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Fred R. Volkmar
Editor

Encyclopedia of Autism Spectrum Disorders

Second Edition

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Encyclopedia of Autism Spectrum Disorders

With 59 Figures and 144 Tables

 Springer

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Preface to Second Edition

Eight years have now passed since the first edition of this Encyclopedia. During that time the field has continued to grow – almost exponentially in some areas! In doing this second edition of the Encyclopedia, we are mindful of the growth as well as some of the advantages for updating entries and including new ones in this much used reference work. It has been gratifying to see this resource being heavily used with around 400,000 downloads since it first appeared!

In this second edition we have added nearly 400 new entries and updates on over 450 previous entries reflecting activity in the field since the first edition.

As with the first edition we have attempted to be comprehensive in scope with entries on a range of topics including not only research issues but biographies of important contributors to the field, legal and social policy issues, educational, behavioral, and medical interventions, treatments, and advances in basic sciences of behavior, communication, neurobiology, genetics, epidemiology, and so forth. For this edition, we have also included a new set of entries on countries giving brief overviews of the history of autism work and the current state of the field in both developed and developing countries. This latter group of entries also reflects the growing interest in autism around the world specifically in developing countries where infrastructure for both service, teaching, and research has become increasingly important. With the addition of our new entries, we have reached nearly 1800 entries in total.

As with the first edition we hope that this work provides an invaluable resource for parents, students, educators, researchers, and professionals alike. Even though these volumes appear in hard copy, in this new second edition we continually update entries and add new ones as these are needed. For this addition, I particularly thank our supporters at Springer – Judy Jones, Tina Shelton, and Sindhu Ramachandran, at Yale my helpful assistants Lori Klein and Monica Mleczek, and at the Autism Center at Southern Connecticut State University my assistant Eileen Farmer. I also particularly thank my Associate Editor Dr. Michael Powers who has assumed an important leadership role in the production of this edition. All of us hope you find this unique resource a valuable and helpful one. We are delighted to welcome you to this second edition.

New Haven, CT, USA
September 1, 2020

Fred R. Volkmar

Preface to First Edition

Why an encyclopedia of autism? There are several answers to this question. They include the need to provide a comprehensive and current guide to the diverse knowledge now available. There has been a significant upsurge in research in autism during the past two decades. Several hundred papers were published in 1991 compared to more than 2,000 articles during 2011. The quantity of research (not even counting non-peer-reviewed publications) has increased so dramatically that it is difficult, if not impossible, for researchers and clinicians to keep up. Access to a reference work that provides an introduction to relevant information is clearly needed.

Although several excellent handbooks and textbooks have been published in recent years, these are, almost intrinsically, fated to become increasingly out of date more and more quickly. Fortunately, many of the same technological advances that have been adapted for use with individuals with autism have uses for those of us who support them. The ability to produce both a print reference work as well as an online version with additional content was a major attraction for us in undertaking this project. It also can be updated easily and will have additional content. The electronic format also provides for an extensive cross-referencing system, which is designed to facilitate rapid searching and information retrieval.

With contributions on a range of topics from leaders in the field, this reference work breaks new ground as a resource. The Encyclopedia contains several thousand entries relevant to autism and related conditions, including new research findings; entries on development and behavior; assessment methods and instruments; treatments and educational interventions; biographies of leaders in the field; and information relevant to epidemiology, social policy, and treatment planning.

Both I and the associate editors of this work hope that you will benefit from using the encyclopedia and welcome your feedback. By the time the print publication of this work appears, the online edition will already have had entries added reflecting new knowledge in various areas. We hope that this resource enhances the work of clinicians and researchers alike.

New Haven, CT, USA
September 2012

Fred R. Volkmar M.D.
Editor

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Fred R. Volkmar is the Irving B. Harris Professor of Child Psychiatry, Pediatrics, and Psychology at the Yale Child Study Center, Yale University School of Medicine, and the Dorothy Goodwin Family Chair of Special Education at Southern Connecticut State University. An international authority on Asperger's disorder and autism, Dr. Volkmar was the primary author of the DSM-IV autism and pervasive developmental disorders section. He has authored several hundred scientific papers and has coedited numerous books, including *Asperger Syndrome, Healthcare for Children on the Autism Spectrum: A Guide to Medical, Nutritional, and Behavioral Issues*, and the recently released third edition of the *Handbook of Autism and Pervasive Developmental Disorders*. He serves as associate editor of the *Journal of Autism*, the *Journal of Child Psychology and Psychiatry*, and the *American Journal of Psychiatry*. He also serves as co-chairperson of the autism/MR committee of the American Academy of Child and Adolescent Psychiatry. Since 2007 he has served editor of the *Journal of Autism* and more recently of the *Encyclopedia of Autism*.

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1-[1-[4,4-Bis(p-fluorophenyl)butyl]-4-piperidyl]-2-benzimidazolinone

► [Pimozide](#)

15q13.3 Microdeletion Syndrome

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Synonyms

[CHRNA7 deletions](#)

Definition

15q13.3 microdeletion syndrome (OMIM 612001, DECIPHER coordinates: chr15: 30,901,306-32,445,407, hg19) is the result of heterozygous deletions at chromosome 15q13.3, ranging in size from ~350 kb to 3.9 Mb. These deletions are mediated by nonallelic homologous recombination (NAHR) between four low copy repeat (LCR) elements: breakpoints (BPs) 3, 4, and 5, as well as the D-CHRNA7-LCR. The most common of these deletions, spanning 1.5 Mb to 2 Mb are mediated by BPs 4 and 5 and encompass six genes: *FAN1*, *MTMR10*, *TRPM1*, *KLF13*, *OTUD7A*, and *CHRNA7*, as well as one microRNA: *hsa-miR-211*. Of these genes, *CHRNA7* and *OTUD7A* are the top candidate genes (Yin et al. 2018; Gillentine and Schaaf 2015).

The estimated frequency of the most common 15q13.3 microdeletions is 1 in 5525 live births (0.19%) and is estimated to be higher (0.29%) among individuals with intellectual disability and idiopathic generalized epilepsy (1%) (Gillentine et al. 2018). However, these deletions also exhibit incomplete penetrance, with about 20% of individuals not having any diagnosed phenotypes. This contributes to 15q13.3 microdeletions being *de novo* (15%) and inherited (85%). Of note, studies have found that a large proportion of 15q13.3 microdeletion probands are adopted; so, while having unknown inheritance, it is likely that these are inherited from affected parents (Ziats et al. 2016).

Individuals carrying 15q13.3 microdeletions have a wide range of phenotypes, including intellectual disability/developmental delay, seizures/epilepsy, autism spectrum disorder (ASD), and schizophrenia (Ziats et al. 2016). In general, probands with 15q13.3 microdeletion syndrome have height, weight, and fronto-occipital circumference within the normal range. Over half of 15q13.3 microdeletion probands exhibit cognitive deficits, with a study of 18 probands finding the average full-scale IQ to be 60 (Ziats et al. 2016; Gillentine and Schaaf 2015). The next most prevalent phenotype is seizures/epilepsy, affecting about one third of probands. Language or speech impairments are also common, affecting just under one third of probands. Other neuropsychiatric phenotypes include schizophrenia, ASD or autistic features, ADHD or attention difficulties, and mood disorders in less than 20% of probands each. Abnormal behaviors, including aggression, and impulsiveness have been observed in about a quarter of the cases. Dysmorphic features are present in about one third of probands, although there is not a consistent pattern of dysmorphia.

While deletions between ~1.5 Mb and ~2 Mb are the most common, but both larger and smaller deletions are reported with similar clinical phenotypes. Notably, homozygous deletions at 15q13.3 have been reported and are phenotypically more severe, with probands exhibiting neonatal encephalopathy. Additionally, the reciprocal microduplication is also pathogenic, with incomplete penetrance as well and a similar range of phenotypes, although typically less severe (Gillentine and Schaaf 2015).

Several hypotheses have been proposed to explain the variable expressivity observed among 15q13.3 microdeletion syndrome probands. These include additional copy number changes and/or single nucleotide variants contributing to phenotypes and epigenetic changes. However, the most prominent hypothesis is the effect of modifier genes, in particular the human-specific fusion gene *CHRFAM7A*, consisting exons 5 through 10 of *CHRNA7* and a sequence of unknown function, *FAM7A*. The fusion gene is copy variable and polymorphic among the

population. Functional studies have shown that increasing amounts of *CHRFAM7A* can contribute to *CHRNA7* dysfunction (Ihnatovych et al. 2019).

Currently, there is no consistent treatment for 15q13.3 microdeletion syndrome. *CHRNA7*, encoding for the $\alpha 7$ nicotinic acetylcholine receptor (nAChR), has been suggested as a candidate gene. Dysfunction of the $\alpha 7$ nAChR is supported molecularly, with a decrease of the receptor resulting in decreased calcium flux through the channel (Gillentine et al. 2017). Due to this, $\alpha 7$ agonists and positive allosteric modulators (PAMs) have been suggested as a possible treatment and assessed among a few individuals with mixed results. One individual carrying a 15q13.3 microdeletion who exhibited recurrent rage outbursts was treated with galantamine, a nAChR allosteric modulator and acetylcholinesterase inhibitor, with positive results, although such drugs are known to have severe side effects (Cubells et al. 2011). Individuals with schizophrenia or autism spectrum disorder have also been treated with nAChR agonists in small studies with positive, but limited results (Olincy et al. 2016). To date, no large clinical trials have been performed using such compounds.

See Also

- [Angelman/Prader-Willi Locus](#)
- [Angelman/Prader-Willi Syndromes](#)
- [Cholinergic System](#)
- [Chromosomal Abnormalities](#)
- [Chromosome 15q11–q13](#)

References and Reading

- Cubells, J. F., DeOreo, E. H., Harvey, P. D., et al. (2011). Pharmacogenetically guided treatment of recurrent rage outbursts in an adult male with 15q13.3 deletion syndrome. *American Journal of Medical Genetics. Part A*, 155, 805–810. <https://doi.org/10.1002/ajmg.a.33917>.
- Gillentine, M. A., Schaaf, C. P. (2015). The human clinical phenotypes of altered *CHRNA7* copy number. *Biochem. Pharmacol.*, 97(4), 352–362. <https://doi.org/10.1016/j.bcp.2015.06.012>

- Gillentine, M. A., Schaaf, C. P., & Patel, A. (2017). The importance of phase analysis in multiexon copy number variation detected by aCGH in autosomal recessive disorder loci. *American Journal of Medical Genetics. Part A*, 173, 2485–2488. <https://doi.org/10.1002/ajmg.a.38328>.
- Gillentine, M. A., Lupo, P. J., Stankiewicz, P., & Schaaf, C. P. (2018). An estimation of the prevalence of genomic disorders using chromosomal microarray data. *Journal of Human Genetics*, 63, 795–801. <https://doi.org/10.1038/s10038-018-0451-x>.
- Ihnatovych, I., Nayak, T. K., Ouf, A., et al. (2019). iPSC model of CHRFAM7A effect on $\alpha 7$ nicotinic acetylcholine receptor function in the human context. *Translational Psychiatry*, 9, 59. <https://doi.org/10.1038/s41398-019-0375-z>.
- Olincy, A., Blakeley-Smith, A., Johnson, L., et al. (2016). Brief report: Initial trial of Alpha7-nicotinic receptor stimulation in two adult patients with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46, 3812–3817. <https://doi.org/10.1007/s10803-016-2890-6>.
- Yin, J., Chen, W., Chao, E. S., et al. (2018). Otud7a knockout mice recapitulate many neurological features of 15q13.3 microdeletion syndrome. *American Journal of Human Genetics*, 102, 296–308. <https://doi.org/10.1016/j.ajhg.2018.01.005>.
- Ziats, M. N., Goin-Kochel, R. P., Berry, L. N., et al. (2016). The complex behavioral phenotype of 15q13.3 microdeletion syndrome. *Genetics in Medicine*, 18, 1111–1118. <https://doi.org/10.1038/gim.2016.9>.
- The importance of deletions and duplications at 16p11.2 in ASD was recognized simultaneously by three research groups (Kumar et al. 2008; Marshall et al. 2008; Weiss et al. 2008). These findings have since been replicated multiple times. 16p11.2 CNVs are found in about 1% of individuals with autism, compared with less than 0.1% of the population. CNVs in this region have also been associated with intellectual disability, developmental delay, schizophrenia (duplications only), and obesity (deletions only) (<http://www.ncbi.nlm.nih.gov/books/NBK11167/>).
- CNVs involving this interval are among the most well-established risk factors for ASD. They also highlight the complexity of the genetic contribution to these syndromes: the CNVs are neither necessary (ASD can occur without 16p11.2 CNVs) nor sufficient (ASD is not always present with the CNV) to cause ASD. Both deletions and duplications can contribute to risk, and these variations may either be de novo or transmitted within families. Moreover, in some families in which one affected child carries a 16p11.2, there may be other affected family members who do not.

The region contains multiple biologically plausible gene candidates for ASD (see list below). At this time, it is not known whether a single gene is responsible for the ASD phenotype or if a combination of genes within the region accounts for the risk.

The genes in the 16p11.2 region are ALDOA, ASPHD1, C16orf53, C16orf54, CDIPT, CORO1A, DOC2A, FAM57B, FLJ25404, GPD3, HIRIP3, INO80E, KCTD13, LOC100271831, LOC440356, MAPK3, MAZ, MVP, PPP4C, PRRT2, QPRT, SEZ6L2, SLC7A5P1, SPN, TAOK2, TBX6, TMEM219, and YPEL3.

16p11.2

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Definition

16p11.2 refers to a particular region on the short (p) arm of chromosome 16 that corresponds to an approximately 500 kilobase copy number variation (CNV) that is strongly associated with the risk for ASD. The region contains 28 genes and is flanked by segmental duplications (stretches of near-identical DNA). These are known to increase the likelihood of a process known as non-homologous allelic recombination, which can lead to gains or losses of the chromosomal segment flanked by these repeats.

See Also

- [Candidate Genes in Autism](#)
- [Chromosomal Abnormalities](#)
- [Common Disease-Rare Variant Hypothesis](#)
- [Copy Number Variation](#)
- [DNA](#)
- [Genetics](#)

References and Reading

- Kumar, R. A., KaraMohamed, S., Sudi, J., Conrad, D. F., Brune, C., Badner, J. A., et al. (2008). Recurrent 16p.112 microdeletions in autism. *Human Molecular Genetics*, 17(4), 628–638.
- Marshall, C. R., Noor, A., Vincent, J. B., Lionel, A. C., Feuk, L., Skaug, J., et al. (2008). Structural variation of chromosomes in autism spectrum disorder. *American Journal of Human Genetics*, 82(2), 477–488.
- Weiss, L. A., Shen, Y., Korn, J. M., Arking, D. E., Miller, D. T., Fossdal, R., et al. (2008). Association between microdeletion and microduplication at 16p.112 and autism. *The New England Journal of Medicine*, 358(7), 667–675.

3-(2-Chloro-10 H-phenothiazin-10-yl)-N,N-dimethylpropan-1-amine Hydrochloride

► Chlorpromazine

3-Chloro-5-[3-(dimethylamino)propyl]-10,11-dihydro-5H-dibenz[b,f]azepine Monohydrochloride

► Clomipramine

3-Day Measles

► Rubella

504 Plan

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Definition

Section 504 is a regulation of the Rehabilitation Act of 1973 that extends civil rights to individuals

with disabilities. Enforced by the Office of Civil Rights (OCR) within the US Department of Health and Human Services, Section 504 states that “*No otherwise qualified individual with a disability in the United States . . . shall, solely by reason of her or his disability, be excluded from the participation in, be denied the benefits of, or be subjected to discrimination under any program or activity receiving Federal financial assistance. . .*” (29 U.S.C. § 794(a)). Section 504 applies to any organization receiving federal funding; thus, it has important implications for individuals with autism spectrum disorders (ASD) and their participation in various educational, recreational, community, and employment settings.

Historical Background

The Civil Rights Act of 1964 and its prohibition of discrimination based on race, color, or national origin was a catalyst for the development of Section 504 of the 1973 Rehabilitation Act. Senator Hubert Humphrey (D., Minnesota) led the work to add an amendment to the Rehabilitation Act of 1973 that would address the discrimination of individuals with disabilities who had not been included under the Civil Rights Act. Section 504 was the first piece of legislation that specifically addressed the civil rights of individuals with disabilities.

Implementation of Section 504 was wrought with challenges. Initial responsibility for writing implementation regulations was left to the US Department of Health, Education, and Welfare (HEW). Though drafts of the regulations were written as early as 1975 (Pfeiffer 2002), by 1977, the regulations had yet to be signed and implementation of Section 504 had stalled. In response, on April 5, 1977, the American Coalition of Citizens with Disabilities (ACCD) led demonstrations in HEW regional offices across the country. These demonstrations and other lobbying efforts led to the signing of the regulations on April 28, 1977. Delays in the creation of government-wide implementation slowed the process of issuing regulations within individual federal agencies (National Council on Disability

2003). Each department within the executive branch of the federal government now has its own regulations for implementing the provisions of Section 504 (Yell 2006).

As the first civil rights legislation for individuals with disabilities, Section 504 of the 1973 Rehabilitation Act paved the way for future legislation for individuals with disabilities, including the 1990 adoption of the Americans with Disabilities Act (ADA) and the Individuals with Disabilities Education Act (IDEA). Together, Section 504, ADA, and IDEA protect the rights and equal participation of individuals with disabilities in employment, in education, and in the community.

Current Knowledge

Qualification Under Section 504

Section 504 specifically states that to be protected under the law, an individual must be determined to (1) have a physical or mental impairment that substantially limits one or more major life activities, (2) have a record of such an impairment, or (3) be regarded as having such an impairment. Though no exhaustive list of specific “mental or physical impairments” covered by Section 504 exists, regulatory provision 34 C.F.R. 104.3(j)(2) (i) defines a physical or mental impairment as “any physiological disorder or condition, cosmetic disfigurement, or anatomical loss . . . or any mental or psychological disorder.” Major life activities, as defined by the Section 504 regulations at 34 C.F.R. 104.3(j)(2)(ii), include functions such as caring for one’s self, performing manual tasks, walking, seeing, hearing, speaking, breathing, learning, and working. It is important to note that this list is also not considered exhaustive, and thus other activities or functions not explicitly stated may be considered “major life activities” under Section 504.

Since autism is a brain-based disorder (Wass 2011), individuals with a diagnosis of ASD would “have record” of a “mental impairment” that could potentially qualify them for protection under Section 504. Qualification is determined based upon the influence of an individual’s autism on his or her ability to perform a “major life activity.”

The characteristics of autism manifest in social interactions, communicative exchanges, and through restricted or stereotyped patterns of behavior, interests, or activities (American Psychiatric Association 2000). Though to qualify for Section 504 each person on the autism spectrum must be evaluated on an individual basis, the disorder could potentially influence many “major life activities.”

Application of Section 504 in Education (From Preschool Through Postsecondary)

The provisions of Section 504 extend civil rights to individuals with disabilities to ensure access to activities and programs for which they “otherwise qualify” (29 U.S.C. § 794(a)). In other words, an individual meets program or employment criteria despite his or her disability. Applied to public education, this means that the individual with a disability is of public school age. Schools providing a public education must ensure that students with disabilities have equal opportunity to benefit from educational programs and facilities under Section 504 (Yell 2006).

A central component of Section 504 as it applies to public schools is the provision of a free appropriate public education (FAPE). FAPE, as defined by Section 504, requires that a student with a disability be provided with regular or special education and related aids and services that are designed to meet his or her individual educational needs. These provisions must meet the individual’s needs as adequately as the needs of students without disabilities are met. Examples relevant to learners with ASD include using visuals to supplement verbal instruction, providing tape recorders, modifying textbooks, using behavior support techniques such as reinforcement, adjusting class schedules, and increasing classroom organization/structure.

Section 504 also requires that all educational programs be accessible to all learners. This does not mean that schools are required to make every room or program accessible to all students but that all learners have equal access to programming. For example, a school may offer multiple sections of a biology lab in three different classrooms. If one of the lab classrooms is accessible and two are not, the school still meets the expectation

of Section 504 because the educational program is accessible to all students. It is not permissible, however, to create a scenario where a disproportionate number of students with disabilities are assigned to the same program or activity because of accessibility issues. Returning to the example of the biology lab, it would not be acceptable for the school to create one section of the lab in which students with disabilities were overrepresented.

This issue of disproportionality, or overrepresentation, is related to the FAPE provision within Section 504 that students with disabilities and students without disabilities should be placed in the same setting, to the maximum extent appropriate to meet the needs of the students with disabilities. In addition, students with disabilities may not be excluded from participating in any school activities, including extracurricular programs such as recreational sports or special interest clubs, in which students without disabilities would participate (US Department of Education, Office of Civil Rights 2010).

Section 504 also requires that students with disabilities access programs and services in “comparable facilities.” In the event that a student with a disability is educated in a separate facility from their peers, a district must ensure that the facility is comparable (i.e., in terms of space, location, size) to the district’s other facilities. Thus, Section 504 protects students with disabilities from the historical practice of establishing special education classrooms in areas not conducive to learning, such as storage rooms or partitioned areas (Yell 2006).

Eligibility Determination

Since Section 504 and IDEA both protect the rights of individuals with disabilities in public education settings (through age 21), there is often confusion about eligibility requirements. It is important to note that not all students with disabilities who qualify for an individualized plan under Section 504 will meet the requirements for special education under IDEA. However, all students protected by IDEA also qualify for protections under Section 504. One reason for this distinction is that under IDEA, a disability must have an adverse impact on a student’s learning

that requires special education and related services. If a student does not require specialized instruction as a result of their disability, then he or she would not meet the requirements of IDEA. While IDEA explicitly requires the involvement of special education programming, implementation of Section 504 is general education responsibility (Yell 2006). Essentially, Section 504 provides *access* to an education (“to and through the schoolhouse door,” Wright and Wright 2008); however, Section 504 includes no guarantee that the individual will receive educational benefit, as specified in IDEA.

In order to determine a student’s eligibility under Section 504, schools are required to follow certain procedural safeguards related to the identification, evaluation, or educational placement of students with a disability (U.S. Department of Education, Office for Civil Rights 2010). An evaluation must occur if a parent or teacher has referred a student, if a student has a medical diagnosis, or if a student has missed an excessive number of school days due to illness. Schools must use an evaluation procedure to determine whether a student’s disability (or perceived disability) limits his or her ability to perform a major life activity, but there is no standardized protocol for how this evaluation should take place.

The FAPE provision requires that once students have been evaluated and determined to meet the criteria for Section 504, school teams must develop an individualized plan that outlines how services and accommodations will be provided. Many students who meet the criteria of Section 504 are also protected under IDEA. These students will therefore have an individualized education program (IEP) that will also constitute their written plan. If a student’s educational needs can be met with accommodations and related services that do not include specialized instruction, they do not typically qualify for special education. These students have only a Section 504 plan that reflects their needs. Finally, a number of rights and safeguards provided by IDEA are not similarly provided to individuals under Section 504, including prior written notice, rights to independent educational evaluations, and protections from permanent expulsion. Table 1

504 Plan, Table 1 Overview of major differences between Section 504 and IDEA

	Section 504	IDEA
Eligibility	Individuals must qualify under the broad definition: (1) have a physical or mental impairment that substantially limits one or more major life activities, (2) have a record of such an impairment, or (3) be regarded as having such an impairment. Need for special education is not a requirement	Students (aged 3–21) must qualify under one of the fourteen disability categories; students must demonstrate need for special education services
Major provisions	No otherwise qualified individual with disability shall solely by reason of his or her disability be: • Excluded from participation in • Denied the benefits of • Be subjected to discrimination under any program or activity receiving federal financial assistance	Procedural safeguards and the right to free appropriate public education in the least restrictive environment as defined by IDEA
Funding	No funding provided for Section 504	Both state and federal funding
Overall responsibility	Local education agency (LEA); general education	State education agency (SEA); special education

A

provides an overview of the supports and services provided under Section 504 and under IDEA.

Application in Postsecondary Education

Any postsecondary institution that receives federal funding is required to apply the regulations of Section 504 for qualifying individuals. Qualifying individuals at the postsecondary education level are those individuals with a disability who also meet the academic or technical standards that are required for admission by the institution. Individuals must also meet the participation requirements for the institution’s activity or program. FAPE does not apply to postsecondary educational settings; instead, institutions are required to provide “appropriate academic adjustments and auxiliary aids and services that are necessary to afford an individual with a disability an equal opportunity to participate in a school’s program” (US Department of Education, Office of Civil Rights 2011). The accommodations and services provided by a postsecondary institution should not alter the individual’s program in a fundamental way nor should they create an “undue burden” on the institution.

Individuals with autism who meet the requirements for Section 504 while in elementary or secondary education should recognize that they might not receive the same services or

accommodations at the postsecondary level. For example, some individuals with autism may be provided support from an educational assistant while in high school. Postsecondary institutions are not required to provide the same service because it may result in an undue financial burden to the institution (US Department of Education, Office of Civil Rights 2007). Another difference in provisions at the postsecondary level is the shift in responsibility. At the elementary and secondary school level, school districts are required to identify, evaluate, and ensure services for an individual with a disability under Section 504. At the postsecondary level, individuals must disclose their disability to the university and follow the institution’s procedures for requesting academic adjustments. Individuals with ASD must be prepared to discuss their individual needs when transitioning to the postsecondary education setting (Adreon and Durocher 2007).

Application in Employment Settings

Any employer who receives federal funding must also fulfill the mandates of Section 504 that protect qualified individuals with a disability. The disability criterion for protection under Section 504 in an employment setting is the same as in educational settings; however, the definition of “qualified” is changed. For the purposes

of employment, in order to be “qualified” an individual with a disability must be able to perform the essential function of the job with reasonable accommodation (US Department of Health and Human Services, Office of Civil Rights 2006). An employer is required to take steps to accommodate an employee’s disability unless doing so would cause an undue burden to the employer.

Workplace accommodations for individuals with disabilities are somewhat intuitive in certain situations (i.e., providing a sign language interpreter for an individual who is deaf or an access ramp for an individual with a physical disability). Workplace accommodations can sometimes be less obvious in the case of an individual with ASD but are no less important in ensuring the individual’s success in the workplace. Accommodations for individuals with autism in the workplace could include minor modifications to work materials or physical changes in the workplace that make the position more accessible. For example, an employer could make the reasonable accommodation of providing a quieter workspace that reduces distractions if such a change would be an appropriate accommodation for the individual with autism.

Future Directions

Increased Prevalence

A recent prevalence study estimated that 2–3% (1:38) of the total school-age population have an autism spectrum disorder (Kim et al. 2011). Many of these students are served in the general education setting (i.e., two-thirds of the sample in the Kim et al. study, 2011) and may not qualify for services under IDEA. This increases the likelihood that individuals with ASD will receive protections under Section 504, which has vast implications for school staff. This resurgence in 504 cases will require that school staff is adept in identifying and implementing appropriate accommodations and modifications for students with ASD – likely requiring additional staff training and expertise. In addition, an increase in litigation around 504 protections is expected as families and schools struggle to identify what accommodations

and auxiliary aids are required. For example, Section 504 does not mandate specific education programs or models nor does it require that students with ASD receive individualized instruction in specialized settings (Katsiyannis and Reid 1999). As this population ages, the demand for Section 504 protections at postsecondary settings, including universities, community colleges, and trade schools, will likely also increase. The resources required to implement these plans, both human resources and financial resources, may create new challenges for these institutions. Finally, employers will likely face similar challenges in supporting employees on the autism spectrum protected by Section 504.

Technology

The use of personal and portable technology with individuals with ASD is on the rise (e.g., iPad, iPod, personal digital assistants, communication devices) (Mechling et al. 2009). These tools are often used to support processing, communication, self-management, self-care, independent functioning, and other “major life activities” (e.g., learning and working, per Section 504). It is not clear, however, whether provisions in Section 504 provide for the procurement/use of these devices, and this ambiguity is likely to be discussed and debated in upcoming years. Though Section 504 requires that auxiliary aids such as technology are provided to individuals with specific disabilities (i.e., hearing or vision impairments) at no additional cost, there is no mention of such supports for individuals with broader developmental delays such as ASD or communication impairments as result of such delays. The fact that no funding is allocated to school districts, postsecondary institutions, or workplaces in association with Section 504 may further complicate the issue of providing technological supports for individuals with ASD.

Social Skills Instruction

Similar questions are likely to arise around the issue of social skills instruction. Because socializing and/or social functioning is described as one of the major life activities under Section 504, accommodations and modifications in this area are

recommended for individuals with ASD (Bellini et al. 2007). These may include peer-mediated strategies, direct social skills instruction, behavioral modification, self-management, and/or other evidence-based social skill strategies. Currently, however, the state of social skills instruction for individuals with ASD who do qualify under IDEA is bleak (Bellini et al.), and little is known about the status of this type of instruction for those who are protected under Section 504. It is safe to assume that services for this population would not exceed that of those who qualify under IDEA and likely also safe to assume that social skills services for the 504 protected group are close to nonexistent. As discussed above, as this population continues to increase, particularly a higher functioning group of students who may not receive services under IDEA, an increased focus on this type of instruction will fall to those implementing Section 504 plans.

See Also

- Academic Supports
- Americans with Disabilities Act
- Employment
- Individual Education Plan
- Individuals with Disabilities Education Act (IDEA)
- Toilet Training

References and Reading

- Adreon, D., & Durocher, J. S. (2007). Evaluating the college transition needs of individuals with high-functioning autism spectrum disorders. *Intervention in School and Clinic*, 42, 271–279.
- Bellini, S., Peters, J., Benner, L., & Hopf, A. (2007). A meta-analysis of school-based social skills interventions for children with autism spectrum disorders. *Remedial & Special Education*, 28, 153–162.
- Katsiyannis, A., & Reid, R. (1999). Autism and section 504: Rights and responsibilities. *Focus on Autism and Other Developmental Disabilities*, 14, 66–72.
- Kim, Y. S., Leventhal, B., Koh, Y. J., Fombonne, E., Laska, E., et al. (2011). Prevalence of autism spectrum disorders in a total population sample. *AJP in Advance*. <https://doi.org/10.1176/appi.ajp.2011.10101532>.
- Mechling, L., Gast, D., & Seid, N. (2009). Using a Personal Digital Assistant to increase independent task completion by students with autism spectrum disorder. *Journal of Autism & Developmental Disorders*, 39, 1420–1434.
- Rehabilitation Act of 1973. Section 504 (34 C.F.R. Part 104), 93rd Congress, H. R. 8070.
- Turnbull, R., Wilcox, B., & Stowe, M. (2002). A brief overview of special education law with focus on autism. *Journal of Autism & Developmental Disorders*, 32, 479–494.
- U.S. Department of Education, Office for Civil Rights. (2010). *Free appropriate public education for students with disabilities: Requirements under section 504 of the rehabilitation act of 1973*. Washington, D.C: U.S. Department of Education, Office for Civil Rights.
- U.S. Department of Health & Human Services. (June 2006). Your rights under the Section 504 and the Americans with Disabilities Act. In *Office for Civil Rights Fact Sheet*. Retrieved May 3, 2011, from <http://www.hhs.gov/ocr/civilrights/resources/factsheets/504.pdf>.
- Wass, S. (2011). Distortions and disconnections: Disrupted brain connectivity in autism. *Brain & Cognition*, 75, 18–28.
- Wright, P. & Wright, P. (March 2, 2008). Key differences between Section 504, the ADA, and the IDEA. In *Wrightslaw.com*. Retrieved May 3, 2011, from <http://wrightslaw.com/>.
- Yell, M. L. (2006). *The law and special education* (2nd ed.). Upper Saddle River: Pearson.

5-HT

- Serotonin

5-Hydroxytryptamine

- Serotonin

7-[4-[4-(2,3-Dichlorophenyl)-1-piperazinyl]butoxy]-3,4-dihydro-2(1H)-quinolinone

- Aripiprazole

7-Dehydrocholesterol Reductase Deficiency

- Smith-Lemli-Opitz Syndrome

7q11.23 Duplications

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Synonyms

[Williams-Beuren region duplication](#)

Definition

7q11.23 duplications are copy number variations (CNVs) in which an extra copy of 1,400 kb of DNA from the long arm of chromosome 7 is present. Duplications in this region are associated with “non-syndromic” ASD (Sanders et al. 2011). The region contains 26 genes, listed below, and is flanked by two segmental duplications (stretches of near-identical DNA). These are known to increase the likelihood of a process known as non-homologous allelic recombination, which can lead to gains or losses of the chromosomal segment flanked by these repeats and account for the common breakpoints seen in the vast majority of individuals carrying duplications in this region. 7q11.23 duplications have also been seen in combination with intellectual disability, speech delay, and cardiac malformations (<http://www.omim.org/entry/609757?search=7q11.23&highlight=7q1123>).

Reciprocal deletions at 7q11.23 cause Williams-Beuren syndrome characterized by distinctive facial features, supravalvular aortic stenosis, and intellectual disability (<http://www.omim.org/entry/194050?search=7q11.23&highlight=7q1123>). Of note, these individuals also are known for highly sociable personalities. The distinctive phenotypes resulting from opposite changes in the number of copies of this region raise the intriguing possibility that the level of expression of a gene, or genes, within the 7q11.23 region plays a key role in the development and/or functioning of the social brain.

The genes in the 7q11.23 region are ABHD11, BAZ1B, BCL7B, CLDN3, CLDN4, CLIP2, DNAJC30, EIF4H, ELN, FKBP6, FZD9, GTF2I, GTF2IRD1, LAT2, LIMK1, MLXIPL,

NSUN5, RFC2, STX1A, TBL2, TRIM50, VPS37D, WBSCR22, WBSCR26, WBSCR27, and WBSCR28.

See Also

- ► [Candidate Genes in Autism](#)
- ► [Chromosomal Abnormalities](#)
- ► [Common Disease-Rare Variant Hypothesis](#)
- ► [Copy Number Variation](#)
- ► [DNA](#)
- ► [Genetics](#)

References and Reading

Sanders, S. J., Ercan-Sencicek, A. G., Hus, V., Luo, R., Murtha, M. T., Moreno-De-Luca, D., et al. (2011). Multiple recurrent de novo CNVs, including duplications of the 7q11.23 Williams syndrome region, are strongly associated with autism. *Neuron*, 70(5), 863–885.

8-Chloro-1-methyl-6-phenyl-4H-s-triazolo [4,3- α] [1,4] Benzodiazepine

► [Alprazolam](#)

AACAP

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Synonyms

[AACAP practice parameters](#)

Definition

One of the first comprehensive guidelines to care of individuals with autism and related disorder, the Practice Parameters of the American Academy

of Child and Adolescent Psychiatry first appears in 1999 (Volkmar et al. 1999) with recommendations for ascertainment and screening, diagnosis, and clinical care. The second version (Volkmar et al. 2014) appeared 15 years later and provided updated guidance for practioners. The original version synthesized available evidence in making recommendations for care anticipating some of the findings and recommendations made by the National Research Council 2 years later (National Research Council 2001).

The initial version was intended to aide in the diagnosis and care and treatment of individuals with autism and related disorder. It provided an overview of the assessment and treatment recommendations with an emphasis on evidence-based treatment practices based on available scientific research. It also noted the need for involvement of multiple care providers with attendant issues of care coordination and so forth.

The second version was updated to reflect the considerable advances in research – particularly treatment research and practice. It focused more specifically on the strength of evidence available in support to the various recommendations in the decade and a half since the first version appeared. The second version explicitly differed in that it explicitly noted the strength of the recommendation – ranging from clinical standard (rigorous evidence), clinical guideline (strong evidence), and clinical option (some but weak or emerging evidence) and not endorsed for treatments that appeared to have no little efficacy based on available research. Explicit distinctions were made based on the strength of the evidence ranging from randomized clinical controlled trials, controlled trials with nonrandomized assignment, uncontrolled trials, and case reports. Issues like care coordination and co-morbidity were also explicitly discussed. Many of the recommendations made were also consistent with the use of the medical home model of care (Hyman and Johnson 2012).

These practice guidelines have many similarities and a few differences from other official guidelines, e.g., relative to issues of screening and early diagnosis; this guideline recommends early screen and encourages early diagnosis while others do not (see, Wilson et al. 2014; McClure 2014, for a discussion). Differences often largely

come from the standards for levels of scientific evidence explicitly adopted by the formulators.

As with all such official guides to care, recommendations should be evaluated in light of current research and practice and the circumstances of the individual case. With that, caveat attempts of this kind are most welcome as they provide clinical guidance for a range of care providers and provide basic recommendations for care.

See Also

- ▶ [Medical Home and ASD](#)
- ▶ [National Guideline for the Assessment and Diagnosis of Autism Spectrum Disorders in Australia](#)
- ▶ [Screening Measures](#)
- ▶ [Sign Language](#)

References and Reading

- Hyman, S. L., & Johnson, J. K. (2012). Autism and pediatric practice: Toward a medical home. *Journal of Autism and Developmental Disorders*, 42(6), 1156–1164.
- McClure, I. (2014). Developing and implementing practice guidelines. In *Handbook of autism and pervasive developmental disorders, volume 2: Assessment, interventions, and policy* (4th ed., pp. 1014–1035). Hoboken: Wiley.
- National Research Council. (2001). *Educating young children with autism*. Washington, DC: National Academy Press.
- Volkmar, F., Cook, E., Jr., Pomeroy, J., Realmuto, G., & Tanguay, P. (1999). Summary of the practice parameters for the assessment and treatment of children, adolescents, and adults with autism and other pervasive developmental disorders. *Journal of the American Academy of Child & Adolescent Psychiatry*, 38(12), 1611–1616.
- Volkmar, F., Siegel, M., Woodbury-Smith, M., King, B., McCracken, J., & State, M. (2014). Practice parameter for the assessment and treatment of children and adolescents with autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(2), 237–257. <https://doi.org/10.1016/j.jaac.2013.10.013>.
- Wilson, C., Roberts, G., Gillan, N., Ohlsen, C., Robertson, D., & Zinkstok, J. (2014). The NICE guideline on recognition, referral, diagnosis and management of adults on the autism spectrum. *Advances in Mental Health and Intellectual Disabilities*, 8(1), 3–14. <https://doi.org/10.1108/AMHID-05-2013-0035>.

AACAP Practice Parameters

► AACAP

Aarskog Syndrome

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Synonyms

[Aarskog-Scott syndrome](#); [Faciogenital dysplasia](#)

Definition

Aarskog syndrome was first reported in 1970 by Aarskog in a seven-patient case series. The syndrome is characterized by short stature with peculiar facies, “shawl” scrotum (the scrotal folds encircle the penis ventrally), cryptorchidism (the testis fails to descend into its normal position in the scrotum), and abnormalities of the hands and feet (Aarskog 1970). Aarskog syndrome can be inherited as an X-linked disorder caused by *FGD1* mutations (Xu et al. 2010; Volter et al. 2014) or possibly in an autosomal dominant or recessive pattern (Xu et al. 2010). Population surveys estimate that Aarskog occurs in approximately 1 per million in the general population (Gorski et al. 2000).

Intelligence ranges from normal to mild mental retardation. A normal IQ distribution has been found (Pilozi-Edwards et al. 2011). Mild learning difficulties and attention deficit hyperactivity disorder have been reported (Pilozi-Edmonds et al.). Comorbidity has been documented with autism (Schwartz et al. 2000).

Birth size is often normal. Alterations occur when individuals are 2–4 years old (Shalev et al. 2006). Until puberty, most patients are short with height at or below the third “centile.” Puberty is often delayed, but these patients do display a growth spurt in the late teens resulting in

adult height in the low-to-normal range. Final height is around the 10th “centile.” Serum growth hormone levels are reported as normal and treatment with growth hormone is ineffective. Spina bifida occulta, cervical spine abnormalities, and scoliosis have been documented (Taub and Stanton 2008).

The nose is often described as short and stubby, with a broad nasal bridge and anteversion of the nostrils. The ears are low set and protuberant. They are fleshy superiorly and referred to as “jug-handle ears.” Maxillary hypoplasia and dental malocclusion has been reported as well as a transverse crease below the lower lip (Taub and Stanton 2008). Associated ophthalmic conditions include hypertelorism, telecanthus, blepharoptosis, and antimongoloid (downward) obliquity of the palpebral fissures. Ophthalmoplegia, strabismus, hyperopic astigmatism, retinal vessel tortuosity, nystagmus, and Brown’s syndrome have also been reported.

The hands and feet are affected by this condition in several ways. The hands are often short and broad with mild syndactyly (interdigital webbing) and/or brachydactyly (shortness in comparison to the other bones and body parts). Hyperextensible joints with concomitant flexion of the distal joints (– a hallmark sign), single palmar creases, and short medially incurved fifth fingers are also found. The feet are broad and flat with metatarsus versus short, splayed bulbous toes (Taub and Stanton 2008).

Genital anomalies include a “shawl” scrotum, bilateral or unilateral cryptorchidism, and macroorchidism (abnormally large testes). Inguinal hernia (a condition in which part of the intestine bulges through a weak area in muscles in the abdomen, specifically the groin) has been found in association with the syndrome. No characteristic anomaly has been documented in females (Taub and Stanton 2008).

There are no specific therapies for Aarskog syndrome. Some features may require surgical intervention (Orrico et al. 2007).

See Also

- [Genetics](#)
- [Strabismus](#)

References and Reading

- Aarskog, D. (1970). A familial syndrome of short stature associated with facial dysplasia and genital anomalies. *The Journal of Pediatrics*, 77, 856–861.
- Gorski, J. L., Estrada, L., Changhzi, H., & Zhou, L. (2000). Skeletal-specific expression of FDG1 during bone formation and skeletal defects in faciogenital dysplasia (FDGY; Aarskog syndrome). *Developmental Dynamics*, 218, 573–586.
- Orrico, A., Galli, L., Obregon, M. G., de Castro Perez, M. F., Falciani, M., & Sorrentino, V. (2007). Unusually severe expression of craniofacial features in Aarskog-Scott syndrome due to a novel truncating variant of the FDG1 gene. *American Journal of Medical Genetics*, 143, 58–63.
- Pilozzi-Edwards, L., Maher, T. A., Basran, R. K., Milunsky, A., Al-Thihli, K., Braverman, N. E., et al. (2011). Fraternal twins with Aarskog-Scott syndrome due to maternal germline mosaicism. *American Journal of Medical Genetics Part A*, 155, 1987–1990.
- Schwartz, C. E., Gillessen-Kaesbach, G., May, M., Cappa, M., Gorski, J., Steindl, K., et al. (2000). Two novel mutations confirm FGD1 is responsible for the Aarskog syndrome. *European Journal of Human Genetics*, 8, 869–874.
- Shalev, S. A., Chevinski, E., Weiner, E., Mazor, G., Friez, M. J., & Schwartz, C. E. (2006). Clinical variation of Aarskog syndrome in a large family with 2189delA in the FGD1 gene. *American Journal of Medical Genetics Part A*, 140(2), 162–165.
- Taub, M. B., & Stanton, A. (2008). Aarskog syndrome: A case report and literature review. *Optometry*, 79, 371–377.
- Volter, C., Martinez, R., Hagen, R., & Kress, W. (2014). Aarskog-Scott syndrome: A novel mutation in the FGD1 gene associated with severe craniofacioal dysplasia. *European Journal of Pediatrics*, 173, 1373–1376.
- Xu, M., Qi, M., Zhou, H., Qui, H., et al. (2010). Familial syndrome resembling Aarskog syndrome. *American Journal of Medical Genetics Part A*, 152A, 2017–2022.

Aarskog-Scott Syndrome

- [Aarskog Syndrome](#)

ABA

- [Didactic Approaches](#)

ABAS, Second Edition

- [Adaptive Behavior Assessment System, Second Edition](#)

ABAS-II

- [Adaptive Behavior Assessment System, Second Edition](#)

ABC

- [Aberrant Behavior Checklist](#)
- [Autism Behavior Checklist](#)

ABC-C

- [Aberrant Behavior Checklist](#)

ABC-R

- [Aberrant Behavior Checklist](#)

Aberrant Behavior Checklist

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Abbreviations

- | | |
|-----|--------------------------|
| ASD | Autism spectrum disorder |
| DD | Developmental disability |

Synonyms

ABC; ABC-C; ABC-R; Aberrant behavior checklist – community; Aberrant behavior checklist – residential

Description

The Aberrant Behavior Checklist (ABC) is an informant rating instrument that was empirically derived by principal component analysis (Aman et al. 1985a). It contains 58 items that resolve onto five subscales. The subscales and the respective number of items are as follows: (a) Irritability (15 items), (b) Social Withdrawal (16 items), (c) Stereotypic Behavior (7 items), (d) Hyperactivity/Noncompliance (16 items), and (e) Inappropriate Speech (4 items). A total score for this instrument was not psychometrically derived and is not valid. The ABC was designed to be completed by any adult who knows the client well. This could be a parent, teacher, workshop supervisor, case worker, or informants in other roles. Depending upon reading ability, completion time varies, but most raters complete the ABC in 10–15 min the first time. Thereafter, rating times usually decline.

A revised version of the ABC was published in 2017, along with a detailed manual (Aman and Singh 2017) and freely available annotated bibliography (<https://psychmed.osu.edu/index.php/instrument-resources>). With respect to the actual content of the scale, although the wording of a handful of items was generalized (e.g., references to “the ward” and “patients” have been altered), the meaning of all items remains the same as in the original version. Subscale titles similarly underwent slight changes; “Irritability, Agitation, Crying” is now entitled *Irritability* and “Lethargy, Social Withdrawal” is now *Social Withdrawal*. Finally, substantive changes to the face sheet were undertaken in an effort to create more usable data. More general terms for school and other settings were used to facilitate comparison, as such terms are variable over time and across geographic location. Rather than querying individual diagnoses, the face sheet now requests explanations and,

where relevant, severity about various conditions that might impact behavior (e.g., sensory or physical impairments, developmental disabilities, medical diagnoses).

The remainder of the face page is unchanged from the previous version; the rater is asked to provide the client’s sex, date of birth, and the rater’s relationship to the client, and a listing of any medicines being used by the client. In the context of treatment studies, this information (other than the subject’s name and date) is often not collected.

Instructions for completing the ABC and its 58 items are found on the second page of the instrument. The period over which informants rate the client defaults to 4 weeks. However, *depending on the clinical or research needs, this period can be increased or decreased*. The instructions ask the informant to rate the client on a scale ranging from 0 (not at all a problem) to 3 (the problem is severe in degree). Further, the instructions ask raters to take relative frequency into account, such that if a given behavior occurs more than the client’s reference group (e.g., other children of the same age and sex), scores greater than or equal to 1 are warranted. The instructions also encourage informants to consider observations and reports of other responsible adults who know the client well when making their ratings. Finally, the instructions indicate that behaviors which interfere with the client’s development, functioning, and/or social relationships should be rated as a problem, even if these behaviors do not interfere with other people around the client. The 58 behavior items consume about 1½ pages of the form.

Initially, the ABC was developed primarily as a measure of treatment effects, especially as an outcome measure for pharmacological intervention. With time, the use of the ABC has expanded, and it has been employed, fairly frequently, for the following applications: (a) to examine psychometric characteristics of other instruments and/or the ABC itself, (b) to study the behavioral phenotypes of individuals with genetic and metabolic conditions, (c) to examine the effects of different environmental variables (e.g., size of housing arrangements) on behavior, (d) to characterize

the composition of subjects within studies and/or programs, (e) to assess the effects of sleep disruption on client behavior, (f) to characterize individuals with different types of psychiatric disorders, and (g) to evaluate quality of life.

There are at least 35 languages into which it has been translated, including the following: Afrikaans, Arabic, Chinese, Czech, Danish, Dutch, Filipino, Finnish, French (Belgian, Canadian, and European), German, Greek, Hebrew, Hungarian, Indonesian, Italian, Japanese, Korean, Lithuanian, Norwegian, Persian (Farsi), Polish, Portuguese, Romanian, Russian, Serbian, Slovak, Slovenian, Spanish (Colombian, Mexican, Spanish, and USA), Swedish, Thai, Turkish, Telugu (regional language of Andhra Pradesh, India), Ukrainian, Urdu, Vietnamese, and Zulu. At the time of this writing, the following language translations were revised for compatibility with the 2017 ABC revision: Afrikaans, Arabic, Canadian French, European French, Chinese (Traditional), English (USA), Filipino, Hebrew, Kannada, Korean, Norwegian, Polish, Portuguese, Russian, Spanish (Spain and USA), and Urdu.

In 2017, a single manual for the community and residential versions of the ABC replaced previous separate versions (Aman and Singh 1986, 1994). This new manual addresses an array of subjects not covered in the original manuals, including sections on giving instructions to raters, practices to avoid, and using the ABC for characterizing change at the individual and group levels. The ABC-Second Edition Community/Residential Manual (Aman and Singh 2017) gives the history of the ABC's development and elaborates upon the meanings of all 58 items. Average subscale scores and standard deviations (normative data) are provided for adults, sourced from developmental centers in the United States and New Zealand. Normative data for teacher ratings of children and adolescents in special educational classes are provided in the following formats: (a) T-scores and percentiles by sex and age, (b) T-scores with all ages and sexes combined, and (c) means and standard deviations broken out by age and sex, as well as collapsed across all ages. The group home norms are presented in

the following ways: (a) T-scores and percentiles by age (10-year groupings) and functional levels (mild, moderate, severe, and profound intellectual disability); (b) T-scores and percentiles collapsed across functional level, summarized for age alone and for sex alone; and (c) means and standard deviations broken out by combinations of functional level and age and summarized by sex alone. Normative data for parent ratings of children and adolescents with intellectual disability are provided as means and standard deviations broken out by age and sex. The manual is also a comprehensive source for information on studies of the psychometric properties of the ABC, including internal consistency, interrater reliability, test-retest reliability, criterion group validity, concurrent and discriminant validity, and correspondence of ratings with direct observation scores. We summarize some of the information contained in the manual herein. There have been about 450 scientific studies conducted with the ABC, providing a rich literature against which new work can be compared.

Historical Background

The development of the ABC grew out of a practical need for an instrument to assess treatment effects in people with DD (e.g., Singh and Aman 1981). Development of the ABC was closely modeled on the Behavior Problem Checklist of Quay and Peterson (Quay 1977) and the enormously popular Conners' Parent and Teacher Rating Scales (Conners 1969, 1970). The initial form of the ABC contained 125 items, developed after a review of residential center case records, a survey of existing instruments, and consultation with direct care staff regarding content and wording. A pilot study obtained ratings from caregivers of 418 adolescents and adults with DDs. Items endorsed for fewer than 10% of subjects were dropped, and a principal factoring method was conducted with oblique rotation, leaving 76 items. The intermediate 76-item scale was then used to rate a new group of 509 adolescents and adults.

The data from both samples were analyzed independently by a principal factoring method

followed by oblique rotation. A five-factor solution seemed most interpretable in both analyses. Items that failed to load on the same respective factors across analyses were deleted, leaving 58 items in the ultimate ABC.

Two important subsequent changes took place more or less simultaneously. First, the original ABC contained some language that was distinctly institutional in flavor (e.g., “excessively active on the ward”). This language was modified in the early 1990s (e.g., “excessively active at home, school, work, or elsewhere”) to form what was then called the ABC-community. At about the same time, investigators assessed the ABC in child samples and found that the original factor structure was maintained for children and adolescents (e.g., Marshburn and Aman 1992; Brown et al. 2002). The earlier version of the ABC was dubbed the ABC-Residential to distinguish it from the newer ABC-Community. Thus, at this stage, there were residential and community versions available, and the Community version’s structure was validated for children, adolescents, and adults.

With time, the ABC came to be used more and more in pharmacological research involving people with intellectual disability and/or autism spectrum disorders (ASDs). Other uses are described under Clinical Uses, below. Much of the published research with the ABC can be accessed through the Annotated Bibliography on the ABC (Aman 2015; available at <http://psychmed.osu.edu/resources.htm>). One important development was the adoption of the ABC’s Irritability subscale as the primary outcome measure by the Research Units on Pediatric Psychopharmacology (RUPP et al. 2002, 2005), a network of experienced psychopharmacology laboratories funded by the US National Institute of Mental Health. In two studies, the RUPP network showed that risperidone was highly effective in reducing agitated and irritable behavior for children and adolescents with autistic disorder chosen for high initial scores on the Irritability subscale. Using data from these pivotal investigations and from another clinical trial, Johnson & Johnson Pharmaceuticals obtained a clinical indication from the United

States Food and Drug Administration for the use of risperidone in children and adolescents with autism and significant agitation and irritability. At that point, it was the only medication approved by the FDA for treating patients with autism. Subsequently, Bristol-Myers Squibb Company launched two pivotal clinical trials of aripiprazole in children and adolescents with autism and agitated/irritable behavior, again with the ABC Irritability subscale as the primary outcome measure. Bristol-Myers Squibb was also able to obtain a clinical indication for its product.

These developments have made the ABC a popular choice as an outcome measure for the pharmaceutical industry when targeting behavior problems in patients with DD. However, it is important to realize that individual academic investigators were using the ABC long before it was adopted as an outcome by industry. In 2015, Bearss et al. published an experiment showing that psychosocial training, administered by parents of children with autism spectrum disorder, was highly effective in reducing disruptive behavior in the children as assessed by parent ratings on the Irritability subscale of the ABC. It seems probable that the ABC will be used widely in future to assess the impact of psychosocial treatments in children with DDs. As noted under Clinical Uses, below, the ABC has been used for approximately 450 pharmacological and non-pharmacological purposes over the last 30+ years.

Psychometric Data

There is a wealth of psychometric data on the ABC.

Construct Validity. There have been several independent factor analyses with the ABC which have supported its construct validity (a) across versions of the ABC, (b) across settings (large residential vs. small, within the community), and (c) across age groups. Most of these studies have been referenced and summarized in the *Annotated Bibliography on the ABC* (Aman 2015; freely available at <http://psychmed.osu.edu/resources.htm>), and they are summarized in Table 1.

Aberrant Behavior Checklist, Table 1 Studies of the construct validity of the ABC

Authors	Residential/ community children/adults	Number of factors	% of items on same factor (mean factor loading)	Coefficient of congruence (mdn)
Aman et al. (1987a)	Res, Adults	5 (Same)	86% (0.58)	0.88–0.96 (0.94)
Newton and Sturmey (1988)	Res, Adults	5 (Same)	78% and 81% ^a	NR
Bihm and Poindexter (1991)	Res, Adults	5 (Same)	NR	NR
Freund and Reiss (1991)	Comm, Childr	5 (Same) (parent)	91%	0.88–0.82 (0.86)
	Comm, Childr	5 (Same) (teacher)	80%	0.65–0.91 (0.81)
Rojahn and Helsel (1991)	Res, Childr	5 (Same)	NR	0.80–0.89 (0.82)
Marshburn and Aman (1992)	Comm, Childr	4 (1–4 Same)	84% (0.65)	0.87–0.96 (0.90)
Aman et al. (1995)	Comm, Adults	5 (Same)	95% (0.59)	0.84–0.97 (0.90)
Ono (1996)	Res, Childr/Adults	5 (Same)	83%	NR
Siegfrid (2000)	Comm, Adults	5 (Same)	84% (0.69)	NR
Brown et al. (2002)	Comm, Childr	4 (1–4 Same)	71% (0.51)	0.62–0.91 (0.85)
Brinkley et al. (2007)	Comm, Childr	5 (Same for low SIB subjects)	78%	NR
		4 (Subscales 2–5 same for high SIB subjects)	60%	NR
Sansone et al. (2012)	Comm, Childr/ Adults	6 (1–5 same)	76%	NR
Kaat et al. (2014)	Comm, Childr	5 (Same)	90%	NR
Wheeler et al. (2014)	Comm, Childr/ Adults	5 (Same)	97%	NR

Same, same factor composition; NR, not reported; mdn, median value

^aUsing ordinal and dichotomous coding (absent/present), respectively

As shown in Table 1, all studies of construct validity essentially verify the ABC factor structure as described in the original report (Aman et al. 1985a). Two studies failed to find the Inappropriate Speech factor in children, possibly because of a lack of participants with ASDs; it is worth noting that a very large study ($n = 1,893$) of children with ASD demonstrated excellent support for the original factor structure (Kaat et al. 2014). One study (Brinkley et al. 2007) found significant changes to the Irritability factor when subjects with high rates of self-injury (SIB) were included, but the factor structure was confirmed when these subjects were excluded.

Other Forms of Validity. The original ABC development study included several validity comparisons (Aman et al. 1985b). Concurrent validity was established through moderate correlations with existing standardized scales (e.g., the AAMD Adaptive Behavior Scale), and comparisons of criterion groups yielded predictable patterns of difference (e.g., individuals who attended formal training activities received lower subscale scores than those who did not). Further, direct observations of the individuals in their residences were well-correlated with ABC scores. Finally, compared to unmedicated individuals, those

prescribed psychotropic medications had significantly higher ABC scores on all domains except Repetitive Speech.

Subsequently, numerous studies have demonstrated the validity of the ABC, and the manual cites about 35 studies addressing validity. Examples of this include concurrent validity between the ABC and other formal instruments, including (a) the Psychopathology Instrument for Mentally Retarded Adults, (b) the Nisonger Child Behavior Rating Form, (c) Conners' Teacher Rating Scale, (d) Diagnostic Assessment for the Severely Handicapped-II, (e) Reiss Screen for Maladaptive Behavior, (f) Stereotyped Behavior Scale, (g) Teacher Report Form, and (h) The ADD-H Comprehensive Teacher's Rating Scale.

Reliability Assessments. Many researchers, especially those who conducted factor analysis with the ABC, reported alpha coefficients – a measure of internal consistency. Generally, coefficient alpha ranged from the low 0.80s to the middle 0.90s, indicating a high level of consistency.

Interrater Reliability. Many of the studies that examined cross-informant reliability are summarized in Table 2. These generally fell into the low 0.50s to high 0.60s range, which is quite adequate for both research and clinical practice. Using criteria established by Cicchetti and Sparrow (1981), these reliabilities fall into the fair to good ranges.

Test-Retest Reliability. Several studies that examined test-retest reliability are summarized in Table 3. Median reliability ranged from the mid-0.60s to highs in the 0.90s. In general, test-retest reliability was quite high, falling within ranges characterized by Cicchetti and Sparrow (1981) as good to excellent.

Clinical Uses

As noted, the ABC was developed as an outcome measure for pharmacological trials in people with developmental disabilities, and it has been used heavily for this purpose (see Annotated Bibliography, Aman 2015). However, use of the scale is not confined to research. The ABC can be used, in combination with other data-based approaches, to monitor the effects of *routine clinical care* in people with intellectual disabilities and/or ASD.

Its early use was primarily among individuals with intellectual disabilities alone, but in recent years it has been used a great deal to assess treatment outcomes in individuals with ASD. This is supported by the available data; one large study ($n = 1,893$) produced very strong evidence for the factor validity of the ABC when used in children and adolescents with ASD. However, it is worth

Aberrant Behavior Checklist, Table 2 Summary of interrater reliability studies with the ABC

Authors	Sample size	Ages of subjects	Correlation range	Median correlation
Aman et al. (1985b)	(a) 35	Adults	0.54–0.67	0.59
	(b) 40	Adults	0.51–0.88	0.71
Aman et al. (1987b)	(a) 28	Adults	0.52–0.74	0.60
	(b) 28	Adults	0.40–0.66	0.59
Freund and Reiss (1991) ^a	94?	Children	0.39–0.49 ^b	0.45 ^b
Rojahn and Helsel (1991)	130	Children/Adolescents	0.39–0.61	0.49
Ono (1996)	33	Children/Adults	0.58–0.78 ^b	0.68
Schroeder et al. (1997)	30	Adults	0.12–0.53	0.45
Siegfrid (2000) ^c	90	Adults	0.67–0.90	0.73
Miller et al. (2004)	22	Children	0.72–0.80	NR

All references can be found in Annotated Bibliography on the ABC (Aman 2015). Unless indicated otherwise, all correlations were Pearson correlation coefficients. Unless coded otherwise, raters had the same roles

^aParent-teacher agreement

^bSpearman correlation coefficients

^cIntraclass correlation coefficients

Aberrant Behavior Checklist, Table 3 Summary of test-retest reliability studies with the ABC

Authors	Lag	Sample size	Age group	Correlation range	Median correlation
Aman et al. (1987b)	4 week	28	Adults	0.55–0.83	0.72 (mean)
Freund and Reiss (1991)	1 month	30 ^a	Children	0.80–0.95	0.88
	1 month	25 ^b	Children	0.50–0.67	0.61
Ono (1996)	4 weeks	43	Children, Adults	0.84–0.90	0.85
Schroeder et al. (1997)	30 days	30	Adults	0.52–0.76	0.59
Siegfrid (2000) ^c	4 week	20	Adults	0.84–0.98	0.94
Miller et al. (2004)	2 weeks	48	Children	0.68–0.85 ^b	NR
				0.74–1.00 ^d	NR
Berry-Kravis et al. (2006)	5 week; 2 week	49	Adults	0.60–0.90 ^e	0.90

All references can be found in the Annotated Bibliography on the ABC (Aman 2015). Unless indicated otherwise, all were Pearson correlation coefficients

- ^aParent ratings
- ^bTeacher ratings
- ^cIntraclass correlation coefficients
- ^dTeaching assistants
- ^eIntraclass correlation coefficient

noting that although several subscales assess features of ASDs (e.g., Social Withdrawal, Stereotypic Behavior, Inappropriate Speech), the ABC was not intended to be a measure of overall autism severity.

As research on specific genetic conditions becomes more common, investigators have attempted to identify syndrome-specific factor structures rather than employing the validated existing structure. This practice is likely to yield unstable results, and researchers are cautioned against this practice (Aman and Singh 2017). Recently, Aman et al. (2020) analyzed extensive data from participants with fragile X syndrome and concluded that the classical scoring algorithm, as presented in the *ABC Manual*, is the optimal way of presenting ABC results.

Periodically, the ABC had been used to assess the effects of behavior intervention, both in formal research (Aman et al. 2009; Bearss et al. 2015) and in everyday care. Obviously, it is important to document the efficacy of such treatment. The ABC has been used to select participants for various forms of research intervention, especially pharmacological investigations. It may serve a similar role in routine clinical care to identify individuals who warrant preventive care and/or

active intervention. As noted earlier, the ABC has been used to monitor behavior in those experiencing transition, such as moving from one living environment to another. It has also been used to assess co-occurring behavioral issues in people with genetic or metabolic syndromes, and this is another likely area of clinical application.

The ABC has primarily been used to assess school-aged children, adolescents, and adults through late middle age. The largest psychometric study of the ABC in preschoolers (*n* = 556, Kaat et al. 2014) produced convincing evidence that it is valid for use in this age group, at least for those with ASD. Although there have been a few studies among elderly people, its utility here has yet to be properly and thoroughly established.

To conclude, the ABC is used to measure and document changes in behavior. These can be changes associated with pharmacological or behavioral intervention or those instigated by environmental alterations. The ABC appears well-suited to assessing a range of ages extending from school-age through late middle age. It has been useful for characterizing the behavior of people with ASD, ID, and a multitude of developmental disability-specific syndromes.

References and Reading

- Aman, M. G. (2015, April). *Annotated bibliography on the Aberrant Behavior Checklist (ABC)* (Unpublished manuscript). The Nisonger Center UAP, Ohio State University, Columbus.
- Aman, M. G., & Singh, N. N. (1986). *Aberrant Behavior Checklist manual*. East Aurora: Slosson Educational Publications.
- Aman, M. G., & Singh, N. N. (1994). *Aberrant Behavior Checklist – Community. Supplementary manual*. East Aurora: Slosson Educational Publications.
- Aman, M. G., & Singh, N. N. (2017). *Aberrant Behavior Checklist manual* (2nd ed.). East Aurora: Slosson Educational Publications.
- Aman, M. G., Singh, N. N., Stewart, A. W., & Field, C. J. (1985a). The Aberrant Behavior Checklist: A behavior rating scale for the assessment of treatment effects. *American Journal of Mental Deficiency, 89*, 485–491.
- Aman, M. G., Singh, N. N., Stewart, A. W., & Field, C. J. (1985b). Psychometric characteristics of the Aberrant Behavior Checklist. *American Journal of Mental Deficiency, 89*, 492–502.
- Aman, M. G., Richmond, G., Stewart, A. W., Bell, J. C., & Kissel, R. C. (1987a). The Aberrant Behavior Checklist: Factor structure and the effect of subject variables in American and New Zealand facilities. *American Journal of Mental Deficiency, 91*, 570–578.
- Aman, M. G., Singh, N. N., & Turbott, S. H. (1987b). Reliability of the Aberrant Behavior Checklist and the effect of variations in instructions. *American Journal of Mental Deficiency, 92*, 237–240.
- Aman, M. G., Burrow, W., & Wolford, P. L. (1995). The Aberrant Behavior Checklist community: Factor validity and effect of subject variables for adults in group homes. *American Journal on Mental Retardation, 100*, 283–292.
- Aman, M. G., Novotny, S., Samango-Sprouse, C., Lecavalier, L., Leonard, E., Gadow, K., et al. (2004). Outcome measures for clinical drug trials in autism. *CNS Spectrums, 9*, 36–47.
- Aman, M. G., McDougle, C. J., Scahill, L., Handen, B., Arnold, L. E., Johnson, C., Stigler, K. A., Bearss, K., Butter, E., Swiezy, N. B., Sukhodolsky, D. D., Ramadan, Y., Pozdol, S. L., Nikolov, R., Lecavalier, L., Kohn, A. E., Koenig, K., Hollway, J. A., Korzekwa, P., Gavaletz, A., Mulick, J. A., Hall, K. L., Dziura, J., Ritz, L., Trollinger, S., Yu, S., Vitiello, B., Wagner, A., & Research Units on Pediatric Psychopharmacology. (2009). Medication and parent training in children with pervasive developmental disorders and serious behavior problems: Results from a randomized clinical trial. *Journal of the American Academy of Child and Adolescent Psychiatry, 48*, 1143–1154.
- Aman, M. G., Norris, M., Kaat, A., Andrews, H., Choo, T.-H., Chen, C., Wheeler, A., Bann, C., & Erickson, C. (2020). *Factor structure of the Aberrant Behavior Checklist in individuals with fragile X syndrome: Clarifications and future guidance* (Unpublished paper). Ohio State University.
- Bearss, K., Johnson, C., Smith, T., Lecavalier, L., Swiezy, N., Aman, M., McAdam, D. B., Butter, E., Stillitano, C., Minshawi, N., Sukhodolsky, D., Mruzek, D. W., Turner, K., Neal, T., Hallett, V., Mulick, J. A., Green, B., Handen, B., Deng, Y., Dziura, J., & Scahill, L. (2015). Parent training for young children with autism spectrum disorder and disruptive behavior: A randomized clinical trial. *Journal of the American Medical Association, 313*, 1524–1533. <https://doi.org/10.1001/jama.2015.3150>.
- Berry-Kravis, E., Krause, S., Block, S., Guter, S., Wu, J., Leurgans, S., et al. (2006). Effect of CX516, an AMPA-modulating compound, on cognition and behavior in fragile X syndrome: A controlled trial. *Journal of Child and Adolescent Psychopharmacology, 16*, 525–540.
- Bihm, E. M., & Poindexter, A. R. (1991). Cross-validation of the factor structure of the Aberrant Behavior Checklist for persons with mental retardation. *American Journal on Mental Retardation, 96*, 209–211.
- Brinkley, J., Nations, L., Abramson, R. K., Hall, A., Wright, H. H., Gabriels, R., et al. (2007). Factor analysis of the Aberrant Behavior Checklist in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 37*(10), 1949–1959.
- Brown, E. C., Aman, M. G., & Haverkamp, S. M. (2002). Factor analysis and norms on parent ratings with the Aberrant Behavior Checklist community for young people in special education. *Research in Developmental Disabilities, 23*, 45–60.
- Cicchetti, D. V., & Sparrow, S. A. (1981). Developing criteria for establishing interrater reliability of specific items: Applications to assessment of adaptive behavior. *American Journal of Mental Deficiency, 86*, 127–137.
- Conners, C. K. (1969). A teacher rating scale for use in drug studies with children. *American Journal of Psychiatry, 126*, 884–888.
- Conners, C. K. (1970). Symptom patterns in hyperkinetic, neurotic, and normal children. *Child Development, 41*, 667–682.
- Freund, L. S., & Reiss, A. L. (1991). Rating problem behaviors in outpatients with mental retardation: Use of the Aberrant Behavior Checklist. *Research in Developmental Disabilities, 12*, 435–451.
- Kaat, A., Lecavalier, L., & Aman, M. (2014). Validity of the Aberrant Behavior Checklist in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 44*, 1103–1116.
- Lecavalier, L., & Aman, M. G. (2005). Rating instruments. In J. L. Matson, R. B. Laud, & M. L. Matson (Eds.), *Behavior modification for persons with developmental disabilities: Treatments and supports* (Vol. 1, pp. 160–189). Kingston: NADD.

- Marshburn, E. C., & Aman, M. G. (1992). Factor validity and norms for the Aberrant Behavior Checklist in a community sample of children with mental retardation. *Journal of Autism and Developmental Disorders*, 22, 357–373.
- Miller, M. L., Fee, V. E., & Netterville, A. K. (2004). Psychometric properties of ADHD rating scales among children with mental retardation I: Reliability. *Research in Developmental Disabilities*, 25, 459–476.
- Newton, J. T., & Sturmey, P. (1988). The Aberrant Behaviour (sic) Checklist: A British replication and extension of its psychometric properties. *Journal of Mental Deficiency Research*, 32, 87–92.
- Ono, Y. (1996). Factor validity and reliability for the Aberrant Behavior Checklist-community in a Japanese population with mental retardation. *Research in Developmental Disabilities*, 17, 303–309.
- Quay, H. C. (1977). Measuring dimensions of deviant behavior: The behavior problem checklist. *Journal of Abnormal Child Psychology*, 5, 277–287.
- Rojahn, J., & Helsel, W. J. (1991). The Aberrant Behavior Checklist with children and adolescents with dual diagnosis. *Journal of Autism and Developmental Disorders*, 21, 17–28.
- RUPP, McCracken, J. T., McGough, J., Shah, B., Cronin, P., Hong, D., et al. (2002). A double-blind, placebo-controlled trial of risperidone in children with autistic disorder. *The New England Journal of Medicine*, 347, 314–321.
- RUPP, Aman, M. G., Arnold, L. E., Lindsay, R., Nash, P., Hollway, J., et al. (2005). Risperidone treatment of autistic disorder: Longer term benefits and blinded discontinuation after six months. *American Journal of Psychiatry*, 162, 1361–1369.
- Sansone, S., Widaman, K., Hall, S., Reiss, A., Lightbody, A., Kaufmann, W., & Hessel, D. (2012). Psychometric study of the Aberrant Behavior Checklist in fragile x syndrome and implications for targeted treatment. *Journal of Autism and Developmental Disorders*, 42, 1377–1392.
- Schroeder, S. R., Rojahn, J., & Reese, R. M. (1997). Reliability and validity of instruments for assessing psychotropic medication effects on self injurious behavior in mental retardation. *Journal of Autism and Developmental Disorders*, 27, 89–102.
- Siegrid, L. W. K. (2000). *A study of the reliability and validity of the Chinese version Aberrant Behavior Checklist* (M.Sc. Thesis, Hong Kong Polytechnic University, Hong Kong).
- Singh, N. N., & Aman, M. G. (1981). Effects of thioridazine dosage on the behavior of severely mentally retarded persons. *American Journal of Mental Deficiency*, 85, 580–587.
- Wheeler, A., Raspa, M., Bann, C., Bishop, E., Hessel, D., Sacco, P., & Bailey, D. (2014). Anxiety, attention problems, hyperactivity, and the Aberrant Behavior Checklist in fragile X syndrome. *American Journal of Medical Genetics Part A*, 164, 141–155.

Aberrant Behavior Checklist – Community

- [Aberrant Behavior Checklist](#)

Aberrant Behavior Checklist – Residential

- [Aberrant Behavior Checklist](#)

ABI, Autism Behavior Inventory

- [Autism Behavior Inventory \(ABI\)](#)

Abilify

- [Aripiprazole](#)

ABI-S, Autism Behavior Inventory-Short Form

- [Autism Behavior Inventory \(ABI\)](#)

ABLLS: Assessment of Basic Language and Learning Skills

- [PEAK Relational Training System](#)

ABLLS-R

- [Assessment of Basic Language and Learning Skills \(ABLLS\)](#)

Abnormal Involuntary Movement Scale

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Synonyms

[AIMS](#)

Definition

A scale used by physicians for evaluating and monitoring abnormal movements such as those associated with tardive dyskinesia which rates the severity of abnormal movements from 0 to 4. The scale is used every 3–6 months to monitor patients taking antipsychotic medications for the development of movement-related side effects. The scale was developed by the Psychopharmacology Research Branch in 1975 and is currently in the public domain.

See Also

- [Atypical Antipsychotics](#)
- [Tardive Dyskinesia](#)

References and Reading

Boyd, M. A. (2008). Appendix D. In *Psychiatric nursing: Contemporary practice* (pp. 891–892). Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins.

Branch, P. R. (1975). Abnormal involuntary movement scale (AIMS). *Early Clinical Drug Evaluation Unit Intercom*, 4, 3–6.

Martinez, M., Marangell, L. B., & Martinez, J. M. (2011). Psychopharmacology. In R. E. Hales, S. C. Yudofsky, & G. O. Gabbard (Eds.), *Essentials of psychiatry* (3rd ed., pp. 455–524). Washington, DC: American Psychiatric Publishing.

Sadock, B. J., & Sadock, V. A. (2003). Biological therapies. In *Synopsis of psychiatry: Behavioral sciences/clinical psychiatry* (pp. 974–1150). Philadelphia: Lippincott Williams & Wilkins.

Wyatt, R. J. (1998). Instructions for using the abnormal involuntary movement scale (AIMS) and AIMS-modified (AIMS-M3D). In *Practical psychiatric practice: Forms and protocols for clinical use* (2nd ed., pp. 77–82). Washington, DC: American Psychiatric Press.

Abnormality

- [Exceptionality](#)

Abolishing Operations

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Definition

Abolishing operations (AO): a general term to describe antecedent events which momentarily decrease the reinforcing or punishing effectiveness of a consequence and therefore alter the future frequency of behavior related to that consequence. AOs, in conjunction with establishing operations (EO; see establishing operation), fall under the greater omnibus term, motivating operation (MO; see motivating operations). AOs involve events which result in a decrease in the effectiveness of a reinforcer or punisher when delivered contingent on a behavior. There are many unconditioned abolishing operations identified in humans. Satiation of food, water, sleep, activity, oxygen, and warmth or cold all function

as abolishing operations for related behavior and reinforcement (Cooper et al. 2007). For example, having just eaten lunch functions as an AO for food as a reinforcer which momentarily decreases any behavior reinforced by food.

Historical Background

Skinner (1938) discussed abolishing operations under the framework of “Drive” and “Drive Conditions,” noting that satiation has an abative effect on behavior. Original work around motivation combined EOs and AOs into one general category. More recently, the AO has been defined and studied as a motivative variable in its own right (Laraway et al. 2003; see motivating operations for a further discussion of the evolution of motivating operations).

Current Knowledge

The concept of the AO has been influential to behavior interventions for individuals with autism spectrum disorders (ASD). The functional assessment of behavior has clearly demonstrated the significance of reinforcement for challenging behavior. Many research studies have focused on reducing challenging behavior by teaching new behaviors which can result in the same reinforcer. Interventions that rely on an AO analysis differ from consequence-based interventions in that they involve the antecedent. Specifically, interventions involving the AO are referred to as antecedent interventions. Antecedent interventions modify or remove the environmental events which precede behavior, to decrease or remove the likelihood of the behavior occurring in the future (Kern and Clemens 2007). An intervention which relies on an AO would seek to abolish or reduce the value of a reinforcer. This reinforcer value-altering effect (Laraway et al. 2003) would subsequently result in a decrease of the target behavior.

For the remainder of this entry, interventions relying on the AO for their effect will be referred to as *AO-based interventions*. For example, an individual with a history of overeating at parties

may instead eat prior to attending a party. This intervention (i.e., satiation of food) would create an AO for food as a reinforcer, thereby decreasing the probability of overeating at the party as previously compared to when an EO for food was in place. The analysis of AOs has successfully contributed to the area of behavior assessment, interventions to reduce behaviors maintained by automatic reinforcement, and interventions to reduce behaviors maintained by social (i.e., positive or negative) reinforcement. These are discussed in detail below.

The Role of AOs in Behavior Assessment

Functional behavior assessment (FBA) and functional analyses (FA) help clinicians to understand how a behavior looks and functions in the environment. These assessments set the foundation for individualized interventions in clinical settings. Furthermore, they have become a “best-practice” component of any program which involves behavior intervention for individuals with ASD. Iwata et al. (1994) established functional analysis (FA) technology to better understand the unique environmental variables that evoke and maintain behaviors of interest. An FA rotates across a variety of conditions which involve an EO for challenging behavior (i.e., demand, alone, denied access, attention). One important component of all functional analyses is a “control” condition (i.e., play condition). In a control condition, there are preferred items (i.e., toys) available, no demands, and social interaction delivered on a time-based schedule. The control condition is specifically set up to function as an AO for challenging behavior. Behavior should not be evoked in this condition unless other reinforcers or processes maintain behavior. Recent research has conceptualized that turning behavior “off” in the control condition (by identifying the relevant AO variables) is just as important as turning it on across other conditions (by identifying relevant EO variables, Hanley et al. 2014). Information about the environmental events acting as an AO for challenging behavior has guided the development of interventions to reduce challenging behavior in individuals with ASD.

Reducing Challenging Behavior with AO-Based Interventions

In addition to contributing to behavior assessment, AOs have been instrumental in developing effective treatment plans. AO-based interventions have demonstrated efficacy with challenging behaviors maintained by automatic reinforcement, as well as those maintained by social reinforcement.

Automatic reinforcement is defined as that which is not mediated by another person. For example, when you feel an itch, scratch it, and it goes away, the reinforcer (i.e., removal of the itch) does not rely on another person. Therefore, the behavior is automatically reinforced. Meaning, in the future, when you feel an itch, you will continue to scratch, because discomfort is removed. This differs from a situation in which you feel an itch, ask someone to scratch the spot, and they scratch it. In the latter scenario, reinforcement is delivered via an individual and is therefore social mediated. Stereotypic behavior, which involves repeated, restricted, and repetitive responses, is frequently maintained by automatic reinforcement. In this case, the behavior may be reinforced by the feeling, sound, or some other quality it itself produces. Automatically maintained challenging behavior can interfere with prosocial repertoires for individuals with ASD and is often difficult to decrease. Historically, interventions targeting behaviors maintained by automatic reinforcement have had limited impact or have necessitated aversive consequences for their effect. When effective, interventions for these types of behaviors often require intensive adult support, which does not lead to maintenance and generalization of suppressed responses (Laprine and Dittrich 2014). AO-based interventions have provided an alternative way to reduce automatically maintained challenging behavior. Examples of these interventions include noncontingent reinforcement (NCR) and pre-session access to reinforcers. The commonality across these interventions is that a known reinforcer is delivered proactively. This differs from procedures which contingently omit reinforcement (i.e., extinction) or deliver an alternate reinforcer (i.e., differential

reinforcement). By satiating an individual on a reinforcer prior to it occurring, an AO may be established which decreases the frequency of the behavior. For example, if a child has previously engaged in repeated scripting of their favorite television show (challenging behavior) because of the preferred sound it creates (reinforcer), delivering access to those sounds proactively may reduce echolalia for a duration of time following the reinforcer delivery. This would be an example of pre-session access to a reinforcer. NCR procedures involve delivering a known reinforcer on a time-based schedule, unrelated to the occurrence or nonoccurrence of a behavior (Cooper et al. 2007). For instance, if a child throws an object (challenging behavior) because of the sound it creates (reinforcer), an AO may be established by proactively providing set times in which designated objects can be thrown to create the same sound. This intervention would create an AO for the sounds as a reinforcer and subsequently reduce throwing behavior for a period of time following intervention. NCR has successfully reduced a variety of topographies of behavior maintained by automatic reinforcement. These include but are not limited to rumination (Carroll et al. 2011), vocal stereotypy (Lang et al. 2009), self-injury (Horner et al. 1991), mouthing (Simmons et al. 2003), as well as behaviors maintained by social reinforcers (discussed in more detail below). Pre-session access to reinforcers has been demonstrated to reduce echolalia (Laprine and Dittrich 2014), vocal stereotypy (Berg et al. 2000; Rispoli et al. 2013), property destruction (O'Reilly et al. 2009), and more general challenging behavior such as aggression or tantrums (Chung and Cannella-Malone 2010; Lang et al. 2010).

AO-based interventions have also been prominently featured in the literature on challenging behaviors maintained by social reinforcement. Social positive reinforcement involves the addition of a preferred stimulus following a behavior (such as attention or a tangible item), while social negative reinforcement involves the removal of something aversive following a behavior (such as a difficult academic assignment). Both positive and negative reinforcement involve reinforcement

that is mediated by someone else. For instance, if a child is presented with academic work, rips it up, and someone takes away the academic demand, the escape (negative reinforcement) is mediated by another person. Several of the same AO-based interventions previously discussed (i.e., NCR and pre-session access to reinforcers) have been used with behaviors maintained by social reinforcement. Vollmer et al. (1995) demonstrated that noncontingent access to escape reduced self-injurious behavior maintained by escape from demands for two young men with developmental disabilities. The researchers provided escape from instruction on a time-based schedule (i.e., every 2 min). The intervention reliably reduced self-injurious behavior across both participating, and over time, the researchers increased the schedule of escape to one that was more naturalistic (i.e., every 10 min), without the reoccurrence of self-injury. Many studies have demonstrated the efficacy of NCR to create AOs for challenging behavior maintained by social negative (Butler and Luiselli 2007) and social positive (Derby et al. 1996; McComas et al. 2003) reinforcement. Along this line of research, several studies have focused on creating an AO for escape-maintained behavior during academic instruction with children with ASD. All interventions involve pairing an environment or environmental situation with high levels of reinforcement. These AO-based interventions have included errorless instruction (Ebanks and Fisher 2003), rate of instruction (Roxburgh and Carbone 2012), stimulus demand fading (Pace et al. 1993), and the high-p request sequence (Mace et al. 1988). One example of an AO-based intervention to decrease escape-maintained problem behavior during academic work involved the technology of pre-session pairing (Kelly et al. 2015). While pre-session pairing is often described a best-practice approach for working with children with ASD, Kelly and colleagues were the first to systematically evaluate the degree to which the intervention created an AO for escape from demands as a reinforcer. In this study the authors provided access to highly preferred reinforcers, in the presence of an instructor, for a set amount of time prior to academic instruction. They found that the intervention

created an AO for escape and attention maintained challenging behavior during academic instruction for three children with ASD who engaged in a variety of challenging behavior (i.e., self-injury, refusal, aggression, and elopement). All participants had decreased levels of challenging behavior and increased responding to instruction following pre-session pairing sessions. Across all studies referenced here, AO-based interventions not only reduced target behaviors but resulted in increased participation with instruction for participants.

A series of studies have also employed the AO to reduce challenging behaviors maintained by social positive reinforcement (Edrisinha et al. 2011; Derby et al. 1996; McComas et al. 2003; McGinnis et al. 2010). McGinnis and colleagues (2010) found that providing pre-session attention to three children with ASD and other developmental disabilities reduced their challenging behavior (self-injury, aggression, property destruction, and tantrums), for up to 15 min in subsequent test sessions. The authors compared a low-AO pre-session condition (with less pre-session exposure to attention) to a high-AO pre-session condition (with more pre-session exposure to attention). The results of the study demonstrated that while both AO conditions resulted in lower levels of problem behavior following exposure, the high-AO sessions resulted in the lowest levels. Marcus and Vollmer (1996) found that noncontingent access to tangible items effectively reduced aggressive and self-injurious behavior in three young children with ASD (two of the participants) and down syndrome (one participant). These studies are example of those which have contributed to the research on AO-based interventions to decrease potentially dangerous and intense challenging behaviors. Importantly, behavior reductions were achieved in many of these studies without the need for punishment or extinction-based interventions.

AO-based interventions have become a popular choice for reducing challenging behaviors for children with ASD. As previously stated, AO-based interventions fall under the umbrella of antecedent interventions. AO-based interventions provide an alternative to interventions that

require intensive schedules of reinforcement, punishment, or extinction (Smith and Iwata 1997). Importantly, these interventions do not rely on another person to respond to each instance of behavior. For this reason, AO-based interventions may require less resources than other interventions such as differential reinforcement. Furthermore, they reduce the probability of a behavior occurring at all, which is important for potentially dangerous behavior (such as self-injury or rumination). Lastly, AO-based interventions may be easier for individuals to implement and reduce the need for hands-on, intensive intervention (Vollmer et al. 1995). One limitation to AO-based interventions is that their impact is relatively short-lived. The reason for this is that MOs function along a continuum. Immediately following the delivery of a reinforcer, an individual is satiated (AO) on that reinforcer. The AO (i.e., satiation) reduces the probability of the behavior related to the reinforcer occurring. As time increases from the delivery of the reinforcer, the satiation will wane and the individual will enter a state of deprivation. This change in states of motivation will create an establishing operation (EO). The EO, by nature of its effect, will increase the probability of the behavior reoccurring. This analysis is important in understanding the long-term effects of AO-based interventions (Michael 2000). Another limitation to these interventions is that they do not change the contingencies which maintain challenging behavior. Therefore, the effect of an AO-based intervention is limited unless the AO remains in place (Iwata et al. 1993).

Future Directions

An understanding of AOs has been demonstrated to be an effective component of interventions to reduce challenging behavior for individuals with ASD. Continued research and application of interventions based in the conceptualization of AOs are necessary to expand the analysis of behavior as it relates to these areas. It is important that the longevity and generality of these interventions continue to be assessed in the literature and in practice with individuals with ASD.

See Also

- Establishing Operations
- Functional Analysis
- Functional Behavior Assessment
- Motivating Operation
- Reinforcement

References and Reading

- Berg, W. K., Peck, S., Wacker, D. P., Harding, J., McComas, J., Richman, D., et al. (2000). The effects of pre-session exposure to attention on the results of assessments of attention as a reinforcer. *Journal of Applied Behavior Analysis*, 33, 463–477.
- Butler, L. R., & Luiselli, J. K. (2007). Escape-maintained problem behavior in a child with autism: Antecedent functional analysis and intervention evaluation of non-contingent escape and instructional fading. *Journal of Positive Behavior Interventions*, 9, 195–202.
- Carroll, R. A., Rapp, J. T., Rieck, T. M., & Siewer, B. N. (2011). The effects of noncontingent reinforcement with alternative oral stimulation in the treatment of rumination. *Journal on Developmental Disabilities*, 17, 72–76.
- Chung, Y. C., & Cannella-Malone, H. I. (2010). The effects of pre-session manipulations on automatically maintained challenging behavior and task responding. *Behavior Modification*, 34, 479–502.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Derby, K. M., Fisher, W. W., & Piazza, C. C. (1996). The effects of contingent and noncontingent attention on self-injury and self-restraint. *Journal of Applied Behavior Analysis*, 29, 107–110.
- Ebanks, M. E., & Fisher, W. W. (2003). Altering the timing of academic prompts to treat destructive behavior maintained by escape. *Journal of Applied Behavior Analysis*, 36, 355–359.
- Edrisinha, C., O'Reilly, M., Sigafoos, J., Lancioni, G., & Choi, H. Y. (2011). Influence of motivating operations and discriminative stimuli on challenging behavior maintained by positive reinforcement. *Research in Developmental Disabilities*, 32, 836–845.
- Hanley, G. P., Jin, C. S., Vanselow, N. R., & Hanratty, L. A. (2014). Producing meaningful improvements in problem behavior of children with autism via synthesized analyses and treatments. *Journal of Applied Behavior Analysis*, 47, 16–36.
- Horner, R. H., Day, H. M., Sprague, J. R., O'Brien, M., & Heathfield, L. T. (1991). Interspersed requests: A nonaversive procedure for reducing aggression and self-injury during instruction. *Journal of Applied Behavior Analysis*, 24, 265–278.
- Iwata, B. A., Dorsey, M. F., Slifer, K. J., Bauman, K. E., & Richman, G. S. (1994). Toward a functional analysis of

- self-injury. *Journal of Applied Behavior Analysis*, 27, 197–209.
- Iwata, B. A., Vollmer, T. R., Zarcone, J. R., & Rodgers, T. A. (1993). Treatment classification and selection based on behavioral function. In R. Van Houten & S. Axelrod (Eds.), *Behavior analysis and treatment* (pp. 101–125). New York: Plenum.
- Kelly, A. N., Axe, J. B., Allen, R. F., & Maguire, R. W. (2015). Effects of pre-session pairing on the challenging behavior and academic responding of children with autism. *Behavioral Interventions*, 30(2), 135–156.
- Kern, L., & Clemens, N. H. (2007). Antecedent strategies to promote appropriate classroom behavior. *Psychology in the Schools*, 44(1), 65–75.
- Lang, R., O'Reilly, M., Sigafoos, J., Lancioni, G. E., Machalicek, W., Rispoli, M., & White, P. (2009). Enhancing the effectiveness of a play intervention by abolishing the reinforcing value of stereotypy: A pilot study. *Journal of Applied Behavior Analysis*, 42, 889–894.
- Lang, R., O'Reilly, M., Sigafoos, J., Machalicek, W., Rispoli, M., Giulio, E. L., et al. (2010). The effects of an abolishing operation intervention component on play skills, challenging behavior, and stereotypy. *Behavior Modification*, 34, 267–289.
- Laprime, A. P., & Dittrich, G. A. (2014). An evaluation of a treatment package consisting of discrimination training and differential reinforcement with response cost and a social story on vocal stereotypy for a preschooler with autism in a preschool classroom. *Education and Treatment of Children*, 37(3), 407–430.
- Laraway, S., Snyderski, S., Michael, J., & Poling, A. (2003). Motivating operations and some terms to describe them: Some further refinements. *Journal of Applied Behavior Analysis*, 36, 407–414.
- Mace, F. C., Hock, M. L., Lalli, J. S., West, B. J., Belfiore, P., Pinter, E., et al. (1988). Behavioral momentum in the treatment of noncompliance. *Journal of Applied Behavior Analysis*, 21, 123–141.
- Marcus, B. A., & Vollmer, T. R. (1996). Combining non-contingent reinforcement and differential reinforcement schedules as treatment for aberrant behavior. *Journal of Applied Behavior Analysis*, 29(1), 43–51.
- McComas, J. J., Thompson, A., & Johnson, L. (2003). The effects of pre-session attention on problem behavior maintained by different reinforcers. *Journal of Applied Behavior Analysis*, 36, 297–307.
- McGinnis, M. A., Houchins-Juárez, N., McDaniel, J. L., & Kennedy, C. H. (2010). Abolishing and establishing operation analyses of social attention as positive reinforcement for problem behavior. *Journal of Applied Behavior Analysis*, 43(1), 119–123.
- Michael, J. (2000). Implications and refinements of the establishing operation concept. *Journal of Applied Behavior Analysis*, 33, 401–410.
- O'Reilly, M. F., Lang, R., Davis, T., Rispoli, M., Machalicek, W., Sigafoos, J., et al. (2009). A systematic examination of different parameters of pre-session exposure to tangible stimuli that maintain problem behavior. *Journal of Applied Behavior Analysis*, 42, 773–783.
- Pace, G. M., Iwata, B. A., Cowdery, G. E., Andree, P. J., & McIntyre, T. (1993). Stimulus (instructional) fading during extinction of self-injurious escape behavior. *Journal of Applied Behavior Analysis*, 26, 205–212.
- Rispoli, M., Camargo, S. H., Neely, L., Gerow, S., Lang, R., Goodwyn, F., et al. (2013). Pre-session satiation as a treatment for stereotypy during group activities. *Behavior Modification*, 38(3), 392–411.
- Roxburgh, C., & Carbone, V. J. (2012). The effects of varying teacher presentation rates on responding during discrete trial training for two children with autism. *Behavior Modification*, 37, 1–26.
- Simmons, J. N., Smith, R. G., & Kliethermes, L. (2003). A multiple-schedule evaluation of immediate and subsequent effects of fixed-time food presentation on automatically maintained mouthing. *Journal of Applied Behavior Analysis*, 36, 541–544.
- Skinner, B. F. (1938). *The behavior of organisms*. Acton: Copley.
- Smith, R. G., & Iwata, B. A. (1997). Antecedent influences on behavior disorders. *Journal of Applied Behavior Analysis*, 30, 343–375.
- Vollmer, T. R., Marcus, B. A., & Ringdahl, J. E. (1995). Noncontingent escape as a treatment for self-injurious behavior maintained by negative reinforcement. *Journal of Applied Behavior Analysis*, 28, 15–26.

Absence Seizures, Second Edition

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Note: In 2017, the International League Against Epilepsy (ILAE) revised the naming of seizures to make them more understandable. Terms like “petit mal” and “pyknolepsy” be avoided

Short Description or Definition

An absence seizure consists of staring as the behavioral change which accompanies abnormal generalized electrical activity in the brain. The

electrical brain activity seen in “typical” absence seizures is generalized 3-Hz spike and wave discharges. Absence seizures are brief (usually less than 15 s) and do not usually result in falling, loss of muscle tone, or jerking of the arms and legs.

Categorization

Absence seizures are categorized as primarily generalized seizures. Childhood absence epilepsy has an onset between 3 and 8 years of age, and juvenile absence epilepsy has onset after 10 years.

Epidemiology

Childhood absence epilepsy (CAE) has an incidence of 6.3–8/100,000 in children less than 15 years of age, and the majority are girls.

CAE represents about 10% of all epilepsies and as such is among the most frequent types of epilepsy. Other seizure types may also occur in children with CAE. It is more common to have other seizure types with juvenile absence epilepsy (JAE).

Absence epilepsy is not reported to occur with greater frequency among children and youth with autism spectrum disorders. It has been associated with specific genes related to GABA function and calcium channel function.

Natural History, Prognostic Factors, and Outcomes

Absence seizures occur most commonly in people under age 20, usually in children ages 6–12. Children who develop typical childhood absence epilepsy (CAE) are usually normal in their development. The seizures are brief, lasting just seconds, but can occur many times a day. They can typically be triggered by hyperventilation. Many children can become seizure-free but the percentage varies. It has been reported that up to 90% of affected children will be seizure-free by adolescence. There may be school and learning difficulties, particularly with verbal memory, seen in patients with CAE. Inattention is reported.

When absence seizures have atypical features, such as EEG findings that are not simply 3-Hz spike and wave, or when the seizures can also be associated with convulsions or myoclonic jerks, it may be harder to become truly seizure-free.

Clinical Expression and Pathophysiology

Absence seizures are related to GABA and voltage-dependent calcium channel functions. Thalamocortical tracts are implicated. Most absence seizures are considered idiopathic or of unknown etiology.

While a genetic association has been identified for a small number of patients, absence epilepsy is idiopathic at this time. It has been reported in children with Angelman syndrome but has not been specifically associated with autism. It may be difficult to clinically differentiate staring episodes from behaviors that occur for other reasons in individuals who are inattentive, who stare, and who might have motor mannerisms on the basis of autism. Absence seizures may be accompanied by a glassy expression (look absent), and affected children may drop things. They may have brief eyelid fluttering or other automatic, subtle movements.

Evaluation and Differential Diagnosis

Evaluation is indicated if staring episodes lasting 5–30 s are observed in children or youth. They may or may not have repetitive motor movements. They will not turn to their name or alert when touched. People do not recall absence seizures.

EEG is the diagnostic study of choice. Characteristic general synchronous, bilateral 2.5–4-Hz spike and slow-wave discharges are seen. Generalized activity on an EEG means that abnormal and synchronized epileptic activity is detected by all EEG electrodes (or most of the cortical surface). If they are frequent enough, a conventional EEG will capture an episode. If less frequent, prolonged monitoring with video may be necessary to identify if a staring episode is a seizure.

As noted, it may be difficult to clinically distinguish absence seizures from inattention,

overfocus, and staring in patients with ASD who might also have stereotyped movements. They are sometimes difficult to distinguish from atypical absence epilepsy and may occur with other seizure types as well. Absence seizures very rarely explain inattention in patients with ADHD.

Since absence epilepsy may result in inattention, there may be a negative effect on school work and social interaction. This may also result in psychosocial stress. The medications used to treat the seizures may further impact attention and learning. Decision on what medication to use must balance all of these factors.

Treatment

The medications used to treat petit mal or absence seizures include ethosuximide, valproic acid, and lamotrigine. The first two have equivalent efficacy but ethosuximide is the initial monotherapy of choice due to fewer cognitive side effects. Forty to seventy percent of children are seizure-free within 4–5 months of therapy. In small studies, topiramate monotherapy has been ineffective for absence seizures. The utility of other anticonvulsants, such as levetiracetam and zonisamide, are under study. Some children with absence seizures that cannot be controlled by any combination of medicines may benefit from a ketogenic diet.

See Also

- [Electroencephalogram \(EEG\)](#)
- [Seizures](#)

References and Reading

- Fisher, R. S., Cross, J. H., D'Souza, C., French, J. A., Haut, S. R., Higurashi, N., et al. (2017). Instruction manual for the ILAE 2017 operational classification of seizure types. *Epilepsia*, 58(4), 531–542. <https://doi.org/10.1111/epi.13671>.
- Glauser, T. A., Cnaan, A., Shinar, S., Hiaz, D. G., Dlugos, P., Masur, D., et al. (2010). Ethosuccimide, valproic acid, and lamictal in children with absence epilepsy. *The New England Journal of Medicine*, 362(9), 790–799.

- Matricardi, S., Verrotti, A., Chiarelli, F., Cerminara, C., & Curatolo, P. (2014). Current advances in childhood absence epilepsy. *Pediatric Neurology*, 50(3), 205–212.

- Spence, S. J., & Schneider, M. T. (2009). The role of epilepsy and epileptiform EEGs in autism spectrum disorders. *Pediatric Research*, 65, 599–606.

- Weiergraber, H., Stephani, U., & Kohling, R. (2010). Voltage gated calcium channels in etiopathogenesis and treatment of absence epilepsy. *Brain Research Reviews*, 62(2), 245–271.

- Yalcin, O. (2012). Genes and molecular mechanisms involved in the epileptogenesis of idiopathic absence epilepsy. *Seizure*, 21(2), 79–86.

Academic Disability

- [Developmental Disabilities](#)

Academic Skills

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Definition

Academic skills have the same meaning within the field of autism as without; they refer to skills in subject areas that form the academic curriculum, available to all children in that country. Increasingly, children and young people within the autism spectrum are entitled to the skills, knowledge, and understanding available to others as a matter of human rights, although there may be problems in exercising these rights where there are additional inherent problems (such as language or intellectual difficulties) or behavioral difficulties. There are also common comorbid conditions that may occur with autism (such as specific learning difficulties: dyslexia, dyspraxia) that may cause particular academic difficulties. However, there are no reasons why individuals with autism should be excluded from any academic area as a result of their autism alone. There may be difficulties in accessing certain

subjects because of the way they are taught or the physical or social context in which they are taught. As with others, success in acquiring academic skills in autism depends on intellectual level, particular talents, and interests, as well as an autism-friendly teaching approach.

Historical Background

Although Kanner (1943) had recognized the biological base of autism, he was later influenced by current psychological theories, which saw autism as a form of childhood schizophrenia with treatment confined to therapy for the child, or the family, depending on the theory of causation adopted. Thus, for two decades following the identification of autism, most children with autism were excluded from academic education of any kind. If there was treatment, it was of a clinical and/or therapeutic kind. It was left to a few pioneering schools (in the UK and Denmark) to demonstrate that these children were able to learn and benefit from education, although even then, the specialist curricula of such schools were largely concerned with teaching adaptive behaviors and practical occupation skills; academic skills were still regarded as largely inappropriate.

Two things changed this picture. Wing (1988) introduced the notion of an autism spectrum that included children and young people with average or above average intellectual ability and good structural language skills (introducing the term “Asperger’s syndrome” to describe such children). It became clear that many of these children (albeit often undiagnosed or misdiagnosed) were already in mainstream schools. Secondly, there grew a worldwide movement for the social inclusion of all children in education with the same entitlement to the culturally valued skills, knowledge, and understanding available to other children and young people in that culture. Inclusion is not about integration alone, where a child may be “allowed” access, but about the designing of curricula and educational systems that take account of all children, in all their diversity and needs, from design to implementation. This is an ideal that is still a “work in progress” in most countries,

but it did open the door to the realization that many children on the autism spectrum could and should benefit from access to the full academic curriculum. The goal was to identify barriers to this process and to seek ways of overcoming them.

The effects of these developments were that children with autism in many countries began to be included in special needs legislation that recognized their entitlement to a broad and relevant curriculum, including academic skills. This did not always mean mainstream education since many children had learning and behavioral difficulties that made full integration problematic, and staffs in mainstream schools were then largely unaware of the special needs of those with autism, and lacked strategies to meet those needs or help the pupils overcome their many barriers to learning. However, special schools often (although not universally) adapted their curricula to include access to academic skills that enabled all their pupils to participate in the national curriculum of their country, albeit often adapted to individual needs. At the same time, as more children with autism were learning to be included in the general educational system available to others, a contrary movement developed from a clinical perspective, which claimed that education for those with autism should first focus on the remedial aspects, training the child in basic adaptive functioning as a precursor to any other form of learning. This was introduced with preschool children and made the claim that such programs would be so successful in remediating core difficulties that no special measures to access the academic curriculum would be needed. Some children appear to have benefited significantly from such intensive behavioral intervention at an early age, although there is no follow-up showing the later effects on learning academic skills (except of the most basic skills of reading and writing). However, research shows that not all children benefit equally (Parsons et al. 2011) and that for some children (especially those of higher ability) it is not relevant to their academic learning. The emphasis on developmental, as opposed to academic, skills, however, has influenced some educational practice, especially in special schools.

The growth of autobiographies of those with autism has also had a profound effect on the understanding of what might be appropriate curricular content for those with autism. Many “successful” individuals with autism demonstrate that success (especially in terms of vocational success and being able to achieve financial independence) depends on building skills and expertise in particular academic subjects at least as much as overcoming supposed “deficits” in functioning. The influence of special interests in guiding and developing academic skills has been shown to be even more important in autism than with other learners and “interest-led” curricula are being developed. Information technology (IT) is not a universal interest of those with autism, but it has been shown to be a valuable medium for learning for many (Murray and Aspinall 2006) and its increasing role in the academic curriculum of many schools has aided participation by those with autism.

The end result has been that many more students with autism are succeeding academically, gaining qualifications at school, and entering further and higher education. Although most people with autism will continue to need understanding and some support even in universities, greater numbers are able to qualify. Sadly, social difficulties and levels of anxiety remain high and may interfere with future job prospects and quality of life (Gelbar et al. 2014). Yet the chance to pursue areas of interest through academic study does of itself improve life for those with autism. Some do attempt to stay within academia, gaining more and more qualifications. Sometimes this is a positive outcome, but for some, it reflects fear and anxiety about moving on from university to the wider world. Those who do succeed provide role models for younger students but also help reinforce the value of academic skills to people with autism. For those with additional learning difficulties, high academic achievement may be out of reach, but academic skills still have relevance in their education. Daily living skills may have a higher priority, but interest and development are fostered by participation in academic tasks and basic academic skills are needed to live a life of dignity and some independence.

Current Knowledge

A study by the Council of Europe a few years ago (Jordan 2009) showed that almost all countries across Europe “included” children and young people with autism within their education systems, although the definition of “education” was varied. For some countries, especially where children with autism had additional learning difficulties, “education” was very like what other countries might describe as “clinical” practice. This was true of some countries that had developed treatment services for people with autism for many decades and where standards of individualized treatment were very high. It is almost as if successful “treatment” is seen as an alternative to inclusive education for some of these children. Even in countries where official policy is for full inclusion for all children with autism (including those with additional difficulties), there remain considerable barriers to its full and successful implementation (Jordan 2008), mostly related to insufficient understanding of autism in mainstream schools. Many developed countries have made significant efforts to increase understanding of autism across the education service with online in-service training of staff and the growth of accredited programs in autism studies.

Even where inclusive practice is well developed, there are usually ways of excluding some children from some aspects of the academic curriculum, where these are not considered relevant to the individual pupil. There will always be some children and young people for whom it is more advantageous to concentrate on a narrower band of academic subjects than is generally taught as part of the national curriculum. This might be because of specific difficulties with subjects that are not considered vital for that individual’s development and future quality of life or it may be because dropping some subjects will allow concentration on other subjects that are more interesting and/or relevant to the individual. The problem is that not all such decisions are evidence based. Ultimately, each decision should be an individual one and there is no good scientific research that can decide which academic subjects will be of benefit to those with autism and which will not.

In fact, it is unlikely that such generalized statements will apply across such a heterogeneous population. Too often, such decisions are made based on assumptions that have not been tested.

Academic Subjects in Relation to Autism

Mathematics: It is often assumed that mathematics will be a strength in autism but this is too broad an assumption. The early stages of mathematics (computation and rule-governed stages) are often areas of strength in autism. However, later stages may produce problems and the aspects that cause problems will vary according to learning style. For visual learners, geometry and graphical work may be strengths but for those who are not visual thinkers (and visual thinking is not universal across the spectrum) this may be a particular difficulty rather than a strength. For the larger group of visual thinkers, algebra rather than geometry may be a problem. Algebra represents a problem because to understand algebra, one has to understand reversibility of operations, which, in turn, requires explicit working memory ability – often a problem in autism. A recent development is software (GRID algebra: Hewett 2016) that makes these internal operations visible (the child can see what operation has been performed and so needs to be reversed), but this awaits evaluation with children with autism.

Even computation skills may be compromised by context and time constraints. When a numeracy program was introduced as a core part of the National Curriculum in the UK, it was expected that this would pose no particular problems for those with autism. But this program emphasized mental arithmetic, conducted at speed in a class context. This proved disastrous for many with autism who could neither concentrate fully in such a group context nor access their answers at speed. It became clear that implicit knowledge of the answer might be there (and could be accessed given time) but there were problems in making the answer explicit and only responding when directed to (inhibiting responses if the teacher did not direct the question specifically at them). As a result many children with autism began to fail at a subject they had previously felt confident in, with disastrous effects on their morale and general learning ability.

One aspect of mathematics, however, has largely unrealized potential in autism: statistics. It is well established that people with autism struggle with uncertainty and that many behavioral issues arise when expected circumstances change or when people find it hard to give definite answers and keep to them. Being told that something “may” happen or that we “will see” if an event unfolds will generally result in much distress in individuals with autism and even challenging behavior. Yet clearly not all of life’s events can be predicted with certainty and people with autism need to be prepared for situations that change. As long as the individual is intellectually able enough to understand, this can be solved by introducing the notion of probability and statistics. In reality, saying that an event has a 90% chance of occurring tomorrow and a 10% chance of not occurring may have little basis in fact, but the numbers seem to make it more acceptable to the person with autism than if one just said it might or might not happen. Degrees of certainty can be refined as the child is taught the variables on which the occurrence depends and the degrees of confidence in that statement. Using such numbers to replace indecisive language not only helps reduce distress and consequent challenging behavior but also gives an acceptable language of numbers for describing and predicting the world. In some cases it can lead to a lifelong interest in statistics and even an occupation using statistics.

On a less positive note, a special ability to calculate at speed may seem like an expression of a high level of mathematical ability that could be utilized in a work situation or be useful for increasing academic ability. But high-speed calculators may have no insight into how answers are reached, that is, no ability to reflect or monitor their own learning. This can be a great drawback when it comes to examinations, where it is important to show working to demonstrate understanding: the actual correct answer carrying less weight than this working out. It can also prevent effective vocational uses of this computational ability. People with autism can sometimes have the capacity to add up a shopping list mentally, for example, but cannot follow the sequential process of recording each item on a cash register. The sad

fact is that no shopper will trust the mental calculations of someone who does not record them on a cash register so an apparent strength ends up having little value.

Literature: Just as mathematics may be assumed to be universally strong in autism, so literature may be seen as a universal problem, but that is equally untrue. Written language is often easier than speech for people with autism, because it does not vary so much between people and situations. Some children with autism come to develop speech through written language for this reason, reversing the typical progression of being able to tell a story by arranging pictures in sequence before learning to read. It is not the sequencing that is a difficulty but the “making sense” of the underlying narrative. It has been suggested, with some research support (Bruner and Feldman 1993; Losh and Capps 2003), that people with autism struggle with many aspects of narrative: understanding the basic narrative structure of events (steady state, event, restoration of the state marked by a coda); telling the gist of an event rather than verbatim details; understanding different roles within an event; keeping track of protagonists within a story by appropriate pronoun use; understanding emotional responses of protagonists; understanding agents and intentional acts. Reading in autism often emerges through reading instructions in computer games or on videos. However, this ability to read short phrases or to memorize large chunks of text is very different from the ability to make sense of longer connected texts such as fictional stories or novels. This is especially true if, as is often the case, there is associated dyslexia in autism. It is paradoxical that individuals with autism may also be hyperlexic, in that they can “read” large chunks of text but in a rote manner, without being able to perceive meaning in the text.

Less commonly, some people with autism are verbal thinkers and have good verbal ability. For these individuals their verbal ability may help with their understanding of the world. For example, linguistic structure can help distinguish actual from reported, or imagined, events and this has been shown to be a factor in some able people with autism learning to develop an understanding of mental states (Theory of Mind). Inasmuch as

literature does involve some of the key difficulties in autism, teaching literature can also be seen as an opportunity to address some of these difficulties: understanding motivations, intentional actions, and their consequences. In written form, these ideas can be addressed at the child’s own pace, rather than trying to be grasped in real-life situations which may pass too quickly and which may be harder to interpret in terms of key events and characters. Literature can provide a structure with which to interpret events and some approaches use written scripts to help the person with autism understand, prepare for, and carry out social actions.

When it comes to writing, there may be dyspraxic or other motor or sensory problems that hinder the development of handwriting skills. It is useful to learn some basic handwriting skills, where possible, and teachers need to take advice from occupational therapists to look at supports (e.g., in posture, in pencil grips) to make this happen. Since typing or touch screen technology means that “writing” (or at least communicating in a visual form) is more accessible to children even with the most severe motor problems, difficulties in handwriting should not be allowed to hinder the expression of ideas. Such technological solutions have enabled some people with autism to demonstrate their ability to think and to express themselves, when it would otherwise have been assumed they were incapable of doing so. Using writing (or an equivalent form), children can also be taught skills such as making a précis of a text, which helps them understand how to extract meaning from a text in a very tangible way.

History: Whether or not history presents a problem for people with autism depends on the nature of the curriculum and how it is taught. If it is presented as a list of facts that can be memorized, then most people with autism (unless they have severe learning difficulties) will manage this without difficulty. However, unless there are clear rules, it can be more difficult to try to assess possible causes for certain events or, even more problematically, try to imagine alternative outcomes. The most difficulties for those with autism, however, are caused by history teaching that requires the pupils to imagine, for example, what it might feel like to have been a Roman

soldier on Hadrian's Wall, or a pilgrim arriving in North America. As with literature, the very fact that history may present some difficulties for pupils with autism can also be seen as an opportunity for teaching. It can be a chance to make explicit some of the things that might affect how someone might feel. This allows pupils with autism to learn more about emotions and to develop a cognitive frame for developing empathy (or at least, sympathy). This does not lead to typical intuitive empathetic understanding, but research shows that a cognitive approach supported with many examples in practice can provide the best approach for people with autism to develop some understanding of others (Mesibov 1986; Ozonoff and Miller 1995); the explicit discussion of motivation and the effects of actions in history may provide this.

Geography: Many individuals with autism prefer to be outdoors rather than confined in buildings (Evans 2015), so they appreciate opportunities to explore their natural environment. For some, this will extend to interest in the geographical features of the outdoors environment and particularly aspects of physical geography. Geographical features of the environment can be explored and explained through laws governing forces of climate, water, volcanoes, particular rock structures, and so on. All of this can provide a logical way of understanding the physical aspects of the environment, without the need for social understanding. On the other hand, the study of populations in social geography can enable some understanding of groups of people, if not of individuals.

Science: Science (and engineering) is usually considered to be one of the most accessible academic subjects for individuals with autism. People with autism are often, mistakenly, thought to have problems with abstract concepts, which would make the abstract concepts of science difficult to master. However, it is not "abstraction" as an explicit description of a concept that causes problems in autism; rather it is the implicit process of abstraction through which everyday "fuzzy" concepts are normally acquired from experience that causes the problems. People with autism therefore have problems with everyday concepts, but

scientific concepts do not rely on this process of abstraction; they are defined explicitly by criterial features and so fit the learning style of those with autism. It is the specificity and explicitness of science that makes it an attractive choice for those with autism. However, there can be some difficulties with the scientific process. People with autism find it difficult to tolerate uncertainty so the scientific method of hypothesis testing can be a problem for them. Once again, however, the process of scientific enquiry can help by specifying the conditions under which facts are established and by being rule governed. Statistics can also help with this understanding and the acceptance of uncertainty.

Foreign Languages: There is a common view in education that, if there is pressure on the curriculum for those with autism because of the need to provide education in social and life skills, then learning a foreign language can be dropped to provide that curricular space. The argument is often made that the person with autism has struggled to master his/her first language so it would be a waste of time to attempt to teach them a second language. There may well be individual cases where this is the correct decision, and certainly curriculum subjects need to be prioritized. But such decisions should always be on an individual basis – not on an assumption that all pupils with autism will struggle with a foreign language. Some may indeed have struggled to acquire their first language and may still have problems with receptive language and with the pragmatic uses of language. A foreign language, however, is not generally taught in the way that a first language is acquired. Everything is made more explicit, so that the processes and structures of the language are much more apparent to the pupils with autism than the implicit understandings that characterize first language acquisition. It may be the first time that students with autism have understood these aspects of language and not only will this make the foreign language easier to acquire but may also help with the understanding of their first language.

In addition, learning a foreign language in a mainstream school is often the only opportunity given to the pupil to be taught everyday social

skills such as greetings, social rules and different language styles, adjusting language to context and useful skills like waiting in restaurants, gaining attention, expressing regret, asking directions, and so on. The fact that these vital social skills are being taught in a foreign language is a minor problem compared with the general failure in mainstream schools to address these important areas of learning at all. Once again, many individuals with autism become very interested in, and skilled at, foreign languages and some are able to obtain employment through acquiring this academic skill.

Few schools remain that teach classical subjects such as Ancient Latin and Greek, but such “dead” languages are also often highly appealing to people with autism. These dead languages do not have the pragmatic learning opportunities of modern foreign languages, but they do offer “pure” academic skills. Because these languages are no longer live, they do not vary according to deictic factors like time, place, and person. Thus, they can be learnt as a system, almost divorced from social meaning, and one that remains unaltered over time.

Information Technology: This relatively new academic subject is not universally attractive or accessible to all individuals with autism, but it has made academic study accessible to many people with autism as well as being a useful tool for accessing other parts of the academic curriculum. Computers can provide a patient, controllable, self-paced, and, above all, nonsocial environment for learning and thus provide access to a large part of the academic curriculum. Information technology can be a rigorous academic subject in its own right also and offer a potential vocational opportunity for many individuals with autism.

Psychology: A minority of schools offer psychology as an academic subject. Although few people with autism will be suited to a career in psychology (in spite of the fact that some have done so), it can be a valuable subject to study as an academic subject. Knowledge of self and others is typically acquired through natural intuitive routes but difficulties in such routes of acquisition are at the heart of autism. People with autism, therefore, have to learn about themselves and others in an

academic way, so the opportunity to engage in this systematically through psychology can be very beneficial. Natural understanding will always be superior (faster and able to happen without effort and alongside other cognitive tasks), but academic psychological skills may be the best route to increased understanding in people with autism. There may still need to be support in applying these academic skills to real-life understanding of self and others, but it is better than having no way to understand.

Future Directions

Technological aids have enabled more individuals with autism gain and demonstrate their potential. This is likely to continue. Technology itself is likely to grow as an academic subject, and there will be more vocational opportunities to develop and apply such technological academic skills. The fact that typical children now also use more technologically driven and explicit ways of learning means that learning styles of students with autism will begin to merge with those of the typical majority of learners. This should aid the development of inclusive practices in education. People with autism may always remain at a disadvantage when it comes to understanding and operating in the social world, but they may be at an advantage when it comes to understanding and operating in the technological world. As technology takes over many low-level cognitive skills (storing and manipulating data, for example), there will be increased need for the exercise of higher-level academic skills – making sense of the data, problem-solving, and interrogating data in meaningful ways. These are high-level skills but they are teachable, and experience shows that what is clearly (and explicitly) taught can be learnt by people with autism, as long as there are no significant learning or other difficulties.

Already it is seen that some academic skills (such as handwriting) have lost some value as other ways of expressing oneself have developed. There may be other academic skills that become redundant, but it is doubtful if humans can flourish and grow without the exercise of some academic skills. It may be that everyone does not need to

learn how to be a historian, say, but everyone needs to understand about how to find sources, how to make sense of them, and to understand notions of trust and reliability in interpreting data. There will be different ways of teaching such skills, but they will be at least as valuable to children with autism as they will be to all.

See Also

- ▶ [Academic Supports](#)
- ▶ [Computer-Based Intervention Assistive Technology](#)
- ▶ [Education](#)
- ▶ [Homework/Assignments, Modifying](#)
- ▶ [Inclusion](#)
- ▶ [Narrative Assessment](#)
- ▶ [Reading](#)
- ▶ [School-Aged Children](#)

References and Reading

- Bruner, J. S., & Feldman, C. (1993). Theories of mind and the problem of autism. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds.), *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.
- Evans, G. (2015). Outdoor adventure programmes: One young man's experiences. *Good Autism Practice*, 16(1), 6–11.
- Gelbar, N., Smith, I., & Reichow, B. (2014). Systematic review of articles describing experience and supports of individuals with autism enrolled in college and university programs. *Journal of Autism and Developmental Disorders*, 44(10), 2593–2601.
- Hewett, D. (2016). GRID algebra Association of Teachers of Mathematics.
- Jordan, R. (2008). Autism spectrum disorders: A challenge and a model for inclusion in education. *British Journal of Special Education*, 35(1), 11–15.
- Jordan, R. (2009). *Education and social integration of children and youth with autism spectrum disorders: Definition, prevalence, rights, needs, provision and examples of good practice*. Strasbourg: Council of Europe.
- Kanner, L. (1943). Autistic disturbance of affective contact. *Nervous Child*, 2, 217–250.
- Losh, M., & Capps, L. (2003). Narrative ability in high-functioning children with autism or Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 33(3), 239–251.
- Mesibov, G. B. (1986). A cognitive program for teaching social behaviors to verbal autistic adolescents and adults. In E. Schopler & G. B. Mesibov (Eds.), *Social behaviour in autism*. New York: Plenum Press.
- Murray, D., & Aspinall, A. (2006). *Getting IT: Using information technology to empower people with communication difficulties*. London: Jessica Kingsley.
- Ozonoff, S., & Miller, J. N. (1995). Teaching theory of mind: A new approach to social skills training for individuals with autism. *Journal of Autism and Developmental Disorders*, 25(4), 415–433.
- Parsons, S., Guldberg, K., MacLeod, A., Jones, G., Prunty, A., & Balfe, T. (2011). International review of the evidence on best practice provision for children on the autism spectrum. *European Journal of Special Needs*, 26(1), 47–63.
- Wing, L. (1988). The continuum of autistic characteristics. In E. Schopler & G. B. Mesibov (Eds.), *Diagnosis & assessment in autism*. New York: Plenum Press.

Academic Supports

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Definition

Academic supports provide students with additional help in specific skill areas or subject areas, such as reading, math, or writing. These may include a small group tutoring session, a test-taking skill program, or other adjustments to the length and difficulty of an assignment, all intended to assist students to reach proficiency in an academic area. Though the term *academic supports* is not used specifically in special education law, it is similar to the term “specially designed instruction,” which is defined in IDEA (Individuals with Disabilities Education Act of 2004) as:

Adapting, as appropriate to the needs of an eligible child...the *content, methodology, or delivery of instruction-*

- i. *To address the unique needs of the child that results from the child's disability; and*
- ii. *To ensure access of the child the general curriculum, so that the child can meet the educational standards within the jurisdiction of the public agency that applies to all children.* [300.39 (b)(3)]

Academic supports can also include accommodations and modifications to a student's scheduling, setting, materials, instruction, and/or student response. *Modifications* change the content that is being taught and/or what is expected of the student, such as providing a text at a different reading level or offering shorter assignments. *Accommodations* change only how the information is received or how the student responds, without altering the content difficulty or student expectations. Accommodations may include providing audiotaped books, allowing answers to be given orally, and using a computer to complete written work. Finally, *supplementary aids* are an additional source of academic support available for students with disabilities, as described in IDEA. These include assistive technology, such as word processors or communication systems; adapted materials, including audio books or highlighted notes; and peer tutors.

Historical Background

Prior to 1975, most individuals with autism spectrum disorders (ASD) in the United States were denied academic instruction in the public schools. These individuals were either not educated or were served in private institutions that focused less on academics and more on the reduction of challenging behavior and/or on the development of life skills (e.g., cooking, cleaning). The passage of the Education for All Handicapped Children Act in 1975 (reauthorized as IDEA in 1990 and including students with autism specifically for the first time) guaranteed for the first time that individuals with ASD and other disabilities could access a free and public education (FAPE). This law also requires that schools and families develop an Individualized Education Program (IEP) which clearly outlines the academic supports (e.g., accommodations, modifications, and supplementary aids) to be provided to the student with ASD. Finally, the law mandates that students with ASD have access to the least restrictive environment (LRE), essentially ensuring that to the maximum extent possible, students with ASD are educated in the general education setting with their nondisabled peers.

Though the law has now been in place for over 30 years, progress in the education of individuals with ASD in the academic domain has been slow. The academic profile of individuals with ASD is complex, and academic skills are often difficult for individuals with ASD to fully demonstrate during assessments and classroom instruction. Historically, most individuals with ASD, as many as 75%, were thought to also have a diagnosis of mental retardation (Ghaziuddin 2000). Due to better instrumentation and understanding of the learning profiles of individuals with ASD, more recent research indicates that approximately 16–30% of the population with ASD has a comorbid condition of mental retardation (now termed “intellectual disability” in the United States) (de Bildt et al. 2004).

Accurately identifying intellectual disabilities in individuals with ASD has been challenging, as has accurately identifying their academic strengths and needs. Individuals with ASD often present an uneven profile of skills, as they may be reading at a very young age (i.e., hyperlexia) but may not be able to describe what they have read or respond verbally to comprehension questions. Similarly, individuals with ASD may have other splinter skills (i.e., a talent or ability in a specific area such as music or calendar knowledge) that may not translate to other areas such as math or reading. Without an accurate understanding of an individual's present level of performance in academic domains, practitioners have had difficulty in developing and implementing appropriate academic supports for students on the autism spectrum.

Current Knowledge

Research in the last decade focused on the cognitive profile of individuals with ASD has informed the field around important and often essential academic supports designed to benefit students with ASD. Following is a brief summary of the processing style of many on the spectrum as well as the state of academic supports currently in use by individuals with ASD. Lastly, a brief description of a number of currently used academic supports will be described.

The Cognitive Profile of Many Individuals with ASD

Auditory and Visual Processing: Research indicates that individuals with ASD may process auditory or linguistic information at a slower rate than their typically developing peers (Cashin and Barker 2009). This auditory processing lag can cause great difficulty during traditional classroom instruction. In addition, research indicates that processing verbal and visual stimuli simultaneously may also be difficult. Visual processing, however, appears to be intact and in fact, can be a strength for individuals on the spectrum.

Weak Central Coherence: Individuals with ASD may have difficulty processing incoming information in context, and instead, the specific details of an event or concept are remembered instead of the “big picture.” This piecemeal processing makes understanding abstract concepts more difficult, as information is stored in chunks without being unified by past experiences or understandings of the world. For example, when recalling a story, individuals with autism are more likely to remember only specific details of the story, perhaps names and locations, rather than the main idea of the story and how it may relate to other stories or past experiences (Hill 2004).

Executive Function: “Executive function” is a term used to describe brain functions such as planning, working memory, and flexibility. These functions are often impaired in individuals with ASD, specifically the ability to plan multi-step sequences of events (e.g., steps required to complete a homework project) and to demonstrate mental flexibility (e.g., shift quickly from one idea or plan to another).

Attention and Inhibition: Individuals with ASD may have difficulty orienting, sustaining, and shifting attention to relevant targets (e.g., the teacher or appropriate topic during instruction) (Patten and Watson 2011). Students with ASD may focus on details that are not relevant, such as a pattern of light created by the blinds or the color of the teacher’s shirt, and miss the most meaningful information or content presented. In addition, individuals with ASD may have difficulty in managing their impulsive behavior (Mesibov et al. 2005).

The State of the Use of Academic Supports

Little is known about what types of academic supports are actually in use by students with ASD, as few researchers have investigated this issue. One source of data, however, has provided the field with a snapshot of the accommodations and modifications used by secondary students with ASD. The National Longitudinal Transition Study 2 (NLTS2) provides data on approximately 1,000 students with ASD ages 14–18 enrolled in secondary education settings. The data indicates that 91% of students with ASD receive some type of academic support or modification in their academic settings (Newman 2007). The types of supports and the percentage of students with ASD who access those supports are listed in Table 1.

Additional learning supports are provided to 81% of the sample (Newman 2007), and those supports are listed below in Table 2.

Finally, 57% of the population used some sort of technology aid to support their academic instruction. See Table 3.

Academic Supports,
Table 1 Accommodations and modifications provided to students with autism

Additional time to complete assignments	52%
More time in taking tests	52%
Alternative tests or assessments	49%
Slower-paced instruction	41%
Shorter or different assignments	38%
Modified tests	33%
Modified grading standards	30%
Tests read to student	25%
Modifications to physical aspects of the classroom	16%

Academic Supports, Table 2 Learning supports provided to students with autism

Monitoring of progress by special education teacher	57%
A teacher’s aide or instructional assistant	55%
More frequent feedback	32%
Learning strategies/study skills assistance	22%
A peer tutor	14%
Self-advocacy training	13%
Tutoring by an adult	9%
A reader or interpreter	6%

Academic Supports, Table 3 Technology aids provided to students with autism

A calculator for activities not allowed other students	28%
Computer software designed for students with disabilities	23%
A computer for activities not allowed other students	16%
Communication aids	16%
Computer hardware adapted for special needs	8%
Books on tape	8%

Description of Commonly Used Academic Supports with Students with ASD

As practitioners gain a better understanding of the cognitive profile of the individuals with ASD that they serve, they are more likely to select meaningful and successful academic supports. Below are some of the most commonly used supports designed to match the academic content and expectations to the strengths and needs of individuals with ASD.

Additional Time: Providing extra time for students with ASD to complete assignments or tests is a common academic support and is recommend for students who have auditory processing lags as described above, as well as for students who may have anxiety, a common co-occurring condition. The time constraints posed by testing protocols may prompt higher levels of anxiety, thus reducing academic success.

Visual Supports: Visuals are a common academic support used by individuals with ASD. Visual supports include any concrete cue that supports verbal explanations and directions provided by teachers. These include diagrams, pictures,

objects to hold, graphic organizers, concept maps, outlines, flowcharts, checklists, and schedules. Descriptions of abstract concepts should include a hands-on and realistic explanation and application, including a visual representation. Students with ASD may benefit from audio recording class lectures and then later transcribing them or using a peer/peer tutor to assist with note-taking. Highlighting text is also a helpful visual support, as students can then clearly “see” what concepts are important. Classroom rules and expectations should also be presented to students with ASD visually to ensure their understanding.

Organizational Supports: Both the instruction and environment should be organized for students with ASD. Assignments should be broken down into clear smaller steps (i.e., task analysis), and those steps may be written or visually represented clearly on a “to-do” list. Feedback and redirection from teachers should be frequent to ensure task completion. Classrooms should be well organized and free of distracters to assist in maintaining the attention of the students with ASD. Establishing a color-coded folder or filing system for the student’s desk or locker may also assist the student in competing and turning in academic assignments.

Computer-Assisted Instruction: Using computers to present academic materials to students with ASD may be beneficial for several reasons, including the increased predictability, frequent feedback and reinforcement, and the limited need for social interaction; another deficit are for students with ASD. Computer-based teaching has been proven to promote achievement in math, spelling, literacy, and problem-solving.

Assistive Technology: Technology can be used as an academic support in a number of other ways including an organizational tool (e.g., using a personal digital assistant to serve as a reminder or provide a to-do list), a teaching tool (e.g., using video to teach a specific academic behavior or skill), a supplement to instruction (e.g., student listens to a book on tape while the class reads it aloud), a communication tool (e.g., a nonverbal student can indicate the correct answer using a communication device), or a basic support (e.g., a calculator).

Strategy Instruction: Learning strategy instruction provides step-by-step processes for students

to follow in classroom settings and situations. For example, individuals with ASD may be taught specific strategies around test-taking, such as how to read instructions, how to respond appropriately (e.g., filling in “bubbles”), and how to reduce anxiety during test-taking. Strategy instruction can also be used to help students with ASD take notes during a lecture, complete large projects such as a term paper, and write an essay. These strategies have been used with students with learning disabilities with great success and have recently been applied to students with ASD (Songlee et al. 2008).

Attention and Motivation Supports: Several supports can contribute to an increased ability to attend to and successfully complete academic tasks. A self-monitoring procedure teaches individuals with ASD to observe their own attending behavior, compare it with predetermined models of behavior (i.e., attending to task), and record if their behavior matches the desired example. Allowing students to choose the sequence in which activities are completed as well as the stimulus used in activities (e.g., choose what color marker to use) is also a proven academic support for students with ASD. Building academic activities around the special interest of a student with ASD can be helpful to increase motivation, as can allowing access to highly preferred materials after the completion of academic work. Finally, pairing nonpreferred academic tasks with preferred academic tasks has been shown to increase task completion as well.

Academic Subject-Specific Supports: The academic supports described above can be applied across academic subjects. Below are supports designed specifically for the following subject areas:

Writing: Writing is often difficult for individuals on the spectrum, likely due to visual-motor and coordination challenges. These may be reduced or alleviated through the use of several supplementary aids such as word processors, voice recognition software, special pencils or grips, or slant boards (Heflin and Alaimo 2007). Teachers may offer reduced writing assignments or allow students to produce outlines rather than lengthier written assignments. Students may also use a note-taker or scribe to assist in reducing the writing load. Beyond the physical difficulties of

writing, the production of written text can be difficult for students with ASD for other reasons, including organizational difficulties and challenges in developing ideas. Academic supports include the use of graphic organizers, planning charts, writing prompts, a word bank, and/or a story grammar map.

Reading: A number of academic supports have been identified to assist in the development of literacy skills. These include several discussed previously, including graphic organizers, multimedia programs, strategy instruction, and highly structured direct instruction (Chiang and Lin 2007). In addition, cooperative groups, one-to-one instruction, interactive books, peer/class-wide tutoring, and flash cards have proven to be effective academic supports in enhancing literacy skills. Reading comprehension can prove especially difficult for students on the spectrum, as broad themes, story meaning, and character motivation may be missed, though recall of specific details and facts may be intact.

Math: Computational skills have generally been a strength for students with ASD; however, difficulty often arises in applying these skills to real tasks or problem-solving (Aspy and Grossman 2007). Academic supports to assist in skill development in this area include the use of practical examples with pictures or diagrams to clarify concepts, the use of visual and tactile cues such as TouchMath, use of graph paper during computation activities to help students organize their problems, increased time to complete math tasks, use of calculators and computer programs, and peer tutoring.

Future Directions

Additional research is needed in the area of academic supports, including a better understanding of what supports are currently in place for elementary-aged students and how effective the supports are in increasing student engagement and academic success. Matching a support with the cognitive strengths and needs of individual students would be most effective, but additional study is required to determine how to accurately assess the academic skills of students with ASD. This is important work, though challenging, as our understanding

of the cognitive profile of students with ASD is changing and evolving as more sophisticated brain research is conducted, including the use of functional MRIs. Additionally, the prevalence of students with ASD appears to be increasing (Kim et al. 2011), which increases the likelihood that all teachers, both special and general education, will be serving students with ASD, thus implementing a number of academic supports. This requires additional staff training, both for in-service and pre-service teachers, as staff must appropriately implement supports determined by the IEP team. Finally, the use of personal and portable technology with individuals with ASD is on the rise (e.g., iPad, iPod, personal digital assistants, communication devices). It is likely that these devices will serve as academic supports for individuals with ASD, as they can provide visual supports (e.g., graphic organizers, video clips), organizational supports (e.g., to-do lists), strategy instruction (e.g., provide step-by-step cues or directions), and motivational supports (e.g., students with ASD are often attracted to the use of technology). Further research on the efficacy of personal technology as an academic support is warranted.

See Also

- Academic Skills
- Computer-Based Intervention Assistive Technology
- Individual Education Plan
- Individuals with Disabilities Education Act (IDEA)
- Modified Testing
- Self-management Interventions
- Visual Supports

References and Reading

- Aspy, R., & Grossman, B. (2007). *The ziggurat model: A framework for designing interventions for individuals with high functioning autism and Asperger syndrome*. Shawnee Mission: Autism Asperger Publishing.
- Cashin, A., & Barker, P. (2009). The triad of impairment in autism revisited. *Journal of Child and Adolescent Psychiatric Nursing*, 22, 189–193.
- Chiang, H., & Lin, Y. (2007). Reading comprehension instruction for students with autism spectrum disorders:

- A review of the literature. *Focus on Autism and Other Developmental Disorders*, 22, 259–267.
- de Bildt, S., Systema, D., Kraijer, A., & Minderaa, R. (2004). Prevalence of pervasive developmental disorders in children and adolescents with mental retardation. *Journal of Child Psychology and Psychiatry*, 46, 275–286.
- Ghaziuddin, M. (2000). Autism in mental retardation. *Current Opinion in Psychiatry*, 13, 481–484.
- Heflin, J., & Alaimo, D. (2007). *Students with autism spectrum disorders*. Upper Saddle River: Pearson.
- Hill, E. (2004). Executive dysfunction in autism. *Trends in Cognitive Sciences*, 8, 26–32.
- Kim, Y. S., Leventhal, B., Koh, Y. J., Fombonne, E., Laska, E., et al. (2011). Prevalence of autism spectrum disorders in a total population sample. *AJP in Advance*. <https://doi.org/10.1176/appi.ajp.2011.10101532>.
- Mesibov, G., Shea, V., & Schopler, E. (2005). *The TEACCH approach to autism spectrum disorders*. New York: Plenum Press.
- Newman, L. (April 2007). Facts from NLTS2: Secondary school experiences of students with autism. Menlo Park: SRI International. Available at www.nlts2.org/fact_sheets/nlts2_fact_sheet_2007_04.pdf.
- Patten, E., & Watson, L. (2011). Interventions targeting attention in young children with autism. *American Journal of Speech-Language Pathology*, 20, 60–69.
- Songlee, D., Miller, S., Tincani, M., Sileo, N., & Perkins, P. (2008). Effects of a test-taking strategy instruction on high functioning adolescents with ASD. *Focus on Autism and Other Developmental Disorders*, 23, 217–228.

Academic Testing

- Educational Testing

Acallosal Syndrome

- Agenesis of Corpus Callosum

ACC

- Anterior Cingulate

Accommodations

- Modified Testing
- Special Needs

Accommodations in Testing

► Modified Testing

Accuracy of the ADOS-2 in Identifying Autism Among Adults with Complex Psychiatric Conditions, The

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Definition

The diagnostic accuracy of the Autism Diagnostic Observation Schedule, Second Edition (ADOS-2; Lord et al. 2012), with adults who present with serious mental illness (e.g., psychosis), chronic mental health problems, and/or multiple comorbid conditions.

Most autistic adults have at least one co-occurring psychiatric condition (e.g., Buck et al. 2014), and overlapping symptoms can make diagnosis difficult. Clinicians need assessment tools that accurately distinguish autism spectrum disorder (ASD) from other psychiatric disorders in adulthood. The ADOS-2 Module 4 is considered a “gold-standard” instrument for collecting standardized and objective information about social communication skills, restricted interests, and repetitive behaviors in adults. It is a widely used, semi-structured assessment tool that allows systematic evaluation of the presence of ASD symptoms. Although the ADOS-2 shows good sensitivity and specificity in university- and lab-based settings (Hus and Lord 2014; Pugliese et al. 2015), it has rarely been studied in community clinics that serve a more psychiatrically impaired population.

In community clinics, ruling out psychosis is likely an important component of an ASD

evaluation. Although distinct in many ways, there are clear areas of symptom overlap between psychosis and ASD (Chisholm et al. 2015; King and Lord 2011). This is particularly true for the negative symptoms of psychosis (e.g., affective flattening, poverty of speech, social withdrawal), which are similar to the core social communication impairments in ASD.

Three studies have shown that Module 4 may not perform well in differentiating between ASD and psychosis. Bastiaansen et al. (2011) found that the ADOS domain and total scores did not significantly differ between autistic adults and adults with schizophrenia using the original algorithm. The Module 4 only correctly classified 74% of cases, with a sensitivity of 61% and specificity of 82%. Using the same sample, de Bildt et al. (2016) applied the revised Module 4 algorithm (Hus and Lord 2014) and also found that the ADOS did not discriminate well between ASD and schizophrenia, with a sensitivity of 61% and specificity of 50%. Bastiaansen et al. (2011), however, found three ADOS items on which autistic adults scored significantly higher than adults with schizophrenia: stereotyped language, quality of social response, and overall quality of rapport. In a separate sample of adults receiving community mental health services ($n = 75$), the ADOS-2 accurately identified all six autistic adults (Maddox et al. 2017). However, there was a high rate of false positives, with a particular limitation in accurately discriminating between ASD and psychosis. All 21 of the false positive cases had a lifetime history of psychosis symptoms. Of the 57 participants with psychosis, 37% exceeded the clinical cut-off score on the ADOS-2. Elevated ADOS-2 scores in the false positive group were driven primarily by high Social Communication domain scores, but not by high Restricted Interests/Repetitive Behaviors domain scores. This finding is likely due to the overlapping nature of the negative symptoms of psychosis and some core ASD symptoms (e.g., flat affect, limited conversation, reduced eye contact).

It is important for clinicians and researchers to remember that prominent social communication difficulties are not specific to ASD, particularly in a clinically complex setting such as community

clinics, where many patients may present with impaired social communication skills. Module 4 of the ADOS-2 provides important information about current social communication skills, restricted interests, and repetitive behaviors. However, the ADOS-2 should be used with caution as a diagnostic instrument with adults, particularly when the differential diagnosis includes the possibility of psychosis.

See Also

- [Bias in Assessment Instruments for Autism](#)
- [Clinical Assessment](#)
- [Mental Health and ASD](#)
- [Psychotic Symptoms in Autism](#)
- [Service Utilization in Autism](#)

References and Reading

- Bastiaansen, J. A., Meffert, H., Hein, S., Huizinga, P., Ketelaars, C., Pijnenborg, M., . . . de Bildt, A. (2011). Diagnosing autism spectrum disorders in adults: The use of Autism Diagnostic Observation Schedule (ADOS) Module 4. *Journal of Autism and Developmental Disorders*, 41, 1256–1266. <https://doi.org/10.1007/s10803-010-1157-x>
- Buck, T. R., Viskochil, J., Farley, M., Coon, H., McMahon, W. M., Morgan, J., & Bilder, D. A. (2014). Psychiatric comorbidity and medication use in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44, 3063–3071. <https://doi.org/10.1007/s10803-014-2170-2>
- Chisholm, K., Lin, A., Abu-Akel, A., & Wood, S. J. (2015). The association between autism and schizophrenia spectrum disorders: A review of eight alternate models of co-occurrence. *Neuroscience and Biobehavioral Reviews*, 55, 173–183. <https://doi.org/10.1016/j.neubiorev.2015.04.012>
- de Bildt, A., Sytema, S., Meffert, H., & Bastiaansen, J. A. (2016). The autism diagnostic observation schedule, Module 4: Application of the revised algorithms in an independent, well-defined, Dutch sample (n = 93). *Journal of Autism and Developmental Disorders*, 46, 21–30. <https://doi.org/10.1007/s10803-015-2532-4>
- Hus, V., & Lord, C. (2014). The Autism diagnostic observation schedule, Module 4: Revised algorithm and standardized severity scores. *Journal of Autism and Developmental Disorders*, 44, 1996–2012. <https://doi.org/10.1007/s10803-014-2080-3>
- King, B. H., & Lord, C. (2011). Is schizophrenia on the autism spectrum? *Brain Research*, 1380, 34–41. <https://doi.org/10.1016/j.brainres.2010.11.031>
- Lord, C., Rutter, M., DiLavore, P. C., Risi, S., Gotham, K., & Bishop, S. L. (2012). *Autism diagnostic observation schedule* (2nd ed.). Torrance: Western Psychological Services.
- Maddox, B. B., Brodtkin, E. S., Calkins, M. E., Shea, K., Mullan, K., Hostager, J., Mandell, D. S., & Miller, J. S. (2017). The accuracy of the ADOS-2 in identifying autism among adults with complex psychiatric conditions. *Journal of Autism and Developmental Disorders*, 47, 2703–2709. <https://doi.org/10.1007/s10803-017-3188-z>
- Pugliese, C. E., Kenworthy, L., Hus-Bal, V., Wallace, G. L., Yerys, B. E., Maddox, B. B., White, S. W., Popal, H., Armour, A. C., Miller, J., Herrington, J. D., Schultz, R. T., Martin, A., & Anthony, L. G. (2015). Replication and comparison of the newly proposed ADOS-2, module 4 algorithm in ASD without ID: A multi-site study. *Journal of Autism and Developmental Disorders*, 45, 3919–3931. <https://doi.org/10.1007/s10803-015-2586-3>

Accuracy of Treatment Implementation

- [Treatment Fidelity](#)

Acetylcholine: Definition

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Synonyms

ACh; Cholinergic

Definition

Acetylcholine (ACh) is a neurotransmitter critical in an individual's ability to assess their surroundings and respond accordingly. More specifically,

ACh functions to evaluate the potential reward and/or threat in a certain stimuli or environmental change and act on it. With roles in regulating attention, cognitive flexibility, social interactions, and stereotypical behaviors, ACh has been heavily implicated in autism. ASD patients show unusually sized, numbered, and structured neurons in the acetylcholine output centers of the basal forebrain as well as decreased concentrations of choline, a precursor of ACh. Low levels of choline have also been correlated with autism severity. Postmortem studies reveal a reduction of ACh receptor and receptor subunits. At the genetic level, mutations and duplications in genes encoding various ACh receptor subunits have been found in ASD patients. Furthermore, mutagenesis, inhibition, and/or deletion of various ACh receptor subunit-encoding genes as well as lesions in ACh-containing cells leads to autistic-like behaviors in rodents. Many of these disturbances in ACh neurotransmission can have direct consequences on synaptic plasticity, a process key in learning and memory. Finally, apart from its direct consequences, ACh also plays an indirect role in the modulation of the balance between excitatory and inhibitory neurons. Perturbations in this balance are hypothesized to contribute greatly to pathogenesis of autism spectrum disorders. No differences have been reported in choline acetyltransferase, involved in the formation of acetylcholine, or acetylcholinesterase, involved in the degradation of acetylcholine, activity between ASD patients and unaffected individuals.

See Also

- [Anticholinesterase Inhibitors](#)
- [Donepezil: Definition](#)

References and Reading

Belmonte, M. K., Cook, E. H., Anderson, G. M., Rubenstein, J. L., Greenough, W. T., Beckel-Mitchener, A., . . . & Tierney, E. (2004). Autism as a disorder of neural information processing: Directions for research and targets for therapy. *Molecular psychiatry*, 9(7), 646–663.

Deutsch, S. I., Urbano, M. R., Neumann, S. A., Burket, J. A., & Katz, E. (2010). Cholinergic abnormalities in autism: Is there a rationale for selective nicotinic agonist interventions? *Clinical Neuropsychopharmacology*, 33(3), 114.

Deutsch, S. I., Schwartz, B. L., Urbano, M. R., Burket, J. A., Benson, A. D., & Herndon, A. L. (2014). Nicotinic acetylcholine receptors in autism spectrum disorders: Therapeutic implications.

Karvat, G., & Kimchi, T. (2013). Acetylcholine elevation relieves cognitive rigidity and social deficiency in a mouse model of autism. *Neuropsychopharmacology*, 39, 831.

Acetylcholinesterase Inhibitors

- [Anticholinesterase Inhibitors](#)

ACh

- [Acetylcholine: Definition](#)

AChE-Inhibitors

- [Anticholinesterase Inhibitors](#)

Achievement Testing

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Definition

Achievement tests are designed to assess an individual's competencies in relation to scholastic material that she/he has been expected to be exposed to in school, home, and community settings (Stetson et al. 2001). Achievement tests are different from intelligence tests. Achievement tests are designed to measure mastery of a specific

subject, or subjects, such as reading ability, number fluency, and scientific knowledge; whereas, intelligence tests are designed to measure both novel problem-solving abilities and stored knowledge (Stedman 2006). Typically, achievement tests are administered in the school setting, as opposed to in mental health clinics (Klin et al. 2005).

Historical Background

Achievement testing has been respected as an accurate tool of academic attainment since 1914 when the Department of Superintendence of the National Education Association officially adopted a favorable view toward educational assessment (Levine 1976), another phrase for achievement testing. Achievement testing was not held in high regard until it was identified as a political tool that both sides, both educators and policymakers, could use to pursue their own interests. However, the origins of achievement testing date back to 1903 when Edward Lee Thorndike and his students developed the Comprehension, Arithmetic, Vocabulary, and Direction following test, better known as the CAVD. Thorndike believed that these four domains were four of the most important dimensions of intellect (Thorndike 1949). In addition to developing four distinct subtests to assess intellect, Thorndike developed scales for the CAVD. While Thorndike was a frontrunner in the development of the achievement test he was primarily interested in measurement of achievement as a utility to establish psychology as a science (Levine 1976).

Achievement tests have come to be critical in the measurement of elementary, middle, and high school students. These tests are used in all states to assess both a student's competency and a school's success. Achievement testing is especially important for high school students hoping to gain entry into college. Lastly, used clinically, achievement tests are administered on a case-by-case basis to identify strengths and weaknesses for academic planning.

The achievement test was revolutionized during the late 1940s and the early 1950s when Henry

Chauncey developed the Census of Abilities. The Census of Abilities was the first test that the Educational Testing Service published, with Chauncey as the first president. Chauncey's goal in creating the first test of achievement was to be able to assess the strengths of every member of society and to utilize these strengths in determining each person's role in society (Lemann 2000). While this ideology would certainly be considered problematic today, the remnants of the Census of Abilities still exist in the form of the Scholastic Aptitude Test, better known as the SAT. The SAT was one of the first standardized tests to assess individual competencies in the subject areas of reading, writing, and math and significantly changed the procedure in which students are selected for admission to university.

Psychologists have been aware of differences between socioeconomic status and race (which are often confounded in the US context), since the beginning of the development of these measures. However, when Alfred Binet determined that significant differences in level of academic functioning existed across different social classes, this information was used to legitimize different educational experiences for different social classes, as opposed to calling to the need for more equitable educational experiences for children across economic background (Levine 1976). Early achievement test findings were also used to discriminate against other marginalized groups such as racial minorities and immigrants deeming them incompetent (Levine 1976).

This pattern of discrimination against lower social classes and marginalized groups continued into the late 1970s, and to some extent still affects minorities and individuals of lower socioeconomic status today. For example, the effects of summer vacation reading recognition regression have been found to be significant among lower-class students, whereas, middle class students saw improvement in this subtest following summer vacations (Cooper et al. 1996).

It has been found that schooling improves achievement and that highly effective schooling raises achievement more. Until recently, achievement testing had been thought to reflect intelligence and the belief was that the influence of

schooling was nonsignificant (Hansen et al. 2004). This new knowledge has many implications for all students, particularly those with some degree of learning difficulty. This new research indicates that quality and fit of schooling could be significant in a child's achievement score.

Current Knowledge

Two types of achievement tests are generally employed: screening for academic delays/deficits and comprehensive tests to characterize profiles of academic achievement functioning. Screening tests are brief and typically contain only one subtest, or a set of questions, for each subject covered. Comprehensive tests utilize more than one subtest for each subject area and generally cover more depth, often in the service of determining appropriate intervention services. Both screening and comprehensive achievement tests routinely assess reading, writing, and mathematics.

Screening tests are generally short and easier to score. This makes them a useful tool to assess whether or not gaps exist within an individual's educational development and prompt whether or not further comprehensive testing may be needed. The Wide Range Achievement Test-4 and the Wechsler Individual Achievement Test-Screener are two commonly used screening tests that have one subtest each of reading, math, and spelling.

Comprehensive tests assess at least three subject areas typically taught in schools, include at least two different subtests from each subject area, and assess both high and lower levels of cognitive ability within each subject area (Stetson et al. 2001). A commonly used comprehensive test is the Wechsler Individual Achievement Test-Comprehensive. A common achievement test used with individuals with an Autism Spectrum Disorder (ASD) is the Woodcock-Johnson III Tests of Achievement. The Woodcock-Johnson III contains 23 different achievement scales or subtests.

In addition to screening and comprehensive achievement tests, there are single-subject versus multiple-subject achievement tests. Single-subject tests include several subtests designed to

explore an individual's competency within one subject area and multiple-subject tests explore several subject areas with one or more subtest (e.g., reading, writing, and mathematics).

Educators and school psychologists often use multiple-subject tests more often than single-subject tests because they assess at least three school subjects and provide preliminary analysis of an individual's overall level of academic achievement. In general, it is recommended that multiple-subject tests be used first in order to assess areas of strengths and weaknesses. Single-subject tests should then be used to further assess an individual's competency in a specific subject area (Stetson et al. 2001).

Single-subject tests allow an assessor to gain a more in-depth understanding of an individual's competency. For example, a single-subject test, such as the Woodcock Reading Mastery Tests – III, includes subtests such as letter identification, word identification, word attack, word comprehension, and passage comprehension. Single-subject tests may be particularly useful in the development of an individualized education plan (IEP) given that they provide detailed information regarding an individual's strengths and weaknesses in a particular subject, thus allowing for a more exact IEP.

Generally, achievement tests are organized with lower-level cognitive tasks first and increase the cognitive difficulty as the task progresses. Achievement tests are organized in this way because the lower the level assessed the less reliable one can predict performance on higher-level skills. Comprehensive tests have several subtests within each subject area and therefore allow several distinct levels of cognition to be assessed, thus allowing a more accurate prediction of achievement. Screener tests, in large part due to only having one subtest per subject, test lower levels of cognition and therefore do not predict achievement as well as comprehensive tests (Stetson et al. 2001).

A note on seasonal norms: achievement tests that include seasonal norms need to be paid close attention to. The difference in standard score of just 1 day can be significant in some tests (Stetson et al. 2001). Additionally, it has been found that

over summer vacation, achievement test scores tend to regress. Of the three core subjects assessed (reading, writing, and mathematics), it was found that math skills seemed to deteriorate the most (Cooper et al. 1996).

When completing achievement testing with an individual who has an ASD, choosing the right achievement test should depend on the specific needs of the individual. For example, some individuals with an ASD struggle with maintaining their attention and should be administered a screening test to maximize concentrated performance (Koegel et al. 1997). Whereas, other individuals with an ASD may be able to focus for long periods of time but may have considerable gaps in knowledge and a more comprehensive test may be the more appropriate choice (Koegel et al. 1997).

Tests that include visual stimuli and that do not require long verbal responses may also be most appropriate for some individuals with ASD. For example, the Peabody Individual Achievement Test – Revised (PIAT-R) touts a multiple choice format that is designed to be easy to use with individuals having severe disabilities. While the simple administration and multiple choice responses certainly make the PIAT-R a desirable choice for testing individuals with severe disability, it should be noted that this test was developed with a typical population and therefore the norms do not address the unique needs of individual special needs populations.

Future Directions

While considerable gains have been made in the development of achievement tests since they were first developed in the early 1900s, it is imperative that research and development of new tests continue to create measures that represent the abilities of all individuals. When considering the development of new measures, it is important to take into consideration the needs of the groups that most often use achievement tests, aside from those used in state and nationwide testing. Additionally, future editions of achievement tests should strive to include norms for different populations. It would be especially useful, given the number of individuals

affected, if norms for the ASD population were provided. These norms would provide helpful insight to providers and parents regarding what is typical and could be expected of children in this population over the course of their development.

See Also

- Educational Testing
- Peabody Individual Achievement Test, Revised
- Psychological Assessment
- Wechsler Preschool and Primary Scale of Intelligence
- Wide Range Assessment of Memory and Learning (WRAML)
- Woodcock-Johnson Cognitive and Achievement Batteries

References and Reading

- Cooper, H., Nye, B., Charlton, K., Lindsay, J., & Greathouse, S. (1996). The effects of summer vacation on achievement test scores: A narrative and meta-analytic review. *Review of Educational Research*, 66(3), 227–268. <https://doi.org/10.3102/00346543066003227>.
- Hansen, K. T., Heckman, J. J., & Mullen, K. J. (2004). The effect of schooling and ability on achievement test scores. *Journal of Econometrics*, 121(1–2), 39–98. <https://doi.org/10.1016/j.jeconom.2003.10.011>.
- Klin, A., Saulnier, C. D., Tsatsanis, K. D., & Volkmar, F. R. (2005). Clinical evaluation in autism spectrum disorders: Psychological assessment within a transdisciplinary framework. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.
- Koegel, L. K., Koegel, R. L., & Smith, A. (1997). Variables related to differences in standardized test outcomes for children with autism. *Journal of Autism and Developmental Disorders*, 27(3), 233–243. 0162-3257/97J0600-0233\$12.50/0.
- Lemann, N. (2000). *The big test*. New York: Farrar, Straus and Giroux.
- Levine, M. (1976). The academic achievement test: Its historical context and social functions. *American Psychologist*, 31(3), 228–238. <https://doi.org/10.1037/0003-066X.31.3.228>.
- Markwardt, F. C. (1997). *Peabody individual achievement test – Revised/normative update*. Bloomington: Pearson Assessments.
- Stedman, T. L. (2006). *Stedman's medical dictionary* (28th ed.). Philadelphia: Lippincott Williams & Wilkins.

- Stetson, R., Stetson, E. G., & Sattler, J. M. (2001). Assessment of academic achievement. In J. M. Sattler (Ed.), *Assessment of children* (4th ed., pp. 576–609). San Diego: Jerome M. Sattler.
- Thorndike, E. L. (1949). *Selected writings from a connectionist's psychology*. New York: Appleton.
- Wechsler, D. (2009). *Wechsler individual achievement test third edition (WIAT III)*. San Antonio: Pearson.
- Wilkinson, G. S., & Robertson, G. J. (2006). *Wide range achievement test 4*. Lutz: Psychological Assessment Resources.
- Woodcock, R. N. (1997). *Woodcock reading mastery test – Revised/normative update*. Circle Pines: American Guidance Service.
- Woodcock, R. W., Mather, N., & McGrew, K. S. (2007). *Woodcock-Johnson III tests of cognitive abilities, normative update (NU) complete*. Rolling Meadows: Houghton Mifflin Harcourt, Riverside.

Achieving a Better Life Experience Savings Account (ABLE Savings Account, ABLE Account, 529A Savings Plan, and 529A Account)

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Definition

An Achieving a Better Life Experience account (ABLE account) is a tax-advantaged savings account for qualified individuals with disabilities (often referred to as “accontholders” or “designated beneficiaries”) (Pub. L. No. 113–295 [2014](#)). ABLE accounts allow beneficiaries to save funds without losing their eligibility for state and federal government benefits programs such as Medicare, Medicaid, Social Security, Supplemental Security Income (SSI), and Supplemental Security Disability Income (SSDI). ABLE account funds can be used for a broad range of health, wellness, and living expenses to support the beneficiary. ABLE accounts are sometimes referred to as “529A accounts” or “529A Savings

Plans” because the ABLE Act amends Section 529 of the US Internal Revenue Code.

History of ABLE Accounts

The Stephen Beck Jr. Achieving a Better Life Experience (ABLE) Act was signed into law on December 19, 2014. The US Congress has since amended the original ABLE Act by passing other legislation including the Consolidated Appropriations Act of 2016 and Tax Cuts and Jobs Act of 2017. The Consolidated Appropriations Act allowed qualified beneficiaries to enroll in any ABLE savings program in any state, not just their state of residence (Pub. L. No 114–113 [2016](#)). The Tax Cuts and Jobs Act made three key changes to ABLE account law by: (1) increasing the yearly contribution amounts allowed from accountholders who are employed (subject to IRS rules); (2) allowing a qualified ABLE accountholder to claim a Retirement Savings Contributions Credit (Saver’s Credit); and (3) allowing funds saved in a government-sponsored college tuition savings account (“529 account”) to roll into a ABLE account (Pub. L. No. 115–9 [2017](#)).

Principles of an ABLE Account

An ABLE account can be a useful special needs planning tool for individuals with autism spectrum disorder (ASD) and their caregivers (US Senate Committee on Finance [2014](#)). See Table 1, Four key benefits of an ABLE account. ABLE accounts were first created in the United States under the Stephen Beck, Jr. Achieving a Better Life Experience Act of 2014 (Pub. L. No. 113–295) and are governed by federal law, the laws of the individual states, and Internal Revenue Service (IRS) administrative rules. Under federal law, ABLE account savings are excluded from the accountholder’s income for the tax year (January 1 through December 31). Accountholders will face taxes and penalty fees if they deposit more than the federal gift tax limit into an ABLE account during a tax year (e.g., \$15,000 in 2020 and adjusted

Achieving a Better Life Experience Savings Account (ABLE Savings Account, ABLE Account, 529A Savings Plan, and 529A Account), Table 1 Four key benefits of an ABLE account

Item number	Summary	Benefit description
1	Tax-free savings	Savings in an ABLE account are not considered part of the accountholder’s income for the tax year (January 1 through December 31).
2	Protected eligibility for government benefits	Savings in an ABLE account cannot cause the accountholder to terminate their eligibility for state and federal government benefits programs.
3	Many possible uses for “qualified disability expenses”	ABLE account funds can be used for a broad range of costs that support the beneficiary. Qualified disability expenses include: education, housing, transportation, employment training/support, assistive technology/ services, health, healthcare services not covered by private insurance or government programs, wellness goods/services, financial planning, financial management, legal assistance, funeral/burial costs, and basic living expenses.
4	Tax deduction incentives (for employed accountholders and for account contributors in certain states)	Any person or organization can contribute to an ABLE account. Several state ABLE account programs offer tax deduction incentives to encourage friends, family, and others to contribute funds to ABLE accounts. Certain ABLE account contributions can qualify as tax deductions for ABLE accountholders who are employed.

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periodically by the IRS to account for inflation). Accountholders who are employed can contribute funds beyond the federal gift tax limit – these amounts depend on the accountholder’s income totals according to IRS rules (US IRS 2020b).

ABLE account savings are designed to supplement the benefits from private healthcare insurance and government healthcare programs. Importantly, ABLE accounts allow beneficiaries to save funds without losing their eligibility for state and federal government benefits programs from Medicare, Medicaid, and the Social Security Administration (SSA). If ABLE accountholders maintain a total account balance of no more than \$100,000, they can continue to receive care and services from their government means-tested benefits without paying additional out-of-pocket costs (US Social Security Administration 2020). ABLE account balances in excess of this threshold are considered “excess resources” and must be spent down before the accountholder can receive additional benefits that are paid for by the government. The ABLE Act specifies that an accountholder’s government benefits “shall

not be terminated, but shall be suspended” if there are excess resources in his or her ABLE account (Pub. L. No. 113–295 2014).

Under current laws, many individuals with serious disabilities do not qualify for ABLE accounts. To enroll in an ABLE account program in any state, individuals must have a qualified disability that occurred before age 26. Provided you satisfy the age requirement, you are automatically eligible to establish an ABLE account if you are a recipient of SSI and/or SSDI benefits. You also can become eligible to open an ABLE account if you can satisfy each of the following criteria: (a) the above-mentioned age requirement, (b) the SSA’s definition and criteria for a disability with significant functional limitations, and (c) receive a letter from a licensed physician that certifies your disability diagnosis.

Any person, for-profit business, or non-profit organization can make a deposit into an ABLE account. Several states offer tax deduction incentives to encourage friends, family, and crowdsourcing initiatives to fund ABLE accounts. Certain ABLE account contributions can qualify as

income tax deductions for ABLE accountholders who are employed (US Internal Revenue Service 2020a).

Each state allows online applications to open an ABLE account. Users can monitor their ABLE savings through their ABLE program's website and make contributions or withdrawals online. A beneficiary's parent, legal guardian, legal conservator, or Power of Attorney agent can assist in creating an ABLE account. Beneficiaries can set up their ABLE accounts directly if they are age 18 or older and have "legally capacity" (Garner 2019). For purposes of entering into any ABLE account contractual agreement, an accountholder's legal capacity is defined as the ability to understand the fundamental benefits, risks, and effect of entering into the agreement (Parker 2016). Individuals do not have legal capacity when a judge rules that they are entirely incapable of managing financial or personal affairs (National Council on Disability 2018). A person who lacks legal capacity is unable to fully safeguard himself or herself against harm to self, wealth, and/or property (see also Kohn et al. 2013 regarding the growing legal and medical field of "supported decision-making").

Qualified Disability Expenses for an ABLE Account

The funds in ABLE accounts can only be spent on expenses that support the accountholder (Morris et al. 2016). Under the guidelines set forth in the federal ABLE Act statute, these are called "qualified disability expenses" (QDE). Federal law defines several wide-ranging categories for QDE as follows:

[A]ny expenses related to the eligible individual's blindness or disability which are made for the benefit of an eligible individual who is the designated beneficiary [accountholder], including the following expenses: education, housing, transportation, employment training and support, assistive technology and personal support services, health, prevention and wellness, financial management and administrative services, legal fees, expenses for oversight and monitoring, funeral and burial expenses, and other expenses... consistent with the purposes of this section [and in support of the accountholder] (Pub. L. No. 113–295).

QDE for ABLE accounts are broadly defined and not tied to medical necessity. This is a stark departure from other federal healthcare spending requirements, such as Medicare's payments for healthcare costs that are "reasonable and medically necessary" for the diagnosis or treatment of illness or injury (Stein and Lipschutz 2019). As a general rule, Medicare and Medicaid spending does not include care that is still considered experimental, investigative, or unproven. In contrast, the only condition for spending on QDE with an ABLE account is that the expense loosely relate back to the accountholder's disability. Best practices for documenting QDE include keeping a Qualified Expenses Withdrawal Log (e.g., Iowa IABLE 2020).

Possible Challenges with an ABLE Account

When either considering or utilizing ABLE accounts, individuals should take care to weigh all potential challenges and benefits (Hershey et al. 2017a). See Table 2, Six key challenges of an ABLE Account. Users must take care to remain informed about each tax year's annual contribution limit (set by the federal gift tax limit and IRS rules) and their maximum account balance limit (which varies according to each state's law). Account contributions that are in excess of annual limits are subject to penalty taxes.

The total account balance limit for an ABLE account is set by individual state programs and modeled after state limits for college tuition savings accounts (also referred to as "529 accounts"). In most states, the total account limit for ABLE savings is \$300,000 or higher. Though individuals cannot deposit a total amount into their ABLE account over time that exceeds their account balance limit, it is impractical for most accountholders to save more than \$100,000 in their ABLE accounts – ABLE accountholders with savings in excess of \$100,000 receive a suspension of their government benefits. In sum, experts recommend that ABLE accountholders keep their balances at \$100,000 or lower at all times to avoid the out-of-pocket costs associated with suspended government benefits.

After an accountholder has passed away, most state governments will claim and collect all of the

Achieving a Better Life Experience Savings Account (ABLE Savings Account, ABLE Account, 529A Savings Plan, and 529A Account), Table 2 Six key challenges of an ABLE account

Item number	Summary	Challenge description
1	Maximum account balance limitations	The total account balance limit for an ABLE account is set by individual state programs and modeled after state limits for college tuition savings accounts (529 accounts). Many states have set the total account limit for ABLE savings at rates of \$300,000 or higher.
2	Annual contribution limitations	Deposits into an ABLE account cannot exceed the threshold of the federal gift tax limit during each tax year (e.g., \$15,000 in 2020). Account contributions that are in excess of federal limits are subject to a penalty tax.
3	Limited free assistance for account management	Most state governments do not provide comprehensive helplines to provide accountholders with meaningful guidance or individualized assistance. Nationwide, state ABLE programs encourage do-it-yourself ABLE account management online. At the same time, all state ABLE contracts strongly encourage ABLE accountholders to manage their finances with a private attorney, financial planner, and/or tax advisor.
4	Remaining funds claimed by the state government after the accountholder has passed away (in most states)	In most states, all funds that remain in an ABLE account after the accountholder's death will be claimed and collected by the state government to recoup expenses paid to the accountholder as government benefits during his or her lifetime.
5	Most beneficial when combined with other special needs planning tools	Finance experts recommend that, if possible, many accountholders should coordinate the spend-down of their ABLE accounts with a special needs trust (SNT) and/or Roth individual retirement account (IRA). As a best practice, accountholders should discuss their ABLE account plans with a special needs planning attorney to ensure all short- and long-term goals for the ABLE account align with the SNT and other legal instruments.
6	Potential for financial loss (depending on the accountholder's investment selections)	ABLE accountholders assume all investment risks as well as responsibility for any federal and state tax consequences related to the account. Depending on the accountholder's investment selections, it is possible to lose all or a portion of the savings in an ABLE account. Accounts with high investment risks can be impacted very negatively by market conditions. As a best practice, accountholders should consult with a special needs financial planner and/or tax advisor before selecting an ABLE account investment structure with high financial risks of loss.

funds that remain in an ABLE account. This process is often referred to as the government's "estate recovery." Under these laws, a state is allowed to recoup expenses that were paid in the form of government benefits to the accountholder during his or her lifetime. Most ABLE accountholders should consider keeping large savings amounts inside a special needs trust

(SNT), a legal instrument which is typically shielded from government collections after the beneficiary's death (Hershey et al. 2017b). Unlike most ABLE accounts, funds protected in an SNT can be transferred to another person or charity organization as part of a beneficiary's estate through a Last Will and Testament (Andersen and Gary 2018).

ABLE accounts are often most advantageous when combined with other special needs planning tools as part of a comprehensive long- and short-term budget plan (Rephan and Groshek 2016). Finance experts recommend that, if possible, many accountholders should coordinate the spend-down of the relatively low funds in their ABLE accounts with spending from higher savings amounts in their SNT (Abbey and Hershey 2016; see also Hershey and Kelly 2019 regarding using a Roth individual retirement account (IRA) as another possible source of funding for special needs planning in coordination with an ABLE account).

ABLE accountholders and their families should be aware that state governments do not provide comprehensive helplines to provide accountholders with meaningful guidance or individualized assistance. All state ABLE contracts strongly encourage ABLE accountholders to manage their finances with a private attorney, financial planner, and/or tax advisor. Hiring professional consultants can be helpful to protect ABLE account users against accidental financial losses – ABLE accountholders assume all financial investment risks related to their accounts.

Helpful Resources for State-by-State Comparisons of ABLE Programs

Qualified individuals can open an account in any state with an active ABLE account program and are not limited to their current state of residency. One of the greatest initial challenges in creating an ABLE account is determining which state ABLE program to utilize. Those who are interested in learning more about ABLE accounts can consult the online resources available from the National Disability Institute's ABLE National Resource Center (ANRC), a leading comprehensive source of independent information about each state's ABLE program. The ANRC website includes two information research functions: a "Search by ABLE Program Features" tool (ABLE National Resource Center 2020a) and a "View All State Programs" tool (ABLE National Resource Center 2020b). These webpages allow individuals to

compare state ABLE program features in several areas, including: debit card availability, estate recovery rules (state government collections of funds after the accountholder's death), and tax deduction options for in-state residents.

Key Considerations When Selecting an ABLE Account Program

There are six key issues to consider when deciding which ABLE program best serves an individual beneficiary's needs:

1. Many state ABLE programs include account fees for the following services: account maintenance, disbursement, account report printing, rollovers, and administration. Will the ABLE program fees in this state unduly burden or otherwise inconvenience the accountholder?
2. What is this state's total account balance limit for ABLE accounts? Moreover, do these limits align with the accountholder's short- and long-term budget goals?
3. Is the ABLE accountholder or those who contribute to an ABLE account eligible for an income tax deduction in this state?
4. Is the accountholder best served by a program that offers a debit or purchasing card to use ABLE account funds? If so, does this state ABLE program offer a card option?
5. State ABLE programs offer allow accountholders to choose between investment options with varying degrees of financial risk. Does this particular state ABLE account program offer the degree of investment risk that most benefits the accountholder?
6. How will the investment-related fees for this particular state ABLE account program impact the beneficiary? Does this state ABLE program have high investment fees that will significantly reduce the ABLE account balance? (Kelly and Hershey 2018).

In addition to the above considerations, accountholders and/or their families should review state ABLE program contracts in detail, ideally with the assistance of a qualified attorney

and/or financial planner. As part of the ABLE enrollment process, each state program asks applicants to perform a thorough review of voluminous contracts and forms. Generally, each state ABLE program requires accountholders – or their designated agents – to sign an account disclosure agreement, which sometimes runs in excess of 100 pages (e.g., Illinois Able 2020). Applicants should be aware that ABLE account program disclosure forms have various titles depending on the state of origin, including – but not limited to – member plan, program disclosure statement, and plan disclosure statement. Some states combine their ABLE disclosures with participation agreement contracts, while others maintain separate documents.

Hiring Legal and/or Financial Professionals as a Recommended Best Practice

Most ABLE savings account program agreements contain links to additional information regarding investment options, strategies, and potential risks of financial losses. Directly or indirectly, a majority of state ABLE programs encourage do-it-yourself account management, while at the same time, explicitly warning individuals against it with legal disclaimers. Consider, for example, the following broad disclaimer which is prominently placed within several state ABLE program websites and contract agreements:

Before investing in any ABLE program, you should consider whether your home state offers an ABLE program that provides its taxpayers with favorable state tax or other benefits that are only available through investment in the home state's ABLE program. You also should consult your financial, tax, or other adviser to learn more about how state-based benefits (or any limitations) would apply to your specific circumstances. You also may wish to directly contact your home state's ABLE program, or any other ABLE program, to learn more about those plans' features, benefits and limitations. Keep in mind that state-based benefits should be one of many appropriately weighted factors to be considered when making an investment decision (e.g. Alaska ABLE Plan: A Member of the National ABLE Alliance 2020)

After reviewing all contract agreements and associated links, it is a recommended best practice for individuals considering an ABLE account to seek personalized, professional guidance (McGee and Ferguson 2017). If feasible, discuss specific legal, investment, and tax situations in detail with a tax advisor, attorney, and/or other financial planner.

See Also

- Conservatorship (Full Conservatorship and Limited Conservatorship)
- Guardianship
- Power of Attorney (Financial and Property Management)
- Power of Attorney for Healthcare (Durable Healthcare Power of Attorney and Medical Power of Attorney)
- Special Needs Planning (SNP)

References and Reading

- Abbey, B., & Hershey, L. (2016). Does the ABLE Act enable the wealthy and disable the poor? *Journal of Financial Service Professionals*, 70(2), 46–52. Retrieved from https://www.emich.edu/cob/documents/2016_does_the_able_act_enable_the_wealthy_and_disable_the_poor.pdf
- ABLE National Resource Center. (2020a). Get started: Search by ABLE program features. <https://www.ablenrc.org/state-plan-search/>
- ABLE National Resource Center. (2020b). Get started: View all state programs. Retrieved from <https://www.ablenrc.org/select-a-state-program/>
- Alaska ABLE Plan: A member of the national ABLE alliance. (2020). Home. Retrieved from <https://savewithable.com/ak/home.html>
- Andersen, R., & Gary, S. (2018). *Understanding trusts and estates* (6th ed.). Durham: Carolina Academic Press.
- Stephen Beck, Jr. Achieving a Better Life Experience Act of 2014, Pub. L. No. 113–295, 128 Stat. 4010, codified at 26 U.S.C. § 529A. Retrieved from <https://www.congress.gov/bill/113th-congress/house-bill/5771/text/enr?r=123>
- Consolidated Appropriations Act of 2016, Pub. L. No. 114–113, codified as amended at 26 U.S.C. § 529A. Retrieved from <https://www.congress.gov/bill/114th-congress/house-bill/2029/text>
- Garner, B. (Ed.). (2019). *Incapacity. Black's Law Dictionary*. 11th ed. St. Paul: Thomson/West.

- Hershey, L., & Kelly, A. (2019). Using Roth conversions of legacy retirement plans to fund special needs planning. *Journal of Financial Service Professionals*, 73(2), 80–88. Retrieved from https://www.emich.edu/cob/documents/2019_hershey_kelly.pdf
- Hershey, L., Kelly, A., & Abbey, B. (2017a). Disabling ABLE: Five possible pitfalls when implementing the ABLE Act. *Journal of Financial Service Professionals*, 71(2), 70–78. Retrieved from https://www.emich.edu/cob/documents/2017_disabling_able.pdf
- Hershey, L., Kelly, A., & Abbey, B. (2017b). Enabling ABLE: Five potential positives for implementing the ABLE Act. *Journal of Financial Service Professionals*, 71(2), 60–68. Retrieved from https://www.emich.edu/cob/documents/2017_enabling_able.pdf
- Illinois Able. (2020). Plan disclosure documents. Retrieved from <https://cdn.unite529.com/jcdn/files/UABLE/pdfs/il-programdescription.pdf>
- Iowa IABLE. (2020). Qualified expenses withdrawal log. Retrieved from <https://www.iabile.gov/resources/qualified-expenses-withdrawal-log>
- Kelly, A., & Hershey, L. (2018). A 50 state review of ABLE Act 529A accounts. *Journal of Financial Service Professionals*, 72(2), 69–84. Retrieved from https://www.emich.edu/cob/documents/a_50_state_review_of_able_act_accounts.pdf
- Kohn, N., Blumenthal, J., & Campbell, A. (2013). Supported decision-making: A viable alternative to guardianship. *Penn State Law Review*, 117(4), 1111. [http://www.pennstatelawreview.org/117/4/%20Final/4-Kohn%20et%20al.%20\(final\)%20\(rev2\).pdf](http://www.pennstatelawreview.org/117/4/%20Final/4-Kohn%20et%20al.%20(final)%20(rev2).pdf)
- McGee, C., & Ferguson, G. (2017). A primer on ABLE accounts. *University of Richmond Law Review*, 52, 149–180. Retrieved from <http://lawreview.richmond.edu/files/2017/11/McGee-521A.pdf>
- Morris, M., Rodriguez, C., & Blanck, P. (2016). ABLE accounts: A down payment on freedom. *Inclusion*, 4(1), 21–29. <https://doi.org/10.1352/2326-6988-4.1.21>
- National Council on Disability. (2018). *Beyond guardianship: Toward alternatives that promote greater self-determination* (pp. 145–152). Retrieved from <https://ncd.gov/publications/2018/beyond-guardianship-toward-alternatives>
- Parker, M. (2016). Getting the balance right: Conceptual considerations concerning legal capacity and supported decision-making. *Journal of Bioethical Inquiry*, 13(3), 381–393. <https://doi.org/10.1007/s11673-016-9727-z>
- Rephan, D. A., & Groshek, J. (2016). ABLE Act accounts: Achieving a better life experience for individuals with disabilities with tax-preferred savings (and the old reliable special and supplemental needs trust). *Mitchell Hamline Law Review*, 42, 963. Retrieved from <https://open.mitchellhamline.edu/cgi/viewcontent.cgi?article=1034&context=mhlr>
- Stein, J., & Lipschutz, D. (2019). Surmounting barriers to Medicare-covered care: The Center for Medicare Advocacy offers advocacy suggestions for accessing medically necessary care via Medicare. *Generations*, 43, 4. Retrieved from <https://www.jstor.org/stable/26908290>
- Tax Cuts and Jobs Act of 2017, Pub. L. No. 115–97, codified as amended at 26 U.S.C. § 529A. Retrieved from <https://www.congress.gov/bill/115th-congress/house-bill/1/text>
- U.S. Internal Revenue Service. (2020a). ABLE accounts – Tax benefit for people with disabilities. Retrieved from <https://www.irs.gov/government-entities/federal-state-local-governments/able-accounts-tax-benefit-for-people-with-disabilities>
- U.S. Internal Revenue Service. (2020b). Instructions for forms 1099-QA and 5498-QA: Distributions from ABLE accounts and ABLE account contribution information. Retrieved from <https://www.irs.gov/instructions/i1099qa>
- U.S. Senate Committee on Finance. (2014, July 23). Saving for an uncertain future: How the ABLE Act can help people with disabilities and their families. Hearing No. 113–598. Retrieved from <https://www.finance.senate.gov/imo/media/doc/937721.pdf>
- U.S. Social Security Administration. (2020). Spotlight on achieving a better life experience (ABLE) accounts. Retrieved from <https://www.ssa.gov/ssi/spotlights/spot-able.html>

Achieving Academic Independence in Middle School-Outpatient (AIMS-O)

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Definition

The Achieving Academic Independence in Middle School-Outpatient (AIMS-O) intervention involves teaching academic executive functioning (EF) skills using behavioral management (e.g., reinforcement, behavioral contract, first-then language, etc.) principles to promote increased independence related to academics. Sessions follow a consistent routine of (1) real-world practice review (discussion of previously taught skill and how it was used at home); (2) teaching component (PowerPoint, video clips, hands-on activities, etc.) that focuses on teens and parents learning key

academic EF skills (e.g., creating a homework system, study cards); and (3) in-session practice of the newly taught concepts/strategies (e.g., parents and teens work together to create a behavioral contract/agreement, study cards, etc.) with coaching from the psychologist. Teens are assigned real-world practice tasks each session that are designed to lead to further mastery of newly taught skills through practice at home with support (as needed) from their parents between sessions. Currently, the seven 90-minute AIMS-O sessions include (1) education related to EF, (2) problem solving, (3) behavioral contracting related to use of academic EF skills, (4) organization and time management skills, (5 and 6) study skills including study cards, memory strategies such as acrostics and acronyms, summarizing skills, and graphic organizers, and (7) planning for use of skills in different settings (e.g., school), school collaboration, and teen initiative. AIMS-O is taught by a clinical psychologist and co-facilitator (e.g., clinical psychology graduate student) and is attended by parents and teens. Parents play a critical role as a coach for their teens to support the acquisition and mastery of key academic EF skills at home. AIMS-O is intended for middle school teens with Autism Spectrum Disorders (ASD) without intellectual disability (ID) who are in the general education setting.

Historical Background

Youth with autism spectrum disorders (ASD) frequently experience significant academic problems in a variety of domains (Whitby and Mancil 2009). Although higher cognitive abilities are associated with better academic performance, youth who have ASD without an intellectual disability (i.e., average IQ) may still struggle academically (Keen et al. 2016). Specific academic challenges in high functioning ASD may include writing (e.g., organizing content), reading comprehension (e.g., understanding how individual details contribute to a greater lesson, taking the perspectives of others), and math problem solving (Keen et al. 2016). While some of these academic

struggles stem from key features of ASD (e.g., social-communication deficits, narrowly defined interests, and concrete/literal thinking), they are also strongly linked to deficits in executive functioning (EF) such as organization, time management, prioritization, and initiation (Pennington and Ozonoff 1996).

EF skills are critical for academic success. Students must be able to initiate tasks, perform multistep sequences of events, reflect, reason, plan, and prioritize (e.g., complete different tasks for several subjects on time), sustain performance and complete tasks, be flexible in their thinking (e.g., select the learning strategy appropriate for each context), and monitor their performance (e.g., manage progress and check for mistakes; (Best et al. 2009, 2011; Endedijk et al. 2011; Fisher and Happe 2005). However, 35–70% of teens with ASD without an intellectual disability present with EF deficits (Blijd-Hoogewys et al. 2014; Pennington and Ozonoff 1996) including deficits in planning, flexibility, shifting set, metacognition (awareness of own thought processes), and monitoring their own behavior (Hill 2004). Common challenges include difficulties getting started on tasks, managing distractions, planning for studying, multitasking, keeping materials organized, and prioritizing tasks. Parents of teens with ASD also report difficulties getting their child to start school work independently (Endedijk et al. 2011; Hampshire et al. 2014). As a result, teens with ASD and their parents may struggle to acquire and manage critical academic behaviors (e.g., material organization, tracking assignments, homework completion, effectively studying, and breaking down large assignments) and experience significant homework issues (e.g., misunderstanding assignments) that are associated with EF deficits.

Persistent EF deficits are clear predictors of poor academic performance (Best et al. 2009, 2011; Fisher and Happe 2005) and poor outcomes in ASD (Clark et al. 2010). There is a clear need for interventions targeting academic EF skills, including planning, organization, time management, and study skills, that lead to more successful outcomes in ASD. Yet, according to the National

Research Council and National Autism Center, there are currently no evidence-based interventions targeting academic EF skills for teens with ASD despite similar interventions already existing for children with similar EF deficits (e.g., attention-deficit/hyperactivity disorder or ADHD). More specifically, to date, there are no randomized clinical trials demonstrating the efficacy of EF interventions for middle school teens with ASD.

The majority of research with children with ASD has focused on the younger ages, with less attention to the transition issues and challenges they face as adolescents (Wong et al. 2015). This is problematic since the inclusion of students with disabilities into general education classrooms is a substantiated transition best practice (Schall et al. 2012). In fact, general education placement of students with ASD has increased at a rate faster than all other disability categories (Whitby 2013). Further, as rates of inclusion for middle school students with high functioning ASD increase, teachers and para-educators do not typically have the support and training to implement evidence-based treatment to meet their needs (Kurth and Mastergeorge 2010). Specifically, most individualized education programs (IEPs) for students with high functioning ASD enrolled in inclusive education settings are more likely to focus on academic progress, but little research has been conducted on how to develop environments and utilize strategies and supports to facilitate academic success (Kurth and Mastergeorge 2010). Given the prevalence of EF deficits coexisting through the transition to middle school, it is unsurprising that students with ASD have significant academic problems in middle school (Adreon and Stella 2001; Mullins and Irvin 2000). In fact, during middle school years, the academic performance of teens with high functioning ASD is approximately 2–3 years below their typical peers (Wagner et al. 2003).

Current Knowledge

AIMS-O is currently still in development. Focus groups with parents, teens with ASD, teachers,

and school administrators confirm that an intervention targeting academic EFs is needed for middle school teens with ASD without an ID. Themes that emerged from parent and teen groups (Tamm et al. 2019) include:

1. *Executive Functioning Is a Barrier to Academic Success*: Both parents and teens agreed that the teens had difficulty with remembering to either do assigned homework or turn in completed homework, which then leads to missing assignments and negatively affects grades. Organization was another prominent challenge noted by both teens and parents. Parents also reported that they provide varying levels of support to help their teen stay organized. Most teens required verbal reminders from teachers to complete assignments. Teens and parents reported that difficulties paying attention also affect school performance. Teens related that they have difficulty listening to others, which gets them in trouble at school and at home. Parents agreed that their teens have difficulty listening and focusing, and they must monitor their teens to ensure tasks are completed. When tasks require sustained attention (e.g., listen to a class lecture), teens often become overwhelmed or frustrated and “shut down,” which then leads to poor performance. Parents noted that teens had difficulties with multitasking. If teens were expected to both pay attention and take notes, only one task would be completed. Additionally, teens reported difficulties at school and home related to a lack of inhibition and self-control (e.g., blurting inappropriate things out in class, working on homework when they should be listening to the teacher). Parents reported that teens respond best to established routines such as a specific time to complete homework.
2. *Expectations for Independence and Socialization Make Middle School Particularly Challenging*: Parents reported that middle school is particularly challenging for their teens. As expectations for independence in the school environment increase, teens with ASD struggle due to their difficulties with organization and planning ahead. Teens’ difficulties with

abstract thinking also interfere with academic success in middle school. Once concepts become more obscure and abstract, teens struggle and take a longer time to complete assignments, which may lead to them avoiding similar tasks in the future. Further, limited abstract thinking causes tension between parents and teens. Unlike other teens, parents reported that teens with ASD often fail to understand that grades are important for future achievements. This inability to connect academic performance with future life aspirations may make teens less apt to excel in school.

3. *Teens with ASD Experience Individual Academic Challenges:* Each teen and parent reported that their teen had different academic challenges. While one teen might have difficulties with English, another would not have problems with English but would report problems with math. Across the groups, teens had difficulties with language arts, math, social studies, science, English, and spelling. Similarly, teens and/or their parents reported using a variety of study strategies including web study, teacher resources, repetitive fact review, reading over textbooks and notes, creating flash cards, and online study programs and apps such as Quizlet.
4. *High Need for Parent Involvement:* More than 80% of parents reported helping teens pack their backpacks and stay organized, as well as helping with homework including determining assignments, helping with completion, and closely monitoring homework. More than 60% reported helping with test studying. All teens and parents reported the use of external rewards to motivate teens to complete work and study for tests. Although formal behavioral contracts were not common, parents and teens reported use of verbal contracts that required teens to perform academic tasks prior to receiving some reward.
5. *Challenges with Academic Accommodations:* A major theme for parents was that their teens were not receiving proper academic accommodations in middle school. Parents felt teens were either given too many, too few, or inappropriate accommodations. The majority of

parents also expressed dissatisfaction with the IEP team at their teen's school stating that if their teen had high grades, then s/he would often not receive assistance with EF deficits (e.g., organization, planning, and prioritizing). Parents reported high levels of frustration as the IEP team would often ignore parents' concerns and sometimes refuse to meet with them. As a result, some parents changed their teen's school in order to receive additional academic supports.

6. *Critical Role for Teachers:* Parents and teens reported that the level and quality of teacher support had a significant impact on these teens' academic success. Teens desired additional help and resources from teachers including homework reminders, study tools, and more in-depth explanations of some assignments. Parents reported that teens had more difficulties when teachers had negative attitudes. These attitudes may have developed because not all teachers are equipped to manage teens with ASD, may not understand how EF deficits affect academic performance, and may benefit from learning to promote the skills taught in the group intervention (e.g., organization, problem solving, and study cards). In contrast, parents noted that when teachers were calm, patient, creative, and persistent, teens were more successful. Overall, parents reported a lack of consistent communication with teachers. They expressed disappointment that teachers sometimes ignored their advice about responding to their teen's behavior. Parents felt that open communication among all involved in their child's education (teachers, parents, aides, IEP teams) was necessary for success in middle school. A blend of in-person and electronic communication strategies was preferred.

A small "proof of concept" trial showed that AIMS-O is possible to deliver, results in meaningful improvements, and is acceptable to parents and teens with ASD (Tamm et al. 2019). For example, attendance was excellent across the seven sessions (100%). Parents rated clinically significant improvements for the teen on the

Children's Organizational Skills Scale (Abikoff and Gallagher 2009) and Homework Problem Checklist (Anesko et al. 1987). On consumer satisfaction ratings, parents gave high ratings for the effectiveness of the group, the content of each session, the effectiveness of the PowerPoint slides, worksheets, visuals, and instructors. However, parents gave lower ratings for how well they thought their teen had understood the content. Teens themselves had generally low ratings for how well they understood their challenges with attention and EF, how to use the study strategies, how to use study cards, how to summarize, how to use time management strategies, and how to create an effective homework system and routine. Given that both teens and parents reported difficulties with teen comprehension of content, additional adaptation of AIMS-O is warranted.

Future Directions

With funding support from the National Institutes of Health, work is ongoing to further adapt AIMS-O for use in the outpatient clinical setting. Based on feedback from expert consultants and key stakeholders, the AIMS-O content and teaching approach is being modified and adapted to address core deficits in ASD. Examples of strategies that will be used to enhance the accessibility of concepts include: use of visual supports (e.g., using graphic organizers as a method of summarizing content), a self-assessment to assist in understanding EF strengths and deficits, use of technology (e.g., videos to teach core components, use of apps for studying and summarizing), worksheets combined with multiple choice lists for new concepts, multiple opportunities for repetition and practice, and hands-on activities to increase engagement while building skills. Also, as schools are increasingly turning to technology (e.g., Blackboard, Power School, Google Classroom, DropBox), strategies for organizing electronic content will be critical. Given the themes identified by parents and teens related to school, an adaptation of AIMS-O for administration in the school setting is warranted. Such work is also underway supported by funding from the Institute of Education Sciences.

See Also

- Academic Skills
- Family-Centered Care, Second Edition
- Functional Life Skills
- High-Functioning Autism (HFA)
- Qualitative Versus Quantitative Approaches

References and Reading

- Abikoff, H., & Gallagher, R. (2009). *The children's organizational skills scales technical manual*. North Tonawanda: Multihealth Systems Inc..
- Adreon, D., & Stella, J. (2001). Transition to middle and high school: Increasing the success of students with Asperger Syndrome. *Intervention in School and Clinic*, 36(5), 268–271.
- Anesko, K. M., Schoiack, G., Ramirez, R., & Levine, F. M. (1987). The homework problem checklist: Assessing children's homework problems. *Behavioral Assessment*, 9, 179–185.
- Best, J. R., Miller, P. H., & Jones, L. L. (2009). Executive functions after age 5: Changes and correlates. *Developmental Review*, 29(3), 180–200. <https://doi.org/10.1016/j.dr.2009.05.002>.
- Best, J. R., Miller, P. H., & Naglieri, J. A. (2011). Relations between executive function and academic achievement from ages 5 to 17 in a large, representative national sample. *Learning and Individual Differences*, 21(4), 327–336. <https://doi.org/10.1016/j.lindif.2011.01.007>.
- Blijd-Hoogewys, E. M., Bezemer, M. L., & van Geert, P. L. (2014). Executive functioning in children with ASD: An analysis of the BRIEF. *Journal of Autism and Developmental Disorders*, 44(12), 3089–3100. <https://doi.org/10.1007/s10803-014-2176-9>.
- Clark, C. A., Pritchard, V. E., & Woodward, L. J. (2010). Preschool executive functioning abilities predict early mathematics achievement. *Developmental Psychology*, 46(5), 1176–1191. <https://doi.org/10.1037/a0019672>.
- Endedijk, H., Denessen, E., & Hendriks, A. W. (2011). Relationships between executive functioning and homework difficulties in students with and without autism spectrum disorder: An analysis of student- and parent-reports. *Learning and Individual Differences*, 21, 765–770.
- Fisher, N., & Happe, F. (2005). A training study of theory of mind and executive function in children with autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 35(6), 757–771. <https://doi.org/10.1007/s10803-005-0022-9>.
- Hampshire, P. K., Butera, G. D., & Dustin, T. J. (2014). Promoting homework independence for students with autism spectrum disorders. *Intervention in School and Clinic*, 49(5), 290–297. <https://doi.org/10.1177/1053451213513955>.
- Hill, E. L. (2004). Executive dysfunction in autism. *Trends in Cognitive Sciences*, 8(1), 26–32.

- Keen, D., Webster, A., & Ridley, G. (2016). How well are children with autism spectrum disorder doing academically at school? *Autism*, 20(3), 276–294.
- Kurth, J. A., & Mastergeorge, A. M. (2010). Individual education plan goals and services for adolescents with autism: Impact of age and educational setting. *The Journal of Special Education*, 44(3), 146–160.
- Mullins, E. R., & Irvin, J. L. (2000). Transition into middle school: What research says. *Middle School Journal*, 31(3), 57–60.
- Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 37(1), 51–87.
- Schall, C., Wehman, P., & McDonough, J. L. (2012). Transition from school to work for students with autism spectrum disorders: Understanding the process and achieving better outcomes. *Pediatric Clinics of North America*, 59(1), 189–202., xii. <https://doi.org/10.1016/j.pcl.2011.10.009>.
- Tamm, L., Duncan, A., Vaughn, A., McDade, R., Estell, N., Birnschein, A., & Crosby, L. (2019). Academic needs in middle school: Perspectives of parents and youth with autism. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03995-1>.
- Wagner, M., Marder, C., Blackorby, J., Cameto, R., Newman, L., Levine, P., & Davies-Mercier, E. (2003). *The achievements of youth with disabilities during secondary school: A report from the National Longitudinal Transition Study-2*. Retrieved from Menlo Park:
- Whitby, P. J. (2013). The Effects of “Solve It!” on the Mathematical Word Problem Solving Ability of Adolescents with Autism Spectrum Disorders. *Focus on Autism and Other Developmental Disabilities*, 28(2), 78–88.
- Whitby, P. J. S., & Mancil, G. R. (2009). Academic achievement profiles of children with high functioning autism and asperger syndrome: A review of the literature. *Education and Training in Developmental Disabilities*, 44(4), 551–560.
- Wong, C., Odom, S. L., Hume, K. A., Cox, A. W., Fettig, A., Kucharczyk, S., . . . & Schultz, T. R. (2015). Evidence-based practices for children, youth, and young adults with autism spectrum disorder: A comprehensive review. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-014-2351-z>.

Acquired Autism

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Synonyms

Autistic regression; Disintegrative disorder; Language/autistic regression; Regressive autism

Definition

Autism (autism spectrum disorders – ASD) typically denotes a static, behaviorally defined, developmental disorder of the immature brain, with identifiable etiologies rare and biologically treatable causes rarer still. Acquired autism implies newly acquired/progressive brain dysfunction, with multiple, mostly undefined, potential causes, presumably affecting similar brain circuitry as developmental ASD. Acquired autism requires prompt neurologic investigation and, in some cases, brain imaging, electrophysiologic, genetic, or other tests to detect potentially medically treatable causes or progressive disease.

Subtypes of acquired autism (discussed in more detailed entries in the encyclopedia):

1. *Language/autistic regression* – Reported by 20–35% of parents, usually between 15 and 30 months. Its causes are unknown because language regression/plateau is rarely studied while in process, especially when its insidious onset is glossed over. It occasionally follows a nonspecific illness or emotional stress. Epilepsy only exceptionally plays a causative role. Regression rarely overlaps acquired epileptic aphasia (Landau-Kleffner syndrome) of preschoolers who all have seizures or epileptiform EEGs, but not autism.
2. *Childhood disintegrative disorder* – Exceptionally rare language/autistic/intellectual but not motor regression of all functions between ages 2 and 10 years following entirely normal earlier development. Its causes are unknown, its prognosis poor, without known medical treatment. It requires thorough neurologic investigation.
3. *Rett syndrome* – Generalized developmental regression in girls, mostly between 6 and 18 months when they cease progressing, head growth stagnates, irritability, hand stereotypies, and a variety of other systemic and neurologic symptoms appear. Severity varies, prognosis is poor. Most are due to mutations of the *MECP2* gene.
4. *Malignant epilepsies of early life* – Infantile spasms with a hypsarrhythmic EEG (West syndrome) in infancy and Lennox-Gastaut syndrome in toddlers with drop and other seizure

types and slow spike waves in the EEG are the most prevalent harbingers of acquired autism with cognitive impairment. They and others have a variety of genetic and acquired etiologies. Prognosis is guarded, but some are medically treatable; so prompt diagnosis is key.

5. *Cerebellar surgery* – Transitory (usually weeks) postoperative mutism with autistic features following removal of midline cerebellar tumors, mostly medulloblastomas.
6. *Psychoses, drug intoxication* – Catatonia may overlap with acquired autism and needs to be diagnosed because treatable. Psychotic depression, mania, and drug intoxication must be considered in unexplained acquired social withdrawal and loss of language and functional skills. Immunizations are not credible causes of autism.
7. *Encephalopathies* – Rarely, acute or chronic infectious, immune, metabolic, or toxic encephalopathies that involve limbic circuitry may result in an acquired autistic state. Diagnosing the causes of encephalopathies is critical because some are treatable, e.g., Hashimoto encephalitis, NMDA receptor limbic encephalitis, herpes simplex, or other infectious encephalitis.

References and Reading

- Dhossche, D. (1998). Brief report: Catatonia in autistic disorders. *Journal of Autism and Developmental Disorders*, 28, 329–331.
- Homan, K. J., Mellon, M. W., Houlihan, D., & Katusic, M. Z. (2011). Brief report: Childhood disintegrative disorder: A brief examination of eight case studies. *Journal of Autism and Developmental Disorders*, 41, 497–504.
- Offit, P. A. (2009). *Autism's false prophets: Bad science, risky medicine, and the search for a cure*. New York: Columbia University Press.
- Riva, D., & Giorgi, C. (2000). The cerebellum contributes to higher functions during development: Evidence from a series of children surgically treated for posterior fossa tumours. *Brain*, 123(Pt 5), 1051–1061.
- Tuchman, R., Cuccaro, M., & Alessandri, M. (2010). Autism and epilepsy: Historical perspective. *Brain & Development*, 32, 709–718.

Acquired Dysgraphia

- [Agraphia](#)

Action Level Imitation

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Definition

An individual copies, or mimics (Lopes and Santos-Victor 2004), the actions of a model (Nehaniv and Dautenhahn 1998). It is considered a lower form of imitation because it is not necessary for the imitator to process the meaning of the actions. Action level imitations can range from single actions (e.g., sticking out tongue, tapping on a table, making a bunny hop) to a string of actions. The imitation is considered successful when the behavior or set of behaviors is repeated exactly as presented by the model (Nehaniv and Dautenhahn 1998). This kind of imitation is seen in human newborns as well as in nonhuman primates such as chimpanzees and apes (Byrne and Russon 1998).

See Also

- [Action on Objects](#)
- [Imitation](#)

References and Reading

- Byrne, R., & Russon, A. (1998). Learning by imitation: A hierarchical approach. *Behavioral and Brain Sciences*, 21(5), 667–721.
- Lopes, M., & Santos-Victor, J. (2004). Visual learning by imitation with motor representations. *IEEE Transactions on Systems, Man and Cybernetics, Part B, Cybernetics, Special Issue on Learning in Computer Vision and Pattern Recognition*, 35(3). Retrieved February 13, 2012, from http://ieeexplore.ieee.org/xpls/abs_all.jsp?arnumber=1430829.
- Nehaniv, C., & Dautenhahn, K. (1998). Mapping between dissimilar bodies: Affordances and the algebraic foundations of imitation. In *Proceedings of the seventh European workshop on learning robots*, Edinburgh.

Action on Objects

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Definition

Movement of an object by another object or person. Action on object imitation trials are often used when studying imitation in children and other nonhuman primates (Tomasello et al. 1993). Some research has shown that adult humans use action on objects to stimulate and engage infants in play.

See Also

► [Action Level Imitation](#)

References and Reading

- Bard, K., & Vauclair, J. (1984). The communicative context of object manipulation in Ape and Human adult-infant Pairs. *Journal of Human Evolution*, 13(2), 181–190.
- Tomasello, M., Savage-Rumbaugh, S., & Kruger, A. (1993). Imitative learning of actions on objects by children, Chimpanzees, and enculturated Chimpanzees. *Child Development*, 64(6), 1688–1705.

Action Prediction in Autism

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Definition

Action prediction is the inherent social cognitive ability to anticipate how another individual's action will unfold over time. Such projections are essential for smooth reciprocal social

interaction and involve the predictions of others' action goals as well as the means they use to achieve their goals. In everyday life, humans constantly coordinate their actions with others. For example, having a cup of coffee at a café involves numerous joint actions, such as ordering the coffee when the waiter is attending, giving the cash and receiving the change, or holding up the cup so that the waiter can refill it with more coffee from the coffeepot. All these actions have to be sensitively attuned in order to successfully enjoy the cup of coffee without dropping money or spilling hot coffee on one's pants.

Eye movements during the observation of another individual's action reveal that, instead of passively following the movement trajectory of this action, humans proactively anticipate the end state as well as the trajectory of an ongoing action (Gredebäck and Falck-Ytter 2015). These gaze patterns are highly similar to eye movements during the performance of one's own actions (Flanagan and Johansson 2003). Developmental psychological research showed that infants in their first year of life already produce such anticipatory eye movements when they observe another's action (Falck-Ytter and von Hofsten 2006).

A substantial body of research suggests that action processing is altered in autism spectrum disorders (ASDs) and that this affects social interaction and communication abilities in individuals with ASD. Recently, it was proposed that impaired action prediction is a key factor that determines social interaction and communication skills in ASD. Yet, theoretical positions and empirical evidence remain controversial. This entry presents current knowledge on if and how action prediction is altered in ASD. Hence, the entry puts emphasis on more recent work on visual action anticipation (often assessed by eye tracking technology) and mentions older work on verbal action prediction only in passing.

Historical Background

Research on action prediction in ASD was mainly framed by two theoretical backgrounds. First, the *theory of mind deficit hypothesis* suggested that

individuals with ASD have difficulties with the prediction of other's actions, because they are impaired in attributing mental states, such as beliefs or desires, to others and themselves (see Frith 2012). This hypothesis is based on the premise that action predictions require the prior representation of another's goal (e.g., the guest at the neighboring table also *wants* a coffee refill) and belief about that goal (e.g., the guest *thinks* that drawing the waiters attention to herself will get her to that goal; cf., Dennett 1989). Thus, if individuals with ASD have difficulties in attributing mental states such as goals and beliefs, they should also be impaired in predicting the corresponding action (e.g., the guest will lift her empty cup of coffee when the waiter passes).

Second, the *broken mirror hypothesis* suggested that the mirror neuron system is dysfunctional in ASD. The mirror neuron system is a network of brain regions that presumably matches observed actions with one's own motor system and thereby enables action imitation and interpretation (Gallese et al. 2004). It was proposed that an ontogenetically early deficit in this neuronal mechanism of mapping another's and one's own actions in ASD leads to impaired imitation, understanding, and prediction of other's actions (Oberman and Ramachandran 2007).

However, mounting evidence is incompatible with both hypotheses. Children and adults with ASD show typical imitation, understanding, and prediction of goal-directed actions in a variety of paradigms (e.g., Cusack et al. 2015; Falck-Ytter 2010; Marsh et al. 2015; Sebanz et al. 2005). On the other hand, studies using different paradigms suggest that in some situations individuals with ASD do have difficulties with accurate action predictions (e.g., von Hofsten et al. 2009; Vivanti et al. 2011). Together, these findings challenge the theory of mind deficit hypothesis and the broken mirror hypothesis because these frameworks cannot fully explain the nuanced characteristics of action prediction in ASD. Consequently, more refined theories that account for impaired and intact aspects of action prediction in ASD are required (Hamilton 2009).

Current Knowledge

Recent years have seen a surge of empirical work on various levels of action processing in ASD, ranging from the detection of biological motion to belief-based action prediction. These findings have in turn driven rapid advance in the development of new theories.

In contrast to previous research (e.g., Blake et al. 2003; Klin and Jones 2008), several recent studies found no group difference between children, adolescents, and adults with ASD and typical participants in cognitive processes that are presumably required for successful action prediction (Cusack et al. 2015; Murphy et al. 2009; Saygin et al. 2010). For example, Cusack et al. (2015) reported intact detection of animacy, detection of biological motion, and discrimination of different types of actions in adolescents with ASD. Thus, deficient precursor mechanisms cannot serve as explanation for altered action prediction in ASD.

Moreover, the prediction of goals and means of a simple "pick-and-place" action was found to be unaffected in 5-year-old children with ASD (Falck-Ytter 2010). The children watched an agent reaching for objects on a table and placing it into a container at the other side of the table. Just as children from the comparison group, 5-year-olds with ASD visually anticipated the end state of these actions, i.e., grasping the corresponding ball and placing it in the container. In contrast, eye movements of children with and without ASD were reactive when the objects were moving self-propelled, i.e., their gaze followed the movement. This suggests that, when observing other agents, children with ASD proactively process the agent's goal and predict according actions.

Yet, action prediction in ASD seems to reach its limits when it becomes necessary to consider another's false beliefs about a certain situation. This was shown in explicit theory of mind tasks testing verbal action predictions (Baron-Cohen et al. 1985), and more recently using implicit theory of mind tasks, eye tracking versions of the classical explicit paradigms. In these implicit tasks, participants are familiarized with an agent's goal to get an object by opening one of two doors. Individuals with and without ASD need only a few

trials to generate predictive eye movements towards the door that is about to be opened. In the subsequent test trial, the agent falsely believes the object would be located behind door A, although it actually is either behind door B or it was removed completely from the scene. To accurately predict the agent's action in this trial (that she will open door A), participants have to take into account that the agent's upcoming action is based on her false belief that the object would still be behind this door. Children and adults with ASD systematically fail to correctly predict this false belief-based action (Schuwerk et al. 2015; Senju et al. 2010). However, it is important to note that, unlike previously thought, individuals with ASD are able to correctly predict the agent's false belief-based action in a variety of explicit theory of mind tasks (Scheeren et al. 2013). One explanation for this finding is that individuals with ASD, especially those with good intellectual and language abilities, develop compensatory strategies to pass these tasks (cf., Livingstone and Happé 2017).

In sum, current mixed and partially inconclusive evidence on intact and impaired aspects of action prediction in ASD speaks against the ideas of a general action prediction deficit in ASD and that single links in the chain of action processing are broken. Individuals with ASD are in principle able to predict other's actions. Rather, the finding that this ability is hampered in some contexts but not in others suggests that computations associated with successful action prediction work less effectively, and/or alternative cognitive routes are taken to get to an accurate action prediction (Livingstone and Happé 2017).

An appealing way to explain these altered cognitive processes that affect action prediction in ASD is offered by the theoretical framework of *hierarchical predictive processing*. In short, this currently prominent theory holds that we do not perceive the world by the unbiased interpretation of the information conveyed by sensory systems. Rather, we have a model of how the world should look like and our brain uses it to actively and optimally predict incoming sensory information. The flow of information is bidirectional: at each level within the postulated cognitive hierarchy, downward driving predictions and upward

transmitted sensory information are compared. The part of sensory input that cannot be explained by the prediction results in a prediction error, which is passed upward so that adjusted and more accurate predictions can be generated. This theory, describing a cognitively very efficient way of making sense of our world, is able to explain cognitive information processing in a variety of domains, ranging from vision to social cognition (Clark 2013).

Related claims have been made in developmental psychology. Ruffman (2014) suggested a reduced ability for learning from statistical information in ASD. Given that action anticipation and social learning might rely on implicit statistical learning (Paulus 2014; Ruffman 2014), such a deficit could account for a variety of problems associated with ASD.

Pellicano and Burr (2012) suggested that these predictions of sensory information are attenuated in ASD. Thus, perception is less biased by prior expectations about sensory information. Assuming that strong expectations help the cognitive system to reduce the complexity of sensory input, attenuated expectations result in an overburdening stream of relatively unfiltered incoming information that has to be processed. This fits well with the clinical observation of sensory sensitivities and repetitive behavior patterns. Within this framework, the latter can be viewed as a way to reduce the need to process unpredictable external information by creating expectable and consistent sensory stimulation.

In the case of action processing, this means that individuals with ASD are affected in the ability to generate action predictions based on prior experience and current observations. This presumably not only affects the control of one's own actions but also the prediction of other people's actions (Sinha et al. 2014). Indeed, individuals with ASD show deficits in motor coordination like action preparation or action planning (Fournier et al. 2010). And also when watching interactions of others, children and adults with ASD are less likely to predict their actions (Chambon et al. 2017; von der Lühé et al. 2016; von Hofsten et al. 2009). This form of interpersonal action prediction is crucial for smooth interactive turn-taking.

Moreover, there is evidence that even very young children with ASD show less anticipation of other's actions. For example, Brisson et al. (2012) retrospectively analyzed home videos of spoon-feeding situations of children around 5 months of age who have been diagnosed with ASD later. In contrast to a control group, they showed less anticipatory mouth opening when the caregiver moved the spoon towards the infant's mouth. Interestingly, typically developing infants who displayed low-anticipation rates, improved rapidly. Although an increase in accurate anticipations was also observed in infants later diagnosed with ASD, they seemed to learn more slowly from experience in such feeding situations.

Also later in life, the ability to exploit past experience to generate action predictions seems to be affected in ASD. For example, adults and 10-year-old children with ASD showed altered action predictions in a task that elicited visual action anticipations of an agent who repeatedly produced one of two possible actions to get to its goal (Schuwerk et al. 2016). The participants with ASD not only showed overall lower rates of action predictions, they also profited less than the respective comparison groups from frequency information. Adults and children from the comparison groups rapidly used the observation that the agent repeatedly acted the same way to predict that, in the same situation, it will produce the same action again. However, participants with ASD showed less improvement in accurate action predictions over time, suggesting that this form of statistical learning is affected in ASD.

In sum, there is growing evidence that the ability to effectively build expectations of another's upcoming actions based on previously observed actions under the same situational constraints is impaired in ASD. This might not affect the anticipation of simple actions with only one plausible outcome (Falck-Ytter 2010), or actions that follow certain rules (e.g., building a tower with colored pieces following the rule "alternate colors"; Vivanti et al. 2011). But, when options for an action become more complex or additional social cues or beliefs are involved, accurate action prediction might become challenging (Senju et al. 2010; Vivanti et al. 2011). It is important to note

that the ability to learn from experience to generate accurate action predictions is not absent in ASD (Chambon et al. 2017; Schuwerk et al. 2015). Yet, it seems that this form of learning from experience works less efficiently in individuals with ASD.

Future Directions

Impaired hierarchical predictive processing is a promising theoretical account, which is able to elucidate a variety of empirical findings on altered action prediction ASD. However, more evidence is needed to firmly conclude that deficient predictive processing is at the core of observed social interaction deficits in ASD. Moreover, it is unclear whether a predictive processing deficit in action prediction sufficiently explains the entire range of symptoms of impaired social interaction and communication. In other words, is navigating the social world challenging for individuals with ASD merely because it is so unpredictable, or do other factors, for example, the motivation to engage with the social world, also play a role (Chevallier et al. 2012)? Further, if it were the case that decelerated learning from experience with past actions underlies altered action processing in ASD, this could be targeted by interventions that, for example, offer additional opportunities to learn from experience, or help to elaborate more explicit and rule-based strategies to predict other's actions. It is up to future research to investigate if these are viable routes to modulate altered action prediction in ASD.

See Also

- [Mirror Neuron System](#)
- [Social Cognition](#)
- [Theory of Mind](#)

References and Reading

- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a "theory of mind"? *Cognition*, 21(1), 37–46.
- Blake, R., Turner, L. M., Smoski, M. J., Pozdol, S. L., & Stone, W. L. (2003). Visual recognition of biological

- motion is impaired in children with autism. *Psychological Science*, 14(2), 151–157.
- Brisson, J., Warreyn, P., Serres, J., Fossier, S., & Adrien-Louis, J. (2012). Motor anticipation failure in infants with autism: a retrospective analysis of feeding situations. *Autism*, 16(4), 420–429.
- Chambon, V., Farrer, C., Pacherie, E., Jacquet, P. O., Leboyer, M., & Zalla, T. (2017). Reduced sensitivity to social priors during action prediction in adults with autism spectrum disorders. *Cognition*, 160, 17–26.
- Chevallier, C., Kohls, G., Troiani, V., Brodtkin, E. S., & Schultz, R. T. (2012). The social motivation theory of autism. *Trends in Cognitive Sciences*, 16(4), 231–239.
- Clark, A. (2013). Whatever next? Predictive brains, situated agents, and the future of cognitive science. *Behavioral and Brain Sciences*, 36(3), 181–204.
- Cusack, J. P., Williams, J. H., & Neri, P. (2015). Action perception is intact in autism spectrum disorder. *Journal of Neuroscience*, 35(5), 1849–1857.
- Dennett, D. C. (1989). *The intentional stance*. Cambridge, MA: MIT press.
- Falck-Ytter, T. (2010). Young children with autism spectrum disorder use predictive eye movements in action observation. *Biology Letters*, 6(3), 375–378.
- Falck-Ytter, T., & von Hofsten, C. (2006). Infants predict other people's action goals. *Nature Neuroscience*, 9(7), 878.
- Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism spectrum disorders: a synthesis and meta-analysis. *Journal of Autism and Developmental Disorders*, 40(10), 1227–1240.
- Frith, U. (2012). Why we need cognitive explanations of autism. *The Quarterly Journal of Experimental Psychology*, 65(11), 2073–2092.
- Gallese, V., Keysers, C., & Rizzolatti, G. (2004). A unifying view of the basis of social cognition. *Trends in Cognitive Sciences*, 8, 396–403.
- Greddebäck, G., & Falck-Ytter, T. (2015). Eye movements during action observation. *Perspectives on Psychological Science*, 10(5), 591–598.
- Hamilton, A. D. C. (2009). Research review: Goals, intentions and mental states: Challenges for theories of autism. *Journal of Child Psychology and Psychiatry*, 50(8), 881–892.
- Klin, A., & Jones, W. (2008). Altered face scanning and impaired recognition of biological motion in a 15-month-old infant with autism. *Developmental Science*, 11(1), 40–46.
- Livingston, L. A., & Happé, F. (2017). Conceptualising compensation in neurodevelopmental disorders: Reflections from autism spectrum disorder. *Neuroscience & Biobehavioral Reviews*, 80, 729–742.
- Marsh, L. E., Pearson, A., Ropar, D., & Hamilton, A. D. C. (2015). Predictive gaze during observation of irrational actions in adults with autism spectrum conditions. *Journal of Autism and Developmental Disorders*, 45(1), 245–261.
- Murphy, P., Brady, N., Fitzgerald, M., & Troje, N. F. (2009). No evidence for impaired perception of biological motion in adults with autistic spectrum disorders. *Neuropsychologia*, 47(14), 3225–3235.
- Oberman, L. M., & Ramachandran, V. S. (2007). The simulating social mind: The role of the mirror neuron system and simulation in the social and communicative deficits of autism spectrum disorders. *Psychological Bulletin*, 133, 310–327.
- Paulus, M. (2014). How and why do infants imitate? An ideomotor approach to social and imitative learning in infancy (and beyond). *Psychonomic Bulletin & Review*, 21, 1139–1156.
- Pellicano, E., & Burr, D. (2012). When the world becomes 'too real': a Bayesian explanation of autistic perception. *Trends in Cognitive Sciences*, 16(10), 504–510.
- Ruffman, T. (2014). To belief or not belief: Children's theory of mind. *Developmental Review*, 34, 265–293.
- Saygin, A. P., Cook, J., & Blakemore, S. J. (2010). Unaffected perceptual thresholds for biological and non-biological form-from-motion perception in autism spectrum conditions. *PLoS one*, 5(10), e13491.
- Scheeren, A. M., de Rosnay, M., Koot, H. M., & Begeer, S. (2013). Rethinking theory of mind in high-functioning autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 54(6), 628–635.
- Schuwerk, T., Vuori, M., & Sodian, B. (2015). Implicit and explicit theory of mind reasoning in autism spectrum disorders: the impact of experience. *Autism*, 19(4), 459–468.
- Schuwerk, T., Sodian, B., & Paulus, M. (2016). Cognitive mechanisms underlying action prediction in children and adults with autism spectrum condition. *Journal of Autism and Developmental Disorders*, 46(12), 3623–3639.
- Sebanz, N., Knoblich, G., Stumpf, L., & Prinz, W. (2005). Far from action-blind: Representation of others' actions in individuals with autism. *Cognitive Neuropsychology*, 22(3–4), 433–454.
- Senju, A., Southgate, V., Miura, Y., Matsui, T., Hasegawa, T., Tojo, Y., et al. (2010). Absence of spontaneous action anticipation by false belief attribution in children with autism spectrum disorder. *Development and Psychopathology*, 22(2), 353–360.
- Sinha, P., Kjelgaard, M. M., Gandhi, T. K., Tsourides, K., Cardinaux, A. L., Pantazis, D., et al. (2014). Autism as a disorder of prediction. *Proceedings of the National Academy of Sciences*, 111(42), 15,220–15,225.
- Vivanti, G., McCormick, C., Young, G. S., Abucayan, F., Hatt, N., Nadig, A., et al. (2011). Intact and impaired mechanisms of action understanding in autism. *Developmental Psychology*, 47(3), 841–856.
- von der Lüh, T., Manera, V., Barisic, I., Becchio, C., Vogeley, K., & Schilbach, L. (2016). Interpersonal predictive coding, not action perception, is impaired in autism. *Philosophical Transactions of the Royal Society B*, 371(1693), 20,150,373.
- von Hofsten, C., Uhlig, H., Adell, M., & Kochukhova, O. (2009). How children with autism look at events. *Research in Autism Spectrum Disorders*, 3(2), 556–569.

Active-But-Odd Group

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Definition

Active but Odd

Lorna Wing and Judith Gould (1979) put forward the concept of a spectrum of autistic conditions.

As part of the spectrum, they described different manifestations of social interaction. These were aloof, passive, active but odd in their interactions. Since their early work, an additional group has been included referred to as “over formal and stilted in their approach to others.”

The active-but-odd group are those individuals who make spontaneous approaches to others, but in a peculiar, naïve, and one-sided way. These individuals are usually more able and they approach others on their own terms and their behavior is not modified according to the needs, interests, and responses of the person approached. Often the person seeks to indulge their special interest by talking at another person but not for the pleasure of reciprocal social interaction.

Compared with the aloof and passive groups, this group has much longer vocabularies and use their language considerably more but their speech characteristically is repetitive, long winded, often pedantic with peculiar intonations.

As children, many in this group have pretend play but this is usually repetitive, stereotyped, and often copied from others or DVD's/TV. This type of play can be misinterpreted as imaginative play but on careful observation over time the quality is not representational or symbolic but is a repetitive routine. As adults, this group shows a lack of social imagination. They are unable to foresee the consequences in social and practical terms of their own and other people's actions and to act appropriately on that knowledge. They find it difficult to learn from experience so tend to make the same mistakes repeatedly.

In his paper, Asperger described some children with this pattern of social interaction (1944).

It must be emphasized that there are no clear dividing lines between any of these groups. It is possible for one person to change from one type of social interaction to another or may even show different types of social interaction in different environments, with different people, in different states of health and at different ages. However, at any one time describing the type of social interaction is a helpful indicator in understanding the needs and supporting the individual.

See Also

- [Asperger, Hans](#)
- [Wing, Lorna](#)

References and Reading

- Asperger, H. (1944). Die ‘Autistischen Psychopathen’ im Kindesalter (Autistic psychopaths in childhood). *Archiv für psychiatrie und nervenkrankheiten*, 117, 76–136. (in German). <http://www.springerlink.com/content/u350x0683r1g6432>
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11–29.

Activities of Daily Living

- [Daily Living Skills](#)

Activities of Daily Living (ADLs)

- [Assessment of Functional Living Skills \(AFLS\)](#)

Activity Schedules

- [Daily Routines](#)

Activity-Based Instruction

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Definition

Activity-based intervention (ABI) refers to instruction that is embedded within children's and families' daily activities and routines. The instructional strategies vary according to child goals and needs, but the approach emphasizes child-directed contexts for instruction and the use of naturally occurring antecedents and consequences to develop functional skills.

Historical Background

Diane Bricker and Juliann Woods-Cripe (1992) distinguished activity-based intervention from more traditional approaches to early intervention in the early 1990s. Procedurally, ABI is similar to intervention practices that came earlier, such as incidental language teaching (Hart and Risley 1968), environmental language intervention (MacDonald et al. 1974), embedded instruction (Neef et al. 1984), and routine-based intervention (Dunst et al. 1987). ABI emphasizes the role of parents as teachers and how to capitalize on the potential advantages of parents teaching their children with disabilities during daily activities and routines. Likewise, educators can be encouraged to embed instruction into naturally occurring, daily classroom activities. Activity-based intervention has provided a foundation for the development and evaluation of a number of related interventions that go by a number of general names, such as embedded instruction, routine-based intervention, and integrated therapy. Although there has been a recent focus on teaching intervention targets in the context of children's daily routines in the home, there are also studies on embedding instruction in community settings as well as many studies applying ABI in daily classroom activities.

Rationale or Underlying Theory

Activity-based intervention sought to improve on traditional teaching approaches in several ways. First, this approach was viewed as a way to increase the amount of instruction provided to children with disabilities by involving caregivers as teachers in contexts that would fit into everyday activities and routines. The idea was to capitalize on the natural instruction that many caregivers use with their young children and to expand upon the quantity and quality of those teaching opportunities. Second, the focus on using everyday contexts also provided an approach that would lessen the need to program generalization from contrived teaching situations into everyday contexts. This approach sought to take advantage of the child's interest in stimuli consistently present in the natural environment and the naturally occurring reinforcers that accompany interactions around those everyday events. Third, by focusing on everyday activities, early interventionists, educators, and families might more carefully consider what objectives would be most functional for children with disabilities in present and future environments that they would naturally encounter in their everyday lives. Analyzing those natural contexts could provide insight into what typically occurring antecedents might evoke learned skills and what natural consequences might maintain or strengthen their use.

Goals and Objectives

The goal of ABI is to teach functional skills in the context of daily activities. The specific objectives cut across developmental domains, such as communication, social, cognitive, adaptive or self-help, and motor skills.

Treatment Participants

ABI has been applied to a variety of populations of individuals with developmental disabilities. The bulk of literature has come from early intervention with participants ranging from infants to school age children. Applicability to children with

autism is obvious, especially with a focus on social and communication skills, which tend to be domains of weakness typically addressed to promote the socialization of individuals with autism in natural environments.

Treatment Procedures

ABI represents a departure from practices that were clinician-directed and that took place in clinical or contrived settings. ABI embraced the idea of “natural environments” as a concept that means more than a location for service delivery. It also recognizes that learning should occur in intervention contexts that represent families’ typical and valued activities, routines, and events. Because children learn through participating in their everyday activities and meaningful experiences, ABI seeks to take advantage of these activities as intervention settings. By teaching caregivers, parents, and teachers to take advantage of these learning opportunities, intervention can be dispersed throughout the day to enhance learning and generalization for the child. Although daily routines may be similar across families, they vary in how and when they are completed. Daily activities that follow consistent, predictable sequences, that are repeated frequently, and that produce meaningful, reinforcing outcomes are especially useful for teaching functional skills. Functional skills improve the child’s ability to participate more fully and independently in their natural environments. During familiar routines, opportunities for communication, social, or other responses can be rather predictable. Thus, caregivers are often taught how to prompt and reinforce targeted responses using a range of facilitative strategies that seem appropriate for the child and the caregiver. For example, some caregivers might be taught how to wait and look expectantly to prompt a response, while others might be taught to prompt the child to ask for help before the child gets frustrated. Some caregivers may be encouraged to model targeted responses, and others may be encouraged to prompt more elaborated responses from their child. Sometimes the focus is limited to getting the caregiver to implement a facilitative strategy in one daily activity, and

sometimes the focus is on getting the caregiver to generalize the use of facilitative strategies to multiple activities across the day.

Woods et al. (2004), McWilliam (2010a), Dunst (2001), and their colleagues are among the investigators who have outlined taxonomies for describing daily activities. For example, Kashinath and Woods (2007) highlighted four major categories of family routines: (a) play routines (including constructive play, pretend play, physical play, and social games), (b) caregiving routines (including disability-, dressing-, hygiene-, and food-related activities), preacademic routines (including reading, singing, watching electronic media (TV, computer, video), and writing or drawing), and (d) community and home routines (including community errands, home chores, arts, cultural, and recreational activities). Such frameworks can help families identify the activities that might provide ample learning opportunities for functional skill development in their child.

Implementation of ABI has been characterized as child-centered and family-centered. The child-centered approach emphasizes following the child’s lead and being responsive to the child’s interests, desires, and initiations especially in educational settings. The family-centered approach to ABI requires a great deal of sensitivity on the part of early interventionists to follow the family’s lead and to form a productive partnership. It may take some time to develop a relationship with caregivers that is conducive to open information exchange, observation and discussion of teaching and learning opportunities, joint problem-solving around which routine and facilitative strategies will be most effective, and thoughtful selection of functional target behaviors that will have a meaningful effect on the child’s life. The early interventionist must be aware of the varied values, goals, and circumstances in families’ lives that must be navigated for ABI to be successfully implemented with sufficient frequency and accuracy to be effective.

Efficacy Information

Reviews of naturalistic instruction approaches highlight the difficulty in summarizing the

empirical support for ABI and similar interventions (Hepting and Goldstein 1996; Milagros-Santos and Lignugaris-Kraft 1997; Rule et al. 1998). That is, examples of ABI found in the literature differ quite a bit procedurally, even when called the same thing. Nevertheless, there are numerous studies that have found positive effects from implementing ABI to teach a variety of behaviors, e.g., social skills, picture naming, instruction following, and counting (Pretti-Frontczak et al. 2003). The bulk of the studies summarized by Pretti-Frontczak et al. investigated ABI within classroom settings.

Few of the studies compared ABI to other approaches, such as direct instruction interventions. The advantage of ABI is not necessarily seen during skill acquisition. However, better results tend to be seen in the demonstrations of the generalized use of those skills (e.g., Losardo and Bricker 1994). When teaching strategies are not embedded in activities frequently, then progress on children's targeted objectives tends to be diminished. Milagros-Santos and Lignugaris/Kraft (1997) provide an analysis of instructional features that are likely to affect learning of new skills.

ABI also has been investigated in parent training programs (McWilliam 2010b; Woods et al. 2004). For example, Woods et al. taught caregivers to implement teaching strategies within daily routines; their toddlers with developmental disabilities learned communication skills and demonstrated generalization across routines to varying extents. This work was extended to children with autism (Kashinath et al. 2006).

ABI has broad applicability to teaching a variety of skills, using a variety of intervention agents in a variety of natural contexts or activities. Although evidence indicates that ABI approaches can be effective, procedures for selecting functional goals and teaching them effectively in everyday activities are increasingly being developed. Moreover, as these treatment approaches are better refined, comparative studies will be needed to investigate whether ABI is shown to increase generalization and improve functioning in natural environments in comparison to other approaches.

Outcome Measurement

Any IEP goals that are amenable to use in natural environments could serve as outcome measures. ABI promotes the identification of functional goals that enhance the ability of the child to participate in daily activities with more meaningful involvement and independence. Thus, the outcome measures that are targeted and measured cut across developmental domains (e.g., communication, social, cognitive, adaptive or self-help, and motor skills). Most often, the occurrence of the targeted behaviors is captured through observational data collection. Sometimes, the environment is arranged to enhance the opportunities for the behavior of interest to be demonstrated.

Qualifications of Treatment Providers

ABI has been implemented by a variety of individuals, typically with training provided by an early intervention professional. Parents, caregivers, general and special educators, related service personnel, and paraprofessionals have been responsible for implementing ABI.

See Also

- [Daily Routines](#)
- [Early Intervention](#)
- [Functional Routines \(FR\), Teaching](#)
- [Home-Based Programs](#)
- [Natural Environment](#)
- [Naturalistic Interventions](#)

References and Reading

- Bricker, D., & Woods-Cripe, J. (1992). *An activity-based approach to early intervention*. Baltimore: Paul H. Brookes.
- Dunst, C. J. (2001). Participation of young children with disabilities in community learning activities. In M. J. Guralnick (Ed.), *Early childhood inclusion: Focus on change* (pp. 307–333). Baltimore: Paul H. Brookes.
- Dunst, C. J., Lesko, J., Holbert, K., Wilson, L., Sharpe, K., & Liles, R. (1987). A systemic approach to infant intervention. *Topics in Early Childhood Special Education*, 7(2), 19–37.

- Dunst, C. J., Herter, S., Shields, H., & Bennis, L. (2001). Mapping community-based natural learning opportunities. *Young Exceptional Children*, 4(4), 16–24.
- Hart, B. M., & Risley, T. R. (1968). Establishing use of descriptive adjectives in the spontaneous speech of disadvantaged preschool children. *Journal of Applied Behavior Analysis*, 1, 109–120.
- Hepting, N. H., & Goldstein, H. (1996). What's natural about naturalistic language intervention? *Journal of Early Intervention*, 20(3), 249–264.
- Kashinath, S., Woods, J., & Goldstein, H. (2006). Enhancing generalized teaching strategy use in daily routines by parents of children with autism. *Journal of Speech, Language, and Hearing Research*, 49(3), 466–485.
- Losardo, A., & Bricker, D. D. (1994). Activity-based intervention and direct instruction: A comparison study. *American Journal of Mental Retardation*, 98, 744–765.
- MacDonald, J. D., Blott, J. P., Gordon, K., Spiegel, B., & Hartmann, M. (1974). An experimental parent-assisted treatment program for preschool language-delayed children. *The Journal of Speech and Hearing Disorders*, 39, 395–415.
- McWilliam, R. A. (2010a). *Routines-based early intervention: Supporting young children and their families*. Baltimore: Paul H. Brookes.
- McWilliam, R. A. (Ed.). (2010b). *Working with families of young children with special needs*. New York: Guilford.
- Milagros-Santos, R., & Lignugaris-Kraft, B. (1997). Integrating research on effective instruction with instruction in the natural environment for young children with disabilities. *Exceptionality*, 7(2), 97–129.
- Neef, N. A., Walters, J., & Egel, A. L. (1984). Establishing generative yes/no responses in developmentally disabled children. *Journal of Applied Behavior Analysis*, 17, 453–460.
- Pretti-Frontczak, K., & Bricker, D. (2004). *An activity-based approach to early intervention* (3rd ed.). Baltimore: Paul H. Brookes.
- Pretti-Frontczak, K. L., Barr, D. M., Macy, M., & Carter, A. (2003). Research and resources relate to activity-based intervention, embedded learning opportunities, and routines-based instruction: An annotated bibliography. *Topics in Early Childhood Special Education*, 23(1), 29–39.
- Rakap, S., & Parlak-Rakap, A. (2011). Effectiveness of embedded instruction in early childhood special education: A literature review. *European Early Childhood Education Research Journal*, 19(1), 79–96.
- Rule, S., Losardo, A., Dinnebeil, L., Kaiser, A., & Rowland, C. (1998). Translating research on naturalistic instruction into practice. *Journal of Early Intervention*, 21, 283–293.
- Schwartz, I. S., Billingsley, F. F., & McBride, B. M. (1998). Including children with Autism in inclusive preschools: Strategies that work. *Young Exceptional Children*, 1(2), 19–26.
- Woods, J. J., & Kashinath, S. (2007). Expanding opportunities for social communication into daily routines. *Early Childhood Services*, 1(2), 137–154.
- Woods, J. J., Kashinath, S., & Goldstein, H. (2004). Effects of embedding caregiver-implemented teaching strategies in daily routines on children's communication outcomes. *Journal of Early Intervention*, 26(3), 175–193.

Acuity

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Definition

Given that detailed or locally oriented perception is a central tenet of visual cognition in autism (Behrmann et al. 2006; Dakin and Frith 2005; Mottron et al. 2006), several studies have systematically assessed the spatial resolution of vision in autism by measuring visual acuity (VA). VA is generally defined as the ability to perceive targets such as optotypes, letters, or numbers of a specific size at a given distance. For example, “normal” Snellen VA, often referred to as 20/20 vision, is a clinical term that reflects a person's ability to recognize a target (i.e., letter E) from 20 ft away when its defining spatial features (i.e., spacing of lines composing an E target) are separated by a visual angle of 1 arc minute.

Several studies have assessed VA in ASD using a variety of clinical screening charts. For the most part, VA has been demonstrated to be unremarkable in ASD when assessed with either the Crowded LogMAR test (Milne et al. 2009), chart and/or computer-based Landolt-C optotype paradigms (de Jonge et al. 2007; Keita et al. 2010; Tavassoli et al. 2011; but see Ashwin, Ashwin, Rhydderch, Howells, and Baron-Cohen (2009) with replies from Bach and Dakin (2009)), or Snellen-type visual charts (Falkmer et al. 2011). These demonstrations of unaffected visual acuity in ASD suggest that detailed or locally oriented visual perception in autism is not of peripheral or ocular origin.

A more direct method of assessing the spatial resolution of the visual system is to measure contrast sensitivity as a function of spatial frequency, thus defining a contrast sensitivity function (CSF) that describes the variation of sensitivity over a range of spatial frequencies (defined by cycles per degree or cpd) from detailed (or high-spatial frequency) to less-detailed (or lower spatial frequency) information. Surprisingly, relatively few direct assessments of contrast sensitivity are available for ASD. de Jonge et al. (2007) assessed contrast sensitivity using the Vistech contrast sensitivity chart, which included spatial frequency gratings of 3, 6, 12, and 18 cpd. Albeit nonsignificant, their ASD group demonstrated increased sensitivity from the mid- to high-spatial frequencies. This trend was consistent with the electrophysiological findings of Jemel et al. (2010), who demonstrated that mid- and high-frequency gratings elicited similar brain responses in their ASD group only (responses segregated in control group), suggesting a response bias toward detailed or high-spatial frequency information. However, in the only published behavioral assessment of contrast sensitivity function (CSF) in ASD to date, Koh, Milne, and Dobkins (2010) demonstrated unremarkable visual acuity, peak spatial frequency, peak contrast sensitivity, and contrast sensitivity at a low-spatial frequency in a small group of participants with ASD.

- de Jonge, M. V., Kemner, C., de Haan, E. H., Coppens, J. E., van den Berg, T. J., & van Engeland, H. (2007). Visual information processing in high-functioning individuals with autism spectrum disorders and their parents. *Neuropsychology*, 21, 65–73.
- Falkmer, M., Stuart, G. W., Danielsson, H., Bram, S., Lönebrink, M., & Falkmer, T. (2011). Visual acuity in adults with Asperger's syndrome: No evidence for "eagle-eyed" vision. *Biological Psychiatry*, 70, 812–816.
- Jemel, B., Mimeault, D., Saint-Amour, D., Hosein, A., & Mottron, L. (2010). VEP contrast sensitivity responses reveal reduced functional segregation of mid and high filters of visual channels in autism. *Journal of Vision*, 10(6), 13.
- Keita, L., Mottron, L., & Bertone, A. (2010). Far visual acuity is unremarkable in autism: Do we need to focus on crowding? *Autism Research*, 3, 333–341.
- Koh, H. C., Milne, E., & Dobkins, K. (2010). Spatial contrast sensitivity in adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40, 978–987.
- Milne, E., Griffiths, H., Buckley, D., & Scope, A. (2009). Vision in children and adolescents with autistic spectrum disorder: Evidence for reduced convergence. *Journal of Autism and Developmental Disorders*, 39, 965–975.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36(1), 27–43.
- Tavassoli, T., Latham, K., Bach, M., Dakin, S. C., & Baron-Cohen, S. (2011). Psychophysical measures of visual acuity in autism spectrum conditions. *Vision Research*, 51, 1778–1780.

References and Reading

- Ashwin, E., Ashwin, C., Rhydderch, D., Howells, J., & Baron-Cohen, S. (2009). Eagle-eyed visual acuity: An experimental investigation of enhanced perception in autism. *Biological Psychiatry*, 65, 17–21.
- Bach, M., & Dakin, S. C. (2009). Regarding "Eagle-eyed visual acuity: An experimental investigation of enhanced perception in autism". *Biological Psychiatry*, 66, e19–e20. author reply e23–14.
- Behrmann, M., Thomas, C., & Humphreys, K. (2006). Seeing it differently: Visual processing in autism. *Trends in Cognitive Sciences*, 10(6), 258–264.
- Crewther, D. P., & Sutherland, A. (2009). The more he looked inside, the more piglet wasn't there: Is autism really blessed with visual hyperacuity? *Biological Psychiatry*, 66, e21–e22. author reply e23–24.
- Dakin, S., & Frith, U. (2005). Vagaries of visual perception in autism. *Neuron*, 48(3), 497–507.

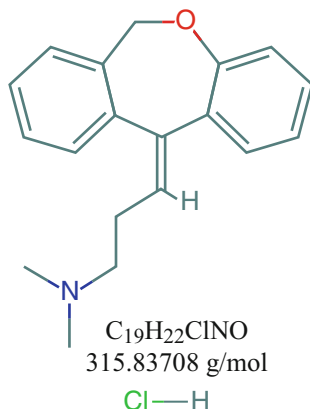
Adapin

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Synonyms

Doxepin; Silenor; Sinequan; Zonalon

Definition



Usually used in the treatment of depression and/or anxiety, Adapin is a member of a class of drugs known as tricyclic antidepressants (TCAs). TCAs work by blocking noradrenaline and serotonin reuptake by presynaptic neurons, resulting in a greater availability and accessibility of these neurotransmitters to the postsynaptic neuron. Long-term administration can result in an increase in sensitivity and number of adrenergic and serotonergic receptors on the postsynaptic neuron. Serotonin is an inhibitory neurotransmitter that is heavily involved in mood and emotion regulation and has been found to be elevated in the whole blood and platelets of ASD patients. However, the use of TCAs in children and adolescents has been fairly limited due to its narrow therapeutic index and high toxicity profile. TCAs have low receptor specificity and, in addition to their reuptake inhibitory effects, exert antagonistic effects on histamine H_2 , serotonin 5-HT_2 , α_1 -adrenergic, and muscarinic acetylcholine receptors. This results in a large array of side effects including, but not limited to, nausea, drowsiness, weakness, dry mouth, changes in appetite or weight gain, constipation, blurred vision, increased sweating, and decreased sexual drive. Evidence for on-target effects and the reduction of autistic symptoms is limited and conflicting. Accordingly, a Cochrane review in 2012 did not recommend the use of tricyclic antidepressants for the treatment of

individuals with autism spectrum disorder. Doxepin has been FDA-approved for the treatment of insomnia.

See Also

- [Antidepressants](#)
- [Serotonin Reuptake Inhibitors \(SRIs\)](#)

References and Reading

- Hurwitz, R., Blackmore, R., Hazell, P., Williams, K., & Woolfenden, S. (2012). Tricyclic antidepressants for autism spectrum disorders (ASD) in children and adolescents. *Cochrane Database of Systematic Reviews*, 3, CD008372.

Adaptive Behavior

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Synonyms

[Functional life skills](#)

Definition

The American Association on Mental Retardation (AAMR 2002) defines adaptive behavior as “the collection of conceptual, social, and practical skills that have been learned by people in order to function in everyday lives.” Adaptive behavior is best understood as the degree to which individuals are able to function and maintain themselves independently and meet cultural expectations for personal and social responsibility at various ages. As such, adaptive behavior involves the person’s physical skills, cognitive ability, affect, motivation, culture, socioeconomic status, family, and environment. Persons with autism spectrum disorders often demonstrate a discrepancy between intellectual potential and

consistently displayed adaptive skills. Assessing adaptive behavior can include standardized adaptive behavior scales, observation, interview, or review of anecdotal records. Some commonly used ratings include the Vineland Adaptive Behavior Scales, Second Edition; Scales of Independent Behavior – Revised (SIB-R); Adaptive Behavior Assessment System – Second Edition (ABAS-II); and the Battelle Developmental Inventory, Second Edition (BDI-2).

See Also

- ▶ [Age Appropriate](#)
- ▶ [Age Equivalents](#)
- ▶ [Daily Living Skills](#)
- ▶ [Developmental Delay](#)
- ▶ [Developmental Milestones](#)
- ▶ [Functional Life Skills](#)
- ▶ [Self-Help Skills](#)

References and Reading

- American Association on Mental Retardation. (2002). *Mental retardation: Definition, classification, and systems of support* (10th ed.). Washington, DC: Author.
- Anderson, S. R., Jablonski, A. L., Thomeer, M. L., & Knapp, V. M. (2007). *Self-help skills for people with autism*. Bethesda: Woodbine House.
- Carter, A. S., Gillham, J. E., Sparrow, S. S., & Volkmar, F. R. (1996). Adaptive behavior in autism. *Mental Retardation*, 5, 945–960.
- Chawarska, K., & Volkmar, F. R. (2005). Autism in infancy and early childhood. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders, volume one: Diagnosis, development, neurobiology, and behavior*. Hoboken: Wiley.
- Harrison, P., & Oakland, T. (2003). *Adaptive behavior assessment system* (2nd ed.). San Antonio: The Psychological Corporation.
- National Research Council. (2001). *Educating children with autism*. (C. Lord & J.P. McGee, Eds.). Committee on Educational Interventions for Children with Autism. Division of Behavioral and Social Sciences and Education. Washington, DC: National Academy Press.
- Openden, D., Whalen, C., Cernich, S., & Vaupel, M. (2009). Generalization and autism spectrum disorders. In C. Whalen (Ed.), *Real life, real progress for children with autism spectrum disorders* (pp. 1–18). Baltimore: Brookes.
- Sattler, J. M., & Hoge, R. D. (2006). *Assessment of children: Behavioral, social, and clinical foundations*. San Diego: Jerome M. Sattler.
- Shea, V., & Mesibov, G. B. (2005). Adolescents and adults with autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders, volume one: Diagnosis, development, neurobiology, and behavior*. Hoboken: Wiley.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales* (2nd ed.). Circle Pines: American Guidance Service.

Adaptive Behavior Assessment System, Second Edition

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Synonyms

[ABAS-II](#); [ABAS, Second Edition](#)

Description

The Adaptive Behavior Assessment System is a reliable, valid, and norms-based questionnaire assessment of adaptive behavior, or the personal and social skills necessary for everyday independent living. Because children and adults with autism spectrum disorders often struggle with practical independent functioning and effective interactions with others, the assessment of adaptive behavior is a crucial part of a comprehensive assessment of individuals on the spectrum.

Now in its second edition, the ABAS-II can be used for individuals across the life span, with norm referenced scores available for ages 0–89. Like other assessments of adaptive behavior, this assessment can be used with individuals with autism spectrum disorders to determine how an individual is responding to day-to-day demands compared to others his/her age, to develop treatment and training goals, to determine eligibility

for services and Social Security benefits, and to assess the capability of adults to live independently. The test may also be used to assess adaptive behavior in individuals with other impairments, including intellectual disability, learning difficulties, or ADHD. The test is published by the Psychological Corporation, and the authors are Patti Harrison & Thomas Oakland.

The ABAS-II assesses three general areas of adaptive behavior: Conceptual, Social, and Practical. These domains were selected according to guidelines of the American Association of Intellectual Disabilities. These three domain areas are divided into ten specific adaptive skill areas, organized according to the specifications of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), which is published by the American Psychiatric Association and provides standard criteria for the classification of mental disorders. These ten skill areas are Communication (e.g., “speaks clearly”), Community Use (e.g., “finds the restroom in public places”), Functional Academics (e.g., “tells time correctly, using a watch or a clock with hands”), Health and Safety (e.g., “carries scissors safely”), Home or School Living (e.g., “sweeps the floor”), Leisure (e.g., “invites others home for fun activity”), Self-Care (e.g., “washes hands with soap”), Self-Direction (e.g., “controls temper when disagreeing with friends”), Social (e.g., “says ‘please’ when asking for something”), Work (e.g., “performs tasks at work neatly”). The Work skills area is optional. Communication, Functional Academics, and Self-Direction areas are a part of the Conceptual domain; Social and Leisure skill areas are a part of the Social domain; and Self-Care, Home or School Living, Community Use, Health and Safety, and Work are a part of the Practical domain. The Motor skills area is not a part of any domain score.

The ABAS-II is available in a five forms, all which assess the same areas of adaptive functioning. Parents of children aged 5–21 may use a rating form; a new form for parents of children aged 0–5 was developed for the second edition. There is also a teacher rating form for individuals aged 5–21, as well as a teacher/day care form for children aged 2–5 (also new to the second edition). Finally, there is an adult form for individuals

aged 16–89, with which adult individuals can report on their own adaptive behavior. Forms include between 193 and 241 items, and items are rated on a 4-point scale, with 0 = is not able, 1 = never when needed, 2 = sometimes when needed, and 3 = always or almost always when needed. An additional category is “check if you guessed,” which helps examiners determine how much confidence to place in responses.

The questionnaire takes approximately 15–20 min to complete and around 5 min to score. Scoring assistance software can aid with the speed and accuracy.

A limited amount of research with the ABAS-II in individuals with autism confirms patterns of adaptive behavior deficits similar to those observed with other assessments of adaptive behavior. For example, in a sample of 40 individuals with high-functioning autism and 30 typically developing controls, individuals with autism demonstrated lower general adaptive composites, as well as specific deficits in social skills. The general adaptive composite was negatively associated with autism symptomatology (Kenworthy et al. 2010).

Historical Background

The Adaptive Behavior Assessment System, Second Edition, is a revision and a downward extension of an earlier first-edition version of the test by the same authors, the Adaptive Behavior Assessment System, published just 3 years prior in 2000. The update was in response to the 2002 AAMR guidelines that suggested looking within conceptual, social, and practical domains of adaptive behavior. The ABAS-II added domain scores for these three areas.

Whereas the first edition was available only for school-aged children and adults, the ABAS-II has two new Infant/Preschool forms to allow for administration to parents of children aged 0–5.

Psychometric Data

The ABAS-II provides scores based on age-related norms, based on a standardization sample

that drew from the US Census data from 1999 to 2000. Thirty-one age groups were assessed for each form, with at least 100 participants per group. In addition to normative samples, the standardization included 20 clinical samples, including a clinical sample for autism. However, these clinical samples were small and not randomly selected, so no autism-specific norms exist for the ABAS-II.

A General Adaptive Composite, with a mean of 100 and a standard deviation of 15, is yielded as an overall measure of an individual's adaptive skills. In the second edition, domain composite scores, also with means of 100 and a standard deviation of 15, are yielded. Skill area standard scores have a mean of 10 and a standard deviation of 3. Confidence intervals and descriptive classifications are also provided. Finally, for individuals up to 22 years, age-based percentile ranks and age equivalencies are yielded. The GAC has a lowest possible score of 40, and the GAC ceiling for 0–5 is 160, and for adults and children over 8 it is 120. In addition to scaled scores, information about relative strengths and weaknesses by skill area as well as base rates in the standardization sample are provided.

On the school-aged parent and teacher data from the standardization sample, girls scored significantly higher than boys on the General Adaptive Composite, and this gender effect was stronger in teachers; however, gender accounted for only a small amount of variance (.6% and 2.7%). These gender differences are consistent with some other adaptive behavior tests (e.g., the Adaptive Behavior Inventory for Children, which showed similar patterns), though not all measures of adaptive behavior (e.g., the Vineland Adaptive Behavior Scales does not demonstrates sex differences). Effects of race were also observed in the standardization sample, with white children scoring higher than Latino children. Again, an ethnicity main effect has been observed in some but not all other assessments of adaptive behavior. Given that adaptive behavior is defined according to the cultural norms and expectations regarding independent behavior and social functioning, sensitivity to cultural context is a critical part of the sensitive assessment of adaptive behavior.

The ABAS-II has shown very strong reliability. Most skill areas have internal consistency of .90 or higher. In studies examining test-retest reliability over a 2-week period, General Adaptive Composite correlations were near or above .90 for all versions of the ABAS-II.

The test also demonstrates adequate validity. Factor analysis supports both the three-factor model and the GAC factor. The factor model is similar for boys and girls (Wei et al. 2008). Comparisons to other adaptive behavior measures, such as the Vineland, show correlations ranging between .70 and .84, demonstrating concurrent validity. Clinical validity studies have also suggested that the ABAS-II is highly sensitive when differentiating clinical and nonclinical samples. Correlations with the Wechsler Intelligence Scale for Children-Third Edition, the Wechsler Adult Intelligence Scale-Third Edition, and the Wechsler Abbreviated Scale of Intelligence were medium-sized, confirming that intelligence and adaptive functioning are inter-related but distinct constructs. No predictive validity studies are known.

Items were selected from an original pool of 1500 generated items, from which a third to a half were used in standardization sampling.

The test has been criticized for requiring a high level of reading comprehension for some items (seventh grade) and for its relatively low ceiling (120) (Sattler 2002).

Clinical Uses

Adaptive skills generate opportunities for independence and meaningful social interaction. Given that core deficits in social and communication skills are at the heart of a diagnosis of Autism Spectrum Disorder, measurement of the adaptive skills that children and adults are using – and where they may need remediation – is a key component of assessment and treatment planning for individuals with ASDs. Indeed, some conceptualizations of developmental disabilities suggest more emphasis be placed on adaptive skills than on IQ, as adaptive skills are modifiable and capture real-world implementation, whereas intellectual ability does not necessarily capture the skills an individual is *using* in a day-to-day context

(Schalock 1999). Individuals with autism, particularly high-functioning ones, typically have a profile that includes adaptive skill levels that are lower than intelligence levels.

The ABAS-II provides a categorical and age-normed assessment of individuals' adaptive skills, which can be used to guide treatment planning. The ABAS-II can also be used to generate a profile of an individual's adaptive skills, so that areas of relative strength and weakness can be better understood. For example, if an individual is shown to demonstrate deficits in a skill area (e.g., Social), then specific behavioral interventions can be built around the specific deficits documented in testing. Assessing adaptive behaviors across a range of settings (e.g., home and school) can also provide information about the generalization of skills. Moreover, using a measure like the ABAS-II over time can document progress in adaptive skills or capture students' response to intervention, a critical component of special education service planning.

The ABAS-II can also be used to determine eligibility for services, such as Social Security and special education services under the Individuals with Disabilities Education Act. A documented deficit in adaptive behavior is necessary for a diagnosis of intellectual disability, which often co-occurs with ASDs. Investigating profiles of adaptive skill strengths and weaknesses using the ABAS can also be helpful in differential diagnosis, as children and adults on the spectrum tend to have particular adaptive skill deficits in social and communication areas of adaptive skills.

Kenworthy, L., Case, L., Harms, M. B., Martin, A., & Wallace, G. L. (2010). Adaptive behavior ratings correlate with symptomatology and IQ among individuals with high-functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(4), 416–423.

Oakland, T., & Algina, J. (2011). Adaptive behavior assessment system-II parent/primary caregiver form: Ages 0–5: Its factor structure and other implications for practice. *Journal of Applied School Psychology*, 27(2), 103.

Oakland, T., & Harrison, P. L. (Eds.). (2008). *Adaptive behavior assessment system II: Clinical use and interpretation*. San Diego: Academic.

Ozonoff, S., Goodlin-Jones, B. L., & Solomon, M. (2005). Evidence-based assessment of autism spectrum disorders in children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, 34(3), 523–540.

Rust, J., & Wallace, M. (2004). Book review: Adaptive behavior assessment system – Second edition. *Journal of Psychoeducational Assessment*, 22, 367–373.

Sattler, J. M. (2002). *Assessment of children: Behavioral and clinical applications*. San Diego: Author.

Schalock, R. L. (Ed.). (1999). *Adaptive behavior and its measurement in the field of mental retardation*. Washington, DC: American Association on Mental Retardation.

Wei, Y., Oakland, T., Algina, J., & MacLean, W. E. (2008). Multigroup confirmatory factor analysis for the adaptive behavior assessment system-II parent form, ages 5–21. *American Journal on Mental Retardation*, 113(3), 178–186.

Adaptive Behavior Predicting Postschool Outcomes

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See Also

- [Maladaptive Behavior](#)
- [Vineland Adaptive Behavior Scales \(VABS\)](#)

References and Reading

- Harrison, P. L., & Oakland, T. (2000). *Adaptive behavior assessment system*. San Antonio: The Psychological Corporation.
- Harrison, P. L., & Oakland, T. (2003). *Adaptive behavior assessment system* (2nd ed.). San Antonio: The Psychological Corporation.

Definition

Adaptive Behavior

The construct of adaptive behavior has been defined by both the American Association on Intellectual and Developmental Disabilities (AAIDD) and the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) as the collection of conceptual, social, and practical skills that are learned and performed by people in their everyday lives. These three domains of adaptive behavior – conceptual, social, and practical skills – have been

consistently identified through factor analytic work on adaptive behavior (Tassé et al. 2012). Conceptual skills refer to language abilities, reading and writing, numbers, time, and money concepts. Social skills include interpersonal skills, friendships, social participation, and social problem-solving. Practical skills encompass self-care, activities of daily living, health and safety, ability to use transportation, etc.

Postschool Outcomes

Researchers have struggled over the years to define what it means for a person with autism to be successful in adulthood (Henninger and Taylor 2013). As adults with autism have increasingly moved out of institutions and into the community, the criteria for positive outcomes have shifted from merely avoiding life in an institution to independently functioning within the community. Three key areas of postschool outcomes are regularly discussed in the literature on disabilities at large, including ASD: postsecondary education, employment, and independent living. In their systematic review of evidence-based predictors of successful postschool outcomes, the National Technical Assistance Center on Transition (NTACT) identified these three areas as the critical outcomes of interest for students with disabilities (Test et al. 2009). Postsecondary education participation is defined as enrollment in any vocational or technical school, 2-year or community college, or 4-year college. Competitive employment – defined as a job at which a majority of the other workers do not have a disability and the person with a disability is earning at least minimum wage – is generally considered the gold standard for employment among young adults with ASD. Residential independence refers to living on one's own or with a roommate, partner, or spouse (i.e., not with caregivers or paid staff, although support from these individuals may be provided). In the literature specific to autism spectrum disorder (ASD), social relationships are often considered another important component of postschool outcomes, including having at least one meaningful friendship with a person in the same age group who is not a paid staff member (Henninger and Taylor 2013).

Historical Background

Transition to Adulthood for Youth with ASD

The transition from secondary school to adulthood is a growing area of concern within the ASD community. In fact, many researchers describe this period of time as a “services cliff” because of the steep decline in services that are available after a student with ASD graduates high school (Roux et al. 2015). It has been well-documented that adults with ASD have poor outcomes compared to their peers without disabilities and peers with other types of disabilities. Data from the National Longitudinal Transition Study-2 (NLTS2) – a large, nationally representative, 10-year longitudinal study of transition-aged youth receiving special education services – is commonly used to summarize the postschool outcomes of youth with ASD and other kinds of disabilities (Newman et al. 2011; Roux et al. 2015). These data show that among young adults with autism ages 23–26, just 44% attended any type of postsecondary education, less than all other disability types except intellectual disability and multiple disabilities. Seventeen percent lived independently, which was lower than all other disability types except multiple disabilities. Twenty-four percent had no socialization with peers, either in person or on a phone call, in the past year, which was lower than all other disabilities studied. Just 63% of the sample with ASD had worked for pay since leaving high school, which was lower than all other disability types except multiple disabilities. Additionally, the majority of people with ASD who had a paid job were working part-time jobs for approximately minimum wage. In many cases, it took 2 years or more to find a job after graduating high school. Findings also show that young adults with ASD have difficulty maintaining a job relative to their peers with other kinds of disabilities. These negative outcomes exist even among adults with ASD who have average or above-average intelligence. Despite average cognitive abilities, they are struggling to engage in typical adult activities. Based on these outcomes, it appears these young adults do not have the skills or supports they need to succeed in their adult lives. Therefore, it is crucial to understand the factors that predict a successful transition.

Adaptive Behavior Profiles in ASD

Research indicates that adaptive skills are likely a key factor in the successful transition to adulthood for youth with ASD. Children and adults with ASD have unique adaptive behavior profiles. It is relatively common for people with ASD to have “scatter” in their skills, meaning that scores on some domains are much higher than others. Research consistently finds that people with ASD have the lowest scores in socialization, as measured by the Vineland Adaptive Behavior Scales (e.g., Kanne et al. 2011). However, some studies have described an “autism profile” in which daily living skills are an area of relative strength, while others find that daily living skills are a relative weakness and communication is the greatest strength. Regardless of the relative strengths and weaknesses found in particular papers, it is important to remember that they are just that: relative. It is well-established that scores on all domains of adaptive behavior for people with ASD are low relative to typically developing peers.

In fact, this pattern of low adaptive behavior scores in people with ASD is a robust finding that exists independent of cognitive functioning. There is a well-documented gap between IQ and adaptive behavior scores in this population. Adaptive functioning often lags behind cognitive level, especially among those with average or above-average IQ (Alvares et al. 2019; Kraper et al. 2017), such that even people with ASD who have high IQ scores often have significantly impaired adaptive behavior. This gap has been shown to emerge as early as 12–36 months of age and widen throughout childhood development and into adolescence (Bradshaw et al. 2019; Kanne et al. 2011; Klin et al. 2007), with gaps as large as 2 standard deviations in some studies (Klin et al. 2007). The gap between IQ and adaptive behavior is largest in people with ASD who are older and who have higher IQs (Kanne et al. 2011). In fact, some studies have found that this gap only exists in those with average intelligence and have found adaptive skills commensurate with mental age in those with intellectual disability (Kanne et al. 2011).

While much of the research on adaptive behavior in ASD has been done on children, some

studies have documented plateaus or declines in adaptive behavior scores through adolescence (Klin et al. 2007; Meyer et al. 2018), where continued improvements are expected. It is important to note that this does not suggest adolescents with ASD are losing adaptive skills, but rather that their rate of progress is slowing down relative to their same-aged peers without ASD. Importantly, age is not associated with either ASD symptom severity or cognitive abilities, so these declines in skill development are found only with regard to adaptive behavior. Therefore, deficits in functional skills are decreasing despite these other two variables remaining the same (Kanne et al. 2011). This trend is worrisome and suggests the need for increased intervention on adaptive skills specifically as children with ASD enter adolescence.

Current Knowledge

In order to improve transition practices and promote positive outcomes for young adults with ASD, it is necessary to understand the factors that influence these outcomes. In the broader disability literature, there is evidence that adaptive behavior skills predict postschool outcomes. For instance, in their systematic review of transition literature across all federal special education disability categories, Test et al. (2009) found that students with more self-care and daily living skills were more likely to participate in all three post-school outcomes of interest: postsecondary education, independent living, and employment. They also found that students with better social skills had improved outcomes in postsecondary education attendance and employment. In a sample of participants with intellectual disability, Dell’Armo and Tassé (2019) found that adaptive behavior variables (social skills, academic performance, and functional skills) were more predictive of postschool outcomes than parent expectations or demographic factors such as race, household income, and parent education level. In a sample with ASD, adaptive behavior – particularly the Daily Living Skills subscale of the Vineland Adaptive Behavior Scales – was most closely correlated with positive adult outcomes

(Farley et al. 2009). Taken together, all of this research suggests that adaptive behavior is likely an important variable in predicting postschool outcomes in ASD, much like in the broader disability population.

Unfortunately, there is relatively little research investigating the effects of adaptive behavior on postschool outcomes. Review papers on predictors of outcomes (e.g., Kirby et al. 2016) have identified relatively few adaptive behavior variables being studied. Much of the research that does exist uses data from the National Longitudinal Transition Study-2 (NLTS). These data are beneficial for studying postschool outcomes as they are longitudinal data that were collected over a period of 10 years. Participants were enrolled in high school at the start of the study and had left high school by the end of the 10 years. Therefore, the data allows researchers to not only describe postschool outcomes but to investigate variables from earlier time points that may be associated with those outcomes. However, a limitation of NLTS2 data is that a standardized adaptive behavior measure was not administered to participants. Therefore, researchers interested in studying adaptive behavior must attempt to use existing variables as a proxy for adaptive behavior (e.g., Dell'Armo and Tassé 2019). Despite this shortcoming, the longitudinal nature of the dataset makes it one of the best available datasets for exploring variables related to postschool outcomes.

Secondary analyses using NLTS2 data have provided further insight into how adaptive behavior predicts postschool outcomes. In a sample of students with autism, Chiang et al. (2012) found that academic performance – which would fall under the conceptual domain of adaptive behavior – was a significant predictor of enrollment in postsecondary education. Similarly, Wei et al. (2013) found that young adults with ASD who had higher levels of basic conceptual skills (i.e., telling time, counting change, reading and understanding signs) were more likely to enroll in postsecondary education.

These same basic conceptual skills, along with conversation ability, have also been associated with social participation outcomes. Orsmond

et al. (2013) found that young adults with ASD who had lower levels of all these skills were less likely to see friends, be called by friends, and be invited to activities (i.e., were more likely to be socially isolated). In terms of employment, Roux et al. (2013) found greater levels of these same basic conceptual skills were associated with greater odds of paid employment, as were greater levels of conversational ability. Another study determined that social skills were a significant predictor of employment after leaving high school (Chiang et al. 2013).

Although studies of adult outcomes do not typically measure psychiatric symptoms, they are important in this population because research suggests that people with ASD experience co-occurring mental health conditions at much greater rates than in the general population (Simonoff et al. 2008). In the only study investigating this topic, Kraper et al. (2017) found that the size of the gap between IQ and adaptive behavior was significantly related to comorbid psychopathology, such that those with greater discrepancies between IQ and adaptive behavior scores were more likely to have another mental health diagnosis like anxiety or depression. While more research is needed to replicate this finding, it appears that adaptive behavior may also impact psychiatric outcomes and psychological well-being.

Future Directions

It should be clear from the information presented that an increased focus on adaptive behavior skills for youth with ASD as they transition to adulthood is warranted. The research that exists on adaptive behavior profiles in this age group suggests that adaptive behavior, and not IQ, is most predictive of postschool outcomes. There are countless examples of young adults with ASD who are of average or above-average intelligence, but adaptive behavior deficits prevent them from living independently, keeping a job, or otherwise succeeding in their adult lives. Future research should put more focus on adaptive behavior for people with ASD in this age group. It would be

useful to know more about typical adaptive behavior profiles (including common areas of relative strength or weakness, as current findings are somewhat conflicting) as well as their longitudinal trajectory (i.e., how do adaptive skills change as youth with ASD age out of school and enter young adulthood?). In addition, these questions should be studied separately in people with ASD who have comorbid ID and those who do not, as prior research suggests that adaptive behavior profiles and outcomes may differ for these two groups.

Despite the importance of adaptive behavior, relatively little work has been done examining the factors that predict or influence adaptive skills (Kraeper et al. 2017). A better understanding of the development of adaptive behavior skills is warranted, with the hope that this would lead to increased knowledge of how to improve adaptive skills in people with ASD. Increased focus should be placed on improving adaptive behavior skills, both in research and in clinical practice. Adaptive behavior skills, in particular those that relate to adult outcomes, should be a target of intervention for all youth with ASD as they transition to adulthood. However, it is still unclear exactly which adaptive skills are most related to adult outcomes. Very little research has specifically investigated how adaptive behavior relates to postschool outcomes, and virtually none of the current research investigating predictors of postschool success use standardized, comprehensive measures of adaptive behavior, as most comes from the same NLTS2 dataset. More research should be conducted to identify which adaptive skills are the most important, as well as develop effective interventions for improving these skills.

See Also

- Factors Affecting Outcomes
- Functional Life Skills
- Outcome Studies
- Quality of Life For Transition-Age Youth with ASD

References and Reading

- Alvares, G. A., Bebbington, K., Cleary, D., Evans, K., Glasson, E. J., Maybery, M. T., Pillar, S., Uljarević, M., Varcin, K., Wray, J., & Whitehouse, A. J. (2019). The misnomer of 'high functioning autism': Intelligence is an imprecise predictor of functional abilities at diagnosis. *Autism*. Advance online publication. <https://doi.org/10.1177/1362361319852831>.
- Bradshaw, J., Gillespie, S., Klaiman, C., Klin, A., & Saulnier, C. (2019). Early emergence of discrepancy in adaptive behavior and cognitive skills in toddlers with autism spectrum disorder. *Autism*, 23(6), 1485–1496.
- Chiang, H. M., Cheung, Y. K., Hickson, L., Xiang, R., & Tsai, L. Y. (2012). Predictive factors of participation in postsecondary education for high school leavers with autism. *Journal of Autism and Developmental Disorders*, 42(5), 685–696. <https://doi.org/10.1007/s10803-011-1297-7>.
- Chiang, H. M., Cheung, Y. K., Li, H., & Tsai, L. Y. (2013). Factors associated with participation in employment for high school leavers with autism. *Journal of Autism and Developmental Disorders*, 43(8), 1832–1842. <https://doi.org/10.1007/s10803-012-1734-2>.
- Dell'Armo, K., & Tassé, M. J. (2019). The role of adaptive behavior and parent expectations in predicting post-school outcomes for young adults with intellectual disability. *Journal of Autism and Developmental Disorders*, 49, 1638–1651. <https://doi.org/10.1007/s10803-018-3857-6>.
- Farley, M. A., McMahon, W. M., Fombonne, E., Jenson, W. R., Miller, J., Gardner, M., et al. (2009). Twenty-year outcome for individuals with autism and average or near-average cognitive abilities. *Autism Research*, 2, 109–118.
- Henninger, N. A., & Taylor, J. L. (2013). Outcomes in adults with autism spectrum disorders: A historical perspective. *Autism*, 17, 103–116.
- Kanne, S. M., Gerber, A. J., Quirnbach, L. M., Sparrow, S. S., Cicchetti, D. V., & Saulnier, C. A. (2011). The role of adaptive behavior in autism spectrum disorders: Implications for functional outcome. *Journal of Autism and Developmental Disorders*, 41(8), 1007–1018.
- Kirby, A. V., Baranek, G. T., & Fox, L. (2016). Longitudinal predictors of outcomes for adults with autism spectrum disorder: Systematic review. *Occupation, Participation, and Health*, 36(2), 55–64.
- Klin, A., Saulnier, C. A., Sparrow, S. S., Cicchetti, D. V., Volkmar, F. R., & Lord, C. (2007). Social and communication abilities and disabilities in higher functioning individuals with autism spectrum disorders: The Vineland and the ADOS. *Journal of Autism and Developmental Disorders*, 37(4), 748–759.
- Kraeper, C. K., Kenworthy, L., Popal, H., Martin, A., & Wallace, G. L. (2017). The gap between adaptive behavior and intelligence in autism persists into

- young adulthood and is linked to psychiatric co-morbidities. *Journal of Autism and Developmental Disorders*, 47, 3007–3017. <https://doi.org/10.1007/s10803-017-3213-2>.
- Meyer, A. T., Powell, P. S., Butera, N., Klinger, M. R., & Klinger, L. G. (2018). Brief report: Developmental trajectories of adaptive behavior in children and adolescents with ASD. *Journal of Autism and Developmental Disorders*, 48(8), 2870–2878.
- Newman, L., Wagner, M., Knokey, A.-M., Marder, C., Nagle, K., Shaver, D., Wei, X., with Cameto, R., Contreras, E., Ferguson, K., Greene, S., and Schwarting, M. (2011). The post-high school outcomes of young adults with disabilities up to 8 years after high school. A report from the national longitudinal transition study-2 (NLTS2) (NCSE 2011-3005). Menlo Park: SRI International.
- Orsmond, G. I., Shattuck, P. T., Cooper, B. P., Sterzing, P. R., & Anderson, K. A. (2013). Social participation among young adults with an autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43, 2710–2719.
- Roux, A. M., Shattuck, P. T., Cooper, B. P., Anderson, K. A., Wagner, M., & Narendorf, S. C. (2013). Postsecondary employment experiences among young adults with an autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(9), 931–939.
- Roux, A. M., Shattuck, P. T., Rast, J. E., Rava, J. A., & Anderson, K. A. (2015). *National autism indicators report: Transition into young adulthood*. Philadelphia: Life Course Outcomes Research Program, A.J. Drexel Autism Institute, Drexel University.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, 47(8), 921–929.
- Tassé, M. J., Schalock, R. L., Balboni, G., Bersani, H., Borthwick-Duffy, S. A., Sprent, S., Thissen, D., Widaman, K. F., & Zhang, D. (2012). The construct of adaptive behavior: Its conceptualization, measurement, and use in the field of intellectual disability. *American Journal on Intellectual and Developmental Disabilities*, 117, 291–303. <https://doi.org/10.1352/1944-7558-117.4.291>.
- Test, D. W., Mazzotti, V. L., Mustian, A. L., Fowler, C. H., Kortering, L., & Kohler, P. (2009). Evidence-based secondary transition predictors for improving post-school outcomes for students with disabilities. *Career Development for Exceptional Individuals*, 32(3), 160–181. <https://doi.org/10.1177/0885728809346960>.
- Wei, X., Jennifer, W. Y., Shattuck, P., McCracken, M., & Blackorby, J. (2013). Science, technology, engineering, and mathematics (STEM) participation among college students with an autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43(7), 1539–1546.

Adaptive Behavior Scales

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Definition

Adaptive behavior scales can provide information about children's communication, socialization, and other everyday behavior relative to their age (Demchak and Drinkwater 1998; Gillham et al. 2000). Adaptive behavior scales are different from intelligence tests in that they measure what a child does in the real world versus what a child is capable of in a structured testing situation (Volkmar 2003). The most widely used adaptive behavior scale is the Vineland Adaptive Behavior Scales (Sparrow et al. 1984, 2005); which is a semi-structured interview with parents or caregivers to assess capacities for self-sufficiency in various areas, including communication, daily living, and social, as well as for young children, motor skills.

Historical Background

Beginning with the first descriptions of mental retardation in the 1800s, limits to or an inability to adapt to the demands of everyday life, that is, adaptive behavior, was emphasized as a main descriptor of people considered to have mental retardation (Bothwick-Duffy 2007). Adaptive behavior scales were developed to identify behavior deficits needing treatment in people who were already known to have a disability. Eventually, assessment of adaptive behavior became used for the purposes of diagnosis and eligibility for special services (Bothwick-Duffy 2007). Although social factors are currently viewed as a central defining characteristic of the autistic syndrome, earlier research did not systematically evaluate social dysfunction in autistic individuals; therefore, the utility of a well-standardized, normative

assessment instrument for documenting autistic social dysfunction in terms of daily adaptive functioning became clear (Volkmar et al. 1987).

Literature describing adaptive deficits in autism emerged with Sparrow et al. (1984) and Volkmar et al. (1987) in the 1980s. The assessment of adaptive behavior in individuals with autism along with standardized measures of intellectual functioning was developed to determine whether or not to assign a diagnosis of mental retardation or intellectual disability as well as to distinguish between Autism Spectrum Disorders and other intellectual and developmental disabilities (Carter et al. 1998).

Multiple assessments were created to measure these adaptive skills. The Behavior Inventory for Rating Development (BIRD) was designed to assess types and levels of adaptive behaviors. The BIRD is classified into several subscales of adaptive behavior; Cognitive Development, Self-Help, Physical Development, Social Behavior, and Self-Control (Sparrow and Cicchetti 1984). Other measures of adaptive behavior include the Comprehensive Test of Adaptive Behavior (Adams 1984); Scales of Independent Behavior (Bruinicks et al. 1984); and the Adaptive Behavior Inventory (Brown and Leigh 1986). The more widely used measure, the Vineland Adaptive Behavior Scales-Survey Form evaluates children's personal and social sufficiency in a semi-structured interview with a primary caregiver (Sparrow et al. 1984). This instrument assesses four areas of adaptive behavior: Communication, Daily Living Skills, Socialization, and Motor Skills (Carter et al. 1998; Sparrow et al. 1984).

Current Knowledge

Adaptive skills include whatever capacities an individual possesses to function within their everyday environment, encompassing self-sufficiency as well as social competence (Demchak and Drinkwater 1998; Paul et al. 2004). These skills are particularly important in individuals with autism and related conditions because they contribute the most to an individual's ability to function successfully and

independently in the world (Liss et al. 2001). Adaptive behavior, or children's ability to take care of themselves and get along with others, is an extremely important aspect of multi-dimensional assessment and interventions for preschool and school-aged children as well as for adolescents and adults. Adaptive behavior assessment is useful for diagnosing possible disabilities and developmental problems of preschoolers, which then can lead to planning effective home, family, and school programs (Harrison and Raineri 2007). Given that adaptive behavior is modifiable, it can lead to planning effective home, family, school, community, and vocational planning through the life span.

As noted, the most widely used measurement of adaptive behavior is the Vineland Adaptive Behavior Scales, which are broken down into four scales. The Communication scale refers to skills required for receptive, expressive, and written language; Daily Living Skills scale includes the practical skills needed to take care of oneself and contribute to a household and community; Socialization scale includes skills needed to get along with others, regulate emotions and behavior, as well as skills involved in leisure activities such as play; and finally the Motor Skills scale, comprising both fine and gross motor items, which is typically assessed in individuals below the age of 6 years or when significant difficulty in motor development is suspected. Additionally, the Vineland also contains a Maladaptive Behavior Domain, which assesses the presence of problematic behaviors that interfere with an individual's functioning. The Maladaptive Behavior Domain can be administered to children aged 5 and older and includes both behaviors that are common in early development but are less common as children get older and more serious behaviors that are of concern throughout development (Carter et al. 1998; Sparrow et al. 1984). Further explanation of these scales is as follows: the Communication scale includes expressive, which is what an individual says, while receptive is what an individual understands, and written is what an individual reads and writes. The Daily Living scale includes personal information such as how an individual eats, dresses, and practices personal hygiene, as

well as domestic information such as what household tasks an individual performs, and finally community, such as how an individual uses time, money, the telephone, and job skills. The Socialization scale includes interpersonal information such as how an individual interacts with others, as well as play and leisure, such as how an individual plays and uses leisure time, as well as coping, or how an individual shows responsibility and sensitivity to others (Paul et al. 2004).

Volkmar and colleagues evaluated the ability of the Vineland Adaptive Behavior Scales to diagnose autism by looking at multiple regression equations to predict expected socialization and communication skills on the basis of age, parent education, and sex of the child (Volkmar et al. 1993). While deficits in both communication and socialization are characteristic of the disorder, individuals with autism tend to evidence greater impairment in socialization relative to both communication and daily living skills (Carter et al. 1996). Children with autism display significantly poorer daily living skills and more serious maladaptive behaviors than those with other developmental disorders (Gillham et al. 2000).

Multiple studies have confirmed that the Vineland Adaptive Behavior Scales (Sparrow et al. 1984), is a well-standardized, semi-structured instrument for assessing adaptive behavior. Gillham et al. (2000) reported that autism could be differentiated from both PDD-NOS and non-autistic developmental disorder (DD) with the Socialization and Daily Living scales of the Vineland Adaptive Behavior Scales (Paul et al. 2004; Sparrow et al. 1984). Children with PDD-NOS, when compared with those with autism, differ only in very specific areas, primarily the use of expressive language for communication – particularly syntax and pragmatics – and the areas of adaptive function on which these skills have a direct effect, such as phone use, manners in conversation, and using language to identify and initiate interaction with others (Paul et al. 2004).

Studies have compared the Vineland Adaptive Behavior Scales with other Adaptive Behavior measures and found significant between score correlations (Villa et al. 2010). An international study that compared the Scales of Independent

Behavior (SIB) and the revised Vineland Adaptive Behavior Scales revealed one similar significant factor, demonstrating personal independence for both tests. The summary scores of both tests were found to correlate moderately with IQ as well as with the extent of integration children achieved in their subsequent school placement (Roberts et al. 1993).

There are state and local differences in the adoption of specific criteria for deficits in adaptive behavior. However, the development of instruments that provide national norms such as the Comprehensive Test of Adaptive Behavior (Adams 1984) and Vineland Adaptive Behavior Scales (Sparrow et al. 1984) have enabled more normalized and quantifiable guidelines that could be widely used (Carter et al. 1998).

Adaptive behavior scales have multiple implications for clinical practice, including assessment, diagnosis, and treatment planning. In contrast to intellectual functioning, adaptive behavior is modifiable (Carter et al. 1998). For all individuals, however, cognitive functioning will set some constraints on the level of adaptive functioning that can be achieved. The adaptive behavior scales are a crucial component of a developmental and diagnostic assessment for children with autism and potential comorbid intellectual disability. Additionally, determining strengths and weaknesses in everyday skills has important implications for intervention planning and family support (Perry et al. 2009) and can inform recommendations for educational and psychotherapeutic interventions for high- and low-functioning individuals (Carter et al. 1996). Adaptive behavior scales have been applied to instructional program planning for disabled preschool and school-aged children, adolescents, and adults (Demchak and Drinkwater 1998). Additionally, the assessment of adaptive behavior can be used as an outcome measure to document the efficacy of intervention programs (Carter et al. 1998).

Future Directions

While considerable gains have been made in the development of adaptive behavior scales,

continued research into their generalizability and cultural sensitivity is imperative, as with all measures. The impact of adaptive behavior scales is widely felt, as they are integral to the diagnosis of intellectual disability and have become a key component of assessment and intervention planning for individuals with autism spectrum disorders. Therefore, it is critical that individuals designing intervention programs set attainable goals across domains of adaptive functioning to lead to increased self-efficacy for all involved (Carter et al. 1998). Further research could explore the connections between outcomes of adaptive behavior scales and successful intervention, providing further guidance for practitioners who design these intervention goals. The importance of intensive intervention in the area of adaptive behavior, particularly for children with autism spectrum disorders, remains clear and continued research into successful interventions is necessary.

See Also

- ▶ [Adaptive Behavior Assessment System, Second Edition](#)
- ▶ [Intellectual Disability](#)
- ▶ [Maladaptive Behavior](#)
- ▶ [Mental Retardation](#)
- ▶ [Vineland Adaptive Behavior Scales \(VABS\)](#)

References and Reading

- Adams, G. L. (1984). *Comprehensive test of adaptive behavior*. San Antonio: Psychological Corporation.
- Bothwick-Duffy, S. (2007). Adaptive behavior. In J. Jacobson, J. Mulick, & J. Rojahn (Eds.), *Handbook of intellectual and developmental disabilities* (pp. 279–293). New York: Springer.
- Brown, L., & Leigh, J. E. (1986). *Adaptive behavior scale*. Austin: PRO-ED.
- Bruinicks, R. H., Woodcock, R. W., Weatherman, R. F., & Hill, B. K. (1984). *Scales of independent behavior*. Allen: DLM Teaching Resources.
- Carter, A., Gillham, J., Sparrow, S., & Volkmar, F. (1996). Adaptive behavior in autism. *Child and Adolescent Psychiatric Clinics of North America*, 5(4), 945–961.
- Carter, A., Volkmar, F., Sparrow, S., Wang, J., Lord, C., Dawson, G., Fombonne, E., Loveland, K., Mesibov, G., & Schopler, E. (1998). The Vineland adaptive behavior scales: Supplementary norms for individuals with autism. *Journal of Autism and Developmental Disorders*, 28(4), 287.
- Demchak, M., & Drinkwater, S. (1998). Assessing adaptive behavior. In V. Boone (Ed.), *Psychological assessment of children: Best practices for school and clinical settings* (2nd ed., pp. 297–322). Hoboken: Wiley.
- Gillham, J. E., Carter, A. S., Volkmar, F. R., & Sparrow, S. S. (2000). Toward a developmental operational definition of autism. *Journal of Autism and Developmental Disorders*, 30(4), 269.
- Harrison, P., & Raineri, G. (2007). Adaptive behavior assessment for preschool children. In B. A. Bruce & R. J. Nagle (Eds.), *Psychoeducational assessment of preschool children* (4th ed., pp. 195–218). Mahwah: Lawrence Erlbaum Associates.
- Liss, M., Harel, B., Fein, D., Allen, D., Dunn, M., Feinstein, C., Morris, R., Waterhouse, L., & Rapin, I. (2001). Predictors and correlates of adaptive functioning in children with developmental disorders. *Journal of Autism and Developmental Disorders*, 31, 219–230.
- Paul, R., Miles, S., Cicchetti, D., Sparrow, S., Klin, A., Volkmar, F., Coffin, M., & Booker, S. (2004). Adaptive behavior in autism and pervasive developmental disorder- not otherwise specified: Microanalysis of scores on the Vineland adaptive behavior scales. *Journal of Autism and Developmental Disorders*, 34(2), 223.
- Perry, A., Flanagan, H., Geier, J. D., & Freeman, N. L. (2009). Brief report: The Vineland adaptive behavior scales in young children with autism spectrum disorders at different cognitive levels. *Journal of Autism and Developmental Disorders*, 39, 1066–1078.
- Roberts, C., McCoy, M., Reidy, D., & Crucitti, F. (1993). A comparison of methods of assessing adaptive behavior in pre-school children with developmental disabilities. *Australia & New Zealand Journal of Developmental Disabilities*, 18(4), 261–272.
- Sparrow, S. S., & Cicchetti, D. V. (1984). The behavior inventory for rating development (BIRD): Assessment of reliability and factorial validity. *Applied Research in Mental Retardation*, 5(2), 219–231.
- Sparrow, S. S., Balla, D., & Cicchetti, D. (1984). *Vineland adaptive behavior scales (expanded form)*. Circle Pines: American Guidance Service.
- Sparrow, S. S., Cicchetti, D., & Balla, D. (2005). *A revision of the Vineland adaptive behavior scales: Survey/care-giver form*. Circle Pines: American Guidance Service.
- Villa, S., Micheli, E., Villa, L., Pastore, V., Crippa, A., & Molteni, M. (2010). Further empirical data on the psychoeducational profile- revised (PEP-R): Reliability and validation with the Vineland adaptive behavior scales. *Journal of Autism and Developmental Disorders*, 40, 334–341.

- Volkmar, F. (2003). Adaptive skills. *Journal of Autism and Developmental Disorders*, 33(1), 3.
- Volkmar, F., Sparrow, S., Goudreau, D., & Cicchetti, D. (1987). Social deficits in autism: An operational approach using the Vineland adaptive behavior scales. *Journal of the American Academy of Child & Adolescent Psychiatry*, 26(2), 156–161.
- Volkmar, F., Carter, A., Sparrow, S., & Cicchetti, D. (1993). Quantifying social development in autism. *Journal of the American Academy of Child Psychiatry*, 32(3), 627–632.

ADHD Rating Scale

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A

Adaptive Skills

- ▶ [Assessment of Functional Living Skills \(AFLS\)](#)

Synonyms

ADHD=Attention deficit/hyperactivity disorder;
ASD=Autism spectrum disorder

ADD

- ▶ [Attention Deficit/Hyperactivity Disorder](#)

Description

The ADHD rating scale is an 18-question informant report that screens for symptoms of ADHD in children and teenagers from 5 to 17 years of age (DuPaul et al. 1998, 2016a). This questionnaire has four forms that are separated by setting and age group. There is one form for caregivers (parents and legal guardians) to complete for children (ages 5–10) and another for adolescents (ages 11–17), and there are parallel age forms for teachers. Each question on the ADHD rating scale asks about the presence and frequency of ADHD symptoms. Nine items ask about behaviors related to inattention (“easily distracted”), and nine questions about hyperactive and impulsive behaviors (“interrupts others”). Frequency is rated on a four-point scale from “never” to “sometimes” to “often” to “very often.” The ADHD rating scale, fifth edition, incorporates questions about how these symptoms cause problems at home or school in six areas: (1) getting along with family members/school professionals, (2) getting along with other children, (3) completing or returning homework, (4) performing academically in school, (5) controlling behavior in school, and (6) feeling good about himself/herself. The ADHD rating scale provides symptom counts and percentile scores relative to a community sample of parents

Adderall

- ▶ [D-Amphetamine](#)
- ▶ [Dexedrine](#)
- ▶ [Dextroamphetamine](#)

Adding

- ▶ [Reinforcement](#)

Addison-Schilder Disease

- ▶ [Adrenoleukodystrophy](#)

ADHD

- ▶ [Attention Deficit/Hyperactivity Disorder](#)

and teachers that were recruited from all regions of the United States.

Historical Background

The ADHD rating scale has been used as an evaluation tool for ADHD for almost 20 years. The ADHD rating scale fourth edition was created with an explicit goal of matching parent and teacher reports of ADHD symptoms to the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition* (DSM-IV; American Psychiatric Association 1994) and the text revised version (DSM-IV-TR; American Psychiatric Association 2000). While the DSM-IV and DSM-IV-TR prohibited a co-occurring diagnosis of ADHD when a diagnosis of autism spectrum disorder (ASD) was made, scientific papers studied the treatment of ADHD symptoms in ASD (Aman et al. 2008; Handen et al. 2000; Posey et al. 2007; Research Units of Pediatric Psychopharmacology 2005), as well as the influence of ADHD symptoms on the clinical presentation of ASD (Corbett and Constantine 2006; Corbett et al. 2009; Gadow et al. 2006; Yerys et al. 2009, 2011) using the ADHD rating scale and other comparable measures. The ADHD rating scale fifth edition (DuPaul et al. 2016a) integrated additional questions about impairment due to increased recognition that both symptom and symptom-related impairments are critical for making a diagnosis (Power et al. 2015).

Psychometric Data

The ADHD rating scale – fifth edition – has excellent reliability and validity metrics (DuPaul et al. 2016a,b). The normative data for the ADHD rating scale fifth edition was collected on 2079 children from 2079 parents and guardians and 2140 students from 1070 teachers (samples did not overlap). Parent and guardian incomes ranged from <\$5000 to \$175,000 or more per year; 64.1% had White non-Hispanic backgrounds, lived in both metropolitan (86.4%) and non-metropolitan areas (13.6%). Teachers included general (83.3%) and special education (16.4%)

teachers who were predominantly White non-Hispanic (87.3%) and reported a mean of 17.88 years of teaching experience (DuPaul et al. 2016b). The rating scale was shown to have excellent reliability for the inattention and hyperactivity/impulsivity domains across age (alphas >0.89), child's and rater's gender (alphas >0.90), ethnicity (alphas >0.90), and assessment language (English vs. Spanish; alphas >0.88). There was acceptable test-retest reliability over a 6-week period for parent ratings on child and adolescent forms for inattention ($r = 0.80$ and 0.70 , respectively) and hyperactivity/impulsivity ($r = 0.83$ and 0.61 , respectively). Test-retest reliability was also acceptable over a 6-week period for teacher ratings on child and adolescent forms for inattention ($r = 0.91$ and 0.85 , respectively) and hyperactivity/impulsivity ($r = 0.90$ and 0.77 , respectively). The impairment ratings introduced in the fifth edition showed better test-retest reliability for both raters in the child form (correlations range from 0.62 to 0.85) than in adolescents (correlations range from -0.06 to 0.81). Criterion validity was also assessed in comparison to the Connors rating scales (Connors 2008), and validity coefficients were acceptable (correlations ranged from 0.81 to 0.89). The ADHD rating scale – fifth edition – also showed excellent factorial validity with the 2-factor structure (inattention and hyperactivity/impulsivity) being the optimum structure. All critical goodness-of-fit statistics were met (comparative fit index and Tucker-Lewis index > 0.91 and root mean square error of approximation < 0.08). Across both rater groups, the factors were shown to be generally invariant to rater gender, rater age, child gender, and child age, though it should be noted that there was some slight strain on the factor structure within African American male students as well as teacher ratings by age. The delta comparative fit index showed a change equal to or less than 0.002 , which suggests this strain is statistically but not clinically significant. This normative study explicitly excluded children with known neurodevelopmental disorders and cognitive impairments, including autism spectrum disorder (ASD); thus, this sample is highly unlikely to represent the psychometric properties in children and adolescents with ASD.

To date, one study evaluated the psychometric properties of the ADHD rating scale fourth edition in children and adolescents with ASD (Yerys et al. 2017). This study demonstrated strong convergent validity in parent and guardian ratings with an informant report of executive function that includes scales on inhibition (i.e., hyperactivity/impulsivity behaviors) and working memory (inattention, forgetfulness, distractibility behaviors) and showed known age-related changes in symptom severity. However, the ADHD rating scale fourth edition demonstrated weak factorial validity for both rater types, suggesting that several items on the ADHD rating scale may require revision in order to better separate ADHD symptoms in children and adolescents with ASD. A major limitation of this study is that it was conducted with a sample of convenience – families willing to travel to a research center to participate in studies – rather than a community-based sample that matches the demographics of the ASD population in the United States.

Clinical Uses

The ADHD rating scale fifth edition can be used to screen for an ADHD diagnosis. Mental and behavioral health specialists with adequate training in psychological and psychiatric assessment can interpret scores generated from the ADHD rating scale. When used in isolation, the ADHD rating scale fifth edition can be used to identify children at risk for an ADHD diagnosis, but a valid diagnosis of ADHD requires follow-up with either an unstructured or structured developmental and psychiatric interview with caregivers to confirm that the symptoms are not better explained by other diagnoses and are truly impairing the individual's functioning in two settings. Some clinicians may also choose to include continuous performance-based measures, like the Conners' Continuous Performance Test. However, it is important to note that concerns have been raised about the ecological validity of continuous performance tests and that research has shown minor incremental utility of these tests when combined with psychiatric interviews and checklists. While the debate regarding the utility

of continuous performance tests continues (Berger et al. 2017), clinicians continue to rely upon the robust psychometrics of measures like the ADHD rating scale.

See Also

- Attention Deficit/Hyperactivity Disorder
- Behavior Assessment System for Children, 2nd Edition
- Comorbidity
- Conners' Continuous Performance Test
- Conners' Parent Rating Scale
- Conners' Teacher Rating Scale
- Executive Function (EF)

References and Reading

- Aman, M. G., Farmer, C. A., Hollway, J., & Arnold, L. E. (2008). Treatment of inattention, overactivity, and impulsiveness in autism spectrum disorders. *Child and Adolescent Psychiatric Clinics of North America*, 17(4), 713–738. vii. <https://doi.org/10.1016/j.jchc.2008.06.009>.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders DSM-IV fourth edition* (4th ed.). Arlington: American Psychiatric Publishing.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders DSM-IV-TR fourth edition* (4th ed.). Arlington: American Psychiatric Publishing.
- Berger, I., Slobodin, O., & Cassuto, H. (2017). Usefulness and validity of continuous performance tests in the diagnosis of attention-deficit hyperactivity disorder children. *Archives of Clinical Neuropsychology: The Official Journal of the National Academy of Neuropsychologists*, 32(1), 81–93. <https://doi.org/10.1093/arclin/acw101>.
- Conners, C. K. (2008). *Conners* (3rd ed.). San Antonio: Pearson Assessments.
- Corbett, B. A., & Constantine, L. J. (2006). Autism and attention deficit hyperactivity disorder: Assessing attention and response control with the integrated visual and auditory continuous performance test. *Child Neuropsychology*, 12(4/5), 335–348. <https://doi.org/10.1080/09297040500350938>.
- Corbett, B. A., Constantine, L. J., Hendren, R., Rocke, D., & Ozonoff, S. (2009). Examining executive functioning in children with autism spectrum disorder, attention deficit hyperactivity disorder and typical development. *Psychiatry Research*, 166(2–3), 210–222. <https://doi.org/10.1016/j.psychres.2008.02.005>.

- DuPaul, G. J., Power, T. J., Anastopoulos, A. D., & Reid, R. (1998). *ADHD rating scale-IV: Checklists, norms, and clinical interpretation*. New York: Guilford Press.
- DuPaul, G. J., Power, T. J., Anastopoulos, A. D., & Reid, R. (2016a). *ADHD rating scale-5 for children and adolescents: Checklists, norms, and clinical interpretation*. New York: Guilford Press.
- DuPaul, G. J., Reid, R., Anastopoulos, A. D., Lambert, M. C., Watkins, M. W., & Power, T. J. (2016b). Parent and teacher ratings of attention-deficit/hyperactivity disorder symptoms: Factor structure and normative data. *Psychological Assessment*, 28(2), 214–225. <https://doi.org/10.1037/pas0000166>.
- Gadow, K. D., DeVincent, C. J., & Pomeroy, J. (2006). ADHD symptom subtypes in children with pervasive developmental disorder. *Journal of Autism and Developmental Disorders*, 36(2), 271–283. <https://doi.org/10.1007/s10803-005-0060-3>.
- Handen, B. L., Johnson, C. R., & Lubetsky, M. (2000). Efficacy of methylphenidate among children with autism and symptoms of attention-deficit hyperactivity disorder. *Journal of Autism and Developmental Disorders*, 30(3), 245–255.
- Posey, D. J., Aman, M. G., McCracken, J. T., Scahill, L., Tierney, E., Arnold, L. E., et al. (2007). Positive effects of methylphenidate on inattention and hyperactivity in pervasive developmental disorders: An analysis of secondary measures. *Biological Psychiatry*, 61(4), 538–544. <https://doi.org/10.1016/j.biopsych.2006.09.028>.
- Power, T. J., Watkins, M. W., Anastopoulos, A. D., Reid, R., Lambert, M. C., & DuPaul, G. J. (2015). Multi-informant assessment of ADHD symptom-related impairments among children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, 1–14. <https://doi.org/10.1080/15374416.2015.1079781>.
- Research Units of Pediatric Psychopharmacology. (2005). Randomized, controlled, crossover trial of methylphenidate in pervasive developmental disorders with hyperactivity. *Archives of General Psychiatry*, 62(11), 1266–1274. <https://doi.org/10.1001/archpsyc.62.11.1266>.
- Yerys, B. E., Wallace, G. L., Sokoloff, J. L., Shook, D. A., James, J. D., & Kenworthy, L. (2009). Attention deficit/hyperactivity disorder symptoms moderate cognition and behavior in children with autism spectrum disorders. *Autism Research*, 2(6), 322–333. <https://doi.org/10.1002/aur.103>.
- Yerys, B. E., Wallace, G. L., Jankowski, K. F., Bollich, A., & Kenworthy, L. (2011). Impaired consonant trigrams test (CTT) performance relates to everyday working memory difficulties in children with autism Spectrum disorders. *Child Neuropsychology*, 17(4), 391–399. <https://doi.org/10.1080/09297049.2010.547462>.
- Yerys, B. E., Nissley-Tsiopinis, J., de Marchena, A., Watkins, M. W., Antezana, L., Power, T. J., & Schultz, R. T. (2017). Evaluation of the ADHD rating scale in youth with autism. *Journal of Autism and Developmental Disorders*, 47(1), 90–100. <https://doi.org/10.1007/s10803-016-2933-z>.

ADHD=Attention Deficit/Hyperactivity Disorder

► ADHD Rating Scale

Adipocytokine

► Plasma Adiponectin and Autism Spectrum Disorder

ADI-R

► Autism Diagnostic Interview-Revised

Adjustment

► Reasonable Accommodation

Admission, Review, and Dismissal Committee (ARD Committee)

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Synonyms

ARD committee

Definition

The Individuals with Disabilities Education Act (IDEA) is a federal law that mandates the availability of a free appropriate public education (FAPE) for all eligible children with disabilities. IDEA

defines “disability” as a person “(1) with mental retardation, hearing impairments . . . speech or language impairments, visual impairments . . . serious emotional disturbance . . . orthopedic impairments, autism, traumatic brain injury, other health impairments, or specific learning disabilities . . . (2) who needs special education and related services because of his or her disability or disabilities” (IDEA § 802, emphasis supplied). Thus, children with ASD are eligible for IDEA-related services.

IDEA Part B, Assistance for Education of All Children with Disabilities, mandates educational services for children from ages three 3 to 21. The educational requirements of Part B include an Individualized Education Program (IEP) for each child and demand the placement of each child in the least restrictive environment (LRE) possible.

An “appropriate” education must address a child’s specific educational needs. Determining what is appropriate entails several steps. The responsible state actor must conduct an individualized assessment to ascertain a student’s strengths and weaknesses. Next, an IEP Team, comprising representative of the school district, a teacher, the child’s parents, and if appropriate, the child, must identify appropriate goals and objectives for the student and construct an IEP designed to aid the student in meeting the goals and objectives. Finally, the IEP Team is charged with identifying the aids and services necessary for the child to succeed in the IEP.

States have discretion regarding the title assigned to the IEP Team. The Texas regulatory framework denotes an IEP Team “The Admission, Review, and Dismissal Committee.” Like any IEP Team, and ARD is charged with determining eligibility for special services (“admission”), conducting periodic reviews of IEPs (“review”), and determining the appropriateness of any disciplinary actions (“dismissal”).

IDEA prohibits students from being punished for actions caused by their disabilities. The IEP Team/ARD must review any proposed disciplinary actions to determine whether the targeted behavior was a manifestation of the student’s disability. If the IEP Team/ARD determines that the behavior was not a manifestation of the disability, the school may impose the sanctions that it would

impose for the same behavior committed by a student without disability. Those sanctions may include suspension or expulsion. Because IDEA mandates a free, appropriate, public education, educational services must be offered during the suspension or expulsion.

See Also

► [Individual Education Plan](#)

References and Reading

- 19 Texas Administrative Code §89.1050. The Admission, Review, and Dismissal (ARD) Committee 34 CFR §300.523 (2011).
- Holland, C. D. (2010). Autism, insurance, and the IDEA: Providing a comprehensive legal framework. *Cornell Law Review*, 95, 1253–1282.
- IDEA Regulations, § 300.8 Child with a disability (2010). Individuals with Disabilities Education Act, §§ 614(d)(1) (B), 615(k)(4), 20 USC §§ 1414 & 1415 (2011).

Adolescents with Autism Spectrum Disorder (ASD) Spontaneously Attending to Real-World Scenes: Use of a Change Blindness Paradigm

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Synonyms

[Change detection](#)

Definition

Change blindness is a perceptual phenomenon that occurs when a change in a visual stimulus is

introduced and the observer does not notice it (Rensink 2002). A classically used paradigm presents flickering stimuli made of repeated sequences of a picture followed by a “masking” stimulus (e.g., blank screen), which is followed by the initial picture with a change. Our sensory system is able to automatically detect change between pictures when they are immediately contiguous, but detection becomes more effortful when they are separated by a mask for an interval exceeding the temporal limits of visible persistence (Shore et al. 2006). Intervals of more than 100 ms render detection challenging, even when changes are large. Due to the mask, which hinders automatic visual change detection processes, orientation of visual attention is guided by controlled mechanisms that reveal the way we prioritize information that enters working memory (Rensink 2002). Indeed, in such circumstances, we need to attend selectively to the most important items in our environment. The detection of change depends on the degree of interest for the object that changes. Detection of change is more likely for parts of the scene that are of central interest rather than of marginal interest. Thus, change blindness is an indicator of where and to which items and features attention is preferentially directed in the presentation of a visual stimulus. Change blindness is assessed by measuring the time taken before noticing the change and the errors in identifying the change and has been extensively used to investigate change detection in natural visual scenes relevant to real-world experience (Rensink 2002).

Though not a diagnostic feature, attentional atypicalities are often found among individuals with autism spectrum disorder (ASD). The literature is mixed, presenting in some instances superior abilities while in other performances are lower. Studies have shown that individuals with ASD are quicker or more successful than typically developing (TD) control participants at various visual-attentional tasks (for a review, see Kaldy et al. 2016), the overall consensus being that across development and symptom severity individuals with ASD outperform controls on visual search. Change blindness relies on processes that are closely related to those involved in visual

search tasks. It is therefore highly relevant for investigating visual attention in ASD.

Change blindness paradigms have been used to explore visual-attentional abilities in ASD with mixed results. In a number of studies, people with autism showed an enhanced effect of blindness to change, while it was attenuated in others (Ames and Fletcher-Watson 2010). These contradictory findings may stem from the interference of impairments in processing speed when performing tasks that otherwise reveal superior visualization skills in ASD (Hochhauser et al. 2018). In addition, developmental transformations may possibly modulate the balance between executive and perceptive abilities, with recent research suggesting that processing speed impairments affect performances in change blindness even in adolescents (Hochhauser et al. 2018).

See Also

Ploog, B. O. (2013). Selective attention. *Encyclopedia of autism spectrum disorders*, 2700–2707.

References and Reading

- Ames, C., & Fletcher-Watson, S. (2010). A review of methods in the study of attention in autism. *Developmental Review*, 30(1), 52–73. <https://doi.org/10.1016/j.dr.2009.12.003>.
- Hochhauser, M., Aran, A., & Grynspan, O. (2018). How adolescents with autism Spectrum disorder (ASD) spontaneously attend to real-world scenes: Use of a change blindness paradigm. *Journal of Autism and Developmental Disorders*, 48(2), 502–510.
- Kaldy, Z., Giserman, I., Carter, A. S., & Blaser, E. (2016). The mechanisms underlying the ASD advantage in visual search. *Journal of Autism and Developmental Disorders*, 46(5), 1513–1527.
- Rensink, R. A. (2002). Change detection. *Annual Review of Psychology*, 53(1), 245–277.
- Shore, D. I., Burack, J. A., Miller, D., Joseph, S., & Enns, J. T. (2006). The development of change detection. *Developmental Science*, 9(5), 490–497.

ADOS

- [Autism Diagnostic Observation Schedule](#)

ADOS-T

- [Autism Diagnostic Observation Schedule \(ADOS\): Toddler Module](#)

Adrenaline

- [Epinephrine](#)

Adrenoleukodystrophy

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Synonyms

[Addison-Schilder disease](#); [ALD](#); [Siemerling-Creutzfeldt disease](#)

Definition

This rare genetic condition is one of a group of disorders termed the leukodystrophies in which myelin (the sheath surrounding nerve cell axons) is damaged. The condition is associated as well with severe damage both to the brain and peripheral nervous system as well as to the adrenal glands. Associated problems can include seizures, movement problems, and loss of function in many areas. Although onset in infancy and adulthood is possible, the onset is usually during childhood, and there may be some confusion early on with other conditions like childhood disintegrative disorder. An adolescent onset type is observed, frequently in males, with more prominent involvement of the spinal cord.

The onset of the condition can be characterized by visual or auditory problems, motor and motor

coordination issues, seizures, and increased behavioral difficulties. Characteristic laboratory findings and MRI findings are observed. A genetic test is available. The prognosis is poor with death after a period of some years of illness. Some dietary interventions are available, and new therapeutic approaches are being investigated.

See Also

- [Childhood Disintegrative Disorder](#)

References and Reading

- Corbett, J., & Harris, R. (1977). Progressive disintegrative psychosis of childhood. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, 18(3), 211–219.
- Darby, J. K. (1976). Neuropathologic aspects of psychosis in children. *Journal of Autism & Childhood Schizophrenia*, 6(4), 339–352.
- Volkmar, F. R., Koenig, K., & State, M. (2005). Childhood disintegrative disorder. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of Autism and pervasive developmental disorders* (Vol. 1, pp. 70–78). Hoboken: Wiley.

Adult Follow-Up Studies

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Definition

Predicting outcome and planning for adult service needs for children with Autism Spectrum Disorders (ASDs) is limited by gaps in current knowledge. The best quality information about autism in adulthood comes from a few population-based longitudinal studies that estimate the full picture of outcomes. However, changes in diagnostic criteria in the 1990s from a comparatively narrow

definition to the broader current criteria for ASD means that studies of adults originally diagnosed as children with historical criteria have limited application to later generations. In addition, longitudinal research that depends on information from aging caregivers has inherent challenges. For example, recall of symptoms from earlier life may be compromised by memory problems and health problems of the aging informant. An alternative research design using cross-sectional samples of adults diagnosed with current, broader criteria can provide data relevant to the future of children being diagnosed today. Such cross-sectional studies are useful adjuncts to population-based, longitudinal research, as they give a more comprehensive understanding of ASD in adulthood and can bring focus to specific issues. For example, the prevalence and variety of behaviors that lead to encounters with law enforcement have been described by Allen et al. (2008).

Two useful prognostic factors for adult outcome in ASD are childhood intellectual ability and onset of communicative speech. Like other people with intellectual disabilities (ID), people with ASD and ID generally achieve a limited range of independence and “success” in adult life as defined in developed Western societies inclusive of gainful employment, a household independent of their parents, and a circle of reciprocal friendships and romantic relationships. Those with average-range intellectual abilities (i.e., ≥ 70) have widely varying adult outcomes. Several longitudinal studies have demonstrated that communicative phrase speech before age 6 and an average-range childhood intelligence quotient (IQ) are necessary for a chance at adult independence but in no way guarantee it (Billstedt et al. 2005; Farley et al. 2009; Howlin et al. 2004; Kobayashi et al. 1992). When assessed in adulthood, barriers to independence in people with ASD and average-range intellectual abilities appear to include co-occurring psychiatric conditions, difficulty with initiation of goal-oriented activities, and poor social skills. There may also be specific genetic variations, developmental processes, educational opportunities, ecological factors, and specialized adult supports that influence levels of independence in adulthood.

Current Knowledge

Natural Course

While ASD is a lifetime diagnosis, several longitudinal studies have shown improvements in autistic symptoms over the life span (Billstedt et al. 2005; Cederlund et al. 2008; Piven et al. 1996; Rumsey et al. 1985; Seltzer et al. 2003). The trend is toward improvement in symptom severity in participants as a group, with the greatest amount of behavioral improvement in individuals who had the highest IQs and the least severe symptom presentation at the initial evaluation. These studies also show that a small proportion of affected individuals no longer meet full diagnostic criteria in adulthood. Importantly, most of these individuals retain subtle impairments that continue to present daily challenges to fully independent functioning.

There also appears to be a small subgroup that experiences significant deterioration in cognitive or behavioral functioning in adolescence (Ballaban-Gil et al. 1996; Eaves and Ho 2008; Kobayashi et al. 1992; Venter et al. 1992). Causes for this deterioration are unknown as yet, but appear unrelated to adolescent seizure-onset that occurs in some individuals with ASD.

Mortality

Studies of mortality in autism have identified a higher rate of mortality for populations with ASD than in the general population, owing largely to complications related to epilepsy and other medical conditions and to accidental deaths that may be associated with ID. Standardized mortality ratios (i.e., the ratio of observed deaths in a specific sample to expected mortality in the general population matched on variables such as age, gender, and length of follow-up period) range from 1.9 to 2.4, approximately twice the expected rate for the general population (Isager et al. 1999; Pickett et al. 2006; Shavelle et al. 2001). Females have had higher mortality rates than males in studied populations, probably associated with a higher rate of ID.

Selected Longitudinal Outcome Studies

A number of authors have categorized outcomes of adults with AD using broad social and

educational or occupational criteria (Howlin et al. 2004). Outcome classifications usually include five nodes and range from Very Poor (i.e., the person cannot function independently in any way) to Very Good (i.e., achieving great independence, having friends and a job). Findings from outcome studies are quite disparate, in spite of considerable similarities between outcome criteria and samples. A consistent finding from published outcome studies is that outcome for a majority (approximately 60%) of individuals with ASD was Fair, Poor, or Very Poor (Billstedt et al. 2005; Eaves and Ho 2008; Farley et al. 2009; Howlin et al. 2004).

Gillberg and Steffenburg (1987) studied outcome for a population-based sample of 23 people with ASD. As children, one-third obtained IQ scores in the mildly mentally retarded range and 26% achieved scores in the normal or near normal ranges. Eight (35%) had communicative speech at age 6. These 23 participants were aged 16–23 years at the time of the follow-up. One person (4% of the sample) obtained a “Good” outcome. Thirty-five percent experienced the “Fair, but restricted outcome.” (i.e., characteristics of “poor” outcome status, but accepted by and included in some social community). Thirteen percent had a “Fair” outcome, and 44% had “Poor” or “Very Poor” outcomes. Childhood IQ and use of communicative speech at age 6 were useful predictors of outcome status. Epileptic seizures were present in one-third of the population, often associated with severe mental retardation and pubertal symptom aggravation.

Kobayashi et al. (1992) conducted a follow-up investigation of 201 adults identified with ASD in childhood through clinical services in Japan. Four of the people had died. The mean age for the remaining 197 young adults was 21 years, 8 months ($S = 3.6$). About one-fourth of the sample had an IQ score of 70 or better at age 6, and about 20% were able to speak without echolalia at that age. An additional 31% used communicative language at age 6 but also used echolalic speech. Forty percent of the sample began school in a general education class, but only 27% remained in general education at the age of 12. At follow-up, 43 (21%) were employed and 11 (6%) were enrolled in higher

education or vocational training programs. Outcome adjustment for 47% was “Good” or “Very Good,” was “Fair” for 32%, and was “Poor” or “Very Poor” for 46%. Childhood IQ was the only strong predictor of outcome in this investigation. Although there were similarities between the sample in this study and others reported, the outcome for these participants was strikingly better, overall. The authors provided some possible explanations including the socio-demographic factors in Japan, advances in public education standards for people with disabilities, intensive intervention histories, and a high proportion of people with ASD and average-range IQ scores at baseline.

Howlin et al. (2004) studied adult outcome for 68 people with ASD who also had a childhood nonverbal IQ score of 50 or better. The mean age at the initial evaluation was 7.24 ($SD = 3.10$) and at follow-up was 29.33 ($SD = 7.97$). Nonverbal IQ scores averaged 80.21 ($SD = 19.28$). At follow-up, the average nonverbal IQ was 75 ($SD = 21.52$). Almost all of the subjects were known to have attended compulsory schooling; however, only 22% left school having achieved formal qualifications. At the time of the follow-up investigation, 23 people were employed. Eight worked in regular, independent jobs; 1 was self-employed as an artist but was unable to earn a living wage; and 14 worked in sheltered or supported employment. Twenty-seven people were occupied in general work/leisure programs at day centers for adults with disabilities. Outcome adjustment ratings for the sample were that 22% had “Good” or “Very Good” outcomes, 19% had “Fair” outcomes, and 58% had “Poor” or “Very Poor” outcomes. Analyses of the assessment results revealed that childhood IQ was a useful predictor of adult adjustment in that those with childhood nonverbal IQ scores of 70 or more were more likely to do well than those with scores below 70. Furthermore, a score of 100 or better did not increase the likelihood that a person would do well in adulthood. For those who were capable of completing a childhood verbal IQ measure, the combination of verbal and nonverbal IQ scores in childhood provided a more precise indication of outcome classification, with scores above 70 in both domains yielding the greatest likelihood of a

“Fair” outcome or better. Specifically, among those with childhood nonverbal IQ scores of 70 or more, 7 had a “Very Good” outcome, 7 had a “Good” outcome, 10 obtained a “Fair” outcome, and 20 had “poor” or “Very Poor” outcomes. Language level at age 5 was useful in predicting overall outcome and residential status but none of the other outcome variables studied demonstrated predictive utility.

Eaves and Ho (2008) followed 48 individuals with ASDs from childhood (mean age = 6.8) to adulthood (mean age = 24) in Canada. Fifty-seven percent of this sample had Autistic Disorder, while the remainder had less severe variants of ASD. Eight of the participants had a childhood IQ score above 70. All participants received special education support during their compulsory schooling years, and 30% engaged in some kind of postsecondary educational program. Overall outcome adjustment ratings were that 21% had “Good” or “Very Good” outcomes, 32% had “fair” outcomes, and 46% had “poor” outcomes. No participants fell within the “Very Poor” outcome categorization. Sixty percent of the sample resided at home with their parents, 19% lived in group homes, and 13% lived in foster care. Almost 80% received a government disability pension and used the services of social workers. In this sample, childhood verbal IQ was most predictive of outcome status. However, the proportion of individuals who were capable of completing an assessment of verbal IQ was not reported.

Also in Cederlund et al. 2008, Cederlund et al. released their study of outcome for 70 adults with autism and 70 adults with Asperger Disorder, after 5 or more years elapsed from original diagnosis. This research team used the same outcome categorization scheme as Gillberg and Steffenburg (1987), with categories of “Good,” “Fair,” “Restricted,” “Poor,” and “Very Poor.” Twenty-seven percent ($n = 19$) of this sample obtained an outcome categorization of “Good,” and 47% ($n = 33$) were categorized as having a “Fair” outcome. Sixteen people, or 23%, obtained “Restricted” outcome status, and two people, or 3%, fell within the “Poor” category. There were no participants with “Very poor” outcome ratings.

Farley et al. (2009) studied 41 adults who had been identified through a population-based study of ASD in Utah in the 1980s. All of these individuals had previous IQ scores of 70 or greater. Mean age at the first assessment was 7.2 years ($SD = 4.1$) and in adulthood was 32.5 years ($SD = 5.7$). Outcome adjustment was somewhat better for this sample than previous samples, with 48% in the “Very Good” and “Good” categories, 34% in the “Fair” category, and 17% in the “Poor” category. No participants fit within the “Very Poor” category of outcome categorization. Six participants did not meet diagnostic criteria for current ASD using gold standard diagnostic procedures, but five of these still retained significant social difficulties reported by themselves or significant others. Half were employed on a full- or part-time basis, and 39% had attended some kind of formal postsecondary education. Over half of the sample (56%) continued to live with their parents, and almost 25% lived in supported living arrangements including a state residential center for people with significant disabilities. Almost 60% of the sample reported co-occurring psychiatric diagnoses. Reported chronic medical conditions were those commonly seen in the general population (e.g., seasonal allergies, gout, high blood pressure).

Cognitive Function

Evidence to date reflects uneven development of cognitive abilities across people with ASD. Initial evaluations during childhood often indicate better nonverbal than verbal abilities. However, many studies show evidence increases in verbal ability and decreases in nonverbal ability during adolescence and adulthood. Group results for individuals with ASD and average-range IQ scores demonstrate consistency in the distribution of subtest scores on Wechsler scales. However, some individuals who have relatively high IQs in childhood demonstrate significant increases in overall ability at follow-up (Gonzales et al. 1993). Disparities among findings may have several causes. Selection of tests at initial evaluation and follow-up for their appropriateness to the research question and participants’ behavior may influence results. Furthermore, tests may not be sufficiently

parallel for comparison, so that some of the variance is attributable to inequality across measures. Variation of tests from the initial evaluation to follow-up further obscures results since within-group variation on measures may be considerable (Howlin et al. 2004). Age at initial IQ also appears to be an important factor, with nonverbal abilities varying more among children initially tested before age 5 (Howlin et al. 2004).

Associated Co-Occurring Conditions

Many of the outcome studies concerning adults with AD provide information concerning co-occurring medical and psychiatric conditions. Few have analyzed the specific contributions these disorders make to restrictions in overall outcome (Danielsson et al. 2005). One of the clearest indicators of the presence of significant co-occurring psychiatric and medical diagnoses is the proportion of individuals who are prescribed anticonvulsant and psychotropic medications. Eaves and Ho (2008) reported that 40% of their sample was prescribed medication for behavioral difficulties. Similarly, 40% of the participants in the population-based study by Billstedt et al. (2005) were prescribed medication for psychiatric disorders, and 40% of the adolescents and adults in another study were prescribed psychotropic medications to control behavior (Ballaban-Gil et al. 1996). Thirty-seven percent of those studied by Farley et al. (2009) were described as taking prescription medications aimed at managing behavioral difficulties.

Epilepsy is a chronic condition involving recurring seizures and is more common in individuals with ASD than in the general population, with an average prevalence rate of 16.8% across epidemiological studies of ASD (Fombonne 1999). This disorder occurs more frequently in individuals with ASD and ID. The onset of seizures typically occurs early in childhood (i.e., before age 2) or in adolescence (Danielsson et al. 2005; Kobayashi et al. 1992). Seizures remit in a fraction of those afflicted (Danielsson et al. 2005). Kobayashi et al. (1992) reported that 19% of their sample, representing the full range of functioning within ASD, had epilepsy, and all took anti-epileptic medication. Nine percent of a sample of

adults with ASD and average-range IQ scores took antiepileptics (Howlin et al. 2004).

Affective disorders challenge a person's capacity to regulate mood and include depression, mania, and bipolar disorder. It is estimated that over 60% of people with AD suffer from a co-occurring affective disorder. In a study of 35 individuals with Asperger syndrome, Ghaziuddin et al. (1998) found that affective disorders were the most common type of psychiatric condition co-occurring in adults, affecting over half of their sample. Figures from outcome studies with adult samples range from 1% to 30% (Billstedt et al. 2005; Farley et al. 2009).

Results of several outcome studies demonstrate that anxiety disorders are present in a large proportion of adults with AD. Rumsey et al. (1985) determined that 50% of their sample was suffering from chronic, generalized anxiety, which they suggested could account for the attention difficulties observed in one-fifth of the sample. Another study of adults with ASD and average-range IQ scores concluded that 40% of their sample had OCD or chronic anxiety (Szatmari et al. 1989). Figures from other outcome studies are much smaller; however, these results may be confounded by the presence of ritualistic characteristics and hyperactivity commonly associated with ASD (Ghaziuddin et al. 1998).

Hyperactivity and short attention span are common in people with ASD. These have been most commonly noted in children, yet some adults present with behavioral characteristics of Attention Deficit-Hyperactivity Disorder (ADHD) as well (Ghaziuddin et al. 1998). Forty (33%) of the adults in the study by Billstedt et al. (2005) presented with hyperactivity.

Psychiatric conditions evident in a small number of people with ASD include tic disorders, psychotic features, and catatonia. Almost 20% of the sample examined by Billstedt et al. (2005) demonstrated tics and 10% of the adults studied by Eaves and Ho (2008) had Tourette's disorder. One of the 15 adults in another investigation presented with Tourette's disorder (Ghaziuddin et al. 1998). A small number of individuals with ASD genuinely have co-occurring psychotic

conditions. Eight percent of the sample in the study of adults with ASD conducted by Billstedt et al. (2005) and 38% of those examined by Szatmari et al. (1989) had characteristics of psychosis. Catatonia is another type of psychiatric disturbance that is rarely observed, but notable in ASD. One of the 15 adults studied by Patricia Howlin et al. (2000) had a sudden-onset catatonic episode during puberty. Billstedt et al. (2005) reported a much higher percentage (12%) in their sample of 120 adults.

While not psychiatric disorders in their own right, maladaptive behaviors are significant deviations from expected behavior for a person's developmental level. They are often disruptive and sometimes dangerous. Maladaptive behaviors are frequently observed in people with ASD of all levels of ability and developmental age. In general terms, maladaptive behaviors have been reported in up to 69% of adults with ASD with no overall difference in frequency between males and females (Ballaban-Gil et al. 1996; Eaves and Ho 2008). Maladaptive behaviors may be relatively infrequent in adults with ASD and average-range IQ scores, but odd or severe enough to preclude acceptance into general social settings over time (Rumsey et al. 1985). Self-injurious behaviors were reported to have occurred in 50% of the sample studied by Billstedt et al. (2005), and have been reported to be more common in females than in males (Ballaban-Gil et al. 1996). Difficulties with toileting and feeding appear to persist in lower functioning individuals, but difficulties with compulsive rituals may develop around these tasks in higher functioning adults as well. Aggression among adults is rarely designed to harm others, but property damage or harm to self may occur intermittently, sometimes in response to unimportant changes or problems in the environment (Rumsey et al. 1985).

Social Relationships

Few adults with ASD develop significant relationships outside of the family of origin in spite of common increases in interest in developing social relationships as individuals with AD age (Rumsey et al. 1985). Almost 75% of family members interviewed in the study by Eaves and Ho (2008)

reported that they enjoyed good to excellent relationships with their affected relative; however, only one-third of the sample of affected adults had one or more friendships outside of the family. Similar results have been found in other studies of adults with ASD (Howlin 2003; Howlin et al. 2004). Females have reportedly experienced greater success with peer relationships than males (Piven et al. 1996). Ten percent of adults in the study by Eaves and Ho (2008) had a romantic relationship at some time in the past, but none of the participants was romantically involved at the time of the investigation. Nineteen percent of the men with Asperger Disorder in the Cederlund et al. (2008) study and 3% of the men with Autistic Disorder were or had been in long-term romantic relationships. Thirty-two percent of those studied by Farley et al. (2009) had dated, and 20% were involved in a serious relationship at the time of the study. In general, very few adults with ASD have been reported to have successful, long-term romantic relationships (Howlin 2003; Howlin et al. 2004).

Education and Employment

Approximately, 15% of adults with ASD studied in outcome research attend postsecondary education programs (Ballaban-Gil et al. 1996; Farley et al. 2009; Kobayashi et al. 1992; Rumsey et al. 1985; Szatmari et al. 1989; Venter et al. 1992). In general, gainful employment for adults with ASD is rare, as is sheltered employment, occupying less than 40% of adults with AD (Howlin 2003; Howlin et al. 2004). While outcome studies of autism into adulthood conducted since 1992 reflect some steady improvements in employment rates, with 22–54% of participants reporting gainful employment on a full- or part-time basis (Ballaban-Gil et al. 1996; Farley et al. 2009; Howlin et al. 2004; Kobayashi et al. 1992; Venter et al. 1992), many of these individuals are underemployed based on their cognitive abilities and academic credentials.

Forensic Problems

Involvement with police officers and other law enforcement agents has been recognized as a major concern for parents of adolescents and

adults with ASD. A study of offending behavior in 33 individuals with Asperger Disorder (Allen et al. 2008) revealed that most engaged in violent or threatening behavior that was related to interpersonal problems including social or sexual rejection, bullying, or family conflict. Investigators have suggested offending behavior in ASD populations was likely to result from coercion by others, misinterpretation of social situations, or obsessional interests, while many with ASD may be protected by their tendency to adhere strictly to rules. Allen et al. (2008) found evidence of this insight in that the least common offenses identified among their population of offenders with Asperger Disorder were drug offenses, theft, fraud, sexual offending, and motor offenses. Cederlund et al. (2008) found that 10% ($n = 7$) of their sample with Asperger Disorder had been involved with law enforcement officers, but the remainder was described as very law-abiding. None of the individuals in their lower functioning sample with autistic disorder had committed legal offenses. In the study by Farley et al. (2009), 29% of the sample was involved with law enforcement officers for infractions after childhood, but these were related exclusively to “suspicious” behaviors deriving from special interests, participants being coerced to engage in antisocial behavior by peers, and social misunderstandings.

Future Directions

The prognosis for a majority of adults with ASD, based on studies conducted to date, is guarded. Future studies are needed to further define the subtypes of ASD, and the factors that influence adult outcome. Studies of genetics, brain imaging, and responses to interventions are likely to yield important information.

See Also

- ▶ [Adulthood, Transition to](#)
- ▶ [Advocacy](#)
- ▶ [Americans with Disabilities Act](#)
- ▶ [Asperger Syndrome Follow-Up Studies](#)

- ▶ [Benhaven Residential Services](#)
- ▶ [Community Services](#)
- ▶ [Community-Integrated Residential Services for Adults with Autism](#)
- ▶ [Competitive Employment](#)
- ▶ [Comprehensive Transition Program](#)
- ▶ [Course of Development](#)
- ▶ [Employment](#)
- ▶ [Employment in Adult Life](#)
- ▶ [Employment Specialist](#)
- ▶ [Functional Life Skills](#)
- ▶ [Guardianship](#)
- ▶ [Independent Living](#)
- ▶ [Individualized Plan for Employment \(IPE\)](#)
- ▶ [Job Carving](#)
- ▶ [Job Coach](#)
- ▶ [Law Enforcement Agencies and Autism](#)
- ▶ [Legal Competency](#)
- ▶ [Living Arrangements in Adulthood](#)
- ▶ [Secure Employment](#)
- ▶ [Self-Advocacy](#)
- ▶ [Sexuality in Autism](#)
- ▶ [Sheltered Employment](#)
- ▶ [Sheltered Workshops](#)
- ▶ [Suicide Rates in Adults with Autism](#)
- ▶ [Supported Employment](#)
- ▶ [Transitional Living](#)
- ▶ [Transition Planning](#)
- ▶ [Travel Training](#)
- ▶ [Trust](#)
- ▶ [Violent/Criminal Behavior in Autism](#)
- ▶ [Vocational Evaluator](#)
- ▶ [Vocational Rehabilitation Act of 1973](#)
- ▶ [Vocational Training](#)

References and Reading

- Allen, D., Evans, C., Hider, A., Hawkins, S., Peckett, H., & Morgan, H. (2008). Offending behaviour in adults with Asperger syndrome. *Journal of Autism and Developmental Disorders*, 38(4), 748–758.
- Ballaban-Gil, K., Rapin, I., Tuchman, R., & Shinnar, S. (1996). Longitudinal examination of the behavioral, language, and social changes in a population of adolescents and young adults with autistic disorder. *Pediatric Neurology*, 15(3), 217–223.
- Billstedt, E., Gillberg, C., & Gillberg, C. (2005). Autism after adolescence: Population-based 13- to 22-year follow-up study of 120 individuals with autism

- diagnosed in childhood. *Journal of Autism and Developmental Disorders*, 35(3), 351–360. <https://doi.org/10.1007/s10803-005-3302-5>.
- Cederlund, M., Hagberg, B., Billstedt, E., Gillberg, I. C., & Gillberg, C. (2008). Asperger syndrome and autism: A comparative longitudinal follow-up study more than 5 years after original diagnosis. *Journal of Autism and Developmental Disorders*, 38(1), 72–85.
- Danielsson, S., Gillberg, I. C., Billstedt, E., Gillberg, C., & Olsson, I. (2005). Epilepsy in young adults with autism: A prospective population-based follow-up study of 120 individuals diagnosed in childhood. *Epilepsia*, 46(6), 918–923.
- Eaves, L. C., & Ho, H. (2008). Young adult outcome of autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(4), 739–747.
- Farley, M. A., McMahon, W. M., Fombonne, E., Jenson, W. R., Miller, J., Gardner, M., et al. (2009). Twenty-year outcome for individuals with autism and average or near-average cognitive abilities. *Autism Research*, 2(2), 109–118. <https://doi.org/10.1002/aur.69>.
- Fombonne, E. (1999). The epidemiology of autism: A review. *Psychological Medicine*, 29, 769–786.
- Ghaziuddin, M., Weidmer-Mikhail, E., & Ghaziuddin, N. (1998). Comorbidity of Asperger syndrome: A preliminary report. *Journal of Intellectual Disability Research*, 42(4), 279–283.
- Gillberg, C., & Steffenburg, S. (1987). Outcome and prognostic factors in infantile autism and similar conditions: A population-based study of 46 cases followed through puberty. *Journal of Autism and Developmental Disorders*, 17(2), 273–288.
- Gonzales, N. M., Murray, A., Shay, J., Campbell, M., & Small, A. M. (1993). Autistic children at follow-up: Change of diagnosis. *Psychopharmacology Bulletin*, 29(3), 353–358.
- Howlin, P. (2003). Outcome in high-functioning adults with autism with and without early language delays: Implications for the differentiation between autism and Asperger syndrome. *Journal of Autism and Developmental Disorders*, 33(1), 3–13.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45(2), 212–229.
- Isager, T., Mouridsen, S. E., & Rich, B. (1999). Mortality and causes of death in pervasive developmental disorders. *Autism*, 3(1), 7–16. <https://doi.org/10.1177/1362361399003001002>.
- Kobayashi, R., Murata, T., & Yoshinaga, K. (1992). A follow-up study of 201 children with autism in Kyushu and Yamaguchi areas, Japan. *Journal of Autism and Developmental Disorders*, 22(3), 395–411. <https://doi.org/10.1007/BF01048242>.
- Patricia Howlin, P., Mawhood, L., & Rutter, M. (2000). Autism and developmental receptive language disorder – A follow-up comparison in early adult life. II: Social, behavioural, and psychiatric outcomes. *Journal of Child Psychology and Psychiatry*, 41, 561–578.
- Pickett, J. A., Paculdo, D. R., Shavelle, R. M., & Strauss, D. J. (2006). 1998–2002 update on “Causes of death in Autism”. *Journal of Autism and Developmental Disorders*, 36(2), 287–288. <https://doi.org/10.1007/s10803-005-0066-x>.
- Piven, J., Harper, J., Palmer, P., & Arndt, S. (1996). Course of behavioral change in autism: A retrospective study of high-IQ adolescents and adults. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35(4), 523–529.
- Rumsey, J. M., Rapoport, J. L., & Sceery, W. R. (1985). Autistic children as adults: Psychiatric, social, and behavioral outcomes. *Journal of the American Academy of Child and Adolescent Psychiatry*, 24(4), 465–473.
- Seltzer, M. M., Krauss, M. W., Shattuck, P. T., Orsmond, G., Swe, A., & Lord, C. (2003). The symptoms of autism spectrum disorders in adolescence and adulthood. *Journal of Autism and Developmental Disorders*, 33(6), 565–581.
- Shavelle, R. M., Strauss, D. J., & Pickett, J. (2001). Causes of death in autism. *Journal of Autism and Developmental Disorders*, 31(6), 569–576.
- Szatmari, P., Bartolucci, G., Bremner, R., Bond, S., & Rich, S. (1989). A follow-up study of high-functioning autistic children. *Journal of Autism and Developmental Disorders*, 19(2), 213–225.
- Venter, A., Lord, C., & Schopler, E. (1992). A follow-up study of high-functioning autistic children. *Journal of Child Psychology and Psychiatry*, 33(3), 489–507. <https://doi.org/10.1111/j.1469-7610.1992.tb00887.x>.

Adult Repetitive Behaviors Questionnaire-2-Revised (RBQ-2A-R)

► Self-Report Autism Scales for Adults

Adult/Adolescent Sensory Profile (AASP)

► Glasgow Sensory Questionnaire (GSQ)

Adult-/Clinician-/Teacher-Directed Approaches

► Didactic Approaches

Adulthood, Transition to

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Definition

For the purposes of this entry, the transition to adulthood is defined as exiting the secondary school system, resulting in the termination of services received through the school system. High school “exit” is differentiated from “graduation,” as some individuals with Autism Spectrum Disorder (ASD) “graduate” with same-aged peers, but continue to receive secondary school services until age 22. We expect that these youth will be more affected by losing school services than by graduation. Thus, high school “exit” refers to the termination of school-based services.

Historical Background

The transition to adulthood for adolescents without disabilities has traditionally been identified as completing a number of developmental tasks of adulthood. These tasks have been well identified, stemming from Freud’s notion of “love and work” (cited by Hazan and Shaver 1990), and most often include leaving the parental home, finishing school and starting employment, and marrying and having children (Fussell and Furstenberg 2005). In recent years, however, the entry into adulthood for typically developing individuals has become much more ambiguous and prolonged and these markers are often achieved more gradually, and not necessarily in as fixed an order as in the past (Furstenberg et al. 2005). It is increasingly common, for example, for youth to move out of the parental home and into a university residence, but then move back into the parental home for a time after the completion of their

university studies. Additionally, it is often the case that men and women are living independently, have finished their schooling, and are engaged in full-time work activities but have not yet married or had children.

Arnett (2000) proposed the concept of emerging adulthood as a way to account for this heterogeneity in how individuals transition from adolescence to adulthood. The emerging adulthood period, Arnett (2000) explained, is distinct from adolescence and early adulthood because of its relative freedom from social roles and societal expectations. He suggested that the transition to adulthood is no longer best represented by demographic transitions, such as ending formal schooling, getting married, or having children. Instead, the criteria for the transition to adulthood are individualistic, encompassing concepts such as independence in decision making, being responsible for one’s own person, and financial independence (Arnett 2000).

When the adolescent in transition has ASD, the complexity implicit in defining the transition to adulthood is multiplied. Some developmental tasks of adulthood are obtained by most individuals with ASD, such as exiting school. Other tasks, such as moving out of the parental home or finding regular employment, are only achieved by a fraction of individuals with ASD, but these milestones can be modified to be attainable by many more (e.g., structured or semi-structured living arrangements, supported employment). Finally, some tasks are attained by few individuals with ASD, such as getting married or having children. The criteria proposed by the emerging adulthood literature are similarly complicated; many individuals with ASD may never have complete independence in decision making nor have financial independence from both their families and federal or state agencies.

Because of these difficulties in definition, many researchers have forgone theory-based ideas of transition and instead defined the transition period for those with intellectual and developmental disabilities using specific ages (such as ages 18–26 in Blacher 2001). Alternatively, our research has chosen high school exit as a key indicator of the transition to adulthood for two

reasons: (1) of all developmental tasks of adulthood, it is the most commonly attained by individuals with ASD; and (2) nearly all transition studies on individuals with intellectual and developmental disabilities (not ASD) center around high school exit.

In contrast to typically developing adolescents in the USA – who exit high school at a prescribed time (at the end of twelfth grade) – considerable variability exists in the age at which adolescents and young adults with ASD exit the school system. Some exit with their same-aged, nondisabled peers, while others take advantage of the Individuals with Disabilities Education Act (IDEA) and remain in secondary school until their 22nd birthday. Although it may be simplistic to only consider high school exit as a marker of the transition to adulthood, this milestone provides a focused lens through which to examine the research related to the transition to adulthood for people with ASD.

Although few studies have focused on high school exit for youth with ASD, there is considerable research among adults with ASD suggesting that they have difficulties integrating into adult society. Adults with ASD tend to live fairly dependent lives, are underemployed, with those who have employment often holding jobs that do not provide a living wage (for a review see Howlin 2005). The transition out of high school for youth with ASD (and other disabilities) has long been recognized by professionals and policy makers as an important turning point that sets the stage for later adult outcomes. Perhaps the greatest evidence of this is the existence of federal legislation mandating specific requirements for transition planning for youth with disabilities, found in the IDEA of 1997 and the Individuals with Disabilities Education Improvement Act (IDEIA) of 2004. These legislative landmarks mandate that a transition plan must be included in the Individualized Education Plan when a student is 16 years of age (although planning can start sooner) which facilitates “real-world” outcomes by focusing on improvement in education (postsecondary, vocational skills), adult services, independent living skills, and community participation. Furthermore, measurable goals must be

developed that take into account the student’s needs, strengths, interests, and preferences.

By examining the corpus of research on the transition to adulthood, it is clear that autism researchers have lagged behind policy makers and practitioners in recognizing the importance of this transition for youth with ASD. The few existing studies are summarized below.

Current Knowledge

The transition to adulthood is associated with a slowing of improvement of the autism behavioral phenotype. (Taylor and Seltzer 2010) examined change over nearly 10 years in autism symptoms and behavior problems for a community sample of over 240 youth with ASD. The vast majority of these youth exited high school over the study period, allowing us to test changes in symptoms and behaviors while youth were in high school, as well as whether leaving high school impacted that change. We found that all subscales of symptoms and behaviors were significantly improving while youth were in high school and that, in general, improvement significantly slowed down after youth with ASD exited the secondary school system. Although youth with ASD who did not have an intellectual disability (ID) had less severe symptoms and behavior problems than those who had ID as well as ASD throughout the study period, the slowing of improvement following high school exit was more pronounced for youth with ASD who did *not* have ID, relative to those who had a comorbid ID. Furthermore, youth with ASD whose families had lower incomes were more negatively impacted by high school exit relative to youth whose families had higher incomes.

Similar patterns were observed in follow-up analyses (Taylor and Seltzer 2011a), which examined the impact of exiting high school on changes in the mother-child relationship over a 7-year period. We found improvements in three indices of the mother-child relationship – mother-child positive affect, subjective burden, and warmth – while youth with ASD were in high school. After high school exit, however, that improvement stopped – even after controlling for concurrent

slowing of improvement in behavior problems. Once again, whether the youth with ASD had a comorbid ID significantly predicted change in maternal warmth; those without an ID were more negatively affected by high school exit relative to those with a comorbid ID. Further, the number of needed services that were currently not being received also predicted change in the mother-child relationship. There was greater slowing of improvement in mother-child positive affect for youth who had more unmet service needs, relative to those who had fewer unmet needs. In sum, these studies provide evidence of a disruption in phenotypic improvement and parent-child relations in the years following high school exit for youth with ASD.

Youth with ASD without an ID might be more negatively impacted by exiting high school because they have a difficult time finding appropriate vocational or educational activities. This hypothesis was supported in a study by (Taylor and Seltzer 2011b), who examined the post-secondary educational and vocational activities of young adults with ASD who had exited high school an average of 2 years previous to data collection. We found that nearly 25% of the young adults who had ASD without ID had no or minimal vocational/educational activities, and those without ID were three times more likely to have no day activities than youths with ASD who also had comorbid ID. This divergent pattern likely does not represent a lack of abilities on the part of the youth with ASD, but instead the inadequacy of the current service system to accommodate the needs of youth with ASD who do not have ID as they are transitioning to adulthood. Indeed, in this sample, only 18% of young adults without ID were getting some sort of employment or vocational services (e.g., supported employment, sheltered workshop) compared to 86% of young adults with ID. Thus, the lack of appropriate services and limited options for educational/vocational activities for youth with ASD without ID after high school exit may be responsible for the slowing of improvement observed during this time. Youth with ASD and a comorbid ID may be less affected as they more easily fit into the existing adult disability service system.

Limited services and opportunities after high school exit might also play a role in the greater negative impact of high school exit on youth with ASD whose families have lower incomes, relative to those whose families have higher income. A recent study by Shattuck et al. (2011) supports this hypothesis. Using a nationally representative sample, the authors found that nearly 40% of youth with ASD were receiving no services in the 2 years following their exit from high school. Furthermore, youth whose families had lower incomes were more likely to be without formal services relative to youth whose families had higher incomes. It appears then that youth with ASD whose families have fewer economic resources also receive fewer adult services once they exit high school and services are no longer mandated, which likely explains (at least in part) why the pattern of improvement in their behavior problems that was observed while they were in high school is more negatively impacted by exiting high school.

In sum, the small body of existing research focused on the transition to adulthood for youth with ASD suggests that it is a disruptive influence in the lives of these families, with the greatest disruption occurring for those who do not have ID, those whose families have fewer resources, as well as those who are underserved by the formal service system. In the following section we discuss the numerous directions for future research.

Future Directions

Although our knowledge of how youth with ASD and their families are impacted by the transition to adulthood is in its infancy, it is critical that we better understand the mutable factors associated with a positive transition. As previously mentioned, employment and vocational outcomes of adults with ASD have much room for improvement. Furthermore, adults with ASD seem to be at additional risk for poor outcomes relative to even adults with other types of developmental disabilities. Esbensen et al. (2010) found that adults with ASD had less optimal outcomes (as defined by less independence in their living arrangements, in

their vocational placements, and less social connectedness) relative to a matched group of adults with Down syndrome. It appears then that adults with ASD might be a particularly vulnerable group as they move out of high school and into adult life.

Future research should focus on the mutable factors that promote a successful transition to adulthood and optimal adult outcomes. So far, studies of risk factors for poor adult outcomes have focused on factors that are static and difficult to change. Adults with ASD who require substantial supports tend to have lower IQ scores, fewer functional abilities, and poor early language skills (Billstedt et al. 2007; Eaves and Ho 2008; Farley et al. 2009; Howlin et al. 2004; Howlin et al. 2000). But while knowing an individual's IQ and early language abilities helps predict adult outcomes, this information is less helpful in considering ways to improve outcomes. Malleable factors that impede positive outcomes or exacerbate negative outcomes may provide better avenues for intervention.

One promising factor is behavioral functioning, and specifically maladaptive behaviors. Maladaptive behaviors can be extremely disruptive for all adults with disabilities, including those with ASD. Taylor and Seltzer (2011b) found that young adults with ASD who had lower levels of maladaptive behaviors were more likely to be in college or working independently in the community in the years after high school exit. Those young adults with higher levels of maladaptive behaviors tended to either spend their time in sheltered settings (day activity programs, sheltered workshops) or to have no vocational activities. Maladaptive behaviors can be changed through both behavioral and pharmacological interventions (Aman et al. 2009; Matson et al. 2009; McCracken et al. 2002; Vismara and Rogers 2010), and thus constitute a promising factor that, if alleviated, could promote independence and employment among adults with ASD.

Environmental resources are another set of malleable factors that have virtually been ignored by researchers studying outcomes for adults with ASD. Not only are the quality and availability of formal services likely important in promoting a

positive transition to adulthood, but also the family environment. Family environments, characterized by high levels of criticism of the individual with ASD, predict significant increases in behavior problems (Greenberg et al. 2006); alternatively, supportive, warm family environments predict decreases in behavior problems for these adults (Smith et al. 2008). Environmental resources can be altered through advocacy for better disability-related services and psychoeducational intervention to improve positivity in the family environment (Bernhard et al. 2006), and thus are also promising avenues for future research focused on promoting a positive transition to adulthood for youth with ASD.

Finally, researchers should continue to consider what is meant by a "positive" transition to adulthood. Based on the current criteria for successful adult outcomes – living independently, working independently, and friendships – it is not difficult to come up with examples of young adults with ASD who appear to be transitioning "unsuccessfully," but in actuality may be doing quite well in adulthood. A more holistic view of the transition to adulthood would be garnered by including measures of life satisfaction, community engagement, sense of purpose, or even by judging outcomes based on individualistic goals for adult life. Measuring constructs broader than employment and living arrangements when examining an individual's transition success may also alleviate some of the bias against a successful transition for those young adults who have more functional limitations. Advocating the inclusion of measures of life satisfaction or purpose does not mean to imply that the difficulties faced by individuals with ASD in attaining community employment and independence are not concerning, only that it does not represent the entirety of the transition to adulthood.

See Also

- ▶ [Adult Follow-Up Studies](#)
- ▶ [Course of Development](#)
- ▶ [Employment](#)
- ▶ [Employment in Adult Life](#)

- ▶ Factors Affecting Outcomes
- ▶ Individual Education Plan
- ▶ Individualized Plan for Employment (IPE)
- ▶ Individualized Transition Plan (ITP)
- ▶ Individuals with Disabilities Education Act (IDEA)

References and Reading

- Aman, M. G., McDougle, C. J., Scahill, L., Handen, B., Arnold, L. E., Johnson, C., et al. (2009). Medication and parent training in children with pervasive developmental disorders and serious behavior problems: Results from a randomized clinical trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48, 1143–1154.
- Arnett, J. J. (2000). Emerging adulthood: A theory of development from the late teens through the twenties. *American Psychologist*, 55, 469–480.
- Bernhard, B., Schaub, A., Kummeler, P., Dittmann, S., Severus, E., Seemuller, F., et al. (2006). Impact of cognitive-psychoeducational interventions in bipolar patients and their relatives. *European Psychiatry*, 21, 81–86.
- Billstedt, E., Gillberg, I. C., & Gillberg, C. (2007). Autism in adults: Symptom patterns and early childhood predictors. Use of the DISCO in a community sample followed from childhood. *Journal of Child Psychology and Psychiatry*, 48, 1102–1110.
- Blacher, J. (2001). Transition to adulthood: Mental retardation, families, and culture. *American Journal on Mental Retardation*, 106, 173–188.
- Eaves, L. C., & Ho, H. H. (2008). Young adult outcome of autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38, 739–747.
- Esbensen, A. J., Bishop, S. L., Seltzer, M. M., Greenberg, J. S., & Taylor, J. L. (2010). Comparisons between individuals with autism spectrum disorders and individuals with Down syndrome in adulthood. *American Journal on Intellectual and Developmental Disabilities*, 115, 277–290.
- Farley, M. A., McMahon, W. M., Fombonne, E., Jenson, W. R., Miller, J., Gardner, M., et al. (2009). Twenty-year outcome for individuals with autism and average or near-average cognitive abilities. *Autism Research*, 2, 109–118.
- Furstenberg, F. F., Rumbaut, R. G., & Settersten, R. A. (2005). On the frontier of adulthood: Emerging theme and new directions. In R. A. Settersten, F. F. Furstenberg, & R. G. Rumbaut (Eds.), *On the frontier of adulthood: Theory, research and public policy* (pp. 3–28). Chicago: University of Chicago Press.
- Fussell, E., & Furstenberg, F. F. (2005). The transition to adulthood during the twentieth century. In R. A. Settersten, F. F. Furstenberg, & R. G. Rumbaut (Eds.), *On the frontier of adulthood: Theory, research and public policy* (pp. 29–75). Chicago: University of Chicago Press.
- Greenberg, J. S., Seltzer, M. M., Hong, J., & Orsmond, G. I. (2006). Bidirectional effects of expressed emotion and behavior problems and symptoms in adolescents and adults with autism. *American Journal on Mental Retardation*, 111, 229–249.
- Hazan, C., & Shaver, P. R. (1990). Love and work: An attachment-theoretical perspective. *Journal of Personality and Social Psychology*, 59, 270–280.
- Howlin, P. (2005). Outcomes in autism spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 201–220). Hoboken: Wiley.
- Howlin, P., Mawhood, L., & Rutter, M. (2000). Autism and developmental receptive language disorder – A follow-up comparison in early adult life. II: Social, behavioural, and psychiatric outcomes. *Journal of Child Psychology and Psychiatry*, 41, 561–578.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45, 212–229.
- Matson, M. L., Mahan, S., & Matson, J. L. (2009). Parent training: A review of methods for children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 3, 868–875.
- McCracken, J. T., McGough, J., Shah, B., Cronin, P., Hong, D., Aman, M. G., et al. (2002). Risperidone in children with autism and serious behavioral problems. *The New England Journal of Medicine*, 347, 314–321.
- Shattuck, P. T., Wagner, M., Narendorf, S., Sterzing, P., & Hensley, M. (2011). Post-high school service use among young adults with an autism spectrum disorder. *Archives of Pediatrics & Adolescent Medicine*, 165, 141–146.
- Smith, L. E., Greenberg, J. S., Seltzer, M. M., & Hong, J. (2008). Symptoms and behavior problems of adolescents and adults with autism: Effects of mother-child relationship quality, warmth, and praise. *American Journal on Mental Retardation*, 113, 378–393.
- Taylor, J. L. (2009). The transition to adulthood for individuals with autism spectrum disorders and their families. *International Review of Research in Mental Retardation*, 38, 1–32.
- Taylor, J. L., & Seltzer, M. M. (2010). Changes in the autism behavioral phenotype during the transition to adulthood. *Journal of Autism and Developmental Disorders*, 40, 1431–1446.
- Taylor, J. L., & Seltzer, M. M. (2011a). Changes in the mother-child relationship during the transition to adulthood for youth with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41, 1397–1410.
- Taylor, J. L., & Seltzer, M. M. (2011b). Employment and post-secondary educational activities for young adults with Autism spectrum disorders during the transition to adulthood. *Journal of Autism and Developmental Disorders*, 41, 556–574.
- Vismara, L. A., & Rogers, S. J. (2010). Behavioral treatments in autism spectrum disorder: What do we know? *Annual Review of Clinical Psychology*, 6, 447–468.

Advocacy

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Definition

Advocacy refers to the process by which an individual or a group of individuals support(s) a cause or protect(s) the rights of an individual or group of individuals. Disability advocates can be parents, professionals, or individuals with disabilities themselves (known as self-advocates). Advocacy exists on multiple levels, ranging from the individual level to advocacy related to change of social policy.

Historical Background

Historically, advocates have been involved in many different issues, including education, healthcare, employment, housing, social opportunities, and more. In the education arena, advocates have been responsible for much of the legislation related to the special education laws in the United States. For example, a group of parents working with the Pennsylvania Association of Retarded Citizens (now known as the ARC) filed a complaint which eventually led to the passage of The Education for All Handicapped Children Act, the precursor to today's Individuals with Disabilities Education Improvement Act (IDEIA). Through advocating at the national and state level, these advocates have sought to ensure that all children with disabilities receive a free and appropriate public education. With regard to autism spectrum disorders (ASD), many advocates have pushed states to pass autism insurance legislation, in an attempt to prevent insurance companies from denying services to individuals with ASD. Currently, almost every state in the country has adopted or is considering autism insurance legislation. Advocates have also been involved in protecting the personal rights of individuals with

disabilities. For example, advocates in states such as Pennsylvania have proposed adult protective services laws, which would protect adults who are physically abused but unable to testify in court due to communication difficulties. This is particularly important in cases where physical evidence points to abuse, but a disability such as autism prevents the victim from testifying.

On an individual level, parents have always needed to advocate for services for their children with disabilities. In the education system, Congress has encouraged the development of parent advocates by increasingly including provisions in legislation which encourage parent involvement in educational decisions for their children. For example, parents are mandated participants in the process of developing individualized education programs (IEPs) for their children and are an integral part of IEP teams.

Similarly, Congress has empowered individuals with disabilities with rights to participate in their own education decisions. This has helped to create a new generation of self-advocates. At the age of 14, students with disabilities are invited to take part in the IEP process. Many IEPs for students with disabilities, including autism, include self-advocacy goals to help students learn to effectively communicate their needs and opinions. As a result, more and more individuals with disabilities are becoming adults who possess effective self-advocacy skills. Not only are these adults adept at advocating on their own behalf, many of them effectively advocate for broader social change. Today, individuals with disabilities sit on national, state, and local advisory boards, which are charged with developing policy that directly affects the disability community.

The role of the professional advocate has also developed over time, particularly as the education and other service systems have expanded and become more complex. Attorneys are advocates by virtue of their training, but increasingly other professionals have labeled themselves as disability or child "advocates." These professionals are not regulated nor accredited by any board. Nonetheless, many of them have a wealth of experience, which families have found helpful in advocating for services for their children.

Current Knowledge

There are a number of different disability advocacy organizations that exist today. Two of the oldest are the ARC (formerly Association of Retarded Citizens) and TASH (formerly American Association on Mental Deficiency). The National Disability Rights Network (formerly the National Association of Protection and Advocacy Systems) began more recently in the 1980s. As the prevalence of autism spectrum disorders has increased over time, advocacy organizations specific to ASD have been formed. Current autism advocacy organizations include the Autism Society of America, Autism Speaks, AutismNOW, Autism National Committee (AUTCOM), Autism Network International, the Autism Self Advocacy Network (ASAN), and the Global and Regional Asperger Syndrome Partnership (GRASP). The last four of these groups are self-advocacy organizations. Local autism support groups may also function as advocacy organizations; additionally, these groups can be effective at teaching parents to become more effective advocates.

Future Directions

Many advocacy organizations set short- and long-term agendas for their advocacy efforts. In the autism community, insurance legislation remains an area of concern in a handful of states across the country. Furthermore, despite new insurance laws in many states, funding for autism services remains incomplete and inadequate. Many advocates are expending great efforts to ensure that Medicaid and other federal and state programs are supported in the budget processes. Other legislations that are currently supported by autism advocates include the IDEA Fairness Restoration Act (to override a Supreme Court decision disallowing parents to be reimbursed for expert witness fees), the Combating Autism Reauthorization Act of 2011 (providing support for research into the causes and treatments for ASD), the Caring for Military Kids with Autism Act (to reverse a Department of Defense healthcare provision which does not recognize

autism as a treatable condition), and the Autism Service and Workforce Acceleration Act of 2011 (to develop comprehensive treatment centers and to improve the transition into adulthood for youth with ASD). When legislation is involved, some advocacy organizations will distribute action alerts to their constituencies to encourage interested parties to send letters to legislators and public officials.

In addition to legislation, court cases may arise which affect the rights of individuals with disabilities. Advocacy organizations may submit amicus briefs related to a particular issue that affects their constituency. The self-advocacy organizations, in particular, have submitted amicus briefs in employment cases and cases involving restraint and seclusion.

Going forward, as more and more children with ASD age into adulthood, advocacy efforts may begin to focus even more toward issues related to employment, housing, and adult services. New legislation regarding autism insurance will lead to more questions related to its interpretation, and advocates will be needed to represent the interests of individuals with ASD and their families. Indeed, funding will likely always remain a key area of advocacy efforts, given persistent budgetary constraints. In the education arena, in addition to the pending legislation related to expert witness fees in special education cases, federal legislation regarding the allocation of burden of proof in special education cases may be proposed. Currently states differ as to who has the responsibility to prove the case when the parent files the lawsuit but the educational authority (the school district) has the most access to evidence.

Another development related to advocacy may be the development of more training programs for professional advocates. There are advocate training programs hosted by a range of organizations, from law schools, to educational agencies, to private individuals and companies. Many of these training opportunities have been helpful in educating parents about their own rights and may offer a broader perspective that enable these parents to better assist other parents as well. Nonetheless, as advocates become more involved in

assisting parents in special education due process proceedings, there could be momentum to regulate advocate certificates (insofar as the certificates being offered do not provide licensure or credentialing).

See Also

- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [PL94-142](#)
- [Procedural Safeguards](#)
- [Self-Advocacy](#)

References and Reading

- Disability Rights Florida. *Self-advocacy*. Retrieved from http://www.disabilityrightsflorida.org/resources/disability_topic_info/category/self-advocacy
- Education for all Handicapped Children Act of 1975, Pub. L., No. 94-142, 89 Stat. 773.
<http://autismnow.org>
<http://autreat.com>
<http://grasp.org>
<http://tash.org>
<http://www.autcom.org>
<http://www.autism-society.org/>
<http://www.autismspeaks.org>
<http://www.autismvotes.org>
<http://www.autisticadvocacy.org>
<http://www.ncd.gov>
<http://www.ndrn.org>
<http://www.thearc.org>
<http://www.wrightslaw.com>
- Individuals with Disabilities Education Improvement Act of 2004, 20 U.S.C. §§ 1400 *et seq.*, Pub. L. No. 108-446, 118 Stat. 2803.
- Kamleiter, M. (n.d.). *Role of the advocate*. Retrieved from http://www.flspedlaw.com/Adv_Roles.html
- Katsiyannis, A., et al. (2001). Reflections on the 25th anniversary of the Individuals with Disabilities Education Act. *Remedial and Special Education*, 22(6), 324–334.
- Pennsylvania Association for Retarded Citizens (PARC) v. *Commonwealth of Pennsylvania*, 343 F. Suppl. 279 (E.D. Pa. 1972).
- PL 94-142: policy, evolution, and landscape shift (2007). Retrieved May 2, 2011, from <http://www.thefreelibrary.com/PL+94-142%3a+policy%2c+evolution%2c+and+landscape+shift.-a0173465140>
- US Office of Special Education Programs. (2000). *Twenty-five years of progress in educating children with disabilities through IDEA*. Retrieved May 2, 2011, from <http://www2.ed.gov/policy/special/leg/idea/history.html>

Affective Development

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Definition

Affective development pertains to the emergence of the emotional capacity to experience, recognize, and express a range of emotions and to adequately respond to emotional cues in others. Emotions such as happiness or fear are defined as subjective reactions to experience that are associated with physiological and behavioral changes. Emotional functioning comprises several aspects, including the inducement and elicitation of internal physiological states, the physiological pathways that mediate these internal states, the emotional expressions, and the perception of affect. Overt manifestations of affective expressions and responses include facial expressions, voice, postures, and movements. Affective development is intertwined with the development of social skills, and this psychosocial combination reflects one's distinctive personality and tendencies when responding to others, engaging in social interactions, and adapting to the interpersonal world (Saarni et al. 2006).

Individuals with autism have difficulties in emotional expressiveness and responsiveness and in the appropriateness of these emotional manifestations to the social context. Individuals with autism may exhibit limited empathic responsiveness and may demonstrate specific difficulties in face perception and face recognition, emotional regulation, and engagement in affective and social behaviors and contact with others. Some individuals with autism seem to manifest emotional flatness or aloofness and seem unresponsive to the social environment. It is most challenging for individuals with autism to reason about the emotional world of oneself and others, thus making it more difficult to successfully engage in social situations (Sigman and Capps 1997).

Historical Background

Kanner (1943) originally wrote that children with autism “have come into the world with innate inability to form the usual, biologically provided affective contact with people, just as other children come into the world with innate physical or intellectual handicaps.” (p. 250). Children with autism were originally described as aloof, unresponsive, or even emotionally detached, and the first studies regarding affective development in autism examined this issue of children’s emotional expressiveness. Early reports indicated that children with autism did not appear less emotionally expressive than children with mental retardation or than typically developing children (Capps et al. 1993; Ricks and Wing 1975). However, parents reported that their children with autism experienced higher levels of negative emotions such as fear, sadness, and anger and lower levels of positive emotions such as joy and interest, compared to the reports of parents of children with mental retardation and typically developing children (Capps et al. 1993). Researchers investigated whether children’s emotional expressions and responses (e.g., smiles, laughter, or even temper tantrums) were socially adequate and context appropriate. Findings revealed that children with autism sometimes manifested discordant affects or deficits in displaying positive affect and coordinating gaze with emotional expression to reveal sharing of emotional experience (Kasari et al. 1990, 1993; Yirmiya et al. 1989). For example, children with autism generally did not look up at their parents and smile when responding to parental praise for an accomplishment, whereas children with typical development or mental retardation generally did. Other studies on children with autism pinpointed difficulties in coordinating and pairing facial expressions with vocal expressions of emotions, with prosodic and linguistic expressions of emotions, or with body gestures (Hobson 1986; Van Lancker et al. 1989). Interestingly, most studies on children and adolescents with high-functioning autism or Asperger syndrome revealed no difficulties in labeling facial expressions, especially of the basic emotions of happiness, sadness, anger, fear, surprise,

and disgust (Braverman et al. 1989; Capps et al. 1992; Hobson et al. 1989; MacDonald et al. 1989; Ozonoff et al. 1990; Yirmiya and Sigman 1991). Current studies are now focusing on measuring emotional recognition abilities and more subtle emotions in individuals with autism with normal intelligence using more fine-grain measures (Golan et al. 2006, 2008; Happé 1994).

These atypicalities in affective development are currently widely accepted as features of autism, but their underlying causes remain a matter of debate. Some investigators consider the difficulties in affective development as secondary to, or as the result of, impairments in the development of social-cognitive abilities such as perspective-taking capacities or theory of mind (ToM) abilities (Baron-Cohen et al. 1985; Happé and Frith 2006), whereas other investigators consider abnormal affective development to be a core deficit in autism (Hobson 1993). According to the latter approach, individuals with autism reveal difficulties in their biologically based and innate capacity to perceive, decode, and understand emotional cues and expressions, which results in a failure to establish the mentalizing functions needed for appropriate social interactions. Today, there is growing awareness that mentalizing and ToM abilities contribute to the understanding of emotions and vice versa. Two-year-old toddlers are already able to decode facial expressions, but only a year later – using the emerging ToM abilities – can they also recognize the internal mental or emotional states that are reflected by these facial expressions. In turn, young children’s growing understanding of basic emotions facilitates and promotes their mentalizing abilities and their comprehension that desires differ from reality (Sigman and Capps 1997).

Current Knowledge

Affective Development in the Early Years

Caregivers facilitate the affective development of their children by supporting and scaffolding the emerging emotional capacities of their children. Infants come into the world equipped with a strong drive to emotionally engage with others.

Newborns are prepared to engage in mutual affective regulation, a process by which the infant and the caregiver communicate emotional states to each other and respond appropriately and sensitively (Jaffe et al. 2001; Kogan and Carter 1996; Stern 1985; Trevarthen 1993; Tronick 1989; Weinberg and Tronick 1996). In the first weeks of life, babies fluctuate between several states of arousal such as crying, sleeping, drowsiness, and alertness, with limited ability to control and regulate these shifts. As the neurological and physiological system becomes more mature and integrated, and the environment provides responsive parental care, infants become better able to regulate states of arousal. They spend more time awake, looking around and exploring social stimuli such as faces, as well as smiling, cooing, and laughing. Their emotional states can be easily seen during parent–child face-to-face interactions, in which infants take an active part in mutual regulation by sending and signaling behavioral and emotional cues such as smiles, gazes, or vocalizations. This synchronized match or “dance” between parent and child is an important mechanism underlying socio-affective development and is considered a prerequisite for later emotional functioning, empathy, and prosocial behaviors (Feldman 2007; Feldman et al. 1999). It was found that toddlers who showed high sensitivity and attention to emotional cues at the age of 2 years were more socially responsive with their peers, both at age 2 and at age 5. These factors may also render reciprocal effects, where children learn about emotions through their relationships with others. In sum, affective development in the first years is influenced by genetic, biological, and environmental factors and is strongly related to children’s temperament and to the development of the parent–child relationship and attachment.

Recent evidence is accumulating regarding different affective developmental trajectories of young children with autism, compared to children with typical development. Retrospective accounts, obtained from parents’ reports and home videotape analyses of the first 2 years, revealed that children with autism differ from children with typical development in social-

emotional behaviors, describing difficulties in affect regulation as well as increased negative affect and ambiguous affective expressions (Baranek 1999; Maestro et al. 2005; Osterling et al. 2002).

Prospective studies of siblings of children with autism – a group considered at risk for the development of autism and related difficulties – demonstrated that 12- to 18-month-old infants later diagnosed with autism are distinguishable from other infants who were not later diagnosed with autism in several social-emotional aspects, such as reductions in expression of positive emotion, social smiling, reactivity, and social interest as well as atypicalities in eye gaze, imitation, and orienting to name (Ozonoff et al. 2010; Young et al. 2009; Zwaigenbaum et al. 2005, 2009). Interestingly, these early manifestations were not extended downward; 6-month-old infants later diagnosed with autism were not distinguishable from 6-month-old infants who were not later diagnosed with autism in their affective expressions or in their social use of gaze and affect during social interactions with mutual sharing of attention and affect (Rozga et al. 2011). Furthermore, 24-month-old toddlers later diagnosed with autism were also distinguishable from their non-diagnosed peers in their temperament profiles, as marked by lower positive affect, difficulties in regulating negative affect, as well as lower feelings of excitement in situations of anticipation (Brian et al. 2008; Bryson et al. 2007). Thus, these important studies on the early affective development of young children with autism provide evidence regarding the presence of difficulties in affect displays and emotional regulation in the first years of life.

Affective Development in Childhood

Emotional development and sense of self are rooted in the experience of early childhood and continue to develop over the childhood years. Typical affective development in these years pertains to understanding and regulating emotions and to the organization of self-concept. As they grow, children become more aware of their own and other people’s emotions, can better regulate and control their feelings, respond with more

empathic behaviors, and show more acceptable emotional expressions. Through interacting with peers and their emerging friendships, children learn about their own emotions, become aware that individuals have different emotional reactions, and can better reflect on others' motives and intentions during complex social-emotional situations. Children must also cope with the emotional challenges associated with social developmental milestones during childhood, such as demands for social conformity, overt competition with others, and mastery of different academic skills (Saarni et al. 2006).

Children with autism face the same challenges as do typically developing children. Although some children with autism may master many academic skills, they have great difficulties managing everyday emotional and social situations in which an array of emotional and social cues must be recognized, interpreted, and synthesized quickly and simultaneously (Baron-Cohen 1995; Bauminger et al. 2008). Clearly, children with autism manifest great variation in their desire to form emotional connections with peers and adults, as well as in their ability to perceive and respond to the emotions of others. Studies regarding the understanding and experience of social emotions such as pride, embarrassment, or empathy revealed that school-age children with autism reported having these feelings as often as typically developing children; however, in their description of situations containing social emotions, they tended to describe more basic emotions (e.g., happy instead of proud) and to describe them more generally and less personally or interpersonally (Kasari et al. 2001).

Researchers examining affective development of children with autism also revealed strong associations between higher cognitive abilities and better understanding of emotional situations (Dyck et al. 2001; Golan et al. 2006), suggesting that cognition is an important moderating variable in affective development, as well as in compensatory strategies that children use to cope in emotional or social situations (Capps et al. 1992; Kasari et al. 2001). It has been suggested that the impaired performance of children with autism on measures of emotional functioning may be

secondary to difficulties in cognitive or ToM abilities, as well as to difficulties in linguistic and pragmatic capacities. Indeed, emotion perception difficulties are not specific to autism but have also been detected in individuals with other disabilities such as learning disabilities, mental retardation, and schizophrenia (Davis and Gibson 2000; Edwards et al. 2001; Zaja and Rojahn 2008).

Most of the evidence regarding affective development during childhood comes from studies of high-functioning children with autism. Children with autism who are low functioning in terms of cognitive abilities and are unable to speak and comprehend language continue to struggle with earlier affective developmental tasks even in childhood. They usually remain more engaged with objects and have few social interactions with peers, and they face challenges in learning alternative ways to communicate (Sigman and Capps 1997).

Affective Development in Adolescence and Adulthood

Adolescence, the developmental transition between childhood and adulthood, entails major physical, cognitive, and psychosocial changes. Adolescence enables vast opportunities for growth and autonomy and for its major developmental task – the search for personal identity. Adolescents must deal with physical alterations and sexual maturity as well as with the development of emotional independence from their parents and families by reorganizing their relationships with parents, siblings, and peers. Their emerging metacognitive thinking enables better comprehension and understanding of complex social and emotional situations, facilitating the capacity for self-consciousness and empathic responsiveness (Saarni et al. 2006).

Adolescents with autism have difficulties talking about their emotional experiences as well as about more complex social emotions other than the basic emotions such as happiness or fear. They also exhibit difficulties in their ability to empathize and recognize the emotions of others compared to adolescents with typical development who are matched on gender and on verbal and cognitive abilities (Capps et al. 1992). In their

descriptions of subjective experiences, adolescents with autism tend to attribute emotions to material circumstances and events rather than to interpersonal interactions or the attainment of a goal to a greater extent than do adolescents with mental retardation or adolescents with typical development (Jaedicke et al. 1994). For example, the descriptions of emotions by adolescents with autism tend to be more idiosyncratic and peculiar than the descriptions of emotions by adolescents in the comparison groups, who tend to link emotions to academic, social, and athletic successes or failures. Furthermore, the task of talking about feelings was more distressful for adolescents with autism; they appeared to struggle with the task and needed prompting and more time to respond compared to adolescents with typical development (Yirmiya and Sigman 1991). Interestingly, it has been demonstrated that adolescents with autism showed better emotional responsiveness abilities than younger children with autism when asked to respond to videotaped stories about children experiencing different events and emotions such as happiness, anger, or sadness (e.g., a boy is sad because he lost his dog). These findings suggest that as children with autism get older, their emotional responsiveness improves. However, these findings were not yet examined using longitudinal research designs and thus need further investigation (Sigman and Capps 1997).

As in childhood, during adolescence verbal and cognitive capacities play a major role in navigating one's developmental course. For some adolescents with autism, the widening gap with typical development may be associated with an aggravation of behavioral symptoms and poorer social functioning. It appears that the increasing complexity of adolescents' social and emotional world, and their engagement in more sophisticated interpersonal interactions, outstrips their advances in social and emotional functioning. Furthermore, difficulties in cognition and social understanding hinder adolescents' adjustment to their own growing physical and psychological alterations, making the adaptation process for this new developmental phase more challenging (Sigman and Capps 1997).

Few longitudinal studies have been conducted to follow children and adolescents with autism into adulthood; therefore, little information is available on affective development after this important turning point in life. The transition from adolescence to adulthood for individuals with autism is usually associated with exiting the school system and entering the adult service system, which is sometimes accompanied by the loss of many entitled services. There is evidence for social and psychiatric disorders in adults with autism that appear to increase with age. For example, adults with autism were found to engage in fewer social and recreational activities and also reported fewer friendships and peer relationships than at younger ages. However, other studies revealed that compared to typically developing individuals, adults with autism did not spend more time alone and were equally involved in social activities; however, they experienced increased social anxiety when in the company of less familiar people. Indeed, more longitudinal research is needed to expand this exploration of social functioning to incorporate emotional abilities in adulthood (Billstedt et al. 2011; Howlin et al. 2004).

Future Directions

Infants share common patterns of affective development; however, each infant shows a distinct emotional profile from the first days of life. Exploring the associations between early emotional style and later development of autism will contribute both to early identification of autism spectrum disorders and to early intervention programs (Dawson et al. 2010; Landa et al. 2010; Rozga et al. 2011). Furthermore, social interventions which are focused on understanding and recognizing more complex social emotions and mental states (e.g., embarrassment, irony) may strongly enhance problem solving abilities in social situations and social engagement (Lopata et al. 2010). Indeed, the issue of generalization of acquired social-emotional abilities to other social situations and to everyday life social interactions is most challenging, and further research is needed to evaluate the efficacy of social-emotional intervention.

See Also

- ▶ Attachment
- ▶ Emotion Regulation
- ▶ Empathy
- ▶ Friendships
- ▶ Interpersonal Skills
- ▶ Self-Recognition
- ▶ Self-Recognition and Self-Referential Behavior
- ▶ Social Cognition
- ▶ Social Interventions
- ▶ Temperament

References and Reading

- Baranek, G. T. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders*, 29(3), 213–224.
- Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory of mind*. Cambridge, MA: The MIT Press.
- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic-child have a theory of mind. *Cognition*, 21(1), 37–46.
- Bauminger, N., Solomon, M., Aviezer, A., Heung, K., Gazit, L., Brown, J., et al. (2008). Children with autism and their friends: A multidimensional study of friendship in high-functioning autism spectrum disorder. *Journal of Abnormal Child Psychology*, 36(2), 135–150.
- Billstedt, E., Gillberg, I. C., & Gillberg, C. (2011). Aspects of quality of life in adults diagnosed with autism in childhood. A population-based study. *Autism*, 15(1), 7–20.
- Braverman, M., Fein, D., Lucci, D., & Waterhouse, L. (1989). Affect comprehension in children with pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 19(2), 301–316.
- Brian, J., Bryson, S. E., Garon, N., Roberts, W., Smith, I. M., Szatmari, P., et al. (2008). Clinical assessment of autism in high-risk 18-month-olds. *Autism*, 12(5), 433–456.
- Bryson, S. E., Zwaigenbaum, L., Brian, J., Roberts, W., Szatmari, P., Rombough, V., et al. (2007). A prospective case series of high-risk infants who developed autism. *Journal of Autism and Developmental Disorders*, 37(1), 12–24.
- Capps, L., Yirmiya, N., & Sigman, M. (1992). Understanding of simple and complex emotions in nonretarded-children with autism. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 33(7), 1169–1182.
- Capps, L., Kasari, C., Yirmiya, N., & Sigman, M. (1993). Parental perception of emotional expressiveness in children with autism. *Journal of Consulting and Clinical Psychology*, 61(3), 475–484.
- Davis, P. J., & Gibson, M. G. (2000). Recognition of posed and genuine facial expressions of emotion in paranoid and nonparanoid schizophrenia. *Journal of Abnormal Psychology*, 109(3), 445–450.
- Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., et al. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The early start Denver model. *Pediatrics*, 125(1), E17–E23.
- Dyck, M. J., Ferguson, K., & Shochet, I. M. (2001). Do autism spectrum disorders differ from each other and from non-spectrum disorders on emotion recognition tests? *European Child & Adolescent Psychiatry*, 10(2), 105–116.
- Edwards, J., Pattison, P. E., Jackson, H. J., & Wales, R. J. (2001). Facial affect and affective prosody recognition in first-episode schizophrenia. *Schizophrenia Research*, 48(2–3), 235–253.
- Feldman, R. (2007). Parent-infant synchrony: Biological foundations and developmental outcomes. *Current Directions in Psychological Science*, 16(6), 340–345.
- Feldman, R., Greenbaum, C. W., & Yirmiya, N. (1999). Mother-infant affect synchrony as an antecedent of the emergence of self-control. *Developmental Psychology*, 35(1), 223–231.
- Golan, O., Baron-Cohen, S., & Hill, J. (2006). The Cambridge Mindreading (CAM) face-voice battery: Testing complex emotion recognition in adults with and without Asperger syndrome. *Journal of Autism and Developmental Disorders*, 36(2), 169–183.
- Golan, O., Baron-Cohen, S., & Golan, Y. (2008). The ‘Reading the Mind in Films’ task [child version]: Complex emotion and mental state recognition in children with and without autism spectrum conditions. *Journal of Autism and Developmental Disorders*, 38(8), 1534–1541.
- Happé, F. (1994). An advanced test of theory of mind – Understanding of story characters thoughts and feelings by able autistic, mentally-handicapped, and normal-children and adults. *Journal of Autism and Developmental Disorders*, 24(2), 129–154.
- Happé, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36(1), 5–25.
- Hobson, R. P. (1986). The autistic child’s appraisal of expressions of emotion – A further study. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 27(5), 671–680.
- Hobson, R. P. (1993). Understanding persons: The role of affect. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds.), *Understanding other minds: Perspectives from autism* (pp. 204–224). Oxford: Oxford University Press.
- Hobson, R. P., Ouston, J., & Lee, A. (1989). Naming emotion in faces and voices – Abilities and disabilities in autism and mental-retardation. *British Journal of Developmental Psychology*, 7, 237–250.

- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45(2), 212–229.
- Jaedicke, S., Storoschuk, S., & Lord, C. (1994). Subjective experience and causes of affect in high-functioning children and adolescents with autism. *Development and Psychopathology*, 6(2), 273–284.
- Jaffe, J., Beebe, B., Feldstein, S., Crown, C. L., & Jasnow, M. D. (2001). Rhythms of dialogue in infancy: Coordinated timing in development – Introduction. *Mono-graphs of the Society for Research in Child Development*, 66(2), vii–viii, 1–151.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kasari, C., Sigman, M., Mundy, P., & Yirmiya, N. (1990). Affective sharing in the context of joint attention interactions of normal, autistic, and mentally-retarded children. *Journal of Autism and Developmental Disorders*, 20(1), 87–100.
- Kasari, C., Sigman, M. D., Baumgartner, P., & Stipek, D. J. (1993). Pride and mastery in children with autism. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 34(3), 353–362.
- Kasari, C., Chamberlain, B., & Bauminger, N. (2001). Social emotions and social relationships in autism: Can children with autism compensate? In J. Burack, T. Charman, N. Yirmiya, & P. Zelazo (Eds.), *Perspectives on development in autism* (pp. 309–323). Hillsdale: Erlbaum.
- Kogan, N., & Carter, A. S. (1996). Mother-infant reengagement following the still-face: The role of maternal emotional availability in infant affect regulation. *Infant Behavior & Development*, 19(3), 359–370.
- Landa, R. J., Holman, K. C., O'Neill, A. H., & Stuart, E. A. (2010). Intervention targeting development of socially synchronous engagement in toddlers with autism spectrum disorder: A randomized controlled trial. *Journal of Child Psychology and Psychiatry*, 52(1), 13–21.
- MacDonald, H., Rutter, M., Howlin, P., Rios, P., Leconteur, A., Evered, C., et al. (1989). Recognition and expression of emotional cues by autistic and normal adults. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 30(6), 865–877.
- Maestro, S., Muratori, F., Cesari, A., Cavallaro, M. C., Paziente, A., Pecini, C., et al. (2005). Course of autism signs in the first year of life. *Psychopathology*, 38(1), 26–31.
- Osterling, J. A., Dawson, G., & Munson, J. A. (2002). Early recognition of 1-year-old infants with autism spectrum disorder versus mental retardation. *Development and Psychopathology*, 14(2), 239–251.
- Ozonoff, S., Pennington, B. F., & Rogers, S. J. (1990). Are there emotion perception deficits in young autistic-children. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 31(3), 343–361.
- Ozonoff, S., Iosif, A. M., Baguio, F., Cook, I. C., Hill, M. M., Hutman, T., et al. (2010). A prospective study of the emergence of early behavioral signs of autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(3), 256–266.
- Ricks, D. M., & Wing, L. (1975). Language, communication, and use of symbols in normal and autistic-children. *Journal of Autism and Childhood Schizophrenia*, 5(3), 191–221.
- Rozga, A., Hutman, T., Young, G. S., Rogers, S. J., Ozonoff, S., Dapretto, M., et al. (2011). Behavioral profiles of affected and unaffected siblings of children with autism: Contribution of measures of mother-infant interaction and nonverbal communication. *Journal of Autism and Developmental Disorders*, 41, 287–301.
- Saarni, C., Campos, J. J., Camras, L. A., & Witherington, D. (2006). Emotional development: Action, communication, and understanding. In W. Damon, R. M. Lerner, & N. Eisenberg (Eds.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 226–299). New York: Wiley.
- Sigman, M., & Capps, L. (1997). Development of social and emotional understanding. In *Children with autism: A developmental perspective* (pp. 34–60). London: Harvard University Press.
- Stern, D. (1985). *The interpersonal world of the infant: A view from psychoanalysis and developmental psychology*. New York: Basic Books.
- Trevarthen, C. (1993). The self born in intersubjectivity: An infant communicating. In U. Neisser (Ed.), *The perceived self: Ecological and interpersonal sources of self-knowledge* (pp. 121–173). New York: Cambridge University Press.
- Tronick, E. Z. (1989). Emotions and emotional communication in infants. *American Psychologist*, 44(2), 112–119.
- Van lancker, D., Cornelius, C., & Kreiman, J. (1989). Recognition of emotional-prosodic meanings in speech by autistic, schizophrenic, and normal-children. *Developmental Neuropsychology*, 5(2–3), 207–226.
- Weinberg, M. K., & Tronick, E. Z. (1996). Infant affective reactions to the resumption of maternal interaction after the still-face. *Child Development*, 67(3), 905–914.
- Yirmiya, N., & Sigman, M. (1991). High functioning individuals with autism – Diagnosis, empirical-findings, and theoretical issues. *Clinical Psychology Review*, 11(6), 669–683.
- Yirmiya, N., Kasari, C., Sigman, M., & Mundy, P. (1989). Facial expressions of affect in autistic, mentally-retarded and normal-children. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 30(5), 725–735.
- Young, G. S., Merin, N., Rogers, S. J., & Ozonoff, S. (2009). Gaze behavior and affect at 6 months: Predicting clinical outcomes and language development in typically developing infants and infants at risk for autism. *Developmental Science*, 12(5), 798–814.
- Zaja, R. H., & Rojahn, J. (2008). Facial emotion recognition in intellectual disabilities. *Current Opinion in Psychiatry*, 21(5), 441–444.
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International*

Journal of Developmental Neuroscience, 23(2–3), 143–152.

Zwaigenbaum, L., Bryson, S., Lord, C., Rogers, S., Carter, A., Carver, L., et al. (2009). Clinical assessment and management of toddlers with suspected autism spectrum disorder: Insights from studies of high-risk infants [Review]. *Pediatrics*, 123(5), 1383–1391.

Affective Disorders (Includes Mood and Anxiety Disorders)

► Anxiety Disorders

Affective Regulation

► Emotional Regulation

Affixes

► Speech Morphology

Affliction

► Disability

Afghanistan and Autism

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Abstract

The people of Afghanistan continue to hurt from the effects of decades of invasion and civil war. While there have been some efforts to support the physically disabled population of Afghanistan, there is less support for individuals

with intellectual and mental disabilities; specifically, there is no support for children and adults with autism. This entry begins by introducing the history of autism-spectrum disorder (ASD) and the various legislative and political efforts in place in Afghanistan to support general disabilities. Furthermore, it addresses the lack of support for people specifically with autism in Afghanistan in regard to research and treatment of the condition. This is measured by the lack of nationwide recognition and awareness of autism, screening and diagnosis, and treatment for those afflicted with it. In addition to recognizing the severe lack of mental health resources in general, this entry also addresses the various cultural and social perceptual issues with autism and mental disabilities in Afghanistan. The aim of this entry is to acknowledge and understand the deep historical, political, economic, and social influences individuals and families affected by autism face in Afghanistan so that the country and its allies may begin to effectively tackle autism.

Historical Background

The earliest known description of autism was by Leo Kanner in the mid-twentieth century. In his report on 11 cases of children with seemingly autistic characteristics, he described these children as being removed from the social world and having a resistance to change or “insistence on sameness” (Kanner 1943). He believed that the condition they had was congenital. Autism was not recognized as an individual diagnosis right away as it was conflated with false leads and unclear symptoms (compared to other mental disorders such as schizophrenia) (Volkmar and Reichow 2013). At the time, parents (mainly mothers) were blamed for the condition of their children as there was a lack of understanding of the genetic cause of the condition. Autism was better understood in the 1970s after further quantitative research showed high rates of associated intellectual disabilities and a genetic basis for the condition (Volkmar and Reichow 2013).

Autism was first recognized as a mental disorder in the *Diagnostic and Statistical Manual of*

Mental Disorders-III (DSM-III) in 1980. The DSM-III was mainly focused on infantile autism and a child's lack of social responsiveness (Volkmar and Reichow 2013). The DSM-III included autism in a class of disorders known as pervasive developmental disorder (PDD) (Volkmar and Reichow 2013). Later revisions of the DSM-III such as the DSM-III-R, DSM-IV, and finally the current revision used today, DSM-V, included more accurate definitions of autism based on various reviews of literature and field studies around the world (Volkmar and Reichow 2013). In addition to having better diagnostic behavioral descriptions, it also adopted a refined description of the class of the disorder known as "autism spectrum disorder" (replacing PDD) (Volkmar and Reichow 2013). Today the DSM-V is used to screen and diagnose individuals who are at risk for autism. According to the Afghan Ministry of Public Health in the *National Mental Health Strategy 2009–2014*, diagnostic descriptions from classification systems such as the DSM-V or the ICD 10 (International Classification of Diseases 10) have limited utility in Afghan society (Ministry of Public Health 2009).

Research shows that structured behavioral, communicative, and educational intervention programs are effective and associated with better outcomes for children with ASD (Volkmar et al. 2014, volume 53, issue 2). The treatment options range from highly intensive and individualized one-on-one teaching every week to various pragmatic language skills training and group social skills workshops (Volkmar et al. 2014, volume 53, issue 2). Various institutions exist in America to support individuals with autism including specialized day schools and boarding schools. Other programs focus on training parents to be better caretakers for their children. Overall, there are a range of effective treatments and solutions for managing individuals with autism and teaching them effective social and life skills. Notably, the majority of these studies and treatments exist in affluent English-speaking countries such as the UK and the USA, and there is a severe lack of literature in effective treatment options for individuals with autism in developing countries (Samadi and McConkey 2011).

Legal Issues, Mandates for Service

When trying to understand the relevant policy and nationwide efforts concerning autism, it is important to note that, as is the case in other developing countries, autism is referred to as a general "disability" in Afghanistan. Therefore, policies and legislations that concern autism will fall into this general category rather than specifically focusing on autism. It is also important to recognize that a significant proportion of people categorized as "disabled" in Afghanistan are those who suffer from war-related injuries and illnesses rather than congenital disabilities such as autism.

In recent history, the Afghan government has taken various legislative steps toward advancing the rights of people with disabilities. First and foremost is the constitution of Afghanistan created in 2004 that addresses the rights and the inclusion into society of people with disabilities in the following articles (Sida 2014):

1. Article 22 prohibits any discrimination between Afghan citizens.
2. Article 53 provides for financial aid to persons with disabilities and guarantees their "active participation and reintegration into society."
3. Article 84 makes provision for two persons with disabilities to be appointed by the president as Members of Parliament in the House of Elders.

In addition to constitutional legislation, various national policies were also put into place to help people with disabilities. This included the *National Policy for Persons with Disabilities* created in 2004 which was joined with the *Afghanistan National Disability Action Plan (ANDAP)* in 2008–2011 to improve the access to education, employment, protection, justice, care, and social assistance and insurance for people with disabilities (Sida 2014). In recent years, the *National Law of Rights and Benefits of People with Disabilities* has further provided economic, social, and political support to people with disabilities and protected their rights and participation in society (Sida 2014). More specifically it allocated 3% of government and private sector

jobs to be reserved for people with disabilities. Another important governmental program is the *National Strategy for Disability and Rehabilitation 2013–2016* developed with the support of the UN and the EU (Sida 2014). However, it is important to note that these programs are largely concerned with physical disabilities, leaving intellectual disabilities on the periphery. Since many individuals with autism do not exhibit symptoms of a severe physical disability, this presents a challenge in addressing their needs.

Other outside parties have also tried to make strides in improving mental health services in Afghanistan for those who are disabled. According to the Human Rights Watch, the United Nations Security Council Resolution 2475 was recently adopted in order to protect and safeguard individuals with disabilities in areas of conflict. The resolution urged governments to enable the participation and representation of individuals with disability in humanitarian action and peacebuilding (Human Rights Watch 2019).

Overview of Current Treatments and Centers

Afghanistan severely lacks the institutions and training for a proper mental health system. Due to the decades of war and instability, much of the educated and professional workforce has left the country (Ventevogel 2006). There is a single mental health hospital in the capital city of Kabul and another single psychiatric hospital in Herat (Ventevogel 2006). Aside from other small inpatient facilities for psychiatric patients scattered around the country, there are no other viable options for individuals seeking mental health services. According to Peter Ventevogel of the UNHCR, the way forward in Afghanistan is to move away from hospital-based psychiatry toward a system of mental health integration in primary care services (Ventevogel et al. 2006).

Educational opportunities and resources for people with disabilities in Afghanistan are also quite limited. The Ministry of Education (MoE) is responsible for supporting inclusive education

and access to schools for children with disabilities as of 2004 (Trani et al. 2009). Other than nongovernmental organizations, the only governmental special education facility in all of Afghanistan is a nonresidential school for children with visual impairments in the capital city Kabul (90 children are enrolled). According to Jean Francois Trani, many of the problems both the MoE and the MLSAMD face in meeting the needs of people with disabilities are due to a lack of clear vision (Trani et al. 2009). There is inability to expand the definition of disability, and while there are many programs that cater to the needs of war-disabled people, there are less programs dedicated to those with congenital disabilities. Other obvious concerns are continual conflict and war, insufficient financial resources, and an inability to reach individuals in rural areas of the country. The lack of infrastructure and efficient transportation routes in Afghanistan makes any effort to send aid to disabled citizens living in rural areas difficult.

When the Republic of Afghanistan was established in 2003, the government inherited decades of turmoil to healthcare institutions and a population in great demand of medical attention. A major problem the country faces is a lack of funding for mental health services. In 2004, the health budget of Afghanistan was 289.4 million USD, but only 100,000 USD was allocated toward mental health (WHO-AIMS 2006, p. 1). A study conducted based on data from 2004 reported that there are only 34 hospital beds and 13 general practitioners per 100,000 population dedicated to mental health (WHO-AIMS 2006, p. 1). The private sector has an additional small percentage of hospital beds. Furthermore, the study showed that there are 3900 physician-based primary healthcare clinics in the country (about half private and half public) and 3100 non-physician-based primary healthcare clinics (WHO-AIMS 2006, p. 1). Recent legislative work in the area has focused on increasing psychosocial services in primary healthcare clinics.

The study by the WHO also showed that there are other mental health centers in the country including mental health outpatient facilities. There are 11 of these facilities in the country, and none of them are for adolescents or children.

The majority of patients seen at these centers are diagnosed with mood disorders or anxiety disorders. Day treatment centers are another type of resource; however, there is only one 1-day treatment center in the entire country. There are five community-based psychiatric inpatient units, but again none of these beds are reserved for children or adolescents, and the majority of patients have schizophrenia or mood disorders (WHO-AIMS 2006, p. 1).

It is important to notice that there is little to no attention to children with mental disorders or disabilities in Afghanistan. Not only are there no hospital beds specifically allocated for adolescents with mental health disorders, but there are also no institutionalized centers for screening or diagnosing mental health disorders. Parents who are worried about their child's behavior in cases such as autism have to take their children to psychiatric centers or mental health outpatient facilities designed for adults. There are no centers in Afghanistan for individuals who may potentially have autism to seek help or even receive a diagnosis.

The security crisis in Afghanistan presents another major challenge for any governmental or NGO initiatives related to autism. According to the WHO, in 2018 there were 85 attacks on healthcare centers in Afghanistan (World Health Organization 2019, p.11). Therefore, any community center or treatment center would have to take security concerns into consideration. For this reason, perhaps home-based or parental training services may be safer and have a larger impact on autism care in the short term.

Overview of Research Directions

A major problem for tackling autism in Afghanistan is surveying the prevalence of the condition in the country. The first step toward helping individuals with autism in Afghanistan is to find the distribution and prevalence of the condition in the country. This is necessary to make meaningful programming or policy-driven change for individuals with autism. In 2005, the National Disability Survey in Afghanistan

(NDSA) surveyed 5250 households for the prevalence of disabilities. The study found a general 4.6% prevalence rate (95% CI 4.4 to 4.8%) of disability in Afghanistan (Trani 2008). This amounts to approximately 1.09 million Afghans with some form of physical disability and/or mental distress (Trani 2008). Furthermore, the prevalence of individuals classified with "severe disabilities" was estimated at 2.7% of the population (Trani 2008). This was marked by individuals with functional limitations due to physical, intellectual, or sensory disabilities as well as mental illness. Jean-Francois Trani argues that surveys such as these must take a multidimensional approach using different instruments such as impairments, activity limitations, and assessments of well-being. Furthermore, studies of disability prevalence must go beyond measurements of prevalence and into the associations these disabilities have with individual's social agency and functioning levels in their environments.

Trani's criticism of the NDSA is important to acknowledge, especially when considering the possibility of a nationwide survey on the prevalence of autism. The National Institute of Mental Health characterizes autism as a "spectrum" disorder because there is a wide variety in the type and severity of symptoms associated with the condition (National Institute of Mental Health 2018). People with ASD have difficulty with social interaction and communication and may show restrictive and repetitive behaviors. However, when training professionals to screen for ASD, especially in developing countries, it is important to note that not all people with ASD will show these behaviors.

A study reviewing a 2011 initiative in Iran to identify autism prevalence in the country described two challenges in screening for autism in developing countries (Samadi and McConkey 2011). Since ASD is a condition with a wide spectrum of associated behaviors, the first challenge to the screening process is training professionals to oversee screening and diagnostic services. Also, since the majority of screening tests are made in developed countries with a different cultural context compared to Afghanistan, there is a need for a culturally relevant screening

process individualized to certain cultural contexts. The second challenge arises when using parental responses to interviews. Oftentimes, parents may not be educated or observant of the various signs that their children display (Samadi and McConkey 2011). As is the case in Iran, parents may even underreport their child's difficulties in order to keep them from being referred to special schools or in fear of social stigmatization (Samadi and McConkey 2011). This can cause survey results to indicate a lower prevalence of ASD than what actually exists. On the other hand, if parents also know that their child will be given specific aid or specialized attention, they may exaggerate their child's condition (Samadi and McConkey 2011). Since Afghanistan has many shared cultural contexts as Iran, many of these lessons are essential to keep in mind when designing a plan to screen the prevalence of ASD in the country.

Overview of Training

Training of mental healthcare professionals in Afghanistan is a present-day challenge, and many of the problems the country faced in the past still remain today. There is a lack of emphasis on the importance of mental health training in the country. A WHO study based on data from 2004 found that less than 1% of training for medical doctors in Afghanistan was dedicated toward mental health. Likewise, only 2% of training for nurses was dedicated to mental health (WHO-AIMS 2006, p. 1). The study further reported that only 2 psychiatrists, 61 other doctors, 37 nurses, and 40 other mental health workers worked in public mental health units (WHO-AIMS 2006, p. 1).

In addition to the serious lack of mental health training in Afghanistan, the WHO study also showed a severe shortage of mental health professionals in the country. The study showed a shocking number of only 0.5 human resource workers per 100,000 in the population (WHO-AIMS 2006, p. 1). There was a deficiency in the number of psychiatrists, medical doctors, nurses, medical assistants, and psychosocial counselors in Afghanistan. Furthermore, of the small number of psychiatrists and mental health

workers that did exist, almost none worked in community-based health centers or outpatient facilities (WHO-AIMS 2006, p. 1). In present-day Afghanistan, a shortage in funding toward mental health training and a shortage in mental healthcare professionals still exist. Furthermore, the majority of mental healthcare professionals work in adult mental hospitals, which, in the case of autism, would not be an appropriate place for one to take their child to be screened or diagnosed (World Health Organization 2019).

In 2009, the Government of the Islamic Republic of Afghanistan (GOIRA) and the Ministry of Public Health (MoPH) published a *National Mental Health Strategy* (NMHS) with the following aims (Sayed 2011):

- To promote mental health of the people of Afghanistan
- To minimize the stigma and discrimination attached to mental disorders
- To reduce the impact of mental disorders on individuals, families, and the community
- To prevent the development of mental health problems and mental disorders, wherever possible
- To provide quality, integrated, evidence and rights-based care for individuals suffering from mental disorders at all levels of health system

The program was completed in 2014 and aided the country in implementing a mental health plan to the country's Basic Package of Health Services. This includes a continuum of mental healthcare for Afghans with mental, neurological, and substance abuse disorders in hospital and community centers. Proving significant strides forward to the future of psychiatry and mental health training in Afghanistan was a study by Yousuf Rahimi in 2012 on the training of mental health professionals in Afghanistan. The study revealed around 60 locally trained psychiatrists working in the country (Rahimi and Azimi 2012). Furthermore, it was shown that neuropsychiatry was being taught in the latter years of medical school and that a 3–5-year postgraduate program was introduced in psychiatry by the

Ministry of Public Health, taking place in the psychiatric hospitals in Kabul and other regional hospitals (Rahimi and Azimi 2012). This shows a promising commitment toward mental health training in Afghanistan since the previous studies done in 2004.

Further showing that the future of mental health in Afghanistan is not all bleak is a recent publication on the importance of developing a culturally relevant counseling psychology degree program in Afghanistan. In the paper, researchers surveyed counselors studying at Kabul University and Herat University for their opinions on the qualities that are important for a counselor to possess. The Afghan counselors surveyed generally agreed that professionals must be knowledgeable on Afghan cultural values and customs, in addition to their expertise in international standards for counseling (Akeson et al. 2018). This is an especially important concept when it comes to leading initiatives related to autism in Afghanistan. This is because the majority of research and methods for coping with autism have been constructed in developed countries using Western individuals with autism. As the counselors recommended in the paper, it is important to consider the Afghan cultural and religious context when approaching any psychological condition.

Social Policy and Current Controversies

An important aspect of analyzing cultural perceptions of disabilities is through the lens of language. Language and words are the tools we use to understand the world around us. *Mayub* is the word in Dari (one of the two most common spoken languages in Afghanistan) that describes people who are disabled by birth through various congenital factors, diseases, or malnutrition (Bakhshi et al. 2006). *Malul* is the word that describes people who are disabled by an accident such as war, land mines, or sickness developed later in life. Finally, *diwana* is a colloquial word that relates to any impairment of the mind. This can mean intellectual disabilities as well as mental illness. People labeled as *diwana* face social stigmatization from both their communities and families (Bakhshi et al. 2006).

Unfortunately, oftentimes in Afghan society, individuals with mental disabilities are seen as *diwana* rather than *mayub*. A child who does not play with other children or does not make proper eye contact is seen as slow or intellectually challenged rather than disabled. Oftentimes families will look down upon this child and give more attention to the child who is funny, witty, or social. This causes further neglect to the child with a disability. Parents will see this as a phase or a learning problem that will fix itself over time. This can cause a major problem for children with autism by delaying the age at which the disability is detected. According to Autism Speaks, a diagnosis and intervention before the age of two maximize the progress of the child and ensure that they do not adopt harmful habits at an early age (Safa 2018). Another major challenge with mental disorders in general in Afghanistan is that oftentimes mothers are blamed for the condition of their child due to their bad parenting. This further delays the family from seeking professional help and opinions.

Cultural and religious stigmas cause problems for adults with mental disabilities and make it hard for these individuals to integrate into society (Trani and Bakhshi 2013). Studies show that in societies with social stigmas toward disabilities, individuals with such conditions are more prone to be poor and excluded from society (Braithwaite 2009). This can be in the form of social outcasting from their family or from society in general. It can cause intense feelings of shame and guilt for the individual with the disability (Trani and Bakhshi 2013). People with disabilities also face difficulty finding employment or finding partners for marriage. These problems are especially severe when the disability is due to congenital factors with no cure rather than war-related physical or mental disabilities (Trani and Bakhshi 2013). A 2004 study on the perception of disability within the Afghan community revealed a complex sentiment toward people with disabilities. War-related disabilities were viewed courageously as a noble sacrifice. This is not the sentiment with which congenital or nonwar-related disabilities are viewed (Bakhshi et al. 2006). Historically, Afghans have believed that individuals with epilepsy are possessed by *djinn* or evil spirits (Miles

2002). For individuals with autism, such religious and cultural stigmatization would further delay a proper diagnosis of the condition and thus delay treatment and rehabilitation.

Conclusion

It is evident that Afghanistan lacks the appropriate institutions and funding to uplift people with autism in the country. Although there has been significant work at a legislative level to be inclusive to people with disabilities, this work often excludes individuals with intellectual disabilities and especially people living in rural areas. A history of war has plagued Afghanistan with a national mental health crisis. This crisis includes not only mental disabilities but also post-traumatic stress disorders and various types of anxiety and depression. Furthermore, society outcasts individuals with intellectual and social disabilities such as autism as people who are *diwana*, or crazy, making it even more important for these issues to be adequately addressed. Unfortunately, international support on autism-related issues is lacking from both nongovernmental organizations and from researchers. There are no known studies looking at the prevalence of autism in Afghanistan, and organizations such as Autism Speaks do not operate in the country. The population of individuals with autism is an especially vulnerable group in Afghanistan, making it all the more necessary to build and fund institutions that support those with autism in Afghanistan.

See Also

- DSM-III
- DSM-III-R
- ICD 10 Research Diagnostic Guidelines

References and Reading

- Akesson, et al. (2018). Developing a culturally relevant counselling psychology degree programme in Afghanistan: Results from a DACUM study. *Intervention*, 16, 231–242. https://doi.org/10.4103/INTV.INTV_54_18.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Braithwaite, J. (2009). Disability and poverty: A survey of World Bank poverty assessments and implications. *Alter*, 3, 219.
- Disability rights in Afghanistan. Human Rights Based Approach at Sida, Sept 2014. www.sida.se/globalassets/sida/eng/partners/human-rights-based-approach/disability/rights-of-persons-with-disabilities-afghanistan.pdf
- Bakhshi, P., Trani, J. F., & Rolland, C. (2006). Conducting surveys on disability: A comprehensive toolkit. Brown School Faculty Publications. 50. https://openscholarship.wustl.edu/brown_facpubs/50
- Human Rights Watch. (2019). Afghanistan: Little help for conflict-linked trauma. 8 Oct 2019. www.hrw.org/news/2019/10/07/afghanistan-little-help-conflict-linked-trauma#
- Islamic Republic of Afghanistan Ministry of Public Health. (2009). National Mental Health Strategy 2009–2014.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Klin, A., Lang, J., et al. (2000). Brief report: Interrater reliability of clinical diagnosis and DSM-IV criteria for autistic disorder: Results of the DSM-IV autism field trial. *Journal of Autism & Developmental Disorders*, 30(2), 163–167.
- Miles, M. (2002). Some historical texts on disability in the classical Muslim world. *Journal of Religion, Disability & Health*, 6(2/3), 77–88.
- National Institute of Mental Health. (2018). Autism spectrum disorder. National Institute of Mental Health, U.S. Department of Health and Human Services. www.nimh.nih.gov/health/topics/autism-spectrum-disorders-asd/index.shtml. March, 2018.
- Rahimi, Y. A., & Azimi, S. (2012). War and the crisis of mental health in Afghanistan. *International Psychiatry: Bulletin of the Board of International Affairs of the Royal College of Psychiatrists*, 9(3), 55–57.
- Safa, T. (2018). *Autism speaks in Rabat and Casablanca*. Worcester Polytechnic Institute. January 29, 2018.
- Samadi, S. A., & McConkey, R. (2011). Autism in developing countries: Lessons from Iran. *Autism Research and Treatment*, 2011, 145359, 11 p. <https://doi.org/10.1155/2011/145359>.
- Sayed, G. D. (2011). *Mental health in Afghanistan: Burdens, challenges, and the way forward*. Washington, DC: The World Bank.
- Trani, J.-F., & Bakhshi, P. (2008). Challenges for assessing disability prevalence: The case of Afghanistan. *Alter*, 2(1), 44–64.
- Trani, J.-F., & Bakhshi, P. (2013). *Vulnerability and mental health in Afghanistan: Looking beyond war exposure*. *Transcultural Psychiatry*, 50(1), 108–139. <https://doi.org/10.1177/1363461512475025>
- Trani, J.-F., Bakhshi, P., Noor, A. A., & Mashkooor, A. (2009). *Lack of a will or of a way? Taking a capability approach for analysing disability policy shortcomings and ensuring programme impact in Afghanistan*. *European Journal of Development Research*, 21(2), 297–319.
- Trani, J.-F., Bakhshi, P., & Nandipati, A. (2012). ‘Delivering’ education; maintaining inequality. The case

- of children with disabilities in Afghanistan. *Cambridge Journal of Education*, 42(3), 345–365.
- Ventevogel, P., et al. (2006). Psychiatry in Afghanistan. *International Psychiatry: Bulletin of the Board of International Affairs of the Royal College of Psychiatrists*, 3(2), 36–38.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 5–41). Hoboken: Wiley.
- Volkmar, F. R., & Reichow, B. (2013). Autism in DSM-5: Progress and challenges. *Molecular Autism*, 4(1), 13. <https://doi.org/10.1186/2040-2392-4-13>.
- Volkmar, F. R., Cicchetti, D. V., et al. (1992). Three diagnostic systems for autism: DSM-III, DSM-III-R, and ICD-10. *Journal of Autism and Developmental Disorders*, 22(4), 483–492.
- Volkmar, F. R., Klin, A., et al. (1994). Field trial for autistic disorder in DSM-IV. *The American Journal of Psychiatry*, 151(9), 1361–1367.
- Volkmar, F., et al. (2014). Practice Parameter for the Assessment and Treatment of Children and Adolescents With Autism Spectrum Disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(2), 237–257.
- WHO-AIMS Report on Mental Health System in Afghanistan, WHO and Ministry of Public Health, Kabul, Afghanistan, 2006.
- World Health Organization. (1994). *Diagnostic criteria for Research*. Geneva: World Health Organization.
- World Health Organization. (2019). WHO Afghanistan Country Office 2019.

AFLS

► Assessment of Functional Living Skills (AFLS)

Age Appropriate

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Definition

Age appropriate refers to a developmental concept whereby certain activities may be deemed appropriate or inappropriate to a child's "stage" or level of development. Specific disabilities as well as lack of exposure to age-appropriate activities and

experiences are commonly thought to prevent a child from gaining the skills necessary for their current and thus their next stage of development. It is thought that development most often occurs in rather predictable stages. Although every child develops in a unique way, all children are expected to interact with their environment at an age-appropriate level. Looking at a child's functional development involves observing whether or not the child has mastered certain developmental milestones and expectations for his or her age.

With this understanding of typical child development, a child may have a special need when he or she has a delay in one or more areas of development listed below:

Body movement
Thinking and learning
Communication
Senses and their integration
Relating to self and others
Self-care and daily living skills

See Also

► Developmental Milestones

References and Reading

- Sattler, J. M., & Hoge, R. D. (2006). *Assessment of children: Behavior, social, and clinical foundations* (5th ed.). San Diego: Jerome M. Sattler.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales* (2nd ed.). Circle Pines: American Guidance Service.

Age Equivalents

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Synonyms

Mental age; Test age

Definition

Age equivalent scores provide an estimate of the chronological age at which typically developing children demonstrate the skills exhibited by the child being assessed. The age equivalent score is based on the mean raw score on a test obtained by the group of children in the normative sample at a specific age. In simple terms, if on average children at 36 months of age obtain a score of 10 correct responses on a particular test, then any child obtaining a score of 10 correct will receive an age equivalent of 36 months. Age equivalent scores are often expressed in years and months (e.g., 5–0 for 5 years, 0 months).

Though age equivalent scores are appealing in that they appear to provide convenient descriptive information, they can be misleading and do not necessarily represent the level of functioning of the individual. For example, a 3-year-old child who receives an age equivalent of 4–6 on a test of expressive vocabulary is only similar to a 4½-year-old child in the number of items answered correctly on the test and does not necessarily share other characteristics of 4-year-old level expressive language ability. As an overall average of abilities, any age equivalent score should be interpreted with caution as the child may actually possess individual skills which fall above or below that level. Especially for children with significant scatter within their profile of abilities, these summary scores may provide an oversimplified picture of the child's skills. As development of skills is not linear, age equivalent scores do not represent equal units. Therefore a 6-month delay will have a different meaning for a 2-year-old child than for a 10-year-old child. Because they are based on ordinal scales, statistical computations such as standard error of measurement cannot be performed and confidence intervals cannot be determined. Correct interpretation of age equivalent scores must take these issues into account. In spite of these limitations, age equivalent scores may be especially useful when standard scores are not available, such as when a test is administered to a child with significant developmental delays whose chronological age falls outside the normative range for that test.

See Also

► [Developmental Milestones](#)

References and Reading

- Anastasi, A. (1988). *Psychological testing*. New York: Macmillan.
- Gilliam, W. S., & Mayes, L. C. (2007). Clinical assessment of infants and toddlers. In A. Martin & F. Volkmar (Eds.), *Lewis's child and adolescent psychiatry: A comprehensive textbook* (pp. 309–322). Philadelphia: Lippincott Williams & Wilkins.
- Sattler, J. M. (2001). *Assessment of children: Cognitive applications*. San Diego: Sattler.
- Tsatsanis, K. (2007). Psychological and neuropsychological assessment of children. In A. Martin & F. Volkmar (Eds.), *Lewis's child and adolescent psychiatry: A comprehensive textbook* (pp. 357–371). Philadelphia: Lippincott Williams & Wilkins.

Age of Onset

► [Onset](#)

Age of Recognition

► [Onset](#)

Age Period Cohort Analysis

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Definition

A “cohort” is a component of the population who shares a significant experience at a certain period of time or has one or more similar characteristics. A common usage for the term is to describe people born in the same time period as a birth cohort

(or generation). In epidemiological terms, it is used to denote a group of individuals sharing a common characteristic or experience, such as the same workplace or living near a waste site, who are observed over time for disease incidence and compared to a group without the characteristic, or to a general population (e.g., cohort study).

“Cohort analysis” is the calculation and analysis of morbidity (or mortality) rates for a particular disease in a birth cohort as they pass through various ages, with different cohorts overlapping at different ages in the same calendar time period.

Historical Background

Cohort analysis began as a tool to describe and understand mortality trends and is now commonly used to identify birth cohorts at higher risk for certain diseases, providing information for both public health surveillance and for the identification of etiologic factors. Age-period-cohort (APC) analysis refers to the interpretation of temporal trends in disease incidence or mortality rates in terms of three scales all related to time: age, calendar date (period), and year of birth (cohort). An *age* effect reflects the change in disease risk as a function of the age of individuals, such as cardiovascular disease, so differences in the age structure of samples being studied could affect disease incidence rates. A *period* effect refers to a change over time that tends to affect everyone regardless of age, such as an epidemic or a food contamination. A *cohort* effect is a variation in disease risk that applies to all individuals sharing a common experience associated with being born around the same time or in the same generation, such as change in exposure to a risk factor. Disentangling these effects can be quite difficult due to their interdependence; e.g., cohort effects are tied to both age and period effects (Fombonne 1994; Keyes et al. 2010).

Various graphical and analytic methods, including parametric and nonparametric approaches, for understanding trends in disease rates have been developed and received considerable attention in the literature (Glenn 1976; Holford 1983, 2005; Keyes et al. 2010; Kupper et al. 1985; Robertson and Boyle 1998). An early

method of statistical modeling of APC data was the multiple classification model, a model containing the effects of age groups (rows), periods of observation (columns), and birth cohorts (diagonals of the age-by-period table) (Kupper et al. 1985). The interpretation of such models is difficult due to the linear dependence between the three APC variables, which must be accounted for in the models. The various models basically treat the definition of the cohort effect in a different way; simplified, some models treat age and period effects as confounders of a cohort effect whereas others model the interaction, or effect modification, of age and period on the cohort. The decision of how to treat these variables is not really a statistical issue but rather depends on the study question of interest and how it is posed. Thus, the APC models are best used to organize and summarize data, potentially pointing out directions for future research to determine the true factors for which time is acting as a proxy.

Current Knowledge

Relevance to Autism

The reporting or prevalence of autism has greatly increased over the past few decades, but the reasons for the temporal increase continue to be debated (Croen et al. 2002; Fombonne 2003; Hertz-Picciotto and Delwiche 2009; King and Bearman 2009; Parner et al. 2008; Rice et al. 2010; Schecter and Grether 2008; Idring et al. 2015; Christensen et al. 2016). Several of these studies have examined age-cohort effects and observe increases for each age group in subsequent (more recent) cohorts. The reasons most commonly cited or examined for the increase include (1) younger age at diagnosis; (2) changes in diagnostic criteria, including shifts from other diagnoses (primarily mental retardation); (3) increased awareness of autism, so that ascertainment is improved or milder cases ascertained; and (4) true changes in the frequency, possibly via introduction of, or increase in, a variety of non-genetic risk factors.

Autism diagnosis is strongly related to age of the child, so shifts to younger ages at diagnosis

could artificially inflate prevalence rates among later cohorts if not taken into account in comparisons (Parner et al. 2008). Further complicating interpretation, age at diagnosis may be related to other factors such as child gender, race/ethnicity, IQ, and degree of impairment, as well as parental education, whose distribution could differ across cohorts (Christensen et al. 2016; Shattuck et al. 2009). Studies have not shown consistent effects of diagnostic shift or substitution on temporal trends, although its occurrence has been supported (King and Bearman 2009; Leonard et al. 2010). Differences in autism rates by race/ethnicity could reflect differential awareness and thus temporal trends as awareness increases and the racial distribution of cohorts change (Rice et al. 2010; Windham et al. 2011). Alternatively, racial differences may reflect access to services, for which temporal trends may be less predictable. As there is no biologic test or marker of autism, diagnosis is somewhat subjective. The medical and psychiatric criteria for diagnosis have changed over time since the 1980s, generally broadening, which might be reflected as period effects. However, the magnitude of effect of this change on prevalence rates is not agreed upon across investigators (Leonard et al. 2010). Further, the impact of the most recent diagnostic changes (DSM-5), which can be considered a tightening of criteria, has not been fully evaluated but may lead to leveling off of the increasing rates (Bennett and Goodall 2016; Maenner et al. 2014).

Two studies using data from California calculated age and birth year (cohort) rates in order to examine the impact of various factors, but without conducting formal APC analysis (Hertz-Picciotto and Delwiche 2009; Schecter and Grether 2008). Both showed that for each successive year of birth, incidence increased for each age, although more steeply for younger children in the 2009 analysis. Similar age-cohort patterns were seen in Stockholm (Idring et al. 2015). Hertz-Picciotto and Delwiche (2009) further attempted to calculate the proportion of increased incidence due to changes in age at diagnosis, diagnostic criteria, or inclusion of milder cases and found that while these explained some, they accounted for only about

a third of the overall change. One recent study based on similar California data but using an APC model (Keyes et al. 2012) reported strong cohort effects so that each successively younger cohort had higher odds of autism diagnosis, controlling for age and period effects. They concluded that the drivers of the increase in autism must be factors that have increased linearly year to year and aggregate in birth cohorts but did not examine specific causes.

Future Directions

Explaining the reasons for temporal trends of a health condition may provide important information for identifying, and thereby potentially ameliorating, risk factors for the disease, and for planning services. Studies show rising rates of autism by birth cohort that are not fully explained by diagnostic changes or awareness, so research to explain the increase is still very much needed. A variety of risk factors, from endogenous (such as parental age shifts) to exogenous (such as environmental exposures or maternal infection), are currently being investigated (Lyall et al. 2017). Formal APC analysis might shed some light by focusing investigators on factors that vary over time by birth cohort. The one study conducted thus far was based on only one of the possible models, however, so other models might not yield consistent results.

See Also

- [Diagnostic Substitution](#)
- [Epidemiology](#)

References and Reading

- Bennett, M., & Goodall, E. (2016). A meta-analysis of DSM-5 autism diagnoses in relation to DSM-IV and DSM-IV-TR. *Review Journal of Autism and Developmental Disorders*, 3, 119–124.
- Christensen, D. L., Baio, J., Van Naarden Braun, K., Bilder, D., Charles, J., Constantino, J. N., Daniels, J., et al. (2016). Prevalence and characteristics of autism

- spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2012. *MMWR Surveillance Summary*, 65, 1–23.
- Croen, L. A., Grether, J. K., Hoogstrate, J., & Selvin, S. (2002). The changing prevalence of autism in California. *Journal of Autism and Developmental Disorders*, 32, 207–215.
- Fombonne, E. (1994). Increased rates of depression: Update of the epidemiological findings and analytical problems. *Acta Psychiatrica Scandinavica*, 90, 145–146.
- Fombonne, E. (2003). The prevalence of autism. *Journal of the American Medical Association*, 289(1), 87–89.
- Glenn, N. D. (1976). Cohort Analysts' futile quest: Statistical attempts to separate age, period, and cohort effects. *American Sociological Review*, 41, 900–905.
- Hertz-Picciotto, I., & Delwiche, L. (2009). The rise in autism and the role of age at diagnosis. *Epidemiology*, 20, 84–90.
- Holford, T. R. (1983). The estimation of age, period and cohort effects for vital rates. *Biometrics*, 39, 311–324.
- Holford, T. R. (2005). Age-period-cohort analysis. *Encyclopedia of Biostatistics*. <https://doi.org/10.1002/0470011815.b2a03003>.
- Ilding, S., Lundberg, M., Sturm, H., Dalman, C., Gumpert, C., Rai, D., Lee, B. K., & Magnusson, C. (2015). Changes in prevalence of autism spectrum disorders in 2001–2011: Findings from the Stockholm youth cohort. *Journal of Autism and Developmental Disorders*, 45(6), 1766–1773.
- Keyes, K. M., Utz, R. L., Robinson, W., & Li, G. (2010). What is a cohort effect? Comparison of three statistical methods for modeling cohort effects in obesity prevalence in the United States, 1971–2006. *Social Science & Medicine*, 70, 1100–1108.
- Keyes, K. M., Susser, E., Cheslack-Postave, K., Fountain, C., Liu, K., & Bearman, P. S. (2012). Cohort effects explain the increase in autism diagnosis among children born from 1992 to 2003 in California. *International Journal of Epidemiology*, 41(2), 495–503.
- King, M., & Bearman, P. (2009). Diagnostic change and the increased prevalence of autism. *International Journal of Epidemiology*, 38(5), 1224–1234.
- Kogan, M. D., Blumberg, S. J., Schieve, L. A., Boyle, C. A., Perrin, J. M., Ghandour, R. M., et al. (2009). Prevalence of parent-reported diagnosis of autism spectrum disorder among children in the US, 2007. *Pediatrics*, 124, 1–9.
- Kupper, L. L., Janis, J. M., Karmous, A., & Greenberg, B. G. (1985). Statistical age-period-cohort analysis: A review and critique. *Journal of Chronic Diseases*, 38(10), 811–830.
- Leonard, H., Dixon, G., Whitehouse, A. J. O., Bourke, J., Aiberti, K., Nassar, N., et al. (2010). Unpacking the complex nature of the autism epidemic. *Research in Autism Spectrum Disorders*, 4, 548–554.
- Lyall, K., Croen, L., Daniels, J., Fallin, M. D., Ladd-Acosta, C., Lee, B. K., Park, B. Y., Snyder, N. W., Schendel, D., Volk, H., Windham, G. C., & Newschaffer, C. (2017). The changing epidemiology of autism spectrum disorders. *Annual Reviews in Public Health*, 38, 81–102.
- Maenner, M. J., Rice, C. E., Ameson, C. L., Cunniff, C., Schieve, L. A., Carpenter, L. A., et al. (2014). Potential impact of DSM-5 criteria on autism spectrum disorder prevalence estimates. *JAMA Psychiatry*, 71(3), 292–300.
- Parner, E. T., Schendel, D. E., & Thorsen, P. (2008). Autism prevalence trends over time in Denmark: Changes in prevalence and age at diagnosis. *Archives of Pediatrics & Adolescent Medicine*, 162(12), 1150–1156.
- Rice, C., Nicholas, J., Baio, J., Pettygrove, S., Lee, L.-C., Braun, K. V. N., et al. (2010). Changes in autism spectrum disorder prevalence in 4 areas of the United States. *Disability and Health Journal*, 3(3), 186–201.
- Robertson, C., & Boyle, P. (1998). Age-period-cohort analysis of chronic disease rates I: Modeling approach. *Statistics in Medicine*, 17(12), 1305–1323.
- Schecter, R., & Grether, J. K. (2008). Continuing increases in autism reported to California's developmental services system. *Archives of General Psychiatry*, 65, 19–24.
- Shattuck, P. T., Durkin, M., Maenner, M., Newschaffer, C., Mandell, D. S., Wiggins, L., et al. (2009). Timing of identification among children with an autism spectrum disorder: Findings from a population based surveillance study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(5), 474–483.
- Windham, G. C., Anderson, M. C., Croen, L. A., Smith, K. S., Collins, J., & Grether, J. K. (2011). Birth prevalence of autism spectrum disorders in the San Francisco Bay Area by demographic and ascertainment source characteristics. *Journal of Autism and Developmental Disorders*, 41, 1362–1372.

Agenesis of Corpus Callosum

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Synonyms

Acallosal syndrome; Callosotomy (surgical severing); Complete agenesis; Dysgenesis (malformation); Hypogenesis (partial formation); Hypoplasia (underdevelopment); Partial agenesis

Short Description or Definition

The corpus callosum (CC) is the largest white matter fiber tract (also known as commissure) connecting the two hemispheres of the human brain. Agenesis of the corpus callosum (AgCC) is present at birth and encompasses structural defects of the development of the corpus callosum that range from partial to complete loss of these connective fiber tracts. Primary AgCC is a complete loss of the CC without other accompanying brain changes. A rare individual diagnosed with ASD is found to have AgCC.

Categorization

The CC is generally divided into seven subregions.

AgCC is divided into *partial* (Fig. 1) and *complete* (Fig. 2) based on whether one or more subregions are missing (Fig. 1) or whether the entire CC is absent (Fig. 2). In both cases, the anterior commissure – a smaller white matter tract connecting ventral frontal regions – is almost always intact, with abnormalities limited to the CC.

Commissurotomy is a surgical procedure that typically involves severing all fiber tracts connecting the hemispheres, including the CC, to treat intractable epilepsy in which seizures that start in one hemisphere propagate to the

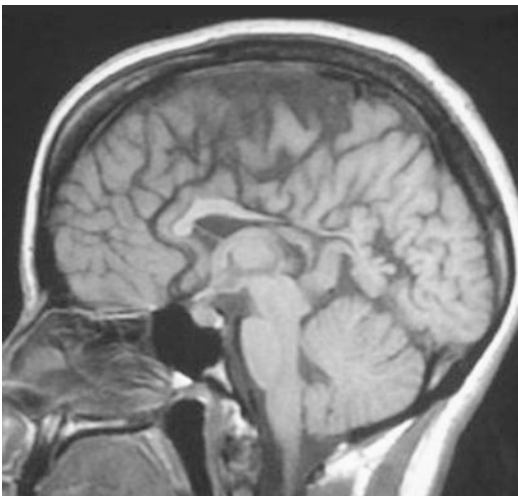
other hemisphere. Callosotomy is a subtype of commissurotomy that involves only severing of the CC.

Epidemiology

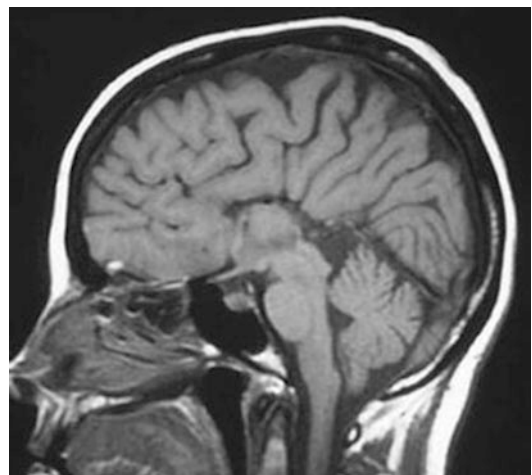
AgCC has been estimated to occur in at least 1 in 4,000 births, with one study identifying two cases in 2,309 neonates (Wang et al. 2004). AgCC is often associated with other developmental or neurogenetic syndromes including Arnold-Chiari malformation, Dandy-Walker syndrome, Aicardi's syndrome, holoprosencephaly, and numerous others. Approximately 30–45% of AgCC cases have currently identifiable genetic syndromes or chromosomal abnormalities. This percentage is likely to increase with advances in the sensitivity of genetic testing and the identification of new genetic disorders (Paul et al. 2007). For example, deletions on chromosome 1q42-q44 (Boland et al. 2007) and 14q12 (Shoicet et al. 2005), as well as multiple other chromosomal abnormalities (O'Driscoll et al. 2010), have been more recently identified.

Natural History, Prognostic Factors, and Outcomes

The CC is the largest and most important structure for interhemispheric transfer of information. It



Agenesis of Corpus Callosum, Fig. 1 Partial AgCC



Agenesis of Corpus Callosum, Fig. 2 Complete AgCC

contains fibers that connect both homotopic and heterotopic interhemispheric regions, meaning some fibers connect regions of the left and right hemispheres that are directly analogous (e.g., the left and right superior temporal regions), while others connect regions that are not directly analogous (e.g., the left superior temporal and right middle temporal regions). CC function was first examined by studying the cognitive skills of individuals who underwent commissurotomy or callosotomy, often called “split-brain” patients. These studies were useful for demonstrating specialization of the left and right halves of the brain. However, because these patients typically had surgical severing later in life, these studies did not provide information about the contributions of the CC to cognitive and brain development. More recent studies of babies and children with AgCC are providing data about the developmental role of the CC.

AgCC involves abnormal formation of the CC, typically between the third and 12th weeks of pregnancy, and is observable at birth via neuroimaging methods such as MRI. It appears that both genetic and environmental factors can play a role in the development of AgCC. Several genetic disorders and syndromes have been associated with AgCC, and evidence from animal work has shown the important roles specific genes play in the normal development of the CC. For example, individuals with X-linked lissencephaly (meaning “smooth brain”) have a mutation in the ARX gene and exhibit AgCC. In the developing brain, ARX proteins are involved with neuronal migration and deficient levels cause abnormal cell cycling and impair the migration of neurons (Friocourt et al. 2008) that should ultimately form the CC. AgCC also occurs in the context of in utero exposure to alcohol with <7% of individuals with fetal alcohol syndrome showing near complete AgCC and a greater proportion having partial AgCC or other CC malformations (Roebuck et al. 1998).

The cognitive impairments associated with AgCC are quite variable, although some consistent findings have emerged. Studies of younger children have identified developmental delay, learning difficulties, or behavior problems in the majority of AgCC cases (Goodyear et al. 2001;

Shevell 2002). Individuals with complete AgCC may have worse cognitive function and outcomes (Paul et al. 2007), although this has not been consistent across studies. Interestingly, although AgCC patients tend to show at least mild cognitive or behavioral difficulties, they do not exhibit the classic disconnection pattern shown by adult “split-brain” patients who had all commissures surgically severed, including the CC. Thus, individuals with congenital AgCC frequently show intact ability to transfer visual and auditory information across the left and right hemispheres. This may be because most AgCC patients have an intact anterior commissure and this structure may support some compensation of interhemispheric transfer.

AgCC can have substantial impact on specific cognitive functions. For example, many individuals with AgCC show significant differences in their verbal and nonverbal (visual) abilities, although which area is stronger varies across individuals (Chiarello 1980; Sauerwein et al. 1994). The most prominent deficits are in complex tasks that involve integration of multiple facets of information or rapid processing of complex arrays of stimuli. Thus, impairments may involve abstract reasoning (Brown and Paul 2000), problem solving (Fischer et al. 1992), and the ability to generalize a rule from one situation to another (Solursh et al. 1965) or to quickly generate examples from a category (e.g., specific names of animals or fruits and vegetables) (David et al. 1993). Deficits have also been observed in understanding pragmatic aspects of language, including problems in understanding idioms, metaphors, sarcasm, and other forms of nonliteral language and humor. Individuals with AgCC often show alexithymia or difficulty with verbally reporting emotional states and experiences. Parents of individuals with AgCC also frequently report social skill deficits.

Not surprisingly, given deficits in the processing of complex social and contextual information and parent reports of social weaknesses, AgCC has been identified in individuals diagnosed with ASD, or perhaps more accurately, some individuals with AgCC have been diagnosed with ASD. However, it is important to note that the vast majority of individuals with

ASD do not have AgCC and not all individuals with AgCC would be diagnosed with ASD. One study that sought to examine the prevalence of ASD-related symptoms in individuals with AgCC reported that 45% of children, 35% of adolescents, and 18% of adults with AgCC met criteria for ASD on the Autism Spectrum Quotient, a parent- and self-report screening tool for autism (Lau et al. 2013). Thus, the two conditions overlap, but are not redundant. The best-known example of this overlap is Kim Peek, the inspiration for the movie *Rain Man* who was widely known for his savant skills. These skills included photographic memory and an amazing ability to read and remember vast amounts of information in a short period of time. However, Kim Peek was not a typical example of primary AgCC because, in addition to having complete AgCC, he also was missing the anterior commissure, had macrocephaly and cerebellar malformation, and may have had a genetic syndrome (FG or Opitz-Kaveggia syndrome) linked to the X chromosome.

Adult outcomes of AgCC, even primary AgCC, are highly variable with some individuals showing intact overall ability and functioning and others showing significant intellectual disability and dependence on caregivers for even basic needs. Important prognostic factors related to this variability may include the level of agenesis (partial or complete) and the extent of other brain abnormalities.

Clinical Expression and Pathophysiology

As discussed above, clinical features are highly variable but include a wide range of deficits in general cognitive ability, large differences between verbal and nonverbal abilities, fairly consistent deficits in specific tasks that require rapid processing of complex information, social perception and skill weaknesses, and impairments in identifying/describing emotions (alexithymia). Developmental manifestations are not well known but are likely to be also highly variable with some individuals showing mild early delays with relatively intact functioning later in life and others showing consistently low levels of ability and functioning throughout the life span.

Evaluation and Differential Diagnosis

Asymptomatic AgCC is by definition hard to identify or diagnose since neuroimaging studies are not conducted without an indication. However, with prenatal ultrasound examination becoming more common and prenatal MRI being further developed, it is possible that identification of CC abnormalities may become more frequent. These alterations can be detected at 20 weeks of gestation, and once identified, associated features should be investigated (Vergani et al. 1994). In the majority of cases, a specific syndrome is diagnosed either during pregnancy or immediately after birth. In young children and older individuals, the presence of associated features such as seizures or developmental delays can prompt a comprehensive evaluation, including brain imaging that leads to diagnosis (see ► “American Academy of Neurology”). When AgCC is associated with a neurogenetic condition, the clinical features of this syndrome will be more evident. The list of conditions associated with anomalies of the CC is long and includes Chiari II malformations, Andermann’s syndrome (intellectual disability and polyneuropathy), and Joubert’s syndrome type III (absence of cerebellar vermis and polymicrogyria).

Treatment

There is no treatment for complete or partial AgCC. CC fibers will not regenerate and appropriately localize after that initial in utero critical period. However, given continued investigation into brain plasticity, even in adults, and a greater appreciation for the efficacy of early, intensive behavioral intervention and training, patients with AgCC may have therapeutic options in the future to help optimize their adaptive functioning. For instance, through greater understanding of the genes and biological pathways involved, it may be possible in the future for a combination of early detection and personalized genetic therapies addressing the specific molecular problems to optimize long-term outcomes in individuals with AgCC. Certainly, eliminating alcohol use in pregnancy, particularly in the first trimester, will reduce the number of cases of AgCC.

See Also

- [Corpus Callosum](#)
- [Corpus Callosum Abnormalities in Autism](#)

References and Reading

- Boland, E., Clayton-Smith, J., Woo, V. G., McKee, S., Manson, F. D. C., Medne, L., ... & Sherr, E. H. (2007). Mapping of deletion and translocation breakpoints in 1q44 implicates the serine/threonine kinase AKT3 in postnatal microcephaly and agenesis of the corpus callosum. *The American Journal of Human Genetics*, 81(2), 292–303.
- Brown, W. S., & Paul, L. K. (2000). Cognitive and psychosocial deficits in agenesis of the corpus callosum with normal intelligence. *Cognitive Neuropsychiatry*, 5, 135–157.
- Chiarello, C. (1980). A house divided? Cognitive functioning with callosal agenesis. *Brain and Language*, 11(1), 128–158.
- David, A. S., Wacharasindhu, A., & Lishman, W. A. (1993). Severe psychiatric disturbance and abnormalities of the corpus callosum: Review and case series. *Journal of Neurology, Neurosurgery, and Psychiatry*, 56(1), 85–93.
- Fischer, M., Ryan, S. B., & Dobyns, W. B. (1992). Mechanisms of interhemispheric transfer and patterns of cognitive function in acallosal patients of normal intelligence. *Archives of Neurology*, 49(3), 271–277.
- Friocourt, G., Kanatani, S., Tabata, H., Yozu, M., Takahashi, T., Antypa, M., ... & Parnavelas, J. G. (2008). Cell-autonomous roles of ARX in cell proliferation and neuronal migration during corticogenesis. *The Journal of Neuroscience*, 28(22), 5794–5805.
- Goodyear, P. W., Bannister, C. M., Russell, S., & Rimmer, S. (2001). Outcome in prenatally diagnosed fetal agenesis of the corpus callosum. *Fetal Diagnosis and Therapy*, 16(3), 139–145. <https://doi.org/10.1159/000053898> (pii).
- Lau, Y. C., Hinkley, L. B. N., Bukshpun, P., Strominger, Z. A., Wakahiro, M. L. J., Baron-Cohen, S., ... & Marco, E. J. (2013). Autism traits in individuals with agenesis of the corpus callosum. *The Journal of Autism and Developmental Disorders*, 43(5), 1106–1118.
- O'Driscoll, M. C., Black, G. C. M., Clayton-Smith, J., Sherr, E. H., & Dobyns, W. B. (2010). Identification of genomic loci contributing to agenesis of the corpus callosum. *The American Journal of Medical Genetics*, 152A(9), 2145–2159.
- Paul, L. K., Brown, W. S., Adolphs, R., Tyszka, J. M., Richards, L. J., & Mukherjee, P. (2007). Agenesis of the corpus callosum: Genetic, developmental and functional aspects of connectivity. *Nature Reviews Neuroscience*, 8, 287–299.
- Roebuck, T. M., Mattson, S. N., & Riley, E. P. (1998). A review of the neuroanatomical findings in children with fetal alcohol syndrome or prenatal exposure to alcohol. *Alcoholism, Clinical and Experimental Research*, 22(2), 339–344.
- Sauerwein, H. C., Nolin, P., & Lasonde, M. (1994). *Callosal agenesis: A natural split brain*. New York: Plenum.
- Shevell, M. I. (2002). Clinical and diagnostic profile of agenesis of the corpus callosum. *Journal of Child Neurology*, 17(12), 896–900.
- Shoicet, S. A., Kunde, S.-A., Viertel, P., Schell-Apacik, C., von Voss, H., Tommerup, N., ... & Kalscheuer, V. M. (2005). Haploinsufficiency of novel FOXG1B variants in a patient with severe mental retardation, brain malformations and microcephaly. *Human Genetics*, 117(6), 536–544.
- Solursh, L. P., Margulies, A. I., Ashem, B., & Stasiak, E. A. (1965). The relationships of agenesis of the corpus callosum to perception and learning. *The Journal of Nervous and Mental Disease*, 141(2), 180–189.
- Vergani, P., Ghidini, A., Strobel, N., Locatelli, A., Mariani, S., Bertalero, C., & Cavallone, M. (1994). Prognostic indicators in the prenatal diagnosis of agenesis of corpus callosum. *American Journal of Obstetrics and Gynecology*, 170(3), 753–758.
- Wang, L. W., Huang, C. C., & Yeh, T. F. (2004). Major brain lesions detected on sonographic screening of apparently normal term neonates. *Neuroradiology*, 46(5), 368–373. <https://doi.org/10.1007/s00234-003-1160-4>.

Ages and Stages Learning Activities

- [Ages and Stages Questionnaire, Second Edition](#)

Ages and Stages Questionnaire, Second Edition

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Synonyms

[Ages and stages learning activities](#); [ASQ family access](#); [ASQ Hub \(for monitoring screening programs of multiple organizations\)](#); [ASQ Pro \(for single-site programs\)](#) and [ASQ Enterprise \(for multisite programs\)](#); [ASQ:SE](#); [ASQ-3 Materials Kit](#); [ASQ-3™](#)

Description

The Ages and Stages Questionnaires (ASQ-3): A Parent-Completed Child Monitoring System, Third Edition (Squires et al. 2009) is a first-level comprehensive screening and monitoring program designed to identify infants and young children who require more extensive assessment to determine whether early intervention is warranted. It is designed to be easy to administer, low-cost, and appropriate for diverse populations, including children suspected of having an autism spectrum disorder. The monitoring system consists of two measures and associated user materials, the ASQ-3 and the ASQ:SE.

The ASQ-3 includes 21 questionnaires, one for each of the following ages: 2, 3, 6, 8, 9, 10, 12, 14, 16, 18, 20, 22, 24, 27, 30, 33, 36, 42, 48, 54, and 60 months. The questionnaires are designed to be completed by parents or other primary caregivers in less than 15 min and scored by a professional in less than 3 min. Each questionnaire contains 30 developmental items which are organized into five areas: Communication, Gross Motor, Fine Motor, Problem Solving, and Personal-Social. An Overall section addresses general parental concerns. In order to make the questionnaires user-friendly, items are written at a fourth to sixth grade level and illustrations are provided. For the 30 developmental items on each questionnaire, parents mark “yes” to indicate that their child performs the behavior, “sometimes” to indicate an occasional or emerging response, or “not yet” to indicate that their child does not yet perform the behavior. During the scoring process, each response is converted to a point value, values are totaled, and totals are then compared to established screening cutoff points. Each questionnaire comes with instructions, an information sheet for identification, activities for each social-emotional area, and an information summary sheet for scoring and general comments.

The ASQ:SE, which is used to screen for the presence of social-emotional delays, includes eight questionnaires, one for each of the following ages: 6, 12, 18, 24, 30, 36, 48, and 60 months. The measure addresses seven social-emotional areas: self-regulation, compliance, communication,

adaptive behaviors, autonomy, affect, and interaction with people. Like the ASQ-3, the ASQ:SE relies on parents to observe their child and complete the measure. Each questionnaire discusses social-emotional activities tied to the age of the child being screened, and by virtue of completing the questionnaire, parents learn about social-emotional milestones as well as their child’s strengths and vulnerabilities. For each item, parents mark “most of the time” to indicate that their child performs the behavior, “sometimes” to indicate an occasional or emerging response, or “rarely or never” to indicate that their child does not yet perform the behavior. Parents are also asked to check whether the behavior in question is a concern for them, and space is provided to allow for open-ended, narrative responding about certain aspects of their child’s social-emotional development. Much like the ASQ-3, each response is converted to a point value, values are totaled, and totals are then compared to established screening cutoff points. Following completion of the questionnaire, professionals may proceed with a referral for additional assessment and/or provide learning activity sheets to support the parent in further promoting social-emotional development.

Historical Background

In the 1970s, researchers at the University of Oregon, led by Dr. Diane Bricker, recognized the need for economical, valid, and culturally sensitive screening tools to identify young children who might be at risk for developmental delays. Following a landmark study on parents’ ability to report on their child’s early development (Knobloch et al. 1979), researchers including Dr. Bricker and Dr. Jane Squires conducted an extensive review of standardized developmental assessments and associated literature, and they selected a set of skills easily observed or elicited by parents within the home environment. Using these skills, Drs. Bricker and Squires created a series of questionnaires that asked parents simple questions about their child’s development. In response to pilot data, the questionnaires were expanded and refined, and in 1995, the

questionnaires were first published commercially by Brookes Publishing as the Ages & Stages Questionnaires® (ASQ): A Parent-Completed, Child-Monitoring System. In 1999, a revised and expanded edition of ASQ was published based on continuing research and user feedback. Data collection on the third edition, ASQ-3, began in 2002, and in 2009, the revised measure was published and an online management and questionnaire completion system was launched.

The ASQ:SE was created in response to growing demand for a screening tool for social-emotional concerns in young children. In 1995, the development process was initiated, and the first version of the Ages & Stages Questionnaires®: Social-Emotional (ASQ:SE) took form. Items in the early version of the ASQ:SE were developed using multiple sources, such as standardized social-emotional and developmental assessments, textbooks and other resources in developmental and abnormal psychology, language and communication materials, and education and intervention resources. In 1996, validity, reliability, and utility studies on a field-test version of the ASQ:SE were initiated. The field-test version was called the Behavior-Ages & Stages Questionnaires (B-ASQ; Squires et al. 1996). Following initial refinement, studies continued between 1996 and 2001 to determine the psychometric properties of the screening instrument, and in 2002, the Ages & Stages Questionnaires®: Social-Emotional (ASQ:SE): A Parent-Completed, Child-Monitoring System for Social-Emotional Behaviors was first published commercially by Brookes Publishing. Research on ASQ:SE is ongoing.

Psychometric Data

ASQ:SE: According to the publisher, normative data for the ASQ:SE were based on 3014 completed questionnaires, and validity studies were conducted using 1041 children. Internal consistency measured by coefficient alpha was found to be high across intervals, ranging from .67 to .91 with an overall alpha of .82. Test-retest reliability, measured as the agreement between two ASQ:SE questionnaires completed by parents at 1- to

3-week intervals was 94%. Sensitivity ranged from 71% at 24 months to 85% at 60 months, with 78% overall sensitivity. Specificity of the questionnaires ranged from 90% at 30 months to 98% at 6 months, with 94% overall. Percent agreement between questionnaires and standardized assessments/disability status ranged from 88% at 30 months to 94% at 60 months, with overall agreement of 92%. Under-referral ranged from 2.4% at 60 months to 4.7% at 12 months, while over-referral ranged from 3.0% at 18 months to 8.6% at 30 months. The ability of the ASQ:SE to detect atypical social-emotional development (sensitivity) was generally lower across intervals, while specificity, or the ability of the ASQ:SE to correctly identify typically developing children, was high. Specificity may have been elevated in the 6-, 12-, and 18-month intervals because of the large number of “identified” children in these samples and the small number of low-moderate risk children.

ASQ-3: The ASQ-3 has a new standardization based on a sample that closely mirrors the US population in geography and ethnicity and includes children of all socioeconomic statuses. The sample includes 15,138 children whose parents completed 18,232 questionnaires. According to the publisher, reliability, validity, sensitivity, and specificity are all excellent: Test-Retest Reliability = .92; Inter-rater Reliability = .93; Validity = .82 to .88; Sensitivity = .86; Specificity = .85.

Clinical Uses

Parents or caregivers complete the ASQ-3 and ASQ:SE questionnaires independently, or, if necessary, with the assistance of a professional. With online questionnaire completion through the web-based ASQ Family Access, parents are able to complete the ASQ-3 anytime, anywhere. The ASQ-3 and ASQ:SE questionnaires can also be completed on paper at home; during home visits by nurses, social workers, or program staff; in waiting areas; or in educational centers. According to the authors, the measures can be adapted to a variety of settings, including primary

care clinics, child care settings, and teen parenting programs. Both measures are designed for easy use and generally require little training, although it is important for professionals to be familiar with the information contained in the User's Guide. Many programs use the available DVD training tools to introduce the ASQ and show staff how to screen, score, and interpret results, and for programs desiring more training, the publishing company regularly hosts remote and on-site training seminars.

See Also

- [Developmental Milestones](#)
- [Early Intervention](#)
- [Screening Measures](#)

References and Reading

- Knobloch, H., Stevens, F., Malone, A., Ellison, P., & Risemberg, H. (1979). The validity of parental reporting of infant development. *Pediatrics*, 63(6), 872–878.
- Squires, J., Bricker, D., Twombly, E., Yockelson, S., & Kim, Y. (1996). *Behavior-ages and stages questionnaires*. Eugene: University of Oregon, Center on Human Development.
- Squires, J., Bricker, D., Twombly, E., Nickel, R., Clifford, J., Murphy, K., Hoselton, R., Potter, L., Mounts, L., & Farrell, J. (2009). *Ages & stages questionnaires*® (3rd ed.. (ASQ-3™)). Baltimore: Paul H. Brookes.

Agnosia

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Definition

It is a partial or complete loss of the ability to recognize and identify familiar objects or persons through sensory stimuli. The specific sense is not defective nor is there any significant memory loss. People with agnosia may retain their cognitive abilities in other areas.

Agnosia may affect any of the senses and is classified accordingly as auditory, visual, olfactory, gustatory, or tactile agnosia. It can result from strokes, dementia, or other neurological disorders and illnesses. It may also be trauma-induced by a head injury, brain infection, or hereditary. Some forms of agnosia have been found to be genetic. It often results from damage to specific brain areas in the occipital or parietal lobes of the brain (Kolb and Whishaw 2003).

Agnosia is found in Landau-Kleffner syndrome, a disorder that is included on the differential diagnosis for autism (Johnson and Myers 2007). Landau-Kleffner syndrome (also known as LKS and acquired epileptic aphasia) is a rare childhood neurological disorder characterized by the loss of previously acquired language milestones, an inability to understand the spoken word and an abnormal electroencephalogram (EEG). These children develop normally until between the ages of 3 to 6 in contrast to autism, which is manifest prior to the age of 3 (Landau and Kleffner 1957; Teplin 1999).

Regardless of cause, there is no direct cure for the agnosia. Patients may improve if information is presented in other modalities than the damaged one. Different types of therapies can help to reverse the effects of agnosia. In some cases, occupational therapy or speech therapy can improve agnosia, depending on its etiology.

See Also

- [Aphasia](#)
- [Electroencephalogram \(EEG\)](#)
- [Inferior Parietal Area](#)
- [Occipital Lobe](#)
- [Occupational Therapy \(OT\)](#)
- [Speech Therapy](#)

References and Reading

- Johnson, C. P., & Myers, S. M. (2007). American Academy of Pediatrics Council on Children with Disabilities. Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120(5), 1183–1215.
- Kolb, B., & Whishaw, Q. (2003). *Fundamentals of human neuropsychology*. New York: Worth.

- Landau, W. M., & Kleffner, F. R. (1957). Syndrome of acquired aphasia with convulsive disorder in children. *Neurology*, 7(8), 523–530.
- Teplin, S. W. (1999). Autism and related disorders. In M. D. Levine, W. B. Carey, & A. C. Crocker (Eds.), *Developmental behavioral pediatrics* (3rd ed., p. 589). Philadelphia: WB Saunders.

Agraphia

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Synonyms

[Acquired dysgraphia](#)

Definition

Agraphia is an impairment or loss in the ability to write in individuals (most often adults) who had typical spelling and/or handwriting prior to brain damage, either sudden or progressive. Agraphia occurs as a result of damage to the cognitive, linguistic, and/or sensorimotor areas of the brain that support spelling and writing (Beeson and Rapczak 2004). Lesions in specific regions in these areas affect the ability to retrieve words and/or their spellings and/or to form the letters to write the words.

Agraphia may be broadly categorized into two types: central or peripheral. Central agraphia affects an individual's ability to spell, while peripheral agraphia is characterized by handwriting difficulties (Beeson and Rapczak 2004). Additionally, visual perceptual changes that impair handwriting are not uncommon in those with brain injury.

Although the characteristics of agraphia are similar to those of developmental dysgraphia, the defining feature of agraphia is a history of typical writing skills before writing difficulties appeared; therefore, agraphia is not seen in children and adolescents with autism spectrum disorders (ASD).

See Also

► [Dysgraphia](#)

References and Reading

- Beeson, P. M., & Rapczak, S. Z. (2004). Agraphia. In R. D. Kent (Ed.), *The MIT encyclopedia of communication disorders* (pp. 233–236). Cambridge, MA: MIT Press.

Aicardi Syndrome

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Definition

A rare genetic in which the corpus callosum (the major connection between the right and left hemispheres of the brain) is either totally or partially missing. It is associated with other abnormalities including seizures and a form of infantile spasms as well as characteristic eye abnormalities. It is thought likely that the source of the condition is on the X chromosome (it is observed only in girls or in boys with Klinefelter's syndrome); it is also possible that the condition is lethal to males with typical XY chromosome patterns – i.e., that the pregnancies miscarry.

First recognized by Jean Aicardi, a French neurologist, in 1976, the condition usually has its onset in the first months of life. The condition is rare. Although very likely to have a genetic cause, it is thought that all cases arise as a result of new mutations.

Treatment involves symptomatic management and treatment of associated problems, e.g., seizures, feeding problems, and sometimes hydrocephalus. Although outcome appears to vary, the condition is associated with significant cognitive delays.

See Also

- [Infantile Spasms/West Syndrome](#)

References and Reading

- Booth, R., Wallace, G. L., et al. (2011). Connectivity and the corpus callosum in autism spectrum conditions: Insights from comparison of autism and callosal agenesis. *Progress in Brain Research*, 189, 303–317.
- Glasmacher, M. A., Sutton, V. R., Hopkins, B., Eble, T., Lewis, R. A., Park Parsons, D., et al. (2007). Phenotype and management of Aicardi syndrome: New findings from a survey of 69 children. *Journal of Child Neurology*, 22, 176–184.
- Kinsman, S. L., & Johnston, M. V. (2007). Chapter 592. Congenital abnormalities of the central nervous system. In R. M. Kliegman, R. E. Behrman, H. B. Jenson, & B. F. Stanton (Eds.), *Nelson textbook of pediatrics* (18th ed.). Philadelphia: Saunders Elsevier.

Aide

- [Para-educator](#)
- [Paraprofessional](#)

AIMS

- [Abnormal Involuntary Movement Scale](#)

Akathisia

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Definition

AKATHISIA: Akathisia is an adverse medication effect described as an unpleasant feeling of restlessness. It is most often associated with antipsychotic

medication, but may occur with antidepressant medications such as serotonin reuptake inhibitors. In the class of antipsychotic medications, akathisia is more likely to occur with the older antipsychotics such as haloperidol, but it may occur with the newer antipsychotics such as risperidone. Patients typically describe a feeling of internal restlessness and an inability to sit still. The observer might see the patient jiggling a leg while sitting or even kicking the leg out from the sitting position. In more extreme cases, the person may be unable to sit at all and will get up and pace around the room.

The cause of akathisia is not completely understood. It usually does not improve with anticholinergic medications that are often effective for other neurologically based adverse effects of antipsychotic medications. The first response is to lower the medication, beta blockers, or switching to another antipsychotic medication may be helpful.

BARNES AKATHISIA SCALE: Barnes Akathisia Rating Scale (BAS) was introduced in late 1980s. It consists of four items that are divided into objective item, subjective item, and an overall global scale. It is the most commonly used scale for measuring akathisia.

References and Reading

- Barnes, T. (1989). A rating scale for drug-induced akathisia. *The British Journal of Psychiatry: the Journal of Mental Science*, 154, 672–676.

Alcohol-Related Neurodevelopmental Disorder

- [Fetal Alcohol Spectrum Disorder](#)

ALD

- [Adrenoleukodystrophy](#)

Aler-Cap [OTC]

- [Diphenhydramine](#)

Aler-Dryl [OTC]

► [Diphenhydramine](#)

Aler-Tab [OTC]

► [Diphenhydramine](#)

Alexithymia

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Autism: Definition

The word autism comes from the Greek word “auto” and can be translated to selfish-ism. The autism spectrum is also called autism spectrum disorders (ASD) or autism spectrum conditions (ASC), with the adjective autistic sometimes replacing the noun autism.

Categorization

Autism is a spectrum of psychological conditions characterized by widespread abnormalities of social interactions and communication as well as restricted interests and repetitive behavior (<http://www.statemaster.com/encyclopedia/Asperger-Syndrome>). Asperger’s syndrome can be seen as a mild form of autism, and in DSM-5, Asperger’s was moved to sort under autism.

Natural History

Autism was first described in 1911 by Swiss psychiatrist Eugen Bleuler (1857–1939) who mentioned, not too distinctly, two forms: schizophrenic autism and infantile autism. Both terms have been taken up by later psychiatrists and applied, confusingly, to a number of apparently different groups of disordered and socially isolated individuals (Tantam 1980). While John Langdon Down (1828–1896) is best known for having described

Down’s syndrome (trisomy 21) and savant syndrome in his 1887 lecture, he made an additional very astute observation about what he called “developmental retardation.” Today, that condition is known as autistic disorder (Treffert 2006).

Some people with autism spectrum disorders give grandiose and detailed descriptions of their special interests. That grandiose factor points to a parallel between narcissism (a.k.a. “psychopathy light”) and alexithymia. All these disorders belong to the (old psychiatry) borderline group, while others are shy, passive in contact with other people, or vice versa: intrusive and talkative.

Michael Rutter (1978) held that all reports of autism must begin with the book *Autistic Disturbances of Affective Contact* (1943) by Leo Kanner (1894–1981) where he described autism and held that the disorder probably was due to an inherited deficiency.

The characteristic symptoms of autism can be summarized as being rigid, a lack of social or emotional reciprocity, reduced communication skills, and limited interests – the last, however, can be very sophisticated (DSM-IV). Author Malika Mokeddem, herself an autist, was addressed by a friend: “You talk like a book.” If an autistic child has difficulties in imagining feelings of other people that may be because such a child has a biological worldview. A personality theory based on a biological approach differs from a psychological theory. Therefore, the claim that autistic children would be able to assess other people’s personality is unlikely to be true. (Pragmatic) autistic children, however, are adept at determining if a person really loves them. They don’t talk to a person who just talks in a friendly way, but does not help them – and as such, one might add that they would not be in the first line of being victims of psychopaths, unless their curiosity takes over. Hans Asperger (1906–1980) wrote (according to Frith 1991): “To our great surprise, we have seen how autistic individuals with good cognitive ability almost always achieve professional success, often in highly specialized academic professions and in very high positions, preferably within theoretical fields” and “A number of successful musicians were treated at our clinic when they were children. This seemingly

surprising fact what such difficult and abnormal children can achieve at acceptable and even high levels of social integration can be explained if one studies this phenomenon somewhat closer.”

“Autistic children may . . . possess a surprisingly well-developed understanding of art and have a solid understanding when distinguishing between genuine art and kitsch. They may even understand works of art that many adults perceive as difficult, such as Romanesque sculptures and paintings by Rembrandt. Autistic individuals may correctly perceive both the events unfolding on the board as to what lies behind, including portrayed individuals’ characters and the mood that pervades the painting. One should not forget that many adults never achieve an equally well-developed understanding of art. This skill is closely related to autistic people’s tendency to engage in a very special form of introspection and their ability to judge other people’s characters.”

Related Studies of the Brain

Research on twins by Folstein and Rutter published in *Nature* 1977 demonstrated the genetic influence on autism. They later wrote that “autism is one of the best validated child psychiatric disorders that exist.” Here are clear key concepts and empirically demonstrated genetic influences (Bailey et al. 1996).

Although brain imaging with MRI has revealed specific anomalies in white and folded gray brain tissue for several of those disorders or conditions, the precise relationship between structural changes and changes in neuronal function and relations remains unclear. Over the past decade, great hopes have been directed toward brain imaging techniques such as PET, SPECT, and functional MRI (fMRI). Torsten Wiesel, Nobel Prize winner in Physiology or Medicine 1981, expressed his surprise at how the research field around the brain and vision has been developed and expressed a wish for more specific cell studies instead of just using MRI for locating different brain functions. Hope of early diagnosis may ultimately be directed toward integration of fMRI, cell studies, and specific genetic tests.

Gender Issues

Hans Asperger said (according to Frith 1991) that he had never encountered a girl with fully

developed autism, and in his textbook of 1952, he reaffirms that all young girls he had met with a full-blown autism had acquired the disease after suspected encephalitis. In the past, such girls were called pseudopsychopaths or were diagnosed ADHD. But those young girls who exhibited the typical Asperger’s variant of autism are now well documented; the fact remains that the vast majority are boys (Frith 1991). A gender-linked form of inheritance is fully consistent with this pattern. The genetic causes of autism are not fully known. Social expectations of boys versus girls should also be observed. In a 2011 summary, Michael Rutter reported significant progress during the years 2007–2010 understanding autism. A little later in 2011, Andrew Whitehouse et al. published their article that autistic traits can be early detected and that they have a rather moderate stability from early childhood into adolescence.

Autism Disorder, Autism Spectrum, Autism Spectrum Disorders, or . . . ?

There is no univocal definition of the term autism spectrum. Connotated terms are attached like epilepsy, learning difficulties, delayed language development, self-injurious behavior, stomach problems, ADHD, tics, clumsiness, etc. Through a critical review of 69 research studies carried out between 1981 and 2010, Sharma et al. (2011) showed that six possible criteria (specifically the age at which signs and symptoms related to autism become apparent, language and social communication abilities, intellectual abilities, motor or movement skills, repetitive patterns of behavior, and the nature of social interaction) for diagnosing Asperger’s overlap with the criteria for diagnosing autism.

Hippler and Klicpera (2005) looked at Hans Asperger’s original data for the period 1950–1986 ($n = 181$) and performed a quantification of the two groups “autistic psychopathy” (AP) and autistic character (AZ). The latter group did produce less severe symptoms, higher intelligence, and communicative difficulties commonly associated with Asperger’s, while the AP group showed a broader symptom picture. Hans Asperger’s old term autistic psychopathy or elements of

psychopathy in autism is still used by some authors as well as in the *ICD-10*. It would be preferable to write autistic personality disorder or autistic personality spectrum. One of the cores of what we understand about psychopathy (nonexistent morals) has no place in an autism-Asperger's diagnosis.

Frith (1991), cited Hans Asperger: With concentrated energy and obvious confidence and, yes, with blinkers toward life's many possibilities, they follow their own path toward that which their talents have directed them since childhood – thus once again realizing the saying: “Man's good and bad qualities are only two sides of the same coin. It is simply impossible to separate them, to choose the good and reject the bad.”

Other researchers have asked autists and aspergians about their own perceptions of their situation. Respondents indicated an unusual perception and information processing, plus difficulty in regulating emotions – descriptions not included in the official DSM criteria. There are obviously different views on how aspergians and autists are to be understood.

The psychological dysfunction underlying the triad of impairments (imaginative thinking, socialization, and communication) could be described as the inability to put oneself in the position of another and to appreciate their thoughts, feelings, and wishes. This triad describes both Asperger's and autism. Obviously there is more to be said about imaginative thinking as, e.g., Einstein is considered to have had Asperger's.

There is no common understanding of the prevalence of Asperger's within the general population. Findings between studies differ but are usually below 1 %. 50 years after Hans Asperger's publication, it entered the *DSM-IV* in 1994. The removal of Asperger's from the *DSM-5* is likely to be controversial as the Asperger diagnosis is used by health insurers, researchers, state agencies, and schools – just to say nothing of people with the diagnosis, many of whom proudly call themselves aspies.

Several of this world's great thinkers are said to have been “suffering” from Asperger's like Socrates, Archimedes, Pythagoras, Julius Caesar, Napoleon Bonaparte, Charles Darwin, Albert

Einstein, Ludwig Wittgenstein, Bertrand Russell, Charles Babbage, Isaac Newton, Nikola Tesla, Kurt Gödel, Charles Lindbergh, John Watson, Alfred Kinsey, George Orwell, H. G. Wells, Ludwig van Beethoven, Wolfgang Amadeus Mozart, Georg Friedrich Händel, Pyotr Illyich Tchaikovsky, H. C. Andersen, Jane Austen, Immanuel Kant, and Alfred Nobel.

What would the world today look like if they had been prescribed selective serotonin reuptake inhibitors (SSRIs), fluoxetine, fluvoxamine, and sertraline for treating their restricted and repetitive interests and behavior?

Alexithymia

Alexithymia was introduced in 1973 by Peter Sifneos and was initially used for patients with psychosomatic disorders including a reduction in emotions, imagination, and finding words to describe their own feelings. Alexithymia is a cluster of cognitive and affective characteristics including difficulty identifying and communicating feelings, trouble distinguishing between feelings and somatic sensations of emotional arousal, impoverished and restrictive imaginative life, and a concrete and reality-oriented style (Taylor et al. 1997). Sometimes alexithymia has come to be associated with traditional masculinity in terms of negative characteristics as homophobia, violence, neglect of health, detached fathering and partnering, substance abuse, etc. (Levant and Richmond 2007).

If there is a common link between alexithymia and psychopathy, it may lie in the characteristic of *impulsivity*, so much in alexithymics that it seems to overcome their natural reluctance to break social conventions. Wastell and Booth (2003) have actually suggested that psychopathy should be viewed as alexithymia. Beginning with Niccolò Machiavelli's text (1513, 1987, *The Prince*), they argued that a psychopath is not a person who knowingly and intentionally manipulates his victims but instead is a victim of his own emotional limitations. A typical person with alexithymia is therefore anxious, overcontrolled, boring (Taylor et al. 1982), and submissive and

has a strict ethical approach. This puts alexithymia far away from psychopathy.

This last thing needs to be clarified; there is a significant positive correlation between secondary (anti-social) psychopathy and alexithymia, but not between primary psychopathy (often seen more stressing on genetic causes) and alexithymia. Alexithymia also seems to parallel Asperger's. Psychopaths on the other hand show low anxiety; are impulsive, dominant, charming, and deceitful; and do not necessarily try to fit in.

One case clearly showing how alexithymia and psychopathy can be mixed up is given by Ellis (2008). He described a man who killed his father without being aware that he was angry. He fully realized intellectually that the evidence (in the researchers' opinion) showed he *must have been* angry with his father, but he could not feel the anger. Perhaps he was not angry but just alexithymic? In short, there are similarities between alexithymia and psychopathy, but on ethical and moral issues, as in being able to lie and charm, psychopathy differs as much from alexithymia as it differs from Asperger's and autism.

There may be reasons for not considering the mild form of autism, Asperger's syndrome, as negatively and pathologizing as is being done today. Besides negative traits that are normally highlighted in the diagnosis, i.e., various aspects of so-called low social competence, one should take into account their high personal moral and personal care that often characterize individuals within the Asperger's syndrome (Dubin 2007).

There is a similarity between autism and narcissism in the young child and sometimes even in adults in their misapprehension of the reality or empathic understanding of other people's existence. "While a normal child is unaware of itself and adequately interacting with others as an integral member of the collective, the autistic child is constantly busy with observing itself. It is itself the object of interest and directs its attention towards its body movements" (Frith 1991). During the 1950s, Carl Rogers shed light on or specified empathy, saying that empathy is the ability to step into another person's shoes – and out again – thereby differentiating empathy from pity, compassion, and sympathy.

Empathy has two components (cognitive empathy and emotional empathy). The extreme emotional component can be illustrated by "rushing to the rescue" and the extreme cognitive aspect carefully planned rescue operation.

Autism, Alexithymia, and Humor

The presence of humor is a strong and useful instrument in psychodiagnostics with its requirements of verbal skills, social skills, and emotional and intellectual self-mirroring. But, as Hooker (1934) said, it can also be a matter of taste and as such difficult or impossible to define. According to psychoanalytic theory, a personality structure more developed than psychosis and borderline is required when dealing with jokes (you don't kid around with a borderline). People with early disorders (psychosis and borderline, i.e., borderline according to the old psychiatric nomenclature, before the DSM) perceive life in a black or white mode, good or bad, us and them (see Youtube.com or Google Videos: Pink Floyd, "Us and them"), and right or wrong – they see nothing in between.

Much of what most people see as humor is a verbal game with nuances and multiple meanings of words or expressions. Only neurotics and psychologically healthy people can make or understand jokes like that. Frith (1991), citing Hans Asperger, wrote that autistic children and those with Asperger's disorder have no sense of humor, especially if the joke is directed toward them. He held that it was partly due to that they are rarely relaxed and unconcerned. So individuals with Asperger's disorder are impaired in humor appreciation although anecdotal and parental reports provide evidence to the contrary.

The language of alexithymic persons has generally been described as flat and humorless, and subjects are characterized by cognitive, operative thinking. An often cited expression is the French "*pensée opératoire*" (operational thinking). It has been used in texts covering psychopathy, Asperger's, autism, and alexithymia but is most often used for the last group. "People who are managed by alexithymics sense their dullness and boredom quickly, and they become frustrated when attempts at interaction fail. Not even humor works" (Kets de Vries 2009).

Borderline individuals with their black or white, matter-of-fact behavior instead of empathic participation rather laugh *at* other people while neurotics and “non-disturbed” individuals laugh *with* other people.

Nature or Nurture?

Over the past 60 years, the pendulum of public and scientific opinion on the etiology of autism has swung between two extreme positions: (1) that autism is caused by some specific genetic abnormality, spawning a search for the “autism gene,” and (2) that autism is the result of some specific environmental factor or condition. There are three related potential explanations: the sociological, the physiological, and the developmental. When trying to explain autism, two positions have been suggested: a genetic abnormality and an environmental factor, including a “lack of maternal warmth.” Although the etiology of autism has remained elusive, the evidence to date has strongly refuted both of these extreme positions (Strathern 2009; Bumiller 2009). In parallel with the sociological explanation of psychopathy, maybe a sociological explanation for autism and Asperger’s can be seen in their wish for social acceptance rather than cures (Lawson 2008).

Some studies have found that parents of children with autism were more likely to have been hospitalized for a mental disorder. Those studies do however not agree on if the parents had their diagnoses *before or after* the birth of their child. Did parental disorder *cause* the child’s disorder or was it the other way around? To that comes the problem of differing between social heritage and genetic heritage. Usually, parents can detect interference patterns long before a formal diagnosis usually is given. Some parents to autistic children seem to feel lack of contact with their children before the children reach 18 months of age. That may explain some parents developing mental disorders *after* their child being born.

Different Levels of Achievement

Some of this world’s great thinkers are said to have been “suffering” from Asperger’s (above).

What a given individual eventually will achieve depends on a combination of genetic heritage and environment. To that come pre-, peri-, and post-natal damages; social heritage, cultural, and social norms and rules – or short: (genetic) heritage sets the roof, environment how close to the roof one will get – and how that roof is defined.

The Very Intelligent Individual’s Problem

More or less, all studies comment on the inability of others to understand the highly gifted. One way of handling that, e.g., was formulated by Leta Stetter Hollingworth (1866–1939): “Of all the special problems of general conduct which the most intelligent children face, I will mention five, which beset them in early years and may lead to habits subversive of fine leadership:

1. To find enough hard and interesting work at school
2. To suffer fools gladly
3. To keep from becoming negativistic toward authority
4. To keep from becoming hermits
5. To avoid the formation of habits of extreme chicanery”

The second point of Hollingsworth above could be of use for the highly gifted, as one way of handling that others don’t understand – although themselves not actually understanding that others don’t understand! A lesson many gifted persons never learn as long as they live is that human beings in general are inherently very different from themselves in thought, in action, in general intention, and in interests. Many a reformer has died at the hands of a mob which he was trying to improve. Leta Stetter Hollingworth also stressed the importance of providing a matching environment for highly gifted children.

Treatment, Education, or Just Acceptance

There is no unanimous definition of a successful treatment. Some say a successful treatment means the patient reports works or study and is happy living in a good relationship, others that the

patient just succeeded in going back to work, while still other definitions could be compared to the surgical concept of arthrodesis, and finally some say the patient just didn't kill himself.

Perhaps we should stop seeing autism as a medical disorder and instead see it as one of life's expressions. One can of course object that this is going a bit too far as there are different kinds of autistic disorders and that perhaps a third of them express some form of incomplete development, i.e., a health problem and not a matter of statistical discrepancy. With a diagnosis, parents may receive medical and psychological/educational support, while the child may be – and sometimes inaccurately – labeled and marked for life. Some autism researchers have expressed: The autism rights movement seeks acceptance, not cures. A parallel might be found in a highly intelligent and stimulus-seeking child misdiagnosed as ADHD.

Asperger/Autist: Truth Teller, Fundamentalist, Terrorist?

What will happen to the Asperger, the truth seeker, the truth teller? Adolescents and adults with Asperger's may engage in activities leading to fundamentalist religions (Attwood 2003). Maybe the Norwegian terrorist Anders Behring Breivik started as a rigid, principle-ridden Asperger's with an ideological goal (Norway only for Norwegians)? Different groups of experts in Norway have suggested diagnoses as narcissism, psychopathy, schizophrenia, or "no psychiatric diagnose at all." Niklas Långström said (2012) that Behring Breivik was neither psychotic nor suffering from substance use disorders, so there must be other explanations. Anders Behring Breivik may perhaps be seen as a truth-seeking aspergian, i.e., totally disregarding other people's views and feelings. With that, a new road for terrorist research on the consequences of fundamentalist personalities is indicated. When, and if, an aspergian develops toward "the pure thinking" of fundamentalism, the "medicine" is a dialogue, however, not an easy one. The method in question has a parallel in international styles of mediation (compared with the work of Jan Eliasson, Deputy Secretary-

General of the United Nations) between conflicting powers where presumably often "pure thinking" is involved among the parties or combatants.

In the Judiciary

Several researchers have pointed to major differences in moral and conventional behavior between most children and children with autism or ADHD and, on the other hand, child psychopaths and adult psychopaths scoring lower. In most countries, psychopaths are seen as fully responsible for their actions. Among those with Asperger's syndrome, there are at most 10 % with savant skills, and they are a problematic group for the judiciary. There is a need for a better understanding of Asperger's for those working within the criminal justice sector that lack the requisite training to respond effectively to those with Asperger's. As stated above, autists should be seen normal beings who want acceptance instead of cures. If that is so, they should be seen as normal responsible people even in a court. Asperger individuals and autists can, so to speak, meet with the judiciary in three instances: police, court, and in the question of sanctions. First, police needs more training. Second, in court psychopaths, Asperger people and autists should be treated as everybody else. Third, when it comes to issues of legal consequence or sanction, i.e., suspended sentence, probation, claim for damages, fines, restraining order, treatment (psychiatric), or prison, more research is needed.

See Also

► [Autism Spectrum Disorders](#)

References

- Attwood, T. (2003). Understanding and managing circumscribed interests. In M. Prior (Ed.), *Learning and behavior problems in Asperger Syndrome*. New York: Guilford Press.
- Bailey, A., Phillips, W., & Rutter, M. (1996). Autism: towards an integration of clinical, genetic, neuropsychological, and neurobiological perspectives. *Journal of Child Psychology and Psychiatry*, 37, 89–126.

- Bumiller, K. (2009). The geneticization of autism: From new reproductive technologies to the conception of genetic normalcy. *Signs*, 34(4), 875–899.
- Dubin, N. (2007). *Asperger syndrome and bullying: Strategies and solutions*. Jessica: Kingsley Publishers.
- Frith, U. (1991). *Autism and Asperger syndrome*. Cambridge: Cambridge University Press.
- Hippler, K., & Klicpera, C. (2005). Hans Asperger und 'seine Kinder' – eine retrospektives Untersuchung des spectrum der "Psychopathia autistischen" anhand von Wiener Krankenakten. *Zeitschrift für Kinder- und Jugendpsychiatrie und Psychotherapie*, 33(1), 35–47.
- Kets de Vries, M. F. R. (2009). *Reflections on character and leaderships*. New York: Wiley.
- Långström, N. (2009). Schizofrenins samband med våldsbrott ifrågasatt (Schizophrenia associated with violent crime challenged). *Läkartidningen (Swedish Medical Journal)*, prepubl. 14 July.
- Långström, N., et al. (2009). Risk factors for violent offending in autism spectrum disorder. A national study of hospitalized individuals. *Journal of Interpersonal Violence*, 24(8), 1358–1370.
- Lawson, W. (2008). *Concepts of normality – The autistic and typical spectrum*. Jessica: Kingsley Publishers.
- Levant, R. F., & Richmond, K. (2007). A review of research on masculinity ideologies using the Male Role Norms Inventory. *Journal of Men's Studies*, 15, 130–146.
- Rutter, M. (1978). Diagnosis and definition of childhood autism. *Journal of Autism and Developmental Disorders*, 8(2), 139–161.
- Rutter, M. (2011). Progress in understanding autism: 2007–2010. *Journal of Autism and Developmental Disorders*, 41, 395–404.
- Rutter, M., Bailey, A., Bolton, P., & Couteur, A. L. (1994). Autism and known medical conditions: Myth and substance. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, 35(2), 311–322.
- Sharma, S., Woolfson, L. M., & Hunter, S. C. (2011). Confusion and inconsistency in diagnosis of Asperger syndrome: A review of studies from 1981 to 2010. *Autism*, 2, 1–22.
- Strathern, L. (2009). The elusive etiology of autism: Nature and nurture? *Frontiers in Behavioral Neuroscience*, 3(11), 1–3.
- Tantam, D. (1988). Lifelong eccentricity and social isolation. I. Psychiatric, social, and forensic aspects. *British Journal of Psychiatry*, 153, 777–782.
- Taylor, G. J., Bagby, R. M., & Parker, G. (1997). *Disorders of affect regulation: Alexithymia in medical and psychiatric illness*. Cambridge: Cambridge University Press.
- Treffert, D. A. (2006). Dr Down and 'developmental disorders'. *Journal of Autism and Developmental Disorders*, 36, 965–966. doi:10.1007/s10803-006-0183-1.
- Wastell, C., & Booth, A. (2003). Machiavellianism: An alexithymic perspective. *Journal of Social and Clinical Psychology*, 22(6), 730–744.
- Whitehouse, A. J. O., Hickey, M., & Ronald, A. (2011). Are autistic traits in the general population stable across development? *PLoS One*, 6(8), e23029. doi:10.1371/journal.pone.0023029.

Allele Similarity

► Zygosity

Allen, Doris

Isabelle Rapin

Neurology and Pediatrics (Neurology), Albert Einstein College of Medicine, Bronx, NY, USA

Name and Degrees

Allen, Doris A., 1932–2002

BA – English/Speech Pathology/Audiology (1954)

MA – Applied Linguistics, Teachers College, Columbia University, New York City (1964)

MA – Psychology, Teachers College, Columbia University, New York City (1971)

EdD – Psycholinguistics, Teachers College, Columbia University, New York City (1973)

Major Appointments (Institution, Location, Dates)

Post doctoral Multidisciplinary Fellowship in Neuroscience, Albert Einstein College of Medicine, Bronx, NY (1974–1976)

Assistant Professor to Professor of Clinical Child Psychiatry and Clinical Pediatrics, Albert Einstein College of Medicine, 1977–2002.

Principal Investigator for the Autism Subproject – Diagnosis and classification of autistic children – of the NIH program project grant: Nosology: Higher Cerebral Function Disorders in Children (NS 20489) (1985–1993).

Major Honors and Awards

President's Scholarship, Teachers College, Columbia University (1964–1966).

Principal Investigator, NIH grant: The development of communicative competence in prematurely born children (1977).

Assistant Professor to Professor of Clinical Child Psychiatry and Clinical Pediatrics, Albert Einstein College of Medicine, 1977–2002.

Principal Investigator for the Autism Subproject – Diagnosis and classification of autistic children – of the NIH program project grant: Nosology: Higher Cerebral Function Disorders in Children (NS 20489) (1985–1993).

Landmark Clinical, Scientific, and Professional Contributions

(All at Albert Einstein College of Medicine, Bronx NY, USA)

- Director of the Therapeutic Nursery in the Division of Child Psychiatry of the Albert Einstein College of Medicine, 1978–1995. Director after its move to Tenafly NJ: 1995–2002.
- Developed a parent-child intervention model in the Nursery for educating preschool children with autism spectrum disorders without mental retardation.
- Trained generations of residents/fellows in child psychiatry, child neurology, and pediatrics to recognize milder autism spectrum disorders and how they can be managed effectively.
- Trained many graduate students and postdoctoral neuropsychology and speech/language pathology fellows in the diagnosis, education, and management of children with autism.
- Led the Einstein research group on language disorders in preschoolers.
- Was principal investigator of the autism subproject and investigator of the Autism Subproject of the multidisciplinary multiinstitutional Nosology project.
- With I. Rapin developed a neurologically and linguistically based clinical classification of developmental language disorders in preschoolers with/without autism for clinicians' use in their offices.

Short Biography

Born and brought up in Indiana, Dr. Doris A. Allen started her professional life as an English

teacher and mother of three sons. She subsequently relocated to New York, was remarried to Dr. Robert L. Allen, Professor of Linguistics at Columbia University where she obtained master's degrees in both psychology and applied developmental psycholinguistics and a doctorate in linguistics. After a 2-year postdoctoral fellowship in neuroscience at Albert Einstein College of Medicine, she was appointed to the faculty and as Director of the Therapeutic Nursery in the Division of Child Psychiatry. She turned it around from Freudian therapy of mothers to education of high functioning preschoolers with autism spectrum disorders (ASD), with a curriculum focused on social skills, communicative language, and self-management (Allen and Mendelson 2000). Dr. Allen recognized much earlier than most investigators that, besides severely impaired and intellectually deficient children with classic autistic disorder, there are many intelligent children on the autism spectrum for whom early, intensive, specialized intervention may enable them to grow up to become independent or nearly independent adults.

Dr. Allen developed the novel and highly effective parent-child model for the Nursery in which a caretaker attends school daily with the preschooler and is trained "in the trenches" to manage severe behavioral outbursts ("meltdowns") and to communicate more effectively with their child. Other family members receive some counseling as well, with tremendous improvement in the quality of life for everyone.

While at Einstein – and even now – the Nursery served as laboratory for research. Equally important, it provided the opportunity for physician trainees in child psychiatry, child neurology, and pediatrics to learn to spot mildly affected children likely to respond to appropriate educational intervention. Dr. Allen trained child psychiatrists, as well as graduate students and postdoctoral fellows, in psychology and speech/language pathology in the diagnosis, education, and treatment of children with autism. She was invited to lecture by many parent groups and at professional meetings in the USA and abroad.

Among her distinguished trainees are the child neuropsychologists Dr. Michelle A. Dunn, an

Einstein Professor, and Dr. Hilary Gomes, a Professor at City University of New York Graduate Center, who use electrophysiology to study language in autism (Dunn et al. 1999; Dunn et al. 1996). Dr. Dunn has developed an innovative visually based curriculum for children with ASD of all ages mainstreamed to regular classes (Dunn 2005; Fein and Dunn 2007). Another trainee, Dr. Mary Jure, has replicated with success the Einstein nursery in Cordoba, Argentina. Still another, Dr. Sylvie Goldman, studies narrative in children with autism (Goldman 2008), its male preponderance (Pfaff et al. 2011), and repetitive movements viewed as movement disorder rather than self-stimulation (Goldman et al. 2009).

Dr. Allen was the leader of the Einstein research group on language deficits in preschoolers (Allen 1988; Rapin and Allen 1987) and Co-principal Investigator for autism in the Nosology project (Fein et al. 1996). She stressed that effective remediation required subtyping of language deficits in order to address each child's needs individually (Allen et al. 1989; Allen 1994). She teamed with Dr. Isabelle Rapin, a child neurologist, to develop a neurologically and linguistically based clinical classification of developmental language disorders for nonspecialists applicable to any young child, whether on the autism spectrum or not. They found that there are several subtypes of language disorders in autism, including some affecting phonology and grammar (Allen and Rapin 1992; Rapin et al. 2009). Major distinctions between autism and developmental language disorders are different subtype prevalences, together with defective comprehension and universal and persistently impaired pragmatics (communication skills) in ASD. Dr. Allen coined the term semantic-pragmatic language disorder, now widely used, to describe chatty children whose expressive language is superior to their comprehension of discourse, whether or not they fulfill criteria for an ASD (Rapin and Allen 1998).

In short, Dr. Allen's interest in preschoolers with inadequate language and behavior and their treatment led to many publications, lectures, and the training of many professionals in the USA and

abroad. Perhaps her most enduring contribution is the innovative and effective model for educating preschoolers with ASD, as indicated by the majority of the graduates of her therapeutic nursery able to be educated in regular classrooms with or without the need for an aide and many among the older ones graduating from college or other higher education who are now independently employed.

References and Reading

- Allen, D. A. (1988). Autistic spectrum disorders: Clinical presentation in preschool children. *Journal of Child Neurology*, 3, s48–s56.
- Allen, D. A. (1994). Tratamiento educativo para niños autistas preescolares. In N. Fejerman, H. A. Arroyo, M. E. Massaro, & V. L. Riggieri (Eds.), *Autismo Infantil Y Otros Trastornos del Desarrollo* (pp. 109–121). Buenos Aires: Paidós.
- Allen, D. A., & Mendelson, L. (2000). Parent, child, and professional: meeting the needs of young autistic children and their families in a multidisciplinary therapeutic nursery model. In S. Epstein (Ed.), *Autistic spectrum disorders and psychoanalytic ideas: Reassessing the fit* (pp. 704–731). Hillsdale: The Analytic Press.
- Allen, D. A., & Rapin, I. (1992). Autistic children are also dysphasic. In H. Naruse & E. Ornitz (Eds.), *Neurobiology of infantile autism* (pp. 73–80). Amsterdam: Excerpta Medica.
- Allen, D. A., Mendelson, L., & Rapin, I. (1989). Syndrome specific remediation in preschool developmental dysphasia. In J. H. French, S. Harel, P. Casaer, M. I. Gottlieb, I. Rapin, & D. C. De Vivo (Eds.), *Child neurology and developmental disabilities* (pp. 233–243). Baltimore: Paul Brookes.
- Dunn, M. (2005). *S.O.S.: Social skills in our schools program (A Social Skills program for children with Pervasive Developmental Disorders and their typical peers)*. Shawnee Mission, KS: Autism and Asperger.
- Dunn, M., Gomes, H., & Sebastian, M. (1996). Prototypicality of responses in autistic language disordered and normal children in a verbal fluency task. *Child Neuropsychology*, 2, 99–108.
- Dunn, M., Vaughan, H. G., Jr., Kreutzer, J., & Kurtzberg, D. (1999). Electrophysiologic correlates of semantic classification in autistic and normal children. *Developmental Neuropsychology*, 16, 75–99.
- Fein, D., & Dunn, M. A. (2007). *Autism in your classroom: A general educator's guide to students with autism spectrum disorders* (1st ed.). Bethesda: Woodbine House.
- Fein, D., Dunn, M., Allen, D. A., Aram, D. M., Hall, N., Morris, R., et al. (1996). Language and neuropsychological findings. In I. Rapin (Ed.), *Preschool children*

- with inadequate communication: *Developmental language disorder, autism, low IQ* (pp. 123–154). London: Mac Keith Press.
- Goldman, S. (2008). Narrative abilities of children with autism and developmental language disorders: Scripts versus stories. *Journal of Autism and Developmental Disorders*, 38, 1982–1988.
- Goldman, S., Wang, C., Salgado, M. W., Greene, P. E., Kim, M., & Rapin, I. (2009). Motor stereotypies in children with autism and other developmental disorders. *Developmental Medicine & Child Neurology*, 51, 30–38.
- Pfaff, D. W., Rapin, I., & Goldman, S. (2011). Male preponderance in autism: Neuroendocrine influences on arousal and social anxiety. *Autism Research*, 4, 1–14.
- Rapin, I., & Allen, D. A. (1987). Developmental dysphasia and autism in preschool children: characteristics and subtypes. In J. Martin, P. Fletcher, P. Grunwell, & D. Hall (Eds.), *Proceedings of the first international symposium on specific speech and language disorders in children* (pp. 20–35). London: AFASIC.
- Rapin, I., & Allen, D. A. (1998). The semantic-pragmatic deficit disorder: Classification issues. *International Journal of Language & Communication Disorders*, 33, 82–87.
- Rapin, I., Dunn, M., Allen, D. A., Stevens, M., & Fein, D. (2009). Subtypes of language disorders in schoolage children with autism. *Developmental Neuropsychology*, 34, 1–9.

Allergies

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Synonyms

Hay fever

Definition

An allergy is the body's exaggerated response to a foreign antigen (substance) or allergen that results

in an immune response leading to a reaction such as allergic conjunctivitis (itchy eyes), allergic rhinitis (runny nose), anaphylaxis (allergic shock), asthma, atopic dermatitis, eczema, hives, serum sickness, or contact dermatitis (skin rash). The body makes antibodies (immunoglobins) that attach to foreign particles like allergens and viruses to allow the immune system to dispose of them. People who are allergic to a compound will make the immunoglobulin type IgE in response to exposure to that compound. Common allergens include dust mites, animal dander, pollen, and foods. Allergic contact dermatitis is not mediated through IgE. While there is genetic predisposition to allergies, it requires a period of exposure (sensitization) for a person to make antibodies and develop symptoms. The production of antibodies in response to an allergen leads to allergic symptoms through release of chemicals such as histamine from the body's own cells which leads to inflammation. Allergies may start at any age. Some allergic manifestations such as asthma may be more problematic in childhood. Food allergies may present as tingling or swelling of the throat and tongue, nausea, diarrhea, skin reactions, or even anaphylaxis. The most common food allergens are milk, fish, shellfish, peanuts, tree nuts, eggs, wheat, and soy. Allergy workup may be initiated after a history of symptoms after exposure to an allergen. Blood tests such as the enzyme linked immunosorbent assay (ELISA) or radioallergen sorbent testing (RAST) may detect specific IgE antibodies associated with allergic response. Blood testing is not as accurate as skin testing. Skin prick, intradermal, or patch testing characterizes an individual's response to allergens administered using standard procedures and measurement of response.

The best treatment for allergies is to avoid the allergen responsible for symptoms. Symptomatic relief may be possible with antihistamines, eye drops, and topical or oral steroid preparations depending on the type of symptom. Treatment of asthma may require both management of the allergy and medication to address lung function. People who respond to allergens with anaphylaxis must carry epinephrine for injection since

anaphylaxis may be fatal. Allergy shots or immunoprophylaxis is a type of treatment that is usually supervised by a medical doctor specializing in allergy and immunology where small amounts of the target allergen are injected into a patient to help build up antibody response.

See Also

► [Food Intolerance](#)

References and Reading

<http://familydoctor.org/online/famdocen/home/common/allergies/basics/083.printerview.html>.

<http://www.jacionline.org/article/S0091-6749%2810%2901566-6/fulltext>.

<http://www.medicinenet.com/allergy/article.htm>.

<http://www.webmd.com/a-to-z-guides/allergy-tests>.

NAIAD Sponsored Expert Panel. (2010). Guideline for diagnosis and management of food allergy in the US: Report of the NAIAD sponsored expert panel. *Journal of Allergy and Immunology*, 126(6), S1–S58.

AllerMax® [OTC]

► [Diphenhydramine](#)

Aloof Group

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Definition

Lorna Wing and Judith Gould (1979) in their epidemiological study identified individual children who did not fit neatly into definitive categories but whose pattern of skills and behavior could be described as part of a spectrum of autistic conditions.

There were three aspects, social interaction, communication (verbal and nonverbal), and imagination. Children with difficulties in these areas also showed repetitive patterns of behavior. The manifestations of different problems in social interaction could be grouped into three types, Aloof, Passive, and Active but Odd.

The aloof group closely resembled the then popular image of autism which had been described by Kanner (1943) and Kanner and Eisenberg (1956). These individuals are the most cut off from social contact. If they do make contact, it is essentially needs led. They may respond to (and may initiate) physical contact only, including rough and tumble games, chasing, cuddling but are otherwise indifferent. This pattern of social interaction is linked with problems in understanding and use of verbal and nonverbal communication. Many persons in this group lack communication skills all their lives. If they do develop speech there are often unusual aspects of communication, e.g., echolalia, reversal of pronouns, repetitiveness, idiosyncratic use of words or phrases, and abbreviations of phrases. If they reach this level of communication, they will use the minimum number of words to convey basic needs. Most importantly, these children do not use speech as a means of social interaction. Speech is simply a way of getting what they want.

As children, most of the aloof group have no symbolic pretend play. They may manipulate objects but show no signs of pretending that toys represent real things. They do not build up an inner world of imagination for themselves. Instead, they fill their time with repetitive, stereotyped activities. Such children may engage for hours on one pursuit which totally absorbs them such as lining up toys or twiddling an object close to their eyes.

The more able aloof individuals may have complex elaborate repetitive routines such as collecting objects, organizing objects into patterns, bedtime rituals, and taking the same route to places. Aloofness and indifference to others are most likely to persist throughout childhood into adult life in individuals who are severely

intellectually disabled. The more intellectually able aloof group may demonstrate special skills usually in visuospatial skills and rote memory.

See Also

- [Kanner, Leo](#)
- [Wing, Lorna](#)

References and Reading

- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250. “Reprint”. *Acta Paedopsychiatr*, 35(4), 100–136. 1968.
- Kanner, L., & Eisenberg, L. (1956). Early infantile Autism 1943–1955. *The American Journal of Orthopsychiatry*, 26, 55–65.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11–29.

Alpha (α) Error

- [False Positive](#)

Alpha-4 Beta-2 Nicotinic Agonists

- [Nicotine and Autism](#)

Alpha-7 Nicotinic Agonists

- [Nicotine and Autism](#)

Alpha-Amino Acid

- [Amino Acids](#)

Alprazolam

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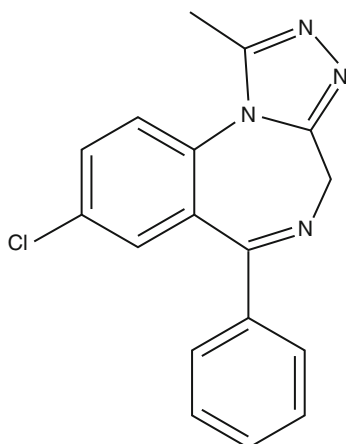
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Synonyms

[8-Chloro-1-methyl-6-phenyl-4H-s-triazolo \[4,3-α\] \[1,4\] benzodiazepine](#); [Niravam](#); [Xanax](#); [Xanax XR](#)

Definition

A prescription drug in the group of triazolobenzodiazepines in the family of benzodiazepines initially FDA-approved for medical use in the year 1981 with the chemical formula C₁₇H₁₃ClN₄. This compound has low water solubility and high lipid solubility. This drug acts as a central nervous system depressant and is mostly metabolized by cytochrome P450 (CYP450) enzyme 3A4. This high-potency benzodiazepine with a half-life of 10–15 h is FDA-approved for the treatment of panic disorder and anxiety disorders and can also be used to treat seizures, premenstrual dysphoric disorder, tricyclic antidepressant-related jitteriness syndrome, and valproate-induced tremors. Observed side effects include drowsiness, light-headedness, dizziness, depression, tiredness, nausea, insomnia, and diarrhea.



See Also

- Benzodiazepines
- Xanax (Alprazolam)

References and Reading

- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001). *Principles and practice of psychopharmacotherapy* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
- Oswald, D. P., & Sonenklar, N. A. (2007). Medication use among children with autism-spectrum disorders. *Journal of Child and Adolescent Psychopharmacology*, 17, 348–355.
- Raj, A., & Sheehan, D. (2006). Benzodiazepines. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 181–197). Washington, DC: American Psychiatric Publishing.
- Stahl, S. M. (2000). Benzodiazepines. Drug treatments for obsessive-compulsive, panic, and phobic disorders. In S. M. Stahl (Ed.), *Essential psychopharmacology: Neuroscientific basis and clinical applications* (pp. 354–355). Cambridge: Cambridge University Press.
- U.S. Food and Drug Administration. (2011). *Drugs@FDA*. Retrieved from <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm>

Altaryl [OTC]

- Diphenhydramine

Alterations in Chromosome Structure or Number

- Chromosomal Abnormalities

Alternative Communication

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Definition

Alternative communication (also called augmentative and alternative communication or AAC) is an area of clinical practice within the field of speech-language pathology. A definition of AAC is provided by the American Speech-Language-Hearing Association (ASHA). According to ASHA, AAC “includes all forms of communication (other than oral speech) that are used to express thoughts, needs, wants, and ideas” (ASHA 1997). The types of AAC include aided and unaided communication systems. Aided systems are those that require something other than the individuals’ body to communicate. That “something” could be picture symbols, written words, or a high-tech, speech generating device. Conversely, unaided systems are those that do not require anything separate from one’s own body to communicate. Essentially, gestures, body language, and sign language are examples of unaided systems.

Historical Background

The field of AAC began with the development of the International Society for Augmentative and Alternative Communication (ISAAC) in 1983. Before this, AAC was not a unified field and existed as a combination of interventions (communication boards and sign language) and products designed for individuals with speech and motor impairments. In fact, the use of augmentative or alternative communication for individuals with speech impairments may have first been introduced in the 1920s in the form of communication boards for individuals with cerebral palsy. The 1960s and 1970s saw an increase in the use of technology for AAC purposes. Typewriters that used innovative input methods such as a sip-and-

puff switch were invented. Speech-output devices were invented in the 1970s, and portable speech-output devices were available shortly thereafter. For a more detailed look at the early history of AAC, see Vanderheiden (2002).

The field of AAC has seen many changes in terms of application and philosophies. Early assessment models focused on AAC candidacy. Often much time and thought was spent examining a client's qualifications for AAC interventions. This resulted in the thought that many individuals were too "something" for AAC and therefore not deemed appropriate for AAC services. Individuals may have been seen as having too little linguistic functioning, too much linguistic functioning, too cognitively impaired, too high functioning, too limited motor abilities, etc. This resulted in many individuals who could have benefited from AAC technologies not receiving proper services.

Another factor which resulted in missed opportunities to provide AAC solutions was the erroneous idea that the use of augmentative communication would act as a crutch for individuals with speech impairments. It was feared by many in the field that those who used AAC would not learn to communicate vocally despite research to the contrary.

Rationale or Underlying Theory

The rationale for augmentative and alternative communication can be found in the term itself. AAC is first augmentative. The purpose for this type of intervention is to augment or supplement the speech an individual naturally possesses. For some individuals, however, this intervention is an alternative form of communication. These individuals have no means of verbal speech and so need to implement an alternative form. AAC is the means by which these individuals communicate.

Goals and Objectives

The goal of AAC is functional communication. Rate of message transfer is different for the different forms of AAC. Sign language, when produced

by a fluent signer, can be produced as quickly as spoken speech (Bellugi and Fischer 1972). However, communication through means of an alternative communication device occurs at an excruciatingly slow 15 words per minute (Foulds 1987) compared to 150–250 words per minute for speakers (Goldman-Eisler 1986). Therefore, rate of communication should not be expected to occur as fast as spoken communication for individuals who use aided systems.

Treatment Participants

Any individual who has impaired communication is a candidate for AAC. Therefore, because communication impairments are a hallmark of autism spectrum disorders (ASDs) (Mirenda 2009), most individuals with ASDs are candidates for a total communication approach.

The currently used assessment model is called the participation model (see Beukelman and Mirenda 2005 for a thorough description of the model). The model emphasizes those areas that an individual is not able to take part in due to their communication impairments. As such, this model is inclusive and appropriate for any individual who has communication needs in any area of their life.

Treatment Procedures

Using the participation model (Beukelman and Mirenda 2005) as a guide, AAC interventions focus on allowing an individual with speech impairment to participate in their environment to the same extent as that of their peers. Beukelman and Mirenda (2005) also give strategies and recommendations for implementing AAC for both nonsymbolic and symbolic beginning communicators. Nonsymbolic beginning communicators are those who use nonsymbolic communication such as gestures, facial expression, cries, or grunts. Symbolic beginning communicators use some form of symbolic communication such as words (spoken or written) or symbols with low- or high-tech communication devices. The authors state that "opportunity for communication is at

least as important to the success of a communication intervention as the availability of an appropriate system” (p. 272). Additionally, the authors provide techniques related to shaping intentional communication, using scripted routines, providing natural consequences, and using structured instructional techniques such as the adapted strategic instruction model (A-SIM), structured practice, and conversational coaching.

Efficacy Information

Efficacy research in the field of AAC is a relatively new addition to the literature. Bedrosian (1999) states that much early research in the field, as it should have been, was devoted to descriptive studies relating to describing the communication of AAC users. Since that publication, many more research studies have been conducted that are devoted to the efficacy of AAC for specific populations. Autism is one of those populations that has been widely studied. Overwhelmingly, the use of AAC has resulted in increased language skills in children with autism over treatment approaches that focus on speech alone. For most individuals with autism, accessing their relative strength in the visual domain has resulted in faster and more complex language growth in both signing and speaking. The use of manual signing in combination with speech training has been shown to increase language skill. The use of nonelectronic-aided systems such as picture use has also been shown to increase functional communication, and a wide range of individuals with autism have been able to make use of this type of communication. High-tech AAC use has been shown to increase language abilities and speech output in individuals with autism as well. See Goldstein (2002) and Mirenda (2002) for reviews.

A meta-analysis of available research related to AAC use was conducted by Millar et al. (2006). Although the meta-analysis was not focused only on individuals with autism, the major finding was that use of AAC does “not have a negative impact on speech production” (p. 257), and, in fact, speech production increased in individuals ages

2–60 years as a result of AAC interventions and across a range of different AAC interventions (aided and unaided).

Outcome Measurement

Because the goal of AAC use is functional communication, the outcome measurement should be the same. Functional communication of course will be defined differently based on the cognitive skills of the individual and the type of AAC system that is in place.

Qualifications of Treatment Providers

AAC interventions are most typically introduced by a speech-language pathologist. Unfortunately, many speech-language pathologists do not report having adequate training or education in the field of AAC (King 1998; Marvin et al. 2003; Simpson et al. 1999), and a survey of education programs for speech-language pathologists has uncovered a need for better education in this area (Ratcliff et al. 2008). Although this is the case, speech-language pathologists are the best equipped of all professionals who work with individuals with autism to provide intervention that includes AAC. A listing of speech-language pathologists who are certified by the American Speech-Language-Hearing Association can be found on their website. A few short questions posed to the speech-language pathologist can reveal whether they are comfortable with the area of AAC.

See Also

- ▶ [American Sign Language \(ASL\)](#)
- ▶ [Assistive Devices](#)
- ▶ [Communication Board](#)
- ▶ [Low-Technology Device](#)
- ▶ [Manual Sign](#)
- ▶ [Pictorial Cues/Visual Supports \(CR\)](#)
- ▶ [Sign Language](#)
- ▶ [Total Communication \(TC\) Approach](#)
- ▶ [Voice Output Communication Aids](#)

References and Reading

- ASHA. (1997). Augmentative and alternative communication (AAC). Retrieved 20 July, 2011 from <http://www.asha.org/public/speech/disorders/AAC.htm>.
- Bedrosian, J. L. (1999). AAC efficacy research: Challenges for the new century. *Augmentative and Alternative Communication*, 15, 2–3.
- Bellugi, U., & Fischer, S. (1972). A comparison of sign language and spoken language. *Cognition*, 1, 173–200.
- Beukelman, D. R., & Mirenda, P. (2005). *Augmentative and alternative communication: Supporting children and adults with complex communication needs*. Baltimore: Brooks Publishing.
- Foulds, R. (1987). Guest editorial. *Augmentative and Alternative Communication*, 3, 169.
- Goldman-Eisler, F. (1986). *Cycle linguistics: Experiments in spontaneous speech*. New York: Academic.
- Goldstein, H. (2002). Communication intervention for children with autism: A review of treatment efficacy. *Journal of Autism and Developmental Disorders*, 32, 373–396.
- King, J. (1998). Preliminary survey of speech-language pathologists providing AAC services in health care settings in Nebraska. *Augmentative and Alternative Communication*, 14, 222–227.
- Marvin, L. A., Montano, J. J., Fusco, L. M., & Gould, E. P. (2003). Speech-language pathologists' perception of their training and experience in using alternative and augmentative communication. *Contemporary Issues in Communication Sciences and Disorders*, 30, 76–83.
- Millar, D. C., Light, J. C., & Schlosser, R. W. (2006). The impact of augmentative and alternative communication intervention on the speech production of individuals with developmental disability: A research review. *Journal of Speech, Language, Hearing Research*, 49, 248–264.
- Mirenda, P. (2002). Toward functional augmentative and alternative communication for students with autism: Manual signs, graphic symbols, and voice output communication aids. *Language, Speech, and Hearing Services in Schools*, 34, 203–216.
- Mirenda, P. (2009). Introduction to AAC for individuals with autism spectrum disorders. In P. Mirenda & T. Iacono (Eds.), *Autism spectrum disorders and AAC*. Baltimore: Paul H. Brookes.
- Ratcliff, A., Koul, R., & Lloyd, L. L. (2008). Preparation in augmentative and alternative communication: An update for speech-language pathology training. *American Journal of Speech-Language Pathology*, 17, 48–59.
- Simpson, K., Beukelman, D., & Bird, A. (1999). Survey of school speech and language service provision to students with severe communication impairments in Nebraska. *Augmentative and Alternative Communication*, 14, 212–221.
- Vanderheiden, G. C. (2002). A journey through early augmentative communication and computer access. *Journal of Rehabilitation Research and Development*, 39, 39–54.

Alternative Diagnostic Concepts

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Definition

Clinical medical work without diagnosis is pointless. There can be no medical epidemiological study of psychiatric or developmental disorder without a consideration of diagnostic boundaries.

Diagnostic systems in psychiatric and developmental medicine are overarching models of symptoms, problems, functional restrictions, impairments, traits, signs, and psychological and biological test markers that constitute a particular disease, disorder, or group of disorders. Among these, the most influential are clinically based, generally agreed models such as the World Health Organization International Classification of Diseases and Disorders (ICD) (WHO 1993) and, for psychiatric disorders, the American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders (DSM) (APA 1994, 2011). However, there are also factor analytic models, signal detection models, continuous distribution models with statistically predetermined cutoff arbiters, and artificial network models, but these, in spite of being important for the development of new operationalized criteria for categorical diagnoses, have, so far, had relatively little impact in clinical practice. There are also taxonomies proposed by individual research groups who have developed alternative diagnostic systems that may – or may not – take into account the existence of the other clinically based models. This entry cannot avoid discussing modeling issues and the most commonly used clinically based systems, before going on to take a look at alternative diagnostic systems, including issues relating to multiple complex developmental disorder (MCDD), deficits in attention, motor control, and perception (DAMP), empathy disorders, nonverbal learning

disability, and early symptomatic syndromes eliciting neurodevelopmental clinical examinations (ESSENCE).

Historical Background

The ICD

The ICD is the international standard diagnostic classification for clinical practice and epidemiological and health management purposes. The current version (ICD-10) has a section for psychiatric disorder (including for autism or ► [“Pervasive Developmental Disorders”](#)) that is similar, but not identical, to that of the DSM-IV, which was published at about the same time as the ICD-10. Attempts were made during the development of the psychiatric section of the ICD-10 and the DSM-IV to streamline the two manuals. This was partly successful, but there are still considerable differences across the texts, criteria, and algorithms for diagnosing particular disorders, and some disorders appear only in one of the manuals.

Given that the DSM, compared to the ICD, has a much longer history when it comes to developing and analyzing operationalized criteria for psychiatric disorder, there will be a more detailed focus on the DSM than on the ICD. Much of what will be said about the DSM-IV (and the development of the DSM-5) applies in principle to the ICD-10 (and the development of the ICD-11, which is scheduled for publication in 2013).

The DSM

The Diagnostic and Statistical Manual of Mental Disorders (DSM-I) was published in 1952. The DSM-II, published in 1968, was 134-page long and listed 182 disorders. Both the DSM-I and the DSM-II reflected the predominantly psychodynamic psychiatry, although they also included biological perspectives and concepts from Kraepelin’s system of classification. Symptoms were not operationalized.

The criteria adopted for many of the mental disorders in the DSM-III (1980) were taken from the Research Diagnostic Criteria (RDC) and the Feighner Criteria, which had already been developed by a group of research-oriented psychiatrists.

Other criteria, and potential new categories of disorder, were established by consensus during meetings of the DSM committee. A key aim was to base categorization on descriptive language rather than assumptions of etiology. A new “multiaxial” system attempted to yield a “bigger picture.” When published, the DSM-III was almost 500-page long and listed 265 diagnostic categories. It rapidly came into widespread international use by multiple stakeholders and has been termed a revolution or transformation in psychiatry.

In 1987, the DSM-III-R was published as a revision of DSM-III. Six categories were deleted while others were added. The DSM-III-R contained 292 diagnoses and was 70 pages longer than the DSM-III.

In 1994, the DSM-IV was published, listing almost 300 disorders in just under 900 pages. The steering committee had created 13 work groups, who conducted a three-step process. First, each group conducted literature reviews of their diagnoses. Then they requested data from researchers, conducting analyses to determine which criteria required change, with instructions to be conservative. Finally, they conducted field trials relating diagnoses to clinical practice. A change from previous versions was the inclusion of a clinical significance criterion to about half of the categories. A “text revision” of the DSM-IV, known as the DSM-IV-TR, was published in 2000. The diagnostic categories and the vast majority of the specific criteria for diagnosis were unchanged (www.wikipedia.com).

Factor Analytic and Latent Class Models

Perhaps the most illustrative example of how factor analysis has been applied in clinical child and adolescent psychiatric/developmental diagnosis comes from the much-researched – and used – material developed by Thomas Achenbach (originally with colleague Edelbrock), often referred to as the “Child Behavior Checklist” (CBCL) or the ASEBA (Achenbach System of Empirically Based Assessment; Achenbach et al. 2008).

The CBCL/1.5–5 and the CBCL/6–18 includes 99/118 problem items that can be scored by parents of children aged 1–18 years. The items

refer to problem behaviors and emotions often encountered in children. A total problem score (comprising an internalizing and an externalizing score) is computed by adding scores for individual items. Subscores for aggressive behavior, anxious/depressed, attention problems, rule-breaking behavior, social problems, somatic complaints, thought problems, and withdrawn/depressed can also be calculated. The six DSM-oriented scales are affective problems, anxiety problems, somatic problems, attention deficit/hyperactivity problems, oppositional defiant problems, and conduct problems. The preschool 99-item version for 1.5–5-year-olds also has a DSM-oriented scale for autism/“pervasive developmental disorder.” Several studies have shown that combinations of subscales and individual items on the CBCL have good sensitivity and specificity for ASD in school-age children. In addition to the CBCL for parent rating, there is a related Teacher’s Report Form (TRF) and a Youth Self Report (YSR) for 11–18-year-olds.

Each item on the CBCL is given the same weight in the scoring system. The various subscales have been developed on the basis of factor and principal component analytic studies, and the DSM-oriented scales have been developed on the basis of a combination of statistical and clinical studies. One of the problems with the factor analytic approach relates to the fact that many of the individual items are completely unrelated and clearly do not have the same clinical weight. In fact, it can be argued that the individual items represent 118 different problems and that the subscales, to a considerable extent, represent artificial statistically derived constructs that do not necessarily correspond to recognizable clinical entities (in spite of having been assigned names that would suggest a clear correlation between the research and clinical concept). This problem is not unique to the development of the CBCL (and related material) but applies equally to a number of other much used scales, including those with subscales or full scales designed for screening and diagnosis of autism, for example, the Strengths and Difficulties Questionnaire (SDQ) (Goodman 1999) and the Autism Spectrum Screening Questionnaire (ASSQ) (Ehlers and Gillberg 1993).

Signal Detection Models and Receiver Operating Characteristic (ROC)

Many diagnostic systems are used to distinguish between two classes of events, essentially “signals” and “noise,” or “diagnosis” and “no diagnosis.” For such systems, analysis in terms of the “relative (or receiver) operating characteristic” (ROC) of signal detection theory provides a fairly precise and valid measure of diagnostic accuracy. It is uninfluenced by decision biases and prior probabilities, and it puts the performances of diverse systems on a common, easily interpreted scale.

The ROC model applied to a diagnostic screening instrument with a wide range of possible scores (such as the CBCL, the SDQ, or the ASSQ) is best presented in a graph detailing the true positive rate (TPR = sensitivity) on the y-axis and the false positive rate (FPR = 1 minus specificity) on the x-axis. The best trade-off for diagnostic purposes is usually seen at the point where the TPR is highest and the FPR lowest, that is, at the inflection point on the curve. The value of TPR times FPR at this point represents the area under the curve (AUC). When the AUC approaches 1.0, the diagnostic precision of the screening instrument is excellent, but when it approaches 0.5, the precision is extremely poor. The use of the AUC concept as a measure in the evaluation of new diagnostic screening tools has become something of a “gold standard” in recent years.

Continuous Distribution Models

Many human traits, functions, or markers of functional systems can be construed as existing on a normal distribution scale which will be relatively smooth when the range of possible scores is large. “Abnormality” is often defined as a specified distance from the mean or median score of such a scale (e.g., ± 2 standard deviations from the mean or under or over the second/98%). A disease or pathological state can be construed as existing when the value of a marker for a biological or psychological function is below a specified level (such as in pathological shortness/“dwarfism” or intellectual developmental disorder/mental retardation) or above a set limit (such as in hyperthyroidism).

Much can be said for diagnosing a number of psychiatric disorders along continuous distribution curves. Autism spectrum disorder (ASD), intellectual developmental disorder, and attention-deficit/hyperactivity disorder (ADHD) are but three examples of “disorders” that can, in many instances, be seen as extremes of “conditions” that exist along a normally/continuously distributed spectrum (Posserud et al. 2006). However, problems arise when it comes to specificity and determining exactly which specific trait should be considered the key marker function for the disorder. For instance, in ADHD, it is still not possible to determine whether attention, activity, or impulsivity aspects/functions should be considered core features of the “disorder.” Similarly, in ASD, it is not possible to assess the core quality of repetitive behaviors or, for that matter, perceptual functions, when it comes to delineating the “syndrome” of ASD. In the latter case – to “fully cover” the clinical spectrum of the “autistic state” in a given individual – it might be necessary to provide centile values for three or more continuous distribution curves, for example, empathy, central coherence, and rigidity-flexibility, and this would entail a great deal of conceptual and practical problems in clinical practice.

There are other problems with the continuous distribution model. First, it is as difficult to reasonably determine cutoff for abnormality under this model as it is in the general medical model of categorical disorders. Second, there are quite a number of instances, for instance, in ASD, when the model is totally inappropriate. It would not be correct or logical to categorize a case of autism caused by herpes encephalitis as being on a distribution curve shading into “normality.” Third, and not the least, there is a need for quick and dirty labels such as ASD and ADHD, much like there is a need for terms like “fever” and “pneumonia” (imprecise and even more vague terms than those used in neuropsychiatry). One of the most important features of a diagnostic label is its “door-opening” quality; by having a label, one will have easy access to knowledge. Having been given a percentage on a normal distribution curve, or worse, multiple different percentages on different curves will possibly be closer to “the

truth” but will often lead to more confusion than clarity. Having said this, the continuous distribution model has much to offer in second-level diagnostics: once a diagnosis of, for instance, ASD has been made, providing information about the individual’s level of functioning on a number of continuous distribution curves might actually help create a much more detailed (and holistic) view of that person’s functioning.

Current Knowledge

The DSM with a Particular Focus on Autism

As more and more research has documented the dimensional nature of so many core psychiatric disorders (including autism), the rigid structure and algorithmic nature of the DSM have come under increasing criticism. The inclusion of dimensional elements in the psychiatric diagnostic systems has been advocated for many years. However, it has been resisted due to concerns about clinical utility.

The categories in DSM are prototypes; a patient with a close approximation to the prototype is said to have that disorder. Each category of disorder has a numeric code taken from the ICD system, used for administrative purposes. One problem with this approach to diagnosis is that it does not properly deal with all those instances when a patient is severely impaired but does not meet *all* the criteria for a given discrete disorder. Every day in clinical practice (and in research), this is illustrated by diagnosis in the field of autism and related disorders. Many Western societies now have legislation specifically for autism. This means that having a “correct” diagnosis (i.e., one that fits with federal legislation) is extremely important. In needy clinical patients and in research prevalence studies, the categorical nature of the DSM system can be the arbiter between help and no help in terms of service provision and between case and noncase in epidemiological studies.

The way in which authors have articulated the multiple manifestations of autism has differed over time. Progress has been made in recent years, and this has brought about a convergence

on a shared definition of autism, including methods of assessment that are acceptable to workers from clinical and research centers across the world. Structured interviews (e.g., the DISCO-11, the ADI-R, and the ASDI) and observation schedules (including the ADOS-G) have brought organizational focus to the traditional psychiatric interview and developmental assessment. Such methods have provided a stricter format and directions to the interviewer, which, in turn, have enabled systematic assessment of *all* the criteria necessary for a diagnosis according to the given diagnostic (e.g., DSM) system. Having a consensually shared set of diagnostic criteria as well as structured assessment devices has helped ensure a more common unit of analysis in clinical practice and research across the globe. Though most workers would consider the operationalization of diagnostic criteria as an advance in psychiatry and developmental medicine, there remain concerns about the impact that the quest for increased diagnostic reliability might have on validity.

Current Clinical Practice and Research Use of the DSM

The DSM is primarily concerned with the symptoms and behavioral manifestation of mental disorders. With the exception of a small number of disorders (including “reactive attachment disorder”), it does not generally attempt to analyze or explain the conditions included in the manual.

The DSM-IV organizes each psychiatric diagnosis into five levels (axes) relating to different aspects of disorder or disability. Appropriate use of the DSM diagnostic criteria requires extensive clinical training, and its contents cannot be applied in a cookbook fashion. There is a risk that patients and nonmedical professionals may use the DSM in a checklist fashion and make “diagnosis” according to number of checked symptoms. It needs to be stressed that the DSM is a manual for medical psychiatric diagnosis. In practice, this means that it can only be used by highly skilled professionals making a definitive clinical diagnosis (i.e., medical doctors with specialist training in psychiatry and for some

disorders, including autism, ADHD, DCD, etc., those with training in neurology and developmental medicine).

Other, highly skilled, professionals use the DSM in clinical research. However, research diagnoses should not uncritically be equated with clinical diagnoses, and if a psychiatrist or other specifically trained medical doctor has not been involved in the diagnostic process, the “DSM diagnosis” should not be considered a psychiatric or medical diagnosis.

The DSM-5 published proposed diagnostic criteria in 2010 and revised proposed criteria in 2011. There was opportunity for specialists and the general public to react to these, and criteria were revised in the process. Once this was accomplished, the criteria were then tested in field trials. The results of these trials are not at hand at the publication of this volume.

Although the DSM-5 may move away from this categorical approach in some limited areas, some argue that a fully dimensional spectrum or complaint-oriented approach would better reflect the evidence (Krueger et al. 2005). Nevertheless, it is very difficult to envisage an overall change leading to fully dimensional diagnostics in psychiatry, given that it would not only be very difficult in practice but that it would entail a break with the tradition of categorical medical diagnosis that has a history of thousands of years.

Alternative Diagnostic Categories and Systems

Multiple Complex Developmental Disorder (MCDD)

The concept of MCDD was introduced by Donald Cohen (Towbin et al. 1993) in an attempt to “define and validate criteria for an early onset, chronic syndrome of disturbances in affect modulation, social relatedness, and thinking.” This syndrome, combining elements of autism, psychosis, and affective disorder, was considered possible to delineate and to be related to earlier onset of symptoms, very poor social and overall functioning, often long periods of inpatient treatment, and poor outcome.

Deficits in Attention, Motor Control, and Perception (DAMP)

The concept of DAMP was introduced by I Carina Gillberg (1987). It refers to the combination of problems in the domain of attentional abilities and motor-perceptual capacities in individuals who do not meet criteria for cerebral palsy. She and her colleagues had researched the clinical concept of minimal brain dysfunction (MBD) for a long time and had found that children thus diagnosed usually had this particular combination of problems (referred to as “perceptual, motor, and attentional deficits” as early as 1982). In later publications (e.g., Kadesjö and Gillberg 1999; Rasmussen and Gillberg 2000), DAMP was seen to correspond to the combination of ADHD and DCD. Gillberg (1983) noted that “severe” DAMP was strongly associated with marked autistic features and found that a large proportion of those diagnosed with “DAMP with autistic features” (=ADHD + DCD + autistic traits) actually met full diagnostic criteria for Asperger syndrome.

Disorders of Empathy

In the early 1990s, Gillberg launched the label of disorders of empathy and suggested that empathy and theory of mind were concepts that referred to closely related or perhaps even identical human functions (Gillberg 1992). He also proposed the concept of an empathy quotient (EQ) that might be used in a fashion similar to IQ when thinking about how ASD and related disorders could best be delineated from each other, from autistic traits and so-called normality. It was envisaged that a battery of tests of empathy including precursors of and mature-level theory of mind (and possibly subtests of facial recognition, central coherence, and set-shifting) would be developed so that disorders within the field could be diagnosed along a scale where an EQ of 70 might be set to demarcate cutoff for milder disorders (including that associated with the “Asperger phenotype”) and an EQ of 50 for more severe disorders (including the phenotype of “classic autism”).

Unfortunately, even though progress has been made regarding the understanding of the relationship between theory of mind, central coherence, executive function, and various types of disorders,

no “IQ-similar” EQ-test battery has been developed over the past two decades. Nevertheless, the concept of disorders of empathy (with autistic traits blending into “normality”) has gained considerable theoretical support over the last 20 years. It is still envisaged that having access to a test battery covering the basic functions and dysfunctions that have been shown to be clearly related to autistic symptoms would be extremely helpful and would pave the way for a “real” alternative ASD diagnostic system, clearly conceptually different from the one that will still be espoused in the DSM-5.

Nonverbal Learning Disability

The concept of nonverbal learning disorder or disability was introduced in a book by Rourke in 1988 and in an influential paper in 1989 (Rourke et al. 1989). The “diagnosis” – which is not in any of the official diagnostic manuals – rests on a considerable discrepancy between verbal and nonverbal skills on tests in individuals who are relatively proficient in expressive language skills. Affected individuals are often motor clumsy, perceptually abnormal, socially awkward, “dyspraxic,” and with poor pragmatic skills (in spite of sometimes superior formal verbal skills). Rourke has suggested that the overlap between nonverbal learning disability and ASD/Asperger syndrome is substantial.

Early Symptomatic Syndromes Eliciting Neurodevelopmental Clinical Examinations (ESSENCE)

The ESSENCE concept was introduced by Gillberg (2010). The acronym refers to early symptomatic syndromes eliciting neurodevelopmental clinical examinations. Gillberg coined this acronym with a view to alerting clinicians and researchers to the reality of a very large number of children (and their parents) presenting in clinical settings with impairing, persistent symptoms before age 3 (to 5) years – symptoms that will endure and overlap for many years, usually into adulthood – in the fields of (a) general development, (b) communication and language, (c) social interrelatedness, (d) motor coordination, (e) attention, (f) activity, (g) behavior, (h) mood, and/or (i) sleep. Children with major difficulties in one or more (usually several) of

these fields, will be referred to and seen by health visitors, nurses, social workers, education specialists, pediatricians, GPs, speech and language therapists, audiologists, child neurologists, child psychiatrists, psychologists, neurophysiologists, dentists, clinical geneticists, occupational therapists, and physiotherapists. Usually they will be seen only by one of these specialists, when they would have needed the input of two or more of the experts referred to. Major problems in at least one ESSENCE domain before age 5 years usually signal major problems in the same or overlapping domains years later. "There is no time to wait; something needs to be done, and that something is unlikely to be just in the area of speech and language, just in the area of autism or just in special education."

ESSENCE is not a new proposed diagnosis but represents an alternative way of approaching the problem of diagnosis in "child neuropsychiatry" and "developmental medicine." At very young ages, children with developmental problems present for diagnosis in a variety of settings, and depending on the type of specialist in charge, one or another of the many possible diagnoses contained in the ESSENCE basket is likely to be made (or not made for that matter). The risk is obvious that only the diagnosed problem type will be intervened for (or that the child excluded from, say, the autism category will not be worked up for his/her very real ADHD and hence excluded from relevant therapy). ESSENCE may be the "only safe label" at an early age. However, ESSENCE is not a diagnosis but a reminder that the child with that "label" will, sooner or later, have one, two, three, or even more diagnoses made. ESSENCE is a label that acknowledges the universal coexistence of symptoms and problems across diagnostic borderlines. All the problems need to be addressed, not just those associated with one discrete diagnostic category.

Future Directions

The DSM-5 and the ICD-11

Major attempts are being made to streamline the DSM-5 and the ICD-11. Several of the personality disorder categories will be gone from the DSM-5,

and a few new categories of psychiatric disorder will be included. It is expected that autism will become one category (no longer referred to as pervasive developmental disorder but, most probably, "autism spectrum disorder") and that subgrouping will be done on the basis of a number of "nonautism" demographics such as level of IQ, language competence, and severity.

Comorbidity and the DSM System

The term "comorbidity" was introduced in medicine to denote those cases in which a "distinct additional clinical entity" occurred during the clinical course of a patient having an index disease. This term has recently become very fashionable in psychiatry and developmental medicine to indicate not only those cases in which a patient receives both a psychiatric and a general medical diagnosis (e.g., autism and tuberous sclerosis) but also those cases in which a patient receives two or more psychiatric diagnoses (e.g., autism and Tourette syndrome). Gillberg (1983) pointed to this overlap of "discrete" psychiatric diagnoses in young children long before the word "psychiatric comorbidity" came into common parlance. The co-occurrence of two or more psychiatric diagnoses has been reported to be very frequent. For instance, in a general population study, 85% of young children with ADHD had at least one additional DSM diagnosis leading to impairment (Kadesjö and Gillberg 2001). In the case of severe autism, it is virtually impossible to find one single case in which there was no other mental or physical disorder. If a diagnosis of autistic disorder according to the DSM-IV-TR is made, one would have to be on the lookout for intellectual developmental disorder/mental retardation/learning disability, epilepsy, a medical disorder such as tuberous sclerosis or 22q11 deletion syndrome, neuropsychiatric disorder such as Tourette syndrome or ADHD, mood disorder, anxiety disorder, eating disorder, sleep disorder, or a specific developmental disorder such as developmental coordination disorder (DCD). There is a further diagnostic problem stemming from the fact that a majority of these other named disorders have a large subgroup with ASD, that the symptoms of all the disorders first appear and

overlap at a very early age, and that it can be very difficult to decide from the start which of the problem types is going to be the “main diagnosis,” that is, the one (or the ones) that will warrant intervention. The acronym ESSENCE has been introduced in order to draw attention to this state of affairs (Gillberg 2010).

The co-occurrence of multiple registered psychiatric diagnoses is now common. This is to some extent due to the use of standardized diagnostic interviews, which helps to identify several clinical aspects that in the past remained unnoticed after the principal diagnosis had been made. Fragmenting a complex clinical condition into several pieces may prevent a holistic approach to the individual.

An obvious determinant of the emergence of the phenomenon of “psychiatric comorbidity” (see below) has been the proliferation of diagnostic categories in recent classifications. If demarcations are made where they do not “really” exist, the probability that several diagnoses have to be made in an individual case will obviously increase.

A coveted tradition in psychiatry and developmental medicine has been to establish a hierarchy of diagnostic categories so that, for example, if autism were present, the possibly concomitant anxiety, depression, or ADHD would not be diagnosed because they would be regarded as part of the clinical picture of autism.

Because everyone has now been using operationalized diagnostic criteria for three decades or more, diagnoses such as autistic disorder have, by some, come to be regarded as more reliable than traditional clinical diagnoses. The old clinical descriptions provided a gestalt of each diagnostic entity. Different emphasis was put on the various clinical aspects, whereas current operational definitions usually give equal weight to a variety of clinical manifestations, *counting* symptoms rather than *weighing* them. Traditional clinical assessment demanded arbiter differential diagnosis, whereas current operational definitions really open up for multiple diagnoses (even though the DSM-IV often actively resists this), possibly in part because they are less able to convey the “essence” of each diagnostic entity.

Along with the trend as regards reliance on operationalized algorithms for diagnosis, has emerged a new insistence on “specific” instruments for these checklist categorical disorders. This is particularly true in autism, where both clinicians and researchers have been overtaken by an industry of diagnostic interviews and observation schedules that purportedly increase the quality of the (single) diagnosis *per se*. It is important to remember that these instruments were developed on the basis of studies using gold standard clinical diagnosis and that they will never, in themselves, be better than such diagnoses. It is envisaged that the heyday of these instruments will be over in the next few years and that they will be replaced by measures more accurately acknowledging and reflecting the developmental and overlapping nature of the conditions in question.

The frequent co-occurrence of the mental disorders has been taken as evidence against the idea that these disorders represent discrete disease entities (Cloninger 2002). The point has been made that psychopathology is usually complex and variable and that what is currently conceptualized as the co-occurrence of multiple disorders could be better reformulated as the complexity of many psychiatric conditions (with increasing complexity being a predictor of greater severity, disability, and service utilization). Even Kraepelin, in one of his later works, dismissed the model of discrete disease entities even for dementia praecox and manic-depressive disorder (Kraepelin 1920).

However, an alternative possibility is that psychopathology does consist of discrete entities, but these entities are not well delineated by current diagnostic categories. If this is the case, then current clinical research on “psychiatric comorbidity” may be helpful in the search for “true” disease entities, contributing in the long term to a rearrangement of present classifications.

There is, of course, another possibility, namely, that the nature of psychopathology is intrinsically heterogeneous, consisting partly of disease entities and categorical disorders, and partly of maladaptive response patterns or of exaggeration of traits that are more or less normally distributed in the general population.

ASD in the DSM-IV and the DSM-5

The DSM-IV comprised of five different autism spectrum disorder categories. The DSM-5 contains only one autism category, incorporating autistic disorder, Asperger's disorder, childhood disintegrative disorder, and PDDNOS into one common coded condition referred to as "ASD" (and leaving, reasonably, Rett syndrome out of the equation).

The change reflects increasing awareness that much of the DSM-IV subgrouping of autism was based on attitudes and personal stance rather than empirical evidence. For instance, most systematic studies have not found support for a clear distinction between autistic disorder and Asperger's disorder. It is also unclear to what extent CDD should be seen as different from autistic disorder with regression, and whether or not "mild" or highly atypical cases of PDDNOS are really related to autistic disorder at all.

There are only seven symptoms in the proposed DSM-5 as compared with 12 in the DSM-IV. There are only two subgroups of symptoms rather than three. The change in number of symptoms superficially gives the impression of a major reconceptualization of the whole category. However, on closer inspection, what has been achieved is a pruning of several symptoms that were felt by many to be vague and relatively unimportant or to be hallmarks of other conditions (such as severe learning disability or severe expressive language disorder), a collapsing of some of the remaining ones, and the addition of a behavioral criterion of perceptual abnormality. Also, the social and communication categories have been collapsed into one. This mirrors the now generally accepted notion that at the root of both the social and communication problems in autism is a shared deficit in intuitive understanding of the meaning of reciprocity. Finally, the three specific social-communication symptoms in the DSM-5 must all be met for a diagnosis to be considered (compared to only two out of four in the DSM-IV), and there must be at least five of the seven total number of symptoms met (compared to "only" 6 of the 12 autistic disorder criteria in the DSM-IV). The age criterion has been changed from delay or abnormal functioning being evident

before age 3 years (DSM-IV) to symptoms having been present from early childhood (DSM-5).

Taken together, it would seem that the proposed DSM-5 might actually restrict somewhat the number of cases of autistic disorder meeting full criteria for autism spectrum disorder compared to the DSM-IV. Also, many of the cases meeting Asperger's disorder symptom criteria (only three symptoms in total needed in the DSM-IV) and PDDNOS "criteria" (that are really extremely vague) would probably fall short of diagnostic status under the DSM-5. The Gillberg's Asperger syndrome category would, on the other hand, at least at a glance usually meet criteria for ASD under the DSM-5. However, all of this is, of course, pure speculation at the present time. Changing the diagnostic criteria, as with the introduction of the DSM-5 (ICD-11), will definitely lead to changes in numbers of cases diagnosed. This, in the case of autism, will, almost certainly, lead to claims of "autism epidemics" or "autism disappearing" in the headlines of many major newspapers from about 2015 onward. This is the extent of what can be reasonably predicted as a result of the introduction of the new diagnostic manuals.

Alternative Diagnostic Systems

MCDD

The following diagnostic criteria for MCDD (or multiplex developmental disorder) have been suggested by the Yale Autism Study Group: (1) *impaired social behavior/sensitivity, similar to that seen in autism*, such as (a) social disinterest, (b) detachment, avoidance of others, or withdrawal, (c) impaired peer relations, (d) highly ambivalent attachments, (e) limited capacity for empathy or understanding what others are thinking or feeling; (2) *affective symptoms*, including (a) impaired regulation of feelings, (b) intense, inappropriate anxiety, (c) recurrent panic, (d) emotional lability without obvious cause; (3) *thought disorder symptoms*, such as (a) sudden, irrational intrusions on normal thoughts, (b) magical thinking, (c) confusion between reality and fantasy, (d) delusions such as paranoid thoughts or fantasies of special powers.

A few studies have tried to examine the relative proportion of MCDD cases within the broader category of ASD. They have found the “condition” to be rare, accounting for fewer than one in ten of all relatively high-functioning cases (Sturm et al. 2004).

It is clear that the combination of problems subsumed under the MCDD heading exists in a small number of individuals and that those affected are very severely impaired. However, studies that have attempted to separate out children with MCDD from those with other “variants” of PDD or schizophrenia have usually not been able to clearly differentiate them from those with other diagnoses. Nevertheless, MCDD, if it will remain as an alternative category, is a diagnostic label that will only be applied in a limited number of patients presenting with ASD symptomatology. In some ways, it resembles the DAMP concept (see below) in that it could possibly be categorized as the concomitant presence of two “discrete” disorders, namely, ASD and schizophreniform disorder.

DAMP

DAMP, when defined as the combination of ADHD and DCD, is a common clinical problem (affecting several percent of all school-age children) that has well-documented ramifications both as regards need for intervention and prognosis (Rasmussen and Gillberg 2000). There are about 50 publications in the scientific literature. Stimulant treatment, cognitive behavioral therapy, special education measures, *and* occupational therapy are likely to be needed in any intervention program. Autistic features are very common and may need special approaches, and there is usually a speech and language component to be taken into account when designing the intervention plan. DAMP has been an accepted alternative clinical diagnostic concept in the Scandinavian countries for many years. However, given its literal meaning when read out as a word rather than as an acronym, it is unlikely that it will become generally accepted as an internationally used diagnostic concept. However, the insight into the common comorbidity

of ADHD with DCD (and of these two problem types with ASD) and the gradually growing awareness among clinicians that DCD is often a problem that should be treated “in its own right” will probably lead to acceptance of the importance of the underlying construct.

Disorders of Empathy

The gradual refinement of concepts such as self-initiated joint attention, theory of mind, central coherence/local-global processing and “connectivity,” facial emotion-recognition, mirror-neuron functions, and certain executive functions (including set-shifting) will possibly pave the way for development of age normed EQ tests that will allow a dimensional approach to diagnosis within the empathy spectrum disorders (or, using another term, ASD). Again, it is possible that the word empathy in itself might be seen by some to be too provocative, seeing as it has come to be associated with a positive (emotional) value (even though this was not its original meaning when the word was coined over a century ago). It could be that “disorders of social communication” will be a preferred term. Even so, it is likely that the concept of EQ (or SCQ, social communication quotient) will get rooted and upon up new avenues of diagnosing autistic traits across a range of problem types, just as the concept of IQ has come to be accepted as something useful when considering any type of problem, regardless of “other diagnoses.”

Nonverbal Learning Disability

One of the problems with the concept of nonverbal learning disability is that there does not seem to be any consensus regarding how it should be diagnosed. Most published studies have relied on results of IQ testing (often with one of the Wechsler scales), and the diagnosis has been made in cases with a verbal IQ that is 15 points (or 15–20% in some studies) higher than performance IQ. However, other authors, including Rourke, would instead use variations on the following diagnostic algorithm: a nonverbal learning disability refers to a subtype of learning-disabled children who have outstanding deficits in interpersonal relationships,

visual spatial organization, organization and planning skills, flexible concept formation, study skills, specific academic areas, and social judgment.

Several studies have attempted to delineate the boundaries between nonverbal learning disability on the one hand and Asperger syndrome on the other. One study has found a very high rate of nonverbal learning disability in young boys with Asperger syndrome; in fact, at least half of all young males with the syndrome had the typical verbal-nonverbal discrepancy (Cederlund and Gillberg 2004). However, when the same individuals were followed up in adult age, only one in five had clear test results indicating persistence of such a discrepancy, meaning that at least half of all those who had childhood indicators had “grown out” of “test evidence” of nonverbal problems after adolescence. Some studies have found no indication of a link between the “neuropsychological disorder” and the clinical syndrome of Asperger.

ESSENCE

It is likely that ESSENCE – or a similar concept – will become influential over the next several years. As has already been pointed out, ESSENCE is not in itself a diagnosis but a broader category covering a variety of neurodevelopmental, psychiatric, and neurological conditions that are sometimes behavioral phenotypes with a known etiology, sometimes empirically derived symptom clusters related to neuronal dysfunction, and sometimes the extreme on curves of normally distributed traits in the general population.

The term ESSENCE acknowledges the very common existence of such conditions and the fact that they are almost always “comorbid” with each other, that the comorbidities (and, indeed, the “main diagnosis”) may vary over time, weave in and out of each other, and that therefore the clinical picture tends to vary with age and time. ASD is but one category (or endpoint on a dimensionally distributed set of traits) within ESSENCE. ASD is virtually never an individual’s *only* problem; there is perhaps *always* an additional impairment that warrants clinical diagnosis and

intervention (including ADHD, tics, depression, anxiety, anorexia nervosa, an associated medical condition, epilepsy, DCD, cerebral palsy, hydrocephalus, catatonia, hyperlexia, dyslexia, speech and language disorder, intellectual developmental disorder, nonverbal learning disability). ESSENCE also flags up the possibility that ASD (or ADHD, tics, etc.) may not be the major clinically impairing problem throughout a person’s life that it can become less impairing with time (to the point that the need for a clinical diagnosis may be called into question) but that other so-called comorbidities (such as ADHD, depression, DCD, intellectual developmental disorder) may be seen as much more impairing and could, in fact, be main drivers of a poor outcome.

It is envisaged that over time, ESSENCE clinics, rather than (“overspecialized”) autism clinics, will be seen as the way forward. Children, adolescents, adults, and their families with one or more (usually several) of the problem types subsumed under the ESSENCE acronym (and remember that in many “ASD cases,” there is not only ESSENCE “comorbidity” in the individual referred for diagnostic workup but one or more of close relatives will also have ESSENCE problems) will need good diagnostic workup and intervention for *all* impairing problems, not “just” for ASD. This is not to say that good autism diagnostics and focused autism intervention will not be needed – quite the opposite – but that the strong emphasis on autism as a unique and separate syndrome may lead to inadvertent, underdiagnosis, and undertreatment of associated, highly treatable ESSENCE problems.

See Also

- ▶ [Asperger Syndrome](#)
- ▶ [Atypical Autism](#)
- ▶ [Autism](#)
- ▶ [Autistic Disorder](#)
- ▶ [Broader Autism Phenotype](#)
- ▶ [Child Behavior Checklist in Autism](#)
- ▶ [Childhood Disintegrative Disorder](#)
- ▶ [Clinical Assessment](#)
- ▶ [Comorbidity](#)

- Dimensional Versus Categorical Classification
- DISCO
- DSM-IV
- Early Diagnosis
- Endophenotypes
- Epidemiology
- Face Validity
- Facilitated Communication
- ICD 10 Research Diagnostic Guidelines
- Medical Conditions Associated with Autism
- Nonverbal Learning Disabilities (NLD)
- Psychotic Disorder
- Schizophrenia
- Screening Measures
- Semantic Pragmatic Disorder
- Sensitivity and Specificity
- Spectrum/Continuum of Autism

References and Reading

- Achenbach, T. M., Becker, A., Dopfner, M., Heiervang, E., Roessner, V., Steinhausen, H. C., et al. (2008). Multicultural assessment of child and adolescent psychopathology with ASEBA and SDQ instruments: Research findings, applications, and future directions. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 49, 251–275.
- American Psychiatric Association. (1994). *The diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- American Psychiatric Association. (2011). Retrieved from <http://www.dsm5.org>
- Cederlund, M., & Gillberg, C. (2004). One hundred males with Asperger syndrome. *Developmental Medicine and Child Neurology*, 46, 652–656.
- Cloninger, C. R. (2002). The discovery of susceptibility genes for mental disorders. *Proceedings of the National Academy of Science in the United States of America*, 99(13), 365–367.
- Ehlers, S., & Gillberg, C. (1993). The epidemiology of Asperger syndrome. A total population study. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 34, 1327–1350.
- Gillberg, C. (1983). Perceptual, motor and attentional deficits in Swedish primary school children. Some child psychiatric aspects. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 24, 377–403.
- Gillberg, I. C. (1987). *Deficits in attention, motor control and perception: follow-up from pre-school to the early teens*. Doctoral thesis, Uppsala University, Uppsala.
- Gillberg, C. (1992). The Emanuel Miller memorial lecture 1991. Autism and autistic-like conditions: Subclasses among disorders of empathy. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 35, 813–842.
- Gillberg, C. (2010). The ESSENCE in child psychiatry: Early symptomatic syndromes eliciting neurodevelopmental clinical examinations. *Research in Developmental Disabilities*, 31, 1543–1551.
- Goodman, R. (1999). The extended version of the strengths and difficulties questionnaire as a guide to child psychiatric caseness and consequent burden. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 40, 791–799.
- Kadesjö, B., & Gillberg, C. (1999). Developmental coordination disorder in Swedish 7-year-old children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38, 820–828.
- Kadesjö, B., & Gillberg, C. (2001). The comorbidity of ADHD in the general population of Swedish school-age children. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 42, 487–492.
- Kraepelin, E. (1920). Die erscheinungsformen des irreseins. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, 62, 1–29.
- Krueger, R. F., Watson, D., & Barlow, D. H. (2005). Introduction to the special section: Toward a dimensionally based taxonomy of psychopathology. *Journal of Abnormal Psychology*, 114, 491–493.
- Posserud, M. B., Lundervold, A. J., & Gillberg, C. (2006). Autistic features in a total population of 7–9-year-old children assessed by the ASSQ (Autism Spectrum Screening Questionnaire). *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 47, 167–175.
- Rasmussen, P., & Gillberg, C. (2000). Natural outcome of ADHD with developmental coordination disorder at age 22 years: A controlled, longitudinal, community-based study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 1424–1431.
- Rourke, B., Young, G., & Leenaars, A. (1989). A childhood learning disability that predisposes those afflicted to adolescent and adult depression and suicide risk. *Journal of Learning Disabilities*, 22, 169–175.
- Sturm, H., Fernell, E., & Gillberg, C. (2004). Autism spectrum disorders in children with normal intellectual levels: associated impairments and subgroups. *Developmental Medicine and Child Neurology*, 46, 444–447.
- Towbin, K. E., Dykens, E. M., Pearson, G. S., & Cohen, D. J. (1993). Conceptualizing “borderline syndrome of childhood” and “childhood schizophrenia” as a developmental disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 775–782.
- World Health Organization. (1993). *International classification of diseases and disorders* (10th ed.). Geneva: Author.
- www.wikipedia.com (2012). Diagnostic manual of mental disorder. Wikipedia text partly cited.

Alti-Haloperidol

- Haloperidol

Amantadine

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Definition

This drug (known as 1-adamantylamine or 1-aminoadamantane) was first approved by the FDA in 1966 for the treatment of influenza; its effectiveness for the treatment of symptoms of Parkinson's disease and drug-induced movement problems (extrapyramidal effects and akathisia) was discovered accidentally. For the treatment of Parkinson's disease, it is used alone or in combination with other agents. The efficacy of its use for Parkinson's disease has been questioned in a recent review (Crosby et al. 2003). Because of growing resistance, it is not now recommended for use in influenza treatment.

There appear to be several mechanisms of action since the agent impacts multiple brain neurotransmitter systems. Central nervous system side effects include anxiety, agitation, and increased seizure activity. Other side effects have included skin problem and suicidal thoughts.

The drug has been used without FDA approval for various other purposes including in autism. In the largest study, King et al. (2001) treated a group of children and adolescents with amantadine using both parent- and clinician-based report measures in a placebo-controlled study. They noted a large placebo effect overall with clinician ratings but not parent ratings suggesting some possible benefit of the agent over placebo. Amantadine was well tolerated. The drug remains one of many agents that deserve study in autism.

References and Reading

Babington, P. W., & Spiegel, D. R. (2007). Treatment of catatonia with olanzapine and amantadine. *Psychosomatics*, 48(6), 534–536.

Crosby, N. J., Deane, K., & Clarke, C. E. (2003). Amantadine in Parkinson's disease. *Cochrane Database of Systematic Reviews*, 1, Art. No.: CD003468. <https://doi.org/10.1002/14651858.CD003468>.

King, B. H., Wright, D. M., et al. (2001). Double-blind, placebo-controlled study of amantadine hydrochloride in the treatment of children with autistic disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40(6), 658–665.

Webb, S. (2010). Drugmakers dance with autism. *Nature Biotechnology*, 28(8), 772–774.

Ambien

► [Zolpidem](#)

American Academy of Clinical Neuropsychology (AACN)

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Membership as of 5/13/20: 1,087 Active, 75 Senior, 643 Affiliate, and 183 Student members.

Mission Statement: AACN is the organization for those psychologists who have achieved board certification in the specialty of Clinical Neuropsychology, by the American Board of Clinical Neuropsychology (ABCN), under the auspices of the American Board of Professional Psychology (ABPP). Board Certification covers neuropsychological aspects of brain-behavior disorders in children, adults, and the elderly. AACN supports continued maintenance of standards in Clinical Neuropsychology through the established board certification process of ABCN. AACN supports the continued development of the ABCN examination process, and advocates for the standards represented by board certification. In addition, Child Subspecialty Certification was added in 2014. Individuals wishing to obtain this certification need to be board certified through ABCN, fill out an added application form, take an added written exam, and undergo an added practice sample review.

Major Activities: AACN has an annual meeting open both to members and nonmembers. The meeting includes an extensive continuing education program which will be of interest to all, including special courses for candidates for board certification and for AACN members and others to maintain specialty knowledge. *The Clinical Neuropsychologist* is the official journal of AACN. In addition to copies of AACN policy statements which can be accessed via the link, <https://www.tandfonline.com/toc/ntcn20/current>, *The Clinical Neuropsychologist* publishes all AACN official policies and documents. AACN also includes the journal *Child Neuropsychology* for all of its members.

American Academy of Neurology

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Major Areas or Mission Statement

American Academy of Neurology (AAN)
1080 Montreal Avenue
Saint Paul, MN 55116
(800) 879-1960
www.aan.com
Child Neurology Society (CNS)
1000 W. County Road E, Suite 290
Saint Paul, MN 55126
(651) 486-9447
www.childneurologysociety.org

The AAN is an international professional association of over 22,000 neurologists and neuroscience professionals dedicated to promoting neurologic care. Members include both adult and child neurologists. The AAN is the primary professional society for clinical neurologists. It is dedicated to maintaining awareness among its membership of clinical and scientific advances

that impact clinical care and to providing educational opportunities for maintaining the knowledge and skills of its members. The AAN also commissions subcommittees to develop practice guidelines that disseminate the state of the science on specific clinical issues that confront neurologists in their daily practices.

The CNS is a professional association of pediatric neurologists and developmental pediatricians in the United States, Canada, and worldwide devoted to optimizing the care of children with neurological and neurodevelopmental disorders. There are over 1500 members. Like the AAN, the CNS has an annual meeting with a program designed to disseminate the latest scientific and clinical advances related to child neurology and to maintain the skills and knowledge of its clinicians. The CNS provides practice guidelines, maintenance of certification support, and CME programming in child neurology and developmental pediatrics, including autism.

The AAN and CNS are dedicated to promoting the highest quality patient-centered neurologic care and enhancing member competence and career satisfaction.

Major Activities

The AAN and CNS provide scientific and clinical education for its members in many formats, commission the development of practice guidelines to support improved standards of care, and public leadership and advocacy for individuals impacted by neurologic and neurodevelopmental disorders. Both organizations have provided educational sessions and practice guidelines on autism and on many related/overlapping issues (see “► [Reading](#)” for examples).

References and Reading

- Ashwal, S., Russman, B., Blasco, P., Miller, G., Sandler, A., Shevell, M., et al. (2004). *Practice parameter: Diagnostic assessment of the child with cerebral palsy*. Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. *Neurology*, 62, 851-863. Current guideline.
- Ashwal, S., Michelson, D., Plawner, L., & Dobyns, B. (2009). *Practice parameter: Evaluation of the*

- child with microcephaly (an evidence-based review):* Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Child Neurology Society. *Neurology*, 73, 887–897. Current guideline.
- Filipek, P. A., Accardo, P. J., Ashwal, S., et al. (2000). *Practice parameter: screening and diagnosis of autism*: Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Child Neurology Society. *Neurology*, 55(4), 468–479. Current guideline.
- French J. A., Kanner A. M., Bautista J., Abou-Khalil B., Browne T., Harden C. L., Theodore W. H., Bazil C., Stern J., Schachter S. C., Bergen D., Hirtz D., Montouris G. D., Nespeca M., Gidal B., Marks W. J. Jr, Turk W. R., Fischer J. H., Bourgeois B., Wilner A., Faught R. E. Jr, Sachdeo R. C., Beydoun A., & Glauser T. A. (2004). *Efficacy and tolerability of the new anti-epileptic drugs I: Treatment of new onset epilepsy*: Report of the Therapeutics and Technology Assessment Subcommittee and Quality Standards Subcommittee of the Neurology and the American Epilepsy Society. *Neurology*, 62, 1252–1260. Update in progress.
- Hirtz, D., Berg, A., Bettis, D., Camfield, C., Camfield, P., Crumrine, P., et al. (2003). *Practice parameter: Treatment of the child with a first unprovoked seizures*. Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. *Neurology*, 166–175. Current guideline.
- Michelson, D. J., Shevell, M. I., Sherr, E. H., Moeschler, J. B., Gropman, A. L., & Ashwal, S. (2011). Evidence report: Genetic and metabolic testing on children with global developmental delay. *Neurology*, 77(17), 1629–1635.
- Shevell M., Ashwal S., Donley D., Flint J., Gingold M., Hirtz D., Majnemer A., Noetzel M., & Sheth R. D. (2003). *Practice parameter: Evaluation of the child with global developmental delay: Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society*. *Neurology*, 60, 367–380. Update in progress.

American Academy of Pediatrics

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Major Areas or Mission Statement

Membership as of August 2017: Approximately 66,000 members in the United States, Canada,

Mexico, and internationally including pediatricians, pediatric subspecialists, and surgical subspecialists belong to the American Academy of Pediatrics (AAP). Thirty-four thousand members are Board Certified in Pediatrics and can be listed as Fellows of the American Academy of Pediatrics or FAAP.

Major Areas or Mission Statement: “The mission of the AAP is to attain optimal physical, mental, and social health and well-being for all infants, children, adolescents, and young adults. To accomplish this mission, the AAP shall support the professional needs of its members.”

Landmark Contributions

Landmark Contributions: It was not until the late 1800s that the care of children began to emerge as a separate area of specialization within medicine. The recognition that growth and development, nutrition, and prevention of infectious diseases in increasingly urbanized communities required focused research led to the founding of the American Pediatric Society in 1888. The increasing number of physicians who limited their practices to the primary care of children in office settings resulted in the formation of the American Medical Association section on pediatrics in 1880. Proposed federal legislation to provide matching funds to states for infant welfare clinics was supported by the American Medical Association section on pediatrics in 1922, but not the leadership of the American Medical Association who saw it a potential initial step to socialized medicine. The commitment by physicians who cared for children to advocate for the welfare of children led to the formation of an independent organization, the American Academy of Pediatrics, in 1929. The original 35 members met in Detroit to establish a professional organization that recognized that the needs for disease prevention and health promotion in children were different than those for adults. In 1930, there were 304 members.

The AAP set out to support and develop the field of pediatrics. The *Journal of Pediatrics* began publication in 1932 and was the official journal until *Pediatrics* assumed that status in

1948. In collaboration with the American Pediatric Society and the AMA section on pediatrics, the AAP supported development of the American Board of Pediatrics in 1934 as an independent organization to establish formal training criteria and certification of expertise in the specialty of pediatrics as well as to approve and certify subspecialists within pediatrics. Specialists and subspecialists must now demonstrate an ongoing commitment to professional education and incorporate quality improvement into their practices to maintain certification.

The AAP has major initiatives regarding the education of professionals and of the public on disorders of childhood in addition to advocacy for the health and well-being of children and families including areas as diverse as disease prevention, behavioral health, education, and the environment. Publications such as the *Red Book* guide practice related to immunization and management of infectious diseases. The efforts of the AAP have been critical in the passage of legislation such as supporting health insurance for children (SCHIP) and Head Start. The policies and recommendations of the AAP guide the health care provided to children by pediatricians and serve to advise other organizations and agencies. In addition to the headquarters in Elk Grove Village, Illinois, it maintains an office in Washington, DC.

Major Activities

The AAP's major activities address member education, public education, advocacy for children and youth, and promotion of community-based research and demonstration projects.

Organization: The AAP is divided into 10 regional districts and 59 state chapters each with elected officials who represent the chapters in the national organization. It is also organized by interest areas within pediatrics into 13 councils and 52 sections. Twenty-seven committees advise the elected leadership of the AAP in the development of the AAP's positions and programs. Committees have interests as varied as injury and poison prevention, children with disabilities, sports medicine, nutrition, and child health financing.

Activities related to autism are primarily managed by the Council on Children with Disabilities, and its Autism Subcommittee, and the Section on Developmental and Behavioral Pediatrics. Other groups with specific interests related to autism include the sections on General Pediatrics in Office Settings, Complementary, Holistic, and Integrative Medicine; Genetics; Gastroenterology, Hepatology, and Nutrition; Injury, Violence, and Poison Prevention; and Neurology and the Council on Environmental Health.

Education: The AAP coordinates continuing education courses, annual scientific meetings, seminars, and online education for pediatricians to address ongoing educational needs. It publishes the journal *Pediatrics* to promote academic understanding of the health needs of children and youth. It also publishes *Pediatrics in Review* as a journal for continuing education, *AAP News* as a member's news magazine, and manuals on topics important to child health such as infectious diseases and school health. Books are written for families on topical areas such as toilet training, Attention Deficit Hyperactivity Disorder, and others. Brochures on many areas relevant to child health, development, behavior, and safety are available to pediatricians to provide information to their patients. To assist child health professionals and policy makers, the AAP committees prepare technical reports and policy statements to summarize current information for the providers and recommend health-care practices. Policies which recommend practice and clinical reports that summarize the medical literature are posted on the AAP website.

Publications related to autism include the informational brochures for families on autism and language delays published in 2007. That year, two clinical reports were published in *Pediatrics* on the assessment and the management of children with autism. Policies of related interest include developmental screening (2006), use of complementary and alternative medicine by children with chronic illness (2001), learning disabilities, dyslexia, and vision (2011). The Autism Toolkit was revised in 2012. A full listing is accessible at www.aap.org.

Public Education: Educational materials for families on common topics are published for

distribution in the context of anticipatory guidance in well-child care and as information related to specific concerns, as well as books on topics such as toilet training and ADHD. Web-based information for families is a priority of the www.healthychildren.org website.

Advocacy: The AAP has an office in Washington, DC that advocates at the federal level for children's health needs in emerging policies and legislation. AAP staff assist the state chapters of the AAP in state and local advocacy around issues such as child safety legislation, Autism insurance legislation, and insurance legislation that assure access to care for low-income children.

Research: Through the Pediatric Research in Office Settings (PROS) network and CATCH grant mechanisms, the AAP promotes research in the community that addresses health needs as well as program development. Interest areas include social, economic, and behavioral research in addition to provision of medical care and disease prevention.

References and Reading

- Adams, R. C., Levy, S. E., & Council on Children with Disabilities. (2017). Shared decision-making and children with disabilities: Pathways to consensus. *Pediatrics*, 139(6), e20170956.
- Council on Children with Disabilities. (2009). Supplemental security income (SSI) for children and youth with disabilities. *Pediatrics*, 124(6), 1702–1708.
- Donoghue, E. A., & Council on Early Childhood. (2017). Quality early education and child care from birth to kindergarten. *Pediatrics*, 140(2), e20171488.
- Handler, S. M., Fierson, W. M., The Section on Ophthalmology and Council on Children with Disabilities, American Academy of Ophthalmology, & American Association for Pediatric Ophthalmology and Strabismus, & American Association of Certified Orthoptists. (2011). Learning disabilities, dyslexia, and vision. *Pediatrics*, 127(3), e818–e856.
- Hauer, J., Houtrow, A. J., & Section on Hospice and Palliative Medicine, Council on Children with Disabilities. (2017). Pain assessment and treatment in children with significant impairment of the central nervous system. *Pediatrics*, 139(6), e20171002.
- <https://www.aap.org/en-us/about-the-aap/Committees-Councils-Sections/Council-on-Children-with-Disabilities/Pages/default.aspx>.
- <https://www.aap.org/en-us/about-the-aap/Committees-Councils-Sections/sodbp/Pages/default.aspx>.
- <https://www.aap.org/en-us/Pages/Default.aspx>. (August 27, 2017).
- Johnson, C. P., Myers, S. M., & Council on Children with Disabilities. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120, 1183–1215.
- Kuo, D. Z., Houtrow, A. J., & Council on Children with Disabilities. (2016). Recognition and management of medical complexity. *Pediatrics*, 138(6), e20163021.
- Lipkin, P. H., Okamoto, J., & the Council on Children with Disabilities and Council on School Health. (2015). The Individuals With Disabilities Education Act (IDEA) for children with special educational needs. *Pediatrics*, 136(6), e1650–e1662.
- Myers, S. M., Johnson, C. P., & Council on Children with Disabilities. (2007). Management of children with autism spectrum disorders. *Pediatrics*, 120, 1162–1182.
- Pearson, H. A. (2006). The 75th anniversary of the American Academy of Pediatrics. *Pediatrics*, 117, 1759–1762.
- Weiss, J. I., & Committee on Violence, and Poison Prevention. (2010). Prevention of drowning. *Pediatrics*, 126(1), e253–e262.

American Association on Intellectual and Developmental Disabilities (AAIDD)

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Major Areas or Mission Statement

The American Association on Intellectual and Developmental Disabilities (AAIDD) is an interdisciplinary professional society of members who have a professional focus in the area of intellectual and developmental disabilities. The AAIDD counts approximately 3,500 interdisciplinary professionals as its members. The membership structure includes professionals working in the field of intellectual disability and related developmental disabilities. Members can select from a tiered membership menu: basic, classic, standard, premium, and corporate. AAIDD is primarily a North

American professional association, but it also offers an “international” membership option and has international members from 55 countries. The corporate membership is available to centers or agencies who may join, garnering a reduction on membership dues for the employees affiliated with the corporate member.

Association members have the option of joining any of its 21 special interest groups (e.g., administration, education, psychology, health and wellness, criminal justice, etc.).

Mission Statement

AAIDD promotes progressive policies, sound research, effective practices, and universal human rights for people with intellectual and developmental disabilities.

AAIDD Has Adopted a 7-Point Set of Principles (or Core Values) Relative to Its Mission

- Cultivate and provide leadership in the field of intellectual and developmental disabilities that encompasses a diversity of disciplines, cultures, and perspectives.
- Enhance the skills, knowledge, rewards, and conditions of people currently working in the field and encourage promising students to pursue careers in the field of intellectual and developmental disabilities.
- Advance basic and applied research to prevent or minimize the effects of intellectual and developmental disabilities.
- Advance the assurance of all human rights of people with intellectual and developmental disabilities, including equality, individual dignity, choice, and respect.
- Promote genuine accommodations to expand participation in all aspects of life for people with intellectual and developmental disabilities, opportunities for choice and self-determination, and access to quality health, education, vocational, and other human services and supports.
- Influence positive attitudes and public awareness to contributions of people with intellectual and developmental disabilities.
- Establish partnerships and strategic alliances with organizations that share our values and goals.

AAIDD's Goals

1. Enhance the capacity of professionals who work with individuals with intellectual and developmental disabilities.
2. Promote the development of a society that fully includes individuals with intellectual and developmental disabilities.
3. Sustain an effective, responsive, well-managed, and responsibly governed organization.

Landmark Contributions

AAIDD was founded in 1876 and has since been the leader in setting the practice standards; publishing books, tests, and other resources; and influencing policy. AAIDD's first president in 1876 was the French physician Édouard Séguin, MD, regarded by many as the father of special education in the USA.

The AAIDD has led the field in establishing the definition and diagnostic criteria for intellectual disability for over a century. It is well established that a significant proportion of individuals with an autism spectrum disorder also have a co-occurring diagnosis of intellectual disability. Since its first definition of intellectual disability in 1910, AAIDD has revised its definition ten times to reflect the changes in research and understanding of this condition (currently in its 11th edition). The AAIDD definition of intellectual disability has historically been adopted by all federal and state governments as well as the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM) in defining intellectual disability. AAIDD is considered the professional authority in the area of intellectual disability.

In examining the history of the AAIDD, one quickly discovers that the organization has undergone a number of changes since it was founded in 1876. The chief among these changes is the position of the organization with regard to issues such as (a) etiology of the disability, (b) systems of classification, and (c) systems of support/intervention. The AAIDD was founded by a small group of superintendents of institutions for people with disabilities. The AAIDD's first annual

meeting was held at the Pennsylvania Training School for Idiotic and Feeble-minded Children (now called Elwyn) in Media, PA, on June 6, 1876 (Sloan and Stevens 1976), where it was founded under the name of “Association of Medical Officers of American Institutions for Idiotic and Feeble-minded Persons” (Sloan and Stevens).

The Association’s first constitution provided a framework for the goals of the association during its earliest days (Sloan and Stevens 1976, p. 1):

Article II: The object of the association shall be the discussion of all questions relating to the causes, conditions, and statistics of idiocy and to the management, training, and education of idiots and feeble-minded persons; it will also lend its influence to the establishment and fostering of institutions for this purpose.

Although the association’s policies have evolved over time, the common goal of reaching a better understanding of intellectual disability and serving to improve the lives of people with intellectual disability has remained unchanged throughout the years.

Changes in the association’s name serve as somewhat of a barometer for the shifting attitudes toward people with intellectual disability within our society at large. The association name changes have largely been driven by a move away from historical terminology that has acquired increasingly pejorative connotations. In 1910, the name of the association was changed to “American Association for the Study of the Feeble-minded.” This was the first of the several name changes for the association. The name was changed again in 1933 to “American Association on Mental Deficiency” (AAMD), which remained until 1987, when it officially became known as the “American Association on Mental Retardation.” The most recent change came in 2007, bringing with it the current name “American Association on Intellectual and Developmental Disabilities.” This change was driven by the increasing acceptance of “intellectual disability” as the replacement terminology for mental retardation. AAIDD also chose to include “developmental disabilities” in its name to reflect its mission and influence in areas such as autism spectrum disorder, cerebral palsy, and other related developmental disabilities.

A landmark change brought about by AAIDD was in 1959 when it introduced the construct of adaptive behavior into its definition of intellectual disability (Heber 1959). The 1959 AAIDD terminology and classification manual first introduced deficits in adaptive functioning as part of the diagnostic criteria for intellectual disability. All other major diagnostic systems (e.g., World Health Organization’s International Classification of Diseases, American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorders*) as well as federal and state agencies followed suit. AAIDD also published the first standardized measure of adaptive behavior in 1969 – titled the AAMD Adaptive Behavior Scale (Nihira et al. 1969).

AAIDD has long been active in influencing legislation and social action toward improving treatment and supports for people with intellectual and developmental disabilities.

Throughout the years, AAIDD has served as *amicus curiae* in many cases regarding the rights of people with intellectual disability (Croser 1999; Herr 1999). James W. Ellis, JD, a Professor of Law at the University of New Mexico and past president of AAIDD, successfully argued before the US Supreme Court (*Atkins v. Virginia* 2002) that the execution of people with ID was cruel and an unusual punishment. The *Atkins v. Virginia* Supreme Court ruling led to the banning of capital punishment for all people diagnosed with ID and upheld in two subsequent Supreme Court decisions (e.g., *Hall v. Florida* 2014; *Moore v. Texas* 2017). AAIDD was prominently mentioned in the 2002 *Atkins v. Virginia* as well as both follow-up Supreme Court decisions as a leading national organization in setting the national consensus regarding the definition and diagnosis of intellectual disability.

Major Activities

The association offers a wide array of trainings, including an annual professional meeting. AAIDD publishes books, journals, assessment instruments, and training materials. Among its publications, AAIDD publishes two of the mostly

highly cited professional journals in the field of disabilities: *American Journal on Intellectual and Developmental Disabilities* and *Intellectual and Developmental Disabilities*. Many of its publications have been translated into dozens of languages and are disseminated and used worldwide.

See Also

- [Developmental Disabilities](#)
- [Diagnosis and Classification](#)
- [Intellectual Disability](#)
- [Mental Retardation](#)

References and Reading

- Atkins V. Virginia. (2002). *536 U.S. 304*.
- Blatt, B., & Kaplan, F. (1974). *Christmas in purgatory*. Syracuse: Human Policy.
- Croser, M. D. (1999). Federal disability legislation: 1975–1999. In R. L. Schalock, P. C. Baker, & M. D. Croser (Eds.), *Embarking on a new century: Mental retardation at the end of the 20th century* (pp. 3–16). Washington, DC: American Association on Mental Retardation.
- Hall V. Florida. (2014). *134 S. Ct. at 1986*.
- Heber, R. (1959). A manual on terminology and classification in mental retardation: A monograph supplement. *American Journal of Mental Deficiency*, 64(2), 1–111.
- Herr, S. S. (1999). Presidential address 1999 – working for justice: Responsibilities for the next millennium. *Mental Retardation*, 37(5), 407–419.
- Moore V. Texas. (2017). *581 U.S. ____*.
- Nihira, K., Foster, R., Shellhaas, M., & Leland, H. (1969). *AAMD adaptive behavior scale*. Washington, DC: American Association on Mental Deficiency.
- Schalock, R. L. (1999). Definitional issues. In R. L. Schalock, P. C. Baker, & M. D. Croser (Eds.), *Embarking on a new century: Mental retardation at the end of the 20th century* (pp. 45–66). Washington, DC: American Association on Mental Retardation.
- Schalock, R. L., Buntinx, W. H. E., Borthwick-Duffy, S., Luckasson, R., Snell, M. E., Tassé, M. J., & Wehmeyer, M. L. (2007). *User's guide: Mental retardation: Definition, classification, and systems of supports, 10th edition. Applications for clinicians, educators, disability program managers, and policy makers*. Washington, DC: American Association on Intellectual and Developmental Disabilities.
- Schalock, R. L., Buntinx, W. H. E., Borthwick-Duffy, S., Bradley, V., Craig, E. M., Coulter, D. L., et al. (2010). *Intellectual disability: Definition, classification, and system of supports* (11th ed.). Washington, DC: American Association on Intellectual and Developmental Disabilities.
- Sloan, W., & Stevens, H. E. (1976). *A century of concern: A history of the American association on mental deficiency*. Washington, DC: American Association on Mental Deficiency.
- Thompson, J. R., Bryant, B., Campbell, E. M., Craig, E. M., Hughes, C., Rotholz, D., et al. (2004). *Supports intensity scale: User manual*. Washington, DC: American Association on Mental Retardation.

American Board of Genetic Counseling

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Major Areas or Mission Statement

American Board of Genetic Counseling (ABGC)

Mission Statement

The American Board of Genetic Counseling establishes standards of competence through accreditation of graduate training programs and certification and recertification of genetic counselors to advance the profession and protect the public.

Membership

Currently there are 3,026 ABGC Certified Genetic Counselors.

The American Board of Genetic Counseling is a nonprofit organization incorporated in 1993 as the accrediting and credentialing body for the genetic counseling profession. The ABGC credential, Certified Genetic Counselor (CGC®), identifies counselors who have met established standards for graduate training with practical clinical experience, passed a comprehensive genetic counseling board examination, and demonstrate a commitment to maintain knowledge through recertification. The ABGC credentials have become recognized as the gold standard in the health care industry.

The ABGC organization is led by ten elected board members who serve a 5-year term. Board

members along with ABGC diplomates run committees, volunteer as item writers, and supervise genetic counseling training programs.

Landmark Contributions

The first genetic counseling training program graduated its master's-level genetic counselors in 1971. Since 1981, the American Board of Medical Genetics (ABMG) had been the body responsible for the certification of genetic counselors. A decade later, the American Board of Medical Specialties recognized genetics as a medical specialty and offered the ABMG an invitation to join. A condition of the membership was that the ABMG was required to exclude non-doctoral-level candidates from its certification process. An agreement was made for the formation of the ABGC. On October 23, 1992, the American Board of Genetic Counseling was incorporated to be the new accrediting and credentialing body for the genetic counseling profession.

The ABGC saw the opportunity to restructure the accreditation guidelines and the overall approach to accreditation. After carefully examining the accreditation practices of other specialties, it elected to accredit entire genetic counseling programs instead of only clinical training sites as had been done previously under the ABMG. In January 1994, a meeting was convened with board members of the ABGC and the genetic counseling program directors. A major objective of the meeting was to draft a set of practice-based competencies that an entry-level genetic counselor needs to demonstrate to effectively manage a genetic counseling session. These competencies served as the basis for the *Requirements for Graduate Programs in Genetic Counseling Seeking Accreditation by the American Board of Genetic Counseling*, adopted by the ABGC in January 1996. The 27 competencies are grouped into four domains (communication skills; critical-thinking skills; interpersonal, counseling, and psychosocial assessment skills; and professional ethics and values). These skills have become the cornerstone for curriculum design for programs seeking to achieve accreditation. The ABGC

moved away from the content-driven accreditation process developed under the ABMG with lists of courses and clinical contact hours, to an accreditation model that encourages the development of practice-based skills that integrate knowledge from several disciplines. With these practice-based competencies, the ABGC can hold the profession to a common set of expectations. The accreditation criteria for training programs are based on the program's ability to successfully develop these competencies in its genetic counseling graduates.

Additionally, ABGC established a recertification requirement for any diplomate certified in 1996 or later. Recertification was initiated to demonstrate a diplomate's commitment to maintaining knowledge in a rapidly evolving field. Through the recertification process, the ABGC strives to protect the public by ensuring the continuing education of genetic counselors. Recertification can be achieved in one of two ways: by either successfully passing another board exam or by collecting a specific number of continuing education units and professional activity credits over a specified period. Recertification has also proven significant for genetic counselors for licensing, professional advancement, hospital credentialing, and insurance reimbursement.

Since the formation of the ABGC, the number of Certified Genetic Counselors has grown from 495 to over 3,000. The number of accredited graduate training programs has increased from 18 to 33. By accrediting training programs, establishing competencies, and implementing recertification, the ABGC has been working hard to protect the public and promote the ongoing growth and development of practitioners in the genetic counseling profession.

Major Activities

The ABGC credentials genetic counselors and accredits genetic counseling training programs.

See Also

► [Genetics](#)

References and Reading

- Begleiter, M. (2002). Training for genetic counsellors. *Nature Reviews*, 3, 557–561.
- Boughman, J. (2007). Looking back; moving forward. *American Journal of Human Genetics*, 81, 422–423.
- Fiddler, B., Fine, B., Baker, D., & ABGC Consensus Development Consortium. (1996). A case-based approach to the development of practice-based competencies for accreditation of and training in graduate programs in genetic counseling. *Journal of Genetic Counseling*, 5, 105–112.
- Fine, B., Baker, D., Fiddler, M., & ABGC Consensus Development Consortium. (1996). Practice-based competencies for accreditation and training in graduate programs in genetic counseling. *Journal of Genetic Counseling*, 5, 113–121.

www.abgc.net

www.nsgc.net

American Congress of Rehabilitation Medicine

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Major Areas or Mission Statement

The purpose of the American Congress of Rehabilitation Medicine (ACRM) is to advance service delivery and research for people who have disabling conditions. There are four major areas of focus for this research:

1. To meet the needs of people with disabilities
2. Educate providers for best practice delivery of care
3. Promote the health, independence, quality of life, and productivity of disabled people
4. To ensure that future research projects are publicly funded

The primary mission of the ACRM is to enhance the lives of disabled people via a multidisciplinary rehabilitation approach. As a leader in the physical medicine and rehabilitation field, their mission is to promote innovative research and new technologies and encourage evidence-

based practices in clinical settings as well as encourage information sharing.

Leadership role ACRM creates forums where all rehabilitation professionals, including clinicians, service managers, administrators, educators, and researchers, can innovate. We call upon the leaders in rehabilitation to identify current best practices and best providers at all levels of care, and share this information via educational meetings and the journal.

Archives of physical medicine and rehabilitation. As rehabilitation science and medicine continues to evolve, the goal is to keep the community connected by creating opportunities to exchange and share information with rehabilitation professionals, corporate providers, health-care payers, and industry regulators.

ACRM aims to provide multidisciplinary leadership and practice innovation to ensure that people living with chronic disease and disabilities have access to effective rehabilitation management throughout their lives. It serves as a forum for creating and discussing new treatment paradigms that define the composition of the rehabilitation team, the duration of care, and the venues required to achieve optimal functional outcomes for people with chronic disease and disabilities.

ACRM is dedicated to

- Serving as advocates for public policy and legislative issues that support individuals with disabilities and providers of rehabilitation services
- Helping develop innovative and cost-effective models of collaborative care and comprehensive rehabilitation management
- Leading research efforts that examine and identify the most effective clinical technology and treatment paradigms
- Initiating dialogue with payers and regulators to communicate the collaborative care models that produce positive rehabilitation outcomes

Landmark Contributions

The current title of American Congress of Rehabilitation Medicine became official in 1966. However, this congress was initially founded in 1923

as the American College of Radiology and Physiotherapy. It began as a professional association of physicians who used physical agents to diagnose and treat disability and illness. In 1925, with medicine already moving more toward specialization, the radiology and physical medicine focuses split. It became the American Congress of Physical Therapy. At that time, the congress' primary journal was the Archives of Physical Therapy, X-ray, Radium, which had been founded in 1920. In 1938, the name was changed to Archives of Physical Therapy, which more accurately reflected its focus.

Over the next 6 years, the congress' focus narrowed further toward physical medicine, and in 1944, the name was again changed to reflect this new direction. It was now the American Congress of Physical Medicine, and in 1945, the name of its journal became the Archives of Physical Medicine. This change in emphasis reflected the distinction that was growing between physical therapy and physical medicine. Physical medicine moved away from a purely clinical approach toward a scientific and diagnostic basis of the medical use of physical agents. It allowed a distinction between physicians and technicians of physical therapy in accord with the new stance of the American Medical Association (AMA).

By 1952, the field of rehabilitation had significantly expanded following WWII. To reflect the close relationship between physical medicine and rehabilitation, the name was changed to the American Congress of Physical Medicine and Rehabilitation. The following year, the journal name was changed to its current version, *Archives of Physical Medicine and Rehabilitation*.

In 1965, the congress formed the Professional Development Committee (PDC) which was pivotal in the management and direction of the ACRM for the next 30 years. This committee's accomplishments included a study of the objectives, constitution, and structure of the congress as well as the sponsorship of interdisciplinary forums and an expansion of the membership.

The following year, several physician members recognized the need for a forum in which professionals of other rehabilitation disciplines could share their professional, scientific, and technical talents. This led to an amendment to the

congress' constitution allowing membership privileges to be extended to persons "holding an earned doctoral degree and active in and contributing to the advancement of the field of rehabilitation medicine." This allowed the membership of psychologists, nurses, physical therapists, occupational therapists, speech pathologists, social workers, vocational counselors, and others. And in the same year, 1966, the name was officially changed to the American Congress of Rehabilitation Medicine.

Major Activities

ACRM membership is focused on interdisciplinary communication and collaboration within the rehabilitation professional community. This is accomplished by providing special interest and networking groups within the community, as well as providing publications and conferences that facilitate ongoing research, reference resources, and up-to-date developments in the field of physical rehabilitation medicine. Some of the resources available include the following:

- Fellows of ACRM
- Archives of Physical Medicine and Rehabilitation – a leading journal in rehabilitation
- Cognitive Rehabilitation Manual: Translating Evidence-Based Recommendations into Practice
- ACRM eNews
- Progress in Rehabilitation Research – an annual conference that brings together experts and participants from 20+ countries
- Midyear meeting for members and leaders within the community to collaborate and share information and refine guideline development
- Community calendar compiles a list of upcoming networking events and educational course offerings

See Also

- ▶ [Certified Rehabilitation Counselor](#)
- ▶ [Occupational Therapy \(OT\)](#)
- ▶ [Physical Therapy](#)

References and Reading

American Academy of Physical Medicine and Rehabilitation (AAPM&R). www.aapmr.org
<http://www.acrm.org>
 International Rehabilitation Medicine Association (IRMA). www.isprm.org

American Medical Association

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Major Areas or Mission Statement

Membership is limited to physicians (with an M.D. or D.O. degree or international equivalent) who are in practice of residents in the United States and its possessions. Medical students can also enroll prior to completion of their training. About one quarter of US physicians are members of the organization.

The mission of the AMA is multifaceted and included improved public health, advocacy for physicians and their patients, and medical education. The AMA plays a major role in maintenance of medical coding that health-care providers use for reimbursement.

Landmark Contributions

The organization was founded in 1847 and incorporated 50 years later. It has a strong record of promotion of the scientific method in the practice of medicine and in the improvement of medical education. It also has had a major role in elaboration of principles of medical ethics and public health measures. It makes substantial contributions in support of medical students in financial need as well as grants for research and community projects. Over the years, many of its political positions have been controversial, for example,

including its initial opposition to Medicare. In recent years, it has focused on the disparities of health care and the special needs of some groups to medical services. Criticism of the organization has come from several sources including the noted economist Milton Friedman who argues that it has acted to limited competition.

Major Activities

The AMA publishes a series of journals in medicine. Of these, the *Journal of the American Medical Association* (JAMA) is the most prominent for the field of medicine in general, and the specialty journals of *Archives of General Psychiatry* and the *Archives of Pediatric and Adolescent Medicine* are most relevant to individuals with autism and other developmental disabilities.

See Also

► [American Psychiatric Association](#)

References

- Cassedy, J. H. (1991). *Medicine in America: A short history*. Baltimore: Johns Hopkins University Press.
 Duffy, J. (1993). *From humors to medical science: A history of American medicine*. Urbana: University of Illinois Press.

American Psychiatric Association

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Major Areas or Mission Statement

The mission of the American Psychiatric Association is to promote the highest quality care for

individuals with mental disorders (including mental retardation and substance-related disorders) and their families, promote psychiatric education and research, advance and represent the profession of psychiatry, and serve the professional needs of its membership.

Landmark Contributions

The American Psychiatric Association, founded in 1844, is the oldest national medical professional association in the United States and the world's largest psychiatric organization. Its member physicians work together to ensure humane care and effective treatment for all persons with mental disorders, including intellectual disability and substance-related disorders.

In 1948, APA formed a small task force to create a new standardized psychiatric classification system. This resulted in the 1952 publication of the first Diagnostic and Statistical Manual of Mental DisordersTM (DSM). The task force is currently developing DSM-5 to be published in May of 2013.

Major Activities

The APA publishes scientific journals:

The American Journal of Psychiatry publishes the latest advances in the diagnosis and treatment of mental illness. The findings presented in this journal explore the full spectrum of issues related to mental health diagnoses and treatment. Psychiatric Services, a journal of the American Psychiatric Association, is a journal for mental health professionals and others concerned with treatment and services for persons with mental illnesses and mental disabilities.

FOCUS: The Journal of Lifelong Learning in Psychiatry addresses clinical issues in psychiatry, featuring articles on current research including influential works selected by experts in the field. It also features an annual self-assessment exam and assists psychiatrists with recertification.

The APA's annual meeting brings together psychiatrists from all over the world to understand new research findings and acquire new knowledge and clinical issues in patient care.

See Also

- ▶ [American Medical Association](#)
- ▶ [DSM-IV](#)
- ▶ [Psychiatrist](#)

References and Reading

- American Psychiatric Association. (1944). *One hundred years of American psychiatry*. New York: Columbia University Press.
- American Psychiatric Association. (1966). *History of the district branches and of the district branch assembly*. Washington, DC: Author.
- Barton, W. E. (1987). *The history and influence of the American Psychiatric Association*. Washington, DC: American Psychiatric Press.
- Baxter, W. E., & Hathcox, D. W., III. (1994). *America's care of the mentally ill: A photographic history*. Washington, DC: American Psychiatric Press.
- Menninger, R. W., & Nemiah, J. C. (2000). *American psychiatry after World War II 1944–1994*. Washington, DC: American Psychiatric Press.
- Obenauf, W. H. (1959). The district branch of the APA: Its origin, present status, and future developments. *Journal of the American Psychiatric Association*, 116, 416–422.
- Sabshin, M. (2008). *Changing American psychiatry: A personal perspective*. Washington, DC: American Psychiatric Publishing.

American Psychological Association

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Major Areas or Mission Statement

The primary mission of the APA is to “advance the creation, communication and application of psychological knowledge to benefit society and

improve people's lives" (APA.org). The APA states that within this mission contains the aspiration and vision to excel as a valuable, effective, and influential organization advancing psychology as a science (American Psychological Association 2002). This is accomplished by the organization's efforts to be:

- A uniting force for the discipline
- The major catalyst for the stimulation, growth, and dissemination of psychological science and practice
- The primary resource for all psychologists
- The premier innovator in the education, development, and training of psychological scientists, practitioners, and educators
- The leading advocate for psychological knowledge and practice informing policy makers and the public to improve public policy and daily living
- A principal leader and global partner promoting psychological knowledge and methods to facilitate the resolution of personal, societal, and global challenges in diverse, multicultural, and international contexts
- An effective champion of the application of psychology to promote human rights, health, well-being, and dignity
- Promoting research in psychology, the improvement of research methods and conditions, and the application of research findings
- Improving the qualifications and usefulness of psychologists by establishing high standards of ethics, conduct, education, and achievement
- Increasing and disseminating psychological knowledge through meetings, professional contacts, reports, papers, discussions, and publications

Landmark Contributions

The APA was founded in 1892 by G. Stanley Hall at Clark University in Worcester, Massachusetts, with approximately 26 individuals accepting membership at the time of its formation. Since the time of its inception, the APA has held prominent and historical members in the field of psychology as its president including William James (1894), James McKeen Cattell (1895), Edward Thorndike (1912), Carl Rogers (1947), Harry Harlow (1958), Abraham Maslow (1968), Albert Bandura (1974), and Phillip Zimbardo (2002).

The APA was responsible for the formation, review, and revision of the ethical codes of conduct and standards of practice. The code itself "is intended to provide guidance for psychologists and standards of professional conduct that can be applied by the APA and by other bodies that choose to adopt them" (APA 2002). The Ethics Code contains the following five general principles that are aspirational in nature and intended to be viewed as a guide to the highest possible standards of ethical practice:

- Continual pursuit of excellence
- Knowledge and application based on methods of science
- Outstanding service to its members and to society
- Social justice, diversity, and inclusion
- Ethical action in all that we do
- Beneficence and nonmaleficence
- Fidelity and responsibility
- Integrity
- Justice
- Respect for people's rights and dignity

The APA espouses the goal of seeking to advance psychology as a science, a profession, and as a means of promoting health, education, and human welfare by promoting and maintaining the following actions:

- Encouraging the development and application of psychology in the broadest manner
- The APA also formulated ten ethical standards of practices with specific guidelines in areas of psychology's application to a variety of domains. The standards set forth by the APA are enforceable by law and provide a guiding framework for

the competent and ethical practice of psychology. The ethical standard domains encompass:

- Resolving ethical issues
- Competence
- Human relations
- Privacy and confidentiality
- Advertising and other public statements
- Record keeping and fees
- Education and training
- Research and publication
- Assessment
- Therapy

Since 1955, the APA has provided the Model Act for State Licensure of Psychologists as a prototype to aid in the drafting of each state's specific legislation regarding the practice and licensing of psychologists in their respective states. The document is also meant to educate and inform legislators about the training and practice of psychology. It has undergone periodic revisions and updates since its inception (APA Committee on Legislation 1955). In 1984, the Council of Representatives directed the Board of Professional Affairs (BPA) to develop another revision of the existing 1967 Model Act for the council's consideration. The Committee on Professional Practice (COPP) prepared the revised document, and it was approved by the Council of Representatives in February 1987 (American Psychological Association 1987). In 2006, the 1987 Model Act was again revised by a task force funded by the APA Board of Directors and Council of Representatives at the recommendation of the Board of Professional Affairs and the Committee for the Advancement of Professional Practice. The primary reason for the changes in the existing Model Act was that it did not reflect the developments in professional practice that had occurred over the preceding 20 years across respective states. Specific developments included the option for prescriptive authority in some states, changes to the provision of industrial/organizational and consulting psychology services encouraging licensure for psychologist practicing in those arenas, and changes in the recommended sequence of education and training for

psychologists. The task force provided a comprehensive review of the 1987 document as well as relevant APA policies and other documents before creating a finalized draft of the new act. The newest revision was approved by council in February 2010 and includes commentary and guidelines for the following areas related to the practice of professional psychology (American Psychological Association 2010):

- Declaration of policy
- Definitions
- State psychology boards
- Requirements for licensure
- Interstate practice of psychology
- Temporary authorization to practice
- Limitations of practice, maintaining and expanding competence
- Inactive status
- Practice without a license
- Exemptions
- Grounds for suspensions or revocation of licenses
- Board hearing and investigations
- Privileged communication
- Severability
- Effective date

The Publication Manual of the American Psychological Association, currently in its sixth edition, has provided guidelines and recommendations for publication style intended for writers, editors, students, and educators in the social and behavioral sciences. It has grown considerably since its first publication in February of 1929 as a seven-page instructional report (American Psychological Association 2001). Over the subsequent 70 years, these suggestions and instructions were revised and expanded across six editions to its present form. The current manual provides guidance on all aspects of the scientific writing process, from the ethics of authorship to the word choice that best reduces bias in language. The manual additionally provides guidance on choosing the headings, tables, figures, and tone that will result in strong, simple, and elegant scientific communication. Every edition of the Publication Manual has been intended to aid authors in the

preparation of manuscripts with the primary goal of providing a standardized communication that will efficiently convey new ideas and research and to simplify the tasks of publishers, editors, authors, and readers. This has further allowed for the linkages of electronic files across publishers and manuscripts as new technological advancements in communication and distribution have emerged. This includes the maintenance and management of the abstract database, PsycINFO, which collects and distributes electronic information from approximately 2,500 journals dating from 1,800 to present (APA.org). Over a thousand journals in psychology, the behavioral sciences, nursing, and personal administration use the *Publication Manual* as their specified style guide (APA 2001).

Major Activities

The APA exists and operates as an executive office, a publishing operation, and an office that addresses administrative, business, information technology, and operational needs. It also contains five substantive directorates that address the needs of the field of psychology in its respective areas:

- The Education Directorate accredits doctoral psychology programs and addresses issues related to psychology education in secondary through graduate education.
- The Practice Directorate engages on behalf of practicing psychologists and health-care consumers.
- The Public Interest Directorate advances psychology as a means of addressing the fundamental problems of human welfare and promoting the equitable and just treatment of all segments of society.
- The Public and Member Communications Directorate is responsible for APA's outreach to its members and affiliates and to the general public.
- The Science Directorate provides support and voice for psychological scientists.

The American Psychologist is the APA's official journal and most highly circulated

peer-reviewed publication. The APA also publishes 57 other journals across a wide range of specialty and focus areas (APA.org). The APA also hosts the largest national convention and gathering of psychologists in the United States in a different host city each year. The convention provides seminars, conferences, presentations, and networking for all areas of psychology in its respective areas of research and practice.

Each year, the APA recognizes the work of psychologists with its "Distinguished Contributions Award." The awards are considered among the highest honors given and include recognition in the following categories:

- Distinguished Scientific Contributions to Psychology
- Distinguished Contributions to Psychology in the Public Interest
- Distinguished Scientific Applications of Psychology
- Distinguished Contributions to Education and Training in Psychology
- Distinguished Professional Contributions to Applied Research
- Distinguished Professional Contributions to Practice in the Public Sector
- Distinguished Contributions to the International Advancement of Psychology

The APA participates in a commitment to be an international partner with the global psychological community. Its office of International Affairs promotes exchange and collaboration with international communities including the United Nations. There are over 7,000 international members and affiliates of the APA (APA.org).

The APA has periodically provided commentary, guidelines, and recommendations to specific issues of practice and applications of psychology that impact current world events and ethical issues. Such issues and world topics have included task force reports on appropriate therapeutic responses to sexual orientation (APA Task Force on Appropriate Therapeutic Responses to Sexual Orientation 2009) as well as the use of military interrogation tactics (American Psychological Association 2007).

Division 33 (Mental Retardation and Developmental Disabilities) of the American Psychological Association was formed in 1973 as a unified division for psychologists committed to advancing psychology practice and research for individuals with mental retardation and developmental disabilities. In order to more accurately recognize the breadth of conditions that are now recognized to constitute developmental disabilities (e.g., autism, Asperger's disorder), the division changed its name from Mental Retardation to Mental Retardation and Developmental Disabilities in 1988 and to Intellectual and Developmental Disabilities in 2007 (APA.org). The division consists of five special interest groups: behavior modification and technology, dual diagnosis, early intervention, aging and adult development, and transitioning into adulthood. Members of Division 33 receive the newsletter "Psychology in Mental Retardation and Developmental Disabilities" three times per year and have access to the division's Listserv.

See Also

- ▶ [American Psychiatric Association](#)
- ▶ [Clinical Psychology](#)
- ▶ [Psychologist](#)

References and Reading

- American Psychological Association. (1987). Model act for state licensure of psychologists. *American Psychologist*, 42, 696–703.
- American Psychological Association. (2001). *Publication manual of the American Psychological Association* (5th ed.). Washington, DC: Author.
- American Psychological Association. (2002). Ethical principles of psychologists and code of conduct. *American Psychologist*, 57, 1060–1073.
- American Psychological Association. (2007). American Psychological Association: Psychology and Interrogations. Submitted to the United States Senate Select Committee on Intelligence. September 21, 2007.
- American Psychological Association. (2010). *Model act for state licensure of psychologists*. Adopted by Council as APA Policy 02/20/2010, 1–16.
- APA Committee on Legislation. (1955). Joint report of the APA and CSPA (Conference of State Psychological Associations). *American Psychologist*, 10, 727–756.
- APA Task Force on Appropriate Therapeutic Responses to Sexual Orientation. (2009). *Report of the task force on appropriate therapeutic responses to sexual orientation*. Washington, DC: American Psychological Association. <http://www.apa.org>

American Sign Language (ASL)

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Definition

American Sign Language (ASL) is the natural and national sign language of the deaf community in the United States and parts of Canada (Neidel et al. 2000). It is a natural language because it has developed out of a need for deaf individuals to communicate with each other, and it is a language that is in constant evolution. It is a national language because it is mutually intelligible and separate from the sign languages that are used in other countries such as British Sign Language (Great Britain), Mexican Sign Language (Mexico), and so forth. ASL is a separate language from spoken English (Lane et al. 1996), and it is distinct from manual codes of English such as Seeing Essential English (SEE I), Signing Exact English (SEE II), Linguistics of Verbal English (LOVE), or Conceptually Accurate Signed English (CASE). Unlike most other languages, ASL is typically learned from peers rather than from one's parents (Padden 1980). This may be due to the fact that most deaf children (about 90 %) are born to hearing parents (Mitchell and Karchmer 2004) rather than to deaf parents who could pass along the language to their children.

Historical Background

The American Sign Language that is used today is a combination of Parisian sign language that was

introduced in 1817 by Laurent Clerc, a teacher of the deaf from France, and the sign language that was used by the large community of deaf Americans at Martha's Vineyard (Baynton 1996). Despite attempts by some members of the normally hearing community to extinguish the language, the ASL that was used in the mid-1800s is still intelligible today to ASL users (Baynton 1996). William C. Stokoe Jr. first described ASL in his publication *Sign Language Structure* (Stokoe 1960). In it, he argued that indeed American Sign Language was a true and natural language and not merely gestures or pantomime. Stokoe followed this work with the first dictionary of American Sign Language.

Current Knowledge

Since the work of William Stokoe, much study has been focused on ASL specifically and sign languages in general. The field of linguistics has a greater understanding of language thanks to comparisons made between spoken languages and sign languages. Like spoken languages, sign language is comprised of syntax, semantics, morphology, and phonology (Sandler and Lillo-Martin 2006). We have much greater understanding of communication processes and language universals due to research on deaf adults who are victims of stroke or traumatic brain injury with resulting aphasia in sign language. The left hemisphere of the brain is largely responsible for language processing of sign language just as it is for spoken language (Corina 1998; Poizner et al. 1987). Both fluent and nonfluent aphasias in sign have been documented as well as paraphasias resulting from disordered phonology and morphology. See Hickok et al. (1998) and Woll and Sharma (2008) for a review of the literature.

Because users of spoken language use gestures to augment their messages, there is recent research on the role of gesture for those who use sign. Vermeerbergen and Demey (2007) show that gesture and sign can coexist and are often combined into one sign. Also, interestingly, the mouth and hands may trade tasks in fluent signers. For nonsigners, the mouth is responsible for transmitting verbal

information, while the hands are largely responsible for gesturing to augment the message. For signers, the mouth may be responsible for gesturing, while the hands convey linguistic information.

Much recent work has been focused on using technology to enhance the lives of the deaf population. There is great potential for converting sign to text and text to sign to create faster and more efficient exchanges between the deaf and hearing populations. The complexities of sign language, however, have made it difficult to automate a translation system to convert signed conversations to text. Two of the most commonly used input devices for capturing sign language gestures are glove-type devices and computer vision systems. Each system has advantages and drawbacks.

There have been a number of different glove-based devices used for input purposes (Hernandez-Rebollar et al. 2004). These devices typically contain several sensors per finger to measure the way the fingers move and the angle of the fingers as well as sensors to measure the pitch and roll of the hand. Proponents of these systems show that these input devices are able to more precisely detect handshapes than video-based systems (Fels and Hinton 1993; Hernandez-Rebollar et al. 2004). There are several disadvantages posed by data-glove devices (Wang et al. 2007). While extremely accurate, these devices were typically bulky and cumbersome as an individual wearing this device needed to be physically attached to the device that connects to a computer by means of cables. This need to tether the device to a computer limited how and where these devices could be used. The need to be physically connected is changing with advances in technology. Newer devices are employing technology such as electro-optical or magnetic sensors and accelerometers along with wireless capabilities to compensate for many of the early data-glove limitations (Dipietro et al. 2008). Even with advances in technology, these devices might interfere with natural movement and thus self-expression for individuals using them. Another factor that limits the use of these devices is the expense, which is typically more than for vision-based systems, although some of the costs have been reduced with new technology.

Gesture recognition based on computer vision systems utilizes a camera to detect hand movements and handshapes. Generally, these systems detect movement or the skin color of the hand to segment and extract features that can be used to model the hand. While the actual processes that each system employs vary, three basic types of methods are used to extract hand features:

1. Model-based or kinematic methods seek to model the angles created by the palm and joints of the hand.
2. View-based or appearance-based methods use multiple two-dimensional intensity images to model gestures as a sequence of views to overcome some of the shortcomings of kinematic models.
3. Low-level feature-based methods utilize low-level measurements of the hand region.

These methods do not rely on re-creating an exact model of the hand but rather attempt to capture just enough of the essential information needed to recognize gestures.

Opponents of video-based gesture recognition state that video-based systems are less able to recognize handshapes (Hernandez-Rebollar et al. 2004; Starner et al. 1997; Starner and Pentland 1998). Other challenges that video-based systems must overcome are specific lighting conditions needed to accurately capture the intended target as well as camera placement. Additionally, the subject being captured must remain in frame and the camera must not be obscured while recognition is underway. These limitations, particularly in earlier systems, made video-based systems difficult to use outside of the laboratory setting. Additionally processing the collected information to extract necessary features requires large amounts of computation that makes real-time processing difficult.

Current techniques in video-based gesture recognition address some of the earlier challenges including using multiple cameras, faster cameras, and better controlled environments and even having the users wear specially colored gloves (Murthy and Jadon 2009; Wang et al. 2007). In addition, the processing of the data collected has

improved by implementing processes such as hidden Markov models and the use of neural networks, but these tasks are still computationally expensive (Murthy and Jadon 2009).

Future Directions

The relationship between language and cognition is an area of continued interest and research. Much more can be learned regarding the processing of visual-spatial information from studies comparing native deaf signers, hearing signers, and hearing nonsigners. The area of normal sign language acquisition is in need of further exploration. With a better understanding of how sign language develops normally, we would be better able to identify disordered or delayed acquisition. The issue of bilingualism in sign language acquisition needs to be appraised more fully. Children who use ASL must become bilingual in their language of conversation (ASL) and their language of instruction which is most often English in many forms (written, signed, and spoken). Therefore, more studies should focus on bilingual acquisition. Few studies use a longitudinal design which would elucidate patterns in the development of sign language and help in the recognition of individual differences. Finally, as technology becomes smaller, less expensive, and more readily available, the applications for those with disabilities are limitless.

See Also

- [Manual Sign](#)
- [Sign Language](#)

References and Reading

- Baynton, D. C. (1996). *Forbidden signs: American culture and the campaign against sign language*. Chicago: The University of Chicago Press.
- Bellugi, U., & Fischer, S. (1972). A comparison of sign language and spoken language. *Cognition*, 1, 173–200.
- Corina, D. P. (1998). Aphasia in users of signed languages. In P. Coppens, Y. Lebrun, & A. Basso (Eds.), *Aphasia in atypical populations* (pp. 261–309). Mahwah: Lawrence Erlbaum.

- Dipietro, L., Sabatini, A. M., & Dario, P. (2008). A survey of glove-based systems and their applications. *IEEE Transactions on Systems, Man, and Cybernetics, Part C: Applications and Reviews*, 38, 461–482.
- Fels, S., & Hinton, G. (1993). Glove-Talk: A neural network interface between a data-glove and a speech synthesizer. *IEEE Transactions on Neural Networks*, 3, 2–8.
- Hernandez-Rebollar, J., Kyriakopoulos, N., & Linderman, R. (2004). A new instrumented approach for translating American Sign Language into sound and text. In *Proceedings of the Sixth IEEE International Conference on Automated Face and Gesture Recognition (FGR '04)* (pp. 547–552). New York: Association for Computing Machinery.
- Hickok, G., Bellugi, U., & Klima, E. S. (1998). The neural organization of language: Evidence from sign language aphasia. *Trends in Cognitive Sciences*, 2, 129–136.
- Lane, H., Hoffmeister, R., & Bahan, B. (1996). *Journey into the deaf-world*. New York: Random House.
- Mitchell, R. E., & Karchmer, M. A. (2004). Chasing the mythical ten percent: Parental hearing status of deaf and hard of hearing children in the United States. *Sign Language Studies*, 4, 138–163.
- Murthy, G., & Jadon, R. (2009). A review of vision based hand gestures recognition. *International Journal of Information Technology and Knowledge Management*, 2, 405–410.
- Neidel, C., Kegl, J., MacLaughlin, C., Bahan, B., & Lee, R. G. (2000). *The syntax of American Sign Language*. Cambridge: The MIT Press.
- Padden, C. (1980). The deaf community and the culture of deaf people. In C. Baker & R. Battison (Eds.), *Sign language and the deaf community: Essays in honor of William Stokoe*. Silver Spring: National Association of the Deaf.
- Poizner, H., Klima, E., & Bellugi, U. (1987). *What the hands reveal about the brain*. Cambridge: The MIT Press.
- Sandler, W., & Lillo-Martin, D. (2006). *Sign language and linguistic universals*. Cambridge: Cambridge University Press.
- Starner, T., & Pentland, A. (1998). Real-time American Sign Language recognition using desk and wearable computer based video. *IEEE Transactions on Pattern Analysis and Machine Intelligence*, 20, 1371–1375.
- Starner, T., Weaver, J., & Pentland, A. (1997). A wearable computer based American Sign Language recognizer. In *First International Symposium on Wearable Computing*. Cambridge: IEEE Computer Society.
- Stokoe, W. C. (1960). *Sign language structure: An outline of the communication systems of the American deaf* (Studies in Linguistics Occasional Papers 8). Buffalo: Department of Anthropology and Linguistics, University of Buffalo.
- Valli, C., Lucas, C., & Mulrooney, K. J. (2005). *Linguistics of American Sign Language* (4th ed.). Washington, DC: Clerc Books.
- Vermeerbergen, M., & Demey, E. (2007). Comparing aspects of simultaneity in Flemish sign language to instances of concurrent speech and gesture. In M. Vermeerbergen, L. Leeson, & O. Crasborn (Eds.), *Simultaneity in sign languages: Form and function*. Philadelphia: John Benjamins.
- Wang, Q., Chen, X., Zhang, L., Wang, C., & Gao, W. (2007). Viewpoint invariant sign language recognition. *Computer Vision and Image Understanding*, 108, 87–97.
- Woll, B., & Sharma, S. (2008). Sign language and English: How the brain processes languages in different modalities. In C. Bidoli & E. Ochse (Eds.), *English in international deaf communication*. Bern: Lang.

American Speech-Language-Hearing Association Functional Assessment of Communication Skills

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Synonyms

[ASHA FACS](#)

Description

The *American Speech-Language-Hearing Association Functional Assessment of Communication Skills* (ASHA FACS) measures and provides tools to monitor the functional communication of adults with certain speech, language, and cognitive impairments. Functional communication is the ability to effectively and independently communicate by sending or receiving messages, whether the individual uses speech, sign, pictures, or a speech-generating machine to convey the message.

Historical Background

This test was first published in 1995 to measure the ability of adults with left-hemisphere stroke and traumatic brain injury to execute their daily communication tasks. An addendum to this test

was published in 2004 that included normative data from individuals with right-hemisphere stroke, progressive neurological disease, and Alzheimer's disease and related dementias but not adults with communication deficits related to autism spectrum disorder (ASD). The extended validation of the test was also designed to support the use of this measure with multicultural populations in the United States and English-speaking populations internationally. The 2017 revised edition of the test includes the same test items and scoring procedures and an updated literature overview and presentation of validation studies in the manual.

Psychometric Data

Comparison data for the *American Speech-Language-Hearing Association Functional Assessment of Communication Skills* (ASHA FACS) test is based on the performance of individuals with aphasia, following left-hemisphere stroke, traumatic brain injury, dementia, and right-hemisphere stroke. While it may not be appropriate to use this data to evaluate the performance of adults with ASD, the test might be used informally to identify specific behaviors important to developing effective functional communication in adults with ASD. An individual's ability to perform the activities outlined on the ASHA FACS should allow the clinician to examine patterns of social communication and the comprehension and use of oral and written language.

Clinical Uses

The *American Speech-Language-Hearing Association Functional Assessment of Communication Skills* (ASHA FACS) is suggested for use with adults with speech, language, and cognitive difficulties following a stroke or traumatic brain injury or in the presence of progressive neurological disease, Alzheimer's disease, and related dementias. The assessment looks at the following areas: social communication, communication of needs,

daily planning, and reading, writing, and number concepts. Although not specifically designed or normed for the ASD population, the measure could be used informally to look at the daily communication abilities of adults and adolescents in this population.

See Also

- ▶ [Augmentative and Alternative Communication \(AAC\) Device](#)
- ▶ [Communicative Functions](#)
- ▶ [Functional Communication Training](#)
- ▶ [Pragmatics](#)
- ▶ [Social Communication](#)

References and Reading

- Adams, B. C. (2009). *The language activities of daily living series* (Sterling ed.). Winooski: Laureate. http://www.laureatelearningsystems.net/pdfs/laureate_ladl_monograph_09.pdf. Retrieved 8 Aug 2012.
- Fratalli, C. M., Holland, A. L., Thompson, C. K., Wohl, C., & Ferketic, M. (2003). *Functional assessment of communication skills for adults*. Rockville: American Speech-Language-Hearing Association.
- Kleinman, L. I. (2003). *Functional communication profile-revised*. East Moline: LinguSystems.
- Light, J. C., Roberts, B., Dimarco, R., & Greiner, N. (1998). Augmentative and alternative communication to support receptive and expressive communication for people with autism. *Journal of Communication Disorders*, 31(2), 153–180.
- Martos Perez, J., & Fortea Sevilla, M. S. (1993). Psychological assessment of adolescents and adults with autism. *Journal of Autism and Developmental Disorders*, 23(4), 653–664.
- Matson, J. L., Rivet, T. T., Fodstad, J. C., Dempsey, T., & Boisjoli, J. A. (2009). Examination of adaptive behavior differences in adults with autism spectrum disorders and intellectual disability. *Research in Developmental Disabilities*, 30(6), 1317–1325.
- Mesibov, G. B., Schopler, E., & Caison, W. (1989). The adolescent and adult psychoeducational profile: Assessment of adolescents and adults with severe developmental handicaps. *Journal of Autism and Developmental Disorders*, 19(1), 33–40.
- Mirenda, P. (2001). Autism, augmentative communication, and assistance technology. *Focus on Autism and Other Developmental Disabilities*, 16(3), 141–151.
- Mirenda, P. (2008). Toward functional augmentative and alternative communication for students with autism:

- Manual signs, graphic symbols, and voice output communication aids. *Language, Speech, and Hearing Services in Schools*, 34, 203–216.
- Ozonoff, S., Goodlin-Jones, B. L., & Solomon, M. (2005). Evidence-based assessment of autism spectrum disorders in children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, 34(3), 523–540.
- Palmen, A., Didden, R., et al. (2012). A systematic review of behavioral intervention research on adaptive skill building in high-functioning young adults with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 6(2), 602–617. <https://doi.org/10.1016/j.rasd.2011.10.001>.
- Persson, B. (2000). Brief report: A longitudinal study of quality of life among adult men with autism. *Journal of Autism and Developmental Disorders*, 30(1), 61–66. <https://doi.org/10.1023/A:1005464128544>.
- Pugliese, C. E., Anthony, L., Strang, J. F., Dudley, K., Wallace, G. L., & Kenworthy, L. (2015). Increasing adaptive behavior skill deficits from childhood to adolescence in autism spectrum disorder: Role of executive function. *Journal of Autism and Developmental Disorders*, 45(6), 1579–1587. <https://doi.org/10.1007/s10803-014-2309-1>.
- Sparrow, S. S., Cicchetti, D., & Balla, D. A. (2016). *Vineland adaptive behavior scales* (3rd ed. manual). Bloomington: NCS Pearson.
- Van Bourgondien, M. E., Reichle, N. C., & Schopler, E. (2003). Effects of a model treatment approach on adults with autism. *Journal of Autism and Developmental Disorders*, 33(2), 131–140. <https://doi.org/10.1023/A:1022931224934>.

Americans with Disabilities Act

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Definition

The *Americans with Disabilities Act* (ADA) is a landmark piece of Federal civil rights legislation which provides equal rights for people with disabilities throughout the United States. The ADA provided civil rights protections for people with disabilities in all programs funded by federal, state, and local governments. It prohibited discrimination on the basis of disability in the areas of employment, state and local government, public accommodations, transportation, telecommunications, and

commercial facilities. In 2008, the ADA was updated with the passage of the *ADA Amendments Act of 2008*, again expanding the coverage of civil rights protections for people with disabilities.

Areas Covered by the ADA

Employment

The ADA stipulates that employers are not allowed to inquire about whether a person has a disability, or the nature or severity of such disability, during the hiring or application process. The ADA also required certain employers (such as employment agencies, labor organizations, and joint labor-management committees) to provide “reasonable accommodations” to qualified individuals with a disability, unless it would impose extreme hardship on the employer. Reasonable accommodations include making existing facilities accessible to people with disabilities, changing work duties (including job restructuring, part-time or modified work schedules, reassignment to a vacant position) and also the provision of equipment, devices, or interpreters to enable a qualified person with a disability to perform the role. Determinations of extreme hardship take into account the size of the firm and the nature and cost of the reasonable accommodation, among other things.

Public Entities and Public Transportation

The ADA also prohibits public entities (such as state or local governments and federal transportation organizations) from engaging in discrimination against people with disabilities in their programs and services. This aspect of the law also requires public entities to provide paratransit and other special transportation services for individuals with disabilities, including making provisions for wheelchair users in public transport.

Public Accommodations and Services Operated by Private Entities

The ADA also prohibits discrimination against people with disabilities in terms of receiving goods, services, facilities, privileges, advantages,

or accommodations from “any place of public accommodation.” The term “public accommodation” is defined very broadly to include such places as hotels and motels; restaurants and bars; movie cinemas and theaters; convention centers and auditoriums; stores that sell food, clothing, or hardware; laundromats; travel centers; banks; pharmacies; parks and zoos; educational institutions (from nursery school to university); day care centers and senior centers; and gyms and health spas. However, religious institutions are not included in the ADA. Any new construction of such facilities must conform to the requirements of the ADA and be accessible to all users.

Telecommunications

Telecommunications carriers were required under the ADA to provide telecommunications relay services such as Teletype Writers and other telecommunications devices, particularly for people who are deaf or who have speech impairments.

Landmark ADA Cases

The ADA has been elaborated and refined under case law – in other words, courts have made rulings about the areas covered under the law over time. Some of the important cases which have affected the way the ADA is interpreted include *Bragdon v. Abbott*, 524 U.S. 624 (1998) which found that people with HIV were included in the ADA; *Sutton v. United Air Lines Inc.*, 119 S. Ct. 2139 (1999) which found that when deciding whether an individual is disabled, courts should consider measures that mitigate the individual’s impairment, such as eyeglasses and contact lenses; *Board of Trustees of University of Alabama v. Garrett*, 531 U.S. 356 (2001), which bars private money damages actions for state violations of employment discrimination against people with disabilities; *Barden v. The City of Sacramento* 292F.3d 1073, 1076 (9th Cir. 2002), which ruled that local governments must make sidewalks accessible when they made street improvements; and *Toyota Motor Manufacturing, Kentucky, Inc. v. Williams*, 534 U.S. 184 (2002), which narrowed the definition of disability by

ruling that an individual must prove they are prevented from performing major life activities in daily life (and not just workplace issues associated with their impairment) before they are considered “disabled” under the ADA.

Historical Background

The Rehabilitation Act of 1973

In the early 1970s, the disability rights movement, inspired by civil rights movement, had increasingly defined itself as a minority which was experiencing widespread discrimination. Its advocates played a key role in developing the legislative precursor to the ADA – the *Rehabilitation Act of 1973*. This Act was the first national piece of civil rights and antidiscrimination legislation for people with disabilities. Section 504 of the *Rehabilitation Act of 1973* which stated that “no qualified individual with a disability in the United States shall be excluded from, denied the benefits of, or be subjected to discrimination under” any program receiving federal funding – specifically the Federal Government, federal contractors, and recipients of federal financial assistance.

Section 504 of the *Rehabilitation Act of 1973* was historic for a number of reasons, including the fact that it recognized that people with disabilities were “a class” who experienced inferior treatment and discrimination because of a widespread pattern of discrimination and prejudice. From this viewpoint, people with disabilities could legitimately be considered a “minority group” – indeed, some activists called it “the biggest minority group in the country” because they estimated 20% of the entire population had a disability. Section 504 also involved treating people with different disabilities as members of the same minority group, replacing a long history of legislation aimed at specific groups of people with disabilities (such as veterans with disabilities, blind people, deaf people, and so on).

For 4 years, the disability rights movement engaged in continuous advocacy over the regulations which would be used to enforce Section 504. They argued that the regulations must require actions that would remove physical and

communicational barriers, as well as providing accommodations. Throughout the USA, disability activists engaged in “sit ins” – the longest of which occurred in San Francisco, lasting 28 days. The final regulations did meet the demands of these disability activists.

In the early 1980s, under the leadership of President Reagan, a task force was established to remove legislation which was excessively burdensome on business. Section 504 was identified as a potential burden for business, but the disability movement waged a 2-year campaign in defense of the legislation, and they were again successful. The regulations stayed in place.

Americans with Disabilities Act of 1990 (P.L. 101-336)

President George H.W. Bush signed into law the Americans with Disabilities Act of 1990 (P.L. 101-336). This was hailed as a major piece of civil rights legislation for people with disabilities. Whereas Section 504 of the Rehabilitation Act prohibited discrimination against individuals on the basis of disability in public entities, and services that received federal funding, the ADA extended the prohibition to private companies as well. Employers were prohibited from engaging in discrimination in every phase of employment: from recruitment and hiring to evaluation and promotion (Wehman 2001). Employers were again prohibited from discriminating against “otherwise qualified” individuals with a disability. The term “otherwise qualified” being a specific legal term. The employer who had an employee or job candidate who was “otherwise qualified” had to make “reasonable accommodations” in the workplace so that the individual could successfully perform his or her job. The scope of this piece of legislation was profound. According to Wehman (2001), this was a considerable challenge to 660,000 private businesses at the time that employed 8.6 million people. In fact, the law set up a timeline by which companies of various sizes had to insure their compliance with the ADA. By 1994, companies with 15 employees or more had to insure their compliance with ADA. Most major companies now employ at least one individual

whose job is to insure compliance with the law. The penalties for noncompliance are similar to those where a company is found guilty of discriminating against a person based upon gender or race. Government agencies are expected to comply with the law and face the same penalties as well.

Current Knowledge

Current knowledge about civil rights legislation for people with disabilities, and the ADA in particular, relies on an updated version of the Act, namely, the ADA Amendments Act of 2008. The central idea behind the ADA – that discrimination against people with disabilities was unlawful – is maintained in this Amendment, but other changes significantly alter the nature of disability rights in the USA.

Under the ADA Amendments Act of 2008, which came into effect on January 1, 2009, the US Congress reversed a series of court rulings which they viewed as limiting the rights of persons with disabilities. The Act specifically criticizes the findings of the judicial system in two of the cases discussed above (*Sutton v. United Air Lines* and *Toyota v. Williams*) for moving away from the initial intent of the ADA, which was to provide a broad-scale remedy to discrimination for people with disabilities.

The *ADA Amendments Act of 2008* also expanded the scope of those covered under the law: it applies not only to programs receiving local, state or federal funding, but also to all private employers with 15 or more employees, as well as businesses with fewer than 15 employees, if they are considered “places of public accommodation.” Such “places of public accommodation” include hotels, educational institutions, care providers, recreation facilities, transportation providers, and restaurants.

While the ADA was marked by conflict between the business community and disability advocates, the *ADA Amendments Act of 2008* broke such patterns of conflict, in some ways, because both business and disability advocates

agreed on a compromise which they unilaterally supported in testimonies to Congress. This was an interesting compromise because business representatives had criticized some disability activists for being “professional plaintiffs” who sought to earn an income by being overly litigious.

New Disability Definition

This Act clarified the intent of Congress to provide a broad definition of “major life activities” which might be affected by a person’s disability. Specifically, it stated that “major life activities include, but are not limited to, caring for oneself, performing manual tasks, seeing, hearing, eating, sleeping, walking, standing, lifting, bending, speaking, breathing, learning, reading, concentrating, thinking, communicating, and working.” The phrase “major life activity” also specifically included “the operation of a major bodily function, including but not limited to, functions of the immune system, normal cell growth, digestive, bowel, bladder, neurological, brain, respiratory, circulatory, endocrine, and reproductive functions.” As well, the Act overrode the findings in the *Sutton* case that “an impairment that substantially limits one major life activity need not limit other major life activities in order to be considered a disability.” Furthermore, the Act stated that defining disability should not be continuously reduced through a series of restrictive court decisions; instead, it states that the definition “shall be construed in favor of broad coverage of individuals under this Act, to the maximum extent permitted. . .”

The Act also stated that determining whether an impairment substantially limits a major life activity must be made without considering various mitigating measures such as medication, equipment, low-vision devices, prosthetics, hearing aids or cochlear implants, oxygen therapy equipment, or assistive technology. However, the Act states that the use of ordinary eyeglasses should be included in determining whether someone has an impairment that limits a major life activity. It also states that the ADA regulations

developed by the Equal Employment Opportunity Commission were inconsistent with congressional intent, relying on an excessively narrow definition of disability.

The Act does not apply retrospectively; it only applies after January 1, 2009.

Future Directions

Future cases will test the redefinition of “disability” through the court system. Congress has indicated that it wanted a more inclusive definition of disability, but how that actually plays out in specific cases and with specific disabilities (and degrees of disability) is yet to be determined. Additionally, upcoming cases will explore issues of compliance with the *Americans with Disabilities Act Amendments of 2008*.

Another issue which will be a major concern in the future is the degree to which the broad definition of disability within the *Americans with Disabilities Act Amendments of 2008* relates to other disability legislation such as the Individuals with Disabilities Education Act (IDEA) which potentially may result in confusion or inconsistent treatment of students in elementary and secondary schools.

See Also

- [Disability](#)
- [Rehabilitation Act of 1973](#)

References and Reading

- Feldblum, C. R., Barry, K., & Benfer, E. A. (2008). The ADA Amendments Act of 2008. *Texas Journal on Civil Liberties & Civil Rights*, 13(2), 187–240.
- Long, A. B. (2008). Introducing the new and improved Americans with Disabilities Act: Assessing the ADA Amendments Act of 2008. *Northwestern University Law Review*, 103, 217–229.
- Rozalski, M., Katsiyannis, A., Ryan, J., Collins, T., & Stewart, A. (2010). Americans with Disabilities Act amendments of 2008. *Journal of Disability Policy Studies*, 21(1), 22–28.

- Thomas, V. L., & Gostin, L. O. (2009). The Americans with Disabilities Act: Shattered aspirations and new hope. *JAMA*, 301(1), 95–97.
- Wehman, P. (2001). *Life beyond the classroom: Transition strategies for young people with disabilities* (3rd ed.). Baltimore: Paul H. Brookes Publishing.

Amino Acid Disorders

► Aminoacidopathies, Disorders of

Amino Acids

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Synonyms

[Alpha-amino acid](#)

Definition

Amino acids are molecules that contain an amine group, a carboxylic acid group, and a side chain built from carbon and hydrogen. Amino acids always include the elements carbon, nitrogen, oxygen, and hydrogen. An amino acid has the generic formula $H_2NCH(R)COOH$, where R is an organic substituent. The amino group is attached to the carbon atom immediately adjacent to the carboxylate group. Amino acids are the building blocks of proteins and important in various metabolic processes. Essential amino acids cannot be built by humans and need to be obtained through food intake.

References and Reading

- Meierhenrich, U. J. (2008). *Amino acids and the asymmetry of life*. Berlin/New York: Springer. ISBN 978-3-540-76885-2.
- Nelson, D. L., & Cox, M. M. (2008). *Lehninger principles of biochemistry* (5th ed.). New York: W.H. Freeman & Company. Hardcover. ISBN 071677108X.

Aminoacidopathies, Disorders of

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Synonyms

[Amino acid disorders](#)

Definition

Aminoacidopathies, or amino acid disorders, are the result of an individual's inability to metabolize certain amino acids in proteins and/or detoxify the resultant by-products of that amino acid metabolism. The buildup of these amino acids and/or amino acid metabolism by-products in the blood can have severe medical implications, including autism spectrum disorders. Because amino acids are the key building blocks of many neurotransmitters, they are crucial to the proper regulation and development of cognitive, social, and emotional brain functions.

Serotonin, essential to the regulation of mood, is a monoamine neurotransmitter derived from tryptophan. Artificial depletion of tryptophan in autistic adults results in an increase in repetitive, anxious, and self-injurious behaviors. Plasma levels of tryptophan are significantly lower in autistic adolescents compared to age-matched unaffected individuals. In concordance with hypotheses of excitatory/inhibitory signaling imbalances in ASD patients, higher levels of glutamic acid and aspartic acid, both major excitatory neurotransmitter precursors, have been reported in ASD patients. Additionally, these patients show increased levels of taurine, an inhibitory neurotransmitter precursor, perhaps in a compensatory effort.

Of aminoacidopathies, phenylketonuria (PKU) probably bares the greatest symptomatic resemblance to ASD if left untreated in early childhood. PKU is caused by a mutation in the hepatic enzyme phenylalanine hydroxylase (PAH) gene, rendering the protein product nonfunctional. The PAH enzyme is responsible for the metabolism of the amino acid phenylalanine into tyrosine. Classic symptoms of PKU include mental retardation, behavioral or social problems, and seizures, all of which are also found in subsets of ASD patients.

Very recently there has been interest in homocysteine, a nonprotein amino acid not derived from diet. Vitamin B6, B9, and B12 deficiencies result in high levels of homocysteine, a neurotoxic state capable of inducing neuronal damage and cell loss through excitotoxicity and apoptosis. High levels of this amino acid have been found in autism patients.

Aminoacidopathies are often autosomal recessive and can be detected in urine samples for some amino acids and dried blood spot samples for others. Treatment usually involves dietary restrictions.

References and Reading

- Croonenberghs, J., Delmeire, L., Verkerk, R., Lin, A. H., Meskal, A., Neels, H., . . . & Maes, M. (2000). Peripheral markers of serotonergic and noradrenergic function in post-pubertal, caucasian males with autistic disorder. *Neuropsychopharmacology*, 22(3), 275–283.
- Kałużna-Czaplińska, J., Zurawicz, E., Michalska, M., & Rynkowski, J. (2013). A focus on homocysteine in autism. *Acta Biochimica Polonica*, 60, 137.
- Lowe, T. L., Tanaka, K., Seashore, M. R., Young, J. G., & Cohen, D. J. (1980). Detection of phenylketonuria in autistic and psychotic children. *JAMA, the Journal of the American Medical Association*, 243(2), 126–128.
- McDougle, C., Naylor, S. T., Cohen, D. J., Aghajanian, G. K., Heninger, G. R., & Price, L. H. (1996). Effects of tryptophan depletion in drug-free adults with autistic disorder. *Archives of General Psychiatry*, 53(11), 993.
- Moreno-Fuenmayor, H., Borjas, L., Arrieta, A., Valera, V., & Socorro-Candanoza, L. (1996). Plasma excitatory amino acids in autism. *Investigación Clínica*, 37(2), 113–128.
- van Spronsen, F. J. (2010). Phenylketonuria: A 21st century perspective. *Nature Reviews Endocrinology*, 6(9), 509–514.
- Warren, R. P., & Singh, V. K. (1996). Elevated serotonin levels in autism: Association with the major histocompatibility complex. *Neuropsychobiology*, 34(2), 72–75.

Amitriptyline

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Synonyms

[Amitriptyline](#)

Indications

Depression, anxiety, enuresis nocturna, neuropathic pain, attention deficit/hyperactivity disorder (ADHD), obsessive-compulsive disorder (OCD).

Mechanisms of Action

Amitriptyline's key mechanism of action lies in the elevation of extracellular biogenic amine levels notably those of noradrenaline and serotonin (with noradrenaline being affected to a greater extent) by its blockade of cellular noradrenaline and serotonin reuptake transporters. In contrast, its metabolite nortriptyline has a more balanced action equally affecting both serotonin and noradrenaline levels. Amitriptyline exerts its function by blocking serotonin and noradrenaline reuptake. It is an antagonist at a number of receptors, notably serotonin 5-HT_{2A}, 5-HT_{2C}, 5-HT₃, 5-HT₆, and 5-HT₇, noradrenaline α_1 , histamine H₁, acetylcholine muscarinic receptors, and opiate σ_1 receptors.

Amitriptyline is absorbed from the gastrointestinal tract with highly varying peak plasma concentrations, occurring between 2 and 12 h after administration, where nearly all (95%) of the available amitriptyline is protein bound. Like all tricyclic antidepressants, amitriptyline is water soluble. The bioavailability of the active drug is between 30% and 60%, due to extensive first pass metabolism of the drug in the liver by the CYP2D6 enzyme.

The elimination half-life varies from 10 to 50 h, with an average of 15 h. Within 24 h, approximately 25–50% of a dose of amitriptyline is excreted in the urine as inactive metabolites; small amounts are excreted in the bile. Amitriptyline is demethylated in the liver to its primary active metabolite, nortriptyline by CYP450 1A2. Circulating therapeutic plasma levels typically lie between 110 and 250 ng/ml.

Specific Compounds and Properties

Amitriptyline is known by several brand names including Elavil, Laroxyl, Lentizol, and Sarotex. A generic version – Tryptizol – is also available. In terms of formulation, amitriptyline is available in solid tablet forms with intramuscular formulations available also. Typically, tablets are available in 50 and 100 mg doses.

Clinical Use (Including Side Effects)

Amitriptyline, whether administered orally or via intramuscular formulation, reaches peak plasma concentrations in both cases approximately 2–12 h after administration with onset commencing after 45 min. As already mentioned, the primary indication for amitriptyline use is as an antidepressant, but it also sometimes used for its sedative action. Therapeutic antidepressant effects are only seen after 2–3 weeks of therapy. In terms of child and adolescent usage, TCAs in general have been employed for more than 40 years and have been indicated not only in depression but also in attention deficit/hyperactivity disorder (ADHD), obsessive-compulsive disorder (OCD), separation anxiety, and nocturnal enuresis. In terms of its antidepressant action in children, this is mixed with its active metabolite nortriptyline showing more favorable effects. Other TCAs including desipramine and clomipramine have shown efficacy in ADHD and OCD, and as such, amitriptyline's usage in these disorders is off-label. In terms of the utility of TCAs in treating separation anxiety, this appears to be modest at best with further improvement seen when TCAs are combined with behavioral therapy.

Amitriptyline is metabolized primarily in the liver and is excreted both in feces and urine. In terms of its half-life, amitriptyline is typically associated with a half-life of between 31 and 36 h dependent on formulation and other factors. In reality, the half-life varies dependent on amitriptyline use and indication with administration for depression usually resorting to formulations with a half-life of 9–25 h, while the treatment of nocturnal enuresis (in children) is associated with dosages/formulations of between 18 and 96 h – where the active metabolite of amitriptyline, nortriptyline, plays a prominent role.

Typically, tricyclic antidepressants are not successful in treating prepubertal depression showing marginally better efficacy in adolescents for this indication. As such, the pharmacological treatment of choice for this indication remains selective serotonin reuptake inhibitors. In those child and adolescent subjects who do not respond to SSRIs, treatment of depression with tricyclic antidepressants (such as amitriptyline) has not been shown to be very productive in clinical studies. Due to their higher rate of metabolism, younger subjects often require higher mg/kg doses when compared to adults if amitriptyline use is warranted in depression. Special attention should be directed toward the higher rate of cardiotoxicity with amitriptyline in young subjects compared to adults, and this should be carefully monitored by both baseline and on-treatment ECG. Coprescribing with medications that prolong the QTc interval is not advised.

Dependent on the indication, dosing schedules of amitriptyline vary. For depression, it is advised to start with a dose of 25–100 mg/day. The dose can be increased up to 300 mg/day, although 100–150 mg per day is the recommended dosage. In elderly subjects, doses for the treatment of depression are typically half or one-third of the recommended adult dose, while its use for childhood depression is not indicated.

In the case of nocturnal enuresis, recommended doses typically range between 1 and 1.5 mg/kg of the body weight per day with doses to be taken in the afternoon. In children between 5 and 10 years old, these doses are usually in the range of

10–25 mg, while for adolescents between 11 and 16 years, these doses are typically 25–50 mg.

The use of amitriptyline against pain typically involves starting doses of 25 mg daily which can be increased up to 100 mg daily with 75 mg daily representing the active clinical dose in most patients. The benefits of amitriptyline treatment against pain are usually seen between 1 and 7 days after treatment onset.

The efficacy of treatment with amitriptyline can be improved and the onset of therapeutic effect hastened by measuring plasma levels to accurately titrate the therapeutic doses required. By monitoring drug compliance, amitriptyline dosing can be optimized. It is important that dosing has been stable for about 1 week prior to the assessment of blood samples with blood drawn between 10 and 14 h after the last intake advised for accurate monitoring. When monitoring plasma levels of amitriptyline, it is advisable to also measure its metabolite nortriptyline as nortriptyline is an active metabolite. Typically, therapeutic plasma levels of amitriptyline lie in the range of 50–200 µg/l, while those for nortriptyline usually are between 100 and 300 µg/l. With regard to drug safety, combined amitriptyline and nortriptyline concentrations of 500 µg/l are toxic.

Due to the high degree of plasma protein binding associated with amitriptyline, patients presenting with renal disorders often demonstrate altered plasma levels of amitriptyline and require careful dose monitoring.

Contraindications: Administration of amitriptyline during the recovery phase of cardiac infarction and glaucoma is highly contraindicated. Administration of amitriptyline in patients with epilepsy, organic brain damage, urine retention, prostate hyperplasia, pyloric stenosis, cardiovascular disease, hyperthyroidism, and diminished liver and kidney function is not advised but is not expressly contraindicated.

Interaction with Other Drugs: All selective serotonin reuptake inhibitors (SSRIs) such as fluvoxamine, with the exception of citalopram, may increase amitriptyline concentrations due to inhibition of the cytochrome CYP 2502D6. Other medications that can cause increased plasma levels are fluvoxamine, cimetidine, haloperidol, cimetidine,

and phenothiazines. Amitriptyline may decrease the effect of antihypertensive medication particularly guanethidine and clonidine, while coadministration with monoamine oxidase inhibitors may even induce a hypertensive crisis and demonstrate atropine-like toxic effects. Coadministration with phenothiazines may increase serum amitriptyline (or any other TCA for that matter) levels, while the effect of amitriptyline (or another TCA) is potentiated in the presence of thyroid preparations. Care should also be exercised requiring careful ECG monitoring when coadministering with thyroid preparations as these together can induce tachycardia and cardiac arrhythmia. For those taking oral contraceptives, these can inhibit the metabolism of TCAs including amitriptyline.

Amitriptyline is associated with strong additive anticholinergic effects when given in combination with anticholinergic agents. This additive action is also seen with CNS depressant ligands causing enhanced depressant effects or even severe cardiac effects such as heart block when combined with quinidine. Potentiation of sympathomimetic effects is also possible when amitriptyline is given in combination with sympathomimetics such as adrenaline.

Side Effects: Side effects associated with amitriptyline use include sedation, anhydrosis (decreased sweating), increased appetite, ataxia, anxiety, blurred vision, glaucoma, dry mouth, mydriasis (oversensitivity to light), headache, heartburn, decreased lacrimation, constipation, orthostatic hypotension, restlessness, sedation, sexual dysfunction (impotence, decreased libido), and urinary hesitancy and retention.

The management of some of the minor side effects is relatively straightforward, e.g., dry mouth can be managed by dry candy or mouth rinsing, mydriasis with sunglasses, orthostatic hypotension with slow positional changes, and decreased lacrimation with artificial tears.

Severe/life-threatening side effects associated with amitriptyline are rare events. However, these severe adverse events can include tachycardia, arrhythmias, extrapyramidal symptoms, glaucoma, hepatic failure, hyperthermia, suicidal ideations, mania, orthostatic hypotension, paralytic ileus, QTc prolongation, and seizures.

Precautions Associated with Amitriptyline

Use: Prior to starting treatment, it is advisable to take a medical history of cardiac problems, glaucoma, and seizures. Due to weight gain associated with amitriptyline use, it is important to measure weight, length, and BMI.

During treatment, it is important to address suicidal ideations, manic symptoms, and side unpleasant effects that might influence therapeutic compliance (e.g., sexual dysfunction). Addressing suicidal ideations is important since amitriptyline can increase suicidal ideations, and fatal overdose does not require a huge increase in dosage. It is therefore also important to be aware of potential hoarding of amitriptyline by patients which can be checked by careful monitoring of drug compliance in blood samples.

Overdosing: Individuals with diminished liver functions, plasma protein count/activity, and decreased total body water are at greater risk for overdose since metabolism occurs in the liver and usually nearly all ingested amitriptyline is bound to plasma proteins. Symptoms of overdose include apathy, coma, convulsions, cardiac arrhythmias, hypotension, and ultimately death.

See Also

► [Elavil \(Amitriptyline\)](#)

References and Reading

- Barbui, C., & Hotopf, M. (2001). Amitriptyline v. the rest: Still the leading antidepressant after 40 years of randomised controlled trials. *British Journal of Psychiatry*, 178, 129–144.
- Guaiana, G., Barbui, C., & Hotopf, M. (2007). Amitriptyline for depression. *Cochrane Database of Systematic Reviews*, 3, CD004186.
- Johns, M. W. (1975). Sleepy and hypnotic drugs. *Drugs*, 9, 448–478.

Amitriptyline Hydrochloride

► [Endep \(Amitriptyline\)](#)

Amitriptyline

► [Amitriptyline](#)

Amphetamine

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Indications

ADHD

Mechanisms of Action

Amphetamines are stimulant medications that enhance the release and block the reuptake of dopamine in the brain. Taking at large doses, this mechanism of action can produce euphoria and increased energy. Because of these effects, amphetamines are subject to abuse. In doses used to treat attention deficit hyperactivity disorder, however, these stimulant effects are not usually present. The enhanced release of dopamine is presumed to be the source of improved attention and decreased activity.

Clinical Use (Including Side Effects)

Amphetamines are associated with many adverse effects that are relevant to children and adults with autism. For example, they can cause insomnia, decreased appetite, increased stereotypic behavior, and irritability. To date, the amphetamines have only been evaluated in small studies in children with autism with equivocal results. It appears

that the potency of the amphetamine compounds makes it difficult to find a dose that is helpful, but not associated with dose-limiting adverse effects.

See Also

► [Attention Deficit/Hyperactivity Disorder](#)

References and Reading

Martin, A., Scahil, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.

Amygdala

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Synonyms

[Amygdaloid complex](#)

Structure

The amygdala is an almond-shaped structure located in the medial temporal lobe bilaterally, comprised of at least 13 nuclei in primates (Amaral et al. 1992; LeDoux 2007). It is a relatively small structure (considerably smaller than the hippocampus, which lies immediately anterior to), with approximately 12 million neurons in total occupying a volume of 4–5 cm³ in an adult human. It is well-documented that the individual subnuclei of the amygdala have distinct functions and distinct anatomical connectivity with other brain regions (both cortically and subcortically). However, given the difficulty in delineating the subnuclei reliably in living humans, the amygdala is often discussed as a single unitary structure

(as in the remainder of this chapter). There are probabilistic atlases for segmenting the amygdala into some of its subnuclei from structural magnetic resonance imaging (MRI), and this can be used to define likely locations that would fall within those nuclei, but not to precisely distinguish their boundaries. Detailed measures of the subnuclei, including stereological counts of the number of neurons, are however possible using postmortem brain tissue.

The largest subnucleus of the amygdala is the lateral nucleus, which receives most of the sensory inputs to the amygdala. In particular, the lateral nucleus receives projections from high-level visual cortex in the temporal lobe, as well as from many other polymodal association cortices. The basal nucleus of the amygdala, often lumped together with the lateral nucleus into the basolateral nucleus, contains neurons that project back to those regions from which the amygdala receives inputs. In primates, the basal nucleus also projects back to all of visual cortex, including early visual cortices (Freese and Amaral 2005). The medial nucleus likely serves an important role in other mammals as it is closely connected with the olfactory system. The central nucleus of the amygdala contains neurons that project to the hypothalamus and brainstem and regulate emotional responses. The internal circuitry of the amygdala is quite complex and is now being unraveled in great detail using optogenetic methods in mice.

Function

The amygdala's function is extremely diverse, reflected in the wide web of anatomical connections that it has with other brain regions. In addition to bidirectional connections with polymodal sensory cortex, it is connected with basal forebrain (modulating attention and flight versus freeze responses), hypothalamus, brainstem nuclei, periaqueductal gray (mediating various emotional behaviors), hippocampus (modulating memory consolidation), prefrontal cortex, and basal ganglia (modulating reward learning and

decision-making), among other regions. This diversity of connections permits the amygdala to modulate a large array of cognitive processes and aspects of behavior, including attention, memory, and reward learning. What ties all these varied aspects of cognition and behavior together is that the amygdala appears to serve a key role in processing those stimuli that have emotional or social value for an animal.

There are two main lines of research that document the amygdala's function in emotion and social behavior. Classic lesion studies in the 1930s suggested that the amygdala was needed for evaluating complex stimuli including social stimuli (Klüver and Bucy 1939), and subsequent lesion studies in monkeys (Emery et al. 2001; Machado and Bachevalier 2006) and humans (Adolphs et al. 1994) have verified this role. Lesions of the amygdala in monkeys produce a lack of cautionary behavior and a propensity to approach objects (including other animals and people) regardless of the context. For instance, whereas normal monkeys are very cautious in approaching novel stimuli or unfamiliar people, monkeys with amygdala lesions approach them readily without hesitation (Machado et al. 2009; Mason et al. 2006). Similarly, humans with amygdala lesions appear not to have a sense of personal space and show abnormally increased approach behaviors and ratings of trustworthiness and approachability of other people (Adolphs et al. 1998; Kennedy et al. 2009). This aspect of amygdala function has been investigated in humans most commonly by showing participants pictures of people and asking them to rate how much they would like to approach that person, or how much they would trust that person.

A second line of research originates primarily on work on rodents and has shown that the amygdala is necessary for learning about stimuli that predict harmful outcomes. The most studied protocol here is called Pavlovian fear conditioning, in which the animal must learn that a conditioned stimulus (such as a tone, or a particular color) predicts electric shock (Davis 2000; LeDoux 2000). Healthy animals, including humans, learn this association rapidly, whereas animals (including humans) with amygdala lesions do not. However, the amygdala is now known to

subserve a much broader role: it is also involved in appetitive learning, and it is also known to modulate declarative memory and instrumental behavior based on the value of stimuli (through projections to such structures as the hippocampus and the basal ganglia) (McGaugh 2004). Neurons recorded in the monkey amygdala show responses to stimuli that predict both aversive and appetitive outcomes, and these neurons appear to be intermingled throughout the basolateral amygdala (Paton et al. 2006). One current view of the amygdala's role in reward learning is thus that it is a primary locus for Pavlovian fear conditioning, but that it participates in other aspects of declarative and instrumental reward learning mostly through its interconnections with other brain structures.

Various attempts have been made to tie together the diverse roles of the amygdala in emotional and social processing. One view is that the amygdala, at least in humans, is somewhat specialized for aspects of social behavior or reward processing. Another view is that the amygdala carries out a much more basic and abstract computation, such as allocating processing resources to any events that are difficult to predict or novel. For instance, some human studies have argued for a fairly specialized role in recognizing social cues from facial expressions and perhaps especially from the eye region of faces (Adolphs et al. 2005). By contrast, other studies have shown broader attentional modulation based on any unpredictable stimulus, regardless of its social meaning (Herry et al. 2007; Whalen 2007). These current frameworks for understanding amygdala function are important for interpreting the amygdala's role in autism spectrum disorders, since they would point to different roles: in aspects of social dysfunction, or in sensory/attentional impairments, for instance.

Pathophysiology

Many have hypothesized that the amygdala plays a key role in the pathophysiology of autism (Bachevalier 1994; Baron-Cohen et al. 2000; Hetzler and Griffin 1981). Initially, however, there was little direct support for amygdala abnormality in autism, and much of the theory was

drawn from observing parallels between the autism phenotype and monkeys or rare humans with amygdala lesions. More recent studies have provided considerable additional evidence directly implicating the amygdala as a key region of neural dysfunction in autism, although the precise nature of the dysfunction, its etiology, and the extent of its contribution to the autism phenotype all remain intensely debated.

The first direct neural evidence to suggest that the amygdala might be abnormal in autism came from postmortem examination of brain tissue (M. Bauman and Kemper 1985), where increased cell-packing density and reduced cell size was noted. A recent follow-up study using modern quantitative methods did not replicate these findings (Schumann and Amaral 2006), likely due to methodological differences and differences in the study sample (e.g., exclusion of individuals with a history of seizures). Importantly, however, they did find significantly fewer neurons in the amygdala in the autism group.

Many studies have further examined the structure of the amygdala in autism using volumetric magnetic resonance imaging (MRI). Although this technique does not have anywhere near the spatial resolution of postmortem studies, the advantages are that it is a noninvasive technique, much larger sample sizes can be included, and one can obtain sufficient statistical power to examine clinical and behavioral correlates, as well as changes across the lifespan. Although volume is largely normal by adulthood, alterations in the early growth trajectory, growth from infancy on to late childhood, have been identified by cross-sectional and longitudinal studies, as well as cross-study comparisons (Mosconi et al. 2009; Schumann et al. 2004, 2009; Sparks et al. 2002). Specifically, the amygdala is enlarged early in development (before 2 years), but growth subsequently slows down and eventually converges with typical volumes by adolescence. In other words, the amygdala in autism undergoes an altered growth trajectory, wherein accelerated growth occurs early on but then gradually slows, so it is at the younger ages (and not adulthood) that the largest volumetric abnormalities can be observed. In addition, several studies have shown amygdala volumes in autism correlate with

various behavioral and clinical measures, such as social functioning, communicative development, and gaze patterns to faces (Mosconi et al. 2009; Munson et al. 2006; Nacewicz et al. 2006; Schumann et al. 2009). This altered growth trajectory, however, may not be specific to the amygdala alone, as total brain volume in autism also undergoes a similar pattern of abnormal development (Redcay and Courchesne 2005).

Studies that have examined the functioning of the amygdala in autism have also identified abnormalities. The primary methodology that is used to measure subcortical (and cortical) brain activity is functional MRI (fMRI) – a noninvasive technique that provides an indirect measure of neuronal activity based on changes in regional blood flow. fMRI provides reasonable spatial and temporal resolution, such that one can determine which stimuli or which cognitive process activates a particular 3–4 mm³ volume of brain tissue. However, subjects undergoing fMRI scanning are required to remain motionless for extended periods of time, and because of this, much of what is known about the functioning of the amygdala in autism comes from older children, adolescents, and adults and not young infants and toddlers (although this is now changing with several sites acquiring resting-state fMRI in sleeping infants). The first series of studies of amygdala functioning in autism found hypoactivation during performance of a variety of tasks, including making mental-state judgments from expressive eyes (Baron-Cohen et al. 1999), implicit processing of emotional faces (Critchley et al. 2000), and passive viewing of nonemotional faces (Pierce et al. 2001). However, these findings are by no means consistent across the literature, possibly due to differences in the tasks and stimuli used, differences in eye movements of participants, or differences in subject samples that reflect the heterogeneity of autism spectrum disorders.

Two notable studies have attempted to provide a more mechanistic account of amygdala abnormality in autism. One study found that amygdala activation in autism correlated positively with gaze to the eye region of faces (Dalton et al. 2005), consistent with other studies implicating the amygdala as involved in guiding eye movements towards eyes in faces (Adolphs et al. 2005;

Gamer and Buechel 2009). Given the well-documented behavioral abnormality that individuals with autism spend reduced time looking at the eyes in faces (Klin et al. 2002; Pelphrey et al. 2002), this may help to explain some of the above-described findings regarding amygdala hypoactivity. Another study (Kleinhans et al. 2009) found that the amygdala in autism exhibits abnormally reduced habituation overtime to repeatedly presented neutral faces, possibly pointing to a basic abnormality in habituation responses that have been found for amygdala neurons in other studies (Herry et al. 2007). The study in autism (Kleinhans et al. 2009) found that although the initial amygdala response was found to be slightly attenuated, the response remained elevated for longer than that observed in controls. The authors suggest that discrepancies across earlier studies might be explained by this altered time course of habituation.

Another promising approach to understanding amygdala dysfunction in autism is to examine the functional interaction between the amygdala and other brain regions. Individual brain regions do not function in isolation from one another, but rather comprise functional networks that exert reciprocal influences on other brain regions and other networks. So far, several studies have found that the amygdala exhibits abnormally reduced functional coupling with other brain regions, at least in the context of face processing tasks (Kleinhans et al. 2008; Rudie et al. 2012; Welchew et al. 2005). It is currently unclear, however, whether the amygdala is the primary source of this abnormality, or whether it simply reflects altered synaptic input (i.e., the abnormality is at the level of input rather than output). Many brain regions exhibit structural and functional abnormalities in autism, and these likely provide altered input to the amygdala.

It should be emphasized that although there is an abundance of evidence identifying the involvement of the amygdala in the social phenotype of autism, amygdala dysfunction is not thought to be the single cause of the autism phenotype. First, whether or not the amygdala contributes to other aspects of the phenotype (language delay, repetitive behaviors, restricted interests) is quite unclear. Second, given that many regions of the

brain are abnormal in autism, it seems likely that these brain regions also contribute to particular aspects of the autistic phenotype. Finally, complete lesions of the amygdala in both monkeys (Emery et al. 2001; Machado et al. 2009; Mason et al. 2006) and humans (Paul et al. 2010) do not result in autism and in several respects show symptoms that are the opposite of autism. There may be some more similarity in regard specifically to infant monkeys who had neonatal amygdala lesions (Bauman et al. 2008; Prather et al. 2001), further emphasizing that autism needs to be understood as emerging throughout a complex and prolonged developmental trajectory.

In sum, a convergence of evidence derived from a wide variety of experimental methods suggests that the amygdala is both structurally and functionally abnormal in autism. It is reasonable to assume that the amygdala is one component among a diverse set of brain regions that likely contribute to particular aspects of the autism phenotype. However, whether the amygdala plays a causal role in producing the core symptoms of autism or whether it is secondary in response to having autism has yet to be determined.

See Also

- [Functional Connectivity](#)
- [Functional MRI](#)

References and Reading

- Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature*, 372, 669–672.
- Adolphs, R., Tranel, D., & Damasio, A. R. (1998). The human amygdala in social judgment. *Nature*, 393, 470–474.
- Adolphs, R., Gosselin, F., Buchanan, T. W., Tranel, D., Schyns, P. G., & Damasio, A. (2005). A mechanism for impaired fear recognition after amygdala damage. *Nature*, 433, 68–72.
- Amaral, D. G., Price, J. L., Pitkanen, A., & Carmichael, S. T. (1992). Anatomical organization of the primate amygdaloid complex. In J. P. Aggleton (Ed.), *The amygdala: Neurobiological aspects of emotion, memory, and mental dysfunction* (pp. 1–66). New York: Wiley-Liss.

- Bachevalier, J. (1994). Medial temporal lobe structures and autism: A review of clinical and experimental findings. *Neuropsychologia*, 32(6), 627–648.
- Baron-Cohen, S., Ring, H. A., Wheelwright, S., Bullmore, E. T., Brammer, M. J., Simmons, A., et al. (1999). Social intelligence in the normal and autistic brain: An fMRI study. *European Journal of Neuroscience*, 11(6), 1891–1898.
- Baron-Cohen, S., Ring, H. A., Bullmore, E. T., Wheelwright, S., Ashwin, E., & Williams, S. C. (2000). The amygdala theory of autism. *Neuroscience and Biobehavioral Reviews*, 24(3), 355–364.
- Bauman, M., & Kemper, T. L. (1985). Histoanatomic observations of the brain in early infantile autism. *Neurology*, 35, 866–874.
- Bauman, M. D., Toscano, J. E., Babineau, B. A., Mason, W. A., & Amaral, D. G. (2008). Emergence of stereotypies in juvenile monkeys with neonatal amygdala or hippocampus lesions. *Behavioral Neuroscience*, 122, 1005–1015.
- Critchley, H. D., Daly, E. M., Bullmore, E. T., Williams, S. C., Van Amelsvoort, T., Robertson, D. M., et al. (2000). The functional neuroanatomy of social behaviour: Changes in cerebral blood flow when people with autistic disorder process facial expressions. *Brain*, 123, 2203–2212.
- Dalton, K. M., Nacewicz, B. M., Johnstone, T., Schaefer, H. S., Gernsbacher, M. A., Goldsmith, H. H., et al. (2005). Gaze fixation and the neural circuitry of face processing in autism. *Nature Neuroscience*, 8, 519–526.
- Davis, M. (2000). The role of the amygdala in conditioned and unconditioned fear and anxiety. In J. Aggleton (Ed.), *The amygdala: A functional analysis* (pp. 213–288). New York: Oxford University Press.
- Emery, N. J., Capitano, J. P., Mason, W. A., Machado, C. J., Mendoza, S. P., & Amaral, D. G. (2001). The effects of bilateral lesions of the amygdala on dyadic social interactions in rhesus monkeys. *Behavioral Neuroscience*, 115, 515–544.
- Freese, J. L., & Amaral, D. G. (2005). The organization of projections from the amygdala to visual cortical areas TE and V1 in the macaque monkey. *The Journal of Comparative Neurology*, 486, 295–317.
- Gamer, M., & Buechel, C. (2009). Amygdala activation predicts gaze toward fearful eyes. *Journal of Neuroscience*, 29, 9123–9126.
- Herry, C., Bach, D. R., Esposito, F., DiSalle, F., Perrig, W. J., Scheffler, K., et al. (2007). Processing of temporal unpredictability in human and animal amygdala. *Journal of Neuroscience*, 27, 5958–5966.
- Hetzler, B. E., & Griffin, J. L. (1981). Infantile autism and the temporal lobe of the brain. *Journal of Autism and Developmental Disorders*, 11(3), 317–330.
- Kennedy, D. P., Gläscher, J., Tyszka, J. M., & Adolphs, R. (2009). Personal space regulation by the human amygdala. *Nature Neuroscience*, 12, 1226–1227.
- Kleinmans, N. M., Richards, T., Sterling, L., Stegbauer, K. C., Mahurin, R., Johnson, L. C., et al. (2008). Abnormal functional connectivity in autism spectrum disorders during face processing. *Brain*, 131(Pt 4), 1000–1012.
- Kleinmans, N. M., Johnson, L. C., Richards, T., Mahurin, R., Greenon, J., Dawson, G., et al. (2009). Reduced neural habituation in the amygdala and social impairments in autism spectrum disorders. *The American Journal of Psychiatry*, 166(4), 467–475.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809–816.
- Klüver, H., & Bucy, P. C. (1939). Preliminary analysis of functions of the temporal lobes in monkeys. *Archives of Neurology and Psychiatry*, 42, 979–997.
- LeDoux, J. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience*, 23, 155–184.
- LeDoux, J. E. (2007). The amygdala. *Current Biology*, 17, R868–R874.
- Machado, C. J., & Bachevalier, J. (2006). The impact of selective amygdala, orbital frontal cortex, or hippocampal formation lesions on established social relationships in rhesus monkeys. *Behavioral Neuroscience*, 120, 761–786.
- Machado, C. J., Kazama, A. M., & Bachevalier, J. (2009). Impact of amygdala, orbital frontal, or hippocampal lesions on threat avoidance and emotional reactivity in nonhuman primates. *Emotion*, 9, 147–163.
- Mason, W. A., Capitano, J. P., Machado, C. J., Mendoza, S. P., & Amaral, D. G. (2006). Amygdalectomy and responsiveness to novelty in rhesus monkeys: Generality and individual consistency of effects. *Emotion*, 6, 73–81.
- McGaugh, J. L. (2004). The amygdala modulates the consolidation of memories of emotionally arousing experiences. *Annual Review of Neuroscience*, 27, 1–28.
- Mosconi, M. W., Cody-Hazlett, H., Poe, M. D., Gerig, G., Gimpel-Smith, R., & Piven, J. (2009). Longitudinal study of amygdala volume and joint attention in 2- to 4-year-old children with autism. *Archives of General Psychiatry*, 66(5), 509–516.
- Munson, J., Dawson, G., Abbott, R., Faja, S., Webb, S. J., Friedman, S. D., et al. (2006). Amygdalar volume and behavioral development in autism. *Archives of General Psychiatry*, 63(6), 686–693.
- Nacewicz, B. M., Dalton, K. M., Johnstone, T., Long, M. T., McAuliff, E. M., Oakes, T. R., et al. (2006). Amygdala volume and nonverbal social impairment in adolescent and adult males with autism. *Archives of General Psychiatry*, 63(12), 1417–1428.
- Paton, J. J., Belova, M. A., Morrison, S. E., & Salzman, C. D. (2006). The primate amygdala represents the positive and negative value of visual stimuli during learning. *Nature*, 439, 865–870.
- Paul, L. K., Corsello, C., Tranel, D., & Adolphs, R. (2010). Does bilateral damage to the human amygdala produce autistic symptoms? *Journal of Neurodevelopmental Disorders*, 2, 165–173.

- Pelphrey, K. A., Sasson, N. J., Reznick, J. S., Paul, G., Goldman, B. D., & Piven, J. (2002). Visual scanning of faces in autism. *Journal of Autism and Developmental Disorders*, 32, 249–261.
- Pierce, K., Muller, R. A., Ambrose, J., Allen, G., & Courchesne, E. (2001). Face processing occurs outside the fusiform “face area” in autism: Evidence from functional MRI. *Brain*, 124, 2059–2073.
- Prather, M. D., Lavenex, P., Mauldin-Jourdain, M. L., Mason, W. A., Capitanio, J. P., Mendoza, S. P., et al. (2001). Increased social fear and decreased fear of objects in monkeys with neonatal amygdala lesions. *Neuroscience*, 106, 653–658.
- Redcay, E., & Courchesne, E. (2005). When is the brain enlarged in autism? A meta-analysis of all brain size reports. *Biological Psychiatry*, 58(1), 1–9.
- Rudie, J. D., Shehzad, Z., Hernandez, L. M., Colich, N. L., Bookheimer, S. Y., Iacoboni, M., et al. (2012). Reduced functional integration and segregation of distributed neural systems underlying social and emotional information processing in autism spectrum disorders. *Cerebral Cortex*, 22(5), 1025–1037.
- Schumann, C. M., & Amaral, D. G. (2006). Stereological analysis of amygdala neuron number in autism. *Journal of Neuroscience*, 26(29), 7674–7679.
- Schumann, C. M., Hamstra, J., Goodlin-Jones, B. L., Lotspeich, L. J., Kwon, H., Buonocore, M. H., et al. (2004). The amygdala is enlarged in children but not adolescents with autism; The hippocampus is enlarged at all ages. *Journal of Neuroscience*, 24(28), 6392–6401.
- Schumann, C. M., Barnes, C. C., Lord, C., & Courchesne, E. (2009). Amygdala enlargement in toddlers with autism related to severity of social and communication impairments. *Biological Psychiatry*, 66(10), 942–949.
- Sparks, B. F., Friedman, S. D., Shaw, D. W., Aylward, E. H., Echelard, D., Artru, A. A., et al. (2002). Brain structural abnormalities in young children with autism spectrum disorder. *Neurology*, 59(2), 184–192.
- Welchew, D. E., Ashwin, C., Berkouk, K., Salvador, R., Suckling, J., Baron-Cohen, S., et al. (2005). Functional disconnectivity of the medial temporal lobe in Asperger’s syndrome. *Biological Psychiatry*, 57(9), 991–998.
- Whalen, P. J. (2007). The uncertainty of it all. *TICS*, 11, 499–500.

Amygdala-Prefrontal Network

► Neural Mechanisms of Emotional Dysregulation

Amygdaloid Complex

► Amygdala

Anafranil

► Clomipramine

Analog Condition Functional Analysis

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Synonyms

Functional analysis

Definition

A functional analysis is a controlled procedure for evaluating behavior function. The completion of an analog condition functional analysis requires the systematic manipulation of controlled environmental variables (Carr and Durand 1985; Iwata et al. 1982/1994). These manipulations are completed, in order, to precisely identify the maintaining variable, or variables, associated with a specific target behavior (e.g., aggression, stereotypy). By executing a series of analog conditions, investigators are able to track relevant reinforcement contingencies and formulate a functional hypothesis of maintaining variables (i.e., situations/factors associated with the problem behavior). These contingencies are considered representative of those occurring, in an integrated fashion, within the natural environment. An operational definition of a target behavior is constructed so that all investigators can reliably observe the occurrence of the response. This definition is then associated with a data collection procedure that is applied throughout all phases of the analysis. The conditions incorporated within the analysis must also be comprehensively defined for identical implementation through the assessment. Common conditions are

typically based on those outlined by Iwata et al. and include escape, attention, control, and alone. Additionally, a tangible condition is often incorporated within the analysis. Each condition is set up in a specific fashion based on certain environmental factors, and there are specific protocols for how the experimenter is to respond when the target behavior occurs. During the control condition, the patient is provided with continual access to high-preference materials, no demands, and high levels of attention. There are no environmental responses to the target behavior. During the alone condition, the patient is observed in a room without any preferred materials, demands, or attention. Again, there are no environmental responses to the target behavior. The demand condition requires continual presentation of specific demands to the patient. Upon an occurrence of the target behavior, the demands are removed for a defined interval, thereby offering negative reinforcement for the target behavior. The attention condition incorporates an introductory period of high-preference attention, which is then removed. The investigator, upon removing attention, remains close by but engaged in other activities. Following the occurrence of the target behavior, the patient is provided with a period of attention, thus offering positive reinforcement of the challenging behavior. The tangible condition allows the patient an introductory period of access to high-preference materials. These are then removed by the investigator but remain in view. The patient is provided defined periods of access to the materials, contingent on the occurrence of the target behavior, thereby establishing a positive reinforcement contingency for the target behavior. The conditions are randomly implemented and replicated until a reliable trend emerges. Defined, individual-specific, modifications to analog conditions are incorporated based upon the needs of the patient and are subject to equal requirements for reliable and replicated implementation.

See Also

- [Functional Analysis](#)
- [Functional Behavior Assessment](#)

References and Reading

- Carr, E. G., & Durand, M. V. (1985). Reducing behavior problems through functional communication training. *Journal of Applied Behavior Analysis*, 18, 111–126.
- Iwata, B. A., Dorsey, M. F., Slifer, K. J., Bauman, K. E., & Richman, G. S. (1994). Toward a functional analysis of self-injury. *Journal of Applied Behavior Analysis*, 27, 197–209. (Reprinted from *Analysis and Intervention in Developmental Disabilities*, 2, 3–20, 1982). 27, 215–240.

Analysis of Verbal Behavior (AVB)

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Definition

In his book *Verbal Behavior* (1957), B. F. Skinner defined verbal behavior as “behavior reinforced through the mediation of other persons” (p. 2). Following this definition, filling a glass with water results in a filled glass and is not verbal behavior, whereas saying, “Can you fill my glass?” depends on the behavior of another person to *mediate* the consequence of the request to fill the glass. Because verbal behavior does not act on the environment directly but rather through the behavior of others, it requires a separate analysis. Nonetheless, Skinner asserts that the same behavioral principles of reinforcement, punishment, and discrimination can account for verbal behavior (i.e., communication) as they do for any other behavior.

Historical Background

In 1934, Alfred North Whitehead challenged Skinner to use behavioral principles to account for language. Despite Whitehead’s cynicism, Skinner began his book *Verbal Behavior* (1957), which took him over 20 years to complete. Shortly

after its publication, Noam Chomsky, who had his own account of language, published a critical review of *Verbal Behavior* and behaviorism. Chomsky's criticisms were not surprising because Skinner's analysis differed significantly from the popular linguist perspective in two important ways. First, the analysis of verbal behavior involved considering units of language based on their function instead of their structure. Second, the analysis of verbal behavior proposed that language is learned behavior, which is shaped and maintained by environmental variables.

Despite the attention this debate attracted, Skinner never responded to Chomsky's review supposing that Chomsky misunderstood the philosophical foundations of behaviorism. In the years since, many have interpreted Skinner's silence as a loss. Although proponents of the analysis of verbal behavior dispute this assumption, it may have impeded widespread adoption of Skinner's analysis of verbal behavior. The linguistic theories that bind language development to physiological processes have flourished despite criticisms about their limited value for language intervention and treatment. However, a theory of language that leads to useful and effective treatments is important, especially for individuals with autism.

In the late 1980s, Ivar Lovaas developed and evaluated a discrete trial training (DTT) model for teaching children with autism. Although based on operant conditioning and behavioral principles, DTT does not align perfectly with Skinner's analysis of verbal behavior. There are two primary differences between a DTT approach to teaching children with autism and an approach based on Skinner's *Verbal Behavior*. First, although some instruction occurs in structured settings, verbal behavior interventions emphasize the importance of natural environment teaching (NET) and make use of naturally occurring learning opportunities. In the Lovaas approach, children are primarily taught in highly structured learning environments. Second, the analysis of verbal behavior suggests that, in addition to antecedents and consequences, motivating variables are crucial in the development of language. Understanding the motivating conditions for the basic verbal behaviors influences the type of antecedents and consequences

used (or captured) for training. In contrast, the Lovaas approach de-emphasizes the motivating variables of the different verbal behaviors and conducts training using edible or tangible consequences and social praise almost exclusively. Although generalization is a key component of both approaches, interventions based on the analysis of verbal behavior are more likely to begin teaching under more naturally occurring motivating conditions, whereas in the Lovaas approach, generalization trials are typically conducted after behaviors are established in highly structured, analog environments.

Rationale or Underlying Theory

There are two kinds of language analyses: formal and functional. A formal analysis considers what verbal behavior looks like or its form (also called topography). The linguistic perspective is formal because words and grammatical structures are the units of analysis. In contrast, Skinner analyzed verbal behavior in terms of functional units. This use of functional does not mean useful but rather causal. In other words, the cause of the behavior is more important to its understanding than what the behavior looks like. A functional unit takes into account the verbal behavior of interest (e.g., mand, tact, intraverbal) and its related antecedents, consequences, and motivating variables. According to this analysis, basic verbal behaviors are defined by the conditions and variables that control them (i.e., their cause).

Defined specifically by the functional variables controlling their use, Skinner proposed a number of elementary verbal behaviors: mand, tact, echoic, and intraverbal. *Mands* are under the functional control of motivating variables (e.g., deprivation, aversive stimulation) and specific reinforcement. Mands are like demands, commands, or requests because they include information about what is wanted or needed. For example, a speaker has not had a drink in a long time (deprivation) says, "Can I have a drink?" (mand) and receives a glass of water (specific reinforcement) from a listener. A *tact* is controlled by nonverbal antecedent stimuli and generalized reinforcement such as attention

or approval. If a glass of water sat on the counter (nonverbal antecedent stimulus) and upon seeing it the speaker said, “water” and was given approval (generalized reinforcement) from a listener, the response “water” is a tact. *Echoic* behaviors are those that are controlled by verbal antecedent stimuli with a matching response form and generalized reinforcement. For example, a person (speaker 1) models the verbal response “water” (verbal antecedent stimulus), and a second person (speaker 2) repeats “water” (echoic) and receives praise (generalized reinforcement) from speaker 1 for making the response sound like the model. *Intraverbals* are also controlled by verbal antecedent stimuli and generalized reinforcement. However, intraverbals are not similar in form to their verbal antecedent stimuli like echoic behaviors. If instead of modeling the verbal behavior “water” in the echoic example, the first speaker had asked, “What is your favorite drink?” (verbal antecedent stimulus) and the second speaker said, “water” and received approval (generalized reinforcement), the response “water” would be an intraverbal.

Goals and Objectives

Skinner’s *Verbal Behavior* is a theoretical framework with direct implications for teaching verbal behavior to individuals with language deficits (e.g., children with autism). A functional analysis of language leads to informative language assessment, a recognition of naturally occurring motivating variables, an emphasis on mands as principal communication skills, and intraverbal instruction to promote language development beyond the basics. Parents and professionals can draw from the analysis of verbal behavior to make decisions regarding instructional approaches such as augmentative communication, discrete trial training vs. natural environment teaching, and inclusion. For example, from a verbal behavior perspective, a more complete language repertoire can be acquired through a combination of discrete trial training (DTT) and natural environment teaching (NET) procedures. For children with autism, inclusion in regular education may be more effective once children master the basic verbal behaviors

(i.e., mand, tact, and intraverbal) necessary to benefit from an integrated learning environment.

Treatment Participants

The analysis of verbal behavior applies to all humans; however, interventions based on this analysis have been designed primarily for children and adults with autism and other developmental disabilities. Skinner’s analysis is not restricted to individuals with language deficits.

Treatment Procedures

Interventions based on the analysis of verbal behavior include a variety of procedures. There is not one standardized model of verbal behavior treatment. However, there are many teaching procedures that are common among them such as the manipulation of motivating variables, prompting, shaping, fading, and transfer of stimulus control. Verbal behavior interventions are likely to balance opportunities for instruction in highly structured, teacher-directed (e.g., discrete trial training) arrangements with opportunities for incidental, child-directed instruction (e.g., natural environment teaching) to capture natural motivating conditions. See ► [“Verbal Behavior Interventions”](#).

Efficacy Information

Based primarily on its conceptual logic, Skinner’s analysis has been applied in the treatment of children with autism for several decades. The Analysis of Verbal Behavior (TAVB), a journal dedicated to publishing verbal behavior research, was first published in 1982. As a result, there is a growing body of literature supporting the main premises of Skinner’s analysis of verbal behavior and demonstrating efficacy of teaching procedures based on the analysis (Sautter and LeBlanc 2006). Much of this literature involves individuals with autism as participants. However, there are no studies that document the outcome of the long-

term application of treatment based on the analysis of verbal behavior and only one study comparing verbal behavior and linguistic approaches to instruction (Carr and Firth 2005).

Outcome Measurement

There are two widely used measurement tools based on Skinner's analysis of verbal behavior. The *Assessment of Basic Language and Learning Skills* (ABLLS; Partington and Sundberg 1998; Partington 2010) is a criterion referenced assessment, curriculum guide, and tracking system for children covering basic learner skills (e.g., imitation, requests, intraverbals), academic skills (e.g., reading, math), self-help skills, and motor skills. A companion manual *Teaching Language to Children with Autism or Other Developmental Disabilities* (Sundberg and Partington 1998) was published at the same time as the ABLLS. In 2010, Partington published the ABLLS-Revised, which is a common tool used for school age children with autism and other developmental disabilities. In 2008, Sundberg published his own assessment tool that integrates developmental milestones with key verbal behaviors. The *Verbal Behavior-Milestone Assessment and Placement Program* (VB-MAPP) includes a stronger focus on placement and individualized education program (IEP) development and subsections for milestones, barriers, and transitions.

Qualifications of Treatment Providers

Although the analysis of verbal behavior can be used to derive treatment procedures, Skinner did not specify a set of tactics to teach verbal behavior. Likewise, there are also no provider qualifications. That being said, Skinner's book *Verbal Behavior* is incredibly complex. Its technical content is appropriate for individuals with an invested interest. Summaries of Skinner's main tenets can be found in more beginner-friendly formats (see References).

Professionals who apply the analysis of verbal behavior in the treatment of individuals with autism need to have advanced training in applied behavior analysis, verbal behavior, and extensive

supervised experience implementing verbal behavior interventions. Preferably, verbal behavior providers have been credentialed by the Behavior Analysis Certification Board (BACB) or have completed the equivalent training. In general, verbal behavior interventions require that providers have more skill and training than discrete trial training (DTT) procedures do.

See Also

- ▶ [Applied Behavior Analysis \(ABA\)](#)
- ▶ [Behavior Analyst Certification Board](#)
- ▶ [Behavior Modification](#)
- ▶ [Behaviorism](#)
- ▶ [Language Acquisition](#)
- ▶ [Language Interventions](#)
- ▶ [Lovaas Approach](#)
- ▶ [Theories of Language Development](#)
- ▶ [Verbal Behavior Interventions](#)

References and Reading

- Barbera, M., & Rasmussen, R. (2007). *The verbal behavior approach: How to teach children with autism and related disorders*. Philadelphia: Jessica Kingsley.
- Carr, J. E., & Firth, A. M. (2005). The verbal behavior approach to early and intensive behavioral intervention for autism: A call for additional empirical support. *Journal of Early and Intensive Behavioral Intervention*, 2, 18–27.
- Chomsky, N. (1959). A review of B.F. Skinner's verbal behavior. *Language*, 35(1), 26–58.
- Hedge, M. N., & Maul, C. A. (2006). *Language disorders in children: An evidence-based approach to assessment and treatment*. Boston: Pearson.
- Lovaas, O. I. (2003). *Teaching individuals with developmental delays: Basic intervention techniques*. Austin: PRO-ED.
- Partington, J. W. (2010). *Assessment of basic language and learning skills revised (ABLLS-R)*. Pleasant Hill: Behavior Analysts, Inc.
- Partington, J. W., & Sundberg, M. L. (1998). *Assessment of basic language and learning skills (The ABLLS): An assessment for language delayed students*. Pleasant Hill: Behavior Analysts.
- Pierce, W. D., & Cheney, C. D. (2004). *Behavior analysis and learning* (3rd ed.). Mahwah: Lawrence Erlbaum Associates.
- Sautter, R. A., & LeBlanc, L. A. (2006). Empirical applications of Skinner's analysis of verbal behavior with humans. *The Analysis of Verbal Behavior*, 22, 35–48.
- Skinner, B. F. (1957). *Verbal behavior*. Acton: Copley.
- Sundberg, M. L. (2007). Verbal behavior. In J. O. Cooper, T. E. Heron, & W. L. Heward (Eds.), *Applied behavior*

analysis (2nd ed., pp. 526–547). Upper Saddle River: Merrill/Prentice Hall.

Sundberg, M. L. (2008). *Verbal behavior milestones assessment and placement program: The VB-MAPP*. Concord: AVB Press.

Sundberg, M. L., & Michael, J. (2001). The benefits of Skinner's analysis of verbal behavior for children with autism. *Behavior Modification*, 25(5), 698–724.

Sundberg, M. L., & Partington, J. W. (1998). *Teaching language to children with autism or other developmental disabilities*. Danville: Behavior Analysts.

Vargas, J. S. (2009). *Behavior analysis for effective teaching*. New York: Routledge.

Definition

An anecdotal observation is a factual account of an incident. The precise sequence of events is documented using descriptive language in order to describe exactly what occurs during a given situation. The setting and context are also carefully described. Subjective statements and judgments should be avoided during anecdotal observations. Therefore, a written anecdotal observation should provide the reader with a clear picture of the event.

In autism, anecdotal observations are often helpful in learning more about a child's behavior. Parents may be asked to make anecdotal observations of their child in order to keep a detailed record of their behavior, monitor their response to particular events, track progress during intervention, or provide information about their behavior following a change. Such information can be valuable for a service provider during assessment or when developing and/or maintaining a therapy program. School staff and treatment providers may decide to use their own anecdotal observations as evidence for the need to implement or modify a treatment program or intervention strategy. For example, an anecdotal observation during the school day may reveal deterioration in a child's behavior whenever there is a school assembly. Tracking these events and responses via direct observations can be useful in determining a pattern of behavioral challenges. They may provide the support necessary to put strategies in place in order to prepare the child for assembly days. Parents and service providers can analyze anecdotal observations to determine patterns such as these and better serve children in need.

Although anecdotal observations can provide a deeper understanding of behavior in one particular individual, caution should be used when applying any conclusions drawn from the observation to other individuals. Because anecdotal observations are individualized to a specific event and person, generalizations to other individuals may not be valid and can at times lead to faulty conclusions, even for those in a similar situation or who have the same diagnosis. Furthermore, anecdotal observations should not replace controlled studies when making judgments about causal relationships because

Analyst

► [Psychologist](#)

Analytic Processing

► [Sequential Processing](#)

Anatomy of Human Ear

► [Auditory System](#)

Androgens (Male Sex Hormones)

► [Sex Hormones](#)

Anecdotal Observation

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Synonyms

[Anecdotal record](#)

they do not include adequate sample sizes and sets of observations that are representative of many individuals.

See Also

- [Behavioral Assessment](#)
- [Direct Observation](#)
- [Functional Behavior Assessment](#)
- [Observational Assessments](#)

References and Reading

- Bentzen, W. R. (2000). *Seeing young children: A guide to observing and recording behavior* (4th ed.). Albany: Delmar Learning.
- Nicolson, S., & Shipstead, S. G. (2002). *Through the looking glass: Observations in the early childhood classroom* (3rd ed.). Upper Saddle River: Prentice Hall.

Anecdotal Record

- [Anecdotal Observation](#)

Angelman/Prader-Willi Locus

- [Chromosome 15q11–q13](#)

Angelman/Prader-Willi Syndromes

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Synonyms

[Prader-Labhart-Willi syndrome](#)

Short Description or Definition

Prader-Willi syndrome (PWS) and Angelman syndrome (AS) are two distinct neurodevelopmental disorders caused by mutations in the same region of the genome, involving chromosome 15q11.2-15q13.3.

Categorization

Genetic syndromes, Neurodevelopmental disorders.

Epidemiology

The prevalence of PWS is approximately 1 in 10,000 individuals (Dykens et al. [2011](#)). The prevalence of AS is 1 in 12,000–20,000 individuals (Williams et al. [2010](#)).

Natural History, Prognostic Factors, Outcomes

Infants with PWS have severe hypotonia and difficulties with feeding. The latter evolves into hyperphagia (excessive eating) and morbid obesity. Obesity-related problems, such as non-insulin-dependent diabetes mellitus, are the most serious health issues. Weight control becomes critical for maximizing health outcomes. Children with PWS experience multiple developmental delays in cognition, language, motor skills, and physical growth. Short stature and hypogonadism are common, the latter affecting pubertal development and resulting in infertility.

Children with AS also experience multiple developmental delays in cognition, language, motor skills (gait ataxia), and physical growth (microcephaly). Seizures, which typically occur during infancy and the toddler years, can be of varied types. In contrast to PWS, individuals with AS have normal pubertal development and fertility. Aside from possible seizures, they typically have good health and normal life spans. They require lifelong supervision.

Clinical Expression and Pathophysiology

A key concept to understanding these syndromes is genomic imprinting. Typically, a child inherits two copies of each gene, one transmitted from the father and one from the mother. In many instances, these pairs of genes work in concert to achieve full function. In the region denoted chromosome 15q11.2–15q13.3, there are a number of genes which are only active (translated to proteins), depending on whether they are inherited from the father or mother. This phenomenon, in which a gene or genes is silent on either maternally or paternally transmitted chromosome, is termed imprinting.

In PWS, 70% of cases are due to a deletion involving the segment 15q11.2–15q13.3 of the paternal chromosome. Because many of the genes in this region are imprinted (or silent) on the maternal chromosome, this results in the loss of all gene products. Another 25% of cases are due to maternal uniparental disomy, a condition in which both copies of the chromosome are inherited from the mother. Five percent of cases are due to chromosomal breakpoints which disrupt genes within the region or mutations which affect the proper imprinting of this interval. While it has not been definitively determined which gene(s) in the interval cause PWS, recently deficiency of paternally expressed small nucleolar RNAs (snoRNAs) has been considered the leading suspects. These RNAs regulate the expression of another gene which is involved in serotonin neurotransmission, the serotonin 2C receptor (Dykens et al. 2011). AS is due to deficiency of the maternally expressed UBE3A gene. This gene shows paternal imprinting, meaning it is silent on the paternal chromosome. Sixty-five to seventy-five percent of cases are due to deletions of the maternal chromosome, 5–11% are due to mutations in the UBE3A gene, 3–7% are due to paternal uniparental disomy (both copies of the chromosome are inherited from the father), and 3% of cases are due to imprinting mutations (Williams et al. 2010).

The chromosome 15q11.2–15q13.3 region has also been implicated in autism spectrum disorders (ASDs). A high proportion of patients

with duplications at this locus meets diagnostic criteria for ASD (Abrahams and Geschwind 2008). Conversely, in some clinical ASD cohorts, up to 1% of patients show maternal duplications of this interval (Sanders et al. 2011). It is among the most common chromosomal rearrangements seen in ASD. There are quite a few overlapping clinical features between PWS and ASD, and it has been suggested that the conventional autism diagnostic tests (ADOS and ADI-R) may not be sufficient to discriminate between PWS and ASDs (Dykens et al. 2011). Greater than 40% of patients with AS have ASD, although the converse is rare (proportion of patients with ASD who have AS).

Evaluation and Differential Diagnosis

PWS is a common cause of hypotonia at birth and may be identified early by genetic testing. If this is not identified early, clinical diagnosis is suspected based on the combination of short stature, behavioral issues, and hyperphagia, typically after age 6. The diagnosis can be confirmed in the vast majority of cases via DNA testing. It is characterized by a wide range of symptoms, many of which are behavioral or endocrine in nature. One of the most common symptoms associated with the disorder is an insatiable appetite that often leads to morbid obesity. This is due to dysfunction of the hypothalamus, the region of the brain which regulates feelings of satiety and hunger (Butler 2011). Patients with PWS have high levels of ghrelin, a compound that is found in the lining of the stomach and stimulates hunger, but whether this finding is a cause or consequence of primary problems in PWS is not known. The typical psychiatric difficulties faced by people with PWS include anxiety and compulsive behavior, including skin picking. Smaller subsets of patients are affected by symptoms such as depression, hallucination, and paranoia. In almost all cases, people with PWS have below average intelligence, with the median IQ being in the 50–70 range (Dykens et al. 2011).

The diagnosis of AS is usually suspected by early developmental delay and behavioral

manifestations and can be confirmed by DNA testing. It is characterized by severe cognitive and neurological impairment. While the manifestation and severity of symptoms varies greatly, there are a few which are the most common, appearing in almost 100% of cases. Patients always experience severe developmental delay as well as movement and balance issues. Consistently, patients are afflicted with speech impairment. Some are nonverbal, while others have very limited vocabulary. One characteristic trait of individuals with AS is their apparently happy demeanor, frequent laughter, and hand flapping. Slightly less common traits are diminished head size and the onset of seizures before the age of 3. Clinical diagnosis of AS can be complicated. Usually, a successful diagnosis involves motor and speech delays, as well as the characteristic motor mannerisms and demeanor (Cassidy et al. 2000). If AS is suspected, an EEG (electroencephalogram) may be performed to rule out gelastic seizure, a rare type of seizure which is accompanied by a burst of energy (Williams 2005).

Treatment

There is no cure for PWS; however, there are treatments to lessen symptoms. These include starting physical therapy early to help with muscle tone. Children should be placed in a structured school environment with close teacher supervision. Occupational and speech therapy should be provided if needed. Strict supervision of diet is required to address hyperphagia and prevent morbid obesity and its attendant health problems. Clinical trials of growth hormone replacement therapy have shown cognitive as well as physical benefits (Cassidy and Schwartz 2009). The latter includes increasing height, lean body mass, and mobility and decreasing fat. Adults with PWS most often require supervised living situations and work environments.

As with PWS, there is no cure for AS, but medications are used to treat the various symptoms. This includes anticonvulsants to combat the seizures, melatonin to encourage regular sleep

patterns, and laxatives for regular bowel movements. Beginning physical and occupational therapy early is also important to promote muscle development and decrease joint stiffness. Given the typically severe speech impairment, speech therapy should emphasize nonverbal methods of communication, such as picture cards (Dagli and Williams 2011). AS is not degenerative; in fact, many symptoms improve with age, such as seizures, sleep issues, and continence. Life expectancy is average, and while people with AS may never be fully independent, adults can learn basic daily living skills.

See Also

► [Chromosome 15q11–q13](#)

References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, 9, 341–355.
- Buiting, K. (1995). Inherited microdeletions in the Angelman and Prader-Willi syndromes define an imprinting centre on human chromosome 15. *Nature Genetics*, 9, 395–400.
- Buiting, K. (2010). Prader-Willi syndrome and Angelman syndrome. *American Journal of Medical Genetics. Part C, Seminars in Medical Genetics*, 154C, 365–376.
- Butler, M. G. (2011). Prader-Willi syndrome: Obesity due to genomic imprinting. *Current Genomics*, 12, 204–215.
- Cassidy, S. B., & Schwartz, S. (2009). Prader-Willi syndrome. *GeneReviews*. Retrieved January, 2012, from <http://www.ncbi.nlm.nih.gov/books/NBK1330/#pws.REF.west.2004.565>
- Cassidy, S. B., Dykens, E., & Williams, C. A. (2000). Prader-Willi and Angelman syndromes: Sister imprinted disorders. *American Journal of Medical Genetics (Seminar Medical Genetics)*, 97, 136–146.
- Christian, S. L., Fantes, J. A., Mewborn, S. K., Huang, B., & Ledbetter, D. H. (1999). Large genomic duplicons map to sites of instability in the Prader-Willi/Angelman syndrome chromosome (15q11–q13). *Human Molecular Genetics*, 8, 1025–1037.
- Dagli, A. I., & Williams, C. A. (2011). Angelman syndrome. *GeneReviews*. Retrieved January, 2012, from <http://www.ncbi.nlm.nih.gov/books/NBK1144/>
- Dykens, E. M., Lee, E., & Roof, E. (2011). Prader-Willi syndrome an autism spectrum disorders: An evolving story. *Journal of Neurodevelopmental Disorders*, 3, 225–237.

- Geshwind, D. H. (2008). Autism: Many genes, common pathways? *Cell*, 135(3), 391–395.
- Matsuura, T., Sutcliffe, J. S., Fang, P., Galjaard, R. J., Jiang, Y. H., Benton, C. S., et al. (1997). De novo truncating mutations in E6-AP ubiquitin-protein ligase gene (UBE3A) in Angelman Syndrome. *Nature Genetics*, 15, 74–77.
- Sanders, S. J., Ercan-Sencicek, A. G., Hus, V., Luo, R., Murtha, M. T., Moreno-De-Luca, D., et al. (2011). Multiple recurrent de novo CNVs, including duplications of 7q11.23 Williams syndrome region, are strongly associated with autism. *Neuron*, 70, 863–885.
- Williams, C. A. (2005). Neurological aspects of the Angelman syndrome. *Brain & Development*, 27, 88–94.
- Williams, C. A., Driscoll, D. J., & Dagli, A. I. (2010). Clinical and genetic aspects of Angelman syndrome. *Genetics in Medicine*, 12(7), 385–395.

Anger Rumination in Children with Autism Spectrum Disorder

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Definition

Anger rumination is a cognitive-emotional process referring to the tendency to dwell on frustrating experiences and recall past anger experiences (Sukhodolsky et al. 2001). More generally, rumination represents a maladaptive form of emotion processing that entails remaining focused on the stressor through repetitive and passive dwelling upon distress, past mistakes, regrets, and shortcomings (Nolen-Hoeksema 1991; Nolen-Hoeksema et al. 2008). Further, rumination may hinder the use of cognitive control strategies such as reappraisal and problem-solving (Nolen-Hoeksema 1991; Nolen-Hoeksema et al. 2008), most likely due to prolongation of negative affect. While rumination is shown to be associated with internalizing disorders such as anxiety and depression (Connor-Smith et al. 2000; Nolen-Hoeksema et al. 2008), rumination may also be a factor in other forms of maladaptive behaviors such as

disruptive behaviors including irritability/anger and aggression (Aldao et al. 2016; McLaughlin et al. 2014; Nolen-Hoeksema and Watkins 2011).

Historical Background

The Anger Rumination Scale (ARS) was developed by Sukhodolsky et al. (2001) to assess cognitive processes that unfold during and continue after the emotion of anger has been generated. The ARS is a widely used and well-established self-report measure of the construct of anger-focused rumination. The ARS includes 19 items assessing the cognitive processes related to feelings of anger, the tendency to think about anger-provoking situations, and tendency to recall past anger episodes. The ARS has four subscales that measure anger afterthoughts, thoughts of revenge, angry memories, and understanding of causes. The subscales anger afterthoughts and thoughts of revenge correspond to thinking about a recent episode or recalling and getting angry about a distant episode, while the subscales angry memories and understanding of causes correspond to thinking about causes of an anger episode in order to achieve a meaningful understanding of the episode. Higher scores on the ARS indicate a greater level of anger rumination. Further, the ARS has demonstrated high internal reliability for use with children with ASD and children with disruptive behaviors (alphas >0.8) (Ibrahim et al. 2019; Patel et al. 2017; Smith et al. 2016), as well as adequate test-retest reliability ($r = 0.77$).

Current Knowledge

Anger Rumination and Associations with Co-occurring Disorders in ASD

Over 50% of children with autism spectrum disorder (ASD) have co-occurring disruptive behavior disorders and/or internalizing disorders (Aldao et al. 2016; McLaughlin et al. 2014; Nolen-Hoeksema and Watkins 2011) that cause distress and impairment in various domains of functioning. Additionally, children and adults with ASD exhibit elevated levels of anger rumination

relative to typically developing controls, which is also associated with co-occurring internalizing and/or externalizing symptoms. For instance, several studies have consistently reported an association between anger rumination and disruptive behaviors (Ibrahim et al. 2019; Patel et al. 2017; Pugliese et al. 2015). Consistent with studies of non-ASD populations indicating greater levels of anger rumination in youths with DBD (Harmon et al. 2017; Smith et al. 2016), recent work also suggests that children with ASD may show levels of anger rumination that are greater relative to typically developing controls, but similar to children with disruptive behavior disorders without ASD (Ibrahim et al. 2019). Further, there is evidence to suggest that children with ASD and co-occurring disruptive behavior may show greater levels of anger rumination relative to children with ASD without disruptive behavior (Ibrahim et al. 2019). The developmental trajectory of rumination broadly may also predict increased disruptive behavior in later adolescence in youths with ASD (Bos et al. 2018). Anger rumination has also been linked to anxiety symptoms and disruptive behaviors in young adults in the general population, in which symptoms of social anxiety may predict greater levels of anger rumination and, thereby, increased levels of disruptive behavior (Pugliese et al. 2015). Lastly, associations between anger rumination and depressive symptoms have also been reported in youths with ASD (Patel et al. 2017). Thus, similar patterns of elevated anger rumination across ASD and disruptive behavior disorders lend support to the notion that rumination more broadly may be a transdiagnostic factor (Aldao et al. 2016). Additionally, similar patterns of elevated anger rumination in ASD and non-ASD populations with disruptive behavior could also suggest shared aberrations in the mechanisms subserving cognitive control.

Anger Rumination and the Relationship with Core ASD Symptoms

Recent work implicates rumination in general with core ASD symptoms such as restricted and repetitive behaviors (RRBs) including an insistence on sameness, inflexible adherence to routines, rigidity of thought, and perseveration (Gotham et al. 2014; Keenan et al. 2017). For

instance, studies have suggested a relationship between a greater tendency to engage in anger rumination and the severity of core ASD symptoms, particularly RRBs (Ibrahim et al. 2019; Patel et al. 2017; Pugliese et al. 2015). A recent study compared samples of children with and without ASD and disruptive behavior and reported an interaction between ASD diagnosis and RRBs in predicting anger rumination; that is, the presence of an ASD diagnosis and greater severity of RRBs was related to higher levels of anger rumination (Ibrahim et al. 2019). Further, levels of anger rumination were found to be positively correlated with the severity of RRBs when modeled dimensionally across the total sample of youths with ASD (Ibrahim et al. 2019). Another study showed that levels of perseveration were found to augment the relationship between anger-focused rumination and disruptive behaviors (Pugliese et al. 2015). It should also be noted that other forms of rumination including depressive rumination have also been shown to be associated with perseveration in young adults (Keenan et al. 2017) as well as adults with ASD (Gotham et al. 2014). Lastly, Patel et al. (2017) demonstrated that anger rumination in children was positively correlated with overall core ASD symptoms, including social communication and interaction and RRBs symptoms. Thus, it is also possible that specific types of RRBs, such as insistence on sameness and perseveration, could differentially contribute to levels of anger rumination in ASD (Gotham et al. 2014; Pugliese et al. 2015). However, future studies are needed that include measures of perseveration on circumscribed interests to understand whether anger rumination is a manifestation of a perseverative type of repetitive behavior or a distinct trait.

Rumination and Emotion Dysregulation in ASD

It has also been hypothesized that the association between rumination more broadly and RRBs may contribute to overall emotion dysregulation in ASD, which could suggest common underlying deficits in cognitive control neural circuitry (Mazefsky et al. 2013). It is possible that anger rumination may be part of the constellation of core ASD symptoms, particularly RRBs including

rigidity of thinking, insistence on sameness, and perseveration. It is also important to emphasize that other forms of ruminative thoughts, including depressive rumination, have been shown to be associated with RRBs in individuals with ASD (Jahromi et al. 2012; Mazefsky et al. 2013, 2014; Rieffe et al. 2011). Thus, greater difficulty disengaging from perseverative thoughts could predispose children with ASD to engage in rumination (Mazefsky et al. 2012). Additionally, in children and adults with ASD, impairments in emotional reactivity and cognitive control in combination with RRBs may hinder the use of adaptive, voluntary emotion regulation strategies, such as reappraisal and problem-solving (Ibrahim et al. 2019).

Models of emotion dysregulation in ASD propose that amplified emotional reactivity paired with poor cognitive control may characterize emotion regulation impairments in ASD, predisposing children with ASD to an increased risk for developing co-occurring psychiatric disorders (Mazefsky et al. 2013). The tendency of individuals with ASD to engage in high levels of rumination or maladaptive cognitive control patterns compared to non-ASD populations could suggest broader deficits in the underlying mechanism of emotion regulation in ASD (Mazefsky et al. 2013). For instance, given that emotion regulation impairments are common in ASD, reliance on involuntary maladaptive strategies such as suppression and rumination may hinder the use of adaptive regulation processes such as cognitive reappraisal or problem-solving (Mazefsky et al. 2012). Thus, the tendency to engage in anger rumination, or ruminative thoughts in general, could also be related to broader deficits in emotion regulation in ASD. Further, the overlap between anger rumination, emotion dysregulation, and RRBs could predispose children with ASD to a heightened risk for disruptive behaviors.

Future Directions

Given the relationships between anger rumination, or rumination more broadly, co-occurring disorders, and repetitive behaviors in ASD such as perseveration, this area of inquiry has the potential to advance understanding of the underlying mechanisms of

emotion dysregulation and the overlap of anger rumination with core ASD symptoms. Future research on rumination in ASD could also pave the way for new, more effective treatments that target perseverative thoughts, as well as for improved diagnostic approaches that can more accurately distinguish between rumination, perseveration, and other associated symptoms such as anxiety and disruptive behavior. Therefore, a better understanding of the mechanisms underlying anger rumination in ASD could contribute to the development of novel treatments for decreasing disruptive behaviors and improving emotion regulation strategies (Jahromi et al. 2012; Mazefsky et al. 2013, 2014; Rieffe et al. 2011). For example, future studies could examine whether perseverative tendencies of individuals with ASD are associated with anxiety, disruptive behavior, and depressive symptoms or whether perseveration could be harnessed in interventions to increase recall of positive autobiographic memories. Given the high prevalence of comorbid psychiatric disorders among individuals with ASD and DBDs (Lecavalier et al. 2019; Leyfer et al. 2006; Simonoff et al. 2008; van Steensel et al. 2013), more studies are needed with sufficiently large samples to compare anger rumination in children with ASD and co-occurring DBDs to children with DBDs without ASD in order to better understand the overlap between core ASD symptoms and disruptive behaviors in predicting anger rumination. Additionally, given the increased prevalence of trauma in children with ASD (Haruvi-Lamdan et al. 2017; Kerns et al. 2015) and the role that rumination may play in strengthening the association between trauma symptoms and anger (Spinhoven et al. 2015), future studies are needed that examine the effects of co-occurring trauma, such as maltreatment or bullying, in ASD and its relationship to anger rumination and core ASD symptoms. Finally, it will be important to understand the impact of sex differences on anger rumination in children with ASD.

See Also

- [Emotional Regulation](#)
- [RDoC and Autism](#)
- [Rumination](#)

References and Reading

- Aldao, A., Gee, D. G., De Los Reyes, A., & Seager, I. (2016). Emotion regulation as a transdiagnostic factor in the development of internalizing and externalizing psychopathology: Current and future directions. *Development and Psychopathology*, 28(4 pt 1), 927–946. <https://doi.org/10.1017/s0954579416000638>.
- Bos, M. G., Diamantopoulou, S., Stockmann, L., Begeer, S., & Rieffe, C. (2018). Emotion control predicts internalizing and externalizing behavior problems in boys with and without an autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48, 2727–2739.
- Connor-Smith, J. K., Compas, B. E., Wadsworth, M. E., Thomsen, A. H., & Saltzman, H. (2000). Responses to stress in adolescence: Measurement of coping and involuntary stress responses. *Journal of Consulting and Clinical Psychology*, 68(6), 976.
- Gotham, K., Bishop, S. L., Brunwasser, S., & Lord, C. (2014). Rumination and perceived impairment associated with depressive symptoms in a verbal adolescent–adult ASD sample. *Autism Research*, 7(3), 381–391.
- Harmon, S. L., Stephens, H. F., Repper, K. K., Driscoll, K. A., & Kistner, J. A. (2017). Children's rumination to sadness and anger: Implications for the development of depression and aggression. *Journal of Clinical Child & Adolescent Psychology*, 48, 622–632.
- Haruvi-Lamdan, N., Horeish, D., & Golan, O. (2017). PTSD and Autism Spectrum Disorder: Co-morbidity, gaps in research, and potential shared mechanisms. *Psychological Trauma*. <https://doi.org/10.1037/tra0000298>.
- Ibrahim, K., Kalvin, C., Marsh, C. L., Anzano, A., Gorynova, L., Cimino, K., & Sukhodolsky, D. G. (2019). Anger rumination is associated with restricted and repetitive behaviors in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49, 3656–3668.
- Jahromi, L. B., Meek, S. E., & Ober-Reynolds, S. (2012). Emotion regulation in the context of frustration in children with high functioning autism and their typical peers. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 53(12), 1250–1258. <https://doi.org/10.1111/j.1469-7610.2012.02560.x>.
- Keenan, E. G., Gotham, K., & Lerner, M. D. (2017). Hooked on a feeling: Repetitive cognition and internalizing symptomatology in relation to autism spectrum symptomatology. *Autism*. <https://doi.org/10.1177/1362361317709603>.
- Kerns, C. M., Newschaffer, C. J., & Berkowitz, S. J. (2015). Traumatic childhood events and autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(11), 3475–3486.
- Lecavalier, L., McCracken, C. E., Aman, M. G., McDougle, C. J., McCracken, J. T., Tierney, E., ... Handen, B. (2019). An exploration of concomitant psychiatric disorders in children with autism spectrum disorder. *Comprehensive psychiatry*, 88, 57–64.
- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., ... Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36(7), 849–861. <https://doi.org/10.1007/s10803-006-0123-0>.
- Mazefsky, C. A., Pelphrey, K. A., & Dahl, R. E. (2012). The need for a broader approach to emotion regulation research in autism. *Child Development Perspectives*, 6(1), 92–97. <https://doi.org/10.1111/j.1750-8606.2011.00229.x>.
- Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 52(7), 679–688.
- Mazefsky, C. A., Borue, X., Day, T. N., & Minshew, N. J. (2014). Emotion regulation patterns in adolescents with high-functioning autism spectrum disorder: Comparison to typically developing adolescents and association with psychiatric symptoms. *Autism Research*, 7(3), 344–354. <https://doi.org/10.1002/aur.1366>.
- McLaughlin, K. A., Aldao, A., Wisco, B. E., & Hilt, L. M. (2014). Rumination as a transdiagnostic factor underlying transitions between internalizing symptoms and aggressive behavior in early adolescents. *Journal of Abnormal Psychology*, 123(1), 13.
- Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, 100(4), 569.
- Nolen-Hoeksema, S., & Watkins, E. R. (2011). A heuristic for developing transdiagnostic models of psychopathology: Explaining multifinality and divergent trajectories. *Perspectives on Psychological Science*, 6(6), 589–609.
- Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science*, 3(5), 400–424.
- Patel, S., Day, T. N., Jones, N., & Mazefsky, C. A. (2017). Association between anger rumination and autism symptom severity, depression symptoms, aggression, and general dysregulation in adolescents with autism spectrum disorder. *Autism*, 21(2), 181–189. <https://doi.org/10.1177/1362361316633566>.
- Pugliese, C. E., Fritz, M. S., & White, S. W. (2015). The role of anger rumination and autism spectrum disorder-linked perseveration in the experience of aggression in the general population. *Autism*, 19(6), 704–712. <https://doi.org/10.1177/1362361314548731>.
- Rieffe, C., Oosterveld, P., Terwogt, M. M., Mootz, S., van Leeuwen, E., & Stockmann, L. (2011). Emotion regulation and internalizing symptoms in children with autism spectrum disorders. *Autism*, 15(6), 655–670. <https://doi.org/10.1177/1362361310366571>.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(8), 921–929.

- Smith, S. D., Stephens, H. F., Repper, K., & Kistner, J. A. (2016). The relationship between anger rumination and aggression in typically developing children and high-risk adolescents. *Journal of Psychopathology and Behavioral Assessment*, 38(4), 515–527.
- Spinhoven, P., Penninx, B. W., Krempehou, A., van Hemert, A. M., & Elzinga, B. (2015). Trait rumination predicts onset of Post-Traumatic Stress Disorder through trauma-related cognitive appraisals: A 4-year longitudinal study. *Behaviour Research and Therapy*, 71, 101–109.
- Sukhodolsky, D. G., Golub, A., & Cromwell, E. N. (2001). Development and validation of the anger rumination scale. *Personality and Individual Differences*, 31(5), 689–700.
- van Steensel, F. J., Bogels, S. M., & de Bruin, E. I. (2013). Psychiatric comorbidity in children with Autism Spectrum Disorders: A comparison with children with ADHD. *Journal of Child and Family Studies*, 22(3), 368–376. <https://doi.org/10.1007/s10826-012-9587-z>.

Animal Models

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Definition

Animal models are useful for testing hypotheses about biological mechanisms underlying the causes and symptoms of human psychiatric disorders and for systematically evaluating the effects of potential treatments. Though animal models cannot fully encapsulate all aspects of autism, mouse behaviors with strong conceptual analogies to the diagnostic symptoms of autism have been identified. Assays currently in use include tests for social approach, reciprocal social interactions, social communication, repetitive behaviors, and restricted interests. These tasks have been employed to test hypotheses about the genetic and environmental causes of autism. Detection of rodent models with endophenotypes highly relevant to the symptoms of autism is likely to enable the discovery of effective therapeutic interventions.

Historical Background

Animal models of human neuropsychiatric disorders are in widespread use for biomedical research. Many rodent behavioral tasks relevant to the symptoms of these disorders have been developed, and psychopharmacological treatments for many major mental illnesses and neurological diseases have been evaluated in translational rodent models (Covington et al. 2010; Crawley 2007b; Higgins and Jacobsen 2003; Moore 2010). Developing animal models relevant to the symptoms of autism spectrum disorders (ASDs) presents a unique challenge to the biomedical research community. Autism is a complex neurodevelopmental disorder marked by considerable clinical heterogeneity. The diagnostic criteria for autism are behaviorally defined by three criteria: (1) aberrant reciprocal social interactions, (2) impaired communication, and (3) stereotyped repetitive behaviors with restricted narrow interests (American Psychiatric Association 1994; Dawson et al. 2002; Kanner 1943; Piven et al. 1997; Volkmar and Pauls 2003). It is important to note that none of the currently available models fully recapitulate all aspects of ASDs. However, fundamental symptoms of autism can be approximated in animal models in order to test hypotheses about mechanisms underlying the etiology and causes of the disorder and to evaluate potential pharmacological, behavioral, and other treatments that may alleviate symptoms associated with ASDs.

Current Knowledge

Strategies for Designing Rodent Models of Autism

Twin and family studies indicate an extraordinarily high degree of heritability for ASDs. Concordance between monozygotic twins approaches 90% for ASDs as compared with 10% or less in dizygotic twins and approximately 0.6–1.0% occurrence in the general population (Abrahams and Geschwind 2008). Several approaches have been used to generate genetic mouse models of autism and to evaluate the contributions of

specific genes to the symptoms of ASDs. Genes implicated in autism include those coding for proteins involved in synapse development, neuronal signaling, neurotransmission, neuron survival, RNA transcription, and DNA methylation. Targeted mutations in genes homologous or orthologous to human candidate genes for autism have generated a large number of genetic mouse models (Bozdagi et al. 2010; Cheh et al. 2006; DeLorey et al. 2008; Etherton et al. 2009; Hines et al. 2008; Kwon et al. 2006; Nakatani et al. 2009; Peca et al. 2011; Shu et al. 2005; Winslow and Insel 2002). *Mus musculus*, the house mouse species used in molecular genetics research, is a social species that engages in high levels of reciprocal social interaction and social communication, communal nesting, sexual and parenting behaviors, territorial scent marking, and aggressive behaviors (Arakawa et al. 2008; Bolivar et al. 2007; Miczek et al. 2001; Moretti et al. 2005; Scattoni et al. 2009; Terranova and Laviola 2001; Winslow and Insel 2002). Table 1 summarizes autism-relevant behavioral phenotypes in some prominent genetic mouse models of autism.

A second approach, also using mouse models, addresses single-gene neurodevelopmental disorders and those resulting from chromosomal deletions and duplications (copy number variations, CNVs), in which a high number of affected individuals display autism-like symptoms. Lines of mice have been generated with targeted gene mutations relevant to disorders such as Angelman syndrome, fragile X syndrome, Rett syndrome, Timothy syndrome, and tuberous sclerosis (Ehninger et al. 2010; Moretti et al. 2005; Spencer et al. 2011). A mutant mouse line with a duplicated chromosome orthologous to human chromosome 15q11–13 has also recently been generated (Nakatani et al. 2009). Table 2 summarizes autism-relevant behavioral phenotypes in selected mouse models of single-gene neurodevelopmental disorders and disorders resulting from rare CNVs.

A third approach is to generate defects in rats or mice that model reports of autism following exposure to teratogenic drugs, environmental toxins, or prenatal insults. For example, increased risk for autism has been associated with prenatal exposure to the anticonvulsant drug valproic acid,

the antiemetic drug thalidomide, and prenatal viral infections. Models that address hypotheses regarding environmental causes of autism include offspring of pregnant rats and mice treated with valproic acid or immunostimulant compounds that simulate viral infection (Ehninger et al. 2010; reviewed in Dufour-Rainfray et al. 2011, and Patterson 2009). Table 3 summarizes findings of autism-relevant behavioral phenotypes in mouse and rat models used to test hypotheses about environmental factors implicated in autism.

A final approach consists of utilizing naturally occurring variation among genetically diverse inbred mouse strains to identify behavioral phenotypes with strong face validity to ASD symptoms (Bolivar et al. 2007; Brodtkin et al. 2004; Moy et al. 2004, 2007, 2008b; Panksepp et al. 2007). Investigation of inbred strains expressing traits relevant to autism is referred to as a “forward genetics” approach and is analogous to human linkage studies aimed at discovering genes linked to autism (Abrahams and Geschwind 2008). Table 4 lists examples of autism-relevant behavioral phenotypes that have been detected in different inbred strains of mice.

Because no consistent biological markers for autism have been identified, the diagnosis of autism is currently based on standardized evaluation instruments such as ADOS and ADI, which score well-defined behavioral symptoms. In consultation with autism clinical experts, behavioral neuroscientists are refining standard behavioral assays available in the literature and developing new behavioral assays which maximize face validity to the diagnostic symptoms of autism. Reviewed here are the tests that have been most useful, along with the essential control measures, for modeling the diagnostic and associated symptoms of autism in animals.

Rodent Behavioral Tasks Relevant to the Diagnostic Symptoms of Autism

Sociability

The first DSM-IV criterion for autism is qualitative and quantitative impairments in social interactions (APA 1994; Lord et al. 2000; Piven et al. 1997; Volkmar and Pauls 2003). These

Animal Models, Table 1 Autism-relevant behavioral phenotypes in selected mouse models with targeted mutations in genes homologous or orthologous to human candidate genes for autism

	Gene	Protein	Autism-relevant behavioral phenotypes
Synaptic cell adhesion molecules	<i>Nlgn2</i>	Neureligin 2	Low sociability ^a
			Increased stereotyped jumping behavior ^a
	<i>Neurexin-1a</i>	Neurexin-1a	Increased repetitive self-grooming ^b
	<i>Shank3</i>	Shank3	Low sociability ^c
			Reduced reciprocal social interactions ^{c, d}
			Reduced ultrasonic vocalizations ^d
			Increased repetitive self-grooming ^d
Signaling, transcription, methylation, and neurotrophic factors	<i>En2</i>	Engrailed-2	Reduced reciprocal social interactions ^e
	<i>Foxp2</i>	Forkhead box protein 2	Reduced pup ultrasonic vocalizations ^f
	<i>Pten</i>	Phosphatase and tensin homolog	Low sociability ^g
			Reduced reciprocal social interactions ^g
	Neurotransmitters	<i>Gabrb3</i>	GABA A receptor beta3 subunit
Lack of preference for social novelty ^h			
Repetitive stereotyped circling behavior ^h			
<i>Oxt</i>		Oxytocin	Impaired social recognition ⁱ
			Reduced pup ultrasonic vocalizations ⁱ

^aHines et al. (2008)
^bEtherton et al. (2009)
^cPeca et al. (2011)
^dBozdagi et al. (2010)
^eChen et al. (2006)
^fShu et al. (2005)
^gKwon et al. (2006)
^hDeLorey et al. (2008)
ⁱWinslow and Insel (2002)

impairments have been characterized as a lack of interest in others, unusual and inappropriate social approach behaviors, lack of social reciprocity, and failure to develop peer relationships appropriate to developmental ages (Kanner 1943; Lord et al. 2000; Piven et al. 1997; Volkmar and Pauls 2003). Assays used to detect social interaction abnormalities in rodent models of autism include measures of social approach, the partition test, reciprocal social interactions, the visible burrow test, social recognition, and social preference tests.

The automated three-chambered social approach apparatus, developed by Nadler, Moy, Crawley, and colleagues (2004), compares time that a subject mouse spends with a novel mouse versus time that a subject mouse spends with a novel object (Brodkin et al. 2004; DeLorey et al. 2008; Hines et al. 2008; McFarlane et al. 2008; Moy et al. 2004, 2009; Nadler et al. 2004; Nakatani et al. 2009; Ryan et al. 2010). Detailed procedures for conducting this task are available (Yang et al. 2011b). The subject mouse is first

Animal Models, Table 2 Selected examples of mouse models of genetic syndromes in which a portion of patients display autistic behaviors

Genetic syndrome	Genetic syndrome characteristics	Mouse model	Autism-relevant behavioral phenotypes
Fragile X syndrome	Lack of fragile X mental retardation protein (FMRP) production; associated with cognitive impairments, hyperactivity, social anxiety, attention problems, executive function impairments, and autistic-like behavior in affected males	Mice with a targeted mutation in the murine <i>Fmr1</i> gene	Low sociability ^{a, b}
			Reduced reciprocal social interactions ^a
			Reduced social interest during a partition test ^a
			High levels of self-grooming ^a
			Increased motor stereotypies and repetitive marble burying ^a
			Resistance to change in a selective attention task ^a
Rett syndrome	Loss of function mutations in the X-linked gene methyl-CpG-binding protein 2 (<i>MECP2</i>); characterized by loss of acquired motor, social, and language skills beginning at 6–18 months of age and nonsyndromic mental retardation	Mice with a heterozygous mutation in the murine <i>Mecp2</i> gene	Social avoidance ^c
			Reduced reciprocal social interactions ^c
Chromosome 15q duplication syndrome	Duplication at chromosome 15q11–13; implicated in ASDs in several association studies	Duplication in the genomic region on the mouse chromosome 7 homologous to the human genomic region 15q11–13	Low sociability ^d
			Ultrasonic vocalizations increased in pups and reduced in adults ^d
			Impaired reversal learning ^d

^aSpencer et al. (2011)

^bMoy et al. (2009)

^cMoretti et al. (2005)

^dNakatani et al. (2009)

placed in the empty center chamber to habituate to the novelty of the environment (shown in Fig. 1). After the 10-min habituation session, the subject mouse is returned to the center chamber, while the targets are placed in the left and right side chambers. A novel object is placed in one side chamber. The novel object is usually an inverted wire pencil cup that elicits considerable exploration and sniffing by the subject mouse. A novel mouse is placed in the other side chamber, inside in a wire cup that permits visual, olfactory, auditory, and

some tactile contact while preventing aggressive or sexual interactions. The number of seconds spent in each chamber, and the number of entries between chambers, is automatically recorded by the software detection of photocell beam breaks in the partitions between the compartments. Sociability in this task is defined as the subject mouse spending more time in the side chamber containing the novel mouse than in the side chamber containing the novel object. Equal or less time spent with the novel object as compared to the

Animal Models, Table 3 Selected examples of mouse and rat models used to test hypotheses about environmental factors implicated in autism

Rodent model	Autism-relevant behavioral phenotypes
Mice with a heterozygous mutation in the murine tuberous sclerosis 2 (<i>Tsc2</i>) gene exposed to an immunostimulant compound during gestation Offspring of rats and mice subjected to immune system challenges during pregnancy	Low sociability ^a
	Reduced reciprocal social interactions ^b
	Reduced ultrasonic vocalizations ^b
	Increased motor stereotypies ^b
Rats and mice prenatally exposed to the antiepileptic drug valproic acid	Reduced reciprocal social interactions ^c
	Increased motor stereotypies ^c

^aEhninger et al. (2010)

^bPatterson (2009)

^cDufour-Rainfray et al. (2011)

novel mouse is interpreted as the absence of sociability in this task. Mice investigate novel conspecifics by sniffing. Thus, to determine whether time spent in the chamber containing the novel mouse reflects true social interactions versus nonsocial exploration of the chamber, a human observer scores, from videotapes of the test session, the amount of time the subject mouse spends sniffing the wire cup containing the novel mouse. Investigating the novel object instead of the novel mouse may be analogous to the tendency of autistic individuals to engage in nonsocial activities such as playing with one toy for an extended period of time or to spend more time visually examining geometric patterns as compared to social images (Frith 2003; Pierce et al. 2011).

The partition test (Spencer et al. 2011) can be used to evaluate social interest as well as basic social recognition. A subject mouse is placed in one side of a standard cage divided in half by a perforated partition made of clear plastic or wire and a partner mouse in the opposite side. The subject mouse can see, hear, and smell the partner mouse, but cannot engage in physical interactions with the partner. Approaches to and time spent at the partition by the subject mouse represent the amount of interest in the social partner. Social

preference and social memory can be evaluated through sequential presentation of different social partners.

To more fully assess the complexity and variability of social behaviors in mice, more fine-grained analyses of reciprocal social interactions can be conducted in freely moving dyads of mice. Behaviors exhibited by two unfamiliar age-matched rats or mice can be detected with automated video-tracking equipment or scored by a human observer. A variety of parameters can be scored depending on the age and sex of the animals, including nose-to-nose sniffing, nose-to-anogenital sniffing, body sniffing, following, pushing past each other with physical contact, crawling over and under each other, chasing, mounting, and wrestling (Bolivar et al. 2007; McFarlane et al. 2008; Terranova and Laviola 2001). Nonsocial behaviors such as self-grooming, repetitive digging in the bedding, and arena exploration are simultaneously scored. Subject animals can be tested at different ages and over repeated test sessions to evaluate trajectories of complex social behaviors across different neurodevelopmental stages. A juvenile play apparatus for scoring reciprocal social interactions in 21-day-old mice is shown in Fig. 2.

The visible burrow system can be used to evaluate social interactions among adult mice in a context that provides many features of rodents' natural habitats, including multiple burrows connected via tunnels to a larger open area (Arakawa et al. 2007). Behaviors displayed in the visible burrow system can be videotaped and scored later by a human observer. Social behaviors such as huddling, chasing, following, and mounting can be scored along with nonsocial behaviors such as self-grooming and fleeing from another animal (Arakawa et al. 2007; Pobbe et al. 2010). Food and water can be provided in the visible burrow system to allow observation of social behaviors at different times of day over several consecutive days or weeks.

Manual scoring of rodent social behaviors requires highly trained human observers, is often time-consuming and is subject to observer bias. A growing number of video-tracking software systems are becoming available to automate

Animal Models, Table 4 Examples of genetically homogeneous inbred mouse strains that display behavioral phenotypes relevant to the diagnostic symptoms of autism

Inbred strain	Autism-relevant behavioral phenotypes
A/J	Low sociability ^{a, b, c}
	Reduced reciprocal social interactions ^d
	Impaired reversal learning ^c
BALB/cJ, BALB/cByJ	Low sociability ^c
	Reduced reciprocal social interactions ^c
	Reduced ultrasonic vocalizations ^c
BTBR T + tf/J	Reduced reciprocal social interactions ^{d, f, g}
	Low sociability ^f
	Increased repetitive self-grooming ^f
	Ultrasonic vocalizations elevated in pups and reduced in adults ^{h, i}
	Unusual repertoire of ultrasonic vocalization call categories as pups and adults ^{h, i}
	Impaired social transmission of food preference ^f
	Impaired reversal learning ^c
	Preference for specific unfamiliar objects and repetitive object exploration patterns ^j
C58/J	Low sociability ^k
	Impaired social transmission of food preference ^k
	High level of repetitive self-grooming and motor stereotypies ^k
NZB/B1NJ	Low sociability ^l
	Impaired reversal learning ^l
129 S1/SvImJ	Low sociability ^l
	Lack of preference for social novelty ^l
	Impaired reversal learning ^k

^aBrodkin et al. (2004)

^bMoy et al. (2004)

^cMoy et al. (2007)

^dBolivar et al. (2007)

^ePanksepp et al. (2007)

^fMcFarlane et al. (2008)

^gDefensor et al. (2011)

^hScattoni et al. (2008)

ⁱScattoni et al. (2011)

^jPearson et al. (2010)

^kRyan et al. (2010)

^lMoy et al. (2008b)

scoring of social behaviors in rodents. Several different software programs have been shown to be reasonably accurate for quantifying social approach behaviors in mouse models of autism using the three-chambered apparatus (e.g., Nadler et al. 2004; Page et al. 2009). Use of more sophisticated software packages to automatically score reciprocal social interactions between pairs of animals is also on the rise (Ahern et al. 2009; Searce-Levie et al. 2008). However, the degree to which these programs accurately track multiple animals has not yet been systematically evaluated,

or has their ability to capture the subtleties inherent to the rodent social behavior repertoire. If their accuracy can be verified, use of automated software programs with standardized quantification methods may allow higher-throughput scoring of rodent social behaviors while improving the chances of reproducibility of results across labs.

Social preference tests can be used to evaluate components of social affiliation, social recognition, and social memory in rodents. In these tests, the subject animal is offered a choice between partners, and time spent with each partner is

measured. In partner preference tests, two stimulus animals with different characteristics (e.g., different strain, familiar versus unfamiliar) are presented simultaneously. The time spent with and number of approaches to each stimulus animal can then be recorded and used to calculate a preference score (Williams et al. 1992). Partner preference tests are often conducted in a Y-maze apparatus where freely moving subject mice spend time with tethered target mice in three cages connected by tunnels (e.g., Lim et al. 2004; Winslow et al. 1993). The three-chambered social approach apparatus (shown in Fig. 1) has been used to investigate preference for social

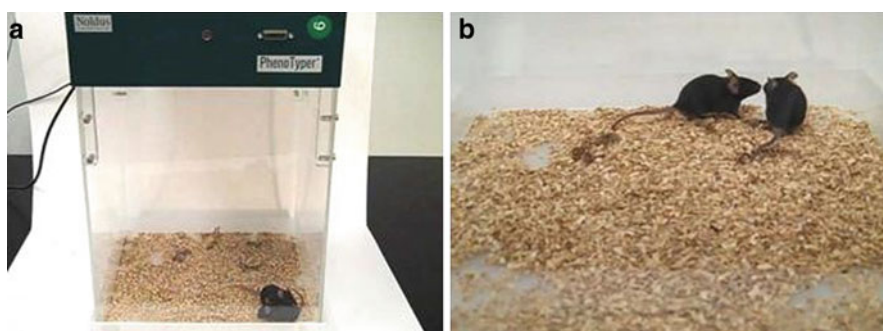


Animal Models, Fig. 1 Three-chambered social approach apparatus used to evaluate sociability and preference for social novelty in mice. (Photograph contributed by Dr. Mu Yang, Laboratory of Behavioral Neuroscience, NIMH)

novelty in mice (DeLorey et al. 2008; Moy et al. 2004, 2009). Preference for social novelty is defined as the subject mouse spending more time in a chamber or in physical contact with a novel mouse in one side chamber than with a familiar mouse in the other side chamber. Mice usually habituate quickly to the presence of a novel conspecific and will move on to approach and investigate another novel conspecific when it is presented. During social approach testing as described above, the subject mouse becomes habituated to the novel mouse. The subject mouse can then be provided access to a second unfamiliar novel mouse, and time spent with the first versus second novel mouse can then be recorded. Partners can also be presented sequentially, with time delays between presentations, to evaluate social recognition memory (Winslow and Insel 2002). A lack of normal preference for a novel social partner or deficits in social recognition may be analogous to the tendency of autistic individuals to avoid unfamiliar individuals or to indiscriminately approach strangers (American Psychiatric Association 1994).

Communication

The second DSM-IV criterion for autism, qualitative impairments in communication (American Psychiatric Association 1994; Frith 2003; Lord et al. 2000), is perhaps the most challenging to model in rodents. The nature of mouse communication is not yet well understood, although considerable interest has recently focused on



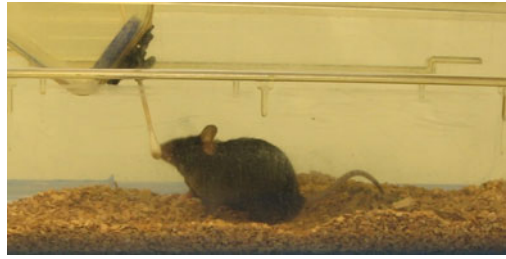
Animal Models, Fig. 2 (a) Noldus PhenoTyper 3000 apparatus for scoring reciprocal social interactions between pairs of age-matched unfamiliar mice. (b) Two

juvenile C57BL/6/J mice engaged in nose-to-nose sniffing. (Photographs contributed by Dr. Mu Yang, Laboratory of Behavioral Neuroscience, NIMH)

ultrasonic vocalizations (Lahvis et al. 2011; Scattoni et al. 2009). Olfaction is the primary sense used by rats and mice for individual recognition and is likely of central importance in rodent communication (Brennan and Kendrick 2006). Information between members of the same species is conveyed using chemical signals commonly termed pheromones. In addition to pheromonal communication, rats and mice emit ultrasonic vocalizations in different social contexts throughout the lifespan (Lahvis et al. 2011; Scattoni et al. 2009). Several behavioral tasks involving the evaluation of responses to olfactory and auditory cues can be used to assay possible communication deficits in rodents.

Tasks designed to assay olfactory communication in rodents include social transmission of food preference, olfactory habituation/dishabituation to social odors, and scent marking. The social transmission of food preference test is a three-stage process. First, a “demonstrator” animal is allowed to eat a novel-flavored food. After consuming the novel food, the demonstrator interacts with an “observer” animal. During this time, the observer animal acquires familiarity with the novel flavor, presumably by sniffing the face, breath, and whiskers of the demonstrator animal. In the final phase, the observer is given a choice between the flavor of the food eaten by the demonstrator and some other novel flavor. The observer animal will express a preference for the now-familiar food as indicated by eating more of it. Normal performance on this task is thought to depend on the observer animal detecting olfactory cues on the breath of the demonstrator, which requires social interactions, particularly nose-to-nose sniffing (Galef and Wigmore 1983; Wrenn 2004).

The olfactory habituation/dishabituation task (shown in Fig. 3) measures the ability to detect and discriminate between different odors. When mice are presented with a cotton swab containing a novel odor, they will investigate it by sniffing. Upon repeated presentations of the same odor, a progressive decrease in sniffing (olfactory habituation), will be seen. Reinstatement of high sniffing levels (dishabituation) will be seen when a novel odor stimulus is subsequently introduced (Ryan



Animal Models, Fig. 3 Olfactory habituation/dishabituation test, showing a mouse sniffing a cotton swab saturated with odors from an unfamiliar mouse. (Photograph contributed by the authors)

et al. 2010; Yang and Crawley 2009). Fresh urine or swipes from the bottom of a soiled cage of unfamiliar mice can be used as social odors. The shapes of the habituation and dishabituation curves reflect the ability to discriminate between same and different odors. The peaks of the curves reflect the level of interest in each odor stimulus. Social odors elicit considerably higher levels of sniffing as compared to nonsocial odors, such as almond and banana extracts (Yang and Crawley 2009).

Olfactory cues influence a variety of social behaviors in rodents, such as kin and individual recognition, bond formation, mate attraction and selection, and communication of danger (Arakawa et al. 2008; Brennan and Kendrick 2006; Hurst 1990). Scent-marking tasks are widely used in mice (Arakawa et al. 2008; Wöhr et al. 2011). Mice deposit urinary steroidal pheromones that serve as territorial scent marks and are distinct among genetically diverse individuals (Brennan and Kendrick 2006). High levels of interest in urinary scents from other mice are indicated by the tendency of a subject mouse to explore the anogenital area of a novel mouse, investigate urinary scent marks in a cage, and sniff a cotton swab soaked in urine from another mouse. When a male mouse encounters a scent mark deposited by another male in its territory, it tends to countermark in response. Countermarking is gradually reduced when a male mouse is repeatedly exposed to scent marks from the same mouse and is increased again when the subject mouse encounters scent marks from a

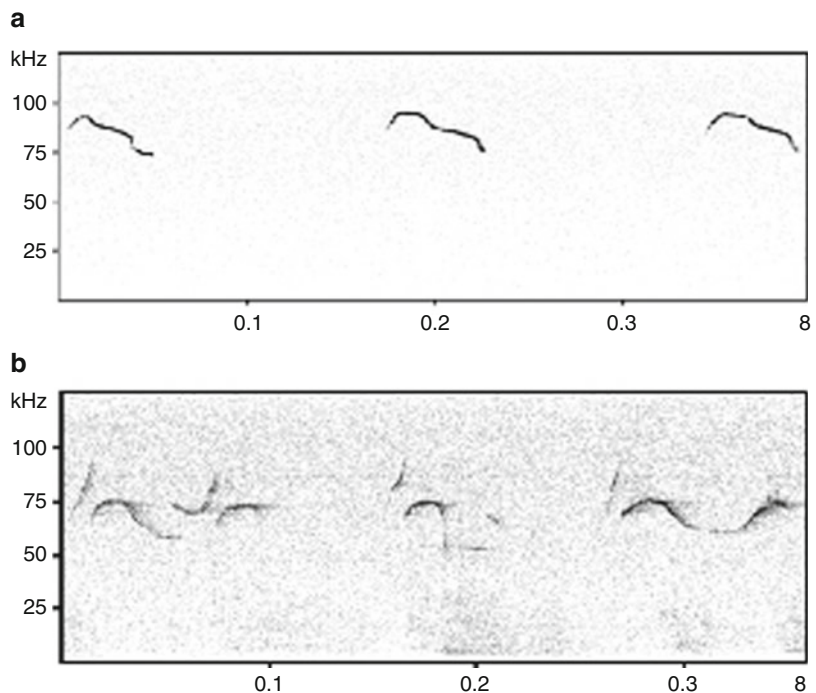
novel, genetically different mouse (Arakawa et al. 2008). Thus, countermarking behavior might be useful for studying the ability to discriminate between different individuals based on olfactory cues. Male mice also deposit scent marks when exposed to urine from a female mouse (Wöhr et al. 2011). Female urine-elicited scent marking is thought to play a role in mate attraction and could serve as a measure of social motivation (Hurst 1990; Wöhr et al. 2011). The importance of olfactory cues across many social contexts suggests that rodent models of autism displaying olfactory communication deficits might be useful for understanding aspects of impaired social communication in autism.

Emission of ultrasonic vocalizations (USVs) in social situations is a consistent and robust phenomenon in rodents. These USVs can be detected using sensitive ultrasonic microphones and recorded using specialized software. Quantitative and qualitative analysis of USVs emitted by mice have been used to examine possible autism-relevant communication deficits in both inbred strains and various genetic mutant mouse lines.

When separated from the nest, mouse pups emit calls that parents use to locate and retrieve the pup (Nakatani et al. 2009; Scattoni et al. 2008; Shu et al. 2005; Winslow and Insel 2002). USVs are also emitted during juvenile interactions, by resident females in a resident-intruder task and by males exposed to a female in estrus or their urine (Bozdagi et al. 2010; Panksepp et al. 2007; Wöhr et al. 2011). Analysis of USV spectrograms (shown in Fig. 4) has allowed researchers to identify discrete categories of ultrasonic calls in mice (e.g., Panksepp et al. 2007; Scattoni et al. 2008; Scattoni et al. 2011). Simultaneous recording of social interactions and USVs have revealed correlations between call emission rates, types of calls emitted, and various social behaviors, suggesting that USVs might convey communicative information during social situations (Panksepp et al. 2007; Scattoni et al. 2011). However, much work remains to be done in order to determine the potential communicative value of rodent USVs and their relevance to the types of communication impairments seen in autistic individuals.

Animal Models,

Fig. 4 Spectrograms of ultrasonic vocalizations emitted by (a) a C57BL/6 J mouse pup separated from the nest and (b) an adult C57BL/6 J male mouse interacting with an unfamiliar C57BL/6 J female mouse in estrus. (Spectrograms contributed by the authors)



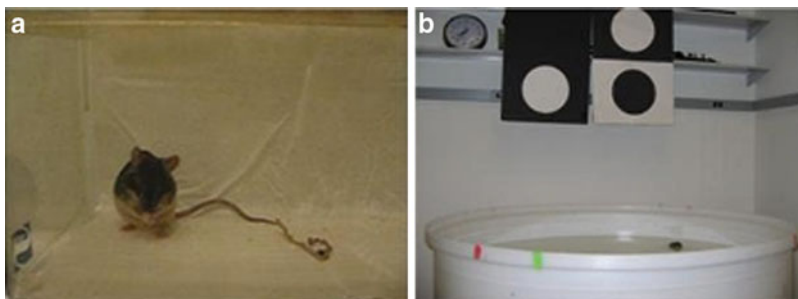
Repetitive Behaviors and Resistance to Change in Routine

Several assays are available to investigate behavioral phenotypes in rodents relevant to the third DSM-IV diagnostic criterion of autism, stereotyped, repetitive behaviors, and patterns with restricted interests or activities (American Psychiatric Association 1994; Lord et al. 2000). Rats and mice display spontaneous motor stereotypies that appear to have no specific function, including circling, back flipping, jumping, and cage bar biting (DeLorey et al. 2008; Hines et al. 2008; Lewis et al. 2007; Ryan et al. 2010). Repetitive behaviors in rodents, which may appear as normal patterns but persist for unusually long periods of time, include self-grooming (shown in Fig. 5a) and marble burying (McFarlane et al. 2008; Ryan et al. 2010; Spencer et al. 2011). Resistance to change has been modeled in rodents using reversal learning tasks, which measure perseverative behavior patterns (Moy et al. 2007, 2008b; Nakatani et al. 2009). Reversal learning tasks measure the flexibility of the animal to switch from an established habit to a new habit. Animals are first well-trained to form a spatial position habit, for example, by placing a food reward in the left arm of a standard T-maze or by placing the escape platform into one quadrant of the Morris water maze (shown in Fig. 5b). The location of the food reward or escape platform is then changed, requiring the development of a new position habit. Successful acquisition of the initial position habit but failure to develop the new one might be analogous to insistence on sameness and inflexibility in routines that

is characteristic of autism (Moy et al. 2008b; Nakatani et al. 2009). Tasks relevant to restricted interests or activities are still under development. One approach measures restricted exploration of only one of the available holes in a hole board (Moy et al. 2008a) or only one of several novel objects in an open field (Pearson et al. 2010).

Associated Symptoms

Additional associated symptoms, which occur in some cases of autism, include intellectual impairments, anxiety, sleep disturbances, aggression, clumsiness, idiosyncratic responses to sensory stimuli, and seizures (Dawson et al. 2002; Lord et al. 2000; Piven et al. 1997). In order to more fully characterize a proposed animal model of autism, it is useful to include behavioral tasks which address phenotypes relevant to these associated symptoms (reviewed in Crawley 2007a, 2007b). Standard tasks available for rats and mice are well-characterized in the behavioral neuroscience literature. Learning and memory tasks (e.g., Morris water maze, contextual and cued fear conditioning, novel object recognition) can be used to detect cognitive deficits that may be relevant to the symptom of mental retardation. Tasks used to assay anxiety-related behaviors (e.g., elevated plus maze, light $\leftrightarrow$ dark exploration) can be used to detect high or low levels of anxiety in an animal model. Disturbances in sleep patterns can be evaluated using electroencephalography (EEG) recordings, circadian running wheels, and home cage monitoring systems. Resident-intruder tasks can be used to measure aggressive behavior



Animal Models, Fig. 5 (a) A BTBR T + tf/J mouse engaged in repetitive self-grooming. Photograph contributed by Dr. Mu Yang, Laboratory of Behavioral Neuroscience,

NIMH. (b) Morris water maze for measuring reversal learning, which evaluates resistance to change an established position habit. (Photograph contributed by the authors)

in males. Motor clumsiness can be tested using the balance beam, rotarod, and footprint tests. Sensory hypersensitivity or hyposensitivity can be detected through the acoustic and tactile startle tests, as well as tests that measure pain sensitivity (e.g., hot plate, tail flick). Spontaneous seizures, audiogenic seizures induced by loud tones, or drug-induced seizures induced by administration of convulsants can be measured using observer scoring or EEG recordings. A potential pitfall of detecting phenotypes relevant to the associated symptoms of autism is that they may complicate interpretation of phenotypes directly relevant to a diagnostic symptom. For example, a mutant mouse line with high-anxiety-like behavior would likely display low levels of exploratory activity in the three-chambered social approach task, confounding interpretation of their social approach behavior. This issue requires careful consideration for each animal model in which autism-relevant behavioral phenotypes have been detected.

Control Parameters

When investigating autism-relevant behavioral phenotypes in animal models of ASDs, it is essential to control for physical disabilities that could produce false positives in many of the behavioral tasks described here (Crawley 2007a). For example, a mutant mouse line with a gene mutation affecting olfactory functions could show deficits on social tasks based on successful detection of conspecific odors. Similarly, rats or mice treated with a drug that produces sedation will likely show impairments in social, cognitive, or motor tasks that are attributable to low overall activity as opposed to a reduction in reciprocal social interactions or a learning deficit. To rule out these types of artifacts, potential rodent models of autism must be evaluated on a series of tasks measuring general health, neurological reflexes, sensory abilities, motor functions, and home cage behaviors (Crawley 2007b).

Future Directions

Autism is a complex disorder with variable symptoms, some of which may be uniquely human. For

example, deficits in theory of mind, or the ability to intuit what another person is thinking or feeling, may be difficult to model in nonhuman animals. However, recent reports suggest that mice display empathy-like behaviors following exposure to cagemates who have experienced a painful stimulus (e.g., Chen et al. 2009). Subtle language and communication deficits, such as the inability to understand humor or sarcasm, are unlikely to be successfully modeled in animals. However, detailed analysis of rodent ultrasonic vocalizations may provide information about their communicative value (Lahvis et al. 2011). Modeling complex cognitive abilities, such as executive functions and joint attention, is also a challenge. Researchers are starting to develop cognitive tasks that evaluate sustained attention and attentional set-shifting abilities in rodents similar to those used to evaluate cognitive abilities in autistic individuals (Brigman et al. 2005).

The occurrence of autism is significantly higher in males than in females, with a male to female ratio of 4:1 (Volkmar and Pauls 2003). Thus, an animal model that displays relevant phenotypes in males but not females could be considered to have face validity with regard to the prevalence of ASDs. Due to the higher prevalence of autism in males, many studies have only tested male animals (e.g., Bolivar et al. 2007; Hines et al. 2008; McFarlane et al. 2008; Moy et al. 2007, 2008b; Nakatani et al. 2009; Pearson et al. 2010; Peca et al. 2011), precluding detection of possible sex differences. However, sex differences have been reported for a few animal models of autism. For example, social deficits have been detected in male but not female mice of the inbred C58/J strain (Ryan et al. 2010) and in male but not female rats exposed prenatally to valproic acid (Dufour-Rainfray et al. 2011). Other studies have tested both males and females and detected autism-relevant behavioral phenotypes in both sexes (e.g., Brodtkin et al. 2004; Cheh et al. 2006; Etherton et al. 2009; Moy et al. 2004; Scattoni et al. 2011). Systematic investigations of sex differences in potential animal models of autism will likely lead to a better understanding of the etiology of ASDs.

Despite these challenges, animal models of autism have been useful for evaluating potential

treatments. Early behavioral interventions have been the most effective treatments for the symptoms of autism (Rogers and Vismara 2008). Medications have been reported to improve associated symptoms of autism such as hyperactivity or mood, but have not been shown to affect the diagnostic features of autism. Both pharmacological and behavioral intervention strategies have been tested in mouse models such as the BTBR T + tf/J strain, which displays behavioral phenotypes relevant to all three diagnostic symptoms of autism (Bolivar et al. 2007; McFarlane et al. 2008; Pearson et al. 2010; Scattoni et al. 2008, 2011). A single injection of the drug 2-methyl-6-(phenylethynyl)pyridine (MPEP), a potent antagonist at mGluR5 subtype glutamate receptors, significantly reduced repetitive self-grooming in BTBR mice (Silverman et al. 2010). Rearing BTBR mice with social C57BL/6J mice as cagemates after weaning significantly improved their sociability in the three-chambered social approach task as adults (Yang et al. 2011a). Several potential drug candidates, such as other mGluR5 antagonists, rapamycin, brain-derived neurotrophic factor (BDNF), and oxytocin, have been shown to prevent or reverse aberrant phenotypes in a variety of mouse models of ASDs (reviewed in Silverman et al. 2010). Animal models with robust and well-replicated behavioral phenotypes will be powerful tools for developing pharmacological and behavioral treatments for autism.

See Also

- [Broader Autism Phenotype](#)
- [Copy Number Variation](#)
- [Genetics](#)
- [Neuroscience](#)
- [Social Behaviors and Social Impairment](#)
- [Social Communication](#)
- [Stereotypic Behavior](#)
- [Synaptic Proteins](#)

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References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, 9(5), 341–355.
- Ahern, T. H., Modi, M. E., Burkett, J. P., & Young, L. J. (2009). Evaluation of two automated metrics for analyzing partner preference tests. *Journal of Neuroscience Methods*, 182(2), 180–188.
- American Psychiatric Association, A. P. (Ed.). (1994). *Diagnostic and statistical manual of mental disorders: DSM-IV* (4th ed.). Washington, DC: American Psychiatric Association.
- Arakawa, H., Blanchard, D. C., & Blanchard, R. J. (2007). Colony formation of C57BL/6 J mice in visible burrow system: Identification of eusocial behaviors in a background strain for genetic animal models of autism. *Behavioural Brain Research*, 176(1), 27–39.
- Arakawa, H., Blanchard, D. C., Arakawa, K., Dunlap, C., & Blanchard, R. J. (2008). Scent marking behavior as an odorant communication in mice. *Neuroscience and Biobehavioral Reviews*, 32(7), 1236–1248.
- Bolivar, V. J., Walters, S. R., & Phoenix, J. L. (2007). Assessing autism-like behavior in mice: Variations in social interactions among inbred strains. *Behavioural Brain Research*, 176(1), 21–26.
- Bozdagi, O., Sakurai, T., Papapetrou, D., Wang, X., Dickstein, D. L., Takahashi, N., et al. (2010). Haploinsufficiency of the autism-associated Shank3 gene leads to deficits in synaptic function, social interaction, and social communication. *Molecular Autism*, 1(1), 15.
- Brennan, P. A., & Kendrick, K. M. (2006). Mammalian social odours: Attraction and individual recognition. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, 361(1476), 2061–2078.
- Brigman, J. L., Bussey, T. J., Saksida, L. M., & Rothblat, L. A. (2005). Discrimination of multidimensional visual stimuli by mice: Intra- and extradimensional shifts. *Behavioral Neuroscience*, 119(3), 839–842.
- Brodtkin, E. S., Hagemann, A., Nemetski, S. M., & Silver, L. M. (2004). Social approach-avoidance behavior of inbred mouse strains towards DBA/2 mice. *Brain Research*, 1002(1–2), 151–157.
- Cheh, M. A., Millonig, J. H., Roselli, L. M., Ming, X., Jacobsen, E., Kamdar, S., et al. (2006). En2 knockout mice display neurobehavioral and neurochemical alterations relevant to autism spectrum disorder. *Brain Research*, 1116(1), 166–176.
- Chen, Q., Panksepp, J. B., & Lahvis, G. P. (2009). Empathy is moderated by genetic background in mice. *PLoS One*, 4(2), e4387.
- Covington, H. E., Vialou, V., & Nestler, E. J. (2010). From synapse to nucleus: Novel targets for treating depression. *Neuropharmacology*, 58(4–5), 683–693.
- Crawley, J. N. (2007a). Mouse behavioral assays relevant to the symptoms of autism. *Brain Pathology*, 17(4), 448–459.

- Crawley, J. N. (2007b). *What's wrong with my mouse? Behavioral phenotyping of transgenic and knockout mice*. Hoboken: Wiley.
- Dawson, G., Webb, S., Schellenberg, G. D., Dager, S., Friedman, S., Aylward, E., et al. (2002). Defining the broader phenotype of autism: Genetic, brain, and behavioral perspectives. *Development and Psychopathology*, 14(3), 581–611.
- Defensor, E. B., Pearson, B. L., Pobbe, R. L., Bolivar, V. J., Blanchard, D. C., & Blanchard, R. J. (2011). A novel social proximity test suggests patterns of social avoidance and gaze aversion-like behavior in BTBR T+tf/J mice. *Behavioural Brain Research*, 217(2), 302–308.
- DeLorey, T. M., Sahbaie, P., Hashemi, E., Homanics, G. E., & Clark, J. D. (2008). Gabrb3 gene deficient mice exhibit impaired social and exploratory behaviors, deficits in non-selective attention and hypoplasia of cerebellar vermal lobules: A potential model of autism spectrum disorder. *Behavioural Brain Research*, 187(2), 207–220.
- Dufour-Rainfray, D., Vourc'h, P., Tourlet, S., Guilleateau, D., Chalon, S., & Andres, C. R. (2011). Fetal exposure to teratogens: Evidence of genes involved in autism. *Neuroscience and Biobehavioral Reviews*, 35(5), 1254–1265.
- Ehninger, D., Sano, Y., de Vries, P. J., Dies, K., Franz, D., Geschwind, D. H., et al. (2010). Gestational immune activation and Tsc2 haploinsufficiency cooperate to disrupt fetal survival and may perturb social behavior in adult mice. *Molecular Psychiatry*, 16, 2010. Advance online publication.
- Etherton, M. R., Blaiss, C. A., Powell, C. M., & Sudhof, T. C. (2009). Mouse neurexin-1 α deletion causes correlated electrophysiological and behavioral changes consistent with cognitive impairments. *Proceedings of the National Academy of Sciences USA*, 106(42), 17998–18003.
- Frith, U. (2003). *Autism: Explaining the enigma* (2nd ed.). Malden: Blackwell Publishing.
- Galef, J. B. G., & Wigmore, S. W. (1983). Transfer of information concerning distant foods: A laboratory investigation of the “information-centre” hypothesis. *Animal Behaviour*, 31(3), 748–758.
- Higgins, G. A., & Jacobsen, H. (2003). Transgenic mouse models of Alzheimer's disease: Phenotype and application. *Behavioral Pharmacology*, 14(5–6), 419–438.
- Hines, R. M., Wu, L., Hines, D. J., Steenland, H., Mansour, S., Dahlhaus, R., et al. (2008). Synaptic imbalance, stereotypies, and impaired social interactions in mice with altered neuroligin 2 expression. *Journal of Neuroscience*, 28(24), 6055–6067.
- Hurst, J. L. (1990). Urine marking in populations of wild house mice *Mus domesticus* Ratty. III. Communication between the sexes. *Animal Behaviour*, 40(2), 233–243.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2(3), 217–250.
- Kwon, C.-H., Luikart, B. W., Powell, C. M., Zhou, J., Matheny, S. A., Zhang, W., et al. (2006). Pten regulates neuronal arborization and social interaction in mice. *Neuron*, 50(3), 377–388.
- Lahvis, G. P., Allewa, E., & Scattoni, M. L. (2011). Translating mouse vocalizations: Prosody and frequency modulation. *Genes, Brain, and Behavior*, 10(1), 4–16.
- Lewis, M. H., Tanimura, Y., Lee, L. W., & Bodfish, J. W. (2007). Animal models of restricted repetitive behavior in autism. *Behavioural Brain Research*, 176(1), 66–74.
- Lim, M. M., Wang, Z., Olazabal, D. E., Ren, X., Terwilliger, E. F., & Young, L. J. (2004). Enhanced partner preference in a promiscuous species by manipulating the expression of a single gene. *Nature*, 429(6993), 754–757.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Leventhal, B. L., DiLavore, P. C., et al. (2000). The autism diagnostic observation schedule-generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30(3), 205–223.
- McFarlane, H. G., Kusek, G. K., Yang, M., Phoenix, J. L., Bolivar, V. J., & Crawley, J. N. (2008). Autism-like behavioral phenotypes in BTBR T + tf/J mice. *Genes, Brain, and Behavior*, 7(2), 152–163.
- Miczek, K. A., Maxson, S. C., Fish, E. W., & Faccidomo, S. (2001). Aggressive behavioral phenotypes in mice. *Behavioural Brain Research*, 125(1–2), 167–181.
- Moore, H. (2010). The role of rodent models in the discovery of new treatments for schizophrenia: Updating our strategy. *Schizophrenia Bulletin*, 36(6), 1066–1072.
- Moretti, P., Bouwknecht, J. A., Teague, R., Paylor, R., & Zoghbi, H. Y. (2005). Abnormalities of social interactions and home-cage behavior in a mouse model of Rett syndrome. *Human Molecular Genetics*, 14(2), 205–220.
- Moy, S. S., Nadler, J. J., Perez, A., Barbaro, R. P., Johns, J. M., Magnuson, T. R., et al. (2004). Sociability and preference for social novelty in five inbred strains: An approach to assess autistic-like behavior in mice. *Genes, Brain, and Behavior*, 3(5), 287–302.
- Moy, S. S., Nadler, J. J., Young, N. B., Perez, A., Holloway, L. P., Barbaro, R. P., et al. (2007). Mouse behavioral tasks relevant to autism: Phenotypes of 10 inbred strains. *Behavioural Brain Research*, 176(1), 4–20.
- Moy, S. S., Nadler, J. J., Poe, M. D., Nonneman, R. J., Young, N. B., Koller, B. H., et al. (2008a). Development of a mouse test for repetitive, restricted behaviors: Relevance to autism. *Behavioral Brain Research*, 188(1), 178–194.
- Moy, S. S., Nadler, J. J., Young, N. B., Nonneman, R. J., Segall, S. K., Andrade, G. M., et al. (2008b). Social approach and repetitive behavior in eleven inbred mouse strains. *Behavioural Brain Research*, 191(1), 118–129.
- Moy, S. S., Nadler, J. J., Young, N. B., Nonneman, R. J., Grossman, A. W., Murphy, D. L., et al. (2009). Social approach in genetically engineered mouse lines relevant to autism. *Genes, Brain, and Behavior*, 8(2), 129–142.
- Nadler, J. J., Moy, S. S., Dold, G., Trang, D., Simmons, N., Perez, A., et al. (2004). Automated apparatus for

- quantitation of social approach behaviors in mice. *Genes, Brain, and Behavior*, 3(5), 303–314.
- Nakatani, J., Tamada, K., Hatanaka, F., Ise, S., Ohta, H., Inoue, K., et al. (2009). Abnormal behavior in a chromosome-engineered mouse model for human 15q11-13 duplication seen in autism. *Cell*, 137(7), 1235–1246.
- Page, D. T., Kuti, O. J., & Sur, M. (2009). Computerized assessment of social approach behavior in mouse. *Frontiers in Behavioral Neuroscience*, 3, 48.
- Panksepp, J. B., Jochman, K. A., Kim, J. U., Koy, J. J., Wilson, E. D., Chen, Q., et al. (2007). Affiliative behavior, ultrasonic communication and social reward are influenced by genetic variation in adolescent mice. *PLoS One*, 2(4), e351.
- Patterson, P. H. (2009). Immune involvement in schizophrenia and autism: Etiology, pathology and animal models. *Behavioural Brain Research*, 204(2), 313–321.
- Pearson, B. L., Pobbe, R. L. H., Defensor, E. B., Oasay, L., Bolivar, V. J., Blanchard, D. C., et al. (2010). Motor and cognitive stereotypies in the BTBR T + tf/J mouse model of autism. *Genes, Brain, and Behavior*, 10(2), 228–235.
- Peca, J., Feliciano, C., Ting, J. T., Wang, W., Wells, M. F., Venkatraman, T. N., et al. (2011). Shank3 mutant mice display autistic-like behaviours and striatal dysfunction. *Nature*, 472(7344), 437–442.
- Pierce, K., Conant, D., Hazin, R., Stoner, R., & Desmond, J. (2011). Preference for geometric patterns early in life as a risk factor for autism. *Archives of General Psychiatry*, 68(1), 101–109.
- Piven, J., Palmer, P., Jacobi, D., Childress, D., & Arndt, S. (1997). Broader autism phenotype: Evidence from a family history study of multiple-incidence autism families. *The American Journal of Psychiatry*, 154(2), 185–190.
- Pobbe, R. L. H., Pearson, B. L., Defensor, E. B., Bolivar, V. J., Blanchard, D. C., & Blanchard, R. J. (2010). Expression of social behaviors of C57BL/6 J versus BTBR inbred mouse strains in the visible burrow system. *Behavioural Brain Research*, 214(2), 443–449.
- Rogers, S. J., & Vismara, L. A. (2008). Evidence-based comprehensive treatments for early autism. *Journal of Clinical Child and Adolescent Psychology*, 37(1), 8–38.
- Ryan, B. C., Young, N. B., Crawley, J. N., Bodfish, J. W., & Moy, S. S. (2010). Social deficits, stereotypy and early emergence of repetitive behavior in the C58/J inbred mouse strain. *Behavioural Brain Research*, 208(1), 178–188.
- Scattoni, M. L., Gandhi, S. U., Ricceri, L., & Crawley, J. N. (2008). Unusual repertoire of vocalizations in the BTBR T + tf/J mouse model of autism. *PLoS One*, 3(8), e3067.
- Scattoni, M. L., Crawley, J., & Ricceri, L. (2009). Ultrasonic vocalizations: A tool for behavioural phenotyping of mouse models of neurodevelopmental disorders. *Neuroscience and Biobehavioral Reviews*, 33(4), 508–515.
- Scattoni, M. L., Ricceri, L., & Crawley, J. N. (2011). Unusual repertoire of vocalizations in adult BTBR T + tf/J mice during three types of social encounters. *Genes, Brain, and Behavior*, 10(1), 44–56.
- Scearce-Levie, K., Roberson, E. D., Gerstein, H., Cholfin, J. A., Mandiyan, V. S., Shah, N. M., et al. (2008). Abnormal social behaviors in mice lacking Fgf17. *Genes, Brain, and Behavior*, 7(3), 344–354.
- Shu, W., Cho, J. Y., Jiang, Y., Zhang, M., Weisz, D., Elder, G. A., et al. (2005). Altered ultrasonic vocalization in mice with a disruption in the Foxp2 gene. *Proceedings of the National Academy of Sciences USA*, 102(27), 9643–9648.
- Silverman, J. L., Tolu, S. S., Barkan, C. L., & Crawley, J. N. (2010). Repetitive self-grooming behavior in the BTBR mouse model of autism is blocked by the mGluR5 antagonist MPEP. *Neuropsychopharmacology*, 35(4), 976–989.
- Spencer, C. M., Alekseyenko, O., Hamilton, S. M., Thomas, A. M., Serysheva, E., Yuva-Paylor, L. A., et al. (2011). Modifying behavioral phenotypes in Fmr1KO mice: Genetic background differences reveal autistic-like responses. *Autism Research*, 4(1), 40–56.
- Terranova, M. L., & Laviola, G. (2001). Scoring of social interactions and play in mice during adolescence. *Current Protocols in Toxicology*, 26, 13.10.1–13.10.11.
- Volkmar, F. R., & Pauls, D. (2003). Autism. *Lancet*, 362(9390), 1133–1141.
- Williams, J. R., Catania, K. C., & Carter, C. S. (1992). Development of partner preferences in female prairie voles (*Microtus ochrogaster*): The role of social and sexual experience. *Hormones and Behavior*, 26(3), 339–349.
- Winslow, J. T., & Insel, T. R. (2002). The social deficits of the oxytocin knockout mouse. *Neuropeptides*, 36(2–3), 221–229.
- Winslow, J. T., Hastings, N., Carter, C. S., Harbaugh, C. R., & Insel, T. R. (1993). A role for central vasopressin in pair bonding in monogamous prairie voles. *Nature*, 365(6446), 545–548.
- Wöhr, M., Roulet, F. I., & Crawley, J. N. (2011). Reduced scent marking and ultrasonic vocalizations in the BTBR T + tf/J mouse model of autism. *Genes, Brain, and Behavior*, 10(1), 35–43.
- Wrenn, C. C. (2004). Social transmission of food preference in mice. *Current Protocols in Neuroscience*, 8, 5 G.1–8.5 G.7.
- Yang, M., & Crawley, J. N. (2009). Simple behavioral assessment of mouse olfaction. *Current Protocols in Neuroscience*, 48, 8.24.1–8.24.12.
- Yang, M., Perry, K., Weber, M. D., Katz, A. M., & Crawley, J. N. (2011a). Social peers rescue autism-relevant sociability deficits in adolescent mice. *Autism Research*, 4(1), 17–27.
- Yang, M., Silverman, J. L., & Crawley, J. L. (2011b). Automated three-chambered social approach task for mice. *Current Protocols in Neuroscience*, 8, 26.1–8.26.16.

Anime, Manga, and the Etiology of Autism

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Historical Overview

The origins of Anime and Manga are difficult to pinpoint. According to some historians, the earliest form of sequential art in Japan emerged in twelfth-century Buddhist scrolls, the most famous of which were created by a monk named Bishop Toba (Brenner 2007). These scrolls were originally created to express religious messages and folklorish narratives. Largely unknown by the general body of society, they soon made their way into popular culture. This form of art went through a series of changes over the next few centuries, eventually entering the scope of entertainment. Illustrators sought to present cartoons in the same fashion, leading to the birth of modern day comics.

The term “Manga” was coined by artist Hokusai Katsuhika in the nineteenth century, which literally means “whimsical pictures” or “sketches.” Bound books with intricate story-lines were soon produced and sold by the thousands. However, the content within the books was considered to be adult in nature and was therefore banned by the Tokugawa regime (Horbinski 2015). The era of modern Anime and Manga would soon be ushered in by the forces of globalization. With Japan’s historically isolated nature dwindling, the advent of modern technology would completely change the trajectory of Anime and Manga.

The arrival of US Commodore Matthew Perry on Japanese shores catalyzed drastic changes in Japan, with a brutal internal struggle between traditional isolationists and those who embraced the West overtaking the entire country. Eventually, Japan would succumb to the influx of the Western way of living, struggling to update its society with technological advances among other

changes. Japanese artists were fascinated by Western forms of art, and eventually created a hybrid that took the form of modern day Manga magazines. The initial forms of Manga magazines were either politically oriented or meant for children. By the middle of the twentieth century, a new genre emerged. Adult Manga comics captivated the entire industry, with explicit representations of crime, sex, and violence. As a result, the more innocent renditions of Manga comics lost business and dwindled in their prevalence.

The development of Anime and Manga snowballed quickly, with comics progressing into television shows, movies, and trading cards. The most significant changes, however, were arguably seen in the fanbase of the genre. Previously aimed at a politically inclined audience as well as children, Anime and Manga morphed into something of a lifestyle for its fans – of all ages, especially adults. One interesting phenomenon in particular is that of cosplay, a play on the words “costume” and “play.” The past few decades have seen cosplay fans gather from all over the world, spending inconceivable monies on intricate costumes and, in some cases, plastic surgery, to resemble their favorite Anime and Manga characters (Winge 2006). These subcommunities of the general Anime genre have experienced significant stigmatization in popular culture.

Among the main factors stigmatizing Anime and Manga fanbase is the reality that the overwhelming majority of amateur Manga artists focus on producing homoerotic and other sexually explicit depictions. As a result, the fanbase became subject to significant controversy in the 1990s (Kinsella 1998). Further, the sheer obsession that Anime and Manga fans hold for their genre of interest is seemingly beyond any other fanbase. With other forms of entertainment typically remaining in the realm of a hobby, Anime and Manga seemed to have a tendency to categorically consume fans. Drastic changes in behavior, dress, speech, lifestyle, spending habits, and other intimately characteristic facets of individuality are not uncommon among fans of Anime and Manga. Anime and Manga fans call themselves “Otaku,” more commonly known with a more general term: “Hikikomori.” Otaku refers to, usually male,

Japanese fans between ages 18 and 40 that fanatically and compulsively consume Anime and Manga products (Azuma 2009). Originally a group of social outcasts bonding over a shared interest, the Otakus now represent a massive portion of the commercial market in Japan.

Of the peculiarities of this demographic is the tendency of psychological uniformity among many of the fans. There exists a surprisingly common set of characteristics among a large portion of Otakus. Researchers have found that there seems to be a surprising association between individuals with autism spectrum disorder (ASD) and the Otaku demographic. While Otakus are often not officially diagnosed as autistic, the culture surrounding Anime and Manga has been called “autism-friendly” (Cowen 2010). The link between Anime, Manga, and ASD begs the question: should this raise any concerns?

Current Research

The affinity that patients with autism spectrum disorder (ASD) have for Anime and Manga products is not an unknown phenomenon to clinical psychologists and therapists. A study carried out with 91 randomized adolescents with ASD found that among the most often visited websites by the subjects fell into the category of anime (Kuo et al. 2013). In fact, this was the second most commonly explored activity among the participants. This is a peculiar pattern, given that individuals with ASD typically exhibit extremely restricted interests (Koegel et al. 2015). High school students with ASD have also been found to gravitate towards only a handful of interests, one of which is the anime fandom (Wolf et al. 2009).

ASD symptoms in adults present in a fairly consistent manner. Common behaviors include preference for social isolation, stunted communication skills, compulsive behavior, difficulty recognizing verbal cues, restriction to either obsession or passiveness towards things, and highly animated approaches to social settings that may include aggression or overly indifferent behavior (*Mayo Clinic*). These behaviors are not alien to the average Otaku. In fact, the

characteristics of Otakus are not much different from the manner in which higher functioning individuals with ASD are described in the West (VanBergeijk 2010).

A study focusing on the Hikikomori (a general term encompassing Otaku) subset of individuals found that about 20% of their Hikikomori patients could be diagnosed with pervasive developmental disorders, or PDD (Tateno et al. 2012). Diagnosis of PDD typically takes place once qualitative impairment in social interaction and restricted and compulsive patterns of behavior and interests are observed in a patient. This is commonly seen in Hikikomori and Otaku. Thus, diagnosis of PDD, especially ASD, should be seriously considered in Hikikomori and Otaku patients. The reality is that ASD is only known by the symptoms exhibited in individuals that have it. There is no biological or genetic factor that has yet been clearly identified by the scientific community (Rozema 2015). This limits our understanding of ASD significantly. Consequently, the etiology of ASD is still unexplored territory. Because the Otaku community heavily exhibits symptoms of autism, a potential environmental etiology must be considered (Vuković 2014).

Future Directions

Why individuals with ASD love Anime and Manga is largely unexplored territory. Robert Rozema mentions that it is estimated that 1 in 68 children fall on the autism spectrum (Rozema 2015). With such a high prevalence, it is important to study the behavioral patterns of this demographic to achieve a firmer understanding of both ASD and the effects of Anime and Manga on a sociological and pathological level. Furthermore, researchers much approach the topic with an open mind, ready to reexamine previously held conceptions about ASD.

The etiology of ASD is still a topic of debate and scarce research. As mentioned, an Otaku is virtually indistinguishable from a high functioning individual with ASD. It is not inconceivable that the captivating tendencies of Anime and Manga produce individuals that operationally

fall on the autism spectrum. In other words, they may not traditionally have ASD as we understand it, but they have grown to develop the same symptoms by way of their fanatical obsession with Anime and Manga products. The umbrella that is ASD, understood to develop in the earliest stages of childhood, must perhaps be expanded to accommodate those adults that develop the same behavioral characteristics later on.

Additionally, the phenomenon of Otaku culture is viewed by many to be a pathological epidemic that is crippling an entire generation. The erosion of social skills, restriction of interests, and displaying of obsessive behaviors are all detriments to the mechanics of day-to-day life. Given the rapidly expanding market for Anime and Manga products as well as the proliferating fanbase, sociologists must allocate efforts towards studying the societal effects of Otaku culture on the development of youth. If the development of the fanbase in the West is towards the same trajectory as Japan, the impacts would be immense in the academic system, the corporate world, the fields of social work and therapy, as well as the structure of society as a whole. The relationship between Anime, Manga, and Autism is one of massive potential in understanding the etiology of ASD and perhaps curbing a looming social epidemic.

- Kuo, M. H., et al. (2013). Media use among adolescents with Autism Spectrum disorder. *Autism*, 18(8), 914–923. <https://doi.org/10.1177/1362361313497832>.
- Rozema, R. (2015). Manga and the autistic mind. *The English Journal*, 105(1), 60–68.
- Tateno, M., et al. (2012). Hikikomori as a possible clinical term in Psychiatry: A Questionnaire survey. *BMC Psychiatry*, 12(1). <https://doi.org/10.1186/1471-244x-12-169>.
- VanBergeijk, E. (2010). Keiko Tobe: With the light: Raising an Autistic child (volume 5). *Journal of Autism and Developmental Disorders*, 41(3), 381–382. <https://doi.org/10.1007/s10803-010-0964-4>.
- Vuković, K. P. (2014). Virtual worlds and Lacan. *Transference in computer games. Phainomena*, 23, 45–68.
- Winge, T. (2006). Costuming the Imagination: Origins of Anime and Manga Cosplay. *Mechademia*, 1, 65–76.
- Wolf, L. E., et al. (2009). *Students with Asperger Syndrome: A guide for college personnel*. Shawnee Mission: AAPC Publishing.

Annual Review

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Synonyms

Present level of growth or knowledge; Report of annual yearly academic progress

References and Reading

- Azuma, H. (2009). *Otaku: Japan's database animals*. Minneapolis: University of Minnesota Press.
- Brenner, R. E. (2007). *Understanding manga and anime*. Westport: Libraries Unlimited.
- Cowen, T. (2010). *The age of the Infovore: Succeeding in the information economy*. Penguin Publishing Group.
- Horbinski, A. (2015). Record of dying days: The alternate history of Ōoku. *Mechademia*, 10, 63–79. <https://doi.org/10.5749/mech.10.2015.0063>.
- Kinsella, S. (1998). Japanese subculture in the 1990s: Otaku and the amateur manga movement. *The Journal of Japanese Studies*, 24(2), 289–316. <https://doi.org/10.2307/133236>.
- Koegel, L. K., et al. (2015). Improving empathic communication skills in adults with Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders*, 46(3), 921–933. <https://doi.org/10.1007/s10803-015-2633-0>.

Definition

The annual review is a formal meeting required by Individuals with Disabilities Education Act (IDEA) and conducted by the school to develop, review, or revise a student's Individualized Education Program (IEP). IEPs also can be reviewed and revised any time during the year but have to be reviewed and updated at least once per year. The purpose of the annual review is to evaluate and revise the basic educational program, instructional guide, placement, services, and, when the child is 14 or older, a transition plan. The participants include the child's educational team members (e.g., the student [when appropriate], both parents, case manager, child's teachers, a school representative other than

the child's teacher, and others can be invited at the discretion of the parents or the district board of education). Schools must provide parents with advanced notice in writing of the annual review meeting. During the meeting, team members review the child's present level of functioning, progress in the general curriculum and towards IEP goals and objectives, and student needs. Teachers should provide progress-monitoring data for each of the goals and objectives; this should include indicating if the goal is met, partially met, or unmet with specific descriptions and explanations as necessary. Also, the team members make recommendations for the next year's program based on the child's needs and progress with current goals and objectives. These include identifying new goals and objectives, determining the necessary levels and types of support for the child to meet IEP goals, and considering and explaining placement options.

See Also

- [Free Appropriate Public Education](#)
- [Individual Education Plan](#)
- [Individualized Plan for Employment \(IPE\)](#)
- [Individualized Transition Plan \(ITP\)](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- Bateman, B. D., & Herr, C. (2006). *Writing measurable IEP goals and objectives*. Verona: Attainment Publications.
- Boutot, E. A., & Myles, B. S. (2011). *Autism spectrum disorders*. Upper Saddle River: Pearson.
- Hall, L. J. (2007). *Autism spectrum disorders: From theory to practice*. Upper Saddle River: Pearson.
- Ruble, L. A., McGrew, J., Dalrymple, N., & Jung, L. A. (2010). Examining the quality of IEPs for young children with autism. *Journal of Autism and Developmental Disorders*, 20, 1459–1470.

Anorexia

- [Eating Disorders](#)

Antecedent-Behavior-Consequence (A-B-C) Analysis

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Definition

An A-B-C analysis is a descriptive assessment that is conducted as an initial part of a complete functional behavior assessment. The goal of this analysis is to develop hypothesis regarding the function that a problem behavior serves for an individual with ASD. A-B-C analysis views behavior (B) as a function of the antecedents (A) that precede it and the consequences (C) that follow it. Typically, an A-B-C chart is used over an extended time period to record events that occur naturally rather than being systematically arranged. These events occur in the natural environment, with the observer recording the environmental events that occur immediately before the behavior in the (A) section, the specific behavior observed in the (B) section, and the events occurring immediately after the behavior in the (C) section.

Historical Background

A-B-C analysis began in the 1960s with the beginnings of applied behavior analysis, with Sidney Bijou and colleagues asserting the importance of collecting direct and repeated data on the observable interactions between the behavior exhibited by the organism and the events in naturally occurring conditions. Interrelationships between the behavior and past and future events were the primary data of interest. The first step of the analysis was a narrative recording, which was a running description of occurrences during an observational period, with no specific behavior targeted for observation. These

descriptions were the first step in identifying a targeted behavior that would be measured formally in further analysis. These temporally sequenced events were translated into A-B-C forms that specified each behavior of interest and the events that occurred immediately before and after the behavior.

In the 1970s, research conducted by Edward Carr and colleagues found that many problem behaviors were logically linked to a small set of antecedents and consequences. Specifically, these researchers stated that an individual with ASD usually exhibited problem behavior to either gain access to attention or a desired item or to escape an undesired event. With the growing body of research studies that supported these findings, the focus of A-B-C analysis narrowed. Currently, many A-B-C analyses focus on more severe problem behavior, such as self-injury, aggression, tantrums, and pica. Antecedent conditions usually consist of (1) demands, (2) attention removed, (3) preferred activity removed, and (4) alone. Similarly, consequence events that follow the problem behavior are often restricted in focus to (1) attention provided in the form of reprimands or soothing statements, (2) removal of demands, (3) access to preferred items, or (4) problem behavior is ignored or neutrally redirected. In addition, initiation of the A-B-C analysis is triggered by concerns regarding the problem behavior voiced by clinical or educational team.

Current Knowledge

The customary usage of the A-B-C form is as one component of a complete functional behavior assessment of a problem behavior exhibited by the individual with ASD. However, these forms can be used for any socially significant behavior of interest. A-B-C forms can be open-ended, where the observer fills in any event that occurs before or after the behavior. Some A-B-C analyses specify time frames and define “immediately” specifically (e.g., as 20 s before or after the behavior occurs).

The categories to be completed in the observation are:

1. The observable behaviors (B) exhibited by the individual with ASD. When defining behavior, it is important to provide clear criteria of the behavior. (e.g., tantrums might be distinguished from whining or crying by being described with an intensity and duration measure, such as screaming and loud crying, that lasts more than 30 s. In addition, tantrums co-occur with one of the following behaviors: lying on the floor, kicking legs, and/or swiping materials off desk). It is also important to record the extent to which the behavior co-occurs with other behaviors in a sequence. Such a sequence might be, for example, first, crying; second, falling on the ground; and third, throwing large objects at adults.
2. Antecedent events (A's) that immediately precede the behavior.
3. Consequent events (C's) that immediately follow the behavior. The consequent events customarily recorded are the social behavior of the adult that is interacting with the individual and include behaviors such as providing attention, feedback, reprimands, access to preferred items/events, and ignoring.

It is also important to include information regarding the setting, other persons present, and materials available and include any other information that may be relevant, such as time of day, day of week, and any unusual events that may effect behavior.

Information gathered from A-B-C analysis is used to develop hypothesis regarding the function (motivation) of the problem behavior and then develop subsequent treatment plans based on this information.

Use of A-B-C forms requires training of observers to limit their recordings to observable and measurable behaviors, as untrained observers have been reported to include subjective impressions of thoughts and feelings of the person observed. This might lead to instances of recording impressions such as “frustrated,” “mad,” “agitated,” or “sad.” In addition, sensitivity to the types of environmental events needs to be trained. It is not uncommon for an untrained observer to record “nothing” as an event, and training on specificity of events to include aspects such as

physical environment, persons present, and materials available is necessary.

Narrative Recordings

These recordings included a description of the setting, time, people present, and materials available. The evaluator begins with a running narrative description of the individual’s behavior, such as “Ed is playing alone in the block center.” When the teacher says “Time to come to circle,” Ed continues to play with the blocks, and the teacher starts the circle without him. When the Aide taps Ed on the shoulder and says “Ed, it’s time to go to circle,” Ed throws the block at the aide. The aide then leaves Ed to play with the blocks, and the teacher conducts circle time with the other children. Here is sample sequence analysis of this recording into a three-column form of antecedents (A), behaviors (B), and consequences (C).

Antecedent (what happened right before the behavior occurred?)	Behavior (record the behavior here)	Consequence (what happened right after the behavior occurred?)
	Ed is playing in the block center	
Teacher says “It’s time for circle”	Ed continues to play with blocks	Aide taps Ed and asks him to join the circle
Aide taps Ed and asks him to join the circle	Ed throws block at aide	Aide leaves Ed alone
Aide leaves Ed alone	Ed plays with blocks	Teacher and aide conduct circle without Ed

The analysis is restricted to describing the participant’s behavior and excludes conjecture regarding the participant’s thoughts and feelings.

For example, “He hits other children because *he does not understand* the situation” would not be included in the analysis, as *understanding* is not observable or measurable behavior. Finally, in A-B-C analysis, generalizations are not made about the environment and behavior, such as “He is a trouble maker who always gives the teacher a hard time.” Finally, in this analysis, consequent events for one behavior can turn into antecedent events for the following behavior.

Open-Ended A-B-C Recording

In this type of analysis, the narrative recording is omitted. The observer uses the A-B-C form when the specific targeted behavior occurs and records the antecedents and consequences that come before and after the targeted behavior. It is recommended that observers include the time the behavior started and ended, the intensity of the behavior, and any other important characteristics of the setting. An example of a form for this type of recording is in Table 1.

Specific A-B-C Recording

In this type of A-B-C analysis, the observer is provided with a specific checklist of A-B-C events to record in a specific ongoing time period. For example, the time period might consist of a 1-h block in the morning, and the observer would record specified behaviors that occurred during that time. In addition, the specified antecedents are recorded whether or not they were followed by problem behavior. This is distinguished from the open-ended recording described above that is only used when the targeted behavior occurs. This type of recording allows a more fine-tuned analysis of the relationship between the antecedent and behavior, as it would detect conditions where the antecedent occurred and the behavior did not

Antecedent-Behavior-Consequence (A-B-C) Analysis, Table 1 Open-ended A-B-C form

Antecedent (what happened right before the behavior occurred?)	Behavior (record the behavior here)	Consequence (what happened right after the behavior occurred?)	Comments
Ex. Teacher said “Time for math” while placing worksheet before student	Ex. Kicked teacher	Ex. Teacher says “It looks like you’re not ready for work” and takes worksheet and walks back to desk	

follow, ruling out faulty correlations between antecedents and consequences.

The specific events recorded can be developed from preliminary information gathered from interviews and/or narrative recordings (see above). The following are possible specific antecedents, behaviors, and consequences used on these forms:

Antecedents

- Demand
- Request
- Feedback
- Denial
- Reprimand
- Transition
- Alone
- Removal or diversion of adult attention
- One-to-one instruction
- Group instruction
- Physical contact
- Social interaction
- Engaged in preferred activity

Behaviors

- Aggression
- Tantrums
- Self-injury
- Bolting
- Pica
- Loud vocalizations
- Stereotyped behavior
- Noncompliance
- Throwing
- Property destruction

Consequences

- Attention
- Corrective feedback
- Access to preferred item
- Ignoring or redirecting behavior
- Task demand
- Task removed
- Physical contact
- Soothing
- Automatic reinforcement (self-stimulation)
- Reactive behavior management procedure

- Time out
- Overcorrection
- Response cost
- Contingent exercise

While the above list provides broad categories of environmental conditions to be analyzed, more fine-grained analysis is often warranted. For example, “task demand” can be specified further as follows:

- Task demand
 - Instruction provided with only auditory cues
 - Instruction with auditory and visual cues
 - Instruction with only visual cues
 - Instruction with auditory, visual, and tactile cues

Or

- Task demand
 - Math task
 - Art task
 - Writing task
 - Expressive speech task

The important point to remember is that the A-B-C analysis should be provided with enough specificity to identify the relevant variables that trigger and maintain the problem behavior. If a student will play with all toys with the exception of puzzles, this should be specified in the analysis.

The sample form in Table 2 lists specific categories of antecedents, behaviors, and consequences for the observer to check off. Using this form, the observer records the antecedent events as they occur, even if the problem behavior does not occur after the antecedent.

After the data are collected, summary statements are developed for each major antecedent or consequence of the behavior and hypotheses are generated regarding the function of the problem behavior.

Future Directions

While there is a growing body of peer-reviewed research studies that shows that while the data

Antecedent-Behavior-Consequence (A-B-C) Analysis, Table 2 Specific A-B-C recording form

Antecedent (circle antecedent)	Behavior (circle behavior)	Consequence (circle consequence)
Demand/instruction	Self-injury	Provide adult attention
Transition	Aggression	Give preferred item or activity
Playing alone	Tantrums	Remove adult demand
Adult attention removed		Provide adult attention
Restrict access to preferred item/activity		Adult reprimand
Other: _____		Other: _____
Demand/instruction	Self-injury	Provide adult attention
Transition	Aggression	Give preferred item or activity
Playing alone	Tantrums	Remove adult demand
Adult attention removed		Provide adult attention
Restrict access to preferred item/activity		Adult reprimand
Other: _____		Other: _____
Repeat above		

collected from A-B-C observations is useful, additional studies have asserted that functional analysis is a more reliable method of identifying variables that control the behavior, and therefore, manipulating these variables lead to more successful treatments. It is therefore recommended that information gathered from the descriptive A-B-C analysis be used as an initial information-gathering step that precedes a formal functional (experimental) analysis. There is controversy regarding this recommendation, as it is argued by some that the information from the A-B-C analysis is sufficient to form hypothesis regarding the motivation of problem behavior that can lead to effective treatments. The time, cost, and controlled clinical settings required to conduct a thorough functional analysis is often not available in customary educational and clinic settings where treatment is provided.

Current A-B-C analyses are restricted to recording observable events in the environment that may predict the occurrence of problem behavior. These events are restricted to immediate antecedents and consequences, which have been referred to as *near triggers*. Future analysis are taking into account *far triggers* such as lack of sleep, a death in the family, moving residences, or other changes in events that may not be immediately apparent in the A-B-C setting. It is recommended to use equipment to measure biological variables, such as increased or decreased heart rate, the need to urinate, physical pain, and

low blood sugar when identifying predictors of problem behavior in the future.

It is argued that underlying characteristics of the individual with ASD can also be strong predictors of behavior. These can include deficiencies such the ability to process complex auditory information, cognitive limitations, and difficulty with abstract reasoning. In addition, the effects of anxiety and mood disorders could be considered as contributing factors to behavior.

See Also

- [Analog Condition Functional Analysis](#)
- [Applied Behavior Analysis \(ABA\)](#)
- [Functional Behavior Assessment](#)

References and Reading

- Bijou, S. W., Peterson, R. F., & Ault, M. H. (1968). A method to integrate descriptive and experimental field studies at the level of data and empirical concepts. *Journal of Applied Behavior Analysis, 1*, 175–191.
- Carr, E. G. (1977). The motivation of self-injurious behavior: A review of some hypotheses. *Psychological Bulletin, 84*, 800–816.
- Feldman, M. A., & Griffiths, D. (1997). Comprehensive assessment of severe behavior problems. In N. N. Singh (Ed.), *Prevention and treatment of severe behavior problems: Models and methods in developmental disabilities*. Pacific Grove: Brookes Publishing Company.
- Lerman, D. C., & Iwata, B. A. (1993). Descriptive and experimental analysis of variables maintaining

- self-injurious behavior. *Journal of Applied Behavior Analysis*, 26, 293–319.
- Mayer, G. R., Sulzer-Azaroff, B., & Wallace, M. (2011). *Behavior analysis for lasting change* (2nd ed.). New York: Sloane Publishing.
- Neef, N., & Peterson, S. (2007). Functional behavior assessment. In J. O. Cooper, T. E. Heron, & W. L. Heward (Eds.), *Applied behavior analysis* (2nd ed., pp. 500–524). Upper Saddle River: Pearson Education.
- O'Neill, R. E., Horner, R. H., Albin, R. W., Sprague, J. R., Storey, K., & Newton, J. S. (1997). *Functional assessment for problem behavior: A practical handbook* (2nd ed.). Pacific Grove: Brooks/Cole.
- Pyles, D. A. M., & Baily, J. S. (1990). Diagnosing severe behavior problems. In A. C. Repp & N. N. Singh (Eds.), *Perspectives on the use of aversive and nonaversive interventions for persons with developmental disabilities* (pp. 381–401). Sycamore: Sycamore Publishing.
- Romanczyk, R. G. (1996). Behavioral analysis and assessment: The cornerstone to effectiveness. In C. Maurice, G. Green, & S. C. Luce (Eds.), *Behavioral intervention for young children with autism* (pp. 195–217). Austin: PRO-ED.
- Sulzer-Azaroff, B., Dyer, K., Soucy, D., & Dupont, S. (2012). *Applying behavior analysis across the autism spectrum: A field guide for practitioners* (2nd ed.). Cornwall-on-Hudson: The Cambridge Center (Sloan Century Series in Behavior Analysis).

Anterior Cingulate

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Synonyms

ACC

Structure

The anterior cingulate cortex (ACC) is in the frontal portion of the cingulate cortex, situated medially just above the corpus callosum. The ACC consists of Brodmann areas 24, 25, 32, and 33. Vogt and colleagues (Vogt 2009) defined four major subdivisions of the rostral ACC. These include a supracallosal portion (above the corpus callosum), designated “midcingulate cortex,”

which is divided into an anterior and a posterior portion. The aspect of the cingulate lying anterior and ventral to the corpus callosum is designated “anterior cingulate.” This region is further divided into a pregenual region (more anterior) and subgenual region (more ventral). Based on observed functional differences (Bush et al. 2000), neuroscientists often distinguish between a “cognitive” dorsal portion of the ACC and an affective ventral portion of the ACC. The dorsal ACC is connected to the prefrontal, parietal and motor cortices, and motor and frontal eye fields whereas the ventral ACC is connected to the more traditional limbic regions including the amygdala, nucleus accumbens, anterior insula, and hypothalamus. More recently, Shackman et al. (2011) provided a coordinate-based meta-analysis of neuroimaging studies suggesting the cognitive-affective demarcation of the ACC is less clear-cut than was originally assumed.

Function

The anterior cingulate’s functions are diverse, including the cognitive control of motor behavior in response monitoring (Botvinick et al. 2001; Devinsky et al. 1995; Holroyd and Coles 2002), reward-based learning (Holroyd & Coles), registering physical (Craig et al. 1996) and social pain (Eisenberger and Lieberman 2004), empathy, consciousness, and autonomic functions.

The ACC is implicated in the related processes of conflict monitoring, response inhibition, and error detection. On simple behavioral paradigms such as the Stroop color word interference task, the individual is charged with responding to the color of a word when the word color differs from the written word (e.g., the word blue written in yellow text). In this task conflict is engaged because the automaticity involved in reading the word conflicts with the different color of the printed word. However, the ACC has been shown to be activated independently of the presence of response alternatives on a Stroop-like task suggesting a role as a top-down regulator increasing the amount of “top-down” regulation required to meet the task demands (Roelofs et al. 2006). In

doing so, the ACC would selectively enhance the activation of a correct response pending some selection threshold to be exceeded (Roelofs et al. 2006). The incorrect response must be inhibited in favor of the correct response. If an incorrect response is executed, it needs to be detected to adjust performance accordingly. In this process, the ACC appears to be involved in error detection, regardless of whether or not errors are consciously perceived, and in the perception of errors committed by others (Gentsch et al. 2009; Hester et al. 2005; Holroyd et al. 2004; Klein et al. 2007; Ullsperger and von Cramon 2001; Ullsperger et al. 2007).

The role of the ACC in response monitoring and reward processing has been linked in a general reinforcement model that attempts to account for error processing, feedback processing, and reinforcement learning more generally. Here, the dorsal ACC is thought to use reward prediction error signals, conveyed via the mesencephalic dopamine system, to reinforce adaptive behavioral responses (Holroyd and Coles 2002). As noted by Holroyd and Coles (2008), two general types of theories have been proposed to describe the role of the dorsal ACC in response monitoring processes. Some theories propose the ACC serves an evaluative role to detect errors or conflict. The response selection perspective suggests ACC is directly involved in the decision making process (Holroyd & Coles). Other neuroimaging work implicates the ACC, but not specifically the dorsal ACC in outcome anticipation, uncertainty of outcome (Critchley et al. 2001a), subjective value of potential rewards (Kable and Glimcher 2007), and imagined or “fictive” rewards (Hayden et al. 2009).

A growing body of work implicates the ACC in physical pain, social pain, and empathy-related processes. In terms of physical pain, recent neuroimaging work indicates the ACC is associated with the unpleasantness aspect of physical pain (Rainville et al. 1997). Studies of social exclusion, a socially painful experience, indicate that some of the same neural circuitry, including the ACC, is involved in the distressing aspect of being excluded by others in a group (Eisenberger and Lieberman 2004). Among typically developing adolescents, neural response to social rejection engages brain

regions involved in affective distress (subgenual anterior cingulate, anterior insula) and affect regulation (ventrolateral PFC, ventral striatum) (e.g., Masten et al. 2011; Sebastian et al. 2011). Interestingly, the anterior cingulate cortex is part of a network consistently engaged in studies of empathy for others’ pain (Krach et al. 2011).

The ventral ACC has been implicated in emotion processing (for reviews see Bush et al. 2000; Shackman et al. 2011). The ventral ACC has been shown to be engaged in modulation of the sympathetic as well as the parasympathetic aspects of the autonomic nervous system (Critchley et al. 2001b; Matthews et al. 2004). As part of a network of higher cortical structures including the insula, amygdala, and hippocampus, the ventral ACC is connected to lower structures that have been dubbed the central autonomic network (Benarroch 1993).

Pathophysiology

Emerging evidence at the levels of cell microstructure, neuronal connectivity, and brain volume suggest abnormalities in the ACC of people with an autism spectrum disorder (ASD). In post-mortem work, Simms and colleagues (Simms et al. 2009) observed that individuals with autism had smaller neurons and reduced neuronal density in the ACC. They specifically examined von Economo neurons (VENs). Interest in VENs in ASD has burgeoned recently given their putative role in emotional regulation and social interaction (Allman et al. 2005; Allman et al. 2010). Simms et al. (2009) found that while VENs did not differ from control brains overall, a subset of ($n = 3$) ASD individuals had significantly increased VEN density whereas the remaining six individuals had reduced VEN density compared to controls. Suda et al. (2011) recently documented the expression of axon guidance proteins were significantly lower in the ACC region among autistic individuals compared to controls (Suda et al.). Similarly, in an examination of ACC single cell axons in brain white matter, Zikopoulos and Barbas (2010) found evidence for a decrease in long axons that communicate over long distances and an

excessive number of thin axons linking the ACC to neighboring areas. Other work points to the role of GABAergic (gamma-aminobutyric acid) function in the ACC in ASD (Zikopoulos and Barbas). GABAergic neurons have chiefly inhibitory action at receptors in the brain. GABA is important for normal cortical functioning, information processing, and cytoarchitecture during brain development (Di Cristo 2007). For instance, in a pair of studies Oblak, Gibbs, and Blatt (2009, 2010) observed reductions in GABA_A and GABA_B receptor densities in the ACC (Oblak et al. 2009, 2010). Lastly, Nakamura et al. (2011) conducted a postmortem study implicating the serotonin (5-HT) system in the ACC to ASD. In the brain, serotonin plays an important role in mood regulation sleep and appetite. Nakamura et al. (2011) observed that the expression of a protein that regulates the serotonin transporter (5-HTT), STX1A, was significantly lower in the ACC region in an autism group compared to controls (Nakamura et al. 2011).

In vivo research documents altered ACC cell membrane metabolism (Levitt et al. 2003). Employing positron emission tomography (PET), Ohnishi et al. (2000) found decreased left ACC cerebral blood flow (Ohnishi et al.). Similarly, Haznedar et al. (1997) observed reduced glucose metabolism throughout the cingulate gyrus and reduced right ACC volume (Haznedar et al.). Moreover, in the ASD group, glucose metabolism was positively associated with social interaction, verbal communication, and nonverbal communication scores.

In terms of connectivity with other brain regions, Welchew et al. (2005) observed atypical connectivity of the ACC with inferior occipital and inferior frontal cortices (Welchew et al.). In the first study using diffusion tensor imaging in ASD, Barnea-Goraly et al. (2004) observed that ASD children had reduced ACC fractional anisotropy (FA), a measure thought to reflect fiber density, axonal diameter, and myelination in white matter, extending to adjacent regions including the ventromedial frontal area and subgenual prefrontal region, bilateral temporoparietal junctions, and adjacent superior temporal gyrus (Barnea-Goraly et al.). Similarly, Noriuchi et al. (2010)

observed that FA was significantly lower in a child ASD group in the mid and right ACC among other regions (Noriuchi et al.). Using diffusion tensor imaging, Ke et al. (2009) observed decreased white matter density in a high-functioning autism group in the right frontal lobe, left parietal lobe, and right anterior cingulate and increased white matter density in the right frontal lobe, left parietal lobe, and left cingulate gyrus compared to control children (Ke et al.). Lastly, in terms of grey matter, Waiter et al. (2004) documented an increase in grey matter volume in the ACC among male adolescent ASD subjects (Waiter et al.).

A growing number of studies find individuals with ASD have deficits in response monitoring. Response monitoring is an executive task subserved by the ACC. Response monitoring specifically refers to evaluating whether one's actions are consistent with one's goals and modifying behavior accordingly to optimize outcomes. In a recent fMRI study, Thakkar et al. (2008) used a performance monitoring task finding that individuals with ASD had increased rostral ACC activation which was related to repetitive behaviors (Thakkar et al.). In terms of behavioral responses, Russell and Jarrold (1998) reported reduced error self-correction among adults with ASD (Russell and Jarrold). Bogte, Flamma, van der Meere, and van Engeland (2007) observed reduced post-error slowing in ASD, an index of behavioral correction to improve performance on a subsequent trial (Bogte et al. 2007). In one of the first ERP studies suggesting abnormal response monitoring in high-functioning ASD, Henderson et al. (2006) observed increased latency in the ERN event-related potential response, and poorer behavioral performance overall. ASD children did not differ from comparison children in terms of ERN amplitude, but ASD probands with higher IQs showed significantly larger ERN responses, suggesting hypersensitivity to errors among this group. In a second study with ASD children, Vlamings, Jonkman, Hoeksma, van Engeland, and Kemner (2008) observed smaller ERNs and a lack of post-error slowing behaviorally (Vlamings et al. 2008). The authors observe this finding, coupled with a comparable correct trial negativity (CRN) for

ASD and typical children is consistent with perseverative behavior seen in ASD children (for a similar finding in adults see Santesso et al. (2010)). Interestingly, a recent study employing a reward-loss feedback task did not find differences in a related brain response thought to be subserved by the ACC, the feedback-related negativity (FRN) (Larson et al. 2011). These data suggest that individuals with ASD process external, concrete feedback similarly to typically developing individuals.

Not surprisingly, anterior cingulate dysfunction also continues to emerge when the experimental paradigm involves social functioning. A meta-analytic examination of 24 studies on social information processing and 15 nonsocial studies by Di Martino et al. (2009) suggests that a distributed system involving the ACC and the anterior insula was hypoactive for individuals with autism – in nonsocial studies the ASD individuals were more likely to show activation in the rostral ACC, which is typically suppressed in attention-demanding tasks. Importantly, we see deficits in the functioning of this specific circuitry in social challenge tasks such as social rejection/exclusion paradigms. Compared to controls, children and adolescents with ASD showed hypoactivation in the ventral ACC and right insula when they were excluded from a simple computer game by same-aged peers (Bolling et al. 2011; Masten et al. 2011).

Other recent social-cognitive work employing experimental paradigms seems to tap monitoring processes as described above, but social monitoring in particular. Recently, Chiu et al. (2008) provided evidence that atypical neural self-representation in ASD involves the cingulate cortex. In typical adolescents and young adults, self-referential compared with other-referential processing preferentially recruited the middle cingulate cortex and ventromedial prefrontal cortex. ASD individuals did not show this self-referential preference. Instead, ventromedial prefrontal cortex responded equally to self and other, while middle cingulate cortex responded more to other-mentalizing than self-mentalizing (Chiu et al.). Importantly, the lack of cingulate “self” response pattern in the ASD group related parametrically to ASD symptom severity. In another

important study, Kennedy and Courchesne (2008) had autism and control participants make true/false judgments for statements about themselves (“self” condition) or a close other person (“other” condition) and related to psychological personality traits (“internal”) or observable characteristics/behaviors (“external”). Within the ventral medial prefrontal cortex and ventral anterior cingulate cortex, activity was reduced for the ASD group across all conditions and also during a rest condition, suggesting task-independent dysfunction in this region (Kennedy and Courchesne).

While clearly a large amount of data supports ACC involvement in the autism phenotype, the ACC should not be considered the only neural structure relevant to autism pathophysiology. First, the ACC is connected to multiple brain and body systems that may be more or less affected in the disorder (see above). Second, and relatedly, functioning in the ACC contributes to self-regulatory and social cognitive abilities, but in concert with other brain and body systems. Third, functioning in the ACC cannot account for all aspects of the autism phenotype more generally (e.g., language delays). Thus, future work examining ACC function in autism will need to incorporate new developments in our understanding of ACC anatomy and function (Shackman et al. 2011; Vogt 2009) coupled with nuanced and yoked paradigms that can be used to parse ACC-relevant functions (Bolling et al. 2011; Chiu et al. 2008) explicit examination of individual differences (Henderson et al. 2006) and a neural systems perspective (Mundy et al. 2010). There again, autism emerges in a developing organism necessitating developmental studies tracking the course of ACC development against the backdrop of typical ACC development (Pelphrey et al. 2011). As of yet, we do not know whether or not the ACC dysfunction plays a causal role in the emergence of the disorder or is secondary to having the condition.

See Also

- [ERN](#)
- [Error-Related Negativity](#)
- [Feedback-Related Negativity](#)

References and Reading

- Allman, J. M., Watson, K. K., Tetreault, N. A., & Hakeem, A. Y. (2005). Intuition and autism: A possible role for Von Economo neurons. *Trends in Cognitive Sciences*, 9(8), 367–373.
- Allman, J. M., Tetreault, N. A., Hakeem, A. Y., Manaye, K. F., Semendeferi, K., Erwin, J. M., et al. (2010). The von Economo neurons in fronto-insular and anterior cingulate cortex in great apes and humans. *Brain Structure & Function*, 214(5–6), 495–517.
- Barnea-Goraly, N., Kwon, H., Menon, V., Eliez, S., Lotspeich, L., & Reiss, A. L. (2004). White matter structure in autism: Preliminary evidence from diffusion tensor imaging. *Biological Psychiatry*, 55(3), 323–326.
- Benarroch, E. E. (1993). The central autonomic network: Functional organization, dysfunction, and perspective. *Mayo Clinic Proceedings*, 68(10), 988–1001.
- Bogte, H., Flamma, B., van der Meere, J., & van Engeland, H. (2007). Post-error adaptation in adults with high functioning autism. *Neuropsychologia*, 45(8), 1707–1714.
- Bolling, D. Z., Pitskel, N. B., Deen, B., Crowley, M. J., McPartland, J. C., Kaiser, M. D., et al. (2011). Enhanced neural responses to rule violation in children with autism: A comparison to social exclusion. *Developmental Cognitive Neuroscience*, 1(3), 280–294.
- Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, 108(3), 624–652.
- Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Science*, 4(6), 215–222.
- Chiu, P. H., Kayali, M. A., Kishida, K. T., Tomlin, D., Klinger, L. G., Klinger, M. R., et al. (2008). Self responses along cingulate cortex reveal quantitative neural phenotype for high-functioning autism. *Neuron*, 57(3), 463–473.
- Craig, A. D., Reiman, E. M., Evans, A., & Bushnell, M. C. (1996). Functional imaging of an illusion of pain. *Nature*, 384(6606), 258–260.
- Critchley, H. D., Mathias, C. J., & Dolan, R. J. (2001a). Neural activity in the human brain relating to uncertainty and arousal during anticipation. *Neuron*, 29(2), 537–545.
- Critchley, H. D., Mathias, C. J., & Dolan, R. J. (2001b). Neuroanatomical basis for first- and second-order representations of bodily states. *Nature Neuroscience*, 4(2), 207–212.
- Devinsky, O., Morrell, M. J., & Vogt, B. A. (1995). Contributions of anterior cingulate cortex to behaviour. *Brain*, 118, 279–306.
- Di Cristo, G. (2007). Development of cortical GABAergic circuits and its implications for neurodevelopmental disorders. *Clinical Genetics*, 72(1), 1–8.
- Di Martino, A., Ross, K., Uddin, L. Q., Sklar, A. B., Castellanos, F. X., & Milham, M. P. (2009). Functional brain correlates of social and nonsocial processes in autism spectrum disorders: An activation likelihood estimation meta-analysis. *Biological Psychiatry*, 65(1), 63–74.
- Eisenberger, N. I., & Lieberman, M. D. (2004). Why rejection hurts: A common neural alarm system for physical and social pain. *Trends in Cognitive Sciences*, 8(7), 294–300.
- Gentsch, A., Ullsperger, P., & Ullsperger, M. (2009). Dissociable medial frontal negativities from a common monitoring system for self- and externally caused failure of goal achievement. *NeuroImage*, 47(4), 2023–2030.
- Hayden, B. Y., Pearson, J. M., & Platt, M. L. (2009). Fictive reward signals in the anterior cingulate cortex. *Science*, 324(5929), 948–950.
- Haznedar, M. M., Buchsbaum, M. S., Metzger, M., Solimando, A., Spiegel-Cohen, J., & Hollander, E. (1997). Anterior cingulate gyrus volume and glucose metabolism in autistic disorder. *The American Journal of Psychiatry*, 154(8), 1047–1050.
- Henderson, H., Schwartz, C., Mundy, P., Burnette, C., Sutton, S., Zahka, N., et al. (2006). Response monitoring, the error-related negativity, and differences in social behavior in autism. *Brain and Cognition*, 61(1), 96–109.
- Hester, R., Foxe, J. J., Molholm, S., Shpaner, M., & Garavan, H. (2005). Neural mechanisms involved in error processing: A comparison of errors made with and without awareness. *NeuroImage*, 27(3), 602–608.
- Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, 109(4), 679–709.
- Holroyd, C. B., & Coles, M. G. (2008). Dorsal anterior cingulate cortex integrates reinforcement history to guide voluntary behavior. *Cortex*, 44(5), 548–559.
- Holroyd, C. B., Nieuwenhuis, S., Yeung, N., Nystrom, L., Mars, R. B., Coles, M. G., et al. (2004). Dorsal anterior cingulate cortex shows fMRI response to internal and external error signals. *Nature Neuroscience*, 7(5), 497–498.
- Kable, J. W., & Glimcher, P. W. (2007). The neural correlates of subjective value during intertemporal choice. *Nature Neuroscience*, 10(12), 1625–1633.
- Ke, X., Tang, T., Hong, S., Hang, Y., Zou, B., Li, H., et al. (2009). White matter impairments in autism, evidence from voxel-based morphometry and diffusion tensor imaging. *Brain Research*, 1265, 171–177.
- Kennedy, D. P., & Courchesne, E. (2008). Functional abnormalities of the default network during self- and other-reflection in autism. *Social Cognitive and Affective Neuroscience*, 3(2), 177–190.
- Klein, T. A., Endrass, T., Kathmann, N., Neumann, J., von Cramon, D. Y., & Ullsperger, M. (2007). Neural correlates of error awareness. *NeuroImage*, 34(4), 1774–1781.
- Krach, S., Cohrs, J. C., de Echeverria Loebell, N. C., Kircher, T., Sommer, J., Jansen, A., et al. (2011). Your flaws are my pain: Linking empathy to vicarious embarrassment. *PLoS One*, 6(4), e18675.

- Larson, M. J., South, M., Krauskopf, E., Clawson, A., & Crowley, M. J. (2011). Feedback and reward processing in high-functioning autism. *Psychiatry Research*, 187(1–2), 198–203.
- Levitt, J. G., O'Neill, J., Blanton, R. E., Smalley, S., Fadale, D., McCracken, J. T., et al. (2003). Proton magnetic resonance spectroscopic imaging of the brain in childhood autism. *Biological Psychiatry*, 54(12), 1355–1366.
- Masten, C. L., Colich, N. L., Rudie, J. D., Bookheimer, S. Y., Eisenberger, N. I., & Dapretto, M. (2011). An fMRI investigation of responses to peer rejection in adolescents with autism spectrum disorders. *Developmental Cognitive Neuroscience*, 1(3), 260–270.
- Matthews, S. C., Paulus, M. P., Simmons, A. N., Nelesen, R. A., & Dimsdale, J. E. (2004). Functional subdivisions within anterior cingulate cortex and their relationship to autonomic nervous system function. *NeuroImage*, 22(3), 1151–1156.
- Mundy, P., Gwaltney, M., & Henderson, H. (2010). Self-referenced processing, neurodevelopment and joint attention in autism. *Autism*, 14(5), 408–429.
- Nakamura, K., Iwata, Y., Anitha, A., Miyachi, T., Toyota, T., Yamada, S., et al. (2011). Replication study of Japanese cohorts supports the role of STX1A in autism susceptibility. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 35(2), 454–458.
- Noriuchi, M., Kikuchi, Y., Yoshiura, T., Kira, R., Shigeto, H., Hara, T., et al. (2010). Altered white matter fractional anisotropy and social impairment in children with autism spectrum disorder. *Brain Research*, 1362, 141–149.
- Oblak, A., Gibbs, T. T., & Blatt, G. J. (2009). Decreased GABAA receptors and benzodiazepine binding sites in the anterior cingulate cortex in autism. *Autism Research*, 2(4), 205–219.
- Oblak, A. L., Gibbs, T. T., & Blatt, G. J. (2010). Decreased GABA(B) receptors in the cingulate cortex and fusiform gyrus in autism. *Journal of Neurochemistry*, 114(5), 1414–1423.
- Ohnishi, T., Matsuda, H., Hashimoto, T., Kunihiro, T., Nishikawa, M., Uema, T., et al. (2000). Abnormal regional cerebral blood flow in childhood autism. *Brain*, 123(Pt 9), 1838–1844.
- Pelphrey, K. A., Shultz, S., Hudac, C. M., & Vander Wyk, B. C. (2011). Research review: Constraining heterogeneity: The social brain and its development in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 52(6), 631–644.
- Rainville, P., Duncan, G. H., Price, D. D., Carrier, B., & Bushnell, M. C. (1997). Pain affect encoded in human anterior cingulate but not somatosensory cortex. *Science*, 277(5328), 968–971.
- Roelofs, A., van Turenout, M., & Coles, M. G. (2006). Anterior cingulate cortex activity can be independent of response conflict in Stroop-like tasks. *Proceedings of the National Academy of Sciences of the United States of America*, 103(37), 13884–13889.
- Russell, J., & Jarrold, C. (1998). Error-correction problems in autism: Evidence for a monitoring impairment? *Journal of Autism and Developmental Disorders*, 28(3), 177–188.
- Santesso, D. L., Drmic, I. E., Jetha, M. K., Bryson, S. E., Goldberg, J. O., Hall, G. B., et al. (2010). An event-related source localization study of response monitoring and social impairments in autism spectrum disorder. *Psychophysiology*, 48, 241–251.
- Sebastian, C. L., Tan, G. C., Roiser, J. P., Viding, E., Dumontheil, I., & Blakemore, S. J. (2011). Developmental influences on the neural bases of responses to social rejection: Implications of social neuroscience for education. *NeuroImage*, 57(3), 686–694.
- Shackman, A. J., Salomons, T. V., Slagter, H. A., Fox, A. S., Winter, J. J., & Davidson, R. J. (2011). The integration of negative affect, pain and cognitive control in the cingulate cortex. *NeuroImage*, 57(3), 154–167.
- Simms, M. L., Kemper, T. L., Timbie, C. M., Bauman, M. L., & Blatt, G. J. (2009). The anterior cingulate cortex in autism: Heterogeneity of qualitative and quantitative cytoarchitectonic features suggests possible subgroups. *Acta Neuropathologica*, 118(5), 673–684.
- Suda, S., Iwata, K., Shimmura, C., Kamenoy, Y., Anitha, A., Thaseem, I., et al. (2011). Decreased expression of axon-guidance receptors in the anterior cingulate cortex in autism. *Molecular Autism*, 2(1), 14.
- Thakkar, K. N., Polli, F. E., Joseph, R. M., Tuch, D. S., Hadjikhani, N., Barton, J. J., et al. (2008). Response monitoring, repetitive behaviour and anterior cingulate abnormalities in autism spectrum disorders (ASD). *Brain*, 131(Pt 9), 2464–2478.
- Ullsperger, M., & von Cramon, D. Y. (2001). Subprocesses of performance monitoring: A dissociation of error processing and response competition revealed by event-related fMRI and ERPs. *NeuroImage*, 14(6), 1387–1401.
- Ullsperger, M., Nittono, H., & von Cramon, D. Y. (2007). When goals are missed: Dealing with self-generated and externally induced failure. *NeuroImage*, 35(3), 1356–1364.
- Vlamings, P. H., Jonkman, L. M., Hoeksma, M. R., van Engeland, H., & Kemner, C. (2008). Reduced error monitoring in children with autism spectrum disorder: An ERP study. *The European Journal of Neuroscience*, 28(2), 399–406.
- Vogt, B. A. (Ed.). (2009). *Cingulate neurobiology and disease*. Oxford: Oxford University Press.
- Waiter, G. D., Williams, J. H., Murray, A. D., Gilchrist, A., Perrett, D. I., & Whiten, A. (2004). A voxel-based investigation of brain structure in male adolescents with autistic spectrum disorder. *NeuroImage*, 22(2), 619–625.
- Welchew, D. E., Ashwin, C., Berkouk, K., Salvador, R., Suckling, J., Baron-Cohen, S., et al. (2005). Functional

disconnectivity of the medial temporal lobe in Asperger's syndrome. *Biological Psychiatry*, 57(9), 991–998.

Zikopoulos, B., & Barbas, H. (2010). Changes in prefrontal axons may disrupt the network in autism. *The Journal of Neuroscience*, 30(44), 14595–14609.

Antianxiety Medication

► Anxiolytics

Anticholinergic

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Definition

Acetylcholine is a chemical that transmits messages between nerve cells in the brain. Centrally acting anticholinergic drugs block the effect of acetylcholine in the brain. These drugs are used to counteract adverse effects of antipsychotic medications. Acetylcholine is a major neurotransmitter in the brain. Acetylcholine and dopamine are in a dynamic balance in the brain. Because many antipsychotic medications block dopamine receptors in motor regions of the brain, there is a relative excess of acetylcholine. This gives rise to the commonly observed neurological side effects of antipsychotic medications such as tremor, dyskinesia, and dystonia. These adverse effects typically occur early in treatment, are unpleasant, and may pose a serious threat to medication adherence. Anticholinergic medications such as benztropine are often useful in reducing these neurological effects of

antipsychotic medications (link to “Psychopharmacology” section of this encyclopedia).

The anticholinergic drugs can also have adverse effects. In the low doses used to treat neurological side effects of antipsychotic medications, adverse effects are not common. At higher doses, adverse effects can include confusion and memory problems, and hallucinations can occur.

See Also

► Neurotransmitter

References and Reading

Martin, A., Scahill, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.

Anticholinesterase Inhibitors

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Synonyms

[Acetylcholinesterase inhibitors](#); [AChE-inhibitors](#)

Definition

Acetylcholine (ACh) is a neurotransmitter key in an individual's ability to adapt to his/her environment and surrounding stimuli. ASD patients show many deficits in ACh production and receptor function. Acetylcholinesterase is an enzyme

involved in the degradation of acetylcholine. Anti-cholinesterase inhibitors, or acetylcholinesterase inhibitors (AChE-inhibitors), seek to block the action of this enzyme, thus increasing ACh levels and action durations. Examples that have been tested and shown beneficial effects in ASD patients include rivastigmine, donepezil, and galantamine. Improvements in expressive speech are consistent across the drugs. Reports also include improvements in irritability, hyperactivity, receptive language, social withdrawal, inattention, and anger management. Side-effect profiles include diarrhea, nausea, and vomiting. Currently AChE-inhibitors are some of the most promising class of drugs in treatment of dementia, especially in Alzheimer's disorder, indicating some degree of alleviation in memory dysfunction. They are delivered orally, rapidly absorbed, and metabolized via CYP P450 isoenzymes CYP2D6 and CYP3A4 in the liver. Their relatively minimal side-effect profiles combined with demonstrated slight improvement in ASD symptoms make them an attractive candidate for ASD pharmacology. However, substantial validation of their consistent effectiveness and minimal side effects in ASD patients specifically is still required.

See Also

- [Acetylcholine: Definition](#)
- [Donepezil: Definition](#)

References and Reading

- Hardan, A. Y., & Handen, B. L. (2002). A retrospective open trial of adjunctive donepezil in children and adolescents with autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, 12(3), 237–241.
- Nicolson, R., Craven-Thuss, B., & Smith, J. (2006). A prospective, open-label trial of galantamine in autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, 16(5), 621–629.
- Niederhofer, H. (2003). Acetylcholinesterase-inhibitors in the treatment of autistic disorders. *Expert Review of Neurotherapeutics*, 3(4), 409–412.
- Rossignol, D. A. (2009). Novel and emerging treatments for autism spectrum disorders: A systematic review. *Annals of Clinical Psychiatry*, 21(4), 213–236.

Anticipated Regression

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Definition

Under current federal law, and as clarified in several court cases and policy explanations, services for the extended school year (ESY) for children with disabilities (either because they have an IEP or 504 plan) can be provided in some contexts. Historically, the potential for the child to regress (anticipated regression) has been regarded as one of the most relevant of these; even here, however, multiple factors should be taken into account. This regression would typically be defined by a loss of knowledge or skills that reflects an interruption of educational programming, placing the gains the child has made at risk. The factors considered by the IEP team have to do with maintenance of skills as well as the nature and severity of the disability.

See Also

- [504 Plan](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Regression](#)

References and Reading

- Mandlawitz, M. R. (2005). Educating children with autism: Current legal issues. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, 3rd ed., pp. 1161–1173). Hoboken: Wiley.
- Volkmar, F., & Wiesner, L. (2009). *A practical guide to autism*. Hoboken: Wiley.

Anticonvulsants

► Antiepileptic Drugs (AEDs)

Antidepressant Medications

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Definition

Medications, including monoamine oxidase inhibitors (MAOIs), tricyclic and tetracyclic antidepressants (TCAs), selective serotonin reuptake inhibitors (SSRIs), and serotonin-norepinephrine reuptake inhibitors (SNRIs), used to treat depressive disorders, conditions characterized by depressed mood often along with other symptoms including the following: changes in appetite, changes in sleep habits, low energy, low self-esteem, poor concentration, and feelings of hopelessness.

Historical Background

The first types of antidepressants to be developed, sometimes referred to as the first-generation antidepressants, include the MAOIs and TCAs. The first of the MAOIs to be developed was iproniazid, a drug initially marketed in 1952 for the treatment of tuberculosis. When it was shown

that iproniazid appeared to induce euphoria in patients and reversed the effects of reserpine, a known depressant, Saunders and Kline began using iproniazid to treat clinical depression. Kline published an article on its clinical use for the treatment of depression in 1958. Although its adverse effects caused its use to be discontinued, iproniazid was replaced by other compounds that inhibit monoamine oxidase. However, the MAOIs were found to exhibit adverse reactions with other drugs and amines. The development of the TCAs began at the same time as that of the MAOIs. The first TCA to be developed, imipramine, was tested as a neuroleptic agent when it was found to relieve depressive symptoms. Comparing the mechanisms of action of the MAOIs and the TCAs caused investigators to recognize that increasing the amount of synaptic neurotransmitter was important for the treatment of depression.

SSRIs, considered to be second-generation antidepressants, were developed to have the specific mechanism of action of the inhibition of the reuptake of serotonin (5-HT) which resulted in similar treatment effectiveness compared to the TCAs, with decreased side effect profiles in all of the SSRIs besides zimelidine. Zimelidine was the first SSRI to be developed, but its production was discontinued due to its toxicity. Fluoxetine, the first SSRI to be marketed that is still in use, was first marketed by Eli Lilly and Co. in 1987. Another antidepressant medication first marketed in the 1980s is bupropion, an antidepressant with the brand name Wellbutrin which is not a serotonin reuptake inhibitor (SRI) but instead may facilitate dopamine (DA) neurotransmission and may affect norepinephrine (NE).

Development of the third-generation antidepressants began with the goal of obtaining compounds which expand upon the functionality of the SSRIs to include other pharmacological effects thought to affect depressive symptoms while maintaining low side effect profiles. The development of SNRIs as antidepressants followed the development of SSRIs starting in the 1980s, although testing for the appropriate approval for clinical use for the first SNRI marketed was not completed until 1993.

Nefazodone is a third-generation antidepressant first marketed in the United States in 1994 which inhibits the reuptake of 5-HT and NE, as well as acting as an antagonist at the 5-HT_{2A} and α_1 -adrenergic receptors. Another third-generation antidepressant, mirtazapine, was first marketed in the United States in 1996 and inhibits the reuptake of 5-HT and NE, as well as acting as a noradrenergic α_2 -autoreceptor blocker and a 5-HT₂ and 5-HT₃ antagonist.

Current Knowledge

Research has suggested that 5-HT, NE, and DA are involved in the pathophysiology of depression. Each of the antidepressants developed to date affects one to all three of these neurotransmitters in the central nervous system (CNS). Although many different antidepressant drugs and families of antidepressant drugs with different functionalities exist, including the SSRIs, SNRIs, TCAs, and MAOIs, currently these drugs do not differ much from one another in efficacy. However, different individuals may have a greater treatment response to one antidepressant medication than to another. Family history of clinical response to a specific antidepressant medication may be a predictor for the response of an individual to that drug in some cases.

The SSRIs are a commonly prescribed group of antidepressant medications which relieve symptoms of depression by selectively blocking the reuptake of 5-HT. This action of SSRIs in the CNS causes an increase in the amount of synaptic 5-HT. Before the neurons are desensitized to drug, this increase in synaptic 5-HT is counteracted by the stimulation of the presynaptic 5-HT_{1A} autoreceptor which inhibits the further release of 5-HT into the synapse. After about 10–14 days of drug treatment, this autoreceptor is desensitized, and the amount of synaptic 5-HT increases. The five SSRIs currently FDA-approved to treat major depressive disorder (MDD) and marketed in the United States are fluoxetine, sertraline, paroxetine, citalopram, and escitalopram. Fluoxetine, with the brand names Prozac and Sarafem, is also marketed for the treatment of obsessive-

compulsive disorder (OCD). Sertraline, with the brand name Zoloft, is also marketed for the treatment of OCD, panic disorder, posttraumatic stress disorder (PTSD), premenstrual dysphoric disorder (PMDD), and social anxiety disorder. Paroxetine, with the brand names Paxil, Paxil CR, and Pexeva, is also marketed for the treatment of OCD, panic disorder, social anxiety disorder, generalized anxiety disorder (GAD), and PTSD. Citalopram, with the brand name Celexa, is only marketed for the treatment of MDD. Escitalopram, with the brand name Lexapro, is also marketed for the treatment of GAD. Additionally, fluvoxamine, with the brand names Luvox and Luvox CR, although only marketed for the treatment of OCD in the United States, is often prescribed for the treatment of depression.

The SNRIs relieve symptoms of depression by blocking the reuptake of 5-HT and NE. These drugs are similar in clinical use to the SSRIs, but two have the additional effects of treating pain and physical symptoms, such as those of fibromyalgia (FM). The three SNRIs currently marketed in the United States for the treatment of depression are duloxetine, venlafaxine, and desvenlafaxine. Duloxetine, with the brand name Cymbalta, is also marketed for the treatment of GAD, diabetic peripheral neuropathy, FM, and chronic musculoskeletal pain. Venlafaxine, with the brand names Effexor and Effexor XR, is also marketed for the treatment of GAD, social anxiety disorder, and panic disorder. Desvenlafaxine, with the brand name Pristiq, is only marketed for the treatment of MDD. Additionally, milnacipran, with the brand name Savella, is marketed for the treatment of MDD in Japan, although it is only marketed for the treatment of FM in the United States.

Other antidepressants with different mechanisms of action than the SSRIs and SNRIs are bupropion, nefazodone, and mirtazapine. Bupropion, with the brand name Wellbutrin, is not an SRI but instead may potentiate DA activity and may affect NE. Nefazodone inhibits the reuptake of 5-HT and NE, as well as acting as an antagonist at the 5-HT_{2A} and α_1 -adrenergic receptors. Mirtazapine, with the brand names Remeron and Remeron SolTab, inhibits the reuptake of

5-HT and NE, as well as acting as a noradrenergic α_2 -autoreceptor blocker and a 5-HT₂ and 5-HT₃ antagonist.

The TCAs are a family of compounds which affect 5-HT and NE, as well as acting as anticholinergic or antimuscarinic agents, α -adrenergic antagonists, and antihistamines. Although these drugs seem to have similar efficacy to the SSRIs and SNRIs and may be more effective than those drugs, the TCAs are not as well tolerated and have more side effects than the SSRIs and the SNRIs. Clinically, the TCAs are rarely used due to their side effects. Although these compounds are named for their chemical rings, their side chains are believed to be more important to their functions. The TCAs with tertiary amine groups on their side chains tend not to be tolerated as well as the TCAs with secondary amine groups on their side chains. The TCAs with tertiary amine groups block the reuptake of 5-HT more strongly than they do NE, whereas the TCAs with secondary amine groups block the reuptake of NE more strongly than they do 5-HT. The nine TCAs currently marketed for the treatment of depression in the United States are doxepin, trimipramine, amoxapine, maprotiline, imipramine, amitriptyline, nortriptyline, protriptyline, and desipramine.

Doxepin, marketed under the brand name Sinequan, is labeled for use as a treatment for psychoneurotic individuals, alcoholic individuals, and individuals with an organic disease with comorbid depression, anxiety, or both, and for individuals with psychotic depressive disorders with anxiety. A formulation of doxepin is also marketed with the brand name Silenor to treat insomnia, and a cream with doxepin hydrochloride as its active ingredient is marketed with the brand name Zonalon for the short-term treatment of pruritus in adults with atopic dermatitis or lichen simplex chronicus. Trimipramine, with the brand name Surmontil; amoxapine, formerly with the brand name Asendin; maprotiline, with the brand name Ludiomil; imipramine, with the brand name Tofranil; a formulation combining amitriptyline hydrochloride with perphenazine, with the brand names Triavil 2–10, Triavil 2–25, Triavil 4–10, Triavil 4–25, and Triavil 4–50;

nortriptyline, with the brand name Pamelor; protriptyline, with the brand name Vivactil; and desipramine, with the brand name Norpramin, are marketed only for the treatment of depression. Additionally, a formulation combining amitriptyline hydrochloride with chlordiazepoxide, with the brand name Limbitrol, is marketed as a treatment for depression associated with anxiety. In addition to these nine TCAs, clomipramine, with the brand name Anafranil, is marketed only for the treatment of OCD in the United States but is marketed for the treatment of MDD in Europe.

Many MAOIs exist for the treatment of various pathologies. MAOIs act by inhibiting monoamine oxidase (MAO) enzymes in the nervous system. Since MAO is located on the outer surface of mitochondria, it can only deaminate species in the cytoplasm and not species inside organelles, thereby keeping the concentration of amines in the cytoplasm low unless inhibited. The inhibition of the MAO enzymes by the MAOIs is not thought to be the direct cause of the alleviation of the symptoms of depression as has been observed from treatment with MAOIs. Secondary effects of these drugs are thought to be important for their use for the treatment of depression. The MAOIs are not widely used to treat depression due to their risks, including the risk of hypertensive crisis.

The four MAOIs currently marketed in the United States for the treatment of depression are phenelzine, isocarboxazid, tranylcypromine, and selegiline. Phenelzine, with the brand name Nardil, is marketed for the treatment of atypical, non-endogenous, or neurotic individuals with depression. Isocarboxazid, with the brand name Marplan, is marketed for the treatment of depression. Tranylcypromine, with the brand name Parnate, is marketed for the treatment of major depressive episodes without melancholia. Selegiline, with the brand name Emsam, is marketed for the treatment of MDD.

Important safety issues must be noted with the use of antidepressant medications. The use of antidepressants in children and adolescents may increase depressive symptoms or cause the onset of suicidal ideation; therefore, appropriate discretion must be used when prescribing SRIs in

children, adolescents, or young adults with depression. Also, the concomitant use of SRIs and MAOIs is a known cause of serotonin syndrome which is potentially lethal; therefore, these drugs should not be prescribed concomitantly, and time should be allowed between discontinuation of one of these types of drugs and the initiation of treatment with a drug of the other type. At least 2 weeks must be allowed before beginning an MAOI after discontinuing most SSRIs, although at least 5 weeks must be allowed before beginning an MAOI after discontinuing fluoxetine. At least a few days must be allowed before beginning an MAOI after discontinuing a TCA.

MAOIs have adverse drug interactions with various drugs including the SRIs, serotonin agonists, stimulants, direct sympathomimetics, indirect sympathomimetics, and antidiabetic agents. Also, MAOIs have adverse interaction with some foods which is thought to be due to increased tyramine levels. Important safety precautions must be taken during the use of MAOIs including the avoidance of certain foods: cheese, overripe fruit, fava beans, sausage, salami, sherry, liqueurs, sauerkraut, monosodium glutamate, pickled fish, brewer's yeast, beef liver, chicken liver, fermented products, and red wine. Additionally, the following foods should be used in moderation during the use of MAOIs: coffee, chocolate, colas, tea, soy sauce, beer, and wines other than those previously listed which should be avoided during the use of MAOIs.

Future Directions

In recent years, the development of new, more efficacious antidepressants has been attempted by combining the effects of SRIs with other pharmacological effects. Other antidepressant drugs similar to the third-generation antidepressants are being developed with novel mechanisms of action. For example, a group of compounds known as serotonin-norepinephrine-dopamine reuptake inhibitors (SNDRIs) are being developed for potential clinical use in the treatment of depressive disorders. At least five SNDRI compounds have been developed. Other

compounds being developed and investigated are SRIs and 5-HT_{2A} antagonists; at least 27 of these compounds have been developed to date. The treatment of depression with a combination of SRIs with atypical antipsychotics is another approach being investigated. Other compounds with SRI activity have reportedly been developed, but these compounds require further testing to determine their viability as medications.

See Also

- [Serotonin Reuptake Inhibitors \(SRIs\)](#)
- [Serotonin Syndrome](#)

References and Reading

- Andreasen, N. C., & Black, D. W. (2001). *Introductory textbook of psychiatry* (3rd ed.). Washington, DC: American Psychiatric Publishing.
- Boland, R. J., & Keller, M. B. (2006). Treatment of depression. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 465–478). Washington, DC: American Psychiatric Publishing.
- Gillman, P. K. (2005). Monoamine oxidase inhibitors, opioid analgesics and serotonin toxicity. *British Journal of Anaesthesia*, 95, 434–441.
- Greden, J. F. (2006). Duloxetine and milnacipran. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 171–180). Washington, DC: American Psychiatric Publishing.
- Jacobsen, E. (1986). The early history of psychotherapeutic drugs. *Psychopharmacology*, 89, 138–144.
- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001). Treatment with antidepressants. In *Principles and practice of psychopharmacotherapy* (3rd ed., pp. 215–325). Philadelphia: Lippincott Williams & Wilkins.
- Kline, N. (1958). Clinical experience with iproniazid. *Journal of Clinical and Experimental Psychopathology*, 19, 72–78.
- Krishnan, K. R. R. (2006). Monoamine oxidase inhibitors. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 113–125). Washington, DC: American Psychiatric Publishing.
- Moltzen, E. K., & Bang-Andersen, B. (2006). Serotonin reuptake inhibitors: The corner stone in treatment of depression for half a century—a medicinal chemistry survey. *Current Topics in Medicinal Chemistry*, 6, 1801–1823.

- Nelson, J. C. (2006). Tricyclic and tetracyclic drugs. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 5–29). Washington, DC: American Psychiatric Publishing.
- Sadock, B. J., & Sadock, V. A. (2003). *Kaplan and Sadock's synopsis of psychiatry: Behavioral sciences/clinical psychiatry*. Philadelphia: Lippincott Williams & Wilkins.
- Sadock, B. J., & Sadock, V. A. (2005). *Kaplan and Sadock's pocket handbook of clinical psychiatry*. Philadelphia: Lippincott Williams & Wilkins.
- Stahl, S. M. (2000). Classical antidepressants, serotonin selective reuptake inhibitors, and noradrenergic reuptake inhibitors. In H. Meltzer (Ed.), *Essential psychopharmacology: Neuroscientific basis and clinical applications* (pp. 199–243). Cambridge: Cambridge University Press.
- Thase, M. E., & Sloan, D. M. E. (2006). Venlafaxine. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 465–478). Washington, DC: American Psychiatric Publishing.
- U.S. Food and Drug Administration. (2011). *Drugs@FDA*. Retrieved June 28, 2012 from <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm>

Antidepressants

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Synonyms

Selective serotonin reuptake inhibitors (SSRIs);
Tricyclic antidepressants (TCAs)

Definition

Antidepressants are a broad group of medications with various mechanisms of action. Until the early 1990s, the most commonly used antidepressants were the so-called tricyclic antidepressants (TCAs). More recently, the most commonly used antidepressants are the selective serotonin

reuptake inhibitors (SSRIs). Other antidepressants include bupropion, serotonin and noradrenaline reuptake inhibitors (SNRIs), and more unique medications like mirtazapine. Medications that are typically used as mood stabilizers (such as lithium) or antipsychotics (such as aripiprazole) also have efficacy for treating depression although they are not typically classified as antidepressants.

SSRIs are a class of medications that includes fluoxetine, fluvoxamine, paroxetine, sertraline, citalopram, and escitalopram. As the name suggests, SSRIs block the reuptake of released serotonin at the presynaptic serotonin transporter. This action prolongs the presence of serotonin in the synapse, which is a major neurotransmitter in the brain.

Neurotransmitters are used to communicate messages from one nerve to the next nerve. Unlike conventional electrical wiring, which requires wire-to-wire contact to move the electrical signal onward, nerve endings do not make physical contact with one another. The transmitting nerve ending comes near, but does not touch, the neighboring nerve ending. The message is carried by a neurotransmitter such as serotonin, dopamine, norepinephrine, or acetylcholine.

Once the neurotransmitter, such as serotonin, is released to carry the message to the neighboring neuron, there is a series of regulatory steps designed to return the system to the resting state. Reuptake, which is an important regulatory mechanism, is an active process of recovering released neurotransmitter back into the transmitting nerve ending. If a drug blocks serotonin reuptake, it permits the neurotransmitter to remain in the synaptic space longer. Although this is a known effect of the SSRIs, there is often a delay between starting the medication and achievement of beneficial effects. This suggests that the blockade of serotonin reuptake, which occurs with the first dose of medication, is not a complete explanation for the therapeutic effect of these medications.

The tricyclic antidepressants (abbreviated TCAs) are an older class of antidepressants that include imipramine, desipramine, clomipramine, amitriptyline, and nortriptyline. The term tricyclic refers to the three-ring structure of this class of antidepressant medications. These medications are not used as commonly as in the past as they

have been largely replaced by the SSRIs. Imipramine has been used to treat both depression and anxiety. Desipramine has been used to treat depression and attention deficit hyperactivity disorder. Clomipramine is often considered a breakthrough because it was the first medication shown to be effective for the treatment of obsessive-compulsive disorder. These three compounds, imipramine, desipramine, and clomipramine, represent three distinct modes of action for drugs in the same class. For example, desipramine has highly selective norepinephrine reuptake inhibitor properties. By contrast, clomipramine is well known for its more selective serotonin reuptake inhibiting properties. Indeed, clomipramine served as a model for the next generation of selective serotonin reuptake inhibitors (see below). Imipramine is intermediate with both norepinephrine and serotonin reuptake inhibiting properties. Other members of the class, such as nortriptyline and protriptyline, are predominately norepinephrine reuptake inhibitors.

The tricyclic antidepressants have several adverse effects in common including dry mouth, urinary retention, constipation, nausea, increased heart rate, dizziness, and, at higher doses, confusion. The tricyclic antidepressants also carry some risk of altering the electrical conduction in the heart. They are well known to be fatal on overdose due to their potential for causing cardiac arrhythmia. Because of their known toxicity at higher doses, treatment with tricyclic antidepressants requires blood-level monitoring and electrocardiogram monitoring as well. Finally, the tricyclic antidepressants are also vulnerable to drug-drug interaction. For example, some medications such as SSRIs or certain antibiotics may interfere with the breakdown of tricyclic antidepressant medications. The interference of metabolism of the tricyclic can cause a sharp increase in the blood levels of the tricyclic antidepressants and increase the vulnerability to toxic effects. The tricyclic medications have not been well studied in children or adults with autism.

See Also

- [Citalopram](#)
- [Clomipramine](#)

- [Escitalopram](#)
- [Fluoxetine](#)
- [Fluvoxamine](#)
- [Paroxetine](#)
- [Sertraline](#)

References and Reading

- Martin, A., Scahil, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.
- McDougle, C. J., Price, L. H., & Goodman, W. K. (1990). Fluvoxamine treatment of coincident autistic disorder and obsessive compulsive disorder: A case report. *Journal of Autism and Developmental Disorders*, 20, 537–543.
- McDougle, C. J., Price, L. H., Volkmar, F. R., Goodman, W. K., Ward-O'Brien, D., Nielsen, J., et al. (1992). Clomipramine in autism: Preliminary evidence of efficacy. *Journal of the American Academy of Child and Adolescent Psychiatry*, 31(4), 746–750.
- McDougle, C. J., Naylor, S. T., Cohen, D. J., Volkmar, F. R., Heninger, G. R., & Price, L. H. (1996). A double-blind, placebo-controlled study of fluvoxamine in adults with autistic disorder [comment]. *Archives of General Psychiatry*, 53(11), 1001–1008.

Antiepileptic Drugs (AEDs)

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Synonyms

[Anticonvulsants](#); [Antiseizure medications](#)

Indications

The decision about which antiepileptic drug (AED) to use is based on both the seizure type and the epilepsy syndrome along with the efficacy and toxicity profile of the AEDs for the various

types of seizures. A list of the most common AEDs used in autism spectrum disorders is described below. Each drug is listed with the following categories: indications, mechanism of action/metabolism, adverse effects, and dosing.

Mechanisms of Action

Mechanisms of action (provided under each entry below)

Specific Compounds and Properties

Phenobarbital

Phenobarbital (PB) is classified as a barbiturate and displays a broad spectrum of anticonvulsant activity. It was first introduced in 1912. It remains the oldest anticonvulsant commonly used and the most widely used around the world.

Indications: Phenobarbital is effective for generalized tonic-clonic seizures as well as partial seizures. PB is also effective for status epilepticus. It is usually the drug of choice for neonatal seizures. It is not effective for absence seizures.

Mechanism of action/metabolism: PB works by enhancing gamma-aminobutyric acid (GABA) inhibition. It is extensively metabolized in the liver. PB undergoes autoinduction whereby clearance may be increased requiring increasing dose adjustment when used as monotherapy.

Adverse effects: The most common adverse effect is sedation; however, tolerance to sedation usually develops with continued use of the drug. Other common side effects include irritability, hyperactivity, ataxia, and cognitive impairment. Decreased bone mineral density may occur. Rash occurs as an idiosyncratic reaction with very rare occurrence of Stevens-Johnson syndrome and toxic epidermal necrolysis. Other rare adverse effects include megaloblastic anemia and respiratory depression. Weight change is not common.

Dosing: PB is available in the following formulations: liquid (20 mg/5 ml), tablets (15 mg, 30 mg, 60 mg, 100 mg). Intravenous preparation is available. Average daily dosing is in the range of 4–11 mg/kg/day in children less than 1 year, 2–7 mg/kg/day for children over 1 year, and

1.5–4 mg/kg/day for children over age 12 and adults. Loading doses are effective ways of rapidly achieving a therapeutic level. PB should be gradually tapered after chronic use to avoid withdrawal seizures, usually over 3–6 months.

Phenytoin (Dilantin)

Phenytoin (PHT) was introduced in 1938 as being useful in controlling seizures without sedative effects as seen in phenobarbital. In addition to its use as an antiepileptic drug, it is also used in treatment of trigeminal neuralgia.

Indications: Phenytoin is effective for partial seizures as well as generalized tonic-clonic seizures. PHT is also highly effective for status epilepticus. It is useful in the treatment of neonatal seizures. It is not effective for absence and myoclonic seizures.

Mechanism of action/metabolism: PHT acts as a use-dependent blocker of voltage-sensitive sodium channels. It inhibits calcium channels and calcium sequestration. PHT is extensively metabolized in the liver. It undergoes autoinduction whereby clearance may be increased requiring increasing dose adjustment when used as monotherapy.

Adverse effects: Common adverse effects are cosmetic including acne, gingival hyperplasia, and hirsutism. Nausea, vomiting, nystagmus, vertigo, ataxia, and lethargy may occur with toxicity. Rare adverse effects include hyperglycemia, movement disorders, and confusional states. More serious side effects include rare hepatotoxicity and hematological abnormalities, including thrombocytopenia, anemia, leukopenia, and agranulocytosis. Other rare life-threatening effects include lymphadenopathy and serious rash including Stevens-Johnson syndrome and toxic epidermal necrolysis. Effect on weight is not common.

Dosing: PHT is available in the following formulations: chewable tablets (50 mg), capsules (30 mg, 100 mg). An oral suspension is available but discouraged from use as it is unstable. Intravenous preparation is available. Average daily dosing is in the range of 4–10 mg/kg/day for children and 300–400 mg/day for adults. Neonates may require more than 10 mg/kg/day.

As with most AEDs, discontinuation should be done with gradual dose reduction over several weeks, unless there is concern for serious adverse effect.

Carbamazepine (Tegretol)

Carbamazepine (CBZ) was initially marketed in 1962 for the treatment of trigeminal neuralgia and shortly after for the treatment of epilepsy. It is particularly effective in the treatment of focal epilepsies. In addition to its use as an anticonvulsant, it is beneficial for neuropathic pain and affective disorders including bipolar disorder.

Indications: Carbamazepine is effective in simple and complex partial seizures as well as generalized tonic-clonic seizures. It is not indicated for use in neonatal or febrile seizures. It is contraindicated in the treatment of generalized seizures seen in idiopathic generalized epilepsy, as well as absence seizures. It is not used in the treatment of epileptic encephalopathies.

Mechanism of action/metabolism: Carbamazepine acts as a use-dependent blocker of voltage-sensitive sodium channels. It inhibits the release of glutamate. CBZ is extensively metabolized in the liver. Carbamazepine exhibits autoinduction whereby clearance may be increased requiring increased dose adjustment when used as monotherapy.

Adverse effects: Common adverse effects include gastrointestinal distress, drowsiness, confusion, headaches, dizziness, ataxia, and blurred or double vision. Aplastic anemia, agranulocytosis, and liver toxicity are rare but nonetheless serious potential reactions that can occur with carbamazepine use. Therefore, hematologic and hepatic parameters should be monitored, especially in the first 6 months of therapy. Rare occurrence of severe rash, including Stevens-Johnson syndrome, and cardiac arrhythmias has been seen. CBZ may cause syndrome of inappropriate antidiuretic hormone (SIADH) with hyponatremia since it both increases the release and potentiates the action of ADH (vasopressin). Weight change is not typical.

Dosing: CBZ is available in the following formulations: liquid (100 mg/5 mL), chewable tablets (100 mg), tablets (200 mg), extended release

sprinkle capsule (Carbatrol) in 100 mg, 200 mg, 300 mg, and extended release tablets (Tegretol-XR) in 100 mg, 200 mg, 400 mg. Average daily dosing is in the range of 10–30 mg/kg/day for children and 600–1,200 mg/day for adults.

Valproic Acid (Depakote)

Valproic acid (VPA) is often referred to as valproate. Its anticonvulsant properties were first discovered in the early 1960's and since then has become one of the most commonly prescribed anticonvulsants worldwide. It is a broad-spectrum AED, effective against all types of seizures and epilepsies. In addition to its use as an AED, it is also used frequently for migraine prophylaxis and treatment of manic episodes of bipolar disorder.

Indications: VPA is highly effective in treatment of generalized epilepsies. It is effective for all types of generalized seizures including myoclonic and absence seizures. It is also used in the treatment of partial seizures. Febrile seizures, refractory status epilepticus, and epileptic encephalopathies, including Lennox-Gastaut syndrome, may be treated with VPA.

Mechanism of action/metabolism: The primary mechanism of action of VPA is not clear but may act by any one of the following: increasing levels of GABA by decreasing its metabolism, blocking voltage-gated sodium channels and T-type calcium channels, or decreasing levels of excitatory amino acid aspartate. VPA is extensively metabolized in the liver.

Adverse effects: Common adverse effects include mild sedation, nausea, vomiting, and anorexia. These side effects commonly occur during initiation of therapy and are usually transient. Alopecia and tremor may occur, but effects on cognition are minimal. The major serious adverse side effects relate to hepatic dysfunction. Fatal hepatotoxicity is considered to be an idiosyncratic reaction rather than a dose-related phenomenon. Children younger than 2 years old are at higher risk. Therefore, serum transaminases (AST, ALT) should be obtained prior to initiation of therapy and periodically during treatment. Thrombocytopenia more than leukopenia can occur and appears to be a dose-related phenomenon. Routine monitoring of CBC and platelets is usually

recommended. Fatal pancreatitis has been reported, albeit rare. If clinically indicated, serum amylase and lipase should be obtained. Hyperammonemia may occur and is often asymptomatic. Usual treatment is L-carnitine but exclusion of urea cycle disorders may be warranted. VPA should not be used in patients with suspected mitochondrial disorders. Weight gain is common.

Dosing: VPA is available in the following formulations: liquid (250 mg/5 mL), sprinkle capsules (125 mg), tablets (125 mg, 250 mg, 500 mg), and extended release tablet (250 mg, 500 mg). It is also available intravenously. Average daily doses are 30–60 mg/kg/day in children and 1,000–3,000 mg/day in adults. L-carnitine supplementation is suggested in certain individuals, especially in young children. As with most AEDs, discontinuation should be done with gradual dose reduction over several weeks, unless concern for serious adverse effect.

Oxcarbazepine (Trileptal)

Oxcarbazepine (OXC) is an analogue of carbamazepine (CBZ) with a keto group at the ten carbon position. It is rapidly metabolized to a 10-monohydroxy metabolite, which is primarily responsible for its anticonvulsant effects. Its anticonvulsant profile is nearly identical to CBZ, but it is better tolerated.

Indications: Oxcarbazepine is similar to carbamazepine in its antiepileptic efficacy. It is effective for simple and complex partial seizures as well as generalized tonic-clonic seizures. It may be particularly useful in individuals who do not tolerate CBZ but respond to CBZ. It is not indicated for use in neonatal or febrile seizures. It is contraindicated in the treatment of generalized seizures seen in idiopathic generalized epilepsy, as well as absence seizures. It is not used in the treatment of epileptic encephalopathies.

Mechanism of action/metabolism: Oxcarbazepine acts a use-dependent blocker of voltage-sensitive sodium channels. It inhibits the release of glutamate. Oxcarbazepine is rapidly metabolized in the liver to 10-hydroxycarbazepine, its pharmacologically active metabolite. Compared with CBZ, OXC has less prominent actions on CYP

450 enzyme systems and therefore, fewer pharmacokinetic interactions. It does not exhibit auto-induction, binds less to serum proteins, has fewer drug interactions, and thus, a lower incidence of side effects than CBZ.

Adverse effects: Common adverse effects include somnolence, headache, dizziness, blurred/double vision, nausea, and vomiting. There is risk of rash, including Stevens-Johnson syndrome and toxic epidermal necrolysis, but the risk is lower with OXC as compared with CBZ. There is a 25–30% incidence of cross-reactive rash with CBZ. As with CBZ, hyponatremia may occur. Hematologic effects, including agranulocytosis and aplastic anemia, are very rare. Hepatotoxicity is not a side effect, as in CBZ. Weight gain is not common.

Dosing: OXC is available in the following formulations: liquid (300 mg/5 mL), tablets (150 mg, 300 mg, 600 mg). Average daily doses are 600–1,200 mg/day for children less than 30 kg and 900–1,800 mg/day for children 30–60 kg. Average doses for adults are 600–2,400 mg/day.

Lamotrigine (Lamictal)

Lamotrigine (LTG) is a broad-spectrum antiepileptic drug that is used for all seizure types with the exception of epilepsies with prominent myoclonic jerks. In addition to its use as an AED, it is also used for treatment of bipolar disorder, migraines, and other headaches, along with trigeminal neuralgia and other neuropathic pain disorders.

Indications: Lamotrigine is effective for the treatment of both partial and generalized seizures, including absence seizures. It is also used in treating Lennox-Gastaut syndrome.

Mechanism of action/metabolism: Lamotrigine acts a use-dependent blocker of voltage-sensitive sodium channels. It inhibits the release of the excitatory amino acid, glutamate. LTG is extensively metabolized in the liver.

Adverse effects: Common adverse effects include rash. Nonspecific rashes occur in approximately 10% of patients and the vast majority of these are benign. However, rare cases of Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) have been reported. The

incidence of SJS and TEN is higher in individuals younger than 16 years of age. Concurrent use of valproic acid and rapid escalation of LTG doses are both thought to be risk factors for the development of these rashes. The risk of rash is thought to be higher in the first 6–8 weeks of therapy. However, SJS has developed in LTG monotherapy and after several months of therapy. Other common risks include headache, nausea, vomiting, diplopia, ataxia, and insomnia, especially when combined with carbamazepine. Hematologic and hepatic effects are rare. Weight gain is not common.

Dosing: LTG is available in the following formulations: chewable tablets (2 mg, 5 mg, 25 mg), orally disintegrating tablets (25 mg, 50 mg, 100 mg, 200 mg), tablets (25 mg, 100 mg, 150 mg, 200 mg), and extended release tablets (25 mg, 50 mg, 100 mg, 200 mg). Average daily doses vary depending on whether LTG is used as monotherapy or with Valproic Acid (VPA) or other enzyme-inducing AEDs. Enzyme-inhibiting drugs such as VPA increase LTG levels, whereas enzyme-inducing drugs such as PB, PHT, and CBZ decrease LTG levels. Therefore, initial and maintenance doses need to be adjusted accordingly. Slow dosage titration is recommended to reduce the risk of potential severe reactions, especially skin rash.

Levetiracetam (Keppra)

Levetiracetam is a broad-spectrum antiepileptic drug. It is widely used due to its low propensity for drug interactions, relatively benign side-effect profile, and effectiveness for nearly all types of epilepsies. It is also used for treatment of neuropathic or chronic pain.

Indications: Keppra is effective in the treatment of both partial and generalized seizures. It is not contraindicated for any seizure type, although experience in neonates and use for febrile seizures is limited. It is used in treatment of status epilepticus.

Mechanism of action/metabolism: The precise mechanism of action of levetiracetam has not yet been established. Levetiracetam is not metabolized in the liver, and thus, its metabolism does not depend on the hepatic cytochrome P450

enzymes. Therefore, doses do not need to be adjusted in those with hepatic impairment.

Adverse effects: Common adverse effects include somnolence, ataxia, and dizziness. Behavioral symptoms including irritability, agitation, aggression, emotional lability, anxiety, and depression may occur and are thought to be more common in children than adults. These symptoms are more common at initiation of the drug and often subside within the first few months of use. Use of pyridoxine (vitamin B6) has been suggested to decrease the occurrence of behavioral side effects, but this has not been proven in controlled data. Behavioral symptoms that persist often require discontinuation of the drug. Levetiracetam has no organ toxicity, and therefore, serious or life-threatening side effects are exceedingly rare. Weight gain is not common.

Dosing: Levetiracetam is available in the following formulations: 100 mg/ml (liquid), tablets (250 mg, 500 mg, 750 mg, 1,000 mg), and extended release tablets (500 mg). Intravenous solution is available. Average daily doses range between 30 and 60 mg/kg/day for children and 1,000–3,000 mg/day for adults.

Zonisamide (Zonegran)

Zonisamide is a broad-spectrum antiepileptic drug. It is not contraindicated for any particular type of epilepsy. In addition to its use as an anti-convulsant, it is used in treatment of migraines, obesity, and bipolar disorder.

Indications: Zonisamide is effective in the treatment of both partial and generalized seizures. It is the drug of choice for myoclonic seizures. It is useful in the management of epileptic encephalopathies along with Lennox-Gastaut syndrome and infantile spasms.

Mechanism of action/metabolism: The exact mechanism of action is not known. Although it may be a carbonic anhydrase inhibitor, this is not how it exerts its antiepileptic effects. It seems to block sodium and T-type calcium channels along with inhibiting the uptake of GABA and enhancing the uptake of glutamate.

Adverse effects: Common adverse effects include drowsiness, dizziness, ataxia, fatigue, nausea, vomiting, decreased appetite, and headache.

Metabolic acidosis, hypohidrosis, and cognitive/behavioral changes occur more commonly in children. Paresthesias and kidney stones are reported but uncommon. Life-threatening side effects such as Stevens-Johnson syndrome, blood dyscrasias, and hyperthermia are extremely rare. Use with caution when combining with other carbonic anhydrase inhibitors or anticholinergics due to risk for hypohidrosis and resultant hyperthermia. Weight loss is common.

Dosing: ZNM is available in the following formulations: capsules (25 mg, 50 mg, 100 mg). Average daily dosing for monotherapy in children is 8 mg/kg/day and 12 mg/kg/day when used with enzyme-inducing AEDs. Average daily doses range between 100–400 mg/day for adults.

Vigabatrin

Vigabatrin (VGB) is primarily used in the treatment of infantile spasms but is also effective in partial epilepsies. Due to its serious potential effects on vision, it had not been approved for use in the United States until 2009. It is now available for use as monotherapy for children ages 1 month to 2 years with infantile spasms and adjunctive therapy for adults with refractory complex partial seizures in whom the potential benefits outweigh the risks for vision loss.

Indications: Vigabatrin is effective against infantile spasms, especially if spasms are due to tuberous sclerosis. It is also used in the treatment of partial seizures. It is contraindicated in absence seizures and may provoke absence status epilepticus.

Mechanism of action/metabolism: Vigabatrin irreversibly inhibits GABA transaminase, the enzyme that breaks down GABA, effectively increasing GABA levels. Vigabatrin is not metabolized in the liver.

Adverse effects: The potential for visual field defects may be idiosyncratic, but dose- and duration-dependent toxicity has been reported. It has been reported in approximately 30% of patients. The onset usually occurs between 6 months and 2 years but is not typically reversible. Therefore, treatment with vigabatrin should not be continued if there is no response to treatment within 3 months. Other common adverse effects include somnolence, dizziness, headache,

and ataxia. Behavioral, mood, and cognitive changes are also reported. Life-threatening side effects are rare, including encephalopathic syndromes. Angioedema, hallucinations, and rash are rare. Weight gain is common.

Dosing: Vigabatrin is available in the following formulations: sachet, i.e., powder (500 mg), tablets (500 mg). Average daily doses for infants with infantile spasms are 100–200 mg/kg/day. Average doses for children are 2,000–3,000 mg/day and 1,000–3,000 mg/day for adults.

Topiramate (Topamax)

Topiramate (TPX) is a broad-spectrum antiepileptic drug that is used for all seizure types. In addition to its use as an AED, it is commonly used for migraine prophylaxis. It is also used in treatment of bipolar disorder and obesity.

Indications: Topiramate is effective for both partial and generalized seizures. It is also used in the treatment of infantile spasms, Lennox-Gastaut syndrome, and progressive and idiopathic myoclonic epilepsies. It is not contraindicated for any type of seizures.

Mechanism of action/metabolism: The exact mechanism of action is not known, but TPX appears to act by inhibiting voltage-dependent sodium channels, enhancing GABA-mediated inhibition, and decreasing glutamate-mediated excitatory neurotransmission. It also inhibits carbonic anhydrase, but this is not how it exerts its antiepileptic effects. It is metabolized in the liver, especially when used with enzyme-inducing AEDs.

Adverse effects: Common adverse effects include somnolence, mental slowing, impaired concentration or confusion, and word-finding difficulties. Paresthesias occur frequently with monotherapy, more frequently in adults than children. Other side effects include dizziness, weight loss, metabolic acidosis, and hypohidrosis. Rare side effects include nephrolithiasis and glaucoma. Serious side effects are related to metabolic acidosis and oligohidrosis that leads to hyperthermia. The risk of these is higher in children than in adults. Hepatotoxicity and bone marrow depression do not occur. Weight loss is common.

Dosing: TPX is available in the following formulations: sprinkle capsules (15 mg, 25 mg) and

tablets (25 mg, 50 mg, 100 mg, 200 mg). Average daily doses for children are 5–10 mg/kg/day and 200–400 mg/day for adults.

Benzodiazepines

Benzodiazepines have been used especially for the treatment of status epilepticus and repetitive or cluster seizures. They are commonly used as adjunctive agents or as temporary drugs while waiting to achieve therapeutic concentrations of mainstay therapy. Diazepam, lorazepam, and midazolam are used for status epilepticus while clobazepam, clonazepam, and clobazam are used for chronic anticonvulsant therapy. Clobazam is not available in the USA.

Major side effects include sedation, ataxia, and behavioral problems such as hyperactivity, irritability, moodiness, restlessness, and aggression. Disinhibition is common. Tolerance to benzodiazepines occurs frequently.

Diastat is the rectal gel preparation of diazepam that has been approved for use with acute repetitive seizures and cluster seizures. Although not approved for use in status epilepticus, it is used for treatment of prolonged seizures at home. It is usually recommended for seizures lasting greater than 5 min in duration. This is very useful as it allows caregivers to intervene early on and potentially avoid the need for emergency room care. It is supplied in doses of 2.5 mg, 5 mg, 10 mg, 15 mg, and 20 mg that is dosed by weight (0.5–0.3 mg/kg). Serious side effects are rare, including respiratory depression.

ACTH and Steroids

ACTH (adrenocorticotrophic hormone) is used in the treatment of infantile spasms. It is also used in other epileptic encephalopathies, such as Lennox-Gastaut syndrome, Landau-Kleffner syndrome, and Dravet syndrome. As such, ACTH is used almost exclusively in children.

Steroids, especially prednisone, have been used to treat acquired epileptic aphasia of childhood (Landau-Kleffner syndrome) and electrical status epilepticus of sleep (ESES). In these disorders, oral prednisone is most commonly used.

Common side effects include irritability, weight gain, hypertension, and hyperglycemia. Serious side effects include peptic ulcers, cataracts/

glaucoma, cardiomyopathy, and brain atrophy. Life-threatening adverse effects include immunosuppression, sepsis, and congestive heart failure.

Clinical Use (Including Side Effects)

AEDs are commonly administered to children and adolescents with ASD, both with and without epilepsy. Two of the most widely used AEDs in the ASD population include valproic acid and lamotrigine. As described herein, many AEDs have a psychotropic effect and are used in treating psychiatric symptoms and disorders, such as bipolar disorder, obsessive-compulsive disorder, mood lability, irritability, and aggressive behaviors. As many children with ASD have coexisting affective disorders, AEDs are an attractive drug of choice for targeting both mood disturbances as well as epilepsy. There are reports of behavioral improvements for children with ASD and epileptiform EEG abnormalities without clinical seizures; however, at present time, there are no data to support the use of antiepileptic drugs in the treatment of these abnormalities in the absence of clinical seizures. Whether these AEDs have a positive psychotropic effect on children with ASD, with and without epilepsy, is not currently known. There is a need for large randomized control trials in this area in order to determine the efficacy of these AEDs in treating the core symptoms of autism.

See Also

- [Depressive Disorder](#)
- [Epilepsy](#)
- [Mania](#)
- [Mood Disorders](#)
- [Seizures](#)

References and Reading

- Browne, T., & Holmes, G. (2008). *Handbook of epilepsy* (4th ed.). Philadelphia: Lippincott Williams & Wilkins.
- Di Martino, A., & Tuchman, R. (2001). Antiepileptic drugs: Affective use in autism spectrum disorders. *Pediatric Neurology*, 25(3), 199–207.

- Patsalos, P., & Bourgeois, B. (2010). *The epilepsy prescriber's guide to antiepileptic drugs*. Cambridge: Cambridge University Press.
- Pellock, J., Bourgeois, B., Dodson, E., Nordli, D., & Sankar, R. (2008). *Pediatric epilepsy: Diagnosis and therapy* (3rd ed.). New York: Demos Medical Publishing.
- Wyllie, E. (Ed.). (2011). *The treatment of epilepsy: Principles and practice* (5th ed.). Philadelphia: Lippincott Williams & Wilkins.

Antigluten Therapy

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Definition

Antigluten therapy is the elimination of gluten from the body by dieting and/or supplemental enzymes. This entry will examine enzyme supplements that break down gluten. (For elimination diets, see ► [“Gluten-Free Diet”](#).)

Historical Background

In 1979, Jaak Panksepp hypothesized that the symptoms of autism may be caused by an opiate excess, although he was unsure how such an excess might come about. Starting in the 1980s, some investigators reported abnormal peptide concentrations in the urine of children with autism and proposed that enzyme deficiencies caused this abnormality (Trygstad et al. 1980; Reichelt et al. 1981, 1990). Additionally, these investigators speculated that the abnormal peptide concentrations reflected abnormal levels of opioid peptides in the brain (Trygstad et al. 1980; Reichelt et al. 1981). More recently, Andrew Wakefield (Wakefield et al. 1998) described intestinal abnormalities in several children with autism and hypothesized that this abnormality could provide another explanation for the opiate-excess theory. More specifically, he hypothesized that

children with autism have a leaky gut that results in the release of opioids that enter blood vessels and circulate into the brain. However, Wakefield's study has since been retracted by The Lancet and is considered to be unreliable.

Rationale or Underlying Theory

Antigluten therapy is based on the opioid-excess, enzyme deficiency, and leaky gut theories. According to the opioid-excess theory, the core symptoms of autism may be explained by disrupted opiate activity in the brain (Panksepp 1979). One proposed explanation for this theory is that children with autism have deficient peptidase enzymes (Trygstad et al. 1980; Reichelt et al. 1981). However, Hunter et al. (2003) did not find dipeptidyl peptidase IV to be defective in children with autism. A “leaky gut” or increased intestinal permeability has also been theorized to cause an opioid excess and to be associated with autism. The theory suggests that undigested proteins and peptides leak into the bloodstream through the intestines, eventually causing damage and/or disrupted opioid receptor activity in the brain. However, this theory lacks empirical support as it is based on a discredited study (Wakefield et al. 1998) that found intestinal abnormalities in several children with autism and that has not been replicated by other investigators (Buie et al. 2010; Fernell et al. 2007; Sandhu et al. 2009).

Another uncorroborated theory that has been adduced to support antigluten therapy is that children with autism have a wheat allergy and other symptoms similar to celiac disease (Lucarelli et al. 1995). However, there is no evidence for increased co-occurrence of wheat allergies or celiac disease and autism spectrum disorders (Fitzgerald et al. 1999; McCarthy and Coleman 1979).

Goals and Objectives

The goals of antigluten therapy include administering digestive enzymes to assist in the breakdown of gluten and preventing undigested gluten and gluten derivatives from affecting the body.

Efficacy Information

For information on the efficacy of gluten-free diets, see ► [“Gluten-Free Diet”](#).

To date, there are only two empirical studies examining the efficacy of enzyme supplements to break down gluten for individuals with autism. Brudnack et al. (2002) placed 46 patients on a combination of several enzymes for 12 weeks. Several behavioral parameters were measured every 2 weeks for the entire 12 weeks. The authors report improvement on every measure including core symptoms. However, there was no control group, the baseline measures were assumed to be zero rather than measured directly, and behavioral evaluators were aware that the children had received a supplement. Additionally, behavioral measurements were collected from an “SOS” form (not shown or explained in the manuscript) in addition to scoring by an observer, and it is not clear whether the observer was distinct from a teacher, parent, or guardian who completed the SOS form. No standardized instruments (i.e., ADOS, Vineland, Mullen, etc.) were used. Despite the reported improvements, the numerous methodological weaknesses in the study make the results unreliable.

Munasinghe et al. (2010) conducted a more scientifically rigorous study that incorporated a randomized, double-blind, crossover design. Only food selection improved significantly at the month 2 measurements. Improvements were not sustained at 3 months. Thus, the two available studies provide insufficient information to recommend antigluten therapy in the form of enzymatic supplements at this time.

Outcome Measurement

Antigluten treatment is intended to reduce autism symptoms and improve adaptive functioning. Therefore, if clinical trials of antigluten therapy are undertaken, outcome measures should include measures of autism symptoms such as the ADOS and measures of adaptive behavior such as the Vineland. Also, because digestive enzymes are administered, measures of nutrition and vital signs should be included.

Qualifications of Treatment Providers

Caregivers should consult a board-certified physician before beginning antigluten therapy. Additionally, the use of enzyme supplements should be supervised by a physician.

See Also

- [Food Intolerance](#)
- [Gastrointestinal Disorders and Autism](#)
- [Gluten-Free Diet](#)
- [Leaky Gut Syndrome](#)
- [Nutritional Interventions](#)

References and Reading

- Brudnack, M. A., Rimland, B., Kerry, R. E., Dailey, M., Taylor, R., Stayton, B., et al. (2002). Enzyme-based therapy for autism spectrum disorders – Is it worth another look? *Medical Hypotheses*, 58, 422–428.
- Buie, T., Campbell, D. B., Fuchs III, G. J., Furuta, G. T., Levy, J., Van de Water, J., et al. (2010). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125, S1–S18.
- Fernell, E., Fagerberg, U. L., & Hellstrom, P. M. (2007). No evidence for a clear link between active intestinal inflammation and autism based on analyses of faecal calprotectin and rectal nitric oxide. *Acta Paediatrica*, 96, 1076–1079.
- Fitzgerald, M., Woods, M., & Matthews, P. (1999). Investigation of possible links between autism and celiac disease. *Autism*, 3, 193–195.
- Hunter, L. C., O'Hare, A., Herron, W. J., Fisher, L. A., & Jones, G. E. (2003). Opioid peptides and dipeptidyl peptidase in autism. *Developmental Medicine and Child Neurology*, 45, 121–128.
- Lucarelli, S., Frediani, T., Zingoni, A. M., Ferruzzi, F., Giardini, O., Quinteri, F., et al. (1995). Food allergy and infantile autism. *Panminerva Medica*, 37, 137–141.
- McCarthy, D. M., & Coleman, M. (1979). Response of intestinal mucosa to gluten challenge in autistic subjects. *The Lancet*, 314, 877–878.
- Munasinghe, S. A., Oliff, C., Finn, J., & Wray, J. A. (2010). Digestive enzyme supplementation for autism spectrum disorders: A double-blind randomized controlled trial. *Journal of Autism and Developmental Disorders*, 40(9), 1131–1138.
- Panksepp, J. (1979). A neurochemical theory of autism. *Trends in Neurosciences*, 2, 174–177.
- Reichelt, K. L., Ekrem, J., & Scott, H. (1990). Gluten, milk proteins and autism: Dietary intervention effects on

- behaviour and peptide secretion. *Journal of Applied Nutrition*, 42, 1–11.
- Reichelt, K. L., Hole, K., Hamberger, A., Saelid, G., Edminson, P. D., Braestrup, C. B., et al. (1981). Biologically active peptide-containing fractions in schizophrenia and childhood autism. *Advances in Biochemical Psychopharmacology*, 28, 627–643.
- Sandhu, B., Steer, C., Golding, J., & Emond, A. (2009). The early stool patterns of young children with autistic spectrum disorder. *Archives of Disease in Childhood*, 94, 497–500.
- Trygstad, O. E., Reichelt, K. L., Foss, I., Edminson, P. D., Selid, G., Bremer, J., et al. (1980). Patterns of peptides and protein-associated-peptide complexes in psychiatric disorders. *British Journal of Psychiatry*, 136, 59–72.
- Wakefield, A. J., Murch, S. H., Anthony, A., Linnell, J., Casson, D. M., Malik, M., et al. (1998). Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *The Lancet*, 351, 637–641.

Anti-Hist [OTC]

► Diphenhydramine

Antihistamines: Definition

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Synonyms

H₁ receptor antagonist; H₁ receptor inverse agonist

Definition

Antihistamines are a class of drugs that inhibit histamine, a neurotransmitter involved in mood and behavior regulation, by either (a) blocking

the action of histamine at the receptor, (b) competing with histamine for binding to the receptor, or (c) displacing histamine from the receptor. In the field of ASD, the majority of interest surrounds mirtazapine and cyproheptadine, both nonselective H₁ receptor (histamine receptor) inverse agonists (similar to antagonist). Mirtazapine specifically shows promise in treating inappropriate sexual behaviors associated with autism. However, the mechanism of action of both these drugs in the context of ASD is likely to be through antihistamines' off-target highly potent antagonism of α -adrenergic receptors (fight-or-flight response) and/or serotonin (mood regulation) receptors as opposed to the H₁ receptor. Antihistamines may be useful in the treatment of sleeping problems associated with autism; however, evidence suggests a greater effectiveness in this regard in typically developing children.

Histamine's more prominent role is as an inflammation mediator, increasing permeability of blood vessels to immune cells. In concordance, antihistamines are normally used in the treatment of allergies by blocking histamine-induced vasodilation and swelling. Common side effects include dry mouth, drowsiness, dizziness, nausea, and vomiting.

References and Reading

- Akhondzadeh, S., Erfani, S., Mohammadi, M. R., Tehrani-Doost, M., Amini, H., Gudarzi, S. S., & Yasamy, M. T. (2004). Cyproheptadine in the treatment of autistic disorder: A double-blind placebo-controlled trial. *Journal of Clinical Pharmacy and Therapeutics*, 29(2), 145–150.
- De Boer, T. (1996). The pharmacologic profile of mirtazapine. *Journal of Clinical Psychiatry*, 57, 19.
- Kanof, P. D., & Greengard, P. (1978). Brain histamine receptors as targets for antidepressant drugs. *Nature*, 272(5651), 329–333.
- Posey, D. J., Guenin, K. D., Kohn, A. E., Swiezy, N. B., & McDougle, C. J. (2001). A naturalistic open-label study of mirtazapine in autistic and other pervasive developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 11(3), 267–277.
- Prell, G. D., & Green, J. P. (1986). Histamine as a neuroregulator. *Annual Review of Neuroscience*, 9(1), 209–254.
- Stone, C. A., Wenger, H. C., Ludden, C. T., Stavorski, J. M., & Ross, C. A. (1961). Antiserotonin-antihistaminic

- properties of cyproheptadine. *Journal of Pharmacology and Experimental Therapeutics*, 131(1), 73–84.
- Thurmond, R. L., Gelfand, E. W., & Dunford, P. J. (2008). The role of histamine H1 and H4 receptors in allergic inflammation: The search for new antihistamines. *Nature Reviews Drug Discovery*, 7(1), 41–53.
- Woosley, R. L. (1996). Cardiac actions of antihistamines. *Annual Review of Pharmacology and Toxicology*, 36(1), 233–252.

Antipsychotic-Induced Dyskinesia

► Tardive Dyskinesia

Antipsychotics: Drugs

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Synonyms

Neuroleptics

Indications

Schizophrenia, bipolar mood disorder, significant irritability, and aggression

Mechanisms of Action

There has been considerable debate about the difference between the traditional antipsychotics and the so-called atypicals. Indeed, the matter is not completely settled. It is generally agreed that the traditional antipsychotics exert their beneficial and adverse effects through dopamine blockade at the dopamine D2 receptor. The traditional antipsychotic, haloperidol, is a potent blocker of dopamine. Its capacity to bind to D2 receptors is strong, and it is not easily displaced by dopamine in the brain. This affinity and persistent binding to D2 receptors in the basal ganglia probably

accounts for the motor side effects described above. By contrast, clozapine (often considered the prototype atypical) has much lower affinity for D2 receptors. Across the current list of atypical antipsychotics, the affinity for D2 receptors varies. For example, risperidone has strong affinity for D2 receptors – but it does not appear to have the same firm hold on these receptors as haloperidol does. Because the hold on the D2 receptors is not firm, endogenous dopamine is more able to bind to the receptors, and we are less likely to see the motor side effects associated with haloperidol.

Specific Compounds and Properties

Antipsychotics

Antipsychotic medications are a large group of medications developed primarily for the treatment of schizophrenia. The antipsychotic medications were introduced in the 1950s with so-called second-generation antipsychotics appearing in the 1990s. These newer medications have properties in common with the older antipsychotics but also important differences.

Atypical Antipsychotics

Because of the adverse neurological effects of the traditional antipsychotic medications, chemists looked for new compounds that would maintain the antipsychotic benefits with decreased risk of neurological side effects. This led to the introduction of the so-called atypical antipsychotics. These medications include clozapine, risperidone, olanzapine, quetiapine, ziprasidone, aripiprazole, asenapine, iloperidone, and lurasidone. As a class, these atypical antipsychotics, also called second-generation antipsychotics, do indeed reduce the risk of neurological adverse effects. However, depending on the actual drug discussed, they have varying degrees of risk for other adverse effects.

See Also

- Chlorpromazine
- Loxitane

- ▶ [Molindone](#)
- ▶ [Perphenazine](#)
- ▶ [Phenothiazine](#)

References and Reading

Martin, A., Seahil, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford.

Antiseizure Medications

- ▶ [Antiepileptic Drugs \(AEDs\)](#)

Antiyeast Therapy

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Definition

Antiyeast therapy includes diets that restrict intake of sugar and yeast as well as supplements and drugs to eliminate yeast from the body.

Historical Background

Candida albicans is a form of yeast and is known to cause human infections. In works published from 1978 to 1981, C. Orian Truss was the first to propose the idea that the natural yeasts in the gastrointestinal tract can cause a variety of symptoms (Edwards 1988). In 1983, William Crook popularized this idea with his book, *The Yeast Connection*, in which he coined the term “Candida syndrome” to explain a host of psychological and neurological syndromes, including learning disabilities. He offered anecdotes as evidence for this syndrome, notably an account of “Candida drunken syndrome” that involved a boy with autism who was said to improve after antiyeast

interventions. The popularity of antiyeast therapies for the treatment of autism increased after Shaw et al. (1995) reported abnormal metabolites in the urine of two brothers with autism.

Rationale or Underlying Theory

Current antiyeast therapy was developed by Shaw et al. (1995) based on a report of abnormally high levels of metabolites in the urine of two siblings with autism, compared to typically developing children. These metabolites included citramalic acid, a citric acid analog, tartaric acid, and a compound assumed to be arabinose. From their case report, Shaw et al. made three assumptions: (1) The metabolites are produced by an overgrowth of a fungus or fermenting yeast such as *Candida albicans*. (2) The overgrowth of fungus or yeast in individuals with autism occurs in the gastrointestinal system rather than the urinary tract. (3) The growth interferes with normal metabolism and produces symptoms of autism. None of these hypotheses have been confirmed by independent investigators in peer-reviewed studies, and some are considered biologically implausible (Lord 2003).

Goals and Objectives

The goal of antiyeast therapy is to rid the body of yeast. William Shaw (2008) recommends probiotics, antiyeast diet, and antifungal products as antiyeast therapies.

Treatment Procedures

Shaw (2008) offers tests for yeast overgrowth through his Great Plains Laboratory and recommends probiotics, antiyeast diet, and antifungal products as antiyeast therapies. Probiotics can be found in yogurt or purchased as supplements from pharmacies or health food stores. An antiyeast diet is a low-sugar diet with the simple mantra: “If it’s sweet, don’t eat”. Nonprescription antifungal supplements include garlic, oregano, caprylic acid, MCT oil, colloidal silver (although Shaw

acknowledges that this may be dangerous), lactoferrin, and biotin. Prescription antifungal drugs that are considered safe because they are poorly absorbed from the intestines include nystatin and amphotericin B. Other prescription antifungals which Shaw does not consider to be as safe are Sporanox (itraconazole) and Lamisil (terbinafine). The use of any prescription drug should be closely supervised by a board-certified physician.

Efficacy Information

Dr. Shaw reports that combining diet and antifungals “double[s] the effectiveness of diet alone in eliminating yeast overgrowth.” However, he does not cite any evidence for a link between autism and yeast and does not report on the validity of his lab tests. Furthermore, he concedes that no “formal assessments” are available to address the effects of the diet and antifungals on the core symptoms of autism (2008). At this time, no clinical trials have been performed, and only anecdotal evidence supports the application of antiyeast diets or medications. Levy and Hyman (2008) have categorized antifungal therapy as a Grade C treatment for autism, supported only by low-quality evidence. According to a consensus statement from an expert panel of clinicians, antiyeast therapy is not recommended for patients with autism spectrum disorders at this time (Buie et al. 2010).

Outcome Measurement

Antiyeast treatment is intended to reduce autism symptoms and improve adaptive functioning. Therefore, if clinical trials of antiyeast therapy are undertaken, outcome measures should include measures of autism symptoms such as the ADOS and measures of adaptive behavior such as the Vineland. Also, because the mechanism by which the treatment is postulated to work is to reduce intestinal yeast overgrowth, well-validated measures of intestinal yeast should be included.

Qualifications of Treatment Providers

A physician should be contacted before beginning an antiyeast therapy. The use of antifungal medications should have ongoing supervision from a board-certified physician.

See Also

- [Gastrointestinal Disorders and Autism](#)
- [Yeast Infection](#)

References and Reading

- Buie, T., Campbell, D. B., Fuchs III, G. J., Furuta, G. T., Levy, J., Van de Water, J., et al. (2010). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125, S1–S18.
- Edwards, J. E. (1988). Systemic symptoms of candida in the gut: Real or imaginary? *Bulletin of the New York Academy of Medicine*, 64, 544–549.
- Levy, S. E., & Hyman, S. L. (2008). Complementary and alternative medicine treatments for children with autism spectrum disorders. *Child and Adolescent Psychiatric Clinics of North America*, 17, 1–15.
- Lord, R. S. (2003). Urinary markers of intestinal yeast. *Townsend Letter for Doctors and Patients*, 245, 96–97.
- Shaw, W. (2008). Biological treatments for autism and PDD (3rd ed.). Lenexa: William Shaw.
- Shaw, W., Kassen, E., & Chaves, E. (1995). Increased urinary excretion of analogs of Krebs cycle metabolites and arabinose in two brothers with autistic features. *Clinical Chemistry*, 41, 1094–1104.

Anxiety

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Synonyms

[Fear](#); [Worry](#)

Definition

Anxiety is a psychological and physiological state characterized by cognitive (e.g., recurrent or obsessive thoughts), somatic (e.g., headache, dizziness, nausea), affective (e.g., dysphoria or negative mood), and behavioral (e.g., trembling, pacing, or restlessness) responses that arise as a result of a perceived threat to the individual. Evolutionarily, these responses are adaptive in allowing individuals to prepare themselves to either flee or fight when faced with a threat, increasing the likelihood of survival. Although periodic anxiety experienced at moderate levels is common to most individuals and can be adaptive, irrational or extreme anxiety over an extended length of time may be indicative of an anxiety disorder. Several studies have shown that children with a pervasive developmental disorder (PDD) exhibit rates of anxiety disorders significantly higher than typically developing children. In addition, it has been speculated that some core autism symptoms may be driven or exacerbated by anxiety and that some anxiety disorder symptoms overlap with PDD features such as perseverative thought and speech.

See Also

- [Amygdala](#)
- [Anxiety Disorders](#)
- [Cognitive Behavioral Therapy \(CBT\)](#)
- [General Anxiety](#)
- [Obsessive-Compulsive Disorder \(OCD\)](#)
- [Separation Anxiety Disorder](#)
- [Social Behaviors and Social Impairment](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.). Washington, DC: APA Press.
- Gillott, A., Furniss, F., & Walter, A. (2001). Anxiety in high-functioning children with Autism. *Autism*, 5(3), 277–286.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kim, J. A., Szatmari, P., Bryson, S. E., Streiner, D. L., & Wilson, F. J. (2000). The prevalence of anxiety and

mood problems among children with autism and Aspergers syndrome. *Autism*, 4(2), 117–132.

- McPheeters, M. L., Davis, A., Navarre, J. R., II, & Scott, T. A. (2010). Family report of ASD concomitant with depression or anxiety among US children. *Journal of Autism and Developmental Disorders*. Retrieved from <http://www.springerlink.com/content/00477205pj2p2383/fulltext.pdf>.

Anxiety and Depression from Adolescence to Old Age in Autism Spectrum Disorder

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Definition and Historical Background

The presence of anxiety in individuals with autism has been recognized since the original description by Leo Kanner (1943), who noted that a number of children from his original sample experienced anxiety in response to particular objects and situations and also in response to sensory stimuli such as loud sounds or mechanical noises. Kanner remarked that children with autism displayed an “anxiously obsessive desire for the maintenance of sameness” (p. 245) and further speculated that

some of the core autism features might be, at least in part, driven by high levels of anxiety. Almost 50 years ago, Rutter (1970) first reported depressive symptoms in a follow-up of an adolescent with autism. Despite these early descriptions and clear indications of pervasiveness and clinical impact, anxiety and depression have become a focus of autism research only in the past 20 years.

Epidemiology

Anxiety

Anxiety has been found to be significantly more prevalent and severe in autism when compared to both the general population (Kim et al. 2000) and a range of other neurodevelopmental and neuropsychiatric disorders, including, but not limited to, Down syndrome (Evans et al. 2005), specific language impairment (Gillott et al. 2001), and Williams syndrome (Rodgers et al. 2012b; Uljarević et al. 2018). Although exact prevalence has varied widely across studies, several relatively recent large-scale studies and systematic reviews have suggested that at least 40% of children and adolescents with autism meet the criteria for clinically significant anxiety (van Steensel et al. 2011; see also White et al. 2009). Prevalence of anxiety in adulthood is less well explored; however, it is clear that it is highly prevalent with recent studies suggesting that up to 60% of adults with autism may be affected (Buck et al. 2014; Lever and Geurts 2016; Uljarević et al. 2019). Results regarding the prevalence of specific anxiety disorders have been mixed. A meta-analysis by van Steensel et al. (2011; see also van Steensel et al. 2014) has suggested specific phobias (29.7%), obsessive-compulsive disorder (OCD; 17.4%), social phobia (16.6%), generalized anxiety disorder (GAD; 15.4%), separation anxiety disorder (9.0%), and panic disorder (1.8%) as the most frequent types of anxiety observed in the autism population. When discussing the prevalence of anxiety subtypes, it is important to note that the above reviewed studies relied on DSM-IV TR anxiety categories. To the best of our knowledge, studies are yet to explore the prevalence of DSM-5 anxiety subtypes. Importantly, several

studies (Halim et al. 2018; Lau et al. 2019; Kerns and Kendal 2012; Kerns et al. 2014) have also described a range of atypical fears and worries that, while common and clinically impactful in autism, do not fit within DSM frameworks.

Depression

Similar to anxiety, reported rates of depression have been highly variable across studies. In their meta-analysis of 66 studies, Hudson et al. (2019) found that rates of depressive disorder were highest when individuals were required to report their own symptoms or when a standardized interview was used, with lifetime rates ranging from 28.5% to 48.6% and current rates from 15.3% to 25.9%. Moreover, individuals with autism were four times more likely to experience lifetime depression than typically developing individuals (e.g., 11.7% to 16.6% in the United States) (Kessler et al. 2005; Merikangas et al. 2010). However, in their meta-analysis of co-occurring mental health diagnoses in autism which included 65 studies, Lai et al. (2019) reported an overall pooled prevalence for current depressive disorders of 11%. In a third meta-analysis, Hollocks et al. (2019) reported pooled current and lifetime rates of depressive disorder of 23% and 37%, respectively, although their analysis included fewer studies overall (29 studies). Finally, it is worth noting findings from a large population-based cohort study by Rai and Heuvelman et al. (2018b), which utilized data from the Stockholm Youth Cohort. Participants included 2,927 individuals with autism without intellectual disability, 1,146 with autism and intellectual disability, and 219,769 without autism, followed up to 27 years of age. Compared to the general population, 19.8% of individuals with autism had a diagnosis of depression by 27 years compared to 6% of the general population, and those with autism who did not have an intellectual disability had a greater relative risk (RR) of a depression diagnosis than those with co-occurring intellectual disability (adjusted RR 4.28 and 1.81, respectively). Furthermore, compared to their siblings without autism, those with autism had a twofold risk of a depression diagnosis in early adulthood.

Suicidal Ideation and Behavior. The prevalence of suicide is much higher in autism than in non-autism samples (Cassidy et al. 2018b; Hedley and Uljarević 2018; Hirvikoski et al. 2016). Systematic research studies of both autism and population samples, supplemented by personal accounts from autistic individuals, show that autistic traits are associated with a significantly increased vulnerability to suicidal ideation, suicide attempts, and deaths by suicide in autism (Adams 2019; Cassidy and Rodgers 2017; Hedley and Uljarević 2018; Luterman 2019; South et al. *in press*). This vulnerability seems to result from a range of individual and environmental factors including perceived social isolation (Cassidy and Rodgers 2017; Hedley et al. 2018b; Pelton and Cassidy 2017), pressure to “camouflage” autism traits to match societal norms (Hull et al. 2017; Leedham et al. 2019; Beck et al. *in press*), and difficulty accessing health services (Camm-Crosbie et al. 2018). Proposed relationships among these variables are complex and in need of further study (Culpin et al. 2018; Gotham et al. 2018; Hedley et al. 2017b; Maddox et al. 2017).

While mental health concerns such as depression and anxiety are associated with suicide risk both generally and in autism (Cassidy et al. 2014; Hedley et al. 2018b), links between mental health concerns, non-suicidal self-injury, and suicidal thoughts and behaviors in autism are not straightforward (Hannon and Taylor 2013; Hedley and Uljarević 2018; Maddox et al. 2017; Richa et al. 2014; Segers and Rawana 2014). One emerging factor that requires further study is that rates of suicide are more uniform between males and females in autism (Hirvikoski et al. 2016; Kirby et al. 2019), in contrast to non-autism populations where men die by suicide more frequently than women. Suggested reasons for more equal gender balance might include factors such as later identification and diagnosis in women and social and emotional differences. For example, increased social motivation and awareness in women with autism may exacerbate feelings of isolation (Ratto et al. 2018; White et al. 2017). Higher rates of camouflaging in women with autism may contribute critical vulnerability (Beck et al. *in press*; Cassidy et al. 2018b; Ratto et al. 2018; White et al. 2017).

Natural History and Outcomes

Anxiety and depression follow distinct and well-established developmental trajectories in the general population. While depression rates show an increase from young to middle adulthood followed by a protracted decline towards older adulthood (Kessler et al. 2005; Suttin et al. 2013), anxiety shows an earlier onset and steeper increase with higher rates observed in young and middle adulthood and moderate decline thereafter (Lee et al. 2016). Sex differences in rates of anxiety and depression are well established such that males experience lower levels of both disorders (Kessler et al. 2005). Of note, sex differences become more pronounced with increasing age (Kessler et al. 2005).

Only four studies to date have explored the course of anxiety and depression across the lifespan in autism. Davis et al. (2011) found that anxiety levels were higher in a group of children with autism aged 3–16 years ($n = 34$) when compared to a group of toddlers ($n = 40$), but lower than in a group of 30 individuals aged 20–48 years, which in turn showed lower anxiety levels than a smaller group ($n = 27$) of individuals aged between 49 and 65 years. Roy et al. (2015) reported lower frequency of anxiety and depression in 26 younger adults (age range: 20–40 years) when compared to 24 older adults (age range: 40–62 years) with autism. A study by Lever and Geurts (2016) explored the pattern of co-occurring psychiatric disorders, including anxiety and depression, in 52 younger (19–38 years), 72 middle-aged (39–54 years), and 48 older (55–79 years) adults with autism and reported that although the severity of both anxiety and depressive symptomatology was high across all age groups, the older group had significantly lower scores compared to the two younger adult groups. Finally, a recent study by Uljarević et al. (2019) examined age trends in anxious and depressive symptoms in a large, cross-sectional sample of 255 adolescents and adults with autism and found that at any life stage, more than one-third of participants reported clinically significant anxiety and depression. Uljarević and colleagues noted a slight increase in the severity of both

anxiety and depression from adolescence to middle adulthood and then a slight decrease in older adulthood; however, these changes were not statistically significant. Although longitudinal studies are missing, it is clear that both anxiety and depression remain prevalent problems across the lifespan of autism.

Anxiety and depression negatively impact concurrent and long-term outcomes. More specifically, elevated levels of anxiety and depression are associated with increased severity of restricted and repetitive behaviors (Uljarević et al. 2017a), a range of externalizing problems (Mattila et al. 2010), loneliness (White and Roberson-Nay 2009), suicidality (Hedley and Uljarević 2018), higher support needs and poorer employment outcomes (Hedley et al. 2017b), and increased parental levels of affective symptoms (Kerns et al. 2015).

Pathophysiology

Research to date has mainly focused on the role of (i) demographic factors, (ii) chronological and developmental age/cognitive functioning, and (iii) autism-specific traits and symptoms, as potential risk factors underlying the high rates of anxiety and depression in autism. More recently, studies have started to explore the role of transdiagnostic risk factors, most notably emotion regulation and intolerance of uncertainty, that have been found to be associated with a range of mental health outcomes across normative and atypical development.

Demographic Factors

A familial history and female sex/gender have been associated with higher prevalence and severity of anxiety and depression (Hedley et al. 2018b; Lai et al. 2019; Rai et al. 2018b; Uljarević et al. 2019). Of note, while adult samples show higher rates of depression in females than males (Hedley et al. 2018a; Lai et al. 2019), rates appear to be more similar for males and females during the school years (Greenlee et al. 2016). A recent meta-analysis by Hudson et al. (2019) reported that being White was associated with higher

lifetime prevalence rates of depression; however, this may also reflect underreporting or underdiagnosis in other races. Effects of race and ethnicity on the rates of anxiety have not been explored in detail.

Cognitive Functioning

Higher cognitive functioning has been associated with higher prevalence and severity of both anxiety (Hallett et al. 2013; Mayes et al. 2011; but see Duvekot et al. 2017 and Eussen et al. 2013 for nonsignificant findings) and depression (Hudson et al. 2019). However, a positive relationship between cognitive functioning and anxiety and depression may reflect expressed rather than experienced symptom levels (Strang et al. 2012). This assertion is supported by the fact that verbal intelligence quotient (IQ), as compared to non-verbal IQ, is more strongly associated with greater levels of anxiety (Gotham et al. 2013; Mayes et al. 2011).

Core Autism Symptoms

It has been hypothesized that core autism symptoms may increase the frequency and severity of everyday stressors, both directly and indirectly (Wood & Wood and Gadow 2010). Studies exploring the relationship between overall autism severity and anxiety have produced inconsistent result – positive (Wigham et al. 2015), negative (Eussen et al. 2013), and lack of significant relationships (Hollocks et al. 2016) have all been reported. However, given the multidimensional and multifactorial nature of autism, studies focusing on the relationship between anxiety and more fine-grained aspect of autism phenotype have produced more consistent results. For instance, preserved social motivation in combination with impaired social and communication skills may lead to repeated social failures, increased emotional pain, and isolation, which, in turn, contributes to the emergence of anxiety (Bellini 2004; Pickard et al. 2017; Phillips et al. 2019; Uljarević et al. 2020). Repetitive behaviors, in particular, insistence on sameness, have been suggested to have an important and somewhat circular role in the development and maintenance of anxiety. More specifically, drawing on the findings from

the normative literature, Uljarević, Richdale, McConachie, and colleagues (2017b; see also Leekam et al. 2011) hypothesized that although during early development insistence on sameness serves to constrain the unpredictability of the environment thus warding off fears and anxieties, over time, due to their inflexible and restrictive nature, these behaviors preclude development of more adaptive forms of self-regulation and thus lead to maintenance of anxiety. Indeed, insistence on sameness has been consistently associated with anxiety across a number of studies (see Leekam et al. 2011; Rodgers et al. 2012a; Gotham et al. 2013; for an overview). Finally, atypical sensory features, in particular sensory hypersensitivity, have been consistently implicated as risk factors for both overall anxiety (Kerns et al. 2014; Lidstone et al. 2014; Uljarević et al. 2016) and anxiety subtypes including specific phobias, GAD, and social phobia (Bitsika et al. 2016, 2019).

Severity of autistic traits has been found to predict depressive symptoms in autism (Hedley et al. 2018a; Rai et al. 2018a), the general population, and individuals experiencing first-episode psychosis (Uptegrove et al. 2018). Rai et al. (2018a) found that 10-year-old children with autism or high levels of autistic traits, particularly social communication impairments, reported higher depressive scores than the general population. The depressive levels remained elevated and on an upward trajectory until 18 years. Bullying accounted for a large portion of risk, and there may well be an interaction between autistic traits, social communication difficulties, and depression (Rai et al. 2018a). Similarly, psychosocial risk factors that may be exasperated by autism, such as loneliness and lack of friendships and social support, have also been shown to predict depressive scores (Hedley et al. 2018b; Mazurek 2014).

Transdiagnostic Factors

Emotion regulation is a complex process that is goal-directed and aims to modify the intensity, duration, and types of emotions experienced (Eisenberg and Spinrad 2004). Research findings in non-autistic populations indicate that the habitual use of certain strategies to regulate negative

emotions (e.g., expressive suppression, avoidance, denial, negative rumination) is related to an increase in internalizing symptoms including anxiety and depression (Aldao et al. 2010; Gross and John 2003). As a result, these strategies are viewed as maladaptive. The habitual use of other strategies to regulate positive emotions (e.g., savoring) and negative emotions (e.g., emotional acceptance) is related to reduced negative emotions, increased positive emotions, and better mental health (Aldao et al. 2010; Campbell-Sills et al. 2006; Troy et al. 2013). Hence, these strategies are typically categorized as adaptive.

Overall, research studies have found that individuals with autism use less adaptive emotion regulation strategies than individuals without autism (e.g., Bruggink et al. 2016; Rieffe et al. 2014; Samson et al. 2015). Findings around maladaptive strategy use are less consistent with studies showing more (e.g., Jahromi et al. 2012; Mazefsky et al. 2014), similar (Rieffe et al. 2014), and less frequent use of maladaptive strategies in autism (Samson et al. 2015). The majority of studies reporting higher internalizing and externalizing symptoms in those with autism relative to controls found individuals with autism used more maladaptive strategies and/or less adaptive ones (Cai et al. 2018c). Recent research examining interactions between adaptive and maladaptive emotion regulation strategy use in autism suggest that the higher use of an adaptive strategy might be a protective factor for psychological well-being in individuals who also show high use of maladaptive strategies (Cai et al. 2019, 2018b).

Intolerance of uncertainty is defined as the “dispositional incapacity to endure the aversive response triggered by the perceived absence of salient, key, or sufficient information, and sustained by the associated perception of uncertainty” (Carleton 2016, p. 31). Individuals who are high in intolerance of uncertainty tend to believe uncertainty is negative and threatening, find ambiguous situations stressful and avoid them, and have problems functioning in uncertain situations (Ladouceur et al. 2000). A meta-analysis by Gentes and Ruscio (2011) has provided robust evidence for the relationship between intolerance of uncertainty, anxiety, and

major depressive disorders. Although the role of intolerance of uncertainty in autism has started to be explored only relatively recently, emerging findings mirror results from the general population. More specifically, it has been consistently reported that children and adults with autism experience higher levels of intolerance of uncertainty when compared to non-autistic controls and that intolerance of uncertainty is associated with anxiety and depression in autism (Boulter et al. 2014; Cai et al. 2018a; Maisel et al. 2016).

Given that individuals with autism present with a maladaptive patterns of emotion regulation strategy use (Cai et al. 2018c) and higher levels of intolerance of uncertainty (Maisel et al. 2016), and both emotion regulation and intolerance of uncertainty strategy use are independently associated with affective symptoms in autism (Samson et al. 2015; Wigham et al. 2015), it is important to clarify the nature of the inter-relationship of intolerance of uncertainty and emotion regulation strategy use in predicting symptoms of anxiety and depression. Indeed, a recent study found that intolerance of uncertainty mediated the relationships between emotion regulation strategy use and symptoms of anxiety and depression (Cai et al. 2018a).

Evaluation and Differential Diagnosis

Depression and anxiety in autism might present differently compared to expression in non-autism clinical groups or might be less apparent. These differences can be attributed to several factors. Firstly, there is considerable overlap between the core autism symptoms and those attributed to anxiety and depression. For instance, while the lack of social approach and engagement can be considered as a symptom of social phobia, it can also be a manifestation of the social communication impairment that characterizes autism. Thus, while it is easy to identify anxiety disorder that manifests with social avoidance in individuals without autism, in the autism population, this symptom might be interpreted as a part of the core autism symptomatology. Consequently, co-occurring mental health disorders can remain undiagnosed and untreated (Uljarević

et al. 2016b). Further, individuals with autism might present with idiosyncratic symptoms including unusual specific phobias (e.g., vacuum cleaners, toilets), fears of change, or novelty; or depression might be expressed through reduction in circumscribed interests (Kerns and Kendal 2012; Uljarević et al. 2016a).

To address these issues, an increasing number of studies have focused on exploring the relevance of existing anxiety and depression measures in autism. For instance, a systematic review by Wigham and McConachie (2014) identified the Spence Children's Anxiety Scale (Spence 1998), the Revised Children's Anxiety and Depression Scale (Chorpita et al. 2000), and the Screen for Child Anxiety-Related Emotional Disorder (Birmaher et al. 1997) as robust outcome measures for Cognitive Behavior Treatment Trials. However, several studies failed to provide the support for the original factor structure of the Spence Children's Anxiety Scale in autism samples (Glod et al. 2017; Jitlina et al. 2017; Magiati et al. 2017). Cassidy et al. (2018a) conducted a systematic review and evaluated instruments that had been used to assess depression in adults with and without autism and without co-occurring intellectual disability. Only one study provided sufficient data to examine psychometric properties in people with autism, leading the authors to conclude that there was only weak support for the use of the Beck Depression Inventory (BDI-II) (Beck et al. 1996) in this population. With regard to other instruments, Uljarević, Richdale, McConachie, and colleagues (2017b) examined the factor structure and psychometric properties of the Hospital Anxiety and Depression Scale (HADS) (Zigmond and Snaith 1983) in representative sample of older adolescents and young adults with autism from the United Kingdom (UK) and Australia. Factor structure was similar to that found in the general population, internal consistency was acceptable, convergent validity was excellent, and divergent validity was found to be acceptable, thus providing support for the HADS in this population.

Given the variable performance of anxiety and depression measures originally designed for the general population when used in autism, and the noted specificity of symptom expression in

this population, several attempts have been made to modify existing instruments. For example, Rodgers et al. (2016) created the Anxiety Scale for Children – autism spectrum disorder (ASC-ASD) by modifying the Revised Children's Anxiety and Depression Scale (RCADS; Chorpita et al. 2000) to incorporate items related to intolerance of uncertainty, sensory oversensitivity, and phobias. The newly developed scale had good internal consistency, validity, and 1-month test-retest reliability. Kerns et al. (2014, 2015, 2016) have also shown that the Autism Spectrum Addendum (ASA) to the Anxiety Disorders Interview Schedule (ADIS; Silverman and Albano 1996) had satisfactory psychometric properties and convergent and discriminant validity in both low anxiety (Kerns et al. 2014) and non-treatment seeking (Kerns et al. 2015) samples of children with autism.

In summary, given that on the one hand, autism-related behaviors can be mistakenly interpreted as signs of a co-occurring anxiety and depression, and on the other hand, anxiety and depression symptoms can be expressed in atypical ways in individuals with autism, and either missed or ascribed to the underlying autism, careful consideration about how best to consider and capture the differential diagnoses of anxiety and depression in individuals with autism is needed. Design of new measures and improvements in the performance of existing measures are currently a priority in the field.

Treatment

Both psychopharmacological and psychosocial intervention approaches have been used to treat anxiety and depression in autism (White et al. 2009; Selles and Storch 2013; Vasa et al. 2014).

Pharmacological Treatments

Pharmacological treatments remain the primary method for treating mental health problems in individuals with autism (Kerns et al. 2016). Pharmacological treatments that have been studied include antidepressants and anxiolytics, but findings regarding treatment effects have been mixed and there is a lack of well-controlled studies looking at anxiety- and depression-

specific treatment effects (Selles and Storch 2013). It is important to highlight the fact that despite somewhat limited evidence concerning the efficacy of pharmacological treatments for anxiety and depression in autism, a review of medical and pharmacy claims data found that 64% of 33,565 children with autism were taking at least 1 psychotropic medication and 35% had evidence of polypharmacy with a median use length of 346 days (Spencer et al. 2013).

Psychosocial Treatments

Cognitive behavioral therapy (CBT), administered individually or in a group format, and mindfulness therapy, may be useful in treating anxiety and depression in individuals with autism (Kerns et al. 2016). One meta-analysis which examined the efficacy of CBT to treat affective disorders in children, adolescents, and adults with autism found CBT was superior to control conditions, returning medium effect sizes ($g = 0.45\text{--}0.59$) (Weston et al. 2016). Building on this evidence as well as robust findings from non-autistic populations that show emotion regulation is malleable to treatment (e.g., Blackledge and Hayes 2011), it has been proposed that targeting emotion regulation impairments may be an effective approach in treating anxiety and depression in the autism population (Weiss et al. 2014). Indeed, researchers have started designing intervention programs to improve emotion regulation in autism (Thomson et al. 2015). Programs aimed at reducing intolerance of uncertainty have also been developed (Rodgers et al. 2016, 2018). Given the inter-relationships between emotion regulation, intolerance of uncertainty, and psychopathology, we suggest that developing interventions aimed at improving the former two constructs may be more effective for improving affective symptoms in individuals with autism than just targeting one construct at a time.

Conclusion

Although the prevalence rates of anxiety and depression in autism have varied significantly across studies, based on the most up-to-date reviews and large-scale studies, it is clear that

both disorders are highly prevalent. The rate of current depressive disorder in autism is likely to range from 11% to 26%, and lifetime rates from 28% to 48%. The rate of anxiety disorders is likely to be around 40% in children and adolescents and up to 60% in adults. It is somewhat difficult to draw firm conclusions about the exact age-related trends of anxiety and depression in autism due to the fact that above reviewed studies were all cross-sectional rather than longitudinal and utilized different measurement instruments. However, it is clear that both anxiety and depression remain prevalent across the lifespan. Given that anxiety and depression have a significant negative impact on the functioning of individuals with autism and their families, often over and above the contribution of core-autism traits and developmental level, clarifying the link of autism with anxiety and depression is currently considered to be a priority in the field.

See Also

- [Emotion Regulation](#)
- [Functional Behavior-Based Cognitive-Behavioral Therapy for Obsessive-Compulsive Behavior in Children with ASD](#)
- [Psychopathology](#)
- [Suicidality in Children and Adolescents with Autism](#)

References and Reading

- Adams, J. (2019, May 3). "Where Do We Go from Here?" *Identifying the Top 10 Priorities to Prevent Suicide in Partnership with Autistic People*. International Society for Autism Research.
- Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review*, 30(2), 217–237. <https://doi.org/10.1016/j.cpr.2009.11.004>.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck depression inventory-II*. San Antonio: The Psychological Corporation.
- Beck, J. S., Lundwall, R. A., Gabrielsen, T. P., Cox, J. C., & South, M. (in press). Looking good but feeling bad: "Camouflaging" behaviors and mental health in women with autistic traits. *Autism. Special Issue on Mental Health Across the Lifespan*.
- Bellini, S. (2004). Social skill deficits and anxiety in high-functioning adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 19(2), 78–86. <https://doi.org/10.1177/10883576040190020201>.
- Birmaher, B., Khetarpal, S., Brent, D., Cully, M., Balach, L., Kaufman, J., & Neer, S. M. (1997). The screen for child anxiety related emotional disorders (SCARED): Scale construction and psychometric characteristics. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(4), 545–553.
- Bitsika, V., Sharpley, C. F., & Mills, R. (2016). Are sensory processing features associated with depressive symptoms in boys with an ASD? *Journal of Autism and Developmental Disorders*, 46(1), 242–252. <https://doi.org/10.1007/s10803-015-2569-4>.
- Bitsika, V., Arnold, W. A., & Sharpley, C. F. (2019). The role of sensory features in mediating associations between autism symptoms and anxiety in boys with autism spectrum disorder. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03917-1>.
- Blackledge, J. T., & Hayes, S. C. (2011). Emotion regulation in acceptance and commitment therapy. *Journal of Clinical Psychology*, 57(2), 243–255. [https://doi.org/10.1002/1097-4679\(200102\)57:2<243::aid-jclp9>3.0.co;2-x](https://doi.org/10.1002/1097-4679(200102)57:2<243::aid-jclp9>3.0.co;2-x).
- Boulter, C., Freeston, M., South, M., & Rodgers, J. (2014). Intolerance of uncertainty as a framework for understanding anxiety in children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(6), 1391–1402. <https://doi.org/10.1007/s10803-013-2001-x>.
- Bruggink, A., Huisman, S., Vuijk, R., Kraaij, V., & Gamefski, N. (2016). Cognitive emotion regulation, anxiety and depression in adults with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 22, 34–44. <https://doi.org/10.1016/j.rasd.2015.11.003>.
- Buck, T. R., Viskochil, J., Farley, M., Coon, H., McMahon, W. M., Morgan, J., & Bilder, D. A. (2014). Psychiatric comorbidity and medication use in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(12), 3063–3071. <https://doi.org/10.1007/s10803-014-2170-2>.
- Cai, R. Y., Richdale, A. L., Dissanayake, C., & Uljarević, M. (2018a). Brief report: Inter-relationship between emotion regulation, intolerance of uncertainty, anxiety, and depression in youth with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(1), 316–325. <https://doi.org/10.1007/s10803-017-3318-7>.
- Cai, R. Y., Richdale, A. L., Foley, K., Trollor, J., & Uljarević, M. (2018b). Brief report: Cross-sectional interactions between expressive suppression and cognitive reappraisal and its relationship with depressive symptoms in autism spectrum disorder. *Research in*

- Autism Spectrum Disorders*, 45, 1–8. <https://doi.org/10.1016/j.rasd.2017.10.002>.
- Cai, R. Y., Richdale, A. L., Uljarević, M., Dissanayake, C., & Samson, A. C. (2018c). Emotion regulation in autism spectrum disorder: Where we are and where we need to go. *Autism Research*, 11(7), 962–978. <https://doi.org/10.1002/aur.1968>.
- Cai, R. Y., Richdale, A. L., Dissanayake, C., Trollor, J., & Uljarević, M. (2019). Emotion regulation in autism: Reappraisal and suppression interactions. *Autism*, 3(3), 737–749. <https://doi.org/10.1177/1362361318774558>.
- Camm-Crosbie, L., Bradley, L., Shaw, R., Baron-Cohen, S., & Cassidy, S. (2018). “People like me don’t get support”: Autistic adults’ experiences of support and treatment for mental health difficulties, self-injury and suicidality. *Autism: The International Journal of Research and Practice*. <https://doi.org/10.1177/1362361318816053/>.
- Campbell-Sills, L., Barlow, D. H., Brown, A., & Hofmann, S. G. (2006). Acceptability and suppression of negative emotion in anxiety and mood disorders. *Emotion*, 6(4), 587–595. <https://doi.org/10.1037/1528-3542.6.4.587>.
- Carleton, R. N. (2016). Into the unknown: A review and synthesis of contemporary models involving uncertainty. *Journal of Anxiety Disorders*, 39, 30–43. <https://doi.org/10.1016/j.janxdis.2016.02.007>.
- Cassidy, S., & Rodgers, J. (2017). Understanding and prevention of suicide in autism. *The Lancet Psychiatry*, 4(6), e11. [https://doi.org/10.1016/S2215-0366\(17\)30162-1](https://doi.org/10.1016/S2215-0366(17)30162-1).
- Cassidy, S., Bradley, P., Robinson, J., Allison, C., McHugh, M., & Baron-Cohen, S. (2014). Suicidal ideation and suicide plans or attempts in adults with Asperger’s syndrome attending a specialist diagnostic clinic: A clinical cohort study. *The Lancet Psychiatry*, 1(2), 142–147. [https://doi.org/10.1016/S2215-0366\(14\)70248-2](https://doi.org/10.1016/S2215-0366(14)70248-2).
- Cassidy, S. A., Bradley, L., Bowen, E., Wigham, S., & Rodgers, J. (2018a). Measurement properties of tools used to assess depression in adults with and without autism spectrum conditions: A systematic review. *Autism Research*, 11(5), 738–754. <https://doi.org/10.1002/aur.1922>.
- Cassidy, S., Bradley, L., Shaw, R., & Baron-Cohen, S. (2018b). Risk markers for suicidality in autistic adults. *Molecular Autism*, 9, 42. <https://doi.org/10.1186/s13229-018-0226-4>.
- Chorpita, B. F., Yim, L., Moffitt, C., Umemoto, L. A., & Francis, S. E. (2000). Assessment of symptoms of DSM-IV anxiety and depression in children: A revised child anxiety and depression scale. *Behaviour Research and Therapy*, 38(8), 835–855. [https://doi.org/10.1016/S0005-7967\(99\)00130-8](https://doi.org/10.1016/S0005-7967(99)00130-8).
- Culpin, I., Mars, B., Pearson, R. M., Golding, J., Heron, J., Bubak, I., Carpenter, P., Magnusson, C., Gunnell, D., & Rai, D. (2018). Autistic traits and suicidal thoughts, plans, and self-harm in late adolescence: Population-based cohort study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 57(5), 313.e6–320.e6. <https://doi.org/10.1016/j.jaac.2018.01.023>.
- Davis, T. E., Hess, J. A., Moree, B. N., Fodstad, J. C., Dempsey, T., Jenkins, W. S., & Matson, J. L. (2011). Anxiety symptoms across the lifespan in people diagnosed with autistic disorder. *Research in Autism Spectrum Disorders*, 5(1), 112–118. <https://doi.org/10.1016/j.rasd.2010.02.006>.
- Duvekot, J., van der Ende, J., Verhulst, F. C., Slappendel, G., van Daalen, E., Maras, A., & Greaves-Lord, K. (2017). Factors influencing the probability of a diagnosis of autism spectrum disorder in girls versus boys. *Autism: The International Journal of Research and Practice*, 21(6), 646–658. <https://doi.org/10.1177/1362361316672178>.
- Eisenberg, N., & Spinrad, T. L. (2004). Emotion-related regulation: Sharpening the definition. *Child Development*, 75(2), 334–339.
- Eussen, M. L., Van Gool, A. R., Verheij, F., De Nijs, P. F., Verhulst, F. C., & Greaves-Lord, K. (2013). The association of quality of social relations, symptom severity and intelligence with anxiety in children with autism spectrum disorders. *Autism*, 17(6), 723–735. <https://doi.org/10.1177/1362361312453882>.
- Evans, D. W., Canavera, K., Kleinpeter, F. L., Maccubbin, E., & Taga, K. (2005). The fears, phobias and anxieties of children with autism spectrum disorders and Down syndrome: Comparisons with developmentally and chronologically age matched children. *Child Psychiatry & Human Development*, 36(1), 3–26.
- Gentes, E. L., & Ruscio, A. M. (2011). A meta-analysis of the relation of intolerance of uncertainty to symptoms of generalized anxiety disorder, major depressive disorder, and obsessive-compulsive disorder. *Clinical Psychology Review*, 31(6), 923–933. <https://doi.org/10.1016/j.cpr.2011.05.001>.
- Gillott, A., Furniss, F., & Walter, A. (2001). Anxiety in high-functioning children with autism. *Autism*, 5(3), 277–286. <https://doi.org/10.1177/1362361301005003005>.
- Glod, M., Creswell, C., Waite, P., Jamieson, R., McConachie, H., Don South, M., & Rodgers, J. (2017). Comparisons of the factor structure and measurement invariance of the Spence children’s anxiety scale-parent version in children with autism spectrum disorder and typically developing anxious children. *Journal of Autism and Developmental Disorders*, 47(12), 3834–3846. <https://doi.org/10.1007/s10803-017-3118-0>.
- Gotham, K., Bishop, S. L., Hus, V., Huerta, M., Lund, S., Buja, A., . . . Lord, C. (2013). Exploring the relationship between anxiety and insistence on sameness in autism spectrum disorders. *Autism Research*, 6(1), 33–41. <https://doi.org/10.1002/aur.1263>.
- Gotham, K. O., Siegle, G. J., Han, G. T., Tomarken, A. J., Crist, R. N., Simon, D. M., & Bodfish, J. W. (2018). Pupil response to social-emotional material is associated with rumination and depressive symptoms in

- adults with autism spectrum disorder. *PLoS One*, 13(8), e0200340. <https://doi.org/10.1371/journal.pone.0200340>.
- Greenlee, J. L., Mosley, A. S., Shui, A. M., Veenstra-VanderWeele, J., & Gotham, K. O. (2016). Medical and behavioral correlates of depression history in children and adolescents with autism spectrum disorder. *Pediatrics*, 137(Supplement 2), S105–S114. <https://doi.org/10.1542/peds.2015-2851I>.
- Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology*, 85(2), 348–362. <https://doi.org/10.1037/0022-3514.85.2.348>.
- Halim, A. T., Richdale, A. L., & Uljarević, M. (2018). Exploring the nature of anxiety in young adults on the autism spectrum: A qualitative study. *Research in Autism Spectrum Disorders*, 55, 25–37. <https://doi.org/10.1016/j.rasd.2018.07.006>.
- Hallett, V., Lecavalier, L., Sukhodolsky, D. G., Cipriano, N., Aman, M. G., McCracken, J. T., ... Scahill, L. (2013). Exploring the manifestations of anxiety in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(10), 2341–2352. <https://doi.org/10.1007/s10803-013-1775-1>.
- Hannon, G., & Taylor, E. P. (2013). Suicidal behaviour in adolescents and young adults with ASD: Findings from a systematic review. *Clinical Psychology Review*, 33(8), 1197–1204. <https://doi.org/10.1016/j.cpr.2013.10.003>.
- Harrop, C., Jones, D., Zheng, S., Nowell, S., Schultz, R., & Parish-Morris, J. (2019). Visual attention to faces in children with autism spectrum disorder: Are there sex differences? *Molecular Autism*, 10(1), 28. <https://doi.org/10.1186/s13229-019-0276-2>.
- Hedley, D., & Uljarević, M. (2018). Systematic review of suicide in autism spectrum disorder: Current trends and implications. *Current Developmental Disorders Reports*, 5(1), 65–76. <https://doi.org/10.1007/s40474-018-0133-6>.
- Hedley, D., Uljarević, M., Cameron, L., Halder, S., Richdale, A., & Dissanayake, C. (2017a). Employment programs and interventions targeting adults with autism spectrum disorder: A systematic review of the literature. *Autism*, 21, 929–941.
- Hedley, D., Uljarević, M., Wilmot, M., Richdale, A., & Dissanayake, C. (2017b). Brief report: Social support, depression and suicidal ideation in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(11), 3669–3677. <https://doi.org/10.1007/s10803-017-3274-2>.
- Hedley, D., Uljarević, M., Foley, K. R., Richdale, A., & Trollor, J. (2018a). Risk and protective factors underlying depression and suicidal ideation in Autism Spectrum Disorder. *Depression and Anxiety*, 35(7), 648–657. <https://doi.org/10.1002/da.22759>.
- Hedley, D., Uljarević, M., Wilmot, M., Richdale, A., & Dissanayake, C. (2018b). Understanding depression and thoughts of self-harm in autism: A potential mechanism involving loneliness. *Research in Autism Spectrum Disorders*, 46, 1–7. <https://doi.org/10.1016/j.rasd.2017.11.003>.
- Hirvikoski, T., Mittendorfer-Rutz, E., Boman, M., Larsson, H., Lichtenstein, P., & Bölte, S. (2016). Premature mortality in autism spectrum disorder. *The British Journal of Psychiatry*, 208(3), 232–238. <https://doi.org/10.1192/bjp.bp.114.160192>.
- Hollocks, M. J., Pickles, A., Howlin, P., & Simonoff, E. (2016). Dual cognitive and biological correlates of anxiety in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46(10), 3295–3307. <https://doi.org/10.1007/s10803-016-2878-2>.
- Hollocks, M. J., Lerh, J. W., Magiati, I., Meiser-Stedman, R., & Brugha, T. S. (2019). Anxiety and depression in adults with autism spectrum disorder: A systematic review and meta-analysis. *Psychological Medicine*, 49(4), 559–572. <https://doi.org/10.1017/s0033291718002283>.
- Hudson, C. C., Hall, L., & Harkness, K. L. (2019). Prevalence of depressive disorders in individuals with autism spectrum disorder: A meta-analysis. *Journal of Abnormal Child Psychology*, 1–11. <https://doi.org/10.1007/s10802-018-0402-1>.
- Hull, L., Petrides, K. V., Allison, C., Smith, P., Baron-Cohen, S., Lai, M.-C., & Mandy, W. (2017). “Putting on my best normal”: Social camouflaging in adults with autism spectrum conditions. *Journal of Autism and Developmental Disorders*, 47(8), 2519–2534. <https://doi.org/10.1007/s10803-017-3166-5>.
- Jahromi, L. B., Meek, S. E., & Ober-Reynolds, S. (2012). Emotion regulation in the context of frustration in children with high functioning autism and their typical peers. *Journal of Child Psychology and Psychiatry*, 53(12), 1250–1258. <https://doi.org/10.1111/j.1469-7610.2012.02560.x>.
- Jitlina, K., Zumbo, B., Mirenda, P., Ford, L., Bennett, T., Georgiades, S., ... Elsabbagh, M. (2017). Psychometric properties of the Spence children’s anxiety scale: Parent report in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(12), 3847–3856. <https://doi.org/10.1007/s10803-017-3110-8>.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kerns, C. M., & Kendal, P. C. (2012). The presentation and classification of anxiety in autism spectrum disorder. *Clinical Psychology: Science and Practice*, 19(4). <https://doi.org/10.1111/cpsp.12013>.
- Kerns, C. M., Kendall, P. C., Berry, L., Souders, M., Franklin, M. E., ... Herrington, J. (2014). Traditional and atypical presentations of anxiety in youth with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(11), 2851–61.
- Kerns, C. M., Kendall, P. C., Zickgraf, H., Franklin, M. E., Miller, J., & Herrington, J. (2015). Not to be overshadowed or overlooked: Functional impairments

- associated with comorbid anxiety disorders in youth with ASD. *Behavior Therapy*, 46(1), 29–39.
- Kerns, C. M., Roux, A. M., Connell, J. E., & Shattuck, P. T. (2016). Adapting cognitive behavioral techniques to address anxiety and depression in cognitively able emerging adults on the Autism Spectrum. *Cognitive and Behavioral Practice*, 23(3), 329–340. <https://doi.org/10.1016/j.cbpra.2016.06.002>.
- Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62(6), 617–627. <https://doi.org/10.1001/archpsyc.62.6.617>.
- Kim, J. A., Szatmari, P., Bryson, S. E., Streiner, D. L., & Wilson, F. J. (2000). The prevalence of anxiety and mood problems among children with autism and Asperger syndrome. *Autism*, 4(2), 117–132. <https://doi.org/10.1177/1362361300004002002>.
- Kirby, A. V., Bakian, A. V., Zhang, Y., Bilder, D. A., Keeshin, B. R., & Coon, H. (2019). A 20-year study of suicide death in a statewide autism population. *Autism Research*, 12, 658. <https://doi.org/10.1002/aur.2076>.
- Kumazaki, H., Muramatsu, T., Kosaka, H., Fujisawa, T. X., Iwata, K., Tomoda, A., Tsuchiya, K., & Mimura, M. (2015). Sex differences in cognitive and symptom profiles in children with high functioning autism spectrum disorders. *Research in Autism Spectrum Disorders*, 13–14, 1–7. <https://doi.org/10.1016/j.rasd.2014.12.011>.
- Ladouceur, R., Dugas, M. J., Freeston, M. H., Léger, E., Gagnon, F., & Thibodeau, N. (2000). Efficacy of a cognitive-behavioral treatment for generalized anxiety disorder: Evaluation in a controlled clinical trial. *Journal of Consulting and Clinical Psychology*, 68(6), 957–964. <https://doi.org/10.1037/0022-006X.68.6.957>.
- Lai, M.-C., Lombardo, M. V., Chakrabarti, B., Ruigrok, A. N., Bullmore, E. T., Suckling, J., Auyeung, B., Happé, F., Szatmari, P., Baron-Cohen, S., & MRC AIMS Consortium. (2018). Neural self-representation in autistic women and association with “compensatory camouflaging.” *Autism: The International Journal of Research and Practice*. <https://doi.org/10.1177/1362361318807159>.
- Lai, M. C., Kasse, C., Besney, R., Bonato, S., Hull, L., Mandy, W., ... Ameis, S. H. (2019). Prevalence of co-occurring mental health diagnoses in the autism population: A systematic review and meta-analysis. *The Lancet Psychiatry*, 6(10), 819–829. [https://doi.org/10.1016/S2215-0366\(19\)30289-5](https://doi.org/10.1016/S2215-0366(19)30289-5).
- Lau, B.-Y., Leong, R., Uljarević, M., Lehr, J. W., Rodgers, J., Hollocks, M. J., ... Magiati, I. (2019). Anxiety in young people with autism spectrum disorder: Common and autism-related anxiety experiences and their associations with individual characteristics. *Autism*. (in press). <https://doi.org/10.1177/1362361319886246>.
- Lee, T. T., Hill, M. N., & Lee, F. S. (2016). Developmental regulation of fear learning and anxiety behavior by endocannabinoids. *Genes, Brain and Behavior*, 15(1), 108–124. <https://doi.org/10.1111/gbb.12253>.
- Leedham, A., Thompson, A., Smith, R., & Freeth, M. (2019). ‘I was exhausted trying to figure it out’: The experiences of females receiving an autism diagnosis in middle to late adulthood. *Autism*. <https://doi.org/10.1177/1362361319853442>.
- Leekam, S. R., Prior, M. R., & Uljarevic, M. (2011). Restricted and repetitive behaviors in autism spectrum disorders: A review of research in the last decade. *Psychological Bulletin*, 137(4), 562–593. <https://doi.org/10.1037/a0023341>.
- Lever, A. G., & Geurts, H. M. (2016). Psychiatric co-occurring symptoms and disorders in young, middle-aged, and older adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(6), 1916–1930. <https://doi.org/10.1007/s10803-016-2722-8>.
- Lidstone, J., Uljarević, M., Sullivan, J., Rodgers, J., McConachie, H., Freeston, M., ... Leekam, S. (2014). Relations among restricted and repetitive behaviors, anxiety and sensory features in children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 8(2), 82–92. <https://doi.org/10.1016/j.rasd.2013.10.001>.
- Luterman, S. (2019, March 12). *Call for help: We need to address suicide risk in autistic women*. <https://www.spectrumnews.org/opinion/viewpoint/call-help-need-address-suicide-risk-autistic-women/>
- Maddox, B. B., Trubanova, A., & White, S. W. (2017). Untended wounds: Non-suicidal self-injury in adults with autism spectrum disorder. *Autism: The International Journal of Research and Practice*, 21(4), 412–422. <https://doi.org/10.1177/1362361316644731>.
- Magiati, I., Lerh, J. W., Hollocks, M. J., Uljarevic, M., Rodgers, J., McConachie, H., ... Simonoff, E. (2017). The measurement properties of the Spence children’s anxiety scale-parent version in a large international pooled sample of young people with autism spectrum disorder. *Autism Research*, 10(10), 1629–1652. <https://doi.org/10.1002/aur.1809>.
- Maisel, M. E., Stephenson, K. G., South, M., Rodgers, J., Freeston, M. H., & Gaigg, S. B. (2016). Modeling the cognitive mechanisms linking autism symptoms and anxiety in adults. *Journal of Abnormal Psychology*, 125(5), 692–703. <https://doi.org/10.1037/abn0000168>.
- Mattila, M. L., Hurtig, T., Haapsamo, H., Jussila, K., Kuusikko-Gauffin, S., Kielinen, M., ... Moilanen, I. (2010). Comorbid psychiatric disorders associated with Asperger syndrome/high-functioning autism: A community- and clinic-based study. *Journal of Autism and Developmental Disorders*, 40(9), 1080–1093. <https://doi.org/10.1007/s10803-010-0958-2>.
- Mayes, S. D., Calhoun, S. L., Murray, M. J., & Zahid, J. (2011). Variables associated with anxiety and depression in children with autism. *Journal*

- of *Developmental and Physical Disabilities*, 23, 325–337. <https://doi.org/10.1016/j.rasd.2010.06.012>.
- Mazefsky, C. A., Borue, X., Day, T. N., & Minshew, N. J. (2014). Emotion regulation patterns in adolescents with high-functioning autism spectrum disorder: Comparison to typically developing adolescents and association with psychiatric symptoms. *Autism Research*, 7(3), 344–354. <https://doi.org/10.1002/aur.1366>.
- Mazurek, M. O. (2014). Loneliness, friendship, and well-being in adults with autism spectrum disorders. *Autism*, 18(3), 223–232. <https://doi.org/10.1177/1362361312474121>.
- Merikangas, K. R., He, J. P., Burstein, M., Swanson, S. A., Avenevoli, S., Cui, L., . . . Swendsen, J. (2010). Lifetime prevalence of mental disorders in U.S. adolescents: Results from the National Comorbidity Survey Replication – Adolescent Supplement (NCS-A). *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(10), 980–989. <https://doi.org/10.1016/j.jaac.2010.05.017>.
- Pelton, M. K., & Cassidy, S. A. (2017). Are autistic traits associated with suicidality? A test of the interpersonal-psychological theory of suicide in a non-clinical young adult sample. *Autism Research*, 10(11), 1891–1904. <https://doi.org/10.1002/aur.1828>.
- Phillips, J. M., Uljarević, M., Schuck, R. K., Frazier, T. W., Schapp, S. S., Solomon, E. M., . . . Hardan, A. Y. (2019). Development of the Stanford Social Dimensions Scale (SSDS): Initial validation in autism spectrum disorder (ASD). *Molecular Autism*, 10(1), 1–16. <https://doi.org/10.1186/s13229-019-0298-9>.
- Pickard, H., Rijdsdijk, F., Happé, F., & Mandy, W. (2017). Are social and communication difficulties a risk factor for the development of social anxiety? *Journal of the American Academy of Child and Adolescent Psychiatry*, 56(4), 344.e3–351.e3. <https://doi.org/10.1016/j.jaac.2017.01.007>.
- Rai, D., Culpin, I., Heuvelman, H., Magnusson, C. M. K., Carpenter, P., Jones, H. J., . . . Pearson, R. M. (2018a). Association of autistic traits with depression from childhood to age 18 years. *JAMA Psychiatry*, 75(8), 835–843. <https://doi.org/10.1001/jamapsychiatry.2018.1323>.
- Rai, D., Heuvelman, H., Dalman, C., Culpin, I., Lundberg, M., Carpenter, P., & Magnusson, C. (2018b). Association between Autism Spectrum Disorders with or without intellectual disability and depression in young adulthood. *JAMA Network Open*, 1(4), e181465. <https://doi.org/10.1001/jamanetworkopen.2018.1465>.
- Ratto, A. B., Kenworthy, L., Yerys, B. E., Bascom, J., Wieckowski, A. T., White, S. W., Wallace, G. L., Pugliese, C., Schultz, R. T., Ollendick, T. H., Scarpa, A., Seese, S., Register-Brown, K., Martin, A., & Anthony, L. G. (2018). What about the girls? Sex-based differences in autistic traits and adaptive skills. *Journal of Autism and Developmental Disorders*, 48(5), 1698–1711. <https://doi.org/10.1007/s10803-017-3413-9>.
- Richa, S., Fahed, M., Khoury, E., & Mishara, B. (2014). Suicide in autism spectrum disorders. *Archives of Suicide Research*, 18(4), 327–339. <https://doi.org/10.1080/13811118.2013.824834>.
- Rieffe, C., De Bruine, M., De Rooij, M., & Stockmann, L. (2014). Approach and avoidant emotion regulation prevent depressive symptoms in children with an autism spectrum disorder. *International Journal of Developmental Neuroscience*, 39, 37–43. <https://doi.org/10.1016/j.ijdevneu.2014.06.003>.
- Rodgers, J., Glod, M., Connolly, B., & McConachie, H. (2012a). The relationship between anxiety and repetitive behaviours in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 42(11), 2404–2409. <https://doi.org/10.1007/s10803-012-1531-y>.
- Rodgers, J., Riby, D. M., Janes, E., Connolly, B., & McConachie, H. (2012b). Anxiety and repetitive behaviours in autism spectrum disorders and Williams syndrome: A cross-syndrome comparison. *Journal of Autism and Developmental Disorders*, 42(2), 175–180. <https://doi.org/10.1007/s10803-011-1225-x>.
- Rodgers, J., Wigham, S., McConachie, H., Freeston, M., Honey, E., & Parr, J. R. (2016). Development of the anxiety scale for children with autism spectrum disorder (ASC-ASD). *Autism Research*, 9(11), 1205–1215. <https://doi.org/10.1002/aur.1603>.
- Rodgers, J., Herrema, R., Honey, R., & Freeston, M. (2018). Towards a treatment for intolerance of uncertainty for autistic adults: A single case experimental design study. *Journal of Autism and Developmental Disorders*, 48(8), 2832–2845. <https://doi.org/10.1007/s10803-018-3550-9>.
- Roy, M., Prox-Vagedes, V., Ohlmeier, M. D., & Dillo, W. (2015). Beyond childhood: Psychiatric comorbidities and social background of adults with Asperger syndrome. *Psychiatria Danubina*, 27(1), 50–59.
- Rutter, M. (1970). Autistic children: Infancy to adulthood. *Seminars in Psychiatry*, 2(4), 435–450.
- Samson, A. C., Hardan, A. Y., Lee, I. A., Phillips, J. M., & Gross, J. J. (2015). Maladaptive behaviour in autism spectrum disorder: The role of emotion experience and emotion regulation. *Journal of Autism and Developmental Disorders*, 45(11), 3424–3432. <https://doi.org/10.1007/s10803-015-2388-7>.
- Segers, M., & Rawana, J. (2014). What do we know about suicidality in autism spectrum disorders? A systematic review. *Autism Research*, 7(4), 507–521. <https://doi.org/10.1002/aur.1375>.
- Selles, R. R., & Storch, E. A. (2013). Translation of anxiety treatment to youth with autism spectrum disorders. *Journal of Child and Family Studies*, 22(3), 405–413. <https://doi.org/10.1007/s10826-012-9593-1>.
- Silverman, W. K., & Albano, A. M. (1996). *The anxiety disorders interview schedule for children and parents – DSM-IV version*. New York: Graywind.
- South, M., Beck, J. S., Lundwall, R., Cutrer, E. C., Christensen, M., Gabrielsen, T. P., Cox, J. C., & Lundwall, R. A. (in press). Persistent depression and suicidality in women with autistic traits. *Journal of*

- Autism and Developmental Disorders: Special Issue on Suicidality and Self Harm in Autism Spectrum Disorder.*
- Spence, S. H. (1998). A measure of anxiety symptoms among children. *Behaviour Research and Therapy*, 36(5), 545–566.
- Spencer, D., Marshall, J., Post, B., Kulakodlu, M., Newschaffer, C., Dennen, T., ... Jain, A. (2013). Psychotropic medication use and polypharmacy in children with autism spectrum disorders. *Pediatrics*, 132(5), 833–840. <https://doi.org/10.1542/peds.2012-3774>.
- Strang, J. F., Kenworthy, L., Daniolos, P., Case, L., Wills, M. C., Martin, A., & Wallace, G. L. (2012). Depression and anxiety symptoms in children and adolescents with autism spectrum disorders without intellectual disability. *Research in Autism Spectrum Disorders*, 6(1), 406–412. <https://doi.org/10.1016/j.rasd.2011.06.015>.
- Suttin, A. R., Terracciano, A., Milaneschi, Y., An, Y., Ferrucci, L., & Zonderman, A. B. (2013). The trajectory of depressive symptoms across the adult life span. *JAMA Psychiatry*, 70(8), 803–811. <https://doi.org/10.1001/jamapsychiatry.2013.193>.
- Thomson, K., Riosa, P. B., & Weiss, J. A. (2015). Brief report of preliminary outcomes of an emotion regulation intervention for children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(11), 3487–3495. <https://doi.org/10.1007/s10803-015-2446-1>.
- Troy, A. S., Shallcross, A. J., & Mauss, I. B. (2013). A person-by-situation approach to emotion regulation: Cognitive reappraisal can either help or hurt, depending on the context. *Psychological Science*, 24(12), 2505–2514. <https://doi.org/10.1177/0956797613496434>.
- Uljarević, M., Lane, A., Kelly, A., & Leekam, S. (2016a). Sensory subtypes and anxiety in older children and adolescents with autism spectrum disorder. *Autism Research*, 9(10), 1073–1078. <https://doi.org/10.1002/aur.1602>.
- Uljarević, M., Nuske, H., & Vivanti, G. (2016b). Anxiety in autism spectrum disorder. In L. Mazzone & B. Vitiello (Eds.), *Psychiatric symptoms and comorbidities in autism spectrum disorder* (pp. 21–38). Cham: Springer International Publishing. https://doi.org/10.1007/978-3-319-29695-1_2.
- Uljarević, M., Richdale, A. L., Evans, D. W., Cai, R. Y., & Leekam, S. R. (2017a). Interrelationship between insistence on sameness, effortful control and anxiety in adolescents and young adults with autism spectrum disorder (ASD). *Molecular Autism*, 8, 36. <https://doi.org/10.1186/s13229-017-0158-4>.
- Uljarević, M., Richdale, A. L., McConachie, H., Hedley, D., Cai, R. Y., Merrick, H., ... Le Couteur, A. (2017b). The hospital anxiety and depression scale: Factor structure and psychometric properties in older adolescents and young adults with autism spectrum disorder. *Autism Research*, 1(2), 258–269. <https://doi.org/10.1002/aur.1872>.
- Uljarević, M., Labuschagne, I., Bobin, R., Atkinson, A., & Hocking, D. (2018). Brief report: The impact of sensory hypersensitivity and intolerance of uncertainty on anxiety in Williams syndrome. *Journal of Autism and Developmental Disorders*, 48(11), 3958–3964. <https://doi.org/10.1007/s10803-018-3631-9>.
- Uljarević, M., Hedley, D., Rose-Foley, K., Magiati, I., Cai, R. Y., Dissanayake, C., ... Trollor, J. (2019). Anxiety and depression from adolescence to old age in autism spectrum disorder. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-04084-z>.
- Uljarević, M., Phillips, J. M., Schuck, R. K., Frazier, T. W., Schapp, S. S., Solomon, E. M., ... Hardan, A. Y. (2020). Exploring social subtypes in autism spectrum disorder: A preliminary study. *Autism Research*, early online.
- Upthegrove, R., Abu-Akel, A., Chisholm, K., Lin, A., Zahid, S., Pelton, M., ... Wood, S. J. (2018). Autism and psychosis: Clinical implications for depression and suicide. *Schizophrenia Research*, 195, 80–85. <https://doi.org/10.1016/j.schres.2017.08.028>.
- van Steensel, F. J. A., Bögels, S. M., & Perrin, S. (2011). Anxiety disorders in children and adolescents with autistic spectrum disorders: A meta-analysis. *Clinical Child and Family Psychology Review*, 14(3), 302–317. <https://doi.org/10.1007/s10567-011-0097-0>.
- van Steensel, F. J. A., Bögels, S. M., Magiati, I., & Perrin, S. (2014). Anxiety in individuals with ASD: Prevalence, phenomenology, etiology, assessment, and interventions. In V. B. Patel, V. R. Preedy, & C. R. Martin (Eds.), *Comprehensive guide to autism* (pp. 601–623). New York: Springer.
- Vasa, R. A., Carroll, L. M., Nozzolillo, A. A., Mahajan, R., Mazurek, M. O., Bennett, A. E., ... Bernal, M. P. (2014). A systematic review of treatments for anxiety in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(12), 3215–3229. <https://doi.org/10.1007/s10803-014-2184-9>.
- Weiss, J. A., Thomson, K., & Chan, L. (2014). A systematic literature review of emotion regulation measurement in individuals with autism spectrum disorder. *Autism Research*, 7(6), 629–648. <https://doi.org/10.1002/aur.1426>.
- Weston, L., Hodgekins, J., & Langdon, P. E. (2016). Effectiveness of cognitive behavioural therapy with people who have autistic spectrum disorders: A systematic review and meta-analysis. *Clinical Psychology Review*, 49, 41–54. <https://doi.org/10.1016/j.cpr.2016.08.001>.
- White, S. W., & Roberson-Nay, R. (2009). Anxiety, social deficits, and loneliness in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(7), 1006–1013. <https://doi.org/10.1007/s10803-009-0713-8>.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229. <https://doi.org/10.1016/j.cpr.2009.01.003>.

- White, E. I., Wallace, G. L., Bascom, J., Armour, A. C., Register-Brown, K., Popal, H. S., Ratto, A. B., Martin, A., & Kenworthy, L. (2017). Sex differences in parent-reported executive functioning and adaptive behavior in children and young adults with autism spectrum disorder. *Autism Research*, 10(10), 1653–1662. <https://doi.org/10.1002/aur.1811>.
- Wigham, S., & McConachie, H. (2014). Systematic review of the properties of tools used to measure outcomes in anxiety intervention studies for children with autism spectrum disorders. *PLoS One*, 9(1), e85268. <https://doi.org/10.1371/journal.pone.0085268>.
- Wigham, S., Rodgers, J., South, M., McConachie, H., & Freeston, M. (2015). The interplay between sensory processing abnormalities, intolerance of uncertainty, anxiety and restricted and repetitive behaviours in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(4), 943–52.
- Wood, J. J., & Gadow, K. D. (2010). Exploring the nature and function of anxiety in youth with autism spectrum disorders. *Clinical Psychology: Science and Practice*, 17(4). <https://doi.org/10.1111/j.1468-2850.2010.01220.x>.
- Zigmond, A. S., & Snaith, R. P. (1983). The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavica*, 67, 361–370.

Anxiety Disorders

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Synonyms

Affective disorders (includes mood and anxiety disorders); Neurotic disorders (DSM-II terminology)

Short Description or Definition

Fear is a normal brain state, a physiologic response in response to a threat, or a dangerous or unexpected stimuli that serves as a warning system to maintain the individual and/or group safety. It is mediated by the activation of orbitofrontal cortex,

anterior cingulate gyrus, hippocampus and amygdala limbic circuits. When the fear occurs in the absence of a threat is called anxiety. Both fear and anxiety are normal reactions to danger. The physiologic normal response of fear and anxiety triggers a “fight, freeze, or flight response” that has been preserved throughout evolution. If anxiety is triggered by a non-dangerous stimuli, or it is too intense or persistent, or it creates impairment, it can be considered as an anxiety disorder. Symptoms are of a cognitive (e.g., worry thoughts), physiological (e.g., tachycardia), or behavioral (e.g., avoidance) nature. Anxiety disorders and mood disorder form the category of affective disorders (Revised in Soutullo and Figueroa 2010).

Categorization

There are several types of anxiety disorders, depending on the stimuli that trigger the anxiety:

- Separation anxiety disorder (SAD)
- Panic disorder
- Agoraphobia
- Generalized anxiety disorder (GAD)
- Simple Phobia that has five subtypes:
 - Animal
 - Environmental (heights, storms, etc.)
 - Pain (injections, blood, injuries, surgeries)
 - Situational (flights, elevators, closed environments, tunnels)
 - “Other types” (choking, vomiting, having an illness)
- Social phobia
- Obsessive-compulsive disorder (OCD)
- Posttraumatic stress disorder (PTSD)

Epidemiology

Anxiety disorders as a group are the most frequent psychiatric disorders in children and adolescents. The epidemiology varies across development. Usually simple phobias and separation anxiety appear first, and then social phobia, GAD, and panic disorder. Some authors suggest that the different anxiety disorders across childhood and

adolescence represent only a developmental variation of the disorder. Available data suggests that 2.8–27% of children and adolescents may have some form of anxiety disorder (Krain et al. 2007). Anxiety disorders are usually more frequent in females, and interestingly, female preponderance emerges before puberty, except in GAD, that only becomes more frequent in females after adolescence (Krain et al. 2007).

Prevalence of GAD is 6.5% in preschoolers, 3.8% in children, and 6.6% in adolescents; of social phobia 3.4% in preschoolers, 1.3% in children, and 1.1% in adolescents; of separation anxiety disorder is 2.4% in preschoolers, 4.1% in children, and 1.4% in adolescents; of simple phobia 1.9% in preschoolers, 5.8% in children, and 4.1% in adolescents; and of panic disorder 0.8% in children, and 2.7% in adolescents. The lifetime prevalence rate of OCD is between 1% and 4% (Keeley and Storch 2009). The mean age of onset is 4.1 years for simple phobia, 4.3 for separation anxiety, 5.3 for agoraphobia, 6 for social phobia, 6.3 for GAD, 6.5 for PTSD, and 8.5 for panic disorder.

Natural History, Prognostic Factors, Outcomes

There is a statistically robust, but modest in effect size (Odds Ratio: 2.0:4.0), association between pediatric anxiety disorders and a range of adult psychiatric disorders, such as mood and anxiety disorders. The most robust association appears to be between GAD and major depression, and anxiety disorder, especially panic disorder (Krain et al. 2007).

Clinical Expression and Pathophysiology

The key characteristics of the different anxiety disorders are (Tables 1 and 2):

- 1. Separation anxiety disorder: Excessive worries concerning separation from loved one, frequently associated with physical symptoms, school avoidance and worries about the loved ones, or about getting lost.

- 2. Social phobia: Fear on social situations that are avoided or endured.
- 3. GAD: Excessive uncontrollable worries about multiple issues during most of the time.
- 4. Specific phobia: Extreme fear and avoidance of specific situations or objects.
- 5. Panic disorder: Unexpected panic attacks, brief in time, with associated physical and psychological symptoms, and fear of having another attack in the future.
- 6. PTSD: Anxiety symptoms after a traumatic event, with associated autonomic hyperarousal, avoidance of the situation, and intrusive memories.
- 7. OCD: Obsessions (intrusive ego-dystonic thoughts) and associated compulsions (behaviors) aimed to reduce anxiety.

Anxiety-related disorders are among the most frequent presenting problems in the clinical setting in children with ASD (Tables 1 and 2).

The etiology and pathophysiology of anxiety is still under study, but we know that there are four factors involved in the development of an anxiety disorder: (1) genetic and environmental influences, (2) the neural circuits underlying emotion process, (3) core psychological processes, and (4) broad behavioral tendencies, including temperament. There are important genetic components in various forms of anxiety. Genetic and environmental influences are likely to shape more basic psychological processes which in

Anxiety Disorders, Table 1 Content of anxious thoughts for specific anxiety disorders

Anxiety disorder: worries, anticipated harm
SAD: Being separated from caretaker, harm to self or caretaker
PD: Being unable to escape the current situation, dying, losing control, going crazy
Social phobia: Negative social judgment embarrassment, negative evaluation, or rejection
PTSD: Posttraumatic event, reexperiencing traumatic event
OCD: Contamination, contracting a disease, doubt, catastrophic outcome
GAD: Routine life issues such as academic performance or social interactions, wide range of possible negative outcomes (e.g., failure, rejection)

Anxiety Disorders, Table 2 Somatic symptoms of anxiety included in the DSM-IV-TR (Keeley and Storch 2009)

System and symptoms
Cardiac
Tachycardia
Palpitations/Heart pounding
Chest pain
Shortness of breath
Gastrointestinal (GI)
Dry mouth
Difficulty swallowing
Nausea/vomiting, diarrhea
GI discomfort
Urogenital
Frequent urination, tenesmus
Respiratory
Shortness of breath
Smothering sensation
Choking sensation
Neurologica
Numbness/tingling
Tremor/shaking
Syncopal episodes/fainting
Sleep
Insomnia
Reluctance/refusal to sleep alone
Nightmares
Sleeptalking/sleepwalking
Excessive tiredness
Dermatological/temperature regulation
Sweating
Hot flashes
Chills
Cold, clammy hands
ENT
Dizziness
Lightheadedness
Feeling unsteady
Others
Increased startle response
Muscle tension

turn influence risk for anxiety. Despite the evidence for genetic contribution, anxiety disorders involve a large environmental component. Parents with anxiety may have distinctive child rearing or parenting practices, and may encourage or train their children to maladaptive patterns of responding to ambiguous situations (Keeley and Storch 2009).

Fear is regulated by connections between pre-frontal cortex (PFC) and the amygdala. When these circuits are altered (by a genetic or by an environmental overactivation), the child perceives a neutral stimuli as dangerous. In PFC the two areas involved in anxiety and fear are the orbitofrontal cortex (OFC) that makes a representation of both negative and positive reinforcers, and the anterior cingulate cortex (ACC), that regulates the emotional response. In addition to these responses, an activation of the amygdala activates:

- The HPA axis, and the hypothalamus secretes CRF (corticotrophin releasing factor), induces the secretion of ACTH, that will induce the secretion of cortisol and adrenaline in the adrenal gland, and causes hyperglycemia and tachycardia, needed for the brain and muscles to respond to danger.
- The parabrachial nuclei that increases respiratory frequency and may cause a sensation of shortness of breath similar to an asthma attack.
- The locus coeruleus, that also releases adrenaline, that raises blood pressure, pulse, activates sweating, and induces tremor (Revised in Soutullo and Figueroa 2010).

Evaluation and Differential Diagnosis

Evidence-based methods of evaluation include diagnostic interview schedules, rating scales, observations, and self-monitoring forms.

Diagnostic Interviews

Diagnostic interviews are reliable and valid instruments to facilitate diagnostic decisions consistent with DSM-IV-TR criteria. These clinician-administered structured diagnostic interviews assess for anxiety disorders and for the presence of other psychiatric disorders. However, these interviews require trained clinicians, and can be time-consuming and expensive (lasting 60–120 min). The most common diagnostic

interviews used in the diagnosis of anxiety disorders include:

1. The *Anxiety Disorders Interview Schedule for DSM-IV: Child and Parent Versions*
2. *K-SADS-PL: Kiddie Schedule for Affective Disorders and Schizophrenia-Present and Lifetime Version*
3. *SCID: Structured Clinical Interview for DSM-IV*

Rating scales: Self-report or parent-report rating scales require minimal training, are easy to administer, can be completed and scored quickly, are useful screening devices, and are easily readministered to capture clinical change over time.

General Anxiety Rating Scales

1. The *SCARED* (Screen for Child Anxiety Related Emotional Disorders-Revised) has five subscales (Panic/Somatic, Separation Anxiety, Social Phobia, General Anxiety, and School Phobia) that help to identify specific anxiety symptoms.
2. The *MASC* (Multidimensional Anxiety Scale for Children) is another commonly used rating scale of general anxiety symptoms with four subscales (Physical Symptoms, Harm Avoidance, Social Anxiety, and Separation/Panic) and an Anxiety Disorders Index, which includes items found to differentiate children with and without an anxiety disorder.
3. The *Fear Survey Schedule for Children-Revised* is a commonly used measure to assess childhood fears, with five subscales: Fear of Failure/Criticism, of the Unknown, of Injury and Small Animals, of Danger/Death, and Fear of Medical Situations.

Syndrome-Specific Anxiety Measures

1. The Social Anxiety Scale for Children-Revised
2. The Children's Yale-Brown Obsessive-Compulsive Scale-Child Report and Parent Report
3. Trauma Symptom Checklist for Children to assess PTSD Symptoms

Observational and Self-monitoring Methods

Direct Observation

1. Social evaluative tasks: In which a child is observed performing in a social situation (e.g., public speaking)
2. Behavioral avoidance tasks: In which a child's response to being exposed to a fear or anxiety-provoking stimuli is observed
3. Parent-child interaction tasks: In which parent and child are observed in a problem-solving task

Self-monitoring Procedures

This is a method to identify and quantify symptoms and behaviors using self-rated via diary-like entries.

Treatment

Practice parameters for the treatment of children with anxiety disorders recommend a multimodal approach to treatment, and comprehensive care should include consideration of:

- Psychoeducation
- Cognitive-behavioral psychotherapy (CBT)
- School consultation
- Family therapy
- Psychodynamic psychotherapy
- Pharmacotherapy (AACAP 2007)

The psychological interventions that have the most empirical support for childhood anxiety to date are *behavioral* and *cognitive-behavioral* interventions and *pharmacotherapy* with selective serotonin reuptake inhibitors (SSRIs) in the short-term treatment of childhood anxiety (AACAP 2007).

Behavioral and Cognitive-Behavioral Psychotherapy (CBT)

Cognitive-behavioral therapy (CBT) has proven to be effective in treating children and adolescents with anxiety disorders. CBT includes:

1. A cognitive-restructuring component
2. Modeling

3. Relaxation skills training
4. Homework
5. Contingency management
6. Most importantly, exposure to feared situations

The exposure (imagined, virtual, or real) is an opportunity for the patient to practice newly learned coping skills in a safe and controlled environment. The cognitive part helps children to change the thinking patterns that support their fears, and the behavioral part helps them to change the way they react to anxiety-provoking situations.

Despite some methodological limitations, mainly the use of a waiting list as a control group, CBT has demonstrated efficacy in the treatment of children with social phobia, GAD, and SAD, in two 16-week randomized controlled trials (Kendall et al. 1997). CBT with a family-based component was also effective, and had added benefits, particularly for younger female children, and treatment gains were maintained at 6-year follow-up (Barrett et al. 2001).

Exposure-based behavioral therapy has been used to treat specific phobias and OCD exposing the child gradually to the object or situation that is feared, perhaps at first only through pictures or tapes, then later face-to-face. Often the therapist will accompany the person to a feared situation to provide support and guidance. CBT is undertaken when the child decides he is ready for it and with his permission and cooperation. To be effective, the therapy must be directed at the person's specific anxieties and must be tailored to his or her needs. There are no side effects other than the discomfort of temporarily increased anxiety. CBT or behavioral therapy often lasts about 12 weeks. It may be conducted individually or with a group of people who have similar problems. Group therapy is particularly effective for social phobia. Often "homework" is assigned for participants to complete between sessions. There is some evidence that the benefits of CBT last longer than those of medication for people with panic disorder, and the same may be true for OCD, PTSD, and social phobia. If a disorder recurs at a

later date, the same therapy can be used to treat it successfully a second time.

Pharmacotherapy with SSRIs

Several recent randomized, placebo-controlled trials of SSRIs have shown evidence for the short-term efficacy of these medications in the treatment of children with anxiety disorders, including:

- GAD (Birmaher et al. 2003; RUPP 2001; Rynn et al. 2001).
- Social phobia (Birmaher et al. 2003; RUPP 2001; Wagner et al. 2004).
- SAD (Birmaher et al. 2003; RUPP 2001)
- OCD (POTS 2004)

No randomized, placebo-controlled trials of SSRIs exist for pediatric Panic Disorder (PD) or PTSD. Uncontrolled trials of SSRIs for pediatric PD suggest that SSRI treatment results in clinically significant reductions in symptoms (Keeley and Storch 2009).

Pharmacotherapy of Anxiety in Children with Autistic Spectrum Disorders (ASD)

There is some very preliminary evidence for the efficacy of sertraline, fluvoxamine, fluoxetine, buspirone, and dextromethorphan. None of these reports included a control group or placebo arm, and the largest sample size was 22 (White et al. 2009).

SSRIs

Two children ages 6 and 13 with DSM-IV ASD and co-occurring anxiety symptoms, treated with sertraline (25–50 mg/day) improved in symptoms of anxiety (Ozbayrak 1997).

An 11-year-old girl with ASD and separation anxiety disorder improved after 8-week treatment with sertraline (150 mg/day) (Bhardwaj et al. 2005).

A 7-year-old girl with PDD-NOS and intellectual disability treated with fluvoxamine had a 15.5 point decrease in the parent-reported CARS (Childhood Autism Rating Scale), and also fewer aggressive behaviors, less nervousness, but no reduction of repetitive behaviors or anxiety.

The child's parents and teachers received concurrent training and behavior interventions, which may have contributed to behavior changes (Kauffmann et al. 2001).

A retrospective chart review of 15 outpatients with ASD treated with citalopram (5–40 mg/day for 14–621 days), found improvements in symptoms of anxiety in 10 of the 15 youth (Namerow et al. 2003).

Silveira et al. (2004) treated with fluoxetine (20 mg daily) a 6-year-old girl with ASD who was also diagnosed with selective mutism and social anxiety. After 8 weeks, her parents reported improvements in symptoms of anxiety and selective mutism.

A retrospective chart review of the effectiveness of citalopram reported that 10 of 17 children with ASD who were treated with the SSRI (5–40 mg/day) showed improvement in target symptoms (Couturier and Nicolson 2002).

Buspirone

Buitelaar et al. (1998) conducted an open trial of buspirone to treat anxiety and irritability in children with ASD. All 22 youth exhibited chronic problems with anxiety, irritability, and/or affective dysregulation. After 6–8 weeks of buspirone treatment (15–45 mg/day), 16 of the 21 patients who completed the trial showed a positive response: nine had marked improvement and seven had moderate improvement on the Clinical Global Impressions-Improvement scale (CGI-I).

Dextromethorphan

Another case study of a 10-year-old boy diagnosed with autistic disorder and GAD reported improvements in target behaviors, leaving the classroom and aggressive tantrums, following treatment with dextromethorphan 30 mg/day (Woodard et al. 2005). Dextromethorphan may have relieved discomfort associated with other illnesses, may have had secondary sedative effects, or may have helped via its glutamate receptor antagonism.

See Also

- ▶ Generalized Anxiety Disorder
- ▶ Obsessive-Compulsive Disorder (OCD)
- ▶ Phobia
- ▶ Posttraumatic Stress Disorder
- ▶ Separation Anxiety Disorder
- ▶ Social Behaviors and Social Impairment

References and Reading

- American Academy of Child and Adolescent Psychiatry. (2007). Practice parameter for the assessment and treatment of children and adolescents with anxiety disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46, 267–283.
- Barrett, P. A., Duffy, A. L., Dadds, M. R., & Rapee, R. M. (2001). Cognitive-behavioral treatment of anxiety disorders in children: Long term (6-year follow-up). *Journal of Consulting and Clinical Psychology*, 69, 135–141.
- Bhardwaj, A., Agarwal, V., & Sitholey, P. (2005). Asperger's disorder with co-morbid separation anxiety disorder: A case report. *Journal of Autism Developmental Disorders*, 35, 135–136.
- Birmaher, B., Axelson, D. A., Monk, K., Kalas, C., Clark, D. B., Ehmann, M., et al. (2003). Fluoxetine for the treatment of childhood anxiety disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 42, 415–423.
- Buitelaar, J. K., van der Gaag, J., & van der Hoeven, J. (1998). Buspirone in the management of anxiety and irritability in children with pervasive developmental disorders: Results of an open-label study. *Journal of Clinical Psychiatry*, 59, 56–59.
- Couturier, J. L., & Nicolson, R. (2002). A retrospective assessment of citalopram in children and adolescents with pervasive developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 12, 243–248.
- Kauffmann, C., Vance, H., Pumariega, A. J., & Miller, B. (2001). Fluvoxamine treatment of a child with severe PDD: A single case study. *Psychiatry: Interpersonal and Biological Processes*, 64, 268–277.
- Keeley, M. L., & Storch, E. A. (2009). Anxiety in youth. *Journal of Pediatric Nursing*, 24(1), 26–40.
- Kendall, P. C., Flannery-Schroeder, E., Panicchelli-Mindel, S. M., Southam-Gerow, M., Henn, A., & Warman, M. (1997). Therapy for youths with anxiety disorders: A second randomized clinical trial. *Journal of Consulting and Clinical Psychology*, 65, 366–380.
- Krain, A. L., Ghaffari, M. Y., Freeman, J., Garcia, A., Leonard, H., & Pine, D. (2007). Anxiety disorder. In

- A. Martin & F. R. Volkmar (Eds.), *Lewis's child and adolescent psychiatry: A comprehensive textbook*. Philadelphia: Williams & Wilkins/Wolters Kluwer Business.
- Namerow, L. B., Thomas, P., Bostic, J. Q., Prince, J., & Monuteaux, M. C. (2003). Use of citalopram in pervasive developmental disorders. *Journal of Developmental and Behavioral Pediatrics*, 24, 104–108.
- Ozbayrak, K. R. (1997). Sertraline in PDD. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 7–8.
- Pediatric OCD Treatment Study (POTS) Team. (2004). Cognitive-behavior therapy, sertraline, and their combination for children and adolescents with obsessive-compulsive disorder. *JAMA: The Journal of the American Medical Association*, 292, 1969–1976.
- Research Units on Pediatric Psychopharmacology Anxiety Study Group (RUPP). (2001). Fluvoxamine for the treatment of anxiety disorders in children and adolescents. *New England Journal of Medicine*, 344, 1279–1285.
- Rynn, M. A., Siqueland, L., & Rickels, K. (2001). Placebo controlled trial of sertraline in the treatment of children with generalized anxiety disorder. *American Journal of Psychiatry*, 158, 2008–2014.
- Silveira, R., Jainer, A. K., & Bates, G. (2004). Fluoxetine treatment of selective mutism in pervasive developmental disorder. *International Journal of Psychiatry in Clinical Practice*, 8, 179–180.
- Soutullo, C., & Figueroa, A. (2010). *Convivir con niños y adolescentes con ansiedad (Living with children and adolescents with anxiety)*. Madrid: Editorial Médica Panamericana.
- Wagner, K. D., Berard, R., Stein, M. B., Wetherhold, E., Carpenter, D. J., & Perera, P. (2004). A multicenter, randomized, double-blind, placebo-controlled trial of paroxetine in children and adolescents with social anxiety disorder. *Archives of General Psychiatry*, 61, 1153–1162.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229.
- Wood, J. J., Drahota, A., Sze, K., Har, K., Chiu, A., & Langer, D. A. (2009). Cognitive behavioral therapy for anxiety in children with autism spectrum disorders: A randomized, controlled trial. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 50(3), 224–234.
- Woodard, C., Groden, J., Goodwin, M., Shanower, C., & Bianco, J. (2005). The treatment of the behavioral sequelae of autism with dextromethorphan: A case report. *Journal of Autism and Developmental Disorders*, 35, 515–518.

Anxiety Disorders Interview Schedule (ADIS)

► [Autism Spectrum Addendum to the Anxiety Disorders Interview Schedule-Parent Interview](#)

Anxiolytic Drugs

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Definition

Anxiolytic medications are used to treat anxiety symptoms. The most common categories involve SSRIs, serotonin agonists, and benzodiazepines. Other medications of interest but with minimal preliminary data include anticonvulsants, glutamate antagonists, and alpha-2 adrenergic antagonists.

Historical Background

There is accumulating evidence to suggest that children and adolescents with ASD have increased comorbidity with anxiety disorders, although there exists controversy on how the diagnosis of anxiety is made in ASD and what specific anxiety disorders are prevalent in this population. In fact, there has been some discussion that certain types of anxiety symptoms may be on a continuum or difficult to differentiate from core symptom domains (e.g., social anxiety and social deficits associated with ASD).

Current Knowledge

There are limited randomized, placebo-controlled trials of anxiolytic drugs for the purposes of reducing anxiety in ASD. Preliminary efficacy for SSRIs/SNRIs, buspirone, dextromethorphan, risperidone, and alpha-adrenergic antagonists exists in this area. Only two of these studies included a control group, and the largest sample size was 31.

SSRIs: There are three children with ASD and comorbid anxiety that have responded to sertraline based on clinician ratings on case reports (Bhardwaj et al. 2005; Ozbayrak 1997). Improvements in “nervousness” with fluvoxamine were reported in a single case by Kauffmann et al. 2001. Silveira et al. (2004) reported on a single case of a 6-year-old girl with ASD, selective mutism, and social anxiety who responded to fluoxetine. Two retrospective case series of citalopram in children with ASD and anxiety symptoms reported improvements in anxiety in response to citalopram (Couturier and Nicolson 2002; Namerow et al. 2003). Of note, there is a debate in the literature about whether repetitive behaviors seen in autism are related to anxiety disorders. Two large, randomized trial studies to date have shown that SSRIs (citalopram and fluoxetine) are not effective in reducing repetitive behavior in youth with ASD.

Bupirone: There is an open-label trial of bupirone, a serotonin agonist, in 22 children with ASD with comorbid anxiety, irritability, and affective dysregulation (Buitelaar et al. 1998). Sixteen of the 22 children were rated as responders at the end of the 8-week study.

Alpha-adrenergic agonists: A small double-blind, placebo-controlled, crossover study of transdermal clonidine in seven children and two adults with ASD and “hyperarousal” reported improvements on the CGI scale (Fankhauser et al. 1992). An open-label study (Ming et al. 2008) also showed improvements in sleep latency and night awakenings.

Glutamatergic agents: A case report suggested improvements in anxiety with dextromethorphan, a weak NMDA inhibitor (Woodard et al. 2005).

SNRIs: An open-label study of mirtazapine in 26 children and young adults with ASD suggested clinically meaningful improvements in 9/26 participants based on improvements in a variety of symptoms including anxiety.

Atypical antipsychotics: In a double-blind, placebo-controlled trial of risperidone in adults with ASD, significant improvements were noted in anxiety in the risperidone group over a 12-week period versus the placebo group (McDougle et al. 1998).

There has been almost no data that examines the role of *benzodiazepines in ASD*. Oswald and Sonenklar (2007) reported that in 2002, less than 5% of ASD patients were prescribed a benzodiazepine, suggesting that this class of medications is not widely used in ASD, despite their known effectiveness for anxiety disorders. Other than being habit forming, there are also reports of paradoxical reactions with the use of benzodiazepines in this population. For example, Marrosu et al. (1987) published a case series of anxiogenic and aggressive responses to diazepam in seven children with ASD. Overall, this class of medication is less attractive for use in pediatrics and especially in children with ASD.

Future Directions

There is also a clear lack of randomized, controlled trials of anxiolytic medications for the treatment of anxiety in ASD. In addition, there is accumulating data to support the use of CBT-based programs for anxiety in this population. There seems an urgent need to identify effective medications, and even more importantly, there is need to examine how medication may facilitate the ability psychoeducational programs to teach new skills and ultimately change the trajectory of anxiety symptoms in this population.

See Also

- ▶ Anxiety
- ▶ Anxiolytics
- ▶ Benzodiazepines
- ▶ Clonidine
- ▶ Diazepam
- ▶ Fluoxetine
- ▶ Fluvoxamine
- ▶ Risperidone
- ▶ Selective Serotonin Reuptake Inhibitors (SSRIs)
- ▶ Sertraline

References and Reading

- Bhardwaj, A., Agarwal, V., & Sitholey, P. (2005). Asperger's disorder with co-morbid separation anxiety disorder: A case report. *Journal of Autism and Developmental Disorders*, 35(1), 135–136.
- Buitelaar, J. K., van der Gaag, R. J., & van der Hoeven, J. (1998). Buspirone in the management of anxiety and irritability in children with pervasive developmental disorders: Results of an open-label study. *The Journal of Clinical Psychiatry*, 59(2), 56–59.
- Couturier, J. L., & Nicolson, R. (2002). A retrospective assessment of citalopram in children and adolescents with pervasive developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 12(3), 243–248.
- Fankhauser, M. P., Karumanchi, V. C., German, M. L., Yates, A., & Karumanchi, S. D. (1992). A double-blind, placebo-controlled study of the efficacy of transdermal clonidine in autism. *The Journal of Clinical Psychiatry*, 53(3), 77–82.
- Gibbs, T. T. (2010). Pharmacological treatment of autism. In G. J. Blatt (Ed.), *The neurochemical basis of autism* (pp. 245–267). New York: Springer Science and Business Media.
- Kauffmann, C., Vance, H., Pumariega, A. J., & Miller, B. (2001). Fluvoxamine treatment of a child with severe PDD: A single case study. *Psychiatry*, 64(3), 268–277.
- Marrosu, F., Marrosu, G., Rachel, M. G., & Biggio, G. (1987). Paradoxical reactions elicited by diazepam in children with classic autism. *Functional Neurology*, 2(3), 355–361.
- McDougle, C. J., Holmes, J. P., Carlson, D. C., Pelton, G. H., Cohen, D. J., & Price, L. H. (1998). A double-blind, placebo-controlled study of risperidone in adults with autistic disorder and other pervasive developmental disorders. *Archives of General Psychiatry*, 55(7), 633–641.
- Ming, X., Gordon, E., Kang, N., & Wagner, G. C. (2008). Use of clonidine in children with autism spectrum disorders. *Brain & Development*, 30(7), 454–460.
- Namerow, L. B., Thomas, P., Bostic, J. Q., Prince, J., & Monuteaux, M. C. (2003). Use of citalopram in pervasive developmental disorders. *Journal of Developmental and Behavioral Pediatrics*, 24(2), 104–108.
- Oswald, D. P., & Sonenklar, N. A. (2007). Medication use among children with autism spectrum disorders. *Journal of Child and Adolescent Psychopharmacology*, 17(3), 348–355.
- Ozbayrak, K. R. (1997). Sertraline in PDD. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(1), 7–8.
- Posey, D. J., Guenin, K. D., Kohn, A. E., Swiezy, N. B., & McDougle, C. J. (2001). A naturalistic open-label study of mirtazapine in autistic and other pervasive developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 11(3), 267–277.
- Silveira, R., Jainer, A. K., & Bates, G. (2004). Fluoxetine treatment for selective mutism in pervasive developmental disorder. *International Journal of Psychiatry in Clinical Practice*, 8, 179–180.
- van Steensel, F. J., Bögels, S. M., & Perrin, S. (2011). Anxiety disorders in children and adolescents with autistic spectrum disorders: A meta-analysis. *Clinical Child and Family Psychology Review*, 14(3), 302–317.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229.
- Woodard, C., Groden, J., Goodwin, M., Shanower, C., & Bianco, J. (2005). The treatment of the behavioral sequelae of autism with dextromethorphan: A case report. *Journal of Autism and Developmental Disorders*, 35(4), 515–518.

Anxiolytics

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Synonyms

[Antianxiety medication](#)

Definition

Anxiolytic is a broad term meant to describe medications that are used to reduce anxiety. Most commonly, these include benzodiazepines and SSRIs.

Benzodiazepines are a class of medications containing several drugs used in the treatment

of insomnia, anxiety, and seizures. The benzodiazepines represent a major advance in psychopharmacology with their introduction in the 1950s. These medications have been commonly used, but are habit-forming. The benzodiazepines have not been well studied in children or adults with autism. The short-acting benzodiazepines (lorazepam and alprazolam) are sometimes used to decrease anxiety prior to medical or dental procedures in children with ASDs. The right dose given at the right time prior to the procedure can be helpful. However, adverse effects of the benzodiazepines may include disinhibition (increased impulsiveness) and poor coordination. The disinhibition can be extreme. Rather than exerting a calming effect, some children have paradoxical activation. It is usually advisable to try a test dose before the actual day of the procedure to estimate the dose and the child's response (Scahill et al. 2010). These adverse effects in the short run and the possibility of habit formation suggest that this class of medications does not have an important role to play in the treatment of adults or children with autism.

SSRIs are approved for the treatment of adults with generalized anxiety disorder, social anxiety disorder, and obsessive-compulsive disorder. But few trials with any medications focused on anxiety symptoms have been conducted in subjects with autism spectrum disorders. More recently, other medications have been added to a list of anxiolytics such as buspirone and mirtazapine, SSRIs, and specific anticonvulsants (e.g., gabapentin and pregabalin). Currently, the SSRIs are likely the most commonly used medications for the treatment of anxiety. Indeed, several of the SSRIs are approved for the treatment of adults with generalized anxiety disorder, social anxiety disorder, and obsessive-compulsive disorder. But few trials with any medications focused on anxiety symptoms have been conducted in subjects with autism spectrum disorders.

In the treatment of anxiety disorders, effective treatment usually combines cognitive behavior therapy with medication. The optimal

treatment plan involves discontinuation of the benzodiazepine after 2–3 months. Long-term use of benzodiazepines can present a significant difficulty in getting the patient off the medication.

See Also

- [Alprazolam](#)
- [Benzodiazepines](#)
- [Diazepam](#)
- [Gabapentin](#)
- [Oxazepam](#)
- [Selective Serotonin Reuptake Inhibitors \(SSRIs\)](#)

References and Reading

- Marrosu, F., Marrosu, G., Rachel, M. G., & Biggio, G. (1987). Paradoxical reactions elicited by diazepam in children with classic autism. *Functional Neurology*, 2(3), 355–361.
- Martin, A., Scahill, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.
- Scahill, L. (2009). Antipsychotic medications. In M. K. Dulcan (Ed.), *Dulcan's textbook of child and adolescent psychiatry* (pp. 775–778). Arlington: American Psychiatric Publishing.
- Scahill, L., Poncin, Y., & Westphal, A. (2010). Alpha-adrenergics, beta-blockers, benzodiazepines, buspirone and desmopressin. In M. K. Dulcan (Ed.), *Dulcan's textbook of child and adolescent psychiatry* (pp. 775–786). Arlington: American Psychiatric Association.

Anxious Personality Disorder

- [Avoidant Personality Disorder](#)

Anxiset E (India)

- [Escitalopram](#)

APA Division 33 Intellectual and Developmental Disabilities

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Definition

Mission Statement (2020)

The American Psychological Association's Division 33 on Intellectual and Developmental Disabilities/Autism Spectrum Disorder (IDD/ASD) is a professional organization that has the mission of advancing psychological research, professional education, and clinical services that address the needs and increase the quality of life of individuals with IDD/ASD across the lifespan.

Some of the goals that drive our work include:

- Promoting social justice and inclusivity through our clinical work, research, and education related to IDD/ASD
- Expanding and exchanging knowledge and information about IDD/ASD through research, education, and professional communication
- Enhancing professional development, education, and the quality of professional services that relate to individuals with IDD/ASD
- Building partnerships with individuals with IDD/ASD, their families, and professional organizations that represent them to ensure that multiple perspectives are reflected in research, education, service, and professional development
- Informing legislative and administrative bodies on the importance of psychological, behavioral, and social factors in IDD/ASD and the value of psychology in contributing to the solution

- Strengthening the practice of psychology in IDD/ASD as a distinct professional and scientific entity
- Pursuing the development of high standards in training, practice, and research for psychologists who work in IDD/ASD

Historical Background

Division History

Prior to the formation of Division 33 in 1973, psychologists in the mental retardation field associated with other divisions in APA including Clinical Psychology, Developmental Psychology, School Psychology, Rehabilitation Psychology, Experimental Analysis of Behavior, and also the oldest organization devoted to intellectual and developmental disabilities, the American Association on Mental Deficiency (AAMD), recently renamed the Association on Intellectual and Developmental Disabilities (AAIDD). Edgar A. Doll served 1932–1933 with Frederick Kuhlman as presiding officers of the Clinical Section of APA and as president of AAMD 1934–1935 as he was developing the Vineland Social Maturity Scale (Doll 1935). He also served 1945–1946 as president of the Division of Clinical and Abnormal Psychology (Division 12) after it was formed as a division of APA (Routh 1997).

A number of these psychologists wished that APA had a division devoted to mental retardation, and during the 1970 meeting of the AAMD Region X in Provincetown, Massachusetts, they formed a steering committee to create the division. The members were Allan G. Barclay, Alfred A. Baumeister, Leonard S. Blackman, Lloyd M. Dunn, Norman R. Ellis, Mortimer Garrison, Olivia J. Hooker, Harris Kahn, Henry Leland, Harold Michal-Smith, Murry Morgenstern, William Sloan, and Sue A. Warren. The purpose would be to promote psychology as the “scientist-practitioner” model in the field of mental retardation. The Division on Mental Retardation (Division 33) was officially founded in January 1973 by a vote of the APA Council of Representatives at the annual meeting in 1972.

Marie Skodak Crissey was the first president, and she was followed by Norman R. Ellis and then Henry Leland (Routh 1999).

The Gatlinburg Conference on Theory and Research in Mental Retardation was first held in 1968 as a forum for the presentation of experimental research as well as for informal interactions among participants. For many of these years, it was held annually in Gatlinburg, Tennessee, but recently it has alternated among eastern, western, and southern cities. After the founding of Division 33, the Gatlinburg Conference served as an important scientific forum of the division. The Division 33 executive council often holds its semiannual meeting at the conference. An important resource to both the division and the conference was Theodore Tjossem, the chief of the mental retardation and behavioral disabilities branch of the National Institute of Child Health and Human Development, which supported the institute's Mental Retardation Research Centers program (Routh 1999).

The field of developmental disabilities was characterized by rapid changes in technologies and advances in research during the 1970s and 1980s. In recognition of the breadth of conditions that were recognized to constitute intellectual and developmental disabilities, the division changed its name from the Division on Mental Retardation to the Division on Mental Retardation and Developmental Disabilities in 1988 and to Division on Intellectual and Developmental Disabilities in 2007 and finally, with the emphasis on ASD, to Division on Intellectual and Developmental Disabilities/Autism Spectrum Disorder in 2015. Membership has grown significantly across the six types of membership, which include fellow, member, associate, life, affiliate, and student. Membership increased from 545 members at the end of 2010 to 717 members on December 31, 2019.

The decade of the brain, 1990–1999, ushered in a wealth of new findings on the neurodevelopmental aspects of intellectual and developmental disabilities. During the early 2000s, members were discussing the need to emphasize neurodevelopmental disorders such as ASD in

the division programming. Division President Sara Sparrow of the Yale Child Study Center at Yale University was acutely aware of the lack of representation of ASD researchers and practitioners in APA and Division 33 in particular. At the annual meeting in 2004, a decision was made to emphasize autistic spectrum disorder as one major focus and to reach out to psychologists, graduate students, and other organizations interested in ASD. The outreach has been successful. The number of presentations pertaining to ASD during the division's allotted programming hours at the annual APA meetings has increased to near 50% as has the number of awards for research related to ASD.

Current Knowledge

Landmark Contributions: Autism Spectrum Disorder

There was little empirical evidence that a person with autism could learn or become a productive member of society until 1987 when a multiyear study based on what is now called applied behavior analysis (ABA) was published by O. Ivar Lovaas. The study demonstrated that early intensive behavioral intervention (<4 years old, 40 h/week, including all significant people in all significant environments) for 2 and up to 6 years can produce large gains in most children. Children were able to pass first grade in either a normal or "aphasia classes" (Lovaas 1987). Follow-up at mean age of 13 years showed that 42% were indistinguishable from average children in terms of IQ and adaptive behavior (McEachin et al. 1993). Lovaas helped create the field of applied behavior analysis (ABA). In 1994, he received the division's highest award, the Edgar A. Doll Award, for revolutionizing the treatment of autism.

Claims of "recovery" for some participants were initially greeted skeptically. A number of replications showing strong gains have occurred. However, none of these found as large a gain, though none provided the intensity and duration of the original study (Thompson 2007a). An

overview of five meta-analyses of early intensive behavioral intervention (EIBI) studies (Reichow 2011) concluded that EIBI can produce “large gains in IQ and/or adaptive behavior” for many children and that “the current evidence on effectiveness of EIBI meets the threshold and criteria for the highest level of evidence-based treatments.” EIBI strategies have been evolving from highly structured programs in one-to-one settings to more naturalistic strategies. Pivotal response training, developed by Robert Koegel and Laura Schreibman (former students of Lovaas), and Lynn Koegel, is a naturalistic extension of ABA that targets pivotal aspects of a child’s development including motivation, responsivity to multiple cues, self-management, and social initiations. Pivotal response treatment, when possible, includes teachers and family members to help provide interventions as often as possible in the natural environment (Schreibman and Koegel 2005).

Recently, there has been an increasing emphasis on gene-brain-behavior relationships to provide a more complete understanding of problem behavior (Schroeder et al. 2002). Travis Thompson, past president of Division 33 and recipient of the division’s Edgar A. Doll Award in 2002, proposed that one of the primary tools of ABA, functional analysis of problem behavior, be extended to include biological measures functionally related to the problem behavior (Thompson 2007b). He also addressed the question of why only approximately half of the children treated by EIBI respond well, by pointing to neurophysiological evidence suggesting that practice enhances synaptic growth, which enables communication both within and among brain networks. If individuals lack sufficient neuroplasticity in critical areas, then new synapses may not be formed. He suggested that children with ASD who were responsive to EIBI may have been able to develop synapses in critical brain areas during treatment, whereas those unresponsive were unable to develop synapses in these areas (Thompson 2005, 2007b).

Accurate diagnosis of individuals with ASD is necessary both for research purposes and for determining effective treatment(s). Adaptive behavior

represents what a person typically does rather than the person’s potential and is perhaps the best overall measure to gauge the baseline level of a person’s functioning and subsequently the person’s response to intervention(s). Division 33 members have been active in extending the work of Doll (1935, 1936) to develop the Vineland Adaptive Behavior Scales (Sparrow et al. 1984) and the Vineland Adaptive Behavior Scales-II (Sparrow et al. 2005, 2008), which has become the most widely used measure of adaptive behavior for persons on the autistic spectrum. The 2008 version includes supplemental norms for ASD (Carter et al. 1998). The Vineland Adaptive Behavior Scales, Third Edition was introduced in 2016 (Sparrow et al. 2016). The new version is currently being investigated to help determine concordance with the second edition (Farmer et al. 2020).

Division Publications

Division 33 has one regular publication, the periodic *Psychology in Intellectual and Developmental Disabilities/Autism Spectrum Disorder*. The first issue was in the winter of 1974. The publication appeared irregularly until 1981 when Robert A. Fox began his term as newsletter editor. Subsequently, the newsletter appeared at least two times per year. Contents included division business, articles based on invited addresses, and talks by Division 33 award winners.

Beginning in the early 1990s, officers of Division 33 hoped to generate greater member interest in participating in the affairs of the division by offering space in the newsletters to special interest groups. Regular contributors to subsequent newsletters included an interest group on aging in mental retardation and one related to behavior modification. For a period of time, the newsletter flourished with these additional contributions, and considerable reader interest was generated by the newsletter. John W. Jacobson and James A. Mulick collaborated on frequent columns for the behavior modification interest group. These columns gradually evolved into pointedly humorous critiques and expositions of important issues in developmental disabilities. Several columns were devoted to problems with various definitions and criteria for diagnosing intellectual disability.

Beginning in the late 1990s, quite a few columns were devoted to early intervention and especially early intensive behavioral intervention for young children with ASD. The issue of facilitated communication was also addressed several times in the newsletter, as well as by other actions taken by the division (details later). Finally, the behavior modification interest group column also considered problems associated with the use of aversive motivation in behavior modification. Newsletters from 2000 to the present are available on the division website <http://www.division33.org>.

Division 33 published its own *Manual of Diagnosis and Professional Practice in Mental Retardation* in 1996, edited by Jacobson and Mulick. The peer-reviewed volume was published by APA books and went on to become a best seller for APA. Revenue from the book augmented the division treasury and led to a long period of financial solvency for Division 33.

Major Activities

The division sponsors a series of programs at the annual meeting of the American Psychological Association each year in August. These symposia and other presentations have increasingly included research on ASD. The percentage of the programming has increased from 30% in 2010 to more than 40% in 2019. Moreover, both Student Research Awards in 2019 were focused on ASD.

Division 33 members have always been involved in advocacy on behalf of people with developmental disabilities. At the same time, they have been wary of fads and ephemeral fashions in advocacy that have arisen from time to time in the general community. Many members worked actively in the deinstitutionalization efforts of the 1970s and 1980s. In doing so, they emphasized improvements in treatment approaches including behavioral intervention and assessment. The treatment of severe behavior disorders, in part a result of the deplorable conditions inside institutions and of the absence of services in community settings, led to widespread application of behavioral treatments to normalize their behavior. As treatment procedure evolved, some in the wider field of developmental

disabilities began to grow concerned about the possible detrimental or inappropriate use of behavioral procedures. Pressure to ready former institutional residents for community life was sometimes associated with an emphasis on quick success and less than thoughtful use of powerful and sometimes aversive and restrictive procedures with little concern about alternative approaches. Then too, many in the broad community could imagine that the labor-intensive and sometimes complicated treatment strategies would lead to a loss of autonomy or even mind control over a vulnerable population. This led advocacy groups to criticize the use of “aversive procedures” and to attempt to use regulations and guidelines to control the treatment options that could be used. Division 33 acted to assert a set of guidelines for the limited, appropriate use of aversive and restrictive procedures that were consistent with the scientific literature on behavior change and the need to control severe aggression, self-injury, and destructive behavior that would otherwise deny people with developmental disabilities the ability to live in the community. These guidelines were published in the newsletter and included in the division’s *Manual of Diagnosis and Professional Practice in Mental Retardation* (Jacobson and Mulick 1996), although they were not adopted as official policy of the division or of the American Psychological Association.

The pressure for universal education and normalization of conditions in society for people with developmental disabilities was very intense throughout the last quarter of the twentieth century and remains so at this writing. Inevitably, the extravagant desire for universal inclusion sometimes clashes with the reality of disability in the context of education. Some students have been found to be unable to benefit from all but the most systematic and individualized behavioral educational services and not to be able to participate in traditional teaching approaches in any practical sense. Usually, this is the result of a lack of any viable communication ability on the part of the person with the disability. Into this vacuum of social and family disappointment, unsubstantiated claims were made for the existence of “hidden literacy” that nevertheless could

be induced to emerge with mere manual support of the disabled person's hand or arm over a keyboard or array of letters. As asserted by Syracuse Professor, Douglas Biklen (1990), the manual support procedure was known as "facilitated communication," and credulous teachers, classroom aides, and hopeful parents have been trained in this technique. Unfortunately, a large body of controlled research has established that facilitated communication was, whenever subjected to empirical evaluation, not the product of the person with a disability but rather the often "non-conscious" result of influence by the facilitator (Spitz 1997). Members of Division 33 contributed a critique of facilitated communication which was published in the *American Psychologist* (Jacobson et al. 1995) and lobbied in APA for the passage of resolution discouraging the use of facilitated communication by psychologists for any purpose and cautioning specifically against relying on it as a means of practical communication in any important context. The resolution was adopted as policy of APA by the Council of Representatives in 1994. The full text of the resolution was included in the *Manual of Diagnosis and Professional Practice in Mental Retardation*.

Division Awards

Two Student Research Excellence Awards (annual) are available for students, for proposals submitted for a presentation at the APA annual meeting. During each of the years 2007–2018, at least one of these awards went for a study on ASD. Both awards in 2019 were for studies aimed toward a better understanding of ASD. One study focused on sensory over-responsivity and anxiety, the other on transitioning to adulthood.

The Edgar A. Doll Award (annual) is named in honor of Edgar A. Doll, the research director of the Vineland Training School from 1925 to 1945 where he made profound contributions in the areas of brain injury, electroencephalography (EEG), and adaptive behavior. He is perhaps best known for developing the Vineland Social Maturity Scale (1935), the revised versions of which are generally considered to provide the most useful measure of the impact of intellectual and developmental disabilities (see above). The Doll

award is the division's highest recognition of outstanding scientific contributions to the field of intellectual and developmental disabilities and was first given in 1981 to Samuel A. Kirk.

The John W. Jacobson Award (biannual) acknowledges John W. Jacobson's dedication to critical thinking in the field (see contributions above). The Jacobson Award recognizes meritorious contributions to the field of intellectual and developmental disabilities in an area directly related to behavioral psychology, evidence-based practice, dual diagnosis, or public policy and was first given in 2007 to Richard Foxx.

The Sara S. Sparrow Early Career Research Award (see her contributions above) of Division 33, alternating biannually with the Jacobson Award, honors an early career individual who has made substantial contributions to the understanding of intellectual or developmental disabilities as reflected in his or her published and presented works. The award was first given in 2008 to Luc Lecavalier.

Future Directions

The division will continue to emphasize autistic spectrum disorder (ASD) as a major focus and to reach out to psychologists, graduate students, and other organizations interested in ASD. Programming at the annual meeting will include research aimed toward a better understanding of the genetic, neurophysiological, psychological, and social factors and their interactions that underlie ASD and other intellectual and developmental disabilities.

Ethical treatment has become more of an issue with the emergence of a movement by people with ASD against ABA, some labeling ABA as abuse (Michelle Dawson 2004). The appliedBehaviorAnalysisEdu.org, (n.d.) addresses this issue and states that "The decision-making process needs to be handled on a case-by-case basis with respect to the severity of each individual's condition." The emphasis on treating individuals in their natural environment with many or most caregivers involved in the treatment plan (Schreibman and Koegel 2005) should help minimize those concerns. There appears to be more of an effort to

address the complex issues regarding which stakeholders have ethical-legal authority to weigh in on decision-making.

The division has five special interest groups: behavior modification and technology, dual diagnosis, early intervention, aging and adult development, and making the transition into adulthood. Division members involved pursue research in these areas and contribute to the scientific and professional literature. They participate in forums at professional meetings to present and discuss the latest findings. In particular, members promote and organize symposia at professional meetings. They also advocate in their communities for treatment services for individuals with ASD and other intellectual and developmental disabilities. Members also provide direct treatment services as well as train others to provide these services.

The Division Executive Council has closely monitored the development of the regulations, required by 2010 health-care legislation, that pertain to psychological services for individuals with developmental disabilities, especially ASD. Division members continue to work with the central APA office to inform HHS and state agencies of best practices for the treatment of individual with ASD and to emphasize the need to provide adequate health insurance coverage for these expensive services.

See Also

- [American Psychological Association](#)
- [Early Intensive Behavioral Intervention \(EIBI\)](#)
- [Pivotal Response Training](#)
- [UCLA Young Autism Project](#)
- [Vineland Adaptive Behavior Scales \(VABS\)](#)

References and Reading

- AppliedBehaviorAnalysisEdu.org (n.d.) What is the neurodiversity movement and autism rights? <https://www.appliedbehavioranalysisedu.org>
- Biklen, D. (1990). Communication unbound: Autism and praxis. *Harvard Educational Review*, 60, 291–314.
- Carter, A., Volkmar, F., Sparrow, S., Wang, J., Lord, C., Dawson, G., et al. (1998). The Vineland adaptive behavior scales: Supplementary norms for individuals with autism. *Journal of Autism and Developmental Disorders*, 28, 287–302.
- Doll, E. A. (1935). The Vineland social maturity scale: Manual of directions. *The Training School Bulletin*, 32, 1–3.
- Doll, E. A. (1936). *The Vineland social maturity scale: Revised condensed manual of directions*. Vineland: The Training School.
- Farmer, C., Adedipe, D., Bal, V., Chlebowski, C., & Thurm, A. (2020). Concordance of the Vineland adaptive behavior scales, second and third editions. *Journal of Intellectual Disability Research: JIDR*, 64(1), 18–26. <https://doi.org/10.1111/jir.12691>.
- Jacobson, J. W., & Mulick, J. A. (Eds.). (1996). *Manual of diagnosis and professional practice in mental retardation*. Washington, DC: American Psychological Association.
- Jacobson, J. W., Mulick, J. A., & Schwartz, A. A. (1995). A history of facilitated communication: Science, pseudoscience, and antiscience. *American Psychologist*, 50, 750–765.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- McEachin, J. J., Smith, T., & Lovaas, O. I. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. *American Journal of Mental Retardation*, 97, 359–372.
- Michelle Dawson, M (2004) The misbehavior of behaviorists – Ethical challenges to the autism-ABA industry. https://www.sentex.ca/~nexus23/naa_aba.html
- Reichow, B. (2011). Overview of meta-analyses on early intensive behavioral intervention for young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(4), 512–520. <https://doi.org/10.1007/s10803-011-1218-9>.
- Reichow, B., & Wolery, M. (2009). Comprehensive synthesis of early intensive behavioral interventions for young children with autism based on the UCLA young autism project model. *Journal of Autism and Developmental Disorders*, 39, 23–41.
- Routh, D. K. (1997). A history of division 12 (clinical psychology): Fourscore years. In D. Dewsbury (Ed.), *Unification through division: Histories of the divisions of the American Psychological Association* (Vol. 2, pp. 55–82). Washington, DC: American Psychological Association.
- Routh, D. K. (1999). A history of division 33 (psychology in mental retardation and developmental disabilities). In D. Dewsbury (Ed.), *Unification through division: Histories of the divisions of the American Psychological Association* (Vol. 3, pp. 117–142). Washington, DC: American Psychological Association.
- Schreibman, L., & Koegel, R. L. (2005). Training for parents of children with autism: Pivotal responses, generalization, and individualization of interventions. In E. D. Hibbs & P. S. Jensen (Eds.), *Psychosocial treatments for child and adolescent disorders: Empirically based strategies for clinical practice* (pp. 605–631). Washington, DC: American Psychological Association.

- Schroeder, S. R., Oster-Granite, M. L., & Thompson, T. (Eds.). (2002). *Self-injurious behavior: Gene-brain-behavior relationships*. Washington, DC: American Psychological Association.
- Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1984). *Vineland adaptive behavior scales: Survey form manual*. Circle Pines: American Guidance Service.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales: Second edition (Vineland II), survey interview form/caregiver rating form*. Livonia: Pearson Assessments.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2008). *Vineland adaptive behavior scales: Second edition (Vineland II), the expanded interview form*. Livonia: Pearson Assessments.
- Sparrow, S., Cicchetti, D., & Saulnier, C. (2016). *Vineland adaptive behavior scales: Third edition*. Pearson: San Antonio.
- Spitz, H. (1997). *Nonconscious movements: From mystical messages to facilitated communication*. Manwah: Lawrence Erlbaum.
- Thompson, T. (2005). Paul E. Meehl & B. F. Skinner: Autitaxia, autitpy and autism. *Behavior and Philosophy*, 33, 101–131.
- Thompson, T. (2007a). *Making sense of autism*. Baltimore: Paul H. Brooks Publishing.
- Thompson, T. (2007b). Relations among functional systems in behavior analysis. *Journal of the Experimental Analysis of Behavior*, 87, 423–440.

Apgar Score

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Definition

The Apgar score is a numerical score developed by Virginia Apgar, MD, an American anesthesiologist, in 1952 to standardize the description of newborn infant medical stability in the delivery room. A scale from 0 (worst) to 2 (normal) is assigned to the following parameters: heart rate (no heart rate to normal >100 beats per minute), respiratory effort (no respiratory effort to cries and has regular breathing), muscle tone (flaccid to

active motion), reflex irritability (no response to grimace and cry), and skin color (dusky blue to pink). The scores are added up to quantify the infant's status at 1 and 5 min. Infants rarely receive perfect scores of 10 because they typically have bluish-colored fingertips even if they are otherwise pink (1 for color). Lower Apgar scores may reflect neonatal stress, use of maternal anesthetic, and immaturity or prematurity. Apgar scores were designed for use with term infants. Scores of 0–3 at 1 and 5 min indicate neonatal depression and suggest the need for medical attention to help the baby adjust to postnatal conditions. A lower Apgar score at 1 min with a normal range score at 5 min is not typically of concern. Apgar scores are not measures of neonatal asphyxia or necessarily predictive of later neurologic impairment.

References and Reading

- American Academy of Pediatrics, Committee on Fetus and Newborn and Committee on Obstetric Practice, American College of Obstetrics and Gynecology. (1996). Use and abuse of the Apgar score. *Pediatrics*, 98, 141–142.
- Health children, American Academy of Pediatrics. <http://www.healthychildren.org/English/ages-stages/prenatal/delivery-beyond/pages/Apgar-Scores.aspx?nfstatus=401&nftoken=00000000-0000-0000-0000-000000000000&nftstatusdescription=ERROR%3a+No+local+token>. Accessed 23 May 2012.
- <https://www.acog.org/Resources-And-Publications/Committee-Opinions/Committee-on-Obstetric-Practice/The-Apgar-Score>. Accessed 25 Aug 2017.
- National Library of Medicine and National Institutes of Health. <http://www.nlm.nih.gov/medlineplus/ency/article/003402.htm>. Accessed 23 May 2012.

Aphasia

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Short Description or Definition

Aphasia, from the Greek term “aphatos” meaning “without language,” is a disorder caused by

damage to the language areas of the brain. Depending on the type and severity of the damage, deficits may be noted in language comprehension and/or production and can include both the spoken and written modalities. Aphasia most commonly occurs secondary to stroke in which brain cells are deprived of oxygen, resulting in tissue death, but it can also be the result of degenerative disorders or traumatic brain injury. Aphasia can co-occur with other conditions including apraxia and dysarthria which are neurologically based motor disorders that can affect speech output.

Categorization

Historically, aphasia has been classified according to the region of the brain that is affected and the symptoms that are displayed. For example, damage to what is considered “Broca’s area,” the region anterior to the Rolandic fissure, often results in a nonfluent form of aphasia in which comprehension is intact, but articulation and speech output, including syntax, is impaired. A disorder in which syntax and language output are preserved, while comprehension is impaired, is often referred to as Wernicke’s aphasia, due to the frequent damage that is observed in Wernicke’s area within the temporal lobe of the brain. However, as research continues to indicate that there is not necessarily a one-to-one correspondence between region in which brain damage is displayed and the type of symptoms that are experienced, other classification systems have developed.

More recently, types of aphasia have been divided into two categories: fluent and nonfluent aphasias. Fluent aphasias include Wernicke’s aphasia (above) and are characterized by individuals speaking in long sentences that often contain unnecessary words and are devoid of meaning. Comprehension in Wernicke’s aphasia is often impaired as well.

Nonfluent aphasias include Broca’s aphasia (above). In addition, global aphasia is another nonfluent aphasia that is characterized by extensive brain damage and severe communication deficits in both receptive and expressive language.

Epidemiology

The incidence of aphasia is largely unknown, given that it occurs in many types of disorders including cerebrovascular, traumatic, and degenerative disorders. In general, it is estimated that about one million individuals in the USA demonstrate aphasia with approximately 80,000 individuals acquiring this disorder every year (ASHA 2008).

Natural History, Prognostic Factors, and Outcomes

Outcomes for patients with aphasia vary greatly depending on the type and location of brain damage and level of severity of the disorder. Recovery is often more favorable for younger individuals or individuals with less extensive brain damage. In addition, language comprehension skills are often recovered more completely than language production skills. Factors including age of onset, health, education level, and how soon treatment takes place after brain damage have been shown to be predictive of recovery in aphasia.

Clinical Expression and Pathophysiology

Aphasia typically manifests itself as a difficulty in language comprehension, production, or both depending on the type and severity of the condition. Aphasia most commonly occurs secondary to stroke, but it can also be the result of degenerative disorders or traumatic brain injury.

Evaluation and Differential Diagnosis

Aphasia is typically initially diagnosed by a neurologist or other physician who is responsible for the treatment of the patient’s physical and neurological symptoms utilizing case history and observation. Further evaluation by a licensed speech-language pathologist often follows. Evaluation

includes the use of language in both comprehension and production contexts, including reading and writing. Evaluations typically include taking a comprehensive case history, observation of the patient in daily contexts, and formal evaluations of language skills, including naming of objects. Standardized evaluation tools that often are used include the Boston Diagnostic Aphasia Examination (Goodglass et al. 2000), the Boston Naming Test (Kaplan et al. 1983), and the Western Aphasia Battery (Kertesz 2006). Once the individual's profile of language strengths and needs is determined, treatment is initiated.

Treatment

Treatment for aphasia is often multifaceted and is typically individualized based on the patient's profile of strengths and needs. Individuals with aphasia often enroll in formal speech-language therapy to address functional communication in a variety of settings in which they are expected to communicate. Therapy goals are focused on maximizing the individual's ability to communicate effectively with peers and family members, given residual strengths. In addition, computer-assisted treatments are beginning to show promise as supports for individuals with aphasia.

Although some individuals recover completely, individuals with aphasia often experience lifelong deficits. In these cases, family member and patient support groups are often a critical piece of the therapeutic process as the patient and family learn to manage their new situation. The National Institute on Deafness and other Communication Disorders (NIDCD 2011) recommends the use of the following caregiver support strategies:

- Simplify language by using short, uncomplicated sentences.
- Repeat the content words or write down key-words to clarify meaning as needed.
- Maintain a natural conversational manner appropriate for an adult.

- Minimize distractions, such as a loud radio or TV, whenever possible.
- Include the person with aphasia in conversations.
- Ask for and value the opinion of the person with aphasia, especially regarding family matters.
- Encourage any type of communication, whether it is speech, gesture, pointing, or drawing.
- Avoid correcting the person's speech.
- Allow the person plenty of time to talk.
- Help the person become involved outside the home. Seek out support groups such as stroke clubs.

See Also

- Broca's Aphasia
- Global Aphasia
- Wernicke's Aphasia

References and Reading

- American Speech-Language-Hearing Association (ASHA). (2008). Incidence and prevalence of speech, voice, and language disorders in adults in the United States. Retrieved May 1, 2011 from www.asha.org/research/reports/speech_voice_language.htm.
- Barresi, B., Goodglass, H., & Kaplan, E. (2001). *The assessment of aphasia and related disorders*. Hagerstown: Lippincott, Williams & Wilkins.
- Chapey, R. (2008). *Language intervention strategies in aphasia and related neurogenic communication disorders*. Philadelphia: Wolters Kluwer/Lippincott, Williams & Wilkins.
- Goodglass, H., Kaplan, E., & Barresi, B. (2000). *Boston diagnostic aphasia examination (BDAE-3)* (3rd ed.). Austin: Pro-Ed.
- Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *The Boston naming test*. Philadelphia: Lea and Febiger.
- Kent, R. D. (1994). *Reference manual for communicative sciences and disorders: Speech and language*. Austin: Pro-Ed.
- Kertesz, A. (2006). *Western aphasia battery-revised (WAB-R)*. Austin: Pro-Ed.
- Lapointe, L. L. (2004). *Aphasia and related neurogenic language disorders*. New York: Thieme Medical Publishers.
- National Institute on Deafness and Other Communication Disorders (NIDCD). (2011). *Aphasia*. Retrieved May 1, 2011 from <http://www.nidcd.nih.gov/health/voice/aphasia.htm>.

Aphonia

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Synonyms

[Loss of voice](#)

Short Description or Definition

Aphonia is the complete loss of voice, typically due to an acquired cause such as vocal cord paralysis or damage to the recurrent laryngeal nerve. In aphonia, phonation (i.e., the process by which sounds are produced through the vibration of the vocal folds) is completely impaired, in contrast to dysphonia in which sound production is limited but not completely absent. Individuals with aphonia are only able to whisper when attempting to speak.

Epidemiology

While specific epidemiologic estimates of the incidence of aphonia are rare, generally speaking, approximately 7.5 million people in the United States demonstrate difficulty with vocal use. Voice disorders are more prevalent in individuals working in occupations that are characterized by frequent or intense vocal use.

Natural History, Prognostic Factors, and Outcomes

Some of the known causes of aphonia include laryngeal or thyroid cancer, vocal fold paralysis, nodules or polyps on the vocal folds, vocal abuse, respiratory problems, injury to the laryngeal nerve, surgical removal of the larynx, vocal fold thickening, or, in rare cases, psychogenic causes. Risk for aphonia increases when an individual is exposed to surgery involving the larynx, when an

individual engages in vocally abusive behaviors such as smoking or vocal overuse, or when an individual experiences anxiety. Prognosis for recovery largely depends upon the cause, severity, and nature of the disorder.

Clinical Expression and Pathophysiology

Aphonia manifests itself as a complete loss of voice. Some individuals are able to whisper, while others demonstrate total vocal loss.

Evaluation and Differential Diagnosis

Aphonia is typically diagnosed by medical professionals who specialize in voice disorders such as physicians who specialize in Ear, Nose, & Throat conditions (ENTs), or medically trained speech-language pathologists. Diagnostic procedures typically involve thorough case histories as well as physical examination to determine possible causes. Diagnostic procedures include analyses of vocal fold function including laryngoscopy and videostroboscopy. In the case of the absence of a clear physical cause, psychological evaluations are often recommended.

Treatment

Treatment of aphonia depends upon the nature and severity of the disorder. Treatments can include surgery, counseling for anxiety and related issues that appear to be causing tension that limits vocal fold function, lifestyle changes such as vocal rest, change of occupation, increased hydration, relaxation techniques, and formal voice therapy. Often therapy with a licensed speech-language pathologist is recommended, especially in cases where specific counseling and treatment techniques appear to be beneficial to the patient in terms of recovery of vocal function.

See Also

► [Speech Impairment](#)

References and Reading

- Aronson, A. E., & Bless, D. M. (2009). *Clinical voice disorders* (4th ed.). New York: Thieme.
- ASHA. (2008). *Incidence and prevalence of speech, voice, and language disorders in adults in the United States: 2008 edition*. Retrieved from 1 May 2011 www.asha.org/research/reports/speech_voice_language.htm
- Boone, D. R., McFarlane, S. C., Von Berg, S. L., & Zraick, R. I. (2009). *The voice and voice therapy* (8th ed.). Boston: Allyn & Bacon.
- Johnson, A. F., & Jacobson, B. H. (2007). *Medical speech-language pathology: A practitioner's guide*. New York: Thieme.
- NIDCD. (2010). *Statistics on voice, speech language from NIDCD*. Retrieved from 1 May 2011 www.nidcd.nih.gov/health/statistics/vsl.asp
- Rammage, L., Morrison, M., & Nichol, H. (2001). *Management of the voice and its disorders* (2nd ed.). San Diego: Singular.
- Stemple, J., Glaze, L., & Gerdeman Klaben, B. (2000). *Clinical voice pathology - Theory and management* (3rd ed.). San Diego: Singular.
- Verdolini, K., Rosen, C. A., Branski, R. C., & Andrews, M. L. (2006). *Classification manual for voice disorders I*. Mahwah: Lawrence Erlbaum.

learning of adaptive, constructive behavior and by reducing excessive problem behavior. Within ABA, the behaviors to be changed and descriptions of interventions responsible for changes are explicitly defined and technologically exact, allowing replication of procedures by others. Scientific methods, including objective description, measurement, and experimentation, allow analysis and establishment of functional relationships between interventions and behavior change. It is these interventions that have been come to be known in the public vernacular as ABA.

Historical Background

In his experiments with non-human animals, B.F. Skinner discovered the principle of *operant conditioning*, where consequences following responses result in changes in future behaviors. These principals of behavior and learning provided the foundation of many experimental studies that were eventually extended to humans in the 1950s. With the publication of the *Journal of Applied Behavior Analysis*, these interventions, previously known as behavior modification, and studies formed the basis of what is now known as *applied behavior analysis*. This branch of behavior analysis had as its goal of the improvement socially important behavior in the real world.

In the 1960s, a groundbreaking study was conducted with a 3-year-old child with autism who was at risk for permanently losing vision because he would not wear his glasses (Wolf et al. 1964). He displayed self-destructive tantrums, had sleeping problems, eating problems, and severe deficits in functional communication. Attempts to treat these problems with sedatives, tranquilizers, and restraints were unsuccessful.

Behavioral intervention consisted of shaping of behavior by reinforcing successively longer periods of glasses wearing with small bits of preferred food and of removal of social attention for tantrums. Gains in appropriate speech resulted from presenting clear cues to verbalize and reinforcement of correct responses with small bites of food. What is now known as *discrete trial training* was pioneered in this original effort. Additional

Apo-Haloperidol

► Haloperidol

Applied Behavior Analysis (ABA)

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Definition

Applied behavior analysis (ABA) is a science that identifies variables that meaningfully and lawfully influence socially significant behavior in real-world settings. This is done by using principles of behavior to successfully teach and support the

hallmarks of this study were staff and parent training, early intervention, and systematic follow-up to ensure maintenance of treatment gains, as well as teaching of new, socially-appropriate behavior. In addition, the natural setting intervention for children with autism, data collection, and early single-subject methodology became foundational practices of applied behavior analysis.

Excitement from these early studies led to the development of laboratories that focused solely on the treatment of children with autism, with the most notable being the Behavioral Intervention Clinic at the University of California, Los Angeles (UCLA), directed by O.I. Lovaas. Rejecting the earlier notion that autism was a psychopathology caused by poor mothering, the behavioral model focused on treating behavioral deficits and excesses exhibited by the children. These early studies also revealed the deleterious effects of institutional environments and the positive effects of intensive, early, comprehensive treatments that included parent training in community settings.

The results of a 1987 study published by the UCLA laboratory, showing that 47% of 19 children achieved normal intellectual functioning, as well as successful inclusion in school, resulted in controversy regarding the methods employed in the study and the dramatic results that were gained (Lovaas 1987). Many replication studies were conducted following this historical intervention that became known as Early Intensive Behavioral Intervention (EIBI) (e.g., Sallows and Graupner 2005). While the results of the subsequent studies did not reveal the extent of improvement in the 1987 study, positive effects of EIBI were still evidenced, with children showing socially meaningful improvements as a result of behavioral intervention. Researchers and scholars in the scientific community responded to findings with questions regarding the effectiveness of this intervention with children of varying severity levels and with comorbid diagnoses. Many scholars cautioned against a “one-size-fits-all” philosophy when considering EIBI interventions.

A change in terminology occurred when the interventions designed from the science of *applied behavior analysis* began to be commonly

referred to as “ABA.” Common use of the term in the public vernacular referred to one-to-one discrete trial interventions in low-distraction environments, where individual skills were taught using a *massed-trial* approach using high rates of positive reinforcement.

However, scholars and researchers continued to use the science of applied behavioral analysis research, including single-subject designs and socially valid outcomes, to expand the intervention strategies. Interventions were developed to increase the amount of child control in the intervention by incorporating children’s choices and preferences and following the child’s lead in language intervention (Koegel et al. 1999). In addition, researchers discovered that many problem behaviors served a communicative function for valid needs, including the need for attention, the need for assistance, and the need to say, “No”, to unpleasant things. This evolved into functional communication training as a major focus of behavioral intervention, where learners were taught appropriate communication to replace severe problem behavior (Carr and Durand 1985).

In addition to an expansion of treatments, applied behavior analysis treatments expanded to include interventions across the age range, including toddlers, older children, adolescents, and adults, with emphasis on appropriate academic skills in the classroom, vocational skills, and peer socialization. Settings expanded to include entire day and residential treatment facilities devoted to behavioral intervention for learners with autism, inclusion models, applications in public schools, home programs, community settings, and adult education programs. Today, ABA procedures are now being implemented with individuals with autism spectrum disorder (ASD) internationally.

Rationale or Underlying Theory

The ABA model addresses behavior scientifically and views behavior from a functional vantage point. This model sees specific responses as those selected for survival by the function they perform. Behavior is examined objectively and viewed as evolving from people’s histories of

interactions with their environments. Scientific investigation is conducted in the real-world laboratory, and behavior is analyzed to determine systematic relationships between conditions of the environment and resultant behavior.

Goals and Objectives

In ABA interventions, socially-valid behavior-change goals that are beneficial to learners are of primary importance. In autism intervention, goals focus on behavior change in areas of behavioral deficits, including communication, social, and play behavior, and areas of behavioral excess, including repetitive behavior patterns and problem behaviors, such as self-injury, aggression, property destruction, and tantrums. Overall goals focus on building age- and developmentally-appropriate skills to improve independent functioning in home, school, and community settings. Questions to guide goal development include:

- What skills are interfering with the learning process, and how can we decrease them?
- What skills are necessary for the learner to function within school settings?
- What skills are necessary for the learner to be able to transition to a less restrictive setting or classroom?
- What skills are appropriate to the learner's developmental level?
- What appropriate skills are needed to serve the function of problem behavior?
- What skills are needed to develop independent functioning in home, vocational, and community settings?
- What skills are needed to increase the ability for the learner to make informed choices, becoming their own advocates, and controlling their environment in an effort to improve overall quality of life?

Treatment Participants

Treatment procedures developed from the science of ABA have been used with individuals with

ASD across the age range. It is recommended that treatment begin as soon as a child receives a diagnosis, and this can now occur before 2 years of age. When ABA interventions are begun at this early age, it is recommended that it be combined with *developmental approaches* to intervention. The majority of research studies, particularly those evaluating the efficacy of discrete trial interventions, have been conducted with younger children on the autism spectrum. Fewer studies have been conducted with adolescents and even fewer with adults. In addition, procedures documented with individuals with Asperger's disorder are limited to *social narratives*, *video modeling*, and *self-management packages* (see below). Finally, at least one study has found negligible effects of intensive ABA interventions for children with Rett syndrome.

Treatment Procedures

Treatment procedures developed from the science of ABA, heretofore referred to as ABA interventions, have a behavioral emphasis as their foundation. The cornerstone is *differential reinforcement*, where desired behaviors are reinforced, and undesired behavioral excesses are not reinforced. By reinforcing behaviors that are more functional than the problem behavior, inappropriate behaviors are thereby replaced with appropriate behavior. For learners with ASD, ABA interventions are commonly on a continuum of instructor-directed activities in low-distraction, isolated environments to interventions that have a higher degree of shared control between the instructor and learner, with more peers, in naturalistic school, home, and community environments. Descriptions of these interventions are as follows:

- *Discrete trial intervention*: This enhances learning through simplifying and individualizing instruction. Initial treatment often focuses on intense training of small, discrete skills through repeated opportunities to respond to trials. This helps present instruction in a clear

manner with the general format for a training trial as follows:

- The instructor presents a clear cue (instruction or question) to the learner who is attending to the instructor or task at hand.
 - The instructional cue may be followed by a prompt to help the learner to respond if needed.
 - The learner responds correctly or incorrectly to the instructor's cue.
 - The instructor delivers a reinforcer if the response is correct or feedback if it is incorrect, with a statement, such as, "Try again", or "No."
 - There is a brief 1–3-s pause before the next trial begins.
- *Antecedent-based interventions:* These involve the modification of environmental events that occur before a target behavior is produced, with the aim of preventing problem target behaviors and setting the occasion for competing appropriate behaviors. For example, providing a fast instructional pace prevents the occurrence of competing repetitive behavior, and *providing choices of tasks and preferred materials* increases interest level and motivation. Other antecedent interventions include (1) providing cues about schedule changes, (2) providing materials that the learner can engage with to compete with interfering behavior (such as using a squeeze ball while walking to reduce hand flapping), (3) allowing the learner to practice known skills while learning new skills to increase success and motivation, (4) errorless learning, and (5) priming, by exposing the student to aspects of the lesson ahead of time.
 - *Errorless learning/teaching:* The instructor prevents or minimizes learner errors by providing the most assistance necessary for the learner to make the correct responses early in teaching the skill. While assistance is gradually faded, it is provided to prevent incorrect responses throughout teaching.
 - *Modeling:* This procedure involves demonstrating to the learners the targeted behavior for them to imitate. This can be done "in vivo" or with videotaped models. With *video modeling*, learners watch a video of the targeted behavior as a preliminary step in teaching. This technique is commonly used to teach social skills, appropriate academic behavior, and play skills.
 - *Natural language interventions:* Hallmarks of natural language interventions are following the learner's communicative initiations for access to preferred items and activities. The instructor sets up the environment with preferred items and activities, and after the learner indicates a desire for the item, the instructor prompts the learner to use an elaborated and appropriate form of communication. Prompts to communicate are faded over repeated opportunities to communicate requests for the items, such that the preferred item becomes the cue for appropriate communication rather than reliance on external instructor prompts. These interventions have been referred to as *incidental teaching*, *the natural language paradigm*, and *pivotal response training*.
 - *Functional communication training:* When children are using problem behavior to communicate a need to gain something desired or avoid something undesired, appropriate communicative behaviors are taught to replace these problem behaviors. Widely targeted communicative responses include requesting preferred items, attention, a break, or protesting undesired activities or items.
 - *Prompts and prompt fading:* Prompts are extra cues used to effectively guide the learner's response and are faded during the course of treatment. Prompting strategies include:
 - *Fading prompt intensity:* This is done gradually, over a series of successive trials, where progressively less intense stimuli are used to guide the learner to make a correct response.
 - *Least-to-most prompting hierarchies:* Also referred to as a system of *least intrusive prompts*, this provides the learner an opportunity to perform the response on each trial. If the learner does not respond correctly after an instructional cue, the teacher

provides more assistance (e.g., a verbal prompt). If, after a short latency, the learner fails to make the correct response, the instructor provides even more assistance (e.g., a model). This is followed by even more intrusive prompting (e.g., a physical prompt) until a correct response is achieved.

- *Graduated guidance*: This employs prompts of decreasing intrusiveness and is typically used to ensure errorless responding. For example, an instructor teaching a learner to ride a bicycle would begin with full physical prompting and gradually fade to partial physical prompting and then to shadow prompting by keeping his hands close to the learner, as the learner gradually gains physical control over the response.
- *Time delay*: When using a time-delay prompting strategy, the instruction is provided, and after a brief delay (usually a few seconds), the prompt is provided.
- *Script/Script fading*: Verbal statements, in written or audio format, are provided to the learner to repeat in social/communicative situations, such as having a conversation and initiating to a peer. The scripts are faded over teaching sessions.
- *Shaping*: New responses that are not yet in the learner's repertoire are shaped through reinforcement of successive approximations to the targeted response. For example, if a learner were learning to request a preferred toy by pointing, the teacher would first reinforce the child if they reached for the object, and then over successive trials, the child would be required to make a more specific finger-pointing response to gain access to the toy.
- *Task analysis/chaining*: This involves breaking down complex skills that have many steps into their component parts, such as multi-step vocational, self-care, leisure, and independent academic behaviors. Then, each step of the chain is taught individually. Examples of behaviors that are task analyzed and taught through chaining are shoe tying, bed making, and operating a computer.
- *Visual supports*: Visual stimuli are used to aid the individual with ASD to engage in appropriate behavior. These stimuli can include pictures, words, objects, labels, scripts, and visual boundaries. Widely used visual supports include (1) *picture activity schedules*, which provide the steps to engage in a sequence of independent play, vocational, or self-care activities; (2) *visual schedules* which provide the learner with support to independently transition across activities; and (3) *scripts*, which can assist individuals during social exchanges.
- *Picture exchange communication system (PECS)*: Learners are provided with *visual supports* in the form of pictures that are exchanged with a listener during communicative interactions. Communicative skills in the PECS system include (1) spontaneous requesting of items, activities, assistance, and breaks; (2) commenting; (3) building sentence structure; and (4) responding to "What do you want?"
- *Pivotal response training*: Pivotal skills known to affect large areas of learning are the focus of this intervention. Attention, motivation, responding to multiple cues, self-management, and self-initiation are skills that provide the foundation upon which widespread generalization of learning can occur. Characteristics of pivotal response training include using learner interests in the context of play; varied materials and responses; reinforcement of attempts to communicate; shared control; and using natural and direct reinforcers.
- *Self-management*: The individual is taught to independently regulate their own behaviors by setting their own goals, accurately record and monitor their own behavior, and reward themselves for engaging in desired targets.
- *Peer- and sibling-based interventions*: Same-aged peers or siblings can support the learning of the individual with ASD using behavioral strategies. While these have customarily involved social skills training, additional areas of training have involved the implementation of *natural language training*, *discrete trial intervention*, and *picture exchange communication systems*.
- *Parent-implemented interventions*: Parents have been successfully trained to use

behavioral intervention strategies to build appropriate skills and reduce problem behaviors using all the ABA-based procedures delineated in this section.

- *Social narratives*: These describe situations with examples of desired responding and relevant cues to display those behaviors and are often presented in a short-story format with salient pictures. They are used as a precursor to an upcoming event and are often used to teach appropriate social skills and address problem behaviors.
- *Programming for generalization*: Skills are taught using the above-described strategies across persons, places, language cues, and settings, including teachers, parents, and other significant others in school, home, and community settings.

Efficacy Information

Expert panels and task forces have reviewed ABA interventions for individuals with ASD. Criteria for an evidenced-based practice included multiple publications of peer-reviewed, experimentally controlled research in scientific journals with individuals with ASD, across different investigators or research groups. ABA practices have been determined as meeting the stringent criteria developed by the National Professional Development Center on Autism Spectrum Disorders and the National Autism Center's Standards Project. In addition, ABA practices are endorsed by the United States Surgeon General and in reports of the New York Department of Health Early Intervention Program, as well as the Maine Administrators of Services for Children with Disabilities. Additional organizations that endorse ABA as a scientifically proven approach include:

- Autism Speaks
- American Academy of Neurology
- American Academy of Family Pediatrics
- American Academy of Pediatrics
- American Academy of Occupational Therapy Association
- American Psychological Association

- American Speech-Language-Hearing Association
- Society for Developmental and Behavioral Pediatrics
- Autism Society of America
- National Institute of Child Health and Human Development
- National Institute of Mental Health

Outcome Measurement

Socially meaningful, observable behavioral outcomes are the foundation of ABA interventions. Individualized, observable, and measurable target behaviors are continually assessed, with 10 to 40 trials per day as a standard in many ABA programs. Data are collected on the frequency, intensity, duration, and accuracy of targeted behaviors to both identify pretreatment levels of functioning, as well as evaluating response to treatment. Criterion is established at the beginning of treatment to provide clear indicators of mastery of the target behavior, and data-based decisions are made regarding modification of treatment, if necessary. Data outcomes are summarized at regular intervals for evaluation from the client, family, funders, and other stakeholders. Successful outcomes are those where the targeted behavior change has been achieved and that the change has maintained in the presence of natural contingencies, as well as generalized across persons, settings, and other relevant situations where the behavior occurs. In addition, social validity data, interviews, and rating scale data are collected typically from consumers, caregivers and other stakeholders to ensure treatment outcomes are socially significant and appropriate. Finally, assessment from other professionals of overall intellectual and academic functioning, as well as medical status, is recommended.

Qualifications of Treatment Providers

It is recommended that ABA programs are supervised by individuals who have certification by the

Behavior Analyst Certification Board® (BACB®) as a Board Certified Behavior Analyst® (BCBA®). Standards for certification as a BCBA® can be found in the Consumer Information section of www.BACB.com. In addition to certification as a BCBA, many states have now enacted licensure regulations to practicing behavior analysis, with certification as a BCBA as a requirement for licensure in most states. BCBA® certification does not guarantee experience in delivering ABA services to persons with ASD. Thus, additional expertise in delivering ABA services to persons with autism is advised.

Additional training in areas including causes and characteristics of autism, curriculum, assessments, autism-specific intervention, and family concerns is recommended. Please refer to *Applied Behavior Analysis Treatment of Autism Spectrum Disorder: Practice Guidelines for Healthcare Funders and Managers* for a complete review of these recommendations.

Persons who deliver treatments that are developed and supervised by a BACB®-approved provider must have demonstrated competency in following written lesson plans, data collection, and behavior reduction plans using ABA procedures described above. In addition to the BACB credential, the BACB® now provides the Registered Behavior Technician® (RBT®) credential for behavioral technicians who implement direct intervention under the supervision of a BCBA®.

The following websites contain further information that may be useful to consumers:

- The Association for Behavior Analysis - www.abainternational.org
- The Association for Science in Autism Treatment - www.asatonline.org
- The ABA Autism Special Interest Group - www.autismsig.org (or www.abainternational.org/Special_Interests/autism.asp).
- The Behavior Analyst Certification Board - www.BACB.com
- The Cambridge Center for Behavioral Studies - www.behavior.org
- The National Standards Report - www.nationalautismcenter.org
- The National Professional Development Center on Autism Spectrum Disorders - <http://autism.fpg.unc.edu>

See Also

- Behavior Analysis
- Behavior Modification
- Behavioral Curricula
- Behaviorism
- Behaviorist Theory
- Early Intensive Behavioral Intervention (EIBI)
- Education
- Lovaas Approach
- Lovaas, O. Ivar
- Motivation
- Operant Conditioning
- UCLA Young Autism Project

References and Reading

- Behavior Analyst Certification Board. (2017). *BCBA/BCaBA task list* (5th ed.). Littleton: Author.
- Carr, E. G., & Durand, V. M. (1985). Reducing behavior problems through functional communication training. *Journal of Applied Behavior Analysis*, 18, 111–126.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2020). *Applied behavior analysis* (3rd ed.). Hoboken: Pearson.
- Green, G. (1996). Evaluating claims about treatments for autism. In C. Maurice (Ed.), G. Green, & S. C. Luce (Co-Eds.), *Behavioral intervention for young children with autism: A manual for parents and professionals* (pp. 15–28). Austin: PRO-ED.
- Green, G. (2001). Behavior analytic instruction for learners with autism: Advances in stimulus control technology. *Focus on Autism and Other Developmental Disabilities*, 16, 72–85.
- Howard, J. S., Sparkman, C. R., Cohen, H. G., Green, G., & Stanislaw, H. (2005). A comparison of intensive behavior analytic and eclectic treatments for young children with autism. *Research in Developmental Disabilities*, 26, 359–383.
- Hyman, S. L., Levy, S. E., & Myers, S. M. (2020). Identification, evaluation, and management of children with autism spectrum disorders. *Pediatrics*, 145(1), e20193447. <https://doi.org/10.1542/peds.2019-3447>.
- Koegel, L. K., Koegel, R. L., Harrower, J. K., & Carter, C. M. (1999). Pivotal response intervention I:

Overview of approach. *Journal of the Association for Persons with Severe Handicaps*, 24, 174–185.

Lovaas, O. I. (1977). *The autistic child: Language development through behavior modification*. New York: Irvington.

Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.

Matson, J. L., Benavidez, D. A., Compton, L. S., Paclawskyj, T., & Baglio, C. (1996). Behavioral treatment of autistic persons: A review of research from 1980 to the present. *Research in Developmental Disabilities*, 17, 433–465.

McClannahan, L. E., & Krantz, P. J. (2006). *Teaching conversation to children with autism: Scripts and script fading*. Bethesda: Woodbine House.

Peters-Scheffer, N., Didden, R., Korzilius, H., & Sturmey, P. (2011). A meta-analytic study on the effectiveness of comprehensive ABA-based early intervention programs for children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5(1), 60–69.

Reichow, B. (2012). Overview of meta-analyses on early intensive behavioral intervention for young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42, 512–520.

Sallows, G. O., & Graupner, T. D. (2005). Intensive behavioral treatment for children with autism: Four-year outcome and predictors. *American Journal on Mental Retardation*, 110, 417–438.

U.S. Department of Health and Human Services. (1999). *Mental health: A report of the surgeon general*. Rockville: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Center for Mental Health Services, National Institutes of Health, National Institute of Mental Health.

Wolf, M. M., Risley, T. R., & Mees, H. L. (1964). Application of operant conditioning procedures to improve behavior problems of an autistic child. *Behavior Research and Therapy*, 1, 305–312.

Wong, C., Odom, S. L., Hume, K. A., Cox, C. W., Fettig, A., Kurcharczyk, S., et al. (2015). Evidence-based practices for children, youth, and young adults with autism spectrum disorder: A comprehensive review. *Journal of Autism and Developmental Disorders*. Advance online publication. <https://doi.org/10.1007/s10803-014-2351-z>.

Zwaigenbaum, L., Bauman, M. L., Choueiri, R., Kasari, C., Carter, A., Granpeesheh, D., Mailloux, Z., Roley, S. S., Wagner, S., Fein, D., Pierce, K., Buie, T., Davis, P. A., Newschaffer, C., Robins, D., Wetherby, A., Stone, W. L., Yirmiya, N., Estes, A., Hansen, R. L., McPartland, J. C., & Natowicz, M. R. (2015). Early intervention for children with autism spectrum disorder under 3 years of age: Recommendations for practice and research. *Pediatrics*, 136(Supplement 1), S60–S81.

Apprenticeships

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Synonyms

Vocational training

Definition

One method to include individuals with autism in the workplace is through the use of apprenticeships. An apprenticeship involves direct on-the-job training by a qualified individual. This structured training may be supplemented by classroom-based training. Apprenticeship programs typically last for 1–5 years and are concluded based upon completing a pre-specified number of hours, meeting specific job competencies, or a combination of both. Apprenticeships allow individuals with autism to gain job skills in integrated settings while earning an income, which typically increases over time.

See Also

- Supported Employment
- Vocational Training

References and Reading

- Lynn, I., & Mack, D. (2008). *Multiple strategies for improving transition. Outcomes of youth with disabilities: Issue paper on increasing access to apprenticeship opportunities*. Washington, DC: Institute for Educational Leadership and HeiTech Services. <http://www.dol.gov/odep/categories/youth/apprenticeship/ApprenticeshipIssuePaper.pdf>.
- Scholl, L., & Mooney, M. (2003, 2004). Youth with disabilities in work-based learning programs: Factors that influence success. *The Journal for Vocational Special Needs Education*, 26 (1, 2), 4–16.

Appropriate Adaptation

- [Reasonable Accommodation](#)

Appropriate Educational Placement

- [Least Restrictive Environment \(LRE\)](#)

Apraxia

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Synonyms

[Dyspraxia](#)

Short Description or Definition

Apraxia is a neurological motor disorder that impairs an individual's ability to perform voluntary movements in the absence of weakness, paralysis, or neuromuscular slowness (Duffy 1995; Freed 2000; Vinson 2007). Apraxia is subtyped according to the area of impairment and ranges in severity from mild to severe (NINDS n.d.).

Categorization

Apraxia is a neurological motor disorder that is subtyped according to the area of impairment. Some apraxias affect general motor movements, while others affect the speech mechanism. Apraxias that affect general motor movements include limb-kinetic apraxia (impairment in the ability to make fine precise movements with an arm or leg), ideomotor apraxia (impairment in the

ability to make appropriate movements in response to a command), and ideational apraxia (impairment in the ability to coordinate activities with sequential movements, such as dressing). Apraxias specific to the speech mechanism are nonverbal oral apraxia (impairment in the ability to carry out facial movements on command) and apraxia of speech (impairment in coordinating mouth and speech movements to produce speech), also known as verbal apraxia or dyspraxia. Different types of apraxia may occur alone or together (Freed 2000; NINDS n.d.).

There are two types of apraxia of speech, acquired and developmental. Acquired apraxia of speech, or adult apraxia of speech, occurs after neurological insult, such as a stroke (e.g., ASHA 2007; Freed 2000). Developmental apraxia of speech, also known as childhood apraxia of speech, is a developmental motor speech disorder. Both acquired and developmental apraxias of speech are characterized by impaired volitional movements of the speech structures. The core features of acquired apraxia of speech and childhood apraxia of speech overlap. However, because of the significant difference in the time in development at which the disorders manifest themselves, there may be important differences in associated features (Massen 2002; ASHA 2007). This encyclopedia entry focuses mainly on childhood apraxia of speech, also known as developmental apraxia.

Epidemiology

Acquired apraxia of speech arises from neurological insult such as stroke, degenerative disease, trauma, or tumor (Freed 2000). Stroke is the leading cause of apraxia of speech in adults followed by degenerative diseases (Duffy 1995). In contrast, childhood apraxia of speech may occur as the result of unknown causes or in association with complex neurobehavioral disorders of known or unknown origins (e.g., fragile X syndrome). Recent research suggests that childhood apraxia of speech may have a genetic basis (for a review, see ASHA 2007). Little data is

available on the prevalence of acquired apraxia of speech or childhood apraxia of speech. Studies examining population estimates of childhood apraxia of speech are based on clinical referral. Population estimates range from one to two children per thousand to 2.4–4.3% of children referred with speech delay of unknown origin, with a 3:1 male to female ratio (Delaney and Kent 2004 as cited by ASHA 2007; Shriberg et al. 1997).

Natural History, Prognostic Factors, and Outcomes

The developmental course of childhood apraxia of speech is not well documented (Shriberg et al. 1997). The signs and symptoms of childhood apraxia of speech may vary across children and within the same child over time (ASHA 2007). This is further complicated by the finding that children appear to move in and out of the childhood apraxia of speech diagnostic category at different points in development. For example, children initially diagnosed with an articulation disorder may go on to receive a diagnosis of childhood apraxia of speech and vice versa (Hall 1989). Research suggests that children with apraxia of speech make improvements between preschool and school-age in articulating single words and in their overall intelligibility. However, these children continue to have difficulty sequencing multisyllabic words and persistent concomitant language impairments (Lewis et al. 2004).

Clinical Expression and Pathophysiology

Children suspected of having apraxia of speech typically demonstrate deficits in at least one of the following domains: nonspeech motor behaviors, motor speech behaviors, speech sounds and structures, prosody, language, metalinguistic/phonemic awareness, and literacy. Nonspeech motor behavior deficits are characterized by general awkwardness or clumsiness, impaired volitional oral movements, mild delays in oral motor

development, mildly low muscle tone, hyper- or hyposensitivity in the oral area, and oral apraxia. Motor speech deficits are characterized by slow development of speech, reduced phonetic inventory, multiple speech sound errors, reduced percentage of consonants correct, and unintelligibility. Both the nonspeech and motor speech deficits observed in apraxia are also found in other speech sound disorders such as speech delay and dysarthria. Characteristics that appear to be distinctive of childhood apraxia of speech include reduced vowel inventory, vowel errors, inconsistency of articulation errors, increased errors in longer or more complex syllable and word shapes, groping, unusual errors, persistent or frequent regression, differences in performance of automatic versus volitional activities (with volitional activities being more affected), and errors in the ordering of sounds, syllables, morphemes, or even words (for a review, see ASHA 2007).

Syllable and prosody production are also affected in children with apraxia of speech. The atypical prosody observed in individuals with suspected childhood apraxia of speech may be attributed to prolonged sound production and prolonged pauses between sounds, syllables, or words. As a result of these prolongations, the sounds, syllables, and/or words are produced as separate entities. This gives the listener the impression of staccato speech. Other prosodic deficits include reduced variability in pitch or loudness, which result in excessive-equal stress (i.e., all or most syllables in a word receiving prominent stress) during speech production (for a review, see ASHA 2007).

Most children suspected of having apraxia of speech have significant concomitant language deficits. These impairments are often more significant and more persistent than in children with other speech sound disorders (Lewis et al. 2004). Language difficulties include poor phonological awareness (a skill that lies at the foundation of literacy development), difficulty perceiving and producing rhymes, and counting syllables. Other areas of difficulty include deficits in word identification and spelling (Lewis et al. 2004; Marquardt et al. 2002).

Evaluation and Differential Diagnosis

There is currently no definitive diagnostic marker for childhood apraxia of speech, and many of the characteristics of childhood apraxia of speech overlap with other speech sound disorders. Thus, the challenge for both researchers and clinicians is to differentiate childhood apraxia of speech from other speech sound disorders. The characteristics that appear to be distinctive to childhood apraxia of speech include reduced vowel inventory, vowel errors, inconsistency of errors, increased errors in longer or more complex syllable and word shapes, groping, unusual errors, regression, differences in performance of automatic versus volitional activities (with volitional activities being more affected), and errors in sequencing. However, these patterns may also be found in children who do not fit the overall pattern of apraxia of speech. Because apraxia impairs motor coordination, clinicians must first rule out muscle weakness, sensory loss, a comprehension deficit, or incoordination as the underlying cause of the impairment. Currently, the minimum age of diagnosis of childhood apraxia of speech ranges from under 2 years of age to under 4 years of age (for a review, see ASHA 2007).

Standardized tests that focus on nonverbal oral motor and/or motor speech performance that may be used to diagnose apraxia include the Apraxia Profile Preschool and School-Age Versions (Hickman 1997), the Kaufman Speech Praxis Test for Children (Kaufman 1995), the Oral Speech Mechanism Screening Examination, Third Edition (St. Louis and Ruscello 2000), Screening Test for Developmental Apraxia of Speech – Second Edition (Blakely 2001), the Verbal Dyspraxia Profile (Jelm 2001), and the Verbal Motor Production Assessment for Children (Hayden and Square 1999). While these tests may assist in diagnosis, they lack normative data and behavioral standards to use in test interpretation and clear behavioral standards on which to base treatment decisions (McCauley and Strand 2008).

Differential diagnosis of childhood apraxia of speech may also include examination of non-speech motor behaviors. Children with apraxia

of speech are more likely to demonstrate general awkwardness or clumsiness, impaired volitional oral movements, mild delays in oral motor development, mildly low muscle tone, hyper- or hypo-sensitivity in the oral area, and oral apraxia (Davis et al. 1998; McCabe et al. 1998; Shriberg et al. 1997). However, many of these motor behaviors are characteristic of dysarthria. In addition, clinicians may use the sequential motion rate task, conversational speech and reading, and repeating words of increasing length to examine motor speech behaviors during diagnostic evaluations (Freed 2000). The sequential motion rate task is one of the most sensitive assessments for differentiating apraxia of speech from other motor disorders (e.g., Davis et al. 1998; Freed 2000; Nijland et al. 2002).

Although there is not currently a validated list of diagnostic features that may be used to differentiate apraxia of speech from other speech sound disorders, three features are consistent with a deficit in the planning and execution of motor movements. These features are (1) inconsistent errors on consonants and vowels in repeated production of syllables or words, (2) lengthened and disrupted coarticulatory transitions between sounds and syllables, and (3) inappropriate prosody (ASHA 2007).

Treatment

There have been few treatment studies of apraxia of speech. Of the treatment studies conducted, none met the highest level of evidence for treatment efficacy (ASHA 2007; Pannbacker 1998). To date, management of childhood apraxia of speech is similar to that of dysarthria and other articulation disorders. Treatment is most often focused on improving speech production. Basic approaches to treating apraxia of speech include (1) linguistic approaches, (2) motor-programming approaches, (3) linguistic-motor programming combinations, and (4) treatments with specific sensory and gestural cueing techniques. Linguistic approaches focus on teaching the child the sounds and the rules regarding sound sequences and sound use. Motor-programming techniques

use principles of motor learning to teach children to acquire the skills needed to make sounds and sequences of sounds accurately and consistently. Other approaches combine linguistic and motor-programming intervention strategies. Finally, there are programs that involve the child's senses such as vision, touch, as well as being touched, to help cue the child about some aspect of the speech sound he or she is attempting to make (Hall 2000; ASHA 2007). For children with significantly reduced intelligibility, treatment goals may focus on facilitating overall communication through the use of Augmentative and Alternative Communication (ASHA 2007).

See Also

- Ataxia
- Developmental Apraxia
- Dyspraxia
- Motor Planning
- Nonverbal Oral Apraxia
- Praxis
- Verbal Apraxia

References and Reading

- American Speech-Language-Hearing-Association. (2007). Childhood apraxia of speech [Technical report]. Available from www.asha.org/policy. Retrieved 25 Jan 2011
- Blakely, R. W. (2001). *Screening test for developmental apraxia of speech* (2nd ed.). Austin: Pro-Ed.
- Davis, B., Jakieslski, K., & Marquardt, T. (1998). Developmental apraxia of speech: Determiners of differential diagnosis. *Clinical Linguistics and Phonetics*, 12, 25–45.
- Delaney, A. L., & Kent, R. D. (2004). *Developmental profiles of children diagnosed with apraxia of speech*. Poster session presented at the annual convention of the American-Speech-Language-Hearing Association, Philadelphia.
- Duffy, J. R. (1995). *Motor speech disorders: Substrates, differential diagnosis, and management*. St. Louis: Mosby.
- Freed, D. (2000). *Motor speech disorders: Diagnosis and treatment*. San Diego: Singular.
- Hall, P. K. (1989). The occurrence of developmental apraxia of speech in a mild articulation disorder: A childhood apraxia of speech study. *Journal of Communication Disorders*, 22, 265–276.
- Hall, P. (2000). Part 1: Speech characteristics of the disorder. *Language, Speech, and Hearing Services in Schools*, 31, 169–172.
- Hayden, D., & Square, P. (1999). *Verbal motor production assessment for children*. San Antonio: The Psychological Corporation.
- Hickman, L. (1997). *Apraxia profile*. San Antonio: The Psychological Corporation.
- Jelm, J. M. (2001). *Verbal dyspraxia profile*. DeKalb: Janelle.
- Kaufman, N. (1995). *Kaufman speech Praxis test for children*. Detroit: Wayne State University Press.
- Lewis, B. A., Freebairn, L. A., Hansen, A. J., Iyengar, S. K., & Taylor, G. H. (2004). School-age follow-up of children with apraxia of speech. *Language, Speech and Hearing Services in Schools*, 35, 122–140.
- Marquardt, T., Sussman, H. M., Snow, T., & Jacks, A. (2002). The integrity of the syllable in developmental apraxia of speech. *Journal of Communication Disorders*, 26, 129–160.
- Massen, B. (2002). Issues contrasting adult acquired versus developmental apraxia of speech. *Seminars in Speech and Language*, 23, 257–266.
- McCabe, P., Rosenthal, J. B., & McLedo, S. (1998). Features of developmental dyspraxia in the general speech impaired population? *Clinical Linguistics and Phonetics*, 12, 105–126.
- McCauley, R. J., & Strand, E. A. (2008). A review of standardized tests of nonverbal oral speech motor performance in children. *American Journal of Speech-Language Pathology*, 17, 81–91.
- National Institute of Neurological Disorders and Stroke. (n.d.). Apraxia information page. Available from www.ninds.nih.gov/disorders/apraxia/apraxia.htm?css=print. Retrieved 25 Jan 2011
- Nijland, L., Maassen, B., van der Meulen, S., Gabreels, F., Kraaimaat, F. W., & Schreuder, R. (2002). Coarticulation patterns in children with developmental apraxia of speech. *Clinical and Linguistic Phonetics*, 16, 461–483.
- Pannbacker, M. (1998). Management strategies for developmental apraxia of speech: A review of the literature. *Journal of Communication Disorders*, 21, 363–371.
- Shriberg, L. D., Aram, D. M., & Kwiatkowski, J. (1997). Developmental apraxia of speech: I. Descriptive and theoretical perspectives. *Journal of Speech, Language, and Hearing Research*, 40, 273–285.
- St. Louis, K. O., & Ruscello, D. (2000). *Oral speech mechanism screening examination* (3rd ed.). Austin: Pro-Ed.
- Vinson, B. (2007). *Language disorders across the lifespan* (2nd ed.). Clifton Park: Thomson Delmar Learning.

Apraxia of Speech (AOS)

- Verbal Apraxia

Arab Views on Autism

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Historical Background

Autism spectrum disorder (ASD) is a neurodevelopmental disorder, mainly presenting with impairments in communication, distinct patterns of social interaction, and restricted or repetitive patterns of behavior (American Psychiatric Association). ASD includes autistic disorder, Asperger syndrome, and pervasive developmental disorder not otherwise specified (PDD-NOS). The symptoms of ASD typically manifest by the age of 3 and persist throughout one's life. However, the condition is commonly missed and not diagnosed until much later in the child's life, especially if the child's symptoms are mild to moderate in severity. Research into ASD has increased dramatically over the past few decades, but unfortunately, most of this research has been limited to affluent, English-speaking nations (Sharpe et al. 2011).

The Arab world consists of 22 member nations in North Africa and the Middle East. These nations include Algeria, Bahrain, the Comoros Islands, Djibouti, Egypt, Iraq, Jordan, Kuwait, Lebanon, Libya, Morocco, Mauritania, Oman, Palestine, Qatar, Saudi Arabia, Somalia, Sudan, Syria, Tunisia, the United Arab Emirates, and Yemen. The population of the Arab world exceeds 400 million, with a diversity of religious, ethnic, and linguistic communities. While the majority of people in the Arab world adhere to Islam and speak some dialect of Arabic, there are various ethno-religious communities which may speak an indigenous language other than Arabic and practice a religion other than Islam.

Historically, children throughout the Arab world who began to display symptoms of autism, such as repetitive behavior and poor social skills, were thought to have been affected by black magic or the "evil eye" and were shunned from society. In recent years, however, pediatricians in

these countries have begun to recognize the symptoms of autism, and countless cases which would previously go unreported are now being diagnosed early enough to allow for successful intervention. ASD research is a very new field in the Arab world (Hussein and Taha 2013).

Organizations such as The Egyptian Autistic Society are being established in order to increase awareness among doctors and parents so as to assist in early diagnosis and treatment of autism (Mendoza 2010). The Autism Research and Treatment (ART) Center at the King Saud University in Saudi Arabia is the first institution in the region to carry out basic research on autism.

The prevalence of ASD worldwide is estimated to be 1 case per 160 children (World Health Organization 2019). However, the vast majority of prevalence studies reviewed by Fombonne (2005, 2009) and Williams (Williams et al. 2006) have been performed in Western nations or affluent Asian nations such as Japan. As such, there is limited research concerning the diagnosis of children with ASD in less affluent, non-Western nations where services and programs for children with ASD are less available. This lack of research has led some writers to make the unwarranted assumption that autism is rare in non-Western cultures (Sanua 1984; Zhang et al. 2006). Available data suggests the prevalence of ASD to be 1.4, 29, and 59 per 10,000 children in Oman (Al-Farsi et al. 2011), the United Arab Emirates (Eapen et al. 2007), and Saudi Arabia (Aljarallah et al. 2006), respectively. The observed lower incidence of ASD in Arab nations in comparison to Western nations is likely due in part to the lack of specialists available in the Arab region to properly diagnose ASD (Mostafa 2011).

While much progress has been made in the past couple of decades in regard to autism research, diagnosis, and treatment in the Arab world, there is still much progress that needs to be made, particularly in autism research. Many experts argue that most of the current research and treatments for autism have been made primarily for an Anglo-Saxon population and thus may not be relevant to Arabs (Sharpe et al. 2011). It is clear that in order to properly address autism in the Arab world, a concerted effort must be

implemented involving government support to research institutions and families, as well as creating campaigns to raise awareness and reduce stigma. Not long ago, many Arab parents would have refused to enroll their child in a school with another autistic child. Although attitudes have now changed, it is not uncommon for parents to hide their autistic child from society due to feelings of shame and embarrassment.

Overview of Current Research

In a review study conducted by Alnemary et al. which examined all published ASD research before January 2014, it was found that autism research was conducted in 13 countries (Alnemary et al. 2017). The majority of the studies were conducted in Saudi Arabia, followed by Egypt and Oman. The majority of the publications addressed the biological aspects of ASD, while the vast minority focused on treatments and interventions. The majority of research areas have begun to show gradual growth over the past few years. Additionally, the majority of ASD publications produced in Arab countries were funded by governmental agencies rather than private organizations. A total of 142 publications were identified from 1992 to January 2014, with a dramatic increase in the number of ASD publications beginning in 2008.

However, while much progress is being made, ASD research in the Arab world remains very limited. There are several factors that may be contributing to the lack of research being conducted. Arab governments and organizations do not prioritize funding for mental health and related fields, putting a constraint on ASD research (Jaalouk et al. 2012). Additionally, many Arab researchers are not able to submit their research to international journals due to language barriers. The majority of publications from the Arab world are written in Arabic, English, or French, and only those written in English are included in international databases (Sarhan 2012).

Another review study analyzing ASD research in Arab countries from the years 1992 to 2012 found that although the majority of identified

papers were published after 2008, autism research is not yet a priority in Arab countries (Hussein and Taha 2013). In accordance with the previous study, it was found that the majority of autism research in Arab countries was concerned with the etiology of the disorder as opposed to treatments or services. This may be due to the fact that medical fields in the Arab world are far more advanced than rehabilitation and educational fields. Furthermore, the majority of etiological studies are concerned with autoimmune and biochemical markers of ASD rather than imaging and genetics. This is most likely due to the lower cost of these methods and the lack of funding available to researchers. Additionally, etiology studies produced in the Arab world do not differ from those produced in Western countries, which, in accordance with previous research conducted by Bristol et al., suggests that no ethnic or environmental factors have been proven to cause autism (Bristol et al. 1996).

The few genetic studies that have been published either have a very limited sample size or are case reports. However, due to Arab culture being characterized by a relatively high amount of consanguineous marriages, there has recently been a growing level interest in genetic research. According to a genetic study conducted in 2009, it was found that one-third of a cohort of Saudi Arabian children with autism had a history of consanguinity (Al-Salehi et al. 2009). In the Arab world, most consanguineous marriages are between first cousins, with the practice being much more prevalent in rural areas. They may also be more prevalent in some countries over others, comprising about 34–80% of all marriages in Saudi Arabia depending on the location. While these findings do not suggest a direct link between consanguinity and autism in Arab countries, the higher incidence of autism among Saudi Arabian families make them ideal candidates for screening studies for genetic variations. An example of this can be seen in a 2008 study in which 88 families with consanguineous marriages and a high incidence of autism were recruited from several countries, including Oman, Saudi Arabia, Jordan, and Kuwait. The DNA of family members was compared in order to identify recessive mutations.

Large chunks of missing DNA which followed the recessive rule was identified in some families, and while the missing regions differed among families, they affected at least six genes that are known to play a role in the development of ASD (Neergaard 2008).

In a 2014 review conducted by Salhia et al., the results of several case-control studies conducted in Bahrain, Saudi Arabia, or Oman were analyzed. The risk factors investigated in these studies included suboptimal breastfeeding, lead exposure, serum osteopontin, maternal and paternal age, cesarean section, and prenatal complications. A lack of colostrum intake and delayed breastfeeding were associated with a higher risk of ASD (Salhia et al. 2014). Additionally, exclusive and prolonged breastfeeding – defined as 24 months or longer – was associated with a lower risk of developing ASD. Higher levels of lead and osteopontin were found in ASD patients, and the disorder was more prevalent among children of mothers who had delivered via cesarean section or experienced antenatal complications.

Additionally, it was found that the levels of several biomarkers were altered in autistic patients compared to healthy controls. Biomarkers that were significantly elevated in autistic patients include lipid peroxidation (Al-Gadani et al. 2009), glutathione peroxidase (Al-Mosalem et al. 2009), superoxide dismutase, sodium-potassium adenosine triphosphatase (Al-Farsi et al. 2012), lactate, saturated fatty acids (El-Ansary et al. 2011), and homocysteine. Biomarkers which were found in lower levels among autistic patients include vitamin E (Al-Gadani et al. 2009), glutathione, folate, vitamin B12 (Ali et al. 2011), and some polyunsaturated acids. Autistic patients also had elevated levels of serum osteopontin, and the levels were positively correlated to the severity of autism (Salhia et al. 2014). The majority of current ASD population studies conducted in Arab countries have several limitations, such as small sample sizes and nonrepresentative populations due to sampling from pediatric wards. Additionally, the majority of ASD research in the Arab world originates from three countries: Saudi Arabia, Egypt, and Oman. Consequently, very little conclusions

or observations may be drawn concerning the Arab world in general.

Future Directions

Although factors such as consanguinity, multiparity, and closely spaced pregnancies are common in the Arab world and would provide for excellent population to study the genetic components of the disorder, there is currently a lack of research into the etiology of ASD in Arab countries. Additionally, research concerning dietary habits and drug usage is almost nonexistent. Research into treatments and services is lacking to an even greater extent, the result of which can be seen in Arab society. There are currently some efforts being done in order to identify autistic symptoms among children in psychiatric wards. The few options available to parents of autistic children are generally very costly and present a large financial burden on the family, and research into the economics of autism in the Arab world is strongly needed.

Furthermore, while general views on autism in Arab societies are slowly changing, there remains much progress to be made in terms of raising awareness and informing the public. It is not uncommon for parents to ignore early symptoms of autism or to hide their children from society due to feelings of shame or not being informed of the options available to them. Raising awareness among parents may allow for early diagnosis and intervention of autistic children. It seems that the most obvious and necessary steps to be taken in the near future involve government funding of research institutions and development of programs specifically geared toward autism diagnosis and intervention.

References and Reading

- Al-Farsi, Y. M., Al-Sharbaty, M. M., Al-Farsi, O. A., Al-Shafae, M. S., Brooks, D. R., & Waly, M. I. (2011). Brief report: Prevalence of autistic spectrum disorders in the Sultanate of Oman. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-010-1094>.

- Al-Farsi, Y. M., Al-Sharbati, M. M., Waly, M. I., Al-Farsi, O. A., Al-Shafae, M. A., Al-Khaduri, M. M., et al. (2012). Effect of suboptimal breast-feeding on occurrence of autism: A case-control study. *Nutrition*, 28, e27–e32.
- Al-Gadani, Y., El-Ansary, A., Attas, O., & Al-Ayadhi, L. (2009). Metabolic biomarkers related to oxidative stress and antioxidant status in Saudi autistic children. *Clinical Biochemistry*, 42, 1032–1040.
- Al-Mosalem, O. A., El-Ansary, A., Attas, O., & Al-Ayadhi, L. (2009). Metabolic biomarkers related to energy metabolism in Saudi autistic children. *Clinical Biochemistry*, 42, 949–957.
- Al-Salehi, S. M., Al-Hifthy, E. H., & Ghaziuddin, M. (2009). Autism in Saudi Arabia: presentation, clinical correlates and comorbidity. *Transcultural Psychiatry*, 46, 340–347.
- Ali, A., Waly, M. I., Al-Farsi, Y. M., Essa, M. M., Al-Sharbati, M. M., & Deth, R. C. (2011). Hyperhomocysteinemia among Omani autistic children: A case-control study. *Acta Biochimica Polonica*, 58, 547–551.
- Aljarallah, A., Alwaznah, T., Alnasari, S., & Alhazmi, M. (2006). A study of autism and developmental disorders in Saudi children. Riyadh: Report, King Abdulaziz City for Science and Technology. <http://www.kacst.edu.sa>. Accessed 20 Apr 2019.
- Alnemary, F. M., et al. (2017). Autism research: Where does the Arab world stand? *Review Journal of Autism and Developmental Disorders*, 4(2), 157–164. <https://doi.org/10.1007/s40489-017-0104-6>.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). American Psychiatric Association Publishing, Washington, DC. text revised.
- Bristol, M. M., Cohen, D. J., Costello, E. J., et al. (1996). State of the science in autism: Report to the National Institutes of Health. *Journal of Autism and Developmental Disorders*, 26, 121–154.
- Eapen, V., Mabrouk, A. A., Zoubeydi, T., & Yunis, F. (2007). Prevalence of pervasive developmental disorders in preschool children in the UAE. *Journal of Tropical Pediatrics*. <https://doi.org/10.1093/tropej/fml091>.
- El-Ansary, A. K., Bacha, A. G., & Al-Ayahdi, L. Y. (2011). Plasma fatty acids as diagnostic markers in autistic patients from Saudi Arabia. *Lipids in Health and Disease*, 10, 62.
- Fombonne, E. (2005). The changing epidemiology of autism. *Journal of Applied Research in Intellectual Disabilities*, 18(4), 281–294.
- Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. *Pediatric Research*, 65(6), 591–598.
- Hussein, H., & Taha, G. R. (2013). Autism spectrum disorders: A review of the literature from Arab countries. *Middle East Current Psychiatry*. <https://doi.org/10.1097/01.XME.0000430433.49160.a4>.
- Jaalouk, D., Okasha, A., Salamoun, M. M., & Karam, E. G. (2012). Mental health research in the Arab world. *Social Psychiatry and Psychiatric Epidemiology*. <https://doi.org/10.1007/s00127-012-0487-8>.
- Mendoza, R. L. (2010). The economics of autism in Egypt. *American Journal of Economics and Business Administration*, 2(1), 12–19.
- Mostafa, A. (2011). Addressing autism in the Arab world. *Nature Middle East*. <https://doi.org/10.1038/middleast.2011.147>.
- Neergaard, L. (2008). Genes from Middle East families yield autism clues. Harvard Medical School, Genetics. Available at: <http://abcnews.go.com/Health/Autism/story?id=5349700&page=1%23.T7AqpOh7ppA>. Accessed 23 Apr 2019.
- Salhia, H. O., et al. (2014). Systemic review of the epidemiology of autism in Arab gulf countries. *Neurosciences (Riyadh, Saudi Arabia)*, 19(4), 291–296.
- Sanua, V. D. (1984). Is infantile autism a universal phenomenon? An open question. *International Journal of Social Psychiatry*, 30(3), 163–177.
- Sarhan, W. (2012). The state of science in mental and behavioral disorders in the Arab region: Research needs and relevance to classification. *The Arab Journal of Psychiatry*, 23(1), 1–11. <http://arabjournalpsychiatry.com/>. Accessed 25 Apr 2019.
- Sharpe, D. L., Baker, D. L., & Mohammad-Reza, M. (2011). The financial side of autism: Private and public costs. In *A comprehensive book on autism spectrum disorders* (pp. 275–296). InTech, IntechOpen. <https://doi.org/10.5772/18588>.
- Williams, J. G., Higgins, J. P. T., & Brayne, C. E. G. (2006). Systematic review of prevalence studies of autism spectrum disorders. *Archives of Disease in Childhood*, 91(1), 8–15.
- World Health Organization. (2019). Questions and answers about autism spectrum disorders. <http://www.who.int/en/>. Accessed 20 Apr 2019.
- Zhang, J., Wheeler, J. J., & Richey, D. (2006). Cultural validity in assessment instruments for children with autism from a Chinese cultural perspective. *International Journal of Special Education*, 21(1), 109–114.

Arciform Rhythm

► Mu Rhythm

ARD Committee

► Admission, Review, and Dismissal Committee (ARD Committee)

Argentina and Autism

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Historical Background

The recognition of autism in Argentina follows, by a few years, the recognition of ASD in Europe and the USA. In general, since then, Argentine physicians and other professionals have been using the DSM classification system (APA 1980).

A few studies by Argentine researchers give an idea about the prevalence of ASD. Lejarraga et al. (2008) conducted developmental screening in a large sample ($n = 839$) of apparently healthy children of 0–5 years of age, in the Municipality of San Isidro, Province of Buenos Aires. In this study, the authors found 11 children with ASD, which indicates a prevalence of 13/1000 (1 in 76 children). Another prospective study was done in 2014 in children of the Province of Santa Fe by Dr. Francisco Astorino and his collaborators (personal communication). The authors of this study found a prevalence of 1/128 in children between 18 and 36 months. No information is available about the prevalence of autism in older children in Argentina.

It is important to understand and acknowledge the strong influence of psychoanalysis among psychologists and psychiatrists in Argentina. Because psychoanalysis puts emphasis on the subjective experiences of the patients, and does not use any systematic classification, it makes it impossible to generate accurate data on prevalence of ASD, or produce replicable descriptions of the treatments.

Legal Issues, Mandates for Service

Until 1970, most Argentine children with disabilities received their grade education in so-called schools for special education (*Escuelas de*

Educación Especial). The process of integrating students with special needs into the regular public school system started in the 1960s, first with integration of children with hearing and blindness disabilities. In the 1970s, the so-called schools for special recovery (*Escuelas de Recuperación*) were intended to serve children with minor difficulties, referred to such schools by professionals in the public school system. In 2008, the country adhered to the International Convention on Disabled Persons; although, the effort to achieve inclusion of students with autism in the Argentine system of public education is progressing slowly.

In the mid-1980s, it was a remarkable increase in work towards the integration of people with developmental disabilities in Argentina. A new presidential administration created the National Advisory Commission for the Integration of Persons with Disabilities in 1987, to provide a nation-wide platform for the coordination of different actions and proposals to achieve greater social integration of people with disabilities. In 1988, the Ministry of Education and Culture of the Nation launched its National Integration Plan. As a consequence of this renewed focus, many jurisdictions began to engage in school integration projects in those years, mostly supported by special education boards.

The National Education Law of 2006 (Argentine Law 26.026) established the right to inclusive school education throughout the country at all educational levels and modalities, to ensure the integration of the students with disabilities, according to each person's potential.

In 2005, the government of the City of Buenos Aires approved a Resolution (Buenos Aires City Law 650/2005) for people with disabilities that mandated early diagnosis and early therapeutic intervention, aiming at reducing the incidence of autism and severe communication disorders. This resolution also mandated a survey of these disorders, and highlighted the need for an increase in identification, and appropriate medical care of autism and severe communication disorders.

In 2014, the National Congress passed Law 27.043 on the "Comprehensive and Interdisciplinary Approach of Persons Who Have Autism Spectrum Disorders." This law established as goals of

national interest all actions conducive to the following: (a) a comprehensive and interdisciplinary approach to people with Autistic Spectrum Disorders (ASD); (b) clinical and epidemiological research; (c) professional training in early detection, diagnosis, treatment, and research; and (d) dissemination and access to benefits for persons who have Autism Spectrum Disorders. Several, but not all, of the country's provinces have adhered to the national law, or have produced similar legislation.

The treatment of these topics generated a public debate around systematic research on diagnostic classifications, the effects of "labeling" when using a diagnosis, and the so-called "pathologization" of children, as well as the appropriate types of early intervention for each child.

Overview of Current Treatments and Centers

Medical Treatment: In Argentina, the overall health-care system is split between four layers of different but often overlapping systems. The country has a public health system, with public health centers and hospitals providing care that is free, or almost free of cost to the patients. These hospital and community health centers are funded by the federal administration, and/or depend on funding provided by provincial and municipal governments. These institutions deliver services to the largest segment of the population. Another layer of health-care services is delivered by social services (*Obras Sociales*) organized and funded by various workers' unions (*Sindicatos*). Most Argentine employers and employees would regularly contribute to these organizations. The quality and volume of health care provided by social services administered by workers' unions is in direct relationship with the number of workers contributing a portion of their salaries to the unions. Some *Obras Sociales* have large hospitals in Buenos Aires, and in other cities. Yet another layer of health care is delivered by private HMO (*organizaciones prepagas para el cuidado de la salud*). People buy a membership, and pay a

monthly fee to a HMO to receive a certain level of health care from professionals affiliated to the organization. Some of these HMOs own large clinics and hospitals in many regions of the country. The fourth layer of health care in the Argentine system is found in some private local clinics and hospitals, and also private individual health professionals across the country. With such web of providers, which often overlap in the most populated areas, it is difficult to evaluate current medical treatments for autism.

Treatment options vary greatly according to region of the country, and type of health insurance coverage. A fair assessment of the situation would indicate that treatment options for autism depend on the qualifications of the centers, and the competence of the professionals delivering such care.

Across the country, many relatively small private interdisciplinary centers deliver care to children with autism. Most of these centers offer a combination of multiple treatments, and the number of professionals using evidence-based therapies in these interdisciplinary centers seems to be increasing. The professionals' combined form of treatments could include therapies such as counseling, speech therapy, occupational therapy, psychomotricity, *psicopedagogía* (psychology of learning), music therapy, and others. Most interdisciplinary clinics include a physician, usually a child neurologist or child psychiatrist. The counseling received by the children is often based on psychoanalysis (Barcala et al. 2003).

Across the country, an increase in public awareness on the need for early detection of autism is evident; see Ministerio de Salud de la Provincia de Buenos Aires, and Rattazzi (2014). Among professionals, there is also increased emphasis in the use of evidence-based treatments like Denver, SCERTS, TEACCH, floor time, etc. For details, see Cadaveira and Waisburg (2014), García Coto (2001), and Valdez (2016).

Barcala and collaborators (2003) conducted a study in Buenos Aires aiming at obtaining an estimate of accessibility to services for children of autism in the public health system in the city. The authors could not obtain information from all Municipal Hospitals due to insufficient systematization, lack of electronic records, and also

because some health care providers reported that they employed psychoanalytical frameworks which precluded them from submitting detailed quantifiable data.

Another comprehensive study by Barcala et al. (2006) presented an excellent picture of the mental health services offered in public hospitals in Buenos Aires. The authors surveyed all the pediatric mental health services (*Servicios de Psicopatología Infantil*) in the city. Although they were unable to obtain the actual number of children with ASD served in those public hospitals, their study included descriptions of the treatment offered in two psychiatric hospitals with specialized treatments. The authors found that among 32 hospitals, only 9 provided treatment for ASD. Most hospitals offered individual therapy delivered by psychologists, many offered treatments delivered by *psicopedagogas* (professionals specialized in psychology of learning), while a few treatments were done by occupational therapists. Only two of the hospitals in this study had child psychiatrists on staff, providing pharmacological treatment.

In 2006, the theoretical framework most often employed by the staff at Mental Health Centers in public Hospitals in Buenos Aires was psychoanalysis (Barcala et al. 2006). Although, a recent review of the number of centers and academic courses that include ABA and other developmental/educational approaches (Denver, TEACH, etc.) indicates an increase in the use of evidence-based treatments in Argentina.

Overview of Research Directions

Currently, it appears that the country does not have an institutional research agenda for ASD. A review of existing research supported by the National Council for Scientific and Technical Research (CONICET), the main public-funded research organization in the country, indicates that many individual projects are targeting basic research on autism; however, apparently there are no programs or established research groups focused on autism. Some clinical studies on autism by individual scientists are also supported by CONICET.

Overview of Training

Ongoing training of providers (teachers, health care professionals, and others): Some universities are beginning to offer postgraduate education on ASD. Currently, the most relevant academic program in Autism and Asperger Syndrome is a postgraduate program offered by Universidad Católica Argentina (UCA). This program, directed by Drs. Víctor Ruggieri and Daniel Valdéz, presents content supported by evidence-based science. Also, Facultad Latinoamericana de Ciencias Sociales (FLACSO), another major university in Argentina, offers an online postgraduate course on practices for education and inclusion of children with ASD.

A review of the curricula offered in Psychology Departments at several universities across the country indicated that psychoanalysis continues to be the most commonly taught theory; although many more syllabi by faculty in Argentine universities now include behavioral and evidence-based approaches for the treatment of ASD.

ASD is part of the curricula in medical residencies in child psychiatry, child neurology, and neuropsychiatry at several universities. However, ASD is not included in the curricula for most pediatric residencies. While most pediatric residents would receive a few lectures on ASD, few of these residents would obtain adequate training for ASD detection, let alone treatment of children with ASD. Lessons on autism are taught in most of the Health Allied Schools, like those offering academic training on occupational therapy, physical therapy, speech therapy, and educational psychology; however, autism rarely appears as the main subject in a course.

Social Policy and Current Controversies

One important controversy in Argentina is found in concerns of parents and professionals regarding the classification of behavioral/mental conditions, and pervasive effects of labeling on the individuals. Labeling manifests conflict between accepting the

child differences as his/her way of being and the attempt to “cure” or make the child as “normal.”

Most of the formal education for children with special needs continues to be segregated in schools for Special Education. Since relevant legislation passed in 2014, there is major push for achieving greater inclusion of children with ASD in public and private schools. Nevertheless, some parents continue to have concerns about the extent to which the trend would mean a genuine move towards inclusion, and wonder about the possibility of ending up with an actual reduction of services.

In sum, the current diagnosis of autism in Argentina shows increase in awareness and decrease in stigmatization, mainly thanks to the efforts of several parents’ organizations and the effects of their public role in educating the community (see, for example, associations such as TGD padres, <http://tgd-padres.com.ar>; Programa Argentino para Niños, Adolescentes y Adultos con Condiciones del Espectro Autista (PANAACEA) <http://www.panaacea.org/>; and Asociación Argentina de Padres de Autistas <http://apadea.org.ar/>).

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Barcala, A., Alvarez Zunino, O., Torricelli, F., Bianchi, V., & Ynoub, R. (2003). *Niños/as con trastornos psicopatológicos graves: representaciones sociales de los profesionales de salud mental que trabajan en los servicios de Salud pertenecientes al gobierno de la Ciudad de Buenos Aires*. Paper presented at Primeras Jornadas sobre Representaciones Sociales, Investigación y Prácticas. Buenos Aires: Universidad de Buenos Aires.
- Barcala, A., Brio, M., Vila, N., Gotlieb, M., Oliva, M., Baumann, N., Bravo, F., Ciolfi, L., Etcheverry, L., & Lefebvre, T. (2004). Caracterización de la consulta institucional en psicosis y autismo infantil en la ciudad de Buenos Aires. Estudio de caso. *Revista de Investigaciones en Psicología*, 9(2), 7–24.
- Barcala, A., Torricelli, F., Brio, M. C., Vila, N., Ynoub, R., & Marotta, J. (2006). Prevalencia institucional en psicosis y autismo infantil en la ciudad de Buenos Aires. In *Anales de la Fundación Alberto J Roemmers* (Vol. XVII). Buenos Aires: Talleres Gráficos de la Prensa Médica Argentina SRL.
- Cadaveira, A., & Waisburg, C. (2014). *Autismo: guía para padres y profesionales*. Buenos Aires: Paidós.
- García Coto, M. A. (2001). Tratamiento del Autismo: Programa neurocognitivo. In *Autismo: enfoques actuales para padres y profesionales de la Salud y la Educación*. Buenos Aires: Fundación para el Desarrollo de los Estudios Cognitivos.
- Guía de ayuda para la detección de los trastornos del espectro autista. (2014). [http://regionsanitaria1.com/documents/GUIA-TEA-2014%20\(1\).pdf](http://regionsanitaria1.com/documents/GUIA-TEA-2014%20(1).pdf).
- Klin, A., et al. (2000). Brief report: Interrater reliability of clinical diagnosis and DSM-IV criteria for autistic disorder: Results of the DSM-IV autism field trial. *Journal of Autism and Developmental Disorders*, 30(2), 163–167.
- Lejarraga, H., Menendez, A. M., Menzano, E., Guerra, L., Biancato, S., Pianelli, P., Del Pino, M., Fattore, M. J., & Contreras, M. M. (2008). Screening for developmental problems at primary care level: A field programme in San Isidro, Argentina. *Paediatric and Perinatal Epidemiology*, 22, 180–187.
- Rattazzi, A. (2014). The importance of early detection and early intervention for children with autism spectrum conditions. *Vertex*, 25(116), 290–294.
- Valdez, D. (Ed.). (2016). *Autismos, Estrategias de Intervención entre lo clínico y lo Educativo*. Buenos Aires: Paidós.

Resources

Parent-Based Organizations

- Asociación Argentina de Padres de Autistas. <http://apadea.org.ar/>
- Programa Argentino para Niños, Adolescentes y Adultos con Condiciones del Espectro Autista (PANAACEA). <http://www.panaacea.org/>
- TGD padres. <http://tgd-padres.com.ar>

Education

- Facultad Latinoamericana de Ciencias Sociales. Academic program on Necesidades educativas y prácticas inclusivas en trastornos del espectro autista. <http://flacso.org.ar/formacion-academica/necesidades-educativas-practicas-inclusivas-y-trastornos-del-espectro-autista/>
- Universidad Católica Argentina, Postgraduate program Diplomatura en Autismo y Síndrome de Asperger. <http://www.uca.edu.ar/index.php/site/index/es/uca/facultad-de-ciencias-medicas/cartelera/diplomatura-en-autismo-y-sindrome-de-asperger/>

Aricept

- Donepezil: Definition

Ariclaim

► Duloxetine: Definition

Aripiprazole

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Synonyms

7-[4-[4-(2,3-Dichlorophenyl)-1-piperazinyl]butoxy]-3,4-dihydro-2(1H)-quinolinone; Abilify

Definition

Aripiprazole is a prescription drug in the family of atypical antipsychotics initially FDA-approved for medical use in the year 2002 with the chemical formula $C_{23}H_{27}Cl_2N_3O_2$ (Fig. 1). This compound is a partial agonist of the dopamine₂ (D₂) receptor and the serotonin type 1A (5-HT_{1A}) receptor and is an antagonist of the serotonin type 2A (5-HT_{2A})

receptor. This drug is mostly metabolized by the enzymes CYP2D6 and CYP3A4 of cytochrome P450. Oral formulations in solution, tablet, and rapidly dissolving forms are FDA-approved for the treatment of schizophrenia in adolescents and adults; the acute treatment of manic and mixed episodes in pediatric patients ages 10 years and older and adults with bipolar I disorder, as an adjunct to lithium or valproate; the treatment of major depressive disorder in adults as an adjunctive treatment; and the treatment of irritability in pediatric patients ages 6–17 years with autistic disorder. A short-acting injectable form is FDA-approved for the acute treatment of agitation in adults with schizophrenia or bipolar I disorder. A long-acting injectable form is FDA-approved for the treatment of schizophrenia in adults. Observed side effects include weight gain, nausea, akathisia, headache, insomnia, agitation, anxiety, and mild transient somnolence.

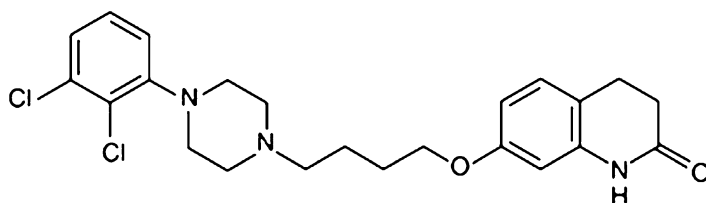
See Also

► Atypical Antipsychotics

References and Reading

- Aripiprazole. (n.d.). Retrieved from the ChemSpider Wiki: <http://www.chemspider.com/RecordView.aspx?rid=365ceb61-2923-4e82-bd96-e849caa18b11>
- Lauriello, J., Biehl, T. K., Bustillo, J. R., & Jenkusky, S. M. (2009). Schizophrenia and other psychotic disorders. In L. W. Roberts (Ed.), *Clinical psychiatry essentials* (pp. 163–180). Philadelphia: Lippincott Williams & Wilkins.
- Marcus, R. N., Owen, R., Kamen, L., Manos, G., McQuade, R. D., Carson, W. H., & Aman, M. G. (2009). A placebo-controlled, fixed-dose study of aripiprazole in children and adolescents with irritability associated with autistic disorder. *Journal of the American Academy of Child and Adolescent Psychiatry* 48(11):1110–1119.

Aripiprazole,
Fig. 1 Chemical Structure



- Owen, R., Sikich, L., Marcus, R. N., Corey-Lisle, P., Manos, G., McQuade, R. D., Carson, W. H., & Findling, R. L. (2009). Aripiprazole in the treatment of irritability in children and adolescents with autistic disorder. *Pediatrics* 124(6):1533–1540.
- Printz, D. J., & Lieberman, J. A. (2006). Aripiprazole. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 277–283). Washington, DC: American Psychiatric Publishing.
- Smith, B. D., & Richards, M. P. (2010). Therapeutic response to psychiatric emergencies. In L. W. Roberts (Ed.), *Clinical psychiatry essentials* (pp. 481–497). Philadelphia: Lippincott Williams & Wilkins.
- U.S. Food and Drug Administration. (2010). *Atypical antipsychotics drug information*. Retrieved from: <http://www.fda.gov/Drugs/DrugSafety/PostmarketDrugSafetyInformationforPatientsandProviders/ucm094303.htm>

corpus callosum, abnormal genitalia, seizures, ataxia and dystonia, and syndromic and non-syndromic intellectual disability. Some people with *ARX* mutations with intellectual disability but without structural brain malformations show features of autism including speech delay, impaired social interactions, and stereotyped repetitive behaviors. However, mutations in this gene are not typically found in individuals with autism.

See Also

- ▶ Epilepsy
- ▶ X-Linked Traits

Aristaless-Related Homeobox Gene

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Synonyms

ARX

Definition

The Aristaless-related homeobox gene on the X chromosome produces a homeodomain transcription factor that, by regulating numerous genes, is crucial for many processes during embryonic development, especially the proliferation and migration of neurons. *ARX* is expressed in forebrain interneurons that release the inhibitory neurotransmitter gamma-aminobutyric acid (GABA).

Mutations in *ARX* can run in families or occur sporadically. These mutations cause a range of X-linked developmental disorders that include lissencephaly (“smooth brain”), agenesis of the

References and Reading

- Chaste, P., Nygren, G., Anckarsater, H., Rastam, M., Coleman, M., Leboyer, M., Gillberg, C., & Betancur, C. (2007). Mutation screening of the *ARX* gene in patients with autism. *American Journal of Medical Genetics Part B Neuropsychiatric Genetics*, 114B(2), 228–230.
- Friocourt, G., & Parnavelas, J. G. (2010). Mutations in *ARX* result in several defects involving GABAergic neurons. *Frontiers in Cellular Neuroscience*, 4(4), 1–11.
- Fulp, C. T., Cho, G., Marsh, E. D., Nasrallah, I. M., Labosky, P. A., & Golden, J. A. (2008). Identification of *Arx* transcriptional targets in the developing basal forebrain. *Human Molecular Genetics*, 17(23), 3740–3760.
- Kato, M., Das, S., Petras, K., Kitamura, K., Morohashi, K., Abuelo, D. N., Barr, M., Bonneau, D., Brady, A. F., Carpenter, N. J., Cipero, K. L., Frisone, F., Fukuda, T., Guerrini, R., Iida, E., Itoh, M., Lewanda, A. F., Nanba, Y., Oka, A., Proud, V. K., Saugier-Verber, P., Schelley, S. L., Selicorni, A., Shaner, R., Silengo, M., Stewart, F., Sugiyama, N., Toyama, J., Toutain, A., Varags, A. L., Yanazawa, M., Zackai, E. H., & Dobyns, W. B. (2004). Mutations of *ARX* are associated with striking pleiotropy and consistent genotype-phenotype correlation. *Human Mutation*, 23(2), 147–159.
- Stromme, P., Mangelsdorf, M. E., Shaw, M. A., Lower, K. M., Lewis, S. M., Bruyere, H., Lutchera, V., Gedeon, A. K., Wallace, R. H., Scheffer, I. E., Turner, G., Partington, M., Frints, S. G., Fryns, J. P., Sutherland, G. R., Mulley, J. C., & Gecz, J. (2002). Mutations in the human ortholog of Aristaless cause X-linked mental retardation and epilepsy. *Nature Genetics*, 30(4), 441–445.

Arlington Central School District v. Murphy 2006 (IDEA Not Authorizing Expert Evaluations)

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Definition

Arlington Central School District v. Murphy
Arlington Central School District (ACSD) v. Murphy concerned parents’ ability to recover costs associated with a successful claim against a local or state educational agency under fee-shifting provisions in the Individuals with Disabilities Act (IDEA). The United States Supreme Court held that expert witness fees were not “costs” as provided for in the act.

Implications for Parents and Professionals Involved in IDEA Actions

The IDEA provides a fee-shifting provision to allow parents prevailing in actions under the Act to recover their costs associated with the litigation. Following *ACSD v. Murphy*, expenses associated with services provided by experts do not fall within the provisions of the act and thus were not recoverable by parents. Allowable costs identified by the Supreme Court as being provided for in IDEA are shown below.

Authorized by 20 U.S.C. 1415(i)(3) (B) (IDEA)	
	Attorney’s fees
Authorized by 28 U.S.C. 1920	
	Fees of the clerk and marshal
	Fees of the court reporter for all or any part of the stenographic transcript necessarily obtained for use in the case
	Fees and disbursements for printing and witnesses
	Docket fees under section 1923 of title 28

(continued)

	Compensation of court-appointed experts (limited to \$40 per diem plus travel expenses under 28 U.S.C. 1821), compensation of interpreters, and salaries, fees, expenses, and costs of special interpretation services under section 1828 of title 28
	Fees for exemplification and copies of papers necessarily obtained for use in the case

Current and Future

On March 17, 2011, in direct response to the Supreme Court’s ruling in *ACSD v. Murphy*, Senator Tom Harkin (D-IA), the Chairman of the Health, Education, Labor, and Pensions Committee; Senator Barbara Mikulski (D-MD); and Senator Bernie Sanders (I-VT) introduced to the Senate the IDEA Fairness Restoration Act, and Congressmen Chris Van Hollen (D-MD 8) and Pete Sessions (R-TX 32) introduced an identical bill in the House of Representatives. This act would amend IDEA’s fee-shifting provision so that “the term ‘attorneys’ fees’ shall include the fees of expert witnesses, including the reasonable costs of any test or evaluation necessary for the preparation of the parent or guardian’s case in the action or proceeding.” The bill is similar to ones that have been introduced in the past two Congresses but which never made it out of committee. As of May, 2011, the bill remains in committee in both the Senate and House of Representatives.

See Also

► [Eligibility \(for Services Under IDEA/ADA, etc.\)](#)

References and Reading

Arlington Central School District v. Murphy, 548 U.S. 291 (2006).
Council of Parent Attorneys and Advocates. (2011). *Reinstate parents’ right to expert witness fees*. Retrieved

July 5, 2012, from <http://www.copaa.org/public-policy/copaas-major-legislative-priorities/reinstate-parents-right-to-expert-witness-fees/>

IDEA Fairness Restoration Act, S.613 HR.1208, 112th Cong., 1st Sess. (2011).

Arousal

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Definition

Arousal is defined as a physiological preparedness to perceive and react to environmental stimuli and is produced by the activation of the sympathetic branch of the autonomic nervous system. An arousal response may be identified through increased heart rate, increased blood pressure, increased sweat gland activity, and dilation of the pupils (Romanczyk and Gillis 2006), and can be indicative of a variety of emotions such as fear, anxiety, excitement, or feelings of competitiveness (Romanczyk and Gillis 2006). Typically, a moderate amount of arousal is optimal for learning (Baron et al. 2006).

Historical Background

An individual's state of arousal can provide valuable insight about a variety of socially significant indicators such as anxiety levels, ability to recognize and react to fearful or stressful situations, and the ability to identify and regulate emotions. Each of these skills is crucial to social functioning and to forming meaningful relationships throughout life. A more in-depth understanding of the history and current state of the arousal literature, as well as a review of typical and atypical demonstrations of arousal, will illustrate the critical role it plays in autism research and the important contributions it can make to interventions. Since the 1960s, numerous studies have measured arousal

in individuals with autism using a variety of different measures. Initially, three main hypotheses explaining arousal dysfunction were studied: hyperarousal, hypoarousal, and difficulties with arousal modulation. Hutt et al. (1965, 1966, 1968) found evidence of hyperarousal which contributed to the hypothesis that individuals with autism are chronically overly aroused and that they regulate their arousal through stereotypical, repetitive motor behaviors. Other studies similarly found that individuals with autism are overly aroused in response to social and nonsocial stimuli and, especially, in response to novel stimuli when compared with typical individuals and individuals with other developmental disabilities (Hermelin and O'Connor 1968; James and Barry 1980). In contrast to the hyperarousal hypothesis, other early investigators found evidence of hypoarousal, that is, chronic underarousal in individuals with autism when compared to typically developing individuals (DesLauriers and Carlson 1969). Early proponents of this hypothesis suggested that individuals with autism engage in stereotypical, repetitive motor behaviors to increase sensory stimulation. Still, others found evidence of fluctuations between both hyper- and hypoarousal dependent on the environment, stimuli, and developmental level of the individuals (Hermelin and O'Connor 1970; Ornitz and Ritvo 1968), thereby forming the hypothesis that individuals with autism experience difficulties in modulating arousal in general, whether hypo or hyper. The varied results noted are likely due to a host of limitations including inconsistencies with terminology and diagnosis identification; most early studies were published prior to the publication of the DSM III-R which more clearly outlined the criterion for an autism diagnosis. This limitation causes uncertainty in the actual diagnosis of participants studied. In addition, the early measurement tools used to measure physiological functioning were likely uncomfortable and may have caused heightened anxiety and arousal for participants. Finally, many studies did not collect baseline data making it difficult to determine resting states of arousal and actual fluctuation, hyper, or hypo states (Goodwin et al. 2006).

Current Knowledge

Technological advances and continued research have provided investigators with the tools to study arousal more uniformly. Currently, heart rate, blood pressure, and skin conductance tests are the most common types of physiological arousal tests studied. Despite the use of more uniform measures, discrepancies in results remain, even within tests. As with early research, more recent studies continue to find that individuals with autism demonstrate elevated levels of physiological arousal (Bellini 2006; Goodwin et al. 2006). One important study found that while individuals with autism were less reactive (i.e., hypoaroused) to environmental stressors than typically developing controls, on average, the autism group demonstrated higher heart rate during baseline and in stressful situations. Interestingly, however, the autism group displayed nearly 50% less variance in heart rate between baseline and stressful situations when compared to the control group (Goodwin et al.). Other studies have found that individuals with autism experienced less arousal than typical individuals when viewing sad or fearful stimuli but more arousal when viewing neutral stimuli (Bolte et al. 2008). Combined, this research may indicate that individuals with autism are chronically hyperaroused or that they experience more arousal than typical individuals in testing situations but demonstrate less arousal during stressful, sad, or fearful situations. Still, other recent work has found no significant group differences in arousal levels between individuals with autism and typical individuals (Ceponiene et al. 2003; Kemner et al. 2002). Inconsistent results may be due to the developmental level of the individuals studied (Dawson and Lewy 1989) and the variability seen within individuals with autism (Zahn 1986), in combination with differences in the measures of arousal (e.g., heart rate, skin conductance).

While much of the discussion in early arousal research was focused on the relations between arousal and stereotypical repetitive motor behaviors, recently, there has been increased discourse regarding the relation between arousal, social

anxiety, and social functioning in individuals with autism. Bellini (2006) recently proposed the developmental pathways model to explain the role of arousal in social anxiety in individuals with autism. Specifically, the model suggests that social anxiety is indirectly the product of temperament, of which physiological hyperarousal is intricately related. Individuals with a temperament marked by increased physiological arousal may withdraw socially in order to prevent overarousal; this social withdrawal may then lead to social skills deficits. Bellini's work is founded in previous work that indicates that individuals that demonstrate hyperarousal levels may be more likely to develop social anxiety in response to negative peer interactions when compared to individuals with chronically lower arousal levels (Biederman et al. 1995). In support of this theoretical perspective, other investigators have recently found that individuals with autism are more aroused by social stimuli than typically developing individuals and, as a result, may avoid them in order to prevent hyperarousal. This avoidance, in turn, may significantly contribute to social functioning deficits (Corden et al. 2008; Nacewicz et al. 2006; Schultz 2005). While more research is needed to confidently draw the link between arousal, social withdrawal, and social competency, recent studies have shown preliminary but promising leads in the field.

Future Directions

Technological advances in data collection will be imperative to the future study of arousal. In the past, intrusive data collection techniques may have skewed results in that individuals studied may have experienced elevated levels of arousal solely based on the testing situation. If this is the case, these results may only reflect arousal conditions during intrusive testing rather than on arousal states in general. Thus, it is critical to develop the least intrusive measures possible in order to accurately and confidently draw conclusions about true arousal levels that mirror conditions in the natural environment. Similarly, in

order to answer questions about neutral arousal levels and about how individuals with and without autism will react to real-life stressors, future studies should attempt to naturalize the testing setting as much as possible and even aim to collect data in the individual's natural environment.

Other considerations that should be made include providing detailed information about the participants studied. Individuals with autism demonstrate a wide range of functioning levels, and results based on the average of high- and low-functioning individuals are difficult to generalize to any particular subset of the disorder. Further, the use of a developmentally matched control group is crucial in order to control for the effects of general maturation delays (James and Barry 1981; Rogers and Ozonoff 2005). Baseline data should be taken on every individual studied, including control subjects, to determine a range of normal and maladaptive arousal levels, and studies should look at within- and between-group variability as Goodwin et al. (2006) have done. Moreover, future researchers should conduct longitudinal studies in order to better understand the developmental course of arousal. Studying patterns and trends within groups over time may also identify individuals at higher risks for social anxiety and other internalizing or externalizing mental health issues. Finally, the recent push for incorporating biological, neurological, and physiological measures in psychological studies will also undoubtedly bring forth large gains in the field of arousal, social anxiety, and social functioning. Similarly, applied research on this topic has the potential to advance clinical work in the field of behavior and emotion regulation, social anxiety, and social functioning.

See Also

- ▶ Hypo-arousal
- ▶ Sensation Avoiding
- ▶ Sensation-Seeking
- ▶ Sensory Experiences Questionnaire
- ▶ Sensory Processing

References and Reading

- Baron, M. G., Groden, J., Groden, G., & Lipsitt, L. P. (2006). *Stress and coping in autism*. New York: Oxford University Press.
- Bellini, S. (2006). The development of social anxiety in adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 21, 138–145.
- Bernal, M. E., & Miller, W. H. (1970). Electrodermal and cardiac responses of schizophrenic children to sensory stimuli. *Psychophysiology*, 7, 155–168.
- Biederman, J., Rosenbaum, J. F., Chaloff, J., & Kagan, J. (1995). Behavioral inhibition as a risk factor for anxiety disorders. In J. S. March (Ed.), *Anxiety in children and adolescents* (pp. 61–81). New York: Guilford Press.
- Bohte, S., Feineis-Matthews, S., & Poustka, F. (2008). Brief report: Emotional processing in high functioning autism- physiological reactivity and affective report. *Journal of Autism and Developmental Disorders*, 38, 776–781. <https://doi.org/10.1007/s10803-007-0443-8>.
- Ceponiene, R., Lepisto, T., Shestakova, A., Vanhala, R., Alku, P., Naatanen, R., et al. (2003). Speech-sound-selective auditory impairment in children with autism: They can perceive but do not attend. *Proceedings of the National Academy of Sciences*, 100, 5567–5572. <https://doi.org/10.1073/pnas.0835631100>.
- Corden, B., Chilvers, R., & Skuse, D. (2008). Avoidance of emotionally arousing stimuli predicts social-perceptual impairment in Asperger's syndrome. *Neuropsychologia*, 46, 137–147. <https://doi.org/10.1016/j.neuropsychologia.2007.08.005>.
- Dawson, G., & Lewy, A. (1989). In Dawson G. (Ed.), *Arousal, attention, and the socioemotional impairments of individuals with autism*. New York: Guilford Press.
- DesLauriers, A. M., & Carlson, C. F. (1969). *Your child is asleep: Early infantile autism*. Homewood, IL: Dorsey Press.
- Goodwin, M., Groden, J., Velicer, W., Lipsitt, L., Baron, G., Hofmann, S., et al. (2006). Cardiovascular arousal in individuals with autism. *Focus on Autism and Other Developmental Disabilities*, 21, 100–123.
- Hermelin, B., & O'Connor, N. (1968). Measures of the occipital alpha rhythm in normal, subnormal, and autistic children. *The British Journal of Psychiatry*, 114, 603–610.
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. Oxford: Pergamon.
- Hutt, S. J., & Hutt, C. (1968). Stereotypy, arousal and autism. *Human Development*, 11, 277–286. <https://doi.org/10.1159/000270612>.
- Hutt, C., & Ounsted, C. (1966). The biological significance of gaze aversion with particular reference to the syndrome of infantile autism. *Behavioral Science*, 11, 346–356. <https://doi.org/10.1002/bs.3830110504>.
- Hutt, S. J., Hutt, C., Lee, D., & Ounsted, C. (1965). A behavioral and electroencephalographic study of

- autistic children. *Journal of Psychiatric Research*, 3, 181–197. [https://doi.org/10.1016/0022-3956\(65\)90028-2](https://doi.org/10.1016/0022-3956(65)90028-2).
- James, A. L., & Barry, R. J. (1980). Respiratory and vascular responses to simple visual stimuli in autistics, retardates, and normals. *Psychophysiology*, 17, 541–547.
- James, A. L., & Barry, R. J. (1981). General maturational lag as an essential correlate of early onset psychosis. *Journal of Autism and Developmental Disorders*, 11, 271–283.
- Kemner, C., Oranje, B., Verbaten, M. N., & van Engeland, H. (2002). Normal P50 gating in children with autism. *The Journal of Clinical Psychiatry*, 63, 214–217.
- Nacewicz, B. M., Dalton, K. M., Johnstone, T., Long, M. T., McAuliff, E. M., Oakes, T. R., et al. (2006). Amygdala volume and nonverbal social impairment in adolescent and adult males with autism. *Archives of General Psychiatry*, 63(12), 1417–1428. <https://doi.org/10.1001/archpsyc.63.12.1417>.
- Ornitz, E. M., & Ritvo, E. R. (1968). Perceptual inconsistency in early infantile autism. *Archives of General Psychiatry*, 18, 76–98.
- Rogers, S., & Ozonoff, S. (2005). Annotation: What do we know about sensory dysfunction in autism? A critical review of the empirical evidence. *Journal of Child Psychology and Psychiatry*, 46, 1255–1268. <https://doi.org/10.1111/j.1469-7610.2005.01431.x>.
- Romanczyk, R. G., & Gillis, J. M. (2006). Autism and the physiology of stress and anxiety. In M. G. Baron, J. Groden, G. Groden, & L. P. Lipsitt (Eds.), *Stress and coping in Autism* (pp. 183–204). New York: Oxford University Press.
- Schultz, R. (2005). Developmental deficits in social perception in autism: The role of the amygdala and fusiform face area. *International Journal of Developmental Neuroscience*, 23, 125–141. <https://doi.org/10.1016/j.ijdevneu.2004.12.012>.
- Zahn, T. P. (1986). Psychophysiological approaches to psychopathology. In M. Coles, E. Donchin, & S. Porges (Eds.), *Psychophysiology: Systems, processes, and applications* (pp. 508–610). New York: Guilford Press.
- media, the creative process, and the resulting artwork to explore their feelings, reconcile emotional conflicts, foster self-awareness, manage behavior and addictions, develop social skills, improve reality orientation, reduce anxiety, and increase self-esteem. A goal in art therapy is to improve or restore a client's functioning and his or her sense of personal well-being. Art therapy practice requires knowledge of visual art (drawing, painting, sculpture, and other art forms) and the creative process, as well as of human development, psychological, and counseling theories and techniques.
- Today, art therapy is widely practiced in a wide variety of settings including hospitals, psychiatric and rehabilitation facilities, wellness centers, forensic institutions, schools, crisis centers, senior communities, private practice, and other clinical and community settings. During individual and/or group sessions, art therapists elicit their clients' inherent capacity for art making to enhance their physical, mental, and emotional well-being. Research supports the use of art therapy within a professional relationship for the therapeutic benefits gained through artistic self-expression and reflection for individuals who experience illness, trauma, and mental health problems and those seeking personal growth (American Art Therapy Association 2013).

Historical Background of Art Therapy

Throughout time humans have used symbols and images to express themselves. From Egyptian hieroglyphics to mask making to other objects used in rituals, art has been important in creating visual records of self-expression and communication. The development of art therapy has been a process which stems from previous interests in the observation of art and human behavior. In the late nineteenth century, French psychiatrists Tardieu and Simon published studies on the similar characteristics of and symbolism in the artwork of the mentally ill. Shortly after, Ernst Kris made connections to art and psychoanalysis believing in strong links between psyche, artistic works, and

Art Therapy and Autism

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Definition of Art Therapy

Art therapy is a mental health profession in which clients, facilitated by the art therapist, use art

creative imagination. Like Freud, he believed that artists had an easier time accessing the “id” for material.

The field of art therapy really took form in the 1950s and 1960s. One of the modern pioneers of art therapy was Margaret Naumburg who was primarily an educator, second a psychotherapist, and third the first art therapist. She believed that art was a powerful vehicle in unlocking repressed material. Her perspective, often referred to as “art psychotherapy,” was based on the recognition that an individual’s most fundamental feelings and thoughts coming from the unconscious were expressed more powerfully through images rather than words. Her book, *Dynamically Oriented Art Therapy* was published in 1966 which still serves as an important text to students of the field.

Edith Kramer was another early contributor to the field. Kramer’s approach, “art as therapy,” was developed with her work with children and adolescents who were often unable to describe their feelings with words. Kramer believed that the process of making art allowed the children to access these feelings and identify them through the creative process. Her first book written in 1958, *Art Therapy in a Children’s Community*, described her initial experiences with her clients. After another 13 years of working in a hospital setting and psychiatric ward, she published *Art as Therapy with Children* in 1971. Edith Kramer along with Dr. Laurie Wilson founded the graduate program at New York University in 1976 which was one of the first successful programs and is still active today. It is important to note that Kramer believed that product was as important as process in art therapy. She felt that denying the client the gratification of the end art product was robbing them. Lastly, Kramer believed that the field of art therapy should be in the category of humanities rather than psychology.

Purpose and Underlying Theory

The intention of the art therapist is to offer and share the creative process with their clients in order for them to access their own inner healing.

Art therapy in its totality can be adapted to various theoretical approaches which exist in today’s mental health field. As part of a comprehensive art therapy training, art therapists study psychoanalytic theories, Freudian, Jungian, and others. In addition, art therapy training includes historical and theoretical perspectives of other approaches, such as gestalt, object relations, humanistic, and family therapy.

Within the context of these theories, the art therapist integrates creative modalities and uses artistic media in the sessions with their clients. Sometimes the sessions may combine verbal or “talk” therapy; however, this is not necessary, and the act of art making can be as far as the client wants to go. In any case, while the creative process is taking place, all the art therapists are constantly assessing and tuning into their clients reactions to the materials, the direction that the art is going as well as subtleties in expression and body language.

Benefits of Art Therapy with Individuals with Autism

Autism is a pervasive developmental disorder in which social interactions are the main impairment along with delayed or impaired language development. Individuals with autism are deprived of the resources from which the mind organizes and develops (Emery 2004). Rigid thinking and inability to read other’s emotions tend to be other characteristics that can impede developing healthy relationships.

There are many issues related to sensory processing that affect most if not all diagnosed with autism. Sensory processing disorder (SPD) is a neurological condition that affects the ability to process information from the five senses. Those who suffer with this condition have sensitivities that can cause great distress, discomfort, and confusion leading to behaviors that are seen as “unacceptable” to the outer world. Because of their sensory processing challenges, art making can be a particularly effective therapy for people with autism. Because autistic individuals tend to have

difficulty processing sensory input and are often nonverbal, they respond well to visual, concrete, hands-on therapies. Many people who work with this population know this and whether or not they have art therapy training, including art making in their clients' activities.

There are limitations to our knowledge of why and how therapeutic art making actually works for autistics. These limitations of understanding result from the difficulty of standardized assessments, the near impossibility of quantifying the experience of making art, and the small number of art therapists publishing on the topic. Nonetheless, the abundant amount of research literature explicates that art making is an effective, clinically sound treatment option for autism when supplemented with studies from the fields of art, art education, psychology, and other creative arts therapies (Martin 2009).

Art therapy when used appropriately with individuals with autism can help increase communication, build better social skills, develop a sense of individuality, build more purposeful relationships, and facilitate sensory integration (Betts 2005). Children in particular who are diagnosed on the autism spectrum struggle with these challenges to varying degrees, but communication in general is probably the most difficult of all (Ullmann 2010). The distinct feature of art therapy is the nonthreatening, unpressured environment that it offers to those who are nonverbal. Engaging with art media can be a fulfilling experience that can often help the individual with autism start to feel relaxed with their therapist. In addition, art therapy can incorporate strategies to help individuals build a sense of accomplishment and self-esteem which then may lead to more refined expression and desire to communicate.

Determining the appropriate art interventions for any given autistic individual relies on assessing the developmental level as well as their functionality. Within the broader developmental context, art therapy can be used to engage an autistic individual's relationships to the areas of communication, socialization, and imagination. Art therapy is known to tap into emotional issues; however, the client will probably need to work in the above three domains, before they can be

successful in accessing this higher functioning and deeper area. This can be somewhat counter-intuitive for a majority of art therapists who have extensive experience with other populations which are more capable of insight. Therefore, it is important that the therapist recognize this and do a full assessment of developmental and functioning levels at the beginning of treatment of an autistic child or adult (Ullmann 2010).

One must also keep in mind that children or adults with autism do not ignore others intentionally, but they will tune out in order to help them make sense of their world and regulate their over- or under-stimulated sensory channels. Therapists need to respect this plain fact and resist any impulse to change the process and force the individual to engage before they are ready to. Art therapy can be an excellent intervention when adapted appropriately and when the therapist has a good understanding of the needs of the population. Treatment must be flexible and open, remembering that each individual with Autism is quite different and requires a customized approach.

References and Reading

- American Art Therapy Association. (2013). Art therapy as an intervention for autism. *Art Therapy: Journal of the American Art Therapy Association*, 143–147. www.arttherapy.org
- Betts, D. J. (2005). The art of art therapy: Drawing individuals out in creative ways. *Advocate: Magazine of the Autism Society of America*, 26–27. Retrieved from <http://www.art-therapy.us/images/art-therapy.pdf>
- Dubowski, J., & Evans, K. (2001). *Art therapy with children on the autistic spectrum: Beyond words*. London: Jessica Kingsley Publishers, LTD. Retrieved from <http://www.amazon.com/Art-Therapy-Children-Autistic-Spectrum/sim/1853028258/2>.
- Emery, M. J. (2004). Art therapy as an intervention for autism, art therapy. *Journal of the American Art Therapy Association*, 21(3), 143–147.
- Martin, N. (2009). Art as an early intervention tool for children with autism. *London, England Therapy Association*, 21(3), 143–147.
- Ullmann, P. (2010). *Art therapy and children with autism: Gaining access to their world through creativity* (Vol. 2, #1). Arlington: Fusion, A Publication of the Art Therapy Alliance and International Art Therapy Association.

Articulation

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Synonyms

Pronunciation; Speech sound production

Definition

Articulation is a general term that refers to the act of producing speech sounds in the vocal tract (i.e., the movement and sequencing of physical structures including the lips, tongue, teeth, jaw, etc.). Speech sounds are often classified based on either the place of articulation (i.e., the physical structures that are involved and where the point of contact occurs between structures) or the manner of articulation (i.e., the amount/type of restriction of airflow involved).

See Also

- ▶ Phonetics
- ▶ Phonology
- ▶ Speech

References and Reading

- American-Speech-Language-Hearing-Association. (n.d.). What is language? What is speech? In *Typical speech and language development*. Retrieved April 25, 2011, from http://www.asha.org/public/speech/development/language_speech.htm
- Bowen, C. (1998). *Children's speech sound disorders: Questions and answers*. Retrieved April 25, 2011, from <http://www.speech-language-therapy.com/phonol-and-artic.htm>
- Crystal, D. (1991). *A dictionary of linguistics and phonetics* (3rd ed.). Cambridge, MA: Basil Blackwell.
- Ladefoged, P., & Maddieson, I. (1996). *The sounds of the world's languages*. Oxford: Blackwell.
- Zemlin, W. R. (1998). *Speech and hearing science: Anatomy and physiology* (4th ed.). Boston: Allyn and Bacon.

Articulation Disorders

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Synonyms

Phonological disorders; Speech delay; Speech disorder; Speech sound disorder

Short Description or Definition

Articulation disorders involve difficulty with the correct production of speech sounds. Within the literature, articulation disorders are often differentiated from phonological disorders in that articulation disorders involve motor movements, while phonological disorders refer to the underlying rules/patterns of sound production within a language.

Categorization

Articulation disorders often are classified in terms of severity (e.g., mild, moderate, severe). This rating is typically based on the type/number of errors the individual produces relative to age/developmental norms, as well as a measure of overall intelligibility.

Epidemiology

Shriberg et al. (1999) reported the prevalence of speech delay in a large sample of 6-year-olds to be 3.8% with a male-to-female ratio of 1.5:1. The comorbidity of speech delay and language impairment was reported to be 1.3%. However, estimates of the prevalence of speech sound disorders within the general population have been reported to be as high as 10%.

Natural History, Prognostic Factors, and Outcomes

Within the pediatric population, outcomes for individuals with articulation disorders range considerably depending on the severity of the disorder and the presence of other co-occurring conditions. For children who have been diagnosed strictly with articulation disorders, evidence suggests that with research-supported intervention, many speech sound disorders can be remediated.

Clinical Expression and Pathophysiology

Articulation disorders are typically characterized by the atypical development or production of a speech sound or group of speech sounds that result in a reduction in intelligibility. An articulation disorder is not the result of a cultural or dialectal difference. Disorders may include sound substitutions, distortions, additions, or omissions that impact an individual's ability to be understood in conversation. Speech sounds may be incorrectly produced due to incorrect placement of articulators, imprecise voicing, and/or structural deficits of the larynx, lips, tongue, palate, teeth, and/or jaw.

Evaluation and Differential Diagnosis

Articulation disorders are assessed using standardized tests as well as observational measures. Examples of formal assessments of articulation abilities include the *Arizona Articulation Proficiency Scale, Third Edition* (Fudala 2000); *Clinical Assessment of Articulation and Phonology, Second Edition* (Secord et al. 2002); and the *Goldman-Fristoe Test of Articulation, Third Edition* (Goldman and Fristoe 2000). In addition to standardized measures, samples of speech taken in single word and conversational contexts can be used to determine the type of speech sound errors that are present. Speech sampling procedures may include the assessment of a child's overall phonetic inventory (i.e., the number and variety of sounds he or she is able to produce), an analysis of syllable shapes and phonetic complexity and an analysis of error patterns. Stimulability measures

(i.e., gradually prompting and shaping sounds using cues and feedback from the clinician) often are used to determine if the individual is able to produce the sound given maximal support. In addition to these procedures, best practice suggests that a complete oral-motor examination of the individual be completed to determine if there are any structural or motor function deficits that are impeding correct speech sound production.

Additionally, articulation disorders typically are differentiated from phonological disorders. Careful assessment of a child's speech patterns may reveal not only a difficulty with speech sound production (i.e., a phonetic disorder) but also difficulties with the patterns of use of sounds within the language (see Phonological Disorders).

Treatment

There are a variety of treatment approaches that are used for the management of articulation disorders. Once an individual's specific areas of deficit have been determined, best practice would target the area of need that would most benefit the individual's intelligibility (i.e., how easily his or her speech is understood). Depending on the nature of the problem, treatment may involve individualized speech therapy in which the individual is taught how to produce the sound correctly through demonstration and repeated practice, learning specific techniques to shape how the speech mechanism is used. Additional techniques that are often used include training in recognizing correct and incorrect productions so that the individual can monitor how his or her speech sounds and practicing in contexts that increase in complexity.

See Also

- [Phonological Disorders](#)
- [Speech Delay](#)

References and Reading

American Speech-Language-Hearing Association, ASHA. (1993). Definitions of communication disorders and variations. *ASHA*, 35(Suppl. 10), 40–41.

- Bleile, K. (1995). *Manual of articulation and phonological disorders: Infancy through adulthood*. San Diego: Singular.
- Fudala, J. B. (2000). *Arizona articulation proficiency scale* (3rd rev.). Los Angeles: Western Psychological Services.
- Gierut, J. (2008). *Treatment efficacy summary: Phonological disorders in children*. Available from <http://www.asha.org/public/EfficacySummaries.htm>
- Goldman, R., & Fristoe, M. (2000). *The Goldman-Fristoe test of articulation* (2nd ed.). Circle Pines: American Guidance Service.
- Secord, W., Donohue, J., & Johnson, C. (2002). *Clinical assessment of articulation and phonology*. Greenville: Super Duper Publications.
- Secord, W., Boyce, S., Donahue, J., Fox, R., & Shine, R. (2007). *Eliciting sounds: Techniques and strategies for clinicians* (2nd ed.). Albany: Thomson Delmar Learning.
- Shriberg, L. D., Tomblin, J. B., & McSweeny, J. L. (1999). Prevalence of speech delay in 6-year-old children and comorbidity with language impairment. *Journal of Speech and Hearing Research*, 42, 1461–1481.

Articulatory Apraxia (or Dyspraxia)

- [Verbal Apraxia](#)

ARX

- [Aristaless-Related Homeobox Gene](#)

ASAS

- [Australian Scale for Asperger's Syndrome](#)

ASAS-R: Australian Scale for Asperger's Syndrome – Revised

- [Australian Scale for Asperger's Syndrome](#)

ASD

- [Pareidolic Faces](#)

ASD (Autism Spectrum Disorder)

- [BRIEF \(Behavior Rating Inventory of Executive Functions\)](#)

ASD=Autism Spectrum Disorder

- [ADHD Rating Scale](#)

ASDI

- [Asperger Syndrome Diagnostic Interview](#)

ASHA FACS

- [American Speech-Language-Hearing Association Functional Assessment of Communication Skills](#)

ASIEP-2

- [Autism Screening Instrument for Educational Planning \(ASIEP-2\)](#)

ASPEN

Lori S. Shery
 ASPEN (asperger/Autism SPectrum Education Network), Edison, NJ, USA

Membership as of October 2016

Membership is open to families and individuals whose lives are affected by autism spectrum disorders (those formerly known as Asperger

syndrome, PDD-NOS, and high-functioning autism) and nonverbal learning disabilities and the professionals who work with them.

Major Areas or Mission Statement

ASPEN is a national volunteer nonprofit organization with headquarters in New Jersey. It was established in 1997 by a group of parents of children diagnosed with Asperger syndrome to address the increasing need for information, support, and advocacy for individuals on the higher-functioning end of the autism spectrum and their families.

Mission

We will:

- Increase awareness and knowledge among the community and the professionals who diagnose, treat, educate, or provide services to individuals with Asperger syndrome (autism spectrum disorder) and related disorders
- Develop and maintain a strong network of families to support one another through the challenges of daily life
- Encourage the development and support of appropriate programs to foster individual independence and community integration. These programs include:
 - Effective social skills training
 - Social and recreational opportunities
 - Transition services
 - Employment possibilities
 - Transportation alternatives
 - Independent living options
- Advocate for individuals with Asperger syndrome (autism spectrum disorder) and related disorders in schools, the community, and the legislature
- Provide a forum to support and educate affected individuals, their families, and the professionals who work with them

Landmark Contributions

Development and distribution of ASD emergency ID cards, *Intricate Minds* peer awareness video,

college personnel training, law enforcement and emergency responder autism training, and series of employment weekend workshops provided at no charge to individuals on the spectrum and their parents.

Major Activities

Chapters meet monthly, alternating parent-support meetings with speaker presentations that are open to families and professionals. Chapters include those focused on family issues, those focused on adult issues, and those focused on fathers' issues. Additionally, social activity groups are run for pre-teens, teens, and adults. The organization publishes an online newsletter and resource directory, maintains a comprehensive website and lending libraries at each chapter location, holds workshops, and hosts semiannual national conferences featuring some of the most prominent names in the research community in addition to providing staff development and training.

Asperger Syndrome

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Synonyms

[Asperger's disorder](#); [Autism spectrum disorder \(ASD\)](#); [PDD](#)

Short Description or Definition

Asperger syndrome (AS) is a developmental disorder characterized by qualitative impairments in social interaction in association with repetitive and ritualistic patterns of behavior. By definition,

there is no clinically significant delay in (1) general cognitive development, as evidenced by IQ in the normal range (i.e., greater than 69), (2) adaptive behaviors, including self-help skills and curiosity about the environment, and (3) expressive language, broadly defined by the use of words by the age of 2 years and phrases by 3 years.

Asperger syndrome, or Asperger's disorder, came to prominence in the 1980s, following the publication of Wing's seminal paper describing 34 young adults with impairments of social interaction and aspects of everyday communication and associated adherence to routine and circumscribed patterns of interest (Wing 1981). The children and young adults described in her paper all exhibited difficulties forming and maintaining relationships with others, with some presenting as aloof and passive, while others actively tried to engage socially, but their communicative exchanges were odd: Unfortunately, therefore, despite their social motivation, their clumsy posture, poor eye contact, and poor vocal intonation denied them the friendships they desired. The majority of the cases Wing described pursued circumscribed, solitary interests with enthusiasm with the result that many acquired a significant knowledgebase on particular subjects.

Wing used the term "Asperger syndrome" to draw attention to the paper first published in 1944 by Hans Asperger, in which four boys with sociocommunicative impairments and repetitive patterns of behavior, including the pursuit of circumscribed interests, were described (Asperger 1944, translated in Frith 1991). She also drew comparisons with the syndrome first described by Kanner in 1943 (Kanner 1943) and, in doing so, brought Asperger and Kanner's syndromes together for the first time and in what has subsequently become known as the "autism spectrum disorders" (ASDs), a tridimensional group of disorders characterized by impairments of social interaction communication and repetitive and ritualistic patterns of behavior.

Since the publication of Wing's paper, there has been considerable interest in Asperger syndrome, as evidenced by the large body of scientific literature devoted to understanding its epidemiology, etiology, and management. There

has also been significant interest in its conceptual relationship to the other "autism spectrum disorders," with much of this research failing to find any evidence of a distinction, thereby supporting the spectral representation (Volkmar and Klin 2005). Indeed, as discussed subsequently, so strong is the evidence that the validity of maintaining Asperger's as a distinct disorder vis-à-vis autistic disorder has been brought into question, and it is quite possible that the term "Asperger's" will not find a place in the subsequent revisions of the World Health Organization's (WHO) International Classification of Diseases eleventh revision (ICD-11) or the American Psychiatric Association's (APA) Diagnostic and Statistical Manual fifth edition (DSM). Nonetheless, as will become apparent, there are a number of reasons for its retention, and even if removed, it is a term that will continue to be used clinically, and therefore it is important for clinicians and health-care workers to have an understanding of its characteristics.

Categorization

In both the American Psychiatric Association's (APA) Diagnostic and Statistical Manual (DSM-IV) and the World Health Organization's (WHO) International Classification of Diseases (ICD-10), Asperger syndrome is categorized along with autistic disorder, Rett's syndrome, childhood disintegrative disorder, and pervasive developmental disorder not otherwise specified (PDDNOS). Much has been written about the relationship between Asperger syndrome and the other PDDs. It is certainly true that the syndromes first described by Kanner and Asperger share many features, and therefore in clinical terms, it is understandable that they have been brought together under the same spectral umbrella. However, what is also apparent is that in bringing these conditions together, many of the features described by Asperger have been subsequently de-emphasized.

For example, Asperger focused on the abnormal patterns of communication that characterized the boys he described. These included abnormalities of social pragmatics, i.e., the everyday

aspects of communication, despite normal formal language skills (such as semantics and syntax). In particular, posture, facial expression, gaze, and other nonverbal communicative gestures were described as notably peculiar. In addition, Asperger commented that language itself, i.e., verbal communication, was of diagnostic importance in view of its peculiarities, which varied from case to case. This included abnormalities with volume of speech (too loud or too quiet), intonation of speech (e.g., talking in a monotone or talking in an overmodulated way resembling exaggerated verse speaking), and in choice of words for communication, which may be formal, pedantic, or otherwise quirky. The importance of the pragmatic aspects of communication is that they do offer some differentiation from the patterns of communication seen in other ASDs, but unfortunately, they are not included in either the DSM-IV or ICD-10.

In addition to this “feature de-emphasis,” the other aspect of our current classification systems that is potentially problematic for the concept of AS is the rule of diagnostic hierarchy. That is, the diagnosis of “autistic disorder” takes precedence over Asperger syndrome, such that if an individual meets the diagnostic criteria for both (and this scenario is not uncommon), then the autistic disorder diagnosis takes priority and the individual is assigned that diagnosis. The result of this is that individuals who may be deemed clinically to have AS are “sucked” into the autistic disorder category.

Although hierarchical diagnosis and symptom de-emphasis may be useful if the spectral conceptualization is correct, as they allow the syndromes of Kanner and Asperger to be more closely aligned, it may be problematic if there is a true difference between the disorders. While it is fair to say that most of the research examining the external validity of differentiating between the two disorders has failed to find any strong evidence for a distinction (discussed in Klin et al. 2005a), much of this research has relied on either the ICD-10 or DSM-IV conceptualizations, and therefore the results come as no great surprise as they are confounded by tautology. In particular, if the two disorders are defined according to the same set of criteria, and if there is a hierarchical

system of diagnosis in place, then the two syndromes may only differ in name.

The only way to overcome this tautological confound will be to re-examine for external validity for groups described according to more robust criteria that offer some possibility of symptom separation (as, it can be argued, would be the case if Kanner’s and Asperger’s original criteria are applied) and if the hierarchical system is removed. One study has explored the external validity of AS in a more objective manner, by comparing features according to three different diagnostic systems, including (1) current DSM-IV criteria, (2) division of the spectrum according to onset of language, and (3) criteria more closely aligned with Asperger’s case studies, which they termed the “new system” (Klin et al. 2005a). This study found that, on balance, their “new system” differentiated greatest between autism, PDDNOS, and Asperger syndrome. Interestingly, while it has also been suggested that IQ profiles differentiate Asperger’s (verbal performance discrepancy favoring the former) from autism (verbal performance discrepancy favoring the latter), no such differences were found for any of the systems used.

Epidemiology

The prevalence of a disorder may vary if researchers use different syndrome defining criteria, and this issue is of crucial significance for AS. For example, before AS was described in the most recent versions of the ICD and DSM, clinicians, eager to diagnose, developed their own criteria. These included those of Ehlers and Gillberg, who subsequently carried out a robust epidemiological study of the prevalence of Asperger syndrome using these criteria (Ehlers and Gillberg 1993). Their criteria were certainly in keeping with characteristics described by Asperger and included the communication items described above, although were fundamentally limited by being very broadly defined. Their study found a point prevalence of 28.5/10,000 (95% CI 0.6–56.5/10,000).

Fombonne (2009), in his overview of ASD epidemiology, took into consideration six more recent

surveys of autism prevalence and found that the rates of AS were consistently lower than autism, with an average ratio of 5:1 for rates of autism versus AS. This translates into a median prevalence estimate of 2.6/10,000 for AS alongside 13/10,000 for autism, and 60/10,000 when more broadly defined cases are included (Fombonne 2009).

In terms of sex ratios, males are more often affected than females, with ratios varying according to diagnostic subtype and level of intellectual ability. In particular, among lower functioning groups, the sex ratio approaches unity, whereas among those who are higher functioning, males are affected more frequently than females. The exact ratio is unclear, with variation between 4:1 and 9:1 being demonstrated between different studies (*ibid.*).

Natural History, Prognostic Factors, Outcomes

Comorbidities

Along with the other ASDs, there are high rates of additional neuropsychiatric disorders among children and adults with AS. Conditions such as epilepsy, tic disorders, and disorders of attention and motor control are known to occur with increased frequency in the ASDs, although no robust data are available for AS. Certainly it is true that for seizure disorders, the highest rates (approaching 20%) are seen among those who are lower functioning, and this is probably true of the attentional and motor disorders too.

In terms of mental health problems, mood and anxiety disorders are particularly common, although due to an absence of epidemiological data, it is not possible to give a true prevalence figure (Woodbury-Smith and Volkmar 2009). In Wing's case series (1981), 8 of the 36 individuals described had "probable depression," and in Tantam's study of 85 individuals with primary social relationship difficulties (1988), many of whom fulfilled the criteria for AS, 11% had clinical depression, this being the most common mental health problem reported (Tantam 1988). It is certainly true that prevalence estimates for comorbid depression vary widely, but taken together,

they suggest that clinical depression is a significant problem in this population. Similarly, anxiety disorders are also commonly reported among individuals with Asperger syndrome. Once again, however, no truly epidemiological study has been carried out, and figures are based on administrative samples. The prevalence of psychotic disorders among AS is less clear, with schizophrenia occurring in three of Tantam's cases (3.5%) and approximately 4% of the "loners" described by Wolff (2000), but none of a clinic-based sample (Ghaziuddin et al. 1998).

Outcome

There is now evidence that as many as 20% will no longer meet the criteria for an ASD as they transition through their adolescent and early adult years, and many others show a significant improvement in their symptoms (Seltzer et al. 2003). Unfortunately, however, studies investigating outcome more generally, including parameters of social inclusion and quality of life such as employment, independent living, and relationships, suggest that the outcome for a significant number is poor (Barnard et al. 2001). This is particularly true of those who are higher functioning, who have the added problem of being excluded from support services because of their normal intellectual function. It is crucial, therefore, that services are developed to meet the needs of this population that will facilitate their social inclusion and thereby improve their quality of life.

It is also apparent that the higher functioning population with ASDs may be at risk of unlawful behavior and contact with the criminal justice system, as discussed elsewhere in this volume. While this may only be true for a small minority, there is some evidence that the core autism phenotype mediates this relationship. In particular, impairment of emotional processing and the pursuit of circumscribed interests may both play a role.

Clinical Expression and Pathophysiology

Clinical Expression

All descriptions of Asperger's have highlighted its core impairment in relating to others. Fairly

consistent has also been the descriptions of communication impairments. Finally, most descriptions highlight the restricted pattern of behaviors, usually taking the form of circumscribed patterns of interest, often solitary, and generally pursued in preference to other activities.

The social impairment is, arguably, the *sine qua non* of Asperger syndrome and all other ASDs. It is characterized by difficulty relating to others. As a result, children with AS are often rejected by, and thereby isolated from, their peers, and as adults may live a fairly solitary existence. In describing Asperger syndrome, Wing highlighted two forms of social impairment, namely, the “aloof” and “active but odd” types, binary categories that have some clinical validation. It is certainly true that most individuals with AS probably fall into the “active but odd type,” with only a minority failing to form social relationships because of aloofness and lack of interest. Instead, many go out of their way to try and form friends, but their approach may be clumsy, with limited use of eye contact, social smiling, or socially recognized greetings. They may dress peculiarly or at least in an unfashionable way. They sometimes fail to appreciate the impact of poor self care on acceptance by others and may stand too close to, or far away from, their interlocutor.

Their communicative exchanges are often formal, particularly noticeable among children who resemble adults in their use of words and formalisms. They may talk in a monotone or over-intonated voice, failing to appreciate the point of “social chit chat,” and may instead chose to present an in-depth monologue about a topic of interest, failing to appreciate whether their listener is interested or bored or, indeed, understands the topic at all. Unfortunately, many people with AS are not interested in the same things as their peers. Among children, for example, an interest in sports, music, and/or fashion is more accepted than cosmology or license plate collecting.

The circumscribed interests are a prominent feature of the disorder, and it is important that they are differentiated from normal patterns of hobbies that many people engage in. Differentiation is unfortunately somewhat arbitrary and

based on interpretation of their intensity and/or focus. To all intents and purpose, an interest is intense to a significant degree if it impinges on other day-to-day activities (such as eating, sleeping, paying bills, and so forth), and is odd in focus if it is not clearly functional (e.g., collecting tin cans). Importantly, it is not unusual for interests to change over time.

Etiology

Much of the literature concerned with the etiology of the ASDs has investigated the spectrum in broad terms, on the assumption that all ASDs share the same causal mechanisms. As indicated previously, there has been research examining the differences between AS and other autistic disorders from a biological (primarily neuropsychological) perspective, but much of this research has failed to differentiate between the disorders. In the discussion that follows, this broader etiological literature will be summarized, but where available, the studies more specifically pertaining to AS will be highlighted.

There is now little doubt that genetic mechanisms play an important role in the etiology of ASDs. Although these same genetic risk factors may be relevant specifically for AS (Rutter 2005), there is a paucity of linkage and association studies specifically examining probands with AS. One study has investigated genetic linkage in AS (Ylisaukko-oja et al. 2004) and observed linkage at 1q21–22, 3p14–24, and 13q31–33 in 17 multiplex families with 119 affected probands, 72 of whom fulfilled the ICD-10 criteria for AS. Interestingly, the loci on chromosomes 1 and 3 overlap with previously identified autism susceptibility loci, and on 1 and 13, with schizophrenia susceptibility loci.

Other research on etiology has focused on looking at neuropsychological mediators of ASDs. This represents a vast literature, although the impairments identified fall into the domains of (1) theory of mind, (2) executive dysfunction, and (3) central coherence (discussed in Klin et al. 2005b). Research using MRI has also identified both structural and functional abnormalities in regions including the fusiform face area, amygdala,

and regions of the dorsolateral and orbitofrontal prefrontal cortices (Schultz et al. 2000).

Unifying all this research into a model of the pathogenesis of autism is difficult. Certainly, the different genes identified all seem to converge on the synapse, and the neuropsychological and neuroimaging research all indicates neural pathways involved in the processing of social and emotional information and mental flexibility. A different perspective aligns the impairments seen in AS with an extreme form of the male brain, with some support for this model existing in the form of the in utero hormonal environment (Baron-Cohen 2005).

Evaluation and Differential Diagnosis

Evaluation

The diagnosis of Asperger syndrome is based upon a detailed clinical assessment, which includes history from an informant who knew the person during their formative years and a direct observation of the person themselves. The autism diagnostic interview (ADI-R) can be used to structure the history, and the autism diagnostic observation schedule (ADOS) can be used to structure the direct observation component. While neither of these contain algorithms specific to the diagnosis of AS, extrapolating from the algorithms that do exist is relatively straightforward. Importantly, these instruments are intended to approximate rather than replace expert clinical opinion. Several other diagnostic instruments have also been developed, including the Australian Scale for Asperger Syndrome (ASAS) and the Gilliam Autism Rating Scale (GARS), and screening instruments specifically for AS are also available (e.g., the autism spectrum quotient (AQ)). Most of these have data on validity and reliability and are commercially available (references available in Woodbury-Smith and Volkmar 2009).

Differential Diagnosis

There are several other disorders that exist at the boundary of the ASDs and which may be

confused with them, particularly among those with ASDs who are higher functioning. This includes schizoid and schizotypal personality disorders, social anxiety disorder, and obsessive-compulsive disorder. The two personality disorders may represent the most diagnostic confusion because of the overlap in clinical symptomatology. Current diagnostic wisdom would argue that the personality disorders develop in late adolescence and early adulthood, which therefore provides a fundamental distinction from ASDs because the latter are of early developmental onset. This is perhaps less helpful than it first appears, however, because in reality, PDs are often symptomatic in earlier adolescence, and the higher functioning ASDs are often characterized by relatively subtle abnormalities during the early years, such that diagnosis is often delayed until the adolescent period and sometimes even adulthood. In all likelihood, the two disorders may represent slightly different manifestations of the same underlying pathological process. Certainly the relationship between Asperger syndrome and the “schizophrenia spectrum” requires further investigation, particularly in light of the genetic evidence discussed above.

Social anxiety disorder is differentiated by the fairly circumscribed nature of the situations that provoke symptoms of anxiety (such as public speaking) and the onset usually in adolescence and beyond. Obsessive-compulsive disorder is differentiated on the basis of the egodystonicity that characterizes the thinking and ritualistic behaviors and the absence of major qualitative social impairments.

Treatment

There is much overlap in the interventions used for AS and other ASDs. In particular, these include those strategies aimed at the core features of the disorder and those aimed at managing comorbidities (Woodbury-Smith and Volkmar 2009). A number of behavioral and educational interventions have been developed aimed at engendering sociocommunicative skills and adaptive functioning and overcoming some of the weaknesses in problem solving and judgment

that occur as a result of executive dysfunction. The evidence base for these interventions is limited and often based on single-case studies or small-case series. Nevertheless, all approaches share a core set of “ingredients,” which include making the treatment individualized, using a “parts to whole” approach, augmented with visual strategies where appropriate, and using explicit, rote verbal learning. Executive dysfunction can be overcome using scheduling, scripts, or lists, and adaptive skills can be taught through practice, rehearsal, and reinforcement. It is also important to recognize that a person with AS can learn though social exposure, either in the form of “buddying” or “circle of friends,” social groups or explicit social skills training. The comorbid mental health problems may also require specific management, through either psychopharmacology or different psychotherapies (as discussed elsewhere in this volume) or a combination of the two.

See Also

- ▶ [Autistic Disorder](#)
- ▶ [Noradrenergic System](#)

References and Reading

- Asperger, H. (1944). Die “autistischen Psychopathen” im Kindersalter. Archive für psychiatrie und Nervenkrankheiten. (U. Frith, Trans.)(Ed.) (1991). *Autism and Asperger's syndrome* (Vol. 117, pp. 76–136). Cambridge: Cambridge University Press.
- Barnard, J., Harvey, V., Prior, A., & Potter, D. (2001). *Ignored or ineligible? The reality for adults with autistic spectrum disorders*. London: National Autistic Society.
- Baron-Cohen, S. (2005). Testing the extreme male brain (EMB) theory of autism: Let the data speak for themselves. *Cognitive Neuropsychiatry*, 10(1), 77–81.
- Ehlers, S., & Gillberg, C. (1993). The epidemiology of Asperger syndrome. A total population study. *Journal of Child Psychology and Psychiatry*, 34(8), 1327–1350.
- Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. *Pediatric Research*, 65(6), 591–598.
- Ghaziuddin, M., Weidmer-Mikhail, E., & Ghaziuddin, N. (1998). Comorbidity of Asperger syndrome: A preliminary report. *Journal of Intellectual Disability Research*, 42(4), 279–283.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Klin, A., McPartland, J., & Volkmar, F. R. (2005a). Asperger syndrome. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 88–125). Hoboken: Wiley.
- Klin, A., Pauls, D., Schultz, R., & Volkmar, F. (2005b). Three diagnostic approaches to Asperger syndrome: Implications for research. *Journal of Autism and Developmental Disorders*, 35(2), 221–234.
- Rutter, M. (2005). Genetic influences and autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 425–452). Hoboken: Wiley.
- Schultz, R. T., Romanski, L. M., & Tsatsanis, K. D. (2000). Neurofunctional models of autistic disorder and Asperger syndrome: Clues from neuroimaging. In A. Klin & F. R. Volkmar (Eds.), *Asperger syndrome* (pp. 172–209). New York: The Guilford Press.
- Seltzer, M. M., Krauss, M. W., Shattuck, P. T., Orsmond, G., Swe, A., & Lord, C. (2003). The symptoms of autism spectrum disorders in adolescence and adulthood. *Journal of Autism and Developmental Disorders*, 33(6), 565–581.
- Tantam, D. (1988). Lifelong eccentricity and social isolation. I. Psychiatric, social, and forensic aspects. *The British Journal of Psychiatry*, 153, 777–782.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 5–41). Hoboken: Wiley.
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11(1), 115–129.
- Wolff, S. (2000). Schizoid personality in childhood and Asperger syndrome. In A. Klin & F. R. Volkmar (Eds.), *Asperger syndrome* (pp. 278–305). New York: The Guilford Press.
- Woodbury-Smith, M. R., & Volkmar, F. R. (2009). Asperger syndrome. *European Child & Adolescent Psychiatry*, 18(1), 2–11.
- Ylisaukko-oja, T., Wendt, T. V., Kempas, E., Sarenius, S., Varilo, T., von Wendt, L., et al. (2004). Genome-wide scan for loci of Asperger syndrome. *Molecular Psychiatry*, 9(2), 161–168.

Asperger Syndrome (AS)

- ▶ [Client Emotional Processing Scale for Autism Spectrum](#)

Asperger Syndrome Diagnostic Interview

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Synonyms

ASDI; High-functioning autism diagnostic interview

Description

The Asperger Syndrome Diagnostic Interview (ASDI) is a diagnostic instrument developed specifically for the assessment of Asperger syndrome (AS) in children and adults. This brief structured interview consists of 20 items divided into six broader areas of behavior that may be indicative of AS. ASDI is an investigator-based interview in which each item is an open-ended question corresponding to a specific part of autistic symptomatology. It is therefore essential that the examiner acquire a sufficient level of clinical material first so that correct assessment of presented behavior can be made. The evaluation is carried out by scoring each item on the interview as either 0 (does not apply) or 1 (applies to some degree or very much). Total scores are obtained within six different areas. Each area may require a different number of items met to fulfill the diagnostic criteria. ASDI is based on Gillberg and Gillberg's (1989) definition of AS and is congruent with Hans Asperger's original clinical account. Parents, siblings, primary caregivers, or any person well acquainted with the individual's developmental history can also be reporters on the ASDI. Although the ASDI addresses major domains that tend to be problematic in individuals with AS, it can only be considered as one of several steps in the diagnostic process and should not be presented as the only source of inquiry.

- *Area 1 (4 Items): Severe Impairments in Reciprocal Social Interaction (Extreme Egocentricity)*

Items in this area map onto the nature of peer interaction, interest in close relationships, ability to follow social rules, appreciate social communication cues, and the presence of socially and emotionally appropriate behaviors.

(Scoring: Two or more scores of 1 = criterion met)

- *Area 2 (3 Items): All-Absorbing Narrow Interest Pattern(s)*

This area addresses the manifestation of restricted interests that could interfere with development in other domains of functioning.

(Scoring: One or more scores of 1 = criterion met)

- *Area 3 (2 Items): Imposition of Routines, Rituals, and Interests*

Following on the restricted interests, the third area deals with the imposition of routines on one self or others to a degree that impedes functioning.

(Scoring: One or more scores of 1 = criterion met)

- *Area 4 (5 Items): Speech and Language Peculiarities*

Language development is examined in Area 4. Delays in language development are assessed as well as abnormalities in the expressive components of language such as odd articulation, verbosity, and characteristic diction. Appropriate language comprehension, including the understanding of literal meaning and metaphors, is also evaluated.

(Scoring: Three or more scores of 1 = criterion met)

- *Area 5 (5 Items): Nonverbal Communication Problems*

Individuals with AS may display a range of marked nonverbal behaviors such as clumsy gestures, inadequate body language, limited repertoire of facial expressions, or disrupted gaze patterns. Such nonverbal communication impairments are the focus of the Area 5.

(Scoring: One or more scores of 1 = criterion met)

- *Area 6 (1 Item): Motor Clumsiness*

The final domain examines difficulties related to motor coordination in everyday functioning and also inquires about the history of gross and fine motor skill development.

(Scoring: Score of 1 = criterion met)

Historical Background

In 1981, Lorna Wing introduced the diagnosis of AS to English speakers in a case report of 34 children and adults whose clinical observation closely matched Hans Asperger's original case studies but did not overlap with Kanner's criteria for autism at that time. This report initiated further research, resulting in a publication of the first diagnostic criteria for AS (Gillberg and Gillberg 1989), which was revised in 1991. By that time, AS was already a widely recognized clinical diagnosis with several existing screening tools but suffered a paucity of reliable diagnostic instruments that could inform an in-depth assessment. In addition, some authors argued that gold standard measures such as the Autism Diagnostic Interview-Revised (ADIR) and Autism Diagnostic Observation Schedule (ADOS) were not sensitive enough to pick up individuals with these milder yet very distinctive difficulties (Klin et al. 2000).

In 2001, Gillberg and colleagues published the ASDI based on their own diagnostic criteria for Asperger disorder (Gillberg and Gillberg 1989; Gillberg 1991). ASDI was a result of long-term work with a large number of individuals with AS and high-functioning autism with symptomatology difficult to notice through ordinary autism screening tools. Following on Hans Asperger's clinical observations, the diagnostic criteria enlisted in ASDI were different from those in DSM-IV and ICD-10. One of the important differences relates to language development. While DSM-IV requires no clinically significant delay in early language development in individuals with AS, Gillberg, Gillberg, Rastam, and Wentz (2001) argued that such a profile was not seen in clinical practice. Therefore, Gillberg et al. (2001) acknowledged early language impairments as

primary characteristics of AS. In addition, other authors raised concerns about the validity of DSM-IV criteria for AS by documenting that Hans Asperger's original clinical cases would not meet those criteria and would instead fall into the autistic disorder category (Leekam et al. 2000; Miller and Ozonoff 1997). These issues continue to be debated as the ASDI is still used in clinical practice. It was not, however, designed in conjunction with DSM-IV and ICD-10.

Psychometric Data

Participants

Reliability and validity of the ASDI have not been studied extensively. Gillberg et al. (2001) reported preliminary findings on the interrater/intrater reliability in a sample of 24 individuals (aged 6–55) where 17 had a clinical diagnosis and 7 were healthy controls. The diagnosed subjects consisted of 12 cases with AS, 2 cases with atypical autism, 2 cases with obsessive-compulsive disorder, and 1 person with multiple personality disorder. The individuals with AS met some of the criteria for the disorder according to the DSM-IV and full criteria for AS as defined by Szatmari, Bremner and Nagy (1989) and Gillberg and Gillberg (1989).

Reliability

Interrater Reliability

In order to determine the degree of agreement among raters using the ASDI, first-degree relatives of 20 individuals were interviewed by two neuropsychiatrists. The raters were blinded to the diagnostic status of the participants. Both raters were present during the interviews, but only one of them performed the interview while the other one was observing and coding independently. Since the ASDI contains 20 items, each rater had to make 200 ratings. In 383 out of 400 ratings, the two raters reached complete agreement (20 paired ratings), which yielded a kappa statistic of .91 (high level of agreement). The raters had a complete agreement across all items for 10 of 20 individuals, an almost complete agreement (19 items)

in six individuals, and in the remaining four subjects, they agreed on 17 and 18 items. Such results are promising and provide support for a good interrater reliability, although it needs to be acknowledged that the authors used a small sample and only two raters. Further investigation is needed to replicate these findings.

Intrater Reliability

Intrater reliability refers to the degree of consistency of a measure over time. In Gillberg et al. (2001) study, the intrater reliability was determined by a repeated evaluation using ASDI at a 10- to 15-month period after the first assessment. Twenty-four individuals participated in this study, and the examiners were still blinded to their diagnostic status. There was an agreement on 465 out of 480 items corresponding to a kappa of .92. In 16 subjects, the examiner scored accordingly with the previous performance (20 items out of 20), in five subjects, there was a disagreement on one item, for two subjects on two items, and finally, in one case, the differences in rating included seven different items. Based on the results from this sample, the ASDI had very good intrater reliability although the same limitations applied as mentioned in the case of interrater reliability.

Validity

In order to evaluate the construct validity of ASDI, the number of correctly diagnosed individuals has been computed. The ASDI correctly detected all of the subjects with a diagnosis of AS or atypical autism as they fulfilled from five or six (out of six) diagnostic areas. Of the remaining sample, one individual also met criteria for autism despite having a different diagnosis – multiple personality disorder. Based on this sample, ASDI was able to discriminate with high accuracy between individuals with AS and other clinical diagnosis. However, the sample did not include individuals with high-functioning autism, and thus, there is no evidence to conclude that this measure could differentiate between those two categories.

Although the psychometric characteristics of ASDI have shown that this measure has good intrater reliability, interrater reliability, and validity, all of these reported findings are

preliminary and have not been replicated with large samples nor has this diagnostic interview been used in conjunction with other AS measures.

Clinical Uses

The ASDI has been used in AS assessment research, although not extensively. Cederlund, Hagberg, and Gillberg (2010) used the ASDI in their follow-up study in a sample of 100 males with AS who were diagnosed in childhood. The aim of the study was to assess the awareness that individuals with AS had of their emotional and cognitive difficulties and to determine to what extent their view was congruent with their parents' opinion. Seven items of ASDI were administered to both the individuals and their parents. The results showed significant differences between the adults and their parents' scores in three out of these seven items (social ability, social cues, and narrow interests) with parents scoring higher than the individuals with AS. The authors emphasized that these items possibly reflected the core deficits of the social impairments seen in AS and therefore may have been the most difficult ones to be assessed accurately by individuals with AS. Such findings also underscore the extent to which diagnostic interviews rely on the insight and honesty of the interviewed person; yet the population with AS may not be fully aware of their emotional impairments or camouflage them by active learning of socially appropriate scripts. Naturally, the assessment of AS demands a more complex approach. An individual's medical, developmental, and family history needs to be acquired in addition to direct observations of social behavior, psychological evaluation of cognitive functioning, coping mechanisms, and communication skills (Klin et al. 2000). Although ASDI can be used for preliminary diagnostic decisions where AS or high-functioning autism symptoms are suspected, a multidisciplinary assessment guided by an experienced clinical judgment will have the best results for informing the subsequent intervention and deciding whether the diagnostic category matches the clinical presentation and the needs of the individual.

See Also

- ▶ Asperger Syndrome
- ▶ Asperger Syndrome Epidemiology
- ▶ Asperger Syndrome Follow-Up Studies
- ▶ Asperger, Hans
- ▶ Autism Diagnostic Interview-Revised
- ▶ Autism Diagnostic Observation Schedule
- ▶ Diagnostic Instruments in Autistic Spectrum Disorders
- ▶ Diagnostic Interviews
- ▶ Diagnostic Process
- ▶ DISCO

References and Reading

Books

- Attwood, T. (2007). *The complete guide to Asperger's syndrome* (1st ed.). London: Jessica Kingsley.
- Gillberg, C. (1991). Clinical and neurobiological aspects of Asperger syndrome in six family studies. In U. Frith (Ed.), *Autism and asperger syndrome*. Cambridge: Cambridge University Press.
- Klin, A., Volkmar, F., & Sparrow, S. S. (Eds.). (2000). *Asperger syndrome*. New York/London: The Guilford Press.
- Ozonoff, S., Dawson, G., & McPartland, J. (2002). *A parent's guide to Asperger syndrome and high-functioning autism: How to meet the challenges and help your child thrive*. New York: The Guilford Press.
- Szatmari, P. (2005). *A mind apart: Understanding children with autism and Asperger syndrome*. New York: The Guilford Press.

Journal Articles

- Cederlund, M., Hagberg, B., & Gillberg, C. (2010). Asperger syndrome in adolescent and young adult males. Interview, self- and parent assessment of social, emotional, and cognitive problems. *Research in Developmental Disabilities*, 31(2), 287–298.
- Gillberg, C., & Gillberg, C. (1989). Asperger syndrome – Some epidemiological considerations: A research note. *Journal of Child Psychology and Psychiatry*, 30(4), 631–638.
- Gillberg, C., Gillberg, C., Rastam, M., & Wentz, E. (2001). The Asperger syndrome (and high-functioning autism) diagnostic interview (ASDI): A preliminary study of a new structured clinical interview. *Autism*, 5(1), 57–66.
- Leekam, S., Libby, S., Wing, L., Gould, J., & Gillberg, C. (2000). Comparison of ICD-10 and Gillberg's criteria for Asperger syndrome. *Autism*, 4(1), 11–28.
- Miller, J. N., & Ozonoff, S. (1997). Did Asperger's cases have Asperger disorder? A research note. *Journal of*

Child Psychology and Psychiatry, and Allied Disciplines, 38(2), 247–251.

Szatmari, P., Bremner, R., & Nagy, J. (1989). Asperger's syndrome: A review of clinical features. *Canadian Journal of Psychiatry*, 34(6), 554–560.

Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11(1), 115–129.

Web Pages

OASIS – Online Asperger Syndrome Information and Support. Retrieved 15 Feb 2011, from <http://www.aspergersyndrome.org>.

Asperger Syndrome Epidemiology

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Definition

The epidemiology of Asperger syndrome refers to what is known about its prevalence and course.

Historical Background

On considering the epidemiology of a disorder, consistency as regards its definition is particularly pertinent, as the prevalence of a disorder may vary if researchers use different syndrome-defining criteria. This is relevant when discussing the epidemiology of Asperger syndrome (AS) for several reasons. First, even before AS was included in the most recent versions of the International Classification of Diseases (ICD, 10th edition, World Health Organization [WHO], 1993) and Diagnostic and Statistical Manual of Mental Disorders (DSM, Volume IV, American Psychiatric Association [APA], 1994), clinicians, eager to diagnose this disorder, developed their own sets of criteria (Klin et al. 2005). Notably, the first two epidemiological surveys of Asperger syndrome, discussed later, both used such “clinician-driven” criteria.

The relatively high-prevalence figure they calculated might reflect the broad nature of the criteria they used, particularly when compared with the generally lower estimates of prevalence for AS subsequently obtained.

Even since its inclusion in the ICD-10 and DSM-IV, with their criteria for AS almost identical, the label Asperger syndrome has often been applied loosely in diagnostic terms, in some cases to mean “mild autism” or “normal IQ autism” or even in everyday parlance synonymously with “loners” or “nerds.” Moreover, even when applying the DSM-IV or ICD-10 criteria, problems with interpretation due to ambiguity of diagnostic items are likely. For example, at what point does an interest become a “circumscribed interest” either in terms of intensity or focus? The subjective threshold of diagnosing clinicians may inflate or reduce prevalence as a result of this ambiguity. And finally, both diagnostic systems include a hierarchy rule, whereby a diagnosis of autism takes precedence over AS, such that if a person meets criteria for both, an autism diagnosis is given. It has been argued that this last point may lead to a situation where an Asperger diagnosis becomes an impossibility, as cases are “sucked into” the autism category. This was formally investigated in a study that revisited the DSM field trial autism-related data. These data included 48 individuals with a clinical diagnosis of AS, of whom 11 (23%) were reassigned a diagnosis of autistic disorder as a result of this hierarchy rule (Woodbury-Smith et al. 2005). As such, the prevalence of the disorder is very likely to vary according to whether a clinician applies this rule.

Current Knowledge

Bearing in mind these caveats, it is perhaps no great surprise that the range of prevalence figures quoted for AS vary widely. For example, the study of Ehlers and Gillberg (1993), using their own diagnostic criteria, found a point prevalence of 36/10,000 among school-aged children (7–16 years) in a school catchment-defined area of central Sweden. This figure rose to 71/10,000 if suspected cases were also included. This same

study also identified a male to female ratio of 4:1 among the definite cases. Ehlers and Gillberg also calculated prevalence using the ICD-10 criteria, which had just been published at the time: a slightly lower figure of 29/10,000 was calculated for definite cases using these criteria. One other study from Sweden (Kadesjo et al. 1999) diagnosed cases according to the ICD-10 criteria and found rates of 48/10,000, with a male to female ratio of 4:1. Due to the fact that samples in both studies originated from small populations (1,401 for Ehlers and Gillberg and 826 for Kadesjo et al.), this prevalence estimate amounted to only a small handful of cases. The Kadesjo et al. study also examined the population prevalence of other autism spectrum disorders, with autism diagnosed according to DSM-III-R and “algorithm ICD-10” criteria. The prevalence of autistic disorder was 60/10,000, suggesting Asperger syndrome is less common than its counterpart.

While the more recent figures also support higher rates of autism than Asperger, the exact prevalence of the latter is somewhat lower than these earlier Swedish studies. The results of more recent prevalence studies (see Fombonne 2009) that include figures for AS are summarized in Table 1. In each of the studies quoted, fairly robust epidemiological methods have been employed and screening and diagnosis are clearly described. On the whole, all these studies seem to agree on a number of points. First, autism is more common than AS. Generally, the ratio was 2:1, although Baird et al. (2000) found a much wider split of 9:1. Secondly, all but one identified prevalence figures between 3 and 10 per 10,000. The one study that found higher figures (Latif and Williams 2007) used Gillberg’s criteria to identify cases which might explain why their figures were closer to those quoted in the earlier studies described above. All the other studies used either ICD-10 and/or DSM-IV.

The lowest prevalence was 3 per 10,000, quoted by Baird et al. (2000). This is the only study that specifically indicated that it ignored the hierarchy rule. It seems reasonable to propose, therefore, that the other four studies quoted in Table 1, which all used the same diagnostic criteria (i.e., DSM-IV or ICD-10), similarly did

Asperger Syndrome Epidemiology, Table 1 Summary of recent epidemiological surveys with Asperger syndrome (AS) data

Country	Size of population	Age range (years)	Autism prevalence (per 10,000)	AS prevalence (per 10,000)	Sex ratio (M:F)	Autism: AS ratio	References
Stafford, UK	15,500	2.5–6.5	16.8 (<i>N</i> = 26)	8.4 (<i>N</i> = 13)	5.5:1	2:1	Chakrabarti and Fombonne (2001)
Stafford, UK	10,903	4.0–6.0	22 (<i>N</i> = 24)	11 (<i>N</i> = 12)	100% M	2:1	Chakrabarti and Fombonne (2005)
South Wales, UK	39,220	Birth–17.0	61.2 (<i>N</i> = 267)	35.4 (<i>N</i> = 154)	6.7–10.5:1	1.7:1	Latif and Williams (2007)
Montreal, Canada	27,749	5–17	21.6 (<i>N</i> = 60)	10.1 (<i>N</i> = 28)	2:1	2:1	Fombonne et al. (2006)
London, UK	16,235	7	27.7 (<i>N</i> = 45)	3.1 (<i>N</i> = 5)	100% M	9:1	Baird et al. (2000)

not apply this rule considering their higher quoted prevalence figures. However, why there should be differences in prevalence when using the same criteria (i.e., ranging from 3/10,000 to 11/10,000) and in urban-based areas in the same country is not clear and may simply be a reflection of methodological differences rather than true prevalence differences.

The studies were fairly consistent in terms of gender split, with the majority of those being identified with AS being males, a figure higher than the 4:1 suggested by Ehlers and Gillberg and Kadesjo and colleagues. This may reflect the fact that diagnosis is more difficult among females or that the phenotype is expressed differently. Of course, the gender difference may be related in some way to the underlying biological mechanisms (such as genes on the X chromosome, or the “extreme male brain” phenotype).

Fombonne (2009), in his overview of ASD epidemiology, took into consideration the six most recent surveys of autism prevalence and found that the rates of AS were consistently lower than autism, with an average ratio of 3.5:1 for rates of autism versus AS. This translates into an estimated prevalence of 6/10,000 for AS alongside 20.6/10,000 for autism and 72.6/10,000 when more broadly defined cases are included (Fombonne 2009).

Future Directions

Therefore, in summary, there are many inconsistencies in the data, but there are a number of factors that might explain these discrepancies. It seems reasonable, however, to conclude that Asperger syndrome is a disorder that predominantly occurs in males and is significantly less common than autistic disorder. Prevalence figures range from 3 to 11 per 10,000 when ICD-10 and DSM-IV criteria are used, ignoring the hierarchy rule, and an estimated median prevalence of 6/10,000 has been suggested. If the hierarchy rule were to be applied, then the figure is likely to be significantly lower. It is also important to recognize that all studies quoted are from Europe or North America, and therefore, the prevalence in other countries is not known. It is even uncertain

as to the prevalence among different ethnic groups in the countries examined.

See Also

- [Asperger Syndrome](#)
- [Epidemiology](#)

References and Reading

- American Psychiatric Association. (1994). *DSM-IV diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Baird, G., Charman, T., Baron-Cohen, S., Cox, A., Swettenham, J., Wheelwright, S., et al. (2000). A screening instrument for autism at 18 months of age: A 6-year follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(6), 694–702.
- Chakrabarti, S., & Fombonne, E. (2001). Pervasive developmental disorders in preschool children. *Journal of the American Medical Association*, 285(24), 3093–3099.
- Chakrabarti, S., & Fombonne, E. (2005). Pervasive developmental disorders in pre-school children: Confirmation of high prevalence. *The American Journal of Psychiatry*, 162, 1133–1141.
- Ehlers, S., & Gillberg, C. (1993). The epidemiology of Asperger syndrome. A total population study. *Journal of Child Psychology and Psychiatry*, 34(8), 1327–1350.
- Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. *Pediatric Research*, 65(6), 591–598.
- Fombonne, E., Zakarian, R., Bennett, A., Meng, L., & McLean-Heywood, D. (2006). Pervasive developmental disorders in Montreal, Quebec, Canada: Prevalence and links with immunizations. *Pediatrics*, 118, e139–e150.
- Kadesjo, B., Gillberg, C., & Hagberg, B. (1999). Brief report: Autism and Asperger syndrome in seven-year-old children: Total population study. *Journal of Autism and Developmental Disorders*, 29(4), 327–331.
- Klin, A., McPartland, J., & Volkmar, F. R. (2005). Asperger syndrome. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 88–125). Hoboken: Wiley.
- Latif, A. H., & Williams, W. R. (2007). Diagnostic trends in autistic spectrum disorders in the South Wales valleys. *Autism*, 11(6), 479–487.
- Woodbury-Smith, M., Klin, A., & Volkmar, F. (2005). Asperger's syndrome: A comparison of clinical diagnoses and those made according to the ICD-10 and DSM-IV. *Journal of Autism and Developmental Disorders*, 35(2), 235–240.
- World Health Organization. (1993). *International classification of diseases (ICD-10)* (10th ed.). Geneva: Author.

Asperger Syndrome Follow-Up Studies

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Definition

Asperger syndrome (AS) is classified as one of several autism spectrum disorders (ASDs) or pervasive developmental disorders (PDDs; American Psychiatric Association 1994). As with other ASDs, the syndrome may be defined as a neurodevelopmental disability that involves significant delays or impairment in social interaction (e.g., age-appropriate friendships, sharing interest or attention with others), communication (conversational skills, nonverbal gestures), and a preference for restricted or atypical interests, stereotypes, or routines. Unlike autistic disorder, individuals with AS do not exhibit clinically significant delays in language development, adaptive functioning, or general intellectual abilities.

There exists considerable variation within clinical work and research as to how AS can be operationally defined using these criteria more precisely. Several researchers have commented that the DSM-IV criteria (APA 1994) are overly restrictive and often result in an underdiagnosis of AS (Cederlund et al. 2008; Howlin 2003; Miller and Ozonoff 1997; Szatmari et al. 1995). Several different definitions have therefore been used in research; however, all share the common features of core ASD deficits in the absence of clinically significant language or general cognitive delay.

Follow-up studies comprise a set of study designs that have the following features in common. The designs are (1) observational, that is, they involve studying individuals that have been naturally selected to a particular group or

exposure as compared to random assignment by researchers to a particular group as in experimental designs; (2) generally prospective, that is, the individuals of interest are followed chronologically and reassessed at one or more later time points; and (3) used to examine predictors and outcomes. Predictors are early factors or characteristics of the individual or his/her environment that are associated with variation in later occurring outcomes. Outcomes may be consequences of a diagnosis or of early predictors of interest.

Follow-up studies are important for several reasons. First, they help to clarify the diagnosis of Asperger syndrome and differentiate it from other ASDs and developmental disabilities. As with many mental health and developmental disorders, the validity of a diagnosis of AS may, at least in part, lie in the ability to distinguish a developmental course with respect to symptoms and functioning over time from other disorders. Second, follow-up studies help individuals, families, and clinicians understand the range of outcomes that may be expected in relation to a diagnosis such as AS, particularly as they relate to important aspects of daily life such as relationships, work, and self-sufficiency.

Prospective cohort studies assemble a group of similar individuals (a “cohort”) at one time point. These individuals are then followed up over one or more time points to determine whether and how variation in certain baseline factors relates to variation in outcomes of interest. If the cohort is followed over three or more time points (including baseline), trajectories or pathways of particular symptoms, abilities, or characteristics may be plotted to describe the rate and shape of change over time. Prospective cohorts are generally considered to produce higher quality evidence among observational studies than cross-sectional or retrospective outcome studies, particularly if they are able to ascertain individuals who are at the same “early” stage of the disorder (e.g., an “inception cohort”). Such designs also minimize error due to recall bias. Drawbacks of these studies include greater expense and length of time to complete data collection.

Retrospective cohort studies also involve assembling a group of similar individuals (e.g.,

individuals with ASD) that nevertheless differ on some traits or factors of interest (e.g., in this case, diagnoses of autism and Asperger syndrome), and are designed to assess whether the type of ASD is associated with differences in outcome. The process of collecting data differs, however. Retrospective studies look back to collect data that has already been recorded in the past to stratify the individuals into subgroups (e.g., records of diagnoses of autism or AS) as well as information on other important predictor (sex) variables or other associated factors. Outcome data may also have been collected in the past or concurrently, as a follow-up to earlier information. Disadvantages of retrospective cohort studies involve sample loss, potentially absent information about important confounders, and reliance on past methods of measurement which may have changed in the interim. Advantages include greater expediency of data collection and lower cost relative to prospective cohort studies.

Finally, *case-control studies* comprise another type of follow-up study, in which individuals with AS are sampled. They are then compared to control groups with respect to rate of earlier predictors or later outcomes of interest. For example, individuals with AS (the “cases”) and higher functioning autism (the “controls”) may be compared with respect to early characteristics and developmental milestones. These studies also have the advantage of saving cost and time to collect data; however, they are at greater risk of bias due to recall effects (e.g., parents of adult children recalling early developmental milestones) and sampling issues (e.g., missing individuals who do not present to a given clinic).

Historical Background

Case reports of children with features resembling Asperger syndrome (AS) were first mentioned in neurological and psychiatric literature in the 1920s (Gillberg 1998). However, Viennese pediatrician Hans Asperger most thoroughly described what he believed to be a new psychiatric disorder, which he termed “autistic psychopathy” (Asperger 1944). His descriptions of children with

disordered “affective contact” were developed around the same time as, but without consultation with, American child psychiatrist Leo Kanner, who also described children with similar traits as “autistic.”

The term “Asperger syndrome” gained significantly greater recognition and interest after it was reintroduced by Lorna Wing in 1981, based on her clinical observations of children and youth who demonstrated obvious autistic features but did not have the cognitive and language delays seen in autism (Wing 1981). An increasing number of publications began appearing to describe individuals with autistic traits who nevertheless demonstrated average or near-average intelligence and language abilities. Asperger syndrome was included in ICD-10 and DSM-IV as one of the pervasive developmental disorders with specific criteria setting it apart from autism and pervasive developmental disorder NOS. Autism and Asperger syndrome were defined as sharing several of the same criteria, with the latter defined as having relatively normal cognitive functioning and language abilities, the absence of language delay, and fewer communication impairments overall. A hierarchical rule was established, such that any individual meeting criteria for both autism and Asperger syndrome would be diagnosed with the former. This rule, as pointed out by many clinical researchers, significantly decreases the number of individuals eligible for a diagnosis of AS (Cederlund et al. 2008; Howlin 2003; Szatmari 2000). Accordingly, definitions of AS have varied across research studies, in efforts to capture samples of individuals who reflect a “true” picture of the disorder.

In spite of this growing literature, there have been relatively few prospective follow-up studies of Asperger syndrome, as distinct from other pervasive developmental disorders and, in particular, high-functioning autism. Gillberg and colleagues followed up young men who had been diagnosed with Asperger syndrome 5 or more years earlier (Cederlund et al. 2008), whereas Szatmari and colleagues followed a cohort of children aged 4–6 recently diagnosed with Asperger syndrome and high-functioning autism every 2–4 years into adolescence (Szatmari et al. 2000). Other studies

used individuals with AS and high-functioning autism who have presented as adolescents or adults to clinical services and then examined current and retrospective features associated with the diagnosis (Gilchrist et al. 2001; Howlin 2003). The differing study designs and definitions of AS have led to some variation in results, particularly regarding the extent to which AS is distinct from high-functioning autism. However, all share the common goal of understanding how individuals with AS fare as they age into adulthood with respect to symptoms, adaptive functioning, and quality of life.

Current Knowledge

Follow-up studies of individuals with Asperger syndrome (AS) have been few in number and have differed widely with respect to their overall design, the definition of Asperger syndrome used, the sampling methods for finding cases with AS, the type of comparison group employed, and how predictors and outcomes are measured (Cederlund et al. 2008; Gilchrist et al. 2001; Howlin 2003; Szatmari et al. 2003, 2009). Nevertheless, they share a common goal of understanding how individuals with AS fare in later childhood, adolescence, and adulthood with respect to important outcomes of interest – their core developmental abilities and their overall level of adaptive functioning as individuals in society. Understanding the course of development and outcomes in Asperger syndrome is related to the predictive validity of the diagnosis: whether the disorder helps forecast a developmental pathway for AS that is distinct from that of Autistic Disorder in a measurable and meaningful way. More importantly, it helps individuals and their families understand the implications of such a diagnosis and plan for their future, while aiding clinicians in service development by anticipating their future needs.

Childhood

Studying the short-term outcomes of children with Asperger syndrome sheds light on baseline variation between children with autism spectrum disorders and the importance of early developmental

“head starts.” For example, Szatmari et al. (2000, 2003) followed up 68 children aged 4–6 years old who were diagnosed with either autistic disorder or Asperger syndrome and had IQs of at least 68 standard score points (Szatmari et al. 2000, 2003). Children diagnosed with Asperger syndrome had significantly better socialization scores on the Vineland Adaptive Behavior Scales at baseline and 2 years later compared to children with autistic disorder, controlling for initial language ability and nonverbal IQ. Children with autistic disorder who gained functional language over the course of the follow-up period achieved socialization scores similar to the Asperger syndrome group at baseline. These early studies indicated that children with Asperger syndrome seem to embark on parallel, but higher functioning, trajectories compared to peers with autistic disorder and that the achievement of verbal fluency may act as an important early differentiating step between developmental pathways (Szatmari et al. 2009).

Adolescence and Early Adulthood

The evaluation of how well individuals with Asperger syndrome fare in adolescence and early adulthood understandably depends upon the group to whom individuals with AS are compared. Researchers using data from two separate prospective follow-up cohort studies (Bennett et al. 2008; Cederlund et al. 2008; Szatmari et al. 2009) found that young adults with AS have better outcomes with respect to ASD symptom burden and adaptive functioning compared to individuals with autistic disorder (including high-functioning autistic disorder with IQ > 70). In a prospective study of young adults with AS, outcomes were classified as poor (“obvious severe handicap, no independent social improvement”), restricted, fair, and good outcomes (engaged in IQ-appropriate work or education and living independently if over 23 years of age or having steady friendships/relationships if younger than 23) (Cederlund et al. 2008). Only 26% of individuals with AS were classified as having “poor” or “restricted” outcome, compared to 64% of those with AD. Retrospective case–control studies have found few if any differences (Gilchrist et al. 2001;

Howlin 2003). A common consensus among studies, however, is that young adults with AS – despite normal-range IQ and absence of early language delays – have striking difficulties across a wide range of domains compared to typically developing individuals. For example, despite a mean full-scale IQ for the AS group of 103.0 in Cederlund et al.'s (2008) study, 47% were classified as having “fair” and only 27% were deemed to have “good” outcomes.

Core ASD Symptoms

A majority of individuals with AS continue to struggle with significant social communication deficits in early adulthood accompanied by significant associated impairment, with the exception of a small number who appear to improve significantly into a relatively unimpaired status (Cederlund et al. 2008). One study found that the mean Global Assessment of Functioning Score (GAF) – a clinical measure of impairment due to symptom burden – was 58.9, indicating moderate symptom burden or impairment. However, 17% of individuals with AS in this study had GAF scores greater than 70, indicating normal or near-normal functioning; 11% of all those diagnosed with AS and later followed up no longer met criteria for AS. As a group, individuals with AS demonstrated less impairment than individuals diagnosed with autism as children (mean GAF = 22.4). However, in a study comparing individuals with AS with high-functioning individuals with autistic disorder, there were no significant differences in ASD symptoms as measured by the autism diagnostic interview-revised (ADI-R; Lord et al. 1994).

Nevertheless, autistic symptoms seem to decrease over time in individuals with AS, as an overall group (Szatmari et al. 2009). Researchers examining the rate of change in core autistic symptoms found a relatively linear rate of decrease from preschool to adolescent years, with a slightly faster rate of change between ages 5–10 years (Szatmari et al. 2009). This rate of change was similar to that for a group of autistic individuals with IQ > 70; however, those with AS maintained a comparatively lower burden of symptoms overall from childhood into late adolescence.

Cognitive Profile

Cognitive abilities as measured by full-scale performance and verbal IQ have generally been found to be stable from childhood to adolescence/early adulthood in AS (Cederlund et al. 2008). There is some evidence that the relative superiority of verbal IQ over performance IQ often described in individuals with AS compared to individuals with autism is less common by adolescence/young adulthood (Cederlund, et al.).

Comorbid Psychiatric Symptoms

Outcome studies indicate that psychiatric comorbidity is a common problem for individuals with AS, with rates similar to those of individuals with high-functioning autistic disorder and PDD-NOS but higher than those seen in the general population (Hofvander et al. 2009; Howlin 2003). Mood and anxiety disorders appear more commonly in adolescence and young adulthood than childhood, occurring in 21–52% of individuals with AS according to two case-control studies (Hofvander et al. 2009; Howlin 2003). Attention-deficit/hyperactivity disorder (36%), tic disorder (21%), and obsessive-compulsive disorder (21%) have also been found to be more common than in controls (Hofvander et al. 2009). Rates of psychotic disorders measured in clinical and population samples of individuals with AS range from 4% to 15% (Cederlund et al. 2008; Hofvander et al. 2009).

Adaptive Functioning

Perhaps the most striking burden of Asperger syndrome in adolescence and adulthood falls under the domain of adaptive functioning – the ability to support oneself in day-to-day self-care, independent living, and financial self-sufficiency, to engage in relationships and to pursue vocational interests. Adaptive functioning is an important measure of impairment related to AS, as well as an indirect measure of burden of care on parents, schools, and community systems of care. Furthermore, adaptive functioning has been found to be associated with self-reported quality of life among individuals with high-functioning ASD (Kamp-Becker et al. 2010). Prospective research has found that individuals with AS

demonstrate significant improvement in adaptive functioning throughout childhood to adulthood, with some slowing of progress during adolescence. It must be remembered, however, that individuals with AS remain significantly impaired compared to the general population (Szatmari et al. 2009).

Educational achievement appears to be a relative strength among individuals with AS. A greater number of individuals with AS achieve advanced levels of schooling, compared to those with autism. In one study, twice as many individuals with AS (52%) completed advanced level high school courses as did those with high-functioning autism (24%) (Howlin 2003). Another study found that 11.4% of individuals with AS enrolled themselves in university, and 5.6% had obtained university degrees, while 33% completed high school studies. Among high-school completers, over half (64%) completed their studies in mainstream classrooms (Cederlund et al. 2008).

Such academic abilities do not seem to translate into longer term vocational success or self-sufficiency, however. Only 5–10% of individuals with AS hold permanent, well-paying jobs in adulthood, whereas a greater number work in short-term, low-pay, or voluntary posts or structured work activities in a support center (Cederlund et al. 2008; Howlin 2003). Furthermore, despite normal IQ abilities, only 35–64% of young adults with AS live independently, the majority of these requiring ongoing parent support (Cederlund et al. 2008; Howlin 2003).

Consistent with reports of persistent social disabilities related to their diagnosis, individuals with AS report ongoing difficulties in maintaining social and romantic relationships. Approximately 4–15% of individuals report long-term relationships such as longstanding close friendships, romantic partnerships, or marriage (Cederlund et al. 2008; Howlin 2003), although a larger number of individuals (40.5%) in one study reported having a range of less intimate friends and acquaintances (Howlin 2003). Nevertheless, individuals with AS remain persistently less impaired than are individuals with autism (including high-functioning autism) from childhood into adulthood (Szatmari et al. 2009).

Predictors of Adolescent Outcome

IQ and language abilities have been found to be important predictors of improved functioning in adolescents and adults with AS and HFA (Bennett et al. 2008; Cederlund et al. 2008; Szatmari et al. 2009). This finding is in keeping with research combining ASDs of all cognitive abilities (Baghdadli et al. 2007). Higher full-scale and verbal IQ scores are associated with improved overall outcome in adolescents and young adults with AS (Cederlund et al. 2008). Because AS is defined largely by the absence of clinically significant language delay in most research studies, this feature has been proposed to account for improved outcomes compared to individuals with autism and IQ > 70 found in prospective studies (Cederlund et al. 2008; Szatmari et al. 2003), although not in retrospective investigations (Gilchrist et al. 2001; Howlin 2003). Structural language impairment at age 6 years, defined as significant deficits in non-pragmatic aspects of language (e.g., grammar, syntax), has been found to predict greater variability in functional outcomes across social, communication, and daily living skill domains in adolescents in high-functioning ASD than the presence of language delay (Bennett et al. 2008; Szatmari et al. 2009). This suggests that structured assessments of language as well as cognitive ability in preschool/early school-aged years are important early steps in understanding the prognosis of children with high-functioning ASDs. There is evidence of persistent language impairment in individuals with AS compared to typically developing norms (Howlin 2003); however, its role in predicting outcomes more specifically within this group has yet to be studied systematically.

Summary and Conclusions

In evaluating how individuals with AS fare in adolescence and adulthood, it is important to consider the standards against which they are being compared. According to prospective follow-up studies, the outcomes of individuals with AS are significantly better compared to those with autistic disorder, including high-functioning autism. This is likely a function of better cognitive and

language ability, which puts them at an early and persistent advantage compared to lower functioning peers with autism. However, given these cognitive capacities, young adults with AS continue to have difficulty living and working independently and remain significantly burdened by social impairment. Furthermore, rates of early improvement in functioning begin to plateau in late adolescence, which may reflect slowed learning or simply an inability for this learning to keep pace with the increasing functional demands of transition to adulthood. These findings highlight the obvious need for continued vocational, social, and daily living supports for teens and adults with Asperger syndrome.

Future Directions

Despite a relatively small number of follow-up studies of young people with Asperger syndrome published to date, the data clearly demonstrate that as a group, their functioning and well-being in adolescence and adulthood are poor relative to typically developing individuals. This consensus underlines the importance of having a better understanding of how early determinants and developmental pathways lead to variation in outcomes. While prospective studies suggest that improved cognitive and early language abilities account for superior outcomes in Asperger syndrome compared to autistic disorder, more work is needed to understand early predictors within groups of individuals diagnosed with AS taking into account the considerable heterogeneity in outcome in this population. Future longitudinal studies should focus on unpacking *how* early predictors lead to later outcomes, for example, through *mediators* (individual or contextual factors that account for the association between predictors and outcomes) or *moderators* (groups or circumstances under which an effect occurs or not). For example, poorer mental health may mediate, or explain, an association between earlier individual traits or cognitive abilities and later adaptive functioning in certain individuals.

Future study designs that combine intervention and longitudinal follow-up will be particularly

important in both elucidating developmental pathways in Asperger syndrome and addressing the suboptimal outcomes that all too often occur. Hybrid studies may include intervention trials (ideally randomized and controlled) and long-term measurement of outcomes. Early social communication interventions, prevention, or treatment trials for depression and anxiety and more intensive social, communication, and vocational supports for adolescents may each demonstrate effects on more global or specific aspects of functioning and quality of life in adulthood. Finally, follow-up and intervention studies should also follow individuals with Asperger syndrome farther into adulthood to continue to track their pathways in learning and functioning and to determine how best to encourage their strengths and support their needs.

See Also

- [Adulthood, Transition to](#)
- [Asperger Syndrome](#)
- [Factors Affecting Outcomes](#)
- [Longitudinal Research in Autism](#)
- [Outcome Studies](#)

References and Reading

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Press.
- Asperger, H. (1944). Die 'autistischen Psychopathen' im Kindesalter. *Arch Psychiatrie Nerven*, 117, 60.
- Baghdadli, A., Picot, M.-C., Michelon, C., Bodet, J., Pernon, E., Berstezjn, C., & Aussiloux, C. (2007). What happens to children with PDD when they grow up? Prospective follow-up of 219 children from pre-school age to mid-childhood. *Acta Psychiatrica Scandinavica*, 115, 9.
- Bennett, T., Szatmari, P., Bryson, S., Volden, J., Zwaigenbaum, L., Vaccarella, L., & Boyle, M. (2008). Differentiating autism and Asperger syndrome on the basis of language delay or impairment. *Journal of Autism and Developmental Disorders*, 38(4), 616–625. <https://doi.org/10.1007/s10803-007-0428-7>.
- Cederlund, M., Hagberg, B., Billstedt, E., Gillberg, I. C., & Gillberg, C. (2008). Asperger syndrome and autism: A comparative longitudinal follow-up study more

- than 5 years after original diagnosis. *Journal of Autism and Developmental Disorders*, 38(1), 72–85. <https://doi.org/10.1007/s10803-007-0364-6>.
- Gilchrist, A., Green, J., Cox, A., Burton, D., Rutter, M., & Le Couteur, A. (2001). Development and current functioning in adolescents with Asperger syndrome: A comparative study. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 42(2), 227–240.
- Gillberg, C. (1998). Long-term course of autistic disorders. *Acta Psychiatrica Scandinavica*, 97, 9.
- Hofvander, B., Delorme, R., Chaste, P., Nyden, A., Wentz, E., Stahlberg, O., & Leboyer, M. (2009). Psychiatric and psychosocial problems in adults with normal-intelligence autism spectrum disorders. *BMC Psychiatry*, 9, 35. <https://doi.org/10.1186/1471-244X-9-35>.
- Howlin, P. (2003). Outcome in high-functioning adults with autism with and without early language delays: Implications for the differentiation between autism and Asperger syndrome. *Journal of Autism and Developmental Disorders*, 33(1), 3–13.
- Kamp-Becker, I., Schroder, J., Remshmidt, H., & Bachmann, C. J. (2010). Helath-related quality of life in adolescents and young adults with high-functioning autism spectrum disorder. *GMS Psycho-Social Medicine*, 7, 10. <https://doi.org/10.3205/psm000065>.
- Longitudinal Studies: Design and Analysis.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24, 26.
- Miller, J. N., & Ozonoff, S. (1997). Did Asperger's cases have Asperger disorder? A research note. *Journal of Child Psychology and Psychiatry*, 38(2), 247–251.
- Szatmari, P. (2000). The classification of autism, Asperger's syndrome, and pervasive developmental disorder. *Canadian Journal of Psychiatry. Revue Canadienne de Psychiatrie*, 45(8), 731–738.
- Szatmari, P., Archer, L., Fisman, S., Streiner, D. L., & Wilson, F. (1995). Asperger's syndrome and autism: Differences in behavior, cognition, and adaptive functioning. *Journal of the American Academy of Child & Adolescent Psychiatry*, 34(12), 1662–1671.
- Szatmari, P., Bryson, S. E., Streiner, D. L., Wilson, F., Archer, L., & Ryerse, C. (2000). Two-year outcome of preschool children with autism or Asperger's syndrome. *The American Journal of Psychiatry*, 157(12), 1980–1987.
- Szatmari, P., Bryson, S. E., Boyle, M. H., Streiner, D. L., & Duku, E. (2003). Predictors of outcome among high functioning children with autism and Asperger syndrome. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 44(4), 520–528.
- Szatmari, P., Bryson, S., Duku, E., Vaccarella, L., Zwaigenbaum, L., Bennett, T., & Boyle, M. H. (2009). Similar developmental trajectories in autism and Asperger syndrome: From early childhood to adolescence. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 50(12), 1459–1467. <https://doi.org/10.1111/j.1469-7610.2009.02123.x>.
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11, 15.

Asperger Syndrome Training & Employment Partnership (ASTEP)

Michael Carley
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The Asperger Syndrome Training & Employment Partnership (ASTEP) is a nonprofit 501(c)3 organization that promotes the inclusion of individuals with Asperger syndrome and high-functioning autism (AS/HFA) in competitive employment through:

1. Awareness and training campaigns aimed at Fortune 1000 companies
2. Establishing relationships between high-quality support programs for adults with Asperger throughout the US and national employers
3. Developing corporate partners to implement integrated employment programs for adults with Asperger syndrome

Founded in 2010 by Marcia Scheiner, ASTEP was created to fill a hole in the autism/Asperger world. Prior to ASTEP, numerous opportunities existed for job placement, but no organizations were helping employers create more inclusive workplace environments for existing employees with AS/HFA or bringing together employers and vocational support professionals to successfully recruit and integrate individuals with AS/HFA. ASTEP does this by educating employers and building relationships.

ASTEP educates employers about the special skills and talents of individuals with AS/HFA, the business benefits of employing people with Asperger syndrome, and the accommodations they may need in the workplace. It encourages employers to incorporate the hiring and retention of individuals with AS/HFA into their diversity and inclusion programs and gives them the tools and training they need to feel confident. ASTEP offers video-based corporate training opportunities, as well as on-site training presentations.

ASTEP also builds relationships with vocational support organizations to facilitate employer access to candidates with AS/HFA. Vocational support organizations can be privately funded organizations, state or federally funded organizations, or postsecondary educational programs that provide specialized support for students and young adults with AS/HFA.

While ASTEP does not provide support services to individuals looking for work opportunities, it offers web resources and programs to assist such individuals on their job hunt on ASTEP's Assistance for Individuals page.

For more information on ASTEP and its programs, you can email them at info@asperger-employment.org, or visit their webpage at www.asperger-employment.org.

Asperger, Hans

Adam Feinstein

Autism Cymru and Looking Up, London, UK

Major Appointments (Institution, Location, Dates)

- Hans Asperger was appointed director of the play-pedagogic station at Vienna University children's clinic in 1935.
- Appointed a lecturer at the University of Vienna, 1944.
- Appointed director of the children's clinic, 1946.
- Named professor at the University of Innsbruck children's clinic, 1957.
- Named professor at the University of Vienna children's clinic, 1962.

Landmark Clinical, Scientific, and Professional Contributions

Dr. Asperger was working in the field of what he called "autistic psychopathy" in Vienna from the early 1930s – several years before Leo Kanner began working on infantile autism at Johns

Hopkins University in Baltimore. His first published paper in this area was not the celebrated 1944 paper but "Das psychisch abnorme Kind," which appeared in the *Wiener Klinischen Wochenschrift* in 1938 (Asperger 1938). This was the transcript of a talk Asperger had given at Vienna University earlier that year. It is a remarkable document: Asperger, concerned to protect the children in his charge from the eugenics law which he feared would be introduced by the Nazis in the newly annexed Austria, carefully used terminology reminiscent of Nazi thinking while at the same time pointing out the valuable contributions the children could make to society. The Gestapo came to arrest him twice, but he received the support of his boss – Franz Hamburger, dean of the university – who ironically, unlike Asperger, was sympathetic to the Nazis. Asperger's 1944 paper, 'Die autistischen Psychopathen' im Kindesalter – which appeared in *Archiv für Psychiatrie und Nervenkrankheiten* – provided detailed descriptions of four children with autistic psychopathy, or what Lorna Wing, in 1981, called "Asperger's syndrome" (Asperger 1944; Wing 1981). Unlike in classic (or "Kanner's") autism – where there is language delay and IQ can be anywhere on the scale – in Asperger's syndrome, there is no language delay and IQ is at least average. Asperger believed that his syndrome was never recognised in infancy and not usually before the third year of life or later. Kanner emphasised onset of his condition from birth or before 30 months. Unlike Kanner, Asperger thought of his condition as a personality disorder with organic causes. While Kanner reported that three of his 11 patients did not speak at all, and the remainder rarely used language to communicate, Asperger noted that his case study patients spoke "like little adults". There were also discrepancies regarding gross co-ordination and fine motor skills. Kanner reported that, although the former was poor the latter was very good. Asperger observed that both were affected. Kanner believed that learning by rote would be the best method of advancing an autistic person, while Asperger suggested that his patients were "abstract thinkers" and therefore performed best spontaneously. Asperger said his patients were highly intelligent and capable of original thought. He referred to them as "little professors".

Short Biography

Hans Asperger was born on a farm outside Vienna on February 18, 1906. He was appointed director of the play-pedagogic station at Vienna University children's clinic. He married in 1935 and had five children, including two daughters who themselves became doctors. In the later part of the Second World War, Asperger served as a doctor in Croatia. His daughter, Dr. Maria Asperger Felder, told Adam Feinstein: "He was against war. He was a nature- and people-loving person, not a soldier." In 1944, he became a lecturer at the University of Vienna and was appointed director of the children's clinic in 1946. In 1957, Asperger became professor at the University of Innsbruck children's clinic and, from 1962, held the same position in Vienna. Despite the fact that he traveled around the world, his writings were not mentioned at a major psychiatry conference in Zurich in April 1957. The veteran French autism authority, Professor Gilbert Lelord, who attended this congress, told Adam Feinstein that this may well have been a consequence of the Second World War: "Even though Asperger was undoubtedly a victim of the war, German-language papers were not popular at the time." Indeed, Asperger's writings did not come to the attention of the English-speaking world until Lorna Wing's 1981 paper and Uta Frith's 1991 translation into English of Asperger's 1944 paper. Leo Kanner never mentioned Asperger in any of his own papers, whereas Asperger often cited Kanner, always insisting that his syndrome was distinct from Kanner's. Asperger's syndrome was listed officially for the first time in ICD-10 in 1992 and in DSM-IV in 1994.

References and Reading

- Asperger, H. (1938). Das psychisch abnorme kind. *Wiener Klinischen Wochenzeitschrift*, 51, 1314–1317.
- Asperger, H. (1944). Die "autistischen Psychopathen" im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136 [Autistic psychopathy in childhood] (U. Frith (Ed.), Trans., (1991), *Autism and Asperger syndrome* (pp. 37–92)). Cambridge: Cambridge University Press.
- Feinstein, A. (2010). *A history of autism: Conversations with the pioneers*. Oxford: Wiley-Blackwell.
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11(1), 115–130.

Asperger's

► [Pareidolic Faces](#)

Asperger's Disorder

► [Asperger Syndrome](#)

Aspire: The Asperger Syndrome Association of Ireland

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Introduction

The Asperger Syndrome Association of Ireland Ltd. (Aspire) was established in 1995 as a registered charity (CHY 11438) and a company (incorporation No. 231996). The original founders were a small group of parents who were concerned at the lack of awareness and information concerning Asperger syndrome or high-functioning autism among parents and professionals and the absence of services specifically designed for those with the condition. The association was set up to provide support to individuals who have Asperger syndrome/high-functioning autism and their families and other caregivers and to encourage and undertake research into the condition.

Address: The Asperger Syndrome Association of Ireland Ltd., Carmichael Centre for Voluntary Groups, Coleraine House, Coleraine Street, Dublin 7, Republic of Ireland.

Membership: On the 31st of December 2013, the number of registered members of the association was 220.

Website: www.aspireireland.ie

Mission Statement: The mission of the association is to assist people with Asperger syndrome/

high-functioning autism to lead more fulfilled lives and to support their caregivers.

Landmark Contributions

The major landmark contributions of the Aspire have been the raising of awareness of Asperger syndrome/high-functioning autism and the development of services to assist people with the condition and their caregivers. When the association was set up, there was very little information available and no services specifically developed for people with this form of autism in Ireland. Since 1995, the Aspire Helpline, the holding of regular conferences/seminars and courses each year together with the development of the Aspire website, has improved the awareness of Asperger syndrome in Ireland. The association has also encouraged and assisted in the production of a range of programs on radio, TV, and other media to highlight the disorder. Professor C. Gillberg addressed a conference in Dublin, Ireland, organized on the tenth anniversary of the founding of the Aspire. In 2006, the Honorary Secretary of the Association received an award from Dublin City Council for his voluntary work, over the previous 11 years, in raising awareness of Asperger syndrome/high-functioning autism in Ireland. Every year visits to schools are a priority in the Aspire awareness-raising campaign.

A major conference was held in October 2010 to address the co-occurrence of conditions such as Asperger syndrome with dyspraxia, hyperactive disorder, and dyslexia. This conference was the first of its kind to be held in Ireland.

In 2009, Aspire in conjunction with Trinity College Dublin produced a DVD entitled "Asperger Syndrome: A Practical Guide for Parents, Teachers, Young People and Other Professionals," and this was circulated to all public schools in the Republic of Ireland. A landmark conference was also held at Trinity College in November 2013 entitled "Challenging DSM 5," and international speakers Professor F. Volkmar and Professor P. Howlin spoke on the changes being introduced in the diagnosis of autism by the American Psychiatric Association under DSM 5.

The setting up of Educational Drama Classes in collaboration with the School of Education at

Trinity College Dublin in September 2004 was a major contribution in the development of services for children, adolescents, and adults affected by Asperger syndrome/high-functioning autism. These classes have made a significant contribution to improving the social and life skills of participants and over the past decade have shed new light on the methods used in the teaching of social skills. The identification of a large number of subtypes within the diagnosis of Asperger syndrome/high-functioning autism by Dr. Carmel O'Sullivan (Director of the Drama Classes) has been a significant milestone in the understanding of the disorder.

Landmark Activities: The most important activities of the association are:

The provision of a Helpline available to Aspire members and the general public from Monday to Friday. These telephone contacts with a large number of people keep Aspire up to date with the issues families are experiencing and identify the problems currently being encountered – mainly by parents. The supports provided by Aspire are tailored to take into account the issues raised on the Helpline.

Raising awareness and understanding of Asperger syndrome using all the media available assists people with a diagnosis of Asperger syndrome to develop their potential in a more understanding and supportive environment.

Provision of Educational Drama Classes to teach social skills to those with a diagnosis of Asperger syndrome/high-functioning autism.

A supported employment and career development service for adults with a diagnosis of Asperger syndrome/high-functioning autism.

Holding regular conferences, seminars, courses, and workshops for all those interested in the condition.

Assisting support groups set up to provide assistance to those affected by Asperger syndrome/high-functioning autism and their caregivers.

Residential unit for adults with Asperger syndrome/high-functioning autism.

Major Areas of Activity

Aspire Helpline service provides contact with over 1,200 callers each year who are seeking

information, support, and advice. Topics of enquiry range from diagnosis, education, assessment, social skills, adult issues, local services, training, and general information. The majority of callers are parents of children with Asperger syndrome or those seeking a diagnosis. The Helpline also receives a large number of calls from adults with the disorder and professionals such as teachers and journalists.

Educational Drama Classes (in Conjunction with Trinity College Dublin) Provide Social Skill Training to About 90 Children, Adolescents, and Adults
Aspire maintains a website with up-to-date information about Asperger syndrome, the latest news and events, including fund-raising, together with general information.

It is divided into information for parents and professionals and information for people with Asperger syndrome. Aspire also keeps up to date using social media through Facebook and Twitter, to ensure that the information we provide is easily accessible to as wide a population as possible.

Aspire supports over 30 groups which were set up to support people with Asperger syndrome and their families. Aspire assists volunteer parents and people with Asperger syndrome to set up the groups and visit and provide informational talks to these groups where required. We also direct any families who contact us to their local group(s). This type of peer support has been found over the years to be extremely important and a hugely valuable resource provided at very low cost.

Aspire visits a large number of schools and third-level institutions each year to promote an understanding of Asperger syndrome and to advise on the supports which need to be provided to students with the disorder.

These visits are made to enhance the educational experience of students with Asperger syndrome and ensure that they have the opportunity to get the best from their educational experience which will aid them in later life in gaining and maintaining employment.

Adults who have Asperger syndrome struggle to find suitable employment as a result of

challenges with communication, social interaction, and anxiety. As adult services are limited in Ireland, Aspire has developed a supported employment and career development program for adults with Asperger syndrome. The needs of participants are identified, and they are supported in areas such as CV preparation, interview skills, interaction with colleagues, and applications. Aspire also liaises with employers to ensure that they have an understanding of Asperger syndrome and provide them with relevant information and support.

ASQ Family Access

- [Ages and Stages Questionnaire, Second Edition](#)

ASQ Hub (for Monitoring Screening Programs of Multiple Organizations)

- [Ages and Stages Questionnaire, Second Edition](#)

ASQ Pro (for Single-Site Programs) and ASQ Enterprise (for Multisite Programs)

- [Ages and Stages Questionnaire, Second Edition](#)

ASQ:SE

- [Ages and Stages Questionnaire, Second Edition](#)

ASQ-3 Materials Kit

- [Ages and Stages Questionnaire, Second Edition](#)

ASQ-3™

- [Ages and Stages Questionnaire, Second Edition](#)
-

ASRS

- [Autism Spectrum Rating Scale](#)
-

Assessing Quality of Life in Autism

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Definition

Given the complex and diverse lives of individuals with autism spectrum disorder (ASD), it is valuable to apply a comprehensive conceptualization of quality of life (QoL) when assessing life course and treatment outcomes for individuals with ASD. In the general population, QoL is considered a multidimensional construct. For example, the World Health Organization defines subjective QoL as an “individuals’ perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards and concerns” (WHOQOL Group 1995, p. 1405). Accordingly, standardized measures of QoL in the general population typically include items in the physical, psychological, and social domains (Danckaerts et al. 2010). For individuals with developmental disabilities, including ASD, researchers have similarly applied a multidimensional framework to the study of QoL. One of the most commonly utilized definitions comes from the work of Schalock and colleagues who drew upon concepts from the literature in the

general population and applied them to individuals with developmental disabilities, resulting in eight key areas of QoL: interpersonal relations, social inclusion, personal development, physical well-being, self-determination, material well-being, emotional well-being, and human and legal rights. For individuals with ASD, other related markers of a high quality of life also have been suggested such as social participation with family and community, having choices, feelings of happiness, and satisfaction and meaning in work (Ruble and Dalrymple 1996). Drawing from these definitions, most contemporary measures of QoL for individuals with ASD include multidimensional components reflecting subjective and objective characterizations of QoL across a range of contexts.

Historical Background and Measurement

In the general population, QoL is typically measured by self-reports capturing personal appraisal of various life domains. However, for individuals with ASD, this measurement strategy can pose unique challenges due to the impairments in communication and/or cognitive functioning that many individuals across the range of the autism spectrum face. To address such potential barriers to the study of QoL, Verdugo et al. (2005) proposed several relevant guidelines for measurement of QoL among individuals with developmental disabilities, namely, that in addition to the individual with the disability acting as the respondent, family members likewise can report on the QoL of the individual. Further, it may be valuable to gather information on QoL using both quantitative and qualitative methodologies and to probe questions related not only to common/universal experiences across physical, social, and cultural contexts, but also to experiences that are more unique to individuals with disabilities (Verdugo et al. 2005). For example, while most population-based measures of quality of life will include multiple general indicators of health, for individuals with disabilities additional questions such as items regarding choice or

empowerment in health care decisions may be needed for a more holistic understanding of quality of life for that dimension. These multi-informant, multimethod approaches allow for a fuller characterization of quality of life for individuals than self-report alone and have been successfully employed in studies of individuals with developmental disabilities including ASD.

Below we discuss specific measures of QoL that have been utilized in the past two decades in samples of individuals with ASD. We summarize measures that include a self-report option and also provide an overview of measures that are exclusively parent or caregiver report. We note that some of these measures were developed for general populations, thus reflecting universal experiences (and subsequently were administered to individuals with ASD or their family members, potentially with modifications), whereas other instruments were intentionally designed to assess QoL dimensions specifically for individuals with disabilities from the onset. Further, some measures focus more on the perspectives of the individual with ASD (either through self-report or proxy report), whereas others gather information about the environment (e.g., presence/absence of activities). Notably, given the diversity of the autism phenotype, particularly in the areas of information processing and reading comprehension, self-report measures designed for the general population may be appropriate for some individuals with ASD without modifications, whereas for other individuals, significant modifications may be needed. For others, some self-report measures simply may not be accessible. In contrast, certain items from measures developed specifically for individuals with intellectual and developmental disabilities and/or ASD may not be relevant for all individuals with ASD depending on age and level of functioning. As such, when selecting measures of QoL, special consideration should be given regarding the intended purpose of the instrument and the populations for whom it was designed and with whom it has been validated.

The *Pediatric Quality of Life Inventory* (PedsQL 4.0; Varni et al. 2001) is a QoL measure designed for pediatric groups in the general population that has both child self-report and parent

proxy report options. The inventory includes subscales (physical functioning, emotional functioning, social functioning, and school functioning) and has 23 items which assess how much of a problem the child has had in the past month on a 5-point scale (0 = never a problem to 4 = almost always a problem). There are versions for different age groups (e.g., young children vs. youth/young adults), with response options reflecting the developmental level of the respondent. For example, the version for young children (ages 5–7) includes simplified language and happy/sad faces. The PedsQL self-report version has been successfully employed in samples of adolescents with ASD with IQs of 70 or greater (Shipman et al. 2011; Sheldrick et al. 2011), with a strong correlation between the adolescent and parent report versions ($r = .40^*$; Shipman et al. 2011). The parent report of the PedsQL also has been used with children with ASD as part of the Autism Treatment Network (Kuhlthau et al. 2010) and in sample of children with Asperger's (Limbers et al. 2009).

The *World Health Organization Quality of Life Instrument, Abbreviated Version* (WHOQOL-BREF; WHOQOL Group, 1998) is a widely used 24-item self-report questionnaire of subjective QoL designed for adults in the general population across multiple cultural groups. However, this measure can also be administered through proxy or other report. The WHOQOL-BREF assesses QoL in four domains: (1) physical health, (2) psychological health, (3) social relationships, and (4) environment. The measure has a 5th-grade reading level and has been successfully implemented in samples of adolescent and young adult males with Asperger syndrome/HFA (Kamp-Becker et al. 2010; Jenees-Coussens et al. 2006). There is also a disability-specific module available (Power et al. 2010). In a recent study of adults with ASD aged 25–50 across a range of intellectual functioning (30% had cooccurring intellectual disability), adults with ASD were shown to rate their own QoL reliably using the WHOQOL-BREF (Hong et al. 2016). Notably, for this study, screening was conducted prior to administration of the measure (e.g., record review to document general comprehension skills and practice items with respondents to gauge

understanding). Modifications also were made to the instrument to increase accessibility such as including additional words/phrases to add clarity when the original wording was difficult to understand (e.g., “Are you able to accept your bodily appearance” was supplemented with “Do you like how you look?” if the participant asked for help in understanding the question) and providing visual supports for response categories (e.g., a response card showing facial expressions, such as a broad smile to a deep frown, for each response option; Hong et al. 2016).

Following the comprehensive conceptualization of QoL put forth by Schalock and colleagues, two self-report QoL measures have been developed specifically for individuals with intellectual and developmental disabilities; these measures include items that cover a range of areas relevant to disability and are accessible for individuals with cognitive limitations. The *Quality of Life Questionnaire* (QOL.Q; Schalock and Keith 1993) is a 40-item self-report interview designed to assess QOL in populations of individuals with intellectual and developmental disabilities. The QOL.Q consists of four subscales with 10 items per subscale: (1) satisfaction, (2) competency/productivity, (3) empowerment/independence, and (4) social belonging/community integration. The *Cross-Cultural Survey of Quality of Life Indicators* (Verdugo et al. 2005) is a related paper/pencil self-report survey for individuals with mild or moderate intellectual disability. A 4-point Likert scale is used to measure importance (*not important at all* to *very important*) and use (*never* to *always*) for 24 indicators across eight domains (emotional well-being, interpersonal relations, material well-being, personal development, self-determination, physical well-being, rights, and social inclusion). A recent analysis suggested that the measure represents three two-order factors: independence (comprising self-determination and personal development), social integration (comprising interpersonal relationship, rights, and social inclusion), and well-being (comprising emotional well-being, material well-being, and physical well-being; Wang et al. 2010).

Other QOL instruments draw on the perspectives and objective reports of others in the lives of

individuals with ASD (e.g., parents, caregivers) to assess QoL rather than employing self-reports of these individuals. These approaches may be especially salient when studying populations of children with ASD or adults with communication challenges. For example, Lee et al. (2008) assessed parent reported QoL for children with ASD utilizing items from the *National Survey of Children's Health*. This assessment includes items in the following areas: family burden, family outings (# times child taken on outing in past week), family meals (# of days in a week family ate together), religious service attendance, work disruption (did parent quit job b/c of child in past 12 months), days of missing school (0–3 scale for how often child missed school), activity participation (yes/no to any organizational involvement in past 12 months), repeated a grade (since kindergarten, yes/no), independence (did child spend time caring for him/herself in past week, yes/no), and community service (any service/volunteer activity in past 12 months, yes/no).

The *Overall Outcome Rating* (Eaves and Ho 2008) is a measure specific to ASD and is based on parental responses to interview questions in three domains: occupation, friendships, and independent living. Scores result in classifications of very good outcome (e.g., high level of independence, some friends, and a job), fair outcome, poor outcome, and very poor outcome (e.g., needing high level of care, no friends, and no autonomy). Notably, this measure does not assess subjective feelings/wishes of the person with ASD but rather the parent's assessment of life outcomes in more objective terms. As such, the Overall Outcome Rating is not technically a measure of life quality which ordinarily implies a subjective component as well as measuring outcomes.

Finally, the *Quality of Life Autism-Friendly Environment* was developed by Billstedt et al. (2011) and is unique, in that it focuses specifically on environmental features that reflect good quality of life for individuals with ASD. This measure includes ratings (1 = very good to 5 = very poor) of the environment in five areas: staff/caregivers have autism-specific knowledge, environmental structure, implementation of

specific treatment/training plans, match of activities to capacity, and overall quality of life.

Current Knowledge and Future Directions

When considering the current state of knowledge for the assessment of quality of life for individuals with ASD, it is clear that researchers are interested in utilizing careful measurement of quality of life not only to benchmark outcomes but also to understand what factors relate to a high quality of life for this population. Enhancing our understanding of what influences quality of life, at what times, and for whom, is critical for informing interventions and services to improve outcomes for individuals with ASD; the field is now beginning to answer these questions. Below we review the emerging literature on QoL for individuals with ASD, highlighting the individual characteristics and contextual factors that are associated with quality of life across the life course.

Findings regarding the influence of the characteristics of the individual with ASD in predicting QoL are mixed, with differences emerging depending on the constructs of focus and the study design. One of the strongest findings is that better adaptive behavior has been repeatedly linked with higher QoL in both children and adults with ASD (Bishop-Fitzpatrick et al. 2016; Kamp-Becker et al. 2010; Kuhlthau et al. 2010; Hong et al. 2016). However, intellectual ability and autism severity have not been consistently related to QoL (Kuhlthau et al. 2010; Kamp-Becker et al. 2010; Renty and Roeyers 2006). For example, in a study of QoL for adults with ASD older than 50 years, limitations in adaptive behavior difficulties and not autism per se were associated with poorer QoL (Totsika et al. 2010). Billstedt et al. (2011) similarly found that IQ, residential placement, and occupational activities were not predictive of QoL when utilizing their measure of “autism-friendly” environmental QoL. In contrast, Eaves and Ho (2008) found that higher childhood IQ and lower autism severity was associated with better overall outcomes during young adulthood when employing their rating

scale of “good” outcomes. These discrepancies are likely related to differences in the emphasis for the various measures employed in these studies (e.g., on subjective vs. objective appraisals).

Contextual factors have also been related to QoL. For example, higher perceived informal support and fewer unmet formal support needs were associated with higher QoL for adults with HFA (Renty and Roeyers 2006). Similarly, having regular recreational activities was associated with higher QoL in a sample of young adults with ASD (Billstedt et al. 2011). In contrast, negative experiences such as bullying and high levels of perceived stress have been linked with poorer quality of life based on adult self-report (Hong et al. 2016). Even during adulthood, the family context also appears to play a role in QoL, with maternal warmth acting as a predictor of QoL, above and beyond individual characteristics (Bishop-Fitzpatrick et al. 2016). Taken together, these findings suggest that individuals as well as their contexts contribute to their overall experience of QoL, with individuals with better adaptive functioning and more supportive environments having more optimal outcomes.

As we consider the next phase of quality of life research for individuals with ASD, there are several important areas of study. First, to date much research on QoL has utilized samples of convenience, often with exclusively male or primarily male respondents and often with few participants from racial, ethnic, or cultural minority groups. Given that most conceptualizations of QoL include a core emphasis on subjective appraisal, which can be deeply shaped by cultures values, identity, etc., understanding the intersections of disability, gender, and culture in defining, measuring, and predicting QoL for all individuals with ASD will be an important area for future research. Second, it will be valuable for future work to incorporate a range of quality of life indicators as outcome measures in intervention and services research. To do this, researchers will need to draw upon measures that are reliable and well-validated for individuals across the full autism spectrum and from multiple cultural groups; this may require additional validation studies for the development of new measures. Including targeted and relevant

QoL indicators in intervention research will be particularly important for understanding which services and supports can be leveraged to promote successful adaptation and healthy outcomes for individuals with ASD. Third, little is known about quality of life for adults on the autism spectrum as they enter later life. New research on aging in ASD is greatly needed. This work may similarly necessitate new research into the assessment of QoL, as our current self-report and parent report instruments may become less relevant or difficult to employ as individuals and their family members age (e.g., siblings may not be as reliable of reporters as parents – this remains to be tested). Finally, considerations of family quality of life, in combination with individual quality of life, continue to be needed. As noted above, some past work has utilized family-level benchmarks in the assessment of QoL, but this was for families of children. Consistent with family systems theory and ecological systems theory, individuals with ASD and their family members have linked life course trajectories. Measuring perspectives on QoL for multiple family members (and the subsequent interplay of stress, coping, and well-being among all family members) is important not only during the time surrounding diagnosis and the childhood years, but across the life course. In conclusion, in order to ultimately inform the implementation of services and supports that promote and maintain a high quality of life for all individuals with ASD, future work will need to continue to develop and confirm careful measures of multiple dimensions of QoL (across a range of individuals). By continuing to test theoretically-informed models of the individual and contextual factors, we will gain new understanding in what influences positive outcomes in the areas that matter most for these individuals and their families.

See Also

- [Ecological Model of Autism](#)
- [Factors Affecting Outcomes](#)
- [Health Disparities](#)
- [Outcome Studies](#)

References and Reading

- Billstedt, E., Gillberg, I. C., & Gillberg, C. (2011). Aspects of quality of life in adults diagnosed with autism in childhood: A population-based study. *Autism, 15*, 7–20.
- Bishop-Fitzpatrick, L., Hong, J., Smith, L. E., Makuch, R. A., Greenberg, J. S., & Mailick, M. R. (2016). Characterizing objective quality of life and normative outcomes in adults with autism spectrum disorder: An exploratory latent class analysis. *Journal of Autism and Developmental Disorders, 46*, 2707–2719.
- Danckaerts, M., Sonuga-Barke, E. S., Banaschewski, T., Buitelaar, J., Döpfner, M., Hollis, C., et al. (2010). The quality of life of children with attention deficit/hyperactivity disorder: A systematic review. *European Child & Adolescent Psychiatry, 19*(2), 83–105.
- Eaves, L. C., & Ho, H. H. (2008). Young adult outcome of autism spectrum disorders. *Journal of Autism and Developmental Disorders, 38*, 739–747.
- Hong, J., Bishop-Fitzpatrick, L., Smith, L., Greenberg, J. S., & Mailick, M. R. (2016). Factors associated with subjective quality of life of adults with autism spectrum disorder: Self-report vs maternal reports. *Journal of Autism and Developmental Disorders, 46*, 1368–1378.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcomes for children with autism. *Journal of Child Psychology and Psychiatry, 45*, 212–229.
- Jennes-Coussens, M., Magill-Evans, J., Koning, C. (2006). The quality of life of young men with Asperger syndrome: A brief report. *Autism, 10*(4), 403–414.
- Kamp-Becker, I., Schoroder, J., Remschmidt, H., & Bachmann, C. J. (2010). Health-related quality of life in adolescents and young adults with high functioning autism-spectrum disorder. *German Medical Science. Psychosocial Medicine, 7*, 1–10. <https://doi.org/10.3205/psm000065>, URN: run:nbn:de:0183-psm0000657.
- Kraemer, B. R., McIntyre, L. L., & Blacher, J. (2003). Quality of life for young adults with mental retardation during transition. *Mental Retardation, 41*, 250–262.
- Kuhlthau, K., Orlich, F., Hall, T. A., Sikora, D., Kovacs, E. A., Delahaye, J., & Clemons, T. E. (2010). Health-related quality of life in children with autism spectrum disorders: Results from the Autism Treatment Network. *Journal of Autism and Developmental Disorders, 40*, 721–729.
- Lee, L.-C., Harrington, R. A., Louie, B. B., & Newschaffer, C. J. (2008). Children with autism: Quality of life and parental concerns. *Journal of Autism and Developmental Disorders, 38*, 1147–1160.
- Limbers, C. A., Heffer, R. W., & Varni, J. W. (2009). Health-related quality of life and cognitive functioning from the perspective of parents of school-aged children with Asperger's syndrome utilizing the PedsQL.

- Journal of Autism and Developmental Disorders*, 39, 1529–1541.
- McGovern, C. W., & Sigman, M. (2005). Continuity and change from early childhood to adolescence in autism. *Journal of Child Psychology and Psychiatry*, 46, 401–408.
- Power, M. J., Green, A. M., & The WHOQOL-Dis Group. (2010). Development of the WHOQOL disabilities module. *Quality of Life Research*, 19, 571–584.
- Renty, J., & Roeyers, H. (2006). Quality of life in high-functioning adults with autism spectrum disorder: The predictive value of disability and support characteristics. *Autism*, 10, 511–524.
- Ruble, L., & Dalrymple, N. (1996). An alternative view of outcomes in autism. *Focus on Autism and Other Developmental Disabilities*, 11, 3–14.
- Schalock, R. L., & Keith, K. (1993). *Quality of life questionnaire manual*. Worthington: IDS.
- Sheldrick, R. C., Neger, E. N., Shipman, D., & Perrin, E. C. (2011). Quality of life of adolescents with autism spectrum disorders: Concordance among adolescents' self-reports, parents' reports, and parents' proxy reports. *Quality of Life Research*, 21(1), 53–57. <https://doi.org/10.1007/s11136-011-9916-5>.
- Shipman, D. L., Sheldrick, C., & Perrin, E. C. (2011). Quality of life in adolescents with autism spectrum disorders: Reliability and validity of self-reports. *Journal of Developmental and Behavioral Pediatrics*, 32, 85–89.
- Totsika, V., Felce, D., Kerr, M., & Hastings, R. P. (2010). Behavior problems, psychiatric symptoms, and quality of life for older adults with intellectual disability with and without autism. *Journal of Autism and Developmental Disorders*, 40, 1171–1178.
- Varni, J. W., Seid, M., & Kurtin, P. S. (2001). PedsQL 4.0: Reliability and validity of the pediatric quality of life inventory version 4.0 generic core scales in healthy and patient populations. *Medical Care*, 39, 800–812.
- Varni, J. W., Seid, M., & Rode, C. A. (1999). The PedsQL: Measurement model for the pediatric quality of life inventory. *Medical Care*, 37, 126–139.
- Verdugo, R. L., Schalock, R. L., Keith, K. D., & Stancliffe, R. J. (2005). Quality of life and its measurement: Important principles and guidelines. *Journal of Intellectual Disability Research*, 49, 707–717.
- Wang, M., Schalock, R. L., Verdugo, M. A., & Jenaro, C. (2010). Examining the factor structure and hierarchical nature of the quality of life construct. *American Journal on Intellectual and Developmental Disabilities*, 115, 218–233.
- WHOQOL Group. (1998). Development of the World Health Organization WHOQOL-BREF quality of life assessment. *Psychological Medicine*, 28(3), 551–558.
- WHOQOL Group. (1995). The World Health Organization quality of life assessment (WHOQOL): Position paper from the World Health Organization. *Social Science & Medicine*, 41(10), 1403–1409.

Assessment of Basic Language and Learning Skills (ABLLS)

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Synonyms

ABLLS-R; Assessment, curriculum guide, and skills tracking

Description

The Assessment of Basic Language and Learning Skills-Revised (ABLLS-R) is a criterion-referenced assessment tool, curriculum planning guide, and tracking system of requisite skills needed for basic language and communication development, as well as skills needed to support learning in important academic, adaptive, and motor areas. ABLLS-R provides a comprehensive review of 544 skills from 25 skill areas. The primary purpose of the ABLLS-R is to assess and identify skills in language/communication and critical academic learning, self-help, and motor areas in order to optimize communication, social interaction, and ongoing learning in everyday situations. A secondary purpose is to assist in determining and prioritizing educational objectives for an individual child. The ABLLS-R assessment can identify skills currently in the child's repertoire, the level of skill attainment, and allows for the ongoing tracking of progression in skill development.

The ABLLS-R is comprised of two separate documents: the ABLLS-R Protocol and the ABLLS-R Scoring Instruction and IEP Development Guide. The ABLLS-R Protocol is the assessment document that provides a task analysis of behaviors and skills in four areas: basic learner skills, academic skills, self-help skills, and motor skills. The basic learner skills assessment is the largest area of consideration and is comprised of

15 subcomponent skill areas including aspects of language and communication, imitation, visual learning ability, play and leisure, social skill interaction, group behavior, responding, and classroom functioning. Academic skill assessment includes task analyses for reading, math, writing, and spelling. The self-help skills assessed include dressing, eating, grooming, and toileting. The motor skills assessment addresses strengths and weaknesses in gross and fine motor abilities. The ABLLS-R Scoring Instruction and IEP Development Guide provide important information regarding scoring, prioritizing educational objectives, and developing an Individualized Education Program (IEP).

The ABLLS-R Protocol

Each language, communication, or learning area included in the ABLLS-R Protocol contains a list of underlying behaviors needed for potential mastery of the domain or skill area. The underlying skills for the domain are identified and numbered as tasks; each task has a corresponding task objective. Individual tasks can be directly observed or assessed for the child, and the level of skill attainment for the task objective determined by a score ranking. The numbered task items for each skill domain are presented in a visual grid display containing eight columns. The first column is the numbered Task, followed by the Score column, the Task Name column, the Task Objective column, the Question column to prompt recall or direct observation of the specific task item being assessed, an Examples column, the Criteria column specifying the standards for scoring, and a Notes column to record related information about the level of performance by the child on the task item. For example, for Task A1 under the skill area *Cooperation and Reinforcer Effectiveness*, the Score column shows a range of scores from 0 to 2, the Task Name states: *Takes reinforcer when offered*, the Task Objective states: *“When offered a known reinforcing item or activity, the students will take/use the item or activity,”* while the Question column provides the following question to prompt a recall or directly observe the requisite behavior: *“When you hold out and offer*

a known reinforcer, will the student take the reinforcer?” The Example column provides an exemplar of the desired behavior: *“M & M taken and eaten.”* The Criteria column specifies the standard that must be met for each of the numbered scores in the Score column: *2 = takes within 3 seconds all the time, 1 = either not all the time or takes more than 3 seconds to respond.*

For each task item in the ABLLS-R, the Score column has four rows of numbers. The initial assessment of the task item is scored in the first row; subsequent updates are scored successively in the rows below. The numbers included in the Score column range from zero to the highest possible score, which varies by task item as 1, 2, or 4. Therefore, depending on the task, the Score column may have four rows, each row with the numbers 0 1; 0 1 2; or 0 1 2 3 4. A score of zero is given when the skill is either absent from the child’s repertoire or the child does not meet the lowest criterion indicated in the Criteria column.

The ABLLS-R Scoring Instructions and IEP Development Guide

The Scoring Instructions and IEP Development Guide provides direction on the initial scoring, how to resolve discrepancies between reports about a specific skill, how to ensure the stability of scores, and how to transfer the scores from the initial assessment (or subsequent update) to the corresponding grid box on the skills tracking system sheets. The skills tracking grid is used by the educational support team, including the parents, teachers, and clinical staff to help determine the skill areas of need for each child and to develop a specific individual educational plan (IEP) for the child. The Scoring Instructions and IEP Development Guide also provides information about how to prioritize the needs of the child in order to develop an optimal IEP.

Historical Background

The ABLLS-R is an assessment and intervention planning tool based on the theory of verbal

behavior and learning of B.F. Skinner. Skinner proposed that language or verbal behavior is a product of an operant stimulus-response-reinforcement/punishment paradigm with additional consideration of the importance of aspects of stimulus control and motivation. Skinner's analysis of verbal behavior centered on a set of functional units called verbal operants; each verbal operant consists of the response or verbal behavior and its controlling antecedents and consequences. Skinner described four verbal operants related to vocal communication including echoic, mand, tact, and intraverbal. Each of these functional units is included in the basic learner skills assessment as vocal imitation, requests, labeling, and intraverbal, respectively. In addition to the functional verbal units, other important areas of cognitive development and behavior that affect verbal learning are included in the basic learner skills, including behaviors associated with motivation, the ability to attend to complex stimuli, generalization, the ability to use language without prompting, termed "spontaneity," the ability to quickly apply a learned verbal behavior, called fluency, joint attention, learner readiness, and social skills development.

The ABLLS-R Protocol and ABLLS-R guide are an update to the Assessment of Basic Language and Learning Skills (ABLLS), first published in 1998. The recent edition incorporates additional skills and two additional areas of importance to verbal learning and communication for children with autism not included in the initial publication. The two new areas include assessment of skill in joint attention and fluency. Fluency is the ability of a child to quickly apply and use a learned skill in a variety of contexts. Additional new items for specific areas were identified from the research literature on autism for inclusion in the 2006 edition, including new items added to the assessment of motivation, response to complex stimuli, generalization, learner readiness, social skills development, and imitation.

Psychometric Data

The ABLLS-R is a criterion-referenced assessment tool. As such, the ABLLS-R is not designed

to compare the child's skill or achievement to a standardized peer group. No age norms, standardized scores, or group comparison data are provided. The ABLLS-R provides a skills tracking system that targets the skill development in each area of assessment. The results can be displayed on a grid that visually portrays the level of functioning in each skill area assessed. The visual grid display allows for the easy identification of areas of significant or moderate deficit for use in identifying needed areas and skills for intervention. The ABLLS-R can be completed by a parent, educator, other professional, or a combination of these. The assessment can be done over a number of days or weeks, and each skill area can be revisited as the child progresses. Each assessment area has a number of task items identified to assess skill development and mastery of the area. The number of task items varies for each area assessed. For example, the receptive language area contains 57 tasks associated with the domain, whereas the vocal imitation domain contains 20 task items.

Clinical Uses

The ABLLS-R is useful for parents, educators, and special education support staff members to assess and identify specific skills needed by a child who is nonverbal or has significant speech and language delays. The ABLLS-R Protocol provides a careful analysis of needed skills that should be the focus of intervention. There are limitations to the ABLLS-R in that it is not an exhaustive list of skills needed, nor does it provide instructions for teaching a desired skill. The ABLLS-R is not standardized; therefore, a child's performance in an area cannot be compared to an age peer group.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavior Analysis](#)
- [Behavior Modification](#)
- [Behavioral Objectives](#)
- [Behaviorism](#)

- ▶ [Criterion-Referenced Testing](#)
- ▶ [Generalization and Maintenance](#)
- ▶ [Imitation](#)
- ▶ [Motivation](#)
- ▶ [Stimulus](#)

References and Reading

- Lerman, D. C., Parten, M., Addison, L. R., Vorndran, C. M., Volkert, V. M., & Kodak, T. (2005). A methodology for assessing the functions of emerging speech in children with developmental disabilities. *Journal of Applied Behavior Analysis*, 38, 303–316.
- Partington, J. W. (2010). *The assessment of basic language and learning skills-revised*. Pleasant Hill: Behavior Analysts.
- Partington, J. W., & Sundberg, M. L. (1998). *The assessment of basic language and learning skills*. Danville: Behavior Analysts.
- Scattone, D., & Knight, K. R. (2008). Current trends in behavioral interventions for children with autism. *International Review of Neurobiology*, 72, 181–193.
- Skinner, B. F. (1957). *Verbal behavior*. New York: Appleton.

Assessment of Functional Living Skills (AFLS)

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Synonyms

[Activities of daily living \(ADLs\)](#); [Adaptive skills](#); [AFLS](#); [Criterion referenced assessment](#); [Functional living skills](#); [Self-help skills](#)

Description

The *Assessment of Functional Living Skills* (AFLS) is a criterion-referenced assessment used to measure essential skills throughout the lifespan for an individual (Partington and Mueller 2016). The AFLS is designed to assess the functional skill

repertoire of a learner across environments including home, school, community, and vocational sites. This comprehensive assessment is comprised of multiple modules that share consistent themes and foster overlapping goal development to increase an individual's learning and independence. For example, skills regarding meal preparation occur across environments throughout the assessment. These modules are represented in protocols which can be administered individually or in combination based on the current questions of the assessment team and characteristics of the individual being assessed (e.g., age, school placement, living arrangements). The ability to customize the assessment allows the evaluator to assess areas or domains that are pertinent to the individual at a given point in time (Partington and Mueller 2016).

The AFLS consists of a guide and six assessment protocols. The AFLS guide contains information about how to implement the assessment, what to teach and how to prompt skills, and task analyses to assist planning and programming. The protocols include assessments of *Basic Living Skills*, *Home Skills*, *Community Participation Skills*, *School Skills*, *Independent Living Skills*, and *Vocational Skills*. Collectively, these protocols assess of 1900 functional daily living skill in 66 skill areas (Partington and Mueller 2016).

AFLS Protocols. There are six assessment protocols included with the AFLS: *Basic Living Skills* (Partington and Mueller 2012a), *Community Participation Skills* (Partington and Mueller 2012b), *Home Skills* (Partington and Mueller 2012c), *School Skills* (Partington and Mueller 2013), *Independent Living Skills* (2015a), and *Vocational Skills* (2015b).

The *Basic Living Skills Assessment Protocol* (Partington and Mueller 2012a) evaluates eight general adaptive living skills that may be assessed across a variety of environments. The domains assessed include self-management, basic communication, dressing, toileting, grooming, bathing, health, safety and first aid, and nighttime routines.

The *Community Participation Assessment Protocol* (Partington and Mueller 2012b) assesses independence in a variety of community environments (e.g., grocery store, restaurants) across

eight domains including basic mobility, community knowledge, shopping, eat in public, money, phone, time, and social awareness and manners.

The *Home Skills Assessment Protocol* (Partington and Mueller 2012c) assesses eight essentials skill areas required for living in a home independently. The domains evaluated include meals at home, dishes, clothing and laundry, housekeeping and chores, household mechanics, leisure, kitchen, and cooking.

The *School Skills Assessment Protocol* (Partington and Mueller 2013) measures eight skill areas required to participate in school including routines and expectations including classroom mechanics, meals at school, routines and expectations, social skills, technology, common knowledge, core academics, and applied academics.

The *Independent Living Assessment Protocol* (Partington and Mueller 2015a) measures 16 skill areas necessary for an individual to live independent of supervision from others. The specific domains include organizational skills, self-care, maintenance and cleaning, mechanics and repairs, community travel, transportation, kitchen tools and appliances, food and meal planning, money management, independent shopping, personal management, safety, problem-solving, social interactions, and interpersonal relationships.

The *Vocational Skills Assessment Protocol* (Partington and Mueller 2015b) assesses 18 skill domains that are essential to acquiring a job as well as performing employment tasks including job search, interview, basic skills, co-worker relations, workplace safety, fixed activity skills, custodial and cleaning, laundry, retail, support personnel, office skills, computer skills, restaurant skills, restaurant kitchen, warehouse, tools, trades and construction, and landscaping.

Each protocol booklet is structured identically including a warning about learner safety when testing skills in potentially dangerous environments (e.g., crossing a street). The protocol booklets provide a brief overall of the *AFLS* assessment, information about the specific protocol, tracking grid to monitor progress for each

skill domain, and modules formatted in tables for each of the skill domains (Partington and Mueller 2016). For ease of use, each module is organized by broad skill domains, which are assigned a reference letter(s). For example, in the *Home Skills Assessment Protocol*, the broad skill area *Meals at Home*, is assigned the letters *MH*. Each individual skill is assigned a reference number that is combined with the domain reference letter(s). For example, the first skill in *Meals at Home* is referenced as item *MH1* (Partington and Mueller 2012c). The skill domain modules are organized similarly with the same headings. The first heading is *Task* which is the reference letter (s) and number for each item. The *Score* column is provided for the assessor to collect data by circling the number that corresponds to the learner's performance. The scoring numbers are presented horizontally in a row with either 0 1 2 or 0 1 2 3 4, which directly correspond to the specific score criteria in the *Criteria* column. The row of numbers is presented vertically four times so that each protocol booklet may be used across four separate scoring sessions. The next header, *Task Objective*, specifies the exact objective the individual must perform in order to receive a score. For example, the task objective for *MH1* is "Learner will identify finger foods versus non-finger foods" (Partington and Mueller 2012c, p. 1). The *Question* presents the objective in a question format which may be used when interviewing caregivers regarding the individual's ability to perform the skill. The *Example* header provides a specific scenario in which the individual should perform the skill. The *Criteria* header provides information regarding the number of targets, prompt levels, and other specific information needed in order to determine the overall quality of the individual's performance of the skill and directly corresponds to the scoring numbers in the *Score* column. A *Comment* column is provided in order to the evaluation to write in data collection and/or other anecdotal information that is important for the testing (Partington and Mueller 2016).

AFLS Guide. The *AFLS Guide* (Partington and Mueller 2016) is the user's manual for the

AFLS, and the information contained in the guide can be applied across all six protocols. The *AFLS Guide* opens with a statement regarding learner safety during across the testing period as many skills assessed may occur in environments in which safety should be a priority (e.g., kitchens, work areas with machinery and tools). Additional information found within the *AFLS Guide* includes an overview of the importance of teaching functional living skills, a review of specific terminology used within the *AFLS*, and a general overview of the *AFLS*. The *AFLS Guide* also contains implementation information for the assessors to follow during assessment periods. Further information located in the guide provides directions for determining which skills to teach, teaching methods, and recommendations for building a task analysis based on the outcomes of the assessment (Partington and Mueller 2016).

Historical Background

Prior to the development of the *AFLS*, James W. Partington and Mark L. Sundberg developed the *Assessment of Behavioral Language and Learning* (*ABLLS*; 1998) based on B. F. Skinner's *Verbal Behavior* (1957). Skinner described a stimulus-response contingency with regard to the development of verbal behavior, identified the speaker-listener relationship, and differentiated a behavioral approach to the development of communication that distinctly differs from traditional models of language development (1957). The *ABLS* (Partington and Sundberg 1998) and subsequently the *Assessment of Behavioral Language and Learning-Revised* (*ABLLS-R*; Partington 2010) established the first assessment for evaluating the basic language skills, social and group skills, and self-help skills for young learners on the autism spectrum and other developmental delays. The primary purpose of the *ABLLS-R* is to provide parents, teachers, and other caregivers a comprehensive method for assessing and tracking early developmental skills in order to guide the development of learning

objectives. The *ABLLS-R* assess 25 developmental domains that typically developing children master at approximately 4–5 years of age. While the *ABLLS-R* provides a foundational start for teaching language skills and learning readiness skills, the *ABLLS-R* is not a comprehensive assessment of independent living skills for young children and adults. Teaching adaptive and independent living skills across home, school, and the community is an essential component of educational plans for individuals with developmental delays. Therefore, Partington and Mueller developed the *AFLS* in order to meet the need for continued assessment of life skills across the age continuum (Partington Behavior Analysts 2016).

Psychometric Data

The *AFLS* is a criterion-referenced assessment and does not provide the standardized scores that accompany standardized assessments. As a criterion-referenced assessment, the *AFLS* provides information regarding the ability of the individual to perform specific skills identified as compared to the learner's previous performance across repeated testing (Powers et al. 2014). Each *AFLS* protocol is scored in the same manner. For every task, evaluators circle the score that corresponds to the learner's current level of performance based on the criteria specified in the protocol. A score of zero indicates a task the learner is unable to perform or does not meet the criterion for the lowest score associated with that task (Partington and Mueller 2016). Skills that may never be applicable or may not be applicable for that learner at the time of the assessment may be scored as not applicable. Specific information regarding the skills can be noted in the comment section for each task. The scores are represented in a tracking grid that corresponds to the specific protocol assessed. The protocol allows for the assessment to be updated three times for each learner. The tracking grid should be marked differentially, using a different color for updates. The use of different colors allows for assessors,

parents, school personnel, and others to visually analyze the learner's progress and skill growth across subsequent reevaluations (Partington and Mueller 2016).

Clinical Uses

The *AFLS* measures and tracks a learner's current level of functioning across a variety of skills that are essential for living and participating in the home, school, community, and vocational settings (Partington and Mueller 2016). The *AFLS* may be completed by school professionals, caregivers, and community service providers to assess a learner's level of independent functioning across a variety of settings. The three primary sources of information required to complete the *AFLS* assessment include interviews of caregivers, direct observation/testing, and/or historical data. Assessors gather information from caretakers who regularly interact, directly observe, and formally present tasks to the learner (Partington and Mueller 2016).

See Also

- Activities of Daily Living (ADLs)
- Adaptive Skills
- Assessment of Basic Language and Learning Skills (ABLLS)
- Criterion Referenced Assessment
- Functional Living Skills
- Skinner's Verbal Behavior
- Verbal Behavior

References and Reading

- Partington, J. W. (2010). *Assessment of basic language and learning skills-revised (ABLLS-R)*. Pleasant Hill: Behavior Analyst.
- Partington Behavior Analysts. (2016). The ABLLS-R and the AFLS: Assessments, curricula and skills tracking systems that work together to guide programming for individuals with autism or other developmental delays. Retrieved from <https://partingtonbehavioranalysts.com/products/ablls-rafls-compatibility/>

- Partington, J. W., & Mueller, M. M. (2012a). *The assessment of functional living skills: Basic living skills assessment protocol* (1.1 ed.). Walnut Creek/Marietta: Behavior Analysts/Stimulus Publications.
- Partington, J. W., & Mueller, M. M. (2012b). *The assessment of functional living skills: Community participation skills assessment protocol* (1.1 ed.). Pleasant Hill/Marietta: Behavior Analysts/Stimulus Publication.
- Partington, J. W., & Mueller, M. M. (2012c). *The assessment of functional living skills: Home skills assessment protocol* (1.1 ed.). Walnut Creek/Marietta: Behavior Analysts/Stimulus Publications.
- Partington, J. W., & Mueller, M. M. (2013). *The assessment of functional living skills: School skills assessment protocol* (1.0 ed.). Walnut Creek/Marietta: Behavior Analysts/Stimulus Publications.
- Partington, J. W., & Mueller, M. M. (2015a). *The assessment of functional living skills: Independent living skills assessment protocol* (1.0 ed.). Pleasant Hill/Marietta: Behavior Analysts/Stimulus Publications.
- Partington, J. W., & Mueller, M. M. (2015b). *The assessment of functional living skills: Vocational skills assessment protocol* (1.0 ed.). Pleasant Hill/Marietta: Behavior Analysts/Stimulus Publications.
- Partington, J. W., & Mueller, M. M. (2016). *Assessment of functional living skills guide* (1.2 ed.). Walnut Creek/Marietta: Behavior Analysts/Stimulus Publications.
- Partington, J. W., & Sundberg, M. L. (1998). *Assessment of basic language and learning skills*. Danville: Behavior Analysts.
- Powers, M. D., Palmieri, M. J., Egan, S. M., Rohrer, J. L., Nulty, E. C., & Forte, S. (2014). Behavioral assessment of individuals with autism: Current practice and future directions. In F. R. Volkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. II, 4th ed., pp. 695–736). Hoboken: Wiley.
- Skinner, B. F. (1957). *Verbal behavior*. Ann Arbor: Copley Custom Textbooks, XanEdu Publishing.

Assessment, Curriculum Guide, and Skills Tracking

- Assessment of Basic Language and Learning Skills (ABLLS)

Assistant

- Paraprofessional

Assistive Devices

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Synonyms

[Augmentative and alternative communication \(AAC\) device](#)

Definition

Assistive devices are aided communication systems used in the area of augmentative and alternative communication (AAC). Aided systems are ones that require something other than the individuals' body to communicate. That "something" could be picture symbols, written words, or a high-tech speech-generating device. Conversely, unaided systems are those that do not require anything separate from one's own body to communicate. Essentially, gestures, body language, and sign language are examples of unaided systems. Examples of assistive devices are individual picture symbols, picture communication boards, mid-tech communication aids such as the MessageMate (sold by Words+), and high-tech communication devices such as the VMax (sold by DynaVox). See Beukelman and Mirenda (2005) for a detailed description of assistive devices and their implementation.

See Also

- ▶ [Alternative Communication](#)
- ▶ [Augmentative and Assistive Technology](#)
- ▶ [Communication Board](#)
- ▶ [Voice Output Communication Aids](#)

References and Reading

Beukelman, D. R., & Mirenda, P. (2005). *Augmentative and alternative communication: Supporting children and adults with complex communication needs*. Baltimore: Brooks Publishing.

Associate

- ▶ [Paraprofessional](#)

Association for Retarded Citizens (Arc)

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Major Areas or Mission Statement

The mission of The Arc is to "promote and protect the human rights of people with intellectual and developmental disabilities and actively supports their full inclusion and participation in the community throughout their lifetimes."

Landmark Contributions

The Arc was founded in 1950 as the National Association of Parents and Friends of Mentally Retarded Children. It initially focused on changing perceptions about I/DD (then referred to as mental retardation), from the assumption that most persons with I/DD necessitated institutionalization, to the recognition of their potential and their rights to employment and education. During the 1960s and 1970s, The Arc advocated for key legislation, including the passage of Public Law 94-142 (guaranteeing a free and appropriate education to children with disabilities) and legislation to create the Supplemental Security Income program to support persons with

disabilities. Since that time, The Arc has contributed significantly to efforts to pass many other laws contributing to increased community-based options for living (e.g., the creation of Medicaid-funded home and community-based waivers) and working (e.g., incentives for employers who hire persons with disabilities), other initiatives related to health (e.g., Medicaid's Early and Periodic Screening, Diagnosis, and Treatment program), and the landmark Americans with Disabilities Act.

The Arc has also contributed to the scientific understanding of I/DD. In the 1960s, The Arc helped to first expose links between lead poisoning and brain damage in children. Research and other work funded by The Arc in the 1970s helped to identify the treatment for phenylketonuria (PKU), to define Fetal Alcohol Spectrum Disorder, and to first suggest infant undernutrition as a cause of developmental disabilities. Since that time, The Arc also began to support the dissemination of scientific findings through its sponsorship or organization of key summits and publications.

Major Activities

The Arc describes itself as “the largest national community-based organization advocating for and serving people with intellectual and developmental disabilities and their families. We encompass all ages and all spectrums from autism, Down syndrome, Fragile X and various other developmental disabilities.” The Arc actively contributes to the development of public policies at the local, state, and federal level.

Education and Activism is a core activity of The Arc at the local, state, and federal level levels, in support of legislation for civil rights, education, employment, health care, housing, and other areas of interest. With funding from Administration on Developmental Disabilities, The Arc recently established the National Autism Resource and Information Center as a “central point of access to high-quality and evidence-based resources and information for individuals with Autism Spectrum Disorder (ASD) and other developmental disabilities, their families, professionals, and other targeted key stakeholders, including underserved and unserved.”

A broad range of supports and services are also offered by individual chapters of The Arc, including information and referral, advocacy and self-advocacy around a broad range of issues, residential support, family support, employment programs, and leisure and recreational programs.

In 2011, The Arc had more than 700 state and local chapters, and more than 140,000 members across the United States. Members come from all walks of life, though most are family members or persons with intellectual and developmental disabilities (I/DD).

See Also

- [Advocacy](#)
- [Disability](#)
- [Intellectual Disability](#)
- [Mental Retardation](#)

Associative Learning

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Definition

Associative learning refers to the process in which a new response is paired with a particular stimulus or response that already exists within the learner's repertoire or experiences; it is based on the ideas that experiences reinforce one another and can be linked to enhance the learning process. Associative learning responses are “associative” in that the responses being learned are associated with previous responses or stimuli, either conditioned or unconditioned. Associative learning, like classical conditioning, involves pairing an unconditioned stimulus (which reflexively produces a response) with another stimulus that is neutral. Over time, the pairing results in the reliable emission of a response that was previously not consistently emitted.

See Also

- [Classical Conditioning](#)
- [Operant Conditioning](#)

References and Reading

- Moran, D., & Malott, R. (2004). *Evidence based educational methods*. New York: Elsevier Academic Press.
- Shanks, D. (1995). *The psychology of associative learning (problems in the behavioral sciences)*. Cambridge: Cambridge University Press.
- Whitehead, W., Lurie, E., & Blackwell, B. (1976). Classical conditioning of decreases in human systolic blood pressure. *Journal of Applied Behavior Analysis*, 9, 153–157.

Ataxia

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Synonyms

[Dystaxia](#)

Definition

Unsteady gait. The term ataxia refers to lack of coordination of motor movements. Ataxia typically arises due to some dysfunction within the central nervous system. It has many different causes, e.g., it can be a sign of dysfunction in the cerebellum (where it usually would be associated with various other difficulties). Other causes can relate to effects of drugs or other substances and problems with the vestibular system and other parts of the brain. Medications given to control seizures are a common cause of ataxia; abuse of some substances (e.g., alcohol) may also lead to ataxia.

Ataxia can also arise as a result of a deficiency in vitamin B₁₂, exposure to certain toxic agents, and so forth. Problems in the peripheral nervous system can also be associated with ataxia. Certain inherited (genetic) disorders can also lead to ataxia.

In autism, problems in posture and gait are relatively common. Problems with clinically significant ataxia often are associated with drug treatments (e.g., for seizures). In Rett's disorder, ataxia and other movement problems are quite common and associated with the underlying pathophysiology of the condition.

Treatment varies depending on the cause. Physical therapy can be useful in some cases, and some drug treatments have also been proposed.

See Also

- [Epilepsy](#)
- [Physical and Neurological Examination](#)
- [Rett's Disorder](#)

References and Reading

- Rinehart, N. J., Tonge, B. J., et al. (2006). Gait function in newly diagnosed children with autism: Cerebellar and basal ganglia related motor disorder. *Developmental Medicine and Child Neurology*, 48(10), 819–824.
- Tachi, N., Kozuka, N., et al. (2000). Hereditary cerebellar ataxia with peripheral neuropathy and mental retardation. *European Neurology*, 43(2), 82–87.
- VanAcker, R., Loncola, J. A., et al. (2005). Rett syndrome: A pervasive developmental disorder. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 126–164). Hoboken: Wiley.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to Autism: What every parent, teacher and family member needs to know*. Hoboken: Wiley.

Ativan

- [Lorazepam](#)

Atomoxetine

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Synonyms

[Strattera \(TM\)](#)

Definition

Atomoxetine is a drug approved for the treatment of attention-deficit hyperactivity disorder (ADHD). This compound is manufactured, marketed, and sold in the United States under the brand name Strattera by Eli Lilly and Company. Generics of atomoxetine are sold in other countries.

Clinical Use

Atomoxetine is approved for use in children, adolescents, and adults; however, its efficacy has not been studied in children under 6 years old. Its advantage over stimulants for the treatment of ADHD is that it has less abuse potential than stimulants (Wee and Woolverton 2004), is not scheduled as a controlled substance, and has shown in clinical trials to offer 24-h coverage of symptoms associated with ADHD in adults and children (May and Kratochvil 2010).

Therapeutic effects of atomoxetine may take a week to be felt and an adequate trial may be up to 8 weeks (May and Kratochvil 2010). Many people respond to atomoxetine who do not respond to stimulants (May & Kratochvil). Atomoxetine may be preferred over amphetamine-based stimulants in patients with psychiatric disorders, those who cannot tolerate stimulants, and those with a substance misuse recurring history. Therapy is

usually initiated by gradually increasing the dose to minimize side effects.

Arnold et al. in 2006 enrolled 16 pediatric patients with pervasive developmental disorder (PDD) in a double-blind, placebo-controlled, crossover study and found that 57% of the pediatric patients responded to atomoxetine (Arnold et al. 2006).

Mechanism of Action

Atomoxetine acts by selectively blocking the norepinephrine transporter. The norepinephrine transporter takes up norepinephrine as well as dopamine in the prefrontal cortex, with little to no activity at the other neuronal reuptake pumps or receptor sites. Blocking the norepinephrine transporter increases norepinephrine as well as dopamine levels; this mechanism was studied in rat prefrontal cortices (Bymaster et al. 2002). Atomoxetine has been shown to improve prefrontal cortices in normal adults with ADHD (Chamberlain et al. 2006, 2007).

Dosing

Once- or twice-daily atomoxetine was effective in the short-term treatment of ADHD in children and adolescents, as observed in several placebo-controlled trials (May and Kratochvil 2010), 0.5–1.4 mg to kilogram of body weight (May & Kratochvil).

Side Effects

The side effects include dry mouth, tiredness, irritability, nausea, decreased appetite, constipation, dizziness, sweating, dysuria, sexual problems, increased obsessive behavior, weight changes, palpitations, and increases in heart rate and blood pressure (Chamberlain et al. 2006).

Two confirmed cases of liver injury have been reported by Eli Lilly and Company out of

approximately two million prescriptions written. In both cases, upon discontinuation of atomoxetine, patients' liver functions returned to normal (Chamberlain et al. 2006).

There is a black box warning for increased risk of suicidality in children and adolescent with ADHD especially during the first month of treatment.

Pharmacology

Pharmacokinetic data: bioavailability, 63–94%; protein binding (primarily albumin), 40%; metabolism, hepatic, via CYP2C19 (minor), 2D6 (major) (Garnock-Jones and Keating 2009).

Bioavailability: 63% in extensive metabolizers; 94% in poor metabolizers (Garnock-Jones and Keating 2009).

Half-life elimination: atomoxetine: 5 h (up to 24 h in poor metabolizers); active metabolites, 4-hydroxyatomoxetine: 6–8 h; N-desmethylatomoxetine: 6–8 h (34–40 h in poor metabolizers) (Chalon et al. 2003; Witcher et al. 2003).

Time to peak, plasma: 1–2 h (Chamberlain et al. 2006).

Excretion: Urine (80%, as conjugated 4-hydroxy metabolite); feces (17%) (Chamberlain et al. 2006).

See Also

► [Attention Deficit/Hyperactivity Disorder](#)

References and Reading

- Arnold, L. E., Aman, M. G., Cook, A. M., Witwer, A. N., Hall, K. L., Thompson, S., et al. (2006). Atomoxetine for hyperactivity in autism spectrum disorders: placebo-controlled crossover pilot trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45(10), 1196–1205.
- Bymaster, F. P., Katner, J. S., Nelson, D. L., Hemrick-Luecke, S. K., Threlkeld, P. G., Heiligenstein, J. H., et al. (2002). Atomoxetine increases extracellular levels of norepinephrine and dopamine in prefrontal cortex of rat: a potential mechanism for efficacy in attention deficit/hyperactivity disorder. *Neuropsychopharmacology*, 27(5), 699–711.
- Chalon, S. A., Desager, J. P., DeSante, K. A., et al. (2003). Effect of hepatic impairment on the pharmacokinetics of atomoxetine and metabolites. *Clinical Pharmacology and Therapeutics*, 73, 178–191.
- Chamberlain, S. R., Müller, U., Blackwell, A. D., Clark, L., Robbins, T. W., & Sahakian, B. J. (2006). Neurochemical modulation of response inhibition and probabilistic learning in humans. *Science*, 311(5762), 861–863.
- Chamberlain, S. R., Del Campo, N., Dowson, J., Müller, U., Clark, L., Robbins, T. W., et al. (2007). Atomoxetine improved response inhibition in adults with Attention Deficit/Hyperactivity Disorder. *Biological Psychiatry*, 62(9), 977–984.
- Garnock-Jones, K. P., & Keating, G. M. (2009). Atomoxetine: a review of its use in attention-deficit hyperactivity disorder in children and adolescents. *Paediatric Drugs*, 11(3), 203–226.
- May, D. E., & Kratochvil, C. J. (2010). Attention-deficit hyperactivity disorder: recent advances in paediatric pharmacotherapy. *Drugs*, 70(1), 15–40.
- Wee, S., & Woolverton, W. L. (2004). Evaluation of the reinforcing effects of atomoxetine in monkeys: Comparison to methylphenidate and desipramine. *Drug and Alcohol Dependence*, 75(3), 271–276.
- Witcher, J., Long, A., Smith, B., et al. (2003). Atomoxetine pharmacokinetics in children and adolescents with attention deficit hyperactivity disorder. *Journal of Child and Adolescent Psychopharmacology*, 13, 53–63.

Attachment

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Definition

According to Bowlby (1969/1982), attachment constitutes the first affective bond that the child forms with the primary caregiver. Bowlby, drawing from object relations theory, suggested that in the first year of life it is in the infant's interest to seek out proximity to the attachment figure when under stress (Bretherton 1985). Thus, to foster proximity, the child and mother are involved in many interactions. According to Bowlby, the

responsiveness of the mother to the child's signals will determine the nature of their relationship, which the child will internalize via working models. The working model comprises the representation of the child's knowledge about the world and about significant persons in the world, including the self (Bowlby 1969/1982). These models are useful in guiding behaviors in new situations. Furthermore, they affect the quality of the child's future relationships throughout life (Sroufe and Fleeson 1986). Once working models are established, they tend to remain stable. The "marker behaviors" of attachment can change throughout stages of child development (e.g., physical proximity or checking in with mother in the first years, verbal negotiation at age 3 or 4). However, the construction of the attachment patterns (secure or insecure) tends to remain stable (Bretherton 1985).

The perception of attachment as an affective bond means that the child is forming long enduring ties with noninterchangeable "significant other/s" (Ainsworth 1989). Thus, the infant's initial ability to differentiate between people and inanimate objects and then the capacity to distinguish the primary caregiver from other individuals are precursors to the ability to form attachment. On the basis of these differentiations, the child directs more proximity-seeking behaviors toward the primary caregiver, shows more distress in the caregiver's absence, and calms down in the caregiver's presence. Behaviors maintaining proximity during infancy include active efforts to stay close to the mother (e.g., approaching, following, clinging) and signaling behaviors (e.g., smiling, crying, calling) (Ainsworth et al. 1978).

A child might be able to differentiate between the mother and other individuals yet nevertheless form an insecure attachment with the mother. Thus, the quality of attachment also needs to be considered. The "strange situation" paradigm, a series of eight episodes in which the infant is given the opportunity to interact with an unfamiliar adult in the mother's presence and absence, was developed to identify individual differences in children's quality of attachment (Ainsworth et al. 1978). The child's reactions to the separation and reunion with the mother during the "strange

situation" episodes enable classification of children as either securely attached to their mothers (e.g., showing distress at separation and attempting to reestablish interaction at reunion) or as insecurely attached. Insecure attachment can be "avoidant" (e.g., showing indifference at separation and actively avoiding parents at reunion), "resistant/ambivalent" (e.g., presenting high distress at separation and responding to reunion with mixed feelings of rejection and approaching), or as later identified by Main and Solomon (1986, 1990), "insecure/disorganized" (e.g., lacking observable goals, intentions, or explanations in the parent's presence, such as stereotypical movements or misdirected and incomplete expressions).

See Also

► [Reactive Attachment Disorder](#)

References and Reading

- Ainsworth, M. S. D. (1989). Attachment beyond infancy. *American Psychologist*, 44, 709–716.
- Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation*. Hillsdale: Erlbaum.
- Bowlby, J. (1969/1982). *Attachment and loss* (Attachment, Vol. 1). New York: Basic Books.
- Bretherton, I. (1985). Attachment theory: Retrospective and prospective. In I. Bretherton & E. Watres (Eds.), *Growing points of attachment theory and research* (Monographs of the Society for Research in Child Development, 50 (1–2, Serial No. 209)). Chicago: University of Chicago Press for the Society for Research in Child Development.
- Main, M., & Solomon, J. (1986). Discovery of an insecure – Disorganized/disoriented attachment pattern. In T. Brazelton & M. W. Yogman (Eds.), *Affective development in infancy* (pp. 95–124). Norwood: Ablex.
- Main, M., & Solomon, J. (1990). Procedures for identifying infants as disorganized/disoriented during the Ainsworth strange situation. In M. T. Greenberg, D. Cicchetti, & E. M. Cummings (Eds.), *Attachment in the preschool years: Theory, research and intervention* (pp. 134–146). Chicago: University of Chicago Press.
- Sroufe, L. A., & Fleeson, J. (1986). Attachment and the construction of relationships. In W. W. Hartup & Z. Rubin (Eds.), *Relationships and development* (pp. 51–71). Hillsdale: Erlbaum.

Attachment Disorder

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Synonyms

Disinhibited attachment disorder; Disinhibited social engagement disorder; Emotionally withdrawn/inhibited attachment disorder; Indiscriminate friendliness; Indiscriminate sociability; Indiscriminate social behavior; Indiscriminately social/disinhibited attachment disorder; Reactive attachment disorder

Short Description or Definition

Attachment disorders describe aberrant social behaviors in young children, particularly regarding how and from whom they seek comfort, support, nurturance, and protection. Two major types have been defined, an emotionally withdrawn/inhibited type, in which the child lacks social and emotional responses, and an indiscriminately social/disinhibited type, in which the child lacks social wariness about unfamiliar adults. The former includes the absence of attachment behaviors directed at caregivers, while the latter refers to social behaviors directed at unfamiliar adults.

Categorization

DSM-IV-TR (American Psychiatric Association [APA] 2000) describes two subtypes of reactive attachment disorder, an emotionally withdrawn/inhibited type and an indiscriminately social/disinhibited type, whereas ICD-10 (World Health Organization [WHO] 1992) defines “reactive attachment disorder” as the emotionally withdrawn/inhibited type and “disinhibited attachment

disorder” as the indiscriminately social/disinhibited type. Recent reviews have concluded that these types actually represent two distinct disorders that share primarily the conditions of risk in which they each occur (Rutter et al. 2009; Zeanah and Gleason 2010). For this reason, DSM 5, in keeping with ICD-10, is proposing to define two distinct disorders.

The emotionally withdrawn/inhibited type of reactive attachment disorder is characterized by a child who appears emotionally unresponsive with limited or no positive affect. These children do not have much of an interest in interaction with any adult, and social reciprocity is minimal or absent. This overt apathy toward relationships extends to the attachment relationship. These children do not exhibit consistent or robustly developed attachment behaviors, that is, behaviors that promote proximity to preferred adults. Children with emotionally withdrawn/inhibited RAD fail to check in with familiar adults, and they neither seek nor accept comfort from caregivers in times of emotional need.

In contrast, children with the indiscriminately social/disinhibited type of reactive attachment disorder are overly familiar, even with adults they do not know. They can be intrusive and lack appropriate social and physical boundaries, as well as emotionally “over bright” and attention seeking. They lack reticence around unfamiliar adults, instead approaching and engaging with anyone. In fact, they are notably willing to “go off” with complete strangers. They may be aggressive as well as uncomfortably “friendly.”

Epidemiology

These disorders are believed to be rare, even in clinic-referred children, but epidemiological data are scarce. In a quasi-epidemiologic study (Egger et al. 2006), for example, 300 children were recruited from pediatric clinics, but there were no cases of reactive attachment disorder identified. A cohort study by Skovgaard showed a prevalence of 0.9% of attachment disorders. In a study of young children with a history of varying

amounts of institutional rearing (range of 6–54 months), only 10% had a diagnosis of RAD at 54 months of age, though a majority of children showed subthreshold signs of the disorder (Gleason et al. 2011). Thus, although signs of the disorder may be evident in children with histories of maltreatment (Pears et al. 2010; Oosterman and Schuengel 2007; Zeanah et al. 2004) or institutional rearing (Chisholm 1998; Gleason et al. 2011; Rutter et al. 2007; Tizard and Rees 1975; Zeanah et al. 2005), children meeting full criteria for the disorder are rare, especially if they are living in families.

Natural History, Prognostic Factors, and Outcomes

Since most children who are severely neglected or raised in deprived institutions do not develop attachment disorders, there must be vulnerability factors that predispose children to one or the other types of RAD. Little progress to date in elucidating those factors has been made. A related question is why children who share risk factors of neglect and deprivation develop such distinctively different phenotypes – one withdrawn and unresponsive and the other overly bright emotionally and attention seeking. Again, there have been no published studies to date that have addressed these issues. Temperamental differences or genetic polymorphisms are potential vulnerability factors.

One of the hallmark features of attachment disorder is that they are supposed to be responsive to changes in the caregiving environment. This is quite clear in the case of emotionally withdrawn/inhibited RAD, but less clear in the case of indiscriminately social/disinhibited RAD as illustrated in the following two examples.

Case 1

Cade is a 30-month-old boy who spent the first 20 months of his life in a run-down apartment. His mother had a serious substance use disorder, which became worse after he turned a year old.

Only rarely did she have someone watch him. Instead, she often left him on the floor in the kitchen, with a bowl of dry cereal, and a pet gate in place so that he would not leave the kitchen. A neighbor called the police, and Cade was taken into State's custody.

Cade was found to be malnourished, but he quickly recovered physically. Most striking about his behavior in the foster home was his eagerness to be held by everyone. He immediately approached any adult, and he showed no preferences nor any reticence. He seemed starved for attention, and his affect was overly bright. Because his mother surrendered her rights to him, Cade was adopted when he was 28 months old. With his adoptive parents, he continued to display intrusive, affectionate behavior. They were concerned because he continued to go readily to any stranger.

They were counseled to restrict his contact with adults other than the two of them for several months. Following this, he began to seek comfort preferentially from his foster parents when he was distressed and to protest when they left him. They felt that he became increasingly affectionate with them. Despite these gains, after several months when they began to take him into public, he still showed occasional lack of reticence with strangers, and they feared that he would be willing to go off with one.

Case 2

Zoe was taken into State's custody when she was 13 months old because of neglect and concerns about her safety. Zoe's mother had been displaying increasingly bizarre behavior, according to the records, and she was later diagnosed with schizophrenia. She wanted to protect and care for Zoe, so she put her in a crib in the closet for hours at a time "to keep her safe." She did not like Zoe being around people because she was afraid of their germs. She refused to take Zoe to the physician because of all of the "sick people" there. Zoe was removed by Child Protective Services and placed in her paternal grandmother's care. She spent her initial days there staring at

the wall or idly touching toys. Her grandmother described her blank stare as “unnerving.” Zoe was easily frustrated and difficult to console. At times, she smiled but her smile had a frozen, empty quality and did not convey any sense of authentic positive affect. Her grandmother described Zoe as “stiff and awkward to hold.” Zoe seemed to be fine as long as she was left alone.

After a few weeks of being in her grandmother’s care, Zoe improved substantially. She began to interact reciprocally with her grandmother, and she ran to her when she wanted comfort. She was easily consoled, but only by her grandmother. She clung tightly to her grandmother when a stranger came into the room. Her frozen smile disappeared, and she readily conveyed moments of genuine enjoyment, though she remained irritable and easily frustrated for several more months.

In studies of children adopted out of institutions, there have been no children identified with emotionally withdrawn/inhibited RAD in follow-ups months to years later (Chisholm 1998; Rutter et al. 2007; Hodges and Tizard 1989). Similarly, in the Bucharest Early Intervention Project (BEIP), children removed from institutions and placed in foster care had a strong and early reduction in signs of emotionally withdrawn/inhibited RAD compared to children who remained institutionalized (Smyke et al. 2012). In other words, once children are removed from socially depriving environments of institutions and are placed with families, signs of emotionally withdrawn/inhibited RAD disappear. On the other hand, for children who remain in institutions, signs of emotionally withdrawn/inhibited RAD are moderately stable over time (Gleason et al. 2011). Thus, when being raised in environments in which opportunities to form selective attachments are limited, children may manifest signs of emotionally withdrawn/inhibited RAD. However, they tend to recover when placed in more normative environments.

The findings regarding the course of indiscriminately social/disinhibited RAD are somewhat different. The stability of indiscriminate behavior is modest to moderate, in both institutionalized (Gleason et al. 2011) and formerly institutionalized

children (Rutter et al. 2007). Both short-term (Chisholm 1998; Gleason et al. 2011) and long-term (Hodges and Tizard 1989; Rutter et al. 2007) longitudinal studies have shown that indiscriminate behavior, once present, is quite persistent in a minority of children who were raised in institutions, even if they are later adopted or placed with families. Indiscriminate behavior that persisted into adolescence was associated with peer problems. Furthermore, in the Bucharest Early Intervention Project, reduction in signs of indiscriminate/disinhibited RAD was less powerful than the reduction in signs of emotionally withdrawn/inhibited RAD following placement in families (Gleason et al. 2011).

Prognostic factors are not well delineated among children with attachment disorders. Generally, the sooner that a young child can be placed within a loving environment the better, but the long-term outcomes of children diagnosed in early childhood with attachment disorders is not well delineated. Signs of both emotionally withdrawn/inhibited and indiscriminately social/disinhibited RAD in children less than 30 months of age were predictive of overall psychiatric impairment at 54 months (Gleason et al. 2011). Still, little is known about individual differences in prognosis.

Clinical Expression and Pathophysiology

Attachment describes a tendency for human infants to seek comfort, support, nurturance, and protection from one or more discriminated caregivers. The tendency for selective seeking of comfort is not apparent at birth, however. Following a period of interaction and comfort with adult caregivers during the first 6 months, two new infant behaviors become apparent at around 7–9 months of age, stranger wariness and separation protest. Stranger wariness describes an apparent discomfort with unfamiliar adults and selectively turning to those the child knows and trusts. Separation protest refers to the infant’s tendency to protest separation from familiar caregivers. Although individual differences in the intensity and expression of these behaviors are clear, they seem to be

universal. When these behaviors appear, the infant is said to be attached to one or more caregivers.

Under species typical rearing conditions, virtually all infants seem to become attached, generally to a relatively small number of caregiving adults with whom they have regular and substantial contact. Once infants reach a cognitive age of 7–9 months, they begin to seek comfort, support, nurturance, and protection from a relatively small number of caregiving adults whom they have learned through repeated experiences are available to them. Research has demonstrated clearly that the quality of infants' attachments to one or more caregivers is predictive of subsequent psychosocial adaptation. Security of attachment has been measured categorically and continuously and predicts subsequent adjustment, particularly in high-risk groups of children.

In extreme rearing conditions; however, such as social neglect or institutional care, attachment may be seriously compromised or even absent. Attachment disorders describe a constellation of aberrant attachment behaviors and other behavioral anomalies that are believed to result from social neglect and deprivation. For this reason, RAD requires a history of "pathogenic caregiving." In response, rather than insecure attachments, young children with attachment disorders display absent or serious aberrations of attachment. Two clinical patterns, described above, have been defined: an emotionally withdrawn/inhibited pattern and an indiscriminately social/disinhibited pattern. In the emotionally withdrawn/inhibited pattern, the child exhibits limited or absent initiation or response to social interactions with caregivers and aberrant social behaviors, such as constricted, hypervigilant, or highly ambivalent reactions. In the indiscriminate pattern, the child exhibits lack of expected selectivity in seeking comfort, support, and nurturance, with lack of social reticence with unfamiliar adults and a willingness to "go off" with strangers.

What is striking about children with the emotionally withdrawn/inhibited RAD is that they have minimal or no signs of attachment to caregiving adults. The lack of selective attachments in children cognitively capable of forming attachments is the essence of the disorder. In contrast,

children may exhibit signs of indiscriminately social/disinhibited RAD whether or not they have formed attachments. The essence of this form of the disorder is socially disinhibited behavior with strangers. Because it has been documented in children with healthy and unhealthy attachments, as well as in children with no attachments, some have suggested that it is not actually an attachment disorder. For this reason, the current DSM 5 proposal is to define it as disinhibited social engagement disorder, distinct from RAD (Zeanah and Gleason 2010).

Evaluation and Differential Diagnosis

In order to assess the presence or absence of attachment in a child, it is necessary to evaluate the relationship of the child with each of her caregivers. A child is able to have different types of attachments with each of her caregivers. Knowing about a child's attachment to one caregiver does not reveal anything about the child's attachment to another caregiver, and not being attached to one caregiver does not mean that the child is not attached to another caregiver. Thus, the child should be seen with different caregivers in order to assess the lack of attachment that is necessary to make the diagnosis of emotionally withdrawn/inhibited RAD.

The first step in the evaluation is to gather a thorough history of the child. This history should include information on the child's current behaviors, past behaviors, social history, developmental history, medical history, and family history. Careful attention to the emergence and expression of selective attachment behaviors is important.

To guide diagnosis of attachment disorders, use of a structured interview with the child's caregiver, such as the Disturbances of Attachment Interview (Gleason et al. 2011), may be useful. This interview systematically inquires about signs of emotionally withdrawn/inhibited RAD, indiscriminately social/disinhibited RAD, and other aberrant attachment behaviors expressed toward caregivers. For children who have experienced pathogenic or grossly inadequate care, identifying a reporter who is knowledgeable about the child's behaviors may be a challenge.

The evaluation of the child should include inquiries about the child's behavior in different settings and with different caregivers to note any differences. Formal observations of the child and parent interactions are also important. Procedures derived from developmental research, such as the Strange Situation Procedure (Ainsworth et al. 1978) or the Crowell procedure (Zeanah et al. 2000), are relatively short observations of child and parent interaction which help the clinician systematically to observe the interaction between the child and caregiver (Zeanah et al. 2011).

During the assessment, there are several other diagnoses to consider since attachment disorders may share features of some other disorders (see Table 1 for details). For example, emotionally withdrawn/inhibited RAD may be confused with autistic spectrum disorders or global developmental delay. The problems with emotional regulation and impaired social reciprocity may resemble the social difficulties of a child with an autistic spectrum disorder. On the other hand, there is little reason to expect restricted interests or repetitive behaviors in children with attachment disorders. A history of adverse caregiving as well as no selective impairment in language or pretend play should point toward a diagnosis of RAD in such

children. Although children with RAD are likely to have cognitive delays, their impaired social responsiveness is not a symptom of intellectual disabilities alone. Children with intellectual disabilities should have social behavior and emotional expressiveness congruent with their cognitive ages. On the other hand, selective reductions in social reciprocity and emotional expressiveness are more indicative of emotionally withdrawn/inhibited RAD.

An important diagnosis to consider with indiscriminately social/disinhibited RAD is attention deficit hyperactivity disorder (ADHD). In RAD, young children have social impulsivity, but this should not be confused with the broader impulsivity and hyperactivity of children with ADHD. It is important to look in detail at how the child interacts in social situations and especially with unfamiliar adults. Children with RAD lack selectivity in directing their social and sometimes attachment behaviors. Children with ADHD may share these features but also demonstrate impulsivity in nonsocial situations. Children with indiscriminately social/disinhibited RAD should show more profound misreading of social cues and situations and engage in more social and physical boundary violations.

Attachment Disorder, Table 1 Differential diagnosis of attachment disorders

Attachment disorder	Differential diagnosis	Similarities	Differences
Emotionally withdrawn/inhibited type	1. Autistic spectrum disorder	1. Disturbances in emotional regulation	1. Attachment disorder does not have selective impairment in pretend play, repetitive preoccupation, or a language abnormality
		2. Impaired or absent social and emotional reciprocity	2. Attachment disorder has a history of seriously adverse caregiving
		3. May involve cognitive delays	
Emotionally withdrawn/inhibited type	1. Intellectual disability	1. Cognitive delays	1. Attachment disorder does not have social/emotional behaviors consistent with developmental age
			2. Attachment disorder has evidence of deviance in social responsiveness and regulations of emotion
Indiscriminately social/disinhibited type	1. Attention deficit hyperactivity disorder	Social impulsivity and attention seeking behavior	1. Attachment disorder in males shows a lack of selectivity in relationships with caregivers and peers

Treatment

There is only one intentional treatment study of attachment disorders that includes pre- and post-assessments and uses random assignment (Smyke et al. in preparation). The BEIP demonstrated substantial treatment effects on reduction of signs of emotionally withdrawn/inhibited RAD and more modest treatment effects of reduction in signs of indiscriminately social/disinhibited RAD for children placed in foster care compared to those who experienced continued institutional care (Smyke et al.). This study bolsters confidence in other less rigorously designed studies that all suggest that signs of emotionally withdrawn/inhibited RAD disappear rapidly when children are placed in reasonably normative caregiving environments. Similarly, the results in BEIP are compatible with studies of internationally adopted children suggesting that signs of indiscriminately social/disinhibited RAD are less responsive to more normative caregiving environments, and that a minority of children have persistent signs of the disorder over years (Smyke et al.).

Future research needs to better determine recommendations for adoptive parents whose young children exhibit signs of RAD and how best to deal not only with the behavioral manifestation but also with the social cognitive abnormalities that presumably underlie the disorder. Further, although there is a clear tendency for signs of both types of disorders to diminish over time, questions about sequelae have not been adequately answered at this point.

See Also

- [Feral Children](#)
- [Posttraumatic Stress Disorder](#)
- [Reactive Attachment Disorder](#)
- [Romanian Adoptive Children](#)

References and Reading

Ainsworth, M. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation*. Hillsdale: Lawrence Erlbaum Associates.

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association. Text Revision.
- Bowlby, J. (1969). *Attachment and loss* (Vol. 1). New York: Basic Books.
- Chaffin, M., Hanson, R., Saunders, B., et al. (2006). Report of the APSAC task force on attachment therapy, reactive attachment disorder, and attachment problems. *Child Maltreatment*, 11(1), 76–89.
- Chisholm, D. (1998). A three-year follow-up of attachment and indiscriminate friendliness in children adopted from Romanian orphanages. *Child Development*, 69, 1092–1106.
- Egger, H., Erkanli, A., Keeler, G., Potts, E., Walter, B., & Angold, A. (2006). Test-retest reliability of the preschool age psychiatric assessment (PAPA). *Journal of the American Academy of Child and Adolescent Psychiatry*, 45, 538–549.
- Gleason, M. M., Fox, N. A., Drury, S., Smyke, A. T., Egger, H. L., Nelson, C. A., et al. (2011). The validity of evidence-derived criteria for reactive attachment disorder: Indiscriminately social/disinhibited and emotionally withdrawn/inhibited types. *Journal of the American Academy of Child and Adolescent Psychiatry*, 50, 216–231.
- Hodges, J., & Tizard, B. (1989). Social and family relationships of ex-institutional adolescents. *Journal of Child Psychology and Psychiatry*, 30, 77–97.
- Oosterman, M., & Schuengel, C. (2007). Autonomic reactivity of children to separation and reunion with foster parents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46, 1196–1203.
- Pears, K. C., Bruce, J., Fisher, P. A., & Kim, H. K. (2010). Indiscriminate friendliness in maltreated foster children. *Child Maltreatment*, 15, 64–75.
- Rutter, M., Colvert, E., Kreppner, J., Beckett, C., Castle, J., Groothues, C., et al. (2007). Early adolescent outcomes for institutionally-deprived and non-deprived adoptees. I: disinhibited attachment. *Journal of Child Psychology and Psychiatry*, 48, 17–30.
- Rutter, M., Kreppner, J., & Sonuga-Barke, E. (2009). Emanuel Miller lecture: attachment insecurity, disinhibited attachment, and attachment disorders: where do research findings leave the concepts? *Journal of Child Psychology and Psychiatry*, 50, 529–543.
- Skovgaard, A. M., Houmann, T., Christiansen, E., Landorph, S., Jørgensen, T., Olsen, E. M., et al. (2007). The prevalence of mental health problems in children 1½ years of age—the Copenhagen Child Cohort 2000. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 48(1), 62–70.
- Smyke, A. T., Zeanah, C. H., Gleason, M. M., Drury, S. S., Fox, N. A., Nelson, C. A., et al. (2012). A randomized controlled trial of foster care vs. institutional care for children with signs of reactive attachment disorder. *American Journal of Psychiatry*, 169, 508–514.
- Tizard, B., & Rees, J. (1975). The effect of early institutional rearing on the behaviour problems and affectional relationships of four-year-old children. *Journal of Child Psychology and Psychiatry*, 16, 61–73.

- World Health Organization. (1992). *ICD-10: International classification of diseases and related health problems*. Geneva: World Health Organization.
- Zeanah, C. H., Berlin, L. J., & Boris, N. W. (2011). Practitioner review: Clinical applications of attachment theory and research for infants and young children. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 52(8), 819–833.
- Zeanah, C., & Gleason, M. M. (2010). Reactive attachment disorders: A review for DSM 5. Retrieved December 29, 2010, from <http://stage.dsm5org/Proposed%20Revision%20Attachments/APA%20DSM-5%20Reactive%20Attachment%20Disorder%20Review.pdf>.
- Zeanah, C., Larrieu, J., Valliere, J., & Heller, S. (2000). Infant-parent relationship assessment. In C. H. Zeanah (Ed.), *Handbook of infant mental health* (2nd ed., pp. 222–235). New York: Guilford Press.
- Zeanah, C. H., Scheeringa, M. S., Boris, N. W., Heller, S. S., Smyke, A. T., & Trapani, J. (2004). Reactive attachment disorder in maltreated toddlers. *Child Abuse and Neglect: The International Journal*, 28, 877–888.
- Zeanah, C., & Smyke, A. (2008). Attachment disorders in relation to deprivation. In M. Rutter & E. Taylor (Eds.), *Rutter's child and adolescent psychiatry* (5th ed., pp. 906–915). Malden/Oxford: Blackwell Publishing.
- Zeanah, C. H., Smyke, A. T., Koga, S., Carlson, E., & The BEIP Core Group. (2005). Attachment in institutionalized and community children in Romania. *Child Development*, 76, 1015–1028.

Attention

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Definition

The ability to orient, sustain, and shift attention on relevant stimuli, using internal and external cues, is a critical skill for learning about the world. Prioritizing stimuli in order to process pertinent, and exclude peripheral, information facilitates selective learning, a skill necessary for many child development processes, including vocabulary development, problem solving, and later, successful classroom learning (Frick and Richards 2001; Kannass and Colombo 2007; Sillar and Sigman 2008). Children with autism often display

atypical development of attention. The processes in which these abnormalities manifest, however, are yet to be determined (Ames and Fletcher-Watson 2010). Despite the high prevalence of attentional difficulties seen in children with autism, these difficulties are not considered a core characteristic of the disorder as specified by the fourth version of the Diagnostic and Statistical Handbook of Mental Disorders (DSM IV 1994), but rather an associated symptom of ASD.

Behavior is acted upon using visual information from the environment. For example, safe driving is largely dependent on drivers attending to stoplights, signs, pedestrians, and other cars and ignoring distracting, irrelevant environmental stimuli. The breadth of attention literature identifies several components of domain-specific and domain-general attending. Visual attention, in particular, plays a large role in domain-specific attention, such as social attention. Social attention is the preferential selection of social over nonsocial stimuli for attention and has been the subject of much research due to its high correlation with later social developmental processes, such as joint attention, theory of mind, and language development (Adamson et al. 2009; Ames and Fletcher-Watson 2010; Mundy and Newell 2007; Sodian and Thoermer 2008). Moreover, social attention is of particular interest to the study of autism due to its relation to social interactions and communication, two core deficits of the disorder.

Attention may be subdivided into the ability to focus, sustain, shift, and encode (Goldstein et al. 2001; Zubin 1975). Focused attention is the ability to concentrate and perform a task on a specific stimulus in the midst of distracting stimuli. Sustained attention is defined as the capacity to maintain attention on a target stimulus over a prolonged period of time. Shifting attention is the ability to effectively transfer concentration from one stimulus to another. Encoding attention is the ability to intake and interpret information from the environment (Goldstein et al. 2001). Research on these specific skills may be used to identify which aspects of attention children with autism seem to struggle with most and, conversely, which areas of attention develop typically.

A comprehensive understanding of attention must include a description of environmental stimuli that help an individual to attend. Attention cueing, that is, attention directed by environmental prompts, affects what stimuli humans attend to; these environmental prompts are identified as exogenous and endogenous factors. Exogenous cues, those that activate “bottom-up” processes, are derived from stimuli properties (e.g., size, color) and evoke involuntary attention (Corbetta and Shulman 2002). Endogenous cues, on the other hand, often characterized as activating “top-down” processes, evoke conscious and voluntary attention control through cognitive processes, learned behavior, or past experiences (Corbetta and Shulman 2002; Hauer and MacLeod 2006). In this way, previous experiences and learned behaviors influence on what or where the child attends.

The multitude of cognitive, social, and language developmental skills learned during play make free play an important setting in which to study attention in children. Ruff and Capozzoli (2003) studied the developmental trajectory of visual attention during play and identified three types of attention. Causal attention is defined as looking at objects (e.g., toys), but not physically engaging with them; settled attention is looking at and manipulating an object; and focused attention is concentrating on an object intently and may include facial expressions and extraneous body movement in order to bring the object closer to the face or body. Collectively, the study of attention covers a wide array of specific topics, all of which hold importance for a comprehensive understanding of the topic and for the development of interventions aimed at healthy development.

Historical Background

Attention has been a topic of study for decades by researchers and clinicians alike. Because of the high occurrence of attention deficits in autism, this topic has been the focus of numerous studies in autism research, in particular. The discourse of processes and causes of this apparent attention

deficit has evolved over time. Early researchers hypothesized that attention difficulties in children with autism were due to hypo- or hyperarousal. That is, some researchers concluded that arousal modulation in particular, was a potential cause of low attention abilities (Hutt et al. 1964; Ornitz and Ritvo 1968). Other investigators attributed limited attention skills to over-selectivity or what some referred to as “tunnel vision,” that is, intense attention to specific details in combination with a lack of interpretation of outside environmental cues (Lovaas et al. 1979; Rincover and Ducharme 1987). More recently, it has been hypothesized that attention problems may be due to difficulties in prioritizing relevant stimuli and disregarding irrelevant stimuli (Bryson et al. 1990; Burack 1994). Furthermore, Ornitz and colleagues (1988) proposed that children with autism struggle in attention shifting, in particular, because they lack an interest in people or social stimuli (Ornitz 1988). Previous studies have also dichotomized attention in studying auditory and visual attention and found that children with autism differed from typical children in auditory attention (Casey et al. 1993) but not in visual attention (Pascualvaca et al. 1998). This finding, however, is inconsistent with more recent findings concerning visual attention in the literature (Goldstein et al. 2001; Leekam et al. 2000) and may be due to differences in measurement (Ames and Fletcher-Watson 2010). With technological advances in detection tools, so came a new wave of studies addressing biological hypotheses for attention deficits. Throughout the past two decades, researchers hypothesized that inattentive behavior may be due to neural abnormalities in areas such as the parietal lobe and the frontal lobe (Courchesne et al. 1993, 1994; Ornitz 1988; Pascualvaca et al. 1998).

Current Knowledge

Developmental studies on attention reveal that attention evolves over the course of childhood and different patterns of attention behaviors are observed over time. The duration of time infants spend looking at objects or people, which reflects

differences in underlying attention processes (Kannass and Oakes 2008), increases from birth through 8 to 10 weeks, then decreases between 3 and 5 or 6 months, and remains stable or slightly increases thereafter (Colombo 2001, 2002). The initial increase of look duration may be due to steady increases in arousal and alertness, whereas the decrease of look duration may be indicative of improvements in information processing. The plateau reached near the first year is likely indicative of endogenous factors or top-down processes manifesting (Corbetta and Shulman 2002; Colombo 2001, 2002).

Children with autism appear to show different patterns of attention development than their typical peers. For example, the top-down processing that develops around the first year appears to pose difficulty for children with autism as compared to typically developing children (Ames and Fletcher-Watson 2010; Ames and Jarrold 2007; Leekam et al. 2000). Goldstein et al. (2001) also found that individuals with autism were different from their typical counterparts in their abilities to focus and shift attention, but were not different in their abilities to sustain and encode attention (Goldstein et al.). Another study found that children with autism had more circumscribed, preservative, and detail-oriented attention (Sasson 2008).

The results of studies involving eye tracking and visual attention in the context of social stimuli have also found differences among individuals with autism. One study indicated that when shown images of objects and people, individuals with autism generally attended to the nonsocial aspects of the picture, that is, objects rather than faces. Further, the investigation found that when individuals with autism did attend to social images, such as a human face, they looked at noncritical social elements, such as the nose rather than the eyes (Klin et al. 2002). Another study found that children with autism have difficulties processing both social and nonsocial information (Leekam et al. 2000). Collectively, the literature suggests that individuals with autism lack attention to social stimuli which undoubtedly affects social and emotional learning.

Future Directions

Attention abilities, from infancy throughout childhood, can have several effects on social relationships, language development, and cognitive development. Future work on attention in autism should aim to examine these processes in natural settings to more appropriately capture variables that facilitate and inhibit successful attention for this population within a variety of contexts, such as their social relationships and academic settings. Moreover, there is a need for greater longitudinal work in this area in order to understand how attentional processes develop over time and the extent to which these processes impact social and cognitive outcomes for individuals with ASDs (Ames and Fletcher-Watson 2010). Finally, attention has the potential to be an invaluable early detection tool for autism diagnoses. Children with autism face early and pervasive abnormalities in attention abilities (Allen and Courchesne 2001; Elsabbagh et al. 2009). The developmental course of attention in typical development has been outlined by previous investigations; future research could use such knowledge to examine attention in children at risk for autism early in infancy. Earlier screening and detection to earlier intervention which has been shown to yield improved outcomes for children with autism and other developmental delays.

See Also

- [Joint Attention](#)
- [RJA/IJA \(Initiating/Responding to Joint Attention\)](#)

References and Reading

- Adamson, L., Bakeman, R., Deckner, D., & Ronski, M. (2009). Joint engagement and the emergence of language in children with autism and down syndrome. *Journal of Autism and Developmental Disorders*, 39, 84–96.
- Allen, G., & Courchesne, E. (2001). Attention function and dysfunction in autism. *Frontiers of Bioscience*, 6, 105–119.

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
- Ames, C., & Fletcher-Watson, S. (2010). A review of methods in the study of attention in autism. *Developmental Review*, 30, 52–73. <https://doi.org/10.1016/j.dr.2009.12.003>.
- Ames, C. S., & Jarrold, C. (2007). The problem with using eye-gaze to infer desire: A deficit of cue inference in children with autism spectrum disorder? *Journal of Autism and Developmental Disorders*, 37, 1761–1775.
- Bartgis, J., Thomas, D., Lefler, E., & Hartung, C. (2008). The development of attention and response inhibition in early childhood. *Infant and Child Development*, 17, 491–502. <https://doi.org/10.1002/icd.563>.
- Bryson, S. E., Wainwright-Sharp, J. A., & Smith, I. M. (1990). Autism: A developmental spatial neglect syndrome? In J. T. Enns (Ed.), *The development of attention: Research and theory* (pp. 405–427). Amsterdam: Elsevier North Holland.
- Burack, J. A. (1994). Selective attention deficits in persons with autism: Preliminary evidence of an inefficient attentional lens. *Journal of Abnormal Psychology*, 103, 535–543.
- Casey, B. J., Gordon, C. T., Mannheim, G. B., & Rumsey, J. M. (1993). Dysfunctional attention in autistic savants. *Journal of Clinical and Experimental Neuropsychology*, 15, 933–946.
- Colombo, J. (2001). The development of visual attention in infancy. *Annual Review of Psychology*, 52, 337–367.
- Colombo, J. (2002). Infant attention grows up: The emergence of a developmental cognitive neuroscience perspective. *Current Directions in Psychological Science*, 11, 196–200.
- Corbetta, M., & Shulman, G. (2002). Control of goal-directed and stimulus driven attention in the brain. *Neuroscience*, 3, 201–215. <https://doi.org/10.1038/nm755>.
- Courchesne, E., Townsend, J., Akshoomoff, N. A., Saitoh, O., Yeung-Courchesne, R., Lincoln, A. J., et al. (1994). Impairment in shifting attention in autistic and cerebellar patients. *Behavioral Neuroscience*, 108, 848–865.
- Courchesne, E., Townsend, J. P., Akshoomoff, N. A., Yeung-Courchesne, R., Press, G. A., Murakmi, J. W., et al. (1993). A new finding: Impairment in shifting of attention in autistic and cerebellar patients. In S. H. Broman & J. Grafman (Eds.), *Atypical deficits in developmental disorders: Implications for brain function* (pp. 101–137). Hillsdale: Erlbaum.
- Elsabbagh, M., Volein, A., Holmboe, K., Tucker, L., Csibra, G., Baron-Cohen, S., et al. (2009). Visual orienting in the early broader autism phenotype: Disengagement and facilitation. *Journal of Child Psychology and Psychiatry*, 50, 637–642.
- Frick, J., & Richards, J. (2001). Individual differences in infants' recognition of briefly presented visual stimuli. *Infancy*, 2, 331–352.
- Goldstein, G., Johnson, C., & Minshew, N. (2001). Attentional processes in autism. *Journal of Autism and Developmental Disorders*, 31, 433–440.
- Hauer, B., & MacLeod, C. (2006). Endogenous versus exogenous attentional cueing effects on memory. *Acta Psychologica*, 122, 305–320. <https://doi.org/10.1016/j.actpsy.2005.12.008>.
- Hutt, C., Hutt, S. J., Lee, D., & Ounsted, C. (1964). Arousal and childhood autism. *Nature*, 204, 908–909.
- Kannass, K., & Colombo, J. (2007). The effects of continuous and intermittent distractors on cognitive performance and attention in preschoolers. *Journal of Cognition and Development*, 8, 63–77.
- Kannass, K., Colombo, J., & Wyss, N. (2010). Now, pay attention! The effects of instruction on children's attention. *Journal of Cognition and Development*, 11, 509–532. <https://doi.org/10.1080/15248372.2010.516418>.
- Kannass, K., & Oakes, L. (2008). The development of attention and its relations to language in infancy and toddlerhood. *Journal of Cognition and Development*, 9, 222–246. <https://doi.org/10.1080/15248370802022696>.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59, 809–815.
- Leekam, S. R., Lopez, B., & Moore, C. (2000). Attention and joint attention in preschool children with autism. *Developmental Psychology*, 36, 261–273.
- Lovaas, I., Koegel, R., & Schreibman, L. (1979). Stimulus overselectivity in autism: A review of research. *Psychological Bulletin*, 86, 1236–1254.
- Mundy, P., & Newell, L. C. (2007). Attention, joint attention and social cognition. *Current Directions in Psychological Science*, 16, 269–274.
- Ornitz, E. M. (1988). Autism: A disorder of directed attention. *Brain Dysfunction*, 1, 309–322.
- Ornitz, E. M., & Ritvo, E. R. (1968). Perceptual inconstancy in early infantile autism. *Archives of General Psychiatry*, 18, 76–98.
- Pascualvaca, D., Fantie, B., Papageorgiou, M., & Mirsky, A. (1998). Attentional capacities in children with autism: Is there a general deficit in shifting focus? *Journal of Autism and Developmental Disorders*, 28, 467–478.
- Rincover, A., & Ducharme, J. M. (1987). Variables influencing stimulus over-selectivity and “tunnel-vision” in developmentally disabled children. *American Journal of Mental Deficiency*, 91, 422–430.
- Ruff, H., & Capozzoli, M. (2003). Development of attention and distractibility in the first 4 years of life. *Developmental Psychology*, 39, 877–890.
- Sasson, N. (2008). Children with autism demonstrate circumscribed attention during passive viewing of complex social and nonsocial picture arrays. *Autism Research*, 1, 31–42.
- Sillar, M., & Sigman, M. (2008). Modeling longitudinal change in the language abilities of children with autism: Parent behaviors and child characteristics as predictors of change. *Developmental Psychology*, 44, 1691–1704.

Sodian, B., & Thoermer, C. (2008). Precursors to a theory of mind in infancy: Perspectives for research on autism. *Quarterly Journal of Experimental Psychology*, 61, 27–39. <https://doi.org/10.1080/17470210701508681>.

Zubin, J. (1975). Problem of attention in schizophrenia. In M. L. Kietzman, S. Sutton, & J. Zubin (Eds.), *Experimental approaches to psychopathology* (pp. 139–166). New York: Academic Press.

Attention Deficit Disorder

► Attention Deficit/Hyperactivity Disorder

Attention Deficit/Hyperactivity Disorder

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Synonyms

ADD; ADHD; Attention deficit disorder; Hyperkinetic disorders; Minimal brain damage; Minimal brain dysfunction; Syndrome of deficits in attention, motor control, and perception (DAMP)

Short Description or Definition

Attention deficit/hyperactivity disorder (ADHD) is one of the most common psychiatric disorders in children and adolescents. It is characterized by inattention, impulsivity, and hyperactivity. Its rate decreases with the increase of age. ADHD usually starts in childhood and continues through adolescence into adulthood. The burden and psychosocial functioning impairment of ADHD is farther than its inattention, impulsivity, and hyperactivity symptoms. There are many controversies and scientific debates about ADHD (Biederman and Faraone 2005; Furman 2008).

Categorization

According to DSM-IV, there are three subtypes of ADHD called “predominantly inattentive,” “predominantly hyperactive-impulsive,” and “combined.” ICD-10 lacks this categorization.

Epidemiology

The prevalence of ADHD in children is estimated to be about 8–12% (Biederman and Faraone 2005). The rate of ADHD in boys is three times more than girls, and this ratio in the clinical sample is six to nine times (Ghanizadeh et al. 2008).

Natural History, Prognostic Factors, and Outcomes

From about two centuries ago, children with symptoms of inattention, impulsivity, and hyperactivity have been described (Crichton 2008; Lange et al. 2010). Heinrich Hoffmann described some symptoms of ADHD in the story of Fidgety Phil (Hoffmann (1948) cited by Lange et al. (2010)). Moral control defect was introduced by Sir George Frederic Still ((Still 1902) cited in Lange et al. (2010)). He reports that these children cannot internalize rules and limits. Then, the term of “postencephalitic behavior disorder” was introduced after the worldwide influenza epidemic (Rothenberger and Neumärker 2005) cited in Lange et al. (2010). The terms of “minimal brain damage” and “minimal brain dysfunction” were described (Hoffmann 1948). The name was changed to “hyperkinetic reaction of childhood” in the second edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-II) (American Psychiatric Association (APA) (1967). *Diagnostic and statistical manual for mental disorders*). Overactivity, restlessness, distractibility, and short attention span were the characteristics of this disorder (APA (1967). *Diagnostic and statistical manual for mental disorders*). In the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III), the disorder was called “attention

deficit disorder (ADD): with and without hyperactivity.” In this edition, the focus was on inattentiveness rather than hyperactivity (APA (1980). *Diagnostic and statistical manual (DSM-III)*). In addition, it was stressed that hyperactivity was no more a necessary criterion for diagnosis of this disorder. From 1987, revision of the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R), this disorder was renamed “attention deficit/hyperactivity disorder” (ADHD) (APA (1987). *Diagnostic and statistical manual (DSM-III, revised)*). In the DSM-III-R, the subtype of “ADD without hyperactivity” was replaced with the category of “undifferentiated ADD” (Lange et al. 2010). From the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (APA (1994). *Diagnostic and statistical manual (DSM-IV)*), the three subtypes of ADHD including “predominantly inattentive type,” “predominantly hyperactive-impulsive type,” and “combined type with symptoms of both dimensions” were presented (Lahey et al. 1994). There was no change regarding ADHD in the text revision of the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (APA (2000). *Diagnostic and statistical manual (DSM-IV, Text Rev.)*). It is expected that DSM-V will be published in 2012.

Multiple comorbid disorders and parent-reported ADHD severity are associated with the poorer psychosocial quality of life (Klassen et al. 2004). The type of comorbidity is also associated with the quality of life. Lower quality of life is associated with the comorbidity of oppositional defiant disorder, conduct disorder, and learning disorder (Klassen et al. 2004). There is a positive short-term effect of medication on quality of life in children, adolescents, and adults with ADHD (Coghill 2010). Comorbidity of ODD with ADHD is associated with more severe ADHD symptoms, peer problems, and family problems (Ghanizadeh and Jafari 2010).

Children and adolescents with ADHD have poorer social and communication skills leading to more peer relationship problems. More than two-thirds of them have no close friends (Wehmeier et al. 2010). So, they are more

frequently rejected by others. This makes them more prone to join to deviant peer groups, injuries, occupational problems, educational problems, cigarettes, and substance use disorders (Biederman and Faraone 2005).

The symptoms of ADHD continue from childhood into adult. However, most of them will not meet the full diagnostic criteria in adult but they will meet the diagnosis of ADHD in partial remission (Fischer et al. 2002).

Clinical Expression and Pathophysiology

While there are many controversies about ADHD, the improvement of some symptoms after pharmacotherapy supports that there are neurobiological causes for the heterogeneous nature of ADHD. There is a large gap in our knowledge and current literature regarding ADHD. However, it is clear that there is not any one specific brain area or genetic or neurochemical factor as the etiology of ADHD.

The etiology of ADHD is complex (Steinhausen 2009). The heritability of ADHD is reported in twin and adoption studies. However, more molecular genetic studies are necessary to indicate the complex genetics and the interaction of gene by environment in ADHD (Biederman and Faraone 2005; Nigg et al. 2010).

There is not enough evidence supporting that ADHD is caused by foods or food additives (Biederman and Faraone 2005), while lead is reported to be associated with ADHD (Ghanizadeh 2011). Exposure to toxins such as mercury, lead, manganese, and polychlorinated biphenyls (PCBs) and pregnancy and delivery complications (such as eclampsia, maternal age, prenatal alcohol exposure, maternal smoking, fetal postmaturity, duration of labor, fetal distress, low birth weight, and hemorrhage) are other risk factors associated with ADHD (Banerjee et al. 2007). Meanwhile, TV viewing is not a risk factor for ADHD (Banerjee et al. 2007).

From the psychosocial factors, low family cohesion, exposure to parental psychopathology especially maternal psychopathology, low maternal education, low social class, and single

parenthood are important risk factors for ADHD (Biederman and Faraone 2005).

Brain structural studies do not report consistent findings for ADHD. However, most of imaging studies delineated overall decrease in total brain size, the caudate nucleus, prefrontal cortex white matter, corpus callosum and the cerebellar vermis (Tripp and Wickens 2009). Some of these areas have a high density of dopamine receptors.

Neuropsychological studies show the impairment of vigilance attention, executive function, working memory response, and motivation in some children with ADHD (Tripp and Wickens 2009). Brain maturation is delayed in ADHD (Curatolo et al. 2009).

Finally, children with ADHD may have difficulties in social exchanges such as sharing and cooperation with peers. They are self-centered, impulsive, and commanding (Wehmeier et al. 2010).

Evaluation and Differential Diagnosis

In many countries, ADHD diagnoses are generally made using Diagnostic and Statistical Manual, Fourth Edition, Text Revision (APA (2000). *Diagnostic and statistical manual (DSM-IV, Text Rev.)*). According to 4th Edition, Text Revision (DSM-IV-TR) criteria, there are two groups of symptoms including (a) attention deficit, (b) hyperactivity, or impulsivity. Six or more items from at least one of the groups are required for ADHD diagnosis. In addition, functional impairments in at least two different settings such as at home, school, and nursery are required.

In other countries, especially in Europe, International Classification of Diseases-10 (ICD-10) is used (World Health Organization (WHO) 1992). Hyperkinetic disorder is the ICD-10 equivalent of ADHD diagnosis (WHO 1992). In ICD-10, several items from attention deficit, hyperactivity, and impulsivity are required to reach diagnosis. Therefore, it is expected that the rate of ADHD in countries using DSM-IV-TR criteria would be reported higher than that of those countries using ICD-10 criteria.

ADHD diagnosis is subjective using the diagnostic systems criteria. There is not any objective

diagnostic test or any biomedical laboratory test for it. However, the ADHD diagnosis is reliable when well-trained raters assess and agree the presence of its symptoms (Biederman and Faraone 2005).

There is a weak correlation between different informants such as parents and teachers for the rating of ADHD symptoms. In other words, they usually do not agree on their assessment of symptoms in children with ADHD. The evaluation of children in different situations can be an explanation for this disagreement. Teachers evaluate children in school while the children are taking medication. Sometime, parents may report some symptoms that the symptoms are not reported by teachers.

In clinical samples, ADHD is usually comorbid with other psychiatric disorders. The rate of at least one comorbid psychiatric disorder in children with ADHD is more than 80% (Ghanizadeh et al. 2008). Other disruptive behavior disorders (oppositional defiant disorder (ODD) or conduct disorder (CD)) and anxiety disorders are the most common comorbid disorders in children with ADHD. The rate for ODD and CD is about 59.3% and 13.6% (Ghanizadeh et al. 2008). Some of the other comorbid disorders are mood disorders, tic disorder, enuresis, and encopresis.

It is interesting that the parent of children with ADHD usually suffer from psychiatric disorders. The lifetime prevalence of ADHD in fathers and mothers of children with ADHD are 45.8% and 17.7%, respectively. Major depressive disorder is very frequent in the parents. The rate in father and mothers are 48.1% and 43.0%, respectively (Ghanizadeh et al. 2008).

Co-occurrence of ADHD and Autism

ADHD DSM-IV-derived items do not overlap with autism spectrum disorder (Ghanizadeh 2010), and the comorbidity of ADHD and autism is precluded in the DSM-IV-TR. Therefore, the symptoms of inattentiveness, hyperactivity, or impulsivity in individuals with autism originate from autism, not ADHD. Meanwhile, there are many individuals who meet diagnostic criteria

for both ADHD and autism. In addition, many patients with Asperger's syndrome are screened with concerns about ADHD (Murray 2010). The children with autism may severely attend to their interest and do not attend to other factors in their environment. It can be interpreted as inattentiveness. Also, sometimes, their stereotypic motor behavior can be interpreted as hyperactivity (Murray 2010). However, there are many published studies reported the co-occurrence of ADHD and autism. About 40–78% of individuals with autism meet diagnostic criteria for DSM-IV ADHD (Murray 2010). Eighty-seven percent of children with autism spectrum disorder have at least one of the three components of ADHD (Ames and White 2011). The rate of autistic traits in children with ADHD is from one-third to one-fifth (Grzadzinski et al. 2011).

In addition, the subtype of ADHD is associated with the severity of difficulties in autism. For example, language and social problems are more common in those with both autism and ADHD-inattentive subtype. Moreover, less symptoms of autism are reported in those with ADHD-hyperactivity subtype. While internalizing behavior problems are usually seen in autism, externalizing behavior problems are more common in those with ADHD. A combination of externalizing and internalizing behavior problems are reported in those with both ADHD and autism (Murray 2010). Clinical profiles and outcomes of children with both ADHD and autism are different with that of those children with autism alone. They have severe social problems and poorer outcomes. Furthermore, executive function is more impaired in the individuals with both ADHD and autism than those with ADHD or autism alone. Motor coordination abnormalities are different between ADHD and autism. While motor response inhibition is more common in ADHD, motor planning impairment is more common in autism (Murray 2010). About two-thirds of children with the syndrome of deficits in attention, motor control, and perception (DAMP) meet diagnostic criteria for autism spectrum disorders. Comorbidity with developmental coordination problems is more likely to co-occur with autism symptoms than those with ADHD alone. Autism,

ADHD, and dyslexia overlap genetically (Smalley et al. 2005).

ADHD can be dissociated from autism spectrum disorders regarding executive dysfunction and response inhibition. Those with autism spectrum disorders are slow and accurate, while those with ADHD are impulsive (Johnston et al. 2011).

It is expected that the comorbidity of ADHD and autism spectrum disorders will be allowed in DSM-V. Then, autism will not be an exclusive criterion for ADHD diagnosis.

Treatment

The educating and counseling of parents (Ghanizadeh 2007), teachers (Ghanizadeh et al. 2006), and general physicians (Ghanizadeh and Zarei 2010) about ADHD is highly necessary and recommended. Many of parents, teachers, and medical service providers have not enough and updated knowledge towards ADHD symptoms and its management. Behavioral parent training is encouraged (van den Hoofdakker et al. 2007).

Drug therapy with stimulant drugs (Cornforth et al. 2010) and atomoxetine (Vaughan et al. 2009) is better than no drug therapy. However, there is not enough evidence indicating any difference between these medications regarding their efficacy or side effects (King et al. 2006).

The precise mechanism of stimulants in ADHD is not known. Noradrenaline and dopamine neurotransmitter systems are involved in ADHD. Methylphenidate and dextroamphetamine are stimulant medications which are effective in the management of ADHD. Atomoxetine is a nonstimulant catecholaminergic medication. They improve ADHD symptoms through increasing activation in cortical and subcortical regions involved in attention and executive functions (Curatolo et al. 2009). Meanwhile, there is a concern about the possible association of atomoxetine and increased suicidal behavior (Garnock-Jones and Keating 2009).

There are concerns about the higher rate of side effects of stimulants in individuals with both autism and ADHD than those with ADHD alone. In addition, methylphenidate efficacy in

autism is less than ADHD (Stigler et al. 2004). While the response rate is limited up to 25%, the rate of side effects reaches to 60% (Stigler et al. 2004). Dexamphetamine may worsen the symptoms (Handen et al. 2000).

Clonidine and guanfacine are α -2 agonists with promising efficacy on hyperactivity, impulsivity, irritability, explosive behaviors, stereotypies, and social interaction (Scahill et al. 2006).

Atomoxetine selectively inhibits the presynaptic norepinephrine transporter. There are contradictory reports about the efficacy of atomoxetine on ADHD symptoms in autism. While an open-label study supported its efficacy (Posey et al. 2006), others did not report a significant effect (Charnsil 2011).

Donepezil as a anticholinesterase inhibitor may decrease some symptoms of ADHD in children with autism (Yoo et al. 2007). Further controlled trials are required to detect the significant gains of these medications on autism.

There are open-label studies promising the efficacy of atypical antipsychotics, such as risperidone, quetiapine, and aripiprazole, on hyperactivity symptom in autism (Murray 2010).

See Also

- ▶ Affective Disorders (Includes Mood and Anxiety Disorders)
- ▶ Antipsychotics: Drugs
- ▶ Aripiprazole
- ▶ Asperger Syndrome
- ▶ Atomoxetine
- ▶ Attention
- ▶ Atypical Antipsychotics
- ▶ Autism
- ▶ Autistic Disorder
- ▶ Behavior
- ▶ Behavior Modification
- ▶ Behavior Rating Scale (BRS)
- ▶ Cerebral Cortex
- ▶ Clonidine
- ▶ Communication Disorder/Communication Impairment
- ▶ Comorbidity
- ▶ Conduct Disorder

- ▶ Developmental Milestones
- ▶ Dextroamphetamine
- ▶ DSM-III
- ▶ DSM-III-R
- ▶ DSM-IV
- ▶ Dyslexia
- ▶ Education
- ▶ Educational Therapy
- ▶ Encopresis
- ▶ Enuresis
- ▶ Epidemiology
- ▶ Executive Function (EF)
- ▶ Expressive Language Disorder
- ▶ Guanfacine
- ▶ ICD 10 Research Diagnostic Guidelines
- ▶ Methylphenidate
- ▶ Mood Disorders
- ▶ Motivation
- ▶ Motor Planning
- ▶ Neurotransmitter
- ▶ Norepinephrine
- ▶ Pervasive Developmental Disorder
- ▶ Repetitive Behavior
- ▶ Risperidone
- ▶ Stereotypic Behavior
- ▶ Stimulant Medications
- ▶ Tics
- ▶ Treatment Integrity

References and Reading

- American Psychiatric Association. (1967). *Diagnostic and statistical manual for mental disorders*. Washington, DC: APA Press.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual (DSM-III)*. Washington, DC: APA Press.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual (DSM-III, revised)*. Washington, DC: APA Press.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual (DSM-IV)*. Washington, DC: APA Press.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual (DSM-IV)*. Washington, DC: APA Press.
- Ames, C. S., & White, S. J. (2011). Brief report: Are ADHD traits dissociable from the autistic profile? Links between cognition and behaviour. *Journal of Autism and Developmental Disorders*, 41(3), 357–363.

- Banerjee, T. D., Middleton, F., & Faraone, S. V. (2007). Environmental risk factors for attention-deficit hyperactivity disorder. *Acta Paediatrica*, 96(9), 1269–1274.
- Biederman, J., & Faraone, S. V. (2005). Attention-deficit hyperactivity disorder. *Lancet*, 366(9481), 237–248.
- Charnsil, C. (2011). Efficacy of atomoxetine in children with severe autistic disorders and symptoms of ADHD: An open-label study. *Journal of Attention Disorders*, 15(8), 684–689.
- Coghill, D. (2010). The impact of medications on quality of life in attention-deficit hyperactivity disorder: A systematic review. *CNS Drugs*, 24(10), 843–866.
- Cornforth, C., Sonuga-Barke, E., & Coghill, D. (2010). Stimulant drug effects on attention deficit/hyperactivity disorder: A review of the effects of age and sex of patients. *Current Pharmaceutical Design*, 16(22), 2424–2433.
- Crichton, A. (2008). An inquiry into the nature and origin of mental derangement: On attention and its diseases. *Journal of Attention Disorders*, 12(3), 200–204. Discussion: 205–206.
- Curatolo, P., Paloscia, C., D'Agati, E., Moavero, R., & Pasini, A. (2009). The neurobiology of attention deficit/hyperactivity disorder. *European Journal of Paediatric Neurology*, 13(4), 299–304.
- Fischer, M., Barkley, R. A., Smallish, L., & Fletcher, K. (2002). Young adult follow-up of hyperactive children: Self-reported psychiatric disorders, comorbidity, and the role of childhood conduct problems and teen CD. *Journal of Abnormal Child Psychology*, 30(5), 463–475.
- Furman, L. M. (2008). Attention-deficit hyperactivity disorder (ADHD): Does new research support old concepts? *Journal of Child Neurology*, 23(7), 775–784.
- Garnock-Jones, K. P., & Keating, G. M. (2009). Atomoxetine: A review of its use in attention-deficit hyperactivity disorder in children and adolescents. *Paediatric Drugs*, 11(3), 203–226.
- Ghanizadeh, A. (2007). Educating and counseling of parents of children with attention-deficit hyperactivity disorder. *Patient Education and Counseling*, 68(1), 23–28.
- Ghanizadeh, A. (2010). Factor analysis on ADHD and autism spectrum disorder DSM-IV-derived items shows lack of overlap. *European Child & Adolescent Psychiatry*, 19(10), 797–798.
- Ghanizadeh, A. (2011). SNAP-25 may mediate the association of lead exposure and ADHD. *European Journal of Paediatric Neurology*, 15(3), 280–281.
- Ghanizadeh, A., & Jafari, P. (2010). Risk factors of abuse of parents by their ADHD children. *European Child & Adolescent Psychiatry*, 19(1), 75–81.
- Ghanizadeh, A., & Zarei, N. (2010). Are GPs adequately equipped with the knowledge for educating and counseling of families with ADHD children? *BMC Family Practice*, 11, 5.
- Ghanizadeh, A., Bahredar, M. J., & Moeini, S. R. (2006). Knowledge and attitudes towards attention deficit hyperactivity disorder among elementary school teachers. *Patient Education and Counseling*, 63(1–2), 84–88.
- Ghanizadeh, A., Mohammadi, M. R., & Moini, R. (2008). Comorbidity of psychiatric disorders and parental psychiatric disorders in a sample of Iranian children with ADHD. *Journal of Attention Disorders*, 12(2), 149–155.
- Grzadzinski, R., Di Martino, A., Brady, E., Mairena, M. A., O'Neale, M., Petkova, E., et al. (2011). Examining autistic traits in children with ADHD: Does the autism spectrum extend to ADHD? *Journal of Autism and Developmental Disorders*, 41(9), 1178–1191.
- Handen, B. L., Johnson, C. R., & Lubetsky, M. (2000). Efficacy of methylphenidate among children with autism and symptoms of attention-deficit hyperactivity disorder. *Journal of Autism and Developmental Disorders*, 30(3), 245–255.
- Hoffmann, H. (1948). *Der Struwwelpeter. Oder lustige Geschichten und drollige Bilder für Kinder von 3 bis 6 Jahren.* Loewes. Stuttgart: Frankfurter Originalausgabe.
- Johnston, K., Madden, A. K., Bramham, J., & Russell, A. J. (2011). Response inhibition in adults with autism spectrum disorder compared to attention deficit/hyperactivity disorder. *Journal of Autism and Developmental Disorders*, 41(7), 903–912.
- King, S., Griffin, S., Hodges, Z., Weatherly, H., Asseburg, C., Richardson, G., et al. (2006). A systematic review and economic model of the effectiveness and cost-effectiveness of methylphenidate, dexamfetamine and atomoxetine for the treatment of attention deficit hyperactivity disorder in children and adolescents. *Health Technology Assessment*, 10(23), iii–iv. xiii–146.
- Klassen, A. F., Miller, A., & Fine, S. (2004). Health-related quality of life in children and adolescents who have a diagnosis of attention-deficit/hyperactivity disorder. *Pediatrics*, 114(5), e541–e547.
- Lahey, B. B., Applegate, B., McBurnett, K., Biederman, J., Greenhill, L., Hynd, G. W., et al. (1994). DSM-IV field trials for attention deficit hyperactivity disorder in children and adolescents. *The American Journal of Psychiatry*, 151(11), 1673–1685.
- Lange, K. W., Reichl, S., Lange, K. M., Tucha, L., & Tucha, O. (2010). The history of attention deficit hyperactivity disorder. *Attention Deficit Hyperactivity Disorder*, 2(4), 241–255.
- Murray, M. J. (2010). Attention-deficit/hyperactivity disorder in the context of autism spectrum disorders. *Current Psychiatry Reports*, 12(5), 382–388.
- Nigg, J., Nikolas, M., & Burt, S. A. (2010). Measured gene-by-environment interaction in relation to attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(9), 863–873.

- Posey, D. J., Wiegand, R. E., Wilkerson, J., Maynard, M., Stigler, K. A., & McDougle, C. J. (2006). Open-label atomoxetine for attention-deficit/hyperactivity disorder symptoms associated with high-functioning pervasive developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 16(5), 599–610.
- Rothenberger, A., & Neumärker, K. J. (2005). *Wissenschaftsgeschichte der ADHS*. Steinkopff, Darmstadt: Kramer-Pollnow im Spiegel der Zeit.
- Scahill, L., Aman, M. G., McDougle, C. J., McCracken, J. T., Tierney, E., Dziura, J., et al. (2006). A prospective open trial of guanfacine in children with pervasive developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 16(5), 589–598.
- Smalley, S. L., Loo, S. K., Yang, M. H., & Cantor, R. M. (2005). Toward localizing genes underlying cerebral asymmetry and mental health. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 135B(1), 79–84.
- Steinhausen, H. C. (2009). The heterogeneity of causes and courses of attention-deficit/hyperactivity disorder. *Acta Psychiatrica Scandinavica*, 120(5), 392–399.
- Stigler, K. A., Desmond, L. A., Posey, D. J., Wiegand, R. E., & McDougle, C. J. (2004). A naturalistic retrospective analysis of psychostimulants in pervasive developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 14(1), 49–56.
- Still, G. F. (1902). Some abnormal psychical conditions in children: The Goulstonian lectures. *Lancet*, 1902, 1008–1012.
- Tripp, G., & Wickens, J. R. (2009). Neurobiology of ADHD. *Neuropharmacology*, 57(7–8), 579–589.
- van den Hoofdakker, B. J., van der Veen-Mulders, L., Sytema, S., Emmelkamp, P. M., Minderaa, R. B., & Nauta, M. H. (2007). Effectiveness of behavioral parent training for children with ADHD in routine clinical practice: A randomized controlled study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46(10), 1263–1271.
- Vaughan, B., Fegert, J., & Kratochvil, C. J. (2009). Update on atomoxetine in the treatment of attention-deficit/hyperactivity disorder. *Expert Opinion on Pharmacotherapy*, 10(4), 669–676.
- Wehmeier, P. M., Schacht, A., & Barkley, R. A. (2010). Social and emotional impairment in children and adolescents with ADHD and the impact on quality of life. *The Journal of Adolescent Health*, 46(3), 209–217.
- World Health Organization (WHO). (1992). *The ICD-10 classification of mental and behavioural disorders. Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organization (WHO).
- Yoo, J. H., Valdovinos, M. G., & Williams, D. C. (2007). Relevance of donepezil in enhancing learning and memory in special populations: A review of the literature. *Journal of Autism and Developmental Disorders*, 37(10), 1883–1901.

Attention Network Tests in ASD

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Definition

Attention has been conceptualized as a multi-component system comprised of three isolable but interacting networks: alerting, orienting, and executive control (Posner and Petersen 1990). Alerting refers to the achievement and maintenance of a state of readiness to respond, orienting refers to the selection of an input pathway for further processing, and executive control refers to resolution of conflict between competing inputs and responses. The Attention Network Test (ANT), first described by Fan et al. (2002), was developed as a computerized tool for assessing the efficacy of each network.

In the ANT, participants are asked to fixate on a central cross responding to target arrows, which appear either above or below fixation. Targets are sometimes preceded by asterisks appearing at the center of the screen (center cue), above and below fixation simultaneously (double cue), or at the location of a subsequent target (spatial cue). In other words, cues may predict the onset (alerting cues) and/or location (orienting cues) of the target arrow. Finally, target arrows are flanked by arrows pointing in the same direction, arrows pointing in the opposite direction, or neutral bars. Network scores are typically calculated as differences in mean reaction time (RT) in opposing conditions. The orienting effect is the RT difference between center cue and spatial cue conditions, the alerting

effect is the RT difference between no-cue and double-cue conditions, and the executive control effect is the RT difference between congruent and flanker conditions.

Historical Background

Since the ANT was first introduced by Fan et al. (2002), substantial evidence has emerged supporting the biological validity of the three-network model. Unique neural patterns associated with each network have been identified using both electroencephalography (Fan et al. 2007) and functional magnetic resonance imaging (Fan et al. 2005). Furthermore, genetic studies suggest that there are heritable factors for executive control, as measured by the ANT (Fan et al. 2001).

Although the three attention networks are biologically and functionally distinct, and network scores are reliable across sessions (Ishigami and Klein 2010), frequently reported interaction effects between cue and flanker type suggest that these networks are not entirely independent (Fan et al. 2002; Ishigami and Klein 2010; Rueda et al. 2004). True independence is theoretically unlikely, assuming there is constant communication among functionally distinct brain regions and networks. Indeed, variations of the ANT have provided compelling evidence that the processes of orienting, alerting, and executive control do, in fact, modulate one another (e.g., Callejas et al. 2005; Fan et al. 2009; Fuentes and Campoy 2008).

Variants of the ANT have been developed to suit a range of purposes. For example, the child-ANT (ANT-C) features colorful, fish-shaped stimuli, which are meant to be more engaging for younger participants (Rueda et al. 2004). A lateralized version of the ANT (Greene et al. 2008) has been used to effectively isolate attentional processes in the left and right hemispheres. To better detect network differences, a revised version of the original ANT (ANT-R; Fan et al. 2009) modified a number of parameters, such as visual angle, cue-to-target interval, target duration, and target placement. The ANT-R also included orienting cues presented at locations

other than that of the target, known as “invalid” cues. In another version known as the ANT-interaction (ANT-I; Callejas et al. 2004), 50% of all spatial cues are invalid (i.e., they are “uninformative”). This version of the task additionally introduced auditory alerting tones to facilitate exploring the interaction between alerting and orienting networks. Recently, the ANT has been adapted into a game-like format in an effort to improve engagement relative to previous versions of the test. In the AttentionTrip (Klein et al. 2017), the participant uses a wheel to steer a spaceship through a wormhole while “shooting” target stimuli as they appear on the screen. This version of the task has been used with high-functioning adults on the autism spectrum (Mash et al. 2018) and may also demonstrate utility in research settings with young or lower-functioning individuals.

Current Knowledge

To date, there is a relatively small but growing body of literature reporting attention network scores in individuals with ASD. These studies have inconsistently reported significant differences between ASD and typically developing (TD) groups in all three major networks. Furthermore, these experiments vary considerably with respect to method (i.e., version of the ANT used), outcome variables reported (accuracy, reaction time), sample size, and age of the sample. In several studies, the sample sizes are quite small (e.g., $N = 12$) and therefore have limited power to detect negative results. However, several general themes have emerged from the extant literature that may guide future research.

Orienting

Problems with attention orienting and disengagement have been well-documented in individuals with ASD across the life span (Sacrey et al. 2014). Reduced orienting efficiency has been corroborated by several studies using versions of the ANT. In a sample of children and adolescents with and without ASD (ages 8–19), Keehn et al. (2010) found smaller orienting network scores on

the original ANT in the ASD group, likely reflecting reduced benefit of an orienting cue compared to the TD group. Less efficient orienting was similarly reported by Mutreja et al. (2016) in younger children with ASD (5–11 years) using the ANT-C. In a very small sample of older adolescents (six ASD and six TD, ages 16–17), Hames et al. (2016) did not find any significant group differences on the ANT-C with respect to orienting reaction time. However, they reported that the ASD group was more error-prone on orienting trials and that they tended to over-recruit brain regions typically associated with executive control during the orienting task. Although the above studies appear to implicate atypical orienting in younger individuals with ASD, other work has found relatively typical orienting on the ANT in both children (Ip et al. 2017; Samyn et al. 2017) and young adults (Fan et al. 2012; Mash et al. 2018) on the autism spectrum. Considering the critical role of attention orienting in early social development (Keehn et al. 2013), this is an important area of clarification for future research.

Alerting

Alerting network scores may reflect changes in both tonic and phasic alertness. Tonic alertness refers to intrinsic arousal, whereas phasic alertness is associated with rapid changes induced by a stimulus. Atypically large network scores may suggest reduced tonic alertness, resulting in slower reaction times in the absence of an alerting cue. Very small network scores, on the other hand, may be interpreted as reduced phasic alertness, resulting in only marginal improvements in reaction time in the presence of an alerting cue. In a study comparing typically developing children to those with ASD and attention-deficit/hyperactivity disorder (ADHD), alerting network scores on the ANT-I were significantly larger in the ADHD group, but were comparable in the ASD and TD groups (Samyn et al. 2017). A study using the original ANT reported no significant group differences in alerting between ASD and TD children and adolescents, but alerting network score size was associated with symptom severity in the ASD group. In contrast,

Mash et al. (2018) reported reduced alerting network scores in young adults using the original ANT, but not the AttentionTrip. In another sample of young adults tested on the ANT-R, Fan et al. (2012) reported that the ASD group made more errors in the absence of a cue, suggesting potentially poorer tonic alertness. Furthermore, using fMRI, they found that alerting errors were associated with reduced brain activity in the medial frontal gyri and caudate. As with orienting network scores, some research reported no significant group differences in alerting networks in ASD (Ip et al. 2017; Mutreja et al. 2016; Ridderinkhof et al. 2018; Samyn et al. 2017).

Executive Control

In general, studies of both children and adults measuring reaction time differences between congruent and incongruent flanker conditions on various versions of the ANT have found similar executive control networks in typical development and ASD (Fan et al. 2012; Hames et al. 2016; Ip et al. 2017; Keehn et al. 2010; Mash et al. 2018; Mutreja et al. 2016; Ridderinkhof et al. 2018; Samyn et al. 2017). One of these studies described an inverse relationship between IQ and the size of the executive control network in children and adolescents with ASD (Keehn et al. 2010), suggesting that inefficient executive processes (i.e., slower reaction time on incongruent trials) may be directly related to general cognitive ability. Supporting this possibility, they did not find any relationship between executive control and ASD symptoms. This study additionally reported that the alerting and executive control networks demonstrated unusually high interdependence in ASD individuals. The authors speculated that this might reflect compensatory executive strategies to control alertness in individuals with poorer intrinsic regulation of arousal. Although reaction times tend to be similar in ASD and TD individuals, there is some evidence that ASD individuals show significantly reduced accuracy on incongruent trials relative to congruent trials, resulting in larger accuracy difference scores. Mutreja et al. (2016) reported this effect in children (ages 5–11) using the ANT-C; a similar finding that did not reach statistical significance

was reported in a larger sample of slightly older group of individuals aged 8–23 (Ridderinkhof et al. 2018). Using the ANT-R, Fan et al. (2012) demonstrated poorer executive control accuracy in adults with ASD; further, they found that less accurate performance on the flanker component of the task was associated with reduced activation in the anterior cingulate cortex and more severe language and communication symptoms in the ASD group. Therefore, although individuals with ASD do not appear to respond more slowly to incongruent flankers, error analysis suggests that they may trade speed for accuracy on this task. Furthermore, it appears that accuracy, but not reaction time, may relate to behavioral and brain markers of ASD.

Future Directions

Studies examining attention using ANT-like tasks have provided some insight into attention network function in individuals with ASD. For example, participants on the autism spectrum generally tend to demonstrate reduced orienting efficiency, as well as poorer task accuracy when resolving conflict. However, even these relatively well-established findings have not been consistent across studies. Although ANT tasks are increasingly used and reported in ASD research, this body of literature remains fairly small and heterogeneous. There is great deal of variability with respect to participant age, sample size, ANT version, and outcome measures reported, which complicates direct comparison of findings. However, further study with adequate sample sizes may help to clarify the details of some of the emerging themes in this field.

A main disadvantage of the ANT is its length and repetitive nature. This may pose a challenge for populations that may have difficulty with sustained attention. Often, such individuals are of greatest interest to researchers studying attention. However, insufficient or variable effort throughout the entire task may preclude accurate interpretation of the resulting data. A recently developed, game-like version of the ANT (the AttentionTrip; Klein et al. 2017) may help to mitigate problems with engagement in

younger or cognitively impaired groups. The AttentionTrip has been used successfully in high-functioning adults on the autism spectrum (Mash et al. 2018), but future work may establish whether it can improve engagement in children or lower-functioning participants with ASD.

See Also

- ▶ Arousal
- ▶ Attention
- ▶ Cognitive Skills
- ▶ Executive Function (EF)
- ▶ Orienting Response

References and Reading

- Callejas, A., Lupiáñez, J., & Tudela, P. o. (2004). The three attentional networks: On their independence and interactions. *Brain and Cognition*, 54(3), 225–227. <https://doi.org/10.1016/j.bandc.2004.02.012>.
- Callejas, A., Lupiáñez, J., Funes, M. J., & Tudela, P. (2005). Modulations among the alerting, orienting and executive control networks. *Experimental Brain Research*, 167(1), 27–37. <https://doi.org/10.1007/s00221-005-2365-z>.
- Fan, J., Wu, Y., Fossella, J. A., & Posner, M. I. (2001). Assessing the heritability of attentional networks. *BMC Neuroscience*, 2, 14. <https://doi.org/10.1186/1471-2202-2-14>.
- Fan, J., McCandliss, B. D., Sommer, T., Raz, A., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience*, 14(3), 340–347. <https://doi.org/10.1162/089892902317361886>.
- Fan, J., McCandliss, B. D., Fossella, J., Flombaum, J. I., & Posner, M. I. (2005). The activation of attentional networks. *NeuroImage*, 26(2), 471–479. <https://doi.org/10.1016/j.neuroimage.2005.02.004>.
- Fan, J., Byrne, J., Worden, M. S., Guise, K. G., McCandliss, B. D., Fossella, J., & Posner, M. I. (2007). The relation of brain oscillations to attentional networks. *Journal of Neuroscience*, 27(23), 6197–6206. <https://doi.org/10.1523/JNEUROSCI.1833-07.2007>.
- Fan, J., Gu, X., Guise, K. G., Liu, X., Fossella, J., Wang, H., & Posner, M. I. (2009). Testing the behavioral interaction and integration of attentional networks. *Brain and Cognition*, 70(2), 209–220. <https://doi.org/10.1016/j.bandc.2009.02.002>.
- Fan, J., Bernardi, S., Van Dam, N. T., Anagnostou, E., Gu, X., Martin, L., . . . Hof, P. R. (2012). Functional deficits of the attentional networks in autism. *Brain and Behavior*, 2(5), 647–660. <https://doi.org/10.1002/brb3.90>.

- Fuentes, L. J., & Campoy, G. (2008). The time course of alerting effect over orienting in the attention network test. *Experimental Brain Research*, 185(4), 667–672. <https://doi.org/10.1007/s00221-007-1193-8>.
- Greene, D. J., Barnea, A., Herzberg, K., Rassiss, A., Neta, M., Raz, A., & Zaidel, E. (2008). Measuring attention in the hemispheres: The lateralized attention network test (LANT). *Brain and Cognition*, 66(1), 21–31. <https://doi.org/10.1016/j.bandc.2007.05.003>.
- Hames, E. C., Rajmohan, R., Fang, D., Anderson, R., Baker, M., Richman, D. M., & O'Boyle, M. (2016). Attentional networks in adolescents with high-functioning autism: An fMRI investigation. *The Open Neuroimaging Journal*, 10, 102–110. <https://doi.org/10.2174/1874440001610010102>.
- Ip, H. H. S., Lai, C. H.-Y., Wong, S. W. L., Tsui, J. K. Y., Li, R. C., Lau, K. S.-Y., & Chan, D. F. Y. (2017). Visuospatial attention in children with Autism Spectrum Disorder: A comparison between 2-D and 3-D environments. *Cogent Education*, 4(1), 1307709. <https://doi.org/10.1080/2331186X.2017.1307709>.
- Ishigami, Y., & Klein, R. M. (2010). Repeated measurement of the components of attention using two versions of the Attention Network Test (ANT): Stability, isolability, robustness, and reliability. *Journal of Neuroscience Methods*, 190(1), 117–128. <https://doi.org/10.1016/j.jneumeth.2010.04.019>.
- Keehn, B., Lincoln, A. J., Müller, R.-A., & Townsend, J. (2010). Attentional networks in children and adolescents with autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 51(11), 1251–1259. <https://doi.org/10.1111/j.1469-7610.2010.02257.x>.
- Keehn, B., Müller, R.-A., & Townsend, J. (2013). Atypical attentional networks and the emergence of autism. *Neuroscience and Biobehavioral Reviews*, 37(2), 164–183. <https://doi.org/10.1016/j.neubiorev.2012.11.014>.
- Klein, R. M., Hassan, T., Wilson, G., Ishigami, Y., & Mülle, J. (2017). The AttentionTrip: A game-like tool for measuring the networks of attention. *Journal of Neuroscience Methods*, 289, 99–109. <https://doi.org/10.1016/j.jneumeth.2017.07.008>.
- Mash, L. E., Klein, R. M., & Townsend, J. (2018). Brief report: A gaming approach to the assessment of attention networks in autism spectrum disorder and typical development. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-018-3635-5>.
- Mutreja, R., Craig, C., & O'Boyle, M. W. (2016). Attentional network deficits in children with autism spectrum disorder. *Developmental Neuropsychology*, 19(6), 389–397. <https://doi.org/10.3109/17518423.2015.1017663>.
- Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, 13, 25–42. <https://doi.org/10.1146/annurev.ne.13.030190.000325>.
- Ridderinkhof, A., de Bruin, E. I., van den Driesschen, S., & Bogels, S. M. (2018). Attention in children with autism spectrum disorder and the effects of a mindfulness-based program. *Journal of Attention Disorders*. <https://doi.org/10.1177/1087054718797428>.
- Rueda, M. R., Fan, J., McCandliss, B. D., Halparin, J. D., Gruber, D. B., Lercari, L. P., & Posner, M. I. (2004). Development of attentional networks in childhood. *Neuropsychologia*, 42(8), 1029–1040. <https://doi.org/10.1016/j.neuropsychologia.2003.12.012>.
- Sacrey, L. A., Armstrong, V. L., Bryson, S. E., & Zwaigenbaum, L. (2014). Impairments to visual disengagement in autism spectrum disorder: A review of experimental studies from infancy to adulthood. *Neuroscience and Biobehavioral Reviews*, 47, 559–577. <https://doi.org/10.1016/j.neubiorev.2014.10.011>.
- Samyn, V., Roeyers, H., Bijttebier, P., & Wiersema, J. R. (2017). Attentional networks in boys with ADHD or autism spectrum disorder and the relationship with effortful control. *Journal of Attention Disorders*, 21(3), 228–239. <https://doi.org/10.1177/1087054712473183>.

Attention Process Training (APT) Program

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Definition

The Attention Process Training (APT and APT-II) program is a cognitive rehabilitation intervention that targets focused, sustained, selective, alternating, and divided attention (Sohlberg and Mateer 1987; Sohlberg et al. 2001). APT developers define focused attention as the ability to respond to specific stimuli. Sustained attention refers to the ability to consistently respond during a continuous or repetitive activity. Selective attention is the ability to activate and inhibit responses based on discrimination of stimuli. Alternating attention refers to aptitude for mental flexibility, and divided attention has been defined as the ability to engage in multiple tasks simultaneously.

In general, process training involves implementing a structured treatment program to improve attention skills in a variety of areas (Sohlberg et al. 2001). The APT materials consist of tasks that are hierarchically organized to target sustained, selective, alternating, and divided attention (Sohlberg et al. 2001). The hierarchical

structure is intended to allow for basic skills to be constantly utilized while developing and practicing more complex skills (Palmese and Raskin 2000). Auditory attention tapes and visual activities are used for some of the tasks. APT also includes exercises to facilitate generalization of skills to daily life (Palmese & Raskin). The APT approach has been referred to as process-specific cognitive rehabilitation because it is intended to improve particular types of attention skills and does not lead to improvements in overall cognitive functioning (Sohlberg and Mateer 1987).

Historical Background

APT was developed by Sohlberg and Mateer (1987) based on experimental attention literature, clinical observation, and patients' subjective reports of symptoms. It frames attention as a multidimensional cognitive capacity (Sohlberg & Mateer). The APT-II is an extension of the original APT and is designed to target more complex attention impairments (Murray et al. 2006).

Rationale or Underlying Theory

APT follows a process-specific approach to cognitive rehabilitation in that it is intended to improve functioning in distinct cognitive areas (Sohlberg and Mateer 1987). The rationale underlying APT is that learning specific skills may help improve some of the cognitive problems that result from acquired brain damage (Park et al. 1999). A process-specific approach can be contrasted with the functional adaptation and the general stimulation perspectives. The functional adaptation approach utilizes task analysis and changes in the environment to assist with the challenges associated with cognitive impairments. The general stimulation approach utilizes tasks that facilitate any type of cognitive processing. These prior approaches to cognitive rehabilitation have been criticized as leading to poor generalizability and lacking a theoretical orientation, respectively (Sohlberg and Mateer 1987; Sohlberg et al. 2001).

Goals and Objectives

The objectives of APT are to improve individuals' focused attention, sustained attention, selective attention, alternating attention, and divided attention skills following an acquired brain injury, although the program has also been used with other populations. Individualized treatment goals are created based on the needs of the client in each of these areas of attention.

Treatment Participants

Although APT was designed for use with individuals who have acquired brain injury and most published research on the APT has been based on this population, some researchers have examined the efficacy of APT for individuals with schizophrenia and aphasia. Little is known about the efficacy of the program with other populations. Some have suggested that APT could be beneficial for individuals with autism spectrum disorders (Ozonoff et al. 2005), although published efficacy research to date has not been conducted with this population.

Treatment Procedures

The APT program is comprised of a set of activities that have a common structure and that range in complexity and processing speed requirements (Sohlberg and Mateer 1987). Treatment goals are individualized based on the client's impairments in each of the attention areas targeted (i.e., sustained, selective, alternating, and divided). Each task is designed to offer practice in one or more levels of attention. The tasks are either client-paced or therapist-paced depending on the nature of the exercise (Park et al. 1999).

The APT-II includes general exercises, each requiring approximately 5 min to complete, for each of the specific areas of attention emphasized in the program (Palmese and Raskin 2000). Four types of activities are incorporated into APT:

visual cancelation, auditory cancelation, mental control, and daily life attentional activities (López-Luengo and Vázquez 2003). Within each exercise, there are tasks that increase in speed and difficulty. When the client completes the final activity for a particular sequence, he or she can advance to the next level. Each exercise is repeated until it is completed successfully according to specified criteria. Some researchers have noted that the linguistic demands of APT tasks need to be taken into account for treatment planning with patients who have language impairments (Murray et al. 2006).

In the area of sustained attention, examples of visual activities include cancelation tasks (e.g., crossing out all the *Ps* and *Cs* in a long series of letters) where the client is scored on completion time, omissions, and errors. Audio activities include tasks such as having the client press a button when he or she hears a target stimulus among a set of distracters (e.g., identifying items that are round from a list of words) and is scored for accuracy (Pero et al. 2006). For selective attention, tasks from sustained attention are included but with more irrelevant and distracting stimuli added (e.g., auditory stimuli recorded over a noisy background). Similar tasks are also incorporated into the alternating attention activities but with instructions to change the target stimuli every 15 seconds. The divided attention activities include completing the visual and auditory cancelation tasks simultaneously, as well as card sorting and Stroop tasks (Pero et al.). Solving math problems and identifying main ideas from paragraphs are also examples of APT tasks (Palmese and Raskin 2000).

The program does not specify a particular number of sessions but recommends that response time should be decreased by 35% before moving on to the next task and that the client achieve 85% accuracy on each task (Pero et al. 2006). Researchers examining the efficacy of the APT program have generally implemented the intervention for a range of 4–10 weeks at a frequency of one to nine sessions per week (e.g., Coelho 2005; Palmese and Raskin 2000; Sohlberg and Mateer 1987; Sohlberg et al. 2000).

Efficacy Information

There is some evidence that APT may lead to improvements in specific attentional skills but not in general cognitive functioning. Most of the efficacy research for APT has been based on single-case designs with small sample sizes (e.g., Coelho 2005; Murray et al. 2006; Palmese and Raskin 2000; Pero et al. 2006; Sohlberg and Mateer 1987), although a few studies have incorporated between-group designs with random assignment (e.g., López-Luengo and Vázquez 2003; Sohlberg et al. 2000).

In general, researchers have found some support for improvement on sustained, selective, and divided attention tasks, as well as reading comprehension, in certain situations (Boman et al. 2004; Coelho 2005; Kurtz et al. 2001; Murray et al. 2006; Palmese and Raskin 2000; Pero et al. 2006; Sohlberg et al. 2000; Sohlberg and Mateer 1987). However, studies have not consistently found evidence of improvement in attention skills resulting from APT (e.g., López-Luengo and Vázquez 2003; Silverstein et al. 2005).

For example, individuals with brain injury who have completed the APT program have shown improvement on the paced auditory serial addition task (PASAT; Gronwall 1977), a measure of sustained attention and information processing speed (Park et al. 1999). However, control subjects who did not receive APT also showed improvement on this task over time (Park et al.). Compared to individuals receiving brain injury education, those who received APT in another study made greater gains on the PASAT (Sohlberg et al. 2000).

Another task on which individuals with brain injury who have completed APT have shown improvement is the consonant trigrams activity (Park et al. 1999), which involves recalling three consonants heard after counting backward by threes. It is intended to measure memory under conditions of distraction.

Researchers have also found some support for improved performance on the Trails-B task for individuals with brain injury who completed APT compared to a group who completed a brain injury education program (Sohlberg et al. 2000). This task requires participants to draw lines connecting a

sequence of ascending numbers and letters (e.g., 1-A-2-B-3-C-4-D...).

In addition to attention tasks, some studies have examined performance on executive function tasks following the APT program in samples with brain injury and schizophrenia (López-Luengo and Vázquez 2003; Sohlberg et al. 2000). One such task on which participants have shown improvement after completing APT is on variations of the Stroop task (Stroop 1935; Mohlman 2008; Sohlberg et al. 2000).

Outcome Measurement

A variety of outcome measures including attention tasks, questionnaires, and participant interviews have been used in APT efficacy research. However the most commonly used outcome measures appear to be the paced auditory serial addition task (PASAT; Gronwall 1977), consonant trigrams (Peterson and Peterson 1959), Trails B, and variations of the Stroop task (Stroop 1935).

Paced Auditory Serial Addition Task

The PASAT measures rate of information processing and was designed to assess the rate and degree of progress for clients recovering from concussion (Gronwall 1977). It is comprised of a randomized presentation of an auditory digit sequence, and the participant is expected to add each new digit to the preceding one (Sohlberg et al. 2000). Subsequent trials are presented at increasingly faster rates. Scores can be calculated as the correct number of responses at each trial pace or average time per correct response (Gronwall 1977). The PASAT is considered to require two types of attention: sustained attention and the ability to identify and correct errors during the activity (Park et al. 1999). Some have questioned whether improvement on this task following APT is due to the intervention or is an effect of repeated testing (Pero et al. 2006).

Consonant Trigrams/Brown-Peterson Task

This task measures memory skills under conditions of distraction (Park et al. 1999). Individuals participating in this task hear three consonants followed by a number. They are then asked to

count backward by threes for a predetermined number of seconds (e.g., 3, 9, 18). After the set time has elapsed, the participant is expected to recall the three consonants heard at the beginning of the trial. Delays of varying lengths between the end of the counting backward and the instruction to recall the consonants are also incorporated into the assessment (Park et al.).

Trails B

Trails B was originally part of the Army Individual Test Battery and is a task that measures visual scanning, mental flexibility, planning abilities, and working memory (Corrigan and Hinkeldey 1987; Sohlberg et al. 2000). Participants are asked to draw lines connecting consecutively numbered and lettered circles and alternate between the two (e.g., in the order 1-A-2-B-3-C-4-D...). Trails B can be scored as number of seconds required to complete the task (Corrigan and Hinkeldey 1987).

The Stroop Task

The Stroop task measures the interference effects of conflicting stimuli (Stroop 1935). Participants are shown a list of color words and asked to name the colors in which the words are printed (e.g., red, yellow) and ignore the words themselves (e.g., naming “yellow” for the word “red” printed in yellow ink). The task can also be completed by having participants read the list of color words while ignoring the ink color in which they are printed. Many variations of this original task have been developed that utilize different types of conflicting stimuli (MacLeod 1991).

Qualifications of Treatment Providers

Psychologists, speech-language pathologists, occupational therapists, special education staff, and related professionals with appropriate training in cognitive rehabilitation would generally be considered qualified to implement APT.

See Also

- [Attention](#)
- [Auditory Discrimination](#)

- ▶ Auditory Processing
- ▶ Executive Function (EF)
- ▶ Information Processing Speed
- ▶ Memory
- ▶ Reaction Time
- ▶ Short-Term Memory
- ▶ Visual Processing
- ▶ Visual Scanning

References and Reading

- Boman, I.-L., Lindstedt, M., Hemmingsson, H., & Bartfai, A. (2004). Cognitive training in home environment. *Brain Injury*, 18, 985–995.
- Coelho, C. A. (2005). Direct attention training as a treatment for reading impairment in mild aphasia. *Aphasiology*, 19, 275–283.
- Corrigan, J. D., & Hinkeldey, N. S. (1987). Relationships between parts A and B of the trail making test. *Journal of Clinical Psychology*, 43, 402–409.
- Gronwall, D. M. A. (1977). Paced auditory serial-addition task: A measure of recovery from concussion. *Perceptual and Motor Skills*, 44, 367–373.
- Kurtz, M. M., Moberg, P. J., Mozley, L. H., Swanson, C. L., Gur, R. C., & Gur, R. E. (2001). Effectiveness of an attention- and memory-training program on neuropsychological deficits in schizophrenia. *Neurorehabilitation and Neural Repair*, 15, 75–80.
- López-Luengo, B., & Vázquez, C. (2003). Effects of attention process training on cognitive functioning of schizophrenic patients. *Psychiatry Research*, 119, 41–53.
- MacLeod, C. M. (1991). Half a century of research on the Stroop effect: An integrative review. *Psychological Bulletin*, 109, 163–203.
- Mohlman, J. (2008). More power to the executive? A preliminary test of CBT plus executive training for treatment of late-life GAD. *Cognitive and Behavioral Practice*, 15, 306–316.
- Murray, L. L., Keeton, R. J., & Karcher, L. (2006). Treating attention in mild aphasia: Evaluation of attention process training-II. *Journal of Communication Disorders*, 39, 37–61.
- Ozonoff, S., South, M., & Provençal, S. (2005). Executive functions. In F. R. Volkmar, R. Paul, A. Kiln, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders: Vol. 1, diagnosis, development, neurobiology, and behavior* (pp. 606–627). Hoboken: Wiley.
- Palmese, C. A., & Raskin, S. A. (2000). The rehabilitation of attention in individuals with mild traumatic brain injury, using the APT-II programme. *Brain Injury*, 14, 535–548.
- Park, N. W., Proulx, G.-B., & Towers, W. M. (1999). Evaluation of the attention process training programme. *Neuropsychological Rehabilitation*, 9, 135–154.
- Pero, S., Incoccia, C., Caracciolo, B., Zoccolotti, P., & Fornisano, R. (2006). Rehabilitation of attention in two patients with traumatic brain injury by means of ‘attention process training’. *Brain Injury*, 20, 1207–1219.
- Peterson, L. R., & Peterson, M. J. (1959). Short-term retention of individual verbal items. *Journal of Experimental Psychology*, 58, 193–198.
- Silverstein, S. M., Hatashita-Wong, M., Solak, B. A., Uhlhaas, P., Landa, Y., Wilkniess, S. M., et al. (2005). Effectiveness of a two-phase cognitive rehabilitation intervention for severely impaired schizophrenia patients. *Psychological Medicine*, 35, 829–837.
- Sohlberg, M. M., & Mateer, C. A. (1987). Effectiveness of an attention-training program. *Journal of Clinical and Experimental Neuropsychology*, 9, 117–130.
- Sohlberg, M. M., McLaughlin, K. A., Pavese, A., Hedrich, A., & Posner, M. I. (2000). Evaluation of attention process training and brain injury education in persons with acquired brain injury. *Journal of Clinical and Experimental Neuropsychology*, 22, 656–676.
- Sohlberg, M. M., Johnson, L., Paule, L., Raskin, S. A., & Mateer, C. A. (2001). *Attention process training-II: A program to address attentional deficits for persons with mild cognitive dysfunction* (2nd ed.). Wake Forest: Lash & Associates.
- Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, 18, 643–662.

Attention Tunnelling

- ▶ Monotropism: An Interest-Based Account of Autism

Attentional Disengagement

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Definition

The first of the three subfunctions necessary for visual-spatial orienting (disengage, shift, and engage); the removal of attention from and/or ocular engagement with a stimulus that enables a shift of attention from one location to another.

Historical Background

Attention and autism spectrum disorder. Although autism spectrum disorder (ASD) is

diagnosed on the basis of impairments in social interaction and communication as well as the presence of repetitive and stereotyped interests and behaviors (APA 2013), differences in attention have been noted as secondary or associated features since the disorder was first described (Asperger 1944; Kanner 1943). For example, in his original account, Asperger (1944) observed that:

We regularly find a disturbance of active attention in autistic children. Here we are not [...] talking about the common-or-garden problems of concentration. These are problems that we find in many [children with other developmental disabilities] who are constantly distracted from work by external stimuli [...]. Autistic children on the other hand are, from the start, not interested in directing their attention to outside stimuli [...]. They follow their own ideas, which are mostly far removed from ordinary concerns, and do not like to be distracted from their thoughts.

Likewise, for one of his original 11 cases, Kanner (1943) remarked that, “to get his attention almost requires one to break down a mental barrier between his inner consciousness and the outside world” (p. 218). However, it was not until the 1960s and 1970s that research focused on the attentional differences in individuals with ASD began in earnest with the work of Hutt et al. (1964), Hermelin and O’Connor (1964), as well as Lovaas et al. (1971). Later, pioneering research led by Michael Posner (1980) investigating *visual-spatial orienting* in neurotypical individuals as well as those with cortical and subcortical lesions (Posner et al. 1982) illuminated this basic cognitive process and its associated brain network. Based on this work, Posner et al. (1984) later outlined the subcomponents of visual-spatial orienting: disengaging, shifting, and reengaging attention. Attentional disengagement thus reflects the initial step of the orienting process and is a prerequisite for shifting and then engaging a new object, person, or location within one’s environment. Subsequent research using Posner’s cuing paradigm in ASD provided the first evidence of impaired nonsocial visual-spatial orienting and in particular showed deficits in disengaging attention (Casey et al. 1993; Townsend et al. 1996; Wainwright-Sharp and Bryson 1993).

Current Knowledge

How do we measure disengagement? In addition to the Posner cuing paradigm, attentional disengagement in ASD has primarily been measured using gap-overlap tasks, which examine differences in the latency of eye movements to peripheral targets appearing with, and without, a central stimulus (Saslow 1967). The time required to execute saccadic eye movements (also referred to as saccadic reaction time; SRT) is reduced when a fixated central stimulus is removed prior to (i.e., gap condition) or simultaneously with (i.e., baseline or step condition) the onset of a peripheral target compared to when the central stimulus remains on screen when the peripheral target appears (i.e., overlap condition). Attentional disengagement, as measured by the gap effect (i.e., overlap SRT – gap SRT), is associated with both attentional and oculomotor components, and arises from two distinct sources: (1) a generalized alerting effect as a consequence of the fixation offset, which cues participants about the impending peripheral target and (2) the release of ocular inhibition due to (a) removal of the foveal stimulus and (b) top-down preparation of a saccade (e.g., Kingstone and Klein 1993). Disengagement abilities have been measured using modified gap-overlap paradigms across the lifespan from infants at risk for ASD (because they have an older sibling diagnosed with the disorder) to adults diagnosed with ASD (see Sacrey et al. 2014, for review).

Attentional disengagement in ASD. Results of studies employing gap-overlap paradigms in ASD are mixed with evidence of equivalent (e.g., Fischer et al. 2016; Schmitt et al. 2014; Zalla et al. 2018), slower (Elison et al. 2013; Elsabbagh et al. 2013; Kawakubo et al. 2007; Kleberg et al. 2017; Landry and Bryson 2004; Sabatos-DeVito et al. 2016), and faster (van der Geest et al. 2001) disengagement. Evidence of impaired disengagement in ASD has received the most support from studies of high-risk infant siblings of children with ASD and has now been replicated by three separate research groups using unique cohorts of infants (Bryson et al. 2018; Elison et al. 2013; Elsabbagh et al. 2013). This

deficit in attentional disengagement emerges around the end of the first year of life and has been shown to persist in children (Kleberg et al. 2017; Landry and Bryson 2004; Sabatos-DeVito et al. 2016) and adults (Kawakubo et al. 2007). Further, while these studies examining attentional disengagement in ASD have almost exclusively used visual stimuli, Keehn et al. (2019) recently showed reduced disengagement efficiency in children with ASD in the auditory domain, suggesting that disengagement impairments in ASD are not specific to the visual modality. Interestingly, this impairment in attentional disengagement is essentially in accord with the early anecdotal accounts by Kanner (1943) and Asperger (1944). That is, difficulties disengaging attention may manifest themselves in becoming “stuck” and subsequently failing to “direct their attention to outside stimuli.”

However, as referenced above, not all investigations have reported impairments in attentional disengagement in ASD. Inconsistent findings may arise from a variety of sources including the heterogeneous nature of ASD, the ages investigated, and differences in task design. Regarding the latter point, Keehn et al. (2019) noted two methodological factors that may contribute to the presence or absence of disengagement differences in ASD: (1) the type of fixation stimulus (e.g., dynamic or static images) and (2) predictability in the sequence of stimuli. For example, disengagement impairments are more commonly reported in studies of infants and young children, which primarily employ dynamic stimuli to maintain engagement of these younger participants, whereas studies of older children, adolescents, and adults with ASD tend to use more basic stimuli (e.g., fixation cross, LEDs). Second, at least two key aspects of gap-overlap tasks can be varied to decrease/increase the predictability of the target onset: (1) whether the central stimulus has a fixed or a variable duration and (2) whether trial types are blocked or randomized. In both cases, studies with more predictable stimulus sequences – central stimulus present for fixed duration and blocked trial type (e.g., all overlap trials presented together) – commonly report no group differences in disengagement, whereas evidence of impaired

disengagement is more consistently found with unpredictable tasks. Furthermore, gap trials provide a predictable sequence of events: (1) fixation cross appears, (2) fixation cross is removed (fixed duration; e.g., 200 ms), and (3) peripheral target onset. Thus, disappearance of the central fixation in gap trials, regardless of study paradigm, always provides a fixed cue associated with the appearance of the target. Group differences in SRT are almost never present in the gap condition; rather, significantly larger gap effect scores in ASD (which are indicative of slower, less efficient disengagement) are the result of longer latencies to shift in overlap trials.

In sum, deficits in attentional disengagement appear to be present within the first year of life and persist across the lifespan in individuals with ASD. Although evidence of equivalent performance on measures of disengagement exists, these findings may result, in part, from specific methodological variations in task design. Together, these results suggest that ASD is associated with impairments in disengaging attention, which are likely exacerbated when engagement with the fixated stimulus is high and/or target onset is unpredictable. Further, while these deficits in attentional disengagement may manifest as subtle differences in the latency of eye movements during well-controlled laboratory experiments, it is likely that these differences are amplified in real-world contexts, which are engaging and unpredictable.

Neural substrates of attentional disengagement. The network of brain regions responsible for orienting attention include the superior parietal lobes, intraparietal sulci, temporal-parietal junction (TPJ), and the frontal eye fields (FEF), as well as the thalamus and superior colliculus (SC) (Corbetta et al. 2008; Petersen and Posner 2012). In particular, the right TPJ may be a critical hub connecting both cortical (i.e., FEF) and subcortical (i.e., SC) brain regions (Bogadhi et al. 2019) and is thought to play a key role in disengaging and reorienting attention. Bryson et al. (1990) previously hypothesized that ASD could be characterized as a developmental spatial neglect syndrome (with acquired spatial neglect in adults generally associated with posterior right

hemisphere brain lesions). More recently, these authors have shown asymmetrical disengagement deficits in high-risk infants. That is, consistent with their prediction of neglect-like patterns of behavior, infants later diagnosed with ASD showed atypically slowed left-directed SRT when the fixation stimulus remained on screen (i.e., overlap trials). This theory and associated empirical results are consistent with electrophysiological (e.g., Orekhova et al. 2009) and neuroimaging findings (e.g., Keehn et al. 2016) of atypical right hemisphere activation in individuals with ASD. However, the particular neural mechanism(s) underlying disengagement deficits in ASD remains unknown.

Clinical significance. Early adaptive allocation of attention to one's environment requires efficient attentional disengagement and shifting. Failure to respond to a caregiver's name call or touch or the appearance of a novel object may result in fewer learning opportunities and affect the development of higher-level cognitive and social communication abilities. High-risk infants later diagnosed with ASD exhibit impairments in attentional disengagement compared both high- and low-risk infants that do not develop ASD. The presence of these early deficits in attentional disengagement has led some authors to hypothesize that they may be one of many factors that contribute to the emergence of the heterogeneous ASD phenotype (Keehn et al. 2013). Findings from prior research has demonstrated that disengagement efficiency in ASD is associated joint attention abilities (Schietecatte et al. 2011), recognition of spoken words (Venker 2017), and emotional distress (Bryson et al. 2018). For example, if infants and toddlers are unable to disengage and shift their attention during early dyadic interactions, then they may not follow a caregiver's point (i.e., respond to joint attention bid) or direct their caregiver's attention to a new item in their environment (i.e., to initiate joint attention). Thus, basic nonsocial attentional processes, such as attentional disengagement, may play a role in the development of core sociocommunicative impairments in ASD. These subsequent impairments (e.g., with joint attention) may have downstream consequences with word learning and language

development and thus may alter developmental trajectories across a variety of domains.

Future Directions

Identifying underlying mechanisms of impaired disengagement. Although strong evidence now exists for slower attentional disengagement in ASD, the mechanisms underlying these differences remain unclear. Elucidating the neurofunctional underpinnings associated with early disengagement impairments in ASD is a necessary next step to understand why this deficit emerges, how it may be used to accurately identify infants at risk, and how to more effectively target this skill in early intervention.

Leveraging attentional disengagement for early diagnosis and intervention. If disengagement impairments are present early (within first year of life) and play a critical role in the development of ASD, then (1) disengagement deficits may be used as an early biobehavioral marker to identify infants at risk for ASD and (2) the development of attention-targeted early interventions may augment early disengagement skills and improve outcomes in children with ASD. Eye-tracking biomarkers for ASD risk that focus on preference for social compared to nonsocial stimuli have shown excellent specificity but poor sensitivity (Pierce et al. 2016). Similar research examining the utility of nonsocial attentional disengagement metrics for classifying ASD risk has not yet been published. Such research – especially in community-based high-risk samples – will assist the field in identifying whether attentional disengagement may be used a biobehavioral marker for ASD risk. Further, research examining the role of atypical attentional disengagement on the development of ASD symptoms will advance our understanding regarding the utility of early attention-targeted interventions. For example, if early disengagement difficulties result in delayed or impaired joint attention in ASD, then targeting these early attention skills may facilitate the acquisition of this pivotal skill (Forssman and Wass 2018) and potentially result in improved outcomes for children with ASD.

See Also

- ▶ Attention
- ▶ Joint Attention
- ▶ Selective Attention
- ▶ Orienting Response

References and Reading

- APA. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychological Association.
- Asperger, H. (1944). Die "Autistischen Psychopathen" im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117(1), 76–136.
- Bogadhi, A. R., Bollimunta, A., Leopold, D. A., & Krauzlis, R. J. (2019). Spatial attention deficits are causally linked to an area in macaque temporal cortex. *Current Biology*, 29(5), 726–736 e724.
- Bryson, S. E., Wainwright-Sharp, J. A., & Smith, I. M. (1990). Autism: A developmental spatial neglect syndrome? In J. T. Enns (Ed.), *The development of attention: Research and theory* (pp. 405–427). Amsterdam: Elsevier North-Holland.
- Bryson, S. E., Garon, N., McMullen, T., Brian, J., Zwaigenbaum, L., Armstrong, V., ... & Szatmari, P. (2018). Impaired disengagement of attention and its relationship to emotional distress in infants at high-risk for autism spectrum disorder. *J Clin Exp Neuropsychol*, 40(5), 487–501.
- Casey, B. J., Gordon, C. T., Mannheim, G. B., & Rumsey, J. M. (1993). Dysfunctional attention in autistic savants. *Journal of Clinical and Experimental Neuropsychology*, 15(6), 933–946.
- Corbetta, M., Patel, G., & Shulman, G. L. (2008). The reorienting system of the human brain: From environment to theory of mind. *Neuron*, 58(3), 306–324.
- Elison, J. T., Paterson, S. J., Wolff, J. J., Reznick, J. S., Sasson, N. J., Gu, H., ... Network, I. (2013). White matter microstructure and atypical visual orienting in 7-month-olds at risk for autism. *Am J Psychiatry*, 170(8), 899–908.
- Elsabbagh, M., Fernandes, J., Jane Webb, S., Dawson, G., Charman, T., & Johnson, M. H. (2013). Disengagement of visual attention in infancy is associated with emerging autism in toddlerhood. *Biological Psychiatry*, 74, 189–194.
- Fischer, J., Smith, H., Martinez-Pedraza, F., Carter, A. S., Kanwisher, N., & Kaldy, Z. (2016). Unimpaired attentional disengagement in toddlers with autism spectrum disorder. *Developmental Science*, 19(6), 1095–1103.
- Forssman, L., & Wass, S. V. (2018). Training basic visual attention leads to changes in responsiveness to social-communicative cues in 9-month-olds. *Child Development*, 89(3), e199–e213.
- Hermelin, B., & O'Connor, N. (1964). Effects of sensory input and sensory dominance on severely disturbed, autistic children and on subnormal controls. *British Journal of Psychology*, 55, 201–206.
- Hutt, C., Hutt, S. J., Lee, D., & Ounsted, C. (1964). Arousal and childhood autism. *Nature*, 204, 908–909.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kawakubo, Y., Kasai, K., Okazaki, S., Hosokawa-Kakurai, M., Watanabe, K., Kuwabara, H., ... & Maekawa, H. (2007). Electrophysiological abnormalities of spatial attention in adults with autism during the gap overlap task. *Clinical Neurophysiology*, 118(7), 1464–1471.
- Keehn, B., Muller, R. A., & Townsend, J. (2013). Atypical attentional networks and the emergence of autism. *Neuroscience and Biobehavioral Reviews*, 37(2), 164–183.
- Keehn, B., Nair, A., Lincoln, A. J., Townsend, J., & Muller, R. A. (2016). Under-reactive but easily distracted: An fMRI investigation of attentional capture in autism spectrum disorder. *Developmental Cognitive Neuroscience*, 17, 46–56.
- Keehn, B., Kadlaskar, G., McNally Keehn, R., & Francis, A. L. (2019). Auditory attentional disengagement in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49, 3999–4008.
- Kingstone, A., & Klein, R. M. (1993). Visual offsets facilitate saccadic latency: Does predisengagement of visuospatial attention mediate this gap effect? *Journal of Experimental Psychology: Human Perception and Performance*, 19(6), 1251–1265.
- Kleberg, J. L., Thorup, E., & Falck-Ytter, T. (2017). Reduced visual disengagement but intact phasic alerting in young children with autism. *Autism Research*, 10(3), 539–545.
- Landry, R., & Bryson, S. E. (2004). Impaired disengagement of attention in young children with autism. *Journal of Child Psychology and Psychiatry*, 45(6), 1115–1122.
- Lovaas, O. I., Schreibman, L., Koegel, R., & Rehm, R. (1971). Selective responding by autistic children to multiple sensory input. *Journal of Abnormal Psychology*, 77(3), 211–222.
- Orekhova, E. V., Stroganova, T. A., Prokofiev, A. O., Nygren, G., Gillberg, C., & Elam, M. (2009). The right hemisphere fails to respond to temporal novelty in autism: Evidence from an ERP study. *Clinical Neurophysiology*, 120(3), 520–529.
- Petersen, S. E., & Posner, M. I. (2012). The attention system of the human brain: 20 years after. *Annual Review of Neuroscience*, 35, 73–89.
- Pierce, K., Marinero, S., Hazin, R., McKenna, B., Barnes, C. C., & Malige, A. (2016). Eye tracking reveals abnormal visual preference for geometric images as an early biomarker of an autism spectrum disorder subtype associated with increased symptom severity. *Biological Psychiatry*, 79(8), 657–666.

- Posner, M. I. (1980). Orienting of attention. *The Quarterly Journal of Experimental Psychology*, 32(1), 3–25.
- Posner, M. I., Cohen, Y., & Rafal, R. D. (1982). Neural systems control of spatial orienting. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 298(1089), 187–198.
- Posner, M. I., Walker, J. A., Friedrich, F. J., & Rafal, R. D. (1984). Effects of parietal injury on covert orienting of attention. *Journal of Neuroscience*, 4(7), 1863–1874.
- Sabatos-DeVito, M., Schipul, S. E., Bulluck, J. C., Belger, A., & Baranek, G. T. (2016). Eye tracking reveals impaired attentional disengagement associated with sensory response patterns in children with autism. *Journal of Autism and Developmental Disorders*, 46(4), 1319–1333.
- Sacrey, L. A., Armstrong, V. L., Bryson, S. E., & Zwaigenbaum, L. (2014). Impairments to visual disengagement in autism spectrum disorder: A review of experimental studies from infancy to adulthood. *Neuroscience and Biobehavioral Reviews*, 47, 559–577.
- Saslow, M. G. (1967). Effects of components of displacement-step stimuli upon latency for saccadic eye movement. *Journal of the Optical Society of America*, 57(8), 1024–1029.
- Schietecatte, I., Roeyers, H., & Warreyn, P. (2011). Exploring the nature of joint attention impairments in young children with autism spectrum disorder: Associated social and cognitive skills. *Journal of Autism and Developmental Disorders*, 42(1), 1–12.
- Schmitt, L. M., Cook, E. H., Sweeney, J. A., & Mosconi, M. W. (2014). Saccadic eye movement abnormalities in autism spectrum disorder indicate dysfunctions in cerebellum and brainstem. *Molecular Autism*, 5(1), 47.
- Townsend, J., Harris, N. S., & Courchesne, E. (1996). Visual attention abnormalities in autism: Delayed orienting to location. *Journal of the International Neuropsychological Society*, 2(6), 541–550.
- van der Geest, J. N., Kemner, C., Camfferman, G., Verbaten, M. N., & van Engeland, H. (2001). Eye movements, visual attention, and autism: A saccadic reaction time study using the gap and overlap paradigm. *Biological Psychiatry*, 50(8), 614–619.
- Venker, C. E. (2017). Spoken word recognition in children with autism spectrum disorder: The role of visual disengagement. *Autism*, 21(7), 821–829.
- Wainwright-Sharp, J. A., & Bryson, S. E. (1993). Visual orienting deficits in high-functioning people with autism. *Journal of Autism and Developmental Disorders*, 23(1), 1–13.
- Zalla, T., Seassau, M., Cazalis, F., Gras, D., & Leboyer, M. (2018). Saccadic eye movements in adults with high-functioning autism spectrum disorder. *Autism*, 22(2), 195–204.

Attributions (First Order/Second Order)

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Synonyms

[Attribution of mental states](#)

Definition

Attribution is a concept in psychology referring to people's tendency to attribute traits and causes to help explain what they observe. First- and second-order attributions refer more specifically to the attribution of mental states to self or others to explain and predict observable behavior (see ► [“Theory of Mind”](#)). Attribution of mental states, such as beliefs and desires, has been widely studied in false belief paradigms (Frith and Frith 2010). First-order mental state attribution tasks require the participant to represent another person's thoughts about the world, e.g., Sally *thinks* the ball is in the basket. Second-order tasks require representation of one person's belief about another person's mental state, e.g., Sally *thinks* Ann *knows* the ball is in the box. An everyday life example of attribution of mental states would be when we understand whether someone is telling a joke or telling a lie: we attribute to the liar, but not to the joker, the intention to make us believe what he or she says. Several tests exist for assessing the ability to attribute mental states (e.g., Happé 1994; White et al. 2009).

A large body of research has demonstrated that most children and many adults with ASD find it difficult to make mental state attributions, especially attributing to another person a state of knowledge that is different from their own or from reality (Baron-Cohen et al. 2000). This may underlie a range of social and communicative

Attribution of Mental States

► [Attributions \(First Order/Second Order\)](#)

symptoms in ASD, such as overliteral language use/understanding, difficulty adapting conversation to listeners' interests/knowledge, and difficulty understanding deception. An interesting question in recent research (e.g., Williams and Happé 2009) is whether some people with ASD may have difficulty attributing mental states to *self*, with implications for self-awareness and the ability to reflect upon one's own thoughts and feelings.

Attribution of mental states has become a key task for use during functional neuroimaging investigations of brain differences in ASD. A range of different tasks suggest key regions including the medial prefrontal cortex are less activated in people with ASD compared to controls when attributing thoughts (in response to, e.g., animated shapes, story vignettes; Frith and Frith 2010).

See Also

► [Theory of Mind](#)

References and Reading

- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. J. (Eds.). (2000). *Understanding other minds*. Oxford: Oxford University Press.
- Frith, U., & Frith, C. (2010). The social brain: Allowing humans to boldly go where no other species has been. *Philosophical Transactions of the Royal Society B*, 365, 165–176.
- Happé, F. G. E. (1994). An advanced test of theory of mind: Understanding of story characters' thoughts and feelings by able autistic, mentally handicapped and normal children and adults. *Journal of Autism and Developmental Disorders*, 24, 129–154.
- White, S. J., Hill, E., Happé, F., & Frith, U. (2009). Revisiting the Strange Stories: revealing mentalising impairments in autism. *Child Development*, 80, 1097–1117.
- White, S. J., Coniston, D., Rogers, R., & Frith, U. (2011). Developing the Frith-Happé animations: A quick and objective test of theory of mind for adults with autism. *Autism Research*, 4, 149–154.
- Williams, D., & Happé, F. (2009). What did I say? Versus what did I think? Attributing false beliefs to self amongst children with and without autism. *Journal of Autism and Developmental Disorders*, 39(6), 865–873.

Atypical

► [Exceptionality](#)

Atypical Antipsychotics

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Synonyms

[Novel antipsychotics](#); [Second-generation antipsychotics \(SGAs\)](#)

Indications

Aripiprazole (Abilify)

Schizophrenia in adults and pediatric patients (age 13–17 years); Acute manic or mixed episodes of bipolar I disorder in adults and pediatric patients (age 10–17 years), alone or as an adjunct to lithium or valproate; Major depressive disorder in adults (adjunctive treatment); Agitation associated with schizophrenia or manic or mixed episodes of bipolar I disorder (adults).

Clozapine (Clozaril)

Acute schizophrenia; Acute schizoaffective disorder; Treatment-refractory schizophrenia; Maintenance therapy in schizophrenia; Manic episodes of bipolar disorder; Depression with psychotic features.

Olanzapine (Zyprexa)

Schizophrenia in adults and pediatric patients (age 13–17 years); Acute manic or mixed episodes of bipolar I disorder in adults and pediatric patients (age 13–17 years), alone or as an adjunct to lithium or valproate; Acute agitation in schizophrenia and mania in bipolar I disorder; in combination with fluoxetine for depressive episodes associated with bipolar I disorder in adults; in combination with fluoxetine for treatment-resistant depression (adults).

Olanzapine and Fluoxetine Hydrochloride (Symbyax)

Acute depressive episodes of bipolar I disorder in adults; Treatment-resistant depression in adults.

Paliperidone (Invega)

Schizophrenia; Acute treatment of schizoaffective disorder, alone or as an adjunct to mood stabilizers and/or antidepressants.

Quetiapine (Seroquel)

Schizophrenia, including global symptoms, positive symptoms, negative symptoms, cognition, and aggression; Bipolar disorder (adults); Major depressive disorder in adults (adjunctive treatment).

Risperidone (Risperdal)

Schizophrenia in adults and pediatric patients (age 13–17 years); Acute manic or mixed episodes of bipolar I disorder in adults, alone or as an adjunct to lithium or valproate; Acute manic or mixed episodes of bipolar I disorder in pediatric patients (age 10–17 years); Irritability associated with autistic disorder in pediatric patients (age 5–16 years).

Ziprasidone Hydrochloride (Geodon) and Ziprasidone Mesylate (Geodon)

Schizophrenia in adults; Acute manic or mixed episodes of bipolar I disorder in adults, alone or as an adjunct to lithium or valproate; Acute agitation of schizophrenia in adults

Mechanisms of Action

When considering mechanisms of action of antipsychotics, it is important to note that the

pathophysiologies of psychiatric conditions treated by these drugs (i.e., schizophrenia, bipolar disorder, and autism) are unknown; therefore, the precise mechanisms of action of the atypical antipsychotics are unknown.

Aripiprazole

Aripiprazole is a dopamine type 2 (D_2) receptor partial agonist, not a full antagonist like the other atypical antipsychotics. This drug acts as a D_2 receptor antagonist when coadministered with a dopamine (DA) agonist but acts as a D_2 receptor agonist when administered without another DA agonist. Aripiprazole acts as an antagonist in overactive DA pathways and an agonist in underactive DA pathways. This drug's antagonist activity at serotonin type 2A ($5-HT_{2A}$) receptors may cause reductions in extrapyramidal symptoms (EPS) and improve the negative symptoms of schizophrenia, and its partial agonist activity at serotonin type 1A ($5-HT_{1A}$) may cause improvement in the negative and cognitive symptoms of schizophrenia, depression, and anxiety.

Clozapine

Clozapine exhibits low affinity for and quick dissociation from dopamine type 2 (D_2) receptors and high affinity for the serotonin type 2A ($5-HT_{2A}$) and serotonin type 1C ($5-HT_{1C}$) receptors, adrenergic receptors, cholinergic receptors, and dopamine type 4 (D_4) receptors, mainly in the extrastriatal cortex as compared to the striatal cortex. This drug also increases the release of dopamine (DA) in the prefrontal cortex. This effect of the drug may alleviate the negative symptoms and cognitive deficits of schizophrenia since these two aspects of the disorder may result from dopaminergic hypoactivity in the prefrontal cortex.

Olanzapine

Olanzapine has high relative serotonin type 2A ($5-HT_{2A}$) receptor blocking activity compared to that of dopaminergic (DA) receptors. This drug increases expression of *c-fos* in the caudate nucleus and increases serum glutamate levels. Also, olanzapine increases brain glutamate levels in patients who exhibit improvement in the negative symptoms of schizophrenia.

Paliperidone

Paliperidone is a dopamine type 2 (D_2), serotonin type 2A ($5-HT_{2A}$), α_1 - and α_2 -adrenergic, and histaminergic 1 (H_1) receptor antagonist. This drug is expected to have a mechanism very similar to that of risperidone since it is the major active metabolite of that drug, although patients have been reported to have responded positively to paliperidone after failing to respond to an adequate trial of risperidone.

Quetiapine

Quetiapine exhibits a high relative blockade of serotonin type 2A ($5-HT_{2A}$), serotonin type 2B ($5-HT_{2B}$), and serotonin type 2C ($5-HT_{2C}$) receptors compared to that of dopamine (DA) receptors. This drug exhibits a greater degree of binding in the extrastriatal cortex than in the striatal cortex. Quetiapine has partial agonist activity at $5-HT_{2A}$ which causes an increased DA level in the mesocortical DA pathway in individuals in which this pathway is hypoactive, thereby causing improvement in the negative and cognitive symptoms of schizophrenia. Also, this compound exhibits brief, high occupancy of dopamine type 2 (D_2) receptors for 2–3 h after dose administration in patients who exhibit improvement in psychosis, extrapyramidal symptoms (EPS), and prolactin. Imaging studies show that this drug has means of 74% $5-HT_{2A}$ receptor binding and 30% D_2 receptor binding for 450 mg/day dosing and means of 76% $5-HT_{2A}$ receptor binding and 41% D_2 receptor binding for 750 mg/day dosing.

Risperidone

Risperidone acts as an antagonist at the serotonin type 2A ($5-HT_{2A}$), dopamine type 2 (D_2), α_1 - and α_2 -adrenergic, and histaminergic 1 (H_1) receptors. Selective $5-HT_{2A}$ antagonists block amphetamine- and phencyclidine-induced locomotor activity and thereby may improve symptoms of psychosis. Also, the dizocilpine-induced disruption of prepulse inhibition of $5-HT_{2A}$ antagonists may improve sensory gating deficits in schizophrenia which may be caused by glutamatergic dysregulation. The α -adrenergic antagonist activity may cause an increase in dopamine (DA) levels in the medial prefrontal cortex which may

improve negative symptoms and cognition in schizophrenia. Dopaminergic hypoactivity in the prefrontal cortex is a potential cause of negative symptoms and cognitive deficits in schizophrenia. The α -adrenergic antagonist activity of this drug also may reduce the risk for the development of extrapyramidal symptoms (EPS) and improve cognition in individuals with frontal dementias. When taken with haloperidol, the selective serotonin type 2 ($5-HT_2$) antagonism reduces neuroleptic-induced parkinsonism and akathisia by increasing DA metabolism in the striatum and preventing an increase in D_2 receptor density which causes a decrease in the effects of D_2 receptor blockade and DA supersensitivity.

Ziprasidone

The antipsychotic effects of ziprasidone may be due to the affinity of this drug for dopamine type 2 (D_2) receptors in the striatum and its strong antagonism for serotonin type 2A ($5-HT_{2A}$) receptors. The $5-HT_{2A}$ receptor antagonism of this drug and its strong serotonin type 1A ($5-HT_{1A}$) receptor agonism may improve the negative and cognitive symptoms of schizophrenia by facilitating the release of dopamine (DA) in the prefrontal cortex.

Specific Compounds and Properties

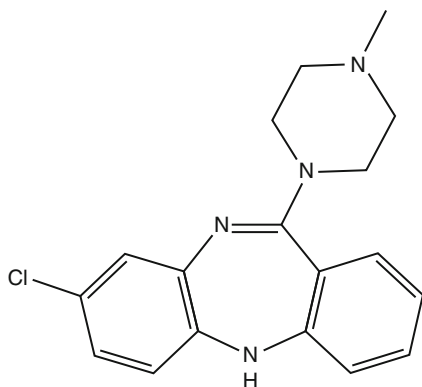
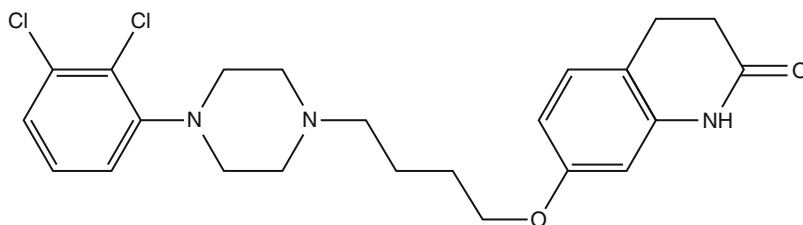
The specific compounds currently marketed in the United States that act as atypical antipsychotics are aripiprazole, clozapine, olanzapine, paliperidone, risperidone, ziprasidone, and quetiapine. The unique chemical structure of each atypical antipsychotic accounts for its binding activity as detailed in the “Mechanisms of Action” section of this entry. The chemical structures of these compounds are pictured in Figs. 1, 2, 3, 4, 5, 6, and 7

Clinical Use (Including Side Effects)

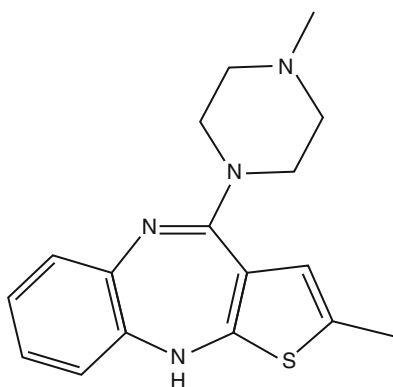
Aripiprazole

Aripiprazole is used in autistic disorder to improve symptoms of aggression, irritability, and self-injurious behavior. Doses used in studies range from 2.5 to 15 milligrams per day (mg/day).

Atypical Antipsychotics, Fig. 1 Chemical structure of aripiprazole



Atypical Antipsychotics, Fig. 2 Chemical structure of clozapine



Atypical Antipsychotics, Fig. 3 Chemical structure of olanzapine

Side effects of aripiprazole include nausea, weight gain, akathisia, headache, insomnia, agitation, anxiety, and mild transient somnolence.

Clozapine

Clozapine is used in autism spectrum disorders (ASDs) to improve symptoms of aggression. Doses of 276 mg/day in an adolescent and

283.33 mg/day in children have been used to treat ASDs. Side effects of clozapine include a very high risk of sedation; a high risk of anticholinergic effects, sialorrhea, orthostasis, and weight gain; a moderate risk of seizures and hematologic effects; a low risk of increased liver enzyme levels; and a very low risk of extrapyramidal symptoms (EPS) and neuroleptic malignant syndrome (NMS).

Olanzapine

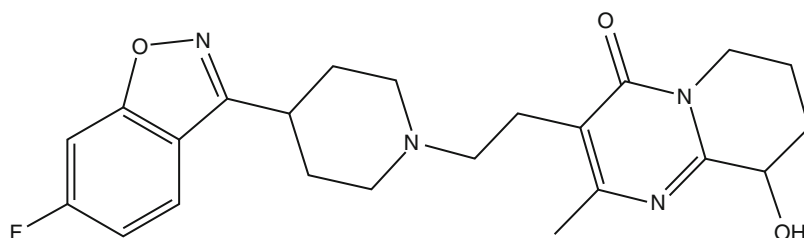
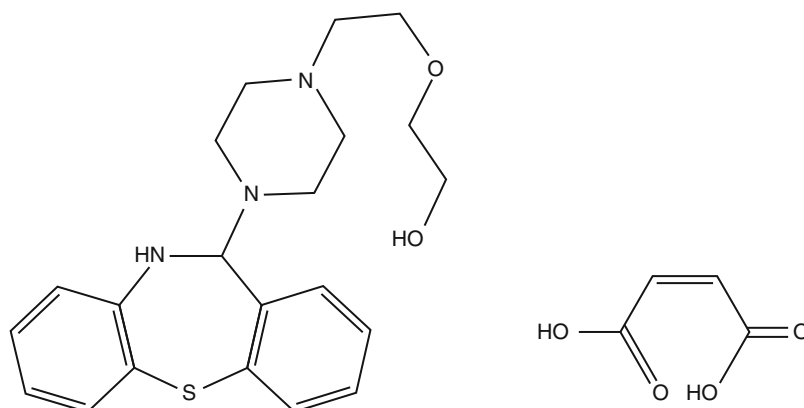
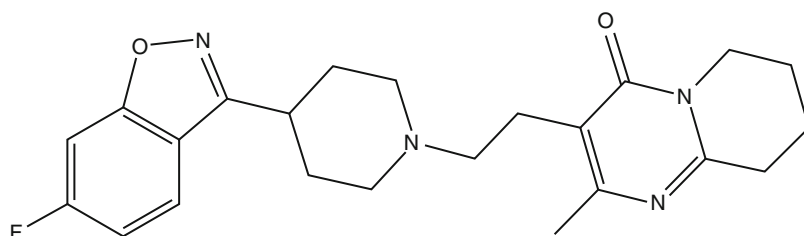
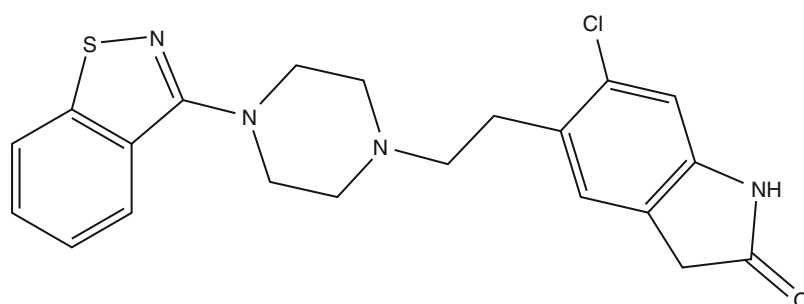
Olanzapine is used in autism spectrum disorders for global improvement of severe behavioral symptoms, overall symptoms of autism, motor restlessness/hyperactivity, social relatedness, affectual relations, sensory responses, language use, self-injurious behaviors, aggression, irritability, anxiety, and depression. The dose for this drug may be between 5 and 20 mg/day and is used in children, adolescents, and adults. Side effects of olanzapine include sedation and weight gain. Also, this drug has a moderate risk of orthostasis and anticholinergic effects; a low, dose-dependent risk of EPS; a low risk of increased liver enzyme levels; and a very low risk of TD, seizures, and hematologic effects.

Paliperidone

Paliperidone has been used in autism spectrum disorders to improve symptoms of irritability, including aggression, self-injurious behaviors, and tantrums. Doses of 6–12 mg/day have been used in adolescents with autism. Side effects of paliperidone include orthostatic hypotension, weight gain, weight loss, and sedation.

Quetiapine

Quetiapine is used in autism spectrum disorders (ASDs) to improve symptoms of aggression,

Atypical Antipsychotics,**Fig. 4** Chemical structure of paliperidone**Atypical Antipsychotics,****Fig. 5** Chemical structure of quetiapine**Atypical Antipsychotics,****Fig. 6** Chemical structure of risperidone**Atypical Antipsychotics,****Fig. 7** Chemical structure of ziprasidone

hyperactivity, and inattention. Doses used in studies of quetiapine for use in the treatment of ASDs include means of 225 mg/day and 477 mg/day in children and adolescents; a mean of 292 mg/day in adolescents; and a mean of 249 mg/day in a group of children, adolescents,

and adults. Side effects of quetiapine include agitation, sedation, weight gain, aggression, and sialorrhea. Also, this drug has a low risk of anticholinergic effects, orthostasis, and increased liver enzyme levels and a very low risk of EPS, NMS, seizures, and hematologic effects.

Risperidone

Risperidone is used in autistic disorder to improve symptoms of aggression, irritability, repetitive behavior and language, hyperactivity, social withdrawal, nonverbal communication, and social responsiveness. An effective dose for children with pervasive developmental disorder (PDD) may range from 1 to 1.2 mg/day, whereas an effective dose for children with autism may be 1.8 mg/day. An effective dose for adults with autism may be 2.9 mg/day. Side effects of risperidone include sedation, increased prolactin, weight gain, and hypersalivation. Also, this drug has a high risk for orthostasis; a moderate, dose-dependent risk of EPS; and a very low risk of tardive dyskinesia (TD), NMS, anticholinergic effects, seizures, hematologic effects, and elevated liver enzyme levels.

Ziprasidone

Ziprasidone is used in autism spectrum disorders to improve symptoms of aggression, irritability, and agitation. A dose used in studies of ziprasidone for use in the treatment of ASDs includes a mean of 59 mg/day in children and adolescents. Side effects of ziprasidone include sedation and mild weight gain. Also, this drug has a low risk of orthostasis and increased liver enzyme levels and a very low risk of EPS, anticholinergic effects, seizures, and hematologic effects.

References and Reading

- Barnard, L., Young, A. H., Pearson, J., Geddes, J., & O'Brien, G. (2002). A systematic review of the use of atypical antipsychotics in autism. *Journal of Psychopharmacology*, 16, 93–101.
- Biederman, J., Spencer, R., & Wilens, T. (2004). Psychopharmacology. In J. M. Wiener & M. K. Dulcan (Eds.), *The American psychiatric publishing textbook of child and adolescent psychiatry* (3rd ed., pp. 931–973). Washington, DC: American Psychiatric Publishing.
- Brown, C. S., Markowitz, J. S., Moore, T. R., & Parker, N. G. (1999). Atypical antipsychotics: Part II: Adverse effects, drug interactions, and costs. *The Annals of Pharmacotherapy*, 33, 210–217.
- Chen, N. C., Bedair, H. S., McKay, B., Bowers, M. B., Jr., & Mazure, C. (2001). Clozapine in the treatment of aggression in an adolescent with autistic disorder. *The Journal of Clinical Psychiatry*, 62, 479–480.
- Citrome, L. (2010). Paliperidone palmitate – Review of the efficacy, safety and cost of a new second-generation depot antipsychotic medication. *International Journal of Clinical Practice*, 64, 216–239.
- Daniel, D. G., Copeland, L. F., & Tamminga, C. (2006). Ziprasidone. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 297–305). Washington, DC: American Psychiatric Publishing.
- Goff, D. C. (2006). Risperidone. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 285–295). Washington, DC: American Psychiatric Publishing.
- Marder, S. R., & Wirshing, D. A. (2006). Clozapine. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 229–243). Washington, DC: American Psychiatric Publishing.
- Martinez, M., Marangell, L. B., & Martinez, J. M. (2011). Psychopharmacology. In R. E. Hales, S. C. Yudofsky, & G. O. Gabbard (Eds.), *Essentials of psychiatry* (3rd ed., pp. 455–524). Washington, DC: American Psychiatric Publishing, Inc..
- Miyamoto, S., Duncan, G. E., Marx, C. E., & Lieberman, J. A. (2005). Treatments for schizophrenia: A critical review of pharmacology and mechanisms of action of antipsychotic drugs. *Molecular Psychiatry*, 10, 79–104.
- Posey, D. J., Stigler, K. A., Erickson, C. A., & McDougale, C. J. (2008). Antipsychotics in the treatment of autism. *Science in Medicine*, 118, 6–14.
- Printz, D. J., & Lieberman, J. A. (2006a). Aripiprazole. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 277–283). Washington, DC: American Psychiatric Publishing.
- Printz, D. J., & Lieberman, J. A. (2006b). Quetiapine. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 263–275). Washington, DC: American Psychiatric Publishing.
- Schatzberg, A. F., Cole, J. O., & DeBattista, C. (2003). Antipsychotic drugs. In *Manual of clinical psychopharmacology* (4th ed., pp. 159–243). Washington, DC: American Psychiatric Publishing.
- Schultz, S. C., Olson, S., & Kotlyar, M. (2006). Olanzapine. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 245–275). Washington, DC: American Psychiatric Publishing.
- Stigler, K. A., Erickson, C. A., Mullet, J. E., Posey, D. J., & McDougale, C. J. (2010). Paliperidone for irritability in autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, 20, 75–78.
- Tsai, L. Y. (2004). Autistic disorder. In J. M. Wiener & M. K. Dulcan (Eds.), *The American psychiatric publishing textbook of child and adolescent psychiatry* (3rd ed., pp. 261–260). Washington, DC: American Psychiatric Publishing.
- U.S. Food and Drug Administration. (2010a). *Atypical antipsychotics drug information*. Retrieved from

<http://www.fda.gov/Drugs/DrugSafety/PostmarketDrugSafetyInformationforPatientsandProviders/ucm094303.htm>

U.S. Food and Drug Administration. (2010b). *Drugs@FDA*. Retrieved from <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm>

Atypical Autism

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Short Description or Definition

Atypical autism is often described as a subthreshold diagnosis, presenting with some symptoms of autism but insufficient to meet criteria for a diagnosis of childhood autism (or autistic disorder). Alternatively, atypical autism can be diagnosed when there is a late onset of symptomatology. Atypical autism (as defined by ICD-10) is seen as being equivalent to the DSM-IV-TR diagnostic category of pervasive developmental disorder not otherwise specified (PDD NOS). DSM-5 does not have a separate diagnostic category for PDD NOS.

Like PDD NOS, atypical autism is poorly defined, resulting in a research literature that can be difficult to interpret and conclusions difficult to reach. Atypical autism, as defined by the ICD, lacks operationalized diagnostic criteria, resulting in inconsistencies and variability in the way in which the diagnosis is applied. Although it now appears to be more common than autistic disorder, in general it remains poorly understood. This is likely due, in no small part, to the lack of a clear definition. Although it is often assumed that findings relating to autism apply to atypical autism, the lack of operationalized diagnostic criteria has undoubtedly hampered specific research into this diagnostic category and contributed to inconsistent findings across studies. Studies often fail to

describe how they operationalized or defined their samples of atypical autism or PDD NOS. The ICD-10 provides specifiers to further define the diagnosis of atypical autism (see section “[Categorization](#)”); however, studies generally do not use these specifiers. Difficulties therefore remain in interpreting and comparing findings across studies. The broadening of the PDD NOS category in DSM-IV (Volkmar et al. 2000) has also contributed to difficulties in interpretability of results across studies, although with DSM-IV-TR (American Psychiatric Association 2000) this was remedied. Further definition of atypical autism or PDD NOS in research (see, e.g., Mandy et al. (2011)) would assist with furthering knowledge in this area.

This entry will focus on research studies involving individuals with atypical autism. Where necessary, this is supplemented with research findings from samples with PDD NOS.

Categorization

The category of *pervasive developmental disorder* (PDD) was introduced in DSM-III (American Psychiatric Association 1980) and included the subthreshold diagnosis of *atypical PDD*, which subsequently became *pervasive developmental not otherwise specified* (PDD NOS) in DSM-III-R (American Psychiatric Association 1987). Reflecting thinking at the time, ICD-9 categorized autism (299.0 *Infantile Autism*) under the category of childhood psychoses and included a code for *other specified early childhood psychoses*, including *atypical childhood psychosis* (299.8) (World Health Organisation 1978). With the revision of these classification systems to the DSM-IV (American Psychiatric Association 2000) and ICD-10 (World Health Organisation 1992), the systems shared a common approach to coding and were seen as conceptually the same (Volkmar 1998).

The ICD-10 (World Health Organisation 1992) provides diagnostic criteria for *atypical autism* (F84.1) under the category of *pervasive developmental disorders*. The diagnosis is for cases

where age of onset is after the age of three (criteria the same for *childhood autism* except for age of onset), or all three sets of criteria for *childhood autism* are not met (subthreshold). Criteria in the domains of abnormalities in reciprocal social interaction, or communication, or restricted, repetitive, and stereotyped patterns of behavior, interests, and activities are the same as for *childhood autism* (F84.0) except that it is not necessary to meet the criteria for number of areas of abnormality. Specifiers can then be used to indicate *atypicality in age of onset* (F84.10), *atypicality in symptomatology* (F84.11), or *atypicality in both age of onset and symptomatology* (F84.12). The DSM-IV (American Psychiatric Association 2000) defines *PDD NOS* as including atypical autism.

The ICD-10 also has two additional diagnoses, namely, *other pervasive developmental disorder* (F84.8, with no diagnostic criteria specified) and *pervasive developmental disorder, unspecified* (F84.9). The latter disorder is defined as a residual category for cases where there is a lack of information or contradictory findings, but where symptomatology fits the general description for a pervasive developmental disorder. The ICD-10 diagnoses of *atypical autism*, *other pervasive developmental disorder*, and *pervasive developmental disorder, unspecified* are considered to be broadly equivalent to the DSM-IV-TR (American Psychiatric Association 2000) diagnosis of *PDD NOS*.

In the current DSM (DSM-5; American Psychiatric Association 2013), the category of *PDD NOS* has been subsumed under autistic spectrum disorder, with the instruction to give the DSM-5 diagnosis of autism spectrum disorder to those with a well-established diagnosis of *PDD NOS*. Concerns have been raised regarding whether children and adolescents with DSM-IV diagnoses of *PDD NOS* or ICD-10 diagnoses of atypical autism would meet the DSM-5 diagnostic criteria for autism spectrum disorder. Using draft criteria, a number of studies reported concerningly low rates (3–28.3%) of cases of *PDD NOS*/atypical autism meeting the DSM-5 criteria for autism spectrum disorder (Barton et al. 2013; Mandy et al. 2011; Mayes et al. 2013; McPartland et al.

2012). Kim et al. (2014) reported a higher rate (63%) and Huerta and colleagues found that the DSM-5 diagnostic criteria resulted in improved specificity compared to the DSM-IV criteria for *PDD NOS* (Huerta et al. 2012). It has been speculated that children without repetitive, restricted, or stereotyped behaviors previously diagnosed with *PDD NOS* may meet the diagnostic criteria for the new DSM-5 Social Communication Disorder category (Ozonoff 2012; Skuse 2012). Prospective research studies using the DSM-5 diagnostic criteria are needed to explore these issues.

Draft guidelines for ICD-11 (due for release in 2018), mirror the DSM-5, subsuming atypical autism into the single diagnostic category of autism spectrum disorder (WHO, GCP Network 2017).

Epidemiology

Atypical autism is rarely the focus of prevalence studies, and differing labels and combining of groups other than autistic disorder can make the extraction and interpretation of prevalence figures difficult. A number of population and birth cohort studies have included figures on the prevalence of atypical autism. The UK-based studies in children have reported differing prevalence figures of 10.5/10,000 (Lingam et al. 2003), 10.9/10,000 (Williams et al. 2008), and 27/10,000 (Baird et al. 2000), while a birth cohort study (6-year-olds) in Stockholm reported a prevalence of 22/10,000 (Fernell and Gillberg 2010). A study in the Faroe Islands (considered a genetic isolate) reported a population prevalence of atypical autism of 0.12%, while acknowledging that this is possibly an underestimate particularly in terms of higher functioning children (Ellefsen et al. 2007). A Danish population study reported separate prevalence rates for atypical autism (3.3/10,000) and *PDD NOS* (14.6/10,000), which when taken together are similar to those rates reported by Fernell and Gillberg (2010) and Baird et al. (2000). A South Korean study provided a prevalence estimate of 1% for *PDD NOS* (Kim et al. 2011). Using data from the national

Danish register, reported rates of Gender ratios have been reported by a very small number of studies, with a higher proportion of males with autistic disorder compared to atypical autism, 6.5:1 compared to 3.8:1 in Stockholm (Fennell and Gillberg 2010), and no reported gender differences between PDD NOS (85.3% male) and autistic disorder (85.9% male) in a birth cohort of 4–6-year-olds in Stafford in the UK (Chakrabarti and Fombonne 2005).

A series of review studies by Fombonne, most recently in 2009, reviewed 43 prevalence surveys, 17 of which provided separate estimates of the prevalence of atypical autistic syndromes (PDD NOS and atypical autism) (Fombonne 2009). Fourteen of these studies reported a higher prevalence of atypical autism syndromes compared to autistic disorder, 37.1/10,000 and 20.6/10,000 respectively. Like the prevalence of autism, the reported prevalence of atypical autism has increased over time. Similarly, this increase is typically discussed in relation to changes in diagnostic criteria, increased awareness, diagnostic substitution, changes in special education policies, and increases in the availability of services. What is, however, clear from these studies is that there is a significantly large population of children with atypical autism who have treatment needs similar to those of children with autism.

Natural History, Prognostic Factors, Outcomes

A small number of studies have investigated the early signs and symptoms in children later diagnosed with atypical autism, with mixed results. One study looked at first symptoms and diagnosis in children with atypical autism, comparing the parent-reported onset of symptomatology to that of children diagnosed with childhood autism (Oslejskova et al. 2007). Significant group differences were found in age of first symptoms, with parents of children with atypical autism reporting first symptoms at an average of 36.7 months (compared to 23.5 months for children with childhood autism). There were however no significant group differences in age at diagnosis. In contrast,

Walker et al. reported no difference between autism and PDD NOS in terms of age at which abnormalities were first identified by parents (2004). Two epidemiological studies found that atypical autism was diagnosed later than childhood autism, with atypical autism generally diagnosed at 5–6 years of age and childhood autism at 3–4 years (Fennell and Gillberg 2010; Lingam et al. 2003).

Research has demonstrated that outcome in autism and other pervasive developmental disorders is associated with the acquisition of expressive language skills by the age of 5–6 years, cognitive ability, and early social-communicative skills (Gillberg and Steffenburg 1987; Kobayashi et al. 1992; Mundy et al. 1990; Nordin and Gillberg 1998; Sigman and Ruskin 1999). Longitudinal studies have reported that initial diagnosis (i.e., atypical autism or PDD NOS compared to autistic disorder) is not related to outcomes (Baghdadli et al. 2007; Turner et al. 2006) and therefore has limited use in predicting developmental outcomes. However, Moulton et al. (2016) reported that a diagnosis of PDD NOS at age 2 was associated with better outcomes at age 4 relative to those children with a diagnosis of autistic disorder, likely due to lower rates of autism symptomatology, particularly restricted and repetitive behaviors.

Clinical Expression and Pathophysiology

The reliability and stability of the diagnoses of atypical autism and PDD NOS has been questioned. In a study of subtypes of pervasive developmental disorders in children, Mahoney et al. (1998) reported interrater agreement for diagnoses of Asperger's disorder, autism, and atypical autism across three raters. Kappa values revealed good agreement for the diagnosis of autism (0.55), Asperger's disorder (0.56), and non-PDD (0.67), but poor agreement in the case of atypical autism (0.18). Consistent with the results of studies in children with atypical autism, research in toddlers with autism and PDD NOS has reported good agreement between clinicians on the diagnosis of autism, but low rates of

agreement for PDD NOS (Chawarska et al. 2007; Stone et al. 1999).

In relation to diagnostic stability, research has focused on individuals with PDD NOS. While diagnoses of autistic disorder have been shown to be relatively stable in toddlers, the same is not true of PDD NOS (Chawarska et al. 2007; Stone et al. 1999; Turner et al. 2006; van Daalen et al. 2009). A meta-analysis of the diagnostic stability of PDD NOS reviewed eight studies, reporting higher rates of stability for a diagnosis of autistic disorder compared to PDD NOS (Rondeau et al. 2010). It was concluded that a diagnosis of PDD NOS prior to 36 months was unstable (35% stability) over time, highlighting the need for reassessment. It has been suggested that low diagnostic stability may be attributable to the later emergence of stereotyped and repetitive behaviors in young children (Kleinman et al. 2008; Suter et al. 2007).

The lack of operationalized diagnostic criteria for atypical autism and the variability in which the diagnosis is applied have possibly resulted in a significant amount of heterogeneity in the presentation of individuals; as such, there is as yet no consensus regarding the symptom profile for atypical autism or PDD NOS (Mandy et al. 2011). Two studies have examined symptom profiles in children with atypical autism, focusing on high-functioning children with atypical autism, Asperger's disorder, and childhood autism (Kanai et al. 2004; Kurita 1997). In a comparison of children with high-functioning atypical autism and childhood autism, symptom patterns were examined using the Childhood Autism Rating Scale (CARS) (Kurita et al. 1989), rated by clinicians blind to the child's diagnosis. The children with atypical autism scored significantly lower on the CARS total score. There were no significant group differences on 11 of the 15 CARS items. After controlling for IQ and total CARS score, the children with atypical autism were found to be significantly less impaired on two items of the CARS (relationships with people and general impressions) and were more impaired in anxiety reaction compared to the children with childhood autism. In a comparison of high-functioning atypical autism and Asperger's disorder, the Asperger's disorder group was significantly less

impaired than the atypical autism group on total CARS score, imitation, visual responsiveness, auditory responsiveness, and nonverbal communication (Kurita 1997). Overall, these findings are consistent with the idea of atypical autism being a subthreshold diagnosis for children with a significant degree of impairment, but not to the degree that criteria for childhood autism are met.

Further information on symptom presentation comes from studies with children with a diagnosis of PDD NOS. Consistent with the results of the studies with children with atypical autism, a number have reported generally finding children with PDD NOS to have significantly less impairment in the social, communication, and restricted and repetitive symptom domains compared to children with autistic disorder (Fodstad et al. 2009; Walker et al. 2004). de Bruin et al. (2006) reported that children with PDD NOS have similar cognitive profiles as children with autism, although in contrast Walker et al. (2004) found that children with PDD NOS scored better than children with autism on measures of adaptive behavior and nonverbal reasoning and problem-solving skills. An investigation of communication impairments using the Children's Communication Checklist (Bishop 1998) with children with high-functioning autism, Asperger's disorder, and PDD NOS found that while all groups demonstrated significantly more impairment than the typically developing control group, there was little difference across the autism subtypes. In a comprehensive study, Mandy et al. (2011) operationalized the definition of PDD NOS and compared the symptom profiles of children with autistic disorder, Asperger's disorder, and PDD NOS on independent measures of symptomatology. They found that the overwhelming majority (97%) of children with PDD NOS presented with a symptom profile characterized by significant impairment in social interaction and communication skills without repetitive stereotyped behavior. The remaining children presented with a symptom pattern of significant social impairment and repetitive stereotyped behavior without communication impairment. These results are inconsistent with the view of PDD NOS being a condition with marked heterogeneity. The children with

PDD NOS demonstrated significantly less routinized and repetitive behaviors, sensory difficulties, feeding, and visuospatial problems compared to the children with autistic disorder and Asperger's disorder. With PDD NOS now subsumed under the DSM-5 diagnostic category of autism spectrum disorder (ASD), it may be that individuals presenting with marked impairments in social interaction and communication, without repetitive stereotyped behavior, will not meet the DSM-5 diagnostic criteria for ASD.

High rates of comorbid mental health problems have been reported in atypical autism and PDD NOS. A Danish study compared a sample of 89 individuals diagnosed as children with atypical autism to a matched control sample from the general population (Mouridsen et al. 2008). Using the Danish Psychiatric Register, they demonstrated that over a 36-year follow-up period, elevated rates of co-occurring psychiatric diagnoses were found in those with atypical autism. The most prevalent of these was schizophrenia spectrum disorder. High levels of depression, anxiety, and disruptive behavior disorder have been reported in children with PDD NOS (de Bruin et al. 2007; Pearson et al. 2006), highlighting the importance of considering comorbid mental health problems when conducting diagnostic assessments for atypical autism.

It has been reported that while comorbid medical conditions in autism are associated with degree of intellectual disability, they may be more frequent in individuals with atypical autism, although results are mixed across studies (Gillberg and Coleman 1996; Juul-Dam et al. 2001; Rutter et al. 1994). A study by Hara (2007) found no differences between individuals with autism and atypical autism in terms of epilepsy. Biological research on atypical autism and PDD NOS, including neuroimaging and genetic studies, has overall found no evidence for differences between these conditions and autistic disorder (Towbin 2005).

Evaluation and Differential Diagnosis

The assessment process for atypical autism is the same as that recommended for autism and other pervasive developmental disorders. In making a

differential diagnosis, whether the criteria are met for a diagnosis of autism or Asperger's disorder needs to be considered, and degree of intellectual disability needs to be taken into account. Differentiating atypical autism from language disorder is also important. It has been demonstrated that children with PDD NOS can be differentiated from children with language disorders on the basis of more severe social impairment and a greater need for routines and order (Mayes et al. 1993). Research with children with a significant degree of disruptive behavior has also highlighted the need to consider a diagnosis of atypical autism. In a cohort of primary school-aged children, significant impairments in social and communication domains were identified in children with significant disruptive behavior, with 28% meeting criteria for a diagnosis of atypical autism (Donno et al. 2010).

Differentiating ADHD and atypical autism in young children can be problematic, with children often first diagnosed with ADHD (Jensen et al. 1997). In a retrospective study, parents of children with PDD NOS or ADHD reported on the symptoms of their children in their first 4 years (Roeyers et al. 1998). Early differences were infrequent, although children with ADHD showed more hyperactive behaviors during the 7–12-month period; this difference was not maintained as the PDD NOS children became active with age. As children aged, the difference became more apparent, with children with PDD NOS demonstrating more pronounced social difficulties, withdrawal, anxiety, stereotyped motor behaviors, unusual behaviors, and better scores on cognitive assessments compared to children with ADHD (Jensen et al. 1997; Luteijn et al. 2000; Roeyers et al. 1998; Scheirs and Timmers 2009).

Treatment

As for autism, treatment for individuals with atypical autism needs to include a range of services and approaches. Behavioral, educational, and developmental approaches to the treatment of communication deficits, social difficulties, and behavior problems have been demonstrated to

result in improvements for individuals with autism and are likely to be helpful for individuals with atypical autism. Although there are no drugs that specifically treat autism, medication and medication in combination with parent training approaches have been shown to reduce severe behavior problems such as aggression, self-injurious behavior, severe tantrums, and irritability (King 2000; Research Units on Pediatric Psychopharmacology (RUPP) Autism Network 2005a, b, 2009).

Early intervention has been highlighted as a specific area of importance in the treatment of children with autism. Treatment gains have been demonstrated in adaptive functioning, developmental skills, symptom severity, and behavior problems (Dawson et al. 1998; Howlin et al. 2009; Rogers and Vismara 2008). Training parents to implement early intervention programs has also demonstrated gains in communicative behavior, knowledge of autism, parent communication style, parent-child interaction, child behavior problems, and a reduction in parent stress and mental health problems (McConachie and Diggle 2005; Tonge et al. 2006; Whittingham et al. 2009). Improvements can also be made in teaching joint attention, symbolic play, and imitation skills to very young children (Drew et al. 2002; Kasari et al. 2001, 2006). Although important gains have been made in the development of evidence-based early interventions, less is known about the role played by mediating or moderating variables in treatment outcomes. Importantly, the impact that early childhood intervention may or may not have on adult outcomes remains unknown.

Research on treatment approaches specifically for individuals with atypical autism is lacking; it is assumed that treatment needs and approaches are similar to those for individuals with autism.

Whether existing evidence-based treatments produce greater effects in individuals with atypical autism remains an area for further research. As is the case for autism, it has been concluded that no single treatment approach or method has been shown to be effective for PDD NOS (Towbin 2005), with treatment approaches needing to take into account the specific strengths, impairments, and needs of each individual.

References

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd. rev. ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders – Text revision* (4th ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5)* (5th ed.). Washington, DC: American Psychiatric Association.
- Baghdadli, A., Picot, M.-C., Michelon, C., Bodet, J., Pernon, E., Burstejn, C., et al. (2007). What happens to children with PDD when they grow up? Prospective follow-up of 219 children from preschool age to mid-childhood. *Acta Psychiatrica Scandinavica*, 115, 403–412.
- Baird, G., Charman, T., Baron-Cohen, S., Cox, A., Swettenham, J., Wheelwright, S., et al. (2000). A screening instrument for autism at 18 months of age: A 6-year follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(6), 694–702.
- Barton, M., Robins, D., Jashar, D., Brennan, L., & Fein, D. (2013). Sensitivity and specificity of proposed DSM-5 criteria for autism spectrum disorder in toddlers. *Journal of Autism and Developmental Disorders*, 45(5), 1184–1195.
- Bishop, D. V. M. (1998). Development of the Children's communication checklist (CCC): A method for assessing qualitative aspects of communicative impairment in children. *Journal of Child Psychology and Psychiatry*, 39(6), 879–891.
- Chakrabarti, S., & Fombonne, E. (2005). Pervasive developmental disorders in preschool children: Confirmation of high prevalence. *The American Journal of Psychiatry*, 162(6), 1133–1141.
- Chawarska, K., Klin, A., Paul, R., & Volkmar, F. (2007). Autism spectrum disorder in the second year: Stability and change in syndrome expression. *Journal of Child Psychology and Psychiatry*, 48(2), 128–138.
- Dawson, J. E., Matson, J. L., & Cherry, K. E. (1998). An analysis of maladaptive behaviors in persons with autism, PDD-NOS, and mental retardation. *Research in Developmental Disabilities*, 19(5), 439–448.
- de Bruin, E. I., Verheij, F., & Ferdinand, R. F. (2006). WISC-R subtest but no overall VIQ-PIQ difference in Dutch children with PDD-NOS. *Journal of Abnormal Child Psychology*, 34(2), 263–271.
- de Bruin, E. I., Ferdinand, R. F., Meester, S., de Nijs, P. F. A., & Verheij, F. (2007). High rates of psychiatric co-morbidity in PDD-NOS. *Journal of Autism and Developmental Disorders*, 37(5), 877–886.
- Donno, R., Parker, G., Gilmour, J., & Skuse, D. H. (2010). Social communication deficits in disruptive primary-

- school children. *British Journal of Psychiatry*, 196, 282–289.
- Drew, A., Baird, G., Baron-Cohen, S., Cox, A., Slonims, V., Wheelwright, S., et al. (2002). A pilot randomised control trial of a parent training intervention for pre-school children with autism. Preliminary findings and methodological challenges. *European Child & Adolescent Psychiatry*, 11(6), 266–272.
- Ellefsen, A., Kampmann, H., Billstedt, E., Gillberg, I. C., & Gillberg, C. (2007). Autism in the Faroe Islands: An epidemiological study. *Journal of Autism and Developmental Disorders*, 37(3), 437–444.
- Fernell, E., & Gillberg, C. (2010). Autism spectrum disorder diagnoses in Stockholm preschoolers. *Research in Developmental Disabilities*, 31(3), 680–685.
- Fodstad, J. C., Matson, J. L., Hess, J., & Neal, D. (2009). Social and communication behaviours in infants and toddlers with autism and pervasive developmental disorder-not otherwise specified. *Developmental Neurorehabilitation*, 12(3), 152–157.
- Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. *Pediatric Research*, 65(6), 591–598.
- Gillberg, C., & Coleman, M. (1996). Autism and medical disorders: A review of the literature. *Developmental Medicine & Child Neurology*, 38(3), 191–202.
- Gillberg, C., & Steffenburg, S. (1987). Outcome and prognostic factors in infantile autism and similar conditions: A population-based study of 46 cases followed through puberty. *Journal of Autism and Developmental Disorders*, 17(2), 273–287.
- Hara, H. (2007). Autism and epilepsy: A retrospective follow-up study. *Brain & Development*, 29(8), 486–490.
- Howlin, P., Magiati, I., & Charman, T. (2009). Systematic review of early intensive behavioral interventions for children with autism. *American Journal on Intellectual and Developmental Disabilities*, 114(1), 23–41.
- Huerta, M., Bishop, S. L., Duncan, A., Hus, V., & Lord, C. (2012). Application of DSM-5 criteria for autism Spectrum disorder to three samples of children with DSM-IV diagnoses of pervasive developmental disorders. *American Journal of Psychiatry*, 169(10), 1056–1064.
- Jensen, V. K., Larrieu, J. A., & Mack, K. K. (1997). Differential diagnosis between attention-deficit/hyperactivity disorder and pervasive developmental disorder – Not otherwise specified. *Clinical Pediatrics*, 36(10), 555–561.
- Juul-Dam, N., Townsend, J., & Courchesne, E. (2001). Prenatal, perinatal, and neonatal factors in autism, pervasive developmental disorder-not otherwise specified, and the general population. *Pediatrics*, 107(4), E63.
- Kanai, C., Koyama, T., Kato, S., Miyamoto, Y., Osada, H., & Kurita, H. (2004). Comparison of high-functioning atypical autism and childhood autism by Childhood Autism Rating Scale-Tokyo version. *Psychiatry & Clinical Neurosciences*, 58(2), 217–221.
- Kasari, C., Freeman, S. F. N., & Paparella, T. (2001). Early intervention in autism: Joint attention and symbolic play. *International Review of Research in Mental Retardation*, 23(23), 207–237.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry*, 47(6), 611–620.
- Kim, Y. S., Leventhal, B. L., Koh, Y.-J., Fombonne, E., Laska, E., Lim, E.-C., Cheon, K.-A., Kim, Y.-K., Lee, H. K., Song, D.-H., & Grinker, R. R. (2011). Prevalence of autism spectrum disorders in a total population sample. *American Journal of Psychiatry*, 168, 904–912.
- Kim, Y. S., Fombonne, E., Koh, Y.-J., Kim, S.-J., Cheon, K.-A., Leventhal, B. A. (2014). Comparison of DSM-IV pervasive developmental disorder and DSM-5 autism spectrum disorder prevalence in an epidemiologic sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(5), 500–508.
- King, B. H. (2000). Pharmacological treatment of mood disturbances, aggression, and self-injury in persons with pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 30(5), 439–445.
- Kleinman, J. M., Robins, D. L., Ventola, P. E., Pandey, J., Boorstein, H. C., Esser, E. L., et al. (2008). The modified checklist for autism in toddlers: A follow-up study investigating the early detection of autism spectrum disorders. *Journal of Autism & Developmental Disorders*, 38(5), 827–839.
- Kobayashi, R., Murata, T., & Yoshinaga, K. (1992). A follow-up study of 201 children with autism in Kyushu and Yamaguchi areas, Japan. *Journal of Autism and Developmental Disorders*, 22(3), 395–411.
- Kurita, H. (1997). A comparative study of Asperger syndrome with high-functioning atypical autism. *Psychiatry & Clinical Neurosciences*, 51(2), 67–70.
- Kurita, H., Miyake, Y., & Katsuno, K. (1989). Reliability and validity of the childhood autism rating scale-Tokyo version (CARS-TV). *Journal of Autism and Developmental Disorders*, 19(3), 389–396.
- Lingam, R., Simmons, A., Andrews, N., Miller, E., Stowe, J., & Taylor, B. (2003). Prevalence of autism and parentally reported triggers in a north east London population. *Archives of Disease in Childhood*, 88(8), 666–670.
- Luteijn, E. F., Serra, M., Jackson, S., Steenhuis, M. P., Althaus, M., Volkmar, F., et al. (2000). How unspecified are disorders of children with a pervasive developmental disorder not otherwise specified? A study of social problems in children with PDD-NOS and ADHD. *European Child & Adolescent Psychiatry*, 9(3), 168–179.
- Mahoney, W. J., Szatmari, P., MacLean, J. E., Bryson, S. E., Bartolucci, G., Walter, S. D., et al. (1998). Reliability and accuracy of differentiating pervasive developmental disorder subtypes. *Journal of the American Academy of Child & Adolescent Psychiatry*, 37(3), 278–285.
- Mandy, W., Charman, T., Gilmour, J., & Skuse, D. (2011). Toward specifying pervasive developmental disorder – not otherwise specified. *Autism Research*, 4, 1–11.

- Mayes, L., Volkmar, F., Hooks, M., & Cicchetti, D. (1993). Differentiating pervasive developmental disorder not otherwise specified from autism and language disorders. *Journal of Autism and Developmental Disorders*, 23(1), 79–90.
- Mayes, S. D., Black, A., & Tierney, C. D. (2013). DSM-5 under-identifies PDD-NOS: Diagnostic agreement between the DSM-5, DSM-IV, and checklist for autism Spectrum disorder. *Research in Autism Spectrum Disorders*, 7(2), 298–306.
- McConachie, H., & Diggle, T. (2005). Parent implemented early intervention for young children with autism spectrum disorder: A systematic review. *Journal of Evaluation in Clinical Practice*, 13, 129–129.
- McPartland, J. C., Reichow, B., & Volkmar, F. R. (2012). Sensitivity and specificity of proposed DSM-5 diagnostic criteria for autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(4), 368–383.
- Moulton, E., Barton, M., Robins, D. L., Abrams, D. N., & Fein, D. (2016). Early characteristics of children with ASD who demonstrate optimal progress between age two and four. *Journal of Autism and Developmental Disorders*, 46(6), 2160–2173.
- Mouridsen, S. E., Rich, B., & Isager, T. (2008). Psychiatric disorders in adults diagnosed as children with atypical autism. A case control study. *Journal of Neural Transmission*, 115(1), 135–138.
- Mundy, P., Sigman, M., & Kasari, C. (1990). A longitudinal study of joint attention and language development in autistic children. *Journal of Autism and Developmental Disorders*, 20(1), 115–128.
- Nordin, V., & Gillberg, C. (1998). The long-term course of autistic disorders: Update on follow-up studies. *Acta Psychiatrica Scandinavica*, 97(2), 99–108.
- Oslejskova, H., Kontrova, I., Foralova, R., Dusek, L., & Nemethova, D. (2007). The course of diagnosis in autistic patients: The delay between recognition of the first symptoms by parents and correct diagnosis. *Neuroendocrinology Letters*, 28(6), 895–900.
- Ozonoff, S. (2012). Editorial perspective: Autism spectrum disorders in DSM-5 – An historical perspective and the need for change. *Journal of Child Psychology and Psychiatry*, 53(10), 1092–1094.
- Pearson, D. A., Loveland, K. A., Lachar, D., Lane, D. M., Reddoch, S. L., Mansour, R., et al. (2006). A comparison of behavioral and emotional functioning in children and adolescents with autistic disorder and PDD-NOS. *Child Neuropsychology*, 12(4–5), 321–333.
- Research Units on Pediatric Psychopharmacology (RUPP) Autism Network. (2005a). Randomized, controlled, crossover trial of methylphenidate in pervasive developmental disorders with hyperactivity. *Archives of General Psychiatry*, 62, 1266–1274.
- Research Units on Pediatric Psychopharmacology (RUPP) Autism Network. (2005b). Risperidone treatment of autistic disorder: Longer-term benefits and blinded discontinuation after 6 months. *The American Journal of Psychiatry*, 162(7), 1361–1369.
- Research Units on Pediatric Psychopharmacology Autism Network. (2009). Medication and parent training in children with pervasive developmental disorders and serious behavior problems: Results from a randomized clinical trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, 48(12), 1143–1154.
- Roeyers, H., Keymeulen, H., & Buysse, A. (1998). Differentiating attention-deficit/hyperactivity disorder from pervasive developmental disorder not otherwise specified. *Journal of Learning Disabilities*, 31(6), 565–571.
- Rogers, S. J., & Vismara, L. A. (2008). Evidence-based comprehensive treatments for early autism. *Journal of Clinical Child & Adolescent Psychology*, 37(1), 8–38.
- Rondeau, E., Klein, L. S., Masse, A., Bodeau, N., Cohen, D., & Guile, J. M. (2010). Is pervasive developmental disorder not otherwise specified less stable than autistic disorder? A meta-analysis. *Journal of Autism & Developmental Disorders*, 41(9), 1267–1276.
- Rutter, M., Bailey, A., Bolton, P., & Le Couteur, A. (1994). Autism and known medical conditions: Myth and substance. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, 35(2), 311–322.
- Scheirs, J. G. M., & Timmers, E. A. (2009). Differentiating among children with PDD-NOS, ADHD, and those with a combined diagnosis on the basis of WISC-III profiles. *Journal of Autism and Developmental Disorders*, 39(4), 549–556.
- Sigman, M., & Ruskin, E. (1999). Continuity and change in the social competence of children with autism, down syndrome, and developmental delays. *Monographs of the Society for Research in Child Development*, 64(1), v–114.
- Skuse, D. H. (2012). DSM-5's conceptualization of autistic disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51, 344–346.
- Stone, W. L., Lee, E. B., Ashford, L., Brissie, J., Hepburn, S. L., Coonrod, E. E., et al. (1999). Can autism be diagnosed accurately in children under 3 years? *Journal of Child Psychology and Psychiatry*, 40(2), 219–226.
- Sutera, S., Pandey, J., Esser, E. L., Rosenthal, M. A., Wilson, L. B., Barton, M., et al. (2007). Predictors of optimal outcome in toddlers diagnosed with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(1), 98–107.
- Tonge, B., Brereton, A. V., Kiamall, M., Mackinnon, A., King, N. M., & Rinehart, N. (2006). Effects on parental mental health of an education and skills training program for parents of young children with autism: A randomized controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45(5), 561–569.
- Towbin, K. E. (2005). Pervasive developmental disorder not otherwise specified. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 165–200). Hoboken: Wiley.
- Turner, L. M., Stone, W. L., Pozdol, S. L., & Coonrod, E. E. (2006). Follow-up of children with autism spectrum disorders from age 2 to age 9. *Autism*, 10(3), 243–265.
- van Daalen, E., Kemner, C., Dietz, C., Swinkels, S. H. N., Buitelaar, J. K., & van Engeland, H. (2009). Inter-rater

- reliability and stability of diagnoses of autism spectrum disorder in children identified through screening at a very young age. *European Child & Adolescent Psychiatry*, 18(11), 663–674.
- Volkmar, F. R. (1998). Categorical approaches to the diagnosis of autism: An overview of DSM-IV and ICD-10. *Autism: The International Journal of Research and Practice*, 2(1), 45–60.
- Volkmar, F. R., Shaffer, D., & First, M. (2000). PDDNOS in DSM-IV. *Journal of Autism and Developmental Disorders*, 30(1), 74–75.
- Walker, D. R., Thompson, A., Zwaigenbaum, L., Goldberg, J., Bryson, S. E., Mahoney, W. J., et al. (2004). Specifying PDD-NOS: A comparison of PDD-NOS, Asperger syndrome, and autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 43(2), 172–180.
- Whittingham, K., Sofronoff, K., Sheffield, J., & Sanders, M. R. (2009). Stepping stones triple P: An RCT of a parenting program with parents of a child diagnosed with an autism spectrum disorder. *Journal of Abnormal Child Psychology*, 37(37), 469–480.
- Williams, E., Thomas, K., Sidebotham, H., & Emond, A. (2008). Prevalence and characteristics of autistic spectrum disorders in the ALSPAC cohort. *Developmental Medicine & Child Neurology*, 50(9), 672–677.
- World Health Organisation. (1978). *International classification of diseases: Mental disorders: Glossary and guide to their classification* (9th ed.). Geneva: World Health Organisation.
- World Health Organisation. (1992). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research*. Geneva: World Health Organisation.
- World Health Organisation, GCP Network (2017). ICD-11 draft guidelines. Available at <https://gcp.network/en/private/icd-11-guidelines/grouping>. Accessed 22 Dec 2017.

Audiovisual Media Content Preferences of Children with Autism Spectrum Disorders

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Definition

Children with autism spectrum disorder (ASD) spend more time with screen media than any other leisure activity (Shane and Albert 2008). Evidence indicates that children with ASD spend most of their screen time with nonsocial media (i.e., television, video games) and less time with

social media. Compared with other disability groups, among ASD youth, rates of nonsocial media use are higher, and that of social media use are lower (Mazurek 2013b). Similarly, children with ASD report more time with television and video games and less time with social media as compared to neurotypical siblings (Mazurek 2013a). Given that children with ASD report difficulty in developing and maintaining friendships compared to typically developing children (i.e., Rowley et al. 2012), the finding that ASD youth spend more time with nonsocial media is not surprising.

Although existing research demonstrates that children with ASD engage in selective exposure to screen media, less attention has been paid to content preferences among this population. There are only a handful of studies that offer some insight into media content preferences of children with ASD. Regarding television, some studies report that children with ASD tend to prefer animated content (e.g., Martins et al. 2019; Shane and Albert 2008) and that this content is typically created for younger audiences (Martins et al. 2019). Martins et al. (2019) argued that children with ASD select programs with content features made to appeal to developmentally similar children; hence programs for the preschool audience are commonly reported as favorites. These programs are slower-paced and more attuned to specific, individual sensory preferences which may aid in comprehension. Considering that parents identify comprehensibility of content as a key factor in program selection by their children (Martins et al. 2019), then we would expect that these programs are what children with ASD like the most.

Comprehensibility is also a key factor in video game content preferences. In their review, Stiller and Mößle (2018) reported that children with ASD prefer role-playing and simulation games. Martins et al. (2019) argued that games like *Minecraft* are popular among this population because children understand the basic functionality for paying and pausing and have full control over the content they view.

As mentioned above, research demonstrates that children with ASD spend little to no time on social media platforms (Martins et al. 2019; Mazurek 2013a, b). There are at least three

reasons why children with ASD spend less time with nonsocial media. First, children with ASD are socially isolated (Rowley et al. 2012) and may not have a network of friends to connect with online. Second, some apps like *Snapchat* or *Facebook* are not developmentally appropriate, and ASD youth may not understand how such apps work. Finally, parental concerns over the safety of social networks may prevent ASD youth from experimenting with the technology. Parents are worried that their ASD child is incapable of recognizing deceit and therefore might be targets of child predators. Other parents were worried that their child could be made fun or bullied for the content they post (see Martins et al. 2019).

Future research should continue to examine content preferences and how such preferences can be used to teach children both academic and socioemotional skills. For example, future work should test whether education skill could be taught using repeated exposure to preferred components of media (i.e., favorite media characters). Such work is particularly important given that parents report that “autism apps” marketed to the ASD community are either unaffordable or not proven to work (see Martins et al. 2019). Future research should also examine whether restricting access to online communities or social networking apps does more harm than good as ASD children transition into adolescence.

See Also

► [Visual Supports](#)

References and Reading

- Martins, N., King, A. J., & Beights, R. (2019). Audiovisual media content preferences of children with autism spectrum disorders: Insights from parental interviews. *Journal of Autism and Developmental Disorders*, 1–9. <https://doi.org/10.1007/s10803-019-03987-1>.
- Mazurek, M. O., & Engelhardt, C. R. (2013a). Video game use in boys with autism spectrum disorder, ADHD, or typical development. *Pediatrics*, 132, 260–266. <https://doi.org/10.1542/peds.2012-3956>.
- Mazurek, M. O., & Wenstrup, C. (2013b). Television, video game and social media use among children with ASD and typically developing siblings. *Journal of Autism and*

Developmental Disorders, 43, 1258–1271. <https://doi.org/10.1007/s10803-012-1659-9>.

- Rowley, E., Chandler, S., Baird, G., Simonoff, E., Pickles, A., Loucas, T., et al. (2012). The experiences of friendship, victimization and bullying in children with an autism spectrum disorder: Associations with child characteristics and school placement. *Research in Autism Spectrum Disorders*, 6, 1126–1134.
- Shane, H. C., & Albert, P. D. (2008). Electronic screen media for persons with autism spectrum disorder: Results of a survey. *Journal of Autism and Developmental Disorders*, 38, 1499–1508. <https://doi.org/10.1007/s10803-007-0527-5>.
- Stiller, A., & Mößle, T. (2018). Media use among children and adolescents with autism spectrum disorder: A systematic review. *Review Journal of Autism and Developmental Disorders*, 5, 227–246. <https://doi.org/10.1007/s40489-018-0135-7>.

Audition

► [Hearing](#)

Auditory Acuity

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Synonyms

[Hearing sensitivity](#); [Hearing threshold](#)

Definition

Auditory acuity describes how sensitive the auditory system is to sound. Auditory acuity is assessed by determining the intensity at which a tone is just audible. Frequencies important for speech perception are typically tested (octave frequencies from 250 to 8,000 Hz). Normal hearing sensitivity is defined as hearing thresholds from 250 to 8,000 Hz between –10 and 15 dB HL. Hearing sensitivity between 16 and 25 dB HL is considered *minimal* or *borderline*; between 26 and 40 dB HL is considered *mild* hearing loss;

between 41 and 55 dB HL is considered *moderate* hearing loss; between 56 and 70 dB HL is considered *moderately severe* hearing loss; between 71 and 90 dB HL is considered *severe* hearing loss; and greater than 91 dB HL is considered *profound* hearing loss. Hearing sensitivity would be evaluated in an individual with autism spectrum disorders if questions regarding hearing abilities existed, but more systematic research needs to be completed regarding auditory acuity in the population of individuals with autism.

See Also

- [Hearing Sensitivity](#)
- [Hearing Threshold](#)

References and Reading

- Hall, J. (1992). *Handbook of auditory evoked responses*. Needham Heights: Allyn & Bacon.
- Justice, L. (2006). *Communication sciences and disorders: An introduction*. Columbus: Pearson.
- Rosenhall, U., Nordin, V., Sandstrom, M., Ahlsen, G., & Gillberg, C. (1999). Autism and hearing loss. *Journal of Autism and Developmental Disorders*, 29(5), 349–357.

Auditory Brain Area

- [Auditory Cortex](#)

Auditory Brainstem Response (ABR)

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Synonyms

[Brainstem auditory evoked response \(BAER\)](#)

Definition

Auditory brainstem response (ABR), sometimes called brainstem auditory evoked response (BAER), is an electrophysiologic test that assesses the auditory system through the low brainstem. This test can assess hearing sensitivity in individuals who cannot respond to traditional testing; thus, it is often used in newborn hearing screenings and on populations that are nonverbal. The ABR is completed by placing electrodes on the individual's head and ears and placing earphones in their ears. Responses are elicited using click and tonal stimuli which are delivered through the earphones. Five waveforms are typically present in the ABR (waves I, II, III, IV, and V); however, wave V is the waveform used for threshold testing. Individuals with autism spectrum disorders might not be able to consistently respond to traditional tests of hearing sensitivity; therefore, ABR may be useful in establishing hearing sensitivity for these individuals.

See Also

- [Auditory Acuity](#)
- [Brainstem Audiometry](#)
- [Hearing](#)

References and Reading

- Hall, J. (1992). *Handbook of auditory evoked responses*. Needham Heights: Allyn & Bacon.
- Rosenblum, S. M., Arick, J. R., Krug, D. A., Stubbs, E. G., Young, N. B., & Pelson, R. O. (1980). Auditory brainstem evoked responses in autistic children. *Journal of Autism and Developmental Disorders*, 10, 215–225.
- Rosenhall, U., Nordin, V., Sandstrom, M., Ahlsen, G., & Gillberg, C. (1999). Autism and hearing loss. *Journal of Autism and Developmental Disorders*, 29(5), 349–357.
- Skoff, B. F., Mirsky, A. F., & Turner, D. (1980). Prolonged brainstem transmission time in autism. *Psychiatry Research*, 2, 157–166.
- Skoff, B. F., Fein, D., McNally, B., Lucci, D., Humes-Bartlo, M., & Waterhouse, L. (1986). Brainstem auditory evoked potentials in autism. *Psychophysiology*, 23, 462.

Auditory Brainstem Response, ABR

- [Brainstem Auditory Evoked Potentials](#)

Auditory Cortex

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Synonyms

[Auditory brain area](#)

Definition

The human auditory cortex occupies a large portion of the superior temporal gyrus located along the sylvian fissure dorsally and the superior temporal sulcus ventrally (Brodmann area 41, 42, and 22). The dorsal surface of the superior temporal gyrus is located within the sylvian fissure and is divided into Heschl's gyrus, the planum temporale, and the planum polare. Studies have suggested that the primary auditory cortex in humans is mainly confined to the anterior-medial wall of Heschl's gyrus. This brain region is vital in decoding and processing spoken language and sounds. The planum temporale, also vital in auditory processing, is located posterior to Heschl's gyrus and lies on the superior surface of the posterior superior temporal sulcus. While high-frequency sounds activate a small lateral region anterior to the intersection of Heschl's gyrus and the superior temporal gyrus and a more extensive medial region posterior to the tip of Heschl's gyrus, low-frequency sounds activate lateral regions centered on mid-Heschl's gyrus and extending posteriorly along the superior temporal gyrus.

Neuroimaging research has identified anatomical and functional abnormalities in the planum temporale in individuals with autism spectrum disorder. While anatomical abnormalities include abnormal asymmetry, altered minicolumn organization, and altered cell type and count, the functional abnormalities include abnormal feature extraction and sensitivity to sounds.

See Also

- ▶ [Auditory Acuity](#)
- ▶ [Auditory Processing](#)
- ▶ [Cortical Language Areas](#)
- ▶ [Primary Sensory Areas](#)
- ▶ [Wernicke's Aphasia](#)

References and Reading

- Binder, J. R., Rao, S. M., Hammeke, T. A., Yetkin, F. Z., Jesmanowicz, A., Bandettini, P. A., et al. (1994). Functional magnetic resonance imaging of human auditory cortex. *Annals of Neurology*, 35, 662–672.
- Boddaert, N., Chabane, N., Belin, P., Bourgeois, M., Royer, V., Barthelemy, C., et al. (2004). Perception of complex sounds in autism: Abnormal auditory cortical processing in children. *American Journal of Psychiatry*, 161, 2117–2120.
- Celesia, G. G. (1976). Organization of auditory cortical areas in man. *Brain*, 99, 403–414.
- Palmen, S., van Engeland, H., Hof, P., & Schmitz, C. (2004). Neuropathological finding in autism. *Brain*, 127, 2572–2583.
- Zatorre, R. J., Belin, P., & Penhune, V. (2002). Structure and function of auditory cortex: Music and speech. *Trends in Cognitive Sciences*, 6, 37–46.

Auditory Discrimination

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Definition

While enhanced discrimination and memory for musical pitch have been widely described in the literature on musical savants with autism, it is only in more recent times that such abilities have been observed in autistic individuals without savant skills (see Heaton 2003). Bonnel et al. (2010) studied auditory perception in individuals with high-functioning autism and Asperger's syndrome and showed that enhanced pitch discrimination was more prevalent in those with late

speech onset and was not associated with atypical discrimination of stimuli that were spectrally and/or temporally complex. Research identifying enhanced discrimination of pitch change in linguistic stimuli (Jarvinen-Pasley and Heaton 2007) has shown that atypical pitch processing is not limited to music but generalizes across auditory domains. This suggests that difficulties in understanding pitch-mediated linguistic cues or prosody, demonstrated in a number of studies (for review McCann and Peppe 2003), are not perceptual in origin but result from abnormalities in higher-order cognitive operations. Building on the enhanced perceptual functioning model, the neural complexity hypothesis (see Samson et al. 2010) is able to account for enhanced pitch discrimination as well as abnormalities in processing acoustically complex stimuli. According to this model, autism is characterized by a bias toward the perceptual features of auditory information. At the behavioral level, this can be associated with enhanced processing of low-level stimuli and atypical processing of higher-order information, such as greater focus toward the perceptual aspects of speech stimuli.

See Also

- [Autistic Savants](#)
- [Enhanced Perceptual Functioning](#)

References and Reading

- Bonnel, A., McAdams, S., Smith, B., Berthiaume, C., Bertone, A., Ciocca, V., et al. (2010). Enhanced pure-tone pitch discrimination among persons with autism but not Asperger syndrome. *Neuropsychologia*, 48(9), 2465–2475.
- Heaton, P. (2003). Pitch memory, labeling and disembedding in autism. *Journal of Child Psychology and Psychiatry*, 44(4), 543–551.
- Jarvinen-Pasley, A., & Heaton, P. (2007). Evidence for reduced domain-specificity in auditory processing in autism. *Developmental Science*, 10(6), 786–793.
- McCann, J., & Peppe, S. (2003). Prosody in autism spectrum disorders: A critical review. *International Journal of Language & Communication Disorders*, 38(4), 325–350.

- Samson, F., Hyde, K. L., Bertone, A., Soulieres, I., Mendrek, A., Ahad, P., et al. (2010). Atypical processing of auditory temporal complexity in autistics. *Neuropsychologia*, 49, 546–555.

Auditory Evoked Potential (AEP)

- [Auditory Potentials](#)

Auditory Integration Therapy

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Definition

Auditory integration training (AIT) is an intervention technique which is currently considered experimental. It was created to attempt to improve the way individuals with autism spectrum disorders (ASD) recognize and respond to sound and to reduce other behaviors associated with ASD. AIT has also been referred to as auditory enhancement training (AET) and audio-psycho-phonology (APP).

Historical Background

Auditory integration training (AIT) was first written about in 1982 in a book by the otolaryngologist Guy Berard, which was translated in 1993 from French to the English title *Hearing Equals Behavior*. In his writing, Berard suggests that various disorders (“autism,” hyperactivity, depression, learning difficulties) are associated with atypical sensitivity to sound.

The AIT technique became widely popular after the 1991 publication of Annabel Stehli’s *The Sound of a Miracle: A Child’s Triumph over Autism*. In this book, Stehli described the full recovery of her

daughter, who was diagnosed with autism and schizophrenia, after 10 h of AIT at Berard's clinic. In 1994, the American Speech-Language-Hearing Association (ASHA) published a review of the existing data on AIT in response to such accounts linking AIT to increased eye contact, social awareness, verbalizations, auditory comprehension, and articulation and reduced tantrums and hyperacusis (i.e., oversensitivity to certain frequency ranges of sound) in children with autism spectrum disorders, learning difficulties, attention deficit disorder, and dyslexia. Currently, several professional organizations (including the American Speech-Language-Hearing Association, the American Academy of Audiology, the Educational Audiology Association, and the American Academy of Pediatrics) indicate that AIT should be considered an experimental rather than an evidence-based treatment due to the lack of scientific data supporting its benefits.

While in the United States the majority of AIT practitioners use the original Berard or a modified methodology, there are other methods of AIT in existence (including the Tomatis and Clark methods).

Rationale or Underlying Theory

Dr. Guy Berard, an ear, nose, and throat (ENT) physician, first introduced auditory integration training (AIT) suggesting that many learning and behavioral disorders, "including autism," are associated with hypersensitivity to sound at particular frequencies possibly resulting in disturbances in learning and discomfort. He suggested that although many children with autism spectrum disorders (ASD) can hear sound, the way in which they process sounds is different and can result in reduced emotional responsiveness and repetitive behaviors even if hypersensitivity to sound does not exist.

Goals and Objectives

In 1982, Dr. Berard suggested that auditory integration training (AIT) would involve a

"reeducation" of the hearing process for individuals with autism spectrum disorders (ASD) targeting the atypical sound perception theorized to be present in a variety of behavioral and learning disorders. Specifically, he suggests the training of the middle ear muscles, and the auditory nervous system is targeted through listening exercises.

Treatment Participants

Auditory integration training (AIT) has been promoted by Dr. Berard as a useful intervention for a variety of disorders (e.g., learning disabilities, behavior disorders, autism, pervasive developmental disorder, attention deficit disorder, attention deficit hyperactivity disorder, tinnitus, progressive deafness, hyperacusis, allergic disorders, depression, suicidal tendencies, poor organizational skills) and has also been recommended for reducing foreign accents and writer's block.

Treatment Procedures

Auditory integration training (AIT) begins with an audiogram (i.e., a graph showing the results of a pure-tone hearing test) to determine whether auditory "abnormalities" exist. The treatment involves ten consecutive days of therapy centered upon listening to music (that has been modified to dampen certain sound frequencies and intensities to correspond to those found abnormal on the audiogram) for 30 min twice a day. It is recommended that sessions occurring on the same day be separated by at least 3 h, while a 2-day interruption of therapy on weekends is allowed.

Audiograms are also used to determine if filter settings need to be adjusted mid-intervention and to monitor response to treatment post-intervention. Berard asserts that following AIT, audiograms show that auditory distortions are eliminated, as they become "flattened." He explains that the "peaks and valleys" in the original audiograms reflect areas of hyper- and hypo-sensitivity, but there is debate as to whether these patterns truly indicate auditory "abnormalities."

Following the recommended 20 auditory integration therapy (AIT) sessions in Dr. Berard's method, an audiogram is obtained and reviewed, while changes in behavior patterns are examined to measure outcome. In efficacy studies of AIT, outcome measures have included post-intervention assessments in the following areas: cognitive ability, core features of autism (i.e., social interaction, communication, and behavioral problems), hyperacusis, auditory processing, behavioral problems, attention and concentration, activity level, quality of life in school and at home, and adverse events.

The US Food and Drug Administration (FDA) banned the import of the Berard's original equipment (Audiokinetrone or Ears Education and Retraining System) used for AIT as a medical device based on finding that there was no sufficient evidence to support that it benefited individuals medically. The FDA regards the Audiokinetrone as an educational aid but not appropriate for the treatment or curing of any medical conditions, such as autism spectrum disorders. The Digital Auditory Aerobics (DAA) device was introduced as a result of this limited access to the Audiokinetrone in the United States. The 20 compact disks (CDs) (each containing 30 min of modulated music) available with this device are believed to match the output of the Audiokinetrone device. Other AIT programs are available (e.g., Samonas Sound Therapy, The Listening Program) which provide music on CDs and promise similar results to Berard's AIT programs.

Efficacy Information

The efficacy of auditory integration training (AIT) continues to be debated. A review of the available existing research indicates that three studies suggest improvements with AIT at 3 months post-intervention based on reported improved performance scores on the *Aberrant Behavior Checklist*. It should be noted that investigators in these studies were associated with organizations that promote or directly provide AIT. Similar results have not yet been replicated by any independent studies. The review highlights the fact that the studies examining

AIT were not randomized controlled trials (used to minimize bias), did not contain control or alternative treatment group, and involved single or very few participants or used surveys or animals.

The American Speech-Language-Hearing Association (ASHA) issued a report on AIT, in which it states that further research in AIT is discouraged given the lack of evidence that it is an effective treatment for individuals with autism spectrum disorder (ASD) but indicates that a "high level of evidence" of its efficacy should be provided if future AIT trials are conducted. ASHA also cautioned parents to take precautions to avoid hearing loss while also being aware of the costs involved in receiving AIT. In studies where children or adults with ASD (ages 3–39 years) were selected and randomly assigned to study treatment groups, though no adverse effects were reported, no noteworthy changes were found in the participants' ability to process sound, their quality of life, or their core and associated features of ASD following AIT. ASHA expressed concerns that clear criteria (based on evidence-based research) are not available, indicating which individuals will be most appropriate for AIT, and families could find both their financial resources and hope strained or depleted by investing in interventions that lack empirical support. In addition, the professional organization had reservations regarding the variability in AIT treatment protocols and the possible noise-induced hearing loss that might be associated with AIT devices, as sufficient data on the risk to participants regarding intensity of sound and length of presentation is not currently available for the devices. In more recent studies (2013–2016), electrophysiological changes and behavioral changes via caregiver report were observed in children with ASD following a series of AIT sessions. Authors of these studies suggested further research to explain the neural mechanisms of how AIT may affect such changes. Still, studies during this same time period suggested the lack of efficacy of AIT, some suggesting increased occurrence of stereotypy post-AIT.

Considering that ASD behaviors can often resemble auditory processing disorders (APD), ASHA has also ruled out the diagnosis of APD, for which AIT is often suggested, in children with

ASD unless reliable testing reveals deficits on multiple assessments. In the case that a child with ASD does meet this guideline, the benefit of receiving intervention involving listening tasks with limited social interaction can also be questioned.

Qualifications of Treatment Providers

The majority of auditory integration training (AIT) practitioners are speech-language pathologists or audiologists but have also included psychologists, physicians, social workers, and teachers. No training is required to operate the Digital Auditory Aerobics (DAA) device that is currently used within the United States to provide AIT based on Berard's method. Other AIT programs do provide trainings to practitioners (e.g., The Listening Program [2½ days], Samonas Sound Therapy [offers a credentialing process following pre-workshop training, initial and advanced workshop training, and a year of practice]). The American Speech-Language-Hearing Association, the American Academy of Audiology, the Educational Audiology Association, and the American Academy of Pediatrics nonetheless all state that AIT should be considered an experimental rather than an evidence-based treatment due to the limited amount of scientific research studies supporting its benefits.

See Also

- [Aberrant Behavior Checklist](#)
- [American Speech-Language-Hearing Association Functional Assessment of Communication Skills](#)
- [Auditory Processing Disorder](#)

References and Reading

- Al-Ayadhi, L. Y., Al-Drees, A. M., & Al-Arfaj, A. M. (2013). Effectiveness of auditory integration therapy in autism spectrum disorders—prospective study. *Autism Insights*, 5, 13.
- American Academy of Audiology. (1993). Position statement: Auditory integration training. *Audiology Today*, 5(4), 21.
- American Academy of Pediatrics. (1998). Auditory integration training and facilitated communication for autism. *Pediatrics*, 102(2), 431–433.
- American Speech-Language-Hearing Association Working Group on Auditory Integration Training. (2003). *Auditory integration training*. (Technical Report). Rockville: Author.. Retrieved from www.asha.org/docs/html/TR2004-00260.html
- Berard, G. (1993). *Hearing equals behaviour*. New Canaan: Keats Publishing. (Original work published 1982).
- Berard, G. (1995). Concerning length, frequency, number, and follow-up AIT sessions. *The Sound Connection Newsletter*, 2(3), 5–6. Available from The Society for Auditory Intervention Techniques.
- Bettison, S. (1996). The long-term effects of auditory training on children with autism. *Journal of Autism and Developmental Disorders*, 26(3), 361–373.
- Brockett, S. S., Lawton-Shirley, N. K., & Kimball, J. G. (2014). Berard auditory integration training: Behavior changes related to sensory modulation. *Autism Insights*, 6, 1.
- Committee on Children With Disabilities. (1998). Auditory integration training and facilitated communication for autism. *Pediatrics*, 102(2), 431–433.
- Edelson, S., Arin, D., Bauman, M., Lukas, S., Rudy, J., Sholar, M., et al. (1999). Auditory integration training: A double-blind study of behavioural and electrophysiological effects in people with autism. *Focus on Autism and Other Developmental Disabilities*, 14(2), 73–81.
- Educational Audiology Association. (1997). Auditory integration training: Educational Audiology Association position statement. *Educational Audiology Newsletter*, 14(3), 16.
- Feigin, J. A., Kapun, J. G., Stelmachowicz, P. G., & Gorga, M. P. (1989). Probe-tube microphone measures of ear canal sound pressure levels in infants and children. *Ear and Hearing*, 10(4), 254–258.
- Gillberg, C., & Coleman, M. (2000). *The biology of autistic syndromes* (3rd ed.). London: MacKeith Press.
- Gilmore, T., Madaule, P., & Thompson, B. (1989). *About the Tomatis method*. Toronto: Listening Center Press.
- Gringras, P. (2000). Practical paediatric psychopharmacological prescribing in autism: The potential and the pitfalls. *Autism*, 4(3), 229–247.
- LaFrance, D. L., Miguel, C. F., Donahue, J. N., & Fechter, T. R. (2015). A case study on the use of auditory integration training as a treatment for stereotypy. *Behavioral Interventions*, 30(3), 286–293.
- Mudford, O. C., Cross, B. A., Breen, S., Cullen, C., Reeves, D., Gould, J., & Douglas, J. (2000). Auditory integration training for children with autism: no behavioral benefits detected. *American Journal on Mental Retardation*, 105(2), 118–129.
- Mudford, O. C., & Cullen, C. (2005). Auditory integration training: A critical review. In J. W. Jacobson, R. M. Foxx, & J. A. Mulick (Eds.), *Controversial therapies for developmental disabilities: Fad, fashion, and science in professional practice* (pp. 351–362). Mahwah: Lawrence Erlbaum Associates.

- Rimland, B., & Edelson, S. M. (1994). The effects of auditory integration training on autism. *American Journal of Speech-Language Pathology*, 3(2), 16–24.
- Rimland, B., & Edelson, S. (1995). Brief report: A pilot study of auditory integration training in autism. *Journal of Autism and Developmental Disorders*, 25(1), 61–70.
- Sinha, Y., Silove, N., Wheeler, D. M., & Williams, K. J. (2009). *Auditory integration training and other sound therapies for autism spectrum disorders (Review)*. Hoboken: Wiley.
- Sokhadze, E. M., Casanova, M. F., Tasman, A., & Brockett, S. (2016). Electrophysiological and behavioral outcomes of berard auditory integration training (AIT) in children with autism spectrum disorder. *Applied psychophysiology and biofeedback*, 41(4), 405–420.
- Stehli, A. (1991). *The sound of a miracle. A child's triumph over autism*. New York: Doubleday.
- Tharpe, A. M. (1998). Treatment fads versus evidence-based practice. In F. H. Bess (Ed.), *Children with hearing impairment: Contemporary trends* (pp. 179–188). Nashville: Vanderbilt Bill Wilkerson Center Press.
- Tochel, C. (2003). *Sensory or auditory integration therapy for children with autistic spectrum disorders*. London: Bazian Ltd (Eds.), Wessex Institute for Health Research and Development, University of Southampton.
- Veale, T. (1993). *Effectiveness of AIT Using the BCG Device (Clark Method): A Controlled Study. Paper Presented at the World of Options International Autism Conference*. Toronto.
- Zollweg, W., Palm, D., & Vance, V. (1997). The efficacy of auditory integration training: A double blind study. *American Journal of Audiology*, 6(3), 39–47.

Auditory Perceptual Disorder

► Central Auditory Processing Disorder

Auditory Potentials

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Synonyms

[Auditory evoked potential \(AEP\)](#)

Definition

An auditory potential is an electroencephalographic (EEG) response, less than a millivolt, time-locked to an auditory sound such as a click, tone, or speech sound. It is recorded from scalp electrodes and consists of averaged responses to a series of sounds. Averaging removes background EEG activity, usually considered to be unrelated to the auditory potential.

A brief sound such as a click triggers at least 15 waveform peaks that unfold over the first second (Picton et al. 1974). These alternating positive and negative peaks reflect the flow of auditory information from the brainstem to the cortex. The short-latency peaks appearing during the first tenth of a second (10 ms) originate from the primary auditory pathway of the brainstem.

Central Auditory Processing Disorders (CAPDs) were described in early 1940s and have recently become of interest to ASD researchers (see Ocak et al. 2018). Auditory middle latency responses are promising auditory tests that allow the identification of functional deficits of the central auditory pathways, and the cerebral hemispheres in school children with reading and writing learning disorders. The recording of these potentials ensure visualization of the electrical activity of the primary auditory cortex and the auditory thalamus-cortical pathways, from the observation of a sequence of waves, negative (N) and positive (P). Na, Pa, Nb, Pb occur in 10–80 ms intervals after stimuli (McPherson et al. 2008).

The later auditory potentials, a subset of event-related potentials (ERPs), represent the sum of neural activity originating from spatially distinct sources. They are usually studied with multiple scalp electrodes that enable determination of waveform scalp topography. Mid-latency auditory peaks, which appear during the 10–50-ms interval, have few well-established clinical findings. Attention effects are seen under some conditions during the later part of this interval.

Long-latency peaks appearing between 50 and 1,000 ms have received the most study. The specific timing of these peaks depends on both the auditory stimulus characteristics and the task demands. They are named starting with the

initial positive peak (P1) at 50 ms usually maximal at the frontocentral electrodes. Next is the negative peak (N1) at around 100 ms, maximal at the vertex. P2 peaks at 150–200 ms. The negative peak (N2) is typically maximal at 200–300 ms at central sites. The P3 peak at 300–400 ms is attention dependent. Amplitude is inversely related to stimulus probability, and latency is positively related to task difficulty. Developmentally, the scalp location of the maximum depends on task conditions.

These waveform peaks each reflect several underlying components. The waveform peaks should be distinguished from the components, which refer to potential neural sources. Unless the component is large such as P3b, it usually needs to be isolated with difference waves or by experimental design (Luck 2005). The component peaks are often identified by the number of milliseconds to peak, e.g., N75 and P100. Auditory ERPs are also used to study language processing. An N400 component, maximal over central and parietal sites, is seen when there is a semantic deviation from expectations, e.g., the last word in a sentence is out of context. P3a, P3b, and N400 components do not appear before ages 3 or 4 years. A central, frontal negative component, at 400–500 ms, reflecting attention has been identified in early infants and labeled “Nc.” A recent review concluded that persons with autism show differences in many of the long-latency components (Jeste and Nelson 2009).

See Also

- Brainstem Auditory Evoked Potentials
- Electroencephalogram (EEG)
- Event-Related Potential (ERP)
- Evoked Potentials

References and Reading

- Andreassi, J. L. (2007). *Psychophysiology: Human behavior and physiological response* (5th ed.). Mahwah: Lawrence Erlbaum Associates.
- Chermak, G. D., & Musiek, F. E. (1992). Managing central auditory processing disorders in children and youth. *American Journal of Audiology*, 1, 61–65.

- Handy, T. C. (Ed.). (2005). *Event-related potentials: A methods handbook*. Cambridge: MIT Press.
- Jeste, S. S., & Nelson, C. A. (2009). Event related potentials in the understanding of autism spectrum disorders: An analytical review. *Journal of Autism and Developmental Disorders*, 39, 495–510.
- Luck, S. J. (2005). *An introduction to the event-related potential technique*. Cambridge: MIT Press.
- McPherson, D. L., Ballachanda, B. B., & Kaf, W. (2008). Middle and long latency evoked potentials. In R. J. Roeser, M. Valente, & H. H. Dunn (Eds.), *Audiology: Diagnosis* (pp. 443–477). New York: Thieme.
- Ocak, E., Eshraghi, R. S., Danesh, A., Mittal, R., & Eshraghi, A. A. (2018). Central auditory processing disorders in individuals with autism spectrum disorders. *Balkan Medical Journal*, 35(5), 367–372. <https://doi.org/10.4274/balkanmedj.2018.0853>.
- Picton, T. W., Hillyard, S. A., Krausz, H. I., & Galambos, R. (1974). Human auditory evoked potentials. I: Evaluation of components. *Electroencephalography and Clinical Neurophysiology*, 36, 179–190.

Auditory Processing

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Synonyms

Central Auditory Processing Disorder (CAPD)

Short Description or Definition

Central auditory processing disorder (CAPD) may be considered when a child is having difficulties producing or understanding verbal language. Lack of appropriate response to what others say may cause people to think the child may be deaf; however, audiological examination of children with CAPD is entirely normal. These children can hear and detect sounds, but their ability to process these sounds meaningfully is not developing as expected. These children may have difficulty recognizing sounds or discriminating between different sounds. CAPD is a controversial diagnosis that is not currently part of conventional diagnostic systems but is increasingly identified in the USA and

Australia and to a more limited extent in the UK and rest of Europe. According to the American Speech-Language-Hearing Association (ASHA) (2005), CAPD refers to difficulties in the perceptual processing of auditory information in the central nervous system and is demonstrated by poor performance in one or more of the following tasks: sound localization and lateralization; auditory discrimination; auditory pattern recognition; temporal aspects of audition, including temporal integration, temporal discrimination (e.g., temporal gap detection), temporal ordering, and temporal masking; auditory performance in competing acoustic signals (including dichotic listening); and auditory performance with degraded acoustic signals. Despite this characterization, there remains little professional agreement about how CAPD should be defined, diagnosed, or treated (Dawes and Bishop 2009).

Epidemiology

There are currently no epidemiological data concerning CAPD in children.

Natural History, Prognostic Factors, and Outcomes

There are currently no longitudinal studies of children with CAPD with which to address questions of history, prognosis, or adult outcomes.

Clinical Expression and Pathophysiology

Reported symptoms of CAPD may include difficulties understanding speech in noise, difficulties following or understanding verbal instructions, poor attention and high distractibility, and communication, language, reading, and academic difficulties.

Evaluation and Differential Diagnosis

ASHA (2005) best practice guidelines recommend diagnosis by a multidisciplinary team that includes a minimum of an audiologist and a

speech-language pathologist. Peripheral hearing should be thoroughly investigated using hearing thresholds, immittance measures, and otoacoustic emissions (Dawes and Bishop 2009). There are, however, no firm guidelines as to what standardized tests of auditory processing should be included, how many tests are required to tap the range of skills that may be compromised, or what cutoff would be indicative of a clinically significant impairment in central auditory functioning.

Part of the controversy surrounding this disorder appears to stem from the methods of assessment and the degree to which they involve speech stimuli (Dawes and Bishop 2009). When such tasks are included, it is difficult to ascertain the origin of the problem: If a child's language is impaired, he or she might perform poorly on tests of speech discrimination in noise because of limitations in linguistic ability rather than a central auditory processing disorder. On the other hand, many language-based tasks will require the auditory processing abilities listed above. ASHA (2005) clarifies the situation to some extent by stating:

although abilities such as phonological awareness, attention to and memory for auditory information, auditory synthesis, comprehension and interpretation of auditorily presented information, and similar skills may be reliant on or associated with intact central auditory function, they are considered higher order cognitive-communicative and/or language-related functions and, thus, are not included in the definition of CAPD.

Differential diagnosis is a clinical concern; Dawes and Bishop (2009) point out that 50% of children meeting criteria for CAPD also meet criteria for other developmental disorders such as ADHD, autism spectrum disorder (ASD), or specific language impairment (SLI). The degree of overlap raises issues about CAPD as a coherent diagnostic entity, and some have argued that the choice of diagnostic label reflects the conceptualization of the problem by the professional assessing the child (Ferguson et al. 2011). In other words, a child with poor attention and language delay may be diagnosed with CAPD by an audiologist, DLD by a speech-language pathologist, or ADHD/ASD by a clinical psychologist.

The difficulty is in determining the nature of the relationship between auditory processing difficulties and the developmental disorders associated with those difficulties. For example, if a child presents with delayed language development, it may be reasonable to assume that these language difficulties are the result of difficulties processing sound. However, as noted above, language difficulties may interfere with the child's ability to do tasks that assess auditory perceptual performance. Equally, there may be a third factor that disrupts both language development and auditory processing, yielding a strong association between the two even though they may be causally unrelated (see Bishop 2011 for discussion).

Tests of CAPD frequently require children to make judgments about sounds; even when the stimuli are tones rather than speech sounds, language ability may affect performance. For example, Marshall et al. (2001) reported that many typically developing children spontaneously adopted a strategy of labeling tones as "high" or "low" and that this labeling facilitated performance on similarity judgment tasks. Thus, children with SLI may be disadvantaged on assessments of CAPD, though it is the case that a substantial minority of children with SLI do experience auditory difficulties (see Dawes and Bishop 2009). It is less clear that these auditory difficulties are causally related to language impairment, though feasibly that may contribute to language learning difficulties (Bishop 2011). However, it is also clear that many children diagnosed with CAPD have considerable language difficulties and often do not differ from children with SLI with regard to language and cognitive profile (Ferguson et al. 2011). These findings again raise the question of whether these are diagnostically and etiologically distinct categories or whether they reflect professional biases.

These tasks also require children to listen carefully and attend to subtle sound differences over a large number of trials. Even typically developed children may find this challenging; for children with ADHD, it may be impossible. In order to differentiate CAPD and ADHD, Dawes and Bishop (2009) advocate the use of behavioral measures that tap visual attention. The prediction

would be that children with ADHD would have difficulties across modalities, whereas children with CAPD would be impaired only on the auditory tests. The more difficult issue to tease apart is whether performance on either measure by children with ADHD reflects attention skills or is indicative of a central processing disorder.

With regard to ASD, perceptual anomalies are frequently reported in both research and clinical settings, though again these are rarely confined to the auditory modality. In addition, the child with ASD is likely to have social deficits that may mimic auditory disorder. For example, not responding to parents calling the child's name is an early indicator of ASD but may also signal an auditory deficit. Dawes and Bishop (2009) reported that children with ASD are overrepresented at assessment centers specializing in CAPD. Research studies that use electrophysiological techniques (e.g., ERP) have suggested that the auditory impairments that characterize ASD arise because of a speech-specific, postsensory impairment related to attentional orienting (Ceponiene et al. 2003; Whitehouse and Bishop 2008). Dawes and Bishop (2009) further suggested that such top-down influences on auditory processing would require a different treatment approach to developing listening skills from the treatments recommended for CAPD.

In sum, it is likely that auditory processing problems are one of a number of "collateral" deficits commonly found in across a range of neurodevelopmental disorders (Dawes and Bishop 2009). Thus, assessment in a multidisciplinary setting will be necessary for documenting auditory deficits and considering these deficits in relation to the child's overall cognitive, linguistic, and social profile. Where possible, assessment of auditory skills that do not explicitly involve speech-based stimuli is preferable in order to avoid the confounding effects of impaired language development.

Treatment

Bishop (2011) highlighted the importance of establishing the causal role of auditory processing

in other developmental disorders because of the implications for treatment. If auditory difficulties contribute to attention or language difficulties, then it would make sense to train auditory skills with positive downstream effects for language and attention. However, if auditory deficits are associated, but do not play a causal role in disorder, such treatments would not be effective. Several computer-based training packages have been developed, with Fast ForWord (Scientific Learning Corporation) being the most popular and widely used in clinical and education contexts. This program was not specifically designed for CAPD but is based on a theoretical framework in which development language and literacy learning difficulties arise from impairments in rapid auditory temporal processing (Tallal and Piercy 1973). Fast ForWord is comprised of adaptive computer games that include acoustically modified speech; the degree of modification gradually diminishes as the child improves performance on the language-based tasks. It is most widely used for children diagnosed with SLI or dyslexia; however, rigorous trials of Fast ForWord and similar computer-based intensive auditory training have not yielded clinically significant improvements in language or literacy functioning (Loo et al. 2010; Strong et al. 2011).

There is currently a dearth of studies investigating treatment efficacy for children diagnosed with CAPD. For the most part, Dawes and Bishop (2009) report that current clinical practices do not aim to treat the auditory deficit directly, but rather aim to reduce the impact of auditory processing deficits through environmental modification (e.g., sitting the child nearer to the classroom teacher, waiting to have the child's visual attention before speaking) or by enhancing the auditory signal (e.g., using a directional microphone in the classroom). However, the effect of these modifications on developing auditory skills or improving language and academic outcomes is largely unknown.

See Also

- Auditory Discrimination
- Hearing

- Language Disorder
- Specific Language Impairment

References and Reading

- American Speech-Language-Hearing Association. (2005). Central auditory processing disorders. Retrieved March 22, 2011. <http://www.asha.org/docs/html/tr2005-00043.html>
- Bishop, D. V. (2011). *Auditory processing disorder – A cause of language problems or an incidental finding?* Wellcome Trust Guest Blog. Retrieved April 11, 2011. <http://wellcometrust.wordpress.com/2011/03/30/auditory-processing-disorder-a-cause-of-language-problems-or-an-incidental-finding/>
- Ceponiene, R., Lepisto, T., Shestakova, A., Vanhala, R., Alku, P., Näätänen, R., & Yaguchi, K. (2003). Speech-sound-selective auditory impairment in children with autism: They can perceive but do not attend. *Proceedings of the National Academy of Sciences*, 100, 5567–5572.
- Dawes, P., & Bishop, D. V. (2009). Auditory processing disorder in relation to developmental disorders of language, communication and attention: A review and critique. *International Journal of Language & Communication Disorders*, 44(4), 440–465.
- Ferguson, M. A., Hall, R. L., Riley, A., & Moore, D. R. (2011). Communication, listening, cognitive and speech perception skills in children with auditory processing disorder or SLI. *Journal of Speech, Language, and Hearing Research*, 54, 211–227.
- Loo, J. H., Bamio, D. E., Campbell, N., & Luxon, L. M. (2010). Computer-based auditory training (CBAT): Benefits for children language and reading-related learning difficulties. *Developmental Medicine and Child Neurology*, 52, 708–717.
- Marshall, C., Snowling, M. J., & Bailey, P. (2001). Rapid auditory processing and phonological ability in normal readers and readers with dyslexia. *Journal of Speech, Language, and Hearing Research*, 44, 925–940.
- Moore, D., Ferguson, M. A., Edmondson-Jones, A. M., Ratib, S., & Riley, A. (2010). Nature of auditory processing disorder in children. *Pediatrics*, 126(2), e382–e390.
- Strong, C., Torgeson, C. J., Torgeson, D., & Hulme, C. (2011). A systematic meta-analytic review of evidence for the effectiveness of the “Fast ForWord” language intervention program. *Journal of Child Psychology and Psychiatry*, 52(3), 224–235.
- Tallal, P., & Piercy, M. (1973). Defects of non-verbal auditory perception in children with developmental aphasia. *Nature*, 241, 468–469.
- Whitehouse, A. J. O., & Bishop, D. V. M. (2008). Do children with autism ‘switch off’ to speech sounds? An investigation using event-related potentials. *Developmental Science*, 11, 516–524.

Auditory Processing Disorder

- ▶ [Central Auditory Processing Disorder](#)
- ▶ [Verbal Auditory Agnosia](#)

Auditory System

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Synonyms

[Anatomy of human ear](#); [Hearing system](#); [Sensory system for sense of hearing](#)

Definition

The auditory system includes the outer, middle, and inner ears, as well as the central auditory nervous system. The outer ear includes the pinna and the external auditory meatus (ear canal). The tympanic membrane (eardrum) is the boundary between the outer and middle ear. The middle ear is housed in the mastoid portion of the temporal bone and is a completely enclosed cavity that is connected to the nasopharynx by the Eustachian tube. The middle ear houses the three smallest bones in the body, the malleus, incus, and stapes, also known as the ossicular chain. The inner ear is called the cochlea, which contains the sensory hair cells and auditory nerve fiber endings that convert mechanical energy from the middle ear into electrical energy. The VIII cranial nerve, vestibulocochlear nerve, brings the auditory information to the central auditory nervous system which consists of the brainstem nuclei (cochlear nuclei, superior olivary complex, lateral lemniscus, inferior colliculus, and medial geniculate body), the primary auditory cortex in the temporal lobe and the association auditory cortices.

The entire auditory system codes frequency, intensity, and time which are essential to the perception of sound and therefore speech.

See Also

- ▶ [Auditory Cortex](#)
- ▶ [Cochlea](#)
- ▶ [Hearing](#)

References and Reading

- Clarke, W., & Ohlemiller, K. (2008). *Anatomy and physiology of hearing for audiologists*. Clifton Park: Thomson, Delmar Learning.
- Musiek, F. E., & Baran, J. A. (2007). *The auditory system: Anatomy, physiology, and clinical correlates*. Boston: Pearson.

Auditory Verbal Agnosia

- ▶ [Verbal Auditory Agnosia](#)

Auditory Verbal Learning

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Definition

Auditory verbal learning refers to the process of acquiring and retaining new information about the sound patterns and/or meanings of words, sentences, stories, and other nonword sequences, after hearing them read aloud. A person's ability to learn the underlying sound structures and meanings of words creates the foundation for that person's ability to ultimately understand speech and use language to communicate with others. One of the core features of ASD is "a

delay in, or total lack of, the development of spoken language” (American Psychiatric Association 2000). Thus, characterizing the strategies that people develop and use to learn language during auditory verbal learning tasks could help to illuminate the mechanisms underlying communication skills in autism.

Historical Background

Research on auditory verbal learning began with the seminal work of Hermann Ebbinghaus, in the late 1800s. Ebbinghaus believed that learning verbal material required the formation of new associations between words. He also posited that the strength of these associations could be intensified with repeated exposure and practice. Thus, he designed a research program to test this hypothesis, using himself as a research subject. He developed lists of “nonsense syllables,” which consisted of consonant-vowel-consonant combinations that have no specific meanings associated with them. For example, DAX and YAT would be considered nonsense syllables, since they are not words in the English language. CAT would not be a nonsense syllable since it has a known meaning. Ebbinghaus attempted to learn his lists of nonsense syllables by slowly reading and repeating the lists to himself. Next, Ebbinghaus tried to recall as many of the syllables as he could. He discovered that his memory for the syllables improved with repeated practice of the material. In addition, he noted that his ability to learn the syllables initially improved rapidly and then more slowly over time, until he learned the material in its entirety. By characterizing these patterns, Ebbinghaus was the first to identify and map out verbal learning curves (patterns of learning over time and with repetition). He similarly identified patterns of forgetting over time and found that forgetting occurs less quickly, when the material is overlearned (repeatedly practiced, even after achieving perfect recall of the list). In addition, Ebbinghaus examined *serial position effects* and discovered that words are easier to learn at the beginning and end of a verbal learning list.

Research on auditory verbal learning continued into the twentieth century, heavily influenced by Ebbinghaus’ work and also by behaviorism, with a focus on stimulus–response aspects of language learning. Then in the 1950s and 1960s there was a shift to studying cognitive “mediators,” which were thought to be conscious mental processes that can be deployed to improve verbal learning performance. This shift was heavily influenced by verbal mediation theory and cognitive psychology, which examined internal cognitive processes rather than focusing specifically on observable behaviors. In the late 1960s and 1970s, John Flavell extended findings related to verbal mediation and described verbal learning abilities from a developmental standpoint, proposing that younger children have more trouble learning verbal information than older individuals because they have a *production deficiency*. In other words, younger children fail to spontaneously produce and use strategies to improve their performance. It was noted these children often showed significant improvements on auditory verbal learning tasks, once they were directly instructed to use specific strategies. For example, Flavell found that younger children were less likely to verbally repeat words to themselves while learning the words from a list, while older children were more likely to use verbal rehearsal with increasing age, and the spontaneous use of this strategy was associated with improvements on task performance.

Flavell’s research initiated a flurry of subsequent training studies examining whether direct instruction in strategy use improved children’s auditory verbal learning abilities. In other words, researchers took children who were not yet actively using strategies on their own and set out to see whether prompting them to use rehearsal, organization, and elaboration improved verbal learning ability. Overall, they found that the ability to use learning strategies typically develops in broad strokes throughout childhood, adolescence, and early adulthood. For example, there are gradual developmental increases in the ability to use semantic strategies and word meaning to aid verbal learning, from the preschool years through adolescence. These advancements in semantic

strategy use are generally accompanied by related improvements in verbal recall performance. Children often begin using word meaning to facilitate verbal learning during elementary school, and as preadolescents they are more likely to use semantic strategies successfully when tasks include words with strong associated meanings, and when there are directions that explicitly instruct them to use these strategies. By adulthood people can use word meaning to facilitate verbal learning, even when there are no explicit directions to do so, and when words are more subtly semantically related to one another. Similarly, verbal rehearsal also changes across development, with younger children rehearsing single words repetitively, while older adolescents rehearse multiple words in clusters. This shift from single-word to multi-word rehearsal is also associated with improved auditory verbal learning performance.

Current Knowledge

In the late 1960s researchers began examining how children with ASD learn words and more complex verbal information. This interest stemmed from the observation that individuals with ASD could engage in echolalia and use stereotyped language without necessarily understanding the core meaning of the words that they echoed. The ability to learn the sound patterns but not the meaning of words was surprising since typically developing people found it easier to learn meaningful information compared to meaningless sets of words or sound strings (Marks and Miller 1964).

Using Word Meaning to Improve Learning: Semantic Strategies

Hermelin and O'Connor were among the first to examine the relationship between word meaning and auditory verbal learning abilities in ASD. They did so by comparing children with ASD and those without ASD on their ability to learn and immediately recall verbal information with varying semantic relationships. They presented children with meaningless word strings and

meaningful sentences. They were asked to recall as much as they could remember, in each condition. Children without autism remembered significantly more sentences than word strings, while children with ASD did not show more efficient learning of meaningful information. Researchers also read children strings of unrelated words and strings of related words from a shared semantic category, such as colors or utensils. Children with ASD were much less likely to group words together from the same category than children without autism. Collectively, these studies suggest that children with ASD were less likely to use word meaning to aid auditory verbal learning. They were also more likely to rely on phonological features or sound patterns of the words rather than word meaning. It is important to note that these early studies primarily involved children who had ASD and intellectual disability. Subsequent research looked at both high- and low-functioning individuals with ASD; although studies yielded mixed findings, they generally support the observation that people with ASD are less likely than those without ASD to use word meaning to improve learning and memory of verbal information.

Using Word Order to Improve Learning: Primacy and Recency Effects

The location and order of words within a word-learning list can also be used to improve auditory verbal learning skills. Scientists have studied whether individuals remember certain parts of a list more readily than other parts, and whether recalling words from the beginning, middle, or end of a list is associated with better learning and memory overall. Remembering words from the beginning or first portion of a list is referred to as the primacy effect. This pattern of recall is thought to reflect the active use of verbal rehearsal, a strategy that involves repeating words over and over again to facilitate retention. Verbal rehearsal has been shown to improve auditory verbal learning in typically developing individuals. Conversely, remembering words from the end of a word list is often referred to as a recency effect, and is thought to reflect a more shallow

level of processing that involves simply echoing back the sounds that were most recently heard. Low-functioning individuals with ASD tend to rely more heavily on rote memory abilities and are more likely than people without autism to simply echo back words from the end of a list. In other words they tend to show a stronger recency effect than people without ASD. This suggests that they rely on more simple and less efficient learning strategies than individuals without autism, who are more likely to use verbal rehearsal to aid learning. Individuals who are high-functioning with ASD show a different pattern of verbal learning and memory. They have demonstrated typical primacy and recency effects when compared to people without autism. The degree to which individuals with autism group words together, based on order, varies across studies; some research has found typical serial position effects while other studies have not. Although overall, individuals with autism appear less able to actively deploy learning strategies efficiently to support their verbal learning.

Using Repetition to Improve Learning: Learning Curves and Retention over Time

To examine auditory verbal learning over time, researchers have used experimental paradigms that involve reading a single list of words over a series of repeated trials. Verbal learning curves are quantified over time to determine how much new information an individual retains with each repetition of the verbal material. Some researchers have used the California Verbal Learning Test (CVLT; a standardized measure of verbal learning and memory) to examine the rate of verbal learning in ASD compared to controls. During the CVLT participants hear a single list of nouns read aloud on five consecutive learning trials. After each trial, participants are asked to immediately recall as many words as they remember. The list has a fixed word order and an underlying semantic structure, meaning that each word on the list belongs to one of a few semantic categories, such as fruits or furniture. When compared to people without ASD, adolescents and adults with high-functioning ASD show typical rates of

verbal learning on early learning trials and poorer recall on later trials. This suggests less efficient auditory verbal learning over time. In other words, their ability to learn new verbal information over time slows down more quickly over repeated trials in comparison to people without ASD. In addition, individuals with ASD were less likely to cluster words together based on shared semantic categories or the order in which they appeared in the original list. In this case, slower learning was likely attributable to less efficient use of learning strategies over time.

To summarize our current knowledge, the research to date suggests some general trends in auditory verbal learning abilities in ASD. First, individuals with ASD are less likely than people without ASD to use word meaning and semantic structure to enhance their learning abilities. Second, they are also less likely to use other active learning strategies, like verbal rehearsal and serial clustering. Finally, when word lists are read repeatedly, individuals with ASD tend to learn words less efficiently over time. Although these are general trends observed in the research literature, patterns of auditory verbal learning have not been entirely consistent across all studies, and these trends are observed more often in low-functioning individuals than in higher-functioning individuals with ASD.

Future Directions

There are a number of possible avenues for future research on auditory verbal learning in ASD. First, future research could adopt a developmental perspective, using longitudinal studies that examine auditory verbal learning abilities as people age and develop throughout their lifespan. Our knowledge about auditory verbal learning in ASD comes largely from cross-sectional studies, which provide a snapshot of verbal learning abilities by capturing performance at a single time point in a person's life. Larger scale longitudinal studies focusing on the emergence and active use of different types of verbal learning strategies at multiple points within a person's life would help

to identify whether specific patterns of learning are simply delayed in ASD or whether they remain consistently impaired throughout the lifespan. Knowing more about the development and use of learning strategies and auditory verbal learning skills over time could help to shape interventions designed to improve verbal learning and communication in this population.

In the same vein, training studies could be conducted to explicitly instruct individuals with ASD to use strategies known to improve auditory verbal learning performance. Such studies could help to evaluate whether explicit instruction on strategy use would improve verbal learning in the short term. In addition, this line of research would also help to determine whether improvements due to instruction could be sustained over time, without additional instruction and maintenance.

Finally, there has been considerable theorizing about underlying neurobiological mechanisms responsible for impaired auditory verbal learning in ASD. In particular, temporal and frontal lobe regions have been implicated, as well as more distributed problems with connectivity. Most studies investigating auditory verbal learning in ASD compare performance on list-learning tasks in individuals with ASD to patterns of performance in individuals with known brain lesions. This approach provides a starting point for identifying underlying neural mechanisms. However, future research could extend this research by utilizing imaging techniques to identify patterns of brain activation during verbal learning tasks in individuals with ASD, and to specify whether particular regions of the brain are related to less efficient verbal learning in this population.

See Also

- [California Verbal Learning Test, Children's Version \(CVLT-C\)](#)
- [Language Acquisition](#)
- [Memory](#)
- [Memory Assessment](#)
- [Semantic Memory](#)
- [Wide Range Assessment of Memory and Learning \(WRAML\)](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
- Aurnhammer-Frith, U. (1969). Emphasis and meaning in recall in normal and autistic children. *Language and Speech*, 12, 29–38.
- Baddeley, A. D., & Hitch, G. (1974). Working memory. In G. A. Bower (Ed.), *The psychology of learning and motivation* (pp. 47–89). New York: Academic.
- Bennetto, L., Pennington, B. F., & Rogers, S. J. (1996). Intact and impaired memory functions in autism. *Child Development*, 67, 1816–1835.
- Beyersdorf, D. Q., Anderson, J. M., Manning, S. E., Nordgren, R. E., Felopulos, G. J., Nadeau, S. E., et al. (1998). The effect of semantic and emotional context on written recall for verbal language in high-functioning adults with autism spectrum disorder. *Journal of Neurology, Neurosurgery, & Psychiatry*, 65, 685–692.
- Bjorklund, D. F., Muir-Broadbent, J. E., & Schneider, W. (1990). The role of knowledge in the development of strategies. In D. F. Bjorklund (Ed.), *Children's strategies: Contemporary views of cognitive development*. Hillsdale: Erlbaum.
- Boucher, J. (1978). Echoic memory capacity in autistic children. *Journal of Child Psychology and Psychiatry*, 19, 161–166.
- Boucher, J. (1981). Immediate free recall in early childhood autism: Another point of behavioural similarity with the amnesic syndrome. *British Journal of Psychology*, 72, 211–215.
- Boucher, J., & Warrington, E. K. (1976). Memory deficits in early infantile autism: Some similarities to the amnesic syndrome. *British Journal of Psychology*, 67, 73–87.
- Bowler, D. M., Gaigg, S. B., & Gardiner, J. M. (2008). Subjective organisation in the free recall learning of adults with Asperger's Syndrome. *Journal of Autism and Developmental Disorders*, 38, 104–113.
- Flavell, J. H., Beach, D. H., & Chinsky, J. M. (1966). Spontaneous verbal rehearsal in a memory task as a function of age. *Child Development*, 39, 53–58.
- Fyffe, C., & Prior, M. (1978). Evidence for language recoding in autistic, retarded and normal children: A re-examination. *British Journal of Psychology*, 69, 393–402.
- Hermelin, B., & O'Connor, N. (1967). Remembering of words by psychotic and subnormal children. *British Journal of Psychology*, 58, 213–218.
- Marks, L. E., & Miller, G. A. (1964). The role of semantic and syntactic constraints on the memorization of English sentences. *Journal of Verbal Learning and Verbal Behavior*, 3, 1–5.
- Minshew, N. J., & Goldstein, G. (1993). Is autism an amnesic disorder? Evidence from the California Verbal Learning Test. *Neuropsychology*, 7, 209–216.
- Minshew, N. J., & Goldstein, G. (2001). The pattern of intact and impaired memory functions in autism. *Journal of Child Psychology and Psychiatry*, 42, 1095–1101.

Pressley, M., & Schneider, W. (1997). *Introduction to memory development during childhood and adolescence*. Mahwah: Lawrence Erlbaum Associates.

Ramondo, N., & Milech, D. (1984). The nature and specificity of the language coding deficit in autistic children. *British Journal of Psychology*, 75, 95–103.

Schneider, W., & Sodian, B. (1997). Memory strategy development: Lessons from longitudinal research. *Developmental Review*, 17, 442–461.

Schwartz, S. (1981). Language disabilities in infantile autism: A brief review and comment. *Applied Psycholinguistics*, 2, 25–31.

Tager-Flusberg, H. (1991). Semantic processing in the free recall of autistic children: Further evidence for a cognitive deficit. *British Journal of Developmental Psychology*, 9, 417–430.

Tulving, E. (1962). Subjective organization in free recall of “unrelated” words. *Psychological Review*, 69, 344–354.

Auditory Verbal Learning Test

► [Rey Auditory Verbal Learning Test \(Rey AVLT\)](#)

Augmentative and Alternative Communication (AAC) Device

- [Assistive Devices](#)
- [Communication Board](#)

Augmentative and Assistive Technology

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Definition

Assistive technology A broad range of devices, services, strategies, and practices that address the

problems faced by individuals who have disabilities (Cook and Polgar 2008)

Assistive technology device “Any item, piece of equipment, or product system, whether acquired commercially off the shelf, modified, or customized, that is used to increase, maintain, or improve functional capabilities of individuals with disabilities” (Assistive Technology Act of 2004)

Assistive technology service “Any service that directly assists an individual with a disability in the selection, acquisition, or use of an assistive technology device” (Assistive Technology Act of 2004)

Augmentative and alternative communication (AAC) One type of assistive technology that specifically focuses on the strategies, interventions, and supports that assist a person with a disability to communicate

Historical Background

In 1988 the Technology-Related Assistance for Individuals with Disabilities Act (1988), called the Tech Act, provided support for activities to help individuals with disabilities to obtain assistive devices and services. The Tech Act focused on helping states create systems that would give individuals with disabilities improved access to assistive technology devices (Bausch et al. 2005). In 2004 the Assistive Technology Act, called the AT Act, continued support for the systems established by the earlier legislation. The AT Act also recognized that “there is a continued need to provide information about the availability of assistive technology, advances in improving accessibility and functionality of assistive technology, and appropriate methods to secure and utilize assistive technology. . .” (AT Act 2004). The new law required majority of states’ funding that was to be used specifically for programs

focusing on assistive technology reutilization, demonstration, and device loans (AT Act 2004).

Both the Individuals with Disabilities Education Act of 2004 (IDEA) and Rehabilitation Act of 1973 speak to the provision of assistive technology in school. IDEA states that assistive technology devices and services must be made available “if required as a part of the child’s special education, related services or supplementary aids and services” (2004). While IDEA uses the AT Act terminology in its definition of assistive technology, it also more specifically outlines the responsibilities related to application in the educational setting. The Rehabilitation Act of 1973 ensures that students who do not qualify for special education but require AT are still provided access to those supports and services.

Current Knowledge

Assistive technology (AT) has been integrated into our work, home, school, and community through programs that focus on identifying, obtaining, and using assistive technology in order to maximize the independence and participation of individuals with disabilities in society (Tech Act 1988). This process of matching the person with a disability, the activity or task, and the assistive technology device is outlined in the definition of assistive technology service found in the AT Act (2004). The types of activities that are part of assistive technology service include evaluation of AT needs of the person, including in relevant and functional environments; procuring the device; conducting tasks associated with obtaining and maintaining the proper device; coordinating different stakeholders to support the assistive technology; training or technical assistance; and expanding access to AT (AT Act 2004).

The definition of a person with a disability, according to the Americans with Disabilities Act, is “a person who has a physical or mental impairment that substantially limits one or more major life activities” (ADA n.d.). In order to access the available AT resources, an individual with autism spectrum disorder or other disabilities will need the support of knowledgeable team

members who can be actively involved in the AT evaluation and implementation. The team can now move toward the identification of appropriate AT tools, training, and technical assistance necessary to increase the person’s abilities to successfully participate in a range of life activities as independently as possible.

The qualifications of the evaluation team members are not specified in the law, but at a minimum the team should be able to execute the steps of an assistive technology assessment including identifying the relevant strengths and challenges of the individual knowledgeable about the range of AT options that are available for consideration (OCALI 2013). AT services are provided by professionals in a number of different fields, such as speech pathology, occupational therapy, physical therapy, engineering, and special education. The law does not require evaluators to have specific credentials; the Rehabilitation Engineering and Assistive Technology Society of North America (RESNA) has developed a certification program so that professionals from related fields can be certified as an Assistive Technology Professional (ATP). ATPs are skilled with evaluating the needs of individuals, matching them to AT, and helping with training in implementation. RESNA has also identified standards of practice in the field of assistive technology which guide the work of ATPs for consistency and fidelity.

Consideration of a person’s assistive technology needs requires an understanding of the user’s abilities and challenges, the context for application, and relevant experiences with other AT supports and strategies. The World Health Organization refers to the term disability as “reflecting the interaction between features of a person’s body and features of the society in which he or she lives” (WHO). This definition highlights the relationship between a person’s abilities and the performance expectations as opposed to a specific diagnosis. The elements of the assessment of the person’s strengths and difficulties are not prescribed by law. Evaluation teams may assess the person’s vision and hearing abilities, as well as their mobility, cognition, learning, communication, and social skills. To understand the impact of the disability fully, functional contexts must be

included in the evaluation. Some other major life activities that could be observed are recreation, daily living, self-care, and working. Activities such as reading, concentrating, standing, lifting, bending, and others (ADA 2011) represent an even broader interpretation of potential applications of assistive technology (Pacer).

Selecting assistive technology is a dynamic process of matching the task, the person, and the tool. There are many resources available online and in texts to guide teams through the process of assessing the need for assistive technology, such as the SETT process (Zabala 2005e), the Wisconsin Assistive Technology Initiative (WATI 2009a), and OCALI AT Resources (2013). In addition to information about specific AT devices, these documents also outline a sequence of steps for an AT evaluation, such as the one from OCALI (2013) below:

1. What are the specific tasks the person needs to perform?
2. What barriers exist, and what is the impact on the person's participation in the task?
3. What strategies and tools have been tried, and what were the results?
4. What AT tools or strategies might be useful in overcoming these barriers or challenges?
5. What is the plan for implementation of the identified AT solutions?

What are the specific tasks the person needs to perform? The identification of appropriate AT starts with determining where and when the AT is needed. Activities that may require AT supports cover all aspects of life, including daily living, vocational, educational, recreational, and social activities (Reed 2009). Within each of these activities, consider the specific tasks that the person wants or needs to perform. These environments and the expectations associated with them represent potential applications of AT.

For the person with autism spectrum disorder or other disabilities, their ability to participate in these settings and activities can be impacted by differences in sensory input, movement, cognition and learning, language and communication, or social skills. Some of the AT resources above

contain checklists that can help the team to focus on identifying the person's abilities in that context as well as their disability-related challenges (Reed 2009).

What barriers exist, and what is the impact on the person's participation in the task? Using information that was collected about important contexts and about the person, the team can begin to record the specific challenges that were observed. This information will inform more clearly the purpose and important features of the AT device. Inaccurate identification of the need will make it much less likely that the AT device will be helpful, so this is a key step in the process.

Before moving ahead to thinking about new AT solutions, it is useful to ask find out what strategies and tools that have been tried for this purpose, and what were the results? Being able to reflect on what has been tried and how well it worked will help narrow the scope of the search. Are there features of previous efforts that were beneficial? What other AT options include these features and might be considered? Collecting this type of information can save time and help generate more focused solutions.

A careful process of looking at the context, identifying challenges and contributing factors, and considering prior AT experiences will form a good foundation for identifying AT solutions. What AT tools or strategies might be useful in overcoming the barriers? As part of making that decision, the team should consider the full range of AT options that are available, from the simplest environmental adaptation to a basic tool to a complex computer-based device. Reed (2009) suggests that teams look at a range of sources to learn about options, including books, catalogs, websites, or actual hardware or software. Here is where it is important to have as part of the team at least one person who has some knowledge about relevant assistive technology (Reed 2009) to help evaluate the findings. Different options of AT to meet different needs are presented in the next section.

Based on the collected information, what would be the best idea to try next? What is the plan for implementation of the identified AT solutions? This is the point in the process where the specific device and services are selected. The clear

determination of the target tasks, the assessment of the person's abilities and challenges in that context, and consideration of prior efforts will prepare the assessment team to make informed decisions and thoughtful recommendations for next steps.

Once a decision has been made about what AT to try, a plan for monitoring the trial should be identified. How will training be provided? How and when will the AT be used? What are the indicators of success? What might suggest that the device is not meeting the identified need? Again, the online AT resources are excellent references for suggestions of questions to ask (WATI 2009b; Zabala 2005d; OCALI 2013). Monitoring the final steps of the process is as important as the other steps in ensuring proper implementation and also documentation of benefits and deficiencies.

Even with a good assessment process, it still may be necessary to conduct trials of multiple tools before a solution is identified (Reed 2009). The team may have already created a list of ideas from which to draw their next AT support. Additional trials should follow the same process of planning and documentation until a successful match is made.

The evaluation process defined the context of the need and the necessary features of the device. The step that involves a discussion of potential AT solutions requires an awareness of the continuum of options available for the identified need. The sections below are organized according to broad areas of need: sensory; seating, positioning, mobility, and transportation; reading, writing and computer; cognition and learning; and communication. A brief description of the skills or functions included in that section is followed by list of examples of AT for those purposes. More general names of different types of supports are used as opposed to naming of specific devices, hardware, software, or materials that would be quickly outdated. The topic of communication is covered in detail following the other areas.

AT options related to vision (e.g., cortical vision, visual acuity) include large-print books, Braille, digital books, screen readers, magnification aids, and optical aids.

AT options related to hearing (e.g., hearing acuity, auditory processing, auditory sensitivity) include hearing aids, speech processors, FM systems, alerting systems, captioning, and noise-cancelling headphones.

AT options related to seating, positioning, mobility, and transportation include manual and power wheelchairs, walker, cane, adapted table, stander, seat cushion, adapted chair, positioning wedges, grab bar, wheelchair van, wheelchair lift, and specialized car seat.

AT options for reading, writing, and computer interface include audiobook, E-book or electronic book, screen reader, alternate keyboard, on-screen keyboard, keyguard, joystick, alternate mouse, stylus, voice commands, speech to text application, word prediction applications, eye gaze technology, electronic switch, adapted pen/pencil, and portable electronic device.

AT options for activities of daily living and environmental control (operation of electronics such as lights, phone) include adaptive switches, adapted utensils, specialized handles and grips, switches, alternate keyboards, computers, remote controls, voice command speakers, and portable electronics.

AT to support cognition and learning, such as attention, memory, information processing, knowledge representation and organization, problem-solving, language, and learning (Cook and Polgar 2008), prioritizing, planning, organizing, self-monitoring, and working memory (Temple 2013) includes electronic and nonelectronic materials such as timers, watches, alarms, calendars, reminders, audio recorders, schedules, task lists, sticky notes, graphic organizers, and work systems.

Assistive technology that specifically addresses the needs associated with impairments in speech and communication is called augmentative and alternative communication or AAC. Augmentative communication (AAC) tools and interventions are typically used to supplement a person's existing communication abilities, including any natural speech, rather than replacing it. The purpose of AAC, according to

Light, McNaughton, and Caron (2019), is to “(a) enhance language learning, (b) facilitate social interaction, (c) improve literacy skills, (d) increase participation in society, and (e) teach partner interaction strategies” (p. 26).

No-tech or unaided communication supports use a person’s own body or the environment with no requirement for equipment. Examples include speaking, pointing, gestures, facial expressions, and manual signing. The strengths of unaided methods are that they do not require preparation or management of materials and they are always available and cannot be lost, broken, or damaged and do not cost anything. When used in a context that has information to support a person’s message, gestures, vocalizations, and facial expressions can be very effective forms of communication.

Low-tech communication systems include nonelectronic and paper-based materials. Communication boards, communication books, and paper communication displays are all examples of low-tech supports. The benefits of low-tech supports are that they are typically inexpensive, lightweight, portable, and easily customized. Individuals who use high-tech communication supports often have a low-tech version, created by printing a copy of the displays to use in situations when the device is unavailable or impractical.

High-tech communication aids are often called “speech-generating devices” or SGDs. Because they are computer-based, high-tech SGDs offer many options for how messages can be organized, displayed, and generated. The type of display used in high-tech devices is called a “dynamic display,” where the message targets displayed on the device will change based on the user’s selection. Most high-tech SGDs allow the user to determine the number of cells, or targets, that are displayed on the screen. Some devices will allow the user to create their own cell size and configuration, while others offer a specific set of page layout options. Many devices come with page sets where the display, messages, representations (symbols), and behaviors (response) have already been programmed. By selecting from a set of preprogrammed displays, the person using the device can make necessary edits to individualize their communication support.

When using a keyboard display, as the user types, the letters and words are displayed in a message window or screen that is usually above the keyboard. The message is then spoken by the device. When the device speaks what a user has typed, it is using a form of technology called “text to speech,” which refers to the process of converting the text entered by the user into spoken output.

Until recently, high-tech dynamic display devices were the least available and most expensive type of communication support. With the increase in the use of portable electronics in the general population, the availability of these devices for individuals requiring AAC has also increased. Software applications that support communication have been developed for commercially available electronic devices and can easily be added to mobile devices such as smartphones and tablets (e.g., iPhones, iPads, android phones, and tablets). McNaughton and Light (2013) noted positive contributions of these options included “increased awareness and social acceptance of AAC in the mainstream, greater consumer empowerment in accessing AAC solutions. . .” (p. 107).

With the rapid development of new applications and changes in operating system features, a list of specific communication apps would not be a very relevant resource for long. Instead, the work on mobile media devices (e.g., tablets, phones) by Caron and Shane (2014) outlines the app features that were most desirable for individuals with ASD: purpose; output (speech produced); speech settings; representation (symbols), display (layout, design), and feedback (visual, auditory, tactile); rate enhancement (prediction), access, and motor; and app interface (social). This might be a more useful reference when considering AAC devices.

Future Directions

In their entry on mobile technology, McNaughton and Light (2013) identify evidence-based practices in the field of AAC that are critical to the implementation of any AAC device: proper

assessment of the individual and communicative contexts; device selection and customization based on assessment; focused intervention to increase competency; and training and support of partners.

The concept of universal design has the potential to reduce the need to use assistive technology or individual adaptations. Applying the principles of universal design increases accessibility for all and decreases the need for specialized products for individuals.

See Also

- [American Sign Language \(ASL\)](#)
- [Facilitated Communication](#)
- [Gestures](#)
- [Manual Sign](#)
- [Nonverbal Communication](#)
- [Pictorial Cues/Visual Supports \(CR\)](#)
- [Picture Exchange Communication System](#)
- [Sign Language](#)
- [Total Communication \(TC\) Approach](#)
- [Visual Scanning](#)
- [Visual Supports](#)
- [Visual-Motor Function](#)

References and Reading

- ADA National Network. (n.d.). <https://adata.org/>
- American Speech-Language-Hearing Association. (n.d.). Autism. <https://www.asha.org/PRPSpecificTopic.aspx?folderid=8589935303§ion=Treatment>
- Assistive Technology Act. (2004). Pub. L. No. 108-364.
- Bausch, M., Mittler, J., Hasselbring, T., & Cross, D. (2005). Assistive Technology Act of 2004: What does it say and what does it mean? *Physical Disabilities: Education and Related Services*, 23(2), 59–67 <https://files.eric.ed.gov/fulltext/EJ842007.pdf>
- Cafiero, J. M. (2005). *Meaningful exchanges for people with autism*. Bethesda: Woodbine House.
- Caron, J. G., & Shane, H. (2014). Mobile media devices: A paradigm shift in communication technology for persons with autism spectrum disorder. In K. I. Boser, M. S. Goodwin, & S. C. Wayland (Eds.), *Technology tools for students with autism: Innovations that enhance independence and learning*. Kindle. Amazon.
- Baltimore, MD: Brookes Publishing.
- Cook, A. M., & Polgar, J. M. (2008). *Cook and Hussey's assistive technologies principles and practice*. St. Louis: Mosby Elsevier.
- Hershberger, D. (2011). Mobile technology and AAC apps from an AAC developer's perspective. *Perspectives on Augmentative and Alternative Communication*, 20(1), 28–33. <https://doi.org/10.1044/aac20.1.28>.
- Individuals with Disabilities Education Act, 20 U.S.C. § 1400 (2004).
- Light, J., McNaughton, D., & Caron, J. (2019). New and emerging AAC technology supports for children with complex communication needs and their communication partners: State of the science and future research directions. *Augmentative and Alternative Communication*, 35(1), 26–41.
- McNaughton, D., & Light, J. (2013). The iPad and mobile technology revolution: Benefits and challenges for individuals who require augmentative and alternative communication. *Augmentative and Alternative Communication*, 29(2), 107–116.
- O'Neill, T., Light, J., & Pope, L. (2018). Effects of interventions that include aided augmentative and alternative communication input on the communication of individuals with complex communication needs: A meta-analysis. *Journal of Speech, Language, and Hearing Research*, 61(7), 1743–1765. https://doi.org/10.1044/2018_JSLHR-L-17-0132.
- Ohio Center for Autism and Low Incidence Assistive Technology Resource Guide. (2013). Assistive Technology & Accessible Educational Materials Center. https://www.ocali.org/up_doc/AT_Resource_Guide_2013.pdf
- Quality Indicators for Consideration of Assistive Technology Needs. (2012). <https://qiat.org/indicators.html>
- Reed, P. (2009). Overview of the assessment and planning process. Assessing students' needs for assistive technology by Wisconsin Assistive Technology Initiative. http://www.wati.org/wp-content/uploads/2017/10/ASNAT_4thEditionDec08.pdf
- Rehab Engineering and Assistive Technology of North America. (2019). AT standards. <https://www.resna.org/at-standards>
- Rehabilitation Act of 1973. (2004). <https://legcounsel.house.gov/Comps/Rehabilitation%20Act%20Of%201973.pdf>
- RESNA Research Committee. (2014). RESNA's guidelines and priorities for assistive technology and rehabilitation engineering research. Rehab Engineering and Assistive Technology of North America. https://www.resna.org/sites/default/files/legacy/library/docs/RESNA%20Research%20Guidelines_February%202017.pdf
- Schlosser, R. W., & Koul, R. (2015). Speech output technologies in interventions for individuals with autism spectrum disorders: A scoping review. *Augmentative and Alternative Communication*, 31, 285–309.
- Technology Related Assistance to Individuals with Disabilities Act of 1988 Pub. L. 100-407. 29 USC 2201 <https://www.govinfo.gov/content/pkg/STATUTE-102/pdf/STATUTE-102-Pg1044.pdf>
- Temple, C. (2013). Executive function skills and assistive technology. *Perspectives on Language and Literacy*, 39(4), 15–17. <https://search-proquest-com.ezproxy.uvm.edu/docview/1498909430?accountid=14679>
- United States., & Job Accommodation Network (U.S.). (2011). The ADA Amendments Act of 2008.

- Morgantown, WV: U.S. Dept. of Labor, Office of Disability Employment Policy, Job Accommodation Network.
- Wisconsin Assistive Technology Initiative. (2009a). Assistive technology consideration to assessment. <http://www.wati.org/free-publications/assistive-technology-consideration-to-assessment/>
- Wisconsin Assistive Technology Initiative. (2009b). Assessing students' needs for assistive technology. <http://www.wati.org/free-publications/assessing-students-needs-for-assistive-technology/>
- World Health Organization. (2020) Health topics: Disabilities. <https://www.who.int/topics/disabilities/en/>
- Zabala, J. (2005a). SETT scaffold for consideration of AT needs. http://joyzabala.com/uploads/Zabala_SETT_Scaffold_Consideration.pdf
- Zabala, J. (2005b). SETT scaffold for data gathering. http://joyzabala.com/uploads/Zabala_SETT_Scaffold_Data_Gathering.pdf
- Zabala, J. (2005c). SETT scaffold for tool selection. http://joyzabala.com/uploads/Zabala_SETT_Scaffold_Tool_Selection.rtf
- Zabala, J. (2005d). SETT implementation and evaluation effectiveness planning. http://www.joyzabala.com/uploads/Zabala_SETT_Scaffold_Implementation.pdf
- Zabala, J. (2005e). SETT Framework Documents. <http://joyzabala.com/Documents.html>

Australia and Autism

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Historical Background

Australia has a land area about 80 % the size of the USA, China, Europe, and Canada, and 30 times the size of the UK. The total population of Australia is 23 million, with most inhabitants living in several large cities, each with a population of between one and four million. There is a federal system of government, as well as six states and two territories, each with its own state legislature and service systems for health, education, community services, and law. Further, each city or municipal area has a local council and

responsibility for services that impact on people with disability. There are thus three levels of government all with involvement in services of various kinds, quite apart from a range of private foundations, organizations, and corporations which also provide service.

The history of autism in Australia follows that in the USA and the UK, beginning with the pioneering work of Kanner in the USA (Kanner 1943). However, specific interest in autism within Australia, apart from individual clinical work, did not emerge until the 1960s. At this time, significant research findings were beginning to emerge from studies in the UK and USA, and trends in child and adolescent psychology and psychiatry in Australia largely followed developments in these countries. Initially most research and clinical interest was in children in early school year (Bettelheim 1967; DeMeyer et al. 1972; Bartak et al. 1975), usually male, with full scale IQ under 70, with quite severe features of autism and with parents who appeared emotionally stressed. Many clinical programs were focused on the involvement of parents in the development of their child's autism. This was partly because autism was still embedded in psychiatry, and child psychiatric programs in Australia had tended to follow a psychodynamic orientation in the absence of much in the way of objective research on causes of behavioral problems generally. Input from other health professionals from the 1950s to the 1980s would often be limited to neurological opinion to exclude underlying medical conditions. The work of Bettelheim (1967) more specifically in the area of autism within a psychodynamic framework also influenced programs in Australia at this time. However, from the 1960s, new approaches, taking into account behavioral criteria for autistic disorder and intellectual ability, were beginning in Australia, following developments overseas, including the work of Rutter and Schopler (1987), Rimland (1964), DeMeyer et al. (1972), and Ornitz and Ritvo (1968) in the USA and Bartak et al. (1975), Hermelin and O'Connor (1970), and Frith (1989) in the UK.

From the 1980s, in line with other changes in the way services were provided to children (see below), Australia has adopted objective

diagnostic criteria as they have been developed and is looking with interest at the new Diagnostic and Statistical Manual-5 criteria for autism from the American Psychiatric Association (2013).

Diagnosis

As mentioned above, diagnosis of autism was done predominantly by psychologists and psychiatrists in Australia until the 1980s. The service context is important here in thinking about the changes that have occurred over time and the current situation. In Australia, the federal government funds health professionals, including doctors, allied health professionals, and psychologists through Medicare funding, to work in private health-care settings which are run as businesses. In 2008, the federal government launched a funding program called “Helping Children with Autism” (HCWA) that provided funding for diagnosis and assessment of children suspected as having autism, using Medicare billing. The HCWA funding also included intervention funding (see below). The states and territories provide funding for public health-care facilities, including the salaries of health-care professionals. Each state and territory can take and has taken a different, and often changing, approach to the service type and location that they will fund for diagnosis and assessment of developmental problems, including autism. Some diagnostic services are based in hospitals and others in community settings. Consistent features across states and territories have been (1) the emergence of multidisciplinary teams based in publicly funded services, from the 1960s but not widespread until the 1980s for the diagnosis and assessment of children with developmental problems, including autism, usually including a pediatrician (less commonly a child psychiatrist), speech pathologist, and psychologist, but with varying composition between states and territories and between regions within states and territories and no given per capita rationale for geographic location; (2) increasing numbers of pediatricians, speech pathologists, and psychologists working in private settings, in isolation but providing parts of the assessment required for a diagnosis of autism; and (3) greater, but not consistent between

services or clinicians, use of standardized assessment instruments (e.g., Autism Diagnostic Observation Schedule (ADOS) (Lord et al. 2001), Ehlers and Gillberg’s High Functioning Autism Syndrome Screening Questionnaire (ASSQ) (1999), the Autism Diagnostic Interview-Revised (ADI-R) (Rutter et al. 2003), or the Diagnostic Interview for Social and Communication Disorders (DISCO) (Wing et al. 2002)). In Western Australia, an active group of clinicians has developed a standard assessment model which has formed the basis of a multidisciplinary diagnostic and assessment procedure. This procedure has not yet been taken up throughout Australia. Consistent with other high-income countries, Australia has seen a trend toward earlier diagnosis of autism, such that it is now common to see children as young as 2 years of age, and sometimes less than two, presenting with concerns. Children with relatively good language skills are sometimes still not being seen by diagnostic teams until 8–12 years of age.

In parallel with other high-income countries, milder cases are also being diagnosed in Australia. The term autism spectrum disorder had high uptake in Australia by the early 2000s. That conceptualization combined with the use of DSM-IV’s diagnostic category pervasive developmental disorder-not otherwise specified (PDD-NOS) and the acceptance of PDD-NOS as sufficient for service entry saw an increasing number of children with fewer diagnostic features and of lower severity level being labeled.

Early Intervention

Australia has followed the USA, Canada, and the UK in embracing early intervention for children with developmental disorders, including autism. Publicly funded early intervention services developed in Australia during the 1960s. Autism associations also began in Australia in the 1960s, and in that role were some of the earliest “early intervention” providers. Traditionally state governments have provided early intervention for children with substantial developmental problems, based on functional ability and needs rather than diagnosis. The HCWA funding of 2008 transformed the early intervention landscape in

Australia, with families receiving some dedicated federal funding for early intervention services and with one early intervention-enriched child care facility being established in every Australian state. The HCWA funding package provides funding for early intervention for children from diagnosis up to their seventh birthday (however, they must be registered by their sixth birthday), with five being the age of school entry for most children in Australia. An essential and increasing feature of these programs has been to provide support and education for parents so that they can supplement the program activities with structured activities at home.

Education

In 1984, the State of Victoria conducted a review of educational services for disabled children. Prior to this, Victoria and other Australian states had a long history of provision of specialized educational settings for children with various disabilities. Some special schools catered for children with IQ under 70, others for children with sensory or motor difficulties. Schools for children with severe intellectual disability were run by the State Health department as were a number of large long-stay residential institutions, some of which had as many as 900 residents many of whom had been there since early childhood for periods of many years. Children with autism had often been thought to be ineducable and tended to end up in small Health Department schools or the larger residential training centers. The review resulted in radical findings and recommended total inclusion in normal schools for all children with a disability. The small schools for children with severe disability were to be transferred to the State Education Department, and it was expected that all specialist schools would be gradually phased out and closed as all children with a disability would eventually be enrolled in local community public or private schools providing general educational facilities. Where a school had more than six children with special needs, the State Education Department would provide resources to employ a special education teacher and classroom aides. Similar changes have occurred elsewhere in Australia. However, in

many regular schools, aides have often been left to work on their own with children with a disability with insufficient specialist guidance. In addition, closure of special schools has not continued. As a result, while many children with autism go to regular schools, a significant number continue to attend specialist schools which were originally set up for children with intellectual disability and have not been geared to the specific needs of children with autism. Outcomes for children with autism in all of these schools have tended to depend upon the quality of individual teachers and in regular schools, upon the degree of support of school principals. In addition, starting in NSW and Victoria in the 1970s, special schools for children with autism were set up with financial support by parents and private charitable foundations. This followed a similar pattern to what had occurred in the USA, UK, and other parts of the world. In Australia, State Educational Authorities have come to provide some degree of financial and administrative support to such schools in most states.

Adult Years

Over time state autism associations and service providers have recognized at least to some extent that children do not grow out of autism but rather grow with it and become adults with autism. One consequence has been the development of social skills programs that are relevant to the development of both casual and more permanent and intimate social relationships. Some of these include sexual and other health topics as well as the development of basic interpersonal communication skills. As an example, one very successful program developed in Victoria was a "Train the Trainer" program. This was an established course already taught at a technical and further education college which qualified people to become adult education trainers. It was modified to focus specifically on autism. Successful graduates were trained to be public speakers and trainers in the field of autism and could run single sessions or short courses for members of the general public to learn about autism. Twenty adults with autistic disorder were recruited to take the course. Much

of the course involved group project activity as well as extensive class discussion. To some degree of surprise amongst both staff and the class members themselves, participants enjoyed the group activities, and ultimately, 17 of the 20 completed the course and obtained graduate certificates as trainers.

Autism Associations

All states in Australia have autism support associations. These were mostly set up by parents of children with autism when there were few facilities and little governmental support, beginning in the 1960s. In most states, the association has at least one specialist school or class for children with autism and also provides services for children and adults with autism as well as provides information for members of the public, advocacy for the autism community, and parental support services.

Legal Issues, Mandates for Service

While there is no legislation specifically directed to people with autism, Australia has for many years had disability relevant legislation federally as well as in all states and territories. In general, most of this was enacted from 1993 following the United Nations Declaration on the Rights of Disabled Persons. In general, legislation either makes discrimination illegal or makes legal provision for supply of services to people with disability. In 2013, the Federal Government announced a National Disability Insurance Scheme (NDIS) to be developed in cooperation with state governments, to provide financial support to people with disability. This is being modeled on the federal scheme for financial support of people with autism and insurance schemes for those left with a disability following an accident.

Overview of Current Treatments and Centers

Today, each state and territory has their own systems for assessment and diagnosis of autism. In

some states, these systems still reside in mental health services, while in others, they are embedded in child health or disability services. Diagnostic practices within these services vary within and between states. A parallel private system for diagnosis and assessment, as described above, continues to operate, and assessment procedures vary between individual clinicians. In response to a need for quality and consistency, as well as recognition of the need for quality and consistency for other service providers, standards and a scheme for certification have been developed. In this scheme schools, clinics and individual practitioners may become certified as skilled practitioners or service providers in the field of autism. This system is similar to the British accreditation scheme for schools and centers in the UK but includes certification of individual practitioners and services for adults as well as of children's services, as in the UK. The Australian system was developed through the local state autism association in Victoria but is supported by the federal peak body for autism in Australia, the Australian Advisory Board on Autism Spectrum Disorders.

Early intervention services continue to be offered by state and federal governments and often operate as parallel services. Many autism associations also provide early intervention services for children and their parents/carers and educational services, either on a fee for service basis or supported by federal or state funding.

This has been identified as a problem by government, professionals, and consumers, and it is hoped that the new National Disability Insurance Scheme will streamline and improve coordination of care for early intervention.

The issue of *inclusion* continues. In some countries other than Australia (e.g., Britain), there are schools that cater specifically for children with autism and some of these extend to full secondary education. Children attending such schools might then have 12 years of school education with only others with autism as fellow students. In Australia, there are a number of special schools for children with autism. However, these are generally at primary level. Few children with autism would stay in an exclusively autism setting to school leaving age of 17–18. Inclusion

in regular schools has generally been actively promoted in Australia. However, the issue remains as to which setting is more effective for children with autism and few controlled studies have been carried out. Those that have been carried out, including the early studies (Bartak and Rutter 1973), have shown clear support for structured educational methods whatever the overall setting.

Awareness and consideration of the needs of adults with autism continues to develop gradually in Australia. This has been referred to already in connection with social skills programs. However, it has become apparent that a much wider range of services than those provided for children is necessary for adults. These will include work preparation and further education, health education and services, counseling and psychiatric services, aged care, housing and independent living options, leisure and recreational training, transport options and training, and forensic and judicial training and orientation.

Social Justice and Forensic Issues

Although there are few studies either in Australia or elsewhere (Bartak 2011; Cashin and Newman 2009; Newman and Ghaziuddin 2008; Mouridsen 2012), it is apparent that a number of adults with autism may be charged with a variety of offenses. In many instances, the offense will have occurred inadvertently and essentially through a lack of comprehension both on the part of the person with autism and through misunderstanding of autistic behavior by police and judicial staff. While the limited evidence suggests that people with autism spectrum disorders are not more likely to offend than others, they are overrepresented in the justice system. In part this may be owing to failure by family members and staff of community agencies (such as schools or medical facilities) to recognize autism in the respective individual. He or she may then inadvertently break the law for one or other of the above reasons. Provision in this area requires better training of police and judicial staff to increase their understanding of autism, and this is starting to occur in

Australia through joint seminars and other training programs with staff from autism associations, university departments, and members of police and justice departments.

Overview of Research Directions

Until recently, Australia had no systems in place or funding models to connect autism researchers beyond informal email networks. Over the last 5 years, the Australasian Society for Autism Research (ASfAR) has been established and now has over 180 members. This year the federal government funded an application for the formation of a Cooperative Research Center (CRC) for living with autism spectrum disorder. The federal government will provide over \$30 million for 8 years, with additional cash and in-kind contributions from participants and partners exceeding \$60 million. The CRC will support an “across the life-span” program of research. In parallel with other high-income countries, Australian scientists, researchers, and professionals with important skill sets for research in autism (e.g., geneticists, information technology experts, bioengineers, ethicists, and lawyers) have been forming transdisciplinary and intersectoral partnerships with autism experts to advance our knowledge of autism in a way that should lead to improved care and outcomes for those affected. Many universities have acknowledged the importance of autism and related disorders by establishing Chairs in autism and developmental medicine, or similar, and Research Institutes have developed relevant research group hubs. Increasingly, autism associations and the Australian autism peak body are linking directly with researchers.

See Also

- ▶ [Adulthood, Transition to](#)
- ▶ [Advocacy](#)
- ▶ [Affective Development](#)
- ▶ [Challenging Behavior](#)
- ▶ [Conduct Disorder](#)
- ▶ [Diagnosis and Classification](#)
- ▶ [Diagnostic Process](#)

- [Early Intervention](#)
- [Education](#)
- [Parent Training](#)
- [Relationship Development Intervention \(RDI\) Model](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders-fifth edition*. Washington, DC: American Psychiatric Publishing.
- Bartak, L. (2011). Educational approaches to the prevention of harmful and unlawful behaviour in later development. Paper presented at Asia Pacific Autism Conference, Perth, Sept 2011.
- Bartak, L., & Rutter, M. (1973). Special educational treatment of autistic children: A comparative study. 1. Design of study and characteristics of units. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, 14(3), 161–179.
- Bartak, L., Rutter, M., & Cox, A. (1975). A comparative study of infantile autism and specific developmental receptive language disorder. 1. The children. *British Journal of Psychiatry*, 126(2), 126–145.
- Bettelheim, B. (1967). *The empty fortress: Infantile autism and the birth of the self*. New York: Free Press.
- Cashin, A., & Newman, C. (2009). Autism in the criminal justice detention system: A review of the literature. *Journal of Forensic Nursing*, 5(2), 70–75.
- DeMeyer, M. K., Alpern, G. D., Barton, S., DeMeyer, W. E., Churchill, D. W., Hingten, J. N., Bryson, C. Q., Pontius, W., & Kimberlin, C. (1971). Imitation in autistic, early schizophrenic and non-psychotic sub-normal children. *Journal of Autism & Childhood Schizophrenia*, 1(3), 311–326.
- Ehlers, S., Gilberg, C., & Wing, L. (1999). A screening questionnaire for Asperger syndrome and other high functioning autisms in school age children. *Journal of Autism & Developmental Disorders*, 29(2), 129–141.
- Frith, U. (1989). *Autism: Explaining the enigma*. Wiley Online Library.
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. New York: Pergamon.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2001). *Autism diagnostic observation schedule*. Los Angeles: Western Psychological Services.
- Mouridsen, S. E. (2012). Current status of research on autism spectrum disorders and offending. *Research in Autism Spectrum Disorders*, 6(1), 79–86.
- Newman, S. S., & Ghaziuddin, M. (2008). Violent crime in Asperger syndrome: The role of psychiatric comorbidity. *Journal of Autism and Developmental Disorders*, 38(10), 1848–1852.
- Ornitz, E. M., & Ritvo, E. R. (1968). Perceptual inconstancy in early infantile autism. The syndrome of early infant autism and its variants including certain cases of childhood schizophrenia. *Archives of General Psychiatry*, 18(1), 76–98.
- Rimland, B. (1964). *Infantile autism: The syndrome and its implications for a neural theory of behaviour*. New York: Appleton-Century-Crofts.
- Rutter, M., & Schopler, E. (1987). Autism and pervasive developmental disorders: Concepts and diagnostic issues. *Journal of Autism and Developmental Disorders*, 17(2), 159–186.
- Rutter, M., Le Couteur, A., & Lord, C. (2003). *Autism diagnostic interview-revised*. Los Angeles: Western Psychological Services.
- Wing, L., Leekam, S., Libby, S., Gould, J., & Larcombe, M. (2002). Diagnostic interview for social and communication disorders: Background, inter-rater reliability and clinical use. *Journal of Child Psychology and Psychiatry*, 43(3), 307–325.

Australian Scale for Asperger's Syndrome

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Synonyms

[ASAS](#); [ASAS-R: Australian Scale for Asperger's Syndrome – Revised](#)

Description

The Australian Scale for Asperger's Syndrome (ASAS) is a rating scale that aims to assist in the identification of children likely to be at risk of the condition. The scale is based on formal diagnostic criteria, research literature on associated conditions and features, and extensive clinical experience of the authors (Garnett and Attwood 1998). While acknowledging the core sets of criteria developed by organizations, viz., American Psychiatric Association (APA) and the World Health Organization (DSM-IV and ICD-10, respectively), they adopted

the clinically derived diagnostic criteria of Gillberg and Gillberg. Attwood viewed these as “clear, concise and comprehensive” (Attwood 1998, p. 23). The ASAS has not been updated to align with the *Diagnostic and Statistical Manual of Mental Disorders* 5th ed. (DSM-5) (APA 2013) in which Asperger's disorder has been subsumed under the umbrella diagnosis autism spectrum disorder (ASD).

The rating scale is designed to be completed by parents, teachers, or other professionals who know the child. It is comprised of six sections, A–F, and covers behaviors and abilities consistent with a primary-school-age child with the former diagnosis of Asperger's syndrome. These include social and emotional issues, communication skills, cognitive skills, specific interests, movement skills, and a range of other characteristics such as sensory sensitivities, age of language development, and facial tics or twitches.

The scale consists of 24 questions, each with an example of the behavior or skill being determined. Responses are scored on a Likert scale from 0 to 6, with 0 indicating rarely and 6 frequently. Within this scale, 0 is deemed the usual level expected of a child of that age.

The final section (F) consists of a further 10 features or behaviors which the respondent completes as appropriate by ticking the box.

While no specific cutoff is noted, the authors suggest that when the majority of the questions are answered in the affirmative with scores between 2 and 6, a referral for full diagnostic assessment may be indicated. High scores do not, by definition, imply the condition. Full clinical assessment, if this were to be undertaken, would enable further examination of the six core areas rated in the scale.

The authors revised the scale for use with children and adolescents aged 5–18 years (ASAS-R, unpublished Ph.D. thesis, 2007). They advised general adherence to the interpretation of scores as per the original measure. No psychometric data are available at this time. Further work has focused on developing a measure that provides an autism symptomatology profile that in turn may be useful in clinical settings for targeting and measuring intervention impact (Garnett et al. 2013).

Historical Background

The ASAS was originally developed by Garnett as part of a master's thesis in 1993. The scale was further developed by Garnett and Attwood and presented at a conference in Australia in 1998. Only one other scale existed *specific* to the higher functioning end of the spectrum, viz., the Autism Spectrum Screening Questionnaire (ASSQ) developed by Ehlers and Gillberg in Sweden in 1993.

Owing to limited knowledge about Asperger's syndrome among professionals at this time, coupled with the sometimes subtle presentation of features, many children were not being identified as warranting referral to specialist assessment services. Developing screening measures was one way to assist in the identification of possible features and abilities consistent with the condition in order to make appropriate onward referrals. While screening measures, such as the ASAS, were overinclusive, they could facilitate the process whereby possible cases were not missed.

In the first instance, the ASAS was developed for use with primary school children. Garnett and Attwood (2006) revised the original scale and adapted it for use with 5- to 18-year-olds (ASAS-R). The ASAS-R was effectively a new measure of core *dimensions* of Asperger's syndrome. Garnett was especially interested in the association of Asperger/autism symptomatology (AAS) with the psychological health of children and adolescents who had an autism spectrum condition (Garnett 2007). She also sought to evaluate the association between family and peer relationships with psychological health of this group and the level of AAS in those with a diagnosis. Garnett conducted a validation study, the results of which were presented in an unpublished Ph.D. in 2007.

The ASAS has been translated into German, and this version has been validated on a German sample of 51 children (Melfsen et al. 2005). It has been used to examine sex differences in comorbidity and clinical presentation in a Polish sample of adolescents (Rynkiewicz and Lucka 2018).

Psychometric Data

Limited data exist on the psychometric properties of the original measure (Thabtah and Peebles 2019). Table 1 gives the areas evaluated by the ASAS.

In the original study (Garnett, unpublished master's thesis, 1993), ASAS ratings were provided for 60 children and adolescents (3–19 years). Each group consisted of 20 children: (1) diagnosed with AS, (2) clinical group with mixed diagnosis, and (3) typically developing controls. In addition, participants were assessed for level of receptive language.

Multivariate analysis of covariance (MANCOVA) was then conducted for each area score by diagnosis with the receptive language score as the covariate. The nonclinical and AS groups were found to be statistically significantly different in each area to <0.0001.

Discriminant function analysis revealed that the ASAS accurately predicted membership of the groups: 90% for AS, 65% for the mixed clinical group, and 100% for the control group.

No further data were available on test-retest reliability, internal consistency, or discriminant validity of the scale. Small sample sizes and no formal testing as a screening instrument were raised as weaknesses in the scale.

The ASAS was translated into German, and the scale was validated in a study by Melfsen et al. (2005). Mothers of children who were inpatients at a local psychiatric hospital were asked to participate. Three groups of children were rated on the ASAS (German version), viz., AS group (18), referred for assessment but not

diagnosed (18), and a group with mixed psychiatric conditions (15).

Melfsen et al. (2005) report ANOVA results which indicate that the scale successfully differentiated between the three groups. Further stepwise discriminant analysis indicated that group membership was accurately predicted (60.78%). On the basis of these findings, the authors concluded that the ASAS was a useful screening instrument for children with Asperger's syndrome.

The authors have conducted reliability and validity studies on the most recent version of the ASAS, but these are not yet available. Furthermore, these data effectively relate to a *different* measure with a *different purpose*. The proposed revised measure aimed to assess five dimensions of AS to provide information on severity in each dimension and would therefore be helpful to assist in guiding treatment postdiagnosis (Garnett, personal communication, 2011).

Since the initial development of the ASAS, there has been a significant increase in screening and diagnostic tools. Garnett et al. (2013) focused on developing a tool that would provide a profile of individual symptomatology of ASD, that is, The Australian Scale for Autism Spectrum Conditions (ASASC). The authors conducted preliminary psychometric evaluation of the measure and confirmed that the 5-subscale measure mapped reasonably well onto to the two domains of ASD in the DSM-5. While the measure did not aim to serve as a screening tool, the authors reported positive indications that the ASASC may have benefits of identifying autism in addition to the core purpose of (i) identifying an autism profile, including strengths and difficulties to target in intervention, and (ii) providing indicators of change during and following intervention. The authors suggest further replication and validation studies.

Clinical Uses

As per the current range of screening measures for children, adolescents, and adults, the ASAS continues to serve the clinical purpose of identifying

Australian Scale for Asperger's Syndrome, Table 1 Areas of behaviors and skills consistent with Asperger's syndrome (Attwood 1998)

Social and emotional abilities	Other characteristics
Communication skills	Sensory
Cognitive skills	Flapping/rocking
Specific interests	High pain threshold
Movement skills	Delayed speech
	Unusual facial expressions/tics

those children and adolescents who are most at risk of having an autism spectrum condition. It is not evident from the authors whether the ASAS has been replaced by the ASASC.

Not unlike *diagnostic* instruments, screening instruments may not identify those who appear to have more subtle difficulties and differences, such as girls, and those of very significant cognitive abilities. In addition, perhaps in a high-risk family, where others may be affected more significantly, the identified child's features or behaviors are not registered as severe, hence not deemed necessary to assess further.

While the ASAS was one of the earliest attempts at screening for Asperger's syndrome in children, an increasing range of screening tools is now available to identify possible ASD. These cover various age ranges, and while some, like the ASAS, may be completed by laypersons, others are clinician-rated scales. Nonetheless, in clinical practice, these are not infrequently employed alongside other measures, hence providing a wealth of information from a range of sources, that is, clinicians, parents, and teachers. With the shift to ASD as the umbrella diagnostic category, there has been an increase in tools that evaluate autistic profiles such as females versus males (e.g., GQ-ASQ).

- Autism Spectrum Quotient (AQ-child)
- Checklist for Autism in Toddlers (18–24 months)
- Child Behavior Checklist (36–71 months)
- Childhood Autism Spectrum Test (4–11 years)
- Developmental Behavior Checklist – Early Screen (20–51 months)
- Gilliam Autism Rating Scale Third Edition (36–71 months)
- Modified Checklist for Autism in Toddlers (17–48 months)
- Screening Tool for Autism in Toddlers (24–35 months)
- Social Communication Questionnaire (48 months; mental age > 24 months)

While a small validation study in Germany (Melfsen et al. 2005) has confirmed the ASAS's ability to differentiate between three clinical

groups, viz., children with a known diagnosis of AS, children referred for suspected AS, and those referred for other psychiatric conditions, little current information is available regarding the scale's current usefulness as a screening measure or how it compares with other screening measures. Furthermore, lack of clear cutoff scores or indication of the meaning of a particular score renders the instrument difficult to interpret in its current form. Since the scale does not require training or qualification and could hence be employed by clinicians and laypersons alike, the lack of clarity about screening scores and their meaning contributes to the difficulties with interpretation. Nonetheless, if employed as a simple guide prior to discussion about full screening and assessment, the brief, structured scale facilitates an initial evaluation of the child's behavior and presentation within clear areas consistent with the autism spectrum.

The ASAS has undergone significant revisions, and it would appear that the purpose of the various versions is necessarily different. The ASAS has *not* been replaced by the ASAS-R, and the former still serves as a screening instrument (Garnett, personal communication, 2011).

Garnett has suggested the potential value of the ASAS-R in providing directions for intervention. When used in conjunction with additional data regarding family cohesion and peer victimization, this may assist in selecting areas for treatment. However, she notes the need for further research to establish the tool's sensitivity to clinical change. The ASASC appears to be fulfill this objective.

See Also

- ▶ [Asperger Syndrome](#)
- ▶ [Autism Spectrum Disorder](#)
- ▶ [Autistic Disorder](#)
- ▶ [Checklist for Autism in Toddlers](#)
- ▶ [Child Behavior Checklist in Autism](#)
- ▶ [Modified Checklist for Autism in Toddlers \(M-CHAT\)](#)
- ▶ [Screening Measures](#)
- ▶ [Screening Tool for Autism in Toddlers](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Attwood, T. (1998). *Asperger's syndrome. A guide for parents and professionals*. London: Jessica Kingsley.
- Attwood, T. (2006). *The complete guide to Asperger's syndrome*. London: Jessica Kingsley.
- Attwood, T., Garnett, M. S., & Rynkiewicz, A. (2018). Profiling Autism Symptomatology: An Exploration of the Q-ASC Parental Report Scale in Capturing Sex Differences in Autism. In: Ormond S, Brownlow C, Garnett MS, Rynkiewicz A & Attwood T (eds.). *Journal of Autism and Developmental Disorders*, 48, 389–403.
- Auyeung, B., Baron-Cohen, S., Wheelwright, S., & Allison, C. (2008). The autism spectrum quotient: Children's version (AQ-Child). *Journal of Autism and Developmental Disorders*, 38(7), 1230–1240.
- Baron-Cohen, S., Hoekstra, R. A., Knickmeyer, R., & Wheelwright, S. (2006). The autism-spectrum quotient (AQ)-adolescent version. *Journal of Autism and Developmental Disorders*, 36(3), 343–350.
- Ehlers, S., & Gillberg, C. (1993). The epidemiology of Asperger's syndrome – A total population study. *Journal of Child Psychology and Psychiatry*, 34, 1327–1350.
- Ehlers, S., Gillberg, C., & Wing, L. (1999). A screening questionnaire for Asperger syndrome and other high-functioning autism spectrum disorders in school age children. *Journal of Autism and Developmental Disorders*, 29, 129–141.
- Garnett, M. S. (2007). *Children and adolescents with Asperger's syndrome: Validation of a new measure of symptomatology and a structural test of family and peer influences*. Ph.D. thesis, School of Psychology, University of Queensland.
- Garnett, M., & Attwood, T. (1998). The Australian Scale for Asperger's Syndrome. Paper presented at the 1995 Australian National Autism Conference, Brisbane, Australia.
- Garnett, M. S., Attwood, T., Peterson, C., & Kelly, A. B. (2013). Autism spectrum conditions among children and adolescents: A new profiling tool. *Australian Journal of Psychology*, 65(4), 206–213. <https://doi.org/10.1111/ajpy.12022>.
- Goldstein, S. (2002). Review of the Asperger's syndrome diagnostic scale. *Journal of Autism and Developmental Disorders*, 32, 611–614.
- Howlin, P. (2000). Assessment instruments for Asperger syndrome. *Child Psychology & Psychiatry Review*, 5(3), 120–129.
- Krug, D. A., Arick, J. R., & Almond, P. (1980). Behavior checklist for identifying severely handicapped individuals with high levels of autistic behavior. *Journal of Child Psychology and Psychiatry*, 21(3), 221–229.
- Melfsen, S., Walitza, S., Attwood, A., & Warnke, A. (2005). Validation of the German version of the Australian Scale of Asperger's Syndrome (ASAS). *Zeitschrift für Kinder- und Jugendpsychiatrie und Psychotherapie*, 33(1), 27–34.
- Rynkiewicz, A., & Lucka, I. (2018). Autism spectrum disorder (ASD) in girls. Co-occurring psychopathology. Sex differences in clinical manifestation. *Psychiatria Polska*. Online First. <https://doi.org/10.12740/PP/OnlineFirst/58837>.
- Scott, F., Baron-Cohen, S., Bolton, P., & Brayne, C. (2002). The CAST (Childhood Asperger Syndrome Test): Preliminary development of UK screen for mainstream primary-school children. *Autism*, 6(1), 9–31.
- Sumiła, A., Buczyński, J., & Burkiwicz, A. (2013). 1969 – Psychopathological representation and treatment in Asperger's syndrome in Polish out-patients. *European Psychiatry*, 28, 1. [https://doi.org/10.1016/s0924-9338\(13\)76909-8](https://doi.org/10.1016/s0924-9338(13)76909-8).
- Thabtah, F., & Peebles, D. (2019). Early autism screening: A comprehensive review. *International Journal of Environmental Research and Public Health*, 16, 3502.
- Woodbury-Smith, M., Robinson, J., Wheelwright, S., & Baron-Cohen, S. (2005). Screening adults for Asperger syndrome using the AQ: A preliminary study of its diagnostic validity in clinical practice. *Journal of Autism and Developmental Disorders*, 35(3), 331–335.

Autism

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Synonyms

Autistic

Definition

The term “autism” (or autistic) has had several uses in psychiatry. Originally introduced by Bleuler to describe self-centered thinking in schizophrenia, he modified the term from the Greek word for self. In the 1930s, the first child psychiatrist, Leo Kanner, became aware of a group of children who had unusual patterns of social engagement and learning. He published his first 11 cases in 1943 using the term “early

infantile autism” to emphasize the apparent congenital lack of social engagement which he believed to be one of the two cardinal features of the disorder (the other being insistence on sameness/resistance to change). Although children with features suggestive of autism had been described for centuries (likely including some feral children like Victor the Wild Boy in France), Kanner was the first to describe the syndrome in detail. Interestingly, independent of Kanner’s work, the Austrian medical student Hans Asperger also used the term in his description of a similar condition in highly verbal but socially isolated and eccentric boys.

Although he initially emphasized the unique aspects of the condition, Kanner’s use of the term also suggested a link to schizophrenia given Bleuler’s earlier use of the term, and indeed until 1980 autism was not recognized as an official diagnosis and children with what today would be said to have autism were instead thought to exhibit a form of childhood schizophrenia. By the late 1970s, this state of affairs changes with recognition of the uniqueness of autism (based on clinical features, onset, family history, neurobiological, and genetic findings) led to its explicit recognition as a category of disorder distinct from schizophrenia.

As time has gone on, the condition now known as Autism Spectrum Disorder has evolved in terms of the stringency of the specific diagnostic concept. At the same time a growing body of both behavioral and genetic work suggests that a broader spectrum of the condition clearly exists likely paralleling the awareness of the diverse number of genes that might contribute to the pathogenesis of the condition.

As work on the social neuroscience of autism has increased, the insight of Kanner in using this term to characterize the condition is increasingly appreciated.

See Also

- [Asperger, Hans](#)
- [Autistic Disorder](#)
- [Broader Autism Phenotype](#)

- [Kanner, Leo](#)
- [Neurodiversity](#)

References and Reading

- Asperger, H. (1944). Die “autistischen Psychopathen” im Kindersalter. *Archive für psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Bleuler, E. (1911). *Dementia praecox oder Gruppe der Schizophrenien* (J. Zinkin, Trans.). New York: International Universities Press.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kanner, L. (1973). The birth of early infantile autism. *Journal of Autism & Childhood Schizophrenia*, 3(2), 93–95.
- Rutter, M. (1972). Childhood schizophrenia reconsidered. *Journal of Autism & Childhood Schizophrenia*, 2(4), 315–337.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 5–41). Hoboken: Wiley.

Autism Acceptance and Mental Health

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Definition

Acceptance, appreciation, and acknowledgement of autism as part of a person, both in terms of self-acceptance and how accepting other people are. Perceiving a lack of acceptance could relate to poorer mental health for autistic people.

Historical Background

Historically, the narratives around autism often focus on deficits, perhaps stemming from its first conception where Kanner (1943) described “autistic disturbances” and autistic behaviors are

often pathologized as not “normal” (Milton and Bracher 2013). For many years, autism research has been dominated by biological studies (e.g., genetic and neuroscientific research) and treatment or intervention studies (Pellicano et al. 2014) which likely proliferates the narrative that autism is something which should be cured rather than accepted. In more recent years, the ethics behind such autism research has been questioned (Pellicano and Stears 2011), with growing acknowledgment of the need for a shift in the narrative (Gillespie-Lynch et al. 2017) as well as increased participatory research whereby autistic people are meaningfully included in autism research (Fletcher-Watson et al. 2019).

The shift in narrative and the idea of accepting autism as an integral part of a person is related to the concept of neurodiversity, a term first credited to Judy Singer (1999). The neurodiversity movement celebrates diverse autistic thinking, views autism as part of identity, and is opposed to autism “cures” (Kapp et al. 2013) while still acknowledging the difficulties autistic people face and not minimizing disability (Den Houting 2019). This movement relates to the social model of disability (Shakespeare 2006) which proposes that disability can be the result of incompatibility with the environment and society. For example, one could argue that society’s lack of acceptance of autism could be a significant factor in the underemployment of autistic people rather than autistic characteristics preventing employment. Generally, these approaches highlight that autism is not perceived to be currently accepted within society.

This poor acceptance could relate to the mental health of autistic people. Autism itself is not a mental health condition, but autistic individuals experience high rates of mental health difficulties, for example, depression (Stewart et al. 2006), anxiety (Gillott and Standen 2007), social anxiety (Maddox and White 2015), and suicidal behavior and ideation (Cassidy et al. 2014). Prevalence estimates vary widely, for example, between 28% and 86% of autistic people have been suggested to experience diagnosable mental health conditions (Howlin and Magiati 2017). Despite this variability, it is asserted that autistic people experience mental health difficulties at

a higher rate than non-autistic people (Lai et al. 2019). These mental health difficulties make everyday life more challenging for autistic people and likely contribute to poorer quality of life (Robertson 2009).

The reasons why mental health conditions are highly prevalent in autism are not well understood and may depend on the specific mental health condition. Further, it is unlikely that the high prevalence is caused by one single factor, but a myriad of both internal and external contributing factors which enhance the risk of poor mental health. Internal factors might include genetic predispositions or certain cognitive biases. For example, in anxiety in autism, a mode of thinking known as “intolerance of uncertainty” has been found to link to increasing anxiety (Boulter et al. 2014). External factors might include aspects of the world dependent on other people, such as lack of social support, or the sensory environment – for example, hypersensitivity to the sensory environment has been noted to play a role in heightening anxiety in autism (Green and Ben-Sasson 2010). More recently, the role of autism acceptance has been examined as an external contributing factor to poor mental health in autism.

Current Knowledge

Autistic adults tend to perceive that within society, they are not accepted for being autistic, and a lack of perceived acceptance from others relates to higher symptoms of depression and stress (Cage et al. 2018). Robertson et al. (2018) found that autistic adults experiencing high rates of anxiety discussed how acceptance from others helped mitigate some of their anxiety symptoms, but they continued to fear the judgment of others. Participants in Cage et al.’s (2018) study described how they were often misunderstood or even completely dismissed by others and they used tactics to hide their autistic characteristics and therefore “pass” within society unnoticed as an autistic person. These tactics have been conceptualized as “camouflaging” (Hull et al. 2017), and the use of camouflaging has been argued as a means of protecting the self against

nonacceptance and discrimination from others (Cage and Troxell-Whitman 2019). Further, high rates of camouflaging have been found to relate to poorer mental health (Hull et al. 2019). Trying to fit into a predominantly non-autistic and non-accepting society is thus a mentally exhausting endeavor.

Autism acceptance can also be measured by examining antonymous concepts such as autism-related stigma. Stigma is the attachment of negative attitudes and stereotypes toward a group of people, and the discreditation and discrimination of the group (Goffman 1990), in this case autistic people. Botha and Frost (2018) argue that autistic people are a stigmatized minority group and are subject to “minority stress” – in other words, the stress associated with being labeled as part of a discriminated minority. Botha and Frost (2018) supported this minority stress model by finding that experiences of discrimination, concealment of autistic status, and internalized stigma related to poorer mental health in a sample of autistic adults. Together, this research suggests that perceived lack of acceptance in the form of stigma can have a negative relationship with mental health.

Other research has examined the self-acceptance of autism, which can also be thought of in terms of autistic identity. The terminology used to describe autism can be classified as either person-first (person with autism) or identity-first (autistic person). Kenny et al. (2016) found in a sample of British autistic participants that the majority preferred identity-first terminology. The participants described how person-first terminology positioned autism as something separate to the self, when in fact autism colors every element of how the individual processes the world and was therefore an inseparable part of the person. Similarly, Kapp et al. (2013) found that stronger identification with an autistic identity was associated with viewing autism more positively and as something that should not be “cured.” Further, Kapp et al. (2013) noted that preference for identity-first language did not mean that these participants lessened the difficulties associated with autism. It is important to bear in mind that acceptance does not mean ignoring or reducing the everyday difficulties associated with autism,

but rather to understand and acknowledge these, alongside autistic strengths (Den Houting 2019; Kapp et al. 2013).

Research has attempted to examine the links between self-acceptance, in terms of identifying with an autistic identity, and mental health. Cooper et al. (2017) measured autistic social identity (i.e., identifying as part of the autistic community) and mental health. They found that autistic identification could protect against anxiety and depression through promoting higher personal self-esteem (the value placed on one’s self) and collective self-esteem (the value placed on autistic people as a group). In this way, identifying with the autistic community could protect against mental health problems through taking pride in being part of this community and potentially drawing on support from other autistic people. In a review and synthesis of qualitative literature, Kim (2019) noted links between positive autistic self-identity and greater self-determination (an attitude toward being in control of one’s own life), which they suggest could link to better quality of life and ability to manage stress. Although the above research suggests that acceptance of being autistic links to better mental health outcomes, it can be challenging to achieve self-acceptance, especially considering the discrimination and stigma experienced by autistic people.

As such, researchers have not only examined autistic people’s perceptions of acceptance but have tried to survey non-autistic people’s attitudes toward autism, to measure the levels of acceptance within society. Knowledge about autism in Western populations is thought to be relatively good (Tipton and Blacher 2014) although some misunderstandings and misconceptions can still occur (Dillenburger et al. 2015) as well as many believing myths about autism (John et al. 2018). Autism-related stigma has also been measured, for example, as “openness” toward autism (Gardiner and Iarocci 2014) or the desire for social distance from autistic people (Gillespie-Lynch et al. 2015), with these studies finding that less stigma is associated with prior experience and contact with autistic people themselves. Notably, non-autistic people can falsely believe that they

are more helpful toward autistic people than they actually are (Heasman and Gillespie 2019), suggesting that non-autistic people likely attempt to provide socially desirable responses that present themselves in a positive light – thus survey measures of stigma are considered with caution.

Experimental research has therefore looked at non-autistic people's acceptance of autism through inventive means which may be less affected by social desirability than survey methods. For example, a growing body of research looks at the "first impressions" of autistic people. First impressions are the rapid judgments made about a person on first interacting with them (Ambady et al. 2000). One approach to test these first impressions is to ask observers to rate people they view in brief video clips. In one of Sasson et al.'s (2017) experiments, it was unknown to the observers that half of the people in the videos were autistic. Sasson et al. (2017) found that more negative judgments of the autistic people were formed, with autistic people rated as more awkward, less likeable, and attractive than non-autistic people, and there was less desire to hang out, talk to, or make friends with the autistic individuals. Interestingly, Sasson and Morrison (2019) found that labeling the video participants as autistic helped to improve first impressions. Further, Morrison et al. (2019) found that personal characteristics of the observers, such as pre-existing stigmatizing attitudes and knowledge about autism, could better explain poor first impressions than the characteristics of the autistic people in the videos. Together, these findings suggest that non-autistic people have negative biases against autism, but with intervention non-autistic people could overcome their difficulties with accepting autistic people.

Future Directions

More research is needed to find means of improving autism acceptance in the non-autistic population. Interventions have been trialled within educational establishments – such as Gillespie-Lynch et al.'s (2015) online training for university

students. Here, the training taught students about a range of different aspects including autistic strengths and difficulties, autism as a spectrum condition, etiology, and neurodiversity. After completing the training, there were improvements in knowledge and reductions in stigma toward autism. These findings are thus promising, with subsequent replications in non-Western countries such as Lebanon (Obeid et al. 2015) and Japan (Someki et al. 2018). Other anti-stigma interventions with school-aged girls have also shown promise in teaching girls about autism, with associated reductions in stigma (Ranson and Byrne 2014). Often research finds that girls and women tend to be more open toward autism (e.g., Cage et al. 2019; Nevill and White 2011); thus future research needs to understand why this is the case and what can be done to improve male's attitudes toward autism. Further, more research is needed on interventions outside of educational settings as well as longitudinal studies to test whether the effects of interventions persist beyond the short term.

Additionally, the difficulties in autistic to non-autistic interactions can be thought of in terms of a "double empathy problem," which can be described as interactional clashing between autistic and non-autistic people, with neither group understanding one another well (Milton et al. 2018). Mitchell et al. (2019) argue that the double empathy problem should be considered in tandem with the mental health difficulties experienced by autistic people. It seems that autistic people expend significant effort to understand and fit in to the predominantly non-autistic world, and this can be mentally and physically exhausting (Cage and Troxell-Whitman 2019). Achieving acceptance may involve reducing the "gap" between autistic and non-autistic perspectives and non-autistics putting in equal effort to understand autistic perspectives. Research on the double empathy problem is in its infancy but shows promise (Milton et al. 2018).

It should also be carefully considered how current practices in education, the media, healthcare and research might be perpetuating stigma. For example, Bottema-Beutel et al. (2018) discuss how social skills training may

implicitly enforce stigma and the necessity of camouflaging, since this training suggests that autistic people need to conform to a non-autistic standard of social communication. Instead, they argue that autistic people need to be able to explore their authentic selves and critically appraise non-autistic social rules, but more research is needed in this area. Furthermore, the narratives perpetuated in the media about autism are also thought to enforce stigma (Holton et al. 2014). Health practitioners too – trained predominantly in terms of a medical or deficit model of autism – need to acknowledge their role in promoting autism acceptance in those just diagnosed as well as in families and caregivers (Nicolaidis 2012). Future research should examine the impact of training, particularly using a social model or neurodiversity approach, on reducing stigma within professionals who encounter autistic people in their work.

Listening to – and including – autistic perspectives within research could be vital in improving autism acceptance. Research which does meaningfully involve autistic people is growing, with several researchers (autistic and non-autistic) developing frameworks and guidelines for this type of research (Chown et al. 2017; Fletcher-Watson et al. 2019). For example, Chown et al. (2017) outline how research should be aligned with the priorities of autistic people themselves, that the social model of disability should be at the research's core and outcomes should center around improving the everyday lives of autistic people. Similarly, Fletcher-Watson et al. (2019) highlight the need for elements such as empathy, authenticity, and respect within autism research. Researchers should consider how these guidelines could be incorporated into their future work and how their research supports the aim of improving autistic people's lives and the inclusion of autistic people in society.

By striving to make the world a more accessible place for autistic people, we may see concurrent improvements in autistic people's mental health. Of course, mental health is affected by numerous factors, and more research is needed to fully understand mental health in autism and appropriate treatments to reduce these difficulties.

Ultimately, autistic people must be accepted for being autistic people, not as camouflaged versions of themselves.

See Also

- Double Empathy
- Mental Health and ASD
- Neurodiversity

References and Reading

- Ambady, N., Bernieri, F. J., & Richeson, J. A. (2000). Toward a histology of social behavior: Judgmental accuracy from thin slices of the behavioral stream. *Advances in Experimental Social Psychology*, 32, 201–271.
- Botha, M., & Frost, D. M. (2018). Extending the minority stress model to understand mental health problems experienced by the autistic population. *Society and Mental Health*. <https://doi.org/10.1177/2156869318804297>.
- Bottema-Beutel, K., Park, H., & Kim, S. Y. (2018). Commentary on social skills training curricula for individuals with ASD: Social interaction, authenticity, and stigma. *Journal of Autism and Developmental Disorders*, 48(3), 953–964.
- Boulter, C., Freeston, M., South, M., & Rodgers, J. (2014). Intolerance of uncertainty as a framework for understanding anxiety in children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(6), 1391–1402.
- Cage, E., & Troxell-Whitman, Z. (2019). Understanding the reasons, contexts and costs of camouflaging for autistic adults. *Journal of Autism and Developmental Disorders*, 49(5), 1899–1911.
- Cage, E., Di Monaco, J., & Newell, V. (2018). Experiences of autism acceptance and mental health in autistic adults. *Journal of Autism and Developmental Disorders*, 48(2), 473–484.
- Cage, E., Di Monaco, J., & Newell, V. (2019). Understanding, attitudes and dehumanisation towards autistic people. *Autism*, 23(6), 1373–1383.
- Cassidy, S., Bradley, P., Robinson, J., Allison, C., McHugh, M., & Baron-Cohen, S. (2014). Suicidal ideation and suicide plans or attempts in adults with Asperger's syndrome attending a specialist diagnostic clinic: A clinical cohort study. *The Lancet Psychiatry*, 1(2), 142–147.
- Chown, N., Robinson, J., Beardon, L., Downing, J., Hughes, L., Leatherland, J., ... MacGregor, D. (2017). Improving research about us, with us: A draft

- framework for inclusive autism research. *Disability & Society*, 32(5), 720–734.
- Cooper, K., Smith, L. G., & Russell, A. (2017). Social identity, self-esteem, and mental health in autism. *European Journal of Social Psychology*, 47(7), 844–854.
- Den Houting, J. (2019). Neurodiversity: An insider's perspective. *Autism*, 23(2), 271–273.
- Dillenburger, K., McKerr, L., & Jordan, J. A. (2015). Creating an inclusive society ... how close are we in relation to autism spectrum disorder? A General Population Survey. *Journal of Applied Research in Intellectual Disabilities*, 28(4), 330–340.
- Fletcher-Watson, S., Adams, J., Brook, K., Charman, T., Crane, L., Cusack, J., ... Pellicano, E. (2019). Making the future together: Shaping autism research through meaningful participation. *Autism*, 23(4), 943–953.
- Gardiner, E., & Iarocci, G. (2014). Students with autism spectrum disorder in the university context: Peer acceptance predicts intention to volunteer. *Journal of Autism and Developmental Disorders*, 44(5), 1008–1017.
- Gillespie-Lynch, K., Brooks, P. J., Someki, F., Obeid, R., Shane-Simpson, C., Kapp, S. K., ... Smith, D. S. (2015). Changing college students' conceptions of autism: An online training to increase knowledge and decrease stigma. *Journal of Autism and Developmental Disorders*, 45(8), 2553–2566.
- Gillespie-Lynch, K., Kapp, S. K., Brooks, P. J., Pickens, J., & Schwartzman, B. (2017). Whose expertise is it? Evidence for autistic adults as critical autism experts. *Frontiers in Psychology*, 8, 438.
- Gillott, A., & Standen, P. J. (2007). Levels of anxiety and sources of stress in adults with autism. *Journal of Intellectual Disabilities*, 11(4), 359–370.
- Goffman, E. (1990). *Stigma: Notes on the management of spoiled identity (3rd Edition)*. London: Penguin Books.
- Green, S. A., & Ben-Sasson, A. (2010). Anxiety disorders and sensory over-responsivity in children with autism spectrum disorders: Is there a causal relationship? *Journal of Autism and Developmental Disorders*, 40(12), 1495–1504.
- Heasman, B., & Gillespie, A. (2019). Participants overestimate how helpful they are in a two-player game scenario towards an artificial confederate that discloses a diagnosis of autism. *Frontiers in Psychology*, 10, 1349.
- Holton, A. E., Farrell, L. C., & Fudge, J. L. (2014). A threatening space? Stigmatization and the framing of autism in the news. *Communication Studies*, 65(2), 189–207.
- Howlin, P., & Magiati, I. (2017). Autism spectrum disorder: Outcomes in adulthood. *Current Opinion in Psychiatry*, 30(2), 69–76.
- Hull, L., Petrides, K. V., Allison, C., Smith, P., Baron-Cohen, S., Lai, M. C., & Mandy, W. (2017). "Putting on my best normal": Social camouflaging in adults with autism spectrum conditions. *Journal of Autism and Developmental Disorders*, 47(8), 2519–2534.
- Hull, L., Mandy, W., Lai, M. C., Baron-Cohen, S., Allison, C., Smith, P., & Petrides, K. V. (2019). Development and validation of the camouflaging autistic traits questionnaire (CAT-Q). *Journal of Autism and Developmental Disorders*, 49(3), 819–833.
- John, R. P., Knott, F. J., & Harvey, K. N. (2018). Myths about autism: An exploratory study using focus groups. *Autism*, 22(7), 845–854.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kapp, S. K., Gillespie-Lynch, K., Sherman, L. E., & Hutman, T. (2013). Deficit, difference, or both? Autism and neurodiversity. *Developmental Psychology*, 49(1), 59.
- Kenny, L., Hattersley, C., Molins, B., Buckley, C., Povey, C., & Pellicano, E. (2016). Which terms should be used to describe autism? Perspectives from the UK autism community. *Autism*, 20(4), 442–462.
- Kim, S. Y. (2019). The experiences of adults with autism spectrum disorder: Self-determination and quality of life. *Research in Autism Spectrum Disorders*, 60, 1–15.
- Lai, M. C., Kasse, C., Besney, R., Bonato, S., Hull, L., Mandy, W., ... Ameis, S. H. (2019). Prevalence of co-occurring mental health diagnoses in the autism population: A systematic review and meta-analysis. *The Lancet*. [https://doi.org/10.1016/S2215-0366\(19\)30289-5](https://doi.org/10.1016/S2215-0366(19)30289-5).
- Maddox, B. B., & White, S. W. (2015). Comorbid social anxiety disorder in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(12), 3949–3960.
- Milton, D. E., & Bracher, M. (2013). Autistics speak but are they heard. *Journal of the BSA Medsoc Group*, 7, 61–69.
- Milton, D. E. M., Heasman, B., & Sheppard, E. (2018). Double empathy. In F. Volkmar (Ed.), *Encyclopedia of autism spectrum disorders*. New York: Springer.
- Mitchell, P., Cassidy, S., & Sheppard, E. (2019). The double empathy problem, camouflage, and the value of expertise from experience. *Behavioral and Brain Sciences*, 42. <https://doi.org/10.1017/S0140525X18002212>.
- Morrison, K. E., DeBrabander, K. M., Faso, D. J., & Sasson, N. J. (2019). Variability in first impressions of autistic adults made by neurotypical raters is driven more by characteristics of the rater than by characteristics of autistic adults. *Autism*. <https://doi.org/10.1177/1362361318824104>.
- Nevill, R. E., & White, S. W. (2011). College students' openness toward autism spectrum disorders: Improving peer acceptance. *Journal of Autism and Developmental Disorders*, 41(12), 1619–1628.
- Nicolaidis, C. (2012). What can physicians learn from the neurodiversity movement? *AMA Journal of Ethics*, 14(6), 503–510.

- Obeid, R., Daou, N., DeNigris, D., Shane-Simpson, C., Brooks, P. J., & Gillespie-Lynch, K. (2015). A cross-cultural comparison of knowledge and stigma associated with autism spectrum disorder among college students in Lebanon and the United States. *Journal of Autism and Developmental Disorders*, 45(11), 3520–3536.
- Pellicano, E., & Stears, M. (2011). Bridging autism, science and society: Moving toward an ethically informed approach to autism research. *Autism Research*, 4(4), 271–282.
- Pellicano, E., Dinsmore, A., & Charman, T. (2014). What should autism research focus upon? Community views and priorities from the United Kingdom. *Autism*, 18(7), 756–770.
- Ranson, N. J., & Byrne, M. K. (2014). Promoting peer acceptance of females with higher-functioning autism in a mainstream education setting: A replication and extension of the effects of an autism anti-stigma program. *Journal of Autism and Developmental Disorders*, 44(11), 2778–2796.
- Robertson, S. M. (2009). Neurodiversity, quality of life, and autistic adults: Shifting research and professional focuses onto real-life challenges. *Disability Studies Quarterly*, 30(1). <https://dsq-sds.org/article/view/1069>.
- Robertson, A. E., Stanfield, A. C., Watt, J., Barry, F., Day, M., Cormack, M., & Melville, C. (2018). The experience and impact of anxiety in autistic adults: A thematic analysis. *Research in Autism Spectrum Disorders*, 46, 8–18.
- Sasson, N. J., & Morrison, K. E. (2019). First impressions of adults with autism improve with diagnostic disclosure and increased autism knowledge of peers. *Autism*, 23(1), 50–59.
- Sasson, N. J., Faso, D. J., Nugent, J., Lovell, S., Kennedy, D. P., & Grossman, R. B. (2017). Neurotypical peers are less willing to interact with those with autism based on thin slice judgments. *Scientific Reports*, 7, 40700.
- Shakespeare, T. (2006). The social model of disability. *The Disability Studies Reader*, 2, 197–204.
- Singer, J. (1999). Why can't you be normal for once in your life? From a 'problem with no name' to the emergence of a new category of difference. In M. Corker & S. French (Eds.), *Disability discourse* (pp. 59–67). Buckingham: Open University Press.
- Someki, F., Torii, M., Brooks, P. J., Koeda, T., & Gillespie-Lynch, K. (2018). Stigma associated with autism among college students in Japan and the United States: An online training study. *Research in Developmental Disabilities*, 76, 88–98.
- Stewart, M. E., Barnard, L., Pearson, J., Hasan, R., & O'Brien, G. (2006). Presentation of depression in autism and Asperger syndrome: A review. *Autism*, 10(1), 103–116.
- Tipton, L. A., & Blacher, J. (2014). Brief report: Autism awareness: Views from a campus community. *Journal of Autism and Developmental Disorders*, 44(2), 477–483.

Autism and Epilepsy

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Synonyms

Epilepsy and autism

Introduction

Autism spectrum disorders (ASD) and epilepsy are both clinically heterogeneous entities whose co-occurrence has long been recognized to exist at a frequency that is greater than could be predicted by chance alone. This observation has led to interest in the possibility of shared causal pathways for both disorders and raises the possibility of future novel interventions that could impact the course of both conditions. Further, the co-occurrence of the disorders presents several diagnostic and treatment challenges and controversies. This encyclopedia entry will provide an overview of terminology, epidemiology and etiology, clinical expression, available findings on developmental course, issues in differential diagnosis, and treatment considerations.

Categorization

Terminology

Terms associated with autism include autism spectrum disorder (ASD), high-functioning autism (HFA), pervasive developmental disorders (PDD), and infantile autism. ASD are behaviorally defined disorders, with literature still utilizing these terms despite the presentation of revised diagnostic criteria in the DSM-5. These terms are defined in introductory chapters in this encyclopedia. Key terms associated with epilepsy include seizure disorder and pediatric seizure disorder. A seizure is commonly understood as uncontrolled electrical activity in the brain, which may produce a physical convulsion, minor physical signs, changes in consciousness, thought disturbances, sensory disturbances, or a combination of symptoms. While the terms seizure disorder and epilepsy are generally used interchangeably, epilepsy is more formally defined as having two or more seizures within a set period of time, most often within 3 years, for which there is no other identifiable cause such as mass lesion, head trauma, infection, toxic exposure, or metabolic derangement (Matson and Neal 2009).

Epidemiology

Epidemiology and Etiology

The prevalence of autism in the pediatric population is approximately 14.6 cases per 1,000 children, with a national rate of approximately 1 in 68 cases at age 8 years. Estimated prevalence was significantly higher for boys at about 23.6 per 1,000, than for females at about 5.3 per 1,000 (Christensen et al. 2016). These rates are higher among non-Hispanic white children at about 15.5 per 1,000 than for non-Hispanic black children (13.2 per 1,000) and Hispanic children (10.1 per 1,000); however, it is suspected that these differences in rates are related more to access to care as opposed to true differences in prevalence across racial and ethnic lines (Durkin et al. 2010; Morbidity and Mortality Weekly Report 2012). Further, autistic traits are about 4.5 more commonly seen in males, with a staggering rate of

1 in 42, than for females (1 in 189) (Christensen et al. 2016).

In the general pediatric and adult population, the prevalence of epilepsy is 2–3% (Canitano 2007), with recent estimated lifetime rates of about 10.2 per 1,000 (Russ et al. 2012). The prevalence of having epilepsy and autism as co-occurring, or perhaps comorbid conditions ranges widely from 8% to 30% (Mouridsen et al. 2013; Russ et al. 2012; Spence and Schneider 2009; Tuchman et al. 2010), with children with ASD having a 7- to 10-fold increased odds compared to controls for having epilepsy (Jokiranta et al. 2014; Tuchman et al. 2013). Viscidi et al. (2013) found that the prevalence of epilepsy in children with ASD was about 12–13% for children ages 2–17, with rates rising to about 26% for adolescents age 13 and above. For children with epilepsy, about 30% will eventually have a diagnosis of ASD (Keller et al. 2017), and individuals with epilepsy diagnosed in childhood are at significant risk for later manifestation of ASD (Sundelin et al. 2016). While there does not appear to be a specific form of epilepsy that is present in children with ASD, epilepsy does appear to be present more frequently in individuals with ASD and Intellectual Disability, with the rates increasing with the severity of the Intellectual Disability (Jokiranta et al. 2014). Of significant concern, there also is increased risk for mortality for individuals with ASD and epilepsy, particularly in the presence of an intellectual disability (Woolfenden et al. 2012).

Etiological Mechanisms

The frequency of co-occurrence of these two disorders has led to interest in the possibility of shared etiological mechanisms in seizure disorders and ASD. Proposed theories of shared causality have been related to the deleterious effects of the seizures themselves and associated imbalances between neuronal excitation and inhibition (Stafstrom and Benke 2015), with a particular focus on impaired GABAergic signaling as being a common denominator for co-occurring ASD and epilepsy (Kang and Barnes 2013; Tuchman et al. 2013). Additionally, contemporary mechanistic understandings of several

key neurodevelopmental disorders have led to new theories about the shared role of impaired plasticity during development (Keller et al. 2017).

For example, portions of the temporal lobe of the brain and associated neural pathways are likely to be key brain regions in the complex network that has been described as “the social brain.” The temporal lobe has long been a suspected region of importance because of the relative frequency of temporal lobe epilepsy both among patients with epilepsy with social challenges and among those with ASD and epilepsy. Animal research using mouse models has demonstrated that mice with induced temporal lobe seizures exhibited less social behavior than control mice (Marin et al. 2008). Neuroimaging studies in patients with temporal lobe epilepsy have provided evidence showing damage to other recognized social brain structures in this network, such as the hippocampus (Dager et al. 2007). Further, such studies also have begun to show a linkage between aberrant neural migration over the course of neurodevelopment (Blackmon 2015) and general neurological vulnerability (Gilby and O’Brien 2013) to seizures in individuals with ASD.

Another set of examples wherein a potential shared mechanism for both ASD and seizure disorders has begun to be explored comes from the study of several recognized genetic syndromes that are associated with both autism features and seizures (Keller et al. 2017; Lee et al. 2015). In this regard, fragile X, tuberous sclerosis complex, and Rett’s syndrome all have been proposed as possible models of overlapping causality in ASD and epilepsy/seizures. For example, Rett’s syndrome is a neurodegenerative disorder that affects girls and is currently understood to be caused by mutations in the gene encoding methyl-CpG binding protein 2 (MeCP2). Rett’s syndrome is characterized by regression of verbal skills along with repetitive hand motions that usually begin to occur between 6 and 18 months of age (Brooks-Kayal 2010). Up to 90% of Rett’s syndrome patients develop seizures (Canitano 2007). Tuberous sclerosis has been associated with both epilepsy and autism (Jeste et al. 2016). The prevalence of tuberous sclerosis in the general population is around 1 case per 10,000 to

20,000 (Sherpherd 1999). Around 1% of children with autism will have tuberous sclerosis (Harrison and Bolton 1997), and approximately 80% of patients with tuberous sclerosis will also have seizures (Canitano 2007). With respect to epilepsy, tubers are thought to be foci of epileptic activity, and many of the ASD symptoms have been linked to tubers found in the temporal lobes of the brain (Bolton et al. 2002). Finally, fragile X syndrome is the most common form of inheritable intellectual disability, and frequently manifests with co-occurring autism and seizures. This syndrome is caused by excessive CGG trinucleotide repeats on the X chromosome, methylating either in whole or in part the Fragile X Mental Retardation gene leading to many of the phenotypic features associated with fragile X syndrome (Brooks-Kayal 2010). With approximately one third of individuals with fragile X syndrome showing co-occurring ASD, this syndrome provides a clear single gene disorder for examining not just autism but its related comorbidity.

While exact mechanisms for the behavioral manifestations remain unknown in each of these disorders, there has been an expanding knowledge base relating to presumed causal genetic defect (s) and their downstream molecular effects. Resultant impaired inhibitory/excitatory regulation and impaired neuroplasticity have been proposed as a possible common explanation for seizures and ASD-related behaviors (Brooks-Kayal 2010). Further, a number of other gene mutations have been associated with ASD, Intellectual Disabilities, and epilepsy/seizures including the genes encoding neuroligins, neurexins, arestelles region X-linked (ARX), and neuropilin-2 (Brooks-Kayal 2010).

Clinical Expression and Pathophysiology

There are a number of ways that the co-occurrence of seizures and ASD can be examined in terms of clinical expression and variables associated with seizure pathophysiology. These include: type of seizures, seizure location, epilepsy syndromes, age of seizure onset, level of intellectual functioning, and developmental course.

Type of Seizures

There are several classification schemas for seizure types and epilepsy. The most commonly used classification is based on the broad categories of generalized seizure onset versus focal onset, each with subcategorizations based on various clinical features and origin of seizure activity. Further, there are numerous recognized epilepsy syndromes. Seizure types in individuals with ASD are highly variable, and multiple seizure types in the same individual are not uncommon. It is important to note, though, that the prevalence of particular seizure types among those with both disorders does not seem to differ significantly from the distribution of seizure types in epilepsy patients in general (Sternberg 2003).

Seizure Location

There is a suggestion that seizure location may point to a relationship with autistic features or autism. In epilepsy in which the seizure activity manifests from the frontal lobe, behavioral changes may include irritability, altered mood, subtle changes in alertness, associated attention dysregulation, and cognitive rigidity features often associated with ASD (Fohlen et al. 2004). Seizures originating in the temporal lobe may be associated with autistic features or autism (Hamiwka and Wirrell 2009) in that the individual may present with affective blunting, odd or impaired language functions, including impairments in core language functions or pragmatics, and poor recognition of faces.

Epilepsy Syndromes

The relationship between ASD and seizures also can be understood by considering the presence of an epilepsy syndrome. There are numerous epilepsy syndromes, and those that are believed to contribute to progressive disturbance in cerebral function may be termed “epileptic encephalopathies.” These disorders begin early in life and are often associated with regression of cognitive, language, and other neurodevelopmental functions. Many children with these disorders may present with features of ASD or they may in fact meet diagnostic criteria for an ASD (Nabbout and Dulac 2003; Nabbout and Dulac 2008). Among these syndromes, infantile spasms (IS), Landau-

Kleffner syndrome (LKS), and epilepsy with continuous spike-waves during slow-wave sleep (CSWS) are most strongly associated with ASD symptomology (Ballaban-Gil and Tuchman 2000).

In IS, the association with ASD may as high as 35%, and this risk seems to increase in the presence of a severe intellectual disability, structural brain lesions, and ongoing epileptiform activity in frontal brain regions (Kayaalp et al. 2007; Saemundsen et al. 2007, 2008). LKS and CSWS have overlapping symptoms in relationship to seizure presentation, and both manifest features that overlap with ASD symptoms. The failure or regression of language development in these disorders often leads to confusion with autistic regression that is reported in children with and without underlying seizure disorders (Canitano 2007).

Age of Seizure Onset

The relationship between ASD and seizures/epilepsy can also be investigated by considering the age of seizure onset. It has been theorized that epilepsy with a late onset during adolescence is brought on by the hormonal fluctuations associated with puberty (Gillberg 1991). One study of children with autism showed that seizure activity peaks between 3 and 10 years of age (Matson and Neal 2009). Other studies, however, have suggested that epilepsy has two peaks in children with autism: one during infancy and another during adolescence (Volkmar and Nelson 1990). The peak during infancy may correlate with the peak of seizure activity that is seen in children with epilepsy without autism, while the second peak during adolescence may be unique to children with autism (Nomura et al. 2010). Recent data have challenged this bimodal distribution, suggesting that the primary peak occurs by 6 years of age (Jokiranta et al. 2014).

Intellectual Functioning

The range of the overall level of intellectual functioning in individuals with ASD is quite large and variable; however, it has been well established that in populations of children with epilepsy, the risk of autism or autistic features is increased among those with the lowest intellectual functioning (Hamiwka and Wirrell 2009; Jokiranta et al. 2014). Among populations of children with ASD,

those with severe intellectual disability, severe receptive language deficits and motor dysfunction (i.e., those with more severe autism symptoms) have the highest risk of epilepsy (El Achkar and Spence 2015; Mulligan and Trauner 2014; Tuchman et al. 2009); and, conversely, children with ASD and epilepsy manifest more cognitive and neuropsychiatric difficulties than those without epilepsy (Viscidi et al. 2014; Weber and Gadow 2017).

Developmental Course

When considered independently, the developmental course, severity, and outcomes of individuals with ASD and epilepsy are highly variable and dependent on numerous factors. To date, there are scant empirical data related to the moderating or mediating effects of epilepsy and ASD on one another in relation to developmental course and outcomes. In general, children with comorbid or co-occurring ASD and seizures/epilepsy have lower IQ, lower adaptive behavior, more emotional problems, and have more frequent use of psychiatric medications (Matson and Neal 2009). Also, a higher rate of seizure activity has been linked to decreased intellectual functioning (Jokiranta et al. 2014; Matson and Neal 2009) but is unclear how medications or other factors (e.g., other neurological factors) may be contributing to this suspected association. Additionally, the presence of temporal lobe seizures has been described as a poor prognostic indicator in relation to social adaptation among individuals with ASD and seizure disorders (Matson and Neal 2009). As noted above, the notion that children with comorbid ASD and seizure disorders have more pronounced social impairment when compared to children with ASD who do not have seizures has been proposed, but this issue is only beginning to be evaluated (Tuchman 2013).

Evaluation and Differential Diagnosis

Issues in Differential Diagnosis

Early diagnosis and treatment of both epilepsy and autism are crucial in order to maximize development and quality of life (Tuchman et al. 2010). Early identification and treatment allow for

the optimal usage of all therapies and resources available. The potential co-occurrence of these disorders does raise several important issues in differential diagnosis. For example, the mainstay of evaluation in seizure disorders is the electroencephalogram (EEG), but a seizure evaluation also can include metabolic and genetic components. It is important to note that abnormal EEG activity can be seen in 7–28% of children with autism, but without any other symptoms of epilepsy (Youroukos 2007). On the other hand, high-functioning individuals with autism may be missed when presenting for epilepsy treatment (Matsuo et al. 2010). The association between autism and seizures has led the Committee on Children with Disabilities of the American Academy of Pediatrics to recommend prolonged sleep-deprived EEG in children with ASD showing developmental regression or in those where there is a high suspicion of subclinical seizures (American Academy of Pediatrics 2001). Due to the current dearth of empirical knowledge about subclinical epileptiform activity and its treatment, universal screening via EEG for all children with ASD has not yet been recommended as a standard of care (Johnson and Myers 2007), but its routine use has been suggested (Swatzyna et al. 2017).

Another important area of concern relates to the convergence of sleep problems in the populations of children with ASD and epilepsy/seizures. Sleep difficulties are common among individuals with neurologic disorders in general as well as in those with ASD and seizure disorders (Malow 2004). Screening for sleep problems and formal sleep evaluations (based on clinical need) are often important for individuals presenting with comorbid ASD and epilepsy (Accardo and Malow 2017). Sleep disorders have significant implications for behavioral functioning and quality of life beyond challenges associated with the underlying disorder (Clarke et al. 2005), such as creating daytime sleepiness, increased irritability, less efficient cognitive functioning (potentially in addition to cognitive impairment), and decreased seizure threshold. Further, sleep studies in children with ASD and sleep problems in rare instances may elucidate a previously unrecognized seizure disorder related to sleep (Accardo and Malow 2017; Malow 2004).

Treatment Considerations

Early recognition of ASD and co-occurring epilepsy is important in that it is hoped that developmental outcomes can be improved via early treatment. Medication is a first-line treatment in children with epilepsy. The chief goal here is to eliminate (or lessen) all seizure activity while minimizing medication-related side effects such as behavioral problems or weight gain. In autism, psychosocial and behavioral interventions are commonly used as first-line interventions for behavioral symptoms. In autism, medication treatment is used as an adjunctive therapy to lessen symptoms of inattention, hyperactivity, repetitive behaviors, impulsivity, irritability, and aggression (Tuchman et al. 2010).

Antiepileptic medications (AEDs) are the mainstay of treatment in epilepsy. Several AEDs are used commonly in general psychiatric practice due to beneficial psychotropic properties, most notably in mood stabilization and the mitigation of aggression. Examples include valproic acid, carbamazepine, lamotrigine, and levetiracetam. While a full discussion of this class of medication is beyond the scope of this chapter, the aforementioned AEDs have been evaluated in the ASD population with and without epilepsy in several case series or small open-label trials. At present, AEDs seemed to have had equivocal results in terms of benefit with irritability, aggression, or behaviors associated with the core features of autism such as repetitive behaviors (Hirota et al. 2014; Tuchman et al. 2010), and concerns always are present for the medications to create affective blunting and/or to negatively impact cognitive and social capabilities. Formal evaluation via large randomized clinical trials in the ASD population with seizures is lacking and will be an important future step in guiding the care of this population (Tuchman et al. 2010).

Epileptic encephalopathies associated with ASD, such as infantile spasms (IS) or Landau-Kleffner syndrome (LKS), are treated early and aggressively with AEDs, adrenocorticotropin hormone (ACTH), steroids, the ketogenic diet, or surgery. The main focus of these interventions is to improve seizure control. Outcomes of these

practices as they relate to mitigation or prevention of ASD features are unknown (Crumrine 2002; Kosso et al. 2005; Trevathan 2002; Wheless 2004).

The treatment of epileptiform activity on EEG, without the presence of clinical seizures, is an area of considerable debate. This debate is most relevant among those with ASD showing cognitive regression, but without a clear epilepsy syndrome or epileptic encephalopathy. Approximately 30% of children with ASD present autistic regression, which is understood as a loss of verbal and nonverbal communication skills between approximately 12 and 24 months of age. The relationship between regression and epileptiform activity noted in this subgroup has been postulated, but remains unclear, and treatment recommendations for this subgroup remain without a clear evidence base (Baird et al. 2006; Venkateswaran and Shevell 2008).

New information about shared genetic and molecular causal pathways may provide new insights about the management of children with epilepsy and autism. For example, in fragile X syndrome, mouse models have provided evidence that FMRP dysfunction may lead to behavioral and cognitive deficits as well as seizure formation (Brooks-Kayal 2010; Penagarikano et al. 2007). A key target in this dysregulation may be the metabotropic glutamate receptor (MgluR). Modulation of MgluR in mouse models has provided promising results in terms of behavior, cognition, and seizure formation (Brooks-Kayal 2010). Several molecules that modulate the function of this receptor are currently in various phases of development. Their role in epilepsy treatment and treatment of any ASD feature remains to be seen, but it is clear that much is to be learned from conditions where ASD and epilepsy coexist (Brooks-Kayal 2010).

Conclusion

This encyclopedia entry provided an overview of the interesting association between autism and seizures disorders. This is an intriguing area for clinical inquiry, but it is also an area ripe for

scientific investigation. With the prevalence of seizure disorders in the general population being approximately 2–3%, the rate of seizures in the population of individuals with autism is arguably as high as 22 times as much, with about one third experiencing at least one seizure by adolescence. With the recently documented prevalence of autism in the population, this combines to create a significantly large number of individuals with comorbid ASD and seizures. As a subgroup of individuals with ASD, however, this area has only begun to receive scientific scrutiny. Increased understanding of the type of seizures, identifiable neurological contributors, other associated conditions, and developmental course all should contribute to improved seizure management. Key to this understanding is early, comprehensive evaluation and associated differential diagnosis. Also, recognizing that at least one-third will manifest a seizure by adolescence implicates the need for routine and thorough developmental surveillance for seizure manifestations by an interdisciplinary group of trained professionals (Eom et al. 2014). Ultimately, coordinated multimodal treatment approaches will be critical to maintaining a good quality of life for individuals with ASD and comorbid seizure disorders. Although treatment of epilepsy is a medical necessity, it typically will not be enough to address the additional symptoms related to ASD and related social and cognitive functioning (Tuchman 2013).

References and Reading

- Accardo, J. A., & Malow, B. A. (2017). Sleep, epilepsy, and autism. *Epilepsy & Behavior*, 47, 202–206.
- American Academy of Pediatrics Committee on Children with Disabilities. (2001). Technical report: The pediatrician's role in the diagnosis and management of autism spectrum disorders in children. *Pediatrics*, 107, 2–18.
- Baird, G., Robinson, R., Boyd, S., & Charman, T. (2006). Sleep electroencephalograms in young children with autism with and without regression. *Developmental Medicine and Child Neurology*, 48, 604–608.
- Ballaban-Gil, K., & Tuchman, R. (2000). Epilepsy and epileptiform EEG: Association with autism and language disorders. *Mental Retardation and Developmental Disabilities Research Reviews*, 6, 300–308.
- Blackmon, K. (2015). Structural MRI biomarkers of shared pathogenesis in autism spectrum disorder and epilepsy. *Epilepsy & Behavior*, 47, 172–182.
- Bolton, P., Park, R., Higgins, N., Griffiths, P., & Pickles, A. (2002). Neuro-epileptic determinants of autism spectrum disorders in tuberous sclerosis complex. *Brain*, 125, 1247–1255.
- Brooks-Kayal, A. (2010). Epilepsy and autism spectrum disorders: Are there common developmental mechanisms? *Brain & Development*, 32, 731–738.
- Canitano, R. (2007). Epilepsy in autism spectrum disorders. *European Child & Adolescent Psychiatry*, 16, 61–66.
- Center for Disease Control. (2012). Prevalence of autism spectrum disorders-autism and developmental disabilities monitoring network, United States. *Morbidity and Mortality Weekly Report*, 61(SS03), 1–19.
- Christensen, D. L., Baio, J., Van Naarden Braun, K., Bilder, D., Charles, J., Constantino, J. N., Daniels, J., Durkin, M. S., Fitzgerald, R. T., Kurzius-Spencer, M., Lee, L., Pettygrove, S., Robinson, C., Schulz, E., Wells, C., Wingate, M. S., Zahorodny, W., & Yeargin-Allsopp, M. (2016). Prevalence and characteristics of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2012. *Morbidity and Mortality Weekly Report. Surveillance Summaries*, 65, 1–23.
- Clarke, D., Roberts, W., Daraksan, M., Dupuis, A., McCabe, J., Wood, H., et al. (2005). The prevalence of autistic spectrum disorder in children surveyed in a tertiary care epilepsy clinic. *Epilepsia*, 46, 1970–1977.
- Crumrine, P. (2002). Lennox-Gastaut syndrome. *Journal of Child Neurology*, 17(Suppl. 1), S70–S75.
- Dager, S., Wang, L., Friedman, S., Shaw, D., Constantino, J., Artru, A., et al. (2007). Shape mapping of the hippocampus in young children with autism spectrum disorder. *American Journal of Neuroradiology*, 28, 672–677.
- Durkin, M. S., Maenner, M. J., Meaney, F. J., Levy, S. E., DiGuseppi, C., Nicholas, J. S., Kirby, R. S., Pinto-Martin, J. A., & Schieve, L. A. (2010). Socioeconomic inequality in the prevalence of autism spectrum disorder: Evidence from a U.S. cross-sectional study. *PLoS One*, 5, e11551.
- El Achkar, C. M., & Spence, S. J. (2015). Clinical characteristics of children and young adults with co-occurring autism spectrum disorder and epilepsy. *Epilepsy & Behavior*, 47, 183–190.
- Eom, S., Fisher, B., Dezort, C., & Berg, A. T. (2014). Routine developmental, autism, behavioral, and psychological screening in epilepsy care settings. *Developmental Medicine and Child Neurology*, 56, 1100–1105.
- Fohlen, M., Bulteau, C., Jalin, C., Jambaque, I., & Delalande, O. (2004). Behavioral epileptic seizures: A clinical and Intracranial EEG study in 8 children with frontal lobe epilepsy. *Neuropediatrics*, 35, 336–345.

- Gabis, L., Pomeroy, J., & Andriola, M. (2005). Autism and epilepsy: Cause, consequence, comorbidity, or coincidence? *Epilepsy & Behavior*, 7, 652–656.
- Gilby, K. L., & O'Brien, T. J. (2013). Epilepsy, autism, and neurodevelopment: Kindling a shared vulnerability? *Epilepsy & Behavior*, 26, 370–374.
- Gillberg, C. (1991). The treatment of epilepsy in autism. *Journal of Autism and Developmental Disorders*, 22, 61–77.
- Hamiwka, L., & Wirrell, E. (2009). Comorbidities in pediatric epilepsy: Beyond “Just” treating the seizures. *Journal of Child Neurology*, 24, 732–742.
- Harrison, J., & Bolton, P. (1997). Annotation: Tuberous sclerosis. *Journal of Child Psychology and Psychiatry*, 38, 603–614. [Review].
- Hirota, T., Veenstra-VanderWeele, J., Hollander, E., & Kishi, T. (2014). Antiepileptic medications in autism spectrum disorder: A systematic review and meta-analysis. *Journal of Autism and Developmental Disorders*, 44, 948–957.
- Jeste, S. S., Varcin, K. J., Hellemann, G. S., Gulsrud, A. C., Bhatt, R., Kasari, C., Wu, J. Y., Sahin, M., & Nelson, C. A. (2016). Symptom profiles of autism spectrum disorder in tuberous sclerosis complex. *Neurology*, 87, 766–772.
- Johnson, C. P., & Myers, S. M. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120, 1183–1215.
- Jokiranta, E., Sourander, A., Suominen, A., Timonen-Soivio, L., Brown, A. S., & Sillanpää, M. (2014). Epilepsy among children and adolescents with autism spectrum disorders: A population-based study. *Journal of Autism and Developmental Disorders*, 44, 2547–2557.
- Kang, J.-Q., & Barnes, G. (2013). A common susceptibility factor of both autism and epilepsy: Function deficiency of GABA_A receptors. *Journal of Autism and Developmental Disorders*, 43, 68–79.
- Kayaalp, L., Dervent, A., Saltik, S., Uluduz, D., Kayaalp, I., Demirbik, V., et al. (2007). EEG abnormalities in West syndrome: Correlation with the emergence of autistic features. *Brain & Development*, 29, 336–345.
- Keller, R., Basta, R., Salerno, L., & Elia, M. (2017). Autism, epilepsy, and synaptopathies: A not rare association. *Neurological Science*, 38, 1353–1361.
- Kosso, E., Thiele, E., Pfeifer, H., McGrogan, J., & Freeman, J. (2005). Tuberous sclerosis complex and the ketogenic diet. *Epilepsia*, 46, 1684–1686.
- Lee, B. H., Smith, T., & Paciorkowski, A. R. (2015). Autism spectrum disorder and epilepsy: Disorders with a shared biology. *Epilepsy & Behavior*, 47, 191–201.
- Malow, B. (2004). Sleep disorders, epilepsy, and autism. *Mental Retardation and Developmental Disabilities Research Reviews*, 10, 122–125.
- Marin, J., Moura, P., Cysneiros, R., Colugnati, D., Cavalheiro, E., Scorza, F., et al. (2008). Temporal lobe epilepsy and social behavior: An animal model for autism? *Epilepsy & Behavior*, 13, 43–46.
- Matson, J., & Neal, D. (2009). Seizures and epilepsy and their relationship to autism spectrum disorders. *Research in Autism Spectrum Disorders*, 3, 999–1005.
- Matsuo, M., Maeda, T., Sasaki, K., Ishii, K., & Hamasaki, Y. (2010). Frequent association of autism spectrum disorder in patients with childhood onset epilepsy. *Brain & Development*, 32, 759–763.
- Morbidity and Mortality Weekly Report Surveillance Summary. (2012). Prevalence of autism spectrum disorders. II. Autism and developmental disabilities monitoring network, 14 sites, United States, 2008. *Morbidity and Mortality Weekly Report Surveillance Summaries*, 61, 1–19.
- Mouridsen, S. E., Rich, B., & Isager, T. (2013). Epilepsy in individuals with a history of Asperger Syndrome: A Danish nationwide register-based cohort study. *Journal of Autism and Developmental Disorders*, 43, 1308–1313.
- Mulligan, C. K., & Trauner, D. A. (2014). Incidence and behavioral correlates of epileptiform abnormalities in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44, 452–458.
- Nabbout, R., & Dulac, O. (2003). Epileptic encephalopathies: A brief overview. *Journal of Clinical Neurophysiology*, 20, 393–397.
- Nabbout, R., & Dulac, O. (2008). Epileptic syndromes in infancy and childhood. *Current Opinion in Neurology*, 21, 161–166.
- Nomura, Y., Nagao, Y., Kimura, K., Hachimori, K., & Segawa, M. (2010). Epilepsy in autism: A pathophysiological consideration. *Brain & Development*, 32, 799–804.
- Penagarikano, O., Mülle, J., & Warren, S. (2007). The pathophysiology of fragile x syndrome. *Annual Review of Genomics and Human Genetics*, 8, 109–129.
- Russ, S. A., Larson, K., & Halfon, N. (2012). A national profile of childhood epilepsy and seizure disorder. *Pediatrics*, 129, 256–264.
- Saemundsen, E., Ludvigsson, P., & Rafnsson, V. (2007). Autism spectrum disorders in children with a history of infantile spasms: A population-based study. *Journal of Child Neurology*, 22, 1102–1107.
- Saemundsen, E., Ludvigsson, P., & Rafnsson, V. (2008). Risk of autism spectrum disorders after infantile spasms: A population-based study nested in a cohort with seizures in the first year of life. *Epilepsia*, 49, 1865–1870.
- Sherpherd, C. (1999). The epidemiology of the tuberous sclerosis complex. In M. Gomez, J. Sampson, & V. Whittemore (Eds.), *Tuberous sclerosis complex* (3rd ed., pp. 24–36). New York: Oxford University Press.
- Spence, S., & Schneider, M. (2009). The role of epilepsy and epileptiform EEGs in autism spectrum disorders. *Pediatric Research*, 65, 599–606.
- Stafstrom, C. E., & Benke, T. A. (2015). Autism and epilepsy: Exploring the relationship using experimental models. *Epilepsy Currents*, 15, 206–210.

- Sternberg, B. S. (2003). Autism. In M. Harris & E. Thackeray (Eds.), *The Gale encyclopedia of mental disorders* (Vol. 1, pp. 97–102). Gale: Detroit.
- Sundelin, H. E. K., Larsson, H., Lichtenstein, P., Almqvist, C., Hultman, C. M., Tomson, T., & Ludvigsson, J. F. (2016). Autism and epilepsy. A population-based nationwide cohort study. *Neurology*, 87, 192–197.
- Swatzyna, R. J., Tarnow, J. D., Turner, R. P., Roark, A. J., MacInerney, E. K., & Koslowski, G. P. (2017). Integration of EEG into psychiatric practice: A step toward precision medicine for autism spectrum disorder. *Journal of Clinical Neurophysiology*, 34, 230–235.
- Trevathan, E. (2002). Infantile spasms and Lennox-Gastaut syndrome. *Journal of Child Neurology*, 17(Suppl. 2), 2S9–2S22.
- Tuchman, R. (2013). Autism and social cognition in epilepsy: Implications for comprehensive epilepsy care. *Current Opinion in Neurology*, 26, 214–218.
- Tuchman, R., Moshe, S., & Rapin, I. (2009). Convulsing toward the pathophysiology of autism. *Brain & Development*, 31, 95–103.
- Tuchman, R., Alessandri, M., & Cuccaro, M. (2010). Autism spectrum disorders and epilepsy: Moving towards a comprehensive approach to treatment. *Brain & Development*, 32, 719–730.
- Tuchman, R., Hirtz, D., & Mamounas, L. A. (2013). NINDS epilepsy and autism spectrum disorders workshop report. *Neurology*, 81, 1630–1636.
- Venkateswaran, S., & Shevell, M. (2008). The case against routine electroencephalography in specific language impairment. *Pediatrics*, 122, e911–e916.
- Viscidi, E. W., Triche, E. W., Pescosolido, M. F., McLean, R. L., Joseph, R. M., Spence, S. J., & Morrow, E. M. (2013). Clinical characteristics of children with autism spectrum disorder and co-occurring epilepsy. *PLoS ONE*, 8, e67797.
- Viscidi, E. W., Johnson, A. L., Spence, S. J., Buka, S. L., Morrow, E. M., & Triche, E. W. (2014). The association between epilepsy and autism symptoms and maladaptive behaviors in children with autism spectrum disorder. *Autism*, 18, 996–1006.
- Volkmar, F., & Nelson, D. (1990). Seizure disorders in autism. *Journal of the American Academy of Child Psychiatry*, 29, 127–129.
- Weber, R. J., & Gadow, K. D. (2017). Relation of psychiatric symptoms with epilepsy, asthma, and allergy in youth with ASD versus psychiatry referrals. *Journal of Abnormal Child Psychology*, 45, 1247–1257.
- Wheless, J. (2004). Nonpharmacologic treatment of the catastrophic epilepsies of childhood. *Epilepsia*, 45 (Suppl. 5), 17–22.
- Woolfenden, S., Sarkozy, V., Ridley, G., Coory, M., & Williams, K. (2012). A systematic review of two outcomes in autism spectrum disorder-epilepsy and mortality. *Developmental Medicine and Child Neurology*, 54, 306–312.
- Youroukos, S. (2007). Autism and epilepsy. *ENCEPHALOS Archives of Neurology and Psychiatry*, 44, 200–203.

Autism and the Caribbean

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Historical Background

The Caribbean comprises over 700 islands, islets, reefs, and cays between North and South America. It is inhabited by approximately 42 million persons. Although many similarities exist between the islands and territories, there are huge variations in the following areas: population, gross domestic product, languages, educational status of the populous, and the healthcare systems. This report captures the status of autism in 12 of the English-speaking countries and territories of the Caribbean (Anguilla, Antigua and Barbuda, the Bahamas, Barbados, Dominica, Grenada, Jamaica, Saint Kitts and Nevis, Saint Lucia, St. Vincent and the Grenadines, Tortola, and Trinidad and Tobago) with information on published research from the English- and Dutch-speaking Caribbean.

There is a dearth of published data on the history of autism in the Anglophone Caribbean. Up until the late twentieth century, a minimum level of basic knowledge, lack of appropriate resources (health, educational, and social), stigma, and discrimination resulted in many individuals with developmental disabilities being hidden away at home or housed in mental institutions. Over the last two decades, there have been a number of drivers for addressing the needs of the disabled community, starting with the adoption of the Panama Commitment to Persons with Disabilities in the American Hemisphere by the Organization of American States (OAS) in 1996 and more recently the signature of the Convention of the Rights of Persons with Disabilities by all of the island states within the last decade (Collamarco et Delamonico 2013). On a regional level, the importance of addressing autism was first

discussed by Heads of Government in the context of a larger discussion on special needs and disability during the 34th regular meeting of the Caribbean Community (CARICOM) Heads of Government in the Port of Spain in July 6, 2013. The call to address special needs and disabilities with a higher-level meeting was raised by Haiti and led by the Prime Minister of the Bahamas the Honorable Perry Christie and Prime Minister of Trinidad and Tobago the Honorable Kamla Persad-Bissessar who reported that their child and grandchild, respectively, are autistic.

Services

No center in the English-speaking Caribbean offers services exclusively for autism. Over the last 40 years with increasing awareness of the need to identify and address early developmental

challenges in children, the power of early intervention, and the rights of all children, the number of child development centers throughout the region has grown (see Table 1). These centers cater to children with all forms of disability, including autism. The most recent additions have been the Marjorie Davis Institute which opened in the Bahamas on April 21, 2015, and the Autism Centre in the British Virgin Islands on October 29, 2012. The Autism Centre in Tortola initially exclusively provided services for children and adults on the autism spectrum; however, its function has evolved to include the provision of services for other disabilities. This evolution captures a trend that resonates throughout the region. Some of the child development centers are publicly funded; however, the majority are formed by non-governmental organization and receive significant subventions from the government and

Autism and the Caribbean, Table 1 Centers providing evaluation or treatment for autism

Country/territory	Centers providing autism services	Services provided	Free services
Bahamas	The Marjorie Davis Institute	Evaluation and treatment	Yes
	Caribbean Center for Child Development	Evaluation and treatment	No
Barbados	Albert Cecil Graham Development Centre	Evaluation and treatment	Yes
	Sunshine Early Stimulation Centre	Treatment	No
	The School for Special Needs	Treatment	No
	The Irvine Wilson School	Treatment	Yes
British Virgin Islands	The Autism Centre	Evaluation and treatment	Yes
Dominica	The Alpha Center	Treatment	No
	The Achievement Centre	Treatment	No
Jamaica	The Pediatric-Adolescent Clinic University Hospital, Kingston	Evaluation	No
	The Early Stimulation Centre and the Early Stimulation School	Evaluation and treatment	Yes
	The Montego Bay Autism Center	Treatment	No
	The McCann Child Development Centre	Treatment	No
	The Promise Learning & Training Centre	Treatment	No
St. Lucia	Child Development Guidance Clinic	Evaluation and treatment	Yes
Trinidad and Tobago	Child Development Clinic, Eric William Complex, Mount Hope	Evaluation	Yes
	The Child Guidance Clinic	Evaluation	Yes
	The Mental Health Clinic, San Fernando	Evaluation	Yes
	The Mental Health Clinic, Tobago	Evaluation	Yes
	The Autism Society of Trinidad and Tobago	Treatment	Yes

donations from philanthropic associations. Their budgets are extremely vulnerable to the fiscal adjustments that frequently occur in small developing economies. This situation also pertains to most of the private special education schools; some of which have closed as a result of inadequate funding.

There are extensive wait periods to access services in the public centers and clinics (up to 3 years in some countries). The extensive wait adds to the delay in diagnosis and commencement of interventions and adversely impacts outcomes. A significant minority of children access services in the private sector where the availability of trained professionals is often greater than the public sector. An effort to expand and decentralize services has been made in Barbados with the addition of speech and language therapy in some of the public clinics. Additionally the evaluation and treatment services are slowly increasing with greater awareness of autism and the benefits of early intervention.

The identification of autism sometimes occurs as a result of concerns raised during routine developmental assessments by pediatricians. However for many of the children who receive services in the public sector, concern about the child's socio-communicative development first occurs in the preschool and elementary school environments. It is also in the school environment that most children receive intervention.

For a vast number of children and adolescents who are identified, the main intervention is placement in a special education class with children who have other developmental disabilities. One-on-one instruction, routine application of applied behavioral analysis, Treatment and Education of Autistic and Related Communication Handicapped Children (TEACCH), and other evidence-based interventions are more common in private schools but seldom present for the majority of children who receive services in the public sector. Where evidence-based interventions for teaching children with autism are available, very few children receive the recommended 20–40 h per week. Speech and language therapy and occupational therapy are seldom integrated into the education system; these therapies are more likely to be integrated in the education plan

of children with autism who attend private schools. The availability of specialists and frequency of receipt of these services vary across the countries and territories with St. Vincent and the Grenadines (population, 109, 000) reporting no speech and language therapist on the island. Parents will often access psychoeducational testing, speech and language, occupational therapy, and behavior therapy privately; the cost of these services presents an additional financial burden in already difficult circumstances.

Some countries maintain a database of persons with disabilities, and in Jamaica the University of the West Indies maintains an autism database; however no other national registry of persons with autism was identified during the preparation of this chapter. Additionally outside of Jamaica and Aruba, no documented monitoring of the situation of persons with autism or studies in the area of autism were identified.

The University of the West Indies, Department of Child and Adolescent Health, Mona campus (Jamaica) has been the major center for research in autism in the region with extensive publication by Professor Maureen Samms-Vaughan, Dr. Mohammed Rahbar, and colleagues on possible etiological factors, associations, parental experience, and barriers to diagnoses and implementation of interventions for autism. Dr. Ingrid Van Balkom from the Child and Adolescent Psychiatry Clinic at Oranjestad (Aruba) has also been a major author of published research on autism in our region. They have contributed a wealth of information on potential causation, risk factors, and barriers to diagnoses and implementation of interventions in the Caribbean region.

Legal Issues, Mandates for Service

All of the ten independent countries described in this report and the United Kingdom (of which Anguilla and Tortola are overseas territories) ratified the Convention on the Rights of the Child in the 1990s. Six of the countries (Barbados, Dominica, Grenada, Jamaica, St. Vincent and the Grenadines, and Trinidad and Tobago) ratified the Convention on the Rights of Persons with Disabilities (CRPD) or joined by accession within the

last 10 years with five of the six joining in the last 3 years. The United Kingdom (of which Anguilla and Tortola are dependencies) ratified the CRPD in 1991.

The Bahamas and Jamaica have enacted disability legislation, and with their ratification of the CRPD, many of the other countries are in the process of developing disability legislation. The absence of disability-specific legislation and mandates has decreased the ability of the disabled community to advocate for its needs. The absence of legislation is also reflected in the lack of a structured approach to the provision of services and entitlements for the disabled community. Going forward it is absolutely essential that the organized leadership lobby their respective policy makers to achieve their support for allocating the drafting of specific disability legislation as a high priority in the face of a multitude of competing demands. Enforcement of the legislation in those countries which have enacted relevant laws encounters problems including but not limited to the availability of funding and skilled professionals. In spite of the above-stated challenges, the Dutch overseas territory Aruba has been making strides with the meeting of high-level national officials with representatives of Autism Speaks on September 22, 2014, to assist with the development of an autism strategy on island.

Most countries have stated in their education act the need to provide education for children with special needs in an appropriate environment; however there is no autism-specific legislation or mandates for provision of services or entitlements specifically for autism.

In the region, the receipt of disability assistance is not automatic; the vast majority of persons with disabilities are required to apply for disability assistance. Furthermore in some countries, the financial assistance can be discontinued if the individual is able to earn any income.

Overview of Current Treatments and Centers

Please see below the table of some of the centers that offer evaluation and/or treatment for autism.

Overview of Research Directions

Most of the research published on autism in the region has focused on causation; to this end the University of the West Indies, Mona campus, has led the way with published research in peer-reviewed journals. Their research has examined the possible role of heavy metals (manganese, cadmium, arsenic, and mercury), the role of glutathione S-transferase (*GST*) genes, parental age, factors inhibiting early identification, and intervention.

Dr. Ingrid Van Balkom from the Child and Adolescent Psychiatry Clinic at Oranjestad (Aruba) has also examined and confirmed paternal age as a risk factor and has studied the prevalence rates of autism spectrum disorder in Aruba which has been determined to be 5.3 per 1,000 for the period 1990–1999.

Overview of Training

Awareness training has been the most common type of training that occurs in the Anglophone Caribbean. Public education on autism is often incorporated into Autism month activities. Educational activities on the identification of children with autism targeting healthcare professionals and educators are usually linked to Autism month and Autism Awareness Day. A program of systematic training and continuous education for teachers who work in special education exist in the Bahamas and Tortola; however no systematic continuous education on autism exist for general teachers and healthcare providers in any of the Anglophone countries and territories of the region.

Social Policy and Current Controversies

In general, with increasing visibility and inclusion of persons with disabilities (e.g., in Barbados the leader of the Senate the Honorable Kerry-Ann Ifill is visually impaired, and a major supermarket chain in the region has integrated employees with disabilities in visible positions), attitudes toward persons with developmental disabilities

are improving with slow but steady movement to inclusivity.

Autism awareness continues to grow in the region as a result of continuous advocacy efforts spearheaded by the autism associations, other service organizations, and prominent families affected in the Caribbean region. Additionally, access to the Internet has allowed parents to become acutely aware of the importance of early diagnosis and interventions. In part, this increased knowledge adds to the frustration around accessing intensive, evidence-based intervention in their countries. Parents are often concerned about the quality of the educational services and are fearful that the emphasis of some special needs schools is skewed toward daycare as oppose to learning.

National autism associations exist in 5 of the 12 countries and territories: Bahamas, Barbados, Jamaica, Trinidad and Tobago, and Tortola. The associations are mainly funded by private sector and philanthropic support with some organizations receiving small subventions from the respective governments. These organizations vary in the scope and continuity of services they offer; at minimum, they offer educational information on autism spectrum disorders and support for affected individuals and their families.

The Autism Society of Trinidad and Tobago is one of the more active autism associations in the region with a register of 650 persons with autism. It trains parents as cotherapists and offers social skills play groups for the children. Additionally they provide recreational activities and life skills training for the adults, music and art therapy on Saturdays, and camps for adults and children during the long holidays.

Possibly the area of greatest concern beyond early identification is the plight of young adults with autism. Crisis often occurs when they surpass the age when schooling is mandated by law, and they have not attained skills for independent living or employment. Many parents (often single mothers who are the sole breadwinners of the household) grapple with the choice of leaving their dependent adult son or daughter at home unsupervised or staying away from work at the risk of compromising employment.

In 2015, the deficit of services for dependent children and adults with disabilities was in part addressed in Barbados and Dominica; in Barbados, through the philanthropic work of Derrick Smith, the state-of-the-art vocational center at the Derrick Smith Secondary School and Vocational Centre was opened. The vocational center caters to adults with developmental disabilities. Additionally on the island of Dominica, advocacy by the Parents Advocating for Children with Disabilities Inclusion in Society (PACIS) and an assessment of the number of children affected by disability on the island culminated in the development of the PACIS Care Center scheduled to be opened in September 2015. The center will provide respite for parents who have to make the difficult decision of earning an income outside of the home or caring for the physical needs of their children with disabilities.

References and Reading

- American Psychiatric Association. (1987). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Colamero, V., & Delamonica, E., et al. (2013). Policies for the inclusion of children with disabilities challenges (Newsletter on progress towards the millennium development goals from a child rights perspective). Number 15, April 2013 ISSN 1816-7551
- Dudzick, P., Elwan, A., & Metts, R. (2002). *Disability policies, statistics, and strategies in Latin America and the Caribbean: A review. Documento de trabajo del Departamento de Desarrollo Sostenible*. Washington, DC: Banco Interamericano de Desarrollo.
- Gjaltema, T., Ebbeson, L., & Gonzales, C. (2011). *An analysis of the status of implementation of the convention on the rights of persons with disabilities in the Caribbean*. Port of Spain: United Nations Economic Commission for Latin America and the Caribbean (ECLAC), Subregional Headquarters for the Caribbean.
- Klin, A., Lang, J., et al. (2000). Brief report: Interrater reliability of clinical diagnosis and DSM-IV criteria for autistic disorder: Results of the DSM-IV autism field trial. *Journal of Autism and Developmental Disorders*, 30(2), 163–167.
- Rahbar, M. H., Samms-Vaughan, M., Loveland, K. A., Pearson, D. A., Bressler, J., Chen, Z., ... Boerwinkle, E. (2012). Maternal and paternal age are jointly associated with childhood autism in Jamaica. *Journal of Autism and Developmental Disorders*, 42(9), 1928–1938.
- Rahbar, M. H., Samms-Vaughan, M., Ardjomand-Hessabi, M., Loveland, K. A., Dickerson, A. S., Chen, Z., ...

- Boerwinkle, E. (2012). The role of drinking water sources, consumption of vegetables and seafood in relation to blood arsenic concentrations of Jamaican children with and without autism spectrum disorders. *Science of the Total Environment*, 433, 362–370.
- Samms-Vaughan, M. E. (2014). The status of early identification and early intervention in autism spectrum disorders in lower-and middle-income countries. *International Journal of Speech-Language Pathology*, 16(1), 30–35.
- van Balkom, I. D., Bresnahan, M., Vogtländer, M. F., van Hoeken, D., Minderaa, R. B., Susser, E., & Hoek, H. W. (2009). Prevalence of treated autism spectrum disorders in Aruba. *Journal of Neurodevelopmental Disorders*, 1(3), 197–204.
- Van Balkom, I. D., Bresnahan, M., Vuijk, P. J., Hubert, J., Susser, E., & Hoek, H. W. (2012). Paternal age and risk of autism in an ethnically diverse, non-industrialized setting: Aruba. *PloS One*, 7(9), e45090–e45090.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 5–41). Hoboken: Wiley.
- Volkmar, F. R., Cicchetti, D. V., et al. (1992). Three diagnostic systems for autism: DSM-III, DSM-III-R, and ICD-10. *Journal of Autism and Developmental Disorders*, 22(4), 483–492.
- Volkmar, F. R., Klin, A., et al. (1994). Field trial for autistic disorder in DSM-IV. *The American Journal of Psychiatry*, 151(9), 1361–1367.
- World Health Organization. (1994). *Diagnostic criteria for research*. Geneva: World Health Organization.

Autism Behavior Checklist

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Synonyms

ABC

Description

The Autism Behavior Checklist (ABC) is one component of the Autism Screening Instrument for Educational Planning (ASIEP) and is the only one

that has been evaluated psychometrically. The ABC is a 57-item behavior rating scale assessing the behaviors and symptoms of autism for children 3 and older. The instrument consists of a list of 57 questions divided into five categories: (1) sensory, (2) relating, (3) body and object use, (4) language, and (5) social and self-help. Each item has a weighted score ranging from 1 to 4. The ABC is designed to be completed independently by a parent or a teacher familiar with the child for at least 3–6 weeks. It should take from 10 to 20 min to complete. The protocol is then returned to a trained professional for scoring and interpretation.

Historical Background

The Autism Behavior Checklist (ABC) was published in 1980 and is part of a broader tool, the Autism Screening Instrument for Educational Planning (ASIEP). The content of the ABC was based on other autism screening instruments available at the time of its development.

Psychometric Data

The ABC's item score weights and cutoff scores were developed using over 1000 completed questionnaires from children and adults with autism, intellectual disabilities, visual and hearing impairments, and emotional disturbance, as well as those with typical developmental profiles. Higher subtest or total scores reflect greater impairments and more severe levels of autism symptomology.

Although widely used for years, several concerns about its psychometric properties have been identified. For example, studies have found interrater reliability to be much lower than those reported in the initial study during development. In addition, the ABC subscales were not empirically derived and were established by grouping items based on face validity. Later studies have also shown significant differences between parent and teacher reports, although it is not clear whether the discrepancies indicate weaknesses specific to the ABC or reflect differences encountered

commonly when using multiple informants. Perhaps more important are questions concerning the sensitivity and specificity of the ABC. Several studies have suggested the cutoff score of 67 leads to a high number of false negatives. Studies lowering of the cutoff scores to 58 and 45 respectively have shown increased sensitivity and decreased specificity. The ABC has been shown to correlate significantly with the Gilliam Autism Rating Scale (GARS), but correlations with the Childhood Autism Rating Scale (CARS) have been variable.

Clinical Uses

The ABC is primarily designed to identify children with autism within a population of school-age children with severe disabilities. When used in conjunction with other diagnostic instruments and methods, the ABC can be useful as a symptom inventory to be used by clinicians in structuring their evaluation.

References and Reading

- Coonrod, E. E., & Stone, W. L. (2005). Screening for autism in young children. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Assessment, interventions, and policy) (Vol. 2). Hoboken: Wiley.
- Krug, D. A., Arisk, J. R., & Almond, P. J. (1980a). *Autism screening instrument for educational planning*. Austin: ProEd.
- Krug, D. A., Arisk, J. R., & Almond, P. J. (1980b). Behavior checklist for identifying severely handicapped individuals with high levels of autistic behavior. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 21(3), 221–229.
- Krug, D. A., Arisk, J. R., & Almond, P. J. (1993). *Autism screening instrument for educational planning* (2nd ed.). Austin: ProEd.
- Lord, C., & Corsello, C. (2005). Diagnostic instruments in autism spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Assessment, interventions, and policy) (Vol. 2). Hoboken: Wiley.
- Volkmar, F. R., Cicchetti, D. V., Dykens, E., Sparrow, S. S., Leckman, J. F., & Cohen, D. J. (1988). An evaluation of the autism behavior checklist. *Journal of Autism and Developmental Disorders*, 18(1), 81–97.

Autism Behavior Inventory (ABI)

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Synonyms

ABI, Autism Behavior Inventory; ABI-S, Autism Behavior Inventory-Short Form

Description

The ABI is a parent- or caregiver-reported outcome measure that was developed to assess change in core and associated symptoms of ASD. The scale consists of 62 items covering 5 domains of behavior. The two core ASD domains are Social Communication (comprised of Reciprocity and Verbal and Nonverbal communication subdomains) and Restrictive Behavior (comprised of Resistance to Change, Restricted Interests, Stereotypical Behavior, Hypersensitivity subdomains). In addition, three associated domains of Mood and Anxiety, Self-Regulation, and Challenging Behavior represent behaviors that are not part of the ASD diagnosis but commonly occur in individuals with ASD and inform clinical consideration. Caregivers endorse items on a 4-point scale relating either to Quality (how *well* behaviors are carried out) or Frequency (how *often* a behavior occurs), over the past 7 days. A shorter version of the ABI (ABI-S), that includes 24 items across all 5 domains, may be used to assess behavior more frequently (e.g., every 2 weeks).

The ABI was developed as an online rating scale, but a paper version is also available. A single form is suitable for caregivers of individuals with ASD aged 3 years through adulthood and may be used with both verbal and minimally verbal groups. Scores are obtained by the averaging of all completed responses for each domain. A core score corresponds to the average of all

responses across the Social Communication and Restrictive Behavior domains.

Historical Background

There are limited reliable, valid, and objective endpoints for measuring clinically and statistically significant changes in the core and associated symptoms of ASD, and this lack hinders the interpretation of treatment outcomes (McConachie et al. 2015). There is a need for measures that have shorter recall periods and reduced burden for respondents, with ample evidence demonstrating that the constructs measured are both relevant and well understood by parents (Aman et al. 2015). The ABI was developed in response to this need, in consultation with regulatory authorities, and in alignment with the FDA (2009) guidelines for patient- and caregiver-reported outcome measures. The iterative scale development of the ABI included two pilot studies (Bangerter et al. 2017), a validation study (Bangerter et al. 2019), and a cognitive interview study (Pandina et al. 2018). Through the entirety of scale development, an initial list of 300 items was refined and reduced through review and testing with multiple stakeholders and based on statistical performance and clinical importance. Caregiver feedback led to the inclusion of additional items on sleep and food sensitivity. Delphi review (Dalkey 1969) by an expert panel of ASD researchers and clinicians led to the selection of items for the ABI-S that were representative across the core and associated domains and were considered to have the most potential for short-term change. Finally, cognitive interviews with caregivers of individuals with ASD, who were representative of a broad range of age and linguistic ability, confirmed that the instructions and items were well understood and that the response options and response period of 1 week were appropriate. Caregivers reported that scale was easy to use and comprehensive in the range of items covered. As a result of this final cognitive debriefing study, a small number of items were removed or reworded resulting in the current 62-item version of the ABI (v1.1) and a 24-item ABI-S (v1.1).

Psychometric Data

Clinical experts assigned ABI items to groups forming domains and subdomains; these were further confirmed with factor analysis in a sample ($n = 353$) of online survey responses (Bangerter et al. 2017). The items were subjected to confirmatory factor analysis (CFA) with principal axis factoring. The Kaiser-Meyer-Olkin measure of the sampling adequacy ratio of total items was 0.932, indicating appropriateness of factor structure. The five factors accounted for 63.74% of the variance in item scores. Items loaded onto respective domains and any cross-loading items were reviewed as part of the item-reduction process. In addition, inter-item correlation, item discrimination, and item information functioning were taken into account to produce a 73-item version of the ABI. This version was further reduced to 62 items comprising the ABI v1.1.

Research in a group of individuals with a clinically confirmed diagnosis of ASD ($n = 144$) demonstrated sound psychometric properties of the ABI v1.1 (Bangerter et al. 2019). Internal consistency across domains was high (Cronbach's α 0.84–0.89). Test-retest reliability 3–5 days after baseline was excellent (ICC values ranging from 0.84 to 0.93). Convergent and discriminant validity were assessed by comparing ABI domain scores with pre-specified subscales of comparison instruments. For convergent validity, strong positive Pearson correlations were found in core ASD domains, for example, ABI Social Communication with Social Responsiveness Scale (SRS), Social Communication and Interaction (0.69), and ABI Restrictive Behavior with Repetitive Behavior Scale-Revised (RBS-R) (0.75). Similarly, for associated domains, ABI Mood and Anxiety correlated with Child and Adolescent Symptom Inventory (CASI) anxiety scale (0.77), ABI Self-Regulation correlated with Aberrant Behavior Checklist (ABC) Hyperactivity and Non-Compliance (0.88), and ABI Challenging Behavior with ABC Irritability (0.76).

Short form items were selected based on their statistical performance and clinical expert feedback. Psychometric properties for the ABI-S v1.1 were similar to those reported for the ABI

v1.1; for example, internal consistency for the ABI-S domains was 0.69–0.79, and test-retest reliability was good (0.77–0.88).

The ABI has been developed as a measure of change in symptoms or behavior over time and is currently being implemented within an interventional clinical trial (NCT03664232), the data from which will allow understanding of the sensitivity to change of the ABI. In an 8–10-week treatment as usual, observational trial (Ness et al. 2019; Bangerter et al. 2019), participants showing improvements in ASD severity based on category change in SRS-2 Total Scores showed analogous ABI domain score improvements in Core ASD Symptoms, Social Communication, and Restrictive Repetitive Behaviors (moderate to large within-group effect sizes of 0.63, 0.50, and 0.41, respectively); these effects were not observed in groups with no documented change or who were reported to have worsened.

Further validation, including sensitivity to change and a confirmatory factor analysis of the ABI v 1.1, is planned as data from ongoing research studies become available. Studies will take place in individuals with broader range of ages and levels of functioning. Translations of the ABI into various languages are also in preparation, alongside associated cross-cultural validation.

Clinical Uses

The ABI and the ABI-S are designed to measure change in response to intervention and allow for the potential to complete one instrument in place of discrete alternatives commonly used in treatment outcome studies and clinical drug trials. Both instruments are freely available in paper form for use in clinical or research settings, subject to terms and conditions, and can be obtained via email at autismbehaviorinventory@its.jnj.com.

Caregivers, including healthcare professionals who spend more than 3–4 h a week with the person who has ASD, can complete the scale. The scale can be repeated at weekly or longer intervals by the same respondent, in order to determine response to an intervention based on

change from baseline administration. The ABI takes approximately 10–20 min to complete and the ABI-S around 5–10 min.

A clinician version of the ABI (ABI-C) is currently being tested. This version allows clinicians and other healthcare professionals to assess the same domains and subdomains of the ABI, using 14 items with a 0–7-point scale of symptom severity.

See Also

- ▶ Aberrant Behavior Checklist
- ▶ Behavior Rating Scale (BRS)
- ▶ Child and Family Characteristics that Predict Clinic Appointment Attendance and Alignment with Providers
- ▶ Cronbach's Alpha
- ▶ Sensory Sensitivity Questionnaire: Revised
- ▶ Social Responsiveness Scale

References and Reading

- Aman, M. G., Arnold, L. E., & Hollway, J. A. (2015). Assessing change in core autism symptoms: Challenges for pharmacological studies. *Journal of Child and Adolescent Psychopharmacology*, 25, 282–285.
- Bangerter, A., Ness, S., Aman, M. G., Esbensen, A. J., Goodwin, M. S., Dawson, G., ... & Pandina, G. (2017). Autism behavior inventory: A novel tool for assessing core and associated symptoms of autism spectrum disorder. *Journal of Child and Adolescent Psychopharmacology*, 27(9), 814–822.
- Bangerter, A., Ness, S., Lewin, D., Aman, M. G., Esbensen, A. J., Goodwin, M. S., ... & Pandina, G. (2019). Clinical validation of the autism behavior inventory: Caregiver-rated assessment of Core and associated symptoms of autism Spectrum disorder. *Journal of Autism and Developmental Disorders*, 1–12.
- Dalkey, N. (1969). An experimental study of group opinion: The Delphi method. *Futures*, 1(5), 408–426.
- Food and Drug Administration. (2009). Guidance for industry: Patient-reported outcome measures: Use in medical product development to support labeling claims. Accessed 28 Nov 2017, from <https://www.fda.gov/downloads/drugs/guidances/ucm193282.pdf>.
- McConachie, H., Parr, J. R., & Glod, M. (2015). Systematic review of tools to measure outcomes for young children with autism spectrum disorder. *Health Technology Assessment*, 19, 1–506.
- Ness, S. L., Bangerter, A., Manyakov, N. V., Lewin, D., Boice, M., Skalkin, A., ... & Hendren, R. (2019). An observational study with the Janssen autism knowledge

engine (JAKE[®]) in individuals with autism spectrum disorder. *Frontiers in Neuroscience*, 13, 111.

Pandina, G., Bangerter, A., Ness, S., Trudeau, J., Stringer, S., Knoble, N., & Lenderking, W. R. (2018). Parent validation of the autism behavior inventory—A cognitive debriefing study (Abstract W143). *Presented at the 57th Annual Meeting of the American College of Neuropsychopharmacology (ACNP)*, 9–13 Dec 2018. https://acnp.societyconference.com/user/server/submission_pdf.php

Autism Collaboration, Accountability, Research, Education, and Support (CARES) Act of 2019 (Also Referred to as the “Autism CARES Act of 2019”)

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Definition

The “Autism Collaboration, Accountability, Research, Education, and Support Act of 2019,” abbreviated as the “Autism CARES Act of 2019” (hereafter “the Act”), is a milestone law in the United States that provides federal funding to the US Department of Health and Human Services (DHHS) for services and research concerning individuals with autism spectrum disorder (ASD) (Pub. L. No. 116–60, 2019).

The Act supports ASD-related programs in three DHHS agencies: National Institutes of Health (NIH), Centers for Disease Control and Prevention (CDC), and Health Resources and Services Administration (HRSA) (DHHS 2017). The programs covered under the Act include biomedical ASD research and the continued development of best practices to enhance the lives of persons with ASD. Importantly, the Act also formally expands the focus of US government programs to include research and services that assist individuals with ASD across their life spans, not only children and young adults (Turcotte et al. 2016).

Legislative Details

Under the Act, the US Congress assigns government funds for a wide range of ASD-related programs with an emphasis on seven key activities: research, surveillance, early detection, prevention, treatment, education, and disability support. The Act has three general goals:

[1] The law expands efforts to conduct research, surveillance, education, detection, and intervention for all individuals with autism spectrum disorder across their lifespan, regardless of age...

[2] [It] also aims to reduce disparities among individuals from diverse racial, ethnic, geographic, or linguistic backgrounds, and [3] directs additional care to rural and underserved areas. (U.S. House of Representatives 2019)

The Act defines “underserved areas” as geographic locations where there are shortages of healthcare professionals available to provide services to the public. In addition, the Act re-establishes the Interagency Autism Coordinating Committee (IACC) federal advisory committee, which is responsible for coordinating and guiding government efforts on issues related to ASD in public forums (IACC 2001–2020). The IACC is made up of federal government workers, industry experts, academic researchers, and members of the public.

The Act’s 5-year reauthorization of funds includes annual authorizations of \$23.1 million for developmental disability surveillance and research; \$50.599 million for ASD education, early detection, and intervention; and \$296 million to carry out the work of the IACC and other DHHS programs. The Act’s funding covers activities through September 30, 2024. Congress will need to provide additional government funding before October 1, 2024, so that the DHHS can continue the services and research facilitated under the Act.

The Autism CARES Act of 2019 is designed to help society obtain new insights regarding effective ASD services, interventions, and treatments (U.S. House of Representatives Congressman Chris Smith 2019). The Act contains eight primary components:

1. Authorizes \$1.8 billion in funds, including \$296 million in annual funding to the NIH

- (2020), \$23.1 million to the CDC (2019), and \$50 million to HRSA (2019)
2. Reauthorizes and expands the IACC
 3. Reauthorizes government program activities including:
 - (a) Expanding ASD research at the NIH (2015)
 - (b) Continuing the CDC’s collection of state-level ASD data (CDC 2020)
 - (c) Continuing the ASD education, early detection, and intervention activities supported by the HRSA and the IACC (HRSA 2020)
 4. Revises the scope of government programs and activities in three key areas:
 - (a) Encompassing ASD individuals of all ages, rather than only children
 - (b) Increasing funding on programs in areas with a shortage of personal health services
 - (c) Reducing health-outcome disparities across diverse populations.
 5. Adds new members of IACC from the Departments of Labor, Justice, Veterans Affairs, and Housing and Urban Development.
 6. Increases from two to three IACC members who are self-advocates, parents or legal guardians and advocacy/service organizations.
 7. Empowers the DHHS Secretary to prioritize grants to rural and underserved areas across the United States
 8. Requires DHHS agencies to compile a comprehensive report to Congress that details current and future outlooks government services and research initiatives concerning ASD. This report must be completed by no later than September 30, 2021, which is 2 years after

the law was first enacted. The Act specifies that the report must contain the following information:

- (a) Demographic factors associated with the health and well-being of individuals with ASD
- (b) Recommendations on establishing best practices to ensure interdisciplinary coordination
- (c) Improvements for health outcomes
- (d) Community-based behavioral support and interventions
- (e) Nutrition, recreational, and social activities
- (f) Personal safety

Legislative History and Background

Congress typically approves funds for DHHS’ ASD-related programs in 5-year appointments. For the past two decades, Congress has continuously recognized the need for the IACC and for increased US government research and support programs regarding ASD. See Table 1. In 2000, Congress first established the IACC under the Children’s Health Act of 2000 – this law intensified US government research, prevention, and treatment activities for a number of conditions that significantly impact children, including ASD (Pub. L. No. 106–310, 2000). In 2006, Congress passed the Combating Autism Act of 2006, which focused on specific DHHS services for populations with ASD and elevated the status of the IACC as an official federal advisory committee that communicates directly with the Office of the Secretary of DHHS (Pub. L. No. 109–416, 2006).

Autism Collaboration, Accountability, Research, Education, and Support (CARES) Act of 2019 (Also Referred to as the “Autism CARES Act of 2019”),

Table 1 Timeline of key federal legislation to support ASD-related government programs: 5-year reauthorization cycles from 2000 to 2019

Year	Law title	Public law number
2000	Children’s Health Act	Public Law 106–310
2006	Combating Autism Act	Public Law 109–416
2011	Combating Autism Reauthorization Act	Public Law 112–32
2014	Autism Collaboration, Accountability, Research, Education, and Support (CARES) Act	Public Law 113–157
2019	Autism Collaboration, Accountability, Research, Education, and Support (CARES) Act	Public Law 116–60

From 2006 to the present, Congress has reauthorized funding for the IACC and steadily expanded funding for ASD-related programs within DHHS. Each act focuses on supporting federal ASD research and services for surveillance, early detection, prevention, treatment, education, and disability programs within DHHS. In 2011, 2014, and 2019, Congress passed the following respective laws: Combating Autism Reauthorization Act of 2011 (Pub. L. No. 112–32, 2011); Autism Collaboration, Accountability, Research, Education, and Support (CARES) Act of 2014 (Pub. L. No. 112–32, 2014); and the Autism Collaboration, Accountability, Research, Education, and Support (CARES) Act of 2019 (Pub. L. No. 116–60, 2019).

On February 7, 2019, the Act was first introduced as a bill within the House of Representatives (House Bill 1058). The House passed the Act on July 24, 2019. The Senate approved it shortly thereafter on September 19, 2019. On September 30, 2019, the Act was signed into law by the president. More than 35 nonprofit organizations endorsed the Act, including the Autism Society of America, Autism Speaks, Autism New Jersey, the Association of University Centers on Disabilities, the Children’s Hospital Association, the National Council on Severe Autism, the Congress, and the National Down Syndrome Society.

See Also

- [Civil Rights Act of 1964](#)
- [Social Security Amendments of 1965 \(or “Medicare Act of 1965” and/or the “Medicaid Act of 1965”\)](#)

References and Reading

- Autism Collaboration, Accountability, Research, Education and Support (CARES) Act of 2014, Pub. L. No. 113–157, 128 Stat. 1831, codified as amended at 42 U.S.C. § 280i (2014). Retrieved from <https://www.govinfo.gov/content/pkg/PLAW-113publ157/pdf/PLAW-113publ157.pdf>
- Autism Collaboration, Accountability, Research, Education, and Support (CARES) Act of 2019, House Bill (H.R.) 1058, 116th Congress. (2019).
- Autism Collaboration, Accountability, Research, Education, and Support (CARES) Act of 2019, Pub. L. No. 116–60, 133 Stat. 1110, codified as amended at 42 U.S.C. § 280i (2019) and 42 U.S.C. § 284g (2019). Retrieved from <https://www.congress.gov/116/plaws/publ60/PLAW-116publ60.pdf>
- Children’s Health Act of 2000, Pub. L. No. 106–310, 114 Stat. 1101, codified as amended at 21 U.S.C. § 802 (2000), 21 U.S.C. § 802 (2000), 21 U.S.C. § 823 (2000), 21 U.S.C. § 824 (2000), 21 U.S.C. § 830 (2000), 21 U.S.C. § 841 (2000), 21 U.S.C. § 853 (2000), 21 U.S.C. § 856 (2000), 21 U.S.C. § 863 (2000), 28 USC § 994 (2000), 42 U.S.C. § 10801 (2000), 42 U.S.C. § 10802 (2000), 42 U.S.C. § 10804 (2000), 42 U.S.C. § 10822 (2000), 42 U.S.C. § 10827 (2000), 42 U.S.C. § 2000 (2000), 42 U.S.C. § 241 (2000), 42 U.S.C. § 243 (2000), 42 U.S.C. § 247 (2000), 42 U.S.C. § 254 (2000), 42 U.S.C. § 256 (2000), 42 U.S.C. § 257 (2000), 42 U.S.C. § 274 (2000), 42 U.S.C. § 280 (2000), 42 U.S.C. § 284 (2000), 42 U.S.C. § 284 (2000), 42 U.S.C. § 285 (2000), 42 U.S.C. § 288 (2000), 42 U.S.C. § 290 (2000), 42 U.S.C. § 294 (2000), 42 U.S.C. § 300 (2000), 42 U.S.C. § 3751 (2000), and 42 U.S.C. § 10801 (2000). Retrieved from <https://www.govinfo.gov/content/pkg/PLAW-106publ310/pdf/PLAW-106publ310.pdf>
- Combating Autism Act of 2006, Pub. L. No. 109–416, 120 Stat. 2821, codified as amended at 42 U.S.C. § 241 (2006), 42 U.S.C. § 281 (2006), and U.S.C. § 284g (2006). Retrieved from <https://www.govinfo.gov/content/pkg/PLAW-109publ416/pdf/PLAW-109publ416.pdf>
- Combating Autism Reauthorization Act of 2011, Pub. L. No. 112–32, 125 Stat. 361, codified as amended at 42 U.S.C. § 280 (2011). Retrieved from <https://www.govinfo.gov/content/pkg/PLAW-112publ32/pdf/PLAW-112publ32.pdf>
- Turcotte, P., Mathew, M., Shea, L., Brusilovskiy, E., & Nonnemacher, S. (2016). Service needs across the lifespan for individuals with autism. *Journal of Autism and Developmental Disorders*, 46(7), 2480–2489. <https://doi.org/10.1007/s10803-016-2787-4>.
- U.S. Department of Health & Human Services. (2017). Autism support: Our commitment to supporting individuals on the autism spectrum and their families. Retrieved from <https://www.hhs.gov/programs/topic-sites/autism/autism-support/index.html>
- U.S. Department of Health & Human Services Centers for Disease Control and Prevention. (2020). Autism and Developmental Disabilities Monitoring (ADDM) Network. Retrieved from <https://www.cdc.gov/ncbddd/autism/addm.html>
- U.S. Department of Health & Human Services Centers of Disease Control and Prevention. (2019). U.S. CDC FY 2021 congressional justification (pp. 175–176). Retrieved from <https://www.cdc.gov/budget/documents/fy2021/FY-2021-CDC-congressional-justification.pdf>

- U.S. Department of Health & Human Services Health Resources & Services Administration. (2019). U.S. HRSA FY 2021 congressional justification (pp. 191–194). Retrieved from <https://www.hrsa.gov/sites/default/files/hrsa/about/budget/budget-justification-fy2021.pdf>
- U.S. Department of Health & Human Services Health Resources & Services Administration. (2020). Maternal & child health: Programs & initiatives: Autism. Retrieved from <https://mchb.hrsa.gov/maternal-child-health-initiatives/autism>
- U.S. Department of Health & Human Services Interagency Autism Coordinating Committee. (2001–2020). Autism reports. Retrieved from <https://iacc.hhs.gov/publications/autism/>
- U.S. Department of Health & Human Services Interagency Autism Coordinating Committee. (2019). Autism CARES Act of 2019. Retrieved from <https://iacc.hhs.gov/about-iacc/legislation/autism/cares-act-2019/>
- U.S. Department of Health & Human Services National Institutes of Health Office of Extramural Research. (2015). Research Portfolio Online Reporting Tools (RePORT): Project listing by category: Autism. Retrieved from https://report.nih.gov/categorical_spending_project_listing.aspx?FY=2018&ARRA=N&DCat=Autism
- U.S. Department of Health & Human Services National Institutes of Health Office of Extramural Research. (2020). Research Portfolio Online Reporting Tools (RePORT): Estimates of funding for various Research, Condition, and Disease Categories (RCDC): Summary table. Retrieved from https://report.nih.gov/categorical_spending.aspx
- U.S. House of Representatives. (2019, July 23). Autism Collaboration, Accountability, Research, Education and Support Act of 2019: To accompany H.R. 1058 (Report 116–177). Retrieved from <https://www.congress.gov/116/crpt/hrpt177/CRPT-116hrpt177.pdf>
- U.S. House of Representatives Congressman Chris Smith. (2019). Autism CARES Act of 2019 signed into law. Retrieved from <https://chrissmith.house.gov/news/documentsingle.aspx?DocumentID=406156>

Autism Cymru

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Major Areas or Mission Statement

Autism Cymru is Wales's pioneering national charity for Wales. It is a practitioner-led charity set up in 2001 to improve the lives of people in

Wales with an autistic spectrum disorder and their families. It has a dedicated national brief in Wales and in the projection of Welsh practice within and outside Wales. Autism Cymru takes the view that everyone with an autistic spectrum disorder in Wales should receive a service appropriate to their assessed needs, whatever their age and wherever they live. In order to achieve this, Autism Cymru actively promotes at both national and local levels the practice of strategic, collaborative, and multidisciplinary partnerships and highly focused coordination of services to people with autistic spectrum disorders and their families. Autism Cymru's primary task was successfully to influence the Welsh Assembly Government to establish the world's first government-led strategy for autism, which was launched at Autism Cymru's third International Autism Conference in Cardiff in April 2008. Autism Cymru's Chief Executive, Hugh Morgan OBE, heads up the implementation of the Assembly Government's Action Plan for Autism.

Landmark Contributions

Wales is the only country in the world with a national strategy for autism.

Major Activities

Autism Cymru runs the pioneering bilingual websites, Awares (www.awares.org). Every November, Autism Cymru's editor, Adam Feinstein, runs the Awares international online autism conference (www.awares.org/conferences), the largest event of its kind in the world, with more than 60 world autism experts taking part, along with thousands of delegates. Professor Simon Baron-Cohen has called this event "the finest online conference on the planet." Autism Cymru together with Autism Northern Ireland, Scottish Society for Autism, and the Irish Society for Autism has launched the Celtic Nations Autism Partnership. This will lead to shared opportunities for joint working in Northern Ireland, Scotland,

and Republic of Ireland, in addition to Autism Cymru's existing work in Wales.

Autism Cymru carries out research in partnerships with universities in Wales. In 2010, Professor Sue Leekam became the first chair of autism at Cardiff University and head of the new Welsh Autism Research Centre, based at the university's school of psychology and supported by Autism Cymru. Autism Cymru works in partnership with Mudiad Ysgolian Meithrin with funding from Children in Need to train Welsh medium playgroup leaders across Wales. Autism Cymru works in partnership with Autism Northern Ireland (PAPA) on UK and European campaigns to improve the lives of those with autism and to project best practice in each country. Autism Cymru works with local authorities and local health boards to develop local strategies for autism. Autism Cymru works with local education authorities in Wales to deliver its Inclusive Schools and ASDs: Whole School Training and Research Project. Autism Cymru works in partnership with the North Wales Police and Dyfed Powys Police to operate the Emergency Services ASD Attention Card Scheme which raises awareness of autism among members of the emergency services in Wales.

Autism Cymru has worked with Bro Morgannwg NHS Trust on a research project connected to the criminal justice system. Autism Cymru operates the AWARES EDUNET website and School Fora for education professionals. Autism Cymru publishes books and bilingual information booklets for professionals, parents, and people with autism. Publications include *All About Autistic Spectrum Disorders* and *My Brother Gwern*, a book for siblings of children with autism which won an award at Welsh Language in Healthcare Awards 2006. Autism Cymru's work also takes place on an international stage and with European partners, including Autism-Europe. For example, its 2009–2012 European-funded *Deis Cyfle* project (Opportunities for people with autism in education and employment) reached out to over 5,700 people across Wales and Ireland. The charity is also the sole national autism charity governed by those living in Wales. Autism Cymru's Chair is Professor Bill Fraser CBE. Its Patron is Lord Dafydd Wigley and its President is Dame Stephanie Shirley.

Autism Diagnostic Interview-Revised

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Abbreviations

ADOS Autism diagnostic observation schedule
ASD Autism spectrum disorders

Synonyms

[ADI-R](#)

Description

The Autism Diagnostic Interview-Revised (ADI-R; Le Couteur et al. 2003; Lord et al. 1994) is a standardized, semistructured, investigator-based interview administered by trained clinicians to parents or caregivers of individuals referred for a possible autism spectrum disorder (ASD). The ADI-R includes 93 items in three domains of functioning: communication; reciprocal social interactions; and restricted, repetitive, and stereotyped patterns of behavior, as well as other aspects of behaviors. All items in the ADI-R are coded for current and past behavior. Current refers to whether the behavior has occurred in the past 3 months. For some items, "past" refers to whether the behavior "ever" occurred, whereas others ask whether the behavior was present during a specifically defined period between 4 and 5 years of age (referred to as "most abnormal 4 to 5").

Up to 42 of the interview items are systematically combined to produce a formal, diagnostic algorithm for autism based on the ICD-10 (World Health Organization [WHO] 1990) and DSM-IV (American Psychiatric Association [APA] 1994) criteria as specified by the authors. In addition to the three domains of behavior, there is a fourth domain, *abnormality of development evident at or before 36 months*, to indicate whether the child meets criteria for age of onset. Each domain has a cutoff; a child must meet or exceed cutoffs in all four areas to receive an ADI-R classification of “autism.” Separate cutoffs are available for the communication domain, depending on whether or not the child is verbal (defined as showing “functional use of spontaneous, echoed, or stereotyped language that, on a daily basis involves phrases of three words or more that at least sometimes include a verb and are comprehensible to other people,” a score of 0 on item 30 *overall level of language*). Other criteria including using lower cutoffs with the same set of items have been used to create an algorithm for broader classification of autism spectrum disorders (ASD) as in several collaborative studies (Dawson et al. 2004; Lainhart et al. 2006; Risi et al. 2006). The diagnostic algorithm for children 4 years old and above is based on the “ever/most abnormal” codes, but current behavior algorithm forms are available to facilitate a clinical diagnosis for children from 2 years old and above.

A toddler version of the ADI-R was also developed several years ago to provide descriptive data for research with children under 4 years of age. The Toddler ADI-R has a total of 125 items, including 32 new questions and codes about the onset of autism symptoms and general development. Other items are identical to the ADI-R, with the exception that the Toddler ADI-R does not have codes for behaviors between 4 and 5 years of age.

Previous analyses suggested that the diagnostic algorithm was useful for children with a nonverbal mental age above 2 years (Le Couteur et al. 1989; Lord et al. 1994; Rutter et al. 2003). Thus, the interview had been appropriate for the diagnostic assessment of any person within the age range extending from early childhood to adult life,

provided that they have a nonverbal mental age above 2 years. Recently, however, newly developed algorithms for toddlers and young preschoolers have shown improved predictive validity compared to the preexisting algorithms for young children from 12 to 47 months of age (Kim and Lord 2011). These algorithms extend the use of the ADI-R to children as young as 12 months and a nonverbal developmental level of at least 10 months. In addition, these new algorithms include items present in both the toddler and standard versions of the ADI-R, allowing for use of the algorithms with either version.

Most items in the ADI-R relate to behaviors that are rare in individuals who do not have ASD and/or who do not have profound intellectual disabilities. Thus, numerical estimates of the scores of typically developing children based on general population have not been obtained. However, there have been several comparisons to children and adolescents with other disorders, which have been used in the development of the diagnostic algorithms (Le Couteur et al. 1989; Lord et al. 1994; Kim and Lord 2011). Researchers have used individual domain scores or an overall total of the three domains as estimates of autistic symptom severity, though the validity of this approach has not been directly tested. Scores have been published for many research populations but not yet systematically dimensionalized.

Historical Background

The ADI was first developed in 1989 (Le Couteur et al. 1989), which was modified in 1994 (Lord et al. 1994). The 1994 version was somewhat shorter than the original in order to make the interview more feasible in both clinical and research settings. The current version of the ADI-R was published in 2003 by Western Psychological Services.

The development of the toddler version of the ADI-R was completed in 2006 for research purposes. Following the development of the toddler version of the ADI-R, there was an increase in demand for diagnostic instruments for very young children, which prompted the development of the

new diagnostic algorithms for toddlers and young preschoolers (Kim and Lord 2011). The final algorithms for toddlers and young preschoolers contain fewer items than the original algorithms and are appropriate for use with children 12–47 months of age.

Psychometric Data

Psychometric properties for the original ADI were reported for a sample of 16 children and adults with autism and 16 children and adults with intellectual disabilities; each group included individuals that spanned wide ranges of age and performance IQ (with a mean age of 12.28 years and a standard deviation of 3.43 from a performance IQ of 43 to 71). Participants were carefully selected and blindly interviewed and coded. Interrater reliability was assessed, with multirater kappas ranging from 0.25 to 1 for each item. Intraclass correlations were above 0.94 for all subdomain and domain scores. The majority of individual items showed good discriminative validity between the autism group and the group of individuals with nonautism intellectual disabilities (Le Couteur et al. 1989).

Psychometric properties for the development of the algorithms for the current ADI-R were based on a sample of 25 children with autism and 25 children with intellectual disabilities who were carefully selected and blindly interviewed and coded (Lord et al. 1994; Rutter et al. 2003). These children ranged in chronological age from 36 to 59 months, with nonverbal mental ages ranging from 21 to 74 months. Using a sample of 10 children, interrater reliability was assessed; multirater kappas ranged from 0.63 to 0.89 for each item. Using the same sample, intraclass correlations were above 0.92 for all subdomain and domain scores. In addition, after the initial standardization of the ADI-R in 1989, a separate sample of 53 children with autism and 41 nonautistic children with intellectual disabilities or language impairments was used to further assess the validity of the ADI-R (Lord et al. 1993). The results of the study showed that the interrater reliability was as high as the initial study, with multirater kappas

ranging from 0.62 to 0.96 for individual items. Test-retest reliability was also very high, with all coefficients in the 0.93–0.97 range.

The majority of individual items in the current ADI-R showed good discriminative validity between children with autism and children with intellectual disabilities (see Lord et al. 1994). The existing algorithms differentiated children with autism over 36 months of age from children with nonspectrum disorders, showing high sensitivity and specificity (both over 0.90). Further analyses of data from preschool children revealed that the ADI-R algorithms differentiated children over 2 years with ASD from those with other developmental disorders. However, for children under 2 years, discrimination between nonverbal children with ASD and nonverbal children without ASD was poor, resulting in low specificity, especially for children with mental ages under 18 months, (Lord et al. 1993).

In a more recent study including a larger sample (Risi et al. 2006), the ADI-R showed high sensitivity (above 80%) for children with ASD under 3 years of age, but lower specificity for the comparison of nonautism ASD versus nonspectrum disorders (around 70%). Ventola et al. (2006) reported that, for children between 16 and 37 months of age, the diagnostic classifications made based upon the ADI-R algorithm resulted in lower sensitivity than those made using the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 1999), Childhood Autism Rating Scale (CARS; Schopler et al. 1980), or clinical judgment using the DSM-IV criteria. Wiggins and Robins (2008) also found that ADI-R algorithms resulted in poor sensitivity for children in the same age range when the standard cutoff for the RRB domain was included in the diagnostic criteria. Given the low sensitivities and specificities being reported for young children, new ADI-R algorithms were developed for toddlers and preschoolers between 12 and 47 months of age using a sample of 491 children with ASD, 136 with nonspectrum disorders (NS), and 67 with typical development (Kim and Lord 2011). The new ADI-R algorithms consist of two different cutoff scores: one for research (more restrictive, higher specificity with lower

sensitivity) and one for clinical purposes (more inclusive, higher sensitivity with lower specificity). They also include “ranges of concern” for clinical use (discussed below). In this sample, sensitivity using the clinical cutoff ranged from 80% to 94% and specificity ranged from 70% to 81% for the comparison of nonautism ASD vs. NS. Using the research cutoffs, the comparison of nonautism ASD vs. NS resulted in sensitivity ranging from 80% to 84% and specificity ranging from 85% to 90%. Another multi-site study (Kim et al. 2013) using two independent datasets provided by National Institute of Health funded consortia, the Collaborative Programs for Excellence in Autism, and Studies to Advance Autism Research and Treatment ($n = 641$) and the National Institute of Mental Health ($n = 167$) replicated the results from the original psychometric study, including the diagnostic validity and factor structure of the new algorithms for toddlers and young preschoolers (Kim and Lord 2011). Results suggested that the new ADI-R algorithms can be appropriately applied to existing research databases with children from 12 to 47 months and down to nonverbal mental ages of 10 months for diagnostic grouping. With a non-US sample, sensitivities, especially for those with phrase speech, were lower, using the new algorithms for toddlers and young preschoolers, suggesting that the algorithms need to be replicated more with other independent, non-US samples (de Bildt et al. 2015).

Clinical Uses

The ADI-R offers a profile of a child, adolescent, or adult which includes information regarding reciprocal social interactions, language and communication, and restricted, repetitive, and stereotyped behaviors and interests. Items are scored based on caregivers’ detailed descriptions of the history and behaviors of their child, thus allowing the clinician to gather both quantitative and qualitative information. One important caveat for clinical users to recognize is that diagnostic classifications based on the algorithms and true clinical diagnoses are not the same. Clinical diagnosis is based on multiple sources of information,

including direct observations (Le Couteur et al. 2007; Risi et al. 2006; Kim and Lord 2012). Risi et al. (2006) found a better balance of sensitivity and specificity when the ADI-R and the ADOS were used in combination compared to when each instrument was used alone. The combined use of these instruments resulted in sensitivity and specificity of 82% and 86%, respectively, for children with autism compared to children with non-spectrum disorders over age 3 years. For younger children, sensitivity and specificity for the same diagnostic comparison using both instruments were 81% and 87%, respectively. In contrast, when each instrument was used alone, specificities ranged from 59% to 72%. Le Couteur and her colleagues (2007) also examined the combined use of the ADOS and ADI-R for preschoolers with ASD using revised ADOS algorithms (Gotham et al. 2007). Consistent with Risi’s 2006 study, the authors found that combining information from both ADOS and ADI-R provided improved diagnostic accuracy compared to either instrument in isolation. Similarly, using the newly developed ADI-R algorithms for toddlers and young preschoolers and the revised ADOS and new ADOS-Toddler algorithms, Kim and Lord (2011) also found that for very young children, the combined use of the ADOS and ADI-R improved diagnostic validity compared to when each instrument was used alone. Thus, even though the ADI-R provides information about the individual’s history and description of his or her current functioning from a broad range of contexts, the ADI-R alone cannot be used to make a clinical diagnosis.

The diagnostic algorithm cutoffs allow classification of ASD based on patterns of behavior, meeting the current DSM-IV or ICD-10 diagnostic criteria for autistic disorder. In addition to single cutoff scores, the new algorithms for toddlers and young preschoolers provide clinicians and researchers with several different options for the diagnostic classification of young children. For clinical purposes, ranges of concern (little-to-no concern, mild-to-moderate concern, and moderate-to-severe concern) that represent the severity of autism symptoms in young children are also provided. A clinician or a researcher can

use these ranges of concern to inform decisions about whether or not a child should be followed up with further assessments or should be quickly referred for treatment services irrespective of diagnostic cutoffs. Scores that fall in the little-to-no range of concern indicate that the child is reported to have no more behaviors associated with ASD than children in the same age range who do not have ASD. On the contrary, a child who scores in the mild-to-moderate range has a number of behaviors consistent with, but perhaps not unique to, ASD. For clinical purposes, children in the mild-to-moderate or moderate-to-severe ranges of concern should receive further evaluation and follow-up, including other cognitive and language assessments, and recommendations for treatment. In addition to ranges of concern, single cutoff score can be used when more strictly stratified groupings are necessary, such as for intervention, neuroimaging, or genetic research. These different alternatives allow clinicians and researchers to be transparent about the choices they make, recognizing that diagnostic decisions about ASD in very young children are less stable and precise than for older children and adolescents.

In addition to the diagnostic algorithms, the ADI-R includes a current behavior algorithm form that can be used in clinical settings to assess changes that occur during or after interventions or that may reflect increasing developmental maturity or changing life circumstances. Because the current behavior algorithm form has not been empirically validated, it is not intended to be used as a diagnostic algorithm. The development of a new algorithm is underway by the authors in anticipation of an updated protocol and algorithm with new criteria. A shorter version of the ADI-R that can be used over the phone is also in the process of being developed and validated.

The ADI-R provides a useful structure to obtain history and understand a caregiver's perspective on his or her child's symptoms associated with ASD. However, it requires substantial practice to administer reliably, and it takes approximately 2–3 h to administer. The ADI-R should only be used by appropriately experienced clinicians who are familiar with ASD and relevant behaviors. Training workshops and videotapes are available to help clinicians and researchers

understand the scoring and administration of the ADI-R. For research use, interviewers must meet standards for reliability.

In a recent effort to identify children with ASD more efficiently, a brief parent interview, Autism Symptom Interview (ASI; 15–20 min), has been designed primarily as a case confirmation tool for ASD (Bishop et al. 2017). The ASI has been based on questions from the ADI-R. Based on school-age children ranging from 5–12 years of age, the verbal algorithm yielded a sensitivity of 0.87 (95% CI = 0.81–0.92) and a specificity of 0.62 (95% CI = 0.53–0.70). When used in conjunction with the ADOS, sensitivity and specificity were 0.82 (95% CI = 0.74–0.88) and 0.92 (95% CI = 0.86–0.96), respectively. Internal consistency and test-retest reliability were both excellent. Based on these results, the authors have concluded that particularly for verbal school age children, the ASI may serve as a useful tool to more quickly ascertain or classify children with ASD for research or clinical triaging purposes. Additional data collection is underway to determine the utility of the ASI in children who are younger and/or nonverbal.

See Also

- [Autism Diagnostic Observation Schedule](#)
- [Autism Diagnostic Observation Schedule \(ADOS\): Toddler Module](#)

References and Reading

- American Psychiatric Association [APA]. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Bishop, S. L., Huerta, M., Gotham, K., Alexandra Havdahl, K., Pickles, A., Duncan, A., . . . Lord, C. (2017). The autism symptom interview, school-age: A brief telephone interview to identify autism spectrum disorders in 5-to-12-year-old children. *Autism Research*, 10(1), 78–88.
- Dawson, G., Webb, S., Carver, L., Panagiotides, H., & McPartland, J. (2004). Young children with autism show atypical brain responses to fearful versus neutral facial expressions of emotion. *Developmental Science*, 7(3), 340–359.
- de Bildt, A., Sytema, S., Zander, E., Bölte, S., Sturm, H., Yirmiya, N., . . . Green, J. (2015). Autism diagnostic interview-revised (ADI-R) algorithms for toddlers and

- young preschoolers: Application in a non-US sample of 1,104 children. *Journal of Autism and Developmental Disorders*, 45(7), 2076–2091.
- DiLavore, P., Lord, C., & Rutter, M. (1995). The prelinguistic autism diagnostic observation schedule (PL-ADOS). *Journal of Autism and Developmental Disorders*, 25, 355–379.
- Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The autism diagnostic observation schedule (ADOS): Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders*, 37(4), 613–627.
- Kim, S., & Lord, C. (2011). New autism diagnostic interview-revised (ADI-R) algorithms for toddlers and young preschoolers from 12 to 47 months of age. *Journal of Autism and Developmental Disorders*, 42, 82. <https://doi.org/10.1007/s10803-011-1213-1>. Epub ahead of print retrieved July 29, 2011.
- Kim, S., & Lord, C. (2012). Combining information from multiple sources in the diagnosis of autism spectrum disorders using the new ADI-R algorithms for toddlers from 12 to 47 months of age. *Journal of Child Psychology and Psychiatry*, 53(2), 143–151.
- Kim, S. H., Thurm, A., Shumway, S., & Lord, C. (2013). Multisite study of new autism diagnostic interview-revised (ADI-R) algorithms for toddlers and young preschoolers. *Journal of Autism and Developmental Disorders*, 43(7), 1527–1538.
- Lainhart, J., Bigler, E., Bocain, M., Coon, H., Dinh, E., et al. (2006). Head circumference and height in autism: A study by the collaborative program of excellence in autism. *American Journal of Medical Genetics Part A*, 140(21), 2256–2274.
- Le Couteur, A., Rutter, M., Lord, C., Rios, P., Robertson, S., Holdgrafer, M., et al. (1989). Autism diagnostic interview: A semistructured interview for parents and caregivers of autistic persons. *Journal of Autism and Developmental Disorders*, 19, 363–387.
- Le Couteur, A., Lord, C., & Rutter, M. (2003). *Autism diagnostic interview-revised*. Los Angeles: Western Psychological Services.
- Le Couteur, A., Haden, G., Hammal, D., & McConachie, H. (2007). Diagnosing autism spectrum disorders in preschoolers using two standardised assessment instruments: The ADI-R and the ADOS. *Journal of Autism and Developmental Disorders*, 38(2), 362–372.
- Lord, C., Storoschuk, S., Rutter, M., & Pickles, A. (1993). Using the ADI-R to diagnose autism in preschoolers. *Infant Mental Health Journal*, 14(3), 234–252.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(5), 659–685.
- Lord, C., Rutter, M., DiLavore, P., & Risi, S. (1999). *Autism diagnostic observation schedule: Manual*. Los Angeles: Western Psychological Services.
- Lord, C., Luyster, R., Gotham, K., & Guthrie, W. J. (2000). *Autism diagnostic observation schedule-toddler module*. Los Angeles: Western Psychological Services.
- Risi, S., Lord, C., Gotham, K., Corsello, C., Chrysler, C., Szatmari, P., et al. (2006). Combining information from multiple sources in the diagnosis of autism spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45, 1094–1103.
- Rutter, M., Le Couteur, A., & Lord, C. (2003). *Autism diagnostic interview-revised*. Los Angeles: Western Psychological Services.
- Schopler, E., Reichler, R. J., & Renner, B. R. (1980). *The childhood autism rating scale (CARS)*. Los Angeles: Western Psychological Services.
- Ventola, P. E., Kleinman, J., Pandey, J., Barton, M., Allen, S., Green, J., et al. (2006). Agreement among four diagnostic instruments for autism spectrum disorders in toddlers. *Journal of Autism and Developmental Disorders*, 36(7), 839–847.
- Wiggins, L. D., & Robins, D. L. (2008). Excluding the ADI-R behavioral domain improves diagnostic agreement in toddlers. *Journal of Autism and Developmental Disorders*, 38(5), 972–976.
- World Health Organization [WHO]. (1990). *International classification of diseases (10th revision)*. Geneva: World Health Organization.

Autism Diagnostic Observation Schedule

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Synonyms

ADOS

Description

The Autism Diagnostic Observation Schedule (ADOS) is a semi-structured observation scale designed to observe social behavior and communication in children and adults referred for possible diagnosis of autism spectrum disorder (ASD). Originally developed as a research instrument, it became commercially available through Western Psychological Services in 2001 (Lord et al. 1999) and is used widely in clinical, school, community, and research settings. The goal of the ADOS is twofold: to help clinicians and researchers discriminate autism from other disorders and

typically developing individuals and to characterize social and communicative behaviors associated with autism (Lord et al. 1989). It is often used in conjunction with the Autism Diagnostic Interview-Revised (ADI-R; Rutter et al. 2003), a parent interview. When used by a skilled clinician, together, these two instruments form the “gold standard” for the diagnosis of ASD.

The format of the ADOS is unique. It is a structured interaction between an examiner and individual in which the examiner’s behavior is standardized using a hierarchy of structured and unstructured social behaviors. The examiner creates a “social world” in which occasions for specific behaviors are purposefully orchestrated in order to observe the presence – or absence – of an expected response. For example, with an older child or adult with fluent language, the examiner might initiate a conversation and observe whether the individual participates in a reciprocal exchange or asks about the examiner’s experiences. With a child or adolescent with limited language, the examiner might observe whether the individual conveys shared enjoyment in an activity, such as bubble play, by smiling, laughing, or requesting for the activity to continue. The ADOS goes beyond measuring the frequency of behaviors and also focuses on the *quality* of social behavior, allowing the examiner to make informed decisions regarding the presence of features associated with a diagnosis of ASD. Because of the movement between structured and unstructured tasks, and the need for keen observation within such tasks, it is imperative the ADOS is administered by a skilled examiner familiar with ASD.

The original version of the ADOS (Lord et al. 2000) consists of four modules based on age and language level, with “higher” modules generally requiring more language and social demands. Each module takes approximately 35–60 min to administer. Module 1 is for individuals with a minimum of no speech or the emergence of simple phrases. Module 2 is designed for individuals who use flexible three-word phrases, but are not yet speaking fluently. Modules 3 and 4 are for individuals with fluent speech. For the purposes of the ADOS, three-word phrases are defined as “regular

spontaneous meaningful use of three-word utterances including a verb,” while fluent speech is defined as “producing a range of flexible sentence types, providing language behavior the immediate context and describing logical connections within a sentence” (Lord et al. 1999).

Though each module of the ADOS has different language requirements, the overall format and structure is the same. In fact, there is considerable overlap of tasks across modules. In each module, the examiner interacts with the individual, administering a series of tasks, or “presses” for particular social behaviors. Modules 1 and 2 are conducted while moving around a room and include play-based tasks appropriate for young children or individuals with very limited language. Modules 3 and 4 generally take place while sitting at a table and include tasks involving more conversation.

Immediately after the administration of all tasks, the examiner rates the individual’s behavior on items across domains including communication, social interaction, play or imagination, and stereotyped behaviors and restricted interests. Ratings, or codes, are made on an ordinal scale from 0 to 3, with 0 indicating no evidence of abnormality related to autism and 3 indicating definite evidence, such that behavior interferes with interaction. Selected items from each domain are used to generate a diagnostic algorithm. These items were selected for their ability to discriminate between ASD and nonspectrum disorders and also for their relevance to DSM-IV and ICD-10 criteria. A classification of autism or non-autism ASD is made when thresholds on the social affect and restricted and repetitive behavior domains, and a combined social affect and restricted and repetitive behavior total, are exceeded. When combined with information from other sources, including but not limited to a parent interview and clinical judgment, an ADOS classification of autism or ASD may lead to a diagnosis on the spectrum.

Since its publication by WPS in 1999, the ADOS has expanded considerably. Revised algorithms for modules 1–3 were developed to improve the instrument’s sensitivity and specificity (Gotham et al. 2007), and a toddler module appropriate for children under 30 months old has

been available for research purposes (Luyster et al. 2009). The revised ADOS algorithms and the new toddler module were released commercially by WPS in 2012 in the second edition of the ADOS (ADOS-2; Lord et al. 2012a, b) (see Table 1 for a summary of ADOS algorithms). Adapted versions of modules 1 and 2 with modified tasks and materials are in development for adolescents and adults with limited language (Hus et al. 2011). The ADOS-Change (ADOS-C; Colombi et al. 2011), a measure using ADOS item descriptions with expanded codes ranging from 0 to 5, has also been created. This measure is scored by watching an unstructured interaction between an adult and child and will be used to measure response to intervention in young children.

Historical Background

The first version of the ADOS was developed primarily as a diagnostic research tool. Direct observation, in addition to observations in familiar settings and parent interviews, was an important part of diagnostic assessment, but such observations were not conducted in a standardized fashion across clinicians or patient. Furthermore, researchers needed a method in which to examine features specific to autism, such as impairments in social interaction and communication, independent of those accounted for by intellectual disability. A series of publications highlight the development of the ADOS from its first version to the significantly expanded versions in use today (Table 2).

The first ADOS (Lord et al. 1989) was intended for individuals between five and 12 years old, with an expressive language level of at least three years. It included only eight tasks, with two sets of materials based on developmental level and chronological age. The validation sample included 20 children and adolescents with autism and 20 children with intellectual disability matched for chronological age, verbal IQ, and gender. The measure showed promise in distinguishing children with autism from those with intellectual disability.

As public awareness of autism increased and more younger and nonverbal children were referred to clinics for diagnostic evaluations, there became a need to develop a “downward extension” of the ADOS that would be appropriate for younger children with no-phrase speech. The Pre-Linguistic Autism Diagnostic Observation Schedule (PL-ADOS; DiLavore and Lord 1995) was intended for children less than 6 years old with limited language. It included 12 tasks with 31 overall ratings. All tasks were administered in the context of play and were informed by the increasing amount of research on early indicators of autism, particularly those studies focusing on joint attention, functional and symbolic play, imitation, and early patterns of language development. The PL-ADOS was validated on a sample of 63 children with autism or developmental delay and matched for chronological age or language level. Overall, the algorithm was successful at differentiating autism from developmental delay, but its performance was not as good when discriminating verbal children with autism from nonverbal children with developmental delay, and children with autism who had some expressive language tended to be underclassified by the instrument.

The ADOS-Generic (ADOS-G; Lord et al. 2000) was developed directly from its original version (Lord et al. 1989) and the PL-ADOS (DiLavore and Lord 1995). It aimed to improve the tendencies to overdiagnose autism in children with insufficient language ability and underdiagnose children with higher language abilities. Furthermore, it sought to extend the current tasks to be appropriate for adolescents and adults. The ADOS-G differed from its predecessors in that it spanned a broader developmental and age range and was the first to introduce the use of modules across different developmental and language levels. It was also the first version to provide continuous scores from ASD to autism, thus making it applicable for children with broader ranges of social and communication impairments.

The ADOS-G was normed on a sample of 381 children, adolescents, and adults spanning a broader diversity of spectrum and nonspectrum disorders. The sample included a group of

Autism Diagnostic Observation Schedule, Table 1 ADOS algorithms

Age	Module T		Module 1	Module 1	Module 1	Module 2	Module 3	Module 4	Adapted module 1		Adapted module 2	
	No words	Some words	<5 words	Single words	Phrases	Fluent	Fluent	Fluent	No words	No words	Some words	
12–30 m	X											
21–30 m	X	X										
30–35 m			X	X	X		X					
3–4 years			X	X	X		X					
5–9 years			X	X	X		X					
10+ years			X	X	X		X	X	X		X	

Autism Diagnostic Observation Schedule, Table 2 History of the ADOS in JADD publications

Publication	Contribution
Autism Diagnostic Observation Schedule: A Standardized Observation of Communicative and Social Behavior (Lord et al. 1989)	First published version of the ADOS
The Pre-Linguistic Autism Diagnostic Observation Schedule (DiLavore and Lord 1995)	Introduction of alternate version of ADOS more appropriate for individuals with very limited language
The Autism Diagnostic Observation Schedule-Generic: A Standard Measure of Social and Communication Deficits Associated with the Spectrum of Autism (Lord et al. 2000)	Consolidation of ADOS and PL-ADOS Introduction of four module structure Appropriate for broader range of social communication deficits and age Accompanied by commercial release of ADOS by Western Psychological Services (Lord et al. 1999)
The Autism Diagnostic Observation Schedule: Revised Algorithms for Improved Diagnostic Validity (Gotham et al. 2007)	Revised algorithms for improved diagnostic validity Algorithms grouped by developmental and language ability Inclusion of restricted and repetitive behaviors in algorithm totals
The Autism Diagnostic Observation Schedule-Toddler Module: A New Module of a Standardized Diagnostic Measure for Autism Spectrum Disorders (Luyster et al. 2009)	Introduction of ADOS-Toddler Appropriate for use in children under 30 months with mental age of at least 12 months
Standardizing ADOS Scores for Measure of Severity in Autism Spectrum Disorders (Gotham et al. 2009)	Created standardized severity metric to measure change in ADOS assessments over time, age, and module

individuals diagnosed with autism, PDD-NOS, and a group designated as “nonspectrum,” which included individuals with diagnoses of mental retardation, language disorder, attention-deficit/hyperactivity disorder, oppositional defiant disorder, anxiety, depression, and obsessive-compulsive disorder and children who were typically developing. The ADOS-G algorithms were successful at discriminating ASD from nonspectrum, but were not as good at making distinctions between children with milder forms of ASD. Upon WPS publication of the ADOS-G in 1999, the “G” was dropped and the instrument became solely known as the ADOS. Gotham et al. (2007) and colleagues sought to improve the diagnostic validity of the ADOS by validating revised algorithms for modules 1–3 on a significantly larger sample of children with ASD and nonspectrum diagnoses. The new algorithms were grouped into developmental cells to reduce the effects of age and IQ and included more similar items across modules with the same number of items per algorithm to increase comparability. Factor analyses yielded two domains representing features of social affect and restricted and repetitive behaviors (RRBs);

thus, the new algorithms required thresholds to be met in social affect, RRB, and a combined total, in order to meet classification criteria for autism or ASD. This was a significant departure from earlier versions of the ADOS in which RRBs were not included on the algorithm and social interaction and communication were considered separately. Specificity in children with nonverbal mental ages of 15 months and younger continued to pose problems in distinguishing children with ASD from those with other language-based disorders or intellectual disability. Since the publication of the revised algorithms, however, several replications with larger and more diverse samples have been conducted with consistent results supporting the improved diagnostic validity of the new algorithms.

Though higher scores on the ADOS do indicate a greater number of behaviors consistent with core deficits of ASD and, to some degree, greater severity of impairment, ADOS scores were not standardized for this purpose. The creation of revised algorithms paved the way for the development of calibrated severity scores (Gotham et al. 2009). Severity scores that reduced the

effects of IQ and chronological age were developed to promote the comparison of ADOS assessments over time, age, and module and to identify trajectories of autism severity. Raw scores have been mapped onto a 10-point severity metric with lower scores indicating less autism impairment.

As calibrated severity scores were being developed, a new module of the ADOS, the ADOS-Toddler, was also underway. Advancements in the understanding of autism in very young children, particularly infants and toddlers, increased the need for diagnostic tools appropriate for use in that developmental level. Because the ADOS, even with revised algorithms, had limited applicability for children with nonverbal mental ages below 15 months, the toddler module was created. The toddler module consists of a combination of ADOS and some new tasks and is intended for use in children 12–30 months chronological age, with nonverbal mental ages of at least 12 months, and who are walking independently. It includes two algorithms, nonverbal 12–20 months/12–30 months and verbal 21–30 months. Because of the relative instability of diagnostic classifications in very young children, the toddler algorithms differ from those of the ADOS-G in two ways. First, they yield research classifications of ASD or nonspectrum and do not make distinctions between autism and ASD, and second, they provide clinical “ranges of concern,” (little-to-no, mild-to-moderate, and moderate-to-severe concern for ASD) indicating the degree of need for continued clinical monitoring.

The ADOS has developed considerably since the first 1989 version, and research on expanded applications of the instrument continues today. Continued testing of the ADOS is occurring in clinical and community-based settings, in addition to the application of translated versions for use in languages other than English.

Psychometric Data

Reliability. Across all ADOS modules, intraclass correlations for the social, communication, social communication, and restricted and repetitive domains were 0.93, 0.84, 0.92, and 0.82, respectively, and mean weighted kappas across items ranged from 0.65 to 0.78. Test-retest reliability ranged from 0.59 to 0.82. For the toddler module, intraclass correlation was 0.96 for the entire protocol and mean weighted kappa was 0.67. Test-retest reliability was 0.86 for the 12–20/21–30 nonverbal algorithm and 0.95 for verbal 21–30. Interrater reliability across all modules is reported in Table 3.

Diagnostic validity. Algorithm cutoffs for the ADOS were excellent for autism and ASD relative to nonspectrum disorders, with even greater performance with the introduction of revised algorithms. Algorithm cutoffs for the toddler module yielded high sensitivity and specificity. Sensitivities and specificities for current and revised algorithms of the ADOS are reported in Tables 4 and 5 and in Table 6 for the toddler module algorithms.

Clinical Uses

The ADOS is intended for use by clinicians familiar with autism. Valid administration and interpretation of results is dependent on the clinical skill of the examiner and requires substantial training. The ADOS can be used clinically upon completion of a two-day WPS-certified clinical course or from WPS training DVDs. Even with training, however, administration of the ADOS should not be attempted without significant practice in administering the tasks, in observing features of autism as specified by the ADOS items, and in

Autism Diagnostic Observation Schedule, Table 3 Interrater reliability: percent agreement

	Toddler ^a	Module 1 ^b	Module 2 ^b	Module 3 ^b	Module 4 ^b
Interrater (items)	84	91.5	89	88.2	88.3
Interrater (algorithm)	87	93	87	81	84

^aLuyster et al. 2009
^bLord et al. 2000

Autism Diagnostic Observation Schedule, Table 4 Sensitivities and specificities for current and revised ADOS algorithms: autism versus nonspectrum (Gotham et al. 2007)

	Current ADOS classification	Current ADOS classification	Revised ADOS classification	Revised ADOS classification
<i>N</i> = 1157	Se	Sp	Se	Sp
Mod 1, no words, nvma ≤15 AUT = 69 NS = 16	100	19	97	50
Mod 1, no words, nvma >15 AUT = 306 NS = 33	97	91	95	94
Mod 1, some words AUT = 201 NS = 76	88	96	97	91
Mod 2, younger AUT = 58 NS = 30	97	93	98	93
Mod 2, age 5+ AUT = 126 NS = 30	96	97	98	90
Mod 3 AUT = 129 NS = 83	86	89	91	84
Mod 4 AUT = 16 NS = 15	93	93	N/A	N/A

Autism Diagnostic Observation Schedule, Table 5 Sensitivities and specificities for current and revised ADOS algorithms: non-autism ASD versus nonspectrum (Gotham et al. 2007)

	Current ADOS classification	Current ADOS classification	Revised ADOS classification	Revised ADOS classification
<i>N</i> = 685	Se	Sp	Se	Sp
Mod 1, no words, nvma ≤15 ASD = 20 NS = 16	95	6	95	19
Mod 1, no words, nvma >15 ASD = 51 NS = 33	88	67	82	79
Mod 1, some words ASD = 75 NS = 76	67	84	77	82
Mod 2, younger ASD = 49 NS = 30	76	70	84	77
Mod 2, age 5+ ASD = 36 NS = 30	86	77	83	83
Mod 3 ASD = 186 NS = 83	68	77	72	76
Mod 4 ASD = 14 NS = 15	86	93	N/A	N/A

scoring. For those using the ADOS in research settings, more rigorous requirements for use exist. Individuals must attend a standardized training workshop and then obtain reliability with workshop leaders and within the research site. As

specified in Lord et al. (2000), research reliability is defined as agreement of 80% or above on ADOS protocols and algorithms on three consecutive scorings for modules 1 and 2 and modules 3 and 4, separately.

Autism Diagnostic Observation Schedule, Table 6 Sensitivities and specificities for toddler module algorithms: ASD versus nonspectrum (Luyster et al. 2009)

<i>N</i> = 234	Se	Sp
12–20/nonverbal 21–30 ASD = 87 NS = 64	87	86
Verbal 21–30 ASD = 59 NS = 24	81	83

Selecting the correct module for use in the ADOS is also crucial for obtaining an accurate classification. Clinicians and researchers can use the results of standardized tests or parent report to inform module choice, but as an individual’s language often varies in unstructured versus structured environments, the collection of a spontaneous language sample at the beginning of an ADOS administration is highly recommended. Administration of an “easier” module (e.g., selecting module 2 for a child with fluent speech because the tasks are “more fun” when module 3 would be more appropriate) can result in under classification. When in doubt, however, a clinician should adopt a conservative approach and chose a lower module as language difficulties may confound the social demands of a higher one.

Perhaps the most important practice in using the ADOS is to recognize its limitations. The ADOS is only one of multiple sources of information that should be considered when determining whether criteria for ASD are met. It is possible to meet classification thresholds on the ADOS algorithm and *not* meet formal criteria for an autism diagnosis. Conversely, a clinician with information from parent report and observations in different settings may assign a diagnosis of ASD even without an accompanying ADOS classification. The ADOS was developed as a companion instrument to the ADI-R, and indeed, both the ADOS and ADI yield higher sensitivities and specificities together than when used separately (Risi et al. 2006). In the hands of a skilled clinician with ample training and multiple sources of information, the ADOS provides a unique contribution to the observation of social and communicative features of autism and greatly aids in the diagnosis of ASD.

See Also

- ▶ [Autism Diagnostic Interview-Revised](#)
- ▶ [Autism Diagnostic Observation Schedule \(ADOS\): Toddler Module](#)
- ▶ [Prelinguistic Autism Diagnostic Observation Schedule](#)

References and Reading

Colombi, C., Carr, T., MacDonald, M., & Lord, C. (2011, March). *Developing a measure of treatment outcomes: The autism diagnostic observation schedule-change*. Poster presented at the Society for Research in Child Development Biennial Conference, Montreal.

DiLavore, P. C., & Lord, C. (1995). The pre-linguistic autism diagnostic observation schedule. *Journal of Autism and Developmental Disorders*, 25, 355–379.

Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The autism diagnostic observation schedule: Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders*, 37, 613–627.

Gotham, K., Pickles, A., & Lord, C. (2009). Standardizing ADOS scores for a measure of severity in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 693–705.

Hus, V., Maye, M., Harvey, L., Guthrie, W., Liang, J., & Lord, C. (2011, May). *The adapted ADOS – Preliminary findings using a modified version of the ADOS for adults who are nonverbal or have limited language*. Poster presented at the International Meeting for Autism Research, San Diego.

Lord, C., Rutter, M., Goode, S., Heemsbergen, J., Jordan, H., & Schopler, E. (1989). Autism diagnostic observation schedule: A standardized observation of communicative and social behavior. *Journal of Autism and Developmental Disorders*, 19, 185–212.

Lord, C., Rutter, M., DiLavore, P., & Risi, S. (1999). *Autism diagnostic observation schedule (ADOS)*. Los Angeles: Western Psychological Services.

Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Leventhal, B. L., DiLavore, P. C., Pickles, A., & Rutter, M. (2000). The autism diagnostic observation schedule – Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30, 205–223.

Lord, C., Luyster, R. J., Gotham, K., & Guthrie, W. (2012a). *Autism diagnostic observation schedule, (ADOS-2), Part II: Toddler module* (2nd ed.). Los Angeles: Western Psychological Services.

Lord, C., Rutter, M., DiLavore, P. C., Risi, S., Gotham, K., & Bishop, S. L. (2012b). *Autism diagnostic observation schedule, (ADOS-2), Part I: Modules 1–4* (2nd ed.). Los Angeles: Western Psychological Services.

- Luyster, R., Gotham, K., Guthrie, W., Coffing, M., Petrak, R., Pierce, K., et al. (2009). The Autism diagnostic observation schedule – Toddler module: A new module of a standardized diagnostic measure for Autism Spectrum Disorders. *Journal of Autism and Developmental Disorders*, 39, 1305–1320.
- Risi, S., Lord, C., Gotham, K., Corsello, C., Chrysler, C., Szatmari, P., Cook, E. H., Leventhal, B. L., & Pickles, A. (2006). *Journal of the American Academy of Child and Adolescent Psychiatry*, 45, 1094–1103.
- Rutter, M., Le Couteur, A., & Lord, C. (2003). *The autism diagnostic interview – revised (ADI-R)*. Los Angeles: Western Psychological Services.

Autism Diagnostic Observation Schedule (ADOS): Toddler Module

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Synonyms

ADOS-T

Description

The Autism Diagnostic Observation Schedule – Toddler Module (or ADOS-T; Luyster et al. 2009; Lord et al. 2012) – is a semi-structured assessment of social engagement, communication, and play using a set of planned “presses” within a naturalistic social interaction. It is intended for children under 30 months of age who have a nonverbal mental age of at least 12 months. Other guidelines for use include independent walking and minimal language; once the child masters three-word phrases, the Toddler Module is no longer considered appropriate.

Eleven activities are included in the Toddler Module, along with 41 overall codes. Two algorithms are associated with the module, including one for all children between 12 and 20 months of age and nonverbal children between 21 and

30 months of age and a second algorithm for verbal children between 21 and 30 months of age. These algorithms include formal cutoffs, which are primarily intended for research use and provide a binary classification of ASD or nonspectrum. Each algorithm also has three “ranges of concern,” which are intended for clinical use and provide three classifications of concern: little to no, mild to moderate, and moderate to severe. The Toddler Module can be administered in a professional’s office or playroom, although a familiar caregiver must be present. Codes are completed immediately after Toddler Module completion and are based on all behaviors during the administration. Each code can be scored between 0 and 3, with higher scores indicative of greater abnormality.

Historical Background

The Toddler Module was developed in response to a research and clinical need for a standardized instrument for use in very young children at high risk for, or suspected of having, an autism spectrum disorder (ASD). Research had indicated that the ADOS Module 1 was over-inclusive (meaning it exhibited relatively poor specificity) for children with nonverbal mental ages under 16 months (Gotham et al. 2007). The Toddler Module was developed for use in this very young population and was intended to aid in both clinical and research efforts targeted at children who fell below the floor of the ADOS.

The creation of the Toddler Module was based primarily on the Module 1 of the ADOS (Lord et al. 2000), which provides a series of semi-structured, play-based tasks and activities to probe for a range of behaviors. Module 1 items that were appropriate for infants and toddlers were included, and additional tasks were created based on a review of the literature on early social and communicative development. Some other important changes were made based on current knowledge of early development in children with ASD, including a shift from three classifications on the algorithm (autism, ASD, nonspectrum) to two

(ASD, nonspectrum), based on extensive evidence of the instability of specific diagnoses within the autism spectrum. For similar reasons, an emphasis was placed on using algorithm ranges of concern in order to encourage a focus on clinical monitoring and follow-up rather than assigning a formal diagnosis to a very young child.

Psychometric Data

Instrument development involved both validity and reliability studies (Lord et al. 2012). The validity study was completed using data from 182 children. Analyses were repeated using two overlapping samples, one of which included each child only once and a second that included multiple visits from some children. The final set of 41 codes was selected in order to yield markedly different distributions across diagnostic groups or to have high clinical or theoretical importance. In addition, codes were chosen in a manner that minimized collinearity with other codes or sample characteristics. Two algorithms were generated by selecting items that met theoretical and empirical thresholds for optimal group classification. Each algorithm includes items in two domains – social affect (SA) and restricted, repetitive behaviors (RRB) – and cutoff scores were selected based on maximal sensitivity and specificity. Using formal cutoffs, sensitivity and specificity exceeded 86% on the younger/nonverbal algorithm, and they exceeded 83% on the verbal algorithm.

The reliability study included ratings from 7 independent, “blind” raters on 14 Toddler Module administrations (8 from children with ASD, 3 from typically developing children, and 2 from children with non-ASD developmental disabilities, one child contributed two administrations). Inter-rater reliability was evaluated using weighted kappas for nonunique pairs of raters, with kappas between 0.4 and 0.74 considered good and kappas at or above 0.75 considered excellent. Three codes were not included in the reliability analyses because of limited variability; 30 codes had kappas equal to or above 0.60, and the remaining eight codes exceeded 0.45. Inter-

rater item reliability was measured using percent agreement and the full range of 0–3 scores: the mean percent agreement was 84%. All items exceeded 71%, and 30 of 41 items had exact agreement of at least 80%. Inter-rater agreement on the algorithms’ (younger/nonverbal and verbal) diagnostic cutoffs was 97% and 87%, respectively; inter-rater agreement for ranges of concern was 70% and 87%, respectively. Test-retest reliability was also satisfactory across both algorithms.

Note that although standardized calibrated severity scores are not available as a formal component of the instrument (Lord et al. 2012), research suggests that they may be helpful in reducing the effects of language level on algorithm totals (Esler et al. 2015).

Clinical Uses

Clinical usage of the Toddler Module should be accompanied by other sources of information. The ranges of concern may be useful in providing an indication of the degree to which a child is exhibiting symptoms consistent with an ASD, but in some cases, these behaviors may be attributable to other, non-ASD etiologies. Therefore, informed clinical judgment is critical in interpreting results within a broader developmental framework. Examining the profile of scores across the 41 codes may be useful in identifying areas of difficulty for the child and can help in education and intervention planning.

See Also

- [Prelinguistic Autism Diagnostic Observation Schedule](#)

References and Reading

- Esler, A. N., Bal, V. H., Guthrie, W., Wetherby, A., Weismer, S. E., & Lord, C. (2015). The autism diagnostic observation schedule, toddler module: Standardized severity scores. *Journal of Autism and Developmental Disorders*, 45(9), 2704–2720.

- Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The autism diagnostic observation schedule: Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders*, 37(4), 613–627.
- Lord, C., Luyster, R., Gotham, K., Guthrie, W., Risi, S., & Rutter, M. (2012). *Autism diagnostic observation schedule – toddler module manual*. Los Angeles: Western Psychological Services.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H. J., Leventhal, B. L., DiLavore, P., et al. (2000). The autism diagnostic observation schedule-generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30(3), 205–223.
- Luyster, R., Gotham, K., Guthrie, W., Coffing, M., Petrak, R., Pierce, K., et al. (2009). The autism diagnostic observation schedule-toddler module: A new module of a standardized diagnostic measure for autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(9), 1305–1320.

Autism Family Experience Questionnaire (AFEQ)

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Synonyms

Autism spectrum disorder (ASD); General health questionnaire-12 (GHQ-12); Pre-school autism communication trial (PACT trial); Pediatric autism communication therapy (PACT therapy); United kingdom (UK); Vineland adaptive behavior scales (VABS); Warwick-edinburgh mental wellbeing scale (WEMWBS)

Description

The Autism Family Experience Questionnaire (AFEQ; Leadbitter et al. 2018) is an ecologically valid questionnaire that measures the intervention priorities of parents/caregivers of children with autism spectrum disorder (ASD) and assesses the impact of interventions on family experience and quality of life. The AFEQ was developed in consultation with parents/caregivers of children with

ASD and was designed to address the paucity of outcome measures that assess parent/caregiver-nominated intervention outcomes for autistic children and their families (McConachie et al. 2015; Morris et al. 2014, 2015). It can be used for both research and clinical purposes.

The AFEQ has 48 items. Items are organized into four domains: (1) experience of being a parent of a child with autism (13 items); (2) family life (9 items); (3) child development (development, understanding, and social relationships; 14 items); and (4) child symptoms (feelings and behavior; 12 items). The questionnaire is designed to be self-rated by parents. Instructions are: “Please read each statement carefully and tick the box which you think best fits your feelings about you, your child with autism and your family life.” Items include both positively and negatively worded statements and are scored on an order scale: 1 = always to 5 = never, with an option for “not applicable”. Items that are negatively worded are reverse scored; individual missing data points can be prorated with the mean score of all items for that participant. The AFEQ produces a total score (range = 48–240) and domain scores which can be used to assess group differences or within-participant/within-group change over time. On the AFEQ a lower score indicates a positive outcome, and a higher score is a poor outcome. There are no clinical or case thresholds.

The AFEQ is being used as an outcome measure in current United Kingdom (UK) and international clinical trials. It is also being used in observational studies in several different countries. We invite other researchers and clinicians to utilize the AFEQ. A copy of the questionnaire can be accessed in open-access at: <https://link.springer.com/article/10.1007/s10803-017-3350-7#SupplementaryMaterial>. Scoring guidelines can be accessed directly from the author (Kathy.Leadbitter@manchester.ac.uk). The questionnaire has been translated from English into several other languages. Please contact the author for further information on available languages and/or the translation procedures. We ask that you appropriately cite our work and keep us informed about your research, its findings, and any planned publications or outputs relating to the AFEQ measure.

Historical Background

The AFEQ was developed prior to and in preparation for the UK Medical Research Council Preschool Autism Communication Trial (PACT Trial) as part of a broader strategy adopted by the trial team and funders to promote the involvement of service users in pretrial research design and out of the recognition that there was a measurement gap in relation to parent-generated outcome measures and assessments of family experience and child and family well-being. The PACT Trial was a two-arm parallel-group randomized controlled trial of Pediatric Autism Communication Therapy (PACT Therapy) – a parent-mediated video-aided communication-focused intervention for preschool children with ASD and their parents. The trial ran between 2006 and 2009 across three UK centers and evaluated the effectiveness of PACT therapy plus treatment-as-usual against treatment-as-usual alone in 152 preschool children with core autism (Green et al. 2010). A subsequent follow-up study assessed outcomes at 6 years after the end of the treatment phase (Pickles et al. 2016). The PACT Trial commissioning and protocol included a pre-specified strategy to develop a new parent-generated change measure of child and family well-being that could be used within the trial and suitable for future intervention research.

The AFEQ questionnaire was developed through three phases. Firstly, a series of pretrial focus groups was conducted with 31 parents of children with ASD to generate a core set of parameters that parents identified as the most important outcomes of a preschool intervention for ASD. Focus group transcripts were analyzed with thematic analysis. A set of 78 individual statements were abstracted from the thematic analysis themes to serve as response items within the draft questionnaire. Secondly, a large pretrial web-based consultation in collaboration with the UK National Autistic Society (www.autism.org.uk) subjected the 78 statements to wider review to evaluate the clarity and usefulness of the items. Using the data from the online consultation, low-performing items were discarded, resulting in a questionnaire with 56 well-performing items. This questionnaire was

rated by 152 parents as part of the baseline and 12-month follow-up assessments of the PACT Trial. Parents reported that they found the questionnaire easy to complete and that they valued the opportunity to report real-life experiences for their children and family on metrics nominated by other parents of autistic children. Following further data cleaning, an additional eight items were excluded as they generated too much missing data. The resulting 48-item questionnaire was named the Autism Family Experience Questionnaire. The 48-item AFEQ was then used in the PACT trial 6-year follow-up study.

A planned analysis within the PACT trial protocol was to use data from the trial to evaluate the psychometric properties of the AFEQ (see below; also Leadbitter et al. 2018). It was also planned to use the questionnaire as a trial outcome measure, to assess the estimation of treatment effect of PACT therapy over treatment-as-usual on parent-prioritized outcomes, family experience, and quality of life. On the 48-item AFEQ total score, there was a statistically significant improvement in the PACT group over the treatment-as-usual group at both trial endpoint (Cohen's $d = -0.29$) and at 6-year follow-up (Cohen's $d = -0.49$). There were also treatment effects at trial endpoint on two domain scores: the “experience of being a parent” domain and the “child development” domain. These findings provided evidence that the AFEQ total score, and to some extent its domain scores, were sensitive to change in response to a parent-mediated intervention for young children with ASD.

Psychometric Data

Descriptive data: In the validation cohort (PACT Trial sample – see above), the range of AFEQ total scores across three measurement timepoints was 64–196, with means of 132–141, medians of 133–141, and standard deviations of 21.3–24.6.

Internal consistency: The AFEQ has good internal consistency. To assess the internal consistency of the AFEQ, we examined the scale reliability based on Cronbach's alpha for the total score and domain scores, calculated from PACT

Trial baseline data. The total score and all domains demonstrated excellent levels of reliability: AFEQ total ($\alpha = 0.92$), parent (0.85), family (0.83), child development (0.81), and child symptoms (0.79).

Criterion validity: We assessed the external criterion validity of the AFEQ against (a) the parental Vineland Adaptive Behavior Scales (VABS; Second Edition; Sparrow et al. 2006), a well-validated parent-rated scale of child adaptive functioning; (b) the General Health Questionnaire-12 (GHQ-12; Goldberg 1992), a well-established measure of adult mental health; and (c) the Warwick-Edinburgh Mental Wellbeing Scale (WEMWBS; Tennant et al. 2007), a widely used measure of adult well-being. The correlations between the VABS total score and AFEQ child development domain score (items 23–36; 14 items) were moderate to strong across the three timepoints ($r = -0.48$ to -0.71 ; positive outcome indicated by a low score on the AFEQ and a high score on the VABS). The correlation between the parent domain score (items 1–13) and GHQ-12 total score was Spearman's $Rho = 0.408$ ($p < 0.001$, $n = 101$; a Spearman's rank correlation was conducted as the GHQ-12 distribution was highly positively skewed). The association between the parent domain score and the WEMWBS total score at trial follow-up was $r = -0.528$ ($p < 0.001$, $n = 103$). The AFEQ therefore showed good convergent validity with well-established measures of child adaptive functioning, parental mental health, and parental well-being.

The psychometric evidence is based on a sample of children with “core autism” aged 2–12 years. It is not yet known how the questionnaire would work outside of these parameters.

Clinical Uses

The AFEQ can be applied in a range of healthcare, child development, educational, or social care settings and developmental settings that support families of children with ASD and similar neurodevelopmental conditions. Its main use would be to make pre- and post-within-participant or within-group comparisons to evaluate the effect of an intervention on parent-nominated intervention

priorities, family experience, and quality of life. At the time of publication, it was in use in this way by several clinicians/clinical teams internationally. The AFEQ could also be used to quantify the experience of families and to make between-family comparisons, in order to identify families who are having a particularly difficult experience and who could benefit from further support.

See Also

- [Assessing Quality of Life in Autism](#)
- [Developmental Intervention Model](#)

References and Reading

- Goldberg, D. (1992). *General health questionnaire (GHQ-12)*. Windsor: Nfer-Nelson.
- Green, J., Charman, T., McConachie, H., Aldred, C., Slonims, V., Howlin, P., Le Couteur, A., Leadbitter, K., Hudry, K., Byford, S., Barrett, B., Temple, K., Macdonald, W., Pickles, A., & The PACT Consortium. (2010). Parent-mediated communication-focused treatment in children with autism (PACT): A randomised controlled trial. *The Lancet*, 375, 2152–2160.
- Leadbitter, K., Aldred, C., McConachie, H., Le Couteur, A., Kapadia, D., Charman, T., Macdonald, W., Salamone, E., Emsley, R., Green, J., & Consortium, T. P. A. C. T. (2018). The autism family experience questionnaire (AFEQ): An ecologically-valid, parent nominated measure of family experience, quality of life and prioritised outcomes for early intervention. *Journal of Autism and Developmental Disorders*, 48, 1052–1062.
- McConachie, H., Parr, J. R., Glod, M., Hanratty, J., Livingston, N., Oono, I. P., et al. (2015). Systematic review of tools to measure outcomes for young children with autism spectrum disorder. *Health Technology Assessment*, 19(41), 1–506.
- Morris, C., Janssens, A., Allard, A., Thompson-Coon, J., Schilling, V., Tomlinson, R., et al. (2014). Informing the NHS outcomes framework: Evaluating meaningful health outcomes for children with neurodisability using multiple methods including systematic review, qualitative research, Delphi survey and consensus meeting. *Health Services & Delivery Research*, 2(15).
- Morris, C., Janssens, A., Shilling, V., Allard, A., Fellowes, A., Tomlinson, R., et al. (2015). Meaningful health outcomes for paediatric neurodisability: Stakeholder prioritisation and appropriateness of patient reported outcome measures. *Health and Quality of Life Outcomes*, 13, 87.
- Pickles, A., Le Couteur, A., Leadbitter, K., Salamone, E., Cole-Fletcher, R., Tobin, H., Grammer, I., Lowry, J.,

- Vamvakas, G., Byford, S., Aldred, C., Slonims, V., McConachie, H., Howlin, P., Parr, J. R., Charman, T., & Green, J. (2016). Parent-mediated social communication therapy for young children with autism (PACT): Long-term follow-up of a randomised controlled trial. *The Lancet*, 388, 2501–2509.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2006). *Vineland adaptive behavior scales: Second edition*. Livonia: Pearson Assessments.
- Tennant, R., Hiller, L., Fishwick, R., Platt, S., Joseph, S., Weich, S., et al. (2007). The Warwick-Edinburgh mental Well-being scale (WEMWBS): Development and UK validation. *Health and Quality of Life Outcomes*, 5(1), 63.

Autism Family Experience Questionnaire (AFEQ), The

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Synonyms

Autism Spectrum Disorder (ASD); General Health Questionnaire (GHQ-12); Pre-school Autism Communication Therapy (PACT); Vineland Adaptive Behavior Scales (VABS); Warwick-Edinburgh Mental Wellbeing Scale (WEMWBS)

Description

The Autism Family Experience Questionnaire (AFEQ) is a measure developed to reflect the intervention priorities of parents of children with autism spectrum disorder (ASD), and to assess the impact of interventions on family experience and quality for life (The PACT Consortium et al. 2018).

The AFEQ consists of 48 items in the domains: (1) experience of being a parent of a child with autism; (2) family life, (3) child development (development, understanding, and social relationships); and (4) child symptoms (feelings and behavior) (The PACT Consortium et al. 2018). All items are scored on an order

scale: 1 = always to 5 = never, with an option for “Not Applicable.”

Historical Background

Quality of life has been widely studied; however, the research on quality of life for people with autism is scarce (Burgess and Gutstein 2007). The AFEQ was developed and tested within the large PACT intervention, Trial and Follow-Up (The PACT Consortium et al. 2018). The AFEQ is developed based on focus and online consultations with parents (The PACT Consortium et al. 2018).

In the focus group phase, 31 parents of pre-school or school-aged children with an autism diagnosis were recruited. The participants were recruited from both local clinical services and parent-support groups (The PACT Consortium et al. 2018). The participants attended one of five focus groups, convened and led by members of the PACT Principal Investigator team, and an independent qualitative researcher. The focus groups explored the specific parameters identified as the most important outcomes in a pre-school communication intervention for parents. A qualitative analysis of the focus groups resulted in 78 questionnaire items. Thirty-five parents rated the 78 questionnaire for clarity and usefulness online.

Based on the online rating, revised questionnaire with 56 items were applied with parents within the PACT trial. A further cleaning resulted in the 48-item questionnaire: the Autism Family Experience Questionnaire (AFEQ).

Psychometric Data

To date there is only one study exploring the psychometric data of the AFEQ (The PACT Consortium et al. 2018). The scale reliability of the AFEQ was initially examined based on 140 participants based on Cronbach's alpha for the domain scores and the total AFEQ score. All domains and the total score demonstrated excellent reliability: parent (alpha = 0.85), family

(0.83), child development (0.81), child symptoms (0.79), and total AFEQ (0.92) (The PACT Consortium et al. 2018).

External criterion validity was explored for the child development domain against parental Vineland Adaptive Behavior Scales (VABS). The correlation were significant ($r = -0.478$ to -0.710 , $p < 0.001$) and indicating a moderate to strong association between AFEQ and VABS at three different time-points (The PACT Consortium et al. 2018). The external criterion validity of the parent domain of the AFEQ was assessed against GHQ-12 and WEMWBS at one time-point. The correlation between the parent domain score and GHQ-12 was Spearman's $Rho = 0.408$ ($p < 0.001$) and with the WEMWBS was $r = -0.528$ ($p < 0.001$).

Clinical Uses

The AFEQ are used in the PACT 6-year follow-up study (Sigafoos and Waddington 2016) and may be used as described in the objective – to reflect the intervention priorities of parents of children with autism spectrum disorder (ASD) and to assess the impact of interventions on family experience and quality for life.

See Also

- [Assessing Quality of Life in Autism](#)
- [Parental Response to Diagnosis](#)
- [Quality of Life for Transition-Age Youth with ASD](#)
- [Social Validity](#)
- [Vineland III](#)

References and Reading

- Burgess, A. F., & Gutstein, S. E. (2007). Quality of life for people with autism: Raising the standard for evaluating successful outcomes. *Child and Adolescent Mental Health*, 12(2), 80–86. <https://doi.org/10.1111/j.1475-3588.2006.00432.x>.
- Sigafoos, J., & Waddington, H. (2016). 6 year follow-up supports early autism intervention. *The Lancet*,

388(10059), 2454–2455. [https://doi.org/10.1016/S0140-6736\(16\)31656-7](https://doi.org/10.1016/S0140-6736(16)31656-7).

The PACT Consortium, Leadbitter, K., Aldred, C., McConachie, H., Le Couteur, A., Kapadia, D., ... Green, J. (2018). The Autism Family Experience Questionnaire (AFEQ): An ecologically-valid, parent-nominated measure of family experience, quality of life and prioritised outcomes for early intervention. *Journal of Autism and Developmental Disorders*, 48(4), 1052–1062. <https://doi.org/10.1007/s10803-017-3350-7>

Autism in Ecuador

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Historical Background

As in other countries from the region, the field of mental health in Ecuador has been documented during pre-Columbian times, the colonial period, and the modern republics. Children and adults with autism spectrum disorder (ASD), especially those situated in the most severe end of the spectrum, may have received treatments available for individuals with atypical and challenging behaviors long before ASD was recognized and named. Different treatments used for mental health conditions have been documented since pre-Columbian times, such as the use of hallucinogens (ayahuasca, bejuco), animals (chickens, guinea pigs), songs, and dances with mythical and religious allusions (Naranjo 1983). In different parts of the country, it is still possible to observe practices of shamanism or crusaderism, offered to individuals with mental health difficulties (Zuniga Carrasco and Riera Recalde 2018), or read billboards advertising the services particularly for children's difficulties, mentioning autism among them. The first hospice and psychiatric asylum, Hospicio Jesus, María y José, was founded under the initiative of the Catholic Church in 1785 in the city of Quito, then a colonial center of the Spanish Crown. Operating on a Western prison model, this center had a

population of individuals suffering from mental health problems but also orphans and beggars (Landazuri 2008).

The birth of psychology in Ecuadorian academic circles is situated toward the end of the nineteenth century. The first chair of psychology was given in 1897 on subjects relating to hypnosis and suggestion by professors of general medicine. The lessons were addressed to teachers trained in philosophy and pedagogy, although the biological paradigm appeared to dominate. The first experimental studies took place shortly after, affirming psychology as a scientific area. The creation of various chairs of psychiatry in different cities succeeded until 1926, influenced by the conference "Psychology and Pedagogy," dictated at the first Ecuadorian Congress of Medicine in 1919 (Zuniga Carrasco and Riera Recalde 2018). By 2007, a total of 17 faculties of psychology had been created in different Ecuadorian universities. One of the most important, the Faculty of Psychological Sciences of the Central University of Ecuador, in Quito, was created in 1972 as a single school, covering four specializations: clinical psychology, special education and psycho-rehabilitation, industrial psychology, and legal psychology.

The first identifications of autism cases likely took place in the 1980s, in the context of psychiatric public and private professional practices, following official recognition of autism by the *Diagnostic and Statistical Manual of Mental Disorders*, third edition (DSM-III), in 1980 and under the influence of European or North American textbooks that university libraries possessed at that time. From the treatment point of view, psychotherapies and psychopharmacology, such as insulin shock therapy, were used for schizophrenia and related disorders from the 1950s (Aguilar 2013). In the 1960s, nongovernmental institutions were created in order to provide care for individuals with mental health conditions. The first organizations created with this purpose were religious, such as the Order of the Sisters of the Hospital, whose work was based on an agreement with the Ministry of Social Protection and Labor. Other nonprofit and for-profit laic foundations, set up to meet the needs of adults and children with

different types of health problems, appear in the following years. The first private centers intending to provide specific assistance to children with autism, by separating them from children with intellectual disabilities, appeared in the late 1980s (Aguirre et al. 2017 cited in Zuniga Carrasco and Riera Recalde 2018).

Currently, it is not possible to identify any specific programs for autism in the registers of public health agencies in Ecuador. The Ministry of Health published the first *Guide for Clinical Practice: Diagnosis, Treatment, Rehabilitation, and Case Management* in 2017. The Ecuadorian health system is based on public and private practices. The public system comprises two subsystems, hospitals, and state social security system institutions, on the one hand, and institutions dependent on the Ministry of Health, on the other. Two major pediatric hospitals, Hospital Baca Ortiz in Quito and Hospital Dr. Francisco de Icaza Bustamante in Guayaquil, depend on the Ministry of Health and provide general and specialized services. There are also private entities which operate in the public sector, such as the Welfare Board of Guayaquil (Hospital Dr. Robert Gilbert E. Guayaquil), the Child Protection Association of Guayaquil, and the Ecuadorian Red Cross. Public services are funded from the general state budget, extra-budgetary funds, and funds from national and international projects and agreements. Private services are funded by selling health-care services to the public sector; by private health insurers, mainly for the middle- and high-income population (Pan American Health Organization 2017); and by the families themselves. Services for people with autism are offered by pediatricians, neuro-pediatricians, psychiatrists, clinical psychologists, and educators, as well as speech and language therapists and occupational therapists, working in public or private centers and private practices but also in nonprofit foundations which are mainly located in the cities.

As it has been the case in other countries, parents' associations have contributed significantly to obtain recognition of ASD through public conferences and free-of-charge training to parents and teachers. In Quito, a group of parents met in 2012 to ask for official recognition of

autism as a handicap. Parents obtained access to a Disability Card (Carnet de Discapacidad) from the National Parliament, which allows individuals with autism to certain rights. In March 2013, those parents united into an association, APADA, aiming to contribute to the development of awareness programs and to work together with the Ministry of Education and the Ministry of Labor on special education plans. This association has also contributed to the development of the National Agenda for Equality in Disabilities 2017–2021, aiming to support the autonomy and productivity of people with disabilities. This agenda also examines how “to define a national instrument for the diagnosis of the Autism Spectrum” and “to implement the screening and diagnosis of the Autism Spectrum in the national territory” (National Council for Equality in Disabilities 2017, p. 54–55). Together with nine other parent associations, Guayaquil (3), Quito (1), Cuenca (1), Machala (1), Santo Domingo (1), Ibarra (1), Los Ríos (1), and Loja (1), it has formed the “Ecuadorian Federation of Autism Spectrum” (Organizaciones de Autismo buscan 2017). Their objective is to protect the well-being of individuals with ASD and their families, promote public policies, and support the work of other organizations. Other parents’ associations are currently being organized in different cities across the country.

Legal Issues, Mandates for Services

Ecuador is a member of the International Convention on the Rights of the Child (The United Nations 1989), which recognizes and protects access to health and education services, and was the 20th state to ratify the Convention on the Rights of Persons with Disabilities (The United Nations 2006) and its optional protocol that entered into force in 2008. The Ecuadorian State has transposed and clarified those rights in its national legislation. The Law on Disabilities (LOD) of the National Health System (National Assembly of the Republic of Ecuador 2012) guarantees the rights of people with ASD to free access to medicines and equipment, technical and

technological aids, adaptations of study plans, and permanent accompaniment of guides (LOD, art. 33). Other benefits, such as financing construction or remodeling housing and a reduction of 50% in the services of water and electricity, are considered by this law. In the field of education, an agreement intends to guarantee the access of individuals with special needs to special education (Ministerio de Educación 2013). In order to be eligible, individuals with ASD must request the Disability Card. Potential beneficiaries need to justify that they are suffering from a “non-evident and non-visible disability.” They also need to present a report from a medical practitioner or a specialist and the results of additional examinations, which may only be issued by the units of the Complementary and Integral Public Health Net (Ministry of Health n.d.). Autism is defined in this context as a “catastrophic disease,” namely, a pathology or chronic disease which poses a grave risk to the life of the person. Their treatment has a high economic cost and social impact, and, being of a prolonged or permanent nature, they must be part of long-term health plan and generally have little or no insurance coverage (Ministry of Health 2012a). In the particular case of individuals in this situation who are living in critical socioeconomic circumstances, this ministerial agreement contemplates the allocation of a monthly voucher of 240 USD under certain conditions. As described, an important legal framework intending to support the development of individuals with ASD exists in Ecuador. However, some critical gaps in terms of training and access to information on good practices exist, hindering the implementation of rules and policies (Educación Inclusiva en Ecuador hay ley 2019).

Overview of Current Treatments and Centers

Many different treatments are offered in public and private centers, as well as in private practices. They include a considerable variety of methods, such as Floortime, Tomatis, hippotherapy, and others, sometimes not specified. Speech therapies, sensory therapies, and cognitive-behavioral

therapies are also commonly offered. Specific evidence-based treatment services are still scarce. To date, two professionals are registered within the Behavior Analyst Certification Board as BCBAs, one in Quito and one in Guayaquil. Only one professional, in Quito, is currently registered on the official list of certified therapists of the ESDM model. TEACCH strategies and alternative/complementary systems of communication are also used within a variety of settings. Drug treatments, generally intended for comorbidities, are prescribed by pediatricians, neuropsychiatrists, and psychiatrists. The annex number ten of the Guide for *Clinical Practice: Diagnosis, Treatment, Rehabilitation, and Case Management* (Health Minister 2017) provides a list of medication endorsed by this document.

Overview of Research Directions

The document “Priority research areas 2013–2017” from the Ministry of Health defines a certain number of fields that had been chosen according to a list of health problems identified in official registers. Mental health issues are the 11th among 19 categories, in which autism and Asperger’s are considered (Ministry of Health 2012b). However, current research literature indicates that areas related to ASD have been underexplored and studies on prevalence at a national level have not yet been conducted. According to the Guide for Clinical Practice (Ministry of Health 2017), ASD prevalence in a child population of 5 years old or less was estimated to be 0.28% (0.18–0.41%) in 2015. According to data provided by the National Directorate of Disabilities of the Ministry of Health, in 2016, 1,266 people diagnosed with ASD were reported. Of these, 254 cases have been registered with a diagnosis of atypical autism, 792 with a diagnosis of childhood autism, 205 with Asperger syndrome, and 15 with Rett syndrome (as cited in Ministry of Health 2017, p. 11). The reasons why estimates of ASD prevalence in Ecuador are remarkably lower than those reported in Western countries remain unclear. As preliminary evidence, a study aiming to estimate school attendance of children with an

ASD diagnosis in the city of Quito found a proportion of 0.11% among 453 pupils in 161 in regular schools, assessed through interviews with school directors (Dekkers et al. 2015).

Preliminary research on the field of assistive technology has also been conducted in different universities. The research group in Artificial Intelligence and Assistive Technology of Salesian Polytechnic University of Ecuador (<https://www.ups.edu.ec/giata>) has carried out a pilot project aiming to explore the functionality of a mobile tool and a robotic assistant for the diagnosis and intervention of children with ASDs (Galán-Mena et al. 2016, June). A project intending to develop an application to support verbal communication and personal autonomy in children and young people has been carried out at the Universidad de las Fuerzas Armadas ESPE (Cárdenas et al. 2015, October).

A research project aiming to identify potential barriers to diagnosis in pediatric environments has been conducted in cooperation with the School of Pediatrics of the Faculty of Medicine at the Catholic University of Quito, the Faculty of Psychology at the University of Geneva, and the AJ Drexel Autism Institute in Philadelphia, with the endorsement of the Ecuadorian Society of Pediatrics (ESP). The results suggest that, as in many other countries, the pediatric community in Ecuador may be facing obstacles in terms of time for screening, training, and resources adapted to their clinical practices. The results also point to a low number of autism cases identified during the professional life of the participants (Buffle et al. 2019). An additional study, aiming to examine the pediatric community’s perception on screening procedures and tools, suggests a preference for observational procedures, over paper parent-administered questionnaires, as well as a clear interest among professionals in acquiring knowledge and expertise on the identification of early signs (Buffle and Gentaz 2019). A pilot project aiming to study the visual social attention with an eye-tracking is currently being conducted in neurotypical preschool age children in Quito. Eye-tracking measures are increasingly proposed as sensitive biomarkers for ASD, particularly convenient to assess the core social attention deficits

contributing to ASD. Remote eye gaze tracking is a noninvasive technique not requiring participants' overt responses and has not significant technical or ethical limitations (Frazier et al. 2018). Furthermore, measures can be rapidly collected across a wide range of ages and probably in different cultural settings. The present study aims to explore the adaptability of this technique and procedures to an Ecuadorian context (www.unige.ch/fapse/babylab/le-babylab/equipe/projet-en-equateur).

An overview of digital repositories from different universities in Ecuador shows an increasing interest among young bachelors in psychology and pedagogy in fields related to intervention in children with ASD, suggesting a potential for the development of new lines of research. An important research priority is the understanding of the cultural fit and adaptations required to implement evidence-based practices originating from the West (Vivanti 2019).

Overview of Training

Ecuadorian universities currently offer postgraduate studies in pediatrics and neurology. The specialization in neuro-pediatrics is not presently available, and this field relies on professionals trained in other countries who return to work in Ecuador. Currently, a training module for pediatricians and pediatric interns on ASD evidence-based practices is being developed in cooperation with the Department of Pediatrics of the Faculty of Medicine of the Catholic University of Quito (www.unige.ch/fapse/babylab/le-babylab/equipe/projet-en-equateur).

Aiming to facilitate the dissemination of scientific research in the field of ASD, the ESP included a session on "Validated screening tools in Spanish and their importance for a diagnostic process" during the 19th Ecuadorian Pediatric Congress in 2017. The newly created Ecuadorian Society of Neuropediatrics, supporting continuous education on early detection, included a session on "The challenges to diagnostic faced in pediatric settings and validated methods of intervention," during the First Ecuadorian Neuropediatric Congress in 2019. The ESP has also supported in February

2018 the organization of workshops intended for service providers on an evidence-based early intervention model in Quito, in collaboration with trainers certified from the UC Davis MIND Institute.

Education intended to service providers, such as speech therapists, psychologists, and occupational therapists, is primarily offered as a bachelor's degree at many official universities across the country. Training on specific topics has been provided within universities, such as an introductory module on ASD evidence-based practices addressed to students on special education of the Faculty of Education within the Universidad de las Américas in 2018. Several universities in major cities hold ad hoc conferences on ASD from a variety of theoretical backgrounds. Free-of-cost conferences organized by different parents' associations try to raise awareness among the general public and particularly among teachers.

Social Policy and Current Controversies

Current controversies include the terminology used for the diagnostic of ASD. Ecuador's public system mainly relies on the World Health Organization's International Classification of Diseases (ICD-10) (WHO 1993), one of the two official diagnostic systems. As ICD-10, which has conserved the traditional three categories dating back to Rutter's (1978) criteria, coexists with the autism spectrum disorder's description of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) (American Psychiatric Association 2013), confusion about the diagnostic is common among parents and individuals with ASD. Furthermore, some families strongly identify to their child's diagnosis of Asperger syndrome, perceived as less stigmatizing and more descriptive of milder conditions. For this reason, some professionals and parents oppose the disappearing of Asperger's diagnosis (APADA, personal communication, December 2, 2019).

Adapted and validated treatments are another important source of controversy. Media has become an authoritative source for families, and

it is quite common to observe parents requesting advice and recommendations through this channel. Furthermore, a miscellaneous offer of services, particularly for children, with sound, little, or no evidence, is advertised through the Internet. Some parents' associations, mainly based in Spain, provide information on good practices and are becoming well known among family circles in Ecuador (e.g., Autismo Diario). However, information about treatments that have little or no evidence (i.e., "Beware of non-evidence-based treatments," 2019) are still scarce in Spanish. This situation highlights the importance of making knowledge easily accessible for all families, independently of their socioeconomic status or level of education, in order to facilitate informed decisions.

Many other areas of controversy can be identified, such as the profile of professionals qualified to give a diagnosis and to carry out interventions. For the time being, multidisciplinary teams do not seem to be constituted at the public level. In private sectors, a common source of concern for families is related to situations where the professional giving a diagnosis of ASD also carries out an intervention with a method that is familiar to the professional but does not correspond to an individual's need, thus excluding other models or strategies of intervention that could be more suitable. Also, in the field of services, the impact of very short-term trainings open to the public and certifications on methods of evaluation and intervention that have significant variability in terms of duration, theoretical background, evidence, and professional supervision need to be studied. Finally, an important concern for families relates to the lack of visibility given to the needs of adults on the spectrum, especially in the cases when the level of functioning compromises autonomy. Those themes illustrate that an absolute priority must be to provide families an access to knowledge on good practices that enable informed decisions on the assessment, intervention, and support processes at each stage of their children's development. Attitudes toward individuals with ASD are changing as society becomes more aware of needs. Initiatives are no longer taken only by parents' associations but also by civil society, as suggested by the organization of a discussion

forum on the social integration of individuals with ASD, by an Ecuadorian NGO traditionally interested in sustainable development (Territorios Sostenibles, October 2019). The needs and constraints of individuals with ASD may be more visible nowadays. However, awareness among the general population specifically about symptoms' manifestation may still be frail. Indeed, early identification of young children requires families' participation in the decision process that leads to professional assistance. This process may not take place if autistic behaviors do not raise a certain level of concern among parents and professionals or if those behaviors are not understood as signs of a potential developmental disorder. In Ecuador, a study conducted on 183 adults concluded that most participants did not endorse many socio-communicative core symptoms as concerning enough to require professional assistance. Only language impairment and self-injurious behaviors attracted attention as concerning behaviors in young children by more than half of the respondents. On the other hand, most of the participants attributed the causes of autistic behaviors to factors unrelated to ASD or neurodevelopmental difficulties, such as child personality (Buffle et al. 2020). Those results suggest that "red flags" may not be recognized by families and non-trained professionals, which may lead to missing critical developmental opportunities. It also suggests that a substantial number of cases may remain invisible, preventing the estimation of individuals needing services in Ecuador.

References

- Aguilar, E. (2013). *Historia de la Psiquiatría y Salud Mental en el Ecuador [History of psychiatry and mental health in Ecuador]*. Quito: Ediciones Abya-Yala.
- Aguirre, R. V., Alemán, M. E., Alvarado, A. L., Borja, L. G., Camacho, L. M., Borja, L. T. (2017). *Manejo de la esquizofrenia en la atención primaria de salud*. Ediciones Médicas.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (DSM-5®). American Psychiatric Pub.

- Autism Science Foundation. (2019). Beware of off non-evidence-based treatments. Retrieved from <https://autismsciencefoundation.org/what-is-autism/beware-of-non-evidence-based-treatments/>.
- Buffle, P., & Gentaz, E. (2019, October). *Perception of autism screening tools in pediatric contexts: An exploratory study in an Ecuadorian sample*. Paper presented at the regional meeting of the International Society for Autism Research (INSAR) Puerto Varas, Chile.
- Buffle, P., Naranjo, A., Gentaz, E., & Vivanti, G. (2019, May). *Barriers to screening for Autism Spectrum disorders in pediatric settings in Ecuador*. Poster session presented at the International Society for Autism Research, Montreal.
- Buffle P., Vivanti G., & Gentaz, E. (2020). How are early signs of autism perceived and interpreted in Ecuador? Manuscript in preparation.
- Cárdenas, A., Segovia, E., Tobar, J., De la Cruz, D., Mejía, P., & Paredes, N. (2015, October). Pictoaprende: Application that contributes to the personal autonomy of children and youth with Autism Spectrum disorder in Ecuador. In 2015 Latin American Computing Conference (CLEI) (pp. 1–8). IEEE.
- Dekkers, L. M., Groot, N. A., Mosquera, E. N. D., Zúñiga, I. P. A., & Delfos, M. F. (2015). Prevalence of autism spectrum disorders in Ecuador: A pilot study in Quito. *Journal of autism and developmental disorders*, 45(12), 4165–4173.
- Educación inclusiva en Ecuador: Hay ley, pero falta formación. (2019, July 14). El Universo. Retrieved from <https://www.eluniverso.com/noticias/2019/07/23/nota/7438443/educacion-inclusiva-ecuador-hay-ley-falta-formacion>.
- Frazier, T. W., Klingemier, E. W., Parikh, S., Speer, L., Strauss, M. S., Eng, C., et al. (2018). Development and validation of objective and quantitative eyetracking-based measures of autism risk and symptom levels. *Journal of the American Academy of Child & Adolescent Psychiatry*, 57(11), 858–866.
- Galán-Mena, J., Ávila, G., Pauta-Pintado, J., Lima-Juma, D., Robles-Bykbaev, V., & Quisi-Peralta, D. (2016, June). An intelligent system based on ontologies and ICT tools to support the diagnosis and intervention of children with autism. In 2016 IEEE Biennial Congress of Argentina (ARGENCON) (pp. 1–5). IEEE.
- Landazuri, M. (2008). Salir del encierro, medio siglo del Hospital San Lázaro-Quito [Exit the confinement, half a century of the San Lázaro-Quito Hospital]. Retrieved December 15, 2019, from https://www.youtube.com/watch?time_continue=296&v=bGhYiagYQ6A&feature=emb_title
- Ministry of Education. (2013). Acuerdo 295–13 para Estudiantes con Necesidades Educativas Especiales [Agreement 295–13 on Students' Special Education needs]. Retrieved from https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&ved=2ahUKEwj2aD_rL_mAhWBpFkKHd_vDJYQFjAAegQIBRAC&url=https%3A%2F%2Feducacion.gob.ec%2Fwp-content%2Fuploads%2Fdownloads%2F2013%2F08%2FACUERDO_295-13.pdf&usq=AOvVaw0rTl2Zlac8yHXvK_Bd40UG.
- Ministry of Health. (2012a). Prioridades de investigación en salud 2013–2017 [Research priorities in health 2013–2017]. Retrieved from https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&cad=rja&uact=8&ved=2ahUKEwjopeertL_mAhUipFkKHciiAnwQFjAAegQIBRAC&url=https%3A%2F%2Fwww.investigacionsalud.gob.ec%2Fwp-content%2Fuploads%2F2019%2F03%2F1_l%25C3%25ADn eas_de_investigaci%25C3%25B3n_priorizadas_por_el_ministerio_de_salud_p%25C3%25BAblca0670108001551892114.pdf&usq=AOvVaw0mEnvTBXXoUWHDDP_asR1S.
- Ministry of Health. (2012b). Acuerdo Ministerial 1829. Emitense los criterios de inclusión de enfermedades consideradas catastróficas, raras y huérfanas para beneficiarios del bono Joaquín Gallegos Lara-M Ministerial [Agreement 1829. Issue the criteria for the inclusion of diseases considered catastrophic, rare and orphan for beneficiaries of the Joaquín Gallegos bond] Official registry 798.
- Ministry of Health. (2017). Guía Práctica Clínica. Trastornos del Espectro Autista en niños y adolescentes: Detección, diagnóstico, tratamiento, rehabilitación y seguimiento [Clinical Practice Guidelines. Autism Spectrum disorders in children and adolescents: Detection, diagnosis, treatment, rehabilitation and follow-up]. Report prepared for Ministry of Health, National Directorate of Normalization. Retrieved from <http://salud.gob.ec>.
- Ministry of Health. (n.d.). Calificación o recalificación de personas con discapacidad [Qualification or requalification of individuals with disabilities]. Retrieved from <https://www.salud.gob.ec/calificacion-o-recalificacion-de-personas-con-discapacidad-2/>.
- Naranjo, P. (1983). *Ayahuasca: Ethnomedicine and mythology*. Quito: Ediciones Libri Mundi.
- National Assembly of the Republic of Ecuador. (2012). Ley Orgánica de discapacidades [Law on disabilities]. Retrieved from https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&cad=rja&uact=8&ved=2ahUKEwifw5oqLibvMAhWypFkKHSyKCGIQFjAAegQIBhAC&url=https%3A%2F%2Fwww.consejodiscapacidades.gob.ec%2Fwp-content%2Fuploads%2Fdownloads%2F2014%2F02%2Fley_organica_discapacidades.pdf&usq=AOvVawIdDBPHgNGm_QMCBUfVVP4U.
- National Council for Equality in Disabilities. (2017). Agenda Nacional para la Igualdad de Discapacidades 2017–2021 [National Agenda for Equality in Disabilities 2017–2021]. Agenda prepared by the National Council for Equality in Disabilities.
- Pan American Health Organization. (2017). Salud en las Américas [Health in the Americas]. Retrieved from <https://www.paho.org/salud-en-las-americas-2017/?p=4272>.
- Redacción Médica. (2017, April 3). Organizaciones de Autismo buscan promover políticas públicas de atención [Autism organizations seek to promote public attention policies]. Retrieved from <https://www.redaccionmedica.ec/secciones/profesionales/federacion-ecuatoriana-de-autismo-promover-politicas-publicas-de-atencion-89944>.

- Rutter, M. (1978). Diagnosis and definition of childhood autism. *Journal of Autism and Childhood Schizophrenia*, 8(2), 139–161.
- The United Nations. (1989). Convention on the rights of the child. *Treaty Series*, 1577, 3.
- The United Nations. (2006). Convention on the rights of persons with disabilities. *Treaty Series*, 2515, 3.
- Vivanti, G. (2019). Towards a culturally informed approach to implementing autism early intervention: A commentary on Ramseur II et al., 2019. *Pediatric Medicine*, 2, 20.
- World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organization.
- World Health Organization. (1993). *The ICD-10 classification of mental and behavioural disorders: diagnostic criteria for research* (Vol. 2). World Health Organization.
- Zuniga Carrasco, D., & Riera Recalde, A. (2018). Historia de la salud mental en Ecuador y el rol de la Universidad Central del Ecuador, viejos paradigmas en una sociedad digitalizada [History of mental health in Ecuador and the role of the Central University of Ecuador, old paradigms in a digitalized society]. *Revista de la Facultad de Ciencias Médicas*, 43(1), 39–45.

Autism in Higher Education: Access, Challenges, and Support Strategies

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Background

The Interagency Autism Coordinating Committee identified the development of services to support transition to adulthood as its first objective within the domain of lifespan research (IACC 2019). Around 50,000 adolescents with autism spectrum disorder (ASD) enter adulthood each year (Shattuck et al. 2012), many of whom have both an interest in higher education and the intellectual

capacity to succeed academically. The developmental period bridging adolescence to adulthood is when multiple essential milestones are generally achieved, including increased independence, autonomy, and responsibility (Arnett 2000). Unfortunately, many youths with ASD either fail to meet typical adult developmental milestones or decline in functioning during late adolescence (Picci and Scherf 2014).

Young adults with ASD often experience lower quality of life than do age- and cognitive ability-matched healthy adults (Bishop-Fitzpatrick et al. 2017). They also face sustained challenges with living independently (Flynn and Healy 2012; Steinhausen et al. 2016) and finding and keeping gainful employment (Engstrom et al. 2003). One pathway to independence in adulthood and financial mobility is through higher education. Based on data from the Bureau of Labor Statistics, education predicts income and people who obtain bachelor's degrees, on average, earn more than those with just a high school diploma (BLS; Torpey 2018). As such, understanding how society, and especially those in mental health and higher education, can best support the needs of college-enrolled and college-bound people with ASD is important.

Transition from High School to College

Federal policies such as the No Child Left Behind Act of 2001 and the Individuals with Disabilities Education Improvement Act have greatly improved educational outcomes for students with ASD in elementary and secondary education (Smith 2005). But while these policy changes have provided more opportunity for students with ASD to pursue a college education, this is not necessarily a smooth or successful transition. Transitioning into college can be both thrilling and difficult for anyone (Shea 2019), but a growing body of research indicates that emerging adults with ASD face unique challenges that can impede access to higher education. Adolescents and emerging adults with ASD often exhibit underdeveloped independence, lagging interpersonal skills, and impaired ability to manage stress

(Elias et al. 2019; Elias and White 2018). Partly because of these factors, it is estimated that only 50% of young adults with ASD pursue a college degree (Taylor and Seltzer 2011), a rate that is lower than the U.S. average college enrollment rate of 70% (U.S. Dept. of Health and Human Services 2017). Prior research also indicates that college students feel more supported academically than socially (Cai and Richdale 2016), a trend which can lead colleges to overlook the social deficits inherent to ASD. However, despite these factors, colleges are still seeing a nationwide increase in applications from students with ASD, and more and more high school students with ASD decide to pursue postsecondary education every year (CITE).

Different College Pathways

Once an adult with ASD decides to go to college, several choices await them. The student must decide on a major, a living arrangement, and indeed the type of college or university they will apply to. These choices are important for all students to consider, especially so for students with ASD. There are multiple different pathways an adolescent with ASD could take, including attending vocational high school, studying at a community college, or pursuing a 4-year degree.

Firstly, where do students with ASD attend? Many students with ASD enroll in community colleges and local institutions. One study reported that around 80% of postsecondary students with ASD were in a community college for at least part of their education (Wei et al. 2014). Some professionals have recommended this pattern as beneficial for students with ASD. For example, according to Adreon and Durocher (2007), Lars Perner (2002) suggested that the increased personal attention found in community colleges can help a student with ASD adjust to the routines and responsibilities of college. Conversely, large universities provide far less opportunity to receive individualized attention, especially in large classes (Freedman 2010). It may also be worth considering what sort of programs colleges have to offer students with disabilities; for example,

Shmulsky et al. (2015) demonstrated that participation in a holistic transition program resulted in students with ASD having a higher first-year completion rate than unsupported typically developing students.

Secondly, what majors do students with ASD choose? Research on this topic is minimal, but studies on the majors chosen by students with ASD suggest that STEM majors are the most commonly chosen areas of study (Baron-Cohen et al. 2007; Fessenden 2013; Wei et al. 2013). In particular, computer science seems to be a popular area of study for these students (Wei et al. 2013). Further research is needed to better understand what students with ASD are interested in studying, so as to better-inform program administrators.

Finally, where do students with ASD choose to live? Some students commute while others live in campus housing. Students with ASD may choose to commute for several reasons. It may be due to family finances and commuting can prevent a family from being charged a housing fee by the college or university (Buescher et al. 2014; Shimabukuro et al. 2008; Wei et al. 2014). It may also be due to personal factors such as deficits in independent living skills (Elias and White 2018; Steinhausen et al. 2016; Van Hees et al. 2015). Conversely, if the student decides to live in campus housing, they may have a roommate. Studies have shown that certain factors in roommates, such as aloofness (preference of solitary activity and decreased social involvement) can play a key role in fostering good relationships with students with ASD, with phenotypes closer to the autism phenotype resulting in higher relationship satisfaction from both roommates (Faso et al. 2016).

Strengths of College Students with ASD

Students with ASD typically bring a variety of strengths with them to college. The prevalence of special abilities and talents among those with ASD has long been recognized (Asperger 1944; Kanner 1943). Around two-thirds of individuals with ASD are thought to possess special isolated skills (Meilleur et al. 2015) which can be

harnessed for success in higher education. Attention to detail, strong memory, adherence to rules and guidelines, passionate interests, and intense knowledge of a particular subject area are commonly noted strengths of these students (Anderson et al. 2017; Gobbo and Shmulsky 2014), as well as openness to feedback and suggestions (Elias et al. 2019). Other strengths which may prove useful in higher education include enhanced perceptual functioning (Motttron et al. 2006), superior pitch discrimination (Heaton et al. 2008), hyperlexia (Ostrolenk et al. 2017), mathematical/calculating skills (Howlin et al. 2009), as well as musical, artistic, and other abilities (Meilleur et al. 2015).

Previous research suggests individuals with ASD tend to do particularly well in STEM areas of study. These students often have a cognitive style that lends itself particularly well to STEM fields: an ability to observe, identify, construct, and apply logical rule-based systems of reasoning to explain the world around them (Cox et al. 2016), i.e., “systemizing” (Baron-Cohen 2009). It has even been suggested that those with ASD may have an innate predisposition for STEM with higher autism rates among children whose parents work in STEM fields (Baron-Cohen 1998; Baron-Cohen and Hammer 1997). Students with ASD may approach problems in science and engineering in unique ways, develop novel and divergent solutions, and show strength, resilience, and determination (Baron-Cohen 2009). Interestingly, students with ASD who pursue STEM fields tend to progress further in their education and are more likely to finish all 4 years or transfer from a community college into a 4-year college or university (Wei et al. 2014). As previously mentioned, college students with ASD are more likely than students in other disability categories, and students in general, to gravitate towards STEM fields, and the efforts of these students are a credit to their fields.

Challenges Experienced by College Students with ASD

Emerging adults with ASD may experience significant difficulties during their college career.

Social interactions, organization and time management, managing anxiety and depression, maintaining motivation, and sensory overload have all been noted as areas of both frequent and severe difficulty for many students with ASD (Alverson et al. 2015; Trembath et al. 2012; White et al. 2016). Loneliness and isolation are common problems for college students with ASD (Madriaga and Goodley 2010). Knowing where to meet other students with similar interests, initiating and maintaining conversations with classmates, and finding ways to connect with other students can all be challenging. Other common situations such as group projects, maintaining appropriate classroom behavior, and in-class debates all demand complex social skills which students with ASD may not have (Cullen 2015). By the students’ own admissions, these social needs often go under-supported (Cai and Richdale 2016).

Furthermore, the college environment is dramatically different from high school, particularly concerning changes in schedule and routine, self-autonomy, and the need for self-advocacy skills (Van Hees et al. 2015). Living on campus also may require negotiating with roommates and independently managing a range of personal responsibilities such as doing laundry, cleaning, self-care, and handling mealtimes. High schools often have a somewhat invisible support system where teachers, staff, and classmates know and understand the student with ASD. Particularly on a large campus, these invisible supports are difficult to replicate in a college environment.

Disclosure of ASD Diagnosis

An additional challenge for students with ASD in college is the issue of disclosure. Many students with a diagnosis of ASD choose not to disclose (Van Hees et al. 2015) for a variety of reasons, which may include relation to self-identity, expected benefits from disclosing, and previous experiences with disclosure. Many college students with disabilities may want to eliminate the label of being disabled to reset their social identity (Marshak et al. 2010). Data from the National

Longitudinal Transition Study-2 indicated that around 33% of students with ASD did identify as “disabled” (Shattuck et al. 2014). The colleges themselves may not do an adequate job of providing information to incoming students about the services and supports which may be available to them and how to navigate the disability services system.

Further complicating the situation, disclosure does not guarantee assistance. One study of disclosing students attending 2-year colleges found that less than half reported receiving any services or accommodations (Roux et al. 2015). Others have reported reluctance to disclose their ASD diagnosis until they encounter a significant problem or are unable to cope (e.g., Gobbo and Shmulsky 2014; Van Hees et al. 2015). Finally, unlike in high school, in college it is the student’s responsibility to take initiative and seek help. Students with ASD, who may have depended upon parents and teachers to set goals (Elias et al. 2019), may fail to access these resources simply because they are not used to doing so.

Attitudes Towards College Students with ASD

Attitudes among students, faculty, staff towards students with ASD are another area of concern. Research has shown a significant lack of knowledge and understanding of ASD among faculty and staff (Glennon 2016; Tipton and Blacher 2014), leading to frustration among faculty and inaccurate interpretations of inappropriate classroom behavior. These and other misconceptions about autism have led to stigmatization and exclusion of these students (Gillespie-Lynch et al. 2015; Gobbo and Shmulsky 2014; Schindler et al. 2015; Wenzel and Brown 2014).

What about students? Student responses vary and seem influenced by major and previous experience. Students often distance themselves from students with ASD (Gardiner and Iarocci 2014) but those more familiar with autism seem to be more accepting (Nevill and White 2011). Students studying engineering and physical sciences have

been shown to be more willing to interact with a student with ASD compared to those majoring in arts and social sciences (Nevill and White 2011). Inclusion in training programs presenting information about ASD has been shown to increase knowledge and decrease stigma among college students (Gillespie-Lynch et al. 2015), since understanding motivation for behavior can help reduce stigma (Butler and Gillis 2011). However, studies by both Gillespie and colleagues and by Matthews et al. (2015) found that training has a greater impact on behavior and cognitive attitudes towards individuals with ASD and less impact on the affect experienced by these trained students.

Related to this work, White et al. (2019) examined student knowledge and attitudes towards other college students with ASD, the underlying factors contributing to such attitudes, and whether attitudes changed over a 5-year period. While the later cohort had greater knowledge and more positive attitudes towards students with ASD, there was no significant relationship between knowledge and attitudes. Even after being presented with an accurate list of traits that might be seen in a student with ASD, students who had previously identified a higher number of aggressive or misleading traits still demonstrated less positive attitudes – their own beliefs still trumped new factual knowledge. These findings imply that despite increasing knowledge and understanding of ASDs in society, negative attitudes remain resistant to change.

Students who personally knew someone with ASD had more positive attitudes toward their peers with ASD, consistent with other research in this area (Gillespie-Lynch et al. 2015; Nevill and White 2011). Students who did not know someone with ASD were more likely to endorse inaccurate traits related to cognitive deficits, perhaps reflective of stereotypes about disability more broadly, which are often perpetuated by a lack of contact. The conclusion that knowledge about ASD does not necessarily mediate attitudes toward peers with ASD is consistent with much of the research on attitudes toward members of minority populations, including those with disabilities and mental health issues (Allport 1954; May 2012; McManus et al. 2011).

Strategies to Support College Students with ASD

With all of this in mind, how can we best support students with ASD? Given the potential for success among college students with ASD, formulating effective support strategies is a priority for an increasing number of higher education institutions. Existing supports available through university counseling centers and learning and academic support services including tutoring and advising are often helpful. Disability services offices also play an important role in setting up academic accommodations which, depending on a student's disabilities and eligibilities, might include extended time for exams, having a note-taker during class, or taking verbal exams (Egan and Giuliano 2009). However, barriers to accessing accommodations are multilayered, beginning with concerns of disclosure, as outlined above. There is a clear need to identify cost-effective programming that can be implemented with efficacy and which improve retention and success for students with ASD (Barnhill 2016).

Supports for Social Skills

As stated previously, college students with ASD often state that they are not receiving adequate social or educational support in this new setting (Cai and Richdale 2016). Students themselves seem willing to attend support groups where they can meet other students with an ASD diagnosis (Van Hees et al. 2015), although colleges offering such groups have reported concerns that students were not receptive to the group, did not show up at the expected time, or that breaking down social skills was not helpful (Barnhill 2016). While an increasing number of institutions are developing tailored support groups, mentoring programs, and special tutoring, empirical support for many of these activities is minimal (Gelbar et al. 2014), and it is not yet clearly understood what supports are most helpful for students with ASD (Cox et al. 2016). It is clear, however, that support for social difficulties is critical (White et al. 2016) and likely has major implications for retention and success.

In part to address these problems, Hillier et al. (2018a) provided a support group program, "Connections," for college students with ASD which had a broad curriculum addressing not only social skills but a range of other potential challenges including academic skills, time and stress management, managing group work, and future plans. Group members indicated significant reductions in loneliness and anxiety and increase in self-esteem at the end of the program. Focus groups were conducted to examine functional changes in academic and social skills, and to hear directly from students themselves, a notable gap in the literature focused on students with ASD (Cox et al. 2016; Gelbar et al. 2014). Five prominent themes were identified in the focus group analysis which reflected how the program had positively impacted participants' skills and coping: executive functioning; goal setting; academics and resources; stress and anxiety; and social. Given that college students typically make their own decisions regarding interventions and services they are willing to receive, the program's social validity was also assessed and participants indicated that the group was acceptable, socially relevant, and useful to them.

Mentoring Programs

Mentoring is a well-established strategy for improving outcomes for a broad range of populations: at-risk youth (Britner et al. 2006), foster children (Rhodes et al. 1999), workplace mentorship (Janssen et al. 2015), and individuals with disabilities (Daughtry et al. 2009). Increasing attention has been paid to the possibility of implementing mentoring programs in higher education institutions for students with ASD. While many factors affect a program's success (Rhodes and DuBois 2008), a meta-analysis of 73 youth mentoring studies identified "best practices" including ongoing training for mentors, recruiting mentors with a background in helping roles or professions, structured activities for mentors and youth, expectations for frequency of contact, mechanisms for support and involvement of parents, and monitoring of overall program implementation (DuBois et al. 2011).

Hillier et al. (2019a) reported on a mentoring program for college students with disabilities, the majority of whom had an ASD diagnosis. The program supported freshmen for one semester and consisted of one-on-one hour-long weekly meetings with a trained mentor, typically an upperclassman at the same school. A structured curriculum with weekly and monthly goals covered topics including socializing on campus, peer pressure, organization and time management, email etiquette, study strategies and academic performance, managing stress, self-care and anxiety, and establishing independence. Mentees were compared with a group of matched students who did not receive mentoring. Self-report measures and focus groups indicated that mentoring had the most impact in knowing how things work at the university, how to meet people on campus, and accessing supports. Although academic outcomes (retention rates, average number of credits earned each semester, and GPA) did not differ between the mentees and a matched comparison group, a follow-up study indicated that mentees continued to experience benefits one-year on. Further, mentors themselves also reported a range of benefits including enhanced interpersonal and communication skills, patience, and compassion, as well as educational benefits and skill development such as thinking on their feet, being supportive rather than condescending, and responding differently to different people based on their needs (Hillier et al. 2018b).

A similar program was implemented in Australia with a group of 10 university students with ASD (Siew et al. 2017). Students met for an hour each week with a trained graduate student and discussed topics based on the needs of the mentees which included time management, academic performance, and communication with professors and peers. Using a pre-post design and a range of self-report measures they found improved social supports and reduced general communication apprehension, although no significant change in anxiety, state communication apprehension, or participants' perceived communication competence. Qualitative analysis of semi-structured interviews indicated that participants appreciated the constant, stable support

from a peer, as well as the individualized nature of the support. Gillespie-Lynch et al. (2017) implemented a two-semester mentoring program for college students with ASD focused on social skills (semester one) and self-advocacy skills (semester two). Participants could access one-on-one mentoring with undergraduate or graduate students, and/or mentor-led group meetings. Again outcomes were positive indicating reduced anxiety and autism symptoms following the social skills training, and increased perceived social support from friends and heightened academic self-efficacy following the self-advocacy training. Mentoring programs focused on preparing for the transition to college prior to matriculation have also shown promise (Burgstahler and Cronheim 2001; Hillier et al. 2019b; Kim-Rupnow and Burgstahler 2004; Lindsay et al. 2016; Patrick and Wessel 2013), as well as summer transition programs specifically for incoming university students with ASD (Hotez et al. 2018; Lei et al. 2018).

Outcomes for College Students with ASD

What happens when students with ASD graduate? The first thing to note is that college is not "job-training," per se. It aims to impart a variety of soft skills, such as problem-solving and critical thinking (Arum and Roksa 2011; Huber and Kuncel 2016; McMillan 1987). This can be a problem for students with ASD, who tend to do better with clear and concrete instructions (Hollander et al. 2011; VanBergeijk et al. 2008), as individuals with ASD often tend to think concretely (Hobson 2012). This can lead to college graduates who are well-taught but who have not been prepared to begin and pursue a career. Job interviews tend to pose a particularly difficult challenge for would-be employees with ASD (Morgan et al. 2014). As nonverbal social behaviors have an impact on interview outcome (Ruben et al. 2015), as well as verbal factors such as prosody, turn-taking, and use of appropriate pauses (Morgan et al. 2014; Nguyen and Gatica-Perez 2015), individuals with ASD often experience difficulty during job interviews.

Despite the challenges, those with ASD who graduate college tend to have better outcomes than those who do not (Migliore et al. 2012; Sung et al. 2015). Participation in postsecondary education is in fact one of the strongest predictors of a good outcome for an adult with ASD (Migliore et al. 2012). Positive outcomes include better job placement, better job retention, and higher wages than their counterparts who do not attend university (Sung et al. 2015). Within college, students with ASD may report high GPAs (Gelbar et al. 2015). Furthermore, as noted by Taylor and Seltzer (2011), college students with ASD sometimes work in addition to studying, meaning that many graduate with not only a degree but with some work experience. Finally, many colleges have career development offices which provide services meant to help students with these “hard skills” needed to earn and keep a job. They teach students how to write a good resumé and offer practice job interviews (Crosby 2009). Some, such as Purdue University’s “Career Closet,” even loan formal interview clothing to students and give fashion advice (“What We Offer,” 2019). These offices and services can prove critical to persons with ASD by teaching the “hard skills” necessary to gain a job (Dey and Cruzvergara 2014).

Future Directions

In summary, an ever-increasing number of individuals with ASD are entering postsecondary education. Researchers and college administrators know enough about these individuals as a group to create services and programs designed to help students with ASD succeed in spite of challenges they may experience. High schools, especially those which tend to send many students to postsecondary education, can also help students with ASD prepare for the transition to college. However, further research is needed to better understand this population. Future work could gather data directly from students with ASD themselves as few studies have asked what they think is effective for preparing to transition and strategies to promote success once matriculated. Although

we have come a long way in recent years, more work is needed to understand the experiences of college students with ASD and the factors that contribute to positive outcomes.

References and Reading

- Adreon, D., & Durocher, J. S. (2007). Evaluating the college transition needs of individuals with high-functioning autism Spectrum disorders. *Intervention in School and Clinic*, 42(5), 271–279.
- Allport, G. W. (1954). *The nature of prejudice*. Cambridge/Reading: Addison-Wesley.
- Alverson, C. Y., Lindstrom, L. E., & Hirano, K. A. (2015). High school to college: Transition experiences of young adults with autism. *Focus on Autism and Other Developmental Disabilities*, 34(1), 1–13.
- Anderson, A. H., Stephenson, J., & Carter, M. (2017). Review: A systematic literature review of the experiences and supports of students with autism spectrum disorder in post-secondary education. *Research in Autism Spectrum Disorders*, 39, 33–53. <https://doi.org/10.1016/j.rasd.2017.04.002>.
- Amett, J. J. (2000). Emerging adulthood: A theory of development from the late teens through the twenties. *American Psychologist*, 55(5), 469–480.
- Arum, R., & Roksa, J. (2011). *Academically adrift: Limited learning on college campuses*. Chicago: University of Chicago Press.
- Asperger, H. (1944). Autistic psychopathy in childhood. In U. Frith (Ed.), *Autism and asperger syndrome*. Translated and annotated by U. Frith (1991) (pp. 37–92). Cambridge: Cambridge University Press.
- Barnhill, G. P. (2016). Supporting students with Asperger syndrome on college campuses current practices. *Focus on Autism and Other Developmental Disabilities*, 31, 3–15.
- Baron-Cohen, S. (1998). Does autism occur more often in families of physicists, engineers and mathematicians? *Autism*, 2(3), 296–301.
- Baron-Cohen, S. (2009). Autism: The empathizing–systemizing (E-S) theory. *Annals of the New York Academy of Science*, 1156, 68–80.
- Baron-Cohen, S., & Hammer, J. (1997). Parents of children with Asperger syndrome: What is the cognitive phenotype? *Journal of Cognitive Neuroscience*, 9(4), 548–554.
- Baron-Cohen, S., Wheelwright, S., Burtenshaw, A., & Hobson, E. (2007). Mathematical talent is linked to autism. *Human Nature*, 18(2), 125–131.
- Bishop-Fitzpatrick, L., Mazefsky, C. A., & Eask, S. M. (2017). The combined impact of social support and perceived stress on quality of life in adults with autism spectrum disorder and without intellectual disability. *Autism*, 22(6), 703–711.
- Britner, P. A., Balcazar, F. E., Blechman, E. A., Blinn-Pike, L., & Larose, S. (2006). Mentoring special youth

- populations. *Journal of Community Psychology*, 34, 747–763.
- Buescher, A. V. S., Cidav, Z., Knapp, M., & Mandell, D. S. (2014). Costs of autism Spectrum disorders in the United Kingdom and the United States. *JAMA Pediatrics*, 168(8), 721–728.
- Burgstahler, S., & Cronheim, D. (2001). Supporting peer–peer and mentor–protégé relationships on the internet. *Journal of Research on Technology in Education*, 34(1), 59–74.
- Butler, R. C., & Gillis, J. M. (2011). The impact of labels and behaviors on the stigmatization of adults with Asperger's disorder. *Journal of Autism and Developmental Disorders*, 41, 741–749.
- Cai, R. Y., & Richdale, A. L. (2016). Educational experiences and needs of higher education students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46, 31–41.
- Cox, B. E., Thompson, K., Anderson, A., Mintz, A., Locks, T., Morgan, L., Edelstein, J., & Wolz, A. (2016). College experiences for students with autism spectrum disorder (ASD): Personal identity, public disclosure, and institutional support. *Journal of College Student Development*, 58, 71–87.
- Crosby, O. (2009). *Resumes, applications, and cover letters* (Occupational Outlook Quarterly, Vol. 53, pp. 18–29).
- Cullen, J. A. (2015). The needs of college students with autism spectrum disorders and Asperger's syndrome. *Journal of Postsecondary Education and Disability*, 28(1), 89–101.
- Daughtry, D., Gibson, J., & Abels, A. (2009). Mentoring students and professionals with disabilities. *Professional Psychology: Research and Practice*, 40(2), 201–205.
- Dey, F., & Cruzvergara, C. Y. (2014). Evolution of career Services in Higher Education. *New Directions for Student Services*, 2014(148), 5–18.
- DuBois, D. L., Portillo, N., Rhodes, J. E., Silverthorn, N., & Valentine, J. C. (2011). How effective are mentoring programs for youth? A systematic assessment of the evidence. *Psychological Science in the Public Interest*, 12(2), 57–91.
- Egan, P. M., & Giuliano, T. A. (2009). Unaccommodating attitudes: Perceptions of students as a function of academic accommodation use and test performance. *North American Journal of Psychology*, 11(3), 487–500.
- Elias, R., & White, S. W. (2018). Autism Goes to college: Understanding the needs of a student population on the rise. *Journal of Autism and Developmental Disorders*, 48(3), 732–746.
- Elias, R., Muskett, A. E., & White, S. W. (2019). Educator perspectives on the postsecondary transition difficulties of students with autism. *Autism*, 23(1), 260–264.
- Engstrom, I., Ekstrom, L., & Emilsson, B. (2003). Psychosocial functioning in a group of Swedish adults with Asperger syndrome or high-functioning autism. *Autism*, 7(1), 99–110.
- Faso, D. J., Corretti, C. A., Ackerman, R. A., & Sasson, N. J. (2016). The broad autism phenotype predicts relationship outcomes in newly formed college roommates. *Autism*, 20(4), 412–424.
- Fessenden, M. (2013). Students with autism gravitate towards STEM majors. *Nature*.
- Flynn, L., & Healy, O. (2012). A review of treatments for deficits in social skills and self-help skills in autism spectrum disorder. *Research in Autism Spectrum Disorders*, 6(1), 431–441.
- Freedman, S. (2010). *Developing college skills in students with autism*. London: Jessica Kingsley Publishers.
- Gardiner, E., & Iarocci, G. (2014). Students with autism spectrum disorder in the university context: Peer acceptance predicts intention to volunteer. *Journal of Autism and Developmental Disorders*, 44(5), 1008–1117.
- Gelbar, N. W., Smith, I., & Reichow, B. J. (2014). Systematic review of articles describing experience and supports of individuals with autism enrolled in college and university programs. *Journal of Autism and Developmental Disorders*, 44, 2593–2601.
- Gelbar, N. W., Shefcyk, A., & Reichow, B. (2015). A comprehensive survey of current and former college students with autism spectrum disorders. *Yale Journal of Biology and Medicine*, 88(1), 45–68.
- Gillespie-Lynch, K., Brooks, P. J., Someki, F., Obeid, R., Shane-Simpson, C., Kapp, S. K., & Smith, D. S. (2015). Changing college students' conceptions of autism: An online training to increase knowledge and decrease stigma. *Journal of Autism and Developmental Disorders*, 45(8), 2553–2566.
- Gillespie-Lynch, K., Bublitz, D., Donachie, A., Wong, V., Brooks, P. J., & D'Onofrio, J. (2017). "For a long time our voices have been hushed": Using student perspectives to develop supports for neurodiverse college students. *Frontiers in Psychology*, 8, 544.
- Glennon, T. J. (2016). Survey of college personnel: Preparedness to serve students with autism spectrum disorder. *American Journal of Occupational Therapy*, 70(2), 7002260010.
- Gobbo, K., & Shmulsky, S. (2014). Faculty experience with college students with autism Spectrum disorders: A qualitative study of challenges and solutions. *Focus on Autism and Other Developmental Disabilities*, 29(1), 13–22.
- Heaton, P., Williams, K., Cummins, O., & Happe, F. (2008). Autism and pitch processing splinter skills: A group and subgroup analysis. *Autism*, 12, 203–219.
- Hillier, A., Goldstein, J., Murphy, D., Trietsch, R., Keeves, J., Mendes, E., & Queenan, A. (2018a). Supporting university students with autism spectrum disorder. *Autism: International Journal of Research and Practice*, 22(1), 20–28.
- Hillier, A., Goldstein, J., Tornatore, L., Byrne, E., Ryan, J., Johnson, H. M., Ratliff, S., Silva, K., & Donnelly, S. M. (2018b). Mentoring college students with autism spectrum disorder: Experiences of the mentors. *International Journal of Mentoring and Coaching in Education*, 7(3), 202–218.

- Hillier, A., Goldstein, J., Tornatore, L., Byrne, E., Diaz, A., Johnson, H. M., Ratliff, S., Silva, K., & Donnelly, S. M. (2019a). Outcomes of a peer mentoring program for university students with disabilities. *Mentoring and Tutoring: Partnership in Learning*. <https://doi.org/10.1080/13611267.2019.1675850>
- Hillier, A., Ryan, J., Donnelly, S., & Buckingham, A. (2019b). Peer mentoring program to prepare high school students with autism spectrum disorder for college. *Advances in Neurodevelopmental Disorders*, 3(4), 411–422.
- Hobson, R. P. (2012). Autism, literal language and concrete thinking: Some developmental considerations. *Metaphor and Symbol*, 27(1), 4–21.
- Hollander, E., Kolevzon, A., & Coyle, J. T. (Eds.). (2011). *Textbook of autism Spectrum disorders*. Arlington: American Psychiatric Publishing.
- Hotez, E., Shane-Simpson, C., Obeid, R., DeNigris, D., Siller, M., Costikas, C., Pickens, J., Massa, A., Giannola, A., D'Onofrio, J., & Gillespie-Lynch, K. (2018). Designing a summer transition program for incoming and current college students on the autism spectrum: A participatory approach. *Frontiers in Psychology*, 9(46), 1–16. <https://doi.org/10.3389/fpsyg.2018.00046>.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2009). Savant skills in autism: Psychometric approaches and parental reports. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 364(1522), 1359–1367.
- Huber, C. R., & Kuncel, N. R. (2016). Does college teach critical thinking? A meta-analysis. *Review of Educational Research*, 86(2), 431–468.
- Janssen, S., van Vuuren, M., & de Jong, M. D. T. (2015). Informal mentoring at work: A review and suggestions for future research. *International Journal of Management Reviews*, 00, 1–20.
- Kanner, L. (1943). Autistic disturbance of affective contact. *Nervous Child*, 2, 217–250.
- Kim-Rupnow, W. S., & Burgstahler, S. (2004). Perceptions of students with disabilities regarding the value of technology-based support activities on postsecondary education and employment. *Journal of Special Education Technology*, 19(2), 43–56.
- Lei, J., Calley, S., Brosnan, M., Ashwin, C., & Russell, A. (2018). Evaluation of a transition to university programme for students with autism Spectrum disorder. *Journal of Autism and Developmental Disorders*, 1–15.
- Lindsay, S., Hartman, R. L., & Fellin, M. (2016). A systematic review of mentorship programs to facilitate transition to post-secondary education and employment for youth and young adults with disabilities. *Disability and Rehabilitation*, 38(14), 1329–1349.
- Madriaga, M., & Goodley, D. (2010). Moving beyond the minimum: Socially just pedagogies and Asperger's syndrome in UK higher education. *International Journal of Inclusive Education*, 14(2), 115–131.
- Marshak, L., Van Wieren, T., Ferrell, D. R., Swiss, L., & Dugan, C. (2010). Exploring barriers to college student use of disability services and accommodations. *Journal of Postsecondary Education and Disability*, 22(3), 151–165.
- Matthews, N. L., Ly, A. R., & Goldberg, W. A. (2015). College students' perceptions of peers with autism Spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(1), 90–99.
- May, C. (2012). An investigation of attitude change in inclusive college classes including young adults with an intellectual disability. *Journal of Policy and Practice in Intellectual Disabilities*, 9(4), 240–246.
- McManus, J. L., Feyes, K. J., & Saucier, D. A. (2011). Contact and knowledge as predictors of attitudes toward individuals with intellectual disabilities. *Journal of Social and Personal Relationships*, 28(5), 579–590.
- McMillan, J. H. (1987). Enhancing college students' critical thinking: A review of studies. *Research in Higher Education*, 26(1), 3–29.
- Meilleur, A. A. S., Jelenic, P., & Mottron, L. (2015). Prevalence of clinically and empirically defined talents and strengths in autism. *Journal of Autism and Developmental Disorders*, 45(5), 1354–1367.
- Migliore, A., Timmons, J., Butterworth, J., & Lugas, J. (2012). Predictors of employment and postsecondary education of youth with autism. *Rehabilitation Counseling Bulletin*, 55(3), 176–184.
- Morgan, L., Leatzow, A., Clark, S., & Siller, M. (2014). Interview skills for adults with autism spectrum disorder: A pilot randomized controlled trial. *Journal of Autism and Developmental Disorders*, 44(9), 2290–2300.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36(1), 27–43.
- Nevill, R. A., & White, S. W. (2011). College students' openness toward autism spectrum disorders: Improving peer acceptance. *Journal of Autism and Developmental Disorders*, 41(12), 1619–1628.
- Nguyen, L. S., & Gatica-Perez, D. (2015). I would hire you in a minute: Thin slices of nonverbal behavior in job interviews. *Proceedings of the 2015 ACM on International Conference on Multimodal Interaction*, 51–58.
- Ostrolenk, A., d'Arc, B. F., Jelenic, P., Samson, F., & Mottron, L. (2017). Hyperlexia: Systematic review, neurocognitive modelling, and outcome. *Neuroscience & Biobehavioral Reviews*, 79, 134–149.
- Patrick, S., & Wessel, R. D. (2013). Faculty mentorship and transition experiences of students with disabilities. *Journal of Postsecondary Education and Disability*, 26(2), 105–118.
- Perner, L. (2002). Preparing to be nerdy where nerdy can be cool: College planning for the high functioning student with autism. Paper presented at the Autism Society of America Annual Conference, Indianapolis, IN.
- Picci, G., & Scherf, K. S. (2014). A two-hit model of autism: Adolescence as the second hit. *Clinical Psychological Science*, 3(3), 349–371.

- Rhodes, J. E., & DuBois, D. L. (2008). Mentoring relationships and programs for youth. *Current Directions in Psychological Science*, 17(4), 254–258.
- Rhodes, J. E., Haight, W. L., & Briggs, E. C. (1999). The influence of mentoring on the peer relationships of foster youth in relative and non-relative care. *Journal of Research on Adolescence*, 9, 185–201.
- Roux, A.M., Shattuck, P.T., Rast, J.E., Rava, J.A., Edwards, A.D., Wei, X., McCracken, M., & Yu, J.W. (2015). Characteristics of two-year college students on the autism spectrum and their support services experiences. *Autism Research and Treatment*, 2015, Article ID 391693, 10 pages.
- Ruben, M. A., Hall, J. A., & Mast, M. S. (2015). Smiling in a job interview: When less is more. *The Journal of Social Psychology*, 155(2), 107–126.
- Schindler, V., Cajiga, A., Aaronson, R., & Salas, L. (2015). The experience of transition to college for students diagnosed with Asperger's disorder. *The Open Journal of Occupational Therapy*, 3(1), 2.
- Shattuck, P. T., Narendorf, S. C., Cooper, B., Sterzing, P. R., Wagner, M., & Taylor, J. L. (2012). Post-secondary education and employment among youth with an autism spectrum disorder. *Pediatrics*, 129(6), 1042–1049.
- Shattuck, P. T., Steinberg, J., Yu, J., Wei, X., Cooper, B. P., Newman, L., & Roux, A. M. (2014). Disability identification and self-efficacy among college students on the autism spectrum. *Autism Research and Treatment*. Available at <https://doi.org/10.1155/2014/924182>
- Shea, L. C. (2019). *From disability to diversity: College success for students with learning disabilities, ADHD, and autism Spectrum disorder*. Columbia: National Resource Center for The First-Year Experience and Students in Transition, University of South Carolina.
- Shimabukuro, T. T., Grosse, S. D., & Rice, C. (2008). Medical expenditures for children with an autism spectrum disorder in a privately insured population. *Journal of Autism and Developmental Disorders*, 38(3), 546–552.
- Shmulsky, S., Gobbo, K., & Donahue, A. (2015). Groundwork for success: A college transition program for students with ASD. *Journal of Postsecondary Education and Disability*, 28(2), 235–241.
- Siew, C. T., Mazzucchelli, T. G., Rooney, R., & Girdler, S. (2017). A specialist peer mentoring program for university students on the autism spectrum: A pilot study. *PLoS One*, 12(7), e0180854.
- Smith, T. C. (2005). IDEA 2004: Another round in the reauthorization process. *Remedial and Special Education*, 26(6), 314–319.
- Steinhausen, H. C., Jensen, C. M., & Lauritsen, M. B. (2016). A systematic review and meta-analysis of the long-term overall outcome of autism spectrum disorders in adolescence and adulthood. *Acta Psychiatrica Scandinavica*, 133, 445–452.
- Sung, C., Sánchez, J., Kuo, H., Wang, C., & Leahy, M. J. (2015). Gender differences in vocational rehabilitation service predictors of successful competitive employment for transition-aged individuals with autism. *Journal of Autism and Developmental Disorders*, 45(10), 3204–3218.
- Taylor, J. L., & Seltzer, M. M. (2011). Employment and post-secondary educational activities for young adults with autism Spectrum disorders during the transition to adulthood. *Journal of Autism and Developmental Disorders*, 41(5), 566–574.
- The Inter-agency Autism Coordinating Committee. (2019, July 9th). *Full committee meeting - July 19th, 2016*. IACC Meetings. <https://iacc.hhs.gov/meetings/iacc-meetings/2016/full-committee-meeting/july19/>
- Tipton, L. A., & Blacher, J. (2014). Brief report: Autism awareness: Views from a campus community. *Journal of Autism and Developmental Disorders*, 44(2), 477–483.
- Torpey, E. (2018). Data on display: Measuring the value of education. Retrieved from <https://www.bls.gov/careeroutlook/2018/data-on-display/education-pays.htm>
- Trembath, D., Germano, C., Johanson, G., & Dissanayake, C. (2012). The experience of anxiety in young adults with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 27(4), 213–244.
- U.S. Department of Health and Human Services. (2017) *Report to congress: Young adults and transitioning youth with autism spectrum disorder. October 2017*. Retrieved from the U.S. Department of Health and Human Services website: <https://www.hhs.gov/sites/default/files/2017AutismReport.pdf>
- Van Hees, V., Moyson, T., & Roeyers, H. (2015). Higher education experiences of students with autism spectrum disorder: Challenges, benefits and support needs. *Journal of Autism and Developmental Disorders*, 45(6), 1673–1688.
- VanBergeijk, E., Klin, A., & Volkmar, F. (2008). Supporting more able students on the autism Spectrum: College and beyond. *Journal of Autism and Developmental Disorders*, 38, 1359–1370.
- Wei, X., Yu, J. W., Shattuck, P., McCracken, M., & Blackorby, J. (2013). Science, technology, engineering, and mathematics (STEM) participation among college students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43, 1539–1546.
- Wei, X., Cristiano, E. R. A., Yu, J. W., Blackorby, J., Shattuck, P., & Newman, L. A. (2014). Post-secondary pathways and persistence for STEM versus non-STEM majors: Among college students with an autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44, 1159–1167.
- Wenzel, C., & Brown, J. T. (2014). Beyond academic intelligence: Increasing college success for students on the autism spectrum. In F. R. Volkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, pp. 918–931). Hoboken: Wiley.
- What We Offer: Career Closet. (2019). Retrieved from <https://www.cco.purdue.edu/Students/WhatWeOffer/#CareerCloset>
- White, S. W., Elias, R., Salinasa, C. E., Capriolaa, N., Connera, C. M., Asselinb, S. B., Miyazakic, Y.,

- Mazefsky, C. A., Howling, P., & Getzel, E. E. (2016). Students with autism spectrum disorder in college: Results from a preliminary mixed methods needs analysis. *Research in Developmental Disabilities, 56*, 29–40.
- White, D., Hillier, A., Frye, A., & Makrez, E. (2019). College students' knowledge and attitudes towards students on the autism spectrum. *Journal of Autism and Developmental Disorders, 49*(7), 2699–2705.

Autism in the Courtroom

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Synonyms

Judgments; Justice; Law; Legal system

Definition

Autistic people are thought to be more likely than non-autistic people to come into contact with the justice system, including the courts. This does not imply that autistic people are more likely to offend; indeed, the limited available evidence suggests that autistic people are generally as law abiding (if not more so) than the general population. Due to vulnerabilities experienced by some autistic people (e.g., diminished social insight coupled with feelings of social alienation and eagerness for peer approval), much of the contact that autistic people have with the justice system may be as victims or witnesses (e.g., because of a limited ability to detect suspicious behavior and mal-intent by others, heightening their risk of manipulation).

Different countries have different judicial systems, but research on autism in the courtroom has

tended to focus on criminal proceedings (addressing matters in relation to criminal law, e.g., murder, robbery, motoring offences) or family proceedings (addressing matters in relation to family law, e.g., custody of children, divorce proceedings). Irrespective of the type of court (e.g., criminal or family), or the capacity in which the autistic person is involved in the justice system (e.g., witness or defendant), there are aspects of the courtroom environment and proceedings that are likely to be particularly problematic for an autistic person.

First, “unusual” behavior or communication in the courtroom may lead to negative perceptions of autistic people (particularly as defendants). This may be particularly relevant if an autism diagnosis is not known or disclosed. Cooper and Allely (2017), for example, refer to the case of *R v Sultan*. Mr. Sultan was a defendant diagnosed as autistic after his trial for rape and indecent assault. Mr. Sultan’s “strange behavior in court, such as reading a book while [the alleged victim] gave her evidence” was noted. Yet even if an autism diagnosis is known pretrial, the autistic witness/defendant may choose not to disclose their diagnosis: research has shown that autistic people are reluctant to tell legal professionals about their diagnosis due to fear of discrimination.

Second, knowledge and experience of autism among courtroom professionals may be pertinent. While many courtroom professionals (e.g., barristers, judges) report that they feel knowledgeable about autism, they often add that they do not feel confident about working with autistic people and are not overly satisfied with their interactions with autistic people in the courtroom. Calls for greater training on supporting autistic people in the courtroom should, therefore, have a distinctly practical focus: improving legal professionals’ self-efficacy, and not just their knowledge of autism.

Recent research has focused on how autistic people are perceived in the courtroom by judges and juries, and this has yielded mixed findings. While concerns have been raised about the perceived unreliability of autistic witnesses, mock jurors’ perceptions of autistic witnesses tend to be positively impacted by the knowledge of a person’s autism diagnosis. This does, however,

appear to depend on the degree of “unusual” behaviors displayed by the witness. Moreover, some evidence suggests that an autism diagnosis may have little impact on decisions of criminal responsibility, while other evidence suggests that judges and jurors may take autism into account as a mitigating factor or that an autistic offender may be less likely to receive a custodial sentence (instead being diverted out of the justice system). Legal professionals report that they struggle to determine what emphasis to place on diagnosis and other information from psychiatric reports; while some use it as a mitigating factor, others consider autism to be an aggravating factor.

Overall, research has shown that autistic people and their families often feel dissatisfied with their experiences in the courtroom. To improve their experiences, an autism diagnosis should, at least, enable reasonable adjustments to ensure fair access to trial, for example, with the provision of an intermediary (a trained professional whose role is to facilitate effective communication between vulnerable witnesses and members of the justice system, a role that is limited to a few countries at present, e.g., England, Australia). It is also vital that appropriate support is provided for both witnesses and defendants (some support, e.g., from an intermediary, is limited to witnesses only). Finally, assistance should also be given to juries, to make decisions informed by expert insights of the needs and complexities associated with autism.

Given the reportedly high rates of autistic people engaged with the courts, this topic is an important avenue for future research, alongside more general awareness and consideration of autism within the courtroom.

See Also

- [Court Decision \(ASD Related\)](#)
- [Criminality, Interactions with Law Enforcement, and Potential Correlates of Juvenile Justice Involvement Among Youth with Autism](#)
- [Law Enforcement Agencies and Autism](#)
- [Law Enforcement Knowledge of Autism](#)
- [Police-Citizen Interactions, Theory of Mind, and ASD](#)

References and Reading

- Cooper, P., & Allely, C. S. (2017). You can't judge a book by its cover: Evolving professional responsibilities, liabilities and 'judgecraft' when a party has Asperger's Syndrome. *Northern Ireland Legal Quarterly*, 68(1), 35–58.
- Freckelton, I. (2013). Forensic issues in autism spectrum disorder: Learning from court decisions. In *Recent advances in autism spectrum disorders – Volume II*. <https://doi.org/10.5772/55400>.
- George, R., Crane, L., Bingham, A., Pophale, C., & Remington, A. (2018). Legal professionals' knowledge and experience of autistic adults in the family justice system. *Journal of Social Welfare and Family Law*, 40(1), 78–97. <https://doi.org/10.1080/09649069.2018.1414381>.
- Maras, K. L., Crane, L., Mulcahy, S., Hawken, T., Cooper, P., Wurtzel, D., & Memon, A. (2017). Brief report: Autism in the courtroom: Experiences of legal professionals and the autism community. *Journal of Autism and Developmental Disorders*, 47, 2610–2620. <https://doi.org/10.1007/s10803-017-3162-9>.
- Maras, K., Marshall, I., & Sands, C. (2019). Mock juror perceptions of credibility and culpability in an autistic defendant. *Journal of Autism and Developmental Disorders*, 49, 996–1010. <https://doi.org/10.1007/s10803-018-3803-7>.

Autism Policy

- [Politics of Autism](#)

Autism Research Priorities

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Definition

Autism research priorities are the areas of scientific study that are deemed most important and valuable by stakeholders within the autism community, including people with autism, their family members and caregivers, and autism service providers (e.g., physicians, educators, etc.). Generally, these priorities range from basic

science studies that include investigations of etiology and neurobiological mechanisms, to translational research focused on using basic knowledge to develop novel interventions and supports, to applied science research testing the efficacy and effectiveness of treatments, and, finally, to clinical implementation studies focused on real-world dissemination and use of effective interventions (Frazier et al. 2018). Research priorities engage the full complement of scientific and clinical disciplines including genetics, molecular and systems biology, neurology and neuroscience, immunology, physiology, psychiatry, behavioral and intervention science, speech and communication science, and public health to advance research and practice for autism (IACC 2012).

Historical Background

Over the last two decades, research has identified and begun to explain the etiologic and phenotypic heterogeneity of autism (de la Torre-Ubieta et al. 2016; Georgiades et al. 2014). Beginning in the early 1990s, the prevalence of children identified with autism spectrum disorders began to increase (Fombonne 2009). This has been attributed to a combination of increased awareness of the disorder's early signs and symptoms among families and health-care providers as well as, in 2013, a change to the clinical definition of autism spectrum disorder. What is now known as autism spectrum disorder was previously classified as three distinct diagnoses in the Diagnostic and Statistical Manual of Mental Disorders (DSM): autism, Asperger's syndrome, and pervasive developmental disorder – not otherwise specified (PDD-NOS). The fifth edition of the DSM (DSM-V) featuring this more expansive definition was published in 2013. Between 2002 and 2014, the prevalence estimates of this heterogeneous group of disorders increased from 1/150 to 1/59 (Baio et al. 2018).

Increasing prevalence rates in the last 20 years have been followed by increased research funding to understand autism's biological underpinnings and develop more effective interventions. Increases in public and private/nonprofit

research funding have been an important part of promoting autism research advances. From 2008 to 2015, autism research funding in the USA (public and private) expanded from \$222 million to \$343 million, with projected increases to \$496 million by 2020 (source: NIH RePORTER) (National Institutes of Health 2017). Expanded funding has paralleled increases in the number of autism publications, from less than 500 in 2000 to more than 3500 in 2015 (source: US National Library of Medicine PubMed database search) (Ncbi Resource Coordinators 2017). Similarly, the number of patent applications relevant to autism increased from 4 in 2001 to almost 60 in 2014 (source: US Patent and Trademark Office AppFT) (US Patent and Trademark Office 2017). While not every research area has received strong funding, investigations have proceeded on a wide front, with prominent topics ranging from genetics and basic neuroscience to cognition and behavior to clinical trials. As the cohorts of young children diagnosed during the expansion of autism awareness have progressed to adolescence and early adulthood, research into understudied topics such as adult transition has also increased. However, absolute funding levels for services and lifespan research – areas important to the quality of life of many individuals and families – lag behind (Interagency Autism Coordinating Committee (IACC) October 2017).

Research priorities in the late twentieth and early twenty-first century focused on understanding if and how autism is inherited, the early emergence of neurodevelopmental symptoms, and understanding the outcomes of existing pharmacologic and behavioral treatments. Over three decades, from 1980 to 2010, these studies identified strong heritability but only a handful of rare genetic causes (Huguet et al. 2016), earlier emergence and ability to identify autism symptoms (Pierce et al. 2019), variable but generally weak pharmacologic benefit outside of atypical antipsychotics for challenging behavior (Goel et al. 2018), and positive short- and long-term outcomes from behavioral treatments (Tiede and Walton 2019; Schreibman et al. 2015; Gengoux et al. 2019; Green et al. 2017; Rogers et al. 2019). The early emergence of autism symptoms and the existence of effective early intervention have

driven the current emphasis on early screening, diagnosis, and intervention. In turn, this emphasis has improved cognitive, functional, and symptom outcomes as well as overall quality of life for many people with autism and their families. However, even through 2019, much work remains in understanding the etiology of autism and translating these findings into more effective biological interventions.

Traditionally, genetic and other basic science investigations of autism spectrum disorder have failed to include the perspectives of stakeholders in the development of research aims. This oversight has often extended to clinical trials and other applied scientific studies. Including viewpoints of those with autism and their families as well as those who work directly with persons on the spectrum, such as therapists and teachers, in decision-making can increase the benefits that future research delivers to individuals and families, iteratively improving community engagement and participation in research (Fletcher-Watson et al. 2017b). The viewpoints of scientists conducting research are important as well, as ideally there is some alignment between researchers and stakeholders directly affected by autism. If there is misalignment, it would be crucial to consider barriers that might discourage scientists from devoting their research to areas considered high priority from those affected directly by autism, such as available funding directed toward those areas.

The preferences and perspectives of stakeholders, including those with autism, their families, and service providers, will differ depending on each person's specific challenges. Understanding the wide range of needs and desires of people that are directly affected by autism, including persons on the spectrum and families, as well as providers (clinicians and educators) and researchers, is important in setting priorities regarding autism research funding (Pellicano et al. 2014a, b). Over the past few years, several investigations have been conducted to better understand research priorities of autism stakeholders and how these priorities can inform future research foci, design, and conduct as well as funding priorities for major funding organizations.

Current Knowledge

In its most recent analysis of autism funding in the USA, the Interagency Autism Coordinating Committee (IACC) reviewed all autism-related projects, which totaled more than \$406 billion dollars in 2010. While previous analysis has found the majority of funding for autism research goes to basic science, including risk factors, biological mechanisms, and genetic factors, the distribution of funding shifted significantly between 2000 and 2010. US funding analyzed by IACC found even priority research areas – biology, risk factors, treatments/interventions, services, infrastructure and monitoring, diagnosis, and lifespan issues. Of these, six received between 11% and 22% of the total autism research funding in 2010. Lifespan issues, however, remained the least-funded research priority area at 2% of total funding (IACC 2012). In several studies, stakeholders ranked lifespan issues and adult outcomes as top research priorities (Gotham et al. 2015), in contrast to its comparably lower current funding levels compared with other research areas.

Community and systemic factors contribute significantly to outcomes of youth with autism transitioning to adulthood – one of the primary targets of lifespan issues in autism. Reviews of the literature and additional studies have identified gaps in the current knowledge regarding these factors. These include the social-ecological determinants of transition outcomes, the broad range of support needs for adults with autism, lack of longitudinal data, lack of comparison of research findings from studies with small sample sizes or other limitations, and outdated quality of life indicators (Shattuck et al. 2018). Stakeholders continue to identify as a top priority the need for more information on building community capacity to support successful outcomes and how to improve systems of care across educational, health, employment, and community settings (Shattuck et al. 2018).

Overall, the pattern of results highlights the importance of collaborating with people with autism and their family members in developing scientific research priorities and the funding process (Pellicano et al. 2014a). Researchers should also consider the broad range of stakeholders'

perspectives when developing their research aims and designing studies. Including multiple caregivers, clinicians, educators, and (of course) people with autism may be important to the development and design of studies to ensure adequate coverage of the large variability of perspectives. Meaningful input from people with autism and their families has the potential to increase the ability to translate findings into practice and make sure outcomes can impart real benefits.

Recognition of the importance of inclusion is driving a movement toward a participatory model to meaningfully engage people with autism and their caregivers and advocates in all aspects of research (Fletcher-Watson et al. 2019). Features of participatory research include collaboration and open dialogue with autism community representatives; listening to and including their feedback in research priorities, funding, aims, and design; and acknowledging a lack of power equity between researchers and study participants in most settings.

People with autism tend to rate researcher priorities as a bit less important than other stakeholders. This may reflect a desire by many verbal, cognitively able people with autism to express that their condition should be understood and accepted rather than pathologized. Some studies have found that current autism research funding levels are not consistent with the priorities of the autism community (Fletcher-Watson et al. 2019), although funding trends show movement in that direction. Some studies in Europe have shown that adults with autism have less favorable attitudes about research than the general population and dislike the clinical terminology “at-risk” when used to describe infants in research studies (Gillespie-Lynch et al. 2017; Fletcher-Watson et al. 2017a).

Applied research tends to be rated as having higher importance than basic or translational science areas, particularly by people with autism. This may reflect the strong desire to identify and implement more effective interventions and supports in the near future. For people with autism, lower ratings for basic science may also reflect a lower average desire to identify etiology versus focusing on acceptance, understanding and modifying current policies and systems to be more supportive and inclusive.

Adult transition, lifespan issues, health and well-being, and co-occurring medical and mental health conditions appear to be very important topics across most stakeholders. Among autism stakeholders who responded to a 2018 survey, areas of applied science are generally deemed more important and valuable than basic science to community stakeholders (Frazier et al. 2018). This reflects the needs and wants of people in this community for quicker access to better treatments and solutions to common challenges. Applied science areas include screening and identification; co-occurring conditions; medical interventions, devices, and other technology; adult transition; lifespan issues; and health and well-being. Respondents gave their highest ratings to research focused on co-occurring conditions, health and well-being, adult transition, and lifespan issues. These results can guide decision-making by public and private funders when developing science funding priorities and engaging in science dissemination activities.

However, basic biological science was also rated an important and valuable area of scientific research by these stakeholders. Basic biological science areas include genetics; molecular studies; cellular studies; animal models; environmental risk and protective factors; biomarkers; immunity and inflammation; and metabolic and mitochondrial function. These topics remain important research priorities due to their potential to lead to better understanding and future treatment options.

Autism research priorities considered primary targets by IACC in the USA include (IACC 2012):

Diagnosis: Diagnostics research aims to develop and validate tools to accurately diagnose autism, look for early signs and biomarkers of risk for autism, define the subtypes or subgroups of people with autism who share similar symptoms and features, and define concretely symptoms that can be reliably and effectively used in autism evaluation.

Biology: This priority area explores the biological mechanisms that underlie the core features of autism and identifies potential targets for interventions to improve function, skill development, and quality of life. Historically, this

research area has focused on early development from the prenatal period through infancy and elementary school age as a means for lowering the potential age for diagnosis and promoting the earliest possible interventions where needed. It also includes investigating the genetic basis for these biological factors.

Risk Factors: Looking for risk factors for autism includes identifying genetic risk factors and genes that cause autism. More recently, this area has broadened to include studying the microbiome and other environmental influences on gene expression (epigenetics).

Treatments/Interventions: This priority area explores the safety and effectiveness of interventions for impairment of function related to core autism features like communication and behavior, as well as other dimensions such as quality of life, mental health, various therapies (e.g., speech, occupational, ABA), and medications. Research in this area also looks for information about medical conditions that often accompany autism and ways to appropriately care for these conditions.

Services: Research into autism services includes various areas, including access to services, dissemination of evidence-based practices, and training of providers.

Lifespan Issues: This research area targets interventions, services, and supports across the lifespan into adulthood, their impact on adult health and quality of life, and the process of transition to adulthood and systemic influences on successful transition.

Infrastructure and Surveillance: Research infrastructure establishes systemic mechanisms to track changes in population outcomes over time. It includes developing networks of researchers to share data, establishing data repositories and biological repositories, and implementing research technology, tools, and protocols to create and access standardized data.

Future Directions

Adult transition, in particular, is garnering increasing attention, both within the broader

autism community and by funding agencies (Gotham et al. 2015). In the early to mid-2000s, increasing diagnosis of autism among childhood cohorts led to an emphasis on early identification and intervention. As these cohorts move through adolescence and into adulthood, the community recognizes the need for evidence-based practices and processes that facilitate a successful transition to adulthood.

While research on adult transition – particularly descriptions of the challenges – has increased, at present there are few evidence-based practices to address this massive public health need. Similarly, little data exist on the lifespan development of autism and issues that might accompany aging. It is likely that both public and private funding will be needed to adequately fill these gaps, raise our understanding of the successes and challenges faced by adults with autism, and develop evidence-based practices that can facilitate health and well-being throughout the lifespan. Autism Speaks, for example, will award nearly \$2.6 million in 2019 on four multi-year studies on issues around transition to adulthood for people with autism.

Federal agencies are also addressing the dearth of knowledge about the range of health and support services facing the adult autism population. The US Congress passed the Autism CARES Act of 2019 as a renewal of previous federal legislation that outlines federal research, service coordination, and surveillance of autism in the USA. The original legislation established the infrastructure need for the Centers for Disease Control and Prevention to study and monitor autism across the country. This infrastructure supports five networks and eight autism intervention research projects (Association of University Centers on Disabilities 2019) to identify areas of need, including screening and intervention services, shortages of qualified personnel in autism diagnosis and treatment, and identifying evidence-based practices for autism and related conditions. The legislation also established the Interagency Autism Coordinating Committee (IACC) to coordinate autism activities among federal agencies and public organizations and advise the Health and Human Services Secretary

on autism science and policy. IACC has identified seven research priority areas in its strategic plan: biology, risk factors, treatments and interventions, services, lifespan issues, infrastructure and surveillance, and screening and diagnosis.

As autism services and research funding has grown, so has our understanding of the health disparities facing many people with autism in certain communities (Karpur et al. 2019). Research into these disparities spans health-care access and utilization, health-care quality, and service quality and delivery and identifies determinants of health. Research is ongoing into the cultural, economic, social, racial, ethnic, policy, and other systemic barriers to autism care in underserved populations, as well as into mechanisms to resolve these disparities. These efforts include the development of valid, reliable, and culturally appropriate diagnostic tools, measures, and interventions for underserved and low-resource populations internationally (Daniels 2019).

References and Reading

- AppFT: Patent Application and Full-Text Image Database. (2017). <http://appft.uspto.gov/netahtml/PTO/search-adv.html>. Accessed 28 Dec 2017.
- Association of University Centers on Disabilities. (2019). *AUCD: Association of University Centers on Disabilities: Research, Education, Service*. Retrieved from <http://aucd.org>.
- Baio, J., Wiggins, L., Christensen, D. L., et al. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 Sites, United States, 2014. *MMWR Surveillance Summaries*, 67(SS-6), 1–23. <https://doi.org/10.15585/mmwr.ss6706a1>.
- Daniels, S. A. on behalf of the Interagency Autism Coordinating Committee (IACC). (2019). IACC committee business. Presentation, IACC meeting July 24, 2019. <https://iacc.hhs.gov/meetings/iacc-meetings/2019/full-committee-meeting/july24/#materials>
- de la Torre-Ubieta, L., Won, H., Stein, J. L., & Geschwind, D. H. (2016). Advancing the understanding of autism disease mechanisms through genetics. *Nature Medicine*, 22(4), 345–361. <https://doi.org/10.1038/nm.4071>.
- Fletcher-Watson, S., Apicella, F., Auyeung, B., Beranova, S., Bonnet-Brilhault, F., Canal-Bedia, R., et al. (2017a). Attitudes of the autism community to early autism research. *Autism*, 21(1), 61–74. <https://doi.org/10.1177/1362361315626577>.
- Fletcher-Watson, S., Larsen, K., Salomone, E., & Members of the, C. E. W. G. (2017b). What do parents of children with autism expect from participation in research? A community survey about early autism studies. *Autism*, 1362361317728436. <https://doi.org/10.1177/1362361317728436>.
- Fletcher-Watson, S., Adams, J., Brook, K., Charman, T., Crane, L., Cusack, J., . . . Pellicano, E. (2019). Making the future together: Shaping autism research through meaningful participation. *Autism: The International Journal of Research and Practice*, 23(4), 943–953. <https://doi.org/10.1177/1362361318786721>.
- Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. In *Pediatric research* (Vol. 65), 591. <https://doi.org/10.1203/PDR.0b013e31819e7203>
- Frazier, T. W., Dawson, G., Murray, D., Shih, A., Snyder Sachs, J., & Geiger, A. (2018). Brief report: A survey of autism research priorities across a diverse community of stakeholders. *Journal of Autism and Developmental Disorders*, 48(11). <https://doi.org/10.1007/s10803-018-3642-6>.
- Gengoux, G. W., Abrams, D. A., Schuck, R., Millan, M. E., Libove, R., Ardel, C. M., Phillips, J. M., Fox, M., Frazier, T. W., & Hardan, A. Y. (2019). A pivotal response treatment package for children with autism spectrum disorder: An RCT. *Pediatrics*, 144(3). <https://doi.org/10.1542/peds.2019-0178>.
- Georgiades, S., Boyle, M., Szatmari, P., Hanna, S., Duku, E., Zwaigenbaum, L., et al. (2014). Modeling the phenotypic architecture of autism symptoms from time of diagnosis to age 6. *Journal of Autism and Developmental Disorders*, 44(12), 3045–3055. <https://doi.org/10.1007/s10803-014-2167-x>.
- Gillespie-Lynch, K., Kapp, S. K., Brooks, P. J., Pickens, J., & Schwartzman, B. (2017). Whose expertise is it? Evidence for autistic adults as critical autism experts. *Frontiers in Psychology*, 8, 438. <https://doi.org/10.3389/fpsyg.2017.00438>.
- Goel, R., Hong, J. S., Findling, R. L., & Ji, N. Y. (2018). An update on pharmacotherapy of autism spectrum disorder in children and adolescents. *International Review of Psychiatry*, 30(1), 78–95. <https://doi.org/10.1080/09540261.2018.1458706>.
- Gotham, K., Marvin, A. R., Taylor, J. L., Warren, Z., Anderson, C. M., Law, P. A., Law, J. K., & Lipkin, P. H. (2015). Characterizing the daily life, needs, and priorities of adults with autism spectrum disorder from interactive autism network data. *Autism*, 19(7), 794–804. <https://doi.org/10.1177/1362361315583818>.
- Green, J., Pickles, A., Pasco, G., Bedford, R., Wan, M. W., Elsabbagh, M., Slonims, V., Gliga, T., Jones, E., Cheung, C., Charman, T., Johnson, M., & British Autism Study of Infant Siblings (BASIS) Team. (2017). Randomised trial of a parent-mediated intervention for infants at high risk for autism: Longitudinal outcomes to age 3 years. *Journal of Child Psychology*

- and *Psychiatry*, 58(12), 1330–1340. <https://doi.org/10.1111/jcpp.12728>.
- Huguet, G., Benabou, M., & Bourgeron, T. (2016). The genetics of autism spectrum disorders. In P. Sassone-Corsi, Y. Christen (Eds.), *A time for metabolism and hormones* [Internet]. Cham: Springer.
- Interagency Autism Coordinating Committee (IACC). (October 2017). 2016–2017 Interagency autism coordinating committee strategic plan for autism spectrum disorder. Retrieved from the U.S. Department of Health and Human Services Interagency Autism Coordinating Committee website <https://iacc.hhs.gov/publications/strategic-plan/2017/>
- Kapur, A., Lello, A., Frazier, T., Dixon, P. J., & Shih, A. J. (2019). Health disparities among children with autism spectrum disorders: Analysis of the National Survey of Children's Health 2016. *Journal of Autism and Developmental Disorders*, 49(4), 1652–1664. <https://doi.org/10.1007/s10803-018-3862-9>.
- National Institutes of Health. (2017). Research portfolio online reporting tools (RePORT): RePORTER manual. (12/19/2017 ed., Vol. 7.24.0).
- Ncbi Resource Coordinators. (2017). Database resources of the National Center for Biotechnology Information. *Nucleic Acids Research*, 45(D1), D12–D17. <https://doi.org/10.1093/nar/gkw1071>.
- Office of Autism Research Coordination, National Institute of Mental Health, on behalf of the Interagency Autism Coordinating Committee (IACC). 2010 IACC Autism Spectrum Disorder Research Portfolio Analysis Report. July 2012. Retrieved from the U.S. Department of Health and Human Services Interagency Autism Coordinating Committee website: <http://iacc.hhs.gov/portfolio-analysis/2010/index.shtml>
- Pellicano, E., Dinsmore, A., & Charman, T. (2014a). Views on researcher-community engagement in autism research in the United Kingdom: A mixed-methods study. *PLoS One*, 9(10), e109946. <https://doi.org/10.1371/journal.pone.0109946>.
- Pellicano, E., Dinsmore, A., & Charman, T. (2014b). What should autism research focus upon? Community views and priorities from the United Kingdom. *Autism*, 18(7), 756–770. <https://doi.org/10.1177/1362361314529627>.
- Pierce, K., Gazestani, V. H., Bacon, E., Barnes, C. C., Cha, D., Nalabolu, S., Lopez, L., Moore, A., Pence-Stophaeos, S., & Courchesne, E. (2019). Evaluation of the diagnostic stability of the early autism spectrum disorder phenotype in the general population starting at 12 months. *JAMA Pediatrics*, 173(6), 578–587. <https://doi.org/10.1001/jamapediatrics.2019.0624>.
- Rogers, S. J., Estes, A., Lord, C., Munson, J., Rocha, M., Winter, J., Greenon, J., Colombi, C., Dawson, G., Vismara, L. A., Sugar, C. A., Helleman, G., Whelan, F., & Talbott, M. (2019). A multisite randomized controlled two-phase trial of the Early Start Denver Model compared to treatment as usual. *Journal of the American Academy of Child and Adolescent Psychiatry*, 58(9), 853–865. <https://doi.org/10.1016/j.jaac.2019.01.004>.
- Schreibman, L., Dawson, G., Stahmer, A. C., Landa, R., Rogers, S. J., McGee, G. G., Kasari, C., Ingersoll, B., Kaiser, A. P., Bruinsma, Y., McNerney, E., Wetherby, A., & Halladay, A. (2015). Naturalistic developmental behavioral interventions: Empirically validated treatments for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(8), 2411–2428. <https://doi.org/10.1007/s10803-015-2407-8>.
- Shattuck, P. T., Lau, L., Anderson, K. A., & Kuo, A. A. (2018). A national research agenda for the transition of youth with autism. *Pediatrics*, 141(Suppl 4), S355–S361. <https://doi.org/10.1542/peds.2016-4300M>.
- Tiede, G., & Walton, K. M. (2019). Meta-analysis of naturalistic developmental behavioral interventions for young children with autism spectrum disorder. *Autism*, 24, 1362361319836371. <https://doi.org/10.1177/1362361319836371>.

Autism Science Foundation

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Major Areas or Mission Statement

The Autism Science Foundation is a 501(c)3 non-profit that supports autism research by providing funding directly to scientists conducting cutting-edge autism research. ASF also shares information about autism with the general public and serves to increase awareness of autism spectrum disorders and the needs of individuals and families affected by autism. The organization was founded in 2009 by Alison Singer and Karen London, parents of children with autism.

ASF adheres to rigorous scientific standards and values and believes that outstanding research is the greatest gift that can be offered to families.

The Autism Science Foundation's mission is premised on the following facts and principles:

- Autism is known to have a strong genetic component. Research must aim to discover the mechanisms of action that trigger autism, as

well as safe, effective, and novel treatments to enhance the quality of life for children and adults currently affected.

- Early diagnosis and early intervention are critical to helping people with autism reach their potential, but educational, vocational, and support services must be applied across the lifespan. Science has a critical role to play in creating evidence-based, effective lifespan interventions.
- Vaccines save lives; they do not cause autism. Numerous studies have failed to show a causal link between vaccines and autism. Vaccine safety research should continue to be conducted by the public health system in order to ensure vaccine safety and maintain confidence in our national vaccine program, but further investment of limited autism research dollars is not warranted at this time.

Major Activities

ASF provides pre- and postdoctoral research fellowships to support promising young researchers working to discover the causes of autism and develop new treatments. The foundation also offers grant to highly dedicated undergraduates to support summer research and to medical school students to support gap-year research.

ASF hosts the annual autism “TED” talks in New York City and San Francisco each year. These days of learning are designed to bring autism researchers and community stakeholders together to exchange ideas. The foundation also supports an annual symposium to share new school-based autism treatment research with classroom teachers.

ASF produces an award-winning weekly “Autism Research” podcast and disseminates research information to families via multiple social media platforms.

In 2017, ASF launched the Autism Sisters Project in an effort to understand why four times as many boys as girls are diagnosed with autism and to study autism’s female protective effect.

ASF also sponsors the international Autism Baby Siblings Research Consortium, which studies the earliest signs of autism, and recently

launched the Next Gen Sibs project to measure recurrence risk and learn about early signs in the children of unaffected siblings.

ASF serves as the community outreach partner for the international Autism BrainNet, encouraging families all over the world to donate postmortem brain tissue for research.

ASF works closely with other health-care entities, including the American Academy of Pediatrics (AAP), the Centers for Disease Control and Prevention (CDC), and the National Institutes of Health (NIH), to ensure that accurate information about autism spectrum disorders is widely available. ASF’s leaders and board members are frequently called upon by major news media to comment on issues relevant to autism research and public policy.

ASF also has major projects underway to study best practices for supported employment, to encourage more families to participate in autism research, to prevent wandering by children with autism, and to encourage families to vaccinate their children and pursue evidence-based treatments.

ASF’s president, Alison Singer served for 12 years as a public member of the Interagency Autism Coordinating Committee. The Interagency Autism Coordinating Committee (IACC) is a federal advisory committee that coordinates all efforts within the Department of Health and Human Services (HHS) concerning autism spectrum disorder (ASD). Through its inclusion of both federal and public members, the IACC helps to ensure that a wide range of ideas and perspectives are represented and discussed in a public forum. This committee provides advice to the Secretary of Health and Human Services regarding federal activities related to autism spectrum disorders, drafts an annual strategic plan to guide federal spending on autism research, and reports annually on the most promising autism research findings.^t

See Also

- [Autism Speaks](#)
- [Vaccinations and Autism](#)

References and Reading

Autism Science Foundation.
 Latest Autism Science.
 Offit, P. (2008). *Autism's false prophets*. New York:
 Columbia University Press.
 Vaccines and Autism.

Autism Screening Instrument for Educational Planning (ASIEP-2)

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Synonyms

ASIEP-2

Description

The Autism Screening Instrument for Educational Planning (ASIEP; Krug et al. 1978) was created to facilitate autism diagnoses and to monitor the educational progress of individuals with autism (Arick et al. 2005). First created in 1978, the ASIEP was revised in 1980 and a second edition, the ASIEP-2, was released in 1993 (Krug et al. 1980, 1993). The authors claim that the ASIEP-2 can identify individuals with high levels of behaviors associated with autism (Frye and Walker 1998) and can be applied to individuals with autism from age 18 months to adults (Olm and Oswald 1998). The autism behavior checklist (ABC) is the most widely used subset of the ASIEP (Olm and Oswald).

Historical Background

The ASIEP was first created in 1978, with the ASIEP-2 following in 1993 (Krug et al. 1978, 1993). The ASIEP was designed to provide clinicians with an additional measure to diagnose

autism (Arick et al. 2005). The ASIEP-2 is different from other diagnostic measures, except the PDD behavior inventory (Cohen and Sudhalter 2005), in that it also provides information helpful in monitoring progress and in creating educational programs tailored to the specific needs of the individual with autism.

Psychometric Data

The ASIEP-2 is comprised of five separately standardized subtests: autism behavior checklist (ABC), sample of vocal behavior, interaction assessment, educational assessment, and prognosis of learning rate (Krug et al. 1993). For each of the subtests, raw scores can be converted to standard scores using tables provided in the ASIEP-2 manual (Olm and Oswald 1998). The ABC can be used for individuals of all ages and levels of autism, while the other four subtests are to be used with individuals whose language and social functioning are between 3 and 49 months (Krug et al.). The ASIEP-2 is meant to be used by professional educators and requires that the examiners are knowledgeable about autism and have had at least three weeks of interaction with the child being assessed (Frye and Walker 1998). Overall, the ASIEP-2 has been shown to have adequate validity and reliability (Frye and Walker). The diagnostic validity has been questioned by other researchers who have found the measure to be an adequate screening measure to identify individuals with high levels of behaviors associated with autism, but not a sufficient diagnostic tool (Volkmar et al. 1988).

The autism behavior checklist (ABC) is a 57-item checklist of behavioral characteristics and is meant to be filled out by individuals being assessed, their parents, and their teachers. Items fall into five behavior categories: sensory, relating, body and object use, language, and social and self-help. Each item is weighted from 1 to 4, and the sum of the scores from the five categories is calculated to produce the total score (Olm and Oswald 1998). Krug et al. (1980) reported good interrater reliability. However, due to some concerns about reliability and validity resulting from their evaluation of the ABC, Volkmar et al. suggest that the ABC is best used as a screening measure for

individuals with frequent autistic behavior, rather than as a diagnostic tool (Volkmar et al. 1988).

The sample of vocal behavior (SVB) subtest assesses the characteristics of preverbal and emerging spontaneous language in the areas of repetitiveness, noncommunication, intelligibility, and babbling (Olmi and Oswald 1998). The goal is to elicit 50 vocalizations from the child to score. Scoring categories include variety, function, articulation, and length. Psychometric studies of the SVB have demonstrated acceptable reliability and validity, but had small sample sizes and thus were less rigorous than those applied to the ABC (Olmi and Oswald). Overall, the authors found that the ASIEP-2 had high test-retest reliability (Frye and Walker 1998). In addition, significant differences between the utterances of preschool- and school-age children with autism compared to those with typical development were observed in standardization studies of matched samples.

During the interaction assessment subtest, four types of behaviors are assessed: interaction, constructive independent play, no response, and aggressive negative. Rater reliability is dependent on training and experience (Olmi and Oswald 1998). One study using the interaction assessment found high median agreement (89%) among the ratings (Frye and Walker 1998). Other reliability statistics are unavailable for this measure.

The educational assessment subtest is designed to assess the child's abilities in five areas: staying in seat, receptive language, expressive language, body concept, and speech imitation (Olmi and Oswald 1998). The educational assessment is intended to assess skills that most children with autism lack (Frye and Walker 1998).

The prognosis of learning rate subtest was created to assess the individual's ability to learn newly presented information based on reinforcement procedures and without verbal or physical cues (Olmi and Oswald 1998). There is limited psychometric data for this subtest.

Clinical Uses

The ASIEP-2 was created not only as an assistive diagnostic tool but also as a method to track

individual progress and aid in the creation of appropriate education strategies (Krug et al. 1993). One way that the ASIEP-2 differs from other psychological diagnostic measures is that it is designed to be administered as often as needed to assess progress without concerns about test-retest effects, as the measure demonstrated a lack of practice effects (Frye and Walker 1998). This makes the ASIEP-2 particularly useful for educational planning. In addition, the ABC can be used in clinical settings to create a behavior description; however, it is not sufficient as a primary diagnostic tool (Frye and Walker).

See Also

- [Autism Behavior Checklist](#)
- [Autism Diagnostic Observation Schedule](#)

References and Reading

- Arick, J. R., Krug, D. A., Fullerton, A., Loos, L., & Falco, R. (2005). School-based programs. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 730–771). Hoboken: Wiley.
- Cohen, I. L., & Sudhalter, V. (2005). *The PDD behavior inventory*. Lutz: Psychological Assessment Resources.
- Frye, V. H., & Walker, K. C. (1998). Book review: Autism screening instrument for educational planning, second edition (ASIEP-2). *Journal of Psychoeducational Assessment*, 16, 280–285.
- Krug, D. A., Arick, J. R., & Almond, P. J. (1978). *Autism screening instrument for educational planning*. Austin: ProEd.
- Krug, D. A., Arick, J. R., & Almond, P. J. (1980). *Autism screening instrument for educational planning* (Revised ed.). Austin: ProEd.
- Krug, D. A., Arick, J. R., & Almond, P. J. (1993). *Autism screening instrument for educational planning* (2nd ed.). Austin: ProEd.
- Olmi, J. D., & Oswald, D. P. (1998). [Review of the test Autism Screening Instrument for Educational Planning, Second Edition]. In *The thirteenth mental measurements yearbook*. Available from <http://www.unl.edu/buros/>
- Volkmar, F. R., Cicchetti, D. V., Dykens, E., Sparrow, S. S., Leckman, J. F., & Cohen, D. J. (1988). An evaluation of the autism behavior checklist. *Journal of Autism and Developmental Disorders*, 18, 81–97.

Autism Screening Questionnaire (ASQ)

► [Social Communication Questionnaire](#)

Autism Services Center (ASC), Huntington, West Virginia

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Major Areas or Mission Statement

ASC was founded in 1979 on the belief that each person with a developmental disability has the capacity for growth and development. Each individual has a right to services that enhance well-being, quality of life, and opportunities to learn. Each should have access to the most normal and least restrictive social and physical environments consistent with his or her needs. Those with even the most challenging behaviors can respond to dignified interaction in a structured, meaningful program with appropriately trained and supervised staff and the appropriate client/staff ratio. ASC encourages the use of best clinical practices and believes everyone has the right to effective treatment.

Landmark Contributions

Major Activities

Serving people with autism, other developmental disabilities, and those who care for and about them.

Autism Services Center employs trained staff to provide a comprehensive array of services for individuals with developmental disabilities.

Service Coordination: Service coordination services are provided for individuals with developmental disabilities who reside in Cabell, Wayne, Mason, and Lincoln Counties in WV. Service coordination establishes a potentially lifelong process

for accessing a range of services, instructions, and assistance. The service coordinator assists an individual with a developmental disability in making meaningful choices and works to ensure quality, accessibility, accountability, and continuity of support services.

Residential Habilitation: Residential Habilitation services, which occur in a client's residence and in the community, provide instruction and assistance for the acquisition and maintenance of skills, which allow for a client to live and socialize more independently. Residential habilitation services may also include behavioral intervention to reduce and eliminate challenging behaviors and replace them with socially valuable, adaptive behaviors, and skills. Autism Services Center owns and/or supervises residences in the Huntington, WV, area.

Day Habilitation: Day habilitation services take place away from an individual's home and include activities in community environments to facilitate skills acquisition. The programs are designed to assist an individual in achieving increased independence and/or to maintain their current skills in activities of daily living.

Prevocational Training: Prevocational training programs are designed to assist an individual in the acquisition and maintenance of basic work and work-related skills.

Supported Employment: Supported employment services enable individuals to engage in paid, competitive employment in integrated community settings. The services are for individuals who have barriers to obtaining employment due to the nature and complexity of their disabilities. These services are designed to assist individuals for whom competitive employment at or above minimum wage is unlikely without the supports.

Respite Care: Respite care is a service provided to an individual by trained staff due to the short-term absence of the primary caregiver.

Adult Companion Services: Adult companion services are complementary to residential habilitation services and provide nonmedical care, supervision, socialization, and assistance in tasks such as meal preparation, laundry, and shopping.

Therapeutic Consultants: Therapeutic consultants provide training for primary care providers

such as direct care staff and family members in person-specific aspects and methods of positive behavior support intervention and instruction.

Nursing Services: Nursing services are provided by registered nurses (RNs) and licensed practical nurses (LPNs) within the scope of the West Virginia Nurse Practice Act.

Family Support: The family support program allocates funds for services and equipment that are not funded by Medicaid or insurance companies. These funds are to assist individuals and their family with such things as clothing, medical care, wheelchair ramps, and respite based on the needs of the family.

Applied Behavioral Analysis (ABA): The applied behavioral analysis (ABA) program or applied behavior analysis is a scientific approach to understanding behavior, how it is affected by the environment and how learning takes place. It is a mixture of psychological and educational techniques tailored to meet the needs of each individual. ABA methods are used to measure behavior, teach functional skills, and evaluate progress.

References and Reading

www.autismservicescenter.org

Autism Society

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Major Areas or Mission Statement

The Autism Society

The Autism Society was founded in 1965 by Dr. Bernard Rimland, Dr. Ruth Sullivan, and a group of parents of children with autism. At that time, little was known about this rare disability. As they met in their living rooms, these parents were determined to create awareness and

understanding of this disorder and to provide support for families living with autism.

The Autism Society mission is to improve the lives of all affected by autism. The Autism Society works to ensure that every child and adult with autism lives an independent, fulfilled, and productive life.

Major Activities

The Autism Society supports individuals with autism and their families through the three critical stages of autism:

- **Early Detection and Intervention:** The Autism Society promotes early identification and access to effective treatment before age 3.
- **Building a Strong Foundation from Childhood through Adolescence:** The Autism Society helps parents and caregivers build education and treatment programs so that each child reaches their fullest potential.
- **A Life of Happiness and Dignity:** The Autism Society works to ensure that every adult with autism has access to services and support systems to ensure they achieve the highest quality of life and personal happiness.

Through its strong chapter network, the Autism Society has spearheaded numerous pieces of state and local legislation and offers family and individual support in over 150 locations nationwide. The Autism Society's website is one of the most visited websites on autism in the world, and its quarterly journal, *Autism Advocate*, has a broad national readership. The Autism Society also hosts a comprehensive national conference on autism that covers issues ranging from early identification to adult options each year. Autism Source, the national information and referral center, and the Autism Society's strong chapter network serve thousands of families each year who are searching for help in their journey with autism.

The Autism Society's national office is headquartered in Bethesda, Maryland, and is governed by a board of directors that includes people on the spectrum. The Autism Society's

Panel of Professional Advisors sets the standards for their Options Policy that governs the organization's programs. The Autism Society's Advisory Panel of People on the Spectrum of Autism is a first-of-its-kind advisory panel comprised solely of individuals with autism, who help Autism Society staff create programs and services that will advocate for the rights of all people with autism to live fulfilling, interdependent lives. The membership base of the Autism Society encompasses a broad and diverse group of parents, family members, special education teachers, administrators, medical doctors, therapists, adult agency personnel, nurses, and aides, as well as countless other personnel involved in the education, care, treatment, and support of individuals on the autism spectrum across the age span.

Recognizing and respecting the diverse range of opinions, needs, and desires of this group, the Autism Society embraces an overall philosophy that chooses to empower individuals with autism and their parents or caregivers to make choices best suited to the needs of the person with autism, a policy it calls the Options Policy. All activities of the Autism Society are guided by the Options Policy. Revisited on a regular basis by the organization, the Options Policy has stood the test of time. It states that:

The Autism Society promotes the active and informed involvement of family members and the individual with autism in the planning of individualized, appropriate services and supports. The Board of the Autism Society believes that each person with autism is a unique individual. Each family and individual with autism should have the right to learn about and then select the options that they feel are most appropriate for the individual with autism. To the maximum extent possible, we believe that the decisions should be made by the individual with autism in collaboration with family, guardians, and caregivers.

Services should enhance and strengthen natural family and community supports for the individual with autism and the family whenever possible. The service option designed for an individual with autism should result in improved quality of life. Abusive treatment of any kind is not an option.

We firmly believe that no single type of program or service will fill the needs of every individual with autism and that each person should have access to support services. Selection of a program, service, or method of treatment should be on the basis of a full assessment of each person's abilities, needs, and interests. We believe that services should be outcome based to insure that they meet the individualized needs of a person with autism.

With appropriate education, vocational training and community living options and support systems, individuals with autism can lead dignified, productive lives in their communities and strive to reach their fullest potential.

In addition to the Options Policy, the Autism Society has created guiding principles to further define their work. These guiding principles include:

- The Autism Society's efforts are focused on meaningful participation and self-determination in all aspects of life for individuals on the autism spectrum and their families.
- The Autism Society promotes individual, parental, and guardian choice to assure that people on the autism spectrum are treated with dignity and respect.
- The Autism Society proactively informs, influences, guides, and develops public policy at the federal, state, and local levels, including setting agendas for policymakers and legislators, for the benefit of the autism community.
- The Autism Society is the respected voice of the autism community and the primary source for information by providing timely, frequent, relevant, and professional communication.
- The Autism Society works to ensure that every chapter is a successful chapter, sustained by a collaborative relationship between the national office and chapters to realize mutual benefit and to protect the interests of both.
- The Autism Society advocates for multidisciplinary approaches to autism research focused on improving the quality of life for individuals across the autism spectrum and their families.

- The Autism Society works to ensure financial self-sufficiency and growth for all Autism Society operating units and integrated operations across all levels of the Autism Society.

At the very core of the Options Policy is the belief that no single program or treatment will benefit all individuals with autism and that ultimate parents should have informed choices. Furthermore, the recommendation of what is “best” or “most effective” for a person with autism should be determined by those people directly involved – the individual with autism, to the extent possible, and the parents or family members.

References and Reading

Autism Society. (2012). For more information about the Autism Society. Retrieved on 28 June 2012, from www.autism-society.org

Autism Speaks

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Major Areas or Mission Statement

Autism Speaks is North America’s largest autism science and advocacy organization. Its goal is to change the future for all who struggle with autism spectrum disorders (ASD). Autism Speaks is dedicated to funding global biomedical research into the causes, prevention, treatments, and cures for ASD; raising public awareness about ASD and its effects on individuals, families, and society; and bringing hope to all who deal with the hardships of this disorder. The organization is committed to raising the funds necessary to support these goals. Autism Speaks aims to bring the autism community together as one strong voice to urge the

government and private sector to listen to the concerns and take action to address this urgent global public health crisis. The core values reflected in Autism Speaks’ mission statement are (1) recognition that individuals with ASD and their families often face struggle, which inspires a sense of urgency; (2) commitment to discovery through scientific excellence; and (3) the belief and commitment that parents are partners in this effort.

Landmark Contributions

Funding Autism Science

Since its inception in 2005, Autism Speaks has made enormous strides, committing over \$170 million to research through 2014. In support of its mission to improve the future for all who struggle with ASD, Autism Speaks provides funding along the entire research continuum – from discovery to development to dissemination – for innovative projects that hold considerable promise in significantly improving the lives of persons with autism. Annually, Autism Speaks accepts applications through a number of grant funding mechanisms for investigator-initiated research projects. This includes cornerstone mechanisms such as the Pilot, Basic & Clinical, Treatment, and Predoctoral Fellowship Awards, as well as targeted mechanisms including Post-doctoral Fellowships in Translational Autism Research and the Suzanne and Bob Wright Trailblazer Award.

Assessing the Impact of Research Grant Funding

A survey was conducted to assess the outcomes and impact of Autism Speaks-funded grants completed by 2010. The vast majority (82%) of respondents reported the major finding as a novel discovery, while only 5% reported a negative result. The impacts of these research findings were most often to inform future research strategies and translate basic science discoveries into novel diagnostic and treatment methods. The 107 completed research grants resulted in over 1000 presentations at scientific conferences,

scientific abstracts, and peer-reviewed journal publications. For fellowship grants that aim to attract new scientists to the field of autism, 88% of fellows reported that it was their first experience in autism research and 95% intended to stay in the field. Finally, for each dollar Autism Speaks invested in these grants, investigators secured \$10 in additional funding, with close \$100 million dollars in leveraged funding to date including over \$77 million in federal grants.

Dissemination of new knowledge and building upon existing findings are critical to maximizing the impact of Autism Speaks' research investments and to accelerating the pace of scientific discovery. To ensure that new knowledge resulting from Autism Speaks-supported research can be accessed, read, applied, and built upon, the organization expects its researchers to publish their findings in peer-reviewed journals. It is a condition of Autism Speaks' Public Access Policy that all peer-reviewed articles supported in whole or in part by its grants must be made available in the PubMed Central online archive.

Science Programs and Initiatives

In addition to investigator-initiated research grants, Autism Speaks supports a number of targeted clinical programs and initiatives. The Autism Treatment Network (ATN) is the first network devoted to addressing the medical conditions associated with ASD and providing comprehensive care. With the help of \$12 million in federal funding, the ATN is developing national standards for the medical treatment of ASD across 17 sites in the United States and Canada. The Autism Genome Project – a collaboration of 120 scientists from 19 countries – uses Autism Speaks genetic database (Autism Genetic Resource Exchange) and brain bank (Autism Tissue Program) to identify new genes that contribute to autism risk, leading to multiple discoveries that impact the understanding of the biology and treatment of autism. The Toddler Treatment Network and High Risk Baby below Siblings Research Consortium are collaborations of 23 scientists from 19 universities who have developed guidelines for early recognition of infants at risk and early intervention approaches for young toddlers

with autism. Autism Speaks funded the launch of the Interactive Autism Network (IAN), the first national online autism registry, which is accelerating autism research by linking more than 10,000 registered families to researchers nationwide. As part of its international development efforts, Autism Speaks launched the Global Autism Public Health Initiative (GAPH), an ambitious advocacy effort that aims to increase autism awareness, enhance capacity and explore unique opportunities in research, and improve service delivery worldwide. Through this effort, Autism Speaks supported the translation and adaptation of diagnostic instruments in languages spoken by 1.75 billion people across the globe. Great advances in the understanding of autism's biology have led Autism Speaks to dedicate increased emphasis to translational research. Their translational research program seeks to accelerate the pace at which basic scientific discoveries are translated into new and effective ways of diagnosing, and treating autism spectrum disorders. This includes "bench to bedside" investigations that move the most promising medicines and other interventions from the laboratory into clinical trials in real world settings such as hospitals, clinics and communities – with the goal of improving outcomes for individuals on the autism spectrum.

Awareness

Autism Speaks' award-winning "Learn the Signs" campaign with the Ad Council has received more than \$258 million in donated media and helped raise awareness of autism to unprecedented levels. Through collaboration between the State of Qatar and Autism Speaks, the UN sanctioned a World Autism Awareness Day to be celebrated in perpetuity on April 2, one of only three disease-specific awareness days of its kind. Autism Speaks celebrates World Autism Awareness Day through its "Light It Up Blue" initiative that has featured the illumination of major US and international landmarks in blue light, including the Empire State Building, Niagara Falls, and the Kingdom Tower in Riyadh, Saudi Arabia. Autism Speaks' web site, autismspeaks.org, has grown to be the most comprehensive and most visited website on autism with over 2.7 million visitors in 2010.

Walk Now for Autism Speaks awareness and fundraising events are held in more than 80 cities across North America, and more than 350,000 individuals participated in 2010.

Family Services

Autism Speaks has provided to families easily accessible and understandable tools and resources for the autism community. The 100 Day Kit – available in English and Spanish – provides a roadmap for newly diagnosed families on how to move forward effectively during the first 100 days following diagnosis. The Asperger/High-Functioning Autism Kit assists families in getting the critical information they need in the first 100 days after a diagnosis specific to Asperger syndrome. The School Community Tool Kit assists members of the school community in understanding and supporting students with autism. Most recently developed, the Transition Tool Kit is a guide to assist families on the journey from adolescence to adulthood. The Autism Video Glossary is a free web-based tool to help parents and professionals learn more about the early warning signs of autism. An online Resource Guide provides families with almost 30,000 resources on everything from diagnosis and treatment centers to autism-friendly barbers. Autism Speaks' Family Services Community Grants program has thus far funded nearly \$3 million to expand innovative and effective community services around the country for people with autism of all ages. The organization is a primary organizer of Advancing Futures for Adults with Autism, which is working to prioritize the needs for adults with autism in order to develop a national policy agenda.

Advocacy

Autism Speaks has played a leading role at the federal and state levels to advocate for legislation that benefits people with autism and their families. The Combating Autism Act of 2006 authorized nearly \$1 billion in autism research and support, and current efforts are focusing on reauthorizing and expanding research and service funding at the federal level. Among the organization's key goals for the next 5 years is to fight for legislation that

will end autism insurance discrimination in all 50 states, as well as at the federal level. Thirty-one states now require insurance companies to cover evidence-based medically necessary autism treatments, including behavioral health treatments, with legislation pending in about ten additional states. It also plans to work with the federal government to set a national policy agenda for services and support of adults with autism.

Major Activities

Research Grant Programs

Autism Speaks offers many types of grants that target critical areas of autism research. The goal is to facilitate and promote efforts that will produce significant findings to lead to discoveries of the causes and development of treatments and improvements in the lives of people with autism.

- *Pilot Research Grants* stimulate the exploration of new avenues of research through 2-year awards aimed at testing novel ideas related to autism. These grants serve to bring new investigators into the field and allow researchers to collect preliminary data, which can permit them to compete for larger grants in future.
- *Treatment Research Grants* address the urgent need to develop effective therapies to treat those living with the disorder today by supporting research focused on all aspects of treatment, including behavioral, psychosocial, biomedical, and technological interventions.
- *Basic and Clinical Research Grants* build upon established research in a broad range of autism-related areas. They provide researchers with larger awards in order to pursue leads that have already shown promise in pilot studies.
- *Dennis Weatherstone Predoctoral Fellowships* are awarded to support highly motivated graduate students with an interest in devoting their careers to autism research.
- *Postdoctoral Fellowships in Translational Autism Research* are designed to support promising, well-qualified postdoctoral scientists in their pursuit of research training that involves translation of biological discoveries toward

novel and more effective methods for treating or diagnosing ASD. This is accomplished by encouraging multidisciplinary collaboration among basic scientists, applied researchers, and clinicians.

- *Suzanne and Bob Wright Trailblazer Awards* are designed to accelerate the pace of autism science. In commemoration of Autism Speaks' fifth anniversary and to honor the organization's pioneering cofounders, the Trailblazer Award is designed to respond quickly in funding highly novel projects with the potential to be transformative and/or to overcome significant research roadblocks.

Science Initiatives

As important as individual grants, initiative projects give Autism Speaks a much more proactive role in promoting specific research. Initiatives frequently involve formation of collaborative research efforts, support of targeted research, organization of research meetings, and creation of research resources.

- The Autism Genome Project (AGP) is the largest study ever conducted to find the genes associated with inherited risk for autism. The ultimate goal is to enable doctors to biologically diagnose autism and researchers to develop universal medical treatments and a cure.
- The *International Autism Epidemiology Network (IAEN)* is an effort to understand the prevalence and causes of autism, particularly across diverse genetic and cultural settings. The activities of this network led to a multinational registry program to examine pre and perinatal factors associated with autism in the largest cohort of children with autism to date.
- The *Global Autism Public Health Initiative (GAPH)* aims to increase public and professional awareness of autism spectrum disorders worldwide, to enhance research expertise and international collaboration, and to improve service delivery in underserved populations.
- The *Environmental Factors in Autism Initiative* targets research that seeks to understand

and identify the potential role environmental factors play in triggering autism.

- The *Innovative Technology for Autism Initiative* was established to lead in the development of products that provide real world solutions to issues faced by those with autism, their families, educators, healthcare specialists, and researchers.
- The *High Risk Baby Siblings Research Consortium (BSRC)* aims to accelerate the understanding of the earliest markers of autism by bringing together the major research groups in the field to investigate infant siblings of children with ASD, including studying the heterogeneity of symptoms and developing best clinical practices.

Clinical Programs

Autism Speaks' clinical programs assist the research community in a variety of ways and include the following:

- The *Autism Genetic Resource Exchange (AGRE)* is a repository (gene bank) of genetic and clinical information from families with two or more members diagnosed with an ASD that is made available to autism researchers worldwide. For over 10 years, AGRE has accelerated the pace of autism research by collecting genetic and clinical data and providing it to researchers, allowing them to focus efforts on their investigations rather than data collection. www.agre.org
- The *Autism Tissue Program (ATP)* is dedicated to increasing and enhancing the availability of postmortem brain tissue to as many qualified scientists as possible to advance autism research. Brain tissue allows scientists to go far beyond the constraints of other technologies and study autism on both a cellular and molecular level. www.autismtissueprogram.org
- The *Autism Treatment Network (ATN)* is a network of hospitals and medical centers working together to improve the quality of care for individuals with autism. The clinicians in the ATN provide comprehensive, coordinated, multidisciplinary care to families in their communities, and are dedicated to establishing standards of care for autism that can be shared

across the wider medical community. www.autismspeaks.org/atn

- The *Autism Clinical Trials Network (ACTN)* is a collaboration of medical and research centers working together on clinical trials of promising pharmaceutical or nutritional treatments for autism. The ACTN approach enables sites to enroll children around the country in a single study, allowing sites to reach recruitment goals in a much shorter amount of time and accelerating progress toward scientifically proven treatments. www.autismspeaks.org/ctn
- The *Interactive Autism Network (IAN)* is an innovative online project designed to accelerate the pace of autism research by linking researchers and families. In addition, families of children with an ASD can share information in a secure online setting and become part of the nation's largest online research effort. www.ianproject.org

To learn more about Autism Speaks, please visit www.AutismSpeaks.org.

Autism Spectrum Addendum (ASA)

► [Autism Spectrum Addendum to the Anxiety Disorders Interview Schedule-Parent Interview](#)

Autism Spectrum Addendum to the Anxiety Disorders Interview Schedule-Parent Interview

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Synonyms

[Anxiety disorders interview schedule \(ADIS\);](#)
[Autism spectrum addendum \(ASA\)](#)

Description

The Autism Spectrum Addendum (ASA) is a series of guidelines, prompts, and questions that can be woven into the Anxiety Disorders Interview Schedule-IV-Child/Parent (ADIS-IV-C/P; Silverman and Albano 1996), a semi-structured diagnostic interview of childhood anxiety disorders and related condition (e.g., obsessive-compulsive disorder [OCD], major depressive disorder, attention deficit hyperactivity disorder) to better tailor it for children with autism spectrum disorder (ASD). For brevity, we will refer to the combined tools as the ADIS/ASA. The purpose of the ASA is: (1) to provide systematic guidelines for differentiating potentially overlapping anxiety and autism spectrum symptoms, such as social avoidance, and (2) to capture distinct worries and fears that arise in ASD but do not fit traditional categories of anxiety as outlined in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, TR (DSM-IV-TR; American Psychological Association 2000), or the more recent, DSM, Fifth Edition (DSM-V; American Psychological Association 2013). The ADIS-IV-C/P assess for DSM anxiety disorders with a series of diagnostic modules. In each module, DSM criteria are queried with a mandatory set of questions, about which the interviewer may follow-up as needed to clarify a participant's meaning, ensure their understanding of the question, and gather more information. In addition, for modules in which symptoms are endorsed, interviewers provide clinician severity ratings (CSR) from 0 (not severe) to 8 (extremely severe), with 4 being the cut-off for diagnosis and 1–3 used to indicate subclinical symptoms. Typically, the ADIS-IV-C/P is administered to both children and parents and diagnoses are based on composite ratings, that is, the highest CSRs provided for each diagnostic module by either the parent or the child interviewer. Though having multiple informants is preferable whenever possible in clinical assessment, initial development of the ASA has focused on the parent interview – the ADIS-IV-P – given research suggesting that child report may be less reliable in children with ASD (Storch et al. 2012) or, in the case of children without communicative language, impossible to collect.

In the ADIS/ASA, the ADIS-IV-P is administered in its standard form with the ASA content woven in to guide interviewer's follow-up questions, systematically gather information important for differentiating anxiety and ASD symptoms, and gather information about distinct presentations of anxiety that may be reported. ASA content supports the assessment of Separation Anxiety Disorder, Social Anxiety Disorder, Specific Phobia, Generalized Anxiety Disorder and OCD. Though the ADIS-IV-P is designed to assess DSM-IV-TR anxiety disorders, it should be noted that the specific criteria for these disorders were not substantially altered in DSM-V (Kupfer 2015; APA 2000, 2013).

In addition to these DSM-consistent diagnoses, the ASA queries for distinct fears and worries that may arise in ASD, including fears of social situations that do not reflect a fear of negative evaluation (referred to as "other social fears"), uncommon phobias (e.g., fears of toilets, mechanical things, men with beards, specific songs), fears of change, and worries related to preoccupations. The ASA also assesses for compulsive behavior (e.g., insistence that doors remain closed or sleeves rolled up) that is associated with distress, but does not clearly have a compensatory function, as is the case for rituals and compulsions in OCD. These symptoms are referred to as Ambiguous OCD. Like the standard ADIS-IV-P modules, these other categories of anxiety, if reported, are assigned a CSR from 0 to 8, with 4 representing the cut off for clinically significant symptoms and 0–3 used for subclinical concerns.

Additional items designed to support differential diagnosis and the assessment of these distinct presentations include an expanded interpersonal relationships sections to assess children's attainment of reciprocal friendships, history of bullying, and levels of social motivation and social awareness. These items provide clinicians with a greater sense of whether social avoidance in a child with ASD may be attributable to social anxiety disorder, other social fears (i.e., fears related to nonverbal communication difficulties), or simply low social interest. They also allow clinicians to assess the extent to which fears of negative evaluation may be adaptive and proportionate rather than maladaptive and excessive (a requirement for

diagnosis). Additional differential diagnosis items include queries into levels of sensory sensitivity and perseverative thinking in each child, which are used to ensure that these ASD-related difficulties are considered and not misinterpreted as symptoms of specific phobia (e.g., phobia of loud sounds) or generalized anxiety. Each differential item is rated on a 0–3 scale, with 0 representing an absence of difficulties or deficits and 3 indicating significant difficulties.

Historical Background

Symptoms of anxiety have long been reported in children with ASD, so much so that anxiety is described as an associated feature of the disorder in the DSM-V and prior version (APA 2000, 2013) as well as the original case descriptions of ASD by Kanner (1943). Nonetheless, the diagnosis of co-occurring anxiety disorders in ASD is not straightforward, given that many symptoms of anxiety are also characteristic of ASD (e.g., social avoidance, ritualistic behavior, perseverative thinking, arousal dysregulation; Kems et al. 2016; Vasa et al. 2016). In addition, deficits in communication in ASD may complicate the assessment of anxiety symptoms, some of which are dependent on language (e.g., worries). Finally, a growing body of research suggests that children and adults with ASD experience fears and worries that are both similar and dissimilar to those of individuals in the general population (Adams et al. 2019; Den Houting et al. 2018; Halim et al. 2018; Kerns et al. 2014, 2017; Magiati et al. 2017; Scahill et al. 2019). Specific, distinct sources of fear include anxiety related to small changes in the environment, nonthreatening items, sounds, or sights (e.g., beards, glasses, specific songs), anxiety linked to a perseverative interests and anxiety related to difficulties understanding social cues and expectations. Whether the fears and worries of a child with ASD should be considered clinically significant as opposed to normative can also be difficult to determine given that the developmental level of children with ASD may not match their chronological age and because children with ASD often experience stressors, such as bullying and academic difficulties, about which some anxiety would be appropriate.

Given these challenges, it is perhaps unsurprising that estimates of the rates and associated characteristics of anxiety disorders in ASD have varied widely in the research literature (11–84% across studies) and even among studies employing diagnostic interviews (39–84%; Kerns and Kendall 2012; Van Steensel et al. 2011; White et al. 2009). This is likely due, in part, to inconsistency around the differential diagnosis, conceptualization, and measurement of anxiety disorders in ASD across these studies. The ADIS/ASA was developed to address this inconsistency, by providing a systematic and guided approach to differential diagnosis of anxiety in ASD and measuring distinct or ASD-related fears and anxieties as opposed to DSM-anxiety disorders alone. Notably, though a meta-analysis of studies assessing only DSM-consistent symptoms concluded that approximately 39.6% of youth with ASD present with clinically significant anxiety (Van Steensel et al. 2011), more recent studies suggest this rate may be closer to 63–69% when distinct as well as traditional fears are assessed (Kerns et al. 2014, 2020; Den Houting et al. 2018).

Psychometrics

The reliability and validity of an initial version of the ADIS/ASA was first examined in a sample of 59 youth, ages 7–17 years with ASD, who were recruited as part of a neuroimaging study of ASD (i.e., not selected for anxiety; Kerns et al. 2014). In this version, both the child and parent interviews were conducted and final diagnoses reflected the composite CSR from both reports. Support was found for the convergent and discriminant validity of the interview. Both traditional and distinct anxiety CSR were significantly correlated with other measures of anxiety, including the Screen for Child Anxiety and Related Emotional Disorders (SCARED; Birmaher et al. 1999) Total Score and Behavior Assessment System for Children-Second Edition Anxiety subscale (BASC-2; Reynolds and Kamphaus 2004). By comparison, neither traditional nor distinct anxiety CSR were significantly associated with externalizing behavior or daily living skills as measured by the BASC-2. Distinct

anxiety CSR, but not traditional anxiety CSR, were significantly associated with each other, but had distinct relationships to other measures of autism-related symptoms. Specifically, only distinct anxiety was significantly associated with higher levels of autism symptoms (Social Responsiveness Scale, Total Score; Constantino and Gruber 2012) and atypical behaviors (BASC-2 atypicality subscale). Inter-rater reliability, assessed in 35% of the sample, suggested good agreement between evaluators on both diagnoses (percent exact agreement: 95–100%) and CSR (*Intraclass Correlation [ICC]* = 0.89–0.99). Retest reliability, assessed in 25% of the sample, indicated that diagnoses (100% exact agreement) and CSR were also consistent over an approximately 2-week time period (0.88–1.00).

Results from this study were used to refine the parent version of the ASA to create the current tool, the psychometrics of which were examined in a new sample of 79 children ages 9–13 years with ASD and no more than mild intellectual impairments (IQ range: 68–143) who were seeking treatment for anxiety as part of a randomized clinical trial (Kerns et al. 2017). In contrast to Kerns et al. (2014), this sample included a larger proportion of children with ASD and co-occurring anxiety disorders, allowing for a more rigorous test of the inter-rater reliability of the different types of anxiety assessed by the ADIS/ASA modules and CSR. Findings again suggested good agreement between independent raters about both DSM-anxiety diagnoses (Cohen's Kappa [*K*] = 0.67–0.91) and CSR (*ICC* = 0.85–0.98) as well as the clinical significance (*K* = 0.77–0.90) and CSR (*ICC* = 0.87–0.95) of distinct anxieties. Support was also found for the discriminant validity of the ADIS/ASA, with partial support for convergent validity. In keeping with Kerns et al. (2014), neither ADIS/ASA traditional nor distinct anxiety CSR were significantly correlated with measures of parent-reported attention or aggression difficulties in the child (as measured by the Child Behavior Checklist; Achenbach et al. 2001). In addition, distinct but not traditional anxiety was associated with a measure of ASD severity (Autism Diagnostic Observation Schedule Comparison Score; Gotham et al. 2009). In contrast to Kerns et al. (2014), traditional but not distinct anxiety CSR were

associated with other measures of parent-reported anxiety (Child Behavior Checklist DSM Anxiety Subscale). Cumulatively these results suggest that the association of traditional anxiety measures and distinct anxiety may vary depending upon sample characteristics and recruitment methods.

Clinical Uses

The ADIS/ASA is designed to help clinicians determine whether a child with ASD meets criteria for a DSM anxiety disorder, other forms of clinically significant anxiety, and OCD. As it is a diagnostic tool, it should only be administered by those with or receiving training in child development and psychological evaluation. Given that administration is typically between 1.5 to 3 h (Mean = 105.36 min, Standard Deviation = 30.32 min; Kerns et al. 2017), the ADIS/ASA is not recommended as a primary or universal screening measure, but rather as a tool for differential diagnosis in children with complex case presentations or when precise clinical characterization or behavioral phenotyping is the goal. Importantly, information on the psychometric properties of the ADIS/ASA has only been published on samples of children with ASD between the ages of 7 and 17 years with no more than mild intellectual impairments. The ADIS/ASA may also provide a useful conceptual framework for clinicians assessing anxiety in preschoolers and children with ASD and more severe intellectual impairments; however, research to support its use in these populations is still ongoing. Similarly, though prior research suggests that the ADIS-IV-P is sensitive to changes in anxiety in children with ASD due to treatment (Reaven et al. 2012; Weiss et al. 2018), whether this also the case for the ADIS/ASA is being studied (see Kerns et al. 2016).

References and Reading

- Achenbach, T. M., Dumenci, L., & Rescorla, L. A. (2001). *Ratings of relations between DSM-IV diagnostic categories and items of the CBCL/6–18, TRF, and YSR*. Burlington: University of Vermont.
- Adams, D., Young, K., Simpson, K., & Keen, D. (2019). Parent descriptions of the presentation and management of anxiousness in children on the autism spectrum. *Autism*, 23(4), 980–992.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders – Fourth edition, text revision*. Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: Author.
- Birmaher, B., Brent, D. A., Chiappetta, L., Bridge, J., Monga, S., & Baugher, M. (1999). Psychometric properties of the screen for child anxiety related emotional disorders (SCARED): A replication study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 38(10), 1230–1236.
- Constantino, J. N., & Gruber, C. P. (2012). Social responsiveness scale: SRS-2 software kit. Western Psychological Services.
- Den Houting, J., Adams, D., Roberts, J., & Keen, D. (2018). Exploring anxiety symptomatology in school-aged autistic children using an autism-specific assessment. *Research in Autism Spectrum Disorders*, 50, 73–82.
- Gotham, K., Pickles, A., & Lord, C. (2009). Standardizing ADOS scores for a measure of severity in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(5), 693–705.
- Halim, A. T., Richdale, A. L., & Uljarević, M. (2018). Exploring the nature of anxiety in young adults on the autism spectrum: A qualitative study. *Research in Autism Spectrum Disorders*, 55, 25–37.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2(3), 217–250.
- Kerns, C. M., & Kendall, P. C. (2012). The presentation and classification of anxiety in autism spectrum disorder. *Clinical Psychology: Science and Practice*, 19(4), 323–347.
- Kerns, C. M., Kendall, P. C., Berry, L., Souders, M. C., Franklin, M. E., Schultz, R. T., ... Herrington, J. (2014). Traditional and atypical presentations of anxiety in youth with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(11), 2851–2861.
- Kerns, C. M., Wood, J. J., Kendall, P. C., Renno, P., Crawford, E. A., Mercado, R. J., ... Small, B. J. (2016). The treatment of anxiety in autism spectrum disorder (TAASD) study: Rationale, design and methods. *Journal of Child and Family Studies*, 25(6), 1889–1902.
- Kerns, C. M., Renno, P., Kendall, P. C., Wood, J. J., & Storch, E. A. (2017). Anxiety disorders interview schedule-autism addendum: Reliability and validity in children with autism spectrum disorder. *Journal of Clinical Child & Adolescent Psychology*, 46(1), 88–100.
- Kerns, C. M., Winder-Patel, B., Iosif, A. M., Nordahl, C. W., Heath, B., Solomon, M., & Amaral, D. G. (2020). Clinically significant anxiety in children with autism spectrum disorder and varied intellectual functioning. *Journal of Clinical Child & Adolescent Psychology*, 1–16.

- Kupfer, D. J. (2015). Anxiety and DSM-5. *Dialogues in clinical neuroscience*, 17(3):245.
- Magiati, I., Ozsivadjian, A., & Kerns, C. M. (2017). Phenomenology and presentation of anxiety in autism spectrum disorder. In *Anxiety in children and adolescents with autism spectrum disorder* (pp. 33–54). Boston: Academic Press.
- Reaven, J., Blakeley-Smith, A., Culhane-Shelburne, K., & Hepburn, S. (2012). Group cognitive behavior therapy for children with high-functioning autism spectrum disorders and anxiety: A randomized trial. *Journal of Child Psychology and Psychiatry*, 53(4), 410–419.
- Reynolds, C. R., & Kamphaus, R. W. (2004). *Behavior assessment system for children – Second edition*. Circle Pines: AGS.
- Scahill, L., Lecavalier, L., Schultz, R. T., Evans, A. N., Maddox, B., Pritchett, J., ... & Aman, M. G. (2019). Development of the parent-rated anxiety scale for youth with autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 58(9), 887–896.
- Silverman, W. K., & Albano, A. M. (1996). The Anxiety Disorders Interview Schedule for DSM-IV: Child and Parent Versions. *The Psychological Corporation TX*: San Antonio.
- Storch, E. A., Ehrenreich May, J., Wood, J. J., Jones, A. M., De Nadai, A. S., Lewin, A. B., ... Murphy, T. K. (2012). Multiple informant agreement on the anxiety disorders interview schedule in youth with autism spectrum disorders. *Journal of Child and Adolescent Psychopharmacology*, 22(4), 292–299.
- Van Steensel, F. J., Bögels, S. M., & Perrin, S. (2011). Anxiety disorders in children and adolescents with autistic spectrum disorders: A meta-analysis. *Clinical Child and Family Psychology Review*, 14(3), 302.
- Vasa, R. A., Mazurek, M. O., Mahajan, R., Bennett, A. E., Bernal, M. P., Nozzolillo, A. A., ... & Coury, D. L. (2016). Assessment and treatment of anxiety in youth with autism spectrum disorders. *Pediatrics*, 137(Supplement 2), S115–S123.
- Weiss, J. A., Thomson, K., Burnham Riosa, P., Albaum, C., Chan, V., Maughan, A., ... & Black, K. (2018). A randomized waitlist-controlled trial of cognitive behavior therapy to improve emotion regulation in children with autism. *Journal of Child Psychology and Psychiatry*, 59(11), 1180–1191.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229.

Autism Spectrum Condition (ASC)

- [Self-Report Autism Scales for Adults](#)

Autism Spectrum Disorder

- [Social Learning Disability/Difference](#)

Autism Spectrum Disorder (ASD)

- [Asperger Syndrome](#)
- [Autism Family Experience Questionnaire \(AFEQ\)](#)
- [Autism Family Experience Questionnaire \(AFEQ\), The](#)
- [DiBAS-R Validation](#)

Autism Spectrum Disorder and PDA Traits

- [Pathological Demand Avoidance \(PDA\)](#)

Autism Spectrum Disorders

- [Pervasive Developmental Disorder](#)

Autism Spectrum Rating Scale

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Synonyms

[ASRS](#)

Description

The Autism Spectrum Rating Scales (ASRS) (Goldstein and Naglieri 2009) are designed to measure behaviors reported by parents and/or teachers

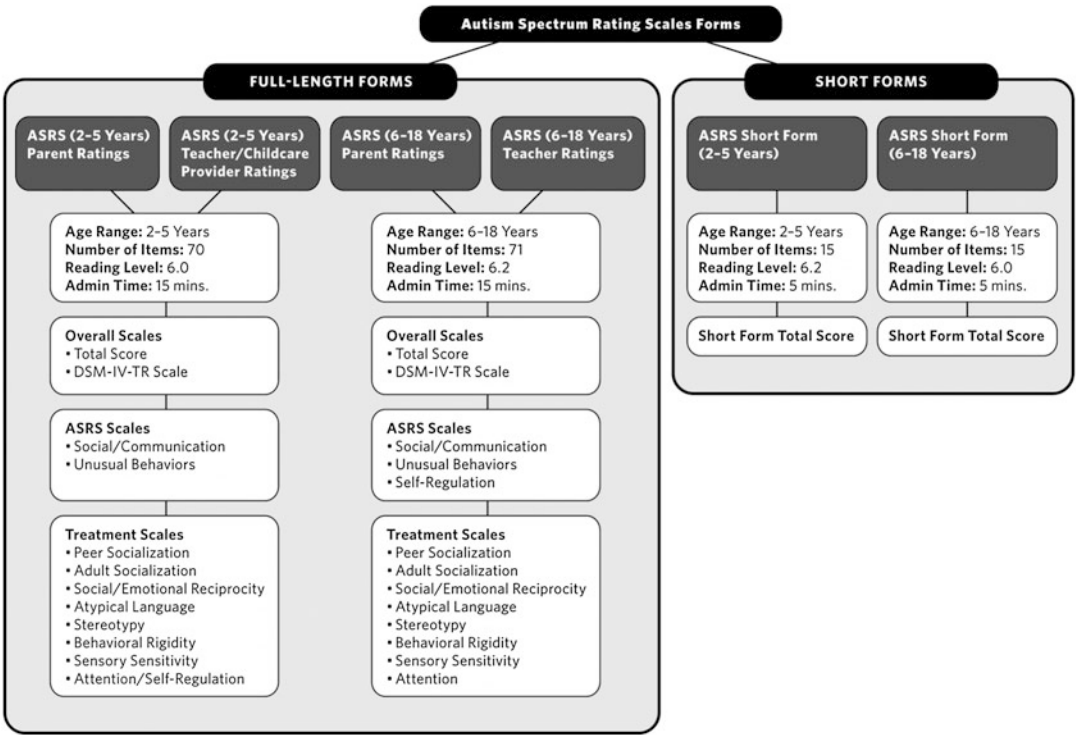
associated with autism spectrum disorders (ASDs) for children and youth aged 2 through 18 years. The ASRS can help guide diagnostic decisions and can be used during treatment planning, ongoing monitoring of response to intervention, and program evaluation. The ASRS includes items related to DSM-IV-TR autistic disorder, Asperger’s disorder, pervasive developmental disorder—not otherwise specified (PDD – NOS), and DSM 5 autism spectrum disorder. As recognition and prevalence of these conditions increase, the risk of over- and underdiagnosis increases in parallel. The need for a valid, reliable, and carefully crafted tool for assessment becomes paramount.

As illustrated in Fig. 1, the ASRS has full-length and short forms for both young children aged 2 to 5 years and youth aged 6–18 years. The full-length ASRS (2–5 years) comprises 70 items, and the full-length ASRS (6–18 years) consists of 71 items. There are separate parent (ASRS parent ratings) and teacher (ASRS teacher ratings) forms for both age groups. The ASRS short forms were developed by selecting the items that best differentiate

between nonclinical youth and youth diagnosed with ASD. The ASRS short form (2–5 years) and ASRS short form (6–18 years) both contain 15 items, and parents and teachers/caregivers complete the same form. All scales are set to the *T*-score metric, which has a normative mean of 50 and standard deviation of 10.

All of the ASRS forms are available in the MHS QuikScore format. The rater writes on the external layers of the form, and the results are transferred to a hidden scoring grid within the internal layers. The assessor then uses the internal layers for tabulating and profiling results. Each ASRS QuikScore form includes profile sheets, which are used to convert raw scores to *T*-scores and percentiles. These profile sheets also include a chart where scores can be plotted for a graphic display of the results. For individuals who wish to use software or online scoring, ASRS items are also provided in a response booklet format that does not include the scoring pages.

The ASRS can be completed and automatically scored online wherever an Internet connection is



Autism Spectrum Rating Scale, Fig. 1 ASRS Scales and Forms

available. Paper-and-pencil forms can also be scored online by entering responses from a paper-and-pencil administration into the online program. All ASRS forms can be scored using the scoring software by entering responses from a completed paper-and-pencil administration into the software program.

In some instances, the assessor may wish to obtain information about a group of youth instead of an individual. In a preschool or school setting, the ASRS can be used to screen a group of children to determine which children might require a full evaluation or to identify children who might benefit from additional support. The ASRS short forms have excellent reliability and validity, are good predictors of the ASRS total score, and were developed for screening purposes. High scores suggest that additional considerations are needed. For example, high scores on the short form might indicate the need for further examination with the full-length form, a more thorough evaluation, and/or some treatment to modify troubling behaviors.

Results from the ASRS can inform decisions about the effectiveness of a particular individual or group intervention. When used in a clinical setting, ASRS results can be collected at the beginning of an intervention and at several points throughout the intervention (in intervals of 4 weeks) in order to evaluate whether a particular program is associated with symptom improvement. In research studies, group data from the ASRS can be analyzed to determine whether change (pre- versus posttreatment or experimental treatment versus control group) is significant. Results from these types of evaluations can be helpful in supporting the need for the continuation of a treatment program or line of research.

ASRS reports can be obtained using the software or online scoring option. There are three report types for all of the ASRS forms: the interpretive report (provides detailed results from one administration), the comparative report (provides a multi-rater perspective by combining results from up to five different raters), and the progress monitoring report (provides an overview of change over time by combining results of up to four administrations from the same rater).

Historical Background

The ASRS development project encompassed 5 years (2004 to 2009), thousands of ratings by parents and teachers, intensive research, sophisticated statistical analyses, and multiple data collection sites. Development of the ASRS occurred in three phases: (1) conceptualization/initial planning, (2) pilot study, and (3) final scale construction (including the normative study). The ASRS was originally conceptualized as an assessment tool that would assess autism spectrum disorder (ASD) symptoms from early childhood through adolescence; therefore, the initial age range of the assessment was 2–18 years. Because of the importance of multi-informant assessment, it was determined from the onset that both parent and teacher forms would be created. Since great importance was placed on the ability to compare results across different raters, a decision was made to include identical items on both the parent and teacher forms.

A comprehensive review of current theory combined with literature on the assessment of ASDs, the DSM-IV-TR and ICD-10 diagnostic criteria, and the authors' experiences were used to determine the preliminary content structure. This structure guided item generation, and multiple items were developed to capture key components of each construct. A DSM-5 scale was added in 2013 as well as an accommodated scoring profile for nonverbal children in 2012.

Psychometric Data

Development of the final scale involved normative and clinical data collection, factor analyses to determine the factor structure of the forms, the creation of the total score, the DSM-IV-TR scale, and the treatment scales. The final scale construction began with the collection of normative and clinical data. The normative samples include 2,560 ratings (640 for the 2–5-year-olds rated by parents and teachers/childcare providers and 1,920 for the 6–18-year-olds also rated by parents and teachers). These samples include ratings of 40 males and 40 females at each age and are representative of the US population across several

demographic variables. The clinical samples include nearly 700 ratings of youth diagnosed with ASD and over 500 ratings of youth diagnosed with other clinical disorders (including delayed cognitive development, delayed communication development, ADHD, anxiety disorders, depressive disorders, and language disorders).

In order to examine the underlying factor structure of the ASRS items, data from both the normative and clinical samples were used in exploratory factor analyses (via principal axis extraction and direct oblimin rotation). Results of these analyses suggested that a two-factor model was most suitable for both the parent and teacher ASRS (2–5 years) forms, while a three-factor model was most suitable for the parent and teacher ASRS (6–18 years) forms. These factor-derived scales were labeled the “ASRS Scales” and include social/communication and unusual behaviors on all forms, as well as self-regulation on the ASRS (6–18 years).

In order to ensure that there was no redundancy in the scales, the ASRS Scale scores were intercorrelated (i.e., redundancy would be implied if the correlations were very high) on the total sample (i.e., the normative plus the clinical sample). Results indicated that the scale intercorrelations met theoretical expectations (i.e., they were moderate in size), providing additional support for the multidimensionality of the measure.

Clinical Uses

The ASRS can be used as an aid in the diagnostic process. Standardized scores from the ASRS allow the assessor to effectively compare an individual to a norm group in an objective and reliable manner. Scores can be integrated with other information to form a complete picture of the individual. When used in combination with other assessment information, results from the ASRS can help guide diagnostic decisions, treatment planning, and ongoing monitoring of response to intervention. The ASRS can also be used to evaluate the effectiveness of a treatment program for a child with ASD.

The ASRS can also be a tool for researchers in a variety of settings and research protocols. There

are several advantages the ASRS offers to researchers. First, the scales were carefully developed to measure a wide spectrum of behaviors associated with ASD. Second, the various scales included provide scores based upon a normative sample aged 2–18 years based on a diverse, representative group of individuals. Third, the scales included on the ASRS have demonstrated reliability, which is particularly important in correlational studies, and validity, which is particularly important for both internal and external validities of any research project. Fourth, the psychometric qualities of the scale are well documented in this manual. Fifth, comparisons to other instruments are easier due to the availability of standard scores.

The ASRS was carefully developed and researched to provide the most useful set of items for ASD identification and intervention. Any rating scale has inherent limitations; however, when used appropriately, the ASRS is a useful tool in the entire process of defining the problem, eliciting information from parents and teachers, planning treatment and intervention, and measuring treatment outcome in ASD.

See Also

- ▶ [Autism Diagnostic Observation Schedule \(ADOS\): Toddler Module](#)
- ▶ [Childhood Autism Rating Scale](#)
- ▶ [DSM-5](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- Biederman, J., Petty, C. R., Fried, R., Wozniak, J., Micco, J. A., Henin, A., Doyle, R., Joshi, G., Galdo, M., Kotarski, M., Caruso, J., Yorks, D., & Faraone, S. V. (2010). Child behavior checklist clinical scales discriminate referred youth with autism Spectrum disorder: A preliminary study. *Journal of Developmental and Behavioral Pediatrics*, 31, 485–490.

- Chlebowski, C., Green, J. A., Barton, M. L., & Fein, D. (2010). Using the childhood autism rating scale to diagnose autism Spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(7), 787–799.
- Cohen, D. J., & Volkmar, F. R. (1997). *Handbook of autism and pervasive developmental disorders*. New York: Wiley.
- DeVincent, C. J., & Gadow, K. (2009). Relative clinical utility of three child symptom inventory-4 scoring algorithms for differentiating children with autism Spectrum disorder vs. attention-deficit hyperactivity disorder. *Journal of Autism Research*, 2(6), 312–321.
- Factor, D. C., Freeman, N. L., & Kardash, A. (1989). A comparison of DSM-III and DSM-III-R criteria for autism. *Journal of Autism and Developmental Disorders*, 19, 637–640.
- Frith, U. (2004). Confusions and controversies about Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 45, 672–686.
- Gillberg, C., & Steffenberg, S. (1987). Outcome and prognostic factors in infantile autism and similar conditions: A population-based study of 46 cases followed through puberty. *Journal of Autism and Developmental Disorders*, 17, 273–287.
- Goldstein, S., & Naglieri, J. (2009). *Autism Spectrum rating scale*. Toronto: Multi-Health Systems.
- Goldstein, S., & Naglieri, J. A. (2010). *The autism Spectrum rating scales*. Toronto: Multi-Health Systems.
- Goldstein, S., & Naglieri, J. A. (2011). Test review: ASRS: Autism Spectrum rating scales. *Journal of Psychoeducational Assessment*, 29(2), 191–195.
- Goldstein, S., & Naglieri, J. A. (2012). *Technical report #1: Scoring the ASRS for individuals who do not speak or speak infrequently*. Toronto: Multi-Health Systems.
- Goldstein, S., & Naglieri, J. A. (2013). *Interventions for autism Spectrum disorders*. New York: Springer.
- Goldstein, S., & Ozonoff, S. (2018). *Assessment of autism Spectrum disorders* (2nd ed.). New York: Springer.
- Goldstein, S., & Schwabach, A. (2004). The comorbidity of pervasive developmental disorder and attention deficit hyperactivity disorder: Results of a retrospective chart review. *Journal of Autism and Developmental Disorders*, 34(3), 329–339.
- Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The autism diagnostic observation schedule: Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders*, 37(4), 613–627.
- Gotham, K., Risi, S., Dawson, G., Tager-Flusberg, H., Joseph, R., Carter, A., Hepburn, S., McMahon, W., Rodier, P., Hyman, S. L., Sigman, M., Rogers, S., Landa, R., Spence, A., Osann, K., Flodman, P., Volkmar, F., Hollander, E., Buxbaum, J., Pickles, A., & Lord, C. (2008). A replication of the autism diagnostic observation schedule (ADOS) revised algorithms. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(6), 642–651.
- Klin, A., Pauls, D., Schultz, R., & Volkmar, F. (2005). Three diagnostic approaches to Asperger syndrome: Implications for research. *Journal of Autism and Developmental Disorders*, 35, 221–234.
- Kurita, H., Osada, H., & Miyake, Y. (2004). External validity of childhood disintegrative disorder in comparison with autistic disorder. *Journal of Autism and Developmental Disorders*, 34, 355–362.
- Mayes, S. D., Calhoun, S. L., & Crites, D. L. (2001). Does DSM-IV Asperger's disorder exist? *Journal of Abnormal Child Psychology*, 29, 263–271.
- Risi, S., Lord, C., Gotham, K., Crosello, C., Chrysler, C., Szatmari, P., et al. (2006). Information from multiple sources in the diagnosis of autism spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45, 1094–1103.
- Tomanik, S. S., Pearson, D. A., Loveland, K. A., Lane, D. M., & Shaw, J. B. (2006). Improving the reliability of autism diagnoses: Examining the utility of adaptive behavior. *Journal of Autism and Developmental Disorders*, 37(5), 921–928.
- Venter, A., Lord, C., & Schopler, E. (1992). A follow-up study of high-functioning autistic children. *Journal of Child Psychology and Psychiatry*, 33, 489–507.
- Ventola, P. E., Kleinman, J., Pandey, J., Barton, M., Allen, S., Green, J., Robins, D., & Fein, D. (2006). Agreement among four diagnostic instruments for autism Spectrum disorders in toddlers. *Journal of Autism and Developmental Disorders*, 36(7), 839–847.
- Witwer, A. N., & Lecavalier, L. (2007). Autism screening tools: An evaluation of the social communication questionnaire and the developmental behaviour checklist-autism screening algorithm. *Journal of Intellectual and Developmental Disabilities*, 32(3), 179–187.
- World Health Organization. (1993). *The ICD-10 classification of mental and behavioral disorders: Diagnostic criteria for research*. Geneva: World Health Organization.

Autism Theory

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Definition

A multitude of theories have attempted to explain certain cognitive characteristics of autism. Although adherents of some of these theories (such as theory of mind) may once have contended that their particular theory could fully

explain autism, it is generally considered nowadays that an explanation of autism requires a synthesis of theory. Three theories – theory of mind, executive (dys)functioning, and central coherence – assume a special place in the pantheon of cognitive autism theory. In the limited space available, we provide a brief introduction to these three “mainstream” theories together with various other “alternative” theories including the single attention/monotropism hypothesis of Murray et al. (2005) which, arguably, explains more characteristics of autism than any other theory.

More than 70 years after Asperger and Kanner first wrote about autism, there is still no definitive autism theory. We doubt there will ever be a full theoretical explanation of *any* neurotype because of the complexities involved. But we also firmly believe that the theories we introduce here are of great value in describing characteristics of autism that can help autistic individuals to understand themselves better and help those who live with and/or support autistic individuals to understand autism. Improved understanding should lead to better interventions and other support for autistic people.

Cognitive Theories of Autism

Theory of Mind

The theory of mind (ToM) is one of the three “big ideas” of autism theory. ToM refers to an individual’s ability to attribute mental states to themselves and to others (Frith and Happé 1999). Put in another way, ToM is the everyday folk psychology that people use to make sense of other people’s behavior by hypothesizing about the beliefs, desires, and feelings that motivate actions. ToM is a way of describing the need for individuals to develop an understanding that objects and other persons have separate existence, that other persons have their own mental state that differs from theirs, and be able to “put on the shoes” of another person mentally. Predicting another person’s likely behavior can be likened to developing a hypothesis about expected behavior. ToM is often referred to as “mind reading” (Baron-Cohen 1995) although it involves the use of

sensory stimuli to guess the mental state of others and there can be no direct access to another person’s mind in the sense of a psychic power!.

An example should better explain the nature of ToM difficulties. It relates to a 16-year-old boy with Asperger syndrome who we will call James. James cannot see things from the perspective of his 8-year-old sister. Like other children of her age, his sister makes statements and asks questions that he finds ridiculous such as: “I want to go to the moon on holiday” and “I’m going to have 20 children when I grow up.” James cannot ignore things like this, agree, or join in on the same line of thought, as he cannot see these comments for what they are, the things a child much younger than him will say. Instead he has to ridicule what she says and explain in detail why it is impossible for her to go to the moon and that it is extremely unlikely that she will ever have so many children. Naturally, this upsets his sister and can make family life stressful. No amount of parental explanation makes any difference to James; his limited ToM prevents him from putting himself in his sister’s shoes and interpreting her comments from her perspective instead of his own.

ToM probably developed in human beings relatively recently (thousands of years ago) to enable us to cope with a social environment that was becoming increasingly complex as the species developed. Social interaction aided by ToM will have had benefits for both reproduction and survival. A newborn child has no understanding that the world exists independently of itself. The child is unable to form mental representations of persons or objects. At this earliest stage of its life, an object exists for the child only while it is in sight and ceases to exist when out of sight. As the child grows, its developing ToM enables it to form mental representations of other objects and persons, and, later on, it learns that other persons have a thinking existence of their own (Frith and Happé 1999). The ability of typically developing children to evaluate the thoughts, emotions, intentions, and beliefs of others grows over time. While major advances in a child’s ToM take place during preschool years, this ability continues to develop throughout childhood and even into adolescence and adulthood. An autistic young person’s ToM

ability may, at least partially, “catch up” with that of their typically developing peers during their adolescence and adulthood.

Executive (Dys)Functioning

A further theory that attempts to explain aspects of autism is known as executive (dys)functioning. Although executive function (EF) was not defined until the 1970s, the beginnings of this concept date right back to a railway accident in 1840. A man called Phineas Gage – sometimes known as “neuroscience’s most famous patient” – was a railway construction foreman in the USA leading a team cutting a railway bed in the state of Vermont. He suffered a very serious head injury when a premature explosion sent a tamping iron – an iron rod – flying in his direction. The tamping iron was nearly 4 feet long, 1 ¼ in. in diameter, and weighed 13 ¼ pounds. It “penetrated Gage’s left cheek, ripped into his brain, and exited through his skull, landing several dozen feet away.” The rod destroyed a large part of Gage’s left frontal lobe. He survived this dreadful accident, but the severe injuries caused a change in his personality and behavior. He became “disinhibited” or “hyperactive,” as is often found in persons with damage to the prefrontal cortex. It was Gage’s accident in particular that caused the medical profession to consider the role of the frontal lobes in what we now call EF, although real progress in developing this concept did not begin until the 1950s.

Delis (2012, p. 14) writes that “Neither a single ability nor a comprehensive definition fully captures the conceptual scope of executive functions: rather, executive functioning is the sum product of a collection of higher level skills that converge to enable an individual to adapt and thrive in complex psychosocial environments.” Others have defined EF as “an overarching term that refers to mental control processes that enable physical, cognitive, and emotional self-control” (Corbett et al. 2009, p. 210) and “several abilities for preparing and engaging in complex organized behaviour” (Macintosh and Dissanayake 2004, p. 426). Although the main components of EF have yet to be established definitively, they are likely to

include formation of abstract concepts, planning, focusing and sustaining attention, shifting focus, and working memory (Macintosh and Dissanayake 2004; Attwood 1998).

Many studies have demonstrated that persons on the autism spectrum often experience difficulties with executive functioning. However, although these difficulties can be pervasive, they are not all necessarily universal in autism. It is also the case that some executive function processes are often less likely to be affected in autism than others (e.g., difficulty with planning is more common than an inability to inhibit impulsive behavior).

Central Coherence Theory

The third main cognitive theory of autism is the theory of central coherence (CC). This theory, developed by Uta Frith and Francesca Happé (1994), attempts to explain why persons with autism exhibit particular strengths in addition to weaknesses. CC is the ability to see the gist – the so-called big picture – rather than just the detail of which something is comprised. CC theory originally proposed that persons with autism will have what Happé and Frith called weak CC in that there will be a tendency for them to focus on the detail at the expense of being able to see things in the round and generalize. Tony Attwood describes weak CC as being “remarkably good at attending to detail but (having) a weakness in perceiving and understanding the overall picture, or gist” (Attwood 1998, p. 241). In accordance with this theory, it should be possible to see strengths in the manipulation of detail in autism in addition to difficulties in forming a holistic picture from the detail.

In a later development of their CC theory, Happé and Frith (2006) contend that there is a *preference* for detail in autism rather than a weakness in CC. They now write of a difference in information processing style in autism, with concomitant strengths, rather than impairment. Happé and Frith also argue that there is a continuum of CC along which all people fit, with autistic individuals lying at the “detail” end of the continuum.

Single Attention/Monotropism

Murray et al. (2005) have investigated the current diagnostic criteria for autism in the light of their theory that autism has its foundation in what they describe as single attention/monotropism. These authors hypothesize that a limited amount of attention available to all people – autistic or not – plays a fundamental part in day-to-day life and is largely a matter of inheritance. They argue that differences in the spread of attention available to individuals are normally distributed between a wide spread of attention over many interests at one end of the distribution and a much narrower focus of attention on only a few interests at the opposite end. Importantly, they regard the “restricted range of interests” referred to in the DSM-IV and ICD-10 diagnostic criteria, and which they call monotropism (A colleague has suggested that the term “monotropism” would be more appropriate for someone with a *single* interest rather than few interests.), as being central to autism.

Instead of a preference for detail over wholes, Murray, Lesser, and Lawson argue for a state of heightened (hyper)awareness inside an “attention tunnel” and lessened (hypo)awareness outside this tunnel. They consider that their proposed tunnel effect also causes the hypersensitivity and hyposensitivity to sensory experiences often seen in autism. Their proposal is that autistic people are hypersensitive within the attention tunnel and hyposensitive outside the tunnel. The single attention/monotropism theory is one of the few that seeks to explain both cognitive *and* sensory differences in autism. The “big three” theories say nothing about the sensory issues so often associated with autism. It is our belief that good autism theory, or theory synthesis, must be capable of explaining the cognitive *and* sensory differences.

Joint Attention

Joint attention refers to a group of nonverbal behaviors for communicating with another person about something, usually an object. Joint attention includes altering eye gaze between the other person and the object and the use of gestures such as pointing. There is no definitive definition of joint

attention but a number of “competing” attempts to set out what it involves. To further complicate matters, there are two aspects of joint attention to be considered, *initiation* of joint attention and *response* to joint attention. Initiation of joint attention refers to a child’s action in seeking the attention of another person. A child’s response to another person’s seeking to gain their attention – which could be either a shift in the gaze of the other person’s eyes or a pointing action – is known as response to joint attention. Joint attention is important because, in a typically developing child, it is associated with the child’s development of a further concept known as intentional communication which has been referred to as understanding and controlling the transfer of information between individual human beings (or members of other species). A communication must involve a “sender,” a recipient, and signals conveying information between sender and recipient. Woodruff and Premack (1979, p. 334) write that communication is intentional if “the sender (i) appreciates the fact that his behavior transmits information, (ii) recognizes that the recipient also knows that his behaviour is informative, and (iii) is able to choose from a set of alternatives that course of action (or inaction) which will provide (or suppress) a given bit of information.” While in any communication there is a transfer of information between sender and recipient, they stress that in intentional communication, the transfer is purposive because the sender knows that their communication may have a particular effect on the recipient and that effect is sought by the sender.

At some point, the young child begins to understand that another person can help them to achieve some end and that they can communicate their wish to that other person by sending signals. As the child develops, their communication increasingly becomes intentional. It is generally considered that a typically developing child learns between the ages of 6 and 9 months that a particular communicative action or signal has the same effect each time. Whether or not joint attention is a type, or a characteristic, of intentional communication, it is regarded as crucial in developmental terms because without it, functional speech would

not be developed by a child. It is argued that in autism, there is either a delay in the development of the joint attention behaviors associated with intentional communication or such behaviors are not developed at all.

Empathizing/Systemizing Theory

Empathizing involves the identification of another person's emotions and thoughts, the ability to respond to emotions and thoughts appropriately, and the ability to predict a person's behavior. Empathizing has been likened to folk psychology which is the common sense ability of human beings to explain and predict behavior and mental states in other human beings. According to Baron-Cohen (2002, p.248), systemizing "is the drive to analyse the variables in a system, to derive the underlying rules that govern the behavior of a system . . . Systemizing allows you to predict the behavior of a *system*, and to control it." Systemizing skills include mathematical reasoning, mental rotation, mechanical reasoning, and spatial visualization. Systemizing has been compared to folk physics, which is the untrained understanding of basic physical phenomena by human beings. Research into sex differences in cognition has led to hypotheses that women are better than men at empathizing but that men are better than women at systemizing. The realization that persons with autism appear to have difficulty empathizing with other people in some cases, but may also exhibit strong systemizing skills, has caused some scholars to wonder if there is any connection between autism and a person's gender. It is Baron-Cohen's contention that a strong tendency to systemize and weak ability to empathize should be seen in autism. Because he thinks that systemizing is stronger and empathizing weaker in autism than in typically developing men, he has gone on to argue that autism is an example of what he refers to as an "extreme male brain."

Interaction Theory

In a challenge to "theory of mind" through claiming that direct access to other minds is not just possible but is the primary means by which individuals understand what another person is thinking, Shaun Gallagher relies on the

developmental psychological concepts of "primary intersubjectivity" and "secondary intersubjectivity." He writes of primary intersubjectivity that "By the end of the first year of life, infants are capable of a non-mentalistic, *perceptually-based* embodied understanding of the intentions and dispositions of other persons" (Gallagher 2008, p. 166, our italics) and that "secondary intersubjectivity builds on these primary perceptual and interactive capabilities . . . when infants start to recognize *context* as significant" (ibid., p. 166, our italics). He goes on to state that many theorists regard "the capabilities of primary and secondary intersubjectivity to be *precursors* to full-blown ToM" (ibid., p. 166, author's italics) in the sense that fully fledged ToM either builds on or replaces primary and secondary intersubjectivity. However, in his opinion "adult phenomenology attests to the continued role of primary and secondary intersubjectivity in our everyday understanding of and interaction with others" (ibid., p.166), quoting Scheler and Wittgenstein in his defense and writing that "Psychologists provide important empirical evidence that our everyday adult interaction is primarily *perceptual* and *contextual*" (ibid., p. 167, our italics). In other words, he considers that direct access to other minds is achieved by means of perception of facial expressions and body language in the light of the context in which the person does the perceiving. Tony Wootton (2002, p. 92) writes that "the pattern of [reduced] interactional involvement displayed by the autistic child will have as its corollary a radically diminished acquaintance with the practice of taking other people's views into account."

Narrative Practice Hypothesis

As previously stated, folk psychology, or commonsense psychology, is the natural capacity to explain and predict the behavior and mental states of other people. Daniel Hutto is sure that there is no need for an innate, hardwired theory of mind ability in human beings because typically developing individuals develop an understanding of folk psychology through continuous exposure during the formative years to stories that teach them about folk psychological practice. He puts it this way: "Encounters with narratives about

those who act for reasons best explain the origins of folk psychological (FP) abilities, both phylogenetically and ontogenetically. Such stories familiarize us with the forms and norms of folk psychology. This is the core claim of the Narrative Practice Hypothesis” (Hutto 2007, pp. 47–48).

Sensorially Disturbed Interaction Hypothesis

Victoria McGeer (2001, p. 129) proposes that sensory disturbances may lie at the heart of autism (as well as deafness and blindness) in that “Being excluded from the regulative influences of other people, autistics will not develop habits of agency that conform to shared norms of what it is to experience, think and act in recognizably normal ways” – which, in autism, could account for a failure to develop non-autistic social understanding [McGeer uses the term psycho-practical expertise] – as well-being “cast back on their own resources for managing their sensory experiences perhaps by reducing, repeating or drowning out incoming sensory stimuli in ways they can control” (ibid., p. 129). This could account for a range of typically autistic symptoms such as repetitive and self-stimulatory behaviors. McGeer writes that her speculations suggest that “becoming minded as others are minded, and sharing thereby in the advantages of normal psychological knowing, may finally depend on something as basic as having sensory access to others in a way that makes possible their regulative influence on us as developing children” (ibid., p. 129) which, if correct, would reconcile the focus of autistic autobiographical accounts of sensory sensitivities with the focus of non-autistic clinicians and researchers of autism on the social difficulties seen in autism.

Time-Parsing Deficit Hypothesis

Jill Boucher (2003, p. 250) refers to the fact that “An earlier hypothesis concerning the psychological cause(s) of language impairment in autism suggested that there is a fundamental deficit in the ability to process transient, sequential stimuli (i.e. stimuli with a temporal dimension) such as speech or manual signing,” which she attempted to revive in the slightly different form of a “time-parsing deficit.” With this theory of autistic language impairment, Boucher claimed that autism

involves varying levels of difficulty in the understanding of conversation exchanges (or signing) in real time “which contributes to the linguistic aspects of their pragmatic impairment” (ibid., p. 250). She considers that the extent of the difficulty in parsing conversation is dependent on where a person lies on the autism spectrum.

Enactive Mind Hypothesis

The enactive mind hypothesis is more of a theoretical framework for understanding autism than an actual theory of autism with Klin et al. writing of their hypothesis as “a framework different from the prevailing computational models of social cognitive development” (Klin et al. 2003, p. 357) involving “disembodied cognition” where cognition and action are separate. The key aspect of the enactive mind hypothesis is that, instead of a child’s mind consisting of certain innate capabilities which are gradually given rein, the mind is an “*active mind* that sets out to make sense of the social environment and that changes itself as a result of this interaction” (ibid., p. 348, our italics). Unlike the disembodied cognition associated with computational models, with an active mind, cognition and action are inextricably linked in the typically developing child *but apparently not in the autistic child*. The alternative framework is centered around:

a different set of social cognitive phenomena, for example people’s predispositions to orient to salient social stimuli, to naturally seek to impose social meaning on what they see and hear, to differentiate what is relevant from what is not, and to be intrinsically motivated to solve a social problem once such a problem is identified. [Their framework] is called EM in order to highlight the *central role of motivational predispositions to respond to social stimuli* and a developmental process in which social cognition results from social action. (ibid., pp. 347/348, our italics)

Enhanced Perceptual Functioning Model

Laurent Mottron and his team have proposed a perception-based model of autism described as the “enhanced perceptual functioning model” along with a set of eight principles of autistic perception (Mottron and Burack 2001; Mottron et al. 2006). The latest version of the enhanced

perceptual functioning model takes account of the researchers' realization "that a *primary* superiority in perceptual analysis could possibly underlie both local biases in hierarchical perception and construction, and exceptionally accurate reproduction of surface properties of the world, like 3-D perspective or absolute pitch values in savants" (Mottron et al. 2006, p. 28, authors' italics). In developing their model, Mottron et al. retained the concept of local bias from the weak central coherence theory (attributing it to superior perceptual functioning in autism) but regarding it as "mandatory" in autism in opposition to Frith and Happé's view of local bias as a cognitive preference.

Future Directions

After many decades of research to explain cognition in autism, there is still no definitive explanation or theory. But some theory does explain aspects of autism and has led to an improved understanding of what it is to be autistic and what needs to be done to support autistic individuals in the community. The vast majority of research into autism continues to be focused on what increasingly appears to be a fruitless search for what causes it. Such research is also potentially dangerous given the risks of it leading to susceptibility testing, genetic engineering, or even full-blown eugenics. Other researchers consider that the primary focus of autism research should be on identifying interventions, and other means of support, to improve the well-being of autistic individuals and their lived experience in a non-autistic world.

There is a distinct tendency for researchers involved in the development of interventions in autism to base their work solely on empirical evidence rather than on evidence *and* theory (Chown 2015). It would seem entirely appropriate for intervention researchers to take account of theories which provide an understanding of autistic cognition in connection with both the design and implementation of their interventions (as is the case with the highly successful Social

Stories™ intervention) or at least explain why they have not done so. Researchers in the intervention field would do well to consider theory and either explicitly include some form of *theoretical justification analysis* in their reporting alongside empirical findings or explain why their proposals require no theoretical justification.

Anecdotal evidence suggests that research into autism causation does not appear to have been as severely affected by austerity measures as has other autism research and diagnosis and support for autistic people. (A London Borough recently attempted to place a limit on the number of persons permitted to receive a diagnosis of autism in their part of the capital in order to meet budget cuts.) Given the commercial and other considerations that drive much autism research, one can only hope that governments will take increasing heed of the full range of views on this subject, and a leading role, so that research priorities cease to be so biased.

See Also

- ▶ [Executive Functioning and Martial Arts Training in Children with Autism Spectrum Disorder](#)
- ▶ [Theory of Mind](#)
- ▶ [Weak Central Coherence](#)

References and Reading

- Attwood, T. (1998). *Asperger's syndrome: A guide for parents and professionals*. London: Jessica Kingsley Publishers.
- Baron-Cohen, S. (1995). *Mind blindness: An essay on autism and theory of mind*. Cambridge, MA: The MIT Press.
- Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences*, 6(6), 248–254.
- Boucher, J. (2003). Language development in autism. *International Journal of Pediatric Otorhinolaryngology*, 67S1, S159–S163.
- Chown, N. (2015). Do researchers evaluate psychosocial interventions for autism from the perspective of the three dominant cognitive autism theories? *Review Journal of Autism and Developmental Disorders*, 2(3), 243–261.
- Corbett, B. A., Constantine, L. J., Hendren, R., Rocke, D., & Ozonoff, S. (2009). Examining executive

- functioning in children with autism spectrum disorder, attention deficit hyperactivity disorder and typical development. *Psychiatry Research*, 166(2), 210–222.
- Delis, D. C. (2012). Introduction: A history of executive functioning as a theoretical and clinical construct. In S. Goldstein, J. A. Naglieri, D. Princiotta, & T. M. Otero (Eds.), *Handbook of executive functioning* (pp. 3–12). New York: Springer. (2014).
- Frith, U., & Happé, F. (1994). Autism: Beyond “theory of mind”. *Cognition*, 50(1), 115–132.
- Frith, U., & Happé, F. (1999). Theory of mind and self-consciousness: What is it like to be autistic? *Mind & Language*, 14, 1–22.
- Gallagher, S. (2008). Inference or interaction: Social cognition without precursors. *Philosophical Explorations*, 11(3), 163–174.
- Happé, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36(1), 5–25.
- Hutto, D. D. (2007). The narrative practice hypothesis: Origins and applications of folk psychology. In D. D. Hutto (Ed.), *Narrative and understanding persons*. Royal Institute of Philosophy Supplement, Cambridge, Cambridge University Press.
- Klin, A., Jones, W., Schultz, R., & Volkmar, F. (2003). The enactive mind, or from actions to cognition: Lessons from autism. *Philos Trans R Soc Lond B: Biol Sci*, 358(1430), 345–360.
- Macintosh, K. E., & Dissanayake, C. (2004). The similarities and differences between autistic disorder and Asperger’s disorder: A review of the empirical evidence. *Journal of Child Psychology and Psychiatry*, 45(3), 421–434.
- McGeer, V. (2001). Psycho-practice, psycho-theory and the contrastive case of autism: How practices of mind become second-nature. *Journal of Consciousness Studies*, 8, 109–132.
- Mottron, L., & Burack, J. A. (2001). Enhanced perceptual functioning in the development of autism. In J. A. Burack, T. Charman, N. Yirmiya, & P. R. Zelazo (Eds.), *The development of autism: Perspectives from theory and research*. Mahwah: Lawrence Erlbaum Associates Publishers.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36(1), 27–43.
- Murray, D., Lesser, M., & Lawson, W. (2005). Attention, monotropism and the diagnostic criteria for autism. *Autism*, 9(2), 139–156.
- Woodruff, G., & Premack, D. (1979). Intentional communication in the chimpanzee: The development of deception. *Cognition*, 7(4), 333–362.
- Wootton, A. J. (2002). Interactional contrasts between typically developing children and those with autism, Asperger’s syndrome, and pragmatic impairment. *Issues in Applied Linguistics*, 13(2), 133–159.

Autism Traits and Parenting

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Definition

Raising children and providing them with protection and care in order to ensure their healthy social, emotional, cognitive, and physical development into adulthood are a critical task of parents. As parenting is known to impact children’s developmental outcomes, examining parenting in the context of autism is an important focus of inquiry.

Historical Background

Little remains known about the lives of adults with autism (Howlin and Magiati 2017), including parenting children. As parenting practices and parenting style are related to the developmental and psychological outcomes of children (Belsky and de Haan 2011), being autistic or having autism traits has the potential to influence parenting.

Autism is associated with difficulties in social communication and social reciprocity, including social-emotional difficulties (American Psychiatric Association (APA) 2013) which can impact the quality of social interactions. Social-cognitive difficulties are also common, including difficulties in taking another’s mental perspective, important in sensitive and responsive parenting (e.g., Laranjo et al. 2008). Furthermore, mental health, specifically depression and anxiety, can influence parenting practices (Berg-Nielsen et al. 2002), with individuals with autism and higher autism traits commonly reporting poorer mental health (Ingersoll and Hambrick 2011; Sucksmith et al. 2011). Good parenting also involves consistency and routine, with

predictability being important in giving children a sense of control, a feature also preferred by those with autism. Thus, much of what is known about both autism and parenting suggests this relationship is an important one to examine, particularly with regard to identifying parenting difficulties and needs that may require support. This understanding can lead to the development of methods to enhance and support parenting outcomes.

Current Knowledge

Only three studies to date have examined parenting with autism. Studying parents with subthreshold traits of autism (as measured by the Autism Spectrum Quotient, AQ; Baron-Cohen et al. 2001), van Steijn et al. (2013) found that mothers with high autism traits use a more permissive parenting style (which is high in warmth but low in control, unlike the optimal authoritative parenting style which is high in both) with their typically developing (TD) child compared to their child with a diagnosis of autism spectrum disorder (ASD). Interestingly, in fathers, it was their ADHD characteristics, not autism traits, that were related to difficulties in parenting of both their affected and unaffected children.

Lau et al. (2016) examined parenting efficacy (rather than parenting style) in mothers and fathers with and without an ASD diagnosis who were raising a child with ASD. They found that while fathers with a diagnosis of ASD reported being less efficacious in their parenting (as measured by the Parenting Sense of Competence scale, PSOC; Johnston and Mash 1989), mothers who had a diagnosis did not differ in their parenting efficacy from mothers without a diagnosis. However, children with a diagnosis of ASD may be difficult to parent because of their characteristics, and, as such, it is impossible to determine (in a cross-sectional study) whether any difficulties in parenting competence observed are due to the child's or the parent's autism traits.

Dissanayake et al. (2019) examined whether autism traits (as measured by the AQ) are uniquely related to parenting a TD child, over

and above other demographic and psychological factors, including the parent's own parenting history. While all parents had a child with ASD, their parenting was examined in relation to the TD child they were raising. None of the demographic variables, nor parenting history, played a role in the relationship between autism traits and the parenting variables studied. However, parental well-being, as measured by the Depression Anxiety and Stress Scale (DASS-21; Lovibond and Lovibond 1995), did contribute to parenting outcomes; thus although a relationship was found between autism traits and satisfaction in the parenting role, with higher traits associated with lower parenting satisfaction, when psychological well-being was accounted for, autism traits did not contribute uniquely to the model. Moreover, autism traits were not related to parenting efficacy. Given that the majority of participants in this study were mothers, this latter finding is consistent with Lau et al.'s (2016) finding that mothers with an ASD diagnosis did not differ in their parenting efficacy from mothers without ASD, although it should be noted that unlike in Dissanayake et al., their findings relate to parenting a child with ASD.

Dissanayake et al. (2019) found that autism traits were associated with parenting difficulties and some aspects of the parent-child relationship (as measured using the Parent-Child Relationship Inventory, PCRI; Gerard 1994) when raising a TD child. Autism traits were negatively related to three of the seven factors examined, with higher traits associated with less perceived enjoyment and fulfilment in the parent-child relationship (*Satisfaction*) and the level of interaction with the child (*Involvement*). As social interaction difficulties (APA 2013) are a core feature of ASD, it is not unexpected that individuals with high autism traits will experience more difficulty interacting with others, including members of their own family. Interestingly, autism traits were not related to other aspects of the parent-child relationship (including *Communication*, *Limit-setting*, *Autonomy*, and *Role Orientation*).

Autism traits were found to be associated with an increase in overall parenting difficulties, as measured by a Parenting Needs Questionnaire

(PNQ) developed by the study authors (Dissanayake et al. 2019). Psychological well-being was also related to parenting difficulties. However, autism traits were also uniquely related to parenting difficulties in all but two parenting domains. Parents with high autism traits reported more difficulties in the subscales of *Modeling/Teaching Behaviors*, *Understanding Needs*, *Emotion Control*, *Attention/Connection*, *Spontaneity*, and *Sensory Issues* compared to parents with low autism traits. Parent with high traits reported moderate difficulties regulating their emotion during parenting situations, and connecting with, or maintaining attention on their child during interactions. They reported more difficulties in modeling and teaching their child behaviors, understanding the needs of their child, being spontaneous in parenting situations, and coping with sensory stimuli around their child. As the development of the subscales of the PNQ was informed by parents who had ASD, it is perhaps unsurprising that those with high autism traits found difficulties in each of these areas.

There were, however, no differences observed in the *Affection Subscale of the PNQ*, indicating that parents with high autism traits do not find it more difficult giving and receiving affection from their child. This finding accords with research findings that both younger and older children with ASD are attached to their parents in the same way as are TD children (Chandler and Dissanayake 2014; Dissanayake and Crossley 1996; Dissanayake and Sigman 2000). The *Danger Awareness Subscale* also failed to differentiate parents with high and low traits, suggesting that these parents do not have difficulty anticipating dangers on behalf of their TD child. Showing and receiving affection and keeping children out of harm's way are critical aspects of parenting which appear to not be related to the presence of high autism traits.

Future Directions

The studies reviewed above on parenting children with and without autism when parents have either a diagnosis of ASD or high autism traits

themselves have identified specific aspects of parenting that may benefit from targeted support which can assist these parents prosper in their parenting role. It is likely that the wealth of available parenting resources will not address the specific needs of parents with high autism traits as the majority of these resources are developed for parents without additional needs, although there are available resources for parents raising children with additional needs. Thus, just as there is a paucity of research on the parents with ASD/high autism traits, there is a lack of parenting resources for them. However, a first step to providing information and resources needed for these parents from which they may benefit is available here: <https://www.amaze.org.au/2017/05/ceo-proud-of-our-new-parenting-skills-guide/>

The few studies to date on parenting with ASD/autism traits have been informed by the parenting literature, where research has been undertaken within the general population. Hence, the specific strengths that autistic parents may bring into their parenting role have not been a focus of study and should be examined in future research. It is also important to focus on the outcomes of the children who are the target of parenting, be they autistic or otherwise. Given the findings presented here that autism traits are related to both parenting abilities and difficulties, it is necessary to examine the positive and/or negative impacts, if any, on children's developmental outcomes.

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from asperger syndrome/high-functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders*, 31(1), 5–17. <https://doi.org/10.1023/A:1005653411471>.
- Belsky, J., & de Haan, M. (2011). Annual research review: Parenting and children's brain development: The end of the beginning. *Journal of Child Psychology and Psychiatry*, 52, 409–428. <https://doi.org/10.1111/j.1469-7610.2010.02281.>

- Berg-Nielsen, T. S., Vikan, A., & Dahl, A. A. (2002). Parenting related to child and parental psychopathology: A descriptive review of the literature. *Clinical Child Psychology and Psychiatry*, 7(4), 529–552. <https://doi.org/10.1177/1359104502007004006>.
- Chandler, F., & Dissanayake, C. (2014). An investigation of the security of caregiver attachment during middle childhood in children with high-functioning autistic disorder. *Autism*, 18(5), 485–492. <https://doi.org/10.1177/1362361313486205>.
- Dissanayake, C., & Crossley, S. A. (1996). Proximity and sociable behaviours in autism: Evidence for attachment. *Journal of Child Psychology and Psychiatry*, 37, 149–156.
- Dissanayake, C., & Sigman, M. (2000). Attachment and emotional responsiveness in children with autism. *International Review of Research in Mental Retardation*, 23, 239–266. [https://doi.org/10.1016/S0074-7750\(00\)80013-0](https://doi.org/10.1016/S0074-7750(00)80013-0).
- Dissanayake, C., Richdale, A. L., Kolivas, N., & Pamment, L. (2019). An exploratory study of autism traits and parenting. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03984-4>.
- Gerard, A. B. (1994). *Parent-child relationship inventory*. Los Angeles: Western Psychological Services.
- Howlin, P., & Magiati, I. (2017). Autism spectrum disorder: Outcomes in adulthood. *Current Opinion in Psychiatry*, 30(2), 69–76. <https://doi.org/10.1097/YCO.0000000000000308>.
- Ingersoll, B., & Hambrick, D. Z. (2011). The relationship between the broader autism phenotype, child severity, and stress and depression in parents of children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5(1), 337–344. <https://doi.org/10.1016/j.rasd.2010.04.017>.
- Johnston, C., & Mash, E. J. (1989). A measure of parenting satisfaction and efficacy. *Journal of Clinical Child Psychology*, 18(2), 167–175. https://doi.org/10.1207/s15374424jccp1802_8.
- Laranjo, J., Bernier, A., & Meins, E. (2008). Associations between maternal mind-mindedness and infant attachment security: Investigating the mediating role of maternal sensitivity. *Infant Behavior and Development*, 31(4), 688–695.
- Lau, W. Y. P., Peterson, C. C., Attwood, T., Garnett, M. S., & Kelly, A. B. (2016). Parents on the autism continuum: Links with parenting efficacy. *Research in Autism Spectrum Disorders*, 26, 57–64. <https://doi.org/10.1016/j.rasd.2016.02.007>.
- Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the depression anxiety stress scales (DASS) with the beck depression and anxiety inventories. *Behaviour Research and Therapy*, 33(3), 335–343. [https://doi.org/10.1016/0005-7967\(94\)00075-U](https://doi.org/10.1016/0005-7967(94)00075-U).
- Sucksmith, E., Roth, I., & Hoekstra, R. (2011). Autistic traits below the clinical threshold: Re-examining the broader autism phenotype in the 21st century. *Neuropsychology Review*, 21(4), 360–389. <https://doi.org/10.1007/s11065-011-9183-9>.
- van Steijn, D. J., Oerlemans, A. M., de Ruiter, S. W., van Aken, M. A., Buitelaar, J. K., & Rommelse, N. N. (2013). Are parental autism spectrum disorder and/or attention-deficit/Hyperactivity disorder symptoms related to parenting styles in families with ASD (+ ADHD) affected children? *European Child & Adolescent Psychiatry*, 22(11), 671–681. <https://doi.org/10.1007/s00787-013-0408-8>.

Autism: Social Communication Disorder

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Synonyms

Pragmatic communication disorder; Pragmatic language disorder; Pragmatic language impairment; Semantic pragmatic deficit disorder; Semantic pragmatic deficit syndrome; Semantic pragmatic language disorder; Social pragmatic communication disorder

Short Description or Definition

In more than 70 years since its first clinical descriptions, conceptualizing autism as a diagnostic category has been an ever-evolving and challenging process due to the broad heterogeneity that exists within this disorder. With the more narrow and rigid diagnostic criteria for autism spectrum disorder (ASD), as described in the DSM-5 (APA 2013), a new diagnostic classification was introduced in an attempt to account for individuals with marked impairments in social and communicative abilities but who fail to meet DSM-5 criteria for ASD: social communication disorder (SCD).

SCD, sometimes referred to as social pragmatic communication disorder, is defined by primary impairments in the pragmatic aspects of

verbal and nonverbal social communication. Specifically, those with SCD will present with significant difficulties interpreting meaning from social cues and adapting their communicative efforts to match social contexts (e.g. following conversation rules, modifying language for different situations or audiences, inferring meaning based on context).

Categorization

Clinical reports on disorders of pragmatic communication were first published in the 1980s (Bishop and Rosenbloom 1987; Rapin and Allen 1983) and described children who possessed relatively unimpaired vocabulary, grammar, and word structure but presented with significant difficulties utilizing and comprehending changes in language based on social contexts (see Semantic Pragmatic Disorder and Pragmatic Language Impairment). Symptomology overlap between these disorders and the social communicative characteristics of ASD were recognized from an early stage. As such, it was common for individuals with these disorders to receive diagnostic labels of Asperger's syndrome or PDD-NOS/unspecified (Bishop 1989).

In the DSM-5, SCD is categorized as a communication disorder along with childhood-onset fluency disorder, language disorder, and speech sound disorder, in the broader domain of neurodevelopmental disorders (the same domain as ASD). Outside the USA (particularly in the UK), the impairments which characterize SCD are approximately equivalent to a condition known as pragmatic language impairment (PLI). Relatedly, though PLI is not included as a diagnostic category in the DSM-5, it is reportedly going to be included in the upcoming ICD-11 as a language disorder (Baird and Norbury 2016). Despite the differences in labels, it is suggested that these two disorders will approximately encompass the same behavioral phenotype and will provide a diagnosis for individuals with the social communicative impairments of ASD but who lack the restricted interest and repetitive behavior characteristics of that disorder.

Epidemiology

As social communication disorder was only introduced as a diagnostic category with the release of the DSM-5 in 2013, epidemiological studies remain rare and limited in scope. Estimates on the prevalence and incidence of SCD, therefore, are still in preliminary stages. Based on the available literature, depending on which diagnostic manual you use, it is estimated that 8.25–15% (DSM-IV-TR) or 19% (ICD-10R) of individuals who would have previously met diagnostic criteria for a pervasive developmental disorder (PDD) would meet DSM-5 criteria for SCD (Kim et al. 2014; Wilson et al. 2013). These studies further noted that the PDD diagnostic subtypes with the greatest incidence of transferring to a SCD diagnosis based on DSM-5 criteria were Asperger's syndrome (6–15%) and PDD-NOS/unspecified (30–32%).

Only Kim et al. (2014) have produced population and gender prevalence estimates for SCD thus far in the extant literature. The findings of their study suggest that SCD has approximately a 0.5% prevalence rate, with a slightly greater occurrence in males than females (1.3:1 sex ratio). However, additional research on this topic (including cross-cultural examinations) will need to be conducted before the actual prevalence of SCD becomes clear.

Natural History, Prognostic Factors, and Outcomes

It is expected that the onset of symptoms associated with SCD will occur during the developmental stages of early childhood, with notable deficits emerging between age 4 and 5 years. For some, however, impairments may not become apparent until later years when social communicative demands eventually exceed the individuals' abilities. Again, due to the relatively new nature of this diagnostic label, there is limited knowledge available with regard to prognostic factors and outcomes.

According to the DSM-5, risk and prognostic factors associated with SCD include familial

history of other communication disorders, specific learning disorder, or ASD (APA 2013). Based on findings with populations with similar pragmatic language communication deficits (i.e., pragmatic language impairment), it can be inferred that some children with SCD will demonstrate improvements in their pragmatic abilities over time (Swineford et al. 2014). However, it is likely that for most, these difficulties will persist into adulthood, and while individuals with SCD may find success academically and reach levels of independence, these lasting communicative deficits are likely to effect the development of meaningful social and romantic relationships (Whitehouse et al. 2009).

Clinical Expression and Pathophysiology

Clinical expression of SCD involves persistent impairments in social communicative abilities that have been present from early development (though may not be apparent until later in life) and result in significant social, academic, and/or occupational limitations for the individual (APA 2013). Specifically, a diagnosis of SCD requires the presence of difficulties in the following areas of verbal and nonverbal communication:

- Utilizing communication for social reasons (e.g., social greetings, joint attention)
- Altering communication to fit social contexts (e.g., adjusting prosody and/or language depending on the setting)
- Following conversational rules (e.g., turn taking, staying on topic)
- Interpreting ambiguous or nonliteral language (e.g., metaphors, idioms)

The manner in which these impairments will present in an individual will depend on their age and the severity of their social and communicative difficulties. Elleseff (2015) details potential areas of difficulty that may be present in individuals at *initial*, *intermediate*, and *advanced* stages of social communicative development:

Initial: During this early stage, impairments may manifest as limitations in communicative

initiation, attention, and understanding of basic rules or conventions.

Intermediate: For those who progress beyond the above stages of social communicative development, impairments may instead present as difficulties with higher-order skills. Potential areas of deficit include perspective taking, abstract language, humor, and social problem-solving.

Advanced: Finally, for individuals whose deficits are not severe enough to become present at the previous stages, impairments may manifest as deficits in areas like social adaptability, theory of mind, monitoring/regulating the effect of their behavior on others, and knowing when/how to use supportive language with peers.

Evaluation and Differential Diagnosis

Standardized and validated evaluation instruments for the diagnostic assessment of SCD have yet to be introduced, and preliminary investigations suggest that the gold standard ASD diagnostic instruments (ADOS, ADI-R) in their current formats would not be reliable resources for identifying individuals with SCD (Foley-Nicpon et al. 2016). Standardized measures of pragmatic language may be useful tools for pre-evaluation screening or to supplement clinical evaluations and inform assessment decisions. Some measures likely to be utilized for these purposes include the Early Social Communication Scales for young children (8–30 months), the Children’s Communication Checklist-2 for older children and adolescents (4–16 years), and the Analysis of Language Impaired Children’s Conversation that can be used from 4 years old into adulthood (for a full review of available measures, see Adams 2015 and Norbury 2014). Such tools, however, should not be used in isolation to make diagnostic decisions. Instead, best practice recommendations suggest diagnostic assessment should be determined through evaluations by a multidisciplinary team of clinicians and include a thorough developmental history along with assessments of cognition and language to sufficiently assess areas of impairment and rule out alternative diagnoses.

Beyond the exclusionary diagnoses of intellectual disability, global developmental delay, or other mental disorders listed in the DSM-5, the primary disorders to consider for differential diagnosis with SCD are social anxiety disorder and ASD. Social anxiety disorder and SCD can be primarily differentiated based on onset of impairments, with SCD being present since early development and deficits associated with social anxiety disorder developing later in life. Meanwhile, ASD and SCD are differentially diagnosed based on the lifetime presence or absence of Domain B items (restricted interests and repetitive behaviors) from the ASD diagnostic criteria. Specifically, the DSM-5 states that a SCD diagnosis should only be considered if “the developmental history fails to reveal any evidence of restricted/repetitive patterns of behaviors, interests, or activities” (APA 2013, p. 49). This stipulation results in a diagnostic gap for individuals who have restricted interest and repetitive behaviors that are too limited to meet the Domain B criteria to obtain an ASD diagnosis but are exclusionary criteria from receiving a SCD diagnosis; and it has yet to be seen how this diagnostic gap will be addressed in practice.

Treatment

Some of the greatest implications of the diagnostic separation of SCD from ASD are with regard to therapeutic interventions. Specifically, over the past few decades the field of ASD has developed a well-established network of foundations and organizations engaged in outreach, education, research, benefits, support services, and public health. Meanwhile, a major area of concern is if the independence of these diagnoses will result in individuals with SCD being excluded from the valuable benefits of these networks (Brukner-Wertman et al. 2016). Further, since DSM-5 diagnostic criteria for SCD exclude the presence of restricted interests and repetitive behaviors, interventions made available to individuals with this diagnostic label may overlook subtle in these areas that can still exist impairments (e.g., difficulties with flexible thinking or change) and are potentially

additional sources of distress within this population (Christ et al. 2010).

Standard practices for treatment of SCD has yet to be determined; however, as communication difficulties are fundamental to the impairments of SCD, recommended intervention strategies will likely revolve around the incorporation of speech and language pathologists. To date, the only published randomized controlled trial of an intervention for SCD was conducted by researchers in the UK (Adams et al. 2012). Comparing the outcomes of children with pragmatic and social communication impairments (PLI) who received 16–20 sessions of direct, manualized intervention from a specialist speech and language pathologist to those who received treatment as usual, Adams and colleagues produced mixed findings. While blind and parent/teacher reports suggested significant improvements in some communication abilities, no improvements in structural language skills were noted based on standardized language assessments (Adams et al. 2012).

With the inclusion of SCD in the DSM-5 (and the anticipated inclusion of PLI in the ICD-11), the increased research and clinical focus on this set of social communicative impairments should result in increased understanding of areas of communicative strengths and deficits, as well as potential underlying weaknesses (e.g. theory of mind, executive functioning, central coherence) for this disorder, prompting the development of effective, disorder-specific intervention strategies. However, until such strategies are determined, it has been suggested that individuals with SCD should maintain access to the supports afforded to those with ASD, as the significant overlap in social communicative difficulties between these disorders would imply that interventions designed for those with ASD would likely benefit individuals with SCD as well (Brukner-Wertman et al. 2016).

See Also

- ▶ [Australian Scale for Asperger’s Syndrome](#)
- ▶ [PDD-NOS \(Pervasive Developmental Disorder Not Otherwise Specified\)](#)
- ▶ [Pragmatic Language Impairment](#)
- ▶ [Semantic Pragmatic Disorder](#)

References and Reading

- Adams, C. (2015). Assessment and intervention for children with pragmatic language impairment. In D. A. Hwa-Froelich (Ed.), *Social communication development and disorders* (pp. 141–170). New York: Psychology Press.
- Adams, C., Lockton, E., Freed, J., Gaile, J., Earl, G., McBean, K., et al. (2012). The social communication intervention project: A randomized controlled trial of the effectiveness of speech and language therapy for school-age children who have pragmatic and social communication problems with or without autism spectrum disorder. *International Journal of Language & Communication Disorders*, 47(3), 233–244.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association.
- Baird, G., & Norbury, C. F. (2016). Social (pragmatic) communication disorders and autism spectrum disorder. *Archives of Disease in Childhood*, 101(8), 745–751.
- Bishop, D. V. (1989). Autism, Asperger's syndrome and semantic-pragmatic disorder: Where are the boundaries? *British Journal of Disorders of Communication*, 24(2), 107–121.
- Bishop, D. V., & Rosenbloom, L. (1987). Classification of childhood language disorders. In W. Yule & M. Rutter (Eds.), *Language development and disorders. Clinics in developmental medicine (double issue)*. London: Mac Keith Press.
- Brukner-Wertman, Y., Laor, N., & Golan, O. (2016). Social (pragmatic) communication disorder and its relation to the autism spectrum: Dilemmas arising from the DSM-5 classification. *Journal of Autism and Developmental Disorders*, 46(8), 2821–2829.
- Christ, S. E., Kanne, S. M., & Reiersen, A. M. (2010). Executive function in individuals with subthreshold autism traits. *Neuropsychology*, 24(5), 590–598.
- Elleseff, T. (2015). Assessing social communication abilities of school-aged children. *SIG 16 Perspectives on School-Based Issues*, 16(3), 79–86.
- Foley-Nicpon, M., Fosenburg, S. L., Wurster, K. G., & Assouline, S. G. (2016). Identifying high ability children with DSM-5 autism spectrum or social communication disorder: Performance on autism diagnostic instruments. *Journal of Autism and Developmental Disorders*, 47(2), 460–471.
- Kim, Y. S., Fombonne, E., Koh, Y. J., Kim, S. J., Cheon, K. A., & Leventhal, B. L. (2014). A comparison of DSM-IV pervasive developmental disorder and DSM-5 autism spectrum disorder prevalence in an epidemiologic sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(5), 500–508.
- Norbury, C. F. (2014). Practitioner review: Social (pragmatic) communication disorder conceptualization, evidence and clinical implications. *Journal of Child Psychology and Psychiatry*, 55(3), 204–216.
- Rapin, I., & Allen, D. (1983). Developmental language disorders: Nosologic consideration. In U. Kirk (Ed.), *Neuropsychology of language, reading, and spelling* (pp. 155–184). New York: Academic.
- Swineford, L. B., Thurm, A., Baird, G., Wetherby, A. M., & Swedo, S. (2014). Social (pragmatic) communication disorder: A research review of this new DSM-5 diagnostic category. *Journal of Neurodevelopmental Disorders*, 6(1), 41.
- Whitehouse, A. J., Line, E. A., Watt, H. J., & Bishop, D. V. (2009). Qualitative aspects of developmental language impairment relate to language and literacy outcome in adulthood. *International Journal of Language & Communication Disorders*, 44(4), 489–510.
- Wilson, C. E., Gillan, N., Spain, D., Robertson, D., Roberts, G., Murphy, C. M., et al. (2013). Comparison of ICD-10R, DSM-IV-TR and DSM-5 in an adult autism spectrum disorder diagnostic clinic. *Journal of Autism and Developmental Disorders*, 43(11), 2515–2525.

Autism-Europe

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Major Areas or Mission Statement



Autism-Europe (AE) is a European umbrella organization representing persons with autism and their families. AE was founded in 1983 and nowadays ensures liaison among more than 80 member associations of parents of persons with autism in 30 European countries, including 20 Member States of the European Union, governments, and European and international institutions. AE is also consulted by the World Health Organisation on matters relating to autism.

Its aim is to raise awareness across Europe of the fundamental rights and needs of people with ASD by representing them at EU level, and to promote positive actions and policies built on the social model of disability and aimed at the active inclusion of people with ASD, in line with the guiding principles of the UN Convention on the rights of persons with disabilities.

Its overarching statutory mission is to improve the life of all persons with autism by promoting their rights. AE members identified the following strategic objectives as their priorities:

1. Representing persons with ASD before all EU institutions.
2. Promoting the rights and dignity of persons with ASD.
3. Promoting awareness of the appropriate care, education, and well-being of persons with ASD.
4. Liaising with other non governmental organizations sharing similar objectives.
5. Promoting the exchange of accurate and evidence-based information about ASD, good practices and experience.

In order to implement its objectives and maximize its impact on EU policies, Autism-Europe has built strategic alliances with European social partners. AE currently holds the vice-Presidency of the European Disability Forum (EDF). It is also a founding member of the European Coalition for Community Living (ECCL) and the Platform of European Social NGOs.

Landmark Contributions

AE is widely recognized as a credible, representative organization across Europe and among parents, decision makers, social partners, the scientific community, and other stakeholders. This is demonstrated by the frequent ongoing contacts and requests for advice, intervention, partnership and collaboration in European and national projects, initiatives, and events.

In 1996, the *Charter of Rights for persons with Autism* was adopted as a written declaration by the European Parliament following its adoption by the Autism-Europe's Congress in Den Haag in 1992.

In March 2004, the Committee of Ministers of the Council of Europe made public the decision taken by the European Committee of Social Rights of November 4, 2003, whereby France was found to have failed to fulfill its educational obligations to persons with autism under the

European Social Charter. This decision upheld the collective complaint that Autism-Europe had lodged in 2003 against France. Autism-Europe's complaint was the first collective action to defend the rights of people with disabilities in Europe. Its importance in this respect was highlighted by the Council of Europe.

Also as a consequence, the Council of Europe published in 2007 the Resolution ResAP(2007)4 *on the education and social inclusion of children and young people with autism spectrum disorders* drafted with the cooperation of Autism-Europe.

Over the years, persons with ASD have been the target of false beliefs and they and their families have constantly suffered the consequences of unreliable treatments. Autism-Europe has made every effort to disseminate reliable, evidence-based information through collaboration with important professional organizations such as IACAPAP, ESCAP, and INSAR.

Autism-Europe's international congresses, organized every 3 years, provide an interdisciplinary forum to examine state-of-the-art scientific knowledge and current cultural approaches in the field of ASD. Autism-Europe ensures the high scientific quality of its international congresses through the support and participation of internationally renowned experts in the field of ASD.

During the VIII Autism-Europe International congress (Oslo, September 2, 2007), a *Position Paper on Care for Persons with Autistic Spectrum disorders* was presented and officially adopted by Autism-Europe, the International Association of Child and Adolescent Psychiatry and Allied Professions (IACAPAP), and the European Society for Child and Adolescent Psychiatry (ESCAP). It reflects the views of Autism-Europe, IACAPAP, and ESCAP on the approach to Autism Spectrum Disorders.

Autism-Europe also published in 2009 the document *Persons with Autistic Spectrum Disorders: Identification, Understanding, Intervention*, drafted by a team of European experts – Catherine Barthélémy, Joaquín Fuentes, Patricia Howlin, and Rutger van der Gaag. The document, which was drafted on a pro-bono basis by these experts, enables a better understanding of ASD and the needs of those affected by this condition. This

document is addressed not only to parents but also for all professionals who are involved in interventions for persons with ASD, and for European and national authorities responsible for the care of individuals with disabilities.

Major Activities

Representing Persons with Autism and Defending Their Interests at the European Level

Autism-Europe's engagement in defending the rights of persons with ASD, by means of legal instruments, such as the collective complaint against France lodged before the Council of Europe's Committee on Social Rights has been widely recognized by the European Institutions.

Autism-Europe is considered as one of the key EU networks in the field of disability and as such is regularly consulted in the process of policy-making to raise the concerns of persons with ASD and also benefit from the support of the European Commission to promote measures against discrimination.

Disseminating Evidence-Based Information About Autism as well as Promoting the Exchange of Knowledge and Best Practices on the Appropriate Care, Education, and Well-Being of Persons with ASD

Disseminating accurate and evidence-based information about autism is key in order to enhance understanding of autism within society and prevent abuse. The recognition of the specific needs of persons with Autistic Spectrum Disorders is essential to foster their inclusion in the community and improve their quality of life.

In order to promote self-advocacy, Autism-Europe has published a number of information documents and toolkits. Many documents of Autism-Europe are translated into easy-to-read format.

Autism-Europe's publications – which are available on its website – include:

- Information documents about Autistic Spectrum Disorders drafted in cooperation with experts.

- Position papers and reports addressed to European decision makers and public authorities.
- Toolkits for self-advocates, taking into account the latest legislative developments at EU level.
- Newsletters about the latest EU developments in the field of disability.
- LINK magazine to share information about important developments at EU and national levels.

Autism-Europe is also involved in a number of European projects – notably in the field of research, life-long learning and deinstitutionalization – in order to share its expertise and disseminate the results across Europe.

Every 3 years, Autism-Europe organizes an International Congress which aims at bringing together self-advocates, families, and professionals in order to share knowledge about state-of-the-art scientific findings in research and intervention.

The IX International Congress took place in Catania in October 2010, all the videos of the session are available on the Congress website and on Youtube. The congress was attended by over 1,200 delegates. Many of the most prominent researchers in the field of autism were present as speakers. AE has built a relationship of trust with both the scientific community and the professionals working in the field of ASD, which allows a fruitful cooperation in order to enhance the rights-based approach to care and intervention.

Promoting General Awareness of Autism

Every year, Autism-Europe also holds the European Days of Autism in October to share information at European level and raise awareness about ASD across Europe.

A wide range of activities are also organized by Autism-Europe and its members to mark the World Autism Awareness Day adopted by the United Nations.

Liaising with Other Non Governmental Organizations Sharing Similar Objectives

Finally, Autism-Europe cooperates closely with other European and international NGOs. It currently holds the vice-Presidency of the European

Disability Forum (EDF). AE strives for the recognition of the complex needs of persons with autism, and other kinds of disabilities requiring a high level of support. It is also a founding member of the World Autism Organization, the European Coalition for Community Living (ECCL), and the Platform of European Social NGOs.

References and Reading

All the publications of Autism-Europe are available on its website. <http://www.autismeurope.org/>

Autistic

► Autism

Autistic Disorder

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Synonyms

Childhood autism; Infantile autism; Kanner's autism

Short Description or Definition

As defined in DSM-IV Autistic disorder was the prototypical autism spectrum/pervasive developmental disorder. The condition, first described by Leo Kanner in 1943, is marked by severe and sustained problems in social development (autism) along with unusual communication and a range of problems typically subsumed under the term “resistance to change” – the last take the form of restricted or stereotyped patterns of behavior and interest as well as literal difficulties tolerating change. The

onset of the condition is in the first years of life. While many individuals with the condition eventually exhibit intellectual disabilities, these rates have decreased with earlier detection and intervention. The revision of the concept in DSM-5 (autism spectrum disorder) reflected a growing body of work suggesting that autism is indeed part of a broader range of conditions characterized by problems of various sorts including in social interaction; unfortunately this DSM-5 definition also excluded many individuals who previously had a diagnosis of autism or a related conditions (Smith et al. 2015).

In his first description of 11 cases, Kanner emphasized two essential features: (1) an inborn disturbance of affective contact, that is, with an apparently congenital “inability to relate” to people in usual ways, and (2) difficulties with change/insistence on sameness, including motor stereotypies, which Kanner viewed as an attempt by the child to maintain sameness. Although he did not emphasize communication problems as central to the definition of the condition, he did note a variety of unusual communication features including mutism (in many cases) and, for those with speech, pronoun reversal and echolalia. Although remaining profoundly influential, his original description also was misleading in some respects, for example, he did not appreciate the extent of cognitive (although often highly scattered) delays and his mention of high SES levels in parents suggested that the disorder was somehow more frequent in highly educated families. The latter contributed to an early mistaken impression that care-taking contributed to pathogenesis. His use of the term “autism” was based on Bleuler’s early use of the word to describe self-centered thinking in schizophrenia – this suggested a connection to childhood schizophrenia/psychosis that proved unwarranted. On the other hand, Kanner’s emphasis on developmental aspects of early social skills and his rich description were a landmark in the field.

Early research on the condition was complicated by confusion of the condition with childhood psychosis/schizophrenia and the emphasis on possible environmental/experiential factors in causation. Over time, the work of Kanner (1971) and Rutter (1972) helped clarify the lack of association with schizophrenia, and follow-up studies noted association with factors strongly suggestive of a familial, brain-based disorder (Folstein and

Rutter 1977; Volkmar and Nelson 1990). Rutter (1978) proposed a highly influential definition of autism based on the presence of social delay and deviance, communication problems, and unusual behaviors. His proposal had a major influence on the criteria for infantile autism when the condition was first recognized officially in DSM-III (APA 1980).

Categorization

Within DSM-IV autism was recognized as one example of the pervasive developmental disorders. The latter term was coined in 1980 for the overarching category of which autism was the prototype in DSM-III (APA 1980) and was synonymous with the more frequently used term “autism spectrum disorder.” Over time, the categorical definition of autism has evolved in some ways since Kanner’s first description. The D-10/DSM-IV definitions of childhood autism were essentially the same (ICD-10, which has both a clinical and research version, provides more potential for differentiation of atypical presentations of autism; see DSM-IV entry). In the current approach, associated medical conditions (if any) and other developmental and psychiatric problems (e.g., intellectual disability) are also coded in the multiaxial approach adopted by DSM-IV (Rutter et al. 1969). Although rates of association of autism with other medical conditions have been much debated, the strongest associations are with a limited number of genetic conditions such as Fragile X syndrome and tuberous sclerosis (Rutter et al. 1994). As noted above the DSM-5 definition is more stringent.

Epidemiology

Many epidemiological studies have been conducted with the DSM-IV/ICD-10 definitions. The median rate of autistic disorder (if strictly defined) is somewhere between 1 in 800 to 1,000 individuals (Hill et al. 2014). Although there is a widespread impression of increased rates of autism changes in diagnostic criteria, increased public awareness, better

diagnosis among more cognitively able individuals, and other factors likely account for much of this impression. Smaller and more thorough studies also report higher rates.

There is a noteworthy male predominance in autism (usually between three and five times as many cases in boys than in girls), but among lower IQ individuals the difference becomes less pronounced. Conversely among the most cognitively able persons, the difference is even more striking. Cultural and ethnic issues have been relatively uncommonly studied. Clearly, the early impression of a high-SES class predominance was unfounded (likely reflecting selective bias in initial referrals) (Grinker 2007). Within the United States, there is more concern about underdiagnosis in individuals coming from lower SES/poverty (Mandell et al. 2007). Cultural issues may impact treatment with considerable variations in entitlements and practice from country to country.

Natural History, Prognostic Factors, and Outcomes

The long-term outcome for children with autism appears to be improving (see Howlin et al. 2015). This does not appear to simply be a result of increased diagnosis among more able individuals. Rather earlier detection and intervention appear to have an important positive benefit for most children (National Research 2001). Over time, the number of individuals with autism who are capable of adult self-sufficiency and independence as adults has increased substantially. That being said, even with provision of good programs, not every child makes substantial improvement. Various issues, including factors apart from the child, can impact outcome, for example, in some countries, available services are limited, and even in more developed countries, factors like poverty may delay or impede case detection and service provision.

Diagnostic issues are most complex in young children (those under 3), although the increased body of work on infants and infant siblings of children with autism has contributed to greater awareness of the diagnostic challenges and need

for more robust methods of early detection (Chawarska et al. 2008). There is reasonable agreement that after age 3 years the diagnosis becomes relatively stable (prior to that time some, but not all diagnostic features, may be apparent). Often social-communication problems are more dramatic in younger children, but the required difficulties with change/stereotyped mannerisms may be the last to develop.

By school age, children with autism often develop more social interest but also may have more behavioral difficulties (Loveland and Tunali-Kotoski 2005). The latter can include agitation, motor mannerisms, and self-injurious behavior. As first noted by Kanner in adolescence, some children make gains while others lose ground (Kanner 1971; Shea and Mesibov 2005). More and more adults are able to be self-sufficient with many now attending college and post-secondary school programs (Volkmar and Wiesner 2009). Positive prognostic factors include higher levels of language and cognitive ability around age 5 years (prognosis can be difficult, however, and presumably depends on a range of factors) (Coplan 2000).

Clinical Expression and Pathophysiology

Marked changes over the course of development are common. A unifying theme, however, across development is the persistence of social difficulties. Although early speculation centered on the possible relevance of experience to pathogenesis, many different lines of research have strongly implicated neurobiological factors. This work includes the observation of markedly increased rates of epilepsy as well as various persistent neurological signs and symptoms (Minshew et al. 2005; Volkmar and Nelson 1990). Over the last decade, new approaches to neuromaging have also illustrated areas of possible difference associated with autism relative to factors such as perception of biological motion or relevance of faces (Schultz et al. 2000; Pinkham et al. 2008). A growing body of research has focused on social information processing in the brain – areas of interest include structures such as the amygdala (e.g., in social perception and social thinking),

frontal lobe regions and other areas involved in social information processing, and the fusiform face area.

Postmortem studies have revealed some abnormalities in specific brain regions as well as changes in overall architecture of the fine structure of the brain (Casanova 2007). Animal model work was originally limited to lesion studies (Bachevalier and Loveland 2006) but now includes genetic studies (e.g., based on knock out gene models) (Gupta and State 2007). The strong role of genetic factors has been suggested by various studies of siblings who are at substantially increased risk for autism. It appears that multiple genes are involved (O’Roak and State 2008). Several approaches have been used to identify potential contributing genetic mechanisms (Abrahams and Geschwind 2008).

Although the lay press has devoted considerable attention to the role of environmental factors (including immunizations) in autism, substantive data are lacking (Offit 2008; Wing and Potter, 2008).

Evaluation and Differential Diagnosis

Autism presents several major challenges for evaluation and diagnosis. Challenges include marked variability in skills, involvement of a range of service providers, and the potential for major change with intervention. Autism should be differentiated from other related disorders as well as other developmental disorders (e.g., of language or intellectual development) and from unusual profiles of development associates with sensory difficulties (e.g., deafness) or with severe neglect. Differences between autism and related disorders as defined in DSM-IV and ICD-10 relate both to historical information and current clinical presentation (key features are summarized in Table 1).

Assessment is complicated by the often strikingly varied cognitive profiles. In autism, nonverbal skills are typically more preserved than verbal ones. In language disorders, social interest and motivation remain even in the face of sometimes severe communication problems. In mental retardation without autism, social skills are usually not dramatically different from other cognitive abilities and may be

Autistic Disorder, Table 1 Differential diagnostic features of autism and nonautistic pervasive developmental disorders

Feature	Autistic disorder	Asperger's disorder	Rett's disorder	Childhood disintegrative disorder	Pervasive developmental disorder NOS
Age at recognition (months)	0–36	Usually >36	5–30	>24	Variable
Sex ratio	M > F	M > F	F (?M)	M > F	M > F
Loss of skills	Variable	Usually not	Marked	Marked	Usually not
Social skills	Very poor	Poor	Varies with age	Very poor	Variable
Communication skills	Usually poor	Fair	Very poor	Very poor	Fair to good
Circumscribed interests	Variable (mechanical)	Marked (facts)	NA	NA	Variable
Family history – similar problems	Sometimes	Frequent	Not usually	No	Sometimes
Seizure disorder	Common	Uncommon	Frequent	Common	Uncommon
Head growth decelerates	No	No	Yes	No	No
IQ range	Severe MR to normal	Mild MR to normal	Severe MR	Severe MR	Severe MR to normal
Outcome	Poor to good	Fair to good	Very poor	Very poor	Fair to good

M male, *F* female, *MR* mental retardation, *NA* not applicable

Adapted, with permission, from Volkmar, F. R., & Cohen, D. (1985). Nonautistic pervasive developmental disorders. In R. Michaels et al. (Eds.), *Psychiatry* (Chap. 27.2, p. 4). Philadelphia: Lippincott-Raven

an area of strength. A common source of confusion is the frequent presence of stereotyped mannerisms in association with severe intellectual impairment. As a practical matter, stereotyped mannerisms are not particularly diagnostic of themselves and have significance for autism only when associated with social-communication deficits.

Children, adolescents, and adults with autism typically have problems in various areas (cognition, adaptive functioning, communication, social skills, and behavioral difficulties). Many different tests have been developed for purposes of screening (Coonrod and Stone 2005) and diagnosis (Lord and Corsello 2005). Children with autism present many challenges for assessment, and considerable skills may be needed (Volkmar and Wiesner 2009). Provision of a comprehensive, integrated view of the individual (attending to both strengths and weaknesses) with provision of an intervention program should be the goal of assessment (Volkmar and Wiesner 2009).

Treatment

A substantial body of work supports the use of behavioral and educational interventions in autism (Volkmar and Wiesner 2009). Increasingly much of this work is strongly evidence based (Reichow and Wolery 2009). The most effective programs rely on behavior modification and special education with a goal of minimizing negative effects of autism on learning and maximizing more normative developmental processes. Drug treatments can be helpful in some instances (particularly for agitation and behavioral difficulties) but do not seem to address core social dysfunction (at least to date). Agents like the second-generation neuroleptic risperidone have been shown, in double-blind studies, to be more effective than placebo (McCracken et al. 2002).

Alternative and complementary treatments are common but lack substantive efficacy data. Parents should be helped to understand the

importance of pursuing proven treatments (Volkmar and Wiesner 2009). Except in selected cases, traditional psychotherapy is not usually helpful, and even in these cases, it often takes on a much more explicit, “life coaching,” model.

See Also

- Asperger's Disorder
- Autism Spectrum Disorder
- DSM-5
- DSM-IV
- Pervasive Developmental Disorder Not Otherwise Specified

References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. (erratum appears in *Nat Rev Genet.* 2008 Jun; 9(6):493). (Research Support, N.I.H., Extramural Research Support, Non-U.S. Gov't Review). *Nature Reviews Genetics*, 9(5), 341–355.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Bachevalier, J., & Loveland, K. A. (2006). The orbitofrontal-amygdala circuit and self-regulation of social-emotional behavior in autism. *Neuroscience and Biobehavioral Reviews*, 30(1), 97–117.
- Casanova, M. F. (2007). The neuropathology of autism. *Brain Pathology*, 17(4), 422–433.
- Chawarska, K., Klin, A., & Volkmar, F. (Eds.). (2008). *Autism spectrum disorders in infants and toddlers: Diagnosis, assessment, and treatment*. New York: Guilford Press.
- Coonrod, E. E., & Stone, W. L. (2005). Screening for autism in young children. In F. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed.). New York: Wiley.
- Coplan, J. (2000). Counseling parents regarding prognosis in autistic spectrum disorder. *Pediatrics*, 105(5), E65.
- Folstein, S., & Rutter, M. (1977). Genetic influences and infantile autism. *Nature*, 265(5596), 726–728.
- Grinker, R. R. (2007). *Unstrange minds*. New York: Basic Books.
- Gupta, A. R., & State, M. W. (2007). Recent advances in the genetics of autism. *Biological Psychiatry*, 61(4), 429–437.
- Hill, A. P., Zuckerman, K. E., Fombonne, E., Volkmar, F. R., Paul, R., Rogers, S. J., & Pelphrey, K. A. (2014). Epidemiology of autism Spectrum disorders. In *Handbook of autism and pervasive developmental disorders* (4th ed.). Hoboken: Wiley.
- Howlin, P., Moss, P., Savage, S., Bolton, P., & Rutter, M. (2015). Outcomes in adult life among siblings of individuals with autism. *Journal of Autism and Developmental Disorders*, 45(3), 707–718.
- Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of Autism and Childhood Schizophrenia*, 1(2), 119–145.
- Lord, C., & Corsello, C. (2005). Diagnostic instruments in autism spectrum disorders. In F. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed.). New York: Wiley.
- Loveland, K. A., & Tunali-Kotoski, B. (2005). The school-age child with an autistic spectrum disorder. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 247–287). Hoboken: Wiley.
- Mandell, D. S., Ittenbach, R. F., Levy, S. E., & Pinto-Martin, J. A. (2007). Disparities in diagnoses received prior to a diagnosis of autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37(9), 1795–1802.
- McCracken, J. T., McGough, J., Shah, B., Cronin, P., Hong, D., Aman, M. G., & Research Units on Pediatric Psychopharmacology Autism, Network. (2002). Risperidone in children with autism and serious behavioral problems. *The New England Journal of Medicine*, 347(5), 314–321.
- Minshew, N. J., Sweeney, J. A., Bauman, M. L., & Webb, S. J. (2005). Neurologic aspects of autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 453–472). Hoboken: Wiley.
- National Research, C. (2001). *Educating young children with autism*. Washington, DC: National Academy Press.
- O’Roak, B. J., & State, M. W. (2008). Autism genetics: Strategies, challenges, and opportunities. *Autism research: Official Journal of the International Society for Autism Research*, 1(1), 4–17. (Review).
- Offit, P. (2008). *Autism's false prophets*. New York: Columbia University Press.
- Pinkham, A. E., Hopfinger, J. B., Pelphrey, K. A., Piven, J., & Penn, D. L. (2008). Neural bases for impaired social cognition in schizophrenia and autism spectrum disorders [Research Support, N.I.H., Extramural Research Support, Non-U.S. Gov't]. *Schizophrenia Research*, 99 (1–3), 164–175.
- Reichow, B., & Wolery, M. (2009). Comprehensive synthesis of early intensive behavioral interventions for young children with autism based on the UCLA young autism project model. *Journal of Autism and Developmental Disorders*, 39(1), 23–41.
- Rutter, M. (1972). Childhood schizophrenia reconsidered. *Journal of Autism & Childhood Schizophrenia*, 2(4), 315–337.

- Rutter, M. (1978). Diagnosis and definitions of childhood autism. *Journal of Autism & Childhood Schizophrenia*, 8(2), 139–161. <https://doi.org/10.1007/BF01537863>.
- Rutter, M., Lebovici, S., Eisenberg, L., Snezhnevskij, A. V., Sadoun, R., Brooke, E., et al. (1969). A tri-axial classification of mental disorders in childhood: An international study. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 10(1), 41–61. Blackwell Publishing, United Kingdom.
- Rutter, M., Bailey, A., Bolton, P., & Le Couter, A. (1994). Autism and known medical conditions: Myth and substance. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 35(2), 311–322.
- Schultz, R. T., Gauthier, I., Klin, A., Fulbright, R. K., Anderson, A. W., Volkmar, F., & Gore, J. C. (2000). Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and Asperger syndrome. *Archives of General Psychiatry*, 57(4), 331–340.
- Shea, V., & Mesibov, G. B. (2005). Adolescents and adults with autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 288–311). Hoboken: Wiley.
- Smith, I. C., Reichow, B., & Volkmar, F. R. (2015). The effects of DSM-5 criteria on number of individuals diagnosed with autism spectrum disorder: A systematic review. *Journal of Autism and Developmental Disorders*, 45(8), 2541–2552.
- Volkmar, F. R., & Nelson, D. S. (1990). Seizure disorders in autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 29(1), 127–129.
- Volkmar, F., & Wiesner, L. (2009). *A practical guide to autism*. Hoboken: Wiley.

individuals with outstanding talents. Subsequent research has suggested that these skills are most commonly seen in the domains of art, music, and numerical calculation. A change in terminology from “idiot savant” to “savant syndrome” was later proposed by Treffert (1989) who also outlined a hierarchical system for categorizing levels of talent proficiency. In addition to avoiding the negative connotations of the earlier term, Treffert’s new term reflected an increased awareness that intellectual disability was not a necessary feature of the savant syndrome.

Although savant skills have been described in individuals with Tourette’s syndrome, frontotemporal dementia, manic depression, language impairment, and congenital blindness, the savant syndrome is most strongly associated with autism spectrum disorders. The early prevalence rate for savant skills, based on parental report, was 9.8%, a figure that was ten times greater than in intellectually handicapped populations (see Heaton and Wallace 2004, for details). However, more recent investigations of splinter and savant skills (Howlin et al. 2009; Bennett and Heaton 2012) observed greatly increased prevalence rates and suggested that up to a third of individuals with ASD may possess unusual talents.

Prominent theoretical accounts of autism have implicated atypical cognitive processing and enhanced perceptual discrimination in the emergence of talents (see Mottron et al. 2009). However, the results from several studies have reported superior working memory (Bölte and Poustka 2004; Bennett and Heaton 2017) and obsessional traits (Bennett and Heaton 2017) in individuals with savant talents, and current theoretical models do not account for these findings

There are several major challenges facing researchers working in this area. First are difficulties in determining exactly how the definitions of Treffert’s (1989) three-tier categories should be operationalized. Quantifying skill levels in domains like art and music, where standardized assessments are not available, introduces a degree of subjectivity that could compromise cross-group comparisons. Some savant skills, for example, calendar calculating, are rare in typical

Autistic Regression

► Acquired Autism

Autistic Savants

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Definition

In 1866, Edouard Seguin coined the term “idiot savant” to describe intellectually handicapped

populations, and questions about appropriate comparison groups must be carefully addressed. There is a final fundamental question that results from the definitional shift from “idiot savant” to savant syndrome. There is currently no consensus about whether intellectually able, talented individuals with autism should be accorded savant status (see Heaton and Wallace 2004; Miller 1998). The rise in the numbers of intellectually able individuals diagnosed with ASD and the observed increase in the prevalence of special talents in these groups (Howlin et al. 2009; Bennett and Heaton 2012) bring the importance of resolving this question into focus. The study of savant syndrome has implications for both theory and practice, and the development of new definitions and methodologies will be an important future goal for psychologists working in this area.

See Also

- [Enhanced Perceptual Functioning](#)
- [Treffert, Darold](#)
- [Weak Central Coherence](#)

References and Reading

- Bennett, E., & Heaton, P. (2012). Is talent in autism spectrum disorders associated with a specific cognitive and behavioural phenotype? *Journal of Autism and Developmental Disorders*, 42(12), 2739–2753.
- Bennett, E., & Heaton, P. (2017). Defining the clinical and cognitive phenotype of child savants with autism spectrum disorder. *Current Pediatric Research*, 21(1), 140–147.
- Bölte, S., & Poustka, F. (2004). Comparing the intelligence profiles of savant and non-savant individuals with autistic disorder. *Intelligence*, 32, 121–131.
- Heaton, P., & Wallace, G. L. (2004). Annotation: The savant syndrome. *Journal of Child Psychology and Psychiatry*, 45(5), 899–911.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2009). Savant skills in autism: Psychometric approaches and parental reports. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, 364(1522), 1359–1367.
- Miller, L. K. (1998). Defining the savant syndrome. *Journal of Developmental and Physical Disabilities*, 10, 73–85.
- Mottron, L., Dawson, M., & Soulières, I. (2009). Enhanced Perception in savant syndrome: Patterns, structure and creativity. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, 364(1522), 1385–1391.
- Treffert, D. A. (1989). *Extraordinary people: Understanding “idiot-savants”*. New York: Harper & Row.

Autistic Spectrum Quotient (AQ-9)

- [Self-Report Autism Scales for Adults](#)

Autistic Traits and Auditory Discrimination Skills

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Definition

Autistic traits are normally distributed, heritable, are stable cross-culturally, and are also apparent in relatives of autistic people. Traits associated with the autism spectrum can be measured with tools such as the Autism-Spectrum Quotient (Baron-Cohen et al. 2001) and allow for designs that take advantage of the variability of autistic traits across individuals with or without a diagnosis. Autistic traits measured in this way correlate with behaviors and performance on tests in non-autistic samples similar to performance in autistic samples (Almeida et al. 2010; Stewart and Ota 2008; Stewart et al. 2009).

In the context of autism research, auditory discrimination has been studied primarily on the dimension of pitch (or rather its acoustic correlate, fundamental frequency), and also of intensity and duration.

Historical Background

Autistic people tend to outperform their non-autistic counterparts on perceptual tasks where there is a benefit in being able to process the local detail over the “gestalt.” Mottron and colleagues suggest that autistic people have Enhanced Perceptual Functioning in which there is an enhanced ability to process detailed perceptual information (see entry ► [Enhanced Perceptual Functioning](#)). Happe and colleagues (see ► [“Weak Central Coherence”](#) entry), in comparison, suggest that autistic people show Weak Central Coherence in which the ability to focus on the “local” detail is preserved or enhanced but there is a weakening of the ability to integrate information into a meaningful whole or a “gestalt.”

Autistic children and adults show enhanced performance in a range of tasks which depend on a detailed, accurate representation of the stimuli. Within the visual domain, autistic individuals show reduced interference from the whole picture when completing whole versus segmented patterns in an adapted block design task (Shah and Frith 1993). Similarly, enhanced performance has been found on the embedded figures task, in which participants are required to find a design which is hidden in a larger picture. Tasks such as these require the participant to ignore the “gestalt” of the whole and focus on the local detail in complex designs. This enhanced performance is also shown in those with high levels of autistic traits but who do not have a diagnosis of autism. In a similarly adapted block design in which there were whole/unsegmented designs and segmented designs, Stewart et al. (2009) found superior performance in the whole/unsegmented designs in those who scored high on autistic character traits versus those who scored low, whereas the groups performed equivalently on the segmented designs. This pattern of performance was also found for a radial search task, where those with high autistic trait scores outperformed those who low autistic trait scores (Almeida et al. 2010).

A similar effect has also been found within the auditory domain. One such demonstration involves the so-called Ganong effect, which reflects the extent to which phonetic categorization shifts to make the percept a known word. For example,

when adults listen to voice onset time (VOT) continua between a real word and a non-word, such as *gift-kift* and *kiss-giss*, their judgment of the mid-point stimuli tend to shift toward the real-word end (i.e., *gift* and *kiss*) even when the onset consonants in the stimuli are acoustically identical. However, those who scored higher on AQ were less influenced by the lexical (“global”) information and were more likely to respond in terms of the actual acoustic (“local”) difference in both adults (Stewart and Ota 2008) and children (Ota et al. 2015). Furthermore, Stewart and colleagues found that auditory discrimination of speech stimuli along the same dimension (i.e., VOT difference) was less categorical in autistic adults than in neurotypicals (Stewart et al. 2018). In other words, while neurotypicals were more sensitive to acoustic differences between categories (i.e., /g/ vs. /k/) than those within categories, autistic individuals showed a more invariant level of sensitivity to acoustic differences. In their literature review, Haesen and colleagues (Haesen et al. 2011) report a range of other evidence consistent with the idea that autistic individuals’ auditory processing is locally oriented and less susceptible to global interference and propose that this pattern of auditory performance in autism can be explained by atypical brain lateralization.

There is also a body of evidence in the auditory domain according to which autistic individuals have an enhanced ability to discriminate perceptual stimuli which is in line with the model of Enhanced Perceptual Functioning (see ► [“Enhanced Perceptual Functioning”](#) entry). Documented most reliably is the observation that autistic individuals exhibit better discrimination of differences in pitch compared to non-autistic individuals. For instance, Bonnel et al. (2010) found enhanced ability in autistic children and young adults to categorize pure tones on the basis of their pitch, but not on the basis of intensity. O’Riordan and Passetti (2006) used an adaptive paradigm in which, across trials, a target tone gradually became closer to a reference tone in fundamental frequency. They found that autistic children tended to shift from a “different” to “same” response later than non-autistic children, which can be interpreted as evidence for higher pitch sensitivity in autistic children. Jones et al.

(2009) assessed individual thresholds on frequency, intensity, and duration in an autistic adolescent sample and controls. While no differences were found at the group level, a subset of the autistic adolescents, who were identified as having delayed language onset and average intellectual ability, showed an enhanced ability to discriminate stimulus differences in fundamental frequency. This effect did not extend to intensity or duration. However, not all studies have found autistic individuals to have better auditory perceptual ability. In fact, Kargas et al. (2015) report deficits, rather than enhancement, in auditory discrimination of frequency, intensity, and duration in autistic adults as compared to age-matched controls.

Current Knowledge

Further evidence for an enhanced perceptual processing in autistic people as well as in those who score high on autistic traits has been found both in the visual and auditory domains. Questions arise, therefore, as to whether there is a common perceptual mechanism across modalities or features, and whether autism or autistic traits explain the differences in perceptual processing, or whether there is another factor associated with this enhancement, and what the mechanism behind such an enhancement would be.

Stewart et al. (2018) asked whether there was a correlational pattern of enhanced discrimination across pitch, time, and intensity in non-autistic participants who scored on autistic character traits. Participants were asked to discriminate between two tones for the pitch and intensity tasks and between two pairs of tones in the time-interval task. Each trial consisted of one reference and one target stimulus, and the position of the target stimulus was randomized across with equal probability across trials. Similar to other studies (Bonnell et al. 2010; Jones et al. 2009), the references were fixed for the pitch task. However, both a fixed and a variable reference task were used for time interval. Stewart and colleagues found that the pattern of enhancement did occur in those who scored high on autistic traits and that autistic trait scores correlated with both pitch discrimination

and time-interval discrimination in the fixed reference tasks. However, they did not find any relationship between autistic traits and discrimination of intensity or time interval in the variable reference task. They suggest that the correlations show an ability to form stable perceptual representations of auditory events in the time and pitch dimensions. Furthermore, they suggest that such stable perceptual representations may be formed because both pitch and time interval can be coded in an absolute fashion, whereas intensity cannot. The findings are in line with the Enhanced Perceptual Functioning model (see ► [“Enhanced Perceptual Functioning”](#) entry), which suggests that autistic people have enhanced low-level processing of basic perceptual information. However, a caveat to this explanation for enhanced discrimination ability is that there could be confounding factors.

On potential factor may be the design of tasks and differing task demands (see, e.g., Barry et al. 2013). Banai and Ahissar (2006) suggest that variation in performance, due to differing task demands, may be due to the ability to form an accurate representation of the auditory stimulus. This ability to form a stable representation of an auditory stimulus is something which is thought to be impaired in dyslexic participants (Banai and Ahissar 2006) but enhanced in those who score high on autistic traits (Stewart et al. 2018).

It may also be important to take into account language and literacy abilities in the design of the study. Pitch (frequency) discrimination deficits are associated with language and literacy impairments, although there may be no causal relationship rather discrimination differences may be a risk factor for differences in reading and literacy (e.g., McArthur and Bishop 2004). To note, is that Jones et al. (2009) showed the converse in autistic people, in that those with delayed language onset had an enhanced ability to discriminate frequency.

Effects could be due to differences in IQ, as evidence shows that perceptual ability, for instance, pitch discrimination, is strongly related to nonverbal or performance IQ ($r = 0.92$; Deary et al. 2004). There are a number of paradigms where IQ is related to discrimination. For instance, those who score high on a measure of non-verbal IQ (Raven's Progressive Matrices)

show shorter response times and higher accuracy on an oddball-type task of auditory discrimination using tones that differed in frequency (De Pascalis et al. 2008). In this study, participants heard a series of tones, 85% of which were the baseline tone and 15% were the target tone with a higher frequency. The participants' task was to indicate when they heard the target tone. In addition, Kuppen and colleagues (Kuppen et al. 2011) found that children with low IQ and who were poor readers had higher thresholds for frequency discrimination (i.e., they were worse at discriminating) than chronologically age-matched controls. Studies in autistic people and on autistic traits assessing perceptual discrimination tend to match on IQ by group but not individually. In studies, where the groups have been matched on IQ, IQ did not have a significant relationship with discrimination for pitch, time, or intensity, nor with autistic traits (e.g., Stewart et al. 2018). However, it may be important to match individually on IQ and on other factors such as reading ability.

It is important to note that IQ tests may be performed differently by autistic people and those who score on autistic character traits. For instance, Dawson found large differences between performance on the Wechsler scales of intelligence versus the Raven's Progressive Matrices in autistic people but not in non-autistic people, suggesting that in some cases, IQ may be underestimated (or potentially overestimated) in autistic people due to the measure used. Even within the Raven's Progressive Matrices, researchers have found that those who score high on autistic traits performed better performance on visuospatial items than on verbal-analytic. Given that IQ is known to relate to perceptual performance, it is of key importance to adequately control for IQ in these studies.

Meilleur et al. (2014) assessed performance in autistic and non-autistic adults across two visual and two auditory tasks, using both the Wechsler Intelligence Scale and the Raven's Progressive Matrices as IQ measures. The visual tasks were a modified block design task and a luminance contrast discrimination, and the auditory tasks were pitch and melody discrimination. The autistic participants showed enhanced pitch discrimination

even when controlling for IQ (both measures). But differences in the performance on the block design disappeared when IQ was controlled for. There were no group differences in the low-level visual task or in the melody discrimination. The authors recommend that a wide range of tasks are used in order to test whether there is an underlying common perceptual factor which may drive performance on perceptual tasks in autistic people but not in non-autistic controls. The tasks chosen were limited to four, and these may not be the most sensitive tasks. In addition, the melody discrimination task may not require "global" processing as the pitch contours that are used consist only of simple ascending and descending pitch directions which may be regarded as a "local" feature.

Future Directions

There is a substantial body of work that assessed perceptual and specifically auditory processing across the autism spectrum, which has helped identify where and why there may be particular enhancements. It is of interest to identify which aspects of behavior also occur in those without a diagnosis of autism but who score highly on autistic character traits. This is important as we know that aspects of autistic traits can be predictive of a range of behaviors. Future research must take into account a range of designs and use a range of stimuli to help identify potential mechanisms behind these identified differences. Techniques such as brain imaging may also be able to shed some light on the underlying mechanisms. In addition, the samples must be well controlled and information regarding IQ, reading, and literacy taken into account. It is of societal interest to assess how far these differences in processing may be related to aspects of functioning, health, and well-being.

See Also

- ▶ [Enhanced Perceptual Functioning](#)
- ▶ [Perception](#)
- ▶ [Weak Central Coherence](#)

References and Reading

- Almeida, R. A., Dickinson, J. E., Maybery, M. T., Badcock, J. C., & Badcock, D. R. (2010). A new step towards understanding Embedded Figures Test performance in the autism spectrum: The radial frequency search task. *Neuropsychologia*, 48(2), 374–381. <https://doi.org/10.1016/j.neuropsychologia.2009.09.024>.
- Banai, K., & Ahissar, M. (2006). Auditory processing deficits in dyslexia: Task or stimulus related? *Cerebral Cortex*, 16, 1718–1728. <https://doi.org/10.1093/cercor/bhj107>.
- Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from Asperger syndrome/high-functioning autism, males and females, scientists and mathematicians. [erratum appears in J Autism Dev Disord 2001 Dec;31(6):603]. *Journal of Autism & Developmental Disorders*, 31(1), 5–17.
- Barry, J. G., Weiss, B., & Sabisch, B. (2013). Psychophysical estimates of frequency discrimination: More than just limitations of auditory processing. *Brain Sciences*. <https://doi.org/10.3390/brainsci3031023>
- Bonnel, A., McAdams, S., Smith, B., Berthiaume, C., Bertone, A., Ciocca, V., . . . Motttron, L. (2010). Enhanced pure-tone pitch discrimination among persons with autism but not Asperger syndrome. *Neuropsychologia*. <https://doi.org/10.1016/j.neuropsychologia.2010.04.020>
- De Pascalis, V., Varriale, V., & Matteoli, A. (2008). Intelligence and P3 components of the event-related potential elicited during an auditory discrimination task with masking. *Intelligence*, 36(1), 35–47. <https://doi.org/10.1016/j.intell.2007.01.002>.
- Deary, I. J., Bell, P. J., Bell, A. J., Campbell, M. L., & Fazal, N. D. (2004). Sensory discrimination and intelligence. *The American Journal of Psychology*, 117(1), 1–18. <https://doi.org/10.2307/1423593>. T4 – Testing Spearman’s Other Hypothesis M4 – Citavi.
- Haesen, B., Boets, B., & Wagemans, J. (2011). A review of behavioural and electrophysiological studies on auditory processing and speech perception in autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5(2), 701–714. <https://doi.org/10.1016/j.rasd.2010.11.006>.
- Jones, C. R. G., Happé, F., Baird, G., Simonoff, E., Marsden, A. J. S., Tregay, J., . . . Charman, T. (2009). Auditory discrimination and auditory sensory behaviours in autism spectrum disorders. *Neuropsychologia*, 47(13), 2850–2858. <https://doi.org/10.1016/j.neuropsychologia.2009.06.015>
- Kargas, N., López, B., Reddy, V., & Morris, P. (2015). The relationship between auditory processing and restricted, repetitive behaviors in adults with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 45(3), 658–668. <https://doi.org/10.1007/s10803-014-2219-2>.
- Kuppen, S., Huss, M., Fosker, T., Fegan, N., & Goswami, U. (2011). Basic auditory processing skills and phonological awareness in low-IQ readers and typically developing controls. *Scientific Studies of Reading*, 15(3), 211–243. <https://doi.org/10.1080/10888431003706291>.
- McArthur, G. M., & Bishop, D. V. M. (2004). Frequency discrimination deficits in people with specific language impairment: Reliability, validity, and linguistic correlates. *Journal of Speech, Language, and Hearing Research*, 47(3), 527–541. [https://doi.org/10.1044/1092-4388\(2004/041\)](https://doi.org/10.1044/1092-4388(2004/041)).
- Meilleur, A. A. S., Berthiaume, C., Bertone, A., & Motttron, L. (2014). Autism-specific covariation in perceptual performances: “g” or “p” factor? *PLoS ONE*. <https://doi.org/10.1371/journal.pone.0103781>
- O’Riordan, M., & Passetti, F. (2006). Discrimination in autism within different sensory modalities. *Journal of Autism & Developmental Disorders*, 36(5), 665–675.
- Ota, M., Stewart, M. E., Petrou, A. M., & Dickie, C. (2015). Lexical effects on children’s speech processing: Individual differences reflected in the Autism-Spectrum Quotient (AQ). *Journal of Speech, Language, and Hearing Research*, 58(2), 422–433. https://doi.org/10.1044/2015_JSLHR-L-14-0061.
- Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 34(8), 1351–1364.
- Stewart, M. E., & Ota, M. (2008). Lexical effects on speech perception in individuals with “autistic” traits. *Cognition*, 109(1), 157–162. <https://doi.org/10.1016/j.cognition.2008.07.010>.
- Stewart, M. E., Watson, J., Allcock, A.-J., & Yaqoob, T. (2009). Autistic traits predict performance on the block design. *Autism*, 13(2), 133–142. <https://doi.org/10.1177/1362361308098515>.
- Stewart, M. E., Griffiths, T. D., & Grube, M. (2015). Autistic traits and enhanced perceptual representation of pitch and time. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-015-2517-3>
- Stewart, M. E., Petrou, A. M., & Ota, M. (2018). Categorical speech perception in adults with autism spectrum conditions. *Journal of Autism and Developmental Disorders*, 48(1), 72–82. <https://doi.org/10.1007/s10803-017-3284-0>.

Autistic Traits in Prison Populations

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Definition

The link between ASD and criminality, including extremely violent offenses as serial killing or mass murdering (Allely et al. 2014), but also

concerning cybercrime (Seigfried-Spellar et al. 2015) has gained widespread public notoriety. Nevertheless, the available evidence does not favor the hypothesis that autistic individuals are more likely to engage in unlawful behavior than the general population and may in fact be less so. Yet, the current literature points to an overrepresentation of people with ASD or autistic traits in the criminal justice system (CJS), even though they are apparently not more likely to commit illegal acts. Besides the implications this has in terms of service provision to this population, whose needs seem to be poorly met in several instances across the legal system, it also highlights the importance of determining which factors increase the risk for autistic individuals to be incarcerated, in order to optimize care and target interventions to prevent this dreadful outcome for autistic people.

Historical Background

Although the field of intellectual disability (ID) in the CJS has a long tradition, with numerous studies on the prevalence of ID in different forensic settings and its implications, the focus in other neurodevelopmental disorders has only recently gained attention from the scientific community, and more so for attention deficit hyperactivity disorder (ADHD), for which a large meta-analysis of prevalence is already available, than for ASD (Young et al. 2015). This is hypothesized to be a consequence of the late recognition of these disorders in adulthood, which were mainly dealt within child services and not officially recognized by the DSM until the 1980s (King and Murphy 2014). Moreover, it is likely that the higher prevalence and the more externalizing symptoms and psychiatric comorbidities of ADHD have led to a greater investment in this area, and not ASD.

However, the association between violent behavior and ASD, independently of ID, was already mentioned by Hans Asperger in 1944 in one of his case descriptions (Im 2016), and nonviolent, but unlawful behavior was also described by Lorna Wing in her 1981 paper on Asperger's (Woodbury-Smith and Dein 2014).

The generalized interest in the relationship between autistic traits and criminality also began to rise in association with extremely violent offenses, committed by (allegedly) autistic individuals, which received extensive media coverage (Allely et al. 2014). Despite the posthumous, not rigorous nature of most diagnosis, the possibility that autistic characteristics could render people on the spectrum liable to violent criminality interested the scientific community, and studies on the subject began to emerge.

Besides analyzing individual cases of offenders and studying criminal behavior in autistic cohorts, some authors have taken a different approach to this subject, beginning in the 1990s, namely, obtaining the prevalence of ASD or autistic traits among prisoners, which has led to the recognition that there is a higher than expected number of autistic individuals in the CJS and has opened a field of interest of its own, expected to affect policy-making and service provision throughout the legal system and specifically in custodial settings (Robertson and McGillivray 2015).

Current Knowledge

Prevalence

Since it emerged, the scientific interest in the subject has grown in the past few years.

The literature in general points to an overrepresentation of individuals with ASD in the CJS, with estimated prevalences ranging from 2% to 27% in a systematic review that included seven studies (King and Murphy 2014). Figures were higher for samples obtained in psychiatric hospitals than in the general prison system, but this is in excess of the expected population prevalence of around 1%, even for the lower end of the interval. Nonetheless, several methodological pitfalls prevent the drawing of rigorous conclusions, as most of the studies employed biased samples (e.g., those referred for forensic psychiatric evaluation only) and substantial heterogeneity existed across studies regarding the terminology and the diagnostic procedures. Also of note, most of the samples were exclusively male, with fewer than 200 women included.

Most prevalence studies use a categorical approach to autism (i.e., either presence or absence of an ASD diagnosis), but there is evidence that individuals without an ASD diagnosis but with a high load of autistic traits are also overrepresented in the CJS, although less publications have addressed this issue. Two studies used the Autism Spectrum Quotient (AQ) to measure autistic traits among prison inmates albeit without a control group. Fazio et al. (2012), in their prevalence study with a sample of 431 male inmates, obtained a mean score of 20; Robinson et al. (2012), in a study with 126 prisoners of varying ages and both genders, reported an average score of 20,1. This contrasts with the reported mean AQ scores of around 18 in the general male adult population. A third study (Loureiro et al. 2018) that measured the AQ in a prison sample of 101 male inmates and included a control group found significant differences regarding autistic traits between the groups (mean AQ of 20,6 in the prisoners and 18,1 in the controls; $OR = 1,13$, $p = 0.002$) after controlling for potential confounders – age, education, psychopathology, psychopathy, and ADHD.

Core Autistic Traits and Imprisonment

Taken together, these studies suggest that autistic individuals (either given an ASD diagnosis or not) are overrepresented in forensic settings. Several reasons are invoked to explain these findings, namely, the characteristics of autistic people themselves, which may confer a higher risk of engaging in illegal activity and/or being sentenced to prison (Haskins and Silva 2006; Lerner et al. 2012; Im 2016).

Individuals with an ASD may be at increased risk for criminality due to the failure to understand social rules and hierarchies and the inadequacy and/or the consequences of some behaviors. They typically have difficulties in evaluating the emotions, thoughts, and intentions of others, leading to misunderstandings in reciprocal relationships. Emotional regulation is commonly impaired, and people on the autism spectrum can be extremely obsessive in the pursuit of their special interests, disregarding dangers and (legal) obstacles in the way. Also, their naivety, paired with an eagerness

to comply and avoid confrontation, renders these individuals susceptible to exploitation by others: they are easy to manipulate into (unintentionally and unknowingly) committing illegal acts.

The same holds true for autistic individuals without an ASD diagnosis, due to the aforementioned cognitive and affective styles, without the social reclusion imposed by more severe phenotypes, not to mention that aggression and other problem behavior are seldom criminalized for individuals with higher degrees of incapacity but are unlikely to be tolerated when no obvious disability is found (Michna and Trestman 2016).

While some studies point to a higher likelihood of criminal behavior among people with an ASD, the majority reject this hypothesis (King and Murphy 2014) and, in fact, find evidence of lower rates of criminality in this population. Only one population-based study tried to address this issue, with a cohort of almost 3.000.000 subjects aged 15–27 years (5739 diagnosed with an ASD). It reported and unadjusted relative risk (RR) of criminality, for ASD individuals without an ID, of 1,39. However, this association did not remain after adjusting for ADHD or conduct disorder (Heeramun et al. 2017). In fact, the tendency to rigidly follow explicit rules has been pointed as an autistic feature that promotes law-abiding behavior (Im 2016).

Moreover, no confirmation was found for the hypothesized link between autistic traits and particularly violent crimes, such as murder or rape, although the available evidence does seem to find an increased risk for arson (King and Murphy 2014) and personal offenses in general and a diminished likelihood of property crimes. The possibility of the highly mediatized link between autism and cybercrime is also at the moment still under debate (although a weak correlation between autistic traits and deviant computer behavior is found in the very few studies available on the subject – Seigfried-Spellar et al. 2015).

So, other hypotheses have been raised regarding the reasons for the overrepresentation of autistic individuals in the CJS, without an obvious tendency among ASD individuals to commit crimes.

One other possibility is that the main characteristics of the core autistic phenotype may

prevent individuals on the spectrum from avoiding incarceration. It has been suggested that features such as the lack of appropriate social support networks, poor communication skills, rigidity, literality, and catastrophic reactions to anxiety not only make it more unlikely for the autistic offender to avoid detection but also have a negative impact on the detention process and especially the trial itself, where the autistic person is often perceived as remorseless and uncaring or giving false accounts (Woodbury-Smith and Dein 2014; Robertson and McGillivray 2015; Michna and Trestman 2016). In that scenario, ASD individuals would have greater rates of incarceration than their counterparts, in the exact same legal contexts.

Although autistic traits can potentially have a negative impact in all the steps that can lead to incarceration, some seem to be especially

detrimental in specific stages of the process. Table 1 attempts to summarize the available literature on how core autistic features are thought to affect unlawful behavior, the sentencing process, and the amount of time spent in prison.

Other Risk Factors

On the other hand, some authors argue that the higher prevalence of illegal behavior in autistic people is more related to psychiatric comorbidities such as psychosis, personality and affective disorders or substance abuse, and not to autistic traits themselves (Im 2016; Heeramun et al. 2017). In general, offenders with ASD are less likely to have coexisting drug or alcohol misuse than the general prison population, but the presence of other mental health issues is a contributing factor for criminality in ASD, as it is for the neurotypical population.

Autistic Traits in Prison Populations, Table 1 Putative factors contributing to the higher than expected prevalence of autistic people in prisons

Factors that affect imprisonment and sentence length	Negative implications of autistic traits
Liability to commit criminal offenses (either by own moto or influenced by others)	Aggressive outbursts directed to others due to poor emotional regulation, intolerance to the unexpected, sensory issues Obsessional pursuit of interests Difficulty interpreting other's intentions and emotions and forming appropriate relationships Difficulty anticipating and appreciating consequences of one's own behavior Utilization as "stooges" (psychopathic individuals prey upon naïve and acquiescent individuals to perform their risky criminal activities)
Increased probability of more severe penalties than neurotypical peers (in the same legal context)	Reaction to arrest (aggression, freezing while others escape, etc.) Tendency to immediately confess, even to additional crimes not under trial Difficulty interacting with different players throughout the CJS Over acquiescence and suggestibility during interviews Facial expression and prosody come across as arrogance and lack of remorse Idiosyncratic use of language, literality, use of words without complete understanding of meaning and poor appraisal of timelines of events may give rise to false testimony
Extended sentences due to inside prison behavior	Victimization by fellow inmates results either in isolation placement for protection (and further failure to socialize) or heteroaggressivity (including displaced toward authority figures), preventing earlier release on "good behavior" terms Failure to engage in and/or successfully complete rehabilitation programs that are usually group-based and poorly fitted for autistic individuals Increased risk of institutionalization

It has also been suggested that the association between criminality and autism may only occur for some individuals on the spectrum who display callous-unemotional traits and are phenotypically closer to psychopaths (Rogers et al. 2006).

Indeed, much debate has been raised about this issue since the original description of Asperger's "Autistic Psychopathy." Although the term psychopathy was, at that time, employed as a synonym of personality disorder, some authors have argued that it may well have "more explanatory power" than previously perceived (Fitzgerald 2001), and the debate around this subject has given rise to a substantial body of knowledge.

Psychopathy is most commonly regarded as a construct that encompasses such features as dominance-seeking, cruelty, manipulation, predatory violence, impulsive, reckless behavior, lack of emotional reactivity, and affective indifference and is primarily associated with poor empathy processing. Empathy is defined as the capacity to feel or imagine another person's emotional experience, and an atypical empathy development results in antisocial behavior. Autism and psychopathy are viewed as two prototypical "disorders of empathy" (Bird and Viding 2014).

So far, the favored hypothesis seems to be that of a divergent profile between psychopaths and autistic people, with the first lacking emotional empathy (the vicarious experiencing of another person's emotions) and the latter lacking cognitive empathy (the capacity of understanding other people's perspectives or theory of mind).

However, regardless of the shared risk factors with the population at large, it should be noted that, in the few studies that looked specifically for the motive why autistic people committed the offenses, they all seemed to be related to the autistic traits themselves, although some sort of acute stressor was usually also found (Haskins and Silva 2006; Helverschou et al. 2015; Im 2016). Moreover, Loureiro et al. (2018) found autistic traits to be independent predictors of incarceration, when taking into account variables as general levels of psychopathology, ADHD, and psychopathy.

In conclusion, autistic characteristics can act as independent risk factors for being sentenced to prison, besides those shared with the population

in general – not only psychiatric comorbidities and psychopathy but also low socioeconomic status, low IQ, and a history of adversity in childhood (Helverschou et al. 2015). This needs to translate into differentiated service provision for this population, who must benefit from tailored interventions that address autism-specific criminogenic needs along with those risk factors shared with the non-autistic offender population.

Service Provision for Autistic People in Prisons

Concerns have been raised about the adequacy of prison or even general psychiatric high-security facilities to accommodate the needs of this particular population during the sentence. There is even less information concerning this subject, although a few studies have begun to shed some light on the experiences of autistic people during the process of arrest, investigation, trial, and imprisonment (Helverschou et al. 2015).

While the personal experiences of people with ASD are primarily negative for the entire process, they are less consistently so when it comes to the imprisonment itself. In fact, the social isolation inherent to life in prison may be perceived as less stressful to autistic individuals than it is for the general population, and some aspects of prison life are regarded as positive, namely, the structured environment, with predictable routines and organized activities that allow for less free time. Not all establishments offer this type of structure, though, and some individuals fare worse under less supervised environments, especially if housed with unselected prisoners, when predation and bullying by fellow inmates is a concern. The ability to avoid conflict in overcrowded facilities with truculent, antisocial individuals is dependent on a highly developed social cognition to interpret and act accordingly to the hierarchies of power and control that ASD people distinctly lack. Indeed, autistic prisoners seem to be at a greater risk of victimization while in prison and to spend more time in solitary confinement for their own protection, although data is particularly scarce and only a few individual cases have been published (Helverschou et al. 2015). It has also been hypothesized that people on the spectrum may be more susceptible to institutionalization (the loss of personal sense of responsibility for one's own acts and dependency

upon others to attend to daily living tasks) and thus have increased difficulties adjusting to life after prison (Robertson and McGillivray 2015).

In the past few years, efforts have been made to accommodate people with ASD throughout the CJS, although these are mainly localized to more affluent countries. For instance, the UK National Autistic Society keeps a comprehensive and updated “Criminal Justice” section with information for professionals who may come into contact with autistic individuals in the different settings along the CJS (<https://www.autism.org.uk/professionals/others/criminal-justice.aspx>) and provides autism accreditation specifically for prisons. The placement of autistic individuals under structured and well-supervised environments, paired with other prisoners that share the same traits and are perceived as more stable and less invasive by the staff, seems to increase the level of adjustment to prison life (Helterschou et al. 2017).

A related problem is the issue of underdetection of autism in prisons. Even if a facility has the capacity to put in place differentiated strategies to accommodate for autistic individuals, they still have to be identified first. In some of the prevalence studies conducted so far, autism was only diagnosed during the study interviews, which implies a low rate of detection through that point (Heeramun et al. 2017; Helterschou et al. 2015). There is a distinct possibility that the majority of autistic individuals, especially those on the less severe end of the spectrum, remain undiagnosed throughout the time that they remain in prison, even more so in less developed countries, where autism awareness levels are much lower and healthcare, social, and legal system’s budgets are tight.

Future Directions

There is now a growing interest in the field of ASD and autistic traits in the CJS and particularly in prison settings that is hoped to aid in the development of better services for the needs of those autistic individuals that come to experience imprisonment. Nevertheless, there is still a wide gap to overcome in many domains, as Woodbury-Smith and Dein (2014) and King and Murphy (2014) have summarized in their reviews. As

estimates of offending in the autistic population are fortunately low and the available estimates of ASD prevalence in custodial settings are not too high either, collaborative efforts among investigation centers across different countries are necessary to draw robust conclusions.

Figures for the prevalence of ASD in prisons require clarification. The available studies have methodological flaws that preclude the drawing of safe conclusions and employ differing diagnostic methodologies that mostly lack developmental corroboration from relatives. It would be beneficial to study unbiased samples with robust and standardized tools that would allow to pool results from different countries to better estimate the true prevalence of autism in prisons, as this is a *sine qua non* condition for budget allocation to specialist services in this area. Woodbury-Smith and Dein (2014) also raise the important question of the almost complete lack of data regarding the female population, which parallels the lag in this regard for the entire ASD population.

Moreover, specific recommendations on diagnostic procedures for those individuals who remain undiagnosed up to their imprisonment would be an important step toward the better care for autistic people while in prison. Neurodevelopmental disorders should be an integral part of standardized interviews that guide the initial health assessment when a person first enters the prison system, as exemplified by the Comprehensive Health Assessment Tool – CHAT (The Offender Health Research Network 2013).

Another important issue that demands a more thorough approach is the systematization of which specific autistic traits and environmental triggers are more likely to contribute or prevent criminal behavior and in what way. Only through extensive data collecting from interviews of convicted autistic offenders and their close contacts could a definite pattern of motives begin to emerge. This would enable the development of specific, individualized interventions to address these issues in a timely fashion, should they emerge, and prevent the offending behavior when possible, either through environmental modifications, therapy, or psychotropic medication.

For those already serving a prison sentence, there is a need to adapt existing programs or

develop other therapeutic interventions aimed at preventing reoffending, as the existing traditional cognitive and group-based alternatives may not be suitable for autistic individuals. Moreover, a more general habilitative approach, including social cognition training, may be necessary to achieve the best results (Woodbury-Smith and Dein 2014). On the whole, it seems that the general risk-need-responsivity method to evaluate and manage risk in offender populations (Bonta and Andrews 2007) can be applied to autistic individuals, as it is already grounded on individual criminogenic needs and static and dynamic risk factors.

Lastly, the interventions would then have to prove their usefulness against objective outcome measures, as living arrangements, employment, relationships, and recidivism. As cost is higher for more specialized services, there is also a need to compare outcomes among different pathways of care (autism accreditation or specialist consultations in prisons or general forensic psychiatric facilities versus specialized autism custodial settings).

See Also

- ▶ [Conduct Disorder](#)
- ▶ [Legal System Involvement](#)
- ▶ [Stalking](#)
- ▶ [Video Games and Violence](#)
- ▶ [Violence and ASD](#)

References and Reading

- Allely, C. S., Minnis, H., Thompson, L., Wilson, P., & Gillberg, C. (2014). Neurodevelopmental and psychosocial risk factors in serial killers and mass murders. *Aggression and Violent Behavior, 19*, 288–301.
- Bird, G., & Viding, E. (2014). The self to other model of empathy: Providing a new framework for understanding empathy impairments in psychopathy, autism and alexithymia. *Neuroscience and Biobehavioral Reviews, 47*, 520–532.
- Bonta, J., & Andrews, D. A. (2007). Risk-need-responsivity model for offender assessment and rehabilitation. *Rehabilitation, 6*(1), 1–22.
- Fazio, R. L., Pietz, C. A., & Denney, R. L. (2012). An estimate of the prevalence of autism-spectrum disorders in an incarcerated population. *Open Access Journal of Forensic Psychology, 4*, 69–80.
- Fitzgerald, M. (2001). Autistic psychopathy. *Journal of the American Academy of Child & Adolescent Psychiatry, 40*(8), 870.
- Haskins, B. G., & Silva, J. A. (2006). Asperger's disorder and criminal behaviour: Forensic-psychiatric considerations. *The Journal of the American Academy of Psychiatry and the Law, 34*, 374–384.
- Helverschou, S. B., Rasmussen, K., Steindal, K., Sondannaa, E., Nilsson, B., Notetstad, J. A. (2015). Offending profiles of individuals with autism spectrum disorder: A study of all individuals with autism spectrum disorder examined by the forensic psychiatric service in Norway between 2000 and 2010. *Autism, 19*(7):850–8.
- Heeramun, R., Magnusson, C., Gumpert, C. H., Granath, S., Lundberg, M., Dalman, C., et al. (2017). Autism and convictions for violent crimes: Population-based cohort study in Sweden. *Journal of the American Academy of Child & Adolescent Psychiatry, 56*(6), 491–497.
- Helverschou, S. B., Steindal, K., Nøttestad, J. A., & Howlin, P. (2017). Personal experiences of the criminal justice system by individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 47*(2), 340–346.
- Im, D. S. (2016). Template to perpetrate: An update on violence in autism spectrum disorder. *Harvard Review of Psychiatry, 24*, 14–35.
- King, C., & Murphy, G. H. (2014). A systematic review of people with autism spectrum disorder and the criminal justice system. *Journal of Autism and Developmental Disorders, 44*(11), 2717–33.
- Lerner, M. D., Haque, O. S., Northrup, E. C., Lawer, L., & Bursztajn, H. J. (2012). Emerging perspectives on adolescents and young adults with high-functioning autism spectrum disorders, violence, and criminal law. *The Journal of the American Academy of Psychiatry and the Law, 40*, 177–190.
- Loureiro, D., Machado, A., Silva, T., Veigas, T., Ramalheira, C., & Cerejeira, J. (2018). Higher autistic traits among criminals, but no link to psychopathy: Findings from a high-security prison in Portugal. *Journal of Autism and Developmental Disorders, 48*(9), 3010–3020.
- Michna, I., & Trestman, R. (2016). Correctional management and treatment of autism spectrum. *The Journal of the American Academy of Psychiatry and the Law, 44*(2), 253–258.
- Robertson, C. E., & McGillivray, J. A. (2015). Autism behind bars: A review of the research literature and discussion of key issues. *The Journal of Forensic Psychiatry & Psychology, 26*(6), 719–736.
- Robinson, L., Spencer, M. D., Thomson, L. D. G., Stanfield, A. C., Owens, D. G. C., Hall, J., et al. (2012). Evaluation of a screening instrument for autism spectrum disorders in prisoners. *PLoS One, 7*(5), e36078.
- Rogers, J., Viding, E., Blair, R. J., & Frith, U. (2006). Autism spectrum disorder and psychopathy: Shared cognitive underpinnings or double hit? *Psychological Medicine, 36*, 1789–1798.

- Seigfried-Spellar, K. C., O'Quinn, C. L., & Treadway, K. N. (2015). Assessing the relationship between autistic traits and cyberdeviancy in a sample of college students. *Behaviour & Information Technology*, 34(5), 533–542.
- The Offender Health Research Network. (2013). Comprehensive Health Assessment Tool (CHAT): Young People in contact with the Youth Offending Service (YOS). Retrieved from <https://www.gmeccsn.nhs.uk/attachments/article/196/CHAT%20Tool%20YOS.pdf>
- Woodbury-Smith, M., & Dein, K. (2014). Autism spectrum disorder (ASD) and unlawful behavior: Where do we go from here? *Journal of Autism and Developmental Disorders*, 44, 2734–2741.
- Young, S., Moss, D., Sedgwick, O., Fridman, M., & Hodgkins, P. (2015). A meta-analysis of the prevalence of attention deficit hyperactivity disorder in incarcerated populations. *Psychological Medicine*, 45, 247–258.

Autobiographical Memory

► Episodic Memory

Autobiographical Writings

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Definition

Autobiographical writings refer to personal narratives about the self. The autobiographical writings of individuals with autism include life stories, memoirs, letters to correspondents, online blogs and articles, entries in online chat rooms, and accounts of experiences (as told to researchers or clinicians). Notable autobiographical writings in the autism field include those of Temple Grandin, Donna Williams, David Miedzianik, Lianne Holliday Willey, Therese Jolliffe, Wendy Lawson, Daniel Tammet, and Tito Mukhopadhyay. While

some autobiographical writings of individuals with autism were originally intended for publication, others were initially private correspondence that has been made public at a later date. Many autobiographical writings are solely the work of an individual with autism, while others have been edited or cowritten, usually by an individual without autism. Although some autobiographical works explicitly outline the degree of editing and/or cowriting by individuals without autism, this is not always the case. Autobiographical writings have been used as a tool through which one can gain an “inside” view of living with an autism spectrum disorder. Individuals with autism have also used their autobiographical writings for purposes of advocacy, using their personal experiences to enable people to have a better understanding of autism.

Historical Background

Early clinical accounts of autism (e.g., Asperger 1944/1991; Kanner 1943) provided an insight into the world of autism from an observer perspective, making inferences about the individual on the basis of parental report, neuropsychological evaluation, and clinical assessment of behaviors. This was followed by a long tradition of research on individuals with autism that provided further insight into the nature of the condition, often on individuals with low intellectual abilities who may not be able to verbally express their experiences (e.g., Hermelin and O'Connor 1970). It was not until much later that (often, but not exclusively, high-functioning) individuals provided their own personal reports of living with autism. Complementing the earlier clinical accounts and experimental investigations, these autobiographical writings enabled a greater understanding of the experience of living with autism.

Current Knowledge

Theories of autism would predict that individuals with autism would not elect to express their thoughts, feelings, and emotions through

autobiographical writings. As well as displaying impairments in aspects of the self (including self-awareness, self-referential cognition, and introspection), individuals with autism characteristically have written and verbal communication difficulties. Despite this, more than 50 autobiographies and memoirs of individuals with autism have been published, and several more appear online. Autobiographical writings have also appeared in the form of entries in chat rooms and blogs, as well as letters or articles. They are clearly a popular method of expression for individuals with autism.

Autobiographical writings have provided researchers, clinicians, and the general public with comprehensive and insightful accounts of living with autism from a personal perspective. One of the most well-known individuals with autism to have produced autobiographical works is Temple Grandin – a highly successful and articulate professor of animal science, who was diagnosed with autism as a child. These writings include her autobiography *Emergence* (which was edited by a children's writer) in addition to a number of articles that were independently written (e.g., Grandin 1984). Marked differences between her entirely self-produced and her externally edited autobiographical writings have been noted, the former exhibiting a higher number of written characteristics that are typical of an individual with autism. These include unexplained changes in topic and a failure to provide readers with pertinent background information that is necessary to fully understand the text (see Happé 1991, for an analysis of this work). Sacks (1995) referred to Grandin's *Emergence* as "unprecedented," as this was the first "inside narrative" of autism.

Another high-functioning female autobiographer who has been diagnosed with autism is Donna Williams. As well as authoring a number of autobiographies (including *Nobody Nowhere* and *Somebody Somewhere*), Williams shares her autobiographical writings through an online blog, which provides regular autobiographical entries. In recent years, several other high-profile individuals with autism have elected to use online blogging as a means of communicating their

autobiographical writings. These include Daniel Tammet (an adult with autism famed for his remarkable memory abilities), who has also published a memoir of his life, *Born on a Blue Day*. Blogging has become an increasingly popular tool for individuals with autism in recent years and, as they are easily updatable, blogs are a useful and rapid means of communicating the current thoughts of individuals with autism.

Although the published autobiographical writings and blogs of high-functioning adults with autism have provided an insight into the experiences of autism at one extreme of the autism spectrum, autobiographical writings have also stemmed from individuals with autism that have more disabling communication impairments. Tito Mukhopadhyay is a writer and poet who is non-verbal, but communicates through his sophisticated writings. He therefore allows readers a window onto life with autism and severe communication difficulties. He is also one of the few individuals with autism to have produced writings as a child, writing *The Voice of Silence* at the age of 8 and *Beyond the Silence* at the age of 11. Tito's autobiographical writings are largely his own work and, to ensure the integrity of his writings, all changes or additions by editors or cowriters have been carefully and clearly noted in the text.

In between these two extremes of the autism spectrum are individuals such as David Miedzianik and Barry (both of which are documented in Happé 1991), who perhaps represent a more typical existence on the (verbal) autism spectrum. Barry's letters to a correspondent were entirely self-motivated works that received no external editing. They displayed features of autism including social naivety, perseveration on topics, the use of parroted material, and the introduction of topics without providing sufficient background information for readers. In contrast, David Miedzianik's autobiographical writings (despite flitting between one subject to another) demonstrate consideration for the reader's state of knowledge, usually introducing information with an explanation. As with the majority of autobiographical writings in autism, these two cases clearly demonstrate the striking range of abilities (and disabilities) found within the autism spectrum.

Researchers and clinicians have used the autobiographical writings of individuals with autism to gain an insight into the mind of individuals with this disorder. Autobiographical writings can provide readers with personal accounts of the core symptoms of autism (for example, specifying real-world instances of social and communication difficulties, or repetitive behaviors, interests, and activities). In particular, they provide a stark reminder of the different manifestations of the key signs and symptoms of autism in different individuals. This can allow professionals to better understand the specific needs of individuals with autism. The actual writings themselves can also provide an insight into the features of the disorder. For example, the writings of individuals with autism often demonstrate commonly noted characteristics of autism including perseveration on topics of interest, unusual changes in topic, a lack of empathy, and a failure to appreciate the prior knowledge of the reader. Writing about personal thoughts, feelings, and experiences may be a medium of choice for individuals with autism as this removes the need for social interactions and spoken communication. This is especially true of online blogging and chat rooms, which have become a very popular means for sharing autobiographical writings and experiences in recent years. This vehicle of communication has been used to help typical individuals understand more about autism. It can also provide a forum for individuals with autism to discuss their interests and may allow an insight into the strengths and weaknesses of the autism community.

Future Directions

Although researchers have attempted to study, and make inferences from, the autobiographical writings of individuals with autism, there are many difficulties faced when interpreting these writings. First, such an analysis often requires a subjective approach to interpretation. Qualitative researchers must make judgments about the underlying motivations and intentions of individuals with autism, which may be erroneous and lead to both false-

positive and false-negative conclusions (see Happé 1991, for a discussion). Using a more quantitative approach, researchers have used content analytic techniques to examine the frequency of specific terminology or phrases within autobiographical writings (e.g., Crane and Goddard 2008; Crane et al. 2010). This technique may be overly superficial and especially problematic given the language and communication atypicalities noted in individuals with autism. Future research should attempt to merge both qualitative and quantitative approaches to the assessment of autobiographical writings in autism.

A second issue regards how the autobiographical writings of individuals with autism are typically from a very able and high-functioning subgroup of individuals with the disorder. Although some published works are from individuals with severe communication difficulties (e.g., Tito Mukhopadhyay), those who produce autobiographical writings must have a relatively high degree of written language abilities. Not only are the majority of these individuals among the most verbally fluent and intellectually able persons on the autism spectrum, their autobiographical writings have tended to make them celebrities within the autism field. As such, their experiences (especially in their latter years) are perhaps somewhat atypical of the everyday experiences of individuals with autism. Problems therefore arise regarding the generalizability of their writings, and it is important for future research to examine a wide range of autobiographical writings, from individuals across different levels of the autism spectrum. Although researchers have explored online writings of individuals with autism (e.g., Jones et al. 2001), which affords an insight into the experiences of a broader range of individuals with autism, questions concerning authenticity arise.

Third, there is a lack of an appropriate comparison group against which to compare the autobiographical writings of individuals with autism. Most published autobiographical writings are from typical adults, usually those in the public eye with rather unusual lives (e.g., politicians, celebrities) or from professional writers. These do not provide suitable comparisons for the

autobiographical writings of individuals with a neurodevelopmental disorder. Some researchers have compared the autobiographical writings of adults with autism with those of adults with schizophrenia (see Happé 1991, for a comparison of Grandin's autobiographical writing with that of a female with schizophrenia), but these comparisons are limited. The selection of a suitable comparison group is also confounded by the lack of interest in works of fiction that is commonly noted in individuals with autism (Happé 1991). As this group may not read fictional works to the same degree as typical adults, this may influence the content and structure of their autobiographical writings. These difficulties mean that it is problematic to draw inferences on the typicality or atypicality of the autobiographical writings of this group, or to determine how they are similar to or different from the writings of individuals without autism. The selection of a range of appropriate comparison works is crucial for future research in this area.

Another important direction for future research is to establish the degree to which the autobiographical writings of individuals with autism are the work of the individual themselves. Published autobiographical writings (in particular, life stories, memoirs, and autobiographies) are often subject to high levels of editing or rewriting by publishers, editors, or cowriters. Indeed, Temple Grandin's autobiographical work *Emergence* was cowritten with a children's writer who rewrote and formatted sections of the book and structured it to make it easier to read. This significantly limits the conclusions that can be drawn from the autobiographical text itself. Indeed, Grandin's autobiographical writing *My Experiences as an Autistic Child* is markedly different to *Emergence* and displays several characteristics that are typical of an adult on the autism spectrum (e.g., switching between topics, failing to provide the reader with pertinent background knowledge regarding a topic). Analysis of writings that clearly delineate the text composed by individuals with autism and that inserted or changed by editors or cowriters is important for future research, as is the analysis of online writings, which tend to be solely the work of the individual with autism (without subsequent editing).

Future research could also consider gender differences in the autobiographical writings of individuals with autism. Despite a higher number of males than females being diagnosed with autism, it appears that more women with autism express themselves in writing and publish their work. Future work should therefore aim to compare the autobiographical writings of males and females with autism, to ascertain whether there are similarities or differences in the expressions of these writings.

A final point to note regarding the autobiographical writings of individuals with autism concerns their recall of personal experiences. Research on autobiographical memories has shown that memories of personal events are not veridical representations of the past – they are reconstructions of experiences. As such, doubts can be raised concerning the accuracy of the events and experiences referred to in the autobiographical writings of individuals with (and without) autism. This is an important factor to take into account when evaluating, and drawing inferences from, the autobiographical writings of individuals with autism (as well as the writings of typical comparison adults).

See Also

- ▶ [Advocacy](#)
- ▶ [Asperger Syndrome](#)
- ▶ [Autistic Savants](#)
- ▶ [Episodic Memory](#)
- ▶ [Expressive Language](#)
- ▶ [Giftedness](#)
- ▶ [High-Functioning Autism \(HFA\)](#)
- ▶ [Memory](#)
- ▶ [Narrative Assessment](#)
- ▶ [Savant Skills \(in Autism\)](#)

References and Reading

- Asperger, H. (1944/1991). "Autistic psychopathy" in childhood. In U. Frith (Ed.), *Autism and Asperger syndrome* (pp. 37–92). Cambridge: Cambridge University Press.

- Chamak, B., Bonniau, B., Jaunay, E., & Cohen, D. (2008). What can we learn about autism from autistic persons? *Psychotherapy and Psychosomatics*, 77, 271–279.
- Crane, L., & Goddard, L. (2008). Episodic and semantic autobiographical memory in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 38(3), 498–506.
- Crane, L., Goddard, L., & Pring, L. (2010). Self-defining and everyday autobiographical memories in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 40(3), 383–391.
- Grandin, T. (1984). My experiences as an autistic child and review of selected literature. *Journal of Orthomolecular Psychiatry*, 13, 144–175.
- Grandin, T., & Scariano, M. (1986). *Emergence: Labeled autistic*. Novato: Arena Press.
- Hacking, I. (2009). Autistic autobiography. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 364(1522), 1467–1473.
- Happé, F. G. E. (1991). The autobiographical writings of three Asperger syndrome adults: Problems of interpretation and implications for theory. In U. Frith (Ed.), *Autism and Asperger syndrome*. Cambridge, MA: Cambridge University Press.
- Hermelin, B., & O'Connor, M. (1970). *Psychological experiments with autistic children*. Oxford: Pergamon Press.
- Holliday Willey, L. (1999). *Pretending to be normal. Living with Asperger's syndrome*. London: Jessica Kingsley.
- Jolliffe, T., Lansdown, R., & Robinson, C. (1992). Autism: A personal account. *Communication*, 26, 12–19.
- Jones, R. S. P., Zahl, A., & Huws, J. C. (2001). First-hand accounts of emotional experiences in autism: A qualitative analysis. *Disability and Society*, 16, 393–401.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Lawson, W. (1998). *Life behind glass. A personal account of autism spectrum*. London: Kingsley.
- Mukhopadhyay, T. R. (2000). *The mind tree: A miraculous child breaks the silence of autism*. New York: Arcade.
- Sacks, O. (1995). *An anthropologist on mars: Seven paradoxical tales*. New York: Knopf.
- Tammet, D. (2006). *Born on a blue day: A memoir of Aspergers and an extraordinary mind*. London: Hodder & Stoughton.
- Volkmar, F. R., & Cohen, D. J. (1985). The experience of infantile autism: A first-person account by Tony W. *Journal of Autism and Developmental Disorders*, 15, 47–54.
- Williams, D. (1992). *Nobody nowhere: The remarkable autobiography of an autistic girl*. London: Jessica Kingsley.
- Williams, D. (1994). *Somebody somewhere: Breaking free from the world of autism*. London: Jessica Kingsley.

Autonomic Nervous System

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Synonyms

Central nervous system (CNS); Peripheral nervous system (PNS)

Definition

The autonomic nervous system (ANS) coordinates the body's fight or flight and rest and digest response through the central and peripheral nervous systems (Rees 2014). Specifically, the ANS maintains homeostasis and adapts physiological, psychological, and behavioral responses to external stressors (Klusek et al. 2015; Rees 2014). Dysfunction of the ANS increases the risk of numerous psychological disorders including panic disorder, anxiety, post-traumatic stress disorder (PTSD), and schizophrenia (Klusek et al. 2015). Several studies showed Autistic Spectrum Disorder (ASD) children revealed diminished parasympathetic response, autonomic response, and vagal tone (Benevides and Lane 2013; Järvinen et al. 2015; Klusek et al. 2015). Furthermore, ASD children exhibited a variety of sensory deficits affecting their capacity to feel and integrate emotions (Järvinen et al. 2015; Legiša et al. 2012). In addition, ASD children showed hyperarousal and high sensitivity to facial expressions, particularly those with fearful, happy, or no emotion (Järvinen et al. 2015). However, the ANS dysfunctions among ASD patients occurs only during goal-directed activities and disappears under normal physiological conditions (Benevides and Lane 2013). As a result, further investigation is needed to elucidate the coordinated interactions between sympathetic and parasympathetic systems, and the behavioral and physiological differences seen in ASD patients.

See Also

- [Beta-Adrenergic Blockers](#)
- [Erythrocyte Glutathione Peroxidase](#)
- [Inositol: Definition](#)

References and Reading

- Benevides, T. W., & Lane, S. J. (2013). A review of cardiac autonomic measures: Considerations for examination of physiological response in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(2), 560–575. <https://doi.org/10.1007/s10803-013-1971-z>.
- Järvinen, A., Ng, R., Crivelli, D., Neumann, D., Grichanik, M., Arnold, A. J., ... Bellugi, U. (2015). Patterns of sensitivity to emotion in children with Williams syndrome and autism: Relations between autonomic nervous system reactivity and social functioning. *Journal of Autism and Developmental Disorders*, 45(8), 2594–2612. <https://doi.org/10.1007/s10803-015-2429-2>.
- Klusek, J., Roberts, J. E., & Losh, M. (2015). Cardiac autonomic regulation in autism and fragile X syndrome: A review. *Psychological Bulletin*, 141(1), 141–175. <https://doi.org/10.1037/a0038237>.
- Legiša, J., Messinger, D. S., Kermol, E., & Marlier, L. (2012). Emotional responses to odors in children with high-functioning autism: Autonomic arousal, facial behavior and self-report. *Journal of Autism and Developmental Disorders*, 43(4), 869–879. <https://doi.org/10.1007/s10803-012-1629-2>.
- Rees, C. A. (2014). Lost among the trees? The autonomic nervous system and paediatrics. *Archives of Disease in Childhood*, 99(6), 552–562. <https://doi.org/10.1136/archdischild-2012-301863>.

Autonomous Living

- [Independent Living](#)

Autonomy

- [Self-Advocacy](#)

Autosomal Recessive Disorder

- [Gangliosidoses](#)

AUTS18

- [CHD8](#)

Aventyl Hydrochloride

- [Nortriptyline](#)

Aversive/Nonaversive Interventions

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Definition

Aversive and nonaversive interventions refer as much to a dynamic yet functional definition of both terms as to a set of intervention procedures. From a technical point of view, an aversive intervention involves the application of an aversive stimulus. This would include a noxious event that serves as a punisher when it follows behavior, one that evokes a behavior that has terminated the noxious stimulus in past circumstances, or one that functions as a reinforcer when it is removed after the occurrence of a behavior (Cooper et al. 2020). A nonaversive intervention involves the application of positive reinforcement and/or extinction contingencies as a consequence to a behavior or alteration of the intensity, duration, or magnitude of a behavior contingent upon the removal or presentation of an antecedent stimulus.

Historical Background

While treatment in autism has, over the years, had many controversies, perhaps none has been so heated as the discussion of the viability and

appropriateness of aversive and nonaversive procedures to treat a variety of problems common to the disorder (and to those with other neurodevelopmental disorders as well). These controversies have pitted, in somewhat of a dichotomous fashion, empirical science against social validity. The result was at once unfortunate and the stimulus for a paradigmatic shift. When in its relative infancy, the science of the experimental analysis of behavior served a very important function: to prove that even the most recalcitrant of human behaviors are subject to the laws of learning and can be improved upon. For generations of clinicians raised on the belief that change was only possible in small increments for those severely affected by autism and then only through rather drastic psychopharmacologic interventions, the opportunity to demonstrate progress in reducing self-injury, aggression, and other destructive behaviors as well as to increase prosocial, adaptive behavior was a breakthrough. Applications of more basic operant conditioning principles such as positive and negative reinforcement, extinction, and punishment were tactics of choice during this period. Indeed, clinical significance was often defined only in terms of the magnitude of behavior reduction (the end product) but rarely so by the means of reduction.

As the 1960s progressed through to the late 1970s, however, means of intervention appropriately became a more prominent consideration. The seminal work on social validity by Kazdin (1977) and Wolf (1978) reshaped the narrative around three key points: not only must the outcome of intervention be socially valid but also the target of intervention (behavior to be changed) and also the means to achieve that outcome. As a construct, social validity imposes the requirement that all factors be considered before, during, and after treatment. This demand served several important functions. It posed the important question, "socially valid for whom?" Were targets, procedures, and outcomes socially valid for the client, the family, and institutionally based caregivers? Social validity also raised the question of relativity. At different points in time, for different clients, and under particular circumstances, a treatment procedure or outcome might

or might not be acceptable. But very importantly, at its base, the question of social validity also raised the issue of the generalizability of behavior change. While behavior analysis had evolved very good technologies of generalization and maintenance (Horner et al. 1988), things did not always work out as planned. So-called treatment failures continued to occur, often under the contingencies of more remote or diverse (and sometimes less well-understood) events. By imposing the demand to assess for social validity, interventionists had a tool to begin to predict potential functional relationships between change agents and the consumers of change and to begin to modify those contingencies that might interfere with long-term maintenance and generalization.

Within the span of a few years, however, a number of flash point events occurred that sharpened the issues concerning treatment of those whose autism placed themselves, and others, at the greatest risk. Highly publicized reports of the deaths of clients in the care of otherwise well-known residential programs following the use of contingent aversive procedures (e.g., white noise) changed the conversation from one of science alone to a discussion of human dignity and the right to effective treatment. Suffice it to say that while at times mean-spirited, personal, and derogatory, the power of the objectivity of science won out. Indeed, not only did the National Institutes of Health fund a number of collaborative research centers with the mandate to investigate and develop effective interventions that were non-aversive, but the NIH later convened a consensus conference (National Institutes of Health 1991) in order to issue guidelines for the use of behavior reduction procedures (including punishment strategies) when treating destructive behavior in those with developmental disabilities. The efforts of established collaborative research centers, other scientists working in basic and applied settings, and the general understanding of the effects (and negative effects) of punishment have led over the past 20 years to a highly developed, evolving, evidence-based technology of behavior change based upon the use of antecedent and consequent control procedures that do not involve the use of aversive stimuli. To be certain, the controversy

has not ended entirely, as those who empirically demonstrate the effective use of punishment procedures *as a component of a comprehensive treatment package* would argue (Axelrod 1990). But, as importantly, the exceptional science being developed to understand the often complex functional motivators behind severe behavior continues as well and is especially visible in the efforts of those promoting positive behavior support initiatives in public schools.

Rationale or Underlying Theory

Given the extensive research base for both aversive and nonaversive interventions, it is reasonable to conclude that considerations about each are evidence-based. The important considerations, however, lie in the issue of negative effects and generalizability of effects. Both sets of procedures are based on the principles of operant conditioning earlier described by Skinner, with many decades of subsequent and substantive empirical extensions of that work. What has evolved over the years is a toolbox of intervention strategies, many working best as part of multi-component procedures. While there may well be occasions for which a punishment procedure – in combination with positive reinforcement procedures designed to increase functionally equivalent, alternative prosocial behavior – is the least restrictive intervention option, intervention based on punishment alone is rarely advised.

Nonaversive interventions are broadly organized around antecedent strategies (those that occur before the problematic behavior is emitted), with the intention of altering the stimulus control and reinforcing value of the existing antecedent “triggers” for the behavior. Consequent procedures are those delivered after behavior has been demonstrated. They can include reinforcement-based procedures, extinction, and variants of interruption and redirection. In contrast, aversive interventions involve the application of an aversive or unpleasant stimulus immediately following the problem behavior, designed to discourage future occurrence of the behavior. In all cases, however, whether an intervention is aversive or

reinforcing to a client is a functional question. If the application of a stimulus immediately following demonstration of a specific behavior increases the probability of that behavior occurring, the stimulus was reinforcing. If presentation of the stimulus immediately following the behavior reduces the likelihood of behavior reoccurrence, then the stimulus was aversive. Referring back to the discussion of social validity earlier, what is aversive to one person may be reinforcing to another. The only solution is to assess functionally before and during treatment implementation.

Ultimately, the rationale about which intervention strategies to employ in a particular case is a functional one, clarified by a thorough functional behavior assessment/analysis and subjected to rigorous outcome evaluation. In the final analysis, intervention must be effective, that is, it must be successful in its outcome and have minimal or no negative effects associated with it. Treatment strategies that are socially valid and empirically based will best serve the interests of persons with autism and related neurodevelopmental disorders.

Goals and Objectives

The selection of intervention strategies is based on behavioral function, not form. Function can be described in several ways. For example, behavior can serve to access positive reinforcement in the form of social attention or access to preferred materials. The behavior can be functionally reinforced by its ability to terminate an aversive or unpleasant event (negative reinforcement). These functions can be observed in the presence of others or when the client is alone. In this latter case, we suggest that the behavior can be maintained by the positive or negative reinforcing contingencies of sensory stimuli impinging on the client. In all cases, the stated goal of intervention should be to improve the behavior of the person with autism by teaching appropriate replacement skills while simultaneously reducing or eliminating the behavior that is problematic or that interferes with more adaptive functioning. Specific procedures to accomplish this are discussed below.

Treatment Participants

Treatment procedures for any given client are selected based upon the results of the functional assessment/analysis but may be modified to address the specific target behaviors selected, the learning history (history of reinforcement) of the client with the particular behavior, and the availability of resources and competencies of intervenors. Consideration is also given to such factors as severity, duration, pervasiveness, and frequency of the target behavior when determining priorities for intervention.

Treatment Procedures

Treatment procedures for nonaversive interventions can be broadly divided into two groups: antecedent interventions that occur prior to the behavior and consequent procedures that are implemented after the behavior has been emitted. Both seek to reduce the likelihood of behavioral expression in the future by emphasizing the use of positive reinforcement procedures as a key or collateral component of the treatment package. Most importantly, all treatment should be preceded by a thorough functional behavior assessment or analysis in order to determine which stimuli in the environment exert control over the target behavior.

Antecedent procedures include errorless learning, whereby the student is prompted to the correct response immediately after the presentation of the request; interspersing mastered or easy tasks with difficult tasks in teaching (Weber and Thorpe 1992); the use of choice in the selection of tasks and reinforcers (Dyer et al. 1990); reducing the information-processing demands of the task or providing an alternative mode of task presentation; use of a high-probability request sequence (Zuluaga and Normand 2008); functional communication training (Carr and Durand 1985); stimulus change procedures, whereby a novel stimulus that is not an antecedent or a consequence to the behavior is interjected into a behavioral sequence, interrupting the response-reinforcer relationship (Carr et al. 1990); and environmental modifications such as use of visual schedules, curriculum

adjustment, etc. (Flannery and Horner 1994; Kern and Dunlap 1998).

Consequent procedures with demonstrated efficacy include positive reinforcement, differential reinforcement, and its variants (differential reinforcement of other, incompatible, high rates or alternative behavior); response interruption and redirection (Underwood et al. 1989); extinction (Lerman and Iwata 1996); and noncontingent reinforcement, whereby reinforcing stimuli are provided to a client independent of the client's behavior (Carr et al. 2009).

Aversive stimuli are noxious events that serve as punishers when following a behavior, evoke a behavior that has terminated the noxious stimulus in past circumstances, or function as a reinforcer when removed after the occurrence of a behavior (Cooper et al. 2020). While the function of an aversive stimulus is always to cause the cessation of a behavior, its forms are virtually limitless (Repp and Singh 1990) and have included smelling aromatic ammonia, contingent water mist to the face, the application of "white noise," and electric shock. It is noteworthy that while the NIH consensus conference clearly emphasized the importance of using treatment procedures based on positive behavioral supports, it also provided clear guidelines for the use of punishment procedures when they might be deemed clinically necessary.

Efficacy Information

The efficacy of antecedent strategies to treat behavior problems has been well documented in the research literature, and several in particular have been identified as evidence-based procedures (Cooper et al. 2020; Powers et al. 2011). It is important to remember, however, that the use of an antecedent (or any other) strategy does not guarantee success. Rather, the use of the procedure must be based on the results of the functional behavior assessment/functional analysis, must be implemented with fidelity, and must be evaluated accurately and objectively. Violation of any of these tenets can (and likely will) reduce the efficacy and efficiency of the correctly chosen treatment strategy.

Outcome Measurement

Objective and reliable measurement of treatment effects and outcomes is essential to the correct use of any procedure designed to increase desirable behavior or to reduce problem behavior. Fortunately, the use of single-subject experimental designs (SSEDs) has predominated in the literature (Kazdin 1982), establishing a robust arsenal of potential designs for outcome measurement. When well used, SSEDs provide excellent internal and external validity, support the development of reliable observations, and ultimately contribute to the serial replication of findings. To this latter point, the aggregation of large numbers of individual studies, each with a small subject pool, can generate strong findings of efficacy (Reichow et al. 2011).

Qualifications of Treatment Providers

While certainly effective when used correctly, the technology of intervention requires training in the principles and strategies of applied behavior analysis. Obviously, with behavior problems of greater significance (e.g., where personal safety of the client or others is at risk and where health status can/may be compromised), the demand for greater levels of sophistication and competency is critical. At a minimum, supervision of assessment and treatment protocols by an individual with Board Certification as a Behavior Analyst (BCBA) or by a clinician with equivalent training and experience would be appropriate. In cases where more extraordinary interventions are necessary or where the risk of harm is greater, it is strongly advisable to have all clinical aspects peer reviewed and vetted by a human rights committee.

See Also

- ▶ Board Certified Associate Behavior Analyst
- ▶ Differential Reinforcement
- ▶ High-Probability Requests

References and Reading

- Axelrod, S. A. (1990). Myths that (mis)guide our profession. In A. C. Repp & N. N. Singh (Eds.), *Perspectives on the use of nonaversive and aversive interventions for persons with developmental disabilities* (pp. 59–72). Sycamore: Sycamore Press.
- Carr, E. G., & Durand, V. M. (1985). Reducing behavior problems through functional communication training. *Journal of Applied Behavior Analysis*, 18, 111–126.
- Carr, E. G., Robinson, S., & Palumbo, L. W. (1990). The wrong issue: Aversive versus nonaversive treatment. The right issue: Functional versus nonfunctional treatment. In A. C. Repp & N. N. Singh (Eds.), *Perspectives on the use of aversive and nonaversive interventions for persons with developmental disabilities* (pp. 361–379). Sycamore: Sycamore Press.
- Carr, J. E., Severtson, J. M., & Lepper, T. L. (2009). Noncontingent reinforcement is an empirically supported treatment for problem behavior exhibited by individuals with developmental disabilities. *Research in Developmental Disabilities*, 30, 44–57.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2020). *Applied behavior analysis* (3rd ed.). Hoboken: Pearson Education.
- Dyer, K., Dunlap, G., & Winterling, V. (1990). Effects of choice-making on the serious problem behaviors of students with severe handicaps. *Journal of Applied Behavior Analysis*, 23, 515–524.
- Flannery, K. B., & Horner, R. H. (1994). The relationship between predictability and problem behavior for students with severe disabilities. *Journal of Behavioural Education*, 4(2), 157–176.
- Horner, R. H., Dunlap, G., & Koegel, R. L. (1988). *Generalization and maintenance: Lifestyle changes in applied settings*. Baltimore: Paul H. Brookes.
- Kazdin, A. E. (1977). Assessing the clinical or applied importance of behavior change through social validation. *Behavior Modification*, 1, 427–451.
- Kazdin, A. E. (1982). *Single-case research designs: Methods for clinical and applied settings*. New York: Oxford University Press.
- Kern, L., & Dunlap, G. (1998). Curricular modifications to promote desirable classroom behavior. In J. K. Luiselli & M. J. Cameron (Eds.), *Antecedent control: Innovative approaches to behavioral support* (pp. 289–307). Baltimore: Paul H. Brookes.
- LaVigna, G. W., & Donnell, A. M. (1986). *Alternatives to punishment: Solving behavior problems with nonaversive strategies*. New York: Irvington.
- Lerman, D. C., & Iwata, B. A. (1996). Developing a technology for the use of operant extinction in clinical settings: An examination of basic and applied research. *Journal of Applied Behavior Analysis*, 29, 345–382.
- National Institutes of Health. (1991). *Treatment of destructive behaviors in persons with developmental disabilities. NIH consensus development conference*. Washington, DC: United States Department of Health and Human Services.

- Powers, M. D., Palmieri, M. J., D'Eramo, K. S., & Powers, K. M. (2011). Evidence-based treatment of behavioral excesses and deficits for individuals with autism spectrum disorders. In B. Reichow, P. Doehring, D. V. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-based practices and treatments for children with autism*. New York: Springer.
- Reichow, B., Doehring, P., Cicchetti, D. V., & Volkmar, F. R. (Eds.). (2011). *Evidence-based practices and treatments for children with autism*. New York: Springer.
- Repp, A. C., & Singh, N. N. (Eds.). (1990). *Perspectives on the use of nonaversive and aversive interventions for persons with developmental disabilities*. Sycamore: Sycamore Press.
- Underwood, L. A., Figueroa, R. G., Thyer, B. A., & Nzeocha, A. (1989). Interruption and DRI in the treatment of self-injurious behavior among mentally retarded and autistic self-restrainers. *Behavior Modification*, 13, 471–481.
- Weber, R. C., & Thorpe, J. (1992). Teaching children with autism through task variation. *Exceptional Children*, 59, 77–86.
- Wolf, M. M. (1978). Social validity: The case for subjective measurement of how applied behavior analysis is finding its heart. *Journal of Applied Behavior Analysis*, 11, 203–214.
- Zuluaga, C. A., & Normand, M. P. (2008). An evaluation of the high-probability instruction sequence with and without programmed reinforcement for compliance with high probability instructions. *Journal of Applied Behavior Analysis*, 27, 649–658.

AVLT

► [Rey Auditory Verbal Learning Test \(Rey AVLT\)](#)

Avoidant Personality Disorder

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Synonyms

[Anxious Personality Disorder](#)

Short Description or Definition

In the most recent *DSM* revision, the *DSM, Fifth Edition (DSM-5)*; American Psychiatric

Association [APA] 2013), AVPD is classified as a personality disorder and is described as “a pervasive pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation that begins by early adulthood and is present in a variety of contexts” (p. 673). As for all personality disorders, “this pattern of inner experience and behavior... deviates markedly from the expectations of the individual’s culture, is pervasive and inflexible,... is stable over time, and leads to distress or impairment” (p. 645).

Categorization

As indicated above, AVPD is classified within the Personality Disorders section in *DSM-5*. Based largely on an earlier, theoretically derived construct (Millon 1981), AVPD first appeared as a diagnostic entity in *DSM, Third Edition (DSM-III*; APA 1980). This category grew from a trifurcation of the *DSM, Second Edition (DSM-II*; APA 1968) diagnosis, schizoid personality – which described individuals with “shyness, oversensitivity, seclusiveness, avoidance of close or competitive relationships, and often eccentricity” (p. 42). The broader *DSM-II* schizoid personality construct was, in *DSM-III*, subdivided into a more narrowly defined schizoid personality disorder, as well as schizotypal and avoidant personality disorders. Schizotypal personality disorder was thought to describe those individuals who had previously been diagnosed with borderline schizophrenia and encompassed the eccentricity noted in the *DSM-II* description. The distinction between *DSM-III* avoidant and schizoid personality disorders was construed as centering on whether or not the individual had the motivation and capacity for emotional involvement with others (APA 1980; Millon 1981).

Beginning with *DSM-III*, personality disorders were placed on axis II within a recommended “multiaxial” approach to psychiatric diagnosis; axis II encompassed specific developmental disorders as well as personality disorders and was felt to be useful in ensuring that “consideration is given to the possible presence of disorders that are frequently overlooked when attention is directly toward the usually more florid Axis

I disorder” (p. 23). Beginning with *DSM, Third Edition, Revised (DSM-III-R; APA 1987)*, AVPD was placed in the cluster C subcategory of personality disorders, which are characterized by “anxious or fearful” (p. 337) clinical presentations. *DSM-III-R* aligned AVPD with the clinical concept of “phobic character” (p. 429) and no longer suggested that it needed to be mutually exclusive with schizoid personality disorder. In *DSM, Fourth Edition (DSM-IV; APA 1994)*, AVPD remained in cluster C, along with dependent and obsessive-compulsive personality disorders, as it does now in *DSM-5*. It is worth noting, however, that the multiaxial system has been eliminated in the current diagnostic manual – and personality disorders are now classified alongside all other relevant diagnoses.

Although initially formulated in *DSM-III* as a monothetic criterion set – requiring, for the diagnosis, all five possible symptom criteria – subsequent revisions have constructed AVPD as a polythetic set, requiring any four of seven possible criteria. Each successive revision – from *DSM-III* to *DSM-III-R*, and from *DSM-III-R* to *DSM-IV* – involved adding, deleting, and rewording various criteria. These changes have been based, in part, on empirical evidence (Baillie and Lampe 1998; Becker et al. 2009; Grilo 2004; Hummelen et al. 2006). No further changes were made to the AVPD criteria in the transition from *DSM-IV* to *DSM-5*.

Epidemiology

Investigations in clinical samples have shown AVPD to be among the most frequently diagnosed personality disorders (Alnæs and Torgersen 1988; Stuart et al. 1998). Although *DSM-III* and *DSM-III-R* had indicated only that AVPD is “apparently common” (APA 1980, p. 323, 1987, p. 352) in the general population, and *DSM-IV* stated that the general prevalence of this disorder is between 0.5% and 1.0% (APA 1994), *DSM-5* cited a prevalence of 2.4% (APA 2013). However, two large, community-based studies – using *DSM-III-R* (Torgersen et al. 2001) and *DSM-IV* (Ekselius et al. 2001) criteria – both yielded much higher rates of 5.0% and 6.6%, respectively. In the

former study, AVPD was more prevalent than any other personality disorder; in the latter study, it was the second most prevalent among these disorders. Ekselius et al. (2001) observed generally that individuals with personality disorders more often were younger, were students or unemployed, received psychiatric treatment, and lacked social supports.

Natural History, Prognostic Factors, and Outcomes

Unfortunately, relatively few studies have directly examined AVPD (Alden et al. 2002). Instead, most have considered AVPD along with other personality disorders – in the service of understanding personality pathology more broadly or within the context of studying the effects of comorbid AVPD on axis I psychiatric disorders. As a result, relatively little is known about the natural history and progression of AVPD. *DSM-5* (APA 2013) notes that avoidance often begins in childhood with shyness – but that, while shyness in most individuals dissipates with age, those who progress to develop AVPD will often become increasingly shy and avoidant during adolescence and young adulthood. Evaluation of the childhood antecedents of AVPD has shown that adults with AVPD – in relation to relevant clinical comparison groups – report poorer athletic performance during childhood and adolescence, less involvement in hobby activities during adolescence, and diminished adolescent popularity (Rettew et al. 2003).

Personality disorder stability has been shown, in general, to be modest; for AVPD, 2-year remission rates as high as 50% have been reported by the Collaborative Longitudinal Personality Disorders Study (Grilo et al. 2004). These investigators have also suggested that personality disorders are hybrids of traits and symptomatic behaviors, with the former being more stable. The interaction of these elements over time helps to determine diagnostic stability. For AVPD, the trait-like criteria – which are the most prevalent and stable – include regarding oneself as socially inept, feeling inadequate compared to others, and wanting evidence of being liked before making social contact

(McGlashan et al. 2005). These observations suggest that the course, persistence, and severity of AVPD – as for all personality disorders – depend upon an interaction of personality traits and the individual's behavioral adaptations to these traits (Lilienfeld 2005). The functional consequences of AVPD are generally significant – having a more profound effect on psychosocial adaptation than, for instance, major depression (Skodol et al. 2002).

Clinical Expression and Psychopathology

In a seminal description of the AVPD construct, Millon (1981) describes four levels of clinical data that may help in the diagnosis: (1) behavioral features (e.g., shyness or timidity, apprehensiveness or guardedness, touchiness, evasiveness, restraint of emotional expression, and physical underactivity with periodic bursts of fidgeting); (2) self-descriptions or complaints (e.g., feeling anxious or ill-at-ease, viewing others as critical or humiliating, and uncertainty about one's self-worth); (3) interpersonal coping style (e.g., anticipation of censure and derision, minimizing involvements that might reactivate or duplicate past humiliations, and diminishing the importance of interpersonal relationships); and (4) inferred intrapsychic dynamics (e.g., conflict between mistrust and the desire for affection, tension between derogation by others and self-deprecation, and tension between the surrounding distress and the emptiness within).

As noted above, *DSM-5* (APA 2013) requires four of seven possible diagnostic criteria:

- Avoids occupational activities that involve significant interpersonal contact, because of fears of criticism, disapproval, or rejection.
- Is unwilling to get involved with people unless certain of being liked.
- Shows restraint within intimate relationships because of the fear of being shamed or ridiculed.
- Is preoccupied with being criticized or rejected in social situations.
- Is inhibited in new interpersonal situations because of feelings of inadequacy.
- Views self as socially inept, personally unappealing, or inferior to others.
- Is unusually reluctant to take personal risks or to engage in any new activities because they may prove embarrassing.

Given the polythetic nature of this and other *DSM-5* personality disorder constructs, psychometric studies – especially those demonstrating a simple factor structure and good internal consistency – have played a key role in establishing construct validity of AVPD. Overall, such studies have demonstrated high internal consistency and a unidimensional structure for the AVPD criterion set adopted in *DSM-IV* and maintained in *DSM-5* (Becker et al. 2009; Grilo 2004; Hummelen et al. 2006).

Evaluation and Differential Diagnosis

Although few data exist regarding the diagnostic process as it relates to AVPD, some evidence has been offered with regard to other personality disorders (Zimmerman and Mattia 1999) or to personality disorders more generally (Zimmerman 1994). Such disorders tend to be diagnosed relatively infrequently within the clinical interview process as compared to when semistructured diagnostic interviews are utilized (Zimmerman and Mattia 1999). This may be due to a general inattention to personality disorder in many clinical settings – or, perhaps, to the polythetic nature of these diagnoses. Although it is therefore preferable that a semistructured diagnostic interview be used in evaluating patients for personality disorders, there is considerable variability among such instruments. Another concern about the assessment process is that the diagnosis of personality disorders is likely to be biased by the patient's acute clinical state (Zimmerman 1994).

With regard to differential diagnosis, consideration should be given especially to social anxiety disorder (social phobia), which is classified as an anxiety disorder in *DSM-5*. Its essential feature is

“a marked... fear or anxiety of social situations in which the individual may be scrutinized by others” (APA 2013, p. 203); social anxiety disorder is therefore phenomenologically similar to AVPD. Indeed, genetic studies have suggested that there is a common genetic vulnerability underlying both disorders (Reichborn-Kjennerud et al. 2010).

Consideration should be given as well to agoraphobia, which is another *DSM-5* (APA 2013) anxiety disorder characterized by avoidance. Finally, with regard to differential diagnosis, some other personality disorders should be considered. These include the other cluster C disorders, characterized by anxiety and fearfulness – especially dependent personality disorder, which can similarly be marked by feelings of inadequacy, sensitivity to criticism, and need for reassurance – as well as the phenomenologically reminiscent, but somewhat more disabling, cluster A conditions: schizoid, schizotypal, and paranoid personality disorders (APA 2013).

Treatment

Studies have shown that psychotherapeutic intervention is the treatment of choice for personality disorders in general – and that this conclusion holds specifically, as well, for AVPD (Verheul and Herbrink 2007). In particular, psychodynamic and cognitive-behavioral therapies have proven effective – especially as individual outpatient modalities but also in group settings and within structured treatment contexts. There is less evidence in support of pharmacotherapeutic intervention, although some have suggested treatment with antidepressant medications – such as selective serotonin reuptake inhibitors – based, in part, on the potential relationship between AVPD and social anxiety disorder (Deltito and Stam 1989; Kapfhammer and Hippus 1998; Ripoll et al. 2011).

See Also

- ▶ Anxiety
- ▶ DSM-5
- ▶ DSM-III

- ▶ DSM-III-R
- ▶ DSM-IV
- ▶ Factor Analysis
- ▶ ICD 10 Research Diagnostic Guidelines
- ▶ Personality Disorders
- ▶ Prevalence
- ▶ Psychotic Disorder
- ▶ Temperament
- ▶ Validity

References and Reading

- Alden, L. E., Laposa, J. M., Taylor, C. T., & Ryder, A. G. (2002). Avoidant personality disorder: Current status and future directions. *Journal of Personality Disorders*, 16, 1–29.
- Alnæs, R., & Torgersen, S. (1988). DSM-III symptom disorders (axis I) and personality disorders (axis II) in an outpatient population. *Acta Psychiatrica Scandinavica*, 78, 348–355.
- American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (Rev. 3rd ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association.
- Baillie, A. J., & Lampe, L. A. (1998). Avoidant personality disorder: Empirical support for DSM-IV revisions. *Journal of Personality Disorders*, 12, 23–30.
- Becker, D. F., Añez, L. M., Paris, M., Bedregal, L., & Grilo, C. M. (2009). Factor structure and diagnostic efficiency of the diagnostic and statistical manual of mental disorders, fourth edition, criteria for avoidant personality disorder in Hispanic men and women with substance use disorders. *Comprehensive Psychiatry*, 50, 463–470.
- Deltito, J. A., & Stam, M. (1989). Psychopharmacologic treatment of avoidant personality disorder. *Comprehensive Psychiatry*, 30, 498–504.
- Ekselius, L., Tillfors, M., Furmark, T., & Fredrikson, M. (2001). Personality disorders in the general population: DSM-IV and ICD-10 defined prevalence as related to sociodemographic profile. *Personality and Individual Differences*, 30, 311–320.
- Grilo, C. M. (2004). Factorial structure and diagnostic efficiency of DSM-IV criteria for avoidant personality

- disorder in patients with binge eating disorder. *Behaviour Research and Therapy*, 42, 1149–1162.
- Grilo, C. M., Sanislow, C. A., Gunderson, J. G., Pagano, M. E., Yen, S., Zanarini, M. C., et al. (2004). Two-year stability and change of schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders. *Journal of Consulting and Clinical Psychology*, 72, 767–775.
- Hummelen, B., Wilberg, T., Pedersen, G., & Karterud, S. (2006). An investigation of the validity of the diagnostic and statistical manual of mental disorders, fourth edition avoidant personality disorder construct as a prototype category and the psychometric properties of the diagnostic criteria. *Comprehensive Psychiatry*, 47, 376–383.
- Kapfhammer, H. P., & Hipplius, H. (1998). Pharmacotherapy in personality disorders. *Journal of Personality Disorders*, 12, 277–288.
- Lilienfeld, S. O. (2005). Longitudinal studies of personality disorders: Four lessons from personality psychology. *Journal of Personality Disorders*, 19, 547–556.
- McGlashan, T. H., Grilo, C. M., Sanislow, C. A., Ralevski, E., Morey, L. C., Gunderson, J. G., et al. (2005). Two-year prevalence and stability of individual DSM-IV criteria for schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders: Toward a hybrid model of axis II disorders. *The American Journal of Psychiatry*, 162, 883–889.
- Millon, T. (1981). *Disorders of personality: DSM-III, axis II*. New York: Wiley.
- Reichborn-Kjennerud, T., Czajkowski, N., Torgersen, S., Neale, M. C., Ørstavik, R. E., Tambs, K., et al. (2010). The relationship between avoidant personality disorder and social phobia: A population-based twin study. *The American Journal of Psychiatry*, 164, 1722–1728.
- Rettew, D. C., Zanarini, M. C., Yen, S., Grilo, C. M., Skodol, A. E., Shea, M. T., et al. (2003). Childhood antecedents of avoidant personality disorder: A retrospective study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 42, 1122–1130.
- Ripoll, L. H., Triebwasser, J., & Siever, L. J. (2011). Evidence-based pharmacotherapy for personality disorders. *The International Journal of Neuropsychopharmacology*, 14(9), 1257–1288 (available online Feb. 15, 2011).
- Skodol, A. E., Gunderson, J. G., McGlashan, T. H., Dyck, I. R., Stout, R. L., Bender, D. S., et al. (2002). Functional impairment in patients with schizotypal, borderline, avoidant, or obsessive-compulsive personality disorder. *The American Journal of Psychiatry*, 159, 276–283.
- Stuart, S., Pfohl, B., Battaglia, M., Bellodi, L., Grove, W., & Cadoret, R. (1998). The cooccurrence of DSM-III-R personality disorders. *Journal of Personality Disorders*, 12, 302–315.
- Torgersen, S., Kringlen, E., & Cramer, V. (2001). The prevalence of personality disorders in a community sample. *Archives of General Psychiatry*, 58, 590–596.
- Verheul, R., & Herbrink, M. (2007). The efficacy of various modalities of psychotherapy for personality disorders: A systematic review of the evidence and clinical recommendations. *International Review of Psychiatry*, 19, 25–38.
- World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organization.
- Zimmerman, M. (1994). Diagnosing personality disorders: A review of issues and research methods. *Archives of General Psychiatry*, 51, 225–245.
- Zimmerman, M., & Mattia, J. I. (1999). Differences between clinical and research practices in diagnosing borderline personality disorder. *The American Journal of Psychiatry*, 156, 1570–1574.

AXCAM

► CNTN4: Contactin 4

Ayres, A. Jean

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Department of Occupational Therapy, San José
State University, San José, CA, USA

Name and Degrees

A. Jean Ayres, PhD, OTR, FAOTA.

Graduated with a BA in Occupational Therapy from University of Southern California in 1945.

Graduated with an MA in Occupational Therapy from University of Southern California in 1954.

Graduated with a PhD in Educational Psychology from University of Southern California in 1961.

Major Appointments (Institution, Location, Dates)

Faculty member in the Department of Occupational Therapy at the University of Southern California (USC) from 1955 to 1964.

Professor in the Department of Special Education at the USC from 1966 to 1977.

Adjunct faculty member in the Department of Occupational Therapy at USC from 1976 to 1984 while running her clinic devoted to serving children with sensory integrative disorders.

Major Honors and Awards

Awarded Fellow of the American Occupational Therapy Association (FAOTA).

Awarded the Eleanor Clark Slagle lectureship in 1963.

Received the highest honor from the American Occupational Therapy Association in 1965, the Award of Merit.

Named as one of the Outstanding Educators of America in 1971.

Charter member of the American Occupational Therapy Association Academy of Research.

Honored by the American Occupational Therapy Association in 1988 with the initiation of the award entitled the A. Jean Ayres Award for Theory Development and Application.

Landmark Clinical, Scientific, and Professional Contributions

Dr. A. Jean Ayres originated the Ayres Sensory Integration theory. She developed the theory into principles of intervention and assessment instruments including the Southern California Sensory Integration Tests (SCSIT) and then revised this instrument as the Sensory Integration and Praxis Tests (SIPT). As an occupational therapist, she introduced the profession to this client-centered, neuroscience-based theory and practice approach to support children with sensory integration disorders/sensory processing disorders.

Short Biography

Biography: A. Jean Ayres

Dr. A. Jean Ayres was born in 1920 in Visalia, CA, and reportedly had challenges learning as a young child, particularly processing various types of

sensory information. She attended the University of Southern California and successfully completed her BA in Occupational Therapy in 1945, her MA in Occupational Therapy in 1954, and her PhD in Educational Psychology in 1961. She completed her postdoctoral training at University of California, Los Angeles (UCLA), Brain Research Institute working with the leading neurophysiologists at that time. Her clinical skills in occupational therapy, with a foundation in the engagement in purposeful activity, and her neuroscience training provided her with the unique perspective to understand how the nervous system can influence functional behaviors.

Dr. Ayres had a long history in academia and was a faculty member in the Department of Occupational Therapy at the University of Southern California (USC) from 1955 to 1964. She then was a professor in the Department of Special Education at the USC from 1966 to 1977. She returned as an adjunct faculty member in the Department of Occupational Therapy at USC from 1976 to 1984 while running her clinic devoted to serving children with sensory integrative disorders.

Dr. Ayres' work as an occupational therapist with children who had learning disabilities and sensory processing challenges served as the impetus for her conceptualization of sensory integrative dysfunctions. She encountered individuals who would complain of how painful it was to have their hair brushed or to wear specific fabrics. This furthered her research endeavors in the area of sensory integration dysfunction and theory development. Her development of the theory of sensory integration expanded, and her numerous publications, books, and approximately 50 scholarly articles provided further evidence of this phenomenon. As a clinician, researcher, and academic, Dr. Ayres recognized the need to establish a mechanism to identify sensory integrative dysfunction and link theory to practice. She developed the Southern California Sensory Integration Tests (SCSIT) in 1972 with intensive training courses on theory, test administration, and interpretation seminars. As the research and theory developed further, Dr. Ayres revised the assessment tool and the Sensory Integration and Praxis Test was published in 1989.

As an occupational therapist, Dr. Ayres sought to support children and provide intervention directed not only to fostering improved functional skills but to develop an explanation regarding the challenges faced by children with sensory integrative disorders. Her scholarship, clinical expertise, and dedication were recognized in several arenas. She was awarded the prestigious Eleanor Clark Sagle lectureship in 1963 by the American Occupational Therapy Association. In her address, she described the theory and practice of sensory integration and how this unique perspective supports participation in everyday tasks. Her substantial contributions to advance the profession of occupational therapy were further recognized when she received the Award of Merit in 1965. This is the highest honor awarded by the American Occupational Therapy Association. In 1971, Dr. A. Jean Ayres was named as one of the Outstanding Educators of America. Dr. Ayres was a charter member of the Academy of Research of the American Foundation of Occupational Therapy, and in 1988, the A. Jean Ayres Award for Theory Development and Application was established in her honor by the American Foundation of Occupational Therapy.

Dr. A. Jean Ayres married Franklin Baker in 1969. She died on December 16, 1988, from complications of cancer. Franklin Baker died on September 2, 1989.

See Also

- [Occupational Therapy \(OT\)](#)
- [Sensory Integration and Praxis Test](#)

References and Reading

Selected Articles by A. Jean Ayres

- Ayres, A. J. (1949). An analysis of crafts in the treatment of electroshock patients. *American Journal of Occupational Therapy*, 3, 195–198.
- Ayres, A. J. (1954). Ontogenetic principles in the development of arm and hand functions. *American Journal of Occupational Therapy*, 8, 95–99.
- Ayres, A. J. (1955a). Proprioceptive facilitation elicited through the upper extremities. Part 3: Specific application to occupational therapy. *American Journal of Occupational Therapy*, 9, 121–126.
- Ayres, A. J. (1955b). Proprioceptive facilitation elicited through the upper extremities. Part 2: Application. *American Journal of Occupational Therapy*, 9, 57–58.
- Ayres, A. J. (1955c). Proprioceptive facilitation elicited through the upper extremities. Part 1: Background. *American Journal of Occupational Therapy*, 9, 1–9.
- Ayres, A. J. (1958a). Basics concepts of clinical practice in physical disabilities. *American Journal of Occupational Therapy*, 12, 300–302.
- Ayres, A. J. (1958b). The visual-motor function. *American Journal of Occupational Therapy*, 12, 130–138.
- Ayres, A. J. (1961). Development of body scheme in children. *American Journal of Occupational Therapy*, 15, 99–102.
- Ayres, A. J. (1963). Eleanor Clark Sagle lecture. The development of perceptual motor abilities: A theoretical basis for treatment of dysfunction. *American Journal of Occupational Therapy*, 17, 221–225.
- Ayres, A. J. (1964). Tactile functions: Their relationship to hyperactivity and perceptual motor behavior. *American Journal of Occupational Therapy*, 18, 6–11.
- Ayres, A. J. (1966a). Interrelationships among perceptual-motor functions in a group of normal children. *American Journal of Occupational Therapy*, 20, 288–292.
- Ayres, A. J. (1966b). Interrelationships among perceptual-motor functions in children. *American Journal of Occupational Therapy*, 20, 68–71.
- Ayres, A. J. (1969). Deficits in sensory integration in educationally handicapped children. *Journal of Learning Disabilities*, 2, 160–168.
- Ayres, A. J. (1971). Characteristics of types of sensory integrative dysfunction. *American Journal of Occupational Therapy*, 25, 329–334.
- Ayres, A. J. (1972a). Types of sensory integrative dysfunction among disabled learners. *American Journal of Occupational Therapy*, 22, 13–18.
- Ayres, A. J. (1972b). Improving academic scores through sensory integration. *Journal of Learning Disabilities*, 5, 338–343.
- Ayres, A. J. (1973). *Sensory integration and learning disorders*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (1974). *The development of sensory integrative theory and practice: A collection of the works of A. Jean Ayres*. Dubuque: Kendall/Hunt Pub.
- Ayres, A. J. (1977a). Dichotic listening performance in learning-disabled children. *American Journal of Occupational Therapy*, 31, 441–446.
- Ayres, A. J. (1977b). Cluster analysis of measures of sensory integration. *American Journal of Occupational Therapy*, 31, 362–366.
- Ayres, A. J. (1977c). Effect of sensory integration on the coordination of children with choreoathetoid movements. *American Journal of Occupational Therapy*, 31, 291–293.
- Ayres, A. J. (1982). *Sensory integration and the child*. Los Angeles: Western Psychological Services.

- Ayres, A. J. (1989). *Sensory integration and Praxis tests*. Los Angeles: Western Psychological Services.
- Ayres, A. J., & Mailloux, Z. (1981). Influence of sensory integrations procedures on language development. *American Journal of Occupational Therapy*, 35, 383–390.
- Ayres, A. J., & Mailloux, Z. K. (1983). Possible pubertal effect on therapeutic gains in an autistic girl. *American Journal of Occupational Therapy*, 37(8), 535–540.
- Ayres, A. J., & Tickle, L. S. (1980). Hyper-responsivity to touch and vestibular stimuli as a predictor of positive response to sensory integration procedures to autistic children. *American Journal of Occupational Therapy*, 34, 375–381.
- Ayres, A. J., Mailloux, Z. K., & Wendler, C. L. (1987). Developmental dyspraxia: Is it a unitary function? *Occupational Therapy Journal of Research*, 7, 93–110.
- Bowman, O. J. (1989). In memoriam: A. Jean Ayres, 1920–1988: Therapist, scholar, scientist, and teacher. *American Journal of Occupational Therapy*, 43, 479–480.

Azaleptin

► [Clozapine](#)

B

Babbling

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Definition

Babbling can be defined as a type of prelinguistic, non-cry vocalization, which typically emerges by 6 or 7 months of age with repetition of the same consonant vowel (CV) syllable (“ba ba”) (Johnson 2008; Paul 2007). This can also be referred to as *canonical babbling* (Oller et al. 1998) or *reduplicative babbling* and is an important part of the developmental process of emerging speech and language. Utterances produced with full stop consonants such as /p/, /b/, /t/, and /d/ and vowels are most common at this stage, resulting in utterances such as /baba/ and /dIdI/ (“dee dee”). *Variegated babbling*, where successive syllables are not identical, begins to appear between 6 and 10 months of age (Paul 2007; Proctor 1989). This consists of a variety of CV and consonant-vowel-consonant (CVC) syllables that are not identical (“pa ta”). By the end of the first year, babbling should begin to imitate the

intonation and prosody of adult speech. This is also referred to as *jargon babble* (Paul 2007).

Historical Background

Research findings from the past several decades on the nature of babbling have documented a shift in the scientific and clinical evidence regarding the connection between babbling and speech and language acquisition. Early literature reported a weak relationship between babbling and early speech development (e.g., Jakobson 1941; Lenneberg 1967). It was not viewed as being composed of linguistic units but rather a biomechanical action where the infant lacks control over the sounds produced. This view, known as the motoric hypothesis, asserts that babbling is just a by-product of motor development. There was also a common misconception that babbling ended prior to the emergence of first words. In recent decades, however, there has been a shift to a linguistic hypothesis, which maintains that babbling has a neurolinguistic foundation and there is a continuity between babbling and early speech forms (Petitto et al. 2000; Vihman et al. 1986). This shift in opinion is based on a strong body of research suggesting that babble and speech share

phonological characteristics within target languages and within individual children (Whitehurst et al. 1991).

Current Knowledge

Progression and presentation of babbling, as well as the acquisition and use of speech and language, can vary greatly among children with autism. It is possible for babbling and other communication milestones to develop normally in this population but then later regress. Approximately 25–30% of children with autism exhibit babbling and begin to say words but then stop speaking between the ages of 15 and 24 months (Johnson et al. 2007). This has been documented by home videos of children who were typically developing, children with early-onset autism, and children with regressive-type autism and reported in a study by Dawson and Werner (2005). They found that the regressed children used complex babbling and words significantly more often than the early-onset children did. Furthermore, the children with regressive-type autism used complex babble nearly twice as often as typical children.

Certain children who present with developmental delays, including those with early-onset autism, may be unusually quiet and make few vocalizations. Others may produce atypical vocalizations such as humming and grunting, and fail to exhibit the typical canonical and variegated babbling within the expected time frames (Johnson 2008). Lack of canonical babbling by 10 months of age has been shown to predict delays in language development in the second year of life (Oller et al. 1998). Current research with infants who are typically developing and those with developmental delays has supported the continuity between babbling and its relationship to patterns in early speech (Davis and MacNeilage 1995; Mitchell 1997).

Typically developing infants exhibit a back-and-forth type pattern of babbling and apparent listening that is coordinated with the caregiver's speech and is similar to the conversational turn-taking that is used by older children (Johnson 2008). Children with autism may continue to

vocalize as if they are not aware of the caregiver's speech, with overlapping vocalizations and lack of eye contact. Parents may report that their child does not seem to recognize their voice or notice when they enter or leave the room. At the jargon babble stage near 1 year of age, they may lack inflection and prosody that is common by this stage.

Since differences and delays in babbling are frequently found in children with autism, an analysis of the child's pre-speech vocalizations by a speech-language pathologist may help to identify children who are at risk (Mitchell 1997). Children who exhibit a loss of babbling should also be referred for an evaluation, as this is a serious red flag. Hearing loss, delayed motor development, and lack of social interactions may also contribute to delays in babbling. For children who were born prematurely, corrected gestational age (CGA) should be used to compare early developmental milestones related to babbling.

A pediatrician can screen children for speech and language delays and may recommend further evaluation by a specialist, such as a speech-language pathologist. Proctor (1989) and Mitchell (1997) have provided instruments and guidelines for assessing vocal development of infants. Standardized evaluation tools, such as the *Communication and Symbolic Behavior Scales Developmental Profile* (Wetherby and Prizant 1993), and criterion-referenced assessments such as the *Rossetti Infant and Toddler Language Scale* (Rossetti 2006) can also be utilized to assess language in the pre-linguistic period.

For children who do not follow the expected progression of babbling and demonstrate a delay in speech and language development, early intervention which is specifically tailored to the individual, targets behavior and communication, and involves the parents or primary caregivers is the best treatment. Typically, a speech-language pathologist implements this intervention.

Future Directions

Many children who are later diagnosed with autism first present to their pediatrician with

delays and differences in speech and language development (Johnson 2008). Still, autism is not typically diagnosed until about 3–5 years of age. Research has shown that early intervention by 2–3 years of age results in more positive outcomes for children with autism (Osterling and Dawson 1994). Since language and communication impairments are part of the diagnostic criteria for autism, and babbling is one of the earliest developmental communication milestones which has been shown to be an important initial phase of speech production ability, lack of babbling by the end of the first year or regression of early speech skills should be recognized as a red flag. More studies on the different patterns and progressions of babbling in children with autism spectrum disorders would help professionals to better understand the link with later speech and language development and help to support earlier identification of children who may be at risk.

See Also

- [Communication and Symbolic Behavior Scale](#)
- [Communicative Acquisition in ASD](#)
- [Rossetti Infant-Toddler Language Scale](#)
- [Speech Delay](#)
- [Vocalization](#)

References and Reading

- American Speech Language Hearing Association. (2010). *How does your child hear and talk: Birth to one year*. Retrieved from: <http://www.asha.org/public/speech/development/01.htm>
- Berko-Gleason, J., & Burstein Ratner, N. (2008). *The development of language* (7th ed.). Upper Saddle River: Prentice Hall.
- Boysson-Bardies, B. (1999). *How language comes to children: From birth to two years*. Cambridge, MA: MIT Press.
- Davis, B., & MacNeilage, P. F. (1995). The articulatory basis of babbling. *Journal of Speech and Hearing Research*, 38, 1199–1211.
- Dawson, G., & Werner, E. (2005). Validation of the phenomenon of autistic regression using home videotapes. *Archives of General Psychiatry*, 62, 889–895.
- Eilers, R. E., Oller, D. K., Levine, S., Basinger, D., Lynch, M. P., & Urbano, R. (1993). The role of prematurity and socioeconomic status in the onset of canonical babbling in infants. *Infant Behavior & Development*, 16, 297–315.
- Jakobson, R. (1941). *Child language, aphasia and phonological universals*. (trans: Keiler, A.R.). The Hague: Mouton.
- Johnson, C. P. (2008). Recognition of autism before age 2 years. *Pediatrics in Review*, 29, 86–96.
- Johnson, C. P., Myers, S. M., & Council on Children with Disabilities. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120(5), 1183–1193.
- Lenneberg, E. H. (1967). *Biological foundations of language*. New York: Wiley.
- McCune, L., & Vihman, M. (2001). Early phonetic and lexical development: A productivity approach. *Journal of Speech, Language, and Hearing Research*, 44, 670–684.
- Mitchell, P. R. (1997). Prelinguistic vocal development: A clinical primer. *Contemporary Issues in Communication Science and Disorders (CICSD)*, 24, 87–92.
- Oller, D. K., Levine, S., Cobo-Lewis, A., Eilers, R., & Pearson, B. (1998). Vocal precursors to linguistic communication: How babbling is connected to meaningful speech. In R. Paul (Ed.), *Exploring the speech-language connection* (pp. 1–23). Baltimore: Paul H. Brookes.
- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders*, 24, 247–257.
- Paul, R. (2007). *Language disorders from infancy through adolescence: Assessment and intervention* (pp. 231–243). St. Louis: Mosby.
- Petitto, L. A., Zatorre, R., Gauna, K., Nikelski, E. J., Dostie, D., & Evans, A. (2000). Speech-like cerebral activity in profoundly deaf people while processing signed languages: Implications for the neural basis of human language. *Proceedings of the National Academy of Sciences*, 97(25), 13961–13966.
- Proctor, A. (1989). States of noncry vocal development in infancy: A protocol for assessment. *Topics in Language Disorders*, 10(1), 26–42.
- Rossetti, L. (2006). *Rossetti infant and toddler language scale: Manual*. East Moline: LinguiSystems.
- Sheinkopf, S. J., Mundy, P., Kimbrough Oller, D., & Steffens, M. (2000). Vocal atypicalities of preverbal autistic children. *Journal of Autism and Developmental Disorders*, 30(4), 345–354.
- Vihman, M. M., Ferguson, C. E., & Elbert, M. (1986). Phonological development from babbling to speech: Common tendencies and individual differences. *Applied Psycholinguistics*, 7, 3–40.
- Wetherby, A. M., & Prizant, B. M. (1993). *Communication and symbolic behavior scales: Manual*. Chicago: Riverside.
- Whitehurst, G. J., Smith, M., Fischel, J. E., Arnold, D. S., & Lonigan, C. J. (1991). The continuity of babble and speech in children with specific expressive language delay. *Journal of Speech and Hearing Research*, 34, 1121–1129.

Babysitter Training Guide for Families with Individuals with ASD

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Synonyms

Caregiver training program

Definition

A training program with supplemental informational materials has been developed with strategies to support short-term care for individuals with autism spectrum disorders (ASD). This program, entitled *Sit for Autism*, was originally designed to inform babysitters by increasing their understanding of young children with ASD and how to best support their needs. However, as the training program began to be implemented, the trainers determined that birth parents, foster parents, grandparents, and other caregivers can benefit from the information presented. These diverse caregivers could then use the tools provided to assist babysitters in caring for their children with ASD. This entry includes a general description of the program and next steps for implementation.

The *Sit for Autism* 2-h training session includes delivery of factual-based information and hands-on activities. In the initial portion of the training, a PowerPoint presentation is used to share

foundational content, including the definition of ASD, strengths of children with ASD, and common social, communication, and behavior characteristics of individuals with ASD. Key theories [Theory of Mind (Baron-Cohen 1995)] and challenges exhibited in the realms of behavior and social communication are explored through content and active engagement of the participants. Along with related content about ASD, targeted evidence-based practices (EBPs) (Wong et al. 2013) are defined and modeled, and a detailed babysitter preparation packet is also provided. Suggestions for implementing the target EBPs in the home and/or community setting are discussed and applied to scenarios presented by the trainer and participants. Opportunities for the participants to reflect on their personal experiences, ask questions, share strategies, and actively engage in dialogue are available throughout the training. Each participant is given the opportunity to practice using these tools during the training session and benefit from presenter feedback.

Participants are given a “Sit Kit” containing PowerPoint with factual information and explanation, written instructions for use of each EBP strategy, Babysitter Sit Preparation guide booklet, and visual supports related to the presented EBPs (visual schedule options, choice board, communication pages, and timer options). A visual timer and instructions on its use are included in each kit.

The included “Babysitter Sit Preparation” guide booklet is to be completed by the caregiver before leaving their child as a means of sharing with the babysitter or other potential temporary caregivers ways to support and understand their child. The information presented in the guide provides the caregiver with a breakdown of the child’s skills and challenges in social, communication, and behavior and allows for personalization of strategies to utilize during the time of short-term care. Pages referred to as “The Sit Prep Kit” components provide the opportunity to share emergency contact information, medical information, food preferences, the child’s typical schedule, special equipment needed, the bedtime routine, adaptive areas in need of assistance, homework routine, activity preferences, and information about siblings and what the child

likes to do with the siblings. The final page includes a space to write down the expected schedule of activities for the time of the babysitting/temporary care event.

At the conclusion of the training, ten tips for success when providing short-term care to an individual with autism are reviewed. These include providing structure; being consistent; using visuals; providing warning time when an activity is over; giving short, clear directions; limiting verbal language; leaving time for the child to answer; being attentive; being understanding and supportive; and supporting with humility. A final question and answer session concludes the training. Trainer contact information is provided to the participants for any follow-up they would like to have with the trainer.

This Sit for Autism training has been conducted 55 times from 2013 through April 2019. A total of 526 participants from a variety of socioeconomic and ethnic backgrounds throughout the state of Connecticut attended the training. In an effort to expand access of the materials by more families, all of the materials have been translated into Spanish. Translators to support the presenters have attended several sessions, with an exclusive training session in Spanish piloted. “Sit Kits” are offered to participants in electronic format so that parents and caregivers can personalize them and use them on more than one occasion. A Train the Trainer model has been developed to begin to train other professionals in order to reach more families in the state.

See Also

- [Autism Theory](#)
- [Children with Autism in Foster Care](#)
- [Parent Training](#)

References and Reading

- Baron-Cohen, S. (1995). *Mind blindness: An essay on autism and theory of mind*. Cambridge, MA: The MIT Press.
- Connecticut Lifespan Respite Coalition, Inc. (2008). Get creative about respite: A parent’s guide. Retrieved from

http://www.ct.gov/dph/lib/dph/family_health/children_and_youth/pdf/59684_parents_guide.pdf

Wong, C., Odom, S. L., Hume, K., Cox, A. W., Fetting, A., Kucharczyk, S., . . . & Schultz, T. R. (2013). *Evidence-based practices for children, youth and young adults with Autism Spectrum Disorders*. Chapel Hill: The University of North Carolina, Frank Porter Graham Child Development Institute, Autism Evidence-Based Practice Review Group.

Bad Science

- [Pseudoscience](#)

Balovaptan

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Synonyms

[RG7314](#); [RO 5028442](#); [RO-5285119](#)

Definition

Balovaptan (previously RG7314) is an investigational drug candidate developed by Hoffman-La Roche for the potential treatment of autism spectrum disorder (ASD). It is an orally active non-peptide vasopressin receptor antagonist (vaptan) with reported selectivity for the vasopressin V1a receptor. Balovaptan is the first vaptan drug to be tested in psychiatry, and its therapeutic potential is yet unknown. Based on the results of a recent phase II clinical trial in adults with ASD (Bolognani et al. 2017), balovaptan was granted “breakthrough therapy” designation by the United States Food and Drug Administration in January

2018. The drug is currently undergoing a phase II clinical trial in children and adolescents with ASD for the alleviation of core ASD symptoms. To date, data on the safety or efficacy of balovaptan have not been published.

Vasopressin, also known as antidiuretic hormone, is a small peptide produced in the hypothalamus. It is a major physiological regulator of water homeostasis, affecting urine concentration and blood volume. Vasopressin binds to three different receptors, V1a, V1b (also called V3), and V2. Activation of V1a receptors on vascular smooth muscle causes vasoconstriction, and activation of V2 receptors promotes water reabsorption in the kidneys. V1a receptors are also expressed on neurons throughout the central nervous system, and vasopressin is known to act as a neuromodulator. The physiological effects of the V1b receptor are less well characterized, though V1b activation is thought to promote the release of adrenocorticotrophic hormone (see ► [“Hypothalamic-Pituitary-Adrenal Axis”](#)). The study of vaptan drugs has primarily focused on agents that antagonize renal V2 receptors (e.g., conivaptan, tolvaptan) and their ability to treat conditions characterized by hyponatremia and fluid overload (Ali et al. 2007). However, a single-dose proof-of-mechanism study testing a small-molecule V1a antagonist (RG7713) in adults with ASD has provided preliminary evidence that targeting this receptor improves social cognition (Umbricht et al. 2017). Thus, in addition to promoting diuresis, vaptan drugs are potentially useful as therapeutics for neuropsychiatric disorders.

Though there is limited evidence supporting vasopressin system dysfunction in the pathogenesis of ASD, both vasopressin and the related neuropeptide oxytocin (see ► [“Oxytocin”](#)) have been implicated in the regulation of social cognition and behavior (Meyer-Lindenberg et al. 2011). Oxytocin and vasopressin interact with a number of other neurotransmitter systems, and the mechanisms by which they alter social functioning in humans have yet to be fully understood. Nevertheless, the oxytocin and vasopressin systems remain feasible targets for novel therapeutics that aim to address the social-communicative

symptoms of ASD (Baribeau and Anagnostou 2015). A phase I clinical trial of balovaptan was completed in 2015, testing the drug in healthy adults between 18 and 45 years of age. The phase I trial attempted to replicate previously reported effects of vasopressin administration on brain activity and functional connectivity during functional MRI tasks. Notably, this trial only included males, as the effects of oxytocin and vasopressin are thought to differ by sex. To establish proof of mechanism, the study assessed the ability of balovaptan to modulate vasopressin-induced changes in brain activity. The results of the trial were not published, though the drug has continued to the next stage of testing.

The most recent data on balovaptan's effects come from the VANILLA (Vasopressin ANtagonist to Improve social communication in Autism) study, a phase II clinical trial in adult males with ASD and normal intellectual functioning, primarily investigating the compound's safety and efficacy in this population. Results of this study were presented at the 2017 International Meeting for Autism Research (Bolognani et al. 2017). A total of 223 patients were randomized to either the placebo condition or 1 of 3 dosages (1.5 mg, 4 mg, 10 mg) for 12 weeks, and of those individuals, 192 (86%) completed the trial. Although the drug appeared to be safe and well-tolerated over the treatment period, there was no change in the primary endpoint (caregiver reported Social Responsiveness Scale Scores) between drug and placebo groups. However, at the 4 and 10 mg doses, significant differences emerged between drug and placebo on the Vineland Adaptive Behavior Scales (VABS), one of the secondary endpoints. Between baseline and the 12-week endpoint, VABS composite scores improved over placebo with effect sizes of 0.59 in the 4 mg group and 0.49 in the 10 mg group. Further analyses of this effect found the improvement in composite scores to be driven by the social and communication domains of the VABS. No consistent treatment effects were noted in any of the other secondary endpoints, including the Aberrant Behavior Checklist (ABC); the Repetitive Behavior Scale-Revised (RBS-R); the State-Trait Anxiety Inventory (STAI); the Anxiety, Depression, and Mood Scale (ADAMS); and the

CGI-I score. While the primary endpoint was not significant in this trial, the compound's effect on VABS scores has caused the drug maker to alter its methods for future investigations. Currently, a second phase II trial of balovaptan (aV1ation; NCT02901431) is under way in children and adolescents with ASD, this time utilizing the VABS as the primary endpoint.

See Also

- [Oxytocin](#)
- [Social Cognition](#)

References and Reading

- A Phase 1, Randomized, Double-blind, Placebo-controlled Crossover Study of RG7314 on the Potential Regulation of Higher Brain Functions in Healthy Male Participants: Proof of Mechanism. (2014). Retrieved from <https://clinicaltrials.gov/show/NCT02205073>. (Identification no. NCT02205073).
- A Study to Investigate the Efficacy and Safety of RO5285119 in Participants With Autism Spectrum Disorder (ASD). (2016). Retrieved from <https://clinicaltrials.gov/ct2/show/NCT02901431>. (Identification no. NCT02901431).
- Ali, F., Guglin, M., Vaitkevicius, P., & Ghali, J. K. (2007). Therapeutic potential of vasopressin receptor antagonists. *Drugs*, 67(6), 847–858.
- Baribeau, D. A., & Anagnostou, E. (2015). Oxytocin and vasopressin: Linking pituitary neuropeptides and their receptors to social neurocircuits. *Frontiers in Neuroscience*, 9, 335.
- Bolognani, F., del Valle Rubido, M., Squassante, L., Wandel, C., Liogier D'ardhuy, X., Boak, L., ... Umbricht, D. (2017). Results of a phase 2 randomized double-blind placebo controlled study (VANILLA) investigating the efficacy and safety of a V1a antagonist (RG7314) in adult men with ASD. Paper presented at the international meeting for autism research, San Francisco.
- Meyer-Lindenberg, A., Domes, G., Kirsch, P., & Heinrichs, M. (2011). Oxytocin and vasopressin in the human brain: Social neuropeptides for translational medicine. *Nature Reviews Neuroscience*, 12(9), 524–538.
- Umbricht, D., del Valle Rubido, M., Hollander, E., McCracken, J.T., Shic, F., Scahill, L., ..., Grundschober, C. (2017). A single dose, randomized, controlled proof-of-mechanism study of a novel vasopressin 1a receptor antagonist (RG7713) in high-functioning adults with autism spectrum disorder. *Neuropsychopharmacology*, 42(9), 1914.

Banophen™ [OTC]

- [Diphenhydramine](#)

Banophen™ Anti-itch [OTC]

- [Diphenhydramine](#)

Barbiturates

- [Sedative Hypnotic Drugs](#)

Barnes Akathisia Scale

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Definition

The Barnes Akathisia Scale is a scale designed to rate the severity of drug-induced or Parkinson disease-based akathisia. Akathisia – literally meaning not sitting – is characterized by an inner restlessness, causing constant motion of hands or feet. Symptoms of akathisia can persist for years, even after discontinuing the precipitating drug. The assessment of akathisia with the Barnes Akathisia Scale includes objective and subjective questions.

See Also

- [Antipsychotics: Drugs](#)
- [Pyramidal System](#)

References and Reading

- Barnes, T. R. (1989). A rating scale for drug-induced Akathisia. *British Journal of Psychiatry*, 154, 672–676.
- Barnes, T. R. (2003). The Barnes Akathisia rating scale—revisited. *Journal of Psychopharmacology*, 17(4), 365–370. Review.

Barriers and Facilitators that Prevent and Enable Physical Healthcare Services Access for Autistic Adults

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Definition

A barrier to physical healthcare access is any component of healthcare provision that negatively impacts the healthcare service access of autistic people. Conversely, facilitators are components of healthcare provision that improve the healthcare service access of autistic people. Barriers that affect the physical healthcare access of autistic people include communication, health professional's knowledge about autism, sensory sensitivities, and cognitive factors (Mason et al. 2019). Brief information is given below about these factors, with examples; more information is contained in the referenced papers.

Autistic people's communication style (e.g., difficulty describing symptoms, particularly those that involve abstract concepts or hyper-specific language) can be a barrier to healthcare provision (Nicolaidis et al. 2015). Moreover, some autistic people may not provide key information unless questions directly ask for this information (Bradshaw et al. 2019). But it is important to be mindful that communication

involves two parties, so a clinician's responsiveness (or lack thereof) to alternative communication styles (e.g., written notes) can act as a facilitator (or barrier) to healthcare provision (Mason et al. 2019).

Autistic people report that some clinicians have limited knowledge about autism. This can lead to clinicians making assumptions about the capabilities of autistic people (e.g., that the autistic person is not fully competent if they attend an appointment with a friend/family member) or clinicians assuming that behavioral expressions of symptoms (e.g., pain) are a part of autism (Nicolaidis et al. 2015). This agrees with many studies which have also identified that clinicians report a lack of knowledge about, but desire to learn more about, autism (Morris et al. 2019). (Note, some data indicate that many clinicians do know the key features of autism; Unigwe et al. 2017.)

Sensory sensitivities can affect multiple aspects of a healthcare visit: travel to a visit, waiting rooms, and during the visit itself. Autistic people can experience sensory issues when using busy or noisy public transport (Bradshaw et al. 2019). Waiting rooms can be problematic for autistic people due to lighting (e.g., bright fluorescent light) or being crowded; this may mean autistic people avoid seeking appointments if this environment cannot be avoided. Likewise, lighting during a healthcare visit may be too bright, or the walk from the waiting room to the healthcare provider's office may be disorientating.

The cognitive factors unique to each autistic person can also be a barrier to healthcare access. Due to the cognitive load of managing social presentation, or trying to process verbal information in "real time," autistic people find it difficult to understand information from health professionals (Mason et al. 2019; Raymaker et al. 2017). Moreover, prospective memory difficulties (e.g., accurately recording future appointments or remembering to take medication) can be a barrier. Therefore, autistic people may need more support in how healthcare information is disseminated (e.g., audio recordings of appointments or a clearly phrased written summary of the appointment) (Mason et al. 2019).

These barriers can interact. For instance, the anxiety brought about by being in a waiting room has a consequence for the subsequent healthcare appointment. An autistic person who is anxious (or exhausted from attempting to manage typical healthcare settings) may then find interacting with the healthcare provider more difficult or may find processing healthcare information, or providing information about their needs, more challenging. This means more time in the appointment will be spent on maintaining the conversation and coping with anxiety, with less time spent processing the content of the healthcare discussion. Thus, the autistic person may leave without sufficient knowledge about the discussion or with multiple questions that were not addressed in the appointment.

See Also

- [Health Disparities](#)
- [Quality of Life for Transition-Age Youth with ASD](#)

References and Reading

- Bradshaw, P., Pellicano, E., van Driel, M., & Urbanowicz, A. (2019). How can we support the healthcare needs of autistic adults without intellectual disability? *Current Developmental Disorders Reports*, 6(2), 45–56.
- Mason, D., Ingham, B., Urbanowicz, A., Michael, C., Birtles, H., Woodbury-Smith, M., ... Nicolaidis, C. (2019). A systematic review of what barriers and facilitators prevent and enable physical healthcare services access for autistic adults. *Journal of autism and developmental disorders*, 8, 3387–3400.
- Morris, R., Greenblatt, A., & Saini, M. (2019). Healthcare providers' experiences with autism: A scoping review. *Journal of Autism and Developmental Disorders*, 49(6), 2374–2388.
- Nicolaidis, C., Raymaker, D. M., Ashkenazy, E., McDonald, K. E., Dem, S., Baggs, A. E., ... Boisclair, W. C. (2015). "Respect the way I need to communicate with you": Healthcare experiences of adults on the autism spectrum. *Autism*, 19(7), 824–831.
- Raymaker, D. M., McDonald, K. E., Ashkenazy, E., Gerrity, M., Baggs, A. M., Kripke, C., ... Nicolaidis, C. (2017). Barriers to healthcare: Instrument development and comparison between autistic adults and adults with and without other disabilities. *Autism*, 21(8), 972–984.
- Unigwe, S., Buckley, C., Crane, L., Kenny, L., Remington, A., & Pellicano, E. (2017). GPs' confidence in caring for their patients on the autism spectrum: An online self-report study. *The British Journal of General Practice*, 67(659), e445–e452.

Barriers to and Facilitators of Successful Early School Transitions for Children with Autism Spectrum Disorders

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Synonyms

[Early school beginnings](#); [Successful school transition](#); [Transition support practices](#)

Definition

The shift from an early childhood setting (e.g., home, preschool) to elementary school can be challenging for children, their families, and their teachers. Families must adapt to important changes once children transition to school (TTS), such as increased academic and social demands, decreased family support, and more transitions throughout the school day (Rimm-Kaufman et al. 2000). This shift tends to be particularly challenging for children with neurodevelopmental disorders, such as autism spectrum disorder (ASD). In addition to the challenges experienced by neurotypical children, the social/communication deficits and challenges adapting to change experienced by children with ASDs make the transition to a novel, social environment like school particularly problematic for children with ASDs and their families (Forest et al. 2004).

Research suggests that the implementation of collaborative transition practices can facilitate the TTS for children with ASDs and their families (Fontil et al. 2019a, b). Transition support practices can be defined as a series of activities that are implemented before, during, and/or after the TTS to support the child and family as they move from one educational environment to the next. Examples of transition support practices include transition planning meetings, school visits, and school orientations. The implementation of high-quality TTS supports (i.e., supports that are individualized to the child and their family's needs and involves direct contact with families) has been correlated with positive social and academic outcomes for children.

Collaboration between key stakeholders in a child's life is essential to facilitating successful school beginnings. That is, home, the sending school (e.g., preschool, intervention center, day care), and the receiving school should be in communication (e.g., transition meetings, sharing information concerning the child between early childhood settings and schools) and aim to develop meaningful partnerships characterized by open communication, valuing insights provided by family members, and considering the needs and values of families. Parents of children with ASDs have reported experiencing more stress than parents of children with other developmental disabilities (DDs; Griffith et al. 2010), which could be attributed to distinct characteristics that are associated with ASDs (i.e., delayed diagnosis, more behavioral problems, lack of reciprocity; Griffith et al. 2010). Furthermore, a common theme in the literature among parents of children with ASDs is a lack of collaboration between home and school during the transition planning (Fontil et al. 2019b). Given that the TTS is often especially stressful for families with children with ASDs, mutual respect and meaningful partnerships need to be underscored and made a priority.

Although collaboration is integral in facilitating successful school beginnings, several other important transition support practices have been highlighted in the literature for children with and without DDs. Child classroom visits, teacher home visits, and caregiver orientation services

are significant predictors of academic achievement (Schulting et al. 2005). Furthermore, increasing the number of school visits (i.e., 5–7 visits), prior to the TTS, is more valuable than having the child visit the receiving school a few times (Schischka et al. 2012), and implementing a greater variety of transition practices is correlated with improved academic outcomes (Ahtola et al. 2011). Early intervention program staff, servicing children with ASDs, report using a diverse set of transition supports to facilitate elementary school entry. For example, program staff provide receiving schools with information concerning a child, discuss the TTS with families, meet staff at the receiving school, encourage families to visit and meet staff at the receiving school, and hold transition planning meetings (Fontil et al. 2019a).

Despite the social and academic implications of implementing high-quality transition supports, several barriers impede the implementation of collaborative transition practices. Commonly cited barriers to collaborative transition support practices for families with children with ASDs include lack of time, lack of resources, insufficient school staff training, and divergent beliefs concerning the transition planning process (Fontil et al. 2019b). Furthermore, families of children with ASDs report being unsatisfied with receiving school support, lack of continuity of care between sending and receiving programs, and a lack of collaboration between home and the receiving school (Fontil et al. 2019b).

The extant literature on TTS has highlighted that there are more commonalities than differences when we compare transition experiences of children with ASDs to those of children with other DDs, with the exception that children with ASD have more difficulty with horizontal transitions (i.e., smaller transitions throughout the day; e.g., moving from classroom to playground). These smaller, daily transitions are more common in elementary schools in comparison to the preschool environment (Fontil et al. 2019b). Additionally, elementary school staff report exhibiting an insufficient amount of ASD-specific knowledge, which may have an impact on their capacity to successfully integrate students (Fontil et al. 2019b). Similarly, global comparisons, between

countries, reveal an international shift in attention toward TTS policy and practices which are supported by the growing number of TTS studies over time (Fontil et al. 2019b).

Based on the literature, systemic changes are needed to promote better TTS for children with ASD. Provided with sufficient financial support, school teachers could be provided with resources to develop their knowledge to facilitate transition practices (i.e., workshops and training on individuals with diverse needs, release time to visit children's sending programs). Additionally, families and caregivers need to be supported throughout the transition process (i.e., providing information about school supports). Finally, transition support practices require involvement of all key stakeholders to facilitate collaborative transition practices; this includes active engagement between families, teachers, and other professionals (i.e., occupational therapists or resource teachers).

Future research should focus on evaluating the effectiveness of specific TTS supports for students with ASD. Current research investigating transition supports tend to be descriptive in nature. Future research should investigate efficacy through randomized control trials. Furthermore, it is important to more clearly differentiate the needs of children with ASDs from those with other DDs at the point of school entry to facilitate a clearer understanding of the specific supports that facilitate TTS for children with ASD.

See Also

- College Transitional Programs
- Family-Centered Care, Second Edition
- Inclusion
- Stepped Transition in Education Program for Students with ASD (STEPS)

References and Reading

Ahtola, A., Silinskas, G., Poikonen, P.-L., Kontoniemi, M., Niemi, P., & Nurmi, J.-E. (2011). Transition to formal schooling: Do transition practices matter for academic performance? *Early Childhood Research Quarterly*, 26, 295–302. <https://doi.org/10.1016/j.ecresq.2010.12.002>.

Fontil, L., Sladeczek, I. E., Gittens, J., Kubishyn, N., & Habib, K. (2019a). From early intervention to elementary school: A survey of transition support practices for children with autism spectrum disorders. *Research in Developmental Disabilities*, 88, 30–41. <https://doi.org/10.1016/j.ridd.2019.02.006>.

Fontil, L., Gitten, J., Beaudoin, E., & Sladeczek, I. E. (2019b). Barriers to and facilitators of successful early school transitions for children with autism spectrum disorders and other developmental disabilities: A systematic review. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03938-w>.

Forest, E. J., Horner, R. H., Lewis-Palmer, T., Todd, A. W., & McGee, G. (2004). Transitions for young children with autism from preschool to kindergarten. *Journal of Positive Behavior Interventions*, 6(2), 103–112. <https://doi.org/10.1177/10983007040060020501>.

Griffith, G., Hastings, R., Nash, S., & Hill, C. (2010). Using matched groups to explore child behavior problems and maternal Well-being in children with down syndrome and autism. *Journal of Autism and Developmental Disorders*, 40(5), 610–619. <https://doi.org/10.1007/s10803-009-0906-1>.

Rimm-Kaufman, S. E., Pianta, R. C., & Cox, M. J. (2000). Teachers' judgments of problems in the transition to kindergarten. *Early Childhood Research Quarterly*, 15(2), 147–166. [https://doi.org/10.1016/S0885-2006\(00\)00049-1](https://doi.org/10.1016/S0885-2006(00)00049-1).

Schischka, J., Rawlinson, C., & Hamilton, R. (2012). Factors affecting the transition to school for young children with disabilities. *Australasian Journal of Early Childhood*, 37(4), 15–23.

Schulting, A. B., Malone, P. S., & Dodge, K. A. (2005). The effect of school-based kindergarten transition policies and practices on child academic outcomes. *Developmental Psychology*, 41(6), 860–871. <https://doi.org/10.1037/0012-1649.41.6.860>.

Barriers to Formal Diagnosis of Autism Spectrum Disorder in Adults

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Definition

Factors that complicate the process of receiving a medical diagnosis of “autism spectrum disorder” by a qualified professional for autistic adults who were not diagnosed in childhood.

Historical Background

Known prevalence of autism in adults is approximately 1.1%, but this is likely underestimated (Brugha et al. 2016). Experts estimate that as many as 40% of autistics are not diagnosed in childhood (Baron-Cohen et al. 2009). Those at highest risk of being misdiagnosed or not recognized during childhood include females, middle-aged and older adults, and individuals with more subtle traits and without cognitive or language delays.

In general, females are at a disproportionate risk of misdiagnosis, delayed diagnosis, and not receiving a diagnosis compared to males across age groups (Loomes et al. 2017). There are many theories on the causes of missed diagnosis of autism among females. Emerging evidence shows a slightly different clinical presentation of autism among females, often referred to as the female autism phenotype, which might be missed by clinicians, parents, teachers, and others looking for “classic” traits more common in males. Females may be more likely to exhibit subtle and internalized symptoms and less likely to show overt restricted interests than males (Bargiela et al. 2016; Loomes et al. 2017). “Camouflaging” is also common among females, in which individuals learn strategies to mask autistic traits and model typical social behaviors, making it more difficult to detect a diagnosis. Little is known about the impact of gender bias on diagnosing individuals who identify with a gender other than sex assigned at birth (e.g., agender, non-binary, genderqueer, transgender), but it is likely that these individuals are also at increased risk of missed diagnosis due to a reliance on male norms for the development of current diagnostic tools.

Middle-aged and older adults are also at risk of missed diagnosis due to lack of awareness and understanding of autism during their childhood, particularly since the categorization of autism in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) has changed significantly over time. Autism did not appear in the DSM until 1980, and prior to the release of the DSM-III-R in 1987, diagnostic criteria for autism limited this

label to individuals with severe symptoms and onset by age 30 months. The DSM-IV, released in 1994, was the first edition to identify autism as a “spectrum” and included the category of Asperger’s disorder for the first time, which could include individuals with more subtle traits. Many adults who grew up prior to these changes in the DSM were not diagnosed with autism because the criteria had not yet been established.

In the most recent edition, the DSM-5 eliminated the term “Asperger’s” and categorized autism as a continuous spectrum called “autism spectrum disorders.” The latest categorization identifies traits that may appear across the lifespan and does not identify a specific window of onset, which may increase clinician’s ability to apply this diagnosis to adults meeting the criteria. However, there is growing evidence that the latest criteria are more likely than the previous categorization to exclude females, older individuals, individuals with above average intelligence, and individuals with subtle traits (Mazurek et al. 2017).

Adults are increasingly becoming aware of their own autistic identity with a rise in media representation of autism, biographical accounts of diagnosis during adulthood, and online presence of support groups and forums related to autism. In addition, many adults recognize their own autistic traits when their children are diagnosed with autism. Self-diagnosis of autism is a growing phenomenon, and many individuals report they are satisfied with a self-diagnosis and confident in their autistic identity without seeking a formal diagnosis (Lewis 2016a). However, others who are self-diagnosed report recurring self-doubt about their autism status and experience unresolved cyclical grief. Without a formal diagnosis, individuals are also ineligible for professional support of any kind and typically lack access to community resources.

There are known benefits to obtaining a diagnosis beyond increased access to services. Individuals who are formally diagnosed report that awareness of their autistic identity was often a relief and led to a new sense of belonging, empowerment, understanding of strategies to improve quality of life, and increased self-acceptance (Crane et al. 2018; Lewis 2016b).

Many autistics are unaware of their diagnosis until adulthood, and awareness of autism can significantly benefit their mental health.

Current Knowledge

Autistic adults seeking diagnosis face significant barriers, and 80% report that obtaining a formal diagnosis was difficult or not possible (Taylor and Marrable 2011). The process of obtaining a formal diagnosis typically takes years, and on average adults see five professionals before receiving a diagnosis (Jones et al. 2014; McKenzie et al. 2015).

Barriers identified by adults seeking formal diagnosis include anxiety; cost; lack of access to adult specialists and limited awareness of autism by most professionals; inability to describe their symptoms; fear of not being believed or understood; lack of rapport or mistrust of healthcare professionals; stigma; and complexity of the healthcare system (Crane et al. 2018; Lewis 2017; Taylor and Marrable 2011; Vogan et al. 2017).

Characteristics of autism can exacerbate these barriers, creating a gap that precludes diagnosis for those with challenges not severe enough to be detected in childhood but too severe to pursue a diagnosis in adulthood. For example, deficits in social functioning often lead to social anxiety, which may prevent individuals from making an appointment with a healthcare professional due to worries about social interactions with receptionists, clinicians, etc. Individuals may also feel anxiety about the sensory experience of the waiting room or office that prevents them from making an appointment or following up.

In addition, approximately half of autistics experience alexithymia, or difficulty identifying and verbalizing their own feelings and emotions, which may make it difficult for them to communicate their symptoms adequately to a professional who does not specialize in autism (Trammell et al. 2013). They may also struggle with introspection or be unable to recognize their own autistic traits.

Individuals who have challenges with executive functioning, which includes tasks such

as organizing, focusing, and multitasking, may have difficulty navigating the healthcare system, planning for transportation, keeping appointments, or coordinating other tasks needed to pursue a diagnosis. Many individuals seeking a diagnosis of autism are also unemployed or employed part time (Happé et al. 2016), which may prohibit them from being able to maintain insurance or to afford the costs associated with evaluation and diagnosis.

Fear of not being believed, being dismissed by clinicians, or being blamed for symptoms is very common among adults seeking diagnosis, particularly among females (Crane et al. 2018; Lewis 2017). Adults report that they feel they are at the mercy of their clinicians for referrals as gatekeepers of diagnosis and fear that lack of clinician awareness of autism presentation in adults or in females may prevent them from receiving a diagnosis. Many fear detrimental effects on their identity formation if they are evaluated and told they do not meet criteria for a diagnosis of autism. Individuals who believe they are autistic also commonly report that they feel they have been misdiagnosed with co-occurring mental health conditions due to a lack of clinician understanding of their autistic traits, for example, that autistic traits are confused with another diagnosis or that difficulties with mental health are indirectly caused by autism (Au-Yeung et al. 2018).

Beyond those barriers identified by autistics, clinicians also cite significant barriers to making a diagnosis of autism in adults. Since autism is a neurodevelopmental condition, it is characterized by the presence of autistic traits in the developmental period. One significant barrier to clinicians making a diagnosis is the practicality of interviewing a parent or other reliable individual who can speak to the presence of these traits during childhood (Lai and Baron-Cohen 2015; Trammell et al. 2013). There may be limitations such as the willingness or availability of informants to meet, their ability to recall details from the individual's early childhood, or their perception of key events compared to the individual seeking a diagnosis (e.g., what they view as culturally "typical" vs. "atypical" behavior). If a parent or childhood caregiver cannot provide this information,

clinicians may opt to meet with other informants who can speak to the individual's childhood such as an older sibling or other relatives. If no such informants are available, clinicians must rely on educational and medical records and the adult's recollection of childhood for information on the developmental period, which may lack sufficient detail to make a diagnosis.

Clinician knowledge of autism is another significant barrier to adult diagnosis. Few instruments exist to assist in the assessment of adults, and even fewer have validated norms for adults. While the Autism Diagnostic Observation Schedule (ADOS) module 4 is the only validated tool for adult diagnosis, this must be used with caution due to its limited sensitivity to detect symptoms in females and individuals who have learned strategies that may camouflage symptoms (Lai and Baron-Cohen 2015; Trammell et al. 2013). Adult diagnosis of autism relies heavily on clinician expertise, and few clinicians specialize in evaluating adults for autism. Primary care providers report limited understanding and lack of training about autism in general, with three out of four clinicians rating their knowledge and skills in providing care to autistic patients as poor or fair (Zerbo et al. 2015).

Diagnosis is further complicated because more than 70% of autistics have at least one co-occurring medical, psychiatric, or developmental condition, most often including anxiety and mood disorders (Happé et al. 2016; Lai and Baron-Cohen 2015; Trammell et al. 2013). Since many differential diagnoses have overlapping symptoms or traits, clinicians may struggle to determine whether characteristics are related to autism, a co-occurring condition, or a differential diagnosis. Making a differential diagnosis is especially challenging when clinicians are missing key information about onset of traits and the developmental period.

Person-environment fit and cultural norms can also significantly impact interpretation of behaviors and traits and prevent diagnosis (Lai and Baron-Cohen 2015). That is, what is considered typical social behavior in one environment may be considered atypical in another environment. Therefore, an individual with many autistic traits

but whose traits do not interfere with functioning might not meet criteria for diagnosis, while an individual with fewer autistic traits that do interfere with functioning might meet criteria for diagnosis. These nuances may complicate the process of determining a diagnosis in adulthood where developmental milestones are more ambiguous.

Given these factors, even if the autistic individual is able to overcome the significant barriers that may prevent them from meeting with a clinician and accurately communicating information about their autistic traits, clinicians still might not be able to make an accurate diagnosis. For those who actually receive a diagnosis, experiences are often negative, with 40% of autistics reporting they were "very/quite dissatisfied" with the diagnostic process (Jones et al. 2014).

Future Directions

Given the potential benefits of recognizing autism for identity building and self-acceptance, it is critical to increase access to diagnosis for those on the spectrum. The World Health Organization recommends screening all children for autism as part of routine care (World Health Organization 2018), and over time, this practice should help reduce the number of adults who were not diagnosed in childhood.

There continues to be a paucity of validated tools for screening and diagnosis of autism in adults, which is a research priority (Wigham et al. 2018). Increased attention must be paid to developing diagnostic tools for all ages that recognize subtle and internalized traits that are more likely to be seen in females and individuals without cognitive impairment or language delays.

Clinicians across practice settings also require increased training on autism. Primary care providers may be the only healthcare professionals in position to detect autism in undiagnosed adults, so knowledge of common presentation is critical. There is a need for increased education in undergraduate and graduate settings for healthcare professionals to increase identification of autism and to minimize the negative experiences of those

who feel that their autistic characteristics are dismissed by clinicians who lack understanding of adult presentation (Zerbo et al. 2015).

All professionals must also be mindful of the significance of self-diagnosis in adults who believe they are autistic. Since such significant barriers to diagnosis exist, many adults rely on a self-diagnosis for autistic identity formation (Lewis 2016a). Dismissal of a self-diagnosis by a professional may be detrimental to self-acceptance and understanding. Professionals must be mindful of their own preconceptions about self-diagnosis and remain open to exploring the potential of an autism diagnosis in those who perceive themselves as being on the autism spectrum even if “classic” autistic traits are absent.

For those who do receive an autism diagnosis in adulthood, little to no post-diagnostic support is offered. Most individuals indicate that they would like counseling, social skills training, and access to support groups. However, as many as 77% receive no support whatsoever after diagnosis (Jones et al. 2014; Taylor and Marrable 2011), and qualitative studies indicate that individuals often feel lost and directionless and lack support after diagnosis (Crane et al. 2018; Lewis 2016b). There is a need for an effective and evidence-based approach to assist adults in managing mental health challenges as well as providing practical supports and resources after diagnosis.

Though formal services are limited, there are several online communities that promote positive images of autism and neurodiversity. Many adults utilize these websites to seek information and connection while working through the diagnostic process and beyond. Table 1 provides examples of some of these sites.

There are many severe barriers to formal diagnosis of autism in adulthood. Primary barriers include autistic traits themselves that prevent the individual from obtaining an assessment, lack of understanding of adult presentation of autism by clinicians, the impracticality of including informants from the developmental period in assessment, and the lack of appropriate tools for adult diagnosis. Given the potential mental health benefits of being aware of autism identity, researchers

Barriers to Formal Diagnosis of Autism Spectrum Disorder in Adults, Table 1 Examples of online communities for adults with ASC

Asperger/Autism Network	https://www.aane.org/
Autism Self Advocacy Network	https://autisticadvocacy.org/
Autism Empowerment	https://www.autismempowerment.org/
Autism Self Advocacy Network	https://autisticadvocacy.org/
Reddit [Autism Subreddit]	https://www.reddit.com/r/autism/
Twainbow	https://www.twainbow.org/
Wrongplanet	https://wrongplanet.net/

and clinicians must prioritize interventions and tools that increase early screening and access to diagnosis for autistic adults.

See Also

- ▶ Accuracy of the ADOS-2 in Identifying Autism Among Adults with Complex Psychiatric Conditions, The
- ▶ Autism Acceptance and Mental Health
- ▶ Diagnostic Instruments in Autistic Spectrum Disorders
- ▶ Social Camouflaging in Adults with ASD

References and Reading

Au-Yeung, S. K., Bradley, L., Robertson, A. E., Shaw, R., Baron-Cohen, S., & Cassidy, S. (2018). Experience of mental health diagnosis and perceived misdiagnosis in autistic, possibly autistic and non-autistic adults. *Autism*, 23(6), 1508–1518. <https://doi.org/10.1177/1362361318818167>.

Bargiela, S., Steward, R., & Mandy, W. (2016). The experiences of late-diagnosed women with autism spectrum conditions: An investigation of the female autism phenotype. *Journal of Autism and Developmental Disorders*, 46(10), 3281–3294. <https://doi.org/10.1007/s10803-016-2872-8>.

Baron-Cohen, S., Scott, F. J., Allison, C., Williams, J., Bolton, P., Matthews, F. E., & Brayne, C. (2009). Prevalence of autism-spectrum conditions: UK school-based population study. *British Journal of Psychiatry*, 194(6), 500–509. <https://doi.org/10.1192/bjp.bp.108.059345>.

- Brugha, T. S., Spiers, N., Bankart, J., Cooper, S.-A., McManus, S., Scott, F. J., ... Tyrer, F. (2016). Epidemiology of autism in adults across age groups and ability levels. *British Journal of Psychiatry*, 209(6), 498–503. <https://doi.org/10.1192/bjp.bp.115.174649>.
- Crane, L., Batty, R., Adeyinka, H., Goddard, L., Henry, L. A., & Hill, E. L. (2018). Autism diagnosis in the United Kingdom: Perspectives of autistic adults, parents and professionals. *Journal of Autism and Developmental Disorders*, 48(11), 3761–3772. <https://doi.org/10.1007/s10803-018-3639-1>.
- Happé, F. G., Mansour, H., Barrett, P., Brown, T., Abbott, P., & Charlton, R. A. (2016). Demographic and cognitive profile of individuals seeking a diagnosis of autism spectrum disorder in adulthood. *Journal of Autism and Developmental Disorders*, 46(11), 3469–3480. <https://doi.org/10.1007/s10803-016-2886-2>.
- Jones, L., Goddard, L., Hill, E. L., Henry, L. A., & Crane, L. (2014). Experiences of receiving a diagnosis of autism spectrum disorder: A survey of adults in the United Kingdom. *Journal of Autism and Developmental Disorders*, 44(12), 3033–3044. <https://doi.org/10.1007/s10803-014-2161-3>.
- Lai, M.-C., & Baron-Cohen, S. (2015). Identifying the lost generation of adults with autism spectrum conditions. *The Lancet Psychiatry*, 2(11), 1013–1027. [https://doi.org/10.1016/S2215-0366\(15\)00277-1](https://doi.org/10.1016/S2215-0366(15)00277-1).
- Lewis, L. F. (2016a). Exploring the experience of self-diagnosis of autism spectrum disorder in adults. *Archives of Psychiatric Nursing*, 30(5), 575–580. <https://doi.org/10.1016/j.apnu.2016.03.009>.
- Lewis, L. F. (2016b). Realizing a diagnosis of autism spectrum disorder as an adult. *International Journal of Mental Health Nursing*, 25(4), 346–354. <https://doi.org/10.1111/inm.12200>.
- Lewis, L. F. (2017). A mixed methods study of barriers to formal diagnosis of autism spectrum disorder in adults. *Journal of Autism and Developmental Disorders*, 47(8), 2410–2424. <https://doi.org/10.1007/s10803-017-3168-3>.
- Loomes, R., Hull, L., & Mandy, W. P. L. (2017). What is the male-to-female ratio in autism spectrum disorder? A systematic review and meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry*, 56(6), 466–474. <https://doi.org/10.1016/j.jaac.2017.03.013>.
- Mazurek, M. O., Lu, F., Symecko, H., Butter, E., Bing, N. M., Hundley, R. J., ... Handen, B. L. (2017). A prospective study of the concordance of DSM-IV and DSM-5 diagnostic criteria for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(9), 2783–2794. <https://doi.org/10.1007/s10803-017-3200-7>.
- McKenzie, K., Forsyth, K., O'Hare, A., McClure, I., Rutherford, M., Murray, A., & Irvine, L. (2015). Factors influencing waiting times for diagnosis of autism spectrum disorder in children and adults. *Research in Developmental Disabilities*, 45–46, 300–306. <https://doi.org/10.1016/j.ridd.2015.07.033>.
- Schuck, R. K., Flores, R. E., & Fung, L. K. (2019). Brief report: Sex/gender differences in symptomology and camouflaging in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49(6), 2597–2604. <https://doi.org/10.1007/s10803-019-03998-y>.
- Taylor, I., & Marrable, T. (2011). *Access to social care for adults with autistic spectrum conditions*. Retrieved from Social Care Institute for Excellence website: <http://rgdoi.net/10.13140/RG.2.1.4623.8248>
- Trammell, B., Wilczynski, S. M., Dale, B., & McIntosh, D. E. (2013). Assessment and differential diagnosis of comorbid conditions in adolescents and adults with autism spectrum disorders. *Psychology in the Schools*, 50(9), 936–946. <https://doi.org/10.1002/pits.21720>.
- Vogan, V., Lake, J. K., Tint, A., Weiss, J. A., & Lunskey, Y. (2017). Tracking health care service use and the experiences of adults with autism spectrum disorder without intellectual disability: A longitudinal study of service rates, barriers and satisfaction. *Disability and Health Journal*, 10(2), 264–270. <https://doi.org/10.1016/j.dhjo.2016.11.002>.
- Wigham, S., Rodgers, J., Berney, T., Le Couteur, A., Ingham, B., & Parr, J. R. (2018). Psychometric properties of questionnaires and diagnostic measures for autism spectrum disorders in adults: A systematic review. *Autism*, 23(2), 287–305. <https://doi.org/10.1177/1362361317748245>.
- World Health Organization. (2018, April 2). *Autism spectrum disorders*. Retrieved from World Health Organization website: <https://www.who.int/news-room/fact-sheets/detail/autism-spectrum-disorders>
- Zerbo, O., Massolo, M. L., Qian, Y., & Croen, L. A. (2015). A study of physician knowledge and experience with autism in adults in a large integrated healthcare system. *Journal of Autism and Developmental Disorders*, 45(12), 4002.

Basal Ganglia

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Synonyms

Striatum; Sub-cortical ganglia

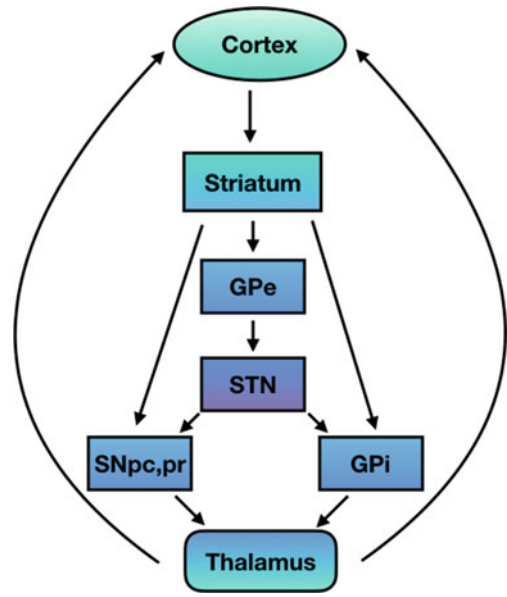
Definition

“Basal Ganglia” refers to a collection of sub-cortically (beneath the cortex) located nuclei in

the brain. Originally, these nuclei were collectively identified as a conduit for information to pass from cortical association regions to motor cortex for the initiation and control of movement. Today, the function of the basal ganglia is thought to include not only the shaping of motor responses, but also the integration of emotional, motivational, and cognitive information. The integration of such a wealth of information has been shown to contribute to motivated behaviors, habit formation, and motor learning. As part of these functions, the basal ganglia provides a locus of control for movement – simultaneously suppressing unwanted movement and enhancing desired movement (Mink 1996).

The subcortical structures typically included as part of the basal ganglia are: caudate nucleus and putamen (collectively called the striatum), nucleus accumbens (included in the ventral striatum), globus pallidus (both internal (GPi) and external (GPe) segments), substantia nigra (both pars compacta (SNpc) and pars reticulata (SNpr)), and the subthalamic nucleus (Groenewegen 2003). The interconnections between the nuclei of the basal ganglia allow for this collection of regions to act as a unit (Fig. 1). Together, they allow the impulses for movement and other behaviors that originate in the cortex to be refined towards a final output and expression.

The main input region of the basal ganglia is considered to be the striatum. The striatum receives cortical information that is topographically distributed based on the type of information. Emotional, associative, cognitive, and motor information from the cortex uniquely arrive in specific regions of the striatum, distributed in a ventral (bottom) to dorsal (top) manner, respectively. Information from the striatum then splits into two main pathways. The “direct” pathway channels information from the striatum to either the substantia nigra or the globus pallidus interna, the two major output regions of the basal ganglia. From these regions, information is then sent to the thalamus; the thalamus sends this, now processed, information back to the cortex. By contrast, the “indirect” pathway passes information first from the striatum to the globus pallidus externa and subthalamic nucleus, and then to the globus pallidus interna and substantia nigra



Basal Ganglia, Fig. 1 Schematic of the direct and indirect pathways of the basal ganglia. SNpc,pr, Substantia Nigra, pars compacta and pars reticulata; GPe, Globus pallidus external segment; GPi, Globus pallidus internal segment; STN, Subthalamic nucleus

for output to the thalamus. As with the direct pathway, the information sent to the thalamus is directed back to the cortex. The indirect and direct paths have distinct neurotransmitters and receptor profiles, and together form the “cortico-basal-ganglia-thalamo-cortical loops” that contain distributed emotional, motivational, associative, cognitive, and motor information (Alexander et al. 1986). These loops allow the impulses that originate in the cortex to be refined through the basal ganglia, and then sent back to cortex for a motor response output.

Abnormalities in the basal ganglia have been proposed to account for some of the symptoms of autism, including repetitive motor behaviors, decreased social interest/ability, and general motor dysfunction. The caudate nucleus, in particular, has been shown to have accelerated growth in children with autism, compared to typically developing children (Langen et al. 2014). This fits with other studies demonstrating an enlarged striatum in patients with autism. In animal models of autism, abnormalities in striatal interneurons, the small, local neurons that are key for regulating the neurons in the

striatum that project to other regions, are thought to underlie striatal pathology. Decreased anticipation and processing of social rewards, and rewards in general, along with decreased recruitment of the ventral striatum have been noted, identifying a potential mechanism for decreased sociability in autism. Given these striatal abnormalities, the question of whether motor abnormalities are primary, and the resulting symptoms of autism are merely secondary to those motor abnormalities, has been raised (Subramanian et al. 2017). Further research is needed to test this and to better understand the specific basal ganglia abnormalities in autism.

See Also

- Caudate Nucleus
- Dopamine
- Reward
- Striatum

References and Reading

- Alexander, G. E., DeLong, M. R., & Strick, P. L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience*, 9, 357–381. <https://doi.org/10.1146/annurev.ne.09.030186.002041>.
- Groenewegen, H. J. (2003). The basal ganglia and motor control. *Neural Plasticity*, 10(1–2), 107–120. <https://doi.org/10.1155/NP.2003.107>.
- Langen, M., Bos, D., Noordermeer, S. D., Nederveen, H., van Engeland, H., & Durston, S. (2014). Changes in the development of striatum are involved in repetitive behavior in autism. *Biological Psychiatry*, 76(5), 405–411. <https://doi.org/10.1016/j.biopsych.2013.08.013>.
- Mink, J. W. (1996). The basal ganglia: focused selection and inhibition of competing motor programs. *Progress in Neurobiology*, 50(4), 381–425. [https://doi.org/10.1016/S0301-0082\(96\)00042-1](https://doi.org/10.1016/S0301-0082(96)00042-1).
- Subramanian, K., Brandenburg, C., Orsati, F., Soghomonian, J. J., Hussman, J. P., & Blatt, G. J. (2017). Basal ganglia and autism – a translational perspective. *Autism Research*, 10(11), 1751–1775. <https://doi.org/10.1002/aur.1837>.

BASC-2

- Behavior Assessment System for Children, 2nd Edition

BASC-3

- BASC-III

BASC-III

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Synonyms

- BASC-3

Description

The Behavior Assessment System for Children, Third Edition (BASC-3) is a multimethod, multi-dimensional system that provides a comprehensive assessment of behavioral and emotional functioning in children, adolescents, and young adults from ages 2 to 25 (Reynolds and Kamphaus 2015). Its multiple components (hence multimethod) can be used individually or in combination and include: The Behavioral and Emotional Screening System (BESS), Teacher Rating Scales (TRS), Parent Rating Scales (PRS), Self-Report of Personality (SRP), Structured Developmental History (SDH), Student Observation System (SOS), Behavior Intervention Guide, Behavioral and Emotional Skill Building Guide, Flex Monitor, and Parenting Relationship Questionnaire (PRQ™). Used together, the information yielded from the components aid in clinical diagnosis and identifying educational supports and services (Reynolds and Kamphaus 2015). Emotional and behavioral functioning specific to a certain setting or context is provided when used individually. Additionally, the BASC-3 assesses positive and negative aspects of personality and behavior, making it multidimensional in nature (Altmann et al. 2018).

A detailed review of each component is provided in the manual (Reynolds and Kamphaus 2015) thus only a brief description of each follows:

- The TRS and PRS each have three age level forms (preschool, child, and adolescent) and measure adaptive and problem behavior in the school and home setting using a four-point Likert scale ranging from *Never* to *Almost Always*.
- The SRP captures respondents' feelings, behaviors, and self-perceptions and has forms for children, adolescents, and young adults (Reynolds and Kamphaus 2015).
- The SDH captures developmental information across a variety of domains that can aid in diagnosis and treatment.
- The SOS allows for a 15-min period of a student's behavior in a classroom to be recorded and evaluated.

The following descriptions are on the BASC-3 components that identify, monitor, and promote optimal behavioral and emotional functioning:

- The BESS is a quick screener that identifies behavioral and emotional problems in children and adolescents and can be filled out by teachers, parents, and students ages 8–18.
- The Behavior Intervention Guide is a compilation of evidence-based strategies for common emotional and behavioral problems that can be applied by a variety of behavioral professionals (Altmann et al. 2018).
- The Behavioral and Emotional Skill Building Guide provides small-group activities that promote and enhance positive behavioral and emotional skills (Reynolds and Kamphaus 2015).
- The Flex Monitor is an Internet-based tool that allows professionals to monitor and track a customizable set of emotions or behaviors which can be compared to a nationally representative population sample, in order to measure behavioral change over a period of time (Reynolds and Kamphaus 2015).
- The PRQ captures the parent's perceived relationship between themselves and the child, and

assesses dimensions such as attachment, involvement, parenting style, stress, and school satisfaction (Reynolds and Kamphaus 2015).

Historical Background

In 1992, after seven years of development, standardization, and validation, the Behavior Assessment System for Children (BASC) was published due to the need for a psychometrically sound and integrated assessment of child and adolescent emotions and behaviors, appropriate for multiple settings (Reynolds and Kamphaus 2015). In 2004, the BASC-2 was released and quickly became the most commonly used set of behavior rating scales in public schools in the USA and internationally (Reynolds and Kamphaus 2015). In 2015, the third edition was published, with the addition of electronic administration and scoring, improved interpretive reporting, and Flex Monitor (see above in “[Description](#)” section) (Reynolds and Kamphaus 2015).

Psychometric Data

The TRS, PRS, and SRP were designed based on a combination of theory and empirical data. Over 120 teachers, 100 parents, and 400 students were surveyed about negative and positive behaviors observed in home and classroom settings (Reynolds and Kamphaus 2015). Approximately 90 negative and 50 positive behaviors were identified and compared to the BASC-2 item pool, and around 10–15 new items were written for the BASC-3 TRS, PSR, and SRP standardization forms (Reynolds and Kamphaus 2015). To ensure each domain had content that was adequate and relevant, a review of the BASC-2 forms, the newly written items, and items on the original BASC served as the basis of the standardized form items (Reynolds and Kamphaus 2015). General norms were derived from data collected from a large and representative sample of children across the United States, while children with a diagnosis of one or more emotional or behavioral problems ages 4 through 18 comprised the clinical norms sample (Reynolds and Kamphaus 2015).

The BASC-3 Manual provides evidence of reliability by reporting internal consistency, test-retest reliability, and interrater reliability for the TRS, PRS, and SRP (Reynolds and Kamphaus 2015). Overall, the scales and composites were found to have reliability coefficients of 0.80 and above, thus demonstrating the ability to reliably estimate behavior for diagnostic and treatment planning. Extensive validity evidence is also provided in the BASC-3 Manual for each scale including scale intercorrelations and factor analyses, correlations with other rating scales, and score profiles of groups of children with particular clinical diagnoses or educational classifications (Reynolds and Kamphaus 2015). Additionally, the BASC-3 contains validity indices that allow for the detection of untruthful responding, carelessness, extreme responding, or other validity threats (Reynolds and Kamphaus 2015).

Clinical Uses

The BASC-3 and its components (TRS, PRS, SRP, SDH, and SOS) can be used in a variety of settings and thus has wide applications including aiding in clinical diagnosis, educational classification, manifestation determination, assessment of individuals with limitations of vision and hearing, program evaluation, forensic evaluation, and research (Reynolds and Kamphaus 2015). In regards to clinical diagnosis, the BASC-3 assesses symptoms found in the DSM-5 for disorders in childhood or adolescence. The rating scales are also sensitive to a variety of classroom problems, which help differentiate academic difficulties from behavioral problems. They also help assess severe emotional disturbance as called for by IDEA (2004) (Reynolds and Kamphaus 2015). The BASC-3 is effective in determining the origins of behavior and can be used by experienced individuals to evaluate children with sensory impairments (Reynolds and Kamphaus 2015). Lastly, the BASC-3 has been found to be helpful in the evaluation of children's progress in programs and/or their response to interventions when administered over multiple

time points, as well as in legal or forensic settings given its psychometric properties and ability to detect dissimulation (Reynolds and Kamphaus 2015).

See Also

- [Behavior Assessment System for Children, 2nd Edition](#)

References and Reading

- Altmann, R. A., Reynolds, C. R., Kamphaus, R. W., & Vannest, K. J. (2018). Behavior assessment system for children. In J. Kreutzer et al. (Eds.), *Encyclopedia of clinical neuropsychology* (3rd ed.). New York: Springer.
- Individuals with Disabilities Education Act, 20 U.S.C.A. § 1400 et seq. (2004)
- Reynolds, C. R., & Kamphaus, R. W. (2015). *Behavior assessment system for children* (3rd ed.). Bloomington: NCS Pearson. (BASC-3).

Baseline

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Definition

An assessment of abilities that serves as an anchor for monitoring subsequent change over time when combined with follow-up assessments. A baseline assessment may occur prior to a child entering school, or, if a child is enrolled in an intervention study, prior to administering the treatment. A baseline assessment may involve more than one assessment point, to determine the stability of a behavior prior to introducing an experimental manipulation (e.g., an intervention designed to change the behavior assessed during the baseline period). Later assessments can be compared to the baseline assessment, so that symptoms or abilities may be tracked over time, and improvements or deterioration in abilities may be noted. This may

be especially helpful for developmental disorders such as autism, in which the symptoms and their severity can change dramatically over time. In cases in which deterioration of skills occurs, the combination of a thorough baseline assessment and appropriate follow-up assessments can help identify specific skills that can be targeted in treatment. Baseline assessments might include measures of language and communication, social skills, self-help skills, play, and IQ.

See Also

- [Course of Development](#)
- [Longitudinal Research in Autism](#)
- [Outcome Studies](#)

References and Reading

- Constantino, J. N., Abbacchi, A. M., Lavesser, P. D., Reed, H., Givens, L., Chiang, L., et al. (2009). Developmental course of autistic social impairment in males. *Development and Psychopathology*, 21, 127–138.
- Gordon, K., Pasco, G., McElduff, F., Wade, A., Howlin, P., & Charman, T. (2011). A communication-based intervention for nonverbal children with autism: What changes? Who benefits? *Journal of Consulting and Clinical Psychology*, 79, 447–457.

Bayley Scales of Infants Development-II

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Synonyms

[Bayley-III](#)

Description

The Bayley-III is a standardized developmental assessment that evaluates the functioning of infants and young children from 1 month to

42 months of age. It is designed to identify children with developmental delays and aid in intervention planning. The test assesses multiple developmental domains, including cognitive, language (both receptive and expressive), motor (both fine and gross), as well as social emotional and adaptive behavior. The cognitive, language, and motor scales are based primarily on direct assessment, whereas the social-emotional and adaptive behavior scales are caregiver questionnaires. Scaled scores are provided for each subtest, with composite scores and percentile ranks for each overall scale. Developmental age equivalents are also provided for cognitive, language, and motor subtests. Growth scores can also be calculated to evaluate a child's growth over time for cognitive, language, and motor subtests.

Historical Background

The Bayley Scales of Infant Development (BSID) were first published in 1969, with revisions in 1993 (BSID-II) and 2006 (Bayley-III). In its most recent edition, the test was updated to reflect updates in the field of child development research, including information processing and preverbal intelligence. However, the Bayley-III still retains its focus on more classic themes in child development (e.g., Piaget, Vygotsky). Additionally, many items from the BSID-II were removed or changed and new items were developed.

Psychometric Data

Normative data for the cognitive, language, and motor scales was collected from 1700 children aged 1 month to 42 months (with 100 individuals in 17 separate age groups) and closely reflected the 2000 US Census in terms of parental education level, race/ethnicity, and geographic region. Only children born between 36 and 42 weeks were included. Children with mental, physical, or behavioral difficulties constituted about 10% of the total sample. The social-emotional scale was normed using 456 children, and the adaptive behavior scale included 1350 children.

Clinical Uses

The Bayley-III is designed to be used to identify children with developmental delays. It is recommended that the Bayley-III be administered by an individual with formal graduate or professional training in developmental assessment. While it is possible for a psychometrician to administer the Bayley-III, test interpretation should occur by an individual with appropriate training to interpret test data.

See Also

- [Developmental Milestones](#)

References and Reading

- Bayley, N. (1993). *Bayley scales of infant and development-second edition*. San Antonio: The Psychological Corporation.
- Bayley, N. (2006a). *Bayley scales of infant and toddler development-third edition: Administration manual*. San Antonio: Harcourt Assessment.
- Bayley, N. (2006b). *Bayley scales of infant and toddler development-third edition: Technical manual*. San Antonio: Harcourt Assessment.

Bayley-III

- [Bayley Scales of Infants Development-II](#)

BCaBA

- [Board Certified Associate Behavior Analyst](#)

BCBA

- [Board Certified Associate Behavior Analyst](#)

BCBA-D

- [Board Certified Associate Behavior Analyst](#)

BDQ

- [Behavior Development Questionnaire](#)
- [Behavioral Development Questionnaire](#)

Beate Hermelin

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Division of Biosciences, Institute of Cognitive Neuroscience UCL, London, UK

Name and Degrees

Beate Hermelin, BA, PhD

Major Appointments (Institution, Location, Dates)

Medical Research Council, London, ca 1960–ca 1985.

Major Honors and Awards

Queen Elizabeth II Silver Jubilee Medal

Landmark Clinical, Scientific, and Professional Contributions

Beate Hermelin was a strikingly original experimental psychologist with an unconventional career and unconventional thinking. It is impossible to talk of her work without also talking of the work of Neil O'Connor, as almost all her publications include both names. Hermelin and O'Connor conducted a series of groundbreaking

experimental studies, which tried to explain and interpret the mind of the autistic child. This work, carried out during the 1960s, culminated in a monograph published in 1970. They were the first to systematically ask questions about the cognitive abilities of severely intellectually impaired children, who had previously been considered untestable and ineducable. In their ingeniously and elegantly designed experiments, they compared learning-disabled children with and without autism, and typically developing children. All were matched for mental age, not chronological age, using a range of cognitive tests, e.g., of memory, vocabulary, and abstract reasoning. This methodology was revolutionary at the time and made visible a variety of cognitive differences, both strengths and weaknesses that could be specifically attributed to autism. Hermelin and O'Connor also studied groups of children with specific disabilities in seeing and hearing, again comparing them with autistic and non-autistic children. In their final working period, they embarked on another pioneering series of experiments into the basis of the previously unexplained phenomenon of savant talent.

O'Connor and Hermelin were the first to apply ideas and methods derived from information processing to the study of autism and are therefore rightly considered the grandparents of modern cognitive theories of autism. They also applied paradigms that were being developed at the same time by neuropsychologists who discovered dissociations of cognitive processes through the study of brain lesions. Thus, Hermelin and O'Connor overcame the limits of purely behaviorist paradigms and were able to probe the neurocognitive causes of the behavioral phenomena of autism. Their legacy is the identification of specific information processing abnormalities over and above cognitive disabilities that are general consequences of atypical brain development.

Short Biography

Beate Hermelin was born August 7, 1919, in Berlin, the daughter of the German Jewish Lawyer

Julius Fliess, who was the brother of Wilhelm Fliess, friend of Sigmund Freud. In 1939 she escaped to Palestine with a boyfriend, while her family survived the war in Switzerland. In 1948 Beate Hermelin arrived with her filmmaker husband, Rolf Hermelin, in London. Here she attended occasional lectures and was recognized as an exceptional student by clinical psychologist Alan Clarke (1922–2011), an authority on intellectual disability, later professor and vice chancellor at Hull University. He encouraged her to read experimental psychology at Reading University and to carry out a PhD on learning and memory in severely learning-disabled children at London University's Institute of Psychiatry. This was when she started her lifelong collaboration with experimental psychologist Neil O'Connor (1917–1997). The equality of their scientific contributions was acknowledged and highlighted by the strict rotation of authorship on their publications. Hermelin was a member of the Medical Research Council Scientific Staff for all of her professional life. After her retirement in ca 1985, she became an honorary professor at Goldsmith College, London University. In 2001 she published a book entitled *Bright Splinters of the Mind*, which, besides a summary of the work on savant talent, also includes autobiographical observations and reminiscences. Beate Hermelin died January 14, 2007.

Despite her pioneering and influential work, and despite her glamorous appearance and her consummate skill in giving inspiring talks, Beate Hermelin eschewed the limelight. Instead she put her lively energy into mentoring her students, many of whom went on to forge distinguished careers in autism research. Among them are Uta Frith, Peter Hobson, Tony Attwood, and Pam Heaton.

References and Reading

- Hermelin, B. (2001). *Bright splinters of the mind. A personal story of research with autistic savants*. London: Jessica Kingsley.
- Hermelin, B., & O'Connor, N. (1965). Visual imperception in psychotic children. *British Journal of Psychology*, 56(4), 455–460.

- Hermelin, B., & O'Connor, N. (1968). Measures of the occipital alpha rhythm in normal, subnormal and autistic children. *British Journal of Psychiatry*, 114, 603–610.
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. Oxford: Pergamon.
- Hermelin, B., & O'Connor, N. (1975). The recall of digits by normal, deaf and autistic children. *British Journal of Psychology*, 66, 203–209.
- Hermelin, B., & O'Connor, N. (1986). Idiot savant calendrical calculators: Rules and regularities. *Psychological Medicine*, 16, 885–893.
- Hermelin, B., & O'Connor, N. (1990a). Art and accuracy: The drawing ability of idiot-savants. *Journal of Child Psychology and Psychiatry*, 31, 217–228.
- Hermelin, B., & O'Connor, N. (1990b). Factors and primes: A specific numerical ability. *Psychological Medicine*, 20, 163–169.
- O'Connor, N., & Hermelin, B. (1963). *Speech and thought in severe subnormality*. Oxford: Pergamon.
- O'Connor, N., & Hermelin, B. (1967). The selective visual attention of psychotic children. *Journal of Child Psychology and Psychiatry*, 8, 167–179.
- O'Connor, N., & Hermelin, B. (1978). *Seeing and hearing and space and time*. Oxford: Pergamon.
- O'Connor, N., & Hermelin, B. (1988). Low intelligence and special abilities. *Journal of Child Psychology and Psychiatry*, 29, 391–396.
- O'Connor, N., & Hermelin, B. (1989). The memory structure of autistic idiot-savant mnemonists. *British Journal of Psychology*, 80, 97–111.
- O'Connor, N., & Hermelin, B. (1994). Two autistic savant readers. *Journal of Autism and Developmental Disorders*, 24, 501–515.

Sample References

- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.). Washington, DC: APA Press.
- Bachevalier, J. (1996). Brief report: Medical temporal love and autism: A putative animal model in primates. *Journal of Autism and Developmental Disorders*, 26(2), 217–220.
- Howlin, P. (2005). Outcomes in autism spectrum disorders. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. I, pp. 640–649). Hoboken: Wiley.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Mesibov, G. B., Shea, V., & Schopler, E. (2004). *The TEACCH approach to autism spectrum disorders*. New York: Springer.

Bed-Wetting

- [Enuresis](#)

Beery VMI

- [Beery-Buktenica Developmental Test of Visual-Motor Integration](#)
- [Visual-Motor Integration, Developmental \(VMI\) Test](#)

Beery VMI Motor Coordination Test

- [Visual-Motor Integration, Developmental \(VMI\) Test](#)

Beery VMI Visual Perception Test

- [Visual-Motor Integration, Developmental \(VMI\) Test](#)

Beery-Buktenica Developmental Test of Visual-Motor Integration

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Synonyms

[Beery VMI](#); [Developmental test of visual-motor integration](#); [VMI](#)

Description

The Beery-Buktenica Developmental Test of Visual-Motor Integration (Beery VMI; Beery

and Beery 2010) is a test of visual-motor coordination. Visual-motor integration is “the degree to which visual perception and finger-hand movements are well coordinated” (Beery 1997, p. 19). This paper-and-pencil test involves examinees copying increasingly complex designs. It is designed to assess visual-motor integration, visual perception, and motor coordination skills and is designed to indicate the need for support services for problems in one or more of these areas.

The Beery VMI includes Short and Full Format tests and supplemental Visual Perception and Motor Coordination tests. None of the Beery VMI tests is timed. The Short and Full Format tests involve the examinee copying increasingly complex designs with a pencil without an eraser. Both the Short and Full Format tests can be administered to individuals and groups (e.g., kindergarten class). The Full Format test contains 30 items and is appropriate for use with children (ages 2–18) and adults (ages 19–100). The items increase in complexity from an imitated mark to a three-dimensional star. The Short Format test contains 21 items and is designed for use with children ages 2–7 years old. It takes about 10–15 min to administer the Short Format or Full Format test. The examiner of the Beery VMI must have Examiner B qualifications, which indicates that examiners must have a graduate degree in psychology or a related field or equivalent training to complete the assessment.

The Full and Short Format test form pages contain a table with six blocks; the top three blocks provide examples of the drawing shapes that the examinee is to copy in the corresponding block below. The blocks represent the boundaries within which the examinee is to draw the design. The Visual Perception supplemental test requires examinees to identify a target design among choices, and the Motor Coordination supplemental test requires examinees to trace a geometric shape with a dashed outline using a pencil without an eraser. Each supplemental test takes about 5 min to complete in addition to administration time for the Short and Full Format tests.

The first three items of the Full Format test, which require scribbling, are designed for use with very young children. For the next three

items, the examiner models drawing the first three shapes in the upper blocks of the test form; after each model, the examinee copies the same shape in the lower blocks of the test form. For examinees aged 19–100, testing starts with item 7 and the examinee copies the printed shape, such as horizontal line, vertical-horizontal cross, or square, in the lower blocks of the test form. Testing is discontinued when an examinee draws three items incorrectly in a row.

Because the Beery VMI is a brief assessment, testing typically can be completed in one session. The Beery VMI is a paper-and-pencil test that must be hand-scored by the examiner. Examinees’ drawings are scored 1 or 0 based on the degree to which each drawing met relevant criteria. Accurate scoring requires the use of a protractor to make judgments about accuracy of angles, etc. The examiner’s manual contains many scoring examples and comments about design attempts, which assist with scoring and interpretation of the examinee’s drawings. A total raw score is obtained by adding the number of designs that were scored as “pass.” The examiner’s manual contains tables to convert raw scores into standard scores, percentiles, and age equivalent scores.

Assessment materials include an examiner’s manual, entitled *Beery VMI With Supplemental Developmental Tests of Visual Perception and Motor Coordination For Children and Adults*, and four different scoring forms: Full, Short, Visual Perception, and Motor Coordination. The examiner’s manual contains administration and scoring instructions and age-specific norms, including about 600 age-specific norms for children from birth to age 6. The examiner’s manual also includes teaching suggestions for improving visual-motor coordination skills.

The authors of the Beery VMI, Keith and Natasha Beery, have also produced additional materials beyond the examiner’s manual to supplement the assessment and aid in the development of related skills. The materials include the following:

- (a) Developmental Teaching Activities: a resource that contains 250+ activities that

parents and teachers can use with young children (birth to age 6) to support the development of skills useful for art, academics, and athletic activities

- (b) My Book of Shapes: a resource that contains 100 geometric paper-and-pencil activities that parents and teachers can use with young children (preschool and kindergarten) to support the development of skills, especially useful for supporting visual-motor skills necessary for early literacy and early numeracy development
- (c) My Book of Letters and Numbers: a resource that contains 100 activities for use with children in the second half of their kindergarten year to support the development of skills necessary for literacy and numeracy activities
- (d) Developmental Wall Chart for Visual-Motor Integration: a wall chart with information about development of gross and fine motor, visual, and visual-motor skills for young children (birth to age 6)
- (e) Beery VMI Stepping Stones Parent Checklist: a checklist created for parents to document their children's progress from preschool through early elementary age

Historical Background

The Beery VMI was first developed in 1967 and is currently in its sixth edition. The most recent normative data was collected in 2010 for children and 2006 for adults. The current version of the assessment looks very similar to its original version, with four major changes as part of past revisions. First, the Visual Perception and the Motor Coordination supplemental tests were added in 1997. The addition of these supplemental tests allows the examiner to obtain additional information to identify specific areas of skill weakness. Second, in 2004, the number of items on the Full Format was increased from the original number of 24–30. Third, the norms were expanded to include a wider age range in 2004 and 2006. Finally, scoring was based on a scale of 1–4 between 1989 and 1996; however, the original and more recent versions use a scoring system with only 1 point possible per item.

Psychometric Data

The Beery VMI “is regarded as one of the most valid and reliable instruments for the assessment of visual-motor integration” (Kulp and Sortor 2003, p. 313) and is used internationally. Standardization studies were conducted on the Beery VMI. The most recent standardization sample for children occurred in 2010 using a nationally representative group of 1,737 children between the ages of 2 and 18 years old. The most recent standardization sample for adults occurred in 2006 using a nationally representative sample 1,021 adults ages 19–100. For more information about psychometric data, the reader is encouraged to refer to the *Encyclopedia of Autism Spectrum Disorders* entry entitled “Visual-Motor Integration, Developmental (VMI) test” (authored by Dr. Ted Brown) or *The Beery-Buktenica Developmental Test of Visual-Motor Integration (Beery VMI) with Supplemental Developmental Tests of Visual Perception and Motor Coordination and Stepping Stones Age Norms: Administration, Scoring and Teaching Manual* (Beery and Beery 2010).

Clinical Uses

The Beery VMI is used in a number of settings and by a variety of professionals to assess the visual-motor integration skills of a wide range of people. Settings of use include schools, hospitals, and clinics; professionals who use the Beery VMI include psychologists, occupational therapists, neurologists, etc. Given the number of disabilities and disorders that include symptoms of visual-motor, visual-perceptual, and motor coordination difficulties, the Beery VMI is applicable for use with many people. Autism spectrum disorder (ASD) is one such condition that typically involves motor and visual deficits (American Psychiatric Association [APA] 2000; Coulter 2009). An advantage of using the Beery VMI for assessment of children with ASD is its nonverbal design, which helps to reduce or eliminate language confounds observed with other psychoeducational assessments.

The Beery VMI is a useful tool as part of psychoeducational evaluations because the Beery VMI provides information about children's writing readiness skills and indicates potential deficits in visual-motor functioning that may require intervention, such as occupational therapy. The supplemental tests help teams identify specific visual-perceptual or motor coordination deficits that might not otherwise be identified on the Short and Full Format tests (Kulp and Sortor 2003). Also, given the importance of early identification of developmental delays, the Beery VMI can be used to identify young children's motor coordination and/or visual perception delays. The Beery VMI is especially helpful in early intervention settings because it provides standard scores for children as young as 2 years old, which is rare among psychological assessments. Additionally, the Beery VMI is useful in educational settings because it can be used as part of universal screening, which involves assessing all children (in a class or school) to determine specific needs. Because it is acceptable for use with groups, the Beery VMI can provide educators with information about the skills of children in an entire class in a short amount of time. There is, however, some evidence that the Beery VMI falls short in identifying older children with handwriting dysfunction (Goyen and Duff 2005) despite its standing as a robust instrument in the use of identifying visual-motor integration. As such, clinicians may be cautioned to not rely solely on the Beery VMI when making decisions about older children's handwriting needs. Another limitation of using the Beery VMI with children with ASD and, potentially, other disabilities is the requirement of the examinee to imitate the examiner and/or printed designs. Individuals who lack adequate attention or memory skills might produce work and earn scores on the Beery VMI that reflect a low estimate of their true visual-motor integration ability.

The Beery VMI boasts solid predictive validity. That is, VMI scores of children in kindergarten "predicted with 85% accuracy those children who had reading problems seven years later" (Brown et al. 2009, p. 395). There is evidence

of associations between children's Beery VMI scores and math and reading performance (Sortor and Kulp 2003). The Beery VMI was created to be compatible with the sequential development of children's skills. Beyond the use of identification of children's needs, the instrument is also designed to support the advance of research. The Beery VMI is described as culture-free and nonverbal, making it appropriate for use with a range of individuals, evidenced by the use of geometric forms instead of letters or numbers. However, recent evidence has called into question whether the Beery VMI is truly culture-free. Specifically, research conducted with a South African preschool population suggested differences in scores between children of socioeconomic status (SES) and race (Dunn et al. 2006), with White children and children of higher SES performing better than their counterparts.

See Also

- [Autism](#)
- [Bender Visual-Motor Gestalt Test II](#)
- [Bruininks-Oseretsky Test of Motor Proficiency](#)
- [Motor Control](#)
- [Motor Planning](#)
- [Occupational Therapy \(OT\)](#)
- [Peabody Developmental Motor Scales \(PDMS\)](#)
- [Psychologist](#)
- [Spectrum/Continuum of Autism](#)
- [Visual-Motor Integration, Developmental \(VMI\) Test](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Rev.). Washington, DC: Author.
- Beery, K. E. (1997). *The Beery-Buktenica VMI: Developmental test of visual-motor integration with supplemental developmental tests of visual perception and motor coordination: administration, scoring, and teaching manual* (4th ed.). Parsippany: Modern Curriculum.
- Beery, K. E. (2006). *The Beery-Buktenica developmental test of visual-motor integration: Beery VMI* (5th ed.). New York: MHS.

- Beery, K. E., & Beery, N. A. (2006). *The Beery-Buktenica developmental test of visual motor integration administration, scoring, and teaching manual*. Bloomington: NCS Pearson.
- Beery, K. E., & Beery, N. A. (2010). *The Beery-Buktenica developmental test of visual-motor integration (Beery VMI) with supplemental developmental tests of visual perception and motor coordination and stepping stones age norms: Administration, scoring and teaching manual*. Minneapolis: NCS Pearson.
- Beery, K. E., Buktenica, N. A., & Beery, N. A. (2004). *The Beery-Buktenica developmental test of visual motor integration* (5th ed.). Bloomington: NCS Pearson.
- Brown, T., Unsworth, C., & Lyons, C. (2009). An evaluation of the construct validity of the developmental test of visual-motor integration using the Rasch measurement model. *Australian Occupational Therapy Journal*, 56(6), 393–402.
- Coulter, R. A. (2009). Understanding the visual symptoms of individuals with autism spectrum disorder (ASD). *Optometry & Vision Development*, 40(3), 164–175.
- Cummings, J. A., Hoida, J. A., Machek, G. R., & Nelson, J. M. (2003). Visual-motor assessment of children. In C. R. Reynolds, R. W. Kamphaus, & C. N. Hendry (Eds.), *Handbook of psychological and educational assessment of children: Intelligence, aptitude, and achievement* (2nd ed.). New York: Guilford Press.
- Dunn, M., Loxton, H., & Naidoo, A. (2006). Correlations of scores on the developmental test of visual-motor integration and copying test in a South African multi-ethnic preschool sample. *Perceptual and Motor Skills*, 103(3), 951–958.
- Goyen, T., & Duff, S. (2005). Discriminant validity of the developmental test of visual-motor integration in relation to children with handwriting dysfunction. *Australian Occupational Therapy Journal*, 52(2), 109–115.
- Kulp, M. T., & Sortor, J. M. (2003). Clinical value of the Beery visual-motor integration supplemental tests of visual perception and motor coordination. *Optometry and Vision Science*, 80(4), 312–315.
- Morr, D., & Corimak, S. (2002). Predicting handwriting performance of early elementary students with the developmental test of visual-motor integration. *Perceptual and Motor Skills*, 95, 661–669.
- Pearson Assessments, Inc. (n.d.). Beery VMI product description. Retrieved February 25, 2011, from <http://www.pearsonassessments.com/haiweb/cultures/en-us/productdetail.htm?pid=pag105&mode=summary>
- Sortor, J. M., & Kulp, M. T. (2003). Are the results of the Beery-Buktenica developmental test of visual-motor integration and its subtests related to achievement test scores? *Optometry and Vision Science*, 80(11), 758–763.
- Volker, M., Lopata, C., Vujnovic, R., Smerbeck, A., Toomey, J., Rodgers, J., et al. (2010). Comparison of the Bender Gestalt-II and VMI-V in samples of typical children and children with high-functioning autism spectrum disorders. *Journal of Psychoeducational Assessment*, 28(3), 187–200.

Behavior

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Definition

Behavior is an action or reaction exhibited by a human or animal in response to stimuli. Stimuli may be external and/or internal. Manipulating stimuli is the way to change any behavior. Specific target behavior chosen for modification must be observable and measurable.

Quite simply, behavior is anything a living organism can do.

References and Reading

- Cooper, J., Heron, T., & Heward, W. (2007). *Applied behavior analysis* (2nd ed.). Columbus: Merrill/Prenice Hall.

Behavior Analysis

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Synonyms

Behavioral specialist

Definition

Behavior analysis is, “the experimental investigation of variables that influence the behavior of

any living organism” (Mayer et al. 2012, p. 6). Often, it is used interchangeably with applied behavior analysis. While there is a relationship between the two, they are not synonymous. There are three branches of the science of behavior analysis – behaviorism, experimental analysis of behavior (EAB), and applied behavior analysis (ABA) (Cooper et al. 2007). ABA, therefore, is one branch of the science of behavior analysis. In addition to these three branches of the science, there is also a focus on practice guided by behavior analysis.

Historical Background

The question of why people behave as they do has been answered in many ways. Over the centuries, many different belief systems have evolved to explain human behavior, including religion, mythology, astrology, and cultural practices. Psychologists, whose focus is on behavior, have developed varying perspectives and theories regarding the causes of behavior, including structuralism and psychoanalysis.

Eventually, there was an attempt to understand whether human behavior might be investigated using the methods of science. At first, Watson employed what the methods of what he called “methodological behaviorism” (Mayer et al. 2012), relying on direct observation and careful manipulation of variables to determine their influence (if any) on behavior. The unique aspect of Watson’s work was to study behavior as a strict scientist, following the strict rules of scientific process. Other psychologists (e.g., Pavlov, Skinner) followed Watson, adhering strictly to the application of the scientific method to study aspects of the human condition. Through this perspective of science, the field has advanced to the point of acknowledging that human behavior follows the laws of nature as do other phenomena. Behavior analysis has remained true to embracing the role of science in the study of human behavior and that is its unique contribution to psychology and education.

The branch of behavior analysis that later became known as ABA can be traced to a publication by Ayllon and Michael (1959), in which

personnel in a mental hospital were trained to use behavior strategies to modify the behaviors of psychotic residents. Pioneers in the 1960s and 1970s made great inroads to changing behavior despite poor funding, the reluctance of the scientific community to publish their work, and the lack of evidence-based strategies to influence their lines of research. In the field of education, exciting results were found with the use of contingent teacher attention (Hall et al. 1968), token economies (Birnbrauer et al. 1965), and programmed instruction (Bijou et al. 1966).

In 1968, the *Journal of Applied Behavior Analysis* was first published. This has been the premiere journal of the discipline since that time. The journal focuses on the use of within-subject designs to experimentally evaluate the effects of treatments and to experimentally identify controlling relationships between variables. For many years, such within-subject design effects were considered less important than group design effects (which are commonly done, e.g., in psychology). In recent years, there has been some progress in this area, as repeated demonstrations in multiple single case designs are now being recognized as scientific evidence.

Also in 1968, the seminal article on the dimensions of ABA was published (Baer et al. 1968). In this article, the authors outline seven critical elements of ABA that define interventions that are behavior analytic: applied, behavioral, analytic, technological, conceptually systematic, and effective.

Current Knowledge

Behaviorism is the theoretical and philosophical branch of the science. Behaviorists analyze at conceptual levels and create theoretical accounts of behavior that are consistent with existing data. Behaviorists may also outline areas in which empirical data are absent and may suggest ways to rectify gaps in our existing knowledge. Behaviorists inspire much of the work of the other branches, and they maintain the focus of the science on the theoretical underpinnings and philosophical stances.

The experimental analysis of behavior (EAB) is the basic science branch. These individuals design and conduct experiments in basic science. They conduct experiments in laboratories and other highly controlled environments. They may use human or nonhuman participants. In their work, they may discover and clarify basic principles of behavior, and they may identify functional relations between variables. EAB is also the branch that creates many of the questions for both ABA and EAB to pursue.

Applied behavior analysis is the branch of behavior analysis in which the tactics derived from the principles of behavior are applied to improve socially significant behavior, and experimentation is used to identify the variables responsible for the improvement in behavior (Cooper et al. 2007). Applied behavior analysts conduct experiments that are designed to identify relations between socially significant behavior and its controlling variables. They do this to add to the technology of humane and effective behavior change procedures.

All three branches of the science are essential, and they influence one another. Research is an essential component to the advancement of the science. Both basic and applied researches help to refine concepts and develop effective procedures/interventions.

The main methodologies utilized within behavior analysis are within-subject designs. These designs experimentally prove the controlling relationships between independent and dependent variables and rule out extraneous explanations. Several commonly used ones are frequently used in behavioral publications: the reversal design, the multiple baseline design, the changing criterion design, and the alternating treatments design.

The Reversal Design: In the reversal design, data on the target behavior are collected prior to intervention (condition A), the intervention is applied (B), the intervention is withdrawn (A), and the intervention is reapplied (B). This is referred to as an ABAB design. The impact of an intervention is examined for its controlling influence. Is it the variable responsible for the change? Does the behavior revert back to pretreatment

levels in the absence of treatment? In this way, one can be more confident that it is the treatment itself effecting change. Variations of the design exist (e.g., ABA, BABA). However, all of the reversal designs use this basic premise of reversing the effect of the intervention by withdrawing treatment.

The Multiple Baseline Design: In this design, the intervention is applied in sequential phases across participants, behaviors, or settings. Essentially, the researcher looks for replication of effect. If an intervention is first applied to one student with good impact, can it then be extended to others? Similarly, can it be applied across settings? If an intervention successfully taught one skill, can it be extended to another? In this way, the confidence about the utility of this intervention in this context increases.

The Changing Criterion Design: In the changing criterion design, the criterion for behavioral effect continually increases. In this design, behaviors may be changed gradually, with increases in expectations shifting over time.

The Alternating Treatments Design: In this design, different approaches or interventions can be directly compared. The level of the target behavior can be compared in different conditions. In other words, the dependent variable is compared in different levels or variations of an independent variable. If there is a question about whether a particular independent variable will make a difference, it can be compared to no treatment. If there is a question about the level of intervention to apply (e.g., # minutes of an activity, richness of reinforcement ratio), the question can be experimentally answered to guide treatment.

In all behavioral research, as well as in applied work inspired by behavioral research, clinicians remain committed to the identification of functional relationships. When appropriate, they utilize within subject designs. This is especially true when they are evaluating the impact of a more experimental treatment. At the level of the individual, the behavior analyst always seeks to demonstrate functional relationships, to identify variables responsible for change.

The delivery of behavior analytic services is a separate domain, as noted above, but is closely

linked to this third branch of the science of behavior analysis, ABA. Practitioners design interventions and evaluate their impact. They use procedures that are derived from basic research and that have been shown to produce socially significant outcomes by applied researchers. In recent years, this application of the science has become increasingly prominent. The effectiveness of ABA in effecting change has been significant, especially in certain populations, such as individuals with autism. This has created a unique and wonderful opportunity for ABA to receive attention in the broader public arena. It has also created threats to the purity of the science, to the portrayal of the science, and to the public's understanding of the core characteristics and commitments of ABA. Misconceptions and misrepresentations abound, and the correction of these misconceptions and misrepresentations has become imperative.

Many myths and misconceptions exist about behavior analysis and, in particular, about behavior analysis in application to clinical practice. In general, the field is often presented as reductionistic and is often contrasted with more humanistic approaches that have more broad appeal. This is a major challenge to the science of behavior, as it impedes the ability to offer these powerful interventions to those most in need of them. Professionals within the applied arena often struggle with core misunderstandings of the science and its applications. In addition, they often are presented with clinical contexts that are ethically challenging. For example, many behaviorally based clinical programs are diluted, combined with other nonverified approaches or delivered at a level of intensity not associated with likely success. There is a need for all branches of the science to promote the accurate and current state of the field, in research and clinical arenas.

Applied refers to the commitment of ABA to improving the lives of those they serve. Behavior analysts seek to effect changes that are socially significant. To achieve this, they select behaviors that are of importance to the individual and to their family. They also assess whether changes have made real-world differences in the lives of the individuals. Many misconceptions exist about

this particular dimension, as many people think of ABA as intervening on all behaviors or as being focused on behavior reduction in the absence of an analysis of importance. In the early days of ABA, when impact was new, the focus was on using the science to reduce intractable behaviors. However, the science has evolved over many decades and is now very focused on the importance of targeting behaviors that make a real-world difference.

Behavioral refers to the focus on behavior. Behaviors targeted must be those in need of improvement, must be measurable, and must be verified to have changed through objective means. This guideline emphasizes the need to target and measure behaviors in the natural setting of the individual and commits the behavior analyst to using behavioral techniques for all intervention and measurement. It distinguishes ABA from other service providing disciplines that often speak in generalities and in global terms. The commitment to the science requires that all behaviors must be measurable, operationally defined, and thoroughly evaluated objectively for change that is empirically verifiable.

Analytic refers to the demonstration of a relationship between the manipulated variables and the documented behavioral effects. Experimenters must be able to control the occurrence and non-occurrence of the behavior. Behavior analysts value this dimension very highly and work to prove that such a functional relation exists between the independent variable (variable that was manipulated) and the dependent variable (behaviors targeted). The behavior analyst is never content with change alone; there must be an understanding of WHY the behavior changed, of the variables responsible for the change.

Technological refers to replicability. Behavior analysts use precision, detail, and clarity in describing their interventions so that others can replicate their work. Behavioral procedures must be replicable to be teachable to others. From both a research and clinical perspective, then, the technological dimension is essential to behavior analysis. This is another hallmark characteristic of science. If a technique is not technological, it cannot be subjected to a test. It then becomes

analogous to anecdotal reports, and it becomes vulnerable to exaggeration and false claims. The requirement for procedures to be technological ensures that they are both teachable and testable.

Conceptually systematic refers to the foundations of behavior analysis. Applied behavior analysts describe their procedures and the impact of these procedures in terms of the basic principles of behavior. This dimension refers to the need for behavior analysts to stay close to their science, to link their findings back to the elemental principles of behavior, and to guard against adding superfluous and false explanations. This principle guards against the dilution of the science at the conceptual and explanatory level.

Effective refers to a core commitment to the improvement of behavior to a practical and meaningful extent. Behavior analysts do not value statistical significance or theoretical significance as much as they value social significance. Effective also implies that behavior analysts choose interventions with empirically verified effects, do not choose interventions that are unproven, and discourage the continuation or pursuit of baseless interventions. In recent years, this has taken the form of commitment to evidence-based practice. While behavior analysts have always valued this dimension, its importance has increased in the context of fad treatments and false claims of effectiveness.

Generality refers to the tendency for behavior changes to last over time, appear in untrained environments, and spread to untrained behaviors. If behaviors are not maintained and do not extend, the changes are far less significant. Behavior analysts are committed to teaching behaviors with enduring and transferrable qualities. In the earliest days of behavioral intervention, this dimension was not as prominently emphasized as it has been in recent years. Demonstration of the generality of behavior change is now routinely expected and sought.

In addition, behavior analysis is defined by several central constructs. Determinism implies that we can determine the cause and effect of various occurrences and can determine the variables responsible for change. Behavior is lawful, and functional relationships can be identified.

Philosophic doubt implies a skeptical worldview. Behavior analysts require empirical verification of hypotheses and do not embrace conclusions without confirming evidence. Parsimony implies that behavior analysts resort to the simplest explanation for events, the explanations that require the least inference and speculation. They stay close to the data and do not go beyond the data in explaining their results.

The strength of the science of behavior analysis comes from commitment to these dimensions and constructs. Furthermore, the integrity of the science depends upon the commitment to the continued development of and adherence to these dimensions and constructs in all branches of the science. Behaviorism, experimental analysis of behavior, and applied behavior analysis are inter-related, and the dimensions of the science fuel and further define one another.

Future Directions

Due to its adherence to the methods of science, behavior analysis has resulted in great strides in understanding, identifying the environmental variables that influence a wide variety of animal and human behavior. One of the areas of the biggest impact has been on persons with disabilities. Acknowledging the benefit of this particular perspective in studying human behavior, future directions of the application of behavior analysis should proceed in at least three directions. First, behavior analysts continue to sharpen its analysis of human behavior in the areas in which they have already studied. For example, deeper analysis of how to treat disabilities would provide significant clinical benefit, as has been shown already. Second, behavior analysis should branch out into other areas of human behavior not yet well studied and submit those areas to extensive scientific analysis. Some of these new areas could include analysis of human creativity and psychological disorders, such as obsessive-compulsive behavior. Third, behavior analysis should proceed more diligently in applying findings from experimental behavior analysis to the testing of and solutions for human behavior. This “translational”

research, in which experimental findings are tested in the human/applied area, has the potential for great payoff.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behaviorism](#)

References and Reading

- Ayllon, T., & Michael, J. (1959). The psychiatric nurse as a behavioral engineer. *Journal of the Experimental Analysis of Behavior*, 2, 323–334.
- Baer, D. M., Wolf, M. M., & Risley, T. (1968). Some current dimensions of applied behavior analysis. *Journal of Applied Behavior Analysis*, 20, 313–327.
- Bijou, S. W., Birnbrauer, J. S., Kidder, J. D., & Tague, C. (1966). Programmed instruction as an approach to teaching of reading, writing, and arithmetic to retarded children. *The Psychological Record*, 16, 505–522.
- Birnbrauer, J. S., Wolf, M. M., Kidder, J. D., & Tague, C. E. (1965). Classroom behavior of retarded pupils with token reinforcement. *Journal of Exceptional Child Psychology*, 2, 219–235.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Hall, R. V., Lund, D., & Jackson, D. (1968). Effects of teacher attention on study behavior. *Journal of Applied Behavior Analysis*, 1, 1–12.
- Mayer, G. R., Sulzer-Azaroff, B., & Wallace, M. (2012). *Behavior analysis for lasting change* (2nd ed.). Cornwall-on-Hudson: Sloan Publishing.

Behavior Analyst Certification Board

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Behavior Analyst Certification Board®

The Behavior Analyst Certification Board®, Inc. (BACB®) is a nonprofit corporation established in 1998 to meet professional credentialing needs

identified by behavior analysts, governments, and consumers of behavior analysis services. The BACB's mission is to develop, promote, and implement an international certification program for behavior analyst practitioners. The BACB has established uniform content, standards, and criteria for the credentialing process that are designed to meet:

1. The legal standards established through state, federal, and case law
2. The accepted standards for national certification programs
3. The “best practice” and ethical standards of the behavior analysis profession

Prior to the creation of the BACB, no uniform standards existed for evaluating the education, training, and experience of a behavior-analytic service provider. Since its inception, the BACB has set the standards for education and training in the field of applied behavior analysis and has certified more than 25,000 individuals (as of 2017).

The BACB adheres to the national standards for organizations that grant professional credentials. The BACB certification procedures and examination content undergo regular psychometric review and validation, pursuant to a job analysis survey of the profession and standards established by content experts in the field.

In 2015, in order to better control experience hours, all BCBAs were required to complete and pass a supervision training curriculum. This change was also supplemented by the change in degree requirements of 2016. In order to be qualified to be a BCBA, the individual must hold a master's degree in behavior analysis, psychology, or education. Other related fields, which were previously accepted by the BACB, are no longer accepted.

The BACB offers four different credentials, BCBA-D, BCBA, BCaBA, and RBT. The RBT credential went into effect in 2016. With the addition of the RBT credential, the BACB became the only organization in the field of behavior analysis to offer credentials at every educational level (graduate, undergraduate, and high school).

The Behavior Analyst Certification Board's BCBA, BCaBA, and RBT credentialing programs are accredited by the National Commission for Certifying Agencies, the accreditation body of the *Institute for Credentialing Excellence*. The BACB is endorsed by the *Association of Professional Behavior Analysts*, the *Association for Behavior Analysis International*, Division 25 (Behavior Analysis) of the American Psychological Association, and the *European Association for Behaviour Analysis*.

The most up-to-date information on the BACB can be found at www.bacb.com.

Behavior Assessment System for Children, 2nd Edition

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Synonyms

[BASC-2](#)

Definition

Acronym: BASC-2

Author: Kamphaus, Randy W.; Reynolds, Cecil R.

Purpose: Designed to determine behavioral and emotional functioning in children and adolescents in preschool through high school

Administration time: 10–20 min (teacher: TRS and parent: PRS), 30 min (self: SRP)

Scores: Scores/Interpretation: T scores and percentiles for general population and clinical populations

Ages/grades: Ages: 2:0 through 21:11 (TRS and PRS); 6:0 through college age (SRP). English and Spanish forms are available.

Scoring/administration programs: BASC-2 ASSIST and ASSIST-plus provide scoring,

reporting, and relationship to DSM-IV-TR diagnostic criteria. Online administration, scoring, and reporting are available for the TRS and PRS scales.

Publisher: Pearson

Publisher address: Pearson, 19500 Bulverde Road, San Antonio, TX 78259; Telephone: 800-627-7271; FAX: 800-632-9011; E-mail: pearsonassessments@pearson.com; Web: www.pearsonassessments.com.

The Behavior Assessment System for Children, 2nd Edition (BASC-2) is a commonly standardized set of rating scales and forms used to assess behavior in children and adolescents. The BASC-2 is normed on current US census population characteristics. Specific norms are not available for individuals with autism spectrum disorders (ASD) or neurodevelopmental disorders. Available scales include the Teacher Rating Scales (TRS), Parent Rating Scales (PRS), Self-Report of Personality (SRP), Student Observation System (SOS), and a Structured Developmental History (SDH).

The Teacher Rating Scales (TRS) measure adaptive and problem behaviors in the pre-school or school setting. Teachers or other qualified observers can rate specific behaviors on a four-point scale of frequency, ranging from "Never" to "Almost Always." The TRS contains 100–139 items. The Parent Rating Scales (PRS) measure both adaptive and problem behaviors in the community and home setting. The form requires a fourth grade reading level and is available in Spanish. Similar to the TRS, parents or caregivers can complete forms at three age levels – preschool (ages 2–5), child (ages 6–11), and adolescent (ages 12–21). The PRS contains 134–160 items and uses a four-choice response format. Both scales capture internalizing and externalizing behavioral adjustment reflected in an overall Behavioral Symptoms Index (BSI). Scales uniquely applicable to children and adolescents with ASD include assessment of functional communication and social skills.

The Self-Report of Personality (SRP) provides self-assessment of a child or adult's thoughts and feelings. Each form – child (ages 8–11), adolescent

(ages 12–21), and college (ages 18–25) – takes about 30 min to complete. The SRP-Interview (SRP-I) form for children 6–7 provides simple yes-or-no responses to questions asked by an examiner. The SRP-I takes about 20 min to complete. Spanish versions are available for the child and adolescent forms. In addition to measuring, internalizing (depression/anxiety/self-esteem), and externalizing problems (impulsivity/attention), the SRP offers self-assessment of interpersonal relationships and social stress.

Recent validity studies of the BASC-2 for use in individuals with ASD have found that the BASC-2 TRS and PRS forms can be effective in differentiating between children with high-functioning autism and typically developing peers. In a recent study (Ensign 2010), significant differences were found between individuals and typically developing groups on all PRS scales. DSM-IV-TR screening indices suggested that the Developmental Social Disorders Scale was highly effective in differentiating between the two groups. Hass et al. (2010) found similar results on the TRS in children receiving an educational classification of autism spectrum disorder.

References and Reading

- Ensign, J. (2010). Psychosocial subtypes on the behavior assessment system for children, second edition following pediatric traumatic brain injury. [Dissertation]. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 71(3-B), 2032.
- Hass, M., Brown, R. S., Brady, J., & Johnson, D. B. (2010). Validating the BASC-TRS for use with children and adolescents with an educational diagnosis of autism. *Remedial and Special Education*, 33, 173–183. <https://doi.org/10.1177/0741932510383160>.
- Mahan, S., & Matson, J. L. (2011). Convergent and discriminant validity of the Autism Spectrum Disorder-Problem Behavior for Children (ASD-PBC) against the Behavioral Assessment System for Children, second edition (BASC-2). *Research in Autism Spectrum Disorders*, 5(1), 222–229.
- Smith, E. A. (2011). Comparing behavior and neuropsychological functioning using NEPSY and BASC-2 scores in a mixed clinical sample. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 71(7-B), 4508.
- Van Slyke, K. B. (2008). Assessing childhood difficulties: Comparing the SDQ and the BASC-2. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 68(11-B), 7289.
- Volker, M. A., Lopata, C., et al. (2010). BASC-2 prs profiles for students with high-functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(2), 188–199.

Behavior Development Questionnaire

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Synonyms

BDQ; Wing Subgroups Questionnaire (WSQ)

Description

The Behavior Development Questionnaire (BDQ), formerly referred to as the Wing Subgroups Questionnaire, is an assessment tool used to classify individuals with autism spectrum disorders into one of three categories based on Wing and Gould's (1979) categorization scheme: *aloof*, *passive*, and *active-but-odd* (Castelloe and Dawson 1993). These classifications are distinguished based on the individual's quality of social interaction. The *aloof* group is considered to rarely display spontaneous social approaches to others, other than for the purpose of making requests, and often rejects social contact from others. The *passive* group shares this lack of spontaneous social approaches but does not reject social approaches from others. The *active-but-odd* group is described as being willing to make social approaches to others, but the approaches are considered unusual in quality (Castelloe and Dawson 1993).

The BDQ is a parent- or teacher-completed questionnaire that is comprised of 13 groups of four behavior descriptions. The 13 groups cover various domains such as patterns of social approaches, response to social approaches,

communication skills, imitation, play skills, unusual motor behavior, resistance to change, physical coordination, and challenging behaviors (Castelloe and Dawson 1993). Parents or teachers are asked to rate the frequency with which the target individual's behavior fits the described behavior (0 = never; 6 = always). An example item is "When my child is with unfamiliar adults or children he readily approaches others to interact and responds easily to others. His manner of interacting is generally appropriate (not awkward or unusual)" (Castelloe and Dawson 1993; p. 240). Summary scores are calculated for each of the four groups (i.e., *aloof*, *passive*, *active-but-odd*, *typical*) by totaling the scores across the 13 groups of behavior descriptions. The group for which the individual receives the highest summary score is assigned as the overall classification (Castelloe and Dawson 1993).

Historical Background

The BDQ was first reported in published research by Castelloe and Dawson (1993), and, at that time, the questionnaire was referred to as the Wing Subgroups Questionnaire, as it is based on clinical subgroups within ASD introduced by Wing and Gould (1979). Wing and Gould developed the classifications to help improve understanding of the relationships between typical autism, mental retardation, and other conditions involving social impairment.

Psychometric Data

Evidence for the validity and reliability of the BDQ has been provided through the examination of the questionnaire's internal consistency, the distinct nature of the three clinical subgroups (i.e., *aloof*, *passive*, *active-but-odd*), interrater agreement, and relationships between BDQ results and other concurrent measures. Internal consistency, as measured by Cronbach's alpha, has been shown to range from .63 for the *passive* classification to .85 for the *active-but-odd*

category on parent-completed questionnaires. For teacher-completed BDQs, Cronbach's alpha has been found to range from .54 for *passive* to .79 for *active-but-odd*. Clinicians' assignments of children with ASD to Wing's groups have been shown to be highly correlated with the results of parent-completed BDQs (Castelloe and Dawson 1993).

Evidence for the distinct nature of the three groups has been found in the strong negative correlation between the *aloof* and *active-but-odd* groups (−.70 for parent-completed BDQs and −.55 for teacher-completed BDQs) and the low correlations between *aloof* and *passive* (−.02 for parent-completed BDQs and −.04 for teacher-completed BDQs) and between *passive* and *active-but-odd* (.17 for parent-completed BDQs and .13 for teacher-completed BDQs; Castelloe and Dawson 1993; O'Brien 1996). However, item analysis has shown that 16 of the 50 items on the BDQ poorly discriminate among the subtypes (O'Brien 1996).

The *aloof* classification has been shown to be associated with lower IQ, lower receptive language skills, and more severe symptoms of autism (Castelloe and Dawson 1993), as compared to the other two classifications. Significant differences have also been found between the *aloof* and *active-but-odd* groups on the Peabody Picture Vocabulary Test and the Vineland Communication and Socialization domains (O'Brien 1996). The *passive* group has been shown to obtain lower Maladaptive Behavior scores on the Vineland and have less physical aggression reported on the Autism Behavior Checklist compared to the other two groups (O'Brien 1996). Wing's subgroup classifications based on clinicians' judgments have been associated with differences in brain activity measured through electroencephalography (EEG; Dawson et al. 1995). In one study, levels of active-but-odd behaviors on the BDQ did not distinguish children with high-functioning autism from a group with ADHD or ODD (Downs and Smith 2004). The ADHD/ODD group actually displayed more *aloof* behavior than the high-functioning autism group (Downs and Smith 2004).

Interrater reliability coefficients, based on pairs of teachers and teaching assistants completing the BDQ for a particular child, were found to be .60 for the *aloof* group, .81 for the *passive* group, .77 for the *active-but-odd* group, and .78 for the *typical* group (O'Brien 1996).

Clinical Uses

The BDQ can be used by clinicians to categorize individuals with ASD as *aloof*, *passive*, or *active-but-odd* and plan intervention goals appropriately (Castelloe and Dawson 1993). It has also been used as an outcome measure in clinical intervention research, and BDQ scores have been found to change following early intervention (Downs et al. 2007). To date, little has been published on the specific clinical uses of the BDQ.

See Also

- [Active-But-Odd Group](#)
- [Aloof Group](#)
- [Passive Group](#)
- [Wing, Lorna](#)

References and Reading

- Castelloe, P., & Dawson, G. (1993). Subclassification of children with autism and pervasive developmental disorder: A questionnaire based on Wing's subgrouping scheme. *Journal of Autism and Developmental Disorders*, 23, 229–241.
- Dawson, G., Klinger, L. G., Panagiotides, H., Lewy, A., & Castelloe, P. (1995). Subgroups of autistic children based on social behavior display distinct patterns of brain activity. *Journal of Abnormal Child Psychology*, 23, 569–583.
- Downs, A., & Smith, T. (2004). Emotional understanding, cooperation, and social behavior in high-functioning children with autism. *Journal of Autism and Developmental Disorders*, 34, 625–635.
- Downs, A., Downs, R. C., Johansen, M., & Fossum, M. (2007). Using discrete trial teaching within a public preschool program to facilitate skill development in students with developmental disabilities. *Education and Treatment of Children*, 30, 1–27.
- O'Brien, S. K. (1996). The validity and reliability of the Wing Subgroups Questionnaire. *Journal of Autism and Developmental Disorders*, 26, 321–335.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11–29.

Behavior Modification

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Definition

Behavior modification is a treatment approach based on Skinner's (1938, 1953) principles of operant conditioning. It seeks to establish desirable behavior and reduce or eliminate undesirable behavior through the use of empirically validated procedures, including but not limited to positive and negative reinforcement, extinction, and punishment. Behavior modification procedures have been used to treat a wide variety of human problems including attention deficit hyperactivity disorder, autism, enuresis and encopresis, fears and phobias, noncompliant behavior, and pica, among others.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavior Therapy](#)
- [Negative Reinforcement](#)
- [Positive Reinforcement](#)

References and Reading

- Martin, G., & Pear, J. (2003). *Behavior modification: What it is and how to do it* (7th ed.). Upper Saddle River: Prentice Hall.
- Skinner, B. F. (1938). *The behavior of organisms*. New York: Appleton-Century-Crofts.
- Skinner, B. F. (1953). *Science and human behavior*. New York: MacMillan.

Behavior Observation Scale

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Synonyms

BOS

Description

The Behavior Observation Scale (BOS) is a clinician-based measure of behaviors associated with autism (Freeman et al. 1978). The authors emphasized that children with autism should be studied within a development context and compared to nonspectrum typical and intellectually impaired children to distinguish behaviors specific to autism that are of diagnostic significance (Freeman et al. 1980).

The BOS is a checklist of 67 objectively defined behaviors. The clinician watches the child interact with age-appropriate toys through a one-way mirror in the presence of an examiner. The observation consists of recording the frequency of the specified behaviors in nine 3-min intervals. Three-minute baseline periods are also documented at the beginning and end of the play period. The examiner in the room presents the child with standard stimuli for seven of the intervals. During one interval, the examiner actively tries to engage the child through ball play. The behaviors are scored as not present or occurring once, twice, or continuously during the three-minute intervals. When not following these specific prompts, the examiner sits in one corner of the room and does not respond to the child if he or she initiates contact (Morgan 1988).

Historical Background

The BOS was one of the first diagnostic instruments for autism. Unlike other diagnostic

measures, the BOS was the first autism scale to emphasize the importance of controlling the observed behaviors of a child, as well as the environment in which the observation took place. The frequency of these observed behaviors was used to differentiate among diagnostic groups. The appearance of some specific rare behaviors during play was found to be a significant indicator, suggesting that the quality of some specific behaviors, rather than the frequency, was more important to diagnose children with autism (Lord and Corsello 2005).

Psychometric Data

The authors of the first factor analyses of the BOS concluded that it is necessary to create age-specific norms for the frequencies of behaviors of children with autism. These norms still need to be created comparing age-matched groups of both nonspectrum typical and intellectually impaired children (Freeman et al. 1978).

Some measures of reliability have been completed for the BOS. Interrater reliability of the BOS was assessed with a sample of 89 children, which included 36 with autism and 30 with nonspectrum intellectual disabilities matched for mental age and 23 typically developing children (Freeman et al. 1978). Correlation coefficients for ratings by the observer (watching through a one-way mirror) and the examiner (sitting in the room) were greater than 0.84 for 55 of the 67 behaviors; the published work did not include the coefficients for the remaining 12 items (Morgan 1988). Internal consistency and test-retest reliability have not been reported for the BOS (Parks 1983).

Various studies have also examined the validity of the BOS. The content validity of the BOS comes from the inclusion of ratable behaviors related to the clinical diagnostic criteria of autism. This is demonstrated by a factor analysis performed from three groups of children: those with autism, those without autism but with intellectual disability, and those with typical development (Freeman et al. 1980). According to their analyses, the authors characterize children with autism as exhibiting “inappropriate interactions

with people and objects,” the nonspectrum intellectually impaired group as having “solitary behaviors,” and the typically developing group as showing “appropriate interactions with people and objects” (p. 344).

In order to determine discriminate validity, Freeman and colleagues compared groups of children with autism and children without autism but with intellectual disabilities and found that they only differed on 11 of the 67 behaviors that compose the BOS (Freeman et al. 1979). However, the authors point out that the behaviors that did not discriminate between these groups were dependent on the developmental variables of mental and/or chronological age. Freeman and Ritvo (1980) compared children with autism, cognitively impaired children matched for mental age, and typically developing children matched for chronological age on the BOS. They found that six items differentiated the low-IQ autism group from the cognitively impaired group. They concluded that the three groups could be discriminated with the BOS if these six items were coded. No studies have examined how well the BOS distinguished between children with autism and children with other behavior problems (Morgan 1988).

Clinical Uses

The purpose of the BOS is to diagnose autism based on objective observation of behavior within a developmental context (Morgan 1988). Though the intention of the authors was to create an agreed-upon diagnostic framework for use in research (Freeman et al. 1978), the BOS is useful clinically as well. Freeman et al. add that the BOS can also be used to document changes in symptoms over time. Diagnosis and symptom changes are necessary for providing adequate therapeutic care for any individual with behavioral or cognitive difficulties, and the BOS provides the means for obtaining that information.

See Also

► [Autism Diagnostic Observation Schedule](#)

References and Reading

- Freeman, B. J., & Ritvo, E. (1980, May). The behavior observation scale for autism (BOS): IQ and behavior of autistic children. Paper presented at the meeting of the Western Psychological Association Honolulu.
- Freeman, B. J., Ritvo, E. R., Guthrie, D., Schroth, P., & Ball, J. (1978). The behavior observation scale for autism: Initial methodology, data analysis, and preliminary findings on 89 children. *Journal of the American Academy of Child Psychiatry*, 17, 576–588.
- Freeman, B. J., Guthrie, D., Ritvo, E. R., Schroth, R., Glass, R., & Frankel, F. (1979). Behavior observation scale: Preliminary analysis of the similarities and differences between autistic and mentally retarded children. *Psychological Reports*, 44, 519–588.
- Freeman, B. J., Schroth, P., Ritvo, E., Guthrie, D., & Wake, L. (1980). The behavior observation scale for autism (BOS): Initial results of factor analysis. *Journal of Autism and Developmental Disorders*, 10, 343–346.
- Lord, C., & Corsello, C. (2005). Diagnostic instruments in autistic spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 730–771). Hoboken: Wiley.
- Morgan, S. (1988). Diagnostic assessment of autism: A review of objective scales. *Journal of Psychoeducational Assessment*, 6, 139–151.
- Parks, S. L. (1983). The assessment of autistic children: A selective review of available instruments. *Journal of Autism and Developmental Disorders*, 13(3), 255–267.

Behavior Plan

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Definition

A behavior plan (behavior support plan or behavior intervention plan) is an organized plan to aid in the reduction of problem behaviors and/or increase desired behaviors. Behavior plans are documents which are usually developed by behavior analysts, teachers, counselors, and school psychologists, of with input from the individual themselves and/or their parent. The documents are suitable for implementation in various settings, such as private homes, public or private schools, residential facilities, or vocational facilities.

Historical Background

In the 1970s, the primary focus of behaviorism and behavior modification shifted from primarily using aversive procedures to eliminate undesirable behaviors toward the use of reinforcement-based techniques to increase desired behaviors (Brown 1987). In many ways, this shift increased the quality and effectiveness of behavioral interventions, as they became more widely accepted and utilized and began to be implemented across individuals with diverse behavioral profiles.

Current Knowledge

Behavior plans may incorporate various methods of behavior modification and are individualized for each person. The procedures outlined in behavior plans should be developed based on evidence-based techniques, such as functional behavior assessment or experimental functional analysis (see Functional Analysis). The behavioral functions identified using these methods are then used to develop an appropriate behavior plan.

The objectives of developing a behavior plan include identifying and defining target behaviors, recommending procedures to decrease challenging behaviors, and recommending procedures to increase appropriate behaviors or replacement skills.

Behaviors targeted to decrease and those targeted to increase must be clearly and operationally defined, so that all personnel implementing the plan will do so consistently. These definitions should be observable, clear, concise, and accurate.

If a behavior plan involves procedures to decrease challenging behaviors, it will necessarily include methods to teach or increase appropriate and functionally equivalent responses.

Behavior plans may include several parts, including antecedent strategies, identified behaviors to decrease, identified behaviors to increase, reinforcement systems, management strategies, data collection procedures, protocols for monitoring effectiveness of interventions, strategies for

maximizing generalization and maintenance, and criteria to discontinue.

Antecedent strategies, or antecedent control procedures, are procedures in which antecedents (i.e., environmental cues, discriminative stimuli, establishing operations, or response effort) are manipulated to influence a desired or undesired behavior. Antecedent strategies may include such techniques as clear delivery of directives, breaking tasks down into smaller components, providing and reviewing visual schedules, reviewing contingencies and expectations prior to each task, providing sequencing choices, providing opportunities for sensory activities, delivering reinforcement for appropriate behavior, providing warnings prior to transitions, modifying academic tasks to match student's ability, and making environmental manipulations. These manipulations may include arranging the environment to decrease the likelihood of problem behavior (e.g., clearing area of extraneous materials or positioning the individual away from dangerous materials). Antecedent strategies can be diverse and individualized for each individual.

Procedures designed to decrease problem behavior and increase replacement skills often include a reinforcement-based system. Examples of positive reinforcement-based systems are token economy systems (based on differential reinforcement), where individuals earn tokens (to be exchanged for identified reinforcers) based on the absence of problem behavior and/or engagement in alternative behaviors. In order to identify effective reinforcers, preference or reinforcer assessments should be conducted. These assessments may be informal, such as interviews or anecdotal reports from parents or caregivers or observation of the individual in various environments to determine where he/she allocates time. Preference or reinforcer assessments can also be formal, using a validated assessment such as a forced-choice preference assessment (Fisher et al. 1992) or multiple-stimulus without replacement (MSWO) preference assessment (DeLeon and Iwata 1996).

Once reinforcers have been identified, a token economy system might be considered as a procedure within a positive reinforcement-based behavior support plan.

A token economy could be based on a DRO (differential reinforcement of other behavior), DRA (differential reinforcement of alternative behavior), or DRI (differential reinforcement of incompatible behavior). In a DRO procedure, reinforcement is delivered solely for the absence of problem behavior, whereas in DRA or DRI procedures, reinforcement is delivered contingent on the occurrence of an alternative response or one that is incompatible with the target behavior. A token system could include tokens that can be physically manipulated by the individual (e.g., stickers, coins, or tickets), or they could simply be checkmarks on a list of completed tasks.

Some positive reinforcement-based procedures may not be as specific or structured as a differential reinforcement procedure and may not involve tokens at all. That is, direct reinforcement may be delivered on a fixed or variable schedule, contingent on appropriate behaviors. Positive reinforcement-based procedures can be implemented on their own or in conjunction with a number of other behavioral intervention procedures.

Additional consequence-based interventions may include procedures such as time-out, response blocking or interruption, physical or verbal redirection, response cost or removal of privileges, restitution, and overcorrection. These procedures involve various behavioral concepts such as extinction and may utilize principles of punishment. Therefore, they are typically used in conjunction with positive reinforcement-based procedures, so as not to focus only on the decrease of aberrant behavior but also the increase of appropriate behavior.

Time-out is a procedure which decreases problem behavior by removing reinforcement contingent on the occurrence of the target behavior(s). Time-out can be inclusionary (the individual remains in the same environment) or exclusionary

(the individual is removed from the environment in which the behavior occurred). It is a punishment-based procedure because a stimulus (reinforcement) is removed contingent on problem behavior, therefore reducing the future likelihood of the occurrence of that behavior.

Response blocking attempts to reduce the reinforcing aspects of the behavior by eliminating contact with the reinforcer. For example, an automatically maintained behavior such as hand flapping would be blocked, therefore restricting access to the reinforcing aspects of the behavior. In a response-interruption procedure such as a “hands-down” procedure, the response is interrupted, and the individual is physically redirected to an alternate response (i.e., putting hands down). This procedure may also function due to the principle of punishment, as the individual may engage less frequently in the behavior in order to avoid the redirection procedure.

Response cost, or removal of privileges, is another consequence-based procedure where a reinforcer (or multiple reinforcers) is removed contingent upon the occurrence of the target behavior. The future of occurrence of the target behavior is then decreased, as the individual avoids coming in contact with this aversive contingency. Restitution and overcorrection are typically used with behaviors where the environment is disturbed, such as property destruction, and refer to procedures where, contingent on the problem behavior, the individual is required to restore the environment to its original state. For example, if the individual dumps juice on the floor, he/she would be required to wipe it up. In overcorrection, the individual might be required to not only clean up the spilled juice but also wipe the rest of the floor.

Within any behavior plan should be a defined system for collecting data, including procedures appropriate to the behaviors being measured. Data collection methods may include event recording, duration recording, latency recording, or interval recording. Event recording refers to a count of behaviors as they occur. When reporting these data, it can be summarized as the total number of

behaviors that occurred, the rate of responding (frequency over time), or percentage of the occurrence of the target behavior as compared to other behaviors. Duration recording refers to how long an individual engages in a certain behavior. This can be reported as total duration per episode, per day, or some other specified time period. Latency recording refers to the amount of time between a stimulus and a response. For example, this type of recording may be used to examine how long it takes an individual to respond once an instruction has been given. Interval recording measures the presence or absence of a target behavior within specified time intervals. Whole interval, partial interval, and momentary time sampling are all types of interval recording. To determine which data collection method to use, it is important to look at the characteristics of the behavior and select a data collection method that will best represent the feature that is to be examined.

Data collection is a crucial part of an effective behavior plan, as it allows careful analysis of the target behaviors as they are influenced by the interventions put in place. Data sheets can be developed by the author of the behavior plan or by staff or caregivers implementing the plan. Once a data collection system has been established, all staff and caregivers involved in the plan's implementation must be aware of the procedures. It is important that the terms and methods for data collection have been reviewed by all people that will be involved in data collection, as this will increase the likelihood that the data collected are valid and useful in analyzing the success of the treatments. All people implementing a behavior support plan should be fully trained in all aspects of the interventions. A system to measure and track treatment fidelity should be in place to minimize procedural drift. Procedural drift refers to when, over time, certain interventions or parts of interventions are not carried out as they should be. This inconsistency can affect the success of the behavior plan and may result in some or all interventions losing effectiveness. Treatment fidelity checks can minimize procedural drift by putting into place specific measures to evaluate the implementation of the plan across implementers.

Future Directions

The individuals who develop behavior plans should always consider the ethical responsibilities involved in any behavior modifications and continue to explore positive reinforcement-based methods before those considered more aversive. Behavior Plans should incorporate the input of the individual and/or their parent whenever possible, to ensure a person-centered treatment plan. As the field of behavior analysis progresses, behavior plans should incorporate the most recent behavioral technologies to best support individuals. Currently, behavior plans are commonly utilized in the field of behavior analysis for individuals with autism and other developmental disabilities. In the future, behavior plans can be used to guide and support the efforts of those who work not only with developmentally disabled individuals but with people of all backgrounds who may benefit from behavioral strategies.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Positive Behavior Support](#)
- [Token Economy](#)

References and Reading

- Brown, D. P. (1987). *Hypnosis and behavioral medicine*. Hillsdale: Lawrence Erlbaum Associates.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (1987). *Applied behavior analysis*. Upper Saddle River: Prentice-Hall.
- DeLeon, I. G., & Iwata, B. A. (1996). Evaluation of a multiple-stimulus presentation format for assessing reinforcer preferences. *Journal of Applied Behavior Analysis*, 29, 519–532.
- Fisher, W., Piazza, C. G., Bowman, L. G., Hagopian, L. P., Owens, J. C., & Slevin, I. (1992). A comparison of two approaches for identifying reinforcers for persons with severe and profound disabilities. *Journal of Applied Behavior Analysis*, 25, 491–498.
- Miltonberger, R. G. (2004). *Behavior modification principles and procedures* (3rd ed.). Belmont: Wadsworth/Thomson Learning.

Behavior Rating Instrument for Autistic and Atypical Children (BRIAAC)

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Synonyms

BRIAAC

Description

The Behavior Rating Instrument for Autistic and other Atypical Children (BRIAAC) was created for the purpose of diagnosing autism (Ruttenberg et al. 1974). The measure was based on observations in a day treatment program of children with autism who had been diagnosed using Kanner's (1943) autism criteria. The measure consists of eight subscales that are completed by a trained examiner who has observed the child for an extensive period of time. The observations lead to descriptive ratings for each subscale within the range characteristic of a 3.5- to 4.5-year-old typically developing child compared to those that are characteristic of a child with severe autism.

Historical Background

The BRIAAC was one of the earliest measures of autism created shortly after Rimland's first diagnostic checklist (Lord and Corsello 2005; Rimland 1964). It was the first measure of autism to utilize direct observation of behaviors as described in the case notes of defined raters, making it a significant milestone of behavior-based measures (Parks 1983).

Psychometric Data

The BRIAAC consists of eight scales that are developmentally ordered, with the lowest level representing behaviors uniquely associated with autism and the highest level representing developmental accomplishments typical of normal 4-year-old children. The scales are communication, drive for mastery, vocalization and expressive speech, sound and speech reception, body movement, social functioning, psychosexual development, and relationship; in the 1977 edition, social functioning and psychosexual development were renamed social responsiveness and psychobiological development, respectively (Ruttenberg et al. 1974). The purpose behind the scoring system is to reflect the entire range of possible behavior and the importance of each behavior within this range (Ruttenberg et al. 1966).

The interrater reliability of the original version of the BRIAAC was examined using trained students as raters (Ruttenberg et al. 1966). Spearman rank correlation coefficients for the four-core scales ranged from 0.85 to 0.88, demonstrating high agreement among raters. However, since all of the children observed had been previously diagnosed with autism, the high reliability does not indicate the ability to diagnose autism accurately with the BRIAAC (Ruttenberg et al.).

Two of the authors also examined the BRIAAC's interrater reliability using the scores of 113 children with autism as determined by seven different pairs of raters (Wenar and Ruttenberg 1976). The correlation coefficients ranged from 0.85 to 0.96 across the eight scales, indicating moderate interrater reliability, as they did not control for response frequencies.

Factor analysis completed by Wenar and Ruttenberg also supported internal consistency because they found a high loading on one factor, which they described as resistance to participation in activities, such as interacting with others or the environment (1976). Cohen et al. also performed factor analysis and similarly found that the same factor accounted for 69% of the variance. All scales, except psychosexual development, loaded at 0.80 or higher, suggesting high internal

consistency (Cohen et al. 1978). This shows that the test is, in fact, measuring a unity factor, leading to high internal consistency.

Both the items and the subscales of the BRIAC were based on frequent observations of children with autism in a daycare center by a highly trained team of specialists. Their observations were incorporated into the items and scales, resulting in good content validity (Wenar and Rutterberg).

The BRIAC presumably has good construct validity because it is based on Kanner's autism criteria (1943), and the children observed were diagnosed according to those same criteria (Morgan 1988). In addition, as previously mentioned, factor analysis demonstrated that the BRIAC does examine one core factor, the resistance to engage with others and the environment (Wenar and Rutterberg; Cohen et al.).

Concurrent validity was studied by comparing the BRIAC scores and clinicians' ratings of 26 children either with autism or typical development (Wenar and Rutterberg). Significant correlations were established between the clinicians' rating and the total BRIAC scores ($r = .69$) and three subscale scores (relationship to an adult, $r = .43$; vocalization and expressive speech, $r = .64$; sound and speech reception, $r = .65$). The authors viewed the examined concurrent validity as satisfactory and expressed the desire to examine the remaining subscales in the future.

Cohen et al. examined the discriminant validity of the BRIAC and found that the total scores did not effectively discriminate among the diagnostic groups of primary-childhood autism, secondary-childhood autism, early-childhood psychosis, developmental aphasia, and mental retardation (1978).

Clinical Uses

The scoring system that addresses the whole range of possible behaviors is clinically relevant because it identifies both signs of progress and problem behaviors (Rutterberg et al. 1966). These areas of needed improvement can be specific for each child evaluated. In addition, the

levels of the scales assist in planning therapeutic programs for children with autism because they indicate upcoming developmental steps and how therapy can progress to meet the child's developmental needs.

See Also

- [Autism Diagnostic Observation Schedule](#)
- [Behavior Observation Scale](#)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- Cohen, D. J., Caparulo, B. K., Gold, J. R., Waldo, M. C., Shaywitz, B. A., Rutterberg, B. A., & Rimland, B. (1978). Agreement in diagnosis: Clinical assessment and behavior rating scales for pervasively disturbed children. *Journal of the American Academy of Child Psychiatry*, 17(3), 589–603.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Lord, C., & Corsello, C. (2005). Diagnostic instruments in autistic spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 730–771). Hoboken: Wiley.
- Morgan, S. (1988). Diagnostic assessment of autism: A review of objective scales. *Journal of Psychoeducational Assessment*, 6, 139–151. (ed.). (730–771). Hoboken: Wiley.
- Parks, S. L. (1983). The assessment of autistic children: A selective review of available instruments. *Journal of Autism and Developmental Disorders*, 13(3), 255–267.
- Rimland, B. (1964). *Infantile Autism: The syndrome and its implications for a neural theory of behavior* (2nd printing). New York: Appleton-Century-Crofts.
- Rutterberg, B. A., Dratman, M. L., Fraknoi, J., & Wenar, C. (1966). An instrument for evaluating autistic children (BRIAC). *Journal of the American Academy of Child Psychiatry*, 5, 453–478.
- Rutterberg, B. A., Kalish, B. I., Wenar, C., & Wolf, E. G. (1974). *Behavior rating instrument for autistic and other atypical children* (rev. ed.). Philadelphia: Developmental Center for Autistic Children.
- Wenar, C., & Rutterberg, B. A. (1976). The use of BRIAC for evaluating therapeutic effectiveness. *Journal of Autism and Childhood Schizophrenia*, 6, 175–191.
- Wenar, C., Rutterberg, B. A., Kalish-Weiss, B., & Wolf, E. G. (1986). The development of normal and autistic children: A comparative study. *Journal of Autism and Developmental Disorders*, 16, 317–333.

Behavior Rating Scale (BRS)

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Synonyms

BRS

Description

The Behavior Rating Scale (BRS) is a subtest within the Bayley Scales of Infants Development, which is an assessment frequently used to assess the development of infants and children, including those with a diagnosis of autism. It is a norm-referenced assessment that was first published in 1969 and later revised in 1993 and provides standard mental and motor indices and a developmental age equivalent for children 2 months to 2.5 years old. The Bayley Scales of Infants Development consists of two subtests in addition to the BRS: the mental development index (MDI) and the psychomotor development index (PDI). The BRS is a form for the evaluator to rate the child's behavior throughout testing, including the ability to pay attention, social engagement, affect and emotions, and the quality of movement and motor control. The BRS, previously known as the infant behavior record (IBR), underwent many changes for the second edition, including a revamp in format and a new scoring system.

The examiner completes the BRS after the other two components of the Bayley are assessed (Nellis and Gridley 1994). The examiner also solicits from the parent or caregiver additional information about the tasks and the session as a whole. This includes whether the child's behavior was typical during the session and if the child's performance on the tasks reflected his or her abilities. This information is not included in the BRS

score, but aids in evaluating the accuracy of those scores. The BRS scores can be interpreted at four different levels: total scores, factor scores, item analysis, and comparisons with the other two tests within the Bayley. The total score compares the child with same-aged peers. Factor scores vary depending on age and are described by qualitative labels.

Historical Background

The BRS was previously known as the infant behavior record (IBR) in the original version of the Bayley Scales of Infant Development (Bayley 1969). Since its creation, the Bayley has remained one of the most standardized and widely used measures for determining the developmental skills of infants and children in both clinical and research settings (Wolf and Lozoff 1985; Klin et al. 2005).

Psychometric Data

The Bayley provides a method for obtaining age-equivalent scores for four facets of development, cognitive, language, social, and motor, but empirical evidence for their validity is limited (Bayley 1993). The authors of the Bayley revision (1993) found that the test has excellent statistical properties and sensitivity to high-risk childhood conditions, but its value for assessing young children with autism is limited (Klin et al. 2005). Children with autism typically present with a varied profile of skills, with higher level nonverbal problem-solving abilities, lower level expressive language, and lowest scores in receptive language. Consequently, any composite index score summarizing performance across domains will misrepresent a child's developmental profile, indicating that the actual profile with varied skill levels is more informative than any composite scores.

The BRS contains 30 items that rate the child's relevant test-taking behaviors and simultaneously measures attention/arousal, orientation/engagement, emotional regulation, and motor quality

(Bayley 1993). The scoring of the BRS is based on rank values and has a five-point ordinal scale for each behavior. There is limited psychometric data for the BRS, as most analyses have been completed on the Bayley as a whole. The authors of the revised edition of the Bayley found that total scores were more highly correlated for the older age range ($r = 0.88$) than for the younger age range ($r = 0.70$), but concluded that the interrater reliability for the BRS was fairly high for an observation-based measure (Bayley 1993, as cited in Koseck 1999).

Clinical Uses

The Bayley is particularly relevant in clinical settings with children suspected of having a developmental delay because it can both identify the presence of a developmental delay and provide information to help the caregiver know which services are necessary to help the child (Washington 1998). It is a relevant measure for children demonstrating signs of autism because it tests a wide variety of behaviors across different domains, but it is most informative when the entire profile is assessed, rather than the total scores (Klin et al. 2005). Another reason that the Bayley is frequently used with children with developmental delays is that the testing materials are of interest for these children and can hold their attention (Nellis and Gridley 1994). These qualities of the Bayley and the BRS make the measure highly informative in both clinical and research settings.

See Also

- [Autism Screening Instrument for Educational Planning \(ASIEP-2\)](#)

References and Reading

- Bayley, N. (1969). *Bayley scales of infant development*. New York: Psychological Corporation.
- Bayley, N. (1993). *Bayley scales of infant development* (2nd ed.). San Antonio: Psychological Corporation.

- Klin, A., Saulnier, C., Tsatsanis, K., & Volkmar, F. (2005). Clinical evaluation in autism spectrum disorders: Psychological assessment within a transdisciplinary framework. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 730–771). Hoboken: Wiley.
- Koseck, K. (1999). Review and evaluation of psychometric properties of the revised Bayley scales of infant development. *Pediatric Physical Therapy*, 11(4), 198–204.
- Nellis, L., & Gridley, B. E. (1994). Review of the Bayley Scales of Infant Development (2nd ed.). *Journal of School Psychology*, 32(2), 201–209.
- Washington, K. (1998). The Bayley scales of infant development-II and children with developmental delays: A clinical perspective. *Journal of Developmental and Behavioral Pediatrics*, 19(5), 346–349.
- Wolf, A. W., & Lozoff, B. (1985). A clinically interpretable method for analyzing the Bayley infant behavior record. *Journal of Pediatric Psychology*, 10(2), 199–214.

Behavior Rehearsal

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Definition

Behavior rehearsal involves practicing appropriate behavior responses within social situations. There are many methods for rehearsing social behaviors. One method may include individuals imagining or thinking about themselves performing and responding appropriately to others. A second method may include individuals practicing social interactions through describing them verbally to others. A third method may include role-playing. With all methods, building fluency through repeated rehearsal is vital to achieving success and increasing appropriate social skills.

See Also

- [Behavior](#)

References and Reading

- Morgan, R. L., & Salzberg, C. L. (1992). Effects of video-assisted training on employment-related social skills of adults with severe mental retardation. *Journal of Applied Behavior Analysis*, 25, 365–383.
- Sarokoff, R., & Sturney, P. (2004). Effects of behavior skills training on staff implementation of discrete trial teaching. *Journal of Applied Behavior Analysis*, 37, 535–538.

Behavior Summarized Evaluation-Revised (BSE-R)

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Synonyms

BSE; BSE-R; IBSE

Description

The Behavior Summarized Evaluation-Revised (BSE-R) is a psychometric instrument designed for research and clinical purposes.

The current version includes 29 items that enable the formalization of behavior observations in the different domains in which specifically autistic difficulties occur.

These domains are touch, eye contact and communication, motor behavior, perception, and imitation. Coding is based on the observations collected by a person who is regularly in touch with the child. The observations are performed in the different situations of daily life by all of the persons who work with the child. A glossary describes briefly the content of each item, which is rated on a 5-point scale ranging from 0 = never to 4 = continuously. The item is rated 0 if the behavior never appears, 1 if it sometimes appears,

2 if often, 3 if very often, and 4 if it is always present. Thus, the total score indicates both the frequency of the behavior disorders and the intensity of the pathology.

Historical Background

Professor Gilbert Lelord and his colleagues designed the first BSE version in 1975 (Laffont et al. 1975). The purpose of this version was to correlate clinical variables with electrophysiological variables. The studies using this scale have demonstrated that certain clinical characteristic such as indifference to others or resistance to change was related to electrophysiological signs such as the low amplitude of the potentials elicited in response to sensory stimuli. The major interest of this technique is to provide a framework for observations that can be repeated in different contexts and at different times in the child's development.

Later, several successive versions involving a varying number of items were developed. The factor analysis provided the means for the classification of the test items into categories.

The 1990 version involved 20 items (Barthelemy et al. 1990). Nine items were later added to the revised version (Barthelemy et al. 1997). A specific version has been developed for infancy and preschool years based on the BSE, and 13 items have been added to describe early manifestations of autism (The infant BSE: IBSE, Adrien et al. 1992).

Psychometric Data

The BSE-R has been validated in a research led by Catherine Barthelemy and her colleagues (Barthelemy et al. 1997); 136 children with autism were included in this study.

Inter-rater Reliability

For the inter-rater reliability study, 29 children (21 boys and 8 girls) were observed and their behaviors assessed with the BSE by two separate groups of nurses trained to use the instrument. The

total score reliability was very high (0.97). Reliability measures were calculated for each item. Three items (1, 10, 29) also had high reliability (0.75–1.0); ten items (2, 4, 5, 6, 9, 12, 14, 20, 27, 28) had good reliability (0.60–0.74); and 12 items (3, 7, 8, 11, 13, 15, 16, 19, 21, 23, 24, 26) had fair reliability (0.40–0.59). Only four items (17, 18, 22, 25) had a low reliability and were therefore excluded from the other analyses.

A factor analysis was performed on the BSE-R results for the 136 children. Six factors were extracted. Two factors accounted for more than 10% of the total variance. Combined, they accounted for a total of 48, 6% of the total variance. The two most loaded factors were labeled “interaction disorder” (items 1, 2, 3, 4, 5, 6, 8, 9, 12, 23, 24, 26, 28) and “modulation disorder” (item 11, 13, 16). A negative correlation was found between the BSE-R score for Factor 1 and Development Quotient. This means that the higher the BSE-R score, the lower the DQ was. No correlation was found for Factor 2 and the DQ.

A criterion validity study was performed on the BSE-R scores for all 136 children. The external criterion was the Expert Severity Score (ESS). This was based on the observation of two experienced staff psychiatrists who were blind to the BSE-R score. The ESS ranged from 1 (minimum) to 5 (maximum). A glossary was available, and for that reason, the ESS had an excellent reliability. Three diagnostic groups were constituted: Autistic Disorders (AD), Pervasive Developmental Disorders Not Otherwise Specified (PDDNOS), and Mental Retardation (MR). A solid relation between the BSE-R score and the ESS was found. Because BSE-R Factor 1 items significantly correlated with the DQ, the variance explained by the DQ was controlled. Each BSE-R Factor Item and BSE Factor 1 score correlated with the ESS. However, the ESS did not correlate with Factor 2 of the BSE-R.

Convergent Validity Study

Seventy-five children were assessed with the Rimland E2 scale. The same subgroups were selected (AD = 51 children; PSSNOS = 8 children; MR = 16 children). Significant differences existed between the three diagnostic subgroups

for the ESS. Correlations were calculated between the BSE-R score for Factor 1, Factor 2, and Rimland E2 score. A significant correlation was found between the BSE-R score for Factor 1 and the Rimland score (0.41). But there was no significant correlation between the BSE-R score for Factor 2 and the Rimland score. Convergent validity was also confirmed in the study by Oneal et al. (2006), where the BSE scores correlated highly with the CARS, a well-validated instrument.

Sensitivity and Specificity Study

Thanks to a ROC analysis, a cutoff of 27 was determined. This score permits a discrimination between autistic children (AD) and nonautistic children (MR + PDDNOS) with a sensitivity of 0.74 and a specificity of 0.71.

Other previously published results concern the first version of the BSE and can be found in different papers (Barthelemy et al. 1990; Reeb et al. 2009).

Recently, a validation study of this scale was carried out in a Lebanese population (Hreich et al. 2016). The scale was first translated into Arabic, and then a back translation was performed. Hundred children with ASD (age range, 35–153 months; DS, 28.0) were evaluated. Their diagnosis was based on DSM4 criteria. The severity of their disorders was measured with CARS. Fifty-eight percent of them had an intellectual disability. Inter-rater fidelity was excellent. The study of internal validity revealed a main factor related to the severity of autistic symptoms (internal consistency of 0.91 in a one-to-one setting and 0.92 in group settings). The external validity was evaluated by the correlation with the CARS score. This study confirmed that the main factor is essentially determined by ASD severity, not by the severity of ID. This factor was named “relational deficiency” according to the initial paper of Barthélémy et al. (1997). In this paper, it is confirmed that BSE-R in Arabic is a practical tool, useful to all team members working with ASD children in Lebanon and the Arab countries. It also allows future research based on reliable tools at an international level.

Clinical Uses

As already indicated, the BSE-R was designed for research and clinical purposes. As the validity and stability of the results obtained with the BSE-R were confirmed, several studies using this instrument were led mainly by Catherine Barthelemy's team.

Biological Measures

Initially, research studies were conducted using the first version (BSE). The objective was to evaluate the severity of behavioral problems in autistic children and to assess the correlations with biological markers. Hameury et al. (1995) using the BSE and other measures distinguished four groups in a population of 202 subjects. Group 1 included subjects with severe autistic behavior, profound mental retardation, and severe neurological symptoms. Group 2 included subjects presenting autistic behavior, language, and communication disorders, with slight or moderate intellectual impairment and mild neurological symptoms. Group 3 included children with severe intellectual impairment and neurological symptoms with no or few autistic behavior patterns. In Group 4, subjects showed multiple but mild disorders. The biochemical parameters of the four groups were compared. The levels of HVA (homovanillic acid) measured in urine samples varied significantly, and Group 3 presented a very high level of HVA compared to the other groups. The authors declare that this approach could make possible the establishment of subgroups in which behavioral clinical profiles could correspond to certain biological profiles (with metabolic characteristics).

Other studies have been undertaken with the BSE revised version. Roux et al. (1997) examined relations between electrophysiological reactivity and BSE-R. In a population of 73 children, they showed that the item "bizarre responses to auditory stimuli" was correlated with abnormalities in frontocentroparietal electrophysiological reactivity, and that the item "unstable attention or easily distracted" corresponded to abnormalities in frontal electrophysiological reactivity.

Hérault et al. (1996) also used the BSE-R in a study on urinary levels of serotonin. No

relationship was found between molecular biology results and clinical scores.

Bruneau et al. (2003) studied relations between late auditory-evoked potentials recorded in the temporal area and autism severity based on BSE-R. They observed a negative correlation between the importance of the right temporal response and the BSE-R score. The greater amplitude of the right temporal responses corresponded to lower (less severe) BSE-R scores on items involving verbal and nonverbal communication skills.

Gomot et al. (2011) examined neural basis of auditory change-detection in children with autism spectrum disorders ($N = 27$) through electrophysiological patterns (MMN, P3a). They wanted to test whether these electrophysiological patterns were quantitatively related to intolerance of change (using the BSE-R scale). Children with ASD displayed significantly shorter MMN latency and larger P3a than controls, indicating a greater tendency to switch attention to deviant events. These electrophysiological abnormalities were significantly more marked in children with greater difficulties in tolerating change. It is therefore confirmed that there is a relationship between the electrophysiological patterns of auditory change-detection and the clinical signs of intolerance to change.

Guimard-Brunault et al. (2013) used eye tracking to compare spontaneous visual attention to a screen displaying a face or an object between children with autism and controls. This study was carried out in a nonconstraint condition. The visual exploration time was measured during passive viewing of static images of faces or objects. The assessment of subjects with autism was conducted with the CARS and the BSE-R. In children with autism, time exploring face screen and time exploring object screen were lower than in control subjects. They were not correlated with intensity of distractibility. No interaction was found between group and type of image on time spent exploring screen. Only time spent in exploring face was correlated with autism symptom severity and gaze impairment.

As the BSE-R is included in the routine assessment of patients in Tours Child Psychiatry Unit, clinical data gathered are available for other

studies in different fields such as genetics (Mbarek et al. 1999).

Sensitivity to Treatment Effects

The BSE and later the BSE-R have been used to evaluate the evolution of children receiving different kinds of treatment. In a study published in 1989, Barthelemy et al. examined the modifications in the BSE scores of 27 children receiving exchange and development therapy over a period of 1–2 years. Different diagnostic subgroups were included (autism, mental retardation, atypical pervasive developmental disorder, developmental delay without autism). The pre- and post-mean BSE scores were compared. The decrease in the scores is interpreted as an improvement. In another study, Barthelemy et al. (1989) assessed changes in BSE scores and biochemical markers in 13 children with autism receiving medication. Significant decreases were observed in a BSE item in responders who also showed significant modifications in serotonin and dopamine levels (Barthelemy et al. 1997). In this study, the treatment lasted 9 months. A significant decrease in BSE-R scores was noted. Other studies (Lelord et al. 1981; Martineau et al. 1988) were led with the BSE as an indicator of improvement. These trials are summarized in Reeb et al. (2009).

In a study carried out by Blanc et al. in 2013, changes induced by EDT (behavior, development, and functioning) were measured at follow-up using the BSE-R among other tools. Thirty-five children with a severe autism associated with a developmental delay followed for 9 months showed improvement in the capacity of exchange and communication.

All these studies suggest that the BSE and the BSE-R are sensitive to treatment effects. However, the number of children included in these trials was usually small and there was no control group. Thus, all these results must be considered with caution.

Family Home Movies: Early Signs

The IBSE has been used in research on early signs based upon family home movies (Adrien et al. 1993). The family movies of 12 autistic children and 12 typically developing children were

analyzed using the IBSE. Two diagnostic-blind raters scored the films. The order of presentation of the videotapes was randomized. The scoring was performed for two different periods, the first and second year, in order to compare the signs observed during these two periods. The analysis of these family movies led to finding specific behaviors that enabled the prediction of the autism diagnosis.

Parents' Rating of Improvement

The BSE is a simple, easy-to-manage tool that has been used for the assessment of improvement by the parents themselves (Oneal et al. 2006). The results show that the BSE presents acceptable psychometric qualities for parent usage when assessing changes in the child's behavior.

The BSE-R is an interesting instrument. It has been validated and can be used in different contexts by professionals from different fields as well as by parents. It can be useful to identify the symptoms of autism, to follow the changes in the expression of these symptoms across age, and to measure the effects of treatment.

References and Reading

- Adrien, J. L., Barthelemy, C., Perrot, A., Roux, S., Lenoir, P., Hameury, L., et al. (1992). Validity and reliability of the infant behavioral summarized evaluation (IBSE): A rating scale for the assessment of young children with autism and developmental disorders. *Journal of Autism and Developmental Disorders*, 22, 375–394.
- Adrien, J., Lenoir, P., Martineau, J., Perrot, A., Hameury, L., Larmande, C., et al. (1993). Blind ratings of early symptoms of autism based upon family home movies. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 617–626.
- Barthelemy, C., Bruneau, N., Jouve, J., et al. (1989). Urinary dopamine metabolites as indicators of the responsiveness to fenfluramine treatment in children with autistic behavior. *Journal of Autism and Developmental Disorders*, 19(2), 241–254.
- Barthelemy, C., Adrien, J. L., Tangay, P., Garreau, B., Fermanian, J., Roux, S., et al. (1990). The behavioural summarized evaluation: Validity and reliability of a scale for the assessment of Autistic behaviours. *Journal of Autism and Developmental Disorders*, 20, 189–203.
- Barthelemy, C., Adrien, J. L., Roux, S., Garreau, B., Perrot, A., & Lelord, G. (1992). Sensitivity and specificity of the behavioural summarized evaluation (BSE) for the

- assessment of Autistic behaviours. *Journal of Autism and Developmental Disorders*, 22(1), 23–31.
- Barthélemy C., Roux S., Adrien J. L., Hameury L., Guérin P., Garreau B., Fermanian J., & Lelord G. (1997). Validation of the revised behaviour summarized evaluation scale (BSE-R). *Journal of Autism and Developmental Disorders*, 27(2), 139–153.
- Blanc, R., Malvy, J., Dansart, P., Bataille, M., Bonnet-Brilhault, F., & Barthélemy, C. (2013). La thérapie d'échange et de développement, une rééducation neurofonctionnelle de la communication sociale. *Neuropsychiatrie de l'Enfance et de l'Adolescence*, 61(5), 288–294.
- Bruneau, N., Bonnet-Brilhault, F., Gomot, M., Adrien, J.-L., & Barthelemy, C. (2003). Cortical auditory processing and communication in children with autism: Electrophysiologically behavioral relations. *International Journal of Psychophysiology*, 51, 17–25.
- Gomot, M., Blanc, R., Clery, H., Roux, S., Barthelemy, C., & Bruneau, N. (2011). Candidate electrophysiological endophenotypes of hyper-reactivity to change in Autism. *Journal of Autism and Developmental Disorders*, 41(6), 705–714. <https://doi.org/10.1007/s10803-010-1091-y>.
- Guimard-Brunault, M., Hernandez, N., Roche, L., Roux, S., Barthelemy, C., Martineau, J., & Bonnet-Brilhault, F. (2013). Back to basic: Do children with autism spontaneously look at screen displaying a face or an object? *Autism Research and Treatment*. <https://doi.org/10.1155/2013/835247:835247>.
- Hameury, L., Roux, S., Barthelemy, C., Adrien, J. L., Desombre, H., Sauvage, D., et al. (1995). Quantified multidimensional assessment of autism and other pervasive developmental disorders. Application for bioclinical research. *European Child & Adolescent Psychiatry*, 4(2), 123–135.
- Héroult, J., Petit, E., Martineau, J., Cherpi, C., Perrot, A., Barthelemy, C., et al. (1996). Serotonin and autism: Biochemical and molecular biology features. *Psychiatry Research*, 65, 33–43.
- Hreich, E. K., Messarra, C., Roux, S., Barthélemy, C., & Richa, S. (2016). Validation in Arabic of the revised autistic behavior summarized evaluation scale (BSE-R). *Encephale*. <https://doi.org/10.1016/j.encep.2016.04.013>.
- Laffont, F., Jusseaume, P., Bruneau, N., Dubost, P., & Lelord, G. (1975). Conditionnement des potentiels évoqués chez des enfants normaux, retardés mentaux et autistiques. *Revue d'Electroencephalographie et de Neurophysiologie*, 5, 369–374.
- Lelord, G., Müh, J. P., Barthelemy, C., Martineau, J., Garreau, B., & Callaway, E. (1981). Effects of pyridoxine and magnesium on autistic symptoms. Initial observations. *Journal of Autism and Developmental Disorders*, 11, 219–230.
- Martineau, J., Barthelemy, C., Cheliakine, C., & Lelord, G. (1988). Brief report: An open middle-term study of combined vitamin B6-magnesium in a subgroup of autistic children selected on their sensitivity to this treatment. *Journal of Autism and Developmental Disorders*, 18, 435–447.
- Mbarek, O., Marouillat, S., Martineau, J., Barthelemy, C., Müh, J. P., & Andres, C. (1999). Association study of the NF1 gene and Autistic disorder. *American Journal of Medical Genetics*, 88, 729–732.
- Oneal, B. J., Reeb, R. N., Korte, J. R., & Butter, E. J. (2006). Assessment of home-based behaviour modification programs for autistic children: Reliability and validity of the behavioural summarized evaluation. *Journal of Prevention & Intervention in the Community*, 32(1–2), 25–39.
- Reeb, R. N., Folger, S. F., & Oneal, B. J. (2009). Behavioural summarized evaluation: An assessment tool to enhance multidisciplinary and parent-professional collaborations in assessing symptoms of Autism. *Children's Health Care*, 38, 301–320.
- Roux, S., Adrien, J. L., Bruneau, N., Garreau, B., Couturier, G., Gomot, M., et al. (1997). Classification of autistic syndrome using behavioural and electrophysiological assessments. *Developmental Brain Dysfunction*, 10, 28–39.

Behavior Therapy

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Definition

Behavior therapy (a term used here interchangeably with behavior modification) is the application of techniques based on empirically derived principles of learning theory to the treatment of human problems, with the goal of reducing or eliminating unwanted behavior and replacing it with behavior that is more adaptive and socially appropriate. While individual strands of behavior therapy differ in several important ways, all share an emphasis on treating behavioral symptoms, with little or no reliance or attention to underlying unconscious processes. With respect to cognitions, behavior therapy proposes that by changing overt behavior (through reinforcement, extinction, punishment, etc.), more adaptive emotional and affective thinking will follow. The understanding of the “here-and-now” in context, rather

than underlying conflicts in a person's past, is a key distinction between behavior therapy and other more psychodynamic or psychoanalytic therapies.

Historical Background

Behavior therapy has evolved over the past six decades from many schools of thought and philosophical systems. This diversity is most evident in the fact that despite the predominance of the discipline of psychology among practitioners of behavior therapy, some of the earliest pioneers were from other fields, for example, the Russian physiologist Ivan Pavlov and the South African psychiatrist Joseph Wolpe. Equally important is the observation that what we today consider behavior therapy generated from the confluence of the work of three groups in different countries. In the United States, the work of Skinner, Lindsley, and others on operant conditioning adopted a more functional approach to assessment and treatment and led to an emphasis on the experimental analysis of behavior best represented in the field of applied behavior analysis. British psychologist Hans Eysenck and his colleagues at the Maudsley Hospital in London emphasized that behavior problems were the result of complex interactions between the client's personality features, the behavior itself, and the environment. Their work targeted these interrelationships through the use of techniques of behavior change based on S-R learning theory (classical conditioning) as an alternative to the then-prevalent psychoanalytic models. In South Africa, Joseph Wolpe, Arnold Lazarus, and others were at work developing techniques that used behavioral principles to treat more common psychological problems, leading to the development of systematic desensitization and psychotherapy by reciprocal inhibition. At the time, these evidence-based procedures were considered both revolutionary and evolutionary and set the stage for the

continuing development of behavior therapy as a scientific discipline with people with a wide range of psychological problems.

Current Knowledge

Contemporary behavior therapy may arbitrarily but conveniently be classified under five broad strands: applied behavior analysis, neo-behavioristic mediational S-R models, social learning approaches, cognitive therapy and cognitive behavior therapy, and "third-generation" approaches. By far, the most widely practiced with respect to understanding and treating autism are those based on applied behavior analysis (ABA). Here, the extension of the work of Skinner and his early colleagues to Donald Baer, Sidney Bijou, Fred Keller, Brian Iwata, and many others has generated a powerful evidence-based technology of change designed to address significant deficiencies in learning as well as behavioral excesses and deficits exhibited by those with ASD. The cornerstone of ABA is function-based assessment and treatment, with data-based decision-making utilizing a variety of methods. Treatment procedures are designed to modify the relationships between antecedent and consequent stimuli that exert influence or control on overt behavior. There is a clear emphasis on what can be observed and measured; cognitive processes and other private events are typically regarded as beyond the domain of scientific analysis. Because ABA directs itself toward the intensive study of the individual, a wide array of intervention and evaluation strategies have been developed and validated scientifically (see Cooper et al. 2020, for a comprehensive review).

Neobehavioristic mediational S-R models derive from classical conditioning and are most frequently associated with the work of Pavlov, Hull, Mowrer, and Miller. In these therapeutic models, hypothetical constructs (e.g., anxiety) are considered to be mediated by cognitive processes, and treatment techniques are designed to put those processes on extinction, resulting in

behavior change. Systematic desensitization is a procedure most commonly associated with this strand of behavior therapy and is used to effectively treat phobias, fears, and other behavioral responses that are triggered by heightened arousal. Social learning approaches are based on the work of Albert Bandura and his colleagues. These approaches, like other behavior therapies, postulate that behavior is controlled by external reinforcement, external stimulus events, and cognitive mediational processes. Importantly, the cognitive mediational processes determine which environmental influences are more or less valued and receive more or less attention. Because the emphasis in social learning theory is on the individual as the agent of change, self-control, self-management, and self-instruction are prominent parts of the treatment plan. Within this paradigm, operant conditioning and especially modeling are more prominent than classical conditioning, although all are considered.

Cognitive behavior therapy (CBT) and cognitive therapy have become among the most prominent and visible of behavior therapy strands, along with applied behavior analysis, over the past 25 years. These approaches are based on the early work of Arnold Lazarus (multimodal therapy), Albert Ellis (rational emotive therapy), and Aaron Beck (cognitive therapy for depression). All share certain core features with contemporary cognitive models, including the understanding that in order to change behavioral responses, one must also alter the prominence or value of the cognitions or thoughts that accompany the response. CBT seeks to develop retrievable memories of more adaptive responses that will then compete with and replace learned problematic responses by suppressing the memory of those responses (Wood et al. 2011). This is achieved through a talk-based therapy process whereby psychoeducation, teaching coping skills, and in vivo exposure are combined to produce specific skills for change, alternative and more adaptive cognitions regarding change, and actual situations in which to practice change. CBT fosters change in behavior by identifying and challenging

irrational beliefs and misinterpretation of events that cause distress (and maladaptive behavioral responses). CBT has been used with individuals with ASD exhibiting anxiety disorders (Chalfant et al. 2007; Sofronoff et al. 2005), anger management problems (Sofronoff et al. 2007), and disruptive behavior (Solomon et al. 2008; Wymbs et al. 2005) as well as for treatment of core social symptoms of ASD (Wood et al. 2009).

The final strand is both a combination of evidence-based treatments and a reaction to earlier iterations of cognitive behavioral models. These so-called third-generation approaches incorporate a broad array of specific procedures, including dialectical behavior therapy (Linehan 1993), functional analytic psychotherapy (Kohlenberg and Tsai 1991), and acceptance and commitment therapy (Hayes et al. 2012). All share the distinction of a general move away from a more cognitive approach and toward a more functional analytic model of assessment and treatment. In many ways, this return to the basics of behavior therapy is consistent with the core features described by Kazdin (1978), namely, that behavior therapists share a common set of assumptions including a focus on current rather than historical determinants of behavior, an emphasis on overt behavior change as the main criterion by which treatment is to be evaluated, specification of dependent variables and treatment parameters in objective terms so that replication is possible, an emphasis on the bilateral relationship between behavior and the environment, a reliance on basic research methods in psychology as a source of hypotheses about treatment and specific therapeutic techniques, and specificity in definition, treatment, and measuring target populations.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavioral Assessment](#)
- [Behaviorism](#)
- [Cognitive Behavioral Therapy \(CBT\)](#)

References and Reading

- Chalfant, A. M., Rapee, R., & Carroll, L. (2007). Treating anxiety disorders in children with high functioning autism spectrum disorders: A controlled trial. *Journal of Autism and Developmental Disorders*, 37, 1842–1857.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2020). *Applied behavior analysis* (2nd ed.). Hoboken: Pearson.
- Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (2012). *Acceptance and commitment therapy: The process and practice of mindful change* (2nd ed.). New York: Guilford.
- Kazdin, A. E. (1978). Behavior therapy: Evolution and expansion. *The Counseling Psychologist*, 23, 34–37.
- Kohlenberg, R. J., & Tsai, M. (1991). *Functional analytic psychotherapy*. New York: Plenum.
- Linehan, M. M. (1993). *Cognitive behavior therapy of borderline personality disorder*. New York: Guilford.
- Sofronoff, K., Attwood, T., & Hinton, S. (2005). A randomized controlled trial of CBT intervention for anxiety in children with Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 46, 1152–1160.
- Sofronoff, K., Attwood, T., Hinton, S., & Levin, I. (2007). A randomized controlled trial of a cognitive behavioral intervention for anger management in children diagnosed with Asperger syndrome. *Journal of Autism and Developmental Disorders*, 37, 1203–1214.
- Solomon, M., Ono, M., Timmer, S., & Goodlin-Jones, B. (2008). The effectiveness of parent child interaction therapy for families of children on the spectrum. *Journal of Autism and Developmental Disorders*, 38, 1767–1776.
- Wood, J. J., Drahota, A., Sze, K., Van Dyke, M., Decker, K., et al. (2009). Brief report: Effects of cognitive behavioral therapy on parent-reported autism symptoms in school-age children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 39, 1609–1612.
- Wood, J. J., Fujii, C., & Renno, P. (2011). Cognitive behavioral therapy in high-functioning autism: Review and recommendations for treatment development. In B. Reichow, P. Doehring, D. V. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-based practices and treatments for children with autism* (pp. 197–230). New York: Springer.
- Wymbs, B. T., Robb, J. A., Chronis, A. M., Massetti, G. M., Fabiano, G. A., et al. (2005). Long-term multimodal treatment of a child with asperger's syndrome and comorbid disruptive behavior problems: A case illustration. *Cognitive and Behavioral Practice*, 12, 338–350.

Behavioral Artistry: The Relationship Between Interpersonal Skills and Effective Treatment Repertoires of Applied Behavior Analysis Practitioners

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Definition

Behavioral Artistry is a repertoire of humanistic, interpersonal behaviors associated with the effective delivery of applied behavior analysis treatment in autism.

Historical Background

In a discussion summarizing 25 years of lessons learned in the development and application of applied behavior analysis practices, Richard Foxx (1998) highlighted a concept from an earlier paper (Foxx 1985) wherein he concluded that the effectiveness of ABA may be negatively impacted by a broad array of deficits in the behavioral repertoires of practitioners. Foxx asserted that there can be important differences in the outcomes achieved by interventionists delivering ABA services in a strictly traditional way (referred to by Foxx as “behavioral technologists”) and those who demonstrate important humanistic, interpersonal behaviors. He observed that some therapists, including individuals with little or no formal behavior analytic training, appear qualitatively better at changing behaviors than their peers with equivalent or even greater levels of training. Foxx described these therapists as “Behavioral Artists” (Foxx 1998, p. 14). Behavioral technologists, according to Foxx, often appear to be practitioners who simply learn a set of scripted,

Behavioral Approaches

► Didactic Approaches

manualized strategies, methods, and procedures and deliver them without much focus on the overall quality of the therapeutic interaction. In contrast to technologists, Behavioral Artists are described as “natural behavior analysts” who demonstrate a set of seven interpersonal characteristics, as well as effective communication skills (Foxy 1998, p. 14).

Foxy hypothesized that there are foundational therapeutic relationship skills associated with the demonstration of “Behavioral Artistry” (BA), including:

- Likes people: Is able to establish rapport; demonstrates concern; wants to facilitate positive change
- Has “perceptive sensitivity”: Pays careful attention to important indicators of client behavior that may be small, subtle, and gradual
- Doesn’t like to fail: Sees difficult clients as a personal challenge to overcome and as an opportunity for the client to succeed
- Has a sense of humor: Recognizes and accepts that much in the educational and human services professions is bizarre, illogical, and humorous
- Looks “for the pony”: Is optimistic and sees behavior change in a “glass half-full” context; always believes programming will be successful; is less likely to burn out
- Is thick-skinned: Doesn’t take negative client actions toward herself or himself personally; maintains objectivity and positivity
- Is “self-actualized”: Does whatever is necessary and appropriate to facilitate and produce positive behavior change; is not under audience control; is creative (Foxy 1985, 1998)

Several of Foxy’s BA components are similar to therapist skills in clinical psychology and counseling and have long been associated with psychodynamic and humanistic/experiential approaches to behavior change. For example, the concept of therapeutic alliance (TA) refers to the collaborative, caring partnership that characterizes a positive client-therapist relationship (Lejuez et al. 2006). TA is seen as a fundamental component of effectiveness within

other therapeutic approaches and has been demonstrated to be a significant predictor of treatment success (Holmqvist 2013; Horvath et al. 2011). Kerns et al. (2018) concluded that a strong therapeutic alliance was related to more positive treatment outcomes in high-functioning children with autism. Similarly, the related concepts of “empathic teaching” in special education (Morgan 1991) and “rapport building” (Shireman et al. 2016) and “compassionate care” (Taylor et al. 2018) in behavior analytic treatment have been highlighted in efficacy literature as essential repertoires among service providers for individuals with disabilities. The influence of empathy, in particular, has been frequently reported within medical and clinical care studies to be a key determinant of positive patient-client relationships and outcomes (e.g., Riess et al. 2012). Nevertheless, the roles and possible significance of these kinds of therapist behaviors in ABA have not been fully explored.

In 2016 Leaf et al. published a review of the skills needed for ABA practitioners to conduct effective programming for individuals with ASD. The authors identified intervention components related to what they describe as a “progressive” and “responsive” approach to the delivery of ABA services. These skills highlight interventionists’ abilities to be flexible and analytical in the implementation of individualized protocols and practices (e.g., while using established EBPs such as discrete trial training and functional analysis) rather than strictly adhering to today’s frequently observed “recipe-based” ABA approach (Leaf et al. 2016, p. 721). Echoing some of the conclusions of Foxy’s research, Leaf and colleagues suggested that the pervasive use of ABA in autism treatment has resulted in changes in its scope and focus, and behavioral interventions have become potentially less effective:

A danger inherent in any large scale, quickly growing area is a loss of focus on meaningful purpose, process, and outcomes. In the field of ABA, this might translate into dogmatic lack of attention to clinical significance, selection of impractical procedures, ritualistic data-collection, over-abundant use of off-putting, dehumanizing terminology, disregard of logistical realities, and insensitivity to consumer issues. (Leaf et al. 2016, p. 728)

Eikeseth (2010) also investigated specific knowledge and skill components necessary to become a technically “competent” provider of early intensive behavioral interventions (EIBI) for children with autism, providing recommendations for assessing and training interventionist skills in the areas of basic intervention, comprehensive curriculum programming, working with families, and supervision. Eikeseth concluded that ineffective EIBI programming is related, in part, to deficiencies in meeting standards of overall program quality: “Highly intensive teaching and supervision will not produce optimal gains if teachers and/or supervisors do not have the necessary qualifications” (Eikeseth 2010, p. 243).

To a large extent, Foxx and other researchers have called attention to the critical need to assess the essential technical skills and related interpersonal characteristics of the individuals delivering ABA services, in order to ensure the future application of ABA as a highly effective and viable treatment approach for persons with ASD. It is possible that the repertoires associated with BA can contribute to sustained improvements in the effective delivery of ABA for individuals with autism and other disabilities.

Current Knowledge

Callahan and colleagues (Callahan et al. 2019) conducted a study to determine if the concept of Behavioral Artistry could be validated and reliably measured using standardized assessments and to determine whether individuals studying and/or working in the field of applied behavior analysis differ from those in other human services professions on important interpersonal skills potentially related to therapeutic effectiveness in autism treatment. In addition, because of the importance of social validity in the selection and effective use of evidence-based treatments in autism (Callahan et al. 2008), these researchers investigated the social validity of characteristics associated with the concept of Behavioral Artistry among the parents of children with ASD. Importantly, Callahan et al. (2019) also assessed preliminary data to determine if there

are differences in the quality of ABA treatment delivered by autism interventionists who have high or low levels of Behavioral Artistry characteristics.

Callahan et al. (2019) determined that the Sixteen Personality Factor Fifth Edition Questionnaire (16PF) instrument could reliably measure Behavioral Artistry characteristics. The 16PF was developed by Raymond B. Cattell in 1949 and is currently in its fifth revision as a widely researched and used, comprehensive, self-report measure of normal adult personality (Institute for Personality and Ability Testing [IPAT] 2009). Designed for individuals aged 16 years and older, the 16PF has been implemented in a variety of research and applied settings (including clinical, counseling, and educational contexts) and has been used to determine and predict levels of creativity, leadership, interpersonal skills, and occupational profiles. The validity and reliability of the 16PF instrument have been well established in more than 4,000 research publications during the past 60 plus years (IPAT 2009). The fifth edition of the 16PF contains 185 multiple-choice items asking simple questions about the respondent’s daily behavior, interests, and opinions. Examples of questions include: “I’d enjoy more being a counselor than an architect (true; false),” and “I believe more in ____ (being properly serious in everyday life; the saying ‘laugh and be merry’ most of the time).” Each question has two narrative choices (“a” or “c”) as well as a “b” choice indicated by a question mark (“?”). Respondents are encouraged to choose the “b” (“?”) response only when neither of the other choices was a better descriptor and are informed that there are no “right” or “wrong” answers.

Responses of the 16PF result in scores on 16 primary personality factors along a bipolar continuum (i.e., each personality factor is represented by two discrete poles, each having a unique, meaningful definition representing a different behavioral profile). For example, on the 16PF personality factor of “Warmth,” respondents could score as being closer to the pole “Reserved, impersonal, distant” or the pole “Warm, outgoing, attentive to others” (IPAT 2009, p. 24).

Using a combination of factor examination, model comparison, and model modification techniques, Callahan et al. determined that eight of the 16PF personality factors are clearly supportive of the BA concept (the factor of “Reasoning,” considered by the test designers to be a brief indicator of intelligence, was omitted from consideration as being largely unrelated to effective ABA intervention). As a result of this statistical modeling, a behavioral artist was hypothesized to represent interpersonal and therapeutic behaviors associated with warmth, emotional stability, liveliness, social boldness, self-assurance, openness to change, self-reliance, and perfectionism.

In order to determine if there was a difference in the level of BA characteristics among university students majoring in ABA and other major areas of study, Callahan et al. conducted an online survey using the 16PF. Undergraduate and graduate students in the majors of ABA, special education, rehabilitation counseling, and other human services majors completed the survey. “Other human services” majors included students in speech and hearing sciences, clinical counseling, psychology, child development, occupational therapy, adapted physical education, and similar majors. Additionally, engineering and computer science students served as a comparison group of persons not expected to pursue professional careers working in human services with individuals with autism or intellectual disabilities.

The 16PF survey results for each respondent were analyzed. For each personality factor, the respondent’s scores indicated whether he or she fell into the BA-compatible pole or the non-BA-compatible pole category. Overall percentages of BA-compatible characteristics were then computed for each respondent. Mean percentages were computed for three main survey respondent groups: (1) Autism Center Group (respondents who were employed part-time or full-time at a university-based autism center delivering ABA therapy); (2) External Group (student respondents not working at the autism center; and (3) Combined Group (all respondents, including undergraduate and graduate students working at the autism center and students external to the autism center).

Finally, Callahan et al. conducted observations of ABA therapists to determine if those with the highest and lowest percentages of BA characteristics looked qualitatively different in their delivery of ABA therapy. The research team posited that therapist behaviors associated with the Foxx BA characteristics “Likes People,” “Thick-Skinned,” “Perceptive Sensitivity,” and “Sense of Humor” could be observed during the delivery of typical discrete trial training (DTT) and naturalistic environment teaching (NET) programming. Operational definitions for each of these BA characteristics were developed. However, during field testing it became apparent that only behaviors associated with “Likes People” occurred frequently enough during therapy sessions to measure meaningfully as a component of BA. It was further observed that behaviors associated with the Foxx characteristics of Thick-Skinned and Sense of Humor were subsumed within the definition of “Likes People.”

“Likes People” was generally defined as observable demonstrations of enjoyment and concern directed toward a client, with four associated behavioral indicators: (a) pleasant facial expression; (b) positive tone of voice; (c) sustained gaze at the client; and (d) body proximity and orientation toward the client. “Likes People” could only be scored during times the therapist was engaged in social interactions with clear communicative and therapeutic intent. The occurrence or nonoccurrence of the four indicators of “Likes People” was scored by data collectors using a partial interval data sheet, on which a 10-minute observation period was divided into 10-second scoring intervals. If the behavioral indicator was observed at any time during an interval, data collectors marked a “+” on the data sheet. A total percentage of occurrence was computed for each of the four behavioral indicators for each observation session. Data collectors were required to demonstrate mastery of the scoring system before beginning BA scoring.

In addition to partial interval scoring of the behavioral indicators of “Likes People,” data collectors recorded a subjective rating of the

therapist's behavior throughout the entire 10-minute therapy session, based on a standardized description of typical examples of the target behavior, as follows: "Likes People will typically appear as a person who is fun, friendly, and child-like; energetic, positive, and affectively expressive; uses appropriate physical touch and gestures; appears attentively interested in what the client is doing; is engaged in activities that demonstrate care for the client's welfare and happiness; demonstrates empathy, respect, and politeness." At the conclusion of a scoring session, data collectors subjectively rated the therapist's "Likes People" behaviors. Behavioral technologist behaviors related to the fidelity of implementation of ABA were also assessed.

The results of the Callahan et al. (2019) study indicate that students majoring in ABA had the lowest overall levels of BA characteristics across all human services majors. Notably, the personality factors of "warmth" and "perfectionism" were significantly lower among ABA majors. Parents of children with autism rated descriptors of BA behaviors as significantly more preferable than non-BA behaviors for the therapists working with their children, providing an indicator of social validation for the concept of Behavioral Artistry by this important consumer group of autism treatments. Importantly, therapists with higher percentages of BA characteristics were rated as delivering higher-quality ABA, including both BA ("Likes People") and behavioral technologist therapeutic behaviors.

Future Directions

Callahan et al. (2019) concluded that the effective practice of ABA for individuals with ASD can be broadened and improved by incorporating BA repertoires into the ongoing delivery of treatment. While acknowledging the preliminary nature and limitations of their research, they identified several key directions for future research. Addressing each of the four questions below will be of paramount importance in further validating the potential impact of BA on the fields of ABA and autism treatment.

1. What are the relationships between the technology and artistry of ABA treatment in autism? For the past several decades, the literature on ABA programming for children with autism has focused almost exclusively on improving the *technology* of treatment. Researchers and practitioners have done an exemplary job identifying evidence-based practices (EBPs) and developing curricula to increase effective autism programming. Indeed, Foxx's seminal articles which introduced the concept of Behavioral Artistry (Foxx 1985, 1998) also included robust emphases on the necessary technological knowledge and skills therapists must possess in order to be effective providers of ABA treatments. In the Callahan et al. (2019) study, therapists with both the highest and lowest levels of Behavioral Artistry demonstrated relatively similar levels of technical competence. Thus, one may conclude that most ABA practitioners, if adequately trained and supervised, can deliver ABA protocols with technological fidelity. However, it can be argued that the repertoires of behavioral technologists, although necessary, are not sufficient to continue to advance the development of the fields of behavior analysis and autism treatment in order to attain maximum, broad-reaching clinical and educational impacts. Researchers must continue to examine the qualities and corresponding behaviors of exemplary behavior analysts, including identifying how components of humanistic therapeutic care may be integrated within the delivery of high-quality ABA treatment. As Taylor et al. (2018) point out, the empirically derived *technical skills* of behavior analysts will always remain a critically important component of client outcomes. Nevertheless, "those methods do not exist separately from relationships with clients and their caregivers" (Taylor et al. 2018, p. 1). Future research should investigate these relationships, and the potential synergies that could result from maximizing technical competence *in coordination with* Behavioral Artistry repertoires. Such research could prove to be a valuable addition

- to the literature on the relationship between therapist interpersonal skills and effective practice (e.g., Anderson et al. 2009; Keijsers et al. 2000; Lambert and Barley 2001).
2. Can the repertoires of Behavioral Artistry be effectively trained? Another important focus for future research is to determine if autism practitioners with lower levels of BA can be taught to consistently demonstrate associated behaviors at a higher level. In addition to recent efforts within the field of ABA (e.g., Lugo et al. 2017; Shireman et al. 2016), other helping professions, such as counseling, have also concluded that the basic skills of establishing therapeutic rapport are, indeed, trainable (Carkhuff 2009; Lambert and Barley 2001). It is possible that by using ABA-based training methods such as Behavioral Skills Training (e.g., Parsons et al. 2012), novice therapists can be taught to recognize skill deficits and receive effective training to remediate them. Crucial additional questions will include how much improvement can be achieved and whether or not the observed changes are clinically significant and appear genuine.
 3. Do the behavioral repertoires associated with Behavioral Artistry improve student/client outcomes? It is important for researchers to demonstrate that the implementation of BA skills results in greater school and life outcomes. Hypothetically and logically, ABA therapists who are warm, attentive, creative, optimistic, and persevering should engage clients instructionally at higher levels and minimize escape-avoidance and problem behaviors, allowing for the more effective delivery of their corresponding technologist repertoires. Nevertheless, future research should examine the interpersonal characteristics and behaviors of ABA practitioners with the intention of identifying more precisely the repertoires and behaviors associated with the most positive client outcomes. The social validation of these outcomes is vitally important, especially for a field which continues to be subject to negative public perceptions and misperceptions, and less than universal acceptability of its methods and language (Critchfield and Reed 2017; Woolfolk et al. 1977). The parent survey results reported by Callahan et al. (2019) suggest that at least this important consumer group supports the inclusion of the interpersonal aspects of Behavioral Artistry as a future hallmark of ABA therapy in autism intervention.
 4. What can the fields of ABA and autism intervention and education do to identify, recruit, and retain practitioners with positive interpersonal behaviors? Expanding the definition of effective ABA practitioners to include the characteristics of Behavioral Artistry can potentially improve the delivery, outcomes, and acceptability of ABA treatment for individuals with ASD. The preliminary results of Callahan et al. (2019) suggest that persons with the highest levels of Behavioral Artistry may often seek other human services professions than ABA in which to apply their therapeutic skills. It is unclear why the students in that study majoring in ABA had the lowest levels of Behavioral Artistry, and, more concerning, the lowest levels of warmth, across all groups of human services providers. However, this is a compelling finding. It could be beneficial for the field of ABA to conduct a large-scale self-study effort to investigate this phenomenon.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Empathy](#)
- [Interpersonal Skills](#)
- [Social Validity](#)

References and Reading

- Anderson, T., Ogles, B. M., Patterson, C. L., Lambert, M. J., & Vermeersch, D. A. (2009). Therapist effects: Facilitative interpersonal skills as a predictor of therapist success. *Journal of Clinical Psychology, 65*, 755–768.
- Callahan, K., Henson, R., & Cowan, A. (2008). Social validation of evidence-based practices in autism by

- parents, teachers, and administrators. *Journal of Autism and Developmental Disorders*, 38, 678–692.
- Callahan, K., Foxx, R. M., Swierczynski, A., Aerts, X., Mehta, S., McComb, M., Nichols, S. M., Segal, G., Donald, A., & Sharma, R. (2019). Behavioral Artistry: Examining the relationship between the interpersonal skills and effective practice repertoires of applied behavior analysis practitioners. *Journal of Autism and Developmental Disorders*, 49, 3557–3570. <https://doi.org/10.1007/s10803-019-04082-1>.
- Carkhuff, R. R. (2009). *The art of healing* (9th ed.). Amherst: Possibilities Publishing.
- Critchfield, T. S., & Reed, D. D. (2017). The fuzzy concept of applied behavior analysis research. *The Behavior Analyst*, 40, 123–159.
- Eikeseth, S. (2010). Examination of qualifications required of an EIBI professional. *European Journal of Behavior Analysis*, 11, 239–246.
- Foxx, R. M. (1985). The Jack Tizzard memorial lecture: Decreasing behaviours: Clinical, ethical, and environmental issues. *Australia and New Zealand Journal of Developmental Disabilities*, 10, 189–199.
- Foxx, R. M. (1998). Twenty-five years of applied behavior analysis: Lessons learned. *Discriminanten*, 4, 13–31.
- Foxx, R. M. (2008). Applied behavior analysis (ABA) treatment of autism: The state of the art. *Child and Adolescent Psychiatric Clinics of North America*, 17, 821–834. <https://doi.org/10.1016/j.chc.2008.06.007>.
- Holmqvist, R. (2013). Therapeutic alliance predicts symptomatic improvement session by session. *Journal of Counseling Psychology*, 60, 317–328.
- Horvath, A. O., Del Re, A. C., Fluckiger, C., & Symonds, D. (2011). Alliance in individual psychotherapy. *Psychotherapy*, 48, 9–16.
- Institute for Personality and Ability Testing. (2009). *16PF fifth edition questionnaire manual*. Champaign: Author.
- Keijsers, G. P. J., Schaap, C. P. D. R., & Hoogduin, C. A. L. (2000). The impact of interpersonal patient and therapist behavior on outcome in cognitive-behavior therapy: A review of empirical studies. *Behavior Modification*, 24, 264–297.
- Kerns, C. M., Collier, A., Lewin, A. B., & Storch, E. A. (2018). Therapeutic alliance in youth with autism spectrum disorder receiving cognitive-behavioral treatment for anxiety. *Autism*, 22, 636–640.
- Lambert, M. J., & Barley, D. E. (2001). Research summary on the therapeutic relationship and psychotherapy outcome. *Psychotherapy*, 38, 357–361.
- Leaf, J. B., Leaf, R., McEachin, J., Taubman, M., Rosales, S., Ross, R. K., ... & Weiss, M. J. (2016). Applied behavior analysis is a science and, therefore, progressive. *Journal of Autism and Developmental Disorders*, 46, 720–731.
- Lejuez, C. W., Hopko, D. R., Levine, S., Gholkar, R., & Collins, L. M. (2006). The therapeutic alliance in behavior therapy. *Psychotherapy: Theory, Research, Practice, Training*, 42, 456–468. <https://doi.org/10.1037/0033-3204.42.4.456>.
- Lugo, A. M., King, M. L., Lamphere, J. C., & McArdle, P. E. (2017). Developing procedures to improve therapist-child rapport in early intervention. *Behavior Analysis in Practice*, 10, 395–401. <https://doi.org/10.1007/s40617-016-0165-5>.
- Morgan, S. R. (1991). The fundamental element of the teaching process: Empathy. In S. R. Morgan & J. Reinhart (Eds.), *Interventions for students with emotional disorders* (pp. 31–49). Austin: Pro-Ed.
- Parsons, M. B., Rollyson, J. H., & Reid, D. H. (2012). Evidence-based staff training: A guide for practitioners. *Behavior Analysis in Practice*, 5, 2–11.
- Riess, H., Kelley, J. M., Bailey, R. W., Dunn, E. J., & Phillips, M. (2012). Empathy training for resident physicians: A randomized controlled trial of a neuroscience-informed curriculum. *Journal of General Internal Medicine*, 27, 1280–1286.
- Shireman, M. L., Lerman, D. C., & Hillman, C. B. (2016). Teaching social play skills to adults and children with autism as an approach to building rapport. *Journal of Applied Behavior Analysis*, 49, 512–531. <https://doi.org/10.1002/jaba.299>.
- Taylor, B. A., LeBlanc, L. A., & Nosik, M. R. (2018). Compassionate care in behavior analytic treatment: Can outcomes be enhanced by attending to relationships with caregivers? *Behavior Analysis in Practice*. <https://doi.org/10.1007/s40617-018-00289-3>.
- Woolfolk, A. E., Woolfolk, R. L., & Wilson, G. T. (1977). A rose by any other name... : Labelling bias and attitudes toward behavior modification. *Journal of Consulting and Clinical Psychology*, 45, 184.

Behavioral Assessment

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Definition

Behavioral assessment is the process of objectively identifying and evaluating units of response (behaviors) and related controlling environmental and organismic variables so that specific behaviors can be better understood and changed (Cooper et al. 2020; Nelson and Hayes 1979). It is pragmatic in nature, in that behavioral assessment seeks to determine and describe underlying functional relationships between behavior and the person in their environment and then uses

that understanding to facilitate the development of new, more adaptive functional responses. By emphasizing objective identification and measurement of environmental and organismic-dependent variables that may influence behavior, behavioral assessment ultimately serves treatment planning and outcome evaluation.

Historical Background

Behavioral assessment has a long past but a relatively short history. With the advent of behavioral approaches to understanding and treating individuals with varying problems (e.g., fears and phobias, depression, anxiety, self-injurious behavior) over the past 60 plus years, behavioral assessment had been somewhat of an informal process until the 1970s when closer attention to those dependent variables that contributed to behavioral treatment success or failure began to receive greater attention from researchers and clinicians. As would be expected, there was an initial emphasis on what behavioral assessment *was not* and in specifying differences between behavioral and so-called traditional or psychodynamic assessment. Those differences were succinctly summarized by Mash (1979), who noted that at a conceptual and applied level, behavioral assessment is characterized by the view that human behavior is predominantly under the control of environmental and organismic events, rather than underlying intrapsychic processes, introspection, or personality traits that are inferred. Further, behavior must be examined in context. While these were radical ideas at the time, particularly as regards the treatment of mood, developmental, and conduct disorders of adults and children, this approach was prescient. As the relationship between brain and behavior is better understood through sophisticated neuroimaging techniques and through advances in neurobiology, genetics, and neurochemistry, it becomes clear that context is everything. These variables, once broadly called *organismic*, now are more precisely described and differentiated. The result is that behavioral assessment procedures are now

better able to help pinpoint functional relationships so that treatment selection and efficacy improve, with greater generalizability beyond the treatment setting.

In practical terms, behavioral assessment evolved initially after behavioral treatments were devised, rather than before it. While this observation helps to understand the recency of more sophisticated assessment strategies, it also provides a context for understanding why so many behavioral interventions for complex psychological disorders have become evidence-based treatments of choice over the past 40 years (e.g., cognitive behavior therapy for individuals with anxiety, depression, and anorexia and bulimia and dialectical behavior therapy for those with borderline personality disorder). Behavioral assessment is rooted in the understanding that behavior must be examined in context, with direct samples taken in multiple settings, utilizing multiple methods of inquiry. With these much more precise, operationalized, and objective formulations, the clinician is able to more accurately specify what is expected or predicted to change and then to evaluate whether change, in fact, occurred after the introduction of treatment. It is this hypothesis-testing process that compliments the rejection of inferred causation and makes the behavioral assessment process inherently objective, dynamic, and responsive to new evidence. Indeed, the reliance on the basics of the scientific method permits the needed flexibility to abandon or modify a treatment approach if it is not working as planned.

Current Knowledge

The technology of behavioral assessment is ever-expanding but is always directed toward understanding behavior functionally (see ► [“Functional Analysis”](#)) through the use of direct and indirect assessment methods. It is important to emphasize, however, that indirect assessment does not imply a reliance on inference. Rather, indirect behavioral assessment methods such as questionnaires (e.g., Questions About Behavioral Function; Paclawsky et al. 2001) and rating scales (Social Responsiveness Scale; Constantino and Gruber

2005) are used in conjunction with direct observation methods to clarify points of behavioral convergence and are themselves designed to measure behaviors that have been more precisely and operationally defined so that interobserver agreement is high.

Identification of target behavior is the first step in a comprehensive behavioral assessment and requires that behavioral form and function be described, including function, topography, duration, frequency, and intensity of the behavior. This is done in such a way that the description becomes an *operational definition*, specifying explicit and precise response parameters. Once completed, determination of controlling variables is undertaken using indirect and direct methods. Indirect methods include third-party interviewing with a structured assessment format such as the functional assessment interview (O'Neill et al. 1990), review of incident reports or permanent products of the behavioral episode, or more informal interviews with parents or caregivers. Direct assessment procedures include direct observation of the target behavior in the natural or analog environment using any number of methods (e.g., momentary time sampling, partial interval recording) as well as descriptive analysis of the behavior using antecedent-behavior-consequence (ABC) analysis. In all cases, the behavioral assessor seeks to describe controlling variables of three types: antecedent stimuli, consequent stimuli, and organismic stimuli. Antecedent stimuli can be discriminative stimuli (they predict the expectation of a particular response because the person has learned that their response is followed by a specific consequence) or elicitors (which evoke automatic, physiological, or emotional responses). These immediate “triggers” help to understand the impact of a particular stimulus event on the person and their behavior.

An assessment of context is undertaken in the form of analysis of setting events and establishing operations. Setting events are variables that influence an ongoing relationship between a stimulus and a response, whereas establishing operations momentarily change the reinforcing value of a discriminative stimulus. There are technical differences between both terms, but both describe antecedent conditions that alter response

probability. For example, when ill or satiated on a specific reinforcing stimulus, a person might respond differently than when healthy or in a deprived state. These are assessed because these variables are more distant from the target behavior (may not occur immediately before the target behavior), but they may persist over time, or exert a cumulative effect, and influence the target behavior. Knowing them highlights a possible point of intervention.

Target behavior can be embedded in a behavioral chain, and assessment for this is important because it may provide an opportunity to intervene at an early point in the chain, thereby interrupting the variables that would normally control the response. Equally important is the assessment of high- and low-probability settings. That is, it is important to understand where and when the target behavior is more or less likely to occur as a method for considering stimulus control.

Behavioral assessment of consequences refers to those stimuli that reliably occur after the target behavior is emitted. These are critical to understand that they represent those contingencies maintaining (reinforcing) the behavior. For behavior change to occur, those contingencies of reinforcement must be modified so that a problematic target behavior is no longer reinforced by the stimulus maintaining it, clearing the way for an alternative, more adaptive response to be reinforced and established to replace the problem behavior.

Organismic stimuli have received somewhat less attention from researchers over the years, but with the advent of more sophisticated technologies (e.g., with neuroimaging or for genetics), a greater emphasis is being placed on the relationship between physical, biological, and neurological status and behavioral expression. For example, identification of lesions in a specific area of the brain (as would occur with tuberous sclerosis) may help the clinician better understand the context for a challenging behavior. This would not necessarily reduce the importance of addressing the target behavior, but it would likely support an interdisciplinary approach to treatment.

Two final areas must be considered in behavioral assessment if the assessment results are to fully inform treatment planning. The first is an assessment of preferences and reinforcers, and

the second is the identification of functionally equivalent behavior(s) that can be taught as a replacement for the target behavior to be reduced or eliminated. There are a number of empirically based strategies for evaluating which stimuli are preferred by a client. They (or caregivers) can be queried, observed, or placed in a formal trial-based assessment environment. An example of the latter is a “forced choice” or paired stimulus presentation whereby two items are presented to a client, all matched randomly. Preferences are determined by frequency of selection of specific items (see Fisher et al. 1992 for an example). In contrast to preferences, reinforcers can only be identified by a functional test. That is, when presented, a stimulus is only considered a reinforcer if it increases the likelihood of reoccurrence of the behavior it followed. There are several ways to conduct a reinforce assessment including those based on concurrent, progressive ratio, or multiple schedules (see Cooper et al. 2020, for examples).

If behavior change is to be achieved, generalized, and maintained, it must gain for the client the same functional outcome, but with greater ease and efficiency, and be more socially desirable and valid than the problem behavior that it replaces. In short, the replacement behavior must work better and faster and be useful and valued in a large number of environments. Determining which behavior to select as the replacement is an essential task in behavioral assessment for several reasons. If left unaddressed, the client may well substitute yet another (and potentially undesirable) behavior in place of the target behavior that has been reduced because the functional need for the behavior still exists. For example, if hitting another person has been a successful means of escaping from a task demand and hitting is reduced without concurrent teaching of a replacement skill that serves the same function, the client may substitute self-injury as a means of escape. If a replacement skill is selected but it is not functionally equivalent, the client will not have an alternative that serves the same functional purpose. In this case, the new skill may be acquired but the target behavior is not reduced. Finally, if the replacement skill to be taught is functionally equivalent, but requires more response effort, or is not reinforced on a sufficiently dense schedule

of reinforcement, then it is less likely to be demonstrated by the client and less likely to serve as a replacement in the long run. In the final analysis, the treatment of behavior problems is better understood as the effective teaching of functionally equivalent, more adaptive replacement skills. This component of the assessment is critical.

The final component of behavioral assessment is a functional demonstration of the relationship between those antecedent, consequent, and organismic variables and the target behavior. This process is termed functional behavior assessment if done without a set of empirical analog conditions and functional analysis if implemented with those analog conditions. Both components are described in detail elsewhere in the *encyclopedia*.

When complete, the behavioral assessment process informs treatment planning and decision-making. In contrast to other, more traditional (and nonbehavioral) assessment approaches, however, behavioral assessment is ongoing. That is, while it does serve a predictive function by helping to elucidate relevant variables that impact the target behavior, it also serves a formative function (informing ongoing decision-making through analysis of treatment effects) as well as a summative function (providing a framework for understanding the target behavior from the point of first identification through resolution and replacement).

Future Directions

If advances in behavioral assessment over the past four decades have emphasized anything, it is that the process is dynamic and data-driven. Whether through the development of more sensitive rating scales for problems experienced by those with ASD or use of microtechnologies to more precisely measure small units of response, the likelihood that future iterations of behavioral assessment will better support treatment planning is without question. Ongoing work in several areas will be especially useful. The continuing analysis of antecedent stimuli, and particularly establishing operations, will continue to support precision intervention. A better understanding of the relationship between information processing

deficits, including those that impact academic performance, will support the development of more sensitive replacement skills that include curriculum modifications and accommodations. Recognizing that exceptional behavioral assessment science and technology is no guarantee that utilization or implementation will proceed correctly, future efforts to better understand the contingencies motivating organizations and systems serving those with ASD will be very valuable.

The availability of sophisticated technologies to objectively evaluate small but significant units of responding in persons presents many exciting opportunities. While the relationship between exciting new findings related to eye gaze in very young infants and toddlers with ASD (Klin et al. 2009) and later social development has yet to translate into intervention protocols demonstrating long-term effects, this area seems one of the top candidates for a marriage of technology with behavioral science. Further, neuroimaging technologies (e.g., fMRI) can map specific brain responses to presented stimuli, and an exciting next step would be to evaluate whether behavioral treatment effects demonstrated through overt behavior lead to discernable change in brain functioning and whether collateral changes are also noted neurologically. Finally, as basic science continues to articulate ways in which those with ASD are (and are not) different from those who are neurotypical, the opportunity for greater interdisciplinary behavioral assessment at the neurological, genetic, and biological levels is exciting. Behavioral assessment is built on the science of specification, not speculation, and good measurement across disciplines has a synergistic effect. The next decades will be exciting ones.

See Also

- [Behavior Rating Scale \(BRS\)](#)
- [Behavior Therapy](#)
- [Functional Analysis](#)
- [Functional Analysis Screening Tool](#)
- [Motivation Assessment Scale](#)

References and Reading

- Constantino, J. N., & Gruber, C. P. (2005). *Social Responsiveness Scale (SRS)*. Los Angeles: Western Psychological Services.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2020). *Applied behavior analysis* (3rd ed.). Hoboken: Pearson Education.
- Fisher, W. W., Piazza, C. C., Bowman, L. G., Hagopian, L. P., Owens, J. C., & Slevin, I. (1992). A comparison of two approaches for identifying reinforcers for persons with severe and profound disabilities. *Journal of Applied Behavior Analysis*, 25, 491–498.
- Klin, A., Lin, D. J., Gorrindo, P., Ramsey, G., & Jones, W. (2009). Two-year-olds with autism fail to orient towards human biological motion but attend instead to non-social, physical contingencies. *Nature*, 459, 257–261.
- Mash, E. J. (1979). What is behavioral assessment? *Behavioral Assessment*, 1, 23–29.
- Michael, J. (1993). Establishing operations. *The Behavior Analyst*, 16, 191–206.
- Nelson, R. O., & Hayes, S. C. (1979). Some current dimensions of behavioral assessment. *Behavioral Assessment*, 1, 1–16.
- O'Neill, R. E., Horner, R. H., Albin, R. W., Storey, K., & Sprague, J. R. (1990). *Functional analysis of problem behavior: A practical assessment guide*. Sycamore: Sycamore Publishing.
- Paclawsky, T. R., Matson, J. L., Rush, K., Smalls, Y., & Vollmer, T. (2001). Assessment of the convergent validity of the questions about behavioral function scale with analogue functional analysis and the motivation assessment scale. *Journal of Intellectual Disability Research*, 45, 484–494.

Behavioral Assessment Scale of Oral Functions in Feeding

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Description

This rating scale assists practitioners to establish a baseline of oral functioning and feeding as well as measure change over time. It should be

utilized in conjunction with an assessment that measures oral structure and muscle tone in children. This scale provides visual feedback of progress as well as assists to plan and justify feeding therapy.

The rating scale includes the following oral motor skills:

- Jaw closure
- Loop closure over a spoon
- Tongue control
- Lip closure while swallowing
- Swallows food without excess loss
- Chews food (tongue/jaw control)
- Sips liquids
- Swallows liquids without excess loss
- Swallows food without coughing

Historical Background

This rating scale was developed by Margret Stratton and published in the *American Journal of Occupational Therapy* in November 1981. The rating scale was developed for people with multiple handicaps and developmental disabilities. Ms. Stratton utilized this rating scale within the J.N. Adam Developmental Center in Perrysburg, New York.

Psychometric Data

This is a criterion-based rating scale. Standard scores are not calculated. The client's oral motor functioning is rated on a scale from 0 to 5 with 5 being normal and 0 being disordered or passive movement.

References and Reading

Stratton, M. (1981). Behavioral assessment scale of oral functions in feeding. *American Journal of Occupational Therapy*, 35(11), 719–721.

Behavioral Curricula

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Definition

Broadly defined, curriculum refers to the content and order of instruction, the instructional strategies used to teach this content, and any assessment or other materials used to implement the educational program (Olley 1999, 2005). Curriculum is also structured by a conceptual framework to guide the treatment model in addressing the core deficits of autism spectrum disorder (ASD) and promoting developmental outcomes (Wong et al. 2015). **Behavioral curricula** are a specific type of curriculum that incorporates behavioral principles (e.g., an emphasis on functional behavior, operationalizing behaviors and objectives, and measuring behavior). Behavioral curricula recognize the importance of individualizing the content, sequence, and method of instruction to best meet each individual's needs (Gould et al. 2011). They also tend to target the reduction and replacement of interfering behaviors (e.g., stereotypic behavior, tantrums; Olley 2005). The characteristics of behavioral curricula are especially appropriate for children with ASD for a couple of reasons. First, behavioral strategies have been found to improve the communication, social, and other skills of children with ASD (Schreibman and Ingersol 2005; Wong et al. 2015). Second, children with ASD differ in their individual strengths and weaknesses and are believed to benefit from individualized educational programs (Olley 1999).

Historical Background

Behavioral treatment approaches are the most common treatment programs for ASD (Weitlauf et al. 2014). Accordingly, recent decades have seen a marked increase in the development of educational curricula for children with ASD (Olley 2005; Wong et al. 2015). Researchers and practitioners have created and evaluated a number of comprehensive and focused (e.g., language, social skills, or academic) curricula (Wong et al. 2015). Many of these have been behaviorally based curricula.

Current Knowledge

Treatment goals for children with ASD include improving social behavior and reducing interfering behavior to increase functional skills, social integration, and independence (Myers and Johnson 2007). Like all curricula, behavioral curricula can use a variety of instructional strategies to target language, social, academic, adaptive, and other skills (Olley 2005). Behavioral curricula incorporate behavioral principles including an emphasis on functional behavior, operationalizing behaviors and objectives, measuring behaviors, individualizing programs to meet each child's needs, and targeting the acquisition of prerequisite skills and the reduction of interfering behaviors. These components are briefly described below.

Functional Behavior

Behavioral curricula focus on functional behaviors. Functional behaviors are behaviors that are useful to the individual. More specifically, behaviors are considered functional if they allow the individual to better navigate his or her current environment, are required to learn or likely to lead to the acquisition of other functional behaviors, increase the individual's ability to navigate other beneficial environments (e.g., general education classrooms), or make others more likely to interact with the individual (e.g., eliminating disruptive or aggressive behaviors may make peers

more willing to initiate interactions with the individual; Cooper et al. 2007). Many individuals with ASD require treatment in numerous areas. Instructors should attempt to identify and target behaviors that are most useful to the individual's current functioning (Cooper et al. 2007). To determine which behaviors would be most useful, instructors should observe the individual in his or her natural environment and include parents and others familiar with the individual (Cooper et al. 2007; Olley 2005).

Operationalizing Behaviors and Objectives

Behavioral curricula emphasize the importance of operationalizing behaviors and objectives. All targeted behaviors should be objectively, clearly, and completely defined. To be objective, the behavior should be described in observable terms. To be clear and complete, the definition should provide comprehensive criteria for behaviors that will be included or excluded (Cooper et al. 2007). Defining targeted behaviors in this way allows teachers and other people working with the individual to count the occurrence of the behaviors and track student progress.

Similarly, behavioral programs operationalize objectives or student goals. For each targeted behavior, there should be specific and objective criterion for mastery (Gould et al. 2011). This criterion should reflect the level of competence that allows the individual to use the behavior to successfully navigate his or her natural environments (Cooper et al. 2007).

Measuring Behavior

Behavioral curricula place a strong emphasis on measuring behavior. Clear and comprehensive operational definitions of targeted behaviors allow teachers and others working with the individual to measure the individual's demonstration of behaviors. An initial measure of the individual's ability allows the instructors to determine his or her current level of ability. Measuring the

targeted behaviors during intervention provides instructors with objective information on student progress (Cooper et al. 2007).

Individualization

Behavioral curricula are usually designed to be individualized and meet each individual's needs (Najdowski et al. 2014; Olley 2005). This may be especially important for individuals with ASD, who share common areas of impairment but demonstrate considerable variability in their abilities and deficits. Measuring the individual's behavior allows instructors to identify that individual's need and focus the curricula accordingly. Regular measurement of the individual's progress provides instructors with information on that individual's response to different instructional strategies. Again, this information can be used to identify instructional strategies that are more effective for that individual student and to update their program content as necessary (Cooper et al. 2007).

Targeting Prerequisite Skills and Interfering Behaviors

Many individuals with ASD lack basic prerequisite skills and demonstrate interfering behaviors. Behavioral curricula tend to target these behaviors early in an individual's program. Prerequisite skills refer to behaviors that facilitate later learning. For example, behavioral programs often target compliance (i.e., following the instructor's instructions), nonverbal and verbal imitation, and attending behaviors (e.g., remaining seated, focusing on presented stimuli; Olley 2005). Consistently demonstrating these and related skills allows the individual to benefit from instruction and facilitates the acquisition of later skills.

Interfering behaviors hinder the individual's ability to learn. These may be inappropriate behaviors, such as stereotypy, aggression, self-injury, or other issues, including sleep disturbances or dietary concerns (Olley 2005). These

and related behaviors and issues affect the individual's ability to participate in and concentrate on instruction. Reducing or eliminating these inappropriate behaviors or concerns increases the individual's ability to focus on and benefit from instruction.

Empirical Support

The components of behavioral curricula are consistent with current best practices for ASD treatment. Existing empirical evidence indicates that effective programs use assessment and progress monitoring to individualize program content and instruction to meet the individual's needs and facilitate his or her independence in his or her natural environments. Therefore, it is recommended that programs for individuals with ASD include these elements (Crimmins et al. 2001; National Research Council 2001; New Jersey Department of Education 2004). Further, there is a large and growing body of research indicating that behavioral strategies can improve the language, social, and other skills of children with ASD. Because behavioral curricula incorporate these components and strategies, there is reason to believe that they may be effective.

However, there is relatively little research examining the effectiveness of curricula content (Olley 1999). More specifically, there is a lack of research examining how specific content and sequence of instruction affect the progress and long-term outcomes of children with ASD. Because of the importance of individualizing each student's program, the necessity of this type of research is unclear (Olley 2005). However, more information could be useful in developing effective programs that best facilitate individual's progress.

Additional research is also needed to validate different behavioral curricula. Researchers and program personnel have conducted studies examining the effects of or outcomes associated with different behavioral curricula (e.g., Arick et al. 2003). However, no single curricula has the empirical support required to meet the criteria for an efficacious treatment (Olley 1999, 2005).

Existing Behavioral Curricula

There are numerous behavioral curricula designed for children with ASD and other disabilities. Three of these are described below. However, these descriptions only provide a brief overview of the program. For more complete information, please refer to the program manuals or other references.

The Early Start Denver Model (ESDM)

The Early Start Denver Model (ESDM; Rogers and Dawson 2010) is a comprehensive and evidence-based behavioral developmental intervention for young children with ASD. Goals of ESDM span all developmental domains with an emphasis on targeting the core deficits of ASD and teaching behaviors in a typical developmental sequence. Research on using the ESDM curriculum and treatment approach has shown gains for young children with ASD in IQ, expressive and receptive language, and social behavior (Dawson et al. 2010). After 1–2 years of treatment, children have also experienced diagnostic shifts toward a milder diagnosis, and electroencephalography (EEG) measures of their engagement and cognitive processing were comparable to typically developing children (Weitlauf et al. 2014).

The structure of ESDM programs is a blend of behavioral, play-based, and relationship-based approaches that are organized by empirical knowledge on child development and ASD. The standardized ESDM curriculum uses the *Early Start Denver Model Curriculum Checklist* assessment to guide individualized behavioral programs. Teaching strategies used are empirically supported methods of applied behavior analysis, and data are used to monitor progress and inform modifications to the program.

There are several core components of ESDM, including a focus on interpersonal interactions and positive affect, reciprocal and functional imitation with facial expressions and objects, shared engagement with joint activities, verbal and non-verbal communication training, and embedding instruction within naturally occurring play and daily routines (Rogers and Dawson 2010).

Each ESDM program has a primary therapist in the home who has ESDM training and holds a certification. Along with a multidisciplinary team, the therapist develops a partnership with parents by teaching strategies to use in the home during daily routines and play (Rogers and Dawson 2010). The ESDM curriculum also offers a companion manual for parents, *An Early Start for Your Child with Autism* (Rogers et al. 2012), that can be used outside of sessions to support the program.

Strategies for Teaching Based on Autism Research (STAR)

The Strategies for Teaching Based on Autism Research (STAR; Arick et al. 2004) program provides instructors with assessment materials, lesson plans, activities, materials, and data collection systems (Arick et al. 2004). The program targets children's receptive, expressive, and spontaneous language; adaptive skills; academics; play; and social skills. These skills are targeted via discrete trial training (DTT), pivotal response training (PRT), and functional routines. All three of these are empirically supported behavioral strategies. The STAR program also recognizes the importance of and provides teachers with strategies for promoting skill integration and generalization.

Research indicates that children with ASD who are exposed to the STAR program do make progress. Special education professionals provided teachers and other staff members with yearly workshops that used the STAR program materials to review DTT, PRT, functional routines, and data collection strategies. Over the next 12–16 months, many of the teachers and staff members' students with ASD demonstrated significant improvement in language, social skills, academics, and autonomy (Arick et al. 2003). Although promising, more research is needed to determine the extent to which the STAR program caused this progress.

The Verbal Behavior Milestones Assessment and Placement Program (VB-MAPP)

The Verbal Behavior Milestones Assessment and Placement Program (VB-MAPP; Sundberg 2008) includes an assessment, curriculum guide, and

progress monitoring system that are used to develop language programs for young children with ASD and other language delays (Sundberg 2008). The targeted skills, assessment, and curriculum suggestions are derived from B. F. Skinner's work on verbal behavior, developmental research, and empirically based behavioral principles and strategies (Sundberg 2008).

The VB-MAPP is composed of the milestone assessment, the barriers assessment, the transition assessment, task analysis and skills tracking, and placement and IEP goals (Gould et al. 2011). The milestone assessment examines the child's current language and related abilities. The barriers assessment is intended to identify existing interfering behaviors or absent prerequisite skills that could affect the child's ability to learn. The transition assessment evaluates skills that the child needs to transition to and succeed in new and less restrictive environments. The task analysis and skills tracking system operationally defines over 900 skills from the different targeted areas. This can provide more detailed data about the child's initial abilities and progress and guide program development. After assessing the child, instructors can consult the placement and IEP section for recommendations for the child's goals and educational settings.

The VB-MAPP has undergone field testing. However, more research is needed to establish its psychometric properties and examine its effectiveness (Gould et al. 2011).

Future Directions

There has been an increase in the development of and research examining behavioral curricula for children with ASD. However, many of these studies have limited internal validity, small samples, and examine program instruction and content together (Arick et al. 2003; Olley 1999). Future research should address these issues. Longitudinal research should also be used to examine the long-term effectiveness of different programs.

In addition, many of the existing behavioral curricula are designed for young or high-functioning children with ASD. There is a need

to develop empirically supported behavioral curricula for older and low-functioning individuals with ASD (Olley 1999, 2005).

These and related areas of research will provide researchers and practitioners with the information needed to develop effective behavioral curricula for individuals with ASD. This information will also help instructors to select appropriate interventions for individuals with ASD. Doing so will facilitate individuals' progress and promote independent adult outcomes.

See Also

- ▶ Curriculum
- ▶ Pivotal Response Training

References and Reading

- Arick, J. R., Young, H. E., Falco, R. A., Loos, L. M., Krug, D. A., Gense, M. H., et al. (2003). Designing an outcome study to monitor the progress of students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 18, 75–88.
- Arick, J. R., Loos, L., Falco, R., & Krug, D. A. (2004). *The STAR program: Strategies for teaching based on autism research, levels I, II, and III*. Austin: PRO-ED.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Merrill/Prentice Hall.
- Crimmins, D. B., Durand, V. M., Theurer-Kaufman, K., & Everett, J. (2001). *Autism program quality indicators: A self-review and quality improvement guide for schools and programs serving students with autism spectrum disorders*. <http://www.p12.nysed.gov/specialed/autism/apqi.htm>. Retrieved 12 May 2011.
- Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., . . . Varley, J. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The Early Start Denver Model. *Pediatrics*, 125(1), e17–e23.
- Gould, E., Dixon, D. R., Najdowski, A. C., Smith, M. N., & Tarbox, J. (2011). A review of assessments for determining the content of early intensive behavioral intervention programs for autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5, 990–1002.
- Myers, S. M., & Johnson, C. P. (2007). Management of children with autism spectrum disorders. *Pediatrics*, 120, 1162–1182.
- Najdowski, A. C., Gould, E. R., Lanagan, T. M., & Bishop, M. R. (2014). Designing curriculum programs for children with autism. In *Handbook of early*

- intervention for autism spectrum disorders (pp. 179–204). New York: Springer.
- National Research Council. (2001). *Educating children with autism (Committee on Educational Interventions for Children with Autism, Division of Behavioral and Social Sciences and Education)*. Washington, DC: National Academy Press.
- New Jersey State Department of Education. (2004). *Autism program quality indicators: A self-review and quality improvement guide for programs serving young students with autism spectrum disorder*. <http://celebratethechildren.org/old/Documents/Indicators.pdf>. Retrieved 12 May 2011.
- Olley, J. G. (1999). Curriculum for students with autism. *School Psychology Review*, 28(4), 595–607.
- Olley, J. G. (2005). Curriculum and classroom structure. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 863–881). Hoboken: Wiley.
- Partington, J. W. (2008). *The assessment of basic language and learning skills-revised: Scoring instructions and IEP development guide*. Pleasant Hill: Behavior Analysts.
- Partington, J. W., Bailey, A., & Pritchard, J. K. (2010). *Data on the developmental patterns of specific language and learning skills of typically developing children as measured by the ABLLS-R*. <http://www.behavioranalysts.com/data.pdf>. Retrieved 30 June 2011.
- Rogers, S. J., & Dawson, G. (2010). *Early start Denver model for young children with autism: Promoting language, learning, and engagement*. New York: Guilford Press.
- Rogers, S. J., Dawson, G., & Vismara, L. A. (2012). *An early start for your child with autism: Using everyday activities to help kids connect, communicate, and learn*. New York: Guilford Press.
- Schreibman, L., & Ingersol, B. (2005). Behavioral interventions to promote learning in individuals with autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 882–896). Hoboken: Wiley.
- Sundberg, M. L. (2008). *Verbal behavior milestones assessment and placement program (VB-MAPP)*. Concord: AVB Press.
- Weitlauf, A. S., McPheeters, M. L., Peters, B., Sathe, N., Travis, R., Aiello, R., . . . Warren, Z. (2014). *Therapies for children with autism spectrum disorder: Behavioral interventions update* [Internet]. Rockville: Agency for Healthcare Research and Quality (US); 2014 Aug. (Comparative Effectiveness Review, No. 137.) Introduction. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK241433/>
- Wong, C., Odom, S. L., Hume, K. A., Cox, A. W., Fettig, A., Kucharczyk, S., et al. (2015). Evidence-based practices for children, youth, and young adults with autism spectrum disorder: A comprehensive review. *Journal of Autism and Developmental Disorders*, 45, 1951–1966.

Behavioral Development Questionnaire

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Synonyms

BDQ; Wing subgroups questionnaire (WSQ)

Description

The Behavioral Development Questionnaire (BDQ) is a measure that assesses several behavioral domains of autism spectrum disorders (ASD) in an attempt to subclassify individuals on the autism spectrum based on their behavioral topography. It is based on the subclassification scheme proposed by Wing and colleagues, which identified the four following ASD subtypes: aloof, passive, active-but-odd, and normal (Wing and Gould 1979, please see section “[Historical Background](#),” below).

The behavioral domains assessed by the BDQ focus on quality of social interaction but also include symbolic play, motor imitation, nonverbal and verbal communication, daily routines, stereotyped behavior, and motor coordination (Castelloe and Dawson 1993). The BDQ consists of 13 groups of four descriptions of behavior, each description corresponding to one of the four ASD subgroups. Parents are asked to rate each description on a 7-point Likert scale with regard to how well it describes their child (0 = never, 6 = always). Additionally, for each group of items, parents are asked to select the one description that best describes their child. Only the item-level ratings are used in scoring the BDQ.

As each description corresponds to a social subtype, the BDQ is scored by summing the ratings for each subtype. Missing items are prorated based on the average score of available items. The child is assigned to the subtype for which he or she received the highest score.

Historical Background

As stated previously, the BDQ is based on the ASD subtyping system developed by Lorna Wing and Judith Gould (1979). Wing and Gould's seminal paper (1979) was an epidemiological study of all children living in Camberwell, London, aged under 15 years who showed ASD-like impairments. The researchers found that the social impairment that characterized this group was expressed in one of four ways. *Social aloofness* was characterized by indifferent social behavior. *Passive interaction* involved the absence of spontaneous approach behavior but the acceptance of approaches made by other children. *Active but odd* interaction included children who spontaneously approached other children but their interactions were one-sided and characterized by repetitive preoccupation with certain phrases or topics of conversation. The fourth group was the *appropriate interaction* subtype, which included children whose social interactions were normal for their level of cognitive development. These groups were proposed to exist on a continuum, with the aloof individuals representing the most severe end of the spectrum of impairment, and the active but odd individuals at the mild end.

It was then hypothesized that individuals with ASD could be subclassified based on these categories of social impairment. Furthermore, Wing proposed that these social classifications would correlate with other patterns of behavior. Indeed, the subtypes were examined and it was found that patterns of behavior tended to occur together. The aloof group comprised the highest proportion of children with autism and was significantly associated with a history of Kanner's (1943) "typical" autism (socially aloof, repetitive routines, speech characterized by reversal of pronouns and idiosyncratic phrases). Patterns of abnormal behavior were also evident between the three groups. Stereotyped and repetitive behavior characterized the aloof group, and repetitive speech and behavior patterns were seen more frequently in the passive and active but odd groups (Wing and Gould 1979).

Since the inception of Wing's subclassification system, it has been accepted by the field as

providing rich clinical descriptions of individuals with ASD (Volkmar and Klin 2005). However, subgroups have largely been assigned based on clinical impression rather than in a systematic fashion. The goal of the BDQ was to create a standardized measure for subclassifying children according to the Wing subtyping system.

Psychometric Data

In the initial study of the BDQ, the questionnaire was completed by parents of children with autism ($n = 34$) or PDD-NOS ($n = 6$) between the ages of 4 and 20 years (Castelloe and Dawson 1993). Separately, clinicians assigned the children to a Wing subtype based on a 30-min observation. Agreement between the BDQ classification and classification based on the clinical observation was good, at 73%. Further analyses revealed that subtype assignment by the clinician was the most powerful predictor of BDQ assignment, indicating good external validity for the BDQ.

The authors also examined the consistency of parents' ratings across the 13 groups of descriptions, to assess the degree to which each subtype was rated in the same manner across behavioral domains. These analyses revealed that for all three subgroups, parents ranked the descriptions in a consistent manner. Correlations between the summary scores were computed to assess the extent to which each subtype was distinct from the others. The correlation between the aloof and passive groups was $-.02$, between the passive and active but odd groups it was $-.17$, and between the aloof and active but odd groups it was $-.70$. The high negative correlation between the aloof and active but odd groups was interpreted as evidence that these subtypes do in fact fall at two ends of a continuum.

In terms of behavioral correlates of the Wing subtypes, Castelloe and Dawson (1993) found that subtype classification was significantly related to mental age and score on the Childhood Autism Rating Scale (CARS), a measure of ASD symptoms (Schopler et al. 1986). The aloof group had the lowest mean mental age, the passive group occupied the intermediate position, and the active

but odd group had the highest mean mental age. CARS scores indicated a similar trend: the aloof group had the most severe ASD symptomatology, the active but odd group had the least severe ASD symptoms, and the passive group occupied the intermediate position. A trend in the same direction was seen for IQ, but it did not reach significance.

Overall, the data support the validity of the BDQ for classifying children with ASD into subgroups based on Wing's classification system.

Clinical Uses

The BDQ has been recommended as a useful addition to the assessment and treatment planning process (Powers 2005). Categorization of individuals with ASD according to social impairments allows for prediction of corresponding behavioral and cognitive differences (Castelloe and Dawson 1993). It has been suggested that the utility of such subtyping within ASD has the greatest clinical implications for the planning of treatment services (Wing and Attwood 1987).

See Also

- [Spectrum/Continuum of Autism](#)
- [Subtyping Autism](#)
- [Wing, Lorna](#)

References and Reading

- Castelloe, P., & Dawson, G. (1993). Subclassification of children with autism and pervasive developmental disorder: A questionnaire based on Wing's subgrouping scheme. *Journal of Autism and Developmental Disorders*, 23, 229–241.
- Kanner, L. (1943). *Childhood psychosis: Initial studies and new insights*. Washington, DC: Winston.
- Powers, M. D. (2005). Behavioral assessment of individuals with autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 817–830). Hoboken: Wiley.
- Schopler, E., Reichler, R. J., & Renner, B. R. (1986). *The childhood autism rating scale (CARS) for diagnostic screening and classification of autism*. New York: Irvington.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 5–41). Hoboken: Wiley.
- Wing, L., & Attwood, A. (1987). Syndromes of autism and atypical development. In D. J. Cohen & A. Donnellan (Eds.), *Handbook of autism* (pp. 3–17). New York: Wiley.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11–29.

Behavioral Disorder

- [Conduct Disorder](#)

Behavioral Flexibility

- [Treatment for Higher-Order Restricted, Repetitive Behaviors](#)

Behavioral Health Rehabilitation (BHR) Services

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Definition

Behavioral Health Rehabilitation Services (BHRS) is a term used for a specific application of a Medicaid-funded interdisciplinary approach for a child or adolescent diagnosed with a serious emotional or behavioral disorder. The type of approach is sometimes referred to as a “wrap-around approach.”

Several states, and primarily Pennsylvania, support the provision of Behavioral Health

Rehabilitation Services when a licensed psychologist has deemed the service medically necessary as part of a Medicaid-funded Early and Periodic Screening, Diagnosis and Treatment (EPSDT) service. “The Early and Periodic Screening, Diagnostic, and Treatment (EPSDT) service is Medicaid’s comprehensive and preventive child health program for individuals under the age of 21. EPSDT was defined by law as part of the Omnibus Budget Reconciliation Act of 1989 (OBRA ‘89) legislation and includes periodic screening, vision, dental, and hearing services” (Centers for Medicare & Medicaid Services n.d.). The Medicaid EPSDT regulations provide for the provision of other necessary health care, when it will “correct or ameliorate defects, and physical and mental illnesses and conditions discovered by the screening services” (Centers for Medicare & Medicaid Services n.d.).

Historical Background

The United States Congress’s Omnibus Reconciliation Act of 1989 created a Medicaid service called the Early and Periodic Screening, Diagnosis, and Treatment (EPSDT) services. During the 1990s, several states, most notably the state of Pennsylvania’s Department of Public Welfare, supported the provision of Behavioral Health Rehabilitation Services when identified as medically necessary by a licensed psychologist or psychiatrist as part of an EPSDT evaluation. While, in theory, Behavioral Health Rehabilitation Services can be provided in any state as part of their Medicaid EPSDT services, Pennsylvania has been the most consistent at regularly providing this service.

Behavioral Health Rehabilitation (BHR) Services is essentially a form of wraparound services that is supported through Medicaid funding. The use of Behavioral Health Rehabilitation Services develops during the 1990s concurrent with the national development of wraparound services for youth with complicated mental health and behavioral needs in several discreet places throughout

the country. See the historical section of the Encyclopedia listing for wraparound services for more information on the development of the wrap-around philosophy.

Rationale or Underlying Theory

The key provision in Medicaid-funded Behavioral Health Rehabilitation Services is that a licensed psychologist or psychiatrist has determined that a child or adolescent has a medical need for the services in order to ensure the correction or amelioration of defects and physical and mental illnesses and conditions.

Behavioral Health Rehabilitation Services are individualized and interdisciplinary services for a child or adolescent with a significant behavioral health disability provided in the natural settings of their family or local community. An essential feature of BHR services is that they are designed and delivered at the sites where the problematic behaviors occur. Based on a philosophy consistent with wraparound services, the goal of BHR services is not to try to understand problematic behaviors in the abstract, but rather to provide direct intervention in the natural context with the professionals designing the interventions able to learn and respond directly from the child’s behavioral responses to the interventions.

Goals and Objectives

Behavioral Health Rehabilitation (BHR) Services are services based on a wraparound philosophy designed to provide comprehensive treatment to children and adolescents with a serious emotional or behavioral disorder who cannot make progress with the usual array of discreet services. An essential feature of BHR services is the coordination, or wraparound, of services in the child or adolescent’s natural environments of home, school, and the local community.

A primary goal of BHR services is to develop a natural community support network, and self-regulating behaviors on the part of the child or

adolescent, that can be maintained with the ordinary array of services. Thus, a key outcome for BHR services is to eliminate the need for BHR services.

BHR clinicians and other clinicians in the child or adolescent's natural environment, as well as with other concerned community members, work with the family of the child receiving services. Other concerned community members may include school administrators and teachers, members of a family's religious congregation, and civic officials, as well as staff at community, health, or recreation centers. BHR clinicians aid in developing and guiding a natural community support network. As a team, they develop individualized goals to promote appropriate behavior, activities, and academic and social skills in the child or adolescent's natural home, school, and community environments.

Treatment Participants

Treatment participants for Behavioral Health Rehabilitation (BHR) Services are children or adolescents who have been diagnosed with a severe emotional or behavioral disorder by a psychiatrist or psychologist after a face-to-face clinical evaluation. The prescribing clinician must identify that the BHR services are necessary in order to ameliorate or correct the identified severe emotional or behavioral disorder.

Treatment Procedures

Behavioral Health Rehabilitation (BHR) Services are not based on a single therapeutic model addressing the therapeutic needs of the children or adolescents with severe emotional or behavioral disorders identified as requiring the BHR services.

BHR services are based upon a wraparound philosophy of an individualized treatment plan utilizing all community resources based in and delivered at the place (or places) where the problematic behaviors occur.

While BHR services are not exactly the same as wraparound services – the implementation of BHR services is consistent with the wraparound philosophy. The National Wraparound Initiative has developed a set of ten Principles of Wraparound. These ten principles are as follows:

1. Family voice and choice: An emphasis on the primary importance of goals and perspective of the individual receiving services and their family and advocates in the development of the wraparound process. This principle stresses the importance of intentional activities to illicit and include the perspective of the individual receiving services and their family and advocates.
2. Team based: This principle stresses the importance of collaborative effort of family members, professionals, and other stakeholders committed to the family's well-being over an extended period of time. The choice of team members should be largely driven by the person receiving services and their family and advocates.
3. Natural supports: To the greatest extent possible, a wraparound plan of service should utilize the natural support systems of family members, friends, neighbors, church, and community members. The plan should also include the regular support structures that exist in the community via school systems, church congregations, community centers, local government, etc.
4. Collaboration: The decision-making process in developing a wraparound plan of service should be based on a consensus approach that includes input from all team members.
5. Community based: Wraparound services should adhere to a principle of provision in the least-restrictive setting possible.
6. Culturally competent: Team designation, service planning, and service delivery should demonstrate "respect for the values, preferences, beliefs, culture and identity of the child/youth and family, and their community" (Bruns et al. 2008, p. 7).
7. Individualized: Wraparound services need to be uniquely developed for the individual in

need and their family. The planning for services should draw upon the best empirical evidence of effective treatment and upon community and professional experience. However, the services should not be assembled from a static list of available services.

8. Strengths based: A key in the development of a wraparound plan of service is to identify, “build on, and enhance the capabilities, knowledge, skills, and assets of the child and family, their community, and other team members” (p. 8).
9. Unconditional: The origins of the wrap-around process grew out of a need to provide quality services to individuals with severe and complex behaviors. It is understood at the outset that this will be a difficult and challenging process. Inherent in the development of a wraparound plan of service is a commitment to see the process through despite setbacks and unanticipated behavior, events, or outcomes. There needs to be an unwavering commitment on the part of the team to continually adapt the plan of service until progress is made and there is consensus that a wraparound process is no longer needed.
10. Outcome based: Wraparound plans of service identify measurable outcomes and indicators of progress and success. The team measures and evaluates these measures on an ongoing basis and modifies plans accordingly (Bruns et al. 2008).

Efficacy Information

As of the summer of 2011, there is little or no published research specifically addressing the efficacy of the Behavioral Health Rehabilitation approach to service delivery.

The Institute for Behavior Change reports on their website information about research conducted by Dr. Natasha K. Brown and Erica Richman of the University of North Carolina at Chapel Hill. As reported on their website, the researchers “studied 301 treatment records of children age 3 to 17 between 2002 and 2007.

They found that Behavioral Health Rehabilitation Services (BHRS) as implemented by the staff of the Institute for Behavior Change had a statistically significant association with reductions in physical aggression, noncompliance with adult prompts, socialization deficits and communication deficits. An association was also found with improvements in the environmental safety of the children” (Institute for Behavioral Change n.d.-b, p. 1).

Behavioral Health Rehabilitation (BHR) Services are not the exact equivalent of wraparound services; nevertheless, their implementation is consistent with the basic philosophy of a wrap-around approach to services. The National Wrap-around Initiative has published a summary of nine controlled studies of wraparound services that had been reported in peer-reviewed journals as of 2010. Their conclusion of this very limited universe of research is that “though many of these studies have significant methodological weaknesses, the ‘weight of evidence’ of these studies indicates superior outcomes for youth who receive wraparound compared to similar youth who receive some alternative service” (p. 5).

Qualifications of Treatment Providers

As identified by Medicaid regulations and implemented by various states, the primary treatment providers for Behavioral Rehabilitation Services fall into three categories:

- *Behavioral Specialist Consultants (BSC)*: Behavioral specialist consultants are clinicians with a Master’s or PhD level of education who work with children, family members, and other members of the treatment team to develop the individualized BHR treatment plan. These clinicians take overall responsibility for overseeing the development and implementation of the treatment plan. In addition to developing and overseeing the treatment plan, they will work as advisors and mentors to all individuals providing services under the plan, including family and community members.

- *Mobile Therapist*: A mobile therapist is a Master's or PhD educated therapist who provides child-centered, family focused, individual, and family-level psychotherapy.
- *Therapeutic Staff Support*: A therapeutic staff support (TSS) worker is an individual with a Bachelors' degree or higher level of education, who provide one-on-one services addressing treatment plan goals. TSS workers are supervised by Behavioral Specialist Consultants and/or Mobile Therapists.

See Also

- [Wraparound Services](#)

References and Reading

- Allegheny HealthChoices, Inc. (2006). Behavioral health rehabilitation services: Brief treatment model. Retrieved from <http://www.ahci.org/Reports/QualityFocusReports/BHRS%20Brief%20Treatment%20Report.pdf>
- Bruns, E. J., & Suter, J. C. (2010). Summary of the wrap-around evidence base. In E. J. Bruns & J. S. Walker (Eds.), *The resource guide to wraparound*. Portland: National Wraparound Initiative.
- Bruns, E. J., Walker, J. S., & The National Wraparound Initiative Advisory Group. (2008). Ten principles of the wraparound process. In E. J. Bruns & J. S. Walker (Eds.), *The resource guide to wraparound*. National Wraparound Initiative: Portland.
- Centers for Medicare & Medicaid Services. (n.d.). Medicaid early & periodic screening & diagnostic treatment benefit: Overview. Retrieved from http://www.cms.gov/MedicaidEarlyPeriodicScrn/01_Overview.asp
- Commonwealth of Pennsylvania Department of Public Welfare. (2009). Health choices behavioral health program: Program standards and requirements: Primary contractor. Retrieved from http://www.dpw.state.pa.us/ucmprd/groups/public/documents/communication/s_002381.pdf
- Institute for Behavior Change. (n.d.-a). A one-page overview of Medicaid, EPSDT and BHRS in Pennsylvania and elsewhere. Retrieved from <http://www.abc-pa.org/A%20one-page%20overview%20of%20Medicaid,%20EPSDT%20and%20BHRS%20in%20Pennsylvania%20and%20elsewhere%20091009.pdf>
- Institute for Behavior Change. (n.d.-b). Promising treatment found for children with inappropriate behavior. Retrieved from <http://www.abc-pa.org/Press%20Release%20and%20BHRS%20study%20071608.pdf>

Behavioral Momentum

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Definition

Behavioral momentum is derived from classical physics. In behavioral momentum, rate of responding is analogous to velocity and largely determined by the schedule of reinforcement, and the characteristic rate or magnitude of the obtained reinforcement in the situation determines the behavioral analogue of mass. The behavior momentum metaphor suggests that the more reinforcement in a condition correlates with greater resistance to change within that condition.

Behavioral momentum describes the relation between resistance to change (persistence of behavior) and the rate of reinforcement obtained in a given situation. Behavioral momentum refers to the tendency for behavior to persist after a change in environmental circumstances. The greater the rate of the reinforcement is the greater level of the behavioral momentum should be.

Behavioral momentum is frequently used as an intervention for noncompliance. Such intervention involves issuing a sequence of instructions with which the learner is most likely to comply (i.e., high-probability instructions) immediately prior to issuing a low-probability instruction.

References

- Dube, W. V., Ahearn, W. H., Lionello-DeNolf, K., & McIlvane, W. J. (2009). Behavioral momentum: Translational research in intellectual and developmental disabilities. *Behavior Analyst Today*, 9, 238–253.
- Mace, F. C., & Belfiore, P. (1990). Behavioral momentum in the treatment of escape-motivated stereotypy. *Journal of Applied Behavior Analysis*, 23, 507–514.
- Mace, F. C., Mauro, B. C., Boyajian, A. E., & Eckert, T. L. (1997). Effects of reinforce quality on behavioral momentum: Coordinated applied and basic research. *Journal of Applied Behavior Analysis*, 30, 1–20.

- Nevin, J. A. (1992). An integrative model for the study of behavioral momentum. *Journal of the Experimental Analysis of Behavior*, 57, 301–316.
- Nevin, J. A. (1996). The momentum of compliance. *Journal of Applied Behavior Analysis*, 29, 535–547.

Behavioral Objective

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Definition

A behavioral objective is a written statement that defines specific action (or pattern of actions) and set of measurements of a target behavior to be expected after an intervention. It contains the following necessary components: a description of the expected behavior itself, environmental circumstance(s) in which the behavior is to occur, and the standard criteria of acceptable behavior performance.

A behavioral objective is often expressed in the following format: *Given a set of conditions or circumstances, an individual will demonstrate the target behavior at performance level determined by rate, frequency, etc. in specified settings or with specific individuals.*

References and Reading

- Cooper, J., Heron, T., & Heward, W. (2007). *Applied behavior analysis* (2nd ed.). Columbus: Merrill/Prentice Hall.
- Yell, M., & Stecker, P. (2003). Developing legally correct and educationally meaningful IEPs using curriculum-based measurements. *Assessment for Effective Intervention*, 28, 73–88.

Behavioral Objectives

► Objective

Behavioral Skills Training

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Synonyms

“Instruction, modelling, rehearsal, and feedback,”
BST

Definition

Behavioural Skills Training (BST) is an evidence-based, multicomponent training method that applies modelling, instruction, rehearsal, and feedback to teach individuals a wide variety of behaviors or skills.

Historical Background

Although there is strong evidence that the use of BST emerged in the late 1960s and early 1970s, it is unclear where the combined use of modelling, instruction, rehearsal, and feedback specifically originated. The main difficulty in finding the origin of BST stems from variations in the use of the BST term and its components in early research. For example, O'Connor (1972) taught 33 socially withdrawn children to engage in more social interactions through the use of video modelling and social reinforcement. In another study, Braukmann (1974) used instructions, modelling, practice, and feedback to teach adolescents interviewing skills. Despite these inconsistencies, the use of BST involving “modelling, instruction, rehearsal, and feedback” was first described within the context of applied behavior analysis (ABA) by Miltenberger (1997) in his conceptualization of the teaching strategy in *Behavior Modification: Principles and Procedures*. In his book, Miltenberger outlined BST as incorporating the following strategies:

1. **Modelling:** The target skill is first demonstrated to the student, which can be done in a variety of ways (e.g., “live” with other people, by video, through cartoons, etc.). For example, when teaching a child to brush his/her teeth, a parent could model grabbing the toothbrush, putting the toothpaste on, brushing his/her teeth, rinsing his/her mouth, and cleaning the toothbrush prior to the child’s brushing attempt. The model is presented first as an example of the behavior(s) that the child is to learn, so that the child can imitate the behaviors displayed. It is important to note that learners must be able to imitate others for this component of BST to be effective.
2. **Instruction:** The child is instructed to complete the behavior modeled in the task, by providing specific instructions regarding the steps needed to complete the behavior, the circumstances in which the behavior should be emitted, and what reinforcers will be provided contingent on engagement in the target behavior. For example, a parent of a child could state, “when it’s bedtime, you must brush the top, bottom, inside, and outside of your teeth. If you do so, you will have clean healthy teeth.” The instruction should incorporate language that will be easily understood by the child, and the instruction should be used in combination with the modelling to ensure acquisition of the behavior(s).
3. **Rehearsal:** The child performs the behavior(s) that were instructed and/or modeled. For instance, the parent who first modeled and instructed the child to brush their teeth would then next observe the child complete a rehearsal of the behavior(s). The purposes of the rehearsal are (a) so the parent can confirm the acquisition of the behavior(s), (b) whether the behavior(s) are completed incorrectly, and (c) contrive an opportunity to correct mistakes and reinforce correct responding.
4. **Feedback:** Immediately after the rehearsal occurs, the student is typically provided with feedback. Feedback always includes praise, and/or other reinforcers, whether the rehearsal was correct or not. In other words, if the student did not rehearse the skill correctly, praise

for attempting the rehearsal is typically provided. If the child in the example forgot to clean his toothbrush before putting it away, the parent could say, “Good job brushing your teeth with the toothpaste! Also, don’t forget to clean the toothbrush after your done. If you don’t it could grow bacteria and make you sick.” After praise has been provided, the parent uses descriptive language to identify correct and incorrect behaviors, avoiding the use of terms such as “bad” or “wrong.” Instead, feedback emphasizes the reason why a behavior was correct/incorrect, how it can be changed, and the reasoning for the form of the behavior.

The first empirical evidence of BST as a training tool for helping people with autism spectrum disorders (ASD) and their caregivers/staff was published in 2004 in a study that used BST to teach Discrete Trial Teaching (DTT) to three teachers of a student with ASD (i.e., Sarokoff & Sturmey, 2004). Since then, BST has become an increasingly popular method for training people with ASD, ASD caregivers, and ASD staff in the field of ABA. Considering that people with ASD often experience difficulties acquiring skills and communicating with others, which may ultimately result in a greater risk of engaging in property destruction, aggression, and self-injurious behaviors without proper, evidence-based treatment (McClintock et al. 2003; Lang et al., 2019; Gormley, Healy, Doherty, O’Regan, & Grey, 2019), the application of training methods, such as BST, is paramount for creating consistent teaching opportunities for people with ASD and to sustain quality of care for caregivers and direct care staff.

Current Knowledge

As BST is an evidence-based intervention, current applications of the method have been implemented to address a variety of behavioral and skill acquisition targets. Studies have demonstrated the utility of BST to teach academic, vocational, and leisure skills to individuals with ASD.

For example, Singh et al. (2017) used BST to effectively increase performance in four different comprehension-fostering areas (i.e., prediction, questioning, summarizing, and clarifying) for a child with ASD. The BST model was implemented for each skill area using explicit instruction, modelling, rehearsal, and feedback using praise or error correction. The results of the study by Singh et al. showed that BST was effective for teaching the fostering skills and improving overall reading comprehension scores (i.e., performance increased from an average of 40% to an average of 71% during intervention and 80% during follow-up). In addition to increasing academic skills, BST has been shown to be effective at teaching vocational skills to individuals with ASD. Morgan and Wine (2018) used a multiple baseline design to evaluate the effects of BST to teach work skills in a restaurant to an 18-year-old with ASD. Instructions regarding each step of a work task were read to the individual followed by modelling of the target skills. The individual was then given the opportunity to rehearse each step of the task and was provided with oral feedback for correct and incorrect steps. Feedback was delivered immediately following each step with specific praise for correct performance of the step or corrective feedback for steps performed incorrectly. Results of the study showed that the individual was able to increase performance for all four work skills (i.e., when rolling silverware, performance increased from 0% to 100% and 100% during maintenance and generalization probes in a novel environment). BST has also been used to teach leisure skills to people with ASD. For example, Thomas et al. (2016) taught five skateboarding skills to a child with ASD through the use of instructions, modelling the target skill, rehearsal, and feedback. In addition to the instructional component, the researcher reviewed the performance of the skill with the child by showing him his performance, describing the data sheet, and giving positive feedback for performing steps correctly and corrective feedback for steps that were performed incorrectly. Following the instructions component, the researcher continued implementing the remaining components of BST (modelling, rehearsal, and feedback) for all five

skills. The participant was able to acquire four out of five skills within two to nine training sessions. Post training, generalization, and novel skateboarding skills were also assessed, demonstrated, and maintained in a novel setting.

BST has also been demonstrated to be effective for teaching caregivers and staff to implement behavior analytic strategies with learners with ASD. This type of application of BST has allowed researchers to examine the effectiveness of both BST to teach a skill as well as the acquisition of the skill by the learner. Researchers have implemented BST with a variety of ASD direct care staff, including but not limited to special education teachers, dentists, and adult behavior technicians with an ASD. For example, Kirkpatrick, Akers, and Rivera (2019) conducted a systematic review of BST literature and found numerous studies that supported BST as a method for training special education teachers to use DTT and preference assessments in the classroom. Graudins et al. (2012) used modelling, instruction, rehearsal, and feedback to train oral care providers to apply basic and effective ABA strategies such as reinforcement or visual supports during routine dental care visits. After BST was implemented, three oral care providers accurately performed the ABA-based techniques while conducting oral exams and dental cleaning with children with ASD. Lerman et al. (2013) used BST to teach four adults with autism to perform discrete trial training (DTT) to a child with ASD. Following the BST-based training, three of four adult behavior technicians with ASD demonstrated an increase in correct responding for implementing DTT with children with ASD.

Traditionally, applications of BST are comprised of the four key components as described by Miltenberger (1997) including modelling, instruction, rehearsal, and feedback. However, research has demonstrated that some variations of this model can be implemented to effectively teach skills. Instructions can be given orally and/or written; modelling can be performed by the experimenter or via video modelling; rehearsal can be performed by the participant or by role-playing with the experimenter; feedback can be provided immediately during rehearsals or after completion

of the entire response; and training can occur in a simulated environment or in situ (i.e., in the environment the behavior is ultimately intended to occur). Unlike traditional BST that evaluates mastery of a target skill in simulated environments, training in situ allows for evaluation of the skills in the environment within which the behavior is most likely occur. Subsequently, in situ training can increase the likelihood that a skill will occur in additional novel, non-training situations and settings. In situ training has been utilized and demonstrated primarily in BST research aimed at teaching safety skills to children with ASD; but it has also been demonstrated to promote learning of other skills. For instance, Gunby and Rapp (2014) taught abduction-prevention skills to three children with ASD using BST and in situ feedback. Specifically, the experimenters taught the children how to respond to different abduction lures. In situ feedback was provided immediately following incorrect responses. For all three participants, abduction prevention skills taught using BST improved and maintained during training and follow-up when novel abduction lures were presented.

Within the BST model, the evaluation of how skills are measured has varied across researchers. Some researchers have measured performance by the percentage of correct opportunities, while others have used rating scales. For example, Singh et al. (2017) and Lerman et al. (2013) evaluated performance by calculating the percentage of correct responses. Alternatively, Gunby and Rapp (2014) and Houvouras and Harvey (2014) created rating scales and designated a score value for a specific behavior or set of behaviors (i.e., Likert scale). Specifically, Houvouras and Harvey evaluated BST to teach three boys with ASD fire safety skills (i.e., how to respond in the presence of a lighter). To assess performance, the researchers created a 4-point Likert scale in which each value specified the quality in which the step or steps were performed by the participants. The results showed that the participants were able to increase their scores (i.e., from a range of 0–1 on the Likert scale to a range of 2–3; with scores of 3 during maintenance probes) following BST using the four key components of BST.

Variations of BST demonstrate how the training method can be individualized to best teach a target skill while also maintaining the components that make up the BST training method. However, because BST is a multicomponent package, one or more of its components may, at times, be solely responsible for increasing and improving target skills. Through the use of component analyses (i.e., analyses that systematically evaluate the effects of individual components that comprise intervention packages to determine the component or components responsible for positive effects), researchers and clinicians can identify the BST components necessary to bring about skill acquisition based on learner needs and the behavior of interest. At the time of this publication, research has shown inconsistent results determining which component or combination of component(s) are necessary. For example, some studies have identified feedback and modelling as necessary components (e.g., Ward & Sturmey, 2012), whereas others have indicated that instructions and modelling are necessary to the effectiveness of BST (e.g., Kornack et al. 2013; Driftke et al. 2017). Findings across numerous studies, however, have been consistent in showing that the use of only one or two components of BST seldom increases skill acquisition to mastery levels. Thus, the use of BST as a full package has been shown to be the most effective way of teaching skills to mastery levels and maintaining performance across time.

Future Directions

The use of BST with people with ASD, their caregivers, and direct care staff is well documented and substantially supported by research. BST can be used in a variety of ways to increase and shape the form of behaviors in a reliable manner. To illustrate, we provided examples of how BST can be used to teach learners with ASD to communicate, engage in social interactions, increase their reading comprehension, and even teach someone with ASD to skateboard. It is likely that BST is an effective and increasingly popular training method because of its versatility.

Specifically, it can be used to teach virtually any behavior, or chain of behaviors, as it can be adapted and used with other resources (e.g., videos, manuals) to provide individualized training for learners. Despite the robust support in the literature for BST methods, continued examination of BST and its components with various populations, target skills, and with more variation has been suggested by researchers.

A consistent topic brought up by current BST researchers is the need for closing the “research-to-practice gap” (i.e., the inconsistent application of relevant and current research in locations/environments where they are most needed). For example, Gormley et al. (2019) noted that although most training of other professionals involved some level of individualization, BST has not been employed despite the evidence supporting BST as a reliable method that can be individualized to train specific behaviors or set of behaviors. Kirkpatrick, Akers, and Rivera (2019), in a systematic review of the literature, noted that BST tends to be used with special education teachers, but not with general education teachers. Further, a review by van der Meer et al. (2017) that examined 22 studies in which training was provided to direct care staff on the implementation of communication interventions found that although all the reviewed interventions indicated positive outcomes for the staff and the individuals with intellectual disabilities, only one study utilized all BST components in its teaching package. Rather, the review indicated that direct care staff were trained with various methods such as presentations, didactic instructions, group discussions, role-play, and structured practice with feedback. In addition to the research-to-practice gap, there is a notable gap between the use of BST between clinicians and non-clinic service providers. For example, Gaudins et al. (2012) indicated that BST was useful in teaching oral care providers how to use ABA-based techniques. Although the results of Gaudins et al. demonstrated effectiveness, there is a paucity of research related to the use of BST in this capacity. The same type of approach used by Gaudins et al. could be done with other community-based individuals such as firemen, policemen, hairdressers, and other

professionals to facilitate interactions between individuals with ASD and professionals in service industries. Given the consistent identification by literature reviews regarding the research-to-practice gap, future studies should try to expand the knowledge base regarding BST with individuals outside the field of ABA.

In addition to closing the research-to-practice gap, there is a need to identify the necessary training components within BST and the effectiveness of BST relative to other teaching methodologies. Some studies have indicated that modelling and feedback are the most important components of BST (e.g., Ward & Sturmey, 2012), while others have indicated that instructions and modelling are more important (e.g., Driftke et al. 2017); other studies have suggested that necessary components are idiosyncratic to each learner (e.g., Kornack et al. 2013). It is important to note that previous BST component analysis-based studies have targeted different skills and behaviors. Thus, caution is needed in terms of inferring external validity with regard to these studies. In other words, behaviors or skills that were taught in respective component analysis-based studies varied across studies, possibly contributing to the varying results. Accordingly, it is important that researchers continue to replicate and extend component analysis-based research regarding BST to continue to evaluate and document the circumstances in which some or all components are necessary to produce positive outcomes. Similarly, more comparative analysis-based research is needed to evaluate the effectiveness of BST relative to other training strategies. Leaf et al. (2015) conducted a review of studies that compared BST to another prominent ABA-based teaching strategy, the Teaching Interaction Procedure (TIP). The authors concluded that even though both interventions were effective at teaching a behavior or skill, none of the studies reviewed provided quantitative measurements that compared one to the other. Future studies could focus on comparing the effectiveness of BST and TIP by measuring trials to criterion, overall training time, and/or other dimensions to determine the most efficient strategy. Leaf et al. also noted that multiple studies have used the

components of BST and mislabelled them as *TIP* and vice versa. It is likely that these misidentifications are due to lack of training with multiple teaching methodologies. Considering the acknowledged presence of a research-to-practice gap, future efforts of researchers should focus on educating professionals on various types of teaching strategies and their respective components.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Imitation](#)
- [Modeling](#)

References and Reading

- Braukmann, C. J., Fixsen, D. L., Phillips, E. L., Wolf, M. M., & Maloney, D. M. (1974). An analysis of a selection interview training package for predeunquents at achievement place. *Criminal Justice and Behavior*, 1, 30–42. <https://doi.org/10.1177/009385487400100105>.
- Driftke, M. A., Tiger, J. H., & Wierzbica, B. C. (2017). Using behavioral skills training to teach parents to implement three-step prompting: A component analysis and generalization assessment. *Learning and Motivation*, 57, 1–14. <https://doi.org/10.1016/j.lmot.2016.12.001>.
- Gormley, L., Healy, O., Doherty, A., O'Regan, D., & Grey, I. (2019). Staff training in intellectual and developmental disability settings: A scoping review. *Journal of Developmental and Physical Disabilities*, 1–26. <https://doi.org/10.1007/s10882-019-09683-3>.
- Graudins, M. M., Rehfeldt, R. A., DeMattei, R., Baker, J. C., & Scaglia, F. (2012). Exploring the efficacy of behavioral skills training to teach basic behavior analytic techniques to oral care providers. *Research in Autism Spectrum Disorders*, 6, 978–987. <https://doi.org/10.1016/j.rasd.2011.12.010>.
- Gunby, K. V., & Rapp, J. T. (2014). The use of behavioral skills training and in situ feedback to protect children with autism from abduction lures. *Journal of Applied Behavior Analysis*, 47, 856–860. <https://doi.org/10.1002/jaba.173>.
- Houvouras, A. J., & Harvey, M. T. (2014). Establishing fire safety skills using behavioral skills training. *Journal of Applied Behavior Analysis*, 47, 420–424. <https://doi.org/10.1002/jaba.113>.
- Kirkpatrick, M., Akers, J., & Rivera, G. (2019). Use of behavioral skills training with teachers: A systematic review. *Journal of Behavioral Education*, 1–18. <https://doi.org/10.1007/s10864-019-09322-z>.
- Kornack, L. T., Ringdahl, J. E., Sjostrom, A., & Neurnberger, J. E. (2013). A component analysis of a behavioral skills training package used to teach conversation skills to young adults with autism spectrum and other developmental disorders. *Research in Autism Spectrum Disorders*, 7, 1370–1376. <https://doi.org/10.1016/j.rasd.2013.07.012>.
- Lang, R., Davis, T., Ledbetter-Cho, K., McLay, L., Erhard, P., & Wicker, M. (2019). Psychological and educational approaches to the treatment of aggression and tantrums in people with intellectual disability. In J. L. Matson (Ed.), *Handbook of intellectual disabilities: Integrating theory, research and practice*. New York: Springer.
- Leaf, J. B., Townley-Cochran, D., Taubman, M., Cihon, J. H., Oppenheim-Leaf, M. L., Kassardjian, A., et al. (2015). The teaching interaction procedure and behavioral skills training for individuals diagnosed with autism spectrum disorder: A review and commentary. *Review Journal of Autism and Developmental Disorders*, 2, 402–413. <https://doi.org/10.1007/s40489-015-0060-y>.
- Lerman, D. C., Hawkins, L., Hoffman, R., & Caccavale, M. (2013). Training adults with an autism spectrum disorder to conduct discrete-trial training for young children with autism: A pilot study. *Journal of Applied Behavior Analysis*, 46, 465–478. <https://doi.org/10.1002/jaba.50>.
- McClintock, K., Hall, S., & Oliver, C. (2003). Risk markers associated with challenging behaviours in people with intellectual disabilities: A meta-analytic study. *Journal of Intellectual Disability Research*, 47, 405–416. <https://doi.org/10.1046/j.1365-2788.2003.00517.x>.
- Miltenberger, R. G. (1997). *Behavior modification: Principles and procedures*. Belmont: Thomson Brooks/Cole Publishing Company.
- Morgan, C. A., & Wine, B. (2018). Evaluation of behavior skills training for teaching work skills to a student with autism spectrum disorder. *Education and Treatment of Children*, 41, 223–232. <https://doi.org/10.1353/etc.2018.0009>.
- O'Connor, R. D. (1972). Relative efficacy of modeling, shaping, and the combined procedures for medication of social withdrawal. *Journal of Abnormal Psychology*, 79, 327–334.
- Sarokoff, R. A., & Sturmey, P. (2004). The effects of behavioral skills training on staff implementation of discrete-trial teaching. *Journal of Applied Behavior Analysis*, 37, 535–538. <https://doi.org/10.1901/jaba.2004.37-535>.
- Singh, B., Moore, D., Furlonger, B., Anderson, A., Busacca, M., & English, D. (2017). Teaching reading comprehension skills to a child with autism using behaviour skills training. *Journal of Autism and Developmental Disorders*, 47, 3049–3958. <https://doi.org/10.1007/s10803-017-3229-7>.
- Thomas, B., Lafasakis, M., & Spector, V. (2016). Brief report: Using behavioral skills training to teach skateboarding skills to a child with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46, 3824–3829. <https://doi.org/10.1007/s10803-016-2900-8>.

van der Meer, L., Matthews, T., Ogilvie, E., Berry, A., Waddington, H., Balandin, S., & Sigafoos, J. (2017). Training direct-care staff to provide communication intervention to adults with intellectual disability: A systematic review. *American Journal of Speech-Language Pathology*, 26, 1279–1295. https://doi.org/10.1044/2017_AJSLP-16-0125.

Ward, H. J., & Sturmey, P. (2012). Component analysis of behavior skills training in functional analysis. *Behavioral Interventions*, 272, 75–92. <https://doi.org/10.1002/bin.1339>.

Behavioral Specialist

► Behavior Analysis

Behaviorally Based Social Skill Groups

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Definition

Today one of the most commonly implemented and endorsed interventions are procedures based upon the principles of applied behavior analysis (ABA; Smith 2012). Although, behaviorally based interventions are commonly implemented in a one-to-one instructional format, research has shown that these interventions can be efficacious within a group instructional format (e.g., Dotson et al. 2010). One form of behaviorally based intervention is the implementation of behaviorally based social skills groups. Reichow and Volkmar (2010) categorize social skills groups as “groups contained two or more like-aged individuals (with and/or without disabilities) meeting in a group instructional format...” (p. 152). For an intervention to be considered a behaviorally based social skills group, the interventionists must implement only behavior

analytic procedures (e.g., behavioral skills training, discrete trial teaching, incidental teaching, or the teaching interaction procedure) and must only implement procedures that are evidence based, empirically based, and not pseudoscientific or antiscientific.

Historical Background

There has been a long history of the implementation of behaviorally based social skills groups for individuals diagnosed with autism spectrum disorder (ASD). Some of the early work took place at the UCLA Young Autism Project, where techniques such as discrete trial teaching occurred in one-to-one instructional formats and group instructional formats (Lovaas 1987; Smith and Lovaas 1998). Since the early days of behaviorally based social skills groups implemented in a clinic-based setting, there have been numerous single-subject and group design studies that have shown the effectiveness of social skills groups (see Williams White et al. (2007) and Reichow and Volkmar (2010) for a more complete review). One of the major milestones in the terms of the empirical research was researchers evaluating social skills groups with randomized group designs, most notably with the evaluation of the UCLA PEERS program (Laugeson et al. 2009). The authors and developers of this model demonstrated that a manualized approach to a social skills group, with parental support, can be effective in improving the lives of adolescents diagnosed with ASD. More recently, Leaf et al. (2017) used a randomized control trial to demonstrate the effectiveness of a behaviorally based social skills group for young children diagnosed with ASD. The results indicated that behaviorally based social skills groups can yield significant improvements to the social behavior of young individuals diagnosed with ASD. The results also demonstrated that a model in which interventionists are permitted to make in-the-moment decisions without a strict protocol (i.e., a progressive model of ABA) can be effective within a behaviorally based social skills group.

Rationale or Underlying Theory

Behaviorally based social skills groups are deeply rooted in the philosophy of behavior analysis. Within this philosophy, behavior is the result of the individual's interaction with his or her environment. More specifically, the probability of a response, or response class, occurring is increased when it is followed by a reinforcing event, and decreased when followed by a punishing event. In addition to the manipulation of reinforcing or punishing events, interventionists manipulate the potency of these events (e.g., establishing and abolishing operations), find effective ways to increase the likelihood of the student(s) responding correctly (e.g., prompting and prompt fading), and transfer learned behaviors with the interventionist, or within the context of the social skills group, to the individual's natural environment. Finally, interventionists make decisions based upon objective data as opposed to anecdotal or subjective evidence.

Goals and Objectives

The overall purpose of behaviorally based social skills groups is to improve specific social behaviors (e.g., joint attention, greetings, and sharing) as well as more global social behaviors (e.g., pragmatics, social learning, and social relatedness) for individuals diagnosed with ASD, with the ultimate goal of improving pro-social relationships and developing meaningful friendships for the individuals receiving intervention. Interventionists should target the improvement of a variety of social behaviors when implementing social skills groups. Some of the skills that can be taught are social communication, social interaction, social awareness, social learning, and social relatedness (Taubman et al. 2011). When interventionists teach social communication skills (e.g., changing the game when bored or providing compliments), the goal is to teach the student how to use and understand the social aspects of communication. When interventionists teach social interaction skills (e.g.,

joining into a game or sportsmanship), the goal is to teach the student skills that will help promote successful interactions with the student's peers. When interventionists teach social awareness skills (e.g., relationship identification or theory of mind), the goal is to teach the student how to pick up on social cues and discriminate between different social cues. When interventionists teach social learning skills (e.g., observational learning or vicarious learning), the goal is to teach the student how to learn from their environment without direct instruction. When interventionists teach social relatedness skills (e.g., tolerating peers or accepting peers), the goal is to teach the student how to have meaningful connections with his or her peers. All of these skills can be taught within the context of a behaviorally based social skills group and can improve the quality of life for individuals diagnosed with ASD. Additionally, within the context of behaviorally based social skills groups, the interventionists might also focus on teaching language, learning how to learn skills, self-help skills, and decreasing any aberrant or interfering behavior(s).

Treatment Participants

Although there is an underlying assumption that behaviorally based social skills groups are only effective for higher functioning and older individuals diagnosed with ASD, they can be successfully implemented with a wide age range of individuals across several demographics. Although behaviorally based social skills groups can be implemented with a heterogeneous group of participants, our research and clinical experience has shown that behaviorally based social skills groups are more effective for the participants and easier for interventionists if the groups are homogenous (Dotson et al. 2010; Leaf et al. 2017). Thus, interventionists should attempt to include participants in the group around the same chronological age (i.e., not developmental age), that display the same deficits in social behavior, have the same level of language, and display the same level of aberrant behavior. Ideally, students

should display minimal amounts of aberrant behavior prior to entering a social skills group, especially, if this behavior is harmful to the individual (i.e., self-injurious) or may be harmful to peers (i.e., aggression). Additionally, if a participant displays high rates of stereotypic behavior, that participant might not be the best fit for a social skills group as stereotypic behavior may interfere with the learning process (Cunningham and Schreibman 2008). Finally, if the interventionists have not identified and/or conditioned effective reinforcers, they should not start the participant in a behaviorally based social skills group, as the interventionists will not be able to effectively reinforce appropriate social behavior.

Treatment Procedures

What distinguishes behaviorally based social skills groups compared to other social skills groups implemented for individuals diagnosed with ASD is that the interventionists only implement procedures based upon the principles of ABA, using only evidenced-based and empirically supported procedures, with the goal to improve observable and measurable behavior. As such, interventionists do not utilize mentalistic interpretations to target social behavior, refrain from utilizing an eclectic approach, and refrain from implementing pseudoscientific procedures. Within a behaviorally based social skills group, the goal of the interventionists should be to minimize downtime of the learner and to maximize teaching opportunities. This can be accomplished by either following strict protocols (Laugeson et al. 2009) or using in-the-moment decision-making informed by clinical judgment (Leaf et al. 2017). Overall, behaviorally based social skills groups should be implemented at least once a week, but it is highly recommended to implement more frequently. It is also recommended that each behaviorally based social skills group run for at least 2 h in duration. A final overall component is that parent involvement should occur at some level, ranging from parents observing sessions (Leaf et al. 2017) to being

directly involved in the intervention (Laugeson et al. 2009).

Within the context of a behaviorally based social skills group, there are several different procedures that can be implemented, all of which have empirical support and have been implemented in one-to-one instructional formats. One procedure that can be implemented in a behaviorally based social skills group is group discrete trial teaching (e.g., Taubman et al. 2001). Within this procedure, the interventionist provides an instruction to a learner(s), allows time to respond, and provides either reinforcement for correct responding or corrective feedback for incorrect responding. The interventionists could provide discrete trials sequentially where one student responds at a time or chorally where all students respond together.

Another procedure that can be implemented in the context of a behaviorally based social skills group is video modeling (Wang and Koyama 2014). Within video modeling, the interventionists would show a video of either an adult, a peer, or the participants themselves engaging in a targeted social behavior. After the video has been watched by the learners, the learners have the opportunity to practice the targeted social behavior(s). Another approach that can be used within behaviorally based social skills groups is incidental teaching (Hart and Risley 1975). In incidental teaching, the interventionists arrange opportunities for the learner to engage in the behavior, follow the learners lead, and when they engage in the target social behavior, the interventionist can provide prompting, error correction, and/or reinforcement. Interventionists can also embed instructions into the context of games. When interventionists do this, they create opportunities in which the learners can respond to multiple simultaneous implicit instructions that occur within the context of the game/activity.

Interventionists can also implement a social discrimination program called the Cool versus Not Cool (CNC; e.g., Leaf et al. 2016) procedure. The CNC procedure consists of the teacher modeling a targeted social behavior correctly (i.e., “Cool”) and incorrectly (i.e., “Not Cool”).

After the demonstration, the learners have an opportunity to state, or label, if the model was “cool” or “not cool” and why the demonstration was “cool” or “not cool.” After the learner labels the social behavior, the learners have an opportunity to role-play the targeted behavior the cool way. A similar procedure that can be implemented in the context of behaviorally based social skills group is the teaching interaction procedure (TIP; Leaf et al. 2015). The TIP is a multicomponent teaching procedure which consists of the interventionist describing the targeted social behavior, providing cues and characteristics of when to engage in the desired social behavior, providing a meaningful rationale of why the learner should engage in the targeted social behavior, breaking the social behavior into smaller components (i.e., task analysis), modeling the targeted social behavior the cool and not cool way, providing the opportunity for the learners to role-play the behavior, and providing differential consequences throughout. Along with these procedures, interventionists could also implement script fading (e.g., Pollard et al. 2012), behavioral skills training (e.g., Miltenberger et al. 2009), and peer-mediated interventions (e.g., Odom et al. 1985) in the context of behaviorally based social skills groups.

Efficacy Information

Through the use of single-subject designs and group designs, researchers have shown that the implementation of behaviorally based social skill groups can be an effective way to improve social behavior for individuals diagnosed with ASD (e.g., Dotson et al. 2010; Leaf et al. 2017). Researchers have evaluated specific interventions (described previously) and have shown that these interventions can lead to learners with ASD learning and engaging in social behaviors such as joint attention, game play, and improved social language (see Williams White et al. (2007) and Reichow and Volkmar (2010) for a more complete review). Researchers have also evaluated the overall effects of behaviorally based social skills groups on improving the overall

social behavior of individuals diagnosed with ASD (e.g., Laugeson et al. 2009; Leaf et al. 2017). In these studies, the results have shown that individuals who receive intervention within behaviorally based social skill groups demonstrate an overall improvement in social behavior as compared to individuals who did not receive intervention within a behaviorally based social skill groups. Furthermore, the results of these group design studies have indicated that the individuals who did receive behaviorally based social skill groups maintained the improvements in social behavior overtime and that these social behaviors generalized to other settings (Leaf et al. 2017).

Outcome Measurement

When measuring improvements in social behavior, it is important to evaluate observable behaviors as opposed to what the learners say they should be doing, or evaluating mentalistic interpretations of behavior. Researchers should use a combination of measures to evaluate improvements in specific social behaviors (e.g., joint attention, sharing, social communication) and overall social improvement (e.g., overall quality of play, overall social interaction, and friendship development). When evaluating improvements on specific social behaviors, the researchers should conduct naturalistic and generalization probes. When implementing naturalistic and generalization probes, the researchers set up an opportunity for the learner(s) to display the targeted social behavior without providing any additional consequences (i.e., consequences other than those occurring naturally) or prompting. For example, if the targeted social behavior is losing graciously, the researcher would set up a game (e.g., the card game war) between a peer/adult and the learner(s) and see how the learner(s) respond when he or she loses the game. Naturalistic probes would be conducted within the context of the social skills group, while generalization probes would be conducted in the learner(s) natural environment (e.g., home, school, community).

Both naturalistic and generalization probes should be evaluated in conjunction with scoring on standardized assessments. There are numerous assessments which could be used when evaluating the overall social behavior of individuals diagnosed with ASD these include: the Vineland Adaptive Behavior Scales, the Social Skills Improvement System, Social Responsiveness Scale, The Walker-McConnell Scale of Social Competence and School Adjustment, the Friendship Qualities Scale, and the Aberrant Behavior Checklist. Within the research, there have been multiple evaluators who have scored these various assessments. The multiple evaluators have included parents, social skills group teachers, outside teachers, and blind evaluators (e.g., Leaf et al. 2017). Within clinical practice and research studies, it is encouraged that multiple people evaluate the participants across these various assessments. It is also encouraged that one of evaluators be blind to the study or clinical implementation of the behaviorally based social skill groups in order to minimize the chance for potential biases.

Qualifications of Treatment Providers

There are no specific credentials or certifications which are required for a professional to implement behaviorally based social skill groups for individuals diagnosed with ASD. What is required is that the professional is well trained in the principles of behavior analysis and is not just trained on how to simply follow a set of procedures or rules. It is not enough for a professional to know how to follow a protocol on the implementation of video modeling, but rather, the professional needs a thorough understanding of the principles behind video modeling. Professionals also need to be fluent in the implementation of a variety of procedures (e.g., video modeling, CNC, TIP) within the context of social skills groups. Additionally, the professional should be well trained in social curriculum and curriculum development to ensure the teaching of meaningful and functional social curriculum. Furthermore, professionals should be trained in

how to work collaboratively and supportively with parents. This training must be intensive (i.e., more than 40 h of general training in behavior analytic principles) and an individual should not be considered competent based on any time requirement (e.g., 40, 1000, or 1500 h) or solely on responses to questions or scenarios (e.g., performance on a multiple choice test). Rather, competency should be determined based on a performance-based assessment, in addition to any written assessments. Although, it is more important that a professional is qualified rather than certified, it may be a requirement by some third-party payers that a professional be certified or licensed. If this is the case, it is important that the professional understand that a certification or license does not necessarily mean that the professional is qualified to implement a behaviorally based social skill group.

See Also

- ▶ [Friendships](#)
- ▶ [Prosocial Behavior](#)
- ▶ [Social Skill Interventions](#)

References and Reading

- Cunningham, A. B., & Schreibman, L. (2008). Stereotypy in autism: The importance of function. *Research in Autism Spectrum Disorders*, 2(3), 469–479.
- Dotson, W. H., Leaf, J. B., Sheldon, J. B., & Sherman, J. A. (2010). Group teaching of conversational skills to adolescents on the autism spectrum. *Research in Autism Spectrum Disorders*, 4(2), 199–209.
- Hart, B., & Risley, T. R. (1975). Incidental teaching of language in the preschool. *Journal of Applied Behavior Analysis*, 8(4), 411–420.
- Laugeson, E. A., Frankel, F., Mogil, C., & Dillon, A. R. (2009). Parent-assisted social skills training to improve friendships in teens with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(4), 596–606.
- Leaf, J. B., Townley-Cochran, D., Taubman, M., Cihon, J. H., Oppenheim-Leaf, M. L., Kassardjian, A., et al. (2015). The teaching interaction procedure and behavioral skills training for individuals diagnosed with autism spectrum disorder: A review and commentary. *Review Journal of Autism and Developmental Disorders*, 2(4), 402–413.

- Leaf, J. B., Taubman, M., Milne, C., Dale, S., & Leaf, J. (2016). Teaching social communication skills using cool versus not cool procedure plus role-playing and a social skills taxonomy. *Education and Treatment of Children*, 39(1), 44–63.
- Leaf, J. B., Leaf, J. A., Milne, C., Taubman, M., Oppenheim-Leaf, M., Torres, N., et al. (2017). An evaluation of a behaviorally based social skills group for individuals diagnosed with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(2), 243–259.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55(1), 3–9.
- Miltenberger, R., Gross, A., Knudson, P., Bosch, A., Jostad, C., & Breitwieser, C. B. (2009). Evaluating behavioral skills training with and without simulated in situ training for teaching safety skills to children. *Education and Treatment of Children*, 32(1), 63–75.
- Odom, S. L., Hoyson, M., Jamieson, B., & Strain, P. S. (1985). Increasing handicapped preschoolers' peer social interactions: Cross-setting and component analysis. *Journal of Applied Behavior Analysis*, 18(1), 3–16.
- Pollard, J. S., Betz, A. M., & Higbee, T. S. (2012). Script fading to promote unscripted bids for joint attention in children with autism. *Journal of Applied Behavior Analysis*, 45(2), 387–393.
- Reichow, B., & Volkmar, F. R. (2010). Social skills interventions for individuals with autism: Evaluation for evidence-based practices within a best evidence synthesis framework. *Journal of Autism and Developmental Disorders*, 40(2), 149–166.
- Smith, T. (2012). Evolution of research on interventions for individuals with autism spectrum disorder: Implications for behavior analysts. *The Behavior Analyst Today*, 35(1), 101–113.
- Smith, T., & Lovaas, I. O. (1998). Intensive and early behavioral intervention with autism: The UCLA young autism project. *Infants & Young Children*, 10(3), 67.
- Taubman, M., Brierley, S., Wishner, J., Baker, D., McEachin, J., & Leaf, R. B. (2001). The effectiveness of a group discrete trial instructional approach for preschoolers with developmental disabilities. *Research in Developmental Disabilities*, 22(3), 205–219.
- Taubman, M., Leaf, R., & McEachin, J. (2011). *Crafting connections: Contemporary applied behavior analysis for enriching the social lives of persons with autism spectrum disorder*. New York: DRL Books.
- Wang, H.-T., & Koyama, T. (2014). An analysis and review of the literature and a three-tier video modeling intervention model. *Research in Autism Spectrum Disorders*, 8(7), 746–758.
- Williams White, S., Keonig, K., & Scahill, L. (2007). Social skills development in children with autism spectrum disorders: A review of the intervention research. *Journal of Autism and Developmental Disorders*, 37(10), 1858–1868.

Behaviorism

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Definition

Behaviorism is a philosophy of science where behavior is the unit of study and several suppositions about the science of behavior are made. Behaviorism focuses on the study of behavioral phenomenon that function under the same principles of conditioning. This includes behaviors that are both public and private. Finally, use of mentalistic terminology (e.g., I feel, I think, I believe) is not helpful in examining behavior and, in fact, ends the examination of a phenomenon.

There is some confusion when discussing behaviorism, particularly the radical behaviorist position of B. F. Skinner by critics wherein people assume that covert behaviors or behavior that occurs “under the skin” such as thoughts and feelings are not important to the study of behavior. The main impediment to using private events, those internal to the individual, is the difficulty in corroborating these events by another individual. Given the need for objective measurement of behavioral phenomenon, the inability to observe internal events makes inclusion of private events a challenge in discussing and defining behavioral principles.

The challenge for behaviorism is presenting behavior as the primary unit of analysis for psychology where the general public tends to support the idea that the “mind” or mental events are the focus and cause of a person’s behavior. Mentalistic concepts such as frustration, anxiety, depression, or anger are not helpful in our understanding of behavior and were deemed “explanatory fictions” by Skinner. Such concepts do not add to our understanding of behavioral phenomena; rather, they end the analysis. Behaviorists look to the behavioral manifestations of what is termed frustration, anxiety, depression, and anger and attempt

to explore the environmental stimuli that function to maintain and reinforce said manifestations.

Behaviorism maintains that behaviors that are overt (observable) and private (“within the skin”) can both be subjected to objective observations with the latter suffering from the challenges noted above with regard to corroboration of a second observer. Therefore, some behaviorists view thinking and feeling as behavior in the same way as overt behaviors such as running, typing, and speaking. While there is some discussion about the utility of attempting to analyze these covert behaviors (see discussion below), there is no argument that individuals engage in covert behavior.

Three type of behaviorism are generally discussed:

Methodological Behaviorism – The study of behavior should focus only on those behaviors that are observable and that no mental states should be considered in the analysis. This is most closely associated with John Watson.

Psychological Behaviorism – Associated with B. F. Skinner, psychological behaviorists focus on the functional relationship between environmental events (antecedents and consequences) and the behaviors produced by those environmental events.

Analytical Behaviorism – A behaviorist position that posits that mental states can be explained through consistent patterns of behavior. These patterns can lead to predictions of an individual’s behavior given a specific set of environmental stimuli.

Historical Background

Behaviorism has links to several philosophical schools including:

Associationism – Classical associationism dealt with the organization of ideas based on relationships between mental states and can be seen in writings as far back as Aristotle. David Hume presented a model of associationism that suggested that our understanding of reality was a product of three laws of

association. These included the Law of Resemblance, things that are similar are associated; the Law of Contiguity, things that occur close in time will be associated; and the Law of Cause and Effect, the most important aspect of associationism wherein the individual identifies causal influences on the environment. This is the basis of scientific inquiry.

Logical Positivism – A philosophical perspective that posits the only true knowledge is knowledge derived from scientific endeavors. Metaphysical explanations are to be abandoned as they cannot be demonstrated empirically.

Behaviorists

John Watson is considered the earliest psychologist to identify himself as a behaviorist. In his work *Psychology as the Behaviorist Views It*, he described the power of behavioral approaches and suggested that psychology should be the science of behavior and not the mind. Watson’s work was with reflexive behavior (see below) and therefore was responding to a limited amount of information on behavior and its relationship with the environment. His work led to significant criticism and a backlash from traditional psychologists who viewed his claims as boastful and whose methods generally consisted of introspection or turning inward for causes of behavior rather than to environmental influences.

Pavlov – Ivan Pavlov’s classic experiments on classical conditioning, (see below), demonstrated a conditioning paradigm that involved reflective behavior similar to Watson. In his classic experiments, Pavlov paired a neutral stimulus (NS), or a stimulus with which the organism does not have any learning history with, with an unconditioned stimulus (UCS), a stimulus that elicits an unconditioned response, a reflex response that occurs in the presence of the UCS. In Pavlov’s experiments, the neutral stimulus was a tone and the unconditioned stimulus was the presentation of food. In response to the presentation of the food, the organism, a dog, salivated. Through repeated pairings of a tone (NS) and the food (UCS), the tone began to elicit the response of salivation without the presence of the food. The tone had become a conditioned stimulus (CS) that elicited

the conditioned response (salivation). The diagram below outlines this process.

E. L. Thorndike – Thorndike’s experimental work led to his theory of Connectionism and the Law of Effect. He examined learning processes in experiments with animals. Animals, generally cats, were placed in a puzzle box that required the animal to perform an action to escape the box and receive a reward. Thorndike observed that the time animals took to perform an action (e.g., lever press) decreased after successful attempts to escape. Additionally, animals did not demonstrate the required action after observing other animals engaging in the behavior. This led to Thorndike’s formulation of a cause/effect description of learning. He tracked “learning curves” in the behavior of animals to demonstrate that learning was a gradual process of trial and error. Thorndike’s Law of Effect indicates that behavior that is followed by positive consequences is likely to be repeated in the future.

Hull – Clark Hull presented a theory of learning termed drive-reduction theory. Drive-reduction theory suggests that behaviors occur in response to internal drives of the organism. Drives are generally important for survival including hunger, thirst, and warmth. Stress on the organism leads to behaviors that reduce the drive and reduce stress. Drive reduction reinforces the organism and those behaviors will occur more frequently in the future. Hull’s theory presents a stimulus-response form of behaviorism where the stimulus (drive) elicits the behavior.

Skinner – Burrhus Frederick (B. F.) Skinner demonstrated operant conditioning procedures in laboratory settings. His work described the principles of behavior that serve as the foundation for the science of the experimental analysis of behavior and applied behavior analysis. Skinner’s radical behaviorism was borne out of his observations during experiments, not based on a theory of why organisms behave in a certain way. Skinner presents a response-stimulus understanding of behavior where the consequences that follow a behavior are crucial to the conditioning of behavior. Operant conditioning is so named as behaviors are emitted and operate on the environment. This is contrasted with behaviors that are elicited

by environmental events and are reflexive in nature. Skinner extended his work in the laboratory to extrapolations to the development of language, social engineering, and education in his later work. All of these extensions of his work were based in operant conditioning methodology.

Current Knowledge

Approaches

Methodological behaviorism is associated with John Watson following the publication of *Psychology as the Behaviorist Views It*. Within this paradigm, observable behavior is the only thing that should be studied and all things within the body should not be considered the realm of psychology.

Radical behaviorism was proposed by B. F. Skinner. The term radical behaviorism referred to the acknowledgement that a science of human behavior must account for covert behaviors (or behaviors within the skin) to be complete. The challenge for establishing the role of internal events into a functional analysis of behavior is that these are not accessible to anyone other than the individual being studied. This, therefore, does not allow for corroboration of these internal events as they are not observable.

Types of Conditioning

Respondent conditioning or classical conditioning is the process of conditioning reflexes to respond to environmental stimuli. This type of conditioning is also known as stimulus-response conditioning where the stimulus (S) precedes the response (R). This relationship is often represented as $S \rightarrow R$. In a traditional classical conditioning arrangement, a neutral stimulus (e.g., a flashing light) that has no previous history of being paired with the occurrence of the reflex (e.g., an eye blink) is presented along with a stimulus that elicits the reflex (e.g., a puff of air). The stimulus that elicits the reflex response is known as the unconditioned stimulus as it does not require a learning history to elicit the reflex or unconditioned response. After repeated pairings of the neutral stimulus with the unconditioned

stimulus, presentation of the neutral stimulus will come to elicit the unconditioned response without presenting the unconditioned stimulus. For this example, presenting the flashing light prior to the puff of air over multiple trials will eventually lead to the flashing light eliciting eye blinking without presenting the puff of air. This arrangement is represented as:

Neutral stimulus → *unconditioned stimulus*
→ *unconditioned response*.

With continued pairing of the neutral stimulus and the unconditioned stimulus, the neutral stimulus, now a conditioned stimulus, comes to control the occurrence of the unconditioned response, now called a conditioned response. This arrangement is represented as:

Conditioned stimulus → *conditioned response*.

When the conditioned stimulus is presented, the response follows as if the unconditioned stimulus had been presented. In this instance, behavior is elicited, that is, behavior is caused by the occurrence of an external stimulus. Continued presentation of the conditioned stimulus without the presentation of the unconditioned stimulus will gradually lead to reductions in the conditioned response. This process is termed extinction.

Operant conditioning occurs when a behavior comes under the control of consequences that follow it. The operant conditioning paradigm can be described in the three-term contingency:

Antecedent → *Behavior* → *Consequence*.

An antecedent is a stimulus event that precedes the occurrence of behavior where as a consequence is a stimulus event that follows the occurrence of the behavior. A behavior is anything an organism does and results in a change in the environment. During operant conditioning, an organism's behavior is subject to consequences that lead to increases (reinforcement) or decreases (punishment) in the future occurrence of that behavior. Along with these increases, antecedent stimulus events come to serve as discriminative

stimuli for the likelihood of reinforcement. That is, environmental events signal the availability of reinforcement if the organism engages in a particular repertoire of behavior. Skinner's work on shaping is instrumental to the development of learned repertoires of behavior. Shaping involves reinforcement of closer and closer approximations to the target behavior. For example, a rat in an operant chamber may be required to push a lever to access food (a reinforcer). As the rat moves about the cage and orients to the lever, a click is followed by the delivery of the reinforcer. As the rat begins to orient toward the lever more frequently, reinforcement is delivered and then withheld. This withholding is called extinction. Extinction leads to variability in responding where the rat may now touch the lever which would be followed by reinforcer delivery. This process continues until the rat reliably presses the lever. Shaping, extinction, and schedules of reinforcement serve as the basis for our understanding of the development of behavioral repertoires.

Molecular Versus Molar Behaviorism

The contrast of molar and molecular behaviorism represents the focus of attention in a functional analysis. Those who support a molecular view of behaviorism support looking at the moment to moment changes in behavior and analyze the direct antecedents to and consequences of those behaviors. This is a view that is in line with Skinner's analyses of behavior in his basic experimental work. A molar perspective looks at behavior over time and views behavior in the context of other, longer sequences (chains) of behavior. That is, when describing an event, one needs to observe the behavior to completion as opposed to a moment in time. Lever pressing is best understood as the duration of engaging in lever pressing and not in the instant where the lever is pressed. The molar view contrasts with the molecular view in terms of how responses are strengthened. The molecular view focuses on increases in response rates as an indicator of response strength. In contrast, the molar view focuses on increased allocation of responding to one or another behavior in a choice paradigm. That is, all behavior requires

choices between responses and the selection of one behavior over another is a function of reinforcement. There is ongoing discussion among behavior analysts as to which perspective best explains behavioral phenomena.

Applications

Experimental Analysis of Behavior – The experimental analysis of behavior has a primary focus on basic research, that is, research on human and nonhuman organisms whose purpose is to develop greater understanding of behavioral principles. This, in turn, enhances our understanding of conditions that reliably predict their occurrence. The experimental analysis of behavior is responsible for our understanding of reinforcement, schedules of reinforcement, and their impact on behavior, punishment, discriminative stimuli, and choice. Basic behavioral principles demonstrated in laboratory settings serve as the basis for procedures used in applied settings.

Applied Behavior Analysis – Applied behavior analysis focuses on the application of behavioral principles to socially important behavior (Baer et al. 1968). Applied behavior analysis limits its scope of focus to the improvement of socially important behavior. This is not a limitation of applied behavior analysis, but rather, the need to focus on those behaviors brought to our attention as needing improvement. Methods for assessing the environmental variables that control behavior are consistent between the experimental analysis of behavior and applied behavior analysis. Applied behavior analysis practices include application of reinforcement contingencies, stimulus control procedures, shaping, chaining, and task analysis and are applied to various populations and areas of practice. Applied behavior analysts have formed an accrediting body and established criteria for university coursework and supervision that leads to certification as a board-certified behavior analyst.

Future Directions

Behaviorists continue to evaluate and extend our understanding of the basic principles of behavior

and application of these principles to socially significant behaviors. Extensions to complex human behaviors continue to fields such as pharmacology, neuroscience, performance management, gun safety, interventions for addiction and gambling, and treatment for individuals with neurodevelopmental disorders including autism spectrum disorders.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Classical Conditioning](#)
- [Functional Analysis](#)
- [Operant Conditioning](#)
- [Punishment](#)
- [Reinforcement](#)

References and Reading

- Baer, D., Wolf, M., & Risley, T. (1968). Some current dimensions of applied behavior analysis. *Journal of Applied Behavior Analysis*, 1, 91–97.
- Baum, W. (2005). *Understanding behaviorism*. Malden: Blackwell Publishing.
- Cooper, J. O., Heron, J., & Heward, W. H. (2007). *Applied behavior analysis* (2nd ed.). New York: Pearson.
- Malone, J. C. (2004). Modern molar behaviorism and theoretical behaviorism: Religion and science. *Journal of the Experimental Analysis of Behavior*, 82, 95–102.
- Skinner, B. F. (1974). *About behaviorism*. New York: Knopf.

Behaviorist Theory

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Definition

Behaviorism is widely used to refer to the philosophy of a science of behavior. More specifically, within the field of psychology, behaviorism

explains responses of humans and other animals only in relation to environmental stimuli and observable and measurable responses to those stimuli. There are various forms of behaviorism: structuralism; behaviorism that uses cognition as causal factors (e.g., cognitive behavior modification); social learning theory, in addition to methodological behaviorism; and radical behaviorism. In his text, *About Behaviorism*, B. F. Skinner (1974) wrote: "Behaviorism is not the science of human behavior, it is the philosophy of that science" (Cooper et al. 2007).

Historical Background

Prior to the introduction of behavioral science, the field of psychology consisted of the study of states of mind and mental processes. There are four historical building blocks of behaviorism: classical conditioning as presented by Pavlov, Thorndike's law of effect, Watson's experiments with human conditioning, and Skinner's conceptualization of operant conditioning.

The development of behaviorism is largely attributed to John B. Watson who wrote a seminal article in 1913 in which he argued that psychology should be viewed as a purely objective experimental branch of natural science. As such, the goal should be to study the prediction and control of behavior through direct observation of the relationship of environmental stimuli and resulting evoked responses. This relationship became known as the *stimulus-response (S-R)* paradigm, and Watson proposed that it could be used to predict and control human behavior in a way that would allow practitioners to improve performance in areas such as education, business, and law. Although Watson later made exaggerated claims about the ability to predict and control human behavior, he is recognized for providing a strong case that the study of behavior as a natural science is on par with physical and biological sciences (Cooper et al. 2007, p. 9). The premise that the study of behavior is a science was further expanded upon in a work by B.F. Skinner who was interested in providing scientific accounts of all behavior. Skinner's publication of *The*

Behavior of Organisms (1938, 1966) summarized his laboratory research and gave rise to two kinds of behavior, *respondent* and *operant*. Respondent behavior is behavior that is elicited by a stimulus; it is reflexive and essentially involuntary. Operant behavior is behavior that is influenced by stimulus changes (consequences) that follow the behavior. Skinner argued that the uniqueness of operant behavior warranted its own field of study (see also ► "[Behavior Analysis](#)"). Skinner conducted thousands of laboratory investigations that allowed him to systematically study functional relationships of antecedent stimuli, responses, and reinforcement of those responses in a controlled environment. Skinner's methodology resulted in the foundation of behavior analysis as we know it today.

Current Knowledge

Behaviorism has evolved into many areas of study. It is most widely represented in the disciplines of *experimental analysis of behavior* and *applied behavior analysis*. Within the field of applied behavior analysis, methods of behaviorism have been used to study and affect services in the areas of verbal behavior, public safety, organizational behavior, education, special education, habit reversal, behavioral medicine, cognitive behavior modification, and therapy including derived relational responding as it is related to relational frame theory and acceptance and commitment therapy, social learning theory, functional analysis and assessment of behaviors, and more. The foundation of behaviorism continues as a philosophy of a science of behavior.

Future Directions

As previously noted, behaviorism has been the underpinning for both experimental analysis of behavior and applied behavior analysis. As such, its methodology can be used to study many branches of behavior as long as the behaviors can be operationally defined and observable. As noted by Pear and Eldridge (1984, p. 459),

“Several alternatives to the operant respondent framework have been proposed, but there is no indication that any of these currently has comparable organizing power. Until such a paradigm is put forth, therefore, we see modification of the operant respondent framework, rather than its elimination, as the more fruitful approach.”

See Also

- [Behavior Analysis](#)
- [Behavior Modification](#)
- [Behaviorism](#)

References and Reading

- Cognitive behavior modification*. Texas guide for effective teaching cognitive behavior modification. Retrieved from http://cdd.unm.edu/swan/autism_course/modules/behavior/cbm/index.htm
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). New Jersey: Pearson Merrill Prentice Hall.
- Dymond, S., & Roche, B. (Eds.). (2013). *Advances in relational frame theory: Research and application*. Oakland: New Harbinger Publications, Inc.
- Harris, R., & Hayes, S. C. (2009). *ACT made simple: An easy-to-read primer on acceptance commitment therapy*. Oakland: New Harbinger Press, Inc.
- Hernandez, P., & Ikkanda, Z. (2011). Applied behavior analysis: Behavior management of children with autism spectrum disorders in dental environments. *The Journal of the American Dental Association*, 143(3), 281–287.
- Methodological behaviorism*. Retrieved from www.ucm.es/info/psi/docs/journal/v6_n2_2003/art133.pdf
- Pear, J. J., & Eldridge, G. D. (1984). The operant-responder distinction: Future directions. *Journal of Experimental Analysis of Behavior*, 42(3), 453–467. <https://www.questia.com/library/psychology/other-types-of-psychology/behaviorism>
- Radical behaviorism*. Retrieved from www.behaviorology.org/pdf/PhilPaperOriginsBk.pdf
- Redd, W. H., Porterfield, A. L., & Andersen, B. L. (1979). *Behavior modification*. New York: Random House.
- Rehfeldt, R. A., Barnes-Holmes, Y., & Hayes, S. C. (2009). *Derived relational responding applications for learners with autism and other developmental disabilities*. Oakland: New Harbinger Publications, Inc..
- Skinner, B. F. (1974). *About behaviorism*. New York: Knopf.
- Social learning theory*. Retrieved from http://teachnet.edb.utexas.edu/~Lynda_abbot/Social.html
- Structuralism*. Retrieved from <http://web.mst.edu/~psyworld/structuralism.htm?pagewanted=all>
- Sulzer-Azaroff, B., & Mayer, G. R. (1977). *Applying behavior analysis procedures with children and youth*. New York: Holt Rinehart and Winston.
- Sulzer-Azaroff, B., & Mayer, G. R. (1991). *Behavior analysis for lasting change*. New York: Holt, Rinehart and Winston.
- Vargas, J. S. (2009). *Behavior analysis for effective teaching*. New York: Routledge.

Bell-Shaped Curve

- [Normal Curve](#)

Benadryl

- [Diphenhydramine HCl](#)

Benadryl® Allergy [OTC]

- [Diphenhydramine](#)

Benadryl® Allergy Quick Dissolve [OTC]

- [Diphenhydramine](#)

Benadryl® Children's Allergy [OTC]

- [Diphenhydramine](#)

Benadryl® Children's Allergy Fastmelt® [OTC]

- [Diphenhydramine](#)

**Benadryl[®] Children’s Allergy
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**Benadryl[®] Children’s Allergy
Quick Dissolve [OTC] [DSC]**

► [Diphenhydramine](#)

**Benadryl[®] Children’s Dye-
Free Allergy [OTC]**

► [Diphenhydramine](#)

**Benadryl[®] Dye-Free Allergy
[OTC]**

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Strength [OTC]**

► [Diphenhydramine](#)

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Benchmark

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Benchmarks

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Bender

► [Bender Visual-Motor Gestalt Test II](#)

**Bender Visual-Motor Gestalt
Test II**

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Synonyms

[Bender](#); [Bender-Gestalt II](#); [BG-II](#)

Description

The Bender Visual Gestalt II testing kit includes 16 stimulus cards that are separated into two tests. These stimulus cards include an improved version of the original nine designs and new cards that were constructed to be more fitting for the age range covered by the test. All of the stimulus cards have been mechanically drawn to increase the clarity of the design.

The administration of the Bender-Gestalt II is considered to be user-friendly and relatively easy. It occurs in two phases: the copy phase and the recall phase. During the copy phase, the examinee is presented with the age-appropriate stimulus cards one at a time and instructed to copy each design onto a blank, white sheet of paper using a No. 2 pencil. In the recall phase, the examinee is instructed to draw as many of the designs as they can from memory onto a new sheet of paper. While there are no time limits for any of the designs or phases, the examiner should begin timing immediately following the presentation of the first design, in order to keep track of the amount of time needed for the examinee to complete each separate design. The examiner should also pay attention to behavioral and physical characteristics of client. Such observation can help determine if poor reproductions of a design are the result of impaired motor or perception abilities.

To score the Bender-Gestalt II, a new Global Scoring System has been outlined. This scoring system evaluates the examinee reproduction of designs at the copy and recall phases and rates the quality on a five-point scale. A score of 0 is given to designs that have no resemblance to the design or are the product of random drawing or scribbling. A score of 4 is given to those designs that are nearly perfect in their resemblance to the design. This scoring system is considered to be fairly simple as specific examples of the Global Scoring System are provided in the manual. However, it requires rigid adherence to the scoring examples and much stricter than previous scoring methods.

Historical Background

The Bender Visual Motor Gestalt Test was first published in 1938 by the American Orthopsychiatric Association under the title of "A Visual Motor Gestalt Test and Its Clinical Use." It evolved from Max Wertheimer's early studies of a Gestalt theory of perception. Lauretta Bender adapted nine of Wertheimer's designs and put them on cards in order to understand the

gestalt experiences of psychiatric patients. Specifically, the test was designed as a screening measure to test the ability of the perceptual system to organize visual stimuli into configural wholes, as a screening measure for neuropsychological damage. It quickly grew in popularity because it was brief and fairly simple to score and administer. Since its original development, the test has undergone many revisions that have largely focused on changes in interpretation and scoring procedures.

A wide variety of scoring procedures have been developed over the years using the original Bender-Gestalt Test. Among the most notable are the Koppitz's Developmental Bender Scoring System, published in 1964 as *The Bender-Gestalt Test for Young Children*, and Max Hutt's Scoring System. Under Koppitz scoring system, 30 discrete errors are scored if present, with each design ranging from 2 to 4 possible errors. This scoring procedure was designed to measure neuropsychological impairment and the developmental maturation of children. Hutt's Scoring System, on the other hand, was designed to use the Bender-Gestalt Test as a projective personality assessment for adults. It scored tests based on the frequency and severity with which an examinee deviated from protocol. Koppitz's original scoring system was adapted after her death by Cecil Reynolds (2007).

Notable psychometric problems with the original version limit interpretation of data from studies utilizing that test. Several studies in the earliest history of autism research utilized the original version of the Bender, but in the context of psychometric problems with the test as well as the lack of standardized diagnostic criteria for autism, these studies are not considered relevant.

The test was included in Norcross et al. (2006) list of "Discredited Psychological Treatments and Tests" based on ratings by a large expert panel, either for use in screening neuropsychological impairment or personality function. This presumably referred to the original version of the test and its uses, rather than to the revised Bender-Gestalt II.

The second edition of the Bender Visual-Motor Gestalt Test was published in 2003. This new

edition is a product of many years of analysis with the first edition of the test, as well as modern research in the fields of psychological testing and test construction. This comprehensive revision added four easier items and three harder items in order to increase the measurement scale. In other words, it lowered the “floor” of the test and created a higher “ceiling” so as to better describe those individuals who score on the extremes of the spectrum.

Psychometric Data

The Bender-Gestalt II was normed from a stratified, random sampling of 4000 subjects that comparatively matched US census data from the year 2000. T-scores, percentile ranks, confidence intervals, and classification labels are available for subjects ages 4 to 85+ years.

The psychometric properties of the test are fairly strong. Interrater reliability is reported at a range of .83 to .84 for the copy phase and .94 to .97 for the recall phase. A validity of .91 was found using split-half procedures. Over a 2–3 week interval, test-retest reliability is between .80 and .88 for the copy phase and .80 to .86 for the recall phase.

Construct validity for the Bender-Gestalt II has been supported by moderate correlations to other measures. For example, it has moderate correlation of .65 with the Beery-Buktenica Developmental Test of Visual-Motor Integration and a correlation of .75 with the Perceptual Organization factor on the WISC-III.

Clinical Uses

The Bender Gestalt II is designed to assess the visual-motor integration abilities of children and adults from 4 to 85+ years of age. It is also designed to be used as a test of motor memory in children and adults ages 5 to 85+. It has been used to identify brain dysfunction in children and adults, and discern emotional problems in children. Generally, if the Bender-Gestalt II is being used to assess for brain damage, it should be

considered a screening device as it is limited to severe forms of brain damage.

Allen and Decker (2008) found significant differences to indicate impaired performance, after controlling for IQ, in a moderately sized sample of children (mean age = 11) diagnosed with attention-deficit/hyperactivity disorder compared to a healthy comparison group, suggesting possible utility as a measure of function in other disorders autism. Effect sizes were very small, however. One study (Volker et al. 2010) has used the Bender Gestalt II to analyze the visual-motor skills of individuals with autism spectrum disorders. In demographically matched subsamples of ASD and healthy children (mean age = 9.7; $n = 27$ for each group), and after statistical control for IQ, a high-functioning autism spectrum disorder group scored lower than the comparison group on the two tests most sensitive to motor function (the copy and supplemental motor scales).

There appears to be substantial justification for continued investigation of atypical motor function in autism. A recent review by Dowd et al. (2010) notes the potential utility of motor function as (a) a diagnostic marker of autism, (b) an endophenotype of autism, and (c) a marker of severity of impairment, including social-communicative impairment. The Bender-Gestalt II could be used in future studies to characterize basic motor deficits and possibly higher order problems with visuomotor planning and organization.

See Also

► [Bruininks-Oseretsky Test of Motor Proficiency](#)

References and Reading

- Allen, R. A., & Decker, S. L. (2008). Utility of the bender visual-motor gestalt test-second edition in the assessment of attention-deficit/hyperactivity disorder. *Perceptual and Motor Skills*, 107, 663–675.
- Brannigan, G. G., & Decker, S. L. (2003). *Bender visual-motor gestalt test* (2nd ed.). Itasca: Riverside Publishing.

- Brannigan, G. G., Decker, S. L., & Madsen, D. H. (2004). *Innovative features of the Bender-Gestalt II and expanded guidelines for the use of the Global Scoring System* (Bender visual-motor gestalt test, Second Edition Assessment Service Bulletin No.1). Itasca: Riverside Publishing.
- Dowd, A. M., Rinehart, N. J., & McGinley, J. (2010). Motor function in children with autism: Why is this relevant to psychologists? *Clinical Psychologist*, 14, 90–96.
- Norcross, J. C., Koocher, G. P., & Garofalo, A. (2006). Discredited psychological treatments and tests: A Delphi poll. *Professional Psychology: Research and Practice*, 37, 515–522.
- Reynolds, C. R. (2007). *Koppitz-2: The Koppitz developmental scoring system for the Bender-Gestalt Test*. Austin: Pro-Ed.
- Volker, M. A., Lopata, C., Vujnovic, R. K., Smerbeck, A. M., Toomey, J. A., Rodgers, J. D., et al. (2010). Comparison of the Bender Gestalt-II and the VMI-V in samples of typical children and children with high-functioning autism spectrum disorders. *Journal of Psychoeducational Assessment*, 28, 187–200.

Bender, Lauretta

Fred R. Volkmar

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Name and Degrees

Lauretta Bender MD

- B.A. (1922) University of Chicago
- M.A. (1923) University of Chicago
- M.D. (1926) State of University of Iowa

Major Appointments (Institution, Location, Dates)

Bender held positions at the Hospital of the University of Chicago, the Boston Psychopathic Hospital, the University of Amsterdam, the Johns Hopkins University Hospital, and Bellevue Hospital in New York, as well as at the University of Maryland.

Major Honors and Awards

In 1955, Dr. Bender was the recipient of the Adolf Meyer Memorial Award from the American Psychiatric Association for her work on severe psychiatric disturbance in children.

Landmark Clinical, Scientific, and Professional Contributions

Loretta Bender was an early pioneer in the study of learning disabilities and severe psychiatric disturbance in children. Highly active at the professional level both as a clinician and researcher, she was involved in development of various approaches to treatment and to theories of childhood psychopathology. The Bender-Gestalt test remains in use today. Her view of learning disabilities was based on a theory related to discrepancies in areas of maturation, and she emphasized the confluence of various problems in children with learning problems that reflected their common origins. She also worked in the area of language difficulty and conducted some of the early work on reading disability. Her work was conducted at a time when childhood schizophrenia/childhood psychosis was used to describe all severe neuropsychiatric disturbance, i.e., before the distinction of autism as a distinctive diagnostic category was made.

Short Biography

A native of Butte, Montana, Lauretta Bender coped with a significant learning difficulty but persevered to become the valedictorian of her high school class. She received her B.A. (1922) and M.A. (1923) from the University of Chicago. She received an M.D. from the State of University of Iowa (1926). Bender held positions at the Hospital of the University of Chicago, the Boston Psychopathic Hospital, the University of Amsterdam, the Johns Hopkins University Hospital, and Bellevue Hospital in New York as well as at the University of Maryland. Bender was active in many ways at the professional level. She was Director of Research of the new Children's Unit

at Creedmoor State Hospital in the 1950s and while there conducted much of her work with severely impaired children.

Her first husband, the psychiatrist Paul Schuler (1886–1940), tragically died after a few years of marriage. She married Henry B. Parkes, a professor at New York University, in 1954.

See Also

► [Bender Visual-Motor Gestalt Test II](#)

References and Reading

- Bender, L. (1969). A longitudinal study of schizophrenic children with autism. *Hospital & Community Psychiatry*, 20(8), 230–237.
- Bender, L. (1971). Alpha and omega of childhood schizophrenia. *Journal of Autism and Childhood Schizophrenia*, 1(2), 115–118.
- Bender, L. (1973). The life course of children with schizophrenia. *American Journal of Psychiatry*, 130(7), 783–786.
- Bender, L. (1974). The family patterns of 100 schizophrenic children observed at Bellevue, 1935–1952. *Journal of Autism and Childhood Schizophrenia*, 4(4), 279–292.

Bender-Gestalt II

► [Bender Visual-Motor Gestalt Test II](#)

Beneficiary

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Definition

Basic Definition

A beneficiary is a person for whose benefit property is placed in trust. The beneficiary is

the third of three ingredients critical to the creation of a trust: (1) property, usually money, placed in a trust administered by (2) a trustee for the benefit of (3) a beneficiary (restatement). A trust cannot exist without a beneficiary (Bogert, 121). On occasion, American courts refer to the beneficiary by the French phrase *cestui que trust*. A trust may have multiple beneficiaries. The trust documents dictate when and how much of the trust property a beneficiary will receive.

Who May Be a Beneficiary

Any legal entity, including individuals or corporations, may be a beneficiary (Bogert, 125).

But, only the entities intended by the creator of the trust, or settlor, to benefit from the trust may be a beneficiary. The settlor may be a beneficiary, and even the trustee may be a beneficiary as long as he or she is not the sole trustee.

Many trusts have multiple beneficiaries who can be named individually or can be designated as a “class,” such as all of the children of a particular person.

Rights of a Beneficiary

A beneficiary’s interest in a trust varies according to the type of trust created (Dietz). In a “fixed trust” in which the benefits are spelled out precisely by the trust documents, the beneficiary has an ownership interest in the trust proceeds. If the trust is a “discretionary trust,” meaning that the trustee has discretion as to when and how much of the trust property to give to the beneficiary, a beneficiary’s interest is subject to the determination of the trustee.

A beneficiary may refuse the benefits of the trust by disclaiming her right to them. The disclaimer may be implied by conduct “inconsistent with a trust for his” (Bogert, 170) ASD-related issues.

See Also

- [Discretionary Trust](#)
► [Support Trust](#)
► [Trust](#)

References and Reading

- Bogert, G. T. (1987). *Trusts* (6th ed.). St. Paul: West Publishing.
- Bogert, G. T., & Bogert, G. B. (1987). *The law of trusts and trustees* (1987). St. Paul: West Publishing.
- Garner, B. A. (Ed.). (2009). *Black's law dictionary* (9th ed.). St. Paul: West Publishing.
- Laura Dietz, L., Lindsley, W., Martin, L., Payne, A., Shampo, J., & Surette, E. C. (1998–2011). *Trusts* (American Jurisprudence). St. Paul: West Publishing.

Benhaven Residential Services

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Definition

Benhaven is an agency that provides educational, day program, and residential supports to adults and adolescents with autism and developmental disabilities in New Haven, Connecticut, and surrounding towns. The residential services division of the program, which is the topic of discussion in this entry, entails a variety of programs including in-home consultation, family respite, professional parent settings, and group homes. In each setting, the emphasis is on individualized programming and is driven by the principles of positive behavioral support (Koegel et al. 1996; Lavigna and Donnellan 1986; Smull and Harrison 1992; Structured Teaching 2010).

Historical Background

Benhaven's residential services developed in response to the overwhelming need for such services in the early 1970s. Amy Lettick, the director and founder of Benhaven School and the mother of an autistic son, Ben, recognized that need. She believed that in order for many children with autism and their families to function optimally, the continuity and consistency of 24-h programming and care was necessary. In addition, during

the late 1960s and early 1970s, quality educational programs were few and far between, so the development of a residential program allowed Benhaven to accept many students in need.

Benhaven School was established in 1967, followed in 1972 by the purchase and development of one home, the beginning of Benhaven's residential program. With many more applicants looking for both school and residential support, more homes were opened, and by 1991, a total of 7 homes served 34 residents. In 1990, Benhaven introduced its Shared Living Program. This program gives people the opportunity to live in a typical home with the skilled support of licensed families and individuals. In 1996, the Individual and Family Support Services Program was created to provide support to children and families in their own home.

Benhaven has had a long-standing supportive relationship with the Yale Child Study Center, particularly with Dr. Fred Volkmar and the late Dr. Donald Cohen. In the mid-1980s and early 1990s, under the direction of its (now retired) Executive Director, Larry Wood, Benhaven underwent a major shift in its understanding of state-of-the-art approaches to teaching and supporting people with autism. Benhaven's administrative, teaching, and managerial staff received extensive training in structured teaching practices, functional behavioral support, and team building, conducted by expert leaders in the field, such as Dr. Gary LaVigna and Dr. Anne Donnellan. This training changed how Benhaven provides services to its program participants and training to its staff. This rigorous approach to staying informed about best practices in the field of autism has continued at Benhaven throughout the years. Under the leadership of Benhaven's new Executive Director, Kathryn DuPree, plans for further residential development to meet the growing need for adult residential services are underway.

Rationale or Underlying Theory

The rationale for Benhaven's approach to residential services is that, based on experience,

individuals with autism have a unique set of learning characteristics that respond well to a visual and structured approach to teaching. Positive behavioral intervention is an established approach that begins with understanding the factors that drive behavior through a process of functional analysis of problem behavior (Fox et al. 2000; O'Neill et al. 1997). When functions of behavior can be identified, an individualized behavior support plan is designed and implemented. The majority of interventions that make up the plan are preventative and positive in nature. They are aimed at maximizing a comfortable physical and social environment for the individual, recognizing and preventing triggers to challenging behavior, and teaching alternative, appropriate means of communicating needs and wishes. The plan also includes carefully planned interventions for managing challenging behavior when it does occur. This approach requires having knowledgeable, educated, and well-trained leadership as well as direct support staff and also requires that supporters' professional development plans include goals to establish positive relationships with the residents in their care.

The most important aspect of the relationship between support provider and the person receiving support is respect. Respect is the cornerstone upon which all support services should be rendered. The roles and tasks assumed by the provider of support will not be as effective if this foundation of respect is missing. The support provider's respect should not have to be earned. It must be there unconditionally.

Part of the challenge of this work is learning how to provide choices, honor preferences, and respect the person's individuality while satisfying the professional responsibilities associated with helping the person stay safe and healthy, helping the person learn, helping the person gain new experiences, and helping the person avoid disappointment or embarrassment. Responding to this challenge is what makes the work more of an art than a science. Balancing the role of supporting what the person wants with the temptation to control because "teacher knows best" can be frustrating, and supporters may find themselves too far in one direction or the other. Approaching the

work from the foundation of respect allows continual assessment of that balance while struggling to carry out multiple and sometimes conflicting roles. Constantly striving to learn and do better is part of the job.

Goals and Objectives

The approaches described here are intended to address issues that exist for people with autism as a result of the inability to communicate or relate to the social world in a typical way (American Psychiatric Association 2000). Challenging behavior is a frequent problem in autism and can be highly interfering to a person's relationship with one's family, to relationships with peers and teachers, to learning, and to one's ability to develop functional life skills and experience a broad range of opportunities. Challenging behavior is most often the result of a person's inability to communicate needs, desires, discomfort, and refusal, and many of the other things a person typically resolves through the use of spoken or symbolic language. Many people with autism, due to the social delay inherent in the disability from infancy, have not learned the vital process of getting needs met through social interactions with others (Volkmar and Wiesner 2009). In the absence of language or another way to express one's needs, maladaptive behavior evolves as an often successful means to quickly and effectively make choices, refuse to participate, or obtain something desired.

Other characteristics of autism, for example, distractibility, or need for sameness, can interfere with learning and compound a cognitive delay. Heightened senses in many people with autism create other barriers to participation. For example, a sensitivity to noises may limit the physical and social environments in which a person may be comfortable, in this way narrowing a person's experiences and learning opportunities. Additionally, each person with autism has a unique set of characteristics and a distinctive learning style. Given all these factors, supporting and teaching people with autism in a residential setting requires a multilayered approach.

One objective in this process is the thoughtful establishment of an environment that makes sense for an individual, taking into consideration both the physical aspects of a setting, such as the amount and type of space that are important, and the social environment, involving the number of others living and working in the setting. Another extremely important factor is the establishment of a means of communication for each individual, including the teaching of the skills necessary to utilize that communication method. A behavior support plan is a vital component of a residential program, which guides instructors in the prevention and response to whatever challenging behaviors may exist, while teaching appropriate alternatives to behavior.

Skill development is another objective of the program. Functional skills are taught in the context of caring for one's home and oneself while living with cognitive, communication, and behavioral challenges. Skills to participate in the community, including recreational as well as functional and employment settings, are also essential. Other goals of the program include establishing and/or building a person's relationship with his or her family and friends and attending to a person's health and medication needs. In short, the responsibility is to teach skills and provide opportunities to empower and equip residents with a range of appropriate choices and productive control that will enhance their quality of life.

Treatment Participants

Those who are most likely to benefit from treatment at Benhaven's residential program are individuals whose primary diagnosis fits the DSM-V diagnostic criteria for autism spectrum disorder and others with cognitive delays and behavioral symptoms similar to those experienced in autism. This is the case because treatment is designed to address the specific learning characteristics of people with autism. However, the strategies described here could be beneficial to others experiencing learning or behavioral challenges, since the primary characteristics involve individualized and structured teaching. While Benhaven's residential program

currently serves adolescents and adults, the treatment procedures are suitable for people with developmental disabilities of any age. Many of the program participants have additional psychiatric diagnoses that are addressed through consultation with outside providers. Because the treatment emphasis is on autism and symptoms of other developmental delay, those with other primary psychiatric diagnoses without cognitive delay would not be well suited to treatment at Benhaven's residential program.

Treatment Procedures

All treatments begin with the process of conducting assessments, which is the first step to designing an individualized program for a person with autism.

Functional Behavioral Assessment and Behavior Support Plan Development

A functional behavioral assessment is a means of identifying the important factors that contribute to the existence of challenging behavior in a person with autism. This process involves interviewing the team of people supporting the individual to determine what factors probably cause and maintain the target behavior(s). This process identifies likely functions of that behavior and tests these hypotheses through observation of the individual in his or her life settings to determine whether the identified functions are supported. Once functions of behavior are established, a series of strategies are developed in the form of an individualized behavior support plan. This plan lays out components to address the target behavior(s) from different angles. Preventative strategies are designed to predict and prevent behavior from happening through management of antecedent settings and events. For example, it might direct instructors to give a person a "heads up" that a difficult transition is coming. Rather than interrupting the person relaxing with a magazine and announcing "It's time to do your laundry," a situation that is likely to cause a target behavior to occur, the instructor may be directed to provide the individual with a verbal and visual countdown. The instructor will

verbally alert the person about the change in activity at 1-min intervals and will use a visual timer to more concretely help the person recognize the passage of time.

The behavior support plan also contains teaching and coping strategies. Coping strategies are means for a person to handle the challenges of the environment or demands in the daily schedule. For example, rather than striking out in frustration when the environment is too noisy or overwhelming, a person may be taught to simply leave the area for a quieter environment within his home. Or, a person may be taught to utilize calming techniques, such as music or deep breathing, when he senses himself becoming overwhelmed. He may also be taught a means to communicate his need, for example, saying “it’s too loud” as a means to ask an instructor to help find a quieter place. Teaching strategies teach a person to engage in alternative actions to the target behavior. These alternatives are meant to be positive and equally efficient ways to get the same needs met. When negative behavior is the result of inability to communicate in a functionally appropriate manner, teaching strategies will involve teaching a person a means to communicate something he was not able to initiate on his own before, such as a need, a desire to change activities or environment, or to refuse something. Teaching strategies also involve teaching a person skills to accomplish things that may have only been accomplished before through behavior, for example, learning to ask for a break rather than hitting the instructor to indicate frustration with the activity. Teaching strategies can also be a means to help someone predict events and have a sense of order in life through the use of visual schedules and scripts. This will be described further below.

Finally, behavior support plans contain strategies for providing consequences to challenging behavior and for responding to challenging behavior if and when it does occur. The term “consequence” refers to the events that occur immediately following the occurrence of a behavior, whether positive or negative. The way instructors respond to the behavior has a major impact on whether the behavior is strengthened or weakened. The goal, of course, is to strengthen positive behaviors and

weaken negative ones, so the strategies are designed to do just that. The consequence for engaging in a positive behavior, such as utilizing communication to ask for help, is to provide the help and offer praise for the communication. Done consistently, this will strengthen the communication response. Done inconsistently, for example, by saying, “I’m glad you asked for help, but I think you can do that on your own,” will result in a weak learning and erratic use of the skill. Concurrently, the response to negative behavior must be clearly designated and consistently applied. If the function of screaming at the instructor is to end the activity, then responding to screaming by ending the activity will maintain that behavior. Rather, the plan might designate that breaks are initially built in after very short work periods, preempting the person from becoming frustrated and acting out. The resident is shown how to ask for a break, perhaps by sign, picture, or words, and then is guided to do that before any frustration sets in. He is immediately rewarded with his break for engaging in the positive behavior. Screaming would be ignored, while the person was helped to utilize communication to ask for a break.

Reactive strategies are designed to manage a crisis situation and direct instructors in how to respond when challenging behavior does occur. These response strategies are not intended to teach the person any new skills. The teaching comes from all the prior components – antecedent, teaching, coping, and consequence strategies. Reactive strategies are an important part of the plan because it is desirable and necessary to train instructors in proper, safe response techniques rather than rely on improvisation in the heat of the moment. Techniques that have proven to be the most effective and safe *for that individual* are carefully designed and trained. Reactive strategies often include some kind of debriefing for the individual and for the instructors to help everyone get back on track once the incident is over.

Skills Assessment and Individualized Program Development

Another important component of teaching individuals with autism in a residential setting is the creation of an individualized learning plan.

While each person's program is specifically tailored to his or her specific learning needs, there are some learning style characteristics that many people with autism share and which form the basis for a learning program for a person living in a residential setting. Examples of some broad learning characteristics in autism include difficulties with changes in routines and schedules, distractibility, trouble organizing and filtering out relevant information, difficulty with auditory processing, and poor understanding of social cues. It is important to design a learning environment and individualized program that takes these characteristics into consideration while also factoring in the unique learning style of the individual himself.

Components of a residential program that utilize a structured approach to teaching include the strong use of visual supports. Visual supports are those environmental and teaching tools that rely primarily on the visual rather than auditory modality to help teach a person with autism. Visual supports are used to help organize the environment, for example, by labeling drawers and cabinets with pictures and/or words, by arranging furniture and other items to create work and recreational spaces, by color coding and using other visual cues, and by storing and arranging materials in clear plastic containers or designated closets so the contents can be accessed easily. Visual supports are also used to help a person organize, understand, and predict his routine. These take the form, for example, of picture schedules, written checklists, or pictorial or written scripts. One type of visual support that is well suited to a learner with autism is the use of a "first . . . then" map, where a person who is anxious for or motivated to engage in a specific activity can see from pictures or words that once the less preferred task is over, the preferred activity can take place:

- "First clean your bedroom, then play video games."

A consequence chain is another visual tool that helps a learner see and predict the course of events:

- "It's sunny outside: I can go swimming.
- It's raining out: I cannot go swimming: I will go to the gym."

There are dozens of tools and variations that can be created to visually assist a learner to understand his routine, including the use of modeling, pointing, sign language, photographs, objects, video, and even handheld devices and applications.

Lastly, visual strategies are very widely used when teaching skill-building activities. Unlike typical teaching approaches done through presentation of verbal material, visual strategies present information and provide direction more concretely. The spoken word is transitory and fleeting in nature and relies heavily on strong auditory processing skills, whereas visual tools are tangible and enduring. Some examples of visual instructional tools are picture recipes that lay out directions for preparing a meal or picture checklists for grocery shopping. The method of displaying the visual tools is based on the individual's strengths. For some, laying the pictures out sequentially on one long strip is effective; for another, gathering them in a photo album which the learner turns as he completes each step is more suitable. Picture strips may be posted on a wall to assist a person through a step-by-step process, such as brushing one's teeth. Cards or pictures can be kept in a wallet which the learner can pull out as needed. Visual tools are also useful in assisting learners to make choices. Giving the learner two or more concrete visual items to choose from increases the likelihood the person will select the truly preferred item, rather than simply repeating the last item heard, which often happens when people are verbally asked to make a choice.

The teaching method most commonly and effectively used at Benhaven is "errorless learning," a system of teaching a person skills through the use of carefully designed and implemented prompts (Etzel and LeBlanc 1979; McDuff et al. 2001). Skills are broken down into a series of steps, called a "task analysis," and instructors provide prompting designed to help the person complete each step before making an error. Prompts are planfully faded as the learner gains independence at each step. Instructors are trained to provide prompts that are best suited to that individual's needs. A person with strong memory and

visual skills may be successful following a series of pointing prompts – the instructor points to each step in the sequence and the learner responds by engaging in the step. To make a sandwich, for example, the instructor points to the loaf of bread, the learner takes out two slices of bread; the instructor points to the filling, the learner places the filling on the bread; etc. Another learner with greater needs may be more successful with physical prompts, where the instructor guides the person's hands to take out the bread and place it on the plate, take the filling and place it on the bread, etc. Other types of prompts include modeling, gesturing, signing, and, of course, visual supports like photos, pictures, or written instructions. Verbal prompts are not widely recommended for people with autism as they rely too heavily on auditory processing skills and, unlike the other types of prompts, are not easily conducive to fading.

Efficacy Information

The methods described here, including functional behavioral assessment, positive behavioral intervention, structured teaching, and errorless learning, are all widely used in the field of education for people with developmental disabilities and are accepted as effective methods to teach positive behavior and functional skills to those on the autism spectrum (Connecticut State Department of Education 2005). Success of these strategies in a residential setting relies heavily on individualized program planning, consistency of implementation by well-trained and caring direct support staff, and strong oversight of routines and practices by knowledgeable administrative personnel.

Outcome Measurement

Treatment outcomes are measured using a variety of tools. Progress toward reducing negative behaviors and increasing adaptive ones are measured through behavioral data charts that track frequency, duration, and intensity of behavior.

Observational and anecdotal reports are commonly used to track the circumstances under which a behavior occurred. Positive behavior is tracked as well, for example, the frequency with which a person engages in a new, positive behavior or skill and under what circumstances. Mood and behavior may also be assessed via rating scales, for example, by rating a person's affect against a certain set of observable and describable attributes. Data is turned into graphs and charts so that it may be more easily reviewed and analyzed by team members.

Progress in skill acquisition is measured in a variety of ways, for example, by recording the number and type of intervention a person needs to perform the steps of a given activity. Progress may be measured in terms of moving from more severe to minimal prompts (prompt fading), by the reduction of the number of prompts or corrections or by tracking the rate at which steps are performed without intervention.

Less quantitative methods of assessing progress are "quality of life" measures. Does the resident's behavior allow him to engage in activities that bring him pleasure and satisfaction, for example, eating out at restaurants, going to the movies, going to parties, and taking a vacation? Can a resident who formerly needed one-to-one supervision when out in the community now go out with others? Can a resident who used to sit in the back seat of a vehicle because of safety concerns now sit up front with the driver if he prefers? The answers to questions like these provide another equally important and valid measure of treatment outcome.

Qualifications of Treatment Providers

Benhaven's residential program is licensed by the Connecticut Department of Developmental Disabilities and must adhere to that agency's licensing requirements, including requirements in the area of staff training. While not required, many management and administrative staff hold licenses and degrees in areas such as special education, psychology, sociology, and social work. Direct line personnel are not required to hold

specific credentials but receive extensive and ongoing training in autism and teaching methods and philosophies as well as other areas vital for supporting people with disabilities, for example, CPR, first aid, medication administration, and health and safety.

What distinguishes Benhaven's treatment procedures from others is not so much the treatments themselves (which are widely accepted and established approaches and strategies in the field of autism) but, rather, other factors that impact the efficacy of treatment. First of all, program plans are individualized in reality, not just on paper. One of Benhaven's primary treatment philosophies is that there must be an adequate number of well-trained staff to carry out these programs so that residents receive individualized training for skill acquisition and maintenance. For a residential program, Benhaven manages to maintain a remarkably high staff ratio, for example, four staff to six residents during programming time in some settings and in one setting two staff to two residents. Strategies for teaching skills and addressing behavior problems are developed to best meet the needs and suit the learning style of the individual; "one size fits all" is never the approach.

Treatment is also enhanced by the relative longevity and retention of skilled and motivated staff at all levels. Benhaven's residential administration has been in place for between 30 and 40 years; senior management of the seven homes averages over 18 years of employment at Benhaven. Advanced direct support staff constitute over 80% of the total supporters, and the average employment among all direct support staff is well over 5–10 years. In addition, and perhaps most importantly, staff receive ongoing training in groups, in team meetings, and as individuals. Staff performance is monitored through formal and informal observation, with feedback and follow-up provided. All staff, full- and part-time, participate in a staff development process that provides people with an opportunity to receive performance feedback on a quarterly basis. Formal staff evaluations are conducted annually, at which time performance goals are established and reviewed.

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.). Washington, DC: APA Press.
- Connecticut State Department of Education, Division of Teaching and Learning Programs and Services, Bureau of Special Education. (2005). *Guidelines for identification and education of children and youth with autism*. Hartford: Connecticut State Department of Education.
- Etzel, B. C., & LeBlanc, J. M. (1979). The simplest treatment alternative: The law of parsimony applied to choosing appropriate instructional control and errorless-learning procedures for the difficult-to-teach child. *Journal of Autism and Developmental Disorders*, 9(4), 361–382.
- Fox, L., Dunlap, G., & Buschbacher, P. (2000). Understanding and intervening with children's challenging behavior: A comprehensive approach. In A. Wetherby & B. Prizant (Eds.), *Autism spectrum disorders: A transactional developmental perspective* (Vol. 9, pp. 109–141). Baltimore: Paul H. Brookes.
- Goldstein, H. (2002). Communication intervention for children with autism: A review of treatment efficacy. *Journal of Autism and Developmental Disorders*, 32(5), 373–396.
- Koegel, L., Koegel, R., & Dunlap, G. (1996). *Positive behavioral support: Including people with difficult behavior in the community*. Baltimore: Paul H. Brookes.
- LaVigna, G. W., & Donnellan, A. M. (1986). *Alternatives to punishment: Solving behavior problems with non-aversive strategies*. New York: Irvington.
- Maurice, C., Green, G., & Luce, S. (Eds.). (1996). *Behavioral intervention for young children with autism: A manual for parents and professionals*. Austin: Pro-Ed.
- McDuff, G. S., Krantz, P. J., & McClannahan, L. E. (2001). Prompts and prompt-fading strategies for people with autism. In C. Maurice, G. Green, & R. M. Foxx (Eds.), *Making a difference: Behavioral intervention for autism* (pp. 37–50). Austin: Pro-Ed.
- Mount, B. (1995). *Capacity works: Finding windows for change using personal futures planning*. New York: Graphic Futures.
- O'Neill, R. E., Horner, R. H., Albin, R. W., Sprague, J. R., Storey, K., & Newton, J. S. (1997). *Functional assessment and program development for problem behavior* (2nd ed.). Pacific Grove: Brooks/Coles Publishing Company.
- Prizant, B., Wetherby, A., & Rydell, P. (2000). Communication intervention issues for young children with autism spectrum disorders. In A. Wetherby & B. Prizant (Eds.), *Autism spectrum disorders: A transactional developmental perspective* (Vol. 9, pp. 109–141). Baltimore: Paul H. Brookes.
- Smull, M., & Harrison, S. B. (1992). *Supporting people with severe reputations in the community: Reviewing essential lifestyle plans*. Alexandria: NASMRPD.

Structured Teaching. (2010). Retrieved 12 June 2010 from <http://www.teacch.com>.

Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know*. Hoboken: Wiley.

Zions, P., & Simpson, R.L. (1992/2000). *Autism: Information and resources for professionals and parents* (2nd ed., p. 3/5). Austin: Pro-Ed.

Benzodiazepine

- [Lorazepam](#)
- [Xanax \(Alprazolam\)](#)

Benzodiazepines

- [Sedative Hypnotic Drugs](#)

Beta-Adrenergic Antagonist

- [Beta-Adrenergic Blockers](#)

Beta-Adrenergic Blockers

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Synonyms

[Beta-adrenergic antagonist](#)

Definition

Beta blockers remain essential pharmacological agents in the management and treatment of cardiovascular and endocrine conditions. With little risk of dependence and high target selectivity, recent clinical trials have investigated the efficacy of beta blockers alongside current treatment guidelines for intellectual disabilities and psychiatric conditions

where first-line therapies have failed. Current research suggests that beta blockers may alleviate the motor and psychiatric symptoms present in intellectual disabilities through CNS or peripheral blockade of sympathetic hyperactivity (Connor et al. 1997). As such, beta blockers may act as a potential adjuvant therapy towards the management and treatment of autism spectrum disorders (ASD). Among autistic patients, 30% exhibit anxiety, irritability, and self-injury comorbidities, which previous pharmacological studies targeted for treatment. Recent studies showed beta blockers, particularly propranolol, reduced these comorbidities, sparking new interest examining beta blockers effects on the language and cognitive deficits in ASD (Sagar-Ouriaghli et al. 2018).

Propranolol is a nonselective beta blocker used extensively to treat test and performance anxiety and cardiovascular disease. Unlike other nonselective beta blockers, such as nadolol, propranolol is a lipophilic beta blocker that blocks both the central nervous system and peripheral β -adrenergic receptors allowing for further modulation of cognitive functions (Beversdorf et al. 2002). Furthermore, propranolol is well-tolerated by children and exhibits less unwanted side effects compared to other pharmaceutical agents (Deepmala and Agrawal 2014). Many ASD patients show a decrease connectivity between brain regions involved in language, social, and motor skills (Koshino et al. 2005). Previous studies using propranolol showed ASD children exhibited less anxiety and more social and adaptive behaviors (Ratey et al. 1987). Further analysis demonstrated propranolol's efficacy improving word fluency, conversation problem solving, and conversational reciprocity in ASD patients (Zamzow et al. 2016). In addition, ASD patients show deficits in phonological processing during social and motor tasks (Schmidt et al. 2008). A functional magnetic resonance imaging (fMRI) of 10 autistic patients showed an increase in neuronal networks with propranolol administration during phonological tasks compared to nadolol (Narayanan et al. 2010).

Although social and cognitive deficits remain the focus of autism research, hypersexuality remains a prevalent comorbidity among ASD patients leading to social embarrassment and

repercussions with limited pharmacological interventions available (Deepmala and Agrawal 2014). However, a recent case report of an adolescent ASD patient demonstrated decreased hypersexual behavior upon administration of propranolol (Deepmala and Agrawal 2014). Although the mechanism behind the observed decreased hypersexual behavior remains unknown, current literature suggest that propranolol decreases testosterone and antagonizes serotonin receptors in the brain (Rosen et al. 1988). Overall, these findings encourage future investigations comparing the efficacy of different beta blockers in managing ASD and the mechanism behind their effects.

See Also

- Autonomic Nervous System
- Irritability in Autism
- Learning Disability

References and Reading

- Beyersdorf, D. Q., White, D. M., Chever, D. C., Hughes, J. D., & Bornstein, R. A. (2002). Central β -adrenergic modulation of cognitive flexibility. *Neuroreport*, 13(18), 2505–2507. <https://doi.org/10.1097/00001756-200212200-00025>.
- Connor, D. F., Ozbayrak, K. R., Benjamin, S., Ma, Y., & Fletcher, K. E. (1997). A pilot study of Nadolol for overt aggression in developmentally delayed individuals. *Journal of the American Academy of Child & Adolescent Psychiatry*, 36(6), 826–834. <https://doi.org/10.1097/00004583-199706000-00021>.
- Deepmala, & Agrawal, M. (2014). Use of propranolol for hypersexual behavior in an adolescent with autism. *Annals of Pharmacotherapy*, 48(10), 1385–1388. <https://doi.org/10.1177/1060028014541630>.
- Koshino, H., Carpenter, P. A., Minshew, N. J., Cherkassky, V. L., Keller, T. A., & Just, M. A. (2005). Functional connectivity in an fMRI working memory task in high-functioning autism. *NeuroImage*, 24(3), 810–821. <https://doi.org/10.1016/j.neuroimage.2004.09.028>.
- Narayanan, A., White, C. A., Saklayen, S., Scaduto, M. J., Carpenter, A. L., Abduljalil, A., . . . Beyersdorf, D. Q. (2010). Effect of propranolol on functional connectivity in autism Spectrum disorder – A pilot study. *Brain Imaging and Behavior*, 4(2), 189–197. <https://doi.org/10.1007/s11682-010-9098-8>.
- Ratey, J. J., Bemporad, J., Sorgi, P., Bick, P., Polakoff, S., O'Driscoll, G., & Mikkelsen, E. (1987). Brief report: Open trial effects of beta-blockers on speech and social behaviors in 8 autistic adults. *Journal of Autism and Developmental Disorders*, 17(3), 439–446. <https://doi.org/10.1007/bf01487073>.
- Rosen, R. C., Kostis, J. B., & Jekelis, A. W. (1988). Beta-blocker effects on sexual function in normal males. *Archives of Sexual Behavior*, 17(3), 241–255. <https://doi.org/10.1007/bf01541742>.
- Sagar-Ouriaghli, I., Lievesley, K., & Santosh, P. J. (2018). Propranolol for treating emotional, behavioural, autonomic dysregulation in children and adolescents with autism spectrum disorders. *Journal of Psychopharmacology*, 32(6), 641–653. <https://doi.org/10.1177/0269881118756245>.
- Schmidt, G. L., Kimel, L. K., Winterrowd, E., Pennington, B. F., Hepburn, S. L., & Rojas, D. C. (2008). Impairments in phonological processing and nonverbal intellectual function in parents of children with autism. *Journal of Clinical and Experimental Neuropsychology*, 30(5), 557–567. <https://doi.org/10.1080/13803390701551225>.
- Zamzow, R. M., Ferguson, B. J., Stichter, J. P., Porges, E. C., Ragsdale, A. S., Lewis, M. L., & Beyersdorf, D. Q. (2016). Effects of propranolol on conversational reciprocity in autism spectrum disorder: A pilot, double-blind, single-dose psychopharmacological challenge study. *Psychopharmacology*, 233(7), 1171–1178. <https://doi.org/10.1007/s00213-015-4199-0>.

Beta-Alanyl-L-Histidine

- Carnosine
-

Bettelheim, Bruno

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Short Biography

A highly controversial figure in the history of autism, Dr. Bettelheim was born in Austria and trained in Art History. His work in history led him to the study of psychology. He became a refugee from the Nazis and moved to the United States.

He eventually moved to Chicago where he became a professor at the University of Chicago (teaching there from 1944 to 1973). He had some psychoanalytic training in Vienna and served, in Chicago, as the Director of the University of Chicago's Sonia Shankman Orthogenic School – a center for treatment of severely disturbed children. He made many claims for successful treatment but did so within the context of claiming that parents were involved in the pathogenesis of autism (a theory now long discredited). His early work on the topic was widely cited, although it is not clear exactly how many children with autism he actually saw. The diagnoses of autism in his patients have also been questioned. His popularization of the concept of the “refrigerator mother” traumatized a generation of parents who were told they were responsible for their child's autism. Questions were raised about possible plagiarism in his scholarly writing and the validity of his work.

References and Reading

- Bettelheim, B. (1950). *Love is not enough: The treatment of emotionally disturbed children*. Glencoe: Free Press.
- Bettelheim, B. (1959). Joey: A mechanical boy. *Scientific American*, 200, 117–126.
- Pollak, R. (1997). *The creation of Dr. B: A biography of Bruno Bettelheim (Hardcover)*. New York: Touchstone.
- Sutton, N. (1996). *Bettelheim: A life and legacy*. New York: Basic Books.

Better Outcomes and Successful Transitions for Autism (BOOST-A) Program, The

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Definition

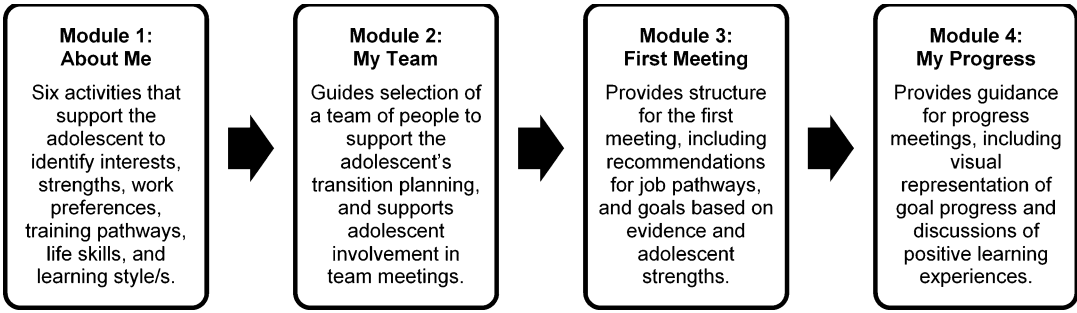
The Better Outcomes & Successful Transitions for Autism (BOOST-A™) is an autism-specific,

web-based program that aims to prepare adolescents on the autism spectrum for the transition from high school to further education, training, or employment. The BOOST-A™ supports adolescents on the autism spectrum to develop career pathways and set goals that enhance their employment-readiness skills. The program consists of four modules, as outlined in Fig. 1.

Module 1, *About Me*, supports career awareness by engaging the adolescent in the following six activities:

- “Interests”: The adolescent completes a questionnaire and is provided with a summary of their key areas of interest related to employment.
- “Strengths”: The adolescent reflects on their areas of strength in the fields of technology, science, physical activities, and arts.
- “Work Preferences”: The adolescent considers the types of work environments they might prefer. They explore factors such as sensory input (e.g., noise and movement), social interactions, and task routines.
- “Training after School”: The adolescent states their preferred post-school training pathway. For example, on-the-job training or an apprenticeship; a vocational training center; or tertiary education at university or college.
- “My skills”: The adolescent explores their current level of performance in activities of daily living, and their participation in community activities.
- “Learning Styles”: The adolescent describes their preferred learning style, which may be one or more of the following: reading, writing, hearing, seeing, or doing.

Module 2, *My Team*, supports the adolescent and their caregiver to identify people who might be best placed to participate in their transition planning, asking them to come together to form a team. In addition, this module provides potential strategies for the adolescent to contribute as actively as possible to the team meetings in a way that they feel comfortable with, as this has been shown to promote increased self-determination (Hendricks and Wehman 2009; Martin and Williams-Diehm 2013).



Better Outcomes and Successful Transitions for Autism (BOOST-A) Program, The, Fig. 1 Description of the four modules of the program

Module 3, *First Meeting*, involves a team meeting that keeps the adolescent at the center of all decision-making. The program provides recommendations for job pathways that leverage the adolescent's strengths. The team decides on a few pathways on which to develop goals. For each goal, the team identifies the actions needed, people responsible, and timeframes for completion. Goals are suggested by the program and encourage the adolescent to gain real-life experiences; e.g., acquiring a part-time job, doing a work experience, finding a mentor, getting more information about potential careers, and developing life skills.

The fourth module, *My Progress*, is completed at subsequent team meetings. The team reflects on the progress toward each goal, and amends and updates the goals and job pathways, as needed. This module encourages the development of resilience through positive reflection, guiding the adolescent to reframe challenging experiences as opportunities for development, rather than failures. Since the program is designed to build self-determination, the adolescent is encouraged to increase their level of involvement at each subsequent meeting. The aim is that the adolescent will eventually lead the meetings and experience a greater sense of control over their plans for the future.

Historical Background

Adolescents on the autism spectrum have numerous strengths; however, many have

difficulty transitioning to post-school employment or higher education (Australian Bureau of Statistics 2012). Furthermore, many people on the autism spectrum are often underemployed or in positions that do not reflect their education level or abilities (Hendricks 2010; Müller et al. 2003). There is increasing recognition of the many strengths people on the autism spectrum bring to the workplace, including low absenteeism, reliability, and excellent recall for information on topics related to their special interests (Mottion 2011; Hillier et al. 2007; Hagner and Cooney 2005). Transition planning enhances post-school outcomes in adolescents with disabilities (King et al. 2005; Wei et al. 2016), but most existing transition planning programs are not autism-specific and may not meet the needs of adolescents on the autism spectrum. Therefore, the current program was developed specifically for adolescents on the autism spectrum to improve transition planning outcomes for this group.

Rationale or Underlying Theory

The program was developed based on three frameworks: self-determination theory, a strengths-based approach, and a technology-based approach. Adolescents with high self-determination are more likely to have post-school employment (Test et al. 2009). The strengths-based approach capitalizes on an individual's expertise and special interests to enhance performance and promote changes in self-determination

(Patten Koenig and Hough Williams 2017). Technology-based interventions are effective in improving outcomes for people on the autism spectrum in areas such as communication, social skills, and emotional recognition (Grynszpan et al. 2014).

Goals and Objectives

Two pilot studies (Pilots A and B) were conducted to determine the feasibility and acceptability of the program (Hatfield et al. 2017b). The effectiveness of the program was then determined in a quasi-randomized controlled trial (RCT) (Hatfield et al. 2016; Hatfield et al. 2017a). Finally, a process evaluation identified the enablers and barriers related to using the program (Hatfield et al. 2016, 2018). The process evaluation involved collecting qualitative and quantitative feedback from the intervention group participants in the RCT.

Treatment Participants

Pilot A consisted of six adolescents on the autism spectrum who trialed the program with their parents and the professionals in their team. Pilot B obtained the feedback from 88 allied health professionals via an online survey. Participants in the RCT and process evaluation were 94 adolescents on the autism spectrum from Australia. The intervention group participants ($n = 49$) received the program and the control group ($n = 45$) participated in usual transition planning practices at their school.

Treatment Procedures

Participants were screened for eligibility and allocated to groups using an alternate allocation method. Outcome measures were completed on enrolment and then 12 months post-intervention, to allow the participants to complete all modules of the program and make progress on their transition planning goals. Outcomes

included self-determination, career planning and exploration, quality of life, environmental support, and domain-specific self-determination. Data were collected from parents and adolescents. Curtin University Human Research Ethics Committee and relevant school sectors provided ethics approval. All participants aged 18+ years provided informed consent and adolescents provided informed assent. For the RCT, normality of the data was determined using the Kolmogorov-Smirnov test. Effectiveness was determined using the independent samples t-test and/or Mann-Whitney U test. An intention-to-treat approach was used, along with the last observation carried forward method.

Efficacy Information

Results from the pilot studies indicated that the program was an acceptable and feasible program (Hatfield et al. 2017b). Modifications to the program were made based on the feedback from participants. Changes included the conversion to a web-based program to improve the usability of the program; a reduction in the content length of the modules; and enhanced use of visuals, such as videos and graphics.

The RCT results indicated significant differences in favor of the intervention group in three areas: (i) opportunity for self-determination at home (parent report); (ii) career exploration (parent and adolescent report); and (iii) transition-specific self-determination (parent report) (Hatfield et al. 2017a). There were no significant differences between groups for the summary scores of the remaining outcomes.

Results from the process evaluation indicated that the program enabled adolescents on the autism spectrum to feel empowered because of the strengths-focus of the program (Hatfield et al. 2018). The program supported parents and adolescents to overcome inertia and take action by providing a structured process to follow, and providing new insights into potential career pathways. Participants were less likely to report benefits from using the program when they did not have a “champion” in their team. The

champion was typically a parent or professional taking charge and moving the transition planning process forward.

Outcome Measurement

The primary outcome of the RCT was self-determination, measured by the AIR Self-Determination Scale (AIR). The four secondary outcomes included: (i) Career planning and exploration, measured by the Career Development Inventory – Australia – Short Form (CDI-A); (ii) Quality of life, measured by the Personal Wellbeing Index-School Children (PWI-SC); (iii) Environment support, measured by the Learning Climate Questionnaire (LCQ); and (iv) Domain specific self-determination, measured by the Transition Planning Objectives Scale.

Qualifications of Treatment Providers

The program is appropriate for use by trusted adults who support the adolescent in transition planning including parents, educators, and allied health professionals, such as occupational therapists, psychologists, and speech pathologists. No formal training, certification, or level of qualification is required.

See Also

- [Education](#)
- [Functional Life Skills](#)
- [Peer Mentors for Students with ASD on College Campuses](#)
- [Preemployment Skills for People with ASD](#)
- [Quality of Life for Transition-Age Youth with ASD](#)
- [Stepped Transition in Education Program for Students with ASD \(STEPS\)](#)
- [Team Approach](#)

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References and Reading

- Australian Bureau of Statistics. (2012). *Autism in Australia* (cat no. 4428.0). Canberra: Australian Bureau of Statistics. Viewed 5 June 2015. Retrieved from <http://www.abs.gov.au/ausstats/abs@.nsf/mf/4428.0>
- Grynszpan, O., Weiss, P., Perez-Diaz, F., & Gal, E. (2014). Innovative technology-based interventions for autism spectrum disorders: A meta-analysis. *Autism, 18*(4), 346–361. <https://doi.org/10.1177/1362361313476767>.
- Hagner, D., & Cooney, B. (2005). “I do that for everybody”: Supervising employees with autism. *Focus on Autism and Other Developmental Disabilities, 20*, 91–97. <https://doi.org/10.1177/10883576050200020501>.
- Hatfield, M., Falkmer, M., Falkmer, T., & Ciccarelli, M. (2016). Evaluation of the effectiveness of an online transition planning program for adolescents on the autism spectrum: Trial protocol. *Child and Adolescent Psychiatry and Mental Health, 10*(48), 1–11. <https://doi.org/10.1186/s13034-016-0137-0>.
- Hatfield, M., Falkmer, M., Falkmer, T., & Ciccarelli, M. (2017a). Effectiveness of the BOOST-A online transition planning program for adolescents on the autism spectrum: A quasi-randomised controlled trial. *Child and Adolescent Psychiatry and Mental Health, 11*(54), 1–12. <https://doi.org/10.1186/s13034-017-0191-2>.
- Hatfield, M., Murray, N., Ciccarelli, M., Falkmer, T., & Falkmer, M. (2017b). Pilot of the BOOST-A: An online transition planning program for adolescents with autism. *Australian Occupational Therapy Journal, 64*(6), 448–456. <https://doi.org/10.1111/1440-1630.12410>.
- Hatfield, M., Falkmer, M., Falkmer, T., & Ciccarelli, M. (2018). Process evaluation of the BOOST-A transition planning program for adolescents on the autism spectrum: A strengths-based approach. *Journal of Autism and Developmental Disorders, 48*(2), 377–388. <https://doi.org/10.1007/s10803-017-3317-8>.
- Hendricks, D. (2010). Employment and adults with autism spectrum disorders: Challenges and strategies for success. *Journal of Vocational Rehabilitation, 32*(2), 125–134. <https://doi.org/10.3233/JVR-2010-0502>.
- Hendricks, D., & Wehman, P. (2009). Transition from school to adulthood for youth with autism spectrum disorders: Review and recommendations. *Focus on Autism and Other Developmental Disabilities, 24*, 77–88. <https://doi.org/10.1177/1088357608329827>.
- Hillier, A., Campbell, H., Mastriani, K., Izzo, M., Kool-Tucker, A., Cherry, L., & Beversdorf, D. (2007).

- Two-year evaluation of a vocational support program for adults on the autism spectrum. *Career Development and Transition for Exceptional Individuals*, 30(1), 35–47. <https://doi.org/10.1177/08857288070300010501>.
- King, G., Baldwin, P., Currie, M., & Evans, J. (2005). Planning successful transitions from school to adult roles for youth with disabilities. *Children's Health Care*, 34, 195–216. https://doi.org/10.1207/s15326888chc3403_3.
- Martin, J., & Williams-Diehm, K. (2013). Student engagement and leadership of the transition planning process. *Career Development and Transition for Exceptional Individuals*, 36, 43–50. <https://doi.org/10.1177/2165143413476545>.
- Mottron, L. (2011). Changing perceptions: The power of autism. *Nature*, 479(7371), 33–35. <https://doi.org/10.1038/479033a>.
- Müller, E., Schuler, A., Burton, B., & Yates, G. (2003). Meeting the vocational support needs of individuals with Asperger syndrome and other autism spectrum disabilities. *Journal of Vocational Rehabilitation*, 18(3), 163–175. <http://content.iospress.com.dbgw.lis.curtin.edu.au/articles/journal-of-vocational-rehabilitation/jvr00193>.
- Patten Koenig, K., & Hough Williams, L. (2017). Characterization and utilization of preferred interests: A survey of adults on the autism spectrum. *Occupational Therapy in Mental Health, Early view*, 1–12. <https://doi.org/10.1080/0164212X.2016.1248877>.
- Test, D., Mazzotti, V., Mustian, A., Fowler, C., Kortering, L., & Kohler, P. (2009). Evidence-based secondary transition predictors for improving post-school outcomes for students with disabilities. *Career Development for Exceptional Individuals*, 32, 160–181. <https://doi.org/10.1177/0885728809346960>.
- Wei, X., Wagner, M., Hudson, L., Yu, J., & Javitz, H. (2016). The effect of transition planning participation and goal-setting on college enrollment among youth with autism spectrum disorders. *Remedial and Special Education*, 37(1), 3–14. <https://doi.org/10.1177/0741932515581495>.

Bias in Assessment Instruments for Autism

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Introduction

Standardized diagnostic tools are critical in terms of assessing Autism Spectrum Disorder (ASD). There is an increasing interest in how culture, sex, social economic status, and other factors affect the performance of concurrent gold standard instruments for the purpose of identifying autism spectrum disorder (ASD). While the performance of, for example, the Autism Diagnostic Observation Schedule (ADOS) (Rutter et al. 2012) and the Autism Diagnostic Interview (ADI-R) (Rutter et al. 2003) performs well in identifying individuals suspected of ASD, and especially when used to complement each other, little is known on the performance of the ADOS and the ADI-R in different cultures, ethnicities, and between sexes. Most of the instruments created for the purpose of identifying ASD are created and validated in the USA and Europe. However, they are often translated into a large range of languages for use in different cultures and ethnicities, without conducting validation studies of the instruments. As a result, it could be discussed how culture, ethnicity, sex, and the translation itself affect test performance of instruments. Even considering how instruments work between sexes are rarely examined in validation studies of instruments. In terms of parent report, it is likely to believe that culture could affect how parents regard various elements of behavior and development, and how

BG-II

► Bender Visual-Motor Gestalt Test II

Bias

► Measurement Error

parents rate their boy or girl in terms of endorsement of, for example, behaviors or development.

Current Knowledge

While we do believe that some of the issues raised could potentially bias diagnostic instruments, little has been reported on ASD-specific diagnostic instruments in terms of potential biases and test performance (Volkmar et al. 2014). A recent study by Vanegas et al. (2016) revealed that the sensitivity and specificity of the ADI-R were moderate, but lower than previously reported values. However, currently we have little information on test performance from different cultures, ethnicities, taking sex also into consideration. Behavioral, developmental and temperamental differences could ultimately affect the performance of screening and diagnostic instruments (Dworzynski et al. 2012; Macari et al. 2017; Øien et al. 2017). In terms of culture, research has revealed cross-cultural prevalence differences (Elsabbagh et al. 2012), and culture is regarded to have a great impact on how ASD is perceived, diagnosed, and treated across cultures (Volkmar et al. 2014). In the western world, early diagnosis is regarded as paramount for early intervention and access to services. It is not sure to what extent, but a diagnosis of ASD could in other cultures impair the access to treatment and services. A great example of how ASD symptomatology could be affected by culture is found in eye contact. As impairment or atypicalities in eye contact is regarded as a core symptom of ASD, avoiding eye contact in some eastern cultures are regarded as appropriate and polite (Volkmar et al. 2014). Non-autism-specific research, among the Sami population of Norway (ethnic minority), revealed that disorders such as ASD and other mental health disorders were not prevalent as in the larger non-ethnic majority of Norway (Nergård 2006).

Future Directions

As there is a pressing need to understand how culture, ethnicity, sex, and other factors affect test performance of a range of different diagnostic

instruments, it is necessary to focus on implementation of validation studies when diagnostic instruments are translated. While translating and back translating is regarded as necessary, translations of instruments can still incorporate wording or translations that might not be suitable for the given culture, ethnicity, or language. Future studies should also examine the performance in sex separately, aiming at exploring if there are different sensitivity and specificity for males and females, respectively. One solution for future research should be to conduct meta-analyses of test performance of the various diagnostic instruments across cultures, ethnicities, and social economic statuses, taking sex into consideration. This could show if the diagnostic instruments perform differently in different conditions, and it could also indicate in which areas the discrepancies arise from. Increasing knowledge on how to identify ASD in third world countries and cultures is of highest importance, and current diagnostic instruments are often too expensive for many cultures and countries. Focusing on the development of community-based identification of ASD in areas with limited resources and utilizing less comprehensive, but culture-sensitive instruments could potentially decrease differences in prevalence and differences across cultures.

References and Reading

- Dworzynski, K., Ronald, A., Bolton, P., & Happé, F. (2012). How different are girls and boys above and below the diagnostic threshold for autism spectrum disorders? *Journal of the American Academy of Child and Adolescent Psychiatry*, 51, 788.
- Elsabbagh, M., Divan, G., Koh, Y.-J., Kim, Y. S., Kauchali, S., Marcin, C., et al. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research*, 5(3), 160–179. <https://doi.org/10.1002/aur.239>
- Macari, S. L., Koller, J., Campbell, D. J., & Chawarska, K. (2017). Temperamental markers in toddlers with autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 58(7), 819–828. <https://doi.org/10.1111/jcpp.12710>
- Nergård, J.-I. (2006). *Den levende erfaring*. Oslo: Cappelen.
- Øien, R. A., Hart, L., Schjølberg, S., Wall, C. A., Kim, E. S., Nordahl-Hansen, A., et al. (2017). Parent-endorsed sex differences in toddlers with and without

- ASD: Utilizing the M-CHAT. *Journal of Autism and Developmental Disorders*, 47(1), 126–134. <https://doi.org/10.1007/s10803-016-2945-8>
- Rutter, M., Le Couteur, A., & Lord, C. (2003). *Autism diagnostic interview-revised* (Vol. 29, p. 30). Los Angeles: Western Psychological Services.
- Rutter, M., DiLavore, P. C., Risi, S., Gotham, K., & Bishop, S. (2012). *Autism diagnostic observation schedule, (ADOS-2)*. Torrance: Western Psychological.
- Vanegas, S. B., Magaña, S., Morales, M., & McNamara, E. (2016). Clinical validity of the ADI-R in a US-based Latino population. *Journal of Autism and Developmental Disorders*, 46(5), 1623–1635. <https://doi.org/10.1007/s10803-015-2690-4>
- Volkmar, F. R., Paul, R., Rogers, S. J., & Pelphrey, K. A. (2014). *Handbook of autism and pervasive developmental disorders*. Hoboken: Wiley.

BIG-2

► CNTN4: Contactin 4

Bilingualism and Language Development in Children with Autism Spectrum Disorders

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Definition

Bilingually exposed children with autism spectrum disorders (ASD) are those who are exposed to two languages from early ages. These children are typically being raised in bilingual families and/or bilingual communities. Bilingualism is a multidimensional characteristic that is influenced by multiple factors such as sociolinguistic context (e.g., majority or minority language), age of acquisition, and amount of exposure or usage (for children) or proficiency (for older children and adults), among others (Surrain and Luk 2017). Additionally, socioeconomic differences

are linked to bilingual status in some contexts. These factors need to be kept in mind when interpreting and comparing research findings (Kay-Raining Bird et al. 2016).

Historical Background

Parents of children with ASD are commonly advised to use only one language when interacting with their children. This often stems from beliefs that bilingualism might be too challenging or confusing for the child and hinder his/her language development (Yu 2013). However, evidence does not support this claim (for reviews see Drysdale et al. 2015; Lund et al. 2017). Research with children under age 6 has repeatedly shown no additional language delays caused by bilingual exposure. Importantly, in the long term, bilingualism can provide social and vocational advantages. For children from bilingual families and communities, it also provides opportunities to maintain familial bonds (Yu 2013) and rich social and linguistic input (Hudry et al. 2018). In situations of highly proficient bilingualism, it may even provide advantages in some executive function skills (Gonzalez-Barrero and Nadig 2017, 2019b; Nadig and Gonzalez-Barrero 2019).

Current Knowledge

Bilingual Language Development in Toddlers and Preschoolers

Research with toddlers and preschool bilingual children with ASD has shown that these children reach language milestones, such as first words and onset of first sentences, at a similar age relative to their monolingual peers with ASD (Hambly and Fombonne 2012; Ohashi et al. 2012; Valicenti-McDermott et al. 2013). Furthermore, their early vocabulary and communication skills develop at a similar rate to that of children with ASD exposed to only one language (Dai et al. 2018; Ohashi et al. 2012; Petersen et al. 2012; Reetzke et al. 2015). Most studies on bilingualism and ASD including children of this age have relied primarily on parent report, which is a valid measure to use early in

development (e.g., Hambly and Fombonne 2012; Reetzke et al. 2015). However, there is a lack of studies examining the language development of school-age bilingual children with ASD. It is important to extend investigations to this older age group as more sophisticated language skills are developed during the school years when language is used as a tool for learning.

Bilingual Language Development at School Age

Only a few studies have directly examined the language skills of school-age bilingual children with ASD (Gonzalez-Barrero and Nadig 2018, 2019a; Meir and Novogrodsky 2019). Importantly, unlike early development, at school age it is well established that typically developing monolinguals outperform bilinguals on standardized language tests administered in one language (Bialystok et al. 2010), which is linked to bilinguals' language exposure being split between languages (Thordardottir 2011).

How do bilingual children with autism, without intellectual disability, fare at school age? Gonzalez-Barrero and Nadig (2019a) assessed the vocabulary and grammatical skills of 13 bilingual and 13 monolingual school-aged children with ASD (age range 5–10 years). Children were recruited in Montreal, Quebec, Canada, a multicultural city where the use of French and English is a common practice and both languages are official languages of the country. Children were speakers of French, English, Spanish, or the combination of two of these languages. To qualify as bilingual in this study, children had to meet a rigorous three-step criterion including amount of exposure above 20% to two languages, proficiency judgments from parents, as well as completion of tasks in both languages. Bilinguals and monolinguals did not differ with respect to chronological age, nonverbal IQ, maternal education, dominant language, or autism symptoms, and a similar percentage of children in each group had language impairment. Standardized tests of vocabulary and grammar were administered to compare the performance of the bilingual children with ASD relative to their monolingual peers with ASD. Results concerning receptive vocabulary

skills in the bilingual children's dominant language showed a trend where monolingual children exhibited higher scores relative to bilinguals. Yet, most of the bilingual children with ASD performed within the average range of the test (within 1 SD above or below the test mean). Results from the expressive grammatical test mirrored those found for vocabulary skills; there were no significant differences between the bilinguals' dominant language scores and those of the monolingual group, although there was a tendency in the bilingual group to score below the average range on this measure. These findings demonstrate the same patterns found in typically developing bilinguals (Bialystok et al. 2010) and highlight the key role that amount of language exposure plays in child language abilities, in autism (Gonzalez-Barrero and Nadig 2018) as in typical development.

Hoang et al. (2018) elicited short narratives in a picture sequencing task from a subsample with similar characteristics to the participants just described ($n = 20$, including 5 bilinguals and 5 monolinguals with ASD). As reported in Gonzalez-Barrero and Nadig (2019a), monolinguals had higher receptive vocabulary scores than bilinguals. Yet, when using language functionally to tell a narrative, bilinguals produced significantly more utterances than monolinguals. With respect to narrative skills, bilinguals and monolinguals did not differ in macrostructure (e.g., sequencing of events and coherence), microstructure (e.g., use of referential terms and conjunctions), or elaborations (e.g., sound effects, character speech). Baldimtsi et al. (2016) used a similar study design to examine narrative production in Greek-speaking bilinguals and monolinguals with and without autism and found that bilingual children with ASD outperformed monolinguals with ASD in some narrative skills. Though preliminary given very small sample sizes, these converging findings suggest that lower standardized language test scores in bilingual children with autism do not reflect a reduced ability to use that language functionally.

This body of work provides information to clinicians working with this population as it demonstrates that many children with ASD can

become bilingual when adequate language exposure is provided (Gonzalez-Barrero and Nadig 2018). Additionally, in contrast to language development during the early years, it is expected that at school age bilinguals will underperform relative to their monolingual peers on standardized language tests, which calls for caution when assessing bilingual children with ASD using monolingual norms (Thordardottir 2015).

Future Directions

With the increasing rate of bilingualism around the world (Surrain and Luk 2017), more research is needed to better understand and describe the language development trajectories of children with ASD being raised in bilingual families and societies. Although there is a growing interest in bilingualism and autism, and some studies have recently been conducted examining the narrative and syntactic abilities in this population (e.g., Hoang et al. 2018; Meir and Novogrodsky 2019), more studies are needed in language domains such as phonology and pragmatics, as well as comparing different contexts of bilingualism.

A review indicated that many practitioners would like to support bilingualism in children with developmental disabilities when relevant for families but that the lack of bilingual specialized services and educational opportunities is a major obstacle (Marinova-Todd et al. 2016). Baker et al. (2018) offer a potential solution through dual immersion educational programs for children with autism, and with respect to how a child's heritage language can be incorporated in therapy, see ► [“Heritage Language Use for Intervention in Autism”](#). The development of bilingual services and research evaluating them is a critical area for future work.

In sum, the available evidence suggests that bilingualism is a possible outcome for children with ASD and that bilingually exposed children with ASD show globally similar language development trajectories to their monolingual peers with ASD. Thus, parents of bilingual children should be encouraged to use the language in

which they can better interact with their child, which could facilitate the delivery of parent-implemented interventions (Lim et al. 2019) and allow parents to provide quality input that supports language development.

See Also

- [Communicative Acquisition in ASD](#)
- [Heritage Language Use for Intervention in Autism](#)
- [Theories of Language Development](#)

References and Reading

- Baker, D., Roberson, A., & Kim, H. (2018). Autism and dual immersion: Sorting through the questions. *Advances in Autism*, 4, 174–183.
- Baldimtsi, E., Peristeri, E., Tsimpli, I. M., & Nicolopoulou, A. (2016). Bilingual children with high functioning autism spectrum disorder: Evidence from oral narratives and non-verbal executive function tasks. In J. Scott & D. Waughtal (Eds.), *Proceedings of the 40th Annual Boston University Conference on Language Development* (pp. 18–31). Somerville: Cascadia Press.
- Bialystok, E., Luk, G., Peets, K. F., & Yang, S. (2010). Receptive vocabulary differences in monolingual and bilingual children. *Bilingualism: Language and Cognition*, 13(4), 525–531.
- Bird, E. K. R., Genesee, F., & Verhoeven, L. (2016). Bilingualism in children with developmental disorders: A narrative review. *Journal of Communication Disorders*, 63, 1–14.
- Dai, Y. G., Burke, J. D., Naigles, L., Eigsti, I. M., & Fein, D. A. (2018). Language abilities in monolingual-and bilingual-exposed children with autism or other developmental disorders. *Research in Autism Spectrum Disorders*, 55, 38–49.
- Drysdale, H., van der Meer, L., & Kagohara, D. (2015). Children with autism spectrum disorder from bilingual families: A systematic review. *Review Journal of Autism and Developmental Disorders*, 2, 26–38.
- Gonzalez-Barrero, A. M., & Nadig, A. (2017). Verbal fluency in bilingual children with autism Spectrum disorders. *Linguistic Approaches to Bilingualism*, 7, 460–475.
- Gonzalez-Barrero, A. M., & Nadig, A. (2018). Bilingual children with autism spectrum disorders: The impact of amount of language exposure on vocabulary and morphological skills at school age. *Autism Research*, 11, 1667–1678.
- Gonzalez-Barrero, A. M., & Nadig, A. (2019a). Brief report: Vocabulary and grammatical skills of bilingual children with autism spectrum disorders at school age.

- Journal of Autism and Developmental Disorders*, 49, 3888–3897.
- Gonzalez-Barrero, A. M., & Nadig, A. S. (2019b). Can bilingualism mitigate set-shifting difficulties in children with autism spectrum disorders? *Child Development*, 90, 1043–1060.
- Hambly, C., & Fombonne, E. (2012). The impact of bilingual environments on language development in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42, 1342–1352.
- Hoang, H., Gonzalez-Barrero, A. M., & Nadig, A. (2018). Narrative skills of bilingual children with autism spectrum disorder. *Discours. Revue de linguistique, psycholinguistique et informatique. A Journal of Linguistics, Psycholinguistics and Computational Linguistics*, (23), 1–33.
- Hudry, K., Rumney, L., Pitt, N., Barbaro, J., & Vivanti, G. (2018). Interaction behaviors of bilingual parents with their young children with autism spectrum disorder. *Journal of Clinical Child & Adolescent Psychology*, 47(sup1), S321–S328.
- Lim, N., O'Reilly, M. F., Sigafos, J., Ledbetter-Cho, K., & Lancioni, G. E. (2019). Should heritage languages be incorporated into interventions for bilingual individuals with neurodevelopmental disorders? A systematic review. *Journal of Autism and Developmental Disorders*, 49, 887–912.
- Lund, E. M., Kohlmeier, T. L., & Durán, L. K. (2017). Comparative language development in bilingual and monolingual children with autism spectrum disorder: A systematic review. *Journal of Early Intervention*, 39, 106–124.
- Marinova-Todd, S. H., Colozzo, P., Mirenda, P., Stahl, H., Bird, E. K. R., Parkinson, K., ... & Genesee, F. (2016). Professional practices and opinions about services available to bilingual children with developmental disabilities: An international study. *Journal of Communication Disorders*, 63, 47–62.
- Meir, N., & Novogrodsky, R. (2019). Syntactic abilities and verbal memory in monolingual and bilingual children with high functioning autism (HFA). *First Language*. <https://doi.org/10.1177/0142723719849981>.
- Nadig, A. S., & Gonzalez-Barrero, A. M. (2019). Proficient bilingualism may alleviate some executive function difficulties in children with autism spectrum disorders. In L. Spradlin, I. Sekerina, & V. Valian (Eds.), *Bilingualism, executive function, and beyond. Questions and insights* (pp. 337–353). Amsterdam: John Benjamins.
- Ohashi, J. K., Mirenda, P., Marinova-Todd, S., Hambly, C., Fombonne, E., Szatmari, P., ... & Volden, J. (2012). Comparing early language development in monolingual-and bilingual-exposed young children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6, 890–897.
- Petersen, J. M., Marinova-Todd, S. H., & Mirenda, P. (2012). Brief report: An exploratory study of lexical skills in bilingual children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 42, 1499–1503.
- Reetzke, R., Zou, X., Sheng, L., & Katsos, N. (2015). Communicative development in bilingually exposed Chinese children with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, 58, 813–825.
- Surraín, S., & Luk, G. (2017). Describing bilinguals: A systematic review of labels and descriptions used in the literature between 2005–2015. *Bilingualism: Language and Cognition*, 22, 401–415.
- Thordardottir, E. (2011). The relationship between bilingual exposure and vocabulary development. *International Journal of Bilingualism*, 15(4), 426–445.
- Thordardottir, E. (2015). Proposed diagnostic procedures for use in bilingual and cross-linguistic contexts. In S. Armon-Lotem, J. de Jong, & N. Meir (Eds.), *Assessing multilingual children: Disentangling bilingualism from language impairment* (pp. 331–358). Bristol: Multilingual Matters.
- Valicenti-McDermott, M., Tarshis, N., Schouls, M., Galdston, M., Hottinger, K., Seijo, R., Shulman, L., & Shinnar, S. (2013). Language differences between monolingual English and bilingual English-Spanish young children with autism spectrum disorders. *Journal of Child Neurology*, 28, 945–948.
- Yu, B. (2013). Issues in bilingualism and heritage language maintenance: Perspectives of minority-language mothers of children with autism spectrum disorders. *American Journal of Speech-Language Pathology*, 22, 10–24.

Biological Motion

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Synonyms

Human or animal motion

Definition

Biological motion refers to the movements of humans or animals including eye, face, and full body motion. Typical observers exhibit robust sensitivity to biological motion cues provided by other people. However, disrupted sensitivity to biological motion, at the behavioral and neural level, is emerging as a hallmark of autism

spectrum disorders (ASD). The lack of tuning to such socially relevant information may reflect some of the pathognomic social deficits associated with ASD.

References and Reading

- Annaz, D., Cambell, R., Coleman, M., Milne, E., & Swettenham, J. (2011). Young children with autism spectrum disorder do not preferentially attend to biological motion. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-011-1256-3>.
- Blake, R., & Shiffrar, M. (2007). Perception of human motion. *Annual Review of Psychology*, 58, 47–73.
- Kaiser, M. D., & Shiffrar, M. (2009). The visual perception of motion by observers with autism spectrum disorder: A review and synthesis. *Psychonomic Bulletin & Review*, 16(5), 761–777.
- Kaiser, M. D., Hudac, C. M., Shultz, S., Lee, S.-M., Cheung, C., Berkena, A. M., et al. (2010). Neural signatures of autism. *Proceedings of the National Academy of Sciences*, 107(49), 21223–21228.
- Klin, A., Lin, D., Gorrindo, P., Ramsay, G., & Jones, W. (2009). Two-year-olds with autism orient to non-social contingencies rather than biological motion. *Nature*, 459, 257–261.

Biomarker Research in Autism Spectrum Disorder

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Definition

A biological marker (biomarker) is defined as a characteristic that is objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or pharmacologic responses to a therapeutic intervention (Biomarkers Definitions Working 2001). Although the term biomarker is often associated with biological processes, many data modalities can be used to objectively measure relevant processes, such as genes, metabolites, brain structure and function, and overt behaviors. In fields like oncology, biomarkers have been categorized into

meaningful subtypes, and similar efforts to classify biomarkers in neuropsychiatry are in progress (Davis et al. 2015; McPartland 2016). Through meaningful subtypes, biomarkers can serve a multitude of purposes in autism spectrum disorder (ASD) research and treatment.

Diagnostic biomarkers are discrete, and objective measures of whether or not an individual has ASD and their identification have been a long-standing objective in the field. Diagnostic biomarkers should not overlap with other conditions, reflected in the sensitivity and specificity of biomarkers (Davis et al. 2015).

Screening biomarkers measure diagnostic risk status and potentially measure processes prior to observable behavioral symptoms. Screening biomarkers would allow for intensive early intervention for individuals which have been shown to improve the prognosis of ASD (Dawson et al. 2010; Estes et al. 2015; Lovaas 1987; Smith et al. 2000).

Stratification biomarkers determine meaningful subgroups of individuals to predict or evaluate treatment (Loth et al. 2016a). Stratification biomarkers, for example, may indicate a group of children with ASD who may be likely to respond to a specific treatment.

Early efficacy biomarkers indicate whether a treatment is altering targeted symptoms or the processes underlying those symptoms (McPartland 2016). In autism, an early efficacy biomarker may reveal symptomology changes from a treatment before they are observable in clinical observation or caregiver report, the current measures used in clinical trials of ASD.

Target engagement biomarkers measure whether an intervention is affecting the intended process (Zhao et al. 2015). For example, in autism, a target engagement biomarker might assist in determining whether a specific medication is affecting neural activity in the intended brain region.

These biomarker subtypes strive to improve the understanding of autism and foster precision-medicine approaches in the diagnosis and treatment of autism (Varcin and Nelson 2016). Determining which biomarker subtype is under research will guide study designs and impact future applicability. Nevertheless, before the

field can reliably utilize biomarkers, key challenges in the identification of biomarkers must be addressed in future study designs.

Historical Background

Biomarker identification and validation in ASD, a condition characterized by two domains of core symptoms, including impairments in social communication and presence of restricted and repetitive behaviors (American Psychiatric Association 2013), hold promise in subtyping the heterogeneous neurodevelopmental disorder and guiding personalized treatments. In other biomedical fields, such as oncology, biomarkers have been successfully implemented to inform personalized treatments as well as measure processes involved in the diagnosis, prognosis, and prevention of various cancers (Nalejska et al. 2014). For example, treatments for different forms of cancer are based on specific genetic mutations (Kalia 2015).

Despite significant biomarker discovery efforts in ASD, biomarkers are not yet readily applied in the treatment and diagnosis of ASD. Clinical judgment of observable behaviors remains the primary clinical tool applied in ASD. The diagnostic criteria, as outlined in the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition, consist of a checklist of behaviors in which a tally of symptoms across domains constitutes as a diagnosis (American Psychiatric Association 2013). The strongest and most reliable diagnostic tools include a parent interview (Lord et al. 1994) and play-based observational assessment (Lord et al. 2012). Furthermore, clinicians determine the best course of treatment and prognoses based on the results of these standardized behavioral assessments and subjective evaluation. Outcomes, from the individual level to multisite clinical trials, are often reported by parent and teacher report or behavioral observation. The applications of these methods have significantly advanced autism, used interchangeably with ASD, research, and these clinical tools can be administered reliably. Nevertheless, the nature of these methods remains remarkably similar to the methods used by Asperger (1944) and Kanner (1943) decades ago.

Biomarker discovery for autism remains in its infancy. The first steps in identifying potential biomarkers are operationalizing the definition for the term “biomarker” and describing various subtypes. Challenges to biomarker research must be highlighted so that future research endeavors can determine the best course of action to address them in study designs. Currently, national and international studies are underway and address many of these challenges to biomarker research, and studies with large cohorts show promising signs of biomarker discovery in ASD. Future studies must consider the objectivity, sensitivity, and scalability of the methodologies used to measure biomarkers to ensure a large public health impact.

Current Knowledge

Specificity of Biomarkers

It is well-established that autism is an extremely heterogeneous disorder containing a large variation in core diagnostic features and associated characteristics, such as cognitive and language ability. Therefore, the search for biomarkers of ASD may prove elusive for potential underlying etiologies. Instead, a more effective approach may be to research potential biomarkers in relation to specific functional processes or symptom indices, as outlined by the Research Domain Criteria approach (<http://www.nimh.nih.gov/research-priorities/rdoc/>), rather than in relation to diagnostic status. Many features present in autism are found in other disorders; for example, repetitive and restricted behaviors are present in obsessive compulsive disorder, and social dysfunction is present in schizophrenia spectrum disorders. Often, treatment goals are centered around variation in a specific area as opposed to diagnostic status more broadly. Biomarkers that measure changes in specific functional domains may prove extremely useful in these cases as opposed to biomarkers measuring diagnostic status.

Individual Versus Composite Biomarkers

Generally, biomarker studies in ASD and other neurodevelopmental disorders focus on utilizing

a specific measure as a biomarker. For example, a study may focus on activation in a specific brain region associated with social behavior. Yet, the complexity of biological processes that give rise to social behavior, as well as the potential for compensatory mechanisms within these processes, and the well-known heterogeneity in autism complicate the potential utility of a single biomarker. It may be necessary to develop biomarkers reflecting multiple measures. An individual's profile as measured through a composite from multiple biomarkers, either within one data modality or across data modalities, may provide more relevant information for prognoses and treatment compared to a single biomarker.

Developmental Considerations

Autism is a developmental disorder wherein early-life symptoms influence experience as well as experience-expectant biological processes (Dawson et al. 2005). Different biological systems may have varying developmental trajectories. Therefore, in individuals with ASD, the patterns of brain activity represent an interplay between early-occurring neural atypicalities and subsequent developmental sequelae. Consequently, biomarker studies conducted at different developmental points may result in disparate findings. A key factor in biomarker discovery in autism will be understanding biomarkers across development. To address this factor, biomarker research must be conducted with sufficiently large samples to analyze developmental effects or performed in tightly developmentally constrained studies.

Differences At the Group and Individual Level

In autism research, and neurodevelopmental disorders more generally, studies report findings as differences in means between groups of individuals. These findings often reflect a shift in the distribution of values between the groups on a biomarker parameter. Thus, there is little information about the biomarker at the individual level unless the value of the biomarker is true for every individual with ASD. Advancing translational goals of biomarkers requires designing studies to evaluate effects at the individual level or individual variation on specific characteristics. Through

these study designs, biomarkers may, at the individual level, provide relevant information.

Current Efforts in Biomarker Discovery

While significant challenges to biomarker discovery in ASD are present, many are addressed through studies developed by collaborative consortia. Researchers are working together to develop protocols for multiple data modalities to identify biomarkers in autism and disseminate these methodologies. Through large cohorts or longitudinal designs, oftentimes both, these studies address many of the challenges to biomarker research in ASD and offer the first opportunities to review biomarkers in autism with sufficiently large sample sizes.

The largest ongoing effort toward autism biomarker measure development is the European Autism Interventions – A Multicenter Study for Developing New Medications (EU-AIMS) Longitudinal European Autism Project (LEAP). The multisite, multidisciplinary study aims to discover stratification biomarkers for ASD. Throughout the study, participants are characterized through their symptom profile, comorbidities, quality of life, adaptive functioning, neurocognitive profile, brain structure and function, biochemical biomarkers, prenatal environmental risk factors, and genomics (Loth et al. 2016b). For this study, validation of biomarkers will be carried out similar to other biomedical fields in which biomarkers are used for specific clinical practice (Lee et al. 2006). Subgroups will be divided through a priori definitions or through data-driven approaches. The protocols will be shared with international research groups to determine reproducibility (Loth et al. 2017).

The Autism Biomarkers Consortium for Clinical Trials (ABC-CT) is a US-based multisite study working in collaboration with EU-AIMS for similar purposes. The ABC-CT is dedicated to utilizing objective approaches to develop reliable measures of social-communicative behaviors in children with autism (www.asdbiomarkers.org). The longitudinal study collects electrophysiological, eye-tracking, and video-tracking data as well as comprehensive characterization of individuals through parent interviews, behavioral

observation, and parent questionnaires at three points over a 6-month period. Additionally, DNA samples are collected from the participants with autism and their biological parents for future genetic analysis. Ultimately, the study is designed to create an infrastructure which will readily transfer into research and treatment areas, such as clinical trials, with tools that will allow for objective and predictive measurements of how individuals with ASD will respond to treatments.

In contrast to these studies oriented toward biomarkers for use in clinical trials, the Infant Brain Imaging Study (IBIS) Network seeks to develop screening and diagnostic biomarkers. The study collects longitudinal MRI data at ages 6, 12, and 24 months from infants who have an older sibling with autism thus have an increased risk for developing autism themselves (Shen et al. 2017). It has already delivered promising results, as elevated extra-axial cerebrospinal fluid (EA-CSF) at 6 months of age predicted the diagnosis of toddlers at 24 months of age where the infants with most severe autistic behaviors at 24 months had the highest EA-CSF volume. This converged with findings from an earlier study with EA-CSF (Shen et al. 2013). The longitudinal study design deepened the developmental understanding of EA-CSF in typical and atypical development, and the automated segmentation algorithm used has the potential to translate to many settings (Pelphrey 2017).

The consortia address many of the challenges to biomarker identification in autism through their study designs. By establishing multisite studies, biomarker measures can be evaluated with sufficiently large cohorts, developmental effects can be examined through large cohorts and longitudinal design, and the feasibility of translating these measures to a larger scale can be assessed.

Future Directions

The efforts in biomarker research are moving toward earlier identification and precision-based treatments of ASD while simultaneously deepening our understanding of the disorder. The success of proposed biomarkers, however, is dependent

on the translational impact of the technologies used to measure these biomarkers. In order to implement biomarker research goals, the biomarker measures must be objective, sensitive, economical, and scalable. Here, electrophysiology is used as an example addressing the aforementioned criteria.

Applicability

Methods that are useful in a large functional range, such as individuals with intellectual disability, and developmental range, as early as infancy, are well suited for future biomarker research. Electrophysiology requires minimal instructions with a straightforward application. Generally, the participant is only required to tolerate sensors on their skin. Artifacts due to movement during a session recording are specific to trials which can be removed from the data or corrected to preserve the integrity of the recording.

Objectivity

Methodological rigor in defining biomarker parameters when compared to other methodologies like behavioral methods will be critical for future research. Consistent data collection across multiple locations is possible through identical equipment and experimental paradigms without the need for developing clinician reliability.

Sensitivity

Electrophysiological measures, for example, can potentially measure processes relevant to biomarker discovery more sensitively than behavioral methods. These recordings may delineate processes that either may never be present in behavior or not present in behavior yet. Many of the features that characterize the ASD diagnosis are not observable until the second year of life; therefore, methods that sensitively index these processes may elucidate atypical processes before overt behaviors are displayed.

Cost and Scalability

Electrophysiological methods are an extremely cost-effective way to measure biological processes; thus prohibitive cost is not a factor compared to other biological assays. Large-scale

implementation with electrophysiological recordings would readily translate into the current healthcare system since these facilities already exist. Psychophysiological methods offer both low cost and widespread availability necessary to utilize biomarkers on a public health level.

Conclusion

Biomarker research has made considerable strides in the last decade. Long-term investments have been made in the hopes of discovering translatable, practicable biomarkers. As the field of biomarker research moves forward, researchers must ascertain the appropriate biomarkers to pursue and whether it is more advantageous to examine processes dimensionally or examine diagnoses categorically. While there are many challenges to biomarker research in autism, identifying and addressing these challenges will increase the ability to discover applicable biomarkers. Large-scale efforts such as the EU-AIMS LEAP and ABC-CT demonstrate collaborative efforts that should have the power to detect modest effect sizes and promise to disseminate the methodologies used to translate these potential biomarkers to other settings. Due to the current efforts in the field, biomarkers may soon be informing individualized treatment plans and implemented in multisite clinical trials and deepening our understanding of autism.

See Also

- [Diagnosis and Classification](#)
- [Longitudinal Research in Autism](#)
- [Neural Signatures of Treatment Response](#)
- [Studies to Advance Autism Research and Treatment](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5*. Washington, DC: American Psychiatric Association.
- Asperger, H. (1944). Die "Autistischen Psychopathen" im Kindesalter. *Archiv für psychiatrie und Nervenkrankheiten*, 117(1), 76–136.
- Biomarkers Definitions Working Group. (2001). Biomarkers and surrogate endpoints: Preferred definitions and conceptual framework. *Clinical Pharmacology and Therapeutics*, 69(3), 89–95. <https://doi.org/10.1067/mcp.2001.113989>.
- Davis, J., Maes, M., Andreazza, A., McGrath, J. J., Tye, S. J., & Berk, M. (2015). Towards a classification of biomarkers of neuropsychiatric disease: From encompass to compass. *Molecular Psychiatry*, 20(2), 152–153. <https://doi.org/10.1038/mp.2014.139>.
- Dawson, G., Webb, S. J., & McPartland, J. (2005). Understanding the nature of face processing impairment in autism: Insights from behavioral and electrophysiological studies. *Developmental Neuropsychology*, 27(3), 403–424. https://doi.org/10.1207/s15326942dn2703_6.
- Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., . . . Varley, J. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The early start Denver model. *Pediatrics*, 125(1), e17–e23.
- Estes, A., Munson, J., Rogers, S. J., Greenson, J., Winter, J., & Dawson, G. (2015). Long-term outcomes of early intervention in 6-year-old children with autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 54(7), 580–587. <https://doi.org/10.1016/j.jaac.2015.04.005>.
- Kalia, M. (2015). Biomarkers for personalized oncology: Recent advances and future challenges. *Metabolism*, 64(3), S16–S21. <https://doi.org/10.1016/j.metabol.2014.10.027>.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2(3), 217–250.
- Lee, J. W., Devanarayan, V., Barrett, Y. C., Weiner, R., Allinson, J., Fountain, S., . . . Wagner, J. A. (2006). Fit-for-purpose method development and validation for successful biomarker measurement. *Pharmaceutical Research*, 23(2), 312–328. <https://doi.org/10.1007/s11095-005-9045-3>.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(5), 659–685.
- Lord, C., Rutter, M., DiLavore, P., Risi, S., Gotham, K., & Bishop, S. (2012). *Autism diagnostic observation schedule—2nd edition (ADOS-2)*. Los Angeles: Western Psychological Corporation.
- Loth, E., Murphy, D. G., & Spooren, W. (2016a). Defining precision medicine approaches to autism spectrum disorders: Concepts and challenges. *Frontiers in Psychiatry*, 7, 188. <https://doi.org/10.3389/fpsy.2016.00188>.
- Loth, E., Spooren, W., Ham, L. M., Isaac, M. B., Auriche-Benichou, C., Banaschewski, T., . . . Murphy, D. G. (2016b). Identification and validation of biomarkers

for autism spectrum disorders. *Nature Reviews: Drug Discovery*, 15(1), 70–73. <https://doi.org/10.1038/nrd.2015.7>.

- Loth, E., Charman, T., Mason, L., Tillmann, J., Jones, E. J. H., Wooldridge, C., ... Buitelaar, J. K. (2017). The EU-AIMS longitudinal European autism project (LEAP): Design and methodologies to identify and validate stratification biomarkers for autism spectrum disorders. *Molecular Autism*, 8, 24. <https://doi.org/10.1186/s13229-017-0146-8>.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55(1), 3.
- McPartland, J. C. (2016). Considerations in biomarker development for neurodevelopmental disorders. *Current Opinion in Neurology*, 29(2), 118–122.
- Nalejska, E., Maczynska, E., & Lewandowska, M. A. (2014). Prognostic and predictive biomarkers: Tools in personalized oncology. *Molecular Diagnosis & Therapy*, 18(3), 273–284. <https://doi.org/10.1007/s40291-013-0077-9>.
- Pelphrey, K. (2017). Charting a course for autism biomarkers. *Biological Psychiatry*, 82(3), 155–156. <https://doi.org/10.1016/j.biopsych.2017.06.002>.
- Shen, M. D., Nordahl, C. W., Young, G. S., Wootton-Gorges, S. L., Lee, A., Liston, S. E., ... Amaral, D. G. (2013). Early brain enlargement and elevated extra-axial fluid in infants who develop autism spectrum disorder. *Brain*, 136(Pt 9), 2825–2835. <https://doi.org/10.1093/brain/awt166>.
- Shen, M. D., Kim, S. H., McKinsty, R. C., Gu, H., Hazlett, H. C., Nordahl, C. W., ... Gu, H. (2017). Increased extra-axial cerebrospinal fluid in high-risk infants who later develop autism. *Biological Psychiatry*, 82(3), 186–193. <https://doi.org/10.1016/j.biopsych.2017.02.1095>.
- Smith, T., Groen, A. D., & Wynn, J. W. (2000). Randomized trial of intensive early intervention for children with pervasive developmental disorder. *American Journal of Mental Retardation*, 105(4), 269–285. [https://doi.org/10.1352/0895-8017\(2000\)105<0269:rtotie>2.0.co;2](https://doi.org/10.1352/0895-8017(2000)105<0269:rtotie>2.0.co;2).
- Varcin, K. J., & Nelson, C. A. (2016). A developmental neuroscience approach to the search for biomarkers in autism spectrum disorder. *Current Opinion in Neurology*, 29(2), 123–129. <https://doi.org/10.1097/WCO.0000000000000298>.
- Zhao, X., Modur, V., Carayannopoulos, L. N., & Laterza, O. F. (2015). Biomarkers in pharmaceutical research. *Clinical Chemistry*, 61(11), 1343–1353. <https://doi.org/10.1373/clinchem.2014.231712>.

Birth Complications

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Definition

Birth is a crucial event in life. The transition from the uterine status to the outside world is a very stressful occasion for parents but also for the newborn child. In retrospect, many parents will impute developmental deviances to a poor start in life. Thus, birth and perinatal complications and the first week of the neonate have been the focus of many research aimed to determine if factors involved with birth and start of life play a role in the etiology of autism.

Definitions

- Birth: the transition from intrauterine life to life outside. This includes the vaginal pathway or the extraction through a so-called caesarian operation.
- Complications: any deviance from normal physiology around the birth (perinatal period).
- Perinatal period: an interval extending from the 28th week of gestation until the 28th day after birth.
- Apgar score: simple repetitive method introduced by Virginia Apgar in 1952 to assess the health of a newborn baby – appearance (skin color), pulse (heart rate), grimace (reflex irritability), activity (muscle tone), and respiration. The five criteria are given marks ranging from 0 to 2. 0 is absent of highly disordered and 2 is fair and normal. Thus, the scale ranges from 0 to 10. It is mostly scored 5 and 10 min after birth. 7–10 is considered normal, 4–7 fairly low, and under 3 critically low.

Biomedical Engineer

► [Rehabilitation Engineer](#)

Historical Background

Direct injuries following forceps extractions or acute termination can cause massive damage to the brain of a neonate, with palsy and severe developmental hazards as consequences. These dramatic circumstances have not been related to the emergence in later life of any form of psychopathology. But over the years, subtle deviances at birth (low Apgar scores, respiratory distress, hypoglycemia, or hyperbilirubinemia after 5–7 days) have been associated with developmental disorders such as attention deficit hyperactivity disorders of autism.

When taking the developmental history in parents of children with developmental disorders, one is often struck by the emphasis put on perinatal hazards. These retrospective recollections are not always reliable. Yet they illustrate how much value is given to the condition of the child just after birth as a potential cause of later disorders. Thus, methodologically retrospective data must be distrusted. In this item, the evidence for associations between birth complications and autism will be reviewed from methodologically sound studies.

Current Knowledge

Gardener et al. (2011) carried out a comprehensive meta-analysis to evaluate the perinatal and neonatal risk factors for autism. After a PubMed, Embase, and PsycINFO search, 60 methodologically sound studies (out of 124 published until 2007) could be retained for a thorough meta-analysis of the possible causal relationship between the occurrence between perinatal and neonatal complications and autism. Since then, only five studies were published to date, implying that the Gardener et al. meta-analysis gives a good summary of the current knowledge with regard to peri- and neonatal factors that are associated with an increased risk for autism. This formulation is of great importance because peri- and neonatal factors appear to be by no means specific for any kind of psychopathology, thus pointing towards a multicausal heterogeneity already hypothesized by Bolton et al. (1997).

But the results from series of well-conducted studies clearly show that there are factors that have no association with autism and others that show a positive association with the occurrence of autism later in life:

Factors that show *no association with autism* are the following: premature rupture of membranes, delayed labor, loss of amniotic fluid on the day before delivery, analgesia during labor, green [meconium holding] amniotic fluid, acidosis ($\text{pH} < 7.2$ in the umbilical cord, shoulder dystocia, near-dead baby, “blue baby,” hypoglycemia, hypocalcemia, infantile vomiting, intracranial hemorrhage, macrocephaly, abnormal fetal cardiac activity, assisted vaginal delivery, post-term birth, a high birth weight, and incubator use! Finally, neither preterm birth nor Cesarean delivery reached statistical significance.

Factors that *do show a significant increase of the risk for autism* are the following: abnormal presentation in general, breech presentation, umbilical cord complications (prolapse, cord wrapping around the neck), multiple birth, (very) low birth weight, small for gestational age, fetal distress, Apgar scores low after 5 min, birth injury or trauma, congenital malformations, meconium aspiration, neonatal anemia, ABO or rhesus incompatibility, and hyperbilirubinemia. There are also two factors that are not related directly to the condition of the child but enhance the risk for autism and those are: maternal bleeding and season of birth (with two high-risk periods, namely, children born in March and in the late summer (August and September)).

Yet, from the discussions around birth complications as a risk factor for autism, it appears clearly that they cannot be perceived independently from earlier prenatal factors. Factors such as advanced age of both mother and father, but also an autistic condition in the offspring as a result of genetic and early embryo-environment interplay early in gestation (viral infections, drugs, etc.), suggest that the birth complications are more the result of prenatal factors that are the cause of autism later on. According to the classic Bolton et al. (1997) study on the “shared risk hypothesis,” this shared risk hypothesis is also supported by the Zwaigenbaum et al. studies

(2002) that show more composite prenatal, perinatal, and neonatal adversity among both affected children and unaffected siblings in families with a high loading for the broader autism phenotype.

Future Directions

In order to fully understand the impact as a risk factor of birth complications on the increased risk for autism, longitudinal studies, starting well before birth like the ABC study in Norway of Generation R, are needed in order to get a better understanding of the interplay between family genetic-embryonic development-birth hazards and neonatal stress on the risk factors for autism.

References and Reading

- Bilder, D., Pinborough-Zimmerman, J., Miller, J., & McMahon, W. (2009). Prenatal, perinatal, and neonatal factors associated with autism spectrum disorders. *Pediatrics*, 123(5), 1293–1300.
- Bolton, P. F., Murphy, M., Macdonald, H., Whitlock, B., Pickles, A., & Rutter, M. (1997). Obstetric complications in autism: Consequences or causes of the condition? *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(2), 272–281.
- Brimacombe, M., Ming, X., & Lamendola, M. (2006). Prenatal and birth complications in autism. *Maternal and Child Health Journal*, 11(1), 73–79.
- Burstyn, I., Sithole, F., & Zwaigenbaum, L. (2011a). Autism spectrum disorders, maternal characteristics and obstetric complications among singletons born in Alberta, Canada. *Chronic Diseases in Canada*, 30(4), 125–134.
- Burstyn, I., Wang, X., Yasui, Y., Sithole, F., & Zwaigenbaum, L. (2011b). Autism spectrum disorders and fetal hypoxia in a population-based cohort: Accounting for missing exposures via estimation-maximization algorithm. *BMC Medical Research Methodology*, 11, 2.
- Cederlund, M., & Gillberg, C. (2004). One hundred males with Asperger syndrome: A clinical study of background and associated factors. *Developmental Medicine and Child Neurology*, 46(10), 652–660.
- Croen, L. A., Yoshida, C. K., Odouli, R., & Newman, T. B. (2005). Neonatal hyperbilirubinemia and risk of autism spectrum disorders. *Pediatrics*, 115(2), e135–e138.
- Gardener, H., Spiegelman, D., & Buka, S. L. (2009). Prenatal risk factors for autism: Comprehensive meta-analysis. *The British Journal of Psychiatry*, 195(1), 7–14.
- Gardener, H., Spiegelman, D., & Buka, S. L. (2011). Perinatal and neonatal risk factors for autism: A comprehensive meta-analysis. *Pediatrics*, 128(2), 344–355.
- Glasson, E. J., Bower, C., Petterson, B., de Klerk, N., Chaney, G., & Hallmayer, J. F. (2004). Perinatal factors and the development of autism: A population study. *Archives of General Psychiatry*, 61(6), 618–627.
- Haglund, N. G., & Källén, K. B. (2011). Risk factors for autism and Asperger syndrome. Perinatal factors and migration. *Autism*, 15(2), 163–183.
- Hultman, C. M., Sparén, P., & Cnattingius, S. (2002). Perinatal risk factors for infantile autism. *Epidemiology*, 13(4), 417–423.
- Juul-Dam, N., Townsend, J., & Courchesne, E. (2001). Prenatal, perinatal, and neonatal factors in autism, pervasive developmental disorder-not otherwise specified, and the general population. *Pediatrics*, 107(4), E63.
- Kern, J. K. (2003). Purkinje cell vulnerability and autism: A possible etiological connection. *Brain & Development*, 25(6), 377–382.
- Kolevzon, A., Gross, R., & Reichenberg, A. (2007). Prenatal and perinatal risk factors for autism: A review and integration of findings. *Archives of Pediatrics & Adolescent Medicine*, 161(4), 326–333.
- Lampi, K. M., Banerjee, P. N., Gissler, M., Hinkka-Yli-Salomäki, S., Huttunen, J., Kulmala, U., et al. (2011). Finnish prenatal study of autism and autism spectrum disorders (FIPS-A): Overview and design. *Journal of Autism and Developmental Disorders*, 41(8), 1090–1096.
- Lyall, K., Pauls, D. L., Spiegelman, D., Ascherio, A., & Santangelo, S. L. (2012). Pregnancy complications and obstetric suboptimality in association with autism spectrum disorders in children of the nurses' health study II. *Autism Research*, 5(1), 21–30.
- Maimburg, R. D., & Vaeth, M. (2006). Perinatal risk factors and infantile autism. *Acta Psychiatrica Scandinavica*, 114(4), 257–264.
- Maimburg, R. D., Vaeth, M., Schendel, D. E., Bech, B. H., Olsen, J., & Thorsen, P. (2008). Neonatal jaundice: A risk factor for infantile autism? *Paediatric and Perinatal Epidemiology*, 22(6), 562–568.
- Simon, E. N. (2004). Autism as a birth defect. *Birth Defects Research. Part A, Clinical and Molecular Teratology*, 70(6), 416; 15211712.
- Sivberg, B. (2003). Parents' detection of early signs in their children having an autistic spectrum disorder. *Journal of Pediatric Nursing*, 18(6), 433–439.
- Stein, D., Weizman, A., Ring, A., & Barak, Y. (2006). Obstetric complications in individuals diagnosed with autism and in healthy controls. *Comprehensive Psychiatry*, 47(1), 69–75.
- Stevens, M. C., Fein, D. H., & Waterhouse, L. H. (2000). Season of birth effects in autism. *Journal of Clinical and Experimental Neuropsychology*, 22(3), 399–407.

- Sugie, Y., Sugie, H., Fukuda, T., & Ito, M. (2005). Neonatal factors in infants with autistic disorder and typically developing infants. *Autism*, 9(5), 487–494.
- Taylor, E. (2011). Antecedents of ADHD: A historical account of diagnostic concepts. *Attention Deficit and Hyperactivity Disorders*, 3(2), 69–75.
- Wilkerson, D. S., Volpe, A. G., Dean, R. S., & Titus, J. B. (2002). Perinatal complications as predictors of infantile autism. *International Journal of Neuroscience*, 112(9), 1085–1098.
- Yeates-Frederikx, M. H., Nijman, H., Logher, E., & Merckelbach, H. L. (2000). Birth patterns in mentally retarded autistic patients. *Journal of Autism and Developmental Disorders*, 30(3), 257–262.
- Zhang, X., Lv, C. C., Tian, J., Miao, R. J., Xi, W., Hertz-Picciotto, I., et al. (2010). Prenatal and perinatal risk factors for autism in China. *Journal of Autism and Developmental Disorders*, 40(11), 1311–1321. Erratum in: *Journal of Autism and Developmental Disorders*, 40(11).
- Zwaigenbaum, L., Szatmari, P., Jones, M. B., Bryson, S. E., MacLean, J. E., Mahoney, W. J., et al. (2002). Pregnancy and birth complications in autism and liability to the broader autism phenotype. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(5), 572–579.

effects have been identified in a few studies. One type of birth order effect that has been observed and replicated is a lower nonverbal IQ score in the second child with autism in the family (Lord 1992; Spiker et al. 2001). Another study found that there was an effect of birth order on multiple aspects of autism including repetitive behaviors, phrase speech, social communication, and non-verbal communication (Reichenberg et al. 2007). Finally, in a more recent study general birth order effects were seen where middle births in multiplex families and later births in simplex families were more likely to develop autism (Turner et al. 2011). There are a number of potential reasons for birth order effects, and these include demographic factors such as stoppage in a family after the first child with autism is born, as well as biological factors including paternal age effects, maternal age effects, maternal-fetal genotype incompatibilities, and potential epigenetic effects. The discovery of birth order effects can help guide researchers in their examination of potential risk factors for autism.

Birth Order

► Birth Order Effects in Autism

Birth Order Effects in Autism

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Synonyms

Birth order; Birth rank effect

Definition

Birth order relates to the order in which a child is born to a set of parents and birth order effects refer to differences seen between individuals that are not of the same birth order. In autism, birth order

References and Reading

- Lord, C. (1992). Birth order effects on nonverbal IQ in families with multiple incidence of autism or pervasive developmental disorder. *Journal of Autism and Developmental Disorders*, 22(4), 663–666.
- Reichenberg, A., Smith, C., Schmeidler, J., & Silverman, J. M. (2007). Birth order effects on autism symptom domains. *Psychiatry Research*, 150(2), 199–204. <https://doi.org/10.1016/j.psychres.2004.09.012>.
- Spiker, D., Lotspeich, L. J., Dimiceli, S., Szatmari, P., Myers, R. M., & Risch, N. (2001). Birth order effects on nonverbal IQ scores in autism multiplex families. *Journal of Autism and Developmental Disorders*, 31(5), 449–460.
- Turner, T., Pihur, V., & Chakravarti, A. (2011). Quantifying and modeling birth order effects in autism. *PLoS One*, 6(10), e26418. <https://doi.org/10.1371/journal.pone.0026418>.

Birth Rank Effect

► Birth Order Effects in Autism

Birth-to-Three

► Early Intervention

BITSEA

► Brief Infant-Toddler Social and Emotional Assessment (BITSEA)

BITSEA-ASD Subscales

► Brief Infant-Toddler Social and Emotional Assessment (BITSEA)

Blindness

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Definition

The relationship of blindness and autism spectrum disorders (ASD) is complex, and, as a group of prominent researchers had aptly noted, it has been a continuing source of interest and perplexity for researchers and clinicians (Hobson et al. 1999). Central to examinations of the relationship is a collection of behaviors regarded as characteristic of children who are blind, in particular children who are congenitally blind, and children who have profound visual impairment. The collection includes stereotyped and ritualistic behaviors, pronounced limitations of social and communicative competence, delayed and limited symbolic play and language, delayed use and reversals of personal pronouns, echolalia and speech limitations, and difficulties with abstract thinking (Gense and Gense 2005; Perez-Pereira and Conti-Ramsden 1999).

Many of these behaviors also are considered to be characteristic of sighted children who have ASD. Because of the seeming commonality of certain behaviors of children who are blind and children who have ASD, researchers and clinicians have been challenged in understanding the nature of the apparent similarities (Fazzi et al. 2007; Hobson and Lee 2010; Parr et al. 2010). Essentially, the question is whether such behaviors, when demonstrated by blind children, indicate ASD. Blindness literature can offer other hypotheses for what are viewed as autistic-like behaviors in blind children: Careful consideration should be given to sensory and social deprivation, early medical complications, limited motor or physical activities, lack of ability to imitate, lack of a variety of activities, self-regulation, and others (Huebner 1986; McHugh and Lieberman 2003; Warren 1984).

The preeminent challenge for both researchers and clinicians may well be definitively identifying ASD in individuals who have significant vision loss. A major hurdle is the lack of appropriate diagnostic screening and assessment tools for this population. Standards, such as the Autism Diagnostic Observation Schedule (ADOS: Lord et al. 1999, 2001, 2002, 2008) and Childhood Autism Rating Scale (CARS: Schopler et al. 1988; Schopler and Van Bourgondien 2010), which are designed for use with sighted individuals, are heavily weighted with visually based tasks. In assessing blind subjects, some researchers have made modifications to these tools, claiming their clinical utility, although the validity of these modifications has not been tested (Jutley-Neilson et al. 2013; Williams et al. 2014). The absence of measures designed for use with children who have significant visual impairments compounds the difficulties of differentiating autistic-like behaviors from ASD in blind children.

Historical Background

In the field of blindness, the classic writings of Selma Fraiberg are most often recognized as the first reports of autistic-like behaviors in blind

children. In 1964, Fraiberg and Freedman published their observations of a group of blind children, stating that nearly a third of the children had “ego deviation.” Keeler (1958), however, is credited with the earliest report of such behaviors in his study of young children with retrolental fibroplasia (otherwise known as retinopathy of prematurity). Several later studies continued investigation of an association between ASD and specific ophthalmological disorders or certain genetic disorders associated with a significant visual impairment. The most frequently researched diagnoses have included Leber’s congenital amaurosis, optic nerve hypoplasia, septo-optic nerve dysplasia, CHARGE syndrome, and retinopathy of prematurity (Bahar et al. 2003; Chase 1972; Ek et al. 1998; Smith et al. 2005). Researchers, however, are not in consensus regarding the association between a specific ophthalmological diagnosis and ASD (Fraiberg 1977; Hobson et al. 1999; Mukaddes et al. 2007).

Some investigators indicated that congenital blindness predisposed a child to ASD (Brown et al. 1997). As researchers sought a better understanding of the relationship between blindness and ASD, the scope of investigations broadened. The degree of vision impairment in relation to the manifestation of autistic-like features or ASD in children who are blind has been a focus of investigators. Some researchers have claimed that more severe vision loss, especially loss of the ability to distinguish forms, increases the likelihood of autistic-like behaviors (Cass et al. 1994). Cognitive impairment reflected in low IQ scores, as well as other additional disabilities, has also been associated with autistic-like behaviors and ASD in blind children (Brown et al. 1997).

Other studies have examined the roles of sensory deprivation and related environmental factors in contributing to the presence of the autistic-like behaviors and ASD. Some investigators have taken a functional perspective of these behaviors, countering that in most cases, such behaviors are adaptive responses to vision loss (Cass 1998; Mottron and Burack 2001). A later review of studies reported that there were no consistent results regarding the relationship and specific types of ophthalmological diagnoses, the severity

of vision impairment, and the role of associated disabilities (Mukaddes et al. 2007).

Tied to the complexities of identification of ASD in blind children have been questions of prevalence. Reported prevalence rates have varied greatly. The US Centers for Disease Control and Prevention (CDC) conducted surveillance of children with visual impairment in metropolitan Atlanta, Georgia, and determined that approximately 6–7% of the children had co-occurring ASD (Kancherla et al. 2013). Studies have reported up to 30 times greater prevalence of ASD in blind children than in sighted children (Cass et al. 1994; Hobson et al. 1999; Jure et al. 2016).

Current Knowledge

A recent review of 11 studies published from 2000 to 2015 focused on the similarity between visual impairment and autistic traits (Buchart et al. 2017). Findings suggested that the presence of autistic traits, such as limited communication and social interaction, together with repetitive and restrictive behavior does not necessarily warrant a diagnosis of ASD. The authors specifically noted that sample sizes were small, and measures used to diagnose ASD have not been systematically tested on a broader visually impaired population. [It is important to recognize, however, the low prevalence rate of childhood blindness. According to a US survey, it is the least prevalent, 0.13%, of all developmental disabilities (Boyle et al. 2011)]. The authors also expressed concern that some studies had adapted standardized autism diagnostic measures, thus undermining their validity and reliability.

Debates have continued regarding the nature of autistic-like behaviors in children with significant vision loss. Andrews and Wyver (2005) have held that blind children who also display such behaviors should be viewed as having specific features rather than being on the autism spectrum. The authors questioned whether these behaviors of blind children are characteristic of *true* ASD or a different developmental pathway. Hobson and Lee (2010) also referred to a distinctive

underlying pathway for blind children's features that seem characteristic of ASD. They, in fact, termed the features as being *quasi*-autistic and suggested the clinicians hold back from the designation of *ASD*. Williams et al. (2014) reported that some behaviors suggestive of ASD in sighted children *do not* distinguish children with ASD and significant vision loss from children without ASD and significant vision loss.

Researchers have given particular attention to young blind children who exhibit autistic-like behaviors. The recent study of Williams et al. (2014) included parent reports based on a modified version of the Autism Diagnostic Interview, Revised (ADI-R; Rutter et al. 2003). Several parents of young blind children noted significant differences in their children's autistic-like symptoms before and after the age of 5 years. They reported that as their children developed and became comfortable exploring their environments, their social and communicative behaviors increased greatly, while their repetitive behaviors decreased proportionally. Similarly, a much earlier study of congenitally blind children found that various stereotypic behaviors occur during the first and second years of life, but many decrease from the age of 3 years onward (Troster et al. 1991). The findings also appear consistent with research by Hobson and Lee (2010), who reassessed nine congenitally blind children and seven sighted comparison children, all of whom had met diagnostic criteria for ASD, 8 years earlier. The results of reassessment determined that only one of the blind children met criteria for ASD, although all seven sighted children continued to meet the criteria. Thus, the message to clinicians is to use caution in diagnosing ASD in very young children with vision impairment, as the symptoms experienced by some children may improve significantly as they develop further (Williams et al. 2014).

Differentiating between autistic-like behaviors related to blindness and those essential to a diagnosis of ASD is particularly challenging, especially if clinicians have limited experience with blind children. When considering children with significant vision loss, there is risk of misinterpreting the basis of a child's behaviors if the

child is not provided appropriate modes and opportunities to develop and demonstrate competence.

Future Directions

Clearly, efforts must be directed toward the development of screening and assessment tools for identification of ASD in individuals with significant vision loss. Current diagnostic screening and assessment tools are heavily weighted with visually based features and tasks, having been designed for use with sighted children. The components of tools for use with individuals who have significant visual loss need to be grounded in modalities other than solely visual. Researchers and clinician will need to devise alternative equivalents for criteria that are basic to current measures, such as eye contact, directed gaze, and joint attention. The development of such measures is in very preliminary stages. A key concern is the lack of information regarding the progression of social development in the very heterogeneous population of individuals with significant vision loss. Much is yet to be learned. Normative data on social development in children with visual impairment are necessary to best inform diagnostic criteria for ASD in this population.

Directly tied to identification of ASD in children who are blind or who have significant visual impairment is the need for appropriate teaching methods, tools, and strategies for this population. It has not been established whether the current instructional interventions used with sighted children who have ASD are the most appropriate for children with significant vision loss, nor whether the various means can be adapted to be such. The question merits investigation, with the ultimate goal of developing strategies, tools, and materials to address the specific learning needs of this population.

Through the review of studies regarding blindness and ASD, it is readily apparent that efforts have been focused nearly exclusively on children, especially young children. A near void exists regarding information about ASD in blind adults. A broader, life course perspective is called for in future studies.

See Also

- Chess, Stella
- Rubella

References and Reading

- Andrews, R., & Wyver, S. (2005). Autistic tendencies: Are there different pathways for blindness and autism spectrum disorder? *British Journal of Visual Impairment*, 23(2), 52–57.
- Bahar, C., Brody, J., McCann, M. E., Mendiola, R. M., & Slot, G. (2003). A multidisciplinary approach to educating preschool children with optic nerve hypoplasia and septo-optic nerve dysplasia. *RE View*, 35(1), 15–21.
- Boyle, C. A., Boulet, S., Schieve, L. A., Cohen, R. A., Blumberg, S. J., Yeargin-Allsopp, M., et al. (2011). Trends in the prevalence of developmental disabilities in US children, 1997–2008. *Pediatrics*, 127(6), 1034–1042.
- Brown, R., Hobson, R. P., & Lee, A. (1997). Are there “autistic-like” features in congenitally blind children? *Journal of Child Psychology and Psychiatry*, 38(6), 693–703.
- Buchart, M., Long, J. J., Brown, M., McMillan, A., Bain, J., & Karatzias, T. (2017). Autism and visual impairment: A review of the literature. *Review Journal of Autism and Developmental Disorders*, 4, 118–131.
- Cass, H. (1998). Visual impairment and autism: Current questions and future research. *Autism*, 2(2), 117–138.
- Cass, H., Sonkensen, P. M., & McCohachie, H. R. (1994). Developmental setback in severe visual impairment. *Archives of Disease in Childhood*, 70, 192–196.
- Chase, J. B. (1972). *Retrolental fibroplasia and autistic symptomatology*. New York: American Foundation for the Blind.
- Chess, S. (1971). Autism in children with congenital rubella. *Journal of Autism and Childhood Schizophrenia*, 1, 33–47.
- Dale, N., & Salt, A. (2008). Social identity, autism and visual impairment in the early years. *British Journal of Visual Impairment*, 26(2), 135–146.
- de Vaan, G., Vervloed, M., Peters-Scheffer, N., van Gent, T., Knoors, H., & Verhoeven, L. (2016). Behavioural assessment of autism spectrum disorders in people with multiple disabilities. *Journal of Intellectual Disability Research*, 60(2), 101–112.
- de Verdier, K., Fernell, E., & Ek, U. (2019). Blindness and autism: Parents’ perspectives on diagnostic challenges, support needs and support provision. *Journal of Autism and Developmental Disorders*, 49, 1–10.
- Ek, U., Fernell, E., Jacobson, L., & Gilberg, C. (1998). Relation between blindness due to retinopathy of prematurity and autism spectrum disorders: A population-based study. *Developmental Medicine and Child Neurology*, 40, 297–301.
- Ek, U., Fernell, E., Jacobson, L., & Gilberg, C. (2005). Cognitive and behavioural characteristics in blind children with bilateral optic nerve hypoplasia. *Acta Paediatrica*, 94, 1421–1426.
- Fazzi, E., Rossi, M., Signorini, S., Rossi, G., Bianchi, P. E., & Lanzi, G. (2007). Leber’s congenital amaurosis: Is there an autistic component? *Developmental Medicine and Child Neurology*, 49, 503–507.
- Fraiberg, S. (1977). *Insights for the blind*. New York: Basic Books.
- Fraiberg, S., & Freedman, D. (1964). Studies in the ego development of the congenitally blind. *The Psychoanalytic Study of the Child*, 19, 113–169.
- Gense, M. H., & Gense, D. J. (2005). *Autism spectrum disorders and visual impairment: Meeting students’ learning needs*. New York: AFB Press.
- Gense, M. H., & Gense, D. J. (2011). Autism spectrum disorders and visual impairment are here to stay: Using an expanded core curriculum to implement a comprehensive program of instruction. *Journal of Visual Impairment & Blindness*, 105(6), 329–334.
- Hobson, R. P., & Lee, A. (2010). Reversible autism among congenitally blind children? A controlled follow-up study. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 51(11), 1235–1241.
- Hobson, R. P., Lee, A., & Brown, R. (1999). Autism and congenital blindness. *Journal of Autism and Developmental Disorders*, 29(1), 45–56.
- Huebner, K. M. (1986). Social skills. In G. T. Scholl (Ed.), *Foundations of education for blind and visually handicapped children and youth* (pp. 341–362). New York: American Foundation for the Blind.
- Jure, R., Pogonza, R., & Rapin, I. (2016). Autism spectrum disorders (ASD) in blind children: Very high prevalence, potentially better outlook. *Journal of Autism and Developmental Disorders*, 46(3), 749–759.
- Jutley-Neilson, J., Harris, G., & Kirk, J. (2013). The identification and measurement of autistic features in children with septo-optic dysplasia, optic nerve hypoplasia and isolated hypopituitarism. *Research in Developmental Disabilities*, 34(12), 4310–4318.
- Kancherla, V., Van Naarden Braun, K., & Yeargin-Allsopp, M. (2013). Childhood vision impairment, hearing loss and co-occurring autism spectrum disorder. *Disability and Health Journal*, 6(4), 333–342.
- Keeler, W. R. (1958). Autistic patterns and defective communication in blind children with retrolental fibroplasia. In P. H. Hoch & J. Zubin (Eds.), *Psychopathology of communication* (pp. 64–83). New York: Grune & Stratton.
- Kiani, R., Bhaumik, S., Tyrer, F., Bankart, J., Miller, H., Cooper, S. A., & Brugha, T. S. (2019). The relationship between symptoms of autism spectrum disorder and visual impairment among adults with intellectual disability. *Autism Research: Official Journal of the International Society for Autism Research*, 12(9), 1411–1422.
- Lord, C., Rutter, M., DeLavore, P. C., & Risi, S. (1999, 2001, 2002, 2008). *Autism diagnostic observation*

- schedule*. Los Angeles: Western Psychological Services.
- McHugh, B. E., & Lieberman, L. J. (2003). The impact of developmental factors on incidence of stereotypic sucking among children with visual impairments. *Journal of Visual Impairment and Blindness*, 97(8), 453–474.
- Mottron, L., & Burack, J. A. (2001). Enhanced perceptual functioning in the development of autism. In J. A. Burack, T. Charman, N. Yirmiya, & P. R. Zelazo (Eds.), *The development of autism* (pp. 131–148). Mahwah: Lawrence Erlbaum Associates.
- Mukaddes, N. M., Kilincasin, A., Kuckyazici, G., Sevetoglu, T., & Tuncer, S. (2007). Autism in visually impaired individuals. *Psychiatry and Clinical Neurosciences*, 61(1), 39–44.
- Parr, J. R., Dale, N. J., Shaffer, L. M., & Salt, A. T. (2010). Social communication difficulties and autism spectrum disorder in young children with optic nerve hypoplasia and/or septo-optic dysplasia. *Developmental Medicine and Child Neurology*, 52(10), 917–921.
- Perez-Pereira, M., & Conti-Ramsden, G. (1999). *Language development and social interaction in blind children*. East Sussex: Psychology Press.
- Pring, L. (Ed.). (2005). *Autism and blindness: Research and reflections*. London: Whurr Publishers.
- Rutter, M., LeCouteur, A., & Lord, C. (2003). *Autism diagnostic interview-revised*. Los Angeles: Western Psychological Services.
- Schopler, E., & Van Bourgondien, M. E. (2010). *The childhood autism rating scale* (2nd ed.). Los Angeles: Western Psychological Services.
- Schopler, E., Reichler, R., & Rothen Renner, B. (1988). *The childhood autism rating scale*. Los Angeles: Western Psychological Services.
- Smith, I. M., Nichols, S. L., Issekutz, K., & Blake, K. (2005). Behavioral profiles and symptoms of autism in CHARGE syndrome. *American Journal of Medical Genetics*, 133A, 248–256.
- Troster, H., Brambring, M., & Beelman, A. (1991). The age dependence of stereotyped behaviours in blind infants and preschoolers. *Child: Care, Health and Development*, 17, 137–157.
- Warren, D. H. (1984). *Blindness and early childhood development* (2nd ed.). New York: American Foundation for the Blind.
- Williams, M. E., Fink, C., Zamora, I., & Borchert, M. (2014). Autism assessment in children with optic nerve hypoplasia and other vision impairments. *Developmental Medicine and Neurology*, 56(1), 66–72.

Block Design Subtest

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Definition

Block design is a subtest that is administered as part of several of the Wechsler Intelligence tests, including the Wechsler Preschool and Primary Scale of Intelligence (WPPSI, the Wechsler Intelligence Scale for Children-fourth edition (WISC-IV; Wechsler 2003) and the Wechsler Adult Intelligence Scale-fourth edition (WAIS-IV; Wechsler 2008). This subtest is included in the calculation of Performance IQ. It is primarily a measure of visual-spatial and organizational processing abilities, as well as nonverbal problem-solving skills. Because it is a timed task, it is also influenced by fine motor skills. The individual is presented with identical blocks with surfaces of solid red, surfaces of solid white, and surfaces that are half red and half white. Using an increasing number of these blocks, the individual is required to replicate a pattern that the test administrator presents to them – first as a physical model, and then as a two-dimensional picture. The number of blocks required to match the presented models increases and the patterns become increasingly difficult to visually dissect into components.

Individuals who do well on this subtest tend to have an aptitude for perceiving spatial patterns and for flexible problem solving; performance is also aided by the ability to work quickly. Conversely, one factor that may hinder an individual's performance on block design is the presence of high anxiety or perfectionistic tendencies (Hopko et al. 2005), as these can lead to an overly cautious approach that causes the individual to finish after the time limit. Poor performance may also be related to a number of factors that affect an individual's ability to perceive spatial patterns, manipulate objects, or integrate visual and spatial information. Of note, individuals

Blitz-Nick-Salaam Krämpfe

► Infantile Spasms/West Syndrome

with autism spectrum disorders have been observed to show superior performance on the block design task (Shah and Frith 1993). This relative strength is described by the hypothesis of the Weak Central Coherence Theory, which suggests individuals with autism have difficulty seeing the “big picture,” and instead may perceive parts of the whole with more relative skill than individuals without autism (Happé and Frith 2006).

While not captured in the final score of block design, it is clinically useful to observe how an individual approaches this task. One such behavior that can be informative to the test administrator includes the above-mentioned perfectionistic tendency, or alternatively, the tendency to be impulsive or careless. An individual's persistence may also be noted, as well as whether the individual tends to approach the pattern in a piecemeal fashion, or in a more global fashion.

See Also

- [Perceptual Organization Index \(POI\)](#)
- [Weak Central Coherence](#)
- [Wechsler Preschool and Primary Scale of Intelligence](#)

References and Reading

- Happé, F., & Frith, U. (2006). The weak central coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36, 5–25.
- Hopko, D. R., Crittendon, J. A., Grant, E., & Wilson, S. A. (2005). The impact of anxiety on performance IQ. *Anxiety, Stress, & Coping: An International Journal*, 18, 17–35.
- Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry*, 34, 1351–1364.
- Wechsler, D. (2002). *The Wechsler preschool and primary scale of intelligence* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2003). *Wechsler intelligence scale for children* (4th ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2008). *Wechsler adult intelligence scale* (4th ed.). San Antonio: Pearson.

Blood-Oxygen-Level Dependence

- [Blood-Oxygen-Level-Dependent \(BOLD\) Signal](#)

Blood-Oxygen-Level-Dependent (BOLD) Contrast

- [Event-Related Functional Magnetic Resonance Imaging \(MRI\)](#)

Blood-Oxygen-Level-Dependent (BOLD) Signal

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Synonyms

[Blood-oxygen-level dependence](#)

Definition

Blood-oxygen-level-dependent (BOLD) signal is the magnetic resonance imaging (MRI) contrast of blood deoxyhemoglobin. Seiji Ogawa and his colleagues first discovered this intrinsic contrast mechanism in 1990. Neurons do not store internal reserves of glucose and oxygen, which are essential to their proper function. Increases in neuronal activity, typically in response to a demand for information processing, require more glucose and oxygen to be rapidly delivered via the blood stream. Via this hemodynamic response, blood releases glucose and oxygen to active neurons at a faster rate relative to inactive neurons. This results in a surplus of oxyhemoglobin localized

to the active area, giving rise to a measureable change in the local ratio of oxy- to deoxyhemoglobin, thus providing a localizable marker of activity for MRI.

See Also

- [Event-Related Functional Magnetic Resonance Imaging \(MRI\)](#)

References and Reading

- Belliveau, J. W., Kennedy, D. N., McKinstry, R. C., Buchbinder, B. R., Weisskoff, R. M., Cohen, M. S., Vevea, J. M., Brady, T. J., & Rosen, B. R. (1991). Functional mapping of the human visual cortex by magnetic resonance imaging. *Science*, 254, 716–719.
- Kwong, K. W., Belliveau, J. W., Chesler, D. A., Goldberg, I. E., Weisskoff, R. M., Poncelet, B. P., Kennedy, D. N., Hoppel, B. E., Cohen, M. S., Turner, R., Cheng, H., Brady, T. J., & Rosen, B. R. (1992). Dynamic magnetic resonance imaging of human brain activity during primary sensory stimulation. *Proceedings of the National Academy of Sciences*, 89, 5951–5955.
- Ogawa, S., Lee, T. M., Nayak, A. S., & Glynn, P. (1990). Oxygenation-sensitive contrast in magnetic resonance image of rodent brain at high magnetic fields. *Magnetic Resonance in Medicine*, 14, 68–78.

Blüm Study

- [CM-AT](#)

Board Certified Associate Behavior Analyst

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Synonyms

[BCaBA](#); [BCBA](#); [BCBA-D](#)

Definition

The Behavior Analyst Certification Board[®], Inc. (BACB[®]) credentials practitioners at four levels. The different categories denote varied depths of training and levels of independence in practice.

Individuals who apply to become *Board Certified Behavior Analysts*[®] (BCBA[®]) must possess at least a master's degree, have 225 classroom hours of specific graduate-level coursework, meet supervised experience requirements, and pass the BCBA examination. In order to use the *Board Certified Behavior Analyst - Doctoral (BCBA-D)* designation, a BCBA must possess an acceptable doctoral degree and meet other criteria. As of 2015, applicants for the BCBA credential will need to have completed 270 h of specific coursework.

Persons who apply to become *Board Certified Assistant Behavior Analysts*[®] (BCaBA[®]) must have at least a bachelor's degree, have 135 classroom hours of specific coursework, meet supervised experience requirements, and pass the BCaBA examination. The BCaBA must have 1000 h of supervised independent field or 670 practicum hours or 500 intensive practicum hours. Once certified, BCaBAs must be supervised by a BCBA; this supervision requirement includes supervision for 5% of hours for the first 1000 h post certification and then ongoing supervision for at least 2% of hours following the initial 1000 post certification hours.

A recently added credential, *Registered Behavior Technician*, has created a professional level for behavior technicians. Individuals with this credential must practice under a BCBA, BCaBA, or FL-CBA. In order to obtain the RBT credential, the individual must have a high school diploma, complete 40 h of training, pass a competency exam, and demonstrate competency across a task list.

Experience and training requirements at all levels of certification are rigorous and ensure that certificants meet minimal competence levels in their knowledge and abilities. BACB certificants must accumulate continuing education credit and recertify over 3 years to maintain their credential. In addition, certificants must annually confirm that they remain in compliance with the

BACB's standards, including ethical guidelines and disciplinary standards.

Because certification requirements periodically change as standards are increased, readers are encouraged to consult (www.bacb.com) for updated information.

Body Movements, Imitation of

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Synonyms

[Gestural imitation](#); [Imitation of intransitive actions](#); [Imitation of nonmeaningful gestures](#)

Definition

Imitation of body movements involves copying acts that do not include the use of objects, do not lead to an end state, do not carry a specific meaning, and can only be described in terms of changes of limb postures in space (e.g., a hand moving across a forehead). Current models of imitation suggest that imitation of body movements is supported by mechanisms that partially differ from those underlying the imitation of actions that carry a semantic meaning (e.g., opening a container or waving goodbye). While imitation of body movement is supported by a “direct visuo-spatial route” in which the visual input is directly mapped into a motor output, imitation of actions that carry a semantic meaning is achieved via a “semantic route” in which previous knowledge on the meaning of the action can be recruited (Tessari and Rumiati 2004). Given that the familiarity with the demonstrator's goals and means cannot be exploited in this type of imitative task, imitation of body movement is considered to provide a rigorous methodology by which to assess “true imitation” in human and comparative research.

Early signs of the ability to imitate body movements are reported to be present since early

infancy, and mutual imitation games between child and caregiver, involving affective mirroring and copying of body movements, are observed throughout infancy and toddlerhood across cultures. These early reciprocal exchanges are thought to promote social bonding and to provide a foundation for social cognitive development (Meltzoff 2002; Stern 1985).

Difficulties in imitating body movements in individuals with autism are reported in many studies that used different stimuli, coding systems, and comparison groups (including different clinical populations) and across a wide range of IQ, language levels, and chronological ages (see Edwards 2014; Rogers and Williams 2006). Differences in the way individuals with autism imitate body movements include (1) reduced frequency of spontaneous imitation and (2) diminished accuracy of imitative performance. While autism-specific deficits are documented in several imitative tasks, imitation of body movements appears to be more impaired than imitation of actions carrying a semantic meaning in this population (Vivanti and Hamilton 2014; Williams et al. 2004). Various explanations have been hypothesized to account for these difficulties in autism, including abnormalities in visual attention, a primary deficit in the perception-action mapping implemented by the mirror neuron system, a reduced motivation to imitate, and a primary deficit in motor execution. However, none of this explanation is supported by unequivocal evidence. Since children with autism have difficulties in many of the neurocognitive processes involved in imitation of body movements, including visual attention to the demonstration, social motivation, motor planning, and executive processes, it is likely that a heterogeneous vulnerability in the components of the imitative process, rather than a single cause, affects the ability to imitate body movements in individuals with autism.

See Also

- ▶ [Apraxia](#)
- ▶ [Imitation](#)
- ▶ [Mirror Neuron System](#)
- ▶ [Motor Planning](#)

References and Reading

- Edwards, L. A. (2014). A meta-analysis of imitation abilities in individuals with autism spectrum disorders. *Autism Research*, 7(3), 363–380.
- Meltzoff, A. N. (2002). Elements of a developmental theory of imitation. In A. N. Meltzoff & W. Prinz (Eds.), *The imitative mind: Development, evolution, and brain bases* (pp. 19–41). Cambridge, England: Cambridge University Press.
- Rogers, S. J., & Williams, J. H. G. (2006). Imitation in autism: Findings and controversies. In S. J. Rogers & J. H. G. Williams (Eds.), *Imitation and the social mind: Autism and typical development* (pp. 277–309). New York: Guilford.
- Stern, D. (1985). *The interpersonal world of the infant*. New York: Basic Books.
- Tessari, A., & Rumiati, R. I. (2004). The strategic control of multiple routes in imitation of actions. *Journal of Experimental Psychology: Human Perception and Performance*, 30, 1107–1116.
- Vivanti, G., & Hamilton, A. F. (2014). Imitation in autism spectrum disorders. In F. Volkmar, R. Paul, & S. Rogers (Eds.), *The handbook of autism and developmental disorders* (pp. 278–301). New York: Wiley.
- Williams, J. H. G., Whiten, A., & Singh, T. (2004). A systematic review of action imitation in autistic spectrum disorder. *Journal of Autism and Developmental Disorders*, 34, 285–299.

IQ scores below average but above an intellectual disability (ID). People with BIF may have adaptive functioning deficits similar to those experienced by people with mild intellectual disabilities (ID) but are often denied critical services that are available to people with ID diagnoses because their IQs scores are too high (Peltopuro et al. 2014).

Adults with borderline intellectual functioning, with or without autism, often require supports similar to those beneficial to people with mild ID. Comprehensive case management is a valuable and effective method of supporting adults with borderline intellectual functioning. Comprehensive case management involves a primary support professional or team of professionals working collaboratively to help individuals set, pursue, and attain goals for health, safety, employment, socialization, and independence. Case management often begins with addressing concrete or urgent needs, such as for housing, healthcare, or employment, and expands to promote social-emotional wellbeing and personal fulfilment. It involves coordination of care across many service systems and domains and is highly individualized.

Bogus Therapy

► Pseudoscience

Borderline Intellectual Functioning and Comprehensive Case Management

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Definition

Borderline intellectual functioning (BIF) is a little-known and little-utilized classification that has historically described individuals who have

Historical Background

The history of borderline intellectual functioning as an official diagnosis is one of progressively declining prominence and usefulness (Wieland and Zitman 2016). In the DSM-II, what is referred to here as borderline intellectual functioning was called borderline mental retardation. In the DSM-III, borderline mental retardation (i.e., BIF) was removed from mental retardation and reclassified as a V code, leaving a whole population of individuals without a suitable diagnosis. It remained a V code in the DSM-IV-TR and was defined using an IQ score range of 71–84. In the DSM-5, the IQ range was removed, leaving it with little definition and limited usefulness as a classification.

The DSM-5 leaves gray area in the diagnosis of intellectual disability as well, by emphasizing a person's adaptive functioning and the diagnosing clinician's judgement over score in making the diagnosis. In practice, this means that some

children and adults whose IQ scores are over 70 (who may have previously been described as having BIF) may now receive ID diagnoses, while others may not. This gray area is particularly concerning considering the difficulty of accurately measuring IQ in people with autism (Rao et al. 2015).

The IQ score range of 71–84 is between 1 and 2 standard deviations below the mean of the standardized distribution of scores. Because the scores fit a normal curve, as much as 13.6% of people fall into this IQ range. The incidence of BIF is nearly twice as high among people with autism. According to the Centers for Disease Control’s 2012 prevalence data, 24.5% of children with autism have IQ scores in the range of 71–84, compared with 31.6% in the range of an intellectual disability and 43.9% with average or above average IQ (Christensen et al. 2016). While they may have a diagnosis of autism without intellectual disability, according to the DSM-5, this system of classification does not capture their borderline intellectual functioning unless it is specifically noted with a V code by the diagnosing clinician. Children with BIF are known to be at increased risk for persistent mental health issues (Jankowska 2016), poor social functioning drug abuse (Gigi et al. 2014), poor parenting, and school adjustment issues (Jankowska et al. 2014), making it critically important that BIF be recognized.

Rationale or Underlying Theory

While some children and adults with borderline intellectual functioning may have only mild adaptive functioning deficits, or even none at all, others experience deficits comparable to those experienced by individuals with mild intellectual disabilities. A review of the literature on BIF shows that, compared with peers who have average IQ scores, people with BIF have lower performance on tests of cognitive and academic skills, hold jobs that are lower-skilled and lower-paid, have poorer executive functioning and abstract reasoning, have slower processing speeds, and are more likely to be incarcerated or

charged with crimes, have mental health problems, or display antisocial behaviors (Peltopuro et al. 2014). On many of these measures, people with BIF fall between people with specific learning disabilities and those with mild intellectual disabilities. Authors of the review conclude that people with BIF may be in a “worse situation” than adults with MID (mild intellectual disability) or with SLDs (specific learning disabilities) (p. 438). They explain that “because the problems with BIF are not as visible as those in MID and not as specific as those in SLDs, they often go unrecognized and, consequently, no support is offered.” In the United States, a diagnosis of an ID is required to receive certain critical supports, services, and government entitlements; people with BIF are denied services that could dramatically improve their life circumstances, all because of their slightly higher IQ scores. Children and adults with a diagnosis of autism who have IQ scores in this range may be able to qualify for some supports and services based on their autism diagnosis, even without an accompanying diagnosis of ID. However, this is not the case in all places or for all services.

In the United States, children with disabilities are guaranteed a free and appropriate public education. A child who fits the, albeit vague, classification for borderline intellectual functioning (but not intellectual disability) may have another diagnosis, like autism, a specific learning disability, or ADHD, that can qualify them for public school services and accommodations. As an adult, these diagnoses do not guarantee comprehensive state or federal services.

Shattuck et al. (2012) note that while research on services and interventions for children with autism has become more robust in sync with the increasing prevalence of autism diagnoses, research on adult services has been slow to follow. It is clear that autism can be impactful across the lifespan and that adults with autism have unmet service needs in multiple life domains (Turcotte et al. 2016). This is particularly true for individuals with BIF or ID, and appropriate services are less available to adults with BIF than to adults with ID. The services that do exist tend to be siloed; for example, an adult with autism and

BIF can access employment supports from their state vocational rehabilitation agency but may need another agency to provide assistance in managing an apartment in order to live independently. Adult service systems are generally not designed to work seamlessly together to support the needs of each individual accessing them, and an adult with BIF may not even qualify for services from all the individual systems that would be relevant for their goals. Those they do qualify for can be difficult for someone with a cognitive impairment to access and utilize effectively. Filling out paperwork, getting to appointments on time, and finding an unfamiliar service location can all be significant challenges for someone with borderline intellectual functioning. Case managers can assist people with BIF find and use the services they need and coordinate care across services.

For adults with intellectual disabilities, case management may be provided by a state developmental disability agency and/or the staff of a residential program and it may or may not be comprehensive. For adults who do not live in staffed residences and who do not qualify for services from a state developmental disability agency due to relatively higher IQ scores, as is the case for most adults with borderline intellectual functioning, comprehensive case management must come from another source. Adults with autism and borderline intellectual functioning, or their families and supporters, should check with their state developmental disability agency to find out whether they qualify for case management from the state even with IQ scores higher than those required for a diagnosis of an intellectual disability. If not, this type of case management may be offered privately by individuals or by disability service organizations, either for profit or not for profit.

Goals and Objectives

The goal of comprehensive case management is to support adults in setting, pursuing, attaining, and maintaining goals of their own choosing and to

promote their overall health, safety, happiness, and well-being. Typically, comprehensive case management is most useful when an individual has multiple or complex goals, or has struggled with setting or attaining goals in the past. It may also be particularly useful for people with a dual diagnosis or a special health care need. Goals may include:

- Finding employment
- Living independently
- Making friends and learning how to manage relationships
- Managing adult responsibilities, like keeping up an apartment and organizing important paperwork
- Learning how to navigate public transportation
- Improved budgeting and money management
- Securing and/or managing entitlements and benefits (health insurance, Social Security payments, affordable housing, etc.)
- Attaining a higher level of education
- Coordinating health care services
- Planning for life after the death of parents or caretakers
- Any other goal that could be supported by a case manager

Goals should be set by the client, with the support of the case manager and of other people who are involved in life-planning with the client (e.g., family members, mental health clinicians, doctors, and educators).

Treatment Participants

Ideal candidates for comprehensive case management are adults with borderline intellectual functioning who require support in one or more areas in order to achieve the level of independence, community participation, and social engagement they desire. Adults with a range of adaptive functioning skills can benefit from case management, and the more significant the person's adaptive functioning deficits, the more intensive and comprehensive the case

management needs to be. Adults of any age can benefit from comprehensive case management and ideal services will follow an individual through their lifespan.

Clients of comprehensive case management may have a range of diagnoses in addition to their below average IQ scores. They may have autism, cerebral palsy, a brain injury, special health care needs, or genetic disorders, to name a few. Conversely, they may have no official diagnosis, but a history of educational and adaptive functioning deficits with no known etiology, other than an IQ score in the borderline range.

Treatment Procedures

While this section is titled “Treatment Procedures” for consistency within the publication, comprehensive case management should not be considered treatment. Rather, it is a system of support and communication that comes together underneath an individual in order to help them rise up to their potential. The professional coordinating an individual’s case management must foster a culture of collaboration with the individual, their family members, and their other providers that supports their self-determination, bolsters their independence, and encourages them to make their own choices.

Comprehensive case management might entail weekly meetings with a client, or may be more or less frequent depending on what the client wants and needs. Some clients may only need relatively short-term intervention, for example, help finding new health insurance when they can no longer be on a family member’s, or may need long-term services if their goals are more complicated. A client with complex goals, fewer state, federal, and nonprofit supports, less family support, or multiple diagnoses may need long-term case management that helps them manage issues and pursue goals in multiple domains and settings throughout their lives. Services should always be highly individualized in their frequency, duration, and in the level of hands-on assistance provided by the case manager.

Organizations offering comprehensive case management should be prepared to follow clients throughout their lives; this reduces the number of transitions for the individual, helps with building provider/client rapport, and streamlines long-term planning efforts and the preparation for later life. Within organizations, providers who are leaving should take care to transition clients thoughtfully to new case managers.

Services must be fully accessible to each individual client. Some common accommodations include:

- Emailing or calling the client to remind them of appointments
- Communicating by the client’s preferred method (email, phone, text, instant messaging, etc.)
- Providing physically accessible meeting spaces
- Travel training to help the client learn how to get to and from the case manager’s office or service location
- Communicating directly with family members, with the client’s permission
- Converting any documents the client needs into accessible formats

Case managers should refer their clients out for specialized services beyond their expertise or licensure, such as psychotherapy and medical care, but should coordinate the connection between the client and the specialist to whatever degree the client requires; this may mean setting up a first appointment together, showing the individual how to get to the specialist, or even traveling to a first appointment with the client if necessary and desired by the individual. Of course, the case manager must receive permission from the client when communicating with any other parties about their case management or other coordinated services. As much as possible, clients should be guided in advocating for themselves with family members and providers, and case managers should avoid speaking for them except when clients request it and when it is

absolutely necessary for clear communication. Instead, case managers can help clients develop scripts for communicating their needs to others, make phone calls together on speaker phone, and attend meetings together, if such support is necessary.

Efficacy Information

There is no body of literature specifically on the efficacy of comprehensive case management in improving outcomes for adults with BIF. However, data from the Lesley University Threshold Program, which serves young adults with BIF, suggests that 2 years of case management in the context of a college-based postsecondary program may contribute to positive employment, social, and independent living outcomes (Lesley University 2015). Graduates of the Threshold program have a paid employment rate of 79% and work an average of 31 hours per week. Threshold program alumni may choose to continue participating in comprehensive case management throughout their lives, provided by the program.

The efficacy of comprehensive case management varies depending on the experience, persistence, and flexibility of the case manager, the motivation of the client, the complexity of the goals being pursued, and the level of other supports available to the client. A highly efficacious case manager will ensure that communication with the client is through a method comfortable and convenient to the client, will be quickly responsive to the client, and will reach out to the client if there is any concern that they need additional supports in order to effectively utilize the case management.

Outcome Measurement

Individual outcomes should be measured by setting goals for case management, directed by the client, and checking-in on those goals together at predetermined times or as necessary. For many clients, comprehensive case management can be

deintensified or even phased out over time as the client develops the skills to manage a wider range of responsibilities on their own.

Qualifications of Treatment Providers

Providers will typically have a background in human services, social work, mental health counselling, special education, vocational rehabilitation, or a related field. Providers need broad knowledge of the educational and adult service systems available to people with disabilities. Depending on the organization through which case management is offered, additional licensure or education may be required.

See Also

- [DSM-5](#)
- [DSM-5 and Autism Spectrum Disorder](#)
- [Higher Education Opportunity Act of 2008 \(HEOA\)](#)
- [Learning Disability](#)

References and Reading

- Christensen, D. L., Baio, J., Braun, K. V. N., Bilder, D., Charles, J., Constantino, J. N., et al. (2016). Prevalence and characteristics of autism spectrum disorder among children aged 8 years — Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2012. *Morbidity and Mortality Weekly Report Surveillance Summaries*, 65(3), 1–23. <http://dx.doi.org/10.15585/mmwr.ss6503a1>.
- Gigi, K., Werbeloff, N., Goldberg, S., Portuguese, S., Reichenberg, A., Fruchter, E., & Weiser, M. (2014). *European Neuropsychopharmacology*, 24(11), 1793–1797.
- Jankowska, A. (2016). Towards a framework for psychological resilience in children and adolescents with borderline intellectual functioning. *Polish Psychological Bulletin*, 47(3), 289–299.
- Jankowska, A., Takagi, A., Bagdanowicz, M., & Jonak, J. (2014). Parenting style and locus of control, motivation, and school adaptation among students with borderline intellectual functioning. *Current Issues in Personality Psychology*, 2(4), 251–266.
- Lesley University (2015). *Annual Threshold alumni survey: Executive summary*. Lesley University.

- Peltopuro, M., Ahonen, T., Kaartinen, J., Seppala, H., & Narhi, V. (2014). Borderline intellectual functioning: A systematic literature review. *Intellectual and Developmental Disabilities*, 52(6), 419–443.
- Rao, P., Raman, V., Thomas, T., & Ashok, M. (2015). IQ in autism: Is there an alternative global cognitive index? *Indian Journal of Psychological Medicine*, 37, 48.
- Shattuck, P., Roux, A., Hudson, L., Taylor, J., Maenner, M., & Trani, J. (2012). Services for adults with an autism spectrum disorder. *Canadian Journal of Psychiatry*, 57(5), 284–291.
- Turcotte, P., Mathew, M., Shea, L. L., Brusilovskiy, E., & Nonnemacher, S. L. (2016). Service needs across the lifespan for individuals with autism. *Journal of Autism and Developmental Disorders*, 46, 2480–2489.
- Wieland, J., & Zitman, F. G. (2016). It is time to bring borderline intellectual functioning back into the main fold of classification systems. *British Journal of Psychiatry Bulletin*, 40, 204–206.

BOS

- [Behavior Observation Scale](#)

BOT-2

- [Bruininks-Oseretsky Test of Motor Proficiency](#)

BOTMP

- [Bruininks-Oseretsky Test of Motor Proficiency](#)

Bound Morphemes

- [Speech Morphology](#)

Brachmann-de Lange Syndrome

- [Cornelia de Lange Syndrome](#)

Brain Connectivity Theories of Autism

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Synonyms

[Functional connectivity](#); [Functional integration](#); [Network coherence](#)

Definition

Brain connectivity refers to both structural connections between distinct regions of the brain as well as coordinated functional activity within networks of different brain regions, which may or may not share direct structural connections. Structural and functional connectivity in the brain are interrelated in that altered structural connections can affect functional coordination within brain networks and altered functional activity can affect structural connections via adaptive changes from synaptic pruning and dendritic arborization. Although there is extensive evidence to support altered structural connectivity in the brain in individuals with autism (for a specific example, see ► [“Corpus Callosum Abnormalities in Autism”](#)), most brain connectivity theories of autism spectrum disorder (ASD) focus on differences in functional connectivity (FC), defined as “temporal correlations between spatially remote neurophysiological events” (Friston et al. 1993). In this regard, FC is most often measured by comparing correlations between brain regions for fluctuations of the blood oxygen-level-dependent (BOLD) response from fMRI data (see Functional Magnetic Resonance Imaging) that is collected during passive rest (no stimulus) or during the completion of a cognitive processing task. Accumulating

evidence from this line of research suggests that the development of typical FC patterns in the brain are altered in individuals with ASD. For instance, early investigations reported general patterns of global hypoconnectivity (under-connectivity) in individuals with ASD (Just et al. 2004), but subsequent studies have indicated more complex patterns of both hypo- and hyper-connectivity across different brain regions and networks. As such, general patterns of global (long-range) hypoconnectivity coupled with local (short-range) hyperconnectivity have also been proposed (Wass 2011). However, these theories oversimplify much more complex alterations of brain connectivity in individuals with ASD, especially regarding the development of FC over time. Models of optimized brain organization exhibit robust FC between neighboring brain regions with some additional long-range connections to more distant regions in order to minimize the metabolic cost of information processing. Consistent with this model, maturational changes of brain networks typically involve functional segregation between non-contributing anatomical neighbors with concurrent integration of more distant brain regions that contribute to the processing of domain-specific information (Fair et al. 2009). Conceptualized within this developmental framework, emerging theories of brain connectivity in ASD suggest that information processing is affected by disruption of the maturation and adaptive development of functional integration within as well as segregation between brain networks (Rudie et al. 2013).

More recent research into disruptions of brain connectivity in ASD have reported some promising results, but the well-known heterogeneity in the etiology, neurobiology, and symptomatology of ASD across individuals suggests that there is most likely not a unique or defining brain connectivity pattern. It is also not yet clear whether FC differences contribute directly to the pathogenesis of the disorder or only emerge as secondary features associated with altered cognitive and behavioral performance. Overall, brain connectivity appears to be altered in individuals with ASD with patterns of both under-connectivity and

over-connectivity depending on the brain region or network that is being evaluated (Abbott et al. 2016). Such alterations may reduce functional network integration to affect cognitive and behavioral processing in some of the core and comorbid domains. However, the substantial variability in reports of FC alterations across individuals coupled with methodological concerns regarding fMRI data processing (Müller et al. 2011) presents major considerations and limitations for previous FC studies in ASD. Brain connectivity will need to be evaluated in larger samples with sufficient power to subgroup individuals with ASD. These subgroups should be compared to individuals with other neuropsychiatric disorders as well as typically developing controls in order to determine whether any ASD-specific alterations in brain connectivity actually exist and elucidate their potential relationship with differences in the pathogenesis of the disorder.

See Also

- ▶ [Corpus Callosum Abnormalities in Autism](#)
- ▶ [Functional Magnetic Resonance Imaging](#)
- ▶ [Neural Signatures of Treatment Response](#)

References and Reading

- Abbott, A. E., Nair, A., Keown, C. L., Datko, M., Jahedi, A., Fishman, I., & Müller, R.-A. (2016). Patterns of atypical functional connectivity and behavioral links in autism differ between default, salience, and executive networks. *Cerebral Cortex*, 26(10), 4034–4045. <https://doi.org/10.1093/cercor/bhv191>.
- Fair, D. A., Cohen, A. L., Power, J. D., Dosenbach, N. U. F., Church, J. A., Miezin, F. M., et al. (2009). Functional brain networks develop from a “local to distributed” organization. *PLoS Computational Biology*, 5(5), e1000381.
- Friston, K. J., Frith, C. D., Liddle, P. F., & Frackowiak, R. S. J. (1993). Functional connectivity: The principal-component analysis of large (PET) data sets. *Journal of Cerebral Blood Flow & Metabolism*, 13(1), 5–14. <https://doi.org/10.1038/jcbfm.1993.4>.
- Just, M. A., Cherkassky, V. L., Keller, T. A., & Minshew, N. J. (2004). Cortical activation and synchronization during sentence comprehension in high-functioning autism: Evidence of underconnectivity. *Brain*, 127(8), 1811–1821.

- Müller, R. A., Shih, P., Keehn, B., Deyoe, J. R., Leyden, K. M., & Shukla, D. K. (2011). Underconnected, but how? A survey of functional connectivity MRI studies in autism spectrum disorders. *Cerebral Cortex*, 21, 2233–2243.
- Rudie, J. D., Brown, J. A., Beck-Pancer, D., Hernandez, L. M., Dennis, E. L., Thompson, P. M., et al. (2013). Altered functional and structural brain network organization in autism. *NeuroImage: Clinical*, 2(0), 79–94. <https://doi.org/10.1016/j.nicl.2012.11.006>.
- Wass, S. (2011). Distortions and disconnections: Disrupted brain connectivity in autism. *Brain and Cognition*, 75(1), 18–28.

traditional tests of hearing sensitivity, and therefore, the ABR may be completed to establish hearing sensitivity.

See Also

- Auditory Acuity
- Auditory Brainstem Response (ABR)
- Brainstem Auditory Evoked Potentials
- Hearing

Brainstem Audiometry

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Synonyms

[Auditory Brainstem Response \(ABR\)](#)

Definition

Brainstem audiometry, sometimes called a brainstem auditory evoked response (BAER) or an auditory brainstem response (ABR), is an electrophysiologic test that assesses the auditory system through the low brainstem. This test can assess hearing sensitivity in individuals who cannot respond to traditional testing; thus, it is often used in newborn hearing screenings and in populations that are nonverbal. The ABR is completed by placing recording electrodes on the individual's head and ears and placing earphones in their ears. Responses are elicited using click and tonal stimuli which are delivered through the earphones. Five waveforms are typically present in the ABR (waves I, II, III, IV, and V); however, wave V is the waveform used for threshold testing. Individuals with autism spectrum disorders might not be able to consistently respond to

References and Reading

- Hall, J. (1992). *Handbook of auditory evoked responses*. Needham Heights: Allyn & Bacon.
- Rosenblum, S. M., Arick, J. R., Krug, D. A., Stubbs, E. G., Young, N. B., & Pelson, R. O. (1980). Auditory brainstem evoked responses in autistic children. *Journal of Autism and Developmental Disorders*, 10, 215–225.
- Rosenhall, U., Nordin, V., Sandstrom, M., Ahlsen, G., & Gillberg, C. (1999). Autism and hearing loss. *Journal of Autism and Developmental Disorders*, 29(5), 349–357.
- Skoff, B. F., Mirsky, A. F., & Turner, D. (1980). Prolonged brainstem transmission time in autism. *Psychiatry Research*, 2, 157–166.
- Skoff, B. F., Fein, D., McNally, B., Lucci, D., Humes-Bartlo, M., & Waterhouse, L. (1986). Brainstem auditory evoked potentials in autism. *Psychophysiology*, 23, 462.

Brainstem Auditory Evoked Potentials

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Synonyms

[Auditory brainstem response, ABR](#); [Brainstem auditory evoked response, BAER](#)

Definition

Brainstem Auditory Evoked Potentials (BAEP) are low-amplitude electrical voltage potentials in the brain that are evoked by a sound, often a click, and recorded using electrodes on the scalp (i.e., electroencephalography or EEG). Since the potentials typically have an amplitude around 1 mV, hundreds of trials are averaged together to provide data with adequate signal to noise ratio. BAEPs have been used as a tool to examine brainstem integrity and hearing ability clinically since the 1970s, helping to diagnose tumors and other diseases such as multiple sclerosis. BAEPs are particularly useful with patients who are difficult to test by traditional audiometry, where feedback is required, because of compromised levels of consciousness, limited communication, or behavioral noncompliance. BAEPs are also useful for detecting subtle changes that may be clinically relevant, and localizing the deficits.

The BAEP reflects the function of the auditory nerve (eighth nerve), cochlear nucleus, superior olive, and inferior colliculus, measuring the time it takes an aural stimulus to travel through the auditory pathway in the brainstem. They are thought to measure action potentials and postsynaptic activity propagating along the auditory nerve, and to other regions along the auditory pathway. When measured in response to a brief stimulus (typically a click) in the ear canal via an inserted earphone or headphone, the elicited waveform response is detected by surface electrodes placed at the base of the scalp and the ear lobes. The BAEP generally includes seven waves, but it is the initial five waves that have been the most extensively studied and are useful for clinical applications. These five waves occur within 6 or 7 ms and are labeled waves I through V. Abnormalities in specific waves are informative, as they are thought to localize the differences to particular parts of the auditory pathway. Wave I is believed to reflect the distal auditory nerve on the ipsilateral side; wave II, proximal auditory nerve on the ipsilateral side; wave III, ipsilateral cochlear nucleus; wave IV, superior olivary nucleus and adjacent brainstem regions bilaterally; and wave V, distal lateral lemniscus and inferior colliculus on the contralateral

side. Interpretation of the BAEP is routinely done by examining the latency and length of these waves and the interpeak intervals between them (IPI), also known as interpeak latencies (IPL). The amplitudes are less frequently interpreted because they are more variable across individuals, and thought to be less reliable indices of dysfunction. The IPIs are considered particularly important, reflecting the conduction times in the auditory pathway through the brainstem (i.e., auditory nerve then cochlear nerve, cochlear nucleus, and lateral lemniscus). Therefore, longer IPIs are thought to indicate impaired function, possibly related to the number, synchronicity, or integrity of the neurons firing in these regions.

The BAEP is relatively adult-like by 18 months of age, though wave V may continue to mature until 3 or 4 years of age. They differ in females and males, with a slightly shorter latency and higher amplitude in females. More recently, the BAEP has been examined in response to a tone (instead of a click), which is thought to measure dysfunction more specific to the cochlear regions.

See Also

- ▶ [Auditory Brainstem Response, ABR](#)
- ▶ [Auditory Potentials](#)
- ▶ [Brainstem Auditory Evoked Response, BAER](#)
- ▶ [Evoked Potentials](#)
- ▶ [Visual Evoked Potential \(VEP\)](#)
- ▶ [Visual/Somatosensory Cognitive Potentials](#)

References and Reading

- Legatt, A. D., Arezzo, J. C., & Vaughan, H. G., Jr. (1988). The anatomic and physiologic bases of brain stem auditory evoked potentials. *Neurologic Clinics*, 6(4), 681–704.
- Moore, J. K., & Lithicum, F., Jr. (2007). The human auditory system: A timeline of development. *International Journal of Audiology*, 46(9), 460–478.
- Starr, A., & Anchor, L. J. (1975). Auditory brain stem responses in neurological disease. *Archives of Neurology*, 32, 761–768.
- Stone, J. L., Calderon-Arnulphi, M., Watson, K. S., Patel, K., Mander, N. S., Suss, N., et al. (2009). Brainstem auditory evoked potentials—a review and modified studies in healthy subjects. *Journal of Clinical Neurophysiology*, 26(3), 167–175.

Brainstem Auditory Evoked Response (BAER)

► Auditory Brainstem Response (ABR)

Brainstem Auditory Evoked Response, BAER

► Brainstem Auditory Evoked Potentials

Brainstem Auditory Evoked Responses in Autism (BAERs)

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Definition

BAERs (brainstem auditory evoked responses; also referred to as brainstem auditory evoked potential, BAEPs, and auditory brainstem response, ABR) measure the electrical voltage potentials in the proximal auditory pathway in response to a noise. This is done via electrodes on the scalp and earlobe (see also definition: ► “Brainstem Auditory Evoked Potentials”). The noise is most frequently a click, but tones and other sounds have also been used (e.g., Russo et al. 2008). BAERs are thought to reflect the function of the auditory pathway through the brainstem, providing insight into both the level of hearing and the integrity of brainstem function in a given individual. When the noise is a click, BAEPs produce seven waves of activity. The first five of these – labeled waves I through V – have been well characterized, with wave V followed by a negative dip (Stone et al. 2009). These initial five waves occur within about 7 ms. The waves are thought to reflect activation progressing as the

aural stimulus moves from more distal regions of the auditory nerve to the more proximal regions. Examining the length of the waves and the latencies between them (the interpeak intervals: IPIs, also known as interpeak latencies IPL) provides insight into whether there is dysfunction along the auditory pathway through the brainstem and, potentially, helps to localize that dysfunction. Waves I, III, and V have been particularly well characterized. Wave I is thought to be generated peripherally, at the auditory or cochlear nerve; wave III at the cochlear nuclei; and wave V at the lateral lemniscus. These signals go from the ipsilateral side (waves I to III in the auditory nerve, cochlear nucleus, and superior olive) to bilateral brainstem regions (wave IV) to contralateral regions (wave V in the lateral lemniscus and inferior colliculus). The wave structure develops an adult-like architecture in the first few years of life, with maturation starting in more peripheral regions (with waves I and III maturing in the first year) and moving to more central regions (with wave V maturing at 3–4 years of age; Fujikawa-Brooks et al. 2010; Moore and Linthicum 2007).

Historical Background

Since sensory modulation is disrupted in ASD, with both under- and over-reactivity to sounds, early theories posited that auditory brainstem function might be affected in ASD (Ornitz et al. 1985; modified in Ornitz 1987). To empirically study this possibility, BAERs were used, examining the integrity of this region and the claim of atypical brainstem function in ASD. Early work on autism in the 1970s and early 1980s was promising, suggesting that there may be abnormalities in BAERs in individuals with ASD. A problem, however, was that what aspect of BAERs actually differed in ASD was not consistent across studies (Klin 1993). In addition, BAERs do not require attention or consciousness, making them useful for testing special populations; however, this fact also led to a very heterogeneous sample being tested in many of these early studies. Some of the participants had known neurological conditions (Klin 1993; Minshew 1991), and in some

studies, many individuals had hearing loss (e.g., Taylor et al. 1982), which create an obvious confound when interpreting these studies. Gender has been shown to affect BAERs, with shorter latencies in women. Therefore, gender also has to be considered since a greater proportion of women in the control group could lead to spurious group differences. Indeed, the conclusion that BAERs were abnormal in ASD was disputed in the mid-1980s by work suggesting that the differences reported in the early studies reflected participant characteristics other than ASD (e.g., other neurological disorder, intellectual disability). Courchesne et al. (1985) tested a cohort of high-functioning individuals with ASD, with well-matched controls, and found no differences in the group with ASD. Once the issues discussed above were taken into account – and the reliability of the measures, as methods were still improving – several reviews argued that differences in individuals with ASD were not evident (Minshew 1991) or less likely (Klin 1993). Klin (1993) pointed out that, while BAERS did not provide convincing evidence of brainstem dysfunction in ASD, they did suggest that peripheral hearing loss might be common in ASD and such hearing loss would be important clinically when treating those with ASD. Tables listing the results and the samples used in these earlier studies are included in Klin (1993) and Wong and Wong (1991).

Current Knowledge

More work has led to further inconsistencies in the data, though several important themes have emerged. In all studies, differences in the BAERs of those with ASD are evident in a subset of participants with ASD and, in some cases, their first-degree relatives (Maziade et al. 2000). This indicates that, while abnormal BAERs are not causal, they may reflect a subgroup which would be important to identify clinically (Nagy and Loveland 2002). Thus, there is still potential for abnormal BAERs to be a biomarker for at least a subset of individuals with ASD, providing insight into the disorder. In addition, what is atypical in the BAERs of the individuals with ASD has

differed both within and across studies, suggesting that there may be multiple ways to disrupt the auditory pathway through the brainstem. These disruptions generally present as prolongations of the waves or IPIs, when they are evident. Nagy and colleagues argue that some of these disruptions may be specific to ASD (e.g., prolongation of waves III to V; on the basis of Bachevalier 1996), while others might be evident in a number of disorders (e.g., speech impairment, ADHD: prolongation of waves I to III) and are potentially related to differences in language acquisition (Nagy and Loveland 2002). In general, it is not clear whether even the differences that have been identified in ASD are specific to this disorder. However, these differences do not generalize to *all* developmental disorders. While individuals with Down's syndrome also display abnormal BAERS, the atypical patterns are distinct from those in autism (Sersen et al. 1990). Finally, abnormalities may have implications clinically, as recent work suggests that there may be some experience-dependent plasticity in the BAER wave pattern that is sensitive to auditory training (Chandrasekaran and Kraus 2010; Skoe and Kraus 2010; see Russo et al. 2010 for training in ASD).

The studies in recent years have shown a prolongation of either the wave itself or – relatedly – the IPI (Gillberg et al. 1983; Kwon et al. 2007; Maziade et al. 2000; Rosenhall et al. 2003; Tanguay et al. 1982; Tas et al. 2007; Wong and Wong 1991), though a few early studies indicated a shortening of waves (see Table 1 in Rosenhall et al. 2003 for a summary of earlier studies). Other conditions, such as Down's syndrome, may tend to exhibit shorter IPIs (Sersen et al. 1990). This longer latency is evident in a subset of those with ASD, generally not more than about 50% of the sample. Which wave (I, III, or V) or IPI the group differences are evident differs between studies; however, wave V appears to be most often affected, especially in the left (L) ear. (See Table 1 for a summary of recent results since 2000 to click tones in BAERs.) This may reflect a more general slowing of auditory processing that differs across this heterogeneous population. This pattern is also evident in many earlier studies.

Brainstem Auditory Evoked Responses in Autism (BAERs), Table 1 Recent literature on BAERs in ASD in response to clicks (see Russo et al. 2008; Tharpe et al.

2006 for recent evidence of differences in BAERs to other sounds, but not to clicks)

Study	N	Potential confounds?	Prolongation?	Prolongation? IPIs I–III, III–V
			Latencies I, III, V	
Tas et al. (2007)	N = 30 ASD N = 15 controls M age = 3 year	Individuals with ASD sedated controls not	None	III–V
Rosenhall et al. (2003)	N = 101 ASD with normal hearing N = 59 controls M age = 8 year	Controls slightly older than individuals with ASD	I, V	III–V
Fujikawa-Brooks et al. (2010)	N = 20 ASD N = 20 controls M age = 10 year		V in L ear	None
Maziade et al. (2000)	N = 73 ASD, 251 relatives N = 521 controls M age = 7 year		Not reported	Longer I–III and I–V in ASD and first-degree relatives especially siblings
Magliaro et al. (2010)	N = 16 ASD N = 25 controls M age = 12 year	Many more F in control group (16 vs. 1)	III, V	I–III, I–V
Kwon et al. (2007)	N = 71 ASD (22 autism) N = 50 controls M age = 3 year	May have had other medical issues, 2 cases with brainstem abnormalities	V in L ear	I–V, III–V bilateral in larger more heterogeneous ASD group only

B

Skoff and colleagues reported prolonged III–V IPIs in the L ear in 33% of their sample (1980). Thivierge et al. found that 80% of their populations had longer I–V and III–V IPIs (1990). Wong and Wong (1991) reported increased latencies of wave V, and I–III, III–V, and I–V IPIs, in sedated individuals with autistic “features,” but not in those with intellectual disability. Later studies (summarized in Table 1) reported longer IPIs I–III in both individuals

with ASD and their first-degree family members (Maziade et al. 2000). However, 52% of the families with ASD had normal BAEPs in everyone in the family. Rosenhall et al. (2003) reported that 58% of children with ASD had longer latencies in waves I and V and IPI in III–V. This study included a large sample, but a portion of the sample had hearing loss. Kwon et al. (2007) reported longer I–V and III–V and wave V in large group of those on the spectrum (ASD)

($N = 71$), but not in those with autism defined more strictly ($N = 22$). The take-home message from Kwon and colleagues was that ASD might have a lot of physiological overlap with central auditory processing disorder (CAPD), on the basis of the ABR results, and that this comorbidity might have clinical implications. In contrast to these positive results, several studies have reported no difference between groups to click stimuli (Courchesne et al. 1985; Rumsey 1984; Tharpe et al. 2006).

Most of the studies do not have a well-matched control group (but see Courchesne et al. 1985), although many of the recent ones do a test for hearing impairment before including participants in the results. In addition, since BAERs are thought to be relatively resistant to age, function level, or other potential confounds such as the effects of sedation, these differences may generally not affect the results or do so only subtly. However, in these studies, there are still issues with the control groups. One such issue is gender. Since females have shorter IPIs, including too many in the control group could bias the IPIs to be shorter in controls and therefore appear longer in ASD. For instance, Magliaro et al. (2010) found prolongation in III and V and IPIs I–III and I–V, but this study included a substantial proportion of females in the control group. Recent studies have attempted to control for gender (Russo et al. 2008), since there are almost always a few more females in the control group, and have found differences. Another issue is that a number of subjects with serious hearing loss *and* ASD have been identified across studies (Rosenhall et al. 2003; Tas et al. 2007). This is an important issue clinically, as it may not be immediately evident in children with ASD that they have hearing loss (Klin 1993). So, while this emphasizes the importance of examining hearing in those with ASD, it also presents confounds in the available data. For instance, Tas (2007) reported a longer III–V bilaterally in young children with ASD. However, five children were identified as having hearing loss, and, while the three with severe loss were excluded, the two with mild hearing loss were not. This study also brings up the issue of using quite

young children, around 2 years old (see also Kwon et al. 2007; Wong and Wong 1991). While the BAER architecture is relatively mature by 18 months of age, there is some evidence that wave V continues to mature until around 3 or 4 years old. While age was approximately matched in many of the studies, differential development across groups may still be influencing the results.

Several studies have examined the BAER response to sounds other than clicks, and these results suggest that group differences might be more likely with sounds other than with the traditional click response. Russo et al. (2008) examined pitch encoding. They found that 20% of children on the autism spectrum had difficulty with pitch, while none showed abnormal BAERs to click sounds, but this result was not correlated with language outcome. The ASD group had more boys and lower IQ, but the results did not change when these issues were controlled statistically. Tharpe et al. (2006) found differences in the BAER when the stimulus was a pure tone, but not when it was a click. This difference was evident in 11 of 22 individuals with ASD. Fujiwaka-Brooks and colleagues (2010) included more clicks per second (61–91 instead of 11–25 used typically), a stressor that is known to lead to longer latencies typically, especially in wave V. These investigators found differences in left ear only, with a trend for latency of wave I and significant results of wave V. They also report a negative correlation between the latency of wave V and verbal IQ, suggesting a relationship between this wave and language skill. About half the sample showed the difference in the L ear for wave V. This group points out the importance of testing from both ears, as some studies have only tested the right ear.

Future Directions

These studies indicate that BAERs *may* be abnormal in ASD, but this is unlikely to reflect important information about etiology across the spectrum. These abnormal BAERs may reflect disrupted auditory processing, possibly deep in

the brainstem. There is not convincing evidence that it is specific to ASD. However, that differences are evident for only a subset of participants with ASD might prove useful for identifying subgroups of ASD. In addition, differences in the developmental pattern in ASD have not been studied but may be enlightening. While TD individuals may show little change in the BAERs after age 4 or due to intellectual disability, this pattern may not be true of those with ASD. Such developmental differences could help explain the discrepancy between the findings of Courchesne et al. (1985) with a high-functioning set of adults with ASD and well-matched controls and the more recent work that generally focuses on children, often very young ones (Kwon et al. 2007; Tas et al. 2007; Wong and Wong 1991). In addition, recent studies have begun to identify plasticity in the BAER in the auditory pathway in the brainstem when training takes place (Chandrasekaran and Kraus 2010; Skoe and Kraus 2010; see Russo et al. 2010 for studies in ASD), and high-functioning individuals with ASD may be able to compensate for their social and communication issues and engage further through language. This plasticity may help to explain the variability in the BAER differences in ASD, in addition to other differences across the spectrum, as well as help to inform potential interventions.

See Also

- Auditory Brainstem Response, ABR
- Auditory Potentials
- Brainstem Auditory Evoked Response, BAER
- Evoked Potentials
- Visual Evoked Potential (VEP)
- Visual/Somatosensory Cognitive Potentials

References and Reading

- Bachevalier, J. (1996). Brief report: Medial temporal lobe and autism: A putative animal model in primates. *Journal of Autism and Developmental Disorders*, 26(2), 217–220.
- Chandrasekaran, B., & Kraus, N. (2010). The scalp-recorded brainstem responses to speech: Neural origins and plasticity. *Psychophysiology*, 47(2), 236–246.
- Courchesne, E., Courchesne, R. Y., Hicks, G., & Lincoln, A. J. (1985). Functioning of the brain-stem auditory pathway in non-retarded autistic individuals. *Electroencephalography and Clinical Neurophysiology*, 61(6), 491–501.
- Fujikawa-Brooks, S., Isenberg, A. L., Osann, K., Spence, M. A., & Gage, N. M. (2010). The effect of rate stress on the auditory brainstem response in Autism: A preliminary report. *International Journal of Audiology*, 49, 129–140. <https://doi.org/10.3109/14992020903289790>.
- Gillberg, C., Rosenhall, U., & Johansson, E. (1983). Auditory brainstem responses in childhood psychosis. *Journal of Autism and Developmental Disorders*, 13(2), 181–195.
- Klin, A. (1993). Auditory brainstem responses in autism: brainstem dysfunction or peripheral hearing loss. *Journal of Autism and Developmental Disorders*, 23, 15–35.
- Kwon, S., Jungmi, K., Choe, B., Ko, C., & Park, S. (2007). Electrophysiologic assessment of central auditory processing by auditory brainstem responses in children with Autism spectrum disorders. *Journal of Korean Medical Science*, 22, 656–659.
- Magliaro, F. C., Scheuer, C. I., Assumpcao, F. B., & Matas, C. G. (2010). Study of auditory evoked potentials in Autism. *Pro-Fono Revista de Atualizacao Cientifica*, 22, 31–37.
- Maziade, M., Merette, C., Cayer, M., Roy, M., Szatmari, P., Cote, R., et al. (2000). Prolongation of brainstem auditory – Evoked responses in Autistic probands and their unaffected relatives. *Archives of General Psychiatry*, 57, 1077–1083.
- Minshew, N. J. (1991). Indices of neural function in Autism: Clinical and biological implications. *Pediatrics*, 87, 774–780.
- Moore, J. K., & Linthicum, F. H. (2007). The human auditory system: A time-line of development. *International Journal of Audiology*, 46(9), 460–478.
- Nagy, E., & Loveland, K. A. (2002). Prolonged brainstem auditory evoked potentials: and autism specific or autism-nonspecific marker. *Archives of General Psychiatry*, 59(3), 288–290.
- Ornitz, E. M., Atwell, C. W., Kaplan, A. R., & Westlake, J. R. (1985). Brain-stem dysfunction in autism. Results of vestibular stimulation. *Archives of General Psychiatry*, 42(10), 1018–1025.
- Rosenhall, U., Nordin, V., Brantberg, K., & Gillberg, C. (2003). Autism and auditory brain stem responses. *Ear and Hearing*, 24, 206–214. <https://doi.org/10.1097/01.AUD.0000069326.11466.7E>.
- Rumsey, J. M. (1984). Auditory brainstem responses in pervasive developmental disorders. *Biological Psychiatry*, 19(10), 1403–1418.
- Russo, N. M., Skoe, E., Trommer, B., Nicol, T., Zecker, S., Brdlow, A., et al. (2008). Deficient brainstem encoding

- of pitch in children with Autism spectrum disorders. *Clinical Neurophysiology*, 119, 1720–1731. <https://doi.org/10.1016/j.clinph.2008.01.108>.
- Russo, N., Zecker, S., Trommer, B., Chen, J., & Kraus, N. (2009). Effects of background noise on cortical encoding of speech in Autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 1185–1186.
- Russo, N. M., Hornickel, J., Nicol, T., Zecker, S., & Kraus, N. (2010). Biological changes in auditory function following training in children with Autism spectrum disorders. *Behavioral and Brain Function*, 16, 6–60. <https://doi.org/10.1186/1744-9081-6-60>.
- Sersen, E. A., Heaney, G., Clausen, J., Belser, R., & Rainbow, S. (1990). Brainstem auditory-evoked responses with and without sedation in Autism and Down's syndrome. *Biological Psychiatry*, 27, 834–840.
- Skoe, E., & Kraus, N. (2010). Auditory brain stem response to complex sounds: A tutorial. *Ear and Hearing*, 31, 302–324.
- Stone, J. L., Calderon-Arnulphi, M., Watson, K. S., Patel, K., Mander, N. S., Suss, N., et al. (2009). Brainstem auditory evoked potentials – a review and modified studies in healthy subjects. *Journal of Clinical Neurophysiology*, 26, 167–175.
- Tanguay, P. E., Edwards, R. M., Buchwald, J., Schwafel, J., & Allen, V. (1982). Auditory brainstem evoked responses in autistic children. *Archives of General Psychiatry*, 39(2), 174–180.
- Tas, A., Yagiz, R., Tas, M., Esme, M., Uzun, C., & Karasalioglu, A. R. (2007). Evaluation of hearing in children with Autism by using TEOAE and ABR. *Autism*, 11, 73–79. <https://doi.org/10.1177/1362361307070908>.
- Taylor, M. J., Rosenblatt, B., & Linschoten, L. (1982). Auditory brainstem response abnormalities in Autistic children. *Canadian Journal of Neurological Sciences*, 9, 429–433.
- Tharpe, A. M., Bess, F. H., Sladen, D. P., Schissel, H., Couch, S., & Schery, T. (2006). Auditory characteristics of children with Autism. *Ear and Hearing*, 27, 430–441.
- Tzounopoulos, T., & Kraus, N. (2009). Learning to encode timing: Mechanisms of plasticity in the auditory brainstem. *Neuron*, 62, 463–469.
- Wong, V., & Wong, S. N. (1991). Brainstem auditory evoked potential study in children with Autistic disorder. *Journal of Autism and Developmental Disorders*, 21, 329–340.

Brainstem Evoked Response (BER)

► Evoked Potentials

Brazil and Autism

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Services and Social Policies

In Brazil, Autism Spectrum Disorder (ASD) is considerably understudied. A single epidemiological study has been conducted, assessing children from one city in the southeast. Results indicated an estimated prevalence of 27.2/10,000, which is believed to be an underestimation due to methodological issues (Paula et al. 2011). If the international prevalence 1% is true in Brazil, approximately 1.5–2 million Brazilian children are affected by ASD.

The National Health System (SUS), still in constant construction, also has shortcomings. Although a national system, it is organized in different ways around the country. The SUS recommends that children with autism have comprehensive care of their needs. Health care occurs primarily in units of ambulatory medical care (AMAS) and psychosocial care centers (CAPS). The Brazilian health system is composed by the primary care for simple demands (Basic Health Units – UBS, Health Nuclei for Family Support – NASF, and Family Health Units – USF), secondary care (CAPS – Outpatient Mental Health) for more difficult cases without need of hospitalization, and tertiary care for hospitalizations and emergency room needs (General Hospitals). It is sure that this network needs to be improved: the CAPS only recently started to assist children with autism in a more regular way. There are some welfare agency in Brazil that plays an important role in the diligence of autism patients and families, such as ABRA – Associação Brasileira de Autismo brings together corporations related to autism in Brazil and represents the interests of Autism patients in different national councils such as the health, social, and the rights of the

individual council; AMA – Associação de Amigos do Autista; and the APAE – Associação de Pais e Amigos dos Excepcionais, and they have an established center in most Brazilian states. They offer different activities for autism patients related to socialization, day care activities, and communication; AUMA – Associação dos Amigos da Criança Autista – since 1990 has the major objective of developing educational programs of social adaptation for autism patients and families.

In São Paulo, the richest state in the country, the health system is designed in a way that the Basic Health Units (UBS), teams of the Family Health Strategy (family physicians, nurses, and dentists), with support from the Family Assistance Center (NASF) are the first place where cases arrive to make diagnosis and also less-severity cases are kept for treatment. The instruments for early identification and diagnosis should be present in established practice in these centers, which unfortunately does not happen. The CAPS is responsible for the establishment of a therapeutic project and is the reference for the UBS (secondary care, in day hospital care scheme, more intensive, with intermediate function between the outpatient and inpatient) and when faced with cases of high complexity, CAPS can trigger other places in the network, as services belonging to the university or references to specific service for ASD. São Paulo is the unique state that has a specific CAPS for adults with autism.

Regarding education, children with autism should be included in regular education (inclusive education), which should provide suitable conditions for integration and development. Teachers and school staff should receive adequate training to work with children with autism. In cases that regular education is not possible given the intensity of symptoms and difficulty of the student to adapt, the option is a special school. Getting a place in special school is very difficult, and parents often have to appeal to justice to have their rights guaranteed. In Brazil, people with autism have all the rights provided for in specific laws for people with disabilities as well as international standards signed by Brazil, such as United Nation Convention on the Rights of

Persons with Disabilities. Moreover, children and adolescents also have all the rights stated in the Statute of Children and Adolescents and the elderly, i.e., over 60 years old, have also the rights of the elderly. People with autism have also the special protection of Federal Law, which ensures proper treatment in public and private health facilities for the specific pathology they have. If the person with autism is demonstrably in need, he/she is entitled to a free pass in state and interstate transportation. The law, establishing the National Policy on Protection of Rights for Persons with Autism Spectrum Disorder, was published in the Official Newspaper in Brazil in December 2012 giving support and emphasis on rights and proper treatment, as well as access to education and to vocational teaching, housing, labor market, and social security and welfare. In cases of proven need, the person with autism spectrum disorder, included in common classes in regular education, will be entitled to have a specialized companion during the activities done in the school. Among the points set out in the Law is community participation in the formulation of public policies for people with autism, in addition to implementation, monitoring, and evaluation of the person with autism. In addition to these duties, the benefit of greater importance to the disabled person and therefore to person with autism is the Continuous Cash Benefit, a social assistance benefit which was regulated by the Organic Law of Social Assistance – LOAS. To get the benefit, the family income must be less than one fourth of the minimum wage, and proof of disability and level of temporary or permanent disability for independent life and work must be attested by medical and social expertise of the INSS. In the next section, we will explore diagnosis and treatment options for autism with an emphasis on research performed in Brazil and the Brazilian limitations.

Instruments for Diagnosis and Standard Scales in Brazil

The official diagnosis of ASD in Brazil follows the ICD-10 criteria (WHO 1993), performed by

Brazil and Autism, Table 1 Translated and validated instruments for screening and diagnosis of Autism Spectrum Disorders

Description of instruments		
<i>The Autistic Traits of Evaluation Scale (ATA)</i>	Authors	Ballabriga et al. 1994
	Proposal	Scale of screening based on observation
	Age of administration	Over 2 years
	Reliability	Favors tracking the evolution of the disease
	Validation for Brazil	Assumpção et al. 1999
<i>Autism Behavior Checklist (ABC)</i>	Authors	Krug et al. 1980
	Proposal	Direct observation and interview with parents and caregivers for screening
	Age of administration	Over 18 months
	Reliability	Identifies autism in both clinical and educational contexts
	Validation for Brazil	Marteletto and Pedromônico 2005
<i>Childhood Autism Rating Scale (CARS)</i>	Authors	Schopler et al. 1986
	Proposal	Assessment scale for observation of behavior for screening
	Age of administration	Over 24 months
	Reliability	High degree of internal consistency and reliability
	Validation for Brazil	Pereira and Wagner 2008
<i>Autism Screening Questionnaire (ASQ)</i>	Authors	Berument et al. 1999
	Proposal	Self-administration questionnaire for parents and caregivers for screening
	Age of administration	Over 6 years
	Reliability	Favors the large-scale use for screening of suspected cases of autism
	Validation for Brazil	Sato et al. 2009
<i>Autism Diagnostic Interview (ADI-R)</i>	Authors	Lord et al. 1994
	Proposal	Semi-structured interviews with parents or guardians for diagnosis and research on autism
	Age of administration	Over 18 months
	Reliability	Validated and reliable instrument for diagnosis of (GDD** TGD) for ASD in preschool aged
	Validation for Brazil	Becker et al. 2012

interviews held with parents and caregivers and by clinical observation of the child. To assist in the diagnostic process, several scales and interviews were validated to Portuguese. Table 1 shows the translated and validated scales and instruments for use in Brazil: The ATA (Escala d'Avaluació dels Trests Autistes), ABC (Autism Behavior Checklist), M-CHAT (Modified Checklist for Autism in

Toddlers), and the ASQ (Autism Screening Questionnaire) are used for screening, while the CARS (Childhood Autism Rating Scale) and ADI-R (Autism Diagnostic Interview-reviewed) are used for diagnosis.

The ATA (Autistic Traits of Evaluation Scale) was the first scale to be translated and adapted in Brazil by Assumpção Jr. et al. The translation of

the ABC made by Marteleto and Pedromônico in 2005 added the direct observation of the child as well as interviews with parents and caregivers. The M-CHAT (Modified scale for screening autism) was translated and adapted to Portuguese in Brazil by Mirella Fiuza Losapio and Milena Pereira Pondé 2008 creating the possibility of early screening, which could be done in public health at the primary care level. The questionnaire for children older than 6 years has been translated and validated in Brazil by Sato et al. in 2009. But, in Brazil there is still no study comparing the use of these scales in our social reality, and it would be important to establish protocols for screening.

The CARS is a scale that helps identify children with autism and it distinguishes from children with developmental impairment and without autism. Its importance is to differentiate mild-moderate autism from severe the CARS has two versions. One observational and one held with parents or guardians. The scale for parents was translated and validated in 2007 in Brazil (Riesgo and Wagner 2008). The ADI has been translated into 11 languages, and it is cited as the gold standard for diagnosis of autism. In Brazil, the translation and validation of the ADI-R (Becker et al. 2012) has recently been published.

It is important to note the CARS alone does not indicate diagnosis (Riesgo and Wagner 2008), it must be used together with the DSM IV diagnostic criteria for autism. Santos et al. (2012) concluded that the CARS fails in the diagnosis of some cases of autism, while the ABC may result in *overdiagnosis*, and it is best to combine the use of both. Corroborating studies show that instruments used in isolation may not be sufficient, and it is important to use at least one questionnaire with parents and an observation scale with the kid. The administration of the ADI-R is complex and lengthy requiring trained professionals to such situations that are nonexistent in our society, even the little time availability of the ADI-R for use in Brazil.

In an attempt to use instruments already validated and translated and applied in Brazilian clinical practice, Duarte et al. (2003) examined the validity of the CBCL/4-18 (Child Behavior

Checklist) for the identification of autism spectrum disorders in the Brazilian population. The CBCL was validated for the administration to the Brazilian population by Bordin et al. (1995). As Albores-Gallo et al. (2008) pointed out, the CBCL is an important instrument to assess the most frequent comorbidities in autism spectrum disorders, such as attention problems, depression, and anxiety, and not automatically valid for diagnosis.

Some Brazilian researchers have been working in the development of national scales for fast and easy administration for early detection of autistic symptoms (children younger than 18 months). The Clinical Indicators of Risk for Child Development (IRDI) was developed in Brazil, and it has the ability to detect a trend of occurrence of problems expressed in the first 18 months of life that extends throughout the development of the child at least until the sixth year, generating impact on the quality of life of the child (Kupfer et al. 2010). They are currently working on the validation of the IRDI for large-scale administration in Brazil, in partnership with the Brazilian government. Braido (2006) reported different behaviors and properties of behaviors (e.g., response latency) that already showed signs of delay in the development of babies (1 year old) who were lately diagnosed with autism. Following this line of research to identify behaviors, Bagaiolo et al. (2010) used the description of the child development from 0 to 3 years in order to analyze home videos of a baby who was lately diagnosed with autism (diagnosed with 3 years of age). Different failures in aspects of child development, before 3 years of age, were analyzed for association with autism risk. This work is still in progress requiring replication.

Regarded as the gold standard for diagnosis, the *Autism Diagnostic Observation Scale* – ADOS (Lord et al. 1994, 2000), ADOS-G has been already translated (personal communication by Maria Clara Pacifico, Cristiane S de Paula, and Guiomar Oliveira 2012) but has not yet been published or validated. The limitation of trained human resources for its administration also prevents the use of the ADOS-G in clinical practice.

Neuropsychological and Speech Pathology Evaluation

In Brazil, several instruments are used for neuropsychological and speech pathology assessment, and there is no standardized protocol. The use of Wechsler Intelligence Scale for Children-III, Wechsler Adult Intelligence Scale-III, and ADAPTATIVE BEHAVIOR SCALES OF VINELAND are recommended for assessment for autism in Brazil. From the standpoint of speech pathology, adapted scales and instruments to the Brazilian society are ABFW tests – Child language test in the areas of phonology, vocabulary, fluency, and pragmatics (Andrade et al. 2004); PPVT– Peabody Picture Vocabulary Test (Capovilla and Capovilla 1997); Language Exam (Braz and Pellicciotti 1988); the Evaluation of Symbolic Maturity (Befi-Lopes et al. 2000); and the language development evaluation (Menezes 2004).

Genetic Evaluation in Brazilian Clinical Practice

In terms of familial and genetic studies conducted in Brazil, we emphasized the study by Mecca et al. (2011) that aimed to track the occurrence of signs and symptoms of ASD in siblings of individuals with this diagnosis. The data indicate higher rates than those reported in the literature (2–6 %) and approach the findings that report 10 % of familial recurrence in dizygotic twins. This result provides evidences of possible neurogenetic factors to explain the occurrence of ASD in relatives of studied probands and emphasizes the need to make the tracking of this disorder in children who are not only evaluated but also their brothers. Brazil is beginning to contribute to genetic studies in the field of ASD and has adequate technology to conduct them. In the last 5 years (2008–2013), more than 11 genetic studies in patients with ASD were made in Brazil, most of which were case studies and some association studies, and case–control or family studies (simplex or multiplex) (Kuczynski et al. 2009; Griesi-Oliveira et al. 2012; Mulatinho et al. 2012;

Baruffi et al. 2012; Griesi-Oliveira et al. 2012; Christofolini et al. 2012; Longo et al. 2009; Castro et al. 2010; Orabona et al. 2009; Campos Junior et al. 2011). Although the Brazilian researches have made some contributions in the genetics of ASD, there is no panel for gene diagnosis in the Brazilian population yet, and the genetic evaluation is not part of the systematic evaluations in private clinics or public ones.

Overview of Current Treatments and Research Related to Treatment Strategies

In Brazil, it is recommended to follow the interventions followed by ABA (Applied Behavior Analysis), TEACCH (Treatment and Education of Autistic and related Communication handicapped Children), and PECS (Picture Exchange Communication System) in order to maximize language acquisition and improve communication skills. All the *AMA – Associação de Amigos do Autista* – in most of Brazilian regions offer different activities mainly based on TEACCH and PECS. Due to the high cost of professional training and the difficulty of administering such interventions in Brazil, it is needed that the research related to modifications of these therapies being either in time, performance in groups, or frequency. Below we present examples of work groups of Brazilian researchers studying single pre-evaluations for appropriateness of inclusion of children at different levels of PECS, time of administration of it as well as the speech pathology therapies in individual or in pairs.

The PECS is a simple and portable equipment, especially produced to ensure that communication with autistic children occurs in spite of their speech difficulties, but previous studies (Flippin et al. 2010) reported that many failures in the *PECS* training may be due to the fact that the child has not acquired all conditional discrimination training required. So before you start training the *PECS*, it is important to assess whether the individual already has such discrimination. In 2003, Guilhardi compared the performance of participants in *ABLA (Assessment of Basic*

Learning Abilities) and conditional discrimination training and equivalence testing. The author aimed to investigate whether the ABLA test results could predict the performance of children with typical development and atypical development (autism, cerebral palsy, congenital syndromes, etc.) in tasks involving the same type of stimulus. Guilhardi (2003) concluded that the predictive power of the ABLA for training equivalence does not happen to all participants and also there were no differences in predictive ability between types of conditional discrimination tests. In Brazil, Godoi et al. (2008) made a study of two cases to investigate the effectiveness of PECS in terms of assessing the amount of training necessary to acquire the skills involved in functional communication exchanging pictures. The authors measured also some side effects of PECS training, i.e., the interference of training on the frequency and appropriate verbalizations and about learning other behaviors specific to each participant. Parallel to the PECS training, additional training for other behaviors was done. These additional training involved differential reinforcement and *fading out* physical tips. As a side effect of this training, Godoi et al. (2008) pointed out the increasing frequency of verbalizations of both groups of participants. Furthermore, according to the authors, the structured and concrete features of the training contributed to the increased frequency of specific behaviors of each participant and independence in an everyday activity. Aiming to understand even more specifically the effects of picture exchange communication on language acquisition, Guilhardi (2009) investigated the functional independence of tacts and mands between verbal responses based on selection of stimuli (PECS). According to the author, the results showed functional independence between operant tacts and mands with verbal responses based on the selection of stimuli.

Cardoso and Fernandes (2006) and Fernandes et al. (2008) did studies with children and adolescents with psychiatric diagnosis within the autistic spectrum at the beginning of the process of speech pathology therapy and they were divided into three groups (individual language

therapy, child in a group with a coordinator, child in a group without a coordinator) according to the therapeutic intervention received for a period of 6 months; the results indicated no statistically significant differences between the groups; however, the group with most progress during the specific period of differentiated intervention was the one where the individuals were treated with a coordinator. The most interesting was that in none of the groups decrease in the levels of improvement obtained after a period of 6 months was observed, and in some situations, the number of individuals with improvement increased after this period. The results of this study reinforce the adequacy of procedures for determining the individual profile of abilities and disabilities of each individual as a basis for definitions regarding to the adopted intervention model.

In terms of pharmacological treatments, not all medications are available in the SUS and there are restrictive conditions according to diagnosis. For example, the majority of atypical antipsychotics are only offered to individuals with the diagnosis of schizophrenia. Besides, we do not have proper clinical trials performed in our country to examine the use of medications in autism. Despite methodological limitations, the study of Novaes et al. (2008) concluded that pharmacological interventions with second-generation antipsychotics seemed useful for the control of behavioral disorders such as psychomotor agitation and aggressive behavior in a sample of Brazilian patients with autistic spectrum disorders corroborating worldwide studies in the area.

Priorities and Future Directions

A systematic review of the Brazilian scientific literature on ASD showed a significant increase in scientific production in this subject over the last 2 years (Teixeira et al. 2010). On the other hand, it showed that most publications are not focused on subjects that can contribute significantly to the improvement of public health relating to autism in Brazil. Most publications make references to intervention studies without controls and small

convenience samples, and the use of validated diagnostic and neuropsychosocial instruments are still required.

Another intriguing finding is the extreme concentration of scientific production in only two regions of the country. Researchers from São Paulo and Rio Grande do Sul are the first authors in 90 % of papers published between 2002 and 2009 (Teixeira et al. 2010). In a recent editorial, De Paula et al. 2011 list the barriers and difficulties as well as the priority areas for investment in autism in Brazil. Some identified challenges include lack of specific funding to support autism research, lack of national multicenter projects, lack of trained researchers and clinicians in various disciplines, lack of robust scientific studies, and lack of campaigns to increase knowledge and understanding by the general public and professionals in education and health. The priorities identified for response to the challenges encountered are by (De Paula et al. 2011).

1. Research areas – to build capacity of research in various disciplines through programs of research training and training of clinicians, including diagnostic skills and tools for early detection and interventions
2. Awareness – to raise public understanding and improve public perception of autism by making information more accessible to the public
3. Services – to build capacity for services in Brazil among professionals and in community-based settings, to establish training program for screening early detection and intervention strategies

All these activities are very important to improve the understanding and assistance of autism in Brazil.

References and Reading

- Albore-Gallo, L., Hernandez-Guzman, L., Diaz-Pichardo, J. A., & Cortes-Hernandez, B. (2008). Dificultades en la evaluación y diagnóstico del autismo: Una discusión. *Salud Mental [online]*, 31(1), 37–44.
- American Psychiatry Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed., pp. 72–73). Washington, DC: American Psychiatry Association.
- American Psychiatry Association. (2002). *Manual diagnóstico e estatístico de transtornos mentais – DSM-IV-TR* (4th ed.). Porto Alegre: Artmed.
- Andrade, C. R. F., Befi-Lopes, D. M., Fernandes, F. D., & Wetzner, H. F. (2004). *ABFW: Teste de linguagem infantil*. São Paulo: Pró-Fono.
- Assumpção Júnior, F. B., Kuczyński, E., Gabriel, M. R., & Rocca, C. C. (1999). Validity and reliability of a scale for the assessment of autistic behaviour. *Arquivos de Neuro-Psiquiatria*, 57(1), 23–29.
- Autism Speaks. (2013). acessado em 20 de março, 2013. Disponível em <http://www.autismspeaks.org/what-autism/learn-signs>
- Ballabriga, M. C. J., Escudé, R. M. C., & Llaberia, E. D. (1994). Escala d'avaluació d'altres autistes (A.T.A.): validez y fiabilidad de una escala para el examen de las conductas autistas. *Revista de Psiquiatria Infanto-Juvenil*, 4, 254–263.
- Baranek, G. T. (2002). Efficacy of sensory and motor interventions of children with autism. *Journal of Autism and Developmental Disorders*, 32(5), 397–422.
- Baruffi, M. R., Souza, D. H., Silva, R. A. B., Ramos, E. S., & Moretti-Ferreira, D. (2012). Autism spectrum disorder in a girl with a De Novo X;19 balanced translocation. *Case Reports in Genetics*, 2012, Article ID 578018.
- Becker, M. M., Wagner, M. B., Bosa, C. A., Schmidt, C., Longo, D., Papaleo, C., & Riesgo, R. S. (2012). Translation and validation of Autism Diagnostic Interview-Revised (ADI-R) for autism diagnosis in Brazil. *Arquivos de Neuro-Psiquiatria*, 70(3), 185–190.
- Befi-Lopes, D. M., Takiuchi, N., & Araújo, K. (2000). Avaliação da maturidade simbólica nas alterações de desenvolvimento da linguagem. *JBF*, 1(3), 6–15.
- Berument, S. K., Rutter, M., Lord, C., & Pickles, A. (1999). Autism screening questionnaire: Diagnostic validity. *British Journal of Psychiatry*, 175, 444–451.
- Bildt, S., Ketelaars, C., Kraijer, D., Mulder, E., & Volkmar, F. (2004). Interrelationship between ADOS-G, ADI-R and DSM-IV classification. *Journal of Autism and Developmental Disorders*, 34, 129–138.
- Bohlander, A. J., Orlich, F., & Varley, C. K. (2012). Social skills training for children with autism. *Pediatric Clinics of North America*, 59(1), 165–174.
- Bondy, A., & Frost, L. (2002). *The picture exchange communication system: Training manual*. Newark: Pyramid Educational Products.
- Bordin, I. A. S., Mari, J. J., & Caeiro, M. F. (1995). Validação da versão brasileira do Child Behavior Checklist (CBCL) – Inventário de Comportamentos da Infância e da Adolescência: dados preliminares. *Revista ABP-APAL/Associação Brasileira de Psiquiatria – Asociación Psiquiátrica de la América Latina*, 17, 55–66.
- Braido, M. L. G. (2006). *Identificação precoce dos Transtornos do Espectro Autista: Um estudo de vídeos familiares*. Dissertação de Mestrado. Departamento de

- Psicologia – Pontifícia Universidade Católica do Rio de Janeiro.
- Braz, H. A., & Pellicciotti, T. H. F. (1988). *Exame de linguagem TIPITI* (3rd ed.). São Paulo: MNJ.
- Bristot Silvestrin, R., Bambini-Junior, V., Galland, F., Daniele Bobermim, L., Quincozes-Santos, A., Torres Abib, R., Zanotto, C., Batassini, C., Brolese, G., Gonçalves, C. A., Riesgo, R., & Gottfried, C. (2013). Animal model of autism Induced by prenatal exposure to valproate: Altered glutamate metabolism in the hippocampus. *Brain Research*, 1495, 52–60.
- Cabezas, H. (2005). A criança com autismo: um programa estruturado para sua educação. In: V. E. Caballo & M. A. Simón (Eds.), *Manual de Psicologia Clínica Infantil e do Adolescente: transtornos específicos* (Cap. 13 – pp. 321–345). São Paulo: Santos.
- Caminha, R. C., & Lampreia, C. (2012). Findings on sensory deficits in autism: Implications for understanding the disorder. *Psychology & Neuroscience*, 5(2), 231–237.
- Canitano, R., & Scandurra, V. (2011). Psychopharmacology in autism: An update. *Progress in Neuropsychopharmacology & Biological Psychiatry*, 35(1), 18–28.
- Capovilla, F. C., & Capovilla, A. G. S. (1997). Desenvolvimento lingüístico da criança dos dois aos seis anos: tradução e padronização do Peabody Picture Vocabulary Test de Dunn&Dunn e da Language-DevelopmentSurvey de Rescorla. *Ciência Cognitiva: Teoria, Pesquisa e Aplicação*, 1(1), 53–380.
- Cardoso, C., & Fernandes, F. D. (2006). Relation between social cognitive aspects and the functional communicative profile in a group of adolescents of the autistic spectrum. *Pro Fono*, 18(1), 89–98.
- Carter, A. C., Volkmar, F. R., Sparrow, S. S., Wang, J. J., Lord, C., Dawson, G., Fombonne, E., Loveland, K., Mesibov, G., & Schopler, E. (1998). The Vineland adaptable scales: Supplementary norms for individuals with autism. *Journal of Autism and Developmental Disorders*, 28(4), 287–302.
- Case-Smith, J., & Arbesman, M. (2008). Evidence-based review of interventions for autism used in or of relevance to occupational therapy. *American Journal of Occupational Therapy*, 62(4), 416–429.
- Castro, F., Barrera, M., & Steiker, L. (2010). Issues and challenges in the design of culturally adapted evidence-based interventions. *Annual Review of Clinical Psychology*, 6, 213–239.
- Centers for Disease Control and Prevention. (2012). Autism spectrum disorders (ASDs): Data & statistics. Acessado em 26 de fevereiro, 2013. Disponível em <http://www.cdc.gov/NCBDDDD/autism/data.html>
- Chawarska, K., Klin, A., & Volkmar, F. R. (2008). *Autism spectrum disorders in infants and toddlers: Diagnosis, assessment and treatment*. New York/London: Guilford.
- Christofolini, D. M., Meloni, V. A., Ramos, M. A. P., Oliveira, M. M., de Mello, C. B., Pellegrino, R., Takeno, S. S., & Melaragno, M. I. (2012). Autistic disorder phenotype associated to a complex 15q intrachromosomal rearrangement. *American Journal of Medical Genetics. Part B*, 159B, 823–828.
- Cohen, D. J., Caparulo, B. K., Gold, J. R., Waldo, M. C., Shaywitz, B. A., Rutenber, B. A., et al. (1978). Agreement in diagnosis, clinical assessment and behavior rating scales for pervasively disturbed children. *Journal of the American Academy of Child Psychiatry*, 17(4), 589–603.
- Constantino, J. N. (2002). *The social responsiveness scale*. Los Angeles: Western Psychological Services.
- Cotugno, A. J. (2009). Social competence and social skills training and intervention for children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(9), 1268–1277.
- Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., Donaldson, A., & Varley, J. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The Early Start Denver Model. *Pediatrics*, 125(1), 17–23.
- De Paula, C. S., Fombonne, E., Gadia, C., Tuchman, R., & Rosanoff, M. (2011). Autism in Brazil – perspectives from science and society. *Revista da Associação Médica Brasileira*, 57(1), 2–5.
- Duarte, C. S., Bordin, I. A. S., de Oliveria, A., & Bird, H. (2003). The CBCL and the identification of children with autism and related conditions in Brazil: Pilot findings. *Journal of Autism and Developmental Disorders*, 33, 703–707.
- Eaves, R. C. (1990). The factor structure of autistic behavior. In *Annual Alabama conference on autism*, Birmingham.
- Eaves, R. C., & Hooper, J. (1988). A factor analysis of psychotic behavior. *Journal of Special Education*, 21(4), 122–132.
- Eldevik, S., Hastings, R. P., Hughes, J. C., Jahr, E., Eikeseth, S., & Cross, S. (2009). Meta-analysis of early intensive behavioral intervention for children with autism. *Journal of Clinical Child and Adolescent Psychology*, 38(3), 439–450.
- Fernandes, F. D. M. (2000). Prova de Pragmática. In C. R. F. Andrade, D. M. Béfi-Lopes, F. D. M. Fernandes, & H. F. Wertzner (Eds.), *Teste ABFW: Teste de linguagem infantil nas áreas de fonologia, vocabulário, fluência e pragmática*. São Paulo: Profono.
- Fernandes, F. D., Cardoso, C., Sassi, F. C., Amato, C. L., & Sousa-Morato, P. F. (2008). Language therapy and autism: Results of three different models. *Pro Fono*, 20(4), 267–272.
- Figueira, S. A. C. M., Souza, I. C. N., Rios, V. G., & Benguigui, Y. (2005). *Manual para vigilância do desenvolvimento infantil no contexto da AIDP-I. Organização Pan-Americana da Saúde*. Washington, DC: OPAS.
- Flippin, M., Reszka, S., & Watson, L. R. (2010). Effectiveness of the picture exchange communication system (PECS) on communication and speech for children with autism spectrum disorders: A meta-analysis.

- American Journal of Speech Language Pathology*, 19(2), 178–195.
- Fombonne, E. (2003). Epidemiological surveys of autism and other pervasive developmental disorders: An update. *Journal of Autism and Developmental Disorders*, 33(4), 365–382.
- Freeman, B. J., Ritvo, E. R., Yokota, A., & Ritvo, A. (1986). A scale for rating symptoms of patients with the syndrome of autism in real life settings. *Journal of the American Psychiatric Association*, 25(1), 130–136.
- Fuentes, J., Bakare, M., Munir, K., Aguayo, P., Gaddour, N., Öner, Ö., & Mercadante, M. (2012). Autism spectrum disorders. In J. M. Rey (Ed.), *IACAPAP e-textbook of child and adolescent mental health*. Geneva: International Association for Child and Adolescent Psychiatry and Allied Professions.
- Ganz, J. B., Davis, J. L., Lund, E. M., Goodwyn, F. D., & Simpson, R. L. (2012). Meta-analysis of PECS with individuals with ASD: Investigation of targeted versus non-targeted outcomes, participant characteristics, and implementation phase. *Research in Development Disabilities*, 33(2), 406–418.
- Gilliam, G. J. E. (1995). *Autism rating scale*. Austin: ProEd.
- Godoi, A. P., Fidalgo, J. P., & Goia, P. S. (2008). *Análise alternativa funcional comunicação's procedure (pictureexchange communication system)*. *Revista brasileira de terapia comportamental cognitiva* 10(1). São: Paulo jun.
- Griesi-Oliveira, K., Moreira, D. P., Davis-Wright, N., Sanders, S., Mason, C., Orabona, G. M., Vadasz, E., Bertola, D. R., State, M. W., & Passos-Bueno, M. R. (2012). A complex chromosomal rearrangement involving chromosomes 2, 5, and X in autism spectrum disorder. *American Journal Medicine Genetic Part B*, 159B, 529–536.
- Il, R. C. W. (1995). Global assessment of functioning: A modified scale. *Psychosomatics*, 36(3), 267–275.
- Guilhardi, H. (2003). O uso de instrumentos padronizados de avaliação comportamental nas sessões de terapia. Recuperado em 10 de Agosto de 2010, de: www.terapiaporcontingencias.com.br/pdf/helio/Uso_instrumentos.pdf
- Guilhardi, H. J. (2009). Terapia por contingências de reforçamento (TCR). Recuperado 29 dezembro, 2100 de http://www.terapiaporcontingencias.com.br/pdf/helio/Terapia_reforcamento2009.pdf
- Howlin, P., Magiati, I., Charman, T., & MacLean, W. E. (2009). Systematic review of early intensive behavioral interventions for children with autism. *American Journal on Intellectual and Developmental Disabilities*, 114(1), 23–41.
- Kerr, M., & Flora, J. (1977). The measurement of motor, visual and auditory discrimination skills in mentally retarded children and adults and in young normal children. *Rehabilitation Psychology*, 24(3), 91–206.
- Kirsten, T. B., Chaves-Kirsten, G. P., Chaible, L. M., Silva, A. C., Martins, D. O., Britto, L. R., Dagli, M. L., Torráo, A. S., Palermo-Neto, J., & Bernardi, M. M. (2012). Hypoactivity of the central dopaminergic system and autistic-like behavior induced by a single early prenatal exposure to lipopolysaccharide. *Journal of Neuroscience Research*, 90(10), 1903–1912.
- Krug, D. A., Arick, J. R., & Almond, P. J. (1980). *Autism screening instrument for educational planning*. Portland: ASIEP Educational.
- Kuczynski, E., et al. (2009). Infantile autism and 47, XYY karyotype. *Arquivos de Neuro-Psiquiatria São Paulo*, 67(3a).
- Kupfer, M. C. M., et al. (2010). Valor preditivo de indicadores clínicos de risco para o desenvolvimento infantil: um estudo a partir da teoria psicanalítica. *Revista Latinoamericana de Psicopatologia Fundamental*, 13(1).
- Le Couteur, A., Lord, C., & Rutter, M. (2003). *Autism diagnostic interview-revised (ADI-R)*. Los Angeles: Western Psychological Services.
- Lerner, R. (2011). *Indicadores Clínicos de Risco para desenvolvimento Infantil – IRDI: verificação da capacidade discriminativa entre autismo, retardo mental e normalidade*. Tese de livre-docência, IP-USP.
- Longo, D., Schuler-Faccini, L., Brandalize, A. P., dos Santos Riesgo, R., & Bau, C. H. (2009). Influence of the 5-HTTLPR polymorphism and environmental risk factors in a Brazilian sample of patients with autism spectrum disorders. *Brain Research*, 1267, 9–17.
- Lord, C., & Corsello, C. (2005). Diagnostic instruments in autistic spectrum disorders. In F. R. Volkmar (Ed.), *Handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.
- Lord, C., Rutter, M. L., & Le Couteur, A. (1994). The autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(5), 659–685.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., DiLavore, P. C., et al. (2000). The autism diagnostic observation schedule-generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30(3), 205–223.
- Losapio, M. F., & Pondé, M. P. (2008). Tradução para o português da escala M-CHAT para rastreamento precoce de autismo. *Revista de Psiquiatria do Rio Grande do Sul*, 30(3), 221–229.
- Luteijn, E., Luteijn, F., Jackson, S., Volkmar, F., & Minderaa, R. (2000). The children's social behavior questionnaire for milder variants of PDD problems: Evaluation of the psychometric characteristics. *Journal of Autism and Developmental Disorders*, 30(4), 317–330.
- Magiati, I., Moss, J., Yates, R., Charman, T., & Howlin, P. (2011). Is the Autism Treatment Evaluation Checklist a useful tool for monitoring progress in children with autism spectrum disorders? *Journal of Intellectual Disability Research*, 55(3), 302–312.

- Marteletto, M. R., & Pedromônico, M. R. (2005). Validade do Inventário de Comportamentos Autísticos (ICA): estudo preliminar. *Revista Brasileira de Psiquiatria*, 27(4), 295–301.
- Matson, M. L., Mahan, S., & Matson, J. L. (2009). Parent training: A review of methods for children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 3(4), 868–875.
- Matson, J. L., Rieske, R. D., & Tureck, K. (2011). Additional considerations for the early detection and diagnosis of autism: Review of available instruments. *Research in Autism Spectrum Disorders*, 5(4), 1319–1326.
- McConachie, H., & Diggle, T. (2007). Parent implemented early intervention for young children with autism spectrum disorder: A systematic review. *Journal of Evaluation in Clinical Practice*, 13(1), 120–129.
- Mecca, T. P., et al. (2011). Rastreamento de sinais e sintomas de transtornos do espectro do autismo em irmãos. *Revista de Psiquiatria do Rio Grande do Sul Porto Alegre*, 33(2).
- Menezes, M. L. N. (2004). Avaliação do desenvolvimento da linguagem [ADL]. *Manual Do Examinador*.
- Mesibov, G. B., Schaffer, B., & Shopler, E. (1988). *Adolescent and adult psychoeducational profile*. Austin: Pro-Ed.
- Ministries of Health and Education. (2008). New Zealand autism spectrum disorder guideline. Wellington: Ministry of Health. Acessado em 15, dezembro de 2012, disponível em <http://www.health.govt.nz/publication/new-zealand-autism-spectrum-disorder-guideline>
- Miral, S., Gencer, O., Inal-Emiroglu, F. N., Baykara, B., Baykara, A., & Dirik, E. (2008). Risperidone versus haloperidol in children and adolescents with AD: A randomized, controlled, double-blind trial. *European Child & Adolescent Psychiatry*, 17(1), 1–8.
- Missouri Department of Mental Health – Division of Developmental Disabilities. (2012). Missouri autism guidelines initiative. Autism spectrum disorders: Guide to evidence-based interventions. Acessado em 28 fevereiro, 2011. Disponível em <http://www.autismguidelines.dmh.mo.gov/documents/Interventions.pdf>
- Mulatinho, M. V., et al. (2012). Severe intellectual disability, omphalocele, hypospadias and high blood pressure associated to a deletion at 2q22.1q22.3: Case report. *Molecular Cytogenetics*, 5, 30.
- Myers, S. M., & Johnson, C. P. (2007). Management of children with autism spectrum disorders. *Pediatrics*, 120(5), 1162–1182.
- National Collaborating Centre for Women's and Children's Health. (2011). *Autism: Recognition, referral and diagnosis of children and young people on the autism spectrum* (Nice clinical guideline, 296 pp). Acessado em 28 fevereiro, 2011. Disponível em <http://www.nice.org.uk/nicemedia/live/13572/56424/56424.pdf>
- New York State Department of Health (NYSDH) – Early Intervention Program. (1999). Clinical practice guideline: Quick reference guide. Autism/pervasive developmental disorders, assessment and intervention for young children (Age 0–3 Years). Publication No. 4216. 108 pp.
- Novaes, C. M., Pondé, M. P., & Freire, A. C. C. (2008). Control of psychomotor agitation and aggressive behavior in patients with autistic disorder. A retrospective chart review. *Arquivos de Neuro-Psiquiatria*, 66(3), 646–651.
- Organização Mundial da Saúde [OMS]. (1993). *Classificação de transtornos mentais e de comportamento da CID-10: descrições clínicas e diretrizes diagnósticas*. Porto Alegre: Artes Médicas.
- Paula, C. S., Ribeiro, S. H., Fombonne, E., & Mercadante, M. T. (2011). Brief report: Prevalence of pervasive developmental disorder in Brazil: A pilot study. *Journal of Autism and Developmental Disorders*, 41(12), 1738–1742.
- Pereira, A. R. R., & Wagner, M. B. A. L. (2008). Childhood autism: Translation and validation of the Childhood Autism Rating Scale for use in Brazil. *Jornal de Pediatria*, 84, 487–494.
- Pondé, M. P., Novaes, C. M., & Losapio, M. F. (2010). Frequency of symptoms of attention deficit and hyperactivity disorder in autistic children. *Arquivos de Neuro-Psiquiatria*, 68(1), 103–106.
- Reichow, B. (2012). Overview of meta-analyses on early intensive behavioral intervention for young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(4), 512–520.
- Reichow, B., & Volkmar, F. R. (2010). Social skills interventions for individuals with autism: Evaluation for evidence-based practices within a best evidence synthesis framework. *Journal of Autism and Developmental Disorders*, 40(2), 149–166. <http://www.ncbi.nlm.nih.gov/pubmed/19655240>
- Ribeiro, S. H. (2007). *Prevalência dos transtornos invasivos do desenvolvimento no município de Atibaia: um estudo-piloto* [dissertação]. São Paulo: Universidade Presbiteriana Mackenzie.
- Rimland, B. (1968). On the objective diagnosis of infantile autism. *International Journal of Child Adolescent Psychiatry*, 35, 146–161.
- Ruttenberg, B. A., Dratman, M. L., Fraknoi, J., & Wenar, C. (1966). An instrument for evaluating autistic children. *Journal of the American Academy of Child Psychiatry*, 5, 453–478.
- Rutter, M. (2005). Autism research: Lessons from the past and prospects for the future. *Journal of Autism and Developmental Disorders*, 35(2), 241–257.
- Santos, T. H., Barbosa, M. R., Pimentel, A. G., Lacerda, C. A., Balestro, J. I., Amato, C. A., & Fernandes, F. D. (2012). Comparing the use of the Childhood Autism Rating Scale and the autism behavior checklist protocols to identify and characterize autistic individuals. *Jornal da Sociedade Brasileira de Fonoaudiologia*, 24(1), 104–106.
- Sato, F. P., Paula, C. S., Lowenthal, R., Nakano, E. Y., Brunoni, D., Schwartzman, J. S., et al. (2009). Instrumento para rastreamento dos casos de transtorno invasivo do desenvolvimento: estudo preliminar de

- validação. *Revista Brasileira de Psiquiatria*, 31(1), 30–33.
- Sato, J. R., Hoexter, M. Q., Oliveira, P. P., Jr., Brammer, M. J., MRC AIMS Consortium, Murphy, D., & Ecker, C. (2013). Inter-regional cortical thickness correlations are associated with autistic symptoms: A machine-learning approach. *Journal of Psychiatry Research*, 47(4), 453–459.
- Schopler, E., & Reichler, R. J. (1979). *Individualized assessment and treatment for autistic and developmentally disabled children: Psychoeducational profile*. Baltimore: University Park Press.
- Schopler, E., Reichler, R. J., & Renner, B. R. (1986). *The Childhood Autism Rating Scale (CARS) for diagnostic screening and classification of autism*. New York: Irvington Publishers.
- Scott, J. B. (2006). *American occupational therapy association fact sheet: Occupational therapy's role with autism*. Bethesda: American Occupational Therapy Association.
- Shaffer, D., Gould, M. S., Brasic, J., Ambrosini, P., Fisher, P., Bird, H., & Aluwahlia, S. (1983). A children's global assessment scale (CGAS). *Archives of General Psychiatry*, 40(11), 1228–1231.
- Sparrow, S. S., Balla, D. A., Cicchetti, D. V., & Doll, E. A. (1984). *Vineland adaptive behavior scales: Interview edition, survey form manual*. Circle Pines: American Guidance Service.
- Teixeira, M. C. T., Meca, T., Velloso, R., Ribeiro, S. H., Mercadante, M. T., et al. (2010). Brazilian scientific production about autism spectrum disorders. *Revista da Associação Médica Brasileira*, 56, 607–614.
- Torrão, A. S., Palermo-Neto, J., & Bernardi, M. M. (2012). Hypoactivity of the central dopaminergic system and autistic-like behavior induced by a single early prenatal exposure to lipopolysaccharide. *Journal of Neuroscience Research*, 90(10), 1903–1912.
- Virués-Ortega, J. (2010). Applied behavior analytic intervention for autism in early childhood: Meta-analysis, meta-regression and dose-response meta-analysis of multiple outcomes. *Clinical Psychology Review*, 30(4), 387–399.
- Volkmar, F. R. (1992). Childhood disintegrative disorder: Issues for DSM-IV. *Journal of Autism and Developmental Disorders*, 22(4), 625–642.
- Wechsler, D. (2002). *WISC-III – Escala de inteligência Wechsler para crianças. Adaptação brasileira V. L. M. Figueiredo*. São Paulo: Casa do Psicólogo.
- Wechsler, D. (2004). *WAIS-III – Escala de inteligência Wechsler para adultos. Adaptação brasileira E. Nascimento*. São Paulo: Casa do Psicólogo.
- White, S. W., Keonig, K., & Scahill, L. (2007). Social skills development in children with autism spectrum disorders: A review of the intervention research. *Journal of Autism and Developmental Disorders*, 37(10), 1858–1868.
- Wing, L., Leekam, S. R., Libby, S. J., Gould, J., & Larcombe, M. (2002). The diagnostic interview for social and communication disorders: Background, inter-rater reliability and clinical use. *Journal of Children Psychology and Psychiatry and Allied Disciplines*, 43(3), 307–325.
- Yeargin-Allsopp, M., Rice, C., Karapurkar, T., Doernberg, N., Boyle, C., & Murphy, C. (2003). Prevalence of autism in a US metropolitan area. *JAMA*, 289, 49–55.

BRI (Behavior Regulation Index)

- BRIEF (Behavior Rating Inventory of Executive Functions)

BRIAAC

- Behavior Rating Instrument for Autistic and Atypical Children (BRIAAC)

BRIEF (Behavior Rating Inventory of Executive Function)

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BRIEF (Behavior Rating Inventory of Executive Functions)

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Synonyms

ASD (autism spectrum disorder); BRI (Behavior Regulation Index); GEC (Global Executive Composite); MI (Metacognition Index)

Description

The Behavior Rating Inventory of Executive Function (BRIEF) is a questionnaire that assesses executive functioning behaviors of school-aged children, ages 5–18 (Gioia et al. 2000). Executive functioning is the ability to actively engage in an assortment of interrelated processes that are responsible for guiding goal-directed cognitive, emotional, and behavioral functioning. Both parent and teacher rating forms of the BRIEF exist, providing opportunities to rate executive functioning across home and school settings. The BRIEF is appropriate for the assessment of children with a variety of developmental, neurological, psychiatric, and medical conditions. It has also been normed and applied in samples of children with autism spectrum disorder (ASD).

The BRIEF consists of 86 items that cluster into eight clinical scales reflecting different aspects of executive functioning. The scales are entitled: Inhibit, Shift, Emotional Control, Initiate, Working Memory, Plan/Organize, Organization of Materials, and Monitor. Three of these scales combine to create the Behavioral Regulation Index (BRI), and five comprise the Metacognition Index (MI). All eight clinical scales of the BRIEF combine to yield the Global Executive Composite (GEC).

The BRI reflects the ability to shift cognitive set and to modulate behavior and emotion by exerting appropriate inhibitory control. The BRI is comprised of the Inhibit, Shift, and Emotional Control scales. The Inhibit scale assesses the ability to resist acting on an impulse or refrain from engaging in an impulsive behavior as appropriate to the situation. The Shift scale measures the ability to respond to changing circumstantial demands by flexibly transitioning from one activity, situation, or aspect of a problem to another. The Emotional Control scale quantifies the ability to regulate emotions and display emotional reactions that are situationally and developmentally appropriate.

The MI reflects the ability to cognitively manage tasks by effectively initiating, planning, organizing, and monitoring goal-oriented behaviors.

The MI is comprised of the Initiate, Working Memory, Plan/Organize, Organization of Materials, and Monitor scales. The Initiate scale measures the ability to independently begin a task or activity, including generating ideas or problem-solving strategies. The Working Memory scale assesses the ability to hold information in mind and to mentally manipulate information for the purpose of completing a task. The capacity to effectively sustain performance and attention is also integral to items on this scale. The Plan/Organize scale evaluates the ability to apply order to information, anticipate future events, set goals, and develop appropriate, often sequential, steps in the service of task completion. The Organization of Materials scale quantifies the child's ability to organize his or her work and play activities, as well as environment (e.g., possessions, storage, and work spaces). The Monitor scale assesses the ability to continuously engage in work-checking behaviors in order to ensure attainment of the designated goal. The scale also measures the child's ability to continuously evaluate how his or her behavior affects others.

In addition to the primary clinical scales, the questionnaire yields two validity scales, assessing inconsistency and negativity of the reporter. The Inconsistency scale of the BRIEF consists of ten pairs of items that are strongly correlated with one another. This scale is included in order to measure rater consistency. The Negativity scale is comprised of the nine items that were least frequently rated as "often" in the normative sample. High Negativity scale scores can indicate an excessively negative perception on the part of the rater, though at times they are also seen in profiles of children with very high levels of executive dysfunction.

The BRIEF is intended to be completed in a single sitting, requiring approximately 10–15 minutes. Each scale and summary index yields a T score, which have a mean of 50 and a standard deviation of 10. Scores above 65 are considered clinically elevated. Percentile scores are also provided. While the BRIEF can be administered by individuals who do not possess formal training, interpretation of scores requires relevant graduate training in the field of psychology.

Historical Background

The BRIEF was developed by neuropsychologists Gerard Gioia, PhD, Peter Isquith, PhD, Steven Guy, PhD, and Lauren Kenworthy, PhD, and published in 2000 by Psychological Assessment Resources, Inc. It was designed to measure children's executive functioning in real-world settings, as observed by parents, teachers, and caregivers. BRIEF items were generated from clinical interviews conducted by the authors, based on common concerns elicited from parents and teachers. The initial draft of the questionnaire included 129 items for the parent form and 127 items for the teacher form. The set of items was determined to match a fourth- to fifth-grade reading level, as intended by the authors.

Following initial item development, twelve neuropsychologists reviewed and categorized all items into their appropriate executive functioning domains. Principal factor analysis was then conducted to further clarify clustering of items. In the final version of the BRIEF, over 80 % of items retained had at least 75 % agreement among authors and expert raters regarding how well they fell within their designated scales.

Within the BRIEF, moderate to high correlations were found between most scales. Therefore, in order to strengthen the validity of the BRIEF scale structure, factor analysis was conducted, using normative and clinical samples for both the parent and teacher forms. Based on the results of a principal factor analysis, a two-factor structure was identified. This two-factor solution accounted for 74 % of the variance in parent forms and 83 % of the variance in teacher forms. The two factors derived from this analysis, entitled the Behavior Regulation Index (BRI) and Metacognition Index (MI), subsequently have been used to capture all eight scales of the BRIEF.

The BRIEF represents the first informant report measure of executive functioning available for use with children and adolescents. Strengths include the ability to measure multiple domains of executive functioning simultaneously. This feature can be contrasted with research and clinical task-based assessments,

which often quantify isolated abilities in distinct executive domains. By utilizing caregiver report of daily functioning, the BRIEF also claims high ecological validity, as findings reflect, and are generalizable to, executive functioning capacity in various real-life settings (Kenworthy et al. 2008). Criticisms of the BRIEF concern the validity and potential for bias inherent in any informant report measure. In addition, concerns have been raised about whether executive functions can truly be parsed into individual domains, as is suggested by the eight BRIEF scales. It has been noted that such parsing of the broader executive functioning construct is not yet well grounded in neuroimaging research regarding brain function (Denckla 2002).

Psychometric Data

The BRIEF was standardized and validated in a sample of school-aged children from diverse racial, socioeconomic, and geographic backgrounds. The normative sample included 1,419 parent forms and 720 teacher forms, for children between 5 and 18 years of age, attending public and private schools in Maryland. Approximately half of the normative sample was male, and no children had a history of special education or psychiatric medication. Among parent responders, 83 % were mothers.

From the normative raw data, T scores and percentile norms were developed for each of the eight BRIEF scales, the BRI and MI, and the GEC. T scores were generated using a linear transformation of raw scores. Percentiles were assigned based on the distribution of raw scores for each scale, index, and GEC. As for other behavior rating scales, BRIEF scales are positively skewed, so that the majority of cases cluster at the lower, normal end of each scale, while scores in the tail represent deviations from the norm.

Several aspects of test reliability were assessed using data from the normative sample. Internal consistency quantifies the degree to which items within individual scales are consistent with each

other and is statistically evaluated using Cronbach's alpha coefficient. Psychometric data indicated that across all individual scales and composite indices, in both parent and teacher forms, Cronbach's alpha values ranged from 0.80 to 0.98. These values indicate good to excellent internal reliability within scales.

Inter-rater reliability was also measured to determine consensus across parent and teacher forms completed for a subset of 296 children within the normative sample. Inter-rater reliability was found to range from 0.15 to 0.50 across the different scales and composites. These values indicate low reliability across raters; however, the authors note that low inter-rater reliability is expected given that these forms are intended to capture any existing variability in children's behavior across different environmental settings. Inter-rater reliability was lowest for the Initiate and Organization of Materials scales; in general, parents tended to endorse more problematic functioning than did teachers.

Finally, test-retest reliability was assessed in order to measure how consistent individual raters were in reporting on a child's behavior at different but proximal time points. For the parent form, this data was computed from a subsample of 54 participants within the normative sample with an average interval of 2 weeks between questionnaire completions for a single rater. Test-retest reliability values ranged from 0.76 to 0.85 across individual scales and from 0.84 to 0.88 for the three composites, indicating good reliability. For the teacher form, test-retest reliability was computed in a subsample of 41 participants, over an average interval of 3.5 weeks. Test-retest reliability for the teacher form ranged from 0.83 to 0.92 for individual BRIEF scales and from 0.90 to 0.92 for the BRI, MI, and GEC. These values indicate good test-retest reliability for individual scales, and excellent reliability for the composites when rated twice by teachers.

In their manual, the BRIEF authors also address two aspects of test validity or the extent to which the BRIEF measures what it proposes to measure. Content validity, reflects the degree to

which the content of the test instrument itself (i.e., BRIEF items) adequately captures all aspects of the construct (i.e., executive functioning) it purports to measure. The fact that items were selected from clinical interviews and that there was strong agreement among pediatric neuropsychologists that items fit within their intended scales, serves to strengthen content validity for the BRIEF. In addition, during test development, scales were refined with item-total correlations (i.e., the extent to which an individual item correlated with the overall score for its scale). Inter-rater agreement among expert reviewers served as an external check for scale membership of each item.

Construct validity speaks to the degree to which the measure adequately captures the construct that it aims to address. It can be quantified by evaluating convergence with other measures that target the same construct, as well as divergence with measures targeting dissociable constructs. Evidence for good construct validity is found in high correlations between BRIEF scales and other pertinent measures, including the ADD-IV scale (Zhang et al. 2005), and several subscales of the Child Behavior Checklist (CBCL) (Achenbach 1991). Specifically, all scales were highly correlated with the ADD-IV summary scores, which is expected given that executive functioning problems are prominent in children with diagnosed attention and behavioral disorders. In addition, there is some evidence for specificity and dissociability of individual BRIEF scales with regard to the constructs they aim to capture. For example, the BRIEF Initiate scale correlated with the Withdrawn, Anxious/Depressed, and Attention Problems scales of the CBCL, whereas the BRIEF Working Memory scale correlated only with CBCL Attention Problems scale. The BRIEF Initiate scale correlated with both the CBCL Attention Problems and Aggressive Behavior scales, whereas the BRIEF Shift and Emotional Control scales correlated with the CBCL Aggressive Behavior scale alone. Evidence of divergence with constructs unrelated to executive functioning is evident in low correlation rates

between BRIEF scales and the CBCL Somatization scale. Convergent and divergent construct validity was also evaluated by comparing the BRIEF, the Behavior Assessment System for Children (BASC) (Reynolds 2004), and the ► [Conners' Parent Rating Scale](#).

Clinical Uses

Findings pertaining to the application of the BRIEF with children and adolescents diagnosed with ASD are available in the BRIEF manual. As part of measure development, the BRIEF was administered to parents ($n = 26$) and teachers ($n = 18$) of children with high-functioning ASD, as well as to parents ($n = 18$) and teachers ($n = 16$) of typically developing children with no psychiatric diagnoses. Relative to controls, scores for children with high-functioning ASD, including Autistic Disorder, Asperger's Disorder, and Pervasive Developmental Disorder – Not Otherwise Specified – were significantly elevated across all BRIEF scales and composite indices.

Similar to findings detailed in the BRIEF manual, primary research literature also confirms that the BRIEF is sensitive to differences between children with ASD and those with no psychiatric diagnoses. Thus, researchers have reported that when comparing BRIEF scores of high-functioning children with ASD to the standardization sample in the BRIEF manual, BRI, MI, and GEC composite scores were all clinically elevated on average (Gilotty et al. 2002; Kenworthy et al. 2005; Kenworthy et al. 2009; Winsler et al. 2007). More specifically, compared to BRIEF norms, approximately two-thirds of children with ASD scored in the clinically impaired range (i.e., T scores over 65) on the BRI, MI, and GEC (Kenworthy et al. 2005). There is also some evidence that when boys with ASD are compared to those with typical development, the pattern of generalized score elevations is most salient for the Shift scale (Mackinlay et al. 2006).

Research conducted within ASD samples has revealed a link between executive functioning

symptom elevations on the BRIEF and greater severity in core symptoms associated with the disorder. Namely, higher scores on the BRI were related to greater impairment in Communication, Reciprocal Social Interaction, and Restricted and Repetitive Behavior domains as assessed by the most widely used diagnostic measures for autism (i.e., Autism Diagnostic Observation Schedule, Autism Diagnostic Interview – Revised) (Kenworthy et al. 2009). Likewise higher BRI scores were associated with score elevations on a measure specifically targeting repetitive behaviors (Boyd et al. 2009). Increased MI scores, in contrast, were related to greater social symptom severity only (Kenworthy et al. 2009). However, when BRIEF scores were compared to scores on a measure of adaptive functioning, MI scores were significantly correlated with impairment in both the socialization and communication domains (Gilotty et al. 2002).

The clinical utility of the BRIEF for children with ASD has been examined in comparison to other clinical populations. Specifically, researchers have shown that individuals with ASD can be differentiated from others with psychiatric, learning, behavioral, and learning disorders based on their BRIEF profiles. For example, children with ASD have more elevated scores on the BRIEF than do those with reading disabilities or traumatic brain injury (Gioia et al. 2002). Some research has demonstrated similarities between the BRIEF profiles of children with ASD and those with attention-deficit/hyperactivity disorder (ADHD) (Gioia et al. 2002; Winsler et al. 2007). However, deficits in flexibility, as indexed by the Shift scale of the BRIEF, appear to be most characteristic of children with ASD (Gioia et al. 2002).

Taken together, research to date has demonstrated that the BRIEF is sensitive to behavioral impairments in regulatory and metacognitive functioning in children with ASD. The level of impairment and relation between BRIEF scores and adaptive functioning in this group underscore the importance of evaluating executive functioning when assessing and treating children with ASD. The BRIEF offers a unique tool for assessing this domain of behavior, as observed by adults

familiar with children's day-to-day functioning in real-world settings.

References and Reading

- Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4-18 and 1991 profile* (p. 288). Burlington: Department of Psychiatry, University of Vermont.
- Boyd, B. A., McBee, M., Holtzclaw, T., Baranek, G. T., & Bodfish, J. W. (2009). Relationships among repetitive behaviors, sensory features, and executive functions in high functioning autism. *Research in Autism Spectrum Disorders*, 3(4), 959–966.
- Denckla, M. B. (2002). The behavior rating inventory of executive function: Commentary. *Child Neuropsychology*, 8(4), 304–306.
- Gilotty, L., Kenworthy, L., Sirian, L., Black, D. O., & Wagner, A. E. (2002). Adaptive skills and executive function in autism spectrum disorders. *Child Neuropsychology*, 8, 241–248.
- Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2000). *Behavior rating inventory of executive function professional manual*. Lutz: Psychological Assessment Resources.
- Gioia, G. A., Isquith, P. K., Kenworthy, L., & Barton, R. M. (2002). Profiles of everyday executive function in acquired and developmental disorders. *Child Neuropsychology*, 8, 121–137.
- Kenworthy, L. E., Black, D. O., Wallace, G. L., Ahluvalia, T., Wagner, A. E., & Sirian, L. M. (2005). Disorganization: The forgotten executive dysfunction in high-functioning autism (HFA) spectrum disorders. *Developmental Neuropsychology*, 28, 809–827.
- Kenworthy, L., Yerys, B. E., Anthony, L. G., & Wallace, G. L. (2008). Understanding executive control in autism spectrum disorders in the lab and in the real world. *Neuropsychology Review*, 18, 320–338.
- Kenworthy, L., Black, D. O., Harrison, B., Della Rosa, A., & Wallace, G. L. (2009). Are executive control functions related to autism symptoms in high-functioning children? *Child Neuropsychology*, 15(5), 425–440.
- Mackinlay, R., Charman, T., & Karmiloff-Smith, A. (2006). High functioning children with autism spectrum disorder: A novel test of multitasking. *Brain and Cognition*, 61, 14–24.
- Reynolds, C. R. (2004). *Behavior assessment system for children*. Wiley.
- Winsler, A., Abar, B., Feder, M. A., Schunn, C. D., & Rubio, D. A. (2007). Private speech and executive functioning among high-functioning children with autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 1617–1635.
- Zhang, S., Faries, D. E., Vowles, M., & Michelson, D. (2005). ADHD rating scale IV: psychometric properties from a multinational study as clinician-administered instrument. *International Journal of Methods in Psychiatric Research*, 14(4), 186–201.

Brief Infant-Toddler Social and Emotional Assessment (BITSEA)

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Synonyms

BITSEA; BITSEA-ASD subscales

Description

The Brief Infant-Toddler Social and Emotional Assessment (BITSEA) (Briggs-Gowan and Carter 2006) is a 42-item caregiver-report broadband screening tool developed to identify social-emotional and behavior problems and delays in the acquisition of competencies in children 11–48-months of age. The BITSEA was derived from the longer (166 item) Infant-Toddler Social and Emotional Assessment (ITSEA; Carter and Briggs-Gowan 2000; Carter et al. 2003), which provides an in-depth analysis of emerging social-emotional development and intervention guidance. The BITSEA can be completed by caregiver report in approximately 5–7 minutes and is written at a fourth to sixth grade reading level (Briggs-Gowan et al. 2004). Items are rated on a three-point Likert scale, with the following anchors: not true/rarely (0), somewhat true/sometimes (1), and very true/often (2). The information gathered from the BITSEA covers a broad range of social-emotional and behavioral functioning and generates scores in internalizing, externalizing, and regulatory domains, as well as other areas that may be indicative of emerging psychopathology. Following the original scoring of the BITSEA, items are grouped into Problem and Competence domains that generate possible problem and possible delay/deviance cut-scores as well as percentiles based on national norms.

Of the total 42 items, the BITSEA contains 19 specific items that are consistent with early-

Brief Infant-Toddler Social and Emotional Assessment (BITSEA), Table 1 Behaviors assessed on BITSEA ASD subscales^a

Subscale	Psychometric properties	Behaviors assessed ^a
ASD-Problems	Sensitivity: 76% Specificity: 72% PPV: 68%	Limited enjoyment of playful activities
		Unresponsive when hurt
		Tactile sensitivities
		Difficulty with transitions
		Repetitive play
		Repetitive speech
		Repetitive motor movements
		Appears unaware of surroundings
		Limited eye contact
		Avoidant of physical contact
ASD-Competence	Sensitivity: 91% Specificity: 80% PPV: 77%	Shares successes
		Seeks out caregiver when upset
		Responds to name with eye contact
		Emotional/physical affection
		Interactive play
		Empathy when someone is hurt
		Imitation skills
		Use of pointing to share interests/joint attention
		Pretend play

^aAdapted from table originally published by Giserman Kiss et al. (2017)

emerging autism spectrum disorder (ASD) symptomatology (See Table 1). An important feature of the BITSEA is its inclusion of both positively and negatively worded items, relating to both problem behaviors and delayed competencies associated with ASD; thus the BITSEA ASD items make up two ASD-specific subscales: ASD-Problems and ASD-Competence. The ASD-Competence subscale consists of nine items that assess early social-emotional and social-communication competencies expected in typical development. The ASD-Problems subscale consists of ten individual items that assess social-communication impairments and repetitive and restricted behaviors commonly seen in young children with ASD. The ASD-Problems and ASD-Competence subscales are calculated and prorated if fewer than three items are missing from either scale. BITSEA ASD subscales can be scored individually or together, as an ASD-Total score; however, findings presented by Giserman Kiss et al. (2017) demonstrate that the ASD-Competence subscale is the most statistically and clinically effective of all three subscales

(see the Psychometric Data section for more information). Children with subscale scores exceeding ASD-Competence, ASD-Problem, or ASD-Total cutoffs are considered “at risk” for ASD, and further developmental assessment is strongly recommended.

The BITSEA allows clinicians to efficiently identify young children who are showing early symptoms of ASD, while simultaneously screening for early-emerging non-ASD social-emotional and behavior problems. Simultaneous screening may allow for more efficient assessments in fast-paced pediatric and early education settings and eliminate providers’ discomfort regarding introducing an ASD-specific screener to families who have not raised any concerns about their children’s social-emotional development, ultimately leading to increased universal screening.

Historical Background

The BITSEA was originally developed in response to the recognition of the importance of

early detection of and early intervention services for young children with social-emotional and behavioral deficits. While the ITSEA responded to this need and effectively identified children with early-emerging psychopathology, authors recognized the limitations of this tool, particularly regarding the time required for completion and scoring. With the recommendation for routine screening during well-child visits by the American Academy of Pediatrics (AAP 2001), as well as the introduction of managed care which resulted in shorter pediatric office visits, a more efficient questionnaire, the BITSEA, was derived from the pool of ITSEA questions. Original and replication studies of the full BITSEA demonstrated excellent test-retest reliability and good interrater agreement between parents when used in a socioeconomically and ethnically diverse community-based population (Briggs-Gowan et al. 2004; Kruzinga et al. 2012). Subsequent studies found strong prediction of concurrent psychiatric disorders (Briggs-Gowan et al. 2013) and good prediction to parent- and teacher-reported school-aged psychopathology (Briggs-Gowan and Carter 2008). In the original BITSEA manual, the authors recommended that the BITSEA be used for identifying young children with ASD, based on an at risk score on the overall competence scale as well as inspection of the scores for a subset of these ASD-consistent items. The ASD item pool was expanded to include sensory over- and under-responsivity following the publication of the DSM-5 criteria (APA 2013). However, cut-scores for the ASD items were not published.

The increased recognition of the benefits of early intervention services for children diagnosed with ASD (e.g., Pickles et al. 2016; Seida et al. 2009; Woods and Wetherby 2003), updated recommendations of the AAP and Centers for Disease Control for frequent developmental surveillance and screening (AAP 2006; Baio 2012), and common use of the BITSEA across research and clinical settings to identify toddlers at risk for ASD lead Giserman Kiss et al. (2017) to assess the feasibility of the BITSEA ASD subscales. The measure was tested in a sample that included young children with ASD, non-ASD

psychopathology, and typical development. Findings optimized cut-scores for each subscale that evidenced moderate to high discriminative power for detecting children with ASD. Of the three subscales, the ASD-Competence scale proved to be the most statistically and clinically effective (see the Psychometric Data section for more information).

Psychometric Data

Giserman Kiss et al. (2017) used receiver-operating characteristic (ROC) plots to determine optimal cut-scores on the BITSEA ASD subscales in a diverse sample of 512 young children (223 in the ASD group, and 289 in the non-ASD group) ranging in age from 15 to 48 months. Children in the non-ASD group included those with typical development as well as non-ASD early-emerging psychopathology. With regard to the ASD-Problems subscale, analyses using the optimized cut-score revealed moderate subscale accuracy (AUC = 0.81), 76% sensitivity, 72% specificity, and 68% positive predictive value (PPV). The ASD-Competence subscale optimized cut-score yielded stronger psychometric properties, as analyses evidenced high subscale accuracy (AUC = 0.92), 91% sensitivity, 80% specificity, and 77% PPV. Finally, the ASD-Total subscale optimized cut-score also yielded high subscale accuracy (AUC = 0.92), as well as 80% sensitivity, 79% specificity, and 77% PPV. Sensitivity and specificity rates for all three subscale optimized cut-scores in the subsample of children 24 months old and younger were comparable or stronger to rates in the overall sample. Importantly, the ASD-Competence subscale outperformed the other subscales in identifying both true positives and true negatives.

Post-hoc analyses explored the clinical characteristics of the children who screened positive despite not receiving an ASD diagnosis. Analyses revealed that children who screened as false positives on the ASD-Competence scale (i.e., screened positive but did not have ASD) had significantly lower nonverbal problem-

solving, receptive language, and expressive language abilities than children that screened as true negatives (i.e., screened negative and did not have ASD). In addition, children who screened as false negatives (i.e., screened negative but had ASD) had significantly higher receptive language scores than true positives (i.e., screened positive and had ASD).

Clinical Uses

It is recommended that the ASD-Competence subscale be used in pediatric primary care, early intervention, and early education settings to detect children at risk for ASD, while simultaneously assessing other areas of a child's development. The ASD-Competence subscale demonstrated strong psychometric properties and is quick and easy for providers to score. While the ASD-Total subscale also demonstrated high sensitivity, specificity, and PPV, scoring this subscale can be more cumbersome, error-prone, and time-consuming due to the additional items and need to perform calculations. Thus, providers are strongly encouraged to use the ASD-Competence subscale in order to examine a child's risk status for ASD, while simultaneously using the overall BITSEA to assess other social-emotional and behavioral domains, thus fulfilling the AAP and CDC's recommendations for regular developmental surveillance and screening. If a child's score on the ASD-Competence subscale exceeds the designated cut-score, caregivers should be counseled to seek further assessment of ASD symptoms, either through a second-stage observational screener, more in-depth conversation about the child's functioning across settings and relationships, or a developmental evaluation.

The broadband nature of the BITSEA may reduce the previously documented stress felt by caregivers during the ASD screening and diagnostic process (Siklos and Kerns 2007). A global screener that has a specific ASD subscale may give the provider additional time and avenues by which to introduce the idea of ASD. Moreover,

the use of positively as well as negatively worded questions on the BITSEA may be more acceptable to parents. Finally, given the increased risk for specific forms of psychopathology among children with ASD (e.g., anxiety, sleeping, and feeding disorders) (Ming et al. 2008), gathering information about symptoms beyond ASD may inform comprehensive intervention services, when needed.

Several limitations of the BITSEA ASD subscales should be kept in mind during use. Giserman Kiss et al. (2017) reported that the ASD-Competence subscale is possibly limited by its overlap with symptoms of general intellectual or developmental disabilities or other early-emerging psychopathology and also may be compromised by advanced language abilities. Thus, like any screening tool, the BITSEA should be used as an initial assessment and not as a diagnostic tool.

References and Reading

- American Academy of Pediatrics. (2001). Committee on children with disabilities. Developmental surveillance and screening of infants and young children. *Pediatrics*, 108, 192–196.
- American Academy of Pediatrics, Council on Children with Disabilities, Section on Developmental and Behavioral Pediatrics, Bright Futures Steering Committee, Medical Home Initiatives for Children with Special Needs Project Advisory Committee. (2006). Identifying infants and young children with developmental disorders in the medical home: An algorithm for developmental surveillance and screening. *Pediatrics*, 118, 405–420. <https://doi.org/10.1542/peds.2006-1231>.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association.
- Baio, J. (2012). Prevalence of autism spectrum disorders: Autism and developmental disabilities monitoring network, 14 sites, United States, 2008. Morbidity and mortality weekly report. Surveillance summaries. *Centers for Disease Control and Prevention*, 61(3), 1–19.
- Briggs-Gowan, M. J., & Carter, A. S. (2006). *Examiner's manual for the Brief Infant-Toddler Social and Emotional Assessment (BITSEA)*. San Antonio: Psychological Corporation, Harcourt Press.
- Briggs-Gowan, M. J., & Carter, A. S. (2008). Social-emotional screening status in early childhood predicts

- elementary school outcomes. *Pediatrics*, 121(5), 957–962. <https://doi.org/10.1542/peds.2007-1948>.
- Briggs-Gowan, M. J., Carter, A. S., Irwin, J., Wachtel, K., & Cicchetti, D. V. (2004). The brief infant-toddler social and emotional assessment: Screening for social-emotional problems and delays in competence. *Journal of Pediatric Psychology*, 29(2), 143–155.
- Briggs-Gowan, M. J., Carter, A. S., McCarthy, K., Augustyn, M., Caronna, E., & Clark, R. (2013). Clinical validity of a brief measure of early childhood social-emotional/behavioral problems. *Journal of Pediatric Psychology*, 38(5), 577–587.
- Carter, A. S., & Briggs-Gowan, M. J. (2000). *Manual of the infant-toddler social-emotional assessment*. New Haven: Yale University.
- Carter, A. S., Briggs-Gowan, M. J., Jones, S. M., & Little, T. D. (2003). The infant-toddler social and emotional assessment: Factor structure, reliability, and validity. *Journal of Abnormal Child Psychology*, 31, 495–514.
- Giserman Kiss, I., Feldman, M. S., Sheldrick, R. C., & Carter, A. S. (2017). Developing autism screening criteria for the Brief Infant Toddler Social Emotional Assessment (BITSEA). *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-017-3044-1>.
- Kruizinga, I., Jansen, W., de Haan, C. L., van der Ende, J., Carter, A. S., & Raat, H. (2012). Reliability and validity of the Dutch version of the Brief Infant-Toddler Social and Emotional Assessment (BITSEA). *PloS One*, 7(6). <https://doi.org/10.1371/journal.pone.0038762>.
- Ming, X., Brimacombe, M., Chaaban, J., Zimmerman-Bier, B., & Wagner, G. C. (2008). Autism spectrum disorders: Concurrent clinical disorders. *Journal of Child Neurology*, 23(1), 6–13. <https://doi.org/10.1177/0883073807307102>.
- Pickles, A., Le Couteur, A., Leadbitter, K., Salomone, E., Cole-Fletcher, R., Tobin, H., et al. (2016). Parent-mediated social communication therapy for young children with autism (PACT): Long-term follow-up of a randomised controlled trial. *The Lancet*, 388, 2501–2509.
- Seida, J., Ospina, M. B., Karkhanav, M., Hartling, L., Smith, V., & Clark, B. (2009). Systematic reviews of psychosocial interventions for autism: An umbrella review. *Developmental Medicine and Child Neurology*, 51(2), 95–104. <https://doi.org/10.1111/j.1469-8749.2008.03211.x>.
- Siklos, S., & Kerns, K. A. (2007). Assessing the diagnostic experiences of a small sample of parents of children with autism spectrum disorders. *Research in Developmental Disabilities*, 28(1), 9–22. <https://doi.org/10.1016/j.ridd.2005.09.003>.
- Woods, J. J., & Wetherby, A. M. (2003). Early identification of and intervention for infants and toddlers who are at risk for autism spectrum disorder. *Language, Speech, and Hearing Services in Schools*, 34(3), 180–193. [https://doi.org/10.1044/0161-1461\(2003/015\)](https://doi.org/10.1044/0161-1461(2003/015)).

Brief Observation of Social Communication Change (BOSCC)

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Abbreviations

ADOS-2	Autism Diagnostic Observation Schedule, second Edition
ASD	Autism spectrum disorder
BOSCC	Brief Observation of Social Communication Change

Description

The Brief Observation of Social Communication Change (BOSCC; Grzadzinski et al. 2016) is a treatment response measure of autism spectrum disorder (ASD) symptoms. The BOSCC is a behavioral coding scheme that is applied to 10–12 min videotaped social/play interactions between a child and a researcher or caregiver (play partner). The coding scheme was developed by expanding items from the Autism Diagnostic Observation Schedule, second Edition (ADOS-2; Lord et al. 2012), to quantify more nuanced variation in ASD symptoms. The BOSCC has been shown to be sensitive to changes in ASD symptoms from pre- to post-treatment (Divan et al. 2018; Frost et al. 2019; Gengoux et al. 2019; Grzadzinski et al. 2016; Kim et al. 2019; Kitzerow et al. 2015; Pijl et al. 2016), was designed to be flexible to use across sites/studies and coded by trained coders who were relatively naïve to ASD. As a measure of global ASD symptom change, studies show the BOSCC is most effective at quantifying treatment response in intervention trials examining improvements across a range of ASD symptoms. A module of the BOSCC for minimally verbal (those using single words or

less) toddlers or preschoolers is currently available to researchers with an applicable research study. Development of BOSCC modules for individuals with phrase to fluent speech are under development. Both the BOSCC play partner and coder can be unaware of the child's treatment status and time point, eliminating any potential bias associated with knowledge of the intervention.

Historical Background

Advances in developing efficacious interventions are limited by a lack of treatment response measures, without which researchers are hindered in evaluating intervention programs (French and Kennedy 2018). Many early interventions focus on improving core ASD symptoms (See ► [Need for Caregiver Support for Families of Children with ASD, The](#), and "Intervention During the Prodromal Stages of ASD"). Often subtle in nature, quantifying changes in core ASD symptoms is difficult (Anagnostou et al. 2015). Intervention programs have used the ADOS-2 to capture changes in ASD symptoms (Dawson et al. 2010; Estes et al. 2015) with limited success – perhaps since the ADOS-2 was developed to provide a diagnosis rather than measure changes in symptoms. There are additional limitations to using the ADOS-2 as well. Reliable use of the ADOS-2 requires substantial training and expertise of ASD, as well as substantial clinician and participant time (~45–60 min), limiting its use across sites and studies. As well as the ADOS-2, studies have used measures developed specifically for their intervention, impeding comparability across studies, or have focused on parent reports of improvement that, while important, may yield subjective information since parents are often aware of the child's treatment status (Bolte and Diehl 2013; Guastella et al. 2015). To address such limitations, the BOSCC was developed by expanding ADOS-2 items to increase sensitivity compared to a diagnostic coding scheme. The BOSCC administration is also shorter, and the coding can be completed by less experienced individuals who are unaware of the child's treatment status and time point.

Psychometric Data

The initial psychometric properties of the BOSCC were evaluated in a sample of 56 children participating in early intervention trials (Grzadzinski et al. 2016). Results of this and subsequent work indicate that the BOSCC has high inter-rater (intra-class correlation coefficients; ICCs = 0.88–0.98) and test-retest (ICCs = 0.79–0.90) reliabilities (Grzadzinski et al. 2016; Kim et al. 2019). These reliabilities have been further confirmed in independent samples (Frost et al. 2019; Kitzerow et al. 2015; Pijl et al. 2016). Results also showed statistically significant decreases (improvements) in BOSCC scores, with small to moderate effect sizes (effect sizes = 0.37–0.60), over 6 to 8 months of intervention (Grzadzinski et al. 2016; Kim et al. 2019). These improvements aligned with improvements observed in parent reports and standardized assessments of communication skills (Grzadzinski et al. 2016; Kim et al. 2019; Mullen 1995; Sparrow et al. 2005). Independent studies have also shown significant decreases in BOSCC scores over the course of intervention (Divan et al. 2018; Frost et al. 2019; Gengoux et al. 2019; Grzadzinski et al. 2016; Kim et al. 2019; Kitzerow et al. 2015; Pijl et al. 2016). However, not all intervention trials using the BOSCC have shown significant improvements. The amount of change observed may be dependent on characteristics of the intervention, such as intensity or duration (Fletcher-Watson et al. 2016; Nordahl-Hansen et al. 2016). See Grzadzinski and Lord, 2019 for a review of the BOSCC development.

Clinical Uses

The BOSCC is a treatment response measure that quantifies changes in ASD symptoms over the course of an intervention (See ► [Need for Caregiver Support for Families of Children with ASD, The](#), and "Intervention During the Prodromal Stages of ASD"). The BOSCC is not currently available for clinical use though can be obtained for use by researchers with an applicable

research study and who have completed appropriate training. The BOSCC is appropriate for minimally verbal (single words or less) toddler/preschoolers with ASD, and modules are currently under development for individuals with phrase to fluent speech. The BOSCC is not intended for use as an ASD screener (See “Screening for ASD and Developmental Delays in Infants and Toddlers”), diagnosis, or severity metric. The BOSCC’s ability to capture treatment response in other clinical conditions that may have overlapping symptoms with ASD (e.g., fragile X) remains an area of exploration.

References and Reading

- Anagnostou, E., Jones, N., Huerta, M., Halladay, A. K., Wang, P., Scahill, L., et al. (2015). Measuring social communication behaviors as a treatment endpoint in individuals with autism spectrum disorder. *Autism, 19*(5), 622–636. <https://doi.org/10.1177/1362361314542955>.
- Bolte, E. E., & Diehl, J. J. (2013). Measurement tools and target symptoms/skills used to assess treatment response for individuals with autism spectrum disorder. *Journal of autism and developmental disorders, 43* (11), 2491–2501.
- Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., et al. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The early start Denver model. *Pediatrics, 125*(1), e17–e23. <https://doi.org/10.1542/peds.2009-0958>.
- Divan, G., Vajaratkar, V., Cardozo, P., Huzurbazar, S., Verma, M., Howarth, E., et al. (2018). The feasibility and effectiveness of PASS plus, a lay health worker delivered comprehensive intervention for autism Spectrum disorders: Pilot RCT in a rural low and middle income country setting. *Autism Research*. in press.
- Estes, A., Munson, J., Rogers, S., Greenson, J., Winter, J., & Dawson, G. (2015). Long-term outcomes of early intervention in 6-year-old children with autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 54*(7), 580–587.
- Fletcher-Watson, S., Petrou, A., Scott-Barrett, J., Dicks, P., Graham, C., O’Hare, A., . . . & McConachie, H. (2016). A trial of an iPad™ intervention targeting social communication skills in children with autism. *Autism, 20*(7), 771–782.
- French, L., & Kennedy, E. M. (2018). Annual research review: Early intervention for infants and young children with, or at-risk of, autism spectrum disorder: A systematic review. *Journal of Child Psychology and Psychiatry, 59*(4), 444–456.
- Frost, K. M., Koehn, G. N., Russell, K. M., & Ingersoll, B. (2019). Measuring child social communication across contexts: Similarities and differences across play and snack routines. *Autism Research, 12*(4), 636–644.
- Gengoux, G. W., Abrams, D. A., Schuck, R., Millan, M. E., Libove, R., Ardel, C. M., . . . & Hardan, A. Y. (2019). A Pivotal Response Treatment Package for Children With Autism Spectrum Disorder: An RCT. *Pediatrics, e20190178*.
- Grzadzinski, R., Carr, T., Colombi, C., McGuire, K., Dufek, S., Pickles, A., & Lord, C. (2016). Measuring changes in social communication behaviors: Preliminary development of the brief observation of social communication change (BOSCC). *Journal of Autism and Developmental Disorders, 46*(7), 2464–2479.
- Grzadzinski, R. & Lord, C. (2019). Commentary: Insights into the Development of the Brief Observation of Social Communication Change (BOSCC). *Journal of Mental Health and Clinical Psychology*.
- Guastella, A. J., Gray, K. M., Rinehart, N. J., Alvares, G. A., Tonge, B. J., Hickie, I. B., et al. (2015). The effects of a course of intranasal oxytocin on social behaviors in youth diagnosed with autism spectrum disorders: A randomized controlled trial. *Journal of Child Psychology and Psychiatry, 56*(4), 444–452. <https://doi.org/10.1111/jcpp.12305>.
- Kim, S. H., Grzadzinski, R., Martinez, K., & Lord, C. (2019). Measuring treatment response in children with autism spectrum disorder: Applications of the brief observation of social communication change to the autism diagnostic observation schedule. *Autism, 23*(5), 1176–1185.
- Kitzerow, J., Teufel, K., Wilker, C., & Freitag, C. M. (2015). Using the brief observation of social communication change (BOSCC) to measure autism-specific development. *Autism Research, 9*, 940–950.
- Lord, C., Rutter, M., DiLavore, P., Risi, S., Gotham, K., & Bishop, S. (2012). *Autism diagnostic observation schedule—2nd edition (ADOS-2)*. Los Angeles: Western Psychological Corporation.
- Mullen, E. M. (1995). *Mullen scales of early learning*. Circle Pines: American Guidance Service.
- Nordahl-Hansen, A., Fletcher-Watson, S., McConachie, H., & Kaale, A. (2016). Relations between specific and global outcome measures in a social-communication intervention for children with autism spectrum disorder. *Research in Autism Spectrum Disorders, 29*, 19–29.
- Pijl, M. K., Rommelse, N. N., Hendriks, M., De Korte, M. W., Buitelaar, J. K., & Oosterling, I. J. (2016). Does the Brief Observation of Social Communication Change help moving forward in measuring change in early autism intervention studies? *Autism, 1362361316669235*.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales, (Vineland-II)*. Circle Pines: American Guidance Services.

Broader Autism Phenotype

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Definition

Autism spectrum disorder (ASD) twin and family studies showed during the 1990s that the behavioral phenotype extends beyond the clinical diagnoses of autism and ASD to include related milder behaviors or personality traits in the relatives of affected individuals. These qualitatively similar ASD-related behaviors in relatives are termed the broader autism phenotype (BAP) (see Losh et al. 2011, for a review). Although several authors have reported that these symptoms and traits are continuously distributed in the general population, the term “BAP” has not been used to describe individuals with social communication difficulties from population samples (see Constantino 2011, for a review of this literature).

Researchers have defined BAP characteristics using interview and questionnaire methods, neuropsychological and neurophysiological testing, and neuroimaging (Bailey and Parr 2003; Dawson et al. 2002; Losh et al. 2011). However, there is no formal definition of the BAP due to variability in approaches and research findings (see section “Historical Background”); indeed BAP is not a “diagnosis” recognized in the international diagnostic classification systems.

Thus, the best working definition of the BAP would be “individuals with the BAP show behavioral characteristics and personality traits similar to, but milder than, their relative with ASD” (see Current Knowledge).

Historical Background

Over the last 20 years, many groups have used a variety of instruments to define various characteristics in relatives of people with ASD. A large body of literature describes the different components of the BAP proposed following studies using a range of methodologies and measures in different populations (see Bailey et al. 1998, and Losh et al. 2011, for reviews). Research shows that in keeping with ASD, impaired social communication and social emotional abilities are core features of the BAP, together with repetitive behaviors (including obsessional behaviors) and behavioral rigidity (for reviews, see Parr et al. 2011 and Losh et al. 2011). Of all the BAP traits, repetitive behaviors, rigidity, perfectionism, obsessions, and circumscribed/special interests have been the most difficult to identify and quantify.

Two approaches have been taken to the investigation of these ASD-like behavioral traits. First, researchers have focused on identifying the BAP in families of one child with ASD (singleton families) and two or more people with ASD (multiplex families). Various groups have continued this research as part of the search for autism susceptibility genes (see the work of Piven, Dawson, and Parr et al. and the International Molecular Genetic Study of Autism Consortium [IMGSAC]). In addition to investigating the behavioral BAP within ASD families, researchers have focused on identifying neuropsychological components of the BAP in the relatives of people with ASD (Dawson et al. 2002). More recently, neurophysiological measurement and neuroimaging studies of relatives have been a focus (see Losh et al. 2011); the rationale for using all these approaches to BAP characterization is described by Bailey and Parr (2003). By contrast to this investigation of the relatives of people with ASD, other groups have conceptualized and measured a range of social communication and other difficulties in the context of normative trait variation in the general population hypothesizing that these traits could be included on a dimension with ASD (see Constantino 2011, for a review).

The BAP has been of increasing interest to researchers due to its potential importance for understanding the neurobiological nature of ASD (for a review, see Lainhart and Lange 2011). From 2000, research groups began collecting data from relatives in an attempt to assist in the search for ASD susceptibility genes, assuming that the BAP indexes a “genetic risk” that may be present in one or both parents and “unaffected” siblings – thus relatives might carry ASD susceptibility genes and express an “ASD-like” phenotype (see Bailey and Parr 2003, and Losh et al. 2011).

The most commonly used measures for the identification of BAP in affected families are summarized in Table 1. In keeping with ASD itself, reliable direct observation of BAP behaviors has been challenging. For this reason, most research groups have used some form of interview data, either exclusively or in combination with other measures. For most ASD molecular genetic studies, the BAP measures have been designed to dimensionalize the social communication difficulties of parents and children (e.g., quantitative trait loci studies) rather than to define an affected/unaffected categorical “cut-off score” – this means “the BAP” is less clearly defined than might be expected.

Current Knowledge

Research studies have shown that relatives of people with ASD have difficulties qualitatively

similar to those seen in ASD, but milder – the individual’s profile of difficulties does not meet clinical ASD threshold. Generally, males are more commonly and more severely affected than females, and relatives from multiplex families are more affected than those from singleton families. Children and young people may have difficulties with developing and maintaining friendships and problems relating to others. Children frequently have less well-developed social play than their same-age peers. Children and adults may be considered aloof. Language and communication difficulties are common. Perfectionism, obsessions, and rigidity may be seen. Considering mental health, the BAP has been associated with affective disorder (particularly depression). Whether depression is part of the BAP or is a function of having a relative with ASD remains unknown (for a detailed review, see Losh et al. 2011).

Investigation of the familial mechanisms that underpin ASD continue and, indeed, the finding that parents from multiplex and simplex ASD families show the BAP at different rates is likely to be important for our understanding of etiology. However, to date, the BAP has contributed only modestly to the understanding of the neurobiology of ASD or the identification of genetic variants (see Lainhart and Lange 2011 and Parr et al. 2011).

The impact of the BAP on the functioning of affected children, young people, and adults is similar to that seen in ASD itself, but milder, and usually results in less impairment in daily life.

Broader Autism Phenotype, Table 1 Broader autism phenotype measures

Instrument	Interview/ questionnaire/ observation	Populations used	References
Social responsiveness scale	Questionnaire	Twin, singleton, multiplex, and general pop samples	Constantino and Todd (2003)
BAPQ	Questionnaire	Singleton and multiplex families	Hurley et al. (2007)
BPASS	Interview and observation	Singleton and multiplex families	Dawson et al. (2007)
Family history interview and impression of interviewee	Interview and observation	Singleton and multiplex families	Parr et al. and the IMGSAC, in preparation

However, BAP traits may lead to difficulties with peer interactions and marital relationships and thus potentially difficulties at home, school, and in the workplace. People with BAP have varying degrees of insight into their difficulties and the impact of their behavior for themselves and others (Losh et al. 2011; Parr et al. 2011).

“BAP” is a term used in research and not usually in clinical practice. However, in clinical settings, with their knowledge of the importance of genetic factors in autism, relatives of people with ASD comment about their own ASD-related difficulties, or those of other family members. For clinicians, the challenge is how best to “classify” these difficulties shown by people who do not have ASD but who do experience some degree of social communication impairment. It is important to be able to effectively describe these difficulties for the affected individual themselves, families, and professionals; this leads to a better understanding of the person’s behaviors and the reason for them. This is likely to be particularly important for individuals who may benefit from specific intervention and resources, for example, mentoring in the workplace for adults with BAP and support from education and/or social care professionals for affected children, young people, and adults (see Parr et al. 2011; Parr and Le Couteur 2011).

Finally, relatively little is known about the neurobiology or pathophysiology of the BAP. It has been hoped that better understanding the BAP will lead to improved neurobiological knowledge about ASD itself. However, in keeping with ASD, replicated neurobiological findings are scarce (comprehensively recently reviewed by Lainhart and Lange 2011). Whether BAP will play a significant role in advancing our understanding of the complexity of ASD remains to be seen (Parr et al. 2011).

Future Directions

During the next decade, studies of parents and other relatives of people with ASD will continue, and this will undoubtedly expand the understanding of subclinical ASD traits. Genetics research is

likely to continue to be a major “driver” of increased knowledge about BAP, for example, there will be great interest in the extent to which the BAP is seen in the relatives of people with ASD who have an identified inherited or de novo causal variant as this will further inform our knowledge of the genetic and environmental contributions to ASD.

Another exciting prospect will be the findings from studies of siblings of children with ASD (“at-risk” or “high-risk” sibling studies). These studies will provide insights into the developmental trajectories of children with ASD and those without ASD who have the BAP; both groups can be compared to siblings who develop typically and to controls. In the future, as “high-risk” siblings move toward and into adolescence and adulthood, the knowledge of how early development and subsequent characteristics relate to individual progress and outcomes will improve.

Finally, one new direction for BAP research relates to intervention. There is currently great interest in whether intervention changes the developmental trajectories and outcomes for “at-risk” siblings (e.g., the study of Green and colleagues in the UK). Projects evaluating the effect of the BAP on the delivery of parent-mediated early intervention for ASD have commenced. If research findings show that the BAP has a negative impact on the effectiveness of parents’ interactions with their child with ASD, identifying the most beneficial intervention strategies for BAP-affected parents will become both a research and a clinical priority to ensure better understanding and effective targeting of evidence-based interventions.

For older children and adults with the BAP, interventions and treatments need to be evaluated. Researchers are, for example, beginning to investigate whether behavioral interventions such as social skills training or social stories might improve the social skills of people with BAP. Indeed, it could be argued that people with BAP might be more responsive to such interventions than individuals with a clinical diagnosis of ASD as they are less likely to have cognitive impairment, will have milder social impairment, and

may well have more insight into their difficulties. Workplace interventions for people with BAP may also be of benefit – whether mentoring or other types of workplace support give adults a greater chance of working more productively with colleagues still remains to be seen.

See Also

- [Autism](#)
- [Perfectionism](#)
- [Repetitive Behavior](#)
- [Social Communication](#)

References and Reading

- Bailey, A., Palferman, S., Heavey, L., & Le Couteur, A. (1998). Autism: The phenotype in relatives. *Journal of Autism and Developmental Disorders*, 28(5), 369–392. Review.
- Bailey, A., & Parr, J. (2003). Implications of the broader phenotype for concepts of autism. *Novartis Foundation Symposium*, 251, 26–35.
- Constantino, J. N. (2011). Autism as a quantitative trait. In D. Amaral, D. Geschwind, & G. Dawson (Eds.), *Autism spectrum disorders* (pp. 510–520). New York: Oxford University Press.
- Constantino, J. N., & Todd, R. D. (2003). Autistic traits in the general population: A twin study. *Archives of General Psychiatry*, 60, 524–530.
- Dawson, G., Estes, A., Munson, J., Schellenberg, G., Bernier, R., & Abbott, R. J. (2007). Quantitative assessment of autism symptom-related traits in probands and parents: Broader phenotype autism symptom scale. *Journal of Autism and Developmental Disorders*, 37(3), 523–536.
- Dawson, G., Webb, S., Schellenberg, G. D., Dager, S., Friedman, S., Aylward, E., et al. (2002). Defining the broader phenotype of autism: Genetic, brain, and behavioral perspectives. *Development and Psychopathology*, 14(3), 581–611.
- Hurley, R. S., Losh, M., Parlier, M., Reznick, J. S., & Piven, J. (2007). The broad autism phenotype questionnaire. *Journal of Autism and Developmental Disorders*, 37(9), 1679–1690.
- Lainhart, J. E., & Lange, N. (2011). The biological broader autism phenotype. In D. Amaral, D. Geschwind, & G. Dawson (Eds.), *Autism spectrum disorders* (pp. 477–509). New York: Oxford University Press.
- Losh, M., Adolphs, R., & Piven, J. (2011). The broad autism phenotype. In D. Amaral, D. Geschwind, & G. Dawson (Eds.), *Autism spectrum disorders* (pp. 457–476). New York: Oxford University Press.
- Parr, J. R., & Le Couteur, A. (2011). The broader autism phenotype. In S. Boelte & J. Hallmayer (Eds.), *International experts answer questions on ASD*. Gottingen/Oxford: Hogrefe.
- Parr, J. R., Wittemeyer, K., & Le Couteur, A. S. (2011). Commentary: The broader autism phenotype implications for research & clinical practice. In D. Amaral, D. Geschwind, & G. Dawson (Eds.), *Autism spectrum disorders* (pp. 521–524). New York: Oxford University Press.

Broca's Aphasia

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Synonyms

[Nonfluent aphasia](#)

Short Description or Definition

Broca's aphasia is a language disorder that is characterized by limited, "telegraphic" spoken language output in the face of intact language comprehension skills. This condition is typically the result of damage to the left frontal lobe of the brain, often due to stroke, but may also result from traumatic brain injury or a degenerative neurological condition.

Categorization

Broca's aphasia is considered a "nonfluent" aphasia under larger aphasia classification systems due to the patient's lack of fluent speech output.

Epidemiology

Estimates of Broca's aphasia in the larger population are largely unknown, though it has been estimated that 80,000 people develop aphasia in the United States each year.

Natural History, Prognostic Factors, and Outcomes

The prognosis for individuals who are diagnosed with Broca's aphasia is largely dependent upon the severity of the condition. Often, people with Broca's aphasia do not completely recover fluent spoken language skills and need to develop compensatory strategies to manage the condition. It is thought that recovery is enhanced depending upon factors such as age of onset, health, education level, and how soon treatment takes place after brain damage has occurred.

assisting individuals with Broca's aphasia to transmit messages.

Family member and patient support groups are often a critical piece of the therapeutic process as the patient and family learn to manage the patient's changed mode of communication. Support groups are often key to recovery.

Please see ► [“Aphasia”](#) for a list of general treatment strategies for aphasia.

See Also

► [Aphasia](#)

Clinical Expression and Pathophysiology

Broca's aphasia is often due to damage in the left frontal lobe of the brain, also referred to as “Broca's area.” Patients demonstrate impaired, effortful speech/language output in the face of relatively intact comprehension skills.

References and Reading

- American Speech-Language-Hearing Association (ASHA). (2008). *Incidence and prevalence of speech, voice, and language disorders in adults in the United States*. Available from www.asha.org/research/reports/speech_voice_language.htm. Retrieved 5 Jan 2011.
- Barresi, B., Goodglass, H., & Kaplan, E. (2001). *The assessment of aphasia and related disorders*. Hagerstown: Lippincott Williams & Wilkins.
- Chapey, R. (2008). *Language intervention strategies in aphasia and related neurogenic communication disorders*. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins.
- Goodglass, H., Kaplan, E., & Barresi, B. (2000). *Boston diagnostic aphasia examination third edition (BDAE-3)* (3rd ed.). Austin: Pro-Ed.
- Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *The Boston naming test*. Philadelphia: Lea and Febiger.
- Kent, R. D. (1994). *Reference manual for communicative sciences and disorders: Speech and language*. Austin: Pro-Ed.
- Kertesz, A. (2006). *Western Aphasia battery- revised (WAB-R)*. Austin: Pro-Ed.
- Lapointe, L. L. (2004). *Aphasia and related neurogenic language disorders*. New York: Thieme Medical Publishers.
- National Institute on Deafness and Other Communication Disorders. (2008). *Aphasia*. Available from <http://www.nidcd.nih.gov/health/voice/aphasia.htm>. Retrieved 5 Jan 2011.

Evaluation and Differential Diagnosis

Please see ► [“Aphasia.”](#)

Treatment

Treatment for Broca's aphasia, as in other aphasias, is typically individualized and is based on the patient's profile of strengths and needs. Formal speech-language therapy is often recommended to address functional communication in a variety of settings in which patients are expected to communicate. Therapy goals are focused on maximizing the individual's ability to communicate effectively with peers and family members, given residual strengths. For individuals with Broca's aphasia, treatment strategies often focus on language output and naming as well as building sentence length, building on their intact comprehension skills. In addition, computer-assisted methods are beginning to show promise in

Brodmann's Area 4

► [Precentral Gyrus](#)

Brodmann's Areas

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Definition

Brodmann's areas (BA) are regions of the cerebral cortex defined by the layer-specific organization of cells. These areas were originally annotated by German neurologist Korbinian Brodmann using basic staining techniques and microscopy in 1909. He initially divided the human cortex into 43 distinct areas. Later studies confirmed these boundaries and identified the functional significance of each area. For example, BA 44 and 45 are part of the left frontal cortex and are important for the motor aspect of speech. Several BAs have been implicated in autism including BA9 (dorsolateral prefrontal cortex), BA 24 (right anterior cingulate gyrus), and BA44 (left inferior frontal gyrus).

See Also

- [Cerebral Cortex](#)
- [Neuroanatomy](#)

References and Reading

- Casanova, M. F., Buxhoeveden, D. P., Switala, A. E., & Roy, E. (2002). Minicolumnar pathology in autism. *Neurology*, 58(3), 428–432.
- Haznedar, M. M., Buchsbaum, M. S., Wei, T.-C., Hof, P. R., Cartwright, C., Bienstock, C. A., & Hollander, E. (2000). Limbic circuitry in patients with autism spectrum disorders studied with positron emission tomography and magnetic resonance imaging. *American Journal of Psychiatry*, 157(12), 1994–2001.
- Zilles, K., & Amunts, K. (2010). Centenary of Brodmann's map – conception and fate. *Nature Reviews Neuroscience*, 11(2), 139–145.

BRS

- [Behavior Rating Scale \(BRS\)](#)

Bruininks-Oseretsky Test of Motor Proficiency

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Synonyms

[BOT-2](#); [BOTMP](#)

Definition

The original version of the Bruininks-Oseretsky Test of Motor Proficiency was abbreviated as the BOTMP. The revised second edition is usually referred to as the BOT-2.

Description

The BOT-2 is designed to assess motor proficiency in children and adults from ages 4 to 21 years and 11 months. This was intended to cover the age range for children served by the American Individuals with Disabilities Education Act (IDEA). It is individually administered, standardized, and norm referenced. It is used for treatment planning and evaluation in clinical and school settings as well as for research. Physical and occupational therapists especially may find the test useful.

The Complete Form version of the BOT-2 includes 53 items based on activities such as cutting out a circle, copying a square, bouncing a ball, and standing on one leg. Items are organized into eight subtests and further categorized into four motor area composites and one comprehensive score. These composites are *strength and*

agility (running speed and agility + strength subtests, meant to measure control of the musculature of body involved in movement); *manual coordination* (manual dexterity and upper limb coordination subtests, meant to measure the ability to manually manipulate objects and the level of coordination in the hands and arms); *body coordination* (bilateral coordination and balance subtests, to measure large musculature control of posture, balance, as well as the sequential and simultaneous coordination of the lower and upper limbs); and *fine manual control* (fine motor precision and fine motor integration subtests, to measure the level of control and coordination of the hand and fingers by looking at an individual's ability to grasp, draw, and cut with scissors).

The nature of the measure makes it fairly easy to administer, as children tend to enjoy performing the variety of activities involved in the testing. The BOT-2 revision has made it much more adaptive for younger children, for instance by increasing the number of blocks to string and adding the balance beam to walk on. The entire battery of tests can take an hour to administer, but a 14-item Short Form of the test is available which only requires 20 min. The short form accounts for 96.3% of the variability in children ages 3–5, so it can be used as a substitute for the complete battery when appropriate. The test manual provides many clear pictures of the tasks being completed. However, scoring of the test is time-intensive, taking at least 20 min according to Deitz et al. (2007). Deitz et al. note that scoring for the BOT-2, although improved over the BOTMP, nonetheless is tedious, sometimes confusing, and easy to make errors. Norm lookup tables are also difficult to use.

Historical Background

The BOTMP was originally developed in Russia by Oseretsky in 1923 (Oseretsky 1923). When it was translated into English by Doll in 1946 (Doll 1946), it was known as the “Oseretsky Test of

Motor Proficiency.” However, because the test had been based on Oseretsky's personal observations of children, it had many problems relating to its psychometric properties. Multiple revisions were made in order to increase the reliability and validity of the measure, and the BOTMP represents the culmination of these revisions. The BOT-2 was published in 2005 with updated and revised materials, items, scales, and norms.

Psychometric Data

Criticisms of the original BOTMP included concerns about the normative sample being racially homogenous and functioning at normal levels both intellectually and motorically. A child's ability to understand and respond to instructions may have confounded motor skill development. Factor analyses showed that 14 of the 17 *fine motor* ability items loaded at significant levels on the general motor ability factor, implying that the BOTMP was not a good stand-alone measure for assessing fine motor abilities and that the grouping of tests into fine and gross motor skills was problematic. The creators of the BOT-2 revision set out to address these and other issues.

The normative sample for the BOT-2 included 1520 individuals from ages 4 to 21, with greater age differentiation for normative comparisons in younger children (in 1-year increments) up to a 5-year age increment for the adult sample. It was targeted to US Census Data from 2001 and included about 11% of children with special education status. Separate clinical samples were tested for autism/Asperger's, developmental coordination disorder, and mild-to-moderate mental retardation.

Interrater reliability reported by the developers of the BOT-2 is above .90 for all but the fine motor scale (adjuster $r = .86$). Test-retest reliability is good for the Total Composite and the Short Form totals, but generally less good (with substantial variability) for the other scale composites and item analyses. Deitz et al. (2007) therefore

recommend that the Composite scores be used wherever possible and that reliance on subscale scores is inadvisable.

Test developers utilized Confirmatory Factor Analysis to document a good fit for the four-factor model of the BOT-2, better than the two-factor (Fine vs. Gross Motor) structure of the original BOTMP. The three clinical samples all scored significantly lower than the normative sample on both the Complete and Short forms. Convergent validity was strong for the original BOTMP (adjusted $r = .80$ for composite scores); the Peabody Test of Developmental Motor Skills – Second Edition (adjusted r s ranging from .51 to .75 for subscales); and the Test of Visual Motor Skills – Revised (comparison of relevant fine motor skills adjusted $r = .74$).

Statistical modeling by Wuang et al. (2009) on a sample of 446 children diagnosed with intellectual disability found that the *manual coordination* and *strength + agility composites* fit the whole sample better than the *fine motor* and *body coordination* composites, which fit the lower-functioning end of the sample better than the higher-functioning end. Their analysis suggested elimination and/or restructuring of a number of items and scales to improve both reliability and discriminant validity.

Clinical Uses

Deitz et al. (2007) note that the inclusion of 11% special education students in the normative sample makes the BOT-2 less likely than its BOTMP predecessor to score children with motor disabilities as significantly below average.

The BOTMP has been used to characterize motor problems in individuals diagnosed with Autism Spectrum Disorders. One study (Ghaziuddin and Butler 1998) compared BOTMP motor coordination between children diagnosed with autism, Asperger's syndrome (AS), and pervasive developmental disorder not otherwise specified (PDDNOS). Of the three groups, those with autism were the most clumsy, followed by those with AS

and then those with PDDNOS. However, there was not a significant difference between the autism group and the AS group. These results indicate that caution should be used before including clumsiness as a diagnostic criterion for only one of the disorders. Dewey et al. (2007), using the BOTMP Short Form, found particular impairment in gestural performance in ASD relative to other clinical groups (developmental motor coordination and ADHD). In the context of generally impaired motor performance for all the clinical groups, Dewey et al. suggest that gestural impairments in autism are not solely attributable to motor problems.

The test is also frequently used in studies of developmental coordination disorders, with a few studies of ADHD. There are very few published studies that have used the BOT-2 instead of the original BOTMP.

See Also

► [Bender Visual-Motor Gestalt Test II](#)

References and Reading

- Beitel, P., & Mead, B. J. (1980). Bruininks-Oseretsky test of motor proficiency: A viable measure for 3- to 5-yr-old children. *Perceptual and Motor Skills*, 5, 919–923.
- Bruininks, R. H. (1978). *Bruininks-Oseretsky test of motor proficiency – Owner's manual*. Circle Pines: American Guidance Service.
- Bruininks, R., & Bruininks, B. (2005). *Bruininks-Oseretsky test of motor proficiency* (2nd ed.). Minneapolis: NCS Pearson.
- Deitz, J. C., Kartin, D., & Kopp, K. (2007). Review of the Bruininks-Oseretsky test of motor proficiency, second edition (BOT-2). *Physical & Occupational Therapy in Pediatrics*, 27, 87–102.
- Dewey, D., Cantell, M., & Crawford, S. G. (2007). Motor and gestural performance in children with autism spectrum disorders, developmental coordination disorder, and/or attention deficit hyperactivity disorder. *Journal of International Neuropsychological Society*, 13, 246–256.
- Doll, E. A. (1946). *The Oseretsky tests of motor proficiency*. Circle Pines: American Guidance Service.
- Ghaziuddin, M., & Butler, E. (1998). Clumsiness in autism and Asperger syndrome: A further report. *Journal of Intellectual Disability Research*, 44, 43–48.

- Hattie, J., & Edwards, H. (1987). A review of the Bruininks-Oseretsky test of motor proficiency. *British Journal of Educational Psychology*, 57, 104–113.
- Oseretsky, N. I. (1923). A metric scale for studying the motor capacity of children. [In Russian].
- Wuang, Y.-P., Lin, Y.-H., & Su, C.-Y. (2009). Rasch analysis of the Bruininks-Oseretsky test of motor proficiency-second edition in intellectual disabilities. *Research in Developmental Disabilities*, 30, 1132–1144.

Bruxism

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Synonyms

Sleep bruxism

Definition

Bruxism is the nonfunctional and involuntary grinding, gnashing, clenching, or tapping of teeth. Bruxism is considered to be common among individuals with developmental delays or disabilities, including those diagnosed with autism spectrum disorders. Bruxism is classified as nocturnal (occurring during sleep) or diurnal (occurring while awake). Bruxism can be audible when teeth are grinding or gnashing or inaudible when teeth are clenching. Many are not aware of their bruxism, but some will develop symptoms such as tooth sensitivity, headaches, or jaw pain. Bruxism is considered to be a psychophysiological and sleep disorder influenced by anatomical and biological (i.e., dental abnormalities), neurological (i.e., mental retardation), and/or psychological (i.e., stress, trauma, anxiety) factors. Studies examining effective treatments for bruxism in individuals with developmental disabilities are limited to date; however, dental-based approaches, biofeedback, behavior therapy, habit

reversal, and stress management appear to be common interventions.

See Also

- Habit Reversal
- Tics

References and Reading

- Allen, K. D., & Polaha, J. (2006). Analysis and treatment of oral-motor repetitive behavior disorders. In D. W. Woods & R. G. Miltenberger (Eds.), *Tic disorders, trichotillomania, and other repetitive behavior disorders: Behavioral approaches to analysis and treatment* (pp. 269–296). New York: Springer.
- Glaros, A. G., & Epkins, C. C. (1995). Habit disorders: Bruxism, trichotillomania, and tics. In M. C. Roberts (Ed.), *Handbook of pediatric psychology* (2nd ed., pp. 558–574). New York: The Guilford Press.
- Glaros, A. G., & Rao, S. M. (1977). Bruxism: A critical review. *Psychological Bulletin*, 4, 767–781.
- Mindell, J. A., & Owen, J. A. (2010). Sleep related rhythmic movements: Bruxism. In *A clinical guide to pediatric sleep: Diagnosis and management of sleep problems* (2nd ed., pp. 90–93). Philadelphia: Lippincott Williams and Wilkins.
- PubMed Health. (2010, February 22). Bruxism: Teeth grinding and clenching. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0002386/>
- US Department of Health and Human Service. (2000). *Oral health in America: A report of the Surgeon General*. Rockville: US Department of Health and Human Services, National Institute of Health and Human Services, National Institute of Dental and Craniofacial Research, National Institute of Health.

BSE

- Behavior Summarized Evaluation-Revised (BSE-R)

BSE-R

- Behavior Summarized Evaluation-Revised (BSE-R)

Buckhannon Versus West Virginia Department of Health and Human Resources: Definition of Prevailing Party

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Definition

In General

The issue in this case was whether the prevailing party in a claim for violating the Americans with Disabilities Act (ADA) should be awarded attorney's fees under the catalyst theory. The Supreme Court, quoting Black's Law Dictionary 1145 (7th ed. 1999), defines "prevailing party" as "[a] party in whose favor a judgment is rendered, regardless of the amount of damages awarded (in certain cases, the court will award attorney's fees to the prevailing party)." The catalyst theory provides fees to a party where the case serves as a "catalyst" for legislative change. The Supreme Court ruled that the "catalyst theory" is not an appropriate basis for attorney's fees under certain civil rights cases, including under the ADA. The prevailing party is only entitled to attorney's fees when they are victorious in court and awarded a judgment in court.

Implications for ASD Students

Buckhannon has implications for those with ASD. First, in the case of discrimination under the ADA, parents may be less willing to bring cases because of the expense of bringing a lawsuit. The parent would not be guaranteed expensive attorney fees if they end up prevailing outside of a courtroom. Only cases heard in court would be awarded attorney's fees. Second, IDEA (Individuals with Disabilities Education Act) has made fees available to parents who win in either administrative or

judicial proceedings. If IDEA follows the *Buckhannon* decision, then this fee payment for parents would become obsolete if a case is heard in an informal proceeding or dismissed because of a change in law.

Litigation Strategies

Buckhannon specifically dealt with an assisted living facility which failed a fire inspection. *Buckhannon* was ordered to be shut down, but filed suit under the ADA. After filing suit, the state legislature removed the language that created the problem. But, because of the Supreme Court decision, *Buckhannon* was not entitled to an award of attorney's fees. Most special education cases settle either because of the speed necessary to allow the child to continue his/her education or the school district changes its behavior before a decision is even made. Under *Buckhannon*, parents would not be able to recover attorney fees. It might be more appropriate to try to mediate between the parents and school district, so that in the mediation agreement the parents can be entitled to some reasonable attorney's fees.

See Also

► [Americans with Disabilities Act](#)

References and Reading

Buckhannon Bd. & Care Home v. W. Va. Dep't of Health & Human Res., 532 U.S. 598 (U.S. 2001).
Weber, M. C. (2004). Litigation under the Individuals with Disabilities Education Act After *Buckhannon* Board & Care Home Inc. v. West Virginia Department of Health & Human Resources. *Ohio State Law Review*, 65, 357–411.

Bulimia Nervosa

► [Eating Disorders](#)

Bullying

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Synonyms

[Peer victimization](#); [School harassment](#)

Short Description or Definition

Bullying is an aggressive behavior perpetrated by those who hold and/or try to maintain a dominant position over others (Morita 1985). It is intended to cause mental and/or physical harm or suffering to another: it is a repetitive behavior and almost always involves an imbalance of power between victim and perpetrator in which the victim is usually not able to defend himself/herself (Farrington 1993). Bullying is also the most common form of school violence.

Bullying can take various forms including: exclusion, verbal abuse, physical abuse, and/or coercion (Kim et al. 2004). Bullying may be “direct” or “indirect.” Direct bullying includes physical and verbal aggression, such as kicking, threatening, name-calling, and insulting. Indirect or covert/relational bullying includes social exclusion/isolation, such as ignoring, cliques, rumor-mongering, insulting, and humiliating with the spread of embarrassing information about an individual (van der Wal et al. 2003).

Students are involved in bullying as victims, perpetrators, victim-perpetrators, or bystanders. Victims may experience many forms of bullying with considerable variability in form. Some students may be involved in bullying as both a victim and perpetrator; that is, they are bullied by one student or a group of students and may also

individually or in a group bully other students (Schwartz 2000).

With recent advances in information technology, bullying has added cyberspace to the schoolyard and neighborhood as sites for bullying. In cyberspace, bullying can take place anonymously, without overtly identifying the perpetrators. Further, children or adolescents may not be safe from bullying even in their homes since unkind text messages, hateful e-mails, videos, or provocatively manipulated messages and materials can reach them 24 h per day, 7 days a week (Pridgen 2009).

Epidemiology/Clinical Expression

Children and adolescents with Autism Spectrum Disorder (ASD) have essential difficulty in reciprocal social interaction along with impairment in communication skills (American Psychiatric Association [APA] 1994; Caronna et al. 2008; Frith and Hill 2004; Gura et al. 2011; Kelly et al. 2008; van Roekel et al. 2010). These difficulties make those with ASD especially vulnerable for involvement with bullying as victims and/or perpetrators since bullying is a form of dynamic and complex social interactions (Cappadocia et al. 2011; Sharp and Cowie 1994). Children with ASD also often demonstrate stereotyped behavior and a limited range of interests, often in unusual topics or objects that can make them stand out as quite different from their peers: this often puts them in the position of becoming targets for ridicule (Cappadocia et al. 2011; Gray 2004). Other challenges often facing children with ASD that include unusual sensory responses such as hyper/hyposensitivity to auditory, olfactory, tactile, or visual stimuli; problems in motor coordination; and poor performance in physical education, can also contribute to the risk of becoming a target of the peer victimization (Bejerot et al. 2011; Kelly et al. 2008).

Perpetrators of bullying intend to cause mental and/or physical harm or suffering on other children; perpetrators identify what would cause pain to their victims and, then, plan and execute their actions accordingly. However, it is difficult for

children and adolescents with ASD to bully others due to their difficulties in understanding and using the rules governing social behaviors and perspectives of other people. Nevertheless, their behaviors may be regarded as bullying for several reasons. First, children and adolescents with ASD may have increased levels of aggressive behaviors (Mandell et al. 2005; van Roekel et al. 2010). Since bullying is a form of aggression, those with ASD who have increased level of aggression may be considered to be bullying other children or adolescents (van Roekel et al.). Second, because adolescents with ASD have limited insight into social processes (Frith and Hill 2004; van Roekel et al. 2010), they may not be aware of the consequences of their own behavior or words; some of these behaviors may be regarded as bullying (van Roekel et al.). For example, children with ASD may say brutally honest things or violate the physical space of others to the extent that they cause discomfort, even though it may not be intended to be bullying (Montes and Halterman 2007; van Roekel et al. 2010).

Although the severity of ASD symptoms is negatively correlated with successful social inclusion and peer relationships, even children and adolescents with high-functioning ASD continue to struggle with social competence as they age (Brauminger and Kasari 2000; Cappadocia et al. 2011; Orsmond et al. 2004); as a result, even with improvement in overall functioning, individuals with ASD remain at increased risks for bullying experiences (Cappadocia et al. 2011).

Indeed, several previous studies have reported that children or adolescents with ASD showed increased involvement in bullying as victims or perpetrators (Cappadocia et al. 2011; Little 2001, 2002; Twyman et al. 2010; van Roekel et al. 2010). Little used a website survey of 411 parents of children with Asperger's disorder (AD) (75% of subjects) or nonverbal learning disorder (25%); they reported that up to 75% of the children with AD were bullied within previous year. The younger children, boys, and children with ASD had greater risk for victimization (Little 2001). In another study of 187 adolescents with ASD attending a special secondary education school,

van Roekel et al. also reported that 7–30% were victimized more than once a month, and 19–46% bullied others, depending on the informants (teacher, peer, or self-report of bullying) (van Roekel et al. 2010). Samson et al. showed individuals with Autistic Disorder recruited from clinics in Germany and Switzerland (40 with autism and 83 control), who reported higher rates of experiencing teasing or being ridiculed, compared to the control group who did not have ASD diagnoses (Samson and Huber 2010). Interestingly, Shtayermann measured the bullying experiences of 10 adolescents or young adults with Asperger's Disorder using mailed or online self- or parent's questionnaires, and reported a negative correlation between the severity of AD symptoms and victimization. The authors considered that children and adolescents with milder AD symptoms received lesser support and supervision from teachers and/or parents than those with severe symptom, leading to greater risks for victimization due to "under-surveillance" by adults (Shtayermman 2007). Although there are significant limitations in his study, including the small number of samples and survey accuracy, this finding suggests that children and adolescents with ASD, irrespective of symptom severity, require appropriate support from caregivers and teachers in order to prevent peer victimization. Additionally, Volker et al., using a standardized behavioral rating scale, demonstrated that children and adolescent with high-functioning ASD recruited from those awaiting participating in social intervention study ($N = 62$) showed increased scores for bullying participation when compared to a control group, even after being controlled for their IQs (Volker et al. 2010).

When examining the experience of school bullying in children and adolescents with ASD, the school setting likely plays an important role: there are advantages and shortcomings in different school settings for children and adolescents with ASD (Burack et al. 1997; Laugeson et al. 2009). On one hand, regular classroom has been associated with increases in the complexity of interactions and decreases in nonsocial activity, in comparison to special education settings. On the other hand, these individuals report often feeling

lonelier and having poorer quality friendships than their typically developing classmates (Capps et al. 1996; Laugeson et al. 2009; Sigman and Ruskin 1999). Another study also implies important feature that in a special educational setting, teachers report higher rates of bullying among students with ASD than those without (van Roekel et al. 2010).

In general, bullying is associated with various psychological problems as consequences or antecedents to bullying experiences (Barker et al. 2008; Kim et al. 2005, 2006; Salmon et al. 1998; Srabstein and Piazza 2008); children and adolescents with ASD who are also involved with bullying are not exceptions. In a study of 192 children diagnosed with ASD recruited from the website for parents of children with ASD or the school system, using parental report of psychopathology, Cappadocia et al. reported that ASD children who were bullied once or more per week had higher levels of anxiety; hyperactivity; self-injurious, stereotypic behaviors; and oversensitivity when compared to those not bullied or bullied less than once per week (Cappadocia et al. 2011). Additionally, correlations between peer victimization and suicidal ideation were reported in adolescents with AD (Asperger's Disorder) (Shtayermman 2007).

Kelly et al. reported that peer victimization was not only directly related to severity of ASD symptoms, but also that poor peer relationship was associated with anxiety and depression symptoms measured by parental survey in 322 children with ASD recruited from the clinics. This suggests that not only do ASD symptoms increased risks for peer victimization but also that victimization may worsen associated symptoms in children with ASD (Kelly et al. 2008). Such bidirectional impacts of social problems and peer victimization on each other have been already demonstrated in a general population of adolescents in a longitudinal study (Kim et al. 2006).

In addition to ASD severity, cognitive function may play roles in the risks for the involvement in bullying and development of psychopathological consequences from bullying experiences. For example, children with milder forms of ASD or higher cognitive function may be more accurate in

recognizing bullying when they are bullied while those with more severe ASD or lower levels of cognitive function might not; this may lead to more serious adverse consequences from bullying experiences in the higher functioning groups (Sofronoff et al. 2011).

The experience of bullying in childhood and adolescence can have long-term sequelae, including in adulthood. Samson et al. recruited 40 adults diagnosed with ASD and 83 adults without ASD to compare their recollection of bullying experience in their childhood and/or youth; compared to the control group, the individuals with Asperger's Disorder report not only higher rates of recollections of being ridiculed or teased in their childhood or youth, but also fear for being ridiculed at present, indicating that the psychological damage of school bullying persists beyond the school years (Samson and Huber 2010).

Evaluation and Differential Diagnosis

Given the high prevalence of bullying and its association with psychiatric and psychological morbidities in children and adolescents with ASD, comprehensive and careful attention and assessment is required for prevention, early identification, and intervention with bullying in ASD children and adolescents.

Due to their impairments in making and recognizing social interactions, the utility of self-report as a tool for identifying bullying experiences in the ASD population may be limited. Indeed, van Roekel et al. showed that teachers reported higher prevalence of bullying compared to peer- and self-reports which indicated much lower rates of school bullying in this population: teachers reported 27% of adolescents frequently involved in school bullying (more than once a week), whereas adolescents themselves reporting only 12% in 230 adolescents with ASD (van Roekel et al. 2010). This was distinctly different from the findings in children and adolescents without ASD, when on average self-report or peer nomination measurement report 35–48% of involvement in bullying as victims and/or perpetrators, but teacher or parent report only have 10–18%

(Cleary 2000; Hunter et al. 2004; Ladd and Kochenderfer-Ladd 2002; Nansel et al. 2001; Rønning et al. 2009). Such a discrepancy may stem from the combination of two factors: First, teachers may have missed opportunities to witness bullying incidences among typically developing children since bullying usually occur in the absence of adults supervision; children and adolescents with ASD receive higher levels of supervision and monitoring from teachers, resulting in more opportunities for teachers to observe peer interactions and bullying in this population. Second, individuals with ASD have difficulties understanding the mental states of other people, and consequently in understanding the intentions of others (Frith and Hill 2004; van Roekel et al. 2010). It may be difficult for children and adolescents with ASD to recognize or identify bullying incidents due to their limited social insights, unlike typically developing children (van Roekel et al.). Therefore, comprehensive assessment with multiple informants including caregivers, teachers, and peers in addition to self-report is crucial for the identification of bullying experience in children and adolescents with ASD (Ladd and Kochenderfer-Ladd 2002; Mandell et al. 2005).

Treatment

Screening for bullying experience and symptoms for ASD in primary care and community setting is an important first step for early identification and intervention since children and adolescents with ASD are at increased risks for the involvement of bullying (Gura et al. 2011; Tantam and Girgis 2009).

Careful school placement is crucial for children and adolescents with ASD. Regular education classroom placement has resulted in mixed outcomes for individuals with ASD (Burack et al. 1997; Laugeson et al. 2009). As mentioned earlier, mainstream classroom placement is associated with the increase of the complexity of interactions and decrease in nonsocial activity in comparison to special education. On the other hand, placement in special education classroom can enhance teachers' capacities for careful

attention and intervention for those students with ASD and protection from undesirable social stigma and traumatization (Laugeson et al.). Having close friends in a classroom is protective of becoming a target of bullying (Cappadocia et al. 2011; Nansel et al. 2001; Williams and Guerra 2007). Therefore, interventions such as social skill training to help these children have better friendship will decrease their risks for peer victimization (Laugeson et al. 2009).

Children and adolescents with ASD have various comorbid psychopathologies including depression, anxiety, and withdrawal, which are reported to be associated with increased risks for the involvement of bullying in general population (Volker et al. 2010). Appropriate assessment and interventions for comorbid conditions in ASD children is warranted (Volker et al.).

When a child is being bullied, particularly a child with a disability, adult support is crucial. Through scaffolding, adults can support children to acquire and develop important social skills such as: adaptive emotional and behavioral regulation strategies and coping skills, identifying and engaging with supportive peers, problem solving, and communicating assertively (Cappadocia et al. 2011; Cummings et al. 2006). Recent research supports the effectiveness and importance of parent-assisted learning with respect to developing social skills among children with ASD (Cappadocia et al. 2011; Frankel et al. 2010; Laugeson et al. 2009). This relationship scaffolding, individualized for each child to capitalize on his or her strengths and support weaknesses, can help the child develop coping skills that may reduce the impact of the bullying on the victimized child and in turn reduce the likelihood of bullying. It is important to encourage children to seek help from a trusted adult and continue to seek help until they find an adult who is willing to listen and offer protection and support. Once the adult understands the particulars of the bullying episodes (e.g., when and where), safe places and safe people can be discussed to minimize the risk for bullying to occur (Cappadocia et al. 2011; Cummings et al. 2006).

Finally, validated effective antibullying campaigns/interventions to decrease bullying in

home, schools, and communities will exert preventive effect on bullying not only for children with ASD but for all children receiving such interventions (Olweus 1994; Olweus and Limber 2010; Vreeman and Carroll 2007).

Understanding the relationships between ASD and bullying has been limited due to the shortcomings of previous studies, including small sample size, limited sampling methods, and/or inadequate measurement of bullying (Mandell et al. 2005). Future study should focus on incidence/prevalence of bullying, the impact of bullying experiences on the natural course of ASD, associations between bullying experiences and other comorbid psychopathology, and development and assessment of intervention programs in larger population-based samples of children and adolescents with ASD.

Conclusion

Bullying is common among all children, but the children with ASD are at even greater risk of this harmful experience. And, just as is the case for typically developing children, reduction of bullying enhances developmental prospects for all children, including those with ASD. While ASD may not be preventable at this time, we can reduce or even prevent bullying experiences for children with ASD, as we can and must for all children.

References and Reading

- American Psychiatric Association. (1994). *DSM-IV*. Washington, DC: American Psychiatric Association.
- Barker, E. D., Arseneault, L., Brendgen, M., Fontaine, N., & Maughan, B. (2008). Joint development of bullying and victimization in adolescence: Relations to delinquency and self-harm. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(9), 1030–1038.
- Bejerot, S., Edgar, J., & Humble, M. B. (2011). Poor performance in physical education – A risk factor for bully victimization. *Acta Paediatrica*, 100, 413–419.
- Brauminger, N., & Kasari, C. (2000). Loneliness and friendship in high functioning children with autism. *Child Development*, 71, 447–456.
- Burack, J. A., Root, R., & Zigler, E. (1997). *Inclusive education for students with autism: Reviewing ideological, empirical, and community considerations*. New York: Harper Collins.
- Cappadocia, M. C., Weiss, J. A., & Pepler, D. (2011). Bullying experiences among children and youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-011-1241-x>.
- Capps, L., Sigman, M., & Yirmiya, N. (1996). Self-competence and emotional understanding in high-functioning children with autism. *Development and Psychopathology*, 7, 137–149.
- Caronna, E. B., Milunsky, J. M., & Tager-Flusberg, H. (2008). Autism spectrum disorders: Clinical and research frontiers. *Archives of Disease in Childhood*, 93, 518–523.
- Cleary, S. D. (2000). Adolescent victimization and associated suicidal and violent behaviors. *Adolescence*, 35(140), 671–682.
- Cummings, J. G., Pepler, P., Mishna, F., & Craig, W. (2006). Bullying and victimization among students with exceptionalities. *Exceptionality Education*, 16, 193–222.
- Farrington, D. P. (1993). Understanding and preventing bullying. In M. Tonry (Ed.), *Crime and justice: A review of research*. Chicago: University of Chicago Press.
- Frankel, F., Myatt, R., Whitham, C., Gorospe, C., & Laugeson, E. A. (2010). A controlled study of parent-assisted children's friendship training with children having autism spectrum Disorders. *Journal of Autism and Developmental Disorders*, 40, 827–842.
- Frith, U., & Hill, E. (2004). *Autism: Mind and brain*. New York: Oxford University Press.
- Gray, C. (2004). Gray's guide to bullying parts I-III. *Jenison Autism Journal*, 16(1), 2–19.
- Gura, G. F., Champagne, M. T., & Blood-Siegfried, J. E. (2011). Autism spectrum disorder screening in primary care. *Journal of Developmental and Behavioral Pediatrics*, 32, 48–51.
- Hunter, S. C., Boyle, J. M. E., & Warden, D. (2004). Help seeking amongst child and adolescent victims of peer-aggression and bullying: The influence of school-stage, gender, victimization, appraisal, and emotion. *British Journal of Educational Psychology*, 74, 375–390.
- Kelly, A. B., Garnett, M. S., Attwood, T., & Peterson, C. (2008). Autism spectrum symptomatology in children: The impact of family and peer relationships. *Journal of Abnormal Child Psychology*, 36, 1069–1081.
- Kim, Y. S., Koh, Y., & Leventhal, B. L. (2004). Prevalence of school bullying in Korean middle school students. *Archives of Pediatric and Adolescent Medicine*, 158, 737–741.
- Kim, Y. S., Koh, Y., & Leventhal, B. (2005). School bullying and suicidal risk in Korean middle school students. *Pediatrics*, 115(2), 357–363.
- Kim, Y. S., Leventhal, B. L., Koh, Y., Hubbard, A., & Boyce, W. T. (2006). School bullying and youth violence: Causes or consequences of psychopathologic

- behavior? *Archives of General Psychiatry*, 63(9), 1035–1041.
- Ladd, G. W., & Kochenderfer-Ladd, B. (2002). Identifying victims of peer aggression from early to middle childhood: Analysis of cross-informant data for concordance, estimation of relational adjustment, prevalence of victimization, and characteristics of identified victims. *Psychological Assessment*, 14(1), 74–96.
- Laugeson, E. A., Frankel, F., Mogil, C., & Dillon, A. R. (2009). Parent-assisted social skills training to improve friendships in teens with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 596–606.
- Little, L. (2001). Peer victimization of children with Asperger spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40, 995–996.
- Little, L. (2002). Middle-class mother's perceptions of peer and sibling victimization among children with Asperger's syndrome and nonverbal learning disorders. *Issue in Comprehensive Pediatric Nursing*, 25, 43–57.
- Mandell, D. S., Walrath, C. M., Manteuffel, B., Sgro, G., & Pinto-Martin, J. A. (2005). The prevalence and correlates of abuse among children with autism served in comprehensive community-based mental health settings. *Child Abuse & Neglect*, 29, 1359–1372.
- Montes, G., & Halterman, J. S. (2007). Bullying among children with autism and the influence of comorbidity with ADHD: A population-based study. *Ambulatory Pediatrics*, 7, 253–257.
- Morita, Y. (1985). *Sociological study on the structure of bullying group*. Osaka: Department of Sociology, Osaka City University.
- Nansel, T. R., Overpeck, M., Pilla, R. S., Ruan, W. J., Simons-Morton, B., & Scheidt, P. (2001). Bullying behaviors among us youth: Prevalence and association with psychosocial adjustment. *Journal of the American Medical Association*, 285(16), 2094–2100.
- Olweus, D. (1994). Bullying at school: Basic facts and effects of a school based intervention program. *Journal of Child Psychology and Psychiatry*, 35(7), 1171–1190.
- Olweus, D., & Limber, S. P. (2010). Bullying in school: Evaluation and dissemination of the olweus bullying prevention program. *The American Journal of Orthopsychiatry*, 80(1), 124–134.
- Orsmond, G. I., Krauss, M. W., & Seltzer, M. (2004). Peer relationships and social and recreational activities among adolescents and adults with autism. *Journal of Autism and Developmental Disorders*, 34, 245–256.
- Pridgen, B. (2009). Book forum: Cyberbullying: Bullying in the digital age. *Journal of the American Academy of Child & Adolescent Psychiatry*, 48(3), 344–346.
- Rønning, J. A., Sourander, A., Kumpulainen, K., Tamminen, T., Niemelä, S., Moilanen, I., et al. (2009). Cross-informant agreement about bullying and victimization among eight-year-olds: Whose information best predicts psychiatric caseness 10–15 years later? *Social Psychiatry and Psychiatric Epidemiology*, 44(1), 15–22.
- Salmon, G., James, A., & Smith, D. M. (1998). Bullying in schools: Self reported anxiety, depression, and self-esteem in secondary school children. *BMJ*, 317(3), 924–925.
- Samson, A. C., & Huber, O. (2010). Teasing, ridiculing and the relation to the fear of being laughed at in individuals with Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 41, 475–483.
- Schwartz, D. (2000). Subtypes of victims and aggressors in children's peer groups. *Journal of Abnormal Child Psychology*, 28(2), 181–192.
- Sharp, S., & Cowie, H. (1994). *School bullying: Insights and perspectives*. London: Routledge.
- Shtayermman, O. (2007). Peer victimization in adolescents and young adults diagnosed with Asperger's syndrome: A link to depressive symptomatology, anxiety symptomatology and suicidal ideation. *Issue in Comprehensive Pediatric Nursing*, 30, 87–107.
- Sigman, M., & Ruskin, E. (1999). Continuity and change in the social competence of children with autism, Down syndrome, and developmental delays. *Monographs of the Society for Research in Child Development*, 64, 114.
- Sofronoff, K., Dark, E., & Stone, V. (2011). Social vulnerability and bullying in children with Asperger syndrome. *Autism*, 15(3), 355–372.
- Srabstein, J., & Piazza, T. (2008). Public health, safety and educational risks associated with bullying behaviors in American adolescents. *International Journal of Adolescent Medicine and Health*, 20(2), 223–233.
- Tantam, D., & Girgis, S. (2009). Recognition and treatment of Asperger syndrome in the community. *British Medical Bulletin*, 89, 41–62.
- Twyman, K. A., Saylor, C. F., Eds, D. S., Macias, M. M., Taylor, L. A., & Spratt, E. (2010). Bullying and ostracism experiences in children with special health care needs. *Journal of Developmental and Behavioral Pediatrics*, 31, 1–8.
- van der Wal, M. F., de Wit, C. A. M., & Hirasing, R. A. (2003). Psychosocial health among young victims and offenders of direct and indirect bullying. *Pediatrics*, 111(6), 1312–1317.
- van Roekel, E., Scholte, R. H. J., & Didden, R. (2010). Bullying among adolescents with autism spectrum disorders: Prevalence and perception. *Journal of Autism and Developmental Disorders*, 40, 63–73.
- Volker, M. A., Lopata, C., Smerbeck, A. M., Knoll, V. A., Thomeer, M. L., Toomey, J. A., et al. (2010). BASC-2 PRS profiles for students with high-functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40, 188–199.
- Vreeman, R. C., & Carroll, A. E. (2007). A systematic review of school-based interventions to prevent bullying. *Archives of Pediatrics & Adolescent Medicine*, 161, 78–88.
- Williams, K. R., & Guerra, N. G. (2007). Prevalence and predictors of internet bullying. *Journal of Adolescent Health*, 41, 14–21.

Bupropion

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Synonyms

[Wellbutrin](#); [Zyban](#)

Definition

Bupropion is a novel antidepressant that is believed to block reuptake of dopamine. It is indicated for the treatment of depression ([Wellbutrin](#)) and for smoking cessation ([Zyban](#)). It has also been evaluated as a treatment for attention deficit/hyperactivity disorder in children and adults. It has not been evaluated systematically in children or adults with autism.

See Also

► [Antidepressants](#)

References and Reading

Martin, A., Scahill, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford.

Buspar

► [Buspirone](#)

Buspirone

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Synonyms

[Buspar](#); [Vanspar](#)

Definition

Buspirone is an antianxiety medication that is an agonist for serotonin 1A receptor. Unlike benzodiazepines, buspirone does not directly affect a GABA system and is not habit-forming. There is limited information on the use of buspirone in children and only one trial in adolescents with pervasive developmental disorders. In that study, buspirone appeared to be only modestly beneficial for disruptive and agitated behavior.

See Also

► [Anxiolytics](#)

References and Reading

Buitelaar, J. K., van der Gaag, R. J., & van der Hoeven, J. (1998). Buspirone in the management of anxiety and irritability in children with pervasive developmental disorders: Results of an open-label study. *The Journal of Clinical Psychiatry*, 59(2), 56–59.

Martin, A., Scahill, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.

CABS

► [Clancy Behavior Scale](#)

California Verbal Learning Test, Children's Version (CVLT-C)

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Synonyms

[CVLT – Children's Version](#); [CVLTC](#); [CVLT-C](#)

Description

The California Verbal Learning Test-Children's Version (CVLT-C; Delis et al. 1994) is an examination of auditory and verbal learning for children between the ages of 5 years and 16 years 11 months. The test makes use of familiar visual categories to generate a measure of short- and long-term memory performance. Encoding and recall are examined via the use of single words verbally presented in the context of “a shopping list” over the course of eight total trials spanning

15–20 min with an additional 20-min period to accommodate delayed recall testing.

During the first five trials of CVLT-C, a list of 15 items consisting of three semantic categories (fruit, clothing, and toys), labeled “list A” or “the Monday list,” is read aloud to the child and he or she is asked to recall as many items as possible following each presentation. During the sixth trial, a second 15-item list containing new words that belong to one of the categories from the original list (fruits) as well as words from two new categories with semantic similarities (furniture and sweets) from the original list are presented as “list B” or “the Tuesday list” to the child as an interference task. The child is then asked to recall as many words as possible. After completion of the list B trial, the child is then asked to recall words from list A without presentation of the items. In the seventh trial, list categories are used as cues to elicit recall from the original list via prompts from the examiner such as “Tell me all the things to wear in the Monday list.” Following trial 7 is a 20-min break from the task during which time the child can complete nonverbal tasks or participate in other activities that provide moderate distraction. Following this “long-delay” interval, the child is asked to recall as many words as he or she can from list A (long-delay free-recall trial), asked to recall words from list A after being provided with the categorical cues (long-delay cued-recall trial), and finally read a 45-item list aloud and asked to indicate whether

or not each word was on list A (yes/no recognition trial). Responses are recorded and documented by the examiner during every trial.

A complete administration of the CVLT-C produces data on eight recall measures, eight learning characteristics, four areas of recall errors, four recognition measures, and five contrast measures. This includes information concerning encoding strategies for success over time as well as the characteristics of errors that occur. In addition to generating information on the quantity of items accurately recalled after each of the eight testing trials, the CVLT-C allows for the detailed examination of characteristics related to acquisition methods utilized during the learning process. Characteristics related to the learning process are examined through the use of learning strategy variables and contrast variables.

The learning strategy variables aid in outlining the characteristics of acquisition and encoding that progress throughout the course of the examination. They include semantic clustering (i.e., consecutive words from the same category), serial clustering (i.e., words recalled in the same order in which they were presented), primacy recall (i.e., percentage of words recalled from the first five items of the list), middle recall (i.e., percentage of words recalled from the middle five items of the list), recency recall (i.e., percentage of words recalled from the last five items of the list), learning slope (i.e., the average number of new words recalled per learning trial), consistency (i.e., percentage of words recalled once that were also recalled on the following trial), recognition hits (i.e., number of words correctly identified as belonging to list A during the recognition trial), and discriminability (i.e., accuracy of distinguishing target words in list A from distraction words in list B). Characteristics of errors are also calculated with regard to perseveration (i.e., words repeated in a trial), free intrusions (i.e., extra-list intrusions on all free-recall trials), cued intrusions (i.e., extra-list intrusions on the cued-recall trials), total intrusions (i.e., extra-list intrusions on all trials), false-positives (i.e., words incorrectly identified as list A items during the recognition trials), and response bias (i.e., the tendency to identify words as belonging on the

target list during recognition trials). The learning slope variable, in particular, allows for a thorough examination of specific learning characteristics that may be evident across differing presentations of clinical populations. Deficits in areas related to learning (i.e., flat learning slope across trials with low amounts of new words learned), encoding (e.g., poor trial 1 performance followed by a normative learning slope), or sustaining focus (i.e., normative recall on initial trials with poor recall on later trials) can be identified with the learning slope, allowing for the closer inspection of learning characteristics and discrimination of other possible domains of learning that may be affected (Spren and Strauss 1998). Children with Down syndrome, attention deficit hyperactivity disorder (ADHD), and other disorders have demonstrated distinct and differentiated characteristics of learning slope in clinical populations (Delis et al. 1994).

The CVLT-C contrast variables (Donders 1999) aid in the identification of trial discrepancies and learning differences that occur throughout the learning process. These include aspects of encoding related to proactive interference (i.e., the contrast between list B recall and list A trial 1 recall), retroactive interference (i.e., the contrast between list A short-delay free recall and list A trial 5), rapid forgetting (i.e., contrast between list A long-delay free recall and list A short-delay recall), and retrieval problems (i.e., contrast between discrimination trial and list A long-delay free recall).

Historical Background

Delis et al. (2000) observed that while there are a variety of verbal learning instruments that measure the amount of material that is recalled, far fewer examine the processes by which the information is learned and retrieved. Construction of CVLT-C in 1994 followed the same process-oriented approach of the original California Verbal Learning Test (CVLT) for adults (Delis et al. 1987). For construction of the task, selection of the target words themselves was chosen based on their frequency of occurrence in the English

language as well as the frequency of reported words by children in the sample. The three most common words in each semantic category were removed to avoid recall confounds associated with item familiarity (Miller et al. 2003). The context of a shopping list was selected for its consistent familiarity with children across a wide range of cultural and demographic variables and mapped closely with the CVLT with regard to presentation, timing, and scoring.

Psychometric Data

The normative sample for the CVLT-C consists of stratified data taken from the 1988 US census findings and is comprised of 920 children in 12 age ranges from 5 years to 16 years 11 months. Standardized scores were derived from accumulative raw score performance per age group, distribution normalization, and elimination of outliers and skewing effects. The remaining learning score components of the CVLT-C were developed via regression analyses (Delis et al. 1987).

Investigations of test-retest reliability among 106 school-age children ranged from .17 (cued-recall intrusions, for 12-year-olds) to .90 (perseverations, for 8-year-olds) (Delis et al. 1987, 1994; Spreen and Strauss 1998). Alternate forms reliability was reported at .84 (Delis et al. 1987, 1994; Spreen and Strauss 1998), indicating appropriate reliability for multiple administrations with children and tracking results and learning characteristics over time.

Gender effects were reported by the authors to be minimal in the initial standardization sample, and significant differences were not found for gender in the 4-year-old sample norms provided by Goodman, Delis, and Mattson (Goodman et al. 1999) for normative populations. However, differences in gender have been reported in follow-up examinations of the standardization sample (Kramer et al. 1997) and have been evidenced in clinical populations of children with ADHD (Cutting et al. 2003) and significant head injury (Warschausky et al. 2005). Gender effects were also evident in examinations of adolescent populations, with girls outperforming boys

(Beebe et al. 2000). Low correlations with measures of executive functioning and moderate associations with intelligence measures such as the Wechsler block design and vocabulary subtests have been reported (Beebe et al. 2000). Donders (1999) also identified a significant link between parental education levels and test performance in the standardized sample, with children of parents with higher education consisting of 22% of the highest performing children and children of parents with lower rates of education accounting for 30% of the children in the below-average range of performance.

Predictably, age effects were observed among the standardization sample, with steeper learning slopes being present in children as age increased and development progressed. Consistency of recalled items and immediate recall scores were also observed to have developmental trends across the sample. The use of semantic clustering strategy as a learning strategy was first emergent among 9–12-year-old participants. Adolescents in the sample exhibited higher degrees of serial clustering strategy use compared to other age groups (Delis et al. 1994). Investigations of executive functioning and CVLT-C process scores further indicate that perseverative errors evidence strong consistency throughout development with minimal improvement, while rates of intrusions and false-positives exhibit considerable improvement as development progresses into adolescence (Beebe et al. 2002; Delis et al. 1994).

Donders (1999) provided maximum likelihood confirmatory factor analysis on 13 qualitative and quantitative variables from the original standardized sample to identify the most salient factors of learning and memory tapped by the CVLT-C. A five-factor model consisting of attention span, learning efficiency, free delayed recall, cued delayed recall, and inaccurate recall showed the greatest fit and was proposed to be a valid and clinically useful predictor of performance on the measure.

The CVLT-C has been co-normed with the children's category test (CCT; Boll 1993), allowing examiners to compare a child's memory and learning performance with other forms of higher order cognitive functioning. Combining the

results of both tasks to generate the learning profile of a child can be clinically valuable as the CCT provides explicit feedback on a nonverbal task, while the CVLT-C provides non-explicit feedback on a verbal task through repetition. By taking advantage of the co-normed scores, clinicians are able to tap a wider range of learning areas and skills for characterizing the cognitive capabilities of the child. Donders (1999) examined the psychometric comparisons of the two measures including the magnitude of difference necessary for statistical significance in scores. Standardized sample data from both measures were used to evaluate covariances and statistically significant discrepancies between the T scores of those instruments as well as the base rate of specific discrepancies among 920 children ranging in age from 5 to 16 years. Results suggested that the CCT and CVLT-C share a small degree of common variance. Statistically significant score discrepancies between the two measures (T-score difference greater than 18 among 5–8-year-olds and greater than 16 among 9–16-year-olds) were common, indicating that evaluation of the potential clinical significance of a discrepancy between the obtained results should also include consideration of base rate statistics when evaluating individual children.

While the standardization sample focused on children ages 5 years through 16 years 11 months, Goodman et al. (1999) provided normative data for 4-year-old participants on the CVLT-C for potential administration with younger populations to aid in early identification and intervention. Each month of the 4-year-old range was represented among the stratified sample of 80 (40 males and 40 females). Performance characteristics of the younger population were considerably similar to that of the normative sample data, apart from a few learning characteristics. The 4-year-old participants had a tendency for higher extra-list intrusions relative to their correct responses on cued recall that were not present on free recall as well as a higher endorsement of distracter items during the recognition trial. Semantic and serial clustering characteristics were also consistent with developmental trends, providing evidence for utility in identifying early

memory and learning characteristics with the younger population.

Clinical Uses

The CVLT-C has been used to assess memory and learning in a wide variety of clinical childhood populations and has been used to examine verbal learning in children with ASD. Early studies of memory and list learning among children with ASD highlighted specific deficits in recall compared to control groups. Boucher and Warrington (1976) used memory tests that employed pictures, lists, and spoken words with 29 children with ASD and compared recall scores against age-matched controls. During trials of forced-choice recall, children with autism showed significantly lower rates of recall than controls but demonstrated considerable improvement when provided with semantic descriptive cues of list items and pictures.

Initial investigations of verbal recall among children with autism spectrum disorder (ASD) utilizing the CVLT also suggested distinct differences in learning and memory profiles when compared to typically developing peers. Minshew and Goldstein (1993) compared the performance of high-functioning children and adults with ASD ranging in age from 12 to 40 years old to age-matched normal controls using the CVLT. The comparison group significantly outperformed the ASD group. Specific scores indicated that while individuals with ASD showed comparable recall and recognition scores when presented with list A of CVLT, they showed significantly more intrusion errors on both list A and list B items and considerably lower recall scores on list B. The authors concluded that the overall characteristics of the ASD scores were indicative of a “subtle inefficiency of verbal memory” that was more suggestive of deficits in mechanisms for effectively organizing information than a reflection of comprehensive memory impairment.

More recent investigations into learning strategies and encoding profiles of children with ASD lend support for this theory and suggest that the CVLT-C may be effective in highlighting specific

characteristics of verbal learning in children with ASD that differ from those of typical developing peers. Phelan, Filliter, and Johnson (Phelan et al. 2010) compared performance and verbal learning characteristics on the CVLT-C between 15 high-functioning children with ASD and typical developing controls. Although the learning profiles and performance characteristics of both groups were comparable, children with ASD demonstrated considerable improvement in their cued-recall scores compared to their free-recall scores, suggesting the need for external supports and cueing opportunities to facilitate verbal memory performance among ASD youth.

Key clinical strengths of the CVLT-C include its relative ease of use and excellent internal consistency. Considerable research and psychometric data have been gathered with CVLT-C, and it has proven useful in predicting a variety of difficulties and deficits that can inform decision making concerning placements in groups such as head trauma patients and other neurodevelopmental disorders (Nagel et al. 2006; Nichols et al. 2004). As previously noted, the test provides a considerable amount of information about the verbal learning process and learning strategies across a relatively short period of time in such a way that recall and cueing effects can be examined efficiently and reliably. Scores on the CVLT-C have been shown to account for a considerable amount of the variance in the prediction of special education services and long-term educational outcome among children with severe head injury that could translate to other clinical populations (Miller and Donders 2003). The CVLT-C's implementation across a wide range of childhood populations illustrates its breadth in utility and efficiency across several domains of care. The provision of normative data for 4-year-olds additionally provides valuable opportunities for early screening, intervention, and tracking among children early in development. While the internal consistency of the test has been thoroughly investigated and validated, stability coefficients of many of the variables examined in the CVLT-C fall below acceptable standards, cautioning against the use of single variables as valid examination of cognitive factors (Spreen and

Strauss 1998). Overall, the test has shown to be an efficient and informative instrument of memory and verbal learning among children that serves as a valuable asset to clinicians involved in diagnostic assessment, treatment planning, service enrollment, and needs assessment.

References and Reading

- Beebe, D. W., Ris, M. D., & Dietrich, K. N. (2000). The relationship between CVLT-C process scores and measures of executive functioning: Lack of support among community-dwelling adolescents. *Journal of Clinical and Experimental Neuropsychology*, 22(6), 779–792.
- Boll, T. (1993). *Children's category test*. San Antonio: The Psychological Corporation.
- Boucher, J., & Warrington, E. K. (1976). Memory deficits in early infantile autism: Some similarities to the amnesic syndrome. *British Journal of Psychology*, 67(1), 73–87.
- Cutting, L. E., Koth, C. W., Mahone, E. M., & Denckla, M. B. (2003). Evidence for unexpected weaknesses in learning in children with attention-deficit/hyperactivity disorder without reading disabilities. *Journal of Learning Disabilities*, 36(3), 257–267.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (1987). *California verbal learning test manual (CVLT)*. San Antonio: The Psychological Corporation.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (1994). *California verbal learning test-children's version (CVLT-C)*. San Antonio: The Psychological Corporation.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000). *The California verbal learning test manual* (2nd ed.). San Antonio: The Psychological Corporation.
- Donders, J. (1999). Structural equation analysis of the California Verbal Learning Test-Children's Version in the standardization sample. *Developmental Neuropsychology*, 15(3), 395–406.
- Goodman, A. M., Delis, D. C., & Mattson, S. N. (1999). Normative data for 4-year-old children on the California Verbal Learning Test-Children's Version. *The Clinical Neuropsychologist*, 13(3), 274–282.
- Kramer, J. H., Delis, D. C., Kaplan, E., O'Donnell, L., & Prifitera, A. (1997). Developmental sex differences in verbal learning. *Neuropsychology*, 11(4), 577–584.
- Miller, J. J., & Donders, J. (2003). Prediction of educational outcome after pediatric traumatic brain injury. *Rehabilitation Psychology*, 48, 237–241.
- Miller, M. J., Bigler, E. D., & Adams, W. V. (2003). Comprehensive assessment of child & adolescent memory: The wide range assessment of memory and learning, the test of memory and learning, and the California verbal learning test-children's version. In C. R. Reynolds & R. W. Kamphaus (Eds.), *Handbook of psychological assessment of children: Intelligence, aptitude, and achievement* (pp. 275–304). New York: Guilford Press.

- Minshew, N. J., & Goldstein, G. (1993). Is autism an amnesic disorder? Evidence from the California verbal learning test. *Neuropsychology*, 7(2), 209–216.
- Nagel, B. J., Delis, D. C., Palmer, S. L., Reeves, C., Gajjar, A., & Mulhern, R. K. (2006). Early patterns of verbal memory impairment in children treated for medulloblastoma. *Neuropsychology*, 20(1), 105–112.
- Nichols, S., Jones, W., Roman, M. J., Wulfeck, B., Delis, D. C., Reilly, J., et al. (2004). Mechanisms of verbal memory impairment in four neurodevelopmental disorders. *Brain and Language*, 88(2), 180–189.
- Phelan, H. L., Filliter, J. H., & Johnson, S. A. (2010). Brief report: Memory performance on the California verbal learning test – children’s version in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41(4), 518–523.
- Spreen, O., & Strauss, E. (1998). *A compendium of neuropsychological tests: Administration, norms, & commentary* (2nd ed.). New York: Oxford University Press.
- Warschawsky, S., Kay, J. B., Chi, P., & Donders, J. (2005). Hierarchical linear modeling of California verbal learning test–children’s version learning curve characteristics following childhood traumatic head injury. *Neuropsychology*, 19(2), 193–198.

Callosotomy (Surgical Severing)

► Agenesis of Corpus Callosum

Cambridge Neuropsychological Test Automated Battery

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Synonyms

CANTAB

Description

The CANTAB[®] tests are simple, computerised, non-linguistic, and culturally blind. They can be

administered by a trained assistant. Importantly, interpretation of a patient’s condition can be easily understood by a clinician. Below is a complete list of all tests, correct at time of publication. The tests are categorised as assessing the following cognitive domains:

1. Induction
2. Visual Memory
3. Executive function
4. Attention
5. Verbal/Semantic Memory
6. Decision Making and Response Control
7. Social Cognition
8. Other tests

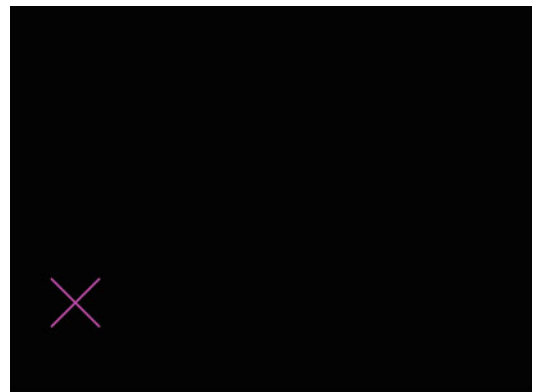
CANTAB – Induction

These very short tests can be used to familiarize participants with the general idea of responding in a task by touching the screen. They can also be regarded as warm-up tasks, getting the participant used to the general testing situation.

They consist of: Motor Screening Task and Big/Little Circle.

Motor Screening (MOT)

See Fig. 1.



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Cambridge Neuropsychological Test Automated Battery, Fig. 1 Motor screening task

Overview

The Motor Screening test is typically administered at the beginning of a test battery, and serves as a simple introduction to the touch screen for the participant. If a participant is unable to comply with the simple requirements of this test, it is unlikely that they will be able to complete other tests successfully. This test therefore screens for visual, movement, and comprehension difficulties.

Administration Time

Around 2 min

Task

Participants must touch the flashing cross which is shown in different locations on the screen.

Outcome Measures

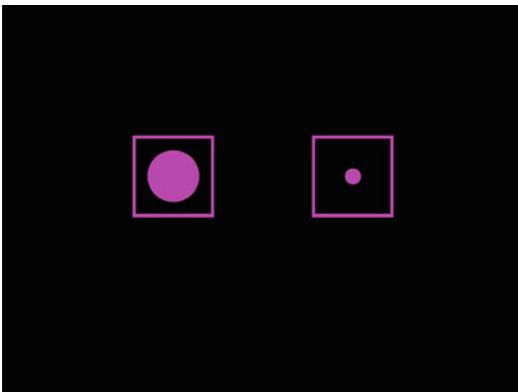
This test has two outcome measures which measure the participant's speed of response and the accuracy of the participant's pointing.

Test Modes

Two modes are available – clinical and high visibility. In high visibility the crosses are drawn using thicker lines and are easier to see.

Big/Little Circle (BLC)

See Fig. 2.



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Cambridge Neuropsychological Test Automated Battery, Fig. 2 Big/Little circle (BLC)

Overview

The Big/Little Circle test assesses comprehension, learning, and reversal. It is also intended to train participants in the general idea of following and reversing a rule, before proceeding to the *Intra-Extra dimensional Shift test (IED)*, so it should ideally precede the IED task in a battery.

Administration Time

Around 2 min.

Task

Participants must first touch the smaller of the two circles displayed, then, after 20 trials, touch the larger circle for 20 further trials.

Outcome Measures

This test has five outcome measures, covering latency (speed of response) and the participant's ability to touch the correct circle.

Test Modes

One mode – clinical

Visual Memory

These tests allow investigation of visual and spatial aspects of memory and consist of: Delayed Matching to Sample, Paired Associates Learning, Pattern Recognition Memory and Spatial Recognition Memory.

Delayed Matching to Sample

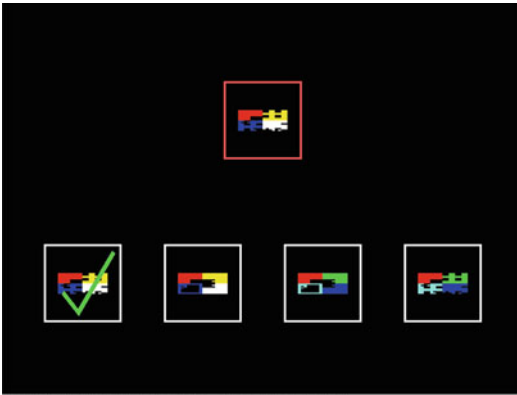
See Fig. 3.

Overview

Delayed Matching to Sample (DMS) assesses forced choice recognition memory for novel non-verbalisable patterns, and tests both simultaneous and short-term visual memory. This test is primarily sensitive to damage in the medial temporal lobe area, with some input from the frontal lobes.

Administration Time

Around 10 min



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Cambridge Neuropsychological Test Automated Battery, Fig. 3 Delayed matching to sample

Task

The participant is shown a complex visual pattern (the sample) and then, after a brief delay, four similar patterns. The participant must touch the pattern which exactly matches the sample.

Outcome Measures

This test has 19 outcome measures, assessing latency (the participant's speed of response), the number of correct patterns selected, and statistical analysis measuring the probability of an error after a correct or incorrect response.

Test Modes

Clinical mode (for testing once); five parallel modes (for repeated testing), and child mode (a simplified version for testing children)

Paired Associates Learning (PAL)

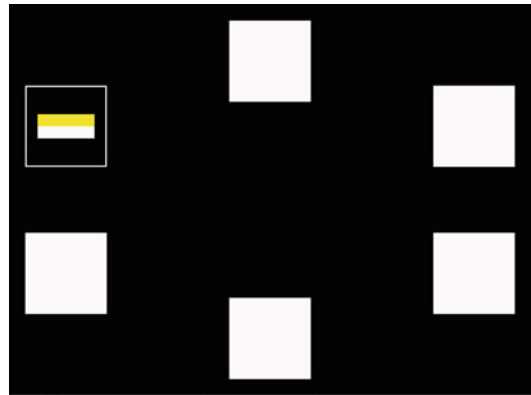
See Fig. 4.

Overview

This challenging test assesses visual memory and new learning, and is a useful tool for assessing patients with questionable dementia, Mild Cognitive Impairment, Alzheimer's disease, and age-related memory loss.

Administration Time

Around 10 min, depending on level of impairment



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Cambridge Neuropsychological Test Automated Battery, Fig. 4 Paired associates learning (PAL)

Task

Boxes are displayed on the screen and are opened in a randomized order. One or more of them will contain a pattern. The patterns are then displayed in the middle of the screen, one at a time, and the participant must touch the box where the pattern was originally located. If the participant makes an error, the patterns are re-presented to remind the participant of their locations. The difficulty level increases through the test. In the clinical mode, the number of patterns increases from one to eight, which challenges even very able participants.

Outcome Measures

This test has 21 outcome measures, covering the errors made by the participant, the number of trials required to locate the pattern(s) correctly, memory scores, and stages completed.

Test Modes

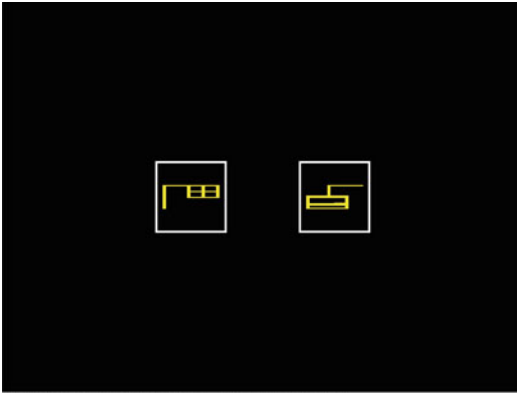
Clinical mode (for testing once); five parallel modes (for repeated testing)

Pattern Recognition Memory (PRM)

See Fig. 5.

Overview

This is a test of visual pattern recognition memory in a two-choice forced discrimination paradigm. This test is often used, in conjunction with *Spatial Recognition Memory (SRM)*, before the *Paired*



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Cambridge Neuropsychological Test Automated Battery, Fig. 5 Pattern recognition memory

Associates Learning (PAL) test, as both these tests help to train the participant for PAL.

PRM and SRM contain different elements of PAL and the results considered together help to decide on the exact nature of the cognitive deficit being considered.

Administration Time

Around 5 min, depending on level of impairment

Task

The participant is presented with a series of 12 visual patterns, 1 at a time, in the center of the screen. These patterns are designed so that they cannot easily be given verbal labels. In the recognition phase, the participant is required to choose between a pattern they have already seen and a novel pattern. In this phase, the test patterns are presented in the reverse order to the original order of presentation.

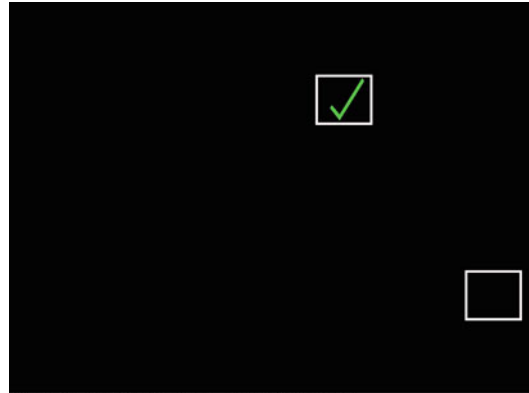
This is then repeated, with 12 new patterns. The second recognition phase can be given either immediately or after a 20 min delay.

Outcome Measures

This test has three outcome measures, including the number and percentage of correct trials and latency (speed of participant's response).

Test Modes

Clinical mode (for testing once); four parallel modes (for repeated testing). Each of these



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Cambridge Neuropsychological Test Automated Battery, Fig. 6 Spatial recognition memory

modes also has separate immediate and delayed versions available.

Spatial Recognition Memory (SRM)

See Fig. 6.

Overview

This is a test of visual spatial recognition memory in a two-choice forced discrimination paradigm. This test is often used, in conjunction with *Pattern Recognition Memory (PRM)*, before the *Paired Associates Learning (PAL)* test, as both these tests help to train the participant for PAL.

PRM and SRM contain different elements of PAL and the results considered together help to decide on the exact nature of the cognitive deficit being considered.

Administration Time

Around 5 min, depending on level of impairment

Task

The participant is presented with a white square, which appears in sequence at five different locations on the screen. In the recognition phase, the participant sees a series of five pairs of squares, one of which is in a place previously seen in the presentation phase. The other square is in a location not seen in the presentation phase. As with the PRM test, locations are tested in the reverse of the presentation order. This subtest is repeated three more times, each time with five new locations.

Outcome Measures

This test has three outcome measures, including the number and percentage of correct trials and latency (speed of subject's response).

Test Modes

Clinical mode (for testing once); four parallel modes (for repeated testing)

CANTAB – Executive Function, Working Memory, and Planning Tests

These tests address executive function, working memory, and planning; all are associated with the frontal area of the brain.

Attention Switching Task (AST)

Overview

AST is a test of the participant's ability to switch attention between the direction or location of an arrow on screen. This test is a sensitive measure of frontal lobe and 'executive' dysfunction.

Administration Time

Around 8 min, depending on level of impairment

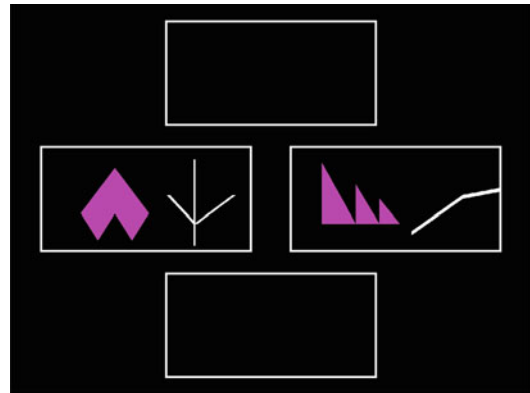
Task

The test begins with an arrow in the centre of the screen which points either to the left or to the right. The participant is introduced to two buttons, one on the left and one on the right, and is asked to press a button corresponding to the direction in which the arrow is pointing.

After this initial training, the participant is then told that the arrow might appear on the left or the right side of the screen, and depending on the cue given at the top of the screen, the participant must either press the left or right button to indicate on which side of the screen the arrow is displayed, or else press the left or right button to correspond with the direction in which the arrow is pointing.

Outcome Measures

AST has 7 outcome measures, each of which can have various options applied to it. The AST measures cover latency, correct and incorrect



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Cambridge Neuropsychological Test Automated Battery, Fig. 7 Intra-extra dimensional set shift

responses, commission errors, omission errors, switch cost and congruency cost.

Test Modes

AST has one mode: Clinical

Intra-Extra Dimensional Set Shift (IED)

See Fig. 7.

Overview

Intra-Extra Dimensional Set Shift is a test of rule acquisition and reversal. It features:

- Visual discrimination and attentional set formation
- Maintenance, shifting, and flexibility of attention

This test is primarily sensitive to changes to the fronto-striatal areas of the brain.

This test is a computerized analogue of the Wisconsin Card Sorting test, and is sensitive to cognitive changes associated with schizophrenia, Parkinson's Disease, and dopaminergic-dependent processes.

Administration Time

Around 7 min, depending on level of impairment

Task

Two artificial dimensions are used in the test:

- Color-filled shapes
- White lines

Simple stimuli are made up of just one of these dimensions, whereas compound stimuli are made up of both, namely white lines overlying color-filled shapes. The participant starts by seeing two simple color-filled shapes, and must learn which one is correct by touching it.

Feedback teaches the participant which stimulus is correct, and after six correct responses, the stimuli and/or rules are changed. These shifts are initially intra dimensional (e.g., color-filled shapes remain the only relevant dimension), then later extra dimensional (white lines become the only relevant dimension).

Participants progress through the test by satisfying a set criterion of learning at each stage (six consecutive correct responses). If at any stage, the participant fails to reach this criterion after 50 trials, the test terminates.

Outcome Measures

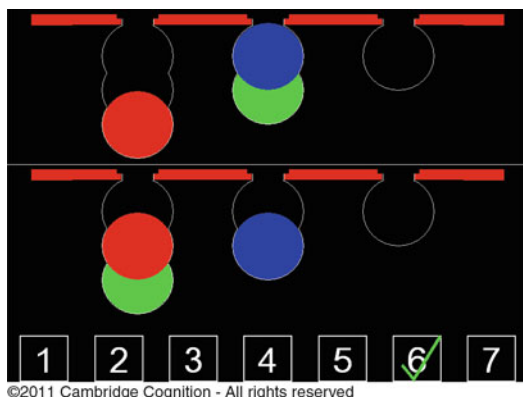
This test has 18 outcome measures, assessing errors, and number of trials and stages completed.

Test Modes

Clinical mode (for testing once); seven parallel modes (for repeated testing)

One Touch Stockings of Cambridge (OTS)

See Fig. 8.



Cambridge Neuropsychological Test Automated Battery, Fig. 8 One touch stockings of cambridge

Overview

One Touch Stockings of Cambridge is a spatial planning task which gives a measure of frontal lobe function. OTS is a variant of the *Stockings of Cambridge* test (see below) and places greater demands on working memory as the participant has to visualize the solution.

Administration Time

Around 10 min, depending on level of impairment

Task

As for SOC (Stockings of Cambridge), the subject is shown two displays containing three colored balls. The displays are presented in such a way that they can easily be perceived as stacks of colored balls held in stockings or socks suspended from a beam. This arrangement makes the 3-D concepts involved apparent to the participant, and fits with the verbal instructions.

There is a row of numbered boxes along the bottom of the screen. The test administrator first demonstrates to the participant how to use the balls in the lower display to copy the pattern in the upper display, and completes one demonstration problem, where the solution requires one move. The participant must then complete three further problems, one each of two moves, three moves, and four moves.

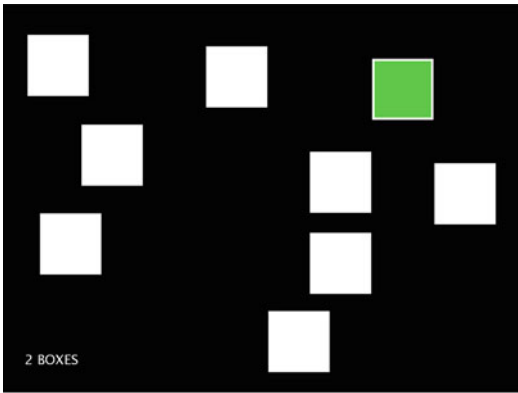
Next the participant is shown further problems, and must work out in their head how many moves the solutions to these problems require, then touch the appropriate box at the bottom of the screen to indicate their response.

Outcome Measures

OTS has four outcome measures – problems solved on first choice, mean choices to correct, mean latency to first choice, and mean latency to correct. Each of these measures may be calculated for all problems, or for problems with a specified number of moves (one move to five or six moves).

Test Modes

OTS has four modes, with varying numbers of problems and boxes.



Cambridge Neuropsychological Test Automated Battery, Fig. 9 Spatial span

Spatial Span (SSP)

See Fig. 9.

Overview

Spatial Span assesses working memory capacity, and is a visuospatial analogue of the Digit Span test.

Administration Time

Around 5 min, depending on level of impairment

Task

White squares are shown, some of which briefly change color in a variable sequence. The participant must then touch the boxes which changed color in the same order that they were displayed by the computer (for clinical mode) or in the reverse order (for reverse mode). The number of boxes increases from two at the start of the test to nine at the end, and the sequence and color are varied through the test.

Outcome Measures

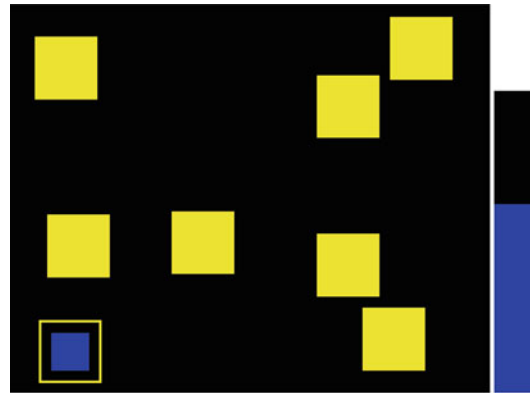
This test has six outcome measures, covering span length (the longest sequence successfully recalled), errors, number of attempts, and latency.

Test Modes

Two modes: clinical mode and reverse mode.

Spatial Working Memory (SWM)

See Fig. 10.



Cambridge Neuropsychological Test Automated Battery, Fig. 10 Spatial working memory

Overview

SWM is a test of the participant's ability to retain spatial information and to manipulate remembered items in working memory. It is a self-ordered task, which also assesses heuristic strategy. This test is a sensitive measure of frontal lobe and "executive" dysfunction. It has been shown in recent studies that impaired performance on SWM emerges as a common factor in prepsychosis.

Administration Time

Around 8 min, depending on level of impairment

Task

The test begins with a number of colored squares (boxes) being shown on the screen. The aim of this test is that, by touching the boxes and using a process of elimination, the participant should find one blue "token" in each of a number of boxes and use them to fill up an empty column on the right hand side of the screen. The number of boxes is gradually increased, until it is necessary to search a total of eight boxes. The color and position of the boxes used are changed from trial to trial to discourage the use of stereotyped search strategies.

Outcome Measures

The 24 outcome measures for SWM include errors (touching boxes that have been found to be empty, and revisiting boxes which have already

been found to contain a token), a measure of strategy, and latency measures.

Test Modes

Clinical mode

Stockings of Cambridge (SOC)

See Fig. 11.

Overview

SOC is a spatial planning test which gives a measure of frontal lobe function

Administration Time

Around 10 min, depending on level of impairment.

Task

The participant is shown two displays containing three coloured balls. The displays are presented in such a way that they can easily be perceived as stacks of coloured balls held in stockings or socks suspended from a beam. This arrangement makes the 3-D concepts involved apparent to the participant, and fits with the verbal instructions.

The participant must use the balls in the lower display to copy the pattern shown in the upper display. The balls may be moved one at a time by touching the required ball, then touching the position to which it should be moved. The time taken to complete the pattern and the number of moves

required are taken as measures of the participant's planning ability.

Outcome Measures

This test has three outcome measures, including the number and percentage of correct trials and latency (speed of participant's response).

Test Modes

Clinical mode

CANTAB Attention Tests

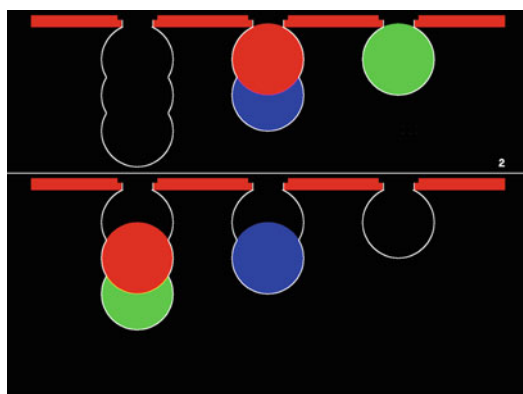
These tests measure different aspects of attention and reaction time. *Choice Reaction Time (CRT)*, *Rapid Visual Information Processing (RVP)*, and *Simple Reaction Time (SRT)* use the press pad exclusively as an input device; *Match to Sample Visual Search (MTS)* and *Reaction Time (RTI)* use both the press pad and the touch screen.

Choice Reaction Time

See Fig. 12.

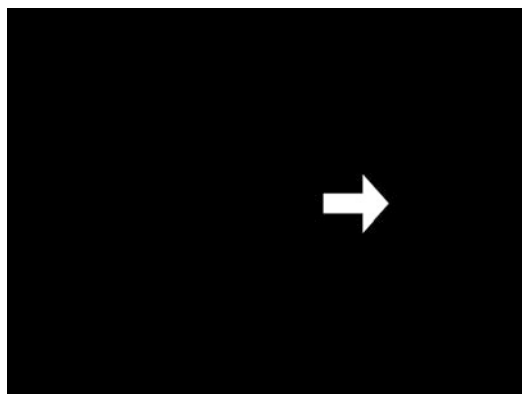
Overview

Choice Reaction Time (CRT) is a two-choice Reaction Time test which is similar to the *Simple Reaction Time (SRT)* test, except that stimulus and response uncertainty are introduced by having two possible stimuli and two possible responses.



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Cambridge Neuropsychological Test Automated Battery, Fig. 11 Stockings of Cambridge



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Cambridge Neuropsychological Test Automated Battery, Fig. 12 Choice reaction time

It is useful for testing general alertness and motor speed.

Administration Time

Around 7 min, depending on level of impairment

Task

An arrow-shaped stimulus is displayed on either the left or the right side of the screen.

The participant must press the left hand button on the press pad if the stimulus is displayed on the left hand side of the screen, and the right hand button on the press pad if the stimulus is displayed on the right hand side of the screen.

Outcome Measures

This test has 13 outcome measures, assessing correct and incorrect responses, errors of commission and omission (late and early responses), and latency (response speed).

Test Modes

Clinical mode

Match to Sample Visual Search (MTS)

See Fig. 13.

Overview

Match to Sample Visual Search (MTS) is a matching test, with a speed/accuracy trade-off. It is a simultaneous visual search task with response

latency dissociated from movement time. Efficient performance on this task requires the ability to search among the targets and ignore the distractor patterns which have elements in common with the target. This test can help to differentiate between Parkinson's disease and Alzheimer's disease, and also between Lewy Body dementia and Alzheimer's disease.

Administration Time

Around 9 min, depending on level of impairment

Task

The participant is shown a complex visual pattern (the sample) in the middle of the screen, and then, after a brief delay, a varying number of similar patterns are shown in a circle of boxes around the edge of the screen. Only one of these boxes matches the pattern in the center of the screen, and the participant must indicate which it is by touching it. Reaction time is measured on the basis of the release of the press pad, which allows for its more accurate measurement.

Outcome Measures

The 12 outcome measures for SOC cover the number of problems solved with minimum moves, the mean number of moves for n-move problems, mean initial thinking time for n-move problems, and mean subsequent thinking time for n-move problems.

Test Modes

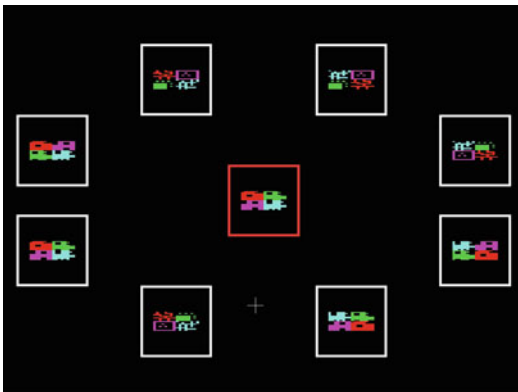
Clinical mode

Rapid Visual Information Processing (RVP)

See Fig. 14.

Overview

Rapid Visual Information Processing (RVP) is a test of sustained attention (similar to the Continuous Performance Task) and has proved useful in many studies in which drugs are used to help develop a disease model. It is sensitive to dysfunction in the parietal and frontal lobe areas of the brain and is also a sensitive measure of general performance.



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Cambridge Neuropsychological Test Automated Battery, Fig. 13 Match to sample visual search



Cambridge Neuropsychological Test Automated Battery, Fig. 14 Rapid visual information processing

Administration Time
Around 7 min

Task

A white box appears in the center of the computer screen, inside which digits, from 2 to 9, appear in a pseudo-random order, at the rate of 100 digits per minute. Participants are requested to detect target sequences of digits (e.g., 2–4–6, 3–5–7, 4–6–8) and to register responses using the press pad.

Outcome Measures

The nine RVP outcome measures cover latency, probabilities, and sensitivity (calculated using Signal Detection Theory), and hits, misses, false alarms, and rejections.

Test Modes

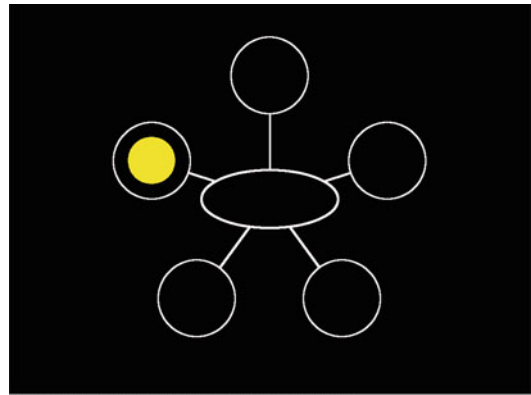
Clinical mode, plus 123 mode (for children aged 4–8) and 357 mode (for children aged 7–14)

Reaction Time (RTI)

See Fig. 15.

Overview

Reaction Time (RTI) is a latency task with a comparative history (the five choice task) and uses a procedure to separate response latency from movement time. It is more useful than CRT or SRT where it is necessary to control for tremor.



Cambridge Neuropsychological Test Automated Battery, Fig. 15 Reaction time

Administration Time

Around 5 min, depending on level of impairment

Task

The task is divided into five stages, which require increasingly complex chains of responses. In each case, the subject must react as soon as a yellow dot appears. In some stages, the dot may appear in one of five locations, and the subject must sometimes respond by using the press pad, sometimes by touching the screen, and sometimes both.

Outcome Measures

The four outcome measures in RTI are divided into Reaction Time (simple and five-choice) and movement time (simple and five-choice)

Test Modes

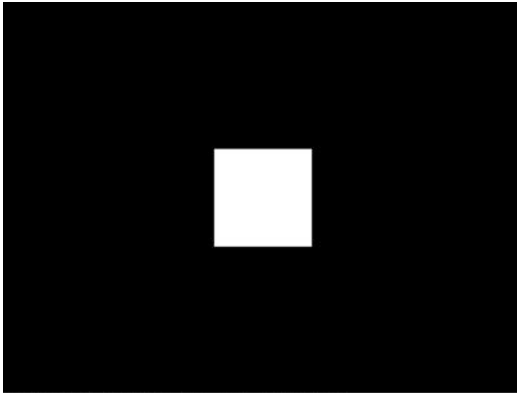
Clinical mode, parallel mode, and child mode

Simple Reaction Time (SRT)

See Fig. 16.

Overview

Simple Reaction Time (SRT) is a test which measures simple Reaction Time through delivery of a known stimulus to a known location to elicit a known response. The only uncertainty is with regard to when the stimulus will occur, by having a variable interval between the trial response and the onset of the stimulus for the next trial. Like



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Cambridge Neuropsychological Test Automated Battery, Fig. 16 Simple reaction time

Choice Reaction Time (CRT), it is useful for testing general alertness and motor speed, and is often sensitive to medication effects.

Administration Time

Around 6 min, depending on level of impairment

Task

As soon as the participant sees the square on the screen, they must press the button on the press pad.

Outcome Measures

The 11 outcome measures for SRT cover latency (response speed), correct responses, and errors of commission and omission.

Test Modes

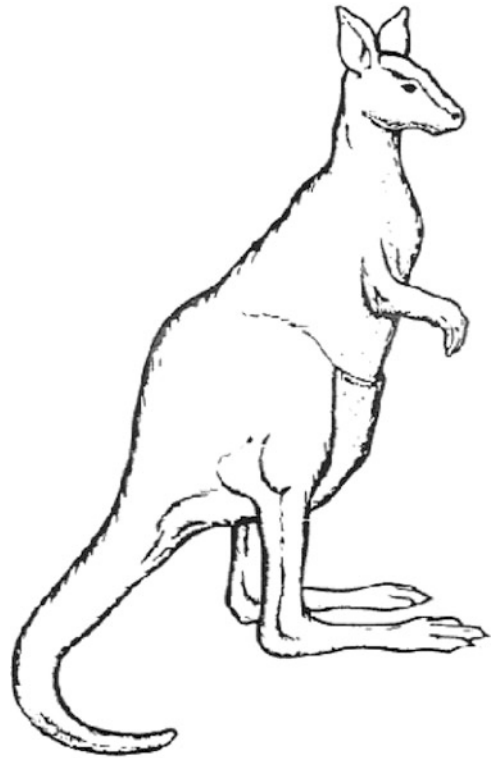
Clinical mode

CANTAB – Semantic/Verbal Memory Tests

These tests, which address semantic and/or verbal memory, are relatively new additions to the CANTAB battery consisting of: Graded Naming Test (GNT) and Verbal Recognition Memory (VRM).

Graded Naming Test (GNT)

See Fig. 17.



Cambridge Neuropsychological Test Automated Battery, Fig. 17 Graded naming test

Overview

The Graded Naming Test has been used extensively in cognitive neuropsychology. The Graded Naming Test (GNT) avoids the problem of ceiling effects in previous naming tests by having participants name drawings of objects in ascending difficulty. Reduced efficiency in retrieving the name of an object can be the first and only indication of impaired language functioning. This test assesses object-naming ability, but is in addition graded in difficulty to allow for individual differences. This means that it may be able to detect any word-finding difficulty even in those with an extensive naming vocabulary.

Administration Time

Around 10 min, depending on level of impairment

Task

Thirty different line drawings are displayed on the screen, 1 at a time. The participant must identify the object depicted in each drawing.

Outcome Measures

This test has six outcome measures, which include total correct, total errors, and normative z-score and percentile.

Notes

Currently available in UK English only (this test is culturally biased and there are no alternative versions at present). A pencil and paper version of this test is also available.

Test Modes

Clinical mode

Verbal Recognition Memory (VRM)

See Fig. 18.

Overview

Despite the general desirability of nonverbal tests because of their culture free applicability, researchers and clinical studies sometimes require verbal tests, perhaps because of need to explore questions relating to language or left hemisphere function. Other verbal tests have a long history of use in psychiatric assessment and clinical studies. The Verbal Recognition Memory test, which assesses immediate and delayed memory of verbal information under free recall and forced choice recognition conditions, should provide comparable results.



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Cambridge Neuropsychological Test Automated Battery, Fig. 18 Verbal recognition memory

Task

In the VRM test, the participant is shown a list of 12 words, 1 at a time, and then asked to:

- Produce as many of the words as possible immediately following the presentation
- Recognize the words they have seen before from a list of 24 words containing the original 12 words and 12 distractors
- Following a delay of 20 min, recognize the words they have seen before from another list of 24 words containing the original list and 12 new distractors

Outcome Measures

The five outcome measures for VRM cover correct and incorrect responses for the recognition and free recall parts of the test.

Notes

Currently available in UK English only

Test Modes

Clinical mode and four parallel modes for repeated testing. Each mode has immediate and delayed parts.

CANTAB – Decision Making and Response Control Tests

These tests add another dimension to cognitive profiling and investigation of frontal lobe function. Most decisions in life have an emotional or risk-related component, and many clinical conditions are associated with inappropriate risk models/strategies.

They consist of Affective Go/No-go (AGN), Information Sampling Task (IST), Cambridge Gambling Task (CGT) and Stop Signal Task (SST).

Affective Go/No-go (AGN)

See Fig. 19.

Overview

This test assesses information processing biases for positive and negative stimuli.



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Cambridge Neuropsychological Test Automated Battery, Fig. 19 Affective Go/No-Go

Affective cognitive functions are thought to be related to the ventral and medial-prefrontal cortex areas of the brain because of the limbic connections with this region. As such, the Affective Go/No-go test represents a powerful research assessment tool for current studies on the neural substrates of depression, bipolar disorder, Post-Traumatic Stress Disorder (PTSD), and many other affective conditions.

Administration Time

Around 10 min, depending on level of impairment

Task

The test consists of several blocks, each of which presents a series of words from two of three different affective categories: Positive (e.g., joyful), Negative (e.g., hopeless), and Neutral (e.g., element). The participant is given a target category, and is asked to press the press pad when they see a word matching this category.

Outcome Measures

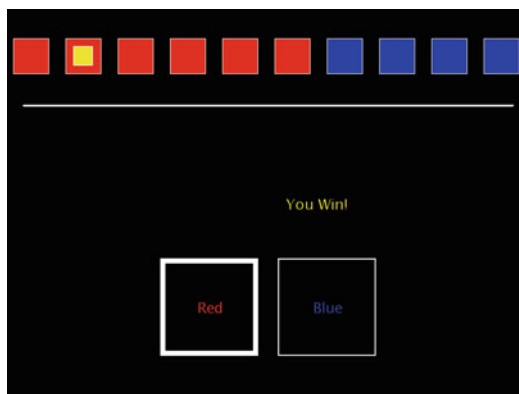
Twelve outcome measures covering latency and errors of commission and omission

Note

Currently available in English only.

Test Modes

Six modes, four using positive and negative stimuli only, and two using positive, negative, and neutral stimuli



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Cambridge Neuropsychological Test Automated Battery, Fig. 20 Cambridge gambling task

Cambridge Gambling Task (CGT)

See Fig. 20.

Overview

The Cambridge Gambling Task was developed to assess decision making and risk-taking behavior outside a learning context. Relevant information is presented to the participants “up-front” and there is no need to learn or retrieve information over consecutive trials.

Unlike other “Gambling” tasks, CGT dissociates risk taking from impulsivity, because in the ascending bet condition, the participant who wants to make a risky bet has to wait patiently for it to appear. The likely neural substrate for this task is the orbitofrontal prefrontal cortex. Traumatic Brain Injury, Alcoholism, and Drug abuse are all conditions sensitive to this test.

Administration Time

Up to 30 min

Task

On each trial, the participant is presented with a row of ten boxes across the top of the screen, some of which are red and some of which are blue. At the bottom of the screen are rectangles containing the words “Red” and “Blue.” The participant must guess whether a yellow token is hidden in a red box or a blue box.

In the gambling stages, participants start with a number of points, displayed on the screen, and can select a proportion of these points, displayed in

either rising or falling order, in a second box on the screen, to gamble on their confidence in this judgment. A stake box on the screen displays the current amount of the bet. The participant must try to accumulate as many points as possible.

Outcome Measures

The six CGT outcome measures cover risk taking, quality of decision making, deliberation time, risk adjustment, delay aversion, and overall proportion bet.

Test Modes

Ascending first (where stakes are displayed in ascending order for two stages, then in descending order for two stages) and Descending first (where stakes are displayed in descending order for two stages, then in ascending order for two stages).

Information Sampling Task (IST)

See Fig. 21.

Overview

The Information Sampling Task (IST) tests impulsivity and decision making.

Administration Time

Up to 15 min

Task

The subject is presented with a 5×5 array of gray boxes on the screen, and two larger colored panels

below these boxes. The subject is instructed that they are playing a game for points, which they can win by making a correct decision about which color is in the majority under the gray boxes. They must touch the gray boxes one at a time, which open up to reveal one of the two colors shown at the bottom of the screen. Once a box has been touched, it remains open. When the subject has made their decision about which color is in the majority, they must touch the panel of that color at the bottom of the screen to indicate their choice. After the subject has indicated their choice, all the remaining gray boxes on the screen reveal their colors and a message is displayed to inform the subject whether or not they were correct. The colors change from trial to trial.

There are two conditions – the fixed win condition, in which the subject is awarded 100 points for a correct decision regardless of the number of boxes opened, and the decreasing win condition, in which the number of points that can be won for a correct decision starts at 250 and decreases by 10 points for every box touched. In either condition, an incorrect decision costs 100 points.

Outcome Measures

The eight IST outcome measures cover errors, latency, total correct trials, mean number of boxes opened per trial, and probability of the subject's decision being correct based on the available evidence at the time of the decision.

Test Modes

IST has two modes:

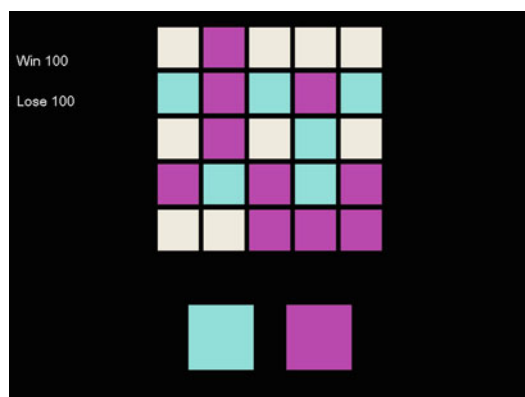
- Fixed win-decreasing win (after practice trials, the fixed win stage precedes the decreasing win stage)
- Decreasing win-fixed win (after practice trials, the decreasing win stage precedes the fixed win stage)

Stop Signal Task (SST)

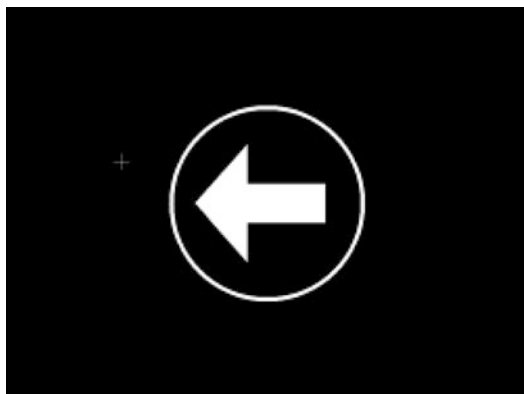
See Fig. 22.

Overview

SST is a classic stop signal response inhibition test, which uses staircase functions to generate an estimate of stop signal reaction time.



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Cambridge Neuropsychological Test Automated Battery, Fig. 22 Stop signal task

This test gives a measure of an individual's ability to inhibit a prepotent response.

Administration Time

Up to 20 min

Task

This test consists of two parts.

In the first part, the participant is introduced to the press pad, and told to press the left hand button when they see a left-pointing arrow, and the right hand button when they see a right-pointing arrow. There is 1 block of 16 trials for the participant to practice this.

In the second part, the participant is told to continue pressing the buttons on the press pad when they see the arrows, as before, but, if they hear an auditory signal (a beep), they should withhold their response and not press the button.

Outcome Measures

SST has five outcome measures, each of which can have various options applied to it. The SST measures cover direction errors, proportion of successful stops, RT on GO trials, SSD (50%), SSRT.

Test Modes

SST has one mode: clinical.

Social Cognition

A range of disorders are known to affect social cognition and there is an expanding research field

examining how such conditions may bias cognitive processes involved in social interaction. This domain is assessed by: Emotion Recognition Task (ERT).

Emotion Recognition Task (ERT)

Overview

ERT measures the ability to identify emotions in facial expressions. The participant is shown a series of faces which appear on the screen briefly and asked to identify the emotion (happiness, sadness, anger, disgust, surprise and fear).

Administration Time

Around 10 min, depending on level of impairment.

Task

One hundred and eighty stimuli, which are computer morphed images derived from the facial features of real individuals each showing a specific emotion, are displayed on the screen, one at a time, in two blocks of ninety. Each face is displayed for a short while (200 ms) and then immediately covered up, and then six buttons are displayed, each describing an emotion which could be portrayed in the photograph. The participant must decide which is the appropriate button to describe the emotion and touch the button. There are fifteen different photographs for each of the six emotions, each showing different levels of intensity.

Outcome Measures

The outcome measures for ERT cover percentages and numbers correct or incorrect, and overall response latencies. Results can be looked at across individual emotions, or across all emotions at once.

Test Modes

ERT is available for clinical trials immediately, and will be available for academic research in CANTABeclipse 5. Please contact Cambridge Cognition for further information.

ERT takes around 10 min to administer in healthy individuals.

Other Tests

Visual Analogue Scales (VAS)

Overview

Visual Analogue Scales are psychometric response scales which can be used as a measurement instrument for subjective states. The CANTAB VAS assess subjective measurements of drug effect, energy levels, sickness, alertness and mood.

Administration Time

Around 5 min, depending on level of impairment.

Task

The participant must respond to sixteen questions as they appear on the screen by touching the on-screen slider and moving it to the appropriate position on the scale.

Outcome Measures

The outcome measures for this test allow you to look at the data on a question-by-question basis.

Test Modes

Please contact Cambridge Cognition for information about availability for academic research.

Historical Background

Grounded in the neurosciences, the CANTAB[®] neuropsychological tests were developed more than 21 years ago at the University of Cambridge by Professors Robbins and Sahakian, to enable detailed translational assessment and evaluation of cognitive function. Lesion, neuroimaging, clinical and psychopharmacological studies have enabled a unique understanding of the structural, clinical and biochemical sensitivities of each of the tests. (CANTAB)

The CANTAB battery was developed for the assessment of cognitive deficits in humans with neurodegenerative diseases or brain damage (Fray and Robbins 1999). It consists of a series of inter-related computerized tests of memory, attention, and executive function, administered via a touch-sensitive screen. It allows a decomposition of

complex tasks commonly used in clinical assessment into their cognitive components and enables the extrapolation of findings from the animal literature. Tests include versions of the Wisconsin Card Sorting Test and the Tower of London and also the Delayed Matching to Sample test, widely used in monkeys for visual recognition memory. The tests are constructed in such a way that they may be given to animals (monkeys) with minimal change. The nonverbal nature of the CANTAB tests makes them largely language independent and culture free. CANTAB has been standardized on a large, predominantly elderly, population and validated in neurosurgical patients as well as in patients with basal ganglia disorders, Alzheimer's disease, depression, and schizophrenia. In addition, CANTAB has been used to evaluate: (a) the therapeutic effects of dopaminergic and cholinergic medication in neurodegenerative disease; (b) cognition in 5–11-year old normal, learning-disabled, and autistic children; (c) deficits in patients with HIV infection; and (d) early, asymptomatic Huntington's disease. The latter illustrate its usefulness in early identification of progressive disorders. It is suggested that the battery should have particular utility across a wide range of age and intelligence in longitudinal assessment after exposure to toxicants, and allow meaningful comparison with experimental studies of toxic effects in other species.

There is emerging evidence to support the involvement of frontal cortex in autism. CANTAB is particularly useful in helping study the cognitive profile of children who have autism and related disorders.

Psychometric Data

CANTAB tests are sensitive to cognitive changes caused by a wide range of CNS disorders and medication effects.

Where error scores are a key outcome measure, CANTAB tests are graded in difficulty to avoid ceiling effects.

Where accurate measurement of latency is important, responses are made via a press pad. Elsewhere, engaging touch-screen technology maximizes compliance.

The majority of CANTAB tests are independent of language and culture.

Clinical Uses

The following cognitive and other disorders have been investigated using CANTAB®:

AD/HD – Attention deficit hyperactivity disorder	Lesion in orbitofrontal cortex
AIDS dementia complex	Liver failure
Alcoholism	Long-term health effects of diving
Amphetamine addiction	Machado-Joseph disease
Amygdalo-hippocampectomy	Mad Hatter's disease
Anorexia nervosa	Manic depression
Anterior parietal damage	Melancholia
Antisocial behavior	Mercury poisoning
Antisocial personality disorder	Mild cognitive impairment (MCI)
Anxiety	Motor neuron disease
Attention deficit-hyperkinetic disorder	Multiple sclerosis
Autism	Multiple system atrophy
Basal ganglia lesions	Narcolepsy
Bipolar disorder	Neuronal migration disorders
Borderline personality disorder	Normal pressure hydrocephalus
Camptocormia	Obsessive compulsive disorder
Capgras syndrome	Organophosphate pesticide exposure
Carcinoid syndrome	Panic disorder
Chronic drug misuse	Paraphrenia
Chronic fatigue syndrome	Parkinson's disease
Chronic occupational solvent encephalopathy	Periventricular brain insult
Critical illness requiring intensive care	Personality disorder
Dementia Alzheimer-type (DAT)	Petrol (gasoline) sniffing
Dementia lewy body type	Phenylketonuria
Dementia of frontal type	Post-concussion syndrome
Developmental dyslexia	Premature birth needing intensive care

(continued)

Diabetes	Premenstrual dysphoric disorder
Dorsolateral frontal cortical compression	Progressive supranuclear palsy
Down's syndrome	Psychopathy
Drug abuse	Psychosis
Dysexecutive syndrome	Questionable Dementia
Frontal lobe damage	Renal Cancer
Frontal lobe excision	Roifman syndrome
Frontal variant frontotemporal dementia	Schizoaffective disorder
Gluten ataxia	Schizophrenia
Hallucinosis	Seasonal affective disorder
Head injury	Self harm
Hearing loss	Semantic dementia
Heart disease	Specific language impairment
Heart failure	Social withdrawal in Schizophrenia
Heavy social drinking	Solvent encephalopathy
Hepatic encephalopathy	Spina bifida
Heroin addiction	Steele-Richardson-Olzewski syndrome
Herpes encephalitis	Stiff Person syndrome
Hippocampal atrophy	Striatocapsular infarct
HIV/AIDS	Subarachnoid hemorrhage
Huntington's disease	Substance abuse
Hydrocephalus	Tardive dyskinesia
Hypercortisolemia	Temporal lobe excision
Hyperostosis frontalis interna	Temporal lobe lesion
Hypertension	Tinnitus
Insomnia	Tourette's syndrome
Korsakoff syndrome	Traumatic brain injury
Late paraphrenia	Trichotillomania
Lead exposure	Tuberous sclerosis
Left ventricular systolic dysfunction	White matter lesions

Drugs

Pharmacological studies (academic research) have been carried out on the following drugs using CANTAB:

Alcohol	Flumazenil	Modafinil
Amisulphiride	Fluoxetine	Neuroleptic
Amphetamine	Galantamine	Nicotine

(continued)

Antipsychotic medication	<i>Ginkgo biloba</i>	Olanzapine
Antiretroviral therapy	Glyburide	Opiates
Atomoxetine	Guanfacine	Paroxetine
Branch chain amino acid drink	Highly active antiretroviral therapy (HAART)	Pergolide
Bromocryptine	Haloperidol	Perindopril
Buspirone	Heroin	Petrol/ Gasoline
Caffeine	Hydrocortisone	Phenserine
Cannabis	Idazoxan	Quetiapine
Chlorpromazine	Idazoxan plus Clonidine	Risperidone
Clonidine	Interferon	Ritalin
Clozapine	Interleukin-2	Rivastigmine
Cocaine	Kava	Rosiglitazone
delta-9 tetrahydrocannabinol	Ketamine	RU-486
Dexamphetamine	L-Dopa	Scopolamine
Diazepam	Lecithin	SGS742
Donepezil	MDMA	Sulpiride
Dopaminergic medication	Metamphetamine	Tacrine
Ecstasy	Methylphenidate	Tryptophan
Endozepines	Mifepristone	Tyrosine

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See Also

► [CANTAB](#)

References and Reading

Cambridge Cognition Ltd. Cambridge automated neuropsychological test automated battery. www.cantab.com

Fray, P. J., & Robbins, T. W. (1999). CANTAB battery: Proposed utility in neurotoxicology. *Neurotoxicology and Teratology*, 18, 499–504.

Ozonoff, S., Cook, I., Coon, H., Dawson, G., Joseph, R. M., Klin, A., et al. (2004). Performance on Cambridge Neuropsychological Test Automated Battery subtests sensitive to frontal lobe function in people with autistic disorder: Evidence from the Collaborative Programs of Excellence in Autism network. *Journal of Autism and Developmental Disorders*, 34, 139–150.

Camouflaging Autistic Traits Questionnaire (CAT-Q)

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Research Department of Clinical, Educational and Health Psychology, University College London, London, UK

Synonyms

[CAT-Q](#)

Description

The Camouflaging Autistic Traits Questionnaire (CAT-Q) is a standardized self-report measure of camouflaging behaviors in autistic and non-autistic adults. It comprises 25 items and takes around 5 min to complete, on paper or online. The scale consists of three sub-scales: compensation (strategies used to overcome social difficulties associated with autism), masking (strategies used to hide autistic characteristics or present a less autistic persona), and assimilation (strategies used to avoid standing out during social interactions). In addition to sub-scale scores, a total camouflaging score can be calculated as the sum of all scores (ranging from 25 to 175, with higher scores indicating greater camouflaging). The CAT-Q is completed by the individual themselves, reflecting on their own behaviors at the present time.

Historical Background

Camouflaging describes the use of strategies, whether deliberate or automatic, to minimize the appearance of autistic characteristics during social interactions and to compensate for social difficulties associated with autism (Hull et al. 2017). Camouflaging has also been proposed as a potential explanation for the underdiagnosis of autism in females (Lai et al. 2015); if girls and



women use more, or more successful, camouflaging strategies to hide or compensate for their autism, they are less likely to be identified by clinical services. This can lead to lack of support and acceptance, as well as the potential for resulting mental health difficulties (Bargiela et al. 2016; Milner et al., 2019). Recent research has also suggested that camouflaging strategies themselves may be associated with negative mental health outcomes for autistic adults (Cage et al. 2018; Hull et al. 2017) and young people (Tierney et al. 2016).

Until recently there has been no way to measure how much someone is camouflaging. Some researchers have quantified camouflaging as the discrepancy between an individual's internal autistic experience (such as level of autistic traits) and the external behavioral presentation (such as ADOS score; Lai et al. 2017). This approach has generally concluded that females camouflage more than males (Lai et al. 2017, 2018; Parish-Morris et al. 2017; Ratto et al. 2018).

However, the discrepancy approach to measuring camouflaging requires multiple, often time-consuming measures to be taken for each individual and only measures the effect camouflaging has on behavior rather than the effort put into camouflaging. An alternative measurement is the CAT-Q, which directly measures the extent of camouflaging strategies self-reported by an individual. This makes measurement of camouflaging quick and easy, and the CAT-Q is freely available to download (Hull et al. 2018).

Psychometric Data

There is limited psychometric data for this measure, particularly across cultures, abilities, and age groups. The CAT-Q has been validated in autistic and non-autistic adult males and females in a large sample ($N = 832$; Hull et al. 2018) and demonstrated good internal consistency ($\alpha = 0.94$ for total camouflaging scale) and acceptable test-retest reliability in a smaller

sample ($ICC [C,1] = 0.77$). Measurement invariance has also been demonstrated between autistic and non-autistic males and females, demonstrating that the CAT-Q can be used with individuals regardless of whether they have received a formal diagnosis of autism. This is particularly important as camouflaging is likely to exist along a continuum, similarly to autistic traits (Constantino, 2011), and individuals who camouflage extensively may consequently not meet current diagnostic criteria for autism (Kreiser and White 2014). There is some evidence to support the convergent validity of the CAT-Q, with higher scores associated with higher levels of autistic-like traits (Hull et al. 2018).

Clinical Uses

Camouflaging has been associated with mental health difficulties including depression (Lai et al. 2017), anxiety (Hull et al. 2018), and suicidal thoughts (Cassidy et al. 2018). The CAT-Q can be used to identify autistic adults who may be a greater risk of these co-occurring conditions and help them access support. However, norms have not yet been established for either autistic or non-autistic adults; therefore clinically meaningful cutoffs have not been identified.

There is also potential for the CAT-Q to be used as part of the autism diagnostic assessment process in adults and adolescents. Adults, particularly women, who have not yet received an autism diagnosis, may camouflage their characteristics during autism assessments, leading to under-recognition of their level of need. Further clinical research is needed to examine exactly how the CAT-Q can be integrated into gold standard assessment processes.

See Also

- [Bias in Assessment Instruments for Autism](#)
- [Social Camouflaging in Adults with ASD](#)
- [Suicide Rates in Adults with Autism](#)

References and Reading

- Bargiela, S., Steward, R., & Mandy, W. (2016). The experiences of late-diagnosed women with autism Spectrum conditions: An investigation of the female autism phenotype. *Journal of Autism and Developmental Disorders*, 46(10), 3281–3294.
- Cage, E., Di Monaco, J., & Newell, V. (2018). Experiences of autism acceptance and mental health in autistic adults. *Journal of Autism and Developmental Disorders*, 48(2), 473–484.
- Cassidy, S., Bradley, L., Shaw, R., & Baron-Cohen, S. (2018). Risk markers for suicidality in autistic adults. *Molecular Autism*, 9(42), 1–14.
- Constantino, J. N. (2011). The quantitative nature of autistic social impairment. *Pediatric Research*, 69(5 Pt 2), 55R–62R. <https://doi.org/10.1203/PDR.0b013e318212ec6e>.
- Hull, L., Petrides, K. V., Allison, C., Smith, P., Baron-Cohen, S., Lai, M.-C., & Mandy, W. (2017). “Putting on my best normal”: Social camouflaging in adults with Autism Spectrum Conditions. *Journal of Autism and Developmental Disorders*, 47(8), 2519–2534.
- Hull, L., Mandy, W., Lai, M.-C., Baron-Cohen, S., Allison, C., Smith, P., & Petrides, K. V. (2018). Development and validation of the camouflaging autistic traits questionnaire (CAT-Q). *Journal of Autism and Developmental Disorders*, 49(3), 819–833.
- Hull, L., Lai, M.-C., Baron-Cohen, S., Allison, C., Smith, P., Petrides, K. V., & Mandy, W. (2019). Gender differences in self-reported camouflaging in autistic and non-autistic adults. *Autism*, 136236131986480. <https://doi.org/10.1177/1362361319864804>.
- Kreiser, N. L., & White, S. W. (2014). ASD in females: Are we overstating the gender difference in diagnosis? *Clinical Child and Family Psychology Review*, 17(1), 67–84.
- Lai, M.-C., Lombardo, M. V., Auyeung, B., Chakrabarti, B., & Baron-Cohen, S. (2015). Sex/gender differences and autism: Setting the scene for future research. *Journal of the American Academy of Child and Adolescent Psychiatry*, 54(1), 11–24.
- Lai, M.-C., Lombardo, M. V., Ruigrok, A. N. V., Chakrabarti, B., Auyeung, B., Szatmari, P., et al. (2017). Quantifying and exploring camouflaging in men and women with autism. *Autism*, 21(6), 690–702.
- Lai, M.-C., Lombardo, M. V., Chakrabarti, B., Ruigrok, A. N., Bullmore, E. T., Suckling, J., et al. (2018). Neural self-representation in autistic men and women and association with ‘compensatory camouflaging’. *Autism*. <https://doi.org/10.1177/1362361318807159>.
- Milner, V., McIntosh, H., Colvert, E., & Happé, F. (2019). A qualitative exploration of the female experience of autism Spectrum disorder (ASD). *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03906-4>.
- Parish-Morris, J., Liberman, M. Y., Cieri, C., Herrington, J. D., Yerys, B. E., Bateman, L., et al. (2017). Linguistic camouflage in girls with autism Spectrum disorder. *Molecular Autism*, 8(1), 48.
- Ratto, A. B., Kenworthy, L., Yerys, B. E., Bascom, J., Trubanova, A., White, S. W., et al. (2018). What about the girls? Sex-based differences in autistic traits and adaptive skills. *Journal of Autism and Developmental Disorders*, 48(5), 1698–1711.
- Tierney, S., Burns, J., & Kilbey, E. (2016). Looking behind the mask: Social coping strategies of girls on the autistic spectrum. *Research in Autism Spectrum Disorders*, 23, 73–83.

Can't Versus Won't Dilemma

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Definition

One common issue faced by parents, teachers, and caregivers of individuals with autism is understanding when a behavioral difficulty is due to a skill deficit (“can’t”), rather than due to deliberate noncompliance (“won’t”). Caregivers who attribute behavior problems to deliberate noncompliance often see the behavior as rooted in laziness, stubbornness, or defiance. This attribution has multiple negative consequences, including increased frustration and stress for the caregiver, as well as use of ineffective or confrontational behavior management strategies.

Even caregivers who have some understanding of autism may believe that the individual with autism is purposely engaging in misbehavior, and consequently become embroiled in an unproductive power struggle. The confusing behavioral picture presented by individuals with autism contributes to this misunderstanding. For example, individuals with autism often have a very typical physical appearance, so the caregivers’ natural inclination is to expect age-appropriate skills and behavior. In addition, many individuals with autism, including those with language impairments, can repeat back

verbal directions even when they have not fully understood the content of what was said, giving a misimpression about their level of understanding. Furthermore, poor social insight and communication deficits may mean that individuals with autism are unable to recognize and communicate their own lack of skill or need for assistance, or may cause them to question directions from others in a manner that is perceived as argumentative or disrespectful. Perhaps most confusing for caregivers is the unusual scatter of strengths and weaknesses shown by individuals with autism, as well as their difficulty in generalizing the use of skills from one context to another. For example, parents of a bright 14 year old with autism may simply have difficulty understanding how their son can have extensive working knowledge of his computer, yet not be able to successfully operate the microwave. A teacher of a more impaired 7 year old may be confused as to why the student can independently use the toilet at home but repeatedly soils her clothing at school.

In general, when faced with a “can’t versus won’t” dilemma, it is more productive to begin by assuming that the individual with autism “can’t” and then conduct a behavioral assessment focused on the symptoms of autism that may be impeding his or her behavioral success. The caregiver should consider the ways in which the individual’s unique profile of strengths and weaknesses in communication, socialization, flexibility and interests, sensory responses, and learning style may be contributing to the behavioral difficulty. That information can then be used to generate positive, proactive strategies to help support desired behaviors in the future.

References and Reading

- Marcus, L. M., & Palmer, A. (2010). Families of children with autism: What educational professionals should know. In F. A. Karnes & K. R. Stephens (Eds.), *The practical strategies series in autism education*. Austin: Prufrock.
- Marcus, L. M., Kuncie, L. J., & Schopler, E. (2005). Working with families. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive*

developmental disorders (Vol. II, pp. 1055–1086). Hoboken: Wiley.

- Notbohm, E. (2005). *Ten things every child with autism wishes you knew*. Arlington: Future Horizons.
- Schopler, E. (1995). *Parent survival manual: A guide to crisis resolution in autism and related developmental disorders*. New York: Plenum.

Canada and Autism

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Background

In Canada, as in many other countries, a growing public awareness of autism spectrum disorder (ASD) has emerged in the context of the evidence for a rising prevalence and the existence of life-long vulnerabilities and complex medical and mental health comorbidities (Anagnostou et al. 2014). Parents and carers have been instrumental in raising this awareness, and the government has responded with the commissioning of new services, principally focused on the needs of children (Motiwala et al. 2006; Auditor General of Ontario 2013). As discussed subsequently, despite increased public funding for ASD-focused services, significant inequalities in service provision exist for specific groups, including adults and higher-functioning individuals (Shattuck et al. 2012), newly arrived immigrants (Khanlou et al. 2017), and individuals with complex health and social care needs (Autism Ontario 2008). Moreover, service inequalities exist between different provinces (Eggleton and Keon 2007). It is now widely recognized that there is an urgent need for uniformly accessible services for all individuals with ASD, irrespective of age or any other characteristic. The provision of a uniform service can only truly be achieved through federal

involvement and ultimately a national policy or legislative framework. Such a strategy has widespread support and in 2007 was a suggestion made by the Senate Select Committee (Eggleton and Keon 2007), although at this stage there is no indication of the adoption of a federal initiative.

By way of background, in Canada, each province (of which there are ten, along with three territories) is responsible for providing healthcare and social services for all individuals. This of course includes children and adults with developmental disabilities, such as ASD. Funds for services are raised through taxation, and each province implements its own model of service delivery. Importantly, for healthcare federal policy – by way of the Canada Health Act – still provides some oversight and direction, including the directive that universal access to publically funded “medically necessary” services must be ensured for all. However, the significant power devolved by the government to provincial policymakers does result in interprovincial variation in services. It is difficult to articulate the minutiae of province-by-province differences, and so this present entry will provide a simple overview, drawing examples from individual provinces but not attempting to present a detailed and comprehensive picture of ASD services across Canada.

Overview of Current Treatments and Centers

Early Diagnosis and Intervention

One significant development across Canada has been the widespread availability of early intervention services for children with ASD (Anagnostou et al. 2014; Volden et al. 2015). Early intervention, based on the principles of Applied Behavior Analysis (ABA), is funded at the provincial level by those Ministries responsible for child, family, and community care. For example, in Ontario such services are commissioned by the Ministry of Child and Youth Services (separate from the Ministry of Health which is responsible for healthcare), in Alberta by the Ministry of Children’s Services, and in British Columbia (BC) by

the Ministry of Children and Family Development. Early intervention programs (Intensive Behavioural Intervention or IBI) involve one-on-one therapy during which the child is engaged in a series of discrete trials involving reward contingencies to facilitate learning and generalization. The trials themselves focus on language and communication, as well as social and adaptive skills. Intensive intervention typically involves 20–40 h of 1:1 therapy per week over a period of 2 years, which has been shown to maximize the chance of improvement (Reitzel et al. 2015 and references therein).

Research has consistently shown that early language and cognition are strong predictors of outcome in later childhood and into adulthood (Henninger and Taylor 2013), although outcome in later adult life may be related more closely to early social adjustment (Howlin et al. 2013). The increased availability of early intervention services has therefore been welcomed by all involved in ASD policy. Moreover, research has shown the success of such programs (Warren et al. 2011), although the research is not clear cut (Reichow et al. 2012). There is still much interprovincial variation in service delivery: for example, in Nova Scotia publicly funded IBI services are available to all young children with ASD, and this level of care is echoed in Ontario. As would be expected, with the evidence for a rising prevalence, currently estimated at ~1% (or 67,000 children age between 3 and 20 years) in Canada (Anagnostou et al. 2014), the demand on these services is large, and consequently wait times are often long between receiving a diagnosis and accessing IBI. Some form of triage is often in place to target those children who are more likely to benefit. For example, in Ontario IBI is reserved for younger children who have “severe autism.” Although “severe” is not explicitly defined, clinicians responsible for intake use a variety of screening tools to determine eligibility. However, even targeting services in this way, wait times are still substantial. For example, in 2013, the waitlist for IBI in Southern Ontario, comprising parts of the Greater Toronto Area (GTA) and the surrounding “Golden Triangle,” included 1748 children (Auditor General of Ontario 2013). Slightly

different service provision is seen in BC and Alberta, where public funding is provided to partly offset the costs of private intervention sourced by the family itself.

Early intervention demands that diagnosis is made as early as possible, which is dependent on the availability of expert clinical diagnostic services. Although certain groups, such as the American Academy of Pediatrics, have recommended universal screening for ASD between the ages of 18 and 24 months (Johnson and Myers 2007), the Canadian Pediatric Society instead advises developmental surveillance (Anagnostou et al. 2014). In parts of Canada, this approach has been facilitated through the use of brief, validated, and reliable screening questionnaires (Zwaigenbaum 2009). While family physicians are in a position to provide early screening, however, the diagnosis itself may be delayed through the unavailability of appropriate expertise. While family physicians are the frontline staff involved in developmental surveillance, the responsibility for early diagnosis rests with existing services, such as developmental pediatrics and child psychiatry. The provision of adequate training to primary and secondary healthcare workers is therefore crucial.

School-Aged Children

Services for school-aged children have also seen progress in recent years. In some provinces, the focus remains on early intervention for pre-schoolers, whereas other provinces have also developed services for children with ASD up to the age of 18 years. In Ontario, for example, services have developed to meet the varying needs of this population using ABA principles. Among some children with ASD, particularly those who function typically, the focus may be on social skills, often, although not necessarily, delivered in a group setting, whereas for others, it may be behavioral or adaptive needs. The emphasis is on mastering skills one at a time and then learning to apply these in everyday settings. In 2013, the median age of children accessing this service was 8 years, with 90% age 14 or younger indicating that older children and those in

transition (i.e., age 17–18 years) may not be accessing services. In addition to therapy that targets the core symptoms of ASD, there is also a need for mental health services to address the high level of emotional distress and comorbidity among children with ASD (Drmic and Szatmari 2014). The impact on later outcome for therapy aimed at school-aged children will need to be fully evaluated.

Adults

Services for adults with ASD in Canada have lagged behind those for children (Stoddart et al. 2013). It is estimated that in the region of 4900, teenagers with ASD in Canada reach their 18th birthday each year (Shattuck et al. 2012). Based on current epidemiological estimates, as many as 70% of these may have IQs in the typical range (Centers for Disease Control and Prevention 2014). Despite this, however, the outcome for many is poor. For example, studies of outcomes consistently find low to modest levels of independence and the persistence of core phenotypic traits and associated developmental and mental health vulnerabilities beyond childhood (Howlin et al. 2013). It is clear, therefore, that for an individual with ASD – irrespective of their IQ – health and community/social care needs will remain significant throughout much of their lives (Stoddart et al. 2013; Autism Ontario 2008). With increases in life expectancy, this potentially represents a public health crisis. Indeed, on an individual basis, the lifetime costs associated with ASD have been estimated at up to \$2.44M US dollars in the USA and UK (Buescher et al. 2014).

Healthcare for adults with ASD in Canada is met according to the universal healthcare principles of the Canada Health Act. Specialist mental health services for adults with developmental disabilities do exist but are not government mandated. Such services focus on the mental health and behavioral needs of adults with IQs below 70, and as such, many adults with ASD will not meet the access criteria. For those adults who do have IQs 70 or above, it is expected that existing mental health services will meet their needs, although this is often not the case, with some excluded from community mental health services

as a result of their ASD diagnosis, essentially leaving them “doubly socially excluded.” This is even more concerning when the statistics are considered: in one study examining comorbidity, as many as 70% of young adults with ASD had experienced one or more episode of major depression, with 50% experiencing recurrent depression and 50% describing an anxiety disorder (Lake et al. 2014).

In Canada, vocational and social care needs, including the provision of supported accommodation, are met by the Ministry responsible for the commission of community and social services. Among those who have an intellectual disability, i.e., evidence of IQ <70 along with adaptive impairments, a variety of services are available, although the large demand for services results in long wait times. Such services include supported living and respite, supported employment and other vocationally centered programs, and behavioral services. Adults with ASD who have IQs above 70, however, are generally denied access to such services (Stoddart et al. 2013; Autism Ontario 2008). As such, the emphasis has been on private initiatives. For example, in Alberta the Sinneave Foundation in collaboration with the consulting firm Meticulon provides individuals with autism an opportunity to work with the IT field while providing them with the appropriate wages. The availability of similar initiatives exist across the country.

Specific Issues

Rural communities have identified a definite lack of resources and services when tending to the needs of their children. The lack of medical support has made life difficult for families and providing proper treatment difficult as there are no professionals or workers to provide the training and insight to how to provide the best environment for their children to grow (Hoogsteen and Woodgate 2013). Similar barriers to service access is seen among newly arrived immigrants to Canada (Khanlou et al. 2017). Rates of ASD have been shown to be 36% higher in children of immigrant mothers. Considering the fact that migration is an integral part of Canadian federal policy and that a significant proportion of

Canada’s population is made up of newly arrived immigrants, this represents a major area of need. Addressing this requires a better understanding of the barriers to care, which will include, for example, language and knowledge of existing structures, as well as federal policy to overcome these barriers.

Other specific issues relate to the organization of existing structures of care. For example, transition planning and implementation continues to present a major challenge for families, with the negotiation of adult services an extra hurdle at an already difficult time (Gorter et al. 2011). Hospitals themselves are often not set up to manage individuals with ASD and other developmental disabilities effectively. For example, it has been argued that emergency departments are poorly equipped to accommodate patients with ASD appropriately (Nicholas et al. 2016). Key problems identified are lack of communication and training. The same concern has been expressed in relation to primary care, although initiatives such as the Primary Care Developmental Disabilities Network in Ontario attempts to overcome this barrier through more adequate training (Sullivan et al. 2011).

Overview of Research Directions

Canada has a long tradition of research in ASD, with basic and applied scientific approaches being used to further the knowledgebase on ASD. These comprise a number of multisite, high-impact, studies. By way of example, two research programs are briefly highlighted below.

Pathways

This large, multisite project comprises researchers across five Canadian provinces (Ontario, Quebec, Nova Scotia, Alberta, and British Columbia). It has recruited newly diagnosed children with ASD, aged between 2 and 4 years, and prospectively following these children to examine their developmental trajectories (Szatmari et al. 2015). Across time, data is collected at four separate intervals, with core symptomatology, behavior, and adaptive function all being measured in detail.

This is a powerful design for a number of reasons. Importantly, most previous studies have recruited participants at different points in the natural history of their disorder. Without sampling an inception cohort (a group assembled at a common time point early in the development of the disorder), there is no way of ensuring that all subgroups of children with ASD are included in the sampling frame. Additionally, in many other studies, individuals with ASD are recruited through clinics, which may introduce bias into the outcomes being observed.

Genetics

Canada has a long tradition of genetics research in autism spectrum disorder, utilizing state-of-the-art techniques to attempt to unravel the genetic component of ASD's etiology. In recent years, the focus has been on identifying de novo and inherited unbalanced copy number variation (CNV) (Pinto et al. 2014), including variation at the single nucleotide level (SNV). The current research activities of this multisite, multidisciplinary group focus on the MSSNG project, a collaboration between Google and Autism Speaks under the directorship of Prof. Steve Scherer that aims to sequence the DNA of over 10,000 families with one or more members affected by autism (Yuen et al. 2017).

Current Controversies

The development of services in Canada in recent years has seen a major injection of money, particularly in relation to IBI programs aimed at young children. These programs have been developed along evidence-based lines and are constantly evaluated to make certain of clinical impact. The provincial-led nature of these programs means that there remain inequalities of care across the country as a whole, with rural communities often having the least joined-up level of care. Furthermore, adults, and particularly those who are higher functioning, have seen the least in the way of service development. The need for a federal approach to ASD-specific policy has been raised but not yet adopted.

See Also

- ▶ [DSM-III](#)
- ▶ [DSM-III-R](#)
- ▶ [ICD 10 Research Diagnostic Guidelines](#)

References and Reading

- Anagnostou, E., et al. (2014). Autism spectrum disorder: Advances in evidence-based practice. *Canadian Medical Association Journal*, 186(7), 509–519.
- Auditor General of Ontario. (2013). *2013 annual report*. Toronto: Queen's Printer for Ontario.
- Autism Ontario. (2008). *Forgotten: Ontario adults with autism and adults with Aspergers*. Toronto: Autism Ontario.
- Buescher, A. V., Cidav, Z., Knapp, M., & Mandell, D. S. (2014). Costs of autism spectrum disorders in the United Kingdom and the United States. *JAMA Pediatrics*, 168(8), 721–728.
- Centers for Disease Control and Prevention. (2014). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2010. *MMWR*, 63(2), 1–21.
- Drmic, I. E., & Szatmari, P. (2014). Emotional dysregulation and co-morbidity in Autism Spectrum Disorder (ASD). *CEPiP*, 1, 119–131.
- Eggleton, A., & Keon, W. J. (2007). *Final report on: The enquiry on the funding for the treatment of autism: Pay now or pay later: Autism families in crisis*. Ottawa: Standing Senate Committee.
- Gorter, J.-W., Stewart, D., & Woodbury-Smith, M. (2011). Youth in transition: Care, health and development. *Child: Care, Health and Development*, 37(6), 757–763.
- Henninger, N. A., & Taylor, J. A. (2013). Outcomes in adults with autism spectrum disorders: A historical perspective. *Autism*, 17, 103–116.
- Hoogsteen, L., & Woodgate, R. (2013). Embracing autism in Canadian rural communities. *Australian Journal of Rural Health*, 21(3), 178–182.
- Howlin, P., Moss, P., Savage, S., & Rutter, M. (2013). Social outcomes in mid- to later adulthood among individuals diagnosed with autism and average nonverbal IQ as children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(6), 572–581.
- Johnson, C. P., & Myers, S. M. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120, 1183–1215.
- Khanlou, N., et al. (2017). Access barriers to services by immigrant mothers of children with autism in Canada. *International Journal of Mental Health & Addiction*, 15, 239–259. <https://doi.org/10.1007/s11469-017-9732-4>.
- Lake, J. K., Perry, A., & Lunskey, Y. (2014). Mental health services for individuals with high functioning autism spectrum disorder. *Autism Research and Treatment*, 2014, 502420.

- Motiwalla, S. S., Gupta, S., Lilly, M. B., Ungar, W. J., & Coyte, P. C. (2006). The cost-effectiveness of expanding intensive behavioural intervention to all autistic children in Ontario. *Healthcare Policy*, 1, 135–151.
- Nicholas, D., Zwaigenbaum, L., Muskat, B., Craig, W., Newton, A., Kilmer, C., et al. (2016). Experiences of emergency department care from the perspective of families in which a child has autism spectrum disorder. *Social Work in Health Care*, 55(6), 409–426.
- Pinto, D., et al. (2014). Convergence of genes and cellular pathways dysregulated in autism spectrum disorders. *American Journal of Human Genetics*, 94(5), 677–694.
- Reichow, B., Barton, E. E., Boyd, B. A., & Hume, K. (2012). Early intensive behavioral intervention (EIBI) for young children with autism spectrum disorders (ASD). *Cochrane Database of Systematic Reviews*, 10, CD009260. <https://doi.org/10.1002/14651858.CD009260.pub2>.
- Reitzel, J.-A., Summers, J., & Drmic, I. (2015). Psychological treatment of autism spectrum disorder. In M. Woodbury-Smith (Ed.), *Clinical topics in disorders of intellectual development* (pp. 201–235). London: Royal College of Psychiatrists.
- Shattuck, P. T., et al. (2012). Services for adults with an autism spectrum disorder. *Canadian Journal of Psychiatry*, 57(5), 284–291.
- Shepherd, C., & Waddell, C. (2015). A qualitative study of autism policy in Canada: Seeking consensus on children's services. *Journal of Autism and Developmental Disorders*, 45(11), 3550–3564.
- Simonoff, E., Pickles, A., Charman, T., et al. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47, 921–929.
- Stoddart, K. P., Burke, L., Muskatt, B., Manett, J., Duhaime, S., Accardi, C., et al. (2013). *Diversity in Ontario's youth and adults with autism spectrum disorders: Complex needs in an unprepared system*. Toronto: The Redpath Centre.
- Sullivan, W. F., et al. (2011). Primary care of adults with developmental disabilities. Canadian consensus guidelines. *Canadian Family Physician*, 57(5), 541–553.
- Szatmari, P., Georgiades, S., Duku, E., et al. (2015). Developmental trajectories of symptom severity and adaptive functioning in an inception cohort of preschool children with autism spectrum disorder. *JAMA Psychiatry*, 72(3), 276–283. <https://doi.org/10.1001/jamapsychiatry.2014.2463>.
- Volden, J., Duku, E., Shepherd, C., Pathways in ASD Study Team, et al. (2015). Service utilization in a sample of preschool children with autism spectrum disorder: A Canadian snapshot. *Paediatric Child Health*, 20(8), e43–e47.
- Warren, Z., PcPheeters, M. L., Sathe, N., Foss-Feig, J. H., Glasser, A., & Veenstra-VanderWeele, J. (2011). A systematic review of early intensive intervention for autism spectrum disorders. *Pediatrics*, 127, e1303–e1311.
- Yuen, R. K. C., et al. (2017). Whole genome sequencing resource identifies 18 new candidate genes for autism spectrum disorder. *Nature Neuroscience*. <https://doi.org/10.1038/nn.4524>.
- Zwaigenbaum, L. (2009). Screening, risk and early identification of autism spectrum disorders. In D. G. Ameral & D. H. Geshwind (Eds.), *Autism spectrum disorders*. Oxford: Oxford University Press.

Canadian Certified Rehabilitation Counselor (CCRC)

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Candidate Genes in Autism

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Definition

Although twin and family studies show that genes play a critical role in determining the risk for autism, its specific genetic etiology remains largely unknown. A candidate gene is one for which there is some evidence of contribution to the etiology of a disorder but for which this has not yet been definitively demonstrated. These genes are identified by a variety of techniques including linkage analysis, association studies, cytogenetic analysis, studies of copy number variation, and next-generation sequencing. Typically, once a candidate gene has been identified, it is reinvestigated via analysis in independent patients' samples. Particularly for studies that rely on case-control comparisons, replication is essential to elevating a candidate gene to a "risk" gene.

Historical Background

Over the past decade, many studies have shown that autism is not a simple Mendelian disorder caused by a single gene at the population level. In the early phase of autism gene discovery, the majority of candidate genes were selected for study based on biological plausibility; that is, they were involved in some biological process that could conceivably play a role in ASD. These genes were then typically evaluated in candidate gene association studies in which one or a small number of common genetic polymorphisms in or near one or a small number of genes were evaluated in cases versus controls. If an overrepresentation of a particular allele or alleles was identified, the gene was considered a candidate ASD gene. These studies were based on the hypothesis that common alleles were responsible for the disorder.

Across all of medicine, the majority of such studies proved difficult to replicate. In retrospect, it is clear that approach had some significant limitations. Among these, the chances of choosing correctly among millions of genetic variations were low, the effect sizes carried by common alleles for most common medical conditions were much smaller than anticipated (resulting in studies that were in retrospect often markedly underpowered), and there were multiple potential confounds, including ancestral mismatching of cases and controls, that were difficult to control for. More recently, the approach has been replaced for the most part by genome-wide association studies, typically of large patient cohorts, that eliminate many of these difficulties. This approach has led to the identification of replicated risk alleles in many common medical conditions, including schizophrenia and bipolar disorder. To date, this approach has led to the identification of several new candidate genes in ASD, but these have not yet replicated in well-powered studies.

Over the last several years, the identification of candidate genes through studies of common variation has been complemented by studies of rare variation. Here again, it is common practice to pursue an initial observation with an attempted independent replication. With rare variations, the

infrequency of individual mutations and the overall genetic heterogeneity of autism may make such studies difficult to mount. A variety of approaches are being developed in an effort to provide a path to confirm candidate loci: these include assessing the total amount of rare variation in a gene in cases versus controls (as opposed to asking questions about one particular rare allele). This approach is often called a mutation burden analysis. In addition, there are ongoing efforts to take advantage of particular types of variation, including *de novo* mutations, to increase the power to detect and confirm the association of a gene or locus with ASD risk (Sanders et al. 2011).

Current Knowledge

Genome-Wide Linkage Studies

Linkage studies identify chromosomal loci inherited by affected individuals more frequently than expected by chance. These studies most often investigate multiplex families in which there is more than one affected person. DNA polymorphisms are used as markers of chromosomal loci throughout the genome. The closer the marker is to a disease gene, the more likely there is cosegregation between the marker and the phenotype under study. The likelihood that a locus is linked to the phenotype is represented as the LOD score (logarithm of the odds). For example, a LOD score of 3 means that there is 1,000 to 1 odds that the locus is linked to the phenotype. When the LOD score is more than 2.2, linkage is considered suggestive; 3.6 is considered significant (Lander and Kruglyak 1995). Linkage peaks have been found on almost every chromosome. As reviewed by Gupta and State (2007), loci with among the highest LOD scores are 3q26.32 (LOD 4.81), 2q31.1 (LOD 4.80), 17q11.2 (LOD 4.3), 17q21.32 (LOD 4.1), and 7q36.1 (LOD 3.7). For the most part, linkage studies in autism have failed to replicate each other, probably due to a number of reasons, such as nonuniform criteria for patient selection, differing sets of polymorphisms, and differing statistical methodologies. A few loci, such as 17q11-q21 and along 7q, have been

highlighted by more than one study (Abrahams and Geschwind 2008). Some of the genes implicated are *CNTNAP2* (contactin-associated protein-like 2), *EN2* (engrailed homeobox 2), *RELN* (reelin), *MET* (MET proto-oncogene), *CADPS2* (Ca²⁺-dependent activator protein for secretion 2), *ITGB3* (integrin beta3), and *SLC6A4* (solute carrier family 6) (Abrahams & Geschwind).

Linkage studies have also been conducted in consanguineous families using homozygosity mapping. Homozygous regions are parts of the genome where the identical chromosomal segment is inherited from both parents due to a recent common ancestor. In homozygosity mapping, it is hypothesized that the disorder is inherited as a recessive trait. Candidate genes found by this method include *DIA1* (deleted in autism-1), *NHE9* (sodium/proton exchanger 9), *PCDH10* (protocadherin 10), and *CNTN3* (contactin 3) (Morrow et al. 2008).

Candidate Gene and Genome-Wide Association Studies

Association studies determine whether there is a statistically significant relationship between exposure to the variant and increased (or decreased) population risk for the phenotype. Numerous genetic association studies have investigated common variants in one or a small number of candidate genes, often selected due to hypothesis-driven disease models. Since these studies are relatively inexpensive, many genes have been evaluated for association with autism, with multiple positive results. However, very few of them have been replicated (Gupta and State 2007). Some genes identified by this method are *GABRB3* (gamma-aminobutyric acid A receptor beta3), *GRIK2* (glutamate receptor ionotropic kainate 2 precursor), *SLC25A12* (solute carrier family 25 member 12), *MET*, *RELN*, *EN2*, *SLC6A4*, and *CNTNAP2* (Abrahams and Geschwind 2008; State 2010). Rare variants can also be investigated by association studies, but this method requires comprehensive resequencing of candidate genes in large cohorts and is expensive. In addition to common variants, rare variants in *CNTNAP2* have been associated with autism (Bakkaloglu et al. 2008).

More recently, high-resolution SNP arrays have enabled genome-wide association studies (GWAS), which query all genes rather than investigating a few candidate genes at a time. Three loci which have been associated with autism are chromosome 5p14.1, between the genes *CDH9* (cadherin 9) and *CDH10* (cadherin 10), chromosome 5p15, near the gene *SEMA5A* (semaphoring 5A), and chromosome 20p12.1, near the gene *MACROD2* (MACRO domain containing 2) (reviewed by State 2010). *CDH9* and *CDH10* are interesting candidate genes since they are involved in neuronal cell adhesion. *SEMA5A* has been implicated in axonal guidance.

Cytogenetic Analysis

Cytogenetic analysis is the study of chromosomal abnormalities such as inversions, translocations, duplications, deletions, and aneuploidies. Traditionally, these abnormalities have been detected via karyotype analysis (microscopic examination of chromosomes). A review by Veenstra-VanderWeele et al. (2004) calculated that 4.3% of the 1826 karyotypes published in the ASD literature are abnormal. Abnormalities have been found on every chromosome, indicating that no one rearrangement is responsible for any substantial fraction of cases. The most common chromosomal abnormality found in ASD is maternally inherited duplications at 15q11-q13 (Abrahams and Geschwind 2008). Some genes which have been implicated by cytogenetic analysis include *NLGN4X* (neuroligin 4X), *UBE3A* (ubiquitin protein ligase E3A), *GABRB3*, *CENTG2* (centaurin gamma 2), *SHANK3* (SH3 and multiple ankyrin repeat domains 3), and *CNTNAP2* (Abrahams & Geschwind; State 2010).

More recently, copy number variations (CNVs) have been investigated using microarrays. Genome-wide CNV analyses have found that CNVs are significantly enriched in neuronal cell adhesion molecules and the ubiquitin pathway (Glessner et al. 2009) and that recurrent de novo copy number variations (CNVs) at 7q11.23, 15q11.2-13.1, 16p11.2, and the *NXRNI* (neurexin 1) locus are strongly associated with autism (Sanders et al. 2011). The 7q11.23 region, the duplication of which is associated with autism in

this study, is previously known to be deleted in Williams-Beuren syndrome, which features a highly social personality, suggesting an intriguing correlation between copy number at this locus and sociability.

Whole-Exome Sequencing

With the development of high-throughput technologies which have been steadily decreasing in cost, it has become possible to obtain the DNA sequence for the entire coding region (exome) of the human genome. This has a profound influence on gene discovery in complex genetic disorders such as ASD. So far, most common variants appear to have small effects on disease risk. Even when large studies have been performed, the vast majority of the genetic contribution to disease risk remains unexplained. These findings suggest that rare variants with relatively large effects may account for a larger fraction of this missing risk than previously anticipated. Whole-exome sequencing enables the identification of rare variants. It can be applied to both large-scale case-control studies and pedigree-based linkage studies.

There are several large whole-exome sequencing studies in progress. Studying simplex families with one affected child, O’Roak et al. (2011) identified de novo mutations in a number of candidate genes: *FOXP1* (forkhead box P1), *GRIN2B* (glutamate receptor, ionotropic, *N*-methyl-D-aspartate 2B), *SCN1A* (sodium channel, voltage-gated, type I, alpha subunit), and *LAMC3* (laminin, gamma3).

Expression Arrays

This method aims at studying alterations in gene expression in autism using postmortem brain tissues or peripheral blood. Some genes implicated include the *EPB41L3* (erythrocyte membrane protein band 4.1-like 3), which interacts with *CNTNAP2*, and the genes relating glutamatergic neurotransmission (Abrahams and Geschwind 2008). Interestingly, genes identified in independent studies share some common pathways such as ubiquitin conjugation, GTPase regulatory activity, and alternative splicing, making them as potential candidates (Abrahams & Geschwind).

Future Directions

Whole-Genome Sequencing: Exome and Regulome

With the rapid development of sequencing technology, whole-genome sequencing has become cost-effective and feasible. The great advantage is the ability to obtain the sequence of regulatory elements (regulome) as well as protein-coding sequence. Regulatory elements regulate the expression of genes and have been understudied in autism. Therefore, whole-genome sequencing has the potential to identify a whole new set of variants which contribute to autism risk.

See Also

- [Functional Analysis](#)
- [Genome-Wide Association](#)

References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, 9, 341–355.
- Bakkaloglu, B., O’Roak, B. J., Louvi, A., Gupta, A. R., Abelson, J. F., & Morgan, T. M. (2008). Molecular cytogenetic analysis and resequencing of contactin associated protein-like 2 in autism spectrum disorders. *American Journal of Human Genetics*, 82, 165–173.
- Choi, M., Scholl, U. I., Ji, W., Liu, T., Tikhonova, I. R., & Zumbo, P. (2009). Genetic diagnosis by whole exome capture and massively parallel DNA sequencing. *Proceedings of the National Academy of Sciences of the United States of America*, 106, 19096–19101.
- El-Fishawy, P., & State, M. W. (2010). The genetics of autism: Key issues, recent findings, and clinical implications. *The Psychiatric Clinics of North America*, 33, 83–105.
- Glessner, J. T., Wang, K., Cai, G., Korvatska, O., Kim, C. E., & Wood, S. (2009). Autism genome-wide copy number variation reveals ubiquitin and neuronal genes. *Nature*, 459, 569–573.
- Gupta, A. R., & State, M. W. (2007). Recent advances in the genetics of autism. *Biological Psychiatry*, 61, 429–437.
- Lander, E., & Kruglyak, L. (1995). Genetic dissection of complex traits: Guidelines for interpreting and reporting linkage results. *Nature Genetics*, 11, 241–247.
- Morrow, E. M., Yoo, S. Y., Flavell, S. W., Kim, T. K., Lin, Y., & Hill, R. S. (2008). Identifying autism loci and

- genes by tracing recent shared ancestry. *Science*, 321, 218–223.
- O’Roak, B. J., & State, M. W. (2008). Autism genetics: Strategies, challenges, and opportunities. *Autism Research*, 1, 4–17.
- O’Roak, B. J., Deriziotis, P., Lee, C., Vives, L., Schwartz, J. J., & Girirajan, S. (2011). Exome sequencing in sporadic autism spectrum disorders identifies severe de novo mutations. *Nature Genetics*, 43, 585–589.
- Sanders, S. J., Ercan-Sencicek, A. G., Hus, V., Luo, R., Murtha, M. T., & Moreno-De-Luca, D. (2011). Multiple recurrent de novo CNVs, including duplications of the 7q11.23 Williams syndrome region, are strongly associated with autism. *Neuron*, 70, 863–885.
- State, M. W. (2010). The genetics of child psychiatric disorders: Focus on autism and Tourette syndrome. *Neuron*, 68, 254–269.
- State, M. W., & Levitt, P. (2011). The conundrums of understanding genetic risks for autism spectrum disorders. *Nature Neuroscience*, 14, 1499–1506.
- Veenstra-Vanderweele, J., Christian, S. L., & Cook, E. H., Jr. (2004). Autism as a paradigmatic complex genetic disorder. *Annual Review of Genomics and Human Genetics*, 5, 379–405.

- Normative data available across age (4–90 years) and IQ levels
- Test-retest reliability data available on many of the tests

The tests that make up the CANTAB are grouped into some of the following general categories (see <http://www.cantab.com/cantab-tests.asp>):

- Screening
- Executive function, planning, and spatial working memory
- Attention and reaction time
- Visual memory and learning
- Decision making and response control
- Semantic/verbal memory
- Social cognition (emotion recognition)

The CANTAB has been used to measure aspects of executive function in individuals with autism including set shifting, planning, and spatial working memory. A brief description of these tasks is as follows (also see <http://www.cantab.com/cantab-tests.asp> for more details):

- Intradimensional/extradimensional (ID/ED) set-shifting task. Assesses the ability to attend to characteristics of simple and compound stimuli, use feedback to learn a rule, and to shift attention within and across dimensions of a stimulus.
- Stockings of Cambridge (SOC) task. Is a spatial planning task based on the Tower of Hanoi task. The SOC task examines the ability to rearrange colored balls in a lower display to match a goal arrangement in an upper display in the least number of moves possible.
- Spatial working memory (SWM) task. Examines the ability to retain spatial information in working memory and also assesses search strategy.

CANTAB

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Synonyms

Cambridge neuropsychological test automated battery

Description

CANTAB is a computerized battery of cognitive-neuropsychological tests that is marketed by Cambridge Cognition. The CANTAB website is www.cantab.com.

CANTAB is promoted as having some of the following features that can be beneficial for use in research (see <http://www.cantab.com/cantab-for-academic-research.asp>):

- Computer touch-screen administration
- Independent of culture

Historical Background

Information about the CANTAB can be found on the CANTAB website at www.cantab.com. The

CANTAB is currently produced and marketed by Cambridge Cognition. The CANTAB was founded by Dr. Trevor W. Robbins at the University of Cambridge and Dr. Barbara J. Sahakian at the Section of Old Age Psychiatry, Institute of Psychiatry, in the United Kingdom and their colleagues (Robbins and Sahakian 2002). The development of the CANTAB was based from cognitive neuropsychological paradigms in animals in order to examine components of cognitive function in humans (beginning with the elderly) and deficits in patients with dementia (Alzheimer's disease); performance on the CANTAB has been linked to the frontal and temporal lobes of the brain (Robbins et al. 1998).

The CANTAB has been used to examine aspects of cognitive function in over 100 psychiatric and neurologic diseases and disorders including Alzheimer's dementia, anxiety disorders, attention deficit hyperactivity disorder, autism spectrum disorder, Parkinson's disease, and schizophrenia. Please see <http://www.cantab.com/disorders.asp> for a full listing of disorders that have been examined using the CANTAB.

The CANTAB was first used in research studies involving individuals with autism in the mid-1990s. Publications on the CANTAB in individuals with autism can be found in the "References and Reading Section." Results on the CANTAB in autism show that the intradimensional/extradimensional (ID/ED) set-shifting task, the Stockings of Cambridge (SOC) task, and the spatial working memory task from the CANTAB have been useful in detecting impairments in executive functioning in individuals with autism; however, there is some inconsistency in the literature on whether deficits are always found in all of these tasks. In addition, performance on the ID/ED, SOC, and SWM tasks from the CANTAB has been examined in siblings as well as in parents of children with autism. The results in siblings showed while there were no group differences in overall means, a subset of the siblings showed deficits at the ED stage on the ID/ED task and difficulty in passing the higher-level planning problems

on the SOC task (Hughes et al. 1999). Parents of children with autism showed impairment on all three of the CANTAB tasks of executive function (fathers in particular, were more impaired on the SOC planning task, Hughes et al. 1997).

Psychometric Data

Normative data on the CANTAB are available for individuals 4–90 years of age in four IQ ranges. (See CANTAB website www.cantab.com for information about norms; also see DeLuca et al. 2003; Luciana and Nelson 2002; Robbins et al. 1994, 1998).

Test-retest reliability data for CANTAB tasks are also available (Cambridge Cognition 2008; Lowe and Rabbitt 1998). Data on the Standard Error of Prediction (SEP) are also available on CANTAB tasks in order to be able to calculate a confidence interval for determining whether a retest score is due to a real effect or a measurement error (Cambridge Cognition 2008).

Clinical Uses

In autism, the CANTAB has generally been used as a research tool rather than for clinical use.

There is one publication in the literature that has used the CANTAB to examine changes following rehabilitation in autism. The study reported changes in executive function abilities on the Stockings of Cambridge planning task and the Spatial Working Memory task in adults with autism following participation in a vocational rehabilitation program compared to prior to enrolling the program (Garcia-Villamizar and Hughes 2007).

See Also

- [Cambridge Neuropsychological Test Automated Battery](#)

References and Reading

- Berger, H. J. C., Aerts, F. H. T. M., van Spaendonck, K. P. M., Cools, A. R., & Teunisse, J.-P. (2003). Central coherence and cognitive shifting in relation to social improvement in high-functioning young adults with autism. *Journal of Clinical and Experimental Neuropsychology*, 25(4), 502–511.
- Cambridge Cognition. (2008). CANTAB topic: Test-retest reliabilities and detecting reliable change. CANTAB Resources. <http://www.cantab.com/cantab-for-academic-research.asp>, <http://www.cantab.com/cantab-tests.asp>, <http://www.cantab.com/disorders.asp>
- CANTAB. Website: www.cantab.com
- DeLuca, C. R., Wood, S. J., Anderson, V., Buchanan, J., Proffitt, T. M., Mahony, K., & Pantelis, C. (2003). Normative data from the Cantab. I: Development of executive function over the lifespan. *Journal of Clinical and Experimental Neuropsychology*, 242–254.
- Garcia-Villamisar, D., & Hughes, C. (2007). Supported employment improves cognitive performance in adults with Autism. *Journal of Intellectual Disability Research*, 51(2), 142–150.
- Goldberg, M. C., Mostofsky, S. H., Cutting, L. E., Mahone, E. M., Astor, B. C., Denckla, M. B., et al. (2005). Subtle executive impairment in children with autism and children with ADHD. *Journal of Autism and Developmental Disorders*, 35(3), 279–293.
- Happé, F., Booth, R., Charlton, R., & Hughes, C. (2006). Executive function deficits in autism spectrum disorders and attention-deficit/hyperactivity disorder: Examining profiles across domains and ages. *Brain and Cognition*, 61, 25–39.
- Hill, E. (2004a). Evaluating the theory of executive dysfunction in autism. *Developmental Review*, 24, 189–233.
- Hill, E. (2004b). Executive dysfunction in autism. *Trends in Cognitive Sciences*, 8(1), 26–32.
- Hughes, C., & Graham, A. (2002). Measuring executive functions in childhood: Problems and solutions? *Child and Adolescent Mental Health*, 3, 131–142.
- Hughes, C., Russell, J., & Robbins, T. W. (1994). Evidence for executive dysfunction in autism. *Neuropsychologia*, 32(4), 477–492.
- Hughes, C., Leboyer, M., & Bouvard, M. (1997). Executive function in parents of children with autism. *Psychological Medicine*, 27, 209–220.
- Hughes, C., Plumet, M.-H., & Leboyer, M. (1999). Towards a cognitive phenotype for autism: Increased prevalence of executive dysfunction and superior spatial span amongst siblings of children with autism. *Journal of Child Psychology and Psychiatry*, 40(5), 1–14.
- Luciana, M., & Nelson, C. A. (2002). Assessment of neuropsychological function through use of the Cambridge Neuropsychological Testing Automated Battery: Performance in 4- to 12-year-old children. *Developmental Neuropsychology*, 22, 595–624.
- Ozonoff, S., South, M., & Miller, J. N. (2000). DSM-IV-defined Asperger syndrome: Cognitive, behavioral and early history differentiation from high-functioning autism. *Autism*, 4, 29–46.
- Ozonoff, S., Cook, I., Coon, H., Dawson, G., Joseph, R. M., Klin, A., et al. (2004). Performance on Cambridge Neuropsychological Test Automated Battery subtests sensitive to frontal lobe function in people with autistic disorder: Evidence from the collaborative programs of excellence in autism network. *Journal of Autism and Developmental Disorders*, 34(2), 139–150.
- Robbins, T. W., & Sahakian, B. J. (2002). Computer methods of assessment of cognitive function. In J. R. M. Copeland, M. T. Abou-Saleh, & D. G. Blazer (Eds.), *Principles and practice of geriatric psychiatry* (2nd ed.). Chichester: Wiley.
- Robbins, T. W., James, T., Owen, A. M., Sahakian, B. J., McInnes, L., & Rabbitt, P. M. (1994). CANTAB: A factor analytic study of a large sample of normal elderly volunteers. *Dementia*, 5, 266–281.
- Robbins, T. W., James, M., Owen, A. M., Sahakian, B. J., Lawrence, A. D., McInnes, L., & Rabbitt, P. M. A. (1998). A study of performance on tests from the CANTAB battery sensitive to frontal lobe dysfunction in a large number of normal volunteers: implications for theories of executive functioning and cognitive aging. *Journal of the International Neuropsychological Society*, 474–490.
- Sinzig, J., Morsch, D., Bruning, N., Schmidt, M. H., & Lehmkuhl, G. (2008). Inhibition, flexibility, working memory and planning in autism spectrum disorders with and without comorbid ADHD-symptoms. *Child and Adolescent Psychiatry and Mental Health*, 2(1), 1–12.
- Steele, S. D., Minshew, N., Luna, B., & Sweeney, J. A. (2007). Spatial working memory deficits in autism. *Journal of Autism and Developmental Disorders*, 37(4), 605–612.
- Teunisse, J.-P., Cools, A. R., van Spaendonck, K. P. M., Aerts, F. H. T. M., & Berger, H. J. C. (2001). Cognitive styles in high-functioning adolescents with autistic disorder. *Journal of Autism and Developmental Disorders*, 31(1), 55–66.

Capgras Delusion

► Capgras Syndrome

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Capgras Syndrome

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Synonyms

Capgras delusion; Capgras delusion syndrome; Delusion of doubles; Delusion of duplicates; Delusion of negative doubles; Delusion of substitution; Delusional hypoidentification; Illusion des sosies

Short Description or Definition

Capgras syndrome is a particular type of delusional misidentification characterized by the inability of recognizing someone (usually a loved one, a close relative, or friend) as the person they claim to be. In this monothematic delusion, the individual recognizes overtly and straightforwardly who that person is meant to be, upholding however a firm belief to the contrary, which is anchored in subjective cues such as an eerie feeling that something is not quite right about that person, complete lack of a sense of familiarity, and missing the proper affective response. Individuals with Capgras syndrome cling to the unshakeable belief that the original person in question was replaced by an impostor, who cunningly is trying to fool them – with no success at all because, of course, they *know* better. The nature of this alleged impostor, an almost exact double, usually is human, but in some cases, it may turn out to be ghostly, alien, or robot. As a consequence of the puzzling dissonance between “looking familiar” and “feeling familiar,” this is

often referred as a most extraordinary and uncanny experience (Young 2009). There is no emotional connectedness whatsoever to the putative impostor, and so a sense of suspicious estrangement ends up prevailing. Therefore, the overall clinical picture is dominated by an intense paranoid tint. A combination of depersonalization (an alteration in the experience of self in which the individual experiences his/her own body or mental activity as changed in quality to become unreal, detached, or automatized) and derealization (an alteration in which it is the individual’s surroundings that are experienced as remote, lacking immediacy, and oddly unreal) is not unusual in the earlier stages of the syndrome (Munro 2009). Sometimes, individuals with Capgras syndrome become enraged and act on their delusion, attacking the “impostor” with violence. But it may also happen that their relationship with the impostor follows the same pattern of the one with the original person.

Categorization

The eponym “Capgras syndrome” was proposed by the French psychiatrist Joseph Levy-Valensi, in 1929 (Blom 2010). The name refers to Jean Marie Joseph Capgras, who described in 1923, in collaboration with Jean Reboul-Lachaux, the case of a psychotic patient who believed that her husband, her children, other inmates, and hospital staff had been replaced by successive and numerous “doubles” or physically identical impostors (Capgras and Reboul-Lachaux 1923). Capgras and Reboul-Lachaux themselves referred to this phenomenon by the French term *illusion des sosies* (illusion of doubles). The patient they described also believed that she herself had many doubles and, in addition to the “delusion of doubles,” she had persecutory and grandiose delusions. However, a narrower connotation for the Capgras syndrome, referring only to the delusional misidentification, gradually evolved (Rodrigues and Banzato 2006).

Even though the classic form of the Capgras syndrome involves the replacement of persons, there are interesting variations in which pets, objects, or even places (such as one’s own

house) are replaced by copies or duplicates. Thus, the syndrome could be further specified by adding the indication of whom or what has allegedly been replaced; there would be then Capgras syndrome for persons, for animals, for objects, for places, etc. The Capgras syndrome is one of the four main delusional misidentification syndromes described in the psychiatric literature, and unlike the other three (Frégoli syndrome, intermetamorphosis syndrome, and the syndrome of Subjective Doubles) where false and positive identification (hyperidentification) phenomena occur, it is marked by false and negative identification (hypoidentification). The Frégoli syndrome is characterized by a delusional false recognition; in short, the individual identifies familiar persons in strangers. The body may be different, but there is no doubt about the presence of the psychological identity of a familiar person. The latter has changed completely his/her physical appearance or taken over someone else's body, a most radical form of disguise (usually with malevolent intentions). In intermetamorphosis, the individual comes to believe that people around have exchanged their identities, so each person involved in this delusional plot becomes somebody else. In the syndrome of the subjective doubles, the individual is convinced of the existence of exact doubles of him/herself (Munro 2009). It has also been described reverse forms of both Capgras and Frégoli syndromes. In the reverse Capgras, the own self of the individual is taken as a sort of psychological impostor, inhabiting a body that does not belong to him/her. In the reverse Frégoli, the psychological identity of one's own self is preserved alongside with radical changes in his/her physical makeup (Rodrigues and Banzato 2006). Delusional misidentification may be a symptom of several psychiatric (most frequently) and neurologic illnesses, or a separate syndrome on its own right. When misidentification takes place in the context of schizophrenia, severe mood disorder, or dementia, it is regarded as a feature of that illness and it should be referred to as a misidentification phenomenon rather than the syndrome in question. But when a delusion such as the ones aforementioned is the principal and most conspicuous aspect of a

psychosis and other conditions can be ruled out (see differential diagnosis below), it should be assigned as a subcategory within persistent delusional disorder (ICD-10) or delusional disorder (DSM-IV) (Munro 2009). Some classify Capgras phenomenon into either "primary," when associated with psychiatric illnesses, or "secondary," when the phenomenon occurs in the context of a neurologic disorder (Barton 2003).

Epidemiology

Estimates of the prevalence rate of Capgras syndrome vary, depending on the settings and facilities where the investigations are carried out. Currently, the syndrome is claimed to be more common than previously assumed, ranging from 1.3% up to 4% of psychiatric inpatients – with lower frequencies being reported in emergency rooms and in private psychiatric practice. Special populations seem to be particularly at risk to develop Capgras syndrome at some point in the course of their illnesses. Prevalence rates as high as 15–40% in patients with schizophrenia and 2–30% in patients with Alzheimer's disease have been reported. Data regarding sex ratio are conflicting, showing either an even distribution of cases or an increased frequency among women – up to twice the frequency found among men (Tamam et al. 2003; Henriët et al. 2008).

Natural History, Prognostic Factors, and Outcomes

The age of onset, course, and outcomes of Capgras syndrome vary, depending on the underlying neuropsychiatric condition. Among psychiatric patients, Capgras delusion may either be present at the clinical onset of the mental disorder or, more frequently, appear later on, after years of evolution. Remission of this delusion may precede the overall clinical improvement, be simultaneous with it or only be achieved after the abatement of other symptoms. The delusional misidentification may also persist in the long

run. When patients with schizophrenia and mood disorders are compared to each other, the latter are seemingly less prone to have unremitting misidentifications and to hold them for longer than the acute phase of the illness (Christodoulou 1978).

Clinical Expression and Pathophysiology

Several theories have been formulated in order to explain the Capgras syndrome, and among them, we have both the psychological comprehensive (in the sense of taking into account meaningful connections within the individual's life and circumstances) and the cognitive neuropsychiatric ones. As they typically share the view that the core emergent phenomenon in Capgras syndrome is the puzzling dissociation between the proper objective recognition of a given percept and a distorted sense of familiarity towards it, it should be recognized that these theories may not be mutually exclusive. Instead, they can even be seen as complementary to each other in some cases, however, with different emphasis, which is placed either on the psychological dynamic aspects or on the neural underpinnings of the phenomena.

Psychodynamic and Other Psychologically Comprehensive Theories

The fact that patients with Capgras syndrome sometimes report feelings of strangeness in respect to both their surroundings and themselves has fostered the hypothesis that experiences of derealization and depersonalization could play a role in the emergence of the Capgras syndrome and other delusional misidentification syndromes. According to this hypothesis, derealization and depersonalization might be conceived as roots to the distorted feelings of familiarity usually held by patients towards their acquainted ones. Whether this distortion is a direct consequence of derealization and depersonalization, or a response to these symptoms, the delusion of substitution is often thought of as having a somewhat appeasing

effect on the individual by explaining away the rather uncomfortable and perplexing experience of unreality (Christodoulou 1991).

Another comprehensive hypothesis about the genesis of this curious phenomenon revolves around the alleged presence of unacceptable or ambivalent feelings toward a close person. A split on such person's identity would then take place in the patient's mind as a means to circumvent the conflict. For example, someone holding unconscious aversive feelings towards his parents would be allowed, by means of the Capgras delusion, to experience unambiguous love and respect towards them, while, at the same time, directing the otherwise unacceptable feelings of despise, hate, distrust, or fear to the "impostors" (Enoch 1986). Similarly, the syndrome could possibly develop in the context of changing interpersonal relationships, when experiences of strangeness and unconscious negative feelings towards a given person might emerge more easily. The ultimate consequence would be the belief that this close person is not who he or she seems to be but an impostor (Berson 1983).

Additionally, the syndrome has also been thought to result from a pathological regression to archaic models of thinking, arguably common in primeval stages of human evolution, possibly inherited by all of us, when the idea of doubles and the theme of dualities in general were usual (Todd 1957).

Cognitive Neuropsychiatric Theories

In contrast to purely psychological and psychodynamic theories, the emphasis given by cognitive neuropsychiatric approaches to the neural underpinnings of Capgras syndrome, whether well-established or still hypothetical, paves the way for putting forward testable hypothesis, improving thus the empirical anchorage of such theories. As these approaches heavily rely on analogies with other conditions where disrupted face recognition processes definitely or presumably occur, such as prosopagnosia, reduplicative paramnesia, autistic disorder, and Asperger syndrome, heuristic gains should be expected.

Several models of this sort have already been proposed, each one of them positing different hypothetical mechanisms that would lead to these diverse, though correlated, phenomena.

One of these models was first presented by Joseph (1986). According to it, putative cortical interhemispheric disconnections would respond for the distortions on the familiarity feelings experienced by patients towards known persons. Dissociation between cerebral hemispheres would lead to two different and segregated images of the percept – one of them produced by analytic strategies in the left hemisphere and another one produced through more global processing in the right hemisphere. These two images would suffice for the patient to recognize the physical features of known people, but their dissociation would also engender a very strange twofold experience of the percept, suitable to delusional interpretation. Nevertheless, individuals with corpus callosum agenesis or those who suffered section of this commissure for treatment of severe epilepsy do not seem to be particularly prone to develop Capgras syndrome, which weakens Joseph's hypothesis.

A more elaborated and highly regarded hypothesis to explain Capgras syndrome was articulated by Ellis and Young (1990), underpinned by Bauer's (1984) postulation of distinct pathways for overt and covert face recognition. According to Bauer, face recognition would involve two different processes and neuroanatomic pathways. A ventral route connecting the visual associative cortex to temporal lobes (especially amygdala) via inferior longitudinal fasciculus would be critical for overt or conscious face recognition. On the other hand, a dorsal route connecting visual associative cortex to cingulate gyrus and hypothalamus via superior temporal lobe and inferior parietal lobule would function as a kind of covert system for the recognition of faces. The latter would not in fact allow someone to know whose is the face seen in a given moment, nor determining whether it is familiar or not. Instead, the authors argue that this route would be relevant in assigning affective significance to faces. Bauer's proposal follows from the observation that one of his patients with bilateral occipito-temporal damage and suffering from

severe prosopagnosia – the impaired ability to recognize previously known faces and learn new ones – could still show distinctive skin conductance responses when pictures of known faces presented to him were paired with their correct names or wrong ones. Although incapable of telling if those faces were known to him, or even guessing the correct face/name pairing, this patient's autonomic responses were taken as indicative that covert recognition was present and, probably, provided by a mechanism independent from the one responsible for overt recognition. This surprising integrity of autonomic responses to unrecognized known faces in prosopagnosic patients has been confirmed by other authors (Tranel and Damasio 1985).

Based on Bauer's distinction, Capgras syndrome, according to Ellis and Young (1990), could be conceived as a clinical and anatomic-functional mirror image of prosopagnosia. While in prosopagnosia, overt recognition pathways are supposedly disrupted and covert recognition route is claimed to be intact, the inverse would arguably happen in Capgras syndrome. In the latter, adequate appraisal of structural and dynamic facial features, as well as correct evocation of related semantic information, would be guaranteed by ventral route proper functioning. At the same time, dorsal route malfunctioning would prevent the patient to ascribe the expected affective tone to familiar faces. Such a strange mismatch would then stimulate rationalization and support the delusional belief that an impostor has replaced an acquainted person.

Departing from Bauer's two-route model of face recognition, as well as from its use by Ellis and Young (1990) to explain Capgras syndrome, Breen et al. (2000) point out that there is very little evidence that the dorsal visual pathway play any role in visual recognition – either in animals or in humans – and even less in ascribing emotional significance to visual percepts. In contrast, they state that inferotemporal area and amygdala, relevant structures in the ventral visual pathway, are respectively regarded as critical in matching seen faces to stored representations and to the emotional responses these faces might evoke. They propose that malfunctioning of the ventral visual

pathway alone may explain both prosopagnosia and Capgras syndrome. As to Capgras syndrome specifically, their suggested explanation is that the activity of ventral visual recognition structures in the ventral temporal lobe would be normal, but it somehow fails to trigger the activity of ventral limbic structures. This would happen due to either a disconnection between these structures or to impairments within the ventral limbic structures.

Perceptual abnormalities engendered by inadequate visual processing of facial features had been also posited as the fundamental dysfunction in Capgras syndrome. Together with clinical and test-generated evidence that patients with Capgras syndrome often present sub-prosopagnosic face recognition defects, the fact that some of them have been reported to show full-blown prosopagnosia and brain damages that include those seen in prosopagnosic patients has led to the so-called prosopagnosia hypothesis for Capgras syndrome. However, each of these alleged links between the two conditions must be taken cautiously. Accumulated evidence indicates that if there is some sort of relationship between prosopagnosia and Capgras syndrome, it is unlikely to be a straightforward one. It seems that at best, there is a heuristically fruitful analogy, such as the one rendered by the models proposed by Ellis and Young (1990) and by Breen et al. (2000).

Two other hypotheses are worth mentioning, as they depart from the emphasis usually given to experiences of depersonalization/derealization, perceptual dysfunctions, or to a primarily disrupted ability to attach familiarity feelings to known faces. One of them, originated from the observation of delusional misidentifications in schizophrenic patients, postulates that in order to be identified and evoke familiarity feelings, a given percept must be subjected to a process of integration of its various perceived features and stored representations. Such integration would be critical for ascribing percepts with a sense of “uniqueness,” a key element for their identification (Margariti and Kontaxakis 2006). This hypothesis had been explored by Cutting (1991), to whom the loss of that sense of “uniqueness” would be related to right

hemisphere dysfunction (allegedly present in schizophrenic patients) and subsequent failure in perceiving and processing information globally. A second theory has postulated that Capgras syndrome, as well as reduplicative paramnesia, would be possibly related to a failure in updating stored representations of an object, thus leading to a mismatch between its currently seen characteristics and those remembered by the patient (Staton and Brumback 1982).

Finally, it must be stressed that the delusional character of Capgras syndrome cannot be explained by the dysfunctions postulated at the core of any of the enlisted theories alone, and this is sometimes acknowledged by their very proponents. Indeed, derealization, depersonalization, and perceptual abnormalities often occur in the absence of impaired reality testing. Likewise, it is argued that even the puzzling experience of missing the affective overtones expected in the sight of a close person should not be so promptly taken as a sufficient condition for a delusion. Accordingly, the fact that patients with Capgras syndrome fail to conceive less unreasonable explanations to their experiences than the existence of an impostor, as well as to revise their odd beliefs despite evidence in contrary, is sometimes suggested to indicate that altered reasoning and disrupted monitoring of decisional processes play a significant role in the clinical picture. If there is actually an altered experience in the encounter with a close person, then a two-stage model for Capgras syndrome may be needed, one accounting for the odd experience itself and another accounting for the creation and maintenance of a delusional explanation for such experience (Barton 2003; Gainotti 2007; Coltheart et al. 2010).

Structural and Functional Brain Findings

Structural abnormalities in brain CT and MRI scans, as well as EEG and functional brain imaging alterations, have been found not only in cases of neurologic Capgras syndrome but, very often, also in those considered to be primary psychiatric cases. These injuries have been shown to be either

diffuse or localized (often numerous) and not rarely a superposition of both. In most cases, the findings are located in the frontal lobes, sometimes exclusively, but frequently in association with abnormalities affecting other brain areas. Right hemisphere is significantly most often affected, as compared to the left hemisphere. This pattern is consistent with the role postulated for the frontal lobes in the genesis of reasoning and decision-making biases that may give a delusional status to abnormal familiarity feelings.

A variety of conditions has been found to be causes of the structural and functional abnormalities mentioned, including tumors, head trauma, strokes, infections, EEG paroxysmic discharges, and metabolic and neurodegenerative disorders (Barton 2003; Gainotti 2007; Devinsky 2009).

Evaluation and Differential Diagnosis

The assessment of individuals with Capgras syndrome is basically clinical, as it happens with delusions in general. Phenomenal experience, i.e., the way a particular content is subjectively experienced by the individual (in this case, the certainty that a replacement has occurred), is the key domain of the clinical evaluation. Compared to other types of delusions (such as persecutory and mystic or religious), it is easier to have Capgras syndrome's delusional character promptly acknowledged by everyone around, due to the clear impossibility of content (hence its classification as a "bizarre delusion"). It is also important to ascertain how broad and systematic the delusion in question is if it is really a monothematic one or just a small part of an overarching delusion. Furthermore, full assessment of all other areas of psychopathology, including consciousness, attention, memory, perception, thinking, language and speech, mood, and motor activity, is required for the sake of differential and precise diagnosis.

It is relevant to identify if Capgras phenomenon occurs in the context of schizophrenia (or schizophrenia spectrum disorders), in other kinds of delusional disorders (where pure Capgras delusion should be included), or in major mood disorders with delusion (Berson 1983). Moreover,

Capgras phenomenon is often associated with organic brain disorders (about one fourth to one third of the cases), such as brain tumors and infarcts, head trauma, subarachnoid hemorrhage, and basilar migraine, so a complete neurological investigation should be carried out in all cases (Barton 2003). Substance-related disorders must be suspected as well, and the history of substance use needs to be properly checked out; laboratory screening tests for drugs may be run as a supplementary measure. Regardless the final diagnosis reached, a careful and comprehensive assessment must be performed to estimate the actual risk of the individual with Capgras syndrome (or more broadly, Capgras phenomenon) acting on such delusion, as, understandably, the putative impostor constitutes an obvious target for violence.

Treatment

To date, no specific treatment is available to Capgras syndrome. When it is part of the clinical picture of some particular medical condition, interventions aiming at the basic disorder should be the first choice. The delusional character of the syndrome prompts the use of antipsychotics. Psychological approaches are unlikely to make the delusion disappear but may be useful to make patients less concerned, isolated, and dysfunctional because of their pathological beliefs. Good estimates of treatment response in Capgras syndrome are not available. Although it is reasonable to assume that prognosis of delusional misidentification will depend on the underlying medical or psychiatric condition, it must be kept in mind that high response rates may be achieved in the treatment of delusions in general, even when they are part of a delusional disorder (typically regarded as having poor therapeutic response) (Munro 2009).

See Also

- [Face Perception](#)
- [Face Recognition](#)
- [Psychosis](#)

References and Reading

- Barton, J. J. (2003). Disorders of face perception and recognition. *Neurologic Clinics of North America*, 21, 521–548.
- Bauer, R. M. (1984). Autonomic recognition of names and faces: A neuropsychological application of the guilty knowledge test. *Neuropsychologia*, 22, 457–469.
- Berson, R. J. (1983). Capgras' syndrome. *The American Journal of Psychiatry*, 140(8), 969–978.
- Blom, J. D. (2010). Capgras' syndrome. In J. D. Blom (Ed.), *A dictionary of hallucinations* (pp. 84–85). New York: Springer.
- Breen, N., Caine, D., & Coltheart, M. (2000). Models of face recognition and delusional misidentification: A critical review. *Cognitive Neuropsychology*, 17, 55–72.
- Capgras, J., & Reboul-Lachaux, J. (1923). L'illusion des "sosies" dans un délire systématisé. *Bulletin de la Société Clinique de Médecine Mentale*, 11, 6–16.
- Christodoulou, G. N. (1978). Course and prognosis of the syndrome of doubles. *The Journal of Nervous and Mental Disease*, 166(1), 68–72.
- Christodoulou, G. N. (1991). The delusional misidentification syndromes. *The British Journal of Psychiatry*, 159(suppl. 14), 65–69.
- Coltheart, M., Langdon, R., & McKay, R. (2010). Delusional belief. *Annual Review of Psychology*, 62, 271–298.
- Cutting, J. (1991). Delusional misidentification and the role of the right hemisphere in the appreciation of identity. *The British Journal of Psychiatry*, 159(Suppl. 14), 70–75.
- Devinsky, O. (2009). Delusional misidentifications and duplications: Right brain lesions, left brain delusions. *Neurology*, 72(1), 80–87.
- Ellis, H. D., & Young, A. W. (1990). Accounting for delusional misidentifications. *The British Journal of Psychiatry*, 157(2), 239–248.
- Enoch, M. D. (1986). Whose double? The psychopathology of the delusional misidentification syndromes, especially the Capgras syndrome. *Bibliotheca Psychiatrica*, 164, 22–29.
- Gainotti, G. (2007). Face familiarity feelings, the right temporal lobe and the possible underlying neural mechanisms. *Brain Research Reviews*, 56(1), 214–235.
- Henriet, K., Haouzir, S., & Petit, M. (2008). L'illusion des sosies de Capgras: une interprétation délirante d'un trouble spécifique de la reconnaissance affective des visages. Revue de la littérature et proposition d'un modèle séquentiel. *Annales Médico-Psychologiques*, 166(2), 147–156.
- Joseph, A. B. (1986). Focal central nervous system abnormalities in patients with misidentification syndromes. *Bibliotheca Psychiatrica*, 164, 68–79.
- Margariti, M. M., & Kontaxakis, V. P. (2006). Approaching delusional misidentification syndromes as a disorder of the sense of uniqueness. *Psychopathology*, 39, 261–268.
- Munro, A. (2009). Persistent delusional symptoms and disorders. In M. G. Gelder, N. C. Andreasen, J. J. López-Ibor Jr., & J. R. Geddes (Eds.), *New Oxford textbook of psychiatry* (Vol. 1, 2nd ed., pp. 609–628). New York: Oxford University Press.
- Rodrigues, A. C. T., & Banzato, C. E. M. (2006). Delusional misidentification syndrome: Why such nosologic challenge remains intractable. *Psychopathology*, 39, 296–302.
- Staton, R. D., & Brumback, R. A. (1982). Wilson H. Reduplicative paramnesia: A disconnection syndrome of memory. *Cortex*, 18, 23–26.
- Tamam, L., Karatas, G., Zeren, T., & Ozpoyraz, N. (2003). The prevalence of Capgras syndrome in a university hospital setting. *Acta Neuropsychiatrica*, 15(5), 290–295.
- Todd, J. (1957). The syndrome of Capgras. *Psychiatric Quarterly*, 31, 250–265.
- Tranel, D., & Damasio, A. R. (1985). Knowledge without awareness: An autonomic index of facial recognition by prosopagnosics. *Science*, 228, 1453–1454.
- Young, G. (2009). In what sense "familiar"? Examining experiential differences within pathologies of facial recognition. *Consciousness and Cognition*, 18, 628–638.

Capute Scales (Along with Cognitive Adaptive Test)

► Clinical Linguistic and Auditory Milestone Scale

Care Pathway for Children and Adolescents with Autism Spectrum Disorder on an Inpatient Psychiatric Service

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Definition

The Autism Spectrum Disorder Care Pathway (ASD-CP) was designed to address the challenges associated with providing care to youth with ASD psychiatrically hospitalized in nonspecialized

settings. The ASD-CP, first implemented in July 2015 in a public hospital, consists of a modular staff training, a set of behavioral intervention strategies focused on prevention and management of challenging behaviors (CBs), and a toolkit to aid in staff implementation of strategies. Data collected in 3 years since its inception suggests that the ASD-CP is associated with significant reductions in the use of crisis interventions in patients with ASD.

Historical Background

Youth with ASD are psychiatrically hospitalized at elevated rates compared to age-matched peers. These youth also have significantly longer hospital stays, and the costs associated with hospitalizing children with ASD are more than double the costs for children without ASD (Croen et al. 2006). Risk factors for hospitalization in ASD include greater adaptive impairments, higher ASD symptom severity, presence of CB, comorbid psychiatric concerns (specifically mood disorder and obsessive-compulsive disorder), concurrent sleep problems, and being of older age or in a single caregiver home (Mandell 2008; Righi et al. 2017). Despite the high prevalence of individuals with ASD receiving inpatient psychiatric services, with over 10% of caregivers reporting that their child with ASD has been hospitalized at least once (Mandell 2008), and the substantial financial resources expended on hospitalization, standard inpatient care frequently fails to meet the unique needs of the autism population.

In general, psychiatric units are designed for verbal, typically developing individuals with acute mental illness, primarily internalizing disorders, and common treatments, such as process groups and family meetings, rely heavily on verbal and social skills. Therefore, the needs of individuals with ASD admitted to the hospital, most frequently for externalizing behavior (i.e., aggression, property destruction, self-injury; Siegel et al. 2012), are often extremely disparate from the typical patient. The inherent social communication and interaction impairments make commonly

used inpatient interventions inaccessible to individuals with ASD. Inpatient care is also complicated by restricted, repetitive behavior symptoms. While schedule changes and frequent staff transitions are common in general psychiatric units, youth with ASD perform best when following a predictable routine with familiar caregivers. Sensory features of the hospital setting (e.g., harsh lighting; novel, unpredictable noises) may also contribute to increased agitation. When patients with ASD present with comorbid intellectual disability (ID) and/or severe language impairments, difficulties with communication are exacerbated, impeding further on medical and psychiatric assessment and treatment and often on staff ability to understand and meet even the basic needs of their patients. Further, hospital personnel at various levels (e.g., direct care workers, child psychiatrists) often report having limited experience and training in ASD, generating further concern regarding both the appropriateness of the services delivered and patient and staff safety (McGuire and Siegel 2018).

In recent years, several specialized inpatient psychiatric units serving exclusively youth with ASD and/or ID have been developed and have demonstrated effectiveness. Specialized units are distinct from general settings in that they provide staff training focused on improving knowledge about the diagnosis of and treatments for ASD/ID; comprehensive medical, developmental, psychiatric, and behavioral assessment to patients; and a biobehavioral approach to treatment where both applied behavior analysis (ABA) and pharmacological interventions are applied. An extensive multidisciplinary team works collaboratively to address patient symptoms and typically includes a child psychiatrist, psychologist, board-certified behavior analyst, occupational and speech therapists, nurses, social workers, and special educators. Specialized units are also equipped with tools to enhance communication (e.g., visual supports), to protect patients and staff in the case of CB (e.g., personal protective equipment), and to meet the sensory needs of children with ASD (e.g., quiet areas). Importantly, most specialized units provide some sort of continuum of services, including care in residential,

intensive day treatment, partial hospitalization, school, and outpatient settings, addressing barriers related to limited options for appropriate follow-up care post discharge (Siegel et al. 2012; Taylor et al. 2019). Though length of stay is longer in specialized units, patients hospitalized in this setting have statistically significantly reduced symptom severity from admission and are less likely to visit the emergency department (ED) within the first 2 months following discharge than patients with ASD psychiatrically hospitalized on general units (Gabriels et al. 2012; Siegel et al. 2014; Taylor et al. 2019).

Unfortunately, only approximately ten specialized units in the country exist and are predominantly located in the Northeastern United States. Consequently, a majority of children with ASD in need of psychiatric hospitalization are currently served within general settings. While the prevalence of specialized units is increasing (Siegel et al. 2012), creating a separate, specialized psychiatric unit for many hospitals, particularly those with limited resources, may not be feasible. Therefore, efforts to improve ASD services in general psychiatric inpatient settings are essential and should occur in tandem with work to expand availability of specialized units. To address this need, experts convened and provided discussion and recommendations on essential components of ASD services that are feasible for integration within general settings. These recommendations were:

1. Obtain detailed information from caregivers about patient preferences, functioning, and presenting problem.
2. Assess first for a potential medical cause of the presenting concern (e.g., pain, medication side effects).
3. Provide comprehensive psychiatric and developmental evaluation as well as evidence-informed pharmacotherapy linked to the resulting diagnostic picture.
4. Evaluate and accommodate communication, sensory, motor, and adaptive behavior needs.
5. Conduct a functional behavior assessment to identify maintaining variables of CB.
6. Provide developmentally appropriate activities and a sensory-friendly environment.
7. Continue educational demands during hospitalization to facilitate the eventual transition back to school.
8. Conduct ASD-specific direct care staff training (McGuire et al. 2015).

While these experts agreed that it is possible to adequately care for youth with ASD within general inpatient units when appropriate accommodations are in place, limited research exists examining the impact of the accommodations on service provision and patient outcome. Therefore, with these recommendations and in collaboration with clinicians in specialized psychiatric units, a multidisciplinary team at a public hospital in New York developed the ASD-CP, a clinical care model aimed at improving inpatient services for youth with ASD served on a general inpatient psychiatric service.

Current Knowledge

The ASD-CP consists of a four-module staff training and toolkit outlining a variety of intervention strategies. Internal supervising psychologists or nurses provide the training once per quarter to all new staff across disciplines, including but not limited to technicians, nurses, and attendings. Each module of the staff training is 45 min long and uses a variety of teaching strategies, such as didactics, video examples, role-plays, and interactive exercises. The first training module defines ASD and presents integral foundational intervention principles, such as first making sure patients' basic needs are met (e.g., assess for the presence of pain/hunger/thirst). The following three modules present key evidence-informed behavioral treatment components of the ASD-CP using the acronym PATHWAY (i.e., Module 2, Predictability, Activity; Module 3, Total communication, High reward; Module 4, WAY to cope). The modular format of the staff training allows for presentation of material over the course of several days or as a comprehensive one half-day training.

The toolkit, which is provided in the form of a three-ring binder to increase portability, builds on the strategies presented in the training and includes a tip sheet, visual supports for patients, and staff supports. Of note, staff interact with the toolkit during the training to facilitate gains in fluency prior to its use with patients on the unit. The tip sheet is an efficient, one-page assessment filled out at admission by parents or guardians. Primarily using a checkbox format, the tip sheet gathers information about how a child communicates and understands language, the topography and antecedents of CB, and patient preferences in activities, calming strategies, rewards, and foods. The back of the tip sheet is reserved for staff to communicate with other personnel any helpful information gathered about the patient throughout their stay. Visual supports include a visual schedule and first-then card, used to enhance communication between staff and patients as well as improve patient compliance and transitions, and a coping card, used to prompt the patient to engage in a calming activity at early signs of agitation to prevent worsening of CB. The stimuli used for the visual supports are pulled from a sizeable bank of laminated images of activities and items typically present during hospitalization (e.g., meet with doctor, lunch, play with ball). A multidisciplinary team worked together to develop the image bank and first piloted the images on the units before including them in the toolkit. Staff supports include a list of developmentally appropriate leisure activities and a staff schedule. The staff schedule lists the order of leisure and therapeutic activities throughout the day with activities of daily living (e.g., toileting, eating) included. At the top of the staff schedule, staff indicate the patient's safety goal and the reward the patient will receive if their safety goal is met. The safety goal is chosen by the treatment team and typically relates to reducing CB and/or increasing adaptive behavior. Checkboxes are used to indicate whether the patient met the requirement for a reward and are presented alongside the schedule of reinforcement for meeting the safety goal (e.g., absence of hitting for 15 min intervals is rewarded with access to iPad).

Patients eligible for the pathway have ASD, no-to-minimal verbal language, and require 1:1 staffing, as per the clinical judgment of the admitting physician, most often due to high levels of CB and/or low levels of adaptive functioning. These eligibility criteria resulted from discussions regarding patient need and organizational feasibility. At the patient level, difficulties associated with adapting to the nonspecialized inpatient setting were hypothesized to be greatest in patients with limited language, substantial adaptive impairments, and severe CB. These youth typically have higher ASD symptomology and, secondary to severe CB, are also at increased risk for crisis interventions. On the provider level, the ASD-CP involves many components of evidence-informed intervention that would be most feasible to provide in the context of 1:1 care.

The ASD-CP is implemented within the pediatric psychiatric acute care program at a public hospital in New York. The acute care program consists of a Children's Comprehensive Psychiatric Emergency Program (CCPEP) and three child and adolescent psychiatric inpatient units. When youth present to the hospital in psychiatric crisis, they are evaluated in the CCPEP. Based on physician evaluation, these youth may be discharged if they do not require further stabilization, admitted for observation to the CCPEP's six-bed brief-stabilization unit where they may be observed for up to 72 h, or admitted directly to one of the psychiatric inpatient units. When a child arrives to the CCPEP, the evaluating physician determines if the child meets criteria for the ASD-CP, and, if they do, the physician asks the caregiver to complete the one-page tip sheet. If the child is then admitted to the brief-stabilization or inpatient unit, the tip sheet along with the toolkit follows the child. Ideally, when preparing for discharge, caregivers of admitted youth are instructed on ASD-CP strategies, and materials used during the inpatient stay are shared.

In regard to evidence of effectiveness, the initial study compared outcomes of first time admits who received the ASD-CP in the 18 months following initiation of implementation to those who would have met criteria to receive ASD-CP but

were admitted in the 18 months prior to its initiation. Initiation of the ASD-CP was associated with a significant reduction in holds and restraints in both brief-stabilization and inpatient settings. Further, a 40% decrease in total length of stay approached statistical significance in this small, initial study (Kuriakose et al. 2018). A subsequent study was then conducted to examine the sustainability of these results, adding a third comparison group of youth who received the ASD-CP in the 18 months following the initial implementation period. Results from this study demonstrated that reductions in the use of crisis interventions, including holds, restraints, and intramuscular medications, were sustained, while the non-statistically significant trend toward decreased length of stay was no longer present (Cervantes et al. 2019). Taken together, current data suggest that the ASD-CP can be implemented and sustained with limited resources and minimal expertise and is associated with improved patient care.

Future Directions

While the results of significant and continued reductions in crisis interventions are exciting and essential, research into the ASD-CP requires further development. First, these initial studies were small, and the samples were heterogeneous. Therefore, replication across sites and with larger samples is needed to improve confidence in the effects of the ASD-CP. We also do not currently have documented evidence of intervention fidelity. However, the process of data abstraction from medical records across the first two studies exposed inconsistent use of some of the ASD-CP tools, particularly those that required documentation (e.g., staff schedule). Although it is undeterminable how ASD-CP components that do not require documentation (e.g., first-then card, simplifying language) were implemented, the inconsistent use of those that do suggests that the improvements seen may be due to a milieu change, such that changes in staff self-efficacy and understanding are responsible for the reductions in their use of crisis interventions (Cervantes

et al. 2019). A more formal evaluation of how patient care and outcome relates to staff fidelity on ASD-CP intervention components and staff acceptability of ASD-CP strategies is currently underway. Importantly though, because fidelity estimates are captured by record review, we are limited by a lack of formal documentation of use across strategies. Therefore, we are now creating a feasible process for assessing implementation of tools and strategies across each staff shift using a brief fidelity checklist. Improved understanding of intervention acceptability and fidelity, and how these factors might influence patient care and outcome, will allow us to identify key components of the ASD-CP and thus pare down the intervention to increase feasibility. Subsequently, staff supports will be further developed to encourage consistent implementation of essential components. For instance, we will be adding periodic booster training sessions for all retained staff.

Further, as reported, the ASD-CP was designed for a distinct subpopulation of the autism spectrum. However, youth with ASD who do not meet criteria for the ASD-CP also require thoughtful adaptations to treatment as usual in psychiatric inpatient settings. Future research should assess the utility and feasibility of implementation of ASD-CP strategies for youth of varying severity levels and presentations. Of note, presenting concerns may differ between and within groups of youth who do and do not meet criteria for the ASD-CP. For example, it is not uncommon for youth with ASD to present with internalizing symptoms, such as anxiety, post-traumatic stress disorder (PTSD), depression, and/or suicidality (Siegel 2018). These individuals would likely require variations in programming that are distinct from both the ASD-CP and treatment as usual. Additional resources for assessing and addressing the unique needs of these children in non-specialized psychiatric inpatient settings are required.

Finally, researchers have found that psychiatric hospitalization in specialized settings is associated with lower recidivism rates for youth with ASD (Gabriels et al. 2012). It is essential that we also study long-term outcomes for patients who receive the ASD-CP. While it is promising there

were demonstrated improvements in care during their stay, understanding if and how the ASD-CP improves patient utilization trajectories and transitions to less restrictive care environments post discharge is integral and would have significant public health implications given the high costs associated with hospitalization. Readmission rates are often elevated in this population of children, as demonstrated by the proportion of youth excluded from evaluation due to readmission status (~10%) across our studies (Kuriakose et al. 2018). While quality of care during psychiatric hospitalization contributes to this, readmission rates are also largely driven by the considerable lack of appropriate community supports available for patients to transition to after their stay. This systemic issue of limited accessibility of supports increases both the prevalence of psychiatric hospitalization and the economic burden of ASD. Importantly, researchers have found that higher spending on ASD-specific outpatient services and on respite care in particular was associated with significant reductions in the likelihood of psychiatric hospitalization (Mandell et al. 2012, 2019). Therefore, not only are improvements in inpatient care necessary, but it is essential that we continue to work to increase accessibility to evidence-based treatments and family supports to prevent hospitalization and keep youth with ASD integrated in the community.

See Also

- [Emergency Department Utilization and Autism](#)
- [Irritability in Autism](#)
- [Mental Health and ASD](#)
- [Suicidality in Children and Adolescents with Autism](#)

References and Reading

- Cervantes, P., Kuriakose, S., Donnelly, L., Filton, B., Marr, M., Okparaek, E., et al. (2019). Sustainability of a care pathway for children and adolescents with autism spectrum disorder on an inpatient psychiatric service. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-04029-6>.
- Croen, L. A., Najjar, D. V., Ray, G. T., Lotspeich, L., & Bernal, P. (2006). A comparison of health care utilization and costs of children with and without autism spectrum disorders in a large group-model health plan. *Pediatrics*, 118(4), e1203–e1211. <https://doi.org/10.1542/peds.2006-0127>.
- Gabriels, R. L., Agnew, J. A., Beresford, C., Morrow, M. A., Mesibov, G., & Wamboldt, M. (2012). Improving psychiatric hospital care for pediatric patients with autism spectrum disorders and intellectual disabilities. *Autism Research and Treatment*, 2012, 1–7. <https://doi.org/10.1155/2012/685053>.
- Kuriakose, S., Filton, B., Marr, M., Okparaek, E., Cervantes, P., Siegel, M., et al. (2018). Does an autism spectrum disorder care pathway improve care for children and adolescents with ASD in inpatient psychiatric units? *Journal of Autism and Developmental Disorders*, 48(12), 4082–4089. <https://doi.org/10.1007/s10803-018-3666-y>.
- Mandell, D. S. (2008). Psychiatric hospitalization among children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(6), 1059–1065. <https://doi.org/10.1007/s10803-007-0481-2>.
- Mandell, D. S., Xie, M., Morales, K. H., Lawer, L., McCarthy, M., & Marcus, S. C. (2012). The interplay of outpatient services and psychiatric hospitalization among Medicaid-enrolled children with autism spectrum disorders. *Archives of Pediatrics & Adolescent Medicine*, 166(1), 68–73.
- Mandell, D. S., Candon, M. K., Xie, M., Marcus, S. C., Kennedy-Hendricks, A., Epstein, A. J., & Barry, C. L. (2019). Effect of outpatient service utilization on hospitalizations and emergency visits among youths with autism spectrum disorder. *Psychiatric Services*. appi.ps.201800290. <https://doi.org/10.1176/appi.ps.201800290>.
- McGuire, K., & Siegel, M. (2018). Psychiatric hospital treatment of youth with autism spectrum disorder in the United States: Needs, outcomes, and policy. *International Review of Psychiatry*, 30(1), 110–115. <https://doi.org/10.1080/09540261.2018.1433134>.
- McGuire, K., Erickson, C., Gabriels, R. L., Kaplan, D., Mazefsky, C., McGonigle, J., et al. (2015). Psychiatric hospitalization of children with autism or intellectual disability: Consensus statements on best practices. *Journal of the American Academy of Child & Adolescent Psychiatry*, 54(12), 969–971. <https://doi.org/10.1016/j.jaac.2015.08.017>.
- Righi, G., Benevides, J., Mazefsky, C., Siegel, M., Sheinkopf, S. J., & Morrow, E. M. (2017). Predictors of inpatient psychiatric hospitalization for children and adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-017-3154-9>.
- Siegel, M. (2018). The severe end of the spectrum: Insights and opportunities from the autism inpatient collection (AIC). *Journal of Autism and Developmental Disorders*, 48(11), 3641–2646. <https://doi.org/10.1007/s10803-018-3731-6>.

- Siegel, M., Doyle, K., Chemelski, B., Payne, D., Ellsworth, B., Harmon, J., et al. (2012). Specialized inpatient psychiatry units for children with autism and developmental disorders: A United States survey. *Journal of Autism and Developmental Disorders*, 42(9), 1863–1869. <https://doi.org/10.1007/s10803-011-1426-3>.
- Siegel, M., Milligan, B., Chemelski, B., Payne, D., Ellsworth, B., Harmon, J., et al. (2014). Specialized inpatient psychiatry for serious behavioral disturbance in autism and intellectual disability. *Journal of Autism and Developmental Disorders*, 44(12), 3026–3032. <https://doi.org/10.1007/s10803-014-2157-z>.
- Taylor, B. J., Sanders, K. B., Kyle, M., Pedersen, K. A., Veenstra-Vanderweele, J., & Siegel, M. (2019). Inpatient psychiatric treatment of serious behavioral problems in children with autism spectrum disorder (ASD): Specialized versus general inpatient units. *Journal of Autism and Developmental Disorders*, 49(3), 1242–1249. <https://doi.org/10.1007/s10803-018-3816-2>.

Caregiver Consent to a Pediatric Neurodevelopmental Research Registry

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Definition

There has been a historical lag in the development of evidenced-based interventions for youth with neurodevelopmental disorders (NDD), including those with autism spectrum disorder (ASD). One well-known barrier to the development of empirically sound interventions is research recruitment. Problems with study recruitment and retention can result in the delay and/or termination of intervention studies. This methodological problem can also result in the selection of study participants who are not representative of the target population, leading to biased study estimates. Recruitment of youth with NDD can be particularly challenging, when compared to the neurotypical

population, since the caregiver will have to simultaneously manage the child's extensive healthcare needs alongside participating in a research protocol.

One potential solution to improve recruitment to NDD studies is through the use of research registries. There are many types of registries, including national or international disorder-specific registries as well as registries that recruit from a particular clinic or institution (hereafter referred to as clinic registries). Joining a clinic registry, which is governed under an institutional review board, offers parents the opportunity to hear about and potentially engage in local research opportunities. For the investigator, it provides a low-cost option to actively recruit participants, rather than simply relying on passive recruitment methods (e.g., flyers, word of mouth).

There is evidence to suggest that most caregivers raising a child with or at risk of NDD are agreeable to joining a clinic registry, when offered the opportunity during their child's evaluation. This finding is valuable as it speaks to parent's overall interest in joining the research enterprise. However, this conclusion is drawn from the only known study of this topic.

There also appears to be disparate trends across settings in terms of the proportion of families who consent to join the clinic registry. Settings that primarily serve the ASD populations, rather than those serving youth with NDD as a whole, may find increased registry consent rates over time. There are many possible reasons for this finding, including the nature of the setting. If it is a one-time evaluation center, families may be less interested at the prospect of an ongoing research relationship compared to a setting where their child may be receiving care over an extended period of time. There may be something unique to the ASD population as well. There are numerous national organizations and initiatives that have brought science to the general conscious of the ASD community, including the federally funded Autism Act, the Simons Foundation SPARK project, and Autism Speaks. These disparate efforts may have created a culture of scientific collaboration not seen in other populations.

Family sociodemographic factors play an important role as to whether families consent to a clinical registry. For the caregiver, race and socioeconomic status likely influence their decision. Race and ethnicity, in particular, may be related to medical mistrust given the historical research injustices (e.g., Henrietta Lacks) faced by people of color. The nature of the clinical registry, which elicits consent to contact for an unspecified prospective project, may exacerbate feelings of mistrust since caregivers are providing consent to hear about an unknown endeavor. Lastly, families with low income may be less likely to consent since they may not have the resources (e.g., ability to travel) or time to become involved in a research project.

Beyond sociodemographics, it is likely the child's clinical characteristics influence caregiver's interest in research. There is evidence to suggest that caregivers of children with increased mental health issues may be more apt to consent. This may reflect the parents desire to enroll in a study that could assist with such problems or the desire to help other children like theirs. It would follow that increased core developmental issues, such as the presence of increased ASD symptoms, would be positively related to caregiver consent, just like mental health symptoms. However, increased ASD severity has not been associated with an increased likelihood of registry consent. There is research to suggest that these core developmental issues are less stressful to caregivers than the presence of behavior problems. Perhaps the consent findings mimic this body of research on stress.

Much more research is needed on trends and predictors of consent to clinic registries involving youth with NDD. Qualitative approaches that identify reasons for consent, or lack thereof, are particularly needed. This work would shed light on the speculative reasons, put forth above, for why particular populations are more or less likely to consent. Future research should also seek to replicate the existing findings, identify novel predictors of consent, and examine this topic among other pediatric populations.

References and Reading

- Bonevski, B., Randell, M., Paul, C., Chapman, K., Twyman, L., Bryant, J., ... & Hughes, C. (2014). Reaching the hard-to-reach: a systematic review of strategies for improving health and medical research with socially disadvantaged groups. *BMC Medical Research Methodology*, 14(1), 42.
- Cleaver, S., Ouellette-Kuntz, H., & Sakar, A. (2010). Participation in intellectual disability research: A review of 20 years of studies. *Journal of Intellectual Disability Research*, 54(3), 187–193.
- Oliver-Africano, P., Dickens, S., Ahmed, Z., Bouras, N., Cooray, S., Deb, S., ... & Bhaumik, S. (2010). Overcoming the barriers experienced in conducting a medication trial in adults with aggressive challenging behaviour and intellectual disabilities. *Journal of Intellectual Disability Research*, 54(1), 17–25.

Caregiver Training Program

- [Babysitter Training Guide for Families with Individuals with ASD](#)

Carnosine

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Synonyms

beta-Alanyl-L-histidine

Definition

Carnosine is a compound formed from two amino acids (histidine and alanine) and is found in several organ systems including muscle and brain. A number of possible biological roles for this compound have been suggested including antioxidant properties. It has been used experimentally

in several disorders. One small double-blind study in 2002 by Chez and colleagues reported positive initial findings, although the study was criticized on various grounds and the results have not yet been well replicated in the scientific literature.

See Also

- [Neurochemistry](#)

References and Reading

Chez, M. G., Buchanan, C. P., Aimonovitch, M. C., Becker, M., Schaefer, K., Black, C., & Komen, J. (2002). Double-blind, placebo-controlled study of L-carnosine supplementation in children with autistic spectrum disorders. *Journal of Child Neurology*, 17(11), 833–837.

Levy, S. E., & Hyman, S. L. (2005). Novel treatments for autistic spectrum disorders. *Mental Retardation and Developmental Disabilities Research Reviews*, 11(2), 131–142.

CARS

- [Childhood Autism Rating Scale](#)

CARS, Second Edition, High-Functioning Version

- [Childhood Autism Rating Scale](#)

CARS, Second Edition, Questionnaire for Parents or Caregivers

- [Childhood Autism Rating Scale](#)

CARS, Second Edition, Standard Version

- [Childhood Autism Rating Scale](#)

CARS2-HF

- [Childhood Autism Rating Scale](#)

CARS2-QPC

- [Childhood Autism Rating Scale](#)

CARS2-ST

- [Childhood Autism Rating Scale](#)

Case Report

- [Case Study](#)

Case Study

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Synonyms

[Case report](#)

Definition

Case studies are frequent in both biomedical and behavioral psychological research. A typical case study (sometimes referred to as case report) provides a focused report of an individual or series of individuals to illustrate some important issue relevant to clinical work or research. Many of the conditions now recognized as significant causes of developmental disability first appeared as case reports, for example, Down syndrome and childhood autism. Sometimes case reports are used to draw attention to other relevant issues, for example, new approaches to treatment. Case studies from the behavioral literature may be used to illustrate the possible effectiveness of a new intervention, for example, the subject is used as his/her own control with data collected pre-, during, and postintervention. In other fields such as business or law, case studies take other formats.

Case studies may be primarily descriptive or may be more theoretical in nature. Sometimes, as in the case of Down syndrome (trisomy 21), the underlying theory may prove profoundly wrong but the observation is very robust (in the case of Down syndrome, the report from Dr. Down appeared well before there was any awareness of the importance of human chromosomes in development and disease). Case studies can bring attention to new phenomena, can serve as a vehicle for teaching or documenting a potentially important clinical issue, and may, over time, lead to more focused hypothesis-based research. As noted, single-subject research methods provide possibilities for statistically based evaluation within a report focused on a single case.

While case studies have importance in focusing attention on new observations and stimulating hypothesis testing and theory building, they also have some important limitations. Various factors can go into the selection of the case that is reported, and generalization is therefore difficult. There is an obvious tendency on the part of editors and reviewers to support positive association reports (rather than negative ones) in case studies and again generalization can be limited. Cases

may also be reported with these multiple publications of the same case contributing to a perception of greater significance than actually is apparent. As a result, many journals now have limited publication of case reports.

One example in the autism research is provided by the many case reports of autism associated with a host of medical conditions ranging from congenital infections, inborn errors of metabolism, obstetrical risk, and so forth. As noted by Rutter et al. (1994) in the 1970s and early 1980s, there were frequent reports of such associations, but the value of such reports was limited given a lack of relevant controls for issues of diagnosis, duplicate reporting, issues in assessment, and lack of comparison groups. Rutter and colleagues emphasized the importance of controlling for these factors and adopting a more epidemiologically based approach in evaluating reports of comorbid associations of autism with these conditions. When this was done, the strongest associations were with a handful of genetic conditions (fragile X and tuberous sclerosis) and with seizure disorder.

See Also

- [Comorbidity](#)
- [Course of Social Avoidance in Fragile X Syndrome, The](#)
- [Qualitative Versus Quantitative Approaches](#)
- [Seizure Disorder](#)
- [Tuberous Sclerosis Complex](#)

References and Reading

- Bailey, D. B., Jr., Mesibov, G. B., Hatton, D. D., Clark, R. D., Roberts, J. E., & Mayhew, L. (1998). Autistic behavior in young boys with fragile X syndrome. *Journal of Autism and Developmental Disorders*, 28(6), 499–508.
- Down, J. L. H. (1866). Observations on an ethnic classification of idiots. *Clinical Lecture Reports of London Hospital*, 3, 259–262.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Rutter, M., Bailey, A., Bolton, P., & Le Couteur, A. (1994). Autism and known medical conditions: Myth and

substance. *Journal of Child Psychology and Psychiatry*, 35(2), 311–322.

Volkmar, F. R., & Nelson, D. S. (1990). Seizure disorders in autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 29(1), 127–129.

Wiznitzer, M. (2004). Autism and tuberous sclerosis. *Journal of Child Neurology*, 19(9), 675–679.

Casein

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Synonyms

Milk protein

Definition

Casein is a milk protein. One type of casein found in human and cow milk, beta-casein, is digested into beta-casomorphins (BCMs). Sun et al. (1999) demonstrated that BCMs affect many regions in the rat brain (i.e., nucleus accumbens, caudate, putamen, ventral tegmental and median raphe nucleus, and orbitofrontal, prefrontal, parietal, temporal, occipital, and entorhinal cortices). These effects are partially blocked by opiate receptor antagonists, indicating that BCMs act like opioids in the mammalian nervous system. Additionally, infusion of BCM has been shown to cause behavioral changes in rats including restlessness followed by inactivity, reduced response to sound, and reduced social interaction (Sun and Cade 1999). These results are used to support to the opioid-excess theory (Panksepp 1979) to explain the symptoms of autism. According to this theory, BCMs become excessive because of an enzyme deficiency (Trygstad et al. 1980; Reichelt et al. 1981) or a leaky gut (Wakefield et al. 1998), and the opioid effects in the human nervous system contribute to the symptoms of autism. However, well-designed studies have not found

abnormal opioid concentrations (Cass et al. 2008) or evidence for GI abnormalities in individuals with autism (Buie et al. 2010; Fernell et al. 2007; Sandhu et al. 2009).

Despite a lack of support for either the enzyme deficiency or leaky gut theory, the opioid-excess theory has led to a focus on eliminating casein, and often gluten, from the diets of children with autism. Most studies have examined a combined gluten-free, casein-free diet. Therefore, it is difficult to assess the effect of eliminating casein alone. However, one study (Lucarelli et al. 1995) did find improvement on five out of seven behavioral scales in children adhering to an only casein-free diet compared to a control group with no dietary restrictions. Worsening on two out of seven of the scales was also observed after a casein challenge. However, the study design had many limitations. Notably, a small sample was studied, and it is unclear if the behavior evaluators were blinded to the diet status of the participants. In addition, no other studies have eliminated only casein. More research and replications are needed before casein-free diets can be considered efficacious. Gluten-free, casein-free diets also lack scientific support. In a 2008 review, Millward, Ferriter, Calver, and Connell-Jones reported mixed results for gluten-free, casein-free diets, with most studies having major methodological limitations and the better designed studies reporting mostly negative findings. Without adequate data, elimination diets are currently not recommended (Buie et al. 2010).

See Also

- [Antigluten Therapy](#)
- [Gluten-Free Diet](#)
- [Nutritional Interventions](#)

References and Reading

Buie, T., Campbell, D. B., Fuchs III, G. J., Furuta, G. T., Levy, J., Van de Water, J., et al. (2010). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125, S1–S18.

- Cass, H., Gringras, P., March, J., McKendrick, I., O'Hare, A. E., Owen, L., et al. (2008). Absence of urinary opioid peptides in children with autism. *Archives of Disease in Childhood*, 93, 745–749.
- Fernell, E., Fagerberg, U. L., & Hellstrom, P. M. (2007). No evidence for a clear link between active intestinal inflammation and autism based on analyses of faecal calprotectin and rectal nitric oxide. *Acta Paediatrica*, 96, 1076–1079.
- Lucarelli, S., Frediani, T., Zingoni, A. M., Ferruzzi, F., Giardini, O., Quinteri, F., et al. (1995). Food allergy and infantile autism. *Panminerva Medica*, 37, 137–141.
- Millward, C., Ferriter, M., Calver, S. J., & Connell-Jones, G. G. (2009). Gluten- and casein-free diets for autistic spectrum disorder. *Cochrane Database of Systematic Reviews*, 2, 1–28.
- Panksepp, J. (1979). A neurochemical theory of autism. *Trends in Neurosciences*, 2, 174–177.
- Reichelt, K. L., Hole, K., Hamberger, A., Saelid, G., Edminson, P. D., Braestrup, C. B., et al. (1981). Biologically active peptide-containing fractions in schizophrenia and childhood autism. *Advances in Biochemical Psychopharmacology*, 28, 627–643.
- Sandhu, B., Steer, C., Golding, J., & Emond, A. (2009). The early stool patterns of young children with autistic spectrum disorder. *Archives of Disease in Childhood*, 94, 497–500.
- Sun, Z., & Cade, J. R. (1999). A peptide found in schizophrenia and autism causes behavioural changes in rats. *Autism*, 3, 85–95.
- Sun, Z., Cade, J. R., Fregly, M. J., & Privette, R. M. (1999). Beta-casomorphin induces Fos-like immunoreactivity in discrete brain regions relevant to schizophrenia and autism. *Autism*, 3, 67–83.
- Trygstad, O. E., Reichelt, K. L., Foss, I., Edminson, P. D., Selid, G., Bremer, J., et al. (1980). Patterns of peptides and protein-associated-peptide complexes in psychiatric disorders. *British Journal of Psychiatry*, 136, 59–72.
- Wakefield, A. J., Murch, S. H., Anthony, A., Linnell, J., Casson, D. M., Malik, M., et al. (1998). Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *The Lancet*, 351, 637–641.

CASL

► [Comprehensive Assessment of Spoken Language](#)

CAT/CLAMS

► [Clinical Linguistic and Auditory Milestone Scale](#)

Catapres

► [Clonidine](#)

Catatonia

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Catatonia, Shutdown and Breakdown in Autism

Catatonia is a complex neuropsychological disorder which can affect individuals with autism spectrum disorder (ASD), including those with high-functioning autism and Asperger syndrome. Most clinicians think about catatonia as a historical and outdated psychiatric disorder and find it difficult to recognize the ways catatonia can manifest in autistic individuals. In autistic individuals, the most severe acute and classic form of catatonia as described in the psychiatric literature can occur but it is rare (Billstedt et al. 2005; Wing and Shah 2000). Manifestations of catatonia in autistic individuals are usually gradual, chronic, and can occur together with shutdown, overall deterioration of functioning, and breakdown. The terms “autism-related catatonia” and “autistic catatonia” are useful to enable clinicians to conceptualise catatonia differently in autistic individuals differently in autistic individuals and distinguish it from general catatonia in the psychiatric literature.

Types of Catatonia Manifestations in Autism

1. Chronic catatonia and catatonia-type deterioration and breakdown
2. Catatonia with shutdown
3. Episodic catatonia type difficulties
4. Acute classic catatonia (rare)

Detailed descriptions of the varied manifestations and case examples are provided in Shah (2019).

The possibility of catatonia should be considered in any autistic individual who shows a marked and obvious deterioration in voluntary movement, speech, level of activity, and independence compared to previous levels (Shah 2019; Wing and Shah 2000).

Possible Early Signs

- “Freezing” or marked hesitations during movement or action
- Slowing down in movements, actions, and/or speech
- Tendency to “shutdown” in overwhelming situations
- Increased reliance on verbal or physical prompts

Common Manifestations and Symptoms

- Increased slowness affecting movements and speech
- Marked reduction in the amount of speech or selective/complete mutism
- Difficulty in initiating and inhibiting actions
- Increased reliance on physical or verbal prompts
- Increased passivity and increased social withdrawal
- Periods of “shutdown”
- Physical or mental freezing episodes lasting minutes or hours

Other Manifestations and Associated Behaviors

These can include difficulty crossing thresholds and transitions, to and fro movements and hesitations, odd stiff posture, parkinsonian features, and episodes of excitement and agitation. There can also be a marked increase in repetitive and ritualistic behaviors.

Secondary Difficulties and Consequences

These include the following:

- Social and communication withdrawal
- Inability to cope with everyday demands and life
- Inability to attend school, college, work, etc.
- Challenging behavior and “meltdowns” due to the frustration of not understanding what is happening to them – this can spiral down to autistic breakdown
- Mobility issues and muscle wastage
- Secondary medical and physical problems

Summary of Key Aspects of Autism-Related Catatonia

(based on research studies and clinical experience)

- Catatonia can affect autistic children (as young as 6 years) and adults of any age.
- The current prevalence rates of catatonia in ASD based on systematic prevalence studies range between 17% and 20% (Billstedt et al. 2005; Breen and Hare 2017; Wing and Shah 2000).
- Catatonia can affect individuals across the whole spectrum including those with intellectual impairment as well as those who are intellectually high-functioning. There are research projects in the pipeline exploring catatonia-associated phenomena such as “inertia” and initiation difficulties which are experienced by many high-functioning autistic individuals.
- Autistic individuals with a “passive” type of social impairment are more likely to have a breakdown in the form of catatonia (Wing and Shah 2000).
- There can be huge variation in symptoms and manifestations in the same individual at different times and in different situations. Some high-functioning autistic individuals show “episodic” catatonia difficulties causing temporary “shutdown” or “freezing” and/or mutism.

- In many individuals, catatonia can occur together with a more general breakdown which can be referred to as “autistic breakdown.”
- At this point in time, we do not have a complete picture of the types and manifestations of catatonia and shutdown in autism. New subtypes are coming to light by clinicians as awareness is increasing.
- Detailed psychological assessment of the person’s underlying autism and possible stress factors
- Eliminating possible culprits such as antipsychotic medication
- Designing a person-centered multidimensional plan of management which reduces the stress and motivates the individual. This includes looking at the individual’s program, environment, occupation, lifestyle, activities, and making changes as needed

Assessment

The general catatonia rating scales are not appropriate for screening or assessing the manifestations of autism-related catatonia. The author recommends a dimensional assessment to gather information from various sources to obtain an individual profile of catatonia-related manifestations and the secondary difficulties. The Autism Catatonia Evaluation (ACE-S) (Shah 2019) has been developed to guide clinicians and researchers.

Interventions and Management

Psycho-Ecological Approach

This is not a specific intervention but a multidimensional approach which has evolved through clinical experience and has been found to be extremely useful and beneficial for individuals and their families at various levels (Shah 2019; Shah and Wing 2006). The aim of this approach is to understand and formulate the individual’s catatonia in the context of their underlying autism, vulnerability and sensitivity, identification of possible causes and stresses, and developing a multidimensional treatment and management plan.

The main components of this approach are listed below. A detailed description and application of the psycho-ecological approach with case examples can be referred to in Shah (2019).

The main components of this approach include the following:

- Timely diagnosis of catatonia-like deterioration at an early stage
- Psycho-education and training
- Providing 1:1 support with specific strategies
- Providing external stimulation and increasing participation with support
- Management of specific problems and secondary consequences
- Psychological interventions and support for high-functioning autistic individuals, for example, adapted Cognitive Behaviour Therapy (CBT), mindfulness, anxiety management training.

Further general and specific psychological intervention and management is described in DeJong et al. (2014); Dhossche et al. (2006b); Hare and Malone (2004); Shah and Wing (2006); and Shah (2019).

Caution About Medical Interventions

There is a very limited literature based on individual case reports and small case series describing effects of medical treatments of catatonia in autistic individuals. These report a range of psychiatric medications, doses, and effects. Most of these reports concern severe, acute manifestation of catatonia in autistic individuals. These have been documented and reviewed in the paper by DeJong et al. (2014). As pointed out by the authors, the outcomes should be interpreted cautiously for various reasons. In general, there is huge individual variation in response to medical treatments used in treating catatonia in autistic individuals. Clinicians warn against the use of antipsychotic medication and generally recommend the use of

benzodiazepines (Dhossche et al. 2006a; Mazzone et al. 2014).

Clinicians who use medication to treat catatonia symptoms in autistic individuals should do with extreme caution and be mindful of the possibility of the side effects of the medication which can trigger catatonia symptoms or make them worse. Medication which is carefully tailored and monitored may be useful as an emergency treatment for acute, severe catatonia or as a short-term treatment trial in selected cases. Before, during, and after medical treatment, it is important to continue using the psycho-ecological approach and strategies for supporting the individual and their families/carers.

See Also

- [Mindfulness Therapy for Individuals with Autism Spectrum Disorder](#)
- [Sensory Impairment in Autism](#)

References and Reading

- Billstedt, E., Gillberg, C., & Gillberg, C. (2005). Autism after adolescence: Population-based 13- to 22-year follow-up study of 120 individuals with autism diagnosed in childhood. *Journal of Autism and Developmental Disorders*, 35(3), 351–360.
- Breen, J., & Hare, D. (2017). The nature and prevalence of catatonic symptoms in young people with autism. *Journal of Intellectual Disability Research*, 61(6), 580.
- DeJong, H., Bunton, P., & Hare, D. J. (2014). A systematic review of interventions used to treat catatonic symptoms in people with autistic spectrum disorders. *Journal of Autism and Developmental Disorders* September, 44(9), 2127–2136.
- Dhossche, D., Wing, L., Ohta, M., & Neumarker, K. (2006a). *Catatonia in autism spectrum disorders*. San Diego: Elsevier.
- Dhossche, D., Wing, L., & Shah, A. (2006b). Blueprints for the assessment, treatment and future study of catatonia in autism Spectrum disorders. *International Review of Neurobiology*, 72, 267–284.
- Hare, D. J., & Malone, C. (2004). Catatonia and autistic spectrum disorders. *Autism*, 8(2), 183–195.
- Mazzone, L., Postorino, V., Valeri, G., & Vicari, S. (2014). Catatonia in patients with autism: Prevalence and management. *CNS Drugs*, 28(3), 205–215.

Shah, A. (2019). *Catatonia breakdown and shutdown in autism. A psycho-ecological approach*. London: Jessica Kingsley Publishers.

Shah, A., & Wing, L. (2006). Psychological approaches to chronic catatonia-like deterioration in autism Spectrum disorders. *International Review of Neurobiology*, 72, 246–263.

Wing, L., & Shah, A. (2000). Catatonia in autistic spectrum disorders. *British Journal of Psychiatry*, 176, 357–362.

CATCH 22 (Chromosome 22q11 Deletion Syndrome)

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Synonyms

[DiGeorge syndrome](#); [Takao syndrome](#); [Velocardiofacial syndrome](#)

Short Description or Definition

22q11.2 deletion syndrome (22q11.2DS) is diagnosed in individuals with a submicroscopic deletion of chromosome; 22. 22q11.2DS encompasses phenotypes previously described as CATCH22, which is an acronym for cardiac defect, abnormal facies, T-cell deficit, cleft palate, and hypocalcemia due to chromosome 22q11 deletion. These are variable features associated with several clinically defined syndromes, including DiGeorge, velocardiofacial, and Takao, that are now recognized to be the same condition with a common cause.

Categorization

The acronym CATCH22 was suggested in the 1990s to encompass the variable features that accompany 22q11 deletion (Wilson et al. 1993).

Clinical diagnoses associated with 22q11 deletion include DiGeorge syndrome, Shprintzen (velocardiofacial) syndrome, and Takao (conotruncal anomaly face) syndrome. These phenotypes were recognized independently due to the prominence of particular clinical features. Identification of a common 22q11 deletion among patients with any of these diagnoses provided the clear unifying factor for these clinically defined syndromes.

An absent thymus and hypocalcemia due to a small parathyroid were the first recognized features of DiGeorge syndrome, establishing the diagnosis in the 1960s (Kirkpatrick and DiGeorge 1968). Additional characteristics including facial features and heart defects were noted as reports of the syndrome accumulated. DiGeorge syndrome is now recognized by a pattern of structural or functional deficits of the thymus, reduced parathyroid function, decreased serum calcium, and congenital heart defects.

Shprintzen, or velocardiofacial syndrome (VCFS), includes palate abnormalities, a characteristic facial appearance, and, in some cases, heart disease (Shprintzen et al. 1978, 1981). Additional features of VCFS include learning disabilities, developmental delay, and a wide array of psychiatric disorders (Motzkin et al. 1993).

Takao, or conotruncal anomaly face syndrome, is identical to DiGeorge syndrome, but the Japanese group was the first to recognize the major contribution of outflow tract defects of the heart (Takao et al. 1980).

In 1981, de la Chapelle and colleagues reported that an unbalanced translocation between chromosome 22 and another chromosome was associated with features of DiGeorge syndrome. The small deletion created by the chromosome rearrangement led to the hypothesis that genes in this region of chromosome 22 were responsible for DiGeorge syndrome (Augusseau et al. 1986). Further prospective analysis of patients with DiGeorge syndrome confirmed the importance of 22q11 deletion in this population, though additional chromosomal abnormalities were found in a few cases (Greenberg et al. 1988). Targeted chromosome studies in patients with VCFS (Driscoll et al. 1992, 1993; Kelly et al. 1993; Scambler et al.

1992) or Takao syndrome (Burn et al. 1993) revealed a similar proportion of 22q11 deletion in these phenotypes as well. A 1.5- or 3-Mb piece of 22q11 is typically lost (Cohen et al. 1999; Jerome and Papaioannou 2001).

Burns suggested using DiGeorge syndrome for the severe presentation at birth, VCFS for children with a prominent craniofacial presentation, and Takao syndrome when cardiovascular features are prominent, with the CATCH phenotype encompassing all of the three diagnoses (Burn 1999). It is now recognized that these clinically defined phenotypes are variable features of the same condition associated with 22q11.2DS (Kobrynski and Sullivan 2007).

Epidemiology

22q11.2DS is the most common microdeletion syndrome. It occurs in about 1 in 2,000 to 1 in 6,000 children and accounts for 2% of all heart defects (Liling et al. 1999; Botto et al. 2003). This is the second most frequent cause of congenital heart disease after Down syndrome. In the United States, as many as 700 infants may be affected annually, with a slightly higher prevalence in Hispanics (Botto et al. 2003). Given the variable expression of 22q11.2DS, the incidence is likely to be higher than estimated.

More than 90% of patients have a de novo deletion of 22q11.2 (McDonald-McGinn et al. 2015).

Natural History, Prognostic Factors, and Outcomes

22q11.2DS is associated with premature mortality, with death occurring within the first year for 4% of all affected infants. Though death is often associated with congenital heart disease, the overall mortality for individuals with 22q11.2DS surpasses that for individuals with non-syndromic forms of similar heart defects (Repetto et al. 2014). Recurrent infections occur across the lifespan. Speech difficulties can abate with therapy and surgery to correct palate abnormalities.

Clinical Expression and Pathophysiology

Congenital heart disease occurs in 75% of individuals (McDonald-McGinn et al. 2015). Associated cardiac malformations typically affect the outflow tract. These include tetralogy of Fallot, type B interrupted aortic arch, truncus arteriosus, right aortic arch, and aberrant right subclavian artery.

Palatal abnormalities occur in 75% of individuals. These include velopharyngeal incompetence, submucosal cleft palate, bifid uvula, and cleft palate. Only 11% of patients with 22q11.2DS have cleft palate, while 65% have more mild palatal abnormalities (McDonald-McGinn et al. 2015).

Immunodeficiency occurs in 75% of individuals. This includes abnormal T-cell production, chronic infection, impaired antibody production impacting vaccine response, allergy and asthma, and other autoimmune disorders (McDonald-McGinn et al. 2015). It can be secondary to absent or impaired thymus development. In infancy, most patients have T-cell counts below age-appropriate levels, which often improves within the first year.

Hypocalcaemia due to hypoparathyroidism occurs in 50% of individuals. In affected infants, hypocalcaemia frequently resolves within the first year, though it can occur at any age, including adulthood (Wilson et al. 1993; McDonald-McGinn et al. 2015).

Facial features include a small mouth, square nose tip with pinched nostrils, unusual earlobe folding, short upper lip folds, and slanting eyes (Wilson et al. 1993).

Gastrointestinal abnormalities are found in 30% of individuals, which can result in feeding and swallowing difficulties (McDonald-McGinn et al. 2015).

Mild to moderate learning difficulties and speech and language delay are common. Autism spectrum disorder is found in 20% of children. Various psychiatric disorders, including paranoid schizophrenia and major depressive illness, have also been described in 25% of adult cases (Motzkin et al. 1993; McDonald-McGinn et al. 2015).

Hearing loss, cleft lip, kidney abnormalities, and low-functioning thyroid can also occur, though these features are less common (Wilson et al. 1993).

Evaluation and Differential Diagnosis

Distinctive facial features together with a heart defect affecting the major outflow tract defect or a history of recurrent infection should raise suspicion. When these features are not present, diagnosis can be missed.

A chest X-ray is necessary for immunological assessment. However, identifying a small thymus by radiography can be challenging in stressed infants. Children with 22q11.2DS may have normal white blood cell counts, while sick infants may instead have a normal thymus and reduced white blood cell counts. To resolve these differences, assess the number of CD4-positive T lymphocytes. Sick infants should be treated as if they have compromised cellular immunity, with transfusion using irradiated blood to avoid graft-versus-host disease until diagnosis is confirmed (Wilson et al. 1993).

Suspicion of 22q11 deletion syndrome should be confirmed using a molecular genetics test. Routine cytogenetic studies can exclude major chromosomal rearrangements, while fluorescent in situ hybridization, multiplex ligation-dependent probe amplification, or copy number variation analyses can more precisely determine deletion size and location. Parents should be screened for carrier status; 10–25% of parents may be asymptomatic carriers (Levy et al. 1997).

Treatment

Clinical management is complex due to the array of phenotypes associated with 22q11.2DS. Heart defects are usually the focus of treatment, though this treatment does not differ from that for other similar heart defects. Early echocardiography is critical in any child with suspected 22q11.2DS.

Hypocalcemia can be treated using calcium supplements and 1,25-cholecalciferol.

The child should be examined for the presence of a submucous cleft, which can elude detection and often requires surgical intervention.

Immunological features manifest as frequent respiratory infections in early childhood with few occurrences of severe immunodeficiency. Early thymus transplantation has been performed to alleviate immunological features, though these features may resolve on their own over time (Markert et al. 1999).

Early diagnosis and intervention for psychiatric illnesses can improve long-term prognosis. Standard treatments for attention deficit, anxiety, and schizophrenia are effective.

See Also

► Velocardiofacial Syndrome

References and Reading

- Augusseau, S., Jouk, S., Jalbert, P., & Prieur, M. (1986). DiGeorge syndrome and 22q11 rearrangements. *Human Genetics*, 74, 206.
- Botto, L. D., May, K., Fernhoff, P. M., Correa, A., Coleman, K., Rasmussen, S. A., Merritt, R. K., O'Leary, L. A., Wong, L. Y., Elixson, E. M., Mahle, W. T., & Campbell, R. M. (2003). A population-based study of the 22q11.2 deletion: Phenotype, incidence, and contribution to major birth defects in the population. *Pediatrics*, 112, 101–107.
- Burn, J. (1999). Closing time for CATCH22. *Journal of Medical Genetics*, 36, 737–738.
- Burn, J., Takao, A., Wilson, D., Cross, I., Momma, K., Wadey, R., Scambler, P., & Goodship, J. (1993). Conotruncal anomaly face syndrome is associated with a deletion within chromosome 22q11. *Journal of Medical Genetics*, 30(10), 822–824.
- Cohen, E., Chow, E. W., Weksberg, R., & Bassett, A. S. (1999). Phenotype of adults with the 22q11 deletion syndrome: A review. *American Journal of Medical Genetics*, 86, 359–365.
- De la Chapelle, A., Herva, R., Koivisto, M., & Aula, P. (1981). A deletion in chromosome 22 can cause DiGeorge syndrome. *Human Genetics*, 57, 253–256.
- Driscoll, D. A., Spinner, N. B., Budarf, M. L., McDonald-McGinn, D. M., Zackai, E. H., Goldberg, R. B., Shprintzen, R. J., Saal, H. M., Zonana, J., Jones, M. C., Mascarello, J. T., & Emanuel, B. S. (1992). Deletions and microdeletions of 22q11.2 in velo-cardio-facial syndrome. *American Journal of Medical Genetics*, 44(2), 261–268.
- Driscoll, D. A., Salvin, J., Sellinger, B., Budarf, M. L., McDonald-McGinn, D. M., Zackai, E. H., & Emanuel, B. S. (1993). Prevalence of 22q11 microdeletions in DiGeorge and velocardiofacial syndromes: Implications for genetic counseling and prenatal diagnosis. *Journal of Medical Genetics*, 30(10), 813–817.
- Greenberg, F., Elder, F. F., Haffner, P., Northrup, H., & Ledbetter, D. H. (1988). Cytogenetic findings in a prospective series of patients with DiGeorge anomaly. *American Journal of Human Genetics*, 43(5), 606–611.
- Jerome, L. A., & Papaioannou, V. E. (2001). DiGeorge syndrome phenotype in mice mutant for the T-box gene, Tbx1. *Nature Genetics*, 27, 286–291.
- Kelly, D., Goldberg, R., Wilson, D., Lindsay, E., Carey, A., Goodship, J., Burn, J., Cross, I., Shprintzen, R. J., & Scambler, P. J. (1993). Conformation that the velo-cardio-facial syndrome is associated with haploinsufficiency of genes at chromosome 22q11. *American Journal of Medical Genetics*, 45(3), 308–312.
- Kirkpatrick, J. A., Jr., & DiGeorge, A. M. (1968). Congenital absence of the thymus. *The American Journal of Roentgenology, Radium Therapy, and Nuclear Medicine*, 103, 32–37.
- Kobrynski, L. J., & Sullivan, K. E. (2007). Velocardiofacial syndrome, DiGeorge syndrome: The chromosome 22q11.2 deletion syndromes. *Lancet*, 370(9596), 1443–1452.
- Levy, A., Michel, G., Lemerer, M., & Philip, N. (1997). Idiopathic thrombocytopenic purpura in two mothers of children with DiGeorge sequence: A new component manifestation of deletion 22q11? *American Journal of Medical Genetics*, 69, 356–359.
- Liling, J., Cross, I., Burn, J., Daniel, C. P., Tawn, E. J., & Parker, L. (1999). Frequency and predictive value of 22q11 deletion. *Journal of Medical Genetics*, 36(10), 794–795.
- Markert, M. L., Boeck, A., Hale, L. P., Kloster, A. L., McLaughlin, T. M., Batchvarova, M. N., Doued, D. C., Koup, R. A., Kostyu, D. D., Ward, F. E., Rice, H. E., & Mahaffey, S. M. (1999). Transplantation of thymus tissue in complete DiGeorge syndrome. *New England Journal of Medicine*, 341, 1180–1189.
- McDonald-McGinn, D. M., Sullivan, K. E., Marino, B., Philip, N., Swillen, A., Vortsman, J. A. S., Zackai, E. H., Emanuel, B. S., Vermeesch, J. R., Morrow, B. E., Scambler, P. J., & Bassett, A. S. (2015). 22q11.2 deletion syndrome. *Nature Reviews Disease Primers*, 1, 15071.
- Motzkkin, B., Marion, R., Goldberg, R., Shprintzen, R., & Saenger, P. (1993). Variable phenotypes in velocardiofacial syndrome with chromosomal deletion. *Journal of Pediatrics*, 123(3), 406–410.
- Repetto, G. M., Guzman, M. L., Delgado, I., Loyola, H., Palomares, M., Lay-Son, G., Vial, C., Benavides, F., Espinoza, K., & Alvarez, P. (2014). Case fatality rate and associated factors in patients with 22q11 microdeletion syndrome: A retrospective cohort study. *British Medical Journal Open*, 4(11), 1–5.
- Scambler, P., Kelly, D., Lindsay, E., Williamson, R., Goldberg, R., Shprintzen, R., Wilson, D. I.,

- Goodship, J. A., Cross, I. E., & Burn, J. (1992). Velo-cardio-facial syndrome associated with chromosome 22 deletions encompassing the DiGeorge locus. *Lancet*, 339(8802), 1138–1139.
- Shprintzen, R. J. (2008). Velo-cardio-facial syndrome: 30 years of study. *Developmental Disability Research Reviews*, 14(1), 3–10.
- Shprintzen, R. J., Goldberg, R. B., Lewin, M. L., Sidoti, E. J., Berkman, M. D., Argamaso, R. V., & Young, D. (1978). A new syndrome involving cleft palate, cardiac anomalies, typical facies, and learning disabilities: Velo-cardio-facial syndrome. *The Cleft Palate Journal*, 15(1), 56–62.
- Shprintzen, R. J., Goldberg, R. B., Young, D., & Wolford, L. (1981). The velo-cardio-facial syndrome: A clinical and genetic analysis. *Pediatrics*, 67, 167–172.
- Takao, A., Ando, M., Cho, K., Kinouchi, A., & Murakami, Y. (1980). Etiologic categorization of common congenital heart disease. In R. Van Praagh & A. Takao (Eds.), *Etiology and morphogenesis of congenital heart disease* (pp. 253–269). Mount Kisco: Futura Publishing.
- Wilson, D. I., Burn, J., Scambler, P., & Goodship, J. (1993). DiGeorge syndrome, part of CATCH 22. *Journal of Medical Genetics*, 30, 852–856.

Catecholamine System

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Structure

The most abundant catecholamines are epinephrine (adrenaline), norepinephrine (noradrenaline), and dopamine. Catecholamines are produced from phenylalanine and tyrosine.

Dopaminergic System

Dopamine is produced by neurons in the substantia nigra, the ventral tegmental area, and hypothalamus. These neurons project to many areas of the brain, including the prefrontal cortex, the amygdala, the hippocampus, and striatum. Dopamine released by the hypothalamus also acts as a neurohormone, inhibiting the release of prolactin from the anterior lobe of the pituitary. In the periphery, dopamine is also produced in the adrenal medulla. Dopamine activates five known types of receptors (D1–D5).

Function

Norepinephrine and dopamine act as neuromodulators in the brain and also as peripheral hormones in the blood circulation. Norepinephrine is a neuromodulator of the peripheral sympathetic nervous system.

Central catecholamine function is important for regulating many behaviors, e.g., cognition, movement, sleep, mood, attention, and learning.

In the periphery, catecholamine release increases heart rate, blood pressure, and blood glucose, generally associated with the response to an environmental stressor.

Pathophysiology

Abnormally high levels of central and peripheral catecholamines can be caused by trauma (brainstem), neuroendocrine tumors (e.g., for the periphery in the adrenal medulla – a condition known as pheochromocytoma). Monoamine oxidase A (MAO-A) deficiency can also lead to elevated levels of central and peripheral catecholamines.

See Also

- ▶ [Catechol-O-methyltransferase](#)
- ▶ [Dopamine](#)
- ▶ [Epinephrine](#)
- ▶ [Norepinephrine](#)

Catechol-O-Methyltransferase

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Synonyms

[COMT](#)

Definition

Enzyme that catalyzes the O-methylation of catecholamine neurotransmitters and catechol hormones, leading to their inactivation.

There are two known isoforms:

A membrane-bound isoform (MB-COMT) and a soluble cytoplasmic isoform (S-COMT).

See Also

- [Catecholamine System](#)
- [Epinephrine](#)

Category Fluency

- [Verbal Fluency](#)

CAT-Q

- [Camouflaging Autistic Traits Questionnaire \(CAT-Q\)](#)

Caudate

- [Caudate Nucleus](#)

Caudate Nucleus

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Synonyms

[Caudate](#); [Neostriatum](#); [Striatum](#)

Structure

Anatomical Structure

The caudate nucleus, along with the putamen, globus pallidus (GP), subthalamic nuclei, and substantia nigra (SN), makes up a larger collection of nuclei called the basal ganglia. The two caudate nuclei, each residing within a hemisphere, sit alongside the lateral ventricles, superior to the thalamus, and laterally bound by the internal capsules. Its C-shaped structure consists of three identifiable regions: (1) the bulbous “head” lying ventral to the putamen and forming the anterior horn of the lateral ventricle, which tapers to (2) the long, curved “body” which moves posteriorly forming the floor of the lateral ventricle and then curves anteriorly to end at (3) the thinner “tail” near the posterior end of the thalamus and forming the roof of the temporal horn of the lateral ventricle. The tail, or *cauda* in Latin, is the namesake for this structure.

The caudate nucleus and putamen together form the striatum (or neostriatum). Although these two structures share embryonic origin, starting as a single nuclear mass, they develop into anatomically distinct structures divided by the internal capsule. Yet, the internal capsule does not completely separate the caudate nucleus from the putamen. At the head of the caudate nucleus, a striated cell bridge, made up of gray matter extensions, joins the caudate nucleus to the putamen, thus giving its name “striatum.” The striatum (caudate nucleus and putamen) and the GP comprise the corpus striatum. Furthermore, the ventral portion of the caudate nucleus, the putamen, nucleus accumbens, and anterior perforated substance make up the ventral striatum. These classifications are used to differentiate structure, afferent and efferent projections, associated neurotransmitters, and functions.

Histology

The majority (~90%) of the neurons that make up the caudate nucleus are efferent spiny dendrites which release gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter. The remaining neurons, those without spines, connect internally and use the excitatory neurotransmitter, acetylcholine (ACh).

Neural Connections

Like the other basal ganglia nuclei, the caudate nucleus has a multitude of nerve connections, serving important and widespread functions, which will be discussed in greater detail in the next section. Here, the major afferent (excitatory) and efferent (inhibitory) projections will be discussed.

Afferent (or Input) Nuclei: The major afferent connections are from the cerebral cortex and substantia nigra. The corticostriatal connection (from the cerebral cortex to the caudate nucleus) originates primarily from the frontal and association cortices, in particular the prefrontal and parietal regions. (The putamen in comparison receives its projections from the primary motor, premotor, supplementary motor, and somatosensory cortices.) All afferent connections are excitatory and glutamergic. Additionally, these afferent connections are ipsilaterally and topographically organized, such that within the same hemisphere, the frontal lobe inputs onto the head of the caudate nucleus, the parietal and occipital lobes onto the body, and the temporal lobe onto the tail.

Efferent (or Output) Nuclei: The major efferent connections of the caudate nucleus are to the internal and external segment of the globus pallidus (GPint and GPext, respectively) and the substantia nigra pars reticulata and compacta (SNr and SNc, respectively). The striatopallidus (from the striatum to the globus pallidus) and striatonigral (from the striatum to the substantia nigra) efferents are inhibitory and GABAergic. The GPint efferents then project to the thalamus, enervating the dorsomedial nucleus, intralaminar nuclei, and parts of the ventral anterior nuclei. The SNr efferents project to the superior colliculus (SC) of the eye and the ventral anterior and ventral lateral thalamic nuclei.

Intrinsic (or Internal) Nuclei: The GABAergic inhibitory striatopallidal and striatonigral connections are not the only intrinsic connections within the striatum. Nigrostriatum connections (originating in the SNr and projecting to the striatum) are dopaminergic may have either excitatory or inhibitory effects, depending on which type of receptor the neurotransmitter binds.

Pathways

The afferent and efferent neurons of the caudate nucleus (and putamen too) participate in the direct and indirect feedback loop pathways of the thalamus, having either excitatory or inhibitory effects, respectively. In the direct pathway, the inhibitory GABAergic effect of the efferent neurons releases the GPint from inhibition, thus creating a net excitatory reaction. Alternatively, in the indirect pathway, GABA from the striatum inhibits the GPext and has downstream effects in the subthalamic nuclei and the GPint, ultimately leading to a net inhibitory effect. Thus, whether the caudate nucleus is involved in engaging or inhibiting an action depends on which pathway wins out (DeLong 2000).

Furthermore, the caudate nucleus may also have excitatory or inhibitory effect in the cortex via dopaminergic neurons within the nigrostriatal pathway. The depolarization (stimulation) or hyperpolarization (inhibition) of a cell is highly dependent on the dopamine receptor on the postsynaptic terminal.

Function

Most of our knowledge of the functionality of the caudate nucleus come from a variety of animal studies, human lesion studies, and more recently, functional magnetic resonance imaging (fMRI). The caudate nucleus, which was once thought to have its influences limited to the sensorimotor system, is now known to be heavily involved in executive function, memory, and even some aspects of social communication. Consequently, most of the caudate's role in sensorimotor functioning, except for that of higher-level control, has since be re-established as being the role of the putamen (for review, see Middleton and Strick 2000).

From a cognitive perspective, convincing evidence points toward the caudate nucleus contributing to goal-oriented behavior (Grahn et al. 2008). Goal-oriented behavior is the appropriate stimulation of action and the selection of goals (and subgoals) based upon the expected outcome of the specific action. Thus, cognitive flexibility

and set-switching between goals become very important in goal-oriented behavior. In animal lesion and neurochemical studies, the caudate nucleus has been directly linked to the rats' ability to change or switch between choices, as it seen in reversal learning tasks (Ragazzino 2003; Ragazzino and Choi 2004), and strategies (Ragazzino et al. 2002; Yin et al. 2005) when task contingencies change (e.g., which item is rewarded, the value of the reward, schedule of reward). Furthermore, the caudate nucleus has been found to be selectively responsible for adapting to these new task contingencies and executing the appropriate switch rather than inhibiting the proponent response as the prefrontal cortex does (Dias et al. 1996). In primates, single-unit recording from the caudate nucleus revealed different patterns depending on whether the expected outcomes of the action are positive or negative (Ravel et al. 2003). Similarly, human neuroimaging evidence has found stronger activation responses in the caudate nucleus to positive reinforcement. In addition, greater activation was seen within the caudate nucleus when subjects thought they had subjective control over the outcome (Grahn et al. 2008). Thus, the caudate nucleus is necessary for both the behavior (the process of selection) and the evaluation of the outcome (choice).

This role in goal-oriented behavior and reward-based learning is not surprising given the caudate nucleus' modulation of dopamine, which is known to be heavily involved in the reward systems (Cools et al. 2009), abundance of dopamine receptors, and influence in updating information during working memory tasks (Frank and O'Reilly 2006). Essentially, the caudate nucleus is active in a constant loop of evaluating feedback, deciding what to do based upon that feedback (e.g., maintaining vs. switching response), and stimulating (or inhibiting) other regions via its multiple neural pathways to execute a response which will provide further feedback to the caudate nucleus.

Social/Language Processing: It is difficult to completely differentiate the caudate nucleus' role in social aspects and language processing from its role in the higher-order cognitive functions

discussed above. For instance, it is not surprising that neuroimaging and lesion studies have found that social rewards activate the caudate nucleus, given the fact that this structure responds similarly to monetary, and even expected (but not necessarily received), rewards (Izuma et al. 2010; Montague et al. 2002; Villablanca 2010). Involvement in social behavior is likely limited to and selectively involved in behavior associated with action-outcomes but may have important implications in social motivation which is reliant on assessing social reward.

In terms of language, evidence shows that the caudate nucleus plays may pay a role in the higher-level language processing involved in bilingualism and deciphering phonemes and meaning of words in ambiguous situations (Crinion et al. 2006). This finding was left-side unilateral which is to be expected as language function as a whole is predominately localized to the left hemisphere. Although its contribution to language processing is not directly related to action-outcome or goal-directed behavior, the caudate nucleus continues to have a critical role in situations which require an active selecting process to yield the best outcome. Here, the caudate nucleus helps determine which phonemes and/or definitions make the most sense given previous knowledge and current context.

In conclusion, the caudate nucleus is highly involved in higher-order cognitive functioning, especially in learning and memory tasks that are highly dependent on reinforcement. Its predominant role in goal-oriented behavior has been shown in rodent, primate, and human studies.

Pathophysiology

Given the structural and functional significance of the caudate nucleus, and the known executive dysfunction in autism, it is not surprising that this structure has been implicated in the pathophysiology of the disorder. Morphological, genetic, and neuroimaging studies have found evidence of abnormalities within the caudate nucleus of individuals with autism and its associated disorders. Although not all results are

consistent with each other, especially in relation to the behavioral and clinical correlates of autism, abnormalities within the caudate nucleus have been repeatedly found and likely contribute in some way to the aberrant functioning of individuals with autism and its associated disorders.

Morphological data has shown a bilateral enlargement of the caudate nucleus in individuals with autism when compared to healthy control groups (Cody Hazlett et al. 2009; Hollander et al. 2005; Langen et al. 2007, 2009; Sears et al. 1999), which remains significant even when total brain volume is taken into account. The volumetric increase (Langen et al. 2009) as well as outward deformation (Qiu et al. 2010) of the caudate nucleus has been localized to the head of the structure. Only one study (Langen et al. 2009) found unilateral malformation, with a significantly greater volumetric increase in the right caudate nucleus. Langen and colleagues additionally found that caudate volume has an atypical developmental trajectory (2009). Caudate volume increased with age in individuals with high-functioning autism compared to the inverted U-shape trajectory in typical development, peaking between the ages 7 and 8. Due to this atypical development, the greatest differences in caudate volume were seen at later ages (Langen et al. 2007, 2009). It should be noted, however, that not all studies have documented this increase in caudate volume (Langen et al. 2011). Age, specific diagnosis, intellectual functioning, and the current or previous usage of medication may have contributed to these nonsignificant findings.

At a microscopic level, Singh and Rivas documented that serum antibodies, which were not present in healthy controls, were most commonly present in the caudate nucleus (49%) of children with autism, compared to the cerebral cortex (18%) and cerebellum (9%; 2004). Although they argue that this supports an autoimmune theory of autism, more importantly, it illustrates an additional abnormality within the caudate nucleus as well as the heterogeneity of these abnormalities.

Additional atypical physiology has been found in the functional connectivity between the caudate nuclei and cerebral cortex (Turner et al. 2006). In a

functional connectivity MRI (fcMRI) study, age-matched males with autism showed decreased connectivity between the right caudate nucleus and occipital-temporal regions but increased connectivity between bilateral caudate nuclei and contralateral motor cortices compared to controls within (Turner et al. 2006). Taken all together, individuals with autism show an aberrant neural organization, which likely contributes to autism's phenotypic expression given the caudate nucleus' role in initiating direct and indirect pathways.

Given the caudate nucleus' diffuse connections throughout the brain via the direct and indirect pathways, this disrupted functional connectivity may have important implications in the executive dysfunction of autism, yet fMRI studies implicating the caudate nucleus have been relatively sparse and inconsistent. Silk and colleagues found reduced activation of the caudate nucleus in individuals with autism compared to controls during a mental rotation task, a paradigm known to rely heavily on executive functioning and working memory (2006). This finding, however, has not been replicated in other tasks relying on visuospatial skills and working memory (Luna et al. 2002). Alternatively, this group found the caudate nucleus to be involved in sensorimotor control associated with saccadic eye movements in individuals with autism but not healthy control individuals. They suggest that the caudate nucleus, as well as other structures within the frontal-striatal circuit, is recruited during saccadic eye movements as a compensatory mechanism due to a defective sensorimotor system (Takarae et al. 2007). If individuals with autism use the caudate nucleus for lower-level functions, like saccadic eye movements, then there may be less resources available for the caudate nucleus to perform higher-level cognitive tasks, like those associated with goal-oriented behavior.

Some of the most intriguing findings are not from those found in individuals with autism but those found in individuals with the genetic disorders associated with autism (see fragile X syndrome and Rett syndrome). Individuals with fragile X syndrome (FXS) not only have an increased caudate nucleus size when compared to controls (Cody Hazlett et al. 2009; Gothelf et al.

2007; Hoefft et al. 2008; Reiss et al. 1995) but also when compared to individuals with non-FXS autism (Cody Hazlett et al. 2009). The Cody Hazlett study further broke down their results to analyze the subgroups of FXS individuals with and without autism compared to autism non-FXS individuals and controls. Their results showed that both FXS groups (those with and without autism) had significantly enlarged caudate nucleus volumes compared to the autism and control groups, and there was no significant difference in the caudate volume between the two FXS groups (Cody Hazlett et al. 2009). This latter finding suggests that although both FXS and autism have been linked to enlargement of the caudate nucleus, this effect is not additive. Such that individuals with both FXS and autism do not have a greater increase in volume of the caudate nucleus. Alternatively, it may mean that individuals with both an autism and a FXS diagnosis have a greater probability of having an enlarged caudate nucleus compared to those individuals with a single diagnosis. Yet, since not all individuals with FXS have autism nor do all individuals with FXS or FXS with autism have enlargements of the caudate nucleus, it is hard to determine how these physiological abnormalities behaviorally manifest themselves in each disorder.

In comparison, age- and gender-matched girls with Rett syndrome showed smaller volumes of the caudate nucleus when compared to controls (Subramaniam et al. 1997). It should be noted, however, that although Rett syndrome is characterized by autistic-like behavior, the study did not indicate whether these individuals had a diagnosis of autism or not. Thus, a decreased caudate volume may be specific to Rett syndrome and not to autistic behavior, however this has been examined.

Although the above studies contribute significantly to the autism literature and begin to delineate the neurophysiological abnormalities in autism, only a few have examined how these structural differences may express themselves phenotypically. Langen and colleagues found significant negative correlations between caudate volume and insistence on sameness (IS) on the

ADI-R (or difficulty changing minor routines; 2009). This is consistent with Sears and colleagues finding negative correlations between caudate volume and higher-order repetitive behaviors (ADI-R C2 algorithm items), including the same IS factor as Langen et al. (2009). Interestingly, a significant positive correlation was found between low-order repetitive behaviors (stereotyped movements) and caudate volume (Sears et al. 2009). These correlations with repetitive behaviors, however, are not consistent. Two groups (Hollander et al. 2005; Rojas et al. 2006) found positive correlations between caudate volume and higher-order repetitive behaviors. These inconsistencies as well as the nonsignificant findings make discussion of this literature. Examining all the results together reveals, at least, some relationship between caudate nucleus enlargement and phenotypic behavior in individuals with autism.

In conclusion, although the caudate nucleus has been implicated in the pathophysiology of autism and its associated genetic disorders, results are relatively inconsistent. Morphological data supporting an enlargement of the caudate nucleus in individuals with autism remains the most replicated, but even these results are not always in agreement, especially when in relation to diagnostic criteria. Findings from the FXS and Rett syndrome studies may have important implications in the genetic pathophysiology of autism and should be examined in greater detail. Additionally, given the known functional importance of caudate nucleus in behavioral flexibility and reversal learning, known to be affected in autism, more studies should aim to identify where the functional abnormalities of the caudate nucleus are in individuals with autism. At this time though, despite inconsistencies in the literature, the caudate nucleus remains an important structure when examining the etiology of autism due to its significant structural, neurochemical, and functional connections.

See Also

► [Executive Function \(EF\)](#)

References and Reading

- Anagnostou, E., & Taylor, M. J. (2011). Review of neuroimaging in autism spectrum disorders: What we have learned and where we go from here. *Molecular Autism*, 2, 4.
- Cody Hazlett, H., Poe, M. D., Lightbody, A. A., Gerig, G., MacFall, J. R., Ross, A. K., Provenza, J., Martin, A., Reiss, A. L., & Piven, J. (2009). Teasing apart the heterogeneity of autism: Same behavior, different brains in toddlers with fragile X syndrome and autism. *Journal of Neurodevelopmental Disorders*, 1, 81–90.
- Cools, R., Frank, M. J., Gibbs, S. E., Miyakawa, A., Jagust, W., & D'Esposito, M. (2009). Striatal dopamine predicts outcome-specific reversal learning and its sensitivity to dopaminergic drug administration. *Journal of Neuroscience*, 29, 1538–1543.
- DeLong, M. R. (2000). The basal ganglia. In E. R. Kandel, J. H. Schwartz, & T. M. Jessell (Eds.), *Principles of neural science* (pp. 853–867). New York: McGraw-Hill.
- Dias, R., Robbins, T. W., & Roberts, A. C. (1996). Dissociation of the prefrontal cortex of affective and attentional shifts. *Nature*, 380, 69–72.
- Frank, M. J., & O'Reilly, R. C. (2006). A mechanistic account of striatal dopamine function in human cognition: Psychopharmacological studies with cabergoline and haloperidol. *Behavioral Neuroscience*, 120, 497–517.
- Gothelf, D., Fufaro, J. A., Fumiko, H., Eckert, M. A., Hall, S. S., et al. (2007). Neuroanatomy of Fragile X syndrome is associated with aberrant behavior and the Fragile X mental retardation protein (FMRP). *Annals of Neurology*, 63, 40–51.
- Grahn, J. A., Parkinson, J. A., & Owen, A. M. (2008). The cognitive functions of the caudate nucleus. *Progress in Neurobiology*, 86, 141–155.
- Hoefel, F., Lightbody, A. A., Cody Hazlett, H., Swetapadma, P., Piven, J., & Reiss, A. L. (2008). Morphometric spatial patterns differentiating boys with Fragile X syndrome, typically developing boys, and developmentally delayed boys ages 1 to 3 years. *Archives of General Psychiatry*, 65, 1087–1097.
- Hollander, E., Anagnostou, E., Chaplin, W., Esposito, K., Haznedar, M., LiCalzi, E., Wasserman, S., Soorya, L., & Buchsbaum, M. (2005). Striatal volume on magnetic resonance imaging and repetitive behaviors in autism. *Biological Psychiatry*, 58, 226–232.
- Izuma, K., Saito, D. N., & Sadato, N. (2010). Processing of the incentive for social approval in the ventral striatum during charitable donation. *Journal of Cognitive Neuroscience*, 22, 621–631.
- Langen, M., Durston, S., Staal, W., Palmen, S., & van Engeland, H. (2007). Caudate nucleus is enlarged in high-functioning medication-naïve subjects with autism. *Biological Psychiatry*, 62, 262–266.
- Langen, M., Schnack, H., Nederveen, H., Bos, D., Lahuis, B., de Jonge, M., van Engeland, H., & Durston, S. (2009). Changes in the developmental trajectories of striatum in autism. *Biological Psychiatry*, 66, 327–333.
- Langen, M., Leemans, A., Johnston, P., Ecker, C., Daly, E., Murphy, C. M., dell'Acqua, F., Durston, S., & The AIMS Consortium, & Murphy, D. G. M. (2011). Fronto-striatal circuitry and inhibitory control in autism: Findings from diffusion tensor imaging tractography. *Cortex*, 30, 1–11.
- Luna, B., Minshew, N. J., Garver, K. E., Lazar, N. A., Thulborn, K. R., Eddy, W. F., & Sweeney, J. A. (2002). Neocortical system abnormalities in autism: An fMRI study of spatial working memory. *Neurology*, 59, 834–840.
- Middleton, F. A., & Strick, R. L. (2000). Basal ganglia and cerebellar loop: Motor and cognitive circuits. *Brain Research Reviews*, 31, 236–250.
- Montague, P. R., Berns, G. S., Cohen, J. D., McClure, S. M., Pagnoni, G., Dhamala, M., Wiest, M. C., Karpov, I., King, R. D., Apple, N., & Fisher, R. E. (2002). Hyperscanning: Simultaneous fMRI during linked social interactions. *NeuroImage*, 16, 1159–1164.
- Patestas, M. A., & Gartner, L. P. (Eds.). (2006). Basal ganglia. *A textbook of neuroanatomy* (pp. 190–211). Oxford: Blackwell.
- Qiu, A., Adler, M., Crocetti, D., Miller, M. I., & Mostofsky, S. H. (2010). Basal ganglia shapes predict social, communication, and motor dysfunctions in boys with autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49, 539–551.
- Ragazzino, M. E. (2003). Acetylcholine actions in the dorsomedial striatum support the flexible shifting of response patterns. *Neurobiology of Learning and Memory*, 80, 257–267.
- Ragazzino, M. E., & Choi, D. (2004). Dynamic changes in acetylcholine output in the medial striatum during place reversal learning. *Learning and Memory*, 11, 70–77.
- Ragazzino, M. E., Jih, J., & Tzavos, A. (2002). Involvement of the dorsomedial striatum in behavioral flexibility: Role of muscarinic cholinergic receptors. *Brain Research*, 953, 205–214.
- Ravel, S., Legallet, E., & Apicella, P. (2003). Responses of tonically active neurons in the monkey striatum discriminate between motivationally opposing stimuli. *Journal of Neuroscience*, 23, 8489–8497.
- Rojas, D. C., Peterson, E., Winterrowd, E., Reite, M. L., Rogers, S. J., & Tregellas, J. R. (2006). Regional gray matter volumetric changes in autism associated with social and repetitive behavior symptoms. *BMC Psychiatry*, 13, 56.
- Sears, L., Vest, C., Mohamed, S., Bailey, J., Ranson, B., & Piven, J. (1999). An MRI study of the basal ganglia in autism. *Progress in Neuropsychopharmacology and Biological Psychiatry*, 23, 613–624.
- Silk, T. J., Rinehart, N., Bradshaw, J. L., Tonge, B., Egan, G., O'Boyle, M. W., & Cunningham, R. (2006). Visuo-spatial processing and the function of prefrontal-parietal networks in autism spectrum disorders: A functional MRI study. *The American Journal of Psychiatry*, 163, 1440–1443.

- Singh, V. K., & Rivas, W. H. (2004). Prevalence of serum antibodies to caudate nucleus in autistic children. *Neuroscience Letters*, 355, 53–56.
- Skuse, D. H., & Gallagher, L. (2000). Dopaminergic-neuropeptide interactions in the social brain. *Trends in Cognitive Neuroscience*, 13, 27–35.
- Subramaniam, B., Naidu, S., & Reiss, A. L. (1997). Neuroanatomy in Rett syndrome: Cerebral cortex and posterior fossa. *Neurology*, 48, 399–407.
- Takarae, Y., Minshew, N. J., Luna, B., & Sweeney, J. A. (2007). Atypical involvement of frontostriatal systems during sensorimotor control. *Psychiatry Research*, 156, 117–127.
- Ten Donkelaar, H. J., van de Warrenburg, B., Willemsen, M., Kusters, B., Hashizume, Y., & Hori, A. (2011). Basal ganglia. In H. J. Ten Donkelaar (Ed.), *Clinical neuroanatomy: Brain circuitry & its disorders* (pp. 495–564). Heidelberg: Springer.
- Turner, K. C., Frost, L., Lisenhardt, D., McIlroy, J. R., & Muller, R.-A. (2006). Atypically diffuse functional connectivity between caudate nuclei and cerebral cortex in autism. *Behavioral and Brain Functions*, 2, 34.
- Verhoeven, J. S., De Cock, P., Lagae, L., & Sunaert, S. (2010). Neuroimaging in autism. *Neuroradiology*, 52, 3–14.
- Villablanca, J. R. (2010). Why do we have a caudate nucleus? *Acta Neurobiologiae Experimentalis*, 70, 95–105.
- Yin, H. H., Ostlund, S. B., Knowlton, B. J., & Balleine, B. W. (2005). The role of the dorsomedial striatum in instrumental conditioning. *European Journal of Neuroscience*, 22, 513–523.

Cause and Effect

- [Qualitative Versus Quantitative Approaches](#)

CBCL 1.5–5

- [Child Behavior Checklist in Autism](#)

CBCL 6–18

- [Child Behavior Checklist in Autism](#)

CCC-2

- [Children’s Communication Checklist \(CCC-2\)](#)

CCPT

- [Conners’ Continuous Performance Test](#)

CDCVH

- [Common Disease-Common Variant Hypothesis](#)

CDIs

- [MacArthur-Bates Communicative Development Inventories, Second Edition](#)

CdLS

- [Cornelia de Lange Syndrome](#)

CDT

- [Clock Drawing](#)

Ceiling Effect

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Definition

This phenomenon occurs when a test item is so easy that a large number of test takers achieve the highest score possible on that item. Another way of viewing the phenomenon would be to describe such easy items as ones that fail to distinguish

between levels of ability of the test takers. If nearly all or all testees get the Vineland adaptive behavior item right, then it is a useless item, from a psychometric perspective and must never see the light of clinical brightness.

CELF Preschool – 2

► [Clinical Evaluation of Language Fundamentals: Preschool – Second Edition \(CELF Preschool-2\)](#)

CELF-5

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Description

The *Clinical Evaluation of Language Fundamentals–Fifth Edition* (CELF-5; Wiig et al. 2013a) is a battery of tests designed to assess, diagnose, and measure changes in oral and written language and verbal and nonverbal communication in individuals 5–21 years of age. The CELF-5 can be used to identify strengths and weaknesses in language, determine service eligibility, provide intervention strategies, and measure intervention efficacy. The CELF-5 is individually administered by speech-language pathologists, school psychologists, special educators, and diagnosticians with training and experience in administration and interpretation of individually administered standardization language tests and knowledge of language structure rules.

The CELF-5 provides the flexibility of administering only the tests needed to answer referral questions for assessment and evaluation. Thus, the testing process can be individualized to meet a student's unique needs, their functional language, and the language behaviors in both clinical and educational settings (Wiig et al. 2013b). Across settings, assessment begins with gathering

information regarding the presence of a language disorder and/or a student's language performance at school and at home. The CELF-5 provides that Observational Rating Scale (ORS), as a tool to systematically document observations as a means to provide descriptive information to help develop plans for intervention.

Diagnostic Battery

The CELF-5 provides a better balance of items across receptive and expressive modalities, language content, and overall structure than its previous editions (Wiig et al. 2013b). It has been developed and researched to enable examiners the flexibility of using each group of items independently (e.g., Linguistic Concepts, Semantic Relationships, and Understanding Spoken Paragraphs) of one another. The CELF-5 battery includes revised tests from previous editions as well as new tests used to evaluate word meaning and vocabulary (semantics), word sentence structure (morphology and syntax), the rules of oral language used in responding to and conveying messages (pragmatics), the recall and retrieval of spoken language (memory), and a new test used to evaluate aspects of literacy (reading comprehension and written language production) (Wiig et al. 2013a).

Administration

As a result of the flexibility in administration, the average length of administration for the CELF-5 can vary across referral questions and age groups. Administration of the Core Language tests takes approximately 34 min for ages 5:0–8:11 and 42 min for ages 9:0–21:11. Administration of the tests needed to derive the Receptive Language Index takes an additional 16 min on average for ages 5:0–8:11, and 9 min for ages 9:0–21:11. No additional time is required to derive the Expressive Language Index for ages 5:0–8:11, as all required tests for this index are part of the Core Language Score. Additionally, in order to derive the Expressive Language Index, an additional 12 min is needed for ages 9:0–21:11. Overall, in order to administer the Core Language, Receptive Language, and Expressive Language Indexes, administration takes an average of 50 min for ages 5:0–8:11 and 62 min for ages 9:0–21:11.

Once the CELF-5 assessment process is complete, clinicians must interpret the results, provide extension testing to test the limits of the student's performance, and synthesize and report all assessment information (Wiig et al. 2013b).

Historical Background

The original CELF was published in 1980 by Eleanor Messing Semel. Since its initial publication, the CELF has undergone four revisions. It was originally developed to identify language disabilities, provide diagnostic information, and identify relative strengths and weaknesses in order to establish priorities for treatment and follow-up intervention. The CELF-3, the second revision to the CELF, was frequently used to evaluate individuals who had suffered traumatic brain injuries (TBI) (Semel et al. 1995; Paslawski 2005). In 2003, the CELF-4 (Semel et al. 2003) was published. It was primarily used to screen for and diagnose language disorders in children and young adults aged 5–21. The norms of the CELF-4 were updated based on a diverse standardization using a US sample of 2,650 individuals that reflected the 2000 US census. Of this sample, 39% identified as minorities and 10% reported having a disability (Wiig et al. 2013b). The CELF-4 was designed as a tool for the clinical decision-making process, including making a diagnosis, determining the severity of a language disorder, identifying relative strengths and weaknesses, making recommendations regarding accommodations and intervention, and measuring the efficacy of intervention (Paslawski 2005).

Additionally, in 2004, the CELF-Preschool, second edition was developed to (CELF Preschool-2) assesses language ability in children ages 3–6 (Wiig et al. 2004; Paslawski 2005). Similar to the CELF-4, the CELF Preschool-2 was used to identify and diagnose language deficits in children and for the purposes of follow-up. The CELF Preschool-2 has four levels of assessment with respect to language disorders, including identification of a language disorder, description of the disorder, assessing the effect of the disorder on classroom functioning, and pragmatics. The

CELF Preschool-2 also overlaps with the CELF-4 for children ages 5 and 6.

Psychometric Data

Standardization for the CELF-5 occurred in 2012, using a sample of 3,250 English-speaking individuals in the USA between the ages of 5 and 21. Participants were gathered from 47 states (Wiig et al. 2013) and were stratified according to age, race/ethnicity (White, Hispanic, African American, Asian, and Other), geographical region (West, Midwest, Northeast, and South), and parent/caregiver education level (less than a high school diploma, high school diploma, some college or technical school, and 4 or more years of college) (Wiig et al. 2013a). Five percent of participants reported having an attention disorder; 1% a learning disability, intellectual disability, pervasive developmental disorder, Down syndrome, or developmental delay; and less than 1% reported having an emotional disturbance, cerebral palsy, color blindness, central auditory processing disorder, visual impairment, autism, or other diagnoses. Approximately 7% were diagnosed with a speech and/or language disorder, 4% with articulation or phonological disorder, and <1% with fluency/voice disorder (Wiig et al. 2013a).

Internal consistency of the CELF-5 was measured using the split-half method with the Spearman–Brown correction formula (Wiig et al. 2013). The average subtest reliability coefficients ranged from acceptable (0.77) to excellent (0.99) for ages 5:0–8:11, while the reliability coefficients for the indexes were excellent and ranged from 0.93 to 0.97. For individuals between the ages of 9:0–21:11, the average subtest reliability coefficients were acceptable (0.60) to excellent (0.99), while the reliability coefficients for the indexes were excellent and ranged from 0.92 to 0.97. Internal consistency was also calculated for individuals from three special populations: language disorders, autism spectrum disorder, and reading and/or writing learning disability (Wiig et al. 2013a). Coefficients for these groups ranged from acceptable (0.75) to excellent (0.99) for the

subtests. Index coefficients were not reported (Wiig et al. 2013a).

Test–retest stability was obtained via Pearson’s product–moment correlation by administering the CELF-5 twice within a 7–46-day interval to 137 participants (Wiig et al. 2013a). Participants were grouped in three age groups (5:0–6:11, 8:0–9:11, and 12:0–16:11). Results for the 5:0–6:11 age group indicated acceptable (0.68) to excellent (0.92) subtest stability and good (0.84–0.89) composite stability (Wiig et al. 2013a). Similarly, results for the 8:0–9:11 age group indicated adequate (0.77) to good (0.89) subtest stability and good (0.87) to excellent (0.92) composite stability. Lastly, results for the 12:0–16:11 age group indicated poor (0.56) to excellent (0.93) subtest stability and good (0.86) to excellent (0.91) composite stability (Wiig et al. 2013a).

The majority of subtests on the CELF-5 are objectively scored (i.e., correct or incorrect), thus they were not analyzed for interrater reliability. However, the following subtests require qualitative judgment for scoring of responses: Word Structure, Formulated Sentences, Word Definitions, and Structured Writing. Overall interrater reliability for these subtests was excellent and ranged from 0.91 (Formulated Sentences) to 0.99 (Word Structure) (Wiig et al. 2013a).

Furthermore, good to strong interrelationships among all subtests and composites support the validity of the CELF-5. Intercorrelations ranged from 0.19 to 0.65 for subtests and from 0.72 to 0.97 for composites. Additionally, the relationship among scores on the CELF-5 and other measures of language development informed the measure’s concurrent validity (Wiig et al. 2013a). Correlations between CELF-5 and CELF-4 subtests were adequate (0.64) to good (0.88), whereas correlations between the indexes were good (0.82) to excellent (0.92). Additional comparisons were made with the Peabody Picture Vocabulary Test–Fourth Edition (PPVT-4; Dunn and Dunn 2007) and the Expressive Vocabulary Test–Second Edition (EVT-2; Williams 2007). The PPVT-4 indicated adequate (0.75) to excellent (0.95) correlations with CELF-5 subtests and adequate (0.68) to good (0.80) correlations with CELF-5

indexes. Similarly, comparisons with the EVT-2 indicated adequate (0.71) to excellent (0.98) correlations with CELF-5 subtests and adequate (0.65–0.78) correlations with CELF-5 indexes (Wiig et al. 2013a).

Clinical Uses

The CELF-5 is a comprehensive assessment that is sensitive to cultural and linguistic diversity and addresses components within the World Health Organization’s International Classification of Functioning, Disability, and Health (2001) (Wiig et al. 2013b). This assessment tool has been developed to aide in the identification of reading and writing difficulties as well as to determine problems with spoken language and the possible impact it may have on a student’s written language. Therefore, the CELF-5 assists clinicians in evaluating a student’s strengths and weaknesses, communicating a student’s needs, addressing parent and teacher concerns, better identifying deficits in social language skills, and identifying the need for an Individualized Education Program (IEP) (Wiig et al. 2013b).

Overall, the CELF-5 allows clinicians to evaluate a student’s general language ability and obtain information that aids in determining if a student has a language disorder by administering four to six tests. Once a language disorder has been determined, the assessment process can be extended in order to further investigate areas of strength and weaknesses. Clinicians are able to determine whether significant differences exist between comprehension and expression, identify weaknesses in the areas of morphology and syntax or semantics, identify how the oral language disorder might affect a student’s written language skills, and examine if the identified language disorder affects the student’s social language interactions.

See Also

- **Peabody Picture Vocabulary Test, Fourth Edition (PPVT)**

References and Reading

- Dunn, L. M., & Dunn, D. M. (2007). Peabody picture vocabulary test (4th ed.). Bloomington: NCS Pearson.
- Paslawski, T. (2005). The clinical evaluation of language fundamentals, fourth edition (CELF-4). *Canadian Journal of School Psychology*, 20(1–2), 129–134. <https://doi.org/10.1177/0829573506295465>.
- Semel, E., Wiig, E., & Secord, W. (1987). *Clinical evaluation of language fundamentals* (Rev. ed.). San Antonio: The Psychological Corp.
- Semel, E., Wiig, E., & Secord, W. A. (1995). *Clinical evaluation of language fundamentals* (3rd ed.). San Antonio: The Psychological Corp.
- Semel, E., Wiig, E. H., & Secord, W. A. (2003). *Clinical evaluation of language fundamentals—fourth edition (CELF-4)*. San Antonio: NCS Pearson.
- Turkstra, L. S. (1999). Language testing in adolescents with brain injury. *Language, Speech, and Hearing Services in Schools*, 30(2), 132–140. <https://doi.org/10.1044/0161-1461.3002.132>.
- Wiig, E. H., Secord, W. A., & Semel, E. (2004). *Clinical evaluation of language fundamentals—Preschool, second edition (CELF Preschool-2)*. Toronto: The Psychological Corporation/A Harcourt Assessment Company.
- Wiig, E. H., Semel, E., & Secord, W. A. (2013a). Clinical evaluation of language fundamentals—fifth edition (CELF-5). *Journal of Psychoeducational Assessment*, 33(5), 495–500.
- Wiig, E. H., Semel, E., & Secord, W. A. (2013b). *Clinical evaluation of language fundamentals—fifth edition (CELF-5)*. Bloomington: NCS Pearson.
- Williams, K. T. (2007). Expressive vocabulary test (2nd ed.). Minneapolis: NCS Pearson.

Center-Based Programs

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Definition

Center-based programs for children, adolescents, and adults with autism spectrum disorders (ASD) typically focus their interventions exclusively on this population of learners and are often based in universities although some are freestanding private programs. Center-based programs include research on intervention with ASD as an important aspect of their work.

Historical Background

In the late 1960s, shortly after Lovaas et al. (1965, 1973) demonstrated that children with autism living on a hospital's inpatient unit could learn adaptive skills, interest in the science of applied behavior analysis (ABA) as a treatment approach for autism increased in universities around the United States. For example, the Koegel Autism Center at UC, Santa Barbara, was opened in 1971 with an outpatient clinic and an experimental classroom. The research on pivotal response treatment coming from that center over the years has been highly influential to the field of ABA (e.g., Koegel et al. 1987).

The Douglass Developmental Disabilities Center opened in 1972 at Rutgers University in New Jersey as a research-based day program for school-age children with autism (Harris and Handleman 2000). That program is noted for research on teaching parents to use ABA methods (Harris 1983), for research on the assessment of children with ASD (e.g., Delmolino 2006), and for developing new ABA methods to teach skill acquisition and behavior management (e.g., Jennett et al. 2007). In 1975, Raymond Romanczyk established the Institute for Child Development at the State University of NY at Binghamton. The work on computer-based curricula coming from that center has been adopted in many places (Romanczyk and Lockshin 1982). The Walden Early Childhood program was opened on the campus of University of Massachusetts in 1985 and has since relocated to the Emory University School of Medicine (McGee et al. 2001). Their emphasis on incidental teaching with preschool-age children has had an important impact on preschool programs for children with ASD (McGee et al. 1999).

Not all centers are university based. For example, the Princeton Child Development Institute in New Jersey (McClannahan and Krantz 2001) is a freestanding private program that has an affiliation with the University of Kansas but is physically far removed from that campus. They have made major contributions to the understanding of the treatment of ASD including a competency-based staff training program and the use of

activity schedules to help students with ASD function independently (McClannahan and Krantz 1999). Another freestanding program located in New Jersey that has a research focus is the Alpine Learning Group which was founded in 1989 and contributes research findings in several areas of ABA (e.g., Meyer et al. 2000).

Rationale or Underlying Theory

Many center-based programs are at universities in which innovative research in the treatment of autism spectrum disorders can most efficiently be done, and others are private programs that place a high value on doing research as part of their mission. Once new ABA teaching techniques have been developed in these environments, they are fine-tuned to work in school-based and home-based settings. Instructional methods developed in research settings have very limited value if they can only be applied in the center where they were created. It is essential that the methods be shown to be effective when used by well-trained staff members in community settings as well. The Princeton Child Development Institute, for example, has consulted to several replication sites that adopted their approach. These sites are located in College Point, NY; New Milford, NJ; Bedminster, NJ; Maplewood, NJ; Gdansk, Poland; and Istanbul, Turkey.

Goals and Objectives

One goal of center-based programs is to develop effective treatments for learners with an autism spectrum disorder (ASD). For university-based programs, another goal is teaching undergraduate and graduate students how to implement these methods. After they leave the university, these students can bring the ABA treatment methods into the wider community and help disseminate cutting-edge techniques in public and private schools. Some center-based programs have staff members who consult to schools and families about the most effective ways to educate students with ASD and share their knowledge through that consultation.

There are significant advantages to providing treatment in a center-based program. One of these is that the entire staff is focused on the treatment of ASD, and this depth of talent ensures that if a teacher is on jury duty or an assistant teacher is on medical leave, there will be other experienced staff members able to step in and maintain a high-quality program for a learner. Public schools rarely have the resources to ensure that kind of coverage, and parents running their own home-based program may find themselves overwhelmed when there are not enough staff members to cover the teaching hours in the day. Another advantage is that center-based programs typically use cutting-edge teaching methods. These data-based methods offer the learner a major advantage in terms of the likelihood of making progress over time.

One potential disadvantage of a center-based program is that there may not be easy access to typically developing peers. By contrast, the public schools are primarily comprised of youngsters in regular education classes who can be invited to serve as role models. To compensate for the lack of neurotypical peers, some center-based programs, especially at the preschool level, include a classroom of typically developing preschool children who can be role models and friends for young children with ASD. This provides an inclusive experience for the child who is getting ready to go to kindergarten in a public school. In addition, when children in a center-based program are ready to be transitioned to their home districts, they will make many visits over an extended period of time to help them feel comfortable when they are fully included in the public school. This transition process allows the center-based staff to identify skill deficits that need to be addressed for the child to fit into the new placement. Older learners who still require intensive services of a center-based program often spend significant amounts of time in community settings where they are exposed to children or adults of their own age.

The extent of parental control varies by instructional setting. In home-based programs, parents are typically present for much of the instructional time and are active in making day-by-day decisions. Some parents value this role and expect to

be very active in their young child's education. However, in families where both parents must work or in single-parent families, it is not feasible for parents to be at home overseeing the teaching programs and still earn a living to support their family. Under these conditions, a center-based program or a school-based program has the advantage of allowing parents to leave much of the daily decision-making to the educational team. By law, parents must have a voice in planning their child's education, but when the program is not home based, they do not have the intensive control of daily decision-making that is possible in their own home.

Treatment Participants

Children of all ages, adolescents, and adults may be served by center-based programs. The centers vary in how they select learners. They may recruit students with specific educational needs, for example, significant speech delays or problems with forming important visual or auditory discriminations, to test a new intervention. Alternatively they may admit students who cannot be accommodated in the public schools because of the complexity of their learning needs, the lack of trained staff with a knowledge of ASD in the district, or seriously challenging behaviors on the part of the learner. Although inclusion in a regular education class is a goal for every child, there are some learners with autism spectrum disorders whose behavioral challenges make that goal difficult, if not impossible, to achieve.

Treatment Procedures

Many center-based programs are at universities with a commitment to developing empirically supported treatments, and others are private programs which share that research goal. Because applied behavior analysis (ABA) has the best track record of providing rigorous evidence, most center-based programs employ a broad array of ABA methods. They range from naturalistic teaching in a playful setting with a young

child or helping a teenager with ASD take public transportation to using more structured methods including discrete trial teaching to help students learn factual knowledge that forms the basis for effective communication and improved cognitive skills. Among the many behaviors that children with ASD have learned with ABA techniques are empathy skills (Schrandt et al. 2009), using a greater diversity of responses (Napolitano et al. 2010), and requesting answers to novel questions (Ingvarsson and Hollobaugh 2010). Rogers and Dawson (2010) have developed ABA techniques that are developmentally informed to work with very young children starting at 1 year of age and continuing to age 5 years.

For difficult-to-manage behavior such as self-injury, aggression, or tantrums, ABA offers sophisticated functional assessment/analysis techniques followed by the development of a treatment intervention to teach the student positive alternatives to disruptive behavior (e.g., Hanley et al. 2003). For example, a child who is motivated to slide to the floor because it gains her teacher's attention might learn to raise her hand or give the teacher a card that says "Talk to me please." Similarly, a teenager who is motivated to avoid a task might learn to ask for a "break please" or give the teacher a "break card."

Efficacy Information

As reflected in this encyclopedia, there is a substantial body of empirical data demonstrating that techniques based on the principles of ABA can be highly effective in teaching new skills in multiple domains including communication, social behavior, adaptive behaviors, vocational skills, and the self-control of maladaptive behaviors. Much of this research comes from center-based programs (e.g., Charania et al. 2010; Koegel et al. 1997; Miguel et al. 2009).

Outcome Measurement

Starting with the pioneering work of Lovaas, much of the published outcome research has

evaluated home-based treatment. Center-based research often results in research articles focused on changes in specific behaviors. For example, R. L. Koegel and L. K. Koegel (Koegel and Koegel 2006) use single-subject designs to illustrate changes in communication and social and academic skills when children are taught skills using pivotal response treatments. Single-subject designs include a multiple baseline design across individuals where two or more people have baseline (untrained) data collected on a target behavior and then one person enters treatment while the others continue in baseline. When the first person reaches criterion, the next person enters treatment and so forth. Multiple baseline designs can also be used for one participant across three or more tasks.

Another single-subject design is called a reversal design, and in using this intervention, baseline data are first collected, then the treatment is introduced, and after changes have been observed, there is return to baseline for a brief period, and finally the treatment, if demonstrated to be effective, is put in place. Single-subject designs are especially useful for the in-depth study of the influence of teaching methods on individual participants.

In addition to single-subject designs, some longer-term follow-up studies of the effectiveness of ABA treatments employ group designs in which participants are assigned randomly to different conditions including a treatment group and a group that receives the usual services available in the community (called treatment as usual, TAU). Data from these studies are analyzed using statistical methods to compare differences between groups (e.g., Harris and Handleman 2000; Rogers and Dawson 2010; Sallows and Graupner 2005; Smith et al. 2000).

Qualifications of Treatment Providers

Treatment providers in many center-based programs include assistant teachers with high school diplomas or who are university undergraduates. They are supervised by special education teachers

who have, or are working toward, their board certification as behavior analysts. Some center-based programs also have speech and language therapists who, in addition to their speech credentials, hold the BCBA certificate. Senior supervisors typically have the BCBA credential, have many years of experience, and are often faculty members engaged in research and staff training. This creates an environment that can be quite dynamic in ensuring that services remain state of the art.

See Also

- [Educational Interventions](#)
- [School to Work Transition Process](#)

References and Reading

- Charania, S. M., LeBlanc, L. A., Sabanathan, N., Ktaech, I. A., Carr, J. E., & Gunby, K. (2010). Teaching effective hand raising to children with autism during group instruction. *Journal of Applied Behavior Analysis, 43*, 493–497.
- Delmolino, L. (2006). Brief report: Use of DQ for estimating cognitive ability in young children with autism. *Journal of Autism and Developmental Disorders, 36*(7), 959–963.
- Hanley, G. P., Iwata, B. A., & McCord, B. E. (2003). Functional analysis of problem behavior: A review. *Journal of Applied Behavior Analysis, 36*, 147–185.
- Harris, S. L. (1983). *Families of the developmentally disabled: A guide to behavioral intervention*. Elmsford: Pergamon.
- Harris, S. L., & Handleman, J. S. (2000). Age and IQ at intake as predictors of placement for young children with autism: A four to six year follow-up. *Journal of Autism and Developmental Disorders, 30*, 137–142.
- Ingvarsson, F. T., & Hollobaugh, T. (2010). Acquisition of intraverbal behavior: teaching children with autism to mand for answers to questions. *Journal of Applied Behavior Analysis, 43*, 1–17.
- Jennett, H. K., Harris, S. L., & Delmolino, L. (2007). Discrete trial instruction vs. mand training for teaching children with autism to make requests. *The Analysis of Verbal Behavior, 24*, 69–85.
- Koegel, R. L., & Koegel, L. K. (2006). *Pivotal response treatments for autism. Communication, social and academic development*. Baltimore: Paul Brookes.
- Koegel, R. L., O'Dell, M. C., & Koegel, L. K. (1987). A natural language teaching paradigm for nonverbal autistic children. *Journal of Autism and Developmental Disorders, 17*, 187–200.

- Koegel, L. K., Camarata, S. M., Valdez-Menchaca, M., & Koegel, R. L. (1997). Setting generalization of question-asking by children with autism. *American Journal on Mental Retardation*, 102, 346–357.
- Lovaas, O. I., Schaeffer, B., & Simons, J. Q. (1965). Experimental studies in childhood schizophrenia. Building social behavior in autistic children by the use of electric shock. *Journal of Experimental Research in Personality*, 1, 99–109.
- Lovaas, O. I., Koegel, R. L., Simmons, J. Q., & Long, J. S. (1973). Some generalization follow-up measures on autistic children in behavior therapy. *Journal of Applied Behavior Analysis*, 6, 131–166.
- McClannahan, L. E., & Krantz, P. (1999). *Activity schedules for children with autism: Teaching independent behavior*. Bethesda: Woodbine House.
- McClannahan, L. E., & Krantz, P. (2001). Behavior analysis and intervention for preschoolers at the Princeton Child Development Institute. In J. S. Handleman & S. L. Harris (Eds.), *Preschool education programs for children with autism* (2nd ed., pp. 191–214). Austin: ProEd.
- McGee, G. G., Morrier, M. J., & Daly, T. (1999). An incidental teaching approach to early intervention for toddlers with autism. *Journal of the Association for Persons with Severe Handicaps*, 24, 133–146.
- McGee, G. G., Morrier, M. J., & Daly, T. (2001). The Walden early childhood programs. In J. S. Handleman & S. L. Harris (Eds.), *Preschool education programs for children with autism* (2nd ed., pp. 157–190). Austin: ProEd.
- Meyer, L. S., Taylor, B., Levin, L., & Fisher, J. (2000). Alpine learning group. In S. L. Harris & J. S. Handleman (Eds.), *Preschool programs in autism* (1st ed., pp. 135–155). Austin, TX: Pro-Ed.
- Miguel, C. E., Clark, K. M., Tereshko, L., & Ahearn, W. H. (2009). The effects of response interruption and redirection and sertraline on vocal stereotypy. *Journal of Applied Behavior Analysis*, 42, 883–888.
- Napolitano, D. A., Smith, T., Zarcone, J. R., Goodkin, K., & McAdam, D. B. (2010). Increasing response diversity in children with autism. *Journal of Applied Behavior Analysis*, 43, 265–271.
- Rogers, S. J., & Dawson, G. (2010). *Early start Denver model for young children with autism*. New York: Guilford Press.
- Romanczyk, R. G., & Lockshin, S. B. (1982). *The IGS curriculum*. Vestal: CBTA.
- Sallows, G. O., & Graupner, T. D. (2005). Intensive behavioral treatment for children with autism: Four-year outcome and predictors. *American Journal on Mental Retardation*, 110, 417–438.
- Schrandt, J. A., Townsend, D. B., & Poulson, C. L. (2009). Teaching empathy skills to children with autism. *Journal of Applied Behavior Analysis*, 42, 17–32.
- Smith, T., Groen, A. D., & Wynn, J. W. (2000). Randomized trial of intensive early intervention for children with pervasive developmental disorders. *American Journal on Mental Retardation*, 105, 269–285.

Central Auditory Processing Disorder

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Synonyms

Auditory perceptual disorder; Auditory processing disorder

Short Description or Definition

A central auditory processing disorder (CAPD) is defined as difficulty with “processing auditory information in the central nervous system and neurobiological activity that underlies and gives rise to the electrophysiologic auditory potentials” (American Academy of Audiology [AAA] 2010). This processing difficulty results in poor performance in localization and lateralization, auditory discrimination, auditory pattern perception, and temporal processing (American Speech-Language-Hearing Association [ASHA] 2005). Individuals diagnosed with CAPD often exhibit trouble following oral instructions, difficulty in background noise, problems with reading, spelling and language, and academic difficulties (Bamiou et al. 2001; Chermak et al. 2002).

Categorization

Currently, there is no consensus on the categorization of CAPD. There are two proposed models of CAPD in children: the Buffalo model (Katz 1992) and the Bellis-Ferre model (Bellis 2003; Ferre 1997). Both of these models attempt to categorize CAPD based on the types of difficulty the individual exhibits. However, neither of these models is based on peer reviewed data, and neither model is able to categorize all children diagnosed with CAPD (Jutras et al. 2007).

Epidemiology

The epidemiology of CAPD is not well documented at this point in time. There is one estimate that 2–3% of children have an auditory processing disorder (Chermak and Musiek 1997). In adults, it is estimated that 10–20% of elderly adults demonstrate an auditory processing disorder (Cooper and Gates 1991).

Natural History, Prognostic Factors, Outcomes

There are no longitudinal studies following individuals with CAPD, therefore, prognostic factors and outcomes cannot be estimated at this point in time. Some risk factors for CAPD have been identified, including neurological dysfunction or disorders, severe jaundice during infancy, chronic otitis media during preschool years, and family history of CAPD, hearing loss, or academic underachievement.

Clinical Expression and Pathophysiology

For most children with CAPD, the exact cause of the disorder is unknown. However, inefficient interhemispheric information transfer and imprecise neural synchrony, among other things, may be involved (Jerger et al. 2002). Neurological disorders, damage, or abnormalities may also be the cause of CAPD if the auditory areas of the brain are affected (Musiek et al. 1994).

Evaluation and Differential Diagnosis

Currently, there is no gold standard test for evaluating and diagnosing auditory processing disorders. Rather, as the definition of CAPD includes disorders of multiple auditory skills, the evaluation of CAPD should also include tests of multiple auditory skills (AAA 2010; ASHA 2005). This is done using a test battery approach. A typical CAPD evaluation will begin with a test of hearing sensitivity to rule out any type of peripheral

hearing loss. Individuals exhibit difficulty with one or more auditory functions as a result of poor processing of auditory information beyond the ear. This represents a problem with the auditory system beyond the inner ear. Therefore, an individual who is seeking a diagnosis of CAPD should not have peripheral hearing loss on a pure tone audiogram test. The current recommendations from the American Academy of Audiology state that a diagnosis of CAPD should be given if a person performs below the age-established norms on two or more tests of central auditory function (AAA 2010). Tests that may be included in a CAPD test battery can be divided into two main categories: behavioral tests and electrophysiological tests.

Behavioral tests are designed to assess one of the many areas of auditory processing. These tests require individuals to participate in the testing process in some way. This includes pressing a button, raising their hand, or repeating what they hear. The main processes evaluated by behavioral tests are binaural integration, binaural separation, auditory closure, temporal resolution, temporal ordering, and understanding speech in noise. Some tests currently available to evaluate binaural integration include the Dichotic Digits Test, Staggered Spondaic Words Test, and the binaural integration subtest of the SCAN (Katz et al. 1963; Keith 2000a; Musiek 1983). These tests involve having different information presented to the left and right ear simultaneously (dichotic testing). Individuals are asked to listen to what is being presented to both ears and repeat back everything they hear. Binaural separation is also tested using a dichotic test procedure; however, they are asked to focus on the sounds presented to one ear while ignoring what they hear in the other ear. Some tests of binaural separation include the competing words and competing sentences subtests of the SCAN and the competing sentences test (Keith 2000a). Auditory closure is the brain's ability to fill in missing information. This is tested by presenting words or sentences that have had some information removed and asking individuals to repeat what they hear. Some examples of auditory closure tests include low-pass filtered speech and compressed speech.

Temporal processing is related to the timing aspects of sound. Temporal resolution is the ability to hear changes in a sound over time (Moore 2003). This is often evaluated using a gap detection task, in which individuals are asked to press a button when they hear a small piece of silence embedded in static noise. Clinically applicable tests of temporal resolution include the Gaps in Noise (GIN) Test (Musiek et al. 2005) and the Random Gap Detection Test (Keith 2000b). Temporal ordering is the skill of determining the order in which multiple stimuli were presented. This is evaluated by presenting two to three sounds that vary in some aspect (frequency or duration) and asking individuals to report in what order they heard the sounds. These tests are the Frequency Pattern Test and the Duration Pattern Test (Musiek 1994).

Because one of the most common complaints of someone with CAPD is difficulty understanding speech in the presence of background noise, many CAPD test batteries include a speech in noise test. These tests present words or sentences with some type of background noise. This noise may be broadband or multi-talker babble. The difference in loudness between the signal that the individual must repeat (words or noise), and the background noise may vary. Some of these tests include the Words in Noise (WIN) test, the QuickSIN, the Hearing in Noise Test (HINT), and the Speech Perception in Noise (SPIN) Test.

The difficulty with all of these CAPD tests for use with individuals diagnosed with autism is that they require the individual to actively and cooperatively participate in the test procedures. These tests also use varying amounts of speech materials for testing and/or instruction. This requires each individual tested to have normal or near normal speech and language abilities and normal cognitive function in order to complete the tests. These requirements would disqualify most individuals with autism from being able to reliably complete the test procedures. Therefore, testing and diagnosis of CAPD is not typically done on individuals with autism.

Electrophysiological (evoked potential) tests involve placing small recording electrodes on the surface of the scalp, forehead, and ears. For this

type of testing, the individual is asked to sit quietly while listening to various sounds. The electrodes record responses from groups of neurons in the brainstem and brain when a sound is presented. Individuals with CAPD may have abnormalities in the size or timing of their evoked potentials responses. Some studies have also found abnormalities on some electrophysiological tests of individuals with autism, although the research findings tend to be mixed (Marco et al. 2011). These tests allow the evaluation of the function of auditory areas of the central nervous system without active participation on the part of the individual. However, electrophysiological tests alone cannot diagnose CAPD. The results of these tests should be combined with behavioral test measures to diagnose CAPD.

Treatment

Treatment options for CAPD fall into four general categories: environmental modifications, informal auditory training, formal auditory training, and computer-based training. Environmental modifications are designed to improve the signal-to-noise ratio (SNR) for the child with CAPD. SNR is the intensity of the signal the listener is meant to attend compared to the intensity of the background noise that should be ignored. Some of these modifications may include offering preferential seating in the classroom, reducing background noise, providing written instructions for assignments and projects, previewing or pre-teaching classroom materials, and some form of assistive listening technology. The goal of these strategies is to improve the individual's functioning in difficult listening situations, not to remediate the CAPD directly.

Informal auditory training and formal auditory training are activities created for each individual that are designed to improve the specific auditory skills with which the child has difficulty. These activities are either completed at home (informal training) or during scheduled rehabilitation sessions (formal training) with a speech-language pathologist or audiologist. The exact activities will be individualized for each individual and

may include auditory skills similar to those used during the test procedures. These activities begin with easier tasks and progress to more difficult assignments.

See Also

- ▶ [American Speech-Language-Hearing Association Functional Assessment of Communication Skills](#)
- ▶ [Auditory Processing](#)
- ▶ [Dichotic Listening](#)

References and Reading

- American Academy of Audiology. (2010). *Clinical Practice Guidelines: Diagnosis, treatment and management of children and adults with central auditory processing disorder*. Reston: American Academy of Audiology.
- American Speech-Language-Hearing Association. (2005). *(Central) Auditory processing disorders-the role of the audiologist*. Bethesda: American Speech-Language-Hearing Association.
- Bamiou, D. E., Musiek, F. E., & Luxon, L. M. (2001). Aetiology and clinical presentations of auditory processing disorders – A review. *Archives of Disease in Childhood*, 85, 361–365.
- Bellis, T. J. (2003). *Assessment and management of central auditory processing disorders in the educational setting: From science to practice* (2nd ed.). Toronto: Thomson Delmar Learning.
- Chermak, G., & Musiek, F. (1997). *Central auditory processing disorders: New perspectives*. San Diego: Singular.
- Chermak, G., Tucker, E., & Seikel, J. A. (2002). Behavioral characteristics of auditory processing disorder and attention-deficit hyperactivity disorder: Predominantly inattentive type. *Journal of the American Academy of Audiology*, 13, 332–338.
- Cooper, J. C., Jr., & Gates, G. A. (1991). Hearing in the elderly – The Framingham cohort, 1983–1985: Part II. Prevalence of central auditory processing disorders. *Ear and Hearing*, 12, 304–311.
- Ferre, J. (1997). *Processing power: A guide to CAPD assessment and management*. San Antonio: Communication Skills Builders.
- Jerger, J., Thibodeau, L., Martin, J., Mehta, J., Tillman, G., Greenwald, R., et al. (2002). Behavioral and electrophysiologic evidence of auditory processing disorder: A twin study. *Journal of the American Academy of Audiology*, 13, 903–912.
- Jutras, B., Loubert, M., Dupuis, J. L., Marcoux, C., Dumont, V., & Baril, M. (2007). Applicability of central auditory processing disorder models. *American Journal of Audiology*, 16, 100–106.
- Katz, J. (1992). A classification of auditory processing disorders. In J. Katz, N. Stecker, & N. Henderson (Eds.), *Central auditory processing: A transdisciplinary view*. Baltimore: Mosby-Yearbook.
- Katz, J., Basil, R. A., & Smith, J. M. (1963). A staggered spondaic word test for detecting central auditory lesions. *The Annals of Otolaryngology, Rhinology, and Laryngology*, 72, 908–917.
- Keith, R. W. (2000a). Development and standardization of SCAN-C test for auditory processing disorders in children. *Journal of the American Academy of Audiology*, 11, 438–445.
- Keith, R. W. (2000b). *Random gap detection test*. St. Louis: Auditec of St Louis Ltd.
- Loo, J. H., Bamiou, D. E., Campbell, N., & Luxon, L. M. (2010). Computer-Based Auditory Training (CBAT): Benefits for children with language- and reading-related learning difficulties. *Developmental Medicine and Child Neurology*, 52, 708–717.
- Marco, E. J., Hinkley, L. B. N., Hill, S. S., & Nagarajan, S. S. (2011). Sensory processing in autism: A review of neurophysiologic findings. *Pediatric Research*, 69, 48R–54R.
- Moore, B. (2003). *An introduction to the psychology of hearing* (5th ed., pp. 163–194). San Diego: Academic.
- Musiek, F. (1983). Assessment of central auditory dysfunction: The dichotic digits test revisited. *Ear and Hearing*, 4, 79–83.
- Musiek, F. (1994). Frequency (pitch) and duration pattern tests. *Journal of the American Academy of Audiology*, 5, 165–168.
- Musiek, F. E., Baran, J., & Pinheiro, M. (1994). *Neuroaudiology: Case studies*. San Diego: Singular Publishing Group.
- Musiek, F., Shinn, J., Jirsa, R., Bamiou, D., Baran, J., & Zaidan, E. (2005). The GIN (Gaps in Noise) Test performance in subjects with and without confirmed central auditory nervous system involvement. *Ear & Hearing*, 26, 608–618.

Central Auditory Processing Disorder (CAPD)

- ▶ [Auditory Processing](#)

Central Nervous System (CNS)

- ▶ [Autonomic Nervous System](#)

Cerebellar Abnormalities in Autism

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Definition

Cerebellar abnormalities consist of some of the earliest neurobiological findings to be described in autism spectrum disorders (ASD). Like other brain anomalies reported in this disorder, cerebellar abnormalities are diverse with varying levels of inconsistency and specificity. Therefore, the contribution of the cerebellum to the pathophysiology of ASD remains unclear. Nevertheless, this structure continues to be of great interest to the autism research community in light of growing evidence suggesting a role that goes beyond motor coordination (see entry: ► [“Cerebellum”](#)). More precisely, recent research has provided clear evidence supporting the involvement of the cerebellum in emotion processing and cognition (Schmahmann and Sherman 1998) which are commonly impaired in individuals with ASD.

This entry briefly summarizes the research literature on the cerebellum as it applies to ASD. Ovid MEDLINE search of the entire medical literature (1948–2010) revealed that this covers approximately 25 years of research. The earliest studies (1985–1999) are briefly summarized in the [“Historical Background”](#) section as there are already many published reviews on this earlier work. In the [“Current Knowledge”](#) section, a more detailed review is provided on research published in the past decade which consists of the majority of published work on cerebellar abnormalities in ASD.

Historical Background

The earliest studies describing cerebellar abnormalities in ASD were published in the mid-1980s. These studies were of two general types: neuropathology and structural neuroimaging. The first neuropathological investigations consisted of case reports or series which reported (Frazier and Hardan 2009) various cerebellar abnormalities, including the well-known finding of decreased Purkinje cell number (Bauman and Kemper 1985; Ritvo et al. 1986) which are distinctive inhibitory output neurons arising from the cerebellar cortex (see entry: ► [“Purkinje Cells”](#)). This finding was replicated a decade later by another group using a different sample, reporting Purkinje cells reduction in postmortem cerebellar tissue (Bailey et al. 1998). These initial neuropathological reports implicating the cerebellum sparked a large number of confirmatory studies with structural MRI being the most commonly used modality. These investigations produced mixed results with respect to cerebellum size and one of its major subdivisions, the vermis, which includes 10 lobules (i.e., anterior vermis, lobules I–V; posterior superior vermis, lobules VI–VII; and posterior inferior vermis, lobules VIII–X). Some reports described smaller cerebellar vermis (Courchesne et al. 1988) while others found normal (Garber and Ritvo 1992) or even larger size (Piven et al. 1997). It should be noted, however, that the initial MRI studies had significant limitations, partially owing to the novelty of this technology in neuropsychiatric research during this time period. For example, many studies reporting on size of the cerebellum refer to area as measured in midsagittal slices and not volume. Finally, a series of studies using clinical assessments to indirectly test for underlying cerebellar neuropathology were also published in the 1980s and 1990s. These investigations assessed a number of abilities believed to depend on the cerebellum, including gait (Hallett et al. 1993), attention (Courchesne et al. 1994), and eye movements (Minshew et al. 1999). These findings were also mixed, though most reports cited abnormalities suggestive of cerebellar pathology.

Current Knowledge

In the past decade, there has been a surge in the number of autism-related published studies. This large body of research was accompanied by major technological advances which enabled clinicians/scientists to study new questions and clarify existing ones. These advances have led to an increase in our understanding of the role of the cerebellum in ASD; however, there is still much to be learned. Inconsistent findings remain a challenge, and the implications of new findings are not entirely clear and/or need replication. This portion of the review is organized by research modality which includes the following: neuropathology, molecular/cellular neurobiology, neuroimaging, and clinical testing. The neuroimaging literature represents the majority of the published work, and this section will be divided further by imaging modality.

Neuropathology

While some of the most informative research has come from postmortem neuropathological examination, this group of studies continues to represent only a small minority of autism-related published studies. Nevertheless, recent research has provided new and informative insights. The well-known observation of reduced Purkinje cell counts was reported to be an inconsistent finding and may only be found in a subpopulation of individuals with ASD (Whitney et al. 2008). Moreover, in a recent neuropathological study, cerebellar pathology was commonly observed, but reduction in Purkinje cells was not consistently found (Wegiel et al. 2010). However, this does not necessarily diminish the importance of Purkinje cells as part of the neuropathology of ASD. Abnormalities in this population of cells may be expressed in other ways such as size reduction which has also been reported (Fatemi et al. 2002). In addition to Purkinje cells, basket and stellate cells (key cerebellar inhibitory interneurons) have been studied, and no abnormalities have been observed in their number or shape, suggesting that Purkinje cell loss is related to a late developmental event (Whitney et al. 2009).

Molecular/Cellular Neurobiology

This category includes human experiments done on the molecular and cellular level, usually involving the use of postmortem neural tissue or blood serum from living participants. As a broad category, these studies represent much of the research in ASD reporting on cerebellar anomalies; however, this grouping is quite diverse and represents mainly preliminary work which needs replication and further investigation. Nevertheless, the resulting research has provided significant leads into the molecular basis of ASD. One way to conceptualize this body of literature is by predicted aberrations in the following: neurotransmission, immune function, apoptosis (programmed cell death), and cell signaling. With respect to neurotransmission, abnormalities have been reported in the gamma-aminobutyric acid (GABA), nicotine, and glutamate neurotransmitter systems. There are now many studies reporting abnormalities in the GABAergic system which mainly includes abnormal expression of GABA receptors (Fatemi et al. 2009) and the rate-limiting, GABA-synthesizing enzyme, glutamic acid decarboxylase (Fatemi et al. 2002; Yip et al. 2007). Studies have also been published, although limited in number, to support abnormalities in the nicotinic (Lee et al. 2002) and glutaminergic systems (Purcell et al. 2001).

Immune dysfunction has also been implicated as part of the pathobiology of ASD. There are several studies reporting on the presence of antibodies to cerebellar proteins in the serum of individuals with ASD (Singer et al. 2006; Wills et al. 2009). A recent investigation specifically identified a neuroinflammatory process in postmortem cerebellum tissue of individuals with ASD (Vargas et al. 2005) which was subsequently supported by a similar study (Laurence and Fatemi 2005). Additionally, there are also several studies that support the presence of aberrant apoptosis in the cerebellum of individuals with ASD. These investigations reported the reduction of antiapoptotic protein Bcl-2 (Fatemi et al. 2001) and increase in proapoptotic proteins, cathepsin D and caspase-3 (Sheikh et al. 2010). Finally, many studies have found

abnormal levels of one or more of the numerous proteins involved in various cell signaling processes. Those proteins found to be abnormal in the cerebellum of individuals with ASD include reelin (Fatemi et al. 2005), phosphodiesterase (Braun et al. 2007), and neurotrophin-3 (Sajdel-Sulkowska et al. 2009).

Neuroimaging

Structural MRI

Structural MRI is one of the most commonly used neuroimaging modalities to study the neurobiology of ASD (see entry: ► [“Magnetic Resonance Imaging”](#)). Major advances in recent structural MRI methodologies or technologies include the use of novel semiautomatic morphometric software that led to improved ability to perform volumetric measurements and separately measure gray and white matter volumes. These new software programs allow the examination of increasingly larger sample sizes, enhancing statistical power and facilitating important group stratification (i.e., by age, gender, etc.). However, the inconsistencies present in the earlier structural MRI literature persisted in the newer studies. In fact, the results have become even more mixed. There are several reports documenting increases in cerebellar size, including total volume (Hardan et al. 2001), gray matter (Ke et al. 2008), white matter (Bloss and Courchesne 2007), and/or vermal lobules (Akshoomoff et al. 2004). In contrast, findings from several other studies revealed reductions in cerebellar size, including total volume (Hallahan et al. 2009), gray matter (Toal et al. 2010), white matter (Courchesne et al. 2001), and vermal lobules (Carper and Courchesne 2000). Finally, reports documenting normal cerebellar volumes have also been published (Hazlett et al. 2005). It is worth noting that most of these newer studies do not restrict their analysis to the cerebellum, and most conduct a whole-brain analysis also reporting on extracerebellar anomalies while examining different age groups. Therefore, future studies should focus on the cerebellum solely while including a large sample of participants and a narrow age range.

Functional MRI

Functional MRI studies are also among the most commonly used neuroimaging modality to study the neurobiology of ASD (see entries: ► [“Functional MRI”](#), and ► [“Magnetic Resonance Imaging”](#)). While some studies reported on the cerebellum in their analyses, a limited number of investigations focused primarily on this structure. Studies have applied motor tasks and paradigms, probing cognitive and emotional processing (Schmahmann and Sherman 1998). Motor tasks commonly involved finger tapping or button pressing, and some investigations have reported reduced (Mostofsky et al. 2009; Muller et al. 2001) while others found increased cerebellar activation (Allen and Courchesne 2003; Allen et al. 2004) in individuals with ASD when compared to controls. Abnormal cerebellar activation has also been reported in nonmotor probes of attention (Allen and Courchesne 2003), executive function (Gilbert et al. 2008), and face processing (Critchley et al. 2000). Abnormalities of how the cerebellum connects to other brain areas have also been observed in a recent investigation examining functional connectivity with evidence indicating a lack of synchronization between this structure and several brain regions during task performance (Belmonte et al. 2010; Mostofsky et al. 2009). Finally, decreased cerebellar activation has been demonstrated in studies examining resting state activity where no tasks are used (Paakki et al. 2010).

Other Neuroimaging Modalities

The remaining neuroimaging modalities, diffusion tensor imaging, magnetic resonance spectroscopy, and single-photon emission computed tomography, are less commonly used (see entries: ► [“Magnetic Resonance Spectroscopy”](#), and ► [“Magnetic Resonance Imaging”](#)). Diffusion tensor imaging has grown tremendously in popularity over the past 5 years, and there are now more than 30 published studies conducted in autism research. However, only a minority of these investigations reported cerebellar abnormalities including impaired white matter integrity as measured by fractional anisotropy. These abnormalities are found in the middle (Cheng et al.

2010) and superior cerebellar peduncles (Catani et al. 2008) which are the major fiber tracts going into and coming out of the cerebellum, respectively.

Most magnetic resonance spectroscopy studies, but not all (Endo et al. 2007), have also described abnormal cerebellar metabolite levels in ASD. Increased myoinositol and choline levels have been observed (Gabis et al. 2008), as well as decreased N-acetylaspartate and glutamate + glutamine (DeVito et al. 2007). These metabolites reflect different functions with myoinositol being essential for cell growth, choline being considered as a measure of membrane synthesis and turnover, N-acetylaspartate as a putative marker of neuronal viability, and glutamate + glutamine being related to excitatory pathways (see entry: ► [“Magnetic Resonance Spectroscopy”](#)). Finally, there is one study using single-photon emission computed tomography which did not find any significant differences in the cerebellum (Hashimoto et al. 2000).

Clinical Testing

Thus far, this review has focused on studies making direct assessments of the cerebellum via imaging or postmortem tissue. An indirect way to assess cerebellar abnormalities is through clinical evaluation. This is analogous to a neurologist determining the location of a stroke by examining the pattern of neurological impairments and clinical symptoms. Many different assessments have been used, and the choice depends on the system or brain region that is being tested. For the cerebellum, motor and nonmotor deficits are usually evaluated. Most, but not all, investigations probing the former have reported alterations suggestive of cerebellar pathology (Goldberg et al. 2000). Specifically, abnormalities have been reported in postural control (Dowell et al. 2009; Minshew et al. 2004), gait (Rinehart et al. 2006), eye movements (Takarae et al. 2004), and hand-eye coordination (Gowen and Miall 2005). Similarly, probing various cognitive functions, thought to depend on cerebellar integrity, have also yielded evidence of alterations in perceptual abilities (Davis et al. 2006) and learning (Mostofsky et al. 2000).

Future Directions

The investigations reviewed here support the integral role that the cerebellum plays in the pathophysiology of ASD. However, the exact involvement of this structure in the development of this disorder remains unclear despite 25 years of intensive research. From the information discussed above, the prevailing evidence in the field is that cerebellar abnormalities do not occur in isolation and do not appear to be specific to ASD. Nevertheless, it remains an important goal to continue investigating the contribution of the cerebellum to the clinical and biological abnormalities observed in ASD. Studies should be comprehensive, applying multimodal imaging and biological techniques, to increase both our general understanding of the cerebellum and the role it plays in autism.

See Also

- [Cerebellum](#)
- [Functional MRI](#)
- [Magnetic Resonance Imaging](#)
- [Magnetic Resonance Spectroscopy](#)
- [Purkinje Cells](#)

References and Reading

- Akshoomoff, N., Lord, C., Lincoln, A. J., Courchesne, R. Y., Carper, R. A., Townsend, J., et al. (2004). Outcome classification of preschool children with autism spectrum disorders using MRI brain measures. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43(3), 349–357.
- Allen, G., & Courchesne, E. (2003). Differential effects of developmental cerebellar abnormality on cognitive and motor functions in the cerebellum: An fMRI study of autism. *The American Journal of Psychiatry*, 160(2), 262–273.
- Allen, G., Muller, R. A., & Courchesne, E. (2004). Cerebellar function in autism: Functional magnetic resonance image activation during a simple motor task. *Biological Psychiatry*, 56(4), 269–278.
- Bailey, A., Luthert, P., Dean, A., Harding, B., Janota, I., Montgomery, M., et al. (1998). Clinicopathological study of autism. *Brain*, 121(5), 889–905.
- Bauman, M., & Kemper, T. L. (1985). Histoanatomic observations of the brain in early infantile autism. *Neurology*, 35(6), 866–874.

- Belmonte, M. K., Gomot, M., & Baron-Cohen, S. (2010). Visual attention in autism families: "Unaffected" sibs share atypical frontal activation. *Journal of Child Psychology and Psychiatry*, 51(3), 259–276.
- Bloss, C. S., & Courchesne, E. (2007). MRI neuroanatomy in young girls with autism: A preliminary study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46(4), 515–523.
- Braun, N. N., Reutiman, T. J., Lee, S., Folsom, T. D., & Fatemi, S. H. (2007). Expression of phosphodiesterase 4 is altered in the brains of subjects with autism. *Neuroreport*, 18(17), 1841–1844.
- Carper, R. A., & Courchesne, E. (2000). Inverse correlation between frontal lobe and cerebellum sizes in children with autism. *Brain*, 123(4), 836–844.
- Catani, M., Jones, D. K., Daly, E., Embiricos, N., Deeley, Q., Pugliese, L., et al. (2008). Altered cerebellar feedback projections in Asperger syndrome. *NeuroImage*, 41(4), 1184–1191.
- Cheng, Y., Chou, K. H., Chen, I. Y., Fan, Y. T., Decety, J., & Lin, C. P. (2010). Atypical development of white matter microstructure in adolescents with autism spectrum disorders. *NeuroImage*, 50(3), 873–882.
- Courchesne, E., Yeung-Courchesne, R., Press, G. A., Hesselink, J. R., & Jernigan, T. L. (1988). Hypoplasia of cerebellar vermal lobules VI and VII in autism. *The New England Journal of Medicine*, 318(21), 1349–1354.
- Courchesne, E., Townsend, J., Akshoomoff, N. A., Saitoh, O., Yeung-Courchesne, R., Lincoln, A. J., et al. (1994). Impairment in shifting attention in autistic and cerebellar patients. *Behavioral Neuroscience*, 108(5), 848–865.
- Courchesne, E., Karns, C. M., Davis, H. R., Ziccardi, R., Carper, R. A., Tigue, Z. D., et al. (2001). Unusual brain growth patterns in early life in patients with autistic disorder: An MRI study. *Neurology*, 57(2), 245–254.
- Critchley, H. D., Daly, E. M., Bullmore, E. T., Williams, S. C., Van Amelsvoort, T., Robertson, D. M., et al. (2000). The functional neuroanatomy of social behaviour: Changes in cerebral blood flow when people with autistic disorder process facial expressions. *Brain*, 123(11), 2203–2212.
- Davis, R. A., Bockbrader, M. A., Murphy, R. R., Hetrick, W. P., & O'Donnell, B. F. (2006). Subjective perceptual distortions and visual dysfunction in children with autism. *Journal of Autism and Developmental Disorders*, 36(2), 199–210.
- DeVito, T. J., Drost, D. J., Neufeld, R. W., Rajakumar, N., Pavlosky, W., Williamson, P., et al. (2007). Evidence for cortical dysfunction in autism: A proton magnetic resonance spectroscopic imaging study. *Biological Psychiatry*, 61(4), 465–473.
- Dowell, L. R., Mahone, E. M., & Mostofsky, S. H. (2009). Associations of postural knowledge and basic motor skill with dyspraxia in autism: Implication for abnormalities in distributed connectivity and motor learning. *Neuropsychology*, 23(5), 563–570.
- Endo, T., Shioiri, T., Kitamura, H., Kimura, T., Endo, S., Masuzawa, N., et al. (2007). Altered chemical metabolites in the amygdala-hippocampus region contribute to autistic symptoms of autism spectrum disorders. *Biological Psychiatry*, 62(9), 1030–1037.
- Fatemi, S. H., Halt, A. R., Stary, J. M., Realmuto, G. M., & Jalali-Mousavi, M. (2001). Reduction in anti-apoptotic protein Bcl-2 in autistic cerebellum. *Neuroreport*, 12(5), 929–933.
- Fatemi, S. H., Halt, A. R., Realmuto, G., Earle, J., Kist, D. A., Thuras, P., et al. (2002a). Purkinje cell size is reduced in cerebellum of patients with autism. *Cellular and Molecular Neurobiology*, 22(2), 171–175.
- Fatemi, S. H., Halt, A. R., Stary, J. M., Kanodia, R., Schulz, S. C., & Realmuto, G. R. (2002b). Glutamic acid decarboxylase 65 and 67 kDa proteins are reduced in autistic parietal and cerebellar cortices. *Biological Psychiatry*, 52(8), 805–810.
- Fatemi, S. H., Snow, A. V., Stary, J. M., Araghi-Niknam, M., Reutiman, T. J., Lee, S., et al. (2005). Reelin signaling is impaired in autism. *Biological Psychiatry*, 57(7), 777–787.
- Fatemi, S. H., Folsom, T. D., Reutiman, T. J., & Thuras, P. D. (2009). Expression of GABA(B) receptors is altered in brains of subjects with autism. *Cerebellum*, 8(1), 64–69.
- Frazier, T. W., & Hardan, A. Y. (2009). A meta-analysis of the corpus callosum in autism. *Biological Psychiatry*, 15(66), 935–941.
- Gabis, L., Wei, H., Azizian, A., DeVincent, C., Tudorica, A., Kesner-Baruch, Y., et al. (2008). 1H-magnetic resonance spectroscopy markers of cognitive and language ability in clinical subtypes of autism spectrum disorders. *Journal of Child Neurology*, 23(7), 766–774.
- Garber, H. J., & Ritvo, E. R. (1992). Magnetic resonance imaging of the posterior fossa in autistic adults. *The American Journal of Psychiatry*, 149(2), 245–247.
- Gilbert, S. J., Bird, G., Brindley, R., Frith, C. D., & Burgess, P. W. (2008). Atypical recruitment of medial prefrontal cortex in autism spectrum disorders: An fMRI study of two executive function tasks. *Neuropsychologia*, 46(9), 2281–2291.
- Goldberg, M. C., Landa, R., Lasker, A., Cooper, L., & Zee, D. S. (2000). Evidence of normal cerebellar control of the vestibulo-ocular reflex (VOR) in children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 30(6), 519–524.
- Gowen, E., & Miall, R. C. (2005). Behavioural aspects of cerebellar function in adults with Asperger syndrome. *Cerebellum*, 4(4), 279–289.
- Hallahan, B., Daly, E. M., McAlonan, G., Loth, E., Toal, F., O'Brien, F., et al. (2009). Brain morphometry volume in autistic spectrum disorder: A magnetic resonance imaging study of adults. *Psychological Medicine*, 39(2), 337–346.
- Hallett, M., Lebedowska, M. K., Thomas, S. L., Stanhope, S. J., Denckla, M. B., & Rumsey, J. (1993). Locomotion of autistic adults. *Archives of Neurology*, 50(12), 1304–1308.

- Hardan, A. Y., Minshew, N. J., Harenski, K., & Keshavan, M. S. (2001). Posterior fossa magnetic resonance imaging in autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40(6), 666–672.
- Hashimoto, T., Sasaki, M., Fukumizu, M., Hanaoka, S., Sugai, K., & Matsuda, H. (2000). Single-photon emission computed tomography of the brain in autism: Effect of the developmental level. *Pediatric Neurology*, 23(5), 416–420.
- Hazlett, H. C., Poe, M., Gerig, G., Smith, R. G., Provenzale, J., Ross, A., et al. (2005). Magnetic resonance imaging and head circumference study of brain size in autism: Birth through age 2 years. *Archives of General Psychiatry*, 62(12), 1366–1376.
- Ke, X., Hong, S., Tang, T., Zou, B., Li, H., Hang, Y., et al. (2008). Voxel-based morphometry study on brain structure in children with high-functioning autism. *Neuroreport*, 19(9), 921–925.
- Laurence, J. A., & Fatemi, S. H. (2005). Glial fibrillary acidic protein is elevated in superior frontal, parietal and cerebellar cortices of autistic subjects. *Cerebellum*, 4(3), 206–210.
- Lee, M., Martin-Ruiz, C., Graham, A., Court, J., Jaros, E., Perry, R., et al. (2002). Nicotinic receptor abnormalities in the cerebellar cortex in autism. *Brain*, 125(7), 1483–1495.
- Minshew, N. J., Luna, B., & Sweeney, J. A. (1999). Oculomotor evidence for neocortical systems but not cerebellar dysfunction in autism. *Neurology*, 52(5), 917–922.
- Minshew, N. J., Sung, K., Jones, B. L., & Furman, J. M. (2004). Underdevelopment of the postural control system in autism. *Neurology*, 63(11), 2056–2061.
- Mostofsky, S. H., Goldberg, M. C., Landa, R. J., & Denckla, M. B. (2000). Evidence for a deficit in procedural learning in children and adolescents with autism: Implications for cerebellar contribution. *Journal of International Neuropsychological Society*, 6(7), 752–759.
- Mostofsky, S. H., Powell, S. K., Simmonds, D. J., Goldberg, M. C., Caffo, B., & Pekar, J. J. (2009). Decreased connectivity and cerebellar activity in autism during motor task performance. *Brain*, 132(9), 2413–2425.
- Muller, R. A., Pierce, K., Ambrose, J. B., Allen, G., & Courchesne, E. (2001). Atypical patterns of cerebral motor activation in autism: A functional magnetic resonance study. *Biological Psychiatry*, 49(8), 665–676.
- Paakki, J. J., Rahko, J., Long, X., Moilanen, I., Tervonen, O., Nikkinen, J., et al. (2010). Alterations in regional homogeneity of resting-state brain activity in autism spectrum disorders. *Brain Research*, 1321, 169–179.
- Piven, J., Saliba, K., Bailey, J., & Arndt, S. (1997). An MRI study of autism: The cerebellum revisited. *Neurology*, 49(2), 546–551.
- Purcell, A. E., Jeon, O. H., Zimmerman, A. W., Blue, M. E., & Pevsner, J. (2001). Postmortem brain abnormalities of the glutamate neurotransmitter system in autism. *Neurology*, 57(9), 1618–1628.
- Rinehart, N. J., Tonge, B. J., Iansek, R., McGinley, J., Brereton, A. V., Enticott, P. G., et al. (2006). Gait function in newly diagnosed children with autism: Cerebellar and basal ganglia related motor disorder. *Developmental Medicine and Child Neurology*, 48(10), 819–824.
- Ritvo, E. R., Freeman, B. J., Scheibel, A. B., Duong, T., Robinson, H., Guthrie, D., et al. (1986). Lower Purkinje cell counts in the cerebella of four autistic subjects: Initial findings of the UCLA-NSAC Autopsy Research Report. *The American Journal of Psychiatry*, 143(7), 862–866.
- Sajdel-Sulkowska, E. M., Xu, M., & Koibuchi, N. (2009). Increase in cerebellar neurotrophin-3 and oxidative stress markers in autism. *Cerebellum*, 8(3), 366–372.
- Schmahmann, J. D., & Sherman, J. C. (1998). The cerebellar cognitive affective syndrome. *Brain*, 121, 561–579.
- Sheikh, A. M., Li, X., Wen, G., Tauqeer, Z., Brown, W. T., & Malik, M. (2010). Cathepsin D and apoptosis related proteins are elevated in the brain of autistic subjects. *Neuroscience*, 165(2), 363–370.
- Singer, H. S., Morris, C. M., Williams, P. N., Yoon, D. Y., Hong, J. J., & Zimmerman, A. W. (2006). Antibrain antibodies in children with autism and their unaffected siblings. *Journal of Neuroimmunology*, 178(1–2), 149–155.
- Takarae, Y., Minshew, N. J., Luna, B., Krisky, C. M., & Sweeney, J. A. (2004). Pursuit eye movement deficits in autism. *Brain*, 127(12), 2584–2594.
- Toal, F., Daly, E. M., Page, L., Deeley, Q., Hallahan, B., Bloemen, O., et al. (2010). Clinical and anatomical heterogeneity in autistic spectrum disorder: A structural MRI study. *Psychological Medicine*, 40(7), 1171–1181.
- Vargas, D. L., Nascimbene, C., Krishnan, C., Zimmerman, A. W., & Pardo, C. A. (2005). Neuroglial activation and neuroinflammation in the brain of patients with autism. *Annals of Neurology*, 57(1), 67–81.
- Whitney, E. R., Kemper, T. L., Bauman, M. L., Rosene, D. L., & Blatt, G. J. (2008). Cerebellar Purkinje cells are reduced in a subpopulation of autistic brains: A stereological experiment using calbindin-D28k. *Cerebellum*, 7(3), 406–416.
- Whitney, E. R., Kemper, T. L., Rosene, D. L., Bauman, M. L., & Blatt, G. J. (2009). Density of cerebellar basket and stellate cells in autism: Evidence for a late developmental loss of Purkinje cells. *Journal of Neuroscience Research*, 87(10), 2245–2254.
- Wills, S., Cabanlit, M., Bennett, J., Ashwood, P., Amaral, D. G., & Van de Water, J. (2009). Detection of autoantibodies to neural cells of the cerebellum in the plasma of subjects with autism spectrum disorders. *Brain, Behavior, and Immunity*, 23(1), 64–74.
- Yip, J., Soghomonian, J. J., & Blatt, G. J. (2007). Decreased GAD67 mRNA levels in cerebellar Purkinje cells in autism: Pathophysiological implications. *Acta Neuropathologica*, 113(5), 559–568.

Cerebellum

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Synonyms

[Little brain](#)

Definition

The cerebellum is a major division of the brain located in the posterior fossa behind the brainstem and underneath the cerebrum. It is attached or connected via three pairs of large fiber bundles (superior, middle, and inferior cerebellar peduncles) and is subdivided by two transverse fissures into three lobes (flocculonodular, anterior, and posterior lobes). Additionally, the cerebellum is divided into longitudinal zones which cut across the three lobes, resulting in two hemispheres (left and right) which flank a narrow midline zone called the vermis. Cerebellar circuitry has a uniform organization: inputs to the cerebellar cortex arrive through the middle/inferior peduncles. Neurons in the cerebellar cortex project to a series of deep cerebellar nuclei (dentate, interposed, emboliform, and globose nuclei) which provide outputs through the superior peduncle. Traditionally, the cerebellum's primary role is in motor control/learning including mediating equilibrium, postural control, and coordination/planning of voluntary movements. More recently, the cerebellum has been implicated in several cognitive functions including attention, learning, and language, as well as in some emotional functions such as regulating fear and pleasure responses. In general, damage to the midline (vermis) causes instability in posture and gait. Lateral damage (hemispheres) causes abnormalities in the limbs,

including decreased muscle tone, reduced reflexes, abnormal speech, tremor, and incoordination. Deficits in these functions have been reported in autism. However, studies of postural control in autism have linked impaired balance to reduced integration of multisensory information (joint-muscle position sense, visual, and vestibular) and not to cerebellar dysfunction. FMRI studies of motor movements have demonstrated deficits in cerebral connectivity rather than the cerebellum as the basis for coordination impairments in autism. The reduction of Purkinje cell number in the cerebellum has been widely found at autopsy brain examination in autism. Purkinje cells are inhibitory neurons which contain the neurotransmitter gamma-aminobutyric acid (GABA) to modulate neuronal transmission. Oculomotor studies in autism have also demonstrated subtle differences related to posterior fossa circuitry. Their implications for the pathophysiology of autism are unknown.

See Also

- ▶ [Cerebellar Abnormalities in Autism](#)
- ▶ [Motor Control](#)
- ▶ [Purkinje Cells](#)

References and Reading

- Manto, M. U., & Pandolfo, M. (Eds.). (2002). *The cerebellum and its disorders*. New York: Cambridge University Press.

Cerebral Cortex

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Synonyms

[Cortex](#); [Gray matter](#)

Definition

The cerebral cortex is the outermost sheet of neural tissue in the brain and plays a critical role in “higher level” cognitive functions, such as perception, attention, memory, language, executive functions, and consciousness.

Anatomical Organization

The most visually apparent features of the human brain are the folds (gyri and sulci), which are necessary products of packing a large cortical sheet into a limited space (i.e., the skull). As such, the folding pattern is most prominent in humans and primates. At the grossest level, the cortex is divided into four lobes: occipital, temporal, parietal, and frontal. At a microscopic scale, the cortex is organized horizontally into up to six layers and vertically into interconnected columns. Based on the thickness of the layers and the predominance of different neuron types, distinct regions of the cortex, called Brodmann’s areas, can be identified under a microscope. The cortex largely consists of cell bodies of neurons, their dendrites, and short range unmyelinated axons, hence the term “gray matter.” Regions of the cortex are connected to one another by myelinated axons that run through the “white matter” underneath the cortex.

Functional Organization

At the grossest functional level, the cortex can be divided into sensory cortex that takes in basic sensory information (auditory, visual, and somatosensory), motor cortex which plans and executes body motions, and association cortex that organizes input from sensory cortex into a unified perceptual world and performs the abstract thought and planning needed to guide the actions of the motor cortex.

Smaller units of function have been reliably localized in human brains using neuropsychological investigations of function after brain injury and in healthy populations using functional magnetic resonance imaging or positron emission tomography. For example, portions of the left

temporal (Wernicke’s area) and frontal cortex (Broca’s area) participate in language function.

See Also

- [Auditory Cortex](#)
- [Visual Cortex](#)

References and Reading

- Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (2000). *Principles of neural science* (4th ed., p. 324). New York: McGraw-Hill.
- Rakic, P. (1988). Specification of cerebral cortical areas. *Science*, 241(4862), 170–176.

Cerebral Gigantism

- [Sotos Syndrome and ASD](#)

Cerebral Palsy

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Synonyms

[CP](#); [Little’s disease](#); [Spastic diplegia](#); [Spastic paralysis](#)

Short Description or Definition

Cerebral palsy (CP) describes a group of disorders of the development of movement and posture, causing activity limitation attributed to nonprogressive disturbances that occur in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation,

cognition, communication, perception, and/or behavior and/or by a seizure disorder.

Categorization

Classification can be helpful in understanding the etiology, choosing a treatment, and even knowing the prognosis.

CP is classified by the type of motor impairment and its distribution:

1. Spastic CP:
 1. Spastic diplegia
 2. Spastic quadriplegia
 3. Spastic hemiplegia
2. Dyskinetic and dystonic CP
3. Hypotonic CP

Epidemiology

It is estimated that 2–3 per 1,000 live newborns have CP. Despite improved obstetric conditions, prevalence remains stable in the last decades, probably related to increase survival of premature infants. Prematurity increases the risk of CP. However, half of the cerebral palsy occurs in term infants.

Natural History, Prognostic Factors, and Outcomes

Natural History

By definition, cerebral palsy is secondary to a static lesion in the developing fetal or infant brain. However, the motor symptoms are progressive and they first appear between the ages of 6 months and 2 years, depending on the subtype of CP.

Functionally, with rehabilitation and physical care, these children can acquire new motor milestones during the first decade of life, remaining stable thereafter. A deterioration in walking ability in adulthood would be expected due to pain,

fatigue, or lack of adapted physical activity, but the few studies that have followed CP patients through adulthood report motor stability during this period for one third to half of the patients.

Prognostic Factors and Outcome

The most important prognostic factor is the type of CP. Most children with spastic diplegia or hemiplegia will acquire independent ambulation. The life span in this group is not shortened. However, children with spastic tetraplegia usually will not walk independently, being the prognosis for the dyskinetic group intermediate. Usually, if the child arrives to sit independently for the age of 2 years, he will arrive to walk alone.

For the quadriplegic group, survival is usually low. Most die from malnutrition, infections, or respiratory problems before they reach adolescence. Those having feeding tubes and inability to support their head, the median survival is 17 years.

The presence of mental retardation, a severe degree of disability, poor socialization, overprotection of parents, and denial of the problem of disability negatively affect a good prognosis for independent living in the adult with cerebral palsy.

Clinical Expression and Pathophysiology

Spastic Syndromes

The symptoms are those of the pyramidal syndrome: hypertonia of the affected body region, spasticity, hyperreflexia, and persistence of archaic reflexes. They are also present with difficulty in fine, rapid, and alternating movements. They are usually associated with some degree of dystonia.

Spastic Diplegia

Spastic diplegia is a clinical syndrome with a greater spasticity in legs than arms, seen most commonly in children born prematurely. It is primarily a disorder of developing white matter, and it is nearly always associated with neuropathological and neuroimaging findings of periventricular leukomalacia. Patients with spastic diplegia are

often identified during the first 6–12 months of life with signs of delayed motor development. Associated symptoms may include strabismus, orthopedic deformities, and oromotor dysfunction. There may be some degree of cognitive dysfunction.

Spastic Quadriplegia

Spastic quadriplegia is presented with bilateral spasticity affecting all extremities, with significant limitations in both mobility and hand use. Associated deficits may be more severe, including intellectual disability, seizures, orthopedic deformities including scoliosis and hip dislocation, and visual impairment. Spastic quadriplegia is the result of a broader range of pathological insults, including genetic and developmental brain malformations, severe periventricular leukomalacia, pre- and postnatal infections, asphyxia, and trauma. As with other cerebral palsy syndromes, low birth weight, prematurity, and complicated neonatal course are important risk factors. Delayed motor development in the first year is usually more prominent than in spastic diplegia.

Spastic Hemiplegia

Spastic hemiplegia represents unilateral spasticity, excluding the face. It often affects children at term. It is produced by damage to one hemisphere, such as prenatal strokes or head trauma. Independent ambulation and normal intelligence are commonly seen. There is no greater language impairment if the dominant hemisphere is affected. There is an increased risk of seizures, growth asymmetry, and sensory impairment of the hemiplegic side. Diagnosis is usually evoked by the end of the first year of life.

Dyskinetic-Dystonic

In children with extrapyramidal syndromes, clinical involvement is characteristically greater in the arms than the legs. Extrapyramidal syndromes are often associated with a marked reduction in speech production, but the child may have relatively preserved intelligence. There is usually involvement of basal ganglia (BG): selective necrosis of neurons in the BG, thalamus, reticular formation, and cerebellum. It is typical of term

infants with perinatal injuries: hypoxic-ischemic encephalopathy, hyperbilirubinemia, etc.

It is usually presented with lethargy, hypotonia, and multisystem involvement. Then psychomotor retardation and hypotonia occur, whereas abnormal movements may appear much later, at about 2 years of age.

Hypotonia and Ataxia

These children present with hypotonia with delayed motor milestones. These patients are distinguished by the preservation of strength and reflexes, suggesting a disorder of the upper motor neurons. This is a heterogeneous group of disorders.

Associated Impairments

Although characterized by their motor dysfunction, children with cerebral palsy frequently have other associated impairments.

Intellectual Disability (ID) Present in 50–75% of patients with CP. Usually more severe in patients with spastic quadriplegia.

Epilepsy Present in 30–50% of cases. More frequent if the lesion affects the cerebral cortex: usually in spastic hemiplegia and quadriplegia. In this group, there is a greater prevalence of ID.

Visual Disturbances Strabismus (50%), retinopathy (10%), cortical deficit (10%), and ocular motility disorders.

Digestive Disorders Malnutrition, dysphagia, gastroesophageal reflux, dental anomalies, and constipation.

Orthopedic Disorders Hip subluxation and osteopenia.

Urinary Disorders Incontinence, urgency, enuresis, detrusor dyssynergia, bladder hypertonia, etc.

Evaluation and Differential Diagnosis

Diagnosis usually requires several consecutive explorations: spasticity does not usually appear

until 6 months of age, dyskinetic movements at 18 months, and ataxia at age 2 years.

The initial symptoms are delayed acquisition of motor milestones, altered tone (hyper- or hypotonia), and persistence of archaic reflexes.

Investigations

MRI

- It is recommended in all cases of suspected CP.
- Ninety percent of children with CP have an altered MRI.
- It helps to know the time and etiology of the CP.

Metabolic Investigations

Less than 5% of the cases are secondary to metabolic disease.

Metabolic tests are only indicated in cases where the history is not typical, for example, no typical MRI, family history of consanguinity, and multiorganic symptoms.

Other Investigations

- EEG if suspected seizures
- Ophthalmologic examination
- ENT evaluation
- Orthopedic evaluation

Treatment

Effective management of cerebral palsy requires a team with medical and rehabilitation specialists to provide careful, coordinated treatment to maximize functional capabilities.

Management should aim to achieve maximal potential in all areas of development and to encourage independence. Realistic, functional goals must be set and periodically reevaluated by the rehabilitative team.

Rehabilitative goals will vary from patient to patient depending on the clinical situation, including ease of care, prevention of orthopedic deformity, or facilitating function.

A range of pharmacological agents are used to treat spasticity:

- For localized or segmental spasticity, recommendations support the use of intramuscular botulinum toxin A.
- For generalized spasticity, oral diazepam and tizanidine should be considered for short-term treatment.
- Surgical procedures are common for orthopedic deformities that arise in spastic patients. These operations have advanced from solo, sequential procedures to simultaneous, collective procedures including both soft tissue and bone.
- For children with severe cerebral palsy, refractory to standard interventions, neurosurgical procedures including intrathecal baclofen, selective dorsal rhizotomy, and deep brain stimulation should be considered.

Physical, occupational, and speech therapies are employed as initial therapies or used in conjunction with medical and surgical treatments, focusing on improving the strength and motion of affected muscles. Occupational and physical therapy play a fundamental role in children. Techniques serve to lessen the effects of inhibitory reflexes, to facilitate the acquisition of gross and fine motor skills, and to encourage language and the promotion of confidence and self-esteem.

See Also

- [Chronic Dyskinesia](#)
- [Developmental Coordination Disorder](#)
- [Hypotonia](#)

References and Reading

- A healthdirect Australia health information service. 2017 Summaries of systematic reviews of the evidence for the effectiveness of treatments for cerebral palsy. http://www.healthinsite.gov.au/topics/Systematic_Reviews_of_Treatments_for_Cerebral_Palsy
- Ashwal, S., Russman, B. S., Blasco, P. A., Miller, G., Sandler, A., Shevell, M., & Stevenson, R. (2004). Practice parameter: Diagnostic assessment of the child with cerebral palsy: Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. *Neurology*, 62, 851–863.

- Bax, M., Goldstein, M., Rosebaum, P., Leviton, A., Paneth, N., Dan, B., Jacobsson, B., Damiano, D., & Executive Committee for the Definition of Cerebral Palsy. (2005). Proposed definition and classification of cerebral palsy. *Developmental Medicine and Child Neurology*, 47, 571–576.
- Clark, S. L., & Hankins, G. D. (2003). Temporal and demographic trends in cerebral palsy- fact and fiction. *American Journal of Obstetrics and Gynecology*, 18, 628–633.
- Fennell, E. B., & Dikel, T. N. (2001). Cognitive and neuropsychological functioning in children with cerebral palsy. *Journal of Child Neurology*, 16, 58–63.
- Grether, J. K., Cummins, S. K., & Nelson, K. B. (1992). The California Cerebral Palsy Project. *Paediatric and Perinatal Epidemiology*, 6, 339–351.
- Imrie, M. N., & Yaszay, B. (2010). Management of spinal deformity in cerebral palsy. *Orthopedic Clinics of North America*, 41(4), 531–547.
- Jaw, T. S., Jong, Y. J., Sheu, R. S., et al. (1998). Etiology, timing of insult, and neuropathology of cerebral palsy evaluated with magnetic resonance imaging. *Journal of the Formosan Medical Association*, 97, 239–246.
- Krigger, K. W. (2006). Cerebral palsy: An overview. *American Family Physician*, 73(1), 91–100. www.aafp.org/afp/2006/0101/p91.html
- Kwong, K. L., Wong, S. N., & So, K. T. (1998). Epilepsy in children with cerebral palsy. *Pediatric Neurology*, 19, 31–36.
- Schenk-Rootlieb, A. J., van Nieuwenhuizen, O., van der Graaf, Y., et al. (1992). The prevalence of cerebral visual disturbance in children with cerebral palsy. *Developmental Medicine and Child Neurology*, 34, 473–480.
- Shapiro, B. K. (2004). Cerebral palsy: A reconceptualization of the spectrum. *Journal of Pediatrics*, 145(Suppl 2), S3–S7.
- Shaw, B. N. J. (1996). The respiratory consequences of neurological deficit. In P. B. Sullivan & L. Rosenbloom (Eds.), *Feeding the disabled child, Clinics in developmental medicine* (Vol. 140, pp. 40–46). New York: Cambridge University Press.
- Sullivan, P. B., Lambert, B., Rose, M., et al. (2000). Prevalence and severity of feeding and nutritional problems in children with neurological impairment: Oxford Feeding Study. *Developmental Medicine and Child Neurology*, 42, 674–680.
- Surveillance of Cerebral Palsy in Europe (SCPE). (2002). Prevalence and characteristics of children with cerebral palsy in Europe. *Developmental Medicine and Child Neurology*, 44, 633–640.
- Taylor, F., & National Institute of Neurological Disorders and Stroke (U.S.), Office of Science and Health Reports. (2001). *Cerebral palsy: Hope through research*. Bethesda: The Institute. Accessed online 28 Sept 2005. http://www.ninds.nih.gov/disorders/cerebral_palsy/detail_cerebral_palsy.htm
- Uvebrant, P., & Carlsson, G. (1994). Speech in children with cerebral palsy. *Acta Paediatrica*, 83, 779.
- Valencia, F. G. (2010). Management of hip deformities in cerebral palsy. *Orthopedic Clinics of North America*, 41(4), 549–559.
- Wu, Y. W., Croen, L. A., Shah, S. J., Newman, T. B., & Najjar, D. V. (2006). Cerebral palsy in a term population: Risk factors and neuroimaging findings. *Pediatrics*, 118, 60–67.

Cerebroatrophic Hyperammonemia

► Rett Syndrome

Cerebrospinal Fluid

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Synonyms

CSF; Spinal fluid

Definition

Cerebrospinal Fluid (CSF) is a clear, colorless liquid with the consistency of water that fills the ventricular system and subarachnoid spaces around the brain and spinal cord. It is produced primarily by the ependymal cells in the choroid plexus and is absorbed via vesicular transport in the arachnoid villi. The production and absorption of CSF are continuous processes that normally occur at equal rates. These processes lead to the complete replacement of the total volume of CSF approximately three times a day. The circulation of CSF is also continuous. The direction of flow is from the lateral ventricles through the cerebral

aqueduct, out either the foramina of Luschka or the foramen of Magendie, and downward posterior to the spinal cord. It then flows upward, anterior to the spinal cord and over the cerebral cortex.

CSF Composition

The CSF is sampled via lumbar puncture and is assayed to provide information relevant to diagnosis, pathophysiology, and treatment. A blood sample is drawn at the same time as the lumbar puncture in order to compare CSF levels with plasma levels of the elements below. The norms for the following can vary with age from premature infant to adult:

1. Osmolality and solute concentrations – CSF is iso-osmolar to blood plasma with normal CSF osmolality being 289 mOSM/L. The concentrations of sodium, magnesium, and bicarbonate are similar to plasma; however, the concentrations of potassium, calcium, and amino acids are lower in CSF than in plasma.
2. Cells – CSF is rather acellular with the normal density of white blood cells being less than five per high-powered field. Red blood cells are not normally present in the CSF.
3. Glucose – The normal concentration of glucose in CSF is 45–80 mg/dL, approximately two-thirds of the level of the normal serum glucose.
4. Protein – There is a rostral to caudal concentration of protein within the nervous system. The normal concentration of protein in CSF is 15–50 mg/dL.

Additional specific assays are performed depending on the differential diagnosis.

CSF Functions

1. Physical support – The brain and spinal cord essentially “float” in the CSF within the skull and spinal column.
2. Protection – CSF prevents the brain from colliding with the bony skull in cases of head injury. Additionally, the volume of CSF can redistribute in order to maintain normal intracranial pressure when volume changes occur in the other intracranial contents.

3. Metabolism – CSF aids in the excretion, elimination, and transport of centrally acting hormones and brain metabolites.

Relevance of CSF to Autism

A lumbar puncture is not a part of the standard workup or evaluation of children with ASD. Studies of CSF have been pursued in ASD as part of research seeking evidence related to various theories about the pathophysiology of autism. It is important to remember that CSF levels are not necessarily representative of brain levels and certainly not of regional or localized brain levels.

1. Neurotransmitters – There are scattered reports of altered neurotransmitter levels and function, including levels of tetrahydrobiopterin (sapropterin), serotonin, norepinephrine, and dopamine. While pervasive evidence of neurotransmitter abnormalities is lacking, alterations or nutritional deficiencies important for neurotransmitter formation and function (e.g., folate) may account for a very small subset of individuals with ASD (Frye 2010).
2. Mitochondrial disease – Elevated CSF lactate may be an important biomarker for individuals with ASD who have an underlying mitochondrial disease. It is unclear whether mitochondrial dysfunction contributes to the pathogenesis of ASD, or whether this is an epiphenomenon (Palmieri and Persico 2010).
3. Inflammatory markers in CSF – There are limited reports of elevated inflammatory markers in the CSF in individuals with ASD. While immune-based therapy for autism has received recent attention, there is a lack of control data to determine the specificity of this finding (Zimmerman et al. 2005).

References and Reading

- Fishman, R. A. (2005). Lumbar puncture and cerebrospinal fluid examination. In L. P. Rowland (Ed.), *Merritt's neurology* (Vol. 11, pp. 123–126). Philadelphia: Lippincott, Williams and Wilkins.
- Frye, R. E. (2010). Central tetrahydrobiopterin concentration in neurodevelopmental disorders. *Frontiers in Neuroscience*, 4, 52.

- Michaelson, D. J. (2006). Spinal fluid examination. In K. F. Swaiman, S. Ashwal, & D. M. Ferriero (Eds.), *Pediatric neurology, principles and practice IV* (Vol. 1, pp. 153–165). Philadelphia: Mosby Elsevier.
- Palmieri, L., & Persico, A. M. (2010). Mitochondrial dysfunction in autism spectrum disorders: Cause or effect? *Biochimica et Biophysica Acta*, 1797, 1130–1137.
- Rossignol, D. A., & Frye, R. E. (2010). Mitochondrial dysfunction in autism spectrum disorders: A systematic review and meta-analysis. *Molecular Psychiatry*, 17, 290–314. Online before print Jan 2011.
- Zimmerman, A. W., Jyonuchi, H., Comi, A. M., Connors, S. L., Milstien, S., Varsou, A., et al. (2005). Cerebrospinal fluid and serum markers of inflammation in autism. *Pediatric Neurology*, 33, 195–201.

Cerebrospinal Fluid 5-Hydroxyindoleacetic Acid

► CSF 5-HIAA

Cerebrospinal Fluid Homovanillic Acid

► CSF HVA

Certified Rehabilitation Counselor

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Synonyms

Canadian Certified Rehabilitation Counselor (CCRC); Certified Rehabilitation Counselor (CRC)

Definition

The Commission on Rehabilitation Counselor Certification (CRCC) sets the standard for quality rehabilitation counseling services in the USA and Canada. It is an independent, not-for-profit

organization. It is this agency that gives the certified designation to a rehabilitation counselor. The CRCC is accredited by the National Commission for Certifying Agencies (NCCA).

According to the Commission on Rehabilitation Counselor Certification (CRCC) agency, this certification designation indicates a “higher level of specialized education and training, a thorough understanding of key competency standards based on current practices in the field, adherence to the Code of Professional Ethics for Rehabilitation Counselors, and an ongoing commitment to continuing education.”

The CRCC mandates that to receive this designation a person must be of good moral character, meet acceptable standards of quality of practice, and have the requisite education and professional background. There are stringent eligibility requirements requiring a minimum of a Masters degree in Counseling or Rehabilitation Counseling together with specified work experience qualifications. The person must take and achieve a passing score on the CRC examination and renew their certification every 5 years via at least 100 h of continuing education or re-examination.

With one exception, Masters and Doctoral degree candidates must have received their education from a program accredited by the Counsel on Rehabilitation Education (CORE) or from a college or university accredited by the Council for Higher Education Accreditation (CHEA). CORE accredits graduate programs which provide academic preparation for a variety of professional rehabilitation counseling positions. CHEA is the largest institutional higher education membership organization in the USA. It is governed by a 20-person board of college and university presidents, institutional representatives, and public members.

If a candidate receives a Masters in Rehabilitation Counseling from a non-CORE program, then they must complete a rehabilitation counseling internship of 600 clock hours supervised by a CRC plus 12 months of acceptable employment experience supervised by a CRC, or 24 months of acceptable employment experience including 12 months supervised by a CRC.

Certification, unlike state licensure, is a voluntary process and is not government regulated. It is

not mandated by any state or federal laws; however, eligibility to sit for the certification exam is federally mandated if a person wishes to work in a state or federal vocational rehabilitation system.

See Also

- [American Congress of Rehabilitation Medicine](#)

References and Reading

<http://innerbody.com/careers-in-health/how-to-become-a-certified-rehab-counselor.html>
http://www.chea.org/pdf/chea_glance_2006.pdf
<http://www.core-rehab.org>
<http://www.crcrcertification.com/>

Certified Rehabilitation Counselor (CRC)

- [Certified Rehabilitation Counselor](#)

CET

- [Cognitive Enhancement Therapy](#)

Chaining

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Definition

Chaining refers to a variety of procedures for teaching behavior chains. A behavior chain is a series of responses in which each step serves both as a reinforcer for the previous step and as a discriminative stimulus for the next step (e.g., Cooper et al. 2007). The reinforcer delivered at the end of the chain maintains all of the previous responses in the chain.

It is important to teach behavior chains for complex sequences of responses that must be maintained at independent levels. Chaining procedures are used to teach many multistep skills, including self-help and daily living skills. The most common variations of chaining are forward and backward chaining. Task analysis is an essential component of chaining. The determination of steps in a chain that will be taught sequentially is complex and must be done competently.

In forward chaining, the sequence of actions is taught in temporal order. The learner is prompted and taught to perform the first step in the chain; the trainer completes the remaining steps. When the learner masters the first step, he or she is taught to do the first two steps. This continues until the entire chain is taught in sequence independently. Recently, forward chaining has been used to teach specific sequences of component skills in playing a game of basketball (Lambert et al. 2016).

A variation of forward chaining is total task chaining, which is also referred to as whole task or total task presentation. In this variation, the learner receives instruction in every step of the chain in every session. The trainer provides assistance for every step on which it is needed, and training continues until all steps are performed to criterion. Recently, total task chaining has been used to teach hygiene skills to young females with autism spectrum disorder (Veazey et al. 2016).

In backward chaining, the sequence is taught in reverse order, from the completion of the task to the start of the task. At the initiation of training, the trainer completes all but the last step of a chain, which is performed by the learner. Upon completion of this last step, the learner receives reinforcement. When competence is achieved on this final step, the trainer then does all but the last two steps of the chain. To receive reinforcement, the learner must complete the last two steps of the chain. This sequence is continued until the learner completes all steps of the chain independently. Backward chains have been utilized to teach a variety of skills. Recently, backward chaining was utilized to effectively increase functional leisure engagement in children with autism (Edwards et al. 2017). A backward chain is sometimes taught in a modified way, known as leaps ahead. In this variation, some steps may be

skipped if there is sufficient evidence that the learner possesses those components. Allowing skipping of steps increases the efficiency of instruction.

Using a limited hold can target the speed of responding within a chain. In a behavior chain with a limited hold, the sequence of responses must be performed correctly and within a specified period of time. The use of a limited hold targets proficiency in addition to accuracy. This can be used to speed slower responders and to ensure that the individual can engage in the targeted responses in a normative duration.

All chaining procedures are associated with positive results and are effective in teaching skills. There may be individual differences among learners, and an assessment may yield a best choice for that person. Furthermore, it has been suggested that total task presentation may make sense for learners who are more disabled (Test et al. 1990) and who have good imitative repertoires. It may also be a good match for tasks that are not too complex (Miltenberger 2001) and for circumstances in which learners know the steps but need to master them sequentially.

See Also

- [Chaining](#)
- [Task Analysis](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Edwards, C. K., Landa, R. K., Frampton, S. E., & Shillingsburg, M. A. (2017). Increasing functional leisure engagement for children with autism using backward chaining. *Behavior Modification*, 42, 9. 0145445517699929.
- Lambert, J. M., Copeland, B. A., Karp, E. L., Finley, C. I., Houchins-Juarez, N. J., & Ledford, J. R. (2016). Chaining functional basketball sequences with embedded conditional discriminations in an adolescent with autism. *Behavior Analysis in Practice*, 9(3), 199–210.
- Miltenberger, R. G. (2001). *Behavior modification: Principles and procedures*. Belmont: Wadsworth Thomson Learning.
- Test, D. W., Spooner, F., Kevl, P. K., & Grossi, T. (1990). Teaching adolescents with severe disability to use the public telephone. *Behavior Modification*, 14, 157–171.
- Veazey, S. E., Valentino, A. L., Low, A. I., McElroy, A. R., & LeBlanc, L. A. (2016). Teaching feminine hygiene skills to young females with autism spectrum disorder and intellectual disability. *Behavior Analysis in Practice*, 9(2), 184–189.

Challenging Behavior

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Definition

Challenging behavior refers to certain behaviors that a person engages in which negatively affect his/her daily functioning. These behaviors are often recognized as being culturally abnormal and occur at such an intensity, frequency, or duration that the safety of the person and/or others is placed in jeopardy. Challenging behaviors may be related to social, academic, communicative, cognitive, vocational, or physical domains, may serve various functions, and should be examined systematically in order to identify these functions. If challenging behavior is to be decreased, it is important to implement functionally and empirically validated interventions. Common challenging behaviors are self-injurious behavior, aggression, property destruction, stereotypic or repetitive behaviors, and sexualized behaviors.

See Also

- [Conduct Disorder](#)
- [Target Behavior](#)

References and Reading

- Cooper, J., Heron, T., & Heward, W. (2007). *Applied behavior analysis* (2nd ed.). Hoboken: Pearson Education.
- Durand, V., & Carr, E. (1991). Functional communication training to reduce challenging behavior: Maintenance

- and application in new settings. *Journal of Applied Behavior Analysis*, 24, 251–264.
- Lindauer, S., Zarcone, J., Richmond, D., & Shroeder, S. (2002). A comparison of multiple reinforcement assessment to identify function or maladaptive behavior. *Journal of Applied Behavior Analysis*, 35, 299–303.
- Thomason, R., & Iwata, B. (2007). A comparison of outcomes from descriptive and functional analyses of problem behavior. *Journal of Applied Behavior Analysis*, 40, 333–338.

Change Detection

- [Adolescents with Autism Spectrum Disorder \(ASD\) Spontaneously Attending to Real-World Scenes: Use of a Change Blindness Paradigm](#)

CHARGE Association

- [CHARGE Syndrome](#)

CHARGE Syndrome

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Synonyms

[CHARGE association](#)

Definition

CHARGE syndrome is a rare genetic syndrome (prevalence of 1:3,000–1:12,000) characterized by a constellation of abnormalities, which may include but is not limited to the following: coloboma, or a hole-shaped malformation, of the eye, resulting in visual impairments; heart defects; atresia of the choanae, or blockage of the nasal passages; retardation of growth and development;

genital abnormalities; and ear abnormalities, such as a cup-shaped ear, and deafness (Nussbaum et al. 2007). The co-occurrence of these features was previously referred to as CHARGE association. However, with the identification of the gene responsible for the majority of cases, the term “syndrome” is now preferred (Nussbaum et al. 2007). Additional features of CHARGE syndrome include abnormalities of the cranial nerves leading to deafness, swallowing difficulties, and facial weakness; cleft palate; and fistulae, or abnormal conduits between the trachea and esophagus (Nussbaum et al.). Behavioral difficulties have also been described, such as hyperactivity and obsessive-compulsive behaviors (Nussbaum et al.).

CHARGE syndrome is one of the rare genetic syndromes that has been associated with autism (Filipek 2005). The first report of this association described three children with CHARGE syndrome, two of whom also had intellectual disability, and clinical features of autism, according to the Autism Diagnostic Interview-Revised, Childhood Autism Rating Scale, and the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* (Fernell et al. 1999). The prevalence of autism in CHARGE syndrome has been estimated to be between 15% and 50% (Grafodatskaya et al. 2010). However, the diagnosis of autism in CHARGE syndrome is complicated by the challenge of quantifying social and communication deficits in a syndrome characterized by visual and hearing impairments and, in some cases, intellectual disability as well (Grafodatskaya et al. 2010).

More than half of all individuals with CHARGE syndrome carry mutations in the gene, *chromodomain helicase DNA binding protein 7 (CHD7)*, which is located on chromosome 8q12. In most cases, this is a de novo, or new, mutation such that the recurrence risk of CHARGE syndrome is typically low, less than 5% if the mutation is not present in either parent (Nussbaum et al. 2007). Because the *CHD7* gene encodes a protein that is involved in altering the structure of chromosomes, it likely functions in an epigenetic manner, regulating the expression of genes that serve critical functions in early development. This accounts for the observation that not

all cases of CHARGE syndrome are due to mutations in *CHD7*, indicating that mutations in other genes can result in a similar clinical presentation. Moreover, as with other rare genetic syndromes associated with an increased risk of autism, studies of the genetic etiology of CHARGE syndrome may provide insight into the genetics of autism.

References and Reading

- Fernell, E., Olsson, V. A., Karlgren-Leitner, C., Norlin, B., Hagberg, B., & Gillberg, C. (1999). Autistic disorders in children with CHARGE association. *Developmental Medicine and Child Neurology*, 41(4), 270–272.
- Filipek, P. A. (2005). Medical aspects of autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 534–578). Hoboken: Wiley.
- Grafodatskaya, D., Chung, B., Szatmari, P., & Weksberg, R. (2010). Autism spectrum disorders and epigenetics. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(8), 794–809.
- Lalani, S. R., Safiullah, A. M., Fernbach, S. D., Harutyunyan, K. G., Thaller, C., Peterson, L. E., et al. (2006). Spectrum of CHD7 mutations in 110 individuals with CHARGE syndrome and genotype-phenotype correlation. *American Journal of Human Genetics*, 78(2), 303–314.
- Nussbaum, R. L., McInnes, R. R., & Willard, H. F. (2007). *Nussbaum: Thompson & Thompson genetics in medicine* (7th ed.). Philadelphia: Saunders Elsevier.
- Visser, L. E., van Ravenswaaij, C. M., Admiraal, R., Hurst, J. A., de Vries, B. B., Janssen, I. M., et al. (2004). Mutations in a new member of the chromodomain gene family cause CHARGE syndrome. *Nature Genetics*, 36(9), 955–957.

Charter School

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Definition

In the United States and other countries, a charter school is a publicly funded but privately run

school. These schools differ from traditional public schools because their existence is contingent upon meeting certain outcomes. When granted a charter, the school sets certain student achievement goals that must be met at the time of charter renewal. The charters are renewed every 3–5 years, depending on the district or state. School leaders at charters have increased autonomy to meet these goals. When the number of applicants for a charter school exceeds available seats, students are admitted based on a lottery.

Charter schools can be primary or secondary schools. They do not charge admission and typically are exempt from some requirements of public (state-run) schools. Students in these schools do participate in state-mandated testing. An increasing number of such schools serve children with special needs including autism.

Reference and Reading

- Lubienski, C. A., & Weitzel, P. C. (Eds.). (2010). *The charter school experiment*. Cambridge, MA: Harvard Educational Press.

CHAT

► [Modified Checklist for Autism in Toddlers \(M-CHAT\)](#)

CHD8

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Synonyms

[AUTS18](#), [Duplin](#), [HELSNF1](#)

Structure

Chromodomain-helicase-DNA-binding protein 8 (CHD8) encodes an ATP-dependent DNA

helicase located on chromosome 14q11.2 in humans. Two human isoforms of CHD8 are produced by alternative splicing, with the canonical sequence spanning 2581 amino acids and 39 exons. CHD8, originally termed Duplin upon its initial discovery, (Sakamoto et al. 2000) is a member of the chromodomain-helicase-DNA-binding protein family. The CHD family is characterized by the SNF-2-like ATPase and two chromodomains (chromatin organization modifier) (Marfella and Imbalzano 2007). Within this protein family, nine genes are organized into three subfamilies according to the presence or absence of specific functional domains. CHD8 bears most functional and structural similarity to CHD7 and CHD9, all of which exhibit a DNA-binding domain as well as a BRK domain located at the C-terminus. Conservation of CHD8 is evident across a diverse array of species including chimpanzees, rhesus monkeys, dogs, cows, mice, rats, zebrafish, and frogs, though 140 organisms have genes orthologous to the human CHD8 gene.

Function

Through its many interactions, CHD8 has been shown to serve as a global regulator of chromatin architecture and gene expression. The discovery of CHD8, initially described as an axis duplication inhibitor, was made through the observation of its novel interaction with the canonical Wnt/ β -catenin pathway (Sakamoto et al. 2000). This pathway, which is implicated in embryonic development and cellular proliferation among other biological processes, is negatively regulated by CHD8 through its binding β -catenin.

Yet the implications for CHD8 in early embryogenesis extend as well to its binding interaction with tumor suppressor protein and transcription factor p53, where CHD8 is believed to recruit histone H1, an essential component in chromatin packing and structural integrity, in order to stave off p53 transactivation activity and ultimately apoptosis (Nishiyama et al. 2009). The hypotheses as to exactly how CHD8 regulates transcription extend to evidence of its interaction with CTCF, an insulator binding protein that blocks the interaction between promoters and

enhancers. In the absence of sufficient CHD8, effects on CpG methylation and histone acetylation were observed around CTCF-binding sites, possibly interfering with proper insulation activity, chromatin structure and integrity, as well as gene regulation and epigenetic control (Ishihara et al. 2006).

Indeed, the functional diversity of CHD8 has been shown through alternate proposals that CHD8 may modulate transcription via histone H3 lysine 4 (H3K4me3) modification (Yuan et al. 2007; Rodriguez-Paredes et al. 2009; Sugathan et al. 2014; Cotney et al. 2015), associations with RNA polymerase II (Rodriguez-Paredes et al. 2009), or remodeling general chromatin architecture by other means.

Path of Physiology

CHD8 is among a small list of genes attributed the highest confidence for its documented contribution to autism spectrum disorder (ASD) risk. This level of confidence in its candidacy as an ASD risk gene is derived from the evidence of 26 de novo mutations or disruptions in the gene, identified through both targeted re-sequencing and whole exome sequencing, among individuals diagnosed with ASD, (Bernier et al. 2014; De Rubeis et al. 2014; Iossifov et al. 2012; McCarthy et al. 2014; O’Roak et al. 2012a, b, 2014; Talkowski et al. 2012) making CHD8 one of the most mutated genes in simplex ASD (Barnard et al. 2015).

Among these mutations are a number of missense variants, many of them predicted in silico to be damaging to protein function, and several implicit loss-of-function (LoF) or likely gene-disrupting (LGD) mutations that result in a premature truncation of the gene, shift in the protein reading frame, or canonical splice site variant. Recorded disruptions of the gene also include chromosomal abnormalities such as the 14q11.1 breakpoint of a de novo balanced translocation of 3q25.31 and 14q11.1 [46 XX, t_(3;14) (q25.31; q11.2)dn] (Talkowski et al. 2012).

These genetic aberrations in CHD8 further seem to exhibit some specificity at the phenotypic level, even among individuals diagnosed with

ASD. Identifying genetic specificity within ASD may ultimately accelerate the search for biologically based diagnostic tools and individualized treatment regimens for patients who exhibit syndromic subtypes of ASD. Individuals with CHD8 mutations exhibit significantly larger head circumferences, known as macrocephaly (O’Roak et al. 2012a, b; Talkowski et al. 2012; Bernier et al. 2014), gastrointestinal problems (Bernier et al. 2014), and often similarly dysmorphic facial features (Bernier et al. 2014; Talkowski et al. 2012). Indeed, one hypothesis has suggested that the observation of macrocephaly in patients with CHD8 mutations may stem from the absence of necessary binding interactions of CHD8 with transcription factors that control cell cycle regulation (Subtil-Rodriguez et al. 2014) or from interference in proper cell cycle progression due to insufficient *Chd8* protein levels (Rodriguez-Paredes et al. 2009). This support for a distinct CHD8 subtype of ASD that arises from disruptions in cellular proliferation and early developmental pathways has been recapitulated among animal models using both mice and zebrafish (Nishiyama et al. 2009; Sakamoto et al. 2000; Bernier et al. 2014).

Several studies have recently sought to explore the impact of heterozygous LoF mutations in CHD8 by investigating the downstream effects of CHD8 knockdown (Sugathan et al. 2014; Cotney et al. 2015; Wilkinson et al. 2015). While the exact mechanisms remain unclear, these studies have replicated the finding that both direct and indirect targets of CHD8 are strongly enriched for genes already known to be associated with ASD risk. Through binding interactions and indirect downregulation, CHD8 has been found to play a role in critical brain-based and neuronal development pathways that control synapse formation, neuron differentiation, and axon guidance as well as chromatin modification and transcriptional regulation.

In summary, evidence from the extensive study of CHD8 since its discovery in 2000 suggests that CHD8 may play a critical role in highly conserved evolutionary pathways. Given the evidence, it has

been proposed that CHD8 likely serves as a master regulator for gene transcription and expression and that the gene sets regulated by CHD8 may in turn tightly control the proper development of the human brain and neuronal development during a key prenatal window (Bernier et al. 2014). Mutations that drastically alter the levels of CHD8 protein likely disrupt these pathways and ultimately give rise to increased ASD risk.

References and Reading

- Barnard, R. A., Pomaville, M. B., & O’Roak, B. J. (2015). Mutations and modeling of the chromatin remodeler CHD8 define an emerging autism etiology. *Frontiers in Neuroscience*, 9, 477.
- Bernier, R., Golzio, C., Xiong, B., Stessman, H. A., Coe, B. P., Penn, O., et al. (2014). Disruptive CHD8 mutations define a subtype of autism early in development. *Cell*, 158(2), 263–276.
- Cotney, J., Muhle, R. A., Sanders, S. J., Liu, L., Willsey, A. J., Niu, W., et al. (2015). The autism-associated chromatin modifier CHD8 regulates other autism risk genes during human neurodevelopment. *Nature Communications*, 6, 6404.
- De Rubeis, S., He, X., Goldberg, A. P., Poultney, C. S., Samocha, K., Cicek, A. E., et al. (2014). Synaptic, transcriptional and chromatin genes disrupted in autism. *Nature*, 515(7526), 209–215.
- Iossifov, I., Ronemus, M., Levy, D., Wang, Z., Hakker, I., Rosenbaum, J., et al. (2012). De novo gene disruptions in children on the autistic spectrum. *Neuron*, 74(2), 285–299.
- Ishihara, K., Oshimura, M., & Nakao, M. (2006). CTCF-dependent chromatin insulator is linked to epigenetic remodeling. *Molecular Cell*, 23(5), 733–742.
- Marfella, C. G., & Imbalzano, A. N. (2007). The Chd family of chromatin remodelers. *Mutation Research*, 618(1–2), 30–40.
- McCarthy, S. E., Gillis, J., Kramer, M., Lihm, J., Yoon, S., Bernstein, Y., et al. (2014). De novo mutations in schizophrenia implicate chromatin remodeling and support a genetic overlap with autism and intellectual disability. *Molecular Psychiatry*, 19(6), 652–658.
- Nishiyama, M., Oshikawa, K., Tsukada, Y., Nakagawa, T., Iemura, S., Natsume, T., et al. (2009). CHD8 suppresses p53-mediated apoptosis through histone H1 recruitment during early embryogenesis. *Nature Cell Biology*, 11(2), 172–182.
- O’Roak, B. J., Vives, L., Fu, W., Egertson, J. D., Stanaway, I. B., Phelps, I. G., et al. (2012a). Multiplex targeted sequencing identifies recurrently mutated genes in autism spectrum disorders. *Science*, 338(6114), 1619–1622.

- O’Roak, B. J., Vives, L., Girirajan, S., Karakoc, E., Krumm, N., Coe, B. P., et al. (2012b). Sporadic autism exomes reveal a highly interconnected protein network of de novo mutations. *Nature*, 485(7397), 246–250.
- O’Roak, B. J., Stessman, H. A., Boyle, E. A., Witherspoon, K. T., Martin, B., Lee, C., et al. (2014). Recurrent de novo mutations implicate novel genes underlying simplex autism risk. *Nature Communications*, 5, 5595.
- Rodriguez-Paredes, M., Ceballos-Chavez, M., Esteller, M., Garcia-Dominguez, M., & Reyes, J. C. (2009). The chromatin remodeling factor CHD8 interacts with elongating RNA polymerase II and controls expression of the cyclin E2 gene. *Nucleic Acids Research*, 37(8), 2449–2460.
- Sakamoto, I., Kishida, S., Fukui, A., Kishida, M., Yamamoto, H., Hino, S., et al. (2000). A novel beta-catenin-binding protein inhibits beta-catenin-dependent Tcf activation and axis formation. *The Journal of Biological Chemistry*, 275(42), 32871–32878.
- Subtil-Rodriguez, A., Vazquez-Chavez, E., Ceballos-Chavez, M., Rodriguez-Paredes, M., Martin-Subero, J. I., Esteller, M., et al. (2014). The chromatin remodeller CHD8 is required for E2F-dependent transcription activation of S-phase genes. *Nucleic Acids Research*, 42(4), 2185–2196.
- Sugathan, A., Biagioli, M., Golzio, C., Erdin, S., Blumenthal, I., Manavalan, P., et al. (2014). CHD8 regulates neurodevelopmental pathways associated with autism spectrum disorder in neural progenitors. *Proceedings of the National Academy of Sciences of the United States of America*, 111(42), E4468–E4477.
- Talkowski, M. E., Rosenfeld, J. A., Blumenthal, I., Pillalamarri, V., Chiang, C., Heilbut, A., et al. (2012). Sequencing chromosomal abnormalities reveals neurodevelopmental loci that confer risk across diagnostic boundaries. *Cell*, 149(3), 525–537.
- Wilkinson, B., Grepo, N., Thompson, B. L., Kim, J., Wang, K., Evgrafov, O. V., et al. (2015). The autism-associated gene chromodomain helicase DNA-binding protein 8 (CHD8) regulates noncoding RNAs and autism-related genes. *Translational Psychiatry*, 5, e568.
- Yuan, C. C., Zhao, X., Florens, L., Swanson, S. K., Washburn, M. P., & Hernandez, N. (2007). CHD8 associates with human Staf and contributes to efficient U6 RNA polymerase III transcription. *Molecular and Cellular Biology*, 27(24), 8729–8738.

Checklist for Autism in Toddlers

► [Modified Checklist for Autism in Toddlers \(M-CHAT\)](#)

Checklist for Autism in Toddlers (CHAT)

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Abbreviations

CHAT	Checklist for autism in toddlers
PDD-NOS	Pervasive developmental disorder-not otherwise specified
ASD	Autism spectrum disorder
PPV	Positive predictive value

Description

The CHecklist for Autism in Toddlers (CHAT; Baron-Cohen et al. 1992, 1996) is a screening tool designed to capture early signs of autism in 18-month-olds by inquiring about milestones related to early social and communicative development. The CHAT consists of nine parent report items and five child observation items completed by the child’s general physician or health visitor. The parent questions (part A) assess abnormal behaviors commonly associated with autism spectrum disorders (i.e., reduced social interest, social play, pretend play, protodeclarative pointing, and joint attention), as well as developmental behaviors that are more likely to be intact in children with autism (i.e., rough and tumble play, motor development, protoimperative pointing, and functional play). The second set of questions (part B) was created to supplement the parent’s report of the child’s behavior. A trained professional administers five items measuring pretend play, protodeclarative pointing (both initiating and responding to another person’s point), eye contact, and functional play. All questions are in a yes/no format and administration time is approximately 15 min.

A high-risk score is obtained if a child fails all five items addressing protodeclarative pointing, pretend play, and gaze monitoring across parent report and clinician observation. A medium-risk score results from failing both items on protodeclarative pointing. All other children are considered to be at low risk for autism. Additionally, a two-stage screening method is recommended in which a child screens positive on the original CHAT administration, as well as upon re-administration 1 month later in attempts to reduce the likelihood of false positive cases (Baron-Cohen et al. 2000).

Historical Background

The CHAT was developed in Great Britain by Baron-Cohen and colleagues as a way for primary care physicians or home visitor nurses to screen for autism in young children. It was the first screening tool to identify autism risk in 18-month-olds. The pilot version of the questionnaire included several parent report items for each of 10 areas of development. In efforts towards quicker administration, items in the imitation domain were dropped, as these behaviors were determined to not be reliably present among most 18-month-olds (more than 20% did not), resulting in the current nine areas of development. Subsequently, only the most frequently passed question for each domain was kept and the rest of the questions were dropped, resulting in the current version of one question for each of the nine areas.

In their initial study, Baron-Cohen et al. (1992) screened 50 randomly selected toddlers from a pediatric setting (low-risk) and 41 high-risk toddlers (younger siblings of children with autism). More than 80% of the randomly selected low-risk toddlers passed all items on the CHAT. Among the high-risk group, four toddlers failed at least two of five target ASD items and later had a diagnosis of ASD at follow-up. This first study, although a small sample, suggested its utility as an ASD screening instrument within a population that had been identified as being at-risk. In a subsequent validation study screening 16,000

children using the CHAT, Baron-Cohen et al. (1996) identified three critical content areas for identifying autism, which include pretend play (parent-report and observation), eye gaze (observation), and pointing (parent-report and observation), totaling five critical items. Twelve of the 16,000 children among the general population were identified as at-risk for autism; risk status was based on a two-stage screening approach in which the high-risk score cutoff of failing all five critical items was met both at the original administration of the CHAT as well as at retest approximately 1 month later. The two-stage method was adopted to help reduce false positive cases. Ten of these children received a diagnosis of autism and two had other developmental delays, suggesting that the measure had adequate utility for use in the general population. Follow-up diagnostic evaluations at 3½ years of age indicated stability of diagnosis. In a follow-up study of the 16,000 children at age 7 years, the effectiveness of one-stage screening was compared to two-stage screening (Baird et al. 2000); see section “Psychometric Data.” In an article summarizing published research on the CHAT, Baron-Cohen et al. (2000) recommended using two-stage screening so as to ensure that failing items on the first CHAT are significant developmental concerns rather than situational concerns on the day of administration (i.e., having a “bad day”) or milder developmental delays.

Several different scoring systems and versions have been developed since the original CHAT. Scambler et al. (2001) published data on a modified scoring system for the CHAT, called the **Denver criteria**. The Denver scoring criteria differed in that they included failing a parent-report item of pretend play or pointing to show an object, as well as clinical observation of pointing impairment.

Additionally, the **Modified Checklist for Autism in Toddlers (M-CHAT)**; Robins et al. (1999) is a parent-report screening measure that was adapted from the original CHAT in order to capture the whole spectrum of disorders, rather than just Autistic Disorder. It consists of 23-item “yes or no” questions. Preliminary results in a mixed low- and high-risk sample indicated promising psychometric properties (Robins et al. 2001)

and a large low-risk sample demonstrated utility in pediatric primary care (Chlebowski et al. 2013).

The latest revision is known as the **Modified Checklist for Autism in Toddlers, Revised with Follow-Up (M-CHAT-R/F)** (Robins et al. 2009, 2014). This version formalized the two-step screening approach, using the structured Follow-Up questions for children who score at risk. The parent questionnaire is slightly shorter than the M-CHAT, consisting of 20 items. Additional changes include removing three items that exhibited poor discriminant validity, re-ordering items to reduce affirmative response bias, adding examples describing target behaviors, and simplifying wording. Children who screen positive (total score ≥ 3) complete the Follow-Up; at-risk score at Follow-Up is ≥ 2 . The M-CHAT-R/F has been adapted for electronic administration (Campbell et al. 2017; Sturmer et al. 2016) and use of drawings to illustrate items; see www.mchatscreen.com for translations including illustrations. See ► “M-CHAT” entry for more details about this instrument and its psychometric data.

Data on the **CHAT-23**, a version of the CHAT applicable for Chinese children, was published by Wong et al. (2004). This version is a combination of both the M-CHAT and CHAT in that it consists of a Chinese translation of the 23-item M-CHAT (part A) plus the five clinical observation items from the CHAT (part B). Initial data on 18- and 24-month-olds identified seven critical items from part A, and four key items in part B. The fifth item in part B assessed general developmental ability (i.e., functional play), which is thought to develop normally in autism and was not included in the statistical analysis. Screen positives on part A include failing two of seven items determined to be critical in this translation or any six of the 23 items overall; screen positives on part B include failing at least two of the four key items. Based on their results, the authors suggest a two-stage algorithm for screening. This includes universal screening using part A, followed by part B screening only for those children who screen positive on part A. See section “[Psychometric Data](#)” for a summary of psychometrics.

Another version is the **Quantitative Checklist for Autism in Toddlers (Q-CHAT)** (Allison et al. 2008), which took the form of a 25-item parent-report scale in which responders quantify behaviors based on a 5-point Likert rating scale. Likert scale response items vary depending on the question and range from, for example, “always” to “never,” “many times a day” to “never,” “very easy” to “impossible,” etc. This allows for demonstration of reduced frequency of particular behaviors that children with an ASD might exhibit, rather than requiring parents to judge absolute absence of these behaviors. In addition to the three key items identified by Baron-Cohen et al. (1996), which are pretend play, eye gaze, and protodeclarative pointing, the Q-CHAT includes other domains, such as language development and repetitive behaviors. The Q-CHAT has been used to measure clinical comparisons, not just for early ASD detection. For example, in a sample of children born premature, the Q-CHAT was used to assess social-communication outcomes in conjunction with measures of sociodemographic factors and cognitive functioning (Wong et al. 2014). Results indicated higher Q-CHAT scores (i.e., greater social-communication difficulties) relative to norms; lower cognitive functioning and ethnic minority status was associated with higher Q-CHAT scores. Additionally, the Q-CHAT has been used to measure ASD traits and sex differences at age 18–24 months and compare to testosterone levels; results have shown prenatal testosterone levels but not postnatal testosterone levels, to be related to later ASD traits and sex differences (Auyeung et al. 2012). This measure has been translated into several languages, and cross-cultural validation studies have been conducted in clinical and unselected samples in Singapore (Magiati et al. 2015), Colombia (Gutiérrez-Ruiz et al. 2019), and Italy (Ruta et al. 2019a, b).

Psychometric Data

The entire screening sample for the initial low-risk CHAT validation included 16,000 children screened at age 18 months (Baron-Cohen et al.

1996), who were later followed up when the children turned 7 years old (Baird et al. 2000) in order to calculate complete psychometric data, which requires ascertainment of missed cases or false negatives. Based on their results, there were 50 cases of autism and 44 cases of PDD-NOS in the sample. The authors compared psychometric data of the CHAT when using one-stage screening versus two-stage screening (two administrations 1 month apart). Based on one-stage screening, 10 of the 50 autism cases were identified by the high-risk score, and an additional 9 cases were identified using the medium-risk score. This yielded a sensitivity of 0.20, specificity of 0.998, and positive predictive value (PPV) of 0.26 using the high-risk score, and sensitivity of 0.38, specificity of 0.98, and PPV of 0.05 for the medium-risk score. Of all 94 ASD cases, medium-risk scoring criteria identified 33 cases whereas high-risk cutoff scores captured 11 cases. The high-risk cutoffs demonstrated a sensitivity of 0.12, specificity of 0.998, and PPV of 0.29; medium-risk scores yielded a sensitivity of 0.35, specificity of 0.98, and PPV of 0.08. When using the two-stage screening in identifying cases of autistic disorder, PPV increased to 0.75 and 0.29 for the high-risk and medium-risk cutoffs, respectively. Specificity remained high, whereas sensitivity somewhat dropped to 0.18 and 0.20 for the high-risk and medium-risk cutoffs, respectively. For all ASD cases, there was a similar pattern with PPV again increasing to 0.83 and 0.59 based on the high-risk and medium-risk scores, respectively, specificity remaining high, and sensitivity somewhat decreasing to 0.11 and 0.21 for high-risk and medium-risk cutoffs, respectively. Overall, two-stage screening increases the CHAT's PPV, which increases the likelihood that a screen positive case will receive an ASD diagnosis; however, the false positive rate is greater in the two-stage approach compared to screening at a single time point, thus reducing the measure's sensitivity (Baird et al. 2000; Baron-Cohen et al. 2000). See Table 1 for a summary of psychometric data.

The Denver criteria (Scambler et al. 2001) were based on post hoc analysis as part of their study on the CHAT as a Level 2 screener. These scoring criteria were compared to original scoring

criteria on a sample of two- to three-year-old children with ASD ($n = 26$) and other developmental disorders (DD; $n = 18$) to determine how well the CHAT distinguishes between the two groups. The Denver scoring criteria yielded 0.85 sensitivity and 1.00 specificity, whereas the sensitivity dropped to 0.65 when using the original CHAT scoring criteria, with specificity remaining at 1.00. A subset of these children (ASD $n = 19$; DD $n = 11$) participated in a follow up study two years later to assess stability of diagnosis (Scambler et al. 2006). Original CHAT scoring at Time 1 correctly classified 83% of the sample at Time 2 (five missed cases of ASD); 93% of the sample was correctly identified at Time 2 based on the Denver scoring criteria of the CHAT at Time 1 (two missed cases of ASD). The CHAT's original scoring and Denver scoring have been assessed for utility in detecting autism in Fragile X syndrome cases (Scambler et al. 2007). On a sample of 17 children (mean age = 34 months), results yielded sensitivity of 0.50 and specificity of 1.00 using CHAT scoring criteria and sensitivity of 0.75 and specificity of 0.92 using the Denver scoring criteria.

Two-stage screening with the CHAT was evaluated in a population-based cross-sectional study in Ireland (VanDenHeuvel et al. 2006). At the initial screen, 29 of 2117 toddlers demonstrated medium or high risk at 18-month developmental checkup, of which at secondary screening 7 continued to screen positive, 12 exhibited low risk, and 10 did not participate. The seven children who screened positive and five of the children who declined secondary screening completed a clinical assessment ($n = 12$), and seven children were diagnosed with ASD, yielding a prevalence of 33.1 per 10,000, 95% CI [12.3, 68.0]. Based on methodological issues, additional psychometric data could not be assessed and is not included in Table 1.

The utility of the CHAT as a tool to detect autism in children younger than 3 years was also investigated in a Swedish population (Höglund-Carlsson et al. 2010). Nurses were instructed to administer the CHAT if the child was identified to be at-risk based on developmental surveillance; those who screened positive on the CHAT were

Checklist for Autism in Toddlers (CHAT), Table 1 Psychometric data for the CHAT

Study	Sample	Sensitivity	Specificity	PPV
Baird et al. 2000	<i>n</i> = 16,000, level 1 Mean age = 18.7 mo.			
	One-stage screening: Autistic disorder			
	High-risk score	0.20	0.998	0.26
	Medium-risk score	0.38	0.98	0.05
	ASD			
	High-risk score	0.12	0.998	0.29
	Medium-risk score	0.35	0.98	0.08
	Two-stage screening: Autistic disorder			
	High-risk score	0.18	0.999	0.75
	Medium-risk score	0.20	0.999	0.29
	ASD			
	High-risk score	0.11	0.999	0.83
	Medium-risk score	0.21	0.999	0.59
Scambler et al. 2001	Autism <i>n</i> = 26; mean age = 33 mo., level 2 DD <i>n</i> = 18; mean age = 34 mo.			
	Denver scoring criteria	0.85	1.00	
	CHAT scoring criteria	0.65	1.00	
Scambler et al. 2006	Fragile X <i>n</i> = 17, level 2 Mean age = 34 mo.			
	Denver scoring criteria	0.75	0.92	
	CHAT scoring criteria	0.50	1.00	
Wong et al. 2004 (CHAT-23)	ASD <i>n</i> = 87; mean age = 51 mo., level 2 DD <i>n</i> = 125; mean age = 29 mo.			
	Part A: Fail 2/7 key items	0.93	0.77	0.74
	Part A: Fail 6/23 total items	0.84	0.85	0.79
	Part B: Fail 2/4 key items	0.74	0.91	0.85
Allison et al. 2012 (Q-CHAT-10)	ASD <i>n</i> = 126; mean age = 36 mo. Control <i>n</i> = 754; mean age = 21 mo.			
	Q-CHAT 10-item version	0.91	0.89	0.58
Raza et al. 2019 (Q-CHAT-10)	High-risk sibling <i>n</i> = 116 (with ASD <i>n</i> = 25) Low-risk control <i>n</i> = 56 (with ASD <i>n</i> = 0)			
	18-month screening	0.75	0.63	0.36
	24-month screening	0.71	0.65	0.34

Note: Details including psychometric properties from studies using the Modified Checklist for Autism in Toddlers (M-CHAT) and the M-CHAT Revised with Follow-Up (M-CHAT-R/F) are not included in this entry. See “► M-CHAT” entry

administered a subsequent CHAT. In a population of 35,990 18-month-olds, 6822 screened positive on developmental surveillance; however, only 18% of these cases were administered a CHAT (*n* = 1230), which was primarily a decision made by the nurses who reported that most often the children in those cases seemed to be non-autistic. Compared to a control study area in which

developmental surveillance as usual was being conducted, an equal number of children were referred for an ASD evaluation. The authors concluded that the use of the CHAT did not help increase the number of children who received an ASD diagnosis before age three. However, the procedures used in the study differed from those used in previous studies. Specifically, the CHAT

was not uniformly administered to the entire sample; in addition, 63% of the nurses reported having deviated from the study protocol. Therefore, one might interpret these results to indicate that when providers select a subset of cases for screening, the use of standardized screening tools may not improve detection of autism.

The psychometric properties of the CHAT-23 (Wong et al. 2004) in a sample of 212 toddlers ages 13 to 86 months yielded a sensitivity of 0.93, specificity of 0.77, and positive predictive value of 0.74 when failing two of the seven key items in part A. Failing any six from the 23 parent items resulted in a sensitivity of 0.84, specificity of 0.85, and positive predictive value of 0.79. Failing two of four key items in part B produced a sensitivity of 0.74, specificity of 0.91, and positive predictive value of 0.85. Given the sensitivity-specificity tradeoff between using the key items for screening in part A compared to part B, the authors proposed two-level screening approach in which part B is only administered to those who initially screen positive on part A. Limitations of the study included the small sample size, and that screening was administered after children had already been evaluated and diagnosed.

Initial publication of Q-CHAT (Allison et al. 2008) data compared total scores within an unselected sample ($n = 779$; mean age = 21 months) to total scores among an ASD sample ($n = 160$; mean age = 45 months). Results demonstrated a significantly higher mean score for the ASD group relative to the control group, whose range of scores approximated a normal distribution. Also, the Q-CHAT demonstrated good test-retest reliability of 0.82 and discrimination between ASD and control groups. However, similar to the CHAT-23, interpretation of findings is preliminary, given the small sample size and because screening was completed after children were already evaluated and diagnosed, which may impact how parents report about their child's behavior. The Q-CHAT has been evaluated across several ethnic groups, including in Singapore (Magiati et al. 2015), Colombia (Gutiérrez-Ruiz et al. 2019), and Italy (Ruta et al. 2019a, b). Some of these studies have compared Q-CHAT results from parents of children already diagnosed with ASD to a sample of typical children, but such use

of preselected samples has limited utility in evaluating screening tools, as this method is not consistent with how the tool was designed to be used. Additionally, parents of children with ASD may have varying degrees of ASD knowledge compared to the general population, which can affect responses.

Allison et al. (2012) sought to develop a 10-item version of the Q-CHAT on a sample of 126 preschool children with an autism spectrum diagnosis and 754 typically developing toddlers. They identified the 10 most discriminating items with a cutoff score of 3, yielding the following psychometric properties: sensitivity = 0.91, specificity = 0.89, PPV = 0.58, and internal consistency >0.85 ; however, these psychometrics should be considered preliminary until a large validation study is conducted. Raza et al. (2019) demonstrated that screening with Q-CHAT-10 distinguished high-risk siblings who were ultimately diagnosed with ASD from other high-risk and low-risk toddlers. However, specificity and PPV were below 70%, and its use as a stand-alone measure for high-risk infants was not recommended.

Clinical Uses

The CHAT is designed for use at 18-month checkups in the pediatric setting to identify children at risk for an autism spectrum disorder.

See Also

► [M-CHAT](#)

References and Reading

- Allison, C., Baron-Cohen, S., Wheelwright, S., Charman, T., Richler, J., Pasco, G., & Brayne, C. (2008). The Q-CHAT (Quantitative CHecklist for Autism in Toddlers): A normally distributed quantitative measure of autistic traits at 18–24-months of age: Preliminary report. *Journal of Autism and Developmental Disorders*, 38(8), 1414–1425.
- Allison, C., Auyeung, B., & Baron-Cohen, S. (2012). Toward brief “red flags” for autism screening: the short autism spectrum quotient and the short

- quantitative checklist in 1,000 cases and 3,000 controls. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(2), 202–212.
- Auyeung, B., Ahluwalia, J., Thomson, L., Taylor, K., Hackett, G., O'Donnell, K. J., & Baron-Cohen, S. (2012). Prenatal versus postnatal sex steroid hormone effects on autistic traits in children at 18 to 24 months of age. *Molecular Autism*, 3(1), 17.
- Baird, G., Charman, T., Baron-Cohen, S., Cox, A., Swettenham, J., Wheelwright, S., & Drew, A. (2000). A screening instrument for autism at 18 months of age: A 6-year follow-up study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 39(6), 694–702.
- Baron-Cohen, S., Allen, J., & Gillberg, C. (1992). Can autism be detected at 18 months? The needle, the haystack, and the CHAT. *British Journal of Psychiatry*, 161, 839–843.
- Baron-Cohen, S., Cox, A., Baird, G., Swettenham, J., & Nightingale, N. (1996). Psychological markers in the detection of autism in infancy in a large population. *British Journal of Psychiatry*, 168(2), 158–163.
- Baron-Cohen, S., Wheelwright, S., Cox, A., Baird, G., Charman, T., Swettenham, J., Drew, A., & Doehring, P. (2000). Early identification of autism by the CHecklist for Autism in Toddlers. *Journal of the Royal Society of Medicine*, 93, 521–525.
- Campbell, K., Carpenter, K. L., Espinosa, S., Hashemi, J., Qiu, Q., Tepper, M., ... & Dawson, G. (2017). Use of a digital Modified Checklist for Autism in Toddlers—Revised with follow-up to improve quality of screening for autism. *The Journal of Pediatrics*, 183, 133–139.
- Chlebowski, C., Robins, D. L., Barton, M. L., & Fein, D. (2013). Large-scale use of the Modified Checklist for Autism in low-risk toddlers. *Pediatrics*, 131(4), e1121–e1127.
- Höglund-Carlsson, L., Gillberg, C., Lannero, E., & Blennow, M. (2010). Autism: screening toddlers with CHAT in a child health care programme did not improve early identification. *Acta Paediatrica*, 99, 1897–1899.
- Magiati, I., Goh, D. A., Lim, S. J., Gan, D. Z. Q., Leong, J. C. L., Allison, C., ... & Chong, Y. S. (2015). The psychometric properties of the Quantitative-Checklist for Autism in Toddlers (Q-CHAT) as a measure of autistic traits in a community sample of Singaporean infants and toddlers. *Molecular Autism*, 6(1), 40.
- Raza, S., Zwaigenbaum, L., Sacrey, L. A. R., Bryson, S., Brian, J., Smith, I. M., ... & Roncadin, C. (2019). Brief Report: Evaluation of the Short Quantitative Checklist for Autism in Toddlers (Q-CHAT-10) as a Brief Screen for Autism Spectrum Disorder in a High-Risk Sibling Cohort. *Journal of autism and developmental disorders*, 49(5), 2210–2218.
- Ruta, L., Arduino, G. M., Gagliano, A., Apicella, F., Leonardi, E., Famà, F. I., ... & Allison, C. (2019a). Psychometric properties, factor structure and cross-cultural validity of the quantitative CHecklist for autism in toddlers (Q-CHAT) in an Italian community setting. *Research in Autism Spectrum Disorders*, 64, 39–48.
- Ruta, L., Chiarotti, F., Arduino, G. M., Apicella, F., Leonardi, E., Maggio, R., ... & Tartarisco, G. (2019b). Validation of the Quantitative CHecklist for Autism in Toddlers (Q-CHAT) in an Italian clinical sample of young children with Autism and Other Developmental Disorders. *Frontiers in psychiatry*, 10, 488.
- Robins, D. L., Fein, D., & Barton, M. (1999). Modified Checklist for Autism in Toddlers (M-CHAT). Self-published. See www.mchatscreen.com.
- Robins, D. L., Fein, D., Barton, M. L., & Green, J. A. (2001). The Modified Checklist for Autism in Toddlers: An initial study investigating the early detection of autism and pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 31(2), 131–144.
- Robins, D. L., Fein, D., & Barton, M. (2009). Modified Checklist for Autism in Toddlers, revised, with follow-up (M-CHAT-R/F). Self-published. See www.mchatscreen.com.
- Robins, D. L., Casagrande, K., Barton, M., Chen, C. M. A., Dumont-Mathieu, T., & Fein, D. (2014). Validation of the modified checklist for autism in toddlers, revised with follow-up (M-CHAT-R/F). *Pediatrics*, 133(1), 37–45.
- Scambler, D., Rogers, S. J., & Wehner, E. A. (2001). Can the Checklist for Autism in Toddlers differentiate young children with autism from those with developmental delays? *Journal of the American Academy of Child & Adolescent Psychiatry*, 40(12), 1457–1463.
- Scambler, D. J., Hepburn, S. L., & Rogers, S. J. (2006). A two-year follow-up on risk status identified by the Checklist for Autism in Toddlers. *Developmental and Behavioral Pediatrics*, 27(2), S104–S110.
- Scambler, D. J., Hepburn, S. L., Hagerman, R. J., & Rogers, S. J. (2007). A preliminary study of screening for risk of autism in children with fragile X syndrome: testing two risk cut-offs for the CHecklist for Autism in Toddlers. *Journal of Intellectual Disability Research*, 51, 269–276.
- Sturner, R., Howard, B., Bergmann, P., Morrel, T., Andon, L., Marks, D., ... & Landa, R. (2016). Autism screening with online decision support by primary care pediatricians aided by M-CHAT/F. *Pediatrics*, 138(3), e20153036.
- VanDenHeuvel, A., Fitzgerald, M., Greiner, B. A., & Perry, I. J. (2006). Screening for autistic spectrum disorder at the 18-month developmental assessment: a population-based study. *Irish Medical Journal*, 100(8), 565–567.
- Wong, V., Hui, L. H., Lee, W. C., Leung, L. S., Ho, P. K., Lau, W. L., Fung, C. W., & Chung, B. (2004). A modified screening tool for autism (Checklist for Autism in Toddlers [CHAT-23]) for Chinese children. *Pediatrics*, 114, e166–e176.
- Wong, H. S., Huertas-Ceballos, A., Cowan, F. M., Modi, N., & Medicines for Neonates Investigator Group. (2014). Evaluation of early childhood social-communication difficulties in children born preterm using the Quantitative Checklist for Autism in Toddlers. *The Journal of Pediatrics*, 164(1), 26–33.

Chelation

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Definition

Chelation involves the use of various agents to remove heavy metals from the body – typically lead – but sometimes arsenic, mercury, or other metals are the targets. Various agents can be used for this process. These agents were first developed in the treatment of poison gas inhalation during World War I and have been substantially modified and refined over the years to increase efficiency while minimizing side effects. Various routes of administration are used for these chelating agents. There are occasional uses in treatment of other diseases, e.g., those that involve excess iron storage.

As an alternative treatment, many claims have been made, but not scientifically substantiated for a range of conditions ranging from atherosclerosis to autism. The use in autism rested, in part, on the unproven suggestion that high mercury levels were involved in the production of autism. There is no scientific justification for this process in autism.

There can be significant risks to chelation – including hypocalcemia, anemia, kidney problems, and cardiac difficulties. There are reports of deaths including one child with autism. Deaths may relate to hypocalcemia. As with all non-proven treatments, particularly when some substantial risk is concerned, parents should be careful to make informed treatment choices.

References and Reading

Beauchamp, R. A., Willis, T. M., Betz, T. G., & Villanacci, J. (2006). Deaths associated with hypocalcemia from chelation therapy - Texas, Pennsylvania, and Oregon, 2003–2005. *Morbidity, Mortality Weekly Review (MMWR)*, 55(8), 204–207.

Volkmar, F., & Wiesner, L. (2009). *A practical guide to autism*. Hoboken: Wiley.

Weber, W., & Newmark, S. (2007). Complementary and alternative medical therapies for attention-deficit/hyperactivity disorder and autism. *Pediatric Clinics of North America*, 54(6), 983–1006.

Chess, Stella

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Name and Degrees

Stella Chess

- B.A., 1935, Smith College, Northampton, MA
- M.D., 1939, New York University College of Medicine, New York, NY

Major Appointments (Institution, Location, Dates)

- Psychiatry Residency, Grasslands Hospital in Eastview, NY
- 1954–1966 Assistant Professor, New York Medical College
- 1966–2007 Associate and Full Professor, New York University School of Medicine

Major Honors and Awards

- Smith College Medal, 1999

Landmark Clinical, Scientific, and Professional Contributions

Stella Chess made many important scientific contributions. She began her New York Longitudinal Study in 1956. This body of work, focused on careful observation of styles of behavior and

personality, led to the development of Chess's concept of varying temperament. Her work helped shift the field from a sole reliance on intrapsychic conflict and anxiety (as exemplified in then popular psychoanalytic work) but instead suggested the importance of understanding individual differences. Chess also elaborated the notion of "goodness of fit," e.g., relative to potential matches and mismatches in parental style and child temperament. This work led to a growing body of work on the basis of individual differences, their stability, and relationship to childhood problems. In 1971, she reported on a possible observation of an association between congenital rubella. In retrospect, the developmental course of many of the patients she first reported seemed less typical of autism, but her work focused attention on a possible biological mechanism in the condition. Chess also was noted for her pioneering work in psychiatric-pediatric liaison work and also edited an influential book series, *Annual Progress in Child Psychiatry and Child Development*, that continues to be published. She founded the first pediatric psychiatry unit at Bellevue Hospital and was a professor at NYU.

Short Biography

Born in New York to immigrant parents from Russia, Chess studied at the Ethical Culture School and then Smith College before entering NYU Medical School in 1935. She met her husband, and research collaborator, Alexander Thomas while they both were in medical school. They married in 1938. While in medical school, she worked with Laretta Bender. Chess began, in collaboration with her husband, the New York Longitudinal Study of Child Development that followed several hundred youth. During the course of their work, they identified a series of basic temperaments and parenting styles and also began to emphasize the importance of "goodness of fit" with parents. Many trainees worked with her. She continued to teach at NYU into her 90s. She was involved in training many of the leaders in the field and collaborated with Michael Rutter among others.

See Also

- [Bender, Laretta](#)
- [Rutter, Michael](#)

References and Reading

- Chess, S. (1971). Autism in children with congenital rubella. *Journal of Autism and Childhood Schizophrenia*, 1(1), 33–47.
- Chess, S. (1977). Follow-up report on autism in congenital rubella. *Journal of Autism and Childhood Schizophrenia*, 7(1), 69–81.
- Chess, S. (1979). Discussion: Language, cognition, and autism by Rutter, Studies of the autistic syndromes by Coleman. *Research Publications – Association for Research in Nervous and Mental Disease*, 57, 277–280.
- Chess, S., & Thomas, A. (1999). *Goodness of fit*. Philadelphia: Brunner/Mazel.
- Chess, S., Fernandez, P., & Korn, S. (1978). Behavioral consequences of congenital rubella. *Journal of Pediatrics*, 93(4), 699–703.
- Rutter, M., Birch, H. G., Thomas, A., & Chess, S. (1964). Temperamental characteristics in infancy and the later development of behavioural disorders. *British Journal of Psychiatry*, 110(468), 651–661. Royal College of Psychiatrists, United Kingdom.

Child Abuse in Autism

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Definition

Children with autism spectrum disorders are significantly more likely than typically developing children to be the victims of abuse, which encompasses emotional abuse, physical abuse, sexual abuse, and neglect.

Historical Background

Child abuse, which includes physical abuse, emotional abuse, sexual abuse, and neglect, is less

studied among children with autism spectrum disorders (ASD) than it is among typically developing children, despite their elevated risk for exposure. However, abuse among ASD and intellectual disability (ID) populations is a growing area of research and awareness, and a specific focus on sexual abuse has emerged. While previous attitudes held that individuals with disabilities were asexual and could not be negatively impacted by others' sexual behaviors, current research is more respectful of the humanity and sexuality of individuals with disabilities, including ASD. Individuals with ASD who have extremely limited or impaired functional communication skills may be particularly at risk, as perpetrators may believe that the individual with ASD will not be able to disclose their role in the abusive incidents to family members or authorities.

Current Knowledge

The Centers for Disease Control and Prevention (CDC) reported that in 2008, approximately 772,000 children in the USA were victims of maltreatment. Of these children, the majority (71%) experienced maltreatment, 16% experienced physical abuse, 9% experienced sexual abuse, and 7% experienced emotional abuse. The rates of child sexual abuse are particularly high: recent studies by the CDC suggest that 16.67% of boys and 25% of girls in the general population experience some form of sexual abuse before the age of 18. It is likely that the actual rate of sexual abuse is even higher than reported by the CDC, given the multiple reasons that victims might be reluctant to disclose or report abuse when it has happened. Also, it is important to keep in mind that the CDC reports statistics for the greater population, and does not compare rates of maltreatment based on children's disability status. However, research has consistently suggested that children with ID are at greater risk than typically developing children to be the victims of all forms of maltreatment (Sobsey 1994). Children with ID are also less likely than typically developing children to report abuse when it has

occurred. Reasons for more limited reporting include communication impairments, social knowledge deficits (e.g., not understanding that the interaction was inappropriate), and greater likelihood of attributing blame for a negative interaction to oneself due to a history of difficult social interactions. The constellation of research documenting greater exposure coupled with reduced likelihood of reporting is very concerning and highlights the importance of protecting the safety and well-being of children with ASD, ID, and other disabilities.

A great deal of what is currently understood about abuse among children with ASD comes from a landmark study by Mandell et al. (2005). This study is unique in that it looks specifically at the experiences of children with ASD, instead of ID more broadly, and considers experiences of both sexual and physical abuse. Unlike previous studies, which drew heavily from institutionalized populations, Mandell et al. recruited participants who received treatment in community settings, much like the majority of children diagnosed with ASD today. This was an important distinction because children who live in hospital and institutional settings are at a greater risk for abuse, and it is problematic to generalize findings from this population to children who live at home with their families. The results of this study revealed high rates of abuse – 18.5% of the 156 children in the sample were reported by their parents to have experienced physical abuse, and 16.6% were reported to have experienced sexual abuse – among children with ASD. While these rates are lower than the ones put forth by the CDC of all children, it is important to consider that the average age of participants in this study was 11 years and the CDC reports their statistics through age 18. Mandell et al. found that children who had experienced physical abuse were more likely than non-abused children to act out sexually, to engage in abusive behavior themselves, to attempt suicide, and to have conduct and/or academic problems. Similarly, children who had experienced sexual abuse were more likely than non-abused children to act out sexually, to engage in abusive behavior themselves, and to attempt suicide. Additionally, these children were also

more likely to engage in self-injurious behavior in addition to suicidal behavior, to run away from home, and to have had a psychiatric hospitalization. Contrary to the previous belief that children with ASD were not susceptible to the effects of abuse, the findings of this study suggest quite the opposite.

In considering recent research about child abuse and ASD, it is important to consider that the rate of abuse is likely even higher than reported since communication deficits associated with ASD may make it more challenging for victims to report abuse, and for these reports to be taken seriously, when it does occur. Some research has been conducted on victims' reactions following sexual abuse, and these findings suggest that children with ASD may respond differently from typically developing children. For example, a child with ASD who has low language abilities may engage in self-injurious or self-stimulatory behavior to try to communicate or cope with the abuse that he or she experienced. Or, a child with ASD who demonstrates echolalia may recount what a perpetrator said during an abusive episode. However, this may not be recognized for what it is by parents or caretakers, who could dismiss the behavior simply as non-functional communication or meaningless jargon. In the absence of recognizing that abuse has occurred and taking appropriate steps to intervene, the abuse could continue. Therefore, the current literature suggests that parents and caretakers of children with ASD should take note of any changes in behavior (including an increase in intensity or frequency of an existing behavior, or the appearance of a new one), as it could indicate abuse. This is not to say that changes in behavior always signal that abuse has occurred – it is prudent, however, to consider the possibility that individuals with ASD may have been exposed to abuse.

The same language impairments that may prevent children with ASD from communicating that abuse has occurred may be part of the reason why they are victimized more often than typically developing children in the first place. Perpetrators may believe that children with ASD would be less likely to report the abuse to others and, in turn, the

perpetrator would not be discovered. Also, the social deficits associated with ASD may also make children on the spectrum appealing to perpetrators. For example, the perpetrator may believe that a child with ASD can be manipulated more easily and be less likely to “fight back” against advances than a typically developing child. Unfortunately, perpetrators may take advantage of children with social difficulties by presenting themselves as a “friend.” Also, children with ASD are encouraged to cooperate with teachers, clinicians, and other professionals from a very early age, and this learned compliance may lead them to follow and not to question the motives or advances of a perpetrator.

Both large-scale and small-scale studies have suggested that children with ASD are at a greater risk for abuse and maltreatment than typically developing children. There are multiple possible explanations for this phenomenon, some of which are related to the nature of ASD symptoms. There is compelling evidence that parents raising children with ASD experience much higher levels of parenting stress and depression than parents raising typically developing children or children with other intellectual and developmental disabilities. Parenting stress and depression have each been linked as risk factors for abuse (Holden and Banez 1996; McPherson et al. 2009). Because of the unpredictability of behavior among children with ASD, parents and caregivers may at times become frustrated with their children's ASD-associated traits and instead of coping with this frustration in constructive ways, they may direct it aggressively and abusively toward their child. The frustrations that lead some parents to abuse their children with ASD may lead others to neglect them. Algood et al. (2011) examine systems-level factors to see which characteristics might contribute to the neglect of children with developmental disabilities more broadly.

When examining the rates and types of maltreatment among children with ASD, it is important to consider who the most common perpetrators are. Current research suggests that the most likely perpetrator differs depending on the type of abuse. In the general population, parents are the most common perpetrators of child

neglect. However, when it comes to the other forms of child maltreatment, perpetrators frequently fall into one of these four categories: disability service providers, acquaintances and neighbors, family members, and peers with disabilities (Sobsey 1994). This information is helpful to consider when assessing whether an individual with ASD has experienced abuse; it can also help in the development of preventative programs, which are discussed in the section below.

Future Directions

Given what is known about the heightened risk of sexual abuse among children with ASD, it is important to provide age- and developmentally appropriate sexuality training to all individuals, regardless of their disability status (Edelson 2010) and to ensure that parents understand the heightened risk and have supportive resources and respite available. While sexuality education is associated with multiple positive outcomes, it serves a particular function for individuals, such as children with ASD, who are susceptible to abuse. Sexuality education can empower individuals so that they may be proactive and take steps to prevent being victimized (although it is important to note here that sexual abuse is *never* the fault of the victim). Sexuality education is also important because it can help individuals to recognize and report sexual abuse when it has occurred. Especially for children with ASD, who may have difficulty navigating social situations and understanding the intentions of others, social skills training can serve a similarly valuable function in protecting against sexual or emotional abuse.

In light of the heightened rates of abuse among children with ASD and its associated detrimental outcomes, it is very important to have valid and reliable instruments that can determine whether a child with ASD has experienced abuse. Edelson (2010) points out that some tools that are used with typically developing children, such as interviews and anatomically detailed dolls, are inappropriate for children with ASD. Children with ASD often prefer familiar routines, environments,

and settings, and to be interviewed by a new clinician when abuse is suspected could be an upsetting and off-putting experience. Also, some of the current tools for assessing abuse require a level of verbal expression that many children with ASD do not possess. Therefore, instruments for detecting abuse must be developed specifically for the needs and capabilities of children with ASD.

Overall, more research is needed to understand the rates and types of abuse experienced specifically by children with ASD, and who is perpetrating this abuse. Additionally, more research is needed on the short- and long-term effects of abuse on children with ASD. Taken together, this information could be useful in preventative, educational programs for both children with ASD and the adults in their lives. Also, this information could help in the interventions and treatments for children who have been victimized.

See Also

- ▶ Parent Training
- ▶ Sex Education
- ▶ Sexuality in Autism

References and Reading

- Algood, C. L., Hong, J., Gourdine, R. M., & Williams, A. B. (2011). Maltreatment of children with developmental disabilities: An ecological systems analysis. *Children and Youth Services Review*, 33(7), 1142–1148.
- Baladerian, N. (2004). *An overview of violence against children with disabilities*. Presentation at the best practice II conference on child abuse & neglect, Mobile.
- Edelson, M. G. (2010). Sexual abuse of children with autism: Factors that increase risk and interfere with recognition of abuse. *Disability Studies Quarterly*, 30(1). Retrieved from <http://dsq-sds.org/article/view/1058/1228>
- Gammicchia, C., & Johnson, C. Living with autism: Information for domestic violence and sexual assault counselors. Retrieved from http://www.leanonus.org/images/Domestic_Violence_and_Sexual_Assault_Counselors.pdf
- Holden, E., & Banez, G. A. (1996). Child abuse potential and parenting stress within maltreating families. *Journal of Family Violence*, 11(1), 1–12.
- Mahoney, A., & Poling, A. (2011). Sexual abuse prevention for people with severe developmental disabilities.

- Journal of Developmental and Physical Disabilities*, 23(4), 369–376.
- Mandell, D. S., Walrath, C. M., Manteuffel, B., Sgro, G., & Pinto-Martin, J. A. (2005). The prevalence and correlates of abuse among young children with autism served in comprehensive community-based mental health settings. *Child Abuse & Neglect*, 29, 1359–1372.
- Marge, D. K. (Ed.). (2003). *A call to action: Ending crimes of violence against children and adults with disabilities: A report to the nation*. Syracuse: SUNY Upstate Medical University.
- McPherson, A. V., Lewis, K. M., Lynn, A. E., Haskett, M. E., & Behrend, T. S. (2009). Predictors of parenting stress for abusive and nonabusive mothers. *Journal of Child and Family Studies*, 18(1), 61–69.
- Sexual Abuse. Autism Speaks. Retrieved from <http://www.autismspeaks.org/family-services/autism-safety-project/abuse>
- Sobsey, D. (1994). *Violence and abuse in the lives of people with disabilities: The end of silent acceptance?* Baltimore: Paul H. Brookes.

Child and Family Characteristics that Predict Clinic Appointment Attendance and Alignment with Providers

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Definition

Children with autism spectrum disorder (ASD) present with a wide range of complex needs related to their mental and physical health. To

adequately address the various needs present in ASD, more families are being referred to ASD specialty clinics. Families who receive services at ASD specialty clinics often come for multiple visits with many interdisciplinary providers (e.g., physicians, psychologists, speech and language pathologists, etc.). For example, there is often an initial diagnostic appointment with a medical and/or psychological provider. After diagnosis, families are frequently referred for further comprehensive assessments related to their cognitive (with neuropsychologists), language (with speech and language pathologists), and physical (with occupational and/or physical therapists) needs.

The provision of specialty services is often from interdisciplinary providers across multiple visits to the clinic, including an initial appointment, the diagnostic evaluation, and follow-up care. Given that families of children with ASD are frequent consumers of specialty services, it is important to examine appointment attendance and alignment with providers about ASD diagnosis. When children are not receiving intervention, families are less likely to keep their initial appointment. It is possible that children who are not receiving intervention are presenting with minimal or inconsistent symptoms that could be temporarily extinguished. As a result, their parents may change their minds when it comes to keeping their initial appointment. These families may be reticent to follow through with their initial appointment seeking due to lack of familiarity with the diagnostic and treatment process. It is essential to spend more time scheduling and providing these families with essential resources to make them more comfortable about seeking services. More specifically, families may benefit from live conversations with a clinic triage specialist. This specialist could inquire about the nature of their concern, and explain the evaluation process, and, where appropriate, assist with the identification of local resources.

Families residing long distances and having older children are less likely to keep their initial and follow-up appointments. Families that live far distances have more difficulty accessing and utilizing specialty care services and, therefore, may rely more heavily on the school system for

services. Older children may be presenting with less severe symptoms. Therefore, these parents may not be scheduling their initial appointment because older and/or less symptomatic children are not experiencing clinically significant impairments that are interfering with daily life and others have tolerated their differences more easily. For these families, more refined communication is necessary to explain the importance of initial visits and follow-up care in order to support parents in service utilization. It is important that parents perceive keeping their appointment to have a high benefit to cost ratio.

African-American families are less likely to keep their initial appointment and express initial doubts with providers about the diagnosis. Therefore, there are barriers that are preventing African-American families from utilizing specialty care services despite them taking initiative. It is important for service provision systems to identify and address the barriers that African-American families may experience during the critical period from service initiation to utilization. African-American families' pre-visit diagnostic beliefs are more likely to be misaligned with providers' delivery of an ASD diagnosis. There is a stigma associated with being "labeled" with a mental health diagnosis, and this is particularly true for ethnic and racial minority families. Lack of alignment may be one probable mechanism through which disparities arise (i.e., later diagnosis, more visits before diagnosis, and/or different diagnosis) between children who are African-American and other races compared to White children. In order for families and providers to be aligned in their diagnostic beliefs, it is imperative that providers communicate in culturally competent ways about ASD symptomology, as well as early detection and intervention.

See Also

- [Early Diagnosis](#)
- [Multidisciplinary Evaluation](#)

- [Parent Responsiveness to Children at Risk of ASD](#)
- [Parent-Professional Partnership](#)

References and Reading

- Cummings, J., Lynch, F., Rust, K., Coleman, K., Madden, J., Owen-Smith, A., . . . Massolo M. (2016). Health services utilization among children with and without autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46(3), 910–920.
- Dantas, L., Fleck, J., Cyrino Oliveira, F., & Hamacher, S. (2018). No-shows in appointment scheduling – A systematic literature review. *Health Policy*, 122(4), 412–421.
- Kalb, L., Freedman, B., Foster, C., Menon, D., Landa, R., Kishfy, L., & Law, P. (2012). Determinants of appointment absenteeism at an outpatient pediatric autism clinic. *Journal of Developmental & Behavioral Pediatrics*, 33(9), 685–697.
- Macari, S., Wu, G., Powell, K., Fontenelle, S., Macris, D., & Chawarska, K. (2018). Do parents and clinicians agree on ratings of autism-related behaviors at 12 months of age? A study of infants at high and low risk for ASD. *Journal of Autism and Developmental Disorders*, 48(4), 1069–1080.

Child and Family-Centered Intervention

- [Role Release](#)

Child Behavior Checklist 1.5–5

- [Child Behavior Checklist in Autism](#)

Child Behavior Checklist 6–18

- [Child Behavior Checklist in Autism](#)

Child Behavior Checklist in Autism

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Synonyms

CBCL 1.5–5; CBCL 6–18; Child Behavior Checklist 1.5–5; Child Behavior Checklist 6–18

Abbreviations

ASD	Autism spectrum disorder
ADHD	Attention-deficit/hyperactivity disorder
ASEBA	Achenbach System of Empirically Based Assessment
CBCL	Child Behavior Checklist
DSM	Diagnostic and Statistical Manual of Mental Disorders
K-	Schedule for Affective Disorders and
SADS	Schizophrenia – Childhood Version
ODD	Oppositional defiant disorder
TRF	Teacher Report Form
YSR	Youth Self-Report

Description

Introduction

Youth diagnosed with autism spectrum disorder (ASD) appear to be at significantly higher risk for a co-occurring emotional and/or behavioral disorder relative to the general pediatric population. According to the *Diagnostic and Statistical Manual of Mental Disorders-Fifth Edition* (DSM-5; American Psychiatric Association [APA] 2013) – approximately 70% of individuals with ASD are likely to have one and 40%

two or more co-occurring disorders. When rendering a diagnosis, the DSM-5 now requires evaluators to specify whether ASD is accompanied by a co-occurring mental or behavioral disorder, which oftentimes requires specific treatment. Therefore, there is a need for reliable and valid measures of emotional and behavioral disorders for youth with ASD.

The Achenbach System of Empirically Based Assessment (ASEBA) consists of several norm-referenced paper and pencil rating scales that were developed to assess for adaptive competencies and a broad range of emotional and behavioral disorders in children and adolescents. Two forms are available for parents: the Child Behavior Checklist 1.5–5 (CBCL 1.5–5; Achenbach and Rescorla 2000) which is used for children aged 1.5–5 years and the Child Behavior Checklist 6–18 (CBCL 6–18; Achenbach and Rescorla 2001) which is used for youth aged 6–18 years. Three other forms are also available: the Caregiver/Teacher Report Form for 1.5–5-year-olds, the Teacher Report Form (TRF) for 6–18-year-olds, and a Youth Self-Report (YSR) for 11–18-year-olds. The various forms that comprise the ASEBA system allow for data integration across multiple informants which provides an understanding of whether and how emotional and/or behavioral disorders manifest across settings. These scales have been normed in many societies around the world, and they reflect one of the best studied measures developed for youth (see Berube and Achenbach 2015). These rating scales assess for emotional and behavioral disorders that are most often observed in youth with ASD such as anxiety, depression, withdrawal, social problems, attention problems, and aggression.

This review focuses on the emotional and behavioral disorder scales of the CBCL 1.5–5 and CBCL 6–18. These ASEBA scales have received the most attention in studies of youth with ASD. Empirical data suggest that these two measures have utility for the assessment of co-occurring disorders in this population.

CBCL Scales

The CBCL 1.5–5 and the CBCL 6–18 contain items pertaining to specific emotional and behavioral responses. Parents rate each item according to how true each statement is about their child: 0 “Not true,” 1 “Somewhat or sometimes true,” or 2 “Very true or often true.” Additionally, several open-ended items allow respondents to provide additional information that may be important for an evaluator to know about the child. The CBCL 1.5–5 ratings describe a child’s functioning during the last 2 months, and ratings on the CBCL 6–18 describe functioning during the past 6 months. Administration time is generally 10–20 min. Each measure can be scored by hand or by ASEBA software.

The CBCL 1.5–5 and CBCL 6–18 each contain two sets of scales: the empirically derived and DSM-oriented scales. The *empirically derived* scales were developed through factor analysis of data collected from a United States standardization sample. Both the CBCL 1.5–5 and the CBCL 6–18 contain two kinds of empirically derived scales. The “narrowband,” or syndrome scales, assess a wide range of specific emotional and behavioral syndromes (e.g., attention problems, social problems, aggressive behavior). The “broadband” scales are called the *Internalizing Domain* and *Externalizing Domain* which assess for broader classes of emotional (internalizing) and behavioral (externalizing) disorders. The *DSM-oriented* scales were *conceptually derived* and were meant to correspond to broad diagnostic categories in the *Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition* (DSM-IV; APA 1994). These scales were recently updated to be more consistent with the DSM-5 (APA 2013), and this is discussed below. The empirically derived and DSM-oriented scales are both norm referenced. Scale scores can be plotted on profiles that allow clinicians to readily examine relative scale elevations across several problem areas.

The CBCL 1.5–5 and CBCL 6–18 are scored and interpreted similarly. The raw scores for items within each of the empirically derived and DSM-oriented scales are summed and converted to

norm-referenced T-scores ($M = 50$, $SD = 10$). A Total Problems T-score is also available and is determined by the sum of all item scores. One set of norms is provided for the CBCL 1.5–5, and separate norms are provided for each gender within the 6–11 and 12–18 year age ranges on the CBCL 6–18. “Clinically significant” elevations are indicated by T-scores ≥ 64 on the broadband scales and ≥ 70 on the syndrome scales. “Borderline” elevations range from 60 to 63 and 65 to 69 on the broadband and narrowband syndrome scales, respectively. These qualitative categories reflect symptom severity, and scores falling within either category suggest the need for a diagnostic assessment.

Historical Background

Studies of the CBCL in ASD Samples

Most studies of the CBCL in samples of youth with ASD examined the extent to which the syndrome and broadband scales discriminated between those with and without ASD. Significant methodological differences were observed across these studies that included varied approaches to confirming an ASD diagnosis, whether the youth were evaluated for a co-occurring disorder, the clinical status and characteristics of non-ASD comparison groups (e.g., typically developing, those with non-ASD developmental disorders or psychiatric disorders), and the specific ASEBA measure used (e.g., see Biederman et al. 2010; Bolte et al. 1999; Duarte et al. 2003; Hurtig et al. 2009; Kanne et al. 2009; Ooi et al. 2010). One consistent finding that emerged from these studies was that youth with ASD often scored significantly higher than youth without ASD across several scales. Many times youth with ASD scored significantly higher than comparison groups on the *Withdrawn/Depressed*, *Social Problems*, and *Thought Problems* scales. However, it is not clear whether scale elevations were related to the presence of co-occurring emotional and behavioral disorders rather than the ASD. Base rate information on specific CBCL profiles would be helpful to

understand how unique such profiles are to the ASD population because recent studies indicated that the CBCL scales are not measures of ASD-related behavior (see Magyar and Pandolfi 2017; Pandolfi et al. 2014).

Although more empirical data are needed, several recent studies provided psychometric support for the CBCL 1.5 and CBCL 6–18 as reliable and valid measures of emotional and behavioral disorders in youth with ASD (see Magyar and Pandolfi 2017; Pandolfi et al. 2012, 2014). The results supported the unidimensionality of nearly all CBCL 1.5–5 and CBCL 6–18 empirically derived syndrome scales, which indicated that each scale measured one construct. The lone exception was the CBCL 1.5–5 *Sleep Problems* scale which was found to consist of two factors: *dysssomnias* and *parasomnias*. The two factor internalizing-externalizing factor structure was supported for both of these measures, consistent with Achenbach and Rescorla (2000, 2001). Scale reliability was generally good to excellent across the syndrome and broadband scales of each measure, although the reliabilities of the *Somatic Complaints* (CBCL 1.5–5) and *Thought Problems* scales (CBCL 6–18) were somewhat lower than desired for a screening measure.

To date, only one study provided evidence on the diagnostic accuracy of the CBCL 6–18 empirically derived scales for identifying co-occurring emotional and behavioral disorders in youth with ASD (Pandolfi et al. 2012). All youth were evaluated for ASD using the Autism Diagnostic Interview- Revised (ADI-R; Rutter et al. 2003) and the Autism Diagnostic Observation Schedule (Lord et al. 2002). Co-occurring psychiatric disorders were evaluated through a standardized multi-method assessment protocol which included the Schedule for Affective Disorders and Schizophrenia – Childhood Version (K-SADS; Kaufman et al. 1996), a semi-structured diagnostic interview. In addition to between-group differences across several empirically derived scales (i.e., ASD only vs. ASD + co-occurring emotional and/or behavioral disorders), the CBCL 6–18 demonstrated good sensitivity

(>.80) for identifying co-occurring depression, anxiety, attention-deficit/hyperactivity disorder (ADHD), and oppositional defiant disorder (ODD) in individuals with ASD. The specific scales with favorable sensitivity were those that were conceptually consistent with the target disorder under investigation. However, specificity was generally low. A subsequent study on the CBCL 6–18 found that those scales that were purported to assess for emotional problems were not measures of ASD: the vast majority of the individual differences in scores for youth on these scales were related to co-occurring emotional disorders, and not to their ASD symptoms (see Pandolfi et al. 2014).

We are aware of only one study on the CBCL 6–18 DSM-oriented scales in youth with ASD (see Magyar and Pandolfi 2017). The only scales evaluated were the *Affective Problems* (recently renamed *Depressive Problems*) and *Anxiety Problems* scales. Findings indicated that each of these scales reliably measured a single construct. The scales did not correlate with the ADI-R current behavior algorithm, but they did correlate with the K-SADS. The results indicated that the scales measured what they purported to measure: *Affective Problems* measured depression and *Anxiety Problems* measured anxiety. Neither scale appeared to be a measure of ASD-specific problems. With respect to diagnostic accuracy, sensitivity was acceptable for both scales, and specificity was acceptable for *Affective Problems* but somewhat low for *Anxiety Problems*.

Research findings lend support for using the CBCL to assess youth with ASD in clinical and research settings. Replication of findings is needed, especially within important subgroups within the ASD population: such as within specific age groups, each gender, and those with various levels of autism severity, language impairment, and cognitive ability. This would provide much more specific information to assist in the clinical decision-making of those professionals who work with this heterogeneous population, many of whom are often in need of both ASD treatment and specific treatment for co-occurring disorders.

Psychometric Data

Test Development and Psychometric Properties

Achenbach and Rescorla (2000, 2001) reported several lines of evidence that supported CBCL scores as indicators of emotional and behavioral disorders in the general population. Although there is close correspondence in the kinds of syndromes that are measured by the CBCL 1.5–5 and CBCL 6–18, some differences exist and are detailed next.

Factor analyses of test items were used to help construct the empirically derived scales for both measures. For the CBCL 1.5–5, seven first-order factors were identified, and these represented the seven narrowband syndrome scales. Two higher order factors were also identified which reflected the broadband scales. One was called the *Internalizing Domain* which consisted of four emotional syndromes (i.e., the first-order factors) that were labeled *Emotionally Reactive*, *Anxious/Depressed*, *Somatic Complaints*, and *Withdrawn*. The other higher order factor, named the *Externalizing Domain*, consisted of two behavioral syndromes which were called *Attention Problems* and *Aggressive Behavior*. One first-order factor, *Sleep Problems*, did not belong to either higher order factor.

A slightly different set of syndrome scales was found for the CBCL 6–18. While it too contained two higher order *Internalizing* and *Externalizing Domains*, the syndromes that belonged to each were different. The *Internalizing Domain* contained the *Anxious/Depressed*, *Withdrawn/Depressed*, and *Somatic Complaints* syndrome scales. The *Externalizing Domain* contained the *Rule-Breaking Behavior* and *Aggressive Behavior* syndrome scales. Three other syndrome scales did not belong to either broadband scale: *Social Problems*, *Thought Problems*, and *Attention Problems*. These are considered mixed syndrome scales because they had sizable factor loadings on both broad domains in the Achenbach and Rescorla (2001) factor analyses.

The DSM-oriented scales were initially developed to be conceptually consistent with broad DSM diagnostic categories. The CBCL 1.5–5

scales included *Affective Problems* (now called *Depressive Problems*, see below), *Anxiety Problems*, *Pervasive Developmental Problems* (now called *Autism Spectrum Problems*, see below), *Attention Deficit/Hyperactivity Problems*, and *Oppositional Defiant Problems*. The CBCL 6–18 scales included *Affective Problems* (now called *Depressive Problems*), *Anxiety Problems*, *Somatic Problems*, *Attention Deficit/Hyperactivity Problems*, *Oppositional Defiant Problems*, and *Conduct Problems*. The DSM-oriented scales complement the empirically derived scales to assist practitioners in the differential diagnostic process. Achenbach and Rescorla (2000, 2001) and Achenbach (2014) described scale construction in detail which included experts in child psychology and psychiatry to help devise scale content. The DSM-oriented scales that were conceptually aligned with the DSM-IV received much psychometric support (described below), and these scales were recently revised to be consistent with the DSM-5.

The revisions featured name changes to some of the scales, and only minor content changes to two of the scales (see Achenbach 2014). On the CBCL 1.5–5, the former *Pervasive Developmental Problems* was renamed *Autism Spectrum Problems*, and the scale's updated content changed only with the deletion of one item. *Affective Problems* was renamed *Depressive Problems* on both the CBCL 1.5–5 and CBCL 6–18. On the CBCL 6–18, the *Anxiety Problems* scale now features three new additional items.

Both test manuals provide several lines of psychometric evidence for the empirically derived and DSM-oriented scales. It is noted that the psychometric evidence pertaining to the DSM-oriented scales was gathered prior to the recent revision to these scales. The authors substantiated the CBCL's content validity by citing years of research, clinical experience, and consultation with several stakeholders in children's mental health assisting in the item selection process. Interested readers should consult the technical manuals for more information about the content of specific scales.

The data presented in each technical manual indicate that the CBCL 1.5–5 and CBCL 6–18

appear to be sufficiently reliable for clinical use with the general population. The technical manuals each reported internal consistencies $\geq .89$ for the *Internalizing* and *Externalizing Domains* and for *Total Problems*. For the CBCL 1.5–5, some of the narrowband and DSM-oriented scales had internal consistencies $< .70$, so it is especially important to interpret these scales in conjunction with other clinical data (see Achenbach and Rescorla 2000). Reported odds ratios for the CBCL 1.5–5 and CBCL 6–18 indicated that those with scale scores in the borderline/clinically significant ranges were far more likely to be referred for mental health services than youth with scores below these ranges. A related finding indicated that a sizable percentage of the youth who were referred for mental health services had scores in these elevated ranges. Achenbach and Rescorla (2000, 2001) also presented significant correlations between CBCL scores and DSM diagnoses. These data suggested that youth with elevated scores on any of the empirically derived or DSM-oriented scales should be referred for a diagnostic assessment.

Clinical Uses

The CBCL 1.5–5 and CBCL 6–18 appear to have utility in ASD assessment. These measures assess for the kinds of disorders that occur at fairly high rates in youth with ASD. These include depression, anxiety, ADHD, and ODD. The empirically derived syndrome scales assess for disorders that cut across DSM categories. These scales can inform practitioners about the range of possible emotional and/or behavioral disorders that might affect an individual. Because the DSM-oriented scales are conceptually consistent with broad DSM-5 diagnostic categories, they can be used to assist in: (a) further understanding the nature of elevations on the syndrome scales, (b) screening for specific emotional and/or behavioral disorders, or (c) diagnostic decision-making.

The CBCL 1.5–5 and CBCL 6–18 should be considered for routine use in ASD *diagnostic assessment*. This is particularly important given that the DSM-5 requires specifying whether a

youth diagnosed with ASD exhibits co-occurring mental and/or behavioral disorders. For those children who do not initially present with a co-occurring disorder, regular *screening* throughout childhood should be completed as a means of monitoring for the emergence of one or more emotional or behavioral disorders. This is particularly so for critical developmental periods such as the later part of early childhood where difficulties with attention and impulsivity may interfere with full participation in an inclusive school setting and in adolescence where increasing self-awareness might increase risk for depression and anxiety. Early detection is critical to informing *treatment planning* specific to the disorder of interest. Without appropriate treatment, the co-occurring disorder might result in additional personal distress and functional impairment for the affected child which might moderate response to ASD-specific treatment. This could result in more restrictive interventions and/or placement, neither of which necessarily addresses the underlying emotional or behavioral disorder. Including the CBCL in ASD intervention *progress monitoring* can help evaluate the child's *response to any interventions* that may be implemented. Finally, the CBCL may play an important role in *eligibility determination* for educational and social services.

See Also

- [Psychotic Disorder](#)
- [Standardized Behavior Checklists](#)

References and Reading

- Achenbach, T. M. (2014). *DSM-oriented guide for the Achenbach System of Empirically Based Assessment (ASEBA)*. Burlington: University of Vermont Research Center for Children, Youth, and Families.
- Achenbach, T. M., & Rescorla, L. A. (2000). *Manual for the ASEBA preschool forms & profiles*. Burlington: University of Vermont Research Center for Children, Youth, and Families.
- Achenbach, T. M., & Rescorla, L. A. (2001). *Manual for the ASEBA school-age forms & profiles*. Burlington: University of Vermont, Research Center for Children, Youth, and Families.

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Berube, R. L., & Achenbach, T. M. (2015). *Bibliography of published studies using the Achenbach System of Empirically Based Assessment (ASEBA)*. Burlington: University of Vermont, Research Center for Children, Youth, & Families. Available online at www.ASEBA.org.
- Biederman, J., Petty, C. R., Fried, R., Wozniak, J., Micco, J. A., Henin, A., et al. (2010). Child behavior checklist clinical scales discriminate referred youth with autism spectrum disorder: A preliminary study. *Journal of Developmental and Behavioral Pediatrics*, 31(6), 485–490.
- Bolte, S., Dickhut, H., & Poustka, F. (1999). Patterns of parent-reported problems indicative in autism. *Psychopathology*, 32, 93–97.
- Duarte, C. S., Bordin, I. A. S., de Oliveira, A., & Bird, H. (2003). The CBCL and the identification of children with autism and related conditions in Brazil: Pilot findings. *Journal of Autism and Developmental Disorders*, 33(6), 703–707.
- Hurtig, T., Kuusikko, S., Mattila, M., Haapsamo, H., Ebeling, H., Jussila, K., et al. (2009). Multi-informant reports of psychiatric symptoms among high-functioning adolescents with Asperger syndrome or autism. *Autism*, 13(6), 583–598.
- Kanne, S. M., Abbacchi, A. M., & Constantino, J. N. (2009). Multi-informant ratings of psychiatric symptom severity in children with autism spectrum disorders: The importance of environmental context. *Journal of Autism and Developmental Disorders*, 39(6), 856–864.
- Kaufman, J., Birmaher, B., Brent, D., Rao, U., & Ryan, N. (1996). *Kiddie-Sads-Present and lifetime Version*. Version 1.0 of October, 1996. <http://www.wpic.pitt.edu/ksads>
- Lord, C., Rutter, M., DiLavore, P., & Risi, S. (2002). *Autism diagnostic observation schedule: Manual*. Los Angeles: Western Psychological Services.
- Magyar, C. I., & Pandolfi, V. (2017). Utility of the CBCL DSM oriented scales in assessing emotional disorders in youth with autism. *Research in Autism Spectrum Disorders*, 37, 11–20.
- Ooi, Y. P., Rescorla, L., Ang, R. P., Woo, B., & Fung, D. S. S. (2010). Identification of autism spectrum disorders using the child behavior checklist in Singapore. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-010-1015-x>.
- Pandolfi, V., Magyar, C. I., & Dill, C. A. (2009). Confirmatory factor analysis of the child behavior checklist 1.5-5 in a sample of children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(7), 986–995.
- Pandolfi, V., Magyar, C. I., & Dill, C. A. (2012). An initial psychometric evaluation of the child behavior checklist 6-18 in a sample of youth with autism spectrum disorders. *Research in Autism Spectrum Disorders*. <https://doi.org/10.1016/j.rasd.2011.03.009>.
- Pandolfi, V., Magyar, C. I., & Norris, M. (2014). Validity study of the CBCL 6-18 for the assessment of emotional problems in youth with ASD. *Journal of Mental Health Research in Intellectual Disabilities*, 7(4), 306–322.
- Rutter, M., LeCouteur, A. L., & Lord, C. (2003). *Autism diagnostic interview- revised*. Los Angeles: Western Psychological Services.

Child Language

► Normal Language Development

Child Psychotherapy

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Synonyms

Clinical psychology; Mental health interventions

Definition

A therapeutic interaction between a child (the client) and a trained therapist to alleviate the child's distress and improve functioning in everyday life. Child psychotherapy is provided by licensed clinicians (e.g., clinical psychologists, clinical social workers, child and family counselors) using a range of therapeutic approaches and strategies in order to alter feelings, thoughts, attitudes, or behaviors. The broad goals of therapy are to improve adjustment and functioning in both intrapersonal and interpersonal spheres and to reduce maladaptive behaviors. Often more specific goals are set for individual clients, depending on the therapeutic modality employed (e.g., Behavior Therapy). Child psychotherapy can include the child's parents as well as other significant members of the child's family and community.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavior Therapy](#)
- [Cognitive Behavioral Therapy \(CBT\)](#)
- [Family Therapy](#)
- [Group Therapy](#)
- [Pivotal Response Training](#)
- [Play Therapy](#)
- [Psychotherapy](#)

References and Reading

- Block, S., & Harari, E. (2001). Psychotherapy, history of: Psychiatric aspects. In N. L. Smelser & P. B. Baltes (Eds.), *International encyclopedia of the social and behavioral sciences* (pp. 12484–12491). Oxford: Elsevier.
- El-Ghoury, N. H., & Krackow, E. (2011). A developmental-behavioral approach to outpatient psychotherapy with children with autism spectrum disorders. *Journal of Contemporary Psychotherapy*, 41(1), 11–17.
- Kazdin, A. E. (2000). *Psychotherapy for children and adolescents: Directions for research and practice*. New York: Oxford University Press.
- Kazdin, A. E., & Johnson, B. (1994). Advances in psychotherapy for children and adolescents: Interrelations of adjustment, development, and intervention. *Journal of School Psychology*, 32(3), 217–246.
- Weisz, J. R., & Kazdin, A. E. (Eds.). (2010). *Evidence-based psychotherapies for children and adolescents* (2nd ed.). New York: Guilford Press.

which, “Children’s preferences guide the selection of materials; adults provide support and encourage, but do not require, that materials are used and activities are carried out in the desired way. Rather than adult-supplied consequences for certain behaviors, internal, naturally occurring reinforcers are assumed to provide the motivation for learning” (National Research Council [NRC] 2001, p. 136). However, other authors note that some children may not learn skills following a “typical” developmental progression and may not provide sufficiently diverse interests to allow educators to follow a child’s interests and therefore may be challenged to teach across variety of priority areas (Volkmar and Wiesner 2009). Volkmar and Wiesner (2009) also note that there is relatively less research on developmental, child-centered approaches than behavioral approaches, and effective implementation of child-centered approaches likely require highly skilled interventionists. The National Research Council (2001) suggested that for children with fewer appropriate initiations, a behavioral approach may be more appropriate than a child-centered approach and noted that more research is needed to demonstrate the effectiveness of child-centered approaches and relative effectiveness compared to other approaches. In contrast, a child-centered approach may be appropriate for children with a variety of interests, thus facilitating teaching across a variety of skills.

Child-Centered Approaches

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Definition

In the National Research Council’s report, *Educating Children with Autism*, the counsel characterizes child-centered approaches as those in

References and Reading

- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know*. Hoboken: Wiley.

Child-Focused Approaches

- [Interventions: Child Centered Approaches](#)

Childhood Aphasia

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Synonyms

Congenital aphasia; Developmental dysphasia;
Language disorder; Specific language impairment

Definition

Aphasia is derived from the Greek word *aphatos*, meaning “speechless,” and is characteristically used to describe the profile of language impairments seen in adults after a stroke or other focal neurological lesion. In the early nineteenth century, physicians and neurologists such as Gall (1935, cited in De Montfort Supple 2010) described seemingly similar language impairments in children. These children could not speak but had apparently normal understanding of language and did not appear to have general cognitive impairments. The term *congenital aphasia* was first used by Vaisse in 1866 (cited in De Montfort Supple 2010), and related terms such as *developmental aphasia* or *dysphasia* were widely used until the mid-twentieth century. The assumption behind the use of these terms was that the neurobiological source of language impairment in children was similar to adult case; however, in recent years, it has become clear that most developmental language disorders do not arise from focal neurological insults. Instead, anomalies in brain development are subtle and not deterministic of language ability. There is also considerable evidence that the etiology of developmental language disorders is more typically genetic, rather than the result of acquired brain damage (Bishop 2009). Finally, most investigators would agree that the boundary between language impairment and normality is somewhat arbitrary, rendering the use of a “medical” term or “disease” category inappropriate (Norbury et al. 2008).

The notion that language could be impaired in the context of “spared” capacities in other aspects of development led to labels such as *specific language impairment (SLI)* replacing *dysphasia*. However, in practice, it is rare to see such discrete linguistic impairments in a developing child, and there is continuing controversy about how best to describe children with more specific language difficulties (Bishop 2010). In addition, there is considerable debate about the nature of language impairment in autism spectrum disorders and whether some children with ASD also have a comorbid SLI (Tomblin 2011). In practice, it is preferable to describe the nature of the child’s language difficulties in detail without recourse to diagnostic labels that make assumptions about etiology.

See Also

- Language
- Language Disorder

References and Reading

- Bishop, D. V. M. (2009). Genes, cognition and communication: Insights from neurodevelopmental disorders. *The Year in Cognitive Neuroscience: Annals of the New York Academy of Sciences*, 1156, 1–18.
- Bishop, D. V. M. (2010). Which neurodevelopmental disorders get researched and why? *PLoS One*, 5(11), e15112. <https://doi.org/10.1371/journal.pone.0015112>.
- De Montfort Supple, M. (2010). Child language disability: A historical perspective. *Topics in Language Disorders*, 30, 72–78.
- Norbury, C. F., Bishop, D. V. M., & Tomblin, J. B. (2008). *Understanding developmental language disorders* (pp. xiii–xv). Hove/New York: Psychology Press.
- Tomblin, J. B. (2011). Co-morbidity of autism and SLI: Kinds, kin and complexity. *International Journal of Language & Communication Disorders*, 46(2), 127–137.

Childhood Apraxia of Speech (CAS)

- Developmental Apraxia
- Verbal Apraxia

Childhood Autism

► Autistic Disorder

Childhood Autism Rating Scale

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Abbreviations

ASD	Autism spectrum disorder
TEACCH	Treatment and education of autistic and communication related handicapped children

Synonyms

CARS; CARS, Second Edition, High-Functioning Version; CARS, Second Edition, Questionnaire for Parents or Caregivers; CARS, Second Edition, Standard Version; CARS2-HF; CARS2-QPC; CARS2-ST

Description

The CARS has a long-standing history as one of the most widely used diagnostic instruments for ASD. A trained observer rates an individual's behavior on 14 items and provides a general impressions score, each of which is rated on a 7-point Likert scale (1–4 with ½ points). Scores represent severity of deviation compared to expectations for one's peers of the same chronological age: a score of 1 represents functioning within normal limits, whereas a score of 4 represents severely abnormal functioning. The CARS total score ranges from 15 to 60 with higher scores indicating a higher probability or severity of autism. The CARS was intended to be used regardless of age or level of functioning. In

2010, the CARS was refined, and separate forms were created based on a person's developmental level. The CARS2-HF was developed for use with individuals over age 6 years, with IQs above 80, and intact verbal communication skills. The CARS2-ST (which is identical to the original CARS) continues to be used for all children under age 6 years and for individuals over age 6 years either with IQ scores of 79 or lower, or who have impaired communication skills (Schopler et al. 2010).

The 14 behavior domains from the CARS and maintained on the CARS2-ST include (1) relating to people; (2) imitation; (3) emotional response; (4) body use; (5) object use; (6) adaptation to change; (7) visual response; (8) listening response; (9) taste, smell, and touch response and use; (10) fear or nervousness; (11) verbal communication; (12) nonverbal communication; (13) activity level; (14) level and consistency of intellectual response; in addition to (15) general impressions. The CARS2-HF maintains the general structure of the CARS, but it does not include imitation and activity level. Instead, it adds social-emotional understanding and thinking/cognitive integration skills. Behavioral descriptions of several other items were also modified to be more applicable for individuals with a higher IQ (Schopler et al. 2010). When rating each domain, a rater considers the peculiarity, frequency, intensity, and duration of a behavioral concern. Brief descriptions of the behaviors to be observed are provided as anchors on the rating forms, but a more detailed description, including a definition and particular considerations for each item, is provided in the CARS2 manual.

The CARS2-QPC is a new form in the second edition. It is an unscored questionnaire completed by others who know the person being evaluated well. The CARS2-QPC can also provide information about a person's early development, which is not captured by the behavioral observations on the CARS2-ST and CARS2-HF, and provide examples of behavior concerns that the parent or caregiver notices. Schopler et al. (2010) stated that parents or other caregivers are not to complete either the CARS2-ST or CARS2-HF but should provide information on the CARS2-QPC, which

can then be used as a guide during a diagnostic or other direct interview.

The CARS surveys a wide range of behaviors. These behaviors related to different conceptualizations of ASD at the time the CARS was developed, but not all of them relate to the DSM-IV-TR or to an earlier predecessor, the DSM-III-R. Although an interested user could compare an individual's score on specific items to any of the diagnostic criteria on which the CARS is based, including the DSM-IV, a weighted score based on the current conceptualization of ASD is not available.

Historical Background

The CARS was developed by Dr. Eric Schopler and colleagues in North Carolina to complement their outpatient treatment program, Division TEACCH. It was included as part of their diagnostic process and educational planning, often being completed as part of the Psycho-educational Profile. Prior to its inception in DSM-III (American Psychiatric Association 1980), there were multiple definitions and diagnostic criteria for what is called ASD today. Schopler and colleagues developed the CARS as their own rating system to distinguish between ASD and other developmental disorders (Reichler and Schopler 1971; Schopler et al. 1980) in an effort to overcome limitations of existing classification systems and diagnostic measures. The CARS was originally called the Childhood Psychosis Rating Scale because it had a broader conceptualization than Kanner's original definition of autism. The name was changed to the CARS as the definition of autism expanded beyond Kanner's strict definition.

The behavior domains of the CARS are largely based on the British Working Party's diagnostic criteria for childhood psychosis (Reichler and Schopler 1971), but it also includes items based on Kanner's primary features of autism and the criteria proposed by Rutter and by Ritvo and Freeman (Schopler et al. 1980, 1988). Although most items were chosen because of their relation to the diagnostic criteria at the time, others were

included because of their clinical or educational relevance (e.g., object use, visual response, auditory response, and taste, smell, and touch response and use). From the time it was developed, the CARS was integrated with the TEACCH program to integrate assessment and intervention.

Psychometric Data

The CARS classifies a person as having minimal-to-no symptoms of ASD, mild-to-moderate symptoms, or severe symptoms of an ASD. Classification cutoffs were originally determined by examining the distribution of CARS scores in a sample of 537 children. Initially, a cutoff of 30 distinguished optimally between those with and without ASD (Schopler et al. 1980). However, the recommended cutoff scores have changed with time and now vary by age and CARS2 form.

In the development sample, the cutoff of 30 on the CARS had a sensitivity of .88 and specificity of .86 (Schopler et al. 1988). Other studies have found similar results with children. Some researchers, however, have recommended higher cutoffs for very young children and lower cutoffs for adolescents and adults. In one large study, a cutoff of 30 was supported among 4-year-olds, but a cutoff of 32 was optimal among 2-year-olds, since this resulted in better specificity (Chlebowski et al. 2010).

On the CARS2-ST, the cutoff of 30 was maintained for all children under age 13 years, but a cutoff of 28 best distinguished between minimal-to-no symptoms of ASD and mild-to-moderate symptoms of an ASD for children over age 13 years. The CARS2-HF also uses a cutoff of 28 to distinguish between minimal-to-no symptoms of ASD and mild-to-moderate symptoms of an ASD. In the CARS2-HF development sample, this resulted in a sensitivity of .81 and a specificity of .87 (Schopler et al. 2010).

The CARS is strongly related to level of functioning. It may falsely identify individuals with language impairments and cognitive impairments as having an ASD. This may be acceptable clinically for diagnostic screening but not for research requiring precise diagnostic distinctions. The

magnitude of the correlations between intellectual and adaptive functioning and CARS scores is quite high (approximately $r = .7$). Although the CARS2-HF was developed to address this weakness, its relationship with IQ has not been researched at the time of this writing.

The CARS has demonstrated good concordant validity with clinical judgment and with other ASD diagnostic instruments, including the Autism Diagnostic Interview-Revised, and the Autism Diagnostic Observation Schedule. It has also shown good convergence with ASD rating scales, including the Autism Behavior Checklist, Real-Life Rating Scale, and the Social Responsiveness Scale.

Evidence for the reliability and validity of the CARS was originally presented by Schopler et al. (1980) for 537 children assessed over a 10-year span as part of the TEACCH program. Internal consistency was .94. Other investigators have replicated this high level of internal consistency for the CARS and the CARS2-ST. Among the 994 participants in the CARS2-HF development sample, coefficient alpha was .96. However, several investigations of the CARS have found negative corrected item-total correlations, specifically for the consistency of intellectual response item.

Early investigations of inter-rater reliability focused on ratings made by other professionals without specialized training in ASD (Schopler et al. 1988). These and subsequent evaluations of inter-rater reliability have found high agreement on diagnostic classifications but lower agreement on specific items. Schopler et al. (2010) found similar results with the CARS2-HF development sample.

Test-retest reliability for the CARS has been examined with a range from as little as 3 months to more than 3 years between assessments. Across these studies, CARS scores are relatively stable ($r_s > .70$), though there is some evidence that scores decrease over time (e.g., Mesibov et al. 1989). Test-retest reliability has not been evaluated for the CARS2-HF at this time.

The CARS has been translated into several languages, including French, Japanese, Swedish, Icelandic, Indian, Spanish, and Korean. Diagnostic cutoffs vary for the different versions, but

overall, they have shown similar psychometric properties as the CARS. Evidence published in English for internal consistency, inter-rater reliability, and diagnostic sensitivity and specificity is available for the Japanese, Swedish, Icelandic, and Indian versions (Nordin et al. 1998; Russell et al. 2010; Saemundsen et al. 2003; Tachimori et al. 2003).

Clinical Uses

The CARS and the CARS2 were designed to be part of a comprehensive diagnostic evaluation for an ASD. Professionals other than clinicians have been shown to make reliable and valid ratings on the CARS after a modest level of training. The CARS also requires a rater to have some knowledge of age-appropriate functioning within each of the behavioral domains. With such training, the tool has been used successfully in clinical and educational settings, as part of a caregiver interview, in a chart review, and as a rating scale. Although it is possible to complete the CARS2-ST based on information from a single source, the CARS2-HF requires that multiple sources of information be considered, one of which must be a direct observation of the person being rated. Multiple sources of information and a behavioral observation are not required for the CARS2-ST but would benefit the diagnostic process. It is recommended that direct behavioral observation by the trained observer be given greater weight in scoring than other information if they conflict (Schopler et al. 2010).

Despite being designed to be completed by a trained clinician, the CARS has been used, with or without adaptations, as a parent rating scale. The CARS2 manual recommends that parents do not complete the CARS2-ST or CARS2-HF as a rating scale. Rather, the unscored CARS2-QPC should be completed, which can guide an interview and provide additional developmental information not captured on the CARS as part of the overall diagnostic process. The psychometric properties of the CARS, when used as a parent rating scale, have not been adequately studied.

The CARS has also found uses within research studies (see Schopler et al. 2010 for examples). It has provided an ASD severity rating or supported an ASD diagnosis. The CARS has also been used as an outcome measure for intervention studies, medication trials, and developmental studies. As an outcome measure, the CARS has shown to be sensitive to treatment effects and to maturational changes.

See Also

- Autism Diagnostic Interview-Revised
- Autism Diagnostic Observation Schedule
- TEACCH Transition Assessment Profile (TTAP)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- Chebowski, C., Green, J. A., Barton, M. L., & Fein, D. (2010). Using the childhood autism rating scale to diagnose autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40, 787–799.
- Mesibov, G. B., Schopler, E., Schaffer, B., & Michal, N. (1989). Use of the childhood autism rating scale with autistic adolescents and adults. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28, 538–541.
- Nordin, V., Gillberg, C., & Nydén, A. (1998). The Swedish version of the childhood autism rating scale in a clinical setting. *Journal of Autism and Developmental Disorders*, 28, 69–75.
- Reichler, R. J., & Schopler, E. (1971). Observations on the nature of human relatedness. *Journal of Autism and Childhood Schizophrenia*, 1, 283–296.
- Russell, P. S. S., Daniel, A., Russell, S., Mammen, P., Abel, J. S., Raj, L. E., et al. (2010). Diagnostic accuracy, reliability, and validity of childhood autism rating scale in India. *World Journal of Pediatrics*, 6, 141–147.
- Saemundsen, E., Magnússon, P., Smári, J., & Sigurdardóttir, S. (2003). Autism diagnostic interview-revised and the childhood autism rating scale: Convergence and discrepancy in diagnosing autism. *Journal of Autism and Developmental Disorders*, 33, 319–328.
- Schopler, E., Reichler, R. J., DeVellis, R. F., & Daly, K. (1980). Toward objective classification of childhood autism: Childhood autism rating scale (CARS). *Journal of Autism and Developmental Disorders*, 10, 91–103.
- Schopler, E., Reichler, R. J., & Renner, B. R. (1988). *The childhood autism rating scale*. Los Angeles: Western Psychological Services.
- Schopler, E., Van Bourgodiën, M. E., Wellman, G. J., & Love, S. R. (2010). *Childhood autism rating scale* (2nd ed.). Los Angeles: Western Psychological Services.
- Tachimori, H., Osada, H., & Kurita, H. (2003). Childhood autism rating scale – Tokyo version for screening pervasive developmental disorders. *Psychiatry and Clinical Neurosciences*, 57, 113–118.

Childhood Disintegrative Disorder

- Noradrenergic System

Childhood Psychosis

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Synonyms

Childhood schizophrenia

Definition

In common use, the term psychosis implies a loss of contact with reality. Typical psychotic phenomena include hallucinations (perceiving things that others do not), delusions, and other behaviors (e.g., catatonia). Often individuals with psychosis have trouble structuring their thinking (a thought disorder). In adolescents and adults, psychosis and psychotic phenomena can arise because of psychiatric or medical illness or exposure to certain substances (e.g., hallucinogenic drugs). In common use, the term is rather broad including a range of conditions. Psychiatric disorders associated with psychosis include schizophrenia and bipolar type 1 disorder (what previously was

termed manic-depressive illness). Psychotic phenomena can be seen in various other conditions and may be more likely with stress.

In children, awareness of psychosis and psychotic phenomena is a relatively historically recent phenomenon (e.g., until the work of Maudsley in the 1800s, it was assumed children were protected from such phenomena). However, the description of what we now recognize as schizophrenia (or as it was once termed *dementia praecox*) led to rapid extension to children (*dementia praecoxissima* (de Sanctis 1906)). Kanner's use of the term autism (Kanner 1943) quickly led to confusion over the issue of whether we now think of as autism is a form of schizophrenia (see Volkmar and Tsatsanis 2002) since the term autism had earlier been used to describe self-centered thinking in schizophrenia (see Volkmar 1996 for a discussion). It took several decades before it became clear that this was not in fact the case and that autistic disorder was a distinctive condition (Kolvin 1971; Rutter 1972).

Given the major changes in children's understanding of reality, the term psychosis can be problematic in childhood. Before puberty, the prototypic psychotic disorder, schizophrenia, is profoundly uncommon although psychotic phenomena can be observed, e.g., in relation to stress, or as isolated phenomena. After age 5, the presence of psychotic symptoms is more concerning, and various factors (medical conditions, drug abuse) can produce such symptoms.

See Also

► [Childhood Schizophrenia](#)

References and Reading

- de Sanctis, S. (1906). On some variations of *dementia praecox*. *Revista Sperimentali di Freniatria*, 32, 141–165.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kolvin, I. (1971). Studies in the childhood psychoses. I. Diagnostic criteria and classification. *The British Journal of Psychiatry*, 118(545), 381–384.

Rutter, M. (1972). Childhood schizophrenia reconsidered. *Journal of Autism and Childhood Schizophrenia*, 2(4), 315–337.

Volkmar, F. R. (1996). Childhood and adolescent psychosis: A review of the past 10 years. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35(7), 843–851.

Volkmar, F. R., & Tsatsanis, K. (2002). Psychosis and psychotic conditions in childhood and adolescence. In D. T. Marsh & M. A. Fristad (Eds.), *Handbook of serious emotional disturbance in children and adolescents*. New York: Wiley.

Childhood Schizophrenia

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Synonyms

[Pediatric onset schizophrenia](#); [Very early-onset schizophrenia](#)

Short Description or Definition

Childhood-onset schizophrenia is defined by onset of psychosis before age 13 and is diagnosed using unmodified DSM-IV criteria for diagnosis of adult-onset schizophrenia.

Categorization

Although the existence of childhood schizophrenia was recognized since early in the twentieth century (Kraepelin 1919; Nicolson and Rapoport 1999), the nosological status of schizophrenia in children was controversial for many years, and the Diagnostic and Statistical Manual of Mental Disorders, Second Edition (DSM-II) category “childhood schizophrenia” included other psychotic disorders in children, as well as autistic disorder, thus limiting the usefulness of early studies. The landmark studies by Kolvin (Kolvin 1971; Kolvin

et al. 1971a, b, c, d, e), however, clearly differentiated schizophrenia with onset in childhood from pervasive developmental disorders, and subsequent research over the years has established the clinical and neurobiological continuity between the childhood- and adult-onset schizophrenia. Thus, COS is more appropriately categorized as the childhood counterpart of the typical adult-onset illness (Gogtay 2008).

Epidemiology

COS is rare and difficult to diagnose. As a result, it is hard to estimate the exact incidence. Furthermore, even today, high rates of misdiagnosis remain as transient psychotic symptoms can occur in healthy children (Caplan 1994; McGee et al. 2000; Schreier 1999), and fleeting hallucinations are not uncommon in nonpsychotic pediatric patients (Lukianowicz 1969; McKenna et al. 1994b) particularly in response to anxiety and stress (Rothstein 1981). Fully developed psychotic disorders in children, however, are rare and tend to be more severe than their adult counterparts (Childs and Scriver 1986), and recent data suggest that psychotic symptoms probably exist as a continuous phenotype rather than an all-or-none phenomenon (Poulton et al. 2000).

Based on the NIMH COS study experience (described later), where over the past 20 years, we have evaluated over 3,000 referrals with a potential diagnosis of schizophrenia. However, diagnosis could be confirmed only in 122 cases to date after careful evaluation, which included inpatient observation and complete medication washout in most cases. These estimates put the approximate incidence to be about 1/300th of the adult-onset illness.

Natural History, Prognostic Factors, and Outcomes

Most reports on the natural history and course of COS come from the NIMH longitudinal study of COS.

Since 1990, children with early-onset psychosis have been recruited nationally for diagnostic screening for COS at the NIMH. Diagnosis of COS is confirmed after an extensive evaluation, which includes inpatient observation during a 3-week drug washout period. To date, 118 patients have participated in the study, including 43 boys and 31 girls with a mean age of 14.06 ± 2.67 years and mean age of onset of psychosis at 10.07 ± 1.9 years. Once the diagnosis is confirmed, a structural brain MRI scan is obtained with prospective re-scans at 2-year intervals.

The general outcome remains poor with most COS children continuing to show residual symptoms: both cognitive deficits and/or psychotic symptoms. In a recent analysis, at 2-year follow-up, almost 75% of COS patients still reported either positive or negative residual symptoms (Greenstein et al. 2008). The clinical course, in general, tends to be non-episodic (unlike that for the adult illness), chronic, and treatment refractory with most children ending up on clozapine (discussed under treatment).

Although there are no specific factors that can be detected in COS either during the premorbid or prodromal course of the illness, however, many features are more striking compared to the AOS during this period which are described under pathophysiology.

Clinical Expression and Pathophysiology

Premorbid Development

A striking phenomenological feature of COS relative to adult-onset schizophrenia appears to be the higher rates of early language, social, and motor developmental abnormalities, possibly reflecting greater impairment in early brain development. In the NIMH sample, premorbid development is defined as development prior to 1 year before psychosis onset and assessed using the Cannon-Spoor Premorbid Adjustment Scale (PAS) (Cannon-Spoor et al. 1982) and the Hollis premorbid development scale (Hollis 1995); social and speech and language impairments were the most common abnormal features in COS, which was also observed by four other

independent research centers (Alaghband-Rad et al. 1995; Asarnow and Ben-Meir 1988; Gogtay et al. 2004b; Green et al. 1992; Hollis 1995; Nicolson et al. 2000; Russell et al. 1989; Watkins et al. 1988).

Risk Factors

Obstetric Complications

An analysis comparing the obstetric records of 60 COS children and 48 healthy siblings using the Columbia Obstetrics Complication Scale (Malaspina 2003), a comprehensive measurement scale consisting of 37 variables, did not find higher incidence of obstetric complications in COS patients compared to the healthy sibling control group (Ordóñez et al. 2005).

Eye Tracking

Smooth pursuit eye movement (SPEM) disorders have been reported in 25–40% of first-degree relatives of schizophrenic probands (Holzman 2000), and other studies have suggested more striking in COS than in AOS with a bilineal pattern of inheritance (Ross et al. 1999). In a recent analysis, we compared 70 COS parents, 64 AOS parents, and 20 COS siblings to separate matched control groups and found that the effect sizes for SPEM abnormalities were higher for COS than for AOS relatives, indicating that genetic factors underlying eye-tracking dysfunction may be more salient for COS (Sporn et al. 2005b).

Familial Schizophrenia Spectrum Disorders

Schizophrenia spectrum disorders consist of schizophrenia and schizoaffective disorders on Axis I and schizotypal, paranoid, and schizoid personality disorders on Axis II (Asarnow and Ben-Meir 1988). A prior study by Asarnow et al. showed higher rates of schizophrenia spectrum diagnoses for COS relatives than for relatives of probands with attention deficit hyperactivity disorder or community controls (Asarnow et al. 2001). Similarly, as expected in our recent analyses of parental diagnosis in 97 parents of COS probands, 97 parents of AOS probands, and matched community controls, it was also found that rate of schizophrenia spectrum disorders was

higher in COS than in AOS, and both were higher than community controls supporting the continuity between COS and AOS, and more salient familial genetic risk in COS (Nicolson et al. 2003).

Familial Neurocognitive Functioning

Cognitive abnormalities, executive functioning, short-term memory, and language function are well documented as endophenotypic measures for family members in AOS (Egan et al. 2001). Particularly striking have been the decreased performance on the Trail Making Test part B (Keefe et al. 1994) and digit span (Tuulio-Henriksson et al. 2002). When we compared neuropsychological deficits in 67 parents and 24 full siblings of COS probands in comparison with matched community controls for Trail Making Tests A and B and Wechsler Intelligence Scale-Revised Digit Span and Vocabulary, COS siblings performed significantly poorer than community controls although the rates of neuropsychological abnormalities for COS were not significantly higher than for AOS (Gochman et al. 2004).

Pervasive Developmental Disorder and COS

Frequently, the diagnosis of autism or pervasive developmental disorder (PDD) has been raised early in the development in our cases, and some studies have claimed that autism per se might be a risk factor for later psychosis (Cantor et al. 1982; Clarke et al. 1989; Petty et al. 1984). In the two large studies examining this systematically, COS is preceded by and comorbid with pervasive developmental disorder in 30–50% of cases. Epidemiologic and family studies also find association between the disorders, and both disorders have evidence for accelerated trajectories of anatomic brain development at ages near disorder onset, and a growing number of shared risk genes and/or rare small chromosomal variants (micro-deletions or duplications). Thus, core neurobiological processes are likely common for subsets of these two heterogeneous clinical groups.

Neurocognitive Functioning in COS Probands

Neuropsychological function in COS has been studied in depth by Robert Asarnow and

colleagues (Asarnow 1999; Asarnow et al. 1994, 1995). While rote language skills and simple perceptual processing are not impaired, these children perform poorly on tasks involving fine motor coordination, attention, and short-term and working memory (Karatekin and Asarnow 1998). Evoked-potential studies show diminished amplitude of brain electrical activity during these tasks suggesting that allocation of necessary attentional resources is deficient, which is also shared by schizophrenic adults (Asarnow et al. 1995). It is generally established for adult schizophrenia that cognitive function deteriorates at onset of psychosis but remains stable afterward (Goldberg et al. 1993; Russell et al. 1997). Our earlier study had shown that COS children ($n = 27$) as well as MDI children ($n = 24$) share similar deficits in attention, learning, and abstraction that resembled the pattern in adult patients with schizophrenia (Kumra et al. 2000). In a recent analysis on 71 COS probands where preadmission IQ data were also available from medical and school record ($n = 27$), post-psychotic cognitive function (defined as >3 years of onset) for up to 8+ years was studied. As expected, all COS patients scored significantly below age norms, but for 46 COS patients seen systematically for follow-up, there was no post-psychotic IQ decline. Thus, in spite of greater severity and generally poor clinical outcome, there was no evidence of a longer-term degenerative cognitive process in COS (Gochman et al. 2003).

Comorbid Disorders

Comorbid psychiatric disorders, particularly DSM-defined mood and anxiety disorders, often coexist with schizophrenia (Bermanzohn et al. 2000; Green et al. 2003; Huppert and Smith 2005), although the hierarchical system for DSM limits independent diagnoses of comorbidities (Bermanzohn et al. 2000), and these disorders may often be part of (or masked by) the symptoms of the primary illness. Alternatively, it is often assumed that symptoms such as severe anxiety are the result of underlying schizophrenic process and that depressive symptoms are almost inevitable in schizophrenia; thus, the diagnoses of independent Axis I conditions are often ignored

(Bermanzohn et al. 2000). However, recent studies indicate that psychiatric comorbidities can significantly alter the presentation, clinical course, or prognosis of the illness, and thus, accurate diagnoses of comorbidities could have useful implications for disease outcome (Fenton and McGlashan 1986; Huppert et al. 2001). As no prior studies have reported comorbidities for childhood-onset schizophrenia (COS), we analyzed the prevalence of comorbid Axis I diagnoses in 76 COS cases at the time of first NIMH admission, and at 4-year follow-up ($n = 28$), and correlated the comorbid diagnoses with age of onset of psychosis, clinical ratings of illness severity, familiarity for schizophrenia spectrum disorders, and early premorbid development.

As has been seen with AOS, the most frequent comorbid diagnosis at NIMH screening was depression (54%) followed by obsessive-compulsive disorder (OCD; 21%), generalized anxiety disorder (GAD; 15%), and attention deficit hyperactivity disorder (ADHD; 15%). The rate of "any" anxiety disorder (GAD, OCD, separation anxiety, PTSD, and panic disorder combined) at screening was 42%. Diagnosis of comorbid depression correlated with poorer global assessment of severity (GAS) scores, and presence of an anxiety disorder only predicted anxiety at 4-year follow-up. No other Axis I diagnoses showed correlations with any clinical measures, and there were no significant associations between comorbid diagnoses and IQ, familiarity, medication status, premorbid functioning, or age of onset at psychosis. Interestingly, there was no "current" comorbid depression at the 4-year follow-up visit, possibly due to our high use of antidepressant treatment (45%). However, the rates of anxiety disorders did not change much at the 4-year follow-up, despite adjuvant anxiety medication use, suggesting either refractory nature of these conditions or their close association with schizophrenia pathology.

Cortical Development in COS

Morphometric studies of COS populations have provided unique insights into schizophrenia brain development. Initial COS studies using whole lobe volumetric measures showed profound and

global GM loss with ventricular expansion in COS (Gogtay 2008; Rapoport et al. 1997, 1999; Rapoport and Inoff-Germain 2000). With novel neuroimaging methodology, finer-scale brain mapping on the longitudinal data revealed that the GM loss in COS had a characteristic back-to-front (parieto-frontal-temporal) pattern of spread during adolescent years (Thompson et al. 2001) which appears to be an exaggeration of the healthy GM developmental pattern (Gogtay et al. 2004a), perhaps reflecting lack of inhibitory controls on the normal maturational GM loss (Schoop et al. 1997; Sowell et al. 2001). As the children mature and become young adults, the GM loss appears to slow down and get circumscribed to prefrontal and temporal cortices and merging into the adult schizophrenia pattern (Greenstein et al. 2006), establishing the neurobiological continuity between the two counterparts of the illness.

The GM deficits in schizophrenia may reflect a disease process that is pronounced earlier in the illness and/or at an earlier age, perhaps reflecting a stronger genetic vulnerability interacting with the early brain developmental windows (Pantelis et al. 2003) and exaggerated (dysregulated) neurodevelopment (Lieberman 1999; Lieberman et al. 2005; Woods 1998). It is also possible that the structural GM differences are most dynamic in the first years around psychosis onset and then vary with the illness over time perhaps influenced by other environmental or illness-related factors such as medication exposure. Indeed a similar pattern of brain changes has also been tracked as psychosis develops in those at risk (Pantelis et al. 2007).

The diagnostic specificity of the GM trajectories was explored by comparing individuals with COS and children who were “ruled out” as having schizophrenia (Kumra et al. 1998). A surprising 40% of those followed longitudinally from this group converted to bipolar I disorder and had pre-onset scans. The developmental trajectories for bipolar I children (with psychosis) showed a subtle but distinct pattern of cortical GM gain in left temporal cortex and loss in right temporal and bilateral subgenual cingulate cortices, pattern that has no overlap with that seen for COS (Gogtay et al. 2007b). These observations point toward

diagnostic specificity of the GM findings in COS (Gogtay et al. 2007a; b). These studies still do not address the effects of medications on “longitudinal” GM trajectories, but a recent analysis comparing GM development between COS subjects treated with clozapine and those with olanzapine showed no differences in GM trajectories (Mattai et al. 2010). Further studies are needed correlating medication exposure as a continuous measure with brain development, or on unmedicated subjects to address this question.

GM abnormalities in schizophrenia may be, at least in part, familial/trait markers (Cannon et al. 2003; Gilbert et al. 2003; Weinberger and McClure 2002; Yucel et al. 2003). We have extended this question in our studies to ask whether GM “trajectories,” rather than deficits, are endophenotypes, indicting dysregulation of development as the crucial defect. Longitudinal GM findings in 52 healthy full siblings of COS patients showed initial cortical GM deficits which not only did not progress during adolescence (unlike their COS probands) but normalized by age 20. A recent analysis using 47 non-overlapping healthy siblings matched with 48 non-overlapping healthy controls replicated these findings (Mattai et al. 2011). Several inferences can be drawn from these findings. First, the pattern of “improving GM deficits” and the localization to “prefrontal and superior temporal areas” in both COS probands and siblings point toward overall similarities in the patterns of GM development in both groups where healthy siblings show a more time limited “shift to the left” compared to the COS probands (earlier deficits which are corrected before adulthood). Second, this points to protective/restitutive factors in sibling brain development, which could relate to functional outcome (Gogtay et al. 2007a). Finally, absence of parietal deficits in healthy siblings may indicate that parietal deficits require a nongenetic trigger as supported by twin studies of adult-onset cases (Cannon et al. 2002).

The profound GM loss in COS could, in theory, be only a perceived loss resulting from the encroachment of continued white matter growth, a process that extends through at least the fourth decade (Benes 1993; Benes et al. 1994; Sowell

et al. 1999). New findings using tensor-based morphometry (TBM) showed that COS patients actually had up to 2% slower WM growth rates per year than healthy controls ($p = 0.02$, all p -values corrected), with greater effect sizes in the right hemisphere ($p = 0.006$) (Gogtay et al. 2008); thus, progressive GM deficits seen in COS do not appear secondary to WM growth (Gogtay 2008).

Genetic Studies

While rare copy number variants (CNVs) have been found to be increased for our COS population (Walsh et al. 2008), only two variants (16p11.2 and 22q11) have shown a unique anatomic brain profile (McCarthy et al. 2009; Usiskin et al. 1999). Recently, genome-wide expression analyses of brain tissue from varied postnatal ages indicated that schizophrenia susceptibility genes are overrepresented during frontal cortical development (Choi et al. 2009; Harris et al. 2009; Webster et al. 2010; Wong et al. 2009). However, given the large number of weak genetic and environmental risk factors and increasing evidence for the dimensional nature of psychosis (Polanczyk et al. 2010), it seems more and more likely that schizophrenia represents a continuum of risk involving many factors. For example, a recent population study found a ninefold risk of schizophrenia if the presence of a parent with psychosis was combined with maternal depression during pregnancy (Maki et al. 2010). Other studies have documented other gene-environmental interactions such as that between genetic risk and urban birth (van Os et al. 2004).

Evaluation and Differential Diagnosis

COS is difficult to diagnose as symptoms of psychosis appear very early in a child's life and are difficult to tease apart from other childhood phenomena such as normal imaginative play, behaviors generated by situations or due to secondary gain. Hallucinations are not uncommon in otherwise healthy children although they tend to be more serious in school-age children (Polanczyk et al. 2010; Poulton et al. 2000).

The disorders most commonly misdiagnosed as childhood-onset schizophrenia are:

1. Severe anxiety can lead to hallucination in children.
2. Affective disorders: Hallucinations are relatively common in pediatric bipolar disorder and major depression (Chambers et al. 1982; Varanka et al. 1988). However, the psychotic symptoms in these conditions tend to be mood congruent, and follow-up studies on this population generally suggest a stable clinical outcome (Garraalda 1984a; McClellan and McCurry 1999; McClellan et al. 1999; Ulloa et al. 2000).
3. Organic psychosis and substance abuse disorders (may mimic withdrawal states or negative symptoms) (Caplan et al. 1991; Garraalda 1984b).
4. Pervasive developmental disorders and childhood disintegrative disorder.
5. Children with conduct disorder and various other behavioral disturbances can show hallucinations (Garraalda 1984a, b).
6. The atypical psychosis group provisionally labeled as "multidimensionally impaired (MDI)" is an important differential diagnosis. These patients are characterized by brief, transient episodes of psychosis and perceptual disturbance, typically in response to stress, emotional lability disproportionate to precipitants, cognitive deficits as indicated by multiple deficits in information processing, no clear thought disorder, and high comorbidity with ADHD. This group of patients is not adequately characterized by existing DSM-IV categories (Kumra et al. 1998; McKenna et al. 1994; Towbin et al. 1993), and in DSM, these patients would be considered as psychosis NOS.

The psychosis of childhood-onset schizophrenia can usually be distinguished by its severe and pervasive nature and its non-episodic, unremitting course (Nicolson and Rapoport 1999). Additionally, these children show poorer premorbid functioning in social, motor, and language domains, learning disabilities, and disruptive behavior

disorders (Alaghband-Rad et al. 1995; Green et al. 1992; Hollis 1995), and although not reported in studies of the premorbid history of adult-onset schizophrenia (Done et al. 1994; Jones et al. 1994), transient autistic symptoms such as hand flapping and echolalia occur in toddler years for a substantial minority of the children (Alaghband-Rad et al. 1995; Russell et al. 1989), probably reflecting compromised early brain development.

Treatment

Although rare, childhood-onset schizophrenia is a devastating disorder, which is frequently resistant to treatment, and unfortunately, there is a narrow evidence base to guide treatment, particularly as there are no trials comparing atypical antipsychotics, which have become the mainstay of current treatment. Two prior randomized controlled trials established the superiority of typical antipsychotics over placebo in COS (Pool et al. 1976; Spencer and Campbell 1994), but only one trial had compared the efficacy and safety of two antipsychotics, demonstrating the therapeutic superiority of clozapine over the typical antipsychotic haloperidol (Kumra et al. 1996). As a result of our prior study and studies in AOS patients (Davis et al. 2003; Moncrieff 2003), clozapine has established itself as the de facto gold standard in studies establishing antipsychotic efficacy – particularly in a pediatric population.

Our recent double-blind randomized controlled trial of comparing clozapine ($n = 12$) with olanzapine ($n = 13$) showed that clozapine was associated with a significant reduction in all outcome measures, whereas olanzapine showed significant improvement only in measures of negative symptoms and in the BPRS. A direct comparison of treatment efficacy showed a significant advantage for clozapine in the alleviation of negative symptoms of schizophrenia (producing a 4% greater reduction in SANS, $p = 0.04$, effect size 0.89), which was not correlated with improvement in mood or extrapyramidal side effects. Clozapine was, however, also associated with more overall side effects, including enuresis, tachycardia, and hypertension. By 2-year follow-up,

15 patients were on clozapine, and there was evidence of sustained clinical improvement, but additional side effects emerged including lipid anomalies ($N = 3$) and seizures ($N = 1$). Both treatments were associated with marked weight gain. This study suggests that clozapine should be the drug of choice in treatment-resistant childhood-onset schizophrenia (Shaw et al. 2006).

Adverse Effects of Clozapine

Clozapine, which is a lifeline for many of the COS children, is associated with several side effects. The NIMH study has started addressing the question of how to manage these side effects so that these children can continue to stay on clozapine.

Neutropenia and Akathisia

Children and adolescents treated with clozapine have increased susceptibility to neutropenia. This can be successfully managed by addition of lithium (Sporn et al. 2003). Similarly, akathisia seen only rarely in adults on clozapine appears more common in children (6 out of 15 children recently treated with clozapine had developed akathisia) and can frequently manifest as worsening of psychotic symptoms or agitation in children, which frequently results in dosage increment. This side effect is responsive to adjunctive propranolol (Gogtay et al. 2002) treatment.

Weight Gain

Weight gain is a significant effect of atypical antipsychotics and is more pronounced in children and adolescents than in adults (Ratzoni et al. 2002). Genetic risk for weight gain on atypical antipsychotics has been suggested (polymorphism in beta3 and alpha 1A adrenergic, 5-HT2C and histamine receptors, and TNF-alpha) (Basile et al. 2001), and a number of biochemical correlates or predictors of weight gain have been reported in the literature (leptin, prolactin, triglyceride, and HDL levels).

In our recent analysis of 23 patients treated with clozapine who had at least one medication-free week, plasma levels of hormones putatively involved in weight and appetite regulation (leptin, insulin, ghrelin, adiponectin, amylin, TNF-alpha) were compared with age, sex, and BMI-matched

healthy controls. After 6 weeks on clozapine, COS children showed increases in BMI ($p = 0.001$) and leptin ($p = 0.01$). For COS patients, BMI at baseline and week 6 correlated with insulin level ($r = 0.5, p = 0.004$). In addition, increase in BMI was positively correlated with clinical improvement in CGI, SAPS, and SANS rating scales ($p < 0.05$). Our findings suggest that clozapine-induced weight gain may be associated with increased leptin, reduced adiponectin and ghrelin, and clinical improvement (Sporn et al. 2005a).

See Also

► Childhood Psychosis

References and Reading

- Alaghband-Rad, J., McKenna, K., Gordon, C. T., Albus, K. E., Hamburger, S. D., Rumsey, J. M., et al. (1995). Childhood-onset schizophrenia: The severity of premorbid course. *Journal of the American Academy of Child and Adolescent Psychiatry*, 34(10), 1273–1283.
- Asarnow, R. F. (1999). Neurocognitive impairments in schizophrenia: A piece of the epigenetic puzzle. *European Child & Adolescent Psychiatry*, 8(Suppl 1), 15–18.
- Asarnow, J. R., & Ben-Meir, S. (1988). Children with schizophrenia spectrum and depressive disorders: A comparative study of premorbid adjustment, onset pattern and severity of impairment. *Journal of Child Psychology and Psychiatry*, 29(4), 477–488.
- Asarnow, R. F., Asamen, J., Granholm, E., Sherman, T., Watkins, J. M., & Williams, M. E. (1994). Cognitive/neuropsychological studies of children with a schizophrenic disorder. *Schizophrenia Bulletin*, 20(4), 647–669.
- Asarnow, R. F., Brown, W., & Strandburg, R. (1995). Children with a schizophrenic disorder: Neurobehavioral studies. *European Archives of Psychiatry and Clinical Neuroscience*, 245(2), 70–79.
- Asarnow, R. F., Nuechterlein, K. H., Fogelson, D., Subotnik, K. L., Payne, D. A., Russell, A. T., et al. (2001). Schizophrenia and schizophrenia-spectrum personality disorders in the first-degree relatives of children with schizophrenia: The UCLA family study. *Archives of General Psychiatry*, 58(6), 581–588.
- Basile, V. S., Masellis, M., McIntyre, R. S., Meltzer, H. Y., Lieberman, J. A., & Kennedy, J. L. (2001). Genetic dissection of atypical antipsychotic-induced weight gain: Novel preliminary data on the pharmacogenetic puzzle. *The Journal of Clinical Psychiatry*, 62(Suppl 23), 45–66.
- Benes, F. M. (1993). The relationship between structural brain imaging and histopathologic findings in schizophrenia research. *Harvard Review of Psychiatry*, 1(2), 100–109.
- Benes, F. M., Turtle, M., Khan, Y., & Farol, P. (1994). Myelination of a key relay zone in the hippocampal formation occurs in the human brain during childhood, adolescence, and adulthood. *Archives of General Psychiatry*, 51(6), 477–484.
- Bermanzohn, P. C., Porto, L., Arlow, P. B., Pollack, S., Stronger, R., & Siris, S. G. (2000). Hierarchical diagnosis in chronic schizophrenia: A clinical study of co-occurring syndromes. *Schizophrenia Bulletin*, 26(3), 517–525.
- Cannon, T. D., Thompson, P. M., van Erp, T. G., Toga, A. W., Poutanen, V. P., Huttunen, M., et al. (2002). Cortex mapping reveals regionally specific patterns of genetic and disease-specific gray-matter deficits in twins discordant for schizophrenia. *Proceedings of the National Academy of Sciences of the United States of America*, 99(5), 3228–3233.
- Cannon, T. D., van Erp, T. G., Bearden, C. E., Loewy, R., Thompson, P., Toga, A. W., et al. (2003). Early and late neurodevelopmental influences in the prodrome to schizophrenia: Contributions of genes, environment, and their interactions. *Schizophrenia Bulletin*, 29(4), 653–669.
- Cannon-Spoor, H. E., Potkin, S. G., & Wyatt, R. J. (1982). Measurement of premorbid adjustment in chronic schizophrenia. *Schizophrenia Bulletin*, 8(3), 470–484.
- Cantor, S., Evans, J., Pearce, J., & Pezzot-Pearce, T. (1982). Childhood schizophrenia: Present but not accounted for. *The American Journal of Psychiatry*, 139(6), 758–762.
- Caplan, R. (1994). Thought disorder in childhood. *Journal of the American Academy of Child and Adolescent Psychiatry*, 33(5), 605–615.
- Caplan, R., Shields, W. D., Mori, L., & Yudovin, S. (1991). Middle childhood onset of interictal psychosis. *Journal of the American Academy of Child and Adolescent Psychiatry*, 30(6), 893–896.
- Chambers, W. J., Puig-Antich, J., Tabrizi, M. A., & Davies, M. (1982). Psychotic symptoms in prepubertal major depressive disorder. *Archives of General Psychiatry*, 39(8), 921–927.
- Childs, B., & Scriver, C. R. (1986). Age at onset and causes of disease. *Perspectives in Biology and Medicine*, 29(3) Pt 1, 437–460.
- Choi, K. H., Zepp, M. E., Higgs, B. W., Weickert, C. S., & Webster, M. J. (2009). Expression profiles of schizophrenia susceptibility genes during human prefrontal cortical development. *Journal of Psychiatry & Neuroscience*, 34(6), 450–458.
- Clarke, D. J., Littlejohns, C. S., Corbett, J. A., & Joseph, S. (1989). Pervasive developmental disorders and psychoses in adult life. *The British Journal of Psychiatry*, 155, 692–699.

- Davis, J. M., Chen, N., & Glick, I. D. (2003). A meta-analysis of the efficacy of second-generation antipsychotics. *Archives of General Psychiatry*, 60(6), 553–564.
- Done, D. J., Crow, T. J., Johnstone, E. C., & Sacker, A. (1994). Childhood antecedents of schizophrenia and affective illness: Social adjustment at ages 7 and 11. *BMJ*, 309(6956), 699–703.
- Egan, M. F., Goldberg, T. E., Gscheidle, T., Weirich, M., Rawlings, R., Hyde, T. M., et al. (2001). Relative risk for cognitive impairments in siblings of patients with schizophrenia. *Biological Psychiatry*, 50(2), 98–107.
- Fenton, W. S., & McGlashan, T. H. (1986). The prognostic significance of obsessive-compulsive symptoms in schizophrenia. *The American Journal of Psychiatry*, 143(4), 437–441.
- Garralda, M. E. (1984a). Hallucinations in children with conduct and emotional disorders: II. The follow-up study. *Psychological Medicine*, 14(3), 597–604.
- Garralda, M. E. (1984b). Hallucinations in children with conduct and emotional disorders: I. The clinical phenomena. *Psychological Medicine*, 14(3), 589–596.
- Gilbert, A. R., Montrose, D. M., Sahni, S. D., Diwadkar, V. A., & Keshavan, M. S. (2003). Obstetric complications correlate with neurobehavioral and brain structural alterations in young relatives at risk for schizophrenia. *Annals of the New York Academy of Sciences*, 1008, 269–275.
- Gochman, P. A., Greenstein, D. K., Sporn, A. L., Gogtay, N., Keller, B., & Rapoport, J. L. *IQ decline and stabilization in Childhood-Onset Schizophrenia*. Submitted, 2003.
- Gochman, P. A., Greenstein, D., et al. (2004). Childhood onset schizophrenia: Familial neurocognitive measures. *Schizophrenia Research*, 71(1), 43–47.
- Gogtay, N. (2008). Cortical brain development in schizophrenia: Insights from neuroimaging studies in childhood-onset schizophrenia. *Schizophrenia Bulletin*, 34(1), 30–36.
- Gogtay, N., Sporn, A., Alfaro, C. L., Mulqueen, A., & Rapoport, J. L. (2002). Clozapine-induced akathisia in children with schizophrenia. *Journal of Child and Adolescent Psychopharmacology*, 12(4), 347–349.
- Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., et al. (2004a). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Sciences of the United States of America*, 101(21), 8174–8179.
- Gogtay, N., Sporn, A., Clasen, L. S., Nugent, T. F., 3rd, Greenstein, D., Nicolson, R., et al. (2004b). Comparison of progressive cortical gray matter loss in childhood-onset schizophrenia with that in childhood-onset atypical psychoses. *Archives of General Psychiatry*, 61(1), 17–22.
- Gogtay, N., Greenstein, D., Lenane, M., Clasen, L., Sharp, W., Gochman, P., et al. (2007a). Cortical brain development in nonpsychotic siblings of patients with childhood-onset schizophrenia. *Archives of General Psychiatry*, 64(7), 772–780.
- Gogtay, N., Ordonez, A., Herman, D. H., Hayashi, K. M., Greenstein, D., Vaituzis, C., et al. (2007b). Dynamic mapping of cortical development before and after the onset of pediatric bipolar illness. *Journal of Child Psychology and Psychiatry*, 48(9), 852–862.
- Gogtay, N., Lu, A., Leow, A. D., Klunder, A. D., Lee, A. D., Chavez, A., et al. (2008). Three-dimensional brain growth abnormalities in childhood-onset schizophrenia visualized by using tensor-based morphometry. *Proceedings of the National Academy of Sciences of the United States of America*, 105(41), 15979–15984.
- Goldberg, T. E., Hyde, T. M., Kleinman, J. E., & Weinberger, D. R. (1993). Course of schizophrenia: Neuropsychological evidence for a static encephalopathy. *Schizophrenia Bulletin*, 19(4), 797–804.
- Green, W. H., Padron-Gayol, M., Hardesty, A. S., & Bassiri, M. (1992). Schizophrenia with childhood onset: A phenomenological study of 38 cases. *Journal of the American Academy of Child and Adolescent Psychiatry*, 31(5), 968–976.
- Green, A. I., Canuso, C. M., Brenner, M. J., & Wojcik, J. D. (2003). Detection and management of comorbidity in patients with schizophrenia. *The Psychiatric Clinics of North America*, 26(1), 115–139.
- Greenstein, D., Lerch, J., Shaw, P., Clasen, L., Giedd, J., Gochman, P., et al. (2006). Childhood onset schizophrenia: Cortical brain abnormalities as young adults. *Journal of Child Psychology and Psychiatry*, 47(10), 1003–1012.
- Greenstein, D. K., Wolfe, S., Gochman, P., Rapoport, J. L., & Gogtay, N. (2008). Remission status and cortical thickness in childhood-onset schizophrenia. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(10), 1133–1140.
- Harris, L. W., Lockstone, H. E., Khaitovich, P., Weickert, C. S., Webster, M. J., & Bahn, S. (2009). Gene expression in the prefrontal cortex during adolescence: Implications for the onset of schizophrenia. *BMC Medical Genomics*, 2, 28.
- Hollis, C. (1995). Child and adolescent (juvenile onset) schizophrenia. A case control study of premorbid developmental impairments. *The British Journal of Psychiatry*, 166(4), 489–495.
- Holzman, P. S. (2000). Eye movements and the search for the essence of schizophrenia. *Brain Research. Brain Research Reviews*, 31(2–3), 350–356.
- Huppert, J. D., & Smith, T. E. (2005). Anxiety and schizophrenia: The interaction of subtypes of anxiety and psychotic symptoms. *CNS Spectrums*, 10(9), 721–731.
- Huppert, J. D., Weiss, K. A., Lim, R., Pratt, S., & Smith, T. E. (2001). Quality of life in schizophrenia: Contributions of anxiety and depression. *Schizophrenia Research*, 51(2–3), 171–180.
- Jones, P., Rodgers, B., Murray, R., & Marmot, M. (1994). Child development risk factors for adult schizophrenia in the British 1946 birth cohort. *Lancet*, 344(8934), 1398–1402.
- Karatekin, C., & Asarnow, R. F. (1998). Working memory in childhood-onset schizophrenia and attention-deficit/

- hyperactivity disorder. *Psychiatry Research*, 80(2), 165–176.
- Keefe, R. S., Silverman, J. M., Roitman, S. E., Harvey, P. D., Duncan, M. A., Alroy, D., et al. (1994). Performance of nonpsychotic relatives of schizophrenic patients on cognitive tests. *Psychiatry Research*, 53(1), 1–12.
- Kolvin, I. (1971). Studies in the childhood psychoses. I. Diagnostic criteria and classification. *The British Journal of Psychiatry*, 118(545), 381–384.
- Kolvin, I., Garside, R. F., & Kidd, J. S. (1971a). Studies in the childhood psychoses. IV. Parental personality and attitude and childhood psychoses. *The British Journal of Psychiatry*, 118(545), 403–406.
- Kolvin, I., Humphrey, M., & McNay, A. (1971b). Studies in the childhood psychoses. VI. Cognitive factors in childhood psychoses. *The British Journal of Psychiatry*, 118(545), 415–419.
- Kolvin, I., Ounsted, C., Humphrey, M., & McNay, A. (1971c). Studies in the childhood psychoses. II. The phenomenology of childhood psychoses. *The British Journal of Psychiatry*, 118(545), 385–395.
- Kolvin, I., Ounsted, C., Richardson, L. M., & Garside, R. F. (1971d). Studies in the childhood psychoses. 3. The family and social background in childhood psychoses. *The British Journal of Psychiatry*, 118(545), 396–402.
- Kolvin, I., Ounsted, C., & Roth, M. (1971e). Studies in the childhood psychoses. V. Cerebral dysfunction and childhood psychoses. *The British Journal of Psychiatry*, 118(545), 407–414.
- Kraepelin, E. (Hrsg.). (1919). *Dementia praecox and paraphrenia*. Huntington: Robert E Krieger.
- Kumra, S., Frazier, J. A., Jacobsen, L. K., McKenna, K., Gordon, C. T., Lenane, M. C., et al. (1996). Childhood-onset schizophrenia. A double-blind clozapine-haloperidol comparison. *Archives of General Psychiatry*, 53(12), 1090–1097.
- Kumra, S., Jacobsen, L. K., Lenane, M., Zahn, T. P., Wiggs, E., Alagband-Rad, J., et al. (1998). “Multidimensionally impaired disorder”: Is it a variant of very early-onset schizophrenia? *Journal of the American Academy of Child and Adolescent Psychiatry*, 37(1), 91–99.
- Kumra, S., Wiggs, E., Bedwell, J., Smith, A. K., Arling, E., Albus, K., et al. (2000). Neuropsychological deficits in pediatric patients with childhood-onset schizophrenia and psychotic disorder not otherwise specified. *Schizophrenia Research*, 42(2), 135–144.
- Lieberman, J. A. (1999). Is schizophrenia a neurodegenerative disorder? A clinical and neurobiological perspective. *Biological Psychiatry*, 46(6), 729–739.
- Lieberman, J. A., Tollefson, G. D., Charles, C., Zipursky, R., Sharma, T., Kahn, R. S., et al. (2005). Antipsychotic drug effects on brain morphology in first-episode psychosis. *Archives of General Psychiatry*, 62(4), 361–370.
- Lukianowicz, N. (1969). Hallucinations in non-psychotic children. *Psychiatr Clin (Basel)*, 2(6), 321–337.
- Maki, P., Riekk, T., Miettunen, J., Isohanni, M., Jones, P. B., Murray, G. K., et al. (2010). Schizophrenia in the offspring of antenatally depressed mothers in the northern Finland 1966 birth cohort: Relationship to family history of psychosis. *The American Journal of Psychiatry*, 167(1), 70–77.
- Malaspina, D. (2003). *Columbia obstetrics complication scale*. In *Diagnostic Center for Schizophrenia Linkage Studies 2003*. New York: New York State Psychiatric Institute.
- Mattai, A., Chavez, A., Greenstein, D., Clasen, L., Bakalar, J., Stidd, R., et al. (2010). Effects of clozapine and olanzapine on cortical thickness in childhood-onset schizophrenia. *Schizophrenia Research*, 116(1), 44–48.
- Mattai, A. A., Weisinger, B., Greenstein, D., Stidd, R., Clasen, L., Miller, R., Tossell, J. W., Rapoport, J. L., & Gogtay, N. (2011). Normalization of cortical gray matter deficits in nonpsychotic siblings of patients with childhood-onset schizophrenia. *Journal of the American Academy of Child and Adolescent Psychiatry*, 50(7), 697–704.
- McCarthy, S. E., Makarov, V., Kirov, G., Addington, A. M., McClellan, J., Yoon, S., et al. (2009). Microduplications of 16p11.2 are associated with schizophrenia. *Nature Genetics*, 41(11), 1223–1227.
- McClellan, J., & McCurry, C. (1999). Early onset psychotic disorders: Diagnostic stability and clinical characteristics. *European Child & Adolescent Psychiatry*, 8 (Suppl 1), 113–119.
- McClellan, J., McCurry, C., Snell, J., & DuBose, A. (1999). Early-onset psychotic disorders: Course and outcome over a 2-year period. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38(11), 1380–1388.
- McGee, R., Williams, S., & Poulton, R. (2000). Hallucinations in nonpsychotic children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(1), 12–13.
- McKenna, K., Gordon, C. T., Lenane, M., Kaysen, D., Fahey, K., & Rapoport, J. L. (1994a). Looking for childhood-onset schizophrenia: The first 71 cases screened. *Journal of the American Academy of Child and Adolescent Psychiatry*, 33(5), 636–644.
- McKenna, K., Gordon, C. T., & Rapoport, J. L. (1994b). Childhood-onset schizophrenia: Timely neurobiological research. *Journal of the American Academy of Child and Adolescent Psychiatry*, 33(6), 771–781.
- Moncrieff, J. (2003). Clozapine v. conventional antipsychotic drugs for treatment-resistant schizophrenia: A re-examination. *The British Journal of Psychiatry*, 183, 161–166.
- Nicolson, R., & Rapoport, J. L. (1999). Childhood-onset schizophrenia: Rare but worth studying. *Biological Psychiatry*, 46(10), 1418–1428.
- Nicolson, R., Lenane, M., Singarachalu, S., Malaspina, D., Giedd, J. N., Hamburger, S. D., et al. (2000). Premorbid speech and language impairments in childhood-onset schizophrenia: Association with risk factors. *The American Journal of Psychiatry*, 157(5), 794–800.
- Nicolson, R., Brookner, F. B., Lenane, M., Gochman, P., Ingraham, L. J., Egan, M. F., et al. (2003). Parental schizophrenia spectrum disorders in childhood-onset and adult-onset schizophrenia. *The American Journal of Psychiatry*, 160(3), 490–495.

- Ordonez, A. E., Bobb, A., Greenstein, D., Baker, N., Sporn, A., Lenane, M., et al. (2005). Lack of evidence for elevated obstetric complications in childhood onset schizophrenia. *Biological Psychiatry*, 58(1), 10–15.
- Pantelis, C., Velakoulis, D., McGorry, P. D., Wood, S. J., Suckling, J., Phillips, L. J., et al. (2003). Neuroanatomical abnormalities before and after onset of psychosis: A cross-sectional and longitudinal MRI comparison. *Lancet*, 361(9354), 281–288.
- Pantelis, C., Velakoulis, D., Wood, S. J., Yucel, M., Yung, A. R., Phillips, L. J., et al. (2007). Neuroimaging and emerging psychotic disorders: The Melbourne ultra-high risk studies. *International Review of Psychiatry*, 19(4), 371–381.
- Petty, L. K., Ornitz, E. M., Michelman, J. D., & Zimmerman, E. G. (1984). Autistic children who become schizophrenic. *Archives of General Psychiatry*, 41(2), 129–135.
- Polanczyk, G., Moffitt, T. E., Arseneault, L., Cannon, M., Ambler, A., Keefe, R. S., et al. (2010). Etiological and clinical features of childhood psychotic symptoms: Results from a birth cohort. *Archives of General Psychiatry*, 67(4), 328–338.
- Pool, D., Bloom, W., Mielke, D. H., Roniger, J. J. J., & Gallant, D. M. (1976). A controlled evaluation of loxitane in seventy-five adolescent schizophrenic patients. *Current Therapeutic Research, Clinical and Experimental*, 19(1), 99–104.
- Poulton, R., Caspi, A., Moffitt, T. E., Cannon, M., Murray, R., & Harrington, H. (2000). Children's self-reported psychotic symptoms and adult schizophreniform disorder: A 15-year longitudinal study. *Archives of General Psychiatry*, 57(11), 1053–1058.
- Rapoport, J. L., & Inoff-Germain, G. (2000). Update on childhood-onset schizophrenia. *Current Psychiatry Reports*, 2(5), 410–415.
- Rapoport, J. L., Giedd, J., Kumra, S., Jacobsen, L., Smith, A., Lee, P., et al. (1997). Childhood-onset schizophrenia. Progressive ventricular change during adolescence. *Archives of General Psychiatry*, 54(10), 897–903.
- Rapoport, J. L., Giedd, J. N., Blumenthal, J., Hamburger, S., Jeffries, N., Fernandez, T., et al. (1999). Progressive cortical change during adolescence in childhood-onset schizophrenia. A longitudinal magnetic resonance imaging study. *Archives of General Psychiatry*, 56(7), 649–654.
- Ratzoni, G., Gothelf, D., Brand-Gothelf, A., Reidman, J., Kikinzon, L., Gal, G., et al. (2002). Weight gain associated with olanzapine and risperidone in adolescent patients: A comparative prospective study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(3), 337–343.
- Ross, R. G., Olincy, A., Harris, J. G., Radant, A., Hawkins, M., Adler, L. E., et al. (1999). Evidence for bilineal inheritance of physiological indicators of risk in childhood-onset schizophrenia. *American Journal of Medical Genetics*, 88(2), 188–199.
- Rothstein, A. (1981). Hallucinatory phenomena in childhood. A critique of the literature. *Journal of the American Academy of Child Psychiatry*, 20(3), 623–635.
- Russell, A., Bott, L., & Sammons, C. (1989). The phenomena of schizophrenia occurring in childhood. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28, 399–407.
- Russell, A. J., Munro, J. C., Jones, P. B., Hemsley, D. R., & Murray, R. M. (1997). Schizophrenia and the myth of intellectual decline. *The American Journal of Psychiatry*, 154(5), 635–639.
- Schoop, V. M., Gardziella, S., & Muller, C. M. (1997). Critical period-dependent reduction of the permissiveness of cat visual cortex tissue for neuronal adhesion and neurite growth. *European Journal of Neuroscience*, 9(9), 1911–1922.
- Schreier, H. A. (1999). Hallucinations in nonpsychotic children: More common than we think? *Journal of the American Academy of Child and Adolescent Psychiatry*, 38(5), 623–625.
- Shaw, P., Sporn, A., Gogtay, N., Overman, G. P., Greenstein, D., Gochman, P., et al. (2006). Childhood-onset schizophrenia: A double-blind, randomized clozapine-olanzapine comparison. *Archives of General Psychiatry*, 63(7), 721–730.
- Sowell, E. R., Thompson, P. M., Holmes, C. J., Jernigan, T. L., & Toga, A. W. (1999). In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. *Nature Neuroscience*, 2(10), 859–861.
- Sowell, E. R., Thompson, P. M., Tessner, K. D., & Toga, A. W. (2001). Mapping continued brain growth and gray matter density reduction in dorsal frontal cortex: Inverse relationships during postadolescent brain maturation. *Journal of Neuroscience*, 21(22), 8819–8829.
- Spencer, E. K., & Campbell, M. (1994). Children with schizophrenia: Diagnosis, phenomenology, and pharmacotherapy. *Schizophrenia Bulletin*, 20(4), 713–725.
- Sporn, A., Gogtay, N., Ortiz-Aguayo, R., Alfaro, C., Tossell, J., Lenane, M., et al. (2003). Clozapine-induced neutropenia in children: Management with lithium carbonate. *Journal of Child and Adolescent Psychopharmacology*, 13(3), 401–404.
- Sporn, A. L., Bobb, A. J., Gogtay, N., Stevens, H., Greenstein, D. K., Clasen, L. S., et al. (2005a). Hormonal correlates of clozapine-induced weight gain in psychotic children: An exploratory study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44(9), 925–933.
- Sporn, A., Greenstein, D., Gogtay, N., Sailer, F., Hommer, D. W., Rawlings, R., et al. (2005b). Childhood-onset schizophrenia: Smooth pursuit eye-tracking dysfunction in family members. *Schizophrenia Research*, 73 (2–3, 243), –252.
- Thompson, P. M., Vidal, C., Giedd, J. N., Gochman, P., Blumenthal, J., Nicolson, R., et al. (2001). Mapping adolescent brain change reveals dynamic wave of accelerated gray matter loss in very early-onset schizophrenia. *Proceedings of the National Academy of Sciences of the United States of America*, 98(20), 11650–11655.
- Towbin, K. E., Dykens, E. M., Pearson, G. S., & Cohen, D. J. (1993). Conceptualizing “borderline syndrome of childhood” and “childhood schizophrenia” as a

developmental disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32(4), 775–782.

- Tuulio-Henriksson, A., Haukka, J., Partonen, T., Varilo, T., Paunio, T., Ekelund, J., et al. (2002). Heritability and number of quantitative trait loci of neurocognitive functions in families with schizophrenia. *American Journal of Medical Genetics*, 114(5), 483–490.
- Ulloa, R. E., Birmaher, B., Axelson, D., Williamson, D. E., Brent, D. A., Ryan, N. D., et al. (2000). Psychosis in a pediatric mood and anxiety disorders clinic: Phenomenology and correlates. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(3), 337–345.
- Usiskin, S. I., Nicolson, R., Krasnewich, D. M., Yan, W., Lenane, M., Wudarsky, M., et al. (1999). Velocardiofacial syndrome in childhood-onset schizophrenia. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38(12), 1536–1543.
- van Os, J., Pedersen, C. B., & Mortensen, P. B. (2004). Confirmation of synergy between urbanicity and familial liability in the causation of psychosis. *The American Journal of Psychiatry*, 161(12), 2312–2314.
- Varanka, T. M., Weller, R. A., Weller, E. B., & Fristad, M. A. (1988). Lithium treatment of manic episodes with psychotic features in prepubertal children. *The American Journal of Psychiatry*, 145(12), 1557–1559.
- Walsh, T., McClellan, J. M., McCarthy, S. E., Addington, A. M., Pierce, S. B., Cooper, G. M., et al. (2008). Rare structural variants disrupt multiple genes in neurodevelopmental pathways in schizophrenia. *Science*, 320(5875), 539–543.
- Watkins, J. M., Asarnow, R. F., & Tanguay, P. E. (1988). Symptom development in childhood onset schizophrenia. *Journal of Child Psychology and Psychiatry*, 29(6), 865–878.
- Webster, M. J., Elashoff, M., & Weickert, C. S. (2010). Molecular evidence that cortical synaptic growth predominates during the first decade of life in humans. *International Journal of Developmental Neuroscience*, 29, 225–236.
- Weinberger, D. R., & McClure, R. K. (2002). Neurotoxicity, neuroplasticity, and magnetic resonance imaging morphometry: What is happening in the schizophrenic brain? *Archives of General Psychiatry*, 59(6), 553–558.
- Wong, J., Webster, M. J., Cassano, H., & Weickert, C. S. (2009). Changes in alternative brain-derived neurotrophic factor transcript expression in the developing human prefrontal cortex. *European Journal of Neuroscience*, 29(7), 1311–1322.
- Woods, B. T. (1998). Is schizophrenia a progressive neurodevelopmental disorder? Toward a unitary pathogenetic mechanism. *The American Journal of Psychiatry*, 155(12), 1661–1670.
- Yucel, M., Wood, S. J., Phillips, L. J., Stuart, G. W., Smith, D. J., Yung, A., et al. (2003). Morphology of the anterior cingulate cortex in young men at ultra-high risk of developing a psychotic illness. *The British Journal of Psychiatry*, 182, 518–524.

Childhood-Onset Pervasive Developmental Disorder

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Synonyms

[Pervasive developmental disorder not otherwise specified](#); [Pervasive developmental disorders](#)

Definition

The term childhood-onset pervasive developmental disorder (COPDD) was originally used in the DSM-III. The essential features of COPDD are profound disturbance in social relations and multiple oddities of behavior all developing after 30 months of age and before 12 years (American Psychiatric Association [APA] 1980). This category was intended to capture the children who presented with features of pervasive developmental disorders but developed the disorder after 30 months of age. In the DSM-III-Revised, the category was removed and the category pervasive developmental disorder not otherwise specified (PDDNOS) was added. PDDNOS now refers to those children who do not meet the criteria for a specific pervasive developmental disorder but demonstrate features.

See Also

- [DSM-III](#)
- [Infantile Autism](#)
- [Pervasive Developmental Disorder](#)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

Children with Autism in Foster Care

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Definition

Foster care is a system in which children, youth, and young adults are temporarily placed outside of their birth family's home. The state maintains oversight of this child-focused system through the involvement of the courts and child protection service agencies. Increasingly, most states have recognized that group care, in which young people are placed in group homes or in a residential setting, is not the best option for youth in need of out-of-home care. As such, other options, such as family care or kinship care and foster parenting, have undergone extensive and robust development and have been fairly widely utilized in various states. The general types of foster care include family/kinship care where the youth is placed with someone related by biology or marriage, placement in emergency shelters, traditional county-level foster care (placement with a certified foster parent in their home), therapeutic foster care (placement with certified foster parents in their home with therapeutic and skill development activities built in), group homes which are within a community setting, and residential placements which offer the highest level of structure and are typically located in a more secure campus-like setting. Residential-based services sometimes include diagnostic settings to help assess treatment options and the development of a youth's individualized program (VanBergeijk and McGowan 2001). The reasons for removal from the family home can include various types of abuse and neglect which frequently may, but do

not always, result in the termination of parental rights. In these cases, guardianship may shift to a relative or to the local department of social services. The type of foster care placement is often impacted by the availability of appropriate placements and is determined by the needs of the specific youth. The primary purpose of foster placement is to provide safety for the youth removed from their family's home. Once in the foster care system, safety, well-being, and permanency are the primary goals typically.

Historical Background

In 1853, Charles Loring Brace founded the Children's Aid Society (CAS) in New York City. The purpose of the CAS was to provide education and housing for homeless youth due to his personal concern over the number of youth who engaged in begging and stealing due to the lack of appropriate living conditions (Ramsey 2007). The Department of Health and Human Services held the first White House conference on the Care of Dependent Children in 1909. Prior to this conference came the creation of the Children's Bureau to oversee the practices "pertaining to the welfare of children and child life among all classes of our people." The establishment of the Children's Bureau led to the standardization of practices governing foster care. In 1974, the Child Abuse Prevention and Treatment Act (CAPTA) of 1974, P.L. 93–247, was passed. The purpose of this law was "to provide financial assistance for a demonstration program for the prevention, identification, and treatment of child abuse and neglect." Subsequently, it was revised in 1978 to help promote healthy development and free individuals for adoption. Since the initial passing of P.L. 93–247, much additional legislation has been passed in the United States dealing with child protection, including the reauthorizing and amending of CAPTA several times, most recently in 2010, and the Preventing Sex Trafficking and Strengthening Families ACT, P.L. 113–183 in 2014. For a full historical account of relevant legislation, see <https://cb100.acf.hhs.gov/childrens-bureau-timeline>.

Foster Care and Autism

Every decade, a federally mandated study is conducted on abuse and neglect to identify the incidence, both reported and unreported, of abuse and neglect of children. The fourth such study, conducted in 2010, covering years 2005–2006 indicated that almost 3 million children (1 in 25) were endangered, with 1.25 million of those experiencing within this time period http://www.acf.hhs.gov/sites/default/files/opre/nis4_report_exec_summ_pdf_jan2010.pdf. Of that number, 29% experienced abuse as opposed to the remainder who experienced neglect. Of those neglected, approximately 72% were either educationally or emotionally neglected. According to the Department of Health and Human Services, there has been a decreasing trend in the number of youth in foster care leading up to 2005, when the number of youth placed out of home was approximately 511,000. Between 2017 and 2018, there was a decrease of approximately 3,388 youth (<http://www.acf.hhs.gov/sites/default/files/cb/afcarsreport26.pdf>). In 2018, the number of youth estimated to be in out-of-home foster care was 437,283.

The estimated number of youth with intellectual and developmental disabilities (IDD) in foster care nationally ranges from 28% to more than 50% (Lightfoot et al. 2011; Ringeisen et al. 2008). With over 30% of the children currently in foster care aged 3 years or younger, identification of developmental delays and early intervention services is critical to the well-being of children within the foster care system (US Department of Health and Human Services 2019). Although children raised in residential foster care display fewer social deficits and ASD symptoms than children raised in institutional settings (Levin et al. 2015), young children enter the child welfare system with significant developmental needs that often are not properly addressed (Casanueva et al. 2008). Systematic screening of all children in the foster care system performed by caregivers can increase the identification of developmental disabilities, including autism among foster care youth (Jee et al. 2010). Screening by developmental pediatricians and other medical

professionals who work with youth in foster care is also critical to identification of autism and improving opportunities for appropriate treatment.

Even when autism is identified, youth with autism spectrum disorders (ASD) are at a much greater risk for poor outcomes and greater lengths of stay in the foster care system (Bilaver and Havlicek 2013). One important contributor to poor outcomes is that individuals in foster care have increased exposure to adverse childhood experiences (ACEs) such as being abused, repeatedly witnessing violence against others, or other forms of trauma. Berg et al. (2016) found that having autism in childhood can in itself be significantly associated with a high number of ACEs. To add to this fact, the impact of foster care plus ASD places these youth at a much higher risk for exposure to ACEs. Youth with autism may be at an even greater risk, for example, for physical abuse, a prominent ACE, as compared to peers with other disabilities and peers without disabilities. The findings of Berg et al. (2016) are consistent with the findings for other children with communication difficulties (Sullivan and Knutson 1998). According to the Centers for Disease Control and Prevention (CDC; Felitti et al. 1998), these experiences are major risk factors for illness, death, social problems such as challenging behavior (e.g., aggression, self-injury), and disability. Similarly, scientists at the Center for the Developing Brain found that ACEs and their associated risk factors have serious implications for disruptions in brain development, particularly when experienced early in life. These disruptions to development may lead to difficulties with emotional regulation and attention difficulties, among other challenges (Center on the Developing Child 2012). The combined risk factors of ASD and ACEs can make the outcomes for these youth quite poor, particularly if they do not receive appropriate assessment or care (Kerns et al. 2015; Simms et al. 2000). Of additional concern for individuals with ASD is the likelihood that these youth may be placed into foster care placement not due to abuse and neglect but rather due to inability of the biological parent(s) to provide adequate support and care given the significant

challenges that parents of youth with ASD face (e.g., challenging behavior) (Estes et al. 2009). Other risk factors attributed to the concomitant impact of both ASD and foster care placement are the number of transitions between homes and providers due to externalizing behaviors (e.g., aggression), which are significant risk factors for placement disruptions. These behaviors may occur at an even higher rate than typical for persons with autism as a result of experiencing frequent transitions in living arrangements due to the difficulty many individuals with ASD have with transitions (Barber et al. 2001). Individuals in foster care often transition between home, community, and residential care settings with some frequency. In fact, data from 2010 revealed that 14.9% of children experienced at least three out-of-home placements in less than a year (Children's Bureau, Administration for Children and Families, US Department of Health and Human Services 2012).

An additional concerning factor affecting foster placement stability for individuals with autism may be the lack of expression of emotion and difficulties with communication between the foster child and foster parent/caregivers (Bernedo et al. 2015). Foster parents typically are motivated to provide care because they believe they are making a difference. When a child can express themselves through words and can show displays of affection toward the foster parent, often the relationship blossoms, and the mutual commitment strengthens. Given communication and expression of emotions are common concerns for individuals with ASD, foster parents may frustrate more easily and may not experience the same level of gratification as with "typical youth." For this, and other reasons (e.g., challenging behavior), foster parents may choose to disrupt the placement more often than with individuals with autism. Finally, placement disruptions, which are highly problematic for all children in care, may be exacerbated by issues affecting individuals with autism. Specifically, issues with change, need for consistency, and the need for sameness/routine can have a significant effect on an individual both emotionally and behaviorally in all settings but particularly when the stability of placement is

so tenuous. For example, placement disruptions can impact an individual's academic performance, with a loss of 4 months of instruction for every move (Mehana and Reynolds 2004). Coupling this with the difficulty individuals with ASD often have with changes in their environment, there is likely to be a significantly greater impact on their functioning.

Future Directions

One way to mitigate the impact of these traumatic experiences for individuals with ASD is to provide permanency (i.e., stable foster placement, adoptive placement, or return to biological family with necessary supports). Given the particular difficulties with transitions that youth with ASD experience, finding a stable placement may be especially important; however, according to Cooley et al. (2015), challenging behaviors, such as those displayed by individuals with ASD, can lower foster parents' satisfaction and motivation to continue to provide care, leading to changes in placement. Adding to this, the particular difficulty of youth with autism to communicate their needs and to display affection to a foster parent, successful placement and stability may be elusive for many individuals with ASD.

Targeted resources and programming must be built into the significantly overtaxed foster care system to both prevent the need for foster care placement and ensure the successful placement and, whenever possible, reunification with the child/youth/young adult's birth family. First, it is critical to teach Departments of Social Services (DSS) case workers who first come into contact with families in need to recognize the signs and needs associated with a diagnosis of ASD and ACEs. This is important to identify in both parents and the youth. Undiagnosed developmental concerns in a parent can lead to challenges in raising a child, particularly one who also has a developmental disability, such as ASD. Furthermore, research suggests that children of mothers who have experienced a high level of ACEs (>3) were 2.2 times more likely to have developmental delay (Folger et al. 2018). Understanding the

needs of the birth parents is critical in promoting support for reunification and obtaining the appropriate services for the family. Additionally, identification of ASD symptoms in the youth may be critical in ensuring the right services are obtained when removing the youth from their home.

Other services that may be critical include access to quality medical care and diagnostics (e.g., to accurately diagnose ASD and/or co-occurring mental health concerns); parent training for both birth parents and foster parents (e.g., Bearss et al. 2015); linkages with early intervention resources, educational resources, and/or vocational resources; and placement in a highly trained therapeutic foster care home when appropriate. Access to evidence-based clinical tools to assess trauma and ACEs in people with ASD do not yet exist (Fuld 2018; Berg et al. 2016). However, using assessment scales to evaluate ACEs like those used by Berg et al. (2016) could be useful in evaluating trauma and guiding treatment.

Access to evidence-based treatments for individuals with ASD in foster care are unfortunately often minimal or nonexistent, including access to applied behavior analysis (ABA) services. Additionally, evidence-based treatments for trauma in children, youth, and young adults such as cognitive behavior therapies (e.g., TF-CBT, DBT) must be evaluated for use with individuals with ASD to mitigate the effects of exposure to adverse childhood events.

References and Reading

- Barber, J. G., Delfabbro, P. H., & Cooper, L. (2001). The predictors of unsuccessful transition to foster care. *Journal of Child Psychology and Psychiatry*, 42(06), 785–790.
- Bearss, K., Johnson, C., Smith, T., Lecavalier, L., Swiezy, N., Aman, M., . . . , & Sukhodolsky, D.G. (2015). Effect of parent training vs parent education on behavioral problems in children with autism spectrum disorder: A randomized clinical trial. *JAMA*, 313, 1524–1533.
- Berg, K. L., Shiu, C. S., Acharya, K., Stolbach, B. C., & Msall, M. E. (2016). Disparities in adversity among children with autism spectrum disorder: A population based study. *Developmental Medicine & Child Neurology*, 58, 1124–1131.
- Bernedo, I. M., García-Martín, M. A., Salas, M. D., & Fuentes, M. J. (2015). Placement stability in non-kinship foster care: Variables associated with placement disruption. *European Journal of Social Work*, 1–14.
- Bilaver, L. A., & Havlicek, J. (2013). Foster children with autism spectrum disorder: Prevalence, length of stay, and placement patterns. *Journal of Public Child Welfare*, 7(5), 496–519.
- Brannan, A. M., Heflinger, C. A., & Bickman, L. (1997). The caregiver strain questionnaire: Measuring the impact on the family of living with a child with serious emotional disturbance. *Journal of Emotional and Behavioral Disorders*, 5(4), 212–222.
- Casanueva, C. E., Cross, T. P., & Ringeisen, H. (2008). Developmental needs and individualized family service plans among infants and toddlers in the child welfare system. *Child Maltreatment*, 13(3), 245–258.
- Center on the Developing Child at Harvard University. (2012). *The science of neglect: The persistent absence of responsive care disrupts the developing brain: Working Paper No. 12*. Retrieved from www.developingchild.harvard.edu
- Children's Rights. (2006). *Forgotten children: Children with disabilities in foster care, policy report*. New York: Children's Rights.
- Cooley, M. E., Farineau, H. M., & Mullis, A. K. (2015). Child behaviors as a moderator: Examining the relationship between foster parent supports, satisfaction, and intent to continue fostering. *Child Abuse & Neglect*, 45, 46–56.
- Estes, A., Munson, J., Dawson, G., Koehler, E., Zhou, X. H., Estes, A., Munson, J., Dawson, G., Koehler, E., Zhou, X. H., & Abbott, R. (2009). Parenting stress and psychological functioning among mothers of preschool children with autism and developmental delay. *Autism*, 13(4), 375–387.
- Felitti, V.J., Anda, R.F., Nordenberg, D., Williamson, D.F., Spitz, A.M., Edwards, V., . . . , & Marks, J.S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, 14, 245–258.
- Folger, A. T., Eismann, E. A., Stephenson, N. B., Shapiro, R. A., Macaluso, M., Brownrigg, M. E., & Gillespie, R. J. (2018). Parental adverse childhood experiences and offspring development at 2 years of age. *Pediatrics*, 141(4). <https://doi.org/10.1542/peds.2017-2826>.
- Fuld, S. (2018). Autism spectrum disorder: The impact of stressful and traumatic life events and implications for clinical practice. *Clinical Social Work Journal*, 46(3), 210–219.
- Jee, S. H., Szilagyi, M., Ovenshire, C., Norton, A., Conn, A. M., Blumkin, A., & Szilagyi, P. G. (2010). Improved detection of developmental delays among young children in foster care. *Pediatrics*, 125(2), 282–289.
- Kerns, C. M., Newschaffer, C. J., & Berkowitz, S. J. (2015). Traumatic childhood events and autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(11), 3475–3486.
- Levin, A. R., Fox, N. A., Zeanah, C. H., Jr., & Nelson, C. A. (2015). Social communication difficulties and

- autism in previously institutionalized children. *Journal of the American Academy of Child & Adolescent Psychiatry*, 54(2), 108–115.
- Lightfoot, E., Hill, K., & LaLiberte, T. (2011). Prevalence of children with disabilities in the child welfare system and out of home placement: An examination of administrative records. *Children and Youth Services Review*, 33, 2069–2075.
- Mehana, M., & Reynolds, A. J. (2004). School mobility and achievement: A meta-analysis. *Children and Youth Services Review*, 26, 93–119.
- Ramsey, P. J. (2007). Wrestling with modernity: Philanthropy and the children's aid society in progressive-era new York City. *New York History*, 88(2), 153–174.
- Ringeisen, H., Casanueva, C., Urato, M., & Cross, T. (2008). Special health care needs among children in the child welfare system. *Pediatrics*, 122(1), 232–241.
- Simms, M. D., Dubowitz, H., & Szilagyi, M. A. (2000). Health care needs of children in the foster care system. *Pediatrics*, 106(3), 909–918.
- Sullivan, P. M., & Knutson, J. F. (1998). The association between child maltreatment and disabilities in a hospital-based epidemiological study. *Child Abuse & Neglect*, 22, 271–288.
- VanBergeijk, E., & McGowan, B. (2001). Children in foster care. In A. Gitterman (Ed.), *Handbook of social work practice with vulnerable and resilient populations* (pp. 399–434). New York: Columbia University Press.

Children's Communication Checklist (CCC-2)

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Synonyms

CCC-2; [Children's communication checklist, version 2](#)

Description

The Children's Communication Checklist (CCC-2) is a checklist that is used to assess aspects of everyday communication that are difficult to evaluate using traditional language tests. It consists of 70 items divided into 10 subscales and is usually completed by a parent or other

caregiver, though useful information can be provided by teachers or other professionals who know the child well. It takes between 5 and 15 min to complete.

The ten scales are:

- A. Speech
- B. Syntax
- C. Semantics
- D. Coherence
- E. Inappropriate initiation (initiation in US version)
- F. Stereotyped language (scripted language in US version)
- G. Use of context
- H. Nonverbal communication
- I. Social relations
- J. Interests

The first four scales, A to D, assess aspects of language structure, vocabulary, and discourse. These are all areas that are often impaired in non-autistic as well as autistic children with language impairments.

The next four scales, E to H, cover aspects of communication that are not easy to assess using conventional language assessments but which are often impaired in children with autistic spectrum disorders.

The last two scales, I and J, assess behaviors that are usually impaired in cases of autistic spectrum disorder.

For each scale, there are seven items, five describing difficulties and two describing strengths. The first 50 items focus on children's difficulties, with items from different scales interleaved, and the last 20 items describe children's strengths. For each item, the respondent completes a rating reflecting the frequency with which a behavior is observed.

- Less than once a week (or never)
- At least once a week, but not every day
- Once or twice a day
- Several times (more than twice) a day (or always)

Uses of the CCC-2

The CCC-2 can be used in three ways:

1. To give a quantitative estimate of pragmatic language impairments in children.
2. To screen children for risk of language impairment. Those identified as at risk can then be referred for more detailed language assessment.
3. To help identify children who may merit further assessment for an autistic spectrum disorder. It is important to stress that CCC-2 cannot be used to diagnose autistic disorder; however, a finding of low scores on scales E to H, plus evidence of impairment on scales I and J, indicates that a more detailed diagnostic evaluation for autism is merited.

Application and Availability

Norms are available for both UK and US standardization samples over the age range 4–16 years. Both UK and US versions are published by Pearson Publishing. An electronic scorer comes with the checklist and is recommended as manual scoring is complex.

Some of the items in the CCC-2 are not suitable for describing adult communication. A modification of the CCC-2, the CC-A, was therefore developed and normed for adults in 2009.

In addition, a self-report version, CC-SR, suitable for literate teenagers and adults was developed in 2009, with UK norms.

Historical Background

Checklist for Language-Impaired Children (CLIC and CLIC-2)

CCC-2's origins were in CLIC, a research instrument that was devised as a means of identifying from within a language-impaired sample those children with a clinical picture of "semantic-pragmatic disorder." This subgroup had been described clinically, and included children who spoke in long and fluent sentences but whose use of language was strange. Utterances may be tangential, off-topic, or long and rambling. The original CLIC had 20 multiple-choice items, with the respondent selecting which of five descriptions best described the child. CLIC was piloted with teachers and therapists but was found to be unsatisfactory because respondents

often felt none of the provided options described the child. Accordingly, the format was revised to create CLIC-2 in which each item described a single communicative behavior which was rated as "applies definitely," "applies somewhat," or "does not apply."

A large-scale reliability study with CLIC-2 was conducted at special schools for language-impaired children using ratings by teachers and therapists. This too was not entirely satisfactory, with inter-rater reliability being low for some items.

The Children's Communication Checklist: Original Version

The original Children's Communication Checklist was developed from CLIC-2 by selecting those items with highest inter-rater reliability and grouping these into new scales on the basis of statistical criterion of internal consistency. This gave a checklist with nine scales: A, speech; B, syntax; C, inappropriate initiation; D, cohesion; E, stereotyped conversation; F, use of context; G, rapport; H, interests; and I, social interaction.

A validation study was conducted with the CCC using a subset of children who had participated in a national study of language-impaired 7-year-olds. Their teachers and therapists completed CCCs independently for the same children, making it possible to assess inter-rater agreement. Inter-rater reliability varied from scale to scale but was good for a pragmatic composite and reasonable for other scales. The distribution of CCC ratings also differentiated children who were categorized on clinical grounds into cases of definite, possible, or no semantic-pragmatic disorder.

Up to this point, the CCC was used only to subclassify children already known to have a communication impairment. However, there was growing interest in its potential in a broader context, both as a screening tool for language and communication problems and as a means of identifying pragmatic difficulties in children with psychiatric impairments. In addition, there seemed to be potential to extend data on the CCC to a broader age range and to explore whether it would yield valid data with parents as respondents.

To consider these questions, a further study with the CCC was carried out in collaboration with Dr. Gillian Baird, a developmental pediatrician at a tertiary referral center in London, with results being published in 2001. CCC data were gathered from a sample of children aged 5–16 years who were referred to the center for diagnostic assessment. Two copies of the CCC were sent to parents with their letter of appointment, and they were asked to have the child's teacher or therapist complete one copy and to complete the other themselves. In addition, CCC data were collected from 31 typically developing children. Agreement between parent and teacher ratings was only modest ($r = 0.45$), but both sets of ratings showed association with the child's clinical diagnosis, with the parent ratings giving particularly strong association. Scores for the typically developing children showed little overlap with those from the clinical sample, suggesting that the CCC might be useful as a means of screening for communication problems in general as well as of identifying pragmatic difficulties. In addition, this study indicated that children with a diagnosis of autism obtained very low scores on the CCC overall.

Development of CCC-2

When CCC had been in use for a few years, it was decided to develop a new version of the checklist for standardization in the UK. One major change between CCC and CCC-2 was in the response format. It was decided that a more concrete rating of frequency of observing a behavior would be less subjective and easier to use than the original response options. The CCC-2 also had the same number of items for all of the scales and gave more emphasis to items assessing non-pragmatic aspects of communication such as speech and syntax. Tables are provided to transform raw scores on each scale to age-scaled scores with mean 10 and SD 3. CCC-2 also provided norms for two composite scores. The first, the General Communication Composite, is based on all the communication scales (A to H). This is effective in discriminating children with *any* clinical diagnosis from typically developing children. The second index, the Social Interaction Deviance

Composite, is an index of mismatch between structural and pragmatic/social skills. This was derived to give optimal discrimination between children with typical SLI and those with evidence of pragmatic difficulties. A low SIDC is seen when a child has intact structural language skills but major pragmatic difficulties. This kind of profile was characteristic of children with a diagnosis of Asperger syndrome.

CCC-2, US version

A US version of CCC-2 was subsequently standardized and was published in 2006. Changes to the checklist itself were minor and just involved alteration of wording to make it more suitable for the US context. The scoring, however, was altered so that the General Communication Composite was scaled with a mean of 100 and SD of 15. It is not therefore comparable to the UK version, which is based on the sum of eight subscales, with expected mean of 80. In addition, some changes were made to the names of scales. The SIDC was renamed the Social Interaction Difference Score (SIDI).

Psychometric Data

The UK version of CCC-2 was standardized on a sample of 542 children aged 4–16 years, which was broadly representative of the socioeconomic distribution of the general population and covered a wide geographic range (though not all regions were represented). Before deriving norms, responses were inspected to find cases where the pattern of responses suggested poor comprehension of instructions; rules were specified to identify these, and they were excluded. Floor effects were obtained on all scales, especially at the older ages (i.e., many children had no evidence of impairment). Norms were derived for each scale from a regression equation that predicted total log score from log age in months. These scores were scaled to mean of 10 and SD of 3. Because of the non-normality of the data, the scaled scores have a ceiling, which means that CCC-2 is not well suited for assessing variations among children who have above-average communication skills.

The test manual reports internal consistency and inter-rater agreement for all scales. Coefficient alpha (internal consistency) was 0.65 or more for all scales. Inter-rater reliability between a parent and a professional (teacher or speech-language therapist) was not impressive for individual subscales. The inter-rater reliability for the General Communication Composite (GCC) was 0.396, and for the Social Interaction Deviance Composite, it was 0.790. Disagreement between parent and professional ratings generally took the form of professionals rating a lower level of impairment.

Validity was assessed using a sample of children with diagnoses of specific language impairment (SLI), pragmatic language impairment (PLI), and high-functioning autism (HFA) or Asperger syndrome as well as twenty typically developing (TD) children. There were striking differences between the clinical groups and the TD group on all ten subscales. On the GCC, there was little overlap between the distribution of scores of the clinical groups and the TD group. However, the GCC did not differentiate well between the different types of disorder. Rather, it acted as a general indicator that the child had communication difficulties. The groups were better differentiated by the Social Interaction Deviance Composite (SIDC), which was formed by subtracting scores on pragmatic/social scales from those on the structural language scales. This showed a progressive increase in abnormality going from the SLI group through the PLI and HFA groups, with the Asperger syndrome group obtaining the lowest scores. Nevertheless, there were no sharp boundaries between the groups, but rather a gradual progression.

Australian Sample

Normative data were also collected for 115 Australian schoolchildren aged 6, 9, or 12 years from the Perth Metropolitan Region. In general, scale means for these children fell around one point below the expected mean of 10. It was recommended therefore that different cutoffs should be used for Australian children.

CCC-2, US Edition

US norms were gathered for the CCC-2 US edition on a sample of 950 children aged 4–16 years. This sample was well matched to US population demographics in terms of race/ethnicity, geographic region, and parental educational level. Norms were developed by a process of inferential norming.

Test-retest reliability was obtained by having a subset of respondents complete the CCC-2 on two occasions within a period of 1–28 days. Values were generally high, above .85, for different age ranges. Internal consistency was somewhat higher than that found for the UK sample. Inter-rater reliability was not assessed.

Validity was assessed by considering scores from children from clinical samples including those with SLI, pragmatic language impairment, and autism spectrum disorder (ASD). The criteria for diagnosing pragmatic language impairment are not provided. As with the UK sample, all three clinical groups showed impairments on all ten subscales. SIDI scores of -11 or less were seen in 6% of children with SLI, none of those with PLI, and 27% of those with ASD.

Data on sensitivity and specificity are presented for different cutoffs on the GCC. In general, as with the UK version, the GCC has good sensitivity and specificity for distinguishing clinical cases from typically developing children, but it is not useful for distinguishing between clinical groups.

Clinical Uses

CCC-2 is a useful screening instrument for communication disorders. The General Communication Composite (GCC) is useful for identifying that a child has communication difficulties and few affected children obtain a GCC above the 10th percentile. The GCC is not, however, useful for distinguishing between different subtypes of disorder.

The SIDC is useful for identifying children who have an uneven communicative profile, with disproportionate impairment in pragmatic aspects of communication relative to structural

language skills. This composite has good reliability and is sensitive to autistic spectrum disorders. It is recommended, however, that it should only be interpreted for a child whose GCC is below the 10th percentile.

The CCC-2 is *not* a diagnostic instrument for autistic spectrum disorder (ASD). It can however be useful in screening for ASD. It is recommended that children who obtain low scores on the GCC, including poor performance on the pragmatic scales, should be referred for full assessment for ASD.

The profile of scores on different subscales is too unreliable to be used diagnostically but can nevertheless provide a useful starting point for a discussion with a caregiver about a child's difficulties.

In research contexts, CCC-2 can be useful for quantifying the extent of communication impairment in different domains. Deficits measured by the CCC-2 have been shown to be highly heritable. The CCC-2 has been shown to be sensitive to the broader autism phenotype in siblings of children with ASD.

CCC-2 has also been used with children with genetic conditions such as Williams syndrome, Down syndrome, and sex chromosome trisomies, where it can be helpful in highlighting different communicative deficits.

See Also

- [Communication Assessment](#)
- [Pragmatic Language Impairment](#)
- [Social Responsiveness Scale](#)

References and Reading

- Bishop, D. V. M. (1998). Development of the Children's Communication Checklist (CCC): A method for assessing qualitative aspects of communicative impairment in children. *Journal of Child Psychology and Psychiatry*, 39, 879–891.
- Bishop, D. V. M. (2003). *The Children's Communication Checklist, version 2 (CCC-2)*. London: Pearson.
- Bishop, D. V. M. (2006). *The Children's Communication Checklist, version 2 (CCC-2) US Edition*. New Jersey: Pearson.
- Bishop, D. V. M., & Baird, G. (2001). Parent and teacher report of pragmatic aspects of communication: Use of the Children's Communication Checklist in a clinical setting. *Developmental Medicine and Child Neurology*, 43, 809–818.
- Bishop, D. V. M., & McDonald, D. (2009). Identifying language impairment in children: Combining language test scores with parental report. *International Journal of Language & Communication Disorders*, 44, 600–615.
- Bishop, D. V. M., Laws, G., Adams, C., & Norbury, C. F. (2006a). High heritability of speech and language impairments in 6-year-old twins demonstrated using parent and teacher report. *Behavior Genetics*, 36, 173–184.
- Bishop, D. V. M., Maybery, M., Wong, D., Maley, A., & Hallmayer, J. (2006b). Characteristics of the broader phenotype in autism: A study of siblings using the Children's Communication Checklist – 2. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 141B, 117–122.
- Bishop, D., Whitehouse, A., & Sharp, M. (2009). *Communication checklist – Self-report (CC-SR)*. London: Pearson Assessment.
- Bishop, D. V. M., Jacobs, P. A., Lachlan, K., Wellesley, D., Barnicoat, A., Boyd, P. A., et al. (2010). autism, language and communication in children with sex chromosome trisomies. *Archives of Disease in Childhood*, 96, 954–959.
- Broeders, M., Geurts, H., & Jennekens-Schinkel, A. (2010). Pragmatic communication deficits in children with epilepsy. *International Journal of Language & Communication Disorders*, 45(5), 608–616.
- Ferguson, M. A., Hall, R. L., Riley, A., & Moore, D. R. (2011). Communication, listening, cognitive and speech perception skills in children with auditory processing disorder (APD) or specific language impairment (SLI). *Journal of Speech, Language, and Hearing Research*, 54(1), 211–227.
- Geurts, H. M., Verté, S., Oosterlaan, J., Roeyers, H., Hartman, C. A., Mulder, E. J., et al. (2004). Can the Children's Communication Checklist differentiate between children with autism, children with ADHD, and normal controls. *Journal of Child Psychology and Psychiatry*, 45, 1437–1453.
- Laws, G., & Bishop, D. V. M. (2004). Pragmatic language impairment and social deficits in Williams syndrome: A comparison with Down's syndrome and specific language impairment. *International Journal of Language & Communication Disorders*, 39, 45–64.
- Norbury, C. F., Nash, M., Bishop, D. V. M., & Baird, G. (2004). Using parental checklists to identify diagnostic groups in children with communication impairment: A validation of the Children's Communication Checklist – 2. *International Journal of Language & Communication Disorders*, 39, 345–364.
- Philofsky, A., Fidler, D. J., & Hepburn, S. (2007). Pragmatic language profiles of school-age children with autism spectrum disorders and Williams syndrome.

- American Journal of Speech-Language Pathology*, 16(4), 368–380.
- Verte, S., Geurts, H. M., Roeyers, H., Rosseel, Y., Oosterlaan, J., & Sergeant, J. A. (2006). Can the Children's Communication Checklist differentiate autism spectrum subtypes? *Autism*, 10(3), 266–287.
- Volden, J., & Phillips, L. (2010). Measuring pragmatic language in speakers with Autism spectrum disorder: Comparing the Children's Communication Checklist-2 and the Test of Pragmatic Language. *American Journal of Speech-Language Pathology*, 19, 204–212.
- Whitehouse, A. J. O., & Bishop, D. V. M. (2009). *Communication Checklist for Adults (CC-A)*. London: Pearson.
- Whitehouse, A. J. O., Coon, H., Miller, J., Salisbury, B., & Bishop, D. V. M. (2010). Narrowing the broader Autism phenotype: A study using the Communication Checklist – Adult version (CC-A). *Autism*, 14(6), 559–574.

Children's Communication Checklist, Version 2

► Children's Communication Checklist (CCC-2)

Children's Global Assessment Scale

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Synonyms

Developmental Disabilities – Children's Global
Assessment Scale (DD-CGAS)

Description

The Children's Global Assessment Scale (CGAS) is a clinician-rated instrument that provides a single score for the overall level of behavioral and emotional functioning of a child aged 4–16 years. The CGAS is completed by a clinician based on information acquired from direct examination and/or derived from informants such as parents, educators, or case managers. Raters score the

child's most impaired level of functioning for the period of interest (usually the past month) on a scale ranging on a continuum from 100 (corresponding to excellent functioning in all areas of life) to 1 (representing very poor functioning with need for constant supervision). Anchoring descriptors are provided for each decile of the CGAS. While a score of 100–91 indicates *superior functioning* and 90–81 *good functioning*, 80–71 applies to children with *no more than slight impairment in functioning at home, at school, or with peers*. A score of 70 or below is usually considered the threshold for the presence of definite, although slight, functional impairment. Most children referred for clinical evaluation and treatment have scores of 60 or below.

The CGAS has been further modified to meet the need of scoring global functioning of children with autism age 4 and older. This scale is called the Developmental Disabilities – CGAS (or DD-CGAS). The information used for scoring the DD-CGAS relates to four main domain of functioning: self-care, communication, social behavior, and school/academic performance. In each of these domains, the level of impairment can range from none to extreme. The reference for determining the level of impairment is the level of functioning that would be expected by a typically developing child of the same chronological age. Impairment in the main domains of functioning is then used by the rating clinician to formulate a final overall score of functioning (the DD-CGAS score) on a scale ranging from 100 (corresponding to superior functioning) to 1 (indicating extreme impairment). Also the DD-CGAS provides descriptors for each decile (i.e., 100–91: superior functioning within family, school, and peers; 90–81: adequate functioning in all areas; 80–71: most daily living activities at age level but with slight impairment in at least one; 70–61: most daily living activities at age level but with moderate impairment in at least one domain; 60–51: moderate impairment in functioning in most domains; 50–41: moderate impairment in functioning in most domains and severe impairment in at least one domain; 40–31: severe impairment in functioning in some domains; 30–21: severe impairment in all domains and settings; 20–11: extreme

impairment in at least one domain; 10–1: extreme and pervasive impairment with danger to self or others and need for intensive constant supervision). The time frame for the rating can vary but typically is in the order of several weeks or months.

Historical Background

The CGAS was introduced by Shaffer et al. (1983) and is a modification of the Global Assessment Scale developed by Endicott and colleagues in 1976, which, in turn, was a revision of the Health-Sickness Rating Scale, originally published by Luborsky in 1962. A similar scale is the Global Assessment of Functioning (GAF), which constitutes the axis V of the DSM-IV multi-axial evaluation. The DD-CGAS is a modification by Wagner et al. (2007) of the CGAS specifically to score the global level of functioning of children autism and other pervasive developmental disorders. Both the CGAS and DD-CGAS have been translated in languages other than English and are used internationally.

Psychometric Data

When used by raters trained in the clinical evaluation of children with mental illness, the CGAS was shown to have excellent inter-rater reliability (e.g., intraclass correlation coefficient around 0.84), good test-retest stability, and acceptable discriminant and concurrent validity. The CGAS can detect treatment effects. For example, it was able to discriminate between active antidepressant treatment and placebo in adolescent depression. The DD-CGAS too was found to have very good inter-rater and test-retest reliability when used by clinicians who were experts in autism and other pervasive developmental disorders and who had been trained in its use. DD-CGAS scores showed moderate correlation with indices of adaptive behavior, intellectual functioning, and severity of psychopathology. Preliminary data obtained before and after 6 months of treatment indicate a moderate correlation between changes in the DD-CGAS scores and changes on the Aberrant

Behavior Checklist and the Clinical Global Impressions-Improvement scores.

Clinical Uses

The CGAS is a clinically useful instrument that provides an overall score of the level of functioning of a child. The DD-CGAS is specifically useful for rating functioning in the context of autism or other pervasive developmental disorder and is a relatively simple way of indicating the observed global functioning relative to the expected functioning based on normal development. The DD-CGAS allows direct comparisons to be made between functioning of children with autism and functioning of children with other mental disorders such as schizophrenia, depression, or anxiety.

See Also

► [Functional Analysis](#)

References and Reading

- Shaffer, D., Gould, M. S., Brasic, J., Ambrosini, P., Fisher, P., Bird, H., et al. (1983). A children's global assessment scale (CGAS). *Archives of General Psychiatry*, 40, 1228–1231.
- Wagner, A., Lecavalier, L., Arnold, L. E., Aman, M. G., Scahill, L., Stigler, K. A., et al. (2007). Developmental disabilities modification of the Children's Global Assessment Scale. *Biological Psychiatry*, 61, 504–511.

Children's Psychiatric Rating Scale

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Synonyms

[CPRS](#)

Abbreviations

ECDEU
Early clinical drug evaluation unit
EMA
European Medicines Agency

Description

The Children’s Psychiatric Rating Scale (CPRS) is a multidimensional rating scale of childhood psychopathology.

The CPRS is not diagnostic but rather a broad-ranging rating scale of symptoms and behaviors which may contribute to diagnosis. In addition, the scoring system enables the rating of *severity* of symptoms and presentation. Since the scale measures the presence or absence of symptoms over a particular period of time, it has been a useful instrument of treatment efficacy and has regularly been the instrument of choice employed in clinical trials.

Owing to the established subscale structure, an abbreviated form, comprised of 14 questions relevant to the autism spectrum, has been employed in studies evaluating treatment efficacy in autistic children. However, more recently the European Medicines Agency (EMA 2017), *Guideline on the clinical development of medicinal products for the treatment of Autism Spectrum Disorder (ASD)*, has suggested the use of the Childhood Autism Rating Scale (CARS) for baseline assessment and outcome measures in clinical trials.

It has also demonstrated value in evaluating psychopathology in autism, clarifying major behavioral dimensions and identifying distinct subtypes (Overall and Campbell 1988; Overall and Pfefferbaum 1982; Pfefferbaum and Overall 1983).

The CPRS is a clinician-rated scale, based on (1) behaviors observed during clinical interview and (2) the child’s reporting of symptoms. The autism-specific scale is based on observation only.

In the *Diagnostic and Statistical Manual*, third edition (DSM-III, American Psychiatric Association [APA] 1980), a diagnosis of infantile autism is made when four behavioral characteristics are

Children’s Psychiatric Rating Scale, Table 1 Items with respective numbers on CPRS (Overall and Campbell 1988)

Withdrawal (8)	Loud voice (25)
Rhythmic motions (28)	Negative and uncooperative (10)
Abnormal object relationships (7)	Hypoactivity (5)
Unspontaneous relationship to examiner (16)	Fidgetiness (3)
Underproductive speech (2)	Hyperactivity (4)
Angry affect (11)	Other speech deviance (27)
Lability of affect (20)	Low voice (24)

present: (1) *pervasive lack of responsiveness to other people*, (2) *gross deficits in language development*, and (3) *bizarre responses to the environment*. These behaviors can be rated, whereas characteristic (4) *speech deviance*, such as echolalia and pronominal reversal, may be more difficult to evaluate in individuals with little speech. These behaviors are deemed well represented by the 14 items on the autism scale of the CPRS (Table 1).

Historical Background

The CPRS was originally developed by the Psychopharmacology Research Branch of the NIMH as a general-purpose instrument (1976). It featured in the ECDEU Assessment Manual for Psychopharmacology Revised (Guy 1976) among other pediatric scales integral to clinical drug evaluation programs. At this stage, the CPRS was regarded as *experimental*, and no standardization data were available.

The instrument was designed to be employed within a semi-structured interview format to be completed by clinicians and generally used alongside parent- and teacher-completed measures. The rating system facilitated assessment at various stages of a clinical trial, generally prior to the commencement of treatment, during the middle and at the end of treatment. It was designed for use with children up to the age of 15 years.

The first 28 items were rated on direct observation of behaviors at interview, while the latter 34 were rated on the basis of the child's verbal reporting of symptom presence at the time of the interview or during the preceding 7 days. Ratings on a Likert scale were possible from *not answered*, *not present*, *very mild*, *mild*, *moderate*, *moderately severe*, and *severe to extremely severe*. The seven-point scale was effectively derived from the Adult Brief Psychiatric Rating Scale (Overall and Gorham 1962).

The scoring was further developed by Fish (1985). The rating scale comprised of two sections (the original 63 items): in Section A, the clinician rated both the observed behavior at interview and the child's reporting of symptoms or behaviors. Section B represented the clinician's overall view based on the integration of a range of data available, including maternal reports and school records. Hence, additional areas were rated by clinicians with respect to clusters of behavior such as withdrawal, aggressive behavior, hyperactive behavior, inadequate or immature behavior, and organic impairment.

Ratings were made on the degree of abnormality from 0 to 9: *none*, *present but not significant*, *significant but mild*, *moderate*, *moderately severe*, *severe*, *very severe and may be paralyzing*, *item not relevant to child*, and *not known or not ascertained*.

The measure has been valuable owing to the breadth of the range of symptoms and behavioral manifestations assessed while not being limited to the DSM diagnostic criteria, since the scale was originally designed prior to the publication of the DSM-III.

Overall and Campbell (1988) proposed an abbreviated version of the CPRS to evaluate psychopathology in autistic children. They evaluated a subtest of the CPRS, comprising of 14 questions relevant to the diagnosis of autism. Fourteen of the 28 questions of the CPRS are included. Since these are based on *observed* behaviors and symptoms, the subtest is useful for those autistic children who have little or no communicative language and who are severely disturbed or severely developmentally delayed.

Psychometric Data

No normative data existed for the CPRS in its original form (Guy 1976).

Factor-analytic studies have subsequently supported a 6-syndrome subscale structure, hence establishing the internal validity of the CPRS (Overall and Pfefferbaum 1982; Pfefferbaum and Overall 1983).

Evaluation of the diagnostic factor structure of the CPRS (Overall and Pfefferbaum 1982; Pfefferbaum and Overall 1983) confirmed the scale's usefulness in evaluating psychopathology and measuring treatment response in different clinical groups. Seven core factors were identified, namely, *behavioral problems*, *depression*, *thought disturbance*, *psychomotor excitation*, *psychomotor retardation*, *nervous/tension*, and *organicity*. Furthermore, cluster analysis revealed six distinct clusters of symptoms and features, thus enabling the grouping together of those DSM-III diagnoses which tend to have core features and symptom profiles in common. Treatment evaluation could thus be focused on the particular dimensions of symptom presentation.

Studies have served to demonstrate both predictive and construct validity in testing diagnostic classifications. However, like other autism rating scales, the CPRS was developed prior to the revision of autism diagnostic classification (APA 2013; Thabtah and Peebles 2019). To date no data are available regarding how well the autism subscale maps onto the two DSM-5 domains of ASD.

Clinical Uses

The CPRS is a general-purpose instrument for assessment of a broad range of childhood psychopathology.

While the measure is used in its complete form, i.e., a 63-item rating scale, autism-specific research has focused on a subset of 14 items relevant to the condition. The first 28 items on the CPRS are deemed valuable since they are items which are rated on the basis of clinical observation of behavior at interview. Hence, they do not rely on a particular level of language development.

Fourteen of these 28 items have been deemed relevant for the assessment and classification of symptoms and features observed in autistic children (Overall and Campbell 1988). The behaviors included in this subset are well matched with the behavioral criteria for infantile autism first described in the DSM-III (APA 1980), including deficits in language development, odd responses to the environment, and lack of responsiveness to other people.

Overall and Campbell (1988) conducted factor analysis of the subset of the CPRS and noted four core aspects which differentiated autistic children, namely, autism, anger/uncooperativeness, hyperactivity, and speech deviance. In other words, scores on the scale differentiated subgroups. However, these did not necessarily differentiate autistic children from children with other psychiatric conditions.

The 14-item CPRS continued to be employed as the measure of choice in clinical trials (Desousa 2010). It is generally completed by the clinician (s) following videotaped observations of autistic children. This is in conjunction with parental ratings of behavior and symptoms, e.g., Aberrant Behavior Checklist (ABC) and the Conners' Parent Rating Scale – Revised (CPRS-R), as well as other clinician ratings such as the Clinical Global Impression Scale (CGI). The rating scale has demonstrated value in open-label and controlled psychopharmacological trials. Improvement of 25% or more on identified symptoms compared with baseline ratings suggests child is a responder to the medication.

Studies have evaluated tolerability, long-term effects, and efficacy of specific psychotropic medication in autistic disorder as well as comparisons of different medication within this group. Studies of specific psychiatric features associated with autism spectrum disorders and effects of psychopharmacology have employed the CPRS-14 (Gagliano et al. 2004; Desousa 2010). More recent clinical trials (Lemonnier et al. 2017) have followed the EMA guidelines (2017) for evaluating efficacy and impact of treatment, employing measures such as the Childhood Autism Rating Scale (CARS), Social Responsiveness Scale (SRS), and the Clinical Global Impressions (CGI).

See Also

- ▶ [Childhood Autism Rating Scale](#)
- ▶ [DSM-5](#)
- ▶ [DSM-III](#)
- ▶ [Risperidone](#)
- ▶ [Screening Measures](#)
- ▶ [Social Responsiveness Scale](#)
- ▶ [Treatment Integrity](#)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text rev.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Campbell, M., & Palij, M. (1985). Documentation of demographic data and family history of psychiatric illness. *Psychopharmacology Bulletin*, 21(4), 719–721.
- Desousa, A. (2010). An open-label trial of risperidone and fluoxetine in children with autistic disorder. *Indian Journal of Psychological Medicine*, 32(1), 17–21. <https://doi.org/10.4103/0253-7176.70522>.
- European Medicines Agency (2017). Guideline on the clinical development of medicinal products for the treatment of Autism Spectrum Disorder (ASD) (EMA/CHMP/598082/2013). Retrieved from https://www.ema.europa.eu/en/documents/scientific-guideline/guideline-clinical-development-medicinal-products-treatment-autism-spectrum-disorder-asd_en.pdf
- Fish, B. (1985). Children's psychiatric rating scale. *Psychopharmacology Bulletin*, 2(4), 753–770.
- Gagliano, A., Germano, E., Pustorino, G., Impallomeni, D., Calamoneri, F., & Spina, E. (2004). Risperidone treatment of children with autistic disorder: Effectiveness, tolerability, and pharmacokinetic implications. *Journal of Child and Adolescent Psychopharmacology*, 14(1), 39–47.
- Guy, W. (1976). *ECDEU assessment manual for psychopharmacology, revised, 1976*. Rockville: United States Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration. (DHEW Publication No. (ADM) 76-338).
- Guy, W. (2000). *Clinical Global Impressions (CGI) scale*. Modified From: Rush, J., et al., *Psychiatric measures*. Washington, DC: APA.
- Lemonnier, E., Villeneuve, N., Sonie, S., Serret, S., Rosier, A., Roue, M., Brosset, P., Viellard, M., Bernoux, D., Rondeau, S., Thummler, S., Ravel, D., & Ben-Ari, Y. (2017). Effects of bumetanide on neurobehavioral function in children and adolescents with autism

- spectrum disorders. *Translational Psychiatry*, 7, e1056. <https://doi.org/10.1038/tp.2017.10>.
- Malone, R. P. (2007). Ziprasidone in adolescents with autism: An open-label pilot study. *Journal of Child and Adolescent Psychopharmacology*, 17(6), 779.
- Niederhofer, H. W., & Mair, S. A. (2003). Tianeptine: A novel strategy of psychopharmacological treatment of children with autistic disorder. *Human Psychopharmacology: Clinical and Experimental*, 18(5), 389–393.
- Overall, J. E., & Campbell, M. (1988). Behavioral assessment of psychopathology in children: Infantile autism. *Journal of Child Psychology*, 44, 708–716.
- Overall, J. E., & Gorham, D. R. (1962). The brief psychiatric scale. *Psychological Reports*, 10, 799–812.
- Overall, J. E., & Pfefferbaum, B. (1982). Brief Psychiatric Rating Scale for Children. *Psychopharmacology Bulletin*, 18, 10–16.
- Overall, J. E., & Pfefferbaum, B. (1984). A brief scale for rating psychopathology in children. *Innovations in Clinical Practice: A Source Book*, 3, 257–266.
- Pfefferbaum, B., & Overall, J. E. (1983). Diagnostic factor structure of the children's psychiatric rating scale (C.P.R.S.). *Journal of Clinical Child Psychology*, 12, 167–173. <https://doi.org/10.1080/15374418309533126>.
- Thabtah, F., & Peebles, D. (2019). Early autism screening: A ss. *International Journal of Environmental Research and Public Health*, 16, 3502.

Children's Social Behavior Questionnaire

► [CSBQ \(Children's Social Behavior Questionnaire\)](#)

Children's Unit for Treatment and Evaluation (State University of New York at Binghamton)

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Definition

The Institute for Child Development (ICD) at the State University of New York at Binghamton

promotes the welfare of children who are challenged by developmental, learning, and emotional disorders. The Institute serves as the focus for service, research, undergraduate, and graduate training programs and the dissemination of basic and applied research. The Institute supports specific units that provide treatment and educational services for children within an evidence-based model. The *Children's Unit for Treatment and Evaluation* provides services for children with autism spectrum disorders and their families in the context of Early Intervention, Preschool, and School Age programs as well as additional complementary programs, such as its Diagnostic Evaluation Clinic.

Historical Background

The ICD was founded by Dr. Raymond G. Romanczyk, a faculty member and clinical psychologist, in 1974, located on the State University of New York (SUNY) at Binghamton campus. An ICD program, the Children's Unit for Treatment and Evaluation, was established in 1975 in cooperation with a small group of parents who wished to receive evidence based and intensive services for their children. Given the efficacy of the program, parents worked with local and state legislators to provide the Unit an appropriate connection to the region's continuum of services. Special status was granted in 1977 through an act of the New York State Legislature (Senate Bill 5911-A) which allows the Unit to exist with a dual status as a fully certified New York State Education Department private school and at the same time organizationally part of SUNY at Binghamton. The bill permits school districts, counties, and other state agencies to contract directly with the Unit for services. This also allows the Unit to function as a separate entity at the University level, rather than as the more typical "lab school" or time-limited grant-funded project. The Unit was the first in New York to provide full-day intensive evidence-based services for children in the early intervention and preschool age range.

At its start, the Unit served just six children from the immediate area. The catchment area has

grown quite large and now includes the New York State counties of Broome, Tioga, Cortland, Tompkins, Chenango, and Onondaga and the Pennsylvania counties of Bradford, Susquehanna, and Sullivan, representing locations across urban, suburban, and rural areas. Currently approximately 65 children commute daily to the program from within an approximately 90 mile radius.

The ICD has had multiple locations on the campus since 1973. In 2001 the Institute was moved to a spacious specially constructed building for the sole use of the ICD. It is located next to the campus preschool services building, permitting ease of cooperative programs for peer-based activities. In 2013 Dr. Jennifer M. Gillis, faculty member, clinical psychologist, and licensed behavior analyst, became the co-director.

Rationale or Underlying Theory

An autism spectrum disorder affects not only the individual but also the family, the community, and the broader society as well. As a group, the impact on families is greater and more complex than many other disorders. This requires an intensity, quality, and precision of educational and clinical services that are not only directed at the individual with an autism spectrum disorder but also the family. Comprehensive service delivery cannot be impeded by bias, inappropriate, and antiquated organizational structures, low expectation, or by compartmentalization of services.

The guiding principle of the Institute for Child Development is that providing a caring, warm, supportive, enriched environment that respects the dignity of individuals and celebrates their unique qualities and potential is the *minimum* starting point for educational and clinical services. This principle is paired with a comprehensive commitment to evidence-based services drawing upon well-conducted, methodologically sound, empirical research. Thus, educational and clinical research is utilized on a continuing basis, and the ICD provides mechanisms and opportunities for all program staff to acquire and use research information on a timely basis, which includes weekly in-service training, journal

club, visiting speakers, and consultants, as well as attendance at professional conferences. Another priority is that there must be extensive, precise, quantitative, and frequent child assessment that permits the daily implementation of an objective feedback loop for decision-making regarding appropriate goals, procedures, and progress.

Given the emphasis on evidence-based approaches to intervention, current practice is based upon research in behavioral approaches (applied behavior analysis and cognitive behavioral therapy), developmental models (Early Start Denver Model), nomothetic and ideographic assessment (such as functional behavior assessment), family systems, curriculum selection, basic attention and learning processes, social development, and comorbid disorders. The program employs a comprehensive model, but it is not based on a specific single "model" or particular "approach," but rather is dynamically based on contemporary, methodologically sound, peer-reviewed research that has been replicated.

Goals and Objectives

The program provides full-day, 12-month services with emphasis on individual evaluation of each child's assets and deficits, past history of services and response, current functioning, the specific parameters of the child's learning pattern, and an analysis of maintaining factors of current behavior patterns using functional behavior analysis. The goal of the program is to remediate skill deficits that prevent children from participating at their potential in the continuum of education services in their community and to provide families with training and support for their own needs. Emphasis is placed on acquisition of communication skills, social interaction skills, self-regulation skills, and reduction of stereotyped behavior and restricted interests. The average length of enrollment is 2.5 years as the emphasis is upon rapid reintegration into services in the child's local community. Thus the ICD is not a long-term alternate educational placement but rather an intensive, focused, short-term intervention program.

Prioritized goals within a comprehensive program produce rapid transition to services in the child's community.

- Ongoing progress monitoring (measurement for decision-making feedback loop)
- Family/caregiver involvement (to address both child and self needs)

Treatment Participants

Children between the age of 1 and 11 are eligible to attend the program. Referrals come from physicians, county health departments, and school districts via the NY system of Early Intervention Officials, the Committee on Preschool Special Education, and the Committee on Special Education. Because the catchment area is so expansive, admission criteria is based on a relative analysis of the child and family's needs in the context of the resources of the continuum of services in the community of residence. The majority of children are diagnosed with autism spectrum disorder and have a history of poor response to intervention prior to admission. Parent willingness and ability to participate in the child's program and attend family service groups are highly variable upon admission and are not a selection criteria. It is explicit that admission is not based on parent's willingness to participate in ongoing research projects, as such a requirement would be deemed coercive.

Treatment Procedures

Comprehensive intervention requires that at minimum primary focus is placed on the core ASD areas of social communication and social interaction and restricted, repetitive, and stereotyped patterns of behavior, interests, and activities. Further, salient comorbid disorders must be assessed and addressed, e.g., anxiety disorders.

Next, a continuum model for comprehensive intervention is used (Romanczyk and Gillis 2010) that includes several critical procedural components:

- Assessment (nomothetic and ideographic)
- Curriculum planning (goal selection, prioritization, and sequencing)
- Intervention methodology (evidence based)

It is these components that are used to achieve acquisition of skills and address behavior problems with emphasis on generalization of skill repertoires in normative settings. A comprehensive developmentally organized curriculum, the IGS Curriculum (Romanczyk et al. 2000), is used to guide and structure assessments and precise goal specification. Utilization of a curriculum that provides a developmentally sequenced compendium of goals permits identification of skills associated with the child's strengths and weaknesses, guides further assessment of the limits of the child's skills and performance, and permits meaningful discussion of goal selection and priorities with parents as it helps in supplementing their knowledge of child development. A comprehensive curriculum that is developmentally sequenced such as the IGS ensures that assessment is directly linked to functional instructional goals. Appropriate staff are also certified Early Start Denver Model (ESDM) therapists and also trained in the use of the Verbal Behavior Milestones Assessment and Placement Program (VB-MAPP).

Settings for instruction, instructional procedures, and specific goals present a complex mix of variables. Their interaction must be addressed to allow optimal configuration from the perspective of each factor. As an example, if emphasis is currently upon transition between two instructional settings, then goals and procedures need to be adjusted to be appropriate within that context.

Likewise, if focus is upon acquisition of a specific goal set, then settings and procedures are adjusted to maximize speed and strength of learning. This is a dynamic process, allowing adjustment to changing child and family characteristics, as well as resource factors, while developing more and more sophisticated child repertoires. No one variable has primacy, with emphasis placed on a coherent comprehensive program.

Social development is a priority, and the program is designed to improve social skills and

social problem-solving. Activities are constructed for individual strengths and deficits. Activities and projects focus on skill strengthening via use of modeling, rehearsal, role-playing, cognitive behavior therapy, and anxiety reduction approaches.

A second component is the strong cooperation with the University Campus Preschool which is in a building physically adjacent to the Institute. Children from the Institute attend the preschool on a part-time basis as a transition step as part of preparation for returning to program's in their school district of residence. Staff of the Institute accompany the children and serve as guides for the preschool staff to integrate the children into typical activities.

A third component is the "Buddy Group," an after school therapeutic social skills program. The program focus is on increasing the quality of social interactions through participating in a variety of on-site and community activities with typical peers. Peer volunteers are area middle-school children who function as "buddies" by relating to children with ASD as they would any other friend. This provides realistic feedback and experiences as would be encountered in typical casual social settings and allows for more success in meeting complex social expectations. Teaching objectives focus on age appropriate activities, learning social expectations, and responding appropriately to the inherently variable consequences of social interaction.

A fourth component is an extension that we term "SLC Saturday." The Institute has a very large specially constructed playground with a state-of-the-art injury prevention focus and various types of play areas that permit quiet self-play as well as areas that support and encourage social interactive play. It can accommodate more than 150 individuals comfortably and safely. It is used on a daily basis for children enrolled at the Institute and is the focus for social development. SLC is the acronym for Social Learning Center. This facility is opened on Saturdays for families of the Institute as well as the community, permitting a safe and choreographed setting for interaction between children with ASD and typically developing children.

Family Focus

A separate but highly intertwined program emphasizes individualization of services. Family involvement is strongly encouraged and supported. Because a child with an ASD affects the whole family, this greatly influences the family services provided. In addition to instruction in specific procedures and skills so that families can conduct teaching programs at home, measure progress on specific skills, and objectively evaluate their child's performance, the staff individually tailor parent services to the needs and values of each family. Families can choose from a variety of ongoing services that include:

- Didactic instruction to implement intensive language and social/emotional programs at home.
- Homework programs with parent with training so that they can conduct more traditional "homework" pre-academic/academic, leisure, and self-help activities at home.
- Periodically themes are identified for a "family-friendly" personal goal. Activities focus on workshops for parents, individual support meetings, and parents participating in school activities. Staff then increase emphasis on addressing these goals at school and paralleling with home programs to maximize generalization and thus family success.

An important additional program component is parent wellness. Wellness sessions are devoted to assisting caregivers in appropriately addressing individual family member needs with particular emphasis on stress management. Components of the program include recognizing and quantifying stress, changing stress responding through relaxation training, diaphragmatic breathing, progressive muscle relaxation, guided imagery, yoga, time management, and cognitive restructuring approaches.

Technological Innovation

There is significant use of technology, particularly computer technology, with individuals with ASD. However, great caution must be exercised in

applying a technological approach to a problem that is at its core a social interaction disorder.

The ICD has been applying technology to the provision of services since the 1970s and has been acknowledged as a pioneer in this area. Staff are provided with sophisticated organizational systems and technology to address the program priorities. Appropriate utilization, however, requires precise matching of need with solution. A major focus has been to provide staff with useful tools that match their needs and abilities for application in complex and changing circumstances.

From an administrative perspective, the problem of efficiently collecting, organizing, interpreting, and monitoring the voluminous information need to achieve comprehensive program goals represents a continuing challenge. We utilize a series of computer databases to organize each student's educational goal plan, specific habilitative goals, daily and monthly progress on each goal, graphs of progress, history of educational goals, and evaluation of goals. Our curriculum database is connected to above the databases, which allows the selected goals from the IGS to be imported into a student's goal plans' database. From this database, printed reports are generated as well as large screen video projection for staff meetings for review of individual children's goals and progress. The use of extensive computer-based analytic tools for staff, high-efficiency database software for goal selection and monitoring, and extensive use of handheld computing devices with custom-developed software for numerous specialized activities is essential for efficient day-to-day operation within a normative, constrained program budget. The twin goals of the technology program are to improve accuracy and speed of data-based decision-making while simultaneously reducing staff "paperwork" tedium which in turn allows more time to focus on child and family needs.

Efficacy Information

The ICD is one of the ten model programs cited in the Educating Children with Autism report of the

National Research Council (2001) that was commissioned by the US Department of Education. The Committee on Educational Interventions for Children with Autism utilized specific selection criteria in their search for model programs, based upon published reports and frequency of citation. They identified ten programs based upon their criteria, to illustrate "state-of-the-art" model approaches, which included the Children's Unit for Treatment and Evaluation.

Because the ICD is an evidence-based program as described above, there is a large body of research studies that are constantly increasing. Some relevant summaries of this research body include:

National Research Council (2001). *Educating Children with Autism*. Committee on Educational Interventions for Children with Autism. Division of Behavioral and Social Sciences and Education. Washington, DC: National Academy Press.

National Autism Center. (2009). National Standards Project - Addressing the need for evidence-based practice guidelines for autism spectrum disorders, from <http://www.nationalautismcenter.org/about/national.php>

The National Professional Development Center on Autism Spectrum Disorders (2010). <http://autismprdc.fpg.unc.edu/>

Odom, S., Boyd, B., Hall, L., & Hume, K. (2010). Evaluation of comprehensive treatment models for individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40, 425–436.

Romanczyk, R.G., Turner, L.B., Seveler, M. and Gillis, J.M., (2015). The Status of Treatment for Autism Spectrum Disorders: The Weak Relationship of Science to Interventions. In Lilienfeld, Lohr, and Lynn (Eds.), *Science and Pseudoscience in Contemporary Clinical Psychology (2nd Edition)*. NY, NY: Guilford Press.

Romanczyk, R.G., and McEachin (Eds), (2016). *Comprehensive Models of Autism Spectrum Disorder Treatment: Points of Divergence and Convergence*. Springer, ISBN: 978–3–319–40903-0.

New York State Department of Health (2017). *New York State Department of Health Clinical Practice Guideline on Assessment and Intervention Services for Young Children (Age 0–3) with Autism Spectrum Disorders (ASD): 2017 Update*. Support from New York State's Title V Maternal and Child Health Block Grant, the New York State Autism Awareness and Research Fund, and the Far Fund. NYS Department of Health, Albany, NY. Retrieved as: https://www.health.ny.gov/community/infants_children/early_intervention/autism/docs/report_recommendations_update.pdf

Outcome Measurement

For an applied educational/clinical setting, it is not possible to determine which specific factor or combination of factors are the most influential in outcome. That requires controlled research with standardized procedures, specifies duration, and appropriate control groups. The explicit goal of the ICD is to quickly transition children from diverse families and communities to their home school districts and to enable them to participate in the services in their community. The specifics of this transition are unique for each child and do not represent the achievement of an absolute level of functioning. The duration of participation is variable within the average of 2.5 years.

Within these non-research parameters, approximately 50% transition to typical educational settings, 25% to “inclusion opportunity” classrooms, and 25% to “self-contained” classrooms. Importantly, recall that our exit criteria are specific to child, family, and school district goals and do not reflect “absolute” criteria. Thus a given family and school district may have typical placement as their goal, while another family and district have the goal of as quickly as possible having the child participate in their continuum of services (this is often the case for children who must travel substantial distances each day to the program).

The formal research that is conducted at the ICD focuses primarily upon measurement/assessment, process, and focused intervention outcomes. Some recent examples are:

- Aponte, C. & Romanczyk, R.G. (2016). Assessment of Feeding Problems in Children with Autism Spectrum Disorder. *Research in Autism Spectrum Disorders*, 21,61–72.
- Cavalari, R.N.S. & Romanczyk, R.G. (2015). Quantifying Supervisory Decision Making: Eye-Tracking Technology Applications for the Promotion of Child Safety. *Journal of Behavioral Decision Making*. DOI: 10.<https://doi.org/10.1002/bdm.1857>.
- Turner, L.B.& Romanczyk, R.G (2012). Assessment of fears and phobias in children with an autism spectrum disorder. *Research in Autism Spectrum Disorders*, 6, 1203–1210.
- Callahan, E. H., Gillis, J. M., Romanczyk, R. G., & Mattson, R. E. (2011). The behavioral assessment of social interactions in young children: An examination of convergent and incremental validity. *Research in Autism Spectrum Disorders*, 5, 768–774.
- Gillis, J.M., Callahan, E.H. & Romanczyk, R.G. (2010). Assessment of social behavior in children with autism: The development of the Behavioral Assessment of Social Interactions in Young Children. *Research in Autism Spectrum Disorders*.

Qualifications of Treatment Providers

All professional staff hold appropriate licenses and certification for their respective professions. Additionally, 30% of the professional staff are also Board Certified Behavior Analysts. The staff represent the following professions:

- Clinical Psychology
- Special Education
- Behavior Analysis
- Nursing
- Speech Pathology

Occupational Therapy School Psychology Adaptive Physical Education

In addition to professional staff, there are full-time staff in teacher aide, administrative, and technical staff positions.

The ICD also has extensive educational programs. At the undergraduate level, there is an intensive four-course sequence, three of which have practicum components that complement the requirements of the major in psychology. The course sequence has been evaluated by the Behavior Analyst Certification Board as a Verified Course Sequence. Selected graduate students in the doctoral clinical psychology program, in addition to the program requirements, participate for 4 years as staff members at the ICD under the supervision of senior staff. Training is also provided for select postdoctoral fellows as well as medical students.

References and Reading

- Eagle, R., Romanczyk, R. G., & Lenzenweger, M. (2010). Classification of children with Autism Spectrum Disorders: A finite mixture modeling approach to heterogeneity. *Research in Autism Spectrum Disorders*, 4(4), 772–781.
- Romanczyk, R. G., & Gillis, J. M. (2006). Autism & the physiology of stress and anxiety. In G. Baron, G. Groden, J. Groden, & L. Lipsitt (Eds.), *Stress and coping in autism*. New York: Oxford University Press.
- Romanczyk, R. G., & Gillis, J. M. (2008). Practice guidelines for autism education and intervention: Historical perspective and recent developments. In J. Luiselli, D. C. Russo, & W. P. Christian (Eds.), *Effective practices for children with autism: Educational and behavior support interventions that work*. New York: Oxford University Press.
- Romanczyk, R. G., & Gillis, J. M. (2010). Continuum-based model of behavioral treatment for children with autism: A multi-factor and multi-dimensional perspective. In J. A. Mulick & E. A. Mayville (Eds.), *Behavioral foundations of effective autism treatment*. Cornwall-on-Hudson: Sloan Publishing.
- Romanczyk, R. G., Lockshin, S., & Matey, L. (2000). Preschool education programs for children with autism. In S. Harris & J. Handleman (Eds.), *Children with autism: The preschool years* (2nd ed.). Austin: Pro-Ed.

Chile and Autism

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Historical Background

The first initiatives providing help for individuals with Autism Spectrum Disorder (ASD) in Chile were, as it is usually the case, spearheaded by parents of autistic children, whose initiatives founded many organizations that provide services to this day. The largest national support organization, ASPAUT, or the Chilean Association of Parents and Friends of Autistics, was founded in 1983 in Santiago. Today, the nonprofit organization has branches in five of Chile's 15 regions, with 1,400 members nationwide. Its services include four schools, five family support groups, and one vocational training center. Though each location is associated by name, each operates as an independent entity.

Legal Issues, Mandates for Services

The department of special education of the Ministry of Education indicates that in 2009, 589 students diagnosed with autism were receiving educational services under the law *Decreto Supremo N° 815/1990* which guarantees special education services to individuals with Autism, severe Dysphasia, and/ or Psychosis.

Overview of Current Treatments and Centers

Multiple research findings indicate that early identification and diagnosis of ASD can improve

opportunities for children to benefit from interventions and lessen the burden on parents (Zwaigenbaum et al. 2013). The key to early diagnosis is access to competent and effective diagnostic and treatment services. In Chile, health care can be accessed through both the public and private systems.

Public Health Care

Though Chile does provide services through a public health-care system, hospitals are not equipped to attend to individuals with an ASD, even though several laws regarding the disability exist. Parents who do receive medical services through the public health care system have great difficulties in making appointments with specialists such as neurologists or psychiatrists, and if they are successful, encounter very long waiting periods, which, in turn, inhibits the possibility of an early diagnosis and thus early intervention. Even after a diagnosis is made, it is practically impossible to access support and treatment from a multidisciplinary team, on a continuous basis, through the public health system. Furthermore, the government does not guaranty coverage of any treatment related to ASD. In addition to the general public health-care system, there is also a list of 69 illnesses or conditions for which the government guarantees free treatment. ASD is not included in this list (Ministerio de Salud 2010).

Private Health Care

In Chile, there is also a private health-care system. Unfortunately, even after paying high premiums for private health insurance, the coverage for the payment of specialist services is very limited, sometimes as low as one or two sessions a year, or the coverage of a very small percentage of the session's actual cost. Typically, professionals in the private sector work independently, not in an integrated center or fashion, leaving parents with no choice but to shuttle their child to different specialists, who do not communicate with one another, for different treatments. High costs and inadequate service provision through the private health system places great financial, emotional, and practical strains on families. Very few private

centers for developmental disabilities have a multidisciplinary staff that works in an integrated fashion.

Obstacles to Quality Service Provision

Autism spectrum disorders place huge strains on families. These can be quantified in terms of financial investment, time lost from work, and time not spent with other family members. Other strains can only be described, such as levels of stress experienced, impacts on relationships, the mental health status of other family members, and lost personal time of professional careers. In fact, many families suffer from severe dysfunctional relationships leading to parental separation, anxious distress, and psychological problems in siblings.

Financial Burden

One of the most common obstacles to receiving treatment services is that of lack of or restriction of financial means. All but one family interviewed for this study stated that their level of financial resources negatively affected the quantity as well as the quality of the treatment their child received. Only one family, one of substantial financial means, stated that the quantity of treatment that their son received was adequate.

Apart from the financial burden placed on families by both the public and private health-care sectors, lack of professionals trained the area of Autism in Chile creates further obstacles to correct diagnosis and early intervention. All children in this study were seen by various professionals including psychologists, neurologists, and child psychiatrists, and received varying diagnoses. Many of these professionals have varying degrees of training and experience in ASD and also use a variety of classifications, nomenclature, and treatment approaches.

Diagnosis

Of four families interviewed and four further cases reviewed, only one child in this study received a diagnosis of Pervasive Developmental Disorder (PDD) as a first diagnosis. Two children received diagnosis of Dysphasia, three of a general language disorder, one of a nonspecific

behavioral disorder, and one of Schizoid Personality Disorder, respectively, all previous to their diagnosis of ASD. Typically, the first professionals to recommend an evaluation were speech therapists, either within the educational setting or in private practice. At least four children received their diagnosis of an ASD from a neurologist. There is a cultural bias against consulting with psychiatrists, leading to underconsultation with these professionals until later in the process. Deficits in professional development and training in the area of ASD can be seen through significant delay in establishing an early and appropriate diagnosis and poor management in several of these cases.

Cultural Aspects

Obstacles to quality service provision and care can also be found in specific cultural aspects. One unexpected cultural aspect that presented itself in two interviews with center directors interviewed in this study was that of, what could be referred to, as *cultural protectiveness*. One director commented that professionals in Chile are often very guarded about their knowledge of a specific area and do not want to share this or work collaboratively with others for fear of competition in the area. One director also stated that there is a lack of trained professionals in the area of ASD in Chile, notwithstanding, she would not employ any foreign professional, regardless of their training or experience, as “they would not know the reality of Chile.” These opinions were spontaneously expressed without prompting through the interviewer’s questioning. Clinical work, following the psychoanalytic method, tends to be considered “in-doors” and “confidential,” which leads to the lack of communication and poor team work in many instances.

Centralization

Centralization of specialists and services that are available, to Santiago, the capital city of Chile and surrounding areas, poses further obstacles to diagnostic, treatment, and support options for families outside the greater metropolitan Santiago area. The limitation of access to services can be seen in the distribution of autism-specific education

and treatment services throughout the country. Except for Valparaiso and Concepcion, the rest of the country lacks trained professionals.

Service Provision

The following diagram presents the governmental bodies responsible for service provision for individuals with disabilities, accessible in the public sector (Fig. 1).

It is important to note that no governmental body provides direct funding or services for the individuals with ASD. This includes the National Service for Disability.

If organizations would like to receive support through this office, they must apply for funding based on a project proposal. Funding is not guaranteed and is very difficult to obtain.

Educational Services

According to the Ministry of Education, in 2013, there are 15 publicly funded schools throughout the country that offer educational services specifically to children with ASD. These schools are located in seven of Chile’s 15 geographical regions, with five located in the Metropolitan Region of Santiago and four in the neighboring region of Valparaiso. Thirteen of these schools are managed by nonprofit organizations that receive funding from the Ministry of Education, on a monthly basis, based on the number of children that attend. Two of these schools receive funding through their municipalities. There is no school directly run by a government body. There are no privately funded schools that specifically support children with autism.

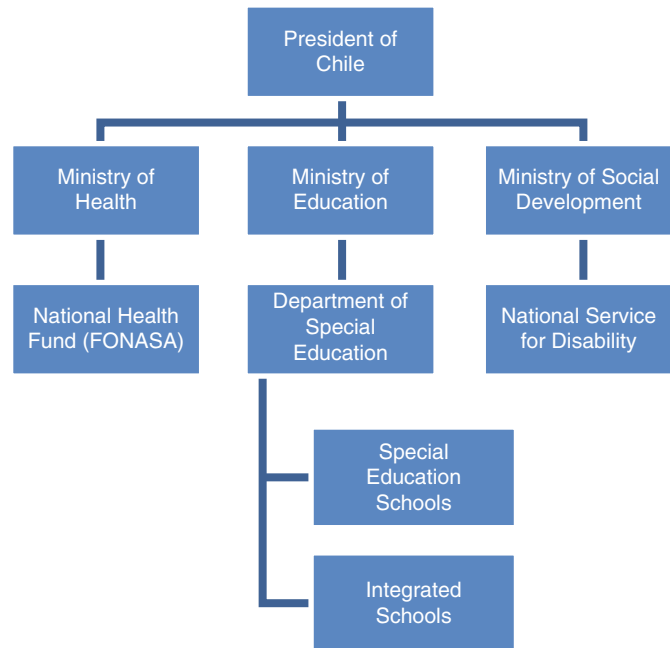
Integration Programs

Chilean law guarantees access to, and integration in, the educational system for all individuals with special education needs. The Ministry of Education satisfies this requirement by giving public schools the opportunity to have an “integration program” by way of contracting a multidisciplinary professional team through government funding.

Public, no profit, or municipality schools receive funding based on what type of disabilities their special needs students have, and how many

Chile and Autism,

Fig. 1 This diagram presents the governmental bodies responsible for service provision for individuals with disabilities in the public sector. Gobierno de Chile (2013)



of them attend. Students with special needs are either considered to have transitory special needs or permanent special needs. Transitory special needs include borderline cognitive disabilities, Attention Deficit Disorders, and specific language and learning disorders. Permanent special needs include cognitive disabilities, physical disabilities, auditory or visual disabilities, and Autism.

According to the Ministry of Education, there are 4,500 public schools in Chile receiving funding for integration programs in 2013. Schools that accept students with special needs are required to redefine their educational projects, adapt their curricula, and implement support systems based on the needs of their students. They must also evaluate and monitor these needs over time as well as train teachers and staff. Students receive a minimum of 10 h of special support per week.

However, in order to receive funding for a multidisciplinary professional team, a school must have at least five transitory special needs students or two permanent special needs students in the first grade level who wish to attend. This begs the question of what kind of support students with special needs in small communities or in

rural areas receive, when pure lack of population limits their access to funding under the governmental initiative.

Some private schools also offer integration programs. The cost of professionals such as educational psychologists is covered by the tuition of all students. However, the parents of a student with special needs must personally cover the costs of any extra support their child might need including special education teachers, classrooms aids, tutors, or “shadows.” Private schools with integration programs often have very strenuous limits on the number of special education students they accept. This may be a limit of five special needs students for a school population of over 1,000 students. Being private institutions, they are under no obligation to accept any student with special needs, if they so choose.

Organizations for ASD

There are currently seven organizations in Chile that provide services specifically to individuals with ASD. These services include schooling options, diagnosis and therapeutic services, and psychoeducation and support for parents and families.

Five of these organizations are located in Santiago, one in Vina del Mar, about 120 km from the capital and one is an internet-based virtual support and advocacy group. Two of these organizations provide support and education through informative websites and chat rooms. One of these specifically supports individuals with Asperger’s Syndrome and their families and only one, ASPAUT, has centers located in various regions of Chile.

Figure 2 below illustrates important aspects of services provided by these organizations including funding, diagnostic tools used, intervention methods utilized, and training of their directors:

Four directors interviewed for this study, three special education teachers and one psychologist, stated that they had not received any education or special training about autism in their University courses. All of them subsequently sought specific education such as training in ABA and PECS either outside of Chile or through visiting international professionals, such as Theo Peeters.

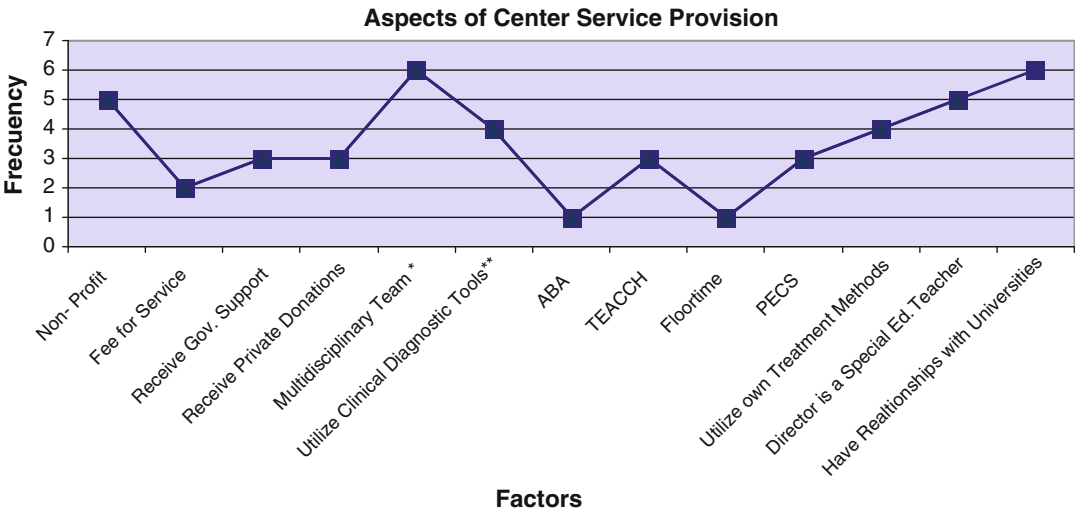
There are also at least two private rehabilitation centers in Santiago that provide diagnostic and therapeutic services to individuals with ASD. Both

provide services through multidisciplinary teams. One center utilizes standardized clinical diagnostic tools while another utilizes its own clinical method. One center provides treatment options based on a mix of ABA, Floortime and TEACCH, while the second provides group therapy, Floortime, and occupational therapy with sensory integration, speech therapy, medication treatment and “Bio- Diets” in connection with two referring pediatricians.

Overview of Research Directions

Children and adolescents under the age of 15 make up 22 % of Chile’s population (WHO 2010).

As stated by the Chilean Ministry of Health, there have been no epidemiological prevalence studies of Autism or ASD conducted in Chile and there is no registry of diagnosed cases. However, based on calculations using the international estimate of prevalence of nine children with autism in 1,000 and 240,569 registered births in Chile in 2007, the Ministry (2011) estimates that



Chile and Autism, Fig. 2 This graph indicates the number centers of service provision in Chile who satisfy each descriptor (*Include Psychologists, Speech- Therapists, Physiotherapists, Special Ed. Teachers, and referring Psychiatrists. **Include ADI-R, M-CHAT, IDEA, Vineland) (Note: *Permissions:* The information presented in the

diagram has been cited. The diagram itself is a creation of the author Sonja Ziegler. The information presented resides in the public domain. The illustration (bar graph) is a visual presentation of data collected during research. It was created by author Sonja Ziegler)

the approximate number of children with a diagnosis of an Autism spectrum disorder would be 2,156. According to the National Inquiry of Disability in 2004, 15,000 individuals presented with severe language disorders, or one in 1,000, using the population registry of 15,000,000 inhabitants of the 2002 census. This estimation does not differentiate between various possible diagnoses.

Though Autism is a global issue, affecting individuals of all ethnicities, clinical and research publications in the field are heavily focused in developed countries, with the United States, Canada, and the United Kingdom being leading publishers. Comparatively, publications in the area of ASD in Latin America are rare, and almost non-existent in Chile (OARC 2012). Thusly, little is known about the magnitude of the problem.

In a review of the Chilean national publication database, only six articles related to ASD have been published in the last 27 years. Three of these articles are clinical descriptions, two are case studies and one is a literature review.

Clinical Description

Though autism is defined as a developmental disorder that manifests itself in the first three years of childhood, its symptoms continue throughout an individual's lifespan. Irrázaval et al. (2005) presented a clinical description of the disorder based on diagnostic guidelines of the DSM- IV- TR, prevalence, and psychopathology from infancy through adolescence. Adult Autism was discussed through a case study of a 23-year old man brought to psychiatric evaluation by his mother. Neurobiological aspects of autism and the use of psychotropic medications in individuals with autism were also discussed. Quejada (2008) presented a clinical description of the disorder through genetic etiology, diagnosis, differential diagnosis, and prognosis. Flora de Barra also presented a clinical description of ASD based on the ICD- 10, genetic etiology, and differential diagnoses (1995).

Genetics

Individuals with autism usually present with cognitive difficulties. About 16–40 % of those with cognitive problems present with profound deficits. Flora de la Barra et al. (1986) reviewed a

case study of two identical twin girls with autism and profound cognitive deficits, who presented with an apparently balanced chromosomal translocation.

Treatment

After a definitive diagnosis of autism, it is important to evaluate how a child's development can be supported and their symptoms alleviated. Morales et al. (1995) presented a case study in which a 9 year-old institutionalized boy with autism improved in the areas of language, social interaction, and stereotypical behavior after several months of systems-focused family therapy with his father, mother, and older brother.

Review of Aetiologies and Alternative Treatments

A growing number of parents are adopting alternative or complementary treatments such as diet restrictions, chelation therapy for heavy metals, the use of hyperbaric oxygen chambers, elimination diets, as well as refusing to vaccinate their children due to a variety of beliefs regarding ASD aetiological hypothesis. Higuerra (2010) presented a critical literature review of studies related to these treatments, highlighting their methodological inadequacies and inconclusiveness. This article is very important to furthering professional development in Latin America. From a cultural perspective, the fact that it is written in Spanish, by a Chilean, adds a great deal of validity and weight to its contents.

Though research in the area of autism in Chile is scarce, the articles that do exist seem to represent the level of knowledge of the field among some professionals, as well as accounts of clinical work with patients.

It is important to note that there are no articles published in PubMed specifically related to Chile and Autism

Government Publications

The Chilean government has published three informative guides on ASD for teachers and health-care professionals, respectively: two through the Ministry of Education and one through the Ministry of Health.

Ministry of Education

Both guides from Ministry of Education are written specifically for teachers. One focuses on detection, intervention, and teaching methods for solely children in the preschool age group, while the second focuses on all schooling levels, including university.

For the preschool level, the guide begins by describing ASD based on the DSM- IV-TR diagnostic criteria, history of the diagnosis and the three areas of impairment. There is also a presentation of aetiological theories. The guide also presents guidelines for early detection and intervention, as well as suggestions on how to speak to families if a need for a clinical evaluation is suspected. Lastly, the guide presents recommendations for the educational-teaching process. It gives concrete examples for preparing lessons, how to structure the learning environment based on the needs presented through the disorder, how to involve the family in the continual learning process at home and provides examples of activity planning and execution (Ministerio de Educación 2010).

Though the guide can be seen as generally helpful, it does have several and significant deficiencies. One being that some information provided is conflicting and at times simply wrong, such as the description of Asperger's Syndrome as a language disorder. Providing false information to a group of professionals who are not likely to receive any further education or training in the area can have limitless repercussions. The guide also gives the impression that intervention at 5 or 6 years of age can still be considered early, and does not emphasize the need for the earliest intervention possible in order to obtain the best outcome for the child (Ministerio de Educación 2010).

The second educator's guide for all school levels also speaks in length about the history of the disorder, diagnostic criteria, and early alert indicators. It presents examples of the M-CHAT and ASSQ for educators' use. The guide describes the specific early intervention methods of Lovaas and TEACCH. For school-aged children, the guide discusses the need for individual evaluation of each child's needs and gives general tips on

supporting a child through their scholastic development. Subsequently, it describes specific characteristics of children with a diagnosis of Asperger's Syndrome.

Lastly, the guide describes the general abilities and impairments that young adults with Asperger's Syndrome typically present in relation to their entrance into the University setting. It provides suggestions for teachers as well as administrators in how to best teach and provide support for individuals with Asperger's Syndrome (Ministerio de Educación 2010).

It is important to note, that in speaking with the author of the guide, she stated that the guide was commissioned as a demonstration of best practice by the Ministry of Education. However to her knowledge, the guide is not actually in significant use within the school system.

Ministry of Health

The Ministry of Health has also published a guide for the clinical detection and diagnosis of ASD, for health-care professionals. All guidelines follow international standard. Steps of early detection, diagnostic evaluation through multidisciplinary teams, and therapeutic interventions are discussed. A multifaceted evaluation using standardized measures is recommended. Behavioral focused treatment options presented include ABA, PTR, and early intensive behavioral intervention. DIR, Floortime, and sensory integration are also discussed. Augmentative communication systems such as PECS, TEACCH, and TCC are presented, and the roles of various medical professionals in the treatment process are explained.

Though this guide presents a best practice model of detection, diagnosis, and treatment for ASD, its application in practice is virtually impossible in Chile due to the multiple hindering factors discussed previously. Based on statements from the four families interviewed and the four cases reviewed, in relation to their diagnostic processes and experiences, it does not seem that the contents of this guide are being taught to the professionals most likely to be involved in the lives of individuals with ASD and their families.

Social Policy and Training

Autism profoundly affects individuals with the diagnosis, their families, and in turn societies. In reviewing the data collected for this chapter on Autism in Chile, it seems as if the knowledge to improve the lives of individuals with ASD and their families through correct early diagnosis, effective early intervention, and treatment as well as long-term support provision is available. It is the lack of implementation of this knowledge that prohibits thousands of individuals with ASD in Chile from receiving the quality and quantity of services needed to reach their full potential.

Governments have an obligation to provide support to individuals with ASD. In Chile, governmental interest and investment in all areas related to autism would be the first step to improving the outcomes for all autistic individuals. Government bodies must guarantee that all resources, including proper diagnosis, high quality treatment, and education are both financially and physically accessible to all its citizens. This can be achieved through proper government investment. This can also be achieved through the regulation of private health sector and the services offered within the system.

The Ministry of Education must ensure that their educational professionals are trained in all aspects of autism and on how to provide students with ASD with the best education and care possible. This would include training for all professionals currently in the field, as well as incorporating autism education and training into the curricula of all public and private educational institutions.

Governments also have a responsibility in overseeing the education and training of its medical professionals, regardless in which capacity they practice or in which school of medicine they have trained. Professionals practicing in Chile need to be trained in the variety of aspects of ASD, its differential diagnosis and treatment options. Diagnosis and treatment methods must be included in the curricula of all medical schools. This training is vital for primary care professionals, who are typically the first professionals to come in contact with children who present with

worrisome signs and symptoms of developmental delays and distortions. Initiatives for early diagnosis and intervention must be made a national medical priority. Correct early diagnosis and intervention also reduces the financial, emotional, and practical strains and burdens experienced by family members.

Governments through their Ministries of Health and Education must also take an active role in providing services to individuals with ASD. These governmental bodies must define a protocol which would clearly delineate the path of "best care" through "best practice strategies." They must train health, educational professionals, and parents about these strategies and enforce their implementation.

Government interest is also needed to improve research in the area of autism in Chile. Without knowing the prevalence and the status of the disorder in a population, it is difficult to develop effective strategies for prevention, intervention, long-term care, and positive outcomes that reflect the specific needs of the country and culture.

In regards to service provision, it is important that all individuals have access to support and services throughout their lifespan: child, adolescence, and adulthood. This promotes personal development, better outcomes, and alleviates some of the strain experienced by family members responsible for lifetime care. Each child diagnosed with autism today will one day be an adult with autism. There is a great need in Chile for more vocational training and rehabilitation centers throughout the country to accommodate the need of adults with ASD.

Lastly, there is a great need for equalization of accessibility to quality care provision between the public and private sectors.

Autism is a universal severe developmental disorder that is also present in Chile. The development of service provision for individuals with autism has sprung from the determination and self-reliance of parents and family members and in most cases it seems still greatly dependent on their resilience and fortitude. Currently, lack of interest, investment, and regulation by government bodies hinders the development of research and practice in the field, as well as access to

quality medical care, treatment, and support services for many Chileans with ASD and their families.

Acknowledgment The authors of this study acknowledge that during its preparation for publication, the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition. (American Psychiatric Association 2013) was published, and acknowledge that under these new diagnostic guidelines, the definition of Autism and ASD has changed. However, the authors also acknowledge that when changes occur, the utilization of new diagnostic guidelines is a scientific and cultural process for both professionals and patients. Thusly, the authors chose to present the study's data as defined by the Diagnostic and Statistical Manual of Mental Disorders 4th Edition, Text Revision (American Psychiatric Association 2000).

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author. Text Revision.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- de Salud, M. (2011). *Guía de Práctica Clínica de Detección y Diagnóstico Oportuno de los Trastornos del Espectro Autista (TEA) [Practical clinical guide the timely detection and diagnosis of autism spectrum disorders (ASD)]*. Santiago, Chile: MINSAL.
- Flora de la Barra, M. (1995). Aspectos Biológicos del Autismo [Biological aspects of Autism]. *La Revista Chilena de Neuropsiquiatría*, 33, 361–365.
- Flora de la Barra, M., Skoknic, V., Allicnde, A., Raimann, E., Cortes, F., & Lacassie, Y. (1986). Autism and mental retardation associated with (7;20) balanced chromosomal translocation in a pair of female twins. *La Revista Chilena de Pediatría*, 57, 549–554.
- Gobierno de Chile. (2013). *Ministros*. Retrieved August 29, 2013 from <http://www.gob.cl/ministros/>
- Higuerra, M. (2010). Biological treatments of Autism, elimination diets: A critical review. *La Revista Chilena de Pediatría*, 81, 204–214. Lit review.
- Irrázaval, M. E., Brokering, W., & Murillo, G. (2005). Autismo: An adult psychiatry perspective. *La Revista Chilena de Neuropsiquiatría*, 43, 51–60.
- Ministerio de Educación. (2009). *Unidad de Educación Especial. Guía de Apoyo Técnico- Pedagógico: Necesidades Educativas Especiales en el Nivel de Educación Parvularia Asociadas al Autismo* [Technical- pedagogical support guide: Special educational needs on the kindergarden educational level associated with autism] (1st ed.) Santiago, Chile: Author.
- Ministerio de Educación. (2010). *Unidad de Educación Especial. Manual de Apoyo a Docentes: Educación de Estudiantes que Presentan con Trastornos del Espectro Autista* [Support manual for teachers: Education for students that present with an autism spectrum syndrome] (1st ed.) Santiago, Chile: Author.
- Ministerio de Educación. (2013). Unidad de Educación Especial. Directorio de Establecimientos. Retrieved May 16, 2013 from http://www.educacionespecial.mineduc.cl/index2.php?id_portal=20&id_seccion=2543&id_contenido=23559. Excel document “Escuelas Especiales”. [Special Education Schools]. Excel document “Escuelas con Programa de Intergración Escolar”. [Public Schools with an Educational Intergration Program].
- Ministerio de Salud. (2010). *Acceso Universal Garantías Explícitas* [Explicit guarantees of universal access]. Chile: Minsal. Retrieved May 27, 2017 from http://www.minsal.gob.cl/portal/url/page/minsalcl/g_gesauge/presentacion.htmlhttp://www.minsal.gob.cl/portal/url/page/minsalcl/g_gesauge/presentacion.html
- Morales, M., Martínez, R., & Valdés, A. (1995). Un modelo de acción con un miembro autista. A model of action with an autistic family member]. *La Revista Chilena de Psiquiatría*. 1, 26–33. Chile.
- Office of Autism Research Coordination. (OARC), National Institute of Mental Health and Thomson Reuters, Inc. on behalf of the Interagency Autism Coordinating Committee (IACC). *IACC/OARC autism spectrum disorder research publications analysis report: The global landscape of autism research*. July 2012. Retrieved May 23, 2013 from the Department of Health and Human Services Interagency Autism Coordinating Committee website: <http://iacc.hhs.gov/publications-analysis/july2012/index.shtml>
- Quijada, C. (2008). Espectro Autista [Autism spectrum]. *La Revista Chilena de Pediatría*, 79, 86–91.
- World Health Organization. (2010). Chile. Retrieved May 27, 2013 from <http://apps.who.int/gho/data/view.country.6300>
- Zwaigenbaum, L., Bryson, S., & Garon, N. (2013). Early identification of autism spectrum disorders. *Behavioral Brain Research*. Retrieved May 27, 2013 from <http://www.ncbi.nlm.nih.gov/pubmed/23588272>

China and Autism

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Historical Background

The history of autism in China is a brief one, as it was not diagnosed there until 1982 (Tao 1987). Since that point, the landscape of research and scholarly work pertaining to the epidemiology

and clinical care of autism and related disorders has been relatively bare. Some of this can be attributed to the fact that dozens of dialects are spoken throughout the mainland, leading to a relative lack of appropriately translated materials relating to the diagnostic and treatment practices of autism (Ming 2013). This has left many doctors, teachers, and a majority of the general public with a lack of awareness and understanding of the disorder.

China has a long history of special education schools, dating back to the early twentieth century and through the times of Mao Zedong (Deng et al. 2001). These institutions have been mostly geared toward those suffering from blindness and deafness, rather than those with intellectual disabilities, however (Yang and Wang 1994). Consequently, programs and schools for those with autism have been much more difficult to come by.

Legal Issues, Mandates for Services

Beginning in 1986, the Chinese Government began enacting a slew of legislation intending to benefit the disabled. The first landmark act was the China Compulsory Education Law, which aimed to require public schools to accept children with special needs (Deng et al. 2001). This was followed in 1990 by the Law of the People's Republic of China on the Protection of Persons with Disabilities, which called for additional protections of Chinese individuals with disabilities under the law (McCabe 2007). Moreover, through its Ninth, Tenth, and Eleventh Five-Year Plans, the Chinese Government continued to outline ways in which to increase support for disabled children as well as improve their enrollment in schools (Ming 2013). Yet, although autism was first diagnosed in 1982, it was not officially included as a disability covered by Chinese law until 2006 (Gu 2007).

Despite these apparent attempts to increase support for those with autism and other disorders, the vague goals outlined in such legislation

have yielded minimal results. Autistic children are still often refused an education from government-run public schools, including special education ones (Huang and Wheeler 2007). Public schools can cost almost half of a Chinese citizen's average annual salary, while private schools often cost two to three times the average amount (Ming 2013). For children that live in rural areas, it can be incredibly difficult to even find a school that offers the proper services (Wang et al. 2011). It is not uncommon for a special education school to admit an autistic child only to later declare that its teachers have an improper background in working with children with such conditions (Rubin 2000).

Due to the shortcomings of China's state-run programs and educational system, many families and autistic individuals seek intervention from private organizations (McCabe 2004). Such treatment is paid for out of pocket by parents or families (Gu 2007; McCabe 2007), which can become an obvious financial burden. The cost of such services in China is an issue for many families.

Overview of Current Treatments and Centers

While Chinese public and private schools have fallen short for most autistic children, a number of nongovernmental organizations have begun to emerge. One of the more notable examples is BARAC: the Beijing Association for the Rehabilitation of Autistic Children (Feinstein 2010). Run completely off of fees and private donations, BARAC offers a hotline for parents of autistic children, publishes newsletters and journals to raise autism awareness, and oversees two schools specifically for autistic children, each with over 100 students (Feinstein 2010). A number of similar centers now exist within China's major cities, though very few exist in China's rural areas and countryside. Many of these organizations have been started by the parents of autistic children who had difficulty finding services or getting an education elsewhere (McCabe 2007).

In terms of the diagnosis of autism within China, many doctors use the *Chinese Classification of Mental Disorders, Third edition* (CCMD-3) (Wu and Zhang 2011). Professionals have argued that changes should be made to improve the accuracy and consistency of diagnoses of autism (Wu and Zhang 2011). Currently, very few internationally recognized clinical diagnostic tests, such as Autism Diagnostic Interview-Revised (ADI-R) and the Autism Diagnostic Observation Schedule (ADOS), are used by doctors (Wu and Zhang 2011).

Overview of Research Directions

Research on autism in China has been very limited, though the volume has been increasing as awareness has increased. Moreover, organizations such as the Autism Consortium China are emerging and beginning to change the landscape of research within the nation (Wu and Zhang 2011). Launched in 2009 by a group of Chinese research scientists and doctors, the Autism Consortium China seeks to spread awareness of autism in Chinese society, help standardize and improve diagnostic procedures, and conduct extensive research on autism within China (Wu and Zhang 2011). While much of the research that has been conducted within China has focused on infantile autism, early intervention, or special education, adults with autism have seemed to be an area that research has neglected.

Overview of Training

Public schools often justify their rejection of autistic children by claiming that their teachers have no training in working with autism (Rubin 2000). Indeed, most instructors in schools and even at intervention programs specifically geared toward autistic children have little to no relevant training, mostly due to the small number of universities throughout the country that offer such a field of

study (McCabe 2013). Many teachers even express a strong desire for more training, especially relating to adolescent intervention (McCabe 2013). At times, passionate teachers with a will to help autistic children are bound by China's limited awareness and lack of resources.

Social Policy and Current Controversies

Autism, along with many other disabilities and illnesses such as ADHD, schizophrenia, and epilepsy, has been severely stigmatized in Chinese culture (Kelly 2007). There are some deeply rooted cultural explanations for such a social context. In the times of Confucius, the mentally and physically disabled were a part of the lowest social status (Deng et al. 2001). In Mainland China, many still refer to autism and other spectrum disorders as “gudu zheng” which translates to “lonely disease” (Feinstein 2010). Moreover, studies have suggested that families of autistic children have experienced increased levels of stress related to pessimism shame (Wang et al. 2011). This social context and intense stigmatization within Chinese culture often incentivizes families or individuals to hide one's autism or disability rather than treat it. A large number of parents cut their disabled children off from outside social interaction, including schooling for this reason (Wang et al. 2011).

See Also

► Culture and Autism

References and Reading

- Deng, M., Poon-Mcbrayer, K. F., & Farnsworth, E. B. (2001). The development of special education in China: A sociocultural review. *Remedial and Special Education*, 22(5), 288–298. <https://doi.org/10.1177/074193250102200504>.
- Feinstein, A. (2010). *A history of autism: Conversations with the pioneers*. Chichester: Wiley-Blackwell.

- Gu, Y. (2007). Gudu zheng ertong qidai geng duo guanzhu [Children with autism look forward to more attention]. *Xinwen Shijie (News World)* January: 11.
- Hu, Y. (2010). Training Needs for Implementing Early Childhood Inclusion in China. *International Journal of Early Childhood Special Education*, 12–30. Retrieved 2 Sept 2014.
- Huang, A. X., & Wheeler, J. J. (2007). Including children with autism in general education in China. *Childhood Education*, 83(6), 356–360. <https://doi.org/10.1080/00094056.2007.10522950>.
- Kelly, T. (2007). Transforming China's mental health system: Principles and recommendations. *International Journal of Mental Health*, 36(2), 50–64. <https://doi.org/10.2753/IMH0020-7411360205>.
- Kuo-Tai, T. (1987). Brief report: Infantile autism in China. *Journal of Autism and Developmental Disorders*, 17(2), 289–296. <https://doi.org/10.1007/BF01495062>.
- McCabe, H. (2004). China: NGOs and education for children with autism. In *Civil society or shadow state: State/NGO relations in education*. Greenwich, CT: Information Age Publishing
- Mccabe, H. (2007). Parent advocacy in the face of adversity: Autism and families in the People's Republic of China. *Focus on Autism and Other Developmental Disabilities*, 22(1), 39–50. <https://doi.org/10.1177/10883576070220010501>.
- Mccabe, H. (2013). Bamboo shoots after the rain: Development and challenges of autism intervention in China. *Autism*, 17(5), 510–526. <https://doi.org/10.1177/1362361312436849>.
- Ming, J. (2013). Autism in China: A biosocial review. *Journal of Global Health*. Retrieved September 2, 2014, from <http://www.ghjournal.org/?p=6140>
- Rubin, K. (2000, October 19). Chinese charities' long March. Retrieved September 2, 2014, from <http://philanthropy.com/article/Chinese-Charities-Long-March/54378/>
- Tao, K. (1987). Brief report: Infantile autism in China. *Journal of Autism and Developmental Disorders*, 17(2), 289–296.
- Wang, P. (2008). Effects of a parent training program on the interactive skills of parents of children with autism in China. *Journal of Policy and Practice in Intellectual Disabilities*, 5(2), 96–104. <https://doi.org/10.1111/j.1741-1130.2008.00154.x>.
- Wang, P., Michaels, C. A., & Day, M. S. (2011). Stresses and coping strategies of Chinese families with children with autism and other developmental disabilities. *Journal of Autism and Developmental Disorders*, 41(6), 783–795. <https://doi.org/10.1007/s10803-010-1099-3>.
- Wu, B., & Zhang, Z. (2011). Current status of autism spectrum disorder in China – Summary on the 369th Xiangshan science conferences. *American Chinese Journal of Medicine and Science*, 4(3), 167. <https://doi.org/10.7156/v4i3p167>.
- Yang, H. L., & Wang, H. B. (1994). Special education in China. *The Journal of Special Education*, 28, 93–105.

Chlorpromazine

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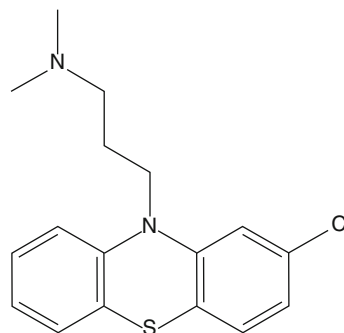
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Synonyms

3-(2-chloro-10 H-phenothiazin-10-yl)-N,N-dimethylpropan-1-amine hydrochloride; Chlorpromazine hydrochloride; Thorazine

Definition



Chlorpromazine is a prescription drug in the group of first-generation antipsychotics initially FDA-approved for medical use in the year 1957 whose active ingredients are chlorpromazine and chlorpromazine hydrochloride which have the chemical formulas $C_{17}H_{19}N_2SCl$ and $C_{17}H_{19}N_2SCl \cdot HCl$, respectively. This drug is currently only available in generic form. This drug can be used for the treatment of schizophrenia,

bipolar mania, some psychotic symptoms of dementia, and serotonin syndrome. Observed side effects include drowsiness/sedation, parkinsonism, orthostatic hypertension, tachycardia, ECG abnormalities, anticholinergic effects, galactorrhea, weight gain, photosensitivity, rashes, and pigmentation.

See Also

► [Antipsychotics: Drugs](#)

References and Reading

- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001). *Principles and practice of psychopharmacotherapy* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
- Stahl, S. M. (2000). Antipsychotic agents. In *Essential psychopharmacology: Neuroscientific basis and clinical applications* (pp. 401–458). Cambridge, MA: Cambridge University Press.
- Thioridazine. (n.d.). Retrieved from the ChemSpider Wiki: <http://www.chemspider.com/Chemical-Structure.5253.html>.
- U.S. Food and Drug Administration. (2011). *Drugs@FDA*. Retrieved from: <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm>.
- Wilkaitis, J., Mulvihill, T., & Nasrallah, H. A. (2006). Classic antipsychotic medications. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 211–228). Washington, DC: American Psychiatric Publishing.

Chlorpromazine Hydrochloride

► [Chlorpromazine](#)

Cholinergic

► [Acetylcholine: Definition](#)

Cholinergic System

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Definition

The cholinergic system utilizes acetylcholine (ACh) neurotransmission to regulate memory, arousal, concentration, attention, and consciousness (Sadock et al. 2009). ACh projects from the brainstem neurotransmitter center and basal forebrain to numerous locations, including the prefrontal cortex, basal forebrain, thalamus, hypothalamus, amygdala, and hippocampus (Stahl 2008). ACh is formed from two precursors: choline, synthesized from the diet and intraneuronal sources, and acetyl coenzyme A (AcCoA), made from glucose in the neuronal mitochondria. The enzyme choline acetyltransferase acts on choline and AcCoA to create ACh.

ACh acts on muscarinic and nicotinic cholinergic receptors. Muscarinic receptors are so-named due to their binding preference for muscarine, a toxin found in poisonous mushrooms (Sadock et al. 2009). The five muscarinic receptors are M₁, M₂, M₃, M₄, and M₅, and each has a different anatomical structure. They are G protein-linked and can be excitatory or inhibitory. The M₁ subtype on the postsynaptic neuron is believed to regulate some memory functions. M₁ receptors blocked by antipsychotic medications can induce sedation and some cognitive dysfunction. The presynaptic M₂ receptor is an autoreceptor, detecting excess ACh in the synapse and preventing further release of ACh. M₃ receptors in pancreatic beta cells cause insulin secretion, so antagonism here by atypical

antipsychotics, like olanzapine and clozapine, can result in decreased insulin secretion.

Nicotinic acetylcholine receptors (nAChR) belong to a class of excitatory, ligand-gated ion channel receptors (Sadock et al. 2009). They bind nicotine, the main addictive substance in tobacco smoke. These receptors have subtypes with variable affinities; the highest-affinity subunits are in the thalamus, followed by the substantia nigra, striatum, hippocampus, and entorhinal cortex. There are fewer high-density receptors in the cerebellar, parietal, and frontal cortices. One of the most notable subtypes is the postsynaptic alpha-4 beta-2 subunit, which is believed to regulate dopamine release in the nucleus accumbens (Stahl 2008). This is the likely target of tobacco nicotine in the brain, strongly contributing to tobacco's addictive qualities. The alpha-7 subunit located on the postsynaptic neuron is thought to regulate cognitive function in the prefrontal cortex, whereas the presynaptic alpha-7 subunit on cholinergic neurons provides positive feedback for continued release of ACh. The alpha-7 subunit of the nicotinic receptor located on dopamine and glutamate neurons also regulates the release of these neurotransmitters when ACh is present.

The neurotransmitter ACh is partly regulated by two degradative enzymes, acetylcholinesterase (AChE) and butyrylcholinesterase (BuChE). These enzymes convert ACh back to choline, which is taken back up into the neuron for resynthesis into ACh (Stahl 2008). AChE is considered the main enzyme that inactivates ACh. It is located throughout the brain, along the major projections as outlined above, as well as within the gastrointestinal (GI) tract, skeletal muscle, red blood cells, lymphocytes, and platelets. The highest density of AChE is located in the caudate and putamen, with lower amounts in areas such as the thalamus, hippocampus, and cortices (frontal, temporal, parietal, occipital, and cerebellum). BuChE is also located throughout the brain, mostly in glial cells, but can also be found in the GI tract, plasma, skeletal muscle, placenta, and liver (Stahl 2008). ACh is partly regulated by cholinergic vesicular transporters (VACHT) on synaptic vesicles, which transport ACh into the vesicle (Sadock et al. 2009). The highest densities

of VACHT are also located in the caudate and putamen, as well as the nucleus accumbens. Lower levels of binding occur in the cerebral cortex and cerebellum.

Anticholinergic medications are some of the most well-known medications to act on the cholinergic system. They block the actions of ACh at either the muscarinic or nicotinic receptors, resulting in side effects such as sedation, analgesia, and management of allergies (Sadock et al. 2009). These drugs impact numerous physiological systems, including the ocular, cardiovascular, respiratory, GI, genitourinary, and the central nervous system (CNS). In the CNS, these medications may be initially stimulating, followed by a longer lasting sedative effect. Adverse effects include confusion, disorientation, hallucinations, and memory impairment. In the eye, anticholinergic agents cause paralysis of the ciliary muscle, leading to loss of accommodation, as well as muscarinic blockade of the iris' sphincter muscle, causing pupillary dilation. Additional ocular effects include blurry vision, anhidrosis, and worsened narrow-angle glaucoma. In the cardiovascular system, anticholinergic agents cause tachycardia due to muscarinic blockade of the parasympathetic fibers in the atria. In toxic doses, they can cause intraventricular conduction block. In the respiratory system, muscarinic blockade causes reduced glandular secretion of the smooth muscle, leading to dry mouth. In the GI tract, inhibited parasympathetic control from anticholinergic blockade leads to decreased motility, causing constipation, delayed gastric emptying, and paralytic ileus. In the genitourinary tract, anticholinergic agents relax the smooth muscle of the bladder and ureter, leading to urinary hesitancy, but they are also known to cause urinary retention. Despite their reputation for adverse effects, anticholinergic agents can be therapeutically useful. They are commonly prescribed to prevent or improve extrapyramidal side effects (EPS) caused by dopamine antagonists. EPS reactions include dystonia, akathisia, and parkinsonism. When antipsychotics block dopamine in the nigrostriatal tract, cholinergic activity is increased, resulting in the above-mentioned side effects. Anticholinergic agents reduce the

increased cholinergic activity, restore balance to the dysfunctioning neurotransmitter system, and relieve symptoms of EPS.

From a pathophysiological standpoint, the cholinergic system is most frequently associated with Alzheimer's disease (AD). In AD, there is degeneration of cholinergic neurons in the nucleus basalis due to deposition of amyloid plaque, leading to memory loss (Sadock et al. 2009). AChE inhibitors prevent the destruction of ACh, which prevents further memory loss in AD. Some AChE inhibitors only inhibit AChE, whereas some inhibit both AChE and BuChE. Depending on the individual, responses to these agents vary, but the overall effect is prevention or slowing of disease progression (Stahl 2008). Examples of AChE inhibitors are donepezil, amantadine, rivastigmine, and galantamine. Another disease process implicated in the pathophysiology of the cholinergic system is schizophrenia, as evidenced by the observation that antimuscarinic drugs improve negative symptoms (Sadock et al.). Anticholinergic agents are known to worsen positive symptoms in patients with unstabilized schizophrenia, but they appear to have no effect on positive symptoms in stabilized patients (Sadock et al.). The cholinergic system is also implicated in Parkinson's disease (PD), which results from dopamine deficiency and cholinergic excess. Anticholinergic agents can help reduce parkinsonian tremor via muscarinic receptor blockade, especially in combination with levodopa, a first-line dopaminergic agent used to treat PD.

Historical Background

ACh was the first neurotransmitter to be discovered. The first individual to uncover its existence was Henry Hallett Dale, a British pharmacologist who lived between 1875 and 1968 (Raju 1999). While studying ergot extracts, Dale found that the extracts reversed the effects of epinephrine and concluded that ergot contained tyramine, histamine, and ACh. In 1914, Dale determined that ACh was the "most suitable chemical" for parasympathetic neurotransmission, and coined the

terms "cholinergic" and "adrenergic." Not long after his discovery, a German physician named Otto Loewi (1873–1961) was researching the autonomic nervous system when he discovered the presence of ACh and adrenaline in isolated hearts. The year was 1921, and Loewi was the first individual to underscore ACh's importance in the nervous system. Loewi initially named ACh "vagusstoff," referencing its release from the vagus nerve. These two men shared the Nobel Prize in Physiology and Medicine in 1936 "for discoveries related to chemical transmission of nerve impulse."

Current Knowledge

Impairment of the cholinergic system has been implicated in the pathophysiology of autism. Postmortem studies by Perry et al. (2001) show a 30% reduction of cortical muscarinic receptor binding in the parietal cortex in autistic individuals compared with age-matched controls. Cholinergic neurons in the basal forebrain, an area thought to play a role in attention, are abnormally large and plentiful in children with autism (Baumann and Kemper 1994). A study by Sokol et al. (2002) found low cytosolic choline concentrations as measured by hydrogen proton magnetic resonance spectroscopy in ten children with autism. Imaging studies have also attempted to link neuroanatomical regions of the brain to core domains of dysfunction observed in autism. Individuals with autism have been noted to have significant deficits in face perception (Grelotti et al. 2002; Schultz 2005), which is believed to play a notable role in social interaction. The neuroanatomical region linked to facial recognition is the fusiform gyrus, which contains the visual pathway. This pathway is regulated by the cholinergic system, suggesting a possible causal relationship between the cholinergic system and autistic social impairment. A study by Suzuki et al. (2011) used positron-emission tomography (PET) and a radiotracer to examine AChE activity in 20 autistic adults compared to 20 age- and IQ-matched controls. The results showed a deficit in cholinergic innervations of the fusiform gyrus in the autistic

subjects, suggesting a possible explanation for social impairment in autism.

There is evidence to suggest that specific cholinergic receptor subtypes play a role in the pathology and symptomatology of autism. It is believed that deficits in alpha4-containing receptors predominate in autism (as well as in Alzheimer's disease), whereas other receptor subtypes are associated with other disorders, like the alpha-7 subtype and schizophrenia (Graham et al. 2002). These observations may lead to drug development targeting specific nicotinic receptor subtypes for alleviation of symptoms in autism. Similarly, a theory by Lippiello (2006) suggests that autism is a disorder of "overfocused attention," unlike attention-deficit/hyperactivity disorder (ADHD), which can be described as a disorder of "underfocused attention." These two disorders theoretically sit at opposite ends of a spectrum with reversed neurophysiological mechanisms underlying their pathophysiology. Lippiello hypothesizes that because nicotinic cholinergic agonists have been shown to improve the symptoms of ADHD (Levin et al. 2001); perhaps nicotinic cholinergic antagonists may ameliorate the symptoms of autism. The concept of nicotinic receptor antagonists treating autism has not yet been explored in the literature, but these concepts may lead to future initiatives in studying the relationship between the anticholinergic system and autism.

Medications affecting the cholinergic system, particularly AChE inhibitors, have been studied to treat symptoms associated with autism. These agents increase ACh in brain regions related to attention and memory, such as the cerebral cortex and basal forebrain (Yoo et al. 2007). Amantadine is a drug approved for the prophylaxis of influenza A, but is commonly used in the treatment of PD and EPS due to its antiparkinsonian effects (Sadock et al. 2009). A double-blind, placebo-controlled study examined the effects of amantadine in 39 autistic children aged 5–19 years (King et al. 2001). The clinician-rated Aberrant Behavior Checklist rating scale (ABC) showed statistical significance in the amantadine-treated group within the domains of hyperactivity and inappropriate speech

(Blakenship et al. 2011). The parent-rated ABC did not show statistically significant improvement between the amantadine and placebo groups. Galantamine, another AChE inhibitor, was examined in 20 males with autism in a double-blind, placebo-controlled study (Niederhofer et al. 2002). Using the ABC as a dependent measure, there were decreases in the domains of irritability, hyperactivity, inadequate eye contact, and inappropriate speech. Despite these promising observations, studies examining the effect of other AChE inhibitors have found dissimilar results. A double-blind, placebo-controlled study by Handen et al. (2011) looked at the effect of donepezil in 34 children and adolescents aged 8–17 years (IQ > 75). The results showed some improvement on a number of measures of executive functioning, but there were no statistically significant differences between the donepezil and placebo groups. The researchers concluded that short-term treatment with donepezil may have limited impact on cognitive functioning in those with autism.

Retrospective and open-label trials are of limited utility in demonstrating effectiveness and safety of a medication due to their lack of experimental design, but they offer a glimpse of possible directions that can be taken in the treatment of symptoms associated with autism. A retrospective study by Hardan and Handen (2002) examined the effects of donepezil, an AChE inhibitor, in the treatment of 8 children with autism, aged 7–19 years. The study found a significant decrease in irritability and hyperactivity according to the ABC, although attention and memory were not measured. An open-label study by Nicolson et al. (2006) examined the effects of galantamine, an AChE inhibitor and nicotine receptor modulator, in the treatment of 13 children with autism. Galantamine demonstrated reductions in parent-rated irritability and social withdrawal on the ABC, improvements in emotional lability and inattention on the Conners' Parent Rating Scales-Revised, and reduced anger on the clinician-rated children's Psychiatric Rating Scale. Hertzman (2003) reported three cases where galantamine promoted verbalization in adults with autism.

Another open-label study by Chez et al. (2004) examined the AChE inhibitor rivastigmine tartrate and found significant improvements in scores of various measurements, including the Childhood Autism Rating Scale, Gardner's Expressive One-Word Picture Vocabulary Test, and Conners' Parent Rating Scale.

Future Directions

Future directions for research into the relationship between the cholinergic system and autism will likely involve investigating cholinergic receptor subtypes, neuroimaging, and pharmacologic treatment development. Cholinergic receptor subtypes occur at variable concentrations in the brain and are implicated in the pathophysiology of autism. Exploring their influence on attention, memory, and cognition, as well as the core diagnostic domain of social impairment, will likely be a continued area of research. Neuroimaging of these receptors will continue to map areas of neuroanatomical dysfunction in autism. Medications affecting the cholinergic system could be further explored as treatments for autism given the mixed results seen in existing studies. Double-blind, placebo-controlled trials are required to draw conclusions about medication safety and efficacy, and currently there are minimal studies examining the effectiveness of AChE inhibitors. Short-term studies of AChE inhibitors have shown mixed results, so it may be of benefit to employ them for longer periods before drawing definitive conclusions about their efficacy. Lastly, the study of nicotinic cholinergic receptor antagonists as a treatment for autism is another possible, untapped direction.

See Also

- Amantadine
- Anticholinergic
- Antipsychotics: Drugs
- Atypical Antipsychotics
- Dopamine

References and Reading

- Baumann, M. L., & Kemper, T. L. (1994). *Neuroanatomic observations of the brain in autism* (pp. 119–145). Baltimore: Johns Hopkins University Press.
- Blakenship, K., Erickson, C. A., et al. (2011). Psychopharmacological treatment of autism, Chapter 69. In D. G. Amaral, G. Dawson, & D. H. Geschwind (Eds.), *Autism spectrum disorders* (pp. 1194–1212). New York: Oxford University Press.
- Chez, M. G., Aimonovitch, M., et al. (2004). Treating autistic spectrum disorders in children: Utility of the cholinesterase inhibitor rivastigmine tartrate. *Journal of Child Neurology*, 19(3), 165–169.
- Graham, A. J., Martin-Ruiz, C. M., et al. (2002). Human brain nicotinic receptors, their distribution and participation in neuropsychiatric disorders. *Current Drug Targets CNS and Neurological Disorders*, 1(4), 387–397.
- Grelotti, D. J., Gauthier, I., et al. (2002). Social interest and the development of cortical face specialization: What autism teaches us about face processing. *Developmental Psychobiology*, 40(3), 213–225.
- Handen, B. L., Johnson, C. R., et al. (2011). Safety and efficacy of donepezil in children and adolescents with autism: Neuropsychological measures. *Journal of Child and Adolescent Psychopharmacology*, 21(1), 43–50.
- Hardan, A. Y., & Handen, B. L. (2002). A retrospective open trial of adjunctive donepezil in children and adolescents with autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, 12(3), 237–241.
- Hertzman, M. (2003). Galantamine in the treatment of adult autism: A report of three clinical cases. *International Journal of Psychiatry in Medicine*, 33(4), 395–398.
- King, B. H., Wright, D. M., et al. (2001). Double-blind, placebo-controlled study of amantadine hydrochloride in the treatment of children with autistic disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40(6), 658–665.
- Levin, E. D., Conners, C. K., et al. (2001). Effects of chronic nicotine and methylphenidate in adults with attention deficit/hyperactivity disorder. *Experimental and Clinical Psychopharmacology*, 9(1), 83–90.
- Lippiello, P. M. (2006). Nicotinic cholinergic antagonists: A novel approach for the treatment of autism. *Medical Hypotheses*, 66(5), 985–990.
- Nicolson, R., Craven-Thuss, B., et al. (2006). A prospective, open-label trial of galantamine in autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, 16(5), 621–629.
- Niederhofer, H., Staffen, W., et al. (2002). Galantamine may be effective in treating autistic disorder. *BMJ*, 325(7377), 1422.
- Perry, E. K., Lee, M. L., et al. (2001). Cholinergic activity in autism: Abnormalities in the cerebral cortex and basal forebrain. *The American Journal of Psychiatry*, 158(7), 1058–1066.

- Raju, T. N. (1999). The Nobel chronicles. 1936: Henry Hallett Dale (1875–1968) and Otto Loewi (1873–1961). *Lancet*, 353(9150), 416.
- Sadock, B. J., Sadock, V. A., & Ruiz, P. (2009). *Kaplan & Sadock's comprehensive textbook of psychiatry, volumes 1 & 2* (9th ed., pp. 67, 279–282, 298, 3014–3021). Philadelphia: Lippincott Williams and Wilkins.
- Schultz, R. T. (2005). Developmental deficits in social perception in autism: The role of the amygdala and fusiform face area. *International Journal of Developmental Neuroscience*, 23(2–3), 125–141.
- Sokol, D. K., Dunn, D. W., et al. (2002). Hydrogen proton magnetic resonance spectroscopy in autism: Preliminary evidence of elevated choline/creatine ratio. *Journal of Child Neurology*, 17(4), 245–249.
- Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd ed., pp. 206–207, 392, 449, 914–926). New York: Cambridge University Press.
- Suzuki, K., Sugihara, G., et al. (2011). Reduced acetylcholinesterase activity in the fusiform gyrus in adults with autism spectrum disorders. *Archives of General Psychiatry*, 68(3), 306–313.
- Yoo, J. H., Valdovinos, M. G., et al. (2007). Relevance of donepezil in enhancing learning and memory in special populations: A review of the literature. *Journal of Autism and Developmental Disorders*, 37(10), 1883–1901.

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Major Appointments

(Institution, Location, Dates) University of Gothenburg (from 1983) and Institute of Child Health, London (from 2004) and University of Glasgow (from 2003)

Major Honors and Awards

Fernström Prize for Medicine in 1991, Ingvar Award in 1995, The Ronald McDonald Major Award for Paediatrics in 1998, Ågrenska Major Medicine Prize in 2001, Philips Nordic Prize in

2004. In 2009, Carl XVI Gustav of Sweden presented Gillberg with The King's Medal of the Seraphim order for his contributions in the field of child and adolescent psychiatry. He received the Dahlberg award for his genetic research and the Life Watch Award for Autism Research in 2010. In 2012 he was awarded one of Sweden's most prestigious scientific honors: the Söderberg Prize for Medicine ("Little Nobel Prize"). In 2016 he was presented with the INSAR Lifetime Achievement Award at the International Meeting for Autism Research (IMFAR).

Landmark Clinical, Scientific, and Professional Contributions

Since the early 1980s, Christopher Gillberg has been publishing practice-changing work on autism: his first paper on the subject is a typically rigorous population-based study on autism and maternal age (Gillberg 1980). His studies have always been highly clinically relevant and, wherever possible, population based, and this has allowed him to see trends in autism spectrum disorders (ASD) that others have missed. He was the first to notice that anorexia nervosa and autism might be related (Gillberg 1983a), and this was later borne out in population-based research. Early in his career, he demonstrated that autism was a more common disorder than first thought (Gillberg et al. 1991) – a finding that was not generally believed at the time, but which is now widely recognized (Lundström et al. 2015).

With child neurologist Mary Coleman, he demonstrated that autism is really "the autisms," with a large proportion of autism diagnoses explained by a multitude of different etiological mechanisms (Gillberg and Coleman 1992) – and that different underlying etiologies might be manifested in different behavioral phenotypes (clinical presentations). This work stimulated an interest in the biological underpinnings of ASD, and a collaboration with Marion Leboyer and with Thomas Bourgeron and colleagues at the Institut Pasteur has elucidated a range of genetic findings that have demonstrated the importance of problems in synapse and cell membrane functioning in

people with autism (Jamain et al. 2003). These findings may well usher in a new era of treatment for ASD.

His rigorous epidemiological approach has shown beyond doubt that autism is a lifelong condition (Hofvander et al. 2009) that is rarely “pure”: co-occurrence with other disorders and problems is the rule (Gillberg 1983b); children with autism are more likely than typically developing children to suffer from attention deficit hyperactivity disorder, tic disorders, and learning and motor problems (Gillberg 2010). An important corollary of this is that “pure” autism may not be problematic across the lifespan – that difficulties experienced often come from the comorbidities, not only from the autism “per se” (Gillberg and Fernell 2014).

Short Biography

As a young boy, Christopher Gillberg’s first wish was to be a film director, and his intense creativity was also expressed in music and painting. However, he was also exceptionally talented academically and had the best exam results in Sweden at the end of his school career. Because he was unusually young when leaving school, he could not enter film school but, instead, was encouraged toward a career in medicine – a field that has since benefited from that intense creativity.

He married Carina Gillberg in 1969, and they have five children. Carina Gillberg is also a child and adolescent psychiatrist, and they have published many papers together, including the widely used Gillberg and Gillberg criteria for Asperger’s syndrome (Gillberg and Gillberg 1989).

As a young doctor, Christopher was asked to oversee a unit for young people with learning disabilities, and his lifelong fascination with autism was born. At that time, autism was usually treated with psychoanalysis, and the frustrations of this approach to what struck Christopher as a brain-based disorder sparked his interest in research.

Throughout his career, his main clinical and research base has been the University of Gothenburg, but his career has been characterized by many fruitful international collaborations. In

1993 he was Fulbright visiting professor at New York University Medical School, and he holds, or has held, full, visiting, or honorary professorships at the Universities of London, Glasgow, Edinburgh, Strathclyde (Glasgow), Kochi (Japan), Odense (Denmark), Bergen (Norway), San Francisco, the Institute of Child Health (London), and the Institut Pasteur (Paris).

He has published more than 650 peer-reviewed scientific papers (more than 600 are on PubMed), and the contribution of his research to clinical practice and social policy is both broad and profound.

References and Reading

- Gillberg, C. (1980). Maternal age and infantile autism. *Journal of Autism and Developmental Disorders*, 10(3), 293–297.
- Gillberg, C. (1983a). Are autism and anorexia nervosa related? *The British Journal of Psychiatry*, 142(4), 428–428.
- Gillberg, C. (1983b). Perceptual, motor and attentional deficits in Swedish primary school children. Some child psychiatric aspects. *Journal of Child Psychology and Psychiatry*, 24(3), 377–403.
- Gillberg, C. (2010). The ESSENCE in child psychiatry: Early symptomatic syndromes eliciting neurodevelopmental clinical examinations. *Research in Developmental Disabilities*, 31(6), 1543–1551.
- Gillberg, C., & Coleman, M. (1992). The biology of the Autisms. In *The autisms* (pp. viii, 317). Oxford: Mac Keith Press.
- Gillberg, C., & Fernell, E. (2014). Autism plus versus autism pure. *Journal of Autism and Developmental Disorders*, 44(12), 3274–3276.
- Gillberg, I. C., & Gillberg, C. (1989). Asperger syndrome—Some epidemiological considerations: A research note. *Journal of Child Psychology and Psychiatry*, 30(4), 631–638.
- Gillberg, C., Steffenburg, S., & Schaumann, H. (1991). Is autism more common now than ten years ago? *The British Journal of Psychiatry*, 158(3), 403–409.
- Hofvander, B., Delorme, R., Chaste, P., et al. (2009). Psychiatric and psychosocial problems in adults with normal-intelligence autism spectrum disorders. *BMC Psychiatry*, 9(1), 1.
- Jamain, S., Quach, H., Betancur, C., et al. (2003). Mutations of the X-linked genes encoding neuroligins NLGN3 and NLGN4 are associated with autism. *Nature Genetics*, 34(1), 27–29.
- Lundström, S., Reichenberg, A., Anckarsäter, H., Lichtenstein, P., & Gillberg, C. (2015). Autism phenotype versus registered diagnosis in Swedish children: Prevalence trends over 10 years in general population samples. *BMJ*, 350, h1961.

CHRNA7 Deletions

► 15q13.3 Microdeletion Syndrome

Chromosomal Abnormalities

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Synonyms

Alterations in chromosome structure or number

Structure

Humans have 22 pairs of autosomes (nonsex chromosomes) and 1 pair of sex chromosomes (XX or XY). Genes are organized in a characteristic pattern on each chromosome. Any disruption of the total number of chromosomes, or the order or amount of genetic material on a given chromosome, is considered to be a chromosomal abnormality. Chromosomal abnormalities occur in about 1 in 150 live births and are the most common cause of intellectual disability and loss of a pregnancy (Jorde et al. 2010). An example of a syndrome caused by disruption of chromosome number is Down syndrome, the most common genetic cause of moderate intellectual disability, which is due to having three copies of chromosome 21, or trisomy 21 (Nussbaum et al. 2007).

In addition to the gain or loss of an entire chromosome, rearrangement of the order of genetic material on a chromosome, or the gain or loss of part of a chromosome, may result in a genetic disorder. Chromosomal abnormalities may be either *balanced* or *unbalanced*, depending on whether the particular alteration results in no net change in the total amount of genetic material, or a net change, respectively. For example, a *translocation* is a chromosomal rearrangement

that occurs when segments of nonhomologous chromosomes break off and are transferred from one chromosome to another. In a *reciprocal translocation*, there is an even exchange of genetic material between the two chromosomes. Other abnormalities include *deletions* and *duplications*, which result in the net loss or gain of genetic material, respectively, and *inversions*, which occur when two breaks occur on the same chromosome, and the piece that is cut out reinserts in the same location, but in the opposite direction (Jorde et al. 2010).

Such abnormalities of chromosome structure may be inherited, or can be new mutations, i.e., occur *de novo*, in the parent's germline. Such abnormalities tend to occur at regions with repetitive sequences of DNA and are due to errors in recombination between homologous chromosomes. Trisomies of the autosomes are most often due to errors in nondisjunction that occur during meiosis, the risk of which increases with maternal age. In general, because most genes in the human genome play a role in the development of the central nervous system, the larger the region of the chromosome that is disrupted, the more genes that are affected, and the greater likelihood that the chromosomal abnormality will result in a developmental disability (Jorde et al. 2010; Nussbaum et al. 2007).

Changes in chromosome number are clearly observable by karyotype. Similarly, large chromosomal abnormalities, such as duplications or deletions that cause the gain or loss of over a few million base pairs, can be detected by the banding pattern on a high-resolution karyotype (Nussbaum et al. 2007). However, the detection of smaller duplications or deletions was not possible until the development of more advanced techniques, including *fluorescence in situ hybridization (FISH)* and *comparative genomic hybridization (CGH)*. FISH utilizes probes that bind to specific regions of DNA to identify the precise location of a chromosomal break point and can be used to identify the genes that are disrupted in that region. CGH involves the binding, or hybridizing, of a patient's genome to a control genome, or in array-based CGH, which improves resolution, to a microarray that contains probes corresponding to

a control genome such that it is possible to detect net gains or losses of chromosomal regions. However, it is not possible to detect abnormalities such as balanced translocations using CGH, because there is no net change in the amount of genetic material in the patient's genome compared to the control genome (Jorde et al. 2010; Nussbaum et al. 2007).

These advances in molecular cytogenetics improved our ability to detect smaller abnormalities in chromosome structure, revealing regions of chromosomes that are associated with specific developmental syndromes, and in some cases, an increased risk of autism spectrum disorders (ASD) (see below). Therefore, investigations of abnormalities of chromosome structure in developmental syndromes have shaped the course and current approach to research in the genetics of ASD (Hoffman and State 2010). For example, Prader-Willi and Angelman syndromes are caused by a microdeletion (loss of less than five million base pairs of DNA) of chromosome 15q11-13. Inheritance of the microdeletion from the father results in Prader-Willi syndrome, while inheritance of a microdeletion in the same region of the maternal chromosome leads to Angelman syndrome, due to imprinting in this region. In addition, DiGeorge syndrome, also called velocardiofacial syndrome, which causes intellectual disability, and craniofacial and heart defects, is caused by a microdeletion of chromosome 22q11.2 (Nussbaum et al. 2007). Structural abnormalities of each of these chromosomal regions are associated with an increased risk of ASD (see below) (Hoffman and State 2010).

Function

The study of chromosomal abnormalities in ASD is particularly germane because individuals with idiopathic autism are more likely than unaffected individuals in the general population to have abnormalities of chromosome structure (O'Roak and State 2008). In particular, abnormalities in specific chromosome regions occur at a higher frequency in individuals with ASD. For example, 1–3% of affected individuals were found to have

maternally inherited duplications of chromosome 15q11-13 (Veenstra-VanderWeele and Cook 2004). Additional chromosomal regions that are more often disrupted by structural abnormalities in ASD include 16p11 and 22q11, both of which have been associated with a range of psychiatric disorders, consistent with the concept of pleiotropy, which is an emerging theme in the genetics of ASD (Hoffman and State 2010; State and Levitt 2011).

Chromosomal abnormalities that are identifiable by cytogenetics occur in an estimated 6–7% of individuals with ASD and in a higher percentage of individuals with ASD who have dysmorphic features and intellectual disability (Abrahams and Geschwind 2008). The likelihood of finding a chromosomal abnormality depends on the resolution of the cytogenetics technique used. For example, approximately 2–5% of individuals with ASD have a chromosomal abnormality identifiable by karyotype (Reddy 2005; Schaefer and Mendelsohn 2008; Shen et al. 2010). Studies have found that yield improves with increasing resolution of the clinical test such that clinical microarray has a higher rate than karyotype of detecting chromosomal abnormalities, though, as discussed, the limitation of this CGH-based test is that it cannot detect balanced translocations (Shen et al. 2010). In addition, the use of FISH for regions where structural abnormalities are more likely to occur in ASD is also likely to improve yield (Reddy 2005).

Recent guidelines recommend obtaining a karyotype as well as testing for fragile X syndrome (FXS) in the evaluation of all individuals with autism spectrum disorders (ASD) (Lintas and Persico 2009; Schaefer and Mendelsohn 2008). Tests for FXS in children undergoing a genetic evaluation for ASD are positive in up to 5% of cases (Reddy 2005; Schaefer and Mendelsohn 2008). Recent studies have suggested that higher resolution cytogenetics tests should also be included as a standard part of the evaluation of a child with ASD, given the increased yield of the higher resolution tests (Reddy 2005; Shen et al. 2010). One of the challenges of conducting these tests remains the limitation in our ability to interpret the findings, particularly given that structural

abnormalities may be inherited or *de novo*, and given the pleiotropy of some of the regions implicated in ASD.

As the technology used to detect chromosomal variation has improved in recent years, our ability to identify abnormalities in chromosomal structure in the research lab has advanced considerably such that it is now possible to detect accurately changes that are in the kilobase range. These submicroscopic gains or losses of genetic material are called *copy number variations* (CNVs). Since 2005, studies of CNVs have made a major contribution to our understanding of the genetic architecture of ASD. Sebat and colleagues were the first to identify an increased association of *de novo* CNVs in families with one affected child (simplex) compared to multiplex families, in which more than one child has autism (Sebat et al. 2007). This finding is consistent with the *rare variant hypothesis*, which predicts that individually rare, *de novo* variation accounts for the risk of autism in families where only one child is affected. At the same time, studies of CNVs in autism have shown that they have similar properties as other types of genetic variation. That is, CNVs can either be inherited or arise *de novo*, common or rare, and may or may not be associated with an increased risk of ASD (Hoffman and State 2010). Although CNVs that are associated with ASD are individually rare, e.g., occur in only 1–2% of individuals with ASD, as a group, these may occur in at least 10–20% of affected individuals (Abrahams and Geschwind 2008).

Pathophysiology

One approach of current research is to utilize the findings of rare chromosomal abnormalities as a route toward understanding the underlying pathophysiology of ASD. Therefore, known genetic syndromes that are associated with an increased risk of symptoms that are similar to those found in ASD may be instrumental in highlighting regions of chromosomes, and therefore, individual genes, that may predispose to ASD. Elucidating the functions of these *candidate genes* can lead us to a

better understanding of biological mechanisms that may be causative.

It is important to observe that while any of the chromosomal abnormalities discussed above, e.g., 15q11-13, are individually rare in idiopathic ASD, symptoms consistent with a diagnosis of ASD may occur at relatively high frequencies in individuals with known genetic syndromes. For example, more than 40% of children with Angelman syndrome and 25% of males with FXS have an ASD (Abrahams and Geschwind 2008). FXS is an important example of how the identification of a cytogenetic abnormality associated with an increased risk of ASD can advance our knowledge of pathophysiology. FXS is the most common inherited cause of intellectual disability (Cornish et al. 2008). The genetic etiology was first identified because the mutation produces a constriction in the X chromosome that is visible by light microscopy. Subsequent studies found that FXS is caused by disruption of a single gene, *fragile X mental retardation 1* (*FMRI*), by a trinucleotide repeat expansion, and that this gene produces a protein that regulates the transport and activation of other molecules that play important roles at the synapse of nerve cells. Further analysis of the function of *FMRI* revealed a potential mechanism for reversing the adverse effects of the mutation pharmacologically (Cornish et al. 2008).

Importantly, *FMRI* was identified as a candidate gene in ASD due to the increased risk of ASD in individuals with FXS, even though mutations in *FMRI* are individually rare in idiopathic autism. Therefore, one approach to the identification of novel candidate genes in ASD that is based on the rare variant hypothesis is to find rare cases of *de novo* chromosomal abnormalities in affected individuals and to identify precisely which genes are disrupted at the break points. This approach has led to crucial discoveries, including the identification of the following susceptibility genes: *contactin-associated protein-like 2* (*CNTNAP2*), *contactin 4* (*CNTN4*), *neuroligin 4X* (*NLGN4X*), and *SH3 and multiple ankyrin repeat domains 3* (*SHANK3*) (Bakkaoglu et al. 2008; Durand et al. 2007; Fernandez et al. 2004; Jamain et al. 2003). While disruptions of these genes are rare in idiopathic autism, their identification as candidates has been

important in elucidating the pathophysiology of ASD by illuminating potential biological mechanisms, such as synapse formation and function.

In recent studies, CNVs serve a similar function as large chromosomal disruptions, highlighting genes that are likely to play a role in ASD. However, one of the challenges that current researchers face is the high degree of structural variation that exists throughout the genomes of affected and unaffected individuals, which often confounds the interpretation of CNV studies. Nonetheless, it is clear that abnormalities in chromosome structure have the potential to offer insight into the genetic etiology of ASD. By following the leads from studies of chromosomal abnormalities, current research aims to illuminate common mechanisms involving candidate genes and, thereby, to identify new approaches for treatment.

See Also

- ▶ [Angelman/Prader-Willi Syndromes](#)
- ▶ [Common Disease-Rare Variant Hypothesis](#)
- ▶ [Course of Social Avoidance in Fragile X Syndrome, The](#)
- ▶ [Karyotype](#)
- ▶ [Pleiotropy](#)

References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, 9(5), 341–355.
- Bakkaloglu, B., O’Roak, B. J., Louvi, A., Gupta, A. R., Abelson, J. F., Morgan, T. M., et al. (2008). Molecular cytogenetic analysis and resequencing of contactin associated protein-like 2 in autism spectrum disorders. *American Journal of Human Genetics*, 82(1), 165–173.
- Cornish, K., Turk, J., & Hagerman, R. (2008). The fragile X continuum: New advances and perspectives. *Journal of Intellectual Disability Research*, 52(Pt 6), 469–482.
- Durand, C. M., Betancur, C., Boeckers, T. M., Bockmann, J., Chaste, P., Fauchereau, F., et al. (2007). Mutations in the gene encoding the synaptic scaffolding protein SHANK3 are associated with autism spectrum disorders. *Nature Genetics*, 39(1), 25–27.
- Fernandez, T., Morgan, T., Davis, N., Klin, A., Morris, A., Farhi, A., et al. (2004). Disruption of contactin 4 (CNTN4) results in developmental delay and other features of 3p deletion syndrome. *American Journal of Human Genetics*, 74(6), 1286–1293.
- Hoffman, E. J., & State, M. W. (2010). Progress in cytogenetics: Implications for child psychopathology. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(8), 736–751; quiz 856–737.
- Jamain, S., Quach, H., Betancur, C., Rastam, M., Colineaux, C., Gillberg, I. C., et al. (2003). Mutations of the X-linked genes encoding neuroligins NLGN3 and NLGN4 are associated with autism. *Nature Genetics*, 34(1), 27–29.
- Jorde, L. B., Carey, J. C., & Bamshad, M. J. (2010). *Jorde: Medical genetics* (4th ed.). Philadelphia: Mosby.
- Lintas, C., & Persico, A. M. (2009). Autistic phenotypes and genetic testing: State-of-the-art for the clinical geneticist. *Journal of Medical Genetics*, 46(1), 1–8.
- Nussbaum, R. L., McInnes, R. R., & Willard, H. F. (2007). *Nussbaum: Thompson & Thompson genetics in medicine* (7th ed.). Philadelphia: Saunders Elsevier.
- O’Roak, B. J., & State, M. W. (2008). Autism genetics: Strategies, challenges, and opportunities. *Autism Research*, 1(1), 4–17.
- Reddy, K. S. (2005). Cytogenetic abnormalities and fragile-X syndrome in autism spectrum disorder. *BMC Medical Genetics*, 6, 3.
- Schaefer, G. B., & Mendelsohn, N. J. (2008). Clinical genetics evaluation in identifying the etiology of autism spectrum disorders. *Genetics in Medicine*, 10(4), 301–305.
- Sebat, J., Lakshmi, B., Malhotra, D., Troge, J., Lese-Martin, C., Walsh, T., et al. (2007). Strong association of de novo copy number mutations with autism. *Science*, 316(5823), 445–449.
- Shen, Y., Dies, K. A., Holm, I. A., Bridgemohan, C., Sobeih, M. M., Caronna, E. B., et al. (2010). Clinical genetic testing for patients with autism spectrum disorders. *Pediatrics*, 125(4), e727–e735.
- State, M. W., & Levitt, P. (2011). The conundrums of understanding genetic risks for autism spectrum disorders. *Nature Neuroscience*, 14(12), 1499–1506.
- Veenstra-VanderWeele, J., & Cook, E. H., Jr. (2004). Molecular genetics of autism spectrum disorder. *Molecular Psychiatry*, 9(9), 819–832.

Chromosome 15q11–q13

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Synonyms

[Angelman/Prader-Willi locus](#)

Definition

This region on the long arm of chromosome 15 is adjacent to the centromere and is 14.6 million bases in size. It spans genomic coordinates 19,000,001–33,600,000 (GRCh37/hg19 assembly, UCSC Genome Browser). It contains numerous genes, some of which are subject to genomic imprinting, a phenomenon by which a gene(s) is silent on either the maternally or paternally transmitted chromosome. Mutations in this locus are responsible for Angelman and Prader-Willi Syndromes. Deficiency of the maternally expressed *UBE3A* gene causes Angelman Syndrome. While it has not been definitively determined which gene(s) in the interval cause PWS, deficiency of paternally expressed small nucleolar RNAs (snoRNAs) has been considered the leading suspect.

Other genes at this locus include those which encode subunits of the GABA receptors, *GABRB3*, *GABRA5*, and *GABRG3*. *CYFIP1* encodes a protein, which interacts with the Fragile X mental retardation protein, FMRP (Schenck et al. 2001). *SNRPN* encodes a small nuclear ribonucleoprotein that is involved in mRNA processing. The upstream untranslated region (5'UTR) of this gene is an imprinting center, which helps to determine the parental imprint of the chromosome (Saitoh et al. 1996).

The entire region is prone to complex rearrangements in chromosomal structure and has been implicated in Autism Spectrum Disorders (ASDs) as well as in schizophrenia, epilepsy, and intellectual disability. Within the interval, there are a number of specific points that are particularly likely to serve as the boundaries for changes in chromosomal structure. These are called breakpoints and have been numbered BP (breakpoint) 1–BP5. Duplications of the maternal chromosome extending from either the centromere or BP1 to BP5 are among the most common changes in chromosomal structure identified in ASD (Sanders et al. 2011). A high proportion of patients with duplications at this locus meet diagnostic criteria for ASD (Abrahams and Geschwind 2008). Conversely, in some clinical

ASD cohorts, up to 1% of patients show maternal duplications of this interval (Sanders et al. 2011). In addition, deletions of the BP1–BP2 region and BP4–BP5 regions have been associated with schizophrenia, deletions of BP4–BP5 have been implicated in epilepsy and intellectual disability, and duplications within the BP4–BP5 interval have been identified in patients with ASD and intellectual disability and are also not infrequently seen in unaffected individuals (Sanders et al. 2011).

See Also

- [Angelman/Prader-Willi Syndromes](#)
- [Copy Number Variation](#)

References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, 9, 341–355.
- Saitoh, S., Buiting, K., Rogan, P. K., Buxton, J. L., Driscoll, D. J., Arneemann, J., et al. (1996). Minimal definition of the imprinting center and fixation of chromosome 15q11-q13 epigenotype by imprinting mutations. *Proceedings of the National Academy of Sciences of the United States of America*, 93, 7811–7815.
- Sanders, S., Ercan-Sencicek, A. G., Hus, V., Luo, R., Murtha, M. T., Moreno-De-Luca, D., et al. (2011). Multiple recurrent de novo CNVs, including duplications of 7q11.23 Williams syndrome region, are strongly associated with autism. *Neuron*, 70, 863–885.
- Schenck, A., Bardoni, B., Moro, A., Bagni, C., & Mandel, J. L. (2001). A highly conserved protein family interacting with the fragile X mental retardation protein (FMRP) and displaying selective interactions with FMRP-related proteins FXR1P and FXR2P. *Proceedings of the National Academy of Sciences of the United States of America*, 98, 8844–8849.
- UCSC Genome Browser, GRCh37/hg19 (February 2009) Assembly. <http://genome.ucsc.edu/>. Retrieved 27 Apr 2012.

Chronic Dyskinesia

- [Tardive Dyskinesia](#)

Chronic Hairpulling

- ▶ [Trichotillomania](#)

Chronological Age Appropriateness

- ▶ [Developmentally Appropriate Practice \(DAP\)](#)

CII

- ▶ [Communication Intention Inventory](#)

Cingulate Cortex

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Definition

The cingulate cortex is a brain region located in the medial portion of the cortex, just above the corpus callosum, continuing through the cingulate sulcus. Traditionally, the cingulate cortex is divided into an anterior portion or anterior cingulate and a posterior portion or posterior cingulate. Broadly, the anterior cingulate is involved in self-regulatory and executive control functions, social and emotional processing, and respiratory control. The posterior cingulate is thought to be involved in supporting internally directed thought. As a key node of the default mode network, the posterior cingulate cortex is thought to play a role in modulating the dynamic interplay between the default mode network and attention networks providing for efficient allocation of attention.

See Also

- ▶ [ERN](#)
- ▶ [Error-Related Negativity](#)
- ▶ [Feedback-Related Negativity](#)

References and Reading

Vogt, B. A. (Ed.). (2009). *Cingulate neurobiology and disease*. Oxford: Oxford University Press.

Cingulum

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Synonyms

[Cingulum bundle](#)

Definition

The cingulum, also known as the cingulum bundle, is a fiber tract in the brain that connects the cingulate gyrus, found in the midline of the brain above the corpus callosum, to the entorhinal cortex, found in the base of the anterior temporal lobe. The tract lies between the cingulate gyrus and the corpus callosum, curving around the back of the callosum and extending into the anterior temporal lobe. The brain structures that the cingulum connects both belong to the limbic system, which is involved in the experience and regulation of emotional states as well as memory. The cingulate gyrus has many functions including helping to selectively attend to relevant stimuli in the environment. Thus, the cingulum may send attended signals from the cingulate gyrus into a pathway that allows salient information to become encoded into long-term memories. Imaging

research using diffusion tensor imaging consistently finds reduced fractional anisotropy, a measure of white matter integrity, in the cingulum bundle among individuals with autism, and in many studies, this abnormality correlated with the degree of social impairment. In individuals without autism but with brain lesions affecting the cingulum, the degree of disruption of this pathway related to mentalizing impairment. This suggests that the cingulum bundle plays an important role in aspects of social cognition that are impaired in autism, and that differences in the development of this pathway may underlie some features of ASD.

See Also

- [Cingulate Cortex](#)
- [Limbic System](#)

References and Reading

- Ameis, S. H., Fan, J., Rockel, C., Soorya, L., Wang, A. T., & Anagnostou, E. (2013). Altered cingulum bundle microstructure in autism spectrum disorder. *Acta Neuropsychiatry*, 25(5), 275–282.
- Catani, M., Dell’Acqua, F., Budisavljevic, S., Howells, H., Thiebaut de Schotten, M., Froudist-Walsh, S., D’Anna, L., Thompson, A., Sandrone, S., Bullmore, E. T., Suckling, J., Baron-Cohen, S., Lombardo, M. V., Wheelwright, S. J., Chakrabarti, B., Lai, M. C., Ruigrok, A. N., Leemans, A., Ecker, C., Consortium, M. A., Craig, M. C., & Murphy, D. G. (2016). Frontal networks in adults with autism spectrum disorder. *Brain*, 139(2), 616–630.
- Chiang, H. L., Chen, Y. J., Lin, H. Y., Tseng, W. I., & Gau, S. S. (2017). Disorder-specific alteration in white matter structural property in adults with autism spectrum disorder relative to adults with ADHD and adult controls. *Human Brain Mapping*, 38(1), 384–395.
- Herbet, G., Lafargue, G., Bonnetblanc, F., Moritz-Gasser, S., Menjot de Champfleury, N., & Duffau, H. (2014). Inferring a dual-stream model of mentalizing from associative white matter fibres disconnection. *Brain*, 137(3), 944–959.
- Ikuta, T., Shafritz, K. M., Bregman, J., Peters, B. D., Gruner, P., Malhotra, A. K., & Szeszko, P. R. (2014). Abnormal cingulum bundle development in autism: A probabilistic tractography study. *Psychiatry Research*, 221(1), 63–68.
- Nezamzadeh, M., Wedeen, V. J., Wang, R., Zhang, Y., Zhan, W., Young, K., Meyerhoff, D. J., Weiner, M. W., & Schuff, N. (2010). In-vivo investigation of the human cingulum bundle using the optimization of MR diffusion spectrum imaging. *European Journal of Radiology*, 75(1), e29–e36.
- Pugliese, L., Catani, M., Ameis, S., Dell’Acqua, F., Thiebaut de Schotten, M., Murphy, C., Robertson, D., Deeley, Q., Daly, E., & Murphy, D. G. (2009). The anatomy of extended limbic pathways in Asperger syndrome: A preliminary diffusion tensor imaging tractography study. *NeuroImage*, 47(2), 427–434.
- van den Heuvel, M., Mandl, R., Luigjes, J., & Hulshoff Pol, H. (2008). Microstructural organization of the cingulum tract and the level of default mode functional connectivity. *Journal of Neurosciences*, 28(43), 10844–10851.

Cingulum Bundle

- [Cingulum](#)

Ciprallex (Canada)

- [Escitalopram](#)

Circle of Friends

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Definition

Circle of friends is a conceptual framework that has been used to develop interventions to promote inclusion of individuals with disabilities in mainstream settings through relationship development with peers, especially individuals at particular risk of rejection or social isolation. A more specific definition refers to “circle of friends” as a group of people who gather around a person who has become excluded or isolated (Falvey et al. 1997).

Historical Background

The circle of friends intervention was initially developed in Canada to facilitate the deinstitutionalization of people with disabilities transitioning to their local communities. Circle of friends was subsequently applied to supporting the inclusion of pupils with special needs in their local mainstream schools (Forest and Lusthaus 1989). Circle of friends also was adapted to support children experiencing emotional, behavioral, and social difficulties in schools (Newton et al. 1996; Pearpoint et al. 1996). The basic implementation of circle of friends involved enlisting the help of classmates through conducting a whole-class meeting and then setting up a voluntary, special group or a “circle of friends.” This circle of friends group helps to set, monitor, and review weekly targets in a meeting facilitated by an adult. Parents also have been encouraged to establish circle of friends (Turnbull et al. 1999). Other social skills interventions that have involved peer-mediated interventions, such as peer buddies (e.g., English et al. 1997) or peer support networks (e.g., Haring and Breen 1992), have sometimes been referred to as consistent with the circle of friends approach.

Rationale or Underlying Theory

The original conceptualization of circle of friends was graphically represented by a set of concentric circles. The center of the circle represents the individual with a disability who is the focus of intervention. The second circle is called the circle of intimacy and includes people who are called “anchors.” This circle represents the close relationships that one cannot live without. The third circle is called the circle of friendship and includes people who are called “allies.” The circle represents friends and close relatives. They are people one can count on in difficult times and who one can confide in. If friendship relationships are sparse, one is prone to isolation, and this may result in anger and depression. The fourth circle is the circle of participation and includes people who are called “associates.” Associates are people with whom one may interact in the community, in

school, in houses of worship, etc. The fifth circle is the circle of exchange and includes people who are “paid.” This would encompass interactions with teachers, aides, medical providers, therapists, hairdressers, etc. People with disabilities may interact with more than the usual number of paid members. These individuals appear to set the agenda for the person with disabilities through scheduled appointments, policy requirements, and their limited availability.

These concentric circles are sometimes referred to as circles of support. They can change over time, especially at life transitions that may change the cast of people in one’s life. This model recognizes the stability of the inner circle of intimacy, but points out that other relationships are important to develop human potential and experience. In particular, the circle of friends intervention emphasizes the need to grow the circle of friendship to maximize inclusion of the person with disabilities in mainstream society and to minimize the likelihood of isolation from or within natural community settings. Building the circle of friendship changes the people who may be moving from other circles, e.g., “associates,” as well as the person with the disability who is at the center of the circle.

As described below, the people in the circle of friendship are provided information to deepen their appreciation of the person at the center of the circle, the characteristics of their disorder, as well as their individual strengths, interests, and desires. They are called upon to identify targets that will enhance the social inclusion of the person, to assist with the learning process, and to track progress. Ultimately, the persons in the peer group are expected to develop authentic friendships characterized by supportive, reciprocal relationships that will expand social inclusion as the person with disabilities learns to adapt successfully to a growing set of social circumstances.

Goals and Objectives

The goal of circle of friends programs is to promote social inclusion of individuals at risk of

rejection or isolation from the community. Circle of friends programs could promote increased support within the different circles or levels, such as extended family, friends, neighbors, and faith communities, but the main objective is to increase the circle of friendship.

Treatment Participants

Circle of friends has been applied to a variety of populations of individuals of all ages with disabilities. Applicability to individuals with autism is readily apparent, as social and communication skills tend to be an area of weakness that typically must be addressed to promote social inclusion of individuals with autism. Peer approaches to promoting social inclusion are prevalent in preschool settings, but have been applied to school age and older individuals as well. Transitions to different school, vocational, and residential settings are each likely to require a reevaluation of one's circle of friends and the need for additional efforts to provide supportive social partners at various levels.

Treatment Procedures

Interventions deemed most consistent with the circle of friends framework are likely to begin with filling in names in the concentric circles of anchors, allies, associates, and paid members. The interventionist uses this information to illustrate the importance of peer friendships, rather than relationships with mainly paid adults. The intervention program usually provides new and accurate information about the nature of the disability, such as the characteristics of autism, and the nature of increased support among allies and associates in particular. Peers are called upon to identify positive features or assets of the individual who is the focus of intervention. As peers discuss strengths, interests, preferences, and desires of their peer with autism, they are guided to recognize that everybody has their own special abilities and areas of needs. The overlap between features of autism and the areas that they may find difficult

when interacting with their peer with autism are highlighted. The peers are encouraged to discuss their own strengths and weaknesses as well. This process is meant to increase social acceptance as well as identifying unique needs or skills that the peers might help the individual with disabilities master to increase acceptance. Weaknesses are discussed as "things one is still learning to do." Thus, the peers are given primary responsibility for helping identify social targets and identifying ways to encourage learning on the part of the person with a disability. They also may be asked to track progress and to be sensitive to new skills that might be needed to adapt to a growing array of social situations. An adult facilitator typically meets with a group of peers and the person with the disability on a regular basis to help sustain the effort and ensure that interactions are supportive, encouraging, and acceptable to all involved.

A number of peer buddy and peer network interventions have been considered exemplars of circle of friends programs, although they have not been developed from the circle of friends conceptual framework.

Efficacy Information

Few evaluations of circle of friends programs have been conducted. Many of those evaluations have reported encouraging results using qualitative case study methodologies or have been largely descriptive in nature (Barrett and Randall 2004; Calabrese et al. 2008; Gus 2000; Newton et al. 1996; Whitaker et al. 1998). A lack of a standard treatment protocol hinders replication and makes evaluations of circle of friends difficult.

Small-scale group design studies have shown improvements in social skills compared to comparison groups (Kalyva and Avramidis 2005). On the other hand, Frederickson and colleagues (Frederickson et al. 2005) found that improved social acceptance on the part of classmates tended to diminish over time and there were no discernable long-term effects seen in the social skills of primary grade children with autism. Owen-DeSchryver et al. (2008) employed peer training

based at least in part on circle of friends and showed, in a multiple baseline design across peer groups, that peers' initiations increased after training and a corresponding increase in initiations and responses was seen in children with ASD. An examination of the single-subject graphs reveals a strong correspondence between peer behavior and the corresponding social behavior of the children with autism. Long-term maintenance and generalization of effects were not evaluated, however. Miller et al. (2003) also used a multiple baseline design across peers and found improved social skills during lunchtime following friendship circle training. They also found impressive maintenance and generalization to recess and other activities for two of the three participants.

Peer-mediated interventions have repeatedly been shown to have robust effects on improving the social behavior of young children with autism (McConnell 2002; Rogers 2000). However, there are few studies that evaluate maintenance of effects and the extent to which peer relationship development results. Perhaps more consistent with the circle of friends framework is the promising research on peer support networks that have been shown to be efficacious in promoting pro-social behavior in youth with ASD (e.g., Haring and Breen 1992; Harrell et al. 1997; Hughes et al. 1999).

Outcome Measurement

One measure of the effects of circle of friends programs is an assessment of the number of people who are identified and who identify themselves within the circles of friendship. In addition, sociometric ratings can be used to determine whether the individual has an elevated social status within a classroom or another social network. Social network analyses also could be used to determine whether individuals with disabilities move from the periphery to more central roles with more reciprocal friendship nominations.

Observational data collection systems typically monitor the rate of social initiations and responses. Alternatively, they could monitor

more specific, targeted behaviors. For example, subsequent to circle of friends training, annoying or disruptive behaviors might be expected to occur less often and appropriate topic shifts, sharing, complimenting, or other positive social behaviors being learned might be expected to occur more frequently.

Social skills rating scales also are available that can be administered to teachers, parents, or peer groups.

Qualifications of Treatment Providers

Circle of friends programs have been implemented by a variety of professionals, including teachers, special educators, counselors, speech-language pathologists, as well as parents.

See Also

- [Inclusion](#)
- [Peer-Mediated Intervention](#)
- [Social Interventions](#)
- [Social Skill Interventions](#)

References and Reading

- Barrett, W., & Randall, L. (2004). Investigating the circle of friends approach: Adaptations and implications for practice. *Educational Psychology in Practice*, 20, 353–368.
- Calabrese, R., Patterson, J., Lieu, F., Goodvin, S., Hummel, C., & Nance, E. (2008). An appreciative inquiry into the circle of friends program: The benefits of social inclusion of students with disabilities. *International Journal of Whole Schooling*, 4(2), 20–49.
- English, K., Goldstein, H., Shafer, K., & Kaczmarek, L. (1997). Promoting interactions among preschoolers with and without disabilities: Effects of a buddy skills-training program. *Exceptional Children*, 63, 229–243.
- Falvey, M., Forest, M., Pearpoint, J., & Rosenberg, R. (1997). *All my life's a circle*. Toronto: Inclusion Press.
- Forest, M., & Lusthaus, E. (1989). Promoting educational equality for all students. Circles and maps. In S. Stainback, W. Stainback, & M. Forest (Eds.), *Educating all students in the mainstream of regular education* (pp. 43–57). Baltimore: Paul H. Brookes.

- Frederickson, N., Warren, L., & Turner, J. (2005). "Circle of friends"-An exploration of impact over time. *Educational Psychology in Practice*, 21, 197–217.
- Gus, L. (2000). Autism: Promoting peer understanding. *Educational Psychology in Practice*, 16(3), 461–468.
- Haring, T. G., & Breen, C. G. (1992). A peer-mediated social network intervention to enhance social integration of persons with moderate and severe disabilities. *Journal of Applied Behavior Analysis*, 25, 319–333.
- Harrell, L. G., Kamps, D., & Kravits, T. (1997). The effects of peer networks on social-communicative behaviors for students with autism. *Focus on Autism and other Developmental Disabilities*, 12, 241–256.
- Hughes, C., Guth, C., Hall, S., Presley, J., Dye, M., & Byers, C. (1999). They are my best friends. Peer buddies promote inclusion in high school. *Teaching Exceptional Children*, 31, 32–37.
- Kalyva, E., & Avramidis, E. (2005). Improving communication between children with autism and their peers through the "circle of friends": A small-scale intervention study. *Journal of Applied Research in Intellectual Disabilities*, 18, 253–261.
- McConnell, S. R. (2002). Interventions to facilitate social interaction for young children with autism: Review of available research and recommendations for educational intervention and future research. *Journal of Autism and Developmental Disorders*, 32(5), 351–372.
- Miller, M., Cooke, N., Test, D., & White, R. (2003). Effects of friendship circles on the social interactions of elementary age students with mild disabilities. *Journal of Behavioral Education*, 12(3), 167–184.
- Newton, C., Taylor, G., & Wilson, D. (1996). Circles of friends: An inclusive approach to meeting emotional and behavioral needs. *Educational Psychology in Practice*, 11(4), 41–48.
- Owen-DeSchryver, J. S., Carr, E. G., Cale, S. I., & Blakely-Smith, A. (2008). Promoting social interactions between students with autism spectrum disorders and their peers in inclusive school settings. *Focus on Autism and Other Developmental Disabilities*, 23(1), 15–28.
- Pearpoint, J., Forest, M., & O'Brien, J. (1996). MAPS, circles of friends and PATH. Powerful tools to help build caring communities. In S. Stainback & W. Stainback (Eds.), *Inclusion: A guide for educators* (pp. 67–86). Baltimore: Paul H. Brookes.
- Rogers, S. J. (2000). Interventions that facilitate socialization in children with autism. *Journal of Autism and Developmental Disorders*, 30, 399–409.
- Turnbull, A., Pereira, L., & Blue-Banning, M. (1999). Parents' facilitation of friendships between their children with a disability and friends without a disability. *Research and Practice for Persons with Severe Disabilities*, 24(2), 85–99.
- Whitaker, P., Barratt, P., Joy, H., Potter, M., & Thomas, G. (1998). Children with autism and peer group support: Using "circles of friends". *British Journal of Special Education*, 25(2), 60–64.

Circumstantial Thinking

► Circumstantiality

Circumstantiality

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Synonyms

[Circumstantial thinking](#)

Definition

A pattern of speech characterized by provision of a mix of relevant and irrelevant information seen in some psychiatric disorders. Typically, although some aspects of narrative are present, the inclusion of extraneous detail and only minimally relevant (or irrelevant) information makes it difficult to follow the speaker's thought. Often individuals need to be reminded of the topic or question because they lose track of the topic. Typically the patient has difficulty in separating relevant from irrelevant information while describing an event. The patient often includes all details and presents them in a sequential order, with the result that the main thread of thought becomes lost as one association leads to another. Frequently the person may need to have questions repeated because the main point of answers has become lost in the confusion of unnecessary detail. Circumstantial thinking/speech is most commonly seen in schizophrenia and obsessive compulsive disorder. It can also be observed in some neurological syndromes (including epilepsy syndromes) at the interface of neurology and psychiatry. Individuals with Asperger's disorder may have difficulties with monitoring conversational cues and often provide tremendous detail

about topics of special interest but usually remain highly focused on their topic.

See Also

- [Asperger's Disorder](#)
- [Obsessive-Compulsive Disorder \(OCD\)](#)
- [Schizophrenia](#)

References and Reading

- Benson, D. F. (1991). The Geschwind syndrome. *Advances in Neurology*, 55, 411–421.
- Hoepfner, J. B., Garron, D. C., et al. (1987). Epilepsy and verbosity. *Epilepsia*, 28(1), 35–40.
- Koyama, T., & Kurita, H. (2008). Cognitive profile difference between normally intelligent children with Asperger's disorder and those with pervasive developmental disorder not otherwise specified. *Psychiatry & Clinical Neurosciences*, 62(6), 691–696.
- North, C. S., Kienstra, D. M., et al. (2006). Interrater reliability and coding guide for nonpsychotic formal thought disorder. *Perceptual & Motor Skills*, 103(2), 395–411.

cis-N,N-Dimethyl-9-[3-(4-methyl-1-piperazinyl)-propylidene] thioxanthene-2-sulfonamide

- [Thiothixene](#)

Citalopram

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Synonyms

[Escitalopram](#)

Definition

Citalopram is a selective serotonin reuptake inhibitor (SSRI) used to treat depression and anxiety disorders. A large-scale, multisite trial of citalopram in 149 children with autism spectrum disorders showed that it was no better than placebo for reducing repetitive behavior. Citalopram is chemically related to the newer SSRI, escitalopram, which is a single chemical isomer of the molecule. By contrast, citalopram is a so-called racemic mixture, which means that there are two isomers in citalopram.

See Also

- [Anxiety Disorders](#)
- [Depressive Disorder](#)
- [Escitalopram](#)

References and Reading

- King, B. H., Hollander, E., Sikich, L., McCracken, J. T., Scahill, L., Bregman, J. D., et al. (2009). Lack of efficacy of citalopram in children with autism spectrum disorders and high levels of repetitive behavior. Citalopram ineffective in children with autism. *Archives of General Psychiatry*, 66(6), 583–590.

Citrate and Autism

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Synonyms

[Citric acid](#)

Definition

Cytoplasmic citrate is an important metabolite at the junction of many important metabolic

pathways, including the tricarboxylic acid (TCA) cycle and the generation of NADH and FADH₂ (Bhutia et al. 2017). The TCA cycle generates citrate within the mitochondrial matrix via citrate synthase (Bhutia et al. 2017). In a well-fed state, citrate is transferred either across the inner mitochondrial membrane through SLC25A1 or from the plasma to the cytoplasm via SLC13A5 (Bhutia et al. 2017). In the cytoplasm, citrate has multiple biological functions related to metabolic regulation and fatty acid and cholesterol synthesis (Bhutia et al. 2017). Increased cytoplasmic citrate inhibits glycolysis by inhibiting the rate-limiting enzyme phosphofructokinase-1 and stimulates gluconeogenesis by activating fructose-1,6-bisphosphatase. Known as SLC13A5 deficiency, patients present with early-onset epilepsy within the first few weeks after birth and persist during childhood. In addition, these patients exhibit developmental delay, slow progression of motor function, and significant impairment in language and speech development, which does not respond to ketogenic diet (Bhutia et al. 2017). Interestingly, these patients also present with defects in teeth development, identified as microdontia (Bhutia et al. 2017). The early onset and severity of epilepsy in SLC13A5-deficiency suggest citrate might serve as an important energy source for neurons. A loss of function of SLC13A5 may increase seizure susceptibility and delay brain development in SLC13A5 patients through an increased energy deficit.

Similar to SLC13A5 deficiency, it is possible that part of the neurological and social deficits observed in autism spectrum disorder (ASD) patients may result from energy deficits within the brain. A recent study examining the bioenergetics of ASD patients found a correlation with poor social function and behavior with increased citrate synthase activity (Delhey et al. 2017). It is believed that dysregulation of mitochondrial metabolism leads to neurodegeneration, oxidative stress, and neuroinflammation, which can drive microglial function and cell loss (Delhey et al. 2017). However, a randomized case-control study of ASD patients from rectal and cecum

biopsies showed ASD patients had a higher complex I activity, while no change was detected in the complex IV or citrate synthase activity (Rose et al. 2017). The results suggest multiple ETC complexes are upregulated in ASD patients (Rose et al. 2017). However, a preliminary communication showed that ASD children had a lower complex I activity although the sample size for the study was small (Giulivi et al. 2010). Overall, the symptomology of ASD seems to be directly affected by the bioenergetics of the brain and activity of mitochondria.

See Also

- Autonomic Nervous System
- Inferior Parietal Area

References and Reading

- Bhutia, Y. D., Kopel, J. J., Lawrence, J. J., Neugebauer, V., & Ganapathy, V. (2017). Plasma membrane Na⁺-coupled citrate transporter (SLC13A5) and neonatal epileptic encephalopathy. *Molecules*, 22(3), 378. Retrieved from <http://www.mdpi.com/1420-3049/22/3/378>.
- Delhey, L., Kilinc, E. N., Yin, L., Slattery, J., Tippet, M., Wynne, R., ... Frye, R. E. (2017). Bioenergetic variation is related to autism symptomatology. *Metabolic Brain Disease*, 32(6), 2021–2031. <https://doi.org/10.1007/s11011-017-0087-0>
- Giulivi, C., Zhang, Y.-F., Omanska-Klusek, A., Ross-Inta, C., Wong, S., Hertz-Picciotto, I., ... Pessah, I. N. (2010). Mitochondrial dysfunction in autism. *JAMA*, 304(21), 2389. <https://doi.org/10.1001/jama.2010.1706>
- Rose, S., Bennuri, S. C., Murray, K. F., Buie, T., Winter, H., & Frye, R. E. (2017). Mitochondrial dysfunction in the gastrointestinal mucosa of children with autism: A blinded case-control study. *PLoS One*, 12(10), e0186377. <https://doi.org/10.1371/journal.pone.0186377>.

Citric Acid

- Citrate and Autism

Civil Rights Act of 1964

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Definition

The Civil Rights Act of 1964 (the “Act”) is a landmark United States federal law that established several new categories of legal protections for individual civil rights (Pub. L. No 88-352). The Act confirms that it is illegal under the US Constitution for private individuals, businesses, and government agencies to discriminate against people on the basis of their race, skin color, religion, sex, or national origin.

The Act requires fair and equitable treatment in employment, voting, and the distribution of government benefits. Additionally, the Act mandates that all people on the US land have equal access to public facilities and places of public accommodation, including hospitals, clinics, schools, and courthouses, among others. Since 1964, this pivotal Act has served as the legal basis for several other equal opportunity and employment discrimination laws (Sandoval-Strausz 2005). Lawmakers have used the Act’s provisions to create protections for the equitable provision of healthcare services as well as protections for workers with disabilities.

Principles of the Civil Rights Act of 1964

The Civil Rights Act of 1964 strengthened the enforcement of anti-segregation laws. It protects against discriminatory acts that fall within one of five protected categories: race, skin color, religion, sex, and national origin. The Act contains 11 segments called Titles. Certain titles have generated a number of important cases in US federal and state courts. These landmark provisions

prohibit discrimination in voting (Title I), public accommodations (Title II), federal funding and benefits (Title VI), and employment (Title VII). Though these key titles are usually referred to with Roman numerals, they are also sometimes written as Sections One, Two, Six, and Seven, respectively.

Title I bars unequal state voter registration requirements for federal elections. Title II bans racial segregation and other discriminatory acts in public places, including courthouses, parks, restaurants, theaters, sports arenas, hotels, and hospitals (U.S. Department of the Interior 2016). The Act’s Title VI prohibits the federal and state governments from discrimination in providing benefits and services. Title VI impacts any program or activity that uses federal funding (U.S. Department of Health and Human Services 2014).

Today, Title VII of the Act is commonly cited in employee-employer human relations lawsuits and administrative court filings across the country. Title VII prohibits harassment and unequal treatment on the basis of race, color, religion, sex, or national origin in all areas of employment. This covers hiring advertisements, on-the-job tasks, terminations, and retirement matters. The Act prohibits retaliation whenever an individual communicates a discrimination concern, files a discrimination charge, or participates in an employment discrimination investigation or lawsuit against an employer (U.S. Department of Labor 2020). Under Title VII, employers must reasonably accommodate the sincerely held religious practices of all job applicants and employees. An employer’s accommodation is deemed as unreasonable – and, therefore, not legally required – if the accommodation would impose an undue hardship on the operation of the employer’s business. US judicial opinions and administrative laws contain examples of situations with “undue hardships” and types “reasonable accommodations” that are recognized under the Civil Rights Act of 1964.

Over the years, judicial court opinions from the US Supreme Court and case decisions from other lower courts have established guidance

for interpreting the Act's various provisions (often called "legal precedent"). Federal and state administrative rules also provide legal insight about the Act's requirements in specific circumstances. This information is available online in the form of agency regulations, registers, manuals, and memoranda (e.g., U.S. Department of Justice Civil Rights Division Federal Coordination and Compliance Section 2016; U.S. Department of Justice Civil Rights Division 2019).

The Civil Rights Act of 1964 is one of several other civil rights statutes within the U.S. Code. Before 1964, the U.S. Congress passed six other laws that used the title "Civil Rights Act." These laws are the Civil Rights Act of 1866, Civil Rights Act of 1870, Civil Rights Act of 1871, Civil Rights Act of 1875, Civil Rights Act of 1957, and Civil Rights Act of 1960 (U.S. Library of Congress 2020). The earliest statutes contained anti-discrimination requirements; however, several protections were ultimately erased by unsound Supreme Court decisions in the late nineteenth century. In a collection of five cases in 1883, the Supreme Court claimed that Congress did not have the Constitutional power to create legislation that prevented racial discrimination by private individuals (*Civil Rights Cases*, 109 U.S. 3 (1883)). Modern courts have since invalidated these rulings and confirmed that the Supreme Court's interpretations of the Constitution in 1883 were legally incorrect. The Civil Rights Act of 1964 both reinserted and strengthened discrimination protections within the US Code.

After 1964, many pivotal laws were modeled after the Civil Rights Act of 1964, including the Americans with Disabilities Act (ADA) (American Bar Association 2004). As a general rule, all civil rights statutes seek to confirm that all people on US land – citizens, residents, and visitors – have fundamental protections against discrimination that are derived from the U.S. Constitution (U.S. Department of Health and Human Services Office for Civil Rights 2020).

Healthcare Discrimination and the Civil Rights Act of 1964

This ground-breaking Act was instrumental in increasing racial equality and fair government funding in health care services settings. As Longest (2016) explains, the Civil Rights Act of 1964 overturned the discriminatory provisions of the Hill-Burton Act of 1946 (more formally known as the Hospital Survey and Construction Act, Pub. L. No. 725). The Hill-Burton Act allocates government funds for the construction and improvement of hospitals across the United States. Before the Civil Rights Act of 1964, the Hill-Burton Act embraced discriminatory "Separate but Equal" Doctrine, which made it legal for patients to be treated in segregated facilities. The Hill-Burton Act incorrectly assumed that patients could receive equal treatment in divided locations. The Civil Rights Act of 1964 removed the "Separate but Equal" provisions in the Hill-Burton Act (U.S. Commission on Civil Rights 1965).

In passing the Civil Rights Act of 1964, the US Congress confirmed that quality of healthcare, available goods, and government funds distributions can never be truly equitable across segregated locations (Hahn et al. 2018). The Civil Rights Act of 1964, in conjunction with seminal Supreme Court rulings like *Brown v. Board of Education*, confirmed that "Separate but Equal" Doctrine violates the law of the US Constitution (347 U.S. 483 (1954)).

Increased Recognition of Discrimination Issues as Social Determinants of Health

Individuals with autism spectrum disorder (ASD) and their caregivers should be knowledgeable about the anti-discrimination protections that are afforded by the Civil Rights Act of 1964 and its legal precedent. It is well-documented that individuals with ASD and

other disabilities are especially vulnerable to experiences of discrimination (e.g., Trump and Ayres 2020; U.S. Department of Health and Human Services Office of Disease Prevention and Health Promotion 2020a). To be actionable under the Civil Rights Act of 1964, a discriminatory action must relate to at least one of the Act's five protected categories.

The US government agencies and the scientific community have increasingly recognized a correlation between discrimination and negative health outcomes (Rosenbaum et al. 2000). At a high level, discrimination is understood as a social stressor that has a physiological effect on individuals (e.g., irregular heartbeat, anxiety, heartburn). Compounded over time, instances of discrimination can lead to long-term negative health outcomes (Pascoe and Richman 2009). The U.S. Department of Health & Human Services (DHHS) classifies discrimination as one of the "Social Determinants" of people's health (U.S. DHHS Centers for Disease Control and Prevention 2019). Social Determinants of Health (sometimes abbreviated as "SDOH") are "conditions in the environments in which people are born, live, learn, work, play, worship, and age that affect a wide range of health, functioning, and quality-of-life outcomes and risks" (U.S. DHHS Office of Disease Prevention and Health Promotion 2020b). SDOH conditions can forecast increased risks of health challenges. Guidance from the DHHS states,

Discrimination can be attributed to social interactions that occur to protect more powerful and privileged groups at the detriment of other groups. While not all stressful experiences negatively affect health, or occur because of discrimination, many do impact health and can be related to discrimination. (U.S. DHHS Office of Disease Prevention and Health Promotion 2020a)

A growing body of scholarly literature emphasizes that individual and structural discrimination can cause intentional or unintentional harm and negative health consequences, regardless of whether it is perceived by an individual or group (e.g., Krahn et al. 2015; Kirschner et al. 2007; Lewis et al. 2009; Major et al. 2018).

History

The Act's provisions were heavily debated in the 88th US Congress (U.S. Government Publishing Office 2020). The House of Representatives approved the Act on February 10, 1964. On June 19, 1964, it was approved in the Senate. President Lyndon B. Johnson signed the Act into law on July 2, 1964.

See Also

- [Americans with Disabilities Act](#)
- [Autism Collaboration, Accountability, Research, Education, and Support \(CARES\) Act of 2019 \(Also Referred to as the "Autism CARES Act of 2019"\)](#)
- [Social Security Amendments of 1965 \(or "Medicare Act of 1965" and/or the "Medicaid Act of 1965"\)](#)

References and Reading

- American Bar Association Section of Civil Rights and Social Justice. (2004). The civil rights act of 1964: Precursors and progeny. *Human Rights Magazine*, 31(3). Retrieved from https://www.americanbar.org/groups/crsj/publications/human_rights_magazine_home/human_rights_vol31_2004/summer2004/irr_hr_summer04_progeny/
- Civil Rights Act of 1964, Pub. L. No 88-352, 78 Stat. 241, codified as amended at 42 U.S.C. § 2000e *et seq.* (1964). Retrieved from <https://www.govinfo.gov/content/pkg/STATUTE-78/pdf/STATUTE-78-Pg241.pdf>
- Civil Rights Cases, 109 U.S. 3 (1883) (containing the combined cases of *U.S. v. Stanley*; *U.S.v. Ryan*; *U.S.v. Nichols*; *U.S.v. Singleton*; and *Robinson et ux. v. Memphis & Charleston R.R. Co.*). Retrieved from <https://www.loc.gov/item/usrep109003/>
- Hahn, R., Truman, B., & Williams, D. (2018). Civil rights as determinants of public health and racial and ethnic health equity: Health care, education, employment, and housing in the United States. *SSM-Population Health*, 4, 17–24. <https://doi.org/10.1016/j.ssmph.2017.10.006>.
- Hospital Survey and Construction Act, Pub. L. No. 725, 60 Stat 1040–1049, codified as amended at 42 U.S.C. §§ 201–209, 210–229, and 241–286 (1946) and at 42 U.S.C. § 291 *et seq.* (1976). Retrieved

- from <https://www.loc.gov/law/help/statutes-at-large/79th-congress/session-2/c79s2ch958.pdf>
- Kirschner, K., Breslin, M., & Iezzoni, L. (2007). Structural impairments that limit access to health care for patients with disabilities. *JAMA*, 297(10), 1121–1125. <https://doi.org/10.1001/jama.297.10.1121>.
- Krahn, G., Walker, D., & Correa-De-Araujo, R. (2015). Persons with disabilities as an unrecognized health disparity population. *American Journal of Public Health*, 105(S2), S198–S206. <https://doi.org/10.2105/AJPH.2014.302182>.
- Lewis, T., et al. (2009). Perceived discrimination and blood pressure in older African American and white adults. *Journal of Gerontology Series A: Biological Sciences and Medical Sciences*, 64(9), 1002–1008. <https://doi.org/10.1093/gerona/glp062>.
- Longest, B. (2016). *Health policymaking in the United States* (6th ed.). Chicago: Health Administration Press.
- Major, B., Dovidio, J., & Link, B. (Eds.). (2018). *The Oxford handbook of stigma, discrimination, and health*. New York: Oxford University Press.
- Oliver Brown et al. v. Board of Education of Topeka et al., 347 U.S. 483 (1954).
- Pascoe, E., & Richman, S. (2009). Perceived discrimination and health: A meta-analytic review. *Psychological Bulletin*, 135(4), 531–554. <https://doi.org/10.1037/a0016059>.
- Rosenbaum, S., Markus, A., & Darnell, J. (2000). US civil rights policy and access to health care by minority Americans: Implications for a changing health care system. *Medical Care Research and Review*, 57(1), 236–259. <https://doi.org/10.1177/1077558700057001S11>.
- Sandoval-Strausz, A. (2005). Travelers, strangers, and Jim Crow: Law, public accommodations, and civil rights in America. *Law and History Review*, 23(1), 53–94. <https://doi.org/10.1017/S0738248000000055>.
- Trump, C. E., & Ayres, K. M. (2020). Autism, insurance, and discrimination: The effect of an autism diagnosis on behavior-analytic services. *Behavior Analysis in Practice*, 13, 282–289. <https://doi.org/10.1007/s40617-018-00327-0>.
- U.S. Commission on Civil Rights. (1965). *Equal opportunity in hospitals and health facilities: Civil rights policies under the Hill-Burton program*. Report No. 2. Retrieved from <https://www.nlm.nih.gov/exhibition/forallthepeople/img/1706.pdf>
- U.S. Department of Health & Human Services Centers for Disease Control and Prevention. (2019). *NCHHSTP social determinants of health: Frequently asked questions*. Retrieved from <https://www.cdc.gov/nchhstp/socialdeterminants/faq.html>
- U.S. Department of Health & Human Services Office for Civil Rights. (2014). *Know the rights that protect us from discrimination based on race, color or national origin*. Retrieved from <https://www.hhs.gov/sites/default/files/yourrightsundertitleviofthecivilrightsactfactsheet.pdf>
- U.S. Department of Health & Human Services Office for Civil Rights. (2020). *FAQS: What are civil rights?*. Retrieved from <https://www.hhs.gov/civil-rights/for-individuals/faqs/what-are-civil-rights/101/index.html>
- U.S. Department of Health & Human Services Office of Disease Prevention and Health Promotion. (2020a). *Social determinants of health: Interventions and resources: Discrimination*. Retrieved from <https://www.healthypeople.gov/2020/topics-objectives/topic/social-determinants-health/interventions-resources/discrimination>
- U.S. Department of Health & Human Services Office of Disease Prevention and Health Promotion. (2020b). *Social determinants of health: Overview*. Retrieved from <https://www.healthypeople.gov/2020/topics-objectives/topic/social-determinants-of-health>
- U.S. Department of Justice Civil Rights Division. (2019). *Regulations, manuals, guidance and reports*. Retrieved from <https://www.justice.gov/crt/regulations-manuals-guidance-and-reports>
- U.S. Department of Justice Civil Rights Division Federal Coordination and Compliance Section. (2016). *Title VI of the Civil Rights Act of 1964: 42 U.S.C. § 2000d et seq.* Retrieved from <https://www.justice.gov/crt/fcs/TitleVI-Overview>
- U.S. Department of Labor Office of the Assistant Secretary for Administration & Management Civil Rights Center. (2020). *Legal highlight: The Civil Rights Act of 1964*. Retrieved from <https://www.dol.gov/agencies/oasam/civil-rights-center/statutes/civil-rights-act-of-1964>
- U.S. Department of the Interior National Park Service. (2016). *Civil Rights Act of 1964*. Retrieved from <https://www.nps.gov/articles/civil-rights-act.htm>
- U.S. Government Publishing Office. (2020). *Content details: 78 Stat. 241*. Retrieved from <https://www.govinfo.gov/app/details/STATUTE-78/STATUTE-78-Pg241>
- U.S. Library of Congress. (2020). *The Civil Rights Act of 1964: A long struggle for freedom*. Retrieved from <https://www.loc.gov/exhibits/civil-rights-act/civil-rights-act-of-1964.html>

CLAMS

► Clinical Linguistic and Auditory Milestone Scale

Clancy Autism Behavior Scale

► Clancy Behavior Scale

Clancy Behavior Scale

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Synonyms

CABS; Clancy autism behavior scale

Description

The Clancy Behavior Scale is an early autism descriptive and classification tool first published in 1969 by Clancy, Dugdale, and Rendle-Short in order to better describe and classify autism in young children. Mothers provided reports of child difficulty across 14 major domains, and the instrument was suggestive of “infantile autism” if seven or more of these domains were endorsed as areas of concern.

Historical Background

In the mid-to-late 1960s, the clinical researchers Helen Clancy, Alan Dugdale, and John Rendle-Short in the Department of Child Health from the University of Queensland, Brisbane, Australia, attempted to develop a tool for more reliably describing and accurately identifying young children with autism. They suggested that accurate

classification could be enhanced by identifying significant vulnerabilities across a 14-point major manifestation scale including great difficulty in mixing and playing with other children, acts as deaf, strong resistance to any learning, lack of fear about realistic dangers, resists change in routine, prefers to indicate needs by gestures, laughing and giggling for no apparent reason, not cuddly as a baby, marked physical overactivity, no eye contact, unusual attachment to a particular object or objects, spins objects especially round ones, repetitive and sustained odd play, and standoffish manner. While the scale promoted the use of specific tools and rating systems for improved descriptive and classification purposes, it was not extensively studied or utilized across clinical populations over time.

Psychometric Data

Fairly limited data regarding the scale’s psychometric properties is available. Capute et al. (1975) conducted a prospective study of 200 children to evaluate the reliability and validity of the Clancy Behavioral Scale. Using only the scale, 48 of 200 children met cutoffs for autism risk; however, only one of these children actually fulfilled Kanner’s (1943) criteria for infantile autism. These false positives were suggested to correlate with increasing severity of cognitive deficits, learning disorders, and hearing loss.

Clinical Uses

This scale utilizes parent report of behavior to indicate symptoms across 14 symptoms of infantile autism. The scale has not been extensively studied, nor is it commonly utilized across clinical populations at present. However, it has seen increasing use by research teams in China (Wu et al. 2017; Sun et al. 2013, 2014; Chen et al. 2007; Ke et al. 2002) in screening for autism, investigating prevalence, validating Mandarin-language screening tools, and differentiating

between other language, cognitive, and behavioral disorders.

See Also

► [Childhood Autism Rating Scale](#)

References and Reading

- Capute, A. J., Derivan, A. T., Chauvel, P. J., & Rodriguez, A. (1975). Infantile autism: I. A prospective study of the diagnosis. *Developmental Medicine and Child Neurology*, 17, 58–62.
- Chen, Y., Chen, Z.-M., Hu R.-L., et al., Language Disorder Center of the First Affiliated Hospital of Jinan University, Guangzhou 510630, China. (2007). Clinical application of Clancy autism behavior scale. *Guangdong Medical Journal*, 28, 375–77.
- Clancy, H., Dugdale, A., & Rendle-Short, J. (1969). The diagnosis of infantile autism. *Developmental Medicine and Child Neurology*, 11, 432–442.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250. Kanner, L. (1968). Reprint. *Acta Paedopsychiatr*, 35(4), 100–136.
- Ke, X. Y., Luo, S. J., Tao G. T., Child Mental Health Research Center of Nanjing Brain Hospital Affiliated of NJMU, Nanjing, 210029, China. (2002). A study of Clancy behavior scale on childhood autism. *Acta Academiae Medicinae Jiangxi*, 142(6), 136–137.
- Rimland, B. (1971). The differentiation of childhood psychoses: An analysis of checklists for 2218 psychotic children. *Journal of Autism and Childhood Schizophrenia*, 1, 161–174.
- Sun, X., Allison, C., Matthews, F. E., et al. (2013). Prevalence of autism in mainland China, Hong Kong and Taiwan: a systematic review and meta-analysis. *Molecular Autism*, 4(1), 7.
- Sun, X., Allison, C., Auyeung, B., et al. (2014). Comparison between a Mandarin Chinese version of the Childhood Autism Spectrum test and the Clancy Autism Behaviour Scale in mainland China. *Research in Developmental Disabilities*, 35, 1599–1608.
- Wu, X., Tao, S., Rutayisire, E., et al. (2017). The relationship between screen time, nighttime sleep duration, and behavioral problems in preschool children in China. *European Child and Adolescent Psychiatry*, 26(5), 541–548.

Classical Conditioning

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Synonyms

[Associative learning](#); [Pavlovian conditioning](#)

Definition

The learning phenomena behind Pavlov's oft-cited salivating dogs (Pavlov 1927) or Little Albert's terror and crying in response to a white rat (Watson and Rayner 1920) are perhaps more scientifically extraordinary than are common sense. It is a unique phenomenon that demonstrates the intricacies of how the brain forms memories and makes meaning. Classical conditioning, or "Pavlovian conditioning," is a type of associative learning. The association between stimuli, or events that are linked in close timing, may be solidified after one or two pairings of the events in close succession. For example, most of us know that a flash of lightning (visual stimuli) will likely result in a thunderous boom (auditory stimuli), which can be loud and frightening. When an individual cringes/winces at the lightning, instead of and before the thunder is emitted, classical conditioning has occurred – that is, the lightning became a "conditioned" trigger for fear. In anticipation of the thunder, the individual reacts to the lightning and not the thunder itself.

Classical conditioning explains a broad number of phenomena in our everyday learning. Not to be confused with operant conditioning, which is a closely related form of associative learning (and the learning phenomenon upon which applied behavioral analysis is based; see Skinner 1953, 1957), classical conditioning is the learning that results from associating two closely timed events: the salivating that takes place upon a dinner bell, instead of for the actual food itself; the sexual

Class Versus Variable

► [Dimensional Versus Categorical Classification](#)

response evoked by a familiar scent or perfume, without the presence of the actual person associated with the memory. Classical conditioning can result in fear, hunger, and sexual and sleep responses, conditioned to a once-neutral event or stimulus. Thus, it is a human ability to predict or anticipate an upcoming pleasurable or aversive event (Rescorla and Wagner 1972). The prediction and anticipation may generalize to other similar stimuli.

Though mathematical models and contemporary descriptions of “configural encoding” have been developed to account for and/or predict the complexities of classical conditioning (Rescorla and Wagner 1972; Rescorla 2003), the occurrence is visible in everyday events. Any environmental event that pairs the human senses (i.e., stimuli) and human need or emotion is a prime opportunity for classical conditioning to take place.

In the applied field of autism intervention and treatment research, classical conditioning approaches take a backseat to operant conditioning approaches. However, a classical conditioning procedure, systematic desensitization, has been used to reduce unwanted fear in children with autism (Love et al. 1990); this procedure involves gradually increasing the intensity of a fear-evoking stimulus. Classical conditioning has also been used in laboratory studies of characteristics associated with autism. For example, Stanton, Peloso, Brown, and Rodier (2007) found that classical conditioning of an eyeblink response occurred more rapidly in a rodent model of autism than in other rodents. Similarly, Sear, Finn, and Steinmetz (1994) found more rapid eyeblink conditioning in children with autism than in typically developing children.

See Also

► [Operant Conditioning](#)

References and Reading

- (1994). *Journal of Autism Developmental Disorders*, 24(6), 737–751.
- Love, S. R., Matson, J. L., & West, D. (1990). Mothers as effective therapists for autistic children’s phobias. *Journal of Applied Behavior Analysis*, 23, 379–385.

- Pavlov, I. P. (1927). *Conditioned reflexes: An investigation of the physiological activity of the cerebral cortex* (G. V. Anrep, Trans.). London: Oxford University Press.
- Poulos, A. M., & Thompson, R. F. (2004). Timing of conditioned responses utilizing electrical stimulation in the region of the interpositus nucleus as a CS. *Integrative Psychological and Behavioral Science: The Official Journal of the Pavlovian Society*, 39, 83–94.
- Rescorla, R. A. (2003). Contemporary study of Pavlovian conditioning. *Spanish Journal of Psychology*, 6, 185–195.
- Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. In A. H. Black & W. F. Prokasy (Eds.), *Classical conditioning II* (pp. 64–99). Appleton: Century-Crofts.
- Sear, L. L., Finn, R. R., & Steinmetz, J. E. (1994). Abnormal classical eye-blink conditioning in autism. *Journal of Autism and Developmental Disorders*, 24, 737–751.
- Skinner, B. F. (1953). *Science and human behavior*. New York: Macmillan.
- Skinner, B. F. (1957). *Verbal behavior*. Englewood Cliffs: Prentice-Hall.
- Stanton, M. E., Peloso, E., Brown, K. L., & Rodier, P. (2007). Discrimination learning and reversal of the conditioned eyeblink reflex in a rodent model of autism. *Behavioral Brain Research*, 176, 133–140.
- Watson, J. B., & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology*, 3, 1–14.

Classroom Aide

- [Para-educator](#)
 ► [Paraprofessional](#)

Classroom Management

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Definition

Classroom management refers to ways of organizing resources, students, procedures, and routines of a classroom so that teaching and learning can proceed in a safe and effective manner.

Historical Background

Historically, specialists in the field of education state that classroom management encourages the establishment of student self-control through positive achievement and behavior. Classroom management is closely linked to issues of motivation, establishing a climate of respect between classroom staff and students and also consistent discipline. The teacher is at a huge advantage when she or he spends the time to set up classroom management that looks at content management (skills that cut across subjects and activities; cf. instructional management skills, sequencing and integrating additional instructional activities, as well as instruction-related discipline problems [Kounin as cited in Froyen and Iverson 1999, p. 128]), conduct management (inclusion of human diversity into one's instructional philosophy), and covenant management (classroom group and social systems). Research demonstrates that a high incidence of disciplinary problems in the classroom results in a significant impact on effectiveness of teaching and learning. Additional research indicates that strong consistent management and organizational skills lead to fewer classroom discipline problems (Johansen et al. (2011), www.intime.uni.edu/model/teacher/teac3_summary.html). Throughout the years, classroom management has created areas of debate among teachers; however, it is widely recognized that a key component of classroom management is the application and implementation of behavioral approaches.

Sulzer-Azaroff (1981 in Bijou and Ruiz, p. 64) stated the use of behavior modification in the classroom parallels the development of behavior modification in the field of mental health. The majority of early studies conducted in the 1960s focused on the reduction of disruptive behaviors by changing teacher behavior; however, this early application of behavior principles did not teach the students an alternative behavior. Careful consideration of research shared by Birnbrauer et al. (1965), Brigham and Sherman (1968), and Buell, Stoddard, Harris, and Baer (1968) yielded the need to focus on using behavioral procedures to teach students in a way that classroom productivity, language development, and social skills were promoted (Sulzer-Azaroff 1981 in Bijou and Ruiz

1981, pp. 65–67). Subsequent research has contributed to a growing body of research that supports positive classroom management through the use of modeling behavior expectations and differential reinforcement procedures (Sulzer-Azaroff and Mayer 1986). Throughout the years, this research has become more refined and focused on a variety of needs that are represented in the learning characteristics of students with autism spectrum disorders (ASD).

Current Knowledge

The current trends in education emphasize the establishment of positive behavior supports (PBS) and the use of positive behavioral interventions and supports (PBIS) to achieve socially important change (Sugai et al. 2000). The application of positive behavior supports (PBS) and positive behavioral interventions and supports (PBIS) is supported by the US Department of Education and Office of Special Education Programs (OSEP), with an emphasis for schools to use the PBIS framework to impact social, academic, and emotional outcomes for students with disabilities (<https://www.pbis.org>). Although the use of such systems is best practice, students with ASD present unique characteristics within a learning environment. The teacher is challenged to incorporate these unique learning needs into meaningful classroom management and instruction. To do this, the teacher must take into account the needs of the learner in a variety of educational settings. These settings necessitate careful thought about physical structure, instructional management, the student's ability in the areas of communication and social skills and the need to teach the student how to learn under a variety of conditions.

Classroom management for the student with ASD should include consideration of the following aspects of instruction:

1. Physical space in the classroom needs to be set up with clearly defined areas that have visual boundaries (e.g., independent work areas, group work areas, an area for use of technology such as a SMART Board or computer, areas that have critical visual information related to

academic subjects, lighting, noise levels, and the like).

2. Tasks should be presented with clear beginnings and ends – students should be able to recognize when they should start and finish work as well as when they should put away materials (they also need to know where the materials go).
3. Routines should be incorporated into the classroom and flexibility taught and incorporated into plans, that is, program for routine and change.
4. Tasks should be clearly organized, and information should be presented visually.
5. Materials and tasks should be structured and modified so that the student is able to independently respond to the task/lesson.
6. Transitions join tasks together in a natural way – specific transitional elements link tasks together into multitask systems.
7. Communication is used to foster independence – systems are designed so that communication takes place as much as possible without adult presence and dependence.
8. Specific work systems are set up (Montgomery County Public Schools, Services for Students with Autism Spectrum Disorders 2009).

Students with ASD rely heavily on structure and predictable routines, and as such, structure and predictable routines should be incorporated into classroom management. It is key to use these structures and routines consistently and with fidelity. Students with ASD also may need customized visual daily schedules, reduced auditory input, succinct verbal instructions that emphasize key points, consideration of reduced visual distractions (e.g., movement, reflections, background patterns), and consideration of reducing other environmental stimuli that may be incompatible with the sensory sensitivities that are associated with autism spectrum disorders (e.g., temperatures, textures, smells, tastes, the need to move or have movement breaks). The student with ASD will also need to have advance warning about changes in his/her environment; they may need a special place to go to that offers opportunity for relaxation and/or relief from stressful situations that may result from innate anxiety. The student also needs to have contact

with peers who model and offer appropriate social interactions. The teacher should consider the needs of the student with ASD with regard to teaching specific communication and social skills and should structure the environment in a way that promotes these skills within the context of daily routines.

Future Directions

Students with ASD are increasingly present in general education settings and classrooms. As such, teachers need to be aware of their unique learning profiles and ways to incorporate their needs into classroom management. The current movement of the use of positive behavioral intervention systems and school-wide positive behavioral intervention supports is a start in this direction; however, individualization will remain paramount if students with ASD are to have a successful educational experience.

See Also

- [Positive Behavior \(al\) Interventions and Supports \(PBIS\)](#)
- [Positive Behavioral Support](#)

References and Reading

- Bijou, S., & Ruiz, R. (Eds.). (1981). *Behavior modification contributions to education*. Hillsdale: Lawrence Erlbaum Associates Publishers.
- Birnbrauer, J. S., Wolf, M. M., Kidder, J. D., & Tague, C. (1965). Classroom behaviour of retarded pupils with token reinforcement. *Journal of Experimental Child Psychology*, 2, 219–235.
- Brigham, T. A., & Sherman, J. A. (1968). An experimental analysis of verbal imitation in preschool children. *Journal of Applied Behavior Analysis, Summer 1*(2), 151–158. <https://doi.org/10.1901/jaba.1968.1-151>. PMID: PMC1310991.
- Buell, Stoddard, Harris, & Baer (1968); (Hart & Risley, 1995). For many years, teachers and 118 / May 2009. *Behavioral Disorders*, 34(3), 118–135.
- Froyen, L. A., & Iverson, A. M. (1999). *Schoolwide and classroom management: The reflective educator-leader* (3rd ed.). Upper Saddle River: Prentice Hall.
- Fullerton, A., Stratton, J., Coyne, P., & Gray, C. (1996). *Higher functioning adolescents and young adults with autism: A teacher's guide*. Austin: Pro-Ed.

- Johansen, A., Little, S.G., & Akin-Little, A. (2011). In B. S. Parsonson (2012). Evidence based classroom behavior management strategies. *Kairaranga*, 13(2), 16–23.
- Montgomery County Public Schools, Montgomery County Maryland, Services for Students with Autism Spectrum Disorders. Unpublished manuscript, Silver Spring.
- Moore, S. T. (2002). *Asperger syndrome and the elementary school experience: Practical solutions for academic & social difficulties*. Shawnee Mission: Autism Asperger Publishing Company.
- Pierangelo, R., & Giuliani, G. (2008). *Teaching students with autism spectrum disorders*. Thousand Oaks: Corwin Press.
- Quill, K. A. (1995). *Teaching children with autism: Strategies to enhance communication and socialization*. New York: Delmar.
- Scott, T. M., Anderson, C. M., & Alter, P. (2012). *Managing classroom behavior using positive behavior supports*. Boston: Pearson.
- Sugai, G., Horner, R. H., Dunlap, G., Hieneman, M., Lewi, T. J., Nelson, C. M., Scott, T., Liaupsin, C., Saylor, W., Turnbull, A. P., Turnbull III, H. R., Wickham, D., Wilcox, B., & Ruef, M. (2000). Applying behavior support and functional behavior assessment in schools. *Journal of Positive Behavior Interventions*, 2(3), 131–143.
- Sulzer-Azaroff, B. (1981). Issues and trends in behavior modification in the classroom. In S. W. Bijou & R. Ruiz (Eds.), *Behavior modification contributions to education* (pp. 63–93). Hillsdale: Lawrence Erlbaum Associates.
- Sulzer-Azaroff, B., & Mayer, G. R. (1986). *Achieving educational excellence using behavioral strategies*. New York: Holt, Rinehart, and Winston.
- www.intime.uni.edu/model/teacher/teac3summary.html
- www.pbis.org/school/what_is_swpbs.aspx
- <https://www.pbis.org>

Classroom Structure

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Synonyms

Organization of the physical learning environment

Definition

How the physical environment is organized to facilitate student success in learning (Interactive

Collaborative Autism Network (ICAN) [n.d.](#)). For students with autism spectrum disorders (ASD), classrooms should include a high degree of structure in order to ensure success (Bodfish 2004; Iovannone et al. 2003; Mesibov and Shea 2010). The Structured Teaching approach (an evidence-based approach devised by the TEACCH [Treatment and Education of Autistic and related Communication-handicapped Children] Program in North Carolina, Mesibov & Shea) includes structuring the physical environment (it also incorporates the strategic environment of learning approaches which will not be considered here).

Effective classroom structure for students with ASD should include (Ball [n.d.](#); Mesibov et al. 2004; Mesibov and Shea 2010):

- Physical structure – using furniture to demonstrate expectations and reduce distractions.
- Visual schedules – using objects, pictures, or the written word to show the student the sequence of events.
- Visually structured individual tasks that incorporate object, picture, and/or written instructions.
- Organizing a sequence of individual tasks using visual work/activity systems – using objects, pictures, letters, numbers, or the written word, tasks are organized to show the student what they have to do, how many tasks they need to do, how they are progressing, when they will be finished, and what they are going to do next. For example, lining up the tasks on the students' left and having them move them to their right when completed.

Within these features, the key to student success is that the details of the structure of the classroom are individualized according to the strengths and weaknesses of each student (Mesibov et al. 2004).

See Also

- Culture and Autism
- Educational Interventions
- Pictorial Cues/Visual Supports (CR)
- Structured Classrooms

- [Structured Teaching](#)
- [TEACCH Transition Assessment Profile \(TTAP\)](#)
- [Visual Schedule](#)
- [Visual Supports](#)

References and Reading

- Ball, J. (n.d.). Structured classrooms, virtual speaker by talk autism [video]. Retrieved 25 Jan 2011, from <http://www.talkautism.com/Components/Video/Video.aspx?v=59>
- Bodfish, J. W. (2004). Treating the core features of autism: Are we there yet? *Mental Retardation and Developmental Disabilities Research Reviews*, 10, 318–326.
- Interactive Collaborative Autism Network (ICAN). (n.d.). *Classroom structure*. Retrieved 25 Jan 2011, from <http://www.autismnetwork.org/modules/envirocnstructure/index.html>
- Iovannone, R., Dunlap, G., Huber, H., & Kincaid, D. (2003). Effective educational practices for students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 18, 150–165.
- Mesibov, G. B., & Shea, V. (2010). The TEACCH program in the era of evidence-based practice. *Journal of Autism and Developmental Disorders*, 40, 570–579.
- Mesibov, G., Shea, V., & Schopler, E. (2004). *The TEACCH approach to autism spectrum disorders*. New York: Springer.

Client Assistance Program

- [Protection and Advocacy System \(P&A\)](#)

Client Emotional Processing Scale for Autism Spectrum

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Synonyms

[Asperger syndrome \(AS\)](#); [Client Emotional Processing Scale for Autism Spectrum \(CEPS-AS\)](#); [Empathy](#); [Mental representation](#)

Description

The Client Emotional Processing Scale for Autism Spectrum (CEPS-AS) is an observer measure that assesses changes in four emotional processing dimensions: (a) emotion encoding and symbolizing, (b) self-reflective processing, (c) empathy, and (d) mental representation. The CEPS-AS is developed as four subscales, each as a five-point scale designed to be applied to video recordings or transcripts of group psychotherapy. The first subscale consists of five scale stages that define the progression of client involvement along emotion processing continuum that runs from a lack of referent to emotional experience (=scale point 1), through externalized experience (=2), to dysregulation (=3), to internally located and encoding experience (=4), and finally to internal symbolizing and interpersonal awareness of emotion (=5). The second subscale defines the progression of client involvement along the self-reflective processing continuum that runs from descriptive accounts with a scripted quality to self-narrative (=scale point 1), through to a deficit referent to AS self-narrative (=2), to immediate awareness of self that possesses a present quality (=3), to new self-insight with interpersonal awareness (=4), and to finally an introspective referent to self that possesses fluidity and complexity (=5). The third subscale defines the progression of client involvement along the empathy processing continuum that runs from a self-absorbed narrative that lacks any empathic attunement (=scale point 1), through narratives that reflect self as oriented towards others, but where cognitive formulations or empathic conjectures are offered but not synchronized (attuned with the other) (=2), to immediate sharing of affect and resonance through empathic attunement (=3), to accurate sensing of the other with an awareness that they can move and be moved by the other (=4), and finally through to narratives that reflect being mobilized into action as a response to sensing the emotion of the other (=5). The fourth subscale defines the progression of client involvement along the mental representation processing continuum that runs from a narrative which projects own thoughts onto others and discourse that assumes that others have

an awareness of the person's implicit mental representations (=scale point 1), through to an awareness of own and other possessing separate mental representations (=2), to flexibility in manipulating and changing own mental representations (=3), to narrative that reflects an emergence of metacognitive processing of misunderstandings (=4), and finally to narrative that reflects metacognitive thinking in how consideration of own and others' mental representations can occur (=5).

The CEPS-AS is an observer measure and requires raters to be sufficiently trained to identify performance markers in the four dimensions and across experiencing levels. Training skilled autism practitioners to rate performance markers to sufficient levels require a minimum of two training sessions using practice video material to rate. Four-minute video segments are observed and rated. The coding procedure uses partial interval sampling (Bakeman and Gottman 1997) by raters coding the presence ("1") or absence ("0") of behavioral indicators of each of the five ordered levels for each of the four subscales. The scoring procedure uses mean values. The mean indicator value is calculated for each segment. Each 4-min video segment is rated for each client times each dimension ($n \times 4$). The mean indicator values are used for reliability analyses. The interrater reliability is calculated for each individual rater using Pearson r and reliability is calculated for cross-judge averaged data using Cronbach alpha. To calculate session-by-session comparisons of client performances, segments are summarized by averaging first across raters, then across segments within sessions and finally across clients.

Historical Background

The CEPS-AS was developed by psychologists Anna Robinson, PhD, and Robert Elliott, PhD, and published in 2016 by *Journal of Autism and Developmental Disorders*. It was designed to measure emotional processing across group psychotherapy, as observed by therapists and researchers. It was modeled on the Client Experiencing Scale (Klein et al. 1986) and

developed through direct observation of client process during Humanistic Experiential Psychotherapy (HEP). CEPS-AS items were generated from the initial and final video-recall sessions taken from small group Emotion-Focused Therapy with adolescent and adults with Asperger syndrome (AS). Discourse analytic methods were used to identify and describe markers of emotional processing performance resulting in 306 performance markers that were organized within each of the emotional processing domains and codified via an open coding process. The emotion processing domain contained 77 performance markers; the empathy processing domain contained 49 performance markers; the self-reflective processing domain contained 86 performance markers, and the fourth, mental representation processing domain contained 94 performance markers. Using the constant comparative method (Glaser and Strauss 1967) these performance markers were clustered into five graded categories across a continuum of processing in each of the four emotional processing domains.

Following the construction of the CEPS-AS initial validation tests were carried out with two raters. Raters tested video material extracted from two adolescent and adult HEP groups. Raters independently assessed for presence absence of emotional processing performance markers followed by level of performance across the four subscales. Interrater reliability was calculated for both individual raters (Pearson r) reliability and cross-judge averaged data (Cronbach alpha). Preliminary interrater reliabilities of presence absence judgments were quite high for overall ratings averaged across dimensions, with an alpha reliability of 0.84 for judgments combined across the two raters. The alpha reliabilities for ratability judgments on the individual dimensions, for ratings averaged across raters, varied from 0.75 to 0.93, indicating consistently good to excellent interrater reliability.

Psychometric Data

Sensitivity of the CEPS-AS was carried out on the performance markers contained within each of the

four domains. Two raters independently rated 42 four-minute segments of video footage across three periods of treatment: the first regular group therapy session, the first video playback/recall session, and the final video playback/recall session, for both adult and adolescent groups. Each 4-min video segment was rated 12 times (3 clients $\times$ 4 dimensions) by each rater. From the initial trials, CEPS-AS results show interrater reliabilities for processing dimension ratings combined across raters (Cronbach alphas) varied from 0.69 (Emotion Regulation) to 0.91 (Mental Representation); interrater reliability for ratings averaged across the four dimensions was 0.91 indicating good to excellent interrater reliability. However, ratings by single raters (correlations) were somewhat lower, varying from 0.53 (Emotion Regulation) to 0.84 (Mental Representation). Initial findings found a high degree of interrater reliability in identifying the presence or absence of performance markers for the four emotional processing dimensions.

Interdimension correlations were analyzed for both raters (Pearson correlations) across the four processing dimensions. All four dimensions were significantly correlated with each other; this varied from 0.66 ($p < 0.01$; empathy and self-reflection) to 0.82 ($p < 0.01$; self-reflection and mental-representation). The overall interdimension reliability statistic for ratings averaged across dimensions (Cronbach alpha) was 0.91. Preliminary findings show a high degree of overlap, indicating that the four items of the CEPS-AS are not in fact independent dimensions but rather closely interwoven components of emotional processing.

The CEPS-AS was constructed to track emotional processing change across treatment of a group HEP. Sensitivity to change was assessed using repeated measures ANOVA for overall emotional processing and for each of the four processing dimensions. The repeated measure ANOVAs were highly significant for each of the four processing dimensions: Emotion Regulation ($F = 32.70$; $df\ 2,7$; $p = 0.01$); Empathy ($F = 50.45$; $df\ 2,9$; $p = 0.01$); Self-Reflection ($F = 12.83$; $df\ 2,11$; $p = 0.01$); Mental Representation ($F = 34.50$; $df\ 2,12$; $p = 0.01$). The CEPS-

AS demonstrated a moderate to high degree of interrater reliability for discriminating experiential levels across each of these dimensions.

Clinical Uses

CEPS-AS is claimed to be the first reported observer measure for emotional processing using a cognitive-affective self-other dimensional framework. The preliminary findings indicate that the CEPS-AS is sensitive to assessing changes in emotional processing across an intervention aimed at helping participants develop better self-empathy and other empathy at both affective and cognitive levels. In the UK, group social psychoeducation intervention is recommended (National Institute for Health and Care Excellence 2012) for adults with Autism Spectrum Conditions (ASC). Therefore, the CEPS-AS may be a useful clinical tool for therapists to monitor change during group therapy. Measuring changes in empathy in autism uses self-assessment measures, such as the Empathy Quotient (Baron-Cohen and Wheelwright 2004). Preliminary findings from the CEPS-AS indicate potential to discriminate observable changes over the course of treatment. Therefore, CEPS-AS has the potential to be a useful observation tool for clinical trials research on both Humanistic-Experiential Psychotherapies (HEP) and Cognitive Behavior Therapies (CBT). Having an empathy observer measure is a potential useful addition in researching evidence-based practice for group psychotherapies. It will enable researchers to triangulate data with self-assessment empathy instruments.

Although preliminary the CEPS-AS can be viewed as a promising new observer instrument for assessing and tracking client emotion processing and empathy over the course of psychotherapeutic treatment. The initial findings reported high interdimension correlations indicating redundancy among the dimensions requiring further testing to ascertain whether the cognitive-affective components are so closely related that they are not distinct constructs but instead may be overlapping components of the same construct.

Observer measures are time-consuming to carry out and often require significant training of raters therefore reducing the number of items may make the CEPS-AS faster and easier to use. This would be a useful step for clinical use to track changes across treatment.

As a new measure the CEPS-AS research to date is limited. There is the need to evaluate convergent or discriminant validity by assessing empathy self-report (such as, the Empathy Quotient (EQ)), emotion self-report measures (such as the Toronto Alexithymia Scale (TAS-20) Bagby et al. 1994). There is a need to test the validity of the CEPS-AS subscales against performance-based measures such as the Revised Eyes Test (Baron-Cohen et al. 2001). However, initial validation demonstrates the CEPS-AS is sensitive to tracking changes in performance markers in emotion processing regulation, empathy, self-reflection, and mental representation in adolescents and adults with ASC. The CEPS-AS offers a unique observer tool for assessing emotional processing domains across group psychotherapy, as observed by researchers.

See Also

- [Asperger Syndrome](#)
- [Empathy](#)

References and Reading

- Bagby, R. M., Parker, J. D. A., & Taylor, G. J. (1994). The twenty-item Toronto alexithymia scale-I. Item selection and cross-validation of the factor structure. *Journal of Psychosomatic Research*, 38, 23–32.
- Bakeman, R., & Gottman, J. M. (1997). *Observing interaction: An introduction to sequential analysis* (2nd ed.). New York: Cambridge University Press.
- Baron-Cohen, S., & Wheelwright, S. (2004). The empathy quotient: An investigation of adults with Asperger syndrome or high functioning autism, and normal sex differences. *Journal of Autism and Developmental Disorders*, 34, 163–175.
- Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The “reading the mind in the eyes” test revised version: A study with normal adults, and adults with Asperger syndrome or high functioning autism. *Journal of Child Psychology and Psychiatry*, 42, 241–251.
- Glaser, B. G., & Strauss, A. L. (1967). *The discovery of grounded theory*. Chicago: Aldine.
- Klein, M. H., Mathieu-Coughlan, P., & Kiesler, D. J. (1986). The experiencing scales. In L. Greenberg & W. Pinsof (Eds.), *The psychotherapeutic process* (pp. 21–71). New York: Guilford Press.
- National Institute for Health and Care Excellence. (2012). *Autism: Recognition, referral, diagnosis and management of adults on the autism spectrum* (NICE clinical guideline, 142). London: British Psychological Society & The Royal College of Psychiatrists.
- Robinson, A., & Elliott, R. (2016). Brief report: An observational measure of empathy for autism spectrum: A preliminary study of the development and reliability of the client emotional processing scale. *Journal of Autism and Developmental Disorders*, 46, 2240–2250.

Client Emotional Processing Scale for Autism Spectrum (CEPS-AS)

- [Client Emotional Processing Scale for Autism Spectrum](#)

Clinical Assessment

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Definition

Clinical assessment is the art and science of understanding a person’s behavior using a variety of perspectives (e.g., biological, psychological, and social/cultural) and within the different contexts in which he or she lives. As a science, clinical assessment strives to develop procedures and judgments based upon empirical evidence and

the *critical evaluation of that evidence*. As an art, clinical assessment relies on the creativity and innovation of the clinician to synthesize relevant findings and to develop new theoretical models, new assessment tools, and new applications of existing knowledge and skills beyond the limits of current research yet still be guided by the values, principles, and consensus expert opinion of what constitutes best practice.

Clinical assessment, in general, is always defined within the current historical and cultural context, by the training and credentials of the clinician, and by the purpose of the evaluation. Similarly, clinical assessment of autism spectrum disorder (ASD) is shaped by the current state of knowledge regarding the characteristics of ASD, licensing requirements for clinical practice, and the referral question(s) that will be addressed by the evaluation. The current best practice use of the biopsychosocial model of clinical assessment integrates the most relevant information about the biological, psychological, and social/cultural processes that contribute to a broad and deep understanding of the presenting problem(s). This multifaceted, investigative model also has the greatest potential to identify the most effective targets or “choice points” of timely interventions.

Currently, ASD is primarily defined by specific developmental and behavioral markers (e.g., American Psychiatric Association 2013; World Health Organization 1993). Consequently, the most valid and reliable measures for ASD are, broadly speaking, psychological (i.e., behavioral, developmental, psychoeducational, and neuropsychological) in nature. Medical assessment is usually undertaken to assess the general health of the individual, contributing medical conditions, comorbid medical disorders, and the response to medications known to impact behavior. With the “epidemic” growth in the number of individuals being identified with ASD (e.g., Wing and Potter 2009), greater public and media attention, and advances in biological knowledge and medical technology, there is new promise for significant advances in biomedical applications in the assessment of ASD (Goldstein et al. 2009; Volkmar et al. 2009).

Historical Background

Kanner’s remarkable description of eleven patients in his 1943 English-language article (Kanner 1943) is considered by many to mark the beginning of the modern era of the use of the term *autism* as a formal diagnosis (although it is not clear if Kanner was aware of the concurrent work of Austrian Hans Asperger whose own descriptions of a similar group of patients did not get translated into English until a later date). Professionals working in this area fell into a few categories: those who used the descriptions very broadly, those who used the symptoms to diagnosis very narrowly, those who said autism was part of other childhood disorders, those who said autism was separate from other childhood disorders, and those who used the terms autism and childhood schizophrenia interchangeably (Feinstein 2010). Other researchers started trying to specify observable behaviors and came up with sets of criteria. Over the years, researchers developed their own criteria for what defined autism behaviors, and as a result, criteria used for diagnosis changed.

Confusion with diagnostic labeling, criteria, and exclusionary criteria persisted for many years even as researchers started assembling lists of criteria to be used to diagnose autism. One of the first assessment tools used was a checklist developed by C. G. Polan and B. L. Spencer in 1959 (Feinstein 2010). A list of other early assessment tools can be found in Table 1.

These instruments were the forerunners of modern clinical assessments used for diagnosing ASD. Analyses regarding the psychometric properties of the tools are not discussed in this entry as their inclusion relates to the available tools for that period of time, as well as to show the historical progression. The best diagnostic assessments focus on social and communication challenges (Ozonoff et al. 2005). Current assessment tools have expanded their items to include subtleties associated with people who are on the milder end of the autism spectrum. Today, the field has behavior checklists, screening measures, and diagnostic instruments. Table 2 includes currently

Clinical Assessment, Table 1 Historical look at the assessment for autism spectrum disorders

Name of instrument	Authors	Year published/used	Purpose
Rimland's Diagnostic Checklist for Behavior-Disturbed Children (Form E-1) (published as an appendix in the book <i>Infantile Autism</i>)	Bernard Rimland	1964	Originally a checklist for parents to complete and submit regarding the child's early development, language development, and behavior
Behavior Rating Instrument for Autistic and Atypical Children (BRIAAC)	Bertram Rutterberg, Mitchell Dratman, Julia Fraknoi, and Charles Wenar	1966	An observation and rating system for assessing the behavior of autistic or autistic-like children
A parental questionnaire for the Diagnosis of Infantile Autism	Helen Clancy, Alan Dugdale, and John Rendle-Short	1969	Questionnaire to assist with the identification of autism in childhood. Data gained by using the Creak Committee's criteria from 1961
Handicap Behavior and Skills (HBS)	Lorna Wing and Judith Gould	1978	Designed to gain information on children with mental retardation or psychosis
Autism Behavior Checklist/Autism Screening Instrument for Educational Planning (ASIEP)	David Krug, Joel Arick, and Patricia Almond	1978–1980	Assess, identify, and program for children with autism within an educational setting
Behavior Observation Scale for Autism (BOS)	B.J. Freeman, Edward Ritvo, D. Guthrie, P. Schroth, and J. Ball	1978	Devise a method for analyzing behavior associated with an autism diagnosis, assist with the diagnosis of autism, and assess behavioral changes over time
Psychoeducational Profile (PEP)	Eric Schopler and Robert Reichler	1979	A developmental assessment designed for autistic and psychotic children to provide a profile of the child's strengths and needs
Childhood Autism Rating Scale (CARS)	Eric Schopler, Robert Reichler, Robert DeVellis, and Kenneth Daly	1980	Assist with diagnosis, help distinguish children with autism from children with other disorders, and help determine severity level
Autism Observation Scale	Bryna Siegel, Thomas Anders, Ronald Ciaranello, Bruce Bienenstock, and Helena Kraemer	1986	Developing a classification system for subtypes of children with autism and autistic-like symptoms
Autism Diagnostic Interview (ADI)	Ann Le Couteur, Michael Rutter, Catherine Lord, Patricia Rios, Sarah Robertson, Mary Holdgrafer, and John McLennan	1989	Interview questions for caregivers to assist professionals with diagnosing and distinguishing among the pervasive developmental disorders
Autism Diagnostic Observation Schedule (ADOS)	Catherine Lord, Michael Rutter, Susan Goode, Jacquelyn Heemsbergen, Heather Jordan, Lynn Mawhood, and Eric Schopler	1989	Observe social and communication behaviors and the quality of those behaviors in children with autism and related disorders. Also helps in distinguishing autism and related disorders from non-autistic disorders and typical development

Clinical Assessment, Table 2 Current instruments and interactive tools for assessing autism spectrum disorders

Title	Author(s)	Year published	Purpose
Autism Diagnostic Interview-Revised (ADI-R)	Michael Rutter, Ann Le Couteur, and Catherine Lord	2003	Assists with the diagnosis of autism and helps differentiate between autism and other developmental disorders
Social Communication Questionnaire (SCQ): (two versions) Current Behavior or Lifetime Behavior	Michael Rutter, Anthony Bailey, and Catherine Lord	2003	Based on the ADI-R. Can be used as a screener or to gain diagnostic information
Autism Diagnostic Observation Schedule, Second Edition (ADOS-2)	Catherine Lord, Michael Rutter, Pamela DiLavore, Susan Rissi, Katherine Gotham, and Somer Bishop	2012	Interactive and semi-structured assessment of characteristics related to autism spectrum disorders, particularly social and communication skills
Parent Interview for Autism (PIA), Clinical Version	Wendy Stone, Elaine Coonrod, Stacie Pozdol, and Lauren Turner	2003	Tracks changes in child's characteristics of autism. Can also differentiate autism from other developmental disorders
Psychoeducational Profile, Third Edition (PEP-3)	Eric Schopler, Margaret Lansing, Robert Reichler, and Lee Marcus	2005	Gathers information relevant for a diagnosis, identifies child's strengths and needs, and provides developmental levels
Autism Spectrum Rating Scale (ASRS)	Sam Goldstein and Jack Naglieri	2009	Rating scale assesses behaviors related to ASDs. Completed by parents or teachers. Provides T-scores. Long and short versions are available. Assists with diagnosis, differential diagnosis, and comparing the individual suspected of having an ASD to a normed group
Childhood Autism Rating Scale, Second Edition – Standard Version (CARS2-ST) and High-Functioning Version (CARS2-HF)	Eric Schopler, Mary Van Bourgondien, Janette Wellman, and Steve Love	2010	Expands the original CARS and provides an updated literature review. Added a HF version for individuals suspected of having HFA/AS/PDD-NOS. The standard version is redesigned. Both versions offer T-scores

used tools related to the screening or diagnosis of ASD. Instruments showing psychometric promise or those tools where reviewers have mentioned the need for stronger psychometric properties were not included. As a result, the reader may find reviews of these and other instruments in other sections of the encyclopedia. As the criteria for ASD broaden and even more subtle characteristics are noticed, assessment developers continue looking for ways to assess high-functioning individuals with ASD.

Current Knowledge

General Considerations in the Clinical Assessment of ASD

Although the more classic forms of autism may be accurately and reliably diagnosed by most professionals who have met minimal requirements (in terms of training and experience) in clinical assessment, variation in characteristics across the spectrum, myriad comorbid conditions, and the complex interplay with environmental factors

(e.g., parenting style and family stress) require a higher standard of expertise in order to be proficient in assessing individuals with ASD. The essential impact of autism on an individual, itself, can make traditional methods of clinical assessment inadequate, even when used by otherwise experienced and competent examiners. Anecdotal evidence gathered from years of experience working with individuals on the spectrum suggests that additional considerations may be as important in conducting a robust and meaningful assessment as are the specific techniques or procedures identified in the professional literature.

The first of these additional considerations has to do with the levels of social expectation that are built into most assessment procedures. Of the four methods used in clinical assessment – interview, observation, informal assessment, and the use of norm-referenced and standardized instruments (e.g., Sattler and Hoge 2006) – the direct use of formal tests with individuals being evaluated for ASD requires that the individual has the ability and motivation to tolerate and cooperate in the socially reciprocal activities that define the evaluation experience. The ability to regulate oneself in the presence of an unfamiliar adult, to attend to the spoken and unspoken expectations for appropriate behavior, and to be motivated to perform “to the best of one’s ability” is an example of prosocial behaviors that are typically learned at a very young age but may be underdeveloped in a person with ASD. Consequently, unless compensatory strategies are effectively utilized by the examiner, the result may be a child who is (inappropriately) described as “untestable” (e.g., Schopler and Mesibov 1988). Adding to these test-taking social challenges, the fact that many individuals with ASD may not be able to meet the receptive and expressive language demands inherent in many tests and the likelihood that the individual with ASD may not find his or her narrow interests stimulated by the standard test items, a generic clinical evaluation may assist in confirming an individual’s diagnosis, but the potential for the individual to learn and adapt with individualized supports may be largely unexplored. The key is to have a thorough understanding of how ASD affects an individual’s ability to learn and adapt

and to have a repertoire of assessment and teaching strategies that can be evaluated along with the individual (e.g., Klinger et al. 2009; Shea and Mesibov 2009).

The second consideration is that establishing rapport with the individual with ASD is just as important as it is with someone who does not have ASD, but that the process of developing rapport may need to be more deliberate and require more creativity, and will likely be facilitated with detailed information about the individual’s unique strengths and interests (things to utilize, such as favorite toys or topics) and challenges (things to avoid, such as excessive talking). It is the clinical experience of the authors that examiners with a genuine fondness for working with individuals with ASD and the knowledge, compassion, persistence, and resilience to go along with that passion tend to obtain the most consistently helpful information from clinical evaluations.

And finally, ASD not only impacts the affected individual, but it can have a profound effect on the individual’s family and on those who work with the individual at school or in the community (e.g., Schopler and Mesibov 1984). Indeed, social opportunities, effective communications, and the pursuit of varied interests can all be compromised in a family with a child with special needs. As a result of reallocating precious family resources, the family of a child with ASD can sometimes become more “autistic” itself with reduced social opportunities to attend church or invite neighbors over for dinner, for example, with little time or energy for couples to go on “dates” or to communicate one-on-one, or by eliminating or modifying leisure options (e.g., a family vacation to Disney World) because of challenges the child with ASD might face. Consequently, the various contexts in which the individual with ASD lives and functions need to be assessed and targeted with constructive suggestions. A supportive and collaborative relationship with families (and other care providers, schools, and community agencies) and a clear, honest, and sensitive presentation of the evaluation findings contribute to a better assessment. These factors together can also have the potentially therapeutic benefit of helping parents come to terms with their child’s diagnosis

in ways that help them obtain services, advocate for their child, and assist all family members to cope more effectively (e.g., Mesibov et al. 2005).

Specific Guidelines and Procedures in the Clinical Assessment of ASD

Several excellent resources have emerged in the past 15 years that outline specific guidelines and evidence-informed procedures for conducting clinical assessments of ASD. For students in training or those professionals interested in a refresher course, textbooks on clinical assessment that include a chapter on autistic disorder are available (e.g., Sattler and Hoge 2006). For those individuals or agencies wishing to establish their expertise in this area, specific practice parameters for what constitutes current best practice have been published (Filipek et al. 1999; Volkmar et al. 2014). For those wanting a comprehensive and concise overview of evidence-informed practices and empirically validated measures, well-written articles and books are easily accessible (Kroncke et al. 2016; Ozonoff et al. 2005). And for those looking for a comprehensive discussion of the relevant issues in the assessment of ASD, there are both earlier and recently published options (Goldstein et al. 2009; Schopler and Mesibov 1988). There is a good deal of consensus regarding current best practice, which will be summarized in this section. Interested readers will find additional detail by consulting these resources mentioned previously.

An *individualized assessment plan* is typically organized around (a) identified and latent concerns, (b) the methods used to gather relevant information, (c) the various contexts in which the individual functions, (d) perspectives from parents and multidisciplinary professionals, and (e) the immediate, intermediate, and long-term goals for the individual (see also Cohen 1976). These multidimensional assessments target the *whole* person, including multiple areas of functioning (e.g., academic, communication, and social) to determine relative strengths and weaknesses (e.g., Goldstein et al. 2009; Schopler and Mesibov 1988), thereby allowing for *strength-based* programming. In addition, *emerging skills* are assessed in order to generate treatment or

educational goals that are specific, concrete, and immediate (e.g., Hogan and Marcus 2009). Practically, predetermined assessment protocols and eligibility requirements of service agencies (e.g., Shea and Mesibov 2009), reimbursement schedules of funding sources, and the time, energy, and expertise of the clinician also factor in assessment planning decisions. The most common types of ASD assessments involve determining (a) whether or not an individual should be referred for a more thorough evaluation; (b) relevant diagnosis(es); (c) strengths and weaknesses in information processing, learning, and performance; and (d) the potential for the individual to live and work independently. Each type of assessment establishes an empirically informed best practice basis for addressing the presenting concerns, whether they are, for example, behavioral, academic, or legal. Each of these types of evaluations will be discussed briefly in the following sections. Clinical assessment to weigh the costs/benefits of specific medication trials, or other experimental and sometimes controversial treatments, is beyond the scope of the present discussion.

Screening Evaluation of ASD There are a number of very good publications that discuss relevant issues and available measures for screening young children for developmental delays, in general, and ASD, in particular (e.g., Barton et al. 2012; Filipek et al. 1999; Rogers 2001). Screening procedures can be categorized as somewhat less structured (e.g., interviews, observations, and play-based interactions) and rely on the clinical expertise of the professional, or they may involve a formal procedure requiring a standardized administration and be adapted to both trained and untrained informants. Screening procedures are oftentimes designed to be used by primary and secondary healthcare providers, and they are evaluated based upon how effectively (i.e., sensitivity) they identify children who should be referred to a secondary care agency for a broad assessment of developmental delays (first-level screening) or to a highly specialized tertiary care agency that has expertise in ASD (second-level screening). The Modified Checklist for Autism in Toddlers (M-CHAT), the Quantitative Checklist for Autism

in Toddler (Q-CHAT), the Social Communication Questionnaire (SCQ), the Pervasive Developmental Disorder Screening Test (PDDST), and the Screening Tool for Autism in Toddlers (STAT) are some of the most frequently cited checklists used to screen for ASD (e.g., Norris and Lecavalier 2010; Rogers 2001; Shea and Mesibov 2009). These measures target a range of observable behaviors (e.g., joint attention, responding to one's name, imaginative play, and repetitive behaviors) present or absent in young developing children that indicate a heightened risk for being diagnosed with ASD.

The need for reliable and effective screening procedures for ASD has received heightened attention recently due to the increasing incidence of ASD worldwide and the importance of effective early intervention programs for decreasing the short- and long-term adverse impact of the disorder (e.g., National Research Council 2001). Early screening and early diagnostic assessment are especially active areas of research currently, and there are a number of recently published references in this area (e.g., Barton et al. 2012; Chawarska et al. 2008; Norris and Lecavalier 2010).

Diagnostic Evaluation of ASD The purpose of the diagnostic assessment is to use valid and reliable methods to get meaningful information about how an individual functions and, as appropriate, to assign a diagnostic label to the individual. The diagnostic label signifies a kind and degree of abnormal behavior and development that characterize a subset of a given population. Diagnostic labels are helpful when they can be used to better manage the uncertainty surrounding the individual's behavior and prognosis, enhance communications about the individual, and facilitate access to available resources and effective treatments. Although ASD is generally considered to be a neurodevelopmental disorder, there are currently no biomedical tests or procedures upon which a diagnosis can be made. Information about an individual's early development and current behaviors gathered through interviews with parents and teachers and observations made during structured

and unstructured interactions with the individual form the basis for determining if the individual meets criteria for an ASD diagnosis. Familiarity with normal child development and the broad spectrum of developmental and psychiatric disorders are essential in determining the appropriate diagnosis(es). Inconsistencies in abilities and performance are, by definition, markers for ASD. Especially for the higher-functioning individual, overt symptoms are frequently *context specific*, and a thorough evaluation will gather information from a variety of settings (see also, e.g., Ozonoff et al. 2005). Autism-informed clinical interviews with parents and autism-informed systematic observations of clients during structured and unstructured interactions, when combined with a review of previous medical and educational records, constitute the core components of a diagnostic evaluation for ASD. Currently, the Autism Diagnostic Interview-Revised (ADI-R) and the Autism Diagnostic Observation Schedule (ADOS) (and, presumably, the recently released ADOS-2) are considered by many to reflect the highest standard of evidence-based practice for both clinical and research purposes (e.g., Ozonoff et al. 2005). A medical evaluation and intellectual, communication, and adaptive behavior testing are essential in ruling out other possible explanations or in ruling in comorbid conditions. Information from other cognitive (e.g., neuropsychological) and behavior assessments can also provide useful information that can help clarify a diagnosis (e.g., Goldstein et al. 2009; Ozonoff et al. 2005).

Psychoeducational Evaluation of ASD There is increasing evidence that the structure and function of the brain are different in individuals with ASD, but the precise and essential nature of the differences remains unclear. Even so, these suspected anomalies are presumed to account for the differences in how individuals with autism process sensory information, learn, reason, and perform daily activities. Understanding these unique patterns of information processing and behavior is the primary goal of the psychoeducational/neuropsychological assessment of ASD. As mentioned earlier, obtaining valid and reliable test results is no easy matter when working with

some individuals on the autism spectrum, but it is essential if meaningful goals are to be developed and if effective strategies are to be prescribed. A comprehensive psychoeducational evaluation, broadly speaking, may begin with an assessment of intellectual functioning, communication skills, academic abilities, and social and adaptive behaviors. More sophisticated assessments may target specific areas of cognitive and emotional functioning known to be relative strengths (e.g., rote memory, visual attention and visual/spatial reasoning, routinized learning and performance) or weaknesses (e.g., verbal abilities, novel problem-solving, integrated and applied skills) in individuals with ASD. Although some of these skills can be assessed in individuals with lower abilities, most of the recent developments in this area stem from work with higher-functioning individuals. Both formal and informal measures are being used to assess cognitive abilities such as executive functioning, perspective-taking, central coherence, cognitive flexibility, and social cognition and problem-solving (e.g., Corbett et al. 2009). Subtle language and communication skills assessed may include understanding figurative language and language concepts and pragmatic communication skills (Paul and Wilson 2009). Imaginative and interactive play skills and individual leisure activities are also typically assessed. The goal of a *diagnostic evaluation* is to determine how the individual is *like* others who share a particular diagnostic label. The goal of a *psychoeducational evaluation* is to determine how the individual is *different* than a generic group of individuals (i.e., individual differences). Individualized treatment and an individualized educational plan are only possible when the unique qualities of how the individual relates to him- or herself and to the surrounding environments are understood.

Vocational Evaluation of ASD Recent changes to state and federal guidelines regarding the education of special needs students have resulted in a renewed interest in developing assessment procedures that can facilitate planning for the transition to adulthood. For some individuals on the autism spectrum, this may involve paid or

voluntary work in the community, as well as semi-independent or independent living arrangements. A comprehensive vocational assessment may outline potential areas of employment or community involvement and the supports and strategies that can maximize independent functioning. Structured and meaningful activities, community inclusion, and greater levels of independence often result in the best possible outcome for adults with ASD. A comprehensive vocational assessment focuses not only on vocational skills and interests but on work habits, communication skills, the ability to adapt to different physical environments, and the necessary stress coping, social, and leisure skills that can help determine the level and the supports that will allow the individual to be successfully integrated into the community.

The recently revised TEACCH Transition Assessment Profile (TTAP), Second Edition (formerly known as the Adolescent and Adult Psychoeducational Profile [AAPEP]), is an example of a comprehensive vocational assessment for adolescents and adults with ASD. Although the strengths of its psychometric properties continue to be researched, this assessment tool is a combination of structured interview, observation, and informal assessment techniques designed to be utilized in the natural home, work, and community settings in which the individual lives. Vocational assessments of individuals with ASD seem to demand a greater degree of ecological validity than some of the other measures discussed in this article, and the TTAP appears to have been designed with this in mind.

Future Directions

Clinical assessment is a dynamic process that is shaped by human nature's indefatigable drive to transcend current limits (of knowledge and practice) and to do so in the most efficient way possible. The recent publication of the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V)*, represents the "next step" in our understanding of what constitutes a formal

diagnosis of ASD. As practitioners develop new tools and techniques for assessing individuals with ASD to accommodate the current paradigms and emerging technologies, cutting-edge researchers are already beginning to shape the field's future. Even though this future is difficult to see clearly, there are several trends that are likely to challenge the field in the coming years. For example, children are being evaluated for ASD at increasingly younger ages, some as young as 12 months. Concerned parents and professionals are looking for reliable assessment tools and procedures that can be used with these very young children. In addition, recent advances in identifying biomarkers of ASD during prenatal development and early childhood may eventually translate into clinical practice. Another area for future research involves highly individualized matching of treatment strategies to specific biological, neuropsychological, or behavioral indicators. Currently, there is an active effort to attempt to match children to treatment protocols based upon characteristics of the child (behavioral markers) to optimize outcomes (e.g., Schreibman et al. 2009). Clinical assessment may at some point in the near future work in conjunction with genetic testing and other medical procedures to determine diagnoses and recommended treatment protocols (including targeted genetic and psychotropic interventions). Assessment in the future may increasingly include more widespread use of teleconferencing and digital surveillance (e.g., Schutte et al. 2015). Computer-based assessment tools for caregivers and clients will likely become mainstream. Practitioners will need to stay alert to the trends, best practices, and ethics related to using technology for assessment and diagnosis. An area of future uncertainty involves changing public policy regarding reimbursement and health insurance reform. Redesigned health insurance policies may cover some assessment procedures but not others. This will likely continue to have an indirect impact on the thoroughness and overall quality of an assessment by setting reimbursement rates for services rendered. While practitioners are becoming more skilled at identifying ASD in even

younger children, a subset of older children and adults exists who are not identified until they are much older. As clinicians become more adept at assessing and diagnosing ASD, professionals need to consider the question of whether everyone who is quirky, rigid, different, or eccentric needs a diagnosis. Because of increased media coverage, an increasing number of referrals are being made to specialized clinics by family members and colleagues of individuals who are clearly unusual but who seem to function reasonably well in society without a diagnosis. Adults who learn about ASD through media or personal experience may wonder if they have a diagnosis on the spectrum. Clinicians will need to understand the diagnostic criteria being used, the strengths and limitations of the instruments available, and the presenting problem to best use their professional judgment in making decisions about further assessment.

See Also

- Academic Skills
- Autism Diagnostic Interview-Revised
- Autism Diagnostic Observation Schedule
- Childhood Autism Rating Scale
- Diagnosis and Classification
- Informal Assessment
- Medical Evaluation in Autism
- Observational Assessments
- Screening Measures
- Social Communication Questionnaire
- TEACCH Transition Assessment Profile (TTAP)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: Author.
- Barton, M. L., Dumont-Mathieu, T., & Fein, D. (2012). Screening young children for autism spectrum disorders in primary practice. *Journal of Autism and Developmental Disorders*, 42(6), 1165–1174. <https://doi.org/10.1007/s10803-011-1343-5>.

- Chawarska, K., Klin, A., & Volkmar, F. R. (2008). *Autism spectrum disorders in infants and toddlers: Diagnosis, assessment, and treatment*. New York: Guilford Press.
- Cohen, D. J. (1976). The diagnostic process in child psychiatry. *Psychiatric Annals*, 6, 29–56.
- Corbett, B. A., Carmean, V., & Fein, D. (2009). Assessment of neuropsychological functioning in autism spectrum disorders. In S. Goldstein, J. A. Naglieri, & S. Ozonoff (Eds.), *Assessment of autism spectrum disorders* (pp. 253–289). New York: Guilford Press.
- Feinstein, A. (2010). *Definitions, diagnosis, and assessment: A history of the tool*. Chichester: Wiley.
- Filipek, P. A., Accardo, P. J., Baranek, G. T., Cook, E. H., Jr., Dawson, G., Gordon, B., . . . , & Volkmar, F. R. (1999). The screening and diagnosis of autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 29(6), 439–484.
- Goldstein, S., Naglieri, J. A., & Ozonoff, S. (Eds.). (2009). *Assessment of autism spectrum disorders*. New York: Guilford Press.
- Hogan, K., & Marcus, L. M. (2009). From assessment to intervention. In S. Goldstein, J. A. Naglieri, & S. Ozonoff (Eds.), *Assessment of autism spectrum disorders* (pp. 318–338). New York: Guilford Press.
- Kanner, L. (1943). Affective disturbances of affective contact. *Nervous Child*, 2, 217–253.
- Klinger, L. G., O’Kelly, S. E., & Mussey, J. L. (2009). Assessment of intellectual functioning in autism spectrum disorders. In S. Goldstein, J. A. Naglieri, & S. Ozonoff (Eds.), *Assessment of autism spectrum disorders* (pp. 209–252). New York: Guilford Press.
- Kroncke, A. P., Willard, M., & Huckabee, H. (2016). *Assessment of autism spectrum disorder: Critical issues in clinical, forensic, and school settings*. Cham: Springer International Publishing.
- Mesibov, G., Shea, V., & Schopler, E. (2005). *The TEACCH approach to autism spectrum disorders*. New York: Kluwer Academic/Plenum Publishers.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Norris, M., & Lecavalier, L. (2010). Screening accuracy of level 2 autism spectrum disorder rating scales. *Autism*, 14(4), 263–284. <https://doi.org/10.1177/1362361309348071>.
- Ozonoff, S., Goodlin-Jones, B. L., & Solomon, M. (2005). Evidence-based assessment of autism spectrum disorders in children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, 34(3), 523–540. https://doi.org/10.1207/s15374424jccp3403_8.
- Paul, R., & Wilson, K. P. (2009). Assessing speech, language, and communication in autism spectrum disorders. In S. Goldstein, J. A. Naglieri, & S. Ozonoff (Eds.), *Assessment of autism spectrum disorders* (pp. 171–208). New York: Guilford Press.
- Rogers, S. J. (2001). Diagnosis of autism before the age of 3. In L. M. Glidden (Ed.), *International review of research in mental retardation: Autism* (pp. 1–31). San Diego: Academic Press.
- Sattler, J. M., & Hoge, R. D. (2006). *Assessment of children: Behavioral, social, and clinical foundations* (5th ed.). San Diego: Jerome M. Sattler, Publisher.
- Schopler, E., & Mesibov, G. B. (Eds.). (1984). *Current issues in autism: The effects of autism on the family*. New York: Plenum Press.
- Schopler, E., & Mesibov, G. B. (Eds.). (1988). *Current issues in autism: Diagnosis and assessment in autism*. New York: Plenum Press.
- Schreibman, L., Stahmer, A. C., Barlett, V. C., & Dufek, S. (2009). Brief report: Toward refinement of a predictive behavioral profile for treatment outcome in children with autism. *Research in Autism Spectrum Disorders*, 3(1), 163–172. <https://doi.org/10.1016/j.rasd.2008.04.008>.
- Schutte, J. L., McCue, M. P., Parmanto, B., McGonigle, J., Handen, B., Lewis, A., . . . , & Saptano, A. (2015). Usability and reliability of a remotely administered adult autism assessment, the autism diagnostic observation schedule (ADOS) module 4. *Telemed J E Health*, 21(3), 176–184. <https://doi.org/10.1089/tmj.2014.0011>.
- Shea, V., & Mesibov, G. B. (2009). Age-related issues in the assessment of autism spectrum disorders. In S. Goldstein, J. A. Naglieri, & S. Ozonoff (Eds.), *Assessment of autism spectrum disorders* (pp. 117–137). New York: Guilford Press.
- Volkmar, F. R., State, M., & Klin, A. (2009). Autism and autism spectrum disorders: Diagnostic issues for the coming decade. *Journal of Child Psychology and Psychiatry*, 50(1–2), 108–115.
- Volkmar, F., Siegel, M., Woodbury-Smith, M., King, B., McCracken, J., State, M., . . . , & Adolescent Psychiatry Committee on Quality, I. (2014). Practice parameter for the assessment and treatment of children and adolescents with autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 53(2), 237–257. <https://doi.org/10.1016/j.jaac.2013.10.013>.
- Wing, L., & Potter, D. (2009). The epidemiology of autism spectrum disorders: Is the prevalence rising? In S. Goldstein, J. A. Naglieri, & S. Ozonoff (Eds.), *Assessment of autism spectrum disorders* (pp. 18–54). New York: Guilford Press.
- World Health Organization. (1993). *The ICD-10 classification of mental and behavioral disorders: Diagnostic criteria for research*. Geneva: Author.

Clinical Depression

► Mood Disorders

Clinical Evaluation of Language Fundamentals: Preschool – Second Edition (CELF Preschool-2)

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Synonyms

[CELF Preschool – 2](#)

Description

The *Clinical Evaluation of Language Fundamentals: Preschool 2* (CELF-P 2) assesses all five domains of language: phonology, morphology, syntax, semantics, and pragmatics in children ages 3–6 years. This assessment includes both expressive and receptive modalities, as the child being assessed is expected to produce language and respond appropriately to instructions through spoken and unspoken modes of communication. The CELF-P 2 can be used to identify a language disorder, determine eligibility for services, and identify strengths and weaknesses, all through the use of performance-based tests that have strong relevance and relation to language and developmental milestones. The assessment is presented as a series of levels, 1–4. Level one is designed to determine whether or not a language disorder is present. Level two determines more information about the child's language including modalities used, content understood and expressed, and structures utilized. Level 3 evaluates the child's early classroom and literacy fundamental language, including preliteracy and phonological awareness ratings. Finally, level four determines the child's pragmatic skills in both an educational and home settings.

The CELF-P 2 items are displayed in the format of an easel book, with pictures for the child to reference, or respond to, either by

pointing to the correct option or verbally responding to the examiner's prompt. The time required to administer this assessment varies depending on how many subtests the evaluator chooses to administer. The manual states that it takes around 15–20 min to complete the three main subtests that make up the “core language score” (sentence structure, word structure, and expressive vocabulary). It is suggested that the evaluator begin with these three subtests, as these are used to determine whether a child needs further assessment. If the child's core language score is below the threshold of 85, the evaluator should then continue onto the remaining subtests for further evaluation. The CELF-P 2 materials include stimulus books 1 and 2, a record form, concept and following direction stimulus sheets, preliteracy rating scale, descriptive pragmatics profile, and the examiner's manual. The examiner's manual includes detailed instructions on how to give the assessment, subtests to use, how to score each, and interpret all of the scores.

The CELF-P 2 manual clearly states all aspects related to scoring for the clinician. Each subtest gives information on scoring including allowance of repetitions, discontinue rule also known as the ceiling, and how many points per correct/incorrect answer. The amount of trials ranges greatly between subtests, from 2 trials for Recalling Sentences, to 24 trials each for word structure and phonological awareness. The test then provides norm-referenced scaled scores for the following subtests as follows: sentence structure, word structure, expressive vocab, concepts and following directions, recalling sentences, basic concepts, and word classes. For each of the subtests that translate to scaled scores, the manual instructs the evaluator to take the raw score that has been calculated and then go to the age-appropriate table in Appendix B of the manual to find the corresponding scaled score. The CELF-P 2 scaled scores are based on a mean of 10 and a standard deviation of 3. As previously mentioned, the CELF-P 2 also provides evaluators with the option to do a shorter version, core language score. This shorter version uses three core subtests to assess the child's overall language abilities and

the obtained scaled scores assist in determining the need for further testing.

Historical Background

The first edition of the *Clinical Evaluation of Language Fundamentals: Preschool* (CELF-P) was published in 1992, authored by Elisabeth H. Wiig, Wayne Secord, and Eleanor Semel. The CELF-P was subsequently updated and built upon ultimately creating the CELF-P 2 (2nd edition) with the aim of being a more complete, adaptable, and multifaceted assessment tool for children ages 3–6:11. New developments with the second edition include a variety of changes, including improved psychometric data made up of stronger reliability and validity, and expansion of both the basal and the ceiling for greater diagnostic value within ages 3–4. Additional changes were included to add clarity and efficiency with administration. For example, the manual was revised to improve precision of the examiner directions, and directions are included in stimulus book 1. Lastly, the second edition also broadened the scope of the assessment itself through the inclusion of pragmatics, semantics, and morphosyntax among the test's subject matter. Overall, the improvements to the CELF-P 2 increase the assessment's value, overall application to relevant populations, and appeal to examiners in related fields.

Psychometric Data

The norm-referenced data used for the CELF-P 2 was derived from a sample of 800 children from 3 to 6 years of age. The standard sample elements for the CELF-P 2 are congruous with the demographic data of the 2000 US Census relating to geographical region, sex, race/ethnicity, and caregiver education level. In developing norms, standardization testing included an interruption rule in four subtest areas that were longer than normal (concepts & following directions, recalling sentences, word classes, and recalling sentences in context); allowing as

much item-specific data to be collected as possible without having to administer items beyond the capacity of the child. Using the first edition of CELF-P 2 as well as CELF-4, functions for each subtest were created based on consistency with expectations and growth pattern curves observed.

CELF-P 2 is both reliable and valid. Test-retest reliability for the subtests and composite scores in all ages ranged from 0.78 to 0.94. Internal consistency reliability coefficients ranged from 0.79 to 0.97, thus, indicating that the scores across subtests as acceptable and composite scores as excellent. The prevalence of validity is presented based on assessment content, response process, internal structure, and intercorrelational studies. Correlations between subtests and composite scores were moderate, and additional validation evidence was documented through comparison with other tests of language disorders in children including the CELF preschool, CELF-4, and the PLS-4. These correlations were reported moderate to high which is considered to be good.

Clinical Uses

The CELF-P 2 can be used in a variety of clinical settings to both assess and diagnose and follow-up with children (ages 3–6) who present with language deficits. The CELF-P 2 provides the option to score the child with the “core language score”; thus, the test can be administered effectively and efficiently within a school or daycare setting, in about 15 min. The use of this assessment provides flexibility, allowing the clinician to only administer subtests relevant to the needs of the client. The assessment manual includes a section in which evidence-based studies are referenced. These studies examine the clinical use of the CELF-P 2 to assess special populations such as autism, specific language disorders, and children who are hard of hearing. The assessment manual notes that depending on the type of impairment (motor, sensory, cognitive), the testing environment, and test itself may be adapted to fit the needs of that specific student. Some adaptations provided by the manual include providing

additional cues, increasing the number of demonstrations and trial items, or skipping items that may not be appropriate for that specific child. The manual clarifies that if adaptations are used, the raw score cannot be translated to a scaled score, and the administrator must score the test solely based on the raw scores of the child.

The CELF-P 2 takes into account cultural differences that can affect the outcome of the test. The assessment stresses the importance of evaluating each child in light of their dialect, culture, community, and ethnicity. To avoid cultural bias, the assessment created a panel of seven licensed speech-language pathologists who were known for their expertise in multicultural issues. The team represented expertise in Hispanic, African American, Native American, and Asian cultures, as well as in men's and women's issues. They were tasked with examining the assessment for cultural, ethnic, gender, socioeconomic, and regional bias. Each member of the panel reviewed the subtest tasks, administration procedures, possible child responses, and stimulus pictures for the proposed CELF-P 2. Their feedback was taken into account and discussions about potential issues took place. To ensure that professionals administering the test consider cultural differences, the testing manual includes a section with peer-reviewed data on how culture may affect assessment, as well as word formation rules for dialectal differences. These charts include possible word formation responses for many of the most used dialects. The test also comes in a Spanish form – CELF Preschool-2 Spanish.

See Also

- [Index of Productive Syntax \(IPSyn\)](#)
- [Pragmatics](#)
- [Semantic Memory](#)
- [Speech Morphology](#)

References and Reading

Paslawski, T. (2005). Clinical evaluation of language fundamentals, fourth edition (CELF-4): A review.

Canadian Journal of School Psychology, 20(1–2), 129–134.

Shurman, J., & Leone, D. (2015). Standardized versus naturalized: An evaluation of child morphological and syntactic assessments. *International Journal of Undergraduate Research and Creative Activities*, 7(1), 6.

Wiig, E. H., Secord, W. A., & Semel, E. M. (1992). *Clinical evaluation of language fundamentals – Preschool*. San Antonio: The Psychological Corporation.

Wiig, E., Secord, W. A., & Semel, E. M. (2004). *Clinical evaluation of language fundamentals – Preschool* (2nd ed.). San Antonio: Harcourt Assessment.

Clinical Linguistic and Auditory Milestone Scale

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Synonyms

[Capute scales \(along with cognitive adaptive test\); CAT/CLAMS; CLAMS](#)

Description

The Clinical Linguistic and Auditory Milestone Scale is a 43-item parent questionnaire that is one of two parts of the 100-item Capute Scales. This measure is administered in conjunction with the Cognitive Adaptive Test (CAT), a 57-item assessment of visual-motor functioning. The test uses standardized methods for obtaining information from parent report and from direct interaction between the examiner and the child. The CLAMS utilizes parent report and focuses on expressive and receptive language skills of children up to 3 years of age.

Historical Background

The Clinical Linguistic and Auditory Milestone Scale (CLAMS) was initially developed by

Dr. Arnold J. Capute to screen for language delays in young children between birth and 3 years of age. The initial version of the scale was developed in 1973 (Capute and Biehl 1973), with revisions in 1978 (Capute and Accardo 1978). It was originally developed as a brief screening measure for pediatricians within an office setting. The measure was first normed in 1986 (Capute et al. 1986). A visual-motor scale, the Cognitive Adaptive Test (CAT), was subsequently added to the assessment protocol in order to assist physicians in the differential diagnosis of isolated language difficulties as opposed to global cognitive impairments. This assessment was commonly referred to as the CAT/CLAMS. The CAT/CLAMS was revised and renamed “The Capute Scales,” with the most recent standardization procedures completed in 2000–2001.

Psychometric Data

Various versions of the CLAMS and Capute Scales have been revised and standardized over a period of 30 years. Most recently, in 2004, the Capute Scales were standardized on a multisite sample of 1055 typically developing children that were balanced for sex, age, and race (Visintainer et al. 2004). Results indicated that both measures adequately estimated age-level performance. It is noteworthy that historically the CLAMS has correlated highly with performance on the Bayley Scales of Infant Development (Capute et al. 1986).

Clinical Uses

The Capute Scales were designed as a screening measure for experienced physicians, speech-language pathologists, and occupational therapists in order to allow them to distinguish between global developmental delay and receptive/expressive language problems. It consists of 100 items that are completed in-office using parent report and direct elicitation. Administration time is estimated to be from 6 to 20 min to complete both “streams,”

the Cognitive Adaptive Test (CAT) and the Clinical Linguistic and Auditory Milestone Scale (CLAMS). Forms are available in multiple languages including English, Spanish, and Russian.

References and Reading

- Accardo, P., & Capute, A. J. (2005). *The Capute scales: Cognitive adaptive test/clinical linguistic and auditory milestone scale*. Baltimore: Paul H. Brookes Publishing.
- Accardo, P., Leppert, M., Lipkin, P., & Rogers, B. (2005). *The Capute scales. Biomedical and social perspectives*. Timonium: York Press.
- Bayley, N. (2006). *Bayley scales of infant and toddler development* (3rd ed.). San Antonio: Harcourt Assessment.
- Beilcher, H., Gittlesohn, A., Capute, A., & Allen, M. (1997). Using the clinical linguistic and auditory milestone scale for developmental screening in high-risk preterm infants. *Clinical Pediatrics*, 36, C635–C664.
- Capute, A. J., & Accardo, P. J. (1978). Linguistic and auditory milestones during the first two years of life: A language inventory for the practitioner. *Clinical Pediatrics*, 17, 847–853.
- Capute, A. J., & Biehl, R. F. (1973). Functional developmental evaluation. Prerequisite to habilitation. *Pediatric Clinics of North America*, 20, 3–26.
- Capute, A., Palmer, F., Shapiro, B., Wachtel, R., Schmidt, S., & Ross, A. (1986). Clinical linguistic and auditory milestone scale: Prediction of cognition in infancy. *Developmental Medicine and Child Neurology*, 28, 762–771.
- Hoon, A. H., Pulsifer, M. B., Gopalan, R., Palmer, F. B., & Capute, A. (1993). Clinical adaptive test/clinical linguistic auditory milestone scale in early cognitive assessment. *Journal of Pediatrics*, 123, S1–S8.
- Kube, D. A., Wilson, W. M., Petersen, M. C., & Palmer, F. B. (2000). CAT/CLAMS: Its use in detecting early childhood cognitive impairment. *Pediatric Neurology*, 23, 208–215.
- Leppert, M., Shank, T., Shapiro, B., & Capute, A. (1998). The Capute scales: CAT/CLAMS – A pediatric assessment tool for the early detection of mental retardation and communicative disorders. *Mental Retardation and Developmental Disabilities Research Reviews*, 4, 14–19.
- Visintainer, P., Leppert, M., Bennett, A., & Accardo, P. (2004). The standardization of the Capute scales: Methods and results. *Journal of Child Neurology*, 19, 967–972.
- Wachtel, R., Shapiro, B., Palmer, F., Allen, M., & Capute, A. (1994). CAT/CLAMS: A tool for the pediatric evaluation of infants and young children with developmental delay. *Clinical Pediatrics*, 33, 410–415.

Clinical Psychology

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Definition

Clinical psychology focuses on the diagnosis and treatment of mental illness, abnormal behavior, and psychological disorders in individuals. In 1907, American psychologist Lightner Witmer first coined the term and defined clinical psychology as the study of individuals through observational or experimental methods to promote change in individuals.

A clinical psychologist has a doctorate (Ph.D. or Psy.D.) in clinical psychology, whereas a psychiatrist has a medical degree (M.D.). A clinical psychologist must be licensed by a state licensing board in order to conduct clinical work as a “clinical psychologist.” A clinical psychologist can be involved in the assessment or treatment of autism spectrum disorder (ASD). In addition, a number of clinical psychologists conduct research on ASD.

See Also

- ▶ [Child Psychotherapy](#)
- ▶ [Clinical Social Worker](#)
- ▶ [Psychiatrist](#)
- ▶ [Psychologist](#)
- ▶ [School Psychologist](#)

References and Reading

- Compas, B., & Gotlib, I. (2002). *Introduction to clinical psychology*. New York: McGraw-Hill Higher Education.
- Witmer, L. (1907). Clinical psychology. *Psychological Clinic*, 1, 1–9.

Clinical Significance

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Definition

Statistical vs. Clinical Significance: Statistical significance means simply that a result in a given comparison occurs beyond what one would expect by chance alone. Here, and by accepted convention, a comparative result is declared statistically significant if it occurs at or beyond the 5% level. By simple subtraction, this means that there is a 95% possibility that the result did not occur by chance.

The caveat here, and one that many journal editors and even some unenlightened biostatisticians fail to grasp, is that given a large enough number of cases or N, a given comparative result will inevitably occur *beyond chance*, at the 5% level of statistical significance.

In order to guard against this so-called big N phenomenon, enlightened biostatisticians have devised guidelines for defining a result as having reached a level of clinical, as well as statistical significance.

A concrete example, albeit an apocryphal one, can be derived easily from the field of autism spectrum research.

Suppose an inexperienced clinician on a scale of 0–100% agrees with the diagnosis of childhood autism, in 500 cases at 10%. With such a large N of cases, the result turns out to be statistically significant with a chance probability at the scientifically acceptable level of 5%.

Well, any self-respecting autism expert would tell you that 10% chance-corrected agreement, from a clinical perspective, is poor or trivial.

Clinical significance to the rescue comes in the form of a set of clinical criteria developed by Cicchetti and Sparrow (1981) by which:

Size of Reliability Coefficient	Clinical Significance
<0.40	Poor
0.40–0.59	Fair
0.60–0.74	Good
0.75–1.00	Excellent

Earlier, in 1977, Landis & Koch provided the following guidelines for also interpreting the level of clinical or practical significance of chance-corrected inter-examiner agreement levels:

Size of Reliability Coefficient	Level of Clinical Significance
Below 0	Poor
0.0–0.20	Slight
0.21–0.40	Fair
0.41–0.60	Moderate
0.61–0.80	Substantial
0.81–1.00	Almost Perfect

Similarly, Fleiss (1981) and Fleiss et al. (2003) also suggested conceptually similar guidelines by which:

Size of Reliability Coefficient	Level of Clinical Significance
<0.40	Poor
0.40–0.75	Fair to Good agreement
Above 0.75	Excellent agreement

Finally, conceptually similar guidelines ranging from, say, Poor to Excellent or Trivial to Very Large, have been developed for correlations of various types, and for judging the internal consistency level of assessment instruments (e.g., Cicchetti 1994).

See Also

► [Statistical Significance](#)

References and Reading

Cicchetti, D. V. (1994). Guidelines, criteria, and rules of thumb for evaluating normed and standardized a assessment instruments. *Psychological Assessment*, 6, 284–290.

Cicchetti, D. V., & Sparrow, S. S. (1981). Developing criteria for establishing interrater reliability of specific items: Applications to assessment of adaptive behavior. *American Journal of Mental Deficiency*, 86, 127–137.

Fleiss, J. L. (1981). *Statistical methods for rates and proportions*. New York: Wiley.

Fleiss, J. L., Levin, B., & Paik, M. C. (2003). *Statistical methods for rates and proportions* (3rd ed.). New York: Wiley.

Landis, J. R., & Koch, G. G. (1977). The measurement of observer agreement for categorical data. *Biometrics*, 33, 159–174.

Clinical Social Worker

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Synonyms

[Clinician](#); [Mental health practitioner](#); [Therapist](#)

Definition

Social work is a broad field with a wide range of variation in areas of professional practice. Clinical social workers are social work professionals who provide direct services to client populations. This is in contrast to non-clinically based social workers who may practice in the realm of social administration or who may be more focused on broad-based advocacy and policy reform at the national, state, and community levels. Clinical social workers strive to improve the quality of life and advocate for clients on a more individual level. The term clinical social worker is most closely associated with social workers who provide direct mental health and behavioral health services. These therapeutic services are provided in a variety of settings including hospitals, community mental health clinics, schools, residential facilities, and correctional facilities. Clinical

social workers provide services for a variety of populations that cover the entire life span including children, adolescents, adults, and the elderly and intervene by working directly with individuals, couples, families, and groups (Hepworth et al. 2006, p. 25).

Clinical social workers are knowledgeable on a wide variety of mental health disorders and are well versed and qualified to conduct mental health assessments, render diagnoses, develop treatment plans, and provide short- and long-term treatment. In comparison to other trained mental health professionals, the social work approach gives special attention to the client's stated goals and incorporates these goals into treatment while also taking into account client's functioning within his or her environment. Additional social work values include high regard for dignity and worth of client, upholding human rights, and respect for diversity (Hepworth et al. 2006, pp. 8–11).

Clinical social workers are the largest group of mental health providers in the United States (Manderscheid and Henderson 2001). As such, clinical social workers are often direct service providers to children and adults with autism spectrum disorders (ASD). Due to being employed in a variety of settings, social workers are often among the first healthcare providers to identify signs and symptoms of developmental delays in children. Additionally, multi-disciplinary ASD assessment team models may include a clinical social worker. Following an ASD diagnosis, clinically trained social workers provide support and education to families, assist in implementing behavioral interventions, and link clients to additional services and resources. Social workers also play a key role as advocates for clients with ASD within a variety of community systems.

Professional social workers typically have a Master of Social Work (MSW) degree. Most MSW programs are accredited by the Council on Social Work Education (CSWE) and require a substantial number of direct practice hours as an intern under close supervision. All social workers must complete a significant number of supervised direct practice hours beyond their degree program

to qualify for advanced licensure. Distinction of advanced practice is commonly noted as Licensed Clinical Social Worker (LCSW) or Licensed Independent Social Worker (LISW). All professional social workers are trained and expected to practice within the Social Work Code of Ethics as established by the National Association of Social Workers (NASW).

See Also

► [Psychiatrist](#)

References and Reading

- Hepworth, D. H., Larsen, J., Rooney, R. H., Rooney, G. D., & Strom-Gottfried, K. (2006). *Direct social work practice: Theory and skills* (7th ed.). Belmont: Thomson.
- National Association of Social Workers. <http://www.naswdc.org/>
- West, J., Kohout, J., Pion, G. M., Wicherski, M. M., Vandivort-Warren, R. E., & Palmiter, M. L. (2001). Mental health practitioners and trainees. In R. W. Manderscheid & M. J. Henderson (Eds.), *Mental health, United States 2000* (pp. 279–315). Washington, DC: Department of Health and Human Services, US Govt. Print.

Clinician

► [Clinical Social Worker](#)
 ► [Psychologist](#)

Clock Drawing

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Synonyms

[CDT](#); [Clock-drawing test](#)

Definition

The clock drawing test (CDT) is a neuropsychological assessment tool completed with pencil-and-paper that examines executive functioning, motor planning, and visuospatial skills. The test typically consists of two phases: command and copy. In the command phase, subjects are asked to draw the face of a clock and add hands to indicate a specific time (usually 10 past 11). In the copy phase, subjects are asked to copy a pre-drawn clock. Performance on the test is assessed by determining whether the hand-drawn clock meets certain criteria. Administrators may score the clock according to the following: number inclusions and location, spacing, organization, and correctness of hand position. Although the above instructions and scaled scoring system are commonly used, there is no standardized method of administration or scoring. Performance errors in the CDT are indicative of impairment in executive functioning.

This test is used in neurological and psychiatric patient populations, most commonly completed with at-risk dementia and Alzheimer's patients. It is sometimes used as a part of a neuropsychological assessment for children with Autism Spectrum Disorder.

See Also

- [Executive Function \(EF\)](#)
- [Motor Planning](#)

References and Reading

- Freedman, M., Leach, L., Kaplan, E., Winocur, G., Shulman, K. I., & Delis, D. C. (1994). *Clock drawing: A neuropsychological analysis*. Oxford: Oxford University Press.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). Oxford: Oxford University Press.
- Sunderland, T., et al. (1989). Clock drawing in Alzheimer's disease. A novel measure of dementia severity. *Journal of American Geriatric Society*, 37(8), 725–729. PMID: 2754157.

Clock-Drawing Test

- [Clock Drawing](#)

Clomipramine

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Synonyms

3-Chloro-5-[3-(dimethylamino)propyl]-10,11-dihydro-5H-dibenz[*b,f*]azepine monohydrochloride; Anafranil; Clomipramine (CMI) hydrochloride

Indications

Clomipramine is a tricyclic antidepressant (TCA). TCAs block the reuptake of serotonin and norepinephrine in the neuronal synapse, resulting in increased concentrations of both neurotransmitters. This is the mechanism by which it is thought that TCAs exert their therapeutic effects. In the United States, clomipramine is FDA-approved for the treatment of obsessive-compulsive disorder (OCD) in children, adolescents, and adults. Behind selective serotonin reuptake inhibitors (SSRIs), clomipramine is among the drugs of

choice for the treatment of OCD (Stahl 2008, 2009). It is also often prescribed for depression (including severe and treatment-resistant depression), cataplexy syndrome, anxiety, insomnia, and neuropathic or chronic pain, although it is not FDA-approved for any of these disorders (Stahl 2009). Unfortunately, its adverse effect profile has limited its use in many patients.

Mechanisms of Action

The exact mechanisms of action are unknown for the tricyclic antidepressants (TCAs) including clomipramine. However, likely mechanisms of action are suggested from the binding activity and receptor affinities of the active compounds of these drugs and from what is known about the pathophysiology of the disorder being treated. Dysregulation of serotonin and dopamine or both is thought to be implicated in OCD. Clomipramine is thought to alleviate obsessions and compulsions in OCD through its inhibition of the reuptake of serotonin in the brain. Its strongest neurological action is its activity as a serotonin reuptake inhibitor (SRI), and it has been described as being “a relatively selective [SRI]” (Andreasen and Black 2001a, “Somatic Treatments,” p. 721).

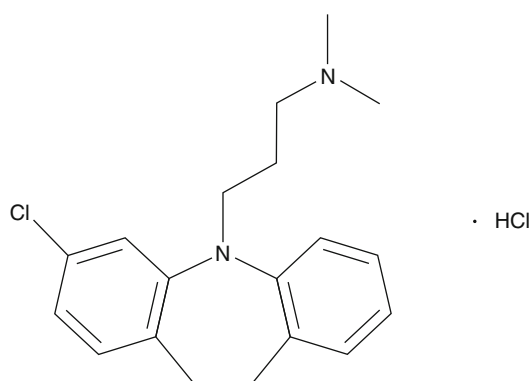
The SRI action of TCAs causes an increase in the amount of synaptic serotonin. Before the neurons are desensitized to the drug, this increase in synaptic serotonin is counteracted by its stimulation of the presynaptic serotonin autoreceptor which inhibits the further release of serotonin into the synapse. After about 10–14 days of drug treatment, this autoreceptor is desensitized, and the amount of synaptic serotonin increases. TCAs also increase the activity of postsynaptic serotonin_{1A} receptors and downregulate serotonin₂ receptors further augmenting the effects of the serotonin.

The active metabolite of clomipramine, desmethylclomipramine, is not an SRI but inhibits the reuptake of norepinephrine. This norepinephrine reuptake inhibition is thought to increase the effects of norepinephrine by increasing the amount of norepinephrine in the synapse by blocking the reuptake of norepinephrine. Before

the neurons are desensitized to the drug, this increase in synaptic norepinephrine is counteracted by its stimulation of the presynaptic α_2 -adrenergic autoreceptor which inhibits the further release of norepinephrine into the synapse. After this autoreceptor is desensitized, the amount of synaptic norepinephrine increases. Although the postsynaptic norepinephrine receptor is down-regulated by the increase in the amount of synaptic norepinephrine, this effect seems less significant than the increase in synaptic norepinephrine. However, no known connection exists between the inhibition of the reuptake of norepinephrine by desmethylclomipramine and the treatment of OCD.

Specific Compounds and Properties

Clomipramine has the International Union of Pure and Applied Chemistry (IUPAC) name 3-chloro-5-[3-(dimethylamino)propyl]-10,11-dihydro-5-*H*-dibenz[*b,f*]azepine monohydrochloride and the chemical formula $C_{19}H_{23}ClN_2 \cdot HCl$. The chemical structure of clomipramine only differs from that of the TCA imipramine in that clomipramine has an additional chloride atom. Pure clomipramine hydrochloride is a solid, white crystalline powder at room temperature and is soluble in the polar solvents water, methanol, and methylene chloride but not in the non-polar solvents ethyl ether and hexanes (Fig. 1).

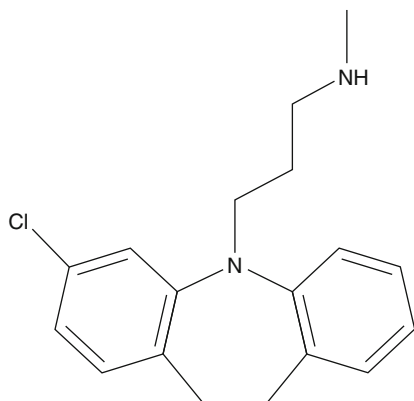


Clomipramine, Fig. 1 Chemical structure of clomipramine

Clomipramine is metabolized by the liver via undergoing N-demethylation of clomipramine to the active metabolite desmethylclomipramine and subsequently by N-oxidation and aromatic hydroxylation. These processes are catalyzed by the enzymes CYP 1A2, 2C19, and 3A3/4, and CYP 2D6 (clomipramine and desmethylclomipramine), respectively. Aromatic hydroxylation is important for the elimination of the drug in the body.

In plasma, clomipramine has a half-life of 15–60 h, a clearance of 20–120 Liters per hour (L/h), and a therapeutic dose range of 150–300 mg per day (mg/day). This drug has a therapeutic plasma level of greater than 150 nanograms (ng) of clomipramine and desmethylclomipramine per milliliter (mL). The main pharmacological action of the TCAs with tertiary amines on their side chains, including clomipramine, is to inhibit the reuptake of serotonin, although it also downregulates serotonin receptors. Clomipramine is the strongest SRI of the TCAs. TCAs with a tertiary amine structure on the side chain of the molecule, such as is the case with clomipramine, have shown less tolerability than those with secondary amines on the side chains (Fig. 2).

Desmethylclomipramine is metabolized by the liver, leading to the elimination of the drug in the body. The main pharmacological action of the TCAs with secondary amines on their side chains, including desmethylclomipramine, is to inhibit the reuptake of norepinephrine.



Clomipramine, Fig. 2 Chemical structure of desmethylclomipramine, active metabolite of clomipramine

Clinical Use (Including Side Effects)

The brand-name drug Anafranil has been FDA-approved for the treatment of obsessions and compulsions in individuals with OCD since 1989. Its active ingredient clomipramine is currently used for the treatment of depression in Europe. Clomipramine has also been shown to be effective in studies to treat hyperactivity in children with autistic disorder and attention deficit hyperactivity disorder (ADHD) and to treat the symptoms of panic disorder, Tourette's syndrome, cataplexy, and body dysmorphic disorder. This drug has also been found to be effective in preventing relapse of anorexia nervosa. Additionally, preliminary studies of the use of TCAs such as clomipramine for the treatment of trichotillomania have suggested at least some efficacy regarding reducing hair-pulling behavior, but controlled studies have yet to be undertaken. Reports have indicated that clomipramine may also be used to treat depersonalization disorder. Clomipramine may be used to treat self-injurious and stereotypic movements in stereotypic movement disorder.

Clomipramine is not currently approved for the treatment of autism or other autism spectrum disorders (ASDs), but researchers have studied its effectiveness in treating symptoms associated with ASDs, particularly autistic disorder. Gordon et al. (1992) found that clomipramine was more efficacious than desipramine and placebo on ratings of autism and anger, as well as repetitive or compulsive behaviors, in children with autism aged 6–18 years. Gordon et al. (1993) found that clomipramine was more efficacious than desipramine and placebo in children with autism with regard to stereotypies, anger, and compulsive, ritualized behavior. It was also superior to placebo in managing hyperactivity. In an open-label trial looking in adults with ASDs, Brodtkin et al. (1997) found that clomipramine may be effective at reducing repetitive thoughts and actions and aggressive behavior and for improving eye contact and verbal responsiveness. In another open-label trial looking at hospitalized children aged 3.5–8.7 years with autistic disorder, Sanchez et al. (1996) found clomipramine to not be therapeutic and posited that young autistic children

may be more prone to experiencing unwanted side effects compared to older patients. Remington et al. (2001) compared clomipramine to haloperidol and placebo in patients with autistic disorder aged 10–36 years and found that clomipramine was comparable to haloperidol in terms of improvement in maladaptive symptoms. Haloperidol was better tolerated than clomipramine. Hazell (2007) reviewed the literature looking at available treatments for attention deficit hyperactivity disorder (ADHD) symptoms in autistic disorder and found clomipramine to be of unlikely benefit.

A typical starting dose in adults is 25–75 mg per day (mg/day) of clomipramine. The dose is titrated up by 75 mg/day each week to the therapeutic dose range of 150–300 mg/day. In children, demethylation of clomipramine to desmethylclomipramine is faster than in adults; as a result, children require higher doses of medication per kilogram (kg) of body weight than do adults since desmethylclomipramine lacks the serotonin reuptake activity of clomipramine. An effective dose for children is generally 2.5–3.5 mg clomipramine/kg body weight. When puberty begins in a child taking clomipramine, the dose can be decreased by up to half of the previous dose. To discontinue a TCA, the drug must be tapered. Clomipramine can be tapered by 25 mg every 2–3 days. Relapse may occur upon discontinuation. The abrupt discontinuation of a dose within the therapeutic range of a TCA may induce cholinergic rebound syndrome, and cases have been reported of rebound manic or hypomanic episodes upon abrupt discontinuation of a TCA.

TCAs become toxic at concentrations greater than 450 ng/mL. Overdose may result in any of the following: agitation, confusion, convulsions, hypotension, tachycardia, cardiac conduction delays, anticholinergic blockade, bowel and bladder paralysis, disturbed temperature regulation, mydriasis (abnormal pupil dilation), and delirium. A TCA overdose may induce a coma, sometimes with shock and respiratory depression, which typically lasts less than 1 day. Patients at risk for making a suicide attempt should be given weekly, nonrefillable prescriptions of their TCAs. Risks include suicidal ideation or increased depression

in individuals with major depressive disorder (MDD), decreased blood pressure, and tachycardia. Adverse effects may include seizure, tremor, sedation, fatigue, dry mouth, dizziness, somnolence, constipation, nausea, increased sweating, impairment of memory, delirium, cardiac arrhythmias, cardiac arrest, headache, abnormal vision, anorexia, dyspepsia, insomnia, orthostasis, sexual dysfunction, weight gain, and anticholinergic effects.

See Also

- Anticholinergic
- Antidepressant Medications
- Desipramine
- Norepinephrine
- Obsessive-Compulsive Disorder (OCD)
- Repetitive Behavior
- Serotonin
- Serotonin Reuptake Inhibitors (SRIs)
- Stereotypic Behavior

References and Reading

- 3-Chloro-10,11-dihydro-N-methyl-5H-dibenz(b,f)azepine-5-propanamine. (n.d.). ChemSpider Wiki. Retrieved from <http://www.chemspider.com/RecordView.aspx?rid=de5fed0d-ac0b-46c1-a74c-6c89dd040665>
- Aman, M. G., & Langworthy, K. S. (2000). Pharmacotherapy for hyperactivity in children with autism and other pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 30, 451–459.
- Anafranil. (n.d.). ChemSpider Wiki. Retrieved from <http://www.chemspider.com/RecordView.aspx?rid=8ccb3154-90d1-41a6-8dec-d574ae7d6bc1>
- Andreasen, N. C., & Black, D. W. (2001a). Somatic treatments. In *Introductory textbook of psychiatry* (3rd ed., pp. 709–759). Washington, DC: American Psychiatric Publishing.
- Andreasen, N. C., & Black, D. W. (2001b). Dissociative disorders. In *Introductory textbook of psychiatry* (3rd ed., pp. 389–401). Washington, DC: American Psychiatric Publishing.
- Brodin, E. S., McDougle, C. J., et al. (1997). Clomipramine in adults with pervasive developmental disorders: A prospective open-label investigation. *Journal of Child and Adolescent Psychopharmacology*, 7(2), 109–121.
- Gordon, C. T., Rapoport, J. L., et al. (1992). Differential response of seven subjects with autistic disorder to

- clomipramine and desipramine. *The American Journal of Psychiatry*, 149(3), 363–366.
- Gordon, C. T., State, R. C., et al. (1993). A double-blind comparison of clomipramine, desipramine, and placebo in the treatment of autistic disorder. *Archives of General Psychiatry*, 50(6), 441–447.
- Hazell, P. (2007). Drug therapy for attention-deficit/hyperactivity disorder-like symptoms in autistic disorder. *Journal of Paediatrics and Child Health*, 43(1–2), 19–24.
- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001a). Treatment with antidepressants. In *Principles and practice of psychopharmacotherapy* (3rd ed., pp. 215–325). Philadelphia: Lippincott Williams & Wilkins.
- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001b). Assessment and treatment of special populations. In *Principles and practice of psychopharmacotherapy* (3rd ed., pp. 559–639). Philadelphia: Lippincott Williams & Wilkins.
- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001c). Assessment and treatment of other disorders. In *Principles and practice of psychopharmacotherapy* (3rd ed., pp. 523–558). Philadelphia: Lippincott Williams & Wilkins.
- Kelly, M. W., & Myers, C. W. (1990). Clomipramine: A tricyclic antidepressant effective in obsessive compulsive disorder. *The Annals of Pharmacotherapy*, 24, 739–744.
- Martin, A., & Volkmar, F. R. (2007). *Lewis's child and adolescent psychiatry: A comprehensive textbook* (4th ed.). Philadelphia: Lippincott Williams & Wilkins.
- Nelson, J. C. (2006). Tricyclic and tetracyclic drugs. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 5–29). Washington, DC: American Psychiatric Publishing.
- Pigott, T. A., & Seay, S. M. (1999). A review of the efficacy of selective serotonin reuptake inhibitors in obsessive-compulsive disorder. *The Journal of Clinical Psychiatry*, 60, 101–106.
- Remington, G., Sloman, L., et al. (2001). Clomipramine versus haloperidol in the treatment of autistic disorder: A double-blind, placebo-controlled, crossover study. *Journal of Clinical Psychopharmacology*, 21(4), 440–444.
- Rosenbaum, J. F., & Tollefson, G. D. (2006). Fluoxetine. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 31–46). Washington, DC: American Psychiatric Publishing.
- Sadock, B. J., & Sadock, V. A. (2005). *Kaplan and Sadock's pocket handbook of clinical psychiatry* (4th ed.). Philadelphia: Lippincott Williams & Wilkins.
- Sanchez, L. E., Campbell, M., et al. (1996). A pilot study of clomipramine in young autistic children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35(4), 537–544.
- Stahl, S. M. (2000). *Essential psychopharmacology: Neuroscientific basis and clinical applications*. Cambridge, NY: Cambridge University Press.
- Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd ed., p. 770). New York: Cambridge University Press.
- Stahl, S. M. (2009). *Stahl's essential psychopharmacology: The prescriber's guide* (3rd ed., pp. 89–95). New Delhi: Cambridge University Press.
- U.S. Food and Drug Administration. (2011). *Drugs@FDA*. Retrieved from <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm>
- Wagner, K. D. (2006). Treatment of childhood and adolescent disorders. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 181–197). Washington, DC: American Psychiatric Publishing.

Clomipramine (CMI) Hydrochloride

► Clomipramine

Clonazepam: Definition

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Synonyms

Clonotril; Klonopin; Linotril; Rivotril

Definition

Clonazepam is a benzodiazepine and a positive allosteric modulator of the GABA_A receptor. Instead of opening the GABA_A receptor chloride channel in the absence of GABA binding, benzodiazepines, like clonazepam, accentuates GABA signaling upon GABA binding to the GABA_A receptor. GABA, or gamma-aminobutyric acid,

is the primary inhibitory neurotransmitter in the mature mammalian CNS, with glutamate being the primary excitatory neurotransmitter. The excitatory/inhibitory neuron imbalance hypothesis of the underlying neurobiology of autism is a highly prominent and well-evidenced one. ASD patients frequently show increased levels of glutamate and excitatory signaling. So, correspondingly increasing levels of GABA, the inhibitory neurotransmitter, should have the potential to reverse some ASD symptoms. However, while the premise of using benzodiazepines is theoretically well-founded, there is little to no evidence of their actual effectiveness in human ASD patients. Clonazepam is able to reverse autistic-like symptoms that accompany Dravet syndrome, a rare epilepsy disorder, in a mouse model. Currently, clonazepam is used to treat panic disorder, anxiety, and seizures. Side effects include sedation, dizziness, loss of orientation, sleep disturbances, and weakness.

See Also

► [Benzodiazepine](#)

References and Reading

- Belmonte, M. K., Allen, G., Beckel-Mitchener, A., Boulanger, L. M., Carper, R. A., & Webb, S. J. (2004a). Autism and abnormal development of brain connectivity. *The Journal of Neuroscience*, 24(42), 9228–9231.
- Belmonte, M. K., Cook, E. H., Anderson, G. M., Rubenstein, J. L., Greenough, W. T., Beckel-Mitchener, A., . . . & Tierney, E. (2004). Autism as a disorder of neural information processing: Directions for research and targets for therapy&ast. *Molecular Psychiatry*, 9(7), 646–663.
- Chouinard, G., Young, S. N., & Annable, L. (1983). Antimanic effect of clonazepam. *Biological Psychiatry*, 18(4), 451.
- Han, S., Tai, C., Westenbroek, R. E., Frank, H. Y., Cheah, C. S., Potter, G. B., . . . & Catterall, W. A. (2012). Autistic-like behaviour in *Scn1a*^{+/-} mice and rescue by enhanced GABA-mediated neurotransmission. *Nature*, 489(7416), 385–390.
- Rubenstein, J. L. R., & Merzenich, M. M. (2003). Model of autism: Increased ratio of excitation/inhibition in key neural systems. *Genes, Brain and Behavior*, 2(5), 255–267.

Clonidine

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Synonyms

[Catapres](#); [Clonidine ER](#); [Kapvay](#)

Definition

Clonidine is an α -2 agonist, typically used as a medication in patients with ASD to target symptoms related to attention, hyperactivity, anxiety, or sleep. It has no FDA-specific indications for this population but is approved for treatment of ADHD in ages 6–17, and is available in multiple preparations including oral and transdermal. Therapeutic effects are thought to be mediated through α -adrenoreceptors that cause a reduction in sympathetic nervous system tone and vital signs such as blood pressure and heart rate. Common side effects include somnolence, dry mouth, constipation, and dizziness. Due to potential effects on blood pressure or heart rate, initiation or discontinuation of this medication should be managed closely by a physician. Studies investigating clonidine in patients with ASD are not robust, with only two small placebo-controlled double-blind trials published that showed some benefit for ADHD/hyperarousal symptoms and irritability (Frankhauser et al. 1992, Jaselskis et al. 1992). Most recently, a retrospective study by Ming et al. (2008) found clonidine to benefit individuals with poor sleep, aggression, mood instability, and

ADHD symptoms. Despite this lack of published evidence in the ASD population, clonidine is commonly used in clinical practice with perceived good therapeutic effect.

See Also

► [Attention Deficit/Hyperactivity Disorder](#)

References and Reading

- Frankhauser, M. P., Karumanchi, V. C., German, M. L., Yates, A., & Karumanchi, S. D. (1992). A double-blind, placebo-controlled study of the efficacy of transdermal clonidine in autism. *The Journal of Clinical Psychiatry*, 53(3), 77.
- Jaselskis, C. A., Cook, E. H., Jr., Fletcher, K. E., & Leventhal, B. L. (1992). Clonidine treatment of hyperactive and impulsive children with autistic disorder. *Journal of Clinical Psychopharmacology*, 12(5), 322–327.
- Ming, X., Gordon, E., Kang, N., & Wagner, G. C. (2008). Use of clonidine in children with autism spectrum disorders. *Brain and Development*, 30(7), 454–460.
- Wolraich, M., Brown, L., Brown, R. T., DuPaul, G., Earls, M., & Feldman, H. M. (2011). ADHD: Clinical practice guidelines for the diagnosis, evaluation, and treatment of Attention-Deficit/Hyperactivity Disorder in children and adolescents. *Pediatrics*, 128(5), 1007–1022.

Clonidine ER

► [Clonidine](#)

Clonotril

► [Clonazepam: Definition](#)

Clopine

► [Clozapine](#)

Clozapine

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Synonyms

[Azaleptin](#); [Clopine](#); [Denzapine](#); [FazaClo](#); [Froidir](#); [Klozapol](#); [Leponex](#); [Zaponex](#)

Definition

Clozapine is an atypical antipsychotic that is FDA-approved for the management of treatment-resistant schizophrenia. It is believed to antagonize dopamine-2 (D₂) receptors, which causes reduced symptoms of psychosis and stabilizes affective symptoms, as well as serotonin 2A (5-HT_{2A}) receptors, causing enhanced dopamine release in certain brain regions and possibly improving cognitive and affective symptoms (Stahl 2009). However, its pharmacological profile is highly complex and likely involves multiple pathways yet to be uncovered.

Clozapine is typically reserved for patients who have not responded to other antipsychotic medications or who have suffered unwanted side effects from such therapies, such as tardive dyskinesia or extrapyramidal symptoms (EPS) (Novartis 2010). Clozapine rarely causes tardive dyskinesia and is even known to reduce dyskinesias and EPS, like dystonia (Saddock and Saddock 2007). It is the only antipsychotic that has demonstrated a reduced risk of suicidal behavior in patients with schizophrenia and schizoaffective disorder (Novartis 2010). For many of these reasons, clozapine is considered the “gold standard” in the treatment of

schizophrenia and schizoaffective disorder, but its use is limited by its side effect profile. A potentially fatal adverse effect is agranulocytosis, characterized by a decreased white blood cell count ($WBC < 2,000/mm^3$) and experienced in 1–2% of patients who take clozapine after 1 year of treatment. Every patient undergoing clozapine treatment receives weekly blood draws for 6 months, followed by biweekly blood draws for an additional 6 months, to monitor for agranulocytosis. This can be a cumbersome and painful process, which is why clozapine is often reserved for those who have had minimal success with other medications. Other adverse effects include an increased risk of seizures, excessive salivation, weight gain, cardiometabolic risk, and myocarditis (Stahl 2008).

Clozapine is not currently approved for treatment of autism spectrum disorders (ASDs). There have been, however, four case reports of clozapine use in the treatment of individuals with autistic disorder (autism). These reports described clozapine use in a 32-year-old autistic male (Gobbi and Pulvirenti 2001), three autistic children ranging in age from 8 to 12 years (Zuddas et al. 1996), a 17-year-old autistic adolescent male with mental retardation (Chen et al. 2001), and a 15-year-old autistic female (Lambrey et al. 2010). Overall, these reports suggested favorable improvement in some symptoms, particularly aggressiveness. Double-blind, placebo-controlled data is needed before recommendations can be made regarding the efficacy of clozapine use in treating ASDs. Clinical benefit must be heavily weighed against clozapine's extensive side effect profile before starting it in any patient.

See Also

- ▶ Antipsychotics: Drugs
- ▶ Atypical Antipsychotics
- ▶ Dopamine
- ▶ Serotonin
- ▶ Tardive Dyskinesia

References and Reading

- Chen, N. C., Bedair, H. S., McKay, B., Bowers, M. B., & Mazure, C. (2001). Clozaril in the treatment of aggression in an adolescent with autistic disorder. *The Journal of Clinical Psychiatry*, 62(6), 479–480.
- Gobbi, G., & Pulvirenti, L. (2001). Long-term treatment with clozapine in an adult with autistic disorder accompanied by aggressive behaviour. *Journal of Psychiatry & Neuroscience*, 26(4), 340–341.
- Lambrey, S., Falissard, B., Martin-Barrero, M., Bonnefoy, C., Quilici, G., Rosier, A., & Guillin, O. (2010). Effectiveness of clozapine for the treatment of aggression in an adolescent with autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, 20(1), 79–80.
- Novartis Pharmaceuticals Corporation. (2010). *Prescribing information*. Retrieved October, 2011, from <http://www.pharma.us.novartis.com/product/pi/pdf/Clozaril.pdf>
- Saddock, B. J., & Saddock, V. A. (2007). *Kaplan & Saddock's Synopsis of psychiatry: Behavioral sciences/clinical psychiatry* (p. 1095). Philadelphia: Lippincott Williams & Wilkins, a Wolters Kluwer Business.
- Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd ed., pp. 409–410). New York: Cambridge University Press.
- Stahl, S. M. (2009). *Stahl's essential psychopharmacology: The prescriber's guide* (3rd ed., pp. 113–118). New Delhi: Cambridge University Press.
- Zuddas, A., Ledda, M. G., Fratta, A., Muglia, P., & Cianchetti, C. (1996). Clinical effects of clozapine on autistic disorder. *The American Journal of Psychiatry*, 153(5), 738.

Cluster Analysis

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Definition

Cluster analysis is a statistical technique which seeks to group individuals according to their similar characteristics. That is, “clusters” of characteristics which might, for example, group individuals who make pottery would include fine motor skill, eye-hand coordination, sensitive

tactile receptors in fingers and hands, high level of ability to perceive moisture content in earth materials, and good color discrimination. While not all individuals with one or two of these characteristics will be able to become a good potter with appropriate training, people who possess all or most of these features will have a distinct advantage in becoming talented potters. Similarly, individuals who exhibit the cluster of symptoms associated with an Autism Spectrum Disorder will be more likely to actually have this condition. However, because there is substantial variation in symptom expression between individuals with particular autism disorders, there has been some conjecture as to what comprises Autistic Disorder versus Aspergers Syndrome or Pervasive Developmental Disorder-Not Otherwise Specified. Cluster analysis allows for identification of clinical subgroups (based on the type, number of, and severity of symptoms) to facilitate recommendations for appropriate treatment – especially early in life when specialized intervention has the potential to result in the greatest positive outcomes for the individual.

Historical Background

The evolution of conceptualizations of autism and reflection of these in the major diagnostic manuals is important in understanding the barriers to accurate diagnosis of those individuals who either do not fit a predetermined diagnostic profile due to subtle expression of symptoms or who present with a complex combination of impairments impacted by comorbid disorders. The transition of autism from an all-or-none condition with one fixed constellation of symptoms to a *spectrum* representing substantial variation in symptom expression has been significant in shaping clinicians' utilization of diagnostic criteria for the purpose of accurate identification. This transition is best exemplified in the changes to diagnostic categories shown in the Diagnostic and Statistical Manual (DSM manual) from its initial publication in 1952 to the 5th edition due for release in 2013. Since the DSM has remained prominent in formal

decisions regarding the standards for diagnosis of autism conditions, the evolution in autism conceptualizations will be examined in relation to DSM adaptations to those standards over time.

Despite its robust history in the clinical research since 1943, autism was not recognized as a distinct disorder of childhood in the DSM until its third edition (DSM-III: APA 1980). The DSM-III conceptualized autism as an all-or-none condition by presenting one diagnostic category (i.e., Infantile Autism) and six diagnostic criteria with the stipulation that evidence of impaired functioning was required for all six of these for a diagnosis of autism to be made. This basis for classification was criticized as being limited because it focused only on those cases in which neurological impairment was evident at birth. Children who exhibited regression in functioning between the ages of 18 and 36 months were not clearly represented, thus leading to subsequent revision of the Infantile Autism category to Autistic Disorder in 1987 with publication of the DSM-III-R. This new diagnostic label acknowledged the possible presence of two autism subtypes (i.e., Infantile Autism and Regressive Autism) distinguished on the basis of the age at which children displayed the pattern of impairments indicative of neurological abnormality.

The DSM-IV (APA 1994) and its revision, the DSM-IV-TR (APA 2000), offered a broader diagnostic framework which allowed for identification of high functioning individuals with average to above average cognitive ability and reasonably well-developed language skills. In addition to specifying five subtypes of Pervasive Developmental Disorder (i.e., Autistic Disorder, Aspergers Disorder, Rett's Disorder, Childhood Disintegrative Disorder, and Pervasive Developmental Disorder-Not Otherwise Specified), there was revision of the diagnostic criteria to encompass a wider range of atypical behaviors and reduce the number of symptoms required for a formal diagnosis. In the case of Autistic Disorder, the DSM-IV-TR (APA 2000) maintained the "triad of impairment" model (Wing and Gould 1979) with stipulation that qualitative discrepancies, between the child and chronologically

similar peers, should occur in *social interaction* (at least two of four symptom clusters) and *communication* (at least one of four symptom clusters), along with presence of *restricted, repetitive, and stereotyped patterns of behavior, interests, and activities* (at least one of four symptoms clusters). Despite this expansion of diagnostic categories, clinical researchers (e.g., Lord and Risi 2000; Schuler and Fletcher 2002; Tidmarsh and Volkmar 2003) continued to argue that the variability in manifestation of symptoms from one individual to another was not adequately addressed by the DSM-IV-TR (APA 2000), thus risking poor identification of high functioning individuals. Also, strict adherence to the “triad of impairment” model did not acknowledge the full constellation of difficulties (e.g., hypersensitivity to sensory stimuli in the environment, restricted diet, and poor sleeping patterns) of clinical significance in understanding the issues which disrupted daily functioning and would require specialized intervention (Keane 2004; Lord and Risi 2000). The latest version of the DSM, currently in draft form, contains some evidence of attempts to encompass a broader range of functional levels in autism and these attempts will be further discussed.

Current Knowledge

The term Autism Spectrum Disorder (ASD), first introduced by Lorna Wing in 1992, has replaced Pervasive Developmental Disorder as the collective label for Autistic Disorder, Aspergers Disorder, Pervasive Developmental Disorder-Not Otherwise Specified, and Childhood Disintegrative Disorder with this last condition being an addition to the group of diagnoses represented by the ASD label. It is also proposed that the three impairment domains listed in earlier editions of the DSM be reduced by merging the communication and social behavior categories (Criterion 1) on the basis that symptoms in these domains are interrelated and possibly due to similar contextual antecedents. In addition, it is argued that atypical communication is not exclusive to autistic disorder nor is it significant to functional impairment in

all cases, thus leading to its re-conception as a contributing rather than defining aspect of ASD. Further, Criterion 1 requires evidence of developmental delay in all three areas of social/communication functioning which include deficits in: social-emotional reciprocity, nonverbal communication, and capacity to maintain relationships. The second diagnostic criterion pertains to presence of fixated interests and repetitive behaviors with stipulation that atypical performance be recorded for two of four symptom clusters: (1) stereotyped or repetitive speech, motor movements, or use of objects; (2) excessive adherence to routines; (3) restricted interests of abnormal intensity; and (4) hyper- or hypo-reactivity to sensory input or unusual interest in sensory aspects of the environment. Interestingly, the DSM-V draft includes a diagnostic criterion which states that symptoms might not become evident until the child is exposed to social stimuli which create demand (s)he is incapable of responding to due to underlying skill deficits. This inclusion in the DSM-V draft provides a basis for continued monitoring of cases with suspected ASD to ensure they are accurately identified and provided with access to specialized educational support at the earliest possible time.

Notwithstanding these potentially advantageous revisions to diagnostic criteria, alternations such as amalgamation of diagnostic categories and removal of Aspergers Disorder as a distinct diagnostic label have created controversy in the clinical field. Further, the anticipated elucidation in methods for identification of complex cases and those with more subtle symptom manifestation has not been realized. Marttila et al. (2011) applied the draft DSM-V diagnostic criteria for ASD to evaluate 26 children with an autism condition confirmed by their Autism Diagnostic Interview-Revised (ADI-R) and Autistic Diagnostic Observation Schedule (ADOS) scores. Those researchers reported that DSM-IV criteria were not of sufficient sensitivity to identify high functioning children with well-developed communication skills and paucity of stereotyped and repetitive behavior. Findings such as these emphasize that the heterogeneity which exists in the autism spectrum cannot be adequately accounted

for by diagnostic categories requiring a fixed profile of symptoms for formal diagnosis. Witwer and Lecavalier (2008) report that there is significant variation in symptom manifestation within DSM diagnostic categories and argue for greater investigation of alternative approaches to categorization.

Cluster analysis shows promise as a system for clarifying differences in functioning across the autism spectrum by leading to identification of subgroups which are potentially independent of diagnostic labels. Cluster analytic investigations of the past decade have focused on detection of those dimensions (arising from skill deficits) most capable of providing a valid basis for differentiating between groups of individuals with an autism condition. While the spectrum concept of autism argues that there is an underlying dimension or set of dimensions along which all individuals will vary, there is still debate over precisely what constitutes this underlying dimension (Szatmari 1992). A large proportion of studies have examined whether impairment in particular aspects of functioning (e.g., cognitive vs. adaptive behavior) or combinations of deficits (e.g., cognitive plus social skill) represent a reliable basis for forming homogeneous autism subgroups. Other studies have sought to examine whether number of symptoms or severity of deficits might result in accurate differentiation to allow for a more inclusive basis for understanding and treating individuals on the autism spectrum.

Eaves et al. (1994) reported on a cluster analysis which grouped children into four subtypes based on number of symptoms and extent of cognitive impairment. Groups were described as representing typical autism (autistic disorder), low functioning autism (intellectually impaired), high functioning autism (Aspergers disorder), and “hard to diagnose” with mild to moderate cognitive disability and a family history of learning difficulties. Researchers such as Prior et al. (1998) have indicated that the distinguishing feature between autism subgroups is related to the severity of social and cognitive impairments rather than distinctive symptom patterns. That study supported the concept of a spectrum of autistic dysfunction which places children on a

continuum from low functioning to high functioning. The concept of a spectrum was previously supported by Sevin et al. (1995) who suggested that the *degree* of impairment in the core features of autism (i.e., social interaction, language and communication, and restricted and ritualistic behaviors) is the most important basis for making sense of the variety of behaviors which fall under the umbrella of autism. Fernell et al. (2010) also argued for the significance of symptom severity in clarifying the variations in functioning which characterize individuals with an autism disorder. Those researchers presented a seven-cluster model of subgroups of preschool children who were differentiated on the basis of cognition, behavior, speech and language, and motor control. Similarly, Wiggins et al. (2009) presented a three-cluster solution for 186 toddlers who fell into homogeneous groups due to variation in social/communication and cognitive skills plus the rate and intensity of repetitive behavior and presence of abnormal sensory responding. Lane et al. (2010) and Lane et al. (2011) used model-based cluster analysis of parent reports of their children’s sensory responses to explore the presence of subgroups differentiated on the basis of type of sensory processing dysfunction. Those researchers identified three sensory subtypes (i.e., sensory-based inattentive seeking, sensory modulation with movement sensitivity, and sensory modulation with taste/smell sensitivity) and argued that these subtypes would have a differential impact on core autism symptoms as well as requiring different remediation strategies. Verte et al. (2006) examined the question of whether children with autism disorder could be differentiated into subgroups based primarily on their scores from the Children’s Communication Checklist and reported a three-cluster solution in which differentiation was achieved in relation to severity of communication deficits such as inappropriate initiation, stereotyped conversation, and interactional rapport. Studies such as this indicate that it is variation in the social-pragmatic aspects of language which allows for separation of individuals into clinical subgroups rather than verbal ability per se. The significance of using language performance to assist in establishing specific

subgroups of individuals has been previously discussed by Tager-Flusberg and Joseph (2003). In both studies, level of cognitive disability was found to mediate the effects of language impairment.

Future Directions

Manualized diagnostic systems, although necessary for standardization of decisions regarding the presence of autism, have not represented all aspects of the spectrum well. The DSM-V draft, despite its proposed revisions in the diagnostic categories for autism disorder, does not appear to offer substantial improvement in accounting for the full range of phenotypes which present in the clinical environment (Marttila et al. 2011). This limitation is exacerbated by the clinical finding that individuals with the same diagnosis can and do exhibit varying profiles of impairment and behavioral disturbance, thus leading to heterogeneity within diagnostic groups (Witwer and Lecavalier 2008). Clinical issues such as these bring into question the common practice of using a diagnostic label as the basis for intervention planning. This label-driven intervention approach, which guides clinicians in the field to apply genetic strategies designed to *remediate aspects of autism*, has not resulted in strong positive outcomes and prompted the call for additional analyses to elucidate specific autism subgroups that can be treated more directly (Bitsika 2008; Perry et al. 2009).

Identification and description of clinical subgroups via statistical methods such as cluster analysis can contribute to development of targeted interventions capable of building competencies to enhance positive outcomes for individuals with an autism disorder in the long term. There are a number of considerations which might be of merit in maximizing the contribution of cluster analysis to effective treatment. Traditionally, studies in this field have relied on total scores from standardized tests (e.g., WISC Full Scale IQ) to establish clinical subgroups which share the same characteristics on specified dimensions

(e.g., social skill and communication). It is suggested that future research elaborates on standardized data via inclusion of subtest scores to facilitate an in-depth investigation of specific abilities. Although differentiation of individuals with autism disorder on the basis of global abilities such as intelligence and adaptive behavior has been useful in more precise classification, it is their behavioral challenges which are particularly disruptive to social functioning and often the reason for referrals for intervention. Researchers (e.g., Bitsika et al. 2008; Prior et al. 1998; Waterhouse et al. 1996) have made some progress in establishing subgroups of individuals with an autism disorder based on the presenting behaviors which interfere with their functioning. However, further investigations are necessary to aid differentiation not only in relation to behavioral topography and intensity but also variations in this behavior resulting from exposure to particular environmental stimuli. Reclassification of individuals with an ASD into clearly defined subgroups, according to suggestions such as those raised here, can only promote a better understanding of their particular needs and clinical treatment to aid effective functioning across the lifespan.

See Also

► [Statistical Approaches to Subtyping](#)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders – DSM-III* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders – DSM-IV* (4th ed.). Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, text revision – DSM-IV-TR* (4th ed.). Washington, DC: Author.
- Bitsika, V. (2008). Including an analysis of difficult behaviour in the assessment of children with an Autism spectrum disorder: Implications for school psychologists. *Australian Journal of Guidance and Counselling*, 18, 1–14.

- Bitsika, V., Sharpley, C. F., & Orapeleng, S. (2008). Using cognitive, adaptive and behavioral indices for cluster analysis of ASD subgroups. *Journal of Intellectual Disability Research*, 52, 973–985.
- Chawarska, K., Klin, A., Paul, R., Macari, S., & Volkmar, F. (2009). A prospective study of toddlers with ASD: Short-term diagnostic and cognitive outcomes. *Child Psychology and Psychiatry*, 50, 1235–1245.
- Eaves, L. C., Ho, H. H., & Eaves, D. M. (1994). Subtypes of Autism by cluster analysis. *Journal of Autism and Developmental Disorders*, 24, 3–22.
- Fernell, E., Hedvall, A., Norrelgen, F., Eriksson, M., Hoglund-Carlsson, L., Barnevik-Olsson, M., et al. (2010). Developmental profiles in preschool children with Autism spectrum disorders referred for intervention. *Research in Developmental Disabilities*, 31, 790–799.
- Keane, E. (2004). Autism: The heart of the disorder? Sensory processing social engagement – Illustrations from autobiographical accounts and selected research findings. *Australian Journal of Early Childhood*, 29, 8–14.
- Lane, A. E., Young, R. L., Baker, A. E., & Angley, M. T. (2010). Sensory processing subtypes in Autism: Association with adaptive behaviour. *Journal of Autism and Developmental Disorders*, 40, 112–122.
- Lane, A. E., Dennis, S. J., & Geraghty, M. E. (2011). Brief report: Further evidence of sensory subtypes in Autism. *Journal of Autism and Developmental Disorders*, 41, 826–831.
- Lord, C., & Risi, S. (2000). Diagnosis of Autism spectrum disorders in young children. In A. Wetherby & B. Prizant (Eds.), *Autism spectrum disorders: A transactional developmental perspective* (pp. 167–190). Baltimore: Paul H. Brookes.
- Marttila, M., Keilinen, M., Linna, S., Jussila, K., Ebeling, H., Bloigu, R., et al. (2011). Autism spectrum disorders according to DSM-IV-TR and comparison with DSM-5 draft criteria: An epidemiological study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 50, 583–592.
- Perry, A., Flanagan, H. E., Grier, J. D., & Freeman, N. L. (2009). Brief report: The Vineland adaptive behavior scales in young children with Autism spectrum disorders at different cognitive levels. *Journal of Autism and Developmental Disorders*, 39, 1066–1078.
- Prior, M., Eisnemajer, R., Leekam, S., Wing, L., Gould, J., Ong, B., et al. (1998). Are there subtypes within the Autistic spectrum? A cluster analysis of a group of children with Autistic spectrum disorders. *Journal of Child Psychology and Psychiatry*, 39, 893–902.
- Schuler, A. L., & Fletcher, C. E. (2002). Making communication meaningful: Cracking the language interaction code. In R. L. Gabriels & D. E. Hill (Eds.), *Autism: From research to individualized practice* (pp. 127–154). London: Jessica Kingsley.
- Sevin, J. A., Matson, J. L., Coe, D., Love, S. R., Matese, M. J., & Benavidez, D. A. (1995). Empirically derived subtypes of pervasive developmental disorders: A cluster analytic study. *Journal of Autism and Developmental Disorders*, 25, 561–578.
- Szatmari, P. (1992). The validity of Autistic spectrum disorders: A literature review. *Journal of Autism and Developmental Disorders*, 22, 583–600.
- Tager-Flusberg, H., & Joseph, R. M. (2003). Identifying neurocognitive phenotypes in Autism. *Philosophical Transactions of the Royal Society of London*, 358, 303–314.
- Tidmarsh, L., & Volkmar, F. (2003). Diagnosis and epidemiology of Autism spectrum disorders. *Canadian Journal of Psychiatry*, 48, 517–525.
- Verte, S., Geurts, H. M., Roeyers, H., Rosseel, Y., Oosterlaan, J., & Sergeant, J. A. (2006). Can the children's communication checklist differentiate Autism spectrum subtypes? *Autism*, 10, 266–287.
- Waterhouse, L., Morris, R., Allen, D., Dunn, M., Fein, D., Feinstein, C., et al. (1996). Diagnosis and classification in Autism. *Journal of Autism and Developmental Disorders*, 26, 59–86.
- Wiggins, L. D., Robins, D. L., Bakeman, R., & Adamson, L. B. (2009). Brief report: sensory abnormalities as distinguishing symptoms of an autism spectrum disorder in young children. *Journal of Autism and Developmental Disorders*, 1087–1091.
- Wiggins, L. D., Robins, D. L., Adamson, L. B., Bakeman, R., & Henrich, C. C. (2011). Support for a dimensional view of Autism spectrum disorders in toddlers. *Journal of Autism and Developmental Disorder*. <https://doi.org/10.1007/s10803-011-1230-0>.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11–29.
- Witwer, A. N., & Lecavalier, L. (2008). Examining the validity of Autism spectrum disorder subtypes. *Journal of Autism and Developmental Disorders*, 38, 1611–1624.

Cluster Reports and Autism

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Definition

Using an epidemiological definition, a “cluster” is an aggregation of health-related events or diseases that are grouped together in time and/or space in

greater than expected (real or perceived) frequencies (Centers for Disease Control [CDC] 1990). “Clustering” is usually used to describe aggregations of relatively uncommon diseases, such as cancer or birth defects, in a population or geographic area for which no known cause exists, but may be suspected. This definition will be used, in contrast to, for example, clustering of disease traits or symptoms, such as in ASD, that might be used to classify subtypes of a condition.

Historical Background

Unusual events such as clusters occur regularly, often by chance alone, so there are many considerations that come into defining or further investigating a cluster of disease. From a statistical perspective, it is almost inevitable that some schools, workplaces, or neighborhoods will be associated with clusters of chronic diseases, which may lead to concern on the part of the seemingly affected group. When first noticed, cases with the disease that appear in a cluster are often suspected of resulting from the same specific cause, rather than as independent events (e.g., coin tosses) that happened to have occurred by chance in one place or time period. A cluster may be investigated to help determine whether there is a common cause, in which case the definition of the underlying population in space and time is critical (see below), but even so there are often no reasons identified for the cluster. For example, for a relatively uncommon disease like autism, with a prevalence of about 1 per 68–100 children, in a small enough population, such as a neighborhood, two families with affected children could constitute a greater than expected occurrence statistically, but yield little information on causes.

Infectious diseases typically have time and place components. Investigating clusters (or “outbreaks” in infectious disease), such as the classic church picnic food poisoning, can lead to identification of the source of the disease and thus the means to stop it or prevent it in the future. If clusters are of sufficient size and

importance, they may eventually turn into epidemics. Often, when clusters are recognized, they are reported to health departments or officials in the local area. Breakthroughs and successes in infectious disease control have resulted from the epidemiologic evaluation of clusters of cases; well-known examples include cholera linked to the Broad Street pump in London by John Snow in the 1850s, the investigation of cases of pneumonia at one hotel in Philadelphia in 1976 that led to the identification of Legionnaires’ disease and the ventilation system as a source, and the 1981 report that seven cases of *Pneumocystis carinii* pneumonia had occurred among young homosexual men in Los Angeles, which began the identification of the AIDS epidemic.

Although less common, investigations of non-infectious disease clusters have also resulted in notable examples of breakthroughs linking a health effect to an exposure, such as angiosarcoma among vinyl chloride workers, adenocarcinoma of the vagina associated with maternal use of diethylstilbestrol (DES), and phocomelia with the use of thalidomide. These tended to have a time (vs. space) component with an alert clinician seeing more cases in a short period than expected, but ended up affecting a widespread population geographically. More often, health investigations may not identify the cause of the cluster due to methodologic limitations, usually of small sample size (Wartenberg and Greenberg 1990) or because of the role of chance.

There are several steps to be taken in a careful cluster evaluation, including establishing a specific case definition, so for example, different types of birth defects or cancers are not grouped together; defining the population denominator in person-time; confirming suspected cases and comparing rates to those in the literature; and accounting for known causes (Agency for Toxic Substances and Disease Registry [ATSDR] 2000; CDC 1990). The step of defining the appropriate population, for example, of an entire school versus a few blocks of a neighborhood, or identifying the appropriate exposure period, may result in realizing that the prevalence of the disease is actually within expected. If a cluster is confirmed,

the next step is to assess common factors or possible exposure pathways to generate hypotheses about etiologic mechanisms.

There are numerous methods developed for identifying clusters, sometimes called cluster or geospatial analysis, to determine whether cases are linked in time or place (Rothenberg and Thacker 1992). These can also be used with routine disease-monitoring data to identify spatially related subgroups that may have a common cause or to learn more about etiologic mechanisms. Detailed description of these methods is beyond the scope of this discussion.

Current Knowledge

Clusters of Autism

Few clusters of autism have been published, but this is not necessarily unexpected as reports of clusters may be handled only at the local level. The best known is the Brick Township cluster in New Jersey, initially reported by a parent group concerned about the number of children with autism and environmental issues due to the proximity of several Superfund sites. Their community survey identified an apparently high rate of autism, so the Centers for Disease Control and Prevention (CDC) and the Agency for Toxic Substances and Disease Registry (ATSDR) conducted an extensive investigation in 1998 to identify and clinically confirm all cases (Bertrand et al. 2001; CDC 2000). The prevalence they determined was about four times higher than the frequently cited prevalence in the literature at the time, of 1 per 1000. The exposure assessment by ATSDR did not reveal any links between environmental contamination and the autism cases. When studies with similar methods of intensive case finding were compared, the prevalence was determined to be more similar. Routine surveillance figures for autism were not available in the USA at that time, so this cluster in part led to the implementation of such a system coordinated by the CDC, i.e., the Autism and Developmental Disabilities Monitoring (ADDM) program, which is ongoing (Christensen et al. 2016).

Another parent-initiated report of an autism cluster occurred in the UK, which was also resolved as being consistent with a higher prevalence of autism than commonly known at the time (Baron-Cohen et al. 1999). Further confirming the difficulties of studying small clusters ($n = 7$) and appropriate comparison groups, the rate would only be considered higher than expected when compared to rates in children under age 5 rather than in all those under age 18. A cluster of autism was reported to the Minnesota Department of Health by parents of Somali children; supported by findings about Somali immigrants in Sweden, this led to further investigations to identify possible risk factors (Hewitt et al. 2016; Esler et al. 2017). As is often the case when studying clusters more rigorously, prevalence in Somalis was not substantially higher than in Whites (although higher than other race/ethnic groups), but Somali children with ASD were more likely to have co-occurring intellectual disability than other groups.

There have been popular media reports of “clusters” or higher rates of autism occurring in areas where information technology (IT) companies cluster, such as the Silicon Valley in California. Although not limited to one workplace as would be typical for a cluster, Baron-Cohen et al. (1997) reported that fathers and grandfathers of children with autism were overrepresented in the field of engineering. Two subsequent studies raised the possibility that differences in ascertainment due to SES might explain the results (Jarrold and Routh 1998; Windham et al. 2009). The latter study, conducted in California, did not find that fathers were overrepresented in “high-tech” fields such as engineering and computer programming, but rather mothers were. Further, that study conducted in a six-county region of the San Francisco Bay Area did not find overrepresentation of cases with autism born in the county that includes Silicon Valley, compared to controls. A more recent study reported higher prevalence of autism in a high-density IT region of the Netherlands compared to two other areas, based on school reports, so further investigation will be done (Roelfsema et al. 2012). Such

observations may be related to “clustering” of autism or autistic traits in families, given the strong genetic component, but this is a different topic (see ► “Genetics”, ► “Broader Autism Phenotype”).

Several studies have used complex statistical methods to identify clusters of autism based on geographic residence in order to generate hypotheses about environmental exposures or other drivers of autism prevalence at the local level. Two studies in CA were based on data from the statewide Department of Developmental Services (DDS) database, which has been used for surveillance purposes (Windham et al. 2011), with slightly different birth years and methods. Clusters were identified in both studies; after accounting for several factors, one found the clustering at birth was related to high parental education (Van Meter et al. 2010), which may be related to successfully seeking services for affected children, confirmed by the other (Mazumdar et al. 2013), which reported clusters loosely defined by residence at diagnosis to be in high SES areas and related to neighborhood-level diagnostic resources. Similarly, two other studies of geographic patterns of ASD concluded that factors impacting diagnosis, including maternal age, likely drove the spatial variation observed (Hoffman et al. 2012, 2017). Although raising the limitations of studying environmental factors across wide geographic areas, environmental causes of autism have been increasingly investigated and associated with ASD (Kalkbrenner et al. 2014).

Future Directions

The occurrence of clusters is not predictable, so the merit of further investigation must be considered on an ad hoc basis. As there are now several surveillance systems for autism, cluster analyses could be conducted to identify so-called hot spots and thus possible risk factors. However, these analyses seem less useful for primary study of the etiology of autism, given its broad multifactorial nature.

See Also

- Broader Autism Phenotype
- Genetics

References and Reading

- Agency for Toxic Substances and Disease Registry (ATSDR). (2000). “Definition of clusters”, *Case Studies in Environmental Medicine (CSEM)*. U.S. Department of Health and Human Services.
- Baron-Cohen, S., Wheelwright, S., Stott, C., Bolton, P., & Goodyer, I. (1997). Is there a link between engineering and autism? *Autism, 1*, 153–163.
- Baron-Cohen, S., Saunders, K., & Chakrabarti, S. (1999). Does autism cluster geographically? A research note. *Autism, 3*, 39–43.
- Bertrand, J., Mars, A., Boyle, C., Bove, F., Yeargin-Allsopp, M., & Decoufle, P. (2001). Prevalence of autism in the United States population: The brick township, New Jersey, investigation. *Pediatrics, 108*, 1155–1161.
- Centers for Disease Control (CDC). (1990). Guidelines for investigating clusters of health events. *MMWR, 39* (RR-11), 1–23.
- Centers for Disease Control and Prevention (CDC). (2000). *Prevalence of autism in Brick Township, New Jersey, 1998: Community report. April 2000*. U.S. Department of Health and Human Services. www.cdc.gov/ncbddd/developmentaldisabilities/documents/brick-report.pdf. Accessed July 2017.
- Christensen, D. L., Baio, J., Van Naarden Braun, K., Bilder, D., Charles, J., Constantino, J. N., Daniels, J., et al. (2016). Prevalence and characteristics of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2012. *MMWR Surveillance Summary, 65*, 1–23.
- Esler, A. N., Hall-Lande, J., & Hewitt, A. (2017). Phenotypic characteristics of autism spectrum disorder in a diverse sample of Somali and other children. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-017-3232-z>. Epub Jul 8.
- Hewitt, A., Hall-Lande, J., Hamre, K., Esler, A. N., Punyko, J., Reichle, J., & Gulaid, A. A. (2016). Autism spectrum disorder (ASD) prevalence in Somali and non-Somali children. *Journal of Autism and Developmental Disorders, 46*, 2599–2608.
- Hoffman, K., Kalkbrenner, A. E., Vieira, V. M., & Daniels, J. L. (2012). The spatial distribution of known predictors of autism spectrum disorders impacts geographic variability in prevalence in central North Carolina. *Environmental Health, 11*, 80–89.

- Hoffman, K., Weisskopf, M. G., Roberts, A. L., Raz, R., Lyall, K., Hoffman, E. M., et al. (2017). Geographic patterns of autism spectrum disorder among children of nurses' health study II women. *American Journal of Epidemiology*. <https://doi.org/10.1093/aje/kwx158>. Epub May 19.
- Jarrold, C., & Routh, D. A. (1998). Is there really a link between engineering and autism? A reply. *Autism*, 2, 281–289.
- Kalkbrenner, A. E., Schmidt, R. J., & Penlesky, A. C. (2014). Environmental chemical exposures and autism spectrum disorders: A review of the epidemiological evidence. *Current Problems in Pediatric and Adolescent Health Care*, 44, 277–318.
- Mazumdar, S., Winter, A., Liu, K. Y., & Bearman, P. (2013). Spatial clusters of autism births and diagnoses point to contextual drivers of increased prevalence. *Social Science & Medicine*, 95, 87–96.
- Roelfsema, M. T., Hoekstra, R. A., Allison, C., Wheelwright, S., Brayne, C., Matthews, F. E., et al. (2012). Are autism spectrum conditions more prevalent in the information technology region? A school-based study of three regions the Netherlands. *Journal of Autism and Developmental Disorders*, 42, 734–739.
- Rothenberg, R. B., & Thacker, S. B. (1992). Guidelines for the investigation of clusters of adverse health events. In P. Elliott, J. Cuziak, D. English, & R. Stern (Eds.), *Geographical and environmental epidemiology: Methods for small-area studies* (pp. 264–277). Oxford: Oxford University Press.
- Van Meter, K. C., Christiansen, L. E., Delwiche, L. D., Azari, R., Carpenter, T. E., & Hertz-Picciotto, I. (2010). Geographic distribution of autism in California: A retrospective birth cohort analysis. *Autism Research*, 3(1), 19–29.
- Wartenberg, D., & Greenberg, M. (1990). Detecting disease clusters: The importance of statistical power. *American Journal of Epidemiology*, 132, S156–S166.
- Windham, G. C., Fessel, K., & Grether, J. K. (2009). Autism spectrum disorders in relation to parental occupation in technical fields. *Autism Research*, 2, 183–191.
- Windham, G. C., Anderson, M. C., Croen, L. A., Smith, K. S., Collins, J., & Grether, J. K. (2011). Birth prevalence of autism spectrum disorders in the San Francisco Bay Area by demographic and ascertainment source characteristics. *Journal of Autism and Developmental Disorders*, 41, 1362–1372.

Cluttering

► Fluency and Fluency Disorders

CM-AT

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Synonyms

Blüm Study

Definition

CM-AT™ is an investigational drug candidate developed to potentially treat the core symptoms of autism spectrum disorder (ASD). The drug has received “fast-track” designation from the US Food and Drug Administration. CM-AT™ is a proprietary mix of digestive enzymes that is sprinkled on food in powder form. Materials distributed by the makers of CM-AT suggest that the drug may improve core and associated symptoms of ASD by enhancing the digestion of proteins. A phase III clinical trial of CM-AT™ was completed in 2011, though no data from that study have been published. Further trials are underway.

The hypothesized effectiveness of CM-AT™ is based on the clinical experience of pediatric chiropractor Joan Fallon, founder and CEO of the company that developed CM-AT™. Investigating the stool samples of children with ASD, Fallon observed that many of these children exhibited low levels of fecal chymotrypsin, a proteolytic digestive enzyme. These observations were not published in a peer-reviewed journal, and fecal chymotrypsin levels have not otherwise been reported in the ASD literature. The makers of CM-AT™ postulate that many children with ASD have deficient protein digestion, potentially manifesting in self-restricted diets that are low in protein (see ► [Feeding Problems](#)). CM-AT™ was

developed to compensate for deficient levels of chymotrypsin, potentially improving ASD symptoms by normalizing the digestion of proteins. The mechanism of this therapeutic effect is purported to be an increase in the levels of essential amino acids, some of which serve as precursors in the biosynthesis of monoamine neurotransmitters (see Amino Acids). This mechanism has not been empirically tested in published research to date.

Some aspects of this theory are empirically supported. Children with ASD have been found to exhibit both reduced protein intake (Sharp et al. 2013) and lower plasma levels of essential amino acids (Arnold et al. 2003) relative to non-autistic peers. However, no research to date has been published linking either dietary protein intake or essential amino acid levels in ASD to a reduced capacity for protein digestion. Moreover, correlations between plasma levels of essential amino and ASD symptoms have not been reported in the literature, and no published data exist to suggest that amelioration of an essential amino acid deficiency will improve core ASD symptoms. One prior trial assessing protease enzyme supplementation in children with ASD did not report any significant improvements in ASD symptoms compared to a placebo, though children in the treatment condition did report an increased variety of foods eaten (Munasinghe et al. 2010).

An initial phase III clinical trial of CM-AT™ was carried out from 2009 to 2011, enrolling 182 children with ASD and low chymotrypsin levels between 3 and 8 years of age. While results of the trial have yet to be published, Fallon has stated in an interview that the CM-AT™-treated group showed improvements in irritability and social withdrawal (Zeliadt 2016). In addition, materials on the drug maker's website contend that CM-AT™ was found to be safe and generally well tolerated. An open label extension of this initial trial is ongoing.

In 2015, a second phase III clinical trial of CM-AT™ was initiated, this time enrolling children with ASD and all levels of fecal chymotrypsin. Termed the biologic autism medication (Blüm) Study, the trial aims to enroll an estimated

300 children, aged 3–8 years. The designated primary outcome of the trial is a change in the irritability/agitation subscale of the aberrant behavior checklist (ABC) (see ► [Aberrant Behavior Checklist](#)). A change in the ABC Lethargy/Social Withdrawal subscale is listed as a secondary outcome for the trial. The Blüm Study and its associated open-label extension study are currently in progress.

See Also

- [Aberrant Behavior Checklist](#)
- [Amino Acids](#)
- [Feeding Problems](#)
- [Nutritional Interventions](#)

References and Reading

- A Trial of CM-AT in Children With Autism With All Levels of FCT (The Blüm Study). (2015). Retrieved from <https://clinicaltrials.gov/show/NCT02410902>. (Identification No. NCT02410902).
- Arnold, G. L., Hyman, S. L., Mooney, R. A., & Kirby, R. S. (2003). Plasma amino acids profiles in children with autism: Potential risk of nutritional deficiencies. *Journal of Autism and Developmental Disorders*, 33(4), 449–454.
- Munasinghe, S. A., Oliff, C., Finn, J., & Wray, J. A. (2010). Digestive enzyme supplementation for autism spectrum disorders: A double-blind randomized controlled trial. *Journal of Autism and Developmental Disorders*, 40(9), 1131–1138.
- Sharp, W. G., Berry, R. C., McCracken, C., Nuhu, N. N., Marvel, E., Saulnier, C. A., et al. (2013). Feeding problems and nutrient intake in children with autism spectrum disorders: A meta-analysis and comprehensive review of the literature. *Journal of Autism and Developmental Disorders*, 43(9), 2159–2173.
- Zeliadt, N. (2016, November 28). Controversial autism drug gets ambitious new trial. *Spectrum*. Retrieved from <https://spectrumnews.org/news/controversial-autism-drug-gets-ambitious-new-trial/>

CNTN4

- [CNTN4: Contactin 4](#)

CNTN4: Contactin 4

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Synonyms

AXCAM; BIG-2; CNTN4

Structure

Contactin 4 is a neuronal membrane protein. In humans, it is encoded by the gene *CNTN4*, located on chromosome 3p26.3-p26.2, containing 24 exons, and spanning 957 kb. Alternative splicing results in multiple transcript variants. The largest splice variant of contactin 4 (1,026 amino acids) shares 44–66% identity with other contactin proteins. It has six immunoglobulin-like domains, four fibronectin type III-like repeats, one C-terminal glycosylphosphatidylinositol (GPI) anchor to the extracellular part of the cell membrane, and one N-terminal signal peptide. These domains are conserved between the human and rat ortholog (BIG-2). A smaller splice variant (CNTN4A, 282 amino acids) has two fibronectin type III-like repeats and one GPI-anchoring domain (Zeng et al. 2002).

Function

Contactin 4 is a member of the immunoglobulin superfamily of neuronal cell adhesion molecules involved in axon growth, guidance, and fasciculation in the central nervous system. It is a member of the contactin subgroup of these molecules and is believed to function in the formation of axon connections and plasticity in the developing nervous system. Yoshihara et al. (1995) studied the rat ortholog protein, BIG-2, in vitro and found that it promoted neurite outgrowth when used as a neuronal substrate. Overall, their results suggest that this protein has a role in the formation and

maintenance of brain neuronal networks. Subsequently, Saito, Mimmack, Kishimoto, Keverne, and Emson (1998) concluded that this protein is involved in synaptogenesis by studying its expression pattern in the development of rat olfactory sensory neurons.

The expression profile of CNTN4 in the adult human brain has been examined by Northern blot analysis, but these studies are limited in scope. CNTN4 expression is most prominent in the cerebellum, occipital lobe, and frontal lobe, followed by thalamus, cerebral cortex, and substantia nigra (Kamei et al. 2000).

Pathophysiology

Several studies of developmental disorders have implicated *CNTN4* and suggest an important role in normal and abnormal central nervous system (CNS) development. Specifically, disruption of this gene may play a role in 3p-deletion syndrome and autism spectrum disorders (ASD), as evidenced by the studies described below.

Fernandez et al. (2004, 2008b) studied the breakpoints of a de novo balanced translocation (t(3,10)(p26;q26)) in a boy with characteristic physical features of 3p-deletion syndrome who met the criteria for ASD with impaired social functioning, verbal and nonverbal developmental delay, and repetitive behaviors. Fine mapping of the breakpoint on chromosome 3 revealed disruption of *CNTN4*, and this gene maps within the previously defined minimal candidate region for 3p-deletion syndrome. These results suggested a causal relationship between *CNTN4* disruption and 3p-deletion syndrome and a possible relationship with ASD.

Roohi et al. (2009) identified paternally inherited copy number variants (CNV) disrupting *CNTN4* in 3 of 92 subjects with ASD (deletions in two affected siblings and a duplication in an unrelated affected child). This report further implicated *CNTN4* as a candidate gene in ASD.

Glessner et al. (2009) reported a study of genome-wide copy number variation in two ASD and control cohorts. Among other ASD candidate genes, they found a significant increase in

inherited CNV deletions ($p = 0.004$) and duplications ($p = 0.013$) overlapping *CNTN4* in cases versus controls.

Most recently, Cottrell et al. (2011) identified unique nonsynonymous variants of *CNTN4* in 4 of 75 ASD-affected individuals versus 1 of 107 unaffected controls. All variants occurred at highly conserved positions in the gene and were predicted to be deleterious. However, this burden differential did not reach statistical significance or segregate with ASD in all affected families, leaving some question about the role of *CNTN4* variants in ASD pathogenesis.

Overall, there is evidence that *CNTN4* plays an important role in the normal development and maintenance of CNS function. It has been proposed that the molecular basis of *CNTN4* neurodevelopmental functions is conferred by interactions with other proteins (Zuko et al. 2011), including protein tyrosine phosphatase receptor gamma (PTPRG) (Bouyain and Watkins 2010) and amyloid precursor protein (APP) (Osterfield et al. 2008).

See Also

► [Contactin-Associated Protein 2](#)

References and Reading

- Bouyain, S., & Watkins, D. J. (2010). The protein tyrosine phosphatases PTPRZ and PTPRG bind to distinct members of the contactin family of neural recognition molecules. *Proceedings of the National Academy of Sciences of the United States of America*, 107(6), 2443–2448.
- Cottrell, C. E., Bir, N., Varga, E., Alvarez, C. E., Bouyain, S., Zernzach, R., et al. (2011). Contactin 4 as an autism susceptibility locus. *Autism Research*, 4(3), 189–199.
- Fernandez, T., Morgan, T., Davis, N., Klin, A., Morris, A., Farhi, A., et al. (2004). Disruption of contactin 4 (CNTN4) results in developmental delay and other features of 3p deletion syndrome. *American Journal of Human Genetics*, 74(6), 1286–1293.
- Fernandez, T., Garcia-Gonzalez, I., Mason, C., Hernandez-Zaragoza, G., Ledezma-Rodriguez, V., Anguiano-Alvarez, V., et al. (2008a). Molecular characterization of a patient With 3p deletion syndrome and a review of the literature. *American Journal of Medical Genetics*, 146A(21), 2746–2752.
- Fernandez, T., Morgan, T., Davis, N., Klin, A., Morris, A., Farhi, A., et al. (2008b). Disruption of contactin 4 (CNTN4) results in developmental delay and other features of 3p deletion syndrome. *American Journal of Human Genetics*, 82(6), 1385. (74, 1286, 2004).
- Glessner, J., Wang, K., Cai, G., Korvatska, O., Kim, C., Wood, S., et al. (2009). Autism genome-wide copy number variation reveals ubiquitin and neuronal genes. *Nature*, 459(7246), 569–573.
- Kamei, Y., Takeda, Y., Teramoto, K., Tsutsumi, O., Taketani, Y., & Watanabe, K. (2000). Human NB-2 of the contactin subgroup molecules: Chromosomal localization of the gene (CNTN5) and distinct expression pattern from other subgroup members. *Genomics*, 69(1), 113–119.
- Online Mendelian Inheritance in Man, OMIM®. McKusick-Nathans Institute of Genetic Medicine, Johns Hopkins University (Baltimore, MD), Dec 20, 2011. World Wide Web: <http://omim.org/entry/607280>
- Osterfield, M., Egelund, R., Young, L. M., & Flanagan, J. G. (2008). Interaction of amyloid precursor protein with contactins and NgCAM in the retinotectal system. *Development*, 135(6), 1189–1199.
- Roohi, J., Montagna, C., Tegay, D. H., Palmer, L. E., DeVincent, C., Pomeroy, J. C., et al. (2009). Disruption of contactin 4 in three subjects with autism spectrum disorder. *Journal of Medical Genetics*, 46(3), 176–182.
- Saito, H., Mimmack, M., Kishimoto, J., Keverne, E. B., & Emson, P. C. (1998). Expression of olfactory receptors, G-proteins and AxCAMs during the development and maturation of olfactory sensory neurons in the mouse. *Brain Research. Developmental Brain Research*, 110(1), 69–81.
- Shimoda, Y., & Watanabe, K. (2009). Contactins: Emerging key roles in the development and function of the nervous system. *Cell Adhesion & Migration*, 3(1), 64–70.
- Yoshihara, Y., Kawasaki, M., Tamada, A., Nagata, S., Kagamiyama, H., & Mori, K. (1995). Overlapping and differential expression of BIG-2, BIG-1, TAG-1, and F3: Four members of an axon-associated cell adhesion molecule subgroup of the immunoglobulin superfamily. *Journal of Neurobiology*, 28(1), 51–69.
- Zeng, L., Zhang, C., Xu, J., Ye, X., Wu, Q., Dai, J., et al. (2002). A novel splice variant of the cell adhesion molecule contactin 4 (CNTN4) is mainly expressed in human brain. *Journal of Human Genetics*, 47(9), 497–499.
- Zuko, A., Bouyain, S., van der Zwaag, B., & Burbach, J. P. (2011). Contactins: Structural aspects in relation to developmental functions in brain disease. *Advances in Protein Chemistry and Structural Biology*, 84, 143–180.

CNVs

► [Copy Number Variation](#)

Coaching Students with Autism

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Definition

The definition of coaching is the same when we consider it with reference to people with autism spectrum disorder (ASD) or the neurotypical population. In a sporting context, a coach is someone who uses sport as a vehicle for the development of individuals as performers and as people. Coaching is a form of teaching or instruction as it primarily involves communicating, learning, and maintaining positive relationships with those being taught. The aims of coaching are to understand: how individuals learn so that an effective learning environment can be created, to improve sports technique and develop skills, and to recognize areas for improvement, inside and outside the sporting realm.

Coaches' responsibilities include influencing the development of sport participants in five specific domains: technically (i.e., develop sport skills and abilities), physically (e.g., improve physical condition and develop good health habits), socially (e.g., enhance social skills of participants), psychologically (e.g., managing and controlling emotions), and personally (e.g., learn life skills). All people, regardless of race, ethnic minority, gender, sexual orientation, or ability should have equal access to sporting activities (United Nations Office on Sport for Development and Peace 2011). With this in mind, coaching should not simply be seen as relevant to competitive athletes or sport teams but to anyone with an interest in sporting activities, including the health, recreational, and pure fun dimensions of sport.

Historical Background

Participation in sport and physical activity (PA) provides multiple benefits for young people, including those living with ASD, and it is seen as useful to address a variety of the typical issues associated with ASD (Sowa and Meulenbroek 2012). Besides general fitness-related benefits, youth with ASD who participate in PA and sport may experience improved cognitive, psychological, behavioral, motor, and social functioning, which may contribute to reducing the typical social, emotional, communication, cognitive, and motor challenges faced by people with ASD (Sowa and Meulenbroek 2012). Furthermore, it is maintained that participation in sport and PA can also help young people with ASD – who tend to be considerably more sedentary than their neurotypical counterpart – to develop critical physical literacies, which may in turn equip them for lifelong participation (Smith and Patterson 2012). In this light, participating in sport and PA is “essential” for youth with ASD, “as it provides a sense of normalcy, allows the children to experience a fun activity with their peers, and develops important interpersonal skills” (Ohrberg 2013, p. 53).

However, it can be difficult for youth with ASD to participate in sports and PA, often due to the same issues that sport participation could seek to overcome. This can include low motivation, poor motor functioning, difficulties in self-monitoring, and socializing (Menear and Smith 2011). Sports typically require good eye-hand coordination, a sense of where one's body is in space, and the ability to track a high-speed object. Depending on where an individual lies on the autism spectrum, many young people with ASD may lack the motor coordination required to participate in sport programs aimed at the general population. Further, the social nature of many sports, including the prevalence of both verbal and nonverbal communication, has been identified as a barrier for participation among individuals with ASD. Another issue refers to the fact that, often, people with autism display markedly uneven ability profiles (especially when compared to the neurotypical population, who tend

to have fairly uniform abilities across all areas), typified by both impaired and spared emotional, motor, social, creative, cognitive, and sensory abilities (Boucher 2009). The typical challenges faced by people with ASD and the likelihood of significant differences between the abilities of individuals within any group of people with ASD make the role of the coaches extremely important in promoting greater involvement in sport and PA among this population.

Coaches assume the role of supporting and guiding individuals and teams when socializing them into sports, encouraging enthusiasm for a range of activities, and imparting knowledge and skills on fitness and healthy lifestyle choices. As such coaches, physical educators, and sport managers play a key role in planning, delivering, and monitoring quality programs for youth with ASD. While specialized training would help coaches to gain an appropriate understanding of ASD, coaches are often volunteers lacking access to specialized support and/or knowledge of effective practices to coach individuals with ASD, resulting in adverse consequences for their continued involvement in sport. In order to provide favorable environments that encourage and motivate individuals with ASD to engage in physical activity, programs need to emphasize adaptations to coaching techniques (from mainstream sport) which take into account the range of issues experienced by youth with ASD.

Current Knowledge

Enabling youth with ASD to maintain a physically active lifestyle is important, as it provides opportunities to learn fundamental movement skills, to enhance social interaction, and to develop lifelong habits that incorporate fitness into their daily routines. While studies using movement to “treat” youth with ASD have been conducted as far back as the 1970s (Best and Jones 1974), the use of sport as a tool to assist individuals with ASD to be both physically active as well as attain other potential benefits has received some focus in more recent time. For individuals on the autism spectrum, sport

participation has been found to help youth with ASD to develop fundamental physical literacies, which may equip them for lifelong participation (Smith and Patterson 2012). Adapted programs suited to the needs of youth with autism can elicit positive effects on social behavior (Pan 2010), academic engagement (Nicholson et al. 2011), as well as sensory skills (Bass et al. 2009).

In all sports programs, the role of the coach is extremely important as coaches need to create supportive environments to help participants to improve their skills, learn to relate to specific sports, enjoy peer companionship, and feel socially accepted in a sport setting. As such, it is imperative that coaches understand the individual needs, strengths, and weaknesses of youth with ASD (Ohrberg 2013). However, research has suggested that most physical education teachers and coaches have had limited to no formal training (i.e., workshops) in teaching young people with disabilities (Meegan and MacPhail 2006).

In addition, methods of how to coach children with ASD is often overlooked in the literature (Ohrberg 2013; Rosso 2016), resulting in limited resources for these coaches to draw upon when adapting their sport program. Despite the scarce literature base, several frameworks have been advanced as to how to adapt physical activity programs for individuals with autism. In one instance, it has been suggested that coaching strategies be informed by the Universal Design for Learning (UDL), a framework where curriculum and coaching methods are as flexible as possible to meet the needs of all learners (Sherlock-Shangraw 2013). UDL guidelines suggest that multiple means should be used for (a) representation, (b) action and expression, and (c) engagement. In regard to having multiple means of representation, adapted sport programs should use visual markers, have various means of content delivery, use cue words, have station activities, provide handouts, and attach new information to previous knowledge. Examples of having multiple means for action and expression include the use of white boards to deliver instructions and involve “start and stop” scrimmages to explain the rules of the sport. Last, strategies to provide multiple means of engagement include

developing strategies to set personal and group goals, building communities of practice, incorporating learners' interests into instruction delivery, and allowing for risk-taking while also maintaining a clear/consistent structure.

Another method which has been used to involve individuals with ASD into physical activity programs is the WPPR principle: Watch, Practice, Perform, Reward (MacPhail et al. 2014). In a sport setting, this would result in first watching the coach perform the activity being learned. This is followed by the individual practicing the task repeatedly with support and guidance from the coach. After some practice, the individual then performs the task to his/her highest ability alongside the coach, and if applicable, other children who also are performing the task. Last, individuals are rewarded for every successful goal reached, in order to develop intrinsic motivation.

Further, looking to the physical education literature, it has been suggested that physical education teachers should adapt their classes for individuals with disabilities and developmental disorders (including autism) in three different avenues: assessment, activity selection, and instructional and management techniques (Houston-Wilson 2016). These same principles can apply to coaching, as coaches should begin by carefully selecting the sporting activity that students will engage in. To select an appropriate sport activity, the most important consideration for the leader is to understand and meet the needs and interests of the learners. Further, the activities should allow a high probability of success for participants, and developmental appropriateness and age appropriateness should be considered when selecting activities. In relation to different instructional and management techniques, Houston-Wilson (2016) highlight that strategies that have been helpful in instructing students in the classroom are transferable to sport contexts. For example, using pictures and communication boards, having a consistent structure and routine, and using natural cues in the environment to facilitate the learning and execution of skills would be important for coaches to utilize.

Though these frameworks exist and provide a foundation for developing adapted sport programs, there is limited empirical research evidence which have actually highlighted the specific barriers that individuals with ASD face in a sport-specific setting, as well as how coaches can tailor their coaching strategies to overcome these barriers and meet the needs of participants (Rosso 2016). Given the gap in the literature surrounding how to coach individuals with ASD, recent research has begun to explore how coaches can successfully adapt their coaching techniques to meet the needs of students with autism (Duquette et al. 2016; Rosso 2016).

Several barriers to coaching individuals with ASD have recently been identified (Duquette et al. 2016; Rosso 2016). One particular barrier that can be frustrating when coaching individuals with ASD are the verbal communication issues that can occur between coach and student. Many children with autism do not speak at all, and others have echolalic speech such that they "echo" (repeat) what has been said without fully comprehending its meaning. Further, individuals with ASD often have difficulty understanding the rules, paying attention to instructions, anticipating, and elaborating good strategies. For instance, explaining activities and giving directions involving multiple instructions can be difficult for individuals with ASD to comprehend.

In order to overcome these communication challenges to engage individuals with ASD in sport, multiple strategies have been suggested by both coaches and parents (Duquette et al. 2016; Kraft 2016; Rosso 2016). These strategies include: (1) grabbing participants' attention by saying their name, (2) adapting the instructions (e.g., give less information, give one instruction at a time), (3) utilize cue words that become familiar to participants over time, (4) make sure the participant understands instructions before they participate, (5) use gestures and/or physically perform the behavior to demonstrate, and (6) reward explanations through repetition and the use of visual cues (e.g., pictograms, gestures). Further, the use of positive reinforcement techniques when celebrating the successes of participants was thought to enhance the participants'

enjoyment and engagement in the sporting activity. These can include both verbal communication (e.g., praise) and explicit physical displays of approval (e.g., high fives, fist bumps, victory dances).

Another challenge that exists relates to individuals' gross motor functioning, as some young people with ASD show a lack of coordination, balance, and strength compared with those without autism (Duquette et al. 2016). However, as ASD manifests itself differently in each person, coaches may be involved with teaching multiple abilities at the same time. As such, the coaching strategies used for one participant may not work for another. To combat these challenges, it has been suggested that coaches provide micro-adaptations within each activity and be flexible in adapting to the needs of the individual (e.g., altering the size of equipment or the distance of a kick/pass according to each participant's abilities, Rosso 2016).

Another challenge faced by coaches is determining an ideal coach-participant ratio, particularly in group sport environments. While some participants require one-on-one support in order to understand the basic concepts behind certain sporting tasks, this may not be feasible from a resources perspective in a group setting when trying to engage multiple participants at the same time. A coach-participant ratio of 1–2 or 1–3 has been suggested as an appropriate target in team/group settings, in order to ensure there is individualized support throughout the sport programming when required, while also providing a reasonable degree of interaction with peers (Rosso 2016). However, for young people with ASD, the social environment can also act as a barrier to participation in sport (Duquette et al. 2016). The attitude and reactions of coaches, parents, and peers can all affect an individual with ASD. Thus, retaining a consistent and predictable structure/routine each session and designing activities organized in stations can help to overcome this barrier.

In summary, recent studies have highlighted coaches should use simple instructions, adopt a step-by-step approach, have suitable adaptations,

develop clear session plans, be consistent and predictable in their coaching yet flexible enough to allow late modifications if required, and often remind and check with athletes what has been learnt and what has been forgotten (Duquette et al. 2016; Kraft 2016; Rosso 2016).

Future Directions

While some research and advice has been developed regarding sport coaching for youth with ASD, there remain a number of areas that require further attention. This includes the development of specialized ASD training (sport education program) for coaches involved in adapted sport programs. Adopting a more focused approach to coach training can assist both those involved in coaching as well as provide quality sporting experiences for youth with ASD. Further to this, sporting organizations should develop sport coaching opportunities and pathways for youth with ASD. Rather than focus on youth as participants of coaching, sporting organizations should be exploring the potential of youth with ASD to be coaches. This would not only provide a further expansion of the potential career pathways for youth with ASD in sport but would also provide the opportunity for younger youth with ASD to be mentored within sport by their older peers.

In relation to research, there is a need to create and promote a clearinghouse of “best practice” coaching techniques, tools, and frameworks. While there are some examples of how sport coaching can be adapted effectively for youth with ASD, this is scattered across a variety of publication types (i.e., peer reviewed journals and grey literature). The development of a clearinghouse of case studies would have the potential of further informing and improving sport coaching for youth with ASD.

Another key aspect required regarding research into sport coaching for youth with ASD is the need for longitudinal research. As Sorensen and Zarrett (2014) highlight, there is a need to identify effective interventions concerning youth with ASD in regard to sport and physical activity. However,

much of the research focusing on sport coaching for youth with ASD has been cross-sectional studies within limited time spans. Thus, there is a need to explore the impact of sport coaching on youth with ASD concerning social, behavioral, and educational changes over time.

See Also

- [Australia and Autism](#)
- [Autism: Social Communication Disorder](#)
- [Gross Motor Skills](#)

References and Reading

- Bass, M. M., Duchowny, C. A., & Llabre, M. M. (2009). The effect of therapeutic horseback riding on social functioning in children with autism. *Journal of Autism and Developmental Disorders*, 39, 1261–1267.
- Best, J. F., & Jones, J. G. (1974). Movement therapy in the treatment of autistic children. *Australian Occupational Therapy Journal*, 21, 72–86.
- Boucher, J. (2009). *The autistic spectrum: Characteristics, causes and practical issues*. Thousand Oaks: Sage.
- Duquette, M.-M., Carboneau, H., Roult, R., & Crevier, L. (2016). Sport and physical activity: Facilitating interventions with young people living with an autism spectrum disorder. *Physical Activity Review*, 4, 40–49.
- Houston-Wilson, C. (2016). Autism spectrum disorder and social communication disorders. In J. Winnick & D. Poretta (Eds.), *Adapted physical education and sport* (6th ed., pp. 197–214). Champaign: Human Kinetics.
- Kraft, E. (2016). *Exploring the experiences of coaching children with Autism Spectrum Disorder in Canadian aquatic programs*. Master's thesis. Retrieved from the University of Ottawa. <http://www.ruor.uottawa.ca/handle/10393/35495>.
- MacPhail, A., Campbell, M., Kenny, I., Tindall, D., & Teannehill, D. (2014). Watch, practice, perform, reward: Meeting (special) learning needs. In K. Armour (Ed.), *Pedagogical cases in physical education and youth sport* (pp. 29–62). Oxford: Routledge.
- Meegan, S., & MacPhail, A. (2006). Irish physical educators' attitude toward teaching students with special education needs. *European Physical Education Review*, 12, 75–97.
- Meneer, K. S., & Smith, S. C. (2011). Teaching physical education to students with Autism Spectrum disorders. *Strategies*, 24, 21–24.
- Nicholson, H., Kehle, T., Bray, M., & van Heest, J. (2011). The effects of antecedent physical activity on the academic engagement of children with autism spectrum disorder. *Psychology in the Schools*, 48, 198–213.
- Ohrberg, N. (2013). Autism Spectrum disorder and youth sports: The role of the sports manager and coach. *Journal of Physical Education, Recreation and Dance*, 84, 52–56.
- Pan, C. (2010). Effects of water exercise swimming program on aquatic skills and social behaviors in children with autism spectrum disorders. *Autism*, 14, 9–28.
- Rosso, E. (2016). Brief report: Coaching adolescents with Autism Spectrum disorder in a school-based multi-sport program. *Journal of Autism and Developmental Disorders*, 46, 2526–2531.
- Sherlock-Shangraw, R. (2013). Creating inclusive youth sport environments with the universal design for learning. *Journal of Physical Education, Recreation & Dance*, 84, 40–46.
- Smith, V., & Patterson, S. Y. (2012). *Getting into the game: Sport programs for kids with autism*. London: Jessica Kingsley Publishers.
- Sorensen, C., & Zarrett, N. (2014). Benefits of physical activity for adolescents with autism spectrum disorders: A comprehensive review. *Review Journal of Autism and Developmental Disorders*, 1, 344–353.
- Sowa, M., & Meulenbroek, R. (2012). Effects of physical exercise on Autism Spectrum disorders: A meta-analysis. *Research in Autism Spectrum Disorders*, 6, 46–57.
- United Nations Office on Sport for Development and Peace (2011). *Achieving the objectives of the United Nations through sport*. Geneva: Publishing Services, United Nations.

Cochlea

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Synonyms

[Inner ear](#)

Definition

The cochlea is the auditory portion of the inner ear which is embedded in the temporal bone. It consists of membranes, fluids, hair cells, and VIII

nerve endings. The organ of Corti, housed in the snail-shaped cochlea, contains many cells, including the inner and outer hair cells, and is responsible for converting mechanical energy from the middle ear into electrical energy. That energy is passed to auditory nerve fibers.

See Also

- Auditory Acuity
- Auditory System
- Hearing

References and Reading

- Clarke, W., & Ohlemiller, K. (2008). *Anatomy and physiology of hearing for audiologists*. Clifton Park: Thomson, Delmar Learning.
- Musiek, F. E., & Baran, J. A. (2007). *The auditory system: Anatomy, physiology, and clinical correlates*. Boston: Pearson.

Codic (Verbal Behavior)

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Skinner (1957) proposed a system of classifying verbal behavior, by function and form, resulting in discrete units of analysis termed *verbal operants*. The taxonomy is based on the forms of the antecedent controlling stimuli and response products and the specific consequences provided for each operant. Skinner's original categorization included the following operants: *echoic*, *mand*, *tact*, and *intraverbal*. However, these categories of behavior do not encompass all forms and functions of communication and language. Two additional categories have since been proposed: *codic* and *duplic* (Michael 1982).

The form of a verbal operant is said to have *point-to-point correspondence* when the individual components (e.g., letters, syllables) of the antecedent stimulus and response product forms match. And, when the sense mode of the stimulus and response is the same (e.g., auditory stimulus and vocal response), they exhibit *formal similarity*. The function of each verbal operant is determined by controlling consequences or motivating operations (*mand* only). Controlling consequences are changes in stimulus conditions that follow the occurrence of verbal operants (e.g., praise), whereas motivating operations are environmental variables that alter the effectiveness of some stimulus as a reinforcer and alter the frequency of behavior that has been reinforced by that stimulus (e.g., hunger).

A *codic* is a verbal operant in which the antecedent stimulus and response product forms exhibit point-to-point correspondence, there is no formal similarity, and that is reinforced by generalized conditioned reinforcement from a listener (usually a parent/guardian or teacher). Common examples of *codics* include reading written words (textual (In the case of the *textual*, reading means "decoding" words and not comprehension)) or writing words that one hears spoken aloud (taking dictation). *Codics* are learned through both direct and incidental teaching strategies and are reinforced by the learner's verbal community with praise or some form of educational reinforcement.

References and Reading

- Michael, J. (1982). Skinner's elementary verbal relations: Some new categories. *The Analysis of Verbal Behavior*, 1(1), 1–3. <https://doi.org/10.1007/BF03392791>.
- Skinner, B. F. (1957). *Verbal behavior*. New York: Appleton-Century-Crofts.

Coefficient Alpha

- Cronbach's Alpha

Cognitive Behavioral Therapy (CBT)

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Definition

Cognitive behavioral therapy (CBT) treatments are based upon cognitive science models of the mind and mental life. Contemporary CBT treatments have been particularly influenced by the memory retrieval competition model (e.g., Brewin 2006). Conceptualized in information processing terms, CBT aims to promote *retrievable memories* of adaptive responses that can successfully compete with and suppress memories of previously learned maladaptive responses evoked under “real-world” conditions outside the therapy office. This process leads to an increase of

realistic appraisals of the environment and the self, more appropriate behavior, and greater capacity to regulate strong emotion.

Historical Background

Cognitive behavioral therapy is rooted in traditions of behavioral and cognitive (or rational) therapy. During the early behavioral learning theory experiments of the early twentieth century, many of which were conducted with animal subjects (e.g., by Pavlov, Thorndike, and Hull), several case studies were conducted with fear conditioning in children. Watson and Rayner (1920) and Mary Cover Jones (1924) used applications of classical conditioning to induce or eradicate fears (phobias) in young children. These are generally considered the earliest behavior therapy cases (Rachman 1997). Skinner, with the advent of radical behaviorism, popularized applications of operant conditioning for modifying human behavior; this was quickly applied to a variety of developmental and psychiatric conditions including autism (e.g., Ferster and Demyer 1962; Lovaas et al. 1971), with some early successes in basic abilities such as imitation and language use. Concurrent with these developments in behavior therapy, prominent researchers were publishing data suggesting that traditional psychodynamic psychotherapies were proving ineffective with individuals with autism (e.g., Kanner and Eisenberg 1955; Rutter 1968). Behavior therapy remains a staple component of modern CBT, including CBT treatments for people with autism.

Cognitive and rational therapies (referred to as cognitive therapy for the remainder of this entry, for simplicity) emerged in the context of the “cognitive revolution” in psychology, which rejected the thesis of radical behaviorism that mental states, such as thoughts, were unmeasurable and inconsequential. Cognitive psychology, with models of attention, memory, perception, and language, and their integration, underlies the original cognitive therapies developed by Ellis (1957) and Beck (1963). A core principle of cognitive therapy is that certain “errors” of thought

(misperceptions, misattributions, all-or-nothing thinking, etc.) lead to inappropriate emotional reactions, behaviors, or both. The underlying model is often referred to as a cognitive appraisal theory of emotion (cf. Lazarus 1991); the idea being that thought in its various forms drives emotion and behavior. A key therapeutic technique in the early cognitive therapies was the use of logic to identify and challenge faulty thought processes. The assumption was that irrational thoughts would be “replaced” with more rational ones, leading to improved emotional states and behavioral responses. Indeed, early research on cognitive therapies offered evidence of good treatment response in terms of symptom reduction, particularly with regard to emotion disorders such as anxiety and depression (Beck 1970; Watkins 1975). Relatively little cognitive therapy research was conducted with individuals on the autism spectrum in this early period.

As the cognitive and behavioral traditions merged into integrative CBT packages, core methods used to achieve symptom remission emerged: psychoeducation (learning about the nature of one’s mental health condition), Socratic questioning and collaborative discussions to build up awareness of thought and emotion and to teach thought- and behavior-based coping skills, and behavioral experimentation, in which alternative responses to challenging situations are attempted in real-world settings and then reflected upon in structured discussions in order to build up potent memories of adaptive patterns of thought and behavior for future use in similar (not necessarily identical) situations (e.g., Brewin 2006). At present, there is increasing interest in the application of CBT to individuals with ASD, as described below.

Rationale or Underlying Theory

With the increasing fund of knowledge emanating from cognitive science research in recent decades, a number of critical findings were published that helped refine the model of mental states in cognitive psychology (see, e.g., Anderson et al. (2004) model of the architecture of the mind).

A generally accepted view from the modern literature is that long-term memories can be nearly indelible and that it is common to have multiple, competing memory representations (schemata) of the same event or concept (Brewin 2006). Hence, rather than “replacing” maladaptive thoughts with adaptive ones, it is more likely that new adaptive thoughts can be learned and strengthened using certain memory enhancement techniques, and, under certain conditions, these new memories can partially or fully suppress older maladaptive memories. Promoting such learning is, in fact, a fundamental goal of contemporary cognitive therapy (e.g., Brewin 2006). Nonetheless, the older maladaptive memories are likely still accessible and can return to consciousness under certain conditions (e.g., Rodriguez et al. 1999) underscoring the “indelibility” of many memories and the challenge facing therapists endeavoring to help individuals cope more effectively and employ more useful skills under varied conditions. These properties of human memory have led cognitive therapists to a memory retrieval competition model of therapy that endeavors to enhance recall of beliefs and skills under challenging conditions that promote adaptive responses (Brewin 2006).

Although cognitive and behavioral therapies were developed in parallel in the middle of the twentieth century, cognitive therapy had behavioral elements right from the beginning. For example, clients were encouraged to challenge their negative views about specific situations by engaging in “behavioral experimentation” and testing these situations out in person (e.g., initiating a brief conversation with others in a social group in which one feels like an outsider to test whether one will be rejected). Although such strategies involve behavioral change, the goal is not specifically to develop a new response linked with a given condition (e.g., a behavioral view of clinical change) but rather to acquire new information that can promote adaptive beliefs about the situation that, with sufficient rehearsal, can occlude the current unrealistic, negative perceptions of the situation and therefore change behavior.

Although behaviorism and the cognitive science perspective emphasize different aspects of

human learning and memory in the service of behavioral change, the two approaches are not necessarily at odds. The extreme view of radical behaviorism, rejecting the importance of mental states, is not commonly practiced in modern psychology, even among those who subscribe to behaviorism as a primary explanatory and clinical tool. Fundamentally, learning via association and learning via higher cognitive processes (e.g., conceptual reasoning) are complementary and may occur either in serial or in parallel in any given learning situation. Cognitive behavioral therapy, accepting this affinity between the behavioral and cognitive perspectives, sought to capitalize on all effective means of human learning – tailored to the cognitive and developmental level of a given population – to promote clinical change. As a result, often multiple treatment techniques spanning both associationist and cognitivist approaches to clinical change are combined into CBT treatment “packages” that layer multiple forms of learning together. Examples are CBT programs for anxiety disorders that involve a number of thought-based approaches to change (e.g., cognitive restructuring; mindful awareness) married together with associationist approaches such as gradual exposure to feared stimuli and self-reward (e.g., Kendall 1994) and CBT programs for depression, which combine identifying and challenging “erroneous” ways of thinking (e.g., all-or-nothing thinking) with behavioral techniques like self-modeling and pleasant activity scheduling (e.g., Weisz et al. 1999). These two basic modalities of learning are thought to complement one another and promote more comprehensive formation of adaptive memories.

Goals and Objectives

CBT is intended to treat a wide variety of mental health and developmental disorders, although the target population is expected to have some capacity to communicate and, thus, benefit from mainline cognitive techniques (in contrast, many purely behavioral therapies do not assume substantial functional communication, particularly those used in the treatment of ASD). Historically,

the most common targets of treatment for CBT have been mood and anxiety disorders (including obsessive-compulsive disorder; OCD), with high levels of evidence for treatment efficacy in both conditions (Butler et al. 2006; James et al. 2005). There are CBT-based programs for many other conditions as well, with somewhat more tentative levels of support due to either a lack of large, rigorous studies or mixed results. Disruptive behavior disorders, habit disorders, substance use disorders, psychotic disorders, and personality disorders are among the other conditions commonly treated with CBT. Autism spectrum disorders have also been the target of some recent CBT programs. Most of these latter programs have focused on associated symptoms (e.g., anxiety) in individuals with ASD (e.g., Sofronoff et al. 2005); several have targeted core autism symptoms (e.g., social engagement) as well (e.g., Wood et al. 2009a).

As noted in the section on rationale, contemporary CBT aims to build up the strength of memory for adaptive responses – thoughts, emotions, and behaviors – when individuals are in challenging situations that typically elicit maladaptive responses. Here are some examples of maladaptive responses of various types, particularly those that are commonly seen in ASD (see, e.g., case studies presented in Sze and Wood 2007, 2008; Lehmkuhl et al. 2008):

- The belief that others are scrutinizing one’s every move (leading to anxiety, dysfluency, tentativeness, and reticence)
- A belief that life is not worthwhile because it is full of challenges (leading to sadness or irritability, retreat into comforting but solitary activities like electronics, or self-harm)
- The belief that nothing short of perfect is good enough, so why try if that is not attainable (leading to avoidance of specific tasks viewed in this light and, often, conflict and anger stemming from social pressure to make an effort anyway)
- The belief that rules are inviolable and absolute (leading to behaviors such as tattletaling as well as the sadness that often ensues after experiencing rejection from others regarding

this behavior or telling on oneself as often seen in pediatric OCD and the anxiety that accompanies that behavior)

- An individual's belief that others are uninterested in talking or playing with him/her because they have not always responded positively to the individual's ideas in the past (leading to anxiety and social avoidance)
- A corresponding belief that playing with others is mostly about getting to choose the game and set the rules, winning, and having the longest possible turn, rather than enjoying an activity together and the sociability that goes with it (leading to egocentric, self-oriented behavior, poor sportsmanship, and, often, ultimately sadness at the negative social feedback that can ensue)

With regard to these types of maladaptive responses, CBT aims to help individuals challenge faulty assumptions by using logic, evidence, and direct observation to arrive at more realistic conclusions and alter behavior accordingly. With the adoption of realistic beliefs and corresponding behaviors often comes emotional relief. For example, in response to anxious beliefs about being scrutinized by others, many CBT programs would encourage an individual to challenge these beliefs by using logic and simple behavioral experiments (e.g., tripping on purpose in public or other "mistakes" to test what types of reactions one receives) to foster new attitudes (e.g., "people really do not care about a lot of minor mistakes you might make, and as for big ones, it is their problem if they laugh at you, not yours") and corresponding behaviors (e.g., taking more risks in public) (e.g., Kendall 1994). Improvement in emotional states is expected to follow successful acquisition of these kinds of adaptive beliefs and behaviors. Some forms of CBT also involve adopting a disengaged attitude with regard to one's negative emotions – an approach that aims to change cognitive appraisal of emotion to reduce the intensity and aversiveness of states such as sadness, anxiety, and perseverative thought (Ost 2008).

A fundamental difference between CBT and strictly behavioral treatments (e.g., operant or classical conditioning-based models) is the

conceptualization of mechanisms of change and complementary intervention techniques. While purely behavioral interventions assume that largely automatic (and unobservable) learning processes (e.g., extinction, associative learning, modeling) promote behavioral change and symptom remission, CBT-based models seek to promote changes in thinking and volitional behavior (e.g., identifying and challenging maladaptive interpretations of social situations) that are adaptable to multiple situational contexts. A simple example of phobia treatment illustrates differences between CBT and purely behavioral approaches: in the former, catastrophic beliefs about a feared stimulus would be identified and challenged to build up to facing the phobic stimulus, and after habituation occurs, the therapy would promote the development of principles for thinking about the feared stimulus differently to build a benign memory schema of the stimulus that could compete with and suppress the fearful schema that the patient had prior to treatment (e.g., Wood and McLeod 2008). In contrast, a purely behavioral approach would involve gradual exposure to a feared stimulus to achieve extinction of the conditioned (fearful) response with no emphasis on related thoughts, and when fear and avoidance were eliminated in one setting, the procedure might be repeated in several other settings in an effort to achieve generalization. Clearly, the putative learning processes and corresponding techniques used to promote change differ significantly in these two types of treatments (further description of CBT technique is given below).

Treatment Participants

CBT has been found efficacious across a wide range of populations, particularly in neurotypical groups. Typical pediatric and adult populations affected by depression, anxiety, and other psychiatric conditions have shown good treatment response with few limitations (Norton and Price 2007; Weisz et al. 2006; In-Albon and Schneider 2007). Although numerous studies of predictors of treatment response have been conducted, few

consistent trends have emerged. The method of delivery is undoubtedly influential, however. The younger the children are, the more likely they are going to need parent involvement. Children with ASD also appear to do better with parent involvement than with child-only CBT (Sofronoff et al. 2005; Puleo and Kendall 2011). No reliable differences have been found with regard to gender or comorbidity. It is assumed that greater intellectual ability should promote greater understanding of the cognitive therapy aspects of treatment, all other things being equal, but this has not been studied carefully. Furthermore, appropriate modifications of treatment for individuals with intellectual disabilities may make CBT accessible and comparable in effectiveness across a wide range of intellectual functioning (Suveg et al. 2006).

Treatment Procedures

CBT is generally presented in an interactive tutorial format, with a lesson plan, some form of instruction, practice, and review. Although it is relatively structured, CBT is responsive to client characteristics, interests, and level of engagement. Forming a positive alliance with the client is an ongoing goal in CBT to enhance the client's motivation to learn and use skills (e.g., Chiu et al. 2009). CBT for mood- and anxiety-related problems typically involves teaching cognitive and behavioral skills followed by a skills practice phase in simulated and "real-world" situations (e.g., Kendall 1994). In one of the more influential clinical trials of CBT for pediatric anxiety disorders in typically developing youth, Kendall et al. (1997) found that the *cognitive* intervention aspects of the treatment (e.g., challenging irrational beliefs) alone were not effective in reducing children's anxiety levels. Only when cognitive training was paired with in vivo exposure elements (facing fears rather than avoiding them) did CBT become an efficacious intervention.

For individuals with ASD and concurrent anxiety, the general CBT approach of challenging irrational fearful beliefs and developing rational beliefs is employed, although other elements of treatment have varied widely from program to

program. In these programs for individuals with ASD, there has been wide variation with regard to the emphasis placed on in vivo exposure relative to other treatment elements (e.g., cartooning, role-playing). At the extremes of the continuum, the Wood et al. (2009b) program involves in vivo exposure at home on a daily basis for the majority of the 16-session treatment, which spans 4–5 months for most youth; whereas the Sofronoff et al. (2005) 6-session treatment focuses on a series of creative anxiety management skills tailored for youth with ASD, but with no explicit in vivo exposure elements. Some (but not all) CBT trials conducted with typically developing children and youth with anxiety disorders (e.g., Barrett et al. 1996; Barrett 1998; Wood et al. 2006) have found that including parent training in the intervention leads to superior intervention effects as compared to exclusively child-focused treatments. Many of the group design studies for youth with ASD and high anxiety have included concurrent child- and parent-intervention components.

The majority of the treatment programs that have been studied for individuals with ASD have used a group-therapy treatment format with a structured sequence of sessions for all participants. Others have used an individual therapy format with modular design (see Chorpita et al. 2004) in which individual treatment components were selected by the therapist and supervisor on a session-by-session basis using a clinical algorithm matching the client's presenting characteristics with corresponding treatment elements (e.g., Wood et al. 2009a, 2009b). As an example, a child who was socially anxious at school would receive a social coaching module in which social approach behaviors are broken down into steps, anxious beliefs about each step are discussed and challenged, and then steps are practiced in various real-world settings such as parks and school playgrounds repeatedly until a sufficiently advanced level of the skill (e.g., joining recess games without fear) is evidenced consistently. No clinical trials thus far have compared the relative efficacy of structured group-format CBT interventions with individually administered, modularized interventions of this kind.

Anger and aggression have been the target of some CBT programs, particularly in neurotypical youth (e.g., Kazdin 2005). In a randomized controlled trial conducted by Sofronoff et al. (2007), a CBT program was devised to address anger problems in youth with ASD. Treatment consisted of six weekly 2-h sessions for both child and parent. The manualized therapy sessions focused on exploring positive and negative emotions, cognitions related to coping with anger, social stories to promote emotion management, and designing individualized coping plans for anger management.

With regard to treatments for ASD symptoms – such as social deficits – only a few programs have been developed within a CBT framework. Bauminger (2002) developed a school-based CBT program, noting that maladaptive cognition accounts for some of the interpersonal behavior in youth with ASD and that, therefore, adaptive alterations to cognitive structures could make a positive impact on interpersonal behavior. In the Bauminger (2002) intervention, several elements are notable: children's classroom teachers are responsible for a 3-h-per-week, 7-month intervention conducted at school that relies heavily on guiding a dyad consisting of the target child and a typically developing peer through a series of 13 social skill lessons (e.g., cooperating) that are to be practiced at recess, on the phone, on playdates, and so forth. Parents are also asked to support children in learning and implementing these social skills. The intervention was presented by the teacher to the dyad, allowing for individualization (e.g., by having pairs of children choose activities that they both liked). The Wood et al. (2009a, b) program, described above, also addresses social and repetitive symptoms of ASD, integrating these target symptoms into the treatment hierarchy with emotion-related symptoms.

Efficacy Information

CBT treatments for anxiety in ASD have been generally successful. Sofronoff et al. (2005) evaluated two variants of a 6-week CBT program for

anxiety in youth with ASD in group-therapy format. Parent-report measures showed declines in child anxiety symptoms in the CBT groups as compared to a wait list group. Similarly, in 12- and 16-week group-therapy CBT interventions for comorbid anxiety and ASD in children, Chalfant et al. (2007) found that anxiety outcomes were superior for the immediate treatment group relative to the wait list arm. However, limitations of these studies were that the study therapists, rather than independent evaluators blind to treatment assignment, administered the posttreatment diagnostic interviews and that treatment fidelity was not assessed. Reaven et al. (2009) studied 33 children (aged 8–14 years) with ASD and comorbid anxiety disorders and, using a nonrandomized assignment paradigm, assigned them to immediate treatment in group-therapy format CBT or a wait list. Outcome measures were child- and parent-reported anxiety symptoms using psychometrically sound questionnaires. Youth in the immediate treatment group improved more than the waiting list group on parent-reported symptoms, but not child-reported symptoms. This may have been attributable to low pretreatment child-report symptom scores.

White et al. (2009) examined a manualized 11-week CBT program for the treatment of anxiety in four youth aged 12–17 years with high-functioning ASD. The program consisted of individualized therapy sessions with high levels of parent involvement and group-therapy sessions mainly for the teaching and practicing of social skills. At posttreatment, three of the four adolescents no longer met diagnostic criteria for their targeted anxiety disorder and showed significant reductions in anxiety based on parent report. In a study of a manualized, individualized CBT program, 40 children aged 7–11 years were randomized to either 16 sessions of CBT or a wait list (Wood et al. 2009b). Participating children had an average of 4.18 psychiatric disorders at intake. Despite the high level of comorbidity, children randomized to CBT had primary outcomes comparable to those of other studies treating childhood anxiety in typically developing patients (see, e.g., Barrett et al. 1996; Wood et al. 2006), with large effect sizes for most outcome measures;

remission of all anxiety disorders for over half of the children by posttreatment or follow-up; and a high rate of positive treatment response on the Clinical Global Impressions–Improvement scale (CGI-I) (78.5% from intent-to-treat analyses). As with the Reaven et al. (2009) study, child-reported anxiety did not differ significantly from pre-treatment to follow-up; however, a floor effect was expected, as baseline levels were low and decreased with treatment.

In the Wood et al. (2009a) CBT intervention ($N = 19$), there was also a statistically significant difference between the CBT group and the wait list group at posttreatment/post-wait-list on total parent-reported autism symptoms on the Social Responsiveness Scale, with a medium to large effect size. Treatment gains were maintained at 3-month follow-up. Of course, this study was limited by a small sample and reliance on parent reports of symptomatology, which are vulnerable to bias. Evidence-based assessments of core autism symptoms based on independent evaluators' ratings and direct observations of children's behavior (e.g., the ADOS) should be employed in future studies of CBT programs like this one to determine their potential for reducing the expression and severity of core autism symptoms.

In the Sofronoff et al. (2007) anger management CBT program, there was a significant reduction in the number of parent-reported anger episodes after treatment in the immediate intervention group, with gains maintained 6 weeks after treatment completion. Qualitative interviews conducted with participants' teachers post-treatment revealed participants' use of strategies they had learned through the program to manage their anger within their classroom. One methodological weakness in this study was that no diagnostic criteria or operational definition of an externalizing disorder was used for case selection at pretreatment. In addition, all outcome measures were parent-report, with the exception of the qualitative interviews with teachers.

In the Bauminger (2002, 2007a) individual interventions for social deficits in ASD, a pre-post design without control group was used, and children approximately doubled their number of observed positive social interchanges with peers

in naturalistic observations at school – particularly eye contact, expressions of interest in others, and talking about their own experiences. They were more likely to initiate positive interactions than they were to respond positively to peers' initiations to them. Teachers also rated children as improved in certain positive social skills on the Social Skills Rating Scale. A 4-month follow-up assessment provided evidence of maintenance of treatment effects (Bauminger 2007a). This treatment model is promising and merits a more thorough evaluation in a randomized trial.

A group-therapy CBT treatment (with 3–6 children per group, at least half of whom were typically developing) with many commonalities with the Bauminger (2002) intervention, but focusing more on within-group interaction as a vehicle for learning, was also evaluated by Bauminger (2007b). Again, an AB design was used ($N = 26$). While there was substantial improvement in social behaviors among the therapy group members while interacting during the sessions from pre- to posttreatment, this effect did not generalize to the playground setting, in which no significant improvement was found in social behaviors over the course of the 7-month interval from baseline to posttreatment. Since Bauminger essentially adapted the therapeutic concepts and methods from her more individually oriented CBT interventions (Bauminger 2002, Bauminger 2007a) for this group-therapy trial, it is worth considering whether there is more merit in individually oriented social interventions in autism (if, as Bauminger notes, the child's ecological influences are addressed through the individual intervention), as compared to group-based interventions, than has traditionally been assumed.

Outcome Measurement

In defining desirable study features for research intended to establish efficacious treatments, Chambless and Hollon (1998) noted that it was important that valid and reliable measures of symptom counts or diagnostic status, preferably including those rated by an evaluator blind to treatment status and study hypotheses, be used

as primary outcome measures. Of the small number of controlled trials of CBT for individuals with ASD, most have included this kind of measure. Many of these have focused on comorbid mental health features such as anxiety (e.g., Chalfant et al. 2007), and one of these trials utilized a parent-rated measure of core autism symptoms that is norm-referenced and used in the diagnosis of ASD (Wood et al. 2009a). The Bauminger studies also used observational measures in the school setting, an objective measure with high ecological validity. Many of the studies of CBT in ASD have also used parent-, teacher-, or self-report, which are useful as secondary indices of treatment outcome but may be biased by expectancy effects and other sequelae of being unblinded.

Qualifications of Treatment Providers

Practitioners are generally licensed clinical psychologists with a doctoral degree in psychology. However, other professionals with licensure to provide psychotherapy to children (e.g., school psychologists, licensed clinical social workers, child and family therapists) with appropriate training can conduct CBT treatments. Regardless of specific license and degree, a specialization in individuals with ASD and specific training (including one-on-one supervision) in CBT for individuals with ASD are essential qualifications for the professional delivery of this type of treatment.

References and Reading

- Anderson, J. R., Bothell, D., Byrne, M. D., Douglass, S., Lebiere, C., & Qin, Y. (2004). An integrated theory of mind. *Psychological Review*, 111(4), 1036–1060.
- Barrett, P. M. (1998). Evaluation of cognitive-behavioral group treatments for childhood anxiety disorders. *Journal of Clinical Child Psychology*, 27, 459–468.
- Barrett, P. M., Dadds, M. R., & Rapee, R. M. (1996). Family treatment of childhood anxiety: A controlled trial. *Journal of Consulting and Clinical Psychology*, 64, 333–342.
- Bauminger, N. (2002). The facilitation of social-emotional understanding and social interaction in high-functioning children with autism: Intervention outcomes. *Journal of Autism and Developmental Disorders*, 32, 283–298.
- Bauminger, N. (2007a). Brief report: Individual social-multi-modal intervention for HFASD. *Journal of Autism and Developmental Disorders*, 37, 1593–1604.
- Bauminger, N. (2007b). Brief report: Group social-multimodal intervention for HFASD. *Journal of Autism and Developmental Disorders*, 37, 1605–1615.
- Beck, A. (1963). Thinking and depression: Idiosyncratic content and cognitive distortions. *Archives of General Psychiatry*, 9(4), 324–333.
- Beck, A. (1970). Cognitive therapy: Nature and relation to behavior therapy. *Behavior Therapy*, 1(2), 184–200.
- Brewin, C. R. (2006). Understanding cognitive behaviour therapy: A retrieval competition account. *Behaviour Research and Therapy*, 44, 765–784.
- Butler, A. C., Chapman, J. E., Forman, E. M., & Beck, A. T. (2006). The empirical status of cognitive-behavioral therapy: A review of meta-analyses. *Clinical Psychology Review*, 26(1), 17–31.
- Chalfant, A. M., Rapee, R., & Carroll, L. (2007). Treating anxiety disorders in children with high functioning autism spectrum disorders: A controlled trial. *Journal of Autism and Developmental Disorders*, 37, 1842–1857.
- Chambless, D. L., & Hollon, S. D. (1998). Defining empirically supported therapies. *Journal of Consulting and Clinical Psychology*, 66, 7–18.
- Chiu, A. W., McLeod, B. D., Har, K., & Wood, J. J. (2009). Child-therapist alliance and clinical outcomes in cognitive behavioral therapy for child anxiety disorders. *Journal of Child Psychology and Psychiatry*, 50, 751–758.
- Chorpita, B. F., Taylor, A. A., Francis, S. E., Moffitt, C., & Austin, A. A. (2004). Efficacy of modular cognitive behavior therapy for childhood anxiety disorders. *Behavior Therapy*, 35, 263–287.
- Ellis, A. (1957). Outcome of employing three techniques of psychotherapy. *Journal of Clinical Psychology*, 13, 344–350.
- Ferster, C. B., & Demyer, M. K. (1962). A method for the experimental analysis of the behavior of autistic children. *The American Journal of Orthopsychiatry*, 32(1), 89–98.
- In-Albon, T., & Schneider, S. (2007). Psychotherapy of childhood anxiety disorders: A meta-analysis. *Psychotherapy and Psychosomatics*, 76, 15–24.
- James, A., Soler, A., & Weatherall, R. (2005). Cognitive behavioral therapy for anxiety disorders in children and adolescents. *Behaviour Change*, 22(2), 55–70.
- Jones, M. C. (1924). A laboratory study of fear: The case of peter. *Pedagogical Seminary*, 31, 308–315.
- Kanner, L., & Eisenberg, L. (1955). Notes on the follow-up studies of autistic children. In P. H. Hoch & J. Zubin (Eds.), *Psychopathology of childhood* (pp. 227–239). Oxford, UK: Grune & Stratton.
- Kazdin, A. (2005). Child, parent, and family-based treatment of aggressive and antisocial child behavior. In

- E. D. Hibbs & P. S. Jensen (Eds.), *Psychosocial treatments for child and adolescent disorders: Empirically based strategies for clinical practice* (2nd ed., pp. 445–476). Washington, DC: American Psychological Association.
- Kendall, P. C. (1994). Treating anxiety disorders in children: Results of a randomized clinical trial. *Journal of Consulting and Clinical Psychology*, 62, 100–110.
- Kendall, P. C., Flannery-Schroeder, E., Panichelli-Mindel, S. M., Southam-Gerow, M., Henin, A., & Warman, M. (1997). Therapy for youths with anxiety disorders: A second randomized clinical trial. *Journal of Consulting and Clinical Psychology*, 65, 366–380.
- Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.
- Lehmkuhl, H. D., Storch, E. A., Bodfish, J. W., & Geffken, G. R. (2008). Brief report: Exposure and response prevention for obsessive compulsive disorder in a 12-year-old with autism. *Journal of Autism and Developmental Disorders*, 38(5), 977–981.
- Lovaas, O. I., Schreibman, L., Koegel, R., & Rehm, R. (1971). Selective responding by autistic children to multiple sensory input. *Journal of Abnormal Psychology*, 77(3), 211–222.
- Norton, P. J., & Price, E. P. (2007). A meta-analytic review of cognitive-behavioral treatment outcome across the anxiety disorders. *The Journal of Nervous and Mental Disease*, 195, 521–531.
- Ost, L.-G. (2008). Efficacy of the third wave of behavioral therapies: A systematic review and meta-analysis. *Behaviour Research and Therapy*, 46(3), 296–321.
- Puleo, C., & Kendall, P. C. (2011). Anxiety disorders in typically developing youth: Autism spectrum symptoms as a predictor of cognitive-behavioral treatment. *Journal of Autism and Developmental Disorders*, 41, 275–286.
- Rachman, S. (1997). The evolution of cognitive behaviour therapy. In D. M. Clark & C. G. Fairburn (Eds.), *Science and practice of cognitive behavior therapy* (pp. 3–26). New York: Oxford University Press.
- Reaven, J. A., Blakeley-Smith, A., Nichols, S., Dasari, M., Flanagan, E., & Hepburn, S. (2009). Cognitive-behavioral group treatment for anxiety symptoms in children with high-functioning autism spectrum disorders: A pilot study. *Focus on Autism and Other Developmental Disabilities*, 24, 27–37.
- Rodriguez, B. I., Craske, M. G., Mineka, S., & Hladek, D. (1999). Context-specificity of relapse: Effects of therapist and environmental context on return of fear. *Behaviour Research and Therapy*, 37, 845–862.
- Rutter, M. (1968). Concepts of autism: A review of research. *Journal of Child Psychology and Psychiatry*, 9(1), 1–25.
- Sofronoff, K., Attwood, T., & Hinton, S. (2005). A randomised controlled trial of CBT intervention for anxiety in children with Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 46, 1152–1160.
- Sofronoff, K., Attwood, T., Hinton, S., & Levin, I. (2007). A randomized controlled trial of a cognitive behavioural intervention for anger management in children diagnosed with Asperger syndrome. *Journal of Autism and Developmental Disorders*, 37, 1203–1214.
- Suveg, C., Comer, J. S., Furr, J. M., & Kendall, P. C. (2006). Adapting manualized CBT for a cognitively delayed child with multiple anxiety disorders. *Clinical Case Studies*, 5, 488–510.
- Sze, K., & Wood, J. (2007). Cognitive behavioral treatment of comorbid anxiety disorders and social difficulties in children with high-functioning autism: A case report. *Journal of Contemporary Psychotherapy*, 37, 133–143.
- Sze, K. M., & Wood, J. J. (2008). Enhancing CBT for the treatment of autism spectrum disorders and concurrent anxiety: A case study. *Behavioural and Cognitive Psychotherapy*, 36, 403–409.
- Watkins, J. T. (1975). Cognitive therapies for depression. *Biological Psychology Bulletin*, 4(3–4), 171–178.
- Watson, J. B., & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology*, 3, 1–14.
- Weisz, J. R., Valeri, S. M., McCarty, C. A., & Moore, P. S. (1999). Interventions for child and adolescent depression: Features, effects, and future directions. In C. A. Essau & F. Petermann (Eds.), *Depressive disorders in children and adolescents* (pp. 383–435). Northvale: Harwood.
- Weisz, J. R., McCarty, C. A., & Valeri, S. M. (2006). Effects of psychotherapy for depression in children and adolescents: A meta-analysis. *Psychological Bulletin*, 132, 132–149.
- White, S. W., Ollendick, T., Scahill, L., Oswald, D., & Albano, A. M. (2009). Preliminary efficacy of a cognitive-behavioral treatment program for anxious youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(12), 1652–1662.
- Wood, J. J., & McLeod, B. M. (2008). *Child anxiety disorders: A treatment manual for practitioners*. New York: Norton.
- Wood, J. J., Piacentini, J. C., Southam-Gerow, M., Chu, B. C., & Sigman, M. (2006). Family cognitive behavioural therapy for child anxiety disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45, 314–324.
- Wood, J. J., Drahota, A., Sze, K., Van Dyke, M., Decker, K., Fujii, C., et al. (2009a). Brief report: Effects of cognitive behavioral therapy on parent-reported autism symptoms in school-age children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 39, 1609–1612.
- Wood, J. J., Drahota, A., Sze, K., Har, K., Chiu, A., & Langer, D. A. (2009b). Cognitive behavioral therapy for anxiety in children with autism spectrum disorders: A randomized, controlled trial. *Journal of Child Psychology and Psychiatry*, 50, 224–234.

Cognitive Behavioral Therapy (CBT) and ASD

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Definition

Cognitive behavioral therapy (CBT) is an evidence-based psychosocial practice for the treatment and primary prevention of anxiety in typically developing children, adolescents, and adults (Chorpita 2007). Recently, different formats of CBT programs have been adapted and used for the treatment of anxiety in children with autism spectrum disorder (ASD). The main purpose of CBT is to help individuals identify and replace negative and unrealistic thoughts with more meaningful positive thoughts, so as to treat both emotional and behavioral problems (Ellis 1962; Beck 1963). CBT programs include a number of cognitive and behavioral problem-solving and coping strategies to enable individuals to identify the contexts, conditions, or precursors that lead to anxiety and then self-manage the symptoms when they appear. Structured manual and modular CBT intervention programs have been implemented in both individual and group formats for varying lengths of time in mental health settings (Perihan et al. 2019). Structured manualized programs include specific sessions for the targeted group. Examples of structured manualized CBT programs are Coping Cat (Kendall and Hedtke 2006) and Skills for Academic and Social Success (Warner et al. 2007). A limitation of the structured manualized programs relevant to ASD is that the manualized approach may not have the flexibility necessary to customize interventions based on the needs of an individual. Modular programs allow for more flexibility and may be tailored to meet and address a wider range of unique needs of children and adolescents (Chorpita 2007).

Current Knowledge

Greater concerns have been expressed in recent years regarding comorbid mental health problems for children with ASD (Ung et al. 2014). Anxiety is a common problem among children and adolescents with ASD (Vasa et al. 2014). Children with ASD tend to be at higher risk for developing significant levels of anxiety than typically developing children (White et al. 2009). While the prevalence of anxiety disorders in typically developing individuals is estimated to be up to 8% (American Academy of Pediatrics 2019), some studies indicated that 40% to 84% children with ASD either meet the diagnosis criteria for various anxiety disorders or report high rates of anxiety (Sukhodolsky et al. 2013). Moreover, the prevalence of anxiety disorders and symptoms in individuals with ASD is also higher than individuals from other disability groups (Vasa et al. 2014). Due to these facts, several studies have been conducted to obtain more information regarding the nature of anxiety symptoms in children with ASD in order to investigate possible overlap in anxiety symptoms and ASD characteristics. Notably, studies have affirmed the similarities between anxiety symptoms and core symptoms of ASD (Wood and Gadow 2010). Recently, Kerns et al. (2014) indicated that some individuals with ASD showed atypical anxiety symptoms that were not consistent with any DSM-5 criteria. As of now, anxiety symptoms of children with ASD are commonly measured by standardized assessment measures that have been developed for assessing anxiety symptoms in typically developing children (Sterling et al. 2015). Examined together, these factors may cause children with ASD to remain unidentified or insufficiently supported in both social and school environments. Nonetheless, while the actual rate of anxiety in children with ASD remains unclear, already identified individuals with a high level of anxiety should be able to receive the best treatment options available.

Children with ASD typically have difficulties in social interactions, communication, and academic performance (American Psychiatric Association [APA] 2013; Leyfer et al. 2006). High levels of anxiety exacerbate these characteristics

and further negatively impact their abilities across the school, home, and community environments (Reaven et al. 2008; Sukhodoldky et al. 2008). Without intervention, high levels of anxiety experienced by many children with ASD may lead to additional mental health problems such as depression, substance abuse, and suicide attempts (Fujii et al. 2012). In recent years, there is also a growing body of research that has illustrated that most children with ASD face difficulties in using their cognitive potential to communicate or build social relationships, which leads to developing high levels of anxiety (Estes et al. 2011). Given the high prevalence of anxiety in children with ASD, there has been a recent push for the implementation of CBT as an evidence-based treatment program to treat anxiety in children with ASD (Clarke et al. 2016).

CBTs are considered primary treatment options for anxiety in typically developing children across different settings. Seligman and Ollendick (2011) indicated that over 269 meta-analytic studies have demonstrated the efficacy of the CBT for the treatment for wide range of problems including the efficacy of CBTs for children and adolescents with anxiety disorders. Overall, these studies indicated that two-thirds of children with anxiety disorders showed no anxiety symptoms after CBT (Seligman and Ollendick 2011). Although much of the current literature on CBT involves typically developing children, increasing attention is currently being given by researchers for using CBT to treat anxiety symptoms in children with ASD. Findings of randomized controlled trial studies, as well as meta-analyses, support that children with ASD benefit from CBT treatments for anxiety disorders (Ung et al. 2014). Moreover, the current evidence provides support to use CBT for children with ASD. However, implementations of CBT programs with adaptations for ASD require additional research on the necessary adaption components, and future studies need to be conducted that have better implementation fidelity of the CBT programs for children with ASD (Perihan et al. 2019).

A growing number of studies have begun to examine the efficacy of CBTs for anxiety in children with ASD, and an increased body of research

has demonstrated that CBT is an effective treatment with adaptations for these youth (Ung et al. 2014). A recent meta-analysis (Perihan et al. 2019) reported that CBT appears as an effective treatment option for children with ASD. Twenty-three published studies of CBT for anxiety in children with ASD were included. Studies varied in sample size from 6 to 69 participants and used varied types of CBTs lasting between 4 and 32 weekly sessions. Outcomes were assessed by examining different types of parent, child, and teacher questionnaires which were designed to measure anxiety in typically developing children. Results yielded a moderate effect size ($g = -0.66$) for the reduction of anxiety symptoms; children who received CBT treatment had reduced levels of anxiety.

To understand potential factors that affect treatment responses of CBT for anxiety, parental involvement and treatment lengths were examined. Several studies have demonstrated that parents of children with ASD are at increased risk of having a higher level of parental stress than parents of typically developing children (Conner et al. 2013). Researchers have paid particular attention to exploring the relationship between parental anxiety and anxiety symptoms in individuals with ASD, as well as possible effects of parental anxiety in treatment response (Reaven et al. 2015). Analysis of parental involvement indicated that individuals with ASD received the most benefit from CBTs when parents were involved in the interventions. This finding provides additional support for the research that expressed that parents may play a critical role in changes in child anxiety (Sofronoff et al. 2005). Moreover, the involvement of parents in CBT programs provides an opportunity to assist children in practicing coping skills when they are faced with challenges outside of school (Weiss et al. 2014). Surprisingly, a few studies showed that parents also derived benefits from their involvement in CBT treatments. They were able to apply the key CBT concepts to themselves to manage their feelings, as well as to reduce both their anxiety and parental stress (Ooi et al. 2008; Reaven et al. 2015). This finding points to the possible impact of parental involvement on

treatment responses of CBT for anxiety in this population.

Recently, studies have consistently noted that children with ASD may need extended time to understand and apply newly learned cognitive strategies for coping with their feelings (Clarke et al. 2016). Thus, the recent meta-analysis (Perihan et al. 2019) aimed to investigate the possible impact of the treatment length on treatment response. Findings demonstrated that treatment effects associated with short-term interventions were significantly weaker than those obtained in standard-term and long-term interventions. Some studies have also aimed to increase the efficacy of interventions in a short period of time (e.g., 1 week) by extending the contact time with clients. However, the results of the studies suggest that extended time rather than more contact time is required for the clients to learn and apply the cognitive strategies implemented. It is thus likely that children also need extended time rather than more contact time to learn and apply cognitive strategies for coping with their anxieties.

Future Directions

A major focus in the development of CBT for children with ASD is the continuing adaptation for school-based settings. In 2014, about 1 in 59 children were identified with ASD by age 8; that statistic is constantly increasing. Furthermore, anxiety-related symptoms are significantly common problems in children with ASD (White et al. 2009). CBT is one of the most effective evidence-based treatments of anxiety in typically developing children, and a growing body of research, including the previously discussed meta-analysis, have clearly documented the impressive results of CBTs in treating anxiety among children with ASD. However, while CBT, especially CBT with modifications or adaptations for ASD (Perihan et al. 2019), appears helpful, some recommendations should be considered for future studies.

The first and most important consideration for future studies is transferring CBTs from clinic to school settings. The focus on inclusion in schools

means a greater number of students with ASD are going to be placed and be educated in general education classrooms with their typically developing peers (Feraoli and Harris 2011). A substantial body of research has demonstrated that children with ASD already face difficulties in social interactions and communication (APA 2013). The move toward inclusion of students with ASD is likely to exacerbate those social interaction and communication programs and increase the level of anxiety experienced by this population. Despite school being an appropriate environment for intervention programs for children with ASD, most of the previous studies were of CBTs delivered in clinic settings by clinicians (Perihan et al. 2019). In a review of the literature, Perihan et al. (2019) found medium to large effect sizes that ranged between -2.40 and -0.40 for school-based CBT treatments in children with ASD (e.g., Clarke et al. 2016; Fujii et al. 2012). These findings provide initial support for the delivery of CBT programs in school settings by a range of professionals – such as teachers and counselors with appropriate training and supervision (Clarke et al. 2016). Introducing CBT programs into school settings that are administered by school professionals will also increase the accessibility of treatments of anxiety for children with ASD (McConachie et al. 2013). Otherwise, the limited number of clinicians will contribute to fewer students being treated in school systems.

Today, the treatment of anxiety symptoms with CBT in children with ASD is still progressing, and researchers are continuing to identify the modifications and adaptations that should be made to existing CBT programs to meet the needs of children with ASD. For future studies, evidence-based guidelines may be required to examine whether these modified CBTs have been implemented accurately with necessary quality components to ensure they are efficient and generalizable (Seligman and Ollendick (2011). As such, it is necessary to include implementation fidelity in studies to investigate the quality of each CBT program or protocol used.

Last, there is continuing uncertainty about the measures used to evaluate anxiety in children with ASD. Most studies have used outcome measures

that have been designed to assess anxiety symptoms in typically developing children. It is becoming clear that children with ASD may possess atypical anxiety symptoms which may not be captured by existing measures (Kerns et al. 2014). There is also emerging evidence for similarities in anxiety symptoms and ASD characteristics (Bellini 2006). Social interactions and communication problems are primary characteristics of children with ASD, which may increase levels of anxiety. Likewise, the difficulty of school settings may increase or intensify the levels of anxiety in children with ASD (White et al. 2009). In order to make a distinction between anxiety symptoms and autism characteristics, future studies should include outcome measures specifically for assessing the autism vs. anxiety symptoms of children with ASD.

See Also

- Autism Spectrum Addendum to the Anxiety Disorders Interview Schedule-Parent Interview
- Cognitive Behavioral Therapy (CBT)
- High-Functioning Autism (HFA)

References and Reading

- American Academy of Pediatrics: *Anxiety Fact sheet*. (n.d.). Retrieved July 25, 2019 from <https://www.aap.org/en-us/advocacy-and-policy/aap-health-initiatives/resilience/Pages/Anxiety-Fact-Sheet.aspx>
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Beck, A. T. (1963). Thinking and depression: Theory and therapy. *Archives of general Psychiatry*, 10, 561–571.
- Bellini, S. (2006). The development of social anxiety in adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 21(3), 138–145.
- Chorpita, B. F. (2007). *Modular cognitive-behavioral therapy for childhood anxiety disorders*. New York: Guilford Press.
- Clarke, C., Hill, V., & Charman, T. (2016). School based cognitive behavioural therapy targeting anxiety in children with autistic spectrum disorder: A quasi-experimental randomised controlled trial incorporating a mixed methods approach. *Journal of Autism and Developmental Disorders*, 47(12), 3883–3895.
- Conner, C. M., Maddox, B. B., & White, S. W. (2013). Parents' state and trait anxiety: Relationships with anxiety severity and treatment response in adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43, 1811–1818.
- Ellis, A. (1962). *Reason and emotions in psychotherapy*. New York: Lyle Stuart.
- Estes, A., Rivera, V., Bryan, M., Cali, P., & Dawson, G. (2011). Discrepancies between academic achievement and intellectual ability in higher-functioning school-aged children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41(8), 1044–1052.
- Ferraioli, S. J., & Harris, S. L. (2011). Teaching Joint Attention to Children with Autism Through A Sibling-Mediated Behavioral Intervention. *Behavioral Interventions*, 26(4), 261–281. <https://doi.org/10.1002/bin.336>
- Fujii, C., Renno, P., Mcleod, B. D., Lin, C. E., Decker, K., Zielinski, K., & Wood, J. J. (2012). Intensive cognitive behavioral therapy for anxiety disorders in school-aged children with autism: A preliminary comparison with treatment-as-usual. *School Mental Health*, 5(1), 25–37. <https://doi.org/10.1007/s12310-012-9090-0>
- Kendall, P. C., & Hedtke, K. (2006). *Cognitive-behavioral therapy for anxious children: Therapist manual* (3rd ed.). Ardmore: Workbook Publishing.
- Kerns, C. M., Kendall, P. C., Berry, L., Souders, M. C., Franklin, M. E., Schultz, R. T., ... Herrington, J. (2014). Traditional and atypical presentations of anxiety in youth with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(11), 2851–2861.
- Leyfer, O. T., Folstein, S. E., Bacalman, S., David, N. O., Dinh, E., Morgan, J., Tager-Flusberg, H., & Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36, 849–861.
- Mcconachie, H., McLaughlin, E., Grahame, V., Taylor, H., Honey, E., Tavernor, L., ... Couteur, A. L. (2013). Group therapy for anxiety in children with autism spectrum disorder. *Autism*, 18(6), 723–732.
- Ooi, Y. P., Lam, C. M., Sung, M., Tan, W. T. S., Goh, T. J., Fung, D. S. S., ... Chua, A. (2008) Effects of cognitive-behavioural therapy on anxiety for children with high-functioning autistic spectrum disorders. *Singapore Medical Journal*, 49(3), 215.
- Perihan, C., Burke, M., Bowman-Perrott, L., Bicer, A., Gallup, J., Thompson, J., & Sallase, M. (2019). Effects of cognitive Behavioral therapy for reducing anxiety in children with high functioning ASD: A systematic review and meta-analysis. *Journal of Autism and Developmental Disorders*, 1–15.
- Reaven, J. A., Blakeley-Smith, A., Nichols, S., Dasari, M., Flanagan, E., & Hepburn, S. (2008). Cognitive-behavioral group treatment for anxiety symptoms in children with high-functioning autism spectrum

- disorders. *Focus on Autism and Other Developmental Disabilities*, 24(1), 27–37.
- Reaven, J., Washington, L., Moody, E. J., Stern, J. A., Hepburn, S. L., & Blakeley-Smith, A. (2015). Examining the relationship between parental anxiety and treatment response in children and adolescents with autism spectrum disorder and anxiety. *Journal of Autism and Developmental Disorders*, 45(8), 2464–2473.
- Seligman, L. D., & Ollendick, T. H. (2011). Cognitive-behavioral therapy for anxiety disorders in youth. *Child and Adolescent Psychiatric Clinics of North America*, 20(2), 217–238. <https://doi.org/10.1016/j.chc.2011.01.003>
- Sofronoff, K., Attwood, T., & Hinton, S. (2005). A randomised controlled trial of a CBT intervention for anxiety in children with Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 46(11), 1152–1160.
- Sterling, L., Renno, P., Storch, E. A., Ehrenreich-May, J., Lewin, A. B., Arnold, E., ... Wood, J. (2015). Validity of the Revised Children's Anxiety and Depression Scale for youth with autism spectrum disorders. *Autism*, 19(1), 113–117.
- Sukhodolsky, D. G., Bloch, M. H., Panza, K. E., & Reichow, B. (2013). Cognitive-behavioral therapy for anxiety in children with high-functioning autism: A meta-analysis. *Pediatrics*, 132(5), 1341–1350.
- Ung, D., Selles, R., Small, B. J., & Storch, E. A. (2014). A systematic review and meta-analysis of cognitive-behavioral therapy for anxiety in youth with high-functioning autism spectrum disorders. *Child Psychiatry & Human Development*, 46(4), 533–547.
- Vasa, R. A., Carroll, L. M., Nozzolillo, A. A., Mahajan, R., Mazurek, M. O., Bennett, A. E., ... Bernal, M. P. (2014). A systematic review of treatments for anxiety in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(12), 3215–3229.
- Warner, C. M., Fisher, P. H., Shrout, P. E., Rathor, S., & Klein, R. G. (2007). Treating adolescents with social anxiety disorder in school: an attention control trial. *Journal of Child Psychology and Psychiatry*, 48(7), 676–686. <https://doi.org/10.1111/j.1469-7610.2007.01737.x>
- Weiss, J. A., Vecili, M. A., & Bohr, Y. (2014). Parenting stress as a correlate of cognitive behavior therapy responsiveness in children with autism spectrum disorders and anxiety. *Focus on Autism and Other Developmental Disabilities*, 30(3), 154–164.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29, 216–229.
- Wood, J. J., & Gadow, K. D. (2010). Exploring the nature and function of anxiety in youth with autism spectrum disorders. *Clinical Psychology: Science and Practice*, 17(4), 281–292.

Cognitive Bias

► Primacy Effects

Cognitive Control Circuitry

► Neural Mechanisms of Emotional Dysregulation

Cognitive Correlates of Reciprocity in Children with Autism Spectrum Disorder

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Definition

The important cognitive factors associated with reciprocity in children with autism spectrum disorder (ASD) include the social-cognitive factor “theory of mind (ToM)” and the cognitive factors “cool” and “hot” executive function (EF).

Historical Background

Reciprocity in Children with ASD

Reciprocal behavior is pivotal to the process of collaborative behavior between individuals who

perform activities to reach shared goals (Cole and Teboul 2004). People who perform reciprocity successfully are aware of others' emotional and interpersonal cues, interpret those cues appropriately, respond suitably to what they interpret, and are motivated to engage in social interactions with others (Constantino et al. 2003). Reciprocity consists of mutual and symmetrical exchanges between individuals as they talk, work, or play together (Gernsbacher 2006). Such collaboration is a critical component of human social behavior, one in which we engage often in social contexts (Hammerstein 2003; Stevens and Hauser 2004). If people do not equally participate and exchange with fine turn-taking, collaboration will fail, leading to unsatisfactory results (Cole and Teboul 2004). A child's ability to perform reciprocal behaviors develops along with age, depending on both the child's cognitive ability and emotional functioning (Perez and Gauvain 2005). Therefore, reciprocity is an important skill for social interaction.

A study by Sakaiya et al. (2013) investigated the neural process of human reciprocity in social interactions with prisoner's dilemma (PD) games, which are used to examine cooperative behavior in adults. An important component of PD games is the Nash equilibrium, which represents mutual defection. Such equilibrium leads to lower payoffs for each player than does mutual cooperation and thus poses a dilemma. To investigate the neural correlates of reciprocal interaction, the researchers manipulated the partner identity (human, computer) and strategy (random, tit-for-tat) in the game. The results showed that, in the human-as-partner condition, tit-for-tat and random strategies were positively and negatively correlated with activation of the left amygdala. This activation represented positive feelings toward reciprocal partners and negative feelings toward nonreciprocal partners. That study also examined the neural process of insight into the reciprocity of partners. Reciprocity influenced differential brain activation in the reward-related circuitry (i.e., the right middle dorsolateral prefrontal cortex and dorsal caudate) and theory of mind (ToM) regions [i.e., the ventromedial prefrontal cortex (VMPFC) and precuneus]. In

participants without insight into their human partners, a tit-for-tat strategy led to asymmetrical activation in the precuneus and VMPFC and activation of the precuneus accompanied by the deactivation of the VMPFC. In summary, the amygdala processes the intensity of emotion in human reciprocity, and the reward-related and ToM regions control the insight into the reciprocity of others.

From infancy, children display motivation for engaging in joint actions and sharing psychological states with others (Carpenter 2009; Feldman and Greenbaum 1997; Trevarthen and Aitken 2001). At as early as 2 months old, mother-infant interactions involve looking at and listening to each other, mutually regulating one another's interests and feelings, exchanging multimodal signals, and imitating vocal, facial, and gestural expressions. During the course of early childhood development, a child's ability to collaborate with others develops from basic to more advanced levels. At around 2 years of age, children begin playing in proximity to each other, doing the same types of activities. In such so-called parallel activity, the children's attention is focused on the same object, but they operate it separately, without engaging in mutual exchanges. In essence, they are playing alone, rather than with other children (van Ommeren et al. 2012). Gradually, the children begin interacting with other children, exhibiting reciprocal behaviors such as equal turn-taking and object sharing (Eckerman et al. 1989; Warneken and Tomasello 2006). At 3 years of age, children begin sharing themes with their playmates, such as building a sandcastle together (Howes 1988). During middle childhood, when children begin to understand others' goals and intentions, they exhibit collaborative reciprocal behaviors. At this age, children have gained the requisite skills and motivation to share psychological states with others (Tomasello et al. 2005). This sharing behavior, which consists of basic to complex reciprocity, enables children to achieve common goals, such as building a small playhouse or drawing a picture (van Ommeren et al. 2012).

Previous studies have investigated the reciprocity ability of children with ASD. In the

study of Sally and Hill (2006), children with ASD and normal IQ were found to reciprocate adequately in a structured test; e.g., on a prisoner's dilemma task. However, adequate performance observed in a structured assessment does not necessarily carry over to real-life interactions. In unstructured real-life interactions, children with ASD are unable to show appropriate reciprocal behaviors with peers and adults. In the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, it is noted that children with ASD have qualitative impairment in social interaction, including failure to develop peer relationships; a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people; and a lack of social or emotional reciprocity (APA 2000). Ozonoff and Miller (1995) found that children with ASD have lower scores in the Socialization domain (which is correlated with reciprocity in daily life) of the Vineland Adaptive Behavior Scale (VABS). The studies of Kasari et al. (2011); Rotheram-Fuller et al. (2010); and Chamberlain et al. (2007) investigated the social networks and friendship reciprocity of children with ASD in elementary school. It was found that children with ASD have lower social network centrality and less friendship reciprocity than their typically developing peers do.

The social reciprocity deficit described above can lead to consequences for children with ASD. School-aged children and adolescents with ASD have increased risk of peer rejection and social isolation (Chamberlain et al. 2007). These children also express that they have less social support and more loneliness than typically developing children (Bauminger and Kasari 2000). The deficit may also lead to underachievement in academics and their later occupations (Howlin and Goode 1998). At the later stages of development, social reciprocity deficit may increase the risk of mood and anxiety problems (Myles et al. 2001; Tantam 2003).

However, the above evidence for this impairment is primarily based on observations of children in daily life, rather than direct assessments with psychological instruments (van Ommeren et al. 2012). Therefore, reciprocity performance

in the laboratory context is not correlated with social interactions in daily life.

Theory of Mind in Children with ASD

Theory of mind (ToM) has been used interchangeably with terms such as "perspective-taking," "social cognition," "metacognition," and "folk psychology." (Astington and Baird 2005; Flavell et al. 2002). ToM is a cognitive ability (Adolphs 2001) whereby a person understands and infers the mental states (e.g., belief, desires, and intentions) of others and uses that information to explain and predict behaviors and actions (Premack and Woodruff 1978; Wellman 1993). ToM assessment traditionally has relied on the false-belief task (Wimmer and Perner 1983), which identifies the ability to realize that people may have different or false understandings and representations of a given situation (Dennett 1978).

However, ToM is understood to be a complex and multifaceted construct. Some researchers have recommended that various aspects of ToM be assessed across levels of complexity (Tager-Flusberg 2001; Wellman et al. 2001) and suggested that it is constructed of related but distinct types of social cognitive understanding (Hughes et al. 2000), such as emotion recognition (Prior et al. 1990), desire-based emotion (e.g., understanding that people are happy when they get what they want), belief-based emotion (e.g., understanding that people will be happy if they think they will get what they want) (Baron-Cohen 1991), understanding that seeing leads to knowing (Perner et al. 1989), and understanding that people see the same object in different ways (e.g., by positioning) (Pillow and Flavell 1986). Complex, or higher-order, ToM is typically measured with a second-order false-belief task (e.g., what Wendy thinks Patty thinks) to assess inferences of both belief and emotion (Hughes et al. 2000).

ToM is a prerequisite skill for developing reciprocal communication and social interactions (Wellman 1990). It helps people understand and distinguish emotional states and complex social information. This ability also helps children to develop more complicated social cognitive skills,

such as perspective-taking, collaboration, and metacognition (Sally and Hill 2006).

Infants display some behaviors that are important beginnings for theory-of-mind development. By 2–3 months, they are able to participate in simple social interactions with others and can coordinate their gestures, vocalizations, and facial expressions with others (Mar et al. 2010). By the age of 2, children clearly show awareness of the difference between thoughts in the mind and things in the world (e.g., pretending a block is a car) (Kavanaugh 2006). They also understand belief-based emotion (e.g., people will feel happy if they get what they want) (Wellman and Banerjee 1991). A crucial development occurs at around 4 years of age, when children realize that thoughts in their minds may not be true. At age 4 or 5, children develop the ability to understand a first-order false-belief task (Wimmer and Perner 1983), and they realize that people talk and act on the basis of the way they think the world is, even when their thoughts do not reflect the actual situation. After 5 years old, children develop advanced mental understanding, such as second-order false-belief, lies, and metacognition (Astington and Gopnik 1991).

Many researchers have concluded that ToM deficits underlie the social, behavioral, and communicative impairments in individuals with ASD (Baron-Cohen et al. 1985). The most common ToM assessment is the classic false-belief task, originally developed by Wimmer and Perner (1983). In this task, children are told a story in which an object is moved from an old location to a new location without the knowledge of the main protagonist. The children are asked, “Where would the main protagonist look for the object?” The old location is the correct answer. As compared with age- and language-matched peers, individuals with ASD generally perform poorly on the false-belief task.

In a study by Baron-Cohen et al. (1985), the Sally-Anne task (first-order false-belief task) was used to examine ToM in children with ASD, and fewer individuals with ASD (20%) passed the test than did typically developing children (85%). In Baron-Cohen (1989), a second-order false-belief task was used to examine ToM in

children with ASD. In that study, none of the children with ASD passed, while 90% of the typically developing children did. In addition, children with ASD are reported to have difficulties in using logic, inferring beliefs, and recognizing emotions (Prior et al. 1990).

In Baron-Cohen (1991), the researchers examined whether individuals with ASD understand some causes (situations, desires, beliefs) of emotions. They found that children with ASD showed deficits in the comprehension of emotions caused by belief as compared with mental age-matched peers. In summary, children with ASD show deficits in both higher-level and lower-level ToM tasks.

Executive Function in Children with ASD

According to Lezak (1995), “Executive functions refer to a collection of interrelated cognitive and behavioral skills that are responsible for purposeful, goal-directed activity, and include the highest level of human functioning.” This collection encompasses the ability to suppress responses, to keep and manipulate information online, to change strategies, and to plan ahead. Executive function allows people to access information, think about solutions, and implement those solutions.

Nowadays, EF is divided into cool and hot aspects. Cool executive function (Cool EF) is defined as goal-directed and future-oriented skills manifested within abstract, decontextualized, nonemotional, and analytical conditions. It is a more purely cognitive aspect of EF usually associated with the dorsolateral prefrontal cortex (DLPFC) (Metcalf and Mischel 1999). Hot executive function (Hot EF) is defined as goal-directed and future-oriented cognitive processes manifested within the contexts of engendering emotion and motivation (Zelazo and Carlson 2012; Zelazo and Müller 2002; Zelazo et al. 2005). It is the affective aspect of executive function and is usually associated with the orbital frontal cortex (OFC) and other medial regions (Happaney et al. 2004).

Despite the numerous investigations of cool executive function in children with ASD, the results have been inconsistent (Geurts et al.

2009; Hill 2004). Some studies have indicated that children with ASD have difficulties in tasks requiring working memory, inhibition, and set shifting ability as compared to their typically developing peers (Hughes et al. 1994; Szatmari et al. 1989), while others have cast doubt on those findings.

Recent studies have found that EF performance is affected by the age of the participants. Older children and adults score worse than their mental age-matched peers, while younger children with ASD exhibit no impairments as compared to their peers (Dawson et al. 1998; McEvoy et al. 1993; Yerys et al. 2007). It is suggested that executive function deficit may not be the primary deficit in children with ASD; rather, it may be a secondary one that manifests as the children develop (Yerys et al. 2007).

Some researchers have suggested that autism and related disorders may involve deficits in global processing and social and emotional functioning (i.e., hot EF) (Happaney et al. 2004), which are controlled by the right and left VMPFC hemispheres. Bechara (2004) suggests that the differential involvement in the right and left hemispheres in avoidance (negative affect) and approach (positive affect) lead to right–left hemispheric asymmetries in the OFC (VM-PFC) (Davidson and Irwin 1999; Davidson et al. 2000). For example, the Iowa Gambling Task requires adaptive decision-making, for seemingly positive responses need to be avoided (a function for which the right VM-PFC may be particularly well suited). The right hemisphere has also been implicated in the mapping of bodily states and the comprehension of somatic information (Cohen et al. 1976), which may help to explain the relative importance of the right OFC in hot EF (Bechara 2004).

Based on a previous literature review on reciprocity, it appears that EF and ToM are important factors in reciprocity in children with ASD. When directly examining the relationships between ToM and reciprocity in children with ASD with the behavioral approach, Sally and Hill (2006) found that children with ASD have impaired ToM ability in both first- and second-order false-belief tasks as compared to their typically developing peers, but

the results for reciprocity in games did not show significant group differences. As explanation, the authors suggested that ToM skills may not be needed in structured computer games. Examinations of the relationships between EF and reciprocity in children with ASD with the behavioral approach have identified the neural correlates of reciprocity (Sakaiya et al. 2013). The DLPFC and VMPFC are active during reciprocal behaviors. Cool EF, controlled by the DLPFC, may affect reciprocity performance. However, no behavior studies have directly investigated the relationship between cool EF and reciprocity in children with ASD. Hot EF, which is controlled by the OFC, one part of the VMPFC, may affect reciprocity performance.

Current Knowledge

ToM and cool EF have been used to explain social deficits. In addition, the brain regions controlling ToM and EF appear to be the neural correlates of reciprocity in children with ASD. However, these two theories, ToM and cool EF, cannot fully explain reciprocity in children with ASD, possibly due to a lack of consideration of the importance of motivation and emotion. To date, only the behavioral study of Lai and her colleagues has examined ToM and EF simultaneously in the reciprocity in children with ASD. In Lai et al.'s study, a total of 59 children with diagnoses of autistic disorder or Asperger's syndrome aged from 4 to 12 years were recruited. In addition to the children's verbal ability and autistic severity, their reciprocity (van Ommeren et al. 2012), ToM, cool EF, and hot EF were examined with the Interactive Drawing Task (IDT), Theory of Mind Task Battery (Hutchins et al. 2012), Dimensional Card Change Sort Task (DCCS) (Diamond and Kirkham 2005; Dichter et al. 2010), and Children's Gambling Task (Kerr and Zelazo 2004).

The IDT was specifically used in Lai et al.'s study to measure children's reciprocity performance (van Ommeren et al. 2012). The IDT is designed to examine reciprocity in a joint, unstructured situation to re-create the unstructured, unpredictable aspects of real-life social

interactions. During the IDT, children are invited to draw a house with the experimenter. Requiring no real drawing skill, the test elicits spontaneous interaction with the experimenter to achieve a mutual goal. The IDT lasts approximately 10 min and requires only a table, drawing paper of size A3, four colored markers (red, blue, black, green), and a video camera. At the beginning of the test, the experimenter says, “We are going to draw together. You may choose a marker.” No other instructions are provided. The goal of the IDT is to elicit equal participation of the participants, with the experimenter setting a good example of how to collaborate with others. To do so, the experimenter performs reciprocal behaviors with increasing dynamics, such as beginning to draw his or her own objects and contributing to the child’s objects. The experimenter also elicits reciprocal behaviors by drawing incomplete objects and by demonstrating reciprocal responses, such as adding elements to an incomplete drawing by the child. Then the experimenter encourages turn-taking by turning the paper to the child, an action that is repeated throughout the test. This assessment measures three types of reciprocal behaviors. The first is the proportion of reciprocal drawing (i.e., collaborating by adding meaningful elements) of the total turns, or total reciprocity. Reciprocal drawing (total reciprocity) is divided into the proportion of reciprocity in the other’s initiative and reciprocity in the child’s own initiative. The former is the amount of reciprocity in the objects drawn by the experimenter, and the latter is the amount of reciprocity in those drawn by the child.

By measuring reciprocity, ToM, hot EF, and cool EF, Lai et al. showed that cool EF and hot EF are both significantly correlated with reciprocity and that in children with ASD, only verbal ability is significantly correlated with reciprocity in the child’s own initiative.

In Lai et al.’s study, cool EF and hot EF were significantly correlated with reciprocity in children with ASD. They also found that cool EF was the significant correlate for reciprocity in the child’s own initiative. However, none of the variables in their study were significantly associated with reciprocity in the other’s initiative.

Therefore, children with better cool EF and hot EF may have better reciprocity capacity and reciprocity in the child’s own initiative. However, the correlate of reciprocity in the other’s initiative was not identified in their study, so the correlates of children’s reciprocity in the other’s initiative remain undetermined.

In addition, Lai et al.’s study found that cool EF and hot EF may be the significant correlates of reciprocity. Children with better cool EF and hot EF ability may perform more reciprocal behaviors. Another important result was the significant correlation between cool EF and reciprocity. The cool EF task, the DCCS, focuses on children’s ability to shift attention. Children who can correctly shift their attention based on the sorting rule may perform more reciprocal behaviors because they can more easily shift their attention to the collaborative theme. This conclusion is consistent with neural process research reporting that the neural correlate of reciprocal behaviors is the DLPFC, the neural region for cool EF (Sakaiya et al. 2013). Hot EF, however, is a goal-directed and future-oriented cognitive process, manifested within the contexts of engendering emotion and motivation (Zelazo and Carlson 2012; Zelazo and Müller 2002; Zelazo et al. 2005). It appears to be significantly associated with the capacity for reciprocal behavior. It is possible that the IDT requires that motivation be maintained; if so, the importance of the drawing themes may need to be reappraised. Children with better hot EF ability, having the ability to keep their attention and motivation focused on the reciprocal themes, can contribute meaningful elements toward the collaborative goals.

The nonsignificant relationship between ToM and reciprocity reported by Lai et al. may have two possible explanations. The first is the obvious nature of the drawing theme of the experimenter. The children may not have needed to employ ToM to speculate on the experimenter’s behaviors. The second is the lack of structure and unpredictability of the IDT. Faced with this unstructured situation, the children with ASD might have found it difficult to generalize their ToM ability to it and thus been unable to respond properly. Despite being able to perform well on the ToM task, the

unstructured context may have limited their ability to perform well on the reciprocity task. This explanation is consistent with findings that, in an unstructured context, children with ASD have difficulties applying their ToM. High-functioning adolescents and adults with ASD can pass ToM tasks at various levels of complexity (Dahlgren and Trillingsgaard 1996). In structured situations, they can respond properly (Begeer et al. 2010), but in unstructured and unpredictable situations, they cannot. However, Tomasello et al. (2005) found that children with ASD have fewer reciprocal behaviors, possibly due to difficulties in understanding others' intentions. This finding is inconsistent with the nonsignificant correlation between ToM and reciprocity of Lai et al.'s study. In summary, the relationship between ToM and reciprocity needs to be further clarified, and the task characteristics, such as the collaborative themes and unstructured or structured tasks used in measuring children's reciprocal behaviors and social interaction, also need to be considered.

As for reciprocity in the child's own initiative and reciprocity in the other's initiative, the present literature suggests that children with ASD present more reciprocity in their own initiative than in another's initiative. It is possible that their restricted behaviors interfere with their ability to shift their attention to another's drawing theme. For instance, in Lai et al.'s study, many of the participants focused on drawing dinosaurs, cars, and busses; they had a hard time following the experimenter's initiative. Another study, van Ommersen et al. (2012), also suggested that children with ASD preferred to lead the drawing themes because they wanted to avoid the confrontation of unexpected situations. To do so, they would perform more reciprocity on their own initiatives.

In addition, children's reciprocity performance on the IDT can be divided into three types: active, passive, and proper reciprocity. Children exhibiting active reciprocity present much reciprocity in their own initiatives and draw typical objects such as cars and busses. Like younger children diagnosed with Asperger's syndrome, these children show only limited responses to the experimenter's initiatives. Children exhibiting passive reciprocity respond well to the

experimenter's initiative, following the experimenter's drawing theme. However, these children rarely initiate their own drawing themes, depending on the experimenter to lead them. Children exhibiting proper reciprocity balance their responses to their own initiatives and to the experimenter's initiatives. They take turns with the experimenter in assuming the leading role, so the dynamic between the experimenter and the child is relatively high.

These types of reciprocal behaviors on the IDT are fairly consistent with the four types of social interaction styles (Castelloe and Dawson 1993; O'Brien 1996; Scheeren et al. 2012): typical, active-but-odd, passive, and aloof. The active type of IDT performance, wherein the children develop their own drawing themes but fail to respond properly to the experimenter's, is similar to the behaviors of active-but-odd children. These children seek out social interaction but do so in an unusual way, such as holding one-sided conversations about their own interests. The passive type of IDT performance, in which the children seldom initiate their own drawings but add proper, meaningful elements to the experimenter's, is similar to the behaviors of passive children. These children do not initiate social interaction, but they respond appropriately to another's initiative. The proper type in IDT performance, wherein the children both initiate their own themes and participate in the experimenter's, resembles the behaviors of typical children, who act properly in social interactions. The absence of any reciprocity on the IDT thus may resemble the behaviors of aloof children, who neither seek out social interaction nor respond to another's initiative. This can be expected, since such children usually have less verbal ability and are low-functioning. Considering the similarities between the types of IDT performance and social interaction styles, an important correlate of reciprocity in the child's own or another's initiative may be the children's social interaction styles.

In conclusion, children with ASD who have better cool EF and hot EF may have better reciprocity capacity, which can be used as the basis for targeting interventions and as the foundation for further study. For clinicians, it is necessary to

focus evaluations and interventions on cool EF and hot EF. With such measurement, clinicians could understand the children's cool EF, hot EF, and reciprocity and thereby detect progress after interventions. Based on the evaluations, interventions targeting cool EF and hot EF could be provided to improve the children's reciprocal behaviors. Targeted interventions could be accordingly developed and provided to children with ASD to improve their reciprocity.

Future Directions

At this stage, the current existing findings cannot be generalized to lower-functioning and nonverbal children with ASD, and their social interaction styles have not been assessed. Future studies should consider recruiting lower-functioning children and those with less verbal ability, use corresponding measurements adjusted for these children, and add a measurement for evaluating social interaction styles. In addition, as regards the relationship between ToM and reciprocity, further study is warranted to better elucidate the relationship between ToM and reciprocity with consideration of the task characteristics, such as the collaborative themes and unstructured or structured tasks used in measuring children's reciprocal behaviors.

See Also

- Components of Advance Theory of Mind in Autism Spectrum Disorder
- Executive Functions, Social Impairment, Friendship Quality, and Adjustment in HFA with ASD
- Theory of Mind

References and Reading

- Adolphs, R. (2001). The neurobiology of social cognition. *Current Opinion in Neurobiology*, 11(2), 231–239.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.
- Astington, J. W., & Baird, J. A. (2005). Introduction: Why language matters. In J. W. Astington & J. A. Baird (Eds.), *Why language matters for theory of mind* (pp. 3–25). Oxford, UK: Oxford University Press.
- Astington, J. W., & Gopnik, A. (1991). Theoretical explanations of children's understanding of the mind. *British Journal of Developmental Psychology*, 9(1), 7–31.
- Baron-Cohen, S. (1989). The autistic child's theory of mind: A case of specific developmental delay. *Journal of Child Psychology and Psychiatry*, 30(2), 285–297.
- Baron-Cohen, S. (1991). Do people with autism understand what causes emotion? *Child Development*, 62(2), 385–395.
- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a "theory of mind"? *Cognition*, 21(1), 37–46.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development*, 71, 447–456.
- Bechara, A. (2004). The role of emotion in decision-making: Evidence from neurological patients with orbitofrontal damage. *Brain and Cognition*, 55(1), 30–40. <https://doi.org/10.1016/j.bandc.2003.04.001>.
- Begeer, S., Malle, B. F., Nieuwland, M., & Keysar, B. (2010). Using theory of mind to represent and take part in social interactions: Comparing individuals with high-functioning autism and typically developing controls. *European Journal of Child Development*, 7, 104–122.
- Carpenter, M. (2009). Just how joint is joint action in infancy? *Topics in Cognitive Science*, 1, 380–392.
- Castelloe, P., & Dawson, G. (1993). Subclassification of children with autism and pervasive developmental disorder: A questionnaire based on Wing's subgrouping scheme. *Journal of Autism and Developmental Disorders*, 23(2), 229–241.
- Chamberlain, B., Kasari, C., & Rotheram-Fuller, E. (2007). Involvement or isolation? The social networks of children with autism in regular classrooms. *Journal of Autism and Developmental Disorders*, 37(2), 230–242. <https://doi.org/10.1007/s10803-006-0164-4>.
- Cohen, H. D., Rosen, R. C., & Goldstein, L. (1976). Electroencephalographic laterality changes during human sexual orgasm. *Archives of Sexual Behavior*, 5(3), 189–199.
- Cole, T., & Teboul, J. C. B. (2004). Non-zero-sum collaboration, reciprocity, and the preference for similarity: Developing an adaptive model of close relational functioning. *Personal Relationships*, 11, 135–160.
- Constantino, J. N., Davis, S. A., Todd, R. D., Schindler, M. K., Gross, M. M., Brophy, S. L., et al. (2003). Validation of a brief quantitative measure of autistic traits: Comparison of the social responsiveness scale with the autism diagnostic interview–revised. *Journal of Autism and Developmental Disorders*, 33(4), 427–433.
- Dahlgren, S. O., & Trillingsgaard, A. (1996). Theory of mind in non-retarded children with autism and

- Asperger's syndrome. A research note. *Journal of Child Psychology and Psychiatry*, 37(6), 759–763.
- Davidson, R. J., & Irwin, W. (1999). The functional neuroanatomy of emotion and affective style. *Trends in Cognitive Science*, 3(1), 11–21.
- Davidson, R. J., Jackson, D. C., & Kalin, N. H. (2000). Emotion, plasticity, context, and regulation: Perspectives from affective neuroscience. *Psychological Bulletin*, 126(6), 890–909.
- Dawson, G., Meltzoff, A. N., Osterling, J., Rinaldi, J., & Brown, E. (1998). Children with autism fail to orient to naturally occurring social stimuli. *Journal of Autism and Developmental Disorders*, 28(6), 479–485.
- Dennett, D. C. (1978). Beliefs about Beliefs. *Behavioral and Brain Sciences*, 1(4), 568–570.
- Diamond, A., & Kirkham, N. (2005). Not quite as grown-up as we like to think: Parallels between cognition in childhood and adulthood. *Psychological Science*, 16(4), 291–297. <https://doi.org/10.1111/j.0956-7976.2005.01530.x>.
- Dichter, G. S., Radonovich, K. J., Turner-Brown, L. M., Lam, K. S., Holtzclaw, T. N., & Bodfish, J. W. (2010). Performance of children with autism spectrum disorders on the dimension-change card sort task. *Journal of Autism and Developmental Disorders*, 40(4), 448–456. <https://doi.org/10.1007/s10803-009-0886-1>.
- Eckerman, C. O., Davis, C. C., & Didow, S. M. (1989). Toddlers' emerging ways of achieving social coordinations with a peer. *Child Development*, 60(2), 440–453.
- Feldman, R., & Greenbaum, C. W. (1997). Affect regulation and synchronicity in mother-infant play as precursors to the development of symbolic competence. *Infant Mental Health Journal*, 18, 4–23.
- Flavell, J. H., Miller, P. H., & Miller, S. A. (2002). *Cognitive development* (4th ed.). Upper Saddle River: Prentice Hall.
- Gernsbacher, M. A. (2006). Toward a behavior of reciprocity. *The Journal of Developmental Processes*, 1, 139–152.
- Geurts, H. M., Corbett, B., & Solomon, M. (2009). The paradox of cognitive flexibility in autism. *Trends in Cognitive Science*, 13(2), 74–82. <https://doi.org/10.1016/j.tics.2008.11.006>.
- Hammerstein, P. (2003). Why is reciprocity so rare in social animals? A protestant appeal. In P. Hammerstein (Ed.), *Genetic and Cultural Evolution of Cooperation* (pp. 83–94). Cambridge, MA: MIT Press.
- Happaney, K., Zelazo, P. D., & Stuss, D. T. (2004). Development of orbitofrontal function: Current themes and future directions. *Brain and Cognition*, 55(1), 1–10. <https://doi.org/10.1016/j.bandc.2004.01.001>.
- Hill, E. L. (2004). Executive dysfunction in autism. *Trends in Cognitive Science*, 8(1), 26–32.
- Howes, C. (1988). Peer interaction of young children. *Monographs of the Society for Research in Child Development*, 53, 1–78.
- Howlin, P., & Goode, S. (1998). Outcome in adult life for people with autism, Asperger syndrome. In F. R. Volkmar (Ed.), *Autism and pervasive developmental disorders* (pp. 209–241). New York: Cambridge University Press.
- Hughes, C., Russell, J., & Robbins, T. W. (1994). Evidence for executive dysfunction in autism. *Neuropsychologia*, 32(4), 477–492.
- Hughes, C., Adlam, A., Happe, F., Jackson, J., Taylor, A., & Caspi, A. (2000). Good test – retest reliability for standard and advanced false-belief tasks across a wide range of abilities. *Journal of Child Psychology and Psychiatry*, 41(4), 483–490.
- Hutchins, T. L., Prelock, P. A., & Bonazinga, L. (2012). Psychometric evaluation of the Theory of Mind Inventory (ToMI): A study of typically developing children and children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 42(3), 327–341. <https://doi.org/10.1007/s10803-011-1244-7>.
- Kasari, C., Locke, J., Gulsrud, A., & Rotheram-Fuller, E. (2011). Social networks and friendships at school: Comparing children with and without ASD. *Journal of Autism and Developmental Disorders*, 41(5), 533–544. <https://doi.org/10.1007/s10803-010-1076-x>.
- Kavanaugh, R. D. (2006). Pretend play and theory of mind. In L. Balter & C. S. Tamis-LeMonda (Eds.), *Child psychology: A handbook of contemporary issue* (2nd ed., pp. 153–166). New York: Psychology Press.
- Kerr, A., & Zelazo, P. D. (2004). Development of “hot” executive function: the children's gambling task. *Brain and Cognition*, 55, 148–157.
- Lai, M. C., Lombardo, M. V., & Baron-Cohen, S. (2014). Autism. *Lancet*, 383(9920), 896–910. [https://doi.org/10.1016/s0140-6736\(13\)61539-1](https://doi.org/10.1016/s0140-6736(13)61539-1).
- Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.
- Mar, R. A., Tackett, J. L., & Moore, C. (2010). Exposure to media and theory of mind development in preschoolers. *Cognitive Development*, 25(1), 69–78. <https://doi.org/10.1016/j.cogdev.2009.11.002>.
- McEvoy, R. E., Rogers, S. J., & Pennington, B. F. (1993). Executive function and social communication deficits in young autistic children. *Journal of Child Psychology and Psychiatry*, 34(4), 563–578.
- Metcalfe, J., & Mischel, W. (1999). A hot/cool-system analysis of delay of gratification: Dynamics of will-power. *Psychological Review*, 106(1), 3–19.
- Myles, B. S., Bock, S. J., & Simpson, R. L. (2001). *Asperger syndrome diagnostic scale*. Austin: Pro-Ed.
- O'Brien, S. K. (1996). The validity and reliability of the wing subgroups questionnaire. *Journal of Autism and Developmental Disorders*, 26(3), 321–335.
- Ozonoff, S., & Miller, J. N. (1995). Teaching theory of mind: A new approach to social skills training for individuals with autism. *Journal of Autism and Developmental Disorders*, 25(4), 415–433.
- Perez, S. M., & Gauvain, M. (2005). The role of child emotionality in child behaviour and maternal instruction on planning tasks. *Social Development*, 14, 250–272.
- Perner, J., Frith, U., Leslie, A. M., & Leekam, S. R. (1989). Exploration of the autistic child's theory of mind:

- Knowledge, belief, and communication. *Child Development*, 60(3), 688–700.
- Pillow, B. H., & Flavell, J. H. (1986). Young children's knowledge about visual perception: Projective size and shape. *Child Development*, 57(1), 125–135.
- Premack, D., & Woodruff, G. (1978). Does the chimpanzee have a theory of mind? *Behavioral and Brain Sciences*, 1(4), 515–526.
- Prior, M., Dahlstrom, B., & Squires, T. L. (1990). Autistic children's knowledge of thinking and feeling states in other people. *Journal of Child Psychology and Psychiatry*, 31(4), 587–601.
- Rotheram-Fuller, E., Kasari, C., Chamberlain, B., & Locke, J. (2010). Social involvement of children with autism spectrum disorders in elementary school classrooms. *Journal of Child Psychology and Psychiatry*, 51(11), 1227–1234. <https://doi.org/10.1111/j.1469-7610.2010.02289.x>.
- Sakaiya, S., Shiraito, Y., Kato, J., Ide, H., Okada, K., Takano, K., & Kansaku, K. (2013). Neural correlate of human reciprocity in social interactions. *Frontiers in Neuroscience*, 7, 239. <https://doi.org/10.3389/fnins.2013.00239>.
- Sally, D., & Hill, E. (2006). The development of interpersonal strategy: Autism, theory-of-mind, cooperation and fairness. *Journal of Economic Psychology*, 27(1), 73–97. <https://doi.org/10.1016/j.joep.2005.06.015>.
- Scheeren, A. M., Koot, H. M., & Begeer, S. (2012). Social interaction style of children and adolescents with high-functioning autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 42(10), 2046–2055. <https://doi.org/10.1007/s10803-012-1451-x>.
- Stevens, J. R., & Hauser, M. D. (2004). Why be nice? Psychological constraints on the evolution of cooperation. *Trends in Cognitive Science*, 8(2), 60–65. <https://doi.org/10.1016/j.tics.2003.12.003>.
- Szatmari, P., Bartolucci, G., Bremner, R., Bond, S., & Rich, S. (1989). A follow-up study of high-functioning autistic children. *Journal of Autism and Developmental Disorders*, 19(2), 213–225.
- Tager-Flusberg, H. (2001). A re-examination of the theory of mind hypothesis of autism. In J. Burack, T. Charman, N. Yirmiya, & P. Zelazo (Eds.), *The development of autism: Perspectives from theory and research* (pp. 173–193). Mahwah: Erlbaum.
- Tantam, D. (2003). The challenge of adolescents and adults with Asperger syndrome. *Child Adolescence and Psychiatric Clinics of North America*, 12, 143–163.
- Tomasello, M., Carpenter, M., Call, J., Behne, T., & Moll, H. (2005). Understanding and sharing intentions: The origins of cultural cognition. *The Behavioral Brain Sciences*, 28(5), 675–691.; discussion 691–735. <https://doi.org/10.1017/s0140525x05000129>.
- Trevarthen, C., & Aitken, K. J. (2001). Infant intersubjectivity: Research, theory, and clinical applications. *Journal of Child Psychology and Psychiatry*, 42(1), 3–48.
- van Ommeren, T. B., Begeer, S., Scheeren, A. M., & Koot, H. M. (2012). Measuring reciprocity in high functioning children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(6), 1001–1010. <https://doi.org/10.1007/s10803-011-1331-9>.
- Warneken, F., & Tomasello, M. (2006). Altruistic helping in human infants and young chimpanzees. *Science*, 311(5765), 1301–1303. <https://doi.org/10.1126/science.1121448>.
- Wellman, H. M. (1990). *The child's theory of mind*. Cambridge, MA: MIT Press.
- Wellman, H. M. (1993). Early understanding of mind: The normal case. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds.), *Understanding other minds: Perspectives from autism* (pp. 10–39). Oxford, England: Oxford University Press.
- Wellman, H. M., & Banerjee, M. (1991). Mind and emotion: children's understanding of the emotional consequences of beliefs and desires. *British Journal of Developmental Psychology*, 9, 191–214.
- Wellman, H. M., Cross, D., & Watson, J. (2001). Meta-analysis of theory-of-mind development: the truth about false belief. *Child Development*, 72(3), 655–684.
- Wimmer, H., & Perner, J. (1983). Beliefs about beliefs: Representation and constraining function of wrong beliefs in young children's understanding of deception. *Cognition*, 13(1), 103–128.
- Yerys, B. E., Hepburn, S. L., Pennington, B. F., & Rogers, S. J. (2007). Executive function in preschoolers with autism: Evidence consistent with a secondary deficit. *Journal of Autism and Developmental Disorders*, 37(6), 1068–1079. <https://doi.org/10.1007/s10803-006-0250-7>.
- Zelazo, P. D., & Carlson, S. M. (2012). Hot and cool executive function in childhood and adolescence: Development and plasticity. *Child Development Perspectives*, 6(4), 354–360.
- Zelazo, P. D., & Müller, U. (2002). Executive function in typical and atypical development. In U. Goswami (Ed.), *Blackwell handbook of childhood cognitive development* (pp. 445–469). Malden, MA: Blackwell.
- Zelazo, P. D., Qu, L., & Müller, U. (2005). Hot and cool aspects of executive function: Relations in early development. In W. Schneider, R. Schumann-Hengsteler, & B. Sodian (Eds.), *Young children's cognitive development: Interrelationships among executive functioning, working memory, verbal ability, and theory of mind* (pp. 71–93). Mahwah, NJ, US: Lawrence Erlbaum Associates, Publishers.

Cognitive Delay

- Intellectual Disability
- Mental Retardation

Cognitive Enhancement Therapy

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Synonyms

CET; Cognitive rehabilitation; Cognitive remediation; Social cognition training

Definition

Cognitive Enhancement Therapy is a comprehensive cognitive rehabilitation intervention designed to improve the social and non-social cognitive impairments that limit adaptive function and quality of life in certain neurodevelopmental disorders. The treatment integrates computer-based cognitive remediation exercises in attention, memory, and problem-solving with a small group-based social cognitive training curriculum designed to improve perspective-taking, social context appraisal, emotion perception and management, and other key aspects of social cognition. Through these integrated activities, Cognitive Enhancement Therapy addresses the core neurocognitive and social cognitive deficits experienced by verbal adults with autism spectrum disorders.

Historical Background

Cognitive Enhancement Therapy (CET) was developed in the 1990s by Professor G. E. Hogarty for the treatment of social and non-social cognitive impairments in schizophrenia. The development of CET was influenced by the holistic cognitive rehabilitation approach of Ben-Yishay et al. (1985) for individuals suffering from a traumatic brain injury, as well as Brenner's (2000) *Integrated Psychological Therapy* for

schizophrenia. During the late 1990s and early 2000s, Hogarty and colleagues conducted a series of research studies supported by the National Institute of Mental Health that developed CET, established its evidence base for patients with schizophrenia and schizoaffective disorder (Hogarty et al. 2004; Eack et al. 2009), and created a comprehensive treatment manual outlining the methods of the approach (Hogarty and Greenwald 2006). A key finding from these studies was that CET could not only improve core social and non-social cognitive impairments in schizophrenia but that its early application could protect against brain loss and support gray matter growth in areas of the brain involved in social cognitive information processing (Eack et al. 2010b).

The positive findings of CET in schizophrenia led the investigators and other researchers to consider the potential applicability of the intervention to other disorders that are characterized by core deficits in information processing and social cognition. Autism spectrum disorders were viewed as primary candidates due to their chronic and debilitating nature, the presence of pervasive impairments in social and non-social cognition, and lack of available interventions for adults. The many similarities between autism and schizophrenia had led classic psychiatric nosologists to characterize the disorders within the same diagnostic category. Although the categorizing of autism with schizophrenia was ultimately found to be inaccurate, due to sharp distinctions relating to the presence of psychosis and age of onset, evidence continues to highlight that both disorders are similarly affected by marked impairments in neurocognitive and social cognitive function (Goldstein et al. 2002; Velikonja et al. 2019), which may reflect similar pathophysiologic processes (Pinkham et al. 2007). Such findings provide early support for the applicability of CET to adults with autism spectrum disorders.

In 2009, CET investigators S. M. Eack, D. P. Greenwald, and S. S. Hogarty began collaborating with autism expert N. J. Minshew at the University of Pittsburgh to adapt and conduct the initial feasibility studies of CET in autism. With early support from the National Institute of Mental

Health and the Pennsylvania Department of Health, CET treatment materials were successfully adapted from schizophrenia to verbal adults with autism spectrum disorders. In addition, an uncontrolled feasibility study was completed to assess the acceptability of the intervention in autism and measure initial levels of efficacy. As expected, CET proved to be highly applicable to adults with autism and satisfying and acceptable to recipients. Subsequently, a first randomized controlled trial was conducted at the University of Pittsburgh to examine efficacy and establish the evidence base for CET in verbal adults with autism spectrum disorders, which indicated significant improvements in cognitive function and employment in adults with autism treated with the approach (Eack et al. 2018).

Underlying Theory

The underlying theoretical framework for CET relies upon a neurodevelopmental model of schizophrenia (Hogarty and Flesher 1999), which may be applicable to other conditions, such as autism. Neurodevelopmental models situate the core disease process in mental and neurological conditions within the brain, and impairments in brain development and function result in core deficits in cognitive functioning that lead to social and vocational disability. Consistent with neurobehavioral models of autism (Minshew and Goldstein 1998), genetic alterations result in early brain abnormalities that affect the development of neural systems responsible for supporting the acquisition of higher-order social cognitive abilities. CET is a developmental approach in that it aims to facilitate cognitive development by shifting reliance from early and effortful serial cognitive processing to a gistful and spontaneous abstraction of social themes needed for effective interpersonal interaction.

The treatment methods employed in CET find their theoretical basis in models of brain plasticity and sociological theories of secondary socialization. The ability of the brain to be shaped, at even a basic molecular level, by environmental experiences has been well-established and is known as

neuroplasticity (Buonomano and Merzenich 1998). Strategic practice of cognitive exercises that activate different brain systems has been shown to enhance brain function, synaptic connections, and cortical organization (Bryck and Fisher 2012). CET builds on this evidence to provide adults with autism the opportunity to engage in targeted cognitive exercises and experiences designed to enhance brain functions associated with improved social and non-social cognition. The enhancement of social cognition is an essential goal of CET and relies on the sociological principles of secondary socialization, where individuals must develop the ability to abstract informal rules for behavior from appraising the context of unrehearsed social situations. Children are initially socialized using primary socialization methods that focus on direct instruction, usually from parents and teachers (e.g., “use your napkin,” “don’t hit your sister,” “wait until it’s your turn”). However, as the complexity of their social world expands, rigid rules for behavior become less useful, and there is a need for cognitive flexibility in socialization that recognizes the fluidity of adult interactions. Further social cognitive development must utilize secondary sources of information to guide behavior, such as observing the social context and understanding the perspective of others. The development of these abilities is a key aspect of the social cognitive curriculum in CET, which are practiced within a small group context.

Goals and Objectives

The overall goal of CET is to enhance neurocognitive and social cognitive functioning as a method of improving social adjustment, adaptive function, and quality of life. This is achieved through targeting of two broad goals in the treatment. The first goal is to foster higher-order thinking among CET participants. The emphasis in CET is on helping individuals learn to shift from an earlier, pre-adolescent cognitive style of processing that is characterized by passivity, concreteness, rigidity, and rehearsal to a more adult style of information processing that is active,

abstract, flexible, and spontaneous. The second goal is to help individuals develop “social wisdom” or the ability to act wisely in social situations, not by learning and rehearsing scripted rules for behavior but by developing the social cognitive abilities that allow for an accurate assessment of and response to spontaneous interpersonal interactions. To accomplish this, individuals learn how to appraise different social contexts and identify the related rules and norms for behavior; take the perspective of others to understand their feelings and intentions; be foresightful in responding to social situations; acknowledge and participate in the reciprocal nature of social interactions; and ultimately develop a greater level of social comfort. These two goals serve as a guiding foundation for all activities in CET and are actively addressed during both neurocognitive training and social cognitive group sessions.

Treatment Participants

Treatment experiences in providing CET to adults with autism spectrum disorder indicate that many individuals with these conditions are likely to benefit from the intervention. Adults with autism have many strengths that they bring to CET, and the goal is to build on these strengths to help improve adaptive function and quality of life. The treatment is applicable to verbal adults with autism spectrum disorder who have an $IQ \geq 80$ and experience significant social and cognitive disability. Currently, CET treatment materials are only available in the English language, and thus proficiency with English is also important. Individuals unlikely to benefit from CET include those with significant intellectual disabilities ($IQ < 80$), individuals who have not developed language skills, and individuals with an organic brain syndrome, substance use problems, persistent suicidality, or disruptive behaviors not conducive to a group context. Individuals with autism commonly experience comorbid psychiatric conditions, such as anxiety or depression, and as long as these conditions are stabilized and managed appropriately, individuals with these conditions may still benefit from CET.

Treatment Procedures

Cognitive Enhancement Therapy integrates 60 h of computer-based neurocognitive training in attention, memory, and problem-solving with 45 1.5 h weekly social cognitive group sessions provided over the course of 18 months. Treatment begins with a thorough assessment of the participant’s cognitive and social difficulties. Subsequently, participants begin neurocognitive training in attention. Neurocognitive training contains three sequential modules: an attention module, a memory module, and a problem-solving module. Attention training focuses on increasing processing speed, sustaining attention, inhibiting irrelevant stimuli, and shifting attention. Memory training focuses on developing a categorizing capacity, cognitive flexibility, and the ability to abstract and recall the “gist” or main point from information to be remembered. Finally, problem-solving training focuses on improving foresightfulness and planning, reasoning, and executive functioning, and it both relies upon and reinforces the attention and memory abilities gained in previous modules. Unlike other cognitive remediation programs, participants in CET receive neurocognitive training in pairs, which affords opportunities for socialization and to begin learning important early aspects of social cognition, such as giving support. Neurocognitive training is facilitated by a therapist “coach” who promotes strategic thinking about how to complete a given exercise and integrates key CET concepts into the sessions, such as managing emotions, gistful processing, working memory, cognitive flexibility, and foresightfulness.

After approximately 2–3 months of attention training, 3–4 participant pairs (6–8 participants) form a small social cognitive group. The emphasis of these weekly social cognitive group sessions is on enhancing social comfort and the abilities needed for effective socialization, interpersonal interactions, and successful adjustment to adult life. All group sessions follow a highly structured format that allows for spontaneity but also keeps the group process predictable and efficient. Each group session begins by distributing an agenda along with handouts for the psychoeducational

lecture for the day. Participants are then asked to present their homework assignment based on the previous week's lecture, and homework presentations are chaired by one of the group members, who asks the members to volunteer to present their homework. The chairperson role is designed to facilitate working memory, organization, and cognitive flexibility. After the homework presentation is finished, select participants complete an in-group cognitive exercise designed to enhance social cognition. Exercises are usually performed in pairs and require participants to practice the social cognitive abilities they are learning in the group, such as perspective-taking, social context appraisal, gistfulness, and reading nonverbal cues. The group members who are not actively completing the exercise are asked to give feedback to their peers on their performance. This important task promotes observational skills and engagement for group members and provides the opportunity to give support and practice giving appropriate feedback in an organized, constructive way. Finally, a psychoeducational lecture is given on a new topic on social cognition, and homework is assigned to facilitate application outside of the group setting.

A broad number of social cognitive topics are covered throughout the 45 group sessions and include perspective-taking, emotion perception, stress management, understanding the social "gist", giving support, reciprocity, and social context appraisal among others. The goal is to provide participants with the core social cognitive abilities they need to succeed in reaching their goals, build better relationships, and communicate and interact effectively with others. Neurocognitive training proceeds concurrently throughout the time individuals are also participating in the social cognitive groups, until the completion of all attention, memory, and problem-solving computer exercises. A complete descriptions of the original treatment methods and procedures used in CET are outlined in detail in *Cognitive Enhancement Therapy: the Training Manual* by Hogarty and Greenwald (2006), and adaptations specific to adults with autism spectrum disorders are forthcoming in a supplement to this treatment manual.

Efficacy Information

The development of CET for individuals with schizophrenia was supported by the National Institute of Mental Health, and primary data regarding its efficacy comes from two randomized controlled trials with individuals with schizophrenia and schizoaffective disorder. Effects of CET on neurocognition and social cognition in both of these studies were large (range of $d = 0.58$ – 3.09), and effects on functional outcome and social adjustment were also highly significant and large (range of $d = 1.40$ to 2.59) (Hogarty et al. 2004; Eack et al. 2009). Further, mediator analyses from these studies indicated that the benefits CET had on cognition were significant contributors to the improved adjustment and adaptive function of participants (Hogarty et al. 2006; Eack et al. 2011). Evidence of the durability of these effects have also been positive, with studies indicating that the effects of CET on cognition and functioning can be maintained for at least 1 year post-treatment (Hogarty et al. 2006; Eack et al. 2010a). Finally, evidence has indicated that CET has a direct effect on the brain, in that it can protect against gray matter loss and even result in increased levels of gray matter in social cognitive brain networks when applied in the early course of schizophrenia (Eack et al. 2010).

Efficacy information on CET effects specific to the autism population is now available, with the completion of the first randomized controlled trial of this intervention in verbal adults with autism spectrum disorder supported by National Institute of Mental Health, Department of Defense, and Autism Speaks (Eack et al. 2018). This 18-month clinical trial randomized 54 verbal adults with autism with $IQ \geq 80$ to either CET or an enriched supportive therapy (EST) comparison condition that focused on education about autism and stress and condition management. The study included an active comparison condition to CET for two reasons. First, the availability of psychological treatments in routine care for adults with autism is quite limited, reducing the feasibility of a study of CET compared to "treatment as usual." Second, EST includes many of the nonspecific components of CET (e.g., provision of a skilled

emphatic therapist, psychoeducation), and comparison of CET to EST provided a particularly stringent test as to whether the specific cognitive training components in CET could result in improved cognitive and functional outcomes in adults with autism.

Results from this first controlled trial indicated significant and medium-sized ($d = 0.46$, $p = 0.013$) effects on non-social cognition favoring CET compared to EST, particularly in speed of processing, which evidence indicates is one the most affected areas of non-social cognitive function in the condition (Haigh et al. 2018; Velikonja et al. 2019). Effect sizes of CET on non-social cognition over 18 months were medium-to-large in magnitude ($d = 0.76$) compared to EST ($d = 0.31$), which also showed some benefits to non-social information processing. With regard to social cognition, individuals treated with CET demonstrated large improvements ($d = 0.89$), particularly in blind-rated clinical assessments of social cognitive behaviors, as well as domains of emotion perception and management. Interestingly and unexpectedly, individuals treated with EST also evidenced significant and medium-to-large levels of improvement in social cognition ($d = 0.62$) after 18 months of treatment. Differential effects on social cognition clearly favored CET by mid-treatment (9 months) ($d = 0.58$, $p = 0.020$); however the advantage of CET compared to EST for improving social cognition was not maintained by the completion of 18 months of treatment ($d = 0.27$, $p = 0.298$). Such findings were not the result of a loss of social cognitive gains in those treated with CET but a larger than expected improvement in this domain during the second half of treatment among individuals receiving EST. We concluded that while CET appears uniquely effective for addressing non-social cognitive impairments in adult autism, both interventions may have important benefits to social cognition, with CET providing a more rapid treatment response in this domain than EST.

The cognitive gains associated with CET observed in this first trial were hypothesized as key mechanisms for supporting improved functional outcomes in verbal adults with autism

spectrum disorder. When examining the efficacy of CET compared to EST on functional outcomes, clear gains in non-sheltered competitive employment were observed in participants treated with CET that were not evident in those who received EST. Overall, individuals receiving CET were nearly 6 times more likely to become competitively employed during the 18-month trial than their EST counterparts ($OR = 6.21$, $p = 0.005$), indicating the benefits of cognitive rehabilitation to important functional outcomes in this population. Furthermore, improved social and non-social cognition significantly predicted competitive employment outcomes and were partial mediators of the impact of CET on employment, supporting hypotheses that the treatment of cognitive impairments in adults with autism is a productive avenue for enhancing functioning and reducing disability. Given the initial nature of this first trial and its modest sample size, a randomized confirmatory efficacy trial supported by the National Institute of Mental Health is now underway with a larger set of participants to determine whether these effects can be replicated. In addition, follow-up studies 1 year after treatment are being conducted to determine what gains can be maintained once treatment is completed.

Outcome Measurement

Outcome assessment is important in CET to guide clinician behavior and provide feedback on success to participants. Several outcome measures are included in the CET programs that are easy to complete by participants and clinicians and provide a sensitive metric upon which to gauge progress. The attention training software utilized in CET developed by Ben-Yishay et al. (1985) contains a simple reaction time assessment of processing speed, and it is recommended that clinicians utilize this assessment before beginning attention training and every 9 months thereafter until the completion of treatment. Two interview-based assessments, the Social Cognition Profile and the Cognitive Styles Inventory (Hogarty et al. 2004), provide assessments of both

neurocognitive and social cognitive improvement during treatment. It is recommended that these assessments be administered to participants prior to beginning treatment and every 9 months thereafter. These standardized assessment instruments will provide both clinicians and participants with a greater understanding of the cognitive strengths and limitations of the participant and his/her degree of improvement during CET.

For clinics and programs that have access to neuropsychological testing materials, several standardized assessments are useful, but not required for judging progress in CET. These include the revised Wechsler Memory Scale, Wisconsin Card Sorting Test, Trails B, and the California Verbal Learning Test. In addition, the Mayer-Salovey-Caruso Emotional Intelligence Test (Mayer et al. 2003) has proven to be an effective assessment of components of social cognition during CET and is self-administered through a computer. Information from these assessments can be useful for treatment planning, as well as examining progress.

Qualifications of Treatment Providers

Qualified providers of CET are not limited to a single profession. In research studies on CET, individuals from many different backgrounds and disciplines have been successfully trained to provide the treatment. This has included social workers, psychologists, and psychiatric clinical nurse specialists, all of which make up the primary workforce who serve adults with autism spectrum disorders. The critical qualifications for providing CET include education in a human service profession (preferably at a master's level), experience (2 or more years) in the treatment of verbal adults with autism or similar conditions, a willingness to resist a traditional psychotherapeutic approach, and an eagerness and commitment to learn new approaches to intervention.

The methods and intervention approaches employed in CET are novel and likely to be different from any treatment approach clinicians have provided before to adults with autism.

Consequently, beyond having the requisite educational and work experience in order to be knowledgeable about the treatment of autism, it is of utmost importance that potential providers are open and willing to learn a new approach. Some providers become comfortable with certain techniques and have considerable difficulty when a new treatment requires them to deviate from their usual practices. In this way, effective CET clinicians must be cognitively flexible themselves, so that they can open up to new ways to help persons with autism. Clinicians that are uncomfortable with deviating from a traditional psychotherapeutic stance where they provide the instruction and advice to solve the problems for their patients (instead of helping the individual learn to solve their own problems) will have difficulty becoming an effective CET coach.

Finally, it should be noted that a qualified clinician is not enough to provide CET. Group sessions are conducted with a minimum of two qualified clinicians, which significantly enhances the process by introducing a greater diversity of ideas and insights into how to help participants. In addition, the feasibility of implementing CET without strong administrative and supervisory support is limited. As with all successful intervention programs, a commitment is required by clinicians and the agency to invest the necessary resources, training, and time to provide this comprehensive approach. The training required to learn CET is extensive and ongoing, making a strong commitment from the agency essential to successful implementation. In summary, CET is an innovative intervention that holds promise for improving adaptive function and lives of verbal adults with autism spectrum disorders.

See Also

- ▶ [Autistic Disorder](#)
- ▶ [Empirically Supported Treatments](#)
- ▶ [Functional Connectivity](#)
- ▶ [Social Behaviors and Social Impairment](#)
- ▶ [Social Cognition](#)
- ▶ [Theory of Mind](#)

References

- Ben-Yishay, Y., Rattok, J., Lakin, P., Piasetsky, E., Ross, B., Silver, S., Zide, E., & Ezrachi, O. (1985). Neuropsychological rehabilitation, quest for a holistic approach. *Seminars in Neurology*, 5, 252–259.
- Brenner, H. D. (2000). Psychological therapy in schizophrenia: What is the evidence? *Acta Psychiatrica Scandinavica*, 102, 74–77.
- Bryck, R. L., & Fisher, P. A. (2012). Training the brain: Practical applications of neural plasticity from the intersection of cognitive neuroscience, developmental psychology, and prevention science. *American Psychologist*, 67, 87–100.
- Buonomano, D. V., & Merzenich, M. M. (1998). Cortical plasticity: From synapses to maps. *Annual Review of Neuroscience*, 21, 149–186.
- Eack, S. M., Greenwald, D. P., Hogarty, S. S., Cooley, S. J., DiBarry, A. L., Montrose, D. M., & Keshavan, M. S. (2009). Cognitive enhancement therapy for early-course schizophrenia: Effects of a two-year randomized controlled trial. *Psychiatric Services*, 60, 1468–1476.
- Eack, S. M., Greenwald, D. P., Hogarty, S. S., & Keshavan, M. S. (2010a). One-year durability of the effects of cognitive enhancement therapy on functional outcome in early schizophrenia. *Schizophrenia Research*, 120, 210–216.
- Eack, S. M., Hogarty, G. E., Cho, R. Y., Prasad, K. M. R., Greenwald, D. P., Hogarty, S. S., & Keshavan, M. S. (2010b). Neuroprotective effects of cognitive enhancement therapy against gray matter loss in early schizophrenia: Results from a two-year randomized controlled trial. *Archives of General Psychiatry*, 67, 674–682.
- Eack, S. M., Hogarty, S. S., Greenwald, D. P., Litschge, M. Y., Porton, S. A., Mazefsky, C. A., & Minshew, N. J. (2018). Cognitive enhancement therapy for adult autism spectrum disorder: Results of an 18-month randomized clinical trial. *Autism Research*, 11, 519–530.
- Eack, S. M., Pogue-Geile, M. F., Greenwald, D. P., Hogarty, S. S., & Keshavan, M. S. (2011). Mechanisms of functional improvement in a 2-year trial of cognitive enhancement therapy for early schizophrenia. *Psychological Medicine*, 41(6), 1253–1261.
- Goldstein, G., Minshew, N. J., Allen, D. N., & Seaton, B. E. (2002). High-functioning autism and schizophrenia: A comparison of an early and late onset neurodevelopmental disorder. *Archives of Clinical Neuropsychology*, 17, 461–475.
- Haigh, S. M., Walsh, J. A., Mazefsky, C. A., Minshew, N. J., & Eack, S. M. (2018). Processing speed is impaired in adults with autism spectrum disorder, and relates to social communication abilities. *Journal of Autism and Developmental Disorders*, 48, 2653–2662.
- Hogarty, G. E., & Flesher, S. (1999). Developmental theory for a cognitive enhancement therapy of schizophrenia. *Schizophrenia Bulletin*, 25, 677–692.
- Hogarty, G. E., & Greenwald, D. P. (2006). *Cognitive enhancement therapy: The training manual*. University of Pittsburgh Medical Center: Authors. Available through www.CognitiveEnhancementTherapy.com.
- Hogarty, G. E., Flesher, S., Ulrich, R., Carter, M., Greenwald, D., Pogue-Geile, M., Keshavan, M., Cooley, S., DiBarry, A. L., Garrett, A., Parepally, H., & Zoretich, R. (2004). Cognitive enhancement therapy for schizophrenia. Effects of a 2-year randomized trial on cognition and behavior. *Archives of General Psychiatry*, 61, 866–876.
- Hogarty, G. E., Greenwald, D. P., & Eack, S. M. (2006). Durability and mechanism of effects of cognitive enhancement therapy. *Psychiatric Services*, 57, 1751–1757.
- Mayer, J. D., Salovey, P., Caruso, D. R., & Sitarenios, G. (2003). Measuring emotional intelligence with the MSCEIT V2.0. *Emotion*, 3, 97–105.
- Minshew, N. J., & Goldstein, G. (1998). Autism as a disorder of complex information processing. *Mental Retardation and Developmental Disabilities Research Reviews*, 4, 129–136.
- Pinkham, A. E., Hopfinger, J. B., Pelphrey, K. A., Piven, J., & Penn, D. L. (2007). Neural bases for impaired social cognition in schizophrenia and autism spectrum disorders. *Schizophrenia Research*, 99, 164–175.
- Velikonja, T., Fett, A. K., & Velthorst, E. (2019). Patterns of nonsocial and social cognitive functioning in adults with autism spectrum disorder: A systematic review and meta-analysis. *JAMA Psychiatry*, 76, 135–151.

Cognitive Flexibility

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Synonyms

[Cognitive shifting](#); [Mental flexibility](#); [Set shifting](#)

Definition

Cognitive flexibility refers to the cognitive function of adaptively shifting mental actions and behaviors based on feedback from the environment. It is a dimension of executive functioning, which is considered to be impaired in many people with autism spectrum disorders (ASD). Executive functioning refers to a broad category of

complex or higher-order cognitive skills that is crucial for organizing and selecting relevant information, planning responses, and executing actions. Compromised cognitive flexibility in ASD may contribute to challenges with appropriately adapting to changes in interpersonal and environmental demands (e.g., social situations). Difficulty switching problem-solving strategies or adapting responses in the face of situational changes characterize the experiences of people with ASD and can be explained by impairments in cognitive flexibility. Some evidence suggests that cognitive flexibility predicts social outcomes in ASD. Better performance on cognitive flexibility measures alone predicted greater improvement in scores of social competence over time in high-functioning adolescents and adults with ASD (e.g., Berger et al. 2003).

Studies assessing cognitive flexibility and related functions in ASD have found moderately robust findings of impaired flexibility, but not without mixed results. Card sorting tests, such as the Wisconsin Card Sorting Test (WCST; Heaton et al. 1993), are one type of neuropsychological assessment that have been sensitive to measuring aspects of executive functioning, particularly cognitive flexibility. Several aspects of impaired cognitive flexibility in ASD have been identified, yet these factors have yet to be uniformly observed across studies. In one study, high-functioning adolescents with ASD performed significantly poorer on the WCST in maintaining their cognitive set (a set of mental plans or rules deduced from direct feedback) relative to a typically developing control group (Kaland et al. 2008). No other significant differences in performance such as perseverative errors (repeatedly using a previously successful strategy despite feedback that it is no longer effective) were observed. Other studies using the WCST have demonstrated that perseverative errors are more common in individuals with ASD (e.g., Shu et al. 2001) as well as poorer performance on the total number of errors, completion of categories, and number of trials required to complete a category relative to control groups (e.g., Pennington and Ozonoff 1996). A lack of uniform findings indicates that cognitive flexibility is complex and multidimensional. It is

likely affected by various factors related to cognitive and executive functioning, explaining the variations in cognitive flexibility observed across the ASD population.

See Also

- [Attention](#)
- [Executive Function \(EF\)](#)
- [Perseveration](#)
- [Sameness, Insistence On](#)
- [Wisconsin Card Sorting Test \(WCST\)](#)

References

- Berger, H. J. C., Aerts, F. H. T. M., van Spaendonck, K. P. M., Cools, A. R., & Teunisse, J. (2003). Central coherence and cognitive shifting in relation to social improvement in high-functioning young adults with autism. *Journal of Clinical and Experimental Neuropsychology*, 25(4), 502–511.
- Heaton, R. K., Chelune, G. J., Talley, J. L., Kay, G. G., & Curtiss, G. (1993). *Wisconsin card sorting test manual: Revised and expanded*. Odessa: Psychological Assessment Resources.
- Kaland, N., Smith, L., & Mortensen, E. L. (2008). Brief report: Cognitive flexibility and focused attention in children and adolescents with Asperger syndrome or high-functioning autism as measured on the computerized version of the Wisconsin card sorting test. *Journal of Autism and Developmental Disorders*, 38(6), 1161–1165.
- Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 37(1), 51–87. Special Issue: Annual Research Review.
- Shu, B., Lung, F., Tien, A. Y., & Chen, B. (2001). Executive function deficits in non-retarded autistic children. *Autism*, 5(2), 165–174.

Cognitive Measures

- [Differential Ability Scales \(DAS and DAS-II\)](#)

Cognitive Rehabilitation

- [Cognitive Enhancement Therapy](#)

Cognitive Remediation

► Cognitive Enhancement Therapy

Cognitive Shifting

► Cognitive Flexibility

Cognitive Skills

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Definition

The term “cognition” refers to mental processes or forms of information processing. These processes include attention, memory, learning, decision making, reasoning, and problem solving. In the study of autism, a distinction often is drawn between social and/or nonsocial forms of cognition given the presumed centrality of social deficits to the disorder. Some also consider language to be a cognitive domain. The focus of this entry, however, is on cognition that is not explicitly social or language related. For more extensive discussions of social cognition and language, the reader is referred to other definitions in this encyclopedia.

Historical Background

Cognition is a term that became very popular in psychology with the onset of the “cognitive revolution” in the 1950s. This “revolution” resulted from the advent and use of computers. Academic psychology borrowed computer and artificial intelligence information processing concepts and

used them to derive testable hypotheses related to human thought processes. This new field, “cognitive psychology,” had a large impact on the larger discipline of psychology and replaced behaviorism as the dominant paradigm. Rapidly advancing fields of neuroscience, social psychology, and developmental psychology also began to influence the larger field of psychology. This resulted in the genesis of cognitive neuroscience, which seeks to understand the neural and other biological mechanisms underlying thought; social cognitive neuroscience, which employs findings from social psychology and attempts to understand their neurobiology; and developmental cognitive neuroscience, which examines the neurobiology of development in an effort to explain typical and atypical growth and development.

Current Knowledge

Many researchers, writing from different perspectives, have arrived at the conclusion that individuals with ASD exhibit a curious set of cognitive strengths and challenges. For example, Minshew and colleagues (Minshew et al. 1997) articulated the point of view that ASD involves spared simple information processing in domains of motor functioning, memory, language, and reasoning, with selective impairment in complex information processing, not involving visual-spatial processing, across these domains. They went on to argue that “this profile is not consistent with a single primary deficit, but with a multiple primary deficit model in which the deficit pattern within and across domains is reflective of the complexity of the information processing demands. This neuropsychological profile is furthermore consistent with the neurophysiologic characterization of autism as a late information processing disorder with sparing of early information processing.” This way of conceptualizing autism also has been extended to the neuroscience of autism. Here, autism has been portrayed as a disorder involving reduced functional connectivity (synchronous activity) between brain regions and neural circuits (Just et al. 2004), resulting in reduced synchrony among cortical regions supporting higher

cognition. Three important topics in higher cognition in ASD are discussed below.

Cognitive Level and IQ in ASD

First, it is important to provide background about cognitive ability level in ASD because it profoundly influences performance in areas of higher cognition, as well as multiple areas of functioning. The study of cognitive abilities in ASD also has attracted considerable research because the patterning of intellectual strengths and challenges in persons with ASD may be very different than that present in typically developing individuals, and this has clear implications for education and intervention strategies.

One of the challenges inherent in working with individuals with ASD is that they display a very wide range of cognitive ability levels ranging from intellectual disability to intellectual giftedness. Diagnostic conventions have changed considerably over the last 20 years, and while it once was believed that 75% of affected individuals displayed intellectual disability, this figure now has dropped to about 40% according to a Centers for Disease Control publication in 2009 (Rice 2009). Twenty years ago, the most common finding was that individuals with ASD had an IQ profile with stronger visuospatial abilities (as assessed by the block design subtest) than verbal (and especially comprehension subtest) abilities (Goldstein et al. 2008). More recently, it has been argued that those with Asperger syndrome display the opposite profile ($VIQ > PIQ$) or that the intellectual profiles of those with ASD are no different than those found in the general population. These discrepancies may in part be explained by variations in the ages of samples and how they were ascertained. Recently, it has been demonstrated that persons with autism and Asperger syndrome perform better on intelligence tests that assess their perceptual and especially visual reasoning abilities (Raven's Progressive Matrices) versus more language-based tests like the Wechsler scales (Dawson et al. 2007).

Attention in Autism

There is an extensive research literature suggesting that individuals with autism exhibit

atypical patterns of attending to their environment. They may be poor at allocating attention to relevant visual stimuli, and they are at times overly selective and focused in what they attend to. They can find it hard to disengage from what they are viewing. They may have problems switching or redirecting attention between divided streams of information (i.e., visual and auditory stimuli). They may not pay attention to novel stimuli the same way as typically developing persons. This unusual attention processing may affect their development and learning and produce pattern of early emerging but persistent socio-communicative deficits (Dawson et al. 1998).

There are several different conceptual models of attention that have been used to study autism. The first was developed by Mirsky et al. (1991). According to this model, which was derived using a factor analysis (mathematical grouping by common features) of commonly used tests, components of attention include encoding (to receive and interpret incoming information), focusing and executing (to focus and perform a task in the face of distraction), sustaining (to maintain attention over time), and shifting (to adaptively shift the focus of attention). One well-known study in this area found that persons with ASD only showed deficits in focusing and executing and shifting (Goldstein, Johnson, and Minshew 2001).

According to another prominent view (Posner et al. 1984), attention is thought to have three components, which each is subserved by different neural networks and their interactions. The first of these, alerting, involves becoming more sensitive to incoming information on either a tonic (steady state) or episodic (in response to an event) basis. This network selects information from sensory input. The second component in this model is orienting. It is similar to attention shifting (see Allen and Courchesne 2001). The third component of attention is executive control, which is a multidimensional system responsible for inhibition, planning, conflict monitoring, and cognitive flexibility. Similar to Goldstein, Johnson, and Minshew (2001), a recent study using the Posner model found that orienting was impaired in individuals with ASD (Keehn et al. 2010).

Learning and Memory in Autism

Memory in autism is characterized by several unique features (see Boucher and Bowler 2008 for a recent volume on memory in autism). Individuals with cognitive ability levels in at least the average range tend to be as good as typically developing individuals on short-term memory tasks, including those involving auditory, visual, and motor stimuli. Across all functioning levels, they also demonstrate largely preserved non-declarative (implicit) memory (memory that occurs largely outside of conscious awareness). They also are unimpaired at memory tasks involving simple cued recall, where cues are provided to them. However, they are inefficient at spontaneously using contextual cues to help them remember and do not naturally use memory strategies that would leverage these cues. For example, they do not reliably employ grouping cues when presented with list learning tasks that contain semantically similar clusters of words that facilitate recall. Sometimes this failure to use presented cues can be an advantage. For example, they may produce fewer “false” memories on tasks that try to trick the participant into remembering nonpresented materials that are semantically similar to presented ones because they are not distracted by such lures. They also appear to lack the facilitated memory for materials related to themselves (self-referenced memory), which is present in typically developing individuals. They also show deficits in memory for information about emotions.

Individuals with ASDs also display a curious pattern of learning strengths and challenges. Their lower level learning involving contingency-shaped procedures, implicit information, single items of information/facts, and habits is intact. The effectiveness of interventions premised on operant conditioning such as applied behavior analysis (ABA), the ability to memorize large bodies of facts about special interests, and the preference for routines and sameness all constitute evidence of their bias toward lower level learning.

However, individuals with ASDs show deficits in higher level learning that relies on abstract goals and reasoning and on the efficient transfer of learning to new situations and problems

(known as generalization). In order to generalize, organisms must make and keep in mind relational links between memory traces that share common elements. This permits the transfer of what has been learned to novel contexts with similar but largely different features. Individuals with ASDs are inefficient learners. They exhibit excessively narrow stimulus discrimination at the expense of generalization (O’Riordan and Plaisted 2001). They often focus on idiosyncratic and overly selective aspects of stimuli. They fail to recognize and/or maintain online important cues signaling similarities across every day events and settings. Consequently, they are unable to leverage what they already know to navigate new situations. These deficits likely underlie the characteristic academic, social, and adaptive functioning problems they face.

Reasoning and Problem Solving in Autism

Abstract reasoning is a form of higher cognition that requires the mental process of considering and manipulating information about events, objects, and concepts not in the immediate environment. Abstract reasoning is thought to involve both the ability to identify concepts (i.e., to recognize underlying category attributes so as to better understand them) and the ability to form concepts based on these discriminations. This latter function has been referred to as generativity.

Concept identification abilities come online during the first year of life in typical development, and children with autism appear to acquire simple classification abilities involving the physical world (e.g., the ability to sort objects) similarly to children with other developmental delays. However, it is unclear whether individuals with autism can categorize based on more representational and abstract criteria. Some have found that lower functioning children have difficulty with sorting tasks that involve abstract categories, though older higher functioning individuals do not appear to have these difficulties.

In contrast to concept identification, concept formation involves an “open-field” situation in which the individual must initiate behaviors to solve a problem. This type of initiation, or

“generativity,” is recognized as a deficit area for children (Bishop and Norbury 2005) and adults (Turner 1999) with HFASD. Concept formation, but not concept identification, as assessed by the Goldstein-Scheerer object sorting task, has been found to differentiate children with HFASD from others.

Deficits in concept formation may limit the ability of individuals with HFASD to generate cognitive schemas that promote the efficient organization of social and nonsocial information. Impairments in the ability to create organizing schemas for initiating new social behaviors and routines (i.e., meeting new people, entering different and unstructured social situations, and/or conducting reciprocal conversations) would greatly disrupt daily social functioning. Similarly, the inability to conceptualize, represent, and integrate multiple perspectives would produce deficits in interpersonal relationships. The replicated finding that generativity is predictive of play quality in young children supports these assertions.

Several studies have used tasks similar to the guessing game “20 questions,” where players must guess the identity of items (persons, places, or things) in less than 20 questions. To perform well, they must ask efficient questions that narrow the possible guesses on each try. Individuals with ASD perform poorly on this task, and their task performance reliably distinguishes them from typically developing individuals. On this type of task, they tend to use strategies that eliminate single items versus groups of items. It has been suggested that this reflects a fundamental problem in concept formation – the spontaneous generation of categories to support goal-directed cognitive processing. These problems appear to be related somewhat to the executive function deficits of affected individuals.

Finally, following the study described above that high-functioning persons with autism perform better on the intelligence tests that involve more visuospatial versus verbal reasoning – the Raven’s Progressive Matrices test (Dawson et al. 2007) – there now have been several that show that aspects of conditional and analogical reasoning are relatively preserved in persons with ASDs.

Future Directions

Cognition is a very important topic in ASD research because it appears that the disorders involve unique strengths and challenges in this area, which are integrally related to educational, social, and behavioral functioning. A better understanding of cognitive style in ASD would produce important information that helps us understand the biological basis of the disorders and what to do to help those with them to achieve their human potential.

Some interesting areas for future research include:

1. More extensive use of evoked response potential studies which are particularly well suited to studying attention. These can be coupled with imaging studies to learn more about the neural substrates of higher cognition problems and what to do to help those with them.
2. The Raven’s Progressive Matrices findings outlined above are intriguing and point to the clear strengths of persons with ASD. We need more research to better elaborate such strengths and how they can be harnessed in the service of educational strategy development and vocational training.
3. The study of development of cognition through the lifespan in persons with ASD is in its infancy. The field is only now publishing findings about cognitive control in older adulthood. More studies are needed that cover the full range of cognitive abilities in toddlers, children, adolescents and adults with ASD.
4. There have been virtually no studies of decision making in persons with ASD. This is an important gap in knowledge, given that such these would be useful to help with the transition to adulthood.
5. More learning studies are needed to examine the neural substrates of learning in persons with ASD. These will help provide an evidence base for ABA and may teach us about effective strategies for helping persons with ASD to overcome their deficits in generalization.

References and Reading

- Allen, G., & Courchesne, E. (2001). Attention function and dysfunction in autism. *Frontiers in Bioscience*, 6, 105–119.
- Bishop, D. V. M., & Norbury, C. (2005). Executive function in children with communication impairments, in relation to autistic symptomatology. 1: Generativity. *Autism*, 9, 7–27.
- Boucher, J., & Bowler, D. (2008). *Memory in autism*. Cambridge: Cambridge University Press.
- Carpenter, M., Pennington, B. E., & Rogers, S. J. (2002). Interrelations among social-cognitive skills in young children with autism. *Journal of Autism and Developmental Disorders*, 32(2), 91–106.
- Dawson, G., Meltzoff, A., Osterling, J., Rinaldi, J., & Brown, E. (1998). Children with autism fail to orient to naturally occurring social stimuli. *Journal of Autism and Developmental Disorders*, 28, 479–485.
- Dawson, M., Soulières, I., Gernsbacher, M., & Mottron, L. (2007). The level and nature of autistic intelligence. *Psychological Science*, 18, 657–662.
- Frith, U. (2000). Cognitive explanations of autism. In K. Lee (Ed.), *Childhood cognitive development: The essential readings* (Essential readings in development psychology) (pp. 324–337). Malden: Blackwell Publishers.
- Just, M., Cherkassy, V. L., Keller, T., & Minshew, N. (2004). Cortical activation and synchronization during sentence comprehension in high-functioning autism: Evidence of underconnectivity. *Brain*, 127, 1811–1821.
- Keehn, B., Lincoln, A., Muller, R.-A., & Townsend, J. (2010). Attentional networks in children and adolescents with autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 51, 1251–1259.
- Minshew, N., Goldstein, G., & Siegel, D. J. (1997). Neuropsychologic functioning in autism profile of a complex information processing disorder. *Journal of the International Neuropsychological Society*, 3, 303–316.
- Mirsky, A. F., Anthony, B. J., Duncan, C. C., Ahearn, M., & Kellam, S. (1991). Analysis of the elements of attention: A neuropsychological approach. *Neuropsychological Review*, 2, 109–145.
- O’Riordan, M., & Plaisted, K. (2001). Enhanced discrimination in autism. *Quarterly Journal of Experimental Psychology*, 54, 961–979.
- Posner, M., Walker, J. A., Friedrich, F. J., & Rafal, R. (1984). Effects of parietal injury on covert orienting of attention. *Journal of Neuroscience*, 4, 1863–1874.
- Russo, N., Flanagan, T., Iarocci, G., Berringer, D., Zelazo, P. D., & Burack, J. A. (2007). Deconstructing executive deficits among persons with autism: Implications for cognitive neuroscience. *Brain & Cognition*, 65(1), 77–86.
- Sahyoun, C., Soulières, I., Belliveau, J. W., Mottron, L., & Mody, M. (2009). Cognitive differences in pictorial reasoning between high-functioning autism and Asperger’s syndrome. *Journal of Autism and Developmental Disorders*, 39(7), 1014–1023.
- Sandberg, A. D., Nyden, A., Gilberg, C., & Hjelmquist, E. (1993). The cognitive profile in infantile autism: A study of 70 children and adolescents using the Griffiths Mental Developmental Scale. *British Journal of Psychology*, 84(3), 365–373.
- Solomon, M., Ozonoff, S. J., Cummings, N., & Carter, C. S. (2008). Cognitive control in autism spectrum disorders. *International Journal of Developmental Neuroscience*, 26(2), 239–247.
- Turner, M. (1999). Generating novel ideas: Fluency performance in high-functioning and learning disabled individuals with autism. *Journal of Child Psychology and Psychiatry*, 40, 189–201.

Cohen Syndrome

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Definition

Cohen syndrome (Mendelian Inheritance in Man [MIM 216550]) is an autosomal recessive disorder which was first described in 1973 by Cohen and coworkers (Cohen et al. 1973). The first patients studied with Cohen syndrome were reported to have mental retardation, microcephaly, antimongoloid slant, mild maxillary hypoplasia, short philtrum, open mouth with prominent maxillary central incisors, micrognathia, highly arched narrow palate, crowded teeth, hypotonia, obesity, narrow hands and feet, tapering extremities, cubitus valgus, genua valga, lumbar lordosis, mild thoracic scoliosis, and hyperextensibility of the joints. Subsequent to this first report, there have been descriptions in the literature of more than 100 patients suggested to have Cohen syndrome Fig. 1.

Historical Background

History

In 1968 and 1972, Dr. Michael Cohen and his collaborators from the United States observed



Cohen Syndrome, Fig. 1 Case 1 showing the characteristic facial appearance (a). Case 2 showing the characteristic body shape of Cohen syndrome: a truncal distribution of body fat, with comparatively slender limbs (b). Case

3 showing the prominent upper incisors, high nasal bridge, and antimongoloid slant of the eyes (c) (From archive of Ozgur Pirgon)

two sibs and a third patient, respectively, with a previously unrecognized pattern of abnormalities. In 1973, they diagnosed these patients with a newly recognized syndrome (Cohen et al. 1973). In another report, Carey and Hall established Cohen syndrome as a clinical entity by presenting four new patients with similar findings (Carey and Hall 1978).

Since then, a large number of patients with Cohen syndrome have been found in Finland. Six of these were reported in 1984 by Norio et al. on a small group of Finnish patients with Cohen syndrome, presenting microcephaly, neutropenia, and specific ophthalmic abnormalities, namely, high myopia and retinal dystrophy (Norio et al. 1984). Recently, a novel disease-causing gene (COH1; chromosome 8q22) was identified in this interval which encodes a transmembrane protein presumably involved in vesicle-mediated sorting and intracellular protein transport (Kolehmainen et al. 2003).

Genetics and Etiology

Many of the reported patients have been sibs, with healthy parents. In five Finnish families, the parents were consanguineous. Thus, Cohen syndrome is considered to be an autosomal recessive disorder. In Finland, linkage studies were performed with the assumption of autosomal recessive inheritance, and the gene was mapped to

the long arm of chromosome 8 (Tahvanainen et al. 1994).

The gene responsible for Cohen syndrome, VPS13B (MIM# 607817), also known as COH1, is located on chromosome 8q22 (Kolehmainen et al. 2003). VPS13B is a large gene: it spans a region of 864 kb and has 62 exons, with putative transmembrane domains and functional motifs specific for intracellular vesicle-mediated protein sorting (VPS) (Kolehmainen et al. 2003; Seifert et al. 2009; Velayos-Baeza et al. 2004). The function of the protein encoded by COH1 is mostly unknown. Although in the majority of patients clinically diagnosed as having Cohen syndrome, homozygous or compound heterozygous mutations in VPS13B are identified, in about 20–30%, only one heterozygous mutation is detected, and in 12%, no mutations are found (Kolehmainen et al. 2004; Seifert et al. 2009). Overall, more than 50 COH1 mutations have been reported in association with Cohen syndrome. Most are termination mutations and predicted to result in a null allele, while missense mutations and larger deletions are less common (Seifert et al. 2006). For these patients, the underlying cause remains uncertain.

The mouse homologue of COH1 is widely expressed in neurons of the postnatal and adult brain suggesting a role in neuronal differentiation (Mochida et al. 2004). This suggests that COH1

primarily functions in postmitotic cells, which may be the reason for the postnatal microcephaly seen in Cohen syndrome (Seifert et al. 2006).

Incidence and Prevalence

Cohen syndrome is a rare autosomal recessive disorder with incidence rates (based on Finnish data) estimated as 1 per 105,000 (Kivitie-Kallio et al. 1999). To date over 100 reports of individuals with Cohen Syndrome have been published, but most are single case or small group studies, and relatively few involve large samples. Patients have been identified worldwide but are overrepresented in the Finnish population.

Diagnosis

Establishing the clinical diagnosis of Cohen syndrome has historically been challenging. The first patients studied with Cohen syndrome were reported to have mental retardation, microcephaly, antimongoloid slant, mild maxillary hypoplasia, short philtrum, open mouth with prominent maxillary central incisors, micrognathia, highly arched narrow palate, crowded teeth, hypotonia, obesity, narrow hands and feet, tapering extremities, cubitus valgus, genua valga, lumbar lordosis, mild thoracic scoliosis, and hyperextensibility of the joints.

The clinical diagnosis of Cohen syndrome is difficult to make in infancy. At infancy, the characteristic facial features are difficult to recognize, but they become more and more evident by 5–10 years of age and remain recognizable for decades but tend to lose their most characteristic appearance after middle age (Kivitie-Kallio et al. 2001). Neonatal feeding problems are common. Babies are often hypotonic during the first months of life.

The initial description of Cohen syndrome features included obesity, mental retardation, hypotonia, narrow hands and feet, and a distinctive craniofacial appearance (Cohen et al. 1973). Broad phenotypic variability in subsequent patients diagnosed with Cohen syndrome has created significant confusion as to diagnostic accuracy (Chandler and Clayton-Smith 2002; Friedman and Sack 1982). The characteristic facial appearance, developmental delay, myopia,

narrow hands with slender and tapering fingers, narrow feet, and generalized joint hyperextensibility were present in all patients with Cohen syndrome investigated for their study. However, microcephaly, short stature, truncal obesity, neutropenia, and retinopathy were not present in some of the patients.

Greater clinical variability is observed in case reports of Cohen syndrome from outside Finland. A comparison of features among different Cohen syndrome populations with shared linkage to the COH1 locus or known COH1 gene mutations may help better define criteria for which to suspect Cohen syndrome.

Although the various physical abnormalities associated with Cohen syndrome have been widely documented, there is still a lack of consistency in diagnosis. The facial features are often a first indication of a diagnostic pathway in the examination of patients presenting with developmental delay.

In 2001, Kivitie-Kallio and Norio proposed the following features as essential for the diagnosis of Cohen syndrome:

1. Nonprogressive mental retardation, motor clumsiness, and microcephaly
2. Typical facial features, including wave-shaped eyelids, short philtrum, thick hair, and low hairline
3. Childhood hypotonia and joint hyperextensibility
4. Retinochoroidal dystrophy and myopia by 5 years of age
5. Periods of isolated neutropenia

In 2003, Chandler et al. linked the diagnosis of Cohen syndrome to the presence of at least two of the following major criteria in a child with significant learning difficulties:

1. Facial gestalt, characterized by thick hair, eyebrows, and eyelashes; wave-shaped, downward slanting palpebral fissures; prominent, beak-shaped nose; short, upturned philtrum with grimacing expression on smiling
2. Pigmentary retinopathy
3. Neutropenia (defined as $<2 \times 10^9/L$)

Cohen Syndrome, Table 1 Diagnostic criteria for diagnosis of Cohen syndrome seen in individuals from various ethnic backgrounds with proven COH1 mutations (Falk et al. 2004)

<i>Major signs</i>	Retinal dystrophy appearing by midchildhood
	Progressive high myopia
	Acquired microcephaly
	Nonprogressive mental retardation, global developmental delay
	Hypotonia
	Joint hyperextensibility
<i>Minor signs</i>	Truncal obesity appearing in or after midchildhood
	Small or narrow hands and feet
	Short stature
	Friendly disposition
	Noncyclic granulocytopenia or low total white blood cell count with or without aphthous ulcers

In association with a number of less specific but supportive criteria, namely:

- 1. Early-onset, progressive myopia
- 2. Microcephaly
- 3. Truncal obesity with slender extremities
- 4. Joint hyperextensibility

See (Table 1)

Current Knowledge

Differential Diagnosis

Conditions to be considered in the differential diagnosis of the Cohen syndrome include the Prader-Willi syndrome, Rubinstein-Taybi syndrome, Borjeson-Forssman-Lehmann syndrome, Bardet-Biedl syndrome, and Mirhosseini-Holmes-Walton syndrome. Mirhosseini-Holmes-Walton syndrome may be the same syndrome as Cohen syndrome (Steinlein et al. 1991). However, the clinical phenotype of these conditions is quite distinct and very different from that of Cohen syndrome. Deafness, diabetes mellitus, and cardiomyopathy are characteristic of Alström syndrome while the patients are usually of normal intellect (Michaud et al. 1996). Postaxial

polydactyly and renal dysplasia are diagnostic features of Bardet-Biedl syndrome (Beales et al. 1997). Prader-Willi syndrome is characterized by severe hypotonia and feeding difficulties in early infancy, followed in later infancy or early childhood by excessive eating and obesity.

Specific Characteristics

Cohen syndrome has been proposed to be subdivided into two types (i.e., Finnish type and Jewish type) (Kondo et al. 1990). Phenotypes common to both types are nonprogressive psychomotor retardation, microcephaly, characteristic facial features, retinal dystrophy, and intermittent neutropenia (Friedman and Sack 1982; Sack and Friedman 1986). The characteristic facial features include high-arched or wave-shaped eyelids, a short philtrum, thick hair, and low hairline. Both types are further classified by the presence (Finnish type) or absence (Jewish type) of retinal anomalies (Kondo et al. 1990).

Neurological

Intracranial abnormalities have been reported in Cohen syndrome patients; however, the findings are inconsistent. Microcephaly, considered by some authors (Fryns et al. 1996; Norio et al. 1984) as a typical and early symptom of the syndrome, was mildly evident from the neonatal period in our two patients and, apart from mild hypotonia, was not associated with major neurological dysfunction (Balestrazzi et al. 1980; Carey and Hall 1978).

Hypotonia can be either of central origin or related to a possible connective tissue disorder (Norio et al. 1984). All patients learn to walk by the age of 2 ± 5 years. The incidence of seizures in Cohen syndrome is approximately 6% (North et al. 1985). EEG abnormalities in Cohen syndrome have also been reported in two cases by Goecke, Majewski, Kauther, and Sterzel (1982). Except for oppiness, no signs of muscle disease are found.

Brain MRI is considered normal, although corpus callosum is relatively enlarged. Previous studies on MRI findings in Cohen patients disclosed no focal signal intensity alterations in the brain and no alterations between the gray and white



matter (Kivitie-Kallio et al. 2001). Furthermore, some authors reported in some patients with Cohen syndrome a relatively enlarged corpus callosum, thus suggesting to consider this a possible hallmark of the syndrome. An increased diameter of the body of the corpus callosum was described in a group of 15 Finnish Cohen syndrome patients (Kivitie-Kallio and Norio 2001), a clinical feature not reported by other large studies of non-Finnish Cohen syndrome patients (Hennies et al. 2004; Kolehmainen et al. 2004; Seifert et al. 2006).

All authors also proposed that though MRI alone cannot confirm the diagnosis and no definite measurements can be recommended for clinical use, any clinical suspicion of Cohen syndrome would have been reinforced by the MRI finding of an enlarged corpus callosum in a microcephalic head and normal signal intensities of gray and white matter (Kivitie-Kallio et al. 2001).

Craniofacial

Craniofacial features are often essential in syndrome diagnosis. Suspicion of Cohen syndrome usually arises when a mentally retarded child has facial features considered typical of this syndrome. Reduced head size (microcephaly), short philtrum, and small cranial base dimensions are essential features in Cohen syndrome. In addition, most patients had forward-inclined upper incisors and maxillary prognathia (Carey and Hall 1978; Ozturk and Weber 1991). Head circumferences of Cohen subjects were very small, in the order of mean values for 5–6-year-old Finnish children and at the level of -4 SD in Finnish head circumference standards (Sorva et al. 1984).

Other craniofacial features, such as anti-mongoloid slant of the eyelids, high-arched or wave-shaped eyelids, long and thick eyebrows, prominent root of nose, short philtrum, prominent upper central incisors, open mouth appearance, maxillary hypoplasia, high and narrow palate, and mandibular micrognathia, have been described (Goecke et al. 1982; Kondo et al. 1990; Warburg et al. 1990).

Despite variability in the facial appearance, several specific features can be identified in patients from different countries. Facial features

also seem to differ between populations. Finnish Cohen syndrome patients (81–100%) are described as having a distinctive facial appearance including wave-shaped eyelids, thick and high-arched eyebrows, long eyelashes, thick hair, low hairline, high nasal bridge, flat philtrum, short upper lip, prominent and broad upper central incisors, open mouth, and micrognathia (Kivitie-Kallio and Norio 2001). The UK cohort was found to have a similar facial appearance as the Finnish, with the additional description of a beak-shaped nose, malar hypoplasia, and a grimace-like smile (Chandler et al. 2003).

Ophthalmologic

Cohen syndrome is also characterized by progressive myopia and pigmentary retinopathy, as first described in two of three patients by Cohen et al. (1973). Other reported ophthalmic features include astigmatism, strabismus, microcornea, microphthalmia, sluggish pupillary reaction, iris atrophy and oval pupil, lens opacities, lens subluxation, optic atrophy, bull's-eye maculopathy, coloboma of the retina or lids, ptosis, exophthalmos, poor vision acuity, nyctalopia, and constricted visual fields (Chandler and Clayton-Smith 2002; Kivitie-Kallio et al. 2000). In the UK study, however, a significant proportion of affected adults were found to develop blindness with time (Chandler et al. 2002).

Retinal dystrophy was recorded by Cohen et al. (1973) in their original description of the syndrome. The pigment deposits increase and approach the posterior pole by 35–40 years of age (Kivitie-Kallio et al. 2000). All except two children aged 2 and 5 years of the 22 Finnish patients showed signs of the pigmentary retinopathy (Kivitie-Kallio et al., 2000). Early studies of Cohen syndrome patients showed that abnormal retinal findings and electroretinographic changes were present much earlier (Chandler et al. 2002). Useful vision is usually preserved until the fourth decade (Kivitie-Kallio et al. 2000).

Retinal dystrophic changes were accompanied by early night blindness. This symptom is commonly seen in adolescents. In Cohen syndrome; ocular anomalies are also common finding

including strabismus, hyperopia, astigmatism, microphthalmia, coloboma of the iris, and, most frequently, severe myopia. Recent reports also indicate involvement of the chorioretinal epithelium (Mendez et al. 1985; Norio et al., 1984).

Myopia and astigmatism are common findings in Cohen syndrome. Myopia was noted in 44% of patients in the literature. Myopia in Cohen syndrome is mainly refractive in type and is due to high corneal and lenticular power (Summanen et al., 2002).

Strabismus was reported in 29% of patients from literature review, with divergent strabismus being more common than convergent (Fryns et al., 1996; Warburg et al., 1990). Chandler et al. (2002) reported strabismus in up to 80% of patients. Downslanting eyelids were present in 71% of patients, while 13% were reported to have ptosis. Lens opacities were present in 13 of 22 Finnish patients (Kivitie-Kallio et al., 2000). Small cortical opacities were noted in patients as early as 15 years of age; biomicroscopy and lens opacitometry show frequent incidence of early nuclear sclerosis in patients with Cohen syndrome (Summanen et al., 2002). Older patients also had posterior subcapsular cataracts, iris atrophy, and iridophacodonesis (Kivitie-Kallio et al., 2000).

Musculoskeletal

In the first report of this syndrome, all of the three patients had lumbar lordosis, mild thoracic scoliosis, cubitus valgus, genu valgum, and narrow hands and feet (Cohen et al., 1973). The hands and fingers of Cohen patients were reported to be long and slender (Kousseff, 1981; Norio et al., 1984). Since the first descriptions, the most common abnormalities reported are kyphoscoliosis and pes calcaneovalgus (Norio et al., 1984; North et al. 1985; Sack and Friedman 1986).

The metacarpophalangeal pattern profile was characteristic: all measured bones of the hands were short, the medial and especially the distal phalanges were the shortest, and the proximal phalanges were relatively the longest. Their hands and feet are small and narrow, as given in earlier reports (Cohen et al. 1973; Norio et al. 1984).

Endocrine

In many previous studies, Cohen syndrome patients have been reported to be short (Carey and Hall 1978; Cohen et al. 1973; North et al. 1995). Short stature is a universal feature among the Amish and Lebanese Cohen syndrome patients, but was seen in only 40% of the Finnish Cohen syndrome patients and 64% of the UK Cohen syndrome patients. Heights were highly variable and ranged from -5.7 to -0.3 SD. Both impaired growth hormone (Massa et al. 1991) and normal growth hormone secretion have been reported (Carey and Hall 1978).

Mild truncal obesity was present in most patients at midchildhood, but that trait may be lacking in adult patients. Other studies have described different frequencies of truncal obesity, from 17% to 100% in patients aged ≥ 8 years (Chandler et al. 2003; Kivitie-Kallio and Norio 2001).

Puberty in Cohen syndrome patients is mostly delayed (Kivitie-Kallio et al. 1999), although they do reach sexual maturity. Delayed puberty has only been reported in the Finnish and UK cohorts (77% and 40%, respectively). No endocrine abnormalities are found. The other reported endocrinologic study showed delayed onset of puberty without luteinizing hormone and follicle-stimulating hormone deficiency in Cohen syndrome (Balestrazzi et al. 1980). However, delayed puberty might be due to the obesity.

Two girls, one with diabetes mellitus (Nambu et al. 1988) and the other with impaired glucose tolerance test (Fuhrmann-Rieger et al. 1984), have been noted. No abnormalities of the hypophyseal-gonadal axis were found. Kivitie-Kallio et al. found no significant endocrinologic abnormalities in their patients with Cohen syndrome; however, they did not perform an oral glucose tolerance test in their obese cases for diabetes or insulin resistance (Kivitie-Kallio et al. 1991).

Pirgon et al. reported that two patients showed the typical characteristics of Cohen syndrome with metabolic syndrome features of acanthosis nigricans, hyperlipidemia, hypertension, and marked hyperinsulinemia (Pirgon et al. 2006).

Developmental Delay and Mental Retardation

All patients had a global developmental delay of variable degree and nonprogressive mental retardation. Intellectual impairment is considered to be an essential criterion by some groups of researchers (Kivitie-Kallio and Norio 2001).

Mental retardation together with motor delay, due to hypotonia, is present from early life in most reported cases of Cohen syndrome. The majority of patients have an IQ less than 50 (Goecke et al. 1982), although there have been reports of children showing mild to moderate degree of retardation. Most reported cases of Cohen syndrome present with mental retardation accompanied by motor delay and hypotonia in early life. Patients are not able to attend normal school; thus, they all need special schools. Mental retardation does not progress, and patients learn new things.

Behavioral

Cohen syndrome patients usually have cheerful disposition and have not been associated with maladaptive behavior. Language is possibly lacking or severely impaired in infancy. Autistic traits are found during childhood (Fryns et al. 1996). Children and young people diagnosed with Asperger's syndrome have significant social-communication difficulties and impaired empathy and theory of mind skills. These difficulties place them at risk of developing mental health problems, particularly anxiety, depression, and obsessive-compulsive disorder.

A recent study of cognitive and adaptive skills (Karpf et al. 2004) indicated that some individuals may have an IQ in the normal range, and although some research (Kivitie-Kallio et al. 1999) has reported low levels of maladaptive behavior and high levels of self-direction, responsibility, and socialization, there are also accounts of greater behavioral disturbance (Chandler et al. 2003). Fryns et al. (1996) reported autistic behavior patterns in four patients and in a postal survey of 33 children and young adults with Cohen syndrome.

Reports from a number of parents belonging to the Cohen Syndrome Support Group in the UK had indicated significant difficulties in social interaction; moreover, there were several cases

of individuals with Cohen syndrome also being diagnosed as having autism. Howlin (2001) found that over half the sample had problems in social understanding, communication, and ritualistic and stereotyped behaviors. Howlin et al. (2001) also suggested that although antisocial behaviors are rare, symptoms of anxiety are common, and in some individuals, autistic-type features are marked. Their investigation of 45 individuals with Cohen syndrome (age 4–48 years) found that although 57% of the sample were reported as showing some behavioral disturbance, problems related mainly to anxiety and social interactions, marked antisocial behaviors were rare.

Kivitie-Kallio and her colleagues (Kivitie-Kallio and Norio 2001) noted that “inappropriate interpersonal manners, stereotyped behavior and odd mannerisms were not uncommon.” One of their cases had also shown autistic behavior as an infant, although this had improved after the age of 3 years. In the Chandler et al. study (2003) of 27 patients, 74% exhibited stereotyped behaviors, such as spinning, and five cases (18%) were observed to show autistic features (communication and social abnormalities and ritualistic and obsessive behavior).

Infections

Many children with Cohen syndrome have recurrent upper respiratory infections. However, patients have no fatal infections, and granulocytes seem to rise normally in cases of severe bacterial infections.

Several of the children had intermittent granulocytopenia. The granulocytopenia is usually mild, not progressive, and does not seem to be associated with recurrent infections. While intermittent granulocytopenia is frequently described in the Finnish group (100%), UK group (77%), and was present in two Amish patients evaluated for this problem with one additional child having symptomatic aphthous ulcers, it was not present in blood counts of any of the Lebanese Cohen syndrome patients. Importantly, no severe infections were reported in association with this finding.

Chandler et al. (2003) reported that stridor secondary to laryngomalacia was common in

infancy among the Jewish-type Cohen syndrome patients and more significant laryngeal abnormalities were also reported, namely, laryngeal stenosis and vocal cord paralysis.

Cardiovascular

Numerous cardiac abnormalities have been reported. These include mitral valve prolapse in two patients (Sack and Friedman 1980; Mehes et al. 1988). Systolic murmur (grades II–IV) in five of six patients (Norio et al. 1984) and a dilated descending aorta (Schlichtemeier et al. 1994) have also been reported. Although 30% of the patients had systolic murmurs, echo studies revealed no abnormalities in their cardiac anatomy.

Future Directions

Management

Early diagnosis of the syndrome is important for appropriate counseling of families with one affected child. Although most of the clinical findings are usually present from an early age, the diagnosis of Cohen syndrome is very difficult in infancy, since the typical morphological stigmata become more evident after the age of 6–8 years (Fryns et al. 1996).

Newborns have low-normal weights, and the onset of obesity is generally in midchildhood (Carey and Hall 1978; Cohen et al. 1973). Severe obesity is rare, and some patients may not develop obesity at all (Friedman and Sack 1982). The relationship among the obesity, hypotonia, and hypogonadism has not been clarified. Nutritional counseling for good long-term weight management should begin in early infancy to prevent the inappropriate weight gain that would otherwise typically begin between 12 and 36 months of age.

Behavioral problems should be detected early and treated appropriately with parental education/training (including consistent limit setting) and, if needed, consideration of counseling and/or psychotropic medication. Developmental assessment should be performed routinely. Physical and occupational therapies should begin in infancy to facilitate the development of motor milestones.

Speech therapy is important in monitoring receptive-expressive language skills. In addition, appropriate educational intervention throughout the school years that addresses individual strengths and challenges as well as behavioral issues can be effectively implemented in both inclusion and self-contained classroom settings depending on individual needs.

Long-term follow-up and clinical information on patients older than 40 years are rare in the literature. Marked deterioration of visual function, and even total blindness, can occur over the age of 50 years (Seifert et al. 2006). Kyphoscoliosis can be observed in patients with Cohen syndrome as teenagers or adults, and this tends to be progressive through adult life.

Appropriate management of children with Cohen syndrome requires collaborative efforts from the geneticist, neurologist, endocrinologist, developmental-behavioral pediatrician, nutritionist, psychologist, psychiatrist, educational specialist, and the family. Once the diagnosis of Cohen syndrome is confirmed, it is important for the child to receive multidisciplinary care in addition to routine preventive health care from the primary care physician.

See Also

► [Intellectual Disability](#)

References and Reading

- Balestrazzi, P., Corini, L., Villani, G., Bolla, M. P., Casa, F., & Bernasconi, S. (1980). The Cohen syndrome: Clinical and endocrinological studies of two new cases. *Journal of Medical Genetics*, 17, 430–432.
- Beales, P. L., Warner, A. M., Hitman, G. A., Thakker, R., & Flintner, F. A. (1997). Bardet-Biedl syndrome: A molecular and phenotypic study of 18 families. *Journal of Medical Genetics*, 34, 92–98.
- Carey, J. C., & Hall, B. D. (1978). Confirmation of the Cohen syndrome. *Journal of Pediatrics*, 93, 239–244.
- Chandler, K. E., & Clayton-Smith, J. (2002). Does a Jewish type of Cohen syndrome truly exist? *American Journal of Human Genetics*, 111, 453–454.
- Chandler, K. E., Kidd, A., Al Gazali, L., Kolehmainen, J., Lehesjoki, A. E., Black, G. C., & Clayton-Smith, J. (2003). Diagnostic criteria, clinical characteristics,

- and natural history of Cohen syndrome. *Journal of Medical Genetics*, 40, 233–241.
- Cohen, M., Hall, B., Smith, D., Graham, B., & Lampert, K. (1973). A new syndrome with hypotonia, obesity, mental deficiency, and facial, oral, ocular and limb anomalies. *Journal of Pediatrics*, 83, 280–284.
- Falk, M. J., Feiler, H. S., Neilson, D. E., Maxwell, K., Lee, J. V., Segall, S. K., Robin, N. H., Wilhelmsen, K. C., Träskelin, A. L., Kolehmainen, J., Lehesjoki, A. E., Wiznitzer, M., & Warman, M. L. (2004). Cohen syndrome in the Ohio Amish. *American Journal of Medical Genetics*, 128, 23–28.
- Friedman, E., & Sack, J. (1982). The Cohen syndrome: Report of five new cases and a review of the literature. *Journal of Craniofacial Genetics and Developmental Biology*, 2, 193–200.
- Fryns, J. P., Leguis, E., Devriendt, K., Meire, F., Standaert, L., Baten, E., & Van den Berghe, H. (1996). Cohen syndrome: The clinical symptoms and stigmata at a young age. *Clinical Genetics*, 49, 237–421.
- Fuhrmann-Rieger, A., Kohler, A., & Fuhrmann, W. (1984). Duplication or insertion in 15q11-13 associated with mental retardation, short stature and obesity, Prader-Willi or Cohen syndrome? *Clinical Genetics*, 25, 347–352.
- Goecke, T., Majewski, F., Kautner, K. D., & Sterzel, U. (1982). Mental retardation, hypotonia, obesity, ocular, facial, dental, and limb abnormalities (Cohen syndrome) report of three patients. *European Journal of Pediatrics*, 138, 338–340.
- Hennies, H. C., Rauch, A., & Seifert, W. (2004). Allelic heterogeneity in the COH1 gene explains clinical variability in Cohen syndrome. *American Journal of Human Genetics*, 75, 138–145.
- Howlin, P. (2001). Autistic features in Cohen syndrome: A preliminary report. *Developmental Medicine and Child Neurology*, 43, 692–696.
- Karpf, J., Turk, J., & Howlin, P. (2004). Cognitive, language and adaptive behaviour skills in individuals with a diagnosis of Cohen syndrome. *Clinical Genetics*, 65, 1–6.
- Katzaki, E., Pescucci, C., Uliana, V., Papa, F. T., Ariani, F., Meloni, I., Priolo, M., Selicorni, A., Milani, D., Fischetto, R., Celle, M. E., Grasso, R., Dallapiccola, B., Brancati, F., Bordinon, M., Tenconi, R., Federico, A., Mari, F., Renieri, A., & Longo, I. (2007). Clinical and molecular characterization of Italian patients affected by Cohen syndrome. *Journal of Human Genetics*, 52, 1011–1017.
- Kivitie-Kallio, S., & Norio, R. (2001). Cohen syndrome: Essential features, natural history, and heterogeneity. *American Journal of Medical Genetics*, 102, 125–135.
- Kivitie-Kallio, S., Eronen, M., Lipsanen-Nyman, M., Marttinen, E., & Norio, R. (1999). Cohen syndrome: Evaluation of its cardiac, endocrine and radiological features. *Clinical Genetics*, 56, 41–50.
- Kivitie-Kallio, S., Summanen, P., Raitta, C., & Norio, R. (2000). Ophthalmologic findings in Cohen syndrome. *Ophthalmology*, 107, 1737–1745.
- Kolehmainen, J., Norio, R., Kivitie-Kallio, S., Tahvanainen, E., de la Chapelle, A., & Lehesjoki, A. E. (1997). Refined mapping of the Cohen syndrome gene by linkage disequilibrium. *European Journal of Human Genetics*, 5, 206–213.
- Kolehmainen, J., Black, C. M., Saarinen, A., Chandler, K., Clayton-Smith, J., Träskelin, A. L., Kivitie-Kallio, S., Norio, R., Warburg, M., Fryns, J. P., de la Chapelle, A., & Lehesjoki, A. E. (2003). Cohen syndrome is caused by mutations in a novel gene, COH1, encoding a transmembrane protein with a presumed role in vesicle-mediated sorting and intracellular protein transport. *American Journal of Human Genetics*, 72, 1359–1369.
- Kolehmainen, J., Wilkinson, R., Lehesjoki, A. E., Chandler, K., Kivitie-Kallio, S., Clayton-Smith, J., Träskelin, A. L., Waris, L., Saarinen, A., Khan, J., Gross-Tsur, V., Traboulsi, E. I., Warburg, M., Fryns, J. P., Norio, R., Black, G. C., & Manson, F. D. (2004). Delineation of Cohen syndrome following a large-scale genotype-phenotype screen. *American Journal of Human Genetics*, 75, 122–127.
- Kondo, I., Nagataki, S., & Miyagi, N. (1990). The Cohen syndrome: Does mottled retina separate a Finnish and a Jewish type? *American Journal of Medical Genetics*, 37, 109–113.
- Kousseff, B. (1981). Cohen syndrome: Further delineation and inheritance. *American Journal of Medical Genetics*, 9, 25–30.
- Massa, G., Doms, L., & Vanderschueren-Lodeweyckx, M. (1991). Growth hormone deficiency in a girl with the Cohen syndrome. *Journal of Medical Genetics*, 28, 48–50.
- Mehes, K., Kosztolanyi, G., Kardos, M., & Horvath, M. (1988). Cohen syndrome: A connective tissue disorder? *American Journal of Medical Genetics*, 31, 131–133.
- Mendez, H. M. M., Paskulin, G. A., & Vallandro, C. (1985). The syndrome of retinal pigmentary degeneration, microcephaly and severe mental retardation (Mirhosseini-Holmes-Walton syndrome): Report of two patients. *American Journal of Medical Genetics*, 22, 223–228.
- Michaud, J. L., Héon, E., Guilbert, F., Weill, J., Puech, B., Benson, L., Smallhorn, J. F., Shuman, C. T., Buncic, J. R., Levin, A. V., Weksberg, R., & Brevière, G. M. (1996). Natural history of Alström syndrome in early childhood: Onset with dilated cardiomyopathy. *Journal of Pediatrics*, 128, 225–229.
- Mochida, G. H., Rajab, A., Eyaid, W., Lu, A., Al-Nouri, D., Kosaki, K., Noruzinia, M., Sadra, P., Ishihara, J., Bodell, A., Apse, K., & Walsh, C. A. (2004). Broader geographical spectrum of Cohen syndrome due to COH1 mutations. *Journal of Medical Genetics*, 41, 87.
- Nambu, M., Oshima, Y., Kakiuchi, T., Hayakawa, Y., Ito, T., Hasegawa, T., et al. (1988). Cohen's syndrome with diabetes mellitus. *Acta Paediatrica Japonica*, 30, 84–88.
- Norio, R., Raitta, C., & Lindahl, E. (1984). Further delineation of the Cohen syndrome: Report on chorioretinal

- dystrophy, leukopenia and consanguinity. *Clinical Genetics*, 25, 1–14.
- North, K. N., Fulton, A. B., & Whiteman, D. A. (1995). Identical twins with Cohen syndrome. *American Journal of Medical Genetics*, 58, 54–58.
- Ozturk, B., & Weber, H. P. (1991). Cohen-Syndrom Eigene Beobachtung und Literaturübersicht. *Monatschrift für Kinderheilkunde*, 139, 844–848.
- Pirgon, O., Atabek, M. E., & Sert, A. (2006). Metabolic syndrome manifestations in Cohen syndrome: Description of two new patients. *Journal of Child Neurology*, 21, 536–538.
- Sack, J., & Friedman, E. (1986). The Cohen syndrome in Israel. *Israel Journal of Medical Sciences*, 22, 766–770.
- Schlichtemeier, T. L., Tomlinson, G. E., Kamen, B. A., Waber, L. J., & Wilson, G. N. (1994). Multiple coagulation defects and the Cohen syndrome. *Clinical Genetics*, 45, 212–216.
- Seifert, W., Holder-Espinasse, M., Spranger, S., Hoeltzenbein, M., Rossier, E., Dollfus, H., Lacombe, D., Verloes, A., Chrzanoswska, K. H., Maegawa, G. H., Chitayat, D., Kotzot, D., Huhle, D., Meinecke, P., Albrecht, B., Mathijssen, I., Leheup, B., Raile, K., Hennies, H. C., & Horn, D. (2006). Mutational spectrum of COH1 and clinical heterogeneity in Cohen syndrome. *Journal of Medical Genetics*, 43, 22.
- Seifert, W., Holder-Espinasse, M., Kuhnisch, J., Kahrizi, K., Tzschach, A., Garshasbi, M., Najmabadi, H., Walter Kuss, A., Kress, W., Laureys, G., Loeys, B., Brilstra, E., Mancini, G. M., Dollfus, H., Dahan, K., Apse, K., Hennies, H. C., & Horn, D. (2009). Expanded mutational spectrum in Cohen syndrome, tissue expression, and transcript variants of COH1. *Human Mutation*, 30, 404–420.
- Sorva, R., Perheentupa, J., & Tolppanen, E. M. (1984). A novel format for a growth chart. *Acta Paediatrica Scandinavica*, 73, 527–529.
- Steinlein, O., Tariverdian, G., Boll, H. U., & Vogel, F. (1991). Tapetoretinal degeneration in brothers with apparent Cohen syndrome: Nosology with Mirhosseini-Holmes-Walton syndrome. *American Journal of Medical Genetics*, 41, 196–200.
- Summanen, P., Kivitie-Kallio, S., Norio, R., Raitta, C., & Kivela, T. (2002). Mechanisms of myopia in Cohen syndrome mapped to chromosome 8q22. *Investigative Ophthalmology & Visual Science*, 43, 686–693.
- Tahvanainen, E., Norio, R., Karila, E., Ranta, S., Weissbach, J., Sistonen, P., & de la Chapelle, A. (1994). Cohen syndrome gene assigned to the long arm of chromosome 8 by linkage analysis. *Nature Genetics*, 7, 201–204.
- Velayos-Baeza, A., Vettori, A., Copley, R. R., Dobson-Stone, C., & Monaco, A. P. (2004). Analysis of the human VPS13 gene family. *Genomics*, 84, 536–549.
- Warburg, M., Pedersen, S. A., & Horlyk, H. (1990). The Cohen syndrome. Retinal lesions and granulocytopenia. *Ophthalmic Paediatrics and Genetics*, 11, 7–13.

Cohen, Donald J.

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Yale Child Study Center, New Haven, CT, USA

Name and Degrees

Donald J. Cohen, M.D. (1940–2001).

Major Appointments (Institution, Location, Dates)

- Director, Yale Child Study Center, 1983–2001.
- President, International Association of Child and Adolescent Psychiatry and Allied Professions, 1992–1998.

Major Honors and Awards

- Doctorate Honoris Causa, Bar-Ilan University, 1997.
- Lifetime Achievement Award, International Meeting for Autism Research, 2001.

Landmark Clinical, Scientific, and Professional Contributions

Donald Cohen was the leading American child psychiatrist of his generation and helped advance the neurobiological study of autism, initially through his work in the 1970s and 1980s on serotonin and monoamines in cerebrospinal fluid and later by developing cross-disciplinary research collaborations at the Yale Child Study Center and beyond. Under his directorship, the research program on autism at Yale broke new ground, among other areas, in nosology (through the DSM field trials led by Fred Volkmar), phenotypic definition (through Ami Klin's eye tracking paradigms), and neural substrates (through Robert Schultz's imaging studies of the fusiform gyrus). In addition, Cohen brought to autism research his background in philosophy and psychoanalysis, as exemplified

in his collaborations on theory of mind with Simon Baron-Cohen, and on the contributions of psychoanalysis to social development with Linda Mayes.

Short Biography

Born in Chicago in 1940 to a humble family, Donald Cohen attended college at Brandeis University. He studied philosophy at Cambridge before enrolling in medical school at Yale. He trained in pediatrics at Children's Hospital Boston and in psychiatry at the Massachusetts Mental Health Center. During his time in Boston, he worked with Ogden Lindsay in his operant conditioning laboratory. During the Vietnam War, he worked in Washington, D.C., as special assistant to Edward Zigler, helping him in the development of the Head Start program. Cohen was recruited back to Yale in 1972 by Albert Solnit, whom he succeeded as director of the Yale Child Study Center in 1983. At Yale, Cohen was able to integrate his background in philosophy, psychoanalysis, and neuroscience particularly on two "model" disorders: Tourette's syndrome and autism. His early studies on monoamine metabolites in serum and CSF were conducted at the Children's Clinical Research Center (CCRC) at Yale-New Haven Children's Hospital. Cohen was codirector of the CCRC from the time of his arrival at Yale until his succession by his close collaborator James Leckman in 1983. Cohen's early studies on monoamine metabolites were followed by his development of a rich multidisciplinary research program for autism at the Yale Child Study Center. Key collaborators in this program were Fred Volkmar (who eventually would go on to lead it), Ami Klin, Robert Schultz, Rhea Paul, and George Anderson. In addition to a wide portfolio on neurobiological studies of autism, Cohen remained interested and made seminal contributions to theory of mind and the inner life of individuals with autism, as well as to the ethical imperative to conduct sound research in

vulnerable populations, including individuals affected with autism. Much of Cohen's later career was devoted to establishing international programs in child and adolescent psychiatry, a focus that he developed as president of the International Association of Child and Adolescent Psychiatry and Allied Professions.

References and Reading

Edited Books

- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. J. (Eds.). (1994). *Understanding other minds: Perspectives from autism* (1st ed.). New York: Oxford University Press.
- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. J. (Eds.). (2000). *Understanding other minds: Perspectives from autism* (2nd ed.). New York: Oxford University Press.
- Cohen, D. J., & Donnellan, A. M. (Eds.). (1987). *Handbook of autism and pervasive developmental disorders* (1st ed.). Chichester: Wiley.
- Cohen, D. J., & Volkmar, F. (Eds.). (1997). *Handbook of autism and pervasive developmental disorders* (2nd ed.). Chichester: Wiley.
- Volkmar, F. R., Paul, R., Klin, A., & Cohen, D. J. (2005). *Handbook of autism and pervasive developmental disorders* (Vol. 2, 3rd ed.). Chichester: Wiley.

Select Articles (In Reverse Chronological Order)

- Anderson, G. M., Freedman, D. X., Cohen, D. J., Volkmar, F. R., Hoder, E. L., McPhedran, P., et al. (1987). Whole blood serotonin in autistic and normal subjects. *Journal of Child Psychology and Psychiatry*, 28(6), 885–900.
- Anderson, G. M., Horne, W. C., Chatterjee, D., & Cohen, D. J. (1990). The hyperserotonemia of autism. *Annals of the New York Academy of Sciences*, 600, 331–340.
- Cohen, D. J. (2001). Into life: Autism, Tourette's syndrome and the community of clinical research. *Israel Journal of Psychiatry and Related Sciences*, 38(3–4), 226–234.
- Cohen, D. J., Caparulo, B. K., Shaywitz, B. A., & Bowers, M. B., Jr. (1977a). Dopamine and serotonin metabolism in neuropsychiatrically disturbed children. CSF homovanillic acid and 5-hydroxyindoleacetic acid. *Archives of General Psychiatry*, 34(5), 545–550.
- Cohen, D. J., Young, J. G., & Roth, J. A. (1977b). Platelet monoamine oxidase in early childhood autism. *Archives of General Psychiatry*, 34(5), 534–537.

- Klin, A., & Cohen, D. J. (1994). The immorality of not-knowing: The ethical imperative to conduct research in child and adolescent psychiatry. In J. Hattab (Ed.), *Ethics in child psychiatry* (pp. 1–17). Jerusalem: Gefen Publishing House.
- Klin, A., Sparrow, S. S., de Bildt, A., Cicchetti, D. V., Cohen, D. J., & Volkmar, F. R. (1999). A normed study of face recognition in autism and related disorders. *Journal of Autism and Developmental Disorders*, 29(6), 499–508.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002a). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809–816.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002b). Defining and quantifying the social phenotype in autism. *American Journal of Psychiatry*, 159(6), 895–908.
- Mayes, L. C., & Cohen, D. J. (1994). Experiencing self and others: Contributions from studies of autism to the psychoanalytic theory of social development. *Journal of the American Psychoanalytic Association*, 42(1), 191–218.
- Schultz, R. T., Gauthier, I., Klin, A., Fulbright, R. K., Anderson, A. W., Volkmar, F., et al. (2000). Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and Asperger syndrome. *Archives of General Psychiatry*, 57(4), 331–340.

Cohort Studies

- [Longitudinal Research in Autism](#)

Collaborative Consultation

- [Role Release](#)

Collaborative Intervention

- [Role Release](#)

Collaborative Program of Excellence in Autism

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Major Areas or Mission Statement

The mission of the CPEA network was to increase empirical knowledge about autism through the use of (1) multiple levels of interdisciplinary investigation into the genetics, neurobiology, and clinical aspects of autism; (2) rigorous phenotyping of research participants to support network-wide studies of large numbers of participants; and (3) a collaborative process of sharing data, findings, and ideas to move the science ahead as quickly as possible. The purpose of this network was to gain knowledge that could lead to prevention, treatment, and/or amelioration of the disabling effects of autism on people with autism and their families.

Landmark Contributions

The CPEA network was created as result of a congressionally mandated conference on the State of the Science in Autism, which took place in April 1995, to identify gaps in the knowledge of autism and directions for future research. The NICHD and the NIDCD joined together in 1997 to fund a 5-year project that consisted of nine clinical centers that each had a unique focus of autism research. The CPEA network was funded through an NIH Cooperative Agreement mechanism. Funding was secured in 2002 for a second

5-year cycle. The primary goal of this program was to bring together expertise, infrastructure, and resources focused on major questions about autism. The research issues addressed include advanced techniques of diagnosis and assessment, population genetics and molecular biology, structural and functional brain imaging, animal models, behavioral and cognitive neuroscience, and focused interventions, to elucidate the neurobiology of autism, with the long-term goal of effective diagnosis, treatment, and prevention. The CPEAs have linked scientists from the United States, Canada, Britain, and five other countries in the study of more than 2,200 families over 10 years. As a result, the CPEAs have data on the genetic and phenotypic characteristics of the world's largest group of well-diagnosed persons with autism. The funding of the network ended in 2007 when the NIH consolidated its funding for autism research into other programs.

Major Activities

The CPEA network conducted basic and clinical research on the possible genetic, immunological, neurobiological, and environmental causes of autism. The network also investigated the developmental course of autism and how specific brain structures were related to autism. These undertakings required that each CPEA implement a cohesive, site-specific, multidisciplinary research program on the causes, brain substrates, functional characteristics, and clinical development of autism. In addition, each site participated in a trans-network collaborative study for which no single site had sufficient expertise and/or participant sample size. Network projects evaluated the effectiveness of the hormone, secretin, in the treatment of autism (Owley et al. 2001; Unis et al. 2002); the candidate autism genes, *HOXA* and *Reelin* (Devlin et al. 2002, 2004); the onset of regression and measles-mumps-rubella vaccination (Richler et al. 2006); early regression in social communication in autism (Richler et al. 2006); the heterogeneous association between *Engrailed-2* and autism (Brune et al. 2008); familial autoimmune thyroid disease and regression in autism

(Molloy et al. 2006); frontal lobe function in people with autism (Ozonoff et al. 2004); the relationship between head circumference and autism (Lainhart et al. 2006); and IQ-based subtypes of autism (Munson et al. 2008).

References and Reading

- Brune, C. W., Korvatska, E., Allen-Brady, K., Cook, E. H., Jr., Dawson, G., Devlin, B., et al. (2008). Heterogeneous association between *enrailed-2* and autism in the CPEA network. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 147B(2), 187–193.
- Devlin, B., Bennett, P., Cook, E. H., Dawson, G., Jr., Gonen, D., Grigorenko, E. L., et al. (2002). No evidence for linkage of liability to autism to *HOXA1* in a sample from the CPEA network. *American Journal of Medical Genetics*, 114(6), 667–672.
- Devlin, B., Bennett, P., Dawson, G., Figlewicz, D. A., Grigorenko, E. L., McMahon, W., et al. (2004). Alleles of a *reelin* CGG repeat do not convey liability to autism in a sample from the CPEA network. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 126B(1), 46–50.
- Lainhart, J. E., Bigler, E. D., Bocian, M., Coon, H., Dinh, E., Dawson, G., et al. (2006). Head circumference and height in autism: A study by the collaborative program of excellence in autism. *American Journal of Medical Genetics. Part A*, 140A, 2257–2274.
- Luyster, R., Richler, J., Risi, S., Hsu, W. L., Dawson, G., Bernier, R., et al. (2005). Early regression in social communication in autism spectrum disorders: A CPEA study. *Developmental Neuropsychology*, 27(3), 311–316.
- Molloy, C. A., Morrow, A. L., Meinzen-Derr, J., Schleifer, K., Dienger, K., Manning-Courtney, P., et al. (2006). Elevated cytokine levels in children with autism spectrum disorder. *Journal of Neuroimmunology*, 172(1), 198–205.
- Munson, J., Dawson, G., Sterling, L., Beauchaine, T., Zhou, A., Elizabeth, K. E., et al. (2008). Evidence for latent classes of IQ in young children with autism spectrum disorder. *American Journal of Mental Retardation*, 113(6), 439–452.
- Owley, T., McMahon, W., Cook, E. H., Laulhere, T., South, M., Mays, L. Z., et al. (2001). Multisite, double-blind, placebo-controlled trial of porcine secretin in autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 40(11), 1293–1299.
- Ozonoff, S., Cook, I., Coon, H., Dawson, G., Joseph, R. M., Klin, A., et al. (2004). Performance on cambridge neuropsychological test automated battery subtests sensitive to frontal lobe function in people with autistic disorder: Evidence from the collaborative programs of excellence in autism network. *Journal of Autism and Developmental Disorders*, 34(2), 139–150.

- Richler, J., Luyster, R., Risi, S., Hsu, W. L., Dawson, G., Bernier, R., et al. (2006). Is there a “regressive phenotype” of autism spectrum disorder associated with the measles-mumps-rubella vaccine? A CPEA study. *Journal of Autism and Developmental Disorders*, 36(3), 299–316.
- Unis, A. S., Munson, J. A., Rogers, S. J., Goldson, E. D., Osterling, J., Gabriels, R., et al. (2002). A randomized, double-blind, placebo-controlled trial of porcine versus synthetic secretin for reducing symptoms of autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 41(11), 1315–1321.

Collaborative Teaming

► Role Release

Collaborative Teaming Model

► Interdisciplinary Team

Collateral Effects of Youth Disruptive Behavior Disorders on Mothers' Psychological Distress: Intellectual Disability, or Typical Development

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Definition

Our focus herein is on how disruptive behavior disorders in youth with intellectual disability and/or autism spectrum disorder impact their families. We further examine the mitigating effects of positive beliefs and dispositional optimism on parents' distress.

Historical Background

Impact of Child Developmental Disability on the Family

It has long been recognized that raising a child with a developmental disability, such as autism, impacts families. These impacts are usually framed as parenting challenges, though more recent researchers have noted positive impacts as well. While some disabilities are apparent at birth (e.g., Down syndrome), others become apparent as the child develops (e.g., mild delays, autism). Thus, four facets of developmental risk and families become obvious: The first is that the impact on families relates to the nature of the child's disorder. The second is that this impact is also related in some ways to the family's strengths and resources. The third is the extent of effective support services that are available to the family. A fourth, which we focus on in this article, is when the developmental disability is compounded by behavior problems and/or mental disorders, which are more likely in children and youth with developmental disabilities. Relatedly, these disorders themselves, especially when compounded by concomitant psychological problems, have collateral effects on parents.

The literature reviewed here speaks to youth with autism spectrum disorder as well as to those with intellectual disabilities, together considered developmental disorders. To a more limited extent, it speaks to impacts on fathers as well as mothers. This section briefly reviews the impacts on families of the child's disability itself, within a historical perspective. The next section addresses in more detail the collateral effects on families when the developmental delay is comorbid with behavior problems and/or mental disorders. The final section provides examples of protective factors for parent and family adjustment, with an illustrative focus on dispositional optimism.

Looking back: Historically, developmental disorders were not only viewed as tragedy, but also with a sense of hopelessness. Some writers spoke to devastating effects, as does the classic paper by Simon Olshansky (1962) who wrote of “chronic sorrow.” He proposed that the adjustment to a retarded child (the terminology of the era) is not

time-bound, but that over time repeated sadness, and even intense grieving, is periodically experienced. Indeed, the common advice of the times was to institutionalize the child, and state-run “training schools” across the USA had become lifelong residences for many.

Now almost six decades later: A number of developments have tempered the negative aspects of raising a child with a disability, in the views of professionals and the lived experience of parents. These changes in the USA, still progressing, have included (a) a marked decline in out-of-home care; (b) legislation mandating free and appropriate education for all handicapped children, codified in the 1975 Public Law 94–142 and more recently in 2004 as IDEA; (c) expanded early childhood programs; (d) considerably increased funding for related research and services; (e) innovations in evaluations and educational curricula; (f) more visibility of children and adults with disabilities in society; and (g) recent media attention to the marked increase in autism spectrum disorder (ASD). These, and other developments, along with more positive family perspectives, are accompanied by increasingly accepting societal views of persons with developmental disabilities.

While parents today certainly still experience the complex of emotions associated with raising a child with a developmental disability – and in many cases experience these sooner with earlier diagnoses – they also find wider community awareness and acceptance and earlier access to services. The developmental disability literature in the past two decades, while acknowledging the panoply of emotions and challenges parents still experience, is increasingly focused on gaining a better understanding of family needs and strengths.

Developmental disorders and effects on families: Over the past two decades, studies of families raising a child with a developmental disability have focused mainly on parenting stress and factors that reduce it. Patton et al. (2018), for example, studied carers for adolescents with ID vs. those with typical cognitive development (TD), finding that parents of youth with ID experienced significantly higher levels of stress.

Similarly, a meta-analysis analyzed parent-reported stress in families of children with autism spectrum disorder (ASD) vs. families with typically developing children (TD) (Hayes and Watson 2013). The authors found a large effect size indicating greater stress with ASD.

While emphases, understandably, have been on negative impacts of child disability on the family, and while perceived positive impacts do not negate these, it is important to report that many parents can readily find the “sunny side of the street” (Blacher et al. 2013a). Studies have examined positive impacts on the family of having a child with a disability and variables associated with positivity (e.g., Bayat 2007; Blacher and Baker 2007). One found that family resources, including higher income, time for interaction with the child, and social support, predicted lower parenting stress better than did aspects of the child’s functioning (Smith et al. 2001). Parents’ social support was also found to be a buffer, as carers with higher social support were no more stressed than carers for typically developing children. Parents’ well-being and positive relationships are also protective factors. Gerstein et al. (2009) examined parents’ resilience and the course of daily parenting stress across the preschool years for their child with intellectual disability (ID). Overall mothers’ daily parenting stress did increase some over time, while fathers’ stress remained more constant. For mothers with decreased stress, this was associated with both mother’s and father’s well-being and their perceived good marital adjustment, as well as a positive father-child relationship. Father’s stress trajectory was affected only by mothers’ well-being and both parents’ marital adjustment. A study of perceptions of positive impact examined 219 families annually from child ages 3 through 9, examining two predictors: child disability status (ID or TD) and family culture (Anglo, Latino) (Blacher et al. 2013b). Anglo mothers initially reported significantly lower positive impact when their child had an ID, but Latino mothers did not. Across all time points, Latino mothers reported higher positive impact than Anglo mothers, regardless of whether they had a child with ID or TD.

While parents today certainly still experience the complex of emotions associated with raising a child with a developmental disability — and in many cases experience these sooner with earlier diagnoses — they also find wider community awareness and acceptance and earlier access to services. The developmental disability literature in the past two decades, while acknowledging the panoply of emotions and challenges that parents still experience, is increasingly focused on gaining a better understanding of family needs and strengths. Studies are going beyond descriptions of family impacts, to examine child, parent, and family characteristics that promote acceptance and support.

Current Knowledge

Child Developmental Disability and Collateral Behavior Problems and/or Mental Disorder

Beyond the impact that children with disabilities have on their parents and families are two related observations: *First, youth with developmental disorders are significantly more likely than typically developing youth to have collateral behavior problems and/or diagnostically classified psychiatric disorders. Second, the combination of youth disabilities and diagnoses will have a heightened impact on their parents and family.* We will first examine the increased prevalence of behavior/mental disorders in youth with ID and ASD and then consider how children with both a developmental disability and also collateral behavior problems/mental disorder impact their families.

Heightened behavior/mental health disorders. Many studies, especially in the last two decades, have addressed comorbid behavioral and mental disorders. We will first focus on ASD. One study conducted a semi-structured interview with parents of school-aged children with ASD or control youth. There were significantly higher rates of ADHD, but also of anxiety disorder, in the youth with ASD (Rosa et al. 2016). This finding is consistent with many other studies (e.g., Leitner 2014; Simonoff et al. 2008). The true incidence may be even higher, as Leitner and colleagues (2014) pointed out that the widely used

Diagnostic and Statistical Manual (DSM-IV) had ASD diagnosis as an exclusion criterion for ADHD, thus limiting research on their common co-occurrence. This exclusion was eliminated in 2013, with the publication of DSM-V, so we might expect even greater comorbidity reported since then.

Focus on Autism: One frequent comorbidity is autism spectrum disorder (ASD) and the mental health diagnosis of attention deficit hyperactivity disorder (ADHD). This paired comorbidity has both high incidence and considerable impact on families. ADHD is defined by impaired functioning in the areas of attention, hyperactivity, and impulsivity, whereas ASD is characterized by core social communication deficits and restrictive-repetitive behaviors. Studies have shown that between 30 and 50% of children with ASD manifest ADHD symptoms (particularly at preschool age), and, similarly, estimates suggest two-thirds of individuals with ADHD show features of ASD (Davis and Kollins 2012).

Other studies of comorbid disorders in young children with ASD have found high rates of language problems, intellectual disability, below average motor function, and severe hyperactivity, ranging from 33% to 78% of this child group (Carlsson et al. 2013). In a major study from 14 sites in the USA and in Canada, Sikora et al. (2012) found that of 1737 young children diagnosed with ASD, 40% reached clinical levels on measures of ADHD.

While the link between ASD and ADHD has received the most research attention, we note that many other problems arise in children and youth with developmental disabilities, either comorbid with ADHD or separately, including anxiety, depression, behavior problems, fewer adaptive skills, and overall poorer quality of life. When a child with ID or ASD manifests any of these comorbid behavior and/or mental disorders, the impact on parents and families becomes more severe. One clear indication is that primary care providers for children with developmental disorders should screen for symptoms of ADHD, other psychiatric disorders, behavior problems, and adaptive skill deficits in these children and consider these broader co-occurring problems when developing a care plan.

Increased impact on families. It is, then, common that when a child has a developmental disorder, many – indeed most – families will encounter additional child-rearing challenges beyond those in the core definitions of intellectual disability and/or autism spectrum disorder. These “collateral effects” from behavior disorder/mental health challenges have negative impacts on the child and also on the family, beyond – *and even greater than* – the impact of the disability itself. *Indeed, evidence supports the conclusion that the behavior disorder/mental health problems greatly overshadow the disability in the challenge facing families.* This section, then, considers studies by researchers who examined youth with a type of dual diagnosis serious enough to put limits on the youth’s opportunities. Our key focus is on the extent to which parenting stress, parent psychological adjustment, and family functioning are impacted by rearing a child with a comorbid developmental disability and behavior/mental disorder.

Looking back: McIntyre et al. (2002) assessed collateral impacts on the family by looking back. They conducted lengthy interviews with the mothers of 103 young adults with severe ID. They assessed not only the young adult’s adaptive functioning, maladaptive behavior, and mental health problems but also the extent to which these had in the past and presently have negative impacts on the family. Behavior and/or mental health problems significantly increased mothers’ perceived negative impact of the YA on the family and predicted the family’s steps toward seeking out-of-home placement.

At the same time (2002) Baker et al., at three universities, examined 225 3-year-old children with developmental delays or typical cognitive development. Parents completed the *Child Behavior Checklist* (CBCL; Achenbach and Edelbrock 2002), a measure of child behavior problems that yields diagnostic categories. They also completed a self-report measure of parenting stress called the *Family Impact Questionnaire* (Donenberg and Baker 1993). Total behavior problems scores within the designated clinical range were three (mothers’ scores) to four (fathers’ scores) times as likely for children with developmental delays.

Collateral effects of disability and behavior disorders were already evident at this young age, as parenting stress scores were significantly higher in families of the children with both developmental delays and clinical level behavior problems. Regression analyses revealed that the extent of child behavior problems was a much stronger contributor to parenting stress than was the child’s cognitive delay, a surprising finding that now has been replicated in many other studies (e.g., McStay et al. 2013).

Other investigators have underscored, and expanded upon, the adverse effects on parents and families from the frequent collateral effects of a child’s developmental disability and accompanying internalizing (e.g., anxiety) or externalizing (e.g., attention or conduct problems) disorders. To cite a few: Yorke et al. (2018) disaggregated child externalizing and internalizing problems and showed that parents’ mental health problems and parenting stress were not solely driven by either, but, rather, that both are causal factors. Carlsson et al. (2013) expanded the scope of outcome from behavior/mental health disorders in young children with ASD, reporting heightened parenting stress from other domains, including comorbid language problems, below average motor function, and severe hyperactivity. Other researchers have found decreased physical health in mothers parenting a child with comorbid disorders (Benson 2017).

In sum, parents and families raising a child with a developmental disorder often face the dual challenge of also coping with comorbid behavior problems/mental health disorder and related effects. As we have noted, the comorbid behavior/mental health conditions often present more difficulties for the child and the family than the developmental disorder. We have spoken mainly of parenting stress, but there are also wide-ranging daily challenges that limit social, educational, employment, and other opportunities for the child, siblings, and parents. Fortunately, there are today many available services that provide support, respite, guidance, and more. What is most encouraging is that so many families have found ways to accept the child’s disabilities and integrate him or her into family life. Our next

section will look at positive parent and family characteristics that normalize and embrace disability.

Dispositional Optimism and Positive Beliefs: Buffering the Impact of Child Developmental Disability and Comorbid Behavior/Mental Health Challenges on Parents and Family Well-Being

Optimists have a favorable outlook on life: they believe that good rather than bad things will happen to them (Olason and Roger 2001).

Over 35 years ago, Crnic et al. (1983) proposed a model for the study of families that presented a more positive view of family outcome, to balance the more negative perspectives so predominant in the literature at that time. Parents have within themselves many ways of coping with child-rearing challenges. We will focus here on parent cognitions, especially dispositional optimism and positive beliefs, as they moderate the collateral effects of child developmental disorder when combined with behavioral/mental health challenges.

We consider here beliefs that will ease child-rearing challenges, drawing primarily on studies of children with dual diagnoses of developmental disability and behavior/mental health disorders (Blacher et al. 2013a). One important finding to remember has been that when causes of increased parenting stress have been parsed into (a) the child's developmental disability itself or (b) the comorbid child behavior problems/mental disorders, the latter accounted for most of the variance (Baker et al. 2002).

Dispositional optimism. Scheier and Carver (1985) proposed dispositional optimism as a relatively stable personality trait that leads to more positive coping with adversity or a "general positive expectation regarding future events regardless of one's control over the outcome" (Scheier et al. 1994). Optimists have a favorable outlook on life: they believe that good rather than bad things will happen to them. Our use of "optimistic" in what follows is referring to dispositional optimism. To maintain such positive beliefs, optimists rely on positive evaluation of the social context and its ability to provide necessary support. Dispositional optimism leads parents to have

an attentional bias toward their children's positive behaviors (Segerstrom 2001).

While dispositional optimism is a general positive expectation about future events, self-mastery is the belief in one's control over specific events. Paczkowski and Baker (2008) examined the role of self-mastery and then examined self-mastery and dispositional optimism together ("positive beliefs"). One's perception of control over life events and one's optimism about the future may be accurate reflections of the true degree of control one has or of the actual likelihood of future positive events. However, many people display unrealistic beliefs about the degree to which they control events in their environments. Regardless, optimism, no matter how unwarranted the positive outlook is, will lead to better coping with child challenges.

Measuring dispositional optimism. The widely used research measure of optimism is the self-report *Life Orientation Test - Revised* (LOT-R), a 6-item self-report scale with four additional filler items (Scheier et al. 1994). Sample items are "In uncertain times, I usually expect the best" and "If something can go wrong for me, it will" (reverse coded). Respondents indicate the extent of agreement on a 5-point Likert scale from 0 (Definitely disagree) to 4 (Definitely agree). The LOT-R has high internal reliability. In one study, parent optimism scores at child ages 3, 6, and 9 years were quite stable across these assessments. In that study, mean optimism scores for parents of children with ID or TD did not differ significantly (Blacher et al. 2013b).

Dispositional optimism further explored. This personality trait of optimism, which is present early in life, is associated with more problem-focused coping, such as seeking social support, actively solving problems, and maintaining a positive outlook on life. The relationship between dispositional optimism and more positive outcomes has been supported by considerable research. In one study higher optimism related to increased positive affect, greater life satisfaction, and psychological well-being (Ekas et al. 2010). In another, optimistic parents of children with a developmental disorder were more likely to engage in problem-solving and seeking social

support (Khan and Alam 2016). In other studies, more optimistic parents were effectively modulating their anger and patience, managing their children more appropriately, and developing more effective coping strategies (Koenig et al. 2010). In short, dispositional optimism and positive beliefs are protective factors for mothers' and fathers' parenting and well-being.

Dispositional optimism, then, moderates the effect of child challenges on parents' psychological distress. As child challenges increase, optimism has a progressively greater benefit. Many studies have found higher dispositional optimism to be related to increased positive and decreased negative parenting. One found that inner-city mothers with higher optimism had more positive parenting (Jones et al. 2002). Ellingsen et al. (2013, 2014) examined parent variables that predicted changes in resilient parenting from ages 3 to 5 and 5 to 8 years. Mothers' education and optimism were protective factors for positive parenting from child ages 3 to 5 and for resilient parenting from child age 5 to 8. Taylor (2010) more generally showed that optimism helps parents maintain positive parenting during adverse times.

Positive beliefs. Related to optimism are parents' positive beliefs about the child and child-rearing. Hastings and Taunt (2002) found that parents spoke to benefits they derived from raising a child with ID or ASD; these included having increased sensitivity and tolerance, a changed perspective on life, improved family dynamics, and opportunities to learn new information. Beighton and Wills (2019) reviewed 22 studies across many countries, reporting how parents described the positive aspects of parenting their child with ID. They concluded that caregivers reporting positive aspects had better self-reported health, fewer depressive symptoms, higher caregiving competence, and greater family adjustment. Fully 75% of mothers and 67% of fathers reported experiencing personal/emotional growth and increased benefit finding. Greater social support led to more positive parent perceptions.

Research published over the past two decades has underscored the power of positivity, whether in the form of positive perceptions, positive

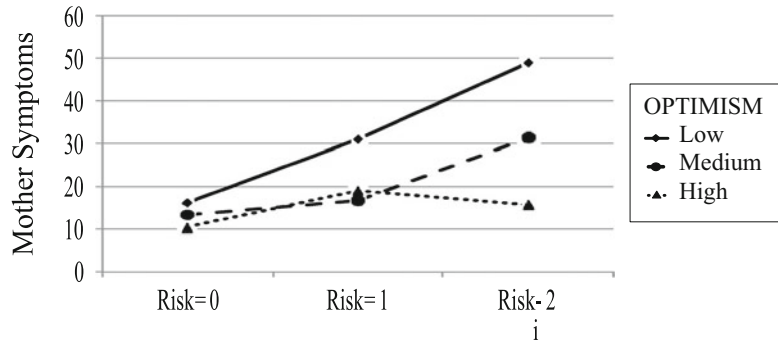
coping, or optimism. To maintain such positive beliefs, optimists rely on a sense of positive evaluation of the social context and its ability to provide necessary support. Optimists are more likely to engage in problem-solving and seeking social support. They effectively modulate their anger and impatience, as well as manage their children more appropriately. Problem-focused coping which emphasizes efforts to seek social support, actively solve the problem, and maintain a positive outlook on life is most adaptive.

Positive impact/positive perceptions. Positive impact can be assessed by a subscale of the *Family Impact Questionnaire* (FIQ; Donenberg and Baker 1993). This 7-item scale is introduced by "We would like to know what impact your child has had on your family compared to the impact other children his/her age have on their families." Sample items are: *I enjoy the time I spend with my child more*, and *My child makes me feel more energetic*. Perceived positive impact was assessed with parents of 92 children with developmental delay (DD) and 122 TD children at ages 3, 4, and 5 years (Blacher and Baker 2007). Even with this large sample, there were no significant differences between mothers or fathers of DD and TD children. However positive perceptions did significantly lessen the relationship between child behavior problems and parenting stress.

We close with an illustration of how mothers' psychological symptoms (e.g., stress, depression) are increased with increasing child risk, but moderated by mothers' optimism (Blacher and Baker 2017) (Fig. 1). Families were assigned to one of three child categories of increasing risk to parent well-being: The child had neither disability nor disorder (Risk 0); had disability *or* disorder (Risk 1); or disability *and* disorder (Risk 2). Mothers' optimism scores were divided into thirds (high, medium, low optimism). Mother's self-reported psychological symptoms were derived from the Symptom Checklist (Derogatis and Savitz 2000). When child risk was low (0), mother's psychological symptoms were also low, and optimism did not matter. As child risk increased, mothers' psychological symptoms also increased. This was most dramatic for mothers with low optimism; mothers with high optimism demonstrated very

Collateral Effects of Youth Disruptive Behavior Disorders on Mothers' Psychological Distress: Intellectual Disability, or Typical Development,

Fig. 1 Mother psychological symptoms by youth risk and maternal optimism



Risk $F=12.99$, $p < .001$ Optimism $F=10.74$, $p < .001$; Risk X Optimism $F=2.63$, $p < .05$

few psychological symptoms. Statistically, main effects of risk and optimism were highly significant, and the risk x optimism interaction was also significant.

Risk $F = 12.99$, $p < 0.001$ Optimism $F = 10.74$, $p < 0.001$; Risk X Optimism $F = 2.63$, $p < 0.05$.

Future Directions

To date, the dominant theme in the literature on family impact of ASD, with or without comorbid ID, has been stress and overall negative effects. Clearly, children with ASD are at great risk of concomitant psychological disorders. Indeed, it is rare to find autism in the absence of other comorbidities (Gillberg and Fernell 2014). Happily, though, there is a trend of work focusing on more positive aspects of rearing a child with behavioral challenges, and these findings pertain to parents of children, youth, and young adults with ASD and comorbid disorders. We are hopeful that the empirical findings reported here will be translated into interventions for parents, in order to foster more adaptive outcomes.

References and Reading

- Achenbach, T. M., & Rescorla, L. A. (2000). Manual for the ASEBA Preschool Forms & Profiles. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families.
- Baker, B. L., & Blacher, J. (2015). Disruptive behavior disorders in adolescents with ASD: Comparisons to youth with intellectual disability or typical cognitive development. *Journal of Mental Health Research in Intellectual Disabilities*, 8(2), 98–116. <https://doi.org/10.1080/19315864.2015.1018395>.
- Baker, B. L., Blacher, J., Cmic, K. A., & Edelbrock, C. (2002). Behavior problems and parenting stress in families of three-year-old children with and without developmental delays. *American Journal on Mental Retardation*, 107(6), 433–444. [https://doi.org/10.1352/0895-8017\(2002\)107<0433:BPAPSI>2.0.CO;2](https://doi.org/10.1352/0895-8017(2002)107<0433:BPAPSI>2.0.CO;2).
- Bayat, M. (2007). Evidence of resilience in families of children with autism. *Journal of Intellectual Disability Research*, 51, 702–714. <https://doi.org/10.1111/j.1365-2788.2007.00960.x>.
- Beighton, C., & Wills, J. (2019). How parents describe the positive aspects of parenting their child who has intellectual disabilities: A systematic review and narrative synthesis. *Journal of Applied Research in Intellectual Disability*, 32, 1255–1279. <https://doi.org/10.1111/jar.12617>.
- Benson, P. R. (2017). The impact of child and family stressors on the self-rated health of mothers of children with autism spectrum disorder: Associations with depressed mood over a 12-year period. *Journal of Autism and Developmental Disorders*, 48(4), 1031–1040. <https://doi.org/10.1177/1362361317697656>.
- Blacher, J., & Baker, B. L. (2007). Positive impact of intellectual disabilities on families. *American Journal on Mental Retardation*, 112, 330–348. [https://doi.org/10.1352/0895-8017\(2007\)112\[0330:PIOIDO\]2.0.CO;2](https://doi.org/10.1352/0895-8017(2007)112[0330:PIOIDO]2.0.CO;2).
- Blacher, J., & Baker, B. L. (2017). Collateral effects of youth disruptive behavior disorders on mothers' psychological distress: Adolescents with autism spectrum disorder, intellectual disability, or typical development. *Journal of Autism and Developmental Disorders*, 43(2), 1–12. <https://doi.org/10.1007/s10803-017-3347-2>.
- Blacher, J., Baker, B. L., & Berkovits, L. D. (2013a). Family perspectives on child intellectual disability: Views from the sunny side of the street. In M. L. Wehmeyer (Ed.), *Handbook of positive psychology* (pp. 166–181). New York: Oxford University Press. <https://doi.org/10.1093/oxfordhb/9780195398786.013.013.0013>.

- Blacher, J., Begum, G. F., Marcoulides, G. A., & Baker, B. L. (2013b). Longitudinal perspectives of child positive impact on families: Relationship to disability and culture. *American Journal on Intellectual and Developmental Disabilities*, 118(2), 141–155. <https://doi.org/10.1352/1944-7558-118.2.141>.
- Carlsson, L. H., Norrelgen, F., & Fernell, E. (2013). Coexisting disorders and problems in preschool children with autism spectrum disorders. *Scientific World Journal*, 213979.
- Crníc, K. A., Friedrich, W. N., & Greenberg, M. T. (1983). Adaptation of families with mentally retarded children: A model of stress, coping, and family ecology. *American Journal of Mental Deficiency*, 88(2), 125–138.
- Davis, N. O., & Kollins, S. H. (2012). Treatment for co-occurring attention deficit/hyperactivity disorder and autism spectrum disorder. *Neurotherapeutics*, 9, 518–530.
- Dekker, M. C., & Koot, H. M. (2003). DSM-IV disorders in children with borderline to moderate intellectual disability: I: Prevalence and impact. *Journal of the American Academy of Child and Adolescent Psychiatry*, 42(8), 915–922. <https://doi.org/10.1097/01.CHI.0000046892.27264.1A>.
- Derogatis, L. R., & Savitz, K. L. (2000). The SCL-90-R and the Brief Symptom Inventory (BSI) in primary care. In M. E. Maruish (Ed.), *Handbook of psychological assessment in primary care settings* (pp. 297–334). Mahwah: Lawrence Erlbaum Associates.
- Donenberg, G., & Baker, B. L. (1993). The impact of young children with externalizing behaviors on their families. *Journal of Abnormal Child Psychology*, 21(2), 179–198. <https://doi.org/10.1007/BF00911315>.
- Ekas, N. V., Lickenbrock, D. M., & Whitman, T. L. (2010). Optimism, social support, and well-being in mothers of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 40, 1274–1284.
- Ekas, N. V., Timmons, L., Pruitt, M., Ghilain, C., & Alessandi, M. (2015). The power of positivity: Predictors of relationship satisfaction for parents of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45, 1997–2007. <https://doi.org/10.1007/s10803-015-2362-4>.
- Ellingsen, R., Baker, B. L., Blacher, J., & Crnic, K. (2013). Resilient parenting of preschool children at developmental risk. *Journal of Intellectual Disability Research*, 58(7), 664–678. <https://doi.org/10.1111/jir.12063>.
- Ellingsen, R., Baker, B. L., Blacher, J., & Crnic, K. (2014). Resilient parenting of children at developmental risk across middle childhood. *Research in Developmental Disabilities*, 35, 1364–1374. <https://doi.org/10.1111/jir.12063>.
- Gerstein, E. D., Crnic, K. A., Blacher, J., & Baker, B. L. (2009). Resilience and the course of daily parenting stress in families of young children with intellectual disabilities. *Journal of Intellectual Disability Research*, 53(12), 981–997. <https://doi.org/10.1111/j.1365-2788.2009.01220.x>.
- Gillberg, C., & Fernell, E. (2014). Autism plus versus autism pure. *Journal of Autism and Developmental Disorders*, 44, 3274–3276. <https://doi.org/10.1007/s10803-014-2163-1>.
- Hastings, R. P., & Taunt, H. M. (2002). Positive perceptions in families of children with developmental disabilities. *American Journal on Mental Retardation*, 107(2), 116–127. [https://doi.org/10.1352/0895-8017\(2002\)107<0116:PPIFOC>2.0.CO;2](https://doi.org/10.1352/0895-8017(2002)107<0116:PPIFOC>2.0.CO;2).
- Hayes, S. A., & Watson, S. L. (2013). The impact of parenting stress: A meta-analysis of studies comparing the experience of parenting stress in parents of children with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43, 629–642. <https://doi.org/10.1007/s10803-012-1604-y>.
- Jones, D. J., Forehand, R., Brody, G. H., & Armistead, L. (2002). Positive parenting and child psychosocial adjustment in inner-city single-parent African American families: The role of maternal optimism. *Behavior Modification*, 26(4), 464–481. <https://doi.org/10.1177/0145445502026004002>.
- Khan, M. F., & Alam, M. A. (2016). Coping trends of parents having children with developmental disabilities: A literature review. *European Journal of Special Education Research*, 1(3), 39–48. <https://oapub.org/edu/index.php/ejse/article/view/362>.
- Koenig, J. L., Barry, R. A., & Kochanska, G. (2010). Rearing difficult children: Parents' personality and children's proneness to anger as predictors of future parenting. *Parenting Science and Practice*, 10(4), 258–273. <https://doi.org/10.1080/15295192.2010.492038>.
- Leitner, Y. (2014). The co-occurrence of autism and attention deficit hyperactivity disorder in children – What do we know? *Frontiers in Human Neuroscience*, 8, 1–8. <https://doi.org/10.3389/fnhum.2014.00268>.
- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., Tager-Flusberg, H., & Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36, 849–861. <https://doi.org/10.1007/s10803-006-0123-0>.
- Marquis, W., & Baker, B. L. (2014). An examination of Anglo and Latino parenting practices: Relation to behavior problems in children with or without developmental delay. *Research in Developmental Disabilities*, 35, 383–392. <https://doi.org/10.1016/j.ridd.2013.11.010>.
- McIntyre, L. L., Blacher, J., & Baker, B. L. (2002). Behaviour/mental health problems in young adults with intellectual disability: The impact on families. *Journal of Intellectual Disability Research*, 46(3), 239–249. <https://doi.org/10.1046/j.1365-2788.2002.00371.x>.
- McStay, R. L., Dissanayake, C., Scheeren, A., Koot, H. M., & Begeer, S. (2013). Parenting stress and autism: The role of age, autism severity, quality of life and problem behaviour of children and adolescents with autism. *Autism*, 18(5) 502–510.
- Neece, C. L., & Baker, B. L. (2008). Predicting maternal parenting stress in middle childhood: The roles of child intellectual status, behaviour problems, and social skills. *Journal of Intellectual Disability*

- Research*, 52, 1114–1128. <https://doi.org/10.1111/j.1365-2788.2008.01071.x>.
- Olason, D., & Roger, D. (2001). Optimism, pessimism and “fighting spirit”: A new approach to assessing expectancy and adaptation. *Personality and Individual Differences*, 31(5), 755–768. [https://doi.org/10.1016/S0191-8869\(00\)00176-8](https://doi.org/10.1016/S0191-8869(00)00176-8).
- Olshansky, S. (1962). Chronic sorrow: A response to having a mentally defective child. *Families in society. Social Casework*, 43, 190–193. <https://doi.org/10.1177/104438946204300404>.
- Paczkowski, E., & Baker, B. L. (2008). Parenting children with developmental delays: The role of positive beliefs. *Journal of Mental Health Research in Developmental Disabilities*, 1, 156–175. <https://doi.org/10.1080/19315860801988392>.
- Patton, K. A., Ware, R., McPherson, L., Emerson, E., & Lennox, N. (2018). Parent-related stress of male and female carers of adolescents with intellectual disabilities and carers of children within the general population: A cross-sectional comparison. *Journal of Applied Research in Intellectual Disabilities*, 31, 51–61. <https://doi.org/10.1111/jar.12292>.
- Reichman, N. E., Corman, H., & Noonan, K. (2008). Impact of child disability on the family. *Journal of Maternal and Child Health*, 12, 679–683. <https://doi.org/10.1007/s10995-007-0307-z>.
- Rosa, M., Puig, O., & Azaro, L. (2016). Socioeconomic status and intelligence quotient as predictors of psychiatric disorders in children and adolescents with high-functioning autism spectrum disorder and in their siblings. *Autism*, 8, 963–972. <https://doi.org/10.1177/1362361315617881>.
- Scheier, M. F., & Carver, C. S. (1985). Optimism, coping, and health: Assessment and implications of generalized outcome expectancies. *Health Psychology*, 4(3), 219–247. <https://doi.org/10.1037/0278-6133.4.3.219>.
- Scheier, M. F., Carver, C. S., & Bridges, M. W. (1994). Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): A reevaluation of the life orientation test. *Journal of Personality and Social Psychology*, 67(6), 1063–1076.
- Segerstrom, S. C. (2001). Optimism and attentional bias for negative and positive stimuli. *Personality and Social Psychology Bulletin*, 7(10), 1334–1343. <https://doi.org/10.1177/01461672012710009>.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(8), 921–929. <https://doi.org/10.1097/CHI.0b013e318179964f>.
- Smith, T. B., Oliver, M. N. I., & Innocenti, M. S. (2001). Parenting stress in families with children with disabilities. *American Journal of Orthopsychiatry*, 71, 257–261. <https://doi.org/10.1037/0002-9432.71.2.257>.
- Taylor, S. E. (2010). Mechanisms linking early life stress to adult health outcomes. *Proceedings of the National Academy of Sciences USA*, 107, 8507–8512. <https://doi.org/10.1073/pnas.1003890107>.
- Trute, B., & Hiebert-Murphy, D. (2002). Family adjustment to childhood developmental disability: A measure of parent appraisal of family impacts. *Journal of Pediatric Psychology*, 27, 271–280. <https://doi.org/10.1093/jpepsy/27.3.271>.
- Wikler, L., Wasow, M., & Hatfield, E. (1981). Chronic sorrow revisited: Parent vs. professional depiction of the adjustment of parents of mentally retarded children. *American Journal of Orthopsychiatry*, 51(1), 63–70. <https://doi.org/10.1111/j.1939-0025.1981.tb01348.x>.
- Yorke, I., White, P., Weston, A., Rafla, M., Charman, T., & Simonoff, E. (2018). The association between emotional and behavioral problems in children with autism spectrum disorder and psychological distress in their parents: A systematic review and meta-analysis. *Journal of Autism and Developmental Disorders*, 48(10), 3393–3415. <https://doi.org/10.1007/s10803-018-3605-y>.

College Transitional Programs

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Definition

College Transition Programs are specialized programs dedicated to helping young adults with ASD to have successful college experiences. The number of these programs has dramatically increased over recent years, and there are programs located across the United States.

Historical Background

The population of individuals with ASD has risen dramatically in the past three decades, from one in 10,000 individuals in the 1980s (Autism Science Foundation 2012) to 1 in 68 children in 2016, according to estimates from the CDC (2016). There has also been a shift in the characteristics of this population. In the years between 1966 and 1998, only about 20% of individuals with an autism diagnosis were found to have IQs in the average range (Fombonne 1999). As of 2010, nearly half of individuals with autism had IQs in

the average range (CDC 2014). In addition to this shift in cognitive ability of those with ASD diagnoses, more effective and widely available early intervention programs have emerged, leading to better outcomes among this group.

The increase in overall numbers of individuals diagnosed with ASD, coupled with this shift in IQ, has vastly increased the number of individuals on the autism spectrum who have the cognitive capacity for college-level academics. As identification of ASD among individuals with no intellectual disability has improved, it is likely that the numbers of college students with ASD will continue to rise (Barnhill 2014). These factors have all impacted the trajectory of the lives of individuals with ASD, leading to a higher level of participation in college environments.

A longitudinal study completed in 2007 revealed that individuals who were diagnosed with an autism spectrum disorder in childhood generally retained this diagnosis into adulthood (Cederlund et al. 2008). Adult outcomes for individuals with ASD have historically been troublesome. Of those individuals with an ASD diagnosis and cognitive abilities in the average range, only 27% achieved what would be described as a “good outcome” in adulthood (Cederlund et al. 2008). Due to the extremely small numbers of individuals with ASD who attended colleges and universities in the twentieth century, little to no historical data about college success exists.

Students with ASD who attended college in previous decades were generally supported through the department/personnel who supported students with any disability, and at many colleges and universities, this remains the case. Legislation such as the Americans with Disabilities Act (ADA 1990) and Section 504 of the Rehabilitation Act of 1973 mandated equal access for individuals with disabilities, as well as provision of certain accommodations. Passage of these laws resulted in an increase in the ability of individuals with a range of disabilities to access higher education. However, as the number of students with ASD who were both interested in and capable of completing a college education rose, programs emerged with their needs in mind. The first university-based program was the college program for students

with Asperger syndrome at Marshall University in Huntington, West Virginia. This program, founded in 2002, was initially developed by the West Virginia Autism Training Center located at Marshall to meet the needs of a single student (Ellison, M., personal communication, 1/11/2017). Due to a high level of demand, it rapidly expanded and remains a pioneering program in the field today.

Current Knowledge

Approximately 50,000 individuals with autism turn 18 each year (Roux et al. 2013), and that number continues to rise. Engagement in productive activities after high school remains a challenge for individuals with ASD. A study conducted in 2012 by Shattuck et al. revealed that 34.7% of individuals with ASD had attended college within 6 years after high school. More than 50% of the young adults with ASD had no participation whatsoever in employment or post-secondary education within the first 2 years after high school. College success for students with ASD has remained a challenge. Even among those students who participate in higher education, high levels of academic achievement and meeting graduation requirements has often been elusive. At the conclusion of Shattuck et al.'s study (2012), less than 20% of students who went directly from high school to a 4-year college had graduated or were on track to graduate.

At the time of this publication, there were at least 31 college transition programs for individuals with ASD known to be in existence in the United States (Barnhill 2016), with that number increasing each year. There are a range of types of programs and supports that are offered. There are some programs that provide individuals with ASD an opportunity to participate in program offerings based on a college campus, outside of the standard coursework that the college offers. Other programs provide supports to individuals as they participate in the standard coursework. Of these programs offering support to take credit-bearing coursework, there are some, such as the program at Marshall, that are operated by the hosting

university or a related entity. Other programs, such as Chapel Haven in New Haven, CT, and Tucson, AZ, are operated by nonprofit agencies and have supports available at multiple colleges and universities. And still others are for-profit corporations offering services at various locations around the United States.

There are varying levels of supports offered at these various programs as well. Some programs offer supports which focus predominantly on academic success, others also include a focus on peer relationships and social skill development, and a few provide a broad range of supports including academic, social, daily living skills, and career development. Most of these programs have oversight provided by a professional experienced in working with students with ASD. Many also have a range of professional staff, while others utilize graduate students from the university, or undergraduate students, often in peer mentor roles.

College transition programs often provide tutoring, organizational support, and/or support accessing on-campus academic resources to help students with ASD succeed in their classes. Some programs also offer supports such as social skills instruction, counseling, or behavioral consultation. Many offer assistance accessing activities with peers, including clubs, sports activities, and residential life activities. A few of the most comprehensive programs also offer supports within the individual's residential environment, whether that be in a college residence hall or elsewhere.

A recent study identified 31 institutions of higher education that provided support services for individuals with ASD beyond those typically offered by college or university disability offices (Barnhill 2016). Of 30 institutions that shared information about their practices for this study, the vast majority provided academic accommodations (e.g., extended time, tutoring, notetakers, etc.) as well as an assigned advisor. More than two thirds of these programs also provided peer mentoring and accommodations in the areas of life skills and social skills. The provision of support services was more variable. Approximately 50% provided social skills groups and 43% provided individual therapy, while just 17% used a

group therapy model and only 13% utilized non-therapeutic groups (Barnhill 2016).

Specifically tailored interventions can allow students with ASD to succeed in the college environment. It is imperative that students and families explore all available options in order to assess the “goodness of fit” of postsecondary programs (VanBergeijk et al. 2008). Finding a program that offers the necessary supports for an individual and also provides access to a college or university that meets the individual's academic needs can be daunting. Families and individuals with ASD may begin the process of searching for the right program significantly in advance of the individual reaching college age. The assistance of educational consultants and other professionals is sometimes sought. The West Virginia Autism Training Center offers a document entitled “Benchmarks of Effective Supports for College Students with Autism Spectrum Disorders” on their website. This document assists students and families in evaluating college transition programs supports as related to the needs of an individual student (Ellison et al. 2012).

Students participating in structured college transition programs have access to substantially more supports than individuals attending college without the assistance of such a program. Even with these supports in place, many individuals on the autism spectrum continue to face some challenges in the postsecondary environment. Challenges faced by college students with ASD include managing new situations and unexpected changes, social relationships, problems with information processing, and time management (Van Hees et al. 2015). They juggle challenges in both college life and basic independent living skills.

An additional consideration when students are exploring college transition programs is cost. A small number of programs are able to provide services at no additional cost to students, outside of college tuition, but the vast majority of support programs for students with ASD require additional tuition and/or fees, which can be significant and pose a barrier for many students and families. These fees may be as low as a few hundred dollars per semester, or up to tens of thousands of dollars

per semester, depending upon the types of supports offered.

The existence of college transition programs offer a path to postsecondary success that many students with ASD may not have otherwise. Selecting the right program, with an appropriate array of supports for an individual student can be life-changing. Anecdotal information and observations suggest that these programs lead to higher levels of college success for individuals with ASD, but thus far, there has been virtually no research done to investigate the success of these programs or of the specific components within these programs. Some programs, including Chapel Haven, have embarked on a path to collect data in order to empirically demonstrate outcomes of their transition programming.

Future Directions

As the rising numbers of individuals with ASD complete high school and begin to enter postsecondary educational institutions, the supports available to them will need to continue to expand to meet this increased need. There will likely be a broadening of supports to meet the unique needs of this population. In a recent survey, individuals with ASD identified that programs facilitating their success should have a personalized approach, a safe/transparent environment, academic accommodations, adequate psychosocial support, leisure activities, and a sufficient amount of rest. They also identified that coaching in education, student life, and daily living should be integral to their supports (Van Hees et al. 2015).

A large majority (79%) of individuals with autism spectrum disorders meet the criteria for a psychiatric disorder at some point in their adult life. These symptoms are most prevalent during the young adult years (Lever and Geurts 2016). These challenges need to be addressed simultaneously to academic and independent living challenges so that the individual may be successful in the college environment. It will be important for college transition programs in the future to continue to provide and/or expand access to these supports to facilitate student success. We have

little detailed empirical data about psychiatric challenges among this population however, so there is a need to continue to investigate to learn more about these challenges can be addressed (Howlin 2014; van Schalkwyk et al. 2016).

There is a dire need for more data and research regarding the types of supports that lead to success in the transition to college for individuals with ASD. As the need for supports have become apparent, many programs have emerged, but information on their effectiveness is scarce. Due to the lack of data on adult interventions in general, most of these programs have needed to rely on information about best practices for children in the development of their programs. Although there is often anecdotal information about what is and is not working for individual participants, there is a dearth of information regarding best practices in transition programming for college students with ASD. In a 2014 systematic review, Gelbar, Smith, and Reichow identified 20 articles, describing a total of 69 college students with ASD, only two of which were empirical studies (Gelbar et al. 2014).

It would be beneficial for the funding sources for college transition programs to be further explored and expanded, in order to allow a greater number of individuals to access these services. There is a significant disparity in college participation for young adults with ASD based upon family income. In a recent study, of those whose parents fell into the lowest identified income bracket, only 19% attended college, while 60% of those with parents in the highest income bracket attended college (Shattuck et al. 2012). Although this disparity certainly exists among young adults without ASD as well, the added expense of transition programming and supports creates an additional barrier for prospective college students from lower-income households.

Finally, as the effects of autism are not mitigated by college graduation, there is a need to explore ongoing adult services and successful transition to these services for many individuals who have participated in college transition programs. A large number of individuals, even those who have successfully completed college, will continue to require supports with various aspects

of adult life, from independent living to developing social networks to employment and career support.

References and Reading

- Americans With Disabilities Act of 1990, Pub. L. No. 101–336, 104 Stat. 328 (1990).
- Autism Science Foundation. (2012). *How common is autism?* Retrieved from <http://autismscience-foundation.org/what-is-autism/how-common-is-autism>.
- Barnhill, G. P. (2014). Supporting students with Asperger syndrome on college campuses: Current practices. *Focus on Autism and Other Developmental Disabilities*. <https://doi.org/10.1177/1088357614523121>.
- Barnhill, G. P. (2016). Supporting students with Asperger syndrome on college campuses: Current practices. *Focus On Autism And Other Developmental Disabilities*, 31(1), 3–15.
- Cederlund, M., Hagberg, B., Billstedt, E., Gillberg, I., & Gillberg, C. (2008). Asperger syndrome and autism: A comparative longitudinal follow-up study more than 5 years after original diagnosis. *Journal of Autism and Developmental Disorders*, 38, 72–85.
- Centers for Disease Control and Prevention. (2014). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 Sites, United States, 2010. Surveillance summaries. Retrieved from http://www.cdc.gov/mmwr/preview/mmwrhtml/ss6302a1.htm?s_cid=ss6302a1_w
- Centers for Disease Control and Prevention. (2016). *Community report on autism*. Atlanta
- Ellison, M., Clark, J., Cunningham, M., & Hansen, R. (2012). *Benchmarks of effective supports for college students with autism spectrum disorders*. Huntington: The West Virginia Autism Training Center at Marshall University. Retrieved from: <http://www.marshall.edu/collegeprogram/files/Benchmarks-of-Effective-Supports.pdf>.
- Fombonne, E. (1999). The epidemiology of autism: A review. *Psychological Medicine*, 29(4), 769–786.
- Gelbar, N., Smith, I., & Reichow, B. (2014). Systematic review of articles describing experience and supports of individuals with autism enrolled in college and university programs. *Journal Of Autism & Developmental Disorders*, 44(10), 2593–2601. <https://doi.org/10.1007/s10803-014-2135-5>.
- Howlin, P. (2014). Outcomes in adults with autism spectrum disorders. In F. R. Volkmar, S. J. Rogers, R. Paul, K. A. Pelphrey, F. R. Volkmar, S. J. Rogers, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders: Diagnosis, development, and brain mechanisms* (pp. 97–116). Hoboken: Wiley.
- Lever, A. G., & Geurts, H. M. (2016). Psychiatric co-occurring symptoms and disorders in young, middle-aged, and older adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(6), 1916–1930. <https://doi.org/10.1007/s10803-016-2722-8>.
- Roux, A. M., Shattuck, P. T., Cooper, B. P., Anderson, K. A., Wagner, M., & Narendorf, S. C. (2013). Post-secondary employment experiences among young adults with an autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 52(9), 931–939. <https://doi.org/10.1016/j.jaac.2013.05.019>.
- Roux, A. M., Shattuck, P. T., Rast, J. E., Rava, J. A., & Anderson, K. A. (2015). *National autism indicators report: Transition into young adulthood*. Philadelphia: Life Course Outcomes Research Program, A.J. Drexel Autism Institute, Drexel University.
- Section 504 of the Rehabilitation Act of 1973, Pub. L. No. 93–112, 87 Stat. 394 (Sept. 26, 1973).
- Shattuck, P. T., Narendorf, S. C., Cooper, B., Sterzing, P. R., Wagner, M., & Taylor, J. L. (2012). Post-secondary education and employment among youth with an autism spectrum disorder. *Pediatrics*, 129(6), 1042. <https://doi.org/10.1542/peds.2011-2864>.
- Van Hees, V., Moyson, T., & Roeyers, H. (2015). Higher education experiences of students with autism spectrum disorder: Challenges, benefits and support needs. *Journal of Autism and Developmental Disorders*, 45(6), 1673–1688. <https://doi.org/10.1007/s10803-014-2324-2>.
- VanBergeijk, E., Klin, A., & Volkmar, F. (2008). Supporting more able students on the autism spectrum: College and beyond. *Journal Of Autism & Developmental Disorders*, 38(7), 1359–1370.
- Van Schalkwyk, G. I., Beyer, C., Martin, A., & Volkmar, F. R. (2016). College students with autism spectrum disorders: A growing role for adult psychiatrists. *Journal of American College Health*, 64(7), 575–579.

Colossal Commissure

► Corpus Callosum

Combat Disorder

► Posttraumatic Stress Disorder

Combat Fatigue

► Posttraumatic Stress Disorder

Combat Neurosis

► Posttraumatic Stress Disorder

Comic Strip Conversations

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Definition

Comic strip conversations are a social narrative that depicts or enhances a conversation or social situation between two individuals by specifying the underlying thought processes and/or communicative exchanges using line drawings incorporating thought bubbles, speaker bubbles, and other symbols. “Comic strip conversations systematically identify what people say and do, and emphasize *what people may be thinking*” (p. 1, Gray 1994).

Historical Background

Comic strip conversations were first described by Carol Gray in 1994 (Gray 1994) and are closely related to Gray’s Social Stories. Since then, little research has evaluated the efficacy of the intervention, and little development beyond what was initially described by Gray has occurred.

Rationale or Underlying Theory

A comic strip conversation is a type of social narrative. Other types of social narratives include Social Stories, cartooning, and Power Cards (Wragge 2011). A social narrative is a written description of various social situations that might

be problematic for an individual with an ASD. The narrative helps explain what typically occurs in a given social situation, thoughts other individuals might have, and how one is expected to act in a given situation. Comic strip conversations enhance a conversation between two individuals by specifying the underlying thought processes and/or communicative exchanges using line drawings incorporating thought bubbles, speaker bubbles, and other symbols. Through the incorporation of these items into a conversation, an individual with an ASD is provided a visual and concrete depiction of the conversation and/or social situation. Providing this pictorial depiction (representation) of an abstract social situation is thought to capitalize on the improved processing of visual information that many individuals with ASDs have. The exact mechanisms by which comic strip conversations and other social narratives are effective remain unknown.

Goals and Objectives

Comic strip conversations aim to help an individual with an ASD understand the underlying thought processes and/or communicative exchanges that occur during conversations and other social situations. Comic strip stories help provide a concrete depiction of the expectations of individuals in specific social situations and the impact that their actions can have on the thoughts of others in that situation, thereby providing the individual with ASD a scheme for improving their behavior in a social situation. Through the use of thought bubbles (described later), one is able to explicitly show a person’s thoughts, thus providing insight into their theory of mind, which is often a difficult area for individuals with ASD. Because comic strip conversations can provide this visual representation of theory of mind, this is also often a target of intervention.

Treatment Participants

Since there has been little empirical study of comic strip conversations, the following

parameters are based on recommendations based on the intervention's components and techniques. Comic strip conversations are likely to be most beneficial to individuals who have higher cognitive functioning skills across all ASD diagnoses. Although young children (e.g., children younger than 5 years) might benefit from comic strip conversations, the heavy emphasis on language and higher-order social thinking makes school-age children, adolescents, and adults to be the ages of individuals who are likely to receive the most benefit.

Treatment Procedures

Comic strip conversations are typically completed in a one-to-one child to therapist (teacher, parent, etc.) ratio. The intervention begins with the adult engaging in small talk to strengthen rapport. The child or therapist then begins the conversation by drawing a social scene involving multiple people. Multiple boxes (or pages) can be used if the scene involves multiple steps or situations. Speaker bubbles can be used to indicate what people are saying in the scene, and thought bubbles can be added to indicate what people are thinking (but not saying) during the scene. Thought bubbles provide a nice way for the therapist to provide perspective of what people are likely to be thinking but not necessarily saying during a situation exchange or interaction. After the therapist feels the child has processed the social situation and the perspectives of those involved, the therapist then can ask the child to summarize the situation and provide solutions to the problems the child is likely to face when they later participate in similar situations.

Efficacy Information

There have been no experimental studies presenting quantitative data demonstrating the efficacy of comic strip conversations for students with ASDs. Two reports (Pierson and Glaeser 2007; Rogers and Myles 2001) presented brief descriptions of the perceived efficacy of comic strip conversations that were used to increase

prosocial behaviors of individuals with ASDs. Pierson and Glaeser reported qualitative data suggesting comic strip conversations increased peer interactions and decreased loneliness for three children with ASDs. Rogers and Myles reported that comic strip conversations appeared to be more effective (as measured by teacher count of the number of redirections for desired behavior) than social stories for helping a 14-year-old boy with Asperger's disorder navigate to PE class. Clearly, more research on the efficacy of comic strip conversations is needed before the technique can be considered an evidence-based practice.

Outcome Measurement

Comic strip conversations are typically writing for individual children targeting specific behaviors. Therefore, outcome measurement is likely to focus on behavioral measures of the target behaviors. Since theory of mind is often targeted, some measures of theory of mind (e.g., faux pas tasks, strange stories, false-belief tasks) might be used to assess the impact of the intervention technique on this construct.

Qualifications of Treatment Providers

There are no formal qualifications (i.e., credentials, licensure) necessary for using comic strip conversations. There is a brief manual (Gray 1994) providing basic guidelines for using the intervention. Additional practice parameters have also been published (Glaeser et al. 2003; Rogers and Myles 2001). What is necessary is that the interventionist has knowledge about how social deficits in ASDs are manifested and how autism can limit an individual's ability to interpret social situations and function independently in a typical social milieu and the ability to integrate that knowledge into meaningful examples of how one can improve their social behavior. Parents, psychologists, special education teachers, and speech-language pathologists are typical groups of interventionists that might use comic strip conversations.

See Also

- [Social Skill Interventions](#)
- [Social Stories](#)
- [Theory of Mind](#)

References and Reading

- Glaeser, B. C., Pierson, M. R., & Fritschmann, N. (2003). Comic strip conversations: A positive behavioural support strategy. *Teaching Exceptional Children*, 36(2), 14–19.
- Gray, C. (1994). *Comic strip conversations: Colorful, illustrated interactions with students with autism and related disorders*. Arlington: Future Horizons.
- Pierson, M. R., & Glaeser, B. C. (2005). Extension of research on social skills training using comic strip conversations to students without autism. *Education and Training in Developmental Disabilities*, 40(3), 279–284.
- Pierson, M. R., & Glaeser, B. C. (2007). Using comic strip conversations to increase social satisfaction and decrease loneliness in students with autism spectrum disorder. *Education and Training in Developmental Disabilities*, 42(4), 460–466.
- Rogers, M. F., & Myles, B. S. (2001). Using social stories and comic strip conversations to interpret social situations for an adolescent with Asperger syndrome. *Intervention in School and Clinic*, 36(5), 310–313.
- Wragge, A. (2011). Social narratives: Online training module. In Ohio Center for Autism and Low Incidence (OCALI), *Autism Internet Modules*, www.autisminternetmodules.org. Columbus: OCALI.

Comics

- [Visual Narrative Comprehension](#)

Command

- [Mands](#)

Comment

- [Protodeclarative](#)

Commercial Hair Analysis

- [Hair Analysis](#)

Common Disease-Common Variant Hypothesis

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Synonyms

CDCVH

Definition

The Common Disease-Common Variant Hypothesis (CDCVH) is a hypothesis that proposes if a disease that is heritable is common in the population (a prevalence greater than 1–5%), then the genetic contributors – specific variations in the genetic code – will also be common in the population. It makes this prediction for diseases whose genetic contribution is believed to come from multiple genes simultaneously, polygenic disorders. Autism is thought to be such a disorder.

The CDCVH is based on evidence from evolutionary theory, specifically that all humans today descended from a small population of roughly 10,000 individuals in Africa a relatively short time ago, approximately 100,000 years ago. Based on evidence about how frequently new mutations enter the population, it states that the extremely rapid population expansion that has occurred over a short period disseminated disease alleles (genetic changes or variations) that were common in the original population at a far greater rate than new mutations have introduced such alleles. Therefore, genetically influenced

diseases that are common today should be the result of disease alleles that were common in the original population and should still be common in today's population, as they would have been widely distributed by the massive population explosion faster than new disease alleles could be introduced.

Several types of studies can be undertaken to identify common genetic variants contributing to common disease. The most reliable of these is called a genome-wide association study (GWAS). These evaluate common genetic variations, often in the form of single nucleotide polymorphisms (SNPs), at every gene in the genome simultaneously. Such studies have reproducibly identified common genetic risks for a wide range of common medical conditions. However, there have only been a small number of cases in which these common genetic variations explain a substantial proportion of the overall predicted genetic risks for the disorder. As a result, some have put forth a competing hypothesis, the Common Disease-Rare Variant Hypothesis, that states that common diseases may be explained by a multiplicity of individually rare disease alleles in the population.

With regard to autism, several GWAS studies have been conducted, and, so far, three SNPs have been found that meet criteria for significance, taking into account the fact that the entire genome has been evaluated simultaneously. However, none of the three studies replicate the others' findings, and combining the data from all three decreases the evidence for any one of these genetic markers being associated with autism spectrum disorders. While it is expected that common genetic polymorphisms carry risks for ASD, there is not yet agreement that any particular variation or gene has been definitively identified.

See Also

- [Common Disease-Rare Variant Hypothesis](#)
- [DNA](#)
- [Genetics](#)
- [Genome-wide Association](#)

References and Reading

- Anney, R., Klei, L., Pinto, D., Regan, R., Conroy, J., Magalhaes, T. R., et al. (2010). A genome-wide scan for common alleles affecting risk for Autism. *Human Molecular Genetics*, 19(20), 4072–4082.
- Borch-Johnsen, K., Burtt, N. P., Chen, H., Chines, P. S., Daly, M. J., Deodhar, P., Ding, C. J., et al. (2008). Meta-analysis of genome-wide association data and large-scale replication identifies additional susceptibility loci for type 2 diabetes. *Nature Genetics*, 40(5), 638.
- Chakravarti, A. (1999). Population genetics-making sense out of sequence. *Nature genetics*, 21(Suppl. 1), 56.
- El-Fishawy, P., & State, M. W. (2010). The genetics of autism: Key issues, recent findings, and clinical implications. *Psychiatric Clinics of North America*, 33(1), 83–105.
- Iyengar, S. K., & Elston, R. C. (2007). The genetic basis of complex traits: Rare variants or “common gene, common disease”. *Methods in Molecular Biology*, 376, 71. (Clifton, NJ).
- Reich, D. E., & Lander, E. S. (2001). On the allelic spectrum of human disease. *Trends in Genetics*, 17(9), 502.
- Wang, K., Zhang, H., et al. (2009). Common genetic variants on 5p14.1 associate with Autism spectrum disorders. *Nature*, 459, 528–533.
- Weiss, L. A., Arking, D. E., & The Gene Discovery Project of Johns Hopkins & the Autism Consortium. (2009). A genome-wide linkage and association scan reveals novel loci for autism. *Nature*, 461(7265), 802–808.

Common Disease-Rare Variant Hypothesis

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Definition

The Common Disease-Rare Variant Hypothesis (CDRVH) hypothesizes that if a disease with genetic causes is common in the population (a prevalence greater than 1–5%), then the genetic causes – specific genetic errors (genetic variants or disease alleles) – will not necessarily be found to be common in the population as suggested by the competing Common Disease-Common Variant Hypothesis (CDCVH) but rather will be comprised of a multiplicity of risk alleles, each of

which is individually rare in the population. It makes this prediction for diseases whose genetic contribution is believed to come from multiple genes simultaneously, polygenic disorders. Autism is thought to be such a disorder.

The CDRVH states that common polygenic disorders may reflect the convergence of multiple, rare variations in the same gene (allelic heterogeneity) or multiple genes (locus heterogeneity). Given a sufficiently large number of disease alleles that could be involved, individually rare mutations could accumulate in the population and account for a significant proportion of a common disorder. Such variation could be transmitted from generation to generation or occur as new mutations (also known as de novo mutation). The latter would give rise to a pattern of inheritance prevalent in autism, where cases are often sporadic (only a single child is affected in a family) and identical (monozygotic) twins share the disorder much more frequently than nonidentical (dizygotic twins).

Proponents of the CDRVH have recently been able to cite considerable empiric evidence supporting this hypothesis. First, to date, it has been difficult to confirm the presence of common variants contributing risk for ASD. Several contemporary well-controlled association studies have implicated biologically plausible candidate genes, including the *MET oncogene*, *Contact-Associated Protein Like 2*, *Cadherin 9* and *Cadherin 10*, *Semaphorin 5A*, and *MACRO domain containing 2* (State and Levitt 2011). However, at present, the field has not reached a clear consensus about which of these genes or loci carry definitive risks. More importantly, were all of these association signals to be confirmed, they would nonetheless account for only a small proportion of the predicted genetic contribution to ASD. Perhaps, more importantly, to date, studies of rare variation have begun to demonstrate that a measurable proportion of the ASD-affected population carries very rare genetic mutations, either in the structure or sequence of the DNA, contributing biological effects that are much larger than those so far associated with common variants. For example, current estimates are that 5–10% of affected

individuals carry large de novo copy number variations (CNVs) that substantially increase the risk for ASD (and other neurodevelopmental disorders). In addition, a measurable proportion of individuals with ASD can be found to have mutations in *FMRP*, *TSC-1*, *TSC-2*, or other rare so-called monogenic forms of autism. Finally, with the advent of new DNA sequencing technology, it is anticipated that additional individuals will be found carrying rare and de novo single nucleotide variants (SNVs) contributing relatively large risks. All told these findings provide substantial evidence in favor of the CDRVH in a proportion of individuals with social disability. It is important to recall, however, that in a genetically heterogeneous disorder such as autism, the contribution of rare variants does not rule out the potential contribution of common variants, either with respect to etiology or as a factor modifying other genetic and environmental risks.

See Also

- ▶ [Candidate Genes in Autism](#)
- ▶ [Common Disease-Common Variant Hypothesis](#)
- ▶ [Copy Number Variation](#)
- ▶ [DNA](#)
- ▶ [Genetics](#)

References and Reading

- El-Fishawy, P., & State, M. W. (2010). The genetics of Autism: Key issues, recent findings, and clinical implications. *The Psychiatric Clinics of North America*, 33(1), 83–105.
- Iyengar, S. K., & Elston, R. C. (2007). The genetic basis of complex traits: Rare variants or “common gene, common disease?” *Methods in Molecular Biology*, 376, 71. Clifton, NJ.
- Ji, W., Foo, J. N., et al. (2008). Rare independent mutations in renal salt handling genes contribute to blood pressure variation. *Nature Genetics*, 40(5), 592.
- Reich, D. E., & Lander, E. S. (2001). On the allelic spectrum of human disease. *Trends in Genetics*, 17(9), 502.
- State, M. W., & Levitt, P. (2011). The conundrums of understanding genetic risks for autism spectrum disorders. *Nature Neuroscience*, 14(12), 1499–1506.

Communication and Symbolic Behavior Scale

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Synonyms

CSBS; CSBS-DP

Description

The *Communication Symbolic and Behavior Scales-Normed Edition* (CSBS; Wetherby and Prizant 1993) is a norm-referenced, standardized test designed to assess infants, toddlers, and pre-schoolers that are at risk for communication delays. In addition, this measure is used to establish a profile of communicative, symbolic, and social-affective functioning of a child, to monitor behavior change over time, and to provide a direction for intervention. The assessment surveys both language skills and symbolic development, including gestures, facial expressions, and play, using 22 5-point rating scales (18 in the communicative domain, 4 in the symbolic domain). This measure can be used with infants and toddlers with functional communication ages of 6–24 months and children up to 72 months exhibiting atypical development. It can be administered by a speech-language pathologist (SLP), psychologist, early interventionist, and other professional trained to work with developmentally young children. As a part of this assessment, parents or caregivers complete a Caregiver Questionnaire, providing background information that can be utilized as a baseline for evaluation. The evaluator samples the child's communicative behaviors in structured and unstructured play-based activities in the child's natural environment. Communicative temptations, sharing books, symbolic play, language comprehension, and constructive play activities are utilized to encourage spontaneous communicative and play behaviors. Ideally, the

sample is videotaped, so as to continue natural interactions during the assessment and to ensure accurate scoring and analysis. This test takes approximately 1 hour to administer and 1 hour to score the videotape. The Caregiver Questionnaire takes about 15 min to complete.

The *Communication Symbolic and Behavior Scales-Developmental Profile* (CSBS-DP; Wetherby and Prizant 2002) is the companion test to the CSBS that is designed to evaluate communication and symbolic abilities of children in the same age range as the CSBS-Normed Edition. However, this test is intended as a guide to indicate areas that may need further assessment or to monitor behavior change. This test includes a one-page Infant-Toddler Checklist for screening, a four-page Caregiver Questionnaire, and Behavior Sample, which is a shorter, more streamlined version of the CSBS-Normed Edition. It should be noted that the CSBS-DP should not be used alone for decisions about program planning. The CSBS-Normed Edition is a more in-depth tool and is designed for making program-planning decisions. The CSBS-DP takes about 30 min to administer, and the Behavior Sample can be scored during the sample or videotaped. The Infant-Toddler Checklist can be completed in 5–10 min, and the Caregiver Questionnaire can be completed in about 20 min.

Historical Background

In 1986, the passage of the Education of the Handicapped Act, Amendments of 1986 (PL 99-457) provided funds to states that chose to develop and implement early identification and intervention services for infants and toddlers beginning in 1991. This included children that were at high risk or children up to their third birthday that had identified disabilities, including delays in speech and language. Often, speech-language delay is the first symptom of developmental delay that parents or professionals notice. Unfortunately, however, early identification was compromised by the limited number of standardized tools for assessing very young children. Early identification of these delays or lack of speech-

language development is crucial, as typical early language development occurs between 12 and 20 months of age. The detrimental effects of early speech and language disorders on later development of peer relations, educational success, as well as emotional and behavioral development have also been well documented in the research (Prizant et al. 1990). Most formal tests used to measure children's communication abilities are clinician-directed and focus on the child as a responder. There were few tests available that sampled communication, especially communicative intent, in a naturalistic way. Communication sampling is needed to supplement formal testing for children who are preverbal or at an early verbal communication stage (Wetherby et al. 1988). Therefore, the CSBS was developed to allow for an informal communication sample. With the implementation of PL 99-457, it was critical that clinicians utilize informal sampling procedures in their assessment process.

The sampling procedures were developed and refined over a 10-year period. Pragmatic and social interactive theories from the 1970s and 1980s (Bloom and Lahey 1978) were utilized to make changes to the sampling procedures. Procedures including the presentation of communicative temptations or structured situations that encourage or entice a child to communicate were adapted from informal procedures to sample communication (Wetherby and Prizant 1989). This included an assessment of both communicative functions and communicative means. Sharing books, which was a temptation originally reported by Wetherby and Prutting (1984), was later considered separate from the communicative temptations, as it is less structured than the other included temptations. Reciprocity and social-affective signaling are also assessed as a part of this tool. Addition of these areas of assessment was based on the work of Stern (1985) and Tronick (1989) in socioemotional development. The communication sample provides a way to measure expressive language abilities, and a small measure of language comprehension was also added. These items were adapted from the work of Miller, Chapman, Branston, and Reichle (1980). Play skills, both symbolic and

constructive, were included in the sample, and toy sets were chosen for children that were developmentally appropriate for children 8 months to 24 months. The development of the symbolic scales was influenced by the model for emergence of symbols (Bates 1979) and based on theories of Piaget (Wetherby 1991).

The *CSBS-DP* (Wetherby and Prizant 2002) *Infant-Toddler Checklist* and the *CSBS-DP Caregiver Questionnaire* were adapted from the *CSBS Caregiver Questionnaire* (Wetherby and Prizant 1993) and from research on the *MacArthur Communicative Development Inventories* (CDI; Fenson et al. 1993, 1994). Studies indicate that parent report is a reliable measure of communication development, and a checklist format was chosen, as this method is more accurate than a diary or free-form format (Fenson et al. 1993). The items on the *CSBS-DP Caregiver Questionnaire* were based on the *CSBS-Normed Edition Caregiver Questionnaire* (Wetherby and Prizant 1993). The questions on the *CSBS-DP Caregiver Questionnaire* are meant to gather similar information to that which is gathered from the Behavior Sample. The words expressed and understood were based on the work of Fenson et al. (1994) and were the first 36 words reported with highest frequency on the MacArthur CDI. The CSBS-DP Behavior Sample was based on the CSBS Behavior Sample (Wetherby and Prizant 1993). However, it was modified by reducing the length of the sample. The scoring procedures were reduced from 23 to 20 scales, which simplified the scoring and enabled the evaluator to score during the observed interactions. Nineteen of the 20 scales were derived from the CSBS; however, the gaze/point-following scale was added based on research findings from Mundy, Kasari, Sigman, and Ruskin (1995).

Psychometric Data

The CSBS-Normed Edition (Wetherby and Prizant 1993) was developed and tested over several years with both normally developing children and children with language delays. This research edition of this test was standardized in 1990 and

1991. The videotaped samples were scored by a group of raters that were trained by the authors. Before beginning the rating for the norming study, they rated two additional tapes to calibrate with ratings of an experienced coder. The samples were taken for 282 children from 24 sites in the United States. The norming sample was weighted for scaling and norming analyses. Information about the sample provided in the manual includes age in months, linguistic stage, gender, race, and Spanish origin. Since the CSBS was intended to monitor a child's progress over several months, one-third of the sample were retested once within 2–3 months of the first administration.

Norms were developed by making the 22 scales comparable by converting individual raw scores to a common metric based on the sample. This was done by deriving percentile ranks of raw scores on each scale based on the frequency distributions of these scores for the weighted sample between 8 and 24 months of age. Cluster scores were established that were based on summing of the scaled scores. The scaled scores for the 18 communication scales were summed to create a larger cluster score, the communication composite. The communication score and all cluster scores are expressed as percentile ranks. Means and standard deviations of raw scores and sums of scaled scores by language age and total unweighted scores are provided in the manual. Reliability of this measure is reported using four common methods. Internal consistency, the degree to which the parts of the instrument measure the same characteristic, yielded a coefficient of 0.91 for the entire sample and 0.84 for children at the multiword stage. Stability, the consistency of the test and retest results for individual test participants whose performance has not changed, is provided. Test-retest scores are provided for all age intervals, including shorter intervals (less than 2 months) and longer intervals (greater than 2 months). Correlations between the test and retest scores for the shorter and longer interval subgroups are also provided. Taken together, this information indicates that the CSBS produces relatively stable rankings when children make significant improvements over shorter periods of time. Interrater reliability coefficients are also

provided. Raters following the CSBS training steps can achieve good agreement with other raters and experienced raters. Standard errors of measurement (SEMs) are provided for all cluster standard scores and communication-composite scores, along with details on confidence ranges. Demonstration of validity is provided, including face validity/ecological validity, criterion-related validity, and construct validity. Further, a complete assessment of gender differences in test outcomes is included in the manual. Studies of the CSBS demonstrate sensitivity and valid norms (Goodwyn and Cruz 1997) and suggest that the instrument has good predictive validity (McCathren et al. 2000).

The CSBS-DP (Wetherby and Prizant 2002) Preliminary Research Edition was developed and field tested with children younger than 24 months of age and was standardized between 1997 and 2000. Collection of Infant-Toddler Checklists, Caregiver Questionnaires, and Behavior Samples was used to derive norms. Raters were trained in the same way as was utilized for the norming of the CSBS. A total of 2,188 Infant-Toddler Checklists, 790 Caregiver Questionnaires, and 337 Behavior Samples were included for the standardization sample from eight sites in the United States and two sites in Canada. A majority of the United States' sample was recruited primarily from Tallahassee, Florida; therefore, the sample is not nationally representative. Information about the sample by age, gender, race and ethnicity, and parent age and ethnicity is included in the manual. Norms were procedurally derived in the same way as the CSBS. All reliability measures including internal consistency, SEMs and confidence intervals, test-retest reliability, and interrater reliability are included in the manual. Validity measures including content, face, construct, criterion, concurrent, and predictive validity are provided. The CSBS-DP shows good reliability and validity, indicating it is a good screening and evaluation tool for use with children between 6 and 24 months of age. Recent research indicates the concurrent and predictive validity of this instrument to be strong, and the findings support the use of this instrument in the screening and evaluation of young children (Wetherby et al. 2003).

Clinical Uses

The CSBS-Normed Edition (Wetherby and Prizant 1993) was designed to be used for early identification of children who have or are at risk for developing communication impairment. In addition, it was intended to establish a profile of communication, symbolic, and social-affective functioning. For this reason, the test helps early intervention providers to gauge further evaluation needs, to prioritize intervention goals, and to monitor progress. In the manual, there are guidelines for using the results of this profile in intervention planning, goal setting, and designating intervention contexts. Areas addressed include expanding the use of social-affective signals, enhancing reciprocity, expanding the range of communicative functions, increasing the sophistication of communicative means, and enhancing symbolic level. The use of this profile is recommended to assign relative strengths and challenges in the areas listed above and to provide an individualized approach to prioritizing goals. It is suggested that the communicative profile be compared to the symbolic profile and to design-focused activities utilizing developmentally age-appropriate toys and objects. The child's behavior displayed during the structured activities of the CSBS should be compared to the child's functioning in less structured environments. The authors suggest that activities that contain increased communicative demands should be planned with the child's strengths in mind. Naturalistic activities that allow for generalization of learning and communication skills are also recommended. Since the CSBS-DP (Wetherby and Prizant 2002) is intended as a guide to indicate areas that may need further assessment or to monitor change in behaviors, the CSBS-DP should not be used alone for decisions about program planning.

See Also

- [Early Intervention](#)
- [MacArthur-Bates Communicative Development Inventories, Second Edition](#)
- [Mullen Scales of Early Learning](#)

- [Normative Data](#)
- [Reciprocal Communication/Interaction](#)
- [Standardized Tests](#)

References and Reading

- Bates, E. (1979). *The emergence of symbols: Cognition and communication in infancy*. New York: Academic.
- Bloom, L., & Lahey, M. (1978). *Language development and language disorders*. New York: Wiley.
- Education of the Handicapped Amendments Act of 1986, Public Law 99-457, 100 Stat, 1145, (1986).
- Fenson, L., Dale, P., Reznick, S., Thal, D., Bates, E., Hartung, J., et al. (1993). *MacArthur communicative development inventories: User's guide and technical manual*. San Diego: Singular.
- Goodwyn, C., & Cruz, R. (1997). Test review: Communication and symbolic behavior scales. *Assessment for Effective Intervention*, 23(1), 233-240.
- McCathren, R., Yoder, P., & Warren, S. (2000). Testing predictive validity of the communication composite of the communication symbolic behavior scales. *Journal of Early Intervention*, 23(1), 36-46.
- Miller, J., Chapman, R., Branston, M., & Reichle, J. (1980). Language comprehension in sensorimotor stages V and VI. *Journal of Speech and Hearing Research*, 23, 284-311.
- Mundy, P., Kasari, C., Sigman, M., & Ruskin, E. (1995). Nonverbal communication and early language acquisition in children with Down syndrome and normal development. *Journal of Speech and Hearing Research*, 38, 157-167.
- Oosterling, I. J., Swinkles, S. H. N., van der Gaag, R. J., Visser, J. C., Dietz, C., & Buitelaar, J. K. (2009). Comparative analysis of three screening instruments for autism spectrum disorders in toddlers at high risk. *Journal of Autism and Developmental Disorders*, 39(6), 897-909.
- Prizant, B., Audet, L., Burke, G., Hummel, L., Maher, S., & Theodore, G. (1990). Communication disorders and emotional/behavioral disorders in children. *The Journal of Speech and Hearing Disorders*, 55, 179-192.
- Stern, D. (1985). *The interpersonal worlds of the infant*. New York: Basic Books.
- Stone, W., & Caro-Martinez, L. (1990). Naturalistic observations of spontaneous communication in autistic children. *Journal of Autism and Developmental Disorders*, 20(4), 437-453.
- Tronick, E. (1989). Emotions and emotional communication in infants. *American Psychologist*, 44, 112-119.
- Wetherby, A. (1991). Profiling pragmatic abilities in the emerging language of young children. In T. Gallagher (Ed.), *Pragmatics of language: Clinical practice issues* (pp. 249-281). San Diego: Singular.
- Wetherby, A., & Prizant, B. (1989). The expression of communicative intent: Assessment guidelines. *Seminars in Speech and Language*, 10, 77-91.

- Wetherby, A., & Prizant, B. (1993). *Communication and symbolic behavior scales*. Chicago: Applied Symbolix.
- Wetherby, A., & Prizant, B. (2002). *Communication and symbolic behavior scales-developmental profile*. Baltimore: Paul H. Brookes.
- Wetherby, A., & Prutting, C. (1984). Profiles of communicative and cognitive-social abilities in autistic children. *Journal of Speech and Hearing Research*, 27, 364–377.
- Wetherby, A., Cain, D., Yonclas, D., & Walker, V. (1988). Analysis of intentional communication in normal children from the prelinguistic to the multiword stage. *The Journal of Speech and Hearing Disorders*, 31, 240–252.
- Wetherby, A., Goldstein, H., Cleary, J., Allen, L., & Kublin, K. (2003). Early identification of children with communication disorders: Concurrent and predictive validity of the CSBS developmental profile. *Infants and Young Children*, 16(2), 161–174.

Communication and Symbolic Behavior Scales – Developmental Profile

► [Infant/Toddler Checklist](#)

Communication and Symbolic Behavior Scales – Normed Edition (2003)

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Abbreviation

CSBS Communication and Symbolic Behavior
Scales-Normed Edition

Description

The Communication and Symbolic Behavior Scales (CSBS; Wetherby and Prizant 2003) looks at the communicative, social-affective, and

symbolic abilities of children whose functional communication age is between 8 months and 2 years. Its purpose is to assess infants, toddlers, and preschool children, and it may be used with children whose chronological age ranges from about 8 months to 5–6 years, if their developmental age is younger than 24 months. The CSBS provides quantitative and qualitative information about a child's communicative and symbolic abilities.

The CSBS contains three formal tests: the CSBS Caregiver Questionnaire, the CSBS Behavior Sample, and the Caregiver Perception Rating form. The CSBS Caregiver Questionnaire gains information from the caregiver regarding the child's communication and symbolic abilities and can be administered prior to the child's direct assessment. The questionnaire can be completed in about 15–20 min. The CSBS Behavior Sample is a play-based sampling procedure. It is broken up into six sampling contexts: warm-up, communicative temptations, sharing books, symbolic play probes, language comprehension probes, and constructive play probes. This takes about 1 h to complete and must be videotaped to complete scoring. A trained evaluator can then score the videotape in about 1 h. Immediately following the Behavior Sample, the caregiver should be asked to complete the Caregiver Perception Rating form. The caregiver rates the typicality of their child's behavior as observed during the Behavior Sample. The examiner may choose to interview the caregiver using this form as an alternative option.

The CSBS kit includes the CSBS Manual, a carrying bag with materials for the CSBS Behavior Sample, two instructional videotapes for the Behavior Sample that demonstrate the sampling procedures and scoring guidelines, Record Forms for scoring and reporting results, and the CSBS Caregiver Questionnaire. The materials included that are used during the CSBS Behavior Sample are action-based toys to get spontaneous communication, books for young children, and play materials that are used to see how a child uses and plays with objects symbolically and constructively.

The behaviors observed during the CSBS Behavior Sample are rated according to

22 5-point scales. These scales measure the following parameters, which are summarized in seven cluster scores: Communicative Function (scales 1–3) which examines (1) behavior regulation, (2) joint attention, and (3) sociability of communicative function; Communicative Means-Gestural (scales 4–6) which examines (4) conventional gestures, (5) distal hand gestures, and (6) coordination of gestures and vocalizations; Communicative Means-Vocal (scales 7–10) which examines (7) vocal acts without gestures, (8) inventory of different consonants, (9) syllables with consonants, and (10) multisyllables; Communicative Means-Verbal (scales 11–12) which examines (11) inventory of different words expressed and (12) inventory of different word combinations; Reciprocity (scales 13–15) which examines (13) respondent acts, (14) rate of communicative acts per minute, and (15) repair strategies; Social-Affective Signaling (scales 16–18) which examines (16) gaze shifts, (17) shared positive affect, and (18) episodes of negative affect; and Symbolic Behavior (scales 19–22) which examines (19) language comprehension, (20) inventory of different action schemes, (21) complexity of action schemes, and (22) constructive play.

The CSBS records three types of scores: raw scores, scaled scores, and normed scores. Raw scores and equivalent scaled scores are reported for the 22 CSBS Behavior Sample scales. Converting raw scores to scaled scores can be done using the Record Form. The scaled scores have a mean of 3 and a standard deviation of 1. Normed scores are reported for the seven clusters and for the communication composite score. Normed scores reflect the performance of children in the standardization sample both by 1-month intervals from 8 to 24 months and by language stage from prelinguistic to multiword. The Communication Composite is a sum of the scaled scores for scales 1–18. The scaled scores of the Communication Composite must be converted to an age-based or stage-based percentile rank and standard score scale that has a mean of 10 and a standard deviation of 3. Since the Communication

Composite scores have a large range and precision, the composite may also be converted to an expanded standard score scale that is widely used for composite scores on other test batteries with a mean of 100 and a standard deviation of 15. The CSBS Caregiver Questionnaire reports cluster raw scores for emotion and eye gaze, communication, gestures, sounds, words, understanding, and object use. The Caregiver Questionnaire also reports a social composite score, a speech composite score, and a symbolic composite score. The social composite score is a sum of the raw scores for emotion and eye gaze, communication, and gestures. The speech composite score is a sum of the raw scores for sounds and words. The symbolic composite score is a sum of the raw scores for understanding and object use.

Historical Background

The purpose of developing the CSBS was to provide an assessment tool for young children that were more representative of current research to identify and assess language and communication delays and disorders at an early age. As stated in the Individuals with Disabilities Education Act (IDEA) Amendments of 1991, and the Handicapped Act Amendments of 1986, a priority is to provide early identification and intervention services for at-risk infants and toddlers. The US Department of Education (1987) note that about 70% of preschool children with developmental disabilities have language and communication disorders, which highlights the need to develop assessments aimed toward the early identification of these children. Early identification and appropriate interventions can be an overarching factor to a child's future educational achievements and overall development (Wetherby and Prizant 2003). The most widely used communication and language assessment instruments for prelinguistic and early linguistic children emphasize the form of communication, rather than the intentions that are expressed with these forms. Previously used instruments are mostly clinician-

directed, limiting the observations of initiated and spontaneous communication used by a child. Language sampling has become more widely used in place of formal language measures for children who engage in conversation. Similarly, communication sampling can be used to supplement children in the preverbal or early verbal stages as it provides a naturalistic environment for assessment (Wetherby and Prizant 2003). This need brought forth the development of the current version of the CSBS that was first made as an informal procedure for sampling communication with preverbal children. The sampling procedures in the CSBS derived from pragmatic and social interaction theories that aimed to mirror a natural, ongoing adult-child interaction, to provide many opportunities for multiple communicative behaviors. The assessment was standardized and normed in response to the need for a more naturalistic assessment environment and the implementation of the IDEA principles to evaluate young children (Wetherby and Prizant 2003).

An initial version of the Communication and Symbolic Behavior Scales-Normed Edition was published in 1993. The previous version of the complete assessment has been changed over time, as scales that proved to be less reliable or discriminating were removed or revised. The Communication and Symbolic Behavior Scales-Developmental Profile (CSBS-DP) was then published in 2002; it is designed as a shortened version of the assessment and includes an Infant-Toddler Checklist. The Communication and Symbolic Behavior Scales-Normed Edition (2003) is the current version of the test that is commonly administered today.

Psychometric Data

The CSBS was standardized in 1990 and 1991, collecting behavior samples of communication and symbolic behaviors from typically developing children and children with delays. Professional scorers were trained to analyze and rate communication and symbolic behavior through

videotaped behavior samples gathered from 282 children. This sample was used to create the norms for the assessment. Children in the standardization sample were recognized by their linguistic stage, gender, race, and Spanish origin. Based on the age of the children in the sample, standard deviations and percentile ranks were created to indicate the level of performance at each linguistic stage. Reliability is reported through measures of internal consistency, stability, and interrater reliability. The internal consistency coefficients reported for the CSBS range from a low of 0.17 for Social-Affective Signaling to a high of 0.91 for Communicative Means-Vocal. The coefficient for the composite is 0.91, indicating strong internal consistency for a total score. Stability was assessed on 66 of the total children participants through a process of test-retest. As expected, correlation of scores was higher for shorter intervals, and effect sizes for the 22 scales are provided, revealing small to medium changes. The authors suggest these changes are likely due to natural period of growth. The interrater reliability was demonstrated by comparing the ratings of 22 children as assessed by 3 raters, which showed good agreement, ranging from 0.83 to 0.90. Standard error of measurement has been calculated and provided for all cluster standard scores as well as the composite score. The construct validity confirms patterns of scores that increased on nearly all 22 scales across selected age intervals, aligning to established developmental expectations. The established means and standard deviations from the raw scores are consistent with known patterns for improvement in communication and symbolic behaviors during the period of early development.

Clinical Uses

The CSBS can be used for clinical purposes by providing quantitative and qualitative information of a child's communicative, social-affective, and symbolic abilities between the ages of 8 months and 2 years. Specific strengths of the assessment

include the ability to capture observation and measurement in the areas of joint attention and intentional communicating. A unique feature of this assessment is that it provides a variety of opportunities for spontaneous initiations of communication, including acts for initiating joint attention (Dos Santos 2009). Joint attention and intentional communication are key areas of deficit in individuals with ASD. The CSBS is a sensitive measure to specific social deficits observed in children with ASD (Wetherby and Prizant 2002).

Research on children with autism spectrum disorder (ASD) has shown how the clinical use of CSBS can be helpful in early diagnoses of ASD given its focus on joint attention and initiating behaviors. For example, it is known that there is an early deficit in shared attention in children with ASD in the second year of life showing lack of appropriate gaze and fewer gaze shifts compared with children with other developmental delays (DD) and typically developing (TD) children (Charman et al. 1997; Swettenham et al. 1998; Wetherby et al. 2004, 2007; Shumway and Wetherby 2009). Additionally, children with ASD evidence a lack of communication involving coordination of at least three of the following: gaze, facial expression, gesture, and sound (Wetherby et al. 2004; Shumway and Wetherby 2009). These well-established early signs of ASD in young children can all be measured through the CSBS and thus has the potential to assist in early diagnosis.

This assessment allows a child's communication and symbolic behavior, which can potentially be missed in other communication assessments, to be measured in different areas based on a clinician's perspective as well as the parent and/or caregiver's perspective. The in-depth nature of this assessment provides quantitative and qualitative information about the child's abilities. The assessment can be administered in a naturalistic environment to observe the child's natural play and allows for multiple developmental domains to be observed in one sitting through an hour of play.

This assessment may take a long time to score based on the video of the Behavior Sample. Fortunately, the authors have developed the CSBS-DP to create a shorter and easier to use tool to

screen and assess young children for early delays, whereas the full CSBS assessment takes twice as long to administer and score and does not provide a checklist that can be used as a screening tool by other medical professionals. The Infant-Toddler Checklist found in the CSBS-DP can be used in clinical practice by a variety of health professionals such as pediatricians and child health nurses to identify infants and toddlers most at risk for a communication delay that may require a referral and further assessment (Eadia et al. 2010). Further research on the CSBS-Normed Edition (2003) would be beneficial to account for the limitations of the assessment and to look at further clinical uses.

See Also

- [Autism: Social Communication Disorder](#)
- [Communication and Symbolic Behavior Scale](#)
- [Symbolic Behavior](#)

References and Reading

- Charman, T., Swettenham, J., Baron-Cohen, S., Cox, A., Baird, G., & Drew, A. (1997). Infants with autism: An investigation of empathy, pretend play, joint attention, and imitation. *Developmental Psychology*, 33, 781–789.
- Dos Santos, K. (2009). *Facilitating initiating joint attention in children with autism spectrum disorder* (Masters dissertation). University of the Witwatersrand, Johannesburg.
- Eadia, P. A., Ukoumunne, O., Skeat, J., Prior, M. R., Bavin, E., Bretherton, L., & Reilly, S. (2010). Assessing early communication behaviours: Structure and validity of the communication and symbolic behaviour scales – Developmental profile (CSBS-DP) in 12-month-old infants. *International Journal of Language & Communication Disorders*, 45(5), 572–585.
- Shumway, S., & Wetherby, A. M. (2009). Communicative acts of children with autism spectrum disorders in the second year of life. *Journal of Speech, Language, and Hearing Research*, 52, 1139–1156.
- Swettenham, J., Baron-Cohen, S., Charman, T., Cox, A., Baird, G., Drew, A., et al. (1998). The frequency and distribution of spontaneous attention shifts between social and non-social stimuli in autistic, typically developing, and non-autistic developmentally delayed infants. *Journal of Child Psychology and Psychiatry*, 39, 747–753.

- Wetherby, A. M., & Prizant, B. M. (1990). *Communication and symbolic behavior scale – Research edition*. Chicago: Riverside.
- Wetherby, A. M., & Prizant, B. M. (2002). *Communication and symbolic behaviour scales – Normed edition*. Baltimore: Paul H. Brooks Publishing Company.
- Wetherby, A. M., & Prizant, B. M. (2003). *Communication and symbolic behavior scale (CSBS)*. Baltimore: Paul H. Brookes Publishing Company.
- Wetherby, A. M., Allen, L., Cleary, J., Kublin, K., & Goldstein, H. (2002). Validity and reliability of the communication and symbolic behavior scales developmental profile with very young children. *Journal of Speech, Language, and Hearing Research*, 45(6), 1202–1218.
- Wetherby, A., Woods, J., Allen, L., Cleary, J., Dickinson, H., & Lord, C. (2004). Early indicators of autism spectrum disorders in the second year of life. *Journal of Autism and Developmental Disorders*, 34, 473–493.
- Wetherby, A., Watt, N., Morgan, L., & Shumway, S. (2007). Social communication profiles of children with autism spectrum disorders late in the second year of life. *Journal of Autism and Developmental Disorders*, 37, 960–975.

future communicative success. The goal of communication assessment is to evaluate a child's current level of communicative skills across various settings and with different partners. Factors included in the assessment of communication are engagement and attention skills, nonverbal behaviors and gestures, and speech and language form and content. Since communication difficulties and pragmatic challenges plague the ability of children with autism to engage in reciprocal exchanges with their communicative partners, it is important to create profiles of communication strengths and challenges to increase understanding of the critical connections between assessment and intervention planning.

There are several screening and diagnostic tools specific to communication that can be used to assess a broad range of skills in children with autism spectrum disorders (ASD). Comprehensive measures are designed to detect problems across speech and language domains, including:

- Receptive and expressive semantic development (assessing vocabulary understanding and expression)
- Receptive and expressive morphosyntactic development (assessing the ability to understand and produce words and sentences)
- Articulation (assessing speech sound production)
- Phonology (assessing the ability to use speech sounds in systematically organized manner)
- Fluency (assessing the smoothness and flow of producing speech)
- Cognitive processes (assessing working memory skills important to the development of language)
- Metalinguistic processes (assessing rhyming and word segmentation abilities critical to the development of language and literacy)

Notably, many communication assessment tools can be used as both screening and identification or diagnostic measures. Although there are comprehensive assessment tools for communication, there are also domain-specific tools for language comprehension, receptive and expressive vocabulary, pragmatics, articulation, etc., which

Communication Assessment

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Definition

Communication assessment is the evaluation of current skills in speech, language, syntax, semantics, morphology, and pragmatics. It is an important process for gathering information to answer questions and make intervention decisions (Schwartz et al. 2001). Importantly, too, the role of assessment extends beyond making diagnoses and identifying skill deficits. A comprehensive communication assessment helps pave the way for intervention planning.

As communication is often an area of difficulty for children with autism, communication assessment is essential in identifying and ultimately addressing the needs of these children to ensure

provide a more detailed examination of a particular area of language development that may be a specific area of challenge for a child with ASD.

Once a diagnosis has been made, the assessment process is not complete until the clinician can profile a child's communication strengths and challenges. Profiling the communication of a child with ASD allows the speech-language pathologist as well as the team, including the family, to develop a more holistic and comprehensive view of the child (Prelock 2006). Creating a communication profile is particularly important for prioritizing communication goals and implementing a developmentally and individually appropriate curriculum (Prelock 2006; Wetherby et al. 2000). Communication profiling requires the clinician to design situations which facilitate a child's ability to communicate and observing what a child does verbally and nonverbally in those situations. An assessment of joint attention is critical as is defining the various gestures, sounds, and words that characterize the child's repertoire (Prelock 2006). Specific assessment strategies are employed for those children with limited or absent verbal skills and those with unconventional verbal behavior. For verbal children and adolescents with ASD, a communication profile needs to carefully evaluate pragmatic language with several communication partners and across a variety of settings. Profiling pragmatic communication in more verbal children with ASD requires an assessment of communicative intent, use of presupposition, and social discourse.

Speech-language pathologists are primarily responsible for the assessment of communication and can provide activities within the home, school, and community to help facilitate communication development and monitor progress (Prelock et al. 2013).

To ensure that a child receives the full benefit of an intervention program, there must be a link between the comprehensive communication assessment (including a profile of communication strengths and challenges) that is completed and the communication goals and curriculum that are established for a child with ASD.

See Also

- Grammar
- Paralinguistic Communication Assessment
- Pragmatics
- Prelinguistic Communication Assessment
- Speech
- Speech Morphology
- Syntax

References and Reading

- Drew, A., Baird, G., Taylor, E., Milne, E., & Charman, T. (2007). The social communication assessment for toddlers with autism (SCATA): An instrument to measure the frequency, form and function of communication in toddlers with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37, 648–666.
- Ogletree, B. T., Pierce, K., Hard, W. E., & Fischer, M. A. (2001–2002). Assessment of communication and language in classical autism: Issues and practices. *Hammill Institute on Disabilities*, 27(1&2), 61–71.
- Paul, R. (2007). *Language disorders from infancy through adolescence: Assessment and intervention* (3rd ed.). St. Louis: Mosby Elsevier.
- Prelock, P. A. (2006). *Communication assessment and intervention in Autism Spectrum Disorders*. Austin: Pro-Ed Publishers.
- Prelock, P., Gulbranson, M., Hutchins, T., Green, E., & Glascoe, F. (2013). Psychosocial risk, language development, and bilingual/dual language learners. In F. P. Glascoe, K. P. Marks, J. K. Poon, & M. M. Macias (Eds.), *Identifying & addressing developmental-behavioral problems: A practical guide for medical and non-medical professionals, trainees, researchers and advocates* (pp. 199–234). Nolensville: PEDStest.com, LLC.
- Schwartz, I. S., Boulware, G. L., McBride, B. J., & Sandall, S. R. (2001). Functional assessment strategies for young children with autism. *Focus on Autism and Other Developmental Disabilities*, 16(4), 222–227.
- Wetherby, A. M., & Prizant, B. M. (1999). Enhancing language and communication development in autism: Assessment and intervention guidelines. In D. B. Zager (Ed.), *Autism: Identification, education, and treatment* (2nd ed., pp. 141–174). Mahwah: Lawrence Erlbaum.
- Wetherby, A. M., Prizant, B. M., & Schuler, A. L. (2000). Understanding the nature of communication and language impairments. In A. M. Wetherby & B. M. Prizant (Eds.), *Autism spectrum disorders: A transactional developmental perspective* (pp. 109–141). Baltimore: Brookes.

Communication Board

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Synonyms

[Assistive devices](#); [Augmentative and alternative communication \(AAC\) device](#); [No-tech communication device](#); [Picture board](#)

Definition

A communication board is a low-tech communication aid that is used in the field of augmentative and alternative communication (AAC). Typically, a grid with pictures on it is created, printed, and then affixed to something like a sturdy manila envelope. This type of assistive communication device is best for individuals with limited verbal output who are either preliterate or nonliterate. The communication board user will point to pictures to communicate wants and needs, to make comments, or to ask questions. See Beukelman and Mirenda (2012) for a detailed description of no- and low-tech communication devices and their implementation.

See Also

- ▶ [Alternative Communication](#)
- ▶ [Assistive Devices](#)
- ▶ [Voice Output Communication Aids](#)

References and Reading

Beukelman, D. R., & Mirenda, P. (2012). *Augmentative and alternative communication: Supporting children and adults with complex communication needs*. Baltimore: Brooks Publishing.

Communication Disorder

- ▶ [Speech/Communication Disabilities](#)

Communication Disorder/ Communication Impairment

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Synonyms

[Communication impairments](#)

Short Description or Definition

A communication disorder is a developmental or acquired impairment which generally affects language, speech, and/or hearing (National Institute on Deafness and Other Communication Disorders [NIDCD] 2010). The American Speech-Language-Hearing Association (ASHA) (1993) describes communication disorders more specifically, as impacting one's ability to "receive, send, process, and comprehend concepts or verbal, non-verbal and graphic symbol systems (p. 2)." These impairments may include, but are not limited to, problems with fluency, articulation, phonology, voice, auditory processing, pragmatics, syntax, semantics, morphology, and hearing loss (American Speech-Language and Hearing Association [ASHA] 2008; NIDCD 2010; Rochester Hearing and Speech Center [RHSC] 2011). Communication disorders generally fall on a continuum of severity, ranging from relatively mild to profound depending on the complexity of the impairment and which processes of communication are affected (ASHA 1993). Individuals may present with a single communication disorder or a

combination of various communication disorders. Depending on the nature of each individual’s impairment, a communication disorder may be a primary disability or it may be secondary to other disabilities (ASHA 1993). A communication disorder or impairment is one of the core deficits identified for individuals with an autism spectrum disorder (American Psychiatric Association 2000).

Categorization

Rather than being a disorder that can be categorized into a group with other like disorders, communication disorder is a category unto itself. Communication disorders include impairments associated with speech, language, and/or hearing, as outlined in Table 1.

Epidemiology

Given the complexity of communication disorders and the endless possibilities for presentations, it is difficult to identify a specific cause or origin. A wide variety of epidemiological studies have been conducted on the topic of communication disorders, and as the prevalence of related disorders increases, the interest in identifying the factors associated with such disorders continues to expand. Recent prevalence rates found that of the 6,068,802 children in the United States being served in public schools under the Individuals with Disabilities Act (IDEA), 1,460,583 (24.1%) are receiving services for speech and/or language disorders (ASHA 2008). It is important to consider that this statistic does not account for children receiving speech and/or language services

secondary to another disability such as autism spectrum disorders (ASD), children who do not qualify for services through the IDEA, for children who have not yet been identified, or children outside of the United States. As with many disorders and diseases, the number of individuals identified and diagnosed with communication disorders continues to grow with increasing knowledge, awareness, and skills of professionals and the general public.

It should be noted that for children to qualify for an autism diagnosis, they must exhibit a qualitative impairment in communication which may be manifested by one or more of the following: (1) delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime); (2) in individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others; (3) stereotyped and repetitive use of language or idiosyncratic language; and/or (4) lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level (American Psychiatric Association 2000). Therefore, all children with autism will have communication disorders of varying degrees that affect their ability to understand, produce, and/or use communication in an effective and efficient manner.

Natural History, Prognostic Factors, and Outcomes

Identification of communication disorders in children can be especially complex, as symptoms, particularly those related to speech and language, do not typically present in the early months of life.

Communication Disorder/Communication Impairment, Table 1 Communication disorders

Speech	Language	Hearing
Articulation disorders	Receptive language disorder	Sensorineural hearing loss
Fluency disorders	Expressive language disorder	Conductive hearing loss
Voice disorders	Mixed receptive-expressive language disorder	Mixed hearing loss
	Specific language impairment	

ASHA (2008), NIDCD (2010), RHSC (2011)

Furthermore, disorder characteristics vary with each unique child (Ogletree et al. 2002). That being said, research has acknowledged factors that may play a role in determining prognosis, including family history of communication disorders or learning impairments, low socioeconomic status (SES), as well as familial hardships, such as single-parent households or parental substance abuse (Johnson et al. 2010). Further addressing issues of prognosis, research indicates that individuals who had early language impairments are more likely to experience unfavorable adult outcomes than those who had early speech impairments (Johnson et al. 2010).

Studies addressing outcome of communication disorders intervention have been mixed, in part due to the breadth of the topic. However, with improved identification and treatment techniques, therapy outcomes appear to be relatively positive, especially for those who were identified early and received intervention (ASHA 2008). While intervention for communication disorders often yields improvements, occasionally even complete remediation, it is essential to consider the additional confounding factors that may inhibit the treatment of individuals with conditions such as autism. Given that autism falls on a spectrum and each person experiences varying levels of cognitive, linguistic, social, and behavioral functioning, it is difficult to make a definitive statement regarding outcome (Howlin et al. 2004).

Clinical Expression and Pathophysiology

Communication disorder is an incredibly broad category, including multiple sub-disorders related to speech, language, and/or hearing. That being said, there are many clinical features that have been identified to facilitate early detection, diagnosis, and treatment (ASHA 2008; Diehl 2003; Drew et al. 2007). Speech disorders can be characterized by atypical articulation of speech sounds, impaired fluency of speech, as well as impaired voice production and/or quality (ASHA 2008). Language disorders can present in various forms but are typically known to affect the comprehension or use of an individual's language in

both verbal and written forms. Within the category of language disorders, individuals can experience difficulties with the form of language, including phonology, morphology, and syntax, the content of language, otherwise known as semantics, as well as the function of language, which is often referred to as pragmatics. Disorders associated with hearing include varying degrees of hearing loss, which can affect a person's ability to detect, comprehend, or discriminate the sounds of speech (ASHA 2008). As indicated above, communication disorders can present with any combination of impairments associated with speech, language, and/or hearing. In consideration of the various presentations of communication disorders as well as the importance of early identification and treatment, it is important for parents and caregivers who suspect a communication disorder to seek the guidance and support of a certified speech-language pathologist. Such a clinical professional can then determine if an evaluation and later treatment is warranted (ASHA 2008).

Evaluation and Differential Diagnosis

As with most disorders, early and accurate identification and diagnosis are essential components in effective intervention of communication disorders. Furthermore, since children with autism have difficulties with one or multiple components of communication, early assessment of communication skills is often necessary to appropriately address the needs of these children and ensure future communicative success. The goal of communication assessment is to evaluate a child's current level of communicative skills across various settings and with different partners (Ogletree et al. 2002). Assessments utilized in the evaluation process can be standardized or non-standardized tools. Standardized measures, often in the form of tests, require, as the name implies, standard administration and scoring to ensure consistent and accurate interpretation of results. Non-standardized measures, such as observations and caregiver interviews, as well as some tests, allows a clinician to obtain information in a flexible and sometimes more functional manner.

Ideally, both standardized and non-standardized data are obtained throughout the assessment process and can be used to plan the most effective and functional course of treatment, given each individual's needs and abilities (Ogletree et al.; Wetherby and Prizant 1999). There are a great deal of components considered in the evaluation process that allow for identification of disorders as well as differential diagnosis. Some components of communication that are often assessed are ability to engage, attention skills, nonverbal behaviors, and gestures, as well as form, content, and use of speech and language.

Treatment

Equally as significant as early identification, early intervention is an important consideration when addressing communication skills. Accounting for the individualized presentation of each child with autism and given the varying levels of skills and abilities, communication intervention should be based on information obtained throughout the assessment process (Ogletree et al. 2002). Communication intervention often seeks to improve skills associated with the form, function, and content of the communicative act, as well as engagement and attention skills, nonverbal behaviors, and gestures (Paul 2008). As communication skills fall on a continuum, there are a plethora of different intervention approaches available. Generally, clinicians should choose an intervention method which is supported with evidence-based research while also fulfilling the personal and clinical needs of the child and their family. Such approaches range from behavioral techniques, which target the functional and social behaviors related to communication, to augmentative and alternative communication aids, which utilize both high- and low-tech aids to facilitate communication (Diehl 2003; Paul 2008).

See Also

- Expressive Language
- Expressive Language Disorder

- Language Disorder
- Pragmatic Communication
- Pragmatic Language Impairment
- Receptive Language Disorders
- Semantic Pragmatic Disorder
- Social Communication
- Speech Impairments
- Speech/Communication Disabilities
- Verbal Communication

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (Test Rev. 4th ed.). Washington, DC: Author.
- American Speech-Language and Hearing Association. (1988). *Definitions of communication disorders and variation* (Relevant paper). Available from www.asha.org/policy
- American Speech-Language and Hearing Association. (1993). *Prevention of communication disorders* (Position statement). Available from www.asha.org/policy
- American Speech-Language and Hearing Association. (2008). *Incidence and prevalence of communication disorders and hearing loss in children*. Available from www.asha.org/research/reports/children
- Diehl, S. F. (2003). Autism spectrum disorder: The context of speech-language pathologist intervention. *Language, Speech, and Hearing Services in Schools*, 34, 177–179.
- Drew, A., Baird, G., Taylor, E., Milne, E., & Charman, T. (2007). The social communication assessment for toddlers with autism (SCATA): An instrument to measure the frequency, form and function of communication in toddlers with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37, 648–666.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Psychology and Psychiatry*, 45(2), 212–229.
- Johnson, C. J., Beitchman, J. H., & Brownlie, E. B. (2010). Twenty-year follow-up of children with and without speech-language impairments: Family, educational, occupational, and quality of life outcomes. *American Journal of Speech-Language Pathology*, 19(1), 51–65.
- National Institute on Deafness and Other Communication Disorders. (2010). *Examples of communication disorders*. Available from www.nidcd.nih.gov/health/voice/speechandlanguage
- Ogletree, B. T., Pierce, K., Harn, W. E., & Fischer, M. A. (2002). Assessment of communication and language in classical autism: Issues and practices. *Assessment for Effective Intervention*, 27(1&2), 61–71.
- Paul, R. (2008). Interventions to improve communication. *Child and Adolescent Psychiatric Clinics of North America*, 17(4), 835–854.

- Rochester Hearing and Speech Center. (2011). *Types of communication disorders: Facts and figures*. Available from <http://www.rhsc.org/files/communication-disorders>
- Wetherby, A. M., & Prizant, B. M. (1999). Enhancing language and communication development in autism: Assessment and intervention guidelines. In D. B. Zager (Ed.), *Autism: Identification, education, and treatment* (2nd ed., pp. 141–174). Mahwah: Lawrence Erlbaum.
- Wrong Diagnosis. (2011). *Prevalence statistics for types of communication disorders*. Available from www.wrongdiagnosis.com/c/communication_disorders/prevalence-types

Communication Impairments

- [Communication Disorder/Communication Impairment](#)

Communication Intention Inventory

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Synonyms

[CII](#)

Description

The *communicative intention inventory* (CII) is an observational system for recording children's early intentional communication, including gestural, vocal, and verbal communicative behaviors, while the child is engaged in a free-play situation with a caregiver.

The CII is designed to support the clinician in observing and coding the intentional communication of children functioning at the 8 to 24-month developmental level. Intentional communication is defined as a child's goal-oriented, purposeful reasons for vocalizing, gesturing, and speaking. Often, particularly in

the preverbal period, children's intentions must be deduced by their behaviors in relation to objects and people. It is these preverbal intentions, and their expression in the use and coordination of gestures, vocalization, and gaze, that serve as the communicative foundation for the emergence of language.

Historical Background

When Drs. Truman Coggins and Robert Carpenter of the University of Washington first introduced the *communicative intention inventory* (CII) in 1981, the assessments available for describing child's communicative intent were limited. The existing measures at that time included a small number of intentional categories used for describing children's behaviors, inadequately described the categories presented, and provided no information to support the reliability of the categories. The authors therefore attempted to develop the CII with good content and construct validity (i.e., with categories that are believed to adequately reflect instances of early intentional communication and the range of communicative functions involved in it).

The *eight intentional categories* included in the CII are:

1. Comment on action
2. Comment on object
3. Request for action
4. Request for object
5. Request for information
6. Answering a request for information
7. Acknowledging that a previous gesture or utterance was received
8. Protesting

Administration

The authors suggest that the free-play session with child and caregiver should be videotaped through a two-way mirror and monitored in a control room by an individual familiar with the CII. Although this is a criterion-referenced measure, the authors indicated that the recording should be approximately 45 minutes in length if the clinician

would like to do comparisons against the preliminary data they reported for the measure. Stimulus materials should include a variety of toys and books that allow the child to create a variety of activities. No other distracting material should be in the assessment room, and only the child and caregiver should be present during the recording. A familiar caregiver is believed to support obtaining a representative sample of the child's communicative functioning regardless of the familiarity of the testing environment. A written script is included to be read to the caregiver prior to beginning the recording. The purpose of the script is to inform the caregiver of the reason for recording the session and to encourage them to follow the child's lead while refraining from interacting minimally or asking too many questions.

Scoring

The authors of the CII suggest that the video be scored as soon as possible and ideally by the person who monitored the recording. A training procedure for all scorers is also outlined to minimize variability in scorer coding. Only intentionally communicative behaviors are to be coded. Therefore, all coded behaviors must occur in a shared or joint activity. Criteria for what constitutes a joint activity are also described. Each instance of an intentionally communicative behavior is assigned to a single category and given a score of 1. Only those behaviors that fit into one of the CII's eight intentional categories can be scored. Specifics on how to handle repetitions of gestures, vocalizations, and verbalizations are also addressed.

Psychometric Data

The *communicative intention inventory* (CII) was designed as a criterion-referenced measure of the intentional communication of a child and thus compares each child's performance in comparison to a standard or objective, rather than a normative sample. The eight intentional categories included were selected based on their content and construct validity. The CII includes

categories that are believed to adequately reflect instances of early intentional communication and the range of communicative functions involved in it based on the existing literature in early intentional communication. The categories included are believed to be necessary for the acquisition of future conversational discourse skills.

The reported internal test reliability was 0.66, based on a sample of 16 monolingual, native English-speaking typically developing children between 15 and 16 months of age in Piaget's sensorimotor stage V. The authors believe the score was affected by the homogeneity of the participants. Percentile ranks and standard deviation scores were also provided for frequency of demonstration of each of the eight intentional categories in the same sample group.

In terms of inter-scorer reliability, 10 graduate students in speech-language pathology programs (who underwent CII's outlined scorer training procedure and independently scored a recording of 33 behavioral sequences) received a mean proportion of correct coding of behavioral sequences score of 91. All judges also coded all four behavioral sequences that did not demonstrate communicative intent as "unscorable."

Content validity was established based on the creation of the inventory format and behavioral definitions founded on existing early communicative development research studies, language acquisition literature, and the authors' observations of parent-child interactions. When this measure was initially published in 1981, specific studies were not undertaken to establish construct validity though the authors believed that indirect evidence (i.e., the existing literature on social development in children with autism spectrum disorder, the literature on onset of intentionality in children with typical development, and hypotheses regarding children's performance on the CII) supports the CII's utility for assessing intentional communication behaviors. The authors published a study 2 years later noting no significant difference in the performance of children with Down syndrome and children with typical development on the CII.

Clinical Uses

The *communicative intention inventory* (CII) allows the clinician to describe a child's early communicative behaviors across eight intentional categories. Communicative intent is believed to form the basis of later social interaction, making the CII a potentially useful tool in determining intervention goals and predicting future social functioning based on a child's performance on the measure. The CII was specifically designed to observe and code the intentional communication of children functioning between Piaget's sensorimotor stages IV and VI. As research in language acquisition supports the idea that nonverbal expression of early communicative intentions precede and may support the development of future conversational skills, the authors suggest that the CII might also be useful with nonverbal children with and without cognitive delays both as an initial assessment tool and a measure of response to therapy.

These eight intentional behaviors (i.e., comment on action, comment on object, request for action, request for object, request for information, answering, acknowledging, and protesting) were selected based on their frequency of use among children in the sensorimotor stage, their likelihood to occur in a clinical setting, and the ability to develop an operational definition for them. Within each intentional category, descriptions of some expected behaviors are presented within subcategories (i.e., "gesture or gestural/vocal" and "verbal") to indicate how the intention was coded. For example, for the *Comment on Object* category, "points to, looks toward, or approaches entity may vocalize" (gestural or gestural/vocal) and "extends arm to show entity in hand and produces a word" (verbal) are both included as specific behaviors that a child may exhibit. General descriptions of the criteria for inclusion within the subcategories of "gesture or gestural/vocal" and "verbal," and samples of parent-child interactions for classification practice are also provided.

The CII allows the clinician to determine which of the eight intentional categories are present and the manner (i.e., gestural, vocal,

or verbal) in which they are expressed in order to determine if a child's language difficulties are specifically associated with their ability to use language effectively. The authors suggest that further analysis may be done regarding other aspects of language, such as productivity, by applying information from existing language acquisition literature. The authors remind the clinician that the percentile ranks and standard deviations reported for the 16 children with typically development are not intended to be used as referenced norms but are simply provided to describe the behavior patterns in that particular group and aid the clinician's interpretations.

See Also

- Communicative Functions
- Communicative Intent
- Intentional Communication
- Piagetian Stages
- Pragmatic Communication
- Pragmatic Language Impairment
- Pragmatics

References and Reading

- Adams, C. (2002). Practitioner review: The assessment of language pragmatics. *Journal of Child Psychology and Psychiatry*, 43, 973–987. <https://doi.org/10.1111/1469-7610.00226>.
- Adams, C., Green, J., Gilchrist, A., & Cox, A. (2002). Conversational behaviour of children with Asperger syndrome and conduct disorder. *Journal of Child Psychology and Psychiatry*, 43, 679–690.
- Austin, J. L. (1962). *How to do things with words*. New York: Oxford University Press.
- Bates, E., Benigni, T., Bretherton, I., Camaioni, D., & Volterra, V. (1979). *The emergence of symbols: Cognition and communication in infancy*. New York: Academic.
- Carpenter, R. L., Mastergeorge, A. M., & Coggins, T. E. (1983). The acquisition of communicative intentions in infants eight to fifteen months of age. *Language and Speech*, 26(2), 101–116.
- Coggins, T., & Carpenter, R. (1981). The communicative intention inventory: A system for observing and coding children's early intentional communication. *Applied PsychoLinguistics*, 2, 235–251.

- Coggins, T., Carpenter, R., & Owings, N. O. (1983). Examining early intentional communication in Down's syndrome and nonretarded children. *International Journal of Language & Communication Disorders*, 18(2), 98–106.
- Creaghead, N. (1984). Strategies for evaluating and targeting pragmatic behaviours in young children. *Seminars in Speech and Language*, 5, 241–252.
- Dore, J. (1979). Conversational acts and the acquisition of language. In E. Ochs & B. Schiefflin (Eds.), *Developmental pragmatics*. New York: Academic.
- Fey, M. E. (1986). *Language intervention with young children*. Boston: College Hill.
- Halliday, M. (1975). *Learning how to mean*. London: Edward Arnold.
- Klecan-Aker, J. S., & Lopez, B. (1984). A clinical taxonomy for the categorization of pragmatic language functions in normal pre-school children. *Journal of Communication Disorders*, 17, 121–131.
- Klecan-Aker, J. S., & Swank, P. (1988). The use of a pragmatic protocol with normal pre-school children. *Journal of Communication Disorders*, 21, 85–102.
- Ninio, A., Snow, C. E., Pan, B. A., & Rollins, P. (1994). Classifying communicative acts in children's interactions. *Journal of Communication Disorders*, 27, 157–188.
- Olswang, T. E., Coggins, L. B., & Guthrie, J. (1987). Assessing communicative intents in young children: Low structured observation or elicitation tasks? *The Journal of Speech and Hearing Disorders*, 52, 44–49.
- Paul, R., & Shiffer, M. E. (1991). Communicative initiations in normal and late-talking toddlers. *Applied PsychoLinguistics*, 12(04), 419–431.
- Roth, F., & Spekman, N. (1984). Assessing the pragmatic abilities of children: Part I. Organizational framework and assessment parameters. *The Journal of Speech and Hearing Disorders*, 49, 2–11.
- Wetherby, A. M., & Prizant, B. M. (1989). The expression of communicative intent: Assessment guidelines. In *Seminars in speech and language* (Vol. 10, No. 01, pp. 77–91). © 1989 by Thieme Medical Publishers.
- Wetherby, A. M., & Rodriguez, G. P. (1992). Measurement of communicative intentions in normally developing children during structured and unstructured contexts. *Journal of Speech, Language, and Hearing Research*, 35(1), 130–138.
- Wetherby, A. M., Cain, D. H., Yonclas, D. G., & Walker, V. G. (1988). Analysis of intentional communication of normal children from the prelinguistic to the multiword stage. *Journal of Speech, Language, and Hearing Research*, 31(2), 240–252.

Communication Intentions

► Communicative Functions

Communication Interventions

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Definition

Early identification and intervention are key factors when addressing communication skills. Upon completion of a comprehensive communication assessment, it is important to address any communication deficits identified. As each child with autism presents with varying levels of skills and abilities, communication intervention should be based on individualized information obtained throughout the assessment process. Communication intervention should focus on improving skills associated with the form, function, and content of the communicative act, as well as increasing engagement and attention skills, nonverbal behaviors, and gestures. Given that communication skills often fall on a continuum, there are a variety of different intervention approaches available, each of which originates from one of many philosophies. Such philosophies range from behavioral techniques that target the functional and social behaviors related to communication to augmentative and alternative communication aids, which utilize both high- and low-tech aids to supplement communication, to naturalistic approaches that seek to foster communication in the natural environment, including home, school, the job setting, and community-based activities.

Children's language and communication abilities are best facilitated through the relationships they establish with family members and their interventionists as well as a result of environmental changes and communication partner development (Prelock 2006). It is important that communication interventions address the core deficits that characterize children with autism spectrum disorders (ASD), including those that can facilitate communication in play and social interaction.

Notably, many communication intervention strategies are rooted in behavioral principles and strategies such as prompting, modeling, and reinforcement (Brown and Murray 2002). Most speech-language pathologists, however, employ social-pragmatic developmental approaches to address the primary social-pragmatic deficits that characterize children with ASD.

Because communication is such a critical skill for learning and connecting with others, children with ASD who have minimal verbal skills are often limited in their ability to participate in play and socialize with adults or peers (Prelock 2006). As a result, some children engage in less conventional ways to communicate their intent, often substituting undesirable behaviors to communicate their needs. Speech-language pathologists have a critical role in addressing the communication needs of children with ASD who exhibit limited verbal abilities. Some of the intervention approaches they might select include but are not limited to joint action routines, minimal speech approach, proximal communication, prelinguistic milieu teaching, augmentative communication, functional communication training, visual supports, parent training, More Than Words™, and the Picture Exchange Communication System™.

As children with ASD begin to develop their oral communication and can communicate verbally, they are likely to require additional support to use their verbal communication skills in the most effective manner to learn and interact across a variety of contexts and with various communication partners. For children with ASD who are verbal, speech-language pathologists might employ interventions such as enhanced milieu teaching, time delay, pivotal response training, modeling, video modeling and video self-modeling, scripting, Talkability™, Social Stories™, and comic strip conversations (Prelock 2006).

Importantly, communication interventions for children with ASD can be used to help manage unconventional verbal behaviors as well as challenging behaviors since behavior is often a child's attempt to communicate. Although not directly related to communication interventions, approximately 52 % of parents of children with ASD have used at least one complementary and

alternative medical (CAM) therapy with their child with ASD to address behavioral and communication challenges (Wong and Smith 2006). Most of the interventions families select are biologically based (e.g., special diets or supplements), and at least 75 % felt the therapies were beneficial (Wong and Smith 2006). CAM interventions, however, have not been widely investigated as a strategy to support communication in children with ASD.

The selection of any communication intervention must consider the context, the available evidence, the child's relative communication strengths and challenges, and the child's communication goals (Prelock and McCauley 2012). Several intervention reviews guide clinicians and families in their pursuit of the most appropriate intervention approaches (for more information, see reports from the Agency for Healthcare Research and Quality 2014; National Autism Center 2009, 2015; Stansberry-Brusnahan and Collet-Klinenberg 2010; Wong et al. 2014).

See Also

- ▶ [Consequence-Based Interventions](#)
- ▶ [Developmental Intervention Model](#)
- ▶ [Developmental-Pragmatic Approaches/Strategies](#)
- ▶ [Early Intensive Behavioral Intervention \(EIBI\)](#)
- ▶ [Early Intervention](#)
- ▶ [Functional Ecological Approach](#)
- ▶ [Hanen Approach](#)
- ▶ [Home-Based Programs](#)
- ▶ [Language Interventions](#)
- ▶ [Natural Environment](#)
- ▶ [Self-Management Interventions](#)
- ▶ [Social Interventions](#)
- ▶ [Speech Therapy](#)
- ▶ [Structured Behavioral Interventions](#)

References and Reading

- Agency for Healthcare Research and Quality. (2014). Therapies for children with autism spectrum disorders: Behavioral interventions update. *Comparative Effectiveness Review*, 137, 1–519.

- Brown, J., & Murray, D. (2002). Communication-based behavioral interventions for children with autism spectrum disorder. *Perspectives on Language Learning and Education*, 9(2), 8–13.
- Diehl, S. F. (2003). Autism spectrum disorder: The context of speech-language pathologist intervention. *Language, Speech, and Hearing Services in Schools*, 34, 177–179.
- National Autism Center. (2009). *National Standards Project, Phase 1: Addressing the need for evidence-based practice guidelines for ASD*. www.nationalautismcenter.org.
- National Autism Center. (2015). *National Standards Project, Phase 2: Addressing the need for evidence-based practice guidelines for ASD*. www.nationalautismcenter.org.
- Ogletree, B. T., Pierce, K., Harn, W. E., & Fischer, M. A. (2002). Assessment of communication and language in classical autism: Issues and practices. *Assessment for Effective Intervention*, 27(1&2), 61–71.
- Paul, R. (2008). Interventions to improve communication. *Child and Adolescent Psychiatric Clinics of North America*, 17(4), 835–854.
- Prelock, P. A. (2006). *Communication assessment and intervention in autism spectrum disorders*. Austin: Pro-Ed Publishers.
- Prelock, P. A., & McCauley, R. J. (2012). *Treatment of autism spectrum disorders: Evidence-based intervention strategies for communication and social interaction*. Baltimore: Paul H. Brookes.
- Stansberry-Brusnahan, L. L., & Collet-Klinenberg, L. L. (2010). Evidence-based practices for young children with autism spectrum disorders: Guidelines & recommendations from the NRC & the National Professional Development Center on ASD. *International Journal of Early Childhood Special Education*, 2(1), 45–56.
- Wong, H. H. L., & Smith, R. G. (2006). Patterns of complementary and alternative medical therapy use in children diagnosed with ASD. *Journal of Autism and Developmental Disorders*, 36(7), 901–909.
- Wong, C., Odom, S. L., Hume, K., Cox, A. W., Fettig, A., Kucharczyk, S., et al. (2014). *Evidence-based practices for children, youth, and young adults with autism spectrum disorder*. Chapel Hill: The University of North Carolina, Frank Porter Graham Child Development Institute, Autism Evidence-Based Practice Review Group.

Communication Services

► Speech-Language Intervention

Communicative Acquisition in ASD

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Short Description or Definition

The development of communicative skills begins in the earliest months of life. Communication can be conceived as any action that conveys information to establish a shared understanding with another individual. It is comprised of a complex set of skills, including language, as well as a number of nonverbal behaviors like gestures, facial expressions, and body posture. Communication – whether verbally or nonverbally expressed – has long been understood to be an area of core impairment in ASD, and research has uncovered deficits in communication development within the first year of life for children who go on to develop ASD. These difficulties are usually persistent, continuing to affect both verbal and nonverbal aspects of communication throughout the lifespan.

Natural History, Prognostic Factors, and Outcomes

Prior to the development of language, typically developing infants make a number of communicative achievements starting only months after birth. They attend preferentially to their caregivers' face (Bushnell 2001) and speech (DeCasper and Fifer 1980), and by 3 months of age, infants smile reciprocally (Emde et al. 1976). By the end of the first year, they produce early speech sounds and perhaps even single words, use gestures to direct others' attention, and follow social cues conveyed in movements and gaze (Fenson et al. 1994).

In contrast, children who are later diagnosed with ASD show broad deficits in early communication. Within the first year of life, children with ASD show reduced social smiling and reciprocal engagement with partners (Zwaigenbaum et al. 2005). Unlike typically developing toddlers, young children with ASD do not preferentially attend to child-directed speech (Kuhl et al. 2005; Paul et al. 2007), instead preferring to listen to nonspeech analog signal. They show delays and deficits in the emergence of early gestures, joint attention, and imitation (Mitchell et al. 2006; Shumway and Wetherby 2009; Wetherby et al. 2007). These skills are all critical developmental precursors to language, and deficits in these non-verbal abilities are associated with deficits in verbal competence (Luyster et al. 2008).

One of the earliest forms of vocal communication in infants is crying, and infants who go on to receive an early ASD diagnosis have cries that are qualitatively different than controls, showing more dysphonation and less modulation (Esposito and Venuti 2009). When the first speech sounds emerge for young children, they are in the form of canonical babbling. In infants and toddlers later identified with ASD, these sounds may have unusual vocal qualities (Schoen et al. 2011; Sheinkopf et al. 2000) or have a restricted consonant range (Wetherby et al. 2004). By around 1 year of age, children with ASD are already falling behind their unaffected peers in receptive and expressive language development, showing delays in the attainment of first words (Landa and Garrett-Mayer 2006; Mitchell et al. 2006; Zwaigenbaum et al. 2005). Receptive language skills are particularly impaired starting in the first few years of life (Hudry et al. 2010; Weismer et al. 2010) and continuing into childhood.

The process of learning words is driven, at least in part, by social engagement and attention (Baldwin et al. 2011; Baldwin and Moses 2001; Tomasello and Barton 1994). Longitudinal studies have suggested that slowed development in language – especially receptive – for children with ASD is partly a product of their overall difficulty with social engagement (Bopp et al.

2009). Indeed, the aforementioned tendency of children with ASD to direct their attention more toward nonspeech sounds than to child-directed speech is associated with lower expressive language development (Kuhl et al. 2005). Similarly, children with ASD who showed greater physiological responsiveness to child-directed speech at an initial evaluation exhibited better communication skills 1 year later (Watson et al. 2010), pointing to a critical link between social attention and communicative development.

The question of how children with ASD, who experience deficits in attending to social input, learn new words has been addressed in a variety of studies. Results generally suggest that children who are able to attend to the social cues of others are able to use those cues to learn new words (Franken et al. 2010; Luyster and Lord 2009; Parish-Morris et al. 2007); the children who experience more profound deficits in social attention may not be able to successfully extract these cues for the purpose of language learning (Baron-Cohen et al. 1997; Preissler and Carey 2005). The importance of relatedness with social partners for language development has also been reported by studies finding an association between parental responsiveness to child interest and child language (McDuffie and Yoder 2010; Siller and Sigman 2008).

A number of other cognitive biases have been proposed as important mechanisms for learning language, including the “noun bias” (by which children map a novel word onto an unknown object rather than an action or feature) and the “shape bias” (which supports children in generalizing words based on similarity of shape). Young children with ASD seem to abide by the former, accurately mapping new words onto objects rather than some other aspect of the visual scene (Swensen et al. 2007). However, they are less adept at following the shape bias, failing to extend terms to same-shaped objects (Tek et al. 2008).

Patterns of change over time in communication development have been a focus of recent research. In general, children with ASD show improvement in language and communication with age

(Ballaban-Gil et al. 1996; Lord et al. 2004a; Paul et al. 2008). However, a minority of children may experience a period of loss early in life. This phenomenon – called “regression” – has been reported in several retrospective and early home video studies (Baird et al. 2008; Hansen et al. 2008; Lord et al. 2004b; Werner and Dawson 2005). The initial portrait of this developmental shift was a sudden-onset loss that primarily affected language skills and occurred around the second birthday. Conceptualizations have gradually broadened to reflect the wider range of social communication skills that seemed to be affected, including reciprocal engagement, social attention, and shared enjoyment (Baird et al. 2008; Luyster et al. 2005). Interestingly, prospective studies have also uncovered evidence for loss of skills, but the characterization of this shift is somewhat different from the retrospective and video review reports. Ozonoff and colleagues noted a gradual deterioration in social communication skills between 6 and 18 months of age (2010), and a similar pattern has been observed elsewhere (Bryson et al. 2007).

The understanding and use of language require the mastery of a complex set of sounds and rules, and individuals with ASD have been found to experience difficulties at nearly every level of language development. It is important to note that, although all individuals with ASD (by definition) experience some sort of impairment in language and communication, any specific area of difficulty is not universal to the conditions. Despite these difficulties, more than two-thirds of individuals with ASD eventually acquire spoken language (Anderson et al. 2007; Turner et al. 2006) though it can range from single words to complex, fluent speech. Early emergence of language (particularly by age 3) is a positive predictor of a number of outcomes (Ben Itzhak and Zachor 2009; Charman et al. 2005). It is associated both concurrently and longitudinally with a variety of skills including joint attention (Adamson et al. 2009; Dawson et al. 2004; Sigman and McGovern 2005), gesture use (Ingersoll and Lalonde 2010; Luyster et al. 2008; Smith et al. 2007), play (Mundy et al. 1987; Sigman and McGovern 2005), imitation

(Carpenter et al. 2002; Charman et al. 2003; Stone et al. 1997), and cognitive skills (Luyster et al. 2008; Thurm et al. 2007). Furthermore, the strength of early verbal skills is positively associated with response to treatment in young children with ASD (Ben Itzhak and Zachor 2011).

Clinical Expression and Pathophysiology

When language is acquired, it is often atypical in a variety of ways. Indeed, some of the first written accounts of the disorder included observations of odd speech patterns (Kanner 1943, 1946). Individuals with ASD may use language without apparent meaning; for instance, they may repeat previously heard words or phrases (termed “echolalia”). This behavior can occur immediately after the child hears a word or phrase, or it may be delayed by several hours or days. The former – “immediate echolalia” – appears to be more common in individuals with limited language (McEvoy et al. 1988). More advanced language users may incorporate chunks of speech heard previously into their speech in a scripted fashion (Nadig et al. 2010). Examples of this could include a phrase spoken by the parent earlier in the day (e.g., “It’s snowing cats and dogs!”) or the introductory sequence to the *Powerpuff Girls* television show. Other individuals may make up words that do not have any conventional meaning (termed “neologisms”) (Volden and Lord 1991). For instance, a child might call all cups “tamots” or referring to anything with stripes as “surry.” In individuals with more advanced language, speech can be idiosyncratic, characterized by overly formal use of words or unusual formation of sentences (Nadig et al. 2010; Paul et al. 2009). For instance, rather than simply saying, “I like reading books,” the individual might use more pedantic phrasing like, “I enjoy engaging in literary endeavors.” Idiosyncratic phrasing could include asking, “How many years are you?” instead of “How old are you?” or referring to rainbows as “color bows.”

There have been some deficits reported in vocabulary acquisition and use, although results are not consistent. On the one hand, vocabulary

knowledge across general categories (e.g., modifiers, nouns, predicates) seems to be indistinguishable from typically developing controls (Charman et al. 2003; Luyster et al. 2007). However, usage of some specific vocabulary types – such as mental state terms (Tager-Flusberg 1992), social-emotional identifiers (Hobson and Lee 1989), or deictic terms (Hobson et al. 2010) – may be impaired in an individual with ASD. An example of this is pronoun reversal, such that the individual uses the term “you” instead of “I” or “me” or refers to himself or herself in the third person (Lee et al. 1994).

The degree to which semantic and lexical organization is disrupted remains unclear. Children with ASD do well on standardized vocabulary tests (Kjelgaard and Tager-Flusberg 2001) and generalize terms in a usual manner. Some studies have reported that individuals with ASD form conceptual categories in a similar fashion to their typically developing peers (Tager-Flusberg 1985), while others reported differences in the connectedness of conceptual and lexical knowledge (Dunn et al. 1996). One possible explanation to this apparent discrepancy is the suggestion that impairment may be specific to certain kinds of conceptual categories (e.g., animate beings; Kelley et al. 2006).

In general, grammar and syntax seem to follow a typical path of development (Tager-Flusberg et al. 1990; Waterhouse and Fein 1982). However, there is some indication that the range of grammatical constructions spontaneously used by individuals with ASD may be limited. Other difficulties have been noted, including morpheme omission and failure to correctly mark tense (Bartolucci et al. 1980; Eigsti and Bennetto 2009; Roberts et al. 2004).

The area of language and communication that is most universally disturbed is pragmatics, or the social use of language. For example, individuals with ASD may use language in restricted ways. That is, whereas language is broadly used for a variety of purposes (making requests, as well as sharing information, conveying interest, or directing attention), individuals with ASD may employ language predominantly to express their own needs, wants, or interests and only minimally

for purely social purposes (Loveland et al. 1988; Wetherby et al. 2007). As a result, maintaining reciprocal conversations is often quite difficult for individuals on the autism spectrum, who experience impairments in turn-taking and using socially appropriate questions and statements (Capps et al. 1998; Loukusa et al. 2007; Paul et al. 2009) (Jones and Schwartz 2009). Difficulty maintaining back-and-forth dialogue may also present as rigidity in language use, sometimes referred to as “verbal rituals.” This pattern of behavior is characterized by a need for a language to follow a certain predictable routine rather than flow naturally. This can consist of a compulsive sequence of utterances spoken by the child – an example might be the need to recite book titles in alphabetical order without interruption – or it could be the desire for an interchange to abide by a particular routine. For instance, the child might have a habit of saying, “Welcome boys and girls! The color of the day is. . .” and insist that the parent answer, “The color of the day is red!” to which the child responds, “And after red is. . .” and expects the parent to answer “After red is orange!” and so on through a series of colors. Any disruption of this specific pattern and phrasing is often distressing for the child, with the focus being on the need for language predictability rather than social usage.

The understanding and use of nonlinguistic communicative cues, such as prosody, gestures, facial expressions, and gaze, commonly present a challenge to individuals with ASD. Understanding and using language that hinges on these nonlinguistic cues (such as humor or irony) are often impaired (MacKay and Shaw 2004; Rundblad and Annaz 2010; Wang et al. 2006) as is the use of a range of nonverbal cues. For instance, the speech of individuals with ASD may sound robotic or monotone; in other cases, it may have exaggerated ups and downs (Paul et al. 2005; Peppé et al. 2011; Shriberg et al. 2001). Vocal atypicalities carry over from speech into laughter, which is restricted in range and variety relative to controls, suggesting that individuals with ASD may not modulate their laughter for different communicative purposes (Hudenko et al. 2009). Difficulties with expressive prosody are accompanied by

deficits in accurately interpreting the significance of others' prosodic cues (Korpilahti et al. 2007; Rutherford et al. 2002).

Individuals with ASD show decreased use of gestures, facial expressions, and gaze in communicative situations whether with or without language use (Garcia-Pérez et al. 2007; Lord et al. 2000; Loveland et al. 1988). Similarly, whereas the perception of gestures facilitates language comprehension for typically developing individuals, individuals with ASD experience a detriment in speech comprehension when the speaker uses concurrent gestures (Silverman et al. 2010), indicating that verbal and nonverbal cues may not be processed as complementary pieces of a unified communicative message.

These observable abnormalities of communication and language are accompanied by atypical structural characteristics of associated brain regions. Volumetric differences in areas underlying social and emotion processing are associated with social communication impairments on behavioral measures (Kim et al. 2011; Mosconi et al. 2009; Parks et al. 2009; Schumann et al. 2009). Similarly, structural differences have emerged in brain regions known to be associated with language processing (McAlonan et al. 2005; Rojas et al. 2006), and some of these structural differences are associated with variability in language for children with ASD (De Fossé et al. 2004; Knaus et al. 2009).

Functional investigations have yielded very promising findings as well. Differences in activation have been noted in regions serving non-linguistic communicative cues, including the amygdala, left temporal lobe, and prefrontal cortex (Critchley et al. 2000; Hesling et al. 2010; Wang et al. 2007). In the case of typical development, language processing is lateralized, occurring predominantly in the left cerebral hemisphere. However, a number of studies have indicated that language processing in individuals with ASD does not follow this pattern. A lack of left hemisphere lateralization has been reported, with profiles of either bilateral or right hemisphere activation being observed instead (Flagg et al. 2005; Kleinhans et al. 2008; Minagawa-Kawai et al. 2009) starting from very early on in life

(Redcay and Courchesne 2008). There seem to be behavioral correlates of these neural differences in that reduced asymmetry is associated with greater language impairment for individuals with ASD (De Fossé et al. 2004; Herbert et al. 2005).

The centrality of communication, whether verbal or nonverbal, to our understanding of ASD ensures that it will remain a primary focus of research and clinical endeavors in the years to come. Strides continue to be made in standardizing definitions and measures (Tager-Flusberg et al. 2010), an important step in unifying efforts and optimizing the applicability of research findings to applied settings.

See Also

- Communication Assessment
- Communication Disorder
- Communicative Functions
- Language
- Language Acquisition
- Language Disorder
- Language Tests
- Speech

References and Reading

- Adamson, L. B., Bakeman, R., Deckner, D. F., & Ronski, M. (2009). Joint engagement and the emergence of language in children with autism and Down syndrome. *Journal of Autism and Developmental Disorders*, 39(1), 84–96. <https://doi.org/10.1007/s10803-008-0601-7>.
- Anderson, D. K., Lord, C., Risi, S., DiLavore, P., Shulman, C., Thurm, A., Welch, K., et al. (2007). Patterns of growth in verbal abilities among children with autism spectrum disorder. *Journal of Consulting and Clinical Psychology*, 75(4), 594–604. <https://doi.org/10.1037/0022-006X.75.4.594>.
- Baird, G., Charman, T., Pickles, A., Chandler, S., Loucas, T., Meldrum, D., et al. (2008). Regression, developmental trajectory and associated problems in disorders in the autism spectrum: The SNAP study. *Journal of Autism and Developmental Disorders*, 38(10), 1827–1836. <https://doi.org/10.1007/s10803-008-0571-9>.
- Baldwin, D. A., & Moses, L. J. (2001). Links between social understanding and early word learning: Challenges to current accounts. *Social Development*, 10(3), 309–329. <https://doi.org/10.1111/1467-9507.00168>.

- Baldwin, D. A., Markman, E. M., Bill, B., Desjardins, R. N., Irwin, J., & Tidball, G. (2011). Infants' reliance on a social criterion for establishing word-object relations. *Child Development*, 67(6), 3135–3153.
- Ballaban-Gil, K., Rapin, I., Tuchman, R., & Shinnar, S. (1996). Longitudinal examination of the behavioral, language, and social changes in a population of adolescents and young adults with autistic disorder. *Pediatric Neurology*, 15(3), 217–223.
- Baron-Cohen, S., Baldwin, D. A., & Crowson, M. (1997). Do children with autism use the speaker's direction of gaze strategy to crack the code of language. *Child Development*, 68(1), 48–57.
- Bartolucci, G., Pierce, S. J., & Streiner, D. L. (1980). Cross-sectional studies of grammatical morphemes in autistic and mentally retarded children. *Journal of Autism and Developmental Disorders*, 10(1), 39–50. Germany: Springer.
- Ben Itzhak, E., & Zachor, D. A. (2009). Change in autism classification with early intervention: Predictors and outcomes. *Research in Autism Spectrum Disorders*, 3(4), 967–976. <https://doi.org/10.1016/j.rasd.2009.05.001>.
- Ben Itzhak, E., & Zachor, D. A. (2011). Who benefits from early intervention in autism spectrum disorders? *Research in Autism Spectrum Disorders*, 5(1), 345–350.
- Bishop, D. V. M. (2009). Genes, cognition and communication: Insights from neurodevelopmental disorders. *Annals of the New York Academy of Sciences*, 1156, 1–18.
- Bopp, K. D., Mirenda, P., & Zumbo, B. D. (2009). Behavior predictors of language development over 2 years in children with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, 52(5), 1106–1120. [https://doi.org/10.1044/1092-4388\(2009/07-0262\)](https://doi.org/10.1044/1092-4388(2009/07-0262)).
- Bryson, S., Zwaigenbaum, L., Brian, J., Roberts, W., Szatmari, P., Rombough, V., & McDermott, C. (2007). A prospective case series of high-risk infants who developed autism. *Journal of Autism and Developmental Disorders*, 37(1), 12–24. United States.
- Bushnell, I. W. R. (2001). Mother's face recognition in newborn infants: Learning and memory. *Infant and Child Development*, 10(1–2), 67–74. <https://doi.org/10.1002/icd.248>.
- Capps, L., Kehres, J., & Sigman, M. (1998). Conversational abilities among children with autism and children with developmental delays. *Autism*, 2(4), 325–344. US: SAGE.
- Carpenter, M., Pennington, B., & Rogers, S. J. (2002). Interrelations among social-cognitive skills in young children with autism. *Journal of Autism and Developmental Disorders*, 32(2), 91–106.
- Charman, T., & Stone, W. (Eds.). (2006). *Social and communication development in autism spectrum disorders: Early identification, diagnosis and intervention*. New York: Guilford Press.
- Charman, T., Drew, A., Baird, C., & Baird, G. (2003). Measuring early language development in preschool children with autism spectrum disorder using the MacArthur Communicative Development Inventory (Infant Form). *Journal Child Language*, 30, 213–236.
- Charman, T., Taylor, E., Drew, A., Cockerill, H., Brown, J.-A., & Baird, G. (2005). Outcome at 7 years of children diagnosed with autism at age 2: Predictive validity of assessments conducted at 2 and 3 years of age and pattern of symptom change over time. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 46(5), 500–513. <https://doi.org/10.1111/j.1469-7610.2004.00377.x>.
- Critchley, H. D., Daly, E. M., Bullmore, E. T., Williams, S. C., Van Amelsvoort, T., Robertson, D. M., et al. (2000). The functional neuroanatomy of social behaviour: Changes in cerebral blood flow when people with autistic disorder process facial expressions. *Brain: A Journal of Neurology*, 123(Pt. 1), 2203–2212.
- Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., et al. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology*, 40(2), 271–283. <https://doi.org/10.1037/0012-1649.40.2.271>.
- De Fossé, L., Hodge, S. M., Makris, N., Kennedy, D. N., Caviness, V. S., McGrath, L., et al. (2004). Language-association cortex asymmetry in autism and specific language impairment. *Annals of Neurology*, 56(6), 757–766. <https://doi.org/10.1002/ana.20275>.
- DeCasper, A., & Fifer, W. (1980). Of human bonding: Newborns prefer their mothers' voices. *Science*, 208(4448), 1174–1176.
- Dunn, M., Gomes, H., & Sebastian, M. (1996). Prototypicality of responses of autistic, language disordered, and normal children in a word fluency task. *Child Neuropsychology*, 2(2), 99–108. United Kingdom: Taylor & Francis.
- Eigsti, I.-M., & Bennetto, L. (2009). Grammaticality judgments in autism: Deviance or delay. *Journal of Child Language*, 36(5), 999–1021. <https://doi.org/10.1017/S0305000909009362>.
- Emde, R. N., Gaensbauer, T. J., & Harmon, R. J. (1976). Emotional expression in infancy: A behavioral study. *Psychological Issues Monograph Series*, 10(1), Serial No. 37.
- Esposito, G., & Venuti, P. (2009). Comparative analysis of crying in children with autism, developmental delays, and typical development. *Focus on Autism and Other Developmental Disabilities*, 24(4), 240–247.
- Fenson, L., Dale, P. S., Reznick, J. S., & Bates, E. (1994). Variability in early communicative development. *Monographs of the Society for Research in Child Development*, 59(5), v-173. US: University of Chicago Press.
- Flagg, E. J., Cardy, J. E. O., Roberts, W., & Roberts, T. P. L. (2005). Language lateralization development in children with autism: Insights from the late field magnetoencephalogram. *Neuroscience Letters*, 386(2), 82–87. <https://doi.org/10.1016/j.neulet.2005.05.037>.

- Franken, T., Lewis, C., & Malone, S. A. (2010). Brief report: Are children with autism proficient word learners? *Journal of Autism and Developmental Disorders*, 40, 255–259. <https://doi.org/10.1007/s10803-009-0847>.
- García-Pérez, R., Lee, A., & Hobson, R. P. (2007). On intersubjective engagement in autism: A controlled study of nonverbal aspects of conversation. *Journal of Autism and Developmental Disorders*, 37(7), 1310–1322. <https://doi.org/10.1007/s10803-006-0276-x>.
- Groen, W. B., Zwiers, M. P., van der Gaag, R. J., & Buitelaar, J. K. (2008). The phenotype and neural correlates of language in autism: An integrative review. *Neuroscience and Biobehavioral Reviews*, 32(8), 1416–1425.
- Hansen, R. L., Ozonoff, S., Krakowiak, P., Angkustsiri, K., Jones, C., Deprey, L. J., et al. (2008). Regression in autism: Prevalence and associated factors in the CHARGE study. *Ambulatory Pediatrics*, 8(1), 13–15.
- Herbert, M. R., Ziegler, D., Deutsch, C. K., O'Brien, L. M., Kennedy, D. N., Filipek, P. A., et al. (2005). Brain asymmetries in autism and developmental language disorder: A nested whole-brain analysis. *Brain: A Journal of Neurology*, 128(Pt. 1), 213–226. <https://doi.org/10.1093/brain/awh330>.
- Hesling, I., Dilharreguy, B., Peppé, S., Amirault, M., Bouvard, M., & Allard, M. (2010). The integration of prosodic speech in high functioning autism: A preliminary fMRI study. *PLoS One*, 5(7), e11571. <https://doi.org/10.1371/journal.pone.0011571>.
- Hobson, R. P., & Lee, A. (1989). Emotion-related and abstract concepts in autistic people: Evidence from the British Picture Vocabulary Scale. *Journal of Autism and Developmental Disorders*, 19(4), 601–623. Germany: Springer.
- Hobson, R. P., García-Pérez, R., & Lee, A. (2010). Person-centred (deictic) expressions and autism. *Journal of Autism and Developmental Disorders*, 40, 403–415.
- Hudenko, W. J., Stone, W., & Bachorowski, J.-A. (2009). Laughter differs in children with autism: An acoustic analysis of laughs produced by children with and without the disorder. *Journal of Autism and Developmental Disorders*, 39(10), 1392–1400. <https://doi.org/10.1007/s10803-009-0752-1>.
- Hudry, K., Leadbitter, K., Temple, K., Slonims, V., McConachie, H., Aldred, C., et al. (2010). Preschoolers with autism show greater impairment in receptive compared with expressive language abilities. *International Journal of Language & Communication Disorders*, 45(6), 681–690. <https://doi.org/10.3109/13682820903461493>.
- Ingersoll, B., & Lalonde, K. (2010). The impact of object and gesture imitation training on language use in children with Autism Spectrum Disorder. *Journal of Speech, Language, and Hearing Research*, 53 (August), 1040–1052.
- Jones, C. D., & Schwartz, I. S. (2009). When asking questions is not enough: An observational study of social communication differences in high functioning children with autism. *Journal of Autism and Developmental Disorders*, 39(3), 432–443. <https://doi.org/10.1007/s10803-008-0642-y>.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kanner, L. (1946). Irrelevant and metaphorical language. *American Journal of Psychiatry*, 103, 242–246.
- Kelley, E., Paul, J. J., Fein, D., & Naigles, L. R. (2006). Residual language deficits in optimal outcome children with a history of autism. *Journal of Autism and Developmental Disorders*, 36(6), 807–828. <https://doi.org/10.1007/s10803-006-0111-4>.
- Kim, J., Lyoo, I., Estes, A., Renshaw, P., Shaw, D., Friedman, S., et al. (2011). Laterobasal amygdalar enlargement in 6- to 7-year-old children with Autism Spectrum Disorder. *Archives of General Psychiatry*, 67(11), 1187–1197.
- Kleinmans, N. M., Müller, R.-A., Cohen, D. N., & Courchesne, E. (2008). Atypical functional lateralization of language in autism spectrum disorders. *Brain Research*, 1221, 115–125. <https://doi.org/10.1016/j.brainres.2008.04.080>.
- Knaus, T. A., Silver, A., Dominick, K. C., Schuring, M., Shaffer, N., Lindgren, K. A., et al. (2009). Age-related changes in the anatomy of language regions in autism spectrum disorder. *Brain Imaging and Behavior*, 3, 51–63.
- Korpilahti, P., Jansson-Verkasalo, E., Mattila, M.-L., Kuusikko, S., Suominen, K., Rytty, S., et al. (2007). Processing of affective speech prosody is impaired in Asperger syndrome. *Journal of Autism and Developmental Disorders*, 37(8), 1539–1549. <https://doi.org/10.1007/s10803-006-0271-2>.
- Kuhl, P. K., Coffey-Corina, S., Padden, D., & Dawson, G. (2005). Links between social and linguistic processing of speech in preschool children with autism: Behavioral and electrophysiological measures. *Developmental Science*, 8(1), F1–F12. <https://doi.org/10.1111/j.1467-7687.2004.00384.x>.
- Landa, R. J., & Garrett-Mayer, E. (2006). Development in infants with autism spectrum disorders: A prospective study. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 47(6), 629–638. <https://doi.org/10.1111/j.1469-7610.2006.01531.x>.
- Lee, A., Hobson, R. P., & Chiat, S. (1994). I, you, me and autism: An experimental study. *Journal of Autism and Developmental Disorders*, 24(2), 155–176.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H. J., Leventhal, B. L., DiLavore, P., et al. (2000). The autism diagnostic observation schedule-generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30(3), 205–223. United States.
- Lord, C., Risi, S., & Pickles, A. (2004a). Trajectory of language development in Autistic Spectrum Disorders. In M. Rice & S. Warren (Eds.), *Developmental language disorders: From phenotypes to etiologies* (pp. 7–29). Mahwah: Lawrence Erlbaum Associates.

- Lord, C., Shulman, C., & DiLavore, P. (2004b). Regression and word loss in autistic spectrum disorders. *Journal of Child Psychology and Psychiatry*, 5(5), 936–955.
- Loukusa, S., Leinonen, E., Jussila, K., Mattila, M.-L., Ryder, N., Ebeling, H., & Moilanen, I. (2007). Answering contextually demanding questions: Pragmatic errors produced by children with Asperger syndrome or high-functioning autism. *Journal of Communication Disorders*, 40(5), 357–379.
- Loveland, K. A., Landry, S. H., Hughes, S. O., & Hall, S. K. (1988). Speech acts and the pragmatic deficits of autism. *Journal of Speech & Hearing Research*, 31(4), 593–604. US: American Speech-Language-Hearing Assn.
- Luyster, R., & Lord, C. (2009). Word learning in children with autism spectrum disorders. *Developmental Psychology*, 45(6), 1774–1786. <https://doi.org/10.1037/a0016223>.
- Luyster, R., Richler, J., Risi, S., Hsu, W.-L., Dawson, G., Bernier, R., Dunn, M., et al. (2005). Early regression in social communication in autism spectrum disorders: A CPEA study. *Developmental Neuropsychology*, 27(3), 311–336. https://doi.org/10.1207/s15326942dn2703_2.
- Luyster, R., Lopez, K., & Lord, C. (2007). Characterizing communicative development in children referred for Autism Spectrum Disorders using the MacArthur-Bates Communicative Development Inventory (CDI). *Journal of Child Language*, 34(03), 623–654. <https://doi.org/10.1017/S0305000907008094>.
- Luyster, R., Kadlec, M., Carter, A., & Tager-Flusberg, H. (2008). Language assessment and development in toddlers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(8), 1426–1438. <https://doi.org/10.1007/s10803-007-0510-1>.
- MacKay, G., & Shaw, A. (2004). A comparative study of figurative language in children with autistic spectrum disorders. *Child Language Teaching and Therapy*, 20(1), 13–32. <https://doi.org/10.1191/0265659004ct2610a>.
- McAlonan, G., Cheung, V., Cheung, C., Suckling, J., Lam, G. Y., Tai, K. S., Yip, L., et al. (2005). Mapping the brain in autism: A voxel-based MRI study of volumetric differences and intercorrelations in autism. *Brain: A Journal of Neurology*, 128(Pt. 2), 268–276. <https://doi.org/10.1093/brain/awh332>.
- McDuffie, A., & Yoder, P. (2010). Types of parent verbal responsiveness that predict language in young children with autism spectrum disorder. *Journal of Speech, Language, and Hearing Research*, 53(4), 1026–1239. [https://doi.org/10.1044/1092-4388\(2009/09-0023\)](https://doi.org/10.1044/1092-4388(2009/09-0023)).
- McEvoy, R. E., Loveland, K. A., & Landry, S. H. (1988). The functions of immediate echolalia in autistic children: A developmental perspective. *Journal of Autism and Developmental Disorders*, 18(4), 657–668. Germany: Springer.
- Minagawa-Kawai, Y., Naoi, N., Kikuchi, N., Yamamoto, J.-I., Nakamura, K., & Kojima, S. (2009). Cerebral laterality for phonemic and prosodic cue decoding in children with autism. *Neuroreport*, 20(13), 1219–1224. <https://doi.org/10.1097/WNR.0b013e32832fa65f>.
- Mitchell, S., Brian, J., Zwaigenbaum, L., Roberts, W., Szatmari, P., Smith, I., & Bryson, S. (2006). Early language and communication development of infants later diagnosed with autism spectrum disorder. *Journal of Developmental and Behavioral Pediatrics*, 27 (2 Suppl), S69–S78.
- Mosconi, M. W., Cody-Hazlett, H., Poe, M. D., Gerig, G., Gimpel-Smith, R., & Piven, J. (2009). Longitudinal study of amygdala volume and joint attention in 2- to 4-year-old children with autism. *Archives of General Psychiatry*, 66(5), 509–516. <https://doi.org/10.1001/archgenpsychiatry.2009.19>.
- Mundy, P., Sigman, M., Ungerer, J., & Sherman, T. (1987). Nonverbal communication and play correlates of language development in autistic children. *Journal of Autism and Developmental Disorders*, 17(3), 349–364.
- Nadig, A. S., Lee, I., Singh, L., Bosshart, K., & Ozonoff, S. (2010). How does the topic of conversation affect verbal exchange and eye gaze? A comparison between typical development and high-functioning autism. *Neuropsychologia*, 48(9), 2730–2739. Elsevier Ltd. <https://doi.org/10.1016/j.neuropsychologia.2010.05.020>.
- Parish-Morris, J., Hennon, E. A., Hirsh-Pasek, K., Golinkoff, R. M., & Tager-Flusberg, H. (2007). Children with autism illuminate the role of social intention in word learning. *Child Development*, 78(4), 1265–1287. <https://doi.org/10.1111/j.1467-8624.2007.01065.x>.
- Parks, L. K., Hill, D. E., Thoma, R. J., Euler, M. J., Lewine, J. D., & Yeo, R. A. (2009). Neural correlates of communication skill and symptom severity in autism: A voxel-based morphometry study. *Research in Autism Spectrum Disorders*, 3(2), 444–454. <https://doi.org/10.1016/j.rasd.2008.09.004>.
- Paul, R. (2006). *Language disorders from infancy through adolescence: Assessment and intervention* (3rd ed.). St. Louis: Mosby.
- Paul, R., Augustyn, A., Klin, A., & Volkmar, F. (2005). Perception and production of prosody by speakers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 35(2), 205–220. <https://doi.org/10.1007/s10803-004-1999-1>.
- Paul, R., Chawarska, K., Fowler, C., Cicchetti, D., & Volkmar, F. (2007). “Listen my children and you shall hear”: Auditory preferences in toddlers with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, 50(October), 1350–1365.
- Paul, R., Chawarska, K., Cicchetti, D., & Volkmar, F. (2008). Language outcomes of toddlers with autism spectrum disorders: A two year follow-up. *Autism Research*, 1(2), 97–107. <https://doi.org/10.1002/aur.12>.
- Paul, R., Orlovski, S. M., Marcinko, H. C., & Volkmar, F. (2009). Conversational behaviors in youth with high-functioning ASD and Asperger syndrome. *Journal of Autism and Developmental Disorders*, 39, 115–125.

- Peppé, S., Cleland, J., Gibbon, F., O'Hare, A., & Castilla, P. M. (2011). Expressive prosody in children with autism spectrum conditions. *Journal of Neurolinguistics*, 24(1), 41–53. Elsevier Ltd. <https://doi.org/10.1016/j.jneuroling.2010.07.005>.
- Preissler, M. A., & Carey, S. (2005). The role of inferences about referential intent in word learning: Evidence from autism. *Cognition*, 97(1), B13–B23. <https://doi.org/10.1016/j.cognition.2005.01.008>.
- Redcay, E., & Courchesne, E. (2008). Deviant functional magnetic resonance imaging patterns of brain activity to speech in 2-3-year-old children with autism spectrum disorder. *Biological Psychiatry*, 64(7), 589–598. <https://doi.org/10.1016/j.biopsych.2008.05.020>.
- Roberts, J., Rice, M., & Tager-Flusberg, H. (2004). Tense marking in children with autism. *Applied Psycholinguistics*, 25(03), 429–448. <https://doi.org/10.1017/S0142716404001201>.
- Rojas, D. C., Peterson, E., Winterrowd, E., Reite, M. L., Rogers, S. J., & Tregellas, J. R. (2006). Regional gray matter volumetric changes in autism associated with social and repetitive behavior symptoms. *BMC Psychiatry*, 6, 56. <https://doi.org/10.1186/1471-244X-6-56>.
- Rundblad, G., & Annaz, D. (2010). The atypical development of metaphor and metonymy comprehension in children with autism. *Autism*, 14(1), 29–46.
- Rutherford, M. D., Baron-Cohen, S., & Wheelwright, S. (2002). Reading the mind in the voice: A study with normal adults and adults with Asperger syndrome and high functioning autism. *Journal of Autism and Developmental Disorders*, 32(3), 189–194.
- Schoen, E., Paul, R., & Chawarska, K. (2011). Phonology and vocal behavior in toddlers with autism spectrum disorders. *Autism Research*. <https://doi.org/10.1002/aur.183>.
- Schumann, C. M., Barnes, C. C., Lord, C., & Courchesne, E. (2009). Amygdala enlargement in toddlers with autism related to severity of social and communication impairments. *Biological Psychiatry*, 66(10), 942–949. Elsevier Inc. <https://doi.org/10.1016/j.biopsych.2009.07.007>.
- Sheinkopf, S. J., Mundy, P., Oller, D. K., & Steffens, M. (2000). Vocal atypicalities of preverbal autistic children. *Journal of Autism and Developmental Disorders*, 30(4), 345–354.
- Shriberg, L. D., Paul, R., McSweeney, J. L., Klin, A., Cohen, D. J., & Volkmar, F. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning autism and Asperger syndrome. *Journal of Speech, Language, and Hearing Research*, 44(5), 1097–1115.
- Shumway, S., & Wetherby, A. (2009). Communicative acts of children with Autism Spectrum Disorders in the second year of life. *Journal of Speech, Language, and Hearing Research*, 52(October), 1139–1157.
- Sigman, M., & McGovern, C. (2005). Improvement in cognitive and language skills from preschool to adolescence in autism. *Journal of Autism and Developmental Disorders*, 35(1), 15–23.
- Siller, M., & Sigman, M. (2008). Modeling longitudinal change in the language abilities of children with autism: Parent behaviors and child characteristics as predictors of change. *Developmental Psychology*, 44(6), 1691–1704. <https://doi.org/10.1037/a0013771>.
- Silverman, L. B., Bennetto, L., Campana, E., & Tanenhaus, M. K. (2010). Speech-and-gesture integration in high functioning autism. *Cognition*, 115(3), 380–393. Elsevier B. V. <https://doi.org/10.1016/j.cognition.2010.01.002>.
- Smith, V., Mirenda, P., & Zaidman-Zait, A. (2007). Predictors of expressive vocabulary growth in children with autism. *Journal of Speech, Language, and Hearing Research*, 50(1), 149–160. [https://doi.org/10.1044/1092-4388\(2007/013\)](https://doi.org/10.1044/1092-4388(2007/013)).
- Stone, W., Ousley, O., & Littleford, C. (1997). Motor imitation in young children with autism: What's the object. *Journal of Abnormal Child Psychology*, 25(6), 475–485.
- Swensen, L. D., Kelley, E., Fein, D., & Naigles, L. R. (2007). Processes of language acquisition in children with autism: Evidence from preferential looking. *Child Development*, 78(2), 542–557. <https://doi.org/10.1111/j.1467-8624.2007.01022.x>.
- Tager-Flusberg, H. (1985). The conceptual basis for referential word meaning in children with autism. *Child Development*, 56(5), 1167–1178.
- Tager-Flusberg, H. (1992). Autistic children's talk about psychological states: Deficits in the early acquisition of a Theory of Mind. *Child Development*, 63(1), 161. <https://doi.org/10.2307/1130910>.
- Tager-Flusberg, H., Calkins, S., Nolin, T., & Baumberger, T. (1990). A longitudinal study of language acquisition in autistic and Down syndrome children. *Journal of Autism and Developmental Disorders*, 20(1), 1–21. Germany: Springer.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 335–364). New York: Wiley Press.
- Tager-Flusberg, H., Rogers, S., Cooper, J., Landa, R., Lord, C., Paul, R., et al. (2009). Defining spoken language benchmarks and selecting measures of expressive language development for young children with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, 52(3), 643–652. [https://doi.org/10.1044/1092-4388\(2009/08-0136\)](https://doi.org/10.1044/1092-4388(2009/08-0136)).
- Tager-Flusberg, H., Rogers, S. J., Cooper, J., Landa, R., Lord, C., Paul, R., et al. (2010). Defining spoken language benchmarks and selecting measures of expressive language development for young children with autism spectrum disorders. *Journal of Speech, Language and Hearing Research*, 52(3), 1–13.
- Tek, S., Jaffery, G., Fein, D., & Naigles, L. R. (2008). Do children with autism spectrum disorders show a shape bias in word learning? *Autism Research*, 1(4), 208–222. <https://doi.org/10.1002/aur.38>.
- Thurm, A., Lord, C., Lee, L.-C., & Newschaffer, C. (2007). Predictors of language acquisition in preschool

- children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 1721–1734.
- Tomasello, M., & Barton, M. E. (1994). Learning words in non-ostensive contexts. *Developmental Psychology*, 30(5), 639–650. <https://doi.org/10.1037/0012-1649.30.5.639>.
- Turner, L. M., Stone, W., Pozdol, S. L., & Coonrod, E. E. (2006). Follow-up of children with autism spectrum disorders from age 2 to age 9. *Autism*, 10(3), 243–265.
- Volden, J., & Lord, C. (1991). Neologisms and idiosyncratic language in autistic speakers. *Journal of Autism and Developmental Disorders*, 21(2), 109–130. Germany: Springer.
- Wang, A., Lee, S., Sigman, M., & Dapretto, M. (2006). Neural basis of irony comprehension in children with autism: The role of prosody and context. *Brain: A Journal of Neurology*, 129(Pt. 4), 932–943. <https://doi.org/10.1093/brain/awl032>.
- Wang, A., Lee, S. S., Sigman, M., & Dapretto, M. (2007). Reading affect in the face and voice: Neural correlates of interpreting communicative intent in children and adolescents with autism spectrum disorders. *Archives of General Psychiatry*, 64(6), 698–708. <https://doi.org/10.1001/archpsyc.64.6.698>.
- Waterhouse, L., & Fein, D. (1982). Language skills in developmentally disabled children. *Brain and Language*, 15(2), 307–333.
- Watson, L. R., Baranek, G. T., Roberts, J., David, F. J., & Perryman, T. Y. (2010). Behavioral and physiological responses to child-directed speech as predictors of communication outcomes in children with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, 53(4), 1052–1064. [https://doi.org/10.1044/1092-4388\(2009/09-0096\)](https://doi.org/10.1044/1092-4388(2009/09-0096)).
- Weismer, S. E., Lord, C., & Esler, A. (2010). Early language patterns of toddlers on the autism spectrum compared to toddlers with developmental delay. *Journal of Autism and Developmental Disorders*, 40(10), 1259–1273. <https://doi.org/10.1007/s10803-010-0983-1>.
- Werner, E., & Dawson, G. (2005). Validation of the phenomenon of autistic regression using home videotapes. *Archives of General Psychiatry*, 62(8), 889–895. <https://doi.org/10.1001/archpsyc.62.8.889>.
- Wetherby, A., Woods, J., Allen, L., Cleary, J., Dickinson, H., & Lord, C. (2004). Early indicators of autism spectrum disorders in the second year of life. *Journal of Autism and Developmental Disorders*, 34(5), 473–493. United States.
- Wetherby, A., Watt, N., Morgan, L., & Shumway, S. (2007). Social communication profiles of children with autism spectrum disorders late in the second year of life. *Journal of Autism and Developmental Disorders*, 37, 960–975.
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, 23(2–3), 143–152. England.

Communicative Act

- Protodeclarative
- Protoimperative

Communicative Development Inventories

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Keywords

CDI-III · CDIs · Infant form · MacArthur-Bates Communicative Development Inventories (MCDIs) · Toddler form

Abbreviations

CDI-WG CDI-Words and Gestures
CDI-WS CDI-Words and Sentences

Description

2nd Edition CDIs (2007)

The MacArthur-Bates Communicative Development Inventories (CDIs) are standardized, norm-referenced, parent-report measures of early language and communication. All reference here pertains to the 2nd edition English-language publication (Fenson et al. 2007), unless otherwise stated. A User's Guide and Technical Manual (hereafter, Manual) accompanies three alternative test forms: CDI-Words and Gestures (CDI-WG), CDI-Words and Sentences (CDI-WS), and CDI-III. The 2nd edition Manual (Fenson et al. 2007) updates and expands upon its predecessor (Fenson et al. 1992), providing details around:

Theoretical rationale for the CDIs

History of development of the CDIs

The various sections and items on each test form

Administration and scoring of the forms and interpretation based on normative data
Clinical and research uses of the CDIs

The Manual Appendix includes some photocopyable forms which (a) facilitate the summary of an individual's CDI scores and (b) guide the types of background data which might be sought concurrently.

CDI-Words and Gestures. With a normative sample of 1,089 infants aged 8–18 months, the CDI-WG evaluates early comprehension and production of language and nonverbal communication. Several subsections are arranged within two major parts. Part 1 evaluates infants' *First Signs of Understanding*, including whether they have begun to respond to language (e.g., recognizing own name) and whether they understand commonly used phrases (e.g., "give it to mommy"). Parents then report on infants' *Starting to Talk* (i.e., word/phrase imitation and early object labeling). The *Vocabulary Checklist* presents 396 words within 19 semantic categories (e.g., animal names, toys, etc.), and parents indicate those words their infant "understands" and "understands and says." Raw expressive vocabulary (i.e., all "understands and says") and receptive vocabulary (i.e., all "understands" or "understands and says") can be totaled.

Part 2 evaluates 63 communicative and symbolic *Actions and Gestures*, presented within five categories. *Early Gestures* include *First Communicative Gestures* (e.g., pointing and showing) and *Games and Routines*, signaling emerging intentional communication and social engagement. *Later Gestures* include *Actions with Objects*, *Imitating other Adult Actions*, and *Pretending to be a Parent*, signaling the developing awareness of objects' functional and representational uses.

CDI-Words and Sentences. With a normative sample of 1,461 toddlers aged 16–30 months, the CDI-WS evaluates later aspects of language production, including expressive vocabulary, grammar, and syntax. Several subsections are again arranged within two parts. Part 1 evaluates *Words Children Use*, presenting a *Vocabulary Checklist* of 680 words arranged within

22 categories. Parents indicate those words their toddler "understands and says," providing an expressive (but not receptive) vocabulary count. *How Children Use Words* considers reference to the past and future, etc. Part 2 evaluates *Sentences and Grammar*, including *Word Endings* (i.e., regular plurals, possessive forms, etc.), and *Word Forms/Endings* (i.e., irregular nouns/verbs, and over-regularizations). Within *Word Combinations*, parents indicate whether their toddler at all combines words, and if so, they transcribe the three longest phrases/sentences recently produced. From these, the *Mean length of Three Longest sentences* (M3L; see Manual, pp. 22–23) is computed (akin to Mean Length of Utterance [MLU]). Finally, parents report on their toddler's current level of *Complexity*, indicating which exemplar within each of 37 pairs of phrases best matches the toddler's current speech (e.g., "that my truck" vs. "that's my truck").

CDI-III. A new addition, the relatively short CDI-III, was designed as an upward extension of the CDI-WS, using a normative sample of 356 children aged 30–37 months. Expressive vocabulary is assessed with a 100-item *Vocabulary Checklist* (around half of items overlap with CDI-WS items). Sentence *Complexity* is assessed using 12 sentence pairs, and *Language Use* questions (not included in the CDI-WS) assess aspects of comprehension, semantics, and syntax. Pilot testing informed the retention of some appropriate items from the CDI-WS and the inclusion of other new items, and the upper age limit of 37 months was adopted given the substantive ceiling scores observed beyond this age.

Spanish CDIs

While numerous other-language CDI adaptations are currently underway (see section "Other Language Adaptations"), two Spanish version CDIs have progressed to the level of Manual publication. The Spanish (Mexican) version comprises a full-test pack, including a Users' Guide and Technical Manual (Jackson-Maldonado et al. 2003), and accompanying CDI-WG and CDI-WS forms. A Spanish (European) version (Lopez Ornat et al. 2005) has been adapted from the former.

Additional Developments

CDI Short Forms

Brief CDI-WG and CDI-WS forms (Fenson et al. 2000) are mentioned within the 2nd edition Manual (Fenson et al. 2007, pp. 13–14), but not published alongside it. These are purchased directly from the authors (refer to *See Also*) and present a subset of the appropriate *Vocabulary Checklist* items. Level 1 maps on to the CDI-WG for 8–18-month-olds, and two (alternate version) level 2 Short Forms map on to the CDI-WS for 16–30-month-olds. Each form also enquires about whether the child yet combines words. Completion time is around 10 min, permitting rapid assessment of vocabulary acquisition, and interview administration format can be used if needed (e.g., in cases of low parent literacy), although normative data were collected using the standard checklist format.

Other Language Adaptations

A CDI Advisory Board promotes (and authorizes) adaptation of the CDIs for other non-English languages, some accompanied by normative data and all available for public use. Section “See Also” contains more information, as does the Advisory Board website: <http://www.sci.sdsu.edu/cdi/>

Historical Background

Interest in language development has a long history, and the current CDIs have evolved over 30 years (Fenson et al. 2007). Parent reports have regularly informed child language and communicative assessment, with diary studies common early on. Into the 1970s and 1980s, research programs led by Elizabeth Bates and Leslie Rescorla endeavored to develop more user-friendly questionnaire formats for parent report. Precursors to the current CDIs were four earlier questionnaires; the Communicative Development Questionnaire (8–12 months), Language and Gesture Inventory (12–18 months), Early Language Inventory (18–24 months), and Grammatical Development Questionnaire (24–36 months). The 1st edition CDI Manual, CDI-WG, and

CDI-WS forms were published in 1992 (Fenson et al. 1992), with Italian versions also developed and published around this same time (Caselli and Casadio 1995). A research monograph on early communication development also appeared in 1994, presenting detailed analysis of data from the English-language normative sample. This included developmental trajectories of the various CDI skills, and evaluation of the correspondence across skills domains, and consideration of the impact of factors such as gender, birth order, and social class on language development (Fenson et al. 1994).

The CDI Manual is currently in its 2nd edition (Fenson et al. 2007). While the inventory forms have seen no substantive alteration, updates to the Manual include an improved normative data set, brief presentation of the Short Forms, and introduction of the CDI-III. Instructions for administering, scoring, and interpreting the CDIs were also expanded upon, and more details were included on the instrument psychometric properties and on research and clinical application. As undertaken with the original normative sample (Fenson et al. 1992, 1994), the updated Manual also includes analysis of developmental trajectories and cross-domain correspondence, along with some evaluation of the impact of other factors on language acquisition/development (Fenson et al. 2007; see section “Psychometric Data”).

Psychometric Data

Normative Samples

Normative data for the CDIs have been collected in two phases. During initial instrument development, 659 CDI-WG forms and 1,130 CDI-WS forms were completed (Fenson et al. 1992). In preparing the 2nd edition Manual (Fenson et al. 2007), an additional 430 CDI-WG and 331 CDI-WS forms were collected. The authors sought to increase the CDI-WG normative range up to 18 months, rather than just 16 months (as in the 1st edition). Increased sample diversity was also sought, to better align with US census records around ethnicity and parent educational level. Ethnic distribution of the 2nd edition normative

sample does better match US census records, albeit with fewer Hispanic respondents than expected, likely due to the inclusion criterion of English as the primary home language, for this sample. Parental education levels remain above the US average, although good variability is included within the 2nd edition normative sample: around 30% of respondents held a high-school diploma or lower level of completed education (Fenson et al. 2007). Geographical sampling (across US states) was also improved for the 2nd edition.

Reliability

The Manual reports reliability in terms of internal consistency (IC) and test-retest reliability (TRT) across the broad skills domains assessed within the CDI-WG and CDI-WS forms (Fenson et al. 2007, pp. 99–102). These various *Vocabulary Checklist* components (CDI-WG comprehension and production and CDI-WS production) all evidence very strong IC, as do the CDI-WS sentence *Complexity* items. IC is lower, however, for CDI-WG *Gestures* items: while the *Actions with Objects* and *Imitating Other Adult Actions* correlate quite highly together, those among the other three sections are lower. TRT reliability is assessed across a maximum inter-assessment interval of 2 months, within a subgroup of the normative sample. CDI-WS expressive vocabulary counts for 216 toddlers were very stable. CDI-WG receptive vocabulary counts and gesture scores were similarly stable for 137 infants, with the single exception of those aged 12 months at initial assessment (see Fenson et al. 2007, p. 101 for discussion). CDI-WG expressive counts were also quite stable, except for infants aged 8–10 months at initial assessment (and likely due to floor effects at these young ages; Fenson et al.).

The authors acknowledge difficulties inherent in assessing the reliability of a parent-report instrument, including the possibility for halo effects and recall bias to impact upon IC and TRT reliability estimates. Ideally, evaluation of inter-rater reliability (IRR) would also be included, but this requires availability of a second respondent, equally familiar with the child's

abilities (frequently improbable). The authors argue, however, that strong evidence of validity (see below) presupposes robust reliability, further strengthening the case for reliability of the inventories (Fenson et al. 2007).

Validity

The Manual reports on face, content, convergent, concurrent, and predictive forms of validity across the broad skills domains assessed within the CDI-WG and CDI-WS (Fenson et al. 2007, pp. 102–114). Face validity is supported by the professional appearance of the checklist forms, along with the breadth and depth with which language and communication skills are addressed in the assessment. The authors feel that parents will consider the CDIs as valid and comprehensive assessments of their children's abilities and thereby provide considered responses. Content validity is evidenced in the authors' use of the language development literature to ensure inclusion of those aspects of early language and communication development most important at the ages assessed. While phonology and communicative pragmatics fail to be represented in the CDIs, the authors maintain that those aspects included are given comprehensive coverage (Fenson et al. 2007).

Convergent validity is evidenced from the observation of correlations between CDI scores and language/communication data obtained through other methods (see Fenson et al. 2007, Chap. 4, and Fenson et al. 1994, for details). In brief, developmental trajectories across various broad communication and language domains evaluated in the CDIs are shown to increase consistently and regularly across ages assessed. Each skill domain assessed demonstrates an onset around the age at which it would be expected (given the broader literature), and rates of development correspond similarly to expectations from past research. Individual variability similarly corresponds to that which would be expected. Fenson et al. (2007) note some clear exceptions, however, including the parents of some 8-month-olds reporting greater receptive vocabularies, and other parents reporting much earlier onset of

some pretense skills, than would be expected (see Manual, p. 103). On the whole, however, evidence for convergent validity is substantial. Fenson et al. (2007) argue that parents and researchers appear to be observing and reporting upon the same development phenomena irrespective of the research method employed (i.e., CDI report vs. laboratory experiment).

Evidence for concurrent validity emerges through the comparison of results from one test (i.e., parts of a CDI form) with those arising from other similar assessments (e.g., formal language tests, naturalistic language samples, etc.). A measure's evaluated concurrent validity is influenced not only by its own indices of reliability and construct validity but also by those of the comparison measure. Furthermore, strong concurrent validity will only be evidenced when the target and comparison measures assess an *equivalent* skill, a potential problem for the CDIs. Parent reports of child language draw on a wealth of knowledge about the child and daily exposure to his/her communication and are sought for the very reason that they are unlikely to correspond perfectly language skills assessed using other means (e.g., during a formal one-off test with an unfamiliar adult, or during a relatively brief naturalistic language sample, Fenson et al. 2007). Notwithstanding, *Vocabulary Checklist* counts show moderate-to-strong correspondence with other vocabulary and language measures (see a tabulation of various study results in Fenson et al. 2007, pp. 106–107). More limited, however, is the evidence around *Gestures*, given the dearth of other accepted measures of gesture to which CDI scores can be compared. These do, however, associate closely with formally assessed aspects of language (i.e., comprehension; see Manual p. 109). CDI-WS *Complexity* has been compared to spontaneous speech transcriptions, with CDI M3L and other complexity scores associated strongly with MLU during unstructured play (Fenson et al.).

Predictive validity is considered in the 2nd edition Manual (Fenson et al. 2007), based on a subset of the normative sample which completed a second inventory form 6 months after initial assessment. For many, this entailed a repeat

assessment with the same form (i.e., 62 parents completed CDI-WG and 228 completed CDI-WS twice). For 217 cases, the interval necessitated initial use of the CDI-WG but follow-up using the CDI-WS. Predictive validity of expressive vocabulary counts was high for all infants and toddlers aged above 11–12 months at the first assessment, with best predictive power observed at an initial 20-month assessment. Only limited validity was shown for expressive skills in infants younger than 11–12 months and for receptive skills (only assessable at two time points for infants initially aged up to 12 months). This pattern of findings is argued to reflect a true lack of stability in the language skills of very young infants (a proposal supported by evidence from other areas of the developmental literature; see Fenson et al. 2007), rather than a flaw of the CDI-WG. CDI-WS grammatical complexity showed good predictive validity, particularly in children aged 20 months and older at initial assessment.

CDI-III

CDI-III normative data yield from 356 children aged 30–37 months. Given the relative recency of this form, only very limited psychometric data are available, summarized in the Manual (Fenson et al. 2007, pp. 154–160). Educational levels of respondents diverge significantly from US census data, limiting the confidence with which scores for children from low socioeconomic backgrounds can be validly interpreted.

CDI Short Forms

The 2nd edition Manual (Fenson et al. 2007) refers only briefly to the CDI-WG and CDI-WS Short Forms, available for purchase directly from the authors (for details, “See Also”). Preliminary normative data exist in a research publication (Fenson et al. 2000), with short-form and full-form vocabulary counts highly intercorrelated. The two (alternate version) level 2 Short Forms demonstrate important ceiling effects for toddlers older than 27–28 months, due to the abbreviated vocabulary list length containing only 100 words (vs. 680 words in the full CDI-WS).

Clinical Uses

Administration and Scoring

Administration of a full-version CDI form requires 20–40 min and should be self-explanatory to parents. While normative data collection was through postal return of forms, the Manual provides suggestions for clarifications to parents and procedures for checking report accuracy (Fenson et al. 2007, pp. 15–18). Both the CDI-WG and CDI-WS forms have norms for 16–18-month-olds, and form selection will therefore depend on the purpose of current and possible future assessments (see pp. 12–13). Options for scoring and obtaining normative CDI data (by hand or using automated methods) are discussed in detail (pp. 18–34). The authors address issues around data interpretation for three subgroups (pp. 34–38):

Children from low SES backgrounds (including where parental education is low)

Children who are learning more than one language

Children who are older than the normative group, but for whom language skills are within the assessable range of the CDIs

Parent-Report Pros and Cons

Parent reports are based on the everyday observation of child language and arguably produce more ecologically valid results than otherwise obtainable (e.g., through formal/direct assessment, naturalistic language sampling, etc.). CDIs benefit from the wealth of parent knowledge and are unlikely to be negatively influenced by aspects of the child's personality (e.g., shyness) or mood on a given day (e.g., fussiness). Clinically, parent report is also time efficient and cost effective; CDIs can be completed prior to attendance at more costly clinic appointments (i.e., permitting better use of the consultation time) and can be completed at multiple time points (i.e., to facilitate developmental monitoring; Fenson et al. 2007).

Other biases may be inherent, however. Parents may consistently over- or underreport their child's abilities, and the experience of

completing an inventory might influence later reports provided using the measure (i.e., through alteration of the parents' behavior with, or subsequent observation of, their child). However, such a possibility has been evaluated for the CDI, with minimal such influence observed (although such contention is more justified for parent reports on other aspects of child developmental skill; Fenson et al. 2007). Furthermore, the CDI format aims specifically to reduce potential respondent bias, addressing current and emerging skills (rather than past abilities) and using a recognition (rather than recall) format to minimize the effects of memory and item interpretation.

Clinical Uses for the Parent-Report CDIs

The Manual presents potential clinical and research uses of the CDIs (Fenson et al. 2007, pp. 40–46). While not uniformly accurate, and therefore inadvisably used in isolation of other information sources, CDIs should yield highly representative language characterization.

Screening for language delay. Firm diagnosis of specific language impairment (SLI) is possible only from around 3 years. However, CDIs permit assessment of conventional early markers for such language disorder (Fenson et al. 2007). Both “delay 3” (Rescorla 1989) and “delay 3+” (Klee et al. 2000) criteria for identifying late talkers can be gauged with the CDIs and associated Basic Information Form (Manual Appendix). Not all late talkers will develop enduring language problems. However, early identification of atypical developmental patterns may indicate further assessment and ongoing monitoring. Rates of communication *growth* (able to be evaluated through repeated CDI completion) better predict later language ability than do the results of any single assessment (Thal 2000). Delays in concurrent language comprehension and production accompanied by a failure to compensate with gestures signify particularly high risk (Thal and Katich 1996), with each component addressed within CDI-WG assessments.

Characterizing special groups. While the CDI normative sample extends only to 30 months

(37 months for CDI-III), they can be used to evaluate the skills of older children whose language falls within the range of assessed domains (i.e., developing vocabulary, emerging grammar, etc.). As such, they are increasingly used clinically/for research with individuals with autism spectrum disorders, Down syndrome, Williams syndrome, and cleft lip/palate, among other groups (e.g., Charman et al. 2003; Mervis and Robinson 2000; Snyder and Scherer 2004). Normative scores are only interpretable where older individuals' raw scores fall at or below the 30-month median level (Fenson et al. 2007). Raw scores are therefore typically of greatest interest (e.g., documenting how many raw words an individual understands/says).

Intervention design and evaluation. CDI results can identify specific intervention targets, such as the promotion of vocabulary growth, specific lack of comprehension skills, development of correct grammar, etc. Furthermore, the inventories can be used to evaluate/demonstrate post-intervention change. The Manual notes some such intervention studies including samples of toddlers with expressive language delays, with cleft lip/palate, and following cochlear implant (Fenson et al. 2007, p. 44).

See Also

- ▶ Communication and Symbolic Behavior Scale
- ▶ Communication Assessment
- ▶ Expressive Language
- ▶ Gestures
- ▶ Grammar
- ▶ Language
- ▶ Language Acquisition
- ▶ Language Development Survey
- ▶ Language Tests
- ▶ Play
- ▶ Pretend Play
- ▶ Preverbal Communication
- ▶ Receptive Language
- ▶ Receptive Vocabulary
- ▶ Symbolic Play
- ▶ Vocabulary

References and Reading

CDI Advisory Board and Website

Additional materials and information on the CDIs, including contact details for purchasing the various publications and inventory forms, are available at: <http://www.sci.sdsu.edu/cdi>. This site is administered by the CDI Advisory Board, a not-for-profit organisation supporting further development of the inventories. Reference lists are included, concerning the various inventory forms and versions, and dating from the 1980s through early 2000s. Some relevant professional organisations, conference groups, and research groups are listed, and a dedications page honours Elizabeth Bates, a key CDI developer, who passed away in 2003.

Other-Language Adaptations

The website and Advisory Board play a key role in CDI other-language adaptations. Guidelines are provided for obtaining appropriate authorisation, and include requirements around the competence and resource-availability necessary to undertake adaptation. Suggestions regarding the adaptation procedure are outlined in an unpublished manuscript http://www.sci.sdsu.edu/cdi/suggestions_adaptations.htm (Dale et al. 1993). Other-language adaptations currently undertaken/underway are tabulated, and entries include investigator contact details, project website links, and publication reference lists (http://www.sci.sdsu.edu/cdi/adaptations_ol.htm).

Lex2005 Database

The Lex2005 Database (Dale and Fenson 1996), available on the CDI Website, (http://www.sci.sdsu.edu/cdi/lexical_e.htm) provides a month-by-month compilation of normative data for all CDI receptive and expressive *Vocabulary Checklist* items (in each of the English and Spanish [Mexican] versions). Users select the language, specific measure (i.e., receptive/expressive), types of word (i.e., all, specified semantic groups, specified words), and ages of interest, and the database returns the proportion of children at each age reported to understand/say each word of interest.

CDI Scoring Program (2004)

The website also allows users to download, free of charge, the *Scoring Program for the CDI (2004)*, with accompanying user's guide.: http://www.sci.sdsu.edu/cdi/scoringp_download.htm. The program aims to facilitate management of CDI data, including entry and scoring, and interfaces with PCs running Microsoft Office 97 or higher. The website mentions CDI Scoring Templates (http://www.sci.sdsu.edu/cdi/scoring_t.htm), and describes the types of scanner system required for automated scoring. Template configurations are indicated to be 'Coming Soon!' However,

like most of the website, this page notes its most recent update as February, 2006.

References

- Caselli, M. C., & Casadio, P. (1995). *Il Primo Vocabolario Del Bambino (Children's early vocabulary)*. Milan: France Angeli.
- Charman, T., Drew, A., Baird, C., & Baird, G. (2003). Measuring early language development in preschool children with autism spectrum disorder using the MacArthur communicative development inventory (Infant Form). *Journal of Child Language*, 30, 213–236.
- Dale, P. S., & Fenson, L. (1996). Lexical development norms for young children. *Behavior Research Methods, Instruments, & Computers*, 28, 125–127.
- Dale, P. S., Fenson, L., & Thal, D. J. (1993). *Development inventories to additional languages*. <http://www.sci.sdsu.edu/cdi/adaptations.htm>
- Fenson, L., Dale, P. S., Reznick, J. S., Thal, D. J., Bates, E., Hartung, J. P., et al. (1992). *MacArthur communicative development inventories: User's guide and technical manual* (1st ed.). Baltimore: Paul H. Brookes.
- Fenson, L., Dale, P. S., Reznick, J. S., Bates, E., Thal, D. J., & Pethick, S. J. (1994). Variability in early communicative development. *Monographs of the Society for Research in Child Development*, 59, 1–173.
- Fenson, L., Pethick, S. J., Renda, C., Cox, J. L., Dale, P. S., & Reznick, J. S. (2000). Short form versions of the MacArthur communicative development inventories. *Applied PsychoLinguistics*, 21, 95–115.
- Fenson, L., Marchman, V. A., Thal, D. J., Dale, P. S., Reznick, J. S., & Bates, E. (2007). *MacArthur-Bates communicative development inventories: User's guide and technical manual* (2nd ed.). Baltimore: Paul H. Brookes.
- Jackson-Maldonado, D., Thal, D. J., Marchman, V. A., Newton, T., Fenson, L., & Conboy, B. (2003). *MacArthur inventarios del desarrollo de habilidades comunicativas. User's guide and technical manual*. Baltimore: Paul H. Brookes.
- Klee, T., Pearce, K., & Carson, D. (2000). Improving the positive predictive value of screening for developmental language disorder. *Journal of Speech, Language, and Hearing Disorders*, 43, 821–833.
- Lopez Ornat, S., Gallego, C., Gallo, P., Karousou, A., Mariscal, S., & Martinez, M. (2005). *MacArthur: Inventario de desarrollo comunicativo. Manual y Cuadernillos*. Madrid: TEA Ediciones.
- Mervis, C. B., & Robinson, B. F. (2000). Expressive vocabulary ability of toddlers with Williams syndrome or Down syndrome: A comparison. *Developmental Neuropsychology*, 17, 111–126.
- Rescorla, L. (1989). The language development survey: A screening tool for delayed language in toddlers. *The Journal of Speech and Hearing Disorders*, 54, 587–599.
- Snyder, L. E., & Scherer, N. (2004). The development of symbolic play and language in toddlers with cleft palate. *American Journal of Speech-Language Pathology*, 13, 66–80.
- Thal, D. J. (2000). *Late-talking toddlers: Are they at risk?* San Diego: San Diego State University Press.
- Thal, D. J., & Katich, J. (1996). Predicaments in early identification of specific language impairment: Does the early bird always catch the worm? In K. Cole, P. S. Dale, & D. J. Thal (Eds.), *Communication and language intervention series* (Advances in assessment of communication and language, Vol. 6, pp. 1–28). Baltimore: Paul H. Brookes.

Communicative Functions

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Synonyms

Communication intentions; Communicative intent

Definition

Communicative functions refer to the purpose of gestural, vocal, and verbal acts intended to convey information to others. Some communicative functions include commenting, requesting, protesting, directing attention, showing, and rejecting. Gestures and vocalizations are often first observed as an indication of intentionality in infants 8–9 months of age. It is at this point many infants appear to begin pursuing their intentions through interactions with others. The development of communicative functions has been described by Bates as occurring in a sequence of three stages: *perlocutionary*, *illocutionary*, and *locutionary*.

The *perlocutionary* stage of intentionality begins at birth and is expected to continue until approximately 8 months of age. During this period, the infant focuses on objects and people and attends, discriminates, and responds to stimuli

through cries and coos. At this developmental level, the child does not possess the mental representational capacity to hold an intention in mind and convey it to another, but his behavior is interpreted and treated by caregivers as if it were intentional. As the predictability of interactions between the caregiver and child increases and cognitive development progresses, infants begin associating their actions with specific responses from their caregivers. Between 8 and 12 months, the *illocutionary* stage begins in which infants begin using gestures and vocalizations coupled with eye gaze and often repeating or modifying communicative acts in an intentional way to convey a message. In this stage, intentional, non-conventional gestures, such as tantrums, also begin to appear. Finally, the *locutionary* stage begins with a child's first meaningful word. This final stage of intentionality development typically occurs at approximately 12 months of age. During this stage, a child uses words in conjunction with gestures, sounds, and gaze to convey, at first, a limited range of communicative functions. As language and social-cognitive development proceed through the preschool and school years, a wider range of communicative intentions is acquired.

See Also

- [Communication Intention Inventory](#)
- [Pragmatics](#)

References and Reading

- Austin, J. L. (1962). *How to do things with words*. New York: Oxford University Press.
- Bates, E. (1976). *Language in context: Studies in the acquisition of pragmatics*. New York: Academic Press.
- Bates, E., Benigni, T., Bretherton, I., Camaioni, D., & Volterra, V. (1979). *The emergence of symbols: Cognition and communication in infancy*. New York: Academic Press.
- Leonard, L. B., Camarata, S., Rowan, L. E., & Chapman, K. (1982). The communicative functions of lexical usage by language impaired children. *Applied Psycholinguistics*, 3(02), 109–125.

- Owens, R. E. (2008). *Language development: An introduction* (7th ed.). Boston: Allyn & Bacon.
- Searle, J. (1965). What is a speech act? In M. Black (Ed.), *Philosophy in America*. New York: Allen & Unwin and Cornell University Press.
- Wetherby, A. M. (1986). Ontogeny of communicative functions in autism. *Journal of Autism and Developmental Disorders*, 16(3), 295–316.
- Wetherby, A. M., Cain, D. H., Yonclas, D. G., & Walker, V. G. (1988). Analysis of intentional communication of normal children from the pre-linguistic to the multiword stage. *Journal of Speech and Hearing Research*, 31, 240–252.

Communicative Intent

- [Communicative Functions](#)

Community Services

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Synonyms

[Community-based services](#)

Definition

Community services include a range of programs that are designed to meet the support and intervention needs of individuals with ASD and their families. Services are delivered in community settings (e.g., community centers, recreational facilities, religious organizations) and include programs for individuals across the autism spectrum and throughout the life span.

The following list includes examples of community services:

- Case management
- Life skills training
- Structured social opportunities

- Employment training
- Community mentors
- Job counselors
- Respite caregiving services
- Recreational opportunities

Developmental Shifts in Service Use: Community service use often changes over time for individuals with ASD. Although children generally receive their primary services through their school, many families also make use of additional community services such as respite caregiving services (i.e., periodic, short-term in-home support to relieve primary caregivers) and specialized recreational activities to develop an area of interest (e.g., art, sports) or to foster a successful social experience (e.g., summer camps). Similarly, adolescents can take advantage of community services to increase their independence and preparation for the transition to adulthood (e.g., structured social activity groups, community mentors). For adults with ASD, specialized job training programs and employment counseling may be utilized.

Obtaining Service: Local and national ASD organizations often compile lists of available community services. These lists are intended to aid families in obtaining the most relevant services for their family member with ASD. Cost and payment requirements vary across services and by state, though many services may be available to individuals at low or no cost through existing Medicaid funding.

Research: In contrast to the accumulating evidence base for traditional interventions for ASD, less work has been done to evaluate the effectiveness of these varied community services. Families typically report involvement with multiple community services at a time to address different needs.

See Also

- [Benhaven Residential Services](#)
- [Independent Living](#)
- [Living Arrangements in Adulthood](#)
- [Self-Help Skills](#)

References and Reading

- Hendricks, D. R. (2009). Transition from school to adulthood for youth with autism spectrum disorders: Review and recommendations. *Focus on Autism and Other Developmental Disabilities*, 24, 77–88.
- http://www.ct.gov/dds/lib/dds/autism/pilot_program_outcome_study.pdf
- Montes, G., Halterman, J. S., & Magyar, C. I. (2009). Access to and satisfaction with school and community health services for US children with ASD. *Pediatrics*, 124, S407–S413.
- Robinson, J., Reed, I., Shugrue, N., Kleppinger, A., & Gruman, G. (2008). *An evaluation of the autism pilot program of the Division of Autism services of the CT Department of Developmental Services*. Report prepared for the Connecticut Department of Developmental Services.
- Ruble, L. (2007). Community service outcomes for families and children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 1, 360–472.

Community Sport

- [Inclusion of Adolescents with ASD in Community Sporting Clubs](#)

Community Work Crew

- [Mobile Work Crew Model](#)

Community-Based Services

- [Community Services](#)

Community-Based Work Team

- [Mobile Work Crew Model](#)

Community-Integrated Residential Services for Adults with Autism

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Definition

Community-integrated residential services are homes located in the community, rather than in disability institutions, and are designed to promote the social inclusion of people with disabilities. They developed as a challenge to the historical segregation and exclusion of people with disabilities from the community. People along the autistic spectrum historically were institutionalized in segregated state institutions, but the exposure of large-scale abuses in disability institutions and the growth of the disability rights movement prompted large-scale deinstitutionalization.

Historical Background

Eugenics and Segregation

In the late nineteenth and early twentieth centuries, eugenics and social Darwinism gained enormous popularity throughout the world. These ideas suggested that disabled people carried defective genes, labeling groups of developmentally disabled people (including people with autism) as “idiots,” “imbeciles” and “morons” – or lumped together through the term “feeble minded” (Noll and Trent 2004). People who were “feeble minded” were blamed for all sorts of social problems, including petty thievery, vagrancy, alcoholism, prostitution and illegitimate children – and they were regarded as a “burden” on society. These ideas, which are recognized as incredibly prejudicial today, were justified as “scientific” by medicine and

psychiatry at the time. Indeed, eugenics inspired a generation of scientists and was taught in 44 universities throughout the United States. The classification, pathologization, and devaluation of disability were integral to the scientific ideas of the time (Snyder and Mitchell 2006).

As a result of such negative attitudes toward disability, people with developmental disabilities were removed from the community and housed in institutions for the “feeble minded.” The following summary of attitudes toward disability highlights the extent of prejudice at that time. Common attitudes included regarding people with disabilities as subhuman, sick, menacing, pitiful, and a burden (Wolfensberger 1975).

Institutionalization and sterilization were linked in the eugenic agenda. By the 1960s, over 60,000 people had been sterilized in the USA (Kerr and Shakespeare 2002). Segregated residential institutions and educational institutions were more results of eugenic ideas. In total, the effect of eugenics was to create a widespread sense that people with disabilities were inferior, and perhaps dangerous, so they should be kept away from the community and locked in institutions.

Eugenic ideas were also central to the Nazi ideology. Their belief in “building a better race” stemmed in large part from the eugenic idea that it was possible to identify and eradicate those with “defective genes.” Under the Nazi eugenic program, “racial cleansing” and mass euthanasia resulted in mass genocide. People with disabilities were bussed into killing centers (rather than concentration camps), and they were diagnosed and killed on the day they arrived – a process which has been called “medicalized killing” (Lifton 2000, p. 14). Nazis sterilized 375,000 people with disabilities and killed approximately 275,000 children and adults with disabilities (Kerr and Shakespeare 2002, pp. 27–44).

Rationale or Underlying Theory

The rationale of community-integrated residential services for adults with autism stems from the

undercovering of the systemic and horrific abuses of congregate care in the late 1960s and early 1970s typified by the Willowbrook case. Willowbrook State Hospital on Staten Island, New York, became the symbol of the deinstitutionalization movement. The thought was that if patients lived and worked in the community, there will be less of a chance of abuse occurring because the eyes of the community were upon the caretakers in the small group homes. Furthermore, people with autism were warehoused in large state-operated psychiatric facilities along with individuals with serious mental illness. This was an inappropriate placement. They received little or no treatment, nor rehabilitation. Nor was the intervention or treatment specific to autism. By being integrated into the community, individuals with autism could learn adaptive behavior to become more independent than in a large institution.

Although the rationale for deinstitutionalization of individuals with autism and other disabilities outlined above is moral and sound, another rationale for community-integrated residential services for adults is economic. The cost of 24 h care in a large state-run psychiatric facility is staggering. Current estimates are between \$150,000 and \$200,000 per year per patient. Small community-integrated residential services, more commonly referred to as group homes, are a fraction of the cost of placement in a state psychiatric facility.

Goals and Objectives

The primary goal of any placement, whether it is a community-integrated residential setting or not, is safety. When evaluating a possible placement, an eye should be kept upon how the person with autism will be kept safe. Questions to ask include the following: (1) What are the staffing and supervision levels? (2) How is wandering prevented? (3) What is the staff training in the areas of (a) CPR, first aid, and the use of automatic external defibrillators; (b) medication management; (c) aggressive behavior management; and (d) psychiatric emergencies and crisis

intervention? (4) How are the residents transported to appointments and recreational activities? (5) Are there any geographic features in the community that might be hazardous to a person with autism (e.g., proximity to a highway, body of water, or railroad tracks)?

The second set of goals should involve maximizing the person's level of independence and integration into the community. The residential services should continuously be an opportunity for the person with autism to learn new independent living skills and adaptive behavior. These skills may be vocational in nature such as working or volunteering on a part-time basis. Or they may involve learning new skills like travel training skills. Instead of relying upon an agency van to drive the person with autism to and from work or recreational activities, the person can be taught how to use mass transit or call a taxicab. Depending upon the skill level of the individual, it may involve learning pedestrian skills. The person may learn to walk to and from the group home to a nearby store. This further increases his or her level of community integration and independence.

The final set of goals should revolve around improving the person with autism's quality of life. What can be done to increase the person's sense of connectedness? What can be done to foster their sense of self-advocacy? What kinds of activities or foods can be made available to the individual to bring joy to his or her life? What can be done to provide this individual with a high quality of life?

Treatment Participants

Community-integrated residential services are generally not considered a treatment in the traditional sense under a medical model. It is, however, a social intervention involving a number of individuals and agencies depending upon the age of the participant. As a part of the transition planning process under IDEA, the IEP team may identify living in a group home as a goal for the student with autism. The IEP team consists of the student with autism or another disability, his or her parents, special educators, and representatives of agencies other than the school district. The

representatives of other agencies can include either private not-for-profit social service agencies subcontracted by the state or state-run social service systems that are known by a variety of acronyms. These can be state offices of Developmental Disabilities Services, Offices of Mental Retardation and Developmental Disabilities (OMRDD), or Offices for Persons with Developmental Disabilities (OPWDD). (It should be noted that the term “mental retardation” is now considered a derogatory term and the preferred term is a “person with a developmental disability.”). These agencies will oversee the provision of residential services in the community. Other entities that may be involved include state offices of Vocational and Rehabilitative Services, Social Security Administration, Medicaid, and Medicare programs, as well as physical and mental health providers.

Group homes will have a variety of staff that are either permanently assigned to the home such as frontline workers or itinerant staff including psychologists, social workers, nurses, occupational therapists, recreational therapists, ABA specialists etc. The provision of services will vary from system to system. There are no universally set standards for the provision of community-integrated residential services for adults with autism. This will vary from state to state.

Treatment Procedures

Challenges to Institutionalization

One of the major reasons why institutionalization was abandoned as the major policy for housing people with disabilities was the public exposure of horrific abuses which were occurring in these institutions. Geraldo Rivera's exposé on the Willowbrook State School, a school in Staten Island for people with developmental disabilities, helped to raise public awareness about this issue. The school was filthy, overcrowded, unsanitary, and rife with physical and sexual abuse. In the words of one scholar, “Willowbrook (and by implication, other residential facilities of its kind) resembled concentration camps. Lacking

cleanliness, privacy, care, affection, and education, Willowbrook would soon become the nation's touchstone for publicly funded abuse and neglect” (Rothman and Rothman 2004, p. 445). A successful class action against New York State followed this public exposure of inhumane treatment.

Another important element of the Willowbrook case was the involvement of a group of scholars from Syracuse University. One leading scholar, Wolf Wolfensberger, developed a theory of “normalization” which suggested that people with disabilities should be afforded every right that other citizens have – they should attend regular schools, live in normal houses in regular communities, and be included in community activities such as leisure, recreation, and sporting events. This philosophy of normalization became a springboard for one section of the disability rights movement who strongly believed that institutions must be dismantled and their residents should be returned to the community. Institutions had become like incarceration, and they were totally inappropriate for people with disabilities, who deserved every opportunity to live a normal life in the community. In the context of the abuses occurring at Willowbrook, Wolfensberger and other scholars suggested that the residents of Willowbrook be immediately placed in group homes instead of the institution (Rothman and Rothman 2004).

The key principle of normalization has been summarized in the following way: “. . . the most explicit and highest goal of normalization must be the creation, support, and defense of *valued social roles* for people who are at risk of social devaluation” (Wolfensberger 2004, p. 43). The philosophy of normalization was later re-named “Social Role Valorization” and became an incredibly popular approach to disability, spurring on community-integrated residential living and giving a sense of what residential inclusion should look like.

Wolfensberger identified three consequences of the devalued social roles which had been attributed to people with disabilities. First, he said that mistreatment is an effect of social devaluation. This was his explanation for the abuse

experienced by disabled people: their devalued social roles encouraged people to treat them in ways that did not recognize their dignity. Second, he said that negative treatment is connected to the forms of devaluation: when people with disabilities are devalued as human beings and regarded as animals, they were put in cages in institutions. Similarly, people who are regarded as a menace will be surrounded by enclosures marked by locks, barred windows, fences, and so on. Third, he said that how people are treated greatly affects how they behave: when people are continuously treated as “deviant,” they will tend to adopt a deviant identity, but if they are treated with respect and dignity and are accorded valued social roles, they will adapt accordingly (Wolfensberger 2004). The implication of this argument was that people with disabilities needed to be located in the regular community – where people with valued social roles can be found – and that any institutions or patterns of behavior which diminished their humanity must be removed.

Wolfensberger believed that addressing the devalued social image of people with disabilities should occur alongside efforts to improve their “competencies.” He suggested that these two processes were interconnected:

A person who is competency-impaired is highly at risk of becoming seen and interpreted as of low value, thus suffering image-impairment; a person who is image-impaired is apt to be responded to by others in ways that impair/reduce his/her competency. . . a person whose social image is positively valued is apt to be provided with experiences, expectancies and other life conditions which generally will also increase his/her competencies, and a person who is highly competent is also more apt to be imaged positively. (Wolfensberger 2004, p. 45)

In conjunction with Social Role Valorization, an approach to evaluating community residences and services was developed entitled “PASSING,” which stood for “Program Analysis of Service Systems’ Implementation of Normalization Goals.” PASSING involved a questionnaire which could be used to evaluate the extent to which residences and services promoted valued social roles. They were evaluated according to whether the following features of the residence

or service enhanced the social role and competencies of people with disabilities: the physical setting of the buildings, the groupings and relationships built into the process, the ways in which time was used and activities were structured, and other miscellaneous areas (Syracuse University Training Institute for Human Service Planning Leadership and Change Agency 2007).

At the same time as the philosophy of normalization and Social Role Valorization was being developed, another key social change was occurring. The disability rights movement, modeled after the African American civil rights movement, was beginning to develop and assert the right to independent living. The first Center for Independent Living was formed in Houston in 1972, and Berkeley and Boston developed their Centers for Independent Living in 1974. Berkeley developed its national reputation from its unique perspective – emphasizing peer counseling, legal assistance, advocacy on access issues, and leadership by people with disability. For instance, disability advocates in Berkeley were able to get curb cuts installed around their city in the 1970s to make the whole community more inclusive and accessible (Fleischer and Zames 2001).

The development of the self-advocacy movement by people with disabilities represented a fundamental shift in the ways in which disability should be understood. No longer were doctors, social workers, welfare workers, bureaucrats, or allied health professionals considered the “experts” on disability; people with disabilities were claiming this role for themselves. Self-advocacy not only occurred among people with physical disabilities; people with developmental disabilities also formed organizations such as People First where they asserted their rights to make important decisions about their own lives (Shapiro 1994).

There were significant differences as well as similarities between the approaches of the emerging disability rights movement and those committed to the principles of Social Role Valorization. One primary goal of the disability rights movement was to remove barriers in the environment – whether those barriers are physical barriers such as steps or attitudinal barriers such as prejudice. But the assertion that people with disabilities must

be in charge of the entire process was not a part of the Social Role Valorization philosophy. It centered on human service and welfare professionals; the disability rights movement mobilized people with disabilities themselves (Linton 1998). Additionally, Social Role Valorization suggested that people with disabilities try to conform to socially valued roles; the disability rights movement asserted a completely different set of values from the rest of society and argued that society should conform to those values, not vice versa. Additionally, Social Role Valorization assumed that disability was generally a negative experience which should be minimized in order to allow the person to fit in to society; the disability rights movement began to promote disability pride, which emphasized and took pride in their differences rather than minimizing them (Sherry 2006).

The development of the disability rights movement had major implications for the ways in which community residential programs responded to the needs of people with disabilities. For instance, they had to begin with the assumption that people with disabilities are capable and did not need to be “fixed” or changed. Additionally, residential services need to provide support to enable people with disabilities, including people with cognitive and developmental disabilities, to control their own lives. Self-determination was now a key component of residential decision making (Mackelprang and Salsgiver 1999).

The deinstitutionalization process, and the growth of the disability rights movement, helped make independent living and supported living in the community a reality for many people with disabilities. Rather than being isolated from their friends and families in institutions, people with disabilities are now living in the community. Social inclusion, including community integration, allows people to make friendships and develop relationships with others and is a cornerstone of equal rights for people with disabilities.

Efficacy Information

The efficacy of community-integrated residential services is difficult to quantify because of the

multiple domains these services impact upon. It appears that these services were effective in moving people with autism out of a warehousing situation (as was the case in the large-scale congregate care) and into the greater community. It is a widely held belief that this move decreased the amount of abuse and neglect that was inflicted upon individuals with autism in large psychiatric hospitals. Likewise, it is believed that the deinstitutionalization movement allowed individuals with autism to receive more appropriate and autism-specific interventions that were empirically based. This led to better outcomes for individuals on the spectrum.

Community-integrated residential services have been effective in alleviating caregiver overload and burn-out. These services have also been effective when families have been overwhelmed with the behavior of the individual (e.g., assaultive or wandering behavior) or when the individual with autism’s parents have become too old and infirmed to care for their adult child. Further discussions regarding the efficacy of the services are dependent upon the particular outcome one is focusing upon.

Outcome Measurement

Outcome measurement is difficult to define in a traditional sense of a single intervention. Community-integrated residential services for adults with autism provide a variety of interventions across multiple domains of a person’s life. Some of the outcomes worth assessing are (1) the level of independence a person attains and the amount of support and supervision he or she might need; (2) the ability of the individual to navigate through the community and use mass transit; (3) the ability of the person to self-advocate when dealing with social service agencies, government entitlement programs, and health-care providers; (4) the skills necessary to manage money, pay bills, and purchase items at a local store; and (5) the frequency and quality of the person’s participation in community events, entertainment, and cultural festivities.

The literature is replete with references to a person being on a spectrum or a continuum when describing their abilities and deficits. Residential services likewise can be thought of as being on a continuum. On one end of the continuum is large-scale congregate care which is generally reserved for the individuals who are most severely impacted by symptoms of autism or have behaviors that prevent them from being in a less restrictive setting (e.g., aggressive/assaultive behavior or self-injurious behavior). On the other end of the continuum is an individual who lives in the community with no formal organization supporting his or her independence. This is done simply through informal support networks (e.g., friends, family, spouse, etc.). In between the two extremes of the continuum of care are group homes, supervised apartments where social service agency staff actually live in the apartment complex and are available 24 h a day/365 days a year for emergencies, and finally a situation where the individual with autism lives completely integrated within the community and social service agencies drop in periodically to help with bill paying, home maintenance issues, and possibly assisting in dealing with in-home emergencies (e.g., what to do if the circuit breaker is tripped; what do if a smoke detector is going off, how and when to use a fire extinguisher, etc.).

An adult with autism may move up and down the continuum of care depending upon where he or she is in the life cycle and how much intervention he or she receives to mitigate the symptoms of autism and any comorbid disorders. The ultimate outcome for any individual on the spectrum is to live in a residential setting that maximizes his or her independence and autonomy while simultaneously ensuring a reasonable level of safety.

Qualifications of Treatment Providers

The qualifications of treatment providers providing community-integrated residential services for adults with autism vary widely. This is a reflection of a variety of state laws governing the provision of residential services to persons with

developmental disabilities. The treatment providers may be state employees working for the Office for Persons with Developmental Disabilities, or they may be employees of a private not-for-profit social service agency. There are for-profit entities that provide care for persons with autism. When looking for a community-integrated residential placement for an adult with autism, consumers should ask where the agency receives its auspice. Who licenses the facility? Who inspects and regulates facility? How often are they inspected? These are all important questions to ask prior to placing an individual with autism into a group home or similar type of facility.

See Also

► [Transition Planning](#)

References and Reading

- Fleischer, D. Z., & Zames, F. (2001). *The disability rights movement: From charity to confrontation*. Philadelphia: Temple University Press.
- Kerr, A., & Shakespeare, T. (2002). *Genetic politics from eugenics to genome*. Cheltenham: New Clarion Press.
- Lifton, R. J. (2000). *The Nazi doctors: Medical killing and the psychology of genocide*. Jackson: Basic Books.
- Linton, S. (1998). *Claiming disability*. New York: New York University Press.
- Mackelprang, R., & Salsgiver, R. (1999). *Disability: A diversity model approach in human service practice*. Pacific Grove: Brooks/Cole Publishing.
- Noll, S., & Trent, J. W. (2004). Introduction. In S. Noll & J. W. Trent (Eds.), *Mental retardation in America: A historical reader* (pp. 1–19). New York: New York University Press.
- Rothman, D. J., & Rothman, S. M. (2004). The litigator as reformer. In S. Noll & J. W. Trent (Eds.), *Mental retardation in America: A historical reader* (pp. 445–465). New York: New York University Press.
- Shapiro, J. (1994). *No pity: People with disabilities forging a new civil rights movement*. New York: Random House.
- Sherry, M. (2006). *If I only had a brain: Deconstructing brain injury*. New York: Routledge.
- Snyder, S., & Mitchell, D. (2006). *Cultural locations of disability*. Chicago: The University of Chicago Press.
- Syracuse University Training Institute for Human Service Planning Leadership and Change Agency. (2007, February). Overview of “Passing”: A tool for analyzing service quality according to social role valorization

criteria. Retrieved 23 May 2011 from http://www.srvip.org/PASSING_overview005.pdf

Wolfensberger, W. (1975). *The origin and nature of our institutional models*. Syracuse: Human Policy Press.

Wolfensberger, W. (2004). Social role valorization: A new term for the principle of normalization. In D. R. Mitchell (Ed.), *Special educational needs and inclusive education: Systems and contexts* (pp. 42–50). New York: Routledge Farmer.

Comorbid ASD

► Risk of ASD in Individuals with Down Syndrome

Comorbidity

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Definition

The presence of a second (comorbid) condition in association with a previous one.

Historical Background

The issue of comorbidity with autism has assumed increasing importance in recent years; it is intimately related to other issues including more general topics in diagnosis and classification as well as the attempt to identify meaningful approaches to subgrouping/subtyping autism. Clearly having any serious disability – such as autism or intellectual disability – only acts to increase vulnerability to other problems. In the past, as in the field of intellectual disability, the problem was complicated by the tendency of autism to “overshadow” other difficulties (Rutter 1994; Volkmar and Klin 2005).

Current Knowledge

Autism has been reported to co-occur with various other developmental, psychiatric, and medical conditions; however, often these associations are based on reports of single, or at best a handful of, cases and often fail to address the more central question of whether associations are observed at greater than chance levels. This has been strongly illustrated in the literature on associations of medical conditions with autism where early efforts, based largely on case reports, suggested hundreds, if not thousands, of such associations (Gillberg 1992), but more careful controlled studies suggested that this list is much smaller, with the strongest associations observed with seizure disorder and a handful of strongly genetic conditions (Rutter et al. 1994).

Evolving diagnostic concepts and research findings have sometimes clarified the significance or meaning of possible associations. For example, Kanner’s original impression (1943) that persons with autism had normal intellectual potential has been shown to be incorrect; although the pattern of cognitive and adaptive abilities in autism is unusual, for many of children with autism, overall scores on cognitive testing are stable within the mentally retarded range (Goldstein et al. 2008; Lord and Schopler 1989). On the other hand, a substantial group of persons with autism have cognitive abilities in the average or above average range and, with early diagnosis and intervention, the overall outcome (and levels of cognitive ability in adulthood) appears to be improving (Howlin 2007), suggesting that perhaps this association reflects an aftereffect of autism rather than one more essentially connected in an etiological sense to autism. On the other hand, seizure disorders of various types are frequently associated with autism if the latter is strictly defined occurring in perhaps 20–25% of cases.

Issues relating to comorbidity for other psychiatric conditions are complicated for several reasons. Firstly there is a major difference between approaches to diagnosis in DSM-IV and ICD-10. While both systems attempt to be comprehensive in coverage, ICD-10 comes from a nosological

tradition of searching for a single, parsimonious diagnostic label (i.e., a “top-down” approach more focused on syndromes rather than symptoms). Conversely DSM-IV and its predecessors have often seemed to encourage multiple diagnoses (a “bottom-up” orientation that starts with symptoms and then moves toward categories) (Ghaziuddin et al. 1992; Rutter 2006; Volkmar and Woolston 1997; Volkmar and Woodbury-Smith 2007). Clearly no single diagnostic label conveys the totality of the individual’s difficulties, and while the multiaxial approach employed in both DSM and ICD helps address this issue, tensions do remain regarding issues of additional diagnoses for persons with autism. Other issues arise given the changes that occur with developmental level and age. Issues of assessment are also a consideration, for example, what would it mean to assess thought disorder in a mute individual with autism?

Other issues arise given that diagnostic systems like DSM-IV and ICD-10 strive for logical consistency in their approach, that is, typically some form of hierarchical process is used so as not to reduce diagnosis entirely to the level of presence/absence of individual symptoms, for example, since stereotyped behaviors are a diagnostic feature of autism, it seems nonsensical to also give a diagnosis of stereotyped movement disorder to most people with autism.

Given these considerations, it is important to interpret reported associations with some caution, particularly if the association is based on case reports in large part. That being said, it is the case that a veritable host of conditions have been reported in individuals with autism. These include hyperactivity, obsessive-compulsive phenomena, self-injury, tics, and affective symptoms (Brasic et al. 1994; Ghaziuddin and Greden 1998; Ghaziuddin and Tsai 1991; Ghaziuddin et al. 1992, 1995; Jaselskis et al. 1992; Kerbeshian et al. 1990; McDougale et al. 1995; Realmuto and Main 1982; Rapoport et al. 2009; White and Schry 2011). Particularly in older and more able individuals, the presence of anxiety and mood

problems is quite frequent (White and Schry 2011). On the other hand, some disorders, notably schizophrenia, do not appear to be significantly increased in samples of individuals with autism (Volkmar and Cohen 1991).

In some cases, possible associations might have significance for treatment. For example, given the use of new pharmacological treatments such as the SSRIs, a possible overlap of autism with obsessive-compulsive disorder (OCD) would be of interest. Clearly phenomena suggestive of obsessions or compulsions are frequent (although highly variable depending on sample selected (Baron-Cohen 1989; Mack et al. 2010; Rumsey et al. 1985)) and may have implications for treatment (McDougale et al. 1995). However, as Baron-Cohen (1989) observed, it does appear that it is not possible to simply equate ritualistic phenomena of autism with the obsessions and compulsions more typical of OCD. Similarly reports of association with Tourette’s syndrome (chronic motor and vocal tics) are of interest, although data on whether or not such associations are more likely than would be expected based on chance alone have yet to be collected.

In recent years, considerable interest has centered on the association of autism with attention deficit disorder (ADD)/attention deficit hyperactivity disorder (ADHD) and the possible implications of such an association for treatment. Early views, as in DSM-III-R, questioned this association given the high frequency of attention problems in children with autism (particularly for social tasks) and what appeared to be, in general, a poor response to stimulant medications, although more recent research has questioned this view while also noting the potential for higher levels of adverse reactions in this population (Aman et al. 2008; Jahromi et al. 2009). The issue may have even greater importance among children with the broader autism spectrum (Barkley et al. 2002); attempts have been made to delineate specific subgroups of cases with problems in social interaction and attention (and other features, e.g., Hellgren et al. 1994).

Future Directions

Clearly the issue of comorbidity is an important one and one where our understanding is likely to significantly shift with advances in research. Complications arise from the dual, and sometimes competing, needs for research-based diagnostic approaches and one focused more on service delivery. In the United States in particular, issues of diagnosis may have very significant implications for eligibility for services. The ability in the coming years to more robustly identify end phenotypes may help resolve some of the issues.

Issues of comorbidity have become more relevant as overall outcome in autism has improved (in part because it becomes easier to document comorbid conditions using more traditional and conventional assessments). A growing awareness of this problem will likely produce a substantial increase in work in this area in the future.

See Also

- [Diagnosis and Classification](#)
- [DSM-IV](#)
- [ICD 10 Research Diagnostic Guidelines](#)
- [Secondary Handicapping Conditions](#)

References and Reading

- Aman, M. G., Farmer, C. A., Hollway, J., & Arnold, L. E. (2008). Treatment of inattention, overactivity, and impulsiveness in autism spectrum disorders [Research Support, N.I.H., Extramural Review]. *Child & Adolescent Psychiatric Clinics of North America*, 17(4), 713–738. vii.
- Barkley, R. A., Cook, E. H., Dulcan, M., Campbell, S., Prior, M., Atkins, M., et al. (2002). Consensus statement on ADHD. *European Child & Adolescent Psychiatry*, 11(2), 96–98.
- Baron-Cohen, S. (1989). Do autistic children have obsessions and compulsions? *British Journal of Clinical Psychology*, 28(Pt 3), 193–200.
- Brasic, J. R., Barnett, J. Y., Kaplan, D., Sheitman, B. B., Aisemberg, P., Lafargue, R. T., et al. (1994). Clomipramine ameliorates adventitious movements and compulsions in prepubertal boys with autistic disorder and severe mental retardation. *Neurology*, 44(7), 1309–1312.
- Ghaziuddin, M., & Greden, J. (1998). Depression in children with autism/pervasive developmental disorders: A case-control family history study. *Journal of Autism and Developmental Disorders*, 28(2), 111–115.
- Ghaziuddin, M., & Tsai, L. (1991). Depression in autistic disorder. *British Journal of Psychiatry*, 159, 721–723.
- Ghaziuddin, M., Ghaziuddin, N., et al. (1992). Comorbidity of autistic disorder in children and adolescents. *European Child & Adolescent Psychiatry*, 1(4), 209–213.
- Ghaziuddin, M., Alessi, N., & Greden, J. F. (1995). Life events and depression in children with pervasive disorders. *Journal of Autism and Developmental Disorders*, 25(5), 495–502.
- Gillberg, C. (1992). Subgroups in autism: Are there behavioural phenotypes typical of underlying medical conditions? *Journal of Intellectual Disability Research*, 36(Pt 3), 201–214.
- Goldstein, G., Allen, D. N., Minshew, N. J., Williams, D. L., Volkmar, F., Klin, A., et al. (2008). The structure of intelligence in children and adults with high functioning autism [Comparative Study Research Support, Non-U.S. Gov't Research Support, U.S. Gov't, Non-P. H.S.]. *Neuropsychology*, 22(3), 301–312.
- Hellgren, L., Gillberg, I., Bagenholm, A., & Gillberg, C. (1994). Children with deficits in attention, motor control and perception (DAMP) almost grown up: Psychiatric personality disorders at age 16 years. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, 35(7), 1255–1271.
- Howlin, P. (2007). *The outcome in adult life for people with ASD. Autism and pervasive developmental disorders* (pp. 269–306). New York: Cambridge University Press.
- Jahromi, L. B., Kasari, C. L., McCracken, J. T., Lee, L. S. Y., Aman, M. G., McDougale, C. J., et al. (2009). Positive effects of methylphenidate on social communication and self-regulation in children with pervasive developmental disorders and hyperactivity [Randomized Controlled Trial Research Support, N.I.H., Extramural Research Support, Non-U.S. Gov't]. *Journal of Autism & Developmental Disorders*, 39(3), 395–404.
- Jaselskis, C. A., Cook, E. H., Jr., Fletcher, K. E., & Leventhal, B. L. (1992). Clonidine treatment of hyperactive and impulsive children with autistic disorder. *Journal of Clinical Psychopharmacology*, 12(5), 322–327.
- Kereshian, J., Burd, L., Randall, T., Martsolf, J., & Jalal, S. (1990). Autism, profound mental retardation and atypical bipolar disorder in a 33-year-old female with a deletion of 15q12. *Journal of Mental Deficiency Research*, 34(Pt. 2), 205–210.
- Lord, C., & Schopler, E. (1989). The role of age at assessment, developmental level, and test in the stability of intelligence scores in young autistic children. *Journal*

of *Autism and Developmental Disorders*, 19(4), 483–499.

- Mack, H., Fullana, M. A., Russell, A. J., Mataix-Cols, D., Nakatani, E., & Heyman, I. (2010). Obsessions and compulsions in children with Asperger's syndrome or high-functioning autism: A case-control study [Comparative Study]. *Australian & New Zealand Journal of Psychiatry*, 44(12), 1082–1088.
- McDougle, C. J., Kresch, L. E., Goodman, W. K., Naylor, S. T., Volkmar, F. R., Cohen, D. J., et al. (1995). A case-controlled study of repetitive thoughts and behavior in adults. *American Journal of Psychiatry*, 152(5), 772–777.
- Rapoport, J., Chavez, A., Greenstein, D., Addington, A., & Gogtay, N. (2009). Autism spectrum disorders and childhood-onset schizophrenia: Clinical and biological contributions to a relation revisited. *Journal of the American Academy of Child & Adolescent Psychiatry*, 48(1), 10–18.
- Realmuto, G. M., & Main, B. (1982). Coincidence of Tourette's disorder and infantile autism. *Journal of Autism and Developmental Disorders*, 12(4), 367–372.
- Rumsey, J. M., Rapoport, J. L., & Sceery, W. R. (1985). Autistic children as adults: Psychiatric, social, and behavioral. *Journal of the American Academy of Child Psychiatry*, 24(4), 465–473.
- Rutter, M. (1994). Comorbidity: Meanings and mechanisms. *Clinical Psychology: Science and Practice*, 1(1), 100–103.
- Rutter, M. (2006). Autism: Its recognition, early diagnosis, and service implications. *Journal of Developmental and Behavioral Pediatrics*, 27(2 Suppl), S54–S58.
- Rutter, M., Bailey, A., Bolton, P., & Le Couteur, A. (1994). Autism and known medical conditions: Myth and substance. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, 35(2), 311–322.
- Volkmar, F. R., & Cohen, D. J. (1991). Comorbid association of autism and schizophrenia. *The American Journal of Psychiatry*, 148(12), 1705–1707.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions, vol 1. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 5–41). Hoboken: Wiley.
- Volkmar, F. R., & Woodbury-Smith, M. (2007). *Clinical diagnosis of autism. Clinical manual for the treatment of autism* (pp. 1–26). Arlington: American Psychiatric Publishing.
- Volkmar, F. R., & Woolston, J. L. (1997). Comorbidity of psychiatric disorders in children and adolescents. ST- An Einstein psychiatry publication, No. 14. In S. Wetzler & W. C. Sanderson (Eds.), *Treatment strategies for patients with psychiatric comorbidity* (An psychiatry publication, No. 14, pp. 307–322). New York: Wiley.
- White, S. W., & Schry, A. R. (2011). *Social anxiety in adolescents on the autism spectrum. Social anxiety in adolescents and young adults: Translating developmental science into practice* (pp. 183–201). Washington, DC: American Psychological Association.

Comparison of Diagnostic Instruments for ASD

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Introduction

The present entry will present a brief introduction to diagnostic instruments commonly used in clinical assessments (and in research) of ASD and a comparison of these instruments. It is essential to distinguish between instruments designed to identify potential cases of autism spectrum disorder (ASD) for further clinical assessment (i.e., screening instruments for parents, teachers, pediatricians, and general practitioners) (Øien et al. 2019) and instruments designed to guide clinical judgment when an individual is referred for ASD specific assessment. Most commonly, a diagnostic includes a wide range of instruments to provide a developmental and behavioral context of understanding the symptom pattern presented at the assessment. Examples are the assessment of the individual's cognitive skills, adaptive skills. However, this entry is focused on instruments measuring autism-specific symptoms and guiding clinicians in the determination of ASD or not. It is of importance that trained professionals providing diagnostic assessments for individuals suspected of ASD, utilize appropriate measures, and are aware of the potentials and pitfalls of these instruments.

Current Knowledge

Since the introduction of the DSM-III and DSM-III-R in the 1980s (Volkmar et al. 1988), multiple

ASD specific diagnostic instruments were developed. Many of these instruments are validated in specific populations and cultures (Øien and Nordahl-Hansen 2018). As presented in the introduction, this entry will provide information about some of the most commonly used ASD-specific diagnostic instruments and the performance of these instruments. Since the introduction of the DSM-III, multiple instruments for diagnosing ASD have been developed.

However, these instruments must not alone serve as a conclusion for clinical decision-making but rather as tools used for guiding clinical judgment.

One of the difficulties with the development of diagnostic instruments for ASD is the heterogeneity of onset and patterns of symptoms and the variability in the severity of both symptoms, IQ and adaptive skills (Klin et al. 2005). This causes significant challenges in the development of one instrument expected to perform well in identifying ASD across the continuum. For more in-depth information and psychometric properties about the various diagnostic instruments Corsello (2013) (Encyclopedia entry on diagnostic instruments). Examples of the most used ASD-specific instruments are:

Interview-Based Instruments

In terms of interview-based instruments, the present entry highlights two, whereas one (ADI-R; Lord 1994) is the most commonly used diagnostic interview.

Diagnostic Interview for Social and Communication Disorders (DISCO; Wing et al. 2002)
It is constructed as a semi-structured interview of parents (primary provider) to diagnose autistic disorder, Asperger’s syndrome, psychiatric disorders, and other developmental disabilities. It is extensive, with over 300 items, whereas 93 items are used for diagnostic guidance. The DISCO is scored from 0 to 2, whereas a lower score indicates more abnormal development.

Instrument item rating	Value
0	Severe problem
1	Minor problem
2	No problem

The scale is furthermore divided into different domains:

Domain	Number of items
Social interaction	38
Communication	15
Stereotyped behaviors	29

The Autism Diagnostic Interview-Revised (ADI-R; Lord 1994)

The ADI-R is a semi-structured interview that contains 93 items concerning behavior at different ages.

The ADI-R has an opposite scoring than the DISCO and is scored as follows: which are scored between 0 and 3, plus additional scoring possibilities. The rating scale includes the following scoring options:

Instrument item rating	Value
0	The behavior of the type specified in the coding is not present
1	The behavior of the type specified is present in an abnormal form, but not sufficiently severe or frequent to meet the criteria for two
2	Definite abnormal behavior
3	The extreme severity of the specified behavior
7	Definite abnormality in the general area of the coding, but not of the type specified
8	Not applicable
9	Not known or asked

The ADI-R is validated and includes four different domains: reciprocal social interaction,



language and communication, stereotyped repetitive behaviors or interests, and age-of-onset criteria.

Cutoff scores are for each domain as following:

Domain	Cutoff score
Social interaction	10
Language and communication	8 (verbal) 7 (non-verbal)
Stereotyped repetitive behaviors or interests	3
Age of onset	1

It is essential to keep in mind that these criteria are tailored to the criteria in ICD-10 or DSM-IV-TR diagnosis of “F84.0 Childhood autism” (WHO 1992) or “299.00 Autistic Disorder” (APA 2002).

Også laget en småbarnsalgoritme for bruk ved ADI-R.

Observation-Based Instruments

The Autism Diagnostic Observation Schedule
The ADOS-G was introduced in 1989 (Lord et al. 1989), later updated to the ADOS-2 (Lord et al. 2012) and is a semi-structured, observer-based instrument that can only be performed by a trained clinician. The procedure is constructed as a standardized play and communication sessions, and the observed behavior is rated 0–3. ADOS2 consists of four different modules, and which one to use is guided by the individual’s level of expressive language and chronological age.

Module	Usage	Number of items
1	Little or no phrase speech	29
2	Phrase speech, but not fluently	28
3	Fluent children and young adolescents	28
4	Fluent adolescents and adults	31

Comprising five different domains:

- Domain
- Language and communication
- Reciprocal social interaction
- Imagination
- Stereotyped behaviors and restricted interests
- Other abnormal behaviors

Gotham et al. (2007) proposed revised algorithms for module 1–3, to increase specificity in determining an ASD diagnosis in lower functioning populations, that was taken into consideration in the ADOS-2 (Lord et al. 2012).

Comparison of Diagnostic Instruments

The diagnostic process can be time-consuming depending on the age of the child/adolescence, complexity of development/difficulties, and presence of comorbidity. Comorbid conditions might cause the symptom expression to be higher as many of the difficulties associated with ASD is not ASD specific. As mentioned, clinicians rely on different tools such as the ADOS and the ADI-R to assist the clinicians in the diagnostic process. While there is a range of instruments designed for autism-specific diagnostic assessments, few of the instruments perform excellently in psychometric properties alone. Current knowledge indicates that the use of autism-specific interviews with parents and caregivers lack the psychometric properties alone to be regarded as “good enough” on their own. However, the combined use of the ADOS and ADI-R are regarded as the gold standard of diagnostic instruments but do require extensive clinical expertise and reliability (Falkmer et al. 2013). According to Falkmer et al. (2013), the ADOS and ADI-R showed that these instruments performed better than any of the other instruments in terms of sensitivity and specificity, i.e., the accurately identifying children with ASD and distinguishing them from children with other developmental issues, combined showing a classification rate of 88%. However, it is crucial to address the fact that diagnostic instruments areas the term indicate: instruments used in a more extensive assessment. Interpreting the results from any

instrument should be conducted with caution, and sound clinical judgment should be penalized in the diagnostic process. Determining ASD by an instrument should be disregarded, and the clinician should always use the instrument as an aid in securing the optimal diagnostic result possible.

In terms of diagnostic performance, a study utilizing the Norwegian Mother and Child cohort, and its nested sub-study the Autism Birth Cohort revealed that the ADI-R cutoffs demonstrated good specificity (SP), while showing lower sensitivity (SE), i.e., missing approximately 43% of toddlers where parents did not have any specific concern about autism spectrum disorder. Furthermore, the same study by Havdahl et al. (2017) revealed that the ADI-R and the ADOS scores agreed well with clinical diagnoses (AOC > 0.85). However, as Havdahl et al. (2017) highlight, the different cutoffs for the ADI-R were needed in the presence or absence of parental concern to achieve comparable SE and SP.

Current results from the ADI-R and ADOS highlight the importance of good clinical judgment and the importance of taking parental concern seriously in the diagnostic process and when interpreting scores from parent endorsed instruments such as the ADI-R and consequently the DISCO. As presented in Havdahl et al. (2017), combining instruments such as the ADI-R and the ADOS provides the optimal prediction and clinical guidance in diagnostic processes (Havdahl et al. 2017).

Future Directions

There is a pressing need to understand how the various instruments, not only the ADOS and the ADI-R, perform across various populations, ethnicities, and cultures. However, it also how the instruments perform in terms of each other. The ADOS and the ADI-R are currently regarded as gold standard instruments, but the gold standard should be considered as relative rather than absolute (Øien et al. 2018). Further research is needed to ascertain how well the parent endorsed instruments are performing in cases without parental concern and for children within the average

range IQ. It is crucial to think about diagnostic instruments such as 3di, DISCO, ADOS, and ADI-R as a supplement and helpful tool in the diagnostic process rather than a tool providing a conclusive decision regarding diagnosis.

See Also

- ▶ ADI-R
- ▶ ADOS
- ▶ Diagnostic Instruments in Autistic Spectrum Disorders
- ▶ Screening Instruments for ASD

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (DSM-IV-TR)* (4th ed.). Washington, DC: American Psychiatric Association.
- Corsello, C. (2013). Diagnostic instruments in autistic Spectrum disorders. In F. R. Volkmar (Ed.), *Encyclopedia of autism spectrum disorders*. New York: Springer.
- Falkmer, T., Anderson, K., Falkmer, M., & Horlin, C. (2013). Diagnostic procedures in autism spectrum disorders: A systematic literature review. *European Child & Adolescent Psychiatry*, 22(6), 329–340. <https://doi.org/10.1007/s00787-013-0375-0>.
- Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The autism diagnostic observation schedule: Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders*, 37(4), 613–627.
- Havdahl, K. A., Bishop, S. L., Surén, P., et al. (2017). The influence of parental concern on the utility of autism diagnostic instruments. *Autism Research*, 10(10), 1672–1686. <https://doi.org/10.1002/aur.1817>.
- Klin, A., Pauls, D., Schultz, R., & Volkmar, F. (2005). Three diagnostic approaches to Asperger syndrome: Implications for research. *Journal of Autism and Developmental Disorders*, 35(2), 221–234. [PUBMED: 15909408].
- Lord, C., Rutter, M., Goode, S., Heemsbergen, J., Jordan, H., Mawhood, L., et al. (1989). Autism diagnostic observation schedule: A standardized observation of communicative and social behavior. *Journal of Autism and Developmental Disorders*, 19(2), 185–212.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(5), 659–685.

- Lord, C., Luyster, R. J., Gotham, K., & Guthrie, W. (2012). *ADOS-2. Autism diagnostic observation schedule* (2nd ed.). Torrance: Western Psychological Services.
- Øien, R. A., & Nordahl-Hansen, A. (2018). *Bias in assessment instruments for autism. Encyclopedia of autism spectrum disorders*. New York: Springer.
- Øien, R. A., Nordahl-Hansen, A., & Schjølberg, S. (2019). *Screening instruments for ASD*. Springer Science+Business Media BV. https://doi.org/10.1007/978-1-4614-6435-8_102295-1. ISBN 978-1-4614-6435-8.s.
- Spitzer, R. L., Gibbon, M. E., Skodol, A. E., Williams, J. B., & First, M. B. (2002). *DSM-IV-TR casebook: A learning companion the diagnostic and statistical manual of mental disorders, text rev.* American Psychiatric Publishing, Inc.
- Stenberg, N., Schjølberg, S., Shic, F., et al. (2020). Functional outcomes of children identified early in the developmental period as at risk for ASD utilizing the the Norwegian mother, father and child cohort study (MoBa). *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-020-04539-8>.
- Volkmar, F. R., Bregman, J., Cohen, D. J., & Cicchetti, D. V. (1988). DSM-III and DSM-III-R diagnoses of autism. *The American Journal of Psychiatry*.
- Wing, L., Leekam, S. R., Libby, S. J., Gould, J., & Larcombe, M. (2002). The diagnostic interview for social and communication disorders: Background, inter-rater reliability and clinical use. *Journal of Child Psychology and Psychiatry*, 43(3), 307–325.
- World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organization.

Comparison of High-Functioning Autism and Asperger Syndrome

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Definition

Since Hans Asperger’s first description, through Lorna Wing’s translation and definition, to its introduction in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), Asperger syndrome has always aroused huge interest and debate, until vanishing in the DSM fifth edition (DSM-5).

The following entry will describe the history of Asperger syndrome, from the original description to the current diagnostic classifications, focusing on clinical profile, boundaries with high-functioning autism and nosographic implications.

Historical Background

In the same years of Leo Kanner’s description of infantile autism (Kanner 1943), Hans Asperger wrote a case report on autistic psychopathy (Asperger 1944), describing children with social-communication impairment, eccentric manners, unusual interests, and cognitive domains of hyperfunctioning. The American Psychiatric Association (APA) did not immediately recognize Autism as a distinct category: it was introduced as infantile autism in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III) and included within pervasive developmental disorders (PDD) in the DSM-III-R. In 1981, Wing resumed Asperger’s researches, renaming the autistic psychopathy as Asperger syndrome (AS) (Wing 1981). In 1989, the first diagnostic criteria for AS were proposed (Gillberg and Gillberg 1989; Szatmari et al. 1989), and in the 1990s, AS appeared in the fourth edition of DSM (DSM-IV) within PDD. The diagnosis of AS required at least two symptoms of social interaction impairment and one symptom of behavioral and interest restriction, a normal cognitive functioning, and the absence of significant general delay in language. Moreover, diagnostic criteria for autistic disorder should not be met (otherwise, autistic diagnosis should have precedence). This implied a differential diagnosis between AS and autism, especially the type without cognitive delay, also known as high-functioning autism (HFA). HFA is not a term used in the DSM, but it is commonly used to identify subjects diagnosed with autistic disorder (AD) or PDD-not other specified (PDD-NOS), with average or above average intellectual abilities (intelligent quotient, IQ, higher than 70). HFA differs from low-functioning autism (IQ lower than 70) in terms of clinical presentation, prognosis and need for support and assistance in daily

life. Since AS and HFA are both characterized by a normal cognitive functioning, there has been considerable debate over whether AS and HFA are distinct conditions, suggesting different etiological and neurobiological mechanism, or share a similar underlying neuropsychological functioning and should therefore be regarded as variants of a single disorder. According to Hans Asperger original description, his patients differ from those described by Kanner (Asperger 1944). Instead, Lorna Wing, translating Asperger's work, named the syndrome and Kanner's autism both part of an autistic continuum (Wing 1981). AS definition and its boundaries with HFA have been the topic of a growing body of literature, providing contradictory results: some authors considered the available evidences insufficient to establish the validity of AS as a syndrome distinct from HFA; others found quantitative rather qualitative differences between them. Sharma et al., in a review of 69 research studies, published from 1981 to 2010, defined "confuse and inconsistent" AS diagnosis, since it is based on criteria mostly overlapping with autism, from which though it could stand out for some fine differences in the nature of social interaction and communication (Sharma et al. 2012).

The unsolved confusion in defining AS criteria and the clinical overlap between HFA and AS led to its merging into one unifying category, on the assumption that they cannot be reliably differentiated from one another; the DSM-5 incorporated AS into autistic spectrum disorder (ASD), removing the previously discrete diagnostic presentation of PDD. According with members of the DSM-5 Neurodevelopmental Disorders Committee, the application of DSM-5 criteria demonstrated adequate sensitivity across all PDD subcategories, also including subjects denoted as cognitively higher functioning and AS (Huerta et al. 2012).

Current Knowledge

Irrespective of the DSM-5 changes, several studies (also published later than 2013) continue to debate about AS and its conceptualization within ASD (Volkmar and McPartland 2014).

Tsai and Ghaziuddin (2014), finding in 95 of 125 comparative studies quantitative and qualitative differences between AS and HFA, expressed their concern about the risk that with the single category of ASD, DSM-5 "moved forward into the past."

A recent study conducted a comparison between 150 children and adolescents with diagnosis of PDD, according to DSM-IV Text Revision (DSM-IV-TR) criteria, with an intelligent quotient average or above, among which 80 AS and 70 HFA (including patients with a diagnosis of autistic disorder or pervasive developmental disorder-not otherwise specified), in term of core clinical features, cognitive functioning, school learning abilities, and comorbidities (de Giambattista et al. 2019). Core clinical features were investigated using the Australian Scale for Asperger's Syndrome (ASAS) (Attwood 2006) and the Michigan Autism Spectrum Questionnaire (MASQ) (Ghaziuddin and Welch 2013). Administering ASAS, social abilities resulted impaired in both groups, even if HFA appeared more compromised above all in avoiding social contact. AS can show more motivation to socialize and desire for friendship, even if this does not translate into superior ability to make friends, because they appeared awkward, eccentric, one-sided, and insensitive. Emotional dysregulation was another common feature in AS and HFA, resulting in need for excessive amount of reassurance and problems especially regard to anger and anxiety management. Among communication abilities, both the conditions presented some deficits in reciprocity of conversation, eye-contact, and modulation of voice tone; nevertheless, over-precise and pedantic speech (characterized by overly formal speech, similar to an in-depth monologue about a topic of special interest, verbose and tangential, lack of the normal prosody, enriched by a sophisticated vocabulary) was significantly more frequent in AS and literal interpretation of comments more common in HFA. The first finding ("over-precise or pedantic speech") is unsurprising and represents one of the most typical clinical feature of AS, firstly described by Hans Asperger's original work (1944, in which

AS were called “little professors” for their way of talking), then included in the following proposed diagnostic criteria sets (Gillberg and Gillberg 1989; Szatmari et al. 1989) and confirmed by subsequent studies (Woodbury-Smith and Volkmar 2009). The second finding (“literal interpretation of comments”) may be related to the HFA worse cognitive and comprehension abilities, which made them less able in recognizing the speaker’s communicative intention and in social understanding, unlike patients with AS, who adopted intellectual strategies of compensation.

Delay in language development resulted significantly more common in HFA, even if also in AS, a mild language delay was reported. In the DSM-IV-TR, the “absence of clinically significant general delay in language (e.g., single words used by age 2 years, communicative phrases used by age 3 years)” was a relevant criterion for AS diagnosis. In contrast with this theoretical construct, extensive literature data showed that language delay could not be considered as a diagnostic exclusion criterion for AS because subtle language problems are expected in AS and also because the recognition of a language delay is not always enough reliable, since it is often dependent on parents recall. Clumsiness, that has been originally considered a distinctive feature of AS appeared indifferently present in AS like in HFA, as well as sensory abnormalities (such as unusual fear/distress due to light touch on skin or scalp, and to unexpected noises), restrictive pattern of interests and behaviors, and difficulties in adaptation to changes. The clinical profile of AS is less likely to include motor mannerism and preoccupation with parts of objects (defined “low order repetitive restricted behaviors”) while more often presented circumscribed interests that consumes a great deal of their time amassing information and facts (defined “high order repetitive restricted behaviors”).

About cognitive functioning, evaluated with Wechsler scales, AS showed Full Scale Intelligence Quotient mean values and sub-values significantly higher than HFA, without differences between verbal and performance sub-values. This overall better performance in AS represented a well-established mark of

differentiation between AS and HFA. Indeed, some authors identified a qualitatively distinct cognitive profile (characterized by a higher verbal IQ and a lower performance IQ and a reversed pattern in HFA), while others reported mixed cognitive patterns. The two conditions could likely be distinguished at a quantitative cognitive level, represented by FSIQ, rather than at a qualitative level, represented by specific cognitive profile. Nevertheless, as for intellectual disability, the presence of very different IQs could denote distinct levels of severity and clinical profile, in the same way a large discrepancy between AS and HFA intellectual scores could have a qualitative effect on general function.

Learning disorders resulted significantly more frequent in HFA, with the exception of dysgraphia, that emerged frequently in both groups, according to the original description of Hans Asperger (Asperger 1944), who described in his patients low performances in handwriting, in terms of forming letters and using the paper space. AS may not show any conspicuous learning disturbances, but an inhomogeneous school profile, characterized by talents in some school subjects, such as precocious learning to read, numerical abilities, or memory, while poor application and performances in other school subjects, perceived as less interesting.

The most prevalent comorbidity both in AS and HFA was attention deficit hyperactivity disorder (ADHD). An association between ASD and ADHD and other externalizing disorders have been reported and supported by clinical population research and twin studies, suggesting substantial genetic overlap (Taylor et al. 2015). Considering that comorbidity between these disorders was a relevant and frequent occurrence, differently from the previous edition, DSM-5 allowed the diagnosis of ADHD in the context of an ASD. Subjects with AS displayed more anxiety and depressive symptoms compared to HFA. This could be explained with AS higher cognitive and communicative levels, that made them more able in introspection, more “active but odd” (Ghaziuddin 2008) in relations attempts, more conscious of social difficulties and so more vulnerable to affective disorders.

Overall, among AS and HFA, differences in neurodevelopmental trajectories and subsequent need for health service could be recognized. Clinical signs in AS might be subtle and camouflaged in preschoolers and could become clear at the age of 7 or 8 years, leading to a later diagnosis.

Another focus of debate is the effect of DSM-5 criteria application on high-functioning ASD. Recent studies indicated a percentage between 50% and 75% of individuals maintaining diagnosis under DSM-5 criteria (Smith et al. 2015). A so wide concordance range could reflect diagnostic methodologies adopted in different studies: the gold standard diagnostic instruments for the assessment of autistic disorder could have low sensitivity when applied to high-functioning subjects. Clinical judgment of qualified professional experts and the use of specific rating scales, designed on the clinical characteristics of HFA and AS, may contribute to reduce the risk of underdiagnosing. According with this perspective, the study of de Giambattista et al. (2019) provided evidences that using brief and simple screening instruments such as ASAS and MASQ, it is more likely to recognize specific features of HFA functioning: indeed, 97.3% of the subjects, previously diagnosed according to DSM-IV-TR, met DSM-5 criteria for ASD and administrating MASQ different scores in HFA and AS were found. Moreover, when DSM-5 specifiers of severity levels were applied to the sample, HFA were more frequently classified in Level 2 (“requiring substantial support”) than AS, while AS were more frequently classified in Level 1 (“requiring support”).

Future Directions

Considering available evidences, it is likely to assume that a complex aged-related different developmental profile between HFA and AS exists, characterized by quantitative and subtle qualitative differences such as language development and communication style, cognitive functioning, academic achievement, and comorbidities profile.

Consistently with clinical findings, biological patterns of differentiation were found: a recent genome-wide association meta-analysis revealed evidences for a qualitatively different genetic architecture between subtypes of ASD (Grove et al. 2019), differences in neuroimaging field, in particular in gray matter distribution (Bi et al. 2018), and in EEG connectivity patterns (Luckhardt et al. 2014).

While acknowledging the merit of the DSM-5 to have solved lots of previous classification discordances and “splitting” approaches (Volkmar and McPartland 2014), it is necessary to consider the risk that defining autism using the umbrella term ASD, could hide its evident heterogeneity (Lai et al. 2013) and particularly that in the severity specifier “Level 1” of the DSM-5, many phenotypic variances could be squeezed making this level very heterogeneous.

It is likely that recognition of different “nuances” of the spectrum may contribute to tailor research, clinical, and assistential approaches. For example, since language development and academic abilities play a central role in global functioning during the first years of life, HFA require more support in terms of rehabilitative treatments and school educational needs than AS, while in middle childhood and adolescence, AS require more support in terms of psychotherapy, presenting more emotional and mood problems than HFA.

Several expert researchers and specialists in the field have invited to reflect about the consequences of removing AS from the available classification, just when it was better understood by the society: the lack of a “label” in which subjects recognized themselves might cause confusion and damage the identity sense of the yet established “Aspie community” (“Aspie” is an affectionate term often used to describe a person with an AS diagnosis or profile).

Future direction in the field of ASD categorization could move forward in the identification of subtypes within the autism spectrum: “subtype specifier” and “partial remission specifier” could promote progress in autism research and improve clinical practice.

DSM-5 allows to use specifiers of language and intellectual impairment that resulted enough restricted to distinguish clinical profile of HFA from classical, low functioning, autism. Nevertheless, a “subtype specifier” for AS could be useful to hypothesize putative developmental trajectories and direct specific paths of treatment.

Moreover, recent theories of neurodiversity, accompanied by the preference of the more neutral term of autism spectrum condition (ASC) rather than ASD, consider AS as a natural variation rather than a syndrome. In fact, AS subjects could, in particular conditions (“Aspie-friendly” or in preschool age), temporarily lacked significant global impairment. Their phenotypes may be considered subclinical-AS-forms. Nevertheless, in most cases, these subjects need a sort of support (cognitive-behavioral treatment, parent/teacher psychoeducation, personalization of school program), also for preventive purpose only. As in ADHD coding of DSM-5, regarding level 1 ASD patients, it could be suggested the use of “subclinical” specifier, for those patients who express only subthreshold autistic-traits and “partial remission” specifier, for those patients who do not meet currently the full diagnostic criteria.

In conclusion, even continuing to use the broad category of ASD, it could be meaningful to introduce, in future revisions of the manual, subtype specifiers that could capture the “nuances” of the spectrum.

Future research about the efficacy of these prospective specifiers could be beneficial.

See Also

- Academic Skills
- Atypical Autism
- Depressive Disorder
- DSM-5 and Autism Spectrum Disorder
- High-Functioning Autism (HFA)
- Neurodiversity

References and Reading

Asperger, H. (1944). Die autistische Psychopathen im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136.

- Attwood, T. (2006). *The complete guide to Asperger's syndrome*. Philadelphia: Jessica Kingsley Publishers.
- Bi, X., Wang, Y., Shu, Q., Sun, Q., & Xu, Q. (2018). Classification of autism spectrum disorder using random support vector machine cluster. *Frontiers in Genetics*, 9, 18.
- de Giambattista, C., Ventura, P., Trerotoli, P., Margari, M., Palumbi, R., & Margari, L. (2019). Subtyping the autism spectrum disorder: Comparison of children with high functioning autism and Asperger syndrome. *Journal of Autism and Developmental Disorders*, 49(1), 138–150.
- Ghaziuddin, M. (2008). Defining the behavioural phenotype of Asperger syndrome. *Journal of Autism and Developmental Disorders*, 38, 138–142.
- Ghaziuddin, M., & Welch, K. (2013). The Michigan autism spectrum questionnaire: A rating scale for high functioning autism spectrum disorders. *Autism Research and Treatment*, 2013, 708273.
- Gillberg, I., & Gillberg, C. (1989). Asperger syndrome: Some epidemiological considerations—a research note. *Journal of Child Psychology and Psychiatry*, 30, 631–638.
- Grove, J., et al. (2019). Identification of common genetic risk variants for autism spectrum disorder. *Nature Genetics*, 51(3), 431–444.
- Huerta, M., Bishop, S. L., Duncan, A., Hus, V., & Lord, C. (2012). Application of DSM-5 criteria for autism spectrum disorder to three samples of children with DSM-IV diagnoses of pervasive developmental disorders. *The American Journal of Psychiatry*, 169(10), 1056–1064.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Lai, M. C., Lombardo, M. V., Chakrabarti, B., & Baron-Cohen, S. (2013). Subgrouping the autism “spectrum”: Reflection on DSM-5. *PLoS Biology*. <https://doi.org/10.1371/journal.pbio.1001544>.
- Luckhardt, C., Jarczok, T. A., & Bender, S. (2014). Elucidating the neurophysiological underpinnings of autism spectrum disorder: New developments. *Journal of Neural Transmission*, 121(9), 1129–1144.
- Sharma, S., Woolfson, L. M., & Hunter, S. C. (2012). Confusion and inconsistency in diagnosis of Asperger syndrome: A review of studies from 1981 to 2010. *Autism*, 16(5), 465–486.
- Smith, I. C., Reichow, B., & Volkmar, F. R. (2015). The effects of DSM-5 criteria on numbers of individuals diagnosed with autism spectrum disorder: A systematic review. *Journal of Autism and Developmental Disorders*, 45(8), 2541–2552.
- Szatmari, P., Bartolucci, G., & Bremner, R. (1989). Asperger's syndrome and autism: Comparison of early history and outcome. *Developmental Medicine and Child Neurology*, 31, 709–720.
- Taylor, M. J., Charman, T., & Ronald, A. (2015). Where are the strongest associations between autistic traits and traits of ADHD? Evidence from a community-based twin study. *European Child and Adolescent Psychiatry*, 24(9), 1129–1138.

- Tsai, L. Y., & Ghaziuddin, M. (2014). DSM-5 ASD moves forward into the past. *Journal of Autism and Developmental Disorders*, 44(2), 321–330.
- Volkmar, F. R., & McPartland, J. C. (2014). From Kanner to DSM-5: Autism as an evolving diagnostic concept. *Annual Review of Clinical Psychology*, 10, 193–212.
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11, 115–129.
- Woodbury-Smith, M. R., & Volkmar, F. R. (2009). Asperger syndrome. *European Child and Adolescent Psychiatry*, 18(1), 2–11.

COMPASS for Hope Training Program

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Definition

COMPASS for Hope (C-HOPE) is a manualized parent-mediated intervention for children with challenging behavior and autism spectrum disorder (ASD). C-HOPE is based on an existing clinical decision-making framework known as the Collaborative Model for Promoting Competence and Success (COMPASS; Ruble et al. 2012). C-HOPE has effectiveness for telehealth or in-person delivery for decreasing behavior problems in children with autism spectrum disorder (ASD), increasing parent competency, and decreasing parent stress.

Historical Background

C-HOPE comes from COMPASS, a validated intervention that has been tested in randomized controlled trials (RCTs) and 20 years of work (Ruble and Dalrymple 1996) in public schools. COMPASS stands for Collaborative Model for Promoting Competence and Success and is an alternative framework for conceptualizing, assessing, and intervening to improve outcomes in autism based on a transactional approach (Fiese

and Sameroff 1989; Ruble and Dalrymple 1996) that considers the complex interplay between the individual, family, school, social, community, and economic resources – the critical proximal (i.e., closely connected to the individual) and distal influences and interactions between individuals with ASD and their environments. We first tested COMPASS for preschool and elementary age students as a consulting intervention to improve IEP outcomes with a large effect size (1.1) compared to IEP outcomes of students receiving services as usual (Ruble et al. 2010). In a second RCT, we replicated our findings and also demonstrated the efficacy of web-based coaching in COMPASS (Ruble et al. 2013). The third RCT, COMPASS for Transition (COMPASS-T), was tested with high school students in their final year of school with a focus on improving IEP and postsecondary outcomes (Ruble et al. 2018). A very large effect size (2.1) was observed for IEP goal attainment outcomes based on an independent observer.

Rationale or Underlying Theory

ASD is a neurodevelopmental disorder that presents many challenges for parents as well as teachers, therapists, and other professionals, often in part because of challenging problem behavior (Crone and Mehta 2016). These problem behaviors are secondary to, or exacerbated by, the core symptoms of ASD [Centers for Disease Control and Prevention (CDC) 2012]. The presence of problem behaviors impacts both children and their families. For children, they experience fewer community outings, less positive interaction with peers, and less access to intervention and education (Anderson et al. 1992; Matson and Nebel-Schwalm 2007). For parents, they report a greater sense of helplessness compared to parents of neurotypical children (Pisula and Kossakowska 2010). A meta-analysis conducted by Hayes and Watson (2013) suggests that the overall level of problem behaviors accounts for the most variance in explaining parent stress when compared to other child factors, such as IQ, autism severity, and adaptive skills, a finding confirmed by others (Krakovich et al. 2016).

Thus, we developed C-HOPE from the existing COMPASS framework to target three outcomes: decreased parent stress, decreased child problem behavior, and increased parent competency. COMPASS is unique in that it focuses on ASD-specific goal areas of social communication skills meaningful for quality of life (QOL). COMPASS is based on the developmental theory that competency is the result of reciprocal and dynamic interactions between individuals and their environments (i.e., transactional) (Ruble and Dalrymple 1996). Thus, if we can carefully examine and identify the contribution that the environment makes toward reducing individual risk factors and enhancing protective factors, we can influence the development of important QOL skills. Competence looks different across the lifespan and is person-specific. Thus C-HOPE behavior plans are sensitive to the specialized learning needs of individuals with ASD and the required instruction across social, communication, and learning skills competencies to continue to be refined and utilized throughout development into adulthood. Further, the extreme heterogeneity in ASD requires a framework, like COMPASS, that can be helpful independent of the language, cognitive, or social abilities of individuals with ASD.

Another unique dimension of C-HOPE, that separates it from other parenting programs, is the emphasis placed on a collaborative, strengths-based approach in working with families. C-HOPE is delivered in an individual and a group format. The group format is intended to be therapeutic as well as educational and draws upon the concept of universality (“I am not the only one struggling”; Yalom 1995) and offers the opportunity for families to also provide emotional support to others and to feel less isolation. C-HOPE requires that the facilitator(s) draw upon basic counselling skills that promote active listening and empathy. Facilitator(s) also have to be aware of group process for the purpose of promoting cohesion among members and to ensure that the goals and needs of the group are met. The importance of a strong therapeutic alliance is foundational to a good helping relationship (Crits-Christoph et al. 2009; Zuroff et al. 2010).

Goals and Objectives

C-HOPE is designed to promote positive parent *and* child outcomes, including decreased child problem behavior, decreased parent stress, and increased parent competency using an evidence-based practices in psychology (EBPP) framework such as COMPASS (McGrew et al. 2016). Parents are the primary intervention targets in C-HOPE. COMPASS emphasizes clinical decision-making based on the three elements of an EBPP-informed approach of child preferences and strengths, parent and family resources, and evidence-based practices. Through positive parent-therapist collaboration, utilizing the COMPASS framework, direct training and support are provided in order to facilitate parent-implemented behavior intervention plans.

Treatment Participants

C-HOPE was developed for parents of young children with ASD and/or intellectual disability or other comorbid diagnoses. C-HOPE is most suited for parents of children between the ages of 3 and 12 years who also experienced challenging behavior. C-HOPE was developed for in-person or telehealth delivery. Thus, parents who are unable to attend in-person sessions may benefit, and this can be a tremendous benefit for families in areas where local resources are scarce.

Treatment Procedures

The C-HOPE curriculum includes activities that support parent-to-parent interaction as well as parent knowledge and skill and include four individual sessions (1 h) and four group sessions (2 h). Prior to the start of the treatment sessions, parents complete a COMPASS profile (see Ruble et al. 2012). The profile guides the discussion for the first individual session and assists with clarifying the problem behavior and possible underlying communicative intents behind the behavior. Once the purposes of behavior are understood, goals are developed that target replacement skills

to increase positive behaviors and decrease problem behaviors.

Individual sessions primarily focus on developing, implementing, and fine-tuning the individualized behavior plan that describes the identified problem behavior(s) and replacement skills. After developing a common understanding and language (e.g., joint attention, antecedents, consequences) to describe child skills, parenting practices such as encouraging positive child behaviors and how to use supports proactively to decrease challenging behaviors are written into the behavior plan. Emphasis is placed on using positive behavior supports (i.e., environmental manipulations) that are designed to promote pro-social skills effective in reducing disruptive behaviors for children with ASD (Buschbacher and Fox 2003; Iovannone et al. 2003; National Research Council 2001; Turnbull et al. 2002). Behavior plans included understanding the antecedents or causes of behavior, making the behavior ineffective, teaching replacement skills that result in desired outcomes for the child, and rewarding positive skills.

The group sessions provide basic information on ASD, including discussion of the learning differences specific to ASD, as well as how these learning differences impact behavior, socialization, and communication of their child. Typically individual sessions follow group sessions so that information learned in group session is discussed and considered during the individual session so that behavior plans are fine-tuned as new information is learned. Psychoeducation also includes information on evidence-based approaches for problem behaviors such as functional behavior assessment including antecedent manipulation, changes in instructional context, differential reinforcement, and self-management strategies (Braithwaite and Richdale 2000; Buggley 2005; Frea et al. 2001; Keeling et al. 2003; Schilling and Schwartz 2004).

In addition, group sessions target parent stress and coping skills. A variety of coping strategies for parental stress are presented, and parents are asked to identify what strategies they find helpful and what new strategies they would consider using in the future. Coping strategies include

general stress reduction techniques, mindfulness-based interventions, and relaxation strategies that have been shown to have long-term positive effects on stress levels and psychological well-being of parents of children with ASD (Cachia et al. 2016).

Order of sessions and content of each individual and group session can be retrieved from Kuravackel et al. (2018). The C-HOPE manual outlining all session details can be requested from the authors.

Efficacy Information

C-HOPE has been tested in two studies. The first was a randomized wait list design that tested telehealth vs face-to-face delivery of C-HOPE. The second was a pre-posttest design that tested an online-only condition where parents met virtually rather than in person (Kuravackel et al. 2018; Rodgers 2018). Both studies examined outcomes of C-HOPE on variables of child problem behavior, parent competency, parent stress, group alliance, and parent satisfaction. Results from the first study indicated significant between-group differences between the treatment and control. Further, pre-posttreatment gains in child problem behavior scores, parent competency, and parent stress were observed for the C-HOPE group. Telehealth modality was equally effective as face-to-face intervention, and no differences were detected with regard to group alliance or parent satisfaction in either modality. Overall parent satisfaction was high across both telehealth and face-to-face modalities (Kuravackel et al. 2018). For the second study when C-HOPE was tested with the group session meeting virtually and the individual sessions conducted using telephone, significant improvements were noted in parent stress and child behaviors and no changes for parent competency (Rodgers 2018).

Outcome Measurement

For primary outcomes of reduced parent stress, increased parent competency, and reduced child

problem behavior, the following were used, respectively: the Parental Stress Index-Fourth Edition Short Form (PSI-4-SF; Abidin 1995) to measure parent stress, the Eyberg Child Behavior Inventory (ECBI; Eyberg and Pincus 1999) to measure child problem behavior, and the Being a Parent Scale (BPS; Johnston and Mash 1989) to measure parent competency. For the secondary outcomes of parent satisfaction and group alliance, the following measures were used, respectively: Consultation Satisfaction Questionnaire (CSQ; Ruble et al. 2010) and the Group Session Rating Scale (GSRS; Duncan and Miller 2007).

Qualifications of Treatment Providers

C-HOPE therapists should have a background in a field that provides mental health intervention services (e.g., clinical/counseling/school psychology, rehabilitation psychology, social work). Therapists who participated in the initial trial of C-HOPE included those with the following backgrounds: doctoral students in school psychology and licensed psychologists. C-HOPE therapists should have prior experience working with parents and clients with ASD, implementing behavioral interventions, and familiarity with evidence-based interventions recommended for ASD. It is recommended that therapists new to C-HOPE receive supervision of their first C-HOPE intervention. A manual has been developed for further reading and intervention fidelity and is available from the authors.

See Also

- Behavior Analysis
- Developmentally Appropriate Practice (DAP)
- Irritability in Autism
- Parental Response to Diagnosis
- Positive Behavioral Interventions and Supports (PBIS)
- Progressive Applied Behavior Analysis

References and Reading

- Abidin, R. (1995). *Parenting Stress Index* (3rd ed.). Lutz: Psychological Assessment Resources.
- Anderson, D. J., Lakin, K. C., Hill, B. K., & Chen, T. (1992). Social integration of older persons with mental retardation in residential facilities. *American Journal on Mental Retardation*, 96(5), 488–501. Multiple imputation with Mplus. Retrieved from <http://www.statmodel.com/download/Imputations7.pdf>
- Braithwaite, K., & Richdale, A. (2000). Functional communication training to replace challenging behaviors across two behavioral outcomes. *Behavioral Interventions*, 15(1), 21–36. [https://doi.org/10.1002/\(SICI\)1099-078X\(200001/03\)15:1<21::AID-BIN45>3.0.CO;2-#](https://doi.org/10.1002/(SICI)1099-078X(200001/03)15:1<21::AID-BIN45>3.0.CO;2-#).
- Buggey, T. (2005). Video self-modeling applications with students with autism spectrum disorder in a small private school setting. *Focus on Autism and Other Developmental Disabilities*, 20(1), 52–63. <https://doi.org/10.1177/10883576050200010501>.
- Buschbacher, P. W., & Fox, L. (2003). Understanding and intervening with the challenging behavior of young children with autism spectrum disorder. *Language, Speech & Hearing Services in Schools*, 34(3), 217–227. <https://doi.org/10.1177/10883576050200010501>.
- Cachia, R. L., Anderson, A., & Moore, D. W. (2016). Mindfulness, stress and well-being in parents of children with autism spectrum disorder: A systematic review. *Journal of Child and Family Studies*, 25(1), 1–14. <https://doi.org/10.1007/s10826-015-0193-8>.
- Centers for Disease Control and Prevention. (2012). Autism spectrum disorders(s). Retrieved from <http://www.cdc.gov/ncbddd/autism/signs.html>
- Crits-Christoph, P., Gallop, R., Temes, C. M., Woody, G., Ball, S. A., Martino, S., & Carroll, K. M. (2009). The alliance in motivational enhancement therapy and counseling as usual for substance use problems. *Journal of Consulting and Clinical Psychology*, 77(6), 1125–1135.
- Crone, R. M., & Mehta, S. S. (2016). Parent Training on Generalized Use of Behavior Analytic Strategies for Decreasing the Problem Behavior of Children with Autism Spectrum Disorder: A Data-Based Case Study. *Education and Treatment of Children*, 39(1), 64–94. Retrieved from <https://search-ebscohost-com.echo.louisville.edu/login.aspx?direct=true&db=eric&AN=EJ1092632&site=ehost-live>.
- Duncan, B. L., & Miller, S. D. (2007). *The Group Session Rating Scale*. Jensen Beach: Author.
- Eyberg, S. M., & Pincus, D. (1999). *Eyberg child behavior inventory and Sutter-Eyberg student behavior inventory-revised: Professional manual*. Odessa: Psychological Assessment Resources.
- Fiese, B. H., & Sameroff, A. J. (1989). Family context in pediatric psychology: A transactional perspective. *Journal of Pediatric Psychology*, 14(2), 293–314.
- Frea, W., Arnold, C., & Vittimberga, G. (2001). A demonstration of the effects of augmentative

- communication on the extreme aggressive behavior of a child with autism within an integrated preschool setting. *Journal of Positive Behavior Interventions*, 3(4), 194–198.
- Hayes, S. A., & Watson, S. L. (2013). The impact of parenting stress: A meta-analysis of studies comparing the experience of parenting stress in parents of children with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43(3), 629–642. <https://doi.org/10.1007/s10803-012-1604-y>.
- Iovannone, R., Dunlap, G., Huber, H., & Kinkaid, D. (2003). Effective educational practices for students with autism spectrum disorders. *Focus on Autism and other Developmental Disabilities*, 18, 150–165.
- Johnston, C., & Mash, E. J. (1989). A measure of parenting satisfaction and efficacy. *Journal of Clinical Child Psychology*, 18(2), 167–175.
- Keeling, K., Smith Myles, B., Gagnon, E., & Simpson, R. (2003). Using the power card strategy to teach sportsmanship skills to a child with autism. *Focus on Autism and Other Developmental Disabilities*, 18(2), 103–109.
- Krakovich, T. M., McGrew, J. H., Yu, Y., & Ruble, L. A. (2016). Stress in parents of children with autism spectrum disorder: An exploration of demands and resources. *Journal of Autism and Developmental Disorders*, 46(6), 2042–2053. <https://doi.org/10.1007/s10803-016-2728-2>.
- Kuravackel, G. M., Ruble, L. A., Reese, R. J., Ables, A. P., Rodgers, A. D., & Toland, M. D. (2018). COMPASS for Hope: Evaluating the effectiveness of a parent training and support program for children with ASD. *Journal of Autism & Developmental Disorders*, 48(2), 404–416. <https://doi.org/10.1007/s10803-017-3333-8>.
- Matson, J. L., & Nebel-Schwalm, M. S. (2007). Comorbid psychopathology with autism spectrum disorder in children: An overview. *Research in Developmental Disabilities: A Multidisciplinary Journal*, 28(4), 341–352.
- McGrew, J. H., Ruble, L. A., & Smith, I. M. (2016). Autism spectrum disorder and evidence-based practice in psychology. *Clinical Psychology: Science and Practice*, 23(3), 239–255.
- National Research Council (NRC). (2001). *Educating students with autism*. Washington, DC: National Academy Press.
- Pisula, E., & Kossakowska, Z. (2010). Sense of coherence and coping with stress among mothers and fathers of children with autism. *Journal of Autism and Developmental Disorders*, 40(12), 1485–1494.
- Rodgers, A. D. (2018). *Examining an asynchronous group discussion board adaptation of a parent-mediated behavior intervention for children with autism spectrum disorders*. Retrieved from <https://search-ebscohost-com.echo.louisville.edu/login.aspx?direct=true&db=ddu&AN=DB9B3D37FB8BF79D&site=ehost-live>
- Ruble, L. A., & Dalrymple, N. J. (1996). An alternative view of outcome in autism. *Focus on Autism and Other Developmental Disabilities*, 11(1), 3–14.
- Ruble, L. A., Dalrymple, N. J., & McGrew, J. H. (2010). The effects of consultation on individualized education program outcomes for young children with autism: The collaborative model for promoting competence and success. *Journal of Early Intervention*, 32(4), 286–301.
- Ruble, L. A., Dalrymple, N. J., & McGrew, J. H. (2012). *Collaborative model for promoting competence and success for students with ASD*. New York: Springer Science + Business Media.
- Ruble, L. A., McGrew, J. H., Toland, M. D., Dalrymple, N. J., & Jung, L. A. (2013). A randomized controlled trial of COMPASS web-based and face-to-face teacher coaching in autism. *Journal of Consulting and Clinical Psychology*, 81(3), 566–572. <https://doi.org/10.1037/a0032003>.
- Ruble, L. A., McGrew, J. H., Toland, M., Dalrymple, N., Adams, M., & Snell-Rood, C. (2018). Randomized control trial of COMPASS for improving transition outcomes of students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(10), 3586–3595.
- Schilling, D., & Schwartz, I. (2004). Alternative seating for young children with autism spectrum disorder: Effects on classroom behavior. *Journal of Autism and Developmental Disorders*, 34(4), 423–432.
- Turnbull, H. R., III, Wilcox, B. L., & Stowe, M. J. (2002). A brief overview of special education law with focus on autism. *Journal of Autism & Developmental Disorders*, 32(5), 479.
- Yalom, I. D. (1995). *The Theory and Practice of Group Psychotherapy* (4th ed.). New York: Basic Books.
- Yalom, I. D., & Leszcz, M. (2005). *The theory and practice of group psychotherapy* (5th ed.). New York: Basic Books.
- Zuroff, D. C., Kelly, A. C., Leybman, M. J., Blatt, S. J., & Wampold, B. E. (2010). Between-therapist and within-therapist differences in the quality of the therapeutic relationship: Effects on maladjustment and self-critical perfectionism. *Journal of Clinical Psychology*, 66(7), 681–697.

Competitive Employment

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Definition

Competitive employment is work that is performed on either a full- or part-time basis in which individuals are compensated for their work.

The compensation paid must be at or above the set minimum wage, but not less than the wages paid to individuals who are not disabled and performing work that is the same or similar. The individual must be employed in an integrated setting in which the individual has the ability to interact with individuals who are not disabled ([NYSED.gov](http://www.nysed.gov), 2011).

Historical Background

Historically, competitive employment was not considered a likely outcome for individuals with autism spectrum disorders (Wehman et al. 2003). Typically, individuals with disabilities were placed in segregated or sheltered vocational settings in which they worked among other individuals with disabilities only to be supervised by non-disabled individuals (Lutfiyya et al. 1988). These sheltered settings were considered to be transitional with the hopes that most individuals placed in these environments would transition into competitive employment. However, Taylor in 2002 noted that only 3.5% of those placed in sheltered work environments move on to competitive employment in any single year.

In the opening of the Americans with Disabilities Act (ADA) in 1990, Congress notes that society has had a tendency to segregate individuals in important areas such as employment and therefore states that “no covered entity shall discriminate against a qualified individual on the basis of disability in regard to job application procedures, the hiring, advancement, or discharge of employees, employee compensation, job training, and other terms, conditions, and privileges of employment” ([ada.gov](http://www.ada.gov), retrieved 2011). The passage of the ADA was to bring about a greater integration of individual with disabilities in the workplace. According to Blanck (2008), discrimination of individuals with disabilities has been reduced and more opportunities have opened.

Current Knowledge

Available statistics on employment by individuals with disabilities vary. However, the National

Survey of Americans with Disabilities (2010) reports that 21% of individuals with disabilities aged 18–64 had been employed with 59% of the general population maintaining employment. Data specific to individuals with autism spectrum disorders (ASD) are harder to find, but the information that is available suggests that those with ASD are less likely to be employed compared to other disability groups. In 2009, the National Longitudinal Transition Study 2 had found the following as it relates to individuals with autism spectrum disorder and employment (www.nlts2.org):

- 32.5% of young adults with autism spectrum disorders currently worked for pay versus an average of 59% of all respondents.
- 47.7% of youth with autism spectrum disorders had worked for pay in the past 2 years versus an average of 78.4% of all participants.
- 29% of young adults with autism spectrum disorders were looking for work if currently unemployed compared to 47.7% of all participants.

The study does not specify if those surveyed are in a competitive employment situation or not. However, it is important to note that individuals that are in competitive employment work a greater number of hours per week and earn more per week than individuals in noncompetitive employment (Schaller and Yank 2005).

Even though there is great benefit to the individual to be working in a competitive employment situation, only about 15% of individuals were doing so (Wehman et al. 2003). The rest were in a number of day habilitation services that were noncompetitive, and oftentimes, these individuals were not integrated with the community.

Current research indicates that individuals with ASD can work in a variety of community-based businesses. However, majority of individuals on the spectrum currently remain unemployed (Dew and Alan 2007).

See Also

- [Sheltered employment](#)
- [Supported employment](#)

References and Reading

- Americans with disabilities act of 1990, as amended. (2008). Retrieved 27 May 2011 from <http://www.ada.gov/pubs/adastatute08.htm>
- Blanck, P. (2008). "The right to live in the world": Disability yesterday, today and tomorrow. *Texas Journal on Civil Liberties and Civil Rights*, 13, 367–401.
- Dew, D. W., & Alan, G. M. (2007). *Rehabilitation of individuals with autism spectrum disorders* (Institute on Rehabilitation Issues Monograph no. 32). Washington, DC: The George Washington University, Center for Rehabilitation Counseling Research and Education.
- Gottlieb, A., Myhill, W. N., & Blanck, P. (2011). Employment of people with disabilities. In J. H. Stone & M. Blouin (Eds.), *International encyclopedia of rehabilitation*. Retrieved 27 May 2011 <http://cirrie.buffalo.edu/encyclopedia/en/article/123/>.
- Gutfiyya, Z. M., Rogan, P., & Shoultz, B. (1988). *Supported employment: a conceptual overview*. Syracuse: Center on Human Policy, Syracuse University. Retrieved 27 May 2011 from <http://thechp.syr.edu/workovw.htm>.
- New York State Education Department, ACCES-VR. (2003). *Employment outcome policy* (010.00P: Employment Outcome Procedure). Albany. Retrieved 27 May 2011 from http://www.access.nysed.gov/vr/current_provider_information/vocational_rehabilitation/policies_procedures/0010_employment_outcome/policy.htm#Definitions
- Schaller, J., & Yank, N. K. (2005). Competitive employment for people with autism: correlates of successful closure in competitive and supported employment. *Rehabilitation Counseling Bulletin*, 49(1), 4, 13.
- Taylor, S. J. (2002, September 2). Disabled workers deserve real choices, real jobs. Retrieved 27 May 2011 from <http://www.accessiblesociety.org/topics/economics-employment/shelteredwksps.html>
- Wehman, P., Revell, W. G., & Brooke, V. (2003). Competitive employment: has it become the: "first choice" yet? *Journal of Disability Policy Studies*, 14(3), 163–173.

Complete Agenesis

- [Agenesis of Corpus Callosum](#)

Complete Exhaustion

- [Posttraumatic Stress Disorder](#)

Components of Advance Theory of Mind in Autism Spectrum Disorder

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Short Description or Definition

In the study of social cognition, the routes to understand others are subserved by separable, independent brain networks. Theory of mind (ToM) otherwise been referred to as "mentalizing" or "mind reading" constitutes one of the neuropsychological mechanisms, providing the understanding of someone else's thoughts or intentions. However, within this concept as a whole, have been identified distinct components of ToM accounting for the appreciation of the socio-affective and socio-cognitive information (Shamay-Tsoory et al. 2002; Hynes et al. 2006). Cognitive ToM reflects the understanding of someone's beliefs, while affective ToM requires the empathic appreciation of someone's emotional state.

Several mechanisms of atypical ToM are associated with autism spectrum disorders (ASD) (Cantio et al. 2016; Happé et al. 2017) converting its measurement into a clinically relevant feature that may explain and mitigate social difficulties in ASD population.

While subjects with autism present difficulties in standard ToM understanding, the majority of individuals with HFA/AS, whose intelligence ranges from normal to near-normal level and does not present language delay, are characterized by higher-order mind-reading abilities, often referred to as "advanced theory of mind."

Consequently, the conceptualization of the term "components of advanced theory of mind in autism spectrum disorder" refers to the assessment of subtle mentalizing difficulties within the autistic spectrum generated by distinct subdomains.

Historical Background

Autism Origins as a Distinct Diagnostic Entity

Only from 1980 and on, autism appeared for the first time as an independent classification in the DSM-3, published in 1980. Until that moment the concept of autism was encompassed in the core symptoms of schizophrenia. At this point, the perception of the condition changed from the description of a symptom to the description of a behavioral, developmental phenotype encompassing a range of symptoms in the social, communicative domain, as well as symptoms of rigidity and repetitiveness (Rapin 1997).

Clinical features, such as the presence of autistic traits in patients with schizophrenia (Kastner et al. 2015) and the development of schizophrenia in a significant proportion of children diagnosed with autism spectrum disorder (Larson et al. 2017), supported the connection between the two spectra at a clinical level in subsequent investigations, leading to numerous comparative studies (e.g., Ozguven et al. 2010; Lugnegard et al. 2013; Tin et al. 2017). However, beyond their similarities in certain features (Tin et al. 2017) to date, there is a clear-cut distinction in respect to neurocognitive profiles and separate diagnosis.

Currently, the umbrella term of ASD is used to describe all people diagnosed within the spectrum, including autism, pervasive developmental disorder not otherwise specified (PDD-NOS), and Asperger syndrome (AS) (APA 2013). Since the disturbances of social cognition are among the central characteristics of ASD, it has been considered as a disorder of the social brain, and it is nowadays categorized in the DSM-5/SCD as a communication disorder in the broader domain of neurodevelopmental disorders.

ASD and ToM Connection

As commonly described, the term theory of mind (ToM) refers to the ability of someone to understand another's thoughts, beliefs, and other internal states such as desires, emotions, or intentions (Frith and Frith 2006). The first application of the term in ASD research was in an experiment which used false belief paradigms to explore ToM in

children with autism (Baron-Cohen et al. 1985). To investigate this, they developed the Sally-Anne false belief task (Baron-Cohen et al. 1985; Wimmer and Perner 1983), in which Sally places an object in a box and leaves the scene. After this, Anne relocates the ball and Sally returns. Participants with well-developed ToM capacities succeeded in predicting Sally's behavior based on her false belief about the ball's location.

Since then a growing body of evidence on social cognition deficits has provided data consistent with the hypothesis that social difficulties in ASD are explained by a decreased ability to use mental state concepts to interpret and predict one's own and other people's behavior (Baron-Cohen et al. 2000; Tager-Flusberg 2007). This theoretical connection with ToM on social abilities remains highly correlated among recent literature. More specifically the two cognitive accounts of ASD that have received the most attention are ToM and executive function.

Likewise, the degree of ToM difficulties may vary widely depending on intelligence quotient (IQ) and language development defining particular ASD diagnosis, being more severe in autism than in Asperger syndrome (AS) or in high-functioning autism (HFA) (Baron-Cohen et al. 2000). Additionally, children diagnosed with ASD generally develop ToM later and continue to fail on these tests until late childhood and even adolescence (Happé 1995); therefore, age might be considered a positive correlate in healthy participants, but not in people diagnosed with ASD.

Current Knowledge

Advanced ToM

Traditionally, ToM is assessed throughout false belief understanding. These tasks relate to the understanding that different people can have different thoughts about the same situation. "First-order" false beliefs indicate what someone thinks about real events, while more advanced "second-order" false beliefs refer to what someone thinks about other people's thoughts. Thus, within the broad construct of ToM, advanced measures were created to examine adults' ability to reason about

mental states in more complex contexts including higher-order false belief understanding, emotion recognition, and social understanding (Livingston et al. 2018).

Impairments in advanced ToM tasks include the selection of subtle nonverbal social cues, interpretation of social stories, construction of social inferences, and comprehension of implied or non-literal meanings in the language (Baron-Cohen et al. 2001; Mathersul et al. 2013). Overall, conducting traditional or advanced ToM research in ASD individuals depends on the clinical expression in which impairments vary according to their age and the severity of their social and communicative difficulties.

In consideration of the diverse structure and performance, ToM may have overdevelopment, and measurements become more task-specific with age. While typically developed children between the ages of 3 and 4 years can solve verbally presented first-order false belief tasks (Wellman et al. 2001; Wimmer and Perner 1983), most children with ASD do not pass these tasks until the age of 11 years (Happé 1995). Then dealing with second-order false belief tasks can be solved by children around the age of 6 and 7 years, whereas advanced ToM abilities are required to process complex social situations in which the wrong behavior has to be represented by a cognitive and affective component, first appearing in typically developed children between the ages of 9 and 11 (Baron-Cohen et al. 1997). Preschoolers, middle childhood, and adults should, therefore, be examined with age-appropriate adaptations.

Distinct Theoretical Components of ToM

Since ToM origins as a concept of investigation, its study expanded to the identification of different aspects, grounded in a distinct neurobiological substrate, brain regions, and differing interacting functions. Understanding others' internal states may be a multidimensional process that interacts with other abilities, a process which may not occur in a single conceptual framework (Warnell and Redcay 2019). Tager-Flusberg and Sullivan (2000) had put forward "the componential view of theory of mind" which suggests the study of ToM as two differentiated processes: the social

cognitive, connected with what has traditionally been called theory of mind and is based on the conceptual understanding of the mind as a representational system, and then the social-perceptual or empathetic component, associated with the affective system and is based on the rapid "online" judgment of another individual's mental state from her/his facial expressions, gestures, tone of voice, movements, and actions.

Considerable research confirm over the years that affective and cognitive aspects of ToM are indeed somewhat separable involving overlapping but dissociable brain systems by highlighting the mediating role of ventromedial (VM) frontal lobes in ToM research (Shamay-Tsoory and Aharon-Peretz 2007; Hynes et al. 2006; Duval et al. 2011; Schaafsma et al. 2015). Depending on which ToM task is used and which aspect of ToM is focused on, research on ToM derived from autism has reported somewhat different results. The bulk of studies consistently support impairments in the general ToM performance of ASD individuals even in high-functioning adults with ASD (Cantio et al. 2016; Happé et al. 2017; Wellman et al. 2001; Ponnet et al. 2004). However, not all research argues for a ToM deficit as the key mechanism underlying the social interaction impairments seen in ASD (Stone and Gerrans 2006; Van de Cruys et al. 2014).

Recent evidence from behavioral data suggested that cognitive and affective ToM is differentially impaired in individuals with ASD. Some ASD literature supports findings for greater impairment in the socio-cognitive component of ToM (Rogers et al. 2007; Dziobek et al. 2008; Mazza et al. 2014) while others for the socio-affective ToM (Lockwood et al. 2013; Shamay-Tsoory et al. 2002). Understanding the way that cognitive and affective aspects of ToM relate to each other is complicated also due to the inconsistent and under-specified uses of relevant terminology. Some of these problems can be reconciled if ToM began to conceptualize as a multifactorial construct, comprised of multiple sub-processes.

Studying the effects of localized brain lesions constitute a set of trustworthy evidence. Comparative studies of the effects of localized brain

lesions suggest that there are some commonalities in the neural mechanisms of emotional processing and ToM. (Mitchell and Phillips 2015). In the past, decoding has often been conflated with emotional processing and reasoning with ToM. Nevertheless, this may not always be appropriate, since basic decoding mechanisms can specialize for either emotional information or intentional information, while other tasks require the perception and combination of both emotional and intentional information (e.g., when trying to decode sarcasm or deception; Adams Jr. and Kleck 2005). In this context, various theoretical models support that emotional perception always precedes ToM in the processing chain, while ToM cannot proceed without input from emotional perception (Baron-Cohen 2005; Mitchell 2006; Ochsner 2008). The fact that emotional process occurs at an earlier temporal stage compared to ToM (Corrigan 1997; Mitchell 2006) further supports the assumption that emotional processing and ToM are indeed related.

Along with the review conducted by Mitchell and Phillips (2015) collecting data from several lesion studies, it is suggested a possible overlap between emotional processing and ToM located in the medial PFC, probably in the right hemisphere. Moreover, neuroimaging data focus on studies that have compared the perception of affective cues against the inference of thoughts, intentions, and beliefs, indicating overlapping function between the substrates of emotional processing and ToM in dorsal PFC regions and at the temporal pole. In other researches have directly been compared the neuroanatomy of affective vs. cognitive ToM by manipulating experimental stimuli (e.g., “what are the characters thinking” vs. “what are they feeling”). Hynes et al. (2006) found a differential response in ventral prefrontal regions of the brain with affective ToM inducing greater activation of ventromedial PFC compared to non-affective ToM. A greater association between dorsolateral PFC activity and cognitive ToM was further supported by transcranial magnetic stimulation (TMS) data where suppression of right dorsolateral PFC activity has been shown to impair only cognitive ToM (Kalbe et al. 2010). In summary affective ToM (which bears some

resemblance to EP) and cognitive ToM partly share neural correlates but can also be differentiated. Therefore, overall research up to date theorizes that EP and ToM share some common components yet without excluding functional independence between the two processes in specific areas.

How Is Cognitive and Affective ToM Being Measured Up to Now?

In the last 15 years, several tests have been developed to tap higher-order mind-reading abilities, which are often referred to as “advanced theory of mind.” Numerous instruments have been proposed to assess ToM, or some aspect(s) of ToM, in individuals with ASD, although there is not yet a universally accepted operationalization of ToM. The more widely used instruments of this kind are “The Strange Stories Test” (Happé 1994), which contains written stories/scenarios with persuasion, white lies, double buffs, and deception; the “Faux Pas Test” (Stone et al. 1998), which involves the understanding of social situations where someone says something without considering the listener’s point of view, with negative consequences that the speaker never intended; and the “Reading the Mind in the Eyes Test” (RMET; Baron-Cohen et al. 2001), which involves recognizing a person’s mental state from the expression in their eyes.

A large number of studies continue to consider ToM a unitary construct, employing only one or two measures in order to capture ToM. Across a variety of specific tasks and modalities which have been argued to be important components of ToM (e.g., verbal versus nonverbal, deliberate versus automatic, implicit versus explicit), the distinction between affective and cognitive ToM captures the use of different cognitive abilities to solve these tasks. Both aspects of mentalizing considered critical predictors of well-being. Affective ToM is most often assessed by presenting either pictorial stimuli depicting complex affective states or verbal narratives that describe a protagonist’s emotion. The Reading the Mind in the Eyes Test reflects this former approach and is one of the most widely used and well-validated measures of this construct. Moreover, in the clinical assessment, it is also used

the Awareness of Social-Inference Test-Revised (TASIT; McDonald et al. 2006) a dynamic audio-visual-based assessment of social cognition. TASIT comprised of a series of 15–60 sec video vignettes and requires participants to implicitly encode and integrate contextual information in order to recognize emotions and determine the meaning and intention of speakers' dialogue, emotional expression, and other paralinguistic information. In respect to cognitive ToM, one of the best-validated methods of assessing this construct is via a comparison of true and false belief tasks. As mentioned before the Strange Stories and the Faux Pas Test tasks tap those domains and are well-validated measures that appear to have good sensitivity to social cognitive failures in clinical groups.

Recent research would ideally study these specific characteristics combining both cognitive and perceptual processes within the same test. In the Cambridge Mindreading (CAM) Face-Voice Battery (Golan et al. 2006) is required recognition of complex emotional mental states from dynamic faces, based on voice prosody and content of the spoken sentences. Recognition of complex emotional states is assumed to involve higher-level integration and mindreading but also low-level perceptual processes (Mitchell and Phillips 2015). Moreover, this test proves good discriminator validity between clinical (autism, Asperger syndrome) and nonclinical groups (Golan et al. 2006), and it is sensitive to developmental changes in ToM.

Novelties Measuring ToM

To date, a series of novel and previously overlooked approaches have been proposed in order to improve the measurement of ToM in research and clinical settings. Classic tests such as those described above "Strange Stories Test" and "Faux Pas Test" are considered "explicit" measures since ToM is prompted by direct mental state questions. Several findings suggest that individuals with ASD show relatively minor impairments on classical tasks (evaluated usually by explicit measurements) compared with their difficulties observed in clinical settings and everyday social situations (e.g., Lever and Geurts 2016;

Scheeren et al. 2013; Wilson et al. 2014). Clements and Perner (1994) were the first to empirically investigate a difference between implicit and explicit ToM; however, this distinction is currently debated (Carruthers 2017).

An implicit measure of social cognition is based on fast spontaneous, unconsciously, and inflexibly action, while explicit ToM reasoning is conquered around age 4 and allows for a deliberate consideration like the judgment of others' mental states. To this end, various studies (e.g., Apperly and Butterfill 2009; Sodian et al. 2014) support the notion of two ToM reasoning systems: an explicit system that verbally explains behavior based on mental states and an implicit system for spontaneous sensitivity to other's mental states. These differences in the representational repertoire of the two systems should thus manifest themselves in distinctive and differential patterns of performance.

Deschrijver et al. (2016) used implicit tasks to measure ToM suggesting that when ToM atypicalities are measured by response time on appropriate cognitive tasks, it may help to understand autistic symptoms in adulthood. Measuring and controlling reaction time (RT) have not been used systematically as a technique within classical ToM, while accuracy is considered as a ToM ability. Nevertheless, measuring accuracy alone can lead to misunderstandings when alternative cognitive strategies might compensate for ToM performance (Scheeren et al. 2013; Livingston et al. 2018). Furthermore, when participants are administered, such tests are given as much time as necessary to process the material, which may explain why individuals may pass the probe test yet still struggle in everyday situations. Thus, combining reaction time and accuracy data could further improve the efficacy of advanced ToM measurements.

Evidence from current studies measuring also implicit mentalizing such as spontaneous looking patterns that reflect intuitive tracking of another person's belief state, documenting clear difficulties in adults with ASD. Besides, recent neuroimaging studies (Kovács et al. 2014; Schneider et al. 2014) showed that tasks developed to measure implicit ToM engage brain areas that are core to

the explicit processing of other's beliefs, suggesting that social cognitive processes most likely underlie implicit ToM. Other recently developed tasks have sought to measure ToM by examining accuracy, via open-ended or multiple-choice questions, of mental state attribution after watching video clips of social situations (Brewer et al. 2017; Dziobek et al. 2008; Murray et al. 2017). The advantage of these tasks resides in the prerequisite of more than simple inferences from images or stories and consequently reflecting more realistic social situations.

Past measures of social cognition tended to focus exclusively on comprehension of a social situation (Mathersul et al. 2013), while recent research has begun to ask participants to generate possible social responses to typical interactions (e.g., "What would you do in this situation?"; Jameel et al. 2014) and found that autistic traits are associated with fewer pro-social responses (Murray et al. 2017).

Future Directions

A critical examination of limitations into existing ToM findings as well as the lack of convergence among conventional ToM measures will offer insight into cognition and behavior more broadly. Inconsistencies in the literature may account for various methodological differences across studies; however, recent work challenges to fill these gaps by understanding and controlling such limitations and further designing improved evaluation tools and theoretical contexts.

Briefly, among the most essential shortcomings, future research needs to disentangle is the variability in the tasks used to measure ToM. The administration of many instruments has not been standardized, and thereby reliable normative data are currently nonexistent. Another dearth emerges from past studies that did not adequately distinguish performance on tasks measuring distinct theoretical components of ToM. Future research needs to clarify theoretically as well as experimentally the extent to which the tasks make emotional and cognitive demands, which components

of ToM are most impacted in ASD, and whether distinct components of ToM predict symptom severity and social functioning in ASD.

Discrepancy also occurs between laboratory and naturalistic settings. Laboratory-based ToM tasks vary in their sensitivity to real-life task demand. For instance, Ponnet et al. (2008) and Chevallier et al. (2015) claim for real-life social contexts when measuring ToM as a parameter to capture the dynamics of social interactions. Deficits linked with ASD in interpreting emotional expressions are observed to be more pronounced when the context was naturalistic and unstructured rather than highly organized or predictable. Finally, the majority of ToM measures present information verbally, while fewer present information visually. Thereupon in many cases, ToM ability is underestimated due to verbal impairments, whereas individuals with good verbal skills pass ToM tasks that are mainly explicit in form presumably due to compensatory strategies.

See Also

- [Autism: Social Communication Disorder](#)
- [Autism Theory](#)

References and Reading

- Adams, R. B., Jr., & Kleck, R. E. (2005). Effects of direct and averted gaze on the perception of facially communicated emotion. *Emotion*, 5, 3–11.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric.
- Apperly, I., & Butterfill, S. A. (2009). Do humans have two systems to track beliefs and belief-like states? *Psychological Review*, 116(4), 953–970.
- Baron-Cohen, S. (2005). The empathizing system: A revision of the 1994 model of the mindreading system. In B. Ellis & D. Bjorklund (Eds.), *Origins of the social mind*. New York: Guilford Press.
- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. J. (2000). *Understanding other minds: Perspectives from developmental cognitive neuroscience* (2nd ed.). New York: Oxford University Press.
- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a theory of mind? *Cognition*, 21, 37–46.

- Baron-Cohen, S., Jolliffe, T., Mortimore, C., & Roberts, M. (1997). Another advanced test of theory of mind: Evidence from very high functioning adults with autism or Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 38, 813–822.
- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. J. (2000). *Understanding other minds: Perspectives from developmental cognitive neuroscience* (2nd ed.). New York: Oxford University Press.
- Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The “Reading the Mind in the Eyes” test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology & Psychiatry*, 42, 241–251.
- Brewer, N., Young, R.L., Barnett, E. (2017). Measuring Theory of Mind in Adults with Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders*, 47, 1927–194.
- Cantio, C., Jepsen, J. R., Madsen, G. F., Bilenberg, N., & White, S. J. (2016). Exploring ‘the autisms’ at a cognitive level. *Autism Research*, 9(12), 1328–1339.
- Carruthers, P. (2017). Mindreading in adults: Evaluating two-systems views. *Synthese*, 194, 673–688.
- Chevallier, C., Parish-Morris, J., McVey, A., Rump, K. M., Sasson, N. J., Herrington, J. D., & Schultz, R. T. (2015). Measuring social attention and motivation in autism spectrum disorder using eyetracking: Stimulus type matters. *Autism Research*, 8, 620–628.
- Clements, W. A., & Perner, J. (1994). Implicit understanding of belief. *Cognitive Development*, 9, 377–395.
- Corrigan, P. W. (1997). The social perceptual deficits of schizophrenia. *Psychiatry*, 60, 309–326.
- Deschrijver, E., Bardi, L., Wiersema, J. R., & Brass, M. (2016). Behavioural measures of implicit theory of mind in adults with high functioning autism. *Cognitive Neuroscience*, 7(1–4), 192–202.
- Duval, C., Piolino, P., Bejanin, A., Eustache, F., & Desgranges, B. (2011). Age effects on different components of theory of mind. *Consciousness and Cognition*, 20, 627–642.
- Dziobek, I., Rogers, K., Fleck, S., Bahnemann, M., Heekeren, H. R., Wolf, O. T., et al. (2008). Dissociation of cognitive and emotional empathy in adults with Asperger syndrome using the multifaceted empathy test (MET). *Journal of Autism and Developmental Disorders*, 38, 464–473.
- Frith, C. D., & Frith, U. (2006). How we predict what other people are going to do. *Brain Research*, 1079, 36–46.
- Golan, O., Baron-Cohen, S., & Hill, J. (2006). The Cambridge mindreading (CAM) face-voice battery: Testing complex emotion recognition in adults with and without Asperger syndrome. *Journal of Autism and Developmental Disorders*, 36, 169–183.
- Happé, F. (1994). An advanced test of theory of mind: Understanding of story characters thoughts and feelings by able autistic, mentally handicapped and normal children and adults. *Journal of Autism and Developmental Disorders*, 24, 129–154.
- Happé, F. (1995). The role of age and verbal ability in the theory of mind task: Performance of subjects with autism. *Child Development*, 66, 843–855.
- Happé, F., Cook, J. L., & Bird, G. (2017). The structure of social cognition: In(ter) dependence of sociocognitive processes. *Annual Review of Psychology*, 68, 243–267.
- Hynes, C. A., Baird, A. A., & Grafton, S. T. (2006). Differential role of the orbital frontal lobe in emotional versus cognitive perspective-taking. *Neuropsychologia*, 44, 374–483.
- Jameel, L., Vyas, K., Bellesi, G., Roberts, V., & Channon, S. (2014). Going ‘Above and Beyond’: Are those high in autistic traits less pro-social? *Journal of Autism and Developmental Disorders*, 44, 1846–1858.
- Kalbe, E., Schlegel, M., Sack, A. T., Nowak, D. A., Dafotakis, M., Bangard, C., Brand, M., Shamay-Tsoory, S., Onur, O. A., & Kessler, J. (2010). Dissociating cognitive from affective theory of mind: A TMS study. *Cortex*, 46, 769–780.
- Kastner, A., Begemann, M., Michel, T. M., Everts, S., Stepniak, B., Bach, C., et al. (2015). Autism beyond diagnostic categories: Characterization of autistic phenotypes in schizophrenia. *BMC Psychiatry*, 15, 115.
- Kovács, A. M., Kühn, S., Gergely, G., Csibra, G., & Brass, M. (2014). Are all beliefs equal? Implicit belief attributions recruiting core brain regions of theory of mind. *PLoS One*, 9(9), e106558.
- Larson, F. V., Wagner, A. P., Jones, P. B., Tantam, D., Lai, M. C., Baron-Cohen, S., et al. (2017). Psychosis in autism: Comparison of the features of both conditions in a dually affected cohort. *British Journal of Psychiatry*, 210(4), 269–275.
- Lever, A. G., & Geurts, H. M. (2016). Age-related differences in cognition across the adult lifespan in autism spectrum disorder. *Autism Research*, 9(6), 666–676.
- Livingston, L. A., Bethany, C., & Punit, S. (2018). Recent advances and new directions in measuring theory of mind in autistic adults. *Journal of Autism and Developmental Disorders*, 49, 1738–1744.
- Lockwood, P. L., Bird, G., Bridge, M., & Viding, E. (2013). Dissecting empathy: High levels of psychopathic and autistic traits are characterized by difficulties in different social information processing domains. *Frontiers in Human Neuroscience*, 7, 760.
- Lugnegard, T., Hallerböck, U. M., Hjäorthag, F., & Gillberg, C. (2013). Social cognition impairments in Asperger syndrome and schizophrenia. *Schizophrenia Research*, 143(2–3), 277–284.
- Mathersul, D., McDonald, S., & Rushby, J. A. (2013). Understanding advanced theory of mind and empathy in high-functioning adults with autism spectrum disorder. *Journal of Clinical and Experimental Neuropsychology*, 35(6), 655–668.
- Mazza, M., Pino, M. C., Mariano, M., Tempesta, D., Ferrara, M., De Berardis, D., et al. (2014). Affective and cognitive empathy in adolescents with autism spectrum disorder. *Frontiers in Human Neuroscience*, 8, 791.

- McDonald, S., Bornhofen, C., Shum, D., Long, E., Saunders, C., & Neulinger, K. (2006). Reliability and validity of the awareness of social inference test (TASIT): A clinical test of social perception. *Disability and Rehabilitation*, 28(24), 1529–1542.
- Mitchell, J. P. (2006). Mentalizing and Marr: An information processing approach to the study of social cognition. *Brain Research*, 1079, 66–75.
- Mitchell, R. L. C., & Phillips, L. H. (2015). The overlapping relationship between emotion perception and theory of mind. *Neuropsychologia*, 70, 1–10.
- Murray, K., Johnston, K., Cunnane, H., Kerr, C., Spain, D., Gillan, N., et al. (2017). A new test of advanced theory of mind: The “Strange Stories Film Task” captures social processing differences in adults with autism spectrum disorders. *Autism Research*, 10(6), 1120–1132.
- Ochsner, K. N. (2008). The social-emotional processing stream: Five core constructs and their translational potential for schizophrenia and beyond. *Biological Psychiatry*, 64, 48–61.
- Ozguven, D. H., Oner, O., Baskak, B., Oktem, F., Olmez, S., & Munir, K. (2010). Theory of mind in schizophrenia and Asperger’s syndrome: Relationship with negative symptoms. US National Library of Medicine. *National Institutes of Health*, 20(1), 5–13.
- Ponnet, K. S., Roeyers, H., Buysse, A., De Clercq, A., & Van Der Heyden, E. (2004). Advanced mind-reading in adults with Asperger syndrome. *Autism*, 8, 249–266.
- Ponnet, K., Buysse, A., Roeyers, H., & Clercq, A. (2008). Mindreading in young adults with ASD: Does structure matter? *Journal of Autism and Developmental Disorders*, 38, 905–918.
- Rapin, I. (1997). Autism. *New England Journal of Medicine*, 337(2), 97–104.
- Rogers, K., Dziobek, I., Hassenstab, J., Wolf, O. T., & Convit, A. (2007). Who cares? Revisiting empathy in Asperger syndrome. *Journal of Autism and Developmental Disorders*, 37, 709–715.
- Schaafsma, S. M., Pfaff, D. W., Spunt, R. P., & Adolphs, R. (2015). Deconstructing and reconstructing theory of mind. *Trends in Cognitive Sciences*, 19(2), 65–72.
- Scheeren, A. M., de Rosnay, M., Koot, H. M., & Begeer, S. (2013). Rethinking theory of mind in high-functioning autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 54(6), 628–635.
- Schneider, D., Slaughter, V. P., & Dux, P. E. (2014). What do we know about implicit false-belief tracking? *Psychonomic Bulletin & Review*, 22(1), 1–12.
- Shamay-Tsoory, S. G., & Aharon-Peretz, J. (2007). Dissociable prefrontal networks for cognitive and affective theory of mind: A lesion study. *Neuropsychologia*, 45(13), 3054–3067.
- Shamay-Tsoory, S. G., Tomer, R., Yaniv, S., & Aharon-Peretz, J. (2002). Empathy deficits in Asperger syndrome: A cognitive profile. *Neurocase*, 8, 245–252.
- Sodian, B., Schuwerk, T., & Kristen, S. (2014). *Implicit and spontaneous theory of mind reasoning in autism spectrum disorders*. <https://doi.org/10.5772/59393>.
- Stone, V. E., & Gerrans, P. (2006). What’s domain-specific about theory of mind? *Social Neuroscience*, 1, 309–319.
- Stone, V. E., Baron-Cohen, S., & Knight, R. T. (1998). Frontal lobe contributions to theory of mind. *Journal of Cognitive Neuroscience*, 10, 640–656.
- Tager-Flusberg, H. (2007). Evaluating the theory-of-mind hypothesis of autism. *Current Directions in Psychological Science*, 16(6), 311–315.
- Tager-Flusberg, H., & Sullivan, K. (2000). A componential view of theory of mind: Evidence from Williams syndrome. *Cognition*, 76, 59–90.
- Tin, L. N. W., Lui, S. S. Y., Ho, K. K. Y., Hung, K. S. Y., Wang, Y., Yeung, H. K. H., et al. (2017). High-functioning autism patients share similar but more severe impairments in verbal theory of mind than schizophrenia patients. *Psychological Medicine*, 48(8), 1264–1273.
- Van de Cruys, S., Evers, K., Van der Hallen, R., Van Eylen, L., Boets, B., de-Wit, L., & Wagemans, J. (2014). Precise minds in uncertain worlds: Predictive coding in autism. *Psychological Review*, 121, 649–675.
- Warnell, K. R., & Redcay, E. (2019). Minimal coherence among varied theory of mind measures in childhood and adulthood. *Cognition*, 191, 103997.
- Wellman, H. M., Cross, D., & Watson, J. (2001). Meta-analysis of theory-of-mind development: The truth about false belief. *Child Development*, 72(3), 655–684.
- Wilson, E., Happé, F., Wheelwright, S. J., Ecker, C., Lombardo, M. V., Johnston, P., et al. (2014). The neuropsychology of male adults with high functioning autism or asperger syndrome. *Autism Research*, 7(5), 568–581.
- Wimmer, H., & Perner, J. (1983). Beliefs about beliefs: Representation and constraining function of wrong beliefs in young children’s understanding of deception. *Cognition*, 13(1), 103–128.

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Comprehension

- Listening Comprehension
- Receptive Language

Comprehensive Assessment of Spoken Language

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Synonyms

CASL

Description

The CASL is an oral language assessment battery for individuals aged 3–21 years and is based on a comprehensive theory of language. Consisting of 15 tests, the CASL measures auditory comprehension, oral expression, and word retrieval processes in four linguistic categories: Lexical/Semantic, Syntactic, Supralinguistic, and Pragmatic. The CASL battery provides an in-depth evaluation of oral language processing systems, knowledge and use of grammatical structures, ability to use language requiring higher-level processing, and adaptation of language across contexts. The results of the CASL assessment provide value in measuring linguistic skill among children with language delays and disorders as well as the competence of individuals who are learning English as a second language.

Each of the 15 CASL tests is constructed differently and measures different aspects of oral language. First, the Lexical/Semantic tests assess knowledge and use of words and word combinations through five tests. *Comprehension of Basic Concepts* measures the ability of young children to comprehend words representing basic perceptual and conceptual relations. *Antonyms* assesses expression of a word that has the opposite meaning of a given word. *Synonyms* assesses the ability to identify a word with the same meaning as a given word. *Sentence Completion* measures the ability to retrieve and express one of the few appropriate words that fit the meaning of spoken

sentence. *Idiomatic Language* measures the expressive knowledge of a group of words that, when used together, have a conventional meaning different from the literal meaning of the individual words.

Next, there are five Syntactic tests that assess knowledge and use of grammar (morphology and syntax). *Syntax Construction* assesses the ability to generate sentences using a variety of morphosyntactic rules. *Paragraph Comprehension* measures the comprehension of syntax through a series of narratives spoken by the examiner. *Grammatical Morphemes* measures the metalinguistic knowledge of the form and meaning of the English language. *Sentence Comprehension* measures the ability to comprehend meaning of the syntactic-structure organization of sentences. *Grammaticality Judgment* assesses the ability to recognize and correct grammatical errors within sentences.

Then, four Supralinguistic tests measure comprehension of complex language in which the meaning is not directly available from lexical or grammatical information. *Nonliteral Language* measures the ability to comprehend figurative speech, indirect requests, and sarcasm. *Meaning from Context* measures inference ability that does not require the examinee to use world knowledge. *Inference* assesses the ability to integrate appropriate world knowledge that is not explicitly stated with information provided by the speaker. *Ambiguous Sentences* assesses the ability to comprehend multiple meanings given sentences containing elements that can be interpreted in more than one way. Finally, *Pragmatic Judgment* measures awareness of the appropriateness of language and ability to modify language in relation to the situation in which it is used.

The CASL is individually administered and requires no reading or writing on part of the examinee. Each test can stand alone, and each score can be reported as a standard score (mean of 100, standard deviation of 15, range of 40–160), percentile, normal curve equivalent (NCE), stanine, and test-age equivalent. These scores allow each test to be compared with every other test, both within a category and between

categories, as well as between tests of comprehension, expression, and retrieval. Additionally, a global measure of oral language can be calculated from a subset of representative tests to derive a CASL Core Composite. Examiners may also administer all of the tests appropriate for the child's age to use as a comprehensive battery. Depending on the students' age, Index Scores may be computed as representative measures of language categories (Lexical, Syntactic, Supralinguistic; available for ages 7–21) and processing (Expressive, Receptive; available for ages 7–10). Administration time varies based on the examinee's age and the number of tests administered. When administering the Core Battery tests only, it takes approximately 30 min to test children aged 3–5 years and 45 min to 1 h for older examinees.

The CASL has two record forms: Record Form 1 is appropriate for children 3–6 years, and Record Form 2 is for those 7–21 years. Three self-standing test easels contain all of the picture stimuli and item text, as well as administration and scoring procedures for each test. General testing information is provided in the easels as well, such as basal and ceiling criteria, allowance of item repetition if the examinee appears to not understand the task, and prompting guidelines for specific tests. Test easels are tabbed for each individual test, and starting pages are marked for the appropriate age ranges. The manual also provides procedural information, interpretation of the results specific to each of the 15 CASL tests, and technical properties such as the description of test development, standardization, reliability, and validity data. A separate Norms Book contains all the normative tables for the CASL in order to convert raw scores to standard scores and provides significance values for comparing differences between tests scores.

Historical Background

Originally published in 1999, the CASL was developed by author Dr. Elizabeth Carrow-Woolfolk as a comprehensive test of oral language based on her Integrative Language Theory, ILT (Carrow-Woolfolk 1988, 1994; Carrow-Woolfolk

and Lynch 1981). ILT combines and relates the linguistic, cognitive, and pragmatic aspects of language, which forms the basis for the classification of the CASL tests. In this view, a comprehensive understanding of language must take into account both the *meaning* of language and its *form*.

The content of language conveys meaning and is largely dependent on substantive words and word combinations. Words that refer to a relatively specific category of things are considered lexical morphemes (vocabulary). Throughout the CASL, the term “semantics” is used to describe the content of only the lexical morphemes of language. Conversely, language can be more abstract with a general meaning that refers to a wide range of things (pronouns, prepositions). These are considered grammatical morphemes (functors and inflections) which along with syntax (word order and structure of language) represent the form or structure of the language.

Language can be ambiguous when word constructions have multiple interpretations. Often an individual must use inference or contextual information to interpret an underlying nonliteral meaning of language. This type of nonliteral language is labeled supralinguistic, which implies that the interpretation of an utterance extends beyond the literal meaning and requires higher-level analysis to comprehend. This category of language emerges later in development and is an integral component in measuring linguistic skill during adolescence. Additionally, the relation of form and meaning is significantly influenced by context including social variables (setting, age, relationship), linguistic variables (type of discourse-conversation, a narrative, a lecture), and personal variables (intention, motivation, knowledge, and style of the sender). These contextual aspects of communication are considered pragmatic and can affect not only the manner in which language is expressed but also how it is interpreted.

The categorization of CASL tests follows the structure of the ILT. Lexical/Semantic tests address the meaning of language (Basic Concepts, Antonyms, Synonyms, Sentence Completion, Idioms). The Syntactic tests address meaning

derived from the structure of language (Syntax Construction, Paragraph Comprehension, Grammatical Morphemes, Sentence Comprehension, Grammaticality Judgment). The Supralinguistic tests measure understanding and use of complex meanings that require analysis above that of lexical and syntactic meaning (Nonliteral Language, Meaning from Context, Inference, Ambiguous Sentences). The Pragmatic test measures understanding of language that is dependent on context and social understanding (Pragmatic Judgment).

The theoretical distinction of these categories is reflected in the developmental literature as well. Children begin to use language at a lexical level and proceed to using two-word phrases and simple morphemes, such as prepositions. Gradually, utterances lengthen by the addition of complex morphemes and the use of structurally advanced syntax, such as noun and verb agreement. Eventually children can adapt their language to the needs and demands of the environment. Only then do children begin to comprehend and express nonliteral language and idioms. Comprehension and expression of complex language in which the utterance units rely on one another for meaning evolves gradually, once the use of basic elements has matured.

The CASL procedures and content reflect the two distinct dimensions of language: knowledge (structure, form, and content of language) and performance (internal systems used to comprehend and express language). As a result, the CASL separates listening comprehension (Receptive Index) from oral expression (Expressive Index). Although these two major processes of language have common elements (e.g., they both draw upon the same basic knowledge of language structure and meaning), there is empirical evidence for their distinction (Carrow-Woolfolk 1985).

Psychometric Data

In developing the CASL, Dr. Woolfolk created items that would elicit information not only about an individual's language knowledge and competency but the individual's use of language and functionality of communication as well.

Initial items were piloted in 1992, and normative data were collected between 1996 and 1997. The CASL was normed on a nationwide standardization sample of 1,700 individuals, 3–21 years, which matched the most current US Census data (1994 Current Population Survey) on gender, race/ethnicity, region, and mother's educational level. Additionally, CASL sampling procedures drew children from public and private schools without respect to special education status. As a result, children receiving various special education services were represented in the normative sample in approximately the same proportions that occur in the US school population. The following five major special education categories were included: Specific Learning Disability (3.4% of CASL sample), Speech or Language Impairments (1.5%), Mental Retardation (0.6%), Emotional Disturbance (0.3%), and Other Impairments (0.4%).

The CASL tests, Index Scores, and Core Composites have high internal consistency and test-retest reliabilities. Item analyses, including Rasch scaling methods, were utilized to obtain the final test items. For each age band (12 groups), all of the Core Composite reliabilities are in the .90s, and the indexes range from .85 to .96. Test-retest reliability was assessed by administering the CASL twice (median 6 week interval) to 148 randomly selected examinees in three age groups: 5–10 to 6–11 (41 cases), 8–10 to 10–11 (38 cases), and 14–16 to 16–17 (69 cases). Results suggest only a minor practice effect and provide strong evidence of the stability of CASL scores.

Five criterion-related validity studies and eight clinical validity studies were conducted during standardization, with over 600 participants. In the first group of studies, CASL scores were compared with four measures of oral language (Test for Auditory Comprehension of Language-Revised [TACL-R]; Listening Comprehension and Oral Expression Scales of the Oral and Written Language Scales [OWLS]; Peabody Picture Vocabulary Test, Third Edition [PPVT-III]; Expressive Vocabulary Test [EVT]) and a measure of cognitive ability (Kaufman Brief Intelligence Test [K-BIT]). The highest overall correlations are with the OWLS, which assesses

the same four linguistic categories as the CASL. While the OWLS assessment has a wide-range approach, the CASL allows for an in-depth study of specific skills. For the clinical validity studies, performance of eight different clinical groups was examined: Speech Impairment, Language Delay, Language Impairment, Mental Retardation, Learning Disability (Reading), Learning Disability (Undifferentiated), Emotional Disturbance, and Hearing Impairment. Each clinical case was matched with a case from the standardization sample on the following variables: age, gender, race/ethnicity, and SES as measured by the mother's education level. Children in the matched control groups were not receiving any special services. The manual includes tables reporting the mean and standard deviation of each clinical group and the matched control groups as well as the significance value of the difference between groups.

In general, the clinical group scored about the same as the control group on most of the CASL tests for the Speech Impairment group. In contrast, the Language Delay, Language Impairment, Emotional Disturbance, and both Learning Disability groups scored significantly lower than their matched control groups. Adolescents with mild mental retardation scored approximately two standard deviations below the mean on all CASL tests. For Hearing Impairment, the clinical group scored lower on all CASL tests (e.g., about one standard deviation below the mean); however, interpretation is limited due to a small sample size for this group.

Clinical Uses

The CASL can assist speech/language pathologists, psychologists, educational diagnosticians, early childhood specialists, and other professionals in measuring oral language processing skills and knowledge in preschoolers through young adults. The age-based norms are useful in identifying language impairments to meet the requirements of Public Law 94-142 (now incorporated into the Individuals with Disabilities Education Act [IDEA] reauthorized as Public

Law 105-17). Further, CASL standard scores can be directly compared with many other tests when using the available standard scores based on the common metric scale (mean of 100, standard deviation of 15).

The CASL assists clinicians in understanding the relation between an individual's score and any delays or disorders in language. The presence of poor performance in one or more of the language categories may be indicative of a language disorder. Further, a significant discrepancy in performance among the categories may indicate a problem. For example, comparisons can be made between receptive and expressive tasks or between tasks highly dependent on retrieval and those that are not. In-depth qualitative information from the CASL may also be used to identify the possibility of a disorder even though the quantitative level of performance is not atypical. Clinicians are also able to create a profile of an examinee's oral language strengths and weaknesses across distinct categories of language. The CASL can also provide a record of growth in language skills and knowledge across a broad time span (3–21 years) through using the same instrument.

Communication deficits are often associated with autism spectrum disorders (ASD). The CASL can provide clinicians with in-depth information about an individual's knowledge and use of language, which may help to identify problem areas associated with ASD. For example, the CASL can be used to identify deficits in expressive compared to receptive language as well as problems in the *use* of language (e.g., pragmatics) as opposed to the *content* or *form* of language (lexical/semantic and syntactic). This may be particularly useful for high-functioning ASD individuals who often have advanced lexical/semantic knowledge and syntactic skills, but relatively poor pragmatic skills associated with difficulties in social interaction (Paul and Wilson 2009). Recent evidence suggests that the Pragmatic and Supralinguistic areas of the CASL are useful particularly in documenting the difficulties that individuals with ASD have in communicating flexibly across contexts (Reichow et al. 2008).

References and Reading

- Carrow-Woolfolk, E. (1985). *Test for Auditory Comprehension of Language-Revised (TACL-R)*. Allen: DLM Teaching Resources.
- Carrow-Woolfolk, E. (1988). *Theory, assessment, and intervention in language disorders: An integrative approach*. Philadelphia: Grune & Stratton.
- Carrow-Woolfolk, E. (1994). *Learning to read: An oral language perspective of beginning reading*. San Antonio: The Psychological Corporation.
- Carrow-Woolfolk, E. (1999/2008). *Comprehensive assessment of spoken language*. Los Angeles: Western Psychological Services.
- Carrow-Woolfolk, E., & Lynch, J. I. (1981). *An integrative approach to language disorders in children*. San Antonio: The Psychological Corporation.
- Paul, R., & Wilson, K. P. (2009). Assessing speech, language, and communication in autism spectrum disorders, Ch. 7. In S. Goldstein, J. A. Naglieri, & S. Ozonoff (Eds.), *Assessment of autism spectrum disorders* (pp. 171–208). New York: Guilford Press.
- Reichow, B., Salamack, S., Paul, R., Volkmar, F. R., & Klin, A. (2008). Pragmatic assessment in autism spectrum disorders: A comparison of a standard measure with parent report. *Communication Disorders Quarterly*, 29, 169–176.

Comprehensive Transition Program

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Definition

A Comprehensive Transition and Postsecondary Program is a college-based program for students with an intellectual disability defined and created by the enacting of Public Law 110–135: the Higher Education Opportunities Act (HEOA, 2008). As defined in the law, a Comprehensive Transition and Postsecondary Program is “designed to support students with intellectual disabilities who are seeking to continue academic, career and technical, and independent living instruction at an institution of higher education in order to prepare for gainful employment” (HEOA, sec. 760). Such a program must be

offered by an institution of higher education and have an advising and curriculum structure. The curriculum must provide for the students with an intellectual disability to spend at least one-half of their time on academic components with non-disabled individuals.

A Comprehensive Transition and Postsecondary Program is a program based at a college or technical school to assist students with an intellectual disability, transitioning out of secondary education, who are not yet ready to enter the workforce or to enroll full-time in a college or technical school. The programs are designed to provide the necessary instruction and support in social functioning and independent living skills to enable the student to prepare for gainful employment.

Institutions of higher education must apply to the United States Department of Education in order to have their program approved as a Comprehensive Transition and Postsecondary Program. In order to be eligible to apply for approval, the institution of higher education must already be approved to administer Federal Financial Aid for its students. A student enrolled in an approved Comprehensive Transition and Postsecondary Program is eligible for Federal financial assistance under the Federal Pell Grant, Federal Supplemental Education Opportunity Grants (FSEOG), and Federal Work Study (FWS) programs. As opposed to the general requirements for a postsecondary student to be eligible for federal financial aid, a student with an intellectual disability in an approved transition program does not have to be enrolled for the purpose of obtaining a degree or certificate and the student is not required to have a high school diploma, a recognized equivalent of a high school diploma, or have passed an ability to benefit test. However, the student must be making satisfactory progress according to the institution’s published standards for students enrolled in its comprehensive transition and postsecondary programs (34 CFR, § 668.233).

See Also

- [Individualized Transition Plan \(ITP\)](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)

- [Intellectual Disability](#)
- [Transition Planning](#)
- [Transitional Living](#)

References and Reading

Higher Education Opportunities Act, Pub. L. No. 110–135 § 760, 34 CFR subpart O § 668.230–233.

Compulsiveness

- [Perfectionism](#)

Computed Axial Tomography (CAT)

- [Computed Tomography](#)

Computed Tomography

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Synonyms

[Computed axial tomography \(CAT\)](#); [CT](#); [X-ray computed tomography](#)

Definition

Computed tomography is a medical imaging method employing tomography created by computer processing of X-ray images. A form of digital geometry processing is used to generate a three-dimensional image of the inside of an object from a large series of two-dimensional X-ray images taken around an axis of rotation. This

volume of data can then be manipulated via computer algorithm to observe and measure bodily structures (including brain structures) based on their ability to block the X-ray beam. This was one of the first techniques available to neuroscientists to study the structure of the living human brain. As such, it was one of the first techniques to be used to study brain structure in individuals with autism providing some of the earliest insights into the neural systems affected by autism.

See Also

- [Magnetic Resonance Imaging](#)

References and Reading

- Damasio, H., Maurer, R. G., Damasio, A. R., & Chui, H. C. (1980). Computerized tomographic scan findings in patients with autistic behavior. *Archives of Neurology*, 37(8), 504–510.
- Harcherik, D. F., Cohen, D. J., Ort, S., Paul, R., Shaywitz, B. A., Volkmar, F. R., et al. (1985). Computed tomographic brain scanning in four neuropsychiatric disorders of childhood. *American Journal of Psychiatry*, 142(6), 731–734.

Computer-Assisted Instruction to Teach Academic Skills

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Definition

Computer-assisted instruction (CAI) has been defined as a systematic approach to developing students' knowledge and/or skills that uses a

computer as a central feature to support instruction via activities including, but not limited to, presenting materials, assessing progress, and guiding activities (Anohina 2005). CAI is a subset of technology-aided instruction, and is distinct from online, Internet, or other network learning in that CAI implies the software is local to the computer, not accessed via the Internet.

Historical Background

Technology has transformed everyday life for many people in the twenty-first century. From its infancy in the form of teaching machines (Pressey 1926; Skinner 1958) to the wide forms and platforms that are ever evolving (Stephenson and Limbrick 2015), instructors and researchers have evaluated the usefulness of technology as the central feature of an intervention to increase academic skills. The current application of CAI originates from the work of psychologists in the early twentieth century.

Sidney Pressey was the first to experiment with teaching machines. He defined the teaching machine as an automatic or self-controlling device that presented a unit of information, provided some means for the learner to respond to the information, and provided feedback about the correctness of the learner's responses (Pressey 1926). Approximately 30 years later, B. F. Skinner (1958) published descriptions of his own teaching machines, which were capable of requiring constructed responses. Skinner attributed the lack of professional enthusiasm and adoption of Pressey's machines to cultural inertia, in that there was not a need for instruction to be automated when there was an abundance of teachers. However, there was an increased interest in teaching machines in the 1950s and 1960s when the baby boom led to a shortage of teachers. The promise of teaching machines was that they provided a solution to the problem of insufficient teacher attention to address all students varying needs. It was presumed that the teaching machine could replace some functions the teacher was currently providing. There were, however, criticisms of teaching machines in the late 1960s, including being

unnecessary for presentation of programmed materials, being too expensive, and individuals' doubts about the extent to which the teaching machines could teach (Benjamin 1988).

The teaching machines developed by Skinner and others, such as the *AutoTutor* (Crowder 1962), were based on the principles of programmed instruction (Molenda 2008). Programmed instruction was described by Bijou et al. (1966) as the process of systematically and effectively arranging and rearranging an environment to elicit a change in a targeted behavior. The intent of programmed instruction is to make the teaching-learning process effective and customized to meet individual needs (Molenda 2008). Skinner's early work in programmed instruction focused on content that was arranged in small chunks of information that built upon each other. In a forward-chaining procedure, the student did not progress forward to a new "chunk" of information until the previous had been mastered. In this form of programmed instruction, learners progressed at an individual pace. The use of reinforcement in the form of knowledge of a correct response increased the likelihood of future correct responding. Programmed instruction was incorporated into textbooks as well, relieving the necessity for hardware. Research using teaching machines to deliver programmed instruction led Skinner to refer to the use the term *technology of teaching* (Skinner 1968).

The evidence-base of CAI to teach academics to students with autism spectrum disorders (ASD) goes back over three decades. Panyan (1984) was one of the first to make the argument for the incorporation of computers into the education of students with disabilities in her review of literature on the use of computers with individuals with ASD. She concluded her review by proposing several distinct benefits computers may offer. These included consistency of instruction, the potential to facilitate generalization, and the inclusion of preferred sensory stimuli into instruction.

As technology has rapidly changed in format and capabilities since Panyan's initial review, the field of special education has followed with empirical evaluations on how this may enhance the lives of individuals with ASD. Because

educators of students with ASD are responsible for providing access to and ensuring progress in the general curriculum (Browder et al. 2007), a specific focus has been on how to effectively use technology to teach academics to students with ASD. Empirical investigations and periodic reviews of research are critical to fulfilling this responsibility because they identify which uses of technology are actually effective, and when educators use practices that are supported by scientific evidence, student outcomes improve (Cook et al. 2008, 2012).

Current Knowledge

In the first literature review on the use of CAI to teach academics to students with ASD, Pennington (2010) found 15 articles that (a) were published in a peer-reviewed journal between the years of 1998 and 2008, (b) were based on experimental research, (c) manipulated an independent variable involving the use of CAI, and (d) collected data related to an academic skill. All of the studies that met inclusion criteria addressed literacy skills. Pennington found a total of 15 articles that met inclusion criteria, and concluded that CAI was effective for a limited number of skills, namely literacy skills, but that many of the designs lacked experimental control.

Knight et al. (2013) had similar findings with 25 out of 25 included studies teaching literacy skills. Inclusion criteria for Knight et al. (2013) consisted of the following: (a) published in a peer reviewed journal between 1973 and 2012, (b) teaching an academic skill, and (c) inclusion of technology as the intervention or a part of the intervention package, including video modeling. Knight et al. (2013) found a total of 25 articles that met these criteria, the majority of which targeted literacy skills. However, the quality was limited as most of the studies met only a few quality indicators (QIs) for single-subject or group design as outlined by Horner et al. (2005).

An updated review by Root et al. (2017) included 29 studies that (a) were published in a peer reviewed journal between 1995 and May 2015, (b) included at least one participant with

ASD, (c) used a recognized experimental or quasi-experimental design, (d) had a dependent variable that measured an academic skill, and (e) CAI served as a key component of the independent variable, excluding video modeling. Authors coded each study for QIs using guidelines of the National Technical Assistance Center on Transition (NTACT 2016). These guidelines provided operational definitions for the QIs outlined for single-case research by Horner et al. (2005) and group experimental research by Gersten et al. (2005). After coding each study for QIs, authors evaluated the evidence to determine if CAI to teach academics to students with ASD was an EBP. Of the 29 studies that met inclusion criteria, 24 used single-case methods. Of those 24, two met criteria for high quality studies and an additional eight met criteria for acceptable quality, therefore satisfying criteria for classification as an EBP. Of the five group experimental studies, two met criteria for high quality, further meeting the NTACT requirements for classification as an EBP.

After determining that CAI was an EBP for teaching academics to students with ASD, Root et al. (2017) further analyzed high and adequate quality studies included in their review for instructional variables to provide a more descriptive picture of the evidence base in terms of content, context, and instructional materials and methods. Nine out of 12 high and acceptable quality studies targeted literacy skills, meaning the evidence for teaching nonliteracy skills (e.g., math, science, and social studies) is not high. The context of CAI was primarily the special education classroom, with other settings included a clinic, conference room, computer lab, and a participant's home. A total of 94 participants with ASD were included in high or adequate quality studies, the majority of whom were male (78%). Unfortunately, only five studies reported ethnicity, making inferences into the generalization of findings according to ethnicity impossible.

The instructional materials used in the high and adequate quality studies included desktops, tablets, and laptops. Interventions were delivered through software or applications in the majority of the studies. Every study used behavioral

instructional methods, aligning with the origin of CAI in Skinner's teaching machines and what is known about the effectiveness of systematic instruction to teach students with ASD.

With the establishment of CAI to teach academics to students with ASD as an EBP, practitioners should consider ways CAI can be incorporated into instruction to overcome barriers to skill acquisition or give students access to academic content. This implication is especially relevant for literacy, as the majority of the evidence to support CAI targets literacy skills. In addition to efficacy, incorporating CAI into instruction can have several other benefits. For instance, the electronic format of many CAI interventions easily facilitates creation, presentation, and transfer of learning materials more feasible for practitioners. This may enable sharing among educators and families, allowing for continuity of interventions across classrooms, school years, and between home and school. In addition, CAI allows for modification to learning materials based on individual student needs (Knight et al. 2013). For example, although one PowerPoint or SMARTboard lesson may be created to address a common learning objective, it can be individualized to address students' interest topics, prerequisite skills, or to adjust for low- or high-ability learners. CAI is not intended to replace teacher instruction; rather, it may supplement or enhance teacher-directed instruction. It may increase the efficiency of instruction by reducing the amount of intensity of teaching monitoring required (Knight et al. 2013). This would allow for teachers to target multiple students' objectives within one lesson.

Future Directions

CAI practices are constantly evolving, and as a result educators may find it challenging to keep pace. Classrooms no longer maintain previous staple items such as chalkboards and overhead projectors, which have given way to items such as SMARTboards and portable tablets. This new technology and the practices associated with them reflect an expansion of the definition of

technology interventions for students with ASD (Wong et al. 2015). Additionally, assistive technology tools, devices, software, and services are increasingly interwoven into the fabric of all settings (e.g., general education, resource, separate), changing the dynamics of today's classrooms. As such, additional research is needed to keep pace with the rapid advances in technology.

First, research into content areas beyond literacy remains a need for students with ASD, as nonliteracy areas can have meaningful impact on students' postsecondary success (Wei et al. 2017). Students with disabilities, including students with ASD, are increasingly being included within the general education classroom for all subject areas. As a result, it is important for future experimental studies to target core content academic skills such as inquiry-based science, and mathematical problem-solving. Current technology can be used to modify content for accessibility, to align content to state standards, and to address the individual needs of students with ASD.

Second, the majority of the research has taken place within separate settings. More and more students with ASD are being educated within less restrictive settings, and CAI can serve as the catalyst to provide access to general education for students with ASD. Therefore, it is important for the context of research to expand beyond the special education classroom and investigate the effects of CAI to teach academics to students with ASD within general education settings as well. Future research should include treatment packages that look to academics for students with ASD through the use of combining CAI and behavior analytic approaches to instruction.

Third, the results of the analysis of instructional materials and methods of high and adequate quality studies may be of interest in future research using CAI. Instructional materials are no longer limited to consumable worksheets; they now include devices, software, assistive technology tools and services to deliver CAI to students with ASD. Looking to future trends in CAI, some of the technology listed should be considered for future research:

1. **Websites:** Although there are many websites available for teachers to use in academic instruction, there is limited experimental support (Bouck et al. 2014). As a result, expanded research using materials available on the Internet is warranted.
2. **Mobile devices** (cell phones): What was once “smart” is now “cognitive.” Smart devices such as meters, lighting, or machines evolve with artificial intelligence enhancements that provide more sensory, context-aware, and complex capabilities. Cognitive technology is modeled on the human brain and has potential to have self-learning abilities. As our mobile devices evolve, research must keep pace with the effects of these devices on student academic progress and how they can best be used within educational environments.
3. **Projection apps:** First introduced for mobile devices, projection apps now work specifically with portable tablets to increase engagement and collaboration. The projection app will allow teachers and students to wirelessly display content from portable tablets on attached projectors. Using the app’s Moderator function, educators can create a collaborative classroom by displaying up to four different portable devices simultaneously, with a maximum of 50 devices connected. Using the app, students and educators also can connect to mobile devices anywhere with wireless connection, making it useful for a one-to-one classroom context. Research is warranted on how to do this effectively.
4. **Virtual reality (VR):** VR is beginning to be used in business communications, retail, entertainment, sports, health, and other industries. Makers of VR have claimed that VR has the potential to completely change how we work, shop, play, and consume media. Future research should include using VR to teach academics to students with and without ASD.
5. **Voice control to sentient:** Voice control software has moved from simply voice-to-text to now include such skills as Internet searches, making phone calls, turning lights on/off, and interfacing with devices that are embedded into our environment (e.g., mobile devices).

Sentient tools are aware of their context, environment, and social interactions. As technologies such as cloud-based artificial intelligence, collaborative robots, and ever-more sophisticated machine-learning algorithms begin to converge, we will start to see the emergence of these capabilities in smart cars, homes, manufacturing, and education. Thus, research is needed for how to use this technology to improve education for students with ASD.

6. **Artificial intelligence personal assistants, chatbots, and conversational interfaces:** Advances in machine learning and natural language processing have already made conversational mediators possible. As they grow in sophistication, they look to move from the fringe and become a primary interface for Internet of Things devices and networks. Research is warranted on how the use of AI can enhance or impede educational and functional skills for students with and without ASD.

These are only a few examples of the multitude of ways CAI instructional materials for students with ASD have made their way into classrooms. Looking to future trends, it is important for educators to continue to investigate all the various assistive technology tools, devices, and services to determine their effectiveness, efficiency, and evidence-base. Finally, future experimental research should adhere to established design QIs. For future studies to be able to contribute to the evidence base, they need to attend to QIs such as those described by Gersten et al. (2005) and Horner et al. (2005).

References and Reading

- Anohina, A. (2005). Analysis of the terminology used in the field of virtual learning. *Journal of Educational Technology & Society*, 8(3), 91–102.
- Benjamin, L. T. (1988). A history of teaching machines. *American Psychologist*, 43, 703–712.
- Bijou, S. W., Birnbrauer, J. S., Kidder, J. D., & Tague, C. (1966). Programmed instruction as an approach to teaching of reading, writing, and arithmetic to retarded children. *The Psychological Record*, 16, 505–524.

- Bouck, E. C., Satsangi, R., Doughty, T. T., & Courtney, W. T. (2014). Virtual and concrete manipulatives: A comparison of approaches for solving mathematics problems for students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44, 180–193.
- Browder, D. M., Wakeman, S. Y., Flowers, C., Rickelman, R. J., Pugalee, D., & Karvonen, M. (2007). Creating access to the general curriculum with links to grade-level content for students with significant cognitive disabilities: An explication of the concept. *The Journal of Special Education*, 41, 2–16.
- Cook, B. G., Tankersley, M., & Harjusola-Webb, S. (2008). Evidence-based special education and professional wisdom: Putting it all together. *Intervention in School and Clinic*, 44, 105–111. <https://doi.org/10.1177/1053451208321566>.
- Cook, B. G., Smith, G. J., & Tankersley, M. (2012). Evidence-based practices in education. In K. R. Harris, S. Graham, & T. Urdan (Eds.), *APA Educational Psychology Handbook* (Vol. 1, pp. 495–528). Washington, DC: American Psychological Association.
- Crowder, N. A. (1962). Intrinsic and extrinsic programming. In J. E. Coulson (Ed.), *Programmed learning and computer-based instruction* (pp. 58–66). New York: Wiley & Sons.
- Gersten, R., Fuchs, L. S., Compton, D., Coyne, M., Greenwood, C., & Innocenti, M. S. (2005). Quality indicators for group experimental and quasi-experimental research in special education. *Exceptional Children*, 71, 149–164.
- Horner, R. H., Carr, E. G., Halle, J., McGee, G., Odom, S., & Wolery, M. (2005). The use of single-subject research to identify evidence-based practice in special education. *Exceptional Children*, 71, 165–179.
- Knight, V., McKissick, B., & Saunders, A. (2013). A review of technology-based interventions to teach academic skills to students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43, 2628–2648.
- Molenda, M. (2008). The programmed instruction era: When effectiveness mattered. *Techtrends: Linking Research and Practice to Improve Learning*, 52(2), 52–58.
- National Technical Assistance Center on Transition. (2016). *Criteria for levels of evidence*. Retrieved from: http://transitionta.org/system/files/effective_practices/EP_Criteria_2017.pdf.
- Panyan, M. V. (1984). Computer technology for autistic students. *Journal of Autism and Developmental Disorders*, 14, 375–382.
- Pennington, R. C. (2010). Computer-assisted instruction for teaching academic skills to students with autism spectrum disorders: A review of literature. *Focus on Autism and Other Developmental Disabilities*, 25, 239–248.
- Pressey, S. L. (1926). A simple apparatus which gives tests and scores-and teaches. *School and Society*, 23, 373–376.
- Root, J. R., Stevenson, B. S., Ley Davis, L., Geddes-Hall, J., & Test, D. W. (2017). Establishing computer-assisted instruction to teach academics to students with autism as an evidence-based practice. *Journal of Autism and Developmental Disorders*, 47, 275–284.
- Skinner, B. F. (1958). Teaching machines. *Science*, 128(3330), 969–977.
- Skinner, B. F. (1968). *The technology of teaching*. New York: Appleton-Century-Crofts.
- Stephenson, J., & Limbrick, L. (2015). A review of the use of touch-screen mobile devices by people with developmental disabilities. *Journal of Autism and Developmental Disorders*, 45, 3777–3791.
- Wei, X., Yu, J. W., Shattuck, P., & Blackorby, J. (2017). High school math and science preparation and post-secondary STEM participation for students with an autism spectrum disorder. *Focus on Autism and Other Developmental Disabilities*, 32, 83–92.
- Wong, C., Odom, S. L., Hume, K. A., Cox, A. W., Fettig, A., Kucharczyk, S., et al. (2015). Evidence-based practices for children, youth, and young adults with autism spectrum disorder: A comprehensive review. *Journal of Autism and Developmental Disorders*, 45, 1951–1966.

Computer-Based Intervention Assistive Technology

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Definition

Computer-based interventions are those that use technology in some form to provide an interactive, multisensory learning experience. Also called computer-assisted instruction (CAI), this form of intervention is used to present information, allow a user to practice certain skills repeatedly, or to test knowledge or comprehension.

Historical Background

Computers have been used with individuals with autism since the 1970s. Colby (1973) used a computer and keyboard to increase the spontaneous verbalizations of children with autism. It was the

intent of the researcher to allow the children to engage in free play with the computer system that was designed to say the names of the letters the children typed or provide some kind of visual reinforcer when a letter was typed. Thirteen out of the 17 participants in the study showed increased language after the therapy. Throughout the rest of the 1970s and early 1980s, most of the evidence that was published regarding computer-based technology was anecdotal and not systematic. These interventions focused on learning a computer language (Goldenberg 1979), playing a computer game (Frost 1981), responsiveness (Geoffrion and Goldenberg 1981), attention (Pleinis and Romanczyk 1983), and social skills (Panyan et al. 1984).

By the mid 1990s, approximately 45 commercially available software programs were marketed for children and young adults with autism. These programs were created by a small number of developers to target cause and effect and language and cognitive development. Higgins and Boone (1996) described best practices for the creation of software for individuals with autism. The authors noted that the symptoms of autism manifest differently in each individual, so they encouraged educators and others working with students with autism to create their own computer-based interventions to implement specific strategies that are known to work with the students. Eighteen software guidelines were provided for those who wished to create software for children with autism. The guidelines cover many aspects of software design such as age appropriateness and principles of repeated practice. Additionally, Higgins and Boone (1996) provided tips and strategies related to creating software for children with autism.

Recently, the field of computer-based interventions has seen a steady growth in the quantity and creativity applied to this population. Virtual reality technology has been utilized to address social difficulties and pragmatic impairments typically seen in individuals with autism (Self et al. 2007), an electronic tutor has been developed to aid in vocabulary development (Bosseler and Massaro 2003), and robots have been created to target social interactions and joint attention (Robins

et al. 2005; Robins et al. 2004). Additionally, with the fervor over the Apple iPod touch, iPad, and approximately 350,000 apps that are available, much attention and speculation has been focused on the use of this new technology. GeekSLP.com is a blog designed for speech-language pathologists focused on technology use. There is also a GeekSLP app available for the iPad which helps identify apps that are appropriate for use by speech-language pathologists in the clinical setting.

Rationale or Underlying Theory

What human beings lack in patience, predictability, and consistency computers make up for. Microcomputers were introduced in the interventions of children with autism due to the finding that children with autism spent a large amount of time playing with machines compared to playing with human beings (Colby 1973). Computer-based interventions have the ability to control where and how stimuli are presented to help children with autism compensate for attentional difficulties and problems with filtering tangential or unnecessary information. Difficulty with unpredictability is a common symptom of autism. Computer-based instruction can vary a routine or program in very small increments, helping an individual with autism to accept these changes and build tolerance for change. See Panyan (1984) for a discussion on the early rationale for computer-based instruction for children with autism. More recently, Blischak and Schlosser (2003) have argued that computer-based interventions can contribute in unique ways to the services provided to individuals with autism due to the cognitive styles and learning preferences of the population.

Goals and Objectives

The goals of computer-based intervention are as varied as the purposes for them. Recently, a computer-based intervention can be found for nearly every area of difficulty afflicting

individuals with autism from vocabulary to attention to pragmatic abilities. The ultimate goal for these programs could be generalization of the skills learned to the individuals' natural environment. However, some programs aim at allowing the individual user practice at a certain skill. In this case, the software is a tool to be used as part of a rehabilitation package. It is the responsibility of the intervener to help the individual with autism to generalize the skills learned and practiced.

Treatment Participants

Due to their versatility, ability to be individualized, and recent availability (related to smaller size and lower price), computers and technology are being used to a much greater extent today than ever before. Technology is a mainstay of the general education curriculum as well as the special education curriculum. Because of this, computer-based interventions could be appropriate for any individual.

Treatment Procedures

The specific procedures vary by the computer-based intervention. Due to the variety of programs available and the myriad of difficulties they target, generalizations regarding treatment procedures cannot be made.

Efficacy Information

Computers have been used successfully in interventions with individuals with autism (Chen and Bernard-Opitz 1993; Colby 1973; Higgins and Boone 1996). Computers are predictable, cueing can be systematically removed to promote independence in a skill, and an individual can practice skills in an environment which is nonjudgmental. These factors have increased the likelihood of the success of an intervention which utilizes some kind of computer-based intervention. The outcome of research that compares computer-based intervention with teacher-based intervention

favors computer-based intervention (Bernard-Opitz et al. 1990). Compared with traditional instruction, children with autism have been shown to attempt more responses, answer more questions correctly, and had improved behavior and developed better literacy skills with a treatment that consisted of computer-based instruction (Chen and Bernard-Opitz 1993; Heimann et al. 1995).

An underlying question in the above studies pertained to generalization. Can an individual with autism transfer the skills learned from a computer-based intervention to real life? A computer-based intervention used by Hertzroni and Tannous (2004) was shown to generalize to the children's natural classroom environment. After children with autism were exposed to the computer-based intervention that focused on the use of relevant utterances and reduction of the amount of echolalic utterances, the children produced more relevant speech and reduced the number of immediate echolalic utterances (Hertzroni and Tannous 2004). A review of computer-based intervention was recently published by Pennington (2010). In that review, the results of 15 studies showed that targeted academic skills were acquired by the participants with autism spectrum disorders. Pennington (2010) concludes that computer-based instruction is a promising area of intervention for this population.

Outcome Measurement

Generalization of the target skills acquired from computer-based intervention to a naturalistic setting is what should be measured as the outcomes of this type of intervention. Although it is important for individuals to be able to acquire and practice skills in the relative safety of a computer program, it is more important for them to be able to use the skills in their everyday lives.

Qualifications of Treatment Providers

Many of the commercially available computer-based intervention software packages are easy to

install and, their use is fairly intuitive. However, for the computer-based intervention to truly have an impact on the language or cognitive skills of an individual with autism, a trained interventionist should prescribe their use and oversee the effectiveness of the tool. Speech-language pathologists, occupational therapists, special educators, and autism specialists are all trained in the use of these types of tools and can help with knowing which computer-based interventions would be most appropriate. These interventionists can also help with monitoring the outcomes of the intervention and will know how quickly cues and prompts can be faded to result in the most efficient and effective treatment.

See Also

- [Academic Supports](#)
- [Education](#)

References and Reading

- Bernard-Opitz, V., Ross, K., & Tuttas, M. L. (1990). Computer assisted instruction for autistic children. *Annals of the Academy of Medicine*, 19, 611–616.
- Blischak, D. M., & Schlosser, R. W. (2003). Use of technology to support independent spelling by students with autism. *Topics in Language Disorders*, 23, 293–304.
- Bosseler, A., & Massaro, D. (2003). Development and evaluation of a computer-animated tutor for vocabulary and language learning in children with autism. *Journal of Autism and Developmental Disorders*, 33, 653–672.
- Chen, S. H., & Bernard-Opitz, V. (1993). Comparison of personal and computer-assisted instruction for children with autism. *Mental Retardation*, 31, 368–376.
- Colby, K. (1973). The rationale for computer-based treatment of language difficulties in non-speaking autistic children. *Journal of Autism and Childhood Schizophrenia*, 3, 254–260.
- Frost, R. E. (1981). An interactive computer environment for autistic children. In *Proceedings of the Johns Hopkins first national search for applications of personal computing to aid the handicapped*. Los Angeles: IEEE Computer Society.
- Goeffrion, L. D., & Goldenberg, E. P. (1981). Computer-based learning systems for communication-handicapped children. *Journal of Special Education*, 15, 325–332.
- Goldenberg, E. P. (1979). *Special technology for special children*. Baltimore: University Park Press.
- Heimann, M., Nelson, K. E., Tjus, T., & Gillberg, C. (1995). Increasing reading and communication skills in children with autism through an interactive multimedia computer program. *Journal of Autism and Developmental Disorders*, 25, 459–480.
- Hertzroni, O. E., & Tannous, J. (2004). Effects of a computer-based intervention program on the communicative functions of children with autism. *Journal of Autism and Developmental Disorders*, 34, 95–113.
- Higgins, K., & Boone, R. (1996). Creating individualized computer assisted instruction for students with autism using multimedia authoring software. *Focus on Autism and Other Developmental Disabilities*, 11, 69–78.
- Panyan, M. V. (1984). Computer technology for autistic students. *Journal of Autism and Developmental Disorders*, 14, 375–382.
- Panyan, M., McGregor, G., Bennett, A., Rysticken, N., & Spurr, A. (1984). *The effects of microcomputer based instruction on the academic and social progress of autistic students*. Paper presented at the CEC technology in special education conference, Reno.
- Pennington, R. C. (2010). Computer-assisted instruction for teaching academic skills to students with autism spectrum disorders: A review of literature. *Focus on Autism and Other Developmental Disabilities*, 25, 239–248.
- Pleinis, A., & Romanczyk, R. G. (1983). *Computer assisted instruction for atypical children: Attention, performance, and collateral behavior*. Paper presented at the applied behavior analysis conference, Milwaukee.
- Robins, B., Dickerson, P., Stribling, P., & Dautenhahn, K. (2004). Robot-mediated joint attention in children with autism. *Interaction Studies*, 5, 161–198.
- Robins, B., Dautenhahn, R., Boekhorst, R. T., & Billard, A. (2005). Robotic assistants in therapy and education of children with autism: Can a small humanoid robot help encourage social interaction skills? *Universal Access in the Information Society*, 4, 105–120.
- Self, T., Scudder, R. R., Weheba, G., & Crumrine, D. (2007). A virtual approach to teaching safety skills to children with autism spectrum disorders. *Topics in Language Disorders*, 27, 242–253.

COMT

- [Catechol-O-Methyltransferase](#)

Conceptually Accurate Signed English (CASE)

- [Manual Sign](#)
- [Sign Language](#)

Conduct Disorder

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Synonyms

Behavioral disorder; Challenging behavior; Disruptive behavior; Dissocial behavior; Problem behavior

Short Description or Definition

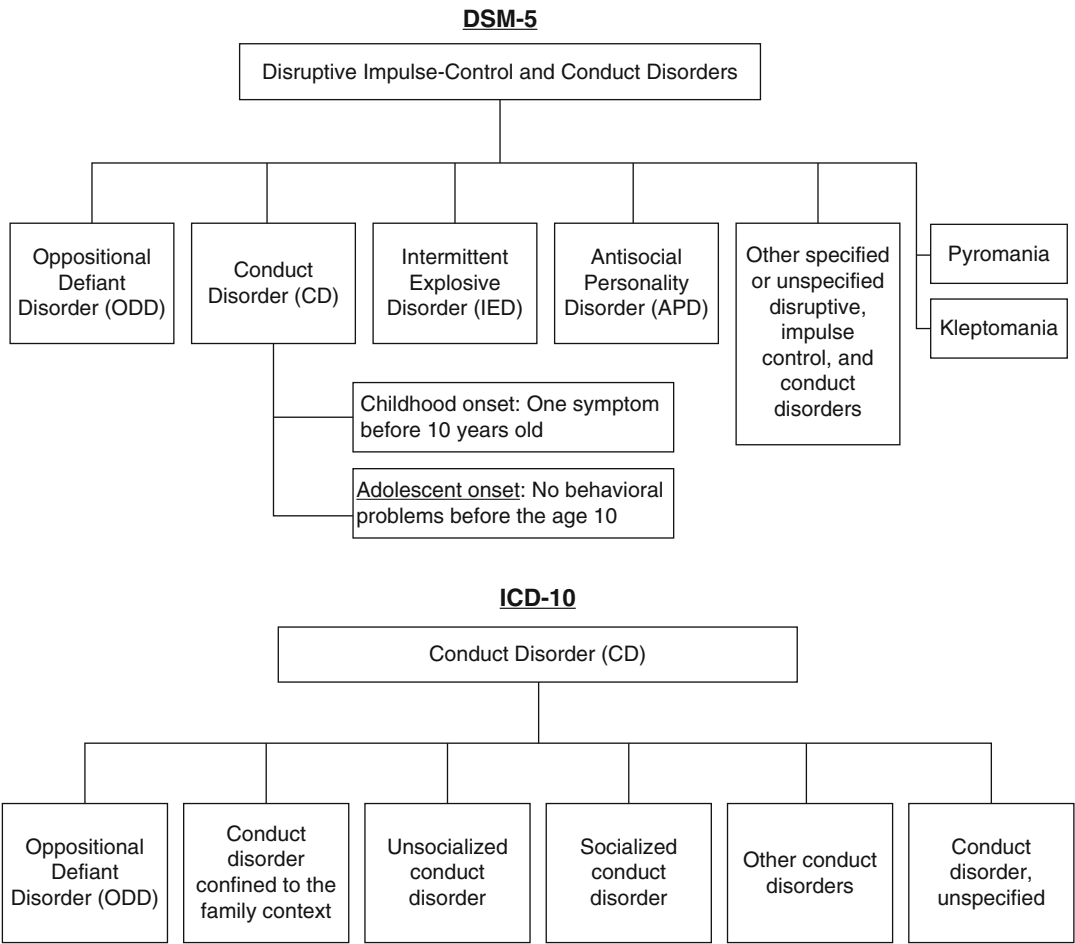
Conduct disorder (CD) is a behavioral disturbance occurring in childhood and adolescence characterized by a persistent and repetitive pattern of behavior that violates the basic rights of others or major age-appropriate societal rules. CD involves a number of problematic behaviors, including oppositional and defiant behaviors, and antisocial activities (e.g., lying, stealing, running away from home, truancy, physical aggression, and coercive behaviors).

People with autism spectrum disorder (ASD) may have several problematic behaviors that are also found in people with CD, such as hyperactivity, impulsivity, aggression, and difficulty with following directions or being cooperative, and both conditions may share other features such as deficit in attributing mental states, poor verbal abilities for describing affective states, or reduced pragmatic skills. It is, however, rare that both conditions occur together (i.e., as comorbid conditions) (Simonoff et al. 2008). While presenting problematic behavior in individuals with CD is considered to arise from abnormal learning, problematic behavior of individuals with ASD can be explained by their difficulties in developing social and communicative skills.

Categorization

CD is classified in the *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition (DSM-5) (American Psychiatric Association [APA] 2013) within the Disruptive, Impulse-Control, and Conduct Disorders group, which also includes Oppositional Defiant Disorder (ODD), Intermittent Explosive Disorder (IED), Antisocial Personality Disorder (APD), Pyromania, Kleptomania, and Other specified or unspecified disruptive, impulse control, and conduct disorders. Because all they share problems with emotional and behavioral self-control, this new group brings together disorders previously described in the chapter Disorders usually first diagnosed in infancy, childhood, or adolescents and in the chapter Impulse control disorders not otherwise specified (see Fig. 1). However, the International Statistical Classification of Diseases (ICD-10) (World Health Organization [WHO] 1992) categorizes the ODD as a particular form of CD, usually occurring in younger children, primarily characterized by markedly defiant, disobedient, disruptive behavior that does not include delinquent acts, or the more extreme forms of problem behavior. On the other hand, dimensional classification systems, based on studies of factor analysis to cluster specific behavioral symptoms, place both disorders within the group of externalizing disorders (Achenbach and Rescorla 2001).

There are several reasons for classifying CD and ODD as two different categories of disorders. First, age of onset for ODD (usually before age 8) is always at an earlier age than the onset of CD, which can have a childhood onset (if at least one symptom is present before 10 years old) or an adolescent onset (if no behavioral problems occur before the age 10). The second reason is that only a small group of children with ODD will develop CD at later ages. Finally, the third reason is that, although both disorders share behavioral characteristics of aggressiveness, aggressive behaviors are qualitatively different for each type of disorder. This distinction has been demonstrated by Frick et al. (1993) who showed that CD and ODD can be differentiated from a dimensional point of view. They conducted a meta-analysis with 60 factors



Conduct Disorder, Fig. 1 Classification of conduct disorder according to DSM-5 (APA 2013) and ICD-10 (WHO 1992)

identifying two bipolar dimensions: “destructive-nondestructive” behaviors and “overt-covert” behaviors. They concluded that most conduct problems could be classified within these two orthogonal dimensions. The overt-nondestructive cluster reflected the criteria for ODD, whereas the other three clusters, with features more indicative of property and status violations, represented CD symptoms (see Fig. 2).

For diagnosis of CD, the individual must have presented in the past 12 months “a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated” (APA 2013). Behaviors that must be present fall into the following four categories of aggressive behavior:

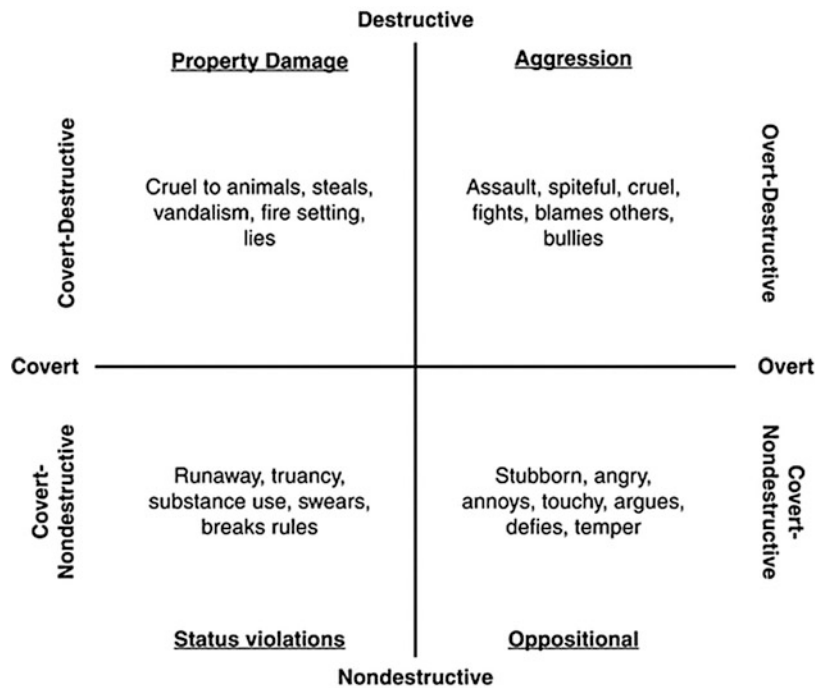
1. Aggressive acts toward people or animals
2. Destruction of property
3. Deceit or theft
4. Violation of rules

At least one of the above criteria must have been presented during the last 6 months. Moreover, the disturbance in behavior must cause clinically significant impairment in social, academic, or occupational functioning.

The age of onset determines the type of CD (see Fig. 1), and the severity depends on the damage caused to others. The behaviors that characterize a mild severity include lying, truancy, and running away at night without permission. Moderate severity may include acts of vandalism and

Conduct Disorder,

Fig. 2 Classification of disruptive behaviors (Adapted from Frick et al. 1993)



theft without confrontation. Severe cases include forced sex, physical cruelty, and use of a weapon and breaking and entering.

Recent studies indicate that a significant proportion of children with ASD may also be characterized with ODD symptoms. For example, two recent studies found that the percentage of children with ASD who meet DSM-IV criteria for ODD is 13% in the age group of 3–5 years and 27% in the age group of 6–12 years according to parental ratings, and when rates from teachers were considered, the numbers were 21% and 25%, respectively (Gadow et al. 2004, 2005).

Problem behaviors that are most often identified in individuals with ASD are physical aggression, self-injury, destruction of property, arguing nature, temper tantrums, and disruption. But other behaviors, such as explosive behavior, running away, stubbornness, violation of rules, defiant, threatening, or not to accept being guilty, have also been identified as moderate or severe conduct problems in people with ASD (Lecavalier 2006). Individuals with ASD present more problem behaviors than typically developing children, and overall levels of problem behavior are positively correlated with severity of ASD (Matson

et al. 2009). About one third of individuals with intellectual disability (ID) who exhibit problem behavior have comorbid diagnosis of ASD (Myrbakk and von Tetzchner 2008), and the more severe the ID, the greater the risk of problem behavior (Holden and Gitlesen 2006).

A common way of categorizing problem behaviors in people with ASD is based on the function of these behaviors in their natural context, but there are no systematic reviews about the possible functions that may play a role. In addition, a behavior problem may have more than one function. The most common functions that can be found in the literature are attention-seeking, avoiding, tangible benefit, or being alone (i.e., nonsocial, self-stimulatory, or automatic reinforcement). Avoiding pain or discomfort has also been described as a possible function of problem behavior.

Epidemiology

Rates of prevalence estimates of CD vary widely depending on the methodology used in each study and on the ascertainment procedures. The disorder

is considered to be a common mental health problem in children and adolescents and appears to have increased in the recent years. CD may be higher in urban than in rural areas and appears more often in boys than girls. Prevalence rates for the disorder in childhood and adolescence range from 2% to 10% in nonreferred samples (APA 2013). Prevalence rates increase from infancy to adolescence and are higher in males than in females. Recent studies show prevalence rates of 9.5% (12% for males and 7.1% for females) (Nock et al. 2006). It seems that male-female ratios might be stronger in childhood than in adolescent-onset groups.

Although individuals with ASD often exhibit behavior problems that could have a negative impact in everyday activities, and several studies suggest a high prevalence of aggressive behavior in people with ASD, few studies have examined the prevalence of maladaptive behaviors that warrant a clinical diagnosis of CD in individuals with ASD. Prevalence of problem behaviors within the ASD population is relatively high. Most studies indicate that at least half of the people with ASD exhibit behavior problems, with an estimated prevalence ranging between 35.8% and 94.3% (Kozlowski and Matson 2012).

Lecavalier (2006) presented a study on prevalence of problem behaviors of children and adolescents with ASD. In a sample of 303 children and adolescents with ASD, he found that the proportion of children and adolescents who, according to parents and teachers, showed “conduct problems” was 13.9% (parents) and 8.4% (teachers). Behavior problems rated by parents and teachers as more frequent were stubbornness, temper tantrums, defiant behavior, arguing nature, not to accept being guilty, and explosive behavior. Stubborn behavior was rated as a moderate or severe problem for 50.7% (parents) and 44.4% (teachers). Also, temper tantrums, defiant behavior, not to accept being guilty, and explosive behavior were classified with a high frequency. Finally, aggressive acts, such as attacking others, were observed by teachers in 14.3% and in 9.9% by parents. Both informants (parents and teachers) indicate physical fights as moderate-to-serious problem for 5.3% of the sample. Property

destruction was a moderate or severe problem for 11–12% (according to information from parents and teachers, respectively), and threatening people was rated as a moderate-to-severe problem for 4.5% (parents) and 7.6% (teachers) of the sample. The study also found that lower adaptive skills were associated with greater problem behaviors among the sample.

Regarding high-functioning individuals with ASD, research and clinical observations suggest that a relatively large number of these individuals have behavioral problems at some point in their development. These symptoms may indicate the presence of ODD comorbid with ASD. They may also have symptoms of CD that are more severe in school-age time than in preschool (Gadow et al. 2005).

Natural History, Prognostic Factors, and Outcomes

The onset of CD can occur very early, even at preschool age, although the most obvious symptoms usually occur between middle childhood and middle adolescence. Onset is rare after age 16. ODD is a common precursor to the childhood-onset type. Other different factors affect the onset of symptoms of CD. The scientific literature highlights three main factors:

1. Personal characteristics such as a difficult temperament in early childhood, a callous-unemotional personality style (lack of empathy, remorselessness, and shallow affected), propensity for risk-taking, low to threatening and emotional reactions stimuli, reduced sensitivity to cues of punishment, and low levels of conscience and moral development
2. Bad parenting practices such as harsh, punitive, abusive, and/or inconsistent discipline
3. Repeated peer rejection and socializing with a deviant peer group (Hughes et al. 2008).

In addition, there is evidence suggesting that childhood-onset CD could be more related to personal and familial factors, whereas adolescent-onset could be more related to exposure to deviant

peers and environmental disadvantages associated with ethnic minority status (McCabe et al. 2001). Finally, CD in childhood is associated with other problems, including the likelihood of repeating a grade in school, being suspended or expelled from school, an earlier age of onset of alcohol dependence, and having to attend a greater number of treatments for drug abuse (Hughes et al. 2008). There is not a single risk factor that determines the onset of the CD. Experts emphasize that the multiple risk factors mentioned above interact to facilitate and perpetuate the disorder.

Problem behaviors play a critical role in ASD. However, the heterogeneity of symptoms present in persons with ASD (differences in cognitive functioning, or in adaptive behavior, the nature and severity of autistic behaviors) and changes in the development difficult to understand how these individual differences affect the occurrence and presentation of behavior problems beyond the core symptoms that define the ASD population.

Despite the differences, consequences of problem behaviors are similar in most cases. These behaviors prevent the development of social relationships (Matson et al. 2010; Myrbakk and von Tetzchner 2008), place the individual and their family members in very difficult situations, and interfere with effective education (Carr et al. 1991). Also, it has been shown that the fact of problem behaviors (specially aggressiveness) causing more distress to caregivers than the core autistic symptoms (Lecavalier et al. 2006) is one of the most important impediments to placement in less-restrictive environments (Shoham-Vardi et al. 1996), can also interfere with intervention efforts, and, if present during early childhood, is of particular concern given that these are critical years for intervention. Thereby problem behaviors impact the long-term prognosis.

Research on risk factors has provided some important data. Tonge and Einfeld (2003) studied a group of 118 children with autism in a period of 8 years. Results indicated that 73.5% of children with autism had behavioral alterations in a clinically significant range, with scores fairly stable over time. These researchers reported that children and adolescents with ASD are at high risk of severe and persistent behavioral disturbances

beyond those that define the disorder. Matson et al. (2009) have studied the potential causal factors of problem behaviors in children with ASD, showing that overall levels of problem behavior were positively correlated with severity of ASD (Matson et al. 2009). Lecavalier (2006) in his epidemiological study found that lower adaptive skills were associated with greater behavioral problems, but age and gender do not seem to influence behavior problems. In a recent study, Hartley et al. (2008) examined a large sample of children with ASD, classifying 27% of sample in the clinically significant range on the CBCL Externalizing Problems' subscale, and 22% fell within the clinically significant range on the aggression subscale. Results indicated that externalizing problems were significantly correlated with poorer adaptive skills, lower nonverbal cognitive functioning, and poorer expressive language. Also Dominick et al. (2007) found that individuals with ASD with low cognitive functioning and adaptive behavior and with low-expressive language skills exhibit more problem behavior than high-functioning individuals (Dominick et al. 2007).

Clinical Expression and Pathophysiology

CD may manifest itself in various symptoms that are classified into four categories: aggression toward people or animals, destruction of property without aggression, deception or theft, and serious violation of the social rules. These symptoms are behaviors that usually occur in early childhood. Many children commit acts of aggression, break property of others, commit petty thefts, say some lies, and violate some social rules. But in the case of children who have a CD, all these behaviors are very frequent and persistent, and some appear in an age too early such as running away from home at night (with no objective reasons to escape such as being abused) or truancy before age 13.

The clinical expression of problem behaviors in ASD will depend on the subject' age and on whether it is associated with ID individuals with greatest deficits engaging in more severe problem behaviors. Severity of ASD symptomatology

affects the severity of problem behaviors. Also, symptoms of other disorders such as attention-deficit hyperactivity disorder or obsessive compulsive disorder will affect the clinical expression of conduct problems in people with ASD. But it remains unknown whether the comorbidity of ASD with these disorders leads to different clinical expressions of behavior problems. This is important to better understand the pathophysiology of CD (and also of ASD). The neurobiological disorder of ASD results in difficulties in social cognition with its own characteristics, such as the difficulty to infer mental states and recognize (e.g., intentions, beliefs, desires, etc.) in self and others, which can interact with environmental factors leading to atypical behavioral patterns and behavior problems.

Some CD symptoms (such as physical aggression, lying, and stealing) are relatively common in early childhood, and to distinguish them from normal childhood behavior, the clinician must take into account the frequency and persistence of problem behavior beyond the age of four. In childhood, most of the manifestations are limited to family and school contexts, but they affect the overall functioning of the child. In adolescence, behavior problems tend to have more serious consequences encompassing the whole adolescent's social setting and including behavior problems that are much more serious.

Evaluation and Differential Diagnosis

CD is a complex problem affecting multiple domains of functioning and often showing a high rate of comorbidity with other disorders. Assessment requires a comprehensive approach encompassing the child, family, school, peers, and community factors. Well-trained professionals should conduct assessments, to ensure the proper treatment. Otherwise, the behavior problems will continue or even worsen.

Language disorders and intellectual disability usually found in ASD complicate the assessment and diagnosis. Even mild deficiencies in language can make challenging the study and identification of subtle differences in emotions, health status

(presence of pain or physical discomfort), or feelings of annoyance at being unable to control own decisions.

Assessment requires combining qualitative and quantitative methods to obtain information, and collecting data from several sources such as parents, teachers, and peers is mandatory. Information from different sources should be compared to detect and prevent possible biases caused by the partial view of each reporter and for a better understanding of the disorder being evaluated.

Behavior rating scales can be completed by parents, teachers, and children to obtain comparable information. Clinicians and researchers consider behavior rating scales a time-efficient method of collecting reliable information and providing an assessment of several domains of behavior, usually both on the healthy functioning and the maladaptive.

Behavioral observation is a useful tool that is normally used in assessing problem behaviors, both to describe the function (i.e., functional assessment) of problem behavior and to monitor treatment progress. Functional assessment is the process of identifying the variables that predict and maintain problem behavior. The literature review on the treatment of problem behaviors suggests that interventions based on functional assessment are more likely to produce the reduction of problem behavior (Homer et al. 2002). The process of conducting a functional assessment typically involves the following: (1) identifying the problem behavior, (2) building hypotheses about the events that occasion and maintain problem behavior, (3) testing/confirming the functional hypothesis, and (4) designing an intervention based on the confirmed information (Homer et al. 2002).

Treatment

A variety of treatment procedures have been developed for children and adolescents with CD, but only some have been shown to reduce CD behaviors. Intervention procedures seem to be more effective in children under 8 years, when behavior problems have recently begun and when it includes a multimodal and multicomponent

strategy, specifically adapted to the individual needs (Hughes et al. 2008).

The evidence-based recommendations emphasize the need for a multicomponent intervention aimed at prevention and early intervention. The treatment mainly consists of psychological, educational, and social interventions focusing on children, parents, and teachers. Psychotropic medication and applied behavior analysis are the most frequently used treatments.

Child-directed interventions aim to improve skills to manage anger and control of aggressive impulses, and improve empathy with others, strengthening relationships with peers. With the family it is necessary first to ensure commitment and motivation and then to begin training within the family context and subsequently generalized to other places of the community (INSERM 2005). Pharmacological treatment is appropriate when used in the context of a comprehensive psychoeducational evaluation and provided as part of a global treatment strategy (Connor 2002).

The best practice for psychoeducational treatment of behavior problems should be based on principles and methods of positive behavior support (Rogers and Vismara 2008). That is, it should be a treatment that uses functional assessment to determine the function (or functions) of problem behavior. After identifying the function, a positive behavior support plan must be designed and implemented, aimed at teaching new functional behaviors that serve to replace the problem behavior. Horner et al. (2002) suggest that a support plan should take into account several elements, and a few of those are as follows:

1. Prevent behavior problems by organizing the environment in order to experience less negative events and greater accessibility of rewarding activities.
2. If there are behavioral problems, conduct a functional assessment.
3. Build a behavioral intervention to make the problem behavior irrelevant, by teaching socially appropriate behaviors that make the person much more competent in the context and produce the same effect in the context of the problem behavior.
4. Organize the consequences for appropriate behavior to compete with problem behavior, avoiding also the reinforcement of problem behavior.
5. Ensure that the procedures are within the skills, resources, and values of those who must implement them.

Parent training based on principles of applied behavior analysis has great empirical support. Successful programs for individuals with ASD and problem behavior include a parent training component usually based on principles of applied behavior analysis. Common parent training elements include teaching behavioral principles and management techniques, role-playing, homework assignments, teaching play and social skills, use of visual schedules, and home visits or telephone consultation, among others.

Pharmacotherapy is common among individuals with ASD with behavior difficulties. The most used agents include selective serotonin reuptake inhibitors, antipsychotics, alpha 2 adrenergic agonists, psychostimulants, and anticonvulsants. But empirical support for use of these agents in ASD varies widely. A classic study on the use of Risperidone (McCracken et al. 2002) concluded that this psychoactive drug is effective and well tolerated for the treatment of tantrums, aggression, or self-injurious behavior in children with ASD, although the short period of the trial in this study limits inferences about adverse effects. Risperidone, like other atypical antipsychotics, is associated with adverse events, such as weight gain, and the subsequent risk of metabolic syndrome. A recent trial by Aman et al. (2009) tested whether combined treatment with risperidone and parent training in behavior management is superior to medication alone in improving severe behavioral problems. Results indicated that parent training plus medication produced greater reduction of problem behavior than medication alone in children with ASD.

See Also

- [Behavior Analysis](#)
- [Behavior Modification](#)

- Behavior Plan
- Behavioral Assessment
- Challenging Behavior
- Maladaptive Behavior
- Positive Behavioral Support

References and Reading

- Achenbach, T. M., & Rescorla, L. A. (2001). *Manual for ASEBA school-age forms & profiles*. Burlington: University of Vermont, Research Center for Children, Youth, & Families.
- Aman, M. G., McDougle, C. J., Scahill, L., Handen, B., Arnold, L. E., et al. (2009). Medication and parent training in children with pervasive developmental disorders and serious behavior problems: Results from a randomized clinical trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(12), 1143–1154.
- American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders*, fifth edition, DSM-5. Washington, DC: American Psychiatric Publishing.
- Carr, E. G., Taylor, J. C., & Robinson, S. (1991). The effects of severe behavior problems in children on the teaching behavior of adults. *Journal of Applied Behavior Analysis*, 24(3), 523–535.
- Connor, D. F. (2002). *Aggression and antisocial behavior in children and adolescents: Research and treatment*. New York: Guilford Press.
- Dominick, K. C., Davis, N. O., Lainhart, J., Tager-Flusberg, H., & Folstein, S. (2007). Atypical behaviors in children with autism and children with a history of language impairment. *Research in Developmental Disabilities*, 28, 145–162.
- Frick, P. J., Van Horn, Y., Lahey, B. B., Christ, M. A. G., Loeber, R., Hart, E. A., et al. (1993). Oppositional defiant disorder and conduct disorder: A meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clinical Psychology Review*, 13, 319–340.
- Gadow, K. D., DeVincent, C. J., Pomeroy, J., & Azizian, A. (2004). Psychiatric symptoms in preschool children with PDD and clinic and comparison samples. *Journal of Autism and Developmental Disorders*, 34(4), 379–393.
- Gadow, K. D., Devincent, C. J., Pomeroy, J., & Azizian, A. (2005). Comparison of DSM-IV symptoms in elementary school-age children with PDD versus clinic and community samples. *Autism*, 9(4), 392–415.
- Hartley, S. L., Sikora, D. M., & McCoy, R. (2008). Prevalence and risk factors of maladaptive behaviour in young children with autistic disorder. *Journal of Intellectual Disability Research*, 52(10), 819–829.
- Holden, B., & Gitlesen, J. P. (2006). A total population study of challenging behaviour in the county of Hedmark, Norway: Prevalence, and risk markers. *Research in Developmental Disabilities*, 27(4), 456–465.
- Horner, R. H., Carr, E. G., Strain, P. S., Todd, A. W., & Reed, H. K. (2002). Problem behavior interventions for young children with autism: A research synthesis. *Journal of Autism and Developmental Disorders*, 32(5), 423–446.
- Hughes, T., Crothers, L., & Jimerson, S. (2008). *Identifying, assessing, and treating conduct disorder at school*. New York: Springer.
- INSERM. (2005). *Conduct disorder in children and adolescents. Collective expert report*. France: Institut national de la santé et de la recherche médicale. Available from <http://www.ncbi.nlm.nih.gov/books/NBK7133/>
- Kim-Cohen, J., Arseneault, L., Caspi, A., Tomás, M. P., Taylor, A., & Moffitt, T. E. (2005). Validity of DSM-IV conduct disorder in 4½-5-year-old children: A longitudinal epidemiological study. *American Journal of Psychiatry*, 162, 1108–1117.
- Kozlowski, A. M., & Matson, J. L. (2012). An examination of challenging behaviors in autistic disorder pervasive developmental disorder not otherwise specified: Significant differences and gender effects. *Research in Autism Spectrum Disorders*, 6, 319–325.
- Lahey, B. B., Loeber, R., Burke, J. D., & Applegate, B. (2005). Predicting future antisocial personality disorder in males from a clinical assessment in childhood. *Journal of Consulting and Clinical Psychology*, 73, 389–399.
- Lecavalier, L. (2006). Behavioral and emotional problems in young people with pervasive developmental disorders: Relative prevalence, effects of subject characteristics, and empirical classification. *Journal of Autism and Developmental Disorders*, 36, 1101–1114.
- Lecavalier, L., Leone, S., & Wiltz, J. (2006). The impact of behaviour problems on caregiver stress in young people with autism spectrum disorders. *Journal of Intellectual Disability Research*, 50(Pt 3), 172–183.
- Matson, J. L., Wilkins, J., & Macken, J. (2009). The relationship of challenging behaviors to severity and symptoms of autism spectrum disorders. *Journal of Mental Health Research in Intellectual Disabilities*, 2, 29–44.
- Matson, J. L., Neal, D., Fodstad, J. C., & Hess, J. A. (2010). The relation of social behaviours and challenging behaviours in infants and toddlers with autism spectrum disorders. *Developmental Neurorehabilitation*, 13(3), 164–169.
- Maughan, B., Rowe, R., Messer, J., Goodman, R., & Meltzer, H. (2004). Conduct disorder and oppositional defiant disorder in a national sample: Developmental epidemiology. *Journal of Child Psychology and Psychiatry*, 45, 609–621.
- McCabe, K. M., Hough, R., Wood, P. A., & Yeh, M. (2001). Childhood and adolescent onset conduct disorder: A test of the developmental taxonomy. *Journal of Abnormal Child Psychology*, 29, 305–316.
- McCracken, J. T., McGough, J., Shah, B., Cronin, P., Hong, D., Aman, M. G., et al. (2002). Risperidone in children with autism and serious behavioral problems. *New England Journal of Medicine*, 347(5), 314–321.

- Myrbakk, E., & von Tetzchner, S. (2008). Psychiatric disorders and behavior problems in people with intellectual disability. *Research in Developmental Disabilities, 29*(4), 316–332.
- Nock, M. K., Kazdin, A. E., Hiripi, E., & Kessler, R. C. (2006). Prevalence, subtypes, and correlates of DSM-IV conduct disorder in the National Comorbidity Survey Replication. *Psychological Medicine, 36*, 699–710.
- Rogers, S. J., & Vismara, L. A. N. (2008). Evidence-based comprehensive treatments for early autism. *Journal of Clinical Child and Adolescent Psychology, 37*, 8–38.
- Shoham-Vardi, I., Davidson, P. W., Cain, N. N., Sloane-Reeves, J. E., Giesow, V. E., Quijano, L. E., et al. (1996). Factors predicting re-referral following crisis intervention for community-based persons with developmental disabilities and behavioral and psychiatric disorders. *American Journal of Mental Retardation, 101*(2), 109–117.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 47*, 921–929.
- Tonge, B. J., & Einfeld, S. L. (2003). Psychopathology and intellectual disability: The Australian child to adult longitudinal study. In L. M. Glidden (Ed.), *International review of research in mental retardation* (Vol. 26, pp. 61–91). San Diego: Academic.
- World Health Organization. (1992). *International classification of diseases: Diagnostic criteria for research* (10th ed.). Geneva: Author.

Web-Sites

American Academy of Child and Adolescent Psychiatry (AACAP). <http://www.aacap.org>
 ConductDisorders. <http://www.conductdisorders.com>
 Internet Mental Health. <http://www.mentalhealth.com/>

Confidentiality

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Definition

Confidentiality refers to a general standard of professional conduct, as well as, in some cases, the legal requirement for providers and/or

researchers to maintain the privacy of a patient's personal health information and records unless consent to release the information is obtained from the patient. In the case of minors, this consent may be obtained from the patient's parent(s) or legal guardian(s), and rules around confidentiality related to minors may vary by state.

Historical Background

Confidentiality is important in order to establish trust between providers and patients and to protect patient privacy. Confidentiality can be bound by both ethical and legal standards. For example, the American Psychological Association's Ethical Principles of Psychologists and Code of Conduct provides ethical guidelines regarding a psychologist's obligation to take reasonable precautions to protect confidential information related to the patient (Standard 4; APA 1992). In 1996, The U.S. Department of Health and Human Services (HHS) established the Health Insurance Portability and Accountability Act (HIPAA), Public Law 104–191, and issued a Privacy Rule to implement the legal requirement for providers to protect the privacy of a patient's medical records or personal health information, including information related to mental health. In the case of minors, according to HIPAA, parents have the legal right to access their child's records and personal information. In certain specific situations, providers may share information without the patient's consent. Common exceptions may include cases that involve a court order to release protected information, cases in which there is suspicion of domestic violence, abuse or neglect of children, the elderly, or people with disabilities, or in order to protect the patient or the public from serious harm (Tarasoff 1974, 1976).

Current Knowledge

Since it was created, the HIPAA has undergone several revisions and updates in order to further protect the confidentiality and privacy of health information, including electronic protected health

information (PHI). In 2002, the HHS modified the Privacy Rule associated with HIPAA to set national standards for the protection of individually identifiable health information by health plans, health care clearinghouses, and health care providers. In 2003, HHS published a final Security Rule to set national standards for protecting the confidentiality of electronic PHI.

Future Directions

Confidentiality remains a sensitive and, at times, controversial topic, particularly as it relates to determining exceptions or limitations to confidentiality, such as in the treatment of minors or when the risks may outweigh the benefits (e.g., Tarasoff 1974, 1976). Some have proposed guidelines and recommendations related to dealing with issues of confidentiality when working with various populations (APA 1992; Gustafson and McNamara 1987; Koocher and Keith-Spiegel 1990). Future investigations may focus on further delineating the ethical and legal boundaries and limits around cases of confidentiality. Furthermore, research should explore the important considerations to make with regards to assessing competence and respecting confidentiality in individuals with autism spectrum disorders and other developmental disabilities.

See Also

- [Consent](#)
- [Ethics](#)

References and Reading

- American Psychological Association. (1992). Ethical principles of psychologists and code of conduct. *American Psychologist*, 47, 1597–1611.
- Gustafson, K. E., & McNamara, J. R. (1987). Confidentiality with minor clients: Issues and guidelines for therapists. *Professional Psychology: Research and Practice*, 18(5), 503–508.
- Health Insurance Portability and Accountability Act of 1996 (HIPAA), Pub. L. No. 104–191, 110 Stat. 1936 (1996).

- Koocher, G. P., & Keith-Spiegel, P. C. (1990). *Children, ethics, and the law: Professional issues and cases*. Lincoln: University of Nebraska Press.
- Tarasoff v. Regents of the University of California, 118 Cal. Rptr. 129, 529 P.2d.533 (Cal. 1974).
- Tarasoff v. Regents of the University of California, 113 Cal. Rptr. 14, 551 P.2d.334 (Cal. 1976).
- U.S. Department of Health and Human Services. Summary of the HIPAA Privacy Rule. Retrieved 1 Dec 2016 from <http://www.hhs.gov/hipaa>

Confirmatory Factor Analysis

- [Latent Variable Modeling](#)

Congenital Aphasia

- [Childhood Aphasia](#)

Congenital Disorders

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Definition

Congenital disorders are those disorders that are present at the time of birth and involve an abnormality of structure and/or function that has arisen during development. Congenital disorders are not necessarily genetic though do include genetic disorders. All genetic disorders are congenital as they are present at birth even if they are not yet detected at birth. Congenital disorders may arise as a result of the intrauterine environment, errors in embryonic development, and infections. The outcome of such disorders varies widely and is dependent upon the disorder itself and the availability of possible postnatal treatments. Examples of congenital disorders include diseases such as cystic fibrosis, physical anomalies such as having a sixth finger on the hand, metabolic diseases such as congenital adrenal hyperplasia, and trisomy 21 which is also known as Down syndrome.

See Also

- [Chromosomal Abnormalities](#)
- [Genetics](#)
- [Inborn Errors of Metabolism](#)

References and Reading

- Kliegman, R. (Ed.). (2007). *Nelson textbook of pediatrics* (18th ed.). Philadelphia: Saunders Elsevier.
- Stocker, J., & Dehner, L. (Eds.). (2001). *Pediatric pathology*. Philadelphia: Lippincott Williams & Wilkins.

Congenital Metabolic Diseases

- [Inborn Errors of Metabolism](#)

Conners' Continuous Performance Test

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Synonyms

CCPT; CPT; CPT-II

Description

The Conners' Continuous Performance Test is an attention test for research and clinical settings (Conners 1995). It is used for measuring processes related to vigilance, response inhibition, signal detection, and other aspects of performance

(Conners et al. 2003). The test is presented in a game-like format where 360 letters (approximately 1 in. in size and bold faced) appear on the computer screen, one at a time, for approximately 250 ms. Respondents are required to press the space bar or click the mouse button when any letter *except* the letter "X" appears on the screen (Conners and MHS Staff 2000). The CPT-II standard paradigm consists of six blocks, with each block divided into three 20-trial sub-blocks. Each sub-block has a separate inter-stimulus interval (i.e., the time in between the letter presentations). The inter-stimulus intervals (ISIs) are 1, 2, or 4 s. The order of the three different ISI conditions varies from block to block (Conners et al. 2003). The CPTII can be completed in 14 min. The test can be administered to participants 6 years of age and above.

After the test session, the program generates a report that includes response times, omission errors (i.e., when a response is not given after a non-X appears on screen), commission errors (i.e., when a response is given after an X appears on screen), change in reaction time speed and consistency as the test progresses, and change in reaction time speed and consistency for different inter-stimulus intervals. Examination of the results by blocks and varying ISIs allows for the assessment of vigilance and the ability to adjust to changing tempo and task demands (Conners and MHS Staff 2000).

Historical Background

The continuous performance test was first introduced by Rosvold and colleagues in 1956 (Spreen and Strauss 1998) to detect lapses of attention in patients with petit mal epilepsy. In this early version, the participants were required to press a key in response to a rare target, such as the letter "X." Subsequent CPTs have made changes to this original paradigm including having the participants press a key when the target letter is preceded by another letter (e.g., "X" preceded by "A") or upon the second successive presentation of a letter (e.g., S-S). There have also been variations with regard to modality (i.e., visual or auditory), the type of

stimuli (e.g., letters, numbers, colors, or geometric figures), and the type of data that are evaluated (e.g., omissions, commissions, inter-stimulus interval, measures of sensitivity; Spreen and Strauss 1998).

Conners' introduction of his version of the CPT in 1995 represented a departure from the more traditional CPT paradigm. In the earlier versions, participants typically sit passively while observing the presentation of nontarget stimuli and must respond to the occasional target stimulus (usually an "X"). In Conners' version, which is also sometimes called the "not-X" CPT, participants are asked to press a button on each trial (usually letters), except for the letter X. Barkley (2006) notes that this task requires a different form of response inhibition. Conners et al. explain that Conners' "not-X" CPT places a greater demand on response inhibition due to the frequent responding interrupted by the occasional nontargets (the less probable "X") as opposed to the more passive responding of the conventional "X" task.

Conners has since come out with an updated version of his CPT, the Conners' Continuous Performance Test (2nd ed.; Conners' CPT-II; Conners and MHS Staff 2000). The updated version differs from the previous version in that it is based on new and expanded norms that include a large subsample of neurologically impaired individuals. This allows for comparison of responses to general population norms, ADHD norms, and neurologically impaired norms. The program itself includes validity checks to flag certain conditions that may adversely affect CPT II administration and a Confidence Index that enables the practitioner to gauge the certainty of the assessment/classification.

Psychometric Data

The CPT-II normative data included 2,521 participants. Of this, 1,920 were healthy individuals from the general population, 378 were diagnosed with Attention-Deficit/Hyperactivity Disorder (ADHD), and 223 were adult individuals identified with some type of neurological impairment

(e.g., head injuries, dementias). Normative data were collected from 30 sites in 16 states and three Canadian provinces. The multi-site, nonclinical data came from schools, organizations, science centers, and controlled research settings. The norms were divided into eight age groups. For children of ages 4 through 17, norms were provided in 2-year increments. For adults aged 18 and older, they were divided into three age groups (18–34, 35–54, and 55 +). The applicability of CPT-II norms to Asian and African American groups was also assessed. Scores for the Asian group were consistent with those obtained in the general population. However, the African American group made slightly fewer commission errors than the general population, and showed slightly better discriminatory power as measured by the statistic d' prime. Overall, the general population norms were reportedly applicable to these minority groups. In fact, there were no significant differences on the overall profile indexes (Conners and MHS Staff 2000).

Three types of reliability information were provided on the CPT-II manual: Split-half reliability, test-retest reliability, and standard error of measurement (Conners and MHS Staff 2000). The split-half reliability information from the original CPT was cited. These appeared adequate and ranged from 0.66 to 0.95. Test-retest reliability was obtained using 23 participants in the standardization of the CPT-II. The average interval between administrations was 3 months. The test-retest reliability estimates ranged from 0.05 to 0.92 with most of the variables showing good consistency across administrations. However, the Block change and ISI change statistics have low test-retest correlations, suggesting that these variables do not produce good consistency across administrations. When measures are combined into indices for ADHD and neurological assessment, the test-retest reliabilities were excellent, 0.89 and 0.92 respectively. Using the same test-retest data, it was also demonstrated that the CPT-II had no significant practice effect. In addition, information on standard error of measurement and standard error of prediction for the various CPT-II measures across gender and age was presented.

Conners and the MHS Staff (2000) cited research to support the clinical utility of the CPT. In a study based on the original standardization sample, significant differences were seen between the ADHD group and other diagnoses across most of the CPT variables. The ADHD group responded more slowly, had greater variability of reaction times, made more omission and commission errors, and was more affected by changes in ISI. In similar analyses using the updated CPT-II data, no significant difference was observed between ADHD and nonclinical groups; for all other analyses, there was a large and significant difference between ADHD and nonclinical groups with the ADHD groups performing worse on all of the measures. For the adults aged 18 years and older, planned comparisons were done to see if the nonclinical group differed from the clinical groups, and if the two clinical groups differed from each other. As predicted, the clinical groups performed significantly worse than the nonclinical group. Compared to the ADHD group, the Neurological group made significantly more omission errors, had significantly slower reaction times, and was significantly less consistent across the interstimulus intervals.

Clinical Uses

The CPT paradigm has traditionally been included in evaluations for ADHD. Barkley (2006) states that, "A wide-ranging literature has shown it to be the most reliable of psychological tests for discriminating groups of children with ADHD from nondisabled children" (p. 377). Spren, Risser, and Edgell (1995) report that on a continuous performance task hyperactive children make more errors of omission and of commission, show more rapid deterioration in performance than controls, and are less able to inhibit premature or repetitive responding, indicating poor impulse control. Lezak, Howieson, and Loring (2004) state that on the CPT, adults with ADHD have a high reaction time variability and higher rate of commission errors than control subjects, which suggests that they have trouble inhibiting responses. According to Spren

and Strauss, the CPTs have also been shown to distinguish between normal controls and certain patient groups including adults with head injuries and children with conduct disorder, learning disabilities, and those at high risk for schizophrenia. In addition, Barkley and Spren and Strauss report that CPTs are sensitive to stimulant drug effects among children and adolescents with ADHD.

Barkley has raised some concern about the diagnostic utility of the Conners' CPT, in particular, in ADHD assessments. Citing one study that investigated associations between Conners' CPT scores and several other measures, including parent and teacher ratings as well as neuropsychological and achievement tests, Barkley reported that the Conners CPT's overall index failed to relate to parent and teacher ratings. In addition, only half of those participants who met criteria for ADHD "failed" the CPT. Barkley also reported poor discriminant validity, in that children with a reading disability actually performed more poorly than children with ADHD. In another study on the ecological validity of the CPT-II in a school-based sample, Barkley cited findings showing nonsignificant relationships between CPT performance and three other kinds of measures (parent ratings, teacher ratings, and classroom observations). He also reported negative correlation between IQ and omission errors on the CPT-II, suggesting that the CPT-II may measure letter recognition skills or phonological awareness rather than impulsivity or inattention per se.

Despite these concerns, Barkley still holds that the CPT is the only psychological measure that seems to directly assess the core symptoms of ADHD, namely, impulsivity and attention. However, he warns that if a child performs well on this measure, it does not indicate that the child is nondisabled or without ADHD because of the high rate of false negatives (i.e., children who are rated by parents and teachers as having ADHD, but who obtain average scores on the test) associated with CPTs. He joins Conners (2000) in reminding the clinician that the test provides one source of information to be integrated with other sources (e.g., self-report data, observer-based data, historical information, interview data,

and results from other tests) in reaching a final diagnostic decision.

The use of continuous performance tests in assessing children with autism spectrum disorders is less common. Three studies were found but none of them used the Conners' CPT-II. In the first study, 23 children with autism were compared with two control groups (one matched based on verbal mental age and another based on performance mental age) on several attention measures including three versions of the traditional CPT paradigm (Pascualvaca et al. 1998). The results showed that none of the CPT versions differentiated between the groups. In the second study, Schatz, Weimer, and Trauner (2002) explored the use of the Test of Variables of Attention (TOVA) in assessing attention deficit symptoms in a group of eight children and young adults with Asperger's Syndrome (AS). The TOVA is a continuous performance test that is similar to the Conners' CPT but with an additional auditory component. Five of eight subjects with AS received scores that suggested the presence of an attention deficit, whereas only two of eight subjects received scores suggestive of an attention deficit. The authors looked at this as a pattern that could be explored using a bigger sample. In the third study, Corbett and Constantine (2006) compared children with autism spectrum disorder (ASD) with those that have been diagnosed with ADHD and typically developing children using the Integrated Visual and Auditory Continuous Performance Test, another version of a CPT. They found that children with ASD show significant deficits in visual and auditory attention and greater deficits in impulsivity than children with ADHD or typically developing children. The authors note that the findings suggest that many of the children with ASD demonstrate significant ADHD-like symptoms. They point out that this study adds to the growing literature that calls into question the current exclusionary practice of offering a diagnosis of ADHD in pervasive developmental disorders.

Two other variables that might be relevant in the performance of individuals with autism spectrum disorders on the CPT-II are anxiety and intelligence. Conners and the MHS Staff presented data showing that anxiety may affect a participant's CPT-II response style and lead to

response inhibition for physiological anxiety, and decrease in response inhibition for cognitive anxiety. In terms of intelligence, the manual included two studies that showed nonsignificant correlations between IQ as measured by the WISC and CPT performance. Still it was noted in the manual that some individuals with severe cognitive impairment, agitation, or severe psychotic symptoms cannot be administered the CPT-II.

See Also

- [Attention](#)
- [Attention Deficit/Hyperactivity Disorder](#)

References and Reading

- Barkley, R. A. (2006). *Attention-deficit hyperactivity disorder: A handbook of diagnosis and treatment* (3rd ed.). New York: The Guilford Press.
- Conners, C. K. (1995). *Conners' continuous performance test computer program: User's manual*. Toronto: Multi-Health Systems.
- Conners, C. K., & MHS Staff. (2000). *Conners' continuous performance test (CPT II): Technical guide and software manual*. Toronto: Multi-Health Systems.
- Conners, C. K., Epstein, J. N., Angold, A., & Klaric, J. (2003). Continuous performance test performance in a normative epidemiological sample. *Journal of Abnormal Child Psychology*, 31, 555–562.
- Corbett, B. A., & Constantine, L. J. (2006). Autism and attention deficit hyperactivity disorder: Assessing attention and response control with the integrated visual and auditory continuous performance test. *Child Neuropsychology*, 12, 335–348.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
- Pascualvaca, D. M., Fantie, B. D., Papageorgiou, M., & Mirsky, A. (1998). Attentional capacities in children with autism: Is there a general deficit in shifting focus? *Journal of Autism and Developmental Disorders*, 28, 467–478.
- Schatz, A. M., Weimer, A. K., & Trauner, D. A. (2002). Brief report: Attention differences in asperger syndrome. *Journal of Autism and Developmental Disorders*, 32, 333–336.
- Spreen, O., & Strauss, E. (1998). *A compendium of neuropsychological tests* (2nd ed.). New York: Oxford University Press.
- Spreen, O., Risser, A. H., & Edgell, D. (1995). *Developmental neuropsychology*. New York: Oxford University Press.

Conners' Parent Rating Scale

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Description

The Conners' Parent Rating Scale (CPRS) is a parent-report measure that assesses children's problem behaviors, particularly symptoms of attention deficit hyperactivity disorder (ADHD) and related disorders (including oppositional defiant disorder and conduct disorder). At the time of publication, the Conners 3-P (2008) is the current version of the CPRS. Parents of children with autism spectrum disorders may be asked to complete the Conners 3-P because of the shared symptoms between ADHD and autism spectrum disorders. The Conners 3-P was developed by C. Keith Conners, Ph.D., who also designed two related measures: the Conners' Teacher Rating Scales (CTRS), a teacher-report measure, and the Conners' Self-Report Scales (CSRS), a self-report measure for children and adolescents. Because these measures are meant to be used in conjunction, the family of Conners' tests is considered to be a "multi-informant" mode of assessment. This is valuable because it can yield information about children's behaviors in multiple settings. For example, asking a parent to complete the Conners 3-P and asking a teacher to complete the Conners 3-T (for "teacher") can shed light on how a child's behavior may differ between home and school.

Historical Background

Gianarris, Golden and Greene (2001) provide a detailed overview of the multiple versions of the Conners' Parent Rating Scales. The roots of the Conners 3-P date back to the 1960s, when C. Keith Conners, Ph.D., created behavior rating scales based on his multiple observations of children and adolescents with behaviors consistent

with what is now known as ADHD. He performed factor analysis to determine how these behaviors fit together and first published his findings in 1970. One of the earliest aims of Conners' work was to track changes in children's behavior following medication use. In 1973, Conners released a 93-item checklist of behaviors that came to be known as the original Conners' Parent Rating Scale. It was quickly adopted as a diagnostic tool even though it did not have a normative sample of the kind structured for empirical support that is required to establish a new assessment tool today. It was not until 1989 that Conners' sample was formalized and expanded, and that the CPRS was published and shared widely. In 1998, the CPRS-R (for "revised") was released, and in 2008, the third and most recent edition, the Conners 3-P, was released. The Conners 3-P is currently available in English and Spanish. The similarities and differences between these versions are explored below in the section "[Psychometric Data](#)."

Psychometric Data

The format and response style of the measure have remained quite consistent throughout the revisions of the Conners 3-P. In all three versions, the respondent (the parent completing the measure) is asked to reflect on his or her child's actions over the past month and to respond to a series of items describing mainly problem behaviors (e.g., "gets distracted when given instructions to do something"). For each item, the respondent is asked to mark 0 ("not true at all/never/seldom"), 1 ("just a little true/occasionally"), 2 ("pretty much true/often/quite a bit"), or 3 ("very much true/very often/very frequent") to describe the extent to which their child engages in or demonstrates the given behavior.

The Conners 3-P has a long-form version (110 questions), a short-form version (45 of the 110 total questions), an ADHD Index (10 of the 110 total questions), and a Global Index (10 of the 110 questions). Previous versions also offered longer and shorter forms. There are circumstances in which one form would be preferable over

another; this largely has to do with the reason for testing. For example, using the long-form version of the Conners 3-P might be preferable to using one of the shorter forms when conducting an initial assessment.

Revisions were made (e.g., transitioning from the CPRS to the CPRS-R, and again from the CPRS-R to the Conners 3-P) in order to strengthen the psychometric properties of the instrument. Most available literature focuses on the psychometric properties of the CPRS-R and the Conners 3-P. The CPRS-R contains the following subscales: Oppositional (long and short forms), Social Problems (long form only), Cognitive Problems/Inattention (long and short forms), Psychosomatic (long form only), Hyperactivity (long and short forms), DSM-IV Symptom Subscales (long form only), Anxious-Shy (long form only), ADHD Index (long and short forms), Perfectionism (long form only), and Conners' Global Index (long form only). The same subscales appear on the CTRS-R with the exception of the Psychosomatic subscale, which is not included. Both raw scores and T scores are generally reported for each subscale; T scores are standardized scores with a mean of 50 and a standard deviation of 10. For example, a child with a T score of 50 on the Oppositional subscale would have about the same level of oppositional behaviors as the average child his or her age in the normative sample. Higher T scores are associated with higher levels of problem behaviors.

Internal consistency coefficients of the CPRS-R (in other words, measures of how well the individual items of the measure "hang" together and form the subscales listed above) for the total sample range from .77 to .96. The test-retest reliability coefficients – a measure of how similarly a child will be rated shortly following an initial assessment with the CPRS-R – range from .47 to .86. As previously discussed, the CPRS-R is designed to be sensitive to changes in behavior, particularly following medication use, and this might explain partially the lower test-retest reliability coefficients.

Some significant changes were made to the CPRS-R to create the Conners 3-P. The publishers of the Conners 3-P point to the large normative sample ($n = 1200$) that reflects the 2000 US

Census information on race and ethnicity, gender, and parental education level as particular strength of the measure. While the test creators were careful to include a diverse normative sample in their development of the Conners 3-P, it is important to point out that the validity of the Conners' tests in diverse cultures has not yet been established and represents an active area to study. The Conners 3-P includes the use of 1-year, instead of 3-year, age bands to compare children's scores to the normative sample (e.g., 4-year-olds are now compared only to other 4-year-olds, instead of to 4-, 5-, and 6-year-olds). Also, the Conners 3-P includes optional combined gender norms for boys and girls – the norms had been strictly separated by gender in the previous versions – and the combined norms can be helpful for understanding behavior within the context of settings, such as the classroom, that are very frequently coed. The Conners 3-P also has a greater focus on differential diagnosis (or, in other words, teasing apart symptoms of ADHD from symptoms of related disorders) and this is reflected in its normative sample. Additionally, the age range of the Conners 3-P, at 6–18 years old, is slightly narrower than the age range, 3–17 years, of the previous CPRS versions. The age range was extended to 18 years, 11 months to capture adjustment through the end of high school; it was limited to 6 years, 0 months so that early experiences can be assessed more thoroughly with a separate measure, the Conners Early Childhood (EC).

The Conners 3-P contains the following content scales: inattention, hyperactivity/impulsivity, learning problems, executive functioning, aggression, and peer/family relations. It also contains four symptoms scales – ADHD Inattentive, ADHD Hyperactive-Impulsive, Conduct Disorder, and Oppositional Defiant Disorder – that map onto the diagnostic criteria put forth in the Diagnostic and Statistical Manual of Mental Disorders, fourth edition, text revision (DSM-IV-TR). New to the Conners 3-P include validity scales (which indicate how a parent is responding to the items and whether or not the information he or she provides is interpretable), screening items for childhood anxiety and depression, critical items (which require immediate follow-up by the researcher or clinician administering the

measure), and impairment items (which indicate decreased functioning in certain life areas, like social relationships). The Conners 3-P is also notable for the way in which it maps onto the Individuals with Disabilities Education Act (IDEA); in other words, how a child is rated by his or her parent on the Conners 3-P might carry implications for the services he or she is eligible to receive in school.

Both test-retest reliability and internal consistency have been found to be very good for the Conners 3-P, and for the overall family of Conners 3 assessments. According to data put forth by the publisher, internal consistency coefficients (in other words, measures of how well the individual items of the measure “hang” together and form the subscales listed above) for the overall content scales is .91 and for the DSM-IV-TR scales, .90. A breakdown of internal consistency coefficients by Conners 3-P subscale are the following: inattention = .93, hyperactivity/impulsivity = .94, learning problems = .90, executive functioning = .92, aggression = .91, peer relations = .85, ADHD Inattentive = .93, ADHD Hyperactive-Impulsive = .92, Conduct Disorder = .83, and Oppositional Defiant Disorder = .83. The 2–4-week test-retest reliability coefficients – a measure of how similarly a child will be rated 2 weeks and again 4 weeks following an initial assessment with the Conners 3-P – was also very good in the overall Conners’ sample (Cronbach’s alpha = .71 to .98, with all correlations significant at the $p < .001$ level). Inter-rater reliability coefficients (a measure of how likely two different respondents, such as a mother and father, or a parent and teacher, are to rate the same child’s behavior) in the overall Conners’ sample are also acceptable to excellent, ranging from .52 to .94. Continuity between the CPRS-R and the Conners 3-P was demonstrated, and tests of factorial, convergent, divergent, and discriminant validity were also performed on the Conners 3-P.

The Conners 3-P may indicate whether a child’s symptoms are consistent with ADHD or a related disorder. While it includes questions about different symptoms in parent-friendly and accessible language, it does not elicit all of the information that would be needed to make a formal diagnosis. It is important to note that a high score on the Conners 3-P alone is not sufficient to diagnose a child; instead, it is only one piece of information that clinicians will consider when making a diagnosis, if one is warranted. If a child has a diagnosis, then the Conners 3-P can be used to track changes in his or her behavior over time; this is especially important if a child receives intervention, medication, or special services to address his or her behavioral challenges. Sometimes, the Conners 3-P is used in the absence of any behavioral problems – it can be used as a screener, or in a routine manner.

Because of the overlap between behaviors – particularly externalizing ones – associated with ADHD and those associated with autism spectrum disorders, it is helpful to understand the function of the Conners 3-P. Also, since autism spectrum disorders sometimes coexist with intellectual disabilities (ID), it is important to understand how the psychometric properties of the Conners’ tests hold up among children with ID. Deb, Dhaliwal and Roy (2008) undertook this research with the CPRS-R and the CTRS-R and found that parents and teachers differed significantly in how they rated children with ID (whereas significant correlations between their reports would be expected, based on previously published psychometric data). Also, the authors noted that some of the items were not applicable to children with severe or profound ID, and/or who were nonverbal (as are some children with autism spectrum disorders). These findings have implications for using the Conners’ tests to assess children with known autism spectrum disorders and ID.

Clinical Uses

The Conners 3-P is a useful tool when a child is experiencing behavioral difficulty at home or at

See Also

- [Attention Deficit/Hyperactivity Disorder](#)
- [Conners’ Teacher Rating Scale](#)

References and Reading

- Conners, K. C. (2008). *Conners* (3rd ed.). Toronto: Multi-Health Systems.
- Conners, C. K., Parker, J. D. A., Sitarenios, G., & Epstein, J. N. (1998). The revised Conners' Parent Rating Scale (CPRS-R): Factor structure, reliability, and criterion validity. *Journal of Abnormal Child Psychology*, 26(4), 257–268.
- Deb, S. S., Dhaliwal, A. J., & Roy, M. M. (2008). The usefulness of Conners' rating scales-revised in screening for attention deficit hyperactivity disorder in children with intellectual disabilities and borderline intelligence. *Journal of Intellectual Disability Research*, 52(11), 950–965.
- Gallant, S. (2008, February). *Conners 3: Psychometric properties and practical applications*. Paper presented at the annual meeting of the National Association of School Psychologists, New Orleans.
- Gallant, S., Conners, C. K., Rzepa, S., Pitkanen, J., Marocco, M., & Sitarenios, G. (2007, August). *Psychometric properties of the Conners, 3rd edition*. Poster presented at the annual meeting of the American Psychological Association, San Francisco. Retrieved from http://downloads.mhs.com/conners/Psychometric_Properties_Conners_3rd_Edition.pdf
- Gianarris, W. J., Golden, C. J., & Greene, L. (2001). The Conners' parent rating scales: A critical review of the literature. *Clinical Psychology Review*, 21(7), 1061–1093.
- Politi, D. M. (2011). *Conners 3rd edition: Introduction and application*. Retrieved from <http://www.msponline.net/Conference2011/Politi2%20Power%20Point.pdf>
- Sparrow, E. P. (2010). *Essentials of Conners' behavior assessments*. Hoboken: Wiley.

Conners' Teacher Rating Scale

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Description

The Conners' Teacher Rating Scale (CTRS) is a teacher-report measure that assesses children's problem behaviors, particularly symptoms of attention deficit hyperactivity disorder (ADHD) and related disorders (including oppositional defiant disorder and conduct disorder). At the time of publication, the Conners 3-T (2008) is the current

version of the CTRS. Teachers of children with autism spectrum disorders or suspected autism spectrum disorders may be asked to complete the Conners 3-T because of the shared symptoms between ADHD and autism spectrum disorders. The Conners 3-T was developed by C. Keith Conners, Ph.D., who also designed two related measures: the Conners' Parent Rating Scales (CPRS), a parent-report measure, and the Conners' Self-Report Scales (CSRS), a self-report measure for children and adolescents. Because these measures are meant to be used in conjunction, the family of Conners' tests is considered to be a "multi-informant" mode of assessment. This is valuable because it can yield information about children's behaviors in multiple settings. For example, asking a teacher to complete the Conners 3-T and a parent to complete the Conners 3-P can shed light on how a child's behavior may differ between home and school.

Historical Background

The Conners 3-T shares a great deal of history with the Conners 3-P. The roots of these assessments date back to the 1960s, when C. Keith Conners, Ph.D., created behavior rating scales based on his multiple observations of children and adolescents with behaviors consistent with what is now known as ADHD. He performed factor analysis to determine how these behaviors fit together and first published his findings in 1970. One of the earliest aims of Conners' work was to track changes in children's behavior following medication use. In 1973, Conners released a 93-item checklist of behaviors that came to be known as the original Conners' Parent Rating Scale. It was quickly adopted as a diagnostic tool even though it did not have a normative sample of the kind structured for empirical support that is required to establish a new assessment tool today. It was not until 1989 that Conners' sample was formalized and expanded, and that the CTRS, along with the CPRS, was published and shared widely. In 1998, the CTRS-R (for "revised") was released, and in 2008, the third and most recent edition, the Conners 3-T, was

released. The Conners 3-T is currently available in English and Spanish. The similarities and differences between these versions is explored below in the section “[Psychometric Data](#).”

Psychometric Data

The format and response style of the measure have remained quite consistent throughout the revisions of the Conners 3-T. In all three versions, the respondent (the teacher completing the measure) is asked to reflect on his or her student's actions over the past month and to respond to a series of items describing mainly problem behaviors (for example, “leaves seat when he/she should stay seated”). For each item, the respondent is asked to mark 0 (“not true at all/never/seldom”), 1 (“just a little true/occasionally”), 2 (“pretty much true/often/quite a bit”), or 3 (“very much true/very often/very frequent”) to describe the extent to which their student engages in or demonstrates the given behavior.

The Conners 3-T has a long-form version (115 questions), a short-form version (41 of the 110 total questions), an ADHD Index (10 of the 110 total questions), and a Global Index (10 of the 110 questions). Previous versions also offered longer and shorter forms. There are circumstances in which one form would be preferable over another; this largely has to do with the reason for testing. For example, using the long-form version of the Conners 3-T might be preferable to using one of the shorter forms when conducting an initial assessment.

Revisions were made (e.g., transitioning from the CTRS to the CTRS-R, and again from the CTRS-R to the Conners 3-T) in order to strengthen the psychometric properties of the instrument. Most available literature focuses on the psychometric properties of the CTRS-R and the Conners 3-T. The CTRS-R, like the CPRS-R, contains the following subscales: Oppositional (long and short forms), Social Problems (long form only), Cognitive Problems/Inattention (long and short forms), Hyperactivity (long and short forms), DSM-IV Symptom Subscales (long form only), Anxious-Shy (long form only),

ADHD Index (long and short forms), Perfectionism (long form only), and Conners' Global Index (long form only). Unlike the CPRS-R, the CTRS-R does not contain the Psychosomatic subscale. Both raw scores and T scores are generally reported for each subscale; T scores are standardized scores with a mean of 50 and a standard deviation of 10. For example, a child with a T score of 50 on the Oppositional subscale would have about the same level of oppositional behaviors as the average child his or her age in the normative sample. Higher T scores are associated with higher levels of problem behaviors.

Internal consistency coefficients of the CPRS-R (in other words, measures of how well the individual items of the measure “hang” together and form the subscales listed above) for the total sample range from .77 to .96. The test-retest reliability coefficients – a measure of how similarly a child will be rated shortly following an initial assessment with the CPRS-R – range from .47 to .86. As previously discussed, the CPRS-R is designed to be sensitive to changes in behavior, particularly following medication use, and this might explain partially the lower test-retest reliability coefficients.

Some significant changes were made to the CTRS-R to create the Conners 3-T. The publishers of the Conners 3-T point to the large normative sample ($n = 1200$) that reflects the 2000 US Census information on race and ethnicity, gender, and parental education level as particular strength of the measure. The Conners 3-T includes the use of 1-year, instead of 3-year, age bands to compare children's scores to the normative sample (e.g., 4-year-olds are now compared only to other 4-year-olds, instead of to 4-, 5-, and 6-year-olds). Also, the Conners 3-T includes optional combined gender norms for boys and girls – the norms had been strictly separated by gender in the previous versions – and the combined norms can be helpful for understanding behavior within the context of settings, such as the classroom, that are very frequently coed. The Conners 3-T also has a greater focus on differential diagnosis (or, in other words, teasing apart symptoms of ADHD from symptoms of related disorders) and this is reflected in its normative sample. Additionally,

the age range of the Conners 3-T, at 6–18 years old, is slightly narrower than the age range, 3–17 years, of the previous CTRS versions. The age range was extended to 18 years, 11 months to capture adjustment through the end of high school; it was limited to 6 years, 0 months so that early experiences can be assessed more thoroughly with a separate measure, the Conners Early Childhood (EC).

The Conners 3-P contains the following content scales: inattention, hyperactivity/impulsivity, learning problems, executive functioning, aggression, and peer relations. It also contains four symptoms scales – ADHD Inattentive, ADHD Hyperactive-Impulsive, Conduct Disorder, and Oppositional Defiant Disorder – that map onto the diagnostic criteria put forth in the Diagnostic and Statistical Manual of Mental Disorders, fourth edition, text revision (DSM-IV-TR). New to the Conners 3-T include validity scales (which indicate how a teacher is responding to the items and whether or not the information he or she provides is interpretable), screening items for childhood anxiety and depression, critical items (which require immediate follow-up by the researcher or clinician administering the measure), and impairment items (which indicate decreased functioning in certain life areas, like social relationships). The Conners 3-T is also notable for the way in which it maps onto the Individuals with Disabilities Education Act (IDEA); in other words, how a child is rated by his or her teacher on the Conners 3-T might carry implications for the services he or she is eligible to receive in school.

Both test-retest reliability and internal consistency have been found to be very good for the Conners 3-T, and for the overall family of Conners 3 assessments. According to data put forth by the publisher, internal consistency coefficients (in other words, measures of how well the individual items of the measure “hang” together and form the subscales listed above) for the total sample range from .77 to .97. The 2–4-week test-retest reliability coefficients – a measure of how similarly a child will be rated 2 weeks and again 4 weeks following an initial assessment with the Conners 3-T – was also very good (Cronbach's $\alpha = .71$ to .98, with all correlations significant

at the $p < .001$ level). Inter-rater reliability coefficients (a measure of how likely two different respondents, such as a mother and father, or a parent and teacher, are to rate the same child's behavior) are also acceptable to excellent, ranging from .52 to .94. Continuity between the CTRS-R and the Conners 3-T was demonstrated, and tests of factorial, convergent, divergent, and discriminant validity were also performed on the Conners 3-T.

Clinical Uses

The Conners 3-T is a useful tool when a child is experiencing behavioral difficulty at school or at home. The Conners 3-P may indicate whether a child's symptoms are consistent with ADHD or a related disorder. While it includes questions about different symptoms in user-friendly and accessible language, it does not elicit all of the information that would be needed to make a formal diagnosis. It is important to note that a high score on the Conners 3-T alone is not sufficient to diagnose a child; instead, it is only one piece of information that clinicians will consider when making a diagnosis, if one is warranted. If a child has a diagnosis, then the Conners 3-T can be used to track changes in his or her behavior over time; this is especially important if a child receives intervention, medication, educational supports, or other services to address his or her behavioral challenges. Sometimes, the Conners 3-T is used in the absence of any behavioral problems – it can be used as a screener, or in a routine manner.

Frick, Barry, and Kamphaus (2009) note that the Conners 3-T has several strengths that suit it well for school-based assessments. For example, it focuses on ADHD and other disorders involving externalizing behaviors that can interfere with children's school performance. Also, its short versions, with demonstrated validity and reliability, may be more accessible and user-friendly for teachers in busy school environments. However, the Conners 3-T has its drawbacks too, which include minimal assessment of childhood depression and anxiety, which frequently include

internalizing symptoms. Also, the normative sample of the Conners 3-T is racially and ethnically diverse, but not to the same degree as the Conners 3-P. Additionally, there is little independent validation of the Conners 3-T, aside from the data put forth by the instrument authors.

Because of the overlap between behaviors – particularly externalizing ones – associated with ADHD and those associated with autism spectrum disorders, it is helpful to understand the function of the Conners 3-T. Also, since autism spectrum disorders sometimes coexist with intellectual disabilities (ID), it is important to understand how the psychometric properties of the Conners' tests hold up among children with ID. Deb et al. (2008) undertook this research with the CPRS-R and the CTRS-R and found that parents and teachers differed significantly in how they rated children with ID (whereas significant correlations between their reports would be expected, based on previously published psychometric data). Also, the authors noted that some of the items were not applicable to children with severe or profound ID, and/or who were nonverbal (as are some children with autism spectrum disorders). These findings have implications for using the Conners' tests to assess children with known autism spectrum disorders and ID.

See Also

► [Attention Deficit/Hyperactivity Disorder](#)

References and Reading

- Conners, K. C. (2008). *Conners* (3rd ed.). Toronto: Multi-Health Systems.
- Conners, C. K., Sitarenios, G., Parker, J. D., & Epstein, J. N. (1998). Revision and restandardization of the Conners Teacher Rating Scale (CTRS-R): Factor structure, reliability, and criterion validity. *Journal of Abnormal Child Psychology*, 26(4), 279–291.
- Cordes, M., & McLaughlin, T. M. (2004). Attention deficit hyperactivity disorder and rating scales with a brief review of the Conners Teacher Rating Scale (1998). *International Journal of Special Education*, 19(2), 23–34.
- Deb, S., Dhaliwal, A.-J., & Roy, M. (2008). The usefulness of Conners' Rating Scales-Revised in screening for

Attention Deficit Hyperactivity Disorder in children with intellectual disabilities and borderline intelligence. *Journal of Intellectual Disability Research*, 52(11), 950–965.

- Frick, P. J., Barry, C. T., & Kamphaus, R. W. (2009). *Clinical assessment of child and adolescent personality and behavior* (3rd ed.). New York: Springer.
- Gallant, S., Conners, C. K., Rzepa, S., Pitkanen, J., Marocco, M., & Sitarenios, G. (2007, August). *Psychometric properties of the Conners 3rd edition*. Poster presented at the annual meeting of the American Psychological Association, San Francisco. Retrieved from http://downloads.mhs.com/conners/Psychometric_Properties_Conners_3rd_Edition.pdf
- Hale, J. B., How, S. K., Dewitt, M. B., & Coury, D. L. (2001). Discriminant validity of the Conners' scales for ADHD subtypes. *Current Psychology: Developmental, Learning, Personality, Social*, 20(3), 231–249.
- Politi, D. M. (2011). *Conners 3rd edition: Introduction and application*. Retrieved from <http://www.mspsaonline.net/Conference2011/Politi2%20Power%20Point.pdf>

Consent

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Synonyms

[Informed consent](#)

Definition

In General

Consent is a voluntary agreement to participate in medical treatment, procedure, or research. A physician or other health care provider must obtain the consent of the patient or of someone legally authorized to give consent for the patient before initiating such activity.

This requirement is based on the principle that every individual of sound mind has a right to determine what shall be done with his own body and to control the course of his medical treatment.

Standard

The historical standard for legally sufficient disclosure was the customary disclosure practices of physicians in the community. The current standard, however, is a more patient-oriented one. It focuses on what material information about risks a reasonable physician would believe a reasonable patient would want to know to make a decision. It thus remains objective, but with due regard for both the patient's informational needs and physician's situation.

Current Law

General principles of consent are the same in all jurisdictions, though specific details of the doctrine may vary. The right of consent requires several elements, including capacity (the patient is competent to make decisions), information (the patient is informed of the benefits and risks), and voluntariness (the patient is not coerced into giving consent). Consent to medical treatment can be oral or written, express, or implied. In some jurisdictions, statutes specify the form that a patient's consent must take. Consent is generally not required in certain circumstances, including emergencies, therapeutic privilege, when the patient is incompetent, and when the patient waives having to consent.

A physician's failure to obtain consent from a patient prior to medical treatment can serve as a factual predicate to a malpractice action.

ASD Application

Patients with developmental disabilities, such as ASD, may have cognitive, social, and mental impairments that limit their ability to provide legal consent.

Minors generally cannot give consent. Instead, parents or legal guardians must give consent, preferably with the child's assent when feasible.

See Also

► [Informed Consent](#)

References and Reading

Canterbury v. Spence, 464F.2d 772 (D.C. Cir. 1972).

Consequence-Based Interventions

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Definition

Consequence-based interventions are implemented in response to inappropriate behaviors. These interventions are designed to decrease the future likelihood of the inappropriate behavior occurring. Interventions are determined based on the results of a functional behavior assessment. For a consequence-based intervention to be successful, it must be in response to the function of the behavior. Often, antecedent and consequence-based interventions are used in combination to decrease the probability of inappropriate behavior occurring in the future. Some examples of consequence-based interventions are differential reinforcement and its variants, extinction, response cost, and redirection.

See Also

► [Punishment](#)
► [Reinforcement](#)

References and Reading

Anderson, C., & Long, E. (2002). Use of structured descriptive assessment method to identify variables affecting problem behavior. *Journal of Applied Behavior Analysis*, 35, 137–154.
Cooper, J., Heron, T., & Heward, W. (2007). *Applied behavior analysis* (2nd ed.). Hoboken: Pearson Education.
Malott, R., Malott, M., & Trojan, E. (2000). *Elementary principles of behavior* (4th ed.). Upper Saddle River: Prentice-Hall.

Conservatorship

► [Guardianship](#)

Conservatorship (Full Conservatorship and Limited Conservatorship)

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Definition

A conservatorship is a legal relationship in which a court gives one person, a conservator, the duty and power to make decisions about financial and property matters for the benefit and protection of a beneficiary (also referred to as a person subject to conservatorship) (Garner 2014).

Principles of Conservatorship

Conservatorships can be a useful special needs planning tool for individuals with autism spectrum disorder (ASD) and their caregivers (Werner & Chabany 2016). The term “conservator” is generally understood as a guardian, protector, or preserver of another person’s property. State courts have jurisdiction in conservatorship matters to protect individuals who are unable to care for their estate because of a severe impairment or disability (Uniform Law Commission, 2007, 2017). A conservatorship beneficiary’s estate includes all personal assets, real property, and funds. Beneficiaries can be minors or adults.

Each conservatorship must be appointed by a judge. A court order authorizes a conservator to manage and safeguard a beneficiary’s estate. Conservators are legally required to act in a beneficiary’s best interests at all times (Devi 2013). Depending on the scope of a judge’s conservatorship order, a conservator’s scope of authority can be broad and include such matters as deciding an individual’s spending priorities, budgets for activities, and trust savings allocations.

In the United States, conservatorship decision-making laws contain three key principles:

1. Ensuring that a conservatorship is invoked only as a last resort and after considering the availability of support to assist people with financial decision-making.
2. Ensuring a conservatorship is as confined in scope and duration as is reasonably possible.
3. A conservator’s decision-making should always respect the will, preferences, and rights of the individual beneficiary (National Council on Disability 2018).

Conservatorship Terminology

In the past, the terms “ward,” “incompetent person,” and “incapacitated person” were regularly used to describe a person with a conservatorship. Today, there is a growing movement among disability rights advocates to discard these terms as outdated, derogatory, and disrespectful. Modern terminology in conservatorship cases increasingly emphasizes the importance of each individual’s right to self-determination and general autonomy (Page & Hinrichs 2017). When describing a person who receives conservatorship services, the following terms are widely used: beneficiary, conservatee, individual subject to conservatorship, adult who is the subject of a conservatorship proceeding, protected individual, protected person, and principal (Dinerstein 2012).

Conservatorships are largely governed by the probate laws of the individual states. The title of conservator is not used consistently across all state codes, and a conservator’s requirements and functions can differ greatly among state laws. Though a small number of state laws consider the term “conservatorship” a synonym for a “guardianship,” the majority of states do not use the words interchangeably (Kohn et al. 2013). As a general rule, a guardian’s decision-making abilities include decisions about both personal well-being and financial/property interests. In contrast, a conservator’s decisions are limited to only financial/property matters (National Council on Disability 2019).

In most states, there are two types of conservatorships: full and limited (American Bar Association Commission on Law and Aging 2016). A full

conservatorship is designed to care for all of the essential elements of a beneficiary's estate. If an individual's circumstances do not require a full conservatorship, a judge may decide to order a limited conservatorship (also known as a partial conservatorship). In limited conservatorship cases, a conservator has a narrow scope of work in matters related to a beneficiary's estate – these conservators can only make decisions about specific situations regarding the beneficiary's property matters (Wilber et al. 2001). In some states, the title of "custodianship" is used to describe a full conservatorship or various types of conservatorship with limited authority.

Legal Capacity in Conservatorship Cases

Before issuing a court order to appoint a conservator, a judge must first declare that a beneficiary lacks "legal capacity (Parker 2016). Historically, the term legal capacity was also referred to as "legal competency." Individuals do not have legal capacity when they are deemed entirely incapable of managing financial affairs. In these instances, the individual is unable to fully safeguard himself or herself against harm to personal wealth and/or property. A judge will determine that a beneficiary lacks legal capacity based on evidence presented to the court, including medical records and testimony from treating physicians, mental health professionals, caregivers, family members, friends, and the individual under review for the conservatorship appointment (United States General Accounting Office 2004).

For a conservatorship appointment, a beneficiary's legal capacity must be distinguished from his or her mental ability (Dudley & Edmonds 2018). Legal capacity embodies individual legal rights to have standing to bring and defend lawsuits and enter into/execute legally binding contracts. In contrast, mental or cognitive capacity issues in conservatorship matters focus on a person's ability to express personal will and intentions. Depending on the jurisdiction in question, state law may require conservators to take a beneficiary's preferences into account when

making life-changing decisions about finances and property (Kohn & Blumenthal 2014).

Under all state laws, if the circumstances that originally mandated a conservatorship have materially changed, courts are obligated to perform a review to confirm whether the conservatorship is still required or if another type of protective measure is more appropriate.

Starting a Conservatorship

When a judge considers whether a conservatorship appointment is necessary, the court may proceed with the following steps:

1. Hold an evidentiary hearing.
2. Order individuals produce evidence or give testimony (including the proposed beneficiary under review for a conservatorship, health-care treatment providers, family members).
3. Order a medical evaluation or assessment of the proposed beneficiary's abilities and impairments.
4. Order any appropriate investigation of any proposed conservator or other individuals involved in a conservatorship proceeding.
5. Review a certified copy of the transcript or other hearing records of from conservatorship proceedings in other courts.
6. Issue an order authorizing the release of medical, financial, criminal, or other relevant information, including protected health information subject to state and federal privacy laws for records.

Ending a Conservatorship

There are two ways to end a conservatorship: with a court order or when the beneficiary passes away. When the beneficiary dies, his or her conservatorship terminates automatically. If a conservator's assistance is no longer needed because of a significant change in the beneficiary's cognitive and/or physical state, the court may decide to formally terminate the conservatorship. Anyone who is interested in the beneficiary's welfare can

petition a court to end a conservatorship, including the conservator, a family member, friend, or the beneficiary himself or herself. Upon this petition, a judge will decide whether a conservatorship should: (1) continue as is, (2) be modified to limit the conservator's responsibilities, or (3) end (Uekert et al. 2018). Ultimately, a judge will consider what is in the best interest of the beneficiary and only deem a conservatorship inappropriate when there is enough evidence in the court's records to show that a "good cause" exists for termination.

If a conservator wishes to resign from his or her appointment, a judge must file a resignation request with the court. Before a judge approves a conservator's resignation request, the judge will appoint another person to serve as conservator (Van Arsdale & Oakes 2020).

See Also

- [Guardianship](#)
- [Power of Attorney \(Financial and Property Management\)](#)
- [Special Needs Planning \(SNP\)](#)
- [Special Needs Trust \(SNT\)](#)

References and Reading

- American Bar Association Commission on Law and Aging. (2016). Guardianship and supported decision-making: Practical tool. Retrieved from https://www.americanbar.org/groups/law_aging/resources/guardianship_law_practice/practical_tool
- Devi, N. (2013). Supported decision-making and personal autonomy for persons with intellectual disabilities: Article 12 of the UN convention on the rights of persons with disabilities. *The Journal of Law, Medicine & Ethics*, 41(4), 792–806. <https://doi.org/10.1111/jlme.12090>.
- Dinerstein, R. (2012). Implementing legal capacity under article 12 of the UN convention on the rights of persons with disabilities: The difficult road from guardianship to supported decision-making. *Human Rights Brief*, 19(2). Retrieved from <https://ssrn.com/abstract=2040938>. <https://doi.org/10.1016/j.dhjo.2013.03.005>.
- Dudley, K., & Edmonds, N. (2018). Psychology's role in guardianship and conservatorship evaluations. In S. S. Bush & A. L. Heck (Eds.), *Forensic geropsychology: Practice essentials* (pp. 257–279). Washington, DC: American Psychological Association. <https://doi.org/10.1037/0000082-013>.
- Garner, B. (Ed.) (2014). *Conservatorship*. Black's law dictionary. 10th ed. St. Paul: Thomson/West. 370.
- Kohn, N., & Blumenthal, J. (2014). A critical assessment of supported decision-making for persons aging with intellectual disabilities. *Disability and Health Journal*, 7(1), S40–S43. <https://doi.org/10.1016/j.dhjo.2013.03.005>.
- Kohn, N., Blumenthal, J., & Campbell, A. (2013). Supported decision-making: A viable alternative to guardianship. *Penn State Law Review*, 117(4), 1111.
- National Council on Disability. (2018). Beyond guardianship: Toward alternatives that promote greater self-determination. Retrieved from <https://ncd.gov/publications/2018/beyond-guardianship-toward-alternatives>
- National Council on Disability. (2019). Turning rights into reality: How guardianship and alternatives impact the autonomy of people with intellectual and developmental disabilities. Retrieved from <https://ncd.gov/publications/2019/turning-rights-into-reality>
- Page, K., & Hinrichs, K. (2017). Swimming against the tide: A case study on the removal of conservatorship and guardianship. *Clinical Gerontologist*, 40(1), 35–42. <https://doi.org/10.1080/07317115.2016.1177767>.
- Parker, M. (2016). Getting the balance right: Conceptual considerations concerning legal capacity and supported decision-making. *Journal of Bioethical Inquiry*, 13(3), 381–393. <https://doi.org/10.1007/s11673-016-9727-z>.
- Uekert, B. et al. (2018). Data quality undermines accountability in conservatorship cases. National Center for State Courts Brief No. 7. NCJ 252660. Retrieved from <http://www.ncjrs.gov/App/publications/abstract.aspx?ID=274885>
- Uniform Law Commission. (2007). Adult guardianship and Protective Proceedings Jurisdiction Act (UAGPPJA). Retrieved from <https://www.uniformlaws.org/committees/community-home?CommunityKey=0f25ccb8-43ce-4df5-a856-e6585698197a>
- Uniform Law Commission. (2017). Guardianship, Conservatorship and Other Protective Arrangements Act (UGCOPAA). Retrieved from <https://www.uniformlaws.org/committees/community-home?CommunityKey=2eba8654-8871-4905-ad38-aabbd573911c>
- United States General Accounting Office. (2004). Guardianships: Collaboration needed to protect incapacitated elderly people. GAO-04-655. Retrieved from <https://www.gao.gov/products/GAO-04-655>.
- Van Arsdale, B., & Oakes, K. (2020). Guardian and ward. *American Jurisprudence*, 2d, 39, 79–80.
- Werner, S., & Chabany, R. (2016). Guardianship law versus supported decision-making policies: Perceptions of persons with intellectual or psychiatric disabilities and parents. *American Journal of Orthopsychiatry*, 86(5), 486–499. <https://doi.org/10.1037/ort0000125>.
- Wilber, K., et al. (2001). New perspectives on conservatorship: The views of older adult conservatees and their conservators. *Aging, Neuropsychology, and Cognition*, 8(3), 225–240. <https://doi.org/10.1076/anec.8.3.225.831>.

Constipation

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Synonyms

Dyschezia; Fecal impaction; Obstipation

Definition

The definition of constipation varies among individuals. To some, it is hard stools; to others, it is large stools; and to many more, it is infrequent stools. Because the word “constipation” has different meanings for different people, it has been difficult to compile data on normal and abnormal patterns in children (Schuster 1984). Webster's English Dictionary reads “a term used to describe the subjective complaint of passage of abnormally delayed or dry, hardened feces, often accompanied by straining and/or pain” (Webster's ninth new collegiate dictionary 1986). Guidelines of the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition (NASPGHAN) similarly define constipation as “a delay or difficulty in defecation, present for 2 or more weeks and sufficient to cause significant distress to the patient” (Baker et al. 1999). At present, the most widely accepted definitions for childhood functional constipation are the Rome III definitions (Hyman et al. 2006 and Rasquin et al. 2006) (12,13). An evidence-based recommendations from ESPGHAN (European Society of Pediatric Gastroenterology, Hepatology and Nutrition) and NASPGHAN in 2014 defined constipation as follows (Tabbers et al. 2014).

In the absence of organic pathology, two of the following must occur:

For a child with a developmental age < 4 years

1. ≤ 2 defecations per week
2. At least 1 episode of incontinence per week after the acquisition of toileting skills

3. History of excessive stool retention
4. History of painful or hard bowel movements
5. Presence of a large fecal mass in the rectum
6. History of large-diameter stools that may obstruct the toilet

Accompanying symptoms may include irritability, decreased appetite, and/or early satiety, which may disappear immediately following passage of a large stool.

For a child with a developmental age 4 years with insufficient criteria for irritable bowel syndrome

1. ≤ 2 defecations in the toilet per week
2. At least 1 episode of fecal incontinence per week
3. History of retentive posturing or excessive volitional stool retention
4. History of painful or hard bowel movements
5. Presence of a large fecal mass in the rectum
6. History of large-diameter stools that may obstruct the toilet

One can conclude then that normal stool frequency ranges from an average of four per day during the first week of life to two per day at 1 year of age. The normal adult range of three per day to three per week is attained by 4 years of age. These data reflect the average stool frequency in normal infants and children in industrialized countries, not in developing countries, where the normal diet is rich in fiber and normal stool frequency may be different.

Many experts believe that constipation is the delay in defecation for approximately 2 weeks or difficulty in defecation. The causes of constipation are many and may be organic or nonorganic; medications can be a potential cause. Children with ASDs can have sensory processing abnormalities and develop stool-withholding behaviors or constipation related to altered pain responses. Even children with ASDs who have daily bowel movements may have retention of stool that is not evident to parents, teachers, or health-care providers (Buie et al. 2010).

Estimates suggest that ~95% of childhood constipation may be functional, without an

underlying physiologic cause, and many children with ASD present with nonorganic toileting problems that may precipitate or play a role in the development of constipation, including absent or delayed acquisition of bowel training (Whiteley 2004) and higher rates of problem behaviors related to changes in toileting routine. Fecal retention in ASD may also occur secondary to difficulty with sensory stimuli, sensory processing, and motor problems, leading to altered gastrointestinal motility and defecation physiology (Peeters et al. 2013). It is also possible that elevated rates of constipation may be related to the ubiquity of food selectivity in this population, as the dietary patterns often associated with ASD involve high intake of processed food and lack fiber-containing fruits and vegetables, which provide a natural laxative effect and decrease intestinal transit time.

The evaluation of all children who present with constipation should include a thorough medical history and physical examination. Understanding what the family or child means when they use the term “constipation,” the frequency of bowel movements, the consistency and size of stool, and the presence or absence of abdominal pain is important. A history of stool-withholding behavior points more toward functional causes of constipation. For children with ASDs, the physical examination may not identify palpable stool, and a careful rectal examination might not be feasible. Every attempt should be made to examine the rectum, although at times it cannot be accomplished. The rectal examination enables the assessment of stool retention, anal tone, and occult mass, as well as the presence or absence of blood, and helps to reassure the family that the child’s anatomy is normal. A plain radiograph of the abdomen may reveal a rectal fecal mass not palpable on the abdominal examination, but due to conflicting evidence for the accuracy of radiologic diagnosis of constipation, routine radiography is not recommended. Diagnostic clues can help to identify some organic causes of constipation. Hirschsprung’s disease is common in children with ASDs, and a history of delayed passage of stool after birth should raise

the suspicion of aganglionosis. Anatomic abnormalities such as an anterior displacement of the anus, which is more common in girls than boys, can be diagnosed by careful inspection of the anal area. Drugs added to behavior management for constipation are often beneficial. Mineral oil, magnesium hydroxide, lactulose, sorbitol, polyethylene glycol (PEG), or a combination of lubricant (mineral oil) and laxative is recommended for the daily management of constipation in children.

See Also

- ▶ [Gastrointestinal Disorders and Autism](#)
- ▶ [Leaky Gut Syndrome](#)

References and Reading

- Baker, S. S., Liptak, G. S., Colletti, R. B., Croffie, J. M., Di Lorenzo, C., Ector, W., et al. (1999). Constipation in infants and children: Evaluation and treatment. A medical position statement of the North American society for pediatric gastroenterology and nutrition. *Journal of Pediatric Gastroenterology and Nutrition*, 29, 612–626.
- Buie, T., Fuchs, G. J., III, Furuta, G. T., Kooros, K., Levy, J., Lewis, J. D., et al. (2010). Recommendations for evaluation and treatment of common gastrointestinal problems in children with ASDs. *Pediatrics*, 125, S19–S29.
- Hyman, P. E., Milla, P. J., Benninga, M. A., et al. (2006). Childhood functional gastrointestinal disorders: Neonate/toddler. *Gastroenterology*, 130, 1519–1526.
- McElhanon, B. O., McCracken, C., Karpen, S., et al. (2014). Gastrointestinal symptoms in autism spectrum disorder: A meta-analysis. *Pediatrics*, 133, 872–883.
- Peeters, B., Noens, I., Philips, E. M., Kuppens, S., & Benninga, M. A. (2013). Autism spectrum disorders in children with functional defecation disorders. *The Journal of Pediatrics*, 163(3), 873–878.
- Rasquin, A., Di Lorenzo, C., Forbes, D., et al. (2006). Childhood functional gastrointestinal disorders: Child/adolescent. *Gastroenterology*, 130, 1527–1537.
- Schuster, M. M. (1984). Chronic constipation in children: The need for hard data about normal stools. *Journal of Pediatric Gastroenterology and Nutrition*, 3, 336–337.
- Tabbers, M. M., DiLorenzo, C., Berger, M. Y., Faure, C., Langendam, M. W., Nurko, S., et al. (2014). Evaluation and treatment of functional constipation in infants and children: Evidence-based recommendations from

- ESPGHAN and NASPGHAN. *Journal of Pediatric Gastroenterology and Nutrition*, 58, 258–274.
- Webster's ninth new collegiate dictionary. (1986). Springfield: Merriam-Webster, Constipation, p. 281.
- Whiteley, P. (2004). Developmental, behavioural and somatic factors in pervasive developmental disorders: Preliminary analysis. *Child: Care, Health and Development*, 30(1), 5–11.

Consulting Teacher

► Itinerant Teacher

Contactin-Associated Protein 2

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The gene CNTNAP2 encodes the protein *contactin-associated protein-like 2* (recommended UniProt name; also known as Caspr2), a member of the neurexin superfamily, and of a class of genes functioning in the nervous system as cell adhesion molecules and receptors acting at the cell membrane ([EntrezGene](#)). This gene is less frequently referred to as AUTS15, CDFE, CASPR2, PTHSL1, NRXN4, KIAA0868, and DKFZ-p781D1846 ([UniProt](#); [BioGrid](#)).

Structure

CNTNAP2 is located at chromosome 7q35; the genic region of this gene is quite large, spanning 2.3 MB (Nakabayashi and Scherer 2001). In humans, there is a single established isoform (or version of the gene) consisting of 24 exons. The final protein product is 1,331 amino acids. The protein spans the cell membrane one single time ([UniProt](#)). The protein consists, in order, of a signal peptide (a short sequence directing the protein product where to go in the cell), a FA58C domain (“cell surface-attached carbohydrate-

binding domain”), two laminin G domains (common in extracellular proteins), an epidermal growth factor-like domain (also frequently found in extracellular proteins), a fibrinogen C-terminal domain, another laminin G domain, another epidermal growth factor-like domain, another laminin G domain, a helical transmembrane domain (spanning the membrane between the inside and outside of the cell), and a cytoplasmic domain (the only portion of the mature protein that is inside the cell). This transmembrane and cytoplasmic end of the protein also contains a putative band 4.1 homologues’ binding motif (common to neurexins as well as syndecans and glycophorin C intracellular C-termini, all of which are cell surface proteins) ([SMART](#); [UniProt](#)).

Function

CNTNAP2 was first described by Poliak, Gollan, Martinez, et al. (1999) and was shown to be a member of the neurexin superfamily that localized within juxtaparanodal regions of myelinated axons and clustering with potassium channels. The juxtaparanode is the region next to the paranode, which is on either side of the node of Ranvier, an unmyelinated (unsheathed) region of the axon that allows for efficient signal conduction. Homologs of CNTNAP2 are found as far back as insects (*D. melanogaster*, *A. gambiae*) and nematodes (*C. elegans*) ([EntrezGene](#)), further implying important neural function. CNTNAP2 was shown to interact with CNTN2 (TAG-1); in the absence of CNTN2, CNTNAP2 failed to localize at juxtaparanodes and potassium channels did not accumulate normally (Traka et al. 2003). The transcription factor FOXP2, itself implicated in language function ([EntrezGene](#)), was shown by Vernes, Newbury, Abrahams, et al. (2008) to directly regulate CNTNAP2 expression. CNTNAP2 was also shown to have higher expression in circuits involved in higher cortical function, like language (Abrahams et al. 2007). A recent mouse knockout (an animal in which both copies of a gene have been removed) of CNTNAP2 showed dysfunction in neuronal migration (ectopic neurons occurring in the corpus callosum) and reduced numbers of

interneurons (Peñagarikano et al. 2011). One study has shown evidence of CNTNAP2 in rat forebrain synapses (Bakkaloglu et al. 2008), but it has not been definitively characterized as a synaptic molecule.

Pathophysiology

CNTNAP2 has been implicated in several psychiatric disease phenotypes, including Tourette syndrome and obsessive-compulsive disorder (Verkerk et al. 2003), but has received particular attention for its possible association with autism. Initially, linkage evidence suggested a language-linked gene on chromosome 7q35 (Alarcon et al. 2002, 2005). A homozygous mutation in CNTNAP2 was associated with a cortical dysplasia and focal epilepsy phenotype, resulting in seizures, language and social impairments, mental disability, and autistic traits (Strauss et al. 2006). CNTNAP2 was additionally implicated in multiplex autism families by a linkage peak at 7q35 and showed subsequent significant association with a single DNA base change, or single nucleotide polymorphism (SNP) within the gene in an analysis of parent-affected child trios (Arking et al. 2008). A fine-scale analysis of the 15 mb region (7q34-36) encompassing the implicated 7q35 language region in 172 parent-autistic child trios reinforced CNTNAP2 as an autism candidate gene; follow-up analysis in 304 separate parent-autistic child trios showed association of a SNP in CNTNAP2 with age at first spoken word, again underscoring a possible connection to language phenotype as suggested by Vernes et al. (2008). Parallel work in this study showed CNTNAP2 expression very specific to brain circuits established as essential for executive function in humans (Alarcon et al. 2008). Also in 2008, large-scale resequencing of the CNTNAP2 gene in 635 autism cases and 942 unaffected controls showed overrepresentation of rare deleterious (i.e., changing the amino acid in a way that is predicted as harmful) mutations in autistic patients compared with unaffected controls, but not at a statistically significant level. A specific mutation, I869T (the 869th amino acid changed from isoleucine to threonine), however, occurred four times in

three unrelated families and was not seen in any controls, reaching statistical significance. In each case, it was inherited from an ostensibly unaffected parent, suggesting again that idiopathic (nonsyndromic) autism may sometimes involve harmful mutations in multiple neurological genes at once. A 2010 study described two siblings with a deletion spanning CNTNAP2 who showed mental retardation and language delay (Sehested et al. 2010); a separate study described a boy with a complex chromosomal rearrangement (pieces of chromosomes breaking off and rearranging spontaneously) disrupting CNTNAP2 who presented with speech delay and ASD (Poot et al. 2010). More recently, a rare, predicted deleterious CNTNAP2 variant was observed when 20 exomes of patients with autism were sequenced (O’Roak et al. 2011), perhaps acting in concert with a FOXP1 mutation in that patient. Finally, a report of a mouse model with both copies of the mouse homolog of CNTNAP2 knocked out (Peñagarikano et al. 2011) described mice with deficits in the 3 ASD core features, in addition to epileptic seizures (reminiscent of Strauss et al. 2006) and hyperactivity. The evidence in CNTNAP2 to date, as well as the implication of several other neurological genes (e.g., NLGN3, NLGN4 X-linked, SHANK3, NRXN1) involved neuron and synapse structure and development, means that further research on CNTNAP2 will undoubtedly continue and, hopefully, better elucidate its precise role(s) in autism spectrum disorders.

See Also

- [Neuroligins](#)
- [SHANK 3](#)

References and Reading

- Abrahams, B. S., Tontler, D., Perederiy, J. V., Oldham, M. C., Coppola, G., & Geschwind, D. H. (2007). Genome-wide analyses of human perisylvian cerebral cortical patterning. *Proceedings of the National Academy of Sciences of the United States of America*, 104(45), 17849–17854.
- Alarcon, M., Cantor, R. M., Liu, J., et al. (2002). Evidence for a language quantitative trait locus on chromosome

- 7q in multiplex autism families. *American Journal of Human Genetics*, 70(1), 60–71.
- Alarcon, M., Yonan, A. L., Gilliam, T. C., et al. (2005). Quantitative genome scan and ordered-subsets analysis of autism endophenotypes support language QTLs. *Molecular Psychiatry*, 10(8), 747–757.
- Alarcon, M., Abrahams, B. S., Stone, J. L., et al. (2008). Linkage, association, and gene-expression analyses identify CNTNAP2 as an autism-susceptibility gene. *American Journal of Human Genetics*, 82(1), 150–159.
- Arking, D. E., Cutler, D. J., Brune, C. W., et al. (2008). A common genetic variant in the neurexin superfamily member CNTNAP2 increases familial risk of autism. *American Journal of Human Genetics*, 82(1), 160–164.
- Bakkaloglu, B., O’Roak, B. J., Louvi, A., et al. (2008). Molecular cytogenetic analysis and resequencing of contactin associated protein-like 2 in autism spectrum disorders. *American Journal of Human Genetics*, 82(1), 165–173.
- BioGrid. Retrieved from <http://thebiogrid.org/>
- EntrezGene. Retrieved from <http://www.ncbi.nlm.nih.gov/gene>
- Nakabayashi, K., & Scherer, S. W. (2001). The human contactin-associated protein-like 2 gene (CNTNAP2) spans over 2 Mb of DNA at chromosome 7q35. *Genomics*, 73(1), 108–112.
- O’Roak, B. J., Derziotis, P., et al. (2011). Exome sequencing in sporadic autism spectrum disorders identifies severe de novo mutations. *Nature Genetics*, 43(6), 585–589.
- Peñagarikano, O., Abrahams, B. S., Herman, E. I., et al. (2011). Absence of CNTNAP2 leads to epilepsy, neuronal migration abnormalities, and core autism-related deficits. *Cell*, 147(1), 235–246.
- Poliak, S., Gollan, L., Martinez, R., et al. (1999). Caspr2, a new member of the neurexin superfamily, is localized at the juxtaparanodes of myelinated axons and associates with K⁺ channels. *Neuron*, 24(4), 1037–1047.
- Poot, M., Beyer, V., Schwaab, I., et al. (2010). Disruption of CNTNAP2 and additional structural genome changes in a boy with speech delay and autism spectrum disorder. *Neurogenetics*, 11(1), 81–89.
- Sehested, L. T., Miller, R. S., Bache, I., Andersen, N. B., Ullmann, R., Tommerup, N., et al. (2010). Deletion of 7q34-q36.2 in two siblings with mental retardation, language delay, primary amenorrhea, and dysmorphic features. *American Journal of Medical Genetics. Part A*, 152A(12), 3115–3119.
- SMART (Simple Molecular Architecture Research Tool). Retrieved from <http://smart.embl-heidelberg.de/>
- Strauss, K. A., Puffenberger, E. G., Huentelman, M. J., et al. (2006). Recessive symptomatic focal epilepsy and mutant contactin-associated protein-like 2. *The New England Journal of Medicine*, 354(13), 1370–1377.
- Traka, M., Goutebroze, L., Denisenko, N., et al. (2003). Association of tag-1 with caspr2 is essential for the molecular organization of juxtaparanodal regions of myelinated fibers. *The Journal of Cell Biology*, 162(6), 1161–1172.
- UCSC Genome Browser. Retrieved from <http://genome.ucsc.edu/>
- UniProt. Retrieved from <http://www.uniprot.org/>
- Verkerk, A. J., Mathews, C. A., Joosse, M., et al. (2003). CNTNAP2 is disrupted in a family with Gilles de la Tourette syndrome and obsessive compulsive disorder. *Genomics*, 82(1), 1–9.
- Vernes, S. C., Newbury, D. F., Abrahams, B. S., et al. (2008). A functional genetic link between distinct developmental language disorders. *The New England Journal of Medicine*, 359(22), 2337–2345.

Contingencies of Reinforcement

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Definition

Contingencies of reinforcement, in their simplest form, are comprised of antecedents (events that occur immediately before a behavior), responses or behaviors, and consequences (events that occur immediately after a behavior). The term *contingencies* refers to the relationship or interrelationship (Skinner 1969) between these events. *Reinforcement* refers to consequences that increase the probability of the behavior occurring again under similar circumstances. Thus, contingencies of reinforcement describe an antecedent-behavior-consequence link in which the consequence increases the likelihood that a behavior will occur again in the presence of an antecedent. Contingencies of reinforcement are a key component in applied behavior analysis (ABA) approaches to the treatment of autism spectrum disorders (ASD). For example, discrete trial teaching includes the presentation of an antecedent (an instruction, often referred to as a *discriminative stimulus* when it comes to occasion a specific response under specific conditions), the learner’s response, and the presentation of reinforcement. In the case of teaching verbal (or social) behavior with learners with ASD,

written or pictorial rules may be used for effectively initiating a social greeting. The rules serve an antecedent function that may occasion the behavior, in the absence of a history of reinforcement. When the behavior is exposed to naturally occurring contingencies, such as a smile in response to eye contact, a shaping process may occur, leading to behavior shaped (better eye contact during a greeting) and maintained by the contingencies of reinforcement, rather than the rule. The behavior required by the rule must be maintained by consequences (part of the contingency) to be effective – an effective rule is supported by the contingencies of reinforcement.

See Also

- ▶ [Negative Reinforcement](#)
- ▶ [Operant Conditioning](#)
- ▶ [Positive Reinforcement](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). Basic concepts. In J. O. Cooper, T. E. Heron, & W. L. Heward (Eds.), *Applied behavior analysis* (2nd ed., pp. 24–46). Upper Saddle River: Pearson.
- Johnson, J. M. (2014). *Radical behaviorism for ABA practitioners* (pp. 110–113). Cornwall on Hudson: Sloan.
- Skinner, B. F. (1969). *Contingencies of reinforcement: A theoretical analysis*. New York: Meredith.

Contingency Contracting

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Definition

A contingency contract is a positive intervention that specifies the behavioral, social, or academic

expectations to be completed in order to access reinforcement. A contract is typically developed by the practitioner providing treatment in collaboration with the client or stakeholders. A clear definition of the task(s), to be completed by the client(s), is outlined, a criterion is established, and a reinforcer is identified. The contract will also monitor individual or group progress after obtaining the baseline level of performance. The implementation of these contracts can target one or more behaviors that are client or group specific. An example of a contingency contract is a token economy system which specifies the target behavior to increase, tokens to be delivered, and the reinforcer as secondary for exhibiting the target behavior and also specifies a variety of highly preferred reinforcers to earn in exchange for the tokens. Contingency contracts are widely used across a variety of settings including home, community, classroom, and clinical settings.

See Also

- ▶ [Positive Reinforcement](#)
- ▶ [Token Economy](#)

References and Reading

- Koegel, L. K., Singh, A. K., & Koegel, R. L. (2010). Improving motivation for academics in children with autism. *Journal of Autism and Developmental Disorders*, 40(9), 1057–1066.
- Skinner, B. F. (1953). *Science and human behavior*. New York: MacMillan.

Continuous Data Collection

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Synonyms

[Continuous measurement](#); [Continuous recording](#)

Definition

Continuous data collection involves methods in which data are collected on all occurrences of behavior during behavioral observations or programmed learning opportunities. When employed during behavior observations, continuous data collection requires the recording of all instances of the response class(es) of interest. When employed during discrete trial training, continuous data collection requires the recording of learner responses and/or prompt levels for each learning trial. Continuous data collection is considered more comprehensive and precise when compared to discontinuous data collection; however, it is also regarded as less practical when available resources preclude recording each instance of the behavior.

See Also

- ▶ [Direct Observation](#)
- ▶ [Discontinuous Data Collection](#)
- ▶ [Massed Practice](#)
- ▶ [Trial](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis*. Hoboken: Pearson Education, Inc..
- Cummings, A., & Carr, J. (2009). Evaluating progress in behavioral programs for children with autism spectrum disorders via continuous and discontinuous measurement. *Journal of Applied Behavior Analysis*, 42, 57–71.
- Lerman, D., Dittlinger, L., Fentress, G., & Lanagan, T. (2011). A comparison of methods for collecting data on performance during discrete trial teaching. *Behavior Analysis in Practice*, 4, 53–62.
- Najdowski, A., Chilingaryan, V., Bergstrom, R., Granpeesheh, D., Balasanyan, S., Aguilar, B., & Tarbox, J. (2009). Comparison of data-collection methods in a behavioral intervention program for children with pervasive developmental disorders: A replication. *Journal of Applied Behavior Analysis*, 42, 827–832.

Continuous Measurement

- ▶ [Continuous Data Collection](#)

Continuous Recording

- ▶ [Continuous Data Collection](#)

Control

- ▶ [Neurotypical](#)

Controlled Oral Word Association

- ▶ [Verbal Fluency](#)

Controlling Prompt

- ▶ [Hand-Over-Hand Assistance](#)

Conversational Discourse

- ▶ [Discourse Management](#)

Conversational Manner

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Definition

Conversational manner refers to the extent to which an individual is able to avoid ambiguity and make clear, concise, and orderly contributions to conversation. The maxim of manner is one of four conversational maxims (general truths or rules of conduct) that guide cooperative conversation in everyday life, as identified by the twentieth-century

linguistic philosopher, Herbert Paul Grice. The other three conversational maxims include the maxims of quality (providing truthful information), quantity (providing sufficient, but not excessive information), and relation (providing relevant information). Failure to adhere to one of these maxims in conversation may lead to the communication breakdowns, such as those that often occur with individuals with autism spectrum disorders (ASD) during discourse. The following response would be considered a violation of the maxim of manner as it lacks clarity as a choice between options has not been made.

Question: Do you want a pepperoni or plain slice?
 Answer: Maybe I'll have the pepperoni or a plain slice.

Alternatively, purposeful violations of a maxim may create what Grice called "implicature," the use of the violation to convey a message that differs from the literal content of the utterance. For example, a young man who violates the maxim of manner when asked about a meeting location by saying "I've been to every last spot in this town and realize that there is not one cool place to hang out. And you know how it is having friends over when you have strict parents" forces his classmate who asked him, "Do you want to hang out with me?" to consider that while, on the surface, he has not clearly answered her question, the implicature she is to draw is that he means that he does not want to hang out with her and is offering a long and vague response in order to avoid directly saying "no" and embarrassing or hurting her.

The existing literature on language use and conversational skills in individuals with ASD indicates that appropriately modifying conversational manner based on listener and context may present a challenge in this group. Individuals with ASD may present ambiguous or disorganized statements without taking into account the interests or knowledge of their listeners.

See Also

► [Discourse Management](#)

References and Reading

- Fine, J., Bartolucci, G., Szatmari, P., & Ginsberg, G. (1994). Cohesive discourse in pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(3), 315–329.
- Frith, U. (1984). A new look at language and communication in autism. *International Journal of Language & Communication Disorders*, 24(2), 123–150.
- Geller, E. (1998). An investigation of communication breakdowns and repairs in verbal autistic children. *The British Journal of Development Disabilities*, 44(87), 71–85.
- Ghaziuddin, M., & Gerstein, L. (1996). Pedantic speaking style differentiates Asperger syndrome from high-functioning autism. *Journal of Autism and Developmental Disorders*, 26(6), 585–595.
- Grice, H. P. (1975). Logic and conversation. In D. Davidson & G. Harman (Eds.), *The logic of grammar*. Encino: Dickenson Press.
- Paul, R. (1984). Responses to contingent queries in adults with mental retardation and pervasive developmental disorders. *Applied PsychoLinguistics*, 5, 349–357.
- Pellegrini, A. D., Brody, G. H., & Stoneman, Z. (1987). Children's conversational competence with their parents. *Discourse Processes*, 10(1), 93–106.
- Surian, L., Baron-Cohen, S., & van der Ley, H. (1996). Are children with autism deaf to Gricean maxims? *Cognitive Neuropsychiatry*, 1(1), 55–71.
- Volden, J. (2004). Conversational repair in speakers with autism spectrum disorder. *International Journal of Language & Communication Disorders*, 39(2), 171–189.

Convulsion

► [Seizure](#)

Co-occurring ASD

► [Risk of ASD in Individuals with Down Syndrome](#)

Cooing

► [Vocalization](#)

Cooperative Groups

► Group Work and Class Discussions

Copy Number Variation

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Synonyms

CNVs

Definition

Copy number variations (CNVs) are deleted or duplicated segments of the genome, typically defined as including ≥ 1000 base pairs of DNA. Deletions and duplications smaller than this are called “indels.” The presence of CNVs in phenotypically normal individuals was not appreciated until 2004 with landmark papers by Iafrate and Sebat (Iafrate et al. 2004; Sebat et al. 2004). Like single-nucleotide polymorphisms (SNPs), CNVs are part of the normal variation between two individuals and may either be inherited from parents or appear de novo. A recent effort to map of CNVs across the human genome (Zarrei et al. 2015) estimated that between 4.8 and 9.5% of the genome varied due to CNVs in healthy individuals, with the number of CNVs falling between roughly 11,500 and 24,000.

The importance of CNVs for ASD was first noted in 2007 by Sebat (Sebat et al. 2007). This study focused on large de novo CNVs (i.e., not present in either parent). These large de novo CNVs were found to be more common in children with autism compared to children without. In this

initial study, the authors also demonstrated an increased rate of de novo CNVs in simplex autism families compared to multiplex autism families. This finding has been replicated multiple times (e.g., Marshall and Scherer 2012; Iossifov et al. 2014 and Sanders et al. 2015) with estimates of ASD risk contribution from over 200 de novo CNVs ranging from approximately 4% to 8%. Recent evidence suggests that small de novo deletions tend to impart risk through the disruption of one major risk gene (as opposed to large CNVs which contribute risk by disrupting multiple genes in parallel). Excitingly, this insight means that small de novo CNVs can be used to enhance the discovery of ASD risk genes (Sanders et al. 2015). Finally, though the vast majority of genetic risk for ASD comes from inherited mutations (Gaugler et al. 2014), the overall contribution of inherited CNVs (both rare and common) has yet to be definitively characterized. However, evidence to date suggests that *rare* inherited CNVs contribute modest risk only (Sanders et al. 2015).

Since the recognition of CNVs’ importance in ASD, multiple studies have aimed to identify specific regions of DNA in which they contribute risk. The best characterized CNVs include 16p11.2 which has been found in 1% of children with autism compared with $<0.1\%$ in unaffected individuals, as well as 15q12, 15q11.2–13.1, 1q21.1, and 2p16.3. Interestingly, some CNVs seem to carry much greater risk as deletions (2p16.3) or duplications (15q12, 15q11.2–13.1, 1q21.1); however others, notably 16p11.2, demonstrate a more balanced split between both cases.

See Also

- 16p11.2
- 7q11.23 Duplications
- CATCH 22 (Chromosome 22q11 Deletion Syndrome)
- Chromosomal Abnormalities
- Common Disease-Rare Variant Hypothesis
- DNA
- Genetics
- Karyotype

References and Reading

- Gaugler, T., Klei, L., Sanders, S. J., Bodea, C. A., Goldberg, A. P., Lee, A. B., Mahajan, M., et al. (2014). Most genetic risk for autism resides with common variation. *Nature Genetics*, 46(8), 881. <https://doi.org/10.1038/ng.3039>.
- Iafrate, A. J., Feuk, L., Rivera, M. N., Listewnik, M. L., Donahoe, P. K., Qi, Y., Scherer, S. W., et al. (2004). Detection of large-scale variation in the human genome. *Nature Genetics*, 36(9), 949–951. <https://doi.org/10.1038/ng1416>.
- Iossifov, I., O’roak, B. J., Sanders, S. J., Ronemus, M., Krumm, N., Levy, D., et al. (2014). The contribution of de novo coding mutations to autism spectrum disorder. *Nature*, 515(7526), 216. <https://doi.org/10.1038/nature13908>.
- Marshall, C. R., & Scherer, S. W. (2012). Detection and characterization of copy number variation in autism spectrum disorder. *Methods in Molecular Biology*, 838, 115–135.
- Sanders, S. J., He, X., Willsey, A. J., Ercan-Sencicek, A. G., Samocha, K. E., Cicek, A. E., et al. (2015). Insights into autism spectrum disorder genomic architecture and biology from 71 risk loci. *Neuron*, 87(6), 1215–1233. <https://doi.org/10.1016/j.neuron.2015.09.016>.
- Sebat, J., Lakshmi, B., Troge, J., Alexander, J., Young, J., Lundin, P., Månér, S., et al. (2004). Large-scale copy number polymorphism in the human genome. *Science*, 305(5683), 525–528. <https://doi.org/10.1126/science.1098918>.
- Sebat, J., Lakshmi, B., Malhotra, D., Troge, J., Lese-Martin, C., Walsh, T., Yamrom, B., et al. (2007). Strong association of de novo copy number mutations with autism. *Science*, 316(5823), 445–449. <https://doi.org/10.1126/science.1138659>.
- Zarrei, M., MacDonald, J. R., Merico, D., & Scherer, S. W. (2015). A copy number variation map of the human genome. *Nature Reviews Genetics*, 16(3), 172. <https://doi.org/10.1038/nrg3871>.

Co-regulation

► Mutual Regulation

Corneal Reflex

► Eyeblick Reflexes

Cornelia de Lange Syndrome

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Synonyms

[Brachmann-de Lange Syndrome](#); [CdLS](#); [de Lange Syndrome](#)

Cornelia de Lange Syndrome (CdLS) is a rare developmental disorder. It was first described by Dutch pediatrician Cornelia de Lange in 1933. Prevalence estimates of CdLS range from 1 in 10,000 births to 1 in 100,000 births. The syndrome is characterized by distinctive facial features, including thick eyebrows that meet at midline, long eyelashes, low-set ears, an upturned nose, a long, smooth philtrum and thin upper lip. CdLS is also associated with small head circumference, in addition to slowed growth before and after birth. Most affected individuals remain below the 5th percentile on the growth curve throughout their lives, and CdLS-specific growth curves have been created for this reason. CdLS patients also typically present with limb abnormalities, ranging from small hands to the absence of forearms, and hirsutism. Intellectual disability is characteristic of CdLS, although individuals with a milder phenotype of the syndrome may present with IQs in the normal range. Reported IQs of affected individuals range from 30 to 102, with an average IQ of 53. Individuals with CdLS usually have gastroesophageal reflux and can present with a variety of other gastrointestinal problems. The majority of children with CdLS experience sensorineural hearing loss. Seizures have been noted in approximately 25% of affected individuals.

Approximately 60% of CdLS cases are attributable to mutations in the NIPBL gene, located on chromosome 5. An additional 10% of CdLS cases

can be attributed to mutations on one of the following genes: SMC1A, HDAC8, RAD21, and SMC3 (accounting for roughly 5%, 4%, <1%, and 1–2% of cases, respectively). In the remaining 30% of cases, the cause of CdLS is unknown. Ninety-nine percent of cases of CdLS come from de novo mutations. A diagnosis of CdLS is established based on the identification of one of the above variants or based on clinical presentation consistent with the syndrome.

Treatment for CdLS varies based on the symptoms with which each patient presents. Gastrointestinal intervention is often required to treat severe gastroesophageal reflux or intestinal malrotation. If limb abnormalities hinder functioning, surgical intervention can be considered. Additionally, physical, speech, and occupational therapies may be utilized in order to optimize developmental outcomes. For individuals who are nonverbal or minimally verbal, alternative communication methods and devices such as PECS and sign language can be used. When hearing loss and seizures occur, they are addressed using standard treatments.

Many individuals affected by CdLS experience autism-like symptoms and self-injurious behavior. Studies have found ASD prevalence among individuals with CdLS ranging from 43% to 85%. There is some thought that the social and communication deficits associated with CdLS are related to high levels of social anxiety among those affected. To date, the literature has not examined whether traditional treatments for ASD are effective in individuals with comorbid ASD and CdLS.

See Also

- [Gastrointestinal Disorders and Autism](#)
- [Speech Therapy](#)

References and Reading

Deardorff, M. A., Noon, S. E., & Krantz, I. D. (2016). Cornelia de Lange syndrome. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics*, 172, 128.

Moss, J., Howlin, P., Magiati, I., & Oliver, C. (2012). Characteristics of autism spectrum disorder in Cornelia de Lange syndrome. *Journal of Child Psychology and Psychiatry*, 53(8), 883–891.

Nakanishi, M., Deardorff, M. A., Clark, D., Levy, S. E., Krantz, I., & Pipan, M. (2012). Investigation of autistic features among individuals with mild to moderate Cornelia de Lange syndrome. *American Journal of Medical Genetics Part A*, 158(8), 1841–1847.

Corpus Callosum

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Synonyms

[Colossal commissure](#); [Interhemispheric white matter tract](#)

Definition

The corpus callosum (CC) is the largest interhemispheric fiber tract of the human brain, consisting of more than 200 million neural fibers that connect homologous (i.e., similar) and heterotopic (i.e., different) regions across the left and right hemispheres. The CC is typically divided into subregions from anterior to posterior using either a three-region or seven-region system. Table 1 presents the three broad subregions and a commonly used seven region subdivision scheme by Witelson (1989), either of which may be used to refer to subregions of the CC. The Witelson scheme, which is often used in research studies, includes seven specific subdivisions: the rostrum, genu, rostral body, anterior midbody, posterior midbody, isthmus, and splenium.

Corpus Callosum, Table 1 Common subregions or segments of the corpus callosum

Region	Anatomical label	Cortical region(s)
Broad regions		
Anterior	Frontal lobe	Prefrontal cortex
Body	Frontal, parietal lobes	Premotor, supplementary motor, sensory, and posterior parietal cortex
Posterior	Temporal, parietal, occipital lobes	Superior and inferior temporal, posterior parietal, visual and secondary occipital cortex
Witelson regions		
1	Rostrum	Caudal/orbital prefrontal, inferior premotor
2	Genu	Prefrontal
3	Rostral body	Premotor, supplementary motor
4	Anterior midbody	Motor
5	Posterior midbody	Somesthetic, posterior parietal
6	Isthmus	Superior temporal, posterior parietal
7	Splenium	Occipital, inferior temporal



Corpus Callosum, Fig. 1 From anterior to posterior, the Witelson (1989) subdivisions include the rostrum (*black*), genu (*yellow*), rostral body (*pink*), anterior midbody (*aqua*), posterior midbody (*green*), isthmus (*deep blue*), and splenium (*red*)

The CC is topographically organized with specific regions connecting fibers originating from defined brain regions (Fig. 1). For example, fibers originating in prefrontal cortex cross the CC in the genu, while fibers starting from the occipital and inferior temporal regions cross at the level of the splenium (Hofer and Frahm 2006). These regions of the CC contain connecting fibers important for many different cognitive functions including attention, memory, language, and other specific abilities. Developmentally, the CC shows an inverted

U-shaped pattern with early increases in volume and integrity through adolescence and early adulthood, followed by later reductions (Giedd et al. 1999; Hasan et al. 2009). Based on its anatomy and functional roles, the CC has been conceptualized as an index of the development of structural and functional connectivity of the hemispheres including aberrant connectivity in neuropsychiatric disorders such as autism (Vidal et al. 2006).

Relevance to ASD

Many research studies have investigated the size of the CC and its segments in autism (see reviews Frazier and Hardan 2009; Lefebvre et al. 2015). The most consistent finding has been a general reduction in the size of the CC in individuals diagnosed with ASD, especially when CC size is considered in relation to total brain volume. Studies that have investigated the size of the CC subregions in ASD have reported somewhat variable results in terms of the subdivisions that are smaller in comparison to controls; however, it appears that the anterior and posterior subdivisions that carry interhemispheric projections from the prefrontal/premotor and posterior parietal areas may be the most affected. This observation, as well as the role of the CC in connecting many brain networks, has provided impetus for further investigation into abnormalities of the interhemispheric pathways

in ASD, especially regarding the hypothesis of abnormal network connectivity in autism (Belmonte et al. 2004). Additionally, a recent longitudinal study that assessed young infants at high-risk for later diagnosis of ASD (6 months, 12 months, and 24 months) suggested that the developmental trajectory of the CC may be altered in ASD (Wolff et al. 2015). Developmental changes were observed in this study with CC thickness being greater during the early postnatal period before a period of relative “normalization” at 24 months. These alterations might precede the abnormalities reported in older children and adults, including the reduction in CC size. Clearly, continued research into the developmental trajectory of the CC in ASD is warranted.

Furthermore, individuals with complete or partial absence of the corpus callosum as a result of abnormal development (agenesis) may exhibit social and communication deficits that result in a diagnosis of ASD in these individuals (Paul et al. 2007). However, agenesis of the corpus callosum is not a characteristic or common finding in ASD and is part of many other congenital syndromes with a wide range of etiologies, including fetal cytomegalovirus infection. The basis of the clinical overlap between ASD and agenesis of the corpus callosum and vice versa is currently under investigation.

See Also

► Corpus Callosum Abnormalities in Autism

References and Reading

- Belmonte, M. K., Allen, G., Beckel-Mitchener, A., Boulanger, L. M., Carper, R. A., & Webb, S. J. (2004). Autism and abnormal development of brain connectivity. *The Journal of Neuroscience*, 24(42), 9228–9231.
- Frazier, T. W., & Hardan, A. Y. (2009). A meta-analysis of the corpus callosum in autism. *Biological Psychiatry*, 66(10), 935–941.
- Giedd, J. N., Blumenthal, J., Jeffries, N. O., Rajapakse, J. C., Vaituzis, A. C., & Liu, H. (1999). Development of the human corpus callosum during childhood and adolescence: A longitudinal MRI study. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 23(4), 571–588.
- Hasan, K. M., Kamali, A., Iftikhar, A., Kramer, L. A., Papanicolaou, A. C., & Fletcher, J. M. (2009). Diffusion tensor tractography quantification of the human corpus callosum fiber pathways across the lifespan. *Brain Research*, 1249, 91–100. <https://doi.org/10.1016/j.brainres.2008.10.026>.
- Hofer, S., & Frahm, J. (2006). Topography of the human corpus-callosum revisited- comprehensive fiber tractography using diffusion tensor magnetic resonance imaging. *NeuroImage*, 32(3), 898–994.
- Lefebvre, A., Beggiano, A., Bourgeron, T., & Toro, R. (2015). Neuroanatomical diversity of corpus callosum and brain volume in autism: Meta-analysis, analysis of the autism brain imaging data exchange project, and simulation. *Biological Psychiatry*, 78(2), 126–134.
- Paul, L. K., Brown, W. S., Adolphs, R., Tyszka, J. M., Richards, L. J., & Mukherjee, P. (2007). Agenesis of the corpus callosum: Genetic, developmental and functional aspects of connectivity. *Nature Reviews Neuroscience*, 8, 287–299.
- Vidal, C. N., Nicolson, R., DeVito, T. J., Hayashi, K. M., Geaga, J. A., Drost, D. J., et al. (2006). Mapping corpus callosum deficits in autism: An index of aberrant cortical connectivity. *Biological Psychiatry*, 60(3), 218–225.
- Witelson, S. F. (1989). Hand and sex differences in the isthmus and genu of the human corpus callosum. *Brain*, 112, 799–835.
- Wolff, J. J., Gerig, G., Lewis, J. D., Soda, T., Styner, M. A., Vachet, C., et al. (2015). Altered corpus callosum morphology associated with autism over the first 2 years of life. *Brain*, 138(7), 2046–2058.

Corpus Callosum Abnormalities in Autism

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Definition

Corpus callosum (CC) abnormalities in autism spectrum disorder (ASD) generally refer to reductions in the midsagittal size, volume, or integrity of the CC in individuals affected by this disorder.

Historical Background

Over the last few decades, case reports have been published supporting an association between partial or complete agenesis or other abnormalities of the CC and social deficits. While not all individuals with agenesis of the corpus callosum meet full DSM criteria for autism spectrum disorder, they often show behavioral, cognitive, and functional patterns reminiscent of autism, particularly individuals with complete agenesis (Paul et al. 2007).

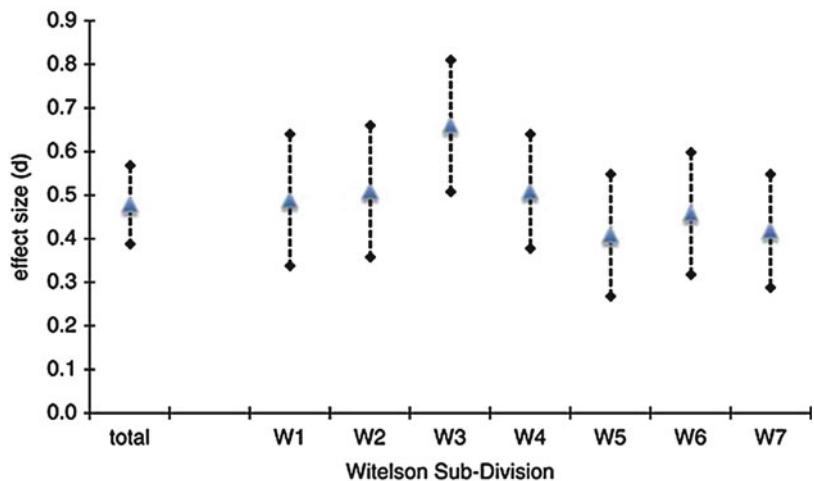
The first careful examination of the CC in autism using magnetic resonance imaging was published by Gaffney et al. (1987). This study did not identify significant differences in midsagittal CC area. However, the lack of significant findings was likely due to a small sample size and the limitation of the early morphometric neuroimaging methodologies. Indeed, following this initial investigation, a series of studies have reported reductions of CC size examining a wide age range of individuals with autism and varying levels of cognitive abilities (Hardan et al. 2000; Just et al. 2007; Manes et al. 1999; Piven et al. 1997; Vidal et al. 2006). For example, studies by Piven et al. (1997) and Hardan et al. (2000) identified reduced midsagittal area of the CC in individuals with ASD. Based on these preliminary investigations, the first meta-analysis of CC size in ASD (Frazier and Hardan 2009) was completed and included ten total studies published between

1987 and 2007, which examined both the total CC and Witelson subdivisions (see Fig. 1 below). Although three of the ten studies conducted over this time period did not find a significant reduction of the CC in autism, when considered together, this pool of studies identified a highly significant reduction of the overall CC in ASD. Additionally, this meta-analysis found that the largest reduction occurred in a subdivision of the CC called Witelson subdivision 3: Rostral body, which is important for motor planning and also includes mirror neurons crucial to representing others' behavior (see Fig. 1 in the Definition of the Corpus Callosum for a view of the whole CC and Witelson subdivisions).

Since the first meta-analysis of CC size in ASD (Frazier and Hardan 2009), 11 additional investigations have been conducted, which largely support a reduced size of the CC in ASD (Alexander et al. 2007; Anderson et al. 2011; Casanova et al. 2011; Frazier et al. 2012; Freitag et al. 2009; Hardan et al. 2009; Hong et al. 2011; Keary et al. 2009; Nordahl et al. 2015; Prigge et al. 2013; Tepest et al. 2010). Two studies conducted during this time failed to find any indication of altered CC size in ASD (Tepest et al. 2010; Hong et al. 2011); however, one study focused only on individuals that received a diagnosis during adulthood (Tepest et al. 2010) and the other reported a significant reduction in density of the CC (Hong et al. 2011), which is also supported by other related studies (Chung et al. 2004; Kumar et al.

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Fig. 1 The magnitude of reductions in the total CC and Witelson subdivisions in individuals with autism. Witelson subdivision 3: rostral body shows the largest reduction



2010; Spencer et al. 2006; Waiter et al. 2005). A new meta-analysis that included the majority of these additional studies was published in 2015 (Lefebvre et al. 2015) and generally supported a reduction in CC size across these investigations, albeit with additional considerations regarding limitations of these previous studies. Following the updated meta-analysis, Lefebvre et al. (2015) also examined CC size in the largest ASD cohort to date, the Autism Brain Imaging Data Exchange (ABIDE), which included 328 patients with high-functioning ASD and 366 controls (7.5–40 years old). Although they failed to find a reduction of CC size in ASD, confounding factors such as participant age, sex, cognitive functioning level, and scanning site may have contributed. Additionally, there appeared to be potential interactions between ASD diagnosis and total brain volume and IQ, suggesting other factors may have contributed to the previously reported group differences or the lack thereof in the ABIDE investigation.

Further supporting CC abnormalities in ASD, diffusion tensor imaging investigations have examined water diffusivity in the CC and generally suggest structural alterations indicative of white matter abnormalities, such as lower fractional anisotropy or higher diffusivity (Alexander et al. 2007; Barnea-Goraly et al. 2004; Hong et al. 2011; Jou et al. 2011a, b; Keller et al. 2007; Kumar et al. 2010; Mengotti et al. 2011; Shukla et al. 2010; Travers et al. 2015) and reduced tract volume or fiber length (Hanaie et al. 2014; Hong et al. 2011; Thomas et al. 2011). These alterations may be due to reductions in the number of axons traveling between hemispheres (hypoplasia) rather than loss of axon integrity or atrophy (Chung et al. 2004); however, a magnetization transfer scan study also reported potentially abnormal myelination of the CC in ASD as well (Gozzi et al. 2012). Overall, it appears that a reduction in CC size and abnormal white matter quality are generally supported in ASD, especially in childhood and adolescence; however, additional research will be necessary to determine the developmental trajectory of CC in individuals with ASD during perinatal development and early infancy.

Current Knowledge

The localization of the exact CC structural abnormalities in ASD has been more thoroughly examined recently. While most studies reported reductions in the total size of the CC, some investigations have suggested that this reduction may be most pronounced in the anterior and posterior subdivisions (Chung et al. 2004; Frazier et al. 2012; Hardan et al. 2009; Keary et al. 2009; Nordahl et al. 2015; Prigge et al. 2013), which are also supported by diffusion tensor imaging studies (Hanaie et al. 2014; Hong et al. 2011; Jou et al. 2011a, b; Thomas et al. 2011; Travers et al. 2015). These findings suggest corresponding structural alterations in frontal, temporal, and parietal regions, which are consistently reported in the literature, see review (Amaral et al. 2008). This pattern is suggestive of early life abnormalities of the CC in ASD. Interestingly, investigations of the CC in young infants that are later diagnosed with ASD suggest potentially altered developmental trajectories of the CC. For instance, some studies report higher fractional anisotropy and increased thickness of the CC in ASD during early infancy (Weinstein et al. 2010; Wolff et al. 2012, 2015) followed by a potential “normalization” by 24 months of age, which seems to precede the aforementioned reduction in CC size during childhood (2–4 years old; Boger-Megiddo et al. 2006; Nordahl et al. 2015) and into adolescence and adulthood (Hardan et al. 2000; Just et al. 2007). This indicates that early CC abnormalities are present in infants that are later diagnosed with autism but the development of hypoplasia of the CC over time corresponds with the presentation of many autism symptoms (Lord et al. 2008; Piven et al. 1996; Seltzer et al. 2003). This potential early overgrowth followed by a relative reduction in size is reminiscent of the global developmental differences in brain size that have been previously reported in ASD (Courchesne et al. 2001), perhaps on an accelerated time course.

Additionally, updated studies and reviews of small case series or small group studies of individuals with agenesis of the CC without intellectual disability have provided further support for a

relationship between the CC and ASD. These observations suggest not only symptom and behavioral patterns similar to what are seen in individuals with ASD but also alterations in thinking abilities consistent with those seen in this disorder. For example, individuals with agenesis of the CC often show cognitive deficits with problems in abstract reasoning, deciphering nonliteral language (metaphors, idioms, sarcasm, humor), and generalization (Brown and Sainsbury 2000; David et al. 1993; Fischer et al. 1992; Solursh et al. 1965; Williams et al. 2006), similar to what has been observed in individuals with ASD.

Future Directions

Progress has been made to date in understanding the pathophysiology of the CC in ASD, but additional studies examining this structure are needed to identify the underpinnings of CC abnormalities and determine how these abnormalities relate to the core features. Longitudinal studies of young children as they grow may be a particularly powerful way to uncover the pattern of development of the CC and relate these changes over time to autism symptomatology. One such investigation found a persistent reduction in CC volume in children and adolescents with ASD over a 2-year period, except for the rostral body, which exhibited relative “normalization” compared to controls (Frazier et al. 2012). Additionally, future studies should apply multimodal imaging techniques over different developmental periods to provide a comprehensive picture of how these abnormalities alter brain function. Finally, relating imaging findings to genetic variation is an important but largely unexplored avenue that will be fruitful for understanding the molecular changes contributing to CC abnormalities and developing new therapeutics that might improve CC abnormalities in individuals with autism.

See Also

- [Agenesis of Corpus Callosum](#)
- [Corpus Callosum](#)

References and Reading

- Alexander, A. L., Lee, J. E., Lazar, M., Boudos, R., DuBray, M. B., & Oakes, T. R. (2007). Diffusion tensor imaging of the corpus callosum in autism. *NeuroImage*, 34, 61–73.
- Amaral, D. G., Schumann, C. M., & Nordahl, C. W. (2008). Neuroanatomy of autism. *Trends in Neuroscience*, 31(3), 137–145.
- Anderson, J. S., Druzgal, T. J., Froehlich, A., DuBray, M. B., Lange, N., Alexander, A. L., . . . Lainhart, J. E. (2011). Decreased interhemispheric functional connectivity in autism. *Cerebral Cortex*, 21(5), 1134–1146.
- Barnea-Goraly, N., Kwon, H., Menon, V., Eliez, S., Lotspeich, L., & Reiss, A. L. (2004). White matter structure in autism: Preliminary evidence from diffusion tensor imaging. *Biological Psychiatry*, 55(3), 323–326. S000632230301151X [pii].
- Boger-Megiddo, I., Shaw, D. W., Friedman, S. D., Sparks, B. F., Artru, A. A., & Giedd, J. N. (2006). Corpus callosum morphometrics in young children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 36, 733–739.
- Brown, L. N., & Sainsbury, R. S. (2000). Hemispheric equivalence and age-related differences in judgments of simultaneity to somatosensory stimuli. *Journal of Clinical and Experimental Neuropsychology*, 22, 587–598.
- Casanova, M. F., El-Baz, Y., Elnakib, A., Switala, A. E., Williams, E. L., Williams, D. L., . . . Conturo, T. E. (2011). Quantitative analysis of the shape of the corpus callosum in patients with autism and comparison individuals. *Autism*, 15(2), 223–238.
- Chung, M. K., Dalton, K. M., Alexander, A. L., & Davidson, R. J. (2004). Less white matter concentration in autism: 2D voxel-based morphometry. *NeuroImage*, 23, 242–251.
- Courchesne, E., Karns, C. M., Davis, H. R., Ziccardi, R., Carper, R. A., Tigue, Z. D., . . . Courchesne, R. Y. (2001). Unusual brain growth patterns in early life in patients with autistic disorder: An MRI study. *Neurology*, 57(2), 245–254.
- David, A. S., Wacharasindhu, A., & Lishman, W. A. (1993). Severe psychiatric disturbance and abnormalities of the corpus callosum: Review and case series. *Journal of Neurology, Neurosurgery and Psychiatry*, 56(1), 85–93.
- Fischer, M., Ryan, S. B., & Dobyns, W. B. (1992). Mechanisms of interhemispheric transfer and patterns of cognitive function in acallosal patients of normal intelligence. *Archives of Neurology*, 49(3), 271–277.
- Frazier, T. W., & Hardan, A. Y. (2009). A meta-analysis of the corpus callosum in autism. *Biological Psychiatry*, 66(10), 9350941.
- Frazier, T. W., Keshavan, M. S., Minshew, N. J., & Hardan, A. Y. (2012). A two-year longitudinal MRI study of the corpus callosum in autism. *Journal of Autism and Developmental Disorders*, 42(11), 2312–2322.

- Freitag, C. M., Luders, E., Hulst, H. E., Narr, K. L., Thompson, P. M., & Toga, A. W. (2009). Total brain volume and corpus callosum size in medication-naïve adolescents and young adults with autism spectrum disorder. *Biological Psychiatry*, 66(4), 316–319. <https://doi.org/10.1016/j.biopsych.2009.03.011>. S0006-3223(09)00360-6 [pii].
- Gaffney, G. R., Kuperman, S., Tsai, L. Y., Minchin, S., & Hassanein, K. M. (1987). Midsagittal magnetic resonance imaging of autism. *British Journal of Psychiatry*, 151, 831–833.
- Gozzi, M., Nielson, D. M., Lenroot, R. K., Ostuni, J. L., Luckenbaugh, D. A., Thurm, A. E., ... Swedo, S. E. (2012). A magnetization transfer imaging study of the corpus callosum myelination in young children with autism. *Biological Psychiatry*, 72(3), 215–220.
- Hanaie, R., Mohri, I., Kagitani-Shimono, K., Tachibana, M., Matsuzaki, J., Watanabe, Y., ... Taniike, M. (2014). Abnormal corpus callosum connectivity, socio-communicative deficits, and motor deficits in children with autism spectrum disorder: A diffusion tensor imaging study. *Journal of Autism and Developmental Disorders*, 44(9), 2209–2220.
- Hardan, A. Y., Minshew, N. J., & Keshavan, M. S. (2000). Corpus callosum size in autism. *Neurology*, 55(7), 1033–1036.
- Hardan, A. Y., Pabalan, M., Gupta, N., Bansal, R., Melhem, N. M., & Fedorov, S. (2009). Corpus callosum volume in children with autism. *Psychiatry Research*, 174(1), 57–61. <https://doi.org/10.1016/j.psychres.2009.03.005>. S0925-4927(09)00086-9 [pii].
- Hong, S., Ke, X., Tang, T., Hang, Y., Kangkang, C., Huang, H., ... Liu, Y. (2011). Detecting abnormalities of the corpus callosum in autism using magnetic resonance imaging and diffusion tensor tractography. *Psychiatry Research: Neuroimaging*, 3(3), 333–339.
- Jou, R. J., Jackowski, A. P., Papademetris, X., Rajeevan, N., Staib, L. H., & Volkmar, F. R. (2011a). Diffusion tensor imaging in autism spectrum disorders: Preliminary evidence of abnormal neural connectivity. *Australian and New Zealand Journal of Psychiatry*, 45(2), 153–162.
- Jou, R. J., Mateljevic, N., Kaiser, M. D., Sugrue, D. R., Volkmar, F. R., & Pelphrey, K. A. (2011b). Structural neural phenotype of autism: Preliminary evidence from a diffusion tensor imaging study using tract-based spatial statistics. *American Journal of Neurology*, 32, 1607–1613.
- Just, M. A., Cherkassky, V. L., Keller, T. A., Kana, R. K., & Minshew, N. J. (2007). Functional and anatomical cortical underconnectivity in autism: Evidence from an fMRI study of an executive function task and corpus callosum morphometry. *Cerebral Cortex*, 17(4), 951–961. <https://doi.org/10.1093/cercor/bhl006>. bhl006 [pii].
- Keary, C. J., Minshew, N. J., Bansal, R., Goradia, D., Fedorov, S., & Keshavan, M. S. (2009). Corpus callosum volume and neurocognition in autism. *Journal of Autism and Developmental Disorders*, 39(6), 834–841. <https://doi.org/10.1007/s10803-009-0689-4>.
- Keller, T. A., Kana, R. K., & Just, M. A. (2007). A developmental study of the structural integrity of white matter in autism. *Neuroreport*, 18(1), 23–27. <https://doi.org/10.1097/01.wnr.0000239965.21685.99>. 00001756-200701080-00005 [pii].
- Kumar, A., Sundaram, S. K., Sivaswamy, L., Behen, M. E., Makki, M. I., Ager, J., ... Chugani, D. C. (2010). Alterations in frontal lobe tracts and corpus callosum in young children with autism spectrum disorder. *Cerebral Cortex*, 20(9), 2103–2113. <https://doi.org/10.1093/cercor/bhp278>.
- Lefebvre, A., Beggato, A., Bourgeron, T., & Toro, R. (2015). Neuroanatomical diversity of corpus callosum and brain volume in autism: Meta-analysis, analysis of the autism brain imaging data exchange project, and simulation. *Biological Psychiatry*, 78(2), 126–134.
- Lord, C., Risi, S., DiLavore, P. S., Shulman, C., Thurm, A., & Pickles, A. (2008). Autism from 2 to 9 years of age. *Archives of General Psychiatry*, 63, 694–701.
- Manes, F., Piven, J., Vrancic, D., Nanclares, V., Plebst, C., & Starkstein, S. E. (1999). An MRI study of the corpus callosum and cerebellum in mentally retarded autistic individuals. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 11, 470–474.
- Mengotti, P., D'Agostini, S., Terlevic, R., De Colle, C., Biasizzo, E., Londero, D., ... Brambilla, P. (2011). Altered white matter integrity and development in children with autism: A combined voxel-based morphometry and diffusion imaging study. *Brain Research Bulletin*, 84(2), 189–195.
- Nordahl, C. W., Iosif, A.-M., Young, G. S., Perry, L. M., Dougherty, R., Lee, A., ... Amaral, D. G. (2015). Sex differences in the corpus callosum in preschool-aged children with autism spectrum disorder. *Molecular Autism*, 6, 26.
- Paul, L. K., Brown, W. S., Adolphs, R., Tyszka, J. M., Richards, L. J., & Mukherjee, P. (2007). Agenesis of the corpus callosum: Genetic, developmental and functional aspects of connectivity. *Nature Reviews Neuroscience*, 8, 287–299.
- Piven, J., Harper, J., Palmer, P., & Arndt, S. (1996). Course of behavioral change in autism: A retrospective study of high-IQ adolescents and adults. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35(4), 523–529.
- Piven, J., Bailey, J., Ranson, B. J., & Arndt, S. (1997). An MRI study of the corpus callosum in autism. *American Journal of Psychiatry*, 154(8), 1051–1056.
- Prigge, M. B. D., Lange, N., Bigler, E. D., Merkley, T. L., Neeley, S., Abildskov, T. J., ... Cooperrider, J. R. (2013). Corpus callosum area in children and adults with autism. *Research in Autism Spectrum Disorders*, 7(2), 221–234.
- Seltzer, M. M., Krauss, M. W., Shattuck, P. T., Orsmond, G., Swe, A., & Lord, C. (2003). The symptoms of autism spectrum disorders in adolescence and adulthood. *Journal of Autism and Developmental Disorders*, 33(6), 565–581.

- Shukla, D. K., Keehn, B., & Müller, R.-A. (2010). Tract-specific analyses of diffusion tensor imaging show widespread white matter compromise in autism spectrum disorder. *The Journal of Child Psychology and Psychiatry*, 52(3), 286–295.
- Solursh, L. P., Margulies, A. I., Ashem, B., & Stasiak, E. A. (1965). The relationships of agenesis of the corpus callosum to perception and learning. *Journal of Nervous and Mental Disease*, 141(2), 180–189.
- Spencer, M. D., Moorhead, W. J., Lymer, K. S., Job, D. E., Muir, W. J., & Hoare, P. (2006). Structural correlates of intellectual impairment and autistic features in adolescents. *NeuroImage*, 33, 1136–1144.
- Tepest, R., Jacobi, E., Gawronski, A., Krug, B., Möller-Hartmann, W., Lehnhardt, F. G., & Vogeley, K. (2010). Corpus callosum size in adults with high-functioning autism and the relevance to gender. *Psychiatry Research: Neuroimaging*, 183(1), 38–43.
- Thomas, C., Humphreys, K., Jung, K.-J., Minshew, N., & Behrmann, M. (2011). The anatomy of the callosal and visual-association pathways in high-functioning autism: A DTI tractography study. *Cortex*, 47(7), 863–873.
- Travers, B. G., Tromp, D. P. M., Adluru, N., Lange, N., Destiche, D., Ennis, C., ... Alexander, A. L. (2015). Atypical development of white matter microstructure of the corpus callosum in males with autism: A longitudinal investigation. *Molecular Autism*, 6, 15.
- Vidal, C. N., Nicolson, R., DeVito, T. J., Hayashi, K. M., Geaga, J. A., & Drost, D. J. (2006). Mapping corpus callosum deficits in autism: An index of aberrant cortical connectivity. *Biological Psychiatry*, 60, 218–225.
- Waiter, G. D., Williams, J. H., Murray, A. D., Gilchrist, A., Perrett, D. I., & Whiten, A. (2005). Structural white matter deficits in high-functioning individuals with autistic spectrum disorder: A voxel-based investigation. *NeuroImage*, 24(2), 455–461. <https://doi.org/10.1016/j.neuroimage.2004.08.049>. S1053-8119(04)00512-9 [pii].
- Weinstein, M., Ben-Sira, L., Levy, Y., Zachor, D. A., Itzhak, E. B., Artzi, M., ... Bashat, D. B. (2010). Abnormal white matter integrity in young children with autism. *Human Brain Mapping*, 32(4), 534–543.
- Williams, D. L., Goldstein, G., & Minshew, N. J. (2006). Neuropsychologic functioning in children with autism: Further evidence for disordered complex information-processing. *Child Neuropsychology*, 12 (4–5), 279–298. <https://doi.org/10.1080/09297040600681190>. W6275407Q443855Q [pii].
- Wolff, J. J., Gu, H., Gerig, G., Elison, J. T., Styner, M., Gouttard, S., ... the IBIS Network (2012). Differences in white matter fiber tract development present from 6 to 24 months in infants with autism. *The American Journal of Psychiatry*, 169(6), 589–600.
- Wolff, J. J., Gerig, G., Lewis, J. D., Soda, T., Styner, M. A., Vachet, C., ... the IBIS Network (2015). Altered corpus callosum morphology associated with autism over the first 2 years of life. *Brain*, 138(7), 2046–2058.

Correlation

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Introduction

The important concept of correlation will cover a number of important areas: defining features, types of correlations, a brief historicity of the concept, and multiple ways to look at the correlation coefficient.

Definition

Correlation refers to a statistical procedure that assesses the association between two variables that we will refer to as X and Y . Correlation coefficients, symbolized as r , can vary between -1 , 0 , and $+1$. Positive correlations occur when Y increases as X increases and negative correlations occur when X increases as Y decreases.

When there is no association between the X and Y variable, the size of r tends in the direction of a 0 value. Finally, correlation rests upon the assumption that there is no so-called causative relationship between the X and Y study variables.

Brief Historicity of the Correlation Coefficient r

As noted by Rodgers and Nicewander (1988), the concept of correlation was introduced, in 1885, by Sir Francis Galton, who developed “the theory of bivariate correlation” as well as the related term “regression.” The concept of r , as we know it today, was introduced a decade later by Karl Pearson (1896). Again, from a historical perspective, the Rodgers and Nicewander publication was written in celebration of the 100th Anniversary of Galton’s critical contribution to the development of the correlation coefficient.

It is important to note also the more far-reaching significance of r when one realizes that “Factor analysis, behavioral genetics models, structural equation models (e.g., LISREL), and other related methodologies use the correlation coefficient as the basic unit of data” (Rodgers and Nicewander 1988, p. 61). In fact, as Henrysson (1971) reminds us, three other correlation coefficients can be accurately defined as special instances of Pearson’s r coefficient: Spearman’s rho and the point-biserial correlation for ordinal data and the phi coefficient for nominal-dichotomous data.

An Application of r

Let us assume that X refers to IQ level and Y to Vineland overall adaptive behavior levels. Assume further that the study group is a random representative sample of typically developing 10-year-olds. Based upon many Vineland standardization samples, we know that the correlation will be of the order of about 0–0.20 between IQ and the adaptive behavior composite, and this is statistically significant at the 5% level of statistical significance. What do we make of the correlation of 0.20 between IQ and overall adaptive behavior? Once again, the all-important level of clinical significance needs to be addressed, this time in the form of criteria developed by Cohen (1988), as expanded by Cicchetti (2008):

Range of correlation, clinical significance:

- <0.10 Trivial
- 0.10–0.29 Small
- 0.30–0.49 Medium
- 0.50–0.69 Large
- 0.70–1.00 Very large

By these clinical criteria, the correlation of 0.20 between IQ level and overall adaptive behavior level is considered small. It should be noted that similar results occur for the Vineland Domains, Daily Living Skills, Socialization, and Motor Skills.

As expected on the basis of the content of the items, the correlation between IQ and the

Communication Domain is somewhat higher, in one study reaching an r value of 0.36 (Sparrow et al. 2005). By the same set of clinical criteria, this represents a medium level of clinical significance.

In the next section, I shall discuss a multitude of different ways to understand the meaning and rather far-reaching implications of the coefficient r . Thirteen were described by Rodgers and Nicewander (1988); a 14th was added by Rovine and von Eye (1997); and a very general purpose r related index was developed and introduced by Rosenthal and Rubin in 1982. Each of these will be discussed in turn:

Thirteen ways to interpret r (derived from Rodgers and Nicewander (1988)):

1. r as Pearson (1896) defined it, or as it is typically applied, based upon raw score and average or mean values
2. r as a ratio of standard deviations
3. r as the standardized slope of the regression line
4. r as the geometric average of the two regression slopes
5. r as the proportion of variability accounted for
6. r as the average cross product of standardized variables
7. r in relation to the angle between two standardized regression lines
8. r in relation to the angle between the two variable vectors
9. r as a rescaled variance of the difference between standardized scores
10. r as estimated from the balloon rule: note that the “balloon” is formed by drawing an ellipse around the scatterplot of the individual X and Y values
11. r as a more formal representation of the balloon rule
12. r as related to test statistics from designed experiments
13. r as the ratio of two means
14. r as the proportion of matches between standardized X and Y values
15. r as its mathematical relationship to the d statistic, as given in Cohen (1988, p. 23), by the following formula: $r = d/(d^2 + 4)^{1/2}$

A fundamental biostatistical research question pertains to the extent to which some future researchers in the field of autism spectrum disorders will find some of these imaginative interpretations of the correlation coefficient useful in providing further clinical insights into the vagaries and vicissitudes of ASD disorders.

In the next section, I shall present some novel interpretations of the meaning of the correlation coefficient r , as presented by Rosenthal and Rubin (1982).

The Binomial Effect Size Display (BESD)

A standard way of interpreting the clinical importance of the size of a correlation coefficient, r , is to simply square its value. This informs us about how much of the explained variance in the Y variable can be attributed to the variance in the X variable. The result is expressed as a percentage score that will vary between 0% (a 0 correlation) and 100% (a perfect correlation, of either +1.00 or -1.00). Thus, a correlation of size 0.32 would be viewed as very low or somewhat trivial, since it accounts for 0.32 squared, or only 10% of the explained variance in the Y variable that can be predicted by the X variable. This would leave as unaccounted variance a whopping 90%! And, in fact, this is the reported success rate of psychotherapy, as reported by Randolph and Edmondson (2000).

While the traditional way of interpreting the psychotherapy outcome study seems rather wimpy, indeed, the work of Rosenthal and Rubin (1982) suggests otherwise. Using what they referred to as a binomial effect size display (BESD), the authors cast a given correlation between the X and Y variables into a 2×2 contingency table, and the approach is applicable whether the original data were derived from categorical or continuous scales of measurement.

Applying the BESD to the Success of Psychotherapy Intervention

The reported r of 0.32, representing the success of psychotherapy intervention, is now considered in

the context of a Treatment Group (the one that receives psychotherapy) and a Control Group (the one that does not receive psychotherapy).

The cells in the 2×2 BESD contingency table are expressed as 4%, each starting and ending at 50%. If the r is 0, then the percentages remain unaltered and indicate 0 success for the psychotherapy intervention. This means that 50% of the subjects in the Treatment Group show positive results and 50% do not. This is no different than the results for the Control Group and makes perfect clinical sense in terms of an r of 0.00 between X , reflecting the type of treatment (Therapy or Not) and Y , reflecting the outcome of the intervention (Success or Failure).

Now, in our application, the actual $r = 0.32$. The BESD will inform how much better than a 0.00 r is one of 0.32.

Using the $r = 0.32$ as a measure of Effect Size (e.g., Cohen 1988; Rosnow et al. 2000), the r is divided in 2, with this value of 0.16 both subtracted and added to the 50% value in each of the two groups. This then represents the amount of improvement provided by the treatment intervention. In our example, this results in percentages of (50% - 16% = 34%) and (50% + 16% = 66%). The result is now interpreted to mean that the r between treatment and success rate of 0.32 indicates that psychotherapy intervention has resulted in a 34–64% increase in success rate or 14% over the 50% one would have expected if the therapy had no effect at all. As noted correctly and initially by Rosenthal and Rubin (1982), even though the r of 0.32 only explains 10% of the variance in success rate, when interpreted in a BESD context, the result can hardly be interpreted as of little clinical value. In summarizing this section of the report, I present, in Table 1, the results of comparing the meaning and interpretation of the various values of a correlation coefficient, r .

The table is constructed to show what happens to the values of r squared, the success of a Treatment intervention, and the level of clinical importance of the research result, as the correlation between the X and Y variable increases from 0 to a perfect correlation of 1.00.

Correlation, Table 1 Comparing and contrasting ways to interpret the meaning of the Pearson correlation coefficient, r

Value of r	r squared	BESD % success	Clinical significance of r
<0.10	<0.01	<55	Trivial
0.10	0.01	55	Small
0.20	0.04	60	Small
0.30	0.09	65	Medium
0.40	0.16	70	Medium
0.50	0.25	75	Large
0.60	0.36	80	Large
0.70	0.49	85	Very large
0.80	0.64	90	Very large
0.90	0.81	95	Very large
1.00	1.00	100	Perfect r

While the levels of clinical significance presented here for the correlation coefficient, r , will be applicable in a multitude of clinical research applications, there are situations in which this is not the case. When this occurs, a statistic other than r will be more appropriate. A classic example has been the binary relationship between whether one takes a daily aspirin or does not and whether one has a heart attack or not. The r between the two binary variables is a mere 0.033. Yet, when the odds ratio is, instead, applied to the data, it produces a value of 1.82, meaning that those not on aspirin had almost twice the odds of experiencing a myocardial infarct than those taking aspirin (Kelley and Preacher 2012; Steering Committee of the Physicians’ Health Study Research Group 1988).

Concluding Comments

This report covers the correlation coefficient for determining the degree of linear relationship between a pair of variables designated as X and Y . It did not cover a number of related issues, not because they are not of research importance in a larger sense but, rather, because studies that focus upon curvilinear relationships among ASD-relevant variables are notable for their absence in the field. Another way of stating this phenomenon is that the relationships we typically study in ASD

research are of a linear nature or quality. For similar reasons, this section on correlation has not focused on multiple correlation.

See Also

► [Statistical Approaches to Subtyping](#)

References and Reading

Cicchetti, D. V. (2008). From Bayes to the just noticeable difference to effect sizes: A note to understanding the clinical and statistical significance of oenologic research findings. *Journal of Wine Research*, 3, 185–193.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale: Erlbaum Associates.

Galton, F. (1985). Regression towards mediocrity in hereditary status. *Journal of the Anthropological Institute*, 15, 246–263.

Henrysson, S. (1971). Gathering, analyzing, and using data on test items. In R. L. Thorndike (Ed.), *Educational measurement* (pp. 130–159). Washington, DC: American Council on Education.

Kelley, K., & Preacher, J. J. (2012). On effect size. *Psychological Methods*, 17, 137–152.

Pearson, K. (1896). Mathematical contributions to the theory of evolution. III. Regression, heredity and panmixia. *Philosophical Transactions of the Royal Society of London*, 187, 253–318.

Randolph, J., & Edmondson, S. (2000). Using the binomial effect size display (BESD) to present the magnitude of effect sizes to the evaluation audience. *Practical Assessment Research and Evaluation*, 10, 1–7.

Rodgers, J. L., & Nicewander, W. A. (1988). Thirteen ways to look at the correlation coefficient. *The American Statistician*, 42, 59–66.

Rosenthal, R., & Rubin, D. B. (1982). A simple general purpose display of magnitude of experimental effect. *Journal of Educational Psychology*, 74, 166–169.

Rosnow, R. L., Rosenthal, R., & Rubin, D. B. (2000). Contrasts and correlations in effect-size estimation. *American Psychological Society*, 11, 446–453.

Rovine, M., & von Eye, A. (1997). A 14th way to look at the correlation coefficient: Correlation as the proportion of matches. *The American Statistician*, 51, 42–46.

Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland II: A revision of the Vineland adaptive behavior scales: I. Survey form*. Circle Pines: American Guidance Service.

Steering Committee of the Physicians’ Health Study Research Group. (1988). *New England Journal of Medicine*, 318, 262–264.

Correlational

- [Qualitative Versus Quantitative Approaches](#)

Cortex

- [Cerebral Cortex](#)

Cortical Deafness

- [Verbal Auditory Agnosia](#)

Cortical Language Areas

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Synonyms

[Language cortex](#)

Structure

Anatomical Organization of Language Cortex in Autism

Cortical language areas are primarily centered around the perisylvian cortex, particularly in the left hemisphere. While the left posterior superior temporal gyrus (LSTG, BA 22, Wernicke's area) has been attributed to decoding language, the left inferior lateral prefrontal cortex (LIFG, BA 44/45, Broca's area) has been associated with motor expression of speech. In other words, Broca's area is the language production center, whereas Wernicke's area is the language comprehension center. In addition to these primary centers, there are several other areas involved in processing

language. For instance, the inferior parietal lobule (IPL) has a critical role in processing language because it is located at the junction of the auditory, visual, and somatosensory cortices. The IPL also shares connections with several other areas in the brain that are implicated in language processing. Other regions involved in language processing include the angular and supramarginal gyri (both are part of IPL), the planum temporale (PT), the insula, Heschl's gyrus (HG), and the parietal operculum. Heschl's gyrus is located within the Sylvian fissure, just anterior to the PT; whereas, the PT is located within the Sylvian fissure, posterior to HG and lies on the superior surface of the posterior STG. Many of these regions are anatomically connected through the arcuate fasciculus (AF), an axonal fiber bundle that arches around the ventral part of the frontal lobe and the posterior part of the temporal lobe.

Impairments in language and communication feature are one of the defining characteristics of autism spectrum disorders (ASD). Neuroimaging studies have found deviant functional brain responses as well as altered anatomical organization of the language cortex in people with ASD. One of the main anatomical abnormalities associated with the cortical language areas in ASD pertains to a reversed asymmetry in the normally left hemisphere dominant structures. For instance, studies have found decreased gray matter in Broca's area in people with ASD in comparison to typically developing individuals. Moreover, the right hemisphere homologue of Broca's area has been found to be much larger in 7–11-year-old children with ASD compared to typically developing peers. Such findings are also supported by evidence from neuronal density and neuronal volume in those with ASD. The differences in asymmetry may be due to a difference in the number of neurons present or possibly how densely they are packed together.

Although there are consistent findings of abnormal asymmetry of language association cortex in Broca's area for individuals with ASD, the picture is less clear for the relatively posterior language areas, such as Wernicke's area and the PT. Typical individuals with leftward lateralized language cortex generally also have leftward

lateralization of the PT. However, there is evidence from PT tracings that suggests there is no association between PT size or asymmetry and the lateralization of language cortex. More recent research noted a significant enlargement of the PT in the right hemisphere of individuals with ASD. Other research, however, has contradicted this finding by demonstrating reduced PT volume in the left hemisphere in ASD and no difference in the volume of Heschl's gyrus. There is also evidence that suggests that PT asymmetry may be related to language impairment; it has been found that individuals both with ASD as well as typical controls with language impairments have more leftward PT asymmetry than individuals with no language impairment. Therefore, language impairment may be an important variable in affecting or causing PT asymmetry. It is possible that age may be another factor that influences the organization of this region. Studies show age-related changes in the PT in both typically developing individuals and in individuals with ASD, with both groups showing increased leftward asymmetry in older than younger individuals.

The difference in asymmetry found in structures like the PT and the STG in people with ASD may directly point to problems associated with auditory processing of language. Children with ASD have been found to process nonspeech sounds abnormally early in development which implies that the deficit may stem from faulty organization of language processing areas within the auditory cortex. For instance, decreased gray matter volume of the left STG has been seen in children with ASD relative to participants without ASD. In addition to the alterations found in PT and STG, increased gray matter volume of the auditory cortex in individuals with ASD has been found in the periamygdaloid cortex, the left middle temporal gyrus, and the right inferior temporal gyrus. These anatomical alterations associated with language processing areas may be a key factor affecting language and communication difficulties in ASD. Many studies point to gray matter anomalies as the causal difference in language processing abilities of individuals with ASD. Such abnormalities are mostly due to the volume,

asymmetry, or neuronal organization of a region rather than lesions of a certain region.

Anatomical Connectivity of Cortical Language Areas

As previously mentioned, morphometry and lesion studies have found alterations in the volume and asymmetry of cortical language areas in individuals with ASD. Such impairments can have a significant impact on the organization and integrity of the anatomical connections among these regions. These connections, formed by axonal fiber tracts, provide the mechanism by which information is transferred from one area to the other. The arcuate fasciculus (AF) is a critical white matter fiber tract for language as it primarily connects the anterior and posterior core language areas. While the direct segment of the AF connects Wernicke's area with Broca's area, an indirect pathway connects these areas with the inferior parietal lobule. Specifically, the anterior segment of the indirect pathway connects Broca's area with the inferior parietal lobule and the posterior segment connects the inferior parietal lobule with Wernicke's area. The functioning of the typical language network relies heavily on the direct and indirect projections from the AF connecting these areas. Microstructural differences in the organization and asymmetry of AF found in people with ASD suggests that atypical asymmetry in people with ASD may be affecting language ability. Another fiber tract that is important for language is the uncinate fasciculus (UF), a hook-shaped fiber bundle that connects the anterior temporal lobe with the orbital frontal cortex including the IFG. Abnormalities in the UF fiber organization in individuals with ASD have recently been found in a Diffusion Tensor Imaging (DTI) study with the people with ASD showing shorter length of the left UF fibers as well as increased length, volume, and density of the right UF. These alterations in fiber organization in AF and UF may underlie the functional abnormalities associated with processing language in people with ASD.

It may not be a coincidence that the asymmetry seen in gray matter associated with language processing is also found in white matter in people with ASD. The differences in hemispheric

lateralization in those with ASD need to be discussed in the context of the structural integrity of the major white matter tract – the corpus callosum – that facilitates the communication between the two hemispheres. Reduced size of the midsagittal area of the corpus callosum in people with ASD has been widely reported. Despite this relatively consistent finding, the size of the subregions of the corpus callosum in those with ASD has been found to vary as some researchers found the reduction to be centered in anterior subregions like the rostrum and genu and others in the body of the callosum or in the splenium. In the context of language processing, the genu and the posterior midbody may be critical as the fibers pass through the former and connect the left and right frontal language areas and fibers passing through the latter connect the left and right temporal and parietal language areas. Research findings indicate the involvement of the corpus callosum in understanding humor, prosody, and decoding the nonliteral meaning within context. These complex linguistic functions may require communication between the two hemispheres that is facilitated by the corpus callosum. Therefore, it is possible that the alterations seen in the organization of the corpus callosum may constrain the language abilities of individuals with ASD.

Function

Functional Specialization of Language Cortex in ASD

Functional specialization and integration go hand-in-hand during brain development. Any faulty element in this delicate process can have a significant impact on how different brain regions respond to a given function. Abnormal specialization of language association cortex in people with ASD has been reported by several studies. For instance, individuals with ASD may have a faulty early perception of language due to impaired feature extraction, such as the correct decoding of phonemes or auditory language encoding. This may result in the left hemisphere language areas not receiving the proper input that

would allow for them to develop normally. When this normal input is not processed correctly early in the pathway, the brain does not receive accurate feedback that allows for the normal maturation of language cortex. In other words, deviant cognitive processing of external stimuli in those with ASD may cause a secondary neurobiological assault, which in turn may result in altered specialization of cortical areas. Due to language specialization occupying abnormal regions of the cortex in people with ASD, those areas may not be capable of functionally supporting language processing to the same extent as seen in typically developing individuals. The alterations in functional specialization in cortical language areas may result in different patterns of recruitment of cortical regions, especially between the two hemispheres.

Differential Recruitment of the Hemispheres

Perhaps mirroring the anatomical differences in the asymmetry of language association cortex, people with ASD have been found to recruit more right hemisphere areas in language tasks. In studies involving the processing of spoken language in individuals with ASD, the main findings have consisted of abnormal frontal and temporal activations, as well as reversed laterality in some regions. Functional MRI scans of babies (2–3 years old) during sleep have reported an increase in activation of right hemisphere regions in children with ASD. In addition to the deficits seen in temporal processing of speech in people with ASD, their difficulty in processing spoken language in the presence of noise was demonstrated by examining cortical encoding of speech in children. The left hemisphere is considered to be responsible for processing the temporal aspects of sound, such as encoding the sound properties of speech. Therefore, sound features that activate higher order language processing areas may not be representing the characteristics of the sound that are required in order to engage left hemisphere areas which are important in language processing and for causing language to lateralize to the correct hemisphere. Several other fMRI and PET studies examining auditory processing also found that people with ASD had less temporal

lobe activation and less activation that was lateralized to the left.

Other aspects of auditory processing, such as phonemic and prosodic variations in the context of spoken word, have also been found to be accomplished differently in people with ASD. They have abnormal functional lateralization within the left temporal area when processing phonemes. This is indicative of the fact that when left hemisphere dominance is not established, language impairments are more prominent. Impairments in the processing of prosody (pitch, rhythm, and stress patterns of language), have been reported in low- and in high-functioning people with ASD. Research shows that people with ASD are worse than typically developing controls at understanding the stress differences within words. At the neural level, while individuals with ASD show decreased recruitment of the left superior temporal sulcus in response to vocal sounds, they display typical activation in response to nonvocal sounds. Correct processing of prosodic information has been found to help initiate language acquisition and to help understand the mental states of others. Therefore, difficulty with prosody can have a significant impact in understanding and using language in social situations.

Differential Functional Activation and Functional Connectivity of Core Language Areas

Semantic and discourse processing (auditory or visual) have also resulted in altered brain responses in people with ASD. In tasks of sentence comprehension, Wernicke's area is more activated and Broca's area is less activated in people with ASD; whereas an opposite trend is observed in typical individuals. This pattern of brain response (increased activation in Wernicke's area) may suggest an increased reliance on word meaning and less emphasis on integrating words at the sentence level to arrive at meaning. This differential recruitment of core language areas has also been found at both the sentence level as well as at the word level in those with ASD. Broca's area has been proposed as a vital component of language processing, especially for mediating semantic integration and for the unification of

the components needed for processing language. Weakened integrative capacities have been reported in ASD in tasks that target contextual processing in semantic anomalies. When examining the integration of speaker information, Broca's area is not activated as highly by individuals with ASD as it is by typically developing participants. Yet, individuals with ASD showed increased activation in the right hemisphere homolog to Broca's area while comprehending speaker-incongruent sentences when compared to controls. The pattern of activation that is evident from these studies suggests that the differences in recruitment of language areas as well as a possible compensatory processing may be due to a spillover effect from the left hemisphere.

In addition to the activation of brain areas for accomplishing a task, the functional connectivity (synchronization of brain activation across activated areas) is critical in solving complex cognitive tasks. For instance, there is significant functional connectivity between Broca's and Wernicke's areas when listening to speech in typically developing individuals. Brain responses in complex language tasks in people with ASD have been found to be characterized by weaker connectivity among core regions. Weaker functional connectivity in ASD has been reported in tasks of active and passive sentence comprehension, sentence imagery, and in discourse processing. Such weak connections are usually found between regions that are critical for the task at hand, for example, there is a weaker connection between Broca's area and IPL in sentence imagery, and between Broca's and Wernicke's areas in sentence comprehension. In people with ASD, connections between the parietal and temporal lobes allow for their increased reliance on this area for processing, perhaps as an alternate route, along with reduced connections between Broca's and Wernicke's areas. Therefore, functional connectivity may play an equally important role in language processing as does functional activation.

Compensatory Strategies

Despite widespread reports of altered recruitment of cortical regions for language comprehension, many studies fail to find a pronounced difference in task performance in people with ASD

suggesting the possible use of compensatory strategies. One such mechanism may be an increased reliance on right hemisphere areas. Additional recruitment of right hemisphere brain regions is usually seen in typically developing individuals when task demands are increased and when higher-level language processing is needed. In studies of discourse processing and in detection of communicative intent, it has been found that people with ASD show increased right hemisphere activation in the homologues of core language areas. Such tasks are complex and may require the involvement of a network of coarse semantic processing, coherence monitoring, text integration, spatial imagery, and perspective taking. While any inference may elicit a right hemisphere response in people with ASD, only certain inferences, which are extremely difficult, may elicit that pattern in typical controls. This spillover effect has been demonstrated in other studies involving linguistic stimuli of differential difficulty in typical individuals and in story comprehension. Lesion studies have shown that damage to the left temporal pole causes difficulties in recalling stories whether they are spoken or written. This occurs even when participants have normal sentence comprehension skills and normal working memory. When listening to stories in particular, people with ASD activate the left temporal pole which is involved in recalling linguistic content. They also activate the right temporal pole which is involved in encoding and storing the prosodic and pragmatic aspects of the story. This finding was also demonstrated when the subjects were required to read the stories. These areas were recruited when the task demand increased, in contrast to simple lexical and semantic processing of single words. Therefore, these studies suggest that individuals with ASD may show bilateral activation regardless of difficulty.

Increased recruitment of relatively more posterior cortical language areas is another alternate neural route seen in those with ASD. In sentence comprehension studies involving high and low visual imagery, greater recruitment of parietal regions was found in participants with ASD irrespective of the presence of high or low imagery content. Similar results were also found with increased occipital activation in individuals

with ASD during semantic decision making tasks with a decrease in activation of the frontal verbal areas. These findings are also in line with Temple Grandin's view of "thinking in pictures" by recruiting visual and visuospatial regions to assist complex cognitive or linguistic processing. Research has also found weaker synchronization between language (left inferior frontal) and spatial (superior parietal) areas in people with ASD as compared to typically developed controls which further suggests focal or modular processing as opposed to integrative processing that is seen in control participants. Such findings were also supported by computational modeling techniques suggesting low level perception in individuals with ASD is superior and accessed easily compared to higher-level "top-down" cognitive processing.

Conclusion

This entry describes the anatomical and functional organization of cortical language areas and the abnormalities associated with them in people with ASD. In addition, it also deals with the ways in which cortical language areas participate and communicate with one another in ASD. The functional abnormalities seen in key language areas (Broca's and Wernicke's areas) as well as the differences in the organization of language association cortex in ASD may produce notable difficulties in language and communication. Explaining any cognitive function in ASD is difficult, as it may reflect the complexity of the disorder itself. The organization of language association cortex during brain development and the potential problems associated with it may affect subsequent functional and anatomical specialization. It is not clear whether the abnormal organization is neurobiological or is in turn the result of a secondary assault on the brain by early deviant behaviors in children with ASD, or both. Nevertheless, it should be noted that the brain of an individual with ASD, perhaps like the typical brain, adapts in certain ways to compensate for weaker connections and altered organization. Such adaptations, reflected in neuroimaging studies of language, may involve increased right

hemisphere recruitment, and an increased recruitment of relatively posterior language areas as well as visuospatial areas. Such atypical use of brain resources for solving cognitive and linguistic tasks may be a cause or a consequence of the altered organization and/or the difference in connectivity seen in individuals with ASD. Another factor that may complicate this topic is the heterogeneity seen in the ASD population in general and in their language abilities in particular. Learning about the organization, recruitment, and connectivity of cortical language areas in ASD should facilitate researchers as well as clinicians in making informed decisions and plans for language-based intervention in people with ASD.

Pathophysiology

The path of physiology of ASD is increasingly complex, perhaps reflecting the intriguing nature of the syndrome itself, with abnormalities found in several brain structures and in neurochemicals on a cellular level in cortical and subcortical structures. Significant cerebral hypo-perfusion (decrease of blood flow within the brain) has been widely reported in individuals with ASD especially in bilateral superior temporal cortices. This decrease in blood flow has been found to be more prevalent as the age of the child increases. A decrease in blood flow to the temporal lobes has been proven to cause impairments in communication, decreased language development, and auditory processing problems. The decreased blood flow has been hypothesized to be a result of vessels within the brain constricting rather than dilating which can lead to hypoxia and result in cell death. The constriction of blood vessels has been attributed to inflammation in the cortex of individuals with ASD. Abnormalities have also been reported in neuronal migration in postmortem cases of people with ASD along with abnormal minicolumn pathology, disrupted neuronal development, and glial cell abnormalities. Many of these abnormalities can have a significant impact on the organization of the brain in ASD.

Abnormalities in neurochemistry can also lead to problems in normal development of the brain. For instance, neurotransmitters like serotonin

regulate neurogenesis, synaptogenesis, neuronal removal, and differentiation. Normal high serotonin synthesis and synaptogenesis that takes place in early childhood is not seen in children with ASD. Although high levels of blood serotonin are found in children with ASD, it seems that they have below normal levels within their brains. It has been proposed that certain binding proteins (Mbd1) that are components of the “methylation-mediated epigenetic gene regulation system” could be an underlying factor in the dysfunctional serotonergic system seen in ASD. Studies show that mice who were missing this gene exhibited autistic like characteristics. Abnormalities in other neurotransmitters, such as the *N*-Acetyl Aspartate (NAA) and in glutamate were also reported in people with ASD. Together, these pathophysiological factors could play a significant role in causing disruption in the typical functioning of the brain in individuals with ASD.

See Also

- [Auditory Cortex](#)
- [Functional Connectivity](#)
- [Language Disorder](#)
- [Neuroanatomy](#)
- [Pragmatic Language Impairment](#)
- [Prosody](#)
- [Theories of Language Development](#)
- [Verbal Comprehension](#)

References and Reading

- Bauman, M. L., & Kemper, T. L. (1995). Neuroanatomical observations of the brain in autism. In J. Panksepp (Ed.), *Advances in biological psychiatry* (pp. 1–2). New York: JAI Press.
- Boddaert, N., Belin, P., Chabane, N., Poline, J. B., Barthélemy, C., Mouren-Simeoni, M. C., et al. (2003). Perception of complex sounds: Abnormal pattern of cortical activation in autism. *American Journal of Psychiatry*, *160*, 2057–2060.
- Catani, M., Jones, D. K., & Ffytche, D. H. (2005). Perisylvian language networks of the human brain. *Annals of Neurology*, *57*, 8–16.
- Dawson, G., Finley, C., Phillips, S., & Galpert, L. (1986). Hemispheric specialization and the language abilities of autistic children. *Child Development*, *57*, 1440–1453.

- De Fossé, L., Hodge, S. M., Makris, N., Kennedy, D. N., Caviness, V. S., Jr., McGrath, L., et al. (2004). Language-association cortex asymmetry in autism and specific language impairment. *Annals of Neurology*, *56*, 757–766.
- Diehl, J. J., Bennetto, L., Watson, D., Gunlogson, C., & McDonough, J. (2008). Resolving ambiguity: A psycholinguistic approach to understanding prosody processing in high-functioning autism. *Brain and Language*, *106*, 144–152.
- Harris, G. J., Chabris, C. F., Clark, J., Urban, T., Aharon, I., Steele, S., et al. (2006). Brain activation during semantic processing in autism spectrum disorders via functional magnetic resonance imaging. *Brain and Cognition*, *61*, 54–68.
- Herbert, M. R., Harris, G. J., Adrien, K. T., Ziegler, D. A., Makris, N., Kennedy, D. N., et al. (2002). Abnormal asymmetry in language association cortex in autism. *Annals of Neurology*, *52*, 588–596.
- Just, M. A., Cherkassky, V. L., Keller, T. A., & Minshew, N. J. (2004). Cortical activation and synchronization during sentence comprehension in high-functioning autism: Evidence of underconnectivity. *Brain*, *127*, 1811–1821.
- Kana, R. K., Keller, T. A., Cherkassky, V. L., Minshew, N. J., & Just, M. A. (2006). Sentence comprehension in autism: Thinking in pictures with decreased functional connectivity. *Brain*, *129*, 2484–2493.
- Kleinhans, N. M., Müller, R.-A., Cohen, D. N., & Courchesne, E. (2008). Atypical functional lateralization of language in autism spectrum disorders. *Brain Research*, *1221*, 115–125.
- Lord, C., & Paul, R. (1997). Language and communication in autism. In D. J. Cohen & F. R. Volkman (Eds.), *Handbook of autism and pervasive developmental disorders*. New York: Wiley.
- Paul, R., Augustyn, A., Klin, A., & Volkmar, F. R. (2005). Perception and production of prosody by speakers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *35*, 205–220.
- Redcay, E., & Courchesne, E. (2008). Deviant functional magnetic resonance imaging patterns of brain activity to speech in 2–3-year-old children with autism spectrum disorder. *Biological Psychiatry*, *64*, 589–598.
- Rojas, D. C., Camou, S. L., Reite, M. L., & Rogers, S. J. (2005). Planum temporale volume in children and adolescents with autism. *Journal of Autism and Developmental Disorders*, *35*, 479–486.
- Tager-Flusberg, H. (1996). Brief report: Current theory and research on language and communication in autism. *Journal of Autism and Developmental Disorders*, *26*, 169–172.

Cortical Remapping

► [Plasticity, Neural](#)

Cortical Rewiring

► [Plasticity, Neural](#)

Cortisol, Serum

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Synonyms

[Hydrocortisone](#)

Definition

Cortisol is a stress hormone secreted into the blood by the adrenal cortex of mammals. Its short-term effects of increasing metabolism, increasing blood sugar, and decreasing inflammation help the organism respond to stress and maintain homeostasis. Long-term elevations in cortisol can have a number of detrimental effects on the body and, perhaps, on the brain. Cortisol secretion by the adrenal is controlled by adrenocorticotrophic hormone (ACTH) secreted by the pituitary, with ACTH secretion in turn controlled by corticotrophin-releasing hormone (CRH) secreted by the hypothalamus. Cortisol exerts its peripheral and central effects by binding to the glucocorticoid receptor (GCR) found on most cells. Cortisol has been measured in the plasma, serum and urine of individuals with autism in order to assess their exposure to and response to stress, and their level of arousal. When taken together, prior studies appear to indicate that cortisol production is similar in individuals with autism compared to controls. However, there is some evidence that the normal diurnal rhythm of cortisol secretion (highest in the morning, lowest at night) might be altered in some individuals with autism.

See Also

► [Anxiety](#)

References and Reading

- Anderson, G. M., & Hoshino, Y. (2005). Neurochemical studies of autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 453–472). Hoboken: Wiley.
- Corbett, B. A., Mendoza, S., Abdullah, M., Wegelin, J. A., & Levine, S. (2006). Cortisol circadian rhythms and response to stress in children with autism. *Psychoneuroendocrinology*, 31(1), 59–68.
- Seltzer, M. M., Greenberg, J. S., Hong, J., Smith, L. E., Almeida, D. M., Coe, C., & Stawski, R. S. (2010). Maternal cortisol levels and behavior problems in adolescents and adults with ASD [research support, N.I.H., extramural research support, Non-U.S. Gov't]. *Journal of Autism and Developmental Disorders*, 40(4), 457–469.
- Tordjman, S., Anderson, G. M., McBride, P. A., Hertzog, M. E., Snow, M. E., Hall, L. M., & Cohe, D. J. (1997). Plasma beta-endorphin, adrenocorticotropin hormone, and cortisol in autism. *Journal of Child Psychology and Psychiatry*, 38(6), 705–715.
- Zinke, K., Fries, E., Kliegel, M., Kirschbaum, C., & Dettenborn, L. (2010). Children with high-functioning autism show a normal cortisol awakening response (CAR) [research support, non-U.S. Gov't]. *Psychoneuroendocrinology*, 35(10), 1578–1582.

Counterfeit Deviance

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Definition

The term “counterfeit deviance” is used to describe inappropriate behavior rooted in factors

other than true deviance. It is most often used in the context of inappropriate sexual behavior, in which the observed behavior looks paraphilic but, upon closer examination, was the result of other factors. Counterfeit deviance has been the focus of more discussion in the intellectual disability literature than in the autism-specific literature. There are clinical examples of its occurrence even among people with ASD who have adequate intellectual ability.

Traits associated with autism spectrum disorder (ASD) – including but not limited to difficulty taking another person’s perspective, social naiveté, preoccupation with particular areas of interest, differences in sensory perception – may make this population prone to situations that cause counterfeit deviance, particularly when the behavior of the person with ASD is assessed by someone unfamiliar with these traits. While one should always consider whether an observed behavior is merely counterfeit deviance, it is possible for a person with ASD to display a real deviant sexual interest or drive.

One clear example of counterfeit deviance is an 18-year-old young man with ASD and intellectual disability who was arrested for indecent exposure. He was taught at school that masturbation was something that he should do only at home. One day at home he masturbated in his backyard. The boy was spotted by a neighbor who called the police. When later asked why he masturbated outside, the boy said he thought it was allowed because he was “at home.” He was not aware that it would be problematic for anyone to see him. Counterfeit deviance occurred when he was viewed masturbating outside and assumed to be engaging in this behavior as a public display (i.e., exhibitionism).

See Also

- [Sex Education](#)
 ► [Sexuality and Problem Behaviors](#)
 ► [Sexuality in Autism](#)

References and Reading

- Dewinter, J., Vermeiren, R., Vanwesenbeeck, I., & Nieuwenhuizen, C. (2013). Autism and normative sexual development: A narrative review. *Journal of Clinical Nursing*, 22(23-24), 3467–3483.
- Griffiths, D., Hingsburger, D., Hoath, J., & Ioannou, S. (2013). 'Counterfeit deviance' revisited. *Journal of Applied Research in Intellectual Disabilities*, 26(5), 471–480.
- Hingsburger, D., & Griffiths, D. (1986). Dealing with sexuality in a community residential service. *Psychiatric Aspects of Mental Retardation Reviews*, 5, 63.
- Lockhart, K., Guerin, S., Shanahan, S., & Coyle, K. (2010). Expanding the test of counterfeit deviance: Are sexual knowledge, experience and needs a factor in the sexualised challenging behaviour of adults with intellectual disability? *Research in Developmental Disabilities*, 31(1), 117–130.
- Michie, A. M., Lindsay, W. R., Martin, V., & Grieve, A. (2006). A test of counterfeit deviance: A comparison of sexual knowledge in groups of sex offenders with intellectual disability and controls. *Sexual Abuse: A Journal of Research and Treatment*, 18(3), 271–278.
- Nichols, S., & Byers, E. S. (2016). Sexual well-being and relationships in adults with autism spectrum disorder. In *Autism spectrum disorder in mid and later life* (Vol. 248). London: Jessica Kingsley.
- Weiss, K. J., & Westphal, A. R. (2015). Autism spectrum disorder and criminal justice. In *Psychiatric expert testimony: Emerging applications*. New York: Oxford University Press.

Course of Development

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Synonyms

[Adult follow-up studies](#); [Adulthood, transition to](#); [Factors affecting outcome](#); [Natural history](#); [Outcome](#); [Outcome studies](#)

Definition

Autism is a lifelong disorder characterized by core problems in social communication and the presence of stereotyped and repetitive behaviors. However, the manifestation of these problems can change over time. In many cases, the severity of autistic symptoms decreases with age; in others, difficulties may become more evident as individuals grow older. In adolescence and adulthood, many individuals also develop additional mental health problems, particularly related to anxiety and depression.

The course of development is highly variable and often very difficult to predict. The most positive outcomes tend to be for individuals who develop useful speech in childhood and have an IQ in the normal range (i.e., 70+). Nevertheless, even among this group, some individuals remain highly dependent as adults. A good outcome also depends on the adequacy of intervention and support available during child- and adulthood.

See Also

- ▶ [Adult Follow-Up Studies](#)
- ▶ [Adulthood, Transition to](#)
- ▶ [Comprehensive Transition Program](#)
- ▶ [Factors Affecting Outcome](#)
- ▶ [Natural History](#)
- ▶ [Outcome Studies](#)

References and Reading

- Howlin, P. (2014). Outcomes in adults with autism spectrum disorders. In F. Volkmar, S. J. Rogers, R. Paul, & K. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (4th ed., pp. 97–116). Hoboken: Wiley.
- Howlin, P., & Magiati, I. (2017). Autism spectrum disorder: outcomes in adulthood. *Current Opinion in Psychiatry*, 302, 69–76.
- Kanner, L. (1973). *Childhood psychosis: Initial studies and new insights* (pp. 189–213). Washington, DC: V. H. Winston Sons.

Course of Social Avoidance in Fragile X Syndrome, The

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Definition

Social avoidance includes failure to initiate interactions, reduced time spent interacting with others, and social interaction restricted to a subset of preferred individuals. Elevated social avoidance is often associated with negative outcomes including reduced relationship quality and educational/vocational difficulties. High levels of social avoidance are commonly observed in individuals with autism spectrum disorder (ASD), attention-deficit/hyperactivity disorder (ADHD), and anxiety.

Fragile X syndrome (FXS) is a monogenic disorder associated with ASD. In males with FXS, social avoidance emerges in infancy, increases through early childhood, and stabilizes at a high level across adolescence and adulthood. Increased social avoidance across infancy and preschool predicts more severe ASD symptoms but reduced ADHD and anxiety symptoms in children with FXS.

Autism is a lifelong disorder characterized by core problems in social communication and the presence of stereotyped and repetitive behaviors. However, the manifestation of these problems can change over time. In many cases, the severity of autistic symptoms decreases with age; in others, difficulties may become more evident as individuals grow older. In adolescence and adulthood, many individuals also develop additional mental health problems, particularly related to anxiety and depression.

The course of development is highly variable and often very difficult to predict. The most positive outcomes tend to be for individuals

who develop useful speech in childhood and have an IQ in the normal range (i.e., 70+). Nevertheless, even among this group, some individuals remain highly dependent as adults. A good outcome also depends on the adequacy of intervention and support available during child- and adulthood.

See Also

- [Social Behaviors and Social Impairment](#)
- [Social Communication](#)
- [Social Motivation and Friendship Experiences of Autistic Adolescents](#)

References and Reading

- Roberts, J., Crawford, H., Hogan, A. L., Fairchild, A., Tonnsen, B., Brewe, A., O'Connor, S., Roberts, D. A., & Abbeduto, L. (2019a). *Journal of Autism and Developmental Disorders*, 49(9), 3753–3766. <https://doi.org/10.1007/s10803-019-04051-8>.
- Roberts, J. E., Crawford, H., Will, E. A., Hogan, A. L., McQuillin, S., Tonnsen, B. L., ... Brewe, A. M. (2019b). Infant social avoidance predicts autism but not anxiety in fragile X syndrome. *Frontiers in Psychiatry*, 10, 199. <https://doi.org/10.3389/fpsy.2019.00199>.

Court Decision (ASD Related)

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Definition

Court decisions involving ASD occur in a number of legal contexts. Perhaps the most common decisions address the issue of insurance coverage for the treatment of ASD. In the private insurance context, the dispute often centers on

coverage of behavioral therapies (Barner 2009). Insurers contend that these treatments are experimental or are educational services rather than health services. Court decisions turn on both the specific language of the policy at issue and the mandates of state laws (Barner 2009). Court decisions often also address the extent to which early intervention, custodial services, and other treatment modalities are covered by specific insurance policies.

Public health insurance also presents issues of ASD treatment coverage. Decisions regarding eligibility for coverage in Medicaid and other governmental programs often depend on whether a given intervention is classified as a mental health treatment, physical health treatment, or a neurodevelopmental therapy (Treatment 2009).

The educational setting provides another major context for court decisions regarding ASD. Litigation in this area often concerns the mandates of the federal Individuals with Disabilities Education Act (IDEA). IDEA's goal is "to ensure that all children with disabilities have available to them a free appropriate public education that emphasizes special education." The statute also mandates the provision of "related services designed to meet their unique needs and prepare them for further education, employment, and independent living." Services must be articulated in an Individualized Education Program (IEP). Litigation and resulting judicial decisions depend on a court's conclusion whether the process provided the student and the IEP itself in accord with the United States Supreme Court's decision in *Board of Education v. Rowley*. There, the Court held that the student must be provided with a fair process and that the IEP must be "reasonably calculated to enable the child to receive educational benefits."

ASD also plays a role in civil litigation between private parties, including the tort litigation contending that the MMR vaccine causes ASD (Thomas 2010). These cases, now totaling nearly 6,000, are being prosecuted under the National Vaccine Injury Compensation Program (Thomas). Individuals claiming an injury from a

covered vaccine must file a claim for "no-fault" compensation with the US Court of Federal Claims. The claims are resolved not by juries but by special masters. To date, the special masters have ruled against the plaintiffs on all six test cases, and all of those decisions appealed to higher courts have been affirmed. The Court of Claims has awarded compensation where the complaining child suffered from a preexisting mitochondrial enzyme deficit (Offit 2008).

On February 22, 2011, the United States Supreme Court ruled that the National Childhood Vaccine Injury Act (NCVIA) prohibits complainants from filing lawsuits against vaccine manufacturers. Rather, all such claims must be filed with the special Federal Court of Claims established under the National Vaccine Injury Compensation Program (Bruesewitz).

References and Reading

Important ASD-Related Court Decisions

- Board of Education of Hendrick Hudson Central School District v. Rowley, 458 U.S. 176 (1982) (all children are entitled to a Free Appropriate Public Education (FAPE)).
- Buckhannon v. West Virginia Dept. Health and Human Resources, 532 U.S. 598 (2001) (prevailing parents may waive their right to recover attorney's fees).
- Burlington School Committee v. Massachusetts Department of Education, 471 U.S. 359 (1985) (parents have a right to reimbursement for necessary private school tuition).
- Cedar Rapids Community School District v. Garret F., 526 U.S. 66 (1999) (schools must provide related educational services regardless of cost).
- Irving Independent School District v. Tatro, 468 U.S. 883 (1984) (except for physician services, schools must provide all necessary supportive services).
- Mills v. Board of Education of District of Columbia, 348 F. Supp. 866 (1972). (Children with disabilities have a right to a public education).
- Pennsylvania Association for Retarded Children (PARC) v. Commonwealth of Pennsylvania, 343 Fed. Supp. 279 (1972) (Children with disabilities have a right to a public education).
- Schaffer v. Weast, 546 U.S. 49 (2005) (burden of persuasion lies with the party seeking relief).
- Smith v. Robinson 468 U.S. 992 (1984) (parents prevailing under IDEA are not entitled to an award of attorney's fees).

COVID-19 and Its Impact on Individuals with Autism Spectrum Disorder

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Description of COVID-19

Coronaviruses (CoVs) are a group of diverse, enveloped, positive-sense, sing-stranded RNA viruses belonging to the subfamily Orthocoronavirinae in the family Coronaviridae, Order Nidovirales (Li et al. 2020). CoVs are known to cause disease in humans and animals, and can affect nervous, respiratory, hepatic, and enteric systems. In December 2019, patients in Wuhan, China, presented with pneumonia of unknown cause. It was determined after genome sequencing that the pneumonia – termed coronavirus disease 2019 (COVID-19) – was caused by a novel CoV, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) (Xu et al. 2020). Symptoms of the disease include mild to moderate respiratory illness, fever, and loss of smell.

Current Knowledge

State of Emergency

On January 20, 2020, the WHO declared COVID-19 a public health emergency, and eventually declared it a pandemic on March 11, 2020 (Dhama et al. 2020). This marked the first pandemic since the 2009 outbreak of H1N1. Initially, the majority of the public health burden associated with COVID-19 was confined mostly to China. However, the disease quickly spread to Europe and the United States of America (USA). The European countries that were affected most severely are Italy and Spain, but the USA has the largest number of confirmed cases and deaths due to COVID-19. The COVID-19 pandemic has resulted in stringent lockdown measures being

implemented worldwide, and has caused severe economic losses due to sudden disruptions in production and trade. Unfortunately, most of the governmental measures implemented worldwide have been reactive rather than proactive, and were not implemented early enough to minimize societal disruptions.

COVID-19 Effects on Mental Health

Many governments worldwide have adopted similar measures to combat COVID-19. These measures typically involve some level of lockdown, wearing masks, quarantining if necessary, and limiting social gatherings. While these measures have proven effective at slowing the spread of COVID-19, they have had a particularly negative impact on regional and global economies (Adhikari et al. 2019). Additionally, these stringent public health measures have caused adverse psychological impacts on the general population.

Studies show that negative psychological effects of the COVID-19 pandemic result from the unpredictability, uncertainty, misinformation, and social isolation surrounding the situation (Zandifar and Badrfam 2020). Vulnerable populations are especially at risk of developing adverse psychological symptoms due to the increased level of stress and panic caused by the possibility of developing complications if affected by COVID-19. The increased level of fear and panic among the general population can be highlighted by certain behaviors such as stockpiling household supplies and behaving violently toward those not following public health guidelines (Shigemura et al. 2020).

Many have hypothesized that the COVID-19 pandemic may lead to a global mental health crisis which would require large-scale psychological interventions and lasting reform of mental health care (Dong and Bouey 2020). Western countries have already begun to address mental health treatment in more recent protocols to combat COVID-19. However, countries such as China have yet to implement psychological interventions to reduce the mental health burden on not only affected individuals, but the general population.

COVID-19 Disruptions in the ASD Community

Those with autism spectrum disorder (ASD) – whether they be high-functioning individuals with Asperger’s syndrome or low-functioning individuals with severely debilitating autism – generally display a preference for specific daily routines (Fuld 2018). Any deviation from their daily routine may result in moderate to severe feelings of anxiety and stress. While governments worldwide have rightly implemented stringent lockdown measures, people with ASD have largely been forgotten. Lockdown, virtual learning, and wearing masks outdoors have resulted in severe disruptions in the lives of those with ASD. Government officials have rarely addressed this significant, vulnerable population in public health protocols. Some countries worldwide, such as the United Kingdom (UK), have relaxed public health protocols for those with ASD (Patel et al. 2020). However, these accommodations only came following legal challenges with the ASD community.

The COVID-19 pandemic poses a significant and continuous source of distress for those with ASD. The uncertainty surrounding the pandemic, as well as the continuous stream of new information regarding COVID-19, may worsen the mental health of those with ASD, even if they do not have an existing mental health comorbidity. One of the most significant sources of psychological stress for those with ASD is the sudden change in routine following the implementation of public health measures (Patel et al. 2020). Caregivers were often unable to provide warning of the impending changes in lifestyle, which likely exacerbated the feelings of psychological distress in those with ASD.

Individuals with ASD often meet with healthcare providers on a regular basis for check-ins and therapy. Additionally, many attend special education schools where they are engaged in various therapeutic educational programming. Due to COVID-19, there are many concerns as to whether such individuals can continue receiving these important services and treatment. A significant portion of schools in the USA and abroad have adjusted their curricula to include some form of virtual learning. This is also the

case for special education schools serving students with ASD. Adjusting to an at-home virtual learning environment provides significant challenges for individuals with ASD who already struggle with maintaining attention and completing tasks in-person (Patel et al. 2020). Without the in-person instruction of specialized educators, the parents must play a larger role in the educational process, posing additional strains on the family of individuals with ASD. Another important factor to consider is physical activity, as the social distancing parameters have caused many to live a sedentary lifestyle (Erkan and Oğuz Kaan 2020). Individuals with ASD often require some form of physical activity throughout the day, and governments have often loosened the outdoor public health protocols for those with ASD due to these requirements. Various physical activities that can be done at home have been outlined by researchers and highlight the importance of family participation (Erkan and Oğuz Kaan 2020).

Advice to Help Individuals with ASD to Cope with the COVID-19 Pandemic

Several studies and reviews have been published on methods to help individuals with ASD cope with the disruption the COVID-19 pandemic has caused. One way to assist those with ASD in coping with the disruptions is to structure their day around various activities using a blackboard or some sort of chart (Narzisi 2020). The pandemic will require families with children who have ASD to find new activities for play, and to schedule these activities at specific times throughout the day. Lego therapy and various video/computer games have also been possible suggestions; however, it is important for parents to find out what works best for their own children (Narzisi 2020). Ordering these activities into a daily schedule will help to minimize any resistance or distress surrounding the changes. Another important aspect to consider is how the current pandemic disrupts education for individuals with ASD. Since many academies and schools have now moved to an online platform, students with ASD will need to make quite an adjustment. One case study of five families with individuals who have ASD in the Philippines reported significant

struggles with adjusting to online schooling (Cahapay 2020). Parents reported the importance of communicating to their children the realities of the current pandemic and its impact on daily life (Cahapay 2020). One parent accomplished this by driving their child around the city so that they could see how businesses were closed down, and also tried to teach their child new social behaviors such as wearing a mask (Cahapay 2020). It is important to communicate to individuals with ASD the realities of this pandemic, and furthermore work with families to teach these individuals new social skills that will help them cope with the extended period of time spent at home.

Future Directions

The COVID-19 pandemic has caused significant worldwide disruptions in nearly every facet of daily life. One population that has been forgotten in the midst of new government regulations and public health protocols is the ASD community. Individuals with ASD typically require a specific daily schedule, any deviation from which may result in significant anxiety and distress. Additionally, individuals with ASD are typically involved in some sort of therapy, social work, or special education. Many of these social services are being moved online, which may pose a significant challenge for individuals with ASD. It is imperative that government officials keep the ASD community in mind when deciding on new public health protocols, and that teachers and parents continue to work together to minimize the disruptions caused by this pandemic on the ASD community.

Bibliography

- Adhikari, S. P., Meng, S., Wu, Y. J., Mao, Y. P., Ye, R. X., Wang, Q. Z., Sun, C., Sylvia, S., Rozelle, S., Raat, H., & Zhou, H. (2019). Epidemiology, causes, clinical manifestation and diagnosis, prevention and control of coronavirus disease (COVID-19) during the early outbreak period: A scoping review. *Infectious Diseases of Poverty*, 9, 29. <https://doi.org/10.1186/s40249-020-00646-x>. U.S. National Library of Medicine.
- Cahapay, M. B. (2020). How Filipino parents home educate their children with autism during COVID-19 period. *International Journal of Developmental Disabilities*, 1–4. <https://doi.org/10.1080/20473869.2020.1780554>.
- Dhama, K., et al. (2020). Coronavirus disease 2019–COVID-19. *Clinical Microbiology Reviews*. American Society for Microbiology. www.ncbi.nlm.nih.gov/pmc/articles/PMC7405836/
- Dong, L., & Bouey, J. (2020). Public mental health crisis during COVID-19 pandemic, China. *Emerging Infectious Diseases*, 26. <https://doi.org/10.3201/eid2607.200407>. U.S. National Library of Medicine.
- Erkan, Y., & Oğuz Kaan, E.. (2020). Promoting physical activity for children with autism spectrum disorders during coronavirus outbreak: Benefits, strategies, and examples. *International Journal of Developmental Disabilities*, 27, 49Z. Taylor & Francis. www.tandfonline.com/doi/full/10.1080/20473869.2020.1756115?casa_token=J-b9hfAYcyIAAAAA%3APb11Pg3qvPxNs9n4sPuuQbQEOPCJZKx4liN-MhkJ9lwBooFav4hNksHRvP5qeZs4ICLL3t-WC2zltg
- Fuld, S. (2018). Autism spectrum disorder: The impact of stressful and traumatic life events and implications for clinical practice. *Clinical Social Work Journal*. <https://doi.org/10.1007/s10615-018-0649-6>. U.S. National Library of Medicine.
- International Journal of Developmental Disabilities. www.tandfonline.com/doi/citedby/10.1080/20473869.2020.1756115?scroll=top&needAccess=true
- Li, H., et al. (2020). Coronavirus disease 2019 (COVID-19): Current status and future perspectives. *International Journal of Antimicrobial Agents*. Elsevier B.V. and International Society of Chemotherapy. www.ncbi.nlm.nih.gov/pmc/articles/PMC7139247/#bib0011
- Narzisi, A.. (2020). Handle the autism spectrum condition during coronavirus (COVID-19) stay at home period: Ten tips for helping parents and caregivers of young children. *MDPI*. Multidisciplinary Digital Publishing Institute. www.mdpi.com/2076-3425/10/4/207/html?fbclid=IwAR2Y511tTSRVy7IA7Jry0qMMc8d-ntEjXBxEenV-Iz5zdpMzlj5oBX0YKu
- Patel, J.A., et al. (2020). COVID-19 and autism: Uncertainty, distress and feeling forgotten. *Public Health in Practice*. Elsevier Ltd on Behalf of The Royal Society for Public Health. www.ncbi.nlm.nih.gov/pmc/articles/PMC7392884/#bib2
- Shigemura, J., Ursano, R. J., Morganstein, J. C., Kurosawa, M., & Benedek, D. M. (2020). Public responses to the novel 2019 coronavirus (2019-NCov) in Japan: Mental health consequences and target populations. *Psychiatry and Clinical Neurosciences*. <https://doi.org/10.1111/pcn.12988>. U.S. National Library of Medicine.
- Xu, X., Chen, P., Wang, J., Feng, J., Zhou, H., Li, X., Zhong, W., & Hao, P. (2020). Evolution of the novel coronavirus from the ongoing Wuhan outbreak and modeling of its spike protein for risk of human

transmission. *Science China Life Sciences*. <https://doi.org/10.1007/s11427-020-1637-5>. U.S. National Library of Medicine.

Zandifar, A., & Badrfam, R. (2020). Iranian mental health during the COVID-19 epidemic. *Asian Journal of Psychiatry*. <https://doi.org/10.1016/j.ajp.2020.101990>. U.S. National Library of Medicine.

CP

- [Cerebral Palsy](#)

CPEP-3

- [Simplified Chinese Psychoeducational Profile Third Edition](#)

CPRS

- [Children's Psychiatric Rating Scale](#)

CPT

- [Conners' Continuous Performance Test](#)

CPT-II

- [Conners' Continuous Performance Test](#)

Craniofacial Microsomia

- [Goldenhar Syndrome](#)

Craniognomy

- [Phrenology](#)

Craniology

- [Phrenology](#)

Cranioscopy

- [Phrenology](#)

Creak, Mildred

Adam Feinstein
Autism Cymru and Looking Up, London, UK

Major Appointments (Institution, Location, Dates)

- Mildred Creak worked as assistant physician at The Retreat, a mental hospital run by Quakers in York, UK, 1924–1928.
- Took up a post at the Maudsley Psychiatric Hospital, London, 1929.
- Appointed physician in psychological medicine at the Hospital for Sick Children, Great Ormond Street, London, 1946–1963.

Landmark Clinical, Scientific, and Professional Contributions

In the early 1960s, Mildred Creak chaired the working party which established the landmark nine-point criteria for the diagnosis of autism, published in 1961. This work was based on a series of 100 children she had collected herself. Creak suggested that autism, far from being caused by parental inadequacies, was primarily due to genetic – or, as she put it, “constitutional” – factors.

Short Biography

Born in Manchester, UK, in 1898, Mildred Creak was an extraordinary figure in the history of child psychiatry. She qualified as a doctor at University College Hospital in London at the end of the First World War. It was at the Children’s Department of London’s Maudsley Psychiatric Hospital, from 1929, that Creak helped to lay the clinical and academic foundations for what is now one of Britain’s leading centers for the study of child psychiatric disorders. During the Second World War, she joined the Women’s Army Corps as a doctor, serving part of her time in India. From 1946 – when she joined the Hospital for Sick Children in London’s Great Ormond Street – until her retirement in 1963, she played a leading role in establishing the practice of child psychiatry in a pediatric setting. After her retirement, Creak lectured in Perth, Western Australia, and had a unit for autistic children named after her there. She died in the UK in 1993 at the age of 95.

References and Reading

- Creak, M. (1961). Schizophrenic syndrome in childhood: Progress report of a working party. *Cerebral Palsy Bulletin*, 3, 501–504.
- Feinstein, A. (2010). *A history of autism: Conversations with the pioneers*. Oxford: Wiley-Blackwell.

Criminality, Interactions with Law Enforcement, and Potential Correlates of Juvenile Justice Involvement Among Youth with Autism

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Objectives

Understanding criminal behavior in juvenile justice-involved youth with autism spectrum disorders (ASD), potential factors associated with involvement in the juvenile justice system (JJS), and experiences of interacting with law enforcement.

ASD and Crime in the Media

In the past decade, the mass media has often inaccurately linked ASD with criminal behavior. For example, perpetrators, including Lauri Love and Gary McKinnon, who hacked into US computer systems, and Adam Lanza, the 2012 Sandy Hook School shooter, were reportedly diagnosed with mild forms of ASD (Holden and Shirbon 2018; Kushner 2011; McCoy 2014). Furthermore, media claimed that Christopher Harper-Mercer, who was the perpetrator in the Oregon community college shooting, and Dylann Roof, the perpetrator in the 2017 Charleston church shooting, also had ASD (Calabresse et al. 2015; Tripp 2017). Even more recently, the media has suggested that Nikolas Cruz, the individual who opened fire at a school in Parkland, Florida, was diagnosed with ASD (Dearen et al. 2018). The mass media typically links ASD to criminality with captivating headlines such as *FLORIDA SCHOOL SHOOTING: Nikolas Cruz was*

Creativity

► [Imagination](#)

diagnosed with autism, ADHD; Students rage against politicians, NRA in anti-gun rally (Dearen et al. 2018) which not only stigmatizes mental health in general but specifically links ASD to criminal behavior, which leads to negative reactions from the public and stigmatizations of the ASD community (Brewer et al. 2017). In fact, ASD advocates have begun to release statements cautioning against connecting ASD to school shootings (Diament 2018).

Moreover, the way in which the mass media links ASD to criminal behavior is problematic methodologically because the media focuses on high-profile, extreme cases which occur rarely within the general population. Furthermore, extensive reviews of the literature have not revealed a direct link between ASD and criminal behavior, including violent criminal activity (Bjorkly 2009; Lerner et al. 2012). Finally, media reports can be misleading because these report do not provide evidence of a formal ASD diagnosis of the perpetrator (Brewer et al. 2017; Maras et al. 2015). Nevertheless, the attention that the ASD community has received in the mass media has led many researchers to turn their attention to understanding criminal behavior among persons, including youth, with ASD.

Youth with ASD in the JJS

Prevalence Rates

ASD prevalence in the general population is 1.7% (Baio et al. 2018) but reports of prevalence rates of JJS involvement among youth with ASD vary widely within the United States (US). The lowest rate of first juvenile court involvement among youth with ASD (i.e., first contact with the JJS) was estimated at 0.02% (Kincaid and Sullivan 2019), which is consistent with another study that revealed the rates of youth with ASD in the JJS in Connecticut between 2006 and 2012 to be 0.04% (Slaughter et al. 2019). However, other studies have reported higher rates of JJS involvement. For example, one study recorded the rate of arrest among youth with ASD to be 4.7% (Rava et al. 2017), and another showed the percentage of youth with ASD who were charged with a crime

to be 5% (Cheely et al. 2012). Among studies conducted outside of the United States, inconsistencies in prevalence rates are also present. Specifically, research in Japan reported ASD prevalence rates ranged from 1.3% to 6.7% across all, but traffic violations and car accidents, charges in three family courts (Kumagami and Matsuura 2009). However, when a specialty family court for sex-related crimes or arson was considered, the prevalence rate was 18.6%. Inconsistencies in these estimates may be related to methodological differences (e.g., samples, statistical approaches), yet it is clear that youth with ASD do come into contact with the JJS.

Types of Crimes

The types of crimes committed by youth with ASD vary, with some studies showing a higher likelihood for these youth of committing crimes against persons compared to youth who did not have a disability (Cheely et al. 2012; Kincaid and Sullivan 2019). Specifically, law enforcers frequently respond to calls related to verbal and physical aggression directed at others, such as family members (Tint et al. 2017). However, other research suggests that youth with ASD are no more likely to commit crimes against person than youth without special educational needs (Slaughter et al. 2019). Special attention has also been paid to sex-related offenses, as one study presented higher rates of sex-related crimes among youth with ASD compared to the general population referred to family courts in Japan (Kumagami and Matsuura 2009). However, when researchers explored sexual offending among individuals with ASD as a follow-up to their original report, there was inconclusive support for the assumption that individuals with ASD commit sex-related offenses (Allely and Creaby-Attwood 2016). With these equivocal findings, it remains unclear as to whether youth with ASD are in fact more likely to commit sex-related offenses.

Studies of property offenses also generated mixed results. Research shows that youth with ASD are less likely to commit property crimes when compared to youth without a disability (Cheely et al. 2012; Slaughter et al. 2019). This may suggest youth with ASD are less likely to be

involved with crimes requiring premeditation when compared to their nondisabled peers. However, this assumption is brought into question with more recent research suggesting that youth with ASD were more likely to commit property crimes compared to youth without disabilities (Kincaid and Sullivan 2019). It is possible that property crimes are committed by youth with ASD who are behaviorally or emotionally dysregulated, as the likelihood of property damage does tend to increase under such circumstances (Hodgetts et al. 2013). In these instances, youth with ASD likely would not have premeditated an offense but could have been arrested for property destruction nonetheless. However, more research is needed to determine intentionality in committing such crimes.

Finally, with regard to recidivism, youth with ASD are not more likely than youth without special educational needs to reoffend (Slaughter et al. 2019), although studies examining recidivism in youth with ASD are scarce. In fact, to our knowledge there have been no other studies that have looked at recidivism rates among youth with ASD. This is surprising since recidivism is a key post-release outcome considered in criminal justice research, and ASD-related work is needed in this area.

A Comparison with Adult Offenders with ASD

Given the shortage in research examining recidivism as well as longitudinal studies of criminal behavior, research on adults with ASD may shed some light as to whether criminal behavior patterns remain consistent over time. A recent literature review examining studies using both criminal records and self-report data revealed that the prevalence of offending behavior among adults with ASD varied across studies ranging from 8% to 26% (Rutten et al. 2017). One study found that the most common convictions (81%) among adults with ASD were for property offenses (Hippler et al. 2010), though other researchers have concluded property crimes were less prevalent among adults with ASD (Rutten et al. 2017). It also appears that adults with ASD may be less likely to commit traffic offenses, and they report taking illicit drugs at lower rates than adults

without ASD (Mouridsen et al. 2008; Woodbury-Smith et al. 2006). With regard to violent crimes or crimes against persons, available research suggests that these crimes are rare in populations of adults with ASD (Björkly 2009; Hippler et al. 2010). However, when these crimes do occur, their common triggers include sensory hypersensitivity, tactile hypersensitivity, and misinterpretation (Björkly 2009). Therefore, it may be informative for future research to attempt to understand offending behavior among those with ASD, especially with regard to crimes against persons, in the context of the disorder.

Taken together, prevalence rates for involvement with the justice system may slightly increase in adulthood when compared to adolescence. There also continues to be some debate as to whether individuals with ASD commit property crimes at higher rates than those without ASD. Furthermore, crimes against persons continue to be prevalent in adulthood, although violent crimes are rare. However, longitudinal research is needed in order to better explore these patterns.

Associated Factors of JJS Involvement

School Discipline

There are several factors that are associated with JJS involvement among youth with ASD. School discipline is one factor that is important to consider as recent research has discovered that school discipline and JJS involvement often co-occur among youth with ASD (Slaughter et al. 2019; Turcotte et al. 2018). Based on teacher reports, some research shows that youth with ASD are 2.5 times more likely to be suspended when compared to youth without disabilities (Ambler et al. 2015). However, other studies suggest they may be less likely to receive such disciplinary actions compared to students who do not have a disability and students with other disabilities, such as emotional disturbance and learning disabilities (Miller and Meyers 2015; Slaughter et al. 2019). Nevertheless, about half of youth with ASD who have come into contact with the JJS have received at least one out-of-school suspension (Slaughter et al. 2019). Additionally, youth with ASD who

have come into contact with the police are more than four times as likely to also have school discipline infractions (Turcotte et al. 2018). Therefore, youth with ASD who are excluded from the supportive environment of the school system through suspensions and expulsions may be at higher risk of coming into contact with the JJS.

Externalizing Behaviors

Additionally, fighting at school that results in a disciplinary referral is associated with reoffending and may serve as a warning sign that youth with ASD are at risk for future JJS involvement (Slaughter et al. 2019). This is supported by other research showing that externalizing behaviors in youth with ASD such as aggression is associated with being stopped, questioned, and arrested by law enforcers (Rava et al. 2017; Tint et al. 2017). Finally, compared to youth whom do not have a disability, youth with ASD are also more likely to be charged for school-based incidents such as disturbing the school, carrying a weapon, assault and battery, or threatening a school employee (Cheely et al. 2012). For these reasons, both externalizing behavior and school discipline may increase the odds of having interactions with law enforcement among youth with ASD.

ASD and Law Enforcement

Interaction with Law Enforcers

Law enforcers may encounter youth with ASD in a variety of situations. Many youth with ASD (i.e., about 49%) elope from the home, and therefore law enforcement may be called when the youth with ASD is lost (Anderson et al. 2012). Officers may also be called when caregivers or teachers cannot manage behaviors like aggression, tantrums, and self-injury (Jang et al. 2011; Matson and Nebel-Schwalm 2007; Tint et al. 2017). Aggressive behaviors are often a primary presenting concern for contacting law enforcers. Finally, youth with ASD may encounter law enforcement in more communal spaces.

Interactions with law enforcement may be extremely difficult for youth with ASD.

Specifically, core characteristics of ASD such as difficulties with social interactions, atypical responses to sensory stimuli, and exhibiting restrictive interests and repetitive behaviors will affect how persons with the diagnosis interact with law enforcers. Law enforcers who are not aware that the youth has been diagnosed with ASD may misinterpret certain behaviors associated with the diagnosis (e.g., not responding to verbal commands, lack of eye contact, repetitive behaviors) as threatening, suspicious, non-compliant, or signaling that the youth is under the influence of drugs or alcohol when these behaviors are merely manifestations of the diagnosis (Teagardin et al. 2012). Additional challenges are present when the youth with ASD is nonverbal and cannot communicate to the law enforcers that they are diagnosed with ASD. Moreover, ASD is often considered a hidden disability, as there are not outward indications of the diagnosis. When law enforcers are not trained to recognize certain characteristics of ASD and respond appropriately, they may unintentionally exacerbate the situation, which may result in trauma, injury, or even fatality (Copenhaver and Tewksbury 2019; Teagardin et al. 2012).

Training Programs for Law Enforcers

There is a growing demand for high-quality training services for law enforcers. For example, one study conducted in England and Wales revealed that only about one-third of police officers reported having received ASD-specific training; and refresher courses are rare (Crane et al. 2016). The number of officers who receive ASD-specific training in the United States is less clear. In general, police officers in England and Wales find ASD-specific trainings to be helpful. However, some are dissatisfied with the training because they felt trainings did not focus on ASD in the context of the justice system, were oversimplified, and lacked practical application as well as relevance to their policing role (Crane et al. 2016). Police officers who have not received police training overwhelmingly report that training would be useful. Work in this area has revealed that police officers would like to receive more information on enhancing communication, minimizing distress,

and specific training for interaction with someone with ASD as it relates to their specific policing role (Crane et al. 2016). This research highlights the need for high-quality training programs that focus on both education and practical utility.

Future Directions

A brief review of the current literature regarding JJS involvement among youth with ASD reveals that much additional work is needed to address both gaps and inconsistencies. This is apparent with regard to the aforementioned equivocal findings such as inconsistencies with regard to prevalence rates, types of crimes, and satisfaction of interactions with law enforcers. Additionally, several limitations exist within the current literature. For one, many studies have been cross-sectional in nature, which highlights the need to study criminal behavior in this population over time. In addition, how ASD was diagnosed (e.g., educational classification, psychological diagnoses) in participants varies across studies, which makes it difficult to draw reliable conclusions. Moreover, few research studies focus on mental health comorbidities and other environmental factors that may increase the risk for JJS involvement (e.g., family factors). This is especially surprising given that many youth with ASD have comorbid medical and psychological diagnoses. There has also been a lack of self-report data collected within this population. Given the limitations of official crime data, which is subject to police and the court's discretion, self-report data (e.g., surveys, qualitative interviews) may, in some cases, provide critical and useful information. Therefore, there are many opportunities for researchers to contribute to the current literature base.

Furthermore, there is a need for more research in the areas of criminal behavior (i.e., types of crime and recidivism), experiences of interactions with law enforcement and the JJS more broadly, as well as factors that may lead to involvement in the JJS (e.g., school discipline, mental health comorbidities, specific ASD traits that increase risk of involvement in the JJS, and social factors). There appears to be a connection between school

discipline and contact with not only law enforcers but also the JJS more broadly. Specifically, more research is needed examining fighting behavior in school. Additional research should also be conducted with youth who have ASD, particularly among those who have been suspended or expelled from the school system. A high number of youth with ASD who have come into contact with the JJS have been suspended from school.

Recidivism rates are important to examine because youth, in general, who have come into contact with the JJS are at an increased risk of coming into contact with the JJS in the future. In theory, youth with ASD are also subject to the increase in risk of coming into contact with the JJS after their first offense. Reasons for how and why youth with ASD recidivate would also be important to explore.

Additionally, many youth in the general population who come into contact with the JJS have mental health comorbidities as well as other environmental factors that increase their risk of involvement with the JJS. However, surprisingly, the research to date has not studied these factors in relation to JJS involvement in youth with ASD. This is especially surprising given medical and psychological comorbidities are very common among youth with ASD (Lai et al. 2013).

Researchers can also use self-report data to understand experiences of police interactions and JJS involvement from youth with ASD. In trying to understand experiences of the JJS, it is important for researchers to study the school environment (e.g., school discipline, arrests on campus, special education services), the crime itself (e.g., triggers), and the experience of interacting with police officers as well as detention staff and juvenile probation officers (e.g., what could have gone better or worse). With regard to rehabilitation, it is also important to examine intent, competency, and remorse.

It also is important to continue to interview law enforcers in order to determine what training they have received and where this training falls short. Current research suggests that specific training topics should include characteristics of the disorder to be aware of as well as effective ways to identify an ASD diagnosis to law

enforcement. Special attention should be paid to how these training programs can incorporate practical skills. Practical skills can be developed via both role-plays and interacting with the ASD community. Moreover, there is a need to develop training programs for youth with ASD and their families so that they are better prepared when they encounter first responders, including law enforcers. Specific topics should include information on how to interact with law enforcers, as well as effective ways to identify an ASD diagnosis to law enforcement.

At a broader level, it appears that aggression is a primary concern for contacting first responders at school, home, and in the community. This highlights the need to develop community-based family support (Tint et al. 2017), as well as support for school personnel so that these behaviors do not escalate to the point that law enforcers must be contacted. It would also be helpful for advocacy groups to create on-hand resources that can be used by first responders in order to become more familiar with ASD in general. To date, there are some online and print resources for first responders and youth with ASD and their families. However, these resources are typically found across many different websites, and it can be difficult to find an aggregated guide of resources. Information in Table 1 provides an overview of currently available resources in this vein. Suggestions below the table include key points from these resource pages, as well as the clinical experiences of the authors. It is important to note that Dennis Debbaudt, an autism advocate and author, has developed many resources for law enforcers and the ASD community; some of these resources can be found in the links provided above.

To conclude, youth with ASD do come into contact with the JJS; however, it seems that these contacts are not ASD-specific. With regard to factors associated with the JJS involvement, there appears to be a link between school-based offenses as well as fighting behavior at school, which may increase the risk of youth with ASD becoming involved in the JJS. Yet, the causal nature of these associations still needs to be

explored. Importantly, interactions with law enforcers can be extremely difficult for youth with ASD given certain characteristics of the disorder. Therefore, it is vital to understand youth with ASD and their families' experiences of the JJS in order to improve justice system interactions with individuals with the disorder and train law enforcers accordingly.

Suggestions for Law Enforcers

- It may be helpful to turn off police lights and cease sirens, as many youth with ASD are sensitive to bright light and loud noises.
- Avoid approaching the youth from behind.
- Avoid touching the youth, which could exacerbate sensory sensitivity.
- Outbursts or impulsive behaviors are not uncommon in autism. If no one is at risk of harm, wait for the behavior to decrease before continuing conversations with the youth.
- The youth may not respond to verbal commands; they may not understand the request or requirement, or they may be unsure how to respond in such a situation. Calmly and reassuringly, try again to communicate. You may have to repeat things multiple times or in different ways.
- Speak slowly and clearly, avoiding slang and sarcasm, which may be difficult for youth with ASD to understand.
- Using simple, plain language, pictures, or symbols may be more effective for communication.
- Allow time for the youth with ASD to respond to commands or requests (10–15 s).
- Check for an identification bracelet or autism handout card.
- If there is a parent, caregiver, or adult support with the youth with ASD, consider speaking to them first as they may be able to tell you the best way to communicate to the youth.
- Do not question youth with ASD without a support (e.g., caregiver, parent, legal guardian) present, as they may be easily influenced by others.
- When questioning the youth, keep in mind that many youth with ASD may:
 - Have difficulty with communicating and/or expressing themselves

Criminality, Interactions with Law Enforcement, and Potential Correlates of Juvenile Justice Involvement Among Youth with Autism, Table 1 Online resources for caregivers and law enforcers

ASAN's Safety Toolkit https://autisticadvocacy.org/safety
Autism & Law Enforcement: 25 Field Response Tips by Dennis Debbaudt https://www.gapost.org/pdf_file/autism.pdf
Autism Alliance of Michigan Safety Training & Support https://autismallianceofmichigan.org/project/safety/
Autism Crime Victims Resource Guide https://www.autism-society.org/wp-content/uploads/2014/04/relative-victim.pdf
Autism First Responder Training Video https://www.madisonhouseautism.org/police-interaction-autism-building-positive-relationships/
Autism Research Institute https://www.autism.com/law_enforcement
Autism Risk and Safety Management https://www.autismriskmanagement.com/
Autism Society https://www.texasautismsociety.org/first-responder-autism-training-video/
Autism Society Resources for First Responders https://www.autism-society.org/wp-content/uploads/files/2014/04/Law_Enforcement_and_Other_First_Responders.pdf
Autism Speaks Resources for First Responders https://www.autismspeaks.org/information-first-responders
Aware Collaboration http://awaare.nationalautismassociation.org/
Emergency Preparation Planning https://aacommunity.net/2018/09/emergency-preparation-for-aac/
Emergency Preparedness Fact Sheet by Autism Society of Minnesota https://www.ausm.org/images/docs/Emergency%20PDFs/EP2015FactSheetv2.pdf
First Responders Guide by TAMU https://disabilitytips.tamu.edu/autism.html
Kentucky Autism Training Center https://louisville.edu/education/kyautismtraining/professionals/training-for-first-responders
Organization for Autism Research https://researchautism.org/autism-spectrum-and-law-enforcement-training/
Prevention Planning Guide https://autisticadvocacy.org/wp-content/uploads/2017/11/Autism-and-Safety-Pt-3.pdf
Safety Products https://www.autismspeaks.org/safety-products-and-services?gclid=Cj0KCQjwy6T1BRDXARIsAlqCTXrkOllzgxE02a5YHdN53iZjmHWYj7ppWN7GcI33PewtsXU7GluiEDMaAv3DEALw_wcB
Spectrum https://www.spectrumnews.org/opinion/viewpoint/training-first-responders-recognize-autism-may-avert-tragedies/
The Arc https://www.thearc.org/NCCJD/training/webinars/archive
The Autism and Law Enforcement Education Coalition https://www.arcsouthnorfolk.org/alec/
The Johnson Center https://www.youtube.com/watch?v=SGCkcPYBXyA&t=224s

- Respond more slowly than others their age
- Not understand verbal commands, especially if they are conveyed using metaphors, sarcasm, slang, or other imprecise terms
- Not understand nonverbal communications (e.g., head nods, hand signals)
- Be nonverbal
- Avoid making eye contact, while others with ASD may have very intense eye contact
- Try to hide their disability and not tell law enforcers that they have been diagnosed with ASD
- Try to run away
- Engage in repetitive behaviors (e.g., twirling objects, rocking, hand flapping,

pacing, talking to themselves), and this can worsen with stress

- Become upset with even slight changes to their routine
- Be interested in shiny objects such as your badge, keys, belt buckle, etc.
- Youth with ASD may need assistance filling out forms. You may have to offer general assistance or may have to read forms to the youth.

Suggestions for Caregivers

- Use an identification bracelet. It is not recommended that your child carries an autism identification card, as law enforcement may assume they are reaching for a weapon when the youth reaches for the card.

- Talk to your child about interacting with law enforcement and make a plan.
- Discuss disclosure of diagnosis to law enforcement.
- Make sure your child knows your phone number and home address or has this information with them so that they can contact you in an emergency.
- Make a plan for what you would want to communicate to the police officer about your child's diagnosis in case you are with your child during the interaction.
- Contact local disability agencies to help you create a plan.
- Think about developing community outreach programs to provide education to law enforcers and the chance for law enforcers to interact with the autism community.
- It may be helpful to provide your local police department with autism resources (e.g., brochures, handouts) so that they may become more familiar with autism in general.

References and Reading

- Allely, C., & Creaby-Attwood, A. (2016). Sexual offending and autism spectrum disorders. *Journal of Intellectual Disabilities and Offending Behaviour*, 7(1), 35–51.
- Ambler, P. G., Eidels, A., & Gregory, C. (2015). Anxiety and aggression in adolescents with autism spectrum disorders attending mainstream schools. *Research in Autism Spectrum Disorders*, 18, 97–109.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Publishing.
- Anderson, C., Law, J. K., Daniels, A., Rice, C., Mandell, D. S., Hagopian, L., & Law, P. A. (2012). Occurrence and family impact of elopement in children with autism spectrum disorders. *American Academy of Pediatrics*, 130(5), 870–877.
- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M., Daniels, J., Warren, Z., . . . Dowling, N. F. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. Surveillance Summaries. *Centers for Disease Control and Prevention*, 67(6), 1–23.
- Bjorkly, S. (2009). Risk and dynamics of violence in Asperger's syndrome: A systematic review of the literature. *Aggression and Violent Behavior*, 14, 306–312.
- Brewer, N., Zoanetti, J., & Young, R. L. (2017). The influence of media suggestions about links between criminality and autism spectrum disorder. *Autism*, 21, 1–5.
- Calabrese, E., Almaguer, M., & Helsel, P. (2015, October 6). Mother of Oregon College shooter wrote about guns, autism. Retrieved from <https://www.nbcnews.com/storyline/oregon-college-shooting/mother-oregon-college-shooter-wrote-about-guns-autism-n439766>
- Cheely, C. A., Carpenter, L. A., Letourneau, E. J., Nicholas, J. S., Charles, J., & King, L. B. (2012). The prevalence of youth with autism spectrum disorders in the criminal justice system. *Journal of Autism and Developmental Disorders*, 42, 1856–1862.
- Copenhaver, A., & Tewksbury, R. (2019). Interactions between autistic individuals and law enforcement: A mixed-methods exploratory study. *American Journal of Criminal Justice*, 44(2), 309–333.
- Crane, L., Maras, K. L., Hawken, T., Mulcathy, S., & Memon, A. (2016). Experiences of autism spectrum disorder and policing in England and Wales: Surveying police and the autism community. *Journal of Autism and Developmental Disabilities*, 46, 2028–2041.
- Dearen, J., Breed, A., & Lush, T. (2018, February 17). Florida School Shooting: Nikolas Cruz was diagnosed with autism, ADHD; Students rage against politicians, NRA in anti-gun rally. *Associated Press*. Retrieved from <https://canoe.com/news/world/florida-school-shooting-nikolas-cruz-was-diagnosed-with-autism-adhd-students-rage-against-politicians-nra-in-anti-gun-rally>
- Diamant, M. (2018, February 20). Advocates caution against autism connection in school shooting. Retrieved from <https://www.disabilitycoop.com/2018/02/20/advocates-caution-school-shooting/24746/>
- Hippler, K., Viding, E., Klicpera, C., & Happe, F. (2010). Brief report: No increase in criminal convictions in Hans Asperger's original cohort. *Journal of Autism and Developmental Disorders*, 40, 774–780.
- Hodgetts, S., Nicholas, D., & Zwaigenbaum, L. (2013). Home sweet home? Families' experiences with aggression in children with autism spectrum disorder. *Focus on Autism and Other Developmental Disorders*, 28(3), 166–174.
- Holden, M., & Shirbon, E. (2018, February 5). Autistic UK man accused of hacking FBI wins appeal against extradition to U.S. Retrieved from <https://www.reuters.com/article/us-britain-usa-hacker/autistic-uk-man-accused-of-hacking-fbi-wins-appeal-against-extradition-to-u-s-idUSKBN1FP11A>
- Jang, J., Dixon, D. R., Tarbox, J., & Granpeesheh, D. (2011). Symptom severity and challenging behavior in children with ASD. *Research in Autism Spectrum Disorders*, 5, 1028–1032.
- Kincaid, A. P., & Sullivan, A. L. (2019). Double jeopardy? Disproportionality in first juvenile court involvement by disability status. *Exceptional Children*, 85(4), 453–470.
- Kumagami, T., & Matsuura, N. (2009). Prevalence of pervasive developmental disorder in juvenile court

- cases in Japan. *The Journal of Forensic Psychiatry and Psychology*, 20, 974–987.
- Kushner, D. (2011, June 27). The autistic hacker: Gary McKinnon hacked thousands of government computers. Retrieved from <https://spectrum.ieee.org/telecom/internet/the-autistic-hacker>
- Lai, M., Lombardo, M. V., & Baron-Cohen, S. (2013). Autism. *The Lancet*, 383(9920), 896–910.
- Lerner, M. D., Haque, O. S., Northrup, E. C., Lawer, L., & Bursztajn, H. J. (2012). Emerging perspectives on adolescents and young adults with high-functioning autism spectrum disorders, violence, and criminal law. *Journal of the American Academy of Psychiatry and the Law*, 40, 177–190.
- Maras, K., Mulcahy, S., & Crane, L. (2015). Is autism linked to criminality? *Autism*, 19(5), 1–2.
- Matson, J. L., & Nebel-Schwalm, M. (2007). Assessing challenging behaviors in children with autism spectrum disorders: A review. *Research in Developmental Disabilities*, 28, 567–579.
- McCoy, T. (2014, May 21). Study: ‘Significant’ statistical link between mass murder, autism, brain injury. Retrieved from <https://www.washingtonpost.com/news/morning-mix/wp/2014/05/21/study-finds-significant-portion-of-mass-murderers-and-serial-killers-had-neurological-disorders-including-autism/?noredirect=on>
- Miller, C. E., & Meyers, S. A. (2015). Disparities in school discipline practices for students with emotional and learning disabilities and autism. *Journal of Education and Human Development*, 4(1), 255–267.
- Mouridsen, S. E., Rich, B., Isager, T., & Nedergaard, N. J. (2008). Pervasive developmental disorders and criminal behaviour. A case control study. *International Journal of Offender Therapy and Comparative Criminology*, 52(2), 196–205.
- Rava, J., Shattuck, P., Rast, J., & Roux, A. (2017). The prevalence and correlates of involvement in the criminal justice system among youth on the autism spectrum. *Journal of Autism and Developmental Disorders*, 44, 340–346.
- Rutten, A. X., Vermeiren, R. R. J. M., & Van Nieuwenhuizen, C. (2017). Autism in adult and juvenile delinquents: A literature review. *Children and Adolescent Psychiatry and Mental Health*, 11(45), 1–12.
- Slaughter, A. M., Hein, S., Hong, J. H., Mire, S., & Grigorenko, E. L. (2019). Criminal behavior and school discipline in juvenile justice-involved youth with autism. *Journal of Autism and Developmental Disorders*, 49(6), 2268–2280.
- Teagardin, J., Dixon, D. R., Smith, M. N., & Granpeesheh, D. (2012). Randomized trial of law enforcement training on autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6, 1113–1118.
- Tint, A., Palucka, A. M., Bradely, E., Weiss, J. A., & Lunsky, Y. (2017). Correlates of police involvement among adolescents and adults with autism spectrum disorder. *Journal of Autism and Developmental Disabilities*, 47, 2639–2647.
- Tripp, D. (2017, May 10). Dylann Roof likely has autism, but preferred death over that label, court records show. Retrieved from <https://abcnews4.com/news/manuel-ame-shooting/dylann-roof-likely-had-autism-but-preferred-death-over-that-label-court-records-show>
- Turcotte, P., Shea, L. L., & Mandell, D. (2018). School discipline, hospitalization, and police contact overlap among individuals with autism spectrum disorder. *Journal of Autism and Developmental Disabilities*, 48, 883–891.
- Woodbury-Smith, M. R., Clare, I. C. H., Holland, A. J., & Kearns, A. (2006). High functioning autistic spectrum disorder, offending, and other law-breaking: Findings from a community sample. *Journal of Forensic Psychiatry and Psychology*, 17(1), 108–120.

Criterion

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Synonyms

Benchmark; Measure; Standard

Definition

Criterion is a standard for minimally acceptable performance of a specific behavior or skill by an individual in order for the skill or behavior to be evaluated and judged as mastered. Criterion provides a defined and measurable answer to questions about how and when an individual has acquired a particular behavior or skill.

Criterion is one of three essential components of a behavioral objective, which is a mandated part of a student’s individualized education plan (IEP). A behavioral objective specifies a target behavior to be taught to a student, the conditions under which the behavior will be taught, and finally, the criterion, how mastery of the behavior will be assessed. An individual might be expected to perform a skill at a certain level of accuracy or

independence across a certain amount of days, number of times, or at a certain rate in order for the skill to be considered mastered. Choices about criterion are to be made based on the skill being taught. For example, an expectation of 80% accuracy may be acceptable in showing mastery of a math skill on a math quiz. However, 80% accuracy would not be an acceptable criterion for teaching an individual how to cross a street because this means that the student would be in danger of not making it across the street 20% of the time.

See Also

- ▶ [Behavioral Objective](#)
- ▶ [Education](#)
- ▶ [Educational Interventions](#)
- ▶ [Functional Goals](#)
- ▶ [Individual Education Plan](#)

References and Reading

- Alberto, P. A., & Troutman, A. C. (2009). *Applied behavior analysis for teachers* (8th ed.). New York: Merrill.
- Snell, M. E., & Brown, F. (2006). *Instruction of students with severe disabilities* (6th ed.). Upper Saddle River: Pearson Education.

Criterion Referenced Assessment

- ▶ [Assessment of Functional Living Skills \(AFLS\)](#)

Criterion-Referenced Assessment

- ▶ [Criterion-Referenced Testing](#)
- ▶ [Verbal Behavior Milestones Assessment and Placement Program \(VB-MAPP\)](#)

Criterion-Referenced Testing

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Synonyms

[Criterion-referenced assessment](#); [Criterion-referenced tests](#)

Definition

Criterion-Referenced Tests

Criterion-referenced tests are designed to assess whether an individual has a particular set of competencies or skills. They aim to answer questions such as “Is this child able to use a spoon for self-feeding or do up shoelaces?” or “Does this individual use sentences of four or more words or know how to subtract two numbers?” The focus of interest is the presence or absence of the criterion behavior and not, as in the case of norm-referenced testing, how the individual functions relative to some normative group.

Criterion-referenced measures may also be used to determine whether the individual meets the requirements for entry in special educational provision or for other decision processes. Psychiatric or other diagnoses are forms of criterion-referenced testing, the presence or absence of signs and symptoms of ASD constituting the criteria. Such uses of criterion-referenced test are sometimes known as “high-stakes testing” because of the major personal consequences of the outcome. In such uses in particular, including for clinical purposes, it is critical that the tests meet key criteria for quality (Kaplan and Saccuzzo 2009); that the tester is competent in administration, scoring, and interpretation of the test; and that the testee was functioning as they typically do.

Criterion-referenced items can function as norm-referenced measures. For instance,

developmental tests will have population norms on sentence length. Hence, a 5-year-old who has not yet achieved four-word sentences may be seen as being severely behind in their expressive speech compared with other 5-year-olds, whereas a 2-year-old with such skills would be seen as within the typical range of development for their age. Used in this way, they should be subject to the same quality standards noted for norm-referenced measures.

See Also

- [Autism Diagnostic Interview-Revised](#)
- [Autism Diagnostic Observation Schedule](#)

References and Reading

Kaplan, R. M., & Saccuzzo, D. P. (2009). *Psychological testing: Principles applications and issues* (7th ed.). Belmont: Wadsworth, Cengage Learning.

Criterion-Referenced Tests

- [Criterion-Referenced Testing](#)

Critical Autism Studies

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Synonyms

[Emancipatory autism studies](#); [Transformative autism studies](#) (Woods et al. 2018)

Definition

Introduction

Critical Autism Studies (CAS) is the only autistic-led community of practice, within autism studies,

and it has a few proposed definitions. A recent entry by notable autistic CAS scholars reviewed the main definitions and its ontology to propose a more inclusive approach between autistic and non-autistic academics; the preferred definition put forward is:

The ‘criticality’ comes from investigating power dynamics that operate in discourses around autism, questioning deficit-based definitions of autism, and being willing to consider the ways in which biology and culture intersect to produce ‘disability’. (Waltz 2014, p. 1337)

For a discussion on the other primary CAS definitions, see Woods et al. (2018). This essay outlines key concepts from disability studies and the nature of the neurodiversity movement before explaining how CAS is relevant to autism. The next section introduces disability studies and contextualizing core ideas with examples relevant to current debates in autism.

Disability Studies

Disability studies started in the 1960s (Woods et al. 2018) and grew out of disability activism (Barnes 2008; Goodley 2011; Oliver 2013; Thomas 1999), generating two terms and definitions that form a novel way of conceptualizing disability, instead of the dominant medical model of disability; these are impairment and disability. Impairment is viewed as the loss functional ability within a person due to the presence of either mental, physical, or sensory impairment. Disability is the loss of opportunities that an impaired person experiences due to physical and social barriers due that prevent living a normal life (Goodley 2011).

A focal point for disability campaigners is created by separating the biological aspects of impairment from the societal impact; it places the emphasis on adapting the environment to meet the needs of the individual. In this capacity the social model of disability is a tool for locating barriers and removing them. Like all tools, it has consequences to its use, as it can be blunt in application. By definition an impaired person’s family is also made disabled if they lose opportunities due to the impairment. For instance, if an autism parent has to sacrifice work to homeschool their child, the parent is made disabled. Some

suggest the social model is a threat to the medical model. Contrarily, the social model encourages the use of approaches from other disability models, especially if they remove barriers to impaired person's inclusion (Barnes 2008). For instance, the social model encourages the use of inclusive practices; for instance, if an autistic person requires augmentative and alternative communication (AAC) to participate in school activities, the AAC should be provided. Despite these positive aspects of the social model, it fails to explain the full nuances of lived experience, and therefore other ideas are required.

Exploring theoretical approaches to disability, Carol Thomas made significant strides in conceptualizing disability. One of Thomas' concepts is impairment effects, the loss of activities resulting from the impairment but not by disability as viewed by the social model. Thomas also developed disableism, as a form of oppression that involves restricting opportunities for impaired persons. The negative impact of such treatment on an impaired person's well-being is called psycho-emotional disableism (Thomas 1999). This can be viewed as part of the second wave of disability studies scholars. In contrast Fiona Campbell, approaching the cultural aspects of disability as part of Critical Disability Studies, coined ableism to investigate cultural structures and practices that reify the perfect human person; in the process impaired persons are viewed as diminished versions of this. Drawing upon critical race theory, Campbell proposed internalized ableism as the processes when parts of an oppressed (impaired) population demographic internalize discourses used to oppress them and then re-emit those discourses creating conflict between members of the oppressed group (Campbell 2008).

Disability studies scholarship is described as falling into a number of waves. The first-wave disability studies originated in the 1980s to the early 1990s, developing the social model and is a sociology derivative (Barnes 2008; Shakespeare 2017). The second wave focused on critique of the social model, investigating lived experience of disabled persons and drawing upon other discourses like feminism. Finally, the third wave, also known as Critical Disability Studies, focuses on the cultural processes contributing to disability

and thus drawing upon critical theory (Shakespeare 2017). However, one can view disability studies as being critical since its inception in the 1960s (Woods et al. 2018). Where CAS fits among these waves will be discussed later.

We have detailed core concepts and debates in disability studies. Now we highlight how their relevance to autism utilize the proposed autism subtype of pathological demand avoidance (PDA). The impairment would be high anxiety, and the impairment effects would be the demand avoidance. For internalized ableism, PDA is the oppressive discourse that is being accepted and expressed by some autistic persons. During the 1980s psychologists sought for traits that are switched off in autistic persons, while switched on non-autistic persons (Fletcher-Watson and Happé 2019). For PDA, the ability to comply with demands of everyday life and other's demands is severely impaired; nonetheless, non-PDA persons, the opposite is applicable. Thus, those who identify with PDA then re-emit the PDA discourse creating conflict within the autistic rights and neurodiversity movements.

For disableism, an example would be when an autistic female is denied an autism diagnosis due to the male bias diagnostic profile; and so some are suggesting PDA is a female form of autism (Gould and Ashton-Smith 2011), and it can be used to remove the barrier to an autism diagnosis for some autistic females. In contrast, PDA has been noted as a misdiagnosis for autistic females (Chown 2017). PDA highlights the complex and sometimes pluripotential nature of disability studies concepts when applied to autism.

As disability rights and disability studies became mainstream, the gains of the social model have receded (Barnes 2008; Oliver 2013). This is partly due to sustained academic challenge to the social model and to its adoption of a social model-light by disability charities (Oliver 2013). Since the 2007 financial crash, austerity measures concerted with disability legislation have eroded rights gained in previous decades, and the latter is used to police impaired persons. (Berghs et al. 2019). Berghs and colleagues (ibid.) went further to argue for a replacement of the social model, specifically a social model of human rights, to enable impaired persons to live full and

flourishing lives that the social model has failed to produce. Autistic persons counter that the social model was never fully implemented for all impaired persons, especially autistic persons (Woods 2017b). Such sentiments are supported by recent reports indicating autistic persons are being increasingly failed by society (All Party Parliamentary Group on Autism 2019). Precisely where autistic rights movement and how autistic persons are treated by society will be discussed in the neurodiversity section.

This section has introduced core terms in disability studies and its history. The discipline evolved from sociology and has drawn heavily from outside areas, such as feminism and critical theory. We provided examples of how these terms apply to autism by utilizing the proposed subtype PDA, such as how it is leading to internalized ableism among autistic persons. Finally, we discuss how the present treatment of impaired persons is leading to calls for a stronger social model of human rights.

Neurodiversity Movement

This year is the 20th birthday of neurodiversity, since Judy Singer's seminal essay in disability studies. The concept proposed is built upon the social model of disability where impairments like attention deficit hyperactivity disorder are perceived as natural variations of human diversity. Since then an entire movement has grown around the concept and it is primarily associated with autism, yet, it encompasses many other impairments. Indeed from its inception in the United Kingdom (UK), there have been substantial debates over conditions and thus who are encompassed by the term (Arnold 2017; Chown 2019a). Likewise, there is an ongoing debate over the strongest theoretical position, with a minority utilizing biological citizenship. This is based on genetic or biological citizens claiming rights on a diagnostic category or condition and is a medical model version (Runswick-Cole et al. 2016), not social model. Considering these contradictory outlooks, there are often intense clashes among supporters of neurodiversity.

There is a complicated relationship between neurodiversity and the wider disability rights

movements. This is reflected on how original disability rights movement focused on physical and sensory impairments (Chown 2019a). Although definition for impairment has been expanded to include mental impairments such those covered by neurodiversity movement (Goodley 2011), the social model is not practiced for mental impairments (Woods 2017b). Logically, those with mental impairments have since formed their own rights movement, which is often centered on the neurodiversity paradigm (Chown 2019a). Crucially, autistic rights movement and neurodiversity are separate and heterogeneous groupings with parallel endeavors (Woods et al. 2018). This variation between neurodiversity supporters is partly due to divergent developmental paths the movement has taken between the UK and North America (Arnold 2017). A problem situated to UK neurodiversity movement are the clashes between social model supporters and those who favor biological citizenship. The latter is favored by those who identify with the proposed form of autism, pathological demand avoidance (PDA) (Milton 2018).

Over the last several years, neurodiversity has been increasingly critiqued within the wider public (Den Houting 2019). Likewise the movement has come under sustained critique in CAS (Bolton 2018; Guest 2020; Runswick-Cole 2014; Runswick-Cole et al. 2016; Woods et al. 2018). CAS is a community of practice that overlaps second- and third-wave disability studies that focus on the autism impairment label. An earlier attempt to stop these straw man attacks on the paradigm seems to have failed. Thus, a more rigorous response is needed. We do not repeat previous dialogues on the debate, instead highlighting the positive contributions of neurodiversity to CAS. The next section explores how neurodiversity paradigm and its supporters have provided core literature to the intrinsic characteristics of CAS.

Current Knowledge

In order to explain how something has contributed to CAS, we need to know what its constituent traits are. The nature of CAS has been explored in a recent redefinition article, which posits CAS has the following characteristics:

CAS definition must be inclusive, be critical, allow new lines of inquiry and have epistemological integrity; the latter is important for the discipline's external acceptance. (Woods et al. 2018, p. 977)

We now explain and explore how neurodiversity supporters have provided key literature to these individual traits. First the article will explore these contributions that provide the critical component of CAS's name.

The origins of CAS are traced back to the birth of autistic self-advocacy, with Jim Sinclair's speech "Don't Mourn For Us" in 1993 (Woods et al. 2018). Sinclair challenges much of the pathologizing discourses about autism and 6 years before Judy Singer's seminal work. This indicates that autistic persons were looking for a movement to rally behind before neurodiversity existed. This view is in line with the historic position that those with mental impairments had not been included in the wider disabled persons movement. Pertinently, since 1999, many autistic persons resonate with the neurodiversity paradigm and have been actively involved in CAS since its inception at academic events almost a decade ago (Woods et al. 2018). Such individuals in 2012 launched the only CAS journal, *Autonomy, the Critical Journal of Interdisciplinary Autism Studies*. The role of this journal in CAS will be discussed later.

Over the last decade, numerous neurodiversity supporters have been challenging the medical model perspectives of autism. For instance, Melanie Yergeau has produced numerous key texts to CAS, where she critiques various concepts like theory of mind to the behaviorist-based intervention, Applied Behavioral Analysis (ABA) (2010, 2013, 2017). The primary motivation for such scholarship is to turn the source of problems experienced by autistic persons away from perceived faults located in autistic individuals and instead onto wider societal processes. Arguably, the most famous example is Damian Milton's double empathy problem that theorizes the communication issues experienced with autistic persons and is the result of a mismatch of saliences between autistic and non-autistic individuals (Milton 2017). This growing body of literature is pivotal to addressing the systemic

oppression faced by autistic persons (Woods 2017b), particularly considering the limited effectiveness of most autism strategies and interventions (Milton 2017). Ironically, the one intervention shown to be effective with high-quality evidence supports the double empathy problem, as it seeks to synchronize behaviors between carer and autistic infant (Pickles et al. 2016). Through such critical perspectives autistic scholarship is providing a turning point to empower themselves.

Autistic persons frequently are aware of the limitations and flaws found within autism studies. One such limitation is that of an academic silo; this is when the disciplines form their own communities of practice and so not understanding the potential contributions other disciplines can make. To assist in breaking down these barriers between silos, autistic persons founded the only CAS journal, the autistic-led journal *Autonomy, the Critical Journal of Interdisciplinary Autism Studies* (Arnold 2012). Many would note that there is a research-to-practice gap. However autistic scholarship has identified a theory-to-research-to-practice gap, with a lack of literature drawing upon autism cognitive theories (Chown 2017). Cross-pollinating disciplines are needed to help close the theory-to-research-to-practice gap by encouraging the use of broader theories in autism research.

Recently there are growing calls for participatory research on ethical and epistemic grounds (Milton 2017; Woods and Waltz 2019). Autistic research priorities tend to be in areas often overlooked by traditional research, such as support and services (Pellicano et al. 2014). Therefore, conducting inclusive research creates novel areas. Investigating the claims of a high-profile journal that we are in a new era of autism research, a preliminary study suggests that 3% of UK autism research is meeting autistic community wishes (Chown 2019b). With autistic persons being commonly unsupported, there is a pressing need for autism research to adopt an emancipatory ethos.

CAS scholarship contains many new lines of inquiry, not necessarily found in the main medical model-based literature, ranging from critique of

historically autism research being typically poor quality and unethically overstating the results (Dawson 2004; Milton 2017; Waltz 2007) to analyzing the activities of autism charities (Rosenblatt 2018; Waltz 2012). Pertinently, CAS draws upon third-wave disability studies by analyzing the cultural aspects contributing to autism. Important aspects include (1) drawing upon feminism, CAS seeks to explore the experiences of autistic persons through intersectionality (Saxe 2017; Strand 2017); (2) how autism is used as a Foucauldian biopower to control individuals (McGuire 2015; Moore 2019); (3) how the construct of autism is commodified into a form of control, frequently to the benefit of non-autistic persons (Runswick-Cole et al. 2016; Woods 2017a), for instance, how stigma around autism is intertwined with the processes that commodify the construct (Grinker 2020); and (4) to the cultural impact, negative medical model-based autism discourse, for instance, is a factor in autism mercy killings (McGuire 2016; Waltz 2008), often this is when a carer kills their child, in the vain hope of killing their autism (McGuire 2016). Through such novel authorship, CAS questions how biology and culture interact to construct the diagnostic category of autism.

There is growing evidence from autistic academics and the autistic population that all autistic individuals' mental health benefits from being in charge of their own lives, partially due to the pressures of conforming to societal demands (Milton 2017; Milton and Moon 2012; Stewart 2012; Woods 2017b). Autistic individuals also face additional societal pressures from psycho-emotional disableism (Milton 2013; Milton and Lyte 2012; Stewart 2012; Woods 2017b). For example, camouflaging is linked to suicidal ideation in autistic persons (Cassidy et al. 2018). Societal pressures contribute to the higher suicide rates and potentially higher rates of self-harming for autistic individuals (Maddox et al. 2017). It is clear that there is a growing momentum to act on the turning point CAS provides for autism studies to be autistic-led.

Neurodiversity supporters' perspectives are source of epistemic integrity to CAS; this is reflected in the growing evidence that

corroborates anecdotal views. Autistic persons often question behaviorist-based autism interpretations and interpretations (Dawson 2004; Milton 2017; Williams 2018; Yergeau 2017), especially aimed on behaviorist approaches that are typically used on vulnerable autistic children. Crucially, such critique is gaining more evidence (Harte 2019; Hassiotis et al. 2018; Kupferstein 2018). Autistic persons frequently challenge the accuracy of functioning-based labels and the negative effects they have for underestimating the challenges faced by all autistic persons. This has recently been supported by a large-scale quantitative study that indicates that IQ scores are an imprecise predictor of functional abilities (Alvares et al. 2020). Comparable observations from good-quality research noting how autism subtypes are clinically unstable and autism subtypes have similar prognosis (Happé 2011), thus supporting autistic preferences for a single diagnostic category (Fletcher-Watson and Happé 2019). The autistic autism theory, monotropism theory, is viewed by many neurodiversity supporters as the strongest autism theory (Woods and Waltz 2019). While it lacks direct empirical testing (Fletcher-Watson and Happé 2019), it is supported by emerging qualitative research (Bertilsson-Rosqvist 2019; Leatherland 2018; Wood 2019). Utilizing the strength of autistic perspectives is crucial to the integrity of autism and helping to improve the quality of life for autistic persons.

A central theme to CAS is the inclusion of autistic perspectives throughout the research process. A misconception of the neurodiversity movement is that it only represents those who are "high functioning." Contrastingly, one of its goals is to ensure all autistic persons (and their families) gain appropriate support (Den Houting 2019; Fletcher-Watson and Happé 2019). After all under the social model of disability, those around impaired persons are also made disabled by how society treats these vulnerable persons. Autistic persons are systematically oppressed. Consequently, this adversely affects autism parents through psycho-emotional disableism (Angell and Solomon 2017; Macleod et al. 2017).

There are numerous signs of this position. Firstly, neurodiversity supporters frequently

argue for the inclusion of all autistic persons' perspectives in autism research for epistemic and ethical grounds (Milton 2017; Woods and Waltz 2019). Secondly, social model supporters have developed inclusive methodologies for underrepresented autistic demographics, including those with learning disability, such as by Bertilsdotter-Rosqvist et al. (2019), Nicolaidis et al. (2019), Ridout (2014, 2016), and Stewart (2012). More recent inclusive methodologies are seen in CAS by other scholars; see Andrews et al. (2019) and Macloud (2019). Thirdly, the National Autistic Taskforce is composed of many neurodiversity supporters, and it is a body specifically seeking to address shortcomings in practice for autistic persons and learning disability. Fourthly, neurodiversity inclined networks, like the Participatory Autism Research Collective, hold annual conferences, in part to provide a platform for those who would not typically be heard. Through such practices, neurodiversity supporters are seeking to remove social barriers to the inclusion of all autistic persons.

One key notion when considering inclusiveness, which is key to CAS, is the recognition of the role of power. As aforementioned, there is an increasing number of autistic scholars and a growing number of publications; autistic academics "upset the apple cart" in terms of power and further complicate notions of power and whose position is more dominant, as autistic people normally considered to be passive participants in research or assessment (Milton et al. 2019), rather than directing their own scholarship. This is fitting with the intersectional nature of current movements including fourth-wave feminism (Evans 2013, p. 7) and, more notably in this case, the autistic civil rights movement (Pountney 2018). Against the backdrop of postmodernist notions of identity and being, we can truly appreciate the importance of examination of power in inclusive research. The Participatory Autism Research Collective and the current drive for "inclusive participatory research" from scholars and activists alike (e.g., Bertilsdotter-Rosqvist et al. 2019; Chown et al. 2017; Fletcher-Watson et al. 2019) have called for reflexivity of standpoints from scholars and researchers, which is applicable to

and necessary in CAS. Furthermore, examination of power and its impact on inclusion allows for further lines of inquiry to be considered, for example, who may define what "good quality of life" is for an autistic person (Waldock 2019) and whose agenda is being served (as discussed in Milton 2017). This demonstrates the importance of recognizing power as part of the inclusive dimension of CAS.

This entry has explored what Critical Autism Studies is and contextualized the background to this growing discipline of autism studies. Initially exploring key points of the derivative of sociology, Disability Studies that CAS heavily draws upon. Subsequently, we discuss the nature of the neurodiversity movement and how this is both separate to and mutually supporting the autistic rights movement. In the final section, we detail how autistic academics have played a leading role to the emergence of CAS and their importance to the constituent parts of the discipline, "critical, allow new lines of inquiry and have epistemological integrity" (Woods et al. 2018, p. 977). In the process we highlight the importance of CAS as being the only discipline of autism studies to be autistic-led due to the involvement of neurodiversity supporters.

Due to the myriad of topics covered in CAS, it is not possible for us to provide more than this descriptive overview of the discipline, and we encourage readers to explore the recommended reading. Moreover, with the growing trend away from tokenistic autism research, by its nature as an autistic-led discipline, it will continue to gain traction in wider autism studies; in the process, it helps to break down the barriers between academic silos common in broader scholarship (Arnold 2012). Future directions for the discipline are likely to focus on inclusive research practices, questioning pathologizing conceptualizations of autism and thus being the pivot for improved quality of life for all autistic persons.

See Also

- [Autism Theory](#)
- [Double Empathy](#)

- England and Autism
- Monotropism: An Interest-Based Account of Autism
- Neurodiversity
- Pathological Demand Avoidance (PDA)
- Personal Perspectives on Autism
- Theoretical Models and Autism

References and Reading

- All Party Parliamentary Group on Autism. (2019). *The Autism Act, 10 years on: A report from the All Party Parliamentary Group on Autism on understanding, services and support for autistic people and their families in England* (Online report). Retrieved from: <https://www.autism.org.uk/get-involved/media-centre/news/2019-09-09-not-enough-campaign.aspx>. Accessed 12 Sept 2019.
- Alvares, G., Bebbington, K., Cleary, D., Evans, K., Glassom, E., Evans, K., ... Whitehouse, A. (2020). The misnomer of 'high functioning autism': Intelligence is an imprecise predictor of functional abilities at diagnosis. *Autism*, 24(1), 221–232.
- Andrews, T., Hodge, N., & Redmore, N. (2019). The potential of the fractions of lifeworld for inclusive qualitative inquiry in the third space. *The International Journal of Inclusive Education*, 1. <https://doi.org/10.1080/13603116.2019.1642398>.
- Angell, A., & Solomon, O. (2017). "If I was a different ethnicity, would she treat me the same?": Latino parents' experiences obtaining autism services. *Disability & Society*, 32(8), 1142–1164.
- Arnold, L. (2012). Introduction. *Autonomy, the Critical Journal of Interdisciplinary Autism Studies*, 1(1).
- Arnold, L. (2017). A brief history of "neurodiversity" as a concept and perhaps a movement. *Autonomy, the Critical Journal of Interdisciplinary Autism Studies*, 1(5).
- Barnes, C. (2008). *Disability and the Academy: a British perspective* (Conference paper). Retrieved from: <https://disability-studies.leeds.ac.uk/wp-content/uploads/sites/40/library/Barnes-Whats-the-point.pdf>. Accessed 18 Oct 2019.
- Berghs, M., Atkin, K., Hatton, C., & Thomas, C. (2019). Do disabled people need a stronger social model: A social model of human rights? *Disability & Society*, 34(7–8), 1034–1039.
- Bertilsdotter-Rosqvist, H. (2019). Doing things together: Exploring meanings of different forms of sociality among autistic people in an autistic work space. *European Journal of Disability Research*, 13(3), 168–178.
- Bertilsdotter-Rosqvist, H., Kourti, M., Jackson-Perry, D., Brownlow, C., Fletcher, K., Bendelman, D., & O'Dell, L. (2019). Doing it differently: Emancipatory autism studies within a neurodiverse academic space. *Disability & Society*, 34(7–8), 1082–1101.
- Bolton, J. (2018). With the silence of a thousand cries: Extremes of autistic advocacy. *Disability & Society*, 33(6), 980–984.
- Campbell, F. (2008). Exploring internalized ableism using critical race theory. *Disability & Society*, 23(2), 151–162.
- Cassidy, S., Bradley, L., Shaw, R., & Baron-Cohen, S. (2018). Risk markers for suicidality in autistic adults. *Molecular Autism*, 9, 42. <https://doi.org/10.1186/s13229-018-0226-4>.
- Chown, N. (2017). *Understanding and evaluating autism theory*. London: Jessica Kingsley Publishers.
- Chown, N. (2019a). Neurodiversity. In F. Volkmar (Ed.), *Encyclopaedia of autism spectrum disorders*. New York: Springer Nature.
- Chown, N. (2019b). Who benefits from autism research? And to what extent is it participatory and/or emancipatory?: A brief follow-up to Pellicano, Dinsmore and Charman (2014). *Autism Policy and Practice*, 2(1), 93–95.
- Chown, N., Robinson, J., Beardon, L., Downing, J., Hughes, L., Leatherland, J., ... & MacGregor, D. (2017). Improving research about us, with us: A draft framework for inclusive autism research. *Disability & Society*, 32(5), 720–734.
- Davidson, J., & Orsini, M. (2013). *Worlds of autism: Across the spectrum of neurological difference*. Minneapolis: University of Minnesota Press.
- Dawson, M. (2004). *The misbehaviour of behaviourists: ethical challenges to the autism-ABA industry* (Online blog). Retrieved from: http://www.sentex.net/~nexus23/naa_aba.html. Accessed 12 Aug 2019.
- Den Houting, J. (2019). Neurodiversity: An insider's perspective. *Autism*, 23(2), 271–273.
- Evans, J. (2013). *Feminist theory today: An introduction to second wave feminism*. London: SAGE.
- Fletcher-Watson, S., & Happé, F. (2019). In 2nd (Ed.), *Autism: A new introduction to psychological theory and current debate*. Abingdon-on-Thames: Routledge.
- Fletcher-Watson, S., Adams, J., Brook, K., Charman, T., Crane, L., Cusack, J., ... & Pellicano, E. (2019). Making the future together: Shaping autism research through meaningful participation. *Autism*, 23(4), 943–953.
- Goodley, D. (2011). *Disability studies: An interdisciplinary introduction*. London: SAGE.
- Gould, J., & Ashton-Smith, J. (2011). Missed diagnosis or misdiagnosis? Girls and women on the autism spectrum. *Good Autism Practice*, 12(1), 34–41.
- Grinker, R. (2020). Autism, "stigma," disability a shifting historical terrain. *Current Anthropology*, 61(21). <https://doi.org/10.1086/705748>.
- Guest, E. (2020). Autism from different points of view: Two sides of the same coin. *Disability & Society*, 35(1), 156–162.
- Happé, F. (2011). Criteria, categories, and continua: Autism and related disorders in DSM-5. *American Academy of Child and Adolescent Psychiatry*, 50(6), 540–542.

- Harte, C. (2019). *Reframing compliance: Exposing violence within applied behaviour analysis*. Masters, City University of Seattle.
- Hassiotis, A., Poppe, M., Strydom, A., & Vickerstaff, V. (2018). Clinical outcomes of staff training in positive behaviour support to reduce challenging behaviour in adults with intellectual disability: Cluster randomised controlled trial. *The British Journal of Psychiatry*, 212(3), 161–168.
- Kupferstein, H. (2018). Evidence of increased PTSD symptoms in autistics exposed to applied behaviour analysis. *Advances in Autism*, 4(1), 19–29.
- Leatherland, J. (2018). *Understanding how autistic pupils experience secondary school: Autism criteria, theory and FAME™*. Ph.D., Sheffield Hallam University.
- Macleod, K., Causton, J., Radel, M., & Radel, P. (2017). Rethinking the individualised education plan process: Voices from the other side of the table. *Disability & Society*, 32(3), 381–400.
- Macloud, A. (2019). Interpretative phenomenological analysis (IPA) as a tool for participatory research within critical autism studies: A systematic review. *Research in Autism Spectrum Disorders*, 64(2019), 49–62.
- Maddox, B., Trubanova, A., & White, S. (2017). Untended wounds: Non-suicidal self-injury in adults with autism spectrum disorder. *Autism*, 21(4), 412–422.
- McGuire, A. (2015). “Life worth defending”: Biopolitical frames of terror in the war on autism. In S. Tremain (Ed.), *Foucault and the government of disability: Enlarged and revised edition* (pp. 350–371). Ann Arbor: University of Michigan Press.
- McGuire, A. (2016). *War on autism*. Ann Arbor: University of Michigan Press.
- Milton, D. (2013). Reversing the vicious circle of psycho-emotional disablism in the education of autistic people. In P. Banerjee, R. Barrie, & M. Hand (Eds.), *Championing research, educating professionals: How compatible are elitism, inclusion and social justice?* (pp. 127–134). Birmingham: University of Birmingham.
- Milton, D. (2017). *A mismatch of salience: Explorations of the nature of autism from theory to practice*. Hove: Pavilion Publishing and Media Ltd.
- Milton, D. (2018). *Pathological Demand Avoidance (PDA) and alternative explanations: a critical overview* (Conference paper). Retrieved from: <https://kar.kent.ac.uk/67064/1/PDA%20and%20alternative%20explanations.pdf>. Accessed 12 Aug 2019.
- Milton, D., & Lyte. (2012). The normalisation agenda and the psycho-emotional disablement of autistic people. *Autonomy, the Critical Journal of Interdisciplinary Autism Studies*, 1(1).
- Milton, D., & Moon, L. (2012). And that, Damian, is what I call life-changing’: Findings from an action research project involving autistic adults in an on-line sociology study group. *Good Autism Practice*, 13(2), 32–39.
- Milton, D., Kapp, S., Bovell, V., Timimi, S., & Russell, G. (2019). Deconstructing diagnosis: Multi-disciplinary perspectives on a diagnostic tool. *Autonomy, the Critical Journal of Interdisciplinary Autism Studies*, 1(6).
- Moore, A. (2019, June 04). *Pathological demand avoidance: What and who are being pathologized and in whose interests?* Paper presented at Participatory Autism Research Collective (PARC) critical autism studies conference 2019, PARC, London
- Nicolaidis, C., Raymaker, D., Kapp, S., Baggs, A., Ashkenazy, E., McDonald, K., ... Joyce, A. (2019). The AASPIRE practice-based guidelines for the inclusion of autistic adults in research as co-researchers and study participants. *Autism*, 23(8), 2007–2019.
- Oliver, M. (2013). The social model of disability: Thirty years on. *Disability & Society*, 28(7), 1024–1026.
- Pellicano, E., Dinsmore, A., & Charman, T. (2014). What should autism research focus upon? Community views and priorities from the United Kingdom. *Autism*, 18(7), 756–770.
- Pickles, A., Le Couteur, A., Leadbitter, K., Salomone, K., Cole-Fletcher, R., Tobin, H., Gammer, I., ... Green, J. (2016). Parent-mediated social communication therapy for young children with autism (PACT): Long-term follow-up of a randomised controlled trial. *Lancet*, 388(2016), 2501–2509.
- Pountney, O. (2018). *Untangling the knots of neuroqueer intersectionality*. A presentation at Autscope 2018. Retrieved from: <http://www.autscope.org/2018/programme/handouts/Untangling%20the%20knots%20of%20neuroqueer%20intersectionality.pdf>. Accessed 19 Aug 2019.
- Ridout, S. (2014). More than picture-making: Reflecting on collage as a narrative tool for opening discourse on the involvement of autistics in autism research. *Autonomy, the Critical Journal of Interdisciplinary Autism Studies*, 1(3).
- Ridout, S. (2016). Well-being and creative methodologies as a tool for communicating with health and social care practitioners. In D. Milton & N. Martin (Eds.), *Autism and intellectual disability in adults* (Vol. 1, pp. 45–47). Hove: Pavilion Publishing and Media Limited.
- Rosenblatt, A. (2018). Autism, advocacy organizations, and past injustice. *Disability Studies Quarterly*, 38(4).
- Runswick-Cole, K. (2014). ‘Us’ and ‘them’: The limits and possibilities of a ‘politics of neurodiversity’ in neoliberal times. *Disability & Society*, 29(7), 1117–1129.
- Runswick-Cole, K., Mallett, R., & Timimi, S. (2016). *Re-thinking autism: Diagnosis identity and equality*. London: Jessica Kingsley Publishers.
- Saxe, A. (2017). The theory of intersectionality: A new lens for understanding the barriers faced by autistic women. *Canadian Journal of Disability Studies*, 6(4), 153–178.
- Shakespeare, T. (2017). *Disability: The basics*. Abingdon: Routledge.
- Sinclair, J. (2013[1999]). Why I dislike “person first” language. *Autonomy, the Critical Journal of Interdisciplinary Autism Studies*, 1(2).

- Stewart, C. (2012). Where can we be what we are?': The experiences of girls with Asperger syndrome and their mothers. *Good Autism Practice*, 13(1), 40–48.
- Strand, L. (2017). Charting relations between intersectionality theory and the neurodiversity paradigm. *Disability Studies Quarterly*, 37(2).
- Thomas, C. (1999). *Female forms: Experiencing and understanding disability* (1st ed.). Buckingham: Open University Press.
- Waldock, K. E. (2019). Commentary on “thinking differently? Autism and quality of life”. *Tizard Learning Disability Review*, 24(2), 77–81.
- Waltz, M. (2007). The relationship of ethics to quality: A particular case of research in autism. *International Journal of Research & Method in Education*, 30(3), 353–361.
- Waltz, M. (2008). Autism = Death: The social and medical impact of a catastrophic medical model of autistic spectrum disorders. *Journal of Popular Narrative Media*, 1(1), 13–24.
- Waltz, M. (2012). Images and narratives of autism within charity discourses. *Disability & Society*, 27(2), 219–233.
- Waltz, M. (2014). Worlds of autism: Across the spectrum of neurological difference. *Disability & Society*, 29(8), 1337–1338.
- Williams, A. (2018). Autonomously autistic: Exposing the locus of autistic pathology. *Canadian Journal of Disability Studies*, 7(2), 60–82.
- Wood, R. (2019). Autism, intense interests and support in school: From wasted efforts to shared understandings. *Educational Review*, 1. <https://doi.org/10.1080/00131911.2019.1566213>.
- Woods, R. (2017a). Pathological demand avoidance: My thoughts on looping effects and commodification of autism. *Disability & Society*, 32(5), 753–758.
- Woods, R. (2017b). Exploring how the social model of disability can be reinvigorated for autism: In response to Jonathan Levitt. *Disability & Society*, 32(7), 1090–1095.
- Woods, R., & Waltz, M. (2019). The strength of autistic expertise and its implications for autism knowledge production: A response to Damian Milton. *Autonomy, the Critical Journal of Interdisciplinary Autism Studies*, 1(6).
- Woods, R., Milton, D., Arnold, L., & Graby, S. (2018). Redefining critical autism studies: A more inclusive interpretation. *Disability & Society*, 33(6), 974–979.
- Yergeau, M. (2010). Circle wars: Reshaping the typical autism essay. *Disability Studies Quarterly*, 30(1).
- Yergeau, M. (2013). Clinically significant disturbance: On theorists who theorize theory of mind. *Disability Studies Quarterly*, 33(4).
- Yergeau, M. (2017). *Authoring autism: On rhetoric and neurological queerness*. Durham: Duke University Press.

Cronbach's Alpha

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Synonyms

Coefficient alpha

Definition

Cronbach's alpha (α) is an estimate of reliability, specifically the internal consistency, of a test or scale. It is widely used in psychological test construction and interpretation (Cortina 1993). When internal consistency is present in a test, it is interpretable (Cronbach 1951). Cronbach's alpha seeks to measure how closely test items are related to one another and thus measuring the same construct.

The formula for Cronbach's alpha is as follows:

$$\alpha = (n/(n - 1)) \times (1 - (\sum s_i^2 / s_T^2))$$

where n is the number of items, s_i^2 is the variance of the i th item, and s_T^2 is the total score variance (Cronbach 1951).

When test items are closely related to one another, Cronbach's alpha will be closer to 1, and when test items are not closely related to one another, Cronbach's alpha will be closer to 0. An α of 0.90–0.95 is desirable for clinical interpretation of tests (Bland and Altman 1997).

References and Reading

- Bland, J., & Altman, D. (1997). Statistics notes: Cronbach's alpha. *British Medical Journal*, 314, 572.
- Cicchetti, D. V., Lord, C., et al. (2008). Reliability of the ADI-R: Multiple examiners evaluate a single case. *Journal of Autism & Developmental Disorders*, 38(4), 764–770.

- Cortina, J. (1993). What is coefficient alpha?: An examination of theory and applications. *Journal of Applied Psychology*, 78, 98–104.
- Cronbach, L. (1951). Coefficient alpha and the internal structure of tests. *Psychometrika*, 16, 297–334.
- Klin, A., Lang, J., et al. (2000). Brief report: Interrater reliability of clinical diagnosis and DSM-IV criteria for autistic disorder: Results of the DSM-IV autism field trial. *Journal of Autism & Developmental Disorders*, 30(2), 163–167.
- Russell, P. S. S., Daniel, A., et al. (2010). Diagnostic accuracy, reliability and validity of childhood autism rating scale in India. *World Journal of Pediatrics*, 6(2), 141–147.

Cross Eye

- [Strabismus](#)

Cross-Training

- [Role Release](#)

Crystallized Intelligence

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Definition

Crystallized intelligence (abbreviated Gc) is reflected in a person's general knowledge, vocabulary, and reasoning based on acquired information. It is contrasted with fluid intelligence (see ► [“Fluid Intelligence”](#)) as one of the two factors of general intelligence first proposed by Cattell (1971). Crystallized intelligence is conceptualized as the product of experience, both cultural and educational, in interaction with fluid intelligence; people with higher levels of fluid intelligence will

generally amass learnt information faster, allowing higher crystallized intelligence. Crystallized intelligence is measured by tests such as vocabulary and general knowledge type assessments.

In ASD, the profile of better performance than verbal IQ subtest scores may reflect, in part, differences between fluid and crystallized intelligence. It has been suggested that standard IQ assessments underestimate intelligence compared to pure fluid assessments such as Raven's Progressive Matrices (Dawson et al. 2007). The acquisition of skills and knowledge, as measured by crystallized intelligence tests, may rely in part on socially mediated learning and be hampered in ASD by problems of social cognition (Scheuffgen et al. 2000). However, Bölte et al. (2009) suggested that discrepancies were small among higher functioning ASD groups and recommended use of Wechsler intelligence scales for comprehensive assessment of abilities in ASD, perhaps supplemented by Raven's Matrices for lower functioning individuals. Recent estimates from epidemiological samples suggest that low measured IQ is not as common among people with ASD as previous figures suggested; Charman et al. (2011) reported just over half their community sample of young people with ASD had IQ below 70, with less than a fifth having IQ below 50, and almost a third of the sample having IQ above 85. Improvements in understanding, recognition, and intervention may have improved accessibility of appropriate education for children with autism, allowing more individuals to come closer to fulfilling their learning potential.

See Also

- [Fluid Intelligence](#)
- [Vocabulary](#)

References and Reading

- Bölte, S., Dziobek, I., & Poustka, F. (2009). Brief report: The level and nature of autistic intelligence revisited. *Journal of Autism and Developmental Disorders*, 39, 678–682.

- Cattell, R. B. (1971). *Abilities: Their structure, growth, and action*. New York: Houghton Mifflin.
- Charman, T., Pickles, A., Simonoff, E., Chandler, S., Loucas, T., & Baird, G. (2011). IQ in children with autism spectrum disorders: Data from the Special Needs and Autism Project (SNAP). *Psychological Medicine*, 41, 619–627.
- Dawson, M., Soulières, I., Gernsbacher, M. A., & Motttron, L. (2007). The level and nature of autistic intelligence. *Psychological Science*, 18, 657–662.
- Scheuffgen, K., Happé, F., Anderson, M., & Frith, U. (2000). High “Intelligence”, low “IQ”? Speed of processing and measured IQ in children with autism. *Development and Psychopathology*, 12, 83–90.

CSBQ (Children's Social Behavior Questionnaire)

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Abbreviations

ASD Autism spectrum disorders

Synonyms

[Children's social behavior questionnaire](#)

Description

Children with an autism spectrum disorder (ASD) form a heterogeneous group. The Children's Social Behavior Questionnaire (CSBQ) charts this heterogeneous behavior through 49 items rated by parents. These items are scored on a three-point scale ranging from “does not apply or occur” to “clearly or often applies.” Items refer directly to DSM-IV criteria for autism but also represent less severe variations of these criteria as well as ASD-associated problems,

such as executive functioning problems and disruptive behavior in difficult social situations. The 49 items aggregate into six problem dimensions which form the subscales of the CSBQ. Children with ASD tend to vary in the extent to which the problems captured by these subscales are present; therefore, each child has his or her own problem profile.

Subscale names are abbreviated as social, tuned, understanding, orientation, change, and stereotypies and capture the following problems:

- First, the subscale “social” measures aspects related to social contact, social interest, and social reciprocity. It refers to both initiation of contact and reaction to social overtures by others.
- Second, the subscale “tuned” measures behavior related to daily adaptation to social situations. Examples of items are as follows: “overreacts to everything and everyone” and “does not know when to stop, goes on and on about things.” While instances of such behavior may also be seen in typically developing children, the tuned subscale depicts the more extreme form manifested by children with ASD.
- Third, children with high scores on the subscale “understanding” have difficulties in understanding the rules of communication and the social use of language (pragmatic communication). Sample items are as follows: “does not understand jokes” and “is extremely naive; believes anything you say.”
- Fourth, the orientation subscale refers to the ability to keep an overview of what goes on, what one is doing, and where one is headed. Sample items are as follows: “gets lost easily” and “has difficulties doing two things simultaneously.”
- Fifth, the items of the subscale “change” measure behavior when confronted with changes, expressed as fear, panicking, resistance, and freezing.

Sixth, the items from the stereotypies subscale tap the various repetitive sensorimotor behaviors seen in children with ASD such as making odd

movements with fingers and hands, smelling objects, and being unusually sensitive to certain sounds.

Both total CSBQ score and individual subscale scores can be used. The profile of scores on the six dimensions simultaneously reveals which problem domains are predominantly present in the child and which problem domains are less prominent. The subscales “orientation” and “tuned” are not specific for ASD, with similar scores in children with ADHD. Thus, if the aim is to focus specifically on the most differentiating ASD core symptoms, scores on the subscales social, understanding, change, and stereotypies are summed.

Historical Background

Parent-report questionnaires are cost- and time-efficient assessment tools in health practice and research. The research program to develop the CSBQ started for two reasons. First, the goal was to quantify the different dimensions on which children with an ASD tend to differ, thus tapping the heterogeneity in this group. The second goal was to specifically include the milder part of the ASD score distribution along with the more severe autistic behaviors in one and the same instrument, thus tapping the full ASD spectrum. The field lacked a questionnaire that tapped not only the severe behaviors seen in autism proper but also these milder and subtler variants of ASD. The inclusion of these (in addition to the more severe autistic behaviors) in an instrument would be helpful in the diagnostic process of ASD, and even in case of a diagnosis outside the ASD spectrum, knowing the presence of such mild problems would be useful. Moreover, the field lacked a questionnaire that summarized the heterogeneous problems seen in ASD by a number of meaningful problem dimensions along which children with an ASD show variation. The peaks and troughs on a profile of different ASD problems would give insight into children's specific clinical presentation within ASD and provide clues for intervention.

With these ideas in mind, the CSBQ was developed about 15 years ago (Luteijn et al. 1998).

In its original form, the CSBQ contained 96 items. Revised in 2006, the 49-item CSBQ gained in specificity by removing problem items that were only marginally characteristic of ASD (Hartman et al. 2006). Instrument development is still in progress. For example, an adult counterpart of the CSBQ, the Adult Social Behavior Questionnaire (ASBQ), with both a self- and other-report version, has been developed and currently is being validated (Horwitz et al. 2012).

Psychometric Data

CSBQ scores are interpreted in relation to norm groups. Norms are available for boys and girls and for six age groups between ages 4 and 18 for children from the general population. Gender-specific norms are also available for general psychiatric child (4–11) and adolescent (12–18) population. From the general psychiatric population, separate norms are available for three subgroups: ADHD, PDD-NOS, and higher functioning autism, respectively. Further, there are norms for children (4–11) and adolescents (12–18) with mild mental retardation and for children (4–18) with moderate mental retardation. These groups are split up further into separate norms for children with both mental retardation and ASD (Hartman et al. 2007).

Several factor analytic studies with varying item pools indicate that the six ASD problem dimensions that are differentiated by the CSBQ are firmly anchored in the data (Hartman et al. 2006; Luteijn et al. 2000a). That is, the ASD problem dimensions emerging from the original item pool of 96 items and from the revised version with the 49 items are highly similar. Additionally, the ASD problem dimensions emerged from a simultaneous factor analysis of the CSBQ with Child Behavior Checklist items. The consistency in factor structure speaks to the construct validity of the problem dimensions.

Multiple studies have shown that the CSBQ has good psychometric properties with regard to test-retest and interrater reliability, internal consistency of the scales (all reliability indices at least .75), and good criterion validity both for

high-functioning children and for children with mild to moderate mental retardation (de Bildt et al. 2005, 2009; Hartman et al. 2006; Luteijn et al. 2000a, b). The CSBQ differentiates between autism and PDD-NOS on the one hand and PDD-NOS and ADHD on the other hand, with decreasing scores for these three conditions, respectively (Hartman et al. 2006). The differences between these diagnostic groups come mostly from the four core autism problem dimensions, i.e., social, understanding, change, and stereotypies. Note however that a combined diagnosis of ADHD and ASD is accompanied by significantly higher scores on the tuned and orientation dimensions than a separate diagnosis of either ADHD or ASD, suggesting that each condition has its own impact on the behaviors measured by these scales (Hartman et al. 2006). Further indices of criterion validity are an association of .75 for the CSBQ with the Autism Behavior Checklist (Krug et al. 1980; Hartman et al. 2007) and associations of around $-.40$ for relevant subscales with Theory of Mind ability (Blijd-Hoogewys et al. 2008). For children with mental retardation, the CSBQ distinguishes between the profiles of the PDD group and the non-PDD group (Hartman et al. 2006). A second study in children with mental retardation showed that the contribution of the CSBQ to a classification of ASD was most specific for the problem dimensions "contact" and "stereotyped," with high coherence with classification methods of ADI-R, ADOS, and clinical DSM-IV-TR classifications.

For research purposes, the instrument has proven useful in genetic (Nijmeijer et al. 2010a, b, 2011), neurocognitive (Geurts et al. 2008; Rommelse et al. 2009), and behavioral (de Bildt et al. 2005; Luteijn et al. 2000c) studies, thus adding to its validity. For example, Nijmeijer et al. showed that the COMT Val/Val genotype interacted with maternal smoking during pregnancy in increasing stereotyped behavior in two independent samples. As a second example, in a sample of 816 children from ADHD and control families, executive functioning and motor impairments were correlated and cross-correlated in siblings to autistic traits, suggesting that ADHD and ASD may possibly share familial/genetic EF and motor deficits. The CSBQ has also aided in characterizing (subthreshold) ASD problems in

populations other than ASD such as ADHD (Nijmeijer et al. 2008) and delinquent groups (Geluk et al. 2011; 't Hart-Kerkhoffs et al. 2009). Finally, early childhood assessments by community pediatric professionals were predictive of CSBQ ratings during adolescence (Jaspers et al. 2011).

Clinical Uses

The CSBQ may be used as a signaling, screening, or describing instrument for children aged 4–18 and for all levels of functioning. Scores on the CSBQ can be interpreted relative to norms based on the general populations or 3 norms based on general child psychiatric outpatient groups as well as on specific psychiatric groups such as autism or ADHD. Norms vary according to age, gender, and level of functioning.

In the orienting stage of the diagnostic procedure, the score profile of the six subscales contributes to identifying whether or not the problem behavior of the child is suggestive of ASD and whether further diagnostic assessments should be focused on ASD.

Additionally, the CSBQ may be of value further in the diagnostic process, clearly not in diagnosing ASD (which should be based on a more extensive diagnostic procedure including observation and interviewing) but in complementing these methods by adding additional information about the child's clinical profile of problems along the six CSBQ problem domains. This profile reveals the child's major ASD problem areas as well as the domains that follow normative development. This may add to the diagnostic process as well as direct treatment choice.

In children with a diagnosis outside the ASD spectrum, the CSBQ may reveal the presence of subthreshold ASD problems which may be helpful for choice of treatment.

See Also

- [Behavior Rating Scale \(BRS\)](#)
- [Behavioral Assessment](#)
- [Pragmatic Communication](#)

- [Spectrum/Continuum of Autism](#)
- [Stereotypic Behavior](#)

References and Reading

- ^t Hart-Kerkhoffs, L. A., Jansen, L. M., Doreleijers, T. A., Vermeiren, R. A., Minderaa, R. B., & Hartman, C. A. (2009). Autism spectrum disorder symptoms in juvenile suspects of sex offenses. *The Journal of Clinical Psychiatry*, 70, 266–272.
- Blijd-Hoogewys, E. M., van Geert, P. L., Serra, M., & Minderaa, R. B. (2008). Measuring theory of mind in children. Psychometric properties of the ToM storybooks. *Journal of Autism and Developmental Disorders*, 38, 1907–1930.
- de Bildt, A., Serra, M., Luteijn, E., Kraijer, D., Sytema, S., & Minderaa, R. B. (2005). Social skills in children with intellectual disabilities with and without autism. *Journal of Intellectual Disability Research*, 49, 317–328.
- de Bildt, A., Mulder, E. J., Hoekstra, P. J., Van Lang, N. D., Minderaa, R. B., & Hartman, C. A. (2009). Validity of the Children's Social Behavior Questionnaire (CSBQ) in children with mental retardation: Comparing the CSBQ with ADI-R, ADOS, and clinical DSM-IV-TR classification. *Journal of Autism and Developmental Disorders*, 39, 1464–1470.
- Geluk, C. A. M. L., Jansen, L. M. C., Vermeiren, R., Doreleijers, T. A. H., van Domburgh, L., de Bildt, A., et al. (2011). Autistic symptoms in childhood arrestees: Longitudinal association with delinquent behavior. *Journal of Child Psychology and Psychiatry*, 53, 160–167.
- Geurts, H. M., Luman, M., & van Meel, C. S. (2008). What's in a game: The effect of social motivation on interference control in boys with ADHD and autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 49, 848–857.
- Hartman, C. A., Luteijn, E., Serra, M., & Minderaa, R. B. (2006). Refinement of the Children's social behavior questionnaire (CSBQ): An instrument that describes the diverse problems seen in milder forms of PDD. *Journal of Autism and Developmental Disorders*, 36, 325–342.
- Hartman, C. A., Luteijn, E., Moorlag, A., de Bildt, A., & Minderaa, R. (2007). *Manual for the CSBQ [Handleiding voor de VISK]*. Amsterdam: Harcourt.
- Horwitz, E. H., Schoevers, R. A., Ketelaars, C. E. J., Kan, C. C., van Lammeren, A. M. D. N., Meesters, Y., et al. (2012). Autism Spectrum Disorders (ASD) in adults assessed by self and other report: Psychometric properties and validity of the Adult Social Behavior Questionnaire (ASBQ) (submitted).
- Jaspers, M., de Winter, A. F., Buitelaar, J. K., Verhulst, F. C., Reijneveld, S. A., & Hartman, C. A. (2011). Early childhood assessments of community pediatric professionals predict autism spectrum and attention deficit hyperactivity problems. *Journal of Abnormal Child Psychology*.
- Krug, D. A., Arick, J. R., & Almond, P. J. (1980). *Autism screening instrument for educational planning*. Portland: ASIEP Education.
- Luteijn, E. F., Jackson, A. E., Volkmar, F. R., & Minderaa, R. B. (1998). The development of the Children's Social Behavior Questionnaire: Preliminary data. *Journal of Autism and Developmental Disorders*, 28, 559–565.
- Luteijn, E. F., Luteijn, F., Jackson, A. E., Volkmar, F. R., & Minderaa, R. B. (2000a). The Children's Social Behavior Questionnaire for milder variants of PDD problems: Evaluation of the psychometric characteristics. *Journal of Autism and Developmental Disorders*, 30, 317–330.
- Luteijn, E., Luteijn, F., Jackson, S., Volkmar, F., & Minderaa, R. (2000b). The children's social behavior questionnaire for milder variants of PDD problems: Evaluation of the psychometric characteristics. *Journal of Autism and Developmental Disorders*, 30, 317–330.
- Luteijn, E. F., Serra, M., Jackson, A. E., Steenhuis, M. P., Althaus, M., Volkmar, F. R., et al. (2000c). How unspecified are disorders of children with a Pervasive Developmental Disorder Not otherwise Specified? A study of social problems in children with PDD-NOS and ADHD. *European Child & Adolescent Psychiatry*, 9, 168–179.
- Nijmeijer, J. S., Hoekstra, P. J., Minderaa, R. B., Buitelaar, J. K., Altink, M. E., Buschgens, C. J., et al. (2008). PDD symptoms in ADHD, an independent familial trait? *Journal of Abnormal Child Psychology*, 37, 443–453.
- Nijmeijer, J. S., Arias-Vasquez, A., Rommelse, N. J., Altink, M. E., Anney, R. J., Asherson, P., et al. (2010a). Identifying loci for the overlap between ADHD and ASD using a genome-wide QTL linkage approach. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49, 675–685.
- Nijmeijer, J. S., Hartman, C. A., Rommelse, N. J., Altink, M. E., Buschgens, C. J. M., Fliers, E., et al. (2010b). Perinatal risk factors interacting with catechol O-methyltransferase and the serotonin transporter gene predict ASD symptoms in children with ADH. *Journal of Child Psychology and Psychiatry*, 51, 1242–1250.
- Nijmeijer, J. S., Arias-Vásquez, A., Lambregts-Rommelse, N. N. J., Altink, M. E., Buschgens, C. J. M., Fliers, E. A., et al. (2011). QTL Linkage for ASD symptoms in ADHD: Significant locus on chromosome 7q11. Submitted.
- Rommelse, N., Altink, M. E., Fliers, E. A., Martin, N. C., Buschgens, C. J., Hartman, C. A., et al. (2009). Comorbid problems in ADHD: Degree of association, shared endophenotypes, and formation of distinct subtypes. Implications for a future DSM. *Journal of Abnormal Child Psychology*, 37, 793–804.

CSBS

- [Communication and Symbolic Behavior Scale](#)

CSBS-DP

- ▶ [Communication and Symbolic Behavior Scale](#)
- ▶ [Infant/Toddler Checklist](#)

CSF

- ▶ [Cerebrospinal Fluid](#)

CSF 5-HIAA

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Synonyms

[Cerebrospinal fluid 5-hydroxyindoleacetic acid](#)

Definition

Cerebrospinal fluid (CSF) levels of 5-hydroxyindoleacetic acid (5-HIAA), the principal metabolic end product produced from the neurotransmitter serotonin, are measured to provide a global index of serotonin production in the brain. Taken together, studies of CSF 5-HIAA in autism indicate that group mean levels are not altered in autism.

See Also

- ▶ [Neurotransmitter](#)
- ▶ [Serotonin](#)

References and Reading

Dhondt, J. L. (2004). Difficulties in establishing reference intervals for special fluids: The example of 5-hydroxyindoleacetic acid and homovanillic acid in

cerebrospinal fluid. *Clinical Chemistry and Laboratory Medicine*, 42(7), 833–841.

Lam, K. S., Aman, M. G., & Arnold, L. E. (2006). Neurochemical correlates of autistic disorder: A review of the literature. *Research in Developmental Disabilities*, 27(3), 254–289.

Narayan, M., Srinath, S., Anderson, G. M., & Meundi, D. B. (1993). Cerebrospinal fluid levels of homovanillic acid and 5-hydroxyindoleacetic acid in autism. *Biological Psychiatry*, 33(8–9), 630–635.

CSF HVA

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Synonyms

[Cerebrospinal fluid homovanillic acid](#)

Definition

Cerebrospinal fluid (CSF) levels of homovanillic acid (HVA), the principal metabolic end product produced from the neurotransmitter dopamine, are measured to provide a global index of dopamine production in the brain. Taken together, studies of CSF HVA in autism indicate that group mean levels are not altered in autism.

See Also

- ▶ [Dopamine](#)
- ▶ [Neurotransmitter](#)

References and Reading

Dhondt, J. L. (2004). Difficulties in establishing reference intervals for special fluids: The example of 5-hydroxyindoleacetic acid and homovanillic acid in cerebrospinal fluid. *Clinical Chemistry and Laboratory Medicine*, 42(7), 833–841.

Lam, K. S., Aman, M. G., & Arnold, L. E. (2006). Neurochemical correlates of autistic disorder: A review of the

literature. *Research in Developmental Disabilities*, 27(3), 254–289.

- Narayan, M., & Anderson, G. M. (1993). CSF HVA in autism (in reply). *Biological Psychiatry*, 32, 746–747.
- Narayan, M., Srinath, S., Anderson, G. M., & Meundi, D. B. (1993). Cerebrospinal fluid levels of homovanillic acid and 5-hydroxyindoleacetic acid in autism. *Biological Psychiatry*, 33(8–9), 630–635.

CT

► Computed Tomography

Culturally Responsive Assessment of Language and the Challenge Within Standardized Tests

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Definition

Cultural responsiveness is an aspect of quality assessment and education that requires being aware of the cultural differences that may affect one's knowledge and consciously adapting to ensure that each child is being accurately assessed and is being taught in a way that is both relevant and effective with respect to their culture (Gay 2000).

Historical Background

Practitioner competence in the use of cultural responsiveness would result in quality assessment and indicate being aware of the cultural

differences that may affect performance, thus lead to consciously adapting evaluation protocols to ensure that each child is being accurately assessed in a way that is both accepting and respectful of their culture (Paul et al. 2018). Evaluation protocols that include assessment batteries that are not culturally responsive have the potential to overdiagnose language disorders in individuals who are culturally diverse (Paul et al. 2018). Culturally diverse children may have language differences, which are styles of language that are different from the standard language of one's culture, yet still follow a rule-governed style (Paul et al. 2018). If an individual has a language difference, it does not imply that this individual has impairment in the ability to learn language nor should they be diagnosed with a language disorder. An individual with a language difference has the capacity to learn the standard language after simply being exposed over time, whereas, individuals with language disorders have impairments that typically require intervention in order to meet social and academic expectations (Paul et al. 2018).

Alternatively, a lack of cultural responsiveness by practitioners can result in the delay of a diagnosis of some developmental disabilities such as Autism Spectrum Disorder. Those of different cultures may lack knowledge of the subtle cues or signs relating to Autism Spectrum Disorder (ASD) and view these changes as a normal process that occurs for all developing children (Danescu 1997). This can impact when or if the child is referred to professionals to then be assessed. For example, socioeconomic status and one's background does not dictate whether or not a child has autism, but may contribute to a physician's decision to screen for ASD (Gibson 2007). As a result, the socioeconomic status of children may cause a delayed diagnosis for certain individuals, especially for African American children. These children are 2.6 times less likely to receive a diagnosis of autism compared to their Caucasian peers (Mandell et al. 2007). Therefore, it is important for practitioners to be aware of these potential culturally related biases and be prepared to proactively reach out to the community to assist in reducing the disparity of diagnosis that occurs (Wiggins et al. 2019). Further, the disparity of

diagnosis of autism in culturally diverse individuals can likely be further confounded when utilizing assessment protocols that are inconsistent with their responsivity to diverse cultures.

In an effort to better understand the degree of cultural responsiveness of a few commonly used test batteries for the development of language, and potentially as they are used as part of a comprehensive evaluation for possible diagnosis of ASD, this review of literature explores the following instruments: Communication and Symbolic Behavior Scale (CSBS), Diagnostic Evaluation of Language Variation-Norm Referenced (DELV-NR), Clinical Evaluation of Language Fundamentals Preschool-2 (CELF-P2), Token Test for Children-Second Edition (TTFC-2), and the Preschool Language Scale-Fifth Edition (PLS-5). The question of the likelihood of overdiagnosing culturally diverse children with language disorders when using these test batteries is explored. The information provided will serve as an initial step in providing knowledge to support use of cultural responsiveness by clinicians as they use these test batteries to assess the language skills of culturally diverse children and those who present with ASD.

Current Knowledge

CSBS

The Communication and Symbolic Behavior Scale assesses the social, communicative, and symbolic abilities of children. It was created for children between the ages of 8 months and 2 years. This assessment is comprised of the Caregiver Questionnaire which collects information about the child's communicative skills, the Behavior Sample, a play-centered method of collecting data that is videotaped for scoring, and the Caregiver Perception Rating form. This form is filled out by the caregiver after observing the Behavior Sample and collects information on how often the child typically exhibits the behavior revealed in the sample. This assessment battery includes toys and books to elicit spontaneous communication. The CSBS is useful in a natural environment to observe a child's play and communicative skills (Wetherby and Prizant 2003).

A shortened version of the CSBS is the CSBS DP: The Communication and Symbolic Behavior Scales Developmental Profile. This was created to be more efficient than the original version.

The CSBS DP has been identified as an assessment tool that can be used cross-culturally with relatively consistent outcomes. In 2016, there were no standardized tests to assess early social communication skills in South African (SA) children (Chambers et al. 2016). The scores of 67 English-speaking South African children on the CSBS DP Behavioral Sample were compared to the standardized scores. The researchers found that from 15 months of age there seemed to be little bias for, or against, SA subjects compared to those in the United States. Based on these results, the researchers concluded that the CSBS DP was culturally fair in assessing South African children from different cultural backgrounds (Chambers et al. 2016). However, the score on the Words cluster subtest revealed differences between children who were monolingual and those who spoke more than one language, with the children who spoke multiple languages scoring lower on the Words cluster section. Yet their scores were still within the limits of the assessment. The toddlers in the SA sample performed well on the gaze tasks, but not as consistently as those in the US groups. As stated previously, this finding may be because of a difference between the cultures being compared. It is possible that the children from the SA sample are more reserved than those from the US sample (Chambers et al. 2016).

Similarly, Roberts et al. (1997) suggest that the CSBS DP is helpful in detecting communication delays in children who are outside of the standardization sample. They assessed 93 African-American 1-year-old children, the majority of them living below poverty level. The results were similar to those of the standardized sample, with the exception of gestural means, which was lower than those of the children in the standardized sample. There are a number of reasons this could be, including differences in how the African-American culture uses gestures or the differences in parents' interaction. The overall results of this group were similar to those of the standardized sample, thus supporting that the

standardized sample is fairly representative cross-culturally (Roberts et al. 1997).

Alternatively, Tek and Landa (2012) argue that the CSBS may not be sensitive enough to cultural differences. When comparing 19 minority toddlers to 65 Caucasian toddlers, the CSBS Caregiver Questionnaire was used. The minority children in this study showed more atypical language and communication scores than their non-minority counterparts, especially on the CSBS Words and the CSBS Understanding of Words. The researchers concluded that minority parents may not view certain behaviors or delayed milestones as concerning, relating to a gap in scores. For example, in some Asian cultures, eye contact with elders is considered disrespectful and a lack of eye contact will result in a lower score on the CSBS Social Composite section. A cultural difference must be determined here so that a child is not wrongly scored. This study was largely made up of children from the upper middle class, independent of ethnicity, so it would be important to compare minorities from a lower class as well (Tek and Landa 2012). Overall, the CSBS is a promising tool for groups that it was not standardized on, and the research thus far shows that it is a culturally responsive assessment tool.

DELV-NR

The Diagnostic Evaluation of Language Variation-Norm Referenced (DELV-NR) is an assessment used to language skills within the domains of phonology, semantics, syntax, and pragmatics of children between the ages of 4 and 9 years, 11 months who speak English as their primary language yet a dialect that is considered varied from Mainstream American English (MAE) (Seymour et al. 2018, p. 1). The DELV-NR can be used to diagnose children with speech and language delays or disorders, and to differentiate delays and disorders from language differences that are seen in dialects other than MAE. Test materials for the administration of the DELV-NR include a stimulus book with colored images as well as a response form. Each domain of language is assessed using multiple subtests and administration of the entire test takes approximately 45 min. Scores can be used to determine

a percentile rank in each domain as well as to diagnose a language or speech disorder.

To limit the impact of cultural bias on the test results of culturally diverse children, the DELV-NR clearly distinguishes features of English that are found in every dialect from features that differ between English dialects and focuses only on the universal features of English dialects. By doing this, it creates a dialect neutral assessment that limits the ability for speaking a dialect other than MAE to impact test results. Therefore, results on the DELV-NR clearly distinguish between a language difference and a disorder. This assessment filled a gap in the available resources for the assessment of children who are culturally and dialectically diverse, and it addressed a significant weakness that is present in many other assessment materials.

Many assessments lack test materials that use only the aspects of language that are universal across all dialects of English. This can lead to struggles in distinguishing language differences from language disorders as many children who speak English dialects other than MAE receive low scores on these assessments. While children may provide responses that are grammatically correct in their dialect, it could result in lower scores if it does not match the desired response in MAE. This often leads to the overdiagnosis of dialectically diverse children, which could lead to unnecessary speech and language services being provided (Losen and Orfield 2002; Robinson and Norton 2012).

One study found that the DELV-NR was able to provide a detailed summary of children's language skills that were highly consistent with their spontaneous speech language samples. They also concluded that the DELV-NR can help improve the quality of assessment and diagnostic accuracy of language impairments in dialectically diverse children (Pearson et al. 2014). Additionally, because the DELV-NR was normed on 900 children who were representative of the demographic diversity of the general population of the United States based on the 2002 U.S. Census, its diagnostic criteria is relevant for speakers of diverse English dialects as well as speakers of MAE (Seymour et al. 2018, p. 109).

By establishing a test that focuses on using the aspects of language that are universal across all dialects of English, the DELV-NR is able to distinguish language differences from disorders with greater accuracy and minimize the overdiagnosis of language disorders in children (Pearson et al. 2014).

CELF-P2

The Clinical Evaluation of Language Fundamentals Preschool-2 assesses morphology, phonology, syntax, semantics, and pragmatics in children ages 3–6 years, 11 months. In this assessment, children respond to picture stimuli in an easel style book, by either verbally responding or pointing. Identifying a language disorder or delay that has a negative impact on a child's success in the classroom is a goal of this assessment. The normative sample of 1500 preschool aged children was representative of the United States population at the time of its creation in 2002. 62.1% of the normative sample was white while 37.9% was non-white. It is important to note although the normative sample would have changed since 2002, it is representative of the population at that time (Wiig et al. 2004).

The CELF-P2 UK is a version of this assessment used in the United Kingdom and it is one of the most widely used standardized assessments in that nation. Similar to the USA, it is maintained that children in the UK who come from disadvantaged backgrounds are more likely to have language delays than those from privileged communities (Ryan et al. 2016). Additionally, a Spanish version of the CELF-P2 has been developed. The norms for this battery were created based on a range of children from culturally, racially, and ethnically different backgrounds. Although not included in the CELF-P2, it is notable that in the fourth revision (CELF-4), there is a section in the manual discussing the influences of African American and Spanish cultures (Paslawski 2005).

Current research suggests the CELF-P2 as mostly culturally responsive. For example, children adopted from Eastern Europe between the ages of 1 and 4 years, 11 months were assessed on the CELF-P2 with Russian, Hungarian, or

Romanian as their first language. The scores of these children were within average range for both expressive and receptive language, displaying cultural relativity with minor language differences not effecting scores. Therefore, this assessment can be appropriate to use on children from different cultures, assuming that they can understand English (Glennen 2015).

In another study examining Aboriginal Australian children, the children who used Aboriginal English performed well on the CELF-P2. The use or lack of use of Aboriginal English did not predict children's performance on the CELF-P2 language sample portion. An explanation for this could be either the assessment tool is not sensitive enough to the differences between Aboriginal English and Standard Australian English, or that the children are code-switching. Overall, it is determined that this is an appropriate assessment to use for children in the early language development stages (Miller et al. 2014).

TTFC-2

The Token Test for Children-Second Edition (TTFC-2) assesses receptive language difficulties in children ages 3–12 years, 11 months. Scores from this assessment reflect the child's ability to complete multiple-step oral directions, understand expressive language, and use their short-term auditory memory. The linguistic components assessed include syntactic structures, knowledge of prepositional and relational concepts, and semantic development. The TTFC-2 includes contextualized stimuli of 46 verbally transmitted commands divided into four parts based on their complexity level, allowing measurements to concretely reflect the child's receptive language and not rely on verbal expression (DiSimoni et al. 2007).

The original TTFC efficacy for evaluating children has not been promising due to its lack of age norms, socioeconomic group comparison and lack of clarity in data collection (DiSimoni 1978). This led to the creation of the TTFC-2 in 2007 by DiSimoni, Ehrlet, and McGhee, which showed improvement from the first edition in validity, reliability, and normative data by having it be a diverse representative of the demographics

of the current United States population. The standardization of the TTFC-2 was based on of geographically diverse communities which included minorities such as those who identify as African American, Hispanic, Asian, and Native American. However, the sample for standardization failed to provide socioeconomic status.

The format of the TTFC-2 assessment holds clinical value for diverse cultural linguistic communities. Several versions have been developed such as Dutch, German, Khmer, Kannada, Polish, Portuguese, and several Spanish-speaking populations (Gallardo et al. 2011). Although many versions lack standardization and psychometric evidence, resulting in poor efficacy, there are two versions that do hold validity and reliability. The A-TTFC was created for native speakers of Arabic (Alkhamra and Al-Jazi 2016) and presents as a valid and reliable assessment. The test was changed into an appropriate Arabic format without adjusting any content and measures of the TTFC and presents with similar clinical strengths for Arabic-speaking children as the TTFC-2 does for English-speaking children. The Revised Token Test (RTT) has shown to be a reliable and valuable tool for assessment for Spanish-speaking children due to its psychometric sensitivity. Spanish speaking children are the most frequent ethnic group receiving services from speech-language pathologists, so it is important to have a reliable assessment for this population in order to avoid under referrals or over referrals (Roseberry-Mckibbin and Eicholtz 1994). Although this revised assessment has shown to result in valid results, additional research is needed with the normative data for these revisions before relying on these versions of assessments throughout practice.

PLS-5

The Preschool Language Scales, Fifth Edition (PLS-5) is used to evaluate the domains of Auditory Comprehension (AC) and Expressive Communication (EC) in children from birth to the developmental age of 7 years, 11 months. This test assesses several domains including: “attention, gesture, play, vocal development, social communication, vocabulary, concepts, language

structure, integrative language, and emergent literacy” (Zimmerman et al. 2011). The results of this test can be used to determine areas of difficulty and strength, and to track the child’s progress over time. The results of this test can identify if the child has a language delay or language disorder, and if it is expressive, receptive, or both. The PLS-5 provides norm-referenced scores for both the AC and EC sections. Additionally, it provides the information necessary to calculate a Total Language score. Based on the standard score that the child receives, the clinician can determine the distance from mean, percentile rank, and age equivalent for that child. These scores can help determine where the child’s communication skills fall compared to other children of the same age (Zimmerman et al. 2011).

The PLS-5 accounts for dialectical differences by providing correct responses for the dialects of African-American English, Appalachian English, English influenced by Chinese languages, English influenced by Spanish, and Southern English. This decreases the likelihood that children will be penalized for speaking with a dialect other than Standard American English provided the clinician is aware of the dialectical difference and references the applicable correct responses.

While there are several benefits of using this test battery, there are also cultural and environmental limitations that have been indicated. For example, when using this assessment on Mandarin-speaking children, most of the 4-year-olds were unable to express what they would do with a coat, towel, or cup, and nearly half of the 3-year-olds were unable to answer the questions about what they would do if they were sick (Ren et al. 2016). In this culture, it is rare for children to be encouraged to participate in self-care tasks, which could negatively affect their performance on sections of this test battery and lead to the over-referral of children from this culture. Additionally, factors such as socioeconomic status or prior exposure to stimuli could affect performance on this assessment. There is evidence that children from low socioeconomic status may perform more poorly on vocabulary tasks than children from higher socioeconomic status homes due to

lack of exposure, even though the ability to learn new vocabulary is not impaired (Horton-Ikard and Weismer 2007). These limitations could lead to the overdiagnosis of culturally diverse children with language disorders in expressive language and auditory comprehension of language and lead to providing treatment services to individuals who do not need these services, unnecessarily taking up the time of both the student and the clinician.

Future Directions

Many assessment batteries that are commonly used in the United States for the assessment of language skills and the diagnosis of language delays and disorders in children have cultural limitations that could potentially lead to the overdiagnosis of culturally diverse children (Horton-Ikard and Weismer 2007; Losen and Orfield 2002; Pearson et al. 2014; Robinson and Norton 2012; Roseberry-Mckibbin and Eicholtz 1994). This initial review of literature has examined the cultural limitations in the commonly used test batteries and reveals the necessity to continue to explore standardized measures for their degree of cultural sensitivity and responsiveness.

While some assessment tools, such as the DELV-NR (Seymour et al. 2018), were designed to be dialectically neutral to improve the diagnostic accuracy of language disorders in this population, other commonly used assessments were found to have weaknesses that could lead to the overdiagnosis of children who are culturally diverse. As the world is becoming more culturally responsive, our language assessments must follow suit. Looking ahead, clinicians must be vigilant in responding to the differences presented by children and acknowledge cultural differences prior to the administration of an assessment. Miller et al. (2014) suggest using language assessments with other language analysis methods that are culturally appropriate. Further, they suggest that speech-language pathologists should be sensitive to the varied cultures and languages that children are exposed to and not make any assumptions regarding their culture and the impact on their language comprehension and expression (Miller

et al. 2014). Lack of awareness of these weaknesses is clinically relevant and could lead to providing services to individuals who do not require intervention or result in a child being delayed in receiving services. A culturally responsive clinician should be aware of the degree of cultural bias in the test batteries they are using to evaluate their clients in order to accurately assess the language skills of culturally diverse children and provide services only if they are necessary (Paul et al. 2018).

See Also

- ▶ [Adaptive Behavior](#)
- ▶ [Clinical Evaluation of Language Fundamentals: Preschool – Second Edition \(CELF Preschool-2\)](#)
- ▶ [Communication and Symbolic Behavior Scale](#)
- ▶ [Culture and Autism](#)
- ▶ [Diagnostic Evaluation of Language Variation \(DELV-Norm Referenced\)](#)
- ▶ [DIR Model \(Developmental, Individual Difference, Relationship Based\): A Parent-Mediated Mental Health Approach to Autism Spectrum Disorders, The](#)
- ▶ [Ecological Model of Autism](#)
- ▶ [Measurement Error](#)
- ▶ [Subtyping Autism](#)
- ▶ [Token Test for Children – Second Edition \(TTFC-2\)](#)

References and Reading

- Alkhamra, R., & Al-Jazi, A. (2016). Validity and reliability of the Arabic Token Test for children. *International Journal of Language & Communication Disorders*, 51(2), 183–191.
- Chambers, N., Stronach, S. T., & Wetherby, A. M. (2016). Performance of South African children on the Communication and Symbolic Behavior Scales-Developmental Profile (CSBS DP). *International Journal of Language & Communication Disorders*, 51(3), 265–275.
- Danescu, E. (1997). Parental beliefs on childhood disability: Insights on culture, child development and intervention. *Journal of Disability, Development and Education*, 44, 41–52.
- DiSimoni, F. (1978). *Token Test for Children*. Austin: PRO-ED.

- DiSimoni, F., McGhee, R., & Ehrler, D. (2007). *The Token Test for Children-second edition*. Austin: PRO-ED.
- Gallardo, G., Guàrdia, J., Villaseñor, T., & McNeil, M. (2011). Psychometric data for the Revised Token Test in normally developing Mexican children ages 4–12 years. *Archives of Clinical Neuropsychology*, 26(3), 225–234.
- Gay, G. (2000). *Culturally responsive teaching: Theory, research, and practice*. New York: Teachers College Press.
- Gibson, D. D. (2007). Racial disparities in the age of diagnosis of autism spectrum disorder: Examining factors that may contribute to delayed diagnosis in African-American children. *Praxis*, 7, 34–38.
- Glennen, S. (2015). Internationally adopted children in the early school years: Relative strengths and weaknesses in language abilities. *Language, Speech, and Hearing Services in Schools*, 46(1), 1–13.
- Horton-Ikard, R., & Weismer, S. (2007). A preliminary examination of vocabulary and word learning in African American toddlers from middle and low socioeconomic status homes. *American Journal of Speech-Language Pathology*, 16(4), 381–392.
- Losen, D. J., & Orfield, G. (Eds.). (2002). *Racial inequity in special education: The Civil Rights Project at Harvard University*. Cambridge, MA: Harvard Education Press.
- Mandell, D., Ittenbach, R., Levy, S., & Pinto-Martin, J. (2007). Disparities in diagnoses received prior to a diagnosis of autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37, 1795–1802.
- Miller, E., Webster, V., Knight, J., & Comino, E. (2014). The use of a standardized language assessment tool to measure the language development of urban Aboriginal preschoolers. *International Journal of Speech-Language Pathology*, 16(2), 109–120.
- Paslawski, T. (2005). Clinical Evaluation of Language Fundamentals, fourth edition (CELF-4): A review. *Canadian Journal of School Psychology*, 20(1–2), 129–134.
- Paul, R., Norbury, C., & Gosse, C. (2018). *Language disorders from infancy through adolescence: Listening, speaking, reading, writing, and communicating*. St. Louis: Elsevier.
- Pearson, B. Z., Jackson, J. E., & Wu, H. (2014). Seeking a valid gold standard for an innovative, dialect-neutral language test. *Journal of Speech, Language, and Hearing Research*, 57(2), 495–508. https://doi.org/10.1044/2013_JSLHR-L-12-0126.
- Ren, Y., Rattanasone, N., Wyver, S., Hinton, A., & Demuth, K. (2016). Interpretation of errors made by Mandarin-speaking children on the Preschool Language Scales-5th edition Screening Test. *Australian Journal of Educational and Developmental Psychology*, 15, 24–34.
- Roberts, J., Medley, L., Swartzfager, J., & Neebe, E. (1997). Assessing the communication of African American one-year-olds using the Communication and Symbolic Behaviour Scales. *American Journal of Speech-Language Pathology*, 6, 59–65.
- Robinson, G., & Norton, P. (2012). How does your state represent? African Americans on speech-language caseloads. Paper presented at the American Speech-Language-Hearing Association Convention, Atlanta.
- Roseberry-Mckibbin, C., & Eicholtz, G. (1994). Serving children with limited English proficiency in the schools: A national survey. *Language, Speech, and Hearing Services in Schools*, 25(3), 156–164.
- Ryan, A., Gibbon, F. E., & O'Shea, A. (2016). Expressive and receptive language skills in preschool children from a socially disadvantaged area. *International Journal of Speech-Language Pathology*, 18(1), 41–52.
- Seymour, H. N., Roeper, T. W., & de Villiers, J. (2018). *Diagnostic Evaluation of Language Variation-Norm Referenced*. Sun Prairie: Ventris Learning.
- Tek, S., & Landa, R. (2012). Differences in autism symptoms between minority and non-minority toddlers. *Journal of Autism and Developmental Disorders*, 42(9), 1967–1973.
- Wetherby, A. M., & Prizant, B. M. (2003). *Communication and Symbolic Behavior Scale (CSBS)*. Baltimore: Paul H. Brookes Publishing.
- Wiggins, L. D., Barger, B., & Moody, E. (2019). Brief report: the ADOS calibrated severity score best measures autism diagnostic symptom severity in pre-school children. *J Autism Dev Disord* 49, 2999–3006.
- Wiig, E., Secord, W. A., & Semel, E. (2004). *The clinical evaluation of language fundamentals – preschool* (2nd ed.). San Antonio: Harcourt Assessment.
- Zimmerman, I. L., Steiner, V. G., & Pond, R. E. (2011). *The preschool language scale-5*. San Antonio: Pearson.

Culturally Sensitive Social Competence Training Program for Chinese High-Functioning Adolescents with ASD, A

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Definition

The CBT-context-based social competence training for ASD (CBT-CSCA) was a social competence training specifically developed for Hong

Kong Chinese adolescents with ASD. The CBT-CSCA was grounded on cognitive-behavioral therapy and incorporated cultural-sensitive elements. It demonstrated the effectiveness in improving social competence among adolescents with ASD in Hong Kong, and the gains were sustained 3 months after the training.

Historical Background

Impaired social functioning is a hallmark feature of autism spectrum disorder (ASD). It becomes a defining feature in the current diagnostic approaches (American Psychiatric Association; APA 2013). Despite improvements shown in other features of ASD upon interventions, social difficulties persist as individuals enter adolescence and young adulthood (Schall and McDonough 2010). This persistence is possibly due to a rapid change in the social landscape and the increase in the complexity of social interaction during adolescence.

In the lack of the necessary social skills to handle complex social situations, adolescents with ASD tend to suffer from a number of negative consequences, including restricted social circle, peer rejection, and bullying (Rieffe et al. 2010). Without pleasant social experiences, adolescents with ASD also suffer from a high prevalence of psychiatric disorders (Simonoff et al. 2008). Presumably, equipping adolescents with ASD with appropriate social skills improves their social deficits as well as their comorbid psychiatric symptoms. Addressing the critical socialization needs of adolescents with ASD, there has been a rapid increase in research on social competence interventions for adolescents with ASD since the previous decade (Reichow et al. 2013).

Group-based intervention has demonstrated initial success (Miller et al. 2014), such as the Program for the Education and Enrichment of Relational Skills (PEERS) (Laugeson et al. 2012), the Multimodal Anxiety and Social Skills Intervention (MASSI) program (White et al. 2013), and Skillstreaming (Lopata et al. 2008). However, most studies published are designed and validated in the Western culture, such as the

USA, Germany, and Scotland, limiting their generalizability. Studies in the East included only the validation studies of the PEERS in Korea (Yoo et al. 2014) and Hong Kong (Shum et al. 2019).

Current Knowledge

In light of the distinctiveness of Chinese culture as compared to the West, the CBT-context-based social competence training for ASD (CBT-CSCA) was developed locally to cater the unique cultural and service needs for Chinese adolescents with ASD. The CBT-CSCA followed the definition of social competence suggested by Bierman and Welsh (2000) “an organisational construct that reflects the child/adolescent’s capacity to integrate behavioural, cognitive and affective skills to adapt flexibly to diverse social contexts and demands”.

The training content of CBT-CSCA followed the model proposed by Yager and Iarocci (2013). The seven domains of social competence were organized into four modules of training, specifically social context (social motivation and social knowledge), behavior (verbal conversational and nonverbal sending skills), emotion (emotion regulation skills and demonstrating empathic concern), and cognition (social inferencing). In each module, specific and concretized behavioral and cognitive skills are listed as targets of training. The training consisted of 15 weekly sessions (see Table 1). Each session lasted for 120 min and followed a standardized format modeling a CBT session. Typically, homework was reviewed at the beginning of each meeting, with compliance and quality recorded. It was followed by didactic teaching and role-played demonstrations on the targeted skills of each session. Newly learned skills were rehearsed by adolescents, during which they received performance feedback. The teaching was followed by a naturalistic game and snack times, during which the use of learned skills from the trainees was systematically reinforced. Before the session ended, a test on teaching materials was distributed to consolidate learning. Homework for the coming week was assigned to encourage skills generalization to natural settings.

Culturally Sensitive Social Competence Training Program for Chinese High-Functioning Adolescents with ASD, A, Table 1 Overview of the CBT-CSCA

Session	Module	Contents	Homework
1	Social context: motivation	Introduce the purpose, structure, and rules of the training program Enhance motivation in group participation	Adolescents record social encounters and the benefits of these encounters
2	Social context: knowledge	Understand hidden social rules in social contexts Learn how to detect hidden social rules in social contexts common to adolescents Introduce perspective-taking questions	Adolescents detect hidden social rules in contexts common to them and evaluate their performance in following them
3	Social context: knowledge	Understand general social rules across different social contexts Learn how to detect general social rules	Adolescents apply skills in detecting general social rules in different settings
4	Behavior: active listening	Learn active listening skills Detect signals of exiting conversations and practice their application	Adolescents apply active listening skills and signals of exiting conversations
5	Behavior: conversation	Learn how to end and maintain conversations Understand four conversation blockers	Adolescents practice skills in ending and maintaining conversations
6	Behavior: conversation	Learn how to initiate and maintain conversations Practice skills in initiate, maintain, and end conversations in different social contexts	Adolescents practice integrated skills in initiating, maintaining, and ending conversations
7	Behavior: electronic communication	Learn how to join a group conversation Learn skills in electronic communication	Adolescents practice skills in joining a group conversation and electronic communication
8	Emotion: facial recognition and expression	Understand how to recognize emotions through facial expressions Practice expressing emotions through facial expressions	Adolescents practice recognizing and expressing emotions using facial expressions with family members
9	Emotion: Gestural and tonal recognition and expression	Understand how to skills emotions through tonal and gestural expressions Practice expressing emotions through tonal and gestural expressions	Adolescents record an emotional incident and retell it with application of facial, tonal, and gestural expressions
10	Emotion: regulation	Learn skills to handle criticisms and related negative emotions	Adolescents record incidents of criticisms and how they apply the skills in handling criticisms
11	Emotion: empathy	Understand what empathy is Learn how to deliver empathic responses	Adolescents record incidents that they apply the skills taught in delivering empathic responses
12	Cognition: social inference	Learn how to make inference on others' thoughts	Adolescents record incidents that they apply skills in making inference on others' thoughts

(continued)

Culturally Sensitive Social Competence Training Program for Chinese High-Functioning Adolescents with ASD, A, Table 1 (continued)

Session	Module	Contents	Homework
13	Integration: friendship building	Understand interpersonal circle Learn how to join group activities and detect unwelcome signals	Adolescents build a friendship with one of the members of the group by phone conversations or electronic communication
14	Integration: planning group activities	Learn how to invite others to join group activities and plan for group activities	Adolescents prepare their speech to be shared at the graduation party
15	Graduation	Share learning in the program Celebrate for graduation	N/A

Regarding cultural-sensitive elements, familiar social contexts unique to local youth were first identified and subsequently included as a basis for training. These social contexts included dining out with family relatives, visiting extended family members, group project discussion in school, and visiting arcade with peers. Besides, training activities and games were designed concerning the local popular culture. For instance, a popular local game that the winner of rock-paper-scissors would become an “emperor” was modified to require players to demonstrate active listening skills in becoming the emperor. Common local board games were also prepared during the naturalistic game time in each session.

The training program was conducted by trained workers, who were registered clinical psychologists, counselors, or social workers, all with a graduate or postgraduate qualifications and substantial experience in conducting social competence training with adolescents with ASD. Workers were fully trained in the CBT-CSCA with confirmed competence. The implementation fidelity of the training was ensured through regular on-site checking regarding the established manual and bi-weekly supervision by the first author (RC).

The CBT-CSCA was validated with local assessment tools on social competence, autistic symptoms, general psychopathology, and parental stress. In a group of 25 adolescents (aged 12 to 17 years, with an FSIQ above 80), significant improvements in social competence, autistic symptoms, and general psychopathology at post-training and 3-month follow-up were reported by

the parents. The findings provided evidence support to the integrative multi-module approach of CBT-CSCA and its crucial training components. The positive results on autistic symptoms and general psychopathology in the present study support the spillover effect of social competence training on improving the related symptoms of ASD. The inclusion of emotion and cognition components in CBT-CSCA is considered to be contributive to the observed significant positive changes. The findings also support the proposition that ASD is primarily a social development disorder and addressing social deficits is a centrality approach for ASD (Chan et al. 2014).

The CBT-CSCA was guided by established theoretical propositions, appropriate cultural adaption, empirically supported training strategies, and relevant contextual contents. It also gives preliminary evidence to the validity of using such an approach in constructing culturally sensitive and effective social interventions for persons challenged by ASD. Apart from the cultural components, CBT-CSCA also adopts an integrative multi-module approach and underlines the importance of context, behavior, emotion, and cognition in social competence learning. Unlike the common practice of translating and adopting foreign-developed training program, the CBT-CSCA was locally developed, with the incorporation of culturally sensitive training contents and activities. The program was not only theoretically valid and empirically supported, but it also accommodated the unique cultural needs of Hong Kong adolescents. The CBT-CSCA also served as an exemplar of developing local

treatment programs via cultural adaptation in the mental health setting.

Future Directions

Despite availability and provision of intervention at the younger age, intervention gains do not seem to translate into adulthood. Prognosis in adulthood was often unsatisfactory (Levy and Perry 2011). Social incompetence often remained to be one of the most disabling impairments among adults with ASD, potentially contributed by the rise in complexity and salience of social relationships (Tantam 2003).

To address the continuity of social impairments, adults with ASD would need psychosocial interventions addressing their unique social challenges. Although there is social skills intervention for adults with high-functioning ASD (see Spain and Blainey 2015 for a review), studies were mostly in lack of cultural diversity, in which all were conducted in the Western countries. There was a need for effective intervention to serve the ASD population in the East. Given the demonstrated effectiveness of CBT-CSCA in adolescents, the extension in the application of CBT-CSCA on adults with ASD shall be investigated.

See Also

- [China and Autism](#)
- [Cognitive Behavioral Therapy \(CBT\)](#)
- [Social Skill Interventions](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). DC: Washington.
- Bierman, K., & Welsh, J. (2000). Assessing social dysfunction: The contributions of laboratory and performance-based measures. *Journal of Clinical Child Psychology*, 29(4), 526–539.
- Chan, R. S. W., Lo, T. Y., & Tang, C. P. (2014). Social competence as the centrality of intervention for autism spectrum disorder (ASD): why and how? *Hong Kong Journal of Mental Health*, 40(1), 5–11.
- Laugeson, E. A., Frankel, F., Gantman, A., Dillon, A. R., & Mogil, C. (2012). Evidence-based social skills training for adolescents with autism spectrum disorders: The UCLA PEERS program. *Journal of Autism and Developmental Disorders*, 42(6), 1025–1036.
- Levy, A., & Perry, A. (2011). Outcomes in adolescents and adults with autism: A review of the literature. *Research in Autism Spectrum Disorders*, 5(4), 1271–1282.
- Lopata, C., Thomeer, M. L., Volker, M. A., Nida, R. E., & Lee, G. K. (2008). Effectiveness of a manualized summer social treatment program for high-functioning children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(5), 890–904.
- Miller, A., Vernon, T., Wu, V., & Russo, K. (2014). Social skill group interventions for adolescents with autism spectrum disorders: A systematic review. *Review Journal of Autism and Developmental Disorders*, 4, 254–265.
- Reichow, B., Steiner, A. M., & Volkmar, F. (2013). Social skills groups for people aged 6 to 21 with autism spectrum disorders (ASD). *Evidence-Based Child Health: A Cochrane Review Journal*, 8(2), 266–315.
- Rieffe, C., Camodeca, M., Pouw, L. B., Lange, A. M., & Stockmann, L. (2010). Don't anger me! Bullying, victimization, and emotion dysregulation in young adolescents with ASD. *European Journal of Developmental Psychology*, 9(3), 351–370.
- Schall, M., & McDonough, J. (2010). Autism spectrum disorders in adolescence and early adulthood: Characteristics and issues. *Journal of Vocational Rehabilitation*, 32(2), 81–88.
- Shum, K. K. M., Cho, W. K., Lam, L. M. O., Laugeson, E. A., Wong, W. S., & Law, L. S. (2019). Learning How to Make Friends for Chinese Adolescents with Autism Spectrum Disorder: A Randomized Controlled Trial of the Hong Kong Chinese Version of the PEERS® Intervention. *Journal of Autism and Developmental Disorders*, 49(2), 527–541.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, 47(8), 921–929.
- Spain, D., & Blainey, S. H. (2015). Group social skills interventions for adults with high-functioning autism spectrum disorders: A systematic review. *Autism*, 19(7), 874–886.
- Tantam, D. (2003). The challenge of adolescents and adults with Asperger syndrome. *Child and Adolescent Psychiatric Clinics of North America*, 12, 143–163.
- White, S. W., Ollendick, T., Albano, A. M., Johnson, C., Oswald, D., Johnson, C., et al. (2013). Randomized controlled trial: multimodal anxiety and social skill intervention for adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43(2), 382–394.
- Yager, J., & Iarocci, G. (2013). The development of the multidimensional social competence scale: a

standardized measure of social competence in autism spectrum disorders. *Autism Research*, 6(6), 631–641.

Yoo, H. J., Bahn, G., Cho, I. H., Kim, E. K., Kim, J. H., Min, J. W., et al. (2014). A Randomized Controlled Trial of the Korean Version of the PEERS® Parent-Assisted Social Skills Training Program for Teens With ASD. *Autism Research*, 7(1), 145–161.

Culture and Autism

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Definition

The understanding of autism can only be gained by further examination of autism both across and within cultures. Careful attention to observed similarities and differences will allow a better understanding of the disorder as well as better design and development of interventions that are applicable to families of all backgrounds. This entry describes research to date on how culture influences the epidemiology, diagnosis, and treatment of autism throughout the world, with a special focus on family functioning, and disparities in diagnosis and care.

Historical Background

While knowledge about autism is increasing rapidly throughout the world, there are to date few scientific studies of the characteristics, prevalence, and phenotypes of autism spectrum disorder (ASD) outside of North America and Western Europe and how culture, race, and ethnicity influence the understanding and management of ASD. Researchers have long agreed that autism occurs

in families across races and socioeconomic backgrounds and, based on the presence of autism parent organizations in more than 100 countries, there is clear evidence that a constellation of behaviors has been recognized as “autism” on every continent. Researchers are just beginning to study, however, the extent to which ASD varies both across and within cultures. While there appear to be similarities in the onset and core symptoms of ASD around the world, this remains an untested assumption. ASD experts to date are gradually learning more about how cultural differences may influence its prevalence, diagnosis, treatment, presentation, course, and family function.

Current Knowledge

Prevalence

The received view is that the prevalence of autism is consistent across races and cultures (Fombonne 2003). However, for many years, epidemiological studies were carried out primarily in North America and Western Europe. Work from outside these areas was limited, with the notable exception of Japan (e.g., Honda et al. 2005; Matsuishi et al. 1987; Sugiyama and Abe 1989). In recent years, reports of prevalence have come from Singapore (Bernard-Optiz et al. 2001), Iran (Samadi et al. 2011), China (Wong and Hui 2008), Oman (Al-Farsi et al. 2011), Venezuela (Montiel-Nava and Pena 2008), Sri Lanka (Perera et al. 2009), and Korea (Kim et al. 2011), among others. Most studies provide prevalence estimates similar to those for the USA, although Kim et al. found more than twice the prevalence of US studies in South Korea. Prevalence studies have used a wide range of methods and are therefore difficult to compare to one another. Moreover, autism epidemiologists face the difficult task of reliably defining cases based on information from teacher and parent reports, both of which can be heavily influenced by cultural attitudes about discipline and what kinds of behaviors are age appropriate. Estimates of autism may appear higher in a country with great awareness among parents, educators, and clinicians and access to services or in

which the national government mandates the use of autism as a diagnostic term. In contrast, estimates may be lower in a country with little awareness, few services, and lack of systematic surveillance through research or administrative means. In the USA, for example, school records of autism grew tremendously following the 1991–1992 school year when the U.S. Department of Education first introduced these terms to the American public school system (Newschaffer et al. 2005).

Research on the prevalence of ASD among children of immigrants dates back more than 20 years. These studies have hypothesized a range of possible explanations for what appears to be higher rates in these children, including vulnerability to intrauterine infections and vitamin deficiencies, among others (Dealberto 2011; Gillberg et al. 1987, 1991, 1995; Narayan et al. 1992). Conclusions from this work are limited by small sample sizes. While some researchers believe they do not support an association, others note that maternal birth abroad represents at least a marginal increase in risk of having a child with ASD (Gardener et al. 2009). Recent work from the UK found a significant association of risk for immigrant mothers, particularly from the Caribbean (Keen et al. 2010). Similarly, data from the 3–4-year-olds in the Somali community in the US city of Minneapolis found the prevalence of ASD to be as much as seven times higher for Somali children than for non-Somali children, although this ratio was observed to decrease dramatically over time (Minnesota Department of Public Health 2009).

Diagnostic Processes

Although researchers may use standardized assessments and criteria – at the very least, The Diagnostic Statistical Manual of Mental Disorders (DSM) and The International Classification of Diseases (ICD) – to determine whether an individual constitutes a “case” of autism, most physicians and psychologists are not integrated into a research community and are likely to rely on past training and personal clinical experience. Even with standardized criteria, considerable subjectivity and differences in clinical assessments exist

because the diagnosis depends on patient or caretaker narrative combined with behavioral observation rather than biological tests. Research in India (Daley and Sigman 2002), Pakistan (Rahbar et al. 2011), and Nigeria (Bakare et al. 2009) shows that professionals can hold different beliefs about the criteria that are important for diagnosis of autism, and many have never even heard of the disorder. In South Korea, children that American clinicians might diagnose with autism are often diagnosed with reactive attachment disorder (RAD), pejoratively referred to as “lack of love” (*aejŏng kyŏlpip*), a term that parallels the older American concept of the “refrigerator mother.”

Cross-cultural variations in diagnostic practices for autism have been found, even among communities whose scientific traditions are often assumed to be similar, such as the USA and western European countries. For example, in the USA, the American Psychiatric Association removed autism from the category of “psychosis” in 1980, but the French child psychiatric establishment, which uses its own indigenous manual of mental disorders, the *Classification Française des Troubles Mentaux de l'Enfant et de l'Adolescent* (CFTMEA), classified autism as a psychosis until 2004. French health professionals also conceptualize the etiology of autism differently from other European countries and consider the American classification of pervasive developmental disorders (PDDs) to be a product of Anglo-American culture. Since French health professionals generally view autism as a problem that lies within family social relationships and with the mother-child relationship in particular, there are only a few psychiatric or medical centers with expertise on autism as a genetic or brain disorder.

Clinicians may not make or record a particular diagnosis, and parents will not seek it, unless the diagnostic term is meaningful and in current use. For example, one report of the Navajo Indians of the American Southwest notes that autism is characterized as “perpetual childhood” (Connors and Donnellan 1995), and through the mid-1990s in India, it was not uncommon for clinicians to refer to children with autism as *paagol*, the Hindi word for madness (Daley 2004). In rural South Korea,

the catch-all “brain disorder” can be used for children with disorders including traumatic brain injury, autism, epilepsy, speech and language disorders, Down syndrome, and other clearly genetic disorders. In addition, many parents in South Korea prefer a RAD diagnosis to autism because (1) it is believed to be a temporary condition treatable by giving love and affection; (2) by blaming the mother, blame is deflected away from the larger family, including the lineage and lineage ancestors; and (3) the diagnosis makes sense in South Korea’s changing social context, which includes the recent integration of mothers into the work force, thereby altering family life and child care and justifying concern about mothers’ attachment to their young children (Grinker 2007).

It is not clear whether diagnostic tools are sensitive to cultural differences, and while many autism screening and diagnostic tools may demonstrate satisfactory properties, they should not be assumed to have applicability in a given culture without some level of validation. Items may need to be tailored to language impairment as it is defined in particular languages and cultures. For example, the Modified Checklist for Autism in Toddlers (M-CHAT) has been translated into more than 40 languages and tested in a number of countries, including China, India, Sri Lanka, Egypt, Kuwait, Jordan, Oman, Qatar, Saudi Arabia, Syria, Tunisia, and Lebanon. While used in many countries, careful validation is not always completed. In at least one location – Sri Lanka – the tool demonstrated unacceptably low specificity (Perera et al. 2009). The authors cite both a lack of cultural relevance of some items, as well as a consistent pattern in which social and communication impairments were not viewed as an abnormality by the mothers. A “symptom” such as poor eye gaze in a 2-year-old may be seen as an impairment in one society but politeness in another. Language is another example of the need to consider cultural practices and norms. For example, while a commonly reported impairment associated with autism among English speakers is pronominal reversal, it occurs rarely in languages, such as Korean and Javanese, in which pronouns are seldom used. Many East Asian languages use

honorifics to denote the status of the speakers in particular conversations and settings, so the inappropriate use of honorifics is an indicator of deficiencies in an individual’s ability to understand the pragmatics of shifting referents and social context.

Within the USA, race, ethnicity, and socioeconomic status all play a role in which children are identified as having autism. Mandell et al. (2002) found that African-American children subsequently diagnosed with autism are at least 2.5 times less likely to receive a diagnosis at their first specialty visit than a white child with autism. Work by Jarquin et al. (2011) found that non-Hispanic Black children receive only the most severe diagnosis of ASD, in contrast to non-Hispanic White children who receive diagnosis at all levels of severity. Using data collected from states on the number of children eligible for special education services under the category of autism, Morrier et al. (2010) found significant underrepresentation for Hispanic children in 95% of US states. Travers et al. (2011) were able to document that this particular finding has persisted between 1998 and 2006, while the likelihood of eligibility based on ASD varied across time for other minority groups. Educational eligibility is clearly not equivalent to diagnosis, although these findings are consistent with the recommendation by Mandell and Novak (2005) to conduct research on “the complex relationship between culture and treatment, focusing on cultural differences in the behavioral phenotype of ASD, recognition of symptoms, interpretation of symptoms, families’ decisions regarding medical and educational interventions, and interactions between families and the healthcare system” (2005, p. 114).

It must be emphasized that cultural variables, such as local conceptions of the normal and abnormal, stigma, and attitudes about disclosure, cannot be easily measured, in large part because there are so many cross-cultural differences in the norms of child rearing. The use of qualitative ethnographic methods, such as cultural consensus analyses, provides systematic data on attitudes toward health and illness that can be quantified and converted into schematic models of shared beliefs

(Romney et al. 1986). Researchers can then quantify levels of agreement among informants and identify both shared cultural categories as well as intercultural differences.

Every geographic location and community may demand different methods and types of description. An ethnographic study of help-seeking for ASD in the USA would likely focus primarily on the relationship between parents and health care providers, while the same study in Kenya would likely focus on an extended-family disease management group and how the family negotiates a plurality of coexisting medical and religious systems. In other words, although autism appears to be universal, the contexts in which it occurs are distinctive.

Treatment

The availability of providers and clinical services that are specific to ASD remains one of largest challenges in most countries in the world. Intervention programs for children with ASD have largely developed under conditions that are both culturally inconsistent and economically untenable for most low- and middle-income countries. Indeed, researchers must also be aware of the cultural fit of many interventions, especially for minority groups within the USA. One intervention approach that is well known for its adaptability in a number of different countries is treatment and education of autistic and communication-related handicapped children or TEACCH (Schopler and Mesibov 2000), a training, services, and research program that has been implemented in a wide range of countries, including Australia, Brazil, Cambodia, Denmark, Iceland, Italy, Germany, Greece, India, Israel, Japan, Kuwait, Mexico, New Zealand, the Netherlands, Republic of Ireland, South Africa, Spain, Sweden, UK, Venezuela, and Vietnam. The flexibility to create new materials and the ability to apply the general principles of the program have likely contributed to its successful adaptation.

School- or classroom-based interventions are only effective, however, if children with ASD attend school. Many children with ASD and other disabilities simply do not attend school at

all. Estimates by the United Nations Children's Fund (UNICEF) and the United Nations Educational Scientific and Cultural Organization (UNESCO) place the percentage of children with disabilities living in developing countries who receive a basic primary education to be between 1% and 5%. In these countries, school-based opportunities are limited primarily to the urban areas, and even in these situations, children with autism are typically in settings where professionals have little knowledge about effective practices specifically for children with ASD. In Ghana, for example, classrooms were described as "crowded, loud, and unpredictable" and "transitions between activities generally occurred without warning, after inconsistent durations and at varying times of the day" (Anthony 2009, p. 9), conditions which are both typical in most low- and middle-income countries and also challenging for many children with ASD.

In the absence of laws and regulations that provide services, parents of children with ASD may engage in a high level of help-seeking and may use a combination of local healers, indigenous systems of medicine, medication, and Western treatments for ASD. In China, for example (Clark and Zhou 2005), there is wide use of sensory integration therapies in addition to applied behavioral analysis (ABA). In India, families have long relied on both special education and traditional healing systems, such as the use of Ayurvedic and homeopathic medicine (Daley 2002). They now also seek treatments for their children that include hyperbaric oxygen chambers, stem-cell replacement, Defeat Autism Now (DAN) diets, auditory integrated therapy, and many others. Of course, such options are not available to most families and in most countries, regardless of whether they are effective. As is true for families throughout the world, parents in Taiwan reported a willingness to try anything that might work, from fortune tellers to vitamin supplements (Shyu et al. 2010).

Family Functioning

Just as the impact of a child with autism on the family varies widely within cultures, it also varies considerably across cultures. In many countries,

having a child with a disability of any type is made even more challenging as a result of the stigma associated with such a difference. Stigma is generally defined as a form of branding of an individual in which a community devalues his or her social identity. In Korea, for example, despite dramatic changes in autism awareness in all segments of society, autism (*chap'ae*) continues to be a highly undesirable disability, and the diagnosis is believed to be applicable primarily to children and adults with profound intellectual impairments. Other cultures provide pathways that minimize stigma. In India, the recent positive portrayal of people with ASD on television and in films, even if inaccurate, has opened dialogue about disability and has provided a point of cultural reference, and in some cases, pride (Singhal 2010). In the USA, many people who have both ASD and above-average intelligence, while facing social challenges, still find gainful employment in the fields of engineering, computers, or mathematics.

Within North America and Europe, the parenting experience of minority groups can involve additional challenges as a result of cultural differences. Kediye et al. (2009) described challenges faced by Somali parents, such as the language barrier in communicating with key professionals; a perception of racism and being judged; misguided advice from the general public who assume poor parenting; and a sense of estrangement in the absence of extended family. However, it is important to recognize the wide variability in experiences across minority groups. For example, Magaña and Smith (2006) found that Latina mothers of children with ASD had significantly better overall well-being than their non-Latina counterparts and reported striking differences in the degree to which Latina mothers held more positive beliefs about their children.

Future Directions

The understanding of autism can only gain by further examination of autism both across and within cultures. Careful attention to observed similarities and differences will help researchers and

clinicians better understand the disorder as well as better design and develop interventions that are applicable to families of all backgrounds. Some specific potential areas for future work include the following areas:

- As immigration patterns continue to shift with political changes in every corner of the globe, researchers are presented with continually changing options for investigation of *immigration and prevalence of ASD*.
- Pediatricians and primary health care workers are in need of *simple, reliable methods of identifying ASD* that do not require extensive training. More research on the *validity of diagnostic tools cross-culturally* is needed to equip these professionals with the instruments they need to accurately diagnose ASD.
- The dramatic gap in appropriate intervention options for the majority of children in the world with ASD suggests the need for researchers to work closely with professionals on the ground to *develop intervention approaches* that are cost-effective, feasible, and culturally relevant.
- Relatively little research has examined whether there are *differences in symptom expression* across cultures. *Genetic studies* are underway in many non-Western countries, which will allow much greater understanding of how genetic heterogeneity and culture interact to influence the presentation of ASD.

See Also

- [Epidemiology](#)
- [M-CHAT](#)
- [Prevalence](#)
- [TEACCH Transition Assessment Profile \(TTAP\)](#)

References and Reading

- Al-Farsi, Y. M., Al-Sharbati, M. M., Al-Farsi, O. A., Al-Shafae, M. S., Brooks, D. R., & Waly, M. I. (2011). Brief report: Prevalence of autistic spectrum disorders in the Sultanate of Oman. *The Journal of Autism and Developmental Disorders*, 41(6), 821–825.

- Anthony, J. H. (2009). Access to education for students with autism in Ghana: Implications for EFA. UNESCO. Retrieved from unesdoc.unesco.org/images/0018/001865/186588e.pdf on 7 Oct 2011.
- Bakare, M. O., Ebigo, P. O., Agomoh, A. O., Eaton, J., Onyeama, G. M., Okonkwo, K. O., Onwukwe, J. U., Igwe, M. N., Orovwigho, A. O., & Aguocha, C. M. (2009). Knowledge about childhood autism and opinion among healthcare workers on availability of facilities and law caring for the needs and rights of children with childhood autism and other developmental disorders in Nigeria. *BMC Pediatrics*, 9, 1–13.
- Bernard-Optiz, V., Kwook, K. W., & Sapuan, S. (2001). Epidemiology of autism in Singapore: Findings of the first autism survey. *International Journal of Rehabilitation Research*, 24(1), 1–6.
- Brown, J. R., & Rogers, S. J. (2003). Cultural issues in autism. In S. Ozonoff, S. J. Rogers, & R. L. Hendren (Eds.), *Autism spectrum disorders: A research review for practitioners*. Washington, DC: American Psychiatric Publishing.
- Clark, E., & Zhou, Z. (2005). Autism in China: From acupuncture to applied behavioral analysis. *Psychology in the Schools*, 42(3), 285–295.
- Connors, J. L., & Donnellan, A. M. (1995). Walk in beauty: Western perspectives on disability and Navajo family/cultural resilience. In H. McCubbin, E. Thomson, A. Thompspon, & J. Fromer (Eds.), *Resiliency in ethnic minority families: Native and immigrant American families* (Vol. 1, pp. 159–182). New York: SAGE.
- Cuccaro, M. L., Wright, H. H., Rownd, C. V., & Abramson, R. K. (1996). Brief report: Professional perceptions of children with developmental difficulties: The influence of race and socioeconomic status. *Journal of Autism and Developmental Disorders*, 26(4), 461–469.
- Daley, T. C. (2002). The need for cross-cultural research on pervasive developmental disorders. *Transcultural Psychiatry*, 39(4), 532–551.
- Daley, T. (2004). From symptom recognition to diagnosis: Children with autism in urban India. *Social Science & Medicine*, 58, 1323–1335.
- Daley, T. C., & Sigman, M. D. (2002). Diagnostic conceptualization of autism among Indian psychiatrists, psychologists, and pediatricians. *Journal of Autism and Developmental Disorders*, 32, 13–23.
- Dealberto, M.-J. (2011). Prevalence of autism according to maternal immigrant status and ethnic origin. *Acta Psychiatrica Scandinavica*, 123, 339–348. Department for International Development (2000), Disability Poverty and Development, Issues, 15. London.
- Duranti, A., & Ochs, E. (1996). Use and acquisition of genitive constructions in Samoan. In D. Slobin, J. Gerhardt, A. Kyratzis, & G. Jiansheng (Eds.), *Social interaction, social context and language: Essays in honor of Susan Ervin-Tripp* (pp. 175–190). Mahwah: Lawrence Erlbaum Associates.
- Fombonne, E. (2003). Epidemiological surveys of autism and other pervasive developmental disorders: An update. *Journal of Autism and Developmental Disorders*, 33(4), 382.
- Gardener, H., Spiegelman, D., & Buka, S. L. (2009). Prenatal risk factors for autism: Comprehensive meta-analysis. *The British Journal of Psychiatry*, 195, 7–14.
- Gillberg, C., Andersson, L., Steffenburg, S., & Börjesson, B. (1987). Infantile autism in children of immigrant parents. *The British Journal of Psychiatry*, 150, 856–858.
- Gillberg, C., Steffenburg, S., & Schaumann, H. (1991). Is autism more common now than 10 years ago? *The British Journal of Psychiatry*, 158, 403–409.
- Gillberg, C., Schaumann, H., & Gillberg, I. C. (1995). Autism in immigrants: Children born in Sweden to mothers born in Uganda. *Journal of Intellectual Disability Research*, 39, 141–144.
- Grinker, R. R. (2007). *Unstrange minds: Remapping the world of autism*.
- Honda, H., Shimizu, Y., Imai, M., & Nitto, Y. (2005). Cumulative incidence of childhood autism: A total population study of better accuracy and precision. *Developmental Medicine and Child Neurology*, 47, 10–18.
- Hugo, C. J., Boshoff, D. E., Traut, A., Zungu-Dirwayi, N., & Stein, D. J. (2003a). Community attitudes toward and knowledge of mental illness in South Africa. *Social Psychiatry and Psychiatric Epidemiology*, 38, 715–719.
- Hugo, C. J., et al. (2003b). Community attitudes toward and knowledge of mental illness in South Africa. *Social Psychiatry and Psychiatric Epidemiology*, 38, 715–719.
- Jarquín, V. G., Wiggins, L. D., Schieve, L. A., & Van Naarden-Braun, K. (2011). Racial disparities in community identification of autism spectrum disorders over time; Metropolitan Atlanta, Georgia, 2000–2006. *Journal of Developmental & Behavioral Pediatrics*, 32(3), 179–187.
- Kediye, F., Valeo, A., & Berman, R. C. (2009). Somali-Canadian mothers' experiences in parenting a child with autism spectrum disorder. *Journal for the Association of Research on Mothering*, 11(1), 211–223.
- Keen, D. V., Reid, F. D., & Arnone, D. (2010). Autism, ethnicity and maternal immigration. *British Journal of Psychiatry*, 196, 274–281.
- Kim, Y. S., Leventhal, B., Koh, Y.-J., Fombonne, E., Laska, E., & Lim, E.-C. (2011). Prevalence of ASD in Korean School-Aged Children. *American Journal of Psychiatry*, 168(9), 904–912.
- Levy, S., & Hyman, S. (2003). Use of complementary and alternative treatments for children with autistic spectrum disorders is increasing. *Pediatric Annals*, 32, 685–691.
- Magaña, S., & Smith, M. J. (2006). Psychological distress and well-being of Latina and Non-Latina white mothers of youth and adults with an autism spectrum disorder: Cultural attitudes towards coresidence status. *American Journal of Orthopsychiatry*, 76(3), 346–357.
- Mandell, D. S., & Novak, M. M. (2005). The role of culture in family's treatment decisions for children with autism spectrum disorders. *Mental Retardation and Developmental Disabilities Research Reviews*, 11(2), 110–115.
- Mandell, D. S., Listerud, J., Levy, S. E., & Pinto-Martin, J. A. (2002). Race differences in the age at diagnosis

- among medicaid-eligible children with autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(12), 1447–1453.
- Mandell, D. S., Novak, M. M., & Zubritsky, C. D. (2005). Factors associated with age of diagnosis among children with autism spectrum disorders. *Pediatrics*, 116(6), 1480–1486.
- Matsuishi, T., Shiotsuki, Y., Yoshimura, K., Shoji, H., Imuta, F., & Yamashita, F. (1987). High prevalence of infantile autism in Kurume city, Japan. *Journal of Child Neurology*, 2(4), 268–271.
- Minnesota Department of Public Health. (2009). *Autism spectrum disorders among preschool children participating in the Minneapolis public schools early childhood special education programs*. St. Paul. Retrieved from www.health.state.mn.us/omh/projects/autism/index.cfm on 7 Oct 2011.
- Montiel-Nava, C., & Pena, J. A. (2008). Epidemiological findings of pervasive developmental disorders in a Venezuelan study. *Autism*, 12(2), 191–202.
- Morrier, M. J., Hess, K. L., & Heflin, L. J. (2010). Ethnic disproportionality in students with autism spectrum disorders. *Multicultural Education*, 16(1), 31–38.
- Narayan, M., Srinath, S., Anderson, G. M., & Meundi, D. B. (1992). Cerebrospinal fluid levels of homovanillic acid and 5-hydroxyindoleacetic acid in autism. *Biological Psychiatry*, 33, 630–635.
- Newschaffer, C. J., Falb, M. D., & Gurney, J. G. (2005). National autism prevalence trends from United States special education data. *Pediatrics*, 115, 277–282.
- Perera, H., Wijewardena, K., & Aluthwelage, R. (2009). Screening of 18-24-month-old children for autism in a semi-urban community in Sri Lanka. *Journal of Tropical Pediatrics*, 55(6), 402–405.
- Rahbar, M. H., Ibrahim, K., & Assassi, P. (2011). Knowledge and attitude of general practitioners regarding autism in Karachi, Pakistan. *Journal of Autism and Developmental Disorders*, 41(4), 465–474.
- Romney, A. K., Weller, S. C., & Batchelder, W. H. (1986). Culture and consensus: A theory of culture and informant accuracy. *American Anthropologist*, 88, 313–338.
- Samadi, S. A., Mahmoodizadeh, A., & McConkey, R. (2011). A national study of the prevalence of autism among five-year-old children in Iran. *Autism*, 16(1), 5–14. Epub ahead of print.
- Schopler, E., & Mesibov, G. B. (2000). Cross-cultural priorities in developing autism services. *International Journal of Mental Health*, 29, 3–21. International Priorities for Developing Autism Services via the TEACCH Model.
- Shin, Y.-J., Lee, K.-S., Min, S.-K., & Emde, R. N. (1999). A Korean syndrome of attachment disturbance mimicking symptoms of pervasive developmental disorder. *Infant Mental Health Journal*, 20(1), 60–76.
- Shyu, Y. I., Tsai, J. L., & Tsai, W. C. (2010). Explaining and selecting treatments for autism: Parental explanatory models in Taiwan. *Journal of Autism and Developmental Disorders*, 40(11), 1323–1331.
- Singhal, N. (2010). *The impact of the popular media on awareness: Aap Ki Antara*. Paper presented at the International Meeting for Autism Research (IMFAR), Philadelphia.
- Stone, W. L. (1987). Cross-disciplinary perspectives on autism. *Journal of Pediatric Psychology*, 12, 615–630.
- Sugiyama, T., & Abe, T. (1989). The prevalence of autism in Nagoya, Japan: A total population study. *Journal of Autism and Developmental Disorders*, 19(1), 87–96.
- Travers, J. C., Tincani, M., & Krezmien, M. P. (2011). A multiyear national profile of racial disparity in autism identification. *Journal of Special Education*, XX(X), 1–9.
- Wong, V. C. N., & Hui, S. L. H. (2008). Epidemiological study of autism spectrum disorder in China. *Journal of Child Neurology*, 23(1), 67–72.

Cumulative Incidence

► Incidence

Cumulative Risk

► Incidence

Curriculum

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Definition

Broadly defined, a curriculum is the set of courses, including the specific course content and sequence of topics, taught in schools. The term *core curriculum* refers to the set of courses and content typically required of all students in a school. In K-12 education, core curriculum would usually include reading, writing, mathematics, science, and social studies, as well as the arts and physical education. Additionally, the term *curriculum* sometimes is applied within a particular domain (e.g., the reading curriculum or the math curriculum). Curricula within a particular domain include specific subtopics or component areas, with some sequencing of subtopics and skills. For instance, a reading curriculum in the primary grades (K-3) should address component areas such as phonemic awareness, phonics,

fluency, vocabulary, and comprehension; within a component area, the curriculum would address easier skills before more difficult ones. In the area of phonics, for example, children would be taught to read simple, one-syllable words before two-syllable words, and two-syllable words before complex multisyllabic words. The term *curriculum* is not synonymous with *instructional program*. A curriculum could be implemented through the use of one particular instructional program or set of programs, but it could also be implemented through instructional activities developed by teachers or schools.

Historical Background

Some countries, such as the United Kingdom, have a national curriculum which standardizes specific course content by grade. Although the United States has no national curriculum, virtually all states have their own standards for important academic domains such as mathematics or reading. These state standards provide some guidance to local school districts about what state education officials view as important content for each grade level, K-12. Professional organizations and scholarly panels (e.g., the National Early Literacy Panel, the National Math Advisory Panel) also provide guidance to educators regarding important curriculum content. Nevertheless, K-12 curricula can vary substantially from one state to the next or even within a state, across districts, meaning that curricular expectations for children at a particular grade level also can vary substantially.

Future Directions

The Common Core State Standards Initiative (www.corestandards.org), a state-led effort coordinated by the National Governors Association Center for Best Practices (NGA Center) and the Council of Chief State School Officers (CCSSO), has outlined evidence-based standards by grade level for K-12 English/language arts and mathematics. States choosing to adopt these standards would be addressing similar skills and content in their core curricula, which might lead to more

consistency across and within states in expectations for students in each grade.

See Also

- [Reading](#)
- [Written Language](#)

References and Reading

- National Council for Teachers of Mathematics. (2000). *Curriculum and evaluation standards for school mathematics*. Reston: Author.
- National Early Literacy Panel. (2008). *Developing early literacy: The report of the National Early Literacy Panel*. Jessup: National Institute for Literacy.
- National Mathematics Advisory Panel. (2008). *Foundations for success: The final report of the National Mathematics Advisory Panel*. Washington, DC: US Department of Education.
- National Reading Panel. (2000). *Teaching children to read: An evidence-based assessment of the scientific research literature on reading and its implications for reading instruction*. Washington, DC: National Institutes of Health.

Curvature of the Spine

- [Scoliosis](#)

Custodial Grandparents of Children with Autism Spectrum Disorder

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Definition

Custodial grandparents of children with autism spectrum disorder (ASD) serve as head of their

household; assume primary responsibility for at least one biological, adoptive, or step-grandchild diagnosed with ASD; and may or may not have their caregiving responsibilities recognized via legal adoption, guardianship, or formal custody arrangements. Families comprised of custodial grandparents and their grandchildren are called grandfamilies, kin care, custodial, and skip-generation families.

Historical Background

Research on custodial grandparents of children with ASD was predicated by general studies of US grandparents who lived in the same household as their grandchildren. Although the Census Bureau began to gather data on grandparents who lived in the same household as their grandchildren in the 1970s, a lack of data regarding the individual characteristics of those grandchildren, coupled with multiple changes in autism's diagnostic nomenclature, has made it difficult, if not impossible, to discern the number of custodial grandparents taking care of a child with ASD.

In 1970, the US Census Bureau reported that 2.2 million grandparents lived in the same household with a grandchild, but it was not possible to determine if those grandparents served as primary caregiver for their grandchild or if they co-resided with additional members of the child's family including the child's parent or parents. Over the next few decades, increasing numbers of grandparents "lived under the same roof" as their grandchildren; by 1997 more than 3.9 million grandparents lived with a grandchild (see Hayslip and Kaminski 2005). Meanwhile, while the 1994 *Diagnostic and Statistical Manual of Mental Disorders* (DSM), 4th edition, identified childhood autism as a developmental disorder that included individual diagnoses of Asperger's disorder, PDD-NOS, autism, childhood disintegrative disorder, and Rett syndrome, the Census Bureau did not collect enough data to determine how many of those 3.9 million grandparents served as custodial, versus co-resident, grandparents, much less custodial grandparents of a child with autism (i.e., ASD).

By the turn of the century, the Census Bureau (1999) collected data that allowed for the

identification of custodial, as well as co-resident, grandparents and reported that of the 4.6 million US grandparents who co-resided with a grandchild, 1.3 million of them served as custodial grandparents. These custodial, compared to co-resident, grandparents were also more likely to be grandmothers, to live in poverty, and to not have health insurance for their grandchild (1999). When the DSM-5 (APA) introduced ASD as an umbrella diagnosis for autism in 2013, it was still not possible to obtain a population count of custodial grandparents of children with ASD via the Census Bureau, and no formal research studies of custodial grandparents of children with ASD existed in the literature. However, recognized experts in grandparenting like Hayslip began to examine the custodial grandparents of children, in general. A review of this early literature on custodial grandparents, in general, revealed five primary themes including the heterogeneity of this population, the personal costs and benefits associated with assuming this role, the use of various parenting styles, a desire for social support and connection, and a need for essential social and health services (Hayslip and Kaminski 2005).

Current Knowledge

It remains impossible to obtain an accurate count of the number of custodial grandparents of children with ASD in the USA; no governmental or private organizations gather the appropriate data to make that official determination. One can, however, estimate how many grandparents currently serve as custodial grandparents for children with ASD in the USA. The Census Bureau indicates that 2.7 million custodial grandparents in the USA currently provide care for 2.5 million grandchildren (Cancino 2016). When combined with current prevalence rates of ASD, in which 1 out of 58 US children receive a diagnosis of ASD (Autism and Developmental Disabilities Monitoring Network 2018), 43,103 of those 2.5 million children cared for by custodial grandparents could be estimated to carry an ASD diagnosis. Because additional census data indicate that custodial grandparents care for grandchildren at a ratio of 1.08 grandparents to 1 grandchild

(Cancino 2016), those 43,103 children diagnosed with ASD would be expected to be cared for by a total of more than 46,500 US custodial grandparents.

Our knowledge of the estimated, more than 46,500, custodial grandparents in the USA currently caring for children with ASD remains limited. Important reference groups for the custodial grandparents of children with ASD include the parents of children with ASD; the parents of children with other disabilities and chronic illnesses; the custodial grandparents of children, in general; and the traditional grandparents of children with ASD. A review of these reference groups will provide useful background information and points of comparison for it currently knows about custodial grandparents of children with ASD.

The parents of children with ASD serve as an essential reference group for custodial grandparents of children with ASD; the custodial grandparents of children with ASD, who assume the role of surrogate parent, can be expected to share similar experiences, challenges, and demands (Hillman and Anderson 2019). It also is essential to note that the parents of children with ASD, compared to the parents of children with other kinds of chronic illness and disabilities (e.g., hearing and vision loss, Down syndrome, cerebral palsy, intellectual disability, and diabetes), experience a variety of unique, additional stressors (see Hillman and Anderson 2019). These stressors include the challenging behavioral and communication problems typically exhibited by children with ASD like elopement, tantrums, meltdowns, insomnia, sensory sensitivity, stimming (e.g., hand flapping, headbanging), an insistence upon sameness, physical aggression, restricted and unusual interests, restricted food choices, and difficulty with verbal and non-verbal communication. Additional, unique challenges for parents of children with ASD, compared to parents of children with other chronic disorders and illnesses, include difficulties in obtaining appropriate ASD-related services, transporting their child to a variety of appointments, and obtaining daycare and respite care, social isolation, and the need to spend more time interacting with insurance companies, school personnel, and various agencies to acquire appropriate services (Cidav et al. 2012).

Among parents of children with ASD, one parent (typically the mother) often takes on the role of the personal case manager, advocates for the child with ASD, and assumes primary responsibility for transportation to ASD-related appointments, management of ASD-related problem behaviors, and before-and-after-school, evening, weekend, holiday, and summer childcare. Because many of these primary caregivers often lose the ability to work a typical workweek outside of the home, mothers of children with ASD earn 56% less than mothers of children in general and 35% less than mothers of children diagnosed with other chronic illnesses and disorders (Zuleyha et al. 2012). This decrease in household income only adds to the increased financial burden of parents of children with ASD who typically need to cover the cost of ASD-related therapies, medical care, specialized school instruction, summer programs, and day and respite care (Cidav et al. 2012).

Parents of children with ASD face additional stressors in their relationships with spouses and partners and often express fear regarding their child's future, particularly after their own deaths. These parents have expressed, "Who will love and care for my child with ASD after I pass away? Will my child be able to live independently, or will they be placed in a group home or institution? Who will make sure that they are cared for and not emotionally, physically or financially abused?" This combination of demands in caregiving, challenges with ASD-related problem behaviors and lost wages can lead parents of children with ASD to experience social isolation, anxiety, and depression (Dyches et al. 2016). It can be expected that the custodial grandparents of children with ASD would have a similar experience.

Another essential reference group for custodial grandparents of children with ASD is that of custodial grandparents of children, in general. In terms of providing long-term social benefits, custodial grandparents of all children prevent those children from entering an already typically underfunded, overworked, and insufficient foster care system (Hayslip et al. 2019). Despite providing this essential social service, however, research suggests that custodial grandparents of children,

overall, represent a disproportionate number of minority group members and are subject to a variety of challenges and stressors. For example, custodial grandparents often assume their role in response to a crisis related to a parent's divorce, death, incarceration, mental or physical illness, military deployment, substance abuse (e.g., increasingly frequent opioid use), or participation in child abuse and neglect (Hayslip et al. 2019). Custodial grandparents of children, overall, are also more likely to be African American, Latino, and grandmothers (Hayslip et al. 2019) living on a fixed income with a chronic illness (or mental or physical disability) and with less formal education (Muthiah et al. 2018) when compared to both partnered and single parents of children, in general.

Custodial grandparents of children, in general, also report feeling guilt, shame, insecurity, sadness, and frustration (Muthiah et al. 2018) within the context of their role, as well as anxiety and depression (Hayslip et al. 2019). This negative effect may arise from stressors including the crisis that led up to their caregiving, ambiguity about their role as grandparent versus parent, uncertainty about their parenting skills, a loss of independence and privacy, increased conflict with their partner or spouse, and increased spending for their grandchild's food, clothing, and other basic needs, challenges in finding same-age custodial peers, and a grandchild's problem behavior. However, custodial grandparents of children, in general (Hayslip et al. 2019), also appear to demonstrate resilience, and they have responded well to small group intervention. In response to feelings of social isolation, custodial grandparents, in general, who participated in both formal and informal support groups (Hayslip et al. 2019) experienced lower levels of stress and depression.

Understanding the experience of traditional, versus custodial, grandparents of children with ASD offers another point of comparison. Based upon the largest national survey of traditional grandparents of children with ASD to date, more than 1800 grandparents of children with ASD who responded provided both emotional and instrumental support to the parents of their grandchildren with ASD (Hillman et al. 2016). Specifically, traditional grandparents of children

with ASD reported that they often provide childcare so that a parent can work outside the home, respite care, transportation to and from ASD-related appointments, advocacy and assistance in dealing with school personnel and insurance agencies, and significant financial support well beyond paying for essentials like food and clothing. To provide this financial support, many of these grandparents made significant personal sacrifices and put off their retirement, went back to work after they had been retired or partially retired, raided their retirement and other savings accounts, and even moved in with, or closer to, their grandchild with ASD. Findings also indicated that traditional grandmothers of children with ASD were more likely to provide emotional and instrumental support, while traditional grandfathers of children with ASD were more likely to provide financial support.

Additional, qualitative analyses of the responses of more than 1800 traditional grandparents of a child with ASD regarding their first-person experience (Hillman et al. 2017) revealed that these grandparents experienced joys as well as challenges in their relationship with their grandchild with ASD. Specifically, these traditional grandparents enjoyed having a special relationship with their grandchild, which often transcended their grandchild's inability to communicate via spoken language. However, this special relationship was often tempered by ASD-related behavior problems like tantrums, meltdowns, elopement, aggressive behavior, and the desire to communicate verbally with a non-verbal grandchild. A central finding was that despite these challenges, these traditional grandparents of children with ASD typically reported that they found solace and joy in celebrating every bit of progress that their grandchild made, no matter how small (Hillman et al. 2017). Additional themes identified by these traditional grandparents of children with ASD included a newly discovered ability to love their grandchild unconditionally and the value they typically found by engaging in ASD-related advocacy. Additional qualitative findings (Prendeville and Kinsella 2019) indicate that although traditional grandparents of children with ASD also experience regret losing some of their personal freedom

when they commit to helping with their grandchild's daily care, they knew they provided an invaluable service. Unlike the parents of children with ASD, however, the traditional grandparents of children with ASD (Hillman et al. 2017; Prendeville and Kinsella 2019) appear to experience a "double dose" of worry and fear in which they express significant fear and anxiety about both the future of their grandchild with ASD and their grandchild's parents, which increased when they thought about becoming too sick or frail to help or passing away.

Although limited, the research available regarding custodial grandparents of children with ASD is drawn primarily from qualitative studies (e.g., Hillman and Anderson 2019). Findings suggest that, like traditional grandparents of children with ASD, the custodial grandparents of a child with ASD experience both challenges and joys in their dual, and often conflicting, role as grandparent and surrogate parent. Specifically, custodial grandparents of children with ASD reported that problematic issues with their grandchildren's parents (i.e., their adult child and/or their adult child's partner) serve as one of their greatest challenges. Challenges include dealing with the original crisis that led to them assuming their role as primary caregiver, obtaining legal custody of their grandchild with ASD, and conflicts that often arose during visitation with the child's parent or parents. The custodial grandparents of children with ASD also reported that caregiver burden, including 24/7 demands, challenging ASD-related problem behaviors like tantrums and eloping, insufficient ASD-related services, increasing financial demands, social isolation, and related fears for their grandchild's future, represented another significant challenge.

However, custodial grandparents of children with ASD also report that they experience joy and positivity in their role (e.g., Hillman and Anderson 2019). Sources of joy include learning to love unconditionally; to celebrate each time their grandchild with ASD made progress, no matter how small or incremental; to focus on the positive; and to draw upon their religious and moral beliefs (e.g., my grandchild is a gift from God, and taking care of him is the right thing to do) during difficult times. Custodial grandparents

of children with ASD also report developing wisdom as a function of their role, including the acknowledgment that it really does "take a village" including friends, teachers, and various professionals to raise a child with ASD and the importance of advocacy and education about ASD for their both own grandchild and for society as a whole.

In sum, research suggests that custodial grandparents of children with ASD face significant stressors and burden, above and beyond those of parents of children with ASD, caregivers of children with other chronic illnesses and disabilities, custodial grandparents of children overall, and traditional grandparents of children with ASD. The custodial grandparents of children with ASD often face additional struggles with living on a fixed or limited income, feeling conflicted about their dual role as parent and grandparent, having few peers, having their own health problems, experiencing a "double dose" of worry for both their grandchild with ASD and their grandchild's parents, and not having their own grandparents to help them with finances or respite care.

Because custodial grandparents of children with ASD have identified social isolation, needing help with ASD-related problem behaviors, around-the-clock demands for caregiving, and financial strain as primary stressors (Hillman and Anderson 2019), the use of online resources available 24-7 from the caregiver's home or portable cell phone (as long as they have Internet access) appears appropriate. Exemplars of potential online resources for custodial grandparents of children with ASD are provided in Table 1. It is notable that *Autism Speaks*, a national nonprofit organization designed to offer advocacy, research, and support for families, was founded in 2005 by two traditional grandparents of a child with ASD who described feeling lost, without adequate resources, when they first learned about their grandson's diagnosis.

Future Directions

Significant gaps in the literature regarding custodial grandparents of children with ASD will be identified here in the hope that they could be

Custodial Grandparents of Children with Autism Spectrum Disorder, Table 1 Websites for custodial grandparents of children with ASD

AARP, Raising Grandchildren: Finances, https://www.aarp.org/relationships/friends-family/info-08-2011/grandfamilies-guide-finances.html
Asperger/Autism Network, How to Handle Meltdowns, https://www.aane.org/how-to-handle-meltdowns/
Autism Speaks, A Grandparent's Guide to Autism, https://www.autismspeaks.org/tool-kit/grandparents-guide-autism
CAR Autism Roadmap, The Children's Hospital of Philadelphia Research Institute, https://www.carautismroadmap.org/resources/
Generations United, Grandfamilies, https://www.gu.org/explore-our-topics/grandfamilies/
Grandparent Autism Network, https://ganinfo.org/
Grandparents Raising Grandchildren, https://www.helpguide.org/articles/parenting-family/grandparents-raising-grandchildren.htm
National Resources for Grandfamilies, Grandfamilies, http://www.grandfamilies.org/
Protections and Advocacy Systems, Inc., https://www.wypanda.com/
Understood, When Grandparents Are Caregivers or Guardians of Kids With Learning and Attention Issues, https://www.understood.org/en/family/managing-everyday-challenges/issues-with-caregivers-babysitters/when-grandparents-are-caregivers-or-guardians-of-kids-with-learning-and-attention-issues

addressed with future research, practice, and policy changes. For example, it would be helpful to obtain an accurate count of the custodial grandparents of children with ASD in the USA and around the world, including their demographic characteristics. Because most research regarding the custodial grandparents of children with ASD available has been conducted with convenience samples, snowball sampling, and primarily US, White, well-educated custodial grandparents, research derived from nationally representative samples would be ideal. Consistent with calls to acknowledge the heterogeneity of custodial grandparents of children, in general (Hayslip et al. 2019), it also remains essential to acknowledge the heterogeneity of custodial grandparents of children with ASD. Additional research can focus upon the experience and needs of custodial grandparents of children with ASD from various understudied and likely underserved groups including immigrants, LGBT adults, and African American, Alaskan Native, American Indian, Asian, Latino, Middle Eastern, and Pacific Islander adults.

Longitudinal studies of custodial grandparents of children with ASD are needed, and both quantitative and qualitative approaches to research should be valued. Consistent with current research on custodial grandparents (Hayslip et al. 2019) of children with ASD (Hillman and Anderson 2019),

future investigations should continue to examine and highlight custodial grandparents' resilience, wisdom, and other personal strengths, as well as their significant challenges. Future research should also investigate the extent to which custodial grandparents of children with ASD might use alcohol and other substances for coping, be physically abused or injured by their grandchild with ASD, and engage in child abuse, themselves. Because societal attitudes toward grandparents who serve as primary caregiver for their grandchildren have traditionally been negative (see Hayslip et al. 2019), it would be helpful to examine the attitudes maintained among case managers, teachers and other school personnel, judges and attorneys, daycare providers, and other individuals who might work with custodial grandparents of children with ASD. It also would be helpful to examine the extent to which custodial grandparents, versus parents, of a child with ASD approach that child's transition to adulthood including their pursuit of job skills, higher education, and options for group and independent living outside the family home.

In terms of policy and practice, a variety of recommendations can be offered to help address the needs of custodial grandparents of children with ASD (e.g., Hillman et al. 2019). Because these grandparents have identified problems with establishing legal custody of their

grandchild with ASD, as well as managing parental contact and behavior during visitation, legal advocates and care providers can offer education and assistance in relation to legal custody, guardianship, visitation rights, permissive visitation, and supervised visitation, which can vary significantly by state. Individual states can sponsor and promote kinship navigators, professionals trained specifically to connect custodial grandparents with state and local ASD- and custody-related resources including legal advocates in family and tax law. For example, many custodial grandparents of children with ASD may not know that they qualify for kinship guardianship payments, foster care payments, or state-supported child health insurance, much less how to apply for those benefits. Because custodial grandparents have expressed significant fears about who will take care of their grandchild after their death (Hillman et al. 2019), these grandparents can be directed to local, state, and national resources that assist in long-term care planning; the creation of a special needs trust and various custodial accounts can help ensure the quality of a child's life with ASD long after a custodial grandparent passes away.

Intergenerational programming for custodial grandparents and their grandchild with ASD could also be offered by local Area Agencies on Aging (e.g., senior centers) and Title VI Native American aging programs, perhaps in conjunction with professionals and student trainees from local colleges and universities. Such intergenerational programming could introduce these custodial grandparents to everything from case management services, legal advocacy for custody and estate planning, the National Family Caregivers Support Program, management of ASD-related problem behaviors, nutrition and feeding services, financial support (e.g., potential kinship care grants, Supplemental Security Income), and in-person and online peer support groups, to state supported respite care and transportation services when available. Ideally, such intergenerational programs would feature simultaneous programming for the grandparents' children with ASD including childcare, social skills training, music and art therapy, and other activities

and services. Addressing the challenges faced by custodial grandparents of children with ASD, while supporting their resilience and benefitting from their wisdom, appears essential.

See Also

- Advocacy
- Applied Behavior Analysis (ABA)
- Asperger Syndrome
- Autism Speaks
- Behavior
- Challenging Behavior
- Child Abuse in Autism
- Court Decision (ASD Related)
- Developmental Disabilities
- Disability
- Estate Planning
- Family Burden
- Family Therapy
- Guardianship
- Health Disparities
- Maladaptive Behavior
- Pervasive Developmental Disorder Not Otherwise Specified
- Qualitative Versus Quantitative Approaches
- Related Services
- Respite Care
- Self-Care
- Support Trust
- Team Approach

References and Reading

- Autism and Developmental Disabilities Monitoring Network Surveillance Year. 2014. Principal Investigators. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *Morbidity and Mortality Weekly Report (MMWR). Surveillance Summaries/CDC*, 67(6), 1–23.
- Cancino, A. (2016). More grandparents raising their grandchildren. Associated Press. Retrieved from <http://www.pbs.org/newshour/runtdown/more-grandparents-raising-their-grandchildren>
- Census Bureau. (1999). Coresident grandparents and grandchildren. *Current Population Reports*, P23–198, 1–10. Retrieved from <https://www.census.gov/prod/99pubs/p23-198.pdf>

- Cidav, Z., Marcus, S. C., & Mandell, D. S. (2012). Implications of childhood autism for parental employment and earnings. *Pediatrics*, 129(4), 617–623.
- Dyches, T. T., Christensen, R., Harper, J. M., Mandleco, B., & Roper, S. O. (2016). Respite care for single mothers of children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46(3), 812–824.
- Hayslip, B., & Kaminski, P. L. (2005). Grandparents raising their grandchildren: A review of the literature and suggestions for practice. *The Gerontologist*, 45(2), 262–269.
- Hayslip, B., Fruhauf, C. A., & Dolbin-MacNab, M. L. (2019). Grandparents raising grandchildren: What have we learned over the past decade? *The Gerontologist*, 57(3), e152–e163.
- Hillman, J. L., & Anderson, C. M. (2019). It’s a battle and a blessing: The experience and needs of custodial grandparents of children with Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders*, 49(1), 260–269. <https://link.springer.com/article/10.1007%2Fs10803-018-3761-0>
- Hillman, J., Marvin, A., & Anderson, C. M. (2016). The experience and contributions of grandparents of children with autism spectrum disorders. *Journal of Intergenerational Relationships*, 14(2), 76–92.
- Hillman, J., Wentzel, M. C., & Anderson, C. M. (2017). Grandparents’ experience of Autism Spectrum Disorder: Identifying primary themes and needs. *Journal of Autism and Developmental Disorders*, 47(10), 2957–2968. <https://link.springer.com/article/10.1007%2Fs10803-017-3211-4>
- Muthiah, N., Keim, S., & Adesman, A. (2018). *Grandparents raising grandchildren: Are they up to the job?* Paper presented at the Conference of the American Academy of Pediatrics, Orlando.
- Prendeville, P., & Kinsella, W. (2019). The role of grandparents in supporting families of children with Autism Spectrum Disorders: A family systems approach. *Journal of Autism and Developmental Disorders*, 49(2), 738–749. <https://link-springer-com.ezaccess.libraries.psu.edu/article/10.1007%2Fs10803-018-3753-0>
- Volkmar, F. R., & Reichow, B. (2014). The evolution of Autism as a diagnostic concept: From Kanner to *DSM-5*: A commentary. In T. E. Davis III, S. W. White, & T. H. Ollendick (Eds.), *Handbook of Autism and Anxiety* (pp. 217–230). New York: Springer.
- Zuleyha, C., Marcus, S. C., & Mandell, D. S. (2012). Implications of childhood autism for parental employment and earnings. *Pediatrics*, 129(4), 617–623.

CVLT – Children’s Version

- [California Verbal Learning Test, Children’s Version \(CVLT-C\)](#)

CVLTC

- [California Verbal Learning Test, Children’s Version \(CVLT-C\)](#)

CVLT-C

- [California Verbal Learning Test, Children’s Version \(CVLT-C\)](#)

Cyclohexitol

- [Inositol: Definition](#)

Cylert

- [Pemoline](#)

Cymbalta

- [Duloxetine: Definition](#)

D

Daily Activities

► Daily Routines

Daily Living Skills

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Synonyms

Activities of daily living; Daily self-care activities; Home living skills; Self-care; Self-help

Definition

The term “daily living skills” refers to a wide range of personal self-care activities across home, school, work, and community settings. Most daily living skills, like food preparation and personal hygiene, need to be performed on a regular basis to maintain a reasonable level of health and safety. Adaptive functioning, or an individual’s ability to care for self and function independently, is a primary consideration

when supporting individuals with autism and other disabilities. Daily living skill activities include:

- Personal hygiene and grooming
- Dressing and undressing
- Meal preparation and feeding
- Mobility and transfer
- Toileting
- Housekeeping
- Laundry
- Home safety
- Health and medication management
- Leisure time and recreation

Children’s abilities to care for themselves have been found to correlate with intellectual functioning and may be a strong predictor of future independence (Carter et al. 1996). Individuals who cannot independently carry out these necessary self-help routines are at greater risk for long-term institutionalization, require more intensive living supports, and are less likely to be employed (Wehman and Targett, 2004). Assessing adaptive functioning is required when measuring intelligence, diagnosing intellectual disability, and determining appropriate treatment goals (Goodlin-Jones and Solomon 2003). The most widely used instrument to assess adaptive behavior functioning is the Vineland Adaptive Behavior

Scales (Sparrow et al. 1984). Selecting skills to teach should focus on the priority tasks required for independent domestic and community living (Wehman and Targett 2004; National Research Council 2001). Daily living skills are usually taught using strategies from applied behavior analysis, specifically task analysis, shaping, chaining, and positive reinforcement. Teaching individuals with autism to generalize learned tasks across settings, people, and materials remains an important aspect of intervention planning when teaching daily living skills.

See Also

- ▶ [Adaptive Behavior](#)
- ▶ [Adaptive Behavior Scales](#)
- ▶ [Chaining](#)
- ▶ [Functional Assessment and Curriculum for Teaching Everyday Routines](#)
- ▶ [Functional Life Skills](#)
- ▶ [Independent Living](#)
- ▶ [Positive Reinforcement](#)
- ▶ [Task Analysis](#)
- ▶ [Vineland Adaptive Behavior Scales \(VABS\)](#)

References and Reading

- Carter, A. S., Gillham, J. E., Sparrow, S. S., & Volkmar, F. R. (1996). Adaptive behavior in autism. *Mental Retardation*, 5, 945–960.
- Goodlin-Jones, B. L., & Solomon, M. (2003). Contributions of psychology. In S. Ozonoff, S. J. Rogers, & R. L. Hendren (Eds.), *Autism spectrum disorders: A research review for practitioners* (pp. 55–85). Washington, DC: American Psychiatric Publishing.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Sparrow, S., Balla, D., & Cicchetti, D. (1984). *Vineland adaptive behavior scales*. Circle Pines: American Guidance Service.
- Wehman, P., & Targett, P. S. (2004). Principles of curriculum design: Road to transition from school to adulthood. In P. Wehman & J. Kregel (Eds.), *Functional curriculum for elementary, middle, and secondary age students with special needs* (2nd ed., pp. 1–36). Austin: Pro-Ed.

Daily Routines

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Synonyms

[Activity schedules](#); [Daily activities](#); [Routine events](#); [Schedules](#); [Visual schedule](#)

Definition

Daily routine is a schedule, custom, or habit that is known to occur similarly on a daily frequency. Daily routines are often preferred by children and adults diagnosed with autism in order to structure their day and provide predictability. Daily routines can be inherently known by the individual without support or review by an outside person, or are scheduled out by another and presented verbally or visually. Visual schedules are often used to act as an aid in conveying the day's event and are often presented pictorially (as with picture icons) or in written form (as in a checklist). Consistent use of daily routines often helps reduce problematic behavior due to issues with transition from activity to activity. Daily routines can be expanded to teach and/or guide most events that occur daily on a large scale (i.e., activities to occur from morning to night) or for specific events (e.g., hand washing, putting away laundry).

See Also

- ▶ [Adaptive Behavior](#)
- ▶ [Daily Living Skills](#)
- ▶ [Functional Assessment and Curriculum for Teaching Everyday Routines](#)
- ▶ [Functional Life Skills](#)
- ▶ [Prompt Hierarchy](#)
- ▶ [Prompting](#)
- ▶ [Visual Schedule](#)
- ▶ [Visual Supports](#)

References and Reading

- Cohen, M. J., & Sloan, D. L. (2007). *Visual supports for people with autism: A guide for parents and professionals*. Bethesda: Woodbine House.
- Etzel, B. C., & LeBlanc, J. M. (1979). The simplest treatment alternative: The law of parsimony applied to choosing appropriate instructional control and errorless learning procedures for the difficult-to-teach child. *Journal of Autism and Developmental Disorders*, 9, 361–382.
- Krantz, P. J., & McClannahan, L. E. (2010). *Activity schedules for children with autism: Teaching independent behavior* (2nd ed.). Bethesda: Woodbine House.
- Lott, J. D., & Kroeger, K. A. (2004). Self-help skills in persons with mental retardation. In J. L. Matson, R. B. Laud, & M. L. Matson (Eds.), *Behavior modification for persons with developmental disabilities: Treatment and supports* (Vol. 2). New York: National Association for the Dually Diagnosed.

Daily Self-Care Activities

- [Daily Living Skills](#)

DAISI

- [Detection of Autism by Infant Sociability Interview](#)

DAMP

- [Deficits in Attention, Motor Control, and Perception](#)

DAMP Syndrome

- [Deficits in Attention, Motor Control, and Perception](#)

D-Amphetamine

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Synonyms

[Adderall](#); [Dexamfetamine](#)

Definition

D-amphetamine, or dextroamphetamine, is a psychostimulant primarily used for the treatment of ADHD and narcolepsy in children and adults. The drugs work by increasing the release of norepinephrine, serotonin, and dopamine from their storage sites within presynaptic nerve terminals while simultaneously also competing for reuptake by their respective transporters, thus preventing the neurotransmitters from being reuptaken effectively. This neurotransmitter release is facilitated by the activation of a membrane transport system that brings in extracellular amphetamine in exchange for pumping out the above neurotransmitters. D-amphetamine may also be a serotonin receptor direct agonist. While there is little to no substantial evidence for the effectiveness of D-amphetamine in treating ASD, use of the drug in ASD patients is high due to the high comorbidity of the disorder with ADHD. Side effects of this drug may include irregular heartbeat, headache, dizziness, weight loss, dry mouth, and others.

See Also

- [ADHD](#)
- [Amphetamine](#)

References and Reading

- Fleckenstein, A. E., Volz, T. J., Riddle, E. L., Gibb, J. W., & Hanson, G. R. (2007). New insights into the mechanism of action of amphetamines. *Annual Review of Pharmacology and Toxicology*, 47, 681–698.
- Pliszka, S. R. (1989). Effect of anxiety on cognition, behavior, and stimulant response in ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28(6), 882–887.
- Solanto, M. V., Arnsten, A. F. T., & Castellanos, F. (2001). *Stimulant drugs and ADHD: Basic and clinical neuroscience*. New York: Oxford University Press.
- Swanson, J. M., McBurnett, K., Christian, D. L., & Wigal, T. (1995). Stimulant medications and the treatment of children with ADHD. *Advances in Clinical Child Psychology*, 17, 265.

DAP:IQ

- [Human Figure Drawing Tests](#)

DAS

- [Differential Ability Scales \(DAS and DAS-II\)](#)

DAS-II

- [Differential Ability Scales \(DAS and DAS-II\)](#)

D-Cycloserine: Definition

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Synonyms

[Seromycin](#)

Definition

D-cycloserine is a partial glycine_B site agonist that binds to the strychnine-insensitive glycine binding site on the NMDA receptor, agonizing the NMDA receptor, a glutamate receptor subtype. The NMDA receptor functions in synaptic plasticity. Alterations in synaptic plasticity are believed to be at the crux of ASD pathogenesis. D-cycloserine has been shown to partially reverse social deficits in mouse models of autism. Similar benefits have been shown in human subjects as well, but further validation is still required. Because of its actions on the NMDA receptor, D-cycloserine is heavily studied in processes of fear conditioning and extinction as well as schizophrenia. In many of these studies, it is used in conjunction with behavioral therapy to allow greater control over any ensuing alterations in synaptic plasticity. Currently, D-cycloserine is only approved for the treatment of tuberculosis. Side effects include headache, drowsiness, dizziness, or shaking.

References and Reading

- Deutsch, S. I., Burket, J. A., Jacome, L. F., Cannon, W. R., & Herndon, A. L. (2011). D-cycloserine improves the impaired sociability of the Balb/c mouse. *Brain Research Bulletin*, 84(1), 8–11.
- Jacome, L. F., Burket, J. A., Herndon, A. L., & Deutsch, S. I. (2011). D-cycloserine enhances social exploration in the Balb/c mouse. *Brain Research Bulletin*, 85(3), 141–144.
- Modi, M. E., & Young, L. J. (2011). D-cycloserine facilitates socially reinforced learning in an animal model relevant to autism spectrum disorders. *Biological Psychiatry*, 70(3), 298–304.
- Posey, D. J., Kem, D. L., Swiezy, N. B., Sweeten, T. L., Wiegand, R. E., & McDougle, C. J. (2004). A pilot study of D-cycloserine in subjects with autistic disorder. *American Journal of Psychiatry*, 161(11), 2115–2117.
- Won, H., Lee, H. R., Gee, H. Y., Mah, W., Kim, J. I., Lee, J., . . . & Kim, E. (2012). Autistic-like social behaviour in Shank2-mutant mice improved by restoring NMDA receptor function. *Nature*, 486(7402), 261–265.

DDST

- [Denver Development Screening Test \(DDST\)](#)

de Lange Syndrome

► [Cornelia de Lange Syndrome](#)

Deaf-Blind

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Synonyms

[Sensory impairment](#)

Short Description or Definition

Deaf-blind individuals have varying degrees of a combination of both hearing and visual impairments. In the United States, the legal definition of blindness is 20/200 in the better eye. An individual with a threshold exceeding 90 dB HL is considered to be deaf. Individuals who are deaf-blind have communication as well as mobility deficits. This dual sensory impairment results in the inability to use one sensory modality to compensate for the other. Services required for individuals who are deaf-blind are different than those required for individuals who are either deaf or blind. Communication and language development are the primary deficits in individuals with deaf-blindness; however, development of social-affective, cognitive, and motor skills is also affected. Individuals with deaf-blindness also exhibit stereotyped behaviors, similar to those seen in children with autism spectrum disorders.

Categorization

Deaf-blindness may be congenital or acquired resulting in a heterogeneous population. It is important to differentiate between these two

groups; those with congenital deaf-blindness have additional handicaps and typically require a substantially greater amount of rehabilitation, including programs that are individually tailored (Rönnerberg and Borg 2000).

Epidemiology

It is estimated that approximately 10,000 children (ages birth to 22 years) in the United States are classified as deaf-blind (Rönnerberg and Borg 2000; The National Consortium on Deaf-Blindness [NCDB] 2008). The adult deaf-blind population numbers are estimated at 35–40,000 individuals (Watson and Taff-Watson 1993).

Congenital deaf-blindness may be caused by hereditary or chromosomal syndromes and disorders, prenatal or congenital complications, complications of prematurity, and undiagnosed causes. Some common hereditary or chromosomal causes are CHARGE syndrome, Usher syndrome, and Down syndrome. Cytomegalovirus (CMV) and microcephaly are some prenatal or congenital complications that may lead to deaf-blindness. In the past, maternal rubella was the leading cause of deaf-blindness. The majority of cases of deaf-blindness are acquired; a variety of causes are responsible such as meningitis, inflicted brain damage, and aging of the sensory organs (Rönnerberg and Borg 2000).

There have been very few reports on the combined disorders of autism and deaf-blindness. The prevalence of deaf-blind individuals with autism is unknown, although it is estimated to be small. Further, because etiological factors and symptoms such as impaired social interaction and communication impairment are associated with both disorders, it is challenging to differentially diagnose between the two.

Natural History, Prognostic Factors, and Outcomes

The history of service for the population of individuals with deaf-blindness is sharply divided between the pre- and post-rubella epidemic of

1964–1965. The first citations of education with this population appeared in the mid-1800s with Laura Bridgman described as the first deaf-blind individual to learn language at the Perkins School for the Blind. Helen Keller was an even more recognized and influential figure in the success of educating deaf-blind individuals. However, even through the 1960s, limited education was available for this population, and individuals were often placed in residential schools or asylums. Congressional legislation, approved in the 1970s and beyond, which mandated education for children with disabilities, had a significant effect in advancing the education of this population (NCDB 2012).

Clinical Expression and Pathophysiology

This severe sensory deficit results in communication disorders and subsequent handicaps in education, social and cultural development, and the acquisition of information. The tactile sense is commonly utilized by these individuals for communication as well as for feelings of security and control. Other methods of compensation include use of the cutaneous senses and vibration for sound localization. Few studies have explored the psychosocial aspects of being deaf-blind and those that do typically focus on adaptation. Depression in adolescents and psychosis has been reported (Rönnerberg et al. 2002).

Evaluation and Differential Diagnosis

The evaluation process for deaf-blindness is focused on determining the extent to which the auditory and visual systems are impaired. Since the characteristics of deaf-blindness are similar to those of autism spectrum disorders, determining if comorbid autism spectrum disorders exist can be challenging. These individuals are also typically difficult to test; therefore, identification is further complicated and standardized tests are nonexistent (Vernon 2010). There tends to be an

overdiagnosis of autism in individuals with deaf-blindness leading to unsuitable intervention (Hoevenaars-van den Boom et al. 2009). These authors studied 10 individuals with deaf-blindness and intellectual disability in order to determine if they could differentiate which of these individuals also had autism (which had been previously diagnosed). They utilized an instrument that they had developed specifically for this purpose. Results indicated the presence of a significantly greater number of impaired behaviors among the individuals with autism in reciprocity of social interaction, quality of initiatives to contact, and use of adequate communicative signals and functions. The authors concluded that their instrument has promise in terms of its utilization in identifying individuals with autism within the deaf-blind population. Operant conditioning techniques have also been used successfully in the assessment of this population (Rönnerberg and Borg 2000).

Treatment

Treatment for deaf-blindness is typically focused on improving the communication, self-help skills, and mobility of the individual. Since great variability exists from individual to individual, it is imperative to establish the degree to which either the auditory system, visual system, or both can be utilized to enhance communication. The predominant therapeutic model is behavior modification (Rönnerberg and Borg 2000). It is recommended that the curriculum addresses five main areas: (1) communication skills, (2) cognitive development, (3) social and emotional development, (4) motor and self-care skills, and (5) sensory development (Murdoch 1986).

Research conducted on communicative and linguistic treatment primarily focuses on the “Tadoma” method in which the Tadoma user places his/her hand on the speaker’s face in a proscribed position. Through the use of perceptual cues, skilled Tadoma users are able to achieve a relatively high level of comprehension

of spoken speech. Intelligibility of their speech production is 60–70%; however, the rate of speech is slower by about 50%. Other methods of communication include the T-code, sign language, and textured symbols (Rönnberg and Borg 2000).

See Also

- ▶ Blindness
- ▶ CHARGE Syndrome
- ▶ Deafness
- ▶ Sensory Processing
- ▶ Sensory Stimuli

References and Reading

Carvill, S. (2001). Sensory impairments, intellectual disability and psychiatry. *Journal of Intellectual Disability Research*, 45, 467–483.

Frith, U. (2003). *Autism: Explaining the enigma*. Malden: Blackwell Publishing.

Gense, M. H., & Gense, D. J. (2005). *Autism spectrum disorders and visual impairment*. New York: American Foundation for the Blind Press.

Hoevenaars-van den Boom, M., Antonissen, A., Knoors, H., & Vervloed, M. (2009). Autism in deaf-blindness. *Journal of Intellectual Disability Research*, 53(6), 548–558.

McKay, V. (2010). Deaf-blindness and autism spectrum disorder. *Journal of American Deafness and Rehabilitation Association*, 44(1), 201–213.

Murdoch, H. (1986). Helping the deaf-blind child in class. *British Journal of Special Education*, 13, 75–77.

Murdoch, H. (2000). Repetitive behaviours in children with sensory impairments and multiple disabilities. *DBI Review*, 26, 7–11.

Rönnberg, J., & Borg, E. (2000). A review and evaluation of research on the deaf-blind from perceptual, communicative, social and rehabilitative perspectives. *Scandinavian Audiology*, 30(2), 67–77.

Rönnberg, J., Samuelsson, E., & Borg, E. (2002). Exploring the perceived world of the deaf-blind: On the development of an instrument. *International Journal of Audiology*, 41, 136–143.

The National Consortium on Deaf-Blindness. (2008). *2007 National child count of children and youth who are deaf-blind*. Monmouth: Teaching Research Division. National Consortium on Deaf-Blindness. Retrieved January 2012 from <http://www.nationaldb.org/>

Van Dijk, J., & Janssen, M. (1993). Deafblind children. In H. Nakken (Ed.), *Multi-handicapped children* (pp. 34–73). Rotterdam: Lemniscaat.

Watson, D., & Taff-Watson, M. (Eds.). (1993). *A model service delivery system for persons who are deaf-blind* (2nd ed.). Arkansas: University of Arkansas.

Deafness

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Synonyms

Profound hearing impairment

Short Description or Definition

“Deafness” is a term that has varying definitions but is characterized by severe-to-profound deficits in the ability to hear. Deaf with a capital “d” is a term used to describe individuals with severe-to-profound hearing loss resulting in little to no usable hearing, even with the use of amplification devices (i.e., hearing aids, assistive listening devices, etc.). Furthermore, individuals who are Deaf belong to the Deaf culture which uses American Sign Language (ASL) as their primary mode of communication. Deaf with a lowercase “d” refers to individuals with severe-to-profound hearing loss who use amplification devices and use oral communication as their primary mode of communication.

Categorization

Degrees of Hearing Impairment in dB HL

Normal	–10 to +15
Minimal	16–25
Mild	26–40
Moderate	41–55

(continued)

Moderately severe	56–70
Severe	71–90
Profound	91+

Epidemiology

According to the National Institute of Deafness and Other Communication Disorders (NIDCD 2010), approximately 2–3 in every 1,000 children born in the United States are born with deafness or some degree of hearing impairment. Hearing loss occurs in approximately 18% of 45- to 64-year-olds, 30% of 65- to 74-year-olds, and 47% of 75-year-olds and older. More systematic research needs to be done regarding the incidence and prevalence of deafness in the population with autism.

Natural History, Prognostic Factors, and Outcomes

Individuals with severe-to-profound hearing loss are not able to hear speech sounds and most environmental sounds without amplification. If individuals with this degree of hearing loss are not treated in the first year of life, severe speech and language delays may occur. Furthermore, learning and attention disorders may often arise. Individuals with severe-to-profound hearing impairment likely need hearing aids or cochlear implants, speech and auditory training/therapy, and special education services.

Prior to the onset of the Early Detection and Intervention (EDHI) program in the late 1980s/early 1990s, children with hearing impairment were not typically diagnosed until age 2 or 3 years when speech and language delays were apparent. Since the beginning of the EDHI program, children with hearing impairment are being identified and treated earlier which is important for speech and language development (either spoken language or American Sign Language (ASL)). Early detection and intervention are critical because a sensitive period exists for speech and language

development. The first few years of life is when the foundation for speech and language is established, and if this period is missed due to an unidentified severe-to-profound hearing loss, the child will not acquire speech and/or language. The development of oral speech and language is possible with appropriate amplification or cochlear implantation in conjunction with speech and language therapy.

In postlingually deafened adults, appropriate amplification is critical to their ability to communicate with spoken language. In order to perceive what is being said, as well as monitor what their own speech, these individuals need to be fit with hearing aids, and/or assistive listening devices, or with cochlear implants.

Clinical Expression and Pathophysiology

Deafness occurs as a result of a sensorineural or mixed (conductive and sensorineural) hearing loss. Conductive hearing losses are those that occur due to pathology in the outer or middle ear. Conductive hearing losses alone only result in at most; moderate hearing losses, however, in conjunction with sensorineural hearing losses can result in severe-to-profound hearing loss. Sensorineural hearing losses occur as a result of pathology (typically hair cell loss) in the cochlea or auditory nerve fibers.

Deafness can be either congenital or acquired. Congenital deafness can be the result of genetic factors, maternal illness, and/or infection. Some examples of syndromes associated with hearing impairment are CHARGE syndrome, Usher syndrome, and Waardenburg’s syndrome. Examples of maternal illness and/or infection include rubella, cytomegalovirus (CMV), diabetes, hypoxia, syphilis, and toxemia. Acquired deafness may be the result of ototoxic medications, infection, meningitis, and encephalitis, or the cause may be unknown. Depending on the cause of the hearing loss, the impairment may, or may not, be progressive in nature. Once hearing impairment is established, annual hearing evaluations are generally recommended.

Evaluation and Differential Diagnosis

The goal of hearing evaluations is to assess the outer, middle, and inner ears. The audiologist will first perform otoscopy to determine if the outer ear (pinna and external auditory meatus) and tympanic membrane have normal appearances. Then tympanometry is completed to determine the status of the middle ear. Finally, behavioral and/or electrophysiologic testing is completed to determine hearing sensitivity at the frequencies important for speech. In behavioral testing, the goal is to complete a pure-tone audiogram which is a graphical depiction of the hearing thresholds of the octave frequencies from 250 to 8,000 Hz. Speech reception threshold and word recognition testing are also done to determine threshold to speech stimuli as well as how accurately words are perceived.

The type of hearing evaluation one undergoes depends on a number of factors, including age and ability to respond to the tonal and speech stimuli. Evaluation methods can be either behavioral or electrophysiologic. Behavioral tests require the listener to respond in some way to the tonal or speech stimuli (i.e., raise hand, turn head, repeat back words, etc.). Some examples of behavioral test procedures are behavioral observation audiometry (BOA), visual reinforcement audiometry (VRA), conditioned play audiometry, and standard audiometry. Behavioral observation audiometry occurs when the audiologist plays tonal and speech stimuli through the sound field or headphones and then watches for a response from the individual. This response might be the cessation of crying or cooing, eyes widening, or turning the head. Visual reinforcement audiometry occurs when lighted puppets positioned in boxes directly above the left and right speakers in the booth are illuminated when the stimulus is presented. This is done repeatedly until the individual is trained to look in the direction of the stimulus. During the actual testing, the stimuli are presented and the light is turned on only after the individual turns and looks toward the light. In conditioned play audiometry, hearing thresholds are obtained by using toys such as blocks. For example, the individual is trained to drop a block in a bucket every

time they perceive the beeping sound. Finally, hearing thresholds using standard audiologic procedures are obtained by having the individual raise their hand or push a button every time they perceive the tonal stimuli.

Physiologic tests, like the otoacoustic emissions (OAEs) and auditory brainstem response (ABR), do not require a behavioral response from the listener and are thus commonly used in newborn hearing screenings, infant hearing tests, as well as hearing tests on individuals who are unwilling, or unable, to respond to behavioral tests. Otoacoustic emissions are generated by the hair cells in the cochlea, so if the hair cells are absent or not functioning properly, the otoacoustic emissions will be absent or reduced. Otoacoustic emissions are often used along with ABR in populations, such as those with autism, that cannot participate in behavioral testing, to differentially diagnose cochlear hearing loss from neural hearing loss.

Treatment

In Deaf populations, no “treatment” is sought since deafness is not considered a problem. Individuals who are Deaf are taught American Sign Language (ASL) and become immersed in Deaf culture. These individuals use manual communication to interact in society.

Individuals who are deaf will often use hearing aids and/or assistive listening devices. With the advancement in technology, individuals who do have a severe-to-profound hearing loss and who do not receive benefit from amplification may get cochlear implants. Children as young as 12 months can receive cochlear implant surgery. Either method (hearing aids or cochlear implantation) must be combined with speech and language therapy in order to train the system to listen as well as produce intelligible speech.

Prognosis, evaluation methods, and treatment of individuals with autism and deafness are contingent upon a number of factors. Some of these factors include the severity of autism, etiology of the hearing loss, comorbid disorders, mode of communication, and candidacy for hearing aids

and/or cochlear implants. Ultimately, a collaborative approach should be taken when treating individuals with autism and deafness.

See Also

- ▶ American Sign Language (ASL)
- ▶ Auditory Brainstem Response (ABR)
- ▶ Auditory System
- ▶ Cochlea
- ▶ Hearing

References and Reading

- Justice, L. (2006). *Communication sciences and disorders: An introduction*. Columbus: Pearson.
- NIDCD. (2010). Quick statistics. Retrieved from <http://www.nidcd.nih.gov/health/statistics/quick.htm>
- Northern, J., & Downs, M. (2002). *Hearing in children* (5th ed.). Philadelphia: Lippincott Williams & Wilkins.

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Major Appointments (Institution, Location, Dates)

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Major Honors and Awards

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Distinguished Contribution to the Science of Psychology award, Connecticut Psychological Association, 2012
Edith Kaplan Award for Outstanding Contributions to Neuropsychology, 2012

Landmark Clinical, Scientific, and Professional Contributions

Deborah Fein has published more than 150 articles and chapters on autism spectrum disorder (ASD) since the late 1970s. Her major contributions span a number of areas, including characterizing children who show optimal outcomes after diagnosis and treatment for autism, screening to promote early detection of autism, developing theoretical models of autism, and writing seminal papers on topics such as the role of oxytocin in autism. Dr. Fein's elucidation of the primacy of social deficits in autism, described in a 1986 paper in the *Journal of the American Academy of Child Psychiatry*, as well as her theories on the neuropsychological underpinnings of autism, seen in her 1996 paper in *Psychological Review*, and her edited volume, *The Neuropsychology of Autism*, as well as her recent honor as the Birch Memorial Lecture speaker at the 2015 annual meeting of the International Neuropsychological Society, have disseminated her work to a broad audience. Her contributions to early screening, as a co-author of the Modified Checklist for Autism in Toddlers (M-CHAT), and the M-CHAT, Revised, with Follow-Up (M-CHAT-R/F) significantly impacted the early detection of autism. Her subsequent work examining the minority of children with a clearly documented history of autism, but no current symptoms, was ground breaking in its characterization of children who achieve this "optimal outcome" and no longer demonstrating deficits associated with their history of autism. Additionally, her work reaches beyond the

professional and academic audiences that access scientific journals, in her publication of a book for educators on adapting classroom strategies for children with autism, and her newest book offering parents activities to do at home with very young children at risk or diagnosed with autism. She is currently developing a website that will be available for free to parents, to teach them principles of behavioral teaching and stimulating attachment.

Short Biography

Dr. Deborah Fein, born on March 21, 1947, is a neuropsychologist who has focused much of her career studying autism. Her theoretical and empirical work in autism was heavily influenced by her training at the Boston Veteran's Affairs Hospital, under the mentorship of Dr. Edith Kaplan (with whom she published several versions of the Wechsler tests as a process instrument), and by a postdoctoral fellowship at BU School of Medicine with Dr. Al Mirsky. In addition to her scientific contributions, Dr. Fein has trained and mentored more than 50 trainees in autism and neuropsychology, many of whom now hold faculty positions in academic or medical institutions, facilitating her tremendous influence on the field. Dr. Fein lives in Western Massachusetts with her husband, and they have two daughters.

See Also

- [Modified Checklist for Autism in Toddlers \(M-CHAT\)](#)
- [Optimal Outcome](#)
- [Recovery in Autism](#)

References and Reading

Fein, D., & Dunn, M. A. (2007). *Autism in your classroom: A general educator's guide to students with autism spectrum disorders*. Bethesda: Woodbine House.

- Fein, D., Pennington, B., Markowitz, P., Braverman, M., & Waterhouse, L. (1986). Toward a neuropsychological model of infantile autism: Are the social deficits primary? *Journal of the American Academy of Child Psychiatry*, 25(2), 198–212.
- Fein, D., Barton, M., Eigsti, I. M., Kelley, E., Naigles, L., Schultz, R. T., et al. (2013). Optimal outcome in individuals with a history of autism. *Journal of Child Psychology and Psychiatry*, 54(2), 195–205.
- Fein, D., Helt, M., Brennan, L., & Barton, M. (2015). *The activity kit for babies and toddlers at risk: How to use everyday routines to build social and communication skills*. New York: Guilford Publications.
- Green, L., Fein, D., Modahl, C., Feinstein, C., Waterhouse, L., & Morris, M. (2001). Oxytocin and autistic disorder: Alterations in peptide forms. *Biological Psychiatry*, 50(8), 609–613.
- Hauck, M., Fein, D., Waterhouse, L., & Feinstein, C. (1995). Social initiations by autistic children to adults and other children. *Journal of Autism and Developmental Disorders*, 25(6), 579–595.
- Helt, M., Kelley, E., Kinsbourne, M., Pandey, J., Boorstein, H., Herbert, M., & Fein, D. (2008). Can children with autism recover? If so, how? *Neuropsychology Review*, 18(4), 339–366.
- Kaplan, E., Fein, D., Kramer, J., Delis, D. C., & Morris, R. (1999). *Wechsler intelligence scale for children—process instrument*. San Antonio: Psychological Corporation.
- Modahl, C., Green, L. A., Fein, D., Morris, M., Waterhouse, L., Feinstein, C., & Levin, H. (1998). Plasma oxytocin levels in autistic children. *Biological Psychiatry*, 43(4), 270–277.
- Obler, L. K., & Fein, D. E. (1988). *The exceptional brain: Neuropsychology of talent and special abilities*. New York: Guilford Press.
- Robins, D. L., Fein, D., Barton, M. L., & Green, J. A. (2001). The modified checklist for autism in toddlers: An initial study investigating the early detection of autism and pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 31(2), 131–144.
- Robins, D. L., Casagrande, K., Barton, M., Chen, C. M. A., Dumont-Mathieu, T., & Fein, D. (2014). Validation of the modified checklist for autism in toddlers, revised with follow-up (M-CHAT-R/F). *Pediatrics*, 133(1), 37–45.
- Sutera, S., Pandey, J., Esser, E. L., Rosenthal, M. A., Wilson, L. B., Barton, M., et al. (2007). Predictors of optimal outcome in toddlers diagnosed with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(1), 98–107.
- Waterhouse, L., Fein, D., & Modahl, C. (1996). Neurofunctional mechanisms in autism. *Psychological Review*, 103(3), 457.

Declarative Memory

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Synonyms

[Explicit memory](#)

Definition

Declarative memory is a type of long-term memory and is memory for facts, data, words, etc. Declarative memory can be divided into three categories: episodic, semantic, and lexical. Episodic memory includes memory for personal events or experiences. Episodic memory is primarily learned consciously and is linked to a certain time and place. Examples include specific events such as walking to the store or cooking dinner. Semantic memory refers to general knowledge or facts, independent of experience. Examples include facts about historical events or types of cars. Lexical memory is the knowledge for words. It has been observed that some individuals with autism have enhanced semantic and lexical memory abilities.

See Also

- ▶ [Episodic Memory](#)
- ▶ [Explicit Memory](#)
- ▶ [Memory](#)
- ▶ [Semantic Memory](#)

References and Reading

- Berger, K. S. (1998). *The developing person through the life span* (4th ed.). New York: Worth.
- Chapey, R. (2001). *Language intervention strategies in aphasia and related neurogenic communication disorders* (4th ed.). Philadelphia: Lippincott Williams and Wilkins.

- Rathus, S. A. (1997). *Essentials of psychology* (5th ed.). Fort Worth: Harcourt Brace.
- Tager-Flusberg, H. (1985). The conceptual basis for referential word meaning in children with autism. *Child Development*, 56, 1167–1178.
- Ullman, M. T. (2004). Contributions of memory circuits to language: The declarative/procedural model. *Cognition*, 92, 231–270.

Decoding Skills

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Synonyms

[Word recognition](#)

Definition

Decoding refers to the word recognition processes in which written words or print are transformed into spoken words. This process is commonly referred to as “sounding out words.” Proficient decoding requires rapid letter recognition, knowledge of sound-letter correspondences, phonemic awareness, and word attack skills (i.e., analysis/segmenting and synthesis/blending of the letter-sound correspondences). Accurate and fluent decoding allows for comprehension of words both in isolation and in context.

Many individuals with autism spectrum disorders (ASD) are able to mentally represent at least some single word meanings; that is, read words in isolation and understand their meanings. Some individuals with ASD spontaneously read words with excellent proficiency at an unexpectedly early age (referred to as hyperlexia); however, it is the ability to read beyond decoding individual words (i.e., reading in context) that presents greater difficulty for individuals with ASD.

References and Reading

- Huemer, S. V., & Mann, V. (2009). A comprehensive profile of decoding and comprehension in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(4), 485–493.
- Kamhi, A. G., Allen, M. M., & Catts, H. W. (2001). The role of the speech-language pathologist in improving decoding skills. *Seminars in Speech and Language*, 22(3), 175–184.
- Shanahan, T. (2006). *The national reading panel report: Practical advice for teachers*. Retrieved from <http://www.learningpt.org/pdfs/literacy/nationalreading.pdf>

Deep Pressure Proprioception Touch Technique

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Synonyms

Wilbarger Protocol

Deep pressure proprioceptive touch technique (DPPT): Previously known as the Wilbarger Protocol, DPPT was developed by two occupational therapists, Patricia and Julia Wilbarger, to address sensory defensiveness. This technique requires specific training and includes three parts where first a client's arms, back, and legs are brushed firmly with a soft-bristled brush similar to a surgical brush. Then joint compressions are applied at specified joints throughout the body, and finally a sensory diet is prescribed to address sensory defensiveness. This technique has been effectively used to reduce sensory defensiveness and has been linked to bringing salivary cortisol levels closer to normal values in children with sensory processing deficits. The cortisol levels have been used as a measure of stress in children, and with the use of the DPPT, the levels of cortisol approached a normal level. The recommended frequency for this technique

is every 2 h during waking hours for 2 weeks to see diminished sensory defensive behaviors. A pilot investigation using the DPPT was successful in reducing self-injurious behaviors in women with depression and a history of self-injurious behaviors. A follow-up 9 months after providing DPPT found women reported a decrease in self-harming behaviors.

References and Reading

- Kimball, J. G., Lynch, K. M., Stewart, K. C., Williams, N. E., Thomas, M. A., & Atwood, K. D. (2007). Using salivary cortisol to measure the effects of a Wilbarger protocol-based procedure on sympathetic arousal: A pilot study. *American Journal of Occupational Therapy*, 61, 406–413.
- Moore, K. M., & Henry, A. D. (2002). Treatment of adult psychiatric patients using the Wilbarger Protocol. *Occupational Therapy in Mental Health*, 18, 43–63.
- Wilbarger, P. (1984). Planning an adequate sensory diet: application of sensory processing theory during the first year of life. *Zero to Three*, 5, 7–12.
- Wilbarger, P., & Wilbarger, J. (1991). *Sensory defensiveness in children aged 2–12: An intervention guide for parents and other caretakers*. Santa Barbara: Avanti Educational Programs.
- Wilbarger, J., & Wilbarger, P. (2002). Wilbarger approach to treating sensory defensiveness and clinical application of the sensory diet. Sections in alternative and complementary programs for intervention. In A. C. Bundy, E. A. Murray, & S. Lane (Eds.), *Sensory integration: Theory and practice* (2nd ed.). Philadelphia: F.A Davis.

Deficits in Attention, Motor Control, and Perception

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Synonyms

DAMP; DAMP syndrome

Definition

DAMP syndrome is a diagnostic concept developed by Gillberg and colleagues in Sweden and used more frequently in Scandinavia. The term refers to a disorder in which aspects of attention-deficit disorder and motor coordination difficulties are present. A close link to PDD-NOS/autism spectrum disorder has been suggested (Gillberg et al. 1993; Kadesjoe and Gillberg 1999). One complexity in this regard is the potential for attentional difficulties to lead to problems with peers and social interaction; this is particularly the case if some degree of language difficulty is involved (Towbin 2005). Issues of diagnosis can also be complex in children with significant intellectual disability, attentional, and motor problems, although it has been suggested that the DAMP concept be restricted to cases where the individual has an IQ no lower than the mild-moderate range of disability. Although the topic has not been the focus of widespread research, the issue of associations between autism spectrum disorder and attentional problems is one of the great interests for both research and clinical purposes (Davis and Kollins 2012).

See Also

- ▶ [Attention Deficit/Hyperactivity Disorder](#)
- ▶ [Developmental Coordination Disorder](#)
- ▶ [Pervasive Developmental Disorder Not Otherwise Specified](#)

References and Reading

- Davis, N. O., & Kollins, S. H. (2012). Treatment for co-occurring attention deficit/hyperactivity disorder and autism spectrum disorder. *Neurotherapeutics*, 9(3), 518–530.
- Gillberg, I., Winnergard, I., & Gillberg, C. (1993). Screening methods, epidemiology and evaluation of intervention in DAMP in preschool children. *European Child & Adolescent Psychiatry*, 2(3), 121–135.
- Hellgren, L., Gillberg, I. C., Bågenholm, A., & Gillberg, C. (1994). Children with deficits in attention, motor control and perception (DAMP) almost grown up: Psychiatric and personality disorders at age 16 years.

Journal of Child Psychology and Psychiatry, and Allied Disciplines, 35(7), 1255–1271.

- Kadesjoe, B., & Gillberg, C. (1999). Developmental coordination disorder in Swedish 7-year-old children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38(7), 820–828.
- Landgren, M., Pettersson, R., Kjellman, B., & Gillberg, C. (1996). ADHD, DAMP and other neurodevelopmental/psychiatric disorders in 6-year-old children: Epidemiology and co-morbidity. *Developmental Medicine and Child Neurology*, 38(10), 891–906.
- Towbin, K. E. (2005). Pervasive developmental disorder not otherwise specified. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders*. Hoboken: Wiley.

Definition: Metaphor

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Synonyms

[Figurative language](#)

Definition

A type of figurative language in which objects or actions are compared in a way that is implied but not literal, such as “She has a heart of gold,” or where one thing is considered as representative of the other as in symbolism (Glucksberg 2001). Typically developing children usually begin to understand and produce simple metaphors as early as 2 years of age and progress to more advanced comparisons with age, showing significant improvements around age 4–5 years and continuing to develop these skills into adulthood (Stites and Ozcaliskan 2012). Children on the autism spectrum with autism demonstrate poorer comprehension of figurative language than their typically developing peers (Kalandadze et al. 2019).

Two theories are proposed to explain difficulties in interpreting figurative language in

individuals with autism. The first theory suggests that this is due to a cognitive deficit in “theory of mind,” or the ability to attribute mental states to self and to others, which leads to impairments in imagination, socialization, and communication (Happé 1995). Since metaphor requires the listener to be able to understand the speaker’s intention, an individual with autism might interpret the language literally and be unable to infer the intended meaning of the metaphor. The second theory proposes that poor figurative language comprehension is related to deficits in structural language skills, including vocabulary and syntax (Whyte et al. 2014).

Figurative language is ubiquitous in social communication, and, therefore, challenges interpreting figurative language can affect social relationships, social participation, and educational achievement (Kalandadze et al. 2016).

See Also

- Cognitive Flexibility
- Figurative Language
- Metaphoric Language

References and Reading

- Glucksberg, S. (2001). *Understanding figurative language: From metaphors to idioms*. New York: Oxford University Press.
- Happé, F. G. (1995). Understanding mind and metaphors: Insights from the study of figurative language in autism. *Metaphor and Symbol*, 10(4), 275–295.
- Kalandadze, T., Norbury, C., Naerland, T., & Naess, K.-A. B. (2016). Figurative language comprehension in individuals with autism spectrum disorder: A meta-analytic review. *Autism*, 22(2), 99–117. <https://doi.org/10.1177/1362361316668652>.
- Kalandadze, T., Bambini, V., & Naess, K.-A. B. (2019). A systematic review and meta-analysis of studies on metaphor comprehension in individuals with autism spectrum disorder: Do task properties matter? *Applied PsychoLinguistics*, 40(6), 1421–1454. <https://doi.org/10.1017/S0142716419000328>.
- Stites, L. J., & Ozcaliskan, S. (2012). Developmental changes in children’s comprehension and explanation of spatial metaphors for time. *Journal of Child Language*, 40(5), 1123–1137. <https://doi.org/10.1017/S0305000912000384>.
- Whyte, E. M., Nelson, K. E., & Scherf, K. S. (2014). Idiom, syntax, and advanced theory of mind abilities in children with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, 57(1), 120–130. <https://doi.org/10.1044/1092-4388>.

DeGangi-Berk

- DeGangi-Berk Test of Sensory Integration

DeGangi-Berk Test of Sensory Integration

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Synonyms

DeGangi-Berk; TSI

Description

This assessment tool offers an objective method to examine the sensory functioning of children aged 3–5 years. This 36-item assessment published in 1983 intended to provide an objective method to determine whether, and to what extent, a preschool child had sensory processing deficits so that the practitioner did not need to rely on clinical judgment alone. At that time, there were only measurements of motor functioning with no other instrument sufficiently sensitive to determine if these motor issues were caused by an underlying sensory integrative difficulty.

Once the items are scored, they are calculated to establish an overall score of sensory integrative functioning (total test score), as well as a score within each of the following subdomains of sensory integration:

DeGangi-Berk Test of Sensory Integration, Table 1 TSI subdomains of sensory integration. All information taken from TSI test manual (p. 1–2)

Sensory integrative subdomain	Names of individual tests	Description and significance
Postural control	• Monkey task • Side-Sit • cocontraction • Prone on elbows • Wheelbarrow walk • Airplane • Scooter board • cocontraction	• Stabilization of the neck, trunk, and upper extremities • Muscle cocontraction of the neck and upper extremities • Includes antigravity postures
Bilateral motor coordination	• Rolling pin activity • Jump and turn • Diadochokinesis • Drumming • Upper extremity control	• Emphasizes bilateral motor coordination • Includes components of laterality including trunk rotation, rapid unilateral and bilateral hand movements, and crossing the midline • Includes stability of the upper and lower extremities in bilateral symmetrical postures and disassociation of trunk and arm movements
Reflex integration	• ATNR – asymmetrical tonic neck • STNR – symmetrical tonic neck • Diadochokinesis	• Quadruped position to observe asymmetrical and symmetrical tonic neck reflexes and associated reactions of the upper extremities

1. Postural control
2. Bilateral motor integration
3. Reflex integration

The above subdomains were identified for inclusion “because of their clinical significance in the development of sensory integrative functions in the preschool child” (DeGangi and Berk 1983, p. 1). Table 1 outlines the components of each subdomain.

This tool was designed to be implemented by occupational or physical therapy practitioners given their training and educational background in the interpretation of sensory integrative information and test results. Therefore, it is suggested that a practitioner outside of these fields (i.e., special educators or motor development specialists) seek the assistance of an occupational or physical therapist for the interpretation of the test scores.

With a baseline understanding of sensory processing, implementers should allow 2 h to learn the items prior to implementation. The assessment

manual is easy to follow, and the specific instructions for item implementation are outlined with pictures to assist. A score of 0 through 1, 2, 3, or 4 is received depending on the child’s response to each item and the quality of the performance indicating that the skill has been developed. The higher the score indicates a more integrated, organized, or normal response. Lower scores qualify the child’s responses, for example, unable to hold, loses grasp, does not cross [midline], no resistance, slight to moderate flexion of the elbow, etc.

The score tallies in each subdomain then result in a “normal,” “at risk,” or “deficient” score profile for a total test score, postural control score, bilateral motor integration score, and a reflex integration score (which is only counted toward the total test score). Score ranges for the varying age ranges are provided on the score sheet which makes scoring very clear. This criterion-referenced assessment tool offers clinicians working with this population of children a structured and organized method to assess sensory integrative functions in children with delays in sensory,

motor, and perceptual skills, or children suspected of having learning problems.

Historical Background

Georgia DeGangi, PhD, OTR (occupational therapist who now practices in clinical psychology) and Ronald Berk, PhD (professor of educational research at Johns Hopkins University at the time and authored the 1980 book titled: *Criterion Referenced Measurements: State of the Art*) developed this objective tool to observe and measure the sensory integrative processes in preschool children, specifically the vestibularly based functions of postural control, bilateral integration, and reflex integration. It was thought that difficulties in sensory integrative processing in preschool children could result in fine or gross motor delays, poor balance, poor hand use, distractibility, and/or visual-spatial organization later in the school years. Rather than wait for these secondary issues to arise, it was thought that intervention could addressed/remediated the sensory concerns before secondary issues arose. This thought, based on sensory integration theory (Ayres 1964, 1972, 1979), continues today. A fuller description of Ayres’ theory of sensory integration can be

found in the links below titled Ayres, A. Jean, sensory processing, and sensory integration therapy.

Psychometric Data

DeGangi began developing test items in 1978, completed psychometric studies, revised the test and the items, completed several rounds of reliability and validity testing, and ultimately identified 73 items. After additional item analysis, which discarded items that did not well discriminate typical from delayed children or were not sufficiently sensitive to typical developmental in these age ranges, only 36 items remained.

The test manual specifically outlines each step of the sampling and statistical procedures. However, it should be noted that there were some sampling difficulties resulting in a disproportionate number of 3–4-year-old children and a low sample population (*n*). The authors therefore suggest that further research with a more representative sample would improve the utility of the tool and the generalizability of the findings.

Table 2 outlines the components of the assessment process including the psychometric procedures associated with the development of this

DeGangi-Berk Test of Sensory Integration, Table 2 DeGangi-Berk TSI assessment review form

Test name: DeGangi-Berk Test of Sensory Integration (TSI)
Author(s): Georgia A. DeGangi PhD, OTR and Ronald Berk, PhD
Publisher: Western Psychological Services, 625 Alaska Ave, Torrance, CA 90503; 800-648-8857
Technical information: 2 h of practice before administering
Age range(s): 3–5 years old
Assessment type: Criterion referenced
The following information was obtained from the TSI Manual
Reliability:
1. <i>Interobserver reliability:</i> Two pairs of examiners were used. Difficulties with implementing repeat testing procedures resulted in a low number in the sample and not fully representative of each age group (i.e., no 5-year olds). Intraclass correlations were .80 and above for postural control, bilateral motor integration, and the total test; and coefficients for the dependability of each observer ranged from .67 to .79 for those same categories. Reflex integration was low within each pair of examiners as well as inconsistent between two pairs of raters.
2. <i>Decision-consistency reliability:</i> The <i>p_o</i> index of decision consistency was used determine “the proportion of children classified as normal and delayed on repeated testings” indicating a degree of confidence for the decision (i.e., the stability of the decisions). A sample of 23 “normal” and 6 “delayed” 3–5-year-old children (10 boys and 19 girls) were tested twice during a 1-week retest interval. Utilizing three observers, the <i>p_o</i> estimates for the three subtests and the total test ranged from 79–93% with the lowest in reflex integration. However, standard error was large and thought to be the result of the small sample size.

(continued)

DeGangi-Berk Test of Sensory Integration, Table 2 (continued)

3. *Test-retest reliability*: The Pearson correlation coefficients between test and retest scores for each subtest and the total score for a period of 1 week ranged from .85 to .96. Postural control was the least stable with anticipation/familiarity with the task thought to be an influence in the second testing, whereas bilateral motor integration and reflex integration, requiring more automatic responses, were thought to be less susceptible to performance changes on test-retest.

Validity:

1. *Content validity*: A two-stage judgmental review occurred to determine test validity.

- *Item-behavior congruence*: Item-behavior congruence and representativeness was rated by eight judges (occupational therapists). The degree of congruence between the items and the subdomain was rated either as poor, moderate, or high for each item. A rating of “high” was obtained for all items in postural control and reflex integration, and for all but one judge for bilateral motor integration.

- *Representativeness*: Twelve judges were asked: Is each collection of items representative of its respective subdomain of behaviors? A score of “high” was obtained by all judges for postural control and 87% of the judges for the bilateral motor integration and reflex integration (with the other 13% scoring as “moderate”).

2. *Construct validity*: Construct validity evidence was found within the item, subtest, and test, and because the specific use of the test score was to identify normal vs. delayed, this was the primary focus of the analysis. Total of 139 children in the sample.

- *Item validity*: The effectiveness of each item was found by computing a discrimination index (DIS) displaying the difference between the mean score for each item in the normal and delayed groups. Statistical significance was then computed using a *t* test and the magnitude of the significance computed via effect size (*d*). Out of the original 73 test items, 37 were taken out after item analysis since they did not discriminate between the groups of delayed and normal, or were not sensitive to the normal developmental status of this population.

- *Decision validity*: The cut-off scores for this tool, and therefore the focus of these analyses, were to minimize the false normal error rate as this was thought to be the most serious of errors. The total test and the three subtests’ error rate ranged from 4 to 9%. The error rate for false delayed ranged from 10 to 26% for all test scores. Sensitivity and specificity were calculated with scores of 71% and 85%, respectively, for the total test.

- *Test structure*: Moderately low subtest correlations (.39–.65) confirms that each subtest is measuring different vestibularly based functions, thus supporting the structure of the test. The correlation of the subtests to sensory integration as a whole ranges from .64 to .93. There was also support that the subdomains of postural control and bilateral motor integration were more vital to overall sensory integration than reflex integration.

Testing procedures

Obtaining information: Thirty-six items should be administered individually and in one sitting; items should be administered exactly as described in the order presented in the manual.

Time to administer: 30 min

Time to score: 10 min

Materials included in the test kit? ☒ yes ☐ no

Additional materials needed: 10 × 15 ft. space, table and chair, masking tape, pencil without eraser, switch-back stopwatch, 3-ft-long wooden dowel, rolling pin, carpeted scooter board, plastic hula hoop, and floor mat

Is the tool easy to learn and administer? ☒ yes ☐ no

How much training or practice is required? 2 h

Who can administer the test? Designed for implementation by occupational therapists and physical therapists; can be implemented by special educators or motor development specialists but seeking an occupational or physical therapist to interpret whether the score is recommended as they have training and education in sensory processing.

Is the manual easy to follow/understand? ☒ yes ☐ no

Are the forms easy to follow/understand? ☒ yes ☐ no

The forms are very clear, easy to follow while administering, and can be quickly scored in a very objective manner.

Domains: Postural control, bilateral integration, and reflex integration

criterion-referenced assessment tool. In summary, the total test score can be used reliably and validly for screening decisions, and the postural control score and bilateral motor integration score can be used reliably and validly for diagnostic decisions based on the following information:

1. **Domain validity:** The total test had a high degree of domain validity

- Consensus among therapists that the items measure the behaviors they were designed to measure, and that the collection of items composing each subtest was representative

of the behaviors defined by the subdomains. (DeGangi and Berk 1983, p. 40)

2. Construct validity:
 - Total test score can be used for screening decisions with better than 80% accuracy and a 9% false normal error rate.
 - Postural control and bilateral motor integration subtests were extremely accurate.
 - Reflex integration was the least effective subtest.
3. Interobserver reliability:
 - Very reliable for postural control, bilateral integration, and total sensory integration behaviors.
 - Considerable subjectivity for reflex integration behaviors.
4. There were high levels of classification consistency in the identification of the classification designated for each item.
5. Test-retest reliability:
 - The results provided substantial evidence of the stability of sensory integrative functions for a 1-week re-test interval using a homogeneous preschool sample. (DeGangi and Berk 1983, p. 41).

Clinical Uses

Any assessment tool should be used in combination with other tools in order to gain the most comprehensive picture of a child's functioning. The DeGangi-Berk TSI was intended to provide information related to the three subdomains noted above as these categories of sensory integrative functioning were thought to have a strong impact on the development of sensory integrative functions in the preschool child. The intent was to administer this assessment to children with delays in sensory, motor, and perceptual skills, or to children suspected of having learning problems.

This tool continues to be utilized today in clinical practice as it is a structured and organized method to investigate the sensory processing abilities in this age group.

See Also

- ▶ Ayres, A. Jean
- ▶ Evaluation of Sensory Processing
- ▶ Occupational Therapy (OT)
- ▶ Sensory Diet
- ▶ Sensory Integration (SI) Therapy
- ▶ Sensory Integration and Praxis Test
- ▶ Sensory Processing
- ▶ Sensory Processing Assessment
- ▶ Sensory Processing Measure
- ▶ Sensory Processing Measure: Preschool (SPM-P)
- ▶ Test of Sensory Functioning in Infants

References and Reading

- Ayres, A. J. (1964). Tactile functions: Their relations to hyperactive and perceptual-motor behaviour. *American Journal of Occupational Therapy*, 18, 6–11.
- Ayres, A. J. (1972). *Sensory integration and learning disorders*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (1979). *Sensory integration and the child*. Los Angeles: Western Psychological Services.
- DeGangi, G. A., & Berk, R. (1983). *DeGangi-Berk Test of Sensory Integration (TSI) manual*. Los Angeles: Western Psychological Services.

Deictic Terms

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Synonyms

Deixis

Definition

Deictic terms are words whose meaning shifts depending on the point of view of the speaker. Examples of deictic terms include “this/that,” “here/there,” “I/you,” and “my/your.” While some personal pronoun contrasts (“I/you,” “my/

your”) are expected to develop before 3 years of age, many typically developing children continue to have difficulty with spatial contrast deictic terms (“this/that,” “here/there”) into the early school age years. This difficulty is thought to be related to the shifting quality of the referents for these terms. That is, “I” does not refer to any particular person but to the person who happens to be talking at a given time. When that person stops talking, the referent for “I” shifts to the next speaker. “Here” refers not to a specific location but rather to a place near the speaker. What is “here” for the speaker may be “there” for the listener. This shifting reference is thought to cause special difficulty for speakers with ASD, possibly due to their difficulties with perspective taking, communicative engagement, flexibility, and change. It is important to note that young children with typical development can also have difficulty with deictic terms.

See Also

- [Pronoun Errors](#)
- [Pronoun Reversal](#)
- [Pronoun Use](#)

References and Reading

- Bartolucci, G., & Albers, R. J. (1974). Deictic categories in the language of autistic children. *Journal of Autism and Developmental Disorders*, 4(2), 131–141.
- Belisle, J., Dixon, M. R., Stanley, C. R., Munoz, B., & Daar, J. H. (2016). Teaching foundational perspective-taking skills to children with autism using the PEAK-T curriculum: Single-reversal “I–You” deictic frames. *Journal of applied behavior analysis*, 49(4), 965–969.
- Gilroy, S. P., Lorah, E. R., Dodge, J., & Fiorello, C. (2015). Establishing deictic repertoires in autism. *Research in Autism Spectrum Disorders*, 19, 82–92.
- Hobson, P. R. (2009). Personal pronouns and communicative engagement in autism. *Journal of Autism and Developmental Disorders*, 40(6), 653–664. <https://doi.org/10.1007/s10803-009-0910-5>.
- Hobson, P. R., Garcia-Perez, R. M., & Lee, A. (2009). Person-centered (deictic) expressions and autism. *Journal of Autism and Developmental Disorders*, 40(4), 403–415. <https://doi.org/10.1007/s10803-009-0882-5>.
- Jackson, M. L., Mendoza, D. R., & Adams, A. N. (2014). Teaching a deictic relational repertoire to children with autism. *The Psychological Record*, 64(4), 791–802.
- Lee, A., Hobson, R. P., & Chiat, S. (1994). I, you, me and autism: An experimental study. *Journal of Autism and Developmental Disorders*, 24, 155–176.
- Owens, R. E. (2008). *Language development: An introduction* (7th ed.). Boston: Allyn & Bacon.

Deixis

- [Deictic Terms](#)

DEL22q13.3 (Entrez Gene, OMIM, Uniprot)

- [SHANK 3](#)

Delaware Autism Program

Vincent Winterling

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Major Areas or Mission Statement

The Delaware Autism Program (DAP) is one of the largest public school programs in the United States specializing in educating children and adolescents with an Autism Spectrum Disorder (ASD). In 2019, it served in excess of 1500 students between 2 and 21 years of age, in the full range of settings (residential programs, separate schools and settings, and integrated school and community sites) in six affiliated school districts. DAP sites employ more than 500 staff, including teachers, assistants, specialists (psychologists, behavior analysts, speech language pathologists, occupational therapists, nurses, etc.), and administrative and support staff.

Retired (former Statewide Director of Autism).

Landmark Contributions

Elements of DAP have been described in various book chapters (Battaglini and Bondy 2006; Bondy 1996; Bondy and Frost 1994, 1995; Doehring and Winterling 2011). The Picture Exchange Communication System (PECS) (Frost and Bondy 2000) was first developed by Andy Bondy and Lori Frost during their tenure at DAP, together with the involvement of other DAP staff. Statewide directors have included Dr. Andy Bondy (1981–1997), Dr. Peter Doehring (1999–2008), Dr. Vincent Winterling (2009–2019), Dr. Mary Whitfield (2019–present).

Major Activities

DAP is public school program that presently consists of affiliated programs in 6 of the 19 school districts (Local Education Agencies, or LEA) across the 3 counties in the State of Delaware, plus other specialized services and supports (e.g., full year programming, respite, and temporary residential), provided through the Office of the Statewide Director of Autism. As a public school program, DAP's services are fully funded by the LEA and the State Education Agency (SEA), at no cost to parents. The six affiliated programs share many key elements, including: (a) programs for children 2 up until 21 years of age, across the autism spectrum; (b) settings ranging from full inclusion to separate classroom for children with ASD, including extended school year services; (c) reliance on teaching methods based on principles of Applied Behavior Analysis (ABA), including PECS; (d) a high staff to student ratio to support more individualized teaching and community integration; (e) opportunities for parents to create local Parent Advisory Committees (PAC) to provide input to the LEA, SEA, and Office of the Statewide Director of Autism; (f) expectations that staff complete a core training program, which for teachers includes a 15-credit graduate teaching certificate in autism and (g) oversight of the program provided by external experts in collaboration with program administration. Many of the programs also coordinate with

other organizations (i.e., daycares, vocational settings, institutes of higher education) to provide community-based services. Three of the six programs operate larger county centers which provide education services to students with more challenging educational and behavioral needs. Through an agreement with the SEA, the Office of the Statewide Director of Autism provides services across the state, including: (a) management of extended educational services (temporary residential programming in community-based settings) and extended support services (in-home respite provided to parents for a nominal co-pay); (b) leadership of various statewide committees that provide consultation to LEAs regarding educational programming, to coordinate parent input from the PACs and provide independent peer review of the assessment and intervention for students with very challenging behaviors; (c) coordination of staff training specific to ASD, and; (d) consultation to all school districts in the state on challenging students. DAP was established in 1976 after parents helped to pass laws defining many of the core elements of the program (specialized positions like the Statewide Director, specialized services like extended educational and support services, additional staffing, extended school year, statewide committees, etc.).

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Educational Interventions](#)
- [Free Appropriate Public Education](#)
- [Individual Education Plan](#)
- [Local Educational Authority](#)
- [Picture Exchange Communication System](#)
- [Regional Centers](#)
- [Statewide Service Programs](#)

References and Reading

- Battaglini, K., & Bondy, A. (2006). Application of the pyramid approach to education model in a public school setting. In J. S. Handleman & S. L. Harris (Eds.), *School-age education programs for children with autism* (pp. 163–194). Austin: Pro-Ed.

- Bondy, A. (1996). What parents can expect from public school programs. In C. Maurice, G. Green, et al. (Eds.), *Behavioral intervention for young children with autism: A manual for parents and professionals* (pp. 323–330). Austin: Pro-Ed.
- Bondy, A., & Frost, L. A. (1994). The Delaware autistic program. In S. L. Harris & J. S. Handleman (Eds.), *Preschool education programs for children with autism* (pp. 37–54). Austin: Pro-Ed.
- Bondy, A. S., & Frost, L. A. (1995). Educational approaches in preschool: Behavior techniques in a public school setting. In E. Schopler, G. Mesibov, et al. (Eds.), *Learning and cognition in autism* (pp. 311–333). New York: Plenum Press.
- Doehring, P., & Winterling, V. (2011). The implementation of evidence-based practices in public schools. In B. Reichow, P. Doehring, D. V. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-based practices and treatments for children with autism* (pp. 343–363). New York: Springer Science + Business Media.
- Frost, L. A., & Bondy, A. S. (2000). *The picture exchange communication system training manual*. Cherry Hill: PECs.

Delay, Deviance Versus

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Definition

Delay versus deviance refers to a debate about the nature of development in autism and other disorders. In general, a child who exhibits a developmental delay follows a progression of development found in the general population, but progress in development at a slower rate. In contrast, a child who exhibits deviance follows a progression of development that is different both in rate and sequence of progression. There is evidence to suggest that children with autism may follow a developmental progression that includes elements of both delay and deviance. In many children with autism, language development is often delayed but occurs in a progression similar to children with typical development. In other children, language development may also include deviant characteristics (e.g., echolalia).

Many children with autism demonstrate deviance in the development of social and pragmatic skills. For example, some children with autism demonstrate deviance in the development of social behaviors such as joint attention.

See Also

► [Speech Delay](#)

References and Reading

- Baron-Cohen, S. (1988). Social and pragmatic deficits in autism: Cognitive or affective? *Journal of Autism and Developmental Disorders*, 18, 379–402.
- Carlisle, P. (2007). *Progress in autism research*. New York: Nova Science.
- Charman, T., & Stone, W. (2006). *Social and communication development in autism spectrum disorders: Early identification, diagnosis, and intervention*. New York: The Guilford Press.
- Tager-Flusberg, H. (1981). On the nature of linguistic functioning in early infantile autism. *Journal of Autism and Developmental Disorders*, 11, 45–56.
- VanMeter, L., Fein, D., Morris, R., Waterhouse, L., & Allen, D. (1997). Delay versus deviance in autistic social behavior. *Journal of Autism and Developmental Disorders*, 27, 557–569.

Delayed Echolalia

- [Echolalia](#)
► [Movie Talk](#)

Delusion of Doubles

- [Capgras Syndrome](#)

Delusion of Duplicates

- [Capgras Syndrome](#)

Delusion of Negative Doubles

- [Capgras Syndrome](#)

Delusion of Substitution

- [Capgras Syndrome](#)

Delusional Hypoidentification

- [Capgras Syndrome](#)

DELV-NR

- [Diagnostic Evaluation of Language Variation \(DELV-Norm Referenced\)](#)

Demand

- [Mands](#)

Demographics and the Age of Autism Diagnosis

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Definition

The role of social and demographic factors on the age at which children are diagnosed with ASD.

Historical Background

A growing body of literature has shed light on the nature of the rapidly rising prevalence of ASD.

Several variables have been suggested in attempts to explain this trend, with the primary question focusing on whether there is an actual rise in incidence or if an increase in awareness has led to higher rates of diagnosis. Variables such as parental age and environmental pollutants have been suggested as influencers of diagnostic trends (Durkin et al. 2008; Raz et al. 2015a). From the social perspective, contributing factors appear to include increased awareness of ASD, changes in the tools used in screening and diagnosis, lower levels of stigma in societies, and shifts in the diagnostic criteria (Leonard et al. 2010). Taken together, the general consensus appears to be that, while the former factors may play a modest role, the rise in incidence is not a true increase in the rates of ASD but rather a social shift expressed in higher numbers of individuals being accurately identified and diagnosed (Isaksen et al. 2013).

Given the above, it follows that these social factors affecting the rates of diagnosis of ASD play out in variable fashion across social and demographic contexts. In the ongoing work by the Centers for Disease Control and Prevention's (CDC) Early Autism and Developmental Disabilities Monitoring (ADDM) Network, national prevalence estimates are based on the average across the states monitored. Within that data exists tremendous variability, strengthening the claim that social factors are central in determining rates of diagnosis (Christensen et al. 2019). In a recent publication evaluating data from 2014, the state with the lowest prevalence was Missouri, at approximately 1 in 104 children. The highest prevalence was found in New Jersey, at 1 in 35. Given the lack of any environmental or biological explanations for this stark discrepancy, it is most logically by the fact that New Jersey has more capacity in the form of trained experts with the ability to diagnose young children as well as an early childhood education system capable of screening for and identify the children.

Notably, even with increased awareness, more accurate diagnostic measures, and reduced stigma, families and children that are not seen by trained professionals with specific expertise in the diagnosis of ASD will not be identified.

Current Knowledge

Concurrent to the aforementioned increase in rates of diagnosis of ASD, the professional community's ability to identify and diagnose ASD in early childhood has improved dramatically. Much research now shows that an evidence-based diagnosis of ASD can be provided to most young children with a high degree of confidence between the ages 18 and 24 months and that such diagnoses are typically stable, meaning they remain accurate when the same child is reevaluated at older ages (Guthrie et al. 2013; Ozonoff et al. 2015). Despite these significant advances in our understanding of the early presentation of ASD and our ability to make accurate early diagnoses, the median age at which a child in the United States receives a diagnosis of ASD is 52 months (Baio et al. 2018).

A number of factors appear to contribute to this significant and costly delay in the age at which children are diagnosed. Children with less pronounced autism symptomatology are often diagnosed later compared to children with higher levels of symptom severity, as are children with average verbal abilities, in contrast to those who are minimally verbal (Salomone et al. 2016). The same is true for children from families with low socioeconomic status (SES) and those whose parents do not express concern regarding the presentation of primary symptoms. On a systemic level, children who do not have access to education and health services are often diagnosed later than peers who have such access (Daniels and Mandell 2014; Mazurek et al. 2014; Shattuck et al. 2009).

Children with comorbid health problems or behavioral difficulties are also likely to receive a diagnosis later, since parents or professionals could potentially attribute symptoms of ASD to these co-occurring conditions (Mandell et al. 2005). Such a situation often leads to an incorrect primary diagnosis of another disorder or a missed diagnosis altogether due to diagnostic overshadowing (Fombonne 2005; Goldstein and Schwebach 2004). For similar reasons, children with autism who have experienced adverse childhood experiences, defined as stressful or traumatic experiences, are likely to receive a diagnosis of ASD later than peers who do not have these experiences (Berg et al. 2018).

Specifically related to SES, studies have consistently found a link between high SES, represented by income, parents' profession, and/or years of parental education, and increased rates of ASD (Durkin et al. 2017; Ng et al. 2017). The primary explanation proposed is that with higher parental education and financial means comes increased access to services, including ASD diagnostic services (Durkin et al. 2010, 2017; Thomas et al. 2012). Work in the United States has found children with ASD from families that speak English as a second language receiving school-based services later than children who come from English-speaking families. When they do eventually receive the services, they receive fewer hours each week than their peers (Nguyen et al. 2016). In contrast, some studies from outside the United States found no particular association between SES and age of diagnosis, and some even found an inverse relationship between family income or parental education and ASD incidence (Khaiman et al. 2015; Larsson et al. 2005). Interestingly, in countries with universal healthcare, such as Canada, Norway, Sweden, and France, where disparities in access to health services between socioeconomic classes are low, higher ASD prevalence was found in lower SES groups, or no link was found between SES and prevalence of ASD (Burstyn et al. 2010; Delobel-Ayoub et al. 2015; Rai et al. 2012).

An extensive review conducted by Daniels and Mandell (2014) examined the association between SES, ethnic or race affiliation, and age of diagnosis. Of the 11 studies that inspected the relationship between SES and age of diagnosis, 5 found an association between high SES and earlier diagnosis, while the remaining studies found no such association. Twelve studies examined the association between ethnic or racial affiliation and the age of diagnosis and revealed contradictory findings. Five of the 12 found that children belonging to ethnic or racial minorities were likely to receive a later diagnosis compared to children of nonminority children, while 5 other studies did not find a correlation between these factors. The remaining two studies found mixed results, showing that children of the main ethnic group were likely to receive a later diagnosis

compared to children of ethnic or race minorities. The researchers posit that these dyssynchronous findings can be attributed to variables that differ between the reviewed studies, such as study duration, sampling method, and the geographic location of the study. The variable findings in this body of literature demand a more nuanced assessment of each individual study, which must each be interpreted within its particular respective context. For instance, a Norwegian study found that ethnicity did not influence the age of diagnosis (Larsen 2015). However, it is imperative to contextualize this finding within the political reality of Norway, where every child has access to health and developmental care at community healthcare centers, regardless of any ethnic difference.

As indicated above, societal shifts in awareness and understanding of ASD are clinically significant and relevant in societal contexts where children and families are able to access proper care. A consistent source of medical or developmental care is essential in order for children to be diagnosed in timely fashion (Emerson et al. 2016). Families with lower SES are also less likely to have such a consistent source of care, thereby minimizing the chance that developmental delays of any kind will be identified and targeted close within the optimal timeframe.

It is noteworthy that the association between consistent care and age of diagnosis was found to differ based on race (Emerson et al. 2016), with African American children's age of diagnosis not decreasing in correlation with consistent care. This could potentially be explained by way of differences in parent and/or professional behavior. Professionals seeing young children of low SES African Americans may be slower to refer the child for a full diagnostic evaluation. Alternatively, or simultaneously, the parents of these children maybe slower to push for such a referral or to question a "wait-and-see" approach.

screening and diagnosis are critical. The conflicting findings that exist in this body of literature point to fact that we do not have a clear understanding of these issues. While some studies find strong associations between variables such as SES and age of diagnosis, others do not. Without considering the context for these studies, we are unable to decipher the meaning of the body of work as a whole.

For instance, while studies have examined differences between ethnic and racial groups within the United States, few have done so internationally. A recent study from Israel (Koller et al. 2019) found that the Arab children diagnosed in Jerusalem prior to the age of 6 were nearly all minimally verbal and displayed severe autism symptomatology. While small, this study highlights the fact that while large-scale, population-based studies are necessary to understand broader trends, more nuanced examinations of specific populations and regions are necessary in order to gain actionable data that can lead to the support of specific sectors.

Significantly, the work done in this area has clear and direct relevance for public policy and community mental health. Efforts to raise awareness of ASD and improve capacity for identification of ASD by community professionals can assist in the early diagnosis of children. The heterogeneity of results seen across studies indicates that region-specific differences in screening, evaluation, and diagnostic practice affect the age of diagnosis. The findings of studies conducted in specific geographical regions or municipalities should be conveyed to policymakers and stakeholders in order to utilize the findings as stepping-stones to improved systems of public awareness, screening, and diagnosis. Continued improvements in these both research and research-driven practice may assist the provision of earlier ASD diagnoses and support, which could lead to improved developmental outcomes.

Future Directions

Further research and taking a more nuanced and subjective perspective on the process of

See Also

- [Epidemiology](#)
- [Prevalence](#)

References and Reading

- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., . . . White, T. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summaries*, 67(6), 1.
- Berg, K. L., Acharya, K., Shiu, C. S., & Msall, M. E. (2018). Delayed diagnosis and treatment among children with autism who experience adversity. *Journal of Autism and Developmental Disorders*, 48(1), 45–54.
- Bhasin, T. K., & Schendel, D. (2007). Sociodemographic risk factors for autism in a US metropolitan area. *Journal of Autism and Developmental Disorders*, 37(4), 667–677.
- Burstyn, I., Sithole, F., & Zwaigenbaum, L. (2010). Autism spectrum disorders, maternal characteristics and obstetric complications among singletons born in Alberta, Canada. *Chronic Diseases in Canada*, 30(4), 125–134.
- Christensen, D. L., Maenner, M. J., Bilder, D., Constantino, J. N., Daniels, J., Durkin, M. S., . . . Shenouda, J. (2019). Prevalence and characteristics of autism spectrum disorder among children aged 4 years – Early autism and developmental disabilities monitoring network, seven sites, United States, 2010, 2012, and 2014. *MMWR Surveillance Summaries*, 68(2), 1.
- Cohen, P., & Hesselbart, C. (1993). Demographic factors in the use of children's mental health services. *American Journal of Public Health*, 83, 49–52.
- Daniels, A. M., & Mandell, D. S. (2014). Explaining differences in age at autism spectrum disorder diagnosis: A critical review. *Autism*, 18(5), 583–597.
- Delobel-Ayoub, M., Ehlinger, V., Klapouszczak, D., Maffre, T., Raynaud, J. P., Delpierre, C., & Arnaud, C. (2015). Socioeconomic disparities and prevalence of autism spectrum disorders and intellectual disability. *PLoS One*, 10(11), e0141964.
- Donohue, M. R., Childs, A. W., Richards, M., & Robins, D. L. (2017). Race influences parent report of concerns about symptoms of autism spectrum disorder. *Autism*, 23, 1–12.
- Durkin, M. S., Maenner, M. J., Newschaffer, C. J., Lee, L. C., Cuniff, C. M., Daniels, J. L., . . . Schieve, L. A. (2008). Advanced parental age and the risk of autism spectrum disorder. *American Journal of Epidemiology*, 168(11), 1268–1276. <https://doi.org/10.1093/aje/kwn250>.
- Durkin, M. S., Maenner, M. J., Meaney, F. J., Levy, S. E., DiGuiseppi, C., Nicholas, J. S., . . . Schieve, L. A. (2010). Socioeconomic inequality in the prevalence of autism spectrum disorder: Evidence from a US cross-sectional study. *PLoS One*, 5(7), e11551.
- Durkin, M. S., Maenner, M. J., Baio, J., Christensen, D., Daniels, J., Fitzgerald, R., . . . Wingate, M. S. (2017). Autism spectrum disorder among US children (2002–2010): Socioeconomic, racial, and ethnic disparities. *American Journal of Public Health*, 107(11), 1818–1826.
- Emerson, N. D., Morrell, H. E., & Neece, C. (2016). Predictors of age of diagnosis for children with autism spectrum disorder: The role of a consistent source of medical care, race, and condition severity. *Journal of Autism and Developmental Disorders*, 46(1), 127–138.
- Fombonne, E. (2005). Epidemiology of autistic disorder and other pervasive developmental disorders. *Journal of Clinical Psychiatry*, 66(10), 3–8.
- Goldstein, S., & Schwabach, A. J. (2004). The comorbidity of pervasive developmental disorder and attention deficit hyperactivity disorder: Results of a retrospective chart review. *Journal of Autism and Developmental Disorders*, 34(3), 329–339.
- Guthrie, W., Swineford, L. B., Nottke, C., & Wetherby, A. M. (2013). Early diagnosis of autism spectrum disorder: Stability and change in clinical diagnosis and symptom presentation. *Journal of Child Psychology and Psychiatry*, 54(5), 582–590.
- Hall, H. R., & Graff, J. C. (2012). Maladaptive behaviors of children with autism: Parent support, stress, and coping. *Issues in Comprehensive Pediatric Nursing*, 35(3–4), 194–214.
- Isaksen, J., Diseth, T. H., Schjølberg, S., & Skjeldal, O. H. (2013). Autism spectrum disorders – are they really epidemic? *European Journal of Paediatric Neurology*, 17(4), 327–333. <https://doi.org/10.1016/j.ejpn.2013.03.003>.
- Khaiman, C., Onnuam, K., Photchanakaew, S., Chonchaiya, W., & Suphateetiporn, K. (2015). Risk factors for autism spectrum disorder in the Thai population. *European Journal of Pediatrics*, 174(10), 1365–1372. <https://doi.org/10.1007/s00431-015-2544-2>.
- Koller, J., Shalev, R., Schallamach, C., Gumpel, T. P., & Begin, M. (2019). The role of demographics in the age of autism diagnosis in Jerusalem. *Journal of Autism and Developmental Disorders*, 1–9.
- Larsen, K. (2015). The early diagnosis of preschool children with autism spectrum disorder in Norway: A study of diagnostic age and its associated factors. *Scandinavian Journal of Child and Adolescent Psychiatry and Psychology*, 3(2), 136–145.
- Larsson, H. J., Eaton, W. W., Madsen, K. M., Vestergaard, M., Olesen, A. V., Agerbo, E., . . . Mortensen, P. B. (2005). Risk factors for autism: Perinatal factors, parental psychiatric history, and socioeconomic status. *American Journal of Epidemiology*, 161(10), 916–925; discussion 926–918. <https://doi.org/10.1093/aje/kwi123>.
- Leonard, H., Dixon, G., Whitehouse, A. J. O., Bourke, J., Aiberti, K., Nassar, N., . . . Glasson, E. J. (2010). Unpacking the complex nature of the autism epidemic. *Research in Autism Spectrum Disorders*, 4(4), 548–554. <https://doi.org/10.1016/j.rasd.2010.01.003>.
- Leonard, H., Glasson, E., Nassar, N., Whitehouse, A., Bebbington, A., Bourke, J., . . . Bower, C. (2011). Autism and intellectual disability are differentially related to sociodemographic background at birth. *PLoS One*, 6(3), e17875.
- Mandell, D. S., Listerud, J., Levy, S. E., & Pinto-Martin, J. A. (2002). Race differences in the age at diagnosis among Medicaid-eligible children with autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 41(12), 1447–1453.

- Mandell, D. S., Novak, M. M., & Zubritsky, C. D. (2005). Factors associated with age of diagnosis among children with autism spectrum disorders. *Pediatrics*, 116(6), 1480–1486.
- Mazurek, M. O., Handen, B. L., Wodka, E. L., Nowinski, L., Butter, E., & Engelhardt, C. R. (2014). Age at first autism spectrum disorder diagnosis: The role of birth cohort, demographic factors, and clinical features. *Journal of Developmental & Behavioral Pediatrics*, 35(9), 561–569.
- Ng, M., de Montigny, J. G., Ofner, M., & Do, M. T. (2017). Environmental factors associated with autism spectrum disorder: A scoping review for the years 2003–2013. *Health Promotion and Chronic Disease Prevention in Canada*, 37(1), 1–23.
- Nguyen, C. T., Krakowiak, P., Hansen, R., Hertz-Picciotto, I., & Angkustsiri, K. (2016). Sociodemographic disparities in intervention service utilization in families of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(12), 3729–3738.
- Ozonoff, S., Young, G. S., Landa, R. J., Brian, J., Bryson, S., Charman, T., . . . Stone, W. L. (2015). Diagnostic stability in young children at risk for autism spectrum disorder: A baby siblings research consortium study. *Journal of Child Psychology and Psychiatry*, 56(9), 988–998.
- Rai, D., Lewis, G., Lundberg, M., Araya, R., Svensson, A., Dalman, C., . . . Magnusson, C. (2012). Parental socioeconomic status and risk of offspring autism spectrum disorders in a Swedish population-based study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(5), 467–476.
- Raz, R., Roberts, A. L., Lyall, K., Hart, J. E., Just, A. C., Laden, F., & Weisskopf, M. G. (2015a). Autism spectrum disorder and particulate matter air pollution before, during, and after pregnancy: A nested case-control analysis within the Nurses' Health Study II cohort. *Environmental Health Perspectives*, 123(3), 264–270. <https://doi.org/10.1289/ehp.1408133>.
- Raz, R., Weisskopf, M. G., Davidovitch, M., Pinto, O., & Levine, H. (2015b). Differences in autism spectrum disorders incidence by sub-populations in Israel 1992–2009: A total population study. *Journal of Autism and Developmental Disorders*, 45(4), 1062–1069.
- Salomone, E., Charman, T., McConachie, H., & Warreyn, P. (2016). Child's verbal ability and gender are associated with age at diagnosis in a sample of young children with ASD in Europe. *Child: Care, Health and Development*, 42(1), 141–145.
- Shattuck, P. T., Durkin, M., Maenner, M., Newschaffer, C., Mandell, D. S., Wiggins, L., . . . Kirby, R. (2009). Timing of identification among children with an autism spectrum disorder: Findings from a population-based surveillance study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 48(5), 474–483.
- Slade, E. (2003). The relationship between school characteristics and the availability of mental health and related health services in middle and high schools in the United States. *The Journal of Behavioral Health Services & Research*, 30, 382–392.
- Thomas, P., Zahorodny, W., Peng, B., Kim, S., Jani, N., Halperin, W., & Brimacombe, M. (2012). The association of autism diagnosis with socioeconomic status. *Autism*, 16(2), 201–213.
- Zwaigenbaum, L., Bauman, M. L., Choueiri, R., Fein, D., Kasari, C., Pierce, K., . . . Hansen, R. L. (2015). Early identification and interventions for autism spectrum disorder: Executive summary. *Pediatrics*, 136(Suppl. 1), S1–S9.

Dendrite

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Definition

A dendrite is one of the four main parts of neurons, which also include the cell body, the axon, and the axon terminals. The dendrite is the part of the neuron that receives incoming signals from other neurons. The signals come in the form of neurotransmitters that cross the area of the synapse between one neuron and another neuron's dendrites. Neurotransmitters bind to receptors on the dendrites, and then the signal passes through the neuron to the cell body. Dendrites may have extensive branching, and each neuron often has multiple dendrites.

The number of dendrites and thus the number of synapses vary with the functions of a neuron. The dendrites of one neuron may receive signals from thousands of other neurons. That one neuron then integrates many signals received and responds accordingly.

See Also

- Neuroanatomy
- Neurochemistry
- Neurotransmitter
- Purkinje Cells

References and Reading

- Corwin, E. (2006). *Handbook of pathophysiology* (3rd ed., pp. 185–187). Philadelphia: Lippincott Williams & Wilkins.

Dendritic Cells and Their Expression of Costimulatory Molecules in Children with Autism Spectrum Disorders

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Background

Autism spectrum disorder (ASD) encompasses early-onset neurodevelopmental mishaps afflicting about 1 in every 160 children worldwide. It develops through a complex set of etiologies that involve immunological, environmental, and genetic factors (Jia et al. 2018a, Bjørklund et al. 2019). Individuals diagnosed with ASD may display a range of problem behaviors such as hyperactivity, poor attention, impulsivity, aggression, self-injury, and tantrums. The lack of successful therapy, genetic heterogeneity, and the increasing incidence make it one of the most challenging disorders. At present, only some comorbid problems of ASD could be treated, but

not the core manifestations (El-Rashidy et al. 2017; Cheng et al. 2017; Shaaban et al. 2018). The etiopathogenesis of ASD is complicated and debatable. Immunological abnormalities have been recently linked with the pathology of ASD. The chemosensory immune system has a vital role in the process of neurodevelopment by regulation of the neuronal proliferation, plasticity, and synapse development. Besides, it removed the apoptotic neurons and contributed to numerous neurological activities (Wang et al. 2014; Bjørklund et al. 2016). Numerous revisions over the last 20 years have recognized immune disorders, including both decreased immune functions and higher autoimmunity among children diagnosed with ASD (see for details Bjørklund et al. 2016). Immune system abnormalities may have a role in autism development; however, there is no consensus regarding whether these disorders initiate the onset of core manifestations or regulate the symptomatology and pathogenesis of autism (Wang et al. 2014). Individuals with ASD are more susceptible to various types of infections, asthma, allergies, and seizures. Also, autistic patients usually have peculiar reactions to vaccines and autoimmune diseases (Hsiao 2013; Wang et al. 2014; Bjørklund et al. 2016).

Dendritic Cells

Dendritic cells (DCs) currently are known as a distinct hematopoietic lineage of myeloid cells, together with monocytes, granulocytes, and macrophages. They originate from the bone marrow (BM) through specific ancestor subclasses and actively contributed to both adaptive and innate immunity and managing the equilibrium between tolerance and immunity. DCs are powerful phagocytic cells and considered one of the most effective specialized antigen-presenting cells (APCs) that are required for initiation and spreading the immune responses (Rhodes et al. 2019). DCs are responsible for uptaking, processing antigens, and then presenting them to T-lymphocytes (Patente et al. 2019). DCs recognize antigens via Toll-like

receptors, which are specific for certain molecules present in bacteria, fungi, viruses, and parasites (Patente et al. 2019). In the peripheral blood, DCs are immature that upon stimulation give rise to two active forms. Based on the functional and phenotypic features, DCs are classified into two lineages. The first type is myeloid dendritic cells (mDCs) CD11c+CD123−, also called conventional DCs, which present mainly in the peripheral blood and lymphoid tissues. mDCs represent the antigen-presenting dendritic cells that summit the adaptive immunity (Patente et al. 2019; Rhodes et al. 2019). mDCs are highly specialized cells similar to monocytes and are secondly divided into *mDC-1* cells which are the main stimulator of CD8⁺ cells, which is very important in antiviral and antitumor activity (Brewitz et al. 2017), and the less common *mDC-2* cells which have several immunoregulatory functions. They are potent inducers for Th1, Th2, and Th17 immune responses. Also, mDC-2 activate T-regulatory cells in the gastrointestinal tract. In addition, mDC2 stimulate CD4⁺-naïve T-lymphocytes to express gut-homing molecules (Segura et al. 2012; Leal Rojas et al. 2017).

The second type is plasmacytoid dendritic cells (pDCs) CD11c−CD123+, which are found in small numbers in the peripheral blood. pDCs have a potential role as APCs, as they express co-stimulatory molecules and MHC II. Also, they have an antiviral role through the production of interferon I and III and priming CD8⁺ T-lymphocytes and natural killer cells (Schlitzer et al. 2018; Rhodes et al. 2019).

Costimulatory Molecules B7 (CD80/CD86)

CD80 and CD86 are a group of cell surface glycoproteins of the B7 family, structurally related to immunoglobulins. These molecules play an important role in the regulation of innate and adaptive immune system (Rhodes et al. 2019). Previous studies have reported that B7-1 (CD80) and B7-2 (CD86) are the most characterized

costimulatory molecules for activation of T-lymphocytes (Fujii et al. 2004; Schlitzer et al. 2018). CD80 is expressed in low levels on the surface of DCs, APCs and monocytes; however, CD86 is highly expressed in these cells. CD80 and CD86 can either stimulate T-lymphocytes through binding to CD28 receptor or deactivate T-cells through binding to CTLA-4 (cytotoxic T-lymphocyte-associated antigen) receptor. CD80 and CD86 are exclusively essential in DCs' activation. When the MHC class II peptide on DCs interacts with T-helper cells, CD80 and CD86 are upregulated, allowing the activation of DCs and contact them to CD8⁺. This supports T-lymphocyte differentiation into cytotoxic T-cell (Fujii et al. 2004; Zheng et al. 2004).

As stated before, these costimulatory molecules have a pivotal role in DCs activation. This activation occurs through CD28 interactions with increase the mobility of DCs for migration and release of cytokines specifically IL-6, with activation of cellular proliferation, and inhibit apoptosis (Zheng et al. 2004; Schlitzer et al. 2018). On the other hand, these molecules have inhibitory effects on DCs through interaction with CTLA-4. This suppressive effect helps regulatory T-lymphocytes to avert any immunological response to self-antigens (Fujii et al. 2004; Zheng et al. 2004).

Dendritic Cells and Costimulatory Molecules in Autism Spectrum Disorders

In our previous work (Saad et al. 2017), we assessed the levels of DCs and CD86 and CD80 costimulatory molecules in children with ASD and compared to normal children:

Myeloid Dendritic Cells (mDCs)

Our study showed significantly augmented levels of mDCs in children with ASD when compared to typically healthy children (2.44 ± 0.20 vs. 1.54 ± 0.09 ; $p = 0.006$). Furthermore, we noticed positive correlations between the frequency of mDCs and autism severity

($p < 0.01$), stereotypy ($p < 0.05$), social understanding ($p < 0.001$), emotional response ($p < 0.01$), and hyperactivity ($p < 0.05$), as measured on CARS and ABC scales (Saad et al. 2017). In line with our findings, Breece et al. (2013) reported significantly increased mDCs frequencies in autistic children as compared to normal controls ($4.6\% \pm 0.41$ vs. $3.70\% \pm 0.38$; $p = 0.03$). They found the percentages of mDCs were higher by 25% to 40% in autistic patients when compared to normal children. Besides, they found significant positive correlations between the frequency of mDCs and stereotypic behavior ($p = 0.02$; effect size = 0.43) and the severity of GIT symptoms especially constipation ($p = 0.002$; effect size 1.85) (Breece et al. 2013). In addition, they reported significant positive associations between mDCs and the volume of the left amygdala ($p = 0.004$), and right amygdala ($p = 0.02$), in autistic children after adjustment of sex and age of the patients' group.

Plasmacytoid Dendritic Cells (pDCs)

In our study, pDCs' frequencies were higher in autistic patients when compared to controls ($1.98\% \pm 0.35$ vs. $1.18\% \pm 0.05$; $p = 0.036$). Furthermore, we found a positive association between mDCs and autism severity ($p = 0.05$). However, we could not find any correlations with autism core manifestations and pDCs (Saad et al. 2017). Contrary to our results, Breece et al. (2013) reported comparable frequencies of pDCs in ASD patients and typically developing controls. They reported significant correlations between pDCs percentages and the right amygdala ($p = 0.03$) and left amygdala volumes ($p = 0.02$) (Breece et al. 2013).

Costimulatory Molecules CD80 and CD86

Our data showed a significant upregulation in the expression of CD80 (49.67 ± 4.67 vs. 23.16 ± 1.39 ; $p = 0.001$) and CD86 (55.37 ± 2.94 vs. 39.42 ± 4.7 ; $p = 0.001$) costimulatory molecules on the entire DCs in autistic children when compared to control group. In addition, the mean fluorescent intensity of CD86+ (149.36 ± 9.52 vs. 111.26 ± 8.26 ; $p = 0.003$) and CD80+ (76.07 ± 3.18

vs. 52.05 ± 3.59 ; $p = 0.001$) were significantly higher in ASD group than controls (Saad et al. 2017).

For the first time, our results showed an inverse association between the frequencies of both types of DCs and vitamin D levels in autistic children. Recently, many pieces of research and reviews focused on the role of vitamin D in CNS and immunological disorders (Saad et al. 2016; Björklund et al. 2016, 2019; Jia et al. 2018a). In addition to the well-known metabolic functions of vitamin D in bone and mineral metabolism, plentiful studies reported that vitamin D has several functions in the process of brain development by affecting the axonal connectivity and neuronal differentiation. In ASD, previous studies have shown significant associations between vitamin D deficiency and increased risk of autism (for details see Jia et al. 2018b). Additionally, vitamin D deficiency during pregnancy was associated with numerous adverse effects on the fetus, e.g., intra-uterine growth retardation (Björklund et al. 2016; Jia et al. 2018b). Vitamin D is a key modulator of the immune system, playing an important physiological role in the regulation of both adaptive and innate immunity. Vitamin D is shared in the process of DC maturation and affected their functions through multiple mechanisms. In DCs, vitamin D can make a resistant phenotype with low HLA-DR, CD80, and CD86 expressions with a high ratio of interleukin10/interleukin12p70 (Ferreira et al. 2015). Furthermore, the differentiation of DCs in the presence of active form of vitamin D ($1,25D_3$ -DCs) leads to failure of DCs to stimulate autoreactive T-lymphocytes, activating in its place antigen-specific T-regulatory cells resulting in induction of infectious tolerance and preservation of DC tolerogenic response (Ferreira et al. 2014, 2015).

Conclusion

Taken together, the previous data suggest many systemic immune abnormalities in patients with ASD. Significant variations in the frequencies and functions of the myeloid lineage cells, specifically DCs, macrophages, and monocytes, have been

extensively investigated in many types of research in children with ASD (see Bjorklund et al. 2016 for details). We suggested that DCs may play a role in the pathogenesis of ASD. However, the exact mechanism of the correlations of DCs and their costimulatory molecules in ASD remains to be elucidated.

References and Reading

- Bjorklund, G., Saad, K., Chirumbolo, S., Kern, J. K., Geier, D. A., Geier, M. R., & Urbina, M. A. (2016). Immune dysfunction and neuroinflammation in autism spectrum disorder. *Acta Neurobiologiae Experimentalis (Wars)*, 76(4), 257–268.
- Bjorklund, G., Waly, M. I., Al-Farsi, Y., Saad, K., Dadar, M., Rahman, M. M., et al. (2019). The role of vitamins in autism spectrum disorder: What do we know? *Journal of Molecular Neuroscience*, 67(3), 373–387.
- Braudeau, C., Graveleau, J., Rimbart, M., Néel, A., Hamidou, M., et al. (2013). Altered innate function of plasmacytoid dendritic cells restored by enzyme replacement therapy in Gaucher disease. *Blood Cells, Molecules & Diseases*, 50(4), 281–288.
- Breece, E., Paciotti, B., Nordahl, C. W., Ozonoff, S., Van de Water, J. A., Rogers, S. J., et al. (2013). Myeloid dendritic cells frequencies are increased in children with autism spectrum disorder and associated with amygdala volume and repetitive behaviors. *Brain, Behavior, and Immunity*, 31, 69–75.
- Brewitz, A., Eickhoff, S., Dähling, S., Quast, T., Bedoui, S., Kroczeck, R. A., et al. (2017). CD8+ T cells orchestrate pDC-XCR1+ dendritic cell spatial and functional cooperativity to optimize priming. *Immunity*, 46, 205–219.
- Cheng, N., Alshammari, F., Hughes, E., Khanbabaei, M., & Rho, J. M. (2017). Dendritic overgrowth and elevated ERK signaling during neonatal development in a mouse model of autism. *PLoS One*, 12(6), e0179409.
- El-Rashidy, O., El-Baz, F., El-Gendy, Y., Khalaf, R., Reda, D., & Saad, K. (2017). Ketogenic diet versus gluten free casein free diet in autistic children: A case-control study. *Metabolic Brain Disease*, 32(6), 1935–1941.
- Ferreira, G. B., Gysemans, C. A., Demengeot, J., da Cunha, J. P., Vanherwegen, A. S., Overbergh, L., et al. (2014). 1,25-Dihydroxyvitamin D3 promotes tolerogenic dendritic cells with functional migratory properties in NOD mice. *Journal of Immunology (Baltimore, Md: 1950)*, 192, 4210.
- Ferreira, G. B., Vanherwegen, A. S., Eelen, G., Gutiérrez, A. C., Van Lommel, L., Marchal, K., et al. (2015). Vitamin D3 induces tolerance in human dendritic cells by activation of intracellular metabolic pathways. *Cell Reports*, S2211–1247(15), 711–725.
- Fujii, S., Liu, K., Smith, C., Bonito, A. J., & Steinman, R. M. (2004). The linkage of innate to adaptive immunity via maturing dendritic cells in vivo requires CD40 ligation in addition to antigen presentation and CD80/86 costimulation. *The Journal of Experimental Medicine*, 199(12), 1607–1618.
- Hsiao, E. Y. (2013). Immune dysregulation in autism spectrum disorder. *International Review of Neurobiology*, 113, 269–302.
- Jia, F., Shan, L., Wang, B., Li, H., Feng, J., Xu, Z., et al. (2018a). Fluctuations in clinical symptoms with changes in serum 25(OH) vitamin D levels in autistic children: Three cases report. *Nutritional Neuroscience*, 8, 1–4.
- Jia, F., Shan, L., Wang, B., Li, H., Miao, C., Xu, Z., et al. (2018b). Bench to bedside review: Possible role of vitamin D in autism spectrum disorder. *Psychiatry Research*, 260, 360–365.
- Leal Rojas, I. M., Mok, W. H., Pearson, F. E., Minoda, Y., Kenna, T. J., Barnard, R. T., et al. (2017). Human blood CD1c+ dendritic cells promote Th1 and Th17 effector function in memory CD4+ T cells. *Frontiers in Immunology*, 8, 563–511.
- Patente, T. A., Pinho, M. P., Oliveira, A. A., Evangelista, G. C. M., Bergami-Santos, P. C., & Barbuto, J. A. M. (2019). Human dendritic cells: Their heterogeneity and clinical application potential in cancer immunotherapy. *Frontiers in Immunology*, 9, 3176.
- Rhodes, J. W., Tong, O., Harman, A. N., & Turville, S. G. (2019). Human dendritic cell subsets, ontogeny, and impact on HIV infection. *Frontiers in Immunology*, 10, 1088.
- Saad, K., Abdel-Rahman, A. A., Elserogy, Y. M., Al-Atram, A. A., Cannell, J. J., Bjorklund, G., et al. (2016). Vitamin D status in autism spectrum disorders and the efficacy of vitamin D supplementation in autistic children. *Nutritional Neuroscience*, 19(8), 346–351.
- Saad, K., Zahran, A. M., Elsayh, K. I., Abdel-Rahman, A. A., Al-Atram, A. A., Hussein, A., & El-Gendy, Y. G. (2017). Frequency of dendritic cells and their expression of costimulatory molecules in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 47(9), 2671–2678.
- Schlitzer, A., Zhang, W., Song, M., & Ma, X. (2018). Recent advances in understanding dendritic cell development, classification, and phenotype. *F1000Res*, 7, pii: F1000. Faculty Rev-1558.
- Segura, E., Valladeau-Guilemond, J., Donnadiou, M.-H., Sastre-Garau, X., Soumelis, V., & Amigorena, S. (2012). Characterization of resident and migratory dendritic cells in human lymph nodes. *The Journal of Experimental Medicine*, 209, 653–660.
- Shaaban, S. Y., El Gendy, Y. G., Mehanna, N. S., El-Senousy, W. M., El-Feki, H. S. A., Saad, K., & El-Asheer, O. M. (2018). The role of probiotics in children with autism spectrum disorder:

- A prospective, open-label study. *Nutritional Neuroscience*, 21(9), 676–681.
- Wang, T. T., DU, L., Shan, L., & Jia, F. Y. (2014). Research advances in immunological dysfunction in children with autism spectrum disorders. *Zhongguo Dang Dai Er Ke Za Zhi*, 16, 1289–1293.
- Zheng, Y., Manzotti, C. N., Liu, M., Burke, F., Mead, K. I., & Sansom, D. M. (2004). CD86 and CD80 differentially modulate the suppressive function of human regulatory T cells. *Journal of Immunology*, 172(5), 2778–2784.

Dental Care

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Definition

Dental care is just as important (if not more important) for children with autism. Unfortunately, various factors make attaining adequate dental care challenging for children with autism. While parents are understandably reluctant to stress their child – particularly a child with oral sensitivities – lack of dental care early in life can lead to major long-term health problems (Volkmar and Wiesner 2009).

Historical Background

As noted elsewhere in this volume, a considerable body of work on the nature of autism and related conditions (Autism Spectrum Disorders, ASDs) exists. These conditions are characterized by major problems in social interaction, communication, and behavior. These individuals range from intellectually deficient to above average IQ. Unfortunately, deficits described above can make providing quality dental health care difficult and unpredictable (Stein et al. 2014). In addition

to difficulties completing dental treatment, parents also have difficulty accessing care (Kogan et al. 2008) or may avoid care because of stress related to appointment experience (Weil and Inglehart 2012). For these reasons, managing their oral health can be very challenging for health care providers.

Current Knowledge

As with regular medical care, establishing a “dental home” is a major step forward in providing comprehensive ongoing dental care for children with autism (Nowak and Casamassimo 2002). Unfortunately, finding a dental home can be difficult because very few general dentists are comfortable treating these individuals because of behavior challenges. Some sources report fewer than one in ten general dentists will treat individuals with special health care needs (Casamassimo et al. 2004). As a result, individuals with autism are often forced to visit multiple offices before finding a dentist who is willing to treat them. Frustration associated with this process can be avoided when patients specifically seek out a pediatric dentist to treat their child’s dental needs. Pediatric dentists complete a 2-year residency program in addition to dental school. In residency, they learn to provide dental care for infants and children through adolescence, including patients with special health care needs. Many pediatric dentists will continue to see patients with special health care needs such as autism well into adulthood. As patients get older, they may start to develop some oral health needs that pediatric dentists are not comfortable addressing. Pediatric dentists can be a helpful resource for finding caregivers who treat more adult needs and are comfortable treating patients with autism.

When searching for a pediatric dentist, parents should be aware that locating a pediatric dentist currently accepting new patients might be difficult (Capozza 2012). The availability of pediatric dental practitioners varies widely by state. Additionally, lower reimbursement by state insurance programs like Medicaid may pose further obstacles for care. The American Academy of Pediatric

Dentistry has developed a provider search engine to help parents find pediatric dentists in their area (<http://www.aapd.org/finddentist/>).

Oral Health Concerns. Individuals with ASD experience many oral health conditions requiring management by dental professionals similar to the general population. Like the general population, the most commonly reported oral health concern individuals with ASD experience is dental decay (Stein et al. 2012). In addition to dental decay, other commonly reported oral health problems include periodontal disease, dental trauma related to coordination problems, and dental trauma related to self-injury (Volkmar and Wiesner 2009). Individuals with ASD also experience orthodontic problems like anterior open bite, crowding, and diastema (Delli et al. 2013).

It is important to understand that dental decay is an enormous problem for all children in the United States regardless of health status. Dental decay is the most common chronic childhood disease today with an estimated 16 million children currently affected. Oral disease is responsible for children missing 51 million school hours and parents missing 25 million work hours annually. Children from low-income families have significantly more untreated decay than the rest of the population.

Considering the high rate of decay experienced by the general population, one may expect individuals with autism to also experience a high rate of dental decay. Current studies, however, do not give clear confirmation that this is the case. Although it is generally agreed that there is a higher risk for dental caries in ASF although data addressing this is rather limited likely related to differences in samples studied (see Loo et al. 2008). Caries prevention in this population is an obvious priority.

Prevention

While the patient, parents, and dentist play the primary role in prevention of dental disease, all members of the health care team need to be aware of dental caries prevention strategies. Establishment of the dental home is the first step in this process preventing dental decay (Nowak and Casamassimo 2002). Ideally, the dental home

should be established within 6 months of the eruption of the first tooth or no later than 1 year of age. Similarly, the medical home should also be established at this time. One goal of the dental home is to break the current cycle of problem-initiated care seeking. The dental home works to help establish good oral health practices through patient and parent education. The dental home also identifies dental disease early in the disease process when it can be most easily managed. The dental home is also responsible for referral to the appropriate specialist when the patient's dental needs are beyond the scope of practice of the dental home.

Caries risk assessment is a tool that uses caries risk indicators to identify habits that are likely to result in the development of dental decay. Identifying these high-risk habits allows preventive strategies to be customized for each patient, so dental decay can be avoided and/or the decay process can be stopped when it is experienced (AAPD 2013). This is especially helpful for patients with ASD because of how little we know about this population's caries experience and the many caries risk indicators with which these patients can present. A risk assessment will ideally have sufficient data to accurately determine disease susceptibility and offer preventive strategies.

Beyond being broadly classified as high caries risk because of special needs status, individuals with autism also have risk factors specifically related to their disorder. Some risk factors include poor oral hygiene, detrimental oral behaviors, medication-induced xerostomia, concurrent medical diagnoses, low cognitive level, poor dietary habits, gastric reflux, a preference for soft/sweet foods, use of sweets for behavior modification, and requiring help with tooth brushing. Some risk factors occur more commonly than others in this population, but no child will display exactly the same risk factors.

The next strategy for preventing dental decay is increasing the patient and caregiver's knowledge through education (Klein and Nowak 1998). Caregivers and patients should have a basic understanding of how dental decay occurs so they can be proactive in making good oral hygiene

decisions. Everyone involved in the patient's care should be aware of the four factors that must come together for a cavity to form, i.e., presence of teeth, the presence of bacteria, the presence of a nutrition source for the bacteria, and time for the decay to develop. While the presence of a tooth and time cannot be modified easily, the other two factors can be. The first modifiable variable is the level of cariogenic bacteria in the mouth. The second factor is the level of fermentable carbohydrates present in the oral environment (Featherstone 2004).

Bacteria levels can be reduced through good oral hygiene (brushing twice per day with fluoride toothpaste and flossing once per day). Fermentable carbohydrates can be reduced through diet control (avoiding foods that are especially cariogenic). Caregivers have total control of diet in this situation and can help prevent decay by drastically reducing fermentable carbohydrates in the diet. In the case of a higher cognitively functioning individual, the child may have more independence when it comes to food choices. With this scenario, effective oral hygiene skills can be taught, therefore reducing bacteria level in the oral environment.

While diet and oral hygiene can both be targeted to reduce caries risk, improving oral hygiene may be the best way to reduce caries risk in the autistic population. Difficulty with home care can be explained by many of the same reasons, and treatment in the dental clinic is also difficult. Tooth brushing can cause problems with individuals who have oral sensitivity, poor motor control, poor understanding, and lack of interest. Fluoride is safe and effective at reducing tooth decay when used as directed (Volkmar and Wiesner 2009). In fact, the Center for Disease Control and Prevention considers water fluoridation to be one of the ten greatest public health achievements of the twentieth century with more than 150 million Americans benefiting from fluoridated water. If there is concern that a patient may swallow toothpaste, extra care should be taken to make sure the proper amount of toothpaste is used during each brushing session. Children under the age of 2 should only have a smear of toothpaste applied to the toothbrush, while children over the

age of 2 can use a pea-sized drop of toothpaste each time they brush (Guidelines on Fluoride Therapy 2014)

Visual teaching can also be used to help improve daily oral hygiene skills. Studies using a simple photo storybook to outline home care and in office procedures have helped improve procedural success (Volkmar and Wiesner 2009). Equally as important, in one study, parents saw the value in the intervention and continued using the storybook when the study ended (Backman and Pilebro 1999).

For the uncooperative child, various strategies may be employed, e.g., three-sided toothbrush. The three-sided toothbrush allows the parent to clean the buccal, lingual/palatal, and occlusal surfaces of the tooth all at once, reducing brushing time and increasing the chance of cleaning all tooth surfaces. Another helpful tool is the mouth prop. Mouth props are placed on the opposite side of the mouth from the side being brushed to prevent the child from biting down. Mouth props can be as simple as a stack of tongue blades taped together or the handle end of a toothbrush. Mouth props can also be purchased online from special needs dental supply websites. Finally, floss sticks and interproximal brushes can be used instead of traditional dental floss. These products may be easier to use when flossing a difficult child, because they can be used with one hand.

Appointment Preparation and the Dental Visit

Challenges for office visits for the child with ASD arise given the children often have difficulties with change, have limited communication skills, can be highly anxious, and variably related socially. These children may dislike being touched and may be alarmed by unusual sounds or bright lights. Accordingly, many behavior guidance techniques commonly used by dental professionals may be of limited use (Marshall et al. 2007). Thus success in the dental chair is largely dependent on preparation for the visit. Caregivers, like parents and therapists, can also do many things to help prepare the patient for the visit. At home, caregivers can help the patient prepare for the visit using exercises to familiarize the patient with what to expect at the appointment by

using visual aids and appointment practice (Pilebro and Backman 2005; Volkmar and Wiesner 2009). Parents are often quite good predictors of the child's behavior. Simply asking things like whether the child can sit for a haircut, if the child is toilet trained, and how successful tooth brushing is at home can serve as initial predictors of cooperativeness. It is important to understand parental concerns or questions, e.g., relative to fluoride, diet, behavior management strategies, and so forth. Parents may have questions about mercury or use of nitrous oxide, radiation, and other materials (Rada 2010). Strategies that can help include predict in use of the dental chair, and visual strategies have been successfully used to manage a range of problems and enhance compliance with health care and make use of the typical strengths children with autism have in the area of nonverbal, visual, static, and sequential information presentation (Hogdon 1995). Storybooks can demonstrate what the dental office, waiting room, and treatment rooms look like. The storybook can also show pictures of dental instruments and pictures of what the dental providers look like. These materials are readily prepared on today's computers and can be used to facilitate cooperation. Realistic goals should be set for each appointment. Staff should notice patterns of success and gradually build upon these. Understanding and consistency on the part of care providers is important, e.g., scheduling so as to avoid waiting time.

For the dental visit, applied behavior analysis (ABA) strategies can help interpret and modify behavior. It is also important for providers to understand that poor behavior can be unintentionally negatively reinforced the same way positive behavior can be reinforced. Children with autism often demonstrate oral defensiveness, escape, and avoidance behaviors at their dental visit attempting to avoid treatment (Delli et al. 2013). Providers must understand they are only reinforcing this behavior by allowing patients to avoid treatment, e.g., patient is experiencing a stimulus, reacting, and is rewarded when the undesired stimulus is removed. Introducing treatment in steps with desensitization visits can also help patients have a positive experience. The first time the patient comes to the office, the visit could simply consist of sitting in the waiting room.

From there, the patient may walk back to a treatment room. Patients continue to experience more each visit until the desired procedure is completed. Although this strategy can be effective, it is also very time consuming and may be difficult to implement in the private dental office. However, desensitization visits can be planned to minimize the amount of time spent on visits that are not truly benefiting the patient. When the first two visits just require visiting different parts of the office, appointments are quick and do not require significant time commitment for the dentist or office staff.

Given problems in relating and communication, it is important to realize that the frequent patterns of becoming overly polite when a person is having problem may be less helpful than direct instruction, e.g., instead of saying, "Please come sit in the chair so we can look at your teeth," the provider may have more success saying, "Sit here." Another way positive outcomes may be increased is by wearing a clear face shield instead of the traditional cloth facemask. Eye-tracking studies show that individuals with autism spend much more time focused on the mouth. Wearing a clear face shield allows individuals with autism to continue to gather information from the dentist's lower face throughout the procedure.

Staff should be aware of sensory overstimulation, e.g., the movement of the dental chair may cause anxiety, and this could be avoided by allowing the patient to stand while the chair reclines. Use of unscented/unflavored products can be helpful. minimize these obstacles. Applying dental products to a few teeth and quickly wiping away the excess as each tooth is cleaned can minimize sensation. "Paste-less" prophylactic angles can be extremely helpful when treating patients who cannot tolerate the sensation or taste of prophylactic paste in their mouth. If use of suction is a problem, gauze can be used to soak up saliva and water. The sound of the high-speed hand piece can also be distressing. When patients are having trouble tolerating bright light, dental mirrors with lights on the handle can be invaluable treatment tools.

Completing dental radiographs is a very important portion of the dental visit and is an essential part of the dental exam and diagnosis. Dental

radiographs provide information about the oral cavity that cannot be discerned any other way. Radiographs can be the deciding factor in determining whether or not a patient needs treatment in the operating room and how much operating room time will be needed. If radiographs cannot be completed, the dental provider must schedule extended operating room time for the patient in case the patient has extensive interproximal dental decay that cannot be visualized clinically. Many of the same strategies previously mentioned can be used in taking radiographs. It traditional bite-wing radiographs are impossible to complete, providers should consider attempting panoramic bitewings

Some papers report that the protective stabilizer (papoose) is used in as high as 44% of the time with children who have ASD. If providers are considering placing the child in a papoose, it should be discussed with parents at length before starting treatment. Every effort should be made to ensure the parent understands the purpose of the protective stabilizer and why it is used (9Eatonk 2005).

Pharmacological Treatment Aids

If other strategies are not successful, pharmacological management of behavior should be considered. There are many sedation protocols available for in-office treatment ranging from minimal to deep sedation. Services offered differ based on the provider's training, experience, and patient factors such as previous sedation experiences, health history, and current health status. In office, sedation is traditionally reserved for healthy patients with no medical complications. Outcomes of sedation procedures can be unpredictable even with the most experienced providers, and unpredictable and paradoxical reaction to some sedative agents have been noted (Volkmar and Wiesner 2009). If all other efforts fail, one option is generally anesthesia; treatment must be completed in the operating room under general anesthesia, and parents are often comfortable with this approach (Cuvo 2010). In such cases, consultation with the primary health care provider is important. The decision to treat a patient under general anesthesia should not be an

excuse to abandon intervention strategies that have been discussed to improve behavior at the dental visit. Behavior is less likely to improve over time in children with ASD than the general population, but improved behavior is possible with hard work. Dental providers and caregivers should continue to work towards successful completion of the dental visit in the office environment.

Future Directions

Regardless of caries rate experienced by the autistic population, all children are at risk for developing dental decay. For that reason, every child requires routine preventative dental care. When treating individuals with autism, it is important to remember that every patient will present with unique challenges and will respond differently to various intervention strategies (Delli et al. 2013). Providers must realize that an intervention strategy may be very effective for one patient and totally ineffective for another. Given the wide range in syndrome expression, clearly there is no single "cook book" to success when treating individuals with ASD.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Dental Care](#)
- [Medical Home and ASD](#)

References and Reading

- Backman, B., & Pilebro, C. (1999). Visual pedagogy in dentistry for children with a autism. *ASDC Journal of Dentistry for Children*, 66(5), 325–331, 294.
- Capozza, L. E., & Bimstein, E. (2012). Preferences of parents of children with autism spectrum disorders concerning oral health and dental treatment. *Pediatric Dentistry*, 34(7), 480–484.
- Casamassimo, P. S., Seale, N. S., & Ruehs, K. (2004). General dentists' perceptions of educational and treatment issues affecting access to care for children with special health care needs. *Journal of Dental Education*, 68(1), 23–28.

- Cuvo, A. (2010). Training children with autism spectrum disorders to be compliant with the oral assessment. *Research in Autism Spectrum Disorders*, 4, 681–696.
- Delli, K., Reinhard, P. A., Bornstein, M. M., & Livas, C. (2013). Management of children with autism spectrum disorder in the dental setting: concerns, behavioural approaches and recommendations. *Medicina Oral, Patología Oral y Cirugía Bucal*, 18(6), e862–e868.
- Featherstone, J. D. (2004). The continuum of dental caries – evidence for a dynamic disease process. *Journal of Dental Research*, 83(Spec No C), C39–C42.
- Guidelines on Fluoride Therapy. (2014). 2014–15 Definitions, oral health policies, and clinical guidelines [cited 2014]. Available from: http://www.aapd.org/media/Policies_Guidelines/G_FluorideTherapy.pdf
- Hogdon, L. A. (1995). *Visual strategies for improving communication: Practical supports for school and home*. Troy: Quick & Roberts Publisher.
- Klein, U., & Nowak, A. J. (1998). Autistic disorder: A review for the pediatric dentist. *Pediatric Dentistry*, 20(5), 312–317.
- Kogan, M. D., Strickland, B. B., Blumberg, S. J., Singh, G. K., Perrin, J. M., & Van Dyck, P. S. C. (2008). A national profile of the health care experiences and family impact of autism spectrum disorder among children in the United States, 2005–2006. *Pediatrics*, 122(6), e1149–e1158.
- Marshall, J., Sheller, B., Mancini, L., & Williams, B. J. (2007). Parental attitudes regarding behavior guidance of dental patients with autism. *Pediatric Dentistry*, 30(5), 400–407.
- Nowak, A. J., & Casamassimo, P. S. (2002). The dental home: A primary care oral health concept. *Journal of the American Dental Association* (1939), 133(1), 93–98.
- Pilebro, C., & Backman, B. (2005). Teaching oral hygiene to children with autism. *International Journal of Paediatric Dentistry*, 15(1), 1–9.
- Rada, R. E. (2010). Controversial issues in treating the dental patient with autism. *Journal of the American Dental Association* (1939), 141(8), 947–953.
- Stein, L. I., Polido, J. C., Najera, S. O., & Cermak, S. A. (2012). Oral care experiences and challenges in children with autism spectrum disorders. *Pediatric Dentistry*, 34(5), 387–391.
- Stein, L. I., Lane, C. I., Williams, M. E., Dawson, M. E., Polido, J. C., & Cermak, S. A. (2014). Physiological and behavioral stress and anxiety in children with autism spectrum disorders during routine oral care. *BioMed Research International*, 2014, 694876.
- Volkmar, F. R., & Wiesner, E. A. (2009). *A practical guide to autism*. Hoboken: Wiley.
- Weil, T. N., & Inglehart, M. R. (2012). Three- to 21-year-old patients with autism spectrum disorders parents perceptions of severity of symptoms, oral health, and oral health-related behavior. *Pediatric Dentistry*, 34(7), 473–479.

Denver Development Screening Test (DDST)

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Synonyms

DDST; Denver II

Description

The Denver Developmental Screening Test, first published in 1967 (Frankenburg and Dodds 1967), was one of the first screening tools developed to identify young children at risk for developmental delay and disability. Its format was similar to the construction of pediatric growth charts, with 105 developmental items for children from birth to 6 years of age aligned chronologically along horizontal age lines, divided into four discrete developmental domains: personal-social, fine motor-adaptive, language, and gross motor. Bar graphs for each developmental item reflect the ages at which 25%, 50%, 75%, and 90% of typically developing children in the standardization sample completed the task. Because of criticisms related to low sensitivity in identifying children with speech and language delays, it was revised to add more language items, restandardized, and remarketed as the Denver II in 1992 (Frankenburg et al. 1992), retaining a similar format to the DDST. Both are administered by individuals who have trained to proficiency using training tapes/DVD or the administration manual, with standardized toys such as blocks, rattles, and pictures included in the toolkit. It is estimated to take 10–20 min to administer and score. Several options regarding item administration were developed and evaluated. The most commonly used approach in primary care settings included administering at least three items in each domain whose bar graphs are closest to but completely to the left

of the age line, indicating items that over 90% of typically developing children should be able to do by that age. Any of these items not successfully completed are considered a delay; items where the age line passes through the 75–90% section of the bar graph which the child cannot accomplish are scored “cautions,” and an algorithm for determining normal, abnormal (two or more delays), and questionable (two cautions or one delay) results is provided in the manual.

Historical Background

The DDST is most important for its historical background rather than as a currently recommended screening tool. It was the first developmental screening tool for young children that was widely marketed to the primary care medical community as well as child care providers and other child health professionals. It played a significant role in widespread recognition of the importance of early identification and intervention from a public health and primary care perspective. The role of parents as accurate observers of their children’s behaviors was also recognized by the developers of the DDST and subsequent screening materials such as the Denver Prescreening Developmental Questionnaire and the Denver II. The DDST and Denver II became widely used in the United States and internationally, being translated and restandardized in many countries.

Psychometric Data

The DDST was originally standardized on 1,036 children from Denver, age 1–72 months, with reported co-positivity scores of .92 and co-negativity scores of .99, using the Bayley Mental and Psychomotor Scales and Stanford-Binet Intelligence Scales as criterion tests. Subsequent studies of concurrent and predictive validity, reviewed by Meisels (1989), found that while the specificity remained high (.87–1.0), the sensitivity was unacceptably low (.13–.46),

particularly when children were reevaluated 14 months to 6 years later (.18). The Denver II was standardized on 2,096 children 0–6.5 years of age, half from Denver and half from rural Colorado. Inter-rater reliability and test-retest validity were reported to be .90 or greater (Frankenburg et al. 1992). Subsequent studies showed that, with these revisions, the Denver II had acceptable sensitivity of .83 reported by Glascoe et al. (1992), but specificity dropped to .43, shifting concerns about the DDST failing to identify children with significant delays to concerns about overreferral of typically developing children using the Denver II.

Clinical Uses

The Denver II is still marketed by Denver Developmental Materials Inc. However, it is not included in the most recent American Academy of Pediatrics guidelines for general developmental surveillance and screening as a recommended tool (2006) nor in the AAP guidelines for ASD screening (2007), as it has never been evaluated as a screening tool for ASD. Its format, however, continues to be useful for pediatric educators as a way of visually illustrating the importance of assessing different developmental domains simultaneously in individual children, of tracking development over time, and of showing the variability in ages at which different developmental items “typically” occur.

References and Reading

- American Academy of Pediatrics. (2006). Identifying infants and young children with developmental disorders in the medical home: An algorithm for developmental surveillance and screening. *Pediatrics*, 118, 405–420.
- Frankenburg, W., & Dodds, J. (1967). The Denver developmental screening test. *Journal of Pediatrics*, 71, 181–191.
- Frankenburg, W. K., Dodds, J., Archer, P., Shapiro, H., & Bresnick, B. (1992). The Denver II: A major revision and restandardization of the Denver developmental screening test. *Pediatrics*, 89, 91–97.

- Glascoe, F. P., Byrne, K. E., Ashford, L. G., Johnson, K. L., Chang, B., & Strickland, B. (1992). Accuracy of the Denver II in developmental screening. *Pediatrics*, 89, 1221–1225.
- Johnson, C. P., & Myers, S. M. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120, 1183–1215.
- Meisels, S. (1989). Can developmental screening tests identify children who are developmental at risk? *Pediatrics*, 83, 578–585.

Denver II

- [Denver Development Screening Test \(DDST\)](#)

Denzapine

- [Clozapine](#)

Deoxyribonucleic Acid

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Synonyms

[DNA](#)

Definition

The instructions for the development and functioning of a living organism are contained in a molecule called deoxyribonucleic acid (DNA) which is a nucleic acid. The instructions are spelled out in a sequence or code of four chemical units called nucleobases (or bases for short). These are adenine, cytosine, guanine, and thymine, abbreviated as A, C, G and T, respectively. DNA is contained within nearly every cell of the human organism. Certain segments of the DNA

molecule called genes contain the code for creating the components of cells, most importantly, molecules called proteins (Alberts et al. 2002). James Watson and Francis Crick described the molecular structure of DNA in 1953 (Watson and Crick 1953).

DNA is passed from one generation to the next. In humans, the DNA molecule is divided up into a set of smaller pieces corresponding to chromosomes. Humans inherit 23 chromosomes from each parent, 22 of them are referred to as autosomes and are numbered 1–22 and one is called a sex chromosome and is either a chromosome X or a chromosome Y. Thus, normal human cells contain 44 autosomes and 2 sex chromosomes. The chromosomes are paired in each cell. For example, each cell will contain two copies of chromosome 1, one from the mother (the maternal chromosome) and one from the father (the paternal chromosome). Each of a pair of autosomes will generally contain the same genes. However, the sequence of DNA at each of the genes will often vary slightly between individuals, and it is also now clear that the structure of the chromosome, so-called copy number variations (CNVs), is also part of the normal complement of human genetic variation.

A change in the sequence or structure of DNA which results in a deviation from the agreed upon reference genome may be referred to in various ways, including an allele, a variant, a variation, a polymorphism, or a mutation. Typically, the word polymorphism is used when one is referring to a change that is present in a percentage of the population and mutation is taken to mean that the variation is rare and relates to a disease or phenotype.

See Also

- [Chromosomal Abnormalities](#)
- [Copy Number Variation](#)
- [Dizygotic \(DZ\) Twins](#)
- [Karyotype](#)
- [Monozygotic \(MZ\) Twins](#)

References and Reading

- Alberts, B., Bray, D., et al. (2002). *The cell*. New York: Garland Science.
- Bailey, A., Le Couteur, A., et al. (1995). Autism as a strongly genetic disorder: Evidence from a British twin study. *Psychological Medicine*, 25(1), 63.
- Strachan, T., & Read, A. P. (2004). *Human molecular genetics*. New York: Garland Press.
- Watson, J. D., & Crick, F. H. C. (1953). Molecular structure of nucleic acids. *Nature*, 171(4356), 737–738.

Depade

► [Naltrexone](#)

Depakene

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Synonyms

[Divalproex](#); [Valproic acid](#)

Indications

Valproic Acid: Valproic acid is a simple carbonic acid. It is available in several preparations including divalproex and valproic acid. It appears to exert its beneficial effects by interfering with the repetitive firing of neurons. This appears to be especially relevant for its treatment of seizures. Valproic acid is approved for the treatment of seizures, migraine, and for the treatment of bipolar disorder.

Clinical Use (Including Side Effects)

It has been studied in children and adults with bipolar illness and appears to be an effective treatment. Valproic acid is often well tolerated, but it can have a range of adverse effects. Sedation and gastrointestinal disturbance with vomiting are common particularly at the start of treatment. Other more significant adverse effects include thrombocytopenia, pancreatitis, and rarely hepatotoxicity. These more severe adverse effects require monitoring of drug level in the blood, platelet counts, amylase, and liver enzymes.

To date, valproic acid has not been well studied in children or adults with autism spectrum disorders. There are some open case studies suggesting benefit for aggression and agitation; however, these studies have not compared valproic acid to placebo.

See Also

► [Mood Stabilizers](#)

References and Reading

- Kowatch, R. A., Strawn, J. R., & Danielyan, A. (2011). Mood stabilizers: Lithium, anticonvulsants and others. In A. Martin, L. Scahill, & C. Kratochvil (Eds.), *Pediatric psychopharmacology: Principles and practice* (pp. 297–311). New York: Oxford University Press.

Department of Vocational Rehabilitation

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Synonyms

[Employment services](#); [Office of rehabilitation](#); [Vocational counseling](#)

Definition

The Department of Vocational Rehabilitation is a broad marker for an organization that provides services for persons identified with developmental disabilities (Rehabilitation Act of 1973, Public Law 93–112 93rd Congress, H. R. 8070 September 26, 1973). In the act, every state arranges a bureau of vocational rehabilitation services. In NY State, the office is frequently mentioned as VESID (VESID is an acronym for Vocational and Educational Services for Individuals with Disabilities).

In all states, the Department of Vocational Rehabilitation (DVR) delivers occupation services and treatment to those with disabilities who want to work but experience obstacles to work due to physical, sensory, and/or mental disability. A DVR therapist works with every person to develop an individually tailored strategy of services intended to aid them in reaching their employment goal. The aid may contain, but is not limited to, the following:

- Counseling and guidance
- Assessment services
- Independent living services
- Assistive technology services
- Training and education

Vocational and Educational Services for Individuals with Disabilities within the New York State Education Department has accountability criteria for meeting the needs of individuals diagnosed with disabilities from early infancy through old age, plus oversight of special education services for pupils with disabilities aged 3–21. Each year VESID offers thousands of New Yorkers who have a disability a chance to be independent through learning, preparation, and employment. In addition, VESID delivers vocational rehabilitation services to eligible individuals to prepare them for appropriate jobs. These jobs might be in the competitive work force, in private businesses, in supported employment on employer sites, or in sheltered shops. Moreover, VESID aids individuals with disabilities who are having trouble keeping their jobs. Offices of Vocational

Services throughout the country provide similar services and oversight.

Offices of Vocational Rehabilitation, or OVR, delivers vocational rehabilitation services to support persons with disabilities to prepare for, obtain, or maintain employment. The office also offers services to qualified persons diagnosed with disabilities, both directly and through a system of appropriate vendors. Services are provided on a personalized base. The therapist, through face-to-face interviews, helps clientele in choosing their choice of occupational goals, services, and service providers. An Individualized Plan for Employment (IPE) is established, charting a vocational objective, services, providers, and responsibilities. Some services are subject to a Financial Needs Test (FNT) and could involve fiscal contribution by the client. Counseling and guidance, diagnostic services, assessments, information and referral, job development and placement, and personal services such as readers or sign language interpreters are provided at no cost to the individual. Also, by law, OVR clientele awarded Social Security benefits for their disability (SSI, SSDI) are relieved from OVR's Financial Needs Test.

Types of Vocational Rehabilitation Services

The OVR runs a variety of services to qualified applicants. Certain services can aid in overcoming or lessening the disability; others can directly support and prepare for a vocation. The services will be organized to meet distinct needs.

The OVR services include:

Diagnostic services: Medical, psychological, and checkups and assessments used to improve understanding of the disability and needs for specific types of services.

Vocational evaluation: Ability, interest, overall ability, academic exams, work tolerance, and “hands-on” job experience used to understand vocational potential.

Counseling: Occupational therapy will help to better understand potential, to rely on abilities,

to set accurate vocational goals, to modify them once needed, to advance fruitful work ways, and to initiate a fulfilling career. Counseling is obtainable throughout rehabilitation program.

Training: Education to prepare for a job including, but not limited to, basic academic, vocational/technical, college, on-the-job training, independent living skills, and personal and work adjustment training.

Restoration services: Medical services and gear such as physical and occupational therapy, wheelchairs, and automobile hand controls can be provided to achieve employment.

Placement assistance: Counseling, job-seeking programs, job clubs, and job development used to upturn your skill to acquire a job.

Assistive technology: Assistive technology includes a wide range of devices and services that can empower individuals with disabilities to make the most of employment, independence, and integration into society. The office can help person with a disability in successfully choosing and obtaining appropriate assistive technology. They can arrange for an adviser to assess the situation and to make appropriate recommendations. The office also functions and maintains Center for Assistive and Rehabilitation Technology (CART) at the Hiram G. Andrews Center. There is no charge for evaluation and vocational counseling services through OVR.

Support services: Additional services are provided for eligible persons if they are essential to start and uphold occupation. Such services may include:

- Room, board, and transportation costs during an evaluation or while completing a rehabilitation program
- Occupational tools, licenses, or equipment
- Home modifications, adaptive or special household equipment; van or car modifications, including special driving devices or lifting devices
- Personal care assistance
- Job site modifications, independent living training
- Text telephone (TT), signaling devices, hearing aids, and interpreter services

- Specialized services such as rehabilitation teaching and orientation and mobility training for persons who are blind or visually impaired

See Also

- ▶ [Americans with Disabilities Act](#)
- ▶ [Individualized Plan for Employment \(IPE\)](#)
- ▶ [Vocational Evaluator](#)
- ▶ [Vocational Training](#)

References and Reading

- Government Accountability Office. (2005). *Special education: Children with autism*. Washington, DC: United States Government Accountability Office.
- Hillier, A., Campbell, H., Mastriana, K., Izzo, M., Kool-Tucker, A., Cherry, L., et al. (2007). Two-year evaluation of a vocational support program for adults on the autism spectrum. *Career Development for Exceptional Individuals*, 30(1), 35–47.
- Müller, E., Schuler, A., Burton, B., & Yates, G. (2003). Meeting the vocational support needs of individuals with Asperger syndrome and other autism spectrum disorders. *Journal of Vocational Rehabilitation*, 18, 163–175.

Depressive Disorder

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Synonyms

[Depressive disorders](#); [Dysthymia](#); [Major depressive disorder](#); [Mood disorders](#)

Short Description or Definition

- Major depressive disorder
- Dysthymia
- Depressive disorders
- Mood disorders

Categorization

According to the DSM-IV-TR (American Psychiatric Association [APA] 2000), depressive disorders include major depressive disorder, dysthymic disorder, and depressive disorder not otherwise specified. Major depressive disorder includes the presence of a major depressive episode, with specifiers to indicate the severity and duration. Major depressive episodes involve symptoms that are present most of the day, nearly every day consisting of either depressed mood (can be irritability in children) or diminished interest or pleasure in activities, and at least four of the following: significant weight loss or weight gain, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue or loss of energy, feelings of worthlessness or excessive or inappropriate guilt, diminished ability to think or concentrate or indecisiveness, or recurrent thoughts of death, suicidal ideation without a plan, or a suicide attempt or specific plan for committing suicide. The symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning, and they must not be due to the direct effects of a general medical condition, a substance, a medication, or other treatment.

Dysthymic disorder includes the presence of depressed mood for most of the day, for more days than not, as indicated either by subjective account or observation by others, for at least 2 years (in children and adolescents, mood can be irritable, and duration must be at least 1 year) and at least two of the following: poor appetite or overeating, insomnia or hypersomnia, low energy or fatigue, low self-esteem, poor concentration or difficulty making decisions, or feelings of hopelessness.

Depressive disorder not otherwise specified includes disorders with depressive features that do not meet the criteria for major depressive disorder but present with subthreshold symptoms that cause clinically significant impairment or distress.

In the ICD-10 (WHO 1992), *depressive disorder* is defined by the presence of at least one depressive episode with the same key characteristics as in the DSM-IV-TR, such as lowering of

mood, decrease in activity, decrease in capacity for enjoyment, and difficulty in concentration. Somatic symptoms, such as marked tiredness, sleep, and appetite disturbance, are present, and ideas of guilt or worthlessness and suicidal thoughts are often present. Depressive disorders are also categorized by the severity and duration of symptoms, into mild, moderate, severe, recurrent, or with psychotic symptoms. The ICD-10 differs in the classification from DSM-IV-TR by its lack of a specific number of symptoms required to meet criteria.

Dysthymia in the ICD-10 is defined similarly as in the DSM-IV-TR, with chronic depression of mood that is not sufficiently severe or prolonged to justify a diagnosis of severe, moderate, or mild recurrent depressive disorder. However, the criteria do not specify a period of 2 years but rather indicates that the symptoms must last at least several years. *Other depressive disorders* include any other disorders that are not of sufficient severity or duration to meet criteria for depressive disorder or dysthymia.

(Note: Major depressive disorder, dysthymia, depressive disorder not otherwise specified, and other depressive disorders will be referred to collectively as “depressive disorders” or “depression” throughout.)

Epidemiology

The occurrence of depressive disorders in individuals with autism spectrum disorders is likely affected by the same complex genetic and environmental interactions seen in typically developing individuals (Ghaziuddin 2005). However, the impact of these factors is far less understood in ASD due to the lack of systematic epidemiological studies. Family studies suggest a genetic component to the presence of depressive disorders in ASD, with rates of major depression increased in first-degree relatives of individuals with autism and parents of children with ASD exhibiting higher risk for depressive disorders than parents of children with other developmental disabilities (Ghaziuddin and Greden 1998; Lainhart 1999). Families of individuals with ASD who present with depression at clinical settings have a history

of depression or suicide at a rate of 50–77% (Lainhart 1999). Stressful life events, including bereavement, peer victimization, and loneliness, could also contribute to the development of depressive symptoms in individuals with ASD (Ghaziuddin et al. 1995; Ghaziuddin 2005).

Despite the recognition that depressive disorders are relatively common in individuals with autism spectrum disorders, true prevalence rates are unknown because most studies have been conducted with psychiatric samples rather than population samples. However, these studies provide initial estimates of the rate of depressive disorders. Reviews of published research on co-occurring depressive disorders in ASD have reported a wide range of prevalence estimates, from 4% to 58% (Lainhart 1999; Lainhart and Folstein 1994; Stewart et al. 2006). The varied rates of depressive disorders in the literature likely reflect the heterogeneity of the samples and different methods of assessing depressive disorders. For example, the estimates differ depending on whether psychiatric or community samples were used, which ASD subtype was included, the age of participants, and whether psychiatric interviews or questionnaire assessment methods were used. Many researchers posit a higher rate of depressive disorders in ASD than in the general population and suggest that the disorders may be underdiagnosed (e.g., Ghaziuddin 2005; Lainhart and Folstein 1994).

In one of the first descriptions of children and adults with Asperger syndrome in a clinical setting, about one third of the adolescents (16 years and above) and young adults presented with clinical levels of depression (Wing 1981). Other reports describe depression as being the most common psychiatric disorder in Asperger syndrome, with 15% of adults referred to a psychiatric setting presenting with symptoms of depression (Tantum 1991). Despite the findings of several clinical studies and the recognition of the risk for developing depressive disorders in ASD, no population-based prevalence studies with adolescents and adults appear to have been completed.

There have been some promising studies examining the prevalence of depressive

symptoms in children with ASD, with larger, more representative samples. For example, a prevalence study of mood and anxiety symptoms among 9- to 13-year-old children with Asperger syndrome and high-functioning autism included a community standardization sample of 1,751 typically developing children (Kim et al. 2000). On a questionnaire of depression symptoms, 16.9% of the ASD sample scored at least two standard deviations above the population mean, suggesting a significantly higher rate of depressive symptoms than in the community sample (Kim et al. 2000).

An Australian study examined emotional and behavioral problems in 4- to 18-year-old children and adolescents diagnosed with ASD and with youngsters diagnosed with intellectual disability (learning disability) and no diagnosis of ASD (Brereton et al. 2006). The ASD group scored significantly higher on a measure of depression than the non-ASD group. Age and IQ in the ASD group also affected depression scores. Older children (13 years or older) scored significantly higher on the depression measure than the youngest age group (less than 6 years old), and those with higher IQs scored higher than individuals with intellectual disability (learning disability).

Standardized interview methods of assessment have been used infrequently in research on depression in ASD. Only one study was found to examine the prevalence rates of psychiatric disorders in a community-derived sample using a standardized interview measure (Simonoff et al. 2008). In this sample of children aged 10–14 years, a surprisingly low rate of depressive disorders (1.4%) was found.

When lifetime occurrence of depressive symptoms and a wider age range was included, a higher rate of depressive disorders was found. In a pilot study for the development of an ASD-specific psychiatric comorbidity interview conducted with 5- to 17-year-olds, 10% of a community sample with higher-functioning ASD met criteria for at least one major depressive disorder in their lifetime, and 25% met criteria for subsyndromal symptoms of depressive disorders (Leyfer et al. 2006).

The prevalence of depressive disorders in the general population varies by age with more

adolescents and adults presenting with depression than children (World Health Organization 2002). Research on depressive disorders in ASD suggests a similar pattern, with more adolescents and adults with ASD presenting with depression than children (e.g., Brereton et al. 2006; Martin et al. 2000; Simonoff et al. 2008). It is currently unknown if more women with ASD present with depression than men, as is the case in the general population (Ghaziuddin 2005).

Natural History, Prognostic Factors, and Outcomes

The presence of depressive symptoms in individuals with autism spectrum disorders was noted in the earliest descriptions of the disorders (Asperger 1944; Kanner 1943; Wing 1981). However, due to the lack of systematic population studies, the course of depressive disorders is not well understood.

Numerous publications have noted that the development of depressive disorders in adolescents and adults with Asperger syndrome and high-functioning autism in particular seems to be related to a developing awareness of “differentness” from their peers and unsuccessful attempts to establish friendships and romantic relationships (e.g., Ghaziuddin 2005; Howlin 1997; Wing 1981). The presence of depressive symptoms in children with Asperger syndrome and high-functioning autism has been found to be associated with higher rates of aggressive and oppositional behavior, along with poorer relationships with teachers, peers, and family members when compared to children with ASD without depressive symptoms (Kim et al. 2000).

In general, the outcomes for adults with autism spectrum disorders, with and without intellectual disability (learning disability), have not been promising, with decreased opportunities for employment, independent living, and access to community services (Howlin 2005). There is little information on the long-term outcome of persons with ASD and depressive disorders. However, clinicians report that the presence of co-occurring depressive disorders can result in

further impairment and disruption in functioning, such as increased morbidity and mortality, and a higher potential for drug interactions due to multiple pharmacotherapy treatment (Ghaziuddin 2005). Depressive illness can become chronic in some individuals, and a family history of mood disorders seems to be associated with a poorer treatment outcome (Ghaziuddin 2005).

Clinical Expression and Pathophysiology

The presentation of depressive symptoms in ASD shares many of the features seen in the general population, such as sadness and lack of interest in formerly pleasurable activities, but individuals with ASD may also present with unique features, due to their restricted range of emotional expression and difficulty in communication. While sad mood and loss of pleasure in activities are defining characteristics of depressive disorders, individuals with ASD are often referred to clinical settings because of changes observed by others, such as facial expressions of sadness or misery, or behavioral expressions, such as increased frequency of crying, irritability, or problem behavior (Stewart et al. 2006). Particular features that must be assessed carefully in individuals with ASD include an increase in social withdrawal, changes in the character of stereotypic and repetitive behavior, and restricted interests, irritability, and regression of skills (Ghaziuddin 2005).

Other factors that are likely to affect the presentation of depressive disorders in ASD include age, gender, cognitive and verbal ability, other psychiatric disorders, and other medical disorders. Younger children may be more likely to present with irritability than with sad or depressed mood, and this is recognized in the DSM-IV-TR criteria, which allows for substitution of irritability for depressed mood in children (APA 2000). Research suggests an increase in depressive symptoms with age (e.g., Brereton et al. 2006). The risk of depression and other psychiatric disorders may be higher in individuals with Asperger syndrome and higher-functioning individuals with autism because their relatively good cognitive and language skills may lead others to

overestimate their abilities and put more pressure on them to “fit in” with peers, while overlooking the severe difficulty they have in understanding social interaction. However, there is not enough evidence at this time to make any conclusions about differential risks of developing depression in these groups (Howlin 2005).

Individuals with Asperger syndrome and higher-functioning autism may present with depressive symptoms differently than individuals with autistic disorder. They may be able to verbally describe feelings of sadness and loneliness, while individuals with more cognitive impairments may not be able to express themselves verbally and may present with more behavioral signs, such as irritability, aggression, and changes in sleep and appetite. However, it is important to recognize that individuals with higher-functioning presentations of ASD and intact language abilities may not be able to accurately describe their emotions and may present with atypical signs and symptoms of depressive disorders, such as irritability or bizarre ideation (Howlin 2005).

The presence of other psychiatric disorders can also affect the presentation of depressive symptoms. The co-occurrence of mood and anxiety disorders is common in the general population, and research suggests that these disorders often co-occur in people with ASD (e.g., Lainhart 1999). The presence of symptoms associated with anxiety, such as increased stereotypic behaviors, may make it more difficult to assess depressive disorders in individuals with ASD.

Evaluation and Differential Diagnosis

The classification of psychiatric disorders in ASD has involved considerable controversy. Many early researchers adopted a hierarchical approach to diagnosis and argued that psychiatric disorders could not occur in individuals with intellectual disability (learning disability) or autism spectrum disorders. A hierarchical approach conceptualizes symptoms that overlap with other disorders as part of the primary disorder, with little room for the diagnosis of co-occurring psychiatric disorders.

An alternative diagnostic approach to the hierarchical approach classifies all symptom constellations that meet criteria for a particular disorder and allows for identification of multiple disorders (Simonoff et al. 2008). Despite the controversy in the literature, many now agree that the full spectrum of psychiatric disorders can co-occur in ASDs (Ghaziuddin 2005; Matson and Nebel-Schwalm 2007; Simonoff et al. 2008).

Despite the recognition that depressive disorders can and do occur in ASD, diagnosing them in individuals with autism spectrum disorders can be particularly difficult due to a variety of factors, including an overlap between symptoms of depressive disorders and features of ASD, such as poor eye contact, restricted affect, and lack of voice inflection. For example, it may be difficult to determine whether the social withdrawal observed in an individual with autism is part of the core social deficits of autism spectrum disorders or is symptomatic of a co-occurring mood disturbance.

An important factor in making an accurate diagnosis of depressive disorders in ASD is having reliable information from multiple sources. If the individual with ASD is able to provide information about symptoms, it is important to assess these carefully. However, given the difficulties that individuals with ASD have in expressing and understanding emotions, it is also important to obtain information from caregivers, teachers, and family members about typical patterns of behavior. Reports by others may also be helpful to interpret self-report of individuals with ASD. The clinician needs to obtain a detailed picture of the individual's baseline levels of social activity, interests, restricted and repetitive behavior, maladaptive behavior, and adaptive skills, in order to detect distinct differences in these areas that may indicate the onset of mood disturbance. Information obtained from parental report concerning developmental and social history, including the presence of significant life events, and the results from prior assessments, such as medical and psychological evaluations, intelligence, and adaptive behavior testing, can complete the diagnostic picture. A detailed physical examination is recommended to rule out other possible causes

of depressive symptoms such as thyroid disorders (Ghaziuddin 2005).

Intellectual disability (learning disability) commonly co-occurs in ASD, with estimates of 30–70% of individuals with ASD functioning in the ID (LD) range (Fombonne 2005). The presence of ID (LD) in this population has important implications for how depression is assessed in this heterogeneous group. The diagnostic manual for intellectual disability (DM-ID) proposed adaptations to the DSM diagnostic criteria for persons with intellectual disabilities based on clinical consensus (Fletcher et al. 2007). The DM-ID includes irritable mood as an acceptable substitute for depressed mood for people with ID which the DSM-IV-TR includes in the criteria for children (APA 2000). The DM-ID reduces the number of symptoms by one for diagnosing major depressive disorder, requiring four symptoms instead of five if the individual has limited expressive language skills. The other important adaptation is that the DM-ID allows observer report for many symptoms (Charlot et al. 2007). This practice is compatible with clinical reports concerning individuals with ASD in which most cases of depression are brought to clinical attention by observations from caregivers rather than by self-report of the individual.

While the alternative diagnostic criteria put forth in the DM-ID represent an important step in the understanding of co-occurring psychiatric disorders in individuals with all types of developmental disabilities, there remains a lack of ASD-specific psychopathology assessment methods. Many studies on depression in ASD have relied on scales or structured interviews designed for the general population or for individuals with intellectual disability. Consequently, it is difficult to determine if these measures are sensitive to the characteristic features of ASD.

A semistructured psychiatric interview was developed to assess psychiatric disorders in children and adolescents with ASD (Leyfer et al. 2006). The Autism Comorbidity Interview – Present and Lifetime Version (ACI-PL) was modified from the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS, Chambers et al. 1985). This measure aims to distinguish the core

symptoms of ASD from symptoms of comorbid psychiatric disorders. It demonstrated good reliability and validity, although the validation sample was limited to individuals with higher-functioning ASD.

The further development of ASD-specific screening measures and structured diagnostic interviews is important to improve the accurate identification of depressive disorders in ASD and to gain access to specific, effective treatment.

Treatment

The treatment of depressive disorders in individuals with ASDs is largely pharmacological in nature, with antidepressant medication being prescribed most often and selective serotonin reuptake inhibitors (SSRIs), such as fluoxetine, sertraline, and fluvoxamine, showing the greatest success in symptom reduction (Lainhart 1999; Stewart et al. 2006). It is difficult to determine if psychopharmacological treatments are being used in this population specifically to treat depressive disorders because a large percentage (30.5%) of individuals with ASD take one or more psychotropic medications (Aman et al. 1995). Antidepressants may be prescribed for repetitive or compulsive behavior as well as for depressive symptoms. A naturalistic study, which examined psychotropic drug use among a sample of 109 individuals with high-functioning ASD, reported that about a third of the participants were prescribed an antidepressant with about one fourth taking an SSRI (Martin et al. 2000). Depression was identified as the reason for taking psychotropic medication in about 30% of participants. The response to antidepressants by patients with ASD is reported to be similar to the general population (Ghaziuddin 2005). Side effects, if they are problematic, tend to be related to other medical issues such as seizures.

There is increasing recognition that cognitive behavior therapy may be an effective treatment for psychiatric disorders in individuals with Asperger syndrome (Anderson and Morris 2006; Attwood 2003). Cognitive behavior therapy for depression has been used with individuals with Asperger

syndrome with resulting improvement (Hare 1997). Depending on the individual's level of functioning and communication abilities, CBT may be an appropriate treatment choice. Further study is needed to determine the effectiveness of CBT with individuals with ASD and to identify the active components of the treatment.

Social skills training, environmental modifications, and behavioral interventions may have a role in addressing depressive symptoms in individuals with ASD. Psychosocial interventions are often used in conjunction with medications to treat depressive disorders.

See Also

- ▶ Affective Disorders (Includes Mood and Anxiety Disorders)
- ▶ Antidepressant Medications
- ▶ Anxiety Disorders
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Mood Disorders
- ▶ Serotonin Reuptake Inhibitors (SRIs)

References and Reading

- Aman, M. G., Van Bourgondien, M. E., Wolford, P. L., & Saphare, G. (1995). Psychotropic and anticonvulsant drugs in subjects with autism: Prevalence and patterns of use. *Journal of the American Academy of Child and Adolescent Psychology*, 34, 1672–1681.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.). Washington, DC: Author.
- Anderson, S., & Morris, J. (2006). Cognitive behaviour therapy for people with Asperger syndrome. *Behavioural and Cognitive Psychotherapy*, 34, 293–303.
- Asperger, H. (1944). Die "Autistischen Psychopathen" im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Attwood, T. (2003). Cognitive behaviour therapy (CBT). In L. H. Willey (Ed.), *Asperger syndrome in adolescence: Living with the ups, the downs and things in between* (pp. 38–68). London: Jessica Kingsley.
- Brereton, A. V., Tonge, B. J., & Einfeld, S. L. (2006). Psychopathology in children and adolescents with autism compared to young people with intellectual disability. *Journal of Autism and Developmental Disorders*, 36, 863–870.
- Chambers, W. J., Puig-Antich, J., Hirsch, M., Paez, P., Ambrosini, P. J., Tabrizi, M. A., et al. (1985). The assessment of affective disorders in children and adolescents by semistructured interview. Test-retest reliability of the schedule for affective disorders and schizophrenia for school-age children, present episode version. *Archives of General Psychiatry*, 42, 696–702.
- Charlot, L., Fox, S., Silka, V. R., Hurley, A., Lowry, M., & Pary, R. (2007). Mood disorders. In R. Fletcher, E. Loschen, C. Stavrakaki, & M. First (Eds.), *Diagnostic manual – Intellectual disability: A clinical guide for diagnosis of mental disorders in persons with intellectual disability* (pp. 157–186). Kingston: NADD Press.
- Fletcher, R., Loschen, E., Stavrakaki, C., & First, M. (Eds.). (2007). *Diagnostic manual – Intellectual disability (DM-ID): A clinical guide for diagnosis of mental disorders in persons with intellectual disability*. Kingston: NADD Press.
- Fombonne, E. (2005). Epidemiology of autistic disorder and other pervasive developmental disorders. *Journal of Clinical Psychiatry*, 66, 3–8.
- Ghaziuddin, M. (2005). *Mental health aspects of autism and Asperger syndrome*. London: Jessica Kingsley.
- Ghaziuddin, M., Alessi, N., & Greden, J. F. (1995). Life events and depression in children with pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 36, 495–502.
- Ghaziuddin, M., & Greden, J. (1998). Depression in children with autism/pervasive developmental disorders: A case-control family history study. *Journal of Autism and Developmental Disorders*, 28, 111–115.
- Hare, D. J. (1997). The use of cognitive-behavioural therapy with people with Asperger syndrome: A case study. *Autism*, 1, 215–225.
- Howlin, P. (1997). *Autism: Preparing for adulthood*. London: Routledge.
- Howlin, P. (2005). Outcomes in autism spectrum disorders. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. I, pp. 640–649). Hoboken: Wiley.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kim, J. A., Szatmari, P., Bryson, S. E., Streiner, D. L., & Wilson, F. J. (2000). The prevalence of anxiety and mood problems among children with autism and Asperger syndrome. *Autism*, 4, 117–132.
- Lainhart, J. E. (1999). Psychiatric problems in individuals with autism, their parents and siblings. *International Review of Psychiatry*, 11, 278–298.
- Lainhart, J. E., & Folstein, S. E. (1994). Affective disorders in people with autism: A review of published cases. *Journal of Autism and Developmental Disorders*, 24, 587–601.
- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., et al. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36, 849–861.

- Martin, A., Patzer, D. K., & Volkmar, F. R. (2000). Psychopharmacological treatment of higher-functioning pervasive developmental disorders. In A. Klin, F. R. Volkmar, & S. S. Sparrow (Eds.), *Asperger syndrome* (pp. 210–228). New York: The Guilford Press.
- Matson, J. L., & Nebel-Schwalm, M. S. (2007). Comorbid psychopathology with autism spectrum disorder in children: An overview. *Research in Developmental Disabilities*, 28, 341–352.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47, 921–929.
- Stewart, M. E., Barnard, L., Pearson, J., Hasan, R., & O'Brien, G. (2006). Presentation of depression in autism and Asperger syndrome: A review. *Autism*, 10, 103–116.
- Tantum, D. (1991). Asperger syndrome in adulthood. In U. Frith (Ed.), *Autism and Asperger syndrome* (pp. 147–183). Cambridge: Cambridge University Press.
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11, 115–129.
- World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: Author.
- World Health Organization. (2002). The World health report 2001 – Mental health: New understanding, new hope. Retrieved from <http://www.who.int/whr/2002/en/> on 2 Nov 2011.

Depressive Disorders

- [Depressive Disorder](#)

Derailment

- [Flight of Ideas](#)

Dermamycin® [OTC]

- [Diphenhydramine](#)

Dermatoglyphic Patterns

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Definition

Dermatoglyphics refers to the study of fingerprints and handprints. Dermatoglyphic patterns are the unique ridges and whorls of the skin of the fingertips and palms. Each individual person (even genetically identical twins) has a slightly different pattern of ridges and whorls on the fingers, especially the tips and palms. There can be unique patterns of the lines that cross the palms and fingers as well. The creases of the palms may be altered in specific genetic syndromes. The dermatoglyphic pattern of the fingerprint is determined prenatally. At this point, the literature on dermatoglyphic patterns in children with ASD is conflicting. The evidence does not indicate that there are differences in the ridges/whorls or palm prints of children with autism spectrum disorders, although there may be differences related to associated or underlying genetic disorders.

See Also

- [Down Syndrome](#)
- [Genetics](#)
- [Physical and Neurological Examination](#)

References and Reading

- Walker, H. A. (1979). A dermatoglyphic study of autistic children. *Journal of Autism and Developmental Disorders*, 7, 11–21.
- Zimmerman, A. W. (2008). *Autism: Current theories and evidence* (p. 193). Boston: Springer.

Descriptive Research

► Qualitative Versus Quantitative Approaches

Desensitization

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Definition

Systematic desensitization refers to the therapeutic technique of gradually exposing an individual to increasingly difficult anxiety-producing conditions in an effort to help an individual reduce anxious responses in those situations by adopting more adaptive ways of coping. The closely related process of counterconditioning consists of coupling opposing, positive responses (e.g., relaxation) to situations known to cause fear or anxiety. By substituting a new, adaptive response to fearful settings, the individual learns to elicit a more appropriate reaction in fearful situations. The development of a hierarchy of fearful conditions (i.e., consists of identifying a range of situations from least to most anxiety provoking) plays a key role in this intervention by serving as a framework for gradual exposure. Anxiety-related situations are introduced in a step-by-step process beginning with the least challenging and progressing to the more difficult. This technique has been used effectively with children and adults with autism.

Historical Background

Systematic desensitization originates from classical conditioning theory. In 1920, John B. Watson demonstrated that fearful responses could be conditioned. Watson conditioned a fear of rats in an infant, referred to as “Little Albert,” by pairing the presence of a white rat with a sudden loud noise (Watson and Rayner 1920). Eventually the presence of the rat by itself elicited a fear response that generalized to all furry objects. Watson postulated that emotional responses could be learned and modified to generalize to a broader category of stimuli. The seminal work by Watson laid forth evidence for the development of phobias.

In response, Mary Carver Jones conducted instrumental research examining the mechanisms in which fearful responses could be reduced and generalization of these new responses achieved (Jones 1924). She treated a young boy, “Peter,” whom she selected partially because of characteristics (i.e., fear of white rats and other furry animals and objects) he shared with “Little Albert.” In the experiment, Peter was gradually presented with increasingly difficult fearful situations while he was simultaneously engaged in a pleasant activity. Initially the rabbit was presented a considerable distance away, and over a period of multiple sessions, it was gradually moved closer to Peter. During each of these sessions, Peter received his favorite food that was presented to him by a friendly peer or adult in the presence of the rabbit (Lang 1966). Peter initially demonstrated fearful responses to the rabbit but gradually exhibited that he was comfortable by holding the rabbit on his lap and he ultimately showed affection towards it. Through the process of gradual exposure, coupled with positive reinforcement (both through tangible rewards and social praise), Jones effectively demonstrated mechanisms through which fear could be reduced.

Building on the findings of Mary Carver Jones, Joseph Wolpe developed and operationalized the method of systematic desensitization to treat clients with anxiety and phobias, especially those connected to situations in which no real danger was present (Wolpe 1958). He based his method on the principle of reciprocal inhibition, which

emphasized that opposite responses to anxiety, such as relaxation, could be utilized to diminish anxiety. Though Wolpe acknowledged that other responses also compete with anxiety (Wolpe 1990a), he incorporated relaxation as the “reciprocally inhibiting” response in his treatment through gradual exposure. Wolpe’s seminal research on systematic desensitization provided a theoretical explanation for the success of gradual approaches through which the therapist determines the degree to which a patient is exposed to anxiety-provoking stimuli (Wolpe 1961). He also developed the method of imagery construction within systematic desensitization (having the client imagine fearful situations) to gradually expose clients to anxiety-provoking scenarios without confronting them directly to begin with. Systematic desensitization was reportedly successful for treating anxiety of nonpersonal stimuli (enclosed spaces, harmless animals, etc.) as well as interpersonal stimuli (fears of specific people or fear of being criticized). Also, Wolpe’s method treated fears that could not be brought into a therapist’s office such as complex social situations in a community setting (Lang 1966). Overall, the method adequately treated abstract fears and anxieties, which separated systematic desensitization from other treatments available at the time.

While Wolpe originally incorporated imagined exposures during treatment, many clinicians now use *in vivo* exposures when implementing systematic desensitization. These *in vivo* exposures involve direct exposure to the actual fear-inducing stimuli. Both imagined exposure and *in vivo* exposure produce diminished anxiety levels in patients. With children, *in vivo* exposure tends to be used more.

Rationale or Underlying Theory

Wolpe hypothesized that the underlying process at work during systematic desensitization is reciprocal inhibition. If a client can be taught to elicit a response oppositional to anxiety (e.g., relaxation) while in the presence of anxiety-evoking stimuli, then the anxiety response weakens eventually leading to diminished or no anxiety (Wolpe

1958). Pairing such a response with systematic presentations of increasingly anxiety-provoking stimuli, fear responses gradually diminish even in response to the most fearful situations.

The theory of habituation provides another explanation for the changes that occur during systematic desensitization (Antony and Stein 2008; Watts 1971). The longer and more often a client is exposed to anxiety-producing stimulus, the less effect that stimulus is presumed to have. When a person is exposed to anxiety-producing stimuli without negative consequences (e.g., petting a dog without getting bitten), negative thoughts are challenged and new, more adaptive information is encoded (Antony and Stein 2008). As a client spends more time exposed to these situations free from negative consequences, the person becomes “habituated” to the stimuli and previously distressing situations no longer cause unmanageable levels of anxiety.

Modern theorists also incorporate cognitive approaches to explain the success of systematic desensitization in treating anxiety symptoms. A retrieval competition theory posits that when clients are exposed to feared stimuli with no negative consequences, they begin to form different mental representations of these situations. Positive aspects of the exposure such as free choice, relative safety, and perceived self-efficacy all contribute to the formation of new mental representations (Brewin 2006). As exposures are repeatedly completed, these more positive representations are primed for retrieval and activated more quickly than the original negative representations.

Goals and Objectives

The overall goal of systematic desensitization is to reduce anxiety in feared situations. The method aims at addressing fears and anxieties through gradual exposures. Systematic desensitization can be used to treat a range of anxiety symptoms, particularly phobias, from the specific (e.g., fear of snakes) to the abstract (e.g., fear of embarrassment). In this way, phobias such as fear of crowds or fear of public speaking can be remediated in behavioral therapy. While some models of the

method utilize only imagined exposure and others incorporate exposure in naturalistic contexts, the objective is to gradually weaken anxious responses to anxiety-evoking stimuli and to minimize the probability of fears returning after being successfully treated (Brewin 2006). Modern research suggests that the “return of fear” phenomenon is most likely to occur when exposure therapy is conducted only in limited contexts (e.g., clinics), rather than also in the naturalistic settings where fear is experienced in everyday life (Mineka et al. 1999; Mystkowski et al. 2003).

Treatment Participants

Systematic desensitization is appropriate to treat phobias or anxiety symptoms in both children and adults. Given that phobias and anxiety present themselves in a range of populations, the process has been effective in treating a variety of conditions (Brooks et al. 2007; Davis et al. 2011; Frank et al. 1988; Morrow 1986). Research indicates particular effectiveness in treating specific phobias (Gelder et al. 1967), such as with snakes (Lang and Lazovik 1963) or claustrophobia (Wolpe 1961). Systematic desensitization has also proven effective to treat fears and anxieties in persons with cognitive limitations (Erfanian and Miltenberger 1990) and autism spectrum disorders (ASD) (Jackson and King 1982; Koegel et al. 2004; Luiselli 1978; Luscre and Center 1996; Wood et al. 2009).

Systematic Desensitization and Autism Spectrum Disorders

Individuals with ASD often experience symptoms of fear and anxiety (Gillot et al. 2001; White et al. 2009). Many systematic desensitization interventions among children with autism involve in vivo exposures within the actual setting (Koegel et al. 2004; Luiselli 1978; Luscre and Center 1996; Wood et al. 2009). Luiselli (1978) used in vivo systematic desensitization to successfully treat a child with autism who feared riding the school bus. Additionally, systematic desensitization significantly reduced fear of dental visits in children with autism (Luscre and Center 1996), and in

another study, it was used to successfully eliminate fearful behaviors to common auditory stimuli (e.g., sounds from a vacuum, flushing toilets) in three children with autism (Koegel et al. 2004).

Wood et al. (2009) incorporated systematic desensitization as a component of their cognitive behavioral therapy. Children with autism moved through individually designed fear hierarchies in which they were presented with in vivo exposures and received a variety of reinforcers for their participation. Immediately before and immediately following each exposure, a therapist guided the client through conversations aimed at restructuring negative thoughts with more adaptive cognition. Case studies of this treatment are given in Sze and Wood (2007, 2008), with anxiety targets ranging from intrusive thoughts (worries and obsessions) to separation anxiety, social avoidance at school, and compulsive behaviors.

These studies suggest that systematic desensitization is effective in treating anxious responses in children with ASD.

Treatment Procedures

Joseph Wolpe’s original treatment paradigm remains at the foundation of systematic desensitization while some elements have been modified. For example, current systematic desensitization procedures now employ more in vivo exposure rather than imagined exposure when possible. In fact, Wolpe himself utilized in vivo exposures when imagined exposure did not effectively treat a patient’s symptoms (Wolpe 1990a). Similarly, though relaxation is used as a means of reciprocal inhibition (Graziano and Kean 1968), other methods have been used as well such as positive reinforcement (Koegel et al. 2004; Luiselli 1978; Wood et al. 2009) and laughter (Jackson and King 1982). All systematic desensitization treatments continue to function on the principles of reciprocal inhibition and/or habituation.

Before implementing an intervention using systematic desensitization, a hierarchy of fears is established. This hierarchy refers to a list of various situations ordered from mildly to severely anxiety provoking (e.g., Wood and McLeod

2008). For example, a mild anxiety-inducing situation in a patient with a fear of snakes would be imagining a snake lying on the ground, while a severe anxiety-provoking situation would be holding a small, defanged, nonpoisonous snake. The hierarchy delineates the full range of conditions that produce fear. The items on the hierarchy can be conceptualized as steps or levels that range from a continuum from least to most anxiety provoking. It is developed before implementing treatment because it serves as a guideline for treatment goals (Lang 1966). Treatment proceeds in a step-wise progression beginning with mild anxiety-producing situations and gradually addresses more severe anxiety-producing scenarios on the hierarchy. The patient should exhibit and report diminished levels of anxiety before moving to the next step in the hierarchy. Employing a hierarchy during treatment has shown to increase the effects of systematic desensitization (Morrow 1986) and has been a constant component of systematic desensitization since Wolpe first described the treatment.

Incorporating relaxation into systematic desensitization treatment produces substantial effects (Wolpe 1958). The presence of a relaxation response inhibits anxiety or fear arousal because it is inherently antagonistic to both (Lomont and Edwards 1966). However, some research indicates that relaxation training is an unnecessary component to treatment (Agras et al. 1971). While the degree of focus on relaxation may vary, many systematic desensitization treatments continue to include it as a component. Given that it requires intensive focus, relaxation training in children may be challenging (King et al. 1990), especially in children with mental retardation or low-functioning autism (Graziano and Kean 1968). To circumvent these challenges, immediate positive reinforcement (praise, edibles, toys, etc.) may substitute as an adequate means of attaining reciprocal inhibition and has been successful in treating anxiety in children with ASD (e.g., Wood et al. 2009). This type of treatment paradigm often occurs when employing in vivo exposures rather than imagined exposures. Luiselli (1978) used verbal praise and edible treats for each successive step a child completed in riding the school bus. In

a study of hypersensitivity to auditory stimuli in children with autism (Koegel et al. 2004), children's favorite snacks and verbal praise were used to reinforce the children during in vivo exposures. Similarly, Luscre and Center (1996) employed individualized rewards (i.e., music, Play-Doh, fruit, etc.) during and after exposures. Jackson and King (1982) found that laughter could be used as an effective means of inhibiting anxiety in a child with autism suffering from a phobia of the sound from flushing toilets. These studies indicate that the effectiveness of systematic desensitization in children with autism may be enhanced when positive reinforcement or pleasant experiences are coupled with anxiety-provoking situations.

Efficacy Information

The efficacy of systematic desensitization is well documented (Chambless et al. 1998; Choy et al. 2007; Wood et al. 2009). Since the treatment requires individualization to the patient, there are numerous single-case studies published (e.g., MacDonald 1975; Sze and Wood 2007, 2008). However, studies have also emphasized the effectiveness of systematic desensitization in larger sample sizes (e.g., Frank et al. 1988; Wood et al. 2009). The degree of efficacy varies and is associated with the type of phobia, the severity of symptoms, the length of intervention, as well as individual attributes (e.g., treatment motivation) that is evidenced across typically developing populations (Wolpe 1990b) and children with ASD (Koegel et al. 2004; Sze and Wood 2007, 2008). Despite these variations, it seems that systematic desensitization is generally an effective treatment method in treating anxious responses in ASD.

Outcome Measurement

Abstract concepts such as "fear" and "anxiety" can be difficult to measure. As a result, outcome measurement varies across studies. One common method is to measure outcome by the number of

steps completed in a fear hierarchy. Many single-case methodological studies utilize this approach as a means of determining the success of treatment in ASD (Koegel et al. 2004; Luscre and Center 1996). Additionally, the occurrence of anxious behavioral responses (e.g., crying, running away) has been measured using frequency counts or calculated as percentages of occurrence within specific timed intervals using direct observation or review of videotaped sessions (Koegel et al. 2004).

Other measures include parent or patient report of anxiety symptoms (Kendall 1994; Wood et al. 2009). These self-report measures are subjective ratings (King et al. 1990) and are often used in conjunction with other measures. In order to limit subjectivity, reports may be recorded using standardized anxiety scales such as subjective units of distress scale, which is a 0–100-point scale usually administered with adults to assess their subjective anxiety levels (Choy et al. 2007). When children are the subjects of treatment, measures of parent report are often obtained.

Since changes in physical (somatic) responses such as increased respiration, cardiac rate, blood pressure, or galvanic skin response (GSR) serve as indicators of fear or anxiety, physiological measures may also be recorded as a means of indicating anxiety (Fisher et al. 2009). All of these outcome measurements are intended to determine the remittance of anxiety behaviors.

Qualifications of Treatment Providers

Trained clinicians familiar with behavioral principles and the treatment population have implemented systematic desensitization. Understanding the rationale of the treatment likely increases the effectiveness of how the treatment is delivered. While therapists typically deliver the treatment, there are resources available for parents, teachers, and even patients themselves to learn to implement the treatment (Merrell 2001); however, the efficacy of such implementation has yet to be determined.

See Also

- Cognitive Behavioral Therapy (CBT)
- Phobia

References and Reading

- Agras, W. S. (1967). Transfer during systematic desensitization therapy. *Behavior Research and Therapy*, 5, 193–199.
- Agras, W. S., Leitenberg, H., Barlow, D. H., Curtis, N. A., Edwards, J., & Wright, D. (1971). Relaxation in systematic desensitization. *Archives of General Psychiatry*, 25(6), 511–514.
- Antony, M. M., & Stein, M. B. (2008). *Oxford handbook of anxiety and related disorders*. Oxford: Oxford University Press.
- Brewin, C. R. (2006). Understanding cognitive behaviour therapy: A retrieval competition account. *Behaviour Research and Therapy*, 44, 765–784.
- Brooks, C. J., Gibbs, P. N., Jenkins, J. L., & McLeod, S. (2007). Desensitizing a pilot with a phobic response to required helicopter underwater escape training. *Aviation, Space, and Environmental Medicine*, 78(6), 618–623.
- Chambless, D. L., Baker, M. J., Baucom, D. H., Beutler, L. E., Calhoun, K. S., Crits-Christoph, P., et al. (1998). Update on empirically validated therapies, II. *Clinical Psychologist*, 51(1), 3–16.
- Choy, Y., Fyer, A. J., & Lipsitz, J. D. (2007). Treatment of specific phobia in adults. *Clinical Psychology Review*, 27, 266–286.
- Davis, T. E., May, A., & Whiting, S. E. (2011). Evidence-based treatment of anxiety and phobia in children and adolescents: Current status and effects on the emotional response. *Clinical Psychology Review*, 31(4), 592–602.
- Erfanian, N., & Miltenberger, R. G. (1990). Brief report: Contact desensitization in the treatment of dog phobias in persons who have mental retardation. *Behavioral Residential Treatment*, 5(1), 55–60.
- Fisher, A. J., Granger, D. A., & Newman, M. G. (2009). Sympathetic arousal moderates self-reported physiological arousal symptoms at baseline and physiological flexibility in response to a stressor in generalized anxiety disorder. *Biological Psychology*, 83(3), 191–200.
- Frank, E., Anderson, B., Stewart, B. D., Dancu, C., & Hughes, C. (1988). Efficacy of cognitive behavior therapy and systematic desensitization in the treatment of rape trauma. *Behavior Therapy*, 19(3), 403–420.
- Gelder, M. G., Marks, I. M., & Wolff, H. H. (1967). Desensitization and psychotherapy in the treatment of phobic states: A controlled inquiry. *The British Journal of Psychiatry*, 113, 53–73.

- Gillot, A., Furniss, F., & Walter, A. (2001). Anxiety in high-functioning children with autism. *Autism, 5*, 277–286.
- Graziano, A. M., & Kean, J. E. (1968). Programmed relaxation and reciprocal inhibition with psychotic children. *Behavior Research and Therapy, 6*, 433–437.
- Hedberg, A. G., & Campbell, L. (1974). A comparison of four behavioral treatments of alcoholism. *Journal of Behavior Therapy and Experimental Psychiatry, 5* (3–4), 251–256.
- Jackson, H. J. E., & King, N. J. (1982). The therapeutic management of an autistic child's phobia using laughter as the anxiety inhibitor. *Behavioural Psychotherapy, 1*, 364–369.
- Jones, M. C. (1924). A laboratory study of fear: The case of Peter. *Pedagogical Seminary, 31*, 308–315.
- Kendall, P. C. (1994). Treating anxiety disorders in children: Results of a randomized clinical trial. *Journal of Consulting and Clinical Psychology, 62*, 100–110.
- King, N. J., Ollendick, T. H., Gullone, E., Cummins, R. A., & Josephs, A. (1990). Fears and phobias in children and adolescents with intellectual disabilities: Assessment and intervention strategies. *Australia and New Zealand Journal of Developmental Disabilities, 16*(2), 97–108.
- Koegel, R. L., Openden, D., & Koegel, L. K. (2004). A systematic desensitization paradigm to treat hypersensitivity to auditory stimuli in children with autism in family contexts. *Research & Practice for Persons with Severe Disabilities, 29*(2), 122–134.
- Lang, P. J. (1964). Experimental studies of desensitization psychotherapy. In J. Wolpe, A. Salter, & L. J. Reyna (Eds.), *The conditioning therapies* (pp. 38–53). New York: Holt, Rinehart, & Winston.
- Lang, P. J. (1966). Experimental studies of fear reduction. *Journal of Dental Research, 45*, 1618–1619.
- Lang, P. J., & Lazovik, A. D. (1963). Experimental desensitization of a phobia. *Journal of Abnormal and Social Psychology, 66*, 519–525.
- Lomont, J. G., & Edwards, J. E. (1966). The role of relaxation in systematic desensitization. *Behavior Research and Therapy, 5*, 11–25.
- Luiselli, J. K. (1978). Treatment of an autistic child's fear of riding a school bus through exposure and reinforcement. *Journal of Behavior Therapy and Experimental Psychiatry, 9*, 169–172.
- Luscre, D. M., & Center, D. B. (1996). Procedures for reducing dental fear in children with autism. *Journal of Autism and Developmental Disorders, 26*(5), 547–556.
- MacDonald, M. L. (1975). Multiple impact behavior therapy in a child's dog phobia. *Journal of Behavior Therapy and Experimental Psychiatry, 6*, 317–322.
- Merrell, K. W. (2001). *Helping students overcome depression and anxiety: A practical guide*. New York: Guilford.
- Mineka, S., Mystkowski, J. L., Hladek, D., & Rodriguez, B. I. (1999). The effects of changing contexts on return of fear following exposure therapy for spider fear. *Journal of Consulting and Clinical Psychology, 67*, 599–604.
- Morrow, G. R. (1986). Effect of the cognitive hierarchy in the systematic desensitization treatment of anticipatory nausea in cancer patients: A component comparison with relaxation only, counseling, and no treatment. *Cognitive Therapy and Research, 10*(4), 421–446.
- Mystkowski, J. L., Mineka, S., Vernon, L. L., & Zinbarg, R. E. (2003). Changes in caffeine states enhance return of fear in spider phobia. *Journal of Consulting and Clinical Psychology, 71*, 243–250.
- Obler, M., & Terwilliger, R. F. (1970). Pilot study on the effectiveness of systematic desensitization with neurologically impaired children with phobic disorders. *Journal of Consulting and Clinical Psychology, 34*, 314–318.
- Paul, G. L. (1966). *Insight versus desensitization in psychotherapy*. Stanford, CA: Stanford University Press.
- Sze, K. M., & Wood, J. J. (2007). Cognitive behavioral treatment of comorbid anxiety disorders and social difficulties in children with high-functioning autism: A case report. *Journal of Contemporary Psychotherapy, 37*, 133–143.
- Sze, K. M., & Wood, J. J. (2008). Enhancing CBT for the treatment of autism spectrum disorders and concurrent anxiety: A case study. *Behavioural and Cognitive Psychotherapy, 36*, 403–409.
- Watson, J. B., & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology, 3*(1), 1–14.
- Watts, F. (1971). Desensitization as an habituation phenomenon: Stimulus intensity as determinant of the effects of stimulus lengths. *Behaviour Research and Therapy, 9*(3), 209–217.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review, 29*, 216–229.
- Wolpe, J. (1958). *Psychotherapy by reciprocal inhibition*. Stanford: Stanford University Press.
- Wolpe, J. (1961). The systematic desensitization treatment of neuroses. *Journal of Nervous and Mental Diseases, 132*, 180–203.
- Wolpe, J. (1990a). *Practice of behavior therapy*. New York: Pergamon Press.
- Wolpe, J. (1990b). *Theme and variations: A behavior therapy casebook*. New York: Pergamon Press.
- Wood, J. J., & McLeod, B. (2008). *Child anxiety disorders: A treatment manual for practitioners*. New York: Norton.
- Wood, J. J., Drahota, A., Sze, K., Van Dyke, M., Decker, K., Fujii, C., et al. (2009). Brief report: Effects of cognitive behavioral therapy on parent-reported autism symptoms in school-age children with high-functioning autism. *Journal of Autism and Developmental Disorders, 39*, 1608–1612.

Desipramine

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Synonyms

[Norpramin](#); [Pertofrane](#)

Definition

Desipramine is a tricyclic antidepressant. The term tricyclic refers to the three-ring structure of this class of antidepressant medications. These medications are not used as commonly as in the past as they have been largely replaced by the SSRIs. Desipramine has been used to treat depression and attention deficit/hyperactivity disorder. Desipramine has highly selective norepinephrine reuptake inhibitor properties.

The tricyclic antidepressants have several adverse effects in common including dry mouth, urinary retention, constipation, nausea, increased heart rate, dizziness, and, at higher doses, confusion. The tricyclic antidepressants also carry some risk of altering the electrical conduction in the heart. They are well known to be fatal on overdose due to their potential for causing cardiac arrhythmia. Because of their known toxicity at higher doses, treatment with tricyclic antidepressants requires blood-level monitoring and electrocardiogram monitoring as well. Finally, the tricyclic antidepressants are also vulnerable to drug-drug interaction. For example, some medications such as SSRIs or certain antibiotics may interfere with the breakdown of tricyclic antidepressant medications. The interference of metabolism of the tricyclic can cause a sharp increase in the blood levels

of the tricyclic antidepressants and increase the vulnerability to toxic effects. The tricyclic medications have not been well studied in children or adults with autism.

See Also

► [Antidepressants](#)

References and Reading

Martin, A., Scahill, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.

Desyrel

► [Trazodone](#)

Detection of Autism by Infant Sociability Interview

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Synonyms

[DAISI](#)

Description

DAISI is a semistructured detailed developmental history interview designed to elucidate a child's early sociability and communication. It is for concurrent or retrospective use with parents in clinical and research investigations, particularly those concerning autism. Wimpory, Williams, Nash, and Hobson (2000) provides

details of DAISI with an appendix clarifying its schedule and criteria for positive and negative responses, as scored from verbatim parental responses.

The instrument focuses on developmental progression in the manifestation of social engagement in young preschool children with autism through a semistructured interview drawing out retrospective reports of social engagement from their parents. It employs a conversational style masking when the interviewer moves from one to another of DAISI's 19 items. The interview is designed to facilitate the development of a relationship between parent(s) and interviewer so that honest/accurate replies are more likely. To this end, DAISI involves sensitive probing with situational prompts. Questions are ordered to increase the likelihood that parents will be able to give an initial positive response before having to describe any negative responses on further detailed questioning. Rather than focusing merely on discrete communicative skills, there is a focus on the infant's role in any interactive flow of preverbal engagement.

The interview schedule is reproduced below from an appendix within a paper by Wimpory et al. (2000) documenting DAISI's initial published use. DAISI can take up to an hour to administer and requires an understanding of typical and autistic early social communication rather than specific training. Although it was originally designed clinical psychologists' use for clinical and research purposes, it has subsequently been successfully employed by a range of professionals and researchers.

Wimpory et al. (2000) retrospectively used of DAISI with twenty 2–4-year-olds, prior to any autism diagnoses being made, found 15 key items differentiating the children who were subsequently diagnosed with autism from those with nonautistic developmental delay. Distinguishing items include engaging in dyadic social interaction (i.e., sociability in play with or without toys, socially directing feelings of anger and distress, frequency and intensity of eye contact, greeting parents, waving, raising arms to be picked up, enjoying lap games, preverbal turn-taking, and

using noises communicatively) as well as “triadic” social behavior, involving an object and another person (i.e., referential eye contact, pointing and following others' points, offering and giving, and showing objects).

The evidence from the study by Wimpory et al. (2000) indicated that infants with autism manifest a range of abnormalities suggestive of profound limitations in social engagement. These abnormalities were specific to the group with autism and were *not* simply an expression of general developmental delays because they were not characteristic of the infancies of children in the matched control group. Moreover, the abnormalities were *both* in the area of person-to-person nonverbal communication and interpersonal contact *and* in triadic person-person-object interactions. In the former respect, it is notable that the abnormalities extended to socially directed expressions of anger and distress as well as signs of positive engagement. In the latter respect, there were a majority of infants without autism but *no* infants with autism who were reported to offer, give, show, or point to objects in relation to someone else. Finally, nearly all of the infants in the control group, but not a single child with autism, were said to have followed another person's point.

These findings are in keeping with autism theories that focus on impairments in primary intersubjectivity as well as recognize the importance of difficulties with secondary intersubjectivity (Hobson 1993; Newson 1984; Rogers and Pennington 1991; Wimpory et al. 2002). Concurrent use of the 15 key DAISI items that Wimpory et al. (2000) found to be significant also determined significant group differences between the infant siblings of autistic spectrum disordered (ASD) and typically developing children as detailed below (Stone et al. 2007). Stone et al. (2007) also found significant correlation between total scores from the DAISI and a direct child measure of social-communicative functioning, The Screening Tool for Autism in 2-Year-Olds.

Findings from Wimpory et al.'s use of DAISI, along those from other studies, determined 25 key items for Dereu et al. in screening for ASDs (Dereu et al. 2010). They successfully evaluated

the Checklist for Early Signs of Developmental Disorders (CESDD) in a population of almost seven thousand 3–39-month-olds. In summary, DAISI has been used for research retrospectively, with children who have autism or non-autistic developmental delay and concurrently with infant siblings of children with autistic spectrum disorders (ASD) and typical development. It has also been employed for diagnostic purposes in clinical/educational settings for two decades.

Historical Background

DAISI's development was influenced by child clinical training at Nottingham University's Child Development Research Unit, and particularly by Professor Elizabeth Newson's guidelines (1990). The originality of data published on DAISI, by Wimpory et al. (2000) lies in the fact that the interview was carried out before any diagnosis was made; so the parents did not have any *a priori* assumption when recounting their child's social behavior.

In this respect, the DAISI differs from earlier questionnaires that were used with parents of affected children or adults, such as Dahlgren and Gillberg (1989) and Wing (1969). The latter were completed after the diagnosis of autism had been made and after years of experience of the person with autism, which might have influenced the results. Also, despite the fact that questionnaires are concise and easy to use, they are less sensitive than interviews and provide less accurate answers. These methodological limitations were a feature of almost all previous studies of this kind, even if the period recalled was not so distant. The DAISI interview is designed to give the impression of a natural conversation. There are opportunities for explanation, discussion, and provision of examples as part of the interchange. While previously published diagnostic instruments for young children with autism, such as the Parent Interview for Autism (PIA; Stone and Hogan 1993) and the revised Autism Diagnostic Interview (ADI-R; Lord et al. 1993), record impairments in social relatedness, they do not focus exclusively on

early social engagement in young preschool children.

The DAISI interview was developed to overcome several of the methodological issues that were a feature of previous research involving retrospective parental accounts. For example, it focuses on aspects of social engagement that are prominent in the behavior of typically developing infants and thus identify what may be abnormal in the case of autism. In addition, interviewing parents of young children with a specific focus on the first 2 years of life means that recall is required over a relatively short period (e.g., over 6–24 months in Wimpory et al. 2000).

Psychometric Data

The internal consistency of the DAISI was determined using the Kuder-Richardson-20-statistic (for dichotomous data) on retrospective use of DAISI with parents of twenty 2–4-year-olds, prior to any autism diagnoses (Wimpory 1995). This gave a standardized item alpha coefficient of 0.9. Significant autistic versus developmentally delayed nonautistic group differences emerged from analysis of variance on the total DAISI scores, $F(1,18) = 166.94, p < .0001$.

As indicated above, Wimpory et al. (2000) reported 15 key items that differentiated the infancies of children subsequently diagnosed with autism (mean score = 3.6, SD 2.4, range 0–7) from those with nonautistic developmental delay (mean score = 15.7, SD 2.5, range 13–19; Mann-Whitney $U = 0, p < .0001$). Distinguishing items, computed on an item by item basis (with Fisher's exact one-tailed test), indicated impairments in frequency/intensity of eye contact ($p < .0001^*$) and its referential use ($p < .0001^*$); pointing ($p < .0001^*$) and following others' points ($p < .0001^*$); using noises communicatively ($p < .0001^*$); preverbal turn-taking ($p < .0004^*$); raising arms to be picked up ($p < .0004^*$); offering and giving ($p < .0004^*$); greeting ($p < .005$); showing objects ($p < .005$); sociability during play with toys ($p < .005$); socially directing anger/distress ($p < .010$); sociability during play

without toys ($p < .016$); waving appropriately ($p < .016$); and enjoying lap games ($p < .043$). Asterisks indicate specific items that individually discriminated between the autistic and developmentally delayed nonautistic groups, following stringent Bonferroni correction for multiple comparisons.

For the above retrospective research, DAISI total scores correlated significantly with Childhood Autism Rating Scales (CARS) scores for the entire group ($-.891, p < .0001$; Wimpory 1995). For the subgroup with developmental delay, DAISI total scores and CARS scores showed a significant negative correlation (Spearman rank correlation $-.86, p = .001$); this was not significant for the group with autism (Spearman rank correlation $-.02, ns$; Wimpory et al. 2000). Within each group, there was a relatively small range of DAISI total scores. The significant negative correlation in the case of control (nonautistic developmentally delayed) individuals is of note because here the individuals who were reported to show a number of social deficits on the DAISI (and thus achieved lower scores) were also those who were given relatively high scores for abnormality on the CARS.

Stone et al. (2007) concurrently employed the 15 key DAISI items that the retrospective study by Wimpory et al. had found significant (2000). Stone et al. (2007) reported significant group differences between infant siblings of 64 autistic spectrum disordered and 42 typically developing (TD) children (MD, 1.32; 95% CI, 0.27–2.37). Autistic siblings' mean scores were 12.8 (SD 3.2, range 0–15), while TD siblings' mean scores were 14.4 (SD 1.2, range 10–15). Stone et al. (2007) also found that DAISI total scores correlated significantly with:

1. CARS total scores (0.74, $p < .01$)
2. Mullen Scales of Early Learning subscores (The Early Learning Composite; Visual Reception, Expressive and Receptive Language; 0.46; 0.28; 0.41; 0.39, respectively, all at $p < .01$)
3. Screening Tool for Autism in 2-Year-Olds total scores (STAT; $-0.37, p < .01$).

This last finding supports face validity for the DAISI as a parental report measure for broadly similar child social-communication constructs that are directly assessed by the STAT.

Clinical Uses

The DAISI interview was designed for both clinical as well as research purposes. It has been employed for multi-agency clinical/educational diagnostic purposes in some services in England and Wales for over two decades. The expanded clinical form includes items assessing the triad of autistic impairments during both early and current functioning, while the published form focuses exclusively on aspects of sociability and communication in infancy. Parental responses are recorded verbatim, and each item that corresponds to a specific domain of behavior is scored as present or absent as indicated above, the interview relies on the relationship between the interviewer and the parent so that accurate and honest answers are more likely to be provided. DAISI's clinical advantage over standardized diagnostic interviews is that it can be administered much more quickly and affords a more conversational experience, so assisting the clinician in gaining a good rapport with clients within routine diagnostic assessments.

The DAISI Schedule

The following section identifies the specific domains of functioning assessed using the DAISI. Each domain contains a key question (italicized in bold below) and may also have associated questions. These are designed to elicit a comprehensive description of behavior relevant for the domain under consideration. The key questions, identified by corresponding item numbers, are those used to determine DAISI scores, as outlined below. These key questions may be substituted by and/or preceded and/or followed by associated questions. This arrangement is designed to allow the interviewer to assist parents both to gain confidence in answering the key questions and to clarify their answers to those questions. Responses to

each key question (either direct or indirect via associated questions) determine the score for its corresponding numbered DAISI item. Examples and criteria for positive and/or negative replies are shown in regular italicized print in the section below.

Questions are arranged below in the order most compatible with the flow of a natural conversation. In this way, responses to more than one domain may be recorded from one segment of conversation. For example, greeting and reaching up to be lifted up from a cot are juxtaposed although these are later analyzed separately as aspects of sociability and gestural communication.

Eye Contact (Item 1)

Did he/she look at you more or less readily as a baby (<2 years) than he/she does nowadays?

Did his/her readiness to give eye contact change at any stage (<2 years)?

Key Question for Item 1: Did he/she have difficulties in the frequency and/or intensity of eye contact? (*This item and item 2 below are subject to a special scoring procedure: They are scored as negative when direct observation of the child reveals poor eye contact at the time of diagnosis and where parents report both that their child's readiness to give eye contact has not changed since infancy, and that they do not see eye contact as a problem for their child.*)

Soothability from Crying (Items 4 and 5)

How would you stop him/her crying as a baby?

Key Question for Item 4: Could you stop him/her crying by picking him/her up? (*Positive responses include those where this strategy worked for at least a few months of infancy.*)

Key Question for Item 5: Could you stop him/her crying by just talking to him/her? (*Positive replies include communicative use of "baby talk," i.e., employing singing, vocalizations, and facial expressions but no physical contact or movement. Negative responses include those for infants described as never interested in social interaction.*)

Greeting, Requesting to Be Picked Up, and Waving (Items 3, 15, and 14, respectively)

What would he/she do when you went to his/her cot after he/she had woken (naturally) from a sleep? Where would he/she be looking?

What would her/his face be like?

Key Question for Item 3: Would he/she greet you? (*Positive responses include manifest pleasure or excitement and/or appropriate facial expression while looking toward parents. Negative responses include a failure to look pleased on most occasions where there was potential for greeting.*)

What would he/she do if he/she wanted to come out of the cot or be lifted from the floor?

Would he/she touch you or the cot while reaching up as if to climb up/out physically?

Did you need to offer your own arms for him/her to lift his/her?

Key Question for Item 15: Would he/she spontaneously lift her arms to be picked up? (*Positive responses cover spontaneous non-tactile gesturing including the support of vocalization, eye contact, etc.*)

Would he/she appear to notice if someone he/she knew well was leaving?

What would he/she do?

Would he/she wave if they (or you) waved?

Would he/she need you to tell him/her to wave or to lift his/her hand for her?

Would he/she wave spontaneously?

How did he/she do it? (i.e., to distinguish from arm flapping)

Where would he/she be looking?

Key Question for Item 14: Would he/she spontaneously and appropriately wave goodbye? (*Positive responses cover spontaneous waving with apparently appropriate communicative intent, as indicated by context, looking toward the other's face, etc. Negative responses include only brief acquisition of waving and/or waving an arm for social or motoric stimulation without apparent understanding of its gestural significance.*)

Lap Games (Items 7 and 8)

What did he/she tend to do during lap games?

Key Question for Item 7: Did he/she enjoy lap games?, e.g., “Round and round the garden,” “Peek a boo.” (*Negative responses included a lack of interest in lap games.*)

Would he/she watch you doing the actions?

Would he/she try to join in?

How did he/she show his/her enjoyment?

Key Question for Item 8: Did he/she actively participate? (*Positive replies require use of body actions, e.g., imitative clapping.*)

Social Engagement During Play With and Without Toys (Items 9 and 6, respectively)

Would he/she be happy for you to play with him/her?

How would he/she react if he/she was already occupied with toys?

Key Question for Item 9: Would he/she be happy for you to join in his/her play with toys or would he/she regard that as an intrusion and prefer to play alone? (*Positive responses include descriptions of infants apparently happy for parents to play alongside them without parents feeling excluded.*)

Would you need toys in order to play with him/her?

Key Question for Item 6: Could you amuse him/her without toys (if say, you were together on a bus or in a doctor’s waiting room where no toys were available)? (*Positive replies may include chatting and/or singing, play with body parts, etc.*)

Showing, Offering and Giving, Referential Eye Contact, Pointing, and Following Points (Items 11, 10, 2, 12, and 13, respectively)

Did he/she sometimes want to draw your attention to his/her toys?

(Or did he/she seem too interested in them to share them with anyone else?)

Key Question for Item 11: Would he/she show you things? (*Positive replies include either holding an object up to another’s field of view or pointing to it and simultaneously looking at the other person. Such referential eye contact also scores positively on item 2, below. Communicative pointing also scores positively on item 12, below.*) (Depending on responses to previous questions. . .)

What would he/she do if he/she wanted you to share his/her experience of a toy?

Would he/she hold it up for you to see? Where would he/she be looking?

Key question for Item 10: Would he/she offer and give objects? (*Positive replies include pausing and looking to the recipient’s face before giving.*)

Would he/she give a toy (or other item) to you?

Was this in response to a request or would it be spontaneous?

Have you known babies who like to give something (e.g., a biscuit) to other people . . . babies who give it very carefully, often breathing heavily as they do so, and then they want it back as soon as they have given it?

Did he/she like to play giving and taking games like that or did he/she tend to “post” or place objects on you instead?

Where would he/she be looking before and during the act of giving?

Key Question for Item 2: Would he/she look both to where he/she was pointing and to you? (Referential eye contact)

What would he/she do if he/she wanted something (e.g., a biscuit) out of reach?

(If reaching) How would he/she position his/her fingers?

Where would he/she be looking?

Key Question for Item 12: Would he/she use pointing communicatively? (*Positive replies include eye- or finger-pointing to request and show items of interest accompanied by eye contact. Negative responses include extension of index finger with no apparent communicative intent.*)

What would he/she do if she saw something of interest like a plane, or an animal across the street?

(If reaching) How would he/she position his/her fingers?

Where would he/she be looking?

Did he/she take notice if you pointed at something or did he/she tend to be preoccupied with his/her own interests?

What would he/she do if you pointed (at near and far objects, e.g., an animal across the street, the correct hole for a puzzle piece, etc.)?

Key Question for Item 13: Could she follow your pointing gestures?

Where would he/she look . . . toward your finger or to where you were pointing?

Expressing Directed Anger and Distress (Item 18)

Did he/she have tantrums?

Where would he/she be looking during these?

What would he/she do if he/she was hurt?

Would he/she let you know how he/she was hurt?

Where would he/she be looking?

Key Question for Item 18: Would he/she appear to direct anger and/or distress with apparent communicative intent? *(Negative responses include toddlers who would avoid looking toward other faces during expressions of anger and/or distress. Positive responses include toddlers who directed anger toward parents when feeling physical pain unrelated to parental behavior.)*

Teasing (Item 16)

Did he/she understand “No” even if he/she chose to ignore it?

Have you noticed some toddlers will still do what they have been told not to do (e.g., touch an electric switch) and will be smiling and looking to their parents at the same time as if they are doing it again *because* they have been told not to do it?

Was he/she a toddler who was interested in doing that?

Can you give examples? Where would he/she be looking?

What would his/her face be like as he/she did it?

Key Question for Item 16: Would he/she tease you? *(Negative responses include enjoyment of playful reprimands, such as being chased, rather than manifesting playful provocation/teasing per se.)*

Can you think of other ways in which he/she would tease you?

Preverbal Turn-Taking and Use of Vocalizations (Items 19 and 17, respectively)

Did he/she make baby noises?

(Positive responses enable progression to the following questions.)

Did he/she make these just for him/herself or did he/she seem to be making them for you to listen to him/her?

How did he/she show that they were for you?

Where would he/she be looking?

Key Question for Item 19: Were his/her baby noises communicative? *(Negative responses include an absence of babbling or parental inability to recall communicative use of babbling despite parental expectation that this occurs.)*

Have you noticed how some babies like you to join in with their babbled noises, so that there is a turn-taking pattern between you and them – as if the two of you are speaking another language? *(Positive answers are required before proceeding.)*

Was he/she the kind of baby who did that?

Were you able to have babbling conversations with him/her?

Did he/she use his/her early words for him/herself or for giving messages to you?

Where would he/she be looking when using them?

Key Question for Item 17: Did he/she take turns before he/she could talk, e.g., with babbled noises? *(Positive responses include turn-taking flows established by (a) infants repeating a babbled noise as if with communicative intent apparently in response to an adult’s imitations of those noises and (b) active silent participation in a flow of interaction using appropriate facial expressions and communicative body actions during a period of mutism.)*

See Also

- [ADI-R](#)
- [CARS](#)
- [STAT](#)

References and Reading

- Dahlgren, S. O., & Gillberg, C. (1989). Symptoms in the first two years of life. *European Archives of Psychiatry and Neurological Sciences*, 238, 169–174.
- Dereu, M., Warreyn, P., Raymaekers, R., Meirsschaut, M., Pattyn, G., Schietecatte, I., et al. (2010). Screening for autism spectrum disorders in Flemish day-care centres with the checklist for early signs of developmental disorders. *Journal of Autism and Developmental Disorders*, 40(10), 1247–1258.
- Hobson, R. P. (1993). *Autism and the development of mind*. Hillsdale: Erlbaum.
- Lord, C., Storoschuk, S., Rutter, M., & Pickles, A. (1993). Using the ADI-R to diagnose autism in preschool children. *Infant Mental Health Journal*, 14, 234–252.
- Mullen, E. (1995). *Mullen scales of early learning*. Circle Pines: American Guidance Service.
- Newson, E. (1984). *The social development of the young autistic child*. Paper presented to the National Autistic Society Conference, Bath.
- Newson, E. (1990). *Questions to ask about the first three years when autism is suspected*. Unpublished manuscript, Child Development Research Unit, Nottingham University.
- Rogers, S. J., & Pennington, B. F. (1991). A theoretical approach to the deficits in infantile autism. *Development and Psychopathology*, 3, 137–162.
- Scholpler, E., Reichler, R., & Renner, B. R. (1986). *The Childhood Autism Rating Scale (CARS) for diagnostic screening and classification of autism*. New York: Irvington.
- Stone, W. L., & Hogan, K. L. (1993). A structured parent interview for identifying young children with autism. *Journal of Autism and Developmental Disorders*, 23, 639–652.
- Stone, W. L., McMahon, C. R., Yoder, P. J., & Walden, T. A. (2007). Early social-communicative and cognitive development of younger siblings of children with autism spectrum disorders. *Archives of Pediatrics & Adolescent Medicine*, 161(4), 384–390.
- Wimpory, D. (1995) *Social engagement in preschool children with autism*. Unpublished doctoral thesis, University of Wales, Bangor, Gwynedd.
- Wimpory, D., Williams, J. M. G., Nash, S., & Hobson, R. P. (2000). Are infants with autism socially engaged? A study of recent retrospective parental reports. *Journal of Autism and Developmental Disorders*, 30(6), 525–536.
- Wimpory, D., Nicholas, B., & Nash, S. (2002). Social timing, clock genes and autism: A new hypothesis. *Journal of Intellectual Disability Research*, 46(4), 352–358.
- Wing, L. (1969). The handicaps of autistic children—a comparative study. *Journal of Child Psychology and Psychiatry*, 10, 1–40.

Detriment in Skill

► Disability

Developing Countries and Autism

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Definition

Autism spectrum disorder (ASD) represents a group of lifelong, complex neurodevelopmental disorders emerging during early childhood and interfering with a person's ability to socially relate to and interact with others. According to the American Psychiatric Association Diagnostic and Statistical Manual, Fifth Revision (DSM-5), the current diagnostic criteria for ASD include deficits in: (a) social interaction and nonverbal communication; and (b) restricted, repetitive movements, behaviors, and interests (American Psychiatric Association 2013). The World Health Organization (WHO) International Classification of Diseases, Tenth Edition (ICD-10) characterizes ASD by impairments in three core areas: social interaction; communication; and restricted, repetitive behaviors (World Health Organization

1994). The ICD-11 is scheduled for release in 2018 and expected to follow the new ASD nosology.

Historical Background

According to the United States Centers for Disease Control and Prevention, ASD now affects 1 in every 68 children (1 in 42 boys and 1 in 189 girls) in the US (Christensen et al. 2016). In 2010, there were approximately 52 million people living with ASD worldwide (Elsabbagh et al. 2012; Baxter et al. 2015). The data on prevalence of ASD in low- and middle-income countries (LMICs) is limited. Most research into the epidemiology, causes, clinical presentations, diagnosis, and treatment of ASD is based on studies conducted in high-income countries (HICs). This has led to a misperception that ASD is more prevalent in HICs versus LMICs. For example, while only 20% of the world's current population resides in HICs, 86.5% of all reported cases of ASD have been in HICs (Elsabbagh et al. 2012; Population Reference Bureau 2015). However, it is important to note that current data in HICs shows that ASD occurs across all levels of socioeconomic status (SES), and the reported association of ASD and higher SES has been due to selected study sampling. Furthermore, uneven rates of diagnosis point to variation in ASD surveillance rates by race and ethnicity, underscoring that children in resource poor regions in HICs may not be appropriately identified (Center for Disease Control and Prevention 2016; Christensen et al. 2016). Barring geographic environmental and genetic variation, there is no reason to expect that prevalence of ASD in LMICs will be lower than that consistently reported in HICs.

In 2007, the United Nations (UN) representative from Qatar had successfully proposed a UN General Assembly resolution creating World Autism Awareness Day. This day, recognized on 2 April every year, encourages all member states to take measures to raise awareness about ASD throughout the world. Further, on World Autism Awareness Day in 2016, the UN General Assembly convened an expert panel that emphasized that

children and adults with ASD and other neurodevelopmental disorders (NDDs) have a special place at the heart of the UN Sustainable Development Agenda and implementation of the Sustainable Development Goals. In 2016, the World Innovation Summit in Health hosted the Autism Forum entitled "A Global Framework for Action" (Munir et al. 2016). There is a growing momentum recognizing ASD globally, and advocating for the needs and rights of children in LMICs with ASD, and their families. There is a need to strengthen national capacities in LMICs within a larger context of global advocacy. This chapter will outline the current knowledge of ASD in LMICs, and highlight challenges currently faced by families, communities, and countries as they aim to advocate for the rights of children with ASD. This chapter will also offer policy recommendations to overcome these challenges.

Current Knowledge

Detection and Diagnosis of ASD

While the timely detection of ASD is a challenge globally, it is a particular problem in LMICs where there is less general surveillance for developmental delays in health and education settings. There are also less opportunities for targeted ASD screening, developmental evaluations, and eventual diagnoses made among high-risk children.

In many LMICs, there is little general knowledge about NDDs, and many parents are not aware of the appropriate timing for developmental milestone skills related to speech and language (Ertem et al. 2007). Review of previous studies in sub-Saharan Africa (SSA) shows that among health workers, a small percentage views ASD as treatable while few others see it as being preventable (Bakare et al. 2016). Community perception of ASD in SSA has a wide range of implications ranging from limited understanding of the disorder, fear of stigmatization, and social exclusion, in the absence of appropriate policies to improve care and community inclusion. As a result, even when parents do have developmental or behavioral concerns, they often still do not reach out to

health care providers because they mostly associate the clinicians with treating acute illnesses. In addition, in many LMICs, the stigma around ASD, as with other NDDs, may be so pervasive as to discourage parents from seeking medical attention when a concern arises, or denying further evaluation or treatment when suggested to them from a medical or educational professional.

In fact, most clinicians in LMICs are not trained in developmental milestones or how to assess them during routine pediatric clinical care (Durkin et al. 2015; Bakare et al. 2016). As a result, developmental delays often go unnoticed also by clinicians, thus children are not referred for further evaluation that could lead to a proper diagnosis. There is also a severe shortage of clinicians who would facilitate more targeted screening, evaluation, and diagnostic procedures for children with ASD and other NDDs in LMICs. Those that do have proper training are mostly congregated in urban settings, leaving large numbers of children in rural regions without diagnostic resources. In the school systems, teachers also lack training and knowledge about ASD, and therefore typically do not pick up on early signs of ASD among children in their classrooms.

Evidence-Based Therapies

Young children with ASD require interventions that target language skills, joint attention, and emotional reciprocity, and therefore treatments should begin as early as possible. Research in HICs has found that early interventions initiated before 3 years of age are likely to have the greatest positive impact on outcomes (Zwaigenbaum et al. 2015). There are evidence-based early interventions, including Applied Behavioral Analysis (ABA); other treatments include speech and language, occupational and physical therapies, social skills groups, as well as management of comorbid conditions such as associated medical (e.g., sleep and gastrointestinal) and neurological (e.g., seizure) disorders, anxiety, and challenging and maladaptive behaviors. In contrast to HICs, the majority of children in LMICs have no access to treatment. In South East Asia with the largest number of children, between 2 and 9 years, the

key barriers to treatment continue to include both the paucity of interventions, as well as lack of trained staff to deliver them outside the reach of specialist centers (Patel et al. 2013). It is feasible to adapt early interventions evidenced in HICs by using “task-shifting” strategies, as demonstrated by the Parent-mediated intervention for ASD in South Asia (PASS) randomized control trial (Rahman et al. 2016). The development of such opportunities is likely to contribute to more optimal care and outcomes in LMICs. The quality of services that are provided is often very variable as well. Children in rural areas, in particular, often have limited access to therapeutic services, which may only be provided in urban settings. Furthermore, many families in LMICs do not have health care coverage to pay for the high cost of services related to ASD and there has been limited adaptation of services that can be provided at lower cost to children and families with few exceptions (Rahman et al. 2016).

Family Support Systems

Families are the constant and consistent source of support for children and as such can wield tremendous influence on the growing and developing child. In all corners of the world, especially where resources are scarce, the family is undoubtedly the vehicle by which education and thus “intervention” and direction for the child can be provided most efficiently and possibly most effectively.

Families whose children are perceived as different feel isolated, alone, and often stigmatized, and too often take on self-blame (Dunst et al. 1994). This is especially true for parents of children with ASD. The pervasive lack of knowledge about ASD magnifies self-doubt, isolation, and stigma that plague many families. Families struggle to know how best to deal with their child’s disability in the home setting or in the community (Hecht et al. 2011) and may not know anyone to ask or to mentor them. The lack of knowledge about ASD coupled with the lack of available role models also prevents the family from learning how their child is best taught and responded to in their homes and communities. This leads to isolation and feelings of stigma.

Many parents of children with disabilities have lower rates of, and diminished opportunities for, employment and advancement than parents of children without disabilities (Emerson 2007). They thus often lack adequate financial resources to pursue formal instruction on how to teach or effectively intervene with their child's special needs; and formal systems' supports are underfunded and scarce (Seltzer et al. 2001; Parish et al. 2009). It is thus incumbent and timely that policy makers assist and support the training of families to best address the needs of the child with ASD and related neurodevelopmental disabilities. This may be especially true in rural and LMIC contexts.

Education

Education has been globally recognized as a basic human right (UN 2008). This perspective has been reflected in a number of international policies including the United Nations Convention on the Rights of the Child, the International Covenant on Economic, Social and Cultural Rights, and the United Nations Convention on the Rights of Persons with Disabilities (UN CRPD; UNESCO 2015). In particular, the UN CRPD, an international treaty passed in 2008, represents an important shift in awareness and advocacy, with an emphasis on considering persons with disabilities as empowered individuals with important rights. These include the right to an "inclusive, quality and free primary and secondary education on an equal basis with others in the communities in which they live." (UN CRPD article 24: UN 2008).

Despite this policy and its current adoption by 162 member states (as of March 22, 2016), children with disabilities continue to face barriers accessing an inclusive public education in many LMICs. In fact, approximately one-third of the over 60 million children still excluded altogether from public schools worldwide are children with disabilities (UNESCO 2013).

The goal of providing children with an inclusive education in mainstream public (government funded) schools is an important one. The path from exclusion to inclusion is a continuum, and many communities fall somewhere in between

with their current practices, which may also vary depending on the severity of a child's disability. While many LMIC countries promote segregated special schools as a means to educate children with ASD and other disabilities, evidence has shown that this can increase prejudice and stigma in societies, and further perpetuate discrimination later in life (Frederickson and Cline 2002). Because segregated schools are often only present in urban areas of LMICs, relying on these separate schools to educate children with disabilities may result in children from rural regions excluded from the education system altogether.

Integration has been a stepping-stone for many countries as they move towards the goal of inclusion. Inclusion with proper support services and accommodation for children with disabilities has been presented as the model learning environment, with evidence showing that it can help all students, both with and without disabilities, reach their full potential (Mariga et al. 2014). Children with moderate to severe disabilities, such as children with both ASD and an intellectual disability, are at highest risk of being excluded from the mainstream educational system in their country. Those with mild disabilities, such as children with ASD without an intellectual disability, may receive their education in the mainstream public education system, but often missing the needed modifications and support services, such as speech therapy and occupational therapy. The lack of enforcement of existing policies that guarantee children with ASD and other disabilities the right to an equal, inclusive education is often due to a shortage of properly trained teachers and appropriate school curriculum. Many LMICs do not provide special education training for teachers, or provide general education teachers with training on how to accommodate a child with a disability in their classroom. Furthermore, appropriate school curriculum and other educational modifications are not present in most school systems in LMICs. Nor do they have resources to develop their own learning programs and materials for children with ASD, and do not have a system for accommodating children with ASD and other disabilities into a regular classroom.

Research and Surveillance

Health research is an important tool for improving the lives of children in LMICs. However, despite the fact that the greatest number of children with ASD live in LMICs, very little relevant research has been conducted in these settings (Durkin et al. 2015). Studies on important topics related to the ASD have mainly been conducted in HIC and therefore provide an incomplete and biased view of the global ASD burden and challenges facing the autism community worldwide. In particular, there is a critical gap in quality epidemiologic research that would accurately describe the prevalence of ASD and other neurodevelopmental disabilities in LMICs.

Proper epidemiologic studies involve the use of validated research tools for systematic clinical screening and diagnosis. Cost of illness studies are also needed to help governments understand the true economic burden of ASD in their country. These types of studies are important for planning and advocacy purposes, resource allocation, and to create a strong justification for the development of provider and researcher training programs, service provision, and additional research. Additional studies are also needed to evaluate the effectiveness of interventions delivered to children with ASD and other neurodevelopmental disabilities in LMICs and the resources required to provide these services. Ideally, results from such studies would enable LMICs to more efficiently utilize their limited mental health resources by taking a systematic approach to prioritize the utilization of the most cost-effective interventions for treating children with ASD and other neurodevelopmental disorders.

However, researchers in LMICs face a number of barriers in their attempt to conduct quality research related to ASD and mental health care more generally (Sharan et al. 2007). For instance, many LMICs do not prioritize the research questions that would maximize health gains and other priorities within their country. As a result, there is a risk that external research funders will pursue a research agenda that meets their own goals, or the goals of the larger international community, which may not be consistent with the needs of the country. In addition, research is often not

prioritized because it rarely informs policy development in the local context. And too few health researchers in LMICs are trained in rigorous standardized methods, including the responsible conduct of research. Researchers without proper training may produce studies that are of poor quality and have biased research results. Data emerging from HICs underscores importance of controlling for biases in detection when conducting epidemiological studies of ASD.

Future Directions

Diagnosis

To increase rates of early diagnosis, there is a need to first increase the surveillance of developmental outcomes in the home, community, health care, and education settings. Education and awareness campaigns have been successful in educating parents in LMICs about ASD. Within the health care system, there is a critical need to provide training and education to clinicians at the primary care level while also developing additional specialized training programs for clinicians to perform more targeted screenings and evaluations for ASD and other neurodevelopmental disorders.

At the primary care level, rates of diagnoses can be increased by providing information to pediatricians about the appropriate developmental milestones as well as signs of ASD and other neurodevelopmental disorders. This information should also be incorporated in the medical curriculum of clinicians. Guidelines should be developed that include strategies to include general developmental surveillance into routine pediatric visits. Structured developmental pediatrics, psychology, and child psychiatry training programs are also needed to train more clinical personnel to perform screenings and evaluation procedures targeted at individuals deemed to be at high risk of ASD and other developmental disabilities based on surveillance in the home, clinic, school, and community settings. The training of these specialists must occur in parallel with greater accessibility to simpler surveillance, screening, and diagnostic tools that can be used with fewer resources than those currently used in HIC.

Teachers and educators are valuable resources who spend their days working with, playing with, and watching children. They are already familiar with some developmental milestones. They are also a trusted source for parents. The teachers in the LMICs can be trained in identifying developmental delays, both as part of their school curriculum and further educated on these needs while working in the classroom. Teachers learn about various milestones to watch for at different ages. These activities need to be appropriately adapted to the sociocultural context.

Evidence-Based Therapy

LMICs need formal training programs for health professionals to develop expertise in evidence-based services. High intensity interventions commonly available in HICs should be adapted to be less resource intensive. Even so, the number of formally trained professionals in LMICs will increase slowly.

While this formal capacity is built up, there is an opportunity to reach out to parents of children diagnosed with ASD and train them to provide services directly to their children in their homes. Some evidence has shown that training parents to provide interventions to their child with ASD can result in favorable outcomes including increased language comprehension and reduction in severity of core symptoms of ASD (Oono et al. 2013, Cochrane Review).

For services provided by health professionals, quality control mechanisms such as certifications for training and licensing for programs are necessary to ensure adequate quality. Importantly, providing health care coverage to children with ASD and other neurodevelopmental disorders would help alleviate the cost burden faced by many families who struggle to pay for 11 intervention services of for their child. Expanding existing programs that provide health care coverage for some infectious diseases may be a starting point for providing coverage for larger range of services for chronic and mental illness, including ASD.

Family

To maximize impact and broadly affect families' quality of life, policy makers, governmental, and

nongovernmental organizations (NGO's), need to create and offer services and support to families raising children who have ASD. This is paramount in order for these families and their loved ones with ASD to attain their optimal health and reach their maximum human potential. This also aligns with Article 25 of the United Nations Convention on the Rights of Persons with Disabilities (United Nations 2008).

Community and governmental efforts that have proven to be most effective in assisting families includes the family voice. That is, by allowing families to become part of the national policy discussion, family concerns are more directly addressed (Hecht et al. 2011). Access to the perspectives of families allows professionals and systems to learn from first-hand experiences. This can result in adjustments to services that meet their needs resulting in improved support to families.

A three pronged approach would include: (1) connecting families to each other; (2) creating programs; and (3) providing sustained support. This is important to up-to-date information, and to policy makers are the three most important elements that families have identified as critical in the effort to support them and their affected child (Hecht et al. 2011).

- (1) *Connecting families to each other:* These connections go a long way towards assisting families in recognizing that they are not alone; that there have been others who have experienced what they are facing and have been successful. Supplying families with needed information is crucial in assisting them in understanding and supporting their family member to reach their potential. These efforts have often been organized and directed by families seeking other families who have experiences similar to their own. Family-led organizations and coalitions have shown to be an effective and low cost way to assist in these connections (will add citation). Policy and program directors can enhance these efforts through providing relatively minimal support to initiate and sustain structures (e.g., developing support groups, family events/

activities) that allow families to connect to other families. In situations where the internet is available, many connections can be initiated online where families can meet and even communicate directly with other families about their concerns (New England Genetics Collaborative 2016). These connections are enhanced when families receive up-to-date information about their child's condition and how best to work with their child (ASC 1016). This is an effective communication strategy that can work especially well in rural and less developed countries when the technology can be utilized. A successful initial effort has been to discover how families currently connect with one another. Leaders in the family movement must be identified and supported so that they can effectively articulate what families need. By inviting families into professional and governmental programs, the needs of families rise to the top and family perspective becomes evident and concerns are heard and addressed (see the ► [Family Therapy](#) case study). Connecting families and family leaders to governmental initiatives is the third part of making connections. This is critical in the development of programs and is a major part of the social strategy that begins to change what families can be offered and how families are supported. These efforts impact the policies that are then developed and that create programs. Policy makers begin to hear the family voice and understand the needs and what would help the most. Integration of the family perspective results in the development of the most effective strategies and policies to address the problems.

- (2) *Creating programs:* Opportunities to participate in programs need to be developed that educate families about the conditions that impact their children. These educational programs can be made readily available by utilizing online training. Policy makers should create the infrastructure that grants the family access to professionals and community health workers. Training opportunities may vary such that information provided to families can range in complexity. Training in skill

development for the families in interacting with the affected family member, and support for parental coping skills may also be provided. This type of system support mechanism can assist in the child's development and help to reduce parental stress. By inviting families to be part of the decision-making process, the programs are more likely to be successful.

- (3) *Providing sustained support:* Although early diagnosis and treatment have been shown to increase the adaptability and skill levels of children with ASD, support for the growing child and family must be sustained over time. The developmental trajectory of the child brings about new challenges and additional or changing needs for support. Much information focuses on the young child and the early interventions provided shortly after diagnosis, but support during development must also be addressed (Hecht et al. 2011). Programs need to be developed that assist the growing child in reaching their full potential throughout their development with an emphasis on supporting an individual's independence and full inclusion into their communities. As families strive to better understand and provide for their children with ASD and related neurodevelopmental disabilities, community health care workers and other professionals (including teachers or school personnel) can be an effective and efficient way to provide the first level of response for families. These teachers or community workers need to be trained to help families understand and cope with the issues they are facing. The Richmond-Kotelchuck Model for Policy Development can serve as a dynamic representation of what is needed for successful policy implementation and outcomes. We have the basic knowledge regarding expanding needs of families whose children have ASD, and there are social strategies that can be utilized that support families. It is up to the political will of policy makers to allow these supports to flourish by welcoming the family perspective into governmental

initiatives in this area (Richmond and Kotelchuck 1983).

Education

There is a critical need to provide a broader set of training programs and certifications to teachers in LMICs that would facilitate the inclusion of children with disabilities into mainstream public schools. Ideally, these programs should be structured to provide special education training to a group of educators as well as provide general education teachers with skills that would allow them to knowledgeably accommodate children with disabilities such as ASD into mainstream, inclusive classes.

There is also an opportunity to train special education teachers in skills and interventions that would improve their work with children with ASD specifically, including applied behavior analysis practices, and peer-mediated intervention (National Autism Center 2009). In parallel to these training programs, there is a need to provide special education teaching and learning resources that can be adapted by individual school systems. For children with ASD and moderate to severe intellectual disabilities, there may be a need to create a separate core curriculum based on the general needs of this group. Goals for these children may include educational, therapeutic, and social ones, including: reading literacy and comprehension, math, social science, sensory therapy, communication and social skills, and functional and life skills. Children with ASD without intellectual disabilities may follow a general education with added support and modifications based on their specific therapeutic and learning needs (Fogarty and Lofland 2015). Efforts should also be made to tailor special education goals to the specific needs of each child. A common strategy for doing this in higher resource settings is to create individual education plans (IEPs) for each child that takes into account their specific needs and goals. These plans can include special arrangements for learning tasks and exams, allowing for exemptions for assignments and assessments, extra time for learning tasks and tests, and

adaptation of the conditions or the format of the task (UNESCO 2015).

And finally, systematically collected data is a central element to monitor educational goals for children with ASD and other disabilities. As part of this, governments need to provide a system and infrastructure that enables school systems to collect and report data on the progress of children in the school environment. This is typically done through annual assessments of student progress. To prioritize and act on these goals, countries should develop an action plan with clear targets and a timetable for implementation. This action plan should be overseen by a core group of administrators within the Ministry of Education that are held accountable for reaching targets by pre-specified deadlines. These goals also create a parallel need for community awareness and education with regards to the benefits of including children with ASD and other disabilities into mainstream schools. These advocacy efforts are often best accomplished by building partnerships between schools, parents, and other stakeholders. It should also be noted that when full inclusion does not seem feasible for a given country, integration of children with ASD and other disabilities into the mainstream school through the coexistence of general education and special education programs within the same school could be an important step to full inclusion.

Research and Surveillance

As an international priority, the World Health Organization (WHO) has urged countries to improve research surveillance frameworks and information systems to better capture data on ASDs (WHO 2013). On a national level, research questions should be prioritized to accommodate both needs and preferences of stakeholders in the local context, and should not be set solely by external research funders. Researchers and policy makers in LMICs should drive a research agenda based on local factors including data that would help with advocacy efforts and creating priorities for national action plan.

There have been different methods used to develop consensus on national research priorities,

and the optimal approach will vary by context. However, nine common themes for best practices in priority setting have emerged in the literature and were summarized by researchers at the WHO in 2010 (Viergever et al. 2010). These best practices are summarized in the appendix. Following the setting of ASD research priorities, it is important to have trained researchers to implement the action plan. However, there are often not enough highly trained researchers in LMICs due to a lack of research-training opportunities. To increase the number of training opportunities, LMIC governments should partner with HIC governments and universities to prepare ASD researchers in high quality research methods. As these training partnerships develop, it is also important to internally strengthen mental health training opportunities in schools of public health and medicine (Sharan et al. 2007), including training in public health research methods, knowledge diffusion, leadership, mentorship, and advocacy (Thorncroft et al. 2012).

While funding for such capacity building is often a barrier, external donors should be encouraged to systematically include capacity-building programs and funding that is earmarked towards capacity-building goals in their projects. As this capacity is being developed, the ASD research environment also needs to be strengthened within LMICs. Critical components of an enabling research environment include appropriate research policies, including procedures to monitor and review ethical considerations in human subject research, basic infrastructure setup that includes administrative support within research centers to oversee research contracts and finance, equipment and supplies, and access to resources such as published literature (Sharan et al. 2007). To help with this last goal, in 2002, the WHO developed the Health InterNetwork Access to Research Initiative (HINARI) in collaboration with major publishers to enable researchers from LMICs to gain access to health research literature. Today approximately 15,000 journals (in 30 different languages) are available to health institutions in more than 100 LMICs (“HINARI Access to Research in Health Programme,” 2016). While the development of a

stable research environment certainly requires upfront investment, over time it will save time and resources by allowing for easier administrative access to research funds and stronger partnerships with funded research programs. Integrating with the health research systems that are already in place, such as those for infectious diseases, can enhance synergies and create a more sustainable national research platform going forward. Strong research partnerships between academic groups, NGO, and governmental entities also create more stable environments and important synergies moving forward.

See Also

- [Autism and the Caribbean](#)
- [Brazil and Autism](#)
- [China and Autism](#)
- [Egypt and Autism](#)
- [Ethiopia and Autism](#)
- [Family Therapy](#)
- [India and Autism](#)
- [Kuwait and Autism](#)
- [Macedonia and Autism](#)
- [Pakistan and Autism](#)
- [Palestine and Autism](#)
- [Singapore and Autism Spectrum Disorder](#)
- [Social Class and Autism](#)
- [South Africa and Autism](#)
- [STEM Education and Autism Spectrum Disorder](#)
- [Turkey and Autism](#)

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References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: APA Press.
- Bakare, M. O., Bello-Mojeed, M. A., Munir, K. M., Ogun, O. C., & Eaton, J. (2016). Neurodevelopmental

- delay among children under the age of three years at immunization clinics in Lagos State, Nigeria – preliminary report. *Scientific Reports*, 2016(6), 25175.
- Baxter, A. J., Brugha, T. S., Erskine, H. E., Scheurer, R. W., Vos, T., & Scott, J. G. (2015). The epidemiology and global burden of autism spectrum disorders. *Psychological Medicine*, 45(3), 601–613.
- Centers for Disease Control and Prevention. (2016). Autism spectrum disorder (ASD), data & statistics. Available from: <http://www.cdc.gov/ncbddd/autism/data.html>.
- Christensen, D. L., Baio, J., Braun, K. V., Bilder, D., Charles, J., Constantino, J. N., et al. (2016). Prevalence and characteristics of autism spectrum disorder among children aged 8 years – Autism and Developmental Disabilities Monitoring Network, 11 sites, United States, 2012. *Morbidity and Mortality Weekly Report. Surveillance Summaries*, 65(3), 1–23.
- Dunst, C. K., Trivette, C. M., & Deal, A. F. (1994). *Supporting and strengthening of families: Methods, strategies and practices* (p. 1). Cambridge, MA: Brookline Books.
- Durkin, M. S., Elsabbagh, M., Barbaro, J., Gladstone, M., Happe, F., Hoekstra, R. A., et al. (2015). Autism screening and diagnosis in low resource settings: Challenges and opportunities to enhance research and services worldwide. *Autism Research*, 8(5), 473–476.
- Elsabbagh, M., Divan, G., Koh, Y. J., Kim, Y. S., Kauchali, S., Marcin, C., et al. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research*, 5(3), 160–179.
- Emerson, E. (2007). Poverty and people with intellectual disabilities. *Mental Retardation and Developmental Disabilities Research Reviews*, 13(2), 107–113.
- Ertem, I. O., Atay, G., Dogan, D. G., Bayhan, A., Bingoler, B. E., Gok, C. G., Ozbas, S., Haznedaroglu, D., & Isikli, S. (2007). Mothers' knowledge of young child development in a developing country. *Child: Care, Health and Development*, 33(6), 728–737.
- Fogarty, B., & Lofland, K. (2015). Curriculum materials and programs for individuals on the autism spectrum. Indiana University Bloomington. Available from: <http://www.iidc.indiana.edu/pages/curriculum-materials-and-programs-for-individuals-on-the-autism-spectrum>. Accessed 22 March 2016.
- Frederickson, N., & Cline, T. (2002). *Special educational needs, inclusion, and diversity: A textbook* (Vol. viii, 520 pp.). Buckingham/Philadelphia: Open University Press.
- Hecht E, Reynolds M, Agosta J, Mc Ginley K. (2011). Building an agenda for supporting families with a member with intellectual and developmental disabilities: Report of the Wingspread Conference on building a family support agenda, March 6–8, Racine, Wisconsin: Johnson Foundation, 2011. Available from: <http://issuu.com/supportstofamilies/docs/wingspreadreport-2012-rev-52912-com/11?e=11660291/7532630>.
- HINARI. (2016). HINARI access to research in Health Programme 2016. Available from: <http://www.who.int/hinari/en/> Accessed 2 April 2016.
- Mariga, L., McConkey, R., & Myezwa, H. (2014). *Inclusive education in low-income countries: A resource book for teacher educators, parent trainers and community development workers*. Cape Town: Atlas Alliance and Disability Innovations Africa.
- Munir, K. M. (2016). The co-occurrence of mental disorders in children and adolescents with intellectual disability/intellectual developmental disorder. *Current Opinion in Psychiatry*, 29(2), 95–102.
- Munir KM, Lavelle TA, Helm DT, Thompson D, Prestt J, Azeem MW (2016). Autism: A global framework for action. Doha: World Innovation Summit for Health. <http://www.wish-qatar.org/wish-2016/forum-reports/forum-reports>
- National Autism Center. (2009). National standards project: May Institute. Available from: <http://www.nationalautismcenter.org>
- New England Genetics Collaborative (NEGC). Resources for families (2016). Available from: <https://www.negenetics.org/resources/families>
- Oono, I. P., Honey, E. J., & McConachie, H. (2013). Parent-mediated early intervention for young children with autism spectrum disorders (ASD). *Cochrane Database of Systematic Reviews*, 4, CD009774.
- Parish, S. L., Rose, R. A., Andrews, M., Grinstein-Weiss, M., Richman, E. L., & Dababnah S. (2009) Material hardship in US families raising children with disabilities: Research summary and policy implications. UNC School of Social Work.
- Patel, C., Kielling, C., Maulik, P. K., & Divan, G. (2013). Improving access to care for children with mental disorders: A global perspective. *Archives of Disease in Childhood*, 98, 323–327.
- Population Reference Bureau. (2015). World population data sheet. Washington, DC. Available from: http://www.prb.org/pdf15/2015-world-population-data-sheet_eng.pdf
- Rahman, A., Divan, G., Hamdani, S. U., Vajaratkar, V., Taylor, C., Leadbitter, K., Aldred, C., Minhas, A., Cardozo, P., Emsley, R., Patel, V., & Green, J. (2016). Effectiveness of the parent-mediated intervention for children with autism spectrum disorder in south Asia in India and Pakistan (PASS): A randomised controlled trial. *Lancet Psychiatry*, 3(2), 128–136.
- Richmond, J. B., & Kotelchuck, M. (1983). Political influences: Rethinking national health policy. In C. Mc Guire, R. Foley, A. Gorr, et al. (Eds.), *Handbook of health professions education: Theory, research and practice* (pp. 386–404). San Francisco: Jossey-Bass.
- Seltzer, M. M., Greenberg, J. S., Floyd, F. J., Pettee, Y., & Hong, J. (2001). Life course impacts of parenting a child with a disability. *American Journal of Mental Retardation*, 106(3), 265–286.
- Sharan, P., Levav, I., Olifson, S., de Francisco, A., Saxena, S. (2007). Research capacity for mental health

in low- and middle-income countries: Results of a mapping project. Geneva, Switzerland: World Health Organization & Global Forum for Health Research. Available from: http://www.who.int/mental_health/MHRC_FullText.pdf.

Thornicroft, G., Cooper, S., Bortel, T. V., Kakuma, R., & Lund, C. (2012). Capacity building in global mental health research. *Harvard Review of Psychiatry*, 20(1), 13–24.

UNESCO. (2013). Equal right, equal opportunity – Inclusive education for all. Available from: http://www.unesco.org/new/en/education/themes/leading-the-international-agenda/right-to-education/single-view/news/equal_right_equal_opportunity_inclusive_education_for_all/? Accessed 22 March 2016

United Nations. (2007). Third committee calls on assembly to designate 2 April World Autism Day, UN General Assembly, 1 November 2007. Available from: <http://www.un.org/press/en/2007/gashc3899.doc.htm>

United Nations. UN Convention on the Rights of Persons with Disabilities (2008). Available from: <http://www.un.org/disabilities/convention/conventionfull.shtml>

Viergever, R. F., Olifson, S., Ghaffar, A., & Terry, R. F. (2010). A checklist for health research priority setting: Nine common themes of good practice. *Health research policy and systems*. *BioMed Central*, 8, 36.

World Health Organization (WHO). (1994). *International classification of diseases, 10th edition (ICD-10)*. Geneva: WHO.

World Health Organization (WHO). (2013). Meeting report on autism spectrum disorders and other developmental disorders: From raising awareness to building capacity. Geneva, Switzerland. Available from: http://apps.who.int/iris/bitstream/10665/103312/1/9789241506618_eng.pdf

Zwaigenbaum, L., Bauman, M. L., Choueiri, R., Kasari, C., Carter, A., Granpeesheh, D., et al. (2015). Early intervention for children with autism spectrum disorder under 3 years of age: Recommendations for practice and research. *Pediatrics*, 136(Suppl 1), S60–S81.

Developmental Apraxia

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Synonyms

Childhood apraxia of speech (CAS)

Short Description or Definition

A motor speech disorder characterized by difficulty acquiring speech, inconsistent sound errors, and groping behaviors during speech in the absence of weakness or paralysis. Symptoms are similar to verbal apraxia in adults; however, the underlying motor impairment significantly impacts phonological development (Maassen 2002). Hallmark characteristics consistent with childhood apraxia of speech include vowel errors or distortions, highly inconsistent speech errors, and inappropriate prosody.

References and Reading

American Speech-Language-Hearing Association. (2007). *Childhood Apraxia of Speech* [Technical Report]. <https://doi.org/10.1044/policy.TR2007-00278.html>.

Aram, D., & Nation, J. (1982). *Child language disorders*. St. Louis: C.V. Mosby.

Crary, M. A. (1984). A neurolinguistic perspective on developmental verbal apraxia. *Communicative Disorders*, 9, 33–49.

Maassen, B. (2002). Issues contrasting adult acquired versus developmental apraxia of speech. *Seminars in Speech and Language*, 23, 257–266.

Morley, M. (1965). *The development and disorders of speech in childhood*. Baltimore: Williams & Wilkins.

Morley, M., Court, D., & Miller, H. (1954). Developmental dysarthria. *British Medical Journal*, 1, 8–10.

Palmer, M., Wuth, C., & Kincheloe, J. (1964). The incidence of lingual apraxia and agnosia in “functional” disorders of articulation. *Cerebral Palsy Review*, 25, 7–9.

Rosenbek, J., & Wertz, R. T. (1972). A review of 50 cases of developmental apraxia of speech. *Language, Speech and Hearing Services in Schools*, 3, 23–33.

Williams, R., Ingham, R., & Rosenthal, J. (1981). A further analysis for developmental apraxia of speech in children with defective articulation. *Journal of Speech and Hearing Research*, 24, 496–505.

Yoss, R., & Darley, F. (1974). Developmental apraxia of speech in children with defective articulations. *Journal of Speech and Hearing Research*, 17, 339–416.

Developmental Apraxia of Speech (DAS)

► Verbal Apraxia

Developmental Change

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Definition

Developmental change is the process of change that occurs in human beings throughout development.

Gene expression, brain function, cognitive processes, behavior, and environmental factors all involve multiple cross-level interactions, and all are characterized by dynamic developmental change over time. The study of any neurodevelopmental disorder, be it autism spectrum disorders (ASDs) or those of known genetic origin like Down syndrome (DS), Williams syndrome (WS), fragile X syndrome (FXS), or velocardiofacial syndrome (VCFS), must focus on full developmental trajectories from infancy to adulthood, examining how domains interact differently over time.

Developmental Change at the Genetic Level

Many studies map specific genes to specific behaviors, but rare are those which take account of changing gene expression over time. Yet, if a gene is expressed widely initially and becomes increasingly confined to certain brain regions, or if a gene is expressed much more during learning but less during subsequent behavior, then the mapping from gene to behavior will change.

Developmental Change at the Neural Level

The brain is not static; it changes significantly after birth in terms of structure and function. Functionally, one often witnesses the child brain initially processing inputs bilaterally. With development, however, neural networks become

increasingly specialized and localized such that, for example, face processing starts out bilaterally and becomes predominantly right lateralized, over developmental time, in a network including the fusiform gyrus. Likewise, the processing of certain aspects of language, for example, the use of, say, articles, starts out bilaterally but becomes increasingly left lateralized. By contrast, in some neurodevelopmental disorders, this progressive fine-tuning of specialization and localization of function fails to occur, and processing continues to be bilateral, even when the relevant overt behavior is quite proficient.

Developmental Change at the Cognitive Level

In the study of neurodevelopmental disorders, it is critical to differentiate between identical overt behavioral scores and the underlying cognitive processes that sustain them. For example, face processing may be proficient in a disorder, with scores “in the normal range,” but the underlying cognitive processes rely on featural analyses, whereas in the typically developing child, processing has moved from featural to configural processing over developmental time.

Developmental Change at the Environmental Level

The environment is not static either. In all neurodevelopmental disorders, parents respond to the subtle differences in their atypical offspring, and thus, the dynamics of parent-child interaction change over time. For example, when learning language, the parents of typically developing children tend to let their children temporarily make overgeneralizations (e.g., “dog” for all animals), whereas parents of atypically developing children tend to correct immediately in the fear, perhaps, that they otherwise may never learn the correct term. However, overgeneralization often helps the development of categories (e.g., “animal”), and subtle differences in the ways in which the environment responds to the atypical child may give

rise to the learning of individual exemplars rather than categories.

In conclusion, developmental changes must be taken into account at every level of analysis.

See Also

- [Developmental Delay](#)
- [Developmental Milestones](#)

References and Reading

- Johnson, M. H. (2011). Interactive specialization: A domain-general framework for human functional brain development? *Developmental Cognitive Neuroscience*, 1(1), 7–21.
- Karmiloff-Smith, A. (2009). Nativism versus neuroconstructivism: Rethinking the study of developmental disorders. Special issue on the interplay of biology and environment. *Developmental Psychology*, 45(1), 56–63.
- Karmiloff-Smith, A. (2010). Neuroimaging of the developing brain: Taking “developing” seriously. *Human Brain Mapping*, 31(6), 934–941.
- Karmiloff-Smith, A., Thomas, M., Annaz, D., Humphreys, K., Ewing, S., Brace, N., et al. (2004). Exploring the Williams syndrome face processing debate: The importance of building developmental trajectories. *Journal of Child Psychology and Psychiatry*, 45(7), 1258–1274.

Developmental Continuum (Principles of TEACCH)

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Synonyms

[Developmental shifts across lifespan](#)

Definition

The developmental continuum is a dynamic process characterized by milestones and challenges

that occur from infancy throughout adulthood. The interaction of typical developmental issues with ASD is complicated. Milestones may be reached earlier or later than typical peers and at an atypical rate or order; across domains, a scattered skill profile may also be noted. The challenges that an individual with ASD faces may mirror those of his or her typical peers (e.g., entering school, coping with bullying, developing self-image, managing life changes) but may be exaggerated or occur at different points in development. In addition, it is now recognized that ASD does not protect one against other possible diagnoses or challenges, such as anxiety, depression, substance abuse, and other co-occurring disorders. Rather, the interaction of ASD with the typical developmental processes may exacerbate or trigger these issues. Therefore, an initial evaluation should be not solely for diagnosis but also for the comprehensive evaluation of cognitive, adaptive, behavioral, emotional, and psychiatric functioning and should be updated over time.

There are currently many evidence-based treatment practices for individuals with ASD. However, the selection of appropriate treatments is complex, leaving families without clear direction in seeking and selecting services. Comprehensive evaluation identifies the current skill level and needs of an individual with ASD and thus facilitates the development of specific goals. In addition, as the individual needs of the person with ASD changes, the appropriate treatment may also change, even throughout adulthood. Updated evaluations reveal the trajectory and pattern of skill development and the efficacy of any treatments being used and allow for selection of new treatments as appropriate. Families' sense of urgency to intervene may vary across the developmental continuum, with life transitions triggering a recognition of increased need.

Like individuals, families are dynamic and follow a general developmental pathway. This is true of families with typical children as well as those with ASD though, as with individual development, the challenges experienced by the latter are likely to be exacerbated and the changes less linear. The particular needs of a family with a child with ASD tend to correspond to the age of

the child. When the child is an infant/toddler/preschooler, families are recognizing developmental differences and managing the impact of these on the family, then dealing with diagnosis of ASD and associated grief. In the middle childhood years, families focus on school concerns, adaptive skills, and issues related to puberty. In adolescence and adulthood, common themes center on collegiate, vocational, and/or residential preparation as well as self-advocacy, guardianship, and interpersonal supports. This pattern of development is not universal, however. Just as professionals must fully assess an individual to ascertain skill level before implementing intervention, they must understand the current family interactions, challenges, needs, and foci so as to have a greater impact on that family. Current recognition of the importance of adaptive behavior provides an opportunity for families to increase positive outcomes through intervention and teaching in the home and community to facilitate increases in independence across the lifespan.

The developmental continuum contributes to a founding principle of the UNC TEACCH Autism Program, which is that of family involvement in service delivery. In working with clients, TEACCH promotes the view that the specific needs of the individual with ASD can be best met by recognizing the family's needs and by working simultaneously with caregivers to address them. TEACCH has always seen parents and caregivers as the experts, advocates, and teachers for their children and the professional's role as one of the facilitators in helping the individual with ASD to maximize their level of independence and in helping family members gain additional tools to be as effective as possible in their roles. There is a current recognition that aging individuals with ASD continue to need support that may differ from that at younger ages, as their health and financial needs increase and their family support circle may decrease. Across the developmental continuum, the needs of individuals, families, and support systems must be the focus of assessment and intervention in order to most effectively serve individuals with ASD.

See Also

- [Clinical Assessment](#)
- [Informal Assessment](#)

References and Reading

- Fiske, K. E., Pepa, L., & Harris, S. L. (2014). Supporting parents, siblings, and grandparents of individuals with autism spectrum disorders. In F. R. Volkmar, S. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders: volume 2, Assessment, interventions, and policy* (4th ed., pp. 932–948). Hoboken: Wiley.
- Marcus, L. M., Kuncie, L. J., & Schopler, E. (2005). Working with families. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 1055–1086). Hoboken: Wiley.
- Mezibov, G. B., & Shea, V. (2011). Evidence-based practices and autism. *Autism*, 15(1), 114–133.
- Van Bourgondien, M. E., & Coonrod, E. (2013). TEACCH: An intervention approach for children and adults with autism spectrum disorders and their families. In S. Goldstein & J. A. Naglieri (Eds.), *Interventions for autism spectrum disorders: Translating science into practice* (pp. 75–105). New York: Springer Science and Business.
- Van Bourgondien, M. E., Dawkins, T., & Marcus, L. (2014). Families of adults with autism spectrum disorders. In F. R. Volkmar, B. Reichow, & J. C. McPartland (Eds.), *Adolescents and adults with autism spectrum disorders* (pp. 15–40). New York: Springer Science and Business.

Developmental Coordination Disorder

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Synonyms

[Cerebral palsy](#); [Dyspraxia](#)

Short Description or Definition

Children who present marked difficulties with motor movements have been known since ancient times. Terms like “cerebral palsy” have been used in the past particularly to refer to situations where these problems appear to relate to some specific process, e.g., birth trauma. Although a medical etiology is sometimes seen, this is less likely in cases that are less severe. The term “developmental coordination disorder” is currently used.

Categorization

In DSM-IV, this condition is defined based on the presence of motor difficulties greater than expected (given age or developmental level) and not due to some other conditions like autism. Motor difficulties are sometimes seen with other developmental problems, e.g., language or learning disorders. Interestingly some work has been done on the constellation of social-emotional difficulties, motor, and attentional problems (the DAMP syndrome; see Ehlers et al. 1997).

Epidemiology

The condition may be seen in up to 6% of children of school age. Boys are more frequently diagnosed than girls (although various factors may make it less likely that subtle difficulties in girls lead to lower rates of referral).

Natural History, Prognostic Factors, and Outcomes

Various factors determine outcome. Often the ultimate outcome is best when motor difficulties are mild and isolated (i.e., not associated with other developmental problems). Sometimes motor delays can lead to other problems such as social isolation and, in turn, to anxiety and mood problems.

Clinical Expression and Pathophysiology

Motor skill difficulties can arise because of a host of factors. These range from problems during pregnancy in the mother, birth trauma, and perinatal difficulties (e.g., hypoxia or severe prematurity). Speech-language issues can be noted reflecting, in some cases, oral motor difficulties. Often a combination of some degree of developmental immaturity and a more specific motor vulnerability is involved.

Evaluation and Differential Diagnosis

Neurological and specialized occupational and physical therapy evaluations are indicated if motor difficulties are severe and/or significant. The presence of unusual movements, problems with hyper- or hypotonia, and of specific neurological symptoms can also prompt referral. Various tests of gross and fine motor skills as well as visual motor integration and of dexterity can be administered. These help to document areas of difficulty and establish baselines for intervention. In some cases, use of auxiliary aids/devices may be helpful, e.g., in children with Asperger’s disorder who have problems with cursive handwriting, use of a laptop to teach keyboarding skills can be indicated.

Treatment

Rehabilitative approaches are helpful. Both occupational and physical therapy approaches can be used to address fine and gross motor problems. Within schools, adaptive physical education can also be helpful.

See Also

- ▶ [DAMP Syndrome](#)
- ▶ [Language Disorder](#)
- ▶ [Occupational Therapy \(OT\)](#)
- ▶ [Physical Therapy](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author. Text revision.
- Ehlers, S., Nyden, A., Gillberg, C., Sandberg, A. D., Dahlgren, S. O., Hjelmquist, E., et al. (1997). Asperger syndrome, autism and attention disorders: A comparative study of the cognitive profiles of 120 children. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, 38(2), 207–217.
- Gillberg, C., & Kadesjo, B. (2003). Why bother about clumsiness? The implications of having developmental coordination disorder (DCD). *Neural Plasticity*, 10(1–2), 59–68.
- Gillberg, C., & Kadesjoe, B. (2000). Attention-deficit/hyperactivity disorder and developmental coordination disorder. In T. E. Brown (Ed.), *Attention-deficit disorders and comorbidities in children, adolescents, and adults* (pp. 393–406). Washington, DC: American Psychiatric Publishing.
- Smyth, M. M., & Mason, U. C. (1997). Planning and execution of action in children with and without developmental coordination disorder. *Journal of Child Psychology and Psychiatry*, 38(8), 1023–1037.
- Volkmar, F. R., & Martin, A. (2011). *Essentials of child and adolescent psychiatry*. Philadelphia: Lippincott, Williams, and Wilkins.
- Wann, J. (2007). Current approaches to intervention in children with developmental coordination disorder. *Developmental Medicine and Child Neurology*, 49(6), 405.

Developmental Delay

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Definition

Developmental delay is a significant lag in reaching the typical childhood milestones in the areas of language; cognition; social, emotional, adaptive functioning; and motor development. Each milestone is reached within a certain number of months based on research of typically developing children. When a child does not reach one or more of the milestones during the expected time frame, then he or she may be suspected of having a developmental delay.

In the context of public education, the IDEA definition of developmental delay is only inclusive of children aged three to nine and lists physical development, cognitive development, communication development, social or emotional development, and adaptive development as the areas to assess for a suspected disability. Children with autism often display delays in several of these areas, which may be the first warning signs that lead to further assessment and evaluation. Assessing developmental delays should be a component of diagnosing autism as the key deficits that characterize the disorder are directly linked to skills typically learned during natural developmental cycles. Skills that may be deficient in early development for individuals with autism include areas such as basic purposeful communication, initiating social interactions, and imitating functional use of objects or toys. Developmental delays may result in gaps in skill acquisition and/or performance and create widely varying strengths and weaknesses in some children.

Evaluations used to assess developmental delays vary among practitioners and typically include a measure of adaptive functioning with assessments such as the Vineland Adaptive Behavior Scales. This rating scale can be used to document delays in social and communicative development in individuals with autism. In addition, there are a number of motor assessments available, tests to measure cognitive levels, and specific communication assessments.

See Also

- [Developmental Milestones](#)
- [Intellectual Disability](#)

References and Reading

- Anderson, D. K., Lord, C., Risi, S., DiLavore, P. S., Shulman, C., Thurm, A., et al. (2007). Patterns of growth in verbal abilities among children with autism spectrum disorder. *Journal of Consulting and Clinical Psychology*, 75(4), 594–604.
- Gillham, J. E., Carter, A. S., Volkmar, F. R., & Sparrow, S. S. (2000). Toward a developmental operational

- definition of autism. *Journal of Autism and Developmental Disorders*, 30(4), 269.
- Gray, K. M., Tonge, B. J., Sweeney, D. J., & Einfeld, S. L. (2008). Screening for autism in young children with developmental delay: An evaluation of the developmental behaviour checklist–early screen. *Journal of Autism and Developmental Disorders*, 38(6), 1003–1010.
- Mayes, S., & Calhoun, S. (2003). Ability profiles in children with autism: Influence of age and IQ. *Autism: The International Journal of Research & Practice*, 7(1), 65–80.
- Openden, D., Whalen, C., Cernich, S., & Vaupel, M. (2009). Generalization and autism spectrum disorders. In C. Whalen (Ed.), *Real life, real progress for children with autism spectrum disorders* (pp. 1–18). Baltimore: Brookes.
- Provost, B., Lopez, B. R., & Heimerl, S. (2007). A comparison of motor delays in young children: Autism spectrum disorder, developmental delay, and developmental concerns. *Journal of Autism and Developmental Disorders*, 37(2), 321–328.
- Weiss, M. J., & LaRue, R. H. (2009). Enhancing generalization of skills taught through discrete trial instruction. In C. Whalen (Ed.), *Real life, real progress for children with autism spectrum disorders* (pp. 41–56). Baltimore: Brookes.

Developmental Disabilities

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Synonyms

Academic disability; Specific learning disability

Definition

Learning disability is not used here to refer to overall intellectual handicap (i.e., “mental retardation”).

- Developmental disability refers to unexpected delay or deficiency apparently healthy young children experience in the acquisition of a learned cognitive/intellectual skill (as opposed to a sensory-motor skill) despite

overall intellectual competence, attention and motivation, lack of auditory or visual handicap, and sufficient exposure to appropriate models and educational opportunity in an adequately supportive and nurturing environment.

- Developmental disabilities are extremely prevalent; they are dimensionally defined with fuzzy borders even though they denote atypical development of particular brain circuitries.
- They are both genetically and environmentally influenced.

Major Types

1. *Developmental language disorder (specific language impairment (SLI), dysphasia)* – in affected infants, the disorder presents as variably delayed/impoverished expressive language. There are three main clinical types, each with subtypes:

- a. *Expressive type*: impaired speech production and articulation (phonology) with adequate comprehension. Prognosis generally fairly good, except in the most severe subtype – verbal dyspraxia (not to be confused with oromotor disability, a deficit in motor control of the speech musculature).
- b. *Mixed receptive/expressive type*: comprehension equal to or somewhat better than expression. Phonology, grammar, and vocabulary affected. Prognosis variable, often the harbinger of dyslexia, and poor when phonologic decoding is severely defective.
- c. *Mainly receptive type*: impaired comprehension of discourse. Often overlooked when speech articulation, grammar, and vocabulary are spared. Particularly frequent but not exclusively so in verbal children on the autism spectrum.

Note: *Language disorders in children on the autism spectrum (ASD)* – Pragmatics, i.e., the communicative/conversational use of language, universally, characteristically, and permanently impaired. The prevalence of types of language disorders in ASD children differs from that of

dysphasic children: some have mixed expressive/receptive disorders; very few have expressive disorders with adequate comprehension; most verbal children have receptive disorders with telltale echolalia, use of scripts, incessant questioning, perseveration on self-selected topics, answering questions off topic, and aberrant prosody.

2. *Reading disability (dyslexia)* – difficulty learning the alphabetical code of written language at school age. Dyslexia is often the residual of a developmental language disorder with difficulty making fine auditory discriminations between speech sounds. Most dyslexic individuals eventually learn to read more or less efficiently, but retain difficulty reading pronounceable non-words and, often, poor spelling (*dysorthographia*). Less frequent causes include visual discrimination difficulties or sequencing problems implicating deficient working memory.
3. *Mathematical disability (dyscalculia)* – difficulty with mental or written arithmetic, geometry, word problems, or other mathematical operations. Identification of its cause requires detailed neuropsychologic investigation. Attention deficit disorder contributes to dyscalculia and complex arithmetical operations. Visuospatial problems impair not only geometry but also written arithmetic.
4. *Dysgraphia* – poor handwriting, associated or not with *dysorthographia*. Either due to an overt or subtle motor deficit or difficulty in learning complex motor skills (*dyspraxia*). A large sloppy handwriting (dyspraxia) with excellent spelling (superior rote memory) often characterizes ASD.
5. *Others* – tone deafness, grossly deficient ability to draw or classify can be considered learning disabilities when they interfere with children’s acquisition of required skills.

References and Reading

Fletcher, J. M. (2009). Dyslexia: The evolution of a scientific concept. *Journal of the International Neuropsychology Society*, 15, 501–508.

Landerl, K., Fussenegger, B., Moll, K., & Willburger, E. (2009). Dyslexia and dyscalculia: Two learning disorders with different cognitive profiles. *Journal of Experimental Child Psychology*, 103, 309–324.

Rapin, I., Dunn, M., & Allen, D. A. (2003). Developmental language disorder. In S. J. Segalowitz & I. Rapin (Eds.), *Part II: Child neuropsychology* (Vol. 8, 2nd ed., pp. 593–630). Amsterdam: Elsevier.

Shaywitz, B. A., Lyon, G. R., & Shaywitz, S. E. (2006). The role of functional magnetic resonance imaging in understanding reading and dyslexia. *Developmental Neuropsychology*, 30, 613–632.

Vernes, S. C., Newbury, D. F., Abrahams, B. S., Winchester, L., Nicod, J., Groszer, M., et al. (2008). A functional genetic link between distinct developmental language disorders. *The New England Journal of Medicine*, 359, 2337–2345.

Developmental Disabilities – Children’s Global Assessment Scale (DD-CGAS)

► [Children’s Global Assessment Scale](#)

Developmental Disability (Ontario)

► [Mental Retardation](#)

Developmental Disability (Ontario), Mental Retardation (Former Term)

► [Intellectual Disability](#)

Developmental Dyscalculia

► [Dyscalculia](#)

Developmental Dysphasia

► [Childhood Aphasia](#)

Developmental Dyspraxia

► Verbal Apraxia

Developmental Intervention Model

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Definition

The developmental approach to intervention draws upon the knowledge of typical development to design treatment objectives for children with autism spectrum disorder (ASD). Child development research informs the developmental processes that determine goals, measure change, and guide treatment practices. The main pillars of this approach include the selection of developmentally appropriate targets, individualized instruction by focusing on child preferences and interests, and the incorporation of family needs, values, and preferences into intervention objectives. A number of developmental models exist in the early intervention literature of children with ASD including the Floortime and Developmental Individual-difference Relationship models (DIR; Greenspan and Weider 1999), Relationship Development Intervention (RDI; Gutstein and Sheely 2002), Hanen Centre programs (Coulter and Gallagher 2001; Sussman 1999), Preschool Autism Communication Trial (PACT; Green et al. 2010), and Social Communication, Emotional Regulation and Transactional Support (SCERTS; Prizant et al. 2006) models, to name a few. Contemporary behavioral approaches to early intervention for children with ASD, often referred to as Naturalistic Developmental Behavioral Interventions (NDBI; Schreibman et al. 2015), also emphasize developmental objectives and to

varying degrees utilize developmental principles (e.g., Joint Attention Symbolic Play Engagement and Regulation (JASPER), Kasari et al. 2006; Pivotal Response Treatment (PRT), Koegel and Koegel 2006; Incidental teaching, McGee et al. 1999; Reciprocal Imitation Training (RIT; Ingersoll 2010); the Denver Model, Rogers et al. 2000; and Early Start Denver Model (ESDM), Rogers and Dawson 2010).

Historical Background

This treatment approach has its origins in our understanding of how children learn through typical daily social interactions. Teaching during naturalistic, play-based, and developmentally appropriate activities has a long history in the typical early childhood literature to ensure motivation, cooperation, and engagement (Fein and Rivkin 1986; Copple and Bredekamp 2009).

In recent years, these principles have also been applied to children with developmental disorders, such as ASD. Most commonly these interventions focus on the core domain of social communication and include specific emphasis on joint attention, social engagement, and early expressive language (e.g., PACT, SCERTS, RDI, JASPER, Hanen). Several comprehensive treatment approaches have also adopted a developmental framework (e.g., Denver model, ESDM, PRT).

Rationale or Underlying Theory

The underlining theory behind developmental treatment approaches is that each child is an individual with corresponding strengths and weaknesses. Not every child has the same profile of development, especially children with a heterogeneous disorder such as ASD. The developmental approach to intervention strives to tailor curriculum to the needs of each child and provide opportunities for learning skills that are appropriate to the child's current level of functioning as well as scaffolding instruction to more developmentally complex skills within the child's milieu. A developmental framework for early social-

communication intervention pulls from the extensive literature on both incidental teaching (McGee et al. 1985) and the importance of linguistic input to language acquisition (Hoff-Ginsberg and Shatz 1982). This teaching approach is child-centered and child-directed and involves such strategies as following the child's motivations and interests, offering choices within activities, responding to child initiations, and expanding on a child's verbal and nonverbal communicative bids (Prizant et al. 2006; Wilcox and Shannon 1998). Unlike traditional behavioral approaches that are adult-facilitated and stick to a strict hierarchy of program objectives, developmental models strive to follow the child's interests in a naturalistic learning environment. The hope is that by embedding learning opportunities within highly motivating and natural contexts, children will be more apt to participate and the total amount of intervention or dosage each day will increase. An underlining goal of developmental approaches is to foster a deeper connection between the child and a social partner (often the parent) that results in meaningful teaching opportunities.

Goals and Objectives

Several common goals exist across early developmental interventions for ASD. These goals include the emphasis on individualized treatment and targeting developmentally appropriate skills. A majority of developmental interventions emphasize social-communication outcomes for children with ASD, with a special importance placed on children becoming effective communicators. Some programs, such as the Walden preschool, also place an emphasis on early interactions with peers. Additionally, there is an emphasis on the child's emotion regulation abilities and the provision on developmentally appropriate supports for student learning. The SCERTS model emphasizes the need to help the child regulate arousal as a foundation to early intervention. Other developmental models also

believe that helping the child reach the optimal level of arousal is an early developmental skill necessary for successful learning (e.g., DIR, JASPER, ESDM). Another common theme is to provide learning opportunities within the natural social context of daily activities. This includes embedding intervention objectives within play-based activities and daily routines of living and providing support and training for parents to generalize treatment objectives to the home environment.

Treatment Participants

Developmental approaches are typically applied during the early intervention period for children with ASD. Some interventions have been applied to children as young as 12 months of age (e.g., ESDM and JASPER). Typically, restrictions regarding participation are not made based upon child developmental characteristics, such as cognitive, language, and adaptive functioning. With the increasing emphasis on early detection and intervention, it follows suit that these models would support the development of the youngest children affected by the disorder.

Some attempts have also been made to apply developmental models of intervention to older children and adults. The JASPER intervention is a modular treatment that has been tested for efficacy in a wide range of children from toddlers to older nonverbal and school-aged children. Principles of PRT are used across the lifespan. Overall, participants are typically in the preschool range and so more research needs to be done to assure that children and adults across the lifespan can benefit from developmental treatment approaches.

More recently there has also been an increased focus on serving children from diverse and under-represented backgrounds in some development intervention models such as JASPER and the Parent-Mediated Intervention for Autism Spectrum Disorders in South Asia (PASS), a modified version of PACT (Rahman et al. 2016).

Treatment Procedures

Developmental interventions use naturalistic teaching strategies in a variety of settings. The goal is for treatment to be embedded within everyday social experiences; thus, many of the developmental intervention models are play-based. Teaching objectives are embedded within these highly motivating play activities. Research in typical development has shown that children who are allowed to follow their preferred attentional focus see direct benefits on communicative growth (Akhtar et al. 1991; Tomasello and Farrar 1986). Thus, it is believed that children with ASD will also benefit from having more autonomy in the choice of activities and more opportunities to lead the interaction.

Developmental interventions incorporate the use of a wide range of treatment settings including clinics, schools, and the home. Many of these treatments are adapted for use across multiple settings. For example, the ESDM can be applied in center-based preschools, inclusive preschools, and the home environment. PRT has been applied and has empirical support for its use across these settings. JASPER has been validated in randomized controlled trials across a number of settings including clinics, homes, inclusive and center-based preschools, and community settings.

Instruction is also delivered using a variety of methods. Many developmental models will have a portion of the instruction delivered in a 1:1 teaching model with a trained therapist and the child (e.g., ESDM, PRT, JASPER, PACT, SCERTS) and also group settings (e.g., Denver Model, JASPER). Most recent interventions involving parents utilize to some degree what is known as a parent-mediated model. In this model, the parent and child are interacting together with a trained clinician who serves as an active “coach” for these social interactions so that the parent receives immediate feedback.

Efficacy Information

Several developmental early intervention programs have been tested for efficacy. In recent

years, there has been a rapid increase in the methodological quality of interventions testing developmental approaches and there is promising evidence of improvement using these models. One frequently studied approach is the Denver Model and ESDM by Rogers and colleagues. To date, they have numerous peer-reviewed papers on the efficacy of this intervention. The early work using this model utilized a within-subject pre-/postdesign, which as a method may not be adequate for determining effective treatments. Later work involved some quasi and true experimental designs including one randomized controlled clinical trial (RCT) of ESDM, which suggests evidence of improvement in the domains of cognition (DQ), language, and adaptive functioning (Dawson et al. 2010). A more recent RCT of a parent-mediated ESDM approach found no treatment effect on parent and child outcomes (Rogers et al. 2012).

Another treatment approach that has gained scientific support is PRT. Utilizing single-subject methodology, PRT has been documented to improve social skills, disruptive behaviors, responsivity, language, and other social behaviors (e.g., Koegel and Frea 1993; Koegel et al. 1992a, b). Recent RCTs of PRT have been conducted finding increased frequency of verbal utterances and mean length of utterances (Hardan et al. 2015; Mohammadzaheri et al. 2014). One study compared PRT to the Picture Exchange Communication System (PECS) and showed no treatment differences, but both groups increased over time on parent-report and standardized measures of expressive language (Schreibman and Stahmer 2014).

Studies by Kasari and colleagues have shown effectiveness for JASPER, a developmental social-communication intervention for toddlers and preschoolers with ASD. In several RCT trials, children randomly assigned to interventions targeting core deficits of ASD including joint attention and symbolic play made significant gains in joint attention initiations and symbolic play, respectively (Kasari et al. 2006), and also both made significant gains in expressive

language at a follow-up visit 1 and 5 years later compared to participants in a control condition (Kasari et al. 2008, 2012). Similarly, in several RCT designs of toddlers and preschoolers with ASD and their caregivers using a parent-mediated approach, children improved in their joint attention, play, and joint engagement with their caregivers (Kasari et al. 2010, 2014, 2015). JASPER has also been tested in a number of deployment trials where the intervention was carried out by teachers and community providers, not trained interventionists, but showed comparable effects to the clinic based trials (Chang et al. 2016; Kaale et al. 2012; Shire et al. 2016).

SCERTS has almost 30 years of clinical practice, and recent research into its efficacy has been conducted through the Early Social Interactions Project (Wetherby et al. 2014). Effects were seen on children's social communication, receptive language, and adaptive functioning. Similarly, DIR/Floortime has recent evidence supporting its efficacy. Three RCTs have been conducted in preschool aged children with some effects seen on children's broad social communicative functioning, regulation, and autism symptomology (Casenhiser et al. 2013; Pajareya and Nopmaneejumruslers 2011; Solomon et al. 2014). The first two trials have some methodological issues that limit the interpretability of the findings, but the most recent trial found that children receiving the DIR/Floortime-based treatment saw improved diagnostic classification, but no effects were observed on standardized language or cognitive measures.

The PACT intervention has been tested in two recent RCTs, one which took place in a clinic setting (Green et al. 2010) and another in community settings in south Asia (Rahman et al. 2016). The clinic-based intervention found positive effects on children's initiations and shared attention, while the community study found effects only for initiations.

These studies and others like them illustrate the trend toward efficacious treatment in ASD research.

Currently, researchers are focused on identifying the moderators of interventions and the exploration of which treatments work best for different populations of children with ASD. There is a clear need for more research into this area to establish stricter guidelines for "best practices" in autism intervention. Although there is promising evidence for the efficacy of developmental approaches in early intervention for ASD, these developmental models need to be tested against other approaches of early intervention (e.g., DTT) to better answer the question of which treatments work best for the wide range of children affected with the disorder. A number of these comparative efficacy trials are currently underway.

Mechanisms of Change

In recent years, there has been a heightened focus on what are often referred to as the *active ingredients* of developmental interventions. Active ingredients are those factors within an intervention that are driving the treatment effect on a specific outcome. Attempts have been made to isolate the active ingredients of both PACT and JASPER. In the PACT trial, ADOS-g scores were mediated by parental synchrony and children's initiations (Pickles et al. 2015) and in JASPER one strategy, Mirrored Pacing, mediated the treatment effect on children's joint engagement (Gulsrud et al. 2015). Further efforts to understand the mechanisms of change of efficacious developmental interventions will continue as the evidence base grows.

Outcome Measurement

Many of the early developmental interventions target social communication in our youngest children affected with ASD. It follows suit that outcome measures for these interventions would tap into domains of social and communicative functioning. Broadly, there are two types of outcome measures to consider, those which are proximal

and distal to the intervention procedure. Proximal outcomes are directly targeted by the intervention and distal outcomes are downstream effects that are expected but not explicitly targeted. The JASPER and PACT interventions have proximal outcomes such as joint engagement and shared attention that are directly targeted in the interventions, and it is expected that these changes in proximal outcomes will lead to distal effects on expressive and receptive language. It is important that both proximal and distal outcomes are carefully selected. An intervention can have strong effects on proximal outcomes but show no effect on distal outcomes, or can show change across both. Important to note is that distal effects cannot be seen without first moving proximal outcomes. The importance of follow-up and long-term measurement is therefore a strong point of emphasis in developmental interventions although the cost of measuring data over time and issues of attrition often limit the feasibility of long-term follow-up periods.

The measures used in developmental interventions vary widely. They include standardized measures of language, cognitive ability, and autism symptomology (often distal outcomes) such as the Mullen Scales of Early Learning, the Reynell Developmental Language Scales (Reynell 1977), and the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2000). Parent reports include measures of language and adaptive functioning such as the MacArthur-Bates Communicative Development Inventory (Fenson et al. 1993) and Vineland Adaptive Behavior Scales (Sparrow et al. 2005). A number of interventions utilize idiosyncratic measures (often proximal outcomes) that code for specific behaviors targeted by the intervention such as engagement, play, and gesture use; these coding measures are usually in the context of unstructured or structured play interactions between adults and children. New standardized observational measures, such as the Brief Observation of Social Communication Change (BOSCC; Grzadzinski et al. 2016), which are more sensitive to subtle changes in

the often complex social communication behaviors of children, are also beginning to emerge.

Qualifications of Treatment Providers

Most developmental interventions require specific training to implement. Trained clinicians carry out the models described above with checks for fidelity to instruction standards occurring at regular intervals for several programs (e.g., PRT, ESDM, JASPER). Hanen requires speech and language therapists to implement the intervention. Many treatments also involve the training of parents to generalize program goals to the home setting. Some models also conduct trainings to extend instruction to teachers and community providers (e.g., PRT, JASPER, PACT).

References and Reading

- Akhtar, N., Dunham, F., & Dunham, P. J. (1991). Directive interactions and early vocabulary development: The role of joint attentional focus. *Journal of Child Language*, 18(01), 41–49.
- Casenhiser, D. M., Shanker, S. G., & Stieben, J. (2013). Learning through interaction in children with autism: Preliminary data from a social-communication-based intervention. *Autism*, 17(2), 220–241.
- Chang, Y. C., Shire, S. Y., Shih, W., Gelfand, C., & Kasari, C. (2016). Preschool deployment of evidence-based social communication intervention: JASPER in the classroom. *Journal of Autism and Developmental Disorders*, 46(6), 2211–2223.
- Copple, C., & Bredekamp, S. (2009). *Developmentally appropriate practice in early childhood programs serving children from birth through age 8*. Washington, DC: National Association for the Education of Young Children.
- Coulter, L., & Gallagher, C. (2001). Evaluation of the Hanen early childhood educators programme. *International Journal of Language & Communication Disorders*, 36, 264–269.
- Dawson, G., Rogers, S. J., Smith, M., Munson, J., & Winter, J. (2010). Randomized controlled trial of the early start Denver model: A relationship-based developmental and behavioral intervention for toddlers with autism spectrum disorders: Effects on IQ, adaptive behavior and autism diagnosis. *Pediatrics*, 125, 1–7.
- Fein, G., & Rivkin, M. (1986). *The young child at play: Reviews of research* (Vol. 4). Washington, DC:

- National Association for the Education of Young Children.
- Fenson, L., Dale, P. S., Reznick, J. S., Thai, D., Bates, E., Hartung, J. P., et al. (1993). *The MacArthur communicative development inventories: User's guide and technical manual*. San Diego: Singular Publishing Group.
- Green, J., Charman, T., McConachie, H., Aldred, C., Slonims, V., Howlin, P., . . . Barrett, B. (2010). Parent-mediated communication-focused treatment in children with autism (PACT): A randomized controlled trial. *The Lancet*, 375(9732), 2152–2160.
- Greenspan, S. I., & Weider, S. (1999). A functional developmental approach to autism spectrum disorders. *The Journal of the Association for Persons with Severe Handicaps*, 24(3), 147–161.
- Grzadzinski, R., Carr, T., Colombi, C., McGuire, K., Dufek, S., Pickles, A., & Lord, C. (2016). Measuring changes in social communication behaviors: Preliminary development of the Brief Observation of Social Communication Change (BOSCC). *Journal of Autism and Developmental Disorders*, 46(7), 2464–2479.
- Gulsrud, A. C., Hellemann, G., Shire, S., & Kasari, C. (2015). Isolating active ingredients in a parent-mediated social communication intervention for toddlers with autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 57(5), 606–613.
- Gutstein, S., & Sheely, R. (2002). *Relationship development intervention with young children: Social and emotional development activities for Asperger syndrome, autism, PDD, and NLD*. London: Jessica Kingsley.
- Hardan, A. Y., Gengoux, G. W., Berquist, K. L., Libove, R. A., Ardel, C. M., Phillips, J., . . . Minjarez, M. B. (2015). A randomized controlled trial of Pivotal Response Treatment Group for parents of children with autism. *Journal of Child Psychology and Psychiatry*, 56(8), 884–892.
- Hoff-Ginsberg, E., & Shatz, M. (1982). Linguistic input and the child's acquisition of language. *Psychological Bulletin*, 92(1), 3.
- Ingersoll, B. (2010). Brief report: Pilot randomized controlled trial of reciprocal imitation training for teaching elicited and spontaneous imitation to children with autism. *Journal of Autism and Developmental Disorders*, 40(9), 1154–1160.
- Kaale, A., Smith, L., & Sponheim, E. (2012). A randomized controlled trial of preschool-based joint attention intervention for children with autism. *Journal of Child Psychology and Psychiatry*, 53(1), 97–105.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry*, 47, 611–620.
- Kasari, C., Paparella, T., Freeman, S., & Jahromi, L. (2008). Language outcome in autism: Randomized comparison of joint attention and play interventions. *Journal of Consulting and Clinical Psychology*, 76, 125–137.
- Kasari, C., Gulsrud, A., Wong, C., Kwon, S., & Locke, J. (2010). Randomized controlled caregiver mediated joint engagement intervention for toddlers with autism. *Journal of Autism and Developmental Disorders*, 40, 1045–1056.
- Kasari, C., Gulsrud, A., Freeman, S., Paparella, T., & Hellemann, G. (2012). Longitudinal follow-up of children with autism receiving targeted interventions on joint attention and play. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(5), 487–495.
- Kasari, C., Lawton, K., Shih, W., Barker, T. V., Landa, R., Lord, C., . . . Senturk, D. (2014). Caregiver-mediated intervention for low-resourced preschoolers with autism: An RCT. *Pediatrics*, 134(1), e72–e79.
- Kasari, C., Gulsrud, A., Paparella, T., Hellemann, G., & Berry, K. (2015). Randomized comparative efficacy study of parent-mediated interventions for toddlers with autism. *Journal of Consulting and Clinical Psychology*, 83(3), 554.
- Koegel, R. L., & Frea, W. D. (1993). Treatment of social behavior in autism through the modification of pivotal social skills. *Journal of Applied Behavior Analysis*, 26, 369–377.
- Koegel, R. L., & Koegel, L. K. (2006). *Pivotal response treatments for autism: Communication, social, and academic development*. Baltimore: Paul Brooks.
- Koegel, L. K., Koegel, R. L., Hurley, C., & Frea, W. D. (1992a). Improving social skills and disruptive behaviors in children with autism through self-management. *Journal of Applied Behavior Analysis*, 25, 341–353.
- Koegel, R. L., Koegel, L. K., & Surratt, A. (1992b). Language intervention and disruptive behavior in preschool children with autism. *Journal of Autism and Developmental Disorders*, 18, 525–538.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Leventhal, B. L., DiLavore, P. C., et al. (2000). The autism diagnostic observation schedule – Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30(3), 205–223.
- McGee, G. G., Krantz, P. J., & McClannahan, L. E. (1985). The facilitative effects of incidental teaching on preposition use by autistic children. *Journal of Applied Behavior Analysis*, 18(1), 17–31.
- McGee, G. G., Morrier, M. J., & Daly, T. (1999). An incidental teaching approach to early intervention for toddlers with autism. *The Journal of the Association for Persons with Severe Handicaps*, 24, 133–146.
- Mohammadzaheri, F., Koegel, L. K., Rezaee, M., & Rafiee, S. M. (2014). A randomized clinical trial comparison between pivotal response treatment (PRT) and structured applied behavior analysis (ABA) intervention for children with autism. *Journal of Autism and Developmental Disorders*, 44(11), 2769–2777.
- Pajareya, K., & Nopmaneejumrulers, K. (2011). A pilot randomized controlled trial of DIR/Floortime™ parent

- training intervention for pre-school children with autistic spectrum disorders. *Autism*, 15(5), 563–577.
- Pickles, A., Harris, V., Green, J., Aldred, C., McConachie, H., Slonims, V., ... Charman, T. (2015). Treatment mechanism in the MRC preschool autism communication trial: Implications for study design and parent-focused therapy for children. *Journal of Child Psychology and Psychiatry*, 56(2), 162–170.
- Prizant, B. M., Wetherby, A. M., Rubin, E., Laurent, A. C., & Rydell, P. J. (2006). *The SCERTS model: A comprehensive educational approach for children with autism spectrum disorders*. Baltimore: Paul H. Brookes.
- Rahman, A., Divan, G., Hamdani, S. U., Vajaratkar, V., Taylor, C., Leadbitter, K., ... Patel, V. (2016). Effectiveness of the parent-mediated intervention for children with autism spectrum disorder in south Asia in India and Pakistan (PASS): A randomised controlled trial. *The Lancet Psychiatry*, 3(2), 128–136.
- Reynell, J. K. (1977). *Reynell developmental language scales*. Windsor: NFER.
- Rogers, S. J., & Dawson, G. (2010). *Early start Denver model for young children with autism: Promoting language, learning and engagement*. New York: Guilford.
- Rogers, S. J., Hall, T., Osaki, D., Reaven, J., & Herbison, J. (2000). The Denver model: A comprehensive, integrated educational approach to young children with autism and their families. In J. S. Handleman & S. L. Harris (Eds.), *Preschool education programs for children with autism* (2nd ed., pp. 95–133). Austin: Pro-Ed.
- Rogers, S. J., Estes, A., Lord, C., Vismara, L., Winter, J., Fitzpatrick, A., et al. (2012). Effects of a brief Early Start Denver Model (ESDM)-based parent intervention on toddlers at risk for autism spectrum disorders: A randomized controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(10), 1052–1065.
- Schreibman, L., & Stahmer, A. C. (2014). A randomized trial comparison of the effects of verbal and pictorial naturalistic communication strategies on spoken language for young children with autism. *Journal of Autism and Developmental Disorders*, 44(5), 1244–1251.
- Schreibman, L., Dawson, G., Stahmer, A. C., Landa, R., Rogers, S. J., McGee, G. G., ... & McNeerney, E. (2015). Naturalistic developmental behavioral interventions: Empirically validated treatments for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(8), 2411–2428.
- Shire, S. Y., Chang, Y. C., Shih, W., Bracaglia, S., Kodjoe, M., & Kasari, C. (2016). Hybrid implementation model of community-partnered early intervention for toddlers with autism: A randomized trial. *Journal of Child Psychology and Psychiatry*.
- Solomon, R., Van Egeren, L. A., Mahoney, G., Huber, M. S. Q., & Zimmerman, P. (2014). PLAY Project Home Consultation intervention program for young children with autism spectrum disorders: A randomized controlled trial. *Journal of Developmental and Behavioral Pediatrics*, 35(8), 475.
- Sparrow, S. S., Cicchetti, V. D., & Balla, A. D. (2005). *Vineland adaptive behavior scales* (2nd ed.). Circle Pines: American Guidance Service.
- Sussman, F. (1999). *More than words: Helping parents promote communication and social skills in children with autism spectrum disorders*. Toronto: The Hanen Centre.
- Tomasello, M., & Farrar, M. J. (1986). Joint attention and early language. *Child Development*, 57, 1454–1463.
- Wetherby, A. M., Guthrie, W., Woods, J., Schatschneider, C., Holland, R. D., Morgan, L., & Lord, C. (2014). Parent-implemented social intervention for toddlers with autism: An RCT. *Pediatrics*, 134(6), 1084–1093.
- Wilcox, M. J., & Shannon, M. S. (1998). Facilitating the transition from prelinguistic to linguistic communication. In A. Wetherby, S. F. Warren & J. Reichle (Eds.), *Transitions in prelinguistic communication* (pp. 385–416). Baltimore: Brookes.

Developmental Language Delay/Disorder

► Expressive Language Disorder

Developmental Language Disorder

► Expressive Dysphasia

Developmental Milestones

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Definition

Developmental milestones are a set of behaviors, skills, or abilities that are demonstrated by

specified ages during infancy and early childhood in typical development. Developmental milestones are often presented in lists broken down by ages, beginning around 1–3 months of age and progressing through approximately 5 years of age. The Centers for Disease Control and Prevention (CDC) provides easily accessible information through their website (2010). Several categories of skills are often focused on including vision and hearing, social, cognitive, language, motor, and self-help. Parents, day-care providers, teachers, child psychologists, and pediatricians often note emerging concerns regarding development when infants and children fail to reach developmental milestones on time.

While some variation is to be expected among individuals, developmental milestones are used as guidelines to assist in the identification of developmental delays, including autism spectrum disorders. When an infant or child is not reaching developmental milestones or is significantly delayed in meeting them, further assessment and evaluation should be completed. Early diagnosis and early intervention for autism are important for best outcomes. Skills that may be deficient in early development for individuals with autism spectrum disorders include social behavior, joint attention, visual orientation, orienting to noise, response to name, imitation of movement or sounds, and language acquisition including both receptive and expressive language (Watson et al. 2003).

See Also

- Developmental Delay
- Early Diagnosis
- Early Intervention
- Expressive Language
- Imitation
- Joint Attention
- Milestone
- Receptive Language

References and Reading

- Centers for Disease Control and Prevention. (2010). *Learn the signs. Act early.*. Retrieved May 6, 2011, from <http://www.cdc.gov/ncbddd/actearly/milestones/>.
- Watson, L. R., Baranek, G. T., & DiLavore, P. C. (2003). Toddlers with autism: Developmental perspectives. *Infants and Young Children*, 16, 201–214.

Developmental Reading Disorder

- Dyslexia

Developmental Right-Hemisphere Syndrome

- Nonverbal Learning Disabilities (NLD), Second Edition
- Right-Hemisphere Syndrome

Developmental Shifts Across Lifespan

- Developmental Continuum (Principles of TEACCH)

Developmental Test of Visual-Motor Integration

- Beery-Buktenica Developmental Test of Visual-Motor Integration

Developmental Verbal Apraxia (or Dyspraxia)

- Verbal Apraxia

Developmental, Dimensional, and Diagnostic Interview (3Di)

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Abbreviations

3Di	Developmental, Dimensional and Diagnostic Interview
ADHD	Attention Deficit Hyperactivity Disorder
ADI-R	Autism Diagnostic Interview – Revised
ADOS	Autism Diagnostic Observation Schedule
ASC	Autism Spectrum Condition
ASD	Autism Spectrum Disorder
DSM-IV-TR	Diagnostic and Statistical Manual of the American Psychiatric Association, 4th Revision, text revision
DSM-5	Diagnostic and Statistical Manual of the American Psychiatric Association, 5th Revision
ICD-10	International Classification of Disease (World Health Organization) 10th Revision
PDD	Pervasive Developmental Disorder
RRBI	Restricted Repetitive Behaviors and Interests

Description

The Developmental, Dimensional and Diagnostic Interview (3Di; Skuse et al. 2004) is a standardized, computer-based diagnostic interview in which individual scores of children from the age of 3 years old and upward in both clinical and normal populations with suspected autism spectrum disorder (ASD) can be scored in terms of their severity, frequency, and comorbidity. It was specifically designed to be easy and practical in its use, requiring minimal training and automation of reporting. The interview offers a dimensional assessment across the full range of autism spectrum characteristics. Subjects, whom the 3Di was primarily developed to assess, were children of school age with normal-range cognitive abilities. With simple modifications, the 3Di can also be used with younger children (from 2 years of age) as well as those with moderate or severe mental retardation. Algorithms can automatically adapt the scoring system for a particular age-group or to allow for a nonverbal subject. If employed for the assessment of preschool-aged children, or those in late adolescence and beyond, some rephrasing of questions will be required. In the case of older adolescents or adults, the interviewer would ask about a specific time when that individual was of an age when the domain of content applied. With minor modification, the interview can be administered as part of the diagnostic evaluation of adults. The 3Di was developed primarily to assist clinicians to make highly reliable diagnoses. It is recommended to use the 3Di in combination with independent observational assessments, such as the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2000). Validity was established in comparison with the ADI-R, and the ADI-R diagnostic algorithm can be emulated (Skuse et al. 2004). A recent modification enables the DSM-5 ASD criteria to be employed in diagnostic decision-making.

The answers to the questions on the 3Di are provided by parents and scored by a trained clinician (Skuse et al. 2004). The interview contains 183 questions concerning demography, family



background, developmental history, and motor skills. Another 266 items concern characteristics of ASD (i.e., social communication/language, social interaction, and restricted repetitive behaviors and interests [RRBI]), while a further 291 questions relate to current mental states relevant to other psychiatric diagnoses. Designed to sound as natural as possible, the questions must be asked in the same way as they are written, which should sound like a normal conversation with the informant. The 3Di is unique in its construction, as it is neither a completely structured nor a semi-structured interview but a hybrid between these two approaches. Moreover, the 3Di's use of computerized administration aims to ensure high reliability, good discriminant validity, and interviewee acceptability. To minimize the time spent in the face-to-face assessment, a pre-interview package of questionnaires has been designed that can be provided by parents in advance of their appointment and entered onto the computer from record forms. With this option, background information on family structure and biological functions such as motor skills, sleep, and eating/toileting behavior are completed in advance. Questions about autistic symptoms are always asked face-to-face. The full ASD interview, which is recommended for complex cases, can usually be conducted in 90–120 min, with pre-entry.

The 3Di was computerized to improve reliability. Computerization would ensure good inter-rater and test-retest reliability with minimal training, for it made rater drift less likely. The 3Di has a computerized interview database using Access Runtime, which is freely available and compatible with all versions of Windows. The system will run on iOS but requires Windows and Office to be installed with Parallels or equivalent. The latest release of the UK 3Di software is compatible with Windows 10. A structured computer-generated report in Microsoft Word is available upon completion. All data are automatically downloadable into spreadsheet format. The software used to construct the interview has a number of features that were developed to enhance the user experience. These include the use of an “interviewer explorer” tree and colored tabs that indicate

whether a question has been answered or not. The interview tailors itself to the gender of the subject and the names of the child and parents. Alternative routes through the interview can be selected, which facilitate the efficient pre-entry of material such as demographic details of the family, as well as allowing specific routes to be selected in order to identify comorbidity. Furthermore, the interview consists of several modules. The standard interview asks about both potential ASD and attention deficit hyperactivity disorder (ADHD). Other modules, which are concerned with the full range of child psychiatric comorbidity, are optional. There are also questions about a range of adaptive functions, including motor skills, sleeping and eating, etc. Algorithms speed the interview by eliminating illogicalities, such as questions about spoken language in a nonverbal child.

The interviewer must make simple coding decisions in response to most questions, as it is a critically important aspect of the design that the interviewer interprets the interviewee's qualitative responses and makes coding decisions based on their clinical expertise. The wording, content, and coding of those questions have been designed to simplify, quicken, and ease the decision-making process. Responses about behaviors are generally coded on three point scales: with “0” indicating no such behavior is or has ever been present; “1” signifying minimal evidence of such behavior; and “2” representing that there is or has ever been definite or persistent evidence of such behavior. For the RRBI domain, a fourth option is often possible, which includes clear evidence of the behavior and clear impact on social functioning. Because coding options are simple and limited, it is possible for the interviewer to quickly verify with the respondent which option is the most appropriate. Moreover, some coding decisions are deliberately skewed (e.g., “often, rarely, never”), in order to magnify differences between populations and increase discriminant validity. The 3Di recognizes that asking parents to describe in detail specific behaviors their child may have demonstrated many years previously produces responses of unverifiable validity. Accordingly, the interview's emphasis is on coding current

behaviors for the most part. Selected questions about repetitive and stereotyped behaviors and sensory sensitivities seek evidence from early childhood onward because these aspects of the autism spectrum tend to ameliorate, especially in those with normal-range intelligence.

Training in the use of the interview can be provided and is brief. It entails role-playing, video rating of model interviews, and an introduction to the interview software (Ix Dx 2019). Interpretation of the DSM-5 algorithms and the interview's integration with observational assessments is covered. Inter-rater reliability for the "blind video rating" is ascertained after every course and is excellent ($kappa > 0.85$). Training courses are held worldwide, and over 2500 trainees are now using the 3Di in clinical practice.

Historical Background

The Developmental, Diagnostic and Dimensional interview was developed in the early 2000s by David Skuse et al. (2004), when changes in the conceptualization of autism as a multidimensional condition raised the need for new methodologies that could capture the severity of autistic traits in clinical as well as general population samples. Both inter-rater and test-retest reliability were potentially enhanced by the computerized format, compared with other autism assessment instruments. The computerization of diagnostic algorithms ensured diagnostic validity was maintained, and the potential to create a diagnostic database facilitated research. The range of questions incorporated allowed users to distinguish between ASD, specific language impairment (SLI), and other comorbidities.

While the original interview takes 2 h to complete, a short 45-min version (3Di-sv) has also been constructed (Santosh et al. 2009). This short version comprises 53 items in 3 domains: reciprocal social interaction (24 items), communication (21 items), and repetitive and stereotyped behavior (8 items). In addition, it originally included eight items concerning language development and age of first symptoms, which were to be used for DSM-IV-TR classification of PDD

subtypes. Concurrent validity with the full version is excellent, but the 3Di-sv was originally not compatible with DSM-5 as it emulates the ADI-R (DSM-IV TR) algorithm. Therefore, in the Netherlands, a study was performed to make the 3Di-sv compatible with DSM-5 (Slappendel et al. 2016). This 3Di DSM-5 short version is currently being further evaluated.

Recently, a version for adults of normal intelligence has been developed: the 3Di-Adult (Mandy et al. 2018). The 3Di-Adult can be administered to a parent or a relative of the client. The items were taken from an initial item pool from the diagnostic algorithm of the original 3Di (Skuse et al. 2004; Santosh et al. 2009), which have previously been identified as being especially discriminating. Subsequently, based on discussion within the study team, the items were matched to the DSM-5 criteria. Next, based on discussion and pilot analyses of existing 3Di data, the questions were sorted into those best suited for discriminating symptoms in either childhood or adulthood, which were then rephrased as either historical questions ($n = 21$) or to assess current behavior ($n = 31$). Lastly, 17 new questions were constructed based upon knowledge of the ASD phenotype in adulthood, to ensure that all aspects of the DSM-5 were covered. This process resulted in a 69-item version for adults. Preliminary analyses revealed high inter-rater reliability and internal consistency of the scales. Furthermore, substantially higher subscale scores for ASD cases than comparisons were found, demonstrating criterion validity. The 3Di-Adult was also highly accurate in discriminating between ASD cases and comparisons and was found to have excellent sensitivity (95%) and specificity (92%). The interview took 50 min for people with ASD on average. Although these findings are preliminary, the 3Di-Adult seems promising as a useful tool in research or clinical practice in identifying ASD in adults based on the DSM-5 criteria. The 3Di-Adult has also been translated into Dutch, in which language its validity is currently being investigated.

The 3Di has previously been translated into several languages: Dutch, Finnish, Italian, Norwegian, Spanish, Cantonese, Mandarin,

Taiwanese, and Thai, of which the latter four are currently only available in the short version. Other translations (e.g., Arabic, Hindi, Swahili, French) are still under development. In 2012, a validation study was published by the Thai translation team (Chuthapisith et al. 2012) in which a group of 63 children with clinically ascertained ASD was compared to a group of 67 typically developing children on their mean 3Di-sv scales scores in each of the 3 domains. The results showed similar sensitivity and specificity to the original UK sv-validation (Santosh et al. 2009). The highly detailed phenotyping that is possible with the 3Di allows one to examine subtle differences in the ASD phenotype that might be due to cultural factors. Considering the reformulation of ASD in the DSM-5, and the condition becoming an increasingly global diagnosis, a study was performed (Mandy et al. 2014) that investigated the DSM-5 version's applicability and cross-cultural stability across the spectrum of symptoms in two different cultures (i.e., UK and Finland). Using confirmatory factor analysis and comparison of model fit between Finnish and UK participants, it was found that the DSM-5 model worked well in ASD participants in both cultures. However, it was found not suitable for measuring the sub-clinical autistic trait characteristic of the broader autism phenotype (BAP) in the Finnish population. This finding might suggest that cross-cultural variability may be greatest for subtle differences in the ASD phenotype.

A study by Slappendel et al. (2016) investigated the items of the short version of the 3Di (3Di-sv; Santosh et al. 2009) which were translated into Dutch. Criterion validity was explored by first comparing the 3Di-sv classifications to clinical diagnoses that were confirmed by an ADOS-2 classification. Data were available for 146 children (50 of whom were considered to have ASD). Although the 3Di-sv classifications showed good sensitivity (.84), specificity was low (.54) when comparing with ADOS-2-confirmed clinical diagnosis. Post hoc analyses with the full sample ($n = 282$) were then conducted, which lacked some ADOS-2 data, but which better reflected the samples used in previous studies

that reported higher specificity for the 3Di-sv's performance (e.g., Chuthapisith et al. 2012). These resulted in a sensitivity of 0.77 and an improved specificity of 0.67. The researchers suggested the initial limited specificity they found, compared to previous studies, was due both to the stringency of their diagnostic criteria and to the clinical characteristics of their population.

Moreover, the PDD module of the 3Di was translated into Cantonese, and its applicability was subsequently tested using a sample of 194 Chinese children aged 6–12 years in Hong Kong (Lai et al. 2015). Using the original authors' cutoff scores, the Cantonese version was found to have excellent psychometric properties, a sensitivity of 95%, and a specificity of 77%. In distinguishing between ASD and non-ASD cases, the agreement was 85.6%, with κ 0.71. However, the relatively higher false-positive rate and relatively high scores on one of the PDD dimensions could be due to cultural differences between England and Hong Kong. For example, children in Hong Kong are less likely than British children to invite friends over after school. In these instances, higher 3Di scores do not indicate autistic symptoms but instead reflect the (social) cultural expectations, habits, and lifestyle of the people of Hong Kong. This finding highlights the need for further validation and development of norms for the 3Di in different cultural contexts.

The 3Di has been used in a wide variety of research studies. The detailed phenotyping generated by the interview has encouraged investigations of conceptual issues and more recently the construct validity of DSM-5 (e.g., Mandy et al. 2012a; Slappendel et al. 2016). Because the instrument seeks information about a wide range of associated symptoms, language development, and comorbidity, it has allowed researchers to employ the interview in studies on these topics. Furthermore, as trait measurement is quintessentially dimensional in character, the 3Di is also well suited for studies of the broader phenotype of ASC (e.g., De la Marche et al. 2015) and sex differences in the phenotype (Mandy et al. 2012b).

Psychometric Data

Reliability studies have been conducted in the development phase of the instrument, as well as “live” measures of reliability which were obtained during training courses. In the original reliability analyses (Skuse et al. 2004), all sections of the interview were administered to parents of 23 PDD and 27 non-PDD cases in a referral sample. The interview was performed by one member of the assessment team, while another scored the responses independently on a computer. Inter-rater and test-retest reliability were analyzed for ICD-10 diagnoses which are generated by the 3Di algorithms, resulting in 96% and 94% agreement, respectively. Intraclass correlation coefficients for both reliabilities were found in excess of 0.86, which is excellent. The reliability of the instrument during live training sessions has also been evaluated, when each group of trainees rates a video recording of an entire interview with the parent of an autistic child. Reliability was evaluated on the basis of ADI-R algorithm scores. For a consecutive training group of 208 psychiatrists, pediatricians, and other professionals, representing approximately 20 training courses, the reference score of the interviewer was compared to the group mean. For the reciprocal social interaction subscale, language and communication skills, and repetitive and stereotyped behaviors, the scores of the trainees were almost identical to the reference scores.

Extensive research on the 3Di has demonstrated its content, concurrent, discriminant, and criterion validity for ASD. The content validity was assessed (Skuse et al. 2004) by reviewing published material on significant discriminating behavioral characteristics in children with ASD as well as the diagnostic criteria as described in the DSM-IV (APA 2013) and ICD-10 (World Health Organization 1993). Concurrent validity of the 3Di with the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2000) has been evaluated in both preschool and school-age children. Duvekot et al. (2015) found that the overall percent agreement between the 3Di and ADOS classification was 63%, with a $kappa$ of 0.20, indicating a slight agreement. However, as the

3Di and ADOS are different types of diagnostic methods (interview and observation), high agreement would not be expected. These instruments should nevertheless be complementary in the diagnostic process, to gain information from observation as well as parent report on current behavior and developmental history. Formal analysis of this aspect of validity is pending.

Furthermore, concurrent validity in autism spectrum conditions has been evaluated using a clinical sample of 60 children (Skuse et al. 2004). The diagnoses by a clinician on the basis of ICD-10 criteria and diagnoses on the basis of 3Di algorithms were compared, which showed that agreement between these methods was very good. Discriminant validity based on the 3Di algorithm's ability to distinguish between autistic and non-autistic conditions was found to be excellent, with a sensitivity of 1.0 and specificity being higher than 0.97. Furthermore, the 3Di's positive and negative predictive powers were found to be 0.93 and 0.91, respectively. The assessment of criterion validity was based on the assessment of 29 children with both the 3Di and the ADI-R, who had been interviewed on both instruments by different interviewers at two different points in time (a 2.4 years apart). Subscales that were similar in broad content and identical in the range of scores to the ADI-R were produced for the 3Di. The agreement on threshold for all three domains was found to be significant with $p < 0.01$, with 86% agreement in the reciprocal social interaction domain, 100% agreement for communication, and 76% for repetitive and stereotyped behaviors. However, complete agreement was not expected, in part because criterion validity was potentially compromised by inter-rater and test-retest variability and in part because the ADI-R algorithm was based largely on historical information, whereas the 3Di seeks contemporaneous information in virtually all domains.

Clinical Uses

The *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association 2013) and the International

Classification of Disease (ICD-10; World Health Organization 1993) are the current accepted classification systems for the diagnosis of ASD. The process of reaching an ASD diagnosis is complex, considering the etiological heterogeneity of ASD, high rates of comorbidity with other medical, psychological or psychiatric conditions, and different levels of severity in symptoms (Falkmer et al. 2013). Furthermore, diagnostic instruments should be highly sensitive and specific, in order to reduce the chances of false positives or negatives. Preferably, diagnostic instruments should also be brief and practical, as well as appropriate for all ages and IQ ranges within the autism spectrum (Mayes et al. 2009). Considering the 3Di's preliminary results indicating excellent reliability and sensitivity, high sensitivity, specificity, and correct classification rate, as well as its practical computerized use and option for the use of a short version, it has proven to be a valuable tool in the diagnostic process.

The current “gold standard” for diagnosis of ASD includes a standardized observation of the child, such as the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2000) and a standard parental interview, for example, the Autism Diagnostic Interview-Revised (ADI-R; Lord et al. 1994) or the Developmental, Dimensional and Diagnostic Interview (3Di; Skuse et al. 2004) (Duvekot et al. 2015). In addition, according to the National Institute for Health and Care Excellence (NICE 2019), the true “gold-standard” clinical assessment should be a multidisciplinary team process which also involves other assessments with consensus clinical judgment such as differential diagnosis, a physical examination, and assessment of comorbid conditions. In a systematic literature review on diagnostic instruments assessing ASD by Falkmer et al. (2013), the 3Di was noted to have the highest correct classification rates of all tools evaluated. However, there are relatively few studies that provide evidence of its utility. Therefore, more research is needed on the 3Di in this regard.

The 3Di was originally devised to be a dimensional measure of autistic traits, although the disorder was predominantly conceptualized in categorical terms at that point in time. Accordingly, it was designed to emulate the algorithmic

content of the ADI-R (Lord et al. 1994), and as such, the 3Di's algorithm agreement with the ADI-R for threshold scores is high (Skuse et al. 2004). Furthermore, the 3Di was meant to also measure symptoms in a far broader context, including associated problems such as sensory sensitivities and pragmatic language skills, which were not at that time part of any diagnostic rubric, thus being wider in scope. The latest release of the 3Di-5 also includes a comorbidities tool, that screens for additional disorders based on DSM-5 diagnostic criteria. Algorithms generate comorbidity risks for all common child diagnoses. If full pre-entry has been achieved, it will be possible without further questioning – upon completion of the interview – to generate comorbidity probabilities for ADHD, conduct disorder, and tic disorders as well as pervasive developmental disorders. Additional modules allow the measurement of anxiety disorders, depression, dysthymia, bipolar affective disorder, eating disorders, post-traumatic stress disorder, and schizotypal disorders. However, it is important to note that the modules of this screening tool have not yet been validated, except for the ADHD module (which is mandatory because of the high overlap of symptoms with ASD), and the anorexia module has a solid foundation of expert input.

When the DSM-5 was published (APA 2013), the new diagnostic criteria were incorporated into a revised 3Di by writing new questions and developing new algorithms which mapped onto the DSM-5 criteria A and B. These included nonverbal interaction, peer relationships, sharing, socio-emotional reciprocity, nonverbal communication skills, conversational abilities, unusual preoccupations, routines and rituals, stereotyped and repetitive motor behavior, preoccupation with parts of objects, and sensory abnormalities. In the revised version, 108 questions contribute to the A-scale and 74 questions to the B-scale DSM-5 diagnostic algorithms. Mandy et al. (2012a) performed confirmatory factor analysis to assess the construct validity of ASD, especially its bidimensional reconceptualization of the autism phenotype. It was found that the two-factor model of autistic symptoms as described in the DSM-5 had superior statistical properties to the

DSM-IV-TR model. The results also showed that it made statistical sense to include stereotyped language usage (a subscale of the Children's Communication Checklist) within the restricted repetitive behavior dimension, together with the newly developed sensory abnormality symptoms. The latest release of the 3Di-5 (available upon request) has greatly increased the number of questions within this latter dimension (especially in the domain hyper- and hyposensitivity to a range of environmental stimuli), which has improved its metrical properties. However, although the 3Di output is tabulated in DSM-5 compatible form, the interpretation of that output has not yet been validated. To explore the compatibility of the 3Di-sv with the DSM-5, Slappendel et al. (2016) performed a study to investigate whether the short version of the Dutch 3Di translation (3Di-sv) would fit the DSM-5's two-factor structure as well as the three-factor structure of the DSM-IV-TR. Confirmatory factor analyses revealed a better fit for a DSM-5 model than DSM-IV-TR model of ASD. As exploration of the content validity for the DSM-5 revealed some construct underrepresentation, thus a panel of 3Di-trained clinicians suggested 14 items be added to improve the 3Di-sv construct, based on a theory-driven, face validity perspective. These suggestions included questions on the topics of insistence of sameness, adherence to routines, and sensory hyper- and hypo reactivity.

For future research, in order to create a new DSM-5 (short) version of the 3Di, adjustments need to be made and subsequently examined thoroughly. As current scoring algorithms still lead to DSM-IV-TR domains, specific DSM-5 3Di algorithms need to be validated and applied in sufficiently large samples and tested in clinical settings in order to achieve a full test of the construct validity of ASD. For example, versions including items that were added to provide better coverage of newly introduced DSM-5 criteria need further validation. Therefore, data-driven research on the suggested items on the basis of clinical consensus by Slappendel et al. (2016) is currently pending. This type of research should also consider evaluating the validity of the novel DSM-5 diagnosis of Social Communication Disorder, which is generated by 3Di algorithms. This diagnosis may apply

to children who were formerly diagnosed by DSM-IV TR with Asperger's disorder or PDD-NOS. Concern has been expressed that they may not fulfil the new DSM-5 criteria for ASD, which might subsequently lead to loss of eligibility for care (McPartland et al. 2012). Furthermore, translation and validation of the 3Di-Adult in other cultures will provide a reliable, valid, and resource-efficient way in helping the "lost generation" of adults with ASD who are currently lacking a formal diagnosis and therefore appropriate treatment (Mandy et al. 2018).

See Also

- [Attention Deficit/Hyperactivity Disorder](#)
- [Autism Diagnostic Interview-Revised](#)
- [Autism Diagnostic Observation Schedule](#)
- [Autism Spectrum Disorder](#)
- [Broader Autism Phenotype](#)
- [Children's Communication Checklist \(CCC-2\)](#)
- [Clinical Assessment](#)
- [Clinician](#)
- [Comorbidity](#)
- [Culture and Autism](#)
- [Diagnosis and Classification](#)
- [Diagnostic Instruments in Autistic Spectrum Disorders](#)
- [Diagnostic Interviews](#)
- [Diagnostic Process](#)
- [DSM-5](#)
- [False Positive](#)
- [ICD 10 Research Diagnostic Guidelines](#)
- [Sensitivity and Specificity](#)
- [Validity](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Publishing.
- Chuthapisith, J., Taycharpipranai, P., Ruangdaraganon, N., Warrington, R., & Skuse, D. (2012). Translation and validation of the developmental, dimensional and diagnostic interview (3Di) for diagnosis of autism spectrum disorder in Thai children. *Autism, 16*(4), 350–356.
- De la Marche, W., Noens, I., Boets, B., Kuppens, S., & Steyaert, J. (2015). The underlying symptom structure of autism spectrum disorders: A factor analytic

- approach using the developmental, dimensional and diagnostic interview. *Research in Autism Spectrum Disorders*, 12, 40–51.
- Duvekot, J., Van der Ende, J., Verhulst, F. C., & Greaves-Lord, K. (2015). The screening accuracy of the parent and teacher-reported Social Responsiveness Scale (SRS): Comparison with the 3Di and ADOS. *Journal of Autism and Developmental Disorders*, 45, 1658–1672.
- Falkmer, T., Anderson, K., Falkmer, M., & Horlin, C. (2013). Diagnostic procedures in autism spectrum disorders: A systematic literature review. *European Child and Adolescent Psychiatry*, 22, 329–340.
- IXDX. (2019). *Getting hold of the 3di*. Retrieved August 1st, 2019, from: <http://www.ixdx.org/3di-courses.html>
- Lai, K. Y. C., Leung, P. W. L., Mo, F. Y. M., Lee, M. M. C., Shea, C. K. S., Chan, G. F. C. (...) & Skuse, D. H. (2015). Validation of the developmental, dimensional and diagnostic interview (3Di) among Chinese children in a child psychiatry clinic in Hong Kong. *Journal of Autism and Developmental Disorders*, 45, 1230–1237.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview – Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24, 659–685.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H. Leventhal, B. L., DiLavore, P. C., (...) & Rutter, M. (2000). The autism diagnostic observation schedule–generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30, 205–223.
- Mandy, W., Charman, T., & Skuse, D. H. (2012a). Testing the construct validity of proposed criteria for DSM-5 autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(1), 41–50.
- Mandy, W., Chilvers, R. J., Chowdhury, U., Salter, G. C., Seigal, A., & Skuse, D. (2012b). Sex differences in autism spectrum disorder: Evidence from a large sample of children and adolescents. *Journal of Autism and Developmental Disorders*, 42(7), 1304–1313.
- Mandy, W., Charman, T., Puura, K., & Skuse, D. (2014). Investigating the cross-cultural validity of DSM-5 autism spectrum disorder: Evidence from Finnish and UK samples. *Autism*, 18(1), 45–54.
- Mandy, W., Clarke, K., McKenney, M., Strydom, A., Crabtree, J., Lai, M.-C., (...) Skuse, D. H. (2018). Assessing autism in adults: An evaluation of the developmental, dimensional and diagnostic interview-adult version (3Di-Adult). *Journal of Autism and Developmental Disorders*, 48, 549–560.
- Mayes, S. D., Calhoun, S. L., Murray, M. J., Morrow, J. D., Yurich, K. K. L., Mahr, F., (...) & Petersen, C. (2009). Comparison of scores on the Checklist for autism spectrum disorder, childhood autism rating scale, and Gilliam Asperger's disorder scale for children with low functioning autism, high functioning autism, Asperger's disorder, ADHD, and typical development. *Journal of Autism and Developmental Disorders*, 39, 1682–1693.
- McPartland, J. C., Reichow, B., & Volkmar, F. R. (2012). Sensitivity and specificity of proposed DSM-5 diagnostic criteria for autism spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(4), 368–383.
- National Institute for Health and Care Excellence (NICE). (2019). *Autism spectrum disorder in under 19s: Recognition, referral and diagnosis*. Retrieved August 27th, 2019, from: <https://www.nice.org.uk/guidance/cg128/chapter/Recommendations#autism-diagnostic-assessment-for-children-and-young-people>
- Santosh, P. J., Mandy, W. P. L., Puura, K., Kaartinen, M., Warrington, R., & Skuse, D. H. (2009). The construction and validation of a short form of the developmental, diagnostic and dimensional interview. *European Child and Adolescent Psychiatry*, 18, 521–524.
- Skuse, D., Warrington, R., Bishop, D., Chowdhury, U., Lau, J., & Mandy, W. (2004). The developmental, dimensional and diagnostic interview (3di): A novel computerized assessment for autism spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45(5), 548–558.
- Slappendel, G., Mandy, W., Van der Ende, J., Verhulst, F. C., Van der Sijde, A., Duvekot, J., Skuse, D., & Greaves-Lord, K. (2016). Utility of the 3di short version for the diagnostic assessment of autism spectrum disorder and compatibility with DSM-5. *Journal of Autism and Developmental Disorders*, 46, 1834–1846.
- World Health Organization. (1993). *The ICD-10 classification of mental and behavioural disorders. Diagnostic criteria for research*. Geneva: World Health Organization.

Developmental, Psychiatric, and Genetic Profiles of the 22q11.2 Deletion Syndrome

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Synonyms

VCFS

Short Description or Definition

One of the most common genomic syndromes involving multiple anomalies is Chromosome 22q11.2 deletion syndrome. Although there are earlier mentions of the disorder clinically, going back to 1829 (Greenberg 1993), the syndrome was first exemplified in detail by Angelo DiGeorge in 1968 (DiGeorge 1968) and by R. J. Shprintzen and colleagues in 1978 (Shprintzen et al. 1978); all presented children demonstrated clinical abnormalities such as underactive parathyroid gland, underdeveloped thymus, palate defects, facial anomalies, heart defects, and cognitive limitations. In the 1970s, the syndrome was also noted by Kinouchi et al. (1976), who labeled the condition as conotruncal anomaly face syndrome. Ultimately, in the 1990s, the specific genetic cause for the condition was identified as a microdeletion (i.e., a deletion of a part of a chromosome) of chromosome 22 at band q11.2 (Scambler et al. 1992). Thus, over the years, this genomic disorder has been called Sedlackova syndrome, DiGeorge syndrome, Shprintzen syndrome, conotruncal anomaly face syndrome, and velocardiofacial syndrome (VCFS). Since the 1990s, the syndrome has been increasingly referred to as 22q11.2 deletion syndrome, 22q11.2DS, which was used as an umbrella term to describe various clinical phenotypes originating from the deletion.

The 22q11.2DS is associated with a vast array of physical, medical, cognitive, behavioral, and psychiatric features. It is characterized by multiple (>180) developmental aberrations, including specific craniofacial features (cleft palate, velopharyngeal insufficiency; 69–100%), thymic and parathyroid defects including hypocalcemia (17–60%), congenital cardiovascular malformations (49–83%), and mild to moderate renal anomalies (36–37%). The most striking feature of the syndrome is the tremendous degree of phenotypic variability, both at the levels of the brain and behavior (Bassett et al. 2011; Kobrynski and Sullivan 2007; Roizen et al. 2007; Wang et al. 1998).

The associated phenotypes range from combinations of serious anomalies to the presence of

isolated mild impairments (McDonald-McGinn and Zackai 2008). Another central feature of the syndrome is the prominent presence of psychopathology (9–50%), with the most frequently observed conditions being developmental delay in infancy (75%) and childhood (45%), speech and language disorders (79–84%), schizophrenia spectrum disorders (1.9–41.7%), autism spectrum disorders (16–26.5%), attention deficit hyperactivity disorder (15–37%), and learning disabilities (50–80%) (Kobrynski and Sullivan 2007; McDonald-McGinn et al. 1999; Polleux and Lauder 2004; Prasad et al. 2008; Ryan et al. 1997; Schneider et al. 2014).

In addition, there are pronounced transformations of the syndrome's presentation across different developmental stages. The causes of such variability within the group of individuals with 22q11.2DS and across the life span within an individual are unclear (Aggarwal and Morrow 2008; Schneider et al. 2014). One of the most consistent findings in the phenotypic presentation of 22q11.2DS is a low average to borderline range of general cognitive functioning (Antshel et al. 2008). Yet, this deficiency is not consistent across different domains of cognitive functioning, although, as a group, individuals with the syndrome tend to function at a lower level than typically developing individuals in all domains of cognitive performance. Verbal functioning in spoken and written domains (e.g., receptive language, decoding and spelling, auditory verbal rote memory, but not higher-level processing such as comprehension) has been observed to be a relative strength, whereas visual-spatial (e.g., space orientation, visuospatial memory, visuospatial perception), quantitative processing (e.g., number and magnitude representation), and executive functioning (e.g., initiation, planning, working memory, and monitoring skills) have been marked as domains of particular weakness. Yet, once again, although this profile appears to be characteristic of individuals with 22q11.2DS as a group, within this group, there is variation in both the direction (i.e., reversed cognitive patterns have been reported) and the magnitude (i.e., the standard scores) of the discrepancy. Of note, data from a collaborative study by the International 22q11.2

Brain Behavior Consortium (Vrotsman et al. 2015) revealed that individuals with the syndrome show a cognitive decline of 7.04 Full Scale IQ points, 9.02 points in Verbal IQ, and 5.09 points in Performance IQ. Additionally, those individuals with a psychotic spectrum disorder experienced even more steeper declines in cognitive functioning.

Epidemiology

Chromosome 22q11.2DS is the most common microdeletion syndrome in humans, with prevalence estimates ranging from one in 2,000 to one in 6,395; with only 5–10% of deletions having been inherited and the rest arising *de novo* (Swillen et al. 1998). However, because many children born with the condition do not have observable abnormalities or immediately detectable medical difficulties, it has been suggested that up to one third of children are not diagnosed until later childhood and that some cases may go completely undetected. Therefore, the population prevalence may be closer to affecting 1 in 2,000–4,000 live births (Schneider et al. 2014; Shprintzen 2008). Boys and girls are affected equally, but the deletion is more prevalent within certain ethnic groups; 22q11.2DS occurs more frequently among Hispanics compared with Whites, African Americans, and Asians (Botto et al. 2003).

Natural History, Prognostic Factors, and Outcomes

Studies have demonstrated that heart defects and severe immune deficiency are the main causes of death in infants with 22q11.2DS. However, with the advancements in neonatal cardiology, many infants undergo early surgical repairs that permit them to survive. Currently, there is limited information regarding the life span of individuals with 22q11.2DS. There are regional data that suggest that if individuals receive adequate medical interventions or do not develop significant medical conditions, then the life span may be normative

(Shprintzen 2008). Similarly, psychological, psychiatric, functional, and vocational outcomes are likely influenced by a host of risk and protective factors, including: the size of the genetic deletion, genes in the region, the number of medical problems, presence of intellectual disability, and co-morbid psychiatric conditions, as well as family and environmental factors such as access to specialized educational and therapeutic interventions (Swillen et al. 2018).

Clinical Expression and Pathophysiology

Clinical Expression

The clinical expression of 22q11.2DS is extremely variable. Common phenotypic presentations may include the following: craniofacial abnormalities (cleft lip, cleft palate, velopharyngeal insufficiency, and microcephaly), feeding difficulties, conotruncal heart defects (tetralogy of Fallot, interrupted aortic arch, and ventricular septal defects), hearing loss or abnormal ear exams, genitourinary anomalies (absent or malformed kidneys), hypocalcemia (low blood calcium), and immunological difficulties due to abnormal T-cells. The facial features of children may also be atypical and may include the following: small ears, hooded eyelids, asymmetric crying facies, and small mouth, chin, and nasal area (Velo-Cardio-Facial Syndrome Educational Inc. 2009). In addition to these various medical vulnerabilities, individuals with 22q11.2DS frequently demonstrate a range of cognitive impairments. Typically, individuals exhibit low average to borderline intellectual functioning. Intellectual disability, typically in the mild range, has been documented in 25–40% of individuals (Shprintzen 2000; Swillen et al. 1997). In early childhood, infants and toddlers typically present with global developmental delays, including speech and language disorders. Later in childhood, specific patterns of cognitive and learning difficulties may become evident. Specifically, relative strengths in rote verbal memory, reading decoding, and spelling have been noted, in contrast to relative weaknesses in visuospatial memory, math computation, and executive functioning

(planning, cognitive flexibility, and working memory) (Simon et al. 2005).

Numerous psychiatric conditions have been reported to occur in individuals with 22q11.2DS. Accordingly, elevated rates of attention deficit hyperactivity disorder (ADHD), anxiety disorders, major depression, obsessive compulsive disorder, and oppositional-defiant disorder have been observed in clinical samples (Antshel et al. 2006). In late adolescence and early adulthood, even more debilitating psychiatric conditions may emerge, such as those characterized by psychotic symptoms, including schizophrenia and schizoaffective disorder. Gothelf et al. (2009) initially suggested that up to one third of patients may acquire psychotic disorders. Yet data from the International Consortium on 22q11.2 indicate that the rates of psychotic disorders vary across age groups, ranging from 1.97% in childhood to 10.2% in adolescence, and 41.3% in mature adults (Schneider et al. 2014).

There have been numerous investigators who have concluded that autism spectrum disorders can be quite prevalent in individuals with 22q11.2DS, with rates ranging from 12% to 50% (Antshel et al. 2007; Chudley et al. 1998; Fine et al. 2005; Niklasson et al. 2001; Schneider et al. 2014; Vorstman et al. 2006). Investigators have also examined how the social deficits observed in children with combined 22q11.2DS and autism are the same or different than children with just autism. There are researchers that have proposed that autism in 22q11.2DS may be qualitatively quite different from idiopathic autism. Specifically, children with combined 22q11.2DS and ASD may have less joint attention, lower levels of make-believe play, and higher levels of repetitive behaviors than those with 22q11.2DS alone (Eliez 2007; Tang et al. 2015). Thus, there is a consensus that children with 22q11.2DS should receive routine, standardized assessments for autism. Furthermore, early assessment for more serious psychopathology such as schizophrenia should also be conducted with children.

Genetics

The pathogenesis of 22q11.2DS is understood relatively well (Grigorenko et al. 2010). Most

cases of 22q11.2DS occur in the general population sporadically, or by chance, but only a portion of cases are inherited as an autosomal dominant trait (Swillen et al. 1998). The syndrome's molecular mechanism is well studied on the cytogenetic level (i.e., at a resolution of hundreds of thousands of bp); the deleted material can range from the more commonly seen 3 Mb deletion (~90%) to the less commonly seen 1.5 Mb deletion (~7%), with the rest having an even smaller deletion with an undetermined minimal size. Deletions in individuals with 22q11.2DS are more frequently of maternal than paternal origin, 56% versus 44% (Hacıhamdioğlu et al. 2015). There appears to be no association between the parental age and the frequency of the deletion (Delio et al. 2013). The pericentromeric region of chromosome 22q, and in particular 22q11, is rich in regions of segmental duplication (SD). SD regions are stretches of the human genome that have one or several highly sequence-homologous (i.e., more than 90% of identical sequence) counterpart(s) elsewhere in the genome, either upstream or downstream on the same chromosome but also on other chromosomes. Such SDs are frequently associated with variability in copy number, even in the normal genome of healthy individuals, as well as with the formation of aberrations leading to genomic disease. In 22q11.2DS, the 3 Mb typical deletion has both of its breakpoints in prominent SD regions. Also, most of the subtypical or atypical deletions have at least one of their endpoints in one such segmentally duplicated stretch of 22q11. The 3 Mb deletion region affects approximately 50 genes and 7 micro-RNAs, whereas the 1.5 Mb region 24 genes. The diminished dose of these genes, or their haploinsufficiency, likely compromises early morphogenesis of the organs impacted by the syndrome. Based on the research in mouse models, however, it appears that no single 22q11.2 deletion region is both necessary and sufficient for the expression of the major features of the syndrome (Amati et al. 2007). Similarly, there appears to be no difference in the clinical severity of the disorder depending on the size of the deletion. Only specific facets of the general clinical manifestation of 22q11.2DS have been understood so far. For example,

malformations of the heart and outflow tracts, as well as facial dysmorphism, palatal defects, hypoplasia of the parathyroid glands and thymus, and dental, feeding, and swallowing problems in individuals with 22q11.2DS are believed to be associated with haploinsufficiency for the *TBX1* (T-box-containing transcription factor 1) gene (Lindsay et al. 1999; Scambler 2010). However, other facets of 22q11.2DS remain etiological puzzles. For example, it is assumed that the syndrome and its psychiatric facets are associated with one or more candidate genes in the deleted region (Prasad et al. 2008). Specifically, among these genes are *COMT* (the gene encoding catechol-O-methyl transferase), *PRODH* (the gene coding for proline dehydrogenase), *GNBIL* (a gene that encodes a protein of unknown function), *ZDHHC8* (the gene coding for zinc finger and DHHC domain-containing protein 8), and *ARVCF* (the armadillo repeat gene deleted in 22q11.2DS, which may have a role in cell-to-cell communication and intracellular transduction during embryonic development). Yet, the results of studies of these candidate genes are provocative, but inconclusive. Similar to the role of *TBX1*, it is possible that haploinsufficiency in specific other genes results in specific features of 22q11.2DS (Meechan et al. 2007). Thus, haploinsufficiency for *GP1BB* might contribute to the mild thrombocytopenia seen in patients, and haploinsufficiency for *COMT* might contribute to cognitive aspects of the 22q11.2DS phenotype (Kobrynski and Sullivan 2007). The genes lost in 22q11.2DS, *PRODH*, *TBX1*, *GNBIL*, *COMT*, *ARVCF*, *DGCR8*, *RANBP1*, *ZDHHC8*, and *PIK4CA*, are located in or relatively close to regions of SD. Each of these genes or a combination of them might contribute to the etiology of the syndrome. However, with the genomic technology that is now allowing high-resolution analyses (i.e., with a resolution of just a few 100 bp and even down to the level of a single bp, in contrast with conventional cytogenetic analyses that have a resolution no better than several tens of thousands and often as many as hundreds of thousands of bp), the picture is changing dramatically. There is a variety of deletions found in chromosome 22q11.2 that are largely submicroscopic, have a

recognizable but often variable phenotype, and show recurrent breakpoints in unrelated individuals (McDonald-McGinn et al. 2015).

Evaluation and Differential Diagnosis

When 22q11.2DS is suspected by a physician (most often due to the presence of heart defects, clefting, facial anomalies, hypocalcemia, or absent thymus), then the most reliable diagnostic tool is a blood test in which the deletion in chromosome 22q11.2 region is detected through the FISH test (Fluorescence In Situ Hybridization in Situ Hybridization) or by other higher-resolution molecular techniques. If no deletion is found in this specific region of the chromosome, then additional chromosomal studies may be conducted to determine whether other types of chromosomal disorders might be present. Once the diagnosis of 22q11.2DS has been made in a child, then typically the parents are encouraged to take the FISH test so that it might be determined whether the child's condition was inherited (Velo-Cardio-Facial Syndrome Educational Inc., 2009). Within the psychological domain, given the preponderance of early developmental delays (language disorders, intellectual disability, and learning disabilities) in addition to the possible emergence of significant behavioral (ADHD), psychiatric (anxiety, depression, and psychosis), and social difficulties (autism), it becomes critical for patients with 22q11.2DS to have repeated comprehensive developmental and neuropsychiatric assessments across the life span.

Treatment

Due to the highly varied needs of children and adolescents with 22q11.2DS, treatment approaches by necessity can be quite complex and multidisciplinary (Shprintzen 2008). The International 22q11.2DS Consortium (Bassett et al. 2011) has developed a range of "Practical Guidelines" for the management of patients with 22q11.2DS which takes into account the multi-systemic nature of the disorder and the varied

clinical considerations across the lifespan. Accordingly, common medical difficulties such as cardiac malformations or heart disease are managed through surgical interventions, and cleft palate repairs can be effectively resolved through palate repair. Similarly, severe feeding difficulties can be temporarily addressed through gastrostomy, but are most often remediated through behavioral feeding programs. For infants and toddlers with early developmental delays, including poor muscle tone and delayed speech, then referrals for early stimulation programs become critical. In later childhood, children with 22q11.2DS may require a range of special education services including speech and language therapy, occupational therapy, physical therapy, learning disabilities instruction, and even specialized school placements. Finally, as discussed, due to the high rates of behavioral and psychiatric conditions in children with 22q11.2DS, early evaluation is warranted through use of standardized instruments and structured observations. Pharmacological interventions to treat ADHD, mood disorders, and psychotic symptoms have been increasingly utilized with patients with 22q11.2DS (Shprintzen 2008), in addition to a range of evidence-based behavioral and cognitive behavioral treatments (Tang et al. 2015) such as cognitive remediation training, applied behavioral analysis, social skills training, CBT for anxiety and depression, and parent training programs.

See Also

- ▶ [CATCH 22 \(Chromosome 22q11 Deletion Syndrome\)](#)
- ▶ [Genetics](#)
- ▶ [Intellectual Disability](#)
- ▶ [Psychosis](#)
- ▶ [Schizophrenia](#)

References and Reading

Aggarwal, V. S., & Morrow, B. E. (2008). Genetic modifiers of the physical malformations in velo-cardio-facial syndrome/DiGeorge syndrome. *Developmental Disabilities Research Reviews*, 14, 19–25.

- Amati, F., Biancolella, M., Farcomeni, A., Giallonardi, S., Bueno, S., Minella, D., et al. (2007). Dynamic changes in gene expression profiles of 22q11 and related orthologous genes during mouse development. *Gene*, 391, 91–102.
- Antshel, K., Fremont, W., Roizen, N., Shprintzen, R., Higgins, A., Dhamoon, A., et al. (2006). ADHD, major depressive disorder, and simple phobias are prevalent psychiatric conditions in youth with velocardiofacial syndrome. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45, 596–603.
- Antshel, K., Aneja, A., Strunge, L., Peebles, J., Fremont, W., Stallone, K., et al. (2007). Autistic spectrum disorders in velo-cardio facial syndrome (22p11.2 deletion). *Journal of Autism and Developmental Disorders*, 37, 1776–1786.
- Antshel, K. M., Fremont, W., & Kates, W. R. (2008). The neurocognitive phenotype in velo-cardio-facial syndrome: A developmental perspective. *Development Disabilities Research Reviews*, 14, 43–51.
- Bassett, A. S., McDonald-McGinn, D. M., Devriendt, K., Diglio, M. C., Goldenberg, P., Habel, A., et al. (2011). Practical guidelines for managing patients with 22q11.2 deletion syndrome. *The Journal of Pediatrics*, 159, 332–339.
- Botto, L. D., May, K., Fernhoff, P. M., Correa, A., Coleman, K., Rasmussen, S. A., Merritt, R. K., O'Leary, L. A., Wong, L. Y., Elixson, E. M., Mahle, W. T., & Campbell, R. M. (2003). A population-based study of the 22q11.2 deletion: Phenotype, incidence, and contribution to major birth defects in the population. *Pediatrics*, 112, 101–107.
- Chudley, J., Jocelyn, L., & Chodirker, B. (1998). Outcomes of genetic evaluation in children with pervasive developmental disorder. *Journal of Developmental and Behavioral Pediatrics*, 19, 321–325.
- Delio, M., Guo, T., McDonald-McGinn, D. M., et al. (2013). Enhanced maternal origin of the 22q11.2 deletion in velocardiofacial and DiGeorge syndromes. *American Journal of Human Genetics*, 92, 439–447.
- DiGeorge, A. (1968). Congenital absence of the thymus and its immunologic consequences: Concurrence with congenital hypoparathyroidism. *Birth Defects*, 4, 116–121.
- Eliez, S. (2007). Autism in children with 22q11.2 deletion syndrome. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46, 433–434.
- Fine, S., Weissman, A., Gerdes, M., Pinto-Martin, J., Zackai, E., McDonald-McGinn, D., et al. (2005). Autism spectrum disorders and symptoms in children with molecularly confirmed 22q11.2 deletion syndrome. *Journal of Autism and Developmental Disorders*, 35, 461–470.
- Gothelf, D., Frisch, A., Michaelovsky, E., Weizman, A., & Shprintzen, R. (2009). Velo-Cardio-facial syndrome. *Journal of Mental Health Research in Intellectual Disabilities*, 2(2), 149–167.
- Greenberg, F. (1993). DiGeorge syndrome: An historical review of clinical and cytogenetic features. *Journal of Medical Genetics*, 30, 803–806.

- Grigorenko, E. L., Urban, A. E., & Mencl, E. (2010). Behavior, brain, and genome in genomic disorders: Finding the correspondences. *Journal of Developmental and Behavioral Pediatrics*, 31, 602–609.
- Hacıhamdioğlu, B., Hacıhamdioğlu, D., & Delil, K. (2015). 22q11 deletion syndrome: Current perspective. *The Application of Clinical Genetics*, 8, 123–132.
- Kinouchi, A., Mori, K., Ando, M., & Takao, A. (1976). Facial appearance of patients with conotruncal anomalies. *Journal of Pediatric Neurology*, 17, 84–87.
- Kobrynski, L. J., & Sullivan, K. E. (2007). Velocardiofacial syndrome, DiGeorge syndrome: The chromosome 22q11.2 deletion syndromes. *The Lancet*, 370, 1443–1452.
- Lindsay, E. A., Botta, A., Jurecic, V., Carattini-Rivera, S., Cheah, Y. C., Rosenblatt, H. M., et al. (1999). Congenital heart disease in mice deficient for the DiGeorge syndrome region. *Nature*, 401, 379–383.
- McDonald-McGinn, D. M., & Zackai, E. H. (2008). Genetic counseling for the 22q11.2 deletion. *Developmental Disabilities Research Reviews*, 14, 69–74.
- McDonald-McGinn, D. M., Kirschner, R., Goldmuntz, E., Sullivan, K., Eicher, P., Gerdes, M., et al. (1999). The Philadelphia story: The 22q11.2 deletion: Report on 250 patients. *Genetic Counseling*, 10, 11–24.
- McDonald-McGinn, D. M., Sullivan, K. E., Marino, B., et al. (2015). 22q11 deletion syndrome. *Nature Reviews Disease Primers*, 1, 15071.
- Meechan, D. W., Maynard, T. M., Gopalakrishna, D., Wu, Y., & LaMantia, A. S. (2007). When half is not enough: Gene expression and dosage in the 22q11 deletion syndrome. *Gene Expression*, 13, 299–310.
- Niklasson, L., Rasmussen, P., Oskarsdottir, S., & Gillberg, C. (2001). Neuropsychiatric disorders in the 22q11deletion syndrome. *Genetics in Medicine*, 3(1), 79–84.
- Polleux, F., & Lauder, J. M. (2004). Toward a developmental neurobiology of autism. *Mental Retardation and Developmental Disabilities Research Reviews*, 10, 303–317.
- Prasad, S. E., Howley, S., & Murphy, K. C. (2008). Candidate genes and the behavioral phenotype in 22q11.2 deletion syndrome. *Development Disabilities Research Reviews*, 14, 26–34.
- Roizen, N. J., Antshel, K. M., Fremont, W., AbdulSabur, N., Higgins, A. M., Shprintzen, R. J., et al. (2007). 22q11.2DS deletion syndrome: Developmental milestones in infants and toddlers. *Journal of Developmental and Behavioral Pediatrics*, 28, 119–124.
- Ryan, A. K., Goodship, J. A., Wilson, D. I., Philip, N., Levy, A., Seidel, H., et al. (1997). Spectrum of clinical features associated with interstitial chromosome 22q11 deletions: A European collaborative study. *Journal of Medical Genetics*, 34, 798–804.
- Scambler, P. J. (2010). 22q11 deletion syndrome: A role for TBX1 in pharyngeal and cardiovascular development. *Pediatric Cardiology*, 31, 378–390.
- Scambler, P., Kelly, D., Lindsay, E., Williamson, R., Goldberg, R., Shprintzen, R., Wilson, D., et al. (1992). Velo-cardio-facial syndrome associated with chromosome 22 deletions encompassing the DiGeorge critical locus. *The Lancet*, 339, 1138–1139.
- Schneider, M., Debbane, M., Bassett, A. S., Chow, E. W. C., Fung, W. L., & van den Bree, M. B. (2014). Psychiatric disorders from childhood to adulthood in 22q11.2 deletion syndrome: Results from the international consortium on brain and behavior in 22q11.2 deletion syndrome. *American Journal of Psychiatry*, 171, 627–639.
- Sedlackova, E. (1955). The syndrome of the congenitally shortening of the soft palate. *Casopis Lekaru Ceskych*, 94, 1304–1307.
- Shprintzen, R. (2000). Velo-cardio-facial syndrome: A distinctive behavioral phenotype. *Mental Retardation and Developmental Disabilities Research Reviews*, 6(2), 142–147.
- Shprintzen, R. (2008). Velo-cardio-facial syndrome: 30 years of study. *Developmental Disabilities Research Reviews*, 14, 3–10.
- Shprintzen, R., Goldberg, R., Lewin, M., Sidoti, E., Berkman, M., Argamaso, R., et al. (1978). A new syndrome involving cleft palate, cardiac anomalies, typical facies, and learning disabilities: Velo-cardio-facial syndrome. *The Cleft Palate Journal*, 15(1), 58–62.
- Simon, T., Bish, J., Bearden, C., Ding, L., Ferrante, S., Nguyen, V., et al. (2005). A multilevel analysis of cognitive dysfunction and psychopathology associated with chromosome 22q11.2 deletion syndrome in children. *Development and Psychopathology*, 17(3), 753–784.
- Swillen, A., Devriendt, K., Legius, E., Eyskens, B., Dumoulin, M., Gewillig, M., et al. (1997). Intelligence and psychosocial adjustment in velocardiofacial syndrome: A study of 37 children and adolescents with VCFS. *Journal of Medical Genetics*, 34(6), 453–458.
- Swillen, A., Devriendt, K., Vantrappen, G., Vogels, A., Rommel, N., Fryns, J., et al. (1998). Familial deletions of chromosome 22q11: The Leuven experience. *American Journal of Medical Genetics*, 80(5), 531–532.
- Swillen, A., Moss, E., & Duijff, S. (2018). Neurodevelopmental outcome in 22q11.2 deletion syndrome and management. *American Journal of Medical Genetics Part A*, 176(10), 2160–2166.
- Tang, K. L., Antshel, K., Fremont, W. P., & Kates, W. R. (2015). Behavioral and psychiatric phenotypes in 22q11.2 deletion syndrome. *Journal of Developmental and Behavioral Pediatrics*, 36, 639–650.
- Velo-Cardio-Facial Syndrome Educational Foundation, Inc. (2009). *What is Velo-cardio-facial syndrome?* Retrieved 10 Jan 2011 from <http://www.vcfsef.org>
- Vorstman, J., Morcus, M., Duijff, S., Klaassen, P., Heineman-deBoer, J., Beemer, F., et al. (2006). The 22q11.2 deletion in children: High rate of autistic disorder and early onset of psychotic symptoms. *Journal*

of the American Academy of Child and Adolescent Psychiatry, 45(9), 1104–1113.

Vortsman, J., Breetvelt, E., Duijff, S. N., Eliez, S., Schneider, M., & Jalbrzikowski, M. (2015). Cognitive decline preceding the onset of psychosis in patients with 22q11.2 deletion syndrome. *JAMA Psychiatry*, 72(4), 377–385.

Wang, P. P., Solot, C., Moss, E. M., Gerdes, M., McDonald-McGinn, D. M., Driscoll, D. A., et al. (1998). Developmental presentation of 22q11.2 deletion (DiGeorge/velocardiofacial syndrome). *Journal of Developmental and Behavioral Pediatrics*, 19, 342–345.

Developmentally Appropriate Practice

► Normalization

Developmentally Appropriate Practice (DAP)

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Synonyms

Chronological age appropriateness; Individual appropriates; Intervention targets and strategies that are related to increased quality of life outcomes

Definition

Developmentally appropriate practice (DAP) refers to providing intervention in a manner that is individually appropriate and culturally relevant for the learner. This term was first introduced by Sue Bredekamp and the National Association for

the Education of Young Children (NAEYC) in 1987 to warn early educators against the trend of pushing typically developing or gifted children too far too fast, or what some developmental psychologists referred to as “robbing children of their childhood” with deleterious effects that may not show up until adolescence or later. It also has important implications for working with students who have cognitive deficits so that families and interventionists interact with people in a manner that is age appropriate and provide opportunities to people that are both age appropriate and matched with individual strengths and preferences.

This concept of DAP is important when planning and implementing interventions for people with ASD. Developmentally appropriate interventions are those that take the student’s chronological as well as developmental age into consideration when identifying targets, materials, places, and strategies for intervention. Interventions that are developmentally appropriate also consider issues of cultural relevance and attempt to insure that the behaviors and skills selected as intervention targets are related to improving the quality of life for the person with ASD and his/her family.

See Also

- Curriculum
- Early Intervention
- Positive Behavior (a) Interventions and Supports (PBIS)

References and Reading

- Carr, E. G. (2007). The expanding vision of positive behavior support: Research perspectives on happiness, helpfulness, hopefulness. *Journal of Positive Behavior Interventions*, 9, 3–14.
- Copple, C., & Bredekamp, S. (2009). *Developmentally appropriate practice (in early childhood programs, serving children from birth through age 8)*. Washington, DC: NAEYC.
- Schwartz, I. S., & McBride, B. (2014). Getting a good start: Effective practices in early intervention. In K. D. Burton & P. Wolfberg (Eds.), *Educating learners on the*

autism Spectrum: Preparing highly qualified educators and related practitioners (2nd ed., pp. 82–105). Kansas City: Autism Asperger Publishing Company.

Schwartz, I. S., Ashmun, J., McBride, B., Scott, C., & Sandall, S. (2017). *The project DATA model for teaching preschoolers with autism*. Baltimore: Brookes.

Schwartz, I. S., Sandall, S. R., McBride, B. J., & Boulware, G. L. (2004). Project DATA (developmentally appropriate treatment for autism): An inclusive, school-based approach to educating children with autism. *Topics in Early Childhood Special Education*, 24, 156–168.

Developmental-Pragmatic Approaches/Strategies

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Definition

Developmental-pragmatic approaches to language intervention have a dual focus: (1) generating treatment goals and procedures based on the child's stage of development as determined by what is known about typical trajectories and (2) generating treatment goals and procedures based on the tenets of social-pragmatic perspectives on language acquisition and use. The child's developmental stage, rather than his or her chronological age, is considered not only in reference to language but also in reference to those areas of development thought to be precursors and cocursors of language, such as symbolic-cognitive (e.g., play abilities) and social-emotional functioning (e.g., interacting with others). Three tenets of social-pragmatic models of language acquisition serve as the foundation for language intervention which is derived from this thinking: (1) language develops within the context of the caregiver-child relationship, with an attuned and loving partner; (2) early developments in engagement, intentionality, and communication set the stage for the ability to comprehend and produce language; and (3) the use of language includes the capacity to know when to say what to whom.

Historical Background

The field of speech-language pathology was dramatically impacted by the introduction of social-pragmatic models of language acquisition in the 1970s and early 1980s (Bates 1976; Bates et al. 1975; Bruner 1975, 1977; Dore 1975; Halliday 1975; Prutting 1982). With these models, the world of communication disorders began to reconsider fundamental questions, such as what defines a language user and how does a language disorder compromise these capacities. Interest in constructs such as intentionality, nonlinguistic communication, functions of language, social interaction, contexts of language learning and language use, discourse, and conversational skills represented a departure from earlier thinking in both language acquisition and language intervention. Taxonomies from the world of typical social-pragmatic development began to appear in language assessment protocols and intervention plans for children with autism spectrum disorders. These included taxonomies of prelinguistic development (communicative intentions), speech acts and functions of language (labels, comments, requests, greetings, etc.), and conversational analysis (initiating and maintaining topics, turn-taking, and contingency). These paradigms were immediately of interest to speech-language pathologists (SLPs) working with children with autism spectrum disorders because for the first time, the nature of these children's language and communication challenges was more adequately described and understood.

Once the breadth of pragmatic models of language acquisition and language use were integrated into the world of communication disorders, the work of speech-language pathologists expanded to include new dimensions of typical language development and language use and greater attention to the contexts of language learning and language use. The earlier "semantic revolution" which brought us from "form" (i.e., the structure of language) to "content" (i.e., the meanings of language) led quite quickly to a "pragmatic revolution" which required professionals to once again rethink the nature and the boundaries of their work as they added a third component to

the definition of language, namely, “use.” Perhaps as important was the shift in thinking about the role of the SLP, from the teacher of language to the partner in the language acquisition process. Encouraging the child’s initiation and intentionality was seen as more important than obtaining responses from the child. Finally, the notion of therapy contexts expanded to potentially include all the contexts of the child’s life, from home to school to play, with a variety of partners, both in terms of number and age (from one adult to groups of peers).

The focus on the interpersonal functions of language was particularly attractive to clinicians who were working with children who could talk but did not use language appropriately for the typical range of communicative intentions (commenting, reporting, requesting answers). In fact, the new taxonomies provided ways of understanding the unconventional linguistic patterns used by some children on the autism spectrum. Prizant and Duchan (1981) noted that children with autism were often expressing typical functions of language with echolalic utterances. Obviously, the relationship between form and function was idiosyncratic (e.g., the child says “Do you want a drink?” to indicate that he or she wants a drink), but nonetheless, the utterances were intentional and certainly could not be simply discounted as inappropriate. The “discovery” that echolalic language was intentional led to shifts in intervention procedures from those which suggested ignoring or discouraging echolalia to those that honored it. The primacy of function over form, a direct outgrowth of pragmatic models, was a theme that resonated with SLPs who were working with children who were nonverbal (e.g., standing in the corner to communicate anxiety) and/or using unconventional means of communication (e.g., repetitively asking questions to deal with transitions).

Although the impact of developmental-pragmatic models of language acquisition had a lasting effect on language assessment and intervention with both children and adults with a range of language disorders, many would agree that speech and language intervention with children with autism spectrum disorders was particularly

effected by embracing this model. The nature and breadth of pragmatic views of language spoke directly to the nature and breadth of the children’s challenges, providing new directions for SLPs working with this group of children.

Rationale or Underlying Theory

A developmental-pragmatic model (DPM) rests on the universals, processes, and facts of typical language acquisition. This information clarifies what is learned by a typically developing child at each point in development, resulting in the child’s acquisition of a symbolic language system and his or her success as a social communicator. The speech-language pathologist who embeds his or her thinking in this theory believes that the language acquisition of children on the autism spectrum includes both typical and atypical parameters, all of which are best understood by reference to the universals of speech, language, and communication development.

Developmental-pragmatic models of language intervention pay particular attention to the social underpinnings of language acquisition and use. While encompassing the interest in the form of language (phonology, morphology, and syntax) and the content of language (semantics), pragmatic thinking leads us to consider the broader interpersonal context of language acquisition and the early ability of the communicator to use gestures, facial expressions, and words for social interaction purposes. Thus, the emphasis on context, both of language learning (i.e., caretaker-child interactions) and language use (e.g., how the intent of our utterances is understood depending on who is being spoken to, what the setting is, and the knowledge of the participants), is considered developmentally from birth to adulthood. The fact that some children on the autism spectrum have strengths in form and content with considerable deficits in pragmatics speaks to the components of language necessary to be a successful and conventional language user.

Regardless of the specific discipline, developmental approaches begin with the assumption that all strategies of intervention, regardless of the

target group or desired outcome, can be derived from normative theories of development. That is to say, the general principles of development apply to all children independent of their biological variability or the range of environments in which they live (National Research Council and Institute of Medicine 2000). We might add to this that goals of intervention can best be derived from normative theories of development as well and that this is particularly compelling when considering children with deficits in social-pragmatic aspects of language.

This overarching understanding of developmental approaches paired with the specifics of pragmatic thinking served as a rich resource for recasting the assessment and intervention of children on the autism spectrum. The emphasis in pragmatic approaches on the intersect between the capacity to interact and the capacity to comprehend and produce language immediately resonated with the challenges and needs of children on the autism spectrum. The models presented in the literature by Bates et al. (1975); Dore (1974, 1975); Halliday (1975); Bloom et al. (1976); Snow (1973, 1978); etc., offered the theory and frameworks for reconsidering what and how we were teaching children with autism spectrum disorders to learn and use language. SLPs began to use and adapt the taxonomies which appeared in the research literature on topics such as the speech acts expressed by young children using single words, the early development of conversational skills, and the nature of adult input to language-learning youngsters.

As this movement continued and moved more deeply into the world of joint attention, early relationships, and affective range (Tomasello 1988; Greenspan and Wieder 1998; Mundy and Sigman 2006), speech-language pathologists continued to integrate the expanding theory into their work with children with challenges (Prizant et al. 2006). More and more intervention programs began to address the social engagement issues of children on the autism spectrum, and, in fact, approaches which differed considerably in the strategies and contexts of intervention now share the emphasis on interaction, reciprocity, and shared attention. The fact that typically

developing infants can easily participate in attuned, communicative exchanges and that children with autism spectrum disorders find this developmental step so challenging is a universal concern in the educational and therapeutic planning for children on the autism spectrum.

Once again, while many of the pragmatic constructs speak to the process of language acquisition and, thus, are relevant for intervention programs for all children with delays and disorders in speech and language development, the nature of autism spectrum disorders has led to a particular interest in this body of work.

The following list of principles reflect the underlying theory of a developmental-pragmatic model of language intervention (Gerber 2003) and are thought to serve as the basis for and rationale for intervention goals and procedures:

- Language is learned in the context of spontaneous, natural everyday interactions between the caregiver and the child.
- The child's language acquisition is embedded in his or her cognitive, affective, and social development and life.
- The development of communicative intentionality precedes the development of language.
- Prelinguistic developments in cognitive, social, emotional, and communicative domains precede the comprehension and production of language.
- The child's communicative interactions include many opportunities to play speaker-initiator and listener-responder discourse roles.
- Both typically and many atypically developing children move through the same general stages of linguistic and communicative development.
- The specifics of language development (e.g., rate, style, strengths) are characterized by individual variation.
- Imitation may play a role in language learning for some children; its role in the development of communication is generally recognized.
- The child plays an active role in his or her language development – meaningful and joyful interactions with the world of objects and the world of people serve as the context for the development of language.

Goals and Objectives

Operationalizing developmental-pragmatic goals and objectives can be thought of in a number of ways, all of which differ from traditional perspectives on how to view the basic components of language intervention. These components include what the goals of intervention are, who the participants are during intervention, what procedures should be used, where the therapy takes place, and what role the adult plays during the interaction. A continuous thread from paradigms of assessment to paradigms of intervention are characteristic of language intervention programs which are based on developmental social-pragmatic theories of language acquisition (Gerber and Prizant 2000; Prutting and Kirschner 1987).

One of the most significant impacts of pragmatic models on the world of language disorders was the rethinking of the intervention goals addressed with children who had challenges in the acquisition of speech, language, and communication. The fact that nonverbal communication including gestures, facial expression, body language, and vocalizations were now considered appropriate goals of intervention represented one significant departure from earlier views which focused on the production of words and sentences. For those children who had not yet developed the capacity to communicate through nonlinguistic forms, the importance of that step in the developmental trajectory on the way to language was more fully recognized. In fact, the notion that nonlinguistic communication continues to be a goal of therapy even for children who are verbal was welcomed by clinicians who were working with children on the autism spectrum who could talk but did not use pointing and showing, eye gaze, and intonation to communicate their intentions.

Further, for children who are at very early developmental stages, pragmatic goals address the social-emotional precursors to language. These include increasing engagement in back and forth adult-child interactions; facilitating affective exchanges between the adult and the child using nonlinguistic communicative forms;

increasing periods of joint attention with caregivers; communicating a range of intentions using differentiated vocalizations, pointing, eye gaze, and word approximations; and facilitating social referencing. Of course, precursory goals related to the content of language and the form of language would also be a part of every child's intervention plan. At these early stages, goals for the caregivers include increasing their responsiveness to the child's potentially communicative attempts and fostering reciprocal interactions, using the child's current repertoire of behaviors.

Of particular relevance for children with autism spectrum disorders was the notion of intentional communication and functions of language which moved to center stage in language intervention as a result of the understanding of pragmatic models of language development. The fact that this group of children did not use their non-linguistic or linguistic systems to communicate a range of intentions had been documented in the research and confirmed by clinical experience. Thus, rather than moving on to the development of larger vocabularies and longer sentences, clinicians began to facilitate the use of the children's existing systems for functions beyond requests, such as greetings, comments, and social routines. The idea that children needed to acquire not only the forms of language but also the interpersonal functions led to an expansion of goals and objectives that has had considerable longevity.

For children whose nonlinguistic and linguistic systems are somewhat unconventional, intervention goals begin with an attempt to analyze the form-function relationship in that child's system. One finding from this type of analysis has been that a child who does not use conventional language may very well be communicating intentions. Unconventional behaviors, echolalia, delayed echolalia, and scripts are often attempts to initiate conversation and/or to communicate particular functions and meanings of language. In terms of a less conventional nonlinguistic system, if a child's tendency to put his or her face close to that of another person is seen as an attempt to start an interaction, the function of the behavior can be acknowledged while more conventional forms are modeled ("let's play").

Responding in this way to a child's behaviors, with an eye toward the function they may serve, came from the focus on function in the pragmatic analysis of language and communication. Similarly, when working with a child who was using a less conventional linguistic system, clinicians began to understand the importance of imbuing the child's echolalic utterances and scripting with communicative intent. This response to the child turns a somewhat ambiguous communicative moment into a productive one and again illustrates how goals informed by pragmatic thinking were drastically different from more traditional ones.

Developmental-pragmatic goals are determined by assessing the child's developmental stage of language acquisition and strengths and challenges in social-pragmatic domains of development. Some examples of typical developmental-pragmatic goals for children functioning at earlier stages of development might include the following, written from the perspective of what the SLP will focus on:

- To facilitate the child's interpersonal engagement and emotional range (e.g., happy, sad, curious, frustrated, angry), with nonlinguistic and/or linguistic forms of communication
- To facilitate the child's participation in joint attention interactions with an adult, with nonlinguistic and/or linguistic forms of communication
- To facilitate the child's range of communicative intentions, with nonlinguistic and/or linguistic forms of communication
- To facilitate the use of spontaneous, self-initiated communication, with nonlinguistic and/or linguistic forms
- To expand the range of forms used to communicate, including both nonlinguistic forms (gestures, signs, visual systems) and linguistic forms (vocalizations, intonation patterns, words, utterances)
- To facilitate the use of social referencing, with nonlinguistic and/or linguistic forms of communication
- To facilitate the child's intention to communicate in a range of naturalistic contexts

(at home, on the playground, with adults, with peers)

- To respond to all of the child's attempts to communicate whether they are conventional or unconventional

For children at higher developmental stages of language, typical developmental-pragmatic goals might include:

- To facilitate participation in conversational exchanges, playing both the speaker and the listener roles
- To facilitate the use of contingent responses during conversational exchanges
- To facilitate an understanding of the listener's perspective and the need to modify one's nonlinguistic and linguistic communication for a range of partners
- To facilitate the ability to repair communication breakdowns
- To facilitate peer interactions, first in dyadic interactions and eventually in larger groups
- To facilitate the coordination of conventional nonlinguistic and linguistic systems to communicate intentions

It should be noted that depending on the child's developmental stage of language, simultaneous goals addressing the comprehension and production of language would be included in an integrated plan of language intervention. Similarly, social-emotional, cognitive-symbolic, and regulation goals will necessarily be considered in all intervention plans that are addressing the further development of language and communication.

Treatment Participants

One of the most vivid and lasting effects of the pragmatic revolution on the field of speech and language was the change of thinking about who the treatment participants should be during intervention. Here, again, this notion had particular resonance for children on the autism spectrum because of the nature of their difficulties in interpersonal interactions.

With the early and continuing interest in pragmatic models and social-emotional approaches to working with children with developmental challenges, SLPs have been exposed to a deepened understanding of the nature of the caregiver-child relationship. This relationship sets the stage for the child's healthy development in all areas of functioning, including the development of the comprehension and production of language. SLPs began to think not only of *what* was learned in the prelinguistic period but *who* was propelling the development and why this relationship was key to the process. The notion that more of the "work" in language intervention should be done with the mother or primary caregiver and the child, rather than the therapist and the child, continues to be difficult to realize during intervention and, yet, is a clear implication of pragmatic models of language acquisition. Even in settings where it is easier to work with the caregiver, such as in early intervention conducted in the child's home, practitioners are not necessarily comfortable with the idea of "coaching" a parent during an interaction and, often, prefer to have the parent observe as the therapist interacts with the child. While understandable, this is not in sync with the research now spanning more than 30 years, suggesting that the caregiver-child interaction is where the "action" is relative to setting the stage for development.

Further, because pragmatic models of language acquisition underscore the fact that language use occurs across contexts with different partners, language intervention which has its roots in this model embraces the notion that the child's ability to use language must be addressed in a range of real-life situations, including his or her other interaction with family, teachers, and peers. Remembering that pragmatics refers to the ability to know what to say when to whom, pragmatic interventions go beyond the traditional therapy room and the SLP-child interaction. The child's interactions with typical and atypical peers must be built into the intervention planning. In fact, quite a few programs have been developed where typical peers coach their classmates who are on the

autism spectrum to enhance the possibility of more frequent and successful exchanges (Kohler et al. 2005).

Prior to the introduction of developmental-pragmatic models, the fact that a successful language user can communicate effectively with many different partners was not recognized as a potential language intervention goal. This notion led to one of the most significant shifts in the intervention paradigms of speech-language pathologists. Improving the child's ability to communicate with different partners requires the SLP to consider the child's interactions with every person in his or her life and to potentially use these interactions as the contexts for language therapy.

Treatment Procedures

The use of developmental-pragmatic models to generate treatment procedures requires an understanding of the way language acquisition progresses and language use is realized in authentic communicative contexts. As mentioned in the previous section, implications from a developmental-pragmatic model affect decisions about all the components of therapy, not only what the goals of intervention are and who the participants are during intervention but also what procedures will be used during treatment. In this discussion, intervention procedures include intervention strategies, intervention contexts, and the role of the adult during the interaction.

The following list captures the nature of *strategies* generated from developmental-pragmatic models of language use:

- Provide many opportunities to facilitate sustained engagement and reciprocal interactions, both nonlinguistically and linguistically.
- Provide many opportunities to expand shared attention and more consistent responsiveness to the communicative partner.
- Interpret all of the child's behaviors, conventional and unconventional, as intentional and meaningful.

- Maintain a reciprocal flow by treating all of the child's behaviors as communicative as you alternate turns in the interaction.
- Model the use of nonlinguistic and linguistic forms of communication to express the child's meanings, messages, and intentions, not yours.
- Engineer the context so that the child has an opportunity to play both initiator and responder roles in the "conversation."
- Engineer "turn-taking," by expectancy, waiting, and nonverbal communication.
- Teach language within the context of natural interactions and discourse.
- Embed language training in contexts that are familiar to the child relative to meaning and affectively laden.
- Reduce the complexity of your language input while maintaining the "grammar" of language, the melody, and the interactive flow of communication.
- Pair language with the child's actions, interests, agenda; timing and contextual support are critical at early stages of language learning.
- Join in the meaning and affective tone of the child's script.
- Teach language when the cognitive, social, affective, and pre- and/or corequisites are in place.
- Teach the child's parents, teachers, and therapists how best to facilitate language throughout the child's daily life.
- Teach language with words and silence.
- Teach language when the interaction is flowing.
- Teach language by matching and meeting the child and then "up the ante."
- Teach pragmatic skills within the context of natural conversation.
- Engineer natural opportunities for promoting nonverbal pragmatic abilities such as proxemics, body language, gestures, and facial expressions.

In reference to intervention *contexts*, pragmatic views of language suggest that all the contexts of a person's life are of interest when we are studying and supporting language use. Much like the idea of different partners, pragmatic models are rooted

in the idea of the range of contexts which make up the person's life and the variations in both non-linguistic (setting, activity, participants) and linguistic (prior discourse) aspects of each context. As a result, attempts were made to expand the notion of "therapy context" to include those activities that reflected the child's typical day (lunch, recess, soccer, classroom, etc.).

Understanding that every moment is a potential language intervention moment pushed the idea of context in language treatment beyond the previous discrete boundaries. In this view, every interaction offers the child an opportunity to learn about language use and to practice how to be a successful language user. For example, a 10-year-old child on the autism spectrum, M., who the author sees for language intervention, was interested in interacting with a 2-year-old boy, J., who he met each week in the waiting room of the clinic. In his attempt to interact with this toddler, M. often hugged J. a bit too vigorously or spoke to him a bit too loudly. Needless to say, the 2-year-old moved away from M. and backed into his caregiver's lap. In order to help M. express his natural interest in interacting with the young child and to have a more successful experience, the clinician practiced with him how he might approach the boy (i.e., from a distance) and what he might say to him (e.g., show him a toy). In fact, from a pragmatic point of view, the SLP welcomes the opportunity to see how the child functions in different contexts in his or her attempt to make the therapeutic experience representative of the child's typical interactions and to address those missteps that may be interfering with his positive opportunities for interaction.

Finally, in reference to the *role of the adult* during the interaction, pragmatic models of language acquisition and use suggest that the clinician assume more of a partner and less of a teacher role during intervention. The adult is seen as sharing the communicative interaction with the child, not directing it. Often, the idea of "following the child's lead" is used to help parents and professionals understand that the child's ideas and intentions should take precedence over the adult's. With the goals in mind, the adult will support, scaffold, and facilitate development, always

beginning from where the child is and engaging in a dynamic that is more reciprocal and less adult led. The full impact of a developmental-pragmatic approach can be seen as therapy sessions which reflect this thinking, where adults are watching, waiting, observing, and then determining based on the child's actions, behaviors, vocalizations, body movements, and words how the next steps in development can be encouraged.

Efficacy Information

Although many language intervention programs integrate aspects of developmental-pragmatic models of language, two programs in particular reflect the basic tenets of this perspective. The Hanen Program, *More Than Words* (Sussman 1999), focuses on teaching parents to use responsive strategies that promote social interaction and, ultimately, language development. The fact that the training is designed to support the primary caregivers in their interactions with their children places this program at the heart of pragmatic thinking. Recent studies (McConachie et al. 2005; Girolametto et al. 2007) have shown that parents who participated in the *More Than Words* program used more responsive interaction strategies than the control group of parents. Moreover, gains in vocabulary, frequency of communication, and/or participation in turn-taking routines were noted in the children.

Other studies have also investigated the effects of responsiveness training on the caregivers' interactive style and the resulting effects on their child's social interaction and communication. Specific improvements in joint attention, initiation of communication, periods of engagement, and expressive language have been noted in children whose parents became more responsive (Aldred et al. 2004; Baker et al. 2010; Mahoney and Perales 2003, 2005; Siller and Sigman 2002).

The SCERTS (Social Communication, Emotional Regulation, and Transactional Support) program developed by Prizant et al. (2006) represents an educational program which embraces the core components of developmental-pragmatic frameworks. This program emphasizes

functional, developmentally appropriate goals and objectives which are addressed in meaningful and purposeful activities throughout the child's day. The child's individual differences, including learning style and interests, are embraced; the family, educators, and clinicians are seen as a collaborative team.

The description of social communication in the SCERTS program (Prizant et al. 2006) as the "development of spontaneous, functional communication, emotional expression, and secure and trusting relationships with children and adults" speaks to the priorities of all pragmatically oriented programs. Similar to *More Than Words* (Sussman 1999), the fact that the family and professionals are taught to respond to the child's needs and interests is derived from the focus in developmental-pragmatic models on the partner's roles in the child's acquisition of language. Prizant et al. (2010) provide many references to support the positive outcomes of training in social communication, with approaches that range from those that are more behavioral to those that are more representative of developmental-pragmatic ones (Kaiser et al. 2000; Wetherby and Woods 2006).

Outcome Measurement

Measuring progress in pragmatic goals and objectives has always presented its own set of challenges for SLPs. The very nature of pragmatics speaks to the way language use varies relative to changing aspects of the context such as the participants, the setting, nonlinguistic supports, ongoing discourse, etc., making measurement for this area of language more complicated than others. Perhaps, the best way to think about the emergence of pragmatic behavior is relative to a continuum of contexts of the child's life, with outcomes measured within specific contexts (e.g., the child's initiation of communication with one particular peer during toy play). From a pragmatic perspective, intervention progress can only be thought of with an understanding of the dimensions of natural contexts and real-life partners.

The outcome measurements of developmental-pragmatic interventions span a wide range of behaviors. Unlike most other approaches, these include both the child's and the partner's behaviors, as the pragmatic approach is anchored in the caregiver's role in creating interactive exchanges. In fact, the responsiveness of the caregiver to the child's behaviors is seen as one of the most important aspects of interaction to measure. Given the underlying theory of typical language learning, clinicians who are working from this framework will want to track the parent's ability to sensitively respond to all of the child's communicative attempts (not just those that involve spoken language). Parent responsiveness provides more opportunities for social interaction and, ultimately, the acquisition of language.

All of the goals and objectives indicated in the previous section are easily translated into outcomes to measure (occasions of intentional communication, use of a range of speech acts, ability to engage in turn-taking exchanges, etc.). Although behavioral principles could be used to conceptualize how to measure a new behavior (e.g., 80% criterion), a developmentalist may be more comfortable with a continuum of criteria, ranging from "emerging" to "achieved." Developmental thinking implies that measures will mirror how typical development proceeds gradually over time rather than thinking in terms of the use of a particular behavior in 8 out of 10 trials.

Developmental-pragmatic models rely heavily on checklists of targeted behaviors, questionnaires, naturalistic observation, language sampling, and semistructured assessment to measure the child's progress in selected goals and objectives. The frequency of assessment will vary with the program and the system, with some measuring outcomes on a daily or weekly basis, and others over a longer span of time. When outcome measurement is being used to determine the success of a particular step in the program, more frequent assessment leads to more frequent modification of the parameters of the treatment plan. Finally, the SLP who is working from a developmental-pragmatic framework will want to periodically measure the child's

progress across the contexts of his or her life, as a reflection of the ability to use language in the learning and social interactions that make up his or her day.

Qualifications of Treatment Providers

The majority of treatment providers for developmental-pragmatic approaches to language intervention are speech-language pathologists. Those SLPs who are working directly with parents will need additional training in how to teach strategies and procedures for affecting change in the caregivers' interactive styles (e.g., Hanen programs). Other more broadly based developmentally oriented models, such as the Developmental, Individual Difference, Relationship-Based (DIR) approach (Greenspan and Wieder 1998), include training components in their certificate process for professionals from a range of disciplines who want to learn how to "coach" parents effectively. Once SLPs begin working with parents closely, they are often aware of the need for further training in counseling in order to deal with the emotional issues typically and understandably raised by the caregivers.

In addition, SLPs working in this model often collaborate with teachers to help them implement a developmental-pragmatic approach in the classroom. The SLP will be called on to help other professionals shift their thinking to implement the goals and strategies in the contexts of the child's academic and social life. Here, again, the developmental-pragmatic approach requires additional programming and planning on the part of the SLP as the work moves beyond the therapy walls and out into the child's everyday world.

See Also

- [Developmental Intervention Model](#)
- [Pragmatic Language Impairment](#)
- [Pragmatic Language Skills Inventory](#)
- [Pragmatic Rating Scale](#)
- [Social Interventions](#)

References and Reading

- Aldred, C., Green, J., & Adams, C. (2004). A new social communication intervention for children with autism: Pilot randomized controlled treatment study suggesting effectiveness. *Journal of Child Psychology and Psychiatry*, 40, 1–11.
- Baker, J., Messinger, D., Lyons, K., & Grantz, C. (2010). A pilot study of maternal sensitivity in the context of emergent autism. *Journal of Autism and Developmental Disorders*, 40, 988–999.
- Bates, E. (1976). *Language and context: The acquisition of pragmatics*. New York: Academic Press.
- Bates, E. C., Camaiono, L., & Volterra, V. (1975). The acquisition of performatives prior to speech. *Merrill-Palmer Quarterly*, 21, 205–216.
- Bloom, L., Rocissano, L., & Hood, L. (1976). Adult-child discourse: Developmental intervention between information processing and linguistic knowledge. *Cognitive Psychology*, 8, 521–552.
- Bruner, J. (1975). The ontogenesis of speech acts. *Journal of Child Language*, 2, 1–19.
- Bruner, J. (1977). Early social interaction and language acquisition. In R. Schaffer (Ed.), *Studies in mother-infant interaction* (pp. 271–289). New York: Academic Press.
- Dore, J. (1974). A pragmatic description of early language development. *Journal of Psycholinguistic Research*, 4(4), 343–350.
- Dore, J. (1975). Holophrases, speech acts, and language universals. *Journal of Child Language*, 2, 21–40.
- Gerber, S. (2003). A developmental perspective on language assessment and intervention for children on the autistic spectrum. *Topics in Language Disorders*, 23(2), 74–95.
- Gerber, S., & Prizant, B. (2000). Speech, language, and communication assessment and intervention for children. In *Clinical practice guidelines: Redefining the standards of care for infants, children, and families with special needs*. Bethesda: ICDL Press.
- Girolametto, L., Sussman, F., & Weitzman, E. (2007). Using case study methods to investigate the effects of interactive intervention for children with autism spectrum disorders. *Journal of Communication Disorders*, 40, 470–492.
- Greenspan, S., & Wieder, S. (1998). *The child with special needs*. Reading: Addison Wesley Longman.
- Halliday, M. A. K. (1975). *Learning how to mean: Explorations in the development of language*. London: Edward Arnold.
- Kaiser, A., Hancock, T., & Nietfeld, J. (2000). The effects of parent-implemented enhanced milieu teaching on the social communication of children who have autism. *Early Education and Development*, 11, 423–446.
- Kohler, F. W., Strain, P. S., & Goldstein, H. (2005). Learning experiences... An alternative program for pre-schoolers and parents: Peer-mediated interventions for young children with autism. In E. D. Hibbs & P. S. Jensen (Eds.), *Psychosocial treatments for child and adolescent disorders: Empirically based strategies for clinical practice* (2nd ed., pp. 659–687). Washington, DC: American Psychological Association.
- Mahoney, G., & Perales, F. (2003). Using relationship-focussed intervention to enhance the social-emotional functioning of your children with autism spectrum disorders. *Topics in Early Childhood Special Education*, 23, 77–89.
- Mahoney, G., & Perales, F. (2005). Relationship-focused intervention with children with pervasive developmental disorders and other disabilities: A comparative study. *Developmental and Behavioral Pediatrics*, 26, 77–85.
- McConachie, H., Randle, V., & Couteur, L. (2005). A controlled trial of a training course for parents of children with suspected autism spectrum disorder. *Journal of Pediatrics*, 147, 335–340.
- Mundy, P., & Sigman, M. (2006). Joint attention, social competence and developmental psychopathology. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology, theory and methods* (2nd ed.). Hoboken: Wiley.
- National Research Council and Institute of Medicine. (2000). *From neurons to neighborhoods: The science of early childhood development*. Washington, DC: National Academy Press.
- Prizant, B., & Duchan, J. (1981). The functions of immediate echolalia in autistic children. *The Journal of Speech and Hearing Disorders*, 46, 241–250.
- Prizant, B., Wetherby, A., Rubin, E., & Rydell, P. (2006). *The SCERTS model: A comprehensive approach for children with autism spectrum disorders*. Baltimore: Paul H. Brookes.
- Prizant, B., Wetherby, A., Rubin, E., & Laurent, A. (2010). The SCERTS model and evidence-based practice. www.scerts.com/docs/SCERTS_EBP%20090810%20v1.pdf.
- Prutting, C. (1982). Pragmatics as social competence. *The Journal of Speech and Hearing Disorders*, 47, 123–134.
- Prutting, C., & Kirschner, D. (1987). A clinical appraisal of the pragmatic aspects of language. *The Journal of Speech and Hearing Disorders*, 52, 105–119.
- Siller, M., & Sigman, M. (2002). The behaviours of parents of children with autism predict the subsequent development of their children's communication. *Journal of Autism and Developmental Disorders*, 32(2), 77–89.
- Snow, C. (1973). Mother's speech to children learning language. *Child Development*, 43, 549–565.
- Snow, C. (1978). The conversational context of language acquisition. In R. Campbell & P. Smith (Eds.), *Recent advances in the psychology of language* (Vol. 4a, pp. 253–269). New York: Plenum Press.
- Sussman, F. (1999). *More than words: Helping parents promote communication and social skills in children with autism spectrum disorder*. Toronto: The Hanen Centre.

Tomasello, M. (1988). The role of joint attentional processes in early language development. *Language Sciences*, 10, 69–88.

Wetherby, A., & Woods, J. (2006). Early social interaction project for children with autism spectrum disorders beginning in the second year of life: A preliminary study. *Topics in Early Childhood Special Education*, 26(2), 67–82.

Deviant Sexual Behavior

► [Sexuality and Problem Behaviors](#)

Dexamfetamine

► [D-Amphetamine](#)

Dexedrine

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Synonyms

[Adderall](#); [Dextroamphetamine](#)

Definition

Dexedrine is a stimulant medication useful for the treatment of ADHD symptoms and narcolepsy. It is available in tablets or extended-release capsules.

Side effects tend to be mild and include insomnia, loss of appetite, weight loss, headaches, dry mouth, and erectile dysfunction. It can also produce transient increases in blood pressure and

may have an effect on seizure threshold and certain heart arrhythmias.

See Also

► [Dextroamphetamine](#)

References and Reading

Handen, B. L., Taylor, J., & Tumuluru, R. (2011). Psychopharmacological treatment of ADHD symptoms in children with autism spectrum disorder. *International Journal of Adolescent Medicine and Health*, 23(3), 167–173.

Dextroamphetamine

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See Also

- [Adderall](#)
- [Dexedrine](#)

References and Reading

- Handen, B. L., Taylor, J., & Tumuluru, R. (2011). Psychopharmacological treatment of ADHD symptoms in children with autism spectrum disorder. *International Journal of Adolescent Medicine and Health*, 23(3), 167–173.

Diagnosis

- [DSM-5](#)

Diagnosis and Classification

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Definition

As used in medicine (including psychiatry), a diagnosis is determined as part of a diagnostic process in the attempt to identify a specific disorder. Diagnoses are used in many ways and for different purposes. As both a term and a process, the issue of diagnosis is very much related to issues of classification. Indeed, the word diagnosis comes through Latin and Greek sources which have to do with understanding/distinguishing things. In clinical medicine, the assignment of a diagnosis often involves various tests, examinations, and so forth; the diagnosis typically guides treatment. In addition, diagnoses have other uses, e.g., in public health, in establishing eligibility for services, and so on. Some special issues arise with

respect to psychiatric diagnosis and are discussed subsequently (see also ► [“DSM-IV”](#) entry).

Historical Background

Although diagnoses have been used since antiquity, it was only as the causes of various medical illness began to be identified in the 1800s that attempts were made to study the issue more systematically, e.g., in relation to causes of death. This effort took place both in Europe and the USA originally focusing on causes of mortality but gradually expanding to include a range of diseases and injuries. This effort results in what is now the *International Classification of Diseases* (ICD-10). In psychiatry, early efforts to assign diagnoses were limited and classification schemes highly theoretical in nature; this limited their use more generally and with clinicians who did not share similar theoretical orientations. This shifted dramatically with the 3rd edition of the American Psychiatric Association's *Diagnostic and Statistical Manual* (DSM-III) (American Psychiatric Association 1980) which adopted an atheoretical approach and which quickly came to dominate psychiatric diagnosis throughout the world.

Current Knowledge

Diagnosis is intimately related to issues of classification. The tendency to engage in the latter activity is an intrinsically human activity that has the potential to facilitate observation and then help generate general principles and hypotheses. When approaches to classification are shared, communication is enhanced. In medicine in particular, the assignment of some specific label to a condition may itself be a source of relief to the patient or family members since it is often (mistakenly) assumed that having a label implies an understanding of etiology and specific treatments. As with any human construction, diagnostic labels can be misused. While official systems like DSM-IV or ICD-10 tend to be organized

around categories, other approaches, e.g., using dimensions of function/dysfunction, could also readily be used. Classification systems vary depending on their purpose but to be generally useful that must be amenable to ready and reliable use by a range of individuals. In the past, theoretically based approaches to classification were common but now have given way to more “phenomenologically based” approaches.

A number of misconceptions regarding issues of diagnosis and classification should be noted: (1) by itself, deviant behavior does not need to imply a disorder, and (2) diagnoses do not necessarily have to have a biological base even when symptoms are expressed somatically (e.g., maladaptive personality traits can be a disorder, and severe psychological stress can give rise to a range of persistent physical symptoms).

Issues for classification arise from numerous sources. One has to do with the primary goal(s) of classification (e.g., to enhance research or to facilitate clinical work). Also, there are some special issues for classification in relation to difficulties of childhood onset. The two major classification systems for psychiatric and developmental disorders (DSM-IV and ICD-10) adopt approaches that are in some ways similar and in other ways quite different. Although it is often assumed that some ideal classification system must exist in reality, many different factors impact approaches to diagnosis. To complicate things further, different apparent etiologies might result in rather similar clinical pictures, while sometimes the same etiological factor is associated with a wide range of clinical outcomes; often intervention is much more concerned with the expression of the clinical problem rather than its cause. With a few interesting exceptions (e.g., reactive attachment or post-traumatic stress disorders in DSM-IV), etiologies have typically not been specified.

Difficulties of childhood onset present special problems for classification and diagnosis. Developmental factors must be considered, e.g., in relation to the ways they may impact symptom expression or in the ways the symptoms may interfere with development. The use of a multi-axial approach helps in dealing with this problem. In the past, theoretically based approaches to classification

were common but now have given way to more “phenomenologically based” approaches.

Contextual factors are of great importance in understanding the clinical expression of conditions in children, i.e., family, school, and ethnic or cultural background may significantly impact the clinical presentation. These issues are often most complicated in very young children where disentangling child-maternal difficulties can sometimes be quite difficult. It should also be noted that disorders not individuals are classified (failure to do this results both in problems of stigmatization and potential adverse effects of labeling).

Having one problem may increase risk for other difficulties (what is termed comorbidity). It has been noted that for individuals with intellectual disability, there has often been a tendency to overlook other problems (what is termed “diagnostic overshadowing”). There are different approaches to the problem of comorbidity, and the problem is a special challenge for childhood-onset disorders since having one problem may contribute to risk for another one.

Future Directions

Particularly in the area of psychiatry, DSM-III marked a watershed event in improving diagnostic reliability and significantly advanced research in the field. In autism and related disorders, similar changes have occurred with diagnostic systems becoming more and more data based and, in turn, more likely to advance research in general. New knowledge in the areas of genetics, biological models, and identification of end phenotypes or intermediate endophenotypes may further advance work on these disorders. As noted in detail elsewhere (e.g., Smith et al. 2015), the new DSM-5 definition has resulted in a significant narrowing of the diagnostic concept.

See Also

- [Comorbidity](#)
- [DSM-5](#)

- ▶ DSM-III
- ▶ DSM-III-R
- ▶ DSM-IV

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual* (3rd ed.). Washington, DC: APA Press.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.). Washington, DC: APA Press.
- First, M. B., & Pincus, H. A. (2002). The DSM-IV text revision: Rationale and potential impact on clinical practice. *Psychiatric Services*, 53(3), 288–292.
- Hobbs, N. (Ed.). (1975). *Issues in the classification of children*. San Francisco: Jossey-Bass.
- Klin, A., Lang, J., Cicchetti, D. V., & Volkmar, F. R. (2000). Brief report: Interrater reliability of clinical diagnosis and DSM-IV criteria for autistic disorder: Results of the DSM-IV autism field trial. *Journal of Autism and Developmental Disorders*, 30(2), 163–167.
- Kupfer, D. A., First, M. B., & Regier, D. A. (2002). *A research agenda for DSM-V*. Washington, DC: American Psychiatric Publishing.
- Ritvo, E. R., & Freeman, B. J. (1977). National society for autistic children definition of the syndrome of autism. *Journal of Pediatric Psychology*, 2(4), 142–145.
- Rutter, M. (1978). Diagnosis and definition of childhood autism. *Journal of Autism and Childhood Schizophrenia*, 8(2), 139–161.
- Rutter, M. (1989). Annotation: Child psychiatric disorders in ICD-10. *Journal of Child Psychology and Psychiatry*, 30, 499–513.
- Rutter, M., & Schopler, E. (1993). Diagnosis by DSM-III-R versus ICD-10 criteria: Response. *Journal of Autism and Developmental Disorders*, 23(3), 573–574.
- Rutter, M., Shaffer, D., & Shepherd, M. (1975). *A multi-axial classification of child psychiatric disorders: An evaluation of a proposal*. Albany: World Health Organization.
- Smith, I. C., Reichow, B., & Volkmar, F. R. (2015). The effects of DSM-5 criteria on number of individuals diagnosed with autism spectrum disorder: A systematic review. *Journal of Autism and Developmental Disorders*, 45(8), 2541–2552.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 5–41). Hoboken: Wiley.
- Volkmar, F. R., Cicchetti, D. V., Bregman, J., & Cohen, D. J. (1992a). Three diagnostic systems for autism: DSM-III, DSM-III-R, and ICD-10. Special issue: Classification and diagnosis. *Journal of Autism and Developmental Disorders*, 22(4), 483–492.
- Volkmar, F. R., Cicchetti, D. V., Bregman, J., & Cohen, D. J. (1992b). Brief report: Developmental aspects of DSM-III-R criteria for autism. Special issue: Classification and diagnosis. *Journal of Autism and Developmental Disorders*, 22(4), 657–662.
- Volkmar, F. R., Schwab-Stone, M., & First, M. (2007). Classification. In A. Martin & F. Volkmar (Eds.), *Lewis's child and adolescent psychiatry: A comprehensive textbook* (4th ed., pp. 302–309). Philadelphia: Wolters Kluwer.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities. *Journal of Autism and Developmental Disorders*, 9(1), 11–29.
- World Health Organization. (1992). Mental and behavioral disorders, clinical descriptions and diagnostic guidelines. In *International classification of diseases* (10th ed.). Geneva: Author.

Diagnosis of Autism with Low Mental Age

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Definition

Important considerations when diagnosing autism spectrum disorder (ASD) in individuals with low mental age (low MA), defined as verbal and nonverbal functioning below a 12-month developmental level.

Historical Background

ASD can be challenging to diagnose in very young children, as many of the defining characteristics of the disorder (e.g., immature peer relationships, limitations in reciprocal conversational skills, stereotyped interests, or repetitive patterns of behavior) are age- or development-specific. That is, consistent with the timing of social communication milestones in typical development, these skills are not usually seen in infants or toddlers, nor are they apparent in older individuals with low MA (Crais et al. 2006; Ventola et al. 2006; Vig and Jedrysek 1999;

Watson et al. 2003). Comorbid intellectual impairment further complicates the diagnosis of ASD, as certain behaviors observed in children under age 5 years with global developmental delay (GDD), or in older children and adults with intellectual disability (ID), mimic the symptoms commonly associated with ASD (e.g., complex motor mannerisms, immature social skills) (Brereton et al. 2002; Gardner et al. 2018). Current estimates say that the prevalence of co-occurring ID in ASD is approximately 31% (CDC 2018). Individuals with ASD and a comorbid cognitive delay appear to be a particularly severe subgroup, as they tend to make limited developmental gains despite intervention, and they exhibit more significant adaptive, social, and behavioral deficits than peers without intellectual impairment (Gardner et al. 2018; Hinnebusch et al. 2017). Despite the challenge of identifying ASD with co-occurring cognitive and developmental delays, it is possible to diagnose ASD in low MA. Furthermore, it is particularly important to *quickly* (i.e., early in development) and *accurately* diagnose ASD in this very low-functioning population in order to facilitate access to appropriate (i.e., intensive or adapted) intervention services and generate research-informed prognoses for affected families.

Current Knowledge

Although the literature regarding ASD with low MA is somewhat limited, recent research has largely focused on three primary topics that require special consideration in this population: (a) differentiation of ASD and global delays; (b) application of commonly used diagnostic tools; and (c) developmental trajectory, including diagnostic stability.

Whereas individuals with cognitive impairment or GDD are expected to show delays across multiple developmental domains, including verbal abilities, nonverbal reasoning, motor functioning, adaptive behavior, and/or social skills, those with ASD should, by diagnostic definition, exhibit well-defined deficits in communication and socialization, in addition to the presence of

atypical restricted, repetitive behaviors (Osterling et al. 2002; Vig and Jedrysek 1999). As detailed above, however, individuals with low MA often display symptoms of both ASD and GDD or ID. When compared to children with ASD, those with GDD exhibit greater social responsiveness and joint attention (Baranek 1999; Miller et al. 2019; Osterling et al. 2002; Vig and Jedrysek 1999). Recent research on social development suggests that toddlers with GDD, but without ASD, display mild to moderate impairments in more advanced social behaviors, such as pointing (i.e., a key joint attention behavior exhibited by age 1 year in many typical toddlers) and creative and imaginative play (i.e., complex make-believe play that builds on sensorimotor exploration and imitation in the second year of life). Thus, these behaviors are unlikely to be particularly helpful in differentiating ASD from global delays, particularly in youngsters with low MA (Miller et al. 2019). There is some suggestion that delayed children, both those with ASD and GDD, engage in repetitive motor mannerisms in infancy and toddlerhood (Osterling et al. 2002), yet that finding is not universal (Miller et al. 2019). When compared to children with just cognitive delays, those with ASD demonstrate atypical language development, social withdrawal, and immature play, consistent with the defining features of ASD. Regardless of mental age, certain features appear to be indicative of ASD in early childhood, namely, a reduction in the following social behaviors: eye contact, social smiling, facial expressions directed toward others, and responsiveness to one's name (Baranek 1999; Miller et al. 2019; Osterling et al. 2002). It is useful to keep in mind that children with ASD usually show deviations from typical development, particularly in terms of their social communication, yet those with global cognitive deficits are more likely to exhibit delays (Vig and Jedrysek 1999). As such, whereas children with GDD might "catch up" to their typically developing peers, those with ASD may continue on an atypical developmental trajectory, especially if not provided with intensive ASD-specific intervention.

Autism-specific diagnostic instruments directly assess core symptoms of ASD, thereby

helping to clarify diagnosis in cases of complex symptom overlap (e.g., ASD and GDD or ID). However, few “gold-standard” tools are designed for use in individuals with low MA, which serves to further complicate the diagnostic process for low-functioning or very young children. The Autism Diagnostic Interview – Revised (ADI-R), a structured interview for the diagnosis of ASD across the life span, is recommended for use in individuals with a mental age above 2 years. Below the age of 3 years, diagnostic utility is mixed, and the measure reportedly overdiagnoses ASD in cognitively delayed and pre-verbal individuals, regardless of chronological age (Gray et al. 2008; Risi et al. 2006; Ventola et al. 2006). The Autism Diagnostic Observation Schedule (ADOS), which is currently in its second edition, uses play as a means of eliciting social communication behaviors in individuals of all ages, yet it is designed for use in those with a mental age of at least 12 months. Research on application of the ADOS to globally delayed populations is mixed. While in some studies the tool adequately discriminates ASD from other diagnoses, even in significantly delayed toddlers (Ventola et al. 2006), in other studies the ADOS is much less accurate in young and cognitively impaired individuals, particularly children functioning below a 15-month level (Gotham et al. 2007; Risi et al. 2006). In toddlers with low MA specifically, the ADOS uniformly overidentifies a large minority of those with GDD as having ASD (Miller et al. 2019). This overclassification appears to be a product of the relatively complex task demands of the ADOS, which are too challenging for low-functioning children (Gotham et al. 2007). The Childhood Autism Rating Scale (CARS), which is also in its second edition at present, offers another method of ASD diagnosis, namely, a clinician rating scale that takes into account information gleaned from a developmental history, as well as clinical observations of behavior. Although this measure is often aligned with clinical best estimate (Gotham et al. 2007; Risi et al. 2006; Ventola et al. 2006), in children with low MA, the CARS both over (i.e., overdiagnoses)- and under-classifies (i.e., misses) a small portion of significantly delayed

youngsters (Miller et al. 2019). It is thus important for clinicians to exercise some caution in applying and interpreting commonly used ASD diagnostic tools in low MA, as these measures may result in misdiagnosis when used blindly in this population.

Limited research exists regarding the developmental trajectory of individuals with ASD and low MA. That said, they do appear to be a particularly severe subgroup of ASD. Even when identified early (i.e., age 2 years), such children show less developmental growth (i.e., slower progress) in the preschool period when compared to their peers with ASD and higher MA (Hinnebusch et al. 2017). It is important to note that the social deficits observed in individuals with low MA are not merely a product of their cognitive delays. In fact, between the ages of 2 and 4 years, stability of ASD diagnoses in children with ASD and low MA is remarkably high; that is, they remain on the autism spectrum and continue to display significant symptoms, even when provided with early and intensive behavioral intervention (Hinnebusch et al. 2017; Miller et al. 2019). The high stability of symptoms in this vulnerable population provides further support for the legitimacy of diagnosing ASD in the presence of low MA.

Future Directions

With growing attention to the diagnosis of ASD with low MA, there is increased need for adequate diagnostic tools, as well as adapted interventions to best meet the needs of this severe population. When used and interpreted with caution by highly trained clinicians, “gold-standard” ASD-specific instruments such as the ADI-R, ADOS, and CARS may be suitable for the diagnosis of ASD in the context of comorbid low MA. In particular, the new Toddler Module of the ADOS, which is designed for children ages 12–30 months, may be better suited developmentally for young children with low MA. Perhaps the most promising measure for the diagnosis of ASD in young and low-functioning individuals is not yet published for clinical use: Autism Observation Scale for

Infants (AOSI; Bryson et al. 2008). The AOSI is intended for 6- to 18-month-olds and thus should suit most of the low MA population, though it is more of a developmental screener. Further research is necessary to determine the best instruments for the diagnosis of ASD with low MA.

Given the apparent severity and stability of the ASD and low MA phenotype, it is imperative that ASD interventions be tailored to support these individuals. Children who have ASD with low MA make limited developmental progress over time and show greater cognitive, adaptive, and social deficits than their peers with intact cognitive functioning even with intensive intervention (Hinnebusch et al. 2017). This pattern suggests that individuals with low MA may struggle to participate in and respond to traditional ASD-specific intervention services (Gardner et al. 2018). While failure to diagnose ASD in low MA may contribute to even poorer outcomes for this subgroup, current intervention approaches are not sufficient to meet the needs of this population. Thus, investigation of appropriate treatments is strongly indicated, drawing from Applied Behavior Analysis (ABA) and other ASD-specific interventions, in addition to knowledge from special education and neurorehabilitation.

Although there are important and unique considerations related to the diagnosis of ASD with low MA, given what is known about this population thus far, clinicians should have greater confidence in diagnosing ASD regardless of the mental age of the patient at the time of evaluation.

See Also

- Factors Affecting Outcomes
- Mental Retardation

References and Reading

- Baranek, G. T. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders*, 29(3), 213–224.
- Brereton, A. V., Tonge, B. J., MacKinnon, A. J., & Einfeld, S. L. (2002). Screening young people for autism with the Developmental Behavior Checklist. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(11), 1369–1375.
- Bryson, S. E., Zwaigenbaum, L., McDermott, C., Rombough, V., & Brian, J. (2008). The autism observation scale for infants: Scale development and reliability data. *Journal of Autism and Developmental Disorders*, 38, 731–738.
- Centers for Disease Control and Prevention. (2018, April 27). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *Morbidity and Mortality Weekly Report Surveillance Summaries*, 67(6), 1–23.
- Crais, E. R., Watson, L. R., Baranek, G. T., & Reznick, J. S. (2006). Early identification of autism: How early can we go? *Seminars in Speech and Language*, 27(3), 143–160.
- Gardner, L. M., Campbell, J. M., Bush, A. J., & Murphy, L. (2018). Comparing behavioral profiles for autism spectrum disorders and intellectual disabilities using the BASC-2 Parent Rating Scales – Preschool Form. *Journal of Psychoeducational Assessment*, 36(6), 535–551.
- Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The autism diagnostic observation schedule: Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders*, 37, 613–627.
- Gray, K. M., Tonge, B. J., & Sweeney, D. J. (2008). Using the autism diagnostic interview – Revised and the autism diagnostic observation schedule with young children with developmental delay: Evaluating diagnostic validity. *Journal of Autism and Developmental Disorders*, 38, 657–667.
- Hinnebusch, A. J., Miller, L. E., & Fein, D. A. (2017). Autism spectrum disorders and low mental age: Diagnostic stability and developmental outcomes in early childhood. *Journal of Autism and Developmental Disorders*, 47, 3967–3982.
- Miller, L. E., Burke, J. D., Robins, D. L., & Fein, D. A. (2019). Diagnosing autism spectrum disorder in children with low mental age. *Journal of Autism and Developmental Disorders*, 49(3), 1080–1095.
- Osterling, J. A., Dawson, G., & Munson, J. A. (2002). Early recognition of 1-year-old infants with autism spectrum disorder versus mental retardation. *Development and Psychopathology*, 14, 239–251.
- Risi, S., Lord, C., Gotham, K., Corsello, C., Chrysler, C., Szatmari, P., . . . Pickles, A. (2006). Combining information from multiple sources in the diagnosis of autism spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45(9), 1094–1103.
- Ventola, P. E., Kleinman, J., Pandey, J., Barton, M., Allen, S., Green, J., . . . Fein, D. (2006). Agreement among four diagnostic instruments for autism spectrum disorders in toddlers. *Journal of Autism and Developmental Disorders*, 36, 839–847.

Vig, S., & Jedrysek, E. (1999). Autistic features in young children with significant cognitive impairment: Autism or mental retardation? *Journal of Autism and Developmental Disorders*, 29(3), 235–248.

Watson, L. R., Baranek, G. T., & DiLavore, P. C. (2003). Toddlers with autism: Developmental perspectives. *Infants and Young Children*, 16(3), 201–214.

Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5)

► [Self-Report Autism Scales for Adults](#)

Diagnostic Behavioral Assessment for Autism Spectrum Disorders-Revised (DiBAS-R)

► [DiBAS-R Validation](#)

Diagnostic Boundaries of Autism

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Definition

The boundaries of autism – an area where the lines of distinction are increasingly unclear.

Historical Background

In psychiatry the publication of the DSM-III as atheoretical and phenomenological orientation (Spitzer et al. 1978) stimulated considerable

research and helped us ponder boundaries or what was “normal” and “pathological” (often related to issues of distress or impairment.). Boundary issues of normal and psychopathological were particularly complex in the area of personality problems/differences and in the area of developmental disorder like autism. Autism and related conditions, including Asperger’s disorder, provide some of the best models for illustrating the importance of the neurodiversity movement and the recognition of a broader range of the autistic phenotype.

Autism itself was first described clinically in the 1940s but only not recognized as a diagnosis until the 1980s in the landmark DSM-III (APA 1980). There were many problems with this definition (including a lack of developmental orientation), but its official recognition stimulated an explosion of research and helped us better identify and offer treatment to children with ASD with a noteworthy improvement in adult levels of independence and self-sufficiency (Magiati and Howlin 2019).

Current Knowledge

The official recognition of autism also was explicitly noted to mark it out as a condition where there was little dispute that it was so distinctive there were no boundary problems with other conditions, thus providing the best example of a “disorder” in child psychiatry (Rutter and Garmezay 1983).

Subsequent work has shown this not to be the case. Just as we have become aware of the very strong and complex genetics of autism (Yuenn et al. 2019) and differences in the way the “social brain” works in autism (McPartland et al. 2014), we also have become aware of the broader autism phenotype (Ingersoll and Wainer 2014) and the importance of a broader view of what is normal (Allen 2013). The ongoing debate on these issues (Silverman 2015; Greenberg 2013) has been helpful in allowing us to recognize a broader acceptance of the range of patterns of development and behavior.

Future Directions

The concept of neurodiversity has assumed increasing importance in helping us recognize the value and significance of neurological differences and in charting boundaries of the broad range of normal and its border with disorder. This concept has been applied to a range of neurobiological “typologies,” e.g., autism, attention deficit disorder, dyspraxia, and others. It emphasizes the potential value of different ways of cognitive processing and viewing the world. The issue of boundaries of autism and related condition with the broad range of normal development and behavior remains an important area for future work (Ingersoll et al. 2014) as are the potential genetic contributions to these phenotypes (Yuenn et al. 2019).

See Also

- [Broader Autism Phenotype](#)
- [Neurodiversity](#)

References and Reading

- Allen, F. (2013). *Saving normal: An insider's revolt against out-of-control psychiatric diagnosis, DSM-5, big pharma, and the medicalization of ordinary life*. New York: Harper Collins.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Asperger, H. (1944). Die “autistischen Psychopathen” im Kindersalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Greenberg, G. (2013). *The book of woe: The DSM and the unmaking of psychiatry*. New York: Penguin.
- Ingersoll, B., & Wainer, A. (2014). *The broader autism phenotype. Handbook of autism and pervasive developmental disorders, volume 1: Diagnosis, development, and brain mechanisms* (pp. 28–56). Hoboken: Wiley.
- Ingersoll, B., A. Wainer, F. R. Volkmar, R. Paul, S. J. Rogers & Pelphrey, K. A. (2014). *The Broader Autism Phenotype. Handbook of Autism and Pervasive Developmental Disorders, Fourth Edition*. John Wiley & Sons, Inc.
- Magiati, I., & Howlin, P. (2019). *Adult life for people with Autism Spectrum Disorders. Autism and Pervasive developmental disorders* (pp. 220–248). Cambridge, UK: F. R. Volkmar/Cambridge University Press.
- McPartland, J. C., Tillman, R. M., Yang, D. Y. J., Bernier, R. A., & Pelphrey, K. A. (2014). *The social neuroscience of autism spectrum disorder. Handbook of autism and pervasive developmental disorders, volume 1: Diagnosis, development, and brain mechanisms* (pp. 482–496). Hoboken: Wiley.
- Rutter, M., & Garmezy, N. (1983). Developmental psychopathology. In E. M. Hetherington (Ed.), *Mussen's handbook of child psychology: Vol. 4. Socialization, personality and child development* (4th ed., p. 911). New York: Wiley.
- Silverman, A. C. (2015). *NeuroTribes: The legacy of autism and the future of neurodiversity*. New York: Penguin Random House.
- Spitzer, R. L., Endicott, J. E., & Robbins, E. (1978). Research diagnostic criteria. *Archives of General Psychiatry*, 35, 773–782.
- Yuenn, R. K. C., Szatmari, P., & Vorstman, J. A. S. (2019). *The genetics of Autism Spectrum Disorders. Autism and the pervasive developmental disorders* (pp. 112–128). Cambridge, UK: F. R. Volkmar/Cambridge University Press.

Diagnostic Evaluation of Language Variation (DELV-Norm Referenced)

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Synonyms

[DELV-NR](#)

Description

The Diagnostic Evaluation of Language Variation (DELV) is a group of tests designed to assess speech and language disorders in children who speak a dialect of English other than Mainstream American English (MAE). The DELV was developed by Harry N. Seymour, at the University of Massachusetts Amherst, and Thomas W. Roeper and Jill de Villiers at the University of Massachusetts and Smith College. Their work was completed in conjunction with Harcourt

Assessment, Inc. and support from a National Institutes of Health (NIH) grant (Seymour and Pearson 2004, p. 30). The DELV consists of three separate tests: the DELV-Screening Test (DELV-ST), the DELV-Criterion Referenced Test (DELV-CR), and the DELV-Norm Referenced (DELV-NR) test. The DELV-NR is a diagnostic test that provides a standardized approach to distinguishing between speech and language differences versus delays and/or disorders in children.

The DELV-NR strives toward being a culturally unbiased assessment by differentiating between non-contrastive features of language that are shared between all English dialects and contrastive features that are not. English dialects may express a sentiment using the same rules or linguistic patterns as other dialects resulting in non-contrastive features. Conversely, they may express a sentiment differently, following rules or linguistic patterns which vary from those of other English dialects, resulting in contrastive features (Seymour et al. 2018, pp. 74–76). For example, in MAE the third person form of the linking/copula verb “be” is obligatory in the sentence “She *is* hungry.” However, in African American English (AAE), it is not obligatory to include “is.” Therefore, the linking/copula verb “be” in the present tense would be considered a contrastive feature of English because its usage varies based on the dialect. In comparison, the past tense, third person form of “be” would be required in all major dialects: “She *was* hungry.” This would be considered a non-contrastive feature of English (Seymour et al. 2018, p. 76). By excluding stimuli items that elicit dialectically specific, contrastive elements of language, the DELV-NR focuses in on the more universal, non-contrastive features of English which are used across all dialects of English. Deficits in the use of non-contrastive features of English would be indicative of a true disorder rather than a dialectal difference.

The DELV-NR assesses the language domains of syntax, pragmatics, semantics, and phonology in children regardless of their dialect of English. Each domain is addressed through multiple subtests. The syntax subdomains are as follows: *wh*-questions (10 items), passive sentence

constructions (10 items), and articles (8 items). The subtest items were selected on the basis of representing syntactic features of English that are universal among all of its dialects. The subtests of the second domain, pragmatics, address communicative role taking (4 items), short narrative (1 story, 4 questions), and question asking (9 items). These items measure the child’s overall ability to use social communication and appropriately respond to questions and situations. The semantics domain contains the most subtests: verb contrast (10 items), preposition contrast (6 items), quantifiers (2 subsections, 9 items total), and fast mapping (25 items). These items provide measures of the child’s lexical organization skills. Finally, phonology as a domain assesses consonant clusters produced in the context of a sentence (25 items) (Seymour et al. 2018, pp. 1–2, 14–48).

The DELV-NR is normed for children aged 4 years, 0 months through 9 years, 11 months. It is designed for children who speak English as their first spoken and primary language (Seymour et al. 2018, p. 1). The materials needed to administer the test consist of the examiner’s manual, stimulus book, record form, and a pencil. The assessment takes approximately 45 minutes to administer and 10–15 minutes to score. Scores in syntax, pragmatics, and semantics are reported as domain-scaled scores, test-age equivalents, and percentile ranks. The range of the domain scores is 1–19, with a mean of 10 and one standard deviation of 3. Phonology is reported in percentile band, with 16th percentile representing one standard deviation. Overall language performance is reported as a composite standard score and percentile rank. The composite standard score represents the sum of the domains scores of syntax, pragmatics, and semantics. It has a range of 40–160, with a mean of 100 and one standard deviation of 15 (Seymour et al. 2018, pp. 57–58).

Historical Background

Historically, research has acknowledged that African American and other minority children are disproportionately represented in special

education. Within speech-language pathology, minority children are often overidentified as having a speech or language disorder. The problem of using MAE as the norm against which to compare the speech and language of AAE speakers was publicly raised by Dr. Orlando Taylor at the 1968 American Speech-Language-Hearing Association (ASHA) convention. Debates around issues of diversity, multiculturalism, and dialectical differences at this seminal convention led to the formation of the ASHA Black Caucus (later the National Black Association for Speech-Language and Hearing (NBASLH)) (Moore 2009). Although a growing body of research about the assessment of speakers of non-mainstream English, including AAE, emerged in subsequent decades, no formal assessment instrument was available until the DELV-ST and DELV-CR were published in 2003. In 2005, DELV-NR became the first standardized assessment tool aimed specifically at assessing children who speak AAE and other variations of English. Based on a 2014 study, the DELV-NR resulted in diagnostic sensitivity and specificity of 0.85 and 0.93, respectively, data that supports the diagnostic validity of the DELV-NR (Pearson et al. 2014, pp. 503–505). In 2018, Ventris Learning reprinted the DELV-NR and the DELV-ST, having made minor updates but no substantial changes to either test (R. Meyer, October 11, 2019, publisher, personal communication).

Psychometric Data

The stimuli items selected for the domains of the DELV-NR were derived from an initial battery of 350 items. A group of 1370 test-takers from across the USA, including 450 speakers of MAE, 800 speakers of AAE, and 120 individuals who spoke other dialects, participated in refining the assessment material. This initial test group included children with language impairments, who together represented nearly 30% of the sample (Bland-Stewart and Pearson 2006). To create an appropriate dialect-neutral set of

questions, specific criteria were required for the final group of items which were included on the DELV-NR. Collectively, items needed to both differentiate between typically developing children and impaired children and discriminate between expected development in the age bands between the ages of 4 and 9 (Bland-Stewart and Pearson 2006). At the same time, items needed to be non-contrastive, resulting in minimal differences regardless of the dialect of English spoken by the test-takers. Researchers analyzed the children's responses to each test item until they determined a set of items which satisfied these criteria (Bland-Stewart and Pearson 2006).

A sample of over 900 participants was used in the standardization of the DELV-NR edition. Data from the 2002 US Census Current Population Survey of children aged 4–9 years was utilized to represent the demographic characteristics within the US population. The authors based their standardization sample on this data in an attempt to closely mirror the percentiles across demographic categories (Seymour et al. 2018, p. 109). The following classifications were examined within the demographic analysis of the sample: age, sex, race/ethnicity, parental education level, and region. Sex, race, and ethnicity (African American, Asian, Hispanic, other, and White), region (North Central, Northeast, South, and West), and parent years of education were categorized. Multiple analyses were completed within each age bracket to further compare sample norms to the equivalent distribution within the general US population. A comparison of race/ethnicity revealed 16.44% of the sample was African American (compared to 15.66% of the US population), 4.89% of the sample was Asian (compared to 4.68% of the US population), 14.00% of the sample was Hispanic (compared to 14.19% of the US population), 1.34% of the sample identified as other (compared to 1.23% of the US population), and lastly 63.33% of the sample was White (as compared to 64.24% of the US population) (Seymour et al. 2018, p. 111). Analysis of dialectic differences within the sample revealed that 80.8% of participants

spoke MAE. Of the remaining participants, 8.0% spoke AAE, another 7.7% of participants spoke Southern American English, and the final 3.5% spoke other dialects (Seymour et al. 2018, p. 110).

The age of sample participants ranged from 4 years (4:0) to 9 years and 11 months (9:11). There was a total of nine age intervals within the standardization sample, including age bands of 4:0–4:5, 4:6–4:11, 5:0–5:5, 5:6–5:11, 6:0–6:5, 6:6–6:11, 7:0–7:11, 8:0–8:11, and 9:0–9:11. Each subsection was represented by 100 children (Seymour et al. 2018, p. 109). Within the standardization sample, there was also a subset of children with clinical speech and language impairments. This population was represented within 6.8% of the sample by children who had been diagnosed with a receptive and expressive language disorder. Additionally, 4.2% of the sample was identified as having an articulation/phonological disorder, 2% were identified as developmentally delayed, and 1.7% was recognized as gifted. No children within the standardization sample were diagnosed with a fluency or voice disorder. Close to 9% of the sample received some type of special education service, a figure which included speech and language services. Some children demonstrated both speech and language disorders (Seymour et al. 2018, p. 110). Data from a study by the National Institute on Deafness and Other Communication Disorders was used to determine the percent of children with language disorders, which determined the prevalence of specific language impairment within the general population of kindergarteners in the USA to be 7.4%. This statistic was used to determine what percentage of the standardized sample should be represented by children with language disorders (Seymour et al. 2018, p. 110).

Reliability was examined using measures of test-retest, internal consistency, and interscorer reliability. The test-retest reliability score was based on data collected from 239 children from the original standardization sample who were given the test twice 6–28 days apart, administered

by the same individual in most cases. The stability coefficient for the individual domains of syntax, pragmatics, semantics, and phonology (for all ages combined) ranged from 0.78 to 0.91. The composite standard score test-retest reliability (for all ages combined) reached a score of 0.90 (Seymour et al. 2018, p. 121).

Internal consistency was measured using two methods and across two population samples: general and clinical. Based on the coefficient alpha technique, the internal consistency reliability coefficient for individual domains ranged from 0.75 (adequate) to 0.93 (excellent) and reached 0.88 for the composite standard score based on data from the general population (Seymour et al. 2018, pp. 122–123). Internal consistency of the individual domains and the test as a whole was even higher when the test-takers were children with a language or articulation disorder. Based on this clinical sample of 118 children, the internal consistency reliability coefficient for individual domains ranged from 0.82 to 0.93 and reached 0.93 for the composite standard score (Seymour et al. 2018, p. 124). The split-half method confirmed that internal consistency was slightly higher for the clinical sample than the general population, both at the level of individual domains and the overall composite score. The average internal consistency reliability coefficient (split-half) was 0.91 based on the general population and 0.94 based on the clinical sample (Seymour et al. 2018, pp. 123–124).

A value for the standard error of measurement (SEM) was also derived to calculate confidence intervals. The average SEM ranged from 0.91 to 1.42 scale-scored units depending on the domain. The average SEM of the composite standard score was 4.69 scale-scored units (Seymour et al. 2018, pp. 125–126). Finally, the average interscorer reliability of the DELV-NR was very high, ranging from 0.93 to 1.00 depending on the domain (Seymour et al. 2018, p. 125).

Some evidence of validity for the DELV-NR is based off information gleaned from correlation studies which looked at subtests of the *Clinical Evaluation of Language Fundamentals*, Fourth

Edition (CELF-4) and *Preschool Language Scale*, Fourth Edition (PLS-4) Articulation Screener. In one validity study, 61 children from the standardization sample of the DELV-NR were also assessed on the CELF-4. Despite some differences in testing format, low to moderate correlations were found between the CELF-4 and various domains of the DELV-NR edition, including a 0.35 correlation between the Expressive Vocabulary subtest of the CELF-4 and the semantics domain of the DELV-NR; a 0.38 correlation was found between the Understanding Spoken Paragraphs subtest of the CELF-4 and the syntax domain of the DELV-NR; and a 0.40 correlation was found between the Sentence Structure subtest of the CELF-4 and the syntax domain of the DELV-NR, as well as a 0.29–0.39 correlation between the Word Classes subtest of the CELF-4 and the semantics domain of the DELV-NR. These results appear to confirm the test developers' expectation that only a low to moderate correlation would exist between the DELV-NR and the CELF-4 because the two tests do not share the same underlying theoretical principles (Seymour et al. 2018, pp. 131–132). As expected, a higher correlation was found between the DELV-NR and the PLS-4 Articulation Screener based on a validity study of 1,104 children, 116 of whom had been diagnosed with articulation disorders. The correlation between PLS-4 screener and the phonology domain of the DELV-NR was 0.87. Together, these scores suggest that the DELV-NR edition has evidence of validity as an assessment tool for language and articulation disorders (Seymour et al. 2018, pp. 133–135).

Another measure which provides salient evidence for the validity of the DELV-NR is the diagnostic validity statistics. These statistics suggest that at one standard deviation (1 SD) below the mean, the DELV-NR has excellent sensitivity and specificity, with sensitivity equaling 0.95 and specificity equaling 0.93 (Seymour et al. 2018, pp. 140–142). One recent study has probed the assessment tool at 1.5 SD below the mean and also found the accuracy of the DELV-NR to be excellent, with a sensitivity of 0.85 and a specificity

of 0.93 when assessing a population of students with a 30% language impairment prevalence (Pearson et al. 2014, pp. 503–505; Seymour et al. 2018, p. 141). The premise of that study was that language sample (LS) measures, such as those analyzed by the Systematic Analysis of Language Transcripts (SALT) protocol, would provide a more accurate reference against which to compare the diagnostic value of the DELV-NR. Results showed that the “accuracy of the DELV-NR relative to LS profiles was greater than that of prior diagnoses, indicating that the DELV-NR was an improvement over preexisting diagnoses for this group” (Pearson et al. 2014, p. 495).

In regard to content validity, the DELV-NR edition includes stimuli items with non-contrastive features shared between speakers of MAE and AAE, a measure which is sensitive to language disorder rather than language difference. It uses this model to assess across the modalities, including semantics, syntax, phonology, and pragmatics (Seymour et al. 2018, p. 127).

Furthermore, evidence based on response processes included a thorough examination of children's incorrect responses to explore the plausibility of other possible answers for each question. This process included an examination of the reasoning behind a child's response and resulted in some changes for assessment questions as deemed appropriate and necessary (Seymour et al. 2018, p. 128).

Additionally, the validity of internal structure was assessed to understand how intercorrelation between the domains may impact testing on the DELV-NR edition. Low to moderate correlations were found which may reflect interconnectivity of language domains in language tasks (Seymour et al. 2018, pp. 128–129).

Clinical Uses

Research supports the usefulness of the DELV-NR as an assessment tool for diagnosing a language disorder. The DELV-NR analyzes scores on multiple domains of language and is shown to have excellent sensitivity (0.95) and specificity

(0.93) to diagnose a language disorder at one standard deviation below the mean as the cutoff score (Seymour et al. 2018, pp. 140–142). Based on the composite standard score, the test was shown to have excellent test-retest reliability, good to excellent internal consistency, and very high interscorer reliability (Seymour et al. 2018).

The usefulness of the DELV-NR as an assessment of articulation is more limited. The DELV-NR appears to have only “good” sensitivity (0.85) and “adequate” specificity (0.75) to diagnose a phonological/articulation disorder at one standard deviation below the mean as the cutoff score. The overall sensitivity is fair compared to other well-known and frequently used assessments (Seymour et al. 2018, pp. 140–142). As stated in the Examiner’s Manual, “The Phonology Domain by itself is not designed to provide in-depth information about the nature of a child’s phonological/articulation disorder. If a child’s percentile band score is below or includes the 16th percentile, you should evaluate the child further to determine the exact nature and scope of the child’s problem” (Seymour et al. 2018, p. 65). Differentiating between a phonological and an articulation disorder would also require the use of additional diagnostic tools.

The DELV-NR is based on robust standardization research, which included 900 children between the ages of 4;0 and 9;11 and was designed to reflect population demographics as reported in 2002 US Census Current Population Survey (Seymour et al. 2018, p. 109). Because the DELV-NR is normed on the general US population, its diagnostic value extends to both speakers of MAE and speakers of nonmainstream English, including AAE (Pearson et al. 2014). However, its greatest strength is in filling the gap in existing standardized language assessments by providing a dialect-neutral testing battery for culturally and dialectically diverse populations.

Children’s dialectal differences may be overidentified as language disorders, requiring students to receive speech and language services, while actual disorders may be under identified. The DELV-NR responds to the need for a systematic means of distinguishing between a language

difference and a disorder. A recent, independent study has concluded that the DELV-NR provides better diagnostic accuracy than prior tests for this population, when using the Systematic Analysis of Language Transcripts (SALT) protocol results as the benchmark. Specifically, the sensitivity and specificity values of the DELV-NR were found to be 0.85 and 0.93, leading the authors to conclude that “even after making allowances for the imperfection of available gold standards, clinical decisions made with the DELV-NR achieved high values on conventional measures of diagnostic accuracy” (Pearson et al. 2014, p. 495).

See Also

- CELF-5
- Clinical Evaluation of Language Fundamentals: Preschool – Second Edition (CELF Preschool-2)
- Culture and Autism
- Participation of Black and African-American Families in Autism Research
- Preschool Language Scale – 5

References and Reading

- Bland-Stewart, L., & Pearson, B. Z. (2006). Difference vs. deficit: Delving into a solution with the new norm-referenced diagnostic evaluation of language variation (DELV-NR). *Perspectives on Communication Disorders and Sciences in Culturally and Linguistically Diverse Populations*, 13(1), 18–24. <https://doi.org/10.1044/cds13.1.18>.
- Moore, M. (2009). 1968: Orlando Taylor looks back. *The ASHA Leader*, 14(4), 20–21. <https://doi.org/10.1044/leader.AN3.14042009.21>.
- Pearson, B. Z., Jackson, J. E., & Wu, H. (2014). Seeking a valid gold standard for an innovative, dialect-neutral language test. *Journal of Speech, Language, and Hearing Research: JSLHR*, 57(2), 495–508. https://doi.org/10.1044/2013_JSLHR-L-12-0126.
- Seymour, H. N., & Pearson, B. Z. (2004). Steps in designing and implementing an innovative assessment instrument. *Seminars in Speech and Language*, 25(01), 27–31. <https://doi.org/10.1055/s-2004-824823>.
- Seymour, H. N., Roeper, T. W., & de Villiers, J. (2018). *Diagnostic evaluation of language variation – Norm referenced*. Sun Prairie: Ventris Learning.

Diagnostic Instruments in Autistic Spectrum Disorders

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Definition

Diagnostic measures are designed to capture behaviors in the areas of communication, reciprocal social interactions, and restricted and repetitive behaviors that characterize an autism spectrum disorder. These measures attempt to quantify behaviors associated with an autism spectrum disorder by assigning them numerical scores. These quantitative behavior scores are then translated into summary scores that result in a classification that is typically either consistent with one of the autism spectrum disorders or not. Current diagnostic instruments include parent questionnaires and interviews as well as standardized observational measures. The time and training required to administer and score these instruments varies from minimal for parent questionnaires to more involved for observational measures and semistructured interviews.

Historical Background

One of the first widely used scales for identifying children with autism was *The Rimland Diagnostic Form for Behavior Disturbed Children* (Form E-1; Rimland 1968). This measure was an important development in the field, as it focused on identifying carefully selected symptoms of autism. Another early diagnostic measure developed at around the same time was the *Behavior Rating Scale for Autistic and Atypical Children* (BRIAC; Rutter et al. 1966). This measure is historically significant because it was the first to be based on actual observations of behavior from clinician case notes rather than parent report.

The *Handicaps, Behaviors and Skills Schedule* (HBS; Wing and Gould 1978) was also an early influential measure because it was the first widely used semistructured parent interview and it contributed to the understanding of the “triad of impairments” that has led to our current understanding of autism. Not technically considered a diagnostic measure, the HBS was a framework for gathering information regarding symptoms and behavior that could be utilized in a clinical evaluation. This measure has been revised and is currently known as the *Diagnostic Interview for Social and Communication Disorders* (DISCO; Wing et al. 2002 – see below).

Another scale of importance in the initial group of diagnostic measures was the *Behavior Autism Rating Scale* (BOS; Freeman et al. 1978). This measure was the first instrument that emphasized controlling the environment in which the child was observed, as well as standardizing the behaviors that were observed (Lord and Corsello 2005).

Most of these measures have been revised, with Rimland’s Form E-1 becoming Form E-2, the HBS now the DISCO, and the BOS leading to the development of the Real Life Rating Scale (RLRS; Freeman et al. 1986).

Diagnostic measures have evolved over time, both because diagnostic criteria have changed with each revision of the Diagnostic and Statistical Manual, Fourth Edition (DSM-IV), and because empirical studies continue to provide information about how well each measure differentiates children with autism from those without autism based on current definitions. The diagnostic measures have changed as we have learned more about the disorder, including the expansion of age range and developmental levels of children included within this diagnostic group.

As the current gold standard in autism diagnostic measures, the *Autism Diagnostic Observation Schedule* (ADOS; Lord et al. 2000) illustrates the change from DSM III to DSM IV criteria to now include Asperger’s syndrome and pervasive developmental disorder, not otherwise specified on the spectrum. This measure evolved from its original form developed in the 1980s to its current form which includes five modules for assessing toddlers through adults with module choice

dependent on the individual's age and language level. The *Childhood Autism Rating Scale* (CARS; Schloper et al. 2010) is another example of this evolution, with the addition of a scale for high-functioning autism, as the original version missed many children with better language and cognitive skills.

Current Knowledge

There are currently several measures available for diagnostic purposes, ranging from those that are very quick to administer and require minimal training to those that take more time and training. As with any other psychometric measure, diagnostic measures are evaluated based on reliability and validity data. This section will cover the most widely used and available diagnostic protocols. For more detailed information on these measures, please see the respective individual entries included within this encyclopedia (Table 1).

Parent Questionnaires

The Autism Behavior Checklist (ABC): This questionnaire is one component of the *Autism Screening Instrument for Education Planning* (ASIEP; Krug et al. 1980), now in its third revision. It builds on several other measures, including Rimland's Form E-2, the BOS, and the BRIAAC. It contains five items across five areas, and ranges are provided to distinguish a high probability of autism, a low probability of autism, or mixed probability. Standard scores are available for children between the ages of 3 and 13 years. It was initially intended to be completed by teachers or other professionals working with a child. This measure requires no special training. It has also been used with parents on a retrospective basis for children with high-functioning autism. One concern regarding this measure, however, is its low sensitivity, as many children on the spectrum appear to be missed using the suggested cutoff score.

The Australian Scale for Asperger's Syndrome (ASAS): This questionnaire includes 19 items covering five areas and is scored on a seven point Likert-type scale. It is designed to be completed

by a teacher or parent and covers ages 3–19 years. The authors recommend that the measure be used as a screener rather than a diagnostic measure because of low specificity. There are little published data available on this measure, and the original study had several methodological issues, including raters who were not blind to diagnosis. Though the measure does not result in a classification of Asperger's disorder, as a screener, it provides information on whether a child should receive a diagnostic evaluation.

Behavior Summarized Evaluation – Revised (BSE-R): This rating form is comprised of items from two overlapping instruments, the *Behavioral Summarized Evaluation Scale* (BSE) and the *Infant Behavioral Summarized Evaluation Scale* (IBSE; Barthelemy et al. 1997). It is primarily designed to document behavioral symptoms associated with autism as they relate to neurophysiological measures. These scales consist of 20 items scored on a five-point Likert scale by trained raters on the basis of direct or videotaped observation, discussion of the child's history, and access to information from multiple sources. It covers the ages of 18 months through 12 years and takes approximately five minutes to administer. Interrater reliability and convergent validity is reported to be strong.

The Childhood Autism Rating Scale – Second Edition (CARS-2): This rating form has been one of the strongest, best documented, and most widely used rating scales for behaviors associated with autism. It consists of 15 items on which children and adults are rated, generally after observation, on a four-point Likert scale and results in classifications of not autistic or mild to severe autism. This measure, most commonly completed by a clinician based on observation, requires minimal training and approximately 15 min to complete. The revision of this measure includes a form to better capture children with high-functioning autism and is recommended for use with individuals whose IQs are above 70 and who are over the age of 6 years. The original CARS form has not changed and is included in the CARS-2 for use with children under the age of 6 years or who have lower IQ scores. The CARS-2 was recently adapted, and there are not

Diagnostic Instruments in Autistic Spectrum Disorders, Table 1 Summary of measures

Measure	Format	Administration time	Age range	Diagnostic criteria used	Training	Suggested use
SRS	Parent/teacher/self-report questionnaire	15 min	Preschool to adulthood	DSM-IV	None	Screening/ response to treatment
SCQ	Parent/caregiver questionnaire	15 min	Preschool to adulthood	DSM-IV	None	Screening
GARS-2	Parent/caregiver questionnaire	15 min	Preschool to adulthood	DSM-IV	None	Screening
CARS-2	Clinician rating based on observation	30 min	Preschool to adulthood	DSM-III-R	Minimal	Diagnostic
BSE-R	Rating based on observation, review of records, and interview	5 min	1½ to 12 years	N/A	Yes	Symptoms for research
ABC	Teacher questionnaire	Not specified	3–13 years	DSM-IV	Minimal	Measure maladaptive behavior
PDDRS	Parent questionnaire	Not specified	Not specified	DSM-III-R	None	Screening
GADS	Parent questionnaire	10 min	3–22 years	DSM-IV	Yes	Assess Asperger's disorder behavior
ASAS	Parent or teacher questionnaire	10 min	3–19 years	Not specified	None	Screening
ADI-R	Semistructured interview	1.5–3 h	Toddler to adulthood	DSM-IV	Yes	Research and clinical diagnosis
DISCO	Semistructured interview	2–3 h	Any age	ICD-10	Yes	Assess individual needs, treatment goals
ASDI	Semistructured interview	Not specified	Not specified	Gillberg's criteria	None	Screening
AOSI	Semistructured observation	20 min	6–18 months	DSM-IV	Yes	Early identification
ADOS	Observation	30–60 min	Toddler to adulthood	DSM-IV	Yes	Research and clinical diagnosis
PEP-III	Caregiver report and clinical observation	45–90 min	1–7 years	DSM-IV	Minimal	Assess development, create treatment goals

yet many research studies evaluating the effectiveness of the rating scale for children with high-functioning autism.

The Gilliam Asperger's Disorder Scale (GADS): This parent questionnaire consists of 32 items and is based on DSM-IV and ICD-10 criteria of Asperger's disorder. It takes

approximately 5–10 min to score and also includes a parent interview section that is not scored but provides information on language and cognitive and adaptive behavior which is important in differentiating Asperger's disorder from other autism spectrum diagnoses. Like the Gilliam Autism Rating Scale (GARS), this measure

results in an Asperger's quotient of low or high probability of an Asperger's disorder. As with the GARS, the standardization sample diagnosis was reported by parent or professional and not confirmed.

The Gilliam Autism Rating Scale – Second Edition (GARS-2): This questionnaire has recently been revised and is now known as the GARS-2. It consists of 56 items across four subscales, covers the ages between 3 and 22 years, and takes approximately 5–10 min to administer. The measure is based on DSM-IV and Autism Society of America criteria and results in an autism quotient that indicates whether a child has a “low probability” or a “high probability” of having autism. No training is required. The measure is intended for screening; however, several studies using the original version of the GARS found that it missed up to 52% of the children who met diagnostic criteria for autism clinically and received scores within the autism range on other standardized diagnostic measures (South et al. 2002). The initial normative sample was large, but the diagnoses were reported by the parent and not confirmed. Revisions to the GARS-2 have attempted to address these concerns by lowering the cutoff score and providing a new normative sample. As with the initial normative sample, not much information is available on the group. While the measure is considered to be appropriate for use with adults, scores should be interpreted cautiously because only 9% of normative sample was over 16 years (Montgomery et al. 2008).

The Pervasive Developmental Disorders Rating Scale (PDDRS): This measure is a revision of an earlier scale (Eaves 2003). It includes 51 items across three subscales and is based on the DSM-III-R. Each behavior is based on a five-point Likert scale. A child is considered to fall within the range of an autism spectrum disorder if both the total score and arousal score fall one standard deviation below the mean. No standard diagnostic procedure was used to define the sample, and therefore the suggested use of the PDDRS is for screening only.

Social Communication Questionnaire (SCQ): This questionnaire, formerly known as the *Autism*

Screening Questionnaire, is based on a well-validated standardized parent interview, the *Autism Diagnostic Interview – Revised (ADI-R)*. It was initially designed as a screening measure and consists of 40 items that cover the areas of communication, reciprocal social interactions, and restricted and repetitive behaviors and interests. It is designed to be filled out by a parent and takes approximately 15 min to complete. No training is required, and scoring instructions are available in the manual.

There are two versions of the measure, a “current” version that is designed for children under the age of 5 years and covers current behavior, and a “lifetime” version that is designed for children over 5 years of age to adulthood and covers early behavior, focusing on the ages between 4 and 5 years. For children under the age of 5 years, several studies have found that a lower cut off score of greater than or equal to 12 results in the greatest diagnostic differentiation. For those older than 5 years of age, scores of greater than or equal to 15 are considered to be significant and suggestive of a possible autism spectrum disorder.

Little information is available on its use with children under the age of 3 years. It works fairly well as a screener for children over the age of 3 years, with the modified cutoff for children under the age of 5 years. It has higher specificity than many screening measures, and its performance has been found to be similar to a standardized diagnostic interview in at least one study (Corsello et al. 2007).

Social Responsiveness Scale (SRS): This questionnaire was initially developed to measure social and communication difficulties along a continuum. It consists of 65 items covering the areas of communication, reciprocal social interactions, and restricted and repetitive behaviors and interests. Gender norms are available, and the measure results in a T-score and a social severity impairment score ranging from typical to severe. Both a teacher version and a caregiver version are currently available, and each takes approximately 15–20 min to complete. The SRS does not require training to administer, and instructions for scoring are included in the manual. The first version of the SRS was designed for children between 4 and

18 years of age. More recently, two additional versions have been developed and are available for research use and are soon to be available for use clinically: an adult version in a self-report and other report form, and a preschool version. Suggested uses include screening and response to treatment.

Semistructured Interviews

The Autism Diagnostic Interview – Revised (ADI-R): The ADI-R is one of the most widely validated diagnostic measures available. Based on DSM-IV criteria, it is administered as a semistructured interview by a clinician to a parent or caregiver, and covers current behavior for all children and historical information for older children and adults. The measure consists of 89 items that are coded on a 0–3 point scale, several of which are transferred to a diagnostic algorithm that results in a diagnostic classification. One of the biggest weaknesses of this measure is administration time, which is between 1.5 and 3 h. The extensive reliability and validity data available for the ADI and the clinically rich information it provides make this measure the gold standard in research despite its lengthy administration time.

The Asperger's Syndrome (and High-Functioning Autism) Diagnostic Interview (ASDI): This measure was designed as a diagnostic tool for verbally fluent individuals with autism and Asperger's disorder. It is a semistructured interview based on Gillberg's criteria and includes 20 items that operationalize six criteria. The interviewer is instructed to obtain descriptions of actual behaviors to accurately code each item. This interview does well in distinguishing Asperger's disorder from psychiatric disorders and normality, but has yet to develop a means of distinguishing Asperger's from autism (Gillberg et al. 2001).

The Diagnostic Interview for Social and Communication Behaviors (DISCO): This measure is a standardized semistructured interview based on the HBS, and it is now in its ninth revision. It was designed to obtain behaviors relevant to the diagnosis of autism for the purpose of assessing individual needs and development across several areas. The DISCO includes items that cover the areas associated with autism spectrum disorders,

as well as developmental items and atypical behaviors. This measure was not originally designed for diagnostic purposes, but rather to assist clinicians in generating recommendations for older individuals with an autism spectrum disorder. Diagnostic algorithms have been developed for research purposes.

Observational Measures

The Autism Diagnostic Observation Schedule (ADOS): The ADOS is one of the most widely studied and used diagnostic instruments, and, along with the ADI-R, is considered the gold standard in research studies. It is a semistructured observational measure that consists of several tasks that are administered to a child or adult for diagnostic purposes. The measure includes a number of coded behaviors that allow for assessment in the areas of communication, reciprocal social interactions, and restricted and repetitive behaviors and interests. Scores are transferred to an algorithm and result in a classification of autism, autism spectrum disorder, or non-spectrum.

The ADOS is based on DSM-IV criteria and takes approximately 30–60 min to administer. It is organized into five modules covering children of toddler age who use little or no phrase speech to older children and adults with fluent language. A toddler module has recently been added. Now there are also revised algorithms designed to improve specificity without sacrificing sensitivity, as well as newly developed severity scores for the purpose of measuring change over time. The ADOS requires training and experience with autism spectrum disorders.

The Autism Observation Scale of Infancy (AOSI): The AOSI is a semistructured, standardized observational measure designed for infants between 6 and 18 months of age. It consists of 18 items covering specific behaviors that have been considered to be early indicators of a later autism spectrum disorder diagnosis based on empirical studies and clinical experience. Training on administration and scoring is required. Interrater reliability on a small sample of infants was good to excellent with the exception of a subset of items at the 6 month assessment. Interrater reliability scores on a small sample

were fair to good. Reliability was calculated using kappas, and a skewed distribution may affect results. Scores at the ages of 12 and 18 months were considered to be predictive of a later diagnosis of autism. Available data has been on high-risk infant groups, primarily high-risk siblings, and on relatively small samples. The intended use of this measure is to identify children who may be likely to later meet criteria for a diagnosis of autism in a high-risk sample, and it is available for use in research protocols (Bryson et al. 2008).

The Psychoeducational Profile Revised (PEP-3): This measure was designed to assess development and diagnostic characteristics of children with an autism spectrum disorder. This measure was designed for children between 12 months and 7 years of age. The PEP-3 consists of a pathology section that is designed to measure the severity of behaviors associated with autism spectrum disorders. There is little information available on the reliability or validity of the pathology section. The PEP-R was primarily designed to assess development and create treatment goals. This measure requires approximately 45–90 min to administer and requires experience with children with autism spectrum disorders. The suggested uses are primarily to create treatment goals and to assess development.

Future Directions

Diagnostic measures continue to be modified and refined as more is learned about their effectiveness, as the diagnostic criteria for autism spectrum disorders changes with new revisions of the diagnostic manual, and as health-care funding changes. There continues to be a need for measures that can be used for research and clinical purposes. Standardized observational measures are generally brief enough to be used as part of a clinical evaluation or research protocol. Several studies suggest that using both a standardized parent interview in conjunction with a standardized diagnostic observational measure and clinical judgment results in the most accurate diagnosis. However, the most well-validated diagnostic interview, the ADI-R, is long and takes time to

administer. As funding changes, there is an increasing need for more efficient diagnostic measures, and consequently, attempts have been made to use more questionnaires and to decrease the length of time required to conduct standardized interviews. Screening measures and questionnaires are also being refined to make them more suitable for diagnostic use in research protocols.

As a field, we have begun to recognize younger children as at risk for autism spectrum disorders, requiring measures that can identify toddlers that may later develop the disorder. Interviews and questionnaires are also being extended, downward to identify the youngest children and upward to better capture older and higher functioning children with autism spectrum disorders. As treatment attempts to address the core deficits of autism spectrum disorders, there have also been requests to develop measures that can capture response to treatment. This has, in part, led to the development of severity scores for the ADOS.

The fifth revision of the DSM is expected within the next few years and will lead to modifications in current measures to match the new diagnostic criteria. Currently there are several very strong diagnostic measures available for use for screening or diagnosis of autism spectrum disorders. The diagnostic measures in the field of autism are dynamic and evolving, with their widespread use in both clinical and research settings leading to modifications for practical application and improved effectiveness.

References and Reading

- Barthelemy, C., Roux, S., Adrien, J. L., Hameury, L., Guerin, P., Garreau, B., et al. (1997). Validation of the revised behavior summarized evaluation scale. *Journal of Autism and Developmental Disorders*, 27(2), 139–153.
- Bryson, S. E., Zwaigenbaum, L., McDermott, C., Rombough, V., & Brian, J. (2008). The autism observation scale for infants: Scale development and reliability data. *Journal of Autism and Developmental Disorders*, 38, 731–738.
- Corsello, C., Hus, V., Pickles, A., Risi, S., Cook, E. H., Jr., Leventhal, B. L., et al. (2007). Between a ROC and a hard place: Decision making and making decisions about using the SCQ. *Journal of Child Psychology and Psychiatry*, 48(9), 932–940.

- Eaves, R. C. (2003). *The pervasive developmental disorders rating scale*. Opelika: Small World.
- Freeman, B. J., Ritvo, E. R., Guthrie, D., Schroth, P., & Ball, J. (1978). The behavior observation scale for autism: Initial methodology, data analysis, and preliminary finding on 89 children. *Journal of the American Academy of Child Psychiatry*, 17, 576–588.
- Freeman, B. J., Ritvo, E. R., Yokota, A., & Ritvo, A. (1986). A scale for rating symptoms of patients with the syndrome of autism in real life settings. *Journal of the American Academy of Child Psychiatry*, 25, 130–136.
- Gillberg, C., Gillberg, C., Rastam, M., & Wentz, E. (2001). The Asperger syndrome (and high-functioning autism) diagnostic interview (ASDI): A preliminary study of a new structure clinical interview. *Autism*, 5(1), 57–66.
- Krug, D. A., Joel, A., & Almond, P. (1980). Behavior checklist for identifying severely handicapped individuals with high levels of autistic behavior. *Journal of Child Psychology and Psychiatry*, 21(3), 221–229.
- Lord, C., & Corsello, C. (2005). Diagnostic instruments in autism spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 730–771). New York: Wiley.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., DiLavore, P. C., et al. (2000). The autism diagnostic observation schedule-generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30(3), 205–223.
- Montgomery, J. M., Newton, B., & Smith, C. (2008). Review of GARS-2: Gilliam autism rating scale-second edition. *Journal of Psychoeducational Assessment*, 26(4), 395–401.
- Rimland, B. (1968). On the objective diagnosis of infantile autism. *Acta Paedopsychiatrica: International Journal of Child and Adolescent Psychiatry*, 35(4/8), 146–161.
- Ruttenberg, B. A., Dratman, M. L., Fraknoi, J., & Wenar, C. (1966). An instrument for evaluating autistic children. *Journal of the American Academy of Child Psychiatry*, 5, 453–478.
- Schloper, E., Van Bourgondien, M. E., Wellman, G. J., & Love, S. R. (2010). *CARS-2: Childhood autism rating scale-second edition*. Torrance: Western Psychological Services.
- South, M., Williams, B. J., McMahon, W. M., Owley, T., Filipek, P. A., Shernoff, E., et al. (2002). Utility of the Gilliam Autism Rating Scale in research and clinical populations. *Journal of Autism and Developmental Disorders*, 32(6), 593–599.
- Wing, L., & Gould, J. (1978). Systematic recording of behaviours and skills of retarded and psychotic children. *Journal of Autism and Childhood Schizophrenia*, 8, 79–97.
- Wing, L., Leekam, L., Libby, S., Gould, J., & Larcombe, M. (2002). The diagnostic interview for social and communication disorders: Background, inter-rater reliability and clinical use. *Journal of Child Psychology and Psychiatry*, 43, 307–325.

Diagnostic Interview for Social and Communication Disorders

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Abbreviations

ADI-R Autism Diagnostic Interview-Revised
PDD Pervasive developmental disorders

Synonyms

DISCO

Description

The Diagnostic Interview for Social and Communication Disorders (DISCO) (Leekam et al. 2002; Wing et al. 2002) is a semi-structured interview schedule used with the parent or carer of an individual to elicit a broad picture of the individual's behaviors and needs. Its primary purpose is to elicit information relevant to the autistic spectrum in order to assist clinicians in their judgment of an individual's level of development, disabilities, and specific needs. It contains sets of algorithms for diagnosis of autism according to the international classification criteria (ICD and DSM) and other sets of diagnostic criteria. Key features of the DISCO are (a) that it can be used for children and adults of any age, (b) that it collects extensive information not only on the core symptoms of autism but also beyond these symptoms (e.g., sensory symptoms, emotion symptoms, gross and fine motor skills, psychiatric and forensic problems, maladaptive behavior, sleep difficulties), and (c) that it has a strong developmental focus, including the detailed assessment of current developmental level and developmental delay.

The Diagnostic Interview for Social and Communication Disorders (DISCO) is based on a concept of a spectrum of autistic disorders that predated the earliest ICD and DSM criteria for

autism and pervasive developmental disorders (Wing 1988, 1996; Wing and Gould 1979). This concept is similar to, but wider than, the concept of pervasive developmental disorders (PDD) described in DSM-IV and ICD-10. Therefore, the use of the DISCO enables diagnosis of specific autism conditions according to DSM-IV, DSM-5, and ICD-10, but the DISCO goes beyond this. Information collected is placed in a broad developmental and behavioral context that reflects a dimensional view of a spectrum of autistic disorder and emphasizes its broad-ranging nature. An individual's difficulties with reciprocal social interaction, communication, and repetitive behavior can therefore be understood against the pattern of their developmental skills and associated abilities and difficulties. In addition, symptoms of other related disorders (e.g., language, attention, or motor impairments) can be elicited for further investigation. Furthermore, as the DISCO is concerned with the assessment of needs as well as with the diagnosis of ASD, the information it collects is relevant for guiding recommendations relating to management and interventions.

The DISCO interview schedule comprises more than 300 questions that are organized into eight parts. Part 1 provides a factual record of family, medical, and identifying information. Part 2 deals with the first 2 years of life. This infancy section consists of medical questions relevant to the diagnostic criteria for Rett's syndrome and a further set of questions relating to behaviors. Part 3 "Developmental Skills" forms the largest part of the DISCO. This section comprises subsections related to the following domains: (a) gross motor skills, (b) self-care, (c) domestic skills, (d) independence, (e) verbal and nonverbal communication, (f) social interaction with adults and peers, (g) social play and leisure, (h) imagination, (i) pictures, reading, and writing, (j) visuo-manual skills, and (k) cognitive skills. All the items are rated by the interviewer in terms of three aspects: (a) current level, (b) delay in acquiring relevant skills, and (c) atypical (or unusual) behavior associated with the relevant skills. The atypical behaviors cover both the past and present behaviors.

Other parts of the DISCO also record both the past and present behavior patterns of the individual. Part 4 on repetitive activities includes subsections on stereotypies, atypical sensory responses, and repetitive routines. Part 5 on emotions includes questions on anxiety and mood changes. Part 6 on maladaptive behavior is concerned with behavior that impinges adversely on other people such as aggression and temper tantrums and disturbances of sleep. Part 8 on psychiatric conditions and forensic problems includes considerations of a range of psychiatric conditions relevant for adolescents and adults that may need further investigations such as symptoms of schizophrenia, personality disorders, and eating disorders, and this part also includes specific subsections on catatonic features and sexual problems.

Finally, there is a separate section (Part 7) to help guide clinicians to arrive at a clinical judgment independent of quantitative results. This part includes the interviewer's judgment on the quality of social interaction, social communication, social imagination, and overall pattern of activities. Whereas elsewhere during the interview, the aim is for the interviewer to establish the facts related to specific skills or behavior, in Part 7, the ratings are made on an overview of all the available information. This part of the schedule usually does not involve direct questioning of the informant and elicits judgments by the interviewer.

As mentioned above, the DISCO enables diagnosis of specific autism conditions according to DSM-IV category "pervasive developmental disorders" (American Psychiatric Association [APA] 1994), DSM-5 category "autism spectrum disorder" (American Psychiatric Association [APA] 2013), and the ICD-10 category "pervasive developmental disorders" (World Health Organization [WHO] 1993). Selected items throughout the interview provide the diagnostic criteria not only for these diagnostic systems but also for other diagnostic systems. Other diagnostic systems include (a) Kanner's early infantile autism (Kanner and Eisenberg 1956), (b) Asperger's syndrome based on Gillberg and Gillberg (1989) (Ehlers and Gillberg 1993; Wing 1981), (c) autistic spectrum disorder (Wing and Gould

1979), and (d) Wing and Gould's definition of social impairment.

Completion of the entire interview takes approximately 2–3 h, and this provides a comprehensive picture of the individual's skills and abilities. This is particularly useful for complex cases. However, it is possible to adapt the DISCO for specific purposes. For example, where the clinician needs only to obtain information on the current clinical picture, questions about delays in development and past behavior can be omitted. It is also possible to complete the interview using only items relevant for the diagnostic algorithms. DSM-5 item sets for a new DISCO-Abbreviated interview have been published (Carrington et al. 2014, 2019).

The DISCO is distinctively different from other interview schedules, such as the Autism Diagnostic Interview-Revised (ADI-R) (Lord et al. 1994), that were designed to be closely related to the ICD-10 research criteria for childhood autism (WHO 1993) and for DSM-IV autistic disorder (APA 1994). The DISCO is more detailed in the information it collects and is broader and more developmental in focus. For example, the interviewer collects information on a very large number of separate items each covering specific examples of types of behavior, from the most common to the rare, in order to facilitate the final clinical judgments. The interviewer also records the individual's current developmental level and their developmental delays for all domains of functioning. Finally, the interviewer can then apply a number of algorithms for different diagnostic systems using the DISCO.

A number of research studies have been published (see also section “[Psychometric Data](#)” below). Research using the DISCO includes examination of its algorithms for, ICD-10 childhood autism (Leekam et al. 2002; Nygren et al. 2009; Maljaars et al. 2012) and for ICD-10 Asperger's syndrome (Leekam et al. 2000). Algorithms have also been published for DSM-5 (Kent et al. 2013), for DSM-5 Abbreviated (Carrington et al. 2014), and for Wing and Gould's autistic spectrum disorder (Leekam et al. 2002). Research using the other algorithms – DSM-III-R pervasive

developmental disorders (American Psychiatric Association 1987), Kanner's and Eisenberg's criteria (Kanner and Eisenberg 1956), and Wing and Gould's definition of social impairment – has not yet been published.

Research using the DISCO has also led to new knowledge about the role of developmental, behavioral, genetic, and neuropsychiatric factors in the autism profile. Research studies have documented ASD symptoms and behavioral profiles in Rett's syndrome (Wulffaert et al. 2009a), Cornelia de Lange syndrome (Wulffaert et al. 2009), mild intellectual disability (Soenen et al. 2009), gender dysphoria (de Vries et al. 2010), fetal alcohol syndrome (Mukherjee et al. 2011), and the link between epilepsy and autism symptoms (Danielsson et al. 2005; Turk et al. 2008). The DISCO has also been used in several epidemiological studies (Ellefsen et al. 2007; Brugha et al. 2011). Research using the DISCO has also given insights into the nature of catatonia in the autism spectrum (Wing and Shah 2000), the nature of pathological demand avoidance within the autism spectrum (O'Nions et al. 2016), adult outcomes of autism (Billstedt et al. 2007; Cederlund et al. 2008), and pattern and frequency of sensory symptoms in those with ASD (Leekam et al. 2007a).

In addition, to research using the DISCO as an interview, items within the DISCO have been extracted to form research questionnaires and checklists for research purposes. Early checklist studies used items later to be included in the DISCO and these examined empirical clustering of symptoms and cognitive abilities (Prior et al. 1998) and the relation between language delay and diagnosis (Eisenmajer et al. 1998). More recently new questionnaires have been created from DISCO interview items. The Repetitive Behaviour Questionnaire-2 for children (Leekam et al. 2007) and Repetitive Behaviour Questionnaire-2A for adults (Barrett et al. 2015) have been used to examine the relationship between repetitive behavior and other factors including imagination, language, sensory symptoms, and anxiety (Honey et al. 2007; Barrett 2018; Lidstone et al. 2014) in both children and

adults with autism. The RBQ2-A has also been used in longitudinal studies of typically developing children to examine different subtypes of repetitive behaviors predict specific developmental outcomes as children get older (Larkin et al. 2017; Uljarević et al. 2017).

Training for the DISCO has been developed by Dr. Judith Gould and Dr. Lorna Wing and consists of a 4-day training course in two stages. Training covers the Lorna Wing Centre's method of diagnosis, the complexities of diagnosis and assessment of needs, and the specialist psychological assessment. Stage 1 is for 2 days preceded by pre-course work and followed by evaluated interim coursework. Stage 2 is for 2 days leading to accreditation. A computer program is available for accredited users. Training is available for clinicians involved in diagnosis and assessment of needs and for professionals who use DISCO for research. Information about training is available from the following email address: lornawing-centre@nas.org.uk.

Historical Background

The origins of the DISCO are to be found in a study comparing children with autism with children with other disabilities (Down's syndrome, developmental receptive language disorders, developmental expressive language disorders, partial sight, and partial hearing) and a group of children with typical development (Wing 1969). The "Childhood Behavior Schedule," a questionnaire sent to parents by post, was designed for this study. It elicited information concerning the social, language, imagination, and motor impairments and the odd responses to sensory input and stereotyped behavior found in autism. These behaviors are now covered in much more detail in the DISCO.

The original questionnaire schedule was reorganized and expanded to include items on developmental skills and was named the "Handicaps, Behavior, and Skills (HBS)" schedule. A variety of sources were used when constructing the developmental items, including Cooper et al.

(1978), Egan et al. (1969), Griffiths (1967) and Sheridan (1973, 1977). The HBS was used for research in an epidemiological study of autism spectrum disorders in children in the former London Borough of Camberwell (Wing and Gould 1979) and in a follow-up of the children into adult life (Wing 1988). The original epidemiological study was designed to identify children with any of the features of autism in order to see if any clinical patterns could be discovered. This distinguished it from previous studies (e.g., Lotter 1966, 1967) which looked specifically for children showing the narrow criteria originally suggested by Kanner and Eisenberg (1956).

The DISCO interview schedule was developed from the HBS schedules for use in diagnostic work for both clinical work and research purposes. It was designed to include referrals with associated physical or psychiatric conditions or other developmental disorders such as dyslexia and dyspraxia. The schedule was developed to be relevant for all these variations in the clinical pictures. It has been expanded to include past behavior from infancy onward as well as for current state. It is also suitable for use with adults (see section on "Clinical Uses" below).

Reliability and validity studies of the DISCO items were published in 2002 (see section on "Psychometric Data" below), when the ninth version was current (Leekam et al. 2002; Wing et al. 2002). The schedule has had two subsequent minor revisions and the current version is the 11th revision, and research has been published on both these versions (see "Psychometric Data" section).

Psychometric Data

The psychometric properties of the DISCO have been examined in studies carried out in the UK, in Sweden, the Netherlands, and Belgium. The UK studies (Leekam et al. 2002; Wing et al. 2002) used DISCO-9 to carry out inter-rater reliability and validity analyses with data from 82 cases aged 3–11 years. Thirty-six had autistic spectrum disorder, 17 had learning disability, and 14 had

language impairments. Inter-rater reliability was analyzed for over 400 items in the interview. Inter-rater reliability was high with kappa coefficient or intra-class correlation at .75 or higher for over 80% of the interview items. Analyses with the same sample examined two algorithms based on the ninth revision of the schedule (DISCO 9). Algorithm diagnoses were applied to interview items in order to analyze the relationship between clinical and algorithm diagnoses and the inter-rater reliability between interviewers for each algorithm output. Results showed that clinical diagnosis was significantly related to the diagnostic outputs for both algorithms and inter-rater reliability was high for both algorithms. The ICD childhood disorder algorithm produced more discrepant diagnoses than the Wing and Gould's autistic spectrum algorithm. Analysis of the ICD-10 algorithm items and combination of items helped to explain the reason for these discrepancies.

The Swedish study (Nygren et al. 2009) used a translation of the tenth version of the DISCO (DISCO-10). Validity analysis compared DISCO-10 algorithm diagnoses with clinical diagnoses and with Autism Diagnostic Interview-Revised (ADI-R) algorithm diagnoses in 57 cases of children and adults. Results showed good to excellent inter-rater reliability in 40 cases. The criterion validity was excellent when compared with clinical diagnoses and the ADI-R. The report concluded that although the DISCO-10 is not as widely used as the ADI-R, the evidence shows that it has the same level of psychometric credibility.

Psychometric research was carried out by Maljaars et al. (2012) using the Dutch translation of the DISCO-11. Their study included young children with different levels of intellectual disability (ID) including no ID, borderline, mild, moderate, and severe ID. DISCO algorithms for ICD-10 childhood autism and atypical autism were used in comparison with clinical classification and the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 1999) and Social Communication Questionnaire (SCQ; Rutter et al. 2003) to examine its criterion and convergent validity. Sensitivity and specificity of the DISCO

were .96 and .79, respectively. Strong agreement was found between DISCO-11 and ADOS classification ($k = 0.69$, $p < 0.001$), although lower agreement was found with the SCQ ($k = 0.49$, $p < 0.001$). Comparisons with clinical diagnosis showed correct classification for the majority of cases with mismatches mainly explained by cases in the moderate and severe ID range. These results confirm that the DISCO has good criterion and convergent validity. This was especially the case for those with average intelligence or mild intellectual disability. However, the specificity was lower for those with moderate and severe levels of intellectual disability ($IQ < 50$), in line with previous findings.

The most recent psychometric study (Kent et al. 2013), used DISCO data to test the DSM-5 Draft description. The study used different ways of combining the rules for a DSM-5 diagnosis. It also tested the DSM-5 criteria on children and adults with different ability levels. Using the optimal rules for diagnosis described in the study (the Modified algorithm), sensitivity and specificity of the DISCO were 1.0 and 0.84, respectively. In a follow-up study, highly discriminating, "essential" items were identified from the DSM-5 algorithm, to try and identify a reduced set of items that could guide diagnosis. Abbreviation of the DSM-5 algorithm did not significantly affect either sensitivity or specificity within the research samples tested (Carrington et al. 2014). Moreover, a subset of items from the abbreviated DSM-5 item that best discriminated between individuals with autism and individuals with non-autism clinical diagnoses were identified. This set of just 14 items had excellent predictive validity according to best estimate clinical diagnosis, and also showed excellent agreement with established DSM-5 and ICD-10 DISCO algorithms (Carrington et al. 2015).

Clinical Uses

The DISCO schedule can be used in clinical practice to fulfill three main purposes – to provide a clinical description, to make a clinical diagnosis, and to provide recommendations.

First, it can be used to provide a *clinical description* by assisting the clinician in collecting information needed to compile a developmental history and a description of the present clinical picture. This information, including current level of development in everyday skills and the pattern of behavior, can be used as the basis of a narrative clinical report. Usually, the informant is someone who has known the person concerned from birth. However, when the DISCO is used with an adult and no informant is available to give an early history, the items of the DISCO schedule can be completed for current skills, deficits, and atypical behavior. Second, the DISCO can be used to assist in making a *clinical diagnosis* of autism spectrum disorders as well as of other disorders of development affecting social interaction and communication. Related to this purpose, the schedule can be used to run a number of different diagnostic algorithms according to different classification systems (see “[Description](#)” section earlier). In the case of an adult, where no developmental history is available, a diagnosis according to DSM and ICD is not possible, but the information gathered from the DISCO allows the experienced clinician to use their clinical judgment to make a working diagnosis in order to plan a management program. Finally, another important purpose of the DISCO is to provide information that will guide a clinician’s recommendations concerning programs of education, adult support, and management of behavior.

See Also

- [Asperger, Hans](#)
- [Autism Diagnostic Observation Schedule](#)
- [Diagnostic Instruments in Autistic Spectrum Disorders](#)
- [Epilepsy](#)
- [Fetal Alcohol Syndrome](#)
- [ICD 10 Research Diagnostic Guidelines](#)
- [Intellectual Disability](#)
- [Kanner, Leo](#)
- [Rett Syndrome](#)
- [Wing, Lorna](#)

References and Reading

- American Psychiatric Association (1987). *Diagnostic and statistical manual of mental disorders: Third edition revised* (pp. 33–39). Washington, DC: American Psychiatric Association.
- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders: Fourth edition* (pp. 65–8). Washington DC: American Psychiatric Association.
- American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders, 5th edn*. Author, Washington DC. American Psychiatric Association.
- Barrett, S. L., Uljarević, M., Baker, E. K., Richdale, A. L., Jones, C. R., & Leekam, S. R. (2015). The Adult Repetitive Behaviours Questionnaire-2 (RBQ-2A): a self-report measure of restricted and repetitive behaviours. *Journal of Autism and Developmental Disorders*, 45(11), 3680–3692.
- Barrett, S. L., Uljarević, M., Jones, C. R., & Leekam, S. R. (2018). Assessing subtypes of restricted and repetitive behaviour using the Adult Repetitive Behaviour Questionnaire-2 in autistic adults. *Molecular Autism*, 9(1), 58.
- Billstedt, E., Gillberg, C., & Gillberg, C. (2007). Autism in adults, symptom patterns and early childhood predictors: Use of the DISCO in a community sample followed from childhood. *Journal of Child Psychology and Psychiatry*, 48(11), 1102–1110.
- Brugha, T. S., McManus, S., Bankart, J., Scott, F., Purdon, S., Smith, J., et al. (2011). Epidemiology of autism spectrum disorders in adults in the community in England. *Archives of General Psychiatry*, 68(5), 459–465.
- Carrington, S. J., Kent, R. G., Maljaars, J., Le Couteur, A., Gould, J., Wing, L., Noens, I., Van Berckelaer-Onnes, I., & Leekam, S. R. (2014). DSM-5 autism spectrum disorder: In search of essential behaviours for diagnosis. *Research in Autism Spectrum Disorders*, 8(6), 701–715.
- Carrington, S., Leekam, S., Kent, R., Maljaars, J., Gould, J., Wing, L., Le Couteur, A., Van Berckelaer-Onnes, I., & Noens, I. (2015). Signposting for diagnosis of autism spectrum disorder using the diagnostic interview for social and communication disorders (DISCO). *Research in Autism Spectrum Disorders*, 9, 45–52.
- Carrington, S.J., Barrett, S.L., Sivagamasundari, U., Fretwell, C., Noens, I., Maljaars, J. & Leekam, S. R., (2019). Describing the profile of diagnostic features in autistic adults using an abbreviated version of the Diagnostic Interview for Social and Communication Disorders (DISCO-Abbreviated). *Journal of autism and developmental disorders*, 49(12), 5036–5046.
- Cederlund, M., Hagberg, B., Billstedt, E., Gillberg, I. C., & Gillberg, C. (2008). Asperger syndrome and autism: A comparative longitudinal follow-up study more than 5 years after original diagnosis. *Journal of autism and developmental disorders*, 38(1), 72–85.

- Cooper, J., Moodley, M., & Reynell, J. (1978). Helping language development. Arnold: London.
- Danielsson, S., Gillberg, C., Billstedt, E., Gillberg, C., & Olsson, I. (2005). Epilepsy in young adults with autism: A prospective population-based follow-up study of 120 individuals diagnosed in childhood. *Epilepsia*, 46, 918–923.
- de Vries, A. L. C., Noens, I. L. G., Cohen-Kettenis, P. T., van Berckelaer-Onnes, I. A., & Doreleijers, T. A. (2010). Autism spectrum disorders in gender dysphoric children and adolescents. *Journal of Autism and Developmental Disorders*, 40(8), 930–936.
- Egan, D., Illingworth, R.S., & MacKeith, R.C. (1969). Developmental screening 0-5 years. Clinics in developmental medicine No. 30. London: Heinemann.
- Ehlers, S., & Gillberg, C. (1993). The epidemiology of Asperger syndrome. A total population study. *Journal of Child Psychology and Psychiatry*, 34, 1327–1350.
- Eisenmajer, R., Prior, M., Leekam, S., Wing, L., Ong, B., Gould, J., et al. (1998). Delayed language onset as a predictor of clinical symptoms in pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 28(6), 527–533.
- Ellefsen, A., Kampmann, H., Billstedt, E., Gillberg, C., & Gillberg, C. (2007). Autism in the Faroe islands: An epidemiological study. *Journal of Autism and Developmental Disorders*, 37, 437–444.
- Gillberg, I. C., & Gillberg, C. (1989). Asperger syndrome—some epidemiological considerations: a research note. *Journal of Child Psychology and Psychiatry*, 30(4), 631–638.
- Griffiths, R. (1967). The abilities of babies. London: University of London Press.
- Honey, E., Leekam, S., Turner, M., & McConachie, H. (2007). Repetitive behaviour and play in typically developing children and children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(6), 1107–1115.
- Kanner, L., & Eisenberg, L. (1956). Early infantile autism, 1943–1955. *American Journal of Orthopsychiatry*, 26(3), 55–65.
- Kent, R. G., Carrington, J. S., Couteur, A., Gould, J., Wing, L., Maljaars, J., Noens, I., van Berckelaer-Onnes, I., & Leekam, S. R. (2013). Diagnosing autism Spectrum disorder: Who will get a DSM-5 diagnosis? *Journal of Child Psychology and Psychiatry*, 54(11), 1242–1250.
- Larkin, F., Meins, E., Centifanti, L. C., Fernyhough, C., & Leekam, S. R. (2017). How does restricted and repetitive behavior relate to language and cognition in typical development? *Development and Psychopathology*, 29(3), 863–874.
- Leekam, S., Libby, S., Wing, L., Gould, J., & Gillberg, C. (2000). Comparison of ICD-10 and Gillberg's criteria for Asperger syndrome. *Autism*, 4(1), 11–28.
- Leekam, S., Libby, S., Wing, L., Gould, J., & Taylor, C. (2002). Diagnostic interview for social and communication disorders: Algorithms for ICD-10 childhood autism and wing and Gould autistic spectrum disorder. *Journal of Child Psychology and Psychiatry*, 43, 327–342.
- Leekam, S. R., Nieto, C., Libby, S., Wing, L., & Gould, J. (2007a). Describing the sensory abnormalities of individuals with autism. *Journal of Autism and Developmental Disorders*, 37(5), 894–910.
- Leekam, S., Tandos, J., McConachie, H., Meins, E., Parkinson, K., Wright, C., et al. (2007b). Repetitive behaviours in typically developing 2-year-olds. *Journal of Child Psychology and Psychiatry*, 48(11), 1131–1138.
- Lidstone, J., Uljarević, M., Sullivan, J., Rodgers, J., McConachie, H., Freeston, M., Le Couteur, A., Prior, M., & Leekam, S., (2014) Relations among restricted and repetitive behaviors, anxiety and sensory features in children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 8(2), 82–92.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism Diagnostic Interview-Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24, 659–686.
- Lord, C., Rutter, M., & DiLavore, P. C. (1999). Autism Diagnostic Observation Schedule—Generic. Dissertation Abstracts International Section A: Humanities and Social Sciences.
- Lotter, V. (1966). Epidemiology of autistic conditions in young children: I. Prevalence. *Social Psychiatry*, 1, 124–137.
- Lotter, V. (1967). Epidemiology of autistic conditions in young children: II. Some characteristics of the parents and children. *Social Psychiatry*, 1, 163–173.
- Maljaars, J., Noens, I., Scholte, E., & Berckelaer-Onnes, I. (2012). Evaluation of the criterion and convergent validity of the Diagnostic Interview for Social and Communication Disorders in young and low-functioning children. *Autism: International Journal of Research and Practice*, 16(5), 487–497.
- Mukherjee, R. A. S., Layton, M., Yacoub, R., & Turk, J. (2011). Autism and autistic traits in people exposed to heavy prenatal alcohol. *Advances in Mental Health and Intellectual Disabilities*, 5(1), 42–49.
- Nygren, G., Hagberg, B., Billstedt, E., Skoglund, A., Gillberg, C., & Johansson, M. (2009). The Swedish version of the diagnostic interview for social and communication disorders (DISCO-10) psychometric properties. *Journal of Autism and Developmental Disorders*, 39(5), 730–741.
- O'Nions, E., Gould, J., Christie, P., Gillberg, C., Viding, E., & Happé, F. (2016). Identifying features of 'pathological demand avoidance' using the Diagnostic Interview for Social and Communication Disorders (DISCO). *European child & adolescent psychiatry*, 25(4), 407–419.
- Prior, M., Eisenmajer, R., Leekam, S., Wing, L., Gould, J., Ong, B., et al. (1998). Are there subgroups within the autistic spectrum? A cluster analysis of a group of children with autistic spectrum disorders. *Journal of*

- Child Psychology and Psychiatry, and Allied Disciplines*, 39(6), 893–902.
- Rutter, M., Bailey, A., & Lord, C. (2003). The social communication questionnaire: Manual. Western Psychological Services.
- Sheridan, M.D. (1973). Children's developmental progress. Windsor: N.F.E.R.
- Sheridan, M.D. (1977). Spontaneous play in early childhood. Windsor: N.F.E.R.
- Soenen, S., Van Berckelaer-Onnes, I., & Scholte, E. (2009). Patterns of intellectual, adaptive and behavioral functioning in individuals with mild mental retardation. *Research in Developmental Disabilities*, 30(3), 433–444.
- Turk, J., Bax, M., Williams, C., Amin, P., Eriksson, M., & Gillberg, C. (2008). Autism spectrum disorder in children with and without epilepsy: Impact on social functioning and communication. *Acta Paediatrica*, 98(4), 675–681.
- Uljarević, M., Arnott, B., Carrington, S. J., Meins, E., Fernyhough, C., McConachie, H., Le Couteur, A., & Leekam, S. R. (2017). Development of restricted and repetitive behaviors from 15 to 77 months: Stability of two distinct subtypes? *Developmental Psychology*, 53(10), 1859–1868.
- Wing, L. (1969). The handicaps of autistic children: A comparative study. *Journal of Child Psychology and Psychiatry*, 10, 1–40.
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11, 115–29.
- Wing, L. (1988). The continuum of autistic characteristics. In E. Schopler & G. Mesibov (Eds.), *Diagnosis and assessment in autism* (pp. 91–110). New York: Plenum.
- Wing, L. (1996). *The autistic spectrum*. London: Constable.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11–29.
- Wing, L., & Shah, A. (2000). Catatonia in autistic spectrum disorders. *British Journal of Psychiatry*, 176, 357–362.
- Wing, L., Leekam, S., Libby, S., Gould, J., & Larcombe, M. (2002). Diagnostic interview for social and communication disorders: Background, inter-rater reliability and clinical use. *Journal of Child Psychology and Psychiatry*, 43, 307–325.
- World Health Organisation (1993). The ICD-10 classification of mental and behavioural disorders. Diagnostic criteria for research. Geneva: World Health Organisation.
- Wulffaert, J., Van Berckelaer-Onnes, I. A., & Scholte, E. M. (2009a). Autistic disorder symptoms in Rett syndrome. *Autism*, 13(6), 567–581.
- Wulffaert, J., van Berckelaer-Onnes, I., Kroonenberg, P., Scholte, E., Bhuiyan, Z., & Hennekam, R. (2009b). Simultaneous analysis of the behavioural phenotype, physical factors, and parenting stress in people with Cornelia de Lange syndrome. *Journal of Intellectual Disability Research*, 53(7), 604–619.

Diagnostic Interviews

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Definition

The diagnostic interview (DI) is a central component of the process (diagnostic process) in which, for a variety of reasons ranging from research to the development of an intervention plan, a decision is made as to whether there is sufficient evidence in an individual's symptoms and signs for a diagnosis of one or more of the "disorder(s)" defined by the criteria of the internationally agreed diagnostic classification systems (► [DSM-5](#)).

Historical Background

Following the initial descriptions of autism and Asperger syndrome in the 1940s, agreed criteria emerged slowly and a number of checklists were developed which matched a list of symptomatology against the criteria evolving at the time in ICD 9 (1975) and DSM-II (1980) (DSM-III) focusing on accounts of observable behavior, particularly in childhood, notably the E-2 (Rimland diagnostic form for behavior disturbed children (E-2)) and the Autism Behavior Checklist (ABC).

In the 1960s, in both America and the UK, the search for greater consistency and precision in psychiatric diagnosis led to the development of standardized diagnostic interviews; initially schedules of standard questions, these became elaborated into a more clinical interview that encouraged the interviewer to cross-examine the patient until the nature of the symptom was clear (Wing et al. 1967). A decade later, the same model led to the development of more systematic interviews in making the diagnosis of autism (as the prototypical disorder of the pervasive developmental disorders). Wing and Gould produced the

Handicaps, Behavior, and Skills Schedule (HBSS) which they later refined into the Diagnostic Interview for Social and Communication Disorder (DISCO), Schopler and Reichler developed the Childhood Autism Rating Scale (CARS), and Le Couteur, Rutter, and Lord produced the Autism Diagnostic Interview (later revised to become the ADI-R) (Autism Diagnostic Interview-Revised). These standardized diagnostic instruments consist of a semi-structured interview (based on the agreed symptom criteria) with an adult informant and became recognized as the “gold standard” in terms of their comprehensiveness and reliability in obtaining a clinical history.

The identification of a broader spectrum of autism disorders (ASD), going beyond the original narrow definition for autism, led to an extension of the content and form of ► [diagnostic instruments](#) in autistic spectrum disorders. Examples of these are the Asperger Syndrome Diagnostic Interview (ASDI) and the Autism Questionnaire (AQ) (Baron-Cohen et al. 2001) for Asperger syndrome, the Pervasive Developmental Disorder in Mental Retardation Scale (PDD-MRS) for people with intellectual disability (Kraijer and de Bildt 2005), and the Diagnostic Interview Guide for use in general adult psychiatry (Royal College of Psychiatrists 2011). The recognition of autistic traits (broader autism phenotype – broader spectrum prevalence) in the relatives of people with ASD has led to the development of a variety of interviews to identify these behavioral and personality characteristics.

In many of these, the emphasis was on obtaining material from informants (usually parents) about behavior. At the end of the 1990s, the Autism Diagnostic Observation Schedule (ADOS) was developed as a play and activities based assessment with the individual; this assessment is described as a series of tightly defined, detailed observations which systematically elicits autistic symptomatology.

In the last decade, the number of instruments, their use varying from screening to diagnosis, has reflected the mounting interest in ASD, while increased public awareness and the Internet have fostered the growth of self-rating scales and the demand for confirmatory diagnostic interviews.

Current Knowledge

The Content of the Interview

There are a variety of models for conducting a diagnostic interview. The structure or framework for the DI is important, but there is no compelling evidence to recommend any particular interview format for any specific situation. For all DIs (irrespective of the interview format), the underlying context is the social engagement and interaction between the interviewer and the interviewee. The interviewing skills and attitudes of the interviewer (clinician or researcher) affect the quality of the interaction which in turn influences the success of the information-gathering process. The responses of the interviewee (also affected by many factors including whether they already know the interviewer; the interviewee is in fact the subject of the interview; his or her intellectual and communicative ability, motivation, emotional state, and so on) and the setting can also influence the outcome and “success” of the diagnostic interview (DI).

It should include:

1. An account of the individual’s current concerns – the symptoms that have brought to interview at this particular time and their development.
2. A systematic survey of the symptomatology associated with ASD, especially that which is directly related to the diagnostic criteria. This review should also include consideration of other behavioral features known to be commonly associated with ASD such as motor coordination, sensory and perceptual symptoms, and feeding and bowel problems. It should include any other behavioral problems recognizing that these can occur in response to a variety of potentially modifiable influences from toothache to a change in school timetable or work colleagues.
3. The wider setting – the individual’s everyday life and activities, relationships, and accomplishments.
4. The structure of their family and any history of developmental or psychiatric disorder.

5. An account of the individual's development and their acquisition of skills, not just in infancy and early childhood but subsequently, through school and after, to give a detailed "developmental history".
6. An account of any other anomaly, past or present, including developmental, psychiatric, or medical disorder as well as of any other adversity including deprivation or substance abuse.

The DI will usually be complimented by a direct examination of the individual together with the collation of background reports (including direct observation in other settings). All these sources of information will contribute to the accuracy and value of the final, "best estimate," diagnostic conclusions which, in turn, will inform the multiagency needs- and skills-based management plan.

While the DI and examination are conceptually distinct, in practice, there is likely to be a substantial overlap. For example, when an individual is being interviewed and asked to provide their own account, the clinician will be considering the way the account is being given, the quality and content of the social interaction, and other individual characteristics (such as their appearance, behavior, and communication). These factors will inevitably affect the interaction between the clinician and the interviewee, thus shaping the course of the DI.

How the DI progresses is at least in part dependent on the skills of the interviewer, their training and expertise, as well as the setting of the interview and the expectations of the interviewees. All these different aspects can foster a "dialogue" between clinician and individual. Instruments may be combined for history-taking and observation although, in the end, the distinction between them is one of emphasis rather than clear-cut. For example, while the framework of observational ratings is central to the ADOS, it is also a semi-structured interview.

The Format of the Diagnostic Interview

The interview may take a range of formats depending on its purpose:

1. *Unstructured.* The structure is not immediately apparent, but the interviewer's impression determines the content, purpose, and conclusions of the interview. Its primary purpose may be a different one with diagnosis as a secondary consideration. Such an assessment depends greatly on the individual clinician's experience, and for this reason it is likely to be difficult to understand or replicate.
2. *Semi-structured/interviewer based.* The interview, usually based on a predetermined diagnostic framework, has well-defined symptoms to be explored. Usually conducted in a conversational style, it takes the form of required questions supplemented by additional, optional, open-ended prompts as necessary until there is sufficient information for the trained interviewer to make the coding judgment for each item and section of the interview. The precision and clarity with which symptoms and their codings are defined contribute to the quality of the instrument.
3. *Structured/respondent based.* The trained interviewer closely follows a defined format without deviation; the interview may be restricted further by giving the interviewee a limited number of choices. The interviewer is not called upon to make any clinical judgment (and, indeed, may not know very much about ASD and other diagnoses to complete the interview).

The result is a relatively high inter-rater reliability and an interview that lends itself to being turned into a self-completion questionnaire. This can be administered as a pre-interview contribution or completed in a computerized format (e.g., the E-2 or Autism Spectrum Quotient (AQ) questionnaires). Increasingly for some individuals, access to this type of questionnaire has been a staging post in their journey to diagnosis.

4. *A composite.* The interview incorporates the material from a preinterview questionnaire. Not only is this a more effective use of time, substantially shortening the DI, but many individuals are more comfortable (and therefore more open) with the impersonality of a self-

completion questionnaire. Examples of DIs that use information-collected preinterview include the Developmental, Dimensional, and Diagnostic Interview (3Di) (Skuse et al. 2004) and the Adult Asperger Assessment (AAA) (Baron-Cohen et al. 2005).

It is difficult to define the point at which the self-completion or screening checklist becomes a more formal diagnostic instrument as this will depend on the skill, experience, and intent of those employing it.

The more standardized the format for gathering and organizing the information, the greater the consistency in the data collected and the diagnoses arrived at by clinicians and researchers of varied experience and views and from different centers. However, validity is lost with increasing rigidity that limits the clinician's skills. Using agreed diagnostic systems permits prospective research as well as making clinical material available for retrospective review for service and academic analysis. The whole process is more transparent and can be taught to trainees.

The style of interview has to be appropriate to the task in hand: a structured interview, with its very narrow, specific remit, will be used for screening or surveys and as such can be administered by a technician. The semi-structured interview provides the framework for a more in-depth assessment when a definitive research or clinical diagnosis is required and usually includes one or more summary algorithms to identify ASD using prespecified thresholds. However, the protean presentations of ASD and the demands of clinical work mean that, in the end, even the best of these instruments does not remove the need for knowledge and experience of ASD in coming to a clinical diagnosis which will inform the diagnostic formulation and intervention planning. There are cases, notably in adulthood, of individuals with less clear-cut presentations where it is difficult to discern the pattern of symptoms. It is here that the experience of working with a wide variety of people across the variations of age, ability, gender, ethnicity, and comorbidity makes it possible to appreciate the characteristic impairments of

ASD. In addition, within the assessment team, there needs to be sufficient knowledge and experience to recognize the developmental and psychiatric disorders that are associated with ASD (notably attention deficit hyperactivity disorder (ADHD) (► [Attention Deficit/Hyperactivity Disorder](#)) and developmental coordination disorder (► [Developmental Coordination Disorder](#))).

The choice to use a particular diagnostic instrument will be informed by both the purpose of the interview and the features of the instrument. For example, the ADI-R (► [Autism Diagnostic Interview-Revised](#)) provides a summary lifetime diagnosis, using information about early childhood and the current state for key aspects of behavior and development and a record of the particular unusual behaviors (such as restricted, repetitive mannerisms and stereotyped behaviors) relevant to the decision as to whether probably all references to Pervasive developmental disorder should now be replaced with autism spectrum disorder is present or not. The frequency and intensity of each symptom is carefully graded to give a detailed quantified picture of key components. The DISCO (► [Diagnostic Interview for Social and Communication Disorders](#)) takes a rather broader approach to arrive at a systematic description that allows the identification of other developmental disorders. The 3Di is a computer-based interview designed to focus on current functioning to assess autistic traits, social impairments, and comorbidities in children of normal ability. The content of the interview generates a structured report with summary algorithms of symptom profiles for autism and common non-autistic comorbidities. By contrast, the CARS (► [Childhood Autism Rating Scale](#)) draws on observation as well as interview. The format is much less structured, guiding the interviewer through the relevant domains rather than individual symptoms, requiring the researcher/clinician to reach the coding decisions through the integration of information from subject and informants

Most structured instruments (► [Diagnostic Instruments in Autistic Spectrum Disorders](#)) have been designed for a specific group, often

defined by age (e.g., childhood) or ability. This means that the phrasing or materials might not be suitable for a different “client” group when adaptation of materials and further reliability and validity studies would be required.

As adults come forward for diagnosis, including, for example, those with a severe intellectual disability, women of normal ability, and individuals with preexisting psychiatric and personality disorder diagnoses, the challenge will be how best to tailor the format and content of the DI appropriately. A particular issue is the necessity of a developmental history to confirm that the evidence of delayed or deviant development dates back to early childhood. This becomes particularly important in adulthood should there be a need to differentiate ASD from other disorders (such as schizophrenia (► [Schizophrenia](#)) or dissocial or obsessive-compulsive personality disorders (► [Obsessive-Compulsive Disorder \(OCD\)](#))). However, it is this client group who may experience real difficulty finding an informant with accurate knowledge about their early development.

Whatever the format of the DI, training in its use is required. This applies especially to standardized instruments where the more structured the interview, the more straightforward the training. While it may be obtained by attending a specific training course, receiving in-house individual tuition, or using a self-taught program, it should include a check that clinician/researcher has reached an acceptable standard of accuracy and reliability. This should be followed by regular opportunities to maintain consistency and reliability over time. Undertaking the rating of standardized videos or attending joint sessions with colleagues can help to maintain best practice in administration of the procedure as well as reliability between colleagues and different centers. However, because this is time consuming and may be seen as additional pressure on scarce resources, it is all too easily overlooked.

Implementing the Interview

A DI may take place as a single event in one setting or be spread across several sessions and settings. The venue (clinic, specialist center,

home, school, or other setting) will depend on the needs of individual, their family/carers, clinicians, and services. For example, a very anxious individual or a disabled relative may only be accessible in the home; a clinic may be the only place to get the opinion of a busy clinician or be the best place to provide the structured, calm setting needed to see someone at their best. It may be necessary to go to a school, nursery, or workplace to see the context and thereby understand what is happening there. Observation in different settings may allow some distinction to be made between what behavior is pervasive and what is situational and in response to a particular environment or set of circumstances.

The DI must provide sufficient information for the interviewer to decide whether the symptoms and signs are:

1. *Sufficiently pronounced in their intensity or frequency* to cross the threshold that separates so-called normal variation for developmental progress and personal characteristics from disorder: threshold that may well vary according to the problems experienced by the individual, the context and situation, and the “demands” and expectations placed upon them. For example, a young child who has managed well in their home with a supportive family may find it much more difficult to settle into an educational setting such as preschool if they do not have sufficiently flexible communication, social and play skills to join in with other young children or cope with new and unexpected changes in routine in an otherwise familiar environment. Similarly, an adult who may have learnt to manage effectively in a particular workplace may still find that he/she is less able to succeed in social and more personal relationships. For the diagnostic interview to be successful, the interviewer needs to understand the importance of gathering information about the development of the individual’s behavior in different settings and contexts over time. This may well require (especially for children and young people but often also for adults) information from other

informants who know the subject well in different settings.

2. *Sufficiently close to the currently agreed criteria* (► **DSM-5**) for a diagnosis of ASD or might be explained better by some other disorder. ASD is a neurodevelopmental disorder defined by its onset in early childhood, something that may be difficult to confirm in later adulthood.

The interview therefore has to enable the clinician to distinguish the signals of ASD against the background noise of other, complicating disorders, particularly other developmental and psychiatric disorders such as intellectual disability, specific speech and language disorders, attention deficit hyperactivity disorder (ADHD), epilepsy, and/or mental health problems such as anxiety or obsessive-compulsive disorder.

The interview must also be appropriate *to its immediate purpose*: for example, the requirements for inclusion in a research study might be more stringent than those needed as the basis for clinical or administrative planning.

A diagnosis may be sought for many reasons, ranging from inclusion in a research study, accessing specific treatments and interventions, eligibility for particular education provision, achieving financial benefits, and gaining family understanding, through to assisting a court to understand the needs of the individual. Most importantly, it can give the individual a more complete understanding of their profile of strengths and impairments. The diagnostic interview also provides a benchmark against which subsequent progress can be measured. It has to be tuned accordingly to meet these specific requirements.

The results of the interview should be valid (i.e., that others would agree with the diagnostic conclusions) and reliable (they would be the same if repeated, whether by the same clinician or others). The process needs to be acceptable to all, sufficiently transparent to be understood, and sufficiently valued for the results to be useful.

Most instruments require the interviewer to make judgments and ascribe a numerical score to each item in the assessment. These scores may be

collated to give symptom and/or domain scores which can be summarized within one or more instrument-specific diagnostic algorithms. For a number of instruments, usually those that have been developed for research, the reliability and validity of the algorithm scores and instrument-specific diagnostic thresholds have been tested and refined in different populations. However, it is important to recognize that a diagnostic algorithm score derived from a particular instrument may contribute to, but is not equivalent to, a clinical diagnosis. This is something broader, using an internationally agreed diagnostic classification system, based on information gathered from several sources, and often involving professionals working in different agencies to provide a multidisciplinary assessment. This information, in turn, will contribute to, but is not sufficient for, the development of a management plan. The DI, which may include the use of a structured instrument, is an opportunity for the development of a dialogue between the interviewer, the individual, and the family/carer and, as such, can also provide the context for sharing the outcome of the multi-agency assessment.

One of the great values of using an agreed diagnostic classification system is that it facilitates the possibility of successful research collaborations between clinical academic centers as well as making clinical material available for service review and analysis. With greater transparency between services and centers, there is an increase in research capacity, the ability to share new knowledge and significant developments, and in opportunities for trainees to learn from the experiences of their colleagues.

Future Directions

A number of standardized instruments are now in routine use for the DI providing both a valuable framework for the history as well as the basis for the start of a therapeutic relationship with individuals and families. Many are time consuming and resource intense, and this has to be balanced against the benefits of the therapeutic alliance and detailed descriptions of behavior. While the use of a detailed

DI may well be appropriate for a behavioral syndrome that has such a variety of presentations and underlying disorders, there is great pressure to develop briefer processes and ever greater consistency while maintaining validity.

The value of increasingly sophisticated online questionnaires as an adjunct to the DI needs to be investigated. New measures will also be required as further understanding of the complexity of the autism spectrum across the lifespan becomes available. However, the development of new instruments is a complex and expensive task. An equally important challenge is to investigate the best ways of getting reliable information from different sources to complement the DI this will enable the clinician/researcher, referred individual, and family to achieve a valid diagnostic formulation that in turn leads to an accurate needs- and skills-based management plan.

The recognition of autistic traits in the families of people with autism has led to the development of instruments to identify these which, once sufficiently validated and standardized, will be published.

In spite of many claims and much research, there is still no reliable laboratory test for ASD. However, even if such a test were ever developed, its results would complement the diagnostic interview rather than replace it, a model seen in other medical conditions as, for example, the use of genetic testing in the clinical diagnosis of Down or Rett syndrome.

With increasing awareness and understanding of ASD, there is likely to be greater emphasis on the identification of the strengths, skills, needs, and impairments of the individual and their family, as well as on diagnosis, to inform a dimensional diagnosis and profile across different domains of functioning. Although separate assessments may be needed to measure different aspects of an individual's functioning (e.g., social responsiveness, language and flexibility, anomalies in sensory sensitivity and motor coordination), this information will always need to be collated alongside the findings of a DI to achieve a diagnostic formulation. At least for the foreseeable future, classification systems used in clinical and research practice, together with other social

and resource pressures, will continue to require a categorical diagnosis of ASD.

See Also

- ▶ Anecdotal Observation
- ▶ Asperger Syndrome Diagnostic Interview
- ▶ Autism Behavior Checklist
- ▶ Autism Diagnostic Interview-Revised
- ▶ Autism Diagnostic Observation Schedule
- ▶ Broader Autism Phenotype
- ▶ Childhood Autism Rating Scale
- ▶ Developmental Coordination Disorder
- ▶ Diagnostic Interview for Social and Communication Disorders
- ▶ Diagnostic Instruments in Autistic Spectrum Disorders
- ▶ Diagnostic Process
- ▶ Dimensional Versus Categorical Classification
- ▶ DISCO
- ▶ DSM-III
- ▶ DSM-5
- ▶ Dyspraxia
- ▶ Evaluation of Sensory Processing
- ▶ Informal Assessment
- ▶ Obsessive-Compulsive Disorder (OCD)
- ▶ Psychotic Disorder
- ▶ Schizophrenia
- ▶ Sensory Impairment in Autism
- ▶ Theory of Mind

References and Reading

- Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from asperger syndrome/high-functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders*, 31(1), 5–17.
- Baron-Cohen, S., Wheelwright, S., Robinson, J., & Woodbury-Smith, M. (2005). The Adult Asperger Assessment (AAA). A diagnostic method. *Journal of Autism and Developmental Disorders*, 35(6), 807–819.
- Kraijer, D., & de Bildt, A. (2005). The PDD-MRS: An instrument for identification of autism spectrum disorders in persons with mental retardation. *Journal of Autism and Developmental Disorders*, 35(4), 499–513.
- Royal College of Psychiatrists. (2011). *Diagnostic interview guide for adults with autism spectrum disorder (ASD)*.

http://www.rcpsych.ac.uk/pdf/asperger_interview_use_this_one.pdf

- Skuse, D., Warrington, R., Bishop, D., Chowdhury, U., Lau, J., & Mandy, W. (2004). *The developmental, dimensional and diagnostic interview (3Di): A novel computerized assessment for autism spectrum disorders*. <http://www.ixdx.org/>
- Wing, J. K., Birley, J. L. T., Cooper, J. E., Graham, P., & Isaacs, A. D. (1967). Reliability of a procedure for measuring and classifying "present psychiatric state." *The British Journal of Psychiatry*, 113, 499–515.

Diagnostic Overshadowing

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Definition

Diagnostic overshadowing refers to the negative bias impacting a clinician's judgment regarding co-occurring disorders in individuals who have intellectual disabilities or other mental illness. Symptoms or behaviors that may be due to a specific mental illness are attributed to another disorder, historically Mental Retardation, without considering alternative etiology.

Historical Background

Reiss, Levitan, and Szyszko first coined the term "diagnostic overshadowing" to describe the tendency to assess individuals with intellectual disability less accurately (Reiss et al. 1982a, b; Reiss and Szyszko 1983). Subsequent research has consistently demonstrated that the cognitive deficits displayed by an individual negatively impacted the ability of clinicians to make accurate judgments with regard to other co-occurring disorders (c.f., Jopp and Keys 2001; White et al. 1995).

Jopp and Keys provide a review of the concept of diagnostic overshadowing in addition to possible moderators (Jopp and Keys 2001). Their

review indicated that most clinician-based variables, such as nature of clinical position (e.g., school, clinical and counseling psychologists, social workers), educational level (e.g., graduate student vs. Ph.D.), and years of experience, were not associated with the strength of the bias. Moreover, though the presence of multiple disabilities would presumably be more inherently difficult to disentangle for a diagnosing clinician, the research clearly indicated that the clinician's perception of the cognitive deficits present in the individual being assessed was the most salient feature reducing diagnostic accuracy.

Diagnostic overshadowing causes clinicians to overlook a range of comorbid mental illness in individuals with intellectual disability, including phobias, schizophrenia, avoidant personality disorder, and depression (Jopp and Keys 2001). As Jopp and Keys note, the bias potentially serves to reduce both sensitivity and specificity – two important components of accurate diagnosis. Sensitivity refers to the ability to accurately diagnose individuals who have a disorder, while specificity refers to the ability to accurately rule out individuals who do not have a particular disorder. Diagnostic overshadowing may reduce sensitivity by creating more false negatives, such as when a child with a cognitive deficit is not diagnosed with an anxiety disorder that they truly have. It may also reduce specificity by increasing the number of false positives, such as when a child is diagnosed with an intellectual disability, when they really have another disorder that has caused the cognitive deficit.

Only one factor has been found to moderate the impact of diagnostic overshadowing, which is how clinicians process information, termed cognitive complexity (Jopp and Keys 2001). That is, when a clinician is able to view a patient's behaviors in a multidimensional fashion, incorporating a wide range of thoughts, feelings, and behaviors, which in turn leads to generating multiple hypotheses, the impact of the patient's cognitive deficits and the resulting diagnostic overshadowing can be reduced.

The concept of diagnostic overshadowing has direct epidemiological implications. If diagnostic accuracy is impacted and individuals are missed

with regard to a diagnosis, or misdiagnosed, then prevalence data may be misleading or incorrect. Moreover, epidemiological studies not only inform prevalence and incidence of a disorder and its associated characteristics, but can also help guide etiological understanding. For example, this was especially the case in autism wherein the initial report of the prevalence of co-occurring epilepsy in autism led to scientists to examine biological mechanisms in contrast to the non-biological theories promulgated at the time (Bryson and Smith 1998; Lotter 1974). If diagnostic overshadowing causes clinicians to overlook important co-occurring disorders, advancements in etiological understanding may also be impacted.

Current Knowledge

More recently, clinicians and researchers have extended the notion of diagnostic overshadowing beyond individuals with cognitive deficits to those with other disorders such as autism. In addition, diagnostic overshadowing has been extended beyond the diagnostic process to discussions regarding how it may impact treatment. For example, some researchers have found that diagnostic overshadowing has direct treatment implications. How an individual is diagnosed affects what treatments are recommended by their treating providers. If the treating provider is affected by diagnostic overshadowing and thus does not recognize other disorders, those other difficulties will not be appropriately treated. Minnes and Steiner found that parents of children with Down syndrome, for example, reported more problems receiving treatment for the co-occurring illnesses, such as cataracts, thyroid problems, and possible dementia (Minnes and Steiner 2009).

Researchers have proposed that the same mechanism biasing clinicians who work with individuals with cognitive deficits may also apply to clinicians who work with individuals with autism. More specifically, given the wide range of cognitive abilities in addition to the other symptoms of autism, such as

communication problems and other challenging behaviors, clinicians may be underdiagnosing comorbid disorders in individuals with autism, despite the accumulation of evidence that demonstrates a high prevalence of co-occurring disorders in autism such as mood disorders, attentional disorders, and behavioral disorders (Simonoff et al. 2008).

Others have demonstrated how diagnostic overshadowing has impacted epidemiological research results. For example, Charman and colleagues, using the Special Needs and Autism Project sample (i.e., total population cohort of 56,946 children in the UK ages 9–10), compared the concordance of their research-based diagnosis to the diagnoses derived from local services in children with IQs above and below 70. They found that the amount of children diagnosed with an autism spectrum disorder from local services who had cognitive impairment was less than those in that group that had been diagnosed through their epidemiological research design, 25% compared to 45% (Charman et al. 2009). These results demonstrate the potential diagnostic overshadowing bias and its impact on prevalence rates of autism depending on method of ascertainment.

Future Directions

In their 2001 review, Jopp and Keys noted four areas in need of research with regard to diagnostic overshadowing which remain relevant despite the broadening of diagnostic overshadowing beyond intellectual disability: (1) improve specification of clinical decisions that make up diagnostic overshadowing, (2) note the processes whereby diagnostic overshadowing occurs, (3) increase the appreciation of other variables such as the environment as they impact overshadowing, and (4) explore overshadowing more fully using qualitative and other methodologies (Jopp and Keys 2001). How much overshadowing actually takes place in local and “real world” clinics, as opposed to the vignettes used in the research that explore its presence, needs to be more fully explored, as well as a better delineation of how diagnostic

overshadowing is impacting other diagnoses, such as autism, in addition to cognitive deficits alone.

See Also

- [Autism](#)
- [Epidemiology](#)

References and Reading

- Bryson, S. E., & Smith, I. (1998). Epidemiology of autism: Prevalence, associated characteristics, and implications for research and service delivery. *Mental Retardation and Developmental Disabilities Research Reviews*, 4, 9–103.
- Charman, T., Pickles, A., Chandler, S., Wing, L., Bryson, S., Simonoff, E., et al. (2009). Commentary: Effects of diagnostic thresholds and research vs service and administrative diagnosis on autism prevalence. *International Journal of Epidemiology*, 38(5), 1234–1238. author reply 1243–1234.
- Jopp, D. A., & Keys, C. B. (2001). Diagnostic overshadowing reviewed and reconsidered. *American Journal on Mental Retardation*, 106(5), 416–433.
- Lotter, V. (1974). Factors related to outcome in autistic children. *Journal of Autism and Childhood Schizophrenia*, 4(3), 263–277.
- Minnes, P., & Steiner, K. (2009). Parent views on enhancing the quality of health care for their children with fragile X syndrome, autism or Down syndrome. *Child: Care, Health and Development*, 35(2), 250–256.
- Reiss, S., & Szyszko, J. (1983). Diagnostic overshadowing and professional experience with mentally retarded persons. *American Journal of Mental Deficiency*, 87(4), 396–402.
- Reiss, S., Levitan, G. W., & McNally, R. J. (1982a). Emotionally disturbed mentally retarded people: An underserved population. *American Psychologist*, 37(4), 361–367.
- Reiss, S., Levitan, G. W., & Szyszko, J. (1982b). Emotional disturbance and mental retardation: Diagnostic overshadowing. *American Journal of Mental Deficiency*, 86(6), 567–574.
- Simonoff, E., Pickles, A., Charman, T., Chandler, T. L., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(8), 921–929.
- White, M. J., Nichols, C. N., Cook, R. S., Spengler, P. M., Walker, B. S., & Look, K. K. (1995). Diagnostic overshadowing and mental retardation: A meta-analysis. *American Journal on Mental Retardation*, 100(3), 293–298.

Diagnostic Process

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Definition

Autism spectrum disorders (ASDs) are a group of heterogeneous disorders that share overlapping diagnostic criteria. These include deficits in communication and socialization, and restricted interests and repetitive behaviors. Deficits in socialization are the hallmark of all ASDs, and deficits in this area are diagnostically required to meet criteria for all of the ASDs. Important to note is that even with these three core domains of impairment, heterogeneity across these symptoms exists on an individual basis. As such, the classification systems used to categorize the core impairments in ASD have been amended over time. Even still, the diagnostic process of ASD is complicated by numerous factors including the differential diagnosis within ASDs, a push to identify symptoms of ASD at very young ages, and the stability of ASD diagnoses over time. Thus, this entry will review these factors in regard to the diagnostic process of ASD.

Historical Background

Autism spectrum disorders were first introduced into the diagnostic nomenclature in 1980 (i.e., *Diagnostic and Statistical Manual of Mental Disorders, Third Edition [DSM-III]*; American Psychiatric Association [APA] 1980) under the category of pervasive developmental disorders. However, two of the currently recognized diagnoses, pervasive developmental disorder not otherwise specified and Asperger's disorder, were not introduced as diagnostic disorders until 1987 and 1994, respectively. Although the diagnostic categories have changed throughout the different editions of the *DSM*, the main areas of impairment (i.e., symptom domains) have remained largely

consistent. For example, deficits in interpersonal relationships, impairment in communication, and bizarre responses to the environment were the three main symptom domains in the *DSM-III*. Currently, the three main symptom domains include impairment in social interaction, impairment in communication, and restricted interests and repetitive behaviors (APA 2000).

Current Knowledge

ASD is an umbrella term used to encompass five disorders: autistic disorder (AD), Asperger's disorder (AS), pervasive developmental disorder not otherwise specified (PDD-NOS), Rett's disorder, and childhood disintegrative disorder. Given the very low incidence of these latter two conditions, the focus of this overview is related to AD, AS, and PDD-NOS. A child is referred for an assessment of ASD if developmental milestones are not met or after observations of behaviors related to diagnoses on the autism spectrum. Initial observations of symptoms or concerns regarding developmental milestones are most often made by teachers, day-care providers, pediatricians, and parents. As with other psychiatric disorders, best practices in regard to the assessment of ASD is to incorporate multiple informants and multiple methods. Informants come in the form of teachers, day-care providers, parents, grandparents, guardians, and other therapists familiar with the child (e.g., physical therapist, speech therapist). The assessment for a diagnosis of ASD should include an interview, an observation, and the administration of at least one assessment measure that has been psychometrically investigated to screen/diagnose ASD. It is also common practice to utilize measures of cognitive functioning and adaptive function to assess for a comorbid diagnosis of intellectual disability. During the entirety of the assessment sessions, clinicians assess for the triad of impairments indicative of an ASD diagnosis: deficits in communication, impairments in socialization, and the presence of repetitive motor movements (e.g., hand flapping) or intense and restricted interests (e.g., will only play with cars). Clinicians should also be mindful

of the high rates of comorbid psychopathology and challenging behaviors, and should use ASD measures that also address these issues.

More recently, there has been a move to diagnose ASD at very young ages. Fortunately, assessments designed to screen for symptoms of ASD in young populations have been developed. The measures with the best research to support them for this purpose are the *Modified Checklist for Autism in Toddlers (M-CHAT*; Robins et al. 2001) and the *Baby and Infant Screen for Children with aUtistic Traits-Part1 (BISCUIT-Part1*; Matson et al. 2007). Both measures are rating scales that can be administered in 30 min or less, have determined cutoff scores, and present with sound psychometric properties. Given the push to identify symptomatology indicative of ASD at younger ages, researchers have explored the diagnostic stability of symptoms using samples of toddlers. Outcomes of such investigations have provided support for the diagnostic stability for ASD for children under age three (Worley et al. 2011). If diagnostic status changes, it is often from one ASD to another (e.g., PDD-NOS to AD; Cox et al. 1999; Eaves and Ho 2004; Kleinman et al. 2008). Thus, at this time, research supports the need and the ability to reliably diagnose ASD during the toddler years. Diagnosing ASD at very young ages is important, as early intervention is key for long-term success.

Another factor to consider when choosing an assessment tool is the ability of the measure to differentiate between the various ASDs, given the blurred boundaries of the various disorders comprising the spectrum. The reader will note that with the appearance of the *DSM-V*, all ASDs will be collapsed together into one diagnostic category. However, for the purpose of service planning, evaluating the severity and symptom profiles will remain very important.

Although ASD can be reliably differentiated from other developmental disorders, differential diagnosis between AD, AS, and PDD-NOS remains difficult. This phenomenon is largely due to the overlapping diagnostic criteria used to define these disorders in the diagnostic nomenclature. More specifically, the diagnostic criteria for PDD-NOS are ill defined with no specific number

of criteria established to obtain this diagnosis. In addition, the diagnostic symptoms for AD and AS overlap exactly in the area of socialization and repetitive behavior and restricted interests. As a result, many researchers have examined differences between disorders comprising the spectrum. However, findings are largely inconsistent. Nonetheless, it is still important to assess for the different ASDs as a means of conforming with the current diagnostic classification system. Two measures that assist in the differential diagnosis between the various ASDs are *Autism Spectrum Disorders Diagnostic for Child (ASD-DC;* Matson and González 2007) and the *Pervasive Developmental Disorders Behavior Inventory (PDDBI;* Cohen and Sudhalter 1999). Both tests are rating scales that can be completed in 20 min or less.

In addition to the need to differentially diagnose between different psychiatric disorders, medical conditions also need to be ruled out as symptoms of certain medical conditions may simulate symptoms of certain psychiatric disorders. As such, a medical assessment should be conducted prior to making an ASD diagnosis. The most important factors to assess during the medical evaluation would be the child's hearing, vision, and oral functioning. Ruling out any problems with the aforementioned is vital to ensure that symptoms of ASD are not better accounted for by medical conditions. For example, individuals with ASD present with delays in communication and socialization. If a child is having trouble hearing or having oral motor problems, these challenges would affect their ability to speak and, subsequently, their ability to socialize with others. In addition, visual impairments could account for other symptoms such as failure to initiate and sustain eye contact and joint attention.

Lastly, intellectual disability (ID) is a highly comorbid condition with ASD. As such, the assessment process should incorporate evaluations of both adaptive skills and intellectual functioning to assess for deficits specific to these areas. Deficits in cognition and adaptive behavior are required to meet criteria for a diagnosis of ID. The assessment of intellectual functioning also assists with the differential diagnosis of

ASDs, specifically between AS and AD. For instance, individuals diagnosed with AS typically fall within the average range of cognitive functioning whereas those diagnosed with AD often have a comorbid diagnosis of ID. However, these boundaries become blurred when examining those with AS and "high-functioning autism" (HFA), most of whom have intelligence quotients (IQ) in the average range. Conversely, individuals diagnosed with AS tend to have higher verbal than performance IQs, and those diagnosed with HFA tend to have higher performance than verbal IQs.

In sum, the assessment process is conducted to arrive at a diagnosis of either AD, AS, or PDD-NOS or to rule out these diagnoses. First, AD is characterized by impairments in all three core domain areas. Children with AD are often referred for an assessment at very young ages since parents' first concerns typically arise during the first year of life. In contrast, individuals meeting diagnostic criteria for AS are often not identified until later in childhood. Likely, this is due to deficits in socialization which are the most impairing symptom associated with a diagnosis of AS. As social demands increase with age, these deficits become more pronounced and more obvious. Thus, deficits in this area become more apparent to the outside observer as the child develops and has more social interactions with others. In addition, unlike children diagnosed with AD, language development is not delayed for children meeting diagnostic criteria for AS. Instead, individuals diagnosed with AS tend to have exceptional vocabularies. Therefore, as toddlers, there is no obvious cause for concern for children eventually meeting criteria for an AS diagnosis. Lastly, a diagnosis of PDD-NOS is given when symptoms of ASD are present, but the individual does not meet the criteria for another disorder on the spectrum. Therefore, the diagnostic category of PDD-NOS is a subthreshold category. Children comprising this diagnostic category have less severe deficits in socialization and may have minimal deficits in communication or less presentation of restricted interests or repetitive behaviors when compared to a child meeting criteria for AD. In addition, it may be that these children present with the same symptoms of a child

meeting criteria for AD, but the age of onset occurs after 36 months of age.

Future Directions

The current classification system for ASD is categorical. However, this approach is problematic due to the poorly defined boundaries of the disorders comprising the autism spectrum. Due to these poorly defined boundaries, there has been a failure to find consistent differences between AD, AS, and PDD-NOS in regard to diagnostic criteria. As such, a dimensional approach to diagnosing has been proposed. This approach to diagnosing ASD is supported in the literature by researchers who have examined the underlying latent structure of symptoms of ASDs utilizing cluster analytic techniques or taxometric analyses. Although results often contradict each other, it has been suggested that the underlying taxon of ASD is dimensional (Boisjoli 2010; Verté et al. 2006).

As a result of the overlap in the behavioral phenotype of ASDs, the APA (2010) has proposed revisions for ASD to be included in the DSM-V, set to be published in 2013. The revisions include utilizing a dimensional approach to diagnosing ASD. As such, there will be no subcategories of ASD, but instead one diagnostic entity referred to as autism spectrum disorder. In regard to diagnostic criteria, impairment related to socialization and communication would be amalgamated into one domain. The second domain refers to symptoms of restricted interests and repetitive behaviors. The third domain would indicate that symptoms need to be present in early childhood; however, early childhood is not further defined. Lastly, the symptoms must cause impairment in everyday functioning (APA 2010).

These changes will bring about further modifications to the diagnostic process. For instance, parceling out differences between the various ASDs would no longer be necessary, since PDD-NOS, AS, and AD would no longer represent discrete diagnostic entities. However, given what will be even greater heterogeneity within the ASD diagnostic category, being able to identify symptom severity will still be critical. Even more

important, existing measures that assess for symptoms of ASD would need to be renormed to follow the new diagnostic criteria and continuing emerging research.

See Also

- ▶ Asperger Syndrome
- ▶ Autistic Disorder
- ▶ Diagnostic Interviews
- ▶ Pervasive Developmental Disorder Not Otherwise Specified

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders – Text revision* (4th ed.). Washington, DC: Author.
- American Psychiatric Association. (2010). *DSM-V*. Retrieved October 20, 2010, from www.dsm5.org
- Boisjoli, J. (2010). *A taxometric analysis of autism spectrum disorders in toddlers*. Unpublished doctoral dissertation, Louisiana State University.
- Cohen, I. L., & Sudhalter, V. (1999). *PDD behavior inventory: Professional manual*. Lutz: Psychological Assessment Resources.
- Cox, A., Klein, K., Charman, T., Baird, G., Baron-Cohen, S., Swettenham, J., et al. (1999). Autism spectrum disorders at 20 and 42 months age: Stability of clinical ADI-R diagnosis. *Journal of Child Psychology and Psychiatry*, 40, 719–732.
- Eaves, L. C., & Ho, H. H. (2004). The very early identification of autism: Outcome to age 4 Â§ – 5. *Journal of Autism and Developmental Disorders*, 34, 367–378.
- Kleinman, J. M., Ventola, P. E., Pandey, J., Verbalis, A. D., Barton, M., Hodgson, S., et al. (2008). Diagnostic stability in very young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38, 606–615.
- Matson, J. L., & González, M. L. (2007). *Autism spectrum disorders – diagnosis – child version*. Baton Rouge: Disability Consultants, LLC. Translated into Italian, Chinese, Hebrew, and Japanese.
- Matson, J. L., Boisjoli, J., & Wilkins, J. (2007). *The baby and infant screen for children with autism traits (BISCUIT)*. Baton Rouge: Disability Consultants, LLC.
- Robins, D. L., Fein, D., Barton, M. L., & Green, J. A. (2001). The modified checklist for autism in toddlers: An initial study investigating the early detection of

autism and pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 31, 131–144.

Verté, S., Geurts, H. M., Roeyers, H., Rosseel, Y., Oosterlaan, J., & Sergeant, J. A. (2006). Can the children's communication checklist differentiate autism spectrum subtypes? *Autism*, 10, 266–287.

Worley, J. A., Matson, J. L., Mahan, S., Kozlowski, A. M., & Neal, D. (2011). Stability of symptoms of autism spectrum disorders in toddlers: An examination using the Baby and Infant Screen for Children with aUtism – Part I. *Developmental Neurorehabilitation*, 14, 36–40.

Diagnostic Substitution

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Definition

Diagnostic substitution has been hypothesized as one possible explanation for why growing numbers of children have been classified with a label of autism in publicly funded service systems such as special education and state systems of care for people with developmental disabilities. The term has been used in two related ways. One refers to a historical shift in the probability of being labeled with autism, whereby some proportion of children labeled with autism in recent years would have been classified with a different label had they been served by the same organization at a previous point in time. The other refers to individual children initially being labeled with one diagnosis and then being reclassified with autism at a later age. In each case, the hypothesis predicts that as enrollment tallies in the autism category increased, there would be some corresponding decrease in the number of children being enrolled and labeled in other administrative categories (e.g., ► [Intellectual Disability](#)).

Evidence testing the diagnostic substitution hypothesis has been mixed. One study of data from California's service system for people with developmental disabilities from 1987 to 1994 found little change in the administrative

prevalence of intellectual disability, whereas autism rates increased nearly fivefold (Croen et al. 2002; Croen and Grether 2003). Another study examined special education enrollment data from Minnesota for the years 1991–2001 and found no substantial decrease in administrative prevalence for other disabilities while autism enrollment counts were increasing (Gurney et al. 2003).

A study using state-level special education data for the whole United States found that the growing administrative prevalence of autism from 1994 to 2003 was strongly associated with decreasing prevalence in other disability categories, though not in every state (Shattuck 2006). A study of special education enrollment in British Columbia from 1996 to 2004 found that nearly one third of growing autism prevalence was explained by children who had initially been classified with some other type of disability being relabeled with autism (Coo et al. 2008).

See Also

► [Intellectual Disability](#)

References and Reading

- Blaxill, M. F., Baskin, D. S., & Spitzer, W. O. (2003). Commentary: Blaxill, Baskin, and Spitzer on Croen et al. (2002), The changing prevalence of autism in California. *Journal of Autism and Developmental Disorders*, 33(2), 223–226.
- Coo, H., Ouellette-Kuntz, H., Lloyd, J., Kasmara, L., Holden, J., & Lewis, M. (2008). Trends in autism prevalence: Diagnostic substitution revisited. *Journal of Autism and Developmental Disorders*, 38, 1036–1047.
- Croen, L. A., & Grether, J. K. (2003). Response: A response to Blaxill, Baskin, and Spitzer on Croen et al. (2002), "The changing prevalence of autism in California". *Journal of Autism and Developmental Disorders*, 33(2), 227–229.
- Croen, L. A., Grether, J. K., Hoogstrate, J., & Selvin, S. (2002). The changing prevalence of autism in California. *Journal of Autism and Developmental Disorders*, 32(3), 207–215.
- Gurney, J. G., Fritz, M. S., Ness, K. K., Sievers, P., Newschaffer, C. J., & Shapiro, E. G. (2003). Analysis of prevalence trends of autism spectrum disorder in

Minnesota. *Archives of Pediatrics and Adolescent Medicine*, 157, 622–627.

Shattuck, P. (2006). The contribution of diagnostic substitution to the growing administrative prevalence of autism in U.S. Special education. *Pediatrics*, 117(4), 1028–1037.

Diastat

► [Diazepam](#)

Diazepam

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Synonyms

[Diastat](#); [Valium](#)

Definition

A long-acting anxiolytic medication in the benzodiazepine class. Diazepam is commonly used in the treatment of anxiety disorders, agitation, and spasticity. Diazepam and other medicines of this class bind to benzodiazepine receptors, enhancing the inhibitory effects of γ -aminobutyric acid (GABA). Side effects of benzodiazepines can include sedation, dizziness, fatigue, and confusion. Additionally, prolonged use of diazepam or other benzodiazepines may lead to tolerance and physical dependence.

See Also

► [Anxiety](#)

References and Reading

Stahl, S. (2009). *The prescriber's guide: Stahl's essential psychopharmacology* (pp. 139–143). Cambridge: Cambridge University Press.

DiBAS-R Validation

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Synonyms

[Autism spectrum disorder \(ASD\)](#); [Diagnostic Behavioral Assessment for Autism Spectrum disorders-Revised \(DiBAS-R\)](#); [Intellectual disability \(ID\)](#); [Social communication and interaction \(SCI\)](#); [Stereotyped and restrictive behaviors and sensory interests \(SRS\)](#)

Description

The Diagnostic Behavioral Assessment for Autism Spectrum disorders-Revised (DiBAS-R) is a quick screening scale for adults with intellectual disability (ID) who are suspected of having autism spectrum disorder (ASD). It is based on the DSM-IV/5 and ICD-10 (APA 2013; Dilling 2014) criteria for ASD and consists of 19 items that are rated on a 4-point scale from “certainly true” to “never true.” It is completed by close caregivers and focuses on current behaviors within the past 3 months. Each question is worded in plain language (e.g., “Can you tell how he/she feels by his/her facial expression?” or “Does he/she show challenging behavior when unpredictable changes occur?”) to allow the administration by individuals without any specific knowledge of ASD. The scale is self-explanatory and thus does not require any preparatory training. Completion and scoring takes about 5 min.

Factor analysis indicated two subscales, namely, the *Social Communication and Interaction* (SCI) and the *Stereotypy, Rigidity, and Sensory Abnormalities* (SRS) subscale. The result of the DiBAS-R is indicative of ASD if an individual shows increased scores on the overall score (>29) and on both subscales (> 21 on the

SCI subscale and > 5 on the SRS subscale). A linear regression model revealed a significant association between the level of ID and the DiBAS-R scores, indicating an association between higher levels of ID and an increased ASD-specific symptom load. Therefore, the DiBAS-R is especially useful in individuals with mild to moderate ID. The questionnaire exists in German and English.

Historical Background

Due to the lack of appropriate screening instruments for adults with ID, a population at risk for comorbid ASD, a 20-item questionnaire, the Diagnostic Behavioral Assessment for ASD (DiBAS), was developed (Sappok et al. 2014b). It is derived from the ICD-10 and DSM-5 criteria for ASD. Information from a review of the literature focusing on symptoms that differentiate between persons with ID with and without additional ASD, an analysis of the *Autism Diagnostic Observation Schedule* (ADOS; Lord et al. 1989) items and the *Autism-Checklist* (ACL; Sappok et al. 2014c) items, and experiences from clinical experts in the field of ID and ASD endorsed the item formulation process. The items score on a 4-point ordinal Likert scale with 3 points for “certainly true,” 2 points for “often true,” 1 point for “sometimes true,” and 0 points for “never true.” The final scores range from 0 to 60 with higher scores indicating a higher ASD symptom load. These items were evaluated in terms of diagnostic validity using the final diagnostic classification of the multidisciplinary case conference as an external criterion. In a pilot study, the DiBAS was applied to 91 patients with ID and suspected ASD (Sappok et al. 2014b). The DiBAS revealed an AUC of 0.81, a sensitivity of 83%, a specificity of 64%, and a kappa of 0.47 (Sappok et al. 2014b). As 8 of the original 20 items did not differentiate sufficiently between ASD and non-ASD, in a next step, these items were replaced by another eight ICD-10/DSM-5-based questions to replenish the DiBAS-Revised (DiBAS-R).

Psychometric Data

A first study assessed the DiBAS-R in a sample that consisted of 219 adults with ID and who were suspected of having ASD who were admitted to a department of psychiatry in Berlin, Germany, from January 2012 to July 2013 (Sappok et al. 2014a). The mean age was 35 years, 57% were males, and 35% of participants were diagnosed with additional ASD. Factor analysis yielded 2 consistent dimensions with 12 items in the *Social Communication and Interaction* subscale (SCI) and 7 items in the *Stereotypy, Rigidity, and Sensory Abnormalities* subscale (SRS). The internal consistencies were *Cronbach's alpha* = 0.91 for the SCI subscale, 0.84 for the SRS subscale, and 0.91 for the overall scale. The DiBAS-R revealed sensitivity and specificity values of 81% each (Sappok et al. 2014b). The reliability was excellent: inter-rater reliability ICC = 0.88 and test-re-test reliability $r = 0.93$. The DiBAS-R scores were highly correlated with other ASD-specific measures (*Social Communication Questionnaire* (SCQ) $r = 0.52$ Berument et al. 1999; Rutter et al. 2003; *Scale of Pervasive Developmental Disorder in Mentally Retarded Persons* (PDD-MRS) $r = 0.5$ Kraijer and de Bildt 2005; *Autism-Checklist* (ACL) $r = 0.59$ Sappok et al. 2014c). The low correlation with the *Modified Overt Aggression Scale* supported the divergent validity of the instrument.

In a second study, an independent sample of 381 adults with ID and who were suspected of having ASD was recruited from August 2013 to December 2016 (Heinrich et al. 2018). The mean age was 40.5 years, 58% were males, and 24% were finally diagnosed with ASD. The diagnostic validity of the DiBAS-R including its given cutoff points was showing an overall agreement with the reference diagnoses of 70% (sensitivity, 82%; specificity, 67%). Sensitivity (79%) and specificity (84%) were more balanced in individuals with mild to moderate ID (sensitivity, 79%; specificity, 84%). Specificity decreased to 34% in persons with severe to profound ID, while the sensitivity was still good (83%). The level of ID as well as its interaction with ASD explained a significant proportion of

the variance in the DiBAS-R scores. Thus, decreasing levels of functioning may result in an increasing overlap in ASD-like symptomatology in individuals with and without ASD, which in turn may lead to a decrease in the diagnostic utility of the DiBAS-R scores.

In a third study, the combination of the DiBAS-R and the ACL were evaluated in 148 persons with ID who were suspected of having ASD (Mutsaerts et al. 2016). The specificity values increased from 75% for each instrument used alone to 88% when two positive screening results were used in combination. The sensitivity values increased from 75% for the DiBAS-R and 91% for the ACL to 95% when at least one positive screening result was used. Different combinations of the ASD screening instruments DiBAS-R and ACL lead to improvements in sensitivity and specificity. The instruments can therefore be used in a complementary way to further improve the overall accuracy of each scale.

Clinical Uses

The DiBAS-R can be used as a quick screening instrument for ASD in adults with ID. It is easy to administer and can be applied in various settings, e.g., hospitals or outpatient clinics or in population-based studies. No special knowledge of the diagnostic criteria of ASD is needed to complete the questionnaire, and no training is necessary to compute the final scoring. Thus, the scale can be used by general practitioners, psychiatrists, or psychologists to screen for ASD in research and clinical practice. The administration of the scale requires about 5 min for scoring and computing and is independent of the collaboration of the examined individual. The validity and reliability results suggest that this scale represents an efficient approach to initial ASD screening of adults with ID. However, it cannot replace a comprehensive assessment for ASD such as interview-based procedures like the Autism Diagnostic Interview-Revised (ADI-R; Lord et al. 1994) or direct behavioral observations in a standardized setting like the Autism Diagnostic Observation Schedule (ADOS; Lord et al.

1989). Its psychometric properties particularly support its use in persons with mild to moderate ID. The DiBAS-R can be applied for clinical and research purposes.

See Also

- [ADI-R](#)
- [ADOS](#)
- [PDD](#)
- [SCQ](#)

References and Reading

- APA. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- Berument, S. K., Rutter, M., Lord, C., Pickles, A., & Bailey, A. (1999). Autism screening questionnaire: Diagnostic validity. *The British Journal of Psychiatry*, 175, 444–451.
- Dilling, H. (Ed.). (2014). *Internationale Klassifikation psychischer Störungen: ICD-10 Kapitel V (F), Klinisch-diagnostische Leitlinien* (9th ed.). Bern: Huber.
- Heinrich, M., Böhm, J., & Sappok, T. (2018). Diagnosing autism in adults with intellectual disability: Validation of the DiBAS-R in an independent sample. *Journal of Autism and Developmental Disorders*, 48(2), 341–350.
- Kraijer, D., & de Bildt, A. (2005). The PDD-MRS: An instrument for identification of autism spectrum disorders in persons with mental retardation. *Journal of Autism and Developmental Disorders*, 35(4), 499–513.
- Lord, C., Rutter, M., Goode, S., Heemsbergen, J., Jordan, H., Mawhood, L., et al. (1989). Autism diagnostic observation schedule: A standardized observation of communicative and social behavior. *Journal of Autism and Developmental Disorders*, 19(2), 185–212.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24, 659–685.
- Mutsaerts, C. G., Heinrich, M., Sterkenburg, P. S., & Sappok, T. (2016). Screening for ASD in adults with ID-moving toward a standard using the DiBAS-R and the ACL. *Journal of Intellectual Disability Research*, 60, 512–522.
- Rutter, M., Bailey, A., Lord, C., & Berument, S. K. (2003). *Social communication questionnaire*. Los Angeles: Western Psychological Services.

- Sappok, T., Gaul, I., Bergmann, T., Dziobek, I., Bölte, S., Diefenbacher, A., et al. (2014a). The diagnostic behavioral assessment for autism spectrum disorder – Revised: A screening instrument for adults with intellectual disability suspected of autism spectrum disorders. *Research in Autism Spectrum Disorders*. <https://doi.org/10.1016/j.rasd.2013.12.016>.
- Sappok, T., Gaul, I., Dziobek, I., Bölte, S., Diefenbacher, A., & Bergmann, T. (2014b). Der Diagnostische Beobachtungsbogen für Autismus Spektrumstörungen (DiBAS): Ein Screeninginstrument für Erwachsene mit Intelligenzminderung bei Autismusverdacht. *Psychiatrische Praxis*, 41, 1–7.
- Sappok, T., Heinrich, M., & Diefenbacher, A. (2014c). Psychometrische Eigenschaften der Autismus-Checkliste (ACL) für erwachsene Menschen mit Intelligenzminderung. *Psychiatrische Praxis*, 41, 37–44.
- Sappok, T., Gaul, I., Bergmann, T., Dziobek, I., Bölte, S., Diefenbacher, A., & Heinrich, M. (2015). *DiBAS-R Der Diagnostische Beobachtungsbogen für Autismspektrum-Störungen – Revidiert. Ein Screening-Instrument für Erwachsene mit Intelligenzminderung*. Bern: Hogrefe Verlag.

Dichotic Listening

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Description

Dichotic listening is the auditory process that involves listening with both ears. Dichotic listening can be broken into two different processes: binaural integration and binaural separation. Binaural integration is the ability to perceive different acoustic messages presented to the left and right ears at the same time. Binaural separation is the ability to perceive an acoustic message in one ear while ignoring a different acoustic message in the other ear. In order to perceive the acoustic messages in both ears, the outer, middle, and inner ears must be working properly, but more importantly, the auditory brainstem nuclei, auditory cortical neurons, as well as neurons in the corpus

callosum must be functioning properly. Individuals with dichotic listening deficits often have difficulty hearing in the presence of background noise.

Historical Background

Dichotic speech testing was first introduced by Broadbent in 1954. It requires the simultaneous presentation of different speech stimuli to each of the ears; the listener must repeat back everything that is heard (binaural integration) or what is heard in one ear only (binaural separation). In 1961, Kimura first used these tests to demonstrate hemispheric asymmetry and cortical dysfunction. She demonstrated contralateral auditory deficits following temporal lobe lesions. These findings indicate that if a lesion is located in the left temporal lobe, the auditory signal presented to the right ear will not be perceived. In typical individuals (those with normal hearing and no known lesions in the central auditory nervous system), a right ear advantage has consistently been reported (Berlin et al. 1973; Dirks 1964; Kimura 1961a, b). Right ear advantage refers to the ability to better perceive the auditory signal presented to the right ear than the speech signal in the left ear. The right ear advantage exists because the hemisphere in the brain responsible for processing the speech signal is in the left hemisphere. Since the contralateral pathways are stronger, the speech signal presented to the right ear travels directly to the left hemisphere for processing; however, the speech signal presented to the left ear must travel to the right hemisphere and across the corpus callosum to the left hemisphere to be processed (creating a slight time delay).

Currently, a variety of dichotic listening tests are available for clinical use. Common dichotic speech tests, specifically binaural integration tests, are the Dichotic Digits Test (Musiek 1983; Musiek and Wilson 1979; Musiek et al. 1979); the Staggered Spondaic Word Test (SSW; Katz 1962), and Dichotic Consonant-Vowels (Dichotic CVs; Berlin et al. 1973). Some common

binaural separation tests are Competing Sentences (Willeford 1977) and the Synthetic Sentence Identification with Contralateral Competing Message (SSI-CCM; Jerger 1970).

Psychometric Data

Dichotic listening tests, such as the Dichotic Digits, Dichotic CVs, SSW, Competing Sentences, and SSI-CCM, have been shown to be sensitive and specific to central auditory nervous system lesions, including interhemispheric lesions (for review, see Musiek and Pinheiro 1985). Peripheral hearing sensitivity should be symmetrical and normal when using dichotic listening tests since any hearing differences between ears can influence test results.

Clinical Uses

Dichotic listening tests are used in clinical audiology to evaluate the central auditory processes of binaural integration and separation. These tests may be used in the assessment of children or adults with possible central auditory nervous system dysfunction. Since dichotic listening tests (tests of binaural integration and separation) evaluate two critical auditory processes, it is important for these tests to be included in a central auditory processing test battery. Clinically, the person administering dichotic listening tests should take into consideration the cognitive, hearing, speech, and language abilities of the individual being tested as these factors may affect performance. Thus, dichotic listening tests are not typically administered to children with autism, and performance on these tests should be interpreted with caution in this population.

See Also

- Central Auditory Processing Disorder
- Corpus Callosum
- Temporal Lobes

References and Reading

- Berlin, C., Lowe-Bell, S., Cullen, J., Thompson, S., & Loovis, C. (1973). Dichotic speech perception: An interpretation of right-ear advantage and temporal offset effects. *Journal of the Acoustical Society of America*, 53, 699–709.
- Broadbent, D. (1954). The role of auditory localization in attention and memory span. *Journal of Experimental Psychology*, 47, 191–196.
- Chermak, G., & Musiek, F. (1997). *Central auditory processing disorders: New perspectives*. San Diego: Singular Publishing Group.
- Dirks, D. (1964). Perception of dichotic and monaural verbal material and cerebral dominance for speech. *Acta Otolaryngologica*, 58, 73–80.
- Efron, R. (1985). The central auditory system and issues related to hemispheric specialization. In M. Pinheiro & F. Musiek (Eds.), *Assessment of central auditory dysfunction: Foundations and clinical correlates* (pp. 143–155). Baltimore: Williams & Wilkins.
- Jerger, J. (1970). Development of the synthetic sentence identification (SSI) as a tool for speech audiometry. In C. Rojskjaer (Ed.), *Speech audiometry*. Odense: Danavox.
- Katz, J. (1962). The use of staggered spondaic words for assessing the integrity of the central auditory system. *Journal of Auditory Research*, 2, 327–337.
- Kimura, D. (1961a). Some effects of temporal lobe damage on auditory perception. *Canadian Journal of Psychology*, 15, 156–165.
- Kimura, D. (1961b). Cerebral dominance and the perception of verbal stimuli. *Canadian Journal of Psychology*, 15, 166–171.
- Musiek, F. (1983). Assessment of central auditory dysfunction: The Dichotic Digit test revisited. *Ear and Hearing*, 4, 79–83.
- Musiek, F., & Pinheiro, M. (1985). Dichotic speech tests in the detection of central auditory dysfunction. In M. Pinheiro & F. Musiek (Eds.), *Assessment of central auditory dysfunction: Foundations and clinical correlates* (pp. 201–219). Baltimore: Williams & Wilkins.
- Musiek, F., & Wilson, D. (1979). SSW and Dichotic Digits results pre- and post-commissurotomy: A case report. *Journal of Speech and Hearing Disorders*, 44, 528–533.
- Musiek, F., Wilson, D., & Pinheiro, M. (1979). Audiological manifestations in split-brain patients. *Journal of the American Auditory Society*, 5, 25–29.
- Willeford, J. (1977). Assessing central auditory behaviour in children: A test battery approach. In R. Keith (Ed.), *Central auditory dysfunction* (pp. 43–73). New York: Grune & Stratton.

Didactic Approaches

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Synonyms

[ABA](#); [Adult-/clinician-/teacher-directed approaches](#); [Behavioral approaches](#); [Direct instruction](#)

Definition

A *didactic approach* to teaching refers to a manner of instruction in which information is presented directly from the teacher to the pupil, in which the teacher selects the topic of instruction, controls instructional stimuli, obligates a response from the child, evaluates child responses, and provides reinforcement for correct responses and feedback for incorrect ones. Intervention methods for early communication in children with autism spectrum disorder (ASD) are often divided into three categories: didactic, naturalistic, and pragmatic or developmental.

Didactic approaches utilize a variety of concepts from behavioral theory, including massed trials, operant conditioning, shaping, prompting, chaining, and reinforcement. Difficulty with the generalization and maintenance of behaviors learned through this method along with the passive communication acquired by many children (i.e., waiting on adults' lead during interactions) are some of the drawbacks associated with this approach. Still, the effectiveness of this approach in initiating and expanding expressive language and developing attention to language and comprehension in preverbal children with ASD is supported by research from numerous case studies and several group studies.

As this approach requires a notable amount of adult direction, a passive role as responder by the child, repetitive drills and

practice, and specific events that should occur before and after a child's response, this method is ideally effective if the instructor consistently monitors the student's interest level, readiness for the information being conveyed, and the motivational value of reinforcers. The passivity and prompt dependence that can result from these methods led to the development of more naturalistic instructional techniques (e.g., contemporary applied behaviors analysis).

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Teach Me Language](#)

References and Reading

- Goldstein, H. (2002). Communication intervention for children with Autism: A review of treatment efficacy. *Journal of Autism and Developmental Disorders*, 32, 373–396.
- Howard, J. S., Sparkman, C. R., Cohen, H. G., Green, G., & Stanislaw, H. (2005). A comparison of intensive behavior analytic and eclectic treatments for young children with Autism. *Research in Developmental Disabilities*, 26(4), 359–383.
- Paul, R. (2008). Interventions to improve communication in Autism. *Child and Adolescent Psychiatric Clinics of North America*, 17(4), 835–856.
- Remington, B., Hastings, R., Kovshoff, H., Degli Espinosa, F., Jahr, E., Brown, T., et al. (2007). Early intensive behavioral intervention: Outcomes for children with Autism and their parents after two years. *American Journal of Mental Retardation*, 112, 418–438.
- Rogers, S. (2006). Evidence-based intervention for language development in young children with Autism. In T. Charman & W. Stone (Eds.), *Social and communication development in autism spectrum disorders: Early identification, diagnosis, and intervention* (pp. 143–179). New York: Guilford Press.

Differential Ability Scales

- [Differential Ability Scales \(DAS and DAS-II\)](#)

Differential Ability Scales (DAS and DAS-II)

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Synonyms

Cognitive measures; DAS; DAS-II; Differential ability scales

Description

The Differential Ability Scales, Second Edition (DAS-II; Elliott 2007) is an individually administered test designed to measure distinct cognitive abilities for children and adolescents ages 2 years, 6 months to 17 years, 11 months. The DAS-II is comprised of individual subtests that evaluate strengths and weaknesses of a broad range of learning processes. A General Conceptual Ability (GCA) composite score is generated that reflects conceptual and reasoning abilities. Three cluster scores of the DAS-II measure more specific learning processes: verbal, nonverbal reasoning, and spatial abilities. There is also a Special Nonverbal Composite that can be derived for an individual of any age where the verbal demands are too taxing to obtain standardized results. The core subtests of the DAS-II tap into specific cognitive processes that are used to estimate the cluster and GCA scores, and the abilities they assess are directly related to educational needs at each age range. There are also Diagnostic subtests that measure memory, processing speed, and early school learning abilities. These scores do not contribute to the overall cluster or GCA scores; however, they are still important foundational skills that address a child's profile of cognitive strengths and weaknesses, as well as educational needs.

Core Batteries of the DAS-II

There are two batteries of the DAS-II: Early Years and School Age. Within Early Years, there are two

levels. The first level is for children ages 2 years, 6 months through 3 years, 5 months. This lower level consists of 4 core subtests (*Verbal Comprehension* [VCom], *Naming Vocabulary* [NVoc], *Picture Similarities* [PSim], and *Pattern Construction* [PCon]) and yields a Verbal Ability (VCom + NVoc) and Nonverbal Ability (PSim + PCon) cluster score, as well as the GCA. The upper level is for children ages 3 years, 6 months to 6 years, 11 months and has 6 core subtests (VCom, NVoc, PSim, *Matrices* [Mat], PCon, and *Copying* [Copy]) that yield three cluster scores: Verbal Ability (VCom + NVoc), Nonverbal Ability (PSim + Mat), and Spatial Ability (PCon + Copy), as well as the GCA.

The School-Age battery of the DAS-II can be administered on children ages 7 years, 0 months to 17 years, 11 months, and it is comprised of six core subtests (*Word Definitions* [WDef], *Verbal Similarities* [VSim], Mat, *Sequential and Quantitative Reasoning* [SQR], *Recall of Designs* [RDes], and PCon) that yield three cluster scores: Verbal Ability, Nonverbal Reasoning Ability, and Spatial Ability, as well as the GCA.

Both the Early Years and School-Age batteries of the DAS-II are normed on children between the ages of 5 years, 0 months and 8 years, 11 months. This allows the School-Age subtests to be administered for brighter young children and, in contrast, the Early Years subtests to be administered for older, less cognitively able children.

Diagnostic Subtests of the DAS-II

The Early Years battery of the DAS-II consists of the following ten Diagnostic subtests: *Early Number Concepts* [ENS], *Matching Letter-like Forms* [MLLF], *Phonological Processing* [PhP], *Recall of Sequential Order* [SeqO], *Recall of Digits Forward* [DigF], *Recall of Digits, Backward* [DigB], *Speed of Information Processing* [SIP], *Rapid Naming* [RNa], *Recall of Objects – Immediate and Delayed* [ROBI, ROBD], and *Recognition of Pictures* [RPic]. Seven of these subtests contribute to three cluster scores: School Readiness (ENC + MLLF + PhP), Working Memory (SeqO + DigB), and Processing Speed (SIP + RNa).

The School-Age battery of the DAS-II only consists of seven Diagnostic subtests that yield

two cluster scores: Working Memory (SeqO + DigB) and Processing Speed (SIP + RNam). The School Readiness subtests from the Early Years battery are not included in the School-Age norms, with the exception of PhP, which has norms up to age 12 years, 11 months.

Historical Background

The original Differential Ability Scales (DAS; Elliott 1990) was modeled after the British Ability Scales (BAS; Elliott et al. 1979). Both instruments were unique in the field of intelligence tests in that their focus was on distinct subtest scores that could be used to flush out cognitive profiles of strengths and weaknesses rather than on an overall intelligence quotient or estimation of IQ. This conceptualization of cognitive assessment sets the DAS and subsequent second edition (DAS-II; Elliott 2007) aside from other commonly used measures, such as the Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV; Wechsler 2003) or Stanford-Binet Intelligence Scales, Fifth Edition (SB5; Roid 2003), where the theoretical models tend to focus more on generalized intelligence than on distinct cognitive abilities. Nevertheless, the DAS and DAS-II have an overall composite score that reflects general cognitive functioning (i.e., General Conceptual Ability score) and that is derived from those subtests which load highest on the factor of general intelligence, or *g*. This results in the GCA being a more refined score than other measures of global intelligence that are derived from a broader collection of subtests. However, examiners are cautioned against interpreting the GCA as a global measure of functioning, as many children have a variable cognitive profile that one general score cannot appropriately encapsulate. This is particularly the case for children with autism spectrum disorders (ASD), where scatter within a cognitive profile is the norm rather than the exception (e.g., Klin et al. 2005).

Although the theoretical development of the BAS, DAS, BAS-II (Elliott 1996), and DAS-II

predated theoretical work on the Cattell-Horn-Carroll theory of intelligence (CHC; McGrew 2005), the structure of the DAS-II fits well into the seven-factor CHC model. For instance, the DAS-II Verbal Ability cluster measures crystallized intelligence (*Gc*), the Nonverbal Reasoning cluster measures fluid intelligence (*Gf*), the Spatial Ability cluster measures visual-spatial processing (*Gv*), the Working Memory diagnostic cluster measures short-term memory (*Gsm*), the Recall of Objects subtest measures long-term storage and retrieval (*Glr*), the Processing Speed cluster measures cognitive processing speed (*Gs*), and the Phonological Processing subtest measures auditory processing (*Ga*).

Psychometric Data

The DAS-II has been standardized on a normative sample of 3480 children ages 2 years, 6 months to 17 years, 11 months that is representative of the general population. Data are also available for a range of clinical samples, including developmental risk, learning disabilities, attention deficit/hyperactivity disorder, mild to moderate intellectual disability, and the gifted and talented.

On the DAS and DAS-II, Verbal, Nonverbal, Spatial, and Special Nonverbal cluster scores, as well as the GCA score, are reported in standard scores that have a mean of 100 and standard deviation of 15 and that range from 30 to 170. Individual subtest scores are reported as T scores that have a mean of 50 and a standard deviation of 10 and that range from 10 to 90. T scores are derived from ability scores, which are based on the number of correct responses (i.e., the raw scores) and on the difficulty of administered items, following the Rasch Model of item response theory. The administration and scoring system of the DAS and DAS-II is also different from other common measures in that raw scores are computed based on the number of items administered within a response set, rather than calculating this number in addition to items

below the basal. In this way, children are administered only those set of items that are appropriate in difficulty to their ability level. Subtest scores can be presented as age equivalents that represent the median ability score for each child's performance, and descriptive categories are provided for standard scores that range from "Very High" (70 and above) to "Very Low" (69 and below).

The DAS-II has strong internal reliability, with average reliability coefficients for the Early Years subtests ranging from .79 to .94 and for the School-Age subtests ranging from .74 to .96. The average reliability for the DAS-II GCA is .95 for Early Years and .96 for School Age. Confirmatory factor analyses were conducted to assess the internal validity of the DAS-II, and general results confirmed the existing clusters; for instance, the structure of cognitive abilities varies with age, with fewer models emerging for the youngest children (e.g., Verbal and Nonverbal clusters) and additional models emerging with age (e.g., Spatial, Short-term Memory, and Cognitive Speed clusters).

Correlations between the DAS and DAS-II are strong, with .88 for the GCA and .85 for the SNC. The correlation between the DAS-II GCA and the Wechsler Preschool and Primary Scale of Intelligence, Third Edition (WPPSI-III; Wechsler 2002) Full Scale IQ is .87; however, WPPSI-III Index and FSIQ scores range from 1.7 to 5.1 points higher than DAS-II cluster scores. WISC-IV Index and FSIQ scores also range from 1.2 to 6.6 points higher than DAS-II cluster scores, with a correlation coefficient of .84 between the two measures.

In nonclinical samples, the correlation between the DAS-II GCA and measures of academic achievement is as follows: .82 with the total score of the Wechsler Individual Achievement Test, Second Edition (WIAT-II; Harcourt Assessment 2005); .81 with the Comprehensive Achievement Composite of the Kaufman Test of Educational Achievement, Second Edition (KTEA-II; Kaufman and Kaufman 2004); and .80 with the Total Achievement score of the

Woodcock-Johnson III Tests of Cognitive Abilities (WJ-III; Woodcock et al. 2001).

Clinical Uses

There are several clinical benefits to using the DAS-II when assessing individuals with autism spectrum disorders (ASD; Klin et al. 2005; Saulnier et al. 2011). These advantages include the following:

1. The teaching items that are provided within each DAS-II domain are extraordinarily useful when complex instructions impede a child's ability to comprehend a given verbal request. When the examiner is allowed to model or demonstrate the correct response, the child is better able to comprehend the nature of the task and successfully complete a subtest on which they otherwise might have failed to obtain a basal level of performance.
2. The extended norms on the DAS-II Early Years battery allow for obtaining standard scores for older, more impaired individuals through age 8 years, 11 months – an option not available in other measures (Elliott 2007).
3. The extended norms of the School-Age battery down to age 5 allow for adequately testing younger children with ASD with more advanced cognitive skills.
4. The Special Nonverbal Composite makes it particularly appealing for individuals on the autism spectrum with significant language vulnerabilities for whom the language demands on the verbal tasks are too taxing. The SNC is also useful for other unique samples, such as children with speech, language, and/or hearing impairments or children who are not fluent in English.
5. The results can generate recommendations for educational and treatment programming that are clinically relevant to each child.

The DAS-II is also extremely useful for clinical research in ASD. First, the extensive age range

makes it possible to conduct scientific studies on both cohort and longitudinal studies of children between the ages of 2 and 17. Second, the extended norms allow for utilizing the same battery for varying levels of functioning. Finally, the core subtests can be administered quickly while generating a more comprehensive measure of cognitive functioning than an abbreviated measure of intelligence.

There have been several studies using the DAS that highlight its utility in detecting learning disabilities and cognitive delays. For instance, in one study comparing composite scores between the DAS and WISC-III, children with learning disabilities evidenced a specific weakness in the Nonverbal Reasoning cluster of the DAS that was not demonstrated on the Perceptual Reasoning Index of the WISC-III (Dumont et al. 1996). The majority of research on cognitive profiles in autism spectrum disorders (ASD) has been conducted using the Wechsler Scales. Less research has investigated DAS and DAS-II profiles in ASD, despite the fact that many researchers have used both measures as part of the characterization process for research paradigms. A study conducted by Joseph, Tager-Flusberg, and Lord (2002) used the DAS on a longitudinal sample of children with and without ASD. They found that the majority of preschool-aged children exhibited lower verbal than nonverbal cluster scores and that greater discrepancies between verbal and nonverbal abilities were detected in ASD vs. the normative sample, with this gap widening with age. Furthermore, children with larger gaps between their verbal and nonverbal skills had greater social impairments, and impaired social functioning was independent of their verbal skills.

See Also

- Achievement Testing
- Cognitive Skills
- Educational Testing
- Intelligence Quotient
- Psychological Assessment
- Standardization

- Standardized Tests
- Wechsler Preschool and Primary Scale of Intelligence
- Woodcock-Johnson Cognitive and Achievement Batteries

References and Reading

- Dumont, R., Cruse, C., Price, L., & Whelley, P. (1996). The relationship between the Differential Ability Scales (DAS) and the Wechsler Intelligence Scale for Children, Third Edition (WISC-III) for students with learning disabilities. *Psychology in the Schools*, 33, 203–209.
- Elliott, C. D. (1990). *Differential ability scales*. San Antonio: The Psychological Corporation.
- Elliott, C. D. (1996). *British ability scales* (2nd ed.). Windsor: NFER-Nelson.
- Elliott, C. D. (2007). *Differential ability scales* (2nd ed.). New York: The Psychological Corporation.
- Elliott, C. D., Murray, D. J., & Pearson, L. S. (1979). *British ability scales*. Windsor: National Foundation for Educational Research.
- Harcourt Assessment. (2005). *Wechsler individual achievement test* (2nd ed.). San Antonio: Author.
- Joseph, R., Tager-Flusberg, H., & Lord, C. (2002). Cognitive profiles and social-communicative functioning in children with autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 43(6), 807–821.
- Kaufman, A. S., & Kaufman, N. L. (2004). *Kaufman Assessment Battery for Children, Second Edition (KABC-II)*. Circle Pines: American Guidance Service.
- Klin, A., Saulnier, C. A., Tsatsanis, K., & Volkmar, F. R. (2005). Clinical evaluation in autism spectrum disorders: Psychological assessment within a transdisciplinary framework. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, pp. 772–798). Hoboken: Wiley.
- McGrew, K. S. (2005). The Catell-Horn-Carroll theory of cognitive abilities: past, present, and future. In D. P. Flanagan & P. L. Harrison (Eds.), *Contemporary intellectual assessment: theories, testing, and issues*. New York: Guilford Press.
- Roid, G. H. (2003). *Stanford-Binet intelligence scales* (5th ed.). Itasca: Riverside.
- Saulnier, C. A., Quirmbach, L., & Klin, A. (2011). Clinical diagnosis of autism. In E. Hollander, A. Kolevzon, & J. T. Coyle (Eds.), *Textbook of autism spectrum disorders* (pp. 25–37). Washington, DC: American Psychiatric Publishing.
- Wechsler, D. (2002). *The Wechsler preschool and primary scale of intelligence* (3rd ed.). San Antonio: Harcourt Assessment.
- Wechsler, D. (2003). *The Wechsler intelligence scale for children* (4th ed.). San Antonio: The Psychological Corporation.
- Woodcock, R. W., McGrew, K. S., & Mather, N. (2001). *Woodcock-Johnson III tests of cognitive abilities*. Itasca: Riverside.

Differential Reinforcement

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Definition

Differential reinforcement is the process of reinforcing a specific response in a particular context and not reinforcing (i.e., extinguishing) other responses. More specifically, differential reinforcement involves providing either positive or negative reinforcement for a targeted response (or targeted member of a response class) and withholding reinforcement from all other responses (or members of a response class). The withholding of reinforcement is defined as “extinction.” Thus, differential reinforcement is a two-part process – reinforcing the desired response(s) and extinguishing all other responses. For example, a parent might reinforce with praise a young child calling out the mother’s name and ignoring (and thus not reinforcing) the child’s behavior of hitting the parent. Another example would be a teacher reinforcing (with praise and attention) a child raising her hand before being called upon to answer a question and ignoring that child if she were to shout out the answer without raising her hand. The goal of differential reinforcement is to increase the strength of the response being reinforced, while weakening the strength of the other responses not being reinforced.

Current Knowledge

A basic principle in understanding differential reinforcement and how people learn in most situations is the concept of discrimination. Basically, discrimination is a process for behaving one way in one situation or context, and behaving in a

completely different way in a different situation or context. Thus, discrimination is the ability to tell the difference between environmental events (or contexts or cues) and behaving accordingly. Discrimination typically develops as a result of differential reinforcement.

Almost all learning occurs due to the concept of discrimination and differential reinforcement. For example, consider learning the letters of the alphabet. When the letter “B” is shown and the learner asked to identify the letter, indicating “B” will be reinforced and naming any other letter will not be. This process of differentially reinforcing the learner’s responses (as correct and incorrect) results in learning of the alphabet. Consider learning to speak. When an infant says “mama” in the presence of the mother, that response will be reinforced with smiles, hugs, and positive attention. If the infant says “mama” in the presence of the father, there will be no reinforcement. Differentially reinforcing a response in one context (i.e., in presence of the mother) and not in another (i.e., in the presence of the father) results in the baby learning what to say in the presence of each parent. Consider the acquisition of social behaviors. Some young children refuse to share their toys. When this occurs, the adult rarely reinforces such selfishness. However, when a child does in fact share her toys, adults provide positive attention and reinforcement. In this case, the adult responds differently to two different behaviors – sharing and not sharing. Through this process, the child learns that sharing is preferred and hoarding toys is not. Thus, virtually all learning is accomplished through the process of learning discriminations via differential reinforcement.

The procedure of differential reinforcement has been used to both increase and decrease the strength (future rate) of specific behaviors. However, even though the goals are different (when considering increasing or decreasing future rates of behaviors), the procedure of differential reinforcement is the same. The basic procedural components of all differential reinforcement programs are these. First, the interventionist must operationally define the target behavior to be changed. That could be an appropriate behavior that must be increased in rate, a behavior deemed inappropriate

that must be decreased in rate, or both. The behavior must be operationally defined to allow for both correct recording of its occurrence (so the interventionist can objectively determine if the differential reinforcement procedure is having the desired effect) as well as for accurate implementation of the procedure (i.e., so that the interventionist(s) reinforce (or not reinforce) the correct response).

The second step in using differential reinforcement is to determine the actual reinforcement that will be made contingent upon the required response. This, by necessity, will vary across the individual due to the fact that what constitutes a motivating reinforcer is so personalized across individuals. However, most of the time, the interventionist will use some form of positive reinforcement, such as praise, smiles, good grades, tokens, or other forms of tangible reinforcement found desirable by the individual. On occasion, the interventionist might use a form of negative reinforcement, such as allowing the individual to escape a work demand contingent upon displaying the targeted response. For example, in the case where an individual tantrums in order to escape or avoid work, the caregiver might allow that person to take a break from work if the individual asks for a break instead of tantruming. Allowing the individual to briefly escape an unpleasant work demand negatively reinforces asking for a break. However, if the individual continues to tantrum and does not ask appropriately for a break, the caregiver would continue to keep the person in the demand situation by requiring work. The use of formal reinforcement preference assessments is considered best practice to determine the most motivating reward items available.

The last step in the procedure is to determine if and how reinforcement can be withheld from the individual when she/he displays a behavior other than the targeted one. In the case of using differential reinforcement to increase the strength of an appropriate behavior, the interventionist must only reinforce the targeted appropriate behavior. In the example of a child shouting out answers instead of raising a hand, the teacher will reinforce hand raising but will have to decide exactly how

to respond to the shouting out of answers. The interventionist will need to ensure that no positive reinforcement follows any behavior other than the targeted one. An important question is whether the inappropriate behavior can be ignored. In the case of shouting out an answer, it is probably the case that planned ignoring can be used effectively. However, in other situations, with other behaviors such as self-injury or aggression, planned ignoring may be difficult.

There are many variations of differential reinforcement procedures that have been used. The most common ones are differential reinforcement of alternative behaviors (DRA), differential reinforcement of incompatible behaviors (DRI), differential reinforcement of other behaviors (DRO), and differential reinforcement of low rate behaviors (DRL; see “See Also” section, below).

Differential reinforcement is one of the most widely used procedures to change behavior. The treatment of problem behaviors has evolved to the point that there is a common assumption that reinforcement-based procedures are considered to be best practice and the most ethical strategies to implement. The procedure is a natural one to most interventionists, in which desired behaviors are rewarded and all other behaviors not rewarded. The research has shown that differential reinforcement procedures can be very effective in changing behaviors, and – since they are based on the use of reinforcement (most of the time, positive, as opposed to negative) – many caregivers are comfortable with using such interventions. An advantage of differential reinforcement procedures is that caregivers have a systematic way to implement a technique that focuses on appropriate (positive) behaviors. Another advantage is that such procedures maintain a positive learning atmosphere and allow the instructional (or work activities) to continue in the context in which these procedures are used. A third advantage is that differential reinforcement can be effective without the addition of aversive or unpleasant procedures, such as punishment. Differential reinforcement is also a good procedure to implement when targeting problem behaviors due to the fact that this procedure can be used before and after the

administration of functional assessment strategies to determine the function of that behavior. In these cases, differential reinforcement can possibly establish appropriate replacement behaviors, by orienting staff to notice and reinforce desired behaviors. This is important because differential reinforcement procedures do not address the function of challenging behaviors. That is, these procedures are used in an attempt to “override” the reinforcing function of problem behaviors.

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Karsten, A. M., & Carr, J. E. (2009). The effects of differential reinforcement of unprompted responding on the skill acquisition of children with autism. *Journal of Applied Behavior Analysis*, 42, 327–334.
- Lennox, D. B., Miltenberger, R. G., Spengler, P., & Erfanian, N. (1988). Decelerative treatment practices with persons who have mental retardation: A review of five years of the literature. *American Journal on Mental Retardation*, 92, 492–501.
- Lerman, D., Kelley, M., Vorndran, C., Kuhn, S., & LaRue, R. (2002). Reinforcement magnitude and responding during treatment with differential reinforcement. *Journal of Applied Behavior Analysis*, 35, 29–48.
- Mayer, G. R., Sulzer-Azaroff, B., & Wallace, M. (2012). *Behavior analysis for lasting change* (2nd ed.). Cornwall-on-Hudson: Sloan Publishing.
- Patel, M. R., Piazza, C. C., Martinez, C. J., Volkert, V. M., & Santana, C. M. (2002). An evaluation of two differential reinforcement procedures with escape extinction to treat food refusal. *Journal of Applied Behavior Analysis*, 35, 363–374.
- Ringdahl, J., Kitsukawa, K., Andelman, M., Call, N., Winborn, L., Barretto, A., et al. (2002). Differential reinforcement with and without instructional fading. *Journal of Applied Behavior Analysis*, 35, 291–294.
- Tiger, J. H., Bouxsein, K. J., & Fisher, W. W. (2007). Treating excessively slow responding by a young man with Asperger syndrome using differential reinforcement of short response latencies. *Journal of Applied Behavior Analysis*, 40, 559–563.
- Vollmer, T. R., & Iwata, B. (1992). Differential reinforcement as treatment for behavior disorders – procedural and functional variations. *Journal of Applied Behavior Analysis*, 13, 393–417.
- Vollmer, T. R., Iwata, B., Smith, R., & Rodgers, T. (1992). Reduction of multiple aberrant behaviors and concurrent development of self-care skills with differential reinforcement. *Research in Developmental Disabilities*, 13, 287–299.

Differential Reinforcement of Low Rates of Responding (DRL)

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Definition

Differential reinforcement of low rates of responding (DRL) is a procedure in which the implementer can lower the rate of a response by reinforcing fewer incidents of that response or by reinforcing longer time intervals between incidents of the response. For example, if an individual makes profanity statements an average of 20 times per half hour, an interventionist could provide a positive reinforcer contingent upon that individual making these statements 18 or fewer times per half hour.

A related term is differential reinforcement of diminishing rates (DRD). The technical difference between DRL and DRD is that in DRD, reinforcement follows a response that has been preceded by a minimum amount of time since the last response. DRL technically refers to providing reinforcement for fewer and fewer responses exhibited by the individual. However, DRL is the most common term and often refers to both of these procedures.

Historical Background

The three general areas of concern for persons with autism are social, behavior, and language. Many persons with this diagnosis display behaviors that are deemed inappropriate, such as aggression, self-stimulation, or self-injury that, if left untreated, can greatly interfere with the individual acquiring positive adaptive skills and becoming more independent. Psychologists and educators

have long investigated the best treatment for these types of concerns. One approach that has been studied extensively has been the use of restrictive or punitive procedures. These involve either presenting a stimulus that is aversive or unpleasant to the individual following the occurrence of the unwanted behavior or removing a desirable stimulus following the display of the unwanted behavior. Although these procedures have been shown to be effective in eliminating a wide variety of unwanted behaviors, they have been associated with a number of negative side effects, as well as potential ethical problems, including misuse and abuse.

Alternatives to punishment have been pursued vigorously in the research over the past few decades. One development has been that of functional assessment procedures, which allow the practitioner to determine the reinforcement maintaining the unwanted behaviors. Research has shown that if the reinforcement maintaining an unwanted behavior can be prevented from occurring, then the unwanted behavior will reduce in strength. Similarly, if an appropriate behavior that will earn the same reinforcement (function) as the unwanted behavior can be taught, then the individual will likely shift to the appropriate replacement behavior and reduce the occurrence of the unwanted behavior.

Along with functional assessment, researchers have developed a set of procedures that emphasize the use of positive reinforcement to reduce unwanted behaviors. Among these is the DRL procedure that focuses on reinforcing less occurrences of unwanted behavior and not reinforcing higher occurrences of unwanted behavior. The findings of dozens of studies show that using reinforcement in particular ways can have the same results as punishment in stopping targeted behaviors.

Current Knowledge

Even though the DRL procedure is used to reduce rates of a problem behavior, the reinforcement is delivered after the occurrence of that behavior,

which may seem counterintuitive. This is in contrast to the differential reinforcement alternative behavior (DRA), which reinforces appropriate replacement behaviors; differential reinforcement of incompatible behavior (DRI), which provides reinforcement for appropriate replacement behaviors that are physically incompatible with the targeted unwanted behaviors; or differential reinforcement of other behavior (DRO) procedure, in which the reinforcement is delivered in the absence of the target behavior. When using DRL, reinforcement occurs following an unwanted response that remains below a certain criterion or following an unwanted response that was preceded by progressively longer intervals of time from the previous response.

It is important to point out that the goal of a DRL procedure is to simply reduce the rate of the targeted behavior but not to eliminate it entirely. Some behaviors that might be considered undesirable at higher rates may be acceptably tolerated at lower rates, without needing to reduce them to zero. For example, perhaps it is acceptable for a child to get out of their seat in school a few times a week, but unacceptable and intolerable if it were to occur several times an hour. A child with autism who spontaneously verbalizes movie scripts only a few times per week could be considered more tolerable than engaging in this behavior several times per half-hour period. Thus, the DRL procedure is typically used when considering reducing behavior that is considered acceptable at lower rates but not at higher levels.

There are several variations of the basic DRL procedure. In “full session DRL,” the implementer provides reinforcement at the end of a session or a predetermined amount of time if the number of incidents of the undesired behavior falls at or below a predetermined criterion level. For example, a teacher divides the school day into 12 30-min sessions or time periods. Each half-hour consists of one “session.” A child engages in tantrums on an average of six per half-hour period. The initial rule for delivering reinforcement in this “full session DRL” program would be that the child engages in five or fewer tantrums in a session. As the rate drops to consistently five or

fewer, a new rule would be implemented, whereby reinforcement would be made contingent upon four or fewer occurrences in the session. Over time, by gradually reducing the criterion level, the DRL program will eventually bring the rate of behavior to an acceptable level. Note also that in full session DRL, the individual has an opportunity to earn reinforcement numerous times, across the multiple sessions, since each new session signals a new opportunity.

Another type of DRL is the “interval DRL,” which is a procedure for implementing DRL in which the total session is divided into equal intervals and reinforcement is provided at the end of each interval in which the number of responses during the interval is equal to or below a criterion limit. Similar to the full session DRL, this would involve taking the full session and breaking it down into smaller intervals and reinforcement could be delivered during each of those intervals. For example, a teacher divides a 30-min lunch period into three 10-min intervals. A child is out of his or her seat on an average of 21 times during the lunch period. The rule for delivering reinforcement in this “interval DRL” would be that if the child was out of seat six or fewer times in each 10-min period, reinforcement would be provided. A potential advantage of an interval DRL program is that the individual has multiple opportunities within a session to earn reinforcement, as opposed to just one opportunity (at the end of the session).

A third variation of the basic DRL procedure is the “space-responding DRL” (sometimes called DRD). This is a procedure for implementing DRL in which reinforcement follows each occurrence of the target behavior that is separated from the previous response by a minimum inter-response time (IRT). For example, a child correctly answers questions asked by the teacher but answers so quickly that other students have no opportunity to be called on. The teacher makes a rule with this student that to be called on to answer a question, 3 min must have elapsed since the child last answered a question. Thus, the rule for reinforcement is that only responses that have been preceded by a minimum of 3 min from the previous response will receive reinforcement.

The basic procedural components of all DRL procedures are these. First, the interventionist must operationally define the targeted unwanted behavior to be changed. This must be done to allow for both correct recording of its occurrence (so the interventionist can objectively determine if the differential reinforcement procedure is having the desired weakening effect) and accurate implementation of the procedure (i.e., so that the interventionist(s) implement the DRL plan consistently).

The second step in using DRL is to determine the current “operant level” of the response. That is, the interventionist must have data showing the current rate of the behavior before implementing DRL. Depending upon the type of DRL procedure used, data might show the total number of responses during a day, the total number of responses (on average) during individual sessions, and/or the average amount of time between occurrences of the targeted undesired behavior.

The third step in using DRL procedures is to determine the actual reinforcement that will be made contingent upon the response meeting the rule for earning reinforcement. This, by necessity, will vary across individuals due to the fact that what constitutes a motivating reinforcer is personalized. However, most of the time, the interventionist will use some form of positive reinforcement, such as praise, smiles, tokens, or other forms of tangible reinforcement desired by the learner. On occasion, the interventionist might use a form of negative reinforcement, such as allowing the individual to escape a work demand contingent upon displaying the targeted response. For example, in the case where a person tantrums in order to escape or avoid work, the caregiver might allow the individual to take a break from work if she/he asks for a break instead of tantruming. In this procedure, asking for a break is negatively reinforced by allowing the individual to briefly escape an unpleasant work demand. However, if the person continues to tantrum and does not ask appropriately for a break, the caregiver would continue to keep the individual in the demand situation and constantly require work. The use of formal reinforcement preference

assessments is considered a best practice to determine the most motivating reward items available.

The last step in the procedure is to determine the actual rule for providing reinforcement. Three rules or criteria must be planned. First, the rule for what level of behavior will be required as the initial new criterion must be established. To determine this, the interventionist would set the initial criterion at or a little below the operant level. For example, if the operant level was ten occurrences per session or interval, the initial DRL criterion would be anywhere from eight to ten. The second rule that must be determined is the criterion for changing from the current criterion to a new, lower one. This criterion would specify the number of sessions that must be at the criterion level before changing to a new one. For example, the interventionist may decide that if the criterion was met, or below, for three consecutive sessions, the next lower criterion would be implemented. The final rule that must be established is the ultimate, terminal criterion at which point the interventionist would consider an acceptable level of the behavior and at which point the DRL plan would be discontinued. For example, if the operant level was ten per session, the interventionist may set two per session as the ultimate criterion to discontinue the DRL.

An excellent example of DRL that has been shown to be effective is called the "Good Behavior Game." This procedure involves dividing a group of individuals (such as students in a classroom) into two or more teams. The goal is to be the team with the fewest occurrences of undesired behaviors. Generally, the interventionist would periodically observe each team and note whether or not undesired behaviors are occurring. After a set period of time (e.g., end of the day, before lunch, etc.), the team with the fewest occurrences of the targeted undesired behavior will earn some type of positive reinforcer.

DRL is a positive procedure in that it utilizes only reinforcement to reduce undesired behaviors. It is also advantageous in that it is more easily tolerated than a behavior-reduction procedure that only provides reinforcement for the *absence* of the targeted behavior (i.e., DRO). The goal of DRO is the cessation of the unwanted behavior.

A complete elimination would generally be considered more difficult to achieve than allowing some (but lower) level of the target behavior. The individual exhibiting the target behavior may more easily tolerate being allowed some amount of unwanted behavior, than attempting to eliminate it altogether. That is why DRL is often successful; it results in a targeted behavior that is inappropriate at higher levels becoming appropriate and tolerated at lower levels. Lastly, DRL procedures have been shown in the literature to be effective procedures with a wide variety of individuals and target problems; thus, it has good generalization evidence.

There are several considerations for using DRL most effectively. Firstly, the interventionist must recognize that the DRL procedure does not produce rapid behavior change; rather, it produces slow and gradual changes. So, one must use DRL to change behaviors that are amenable to gradual change. Secondly, practitioners should not use DRL when targeting behaviors that could be physically harmful to the individual or others (such as self-injury, aggression, etc.). For those categories of behaviors, the interventionist should use procedures that have more of an immediate impact or combine DRL with such strategies. Lastly, DRL does, by its nature, focus on the undesired behavior, rather than reinforcing appropriate replacement behaviors. This suggests that the implementer combines DRL with procedures that target and reinforce appropriate replacement behaviors.

Future Directions

When developing programs for children with autism, often part of that programming focuses on attempting to reduce problem behaviors. The set of DRL programs could be useful in that regard, depending upon the characteristics of the undesired behavior. Future directions could include clarifying the specific behavioral or contextual variables that would suggest a particular DRL program be used over another type of program, such as DRA, DRI, DRO, or more restrictive techniques. In addition, rules for determining

the combination of DRL with other programs to specifically teach, model, and reinforce appropriate incompatible behaviors (to the undesired ones) would be useful for practitioners.

See Also

► [Differential Reinforcement](#)

References and Reading

- Anglesea, M. M., Hoch, H., & Taylor, B. A. (2008). Reducing rapid eating in teenagers with autism: Use of a page prompt. *Journal of Applied Behavior Analysis*, 41(1), 107–111.
- Barrish, H. H., Saunders, M., & Wolf, M. M. (1969). Good behavior game: Effects of individual contingencies for group consequences on disruptive behavior in a classroom. *Journal of Applied Behavior Analysis*, 2, 119–124.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2020). *Applied behavior analysis* (3rd ed.). Upper Saddle River: Pearson.
- Deitz, S. M., & Repp, A. C. (1973). Decreasing classroom misbehavior through the use of DRL schedules of reinforcement. *Journal of Applied Behavior Analysis*, 6, 457–463.
- Hagopian, L. P., & Kuhn, D. E. (2009). Targeting social skills deficits in an adolescent with pervasive developmental disorder. *Journal of Applied Behavior Analysis*, 42(4), 909–911.
- Lennox, D. B., Miltenberger, R. G., Spengler, P., & Erfanian, N. (1988). Decelerative treatment practices with persons who have mental retardation: A review of five years of the literature. *American Journal on Mental Retardation*, 92, 492–501.
- Mayer, G. R., Sulzer-Azaroff, B., & Wallace, M. (2019). *Behavior analysis for lasting change* (4th ed.). Cornwall-on-Hudson: Sloan Publishing.
- Rolider, A., & van Houten, R. (1990). The role of reinforcement in reducing inappropriate behavior: Some myths and misconceptions. In A. C. Repp & N. N. Singh (Eds.), *Perspectives on the use of nonaversive and aversive interventions for persons with developmental disabilities* (pp. 119–127). Sycamore: Sycamore.
- Shaw, R., & Simms, T. (2009). Reducing attention-maintained behavior through the use of positive punishment, differential reinforcement of low rates, and response marking. *Behavioral Interventions*, 24, 249–263.
- Wright, C. S., & Vollmer, T. R. (2002). Evaluation of a treatment package to reduce rapid eating. *Journal of Applied Behavior Analysis*, 35(1), 89–93.

Differential Reinforcement Procedures of Alternative Behavior (DRA/DRAIt) of Incompatible Behavior (DRI)

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Definition

Differential reinforcement of alternative behaviors (DRA) and differential reinforcement of incompatible behaviors (DRI) are both procedures designed to decrease the rate of targeted unwanted behaviors. Targeted behaviors decrease due to two mechanisms – reinforcement of appropriate behaviors that will replace the unwanted behavior and the withholding of reinforcement that historically followed the unwanted behavior. DRI is often considered a type of DRA procedure.

With both of these procedures, reinforcement is contingent upon specific behaviors that will replace the unwanted behavior. The nature of the replacement behaviors marks the difference between DRA and DRI. In DRI, the replacement behaviors are physically incompatible with the unwanted behavior. They both cannot be done at the same time. For example, if the unwanted behavior were out of seat, a physically incompatible behavior would be staying in seat. If the unwanted behavior were putting fingers in the mouth, a physically incompatible behavior would be putting hands in pants pockets. Thus, with a DRI procedure, the replacement behavior is both an appropriate one, as well as physically incompatible with the unwanted behavior. In DRA, there is no concern about the replacement behaviors being physically incompatible; it is simply an appropriate behavior that could fulfill the same function as the unwanted behavior. For example, if the unwanted behavior were screaming (to indicate a need to escape a work demand),

an interventionist might use a DRA procedure and select an appropriate replacement behavior such as pointing to a card to signal a need to break from work. Another example would be that if the unwanted behavior were a child inappropriately calling out answers in class (without waiting for the teacher to call on her), an appropriate replacement behavior would be to raise her hand to be called on. Note that in both of these examples, the appropriate replacement behavior is *not* physically incompatible with the targeted unwanted behavior; both the unwanted behavior and the replacement behavior could be displayed simultaneously. In both procedures, reinforcement is delivered for the alternative or incompatible behavior, and reinforcement is withheld (extinguished) from the targeted unwanted behavior. Both procedures result in a decrease in rate of the unwanted behavior. The strength of these procedures lies in the discrimination between the two – the alternative or incompatible behaviors are reinforced, while the unwanted behaviors are not.

Historical Background

The three general areas of concern for persons with autism are social, behavior, and language. Many persons with this diagnosis display behaviors that are deemed inappropriate, such as aggression, self-stimulation, or self-injury and that greatly interfere with the person learning positive adaptive skills and increasing their independence in life. Psychologists and educators have long investigated the best treatment for these types of concerns. One approach that has been studied extensively has been the use of restrictive or punitive procedures. These involve either presenting a stimulus that is aversive or unpleasant to the individual following the occurrence of the unwanted behavior or by removing a desirable stimulus following the display of the unwanted behavior. Although these procedures have been shown to be effective in eliminating a wide variety of unwanted behaviors, they have been associated with a number of negative side effects, as well as

potential ethical problems, including misuse and abuse.

Alternatives to punishment have been pursued vigorously in the research over the past few decades. One development has been that of functional assessment procedures, which allow the practitioner to determine the reinforcement maintaining the unwanted behaviors. Research has shown that if the reinforcement maintaining an unwanted behavior can be prevented from occurring, then the unwanted behavior will reduce in strength. Similarly, if an appropriate behavior that will earn the same reinforcement (function) as the unwanted behavior can be taught, then the individual will likely shift to the appropriate replacement behavior and reduce the occurrence of the unwanted behavior.

Along with functional assessment, researchers have developed a set of procedures that emphasize the use of positive reinforcement to reduce unwanted behaviors. Among these are the DRA and DRI procedures that focus on simultaneously reinforcing behaviors that can replace the unwanted behavior and removing the reinforcement that has maintained the targeted unwanted behavior. Findings of dozens of studies show that using reinforcement in particular ways can have the same results as punishment in stopping targeted behaviors.

Current Knowledge

Differential reinforcement procedures have been found to be some of the most frequently used procedures to reduce and eliminate unwanted behaviors, across educational, social, and vocational contexts. DRA is useful for behaviors that may occur at high or low rates, as this procedure involves teaching the individual to engage in a more appropriate behavior than the behavior targeted for reduction. Often, DRA is combined with DRI. DRI is preferable, as the student cannot engage in the targeted behavior for reduction since the reinforced response is physically incompatible with the unwanted behavior.

The procedural steps for both DRA and DRI are similar. First, the implementer must

operationally define the targeted unwanted behavior to be reduced or eliminated so that the implementer(s) will not deliver reinforcement after its occurrence and that there will be increased accuracy in data collection, to confirm (or not) if the differential reinforcement procedure is having the desired effect. With both of these procedures, the implementer must track the occurrence of both the targeted unwanted behaviors, as well as the alternative and incompatible ones.

Second, the interventionist should determine the function of the unwanted behavior. This information is helpful when deciding the procedures to use to prevent the reinforcement of the unwanted behavior (see below), as well as in guiding the selection of the appropriate replacement behaviors, which is the third step. The implementer must operationally define one or more behaviors that will be (a) desirable alternatives to the unwanted behaviors, (b) fulfill the same function as the unwanted behaviors, and (c) preferably be physically incompatible or compete with the unwanted behaviors. For example, if the unwanted behavior is swearing when frustrated, then an alternative behavior to strengthen could be having the individual write down what is frustrating. When planning on using a DRI procedure, the implementer must define an appropriate behavior that is physically incompatible with the targeted inappropriate one. In the example of an individual swearing, an incompatible behavior to reinforce could be saying, "oh, I am so frustrated, I need help!" instead of swearing. Note that expressing frustrating using that phrase is physically incompatible with swearing.

The fourth step in using DRA or DRI is to determine the actual reinforcement that will be made contingent upon the required alternative or incompatible response. The implementer will be guided by two considerations here – the function of the unwanted behavior (determined through a functional assessment) and the preferences of the individual. Reinforcement needs to be determined based upon the particular individual with whom the implementer is working, since reinforcement is so individualized. Most of the time, the implementer will use some form of positive reinforcement, such as praise, smiles, good grades, tokens,

or other forms of tangible reinforcement desired by the person. On occasion, the implementer might use a form of negative reinforcement, such as allowing the individual to escape a work demand contingent upon displaying the targeted response. These procedures are referred to as differential negative reinforcement of alternative behaviors (DNRA) and differential negative reinforcement of incompatible behaviors (DNRI). For example, in the case where a person tantrums in order to escape or avoid work, the caregiver might allow the individual to take a break from work if she/he asks for a break instead of tantruming. Thus, in this procedure, asking for a break is negatively reinforced by allowing the person to briefly escape an unpleasant work demand. However, if the individual continues to tantrum and does not ask appropriately for a break, the caregiver would keep the individual in the demand situation and continue to present work demands. The use of formal reinforcement preference assessments is considered best practice to determine the most motivating reward items available.

The last step in the procedure is to identify the extinction procedures to implement contingent upon the occurrence of the targeted unwanted behavior. The results of the functional assessment are critically important here. Once the reinforcement for the unwanted behavior has been determined, the interventionist must plan on how to prevent that reinforcement from occurring when the unwanted behavior is emitted. In the case of using DRA and DRI, the implementer will need to ensure that no reinforcement follows the unwanted behavior. For example, when an individual swears, how will the implementer react? In DRA and DRI, the implementer must ignore the swearing and not comment or react to it and focus on reinforcing the occurrence of the alternative or incompatible behavior.

There are several advantages to DRA procedures. Of particular importance is the focus on appropriate behavior. These procedures require specification of appropriate and positive behaviors to strengthen in the individual, which will contribute to the individuals' overall level of reinforcement. They learn what to do, not just what *not* to do. A second advantage of this group of

procedures is that they are associated with few or no negative side effects, unlike more restrictive procedures, such as time-out, overcorrection, and other forms of punishment. Since DRA/DRI is associated with reinforcement for appropriate responding, the individual receiving the reinforcement will likely show positive affect, demonstrate generalized responding, and develop a positive relationship with the interventionist.

A third and equally important advantage is that practitioners view these procedures quite positively, much more so than punitive or restrictive ones. Caregivers, thus, are more likely to carry out these procedures with greater willingness and fidelity. A final advantage is that DRA procedures are associated with long-term positive change. As the unwanted behavior decreases in strength, and the appropriate behaviors increase, there should be continued suppression and elimination of the unwanted behavior.

When considering the use of this group of procedures, it has been shown that the effect on the targeted replacement behavior may take some time. Reinforcement does result in behavior change, but the change may not be that rapid. To increase the speed of behavior change, it is recommended selecting powerful reinforcers. Another way to further increase the speed of further progress, one should select alternative or incompatible behaviors that already exist in the individual's repertoire. These appropriate behaviors should already be occurring at some level so that the implementer has opportunities to reinforce them when they occur. Although interventionists could teach a new skill or behavior as the replacement behavior, this simply complicates the effort involved. As with most behaviors that are targeted for increase, it would be important to select these appropriate behaviors that will likely be naturally reinforced in the individual's daily environment. It is also good practice to select these alternative and incompatible behaviors that will be less effort to emit than the targeted unwanted one. As noted earlier, of equal importance is to select replacement behaviors that are incompatible with the unwanted behavior.

In addition, there is a potential danger of a DRA procedure that focuses on a limited group

of replacement behaviors of reducing the strength of other, equally appropriate replacement behaviors. For example, consider an unwanted behavior of screaming to escape or avoid a work situation. If the interventionist selected one appropriate replacement behavior, that of pointing to a break card, the individual may learn to use that card when a break is desired but at the same time, no longer asks for a break using words or a communication device. To avoid this potential result, the interventionist should select all replacement behaviors that could serve the same function as the targeted unwanted behavior.

Lastly, the implementer must *consistently* reinforce the alternative and incompatible behaviors and *consistently* extinguish the unwanted behavior. The procedures are less effective when some instances of the alternative or incompatible behaviors are not reinforced and some instances of the unwanted behavior continue to achieve reinforcement. Extinction of the unwanted behavior seems to be important in the success of DRA/DRI. Research has shown that these procedures will be less effective if the unwanted behavior continues to result in reinforcement.

Future Directions

Differential reinforcement of alternative/incompatible behaviors should be seriously considered when planning on addressing unwanted behaviors. To use these procedures effectively, the practitioner must carefully determine the reinforcement for the unwanted behavior, plan powerful reinforcement to strengthen the appropriate behavior, and develop procedures for preventing the unwanted behavior from being rewarded. When working with individuals who display unwanted behaviors in which it may be difficult to prevent the reinforcement for those behaviors, caregivers will need to determine how to manipulate the reinforcement for the replacement behaviors in a way to promote their increase, regardless of the reinforcement for the unwanted behaviors. For example, the use of intermittent

reinforcement, increased duration of reinforcement, or a greater magnitude of reinforcement for the appropriate replacement behaviors could be considered.

See Also

- [Differential Reinforcement](#)
- [Functional Assessment](#)

References and Reading

- Athens, E. S., & Vollmer, T. R. (2010). An investigation of differential reinforcement of alternative behavior without extinction. *Journal of Applied Behavior Analysis*, 43, 569–589.
- Beare, P. L., Severson, S., & Brandt, P. (2004). The use of a positive procedure to increase engagement on-task and decrease challenging behavior. *Behavior Modification*, 28, 28–44.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Heinicke, M. R., Carr, J. E., & Mozzoni, M. P. (2009). Using differential reinforcement to decrease academic response latencies of an adolescent with acquired brain injury. *Journal of Applied Behavior Analysis*, 42, 861–865.
- Mayer, G. R., Sulzer-Azaroff, B., & Wallace, M. (2012). *Behavior analysis for lasting change* (2nd ed.). Cornwall-on-Hudson: Sloan Publishing.
- Petscher, E. S., & Bailey, J. (2008). Comparing main and collateral effects of extinction and differential reinforcement of alternative behavior. *Behavior Modification*, 32(4), 468–488.
- Petscher, E. S., Rey, C., & Bailey, J. S. (2009). A review of empirical support for differential reinforcement of alternative behavior. *Research in Developmental Disabilities*, 30, 409–425.
- Pipkin, C. S. P., Vollmer, T. R., & Sloman, K. N. (2010). Effects of treatment integrity failures during differential reinforcement of alternative behavior: A translational model. *Journal of Applied Behavior Analysis*, 43, 47–70.
- Tiger, J. H., Bousse, K. J., & Fisher, W. W. (2007). Treating excessively slow responding of a young man with Asperger syndrome using differential reinforcement of short response latencies. *Journal of Applied Behavior Analysis*, 40, 559–563.
- Vollmer, T. R., Roane, H. S., Ringdahl, J. E., & Marcus, B. A. (1999). Evaluating treatment challenges with differential reinforcement of alternative behavior. *Journal of Applied Behavior Analysis*, 32, 9–23.

Differential Reinforcement Procedures of Other Behavior (DRO)

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Definition

Differential reinforcement of other behaviors (DRO) is a reinforcement procedure in which reinforcement is delivered for any response *other than* a specific target behavior. This procedure results in a decrease in that specific target behavior because that behavior is never followed by reinforcement; thus, it weakens in future rate. For example, if a child with autism displays self-stimulatory behavior in the form of waving both hands in front of his face, a DRO procedure would be to provide a positive reinforcement for a 10-s period during which his hands were *not* waving in front of his face. Other names for this procedure include differential reinforcement of zero occurrences or omission training.

Historical Background

When considering interventions for undesirable behaviors, interventionists initially found punishment procedures to be effective. Although such procedures as overcorrection, time-out, and response cost do indeed reduce unwanted behavior, there are often negative side effects for the individual being exposed to those procedures, and historically, there have been abuses using aversive techniques. As more research was conducted on dealing with unwanted behaviors, professionals learned that using positive procedures could be an effective tool in obtaining reductions in these

behaviors. Generally speaking, differential reinforcement has been shown to both increase appropriate behaviors and reduce the strength of unwanted responses. One form of differential reinforcement that has been shown in the research to be quite effective in weakening problem behaviors is DRO. This technique has been shown to be effective across a wide variety of unwanted behaviors exhibited by a variety of individuals.

Current Knowledge

Differential reinforcement of other behaviors (DRO) is a procedure for decreasing problem behavior in which reinforcement is contingent on the absence of the problem behavior during or at specific times. DRO is perhaps the simplest of all behavior reduction procedures as it involves the simple rule of providing reinforcement whenever the specific undesirable behavior is *not* displayed. DRO differs from differential reinforcement of alternative behaviors (DRA) and differential reinforcement of incompatible behaviors (DRI) in that with those two procedures, reinforcement follows specific appropriate responses. In DRO, reinforcement is provided contingent upon passage of time in which the targeted undesired behavior does not occur. Note that reinforcement does not follow any specific response; it can follow any response as long as that response is not the targeted undesirable behavior. Because the “other” behaviors are not defined, no one behavior is reinforced so much that it is likely to increase in strength. But what does happen is that the targeted undesirable behavior is never reinforced, so over time, it reduces in rate.

There are basically two types of DRO, whole-interval and momentary-interval. The whole-interval DRO is a procedure in which reinforcement is available at the end of a fixed interval of duration if the targeted unwanted behavior did not occur at any time throughout that interval. For example, a child with autism is often out of her seat during independent work time. A whole-interval DRO procedure could involve dividing the independent work time period into six 5-min

periods. During each 5-min period, the teacher observes the child, and if the child does not get out of seat at all during a 5-min period, the teacher delivers reinforcement. However, if the child did get out of seat during a 5-min period, no reinforcement will be provided; the child will have another opportunity at the beginning of the next 5-min interval. Since the out-of-seat behavior is not reinforced by the teacher, and other behaviors are, the out-of-seat behavior should begin to diminish in rate. This procedure requires constant vigilance and observation on the part of the interventionist throughout the interval, so as to observe any occurrence of the target behavior. This DRO is appropriate for high or low rates of challenging behaviors, as the interval can be set according to the rates of challenging behaviors. Typically, one sets the interval just below the pre-intervention IRT duration of the problem behavior (see below).

The momentary DRO is a procedure whereby reinforcement is available at specific moments of time and delivered contingent on the problem behavior not occurring at that those precise moments. For example, a child with autism often whines while playing at home. A caregiver could make a rule that every 2 min (and exactly at the 2-min mark), the child will be provided a reinforcer if, *at that very moment of observation*, there is no whining being emitted. Thus, reinforcement is delivered at the moment of observation if the individual is doing anything other than whining. Note that this DRO procedure does not demand constant vigilance and attention on the part of the interventionist as does the whole-interval DRO. Using a momentary DRO allows the interventionist to be attentive to the individual only at the precise moment specified by the DRO schedule. Whether reinforcement is delivered is not dependent upon whether the targeted behavior was present or absent before or after the moment of observation; reinforcement is entirely dependent upon whether it is occurring at the precise observational moment.

There are two variations of the whole- and momentary-interval DRO procedures. The intervals can be a fixed or variable duration. Thus, a fixed-whole-interval DRO consists of the interval size being standard across all intervals. However,

a variable-whole-interval DRO consists of the interval duration varying per interval. For example, the intervals could range from 5, 10, 35, 3, and so forth but varying around a set mean. Momentary-interval DRO programs can be either fixed or variable. A fixed-momentary-interval DRO consists of the interval size being standard across all intervals; a variable-momentary-interval DRO plan allows each interval to vary around some average duration. The advantage of the variable DRO is that individuals cannot predict when the interval will end and reinforcement is available. All DRO procedures target the reduction of targeted inappropriate behavior. The research on which DRO procedure to use shows mixed results; both types of DRO plans can be effective in reducing the targeted undesired behavior.

The basic procedural components of all DRO procedures are these. First, the interventionist must operationally define the target behavior to be changed. That requires carefully specifying the targeted unwanted behavior to allow for both correct recording of its occurrence (so the interventionist can objectively determine if the differential reinforcement procedure is having the desired weakening effect) and accurate implementation of the procedure (i.e., so that the interventionist (s) know exactly when reinforcement should or should not be provided). The second step in using DRO is to determine the actual reinforcement that will be made contingent upon the absence of the unwanted response and how it will be delivered. This, by necessity, will vary across individuals due to the fact that what constitutes a motivating reinforcer is so personalized. However, most of the time, the interventionist will use some form of positive reinforcement, such as praise, smiles, tokens, or other forms of tangible reinforcement desired by the learner. On occasion, the interventionist might use a form of negative reinforcement, (termed differential negative reinforcement of other behavior, or DNRO) such as allowing the learner to escape a work demand contingent upon displaying the targeted response. For example, in the case where an individual tantrums in order to escape or avoid work, the caregiver might allow the person to take a break from work if she/he asks

for a break instead of tantruming. In this procedure, asking for a break is negatively reinforced by allowing the person to briefly escape an unpleasant work demand. However, if the individual continues to tantrum and does not ask appropriately for a break, the caregiver would continue to keep the person in the demand situation and constantly require work. The use of formal reinforcement preference assessments is considered best practice to determine the most motivating reward items available.

The third step in implementing a DRO procedure is to determine which type of DRO will be used, interval or momentary, and the criteria for establishing the initial interval size and increasing the interval size as the behavior begins to weaken. Once the type of DRO program is decided, the interventionist must determine the interval size to use to begin the procedure. Research has shown the most success is seen when the initial interval size is set small and gradually lengthened over time, as the targeted behavior reduces in rate. This should be based upon pre-intervention levels of the problem behavior and the average inter-response time (IRT) duration historically observed. The formula for calculating IRT is to divide the total number of responses observed during a certain time interval by the total amount of time of that interval. For example, if during pre-intervention conditions, the individual exhibits the target behavior, on average, ten times every hour, the mean interval between occurrences is 6 min (ten occurrences of the behavior divided by 60 min). That information can then be used to establish the initial interval size for the DRO procedure. Next, the interventionist must develop a criterion for increasing the interval duration as the DRO program demonstrates success. For example, if the practitioner begins with an interval size of 6 min and over 90% of the intervals shows no targeted problem behavior over 3 consecutive days, then the interval size could be increased to 7 min. Such a mastery criterion if developed, in advance, will result in both increased progress in decreasing the problem behavior and a procedure gradually easier to implement.

The last step in the procedure is to determine exactly how to respond to the display of the

targeted undesired behavior. The rule in DRO is to not provide any reinforcement (regardless of function) for its occurrence. So, the interventionist must be careful not to react in any way that could possibly provide any source of reinforcement for its occurrence. An important question is whether the inappropriate behavior can be ignored or if it is such a serious behavior that some sort of intervention must apply. In the case of shouting out an answer, it is probably the case that ignoring it can be done effectively. However, in other situations, with other behaviors such as self-injury or aggression, not reacting may be difficult, due to potential safety issues. In those cases, DRO may not be the method of choice.

There are several advantages to DRO such as the procedure is positive, is easy to implement, and focuses solely on the use of reinforcement to decrease undesired behaviors. Reinforcers are not removed from the individual, and few to no negative side effects are reported. Interventionists appreciate and are more willing to use positive procedures as opposed to more aversive or unpleasant interventions. Since these procedures are generally effective and positive, they are more ethically appropriate as a treatment choice. DRO is easy for teachers to use in most classrooms and school settings and has been shown to work across a wide variety of populations and contexts. The effect of such procedures is more rapid than simply extinguishing the targeted undesired behavior; although extinction can work, the application of DRO produces quicker change. Additionally, the effects of DRO have been shown to be long-lasting, producing durable response suppression. A particular advantage of a momentary DRO is that it does not require such continuous attention, and for a busy teacher or parent, that can be a useful feature. With this procedure, at the moment of observation, the interventionist can interrupt what she/he is doing, observe whether or not the targeted undesirable behavior is occurring, and deliver (or not deliver) the reinforcement based upon that immediate observation.

However, there are several potential disadvantages to DRO procedures. One is that such

procedures are not designed to teach and/or increase any particular appropriate behavior. Its inherent characteristic is to focus on the *absence* of the targeted behavior, and there is no attempt to operationally define and strengthen an appropriate replacement behavior. Another potential limitation of this procedure is that it focuses the attention of the interventionist on the negative or undesired behavior. Since its occurrence triggers whether or not reinforcement is delivered, the interventionist is paying attention primarily to whether or not the problem behavior occurs. This may result in the individual inadvertently getting attention for the problem behavior. Thus, caregivers need to be aware of any potential reaction being given to the individual following the occurrence of the targeted unwanted behavior. Another potential disadvantage of the DRO procedures is that since reinforcement is delivered for any response *other than* the targeted undesired behavior, there is a risk that other behaviors equally undesirable may inadvertently be reinforced and thus strengthened. For example, consider a DRO procedure used to reduce the self-stimulatory behavior of jumping up and down repeatedly. With DRO, reinforcement is given whenever jumping is not occurring. However, if the individual is not jumping but instead waving fingers in front of the face, reinforcement would be allowed (since the rule is to provide reinforcement for any response other than jumping). This potential disadvantage is possible when working with an individual who displays a large number of undesired behaviors. If this potential exists, a recommendation would be to provide the reinforcement only when none of the undesired behaviors are occurring or to use a procedure other than DRO (such as DRA or DRI).

Future Directions

DRO procedures are effective, show long-lasting results, are relatively easy to implement, and are preferred by interventionists due to their positive nature. Further clarification of the behavioral

characteristics of when to use which type of DRO would enhance its use and effectiveness. Guidelines for establishing initial interval size and criterion for increasing the interval duration would be helpful as well.

See Also

► [Differential Reinforcement](#)

References and Reading

- Conyers, C., Miltenberger, R., Romaniuk, C., Kopp, B., & Himle, M. (2003). Evaluation of DRO schedules to reduce disruptive behavior in a preschool classroom. *Child and Family Behavior Therapy*, 25(3), 1–6.
- Conyers, C., Miltenberger, R., Maki, A., Barenz, R., Jurgens, M., Sailer, A., et al. (2004). A comparison of response cost and differential reinforcement of other behavior to reduce disruptive behavior in a preschool classroom. *Journal of Applied Behavior Analysis*, 37, 411–415.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2020). *Applied behavior analysis* (3rd ed.). Upper Saddle River: Pearson.
- Cowdery, G., Iwata, B., & Pace, G. (1990). Effects and side effects of DRO as treatment for self-injurious behavior. *Journal of Applied Behavior Analysis*, 23(4), 497–506.
- Daddario, R., Anhalt, K., & Barton, L. (2007). Differential reinforcement of other behavior applied classwide in a child care setting. *International Journal of Behavioral Consultation and Therapy*, 3(3), 342–348.
- Hegel, M. T., & Ferguson, R. J. (2000). Differential reinforcement of other behavior (DRO) to reduce aggressive behavior following traumatic brain injury. *Behavior Modification*, 24(1), 94–101.
- Homer, A. L., & Peterson, L. (1980). Differential reinforcement of other behavior: A preferred response elimination procedure. *Behavior Therapy*, 11, 449–471.
- Kodak, T., Miltenberger, R. G., & Romaniuk, C. (2003). The effects of differential negative reinforcement of other behavior and noncontingent escape on compliance. *Journal of Applied Behavior Analysis*, 36, 379–382.
- Mayer, G. R., Sulzer-Azaroff, B., & Wallace, M. (2019). *Behavior analysis for lasting change* (4th ed.). Cornwall-on-Hudson: Sloan Publishing.
- Miltenberger, R. G. (2016). *Behavior modification: Principles and procedures* (6th ed.). Boston: Cengage Learning.

Diffusion Tensor Magnetic Resonance Imaging

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Definition

Diffusion tensor imaging (DTI) is a magnetic resonance imaging (MRI) modality used in brain imaging which measures characteristics of water diffusion in vivo to make inferences on the underlying neuroanatomy, such as the structural integrity of white matter. White matter structures probed include major neuronal fiber tracts such as association (e.g., superior longitudinal fasciculus), commissure (e.g., corpus callosum), and projection (e.g., corticospinal tract) fibers. Water diffusion can be characterized at each anatomical location by the diffusion tensor, a second-order model which provides the direction and the degree of anisotropy (i.e., directionality). The diffusion tensor can be visualized as an ellipsoid and generally aligns with the underlying white matter fibers. Diffusion properties in tissue can then be captured using various numeric metrics computed from the tensor and commonly include fractional anisotropy (FA), radial diffusivity (RD), axial diffusivity (AD), mean diffusivity (MD), and apparent diffusion coefficient (ADC), with FA being the most widely utilized in neuropsychiatric research.

There are some general relationships between the aforementioned metrics and biological features of tissue. Thus, knowledge of these inferences can guide the interpretation of research findings using DTI. Each of these measures aims to characterize the restriction of water diffusion due to physical barriers such as membranes and myelin; therefore, they are used as surrogates for white matter structural integrity in DTI studies.

RD is a measure of the inhibited water diffusion occurring across or perpendicular to nerve fibers. Causes of increased restriction perpendicular to the fiber (a low RD) include thicker myelin, a more water-impermeable myelin, denser packing of fibers, and/or smaller fiber diameters. Less restriction (a high RD) could be due to delayed myelination, loss of myelin, more water-permeable myelin, loss of axonal membrane integrity, looser fiber packing, disorganized fiber packing, and/or larger-diameter fibers. On the other hand, AD is a measure of water diffusion occurring along or parallel to nerve fibers. A higher AD value indicates less hindrance to water movement along axons and could be due to axonal loss and/or less-dense fiber packing. MD and ADC measure average water diffusion in all directions. Finally, FA ranges from zero to one and describes the degree to which water diffusion is directionally dependent. A value of zero means that water diffusion is isotropic; it is equally restricted in all directions such that the pattern of diffusion resembles a sphere. A value of one means that water diffusion is completely restricted to a single direction. FA is most frequently used to characterize white matter integrity. Regardless of the specific measure, however, each of these parameters provides different information about the underlying white matter architecture. Because of this, considering multiple measures has become a common approach in DTI studies.

Moreover, DTI can also be used to reconstruct the 3D structure of white matter fiber tracts using a technique called tractography or “fiber tracking.” Algorithms are used to determine 3D curves which trace fibers by following the orientation of maximum water diffusion. These fibers can then be visualized resulting in spectacular images of multiple fiber pathways. Tractography is now being used regularly in the study of neuropsychiatric disorders such as autism. Previously, such white matter anatomy could only be studied by postmortem dissection or invasive tracing studies in nonhuman animals. Because of this, tractography has been referred to as “virtual dissection.”

DTI has revolutionized the study of structural brain connectivity in humans and is extensively

used in the field of autism research to study alterations in neural connectivity. In the past 5 years, there has been a surge in the number of DTI studies published in autism research with the overall consensus being that some level of impairment exists in structural brain connectivity likely in the direction of underconnectivity. The focus of this chapter is the application of DTI in the study of the neurobiology of autism spectrum disorders (ASD). While a brief description of the technical aspects of DTI has been provided, the reader is referred to other reading which covers the technical details of DTI in much greater detail (Mori 2007; Mori and Zhang 2006).

Historical Background

Interestingly, diffusion MRI had been known for many decades as a source of obtaining tissue contrast. Early work in the 1950s by Hahn (1950), Carr and Purcell (1954), and Torrey (1956) laid the groundwork for diffusion measurements from magnetic resonance, providing an understanding of the change in the magnetic resonance signal in the presence of water diffusion. Stejskal and Tanner (1965) advanced this formulation and incorporated the diffusion tensor. Finally, Basser and colleagues (1994) developed the acquisition strategy that allowed computation of the diffusion tensor. Using multiple acquisitions, each sensitive to diffusion in a specified direction, the diffusion tensor can be reconstructed at each location in the brain image.

Current Knowledge

At the time of this writing, there are over 30 studies using diffusion imaging, investigating the neurobiology of ASD since the first study was published in 2004 (Barnea-Goraly et al. 2004). In recent years, the number of studies has increased sharply: one study in 2004, five studies in 2007, eight studies in 2009, and 13 studies in 2010 (all reviewed below). The methods implemented in these studies are diverse, ranging from voxel-wise comparisons to tractography-based studies. Some studies use a combination

of methods or other MRI modalities such as structural and/or functional MRI. The DTI studies reviewed in this chapter are presented according to their methodology which will include voxel-wise, region of interest (ROI), tractography, combination DTI, and multimodal MRI studies. White matter properties vary with age through development. Thus, in order to better appreciate the developmental aspects of ASD, these DTI studies are further subdivided into child (age <13 years), adolescent (13–20 years), and adult (age $\geq$ 21 years) categories based mainly on the average age of ASD participants.

Voxel-Wise

The first DTI study published in the autism research literature was a voxel-wise study. Thus, it comes as no surprise that voxel-wise studies are the most common of the DTI studies in ASD. In general, image volumes are warped to a common space, and then, groups are compared on a voxel-by-voxel basis within the white matter. A variety of statistical procedures are used to identify significant differences and control for false positives which can result from the large numbers of comparisons made (each brain contains thousands of voxels). At the time of this writing, there are 10 DTI studies which utilize a voxel-wise approach in the study of ASD. Overall, these studies demonstrate diffuse abnormalities in white matter using the previously mentioned metrics, though the most commonly reported abnormality is a reduction in FA.

There are four studies which implement a voxel-wise analysis studying children with ASD. Cheung and colleagues (2009) reported on a comparison of 13 children with autism (9.3 ± 2.6 years) and 14 controls (9.9 ± 2.5 years) where FA in the autism group was significantly lower than controls in bilateral prefrontal and temporal regions, particularly in the right ventral temporal lobe adjacent to the fusiform gyrus. Additionally, FA was greater in the right inferior frontal gyrus and left occipital lobe. Barnea-Goraly and colleagues (2010) reported on a comparison of 13 children with autism (10.5 ± 2.0 years), 13 of their unaffected siblings (8.9 ± 1.9 years), and 11 controls (9.6 ± 2.1 years). Both the autism and unaffected sibling groups had widespread

FA reductions in the frontal, parietal, and temporal lobes, including regions known to be important for social cognition. Within regions of reduced FA, reductions in AD with preserved RD were observed. There were no differences in white matter structure between autism and unaffected sibling groups. Sahyoun and colleagues (2010a) reported on a comparison of nine children with autism (12.8 ± 1.5 years) and 12 controls (13.3 ± 2.45 years). Controls showed increased FA within frontal white matter and the superior longitudinal fasciculus. The autism group showed increased FA within peripheral white matter, including the ventral temporal lobe. Shukla and colleagues (2011) reported on a comparison of 26 children with ASD (12.8 ± 0.6 years) and 24 controls (13.0 ± 0.6 years). The ASD group demonstrated decreased FA and increased MD and RD in numerous white matter structures: corpus callosum, anterior and posterior limbs of the internal capsule, inferior longitudinal fasciculus, inferior fronto-occipital fasciculus, superior longitudinal fasciculus, cingulum, anterior thalamic radiation, and corticospinal tract. There were no areas of increased FA, reduced MD, or RD in the ASD group.

There are four studies which implement a voxel-wise analysis studying adolescents with ASD. In the first published DTI study in ASD, Barnea-Goraly and colleagues (2004) reported on a comparison of seven adolescents with autism (14.6 ± 3.4 years) and nine controls (13.4 ± 2.8 years). The autism group demonstrated reduced FA in white matter adjacent to the ventromedial prefrontal cortices, anterior cingulate gyri, and temporoparietal junctions. FA reductions were also seen adjacent to the superior temporal sulcus bilaterally, temporal lobes approaching the amygdala bilaterally, occipitotemporal tracts, and corpus callosum. Cheng and colleagues (2010) compared 25 adolescents with ASD (13.71 ± 2.54 years) and 25 controls (13.51 ± 2.20 years), reporting reduced FA in the right posterior limb of internal capsule with increased RD distally and reduced AD centrally. ASD adolescents also demonstrated greater FA with reduced RD in the frontal lobe, greater FA with reduced RD in the right cingulate gyrus, greater FA with reduced RA with increased AD

in the bilateral insula, greater FA with reduced RD in the right superior temporal gyrus, and greater FA with reduced RD in the bilateral middle cerebellar peduncle. Noriuchi and colleagues (2010) reported on a comparison of seven adolescents with ASD (13.96 ± 2.68 years) and seven controls (13.36 ± 2.74 years). For the ASD group, FA and AD were lower in the white matter around left dorsolateral prefrontal cortex, posterior superior temporal sulcus/temporoparietal junction, right temporal pole, amygdala, superior longitudinal fasciculus, occipitofrontal fasciculus, mid- and left anterior corpus callosum, and mid- and right anterior cingulate cortex. Higher AD values were observed in the cerebellar vermis lobules in the ASD group. Groen and colleagues (2011) reported on a comparison of 17 adolescents with autism (14.4 ± 1.6 years) and 25 controls (15.5 ± 1.8 years). Participants with autism had lower FA in the left and right superior and inferior longitudinal fasciculi which lost significance after controlling for age and IQ. MD levels were markedly increased in the autism group throughout the brain.

In the two remaining voxel-wise studies, one examined adults only, and the other included subjects from the entire age range from children to adults. Bloemen and colleagues (2010) reported on a comparison of 13 adults with Asperger syndrome (39.0 ± 9.8 years) and 13 controls (37.0 ± 9.6 years). Adults with Asperger syndrome had lower FA than controls in 13 clusters which were largely bilateral and included white matter in the internal capsule; frontal, temporal, parietal, and occipital lobes; cingulum; and corpus callosum. Keller and colleagues (2007) reported on a comparison of 34 children, adolescents, and adults with ASD (18.9 ± 7.3 years) and 31 controls (18.9 ± 6.2 years). Participants with ASD had lower FA in areas within and near the corpus callosum and in the right retrolenticular portion of the internal capsule.

Region of Interest (ROI)

In using the ROI method, anatomical area(s) which are to be studied are traced for each individual participant, usually by hand and without knowledge of group membership, in order to

obtain averaged measures (e.g., FA, RD) within the ROI that characterize the selected region for a particular participant. Comparisons can then be made testing for significant group differences. ROI studies are particularly useful when particular brain structures, which can be readily defined, are suspected to be abnormal. By focusing on hypothesized regions, the problem of multiple comparisons is greatly reduced. At the time of this writing, there are seven DTI studies which use an ROI approach in the study of ASD. Overall, these studies demonstrate various diffusion abnormalities in most areas studied with the most common abnormality being a reduction in FA.

There are four studies which implement an ROI approach studying children with ASD. Ben Bashat and colleagues (2007) reported on a comparison of seven toddlers with autism with ages ranging from 1.8 to 3.3 years. ROI measurements in different anatomical regions revealed an increase in FA with dominance in the left hemisphere and frontal lobe. Sivaswamy and colleagues (2010) reported on a comparison of 27 children with ASD (mean age 5.0 years) and 16 controls (mean age 5.9 years) where ROIs were placed in the cerebellar peduncles. In the ASD group, there was an increase in the MD of bilateral superior cerebellar peduncles and reversal of asymmetry in FA of the middle cerebellar peduncle and inferior cerebellar peduncle. Brito and colleagues (2009) compared eight children with ASD (9.53 ± 1.83 years) and eight controls (9.57 ± 1.36 years). In the ASD group, they reported reduced FA in ROIs corresponding to the anterior corpus callosum, right corticospinal tract, posterior limb of right and left internal capsules, left superior cerebellar peduncle, and right and left middle cerebellar peduncles. Shukla and colleagues (2010) reported on a comparison of 26 children with ASD (12.7 ± 0.6 years) and 24 controls (13.0 ± 0.6 years). ASD children demonstrated reduced FA and increased RD for whole-brain white matter and ROIs corresponding to the corpus callosum and internal capsule. Additionally, there was increased MD for whole-brain white matter and ROIs corresponding to the anterior and posterior limbs

of the internal capsule. Finally, reduced AD was reported for the ROI of the body of the corpus callosum, and reduced FA was also found for the ROI of the middle cerebellar peduncle.

In the three remaining studies, analyses included subjects across the entire age range including children, adolescents, and adults. Lee and colleagues (2007) reported on a comparison of 43 individuals with ASD (16.2 ± 6.7 years) and 34 controls (16.4 ± 6.0 years) with ROIs capturing the superior temporal gyrus and temporal stem. In all examined regions, the ASD group demonstrated decreased FA and increased MD and RD. Lange and colleagues (2010) reported on a comparison of 30 individuals with autism (15.78 ± 5.6 years) and 30 controls (15.79 ± 5.5 years) with ROIs including superior temporal gyrus and temporal stem. Tensor skew, a measure of tensor shape, was used in addition to the more common metrics. In the superior temporal gyrus, reversed hemispheric asymmetry was reported for the autism group: tensor skew was greater on the right, and FA was decreased on the left. Moreover, there was also increased AD bilaterally. In the right temporal stem (but not the left), increases in MD, AD, and RD were exhibited in the autism group. Alexander and colleagues (2007) reported on a comparison of 43 individuals with ASD (16.23 ± 6.70 years) and 34 controls (16.44 ± 5.97 years) using a corpus callosum ROI. There were significant group differences in white matter volume, FA, MD, and RD which appeared to be driven by an autism subgroup with small corpus callosum volumes, high MD, low FA, and increased RD. Compared to other individuals with autism or the controls, this subgroup had lower performance IQ measures.

Tractography

Tractography studies have similarities to ROI studies, except the area of interest is defined using tractography. The results of tractography are very sensitive to the method and parameters used in creating these tract volumes; thus, great care must be taken to ensure reliability and blindness. In a manner analogous to ROI studies, diffusion metrics captured within the tract volume are analyzed. In addition, geometric properties of

the tracts can also be obtained (e.g., lengths, volumes). Comparisons can be made by averaging these measures and comparing means between groups. At the time of this writing, there are six DTI studies which utilize a tractography approach in the study of the neurobiology of ASD. Overall, studies using tractography demonstrate diffusion abnormalities in many fiber tracts, again with the most common abnormality being a reduction in FA.

There are two studies which implement the tractography approach studying children and adolescents with ASD. Sundaram and colleagues (2008) reported on a comparison of 50 children with ASD (4.79 ± 2.43 years) and 16 controls (6.84 ± 3.45 years). Tractography was performed on frontal lobe long- and short-range pathways. The ADC was significantly higher for whole frontal lobe, long- and short-range association fibers in the ASD group. FA was significantly lower in the ASD group for short-range fibers but not for long-range fibers. There was no between-group difference in the number of frontal lobe fibers (short and long); however, the long-range association fibers of frontal lobe were significantly longer in ASD group. Fletcher and colleagues (2010) reported on a comparison of 10 adolescents with autism (14.25 ± 1.92 years) and 10 controls (13.36 ± 1.34 years), performing tractography of the arcuate fasciculus (superior longitudinal fasciculus). The results showed an increase in MD in the autism group, due mostly to an increase in the RD. Both MD and FA were less lateralized in the autism group.

The remaining four tractography studies include adults with one study including participants across the entire age range. Catani and colleagues (2008) reported on a comparison of 15 adults with Asperger syndrome (31 ± 9 years) and 16 controls (35 ± 11 years). Tractography was performed on short intracerebellar connections, long-range afferent (i.e., corticopontocerebellar and spinocerebellar tracts) and efferent (i.e., superior cerebellar tracts) connections. The Asperger group had significantly lower FA in the short intracerebellar fibers and right superior cerebellar peduncles, but no difference in the afferent tracts. Conturo and

colleagues (2008) reported on a comparison of 17 adults with autism (26.46 ± 2.73 years) and 17 controls (26.08 ± 2.69 years), performing tractography of hippocampo-fusiform and amygdalo-fusiform pathways. While these pathways had normal size and shape, the right hippocampo-fusiform had reduced RD compared with controls, opposite to the whole-brain effect of increased RD. In contrast, left hippocampo-fusiform, right arcuate fasciculus, and left arcuate fasciculus had increased RD and increased AD in autism. There was a general loss of lateralization compared with controls. Thomas and colleagues (2011) reported on a comparison of 12 adults with autism (28.5 ± 9.7 years) and 18 controls (22.4 ± 4.1 years), performing tractography on callosal and visual-association pathways. Compared with the control group, the autism group demonstrated an increase in the volume of the intra-hemispheric fibers, particularly in the left hemisphere, and a reduction in the volume of the forceps minor and the body of the corpus callosum. Finally, Pugliese and colleagues (2009) compared 24 children, adolescents, and adults with Asperger syndrome (23.3 ± 12.4 years) and 42 controls (25.3 ± 10.3 years), performing tractography on the following limbic pathways: inferior longitudinal fasciculus, inferior frontal occipital fasciculus, uncinate, cingulum, and fornix. There were no significant between-group differences in FA and MD. However, the Asperger group had a significantly higher number of streamlines in the right and left cingulum and in the right and left inferior longitudinal fasciculus. In contrast, the group with Asperger syndrome had a significantly lower number of streamlines in the right uncinate.

Combination DTI

While each of the DTI methods described above has limitations when used alone, these can be overcome by using the methods in combination with one another, ideally in a synergistic manner. Kumar and colleagues (2010) reported on a comparison of 32 children with ASD (mean age 5.0 years), 12 developmentally impaired children without ASD (mean age 4.6 years), and 16 controls (mean age 5.5 years). They essentially

performed two separate analyses on the same group of participants: voxel-wise and tractography study. In the voxel-wise portion of the study, when the ASD and developmentally impaired children were compared with controls, FA was lower in the right uncinate fasciculus, right cingulum, and corpus callosum in both affected groups. There was also reduced FA in right arcuate fasciculus when ASD children were compared with controls and reduced FA in the bilateral inferior fronto-occipital fasciculus when developmentally impaired children were compared with controls. ADC was increased in right arcuate fasciculus in both ASD and developmentally impaired children. In the tractography portion of the study, the ASD group showed shorter length of the left uncinate fasciculus and increased length, volume, and density of the right uncinate fasciculus; increased length and density of the corpus callosum; and higher density of the left cingulum compared with the control group. Compared with the developmentally impaired group, the ASD group had increased length, volume, and density of the right uncinate fasciculus; higher volume of the left uncinate fasciculus; and increased length of the right arcuate fasciculus and corpus callosum. Jou and colleagues (2011) reported on a comparison of 10 ASD adolescents (13.06 ± 3.85 years) and 10 controls (13.94 ± 4.23 years). DTI data was analyzed in a synergistic manner by performing a voxel-wise comparison with follow-up tractography to identify underlying affected white matter structures. The regions of lower FA, as confirmed by tractography, involved the inferior longitudinal fasciculus/inferior fronto-occipital fasciculus, superior longitudinal fasciculus, and corpus callosum/cingulum. Notably, some volumes of interest were adjacent to the fusiform face area, bilaterally, corresponding to involvement of the inferior longitudinal fasciculus. The largest effect sizes were noted for volumes of interest in the right anterior radiation of the corpus callosum/cingulum and the right fusiform face area (inferior longitudinal fasciculus). Finally, Pardini and colleagues (2009) reported on a comparison of 10 adults with autism (19.7 ± 2.83 years) and 10 controls (19.9 ± 2.64 years). They compared

FA within orbitofrontal cortex volumes defined by tractography in addition to voxel-wise comparison of FA. The low-functioning group with autism demonstrated reduced tract volume and lower mean FA values in the left orbitofrontal cortex network compared with controls.

Multimodal MRI

While an extremely powerful technology, DTI remains an indirect probe of white matter integrity based on measuring properties of restricted water diffusion. One strategy to augment this data is to use multiple modalities in search for converging evidence supporting a particular neurobiological hypothesis. At the time of this writing, there are a total of five published studies taking a multimodal MRI approach: two combining with structural MRI, two combining with functional MRI, and one combining with both structural and functional MRI.

Ke and colleagues (2009) reported on a comparison of 12 children with autism (8.75 ± 2.26 years) and 10 controls (9.40 ± 2.07 years) using voxel-wise comparison of both white matter density (structural MRI) and FA (DTI). In the autism group, there was a decrease of the white matter density in the right frontal lobe, left parietal lobe, and right anterior cingulate. Moreover, there was an increase of the white matter density in the right frontal lobe, left parietal lobe, and left cingulate gyrus. The autism group also exhibited reductions of FA in the frontal lobe and left temporal lobe. Mengotti and colleagues (2011) reported on a comparison of 20 children with autism (7.00 ± 2.75 years) and 22 controls (7.68 ± 2.03 years) using a combination of voxel-wise comparison in gray/white matter and ROIs (corpus callosum, frontal, temporal, parietal, and occipital lobes) comparing ADC. Compared to controls, the autism group exhibited increased white matter volumes in the right inferior frontal gyrus, right fusiform gyrus, left precentral and supplementary motor areas, and left hippocampus. Moreover, there were increased gray matter volumes in the inferior temporal gyri bilaterally, right inferior parietal cortex, right superior occipital lobe, and left superior parietal lobule. Additionally, there were decreased gray matter volumes in the right

inferior frontal gyrus and left supplementary motor area. Finally, the autism group exhibited abnormally increased ADC in the bilateral frontal cortex and left genu of the corpus callosum.

Using a combination of DTI and functional MRI, Sahyoun and colleagues (2010b) reported on a comparison of 12 adolescents with autism (13.3 ± 2.1 years) and 12 controls (13.3 ± 2.5 years). DTI analysis included a tractography approach in which fiber tracking was aided by functional MRI. FA was captured within these tracts, and mean FA was compared between groups. The functional MRI included response time on pictorial problem-solving task. Autism and control groups showed similar networks: linguistic processing activated inferior frontal, superior and middle temporal, ventral visual, and temporoparietal areas, whereas visuospatial processing activated occipital and inferior parietal areas. However, the autism group activated occipitoparietal and ventral temporal areas, whereas controls activated frontal and temporal language regions. The autism group relied more heavily on visuospatial abilities as evidenced by intact connections between the inferior parietal and ventral temporal ROIs. There was impaired activation of frontal language areas in the autism group as evidenced by reduced connectivity of the inferior frontal region to the ventral temporal/middle temporal regions.

In another combination DTI and functional MRI study, Thakkar and colleagues (2008) reported on a comparison of 12 ASD adults (30 ± 11 years) and 14 controls (27 ± 8 years). DTI analysis included a comparison of FA performed 2 mm below the white/gray matter boundary. Functional MRI included a saccadic paradigm where activation was compared in error versus correct antisaccades, and in both correct and error antisaccades versus fixation, both within and between groups using a random effects model. Relative to controls, the ASD group made more antisaccade errors and responded more quickly on correct trials. The ASD group also showed reduced discrimination between error and correct responses in rostral anterior cingulate cortex and reduced FA in white matter underlying anterior cingulate cortex. Finally, in the ASD

group, there was increased activation on correct trials and reduced FA in rostral anterior cingulate, both of which were related to repetitive behavior.

Using a combination of DTI and structural and functional MRI, Knaus and colleagues (2010) reported on a comparison of 14 ASD adolescents (age range 11–19 years) and 20 controls (age range 11–19 years). Structural MRI analysis included volumetric measurements of language areas. DTI analysis included tractography to delineate a pathway between temporal and frontal language areas to compare mean FA. Functional MRI was used to divide participants into typical (leftward) and atypical (rightward) language laterality groups. Participants with typical left-lateralized language activation had smaller frontal language region volume and higher FA of the arcuate fasciculus compared to the group with atypical language laterality, across both ASD and controls. The group with typical language asymmetry included the most right-handed controls and fewest left-handers with ASD. Atypical language laterality was more prevalent in the ASD than in controls.

Future Directions

Future directions include further refinement of DTI techniques, sophistication in the integration of multiple imaging modalities, and multi-dimensional longitudinal designs. Improvements in technology include higher scan resolution, improving signal-to-noise ratio while maintaining tolerability, and developing novel metrics with higher pathological specificity. Other improvements go beyond the tensor model to examine the directional variation of diffusion in more detail (Lo et al. 2011). Tractography faces challenges in its ability to resolve multiple fiber populations in a single voxel (e.g., crossing and kissing fibers), growing usage as a more quantitative measure, and lack of standardized technique supported by gold-standard postmortem studies. While several multimodal studies have been published, there could be tighter integration of more modalities (MRI and beyond) to create novel study designs with higher synergy. The studies reviewed in this chapter are all cross-sectional; thus, longitudinal

studies would be optimal to fill in the gaps in current knowledge. In addition to longitudinal imaging across the life span, there should be longitudinal clinical assessments designed to give further meaning to imaging data.

See Also

- [Functional Connectivity](#)
- [Magnetic Resonance Imaging](#)

References and Reading

- Alexander, A. L., Lee, J. E., Lazar, M., Boudos, R., Dubray, M. B., Oakes, T. R., et al. (2007). Diffusion tensor imaging of the corpus callosum in Autism. *NeuroImage*, 34(1), 61–73.
- Barnea-Goraly, N., Kwon, H., Menon, V., Eliez, S., Lotspeich, L., & Reiss, A. L. (2004). White matter structure in autism: Preliminary evidence from diffusion tensor imaging. *Biological Psychiatry*, 55(3), 323–326.
- Barnea-Goraly, N., Lotspeich, L. J., & Reiss, A. L. (2010). Similar white matter aberrations in children with autism and their unaffected siblings: A diffusion tensor imaging study using tract-based spatial statistics. *Archives of General Psychiatry*, 67(10), 1052–1060.
- Basser, P. J., Mattiello, J., & LeBihan, D. (1994). MR diffusion tensor spectroscopy and imaging. *Biophysical Journal*, 66(1), 259–267.
- Ben Bashat, D., Kronfeld-Duenias, V., Zachor, D. A., Ekstein, P. M., Hendler, T., Tarrasch, R., et al. (2007). Accelerated maturation of white matter in young children with autism: A high b value DWI study. *NeuroImage*, 37(1), 40–47.
- Bloemen, O. J., Deeley, Q., Sundram, F., Daly, E. M., Barker, G. J., Jones, D. K., et al. (2010). White matter integrity in Asperger syndrome: A preliminary diffusion tensor magnetic resonance imaging study in adults. *Autism Research*, 3(5), 203–213.
- Brito, A. R., Vasconcelos, M. M., Domingues, R. C., Hygino da Cruz, L. C., Jr., Rodrigues Lde, S., Gasparetto, E. L., et al. (2009). Diffusion tensor imaging findings in school-aged autistic children. *Journal of Neuroimaging*, 19(4), 337–343.
- Carr, H. Y., & Purcell, E. M. (1954). Effects of diffusion on free precession in nuclear magnetic resonance experiments. *Physical Review*, 94(3), 630–638.
- Catani, M., Jones, D. K., Daly, E., Embiricos, N., Deeley, Q., Pugliese, L., et al. (2008). Altered cerebellar feedback projections in Asperger syndrome. *NeuroImage*, 41(4), 1184–1191.
- Cheng, Y., Chou, K. H., Chen, I. Y., Fan, Y. T., Decety, J., & Lin, C. P. (2010). Atypical development of white

- matter microstructure in adolescents with autism spectrum disorders. *NeuroImage*, 50(3), 873–882.
- Cheung, C., Chua, S. E., Cheung, V., Khong, P. L., Tai, K. S., Wong, T. K., et al. (2009). White matter fractional anisotropy differences and correlates of diagnostic symptoms in autism. *Journal of Child Psychology and Psychiatry*, 50(9), 1102–1112.
- Conturo, T. E., Williams, D. L., Smith, C. D., Gultepe, E., Akbudak, E., & Minshew, N. J. (2008). Neuronal fiber pathway abnormalities in autism: An initial MRI diffusion tensor tracking study of hippocampo-fusiform and amygdalo-fusiform pathways. *Journal of the International Neuropsychological Society*, 14(6), 933–946.
- Fletcher, P. T., Whitaker, R. T., Tao, R., DuBray, M. B., Froehlich, A., Ravichandran, C., et al. (2010). Microstructural connectivity of the arcuate fasciculus in adolescents with high-functioning autism. *NeuroImage*, 51(3), 1117–1125.
- Groen, W. B., Buitelaar, J. K., van der Gaag, R. J., & Zwiers, M. P. (2011). Pervasive microstructural abnormalities in autism: A DTI study. *Journal of Psychiatry & Neuroscience*, 36(1), 32–40.
- Hahn, E. L. (1950). Spin echoes. *Physical Review*, 80(4), 580–594.
- Jou, R. J., Jackowski, A. P., Papademetris, X., Rajeevan, N., Staib, L. H., & Volkmar, F. R. (2011). Diffusion tensor imaging in autism spectrum disorders: Preliminary evidence of abnormal neural connectivity. *The Australian and New Zealand Journal of Psychiatry*, 45(2), 153–162.
- Ke, X., Tang, T., Hong, S., Hang, Y., Zou, B., Li, H., et al. (2009). White matter impairments in autism, evidence from voxel-based morphometry and diffusion tensor imaging. *Brain Research*, 1265, 171–177.
- Keller, T. A., Kana, R. K., & Just, M. A. (2007). A developmental study of the structural integrity of white matter in autism. *Neuroreport*, 18(1), 23–27.
- Knaus, T. A., Silver, A. M., Kennedy, M., Lindgren, K. A., Dominick, K. C., Siegel, J., et al. (2010). Language laterality in autism spectrum disorder and typical controls: A functional, volumetric, and diffusion tensor MRI study. *Brain and Language*, 112(2), 113–120.
- Kumar, A., Sundaram, S. K., Sivaswamy, L., Behen, M. E., Makki, M. I., Ager, J., et al. (2010). Alterations in frontal lobe tracts and corpus callosum in young children with autism spectrum disorder. *Cerebral Cortex*, 20(9), 2103–2113.
- Lange, N., DuBray, M. B., Lee, J. E., Froimowitz, M. P., Froehlich, A., Adluru, N., et al. (2010). Atypical diffusion tensor hemispheric asymmetry in autism. *Autism Research*, 3(6), 350–358.
- Lee, J. E., Bigler, E. D., Alexander, A. L., Lazar, M., DuBray, M. B., Chung, M. K., et al. (2007). Diffusion tensor imaging of white matter in the superior temporal gyrus and temporal stem in autism. *Neuroscience Letters*, 424(2), 127–132.
- Lo, Y. C., Soong, W. T., Gau, S. S., Wu, Y. Y., Lai, M. C., Yeh, F. C., et al. (2011). The loss of asymmetry and reduced interhemispheric connectivity in adolescents with autism: A study using diffusion spectrum imaging tractography. *Psychiatry Research*, 192(1), 60–66.
- Mengotti, P., D'Agostini, S., Terlevic, R., De Colle, C., Biasizzo, E., Londero, D., et al. (2011). Altered white matter integrity and development in children with autism: A combined voxel-based morphometry and diffusion imaging study. *Brain Research Bulletin*, 84(2), 189–195.
- Mori, S. (2007). *Introduction to diffusion tensor imaging*. Amsterdam: Elsevier.
- Mori, S., & Zhang, J. (2006). Principles of diffusion tensor imaging and its applications to basic neuroscience research. *Neuron*, 51(5), 527–539.
- Noriuchi, M., Kikuchi, Y., Yoshiura, T., Kira, R., Shigeto, H., Hara, T., et al. (2010). Altered white matter fractional anisotropy and social impairment in children with autism spectrum disorder. *Brain Research*, 1362, 141–149.
- Pardini, M., Garaci, F. G., Bonzano, L., Roccatagliata, L., Palmieri, M. G., Pompili, E., et al. (2009). White matter reduced streamline coherence in young men with autism and mental retardation. *European Journal of Neurology*, 16(11), 1185–1190.
- Pugliese, L., Catani, M., Ameis, S., Dell'Acqua, F., Thiebaut de Schotten, M., Murphy, C., et al. (2009). The anatomy of extended limbic pathways in Asperger syndrome: A preliminary diffusion tensor imaging tractography study. *NeuroImage*, 47(2), 427–434.
- Sahyoun, C. P., Belliveau, J. W., & Mody, M. (2010a). White matter integrity and pictorial reasoning in high-functioning children with autism. *Brain and Cognition*, 73(3), 180–188.
- Sahyoun, C. P., Belliveau, J. W., Soulieres, I., Schwartz, S., & Mody, M. (2010b). Neuroimaging of the functional and structural networks underlying visuospatial vs. linguistic reasoning in high-functioning autism. *Neuropsychologia*, 48(1), 86–95.
- Shukla, D. K., Keehn, B., Lincoln, A. J., & Muller, R. A. (2010). White matter compromise of callosal and subcortical fiber tracts in children with autism spectrum disorder: A diffusion tensor imaging study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(12), 1269–1278.
- Shukla, D. K., Keehn, B., & Muller, R. A. (2011). Tract-specific analyses of diffusion tensor imaging show widespread white matter compromise in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 52(3), 286–295.
- Sivaswamy, L., Kumar, A., Rajan, D., Behen, M., Muzik, O., Chugani, D., et al. (2010). A diffusion tensor imaging study of the cerebellar pathways in children with autism spectrum disorder. *Journal of Child Neurology*, 25(10), 1223–1231.
- Stejskal, E. O., & Tanner, J. E. (1965). Spin diffusion measurements: Spin echoes in the presence of a time-dependent field gradient. *Journal of Chemical Physics*, 42(1), 288–292.
- Sundaram, S. K., Kumar, A., Makki, M. I., Behen, M. E., Chugani, H. T., & Chugani, D. C. (2008). Diffusion tensor imaging of frontal lobe in autism spectrum disorder. *Cerebral Cortex*, 18(11), 2659–2665.
- Thakkar, K. N., Polli, F. E., Joseph, R. M., Tuch, D. S., Hadjikhani, N., Barton, J. J., et al. (2008). Response

monitoring, repetitive behaviour and anterior cingulate abnormalities in autism spectrum disorders (ASD). *Brain*, 131(9), 2464–2478.

Thomas, C., Humphreys, K., Jung, K. J., Minshew, N., & Behrmann, M. (2011). The anatomy of the callosal and visual-association pathways in high-functioning autism: A DTI tractography study. *Cortex*, 47(7), 863–873.

Torrey, H. C. (1956). Bloch equations with diffusion terms. *Physical Review*, 104(3), 563–565.

DiGeorge Syndrome

► [CATCH 22 \(Chromosome 22q11 Deletion Syndrome\)](#)

Digital, Block-Based Coding of Robots for Students with Autism Spectrum Disorder

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Definition

Background. Robotics and computer programming (i.e., coding) are becoming universal in K-12 STEM classrooms. Some educators and experts are calling coding the “new literacy” because every child will need to be literate in coding in order to excel in the twenty-first century (Burke et al. 2016). Participation in robotics and computer coding/programming have shown a wide range of benefits for typically developing children, including increasing student knowledge and participation in the practices of science, engineering, and in problem-solving and team building skills (e.g., Karp and Maloney 2013).

Coding (i.e., programming or developing) is the process of instructing a computer, app, phone, or website. **Robotics** is the field of computer science dedicated to engineering robots. Students

might be especially engaged when **coding robots** because they can use computer programming to interact with physical robots. To code a robot, students must determine the task they want the robot to complete, design the code to complete the task, and then ensure the robot receives the message in code and completes the desired task.

Block-based coding is a type of coding used primarily to teach introductory coding skills to children and includes predesigned sets of codes the child “drags and drops” from a computer program in order to create a set of instructions. These instructions can be used to run a computer program, control a robot, or manipulate a variety of technologies. One type of block-based coding editor is Ozo-blocky, which allows for students to increase the complexity of their coding skills over time, leading to learning of functions and even text-based coding. Learning these block-based coding skills could be a precursor to understanding more advanced skills, such as learning a computer programming language in which text-based coding is used (e.g., JAVA).

Research. Preliminary evidence suggests youth with autism spectrum disorder (ASD) and challenging behavior can learn how to manipulate a robot’s actions after being explicitly taught how to drag and drop code using block-based code (Knight et al. 2019a, b). High school students in the Knight et al. (2019a, b) study also created novel codes and generalized the skills in a self-directed manner by determining the steps they wanted the robot to follow, programming for those steps, and then assessing whether the robots followed the desired sequence (problem-solving if the robots did not). In a similar study, an elementary-aged student with ASD and challenging behavior was explicitly taught how to program robots by creating color sequences of code using paper and markers for the robots to follow (Knight et al. 2019a, b). In this study, the student learned how to calibrate, draw various tracks for the robot to follow, and draw a three-color code which programmed the robot to move at nitro speed. This student also generalized the coding skill to novel codes and without prompting, created his own three-dimensional paper robot, complete with numerical formulas the robot used to follow directions. None of the children in either study exhibited

challenging behavior at any time throughout the study, even when asked questions for which they did not know the answers (e.g., “Code the robot to go fast” during baseline sessions).

Why teach coding of robotics to children with ASD? Including students with ASD in coding and robotics opportunities throughout their K-12 education could lead to future hobbies, career goals, or an interest in pursuing science, technology, engineering, and math (STEM) college courses or majors. Wei et al. (2013) found individuals with ASD less likely than their typically developing peers and peers with other disabilities to enter college; however, individuals with ASD were enrolled in STEM majors at a disproportionately *higher rate* than their peers. Increasingly, the tech sector (e.g., Microsoft) is examining ways to hire the untapped talent pool of employees with ASD, including allowing performance-based interviews rather than traditional face to face ones. Many of these companies report “finding a return on their investment” in terms of hiring employees with ASD (Eng 2018). Longitudinal studies should examine whether students with ASD who are exposed to STEM skills in a similar way to the students in the current study are more likely to be successful in STEM courses, fields, and careers in their futures.

References and Reading

- Burke, Q., O’Byrne, W. I., & Kafai, Y. B. (2016). Computational participation: Understanding coding as an extension of literacy instruction. *Journal of Adolescent & Adult Literacy*, 59(4), 371–375.
- Eng, D. (2018, July 24). *Where autistic workers thrive*. Fortune Media IP Limited. Retrieved from <https://fortune.com/2018/06/24/where-autistic-workers-thrive/>
- Geist, E. (2016). Robots, programming and coding, oh my! *Childhood Education*, 92, 547–554.
- Gülbahar, Y., & Kalelioglu, F. (2014). The effects of teaching programming via scratch on problem solving skills: A discussion from learners’ perspective. *Informatics in Education*, 13, 33–50.
- Karp, T., & Maloney, P. (2013). Exciting young students in grades K-8 about STEM through an afterschool robotics challenge. *American Journal of Engineering Education*, 4, 39–54.
- Knight, V. F., Wright, J., Wilson, K., & Hooper, A. (2019a). Teaching digital, block-based coding of robots to high school students with autism spectrum disorder and challenging behavior. *Journal of Autism and Developmental Disorders*, 49, 3113–3126. <https://doi.org/10.1007/s10803-019-04033-w>.
- Knight, V. F., Wright, J., & DeFreese, A. (2019b). Teaching robotics coding to a student with ASD and severe problem behavior. *Journal of Autism and Developmental Disorders*, 49, 2632–2636. <https://doi.org/10.1007/s10803-019-03888-3>.
- National Science Foundation. (2017). *National Center for Science and Engineering Statistics. Women, Minorities, and Persons with Disabilities in Science and Engineering: 2017*. Special Report NSF 17-310. Arlington.
- Sullivan, F. R., & Heffernan, J. (2016). Robotic construction kits as computational manipulatives for learning in the STEM disciplines. *Journal of Research on Technology in Education*, 48, 105–128. <https://doi.org/10.1080/15391523.2016.1146563>.
- Taylor, M. S. (2018). Computer programming with pre-K through first-grade students with intellectual disabilities. *The Journal of Special Education*, 52, 78–88. <https://doi.org/10.1177/0022466918761120>.
- Taylor, M. S., Vasquez, E., & Donehower, C. (2017). Computer programming with early elementary students with down syndrome. *Journal of Special Education Technology*, 32, 149–159. <https://doi.org/10.1177/0162643417704439>.
- Wei, X., Yu, J. W., Shattuck, P., McCracken, M., & Blackorby, J. (2013). Science, technology, engineering, and mathematics (STEM) participation among college students with an autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43, 1539–1546. <https://doi.org/10.1007/s10803-012-1700-z>.

Digitigrade Gait

► Toe Walking

Dimensional Versus Categorical Classification

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Synonyms

Class versus variable; Discrete versus continuous

Definition

Is autism a distinct and discrete abnormality or is it simply the upper end of some dimension of normal human variability? Such a contrast of categorical and dimensional conceptualizations of mental health has a long history, especially relevant to the discussion about an autism spectrum and “the autisms.” It should be noted that the question conflates at least two issues: one is the contrast between discrete and continuous but the other is the implicit value judgment that is associated with abnormal and normal. The choice has broad consequences for almost all aspects of measurement, explanation, and much of treatment and policy formulation. It is also the basis of much unproductive and confused debate.

Essentially, all our clinical measurement of autism starts with sets of categorical symptoms or items, though implicit judgments about dimensional severity may be implicit in the scoring of each item. From these, the international diagnostic systems such as DSM (American Psychiatric Association [APA] 1994) spent decades refining rules for combining these items into categorical diagnostic categories. Screening questionnaires (e.g., Charman et al. 2007) commonly start with a similar item set and form total scores that might be considered a dimension. However, the items chosen commonly identify clear abnormality (high threshold or difficult items in the terminology of psychometrics), generate strongly non-normal item-total distributions when applied to a general population, and, through use of cut points, are intended to increase the proportion or probability of caseness rather than to provide a metric. They are not intended to differentiate among the majority who fall within the normal range. By contrast, questionnaires such as the Autism Quotient (e.g., Wheelwright et al. 2010) have items with a range of difficulties and are quite explicitly orientated toward measurement of an autism-related dimension. Questions then arise as to whether the variation that is being differentiated among the normal and supernormal is the same dimension as the variation being

differentiated among the abnormal or whether instead we have a mixture of normal and abnormal populations. Some formal tests have been proposed (e.g., Meehl 1995).

The use of a categorical diagnostic tool and a separate dimensional severity has the potential to lead to inconsistencies, a problem that can be resolved if both are derived from the same instrument, for example, the Autism Diagnostic Observation Schedule (Gotham et al. 2009). It is however crucial to distinguish a dimensional severity measure that relates to symptom abnormality from one that relates to level of impairment. While these are correlated, they are not the same.

So should autism be treated as a category or as a high score on a dimension? The arguments of Pickles and Angold (2003) imply that while a dimensional perspective may be more appropriate when considering some aspects of autism, such as when assessing treatment outcome effects, a categorical perspective may be better, even necessary, when considering another, notably treatment eligibility. Whether that dimension should be severity or impairment will depend on the circumstance.

See Also

- ▶ [Atypical Autism](#)
- ▶ [Autism](#)
- ▶ [Autism Diagnostic Observation Schedule](#)
- ▶ [Broader Autism Phenotype](#)
- ▶ [DSM-IV](#)
- ▶ [Factor Analysis](#)
- ▶ [Latent Variable Modeling](#)
- ▶ [Screening Measures](#)

References and Reading

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Charman, T., Baird, G., Simonoff, E., Loucas, T., Chandler, S., Meldrum, D., et al. (2007). Efficacy of three

- screening instruments in the identification of autism spectrum disorder. *The British Journal of Psychiatry*, 191, 554–559.
- Gotham, K., Pickles, A., & Lord, C. (2009). Standardizing ADOS scores for a measure of severity in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 693–705.
- Meehl, P. (1995). Bootstraps taxonometrics: Solving the classification problem in psychopathology. *American Psychologist*, 50, 266–275.
- Pickles, A., & Angold, A. (2003). Natural categories or fundamental dimensions: On carving nature at the joints and the re-articulation of psychopathology. *Development and Psychopathology*, 15, 529–551.
- Wheelright, S., Auyeung, B., Allison, C., & Baron-Cohen, S. (2010). Defining the broader, medium and narrow autism phenotype among parents using the autism spectrum quotient (AQ). *Molecular Autism*, 1, 1–9.

trials revealed no significant differences in behavior in individuals with autism after taking DMG (Bolman and Richmond 1999; Kern et al. 2001).

References and Reading

- Bolman, W. M., & Richmond, J. A. (1999). A double-blind, placebo controlled pilot trial of low dose dimethylglycine in patients with autistic disorder. *Journal of Autism and Developmental Disorders*, 29(3), 191–194.
- Kern, J. K., Miller, V. S., Cauller, P. L., Kendall, P. R., Mehta, P. J., & Dodd, M. (2001). Effectiveness of N, N-dimethylglycine in autism and pervasive developmental disorder. *Journal of Child Neurology*, 16(3), 169–173.

D

Dimethylglycine

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Synonyms

DMG

Definition

Dimethylglycine (DMG) is a natural substance thought to inhibit the buildup of certain amino acids in the body and enhance the immune response in children with ASD. It is a derivative of the amino acid, glycine. It is found in foods, such as beans, grains, and liver.

DMG supplementation has been proposed as a treatment for autism. Anecdotal reports have suggested that use of DMG improved social behavior, frustration tolerance, speech, and reduced aggressive behavior in individuals with autism. However, two randomized clinical

Diminished Capacity

► [Diminished Responsibility](#)

Diminished Responsibility

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Synonyms

[Diminished capacity](#)

Definition

In criminal law, the defense of diminished responsibility reduces a person's liability in connection with the killing of another if it can be argued that they were suffering from an "abnormality of mind (whether arising from a condition of arrested or

retarded development of mind or any inherent causes or induced by disease or injury) as substantially impaired his mental responsibility for his acts and omissions in doing or being a party to the killing” (Homicide Act (England & Wales) 1957). As this definition from English law indicates, this defense can only be used in connection with charges of murder and, if successful, reduces a person’s culpability such that they are found guilty of the lesser charge of manslaughter rather than murder. It is particularly useful in this context as there are many “disposal” options available to the court for a charge of manslaughter, whereas murder carries the mandatory life sentence.

The defense itself was first recognized under the common law in Scotland and is recognized in several jurisdictions across the globe, including several states in the USA; certain territories in Australia, Hong Kong, and Singapore; and several Caribbean countries. Moreover, in certain jurisdictions without this defense, there have been a number of cases described where a defense of “lack of intent” has been advanced on the grounds of a mental disorder not amounting to insanity, essentially amounting to the same thing as a diminished defense.

It is important to contrast diminished responsibility with defense of insanity, which states that if a person, at the time of the act or omission, was, due to a severe mental disease or defect, unable to appreciate the nature or quality of their act, then they cannot be held criminally responsible for their behavior. As a result, and in contrast to the defense of criminal responsibility, they are deemed to be “not guilty.” Both diminished responsibility and insanity are therefore interpreted at the level of a person’s mens rea (i.e., their ability to form a “guilty mind”).

The relevance of this defense to individuals with ASDs will therefore only really arise in connection with allegations of murder. Such an occurrence will be extremely uncommon, and at the time of writing, no case law on the use of this defense for an individual with ASDs is available.

See Also

► [Violent/Criminal Behavior in Autism](#)

References and Reading

- Bowden, P. (1995). Psychiatry in criminal proceedings. In D. Chiswick & R. Cope (Eds.), *Seminars in practical forensic psychiatry*. London: Royal College of Psychiatrists.
- Samuels, A., O’Driscoll, C., & Allnutt, S. (2007). When killing isn’t murder: Psychiatric and psychological defenses to murder when the insanity defense is not applicable. *Australasian Psychiatry*, 15(6), 474–479.

Diphen [OTC]

► [Diphenhydramine](#)

Diphenhist[®] [OTC]

► [Diphenhydramine](#)

Diphenhydramine

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Synonyms

Aler-Cap [OTC]; Aler-Dryl [OTC]; Aler-Tab [OTC]; AllerMax[®] [OTC]; Altaryl [OTC]; Anti-Hist [OTC]; Banophen[™] [OTC]; Banophen[™] anti-itch [OTC]; Benadryl[®] allergy [OTC]; Benadryl[®] allergy quick dissolve [OTC]; Benadryl[®] children’s allergy [OTC]; Benadryl[®] Children’s Allergy Fastmelt[®] [OTC]; Benadryl[®] Children’s Allergy Perfect Measure[™]; Benadryl[®] children’s allergy quick dissolve [OTC] [DSC]; Benadryl[®] children’s dye-free allergy [OTC]; Benadryl[®] dye-free allergy [OTC]; Benadryl[®] itch relief extra strength [OTC]; Benadryl[®] itch stopping [OTC]; Benadryl[®] itch stopping extra strength [OTC]; Compoz[®] nighttime sleep aid [OTC];

Dermamycin[®] [OTC]; Diphen [OTC]; Diphenhist[®] [OTC]; Dytran[™]; Genahist[™] [OTC]; Histaprin [OTC]; Hydramine [OTC] [DSC]; Nytol[®] quick caps [OTC]; Nytol[®] quick gels [OTC]; PediaCare[®] children's allergy [OTC]; PediaCare[®] children's NightTime cough [OTC]; Siladryl allergy [OTC]; Silphen cough [OTC]; Simply Sleep[™] [OTC]; Sleep-ettes D [OTC]; Sleepinal[®] [OTC]; Sleep-tabs [OTC]; Sominex[®] [OTC]; Sominex[®] maximum strength [OTC]; Theraflu[®] Thin Strips[®] multi symptom [OTC]; Triaminic Thin Strips[®] children's cough and runny nose [OTC]; Twilite[®] [OTC]; Unisom[®] SleepGels[®] maximum strength [OTC]; Unisom[®] SleepMelts[™] [OTC]

Definition

Diphenhydramine (generic name) is also known as Benadryl[®]. Diphenhydramine acts by blocking the effect of histamine on the H1 receptor site. Diphenhydramine inhibits most responses of smooth muscle to histamine. It acts as a vasoconstrictor by inhibiting the vasodilator effects of histamine.

Diphenhydramine is used to provide relief to allergic symptoms caused by histamine release, for sedation, as prevention of motion sickness, as an antitussive, as treatment of phenothiazine-induced dystonic reactions, as adjunct to epinephrine in the treatment of anaphylaxis, and topically for relief of pain and itching.

Diphenhydramine is often used to control agitation or aggression in children; however, it does not have an FDA indication for this use.

Pharmacodynamics/Kinetics:

- Onset of action: Maximum sedative effect: 1–3 h
- Duration: 4–7 h
- Distribution: V_d : 3–22 L/kg
- Protein binding: 78%
- Metabolism: Extensively hepatic n-demethylation via CYP2D6; minor demethylation via CYP1A2, 2C9, and 2C19; smaller degrees in pulmonary and renal systems; significant first-pass effect

- Bioavailability: Oral: ~40–70%
- Half-life elimination: 2–10 h; elderly: 13.5 h
- Time to peak, serum: 2–4 h
- Excretion: Urine (as unchanged drug)

Side Effects

Since diphenhydramine acts by blocking the effect of histamine on the H1 receptor site, it can cause significant anticholinergic side effects such as ataxia; loss of coordination; decreased mucus production; consequent dry, sore throat; xerostomia or dry mouth with possible acceleration of dental caries; cessation of perspiration; consequent decreased epidermal thermal dissipation leading to warm, blotchy, or red skin; increased body temperature; pupil dilation (mydriasis); consequent sensitivity to bright light (photophobia); loss of accommodation (loss of focusing ability, blurred vision (cycloplegia)); double vision (diplopia); increased heart rate (tachycardia); tendency to be easily startled; urinary retention; diminished bowel movement, sometimes ileus; increased intraocular pressure; and shaking.

In high enough doses, diphenhydramine can cause a cholinergic delirium (children and elderly are more prone), and may include confusion, disorientation, agitation, euphoria, or dysphoria; respiratory depression; memory problems; inability to concentrate; wandering thoughts; inability to sustain a train of thought; incoherent speech; wakeful myoclonic jerking; unusual sensitivity to sudden sounds; illogical thinking; visual disturbances (periodic flashes of light, periodic changes in visual field, restricted vision); visual, auditory, or other sensory hallucinations (warping or waving of surfaces and edges, textured surfaces, “dancing” lines, “spiders,” insects); and, rarely, seizures, coma, and death.

Diphenhydramine may cause paradoxical excitation in young children.

Its chemical name is 2-(Diphenylmethoxy)-N, N-dimethylethylamine hydrochloride, and it has a molecular weight of 291.82. The molecular formula is $C_{17}H_{21}NO \cdot HCl$.

See Also

- [Anxiolytics](#)
- [Benzodiazepines](#)
- [Diazepam](#)
- [Gabapentin](#)
- [Oxazepam](#)

References and Reading

- Akutsu, T., Kobayashi, K., Sakurada, K., Ikegaya, H., Furihata, T., & Chiba, K. (2007). Identification of human cytochrome P450 isozymes involved in diphenhydramine N-demethylation. *Drug Metabolism and Disposition*, 35(1), 72–78.
- Blyden, G. T., Greenblatt, D. J., Scavone, J. M., & RI, S. (1986). Pharmacokinetics of diphenhydramine and a demethylated metabolite following intravenous and oral administration. *Journal of Clinical Pharmacology*, 26(7), 529–533.
- Deshmukh, P., Kulkarni, G., & Barzman, D. (2010). Recommendations for pharmacological management of inpatient aggression in children and adolescents. *Psychiatry (Edmont)*, 7(2), 32–40.
- Garnett, W. R. (1986). Diphenhydramine. *American Pharmacology*, NS26(2), 35–40.
- http://www.pfizer.com/files/products/uspi_benadryl.pdf

Diphenhydramine HCl

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Synonyms

[Benadryl](#)

Definition

Diphenhydramine is a histamine H₁ receptor inverse agonist/antagonist as well as a potent

muscarinic acetylcholine receptor antagonist. Being blood–brain barrier permeable, diphenhydramine’s action on CNS H₁ receptors results in a drowsy state. While this drowsy state may be beneficial to ASD patients with sleep problems, there is no evidence indicating diphenhydramine effectiveness in treating ASD’s core symptoms. The drug is most commonly used for allergies and common cold. Side effects may include nausea, vomiting, drowsiness, dizziness, loss of appetite, headache, muscle weakness, and excitement.

See Also

- [Antihistamines: Definition](#)

References and Reading

- Gengo, F., Gabos, C., & Miller, J. K. (1989). The pharmacodynamics of diphenhydramine-induced drowsiness and changes in mental performance. *Clinical Pharmacology and Therapeutics*, 45(1), 15–21.
- McGeer, P. L., Boulding, J. E., Gibson, W. C., & Foulkes, R. G. (1961). Drug-induced extrapyramidal reactions treatment with diphenhydramine hydrochloride and dihydroxyphenylalanine. *JAMA, the Journal of the American Medical Association*, 177(10), 665–670.
- Reiner, P., & Kamondi, A. (1994). Mechanisms of antihistamine-induced sedation in the human brain: H₁ receptor activation reduces a background leakage potassium current. *Neuroscience*, 59(3), 579–588.

DIR Model (Developmental, Individual Difference, Relationship Based): A Parent-Mediated Mental Health Approach to Autism Spectrum Disorders, The

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Definition

The *Developmental, Individual Difference, Relationship-Based* model of intervention (DIR)

provides a developmental framework for interdisciplinary assessment and parent-mediated intervention for autism spectrum and related disorders. This comprehensive model utilizes affect-based interactions and experiences tailored to individual differences to promote development. “D” refers to developmental capacities for shared attention and regulation, engagement across a wide range of emotions, two-way communication, and complex social problem solving which underlie the development of symbol formation, language, and intelligence. Intervention starts with pleasurable synchronous interactions between children and parents, the heart of relationships, which support progress. “I” refers to individual differences related to sensory reactivity and regulation, visual-spatial and auditory/language processing, and purposeful movement. Challenges in these neurobiological factors can make it difficult to participate in the emotional interactions that enable mastery of the developmental capacities (“D”). “R” refers to relationships with caregivers that are the vehicle for affect-based developmentally appropriate interactions. DIR pioneered parent-mediated intervention because of *parents’ ongoing opportunities to support their child’s* everyday functioning to carry out emotionally meaningful goals and provides the essential relationship every child needs. Cultural and environmental influences are also considered. By taking all three dimensions of DIR into account, the foundation for functioning, learning, and relating to others in meaningful ways is established. Current developmental science’s shift away from behavioral reductionism to a relational developmental perspective highlights DIR’s original dynamic and systems model where its three dimensions mutually influence each other throughout the life span.

DIR is commonly known as *Floortime*, the heart of DIR’s intervention. Floortime is both a philosophy and specific technique where caregivers follow the child’s natural emotional interests and create states of heightened pleasure in playful interactions tailored to the child’s unique motor and sensory-processing profile to strengthen the connection between sensation, affect, and motor action. Connecting words to underlying affects that give them purpose and

meaning leads to the formation of symbols, imaginative play, and reflective conversations. Additionally, semi-structured problem solving and social activities; play dates, language, sensory-motor, and visual-spatial therapies; educational programs; family support; and augmentative and biomedical interventions make DIR a comprehensive model. The child’s evolving DIR profile determines the individualized intervention as he progresses. While DIR emphasizes early identification and early intervention, it guides developmental intervention at all ages (Greenspan and Wieder 1998, 2006, 2011; Wieder 2011; Wieder and Greenspan 2001). As an infant mental health model, DIR is strength based; focuses on the child’s emotional life and challenges; addresses anxiety, emotional and behavioral regulation, parent stress, and depression; and engages in reflective practice.

Historical Background

The components of the DIR Model have long theoretical, clinical, and research traditions. Developmental frameworks go as far back as Freud, Erikson, Piaget, Anna Freud, and Mahler and were enhanced by the clinical reports of Spitz, Bowlby, Winnicott, Fraiberg, Lourie, Provence, and others who described the critical impact disrupted and impoverished environments had on early relationships and development. Soon the nuances and bidirectionality of parent infant interaction came under scrutiny as Tronick, Feldman, Fonagy, and others studied synchrony, contingency, affect attunement, match-mismatch, and repair. The importance of nurturing responsive parents and strong parent-child relationships that could protect the family from the disruptions of stress and harmful life events, as well as social emotional development, shaped the new field of infant mental health. Meanwhile, Escalona, Ayres, Brazelton, and others were identifying biological influences in development. This coincided with rejection of psychogenic theory blaming parents for their children’s autism implied by Kanner, Asperger, and Bettelheim in prior decades and opened the door to understanding individual differences (Greenspan et al. 2001; Wieder 2011).

Developmental processes underlie every aspect of experience. Far from automatic, far from milestones, development is a process that captures the essential pathways to functional emotional capacities, which, in turn, contribute to mental health and resilience. This chapter will describe how the DIR Model by Greenspan and Wieder integrated IMH into development in a relationship-based intervention model emphasizing therapeutic pathways that facilitate affect signaling, shared and joint attention, reciprocal communication, problem solving, and symbolic process. It will illustrate how symbolic expression reflects capacities for the full range of emotions within the reciprocal relationship between the child's developmental capacities and the caregiving environment.

The initial formulation, the developmental structuralist theory, developed during an NIMH preventive intervention research study of multi-risk families, described the processes that organize experience at successively higher levels and provide the structure for development (Greenspan et al. 1987; Greenspan and Lourie 1981). As more children at risk for disorders in relating and communicating were identified, this model began to serve children with autism and other special needs providing the first relationship-based developmental approach recognizing individual differences and how emotions impact cognitive and language abilities, as well as social and self-regulation skills, and became known as DIR (Greenspan and Wieder 1998, 2006, 2011).

Rationale or Underlying Theory

Autism's deficits relate to the inability to interact with emotional signals, gestures, and vocalizations, and maintain these interactions with others. DIR hypothesizes these deficits stem from a compromised capacity to connect emotions or intent to motor planning/sequencing, sensations, and later to early forms of symbolic expression of intent or emotions (Greenspan and Shanker 2004; Greenspan and Wieder 1998, 2006). Usually, an

infant connects the sensory system to the motor system through affect, e.g., seeing the caregiver's smiling face or hearing her wooing voice entices the infant to turn and look and listen and smile back. Through many of these interactions, the infant begins to recognize patterns as they share attention, take pleasure in interactions, read each other's cues, and respond to each other over and over again through gaze, vocalizations, and gestures. By the end of the first year, the infant recognizes variations in his caregiver's affect as well as his own feelings related to love, anger, feeling proud, disapproved of, etc. By the second year, these patterns lead to sense of self as purposeful and differentiated sense of others. By the third year, these interactions enable a child to form and give meaning to symbols leading to higher levels of thinking. When challenges with sensory-motor, language comprehension, and visual-spatial knowledge derail this process, affect must be brought in to strengthen connections and enable simultaneous looking, listening, and moving while engaging in meaningful problem-solving interactions, through heightened states of pleasure and other affects. Longer chains of co-regulated affective gesturing will enable the child to recognize variations in the caregiver's gestures, facial expressions, and tone of voice and become aware of his anxiety and repetitive behavior. The relationship becomes the vehicle for affect that transforms labels into meanings leading to symbolic thinking and more complex and abstract reasoning (Greenspan and Shanker 2004; Greenspan and Wieder 1998; Wieder and Greenspan 2003).

DIR theory identifies six fundamental capacities that *emerge* in infancy and *expand* in duration, range, and stability as the child develops. These capacities are necessary for functioning across the life span and can be observed at any point in time.

1. *Regulation and Shared Attention (Between Infant and Caregiver)*. From birth to 3 months, an infant's capacity grows for calm, focused interest in the sights and sounds of the outer

world while she begins to share her interests with the caregiver.

2. *Forming Attachments and Engaging in Relationships.* During the next first 4 months, infants and parents become more intimate as they interact with warmth, trust, and intimacy. They each use their senses to enjoy each other through looks, hugs, songs, and dancing together. Over time, the infant will need to remain related and engaged across the full range of emotions, even when disappointed, scared, angry, or stressed.
3. *Intentional Two-Way Affective Communication.* Between 4 and 10 months, purposeful, continuous flow of interactions with gestures and reciprocating emotions gets underway. The infant begins to act purposefully, more aware of her body and the functions it can perform. As the infant gains motor control over her body and intent, she is better able to communicate her desires. With emerging abilities to reach, sit and turn, crawl and creep, and give, take or drop objects, the infant's awareness of the interpersonal world develops, as does awareness of her body in space and in relation to others who may be moving.
4. *Complex Social Problem Solving.* Between 9 and 18 months, an infant has learned the back and forth rhythm of interactive emotional signaling and begins to use this ability to think about and solve problems that are emotionally meaningful to get what he wants, such as pulling mommy to the door to go outside and play. All of the child's senses work with his motor system as he interacts with others to solve problems. Difficulties arise when he encounters new difficulties to solve as experience expands.
5. *Emotional Ideas.* Between 18 and 36 months, the toddler begins to represent or symbolize intentions, feelings, and ideas in imaginative play and/or language, using gestures, words, and symbols. The toddler now calls on a toy phone, sets up a picnic or tea party, takes the sick baby to the doctor, or repairs his car before driving. These first ideas come from

experiences in real life that are enacted in pretend dramas as the child experiments with different roles and feelings.

6. *Emotional Thinking, Logic, and Sense of Reality.* At about three, the child begins to combine ideas to tell a story as he develops more logic and understanding of himself and others. His stories use imaginative characters and animal figures that talk and may have magic as he discovers he needs more power to encounter the fears and conflicts in life, but reasoning skills click in to elaborate sequences, and stories become increasingly logical and realistic. Over the next few years, the child's emotional and mental abilities move toward abstract thinking, and he develops the ability to distinguish reality from fantasy, self from nonself, and one feeling from another and makes distinctions concerning time and space.

Level six later expands to:

7. *Multicausal and Comparative Thinking.* The child "deepens the plot," explores multiple motives, gets opinions, and compares and contrasts ideas. The child can step into "your shoes" and predicts what you will do based on "affect cues" related to deception, fairness, and justice.
8. *Relativistic or Gray-Area Thinking.* Here, the child differentiates more of his thoughts, rather than thinking only in "black and white" terms. For example, the lion may pay a price for killing the zebra who breaks his leg before being devoured. The child now considers different possibilities and contingencies and is aware of different outcomes and of how he would feel under different circumstances.
9. *Self-Reflection or Thinking Using an Internal Standard.* Now the child has a sense of herself; she can look at and reflect on her performance and feelings. She can question why she is feeling a certain way and contrast this with how she usually feels, or she can compare her current efforts with earlier ones. This kind of thinking

allows her to make inferences about herself and others and create new choices and ideas.

Each of these capacities can be framed as indicators of emotional development throughout life. For example, engagement builds on shared attention with another and must withstand negative feelings, as well as love, trust, and security. Gestures, expressed through tone of voice, movement, facial expressions, and body language continue to be the fundamentals of communication giving meaning to words and negotiate acceptance, approval, ambivalence, and rejection. Complex social problem solving relates to perspective taking and reciprocity that underlie cooperation and collaboration. As language and symbolic development emerge, it is possible to pursue the inner experience of children as they use words and play to express emotions, develop and bridge ideas in logical sequences, and develop reality testing. The progressive levels of thinking are a reflection of emotional thinking and the development of self.

Goals and Objectives

The goal of the DIR model is to enable children on the autism spectrum to form a sense of themselves as intentional, interactive individuals, who can develop cognitive, language, and social and emotional capacities. This calls for the mastery of six functional developmental levels and comprehensive interventions that treat problems related to gaps or variability in these capacities.

Specific objectives:

- To identify the degree to which each developmental level is mastered fully, partially, or unmastered and how stable or consistent. The critical principle is to engage the child at his or her level and to help the child master that level and subsequent levels. When a child has partial mastery of a higher level, e.g., using ideas, but is not fully engaged or interactive, he still needs work at the earlier levels.
- To identify and treat the bioneurological regulatory, sensory, and motor-processing challenges that effect developmental processes.
- To identify gaps in daily adaptation and expected competencies in executive functions.
- To identify family needs for counseling, functioning, and advocacy.
- To organize comprehensive individualized programs that apply principles of affect-based interactions throughout all interventions to support meanings and comprehension.
- To use developmentally appropriate practices which support child initiation, intentionality, communication, and discovery.
- To keep intervention dynamic and flexible, modifying as needed to support rate of progress.

Treatment Participants

This model provides a road map for the treatment of ASD and other developmental, learning, and emotional challenges and diagnoses across the life span. This widespread applicability is possible because it is based on a theory that focuses on capacities fundamental to the development of all individuals. It is also a comprehensive model with a range of interventions that can be tailored to specific underlying sensory processing, motor, and learning challenges, as well as family and cultural factors. Since autism is so heterogeneous, DIR can guide each family to identify the most appropriate program for the child and family based on their individual profiles and helps set priorities. This theory of development is especially useful for early identification in infancy when capacities for regulation and shared attention, engagement, and communicative intent begin and red flags become evident. The intervention begins as soon as challenges are evident or at risk. Infant siblings of children with autism should be monitored. With older ages (children, adolescents, and adults) intervention ensues when gaps in development are identified, rate of progress is less than expected, core capacities need strengthening, and anxiety, stress, and social emotional difficulties arise. DIR intervention can support

other interventions and educational challenges and especially supports families.

Treatment Procedures

Appropriate assessment of all functional areas requires multiple sessions with the child and family. A senior DIR clinician and/or multidisciplinary team determines which developmental capacities have been mastered fully, partially, or not at all and how individual differences in sensory modulation, processing, and motor planning effect each level and underlie particular symptoms, behaviors, and learning challenges. These sessions begin with parent discussions, two 45-min observations of child-caregiver and/or clinician-child interactions, developmental history and review of current functioning, family functioning, prior diagnostic and educational assessments, current programs, consultation with speech and language, occupational, physical, and arts therapists, educators, developmental-pediatricians, optometrists, and mental health colleagues when indicated. Structured tests (ADOS, neuropsychological, educational, speech and language, OT, PT, etc.) are recommended as needed, rather than routine bases, and biomedical evaluation. Intervention planning and priorities are set with the family and team.

DIR is unique in its comprehensiveness, its developmental focus, the role of the family, its emphasis on emotional and symbolic development, and its long-term developmental perspective. As a dynamic model, it is flexible and changes as the child progresses moving onto typical activities. There is no attempt to fit the child into a program, and the specific interventions and frequency depend on individual needs. While these therapies are common to other treatments, DIR provides the unifying goals and principles for an integrated approach. The sessions may be individual and/or group based, in schools or therapy offices, and parents participate (Greenspan and Wieder 2000, 1998, 2006).

DIR interventions include the following:

- Floortime starts with 4–6 daily *spontaneous unstructured “play”* sessions of 20–30 min (15 h weekly) provided by parents and caregivers that focus on obtaining a continuous flow of synchronous interactions as the child climbs the developmental ladder, e.g., 20–50 circles of communication during preschool years. Teachers, therapists, and Floortime players can provide these experiences. Key elements include observe child’s interests, wait for his initiation and response, follow his intentions, and engage in what gives him pleasure using affect cues to sustain shared and joint attention; expand back and forth interactions by helping child do what he intends, becoming playfully obstructive, and increase problem solving in gestures or words to get the child to further elaborate his intent and reciprocity. The parent does not change topics or direct but works within the child’s interests to deepen engagement and expand ideas at pre-symbolic and symbolic levels where imaginative play focuses on emotions and abstract thinking. These Floortime principles also inform all education and therapies so that children are maximally interested and engaged in learning interactions.
- Semi-structured problem-solving interventions and routines. The child with autism may not benefit just from exposure to experiences and needs mediation and systematic implementation. Natural learning from the environment gets derailed by constricted interests, repetitive behaviors, poor imitation, poor auditory and visual-spatial comprehension, and motor planning difficulties (praxis), as well as hypersensitivity and/or under-responsiveness. Opportunities are created daily to get the child to tune into his environment and think when his expectations are challenged, and changes poses a problem for him. These situations are always meaningful and relevant to his emotions such as desire for more or less of something; concern something he is missing or broken, or not finding what he wants in usual places; feeling challenged when needing better motor skills to open containers, or unwrap books or toys; and having to pack his

backpack, serve as a messenger, follow multiple directions, and get ready independently for routines, and other executive function tasks. Reasoning is inserted to comprehend the problem and helps the child feel that the new expectations are not arbitrary, with co-regulated interactions to deal with frustration or disappointment, as well as excitement and success. The challenge increases as the child progresses and involves more elaborate sequences of actions and thoughts with the larger goal of helping child develop competencies off of real-life experiences. In the DIR model, these competencies are part of the Foundational Capacities for Development (FCD) (Wieder and Wachs 2012).

- Social Games and Activities. When meaningful and fun, typical social activities can be practiced with an adult and then peers, e.g., songs and movement such as ring-around-the-rosy, red light-green light, red rover, relay races, treasure hunts, or tag. Secondary goals are to help the child learn to negotiate, make deals, learn turn taking and chance games, and resolve conflicts. At older later, social thinking and group activities and therapy may be useful.
- Play dates and social activities with peers to form friendships and spontaneous interactions, sharing ideas, and negotiations. Number per week depends on age of the child.
- Sensory Motor/Visual-Spatial Activities for several hours daily. Most children with autism have motor planning, coordination, or executive function challenges and rely on memory to stay oriented in space. Many have reduced muscle tone and movement/discriminative movement difficulties. Ocular motor and other visual-spatial processing challenges contribute to attention and learning difficulties as well as daily adaptation. Therefore, intensive daily practice to strengthen these areas is beneficial. Activities range from specific fun exercise routines, involving running, jumping, climbing, chase, and range from solo sports, gymnastic, biking, swimming, or track, to interactive ball sports. These activities support competence and fun and are opportunities for interaction and negotiation.
- Individual and group language, occupational, physical, visual-spatial, and creative art therapies are determined by individual needs of the child. DIR therapists maintain a developmental perspective, include parents in the sessions, and guide home activities between sessions.
- Educational programs range from inclusion in regular education and public and private special education with varying degrees of inclusion, hybrid programs of school- and home-based intervention, tutoring programs, etc. These programs vary in the level of structure provided and are selected on the basis of which setting will best insure comprehension, social interaction, and learning.
- Augmentative and assistive technologies as indicated.
- Family counseling to help parents implement interventions, plan transitions and prepare for adulthood, support family functioning, and provide advocacy when needed.
- Developmental psychotherapy and mental health intervention may be indicated as children and adolescents get older and are struggling with anxiety, depression, mood disorder, etc. Some may also benefit from medications.
- Consideration of stress-related interventions including nutrition and diet, sleep, biomedical interventions, and when indicated, medications addressing regulation and anxiety, possible seizures, impulsiveness, and attention disorders.

Efficacy Information

DIR and Parent Mediated Intervention (PMI) provide research evidence for autism treatment targeting core deficits by strengthening essential relationships every child needs and demonstrates long-term benefits. Research with at risk infant siblings of children with autism under 1 year of age further highlight the potential value of prodromal relationship interventions. Major studies are reviewed here.

The PLAY Project, a DIR-based intervention that found large treatment effects for parent-child interactional behaviors in 2007, was followed by a study of 128 randomized preschool aged children who received 3 h monthly meetings using coaching, modeling, and video feedback for 1 year with a home consultant. Parents also provided 15 h of Floortime weekly and all children received community services. Large treatment effects were reported for parent-child interactional behaviors, improved diagnostic categories on ADOS, and reduced autism symptomology along with reduced parent stress and depression. No differences were found on IQ or language scores (Solomon et al. 2007, 2014).

Another DIR evidence-based study at York University compared 12-month DIR/Floortime treatment for 51 children ages 30–51 months with community standard and found significant gains in initiation of joint attention, enjoyment, fewer ADOS symptoms, changes in interaction skills, and less parent stress and depression (Casenhiser et al. 2011). A subsequent analysis of communicative language acts in conversations with parents was significant, whereas language tests were not (Casenhiser et al. 2014). Parent stress has been reported by others and this study, as well as the PLAY Project, highlights the importance of relationship-based interventions in reducing stress and depression as parents are appreciated for their central to the intervention process.

The Preschool Autism Communication Trial (PACT) offered PMI 2 h, twice monthly for 6 months, and monthly for the next 6 months to randomized group of 152 children between 2 and 4–11 years. Intervention targeted social interactions and communication, was video aided, and provided plans for daily play at home after each session. Significant findings were reported for reduced severity of autism symptoms and increased parental synchronous response to child, child initiations, and shared attention. Effects on language and adaptive functioning in school were small (Green et al. 2010). Although no further intervention was offered, a rare long-

term follow-up study of 80% of the children 6 years later found the PMI group continued to show less severe symptoms, with improved social communication skills and reduced repetitive behavior, but no differences in language or anxiety (Pickles et al. 2016).

These researchers also conducted the iBasis (Intervention British Autism Study of Infant Siblings), the first pre-emptive parent-mediated intervention with 54 infants at familial risk. The 5-month home-based video-aided intervention (9–14 months) reduced the severity of subsequent prodromal autism symptoms over the period to 39-month follow-up, as well as producing positive impact on dyadic parent–infant interactions and child attention and initiation. They focused on interpreting the infant's behavior, recognizing intentions, and enhancing sensitive responding, emotional attunement, and patterns of verbal and nonverbal interaction (Green et al. 2017). Replication of these studies is underway.

Neuroscientists in the IBIS (Infant Brain Imaging Study) Network are using fMRI images from high risk siblings and low risk infants to develop early prediction models, “neural signatures,” for autism. Images found specific brain differences at 6 months that predicted autism at 24 months in group data. In a study of typical and high risk 260 babies, the high risk group showed neural inefficiencies in the right auditory cortex (processes speech sounds) and by 12 month in areas critical for language, touch, and self-awareness. Early brain and behavior changes in autism can be detected before symptoms appear and likely have downstream cascading effects, producing later manifestations and later brain changes in autism. Lewis reported, “Without processing efficiently and pruning, the brain just passes on noise.” They found 17% of high risk infants were diagnosed at 2, and 1.3% of the control group (Lewis et al. 2017). While these prediction models have potential public health implications, they need to be replicated and are not yet available for clinical application now.

Another IBIS prospective longitudinal intervention study of infant siblings used

electrophysiological (EEG) and habituation measures to target social attention. Measures of 33 high risk infants were taken at 6, 12, 18 months in a randomized control study. Between 9 and 11 months, the 19 randomized intervention group received *Promoting First Relationships*, a parent-mediated intervention of 10 weekly sessions, and showed improved social attention in reduced habituation times to face versus object stimuli and greater increase in frontal EEG theta power, closer to the normative responses of matched low risk controls. Replication is necessary, but results suggest relationship-based intervention has the potential to alter the brain systems underpinning social attention and improve outcomes (Jones et al. 2017). On another frontier, Torres et al. (2016) used new mathematical analytics to profile growth data longitudinally from 36 newborn babies with and without complications over 5 months using sensors that hinted at less overall motion in the infants at risk, and detected very early stunting in the development of voluntary neuromotor control flagging risk of neurodevelopmental derailment and potential of early motor-based intervention in high risk infants for autism. These recent findings hold promise for relationship based intervention changing trajectories when identified very early to improve outcomes for children with autism.

Other methods and research also support elements of DIR's complex model. Responsive parent-child interactions have been found to improve social engagement and communication (Gernsbacher 2006; Gutstein 2005; Mahoney and Perales 2005; Prizant et al. 2003; Schreibman and Koegel 2005; Vismara and Rogers 2009). Studies on joint attention, emotional attunement, and play reported gains in language and symbolic thought (Kasari et al. 2006; Kasari et al. 2008a; Mundy et al. 1990). Following a child's lead improved communication as well as language development over long-term periods (Schreibman and Koegel 2005; Siller and Sigman 2002). The strength of relationships and attachment is tied to parent's sensitive responsiveness just as with typical children (Capps et al. 1994; Oppenheim 2009; Rogers et al. 1993). The Early Start Denver model reported affectively engaging social interactions,

improved IQ, language, social interaction, initiative, behavior, and adaptive skills and decreased severity of ASD symptoms (Rogers and Dawson 2010). A 10- to 15-year follow-up study of a subset of 16 boys between 12 and 17, who received preschool DIR intervention, showed empathetic, creative, abstract, and reflective adolescents (Greenspan and Wieder 2005). There is still no definitive evidence on any one method being better than others, but relationships belong to everyone and should be part of all interventions.

Outcome Measurement

DIR studies utilize the standard outcome measures in autism research, including the Autism Diagnostic Observation Scale (ADOS), Achenbach Child and Adolescent Behavior Checklists, BASC (Behavior Assessment System for Children), Greenspan Social-Emotional Growth Chart, Mahoney Maternal and Child Behavior Rating Scales, Mullen Scales of Early Learning, MacArthur CDI, Reynell Developmental Language Scales, Vineland-II, Parenting Stress Index, CES-D Depression Scale, Functional Emotional Assessment Scale, Functional Emotional Developmental Level rating scales, sensory motor profiles related to individual differences, symbolic play scales, and clinical reports.

Qualifications of Treatment Providers

Treatment is provided by multidisciplinary licensed/credentialed professionals who complete a multiyear certificate process to develop competencies within their discipline. They coordinate, consult, and/or oversee intervention teams and supervise paraprofessional Floortime players and assistants who implement specified activities in schools, social activities, and homes. Professionals include clinical and developmental psychologists, regular and special educators, and speech and language, occupational, physical, movement, and creative arts therapists. Senior professionals coordinate teams. Parents work

side by side with the therapists, are coached to provide Floortime, implement the home programs, and have free access to the Profectum Parent Toolbox series of training webcasts (PPT 2017). Developmental pediatricians, pediatricians, child psychiatrists, neurologists, nutritionists, and other specialists are consulted as needed.

See Also

- ▶ [Developmental Intervention Model](#)
- ▶ [Developmental-Pragmatic Approaches/Strategies](#)
- ▶ [Early Start Denver Model](#)
- ▶ [Mutual Regulation](#)
- ▶ [RJA/IJA \(Initiating/Responding to Joint Attention\)](#)
- ▶ [Sensory Processing Assessment](#)

References and Reading

- Capps, L., Sigman, M., & Mundy, P. (1994). Attachment security in children with autism. *Development and Psychopathology*, 6, 249–261.
- Casenheiser, D. M., Shanker, S. G., & Stieben, J. (2011). Learning through interaction in children with autism: Preliminary data from a social-communication-based intervention. *Autism*, published online 2011.
- Casenhisier, D. M., Binns, A., McGill, F., Morderer, P., & Shanker, S. G. (2014). Measuring and supporting language function for children with autism: Evidence from a randomized control trial of a social-interaction-based therapy. *Journal of Autism and Developmental Disorders*, 45(3).
- Dawson, G., & Galpert, I. (1990). Mother's use of imitative play for facilitating social responsiveness and toy play in young autistic children. *Development and Psychopathology*, 2, 151–162.
- Gernsbacher, M. (2006). Toward a behavior of reciprocity. *Journal of Developmental Processes*, 1, 139–152.
- Green, J., Charman, T., McConachie, H., Aldred, C., Slonims, V., Howlin, P., Le Couteur, A., Leadbitter, K., Hudrey, K., Byford, S., Barrett, B., Temple, K., Macdonald, W., Pickles, A., & The PACT Consortium. (2010). Parent-mediated communication-focused treatment in children with autism (PACT): A randomized controlled trial. *Lancet*, 375, 2152–2160.
- Green, J., Wai Wan, M., Gulraud, J., Holsgrove, S., McNally, J., Slonims, V., Elsabbagh, M., Charman, T., Pickles, A., & Johnson, M. (2013). The BASIS team. *Journal of Autism and Developmental Disorders*, 43, 2502–2514.
- Green, J., Pickles, A., Pasco, G., Bedford, R., Wai Wan, M., Elsabbagh, M., Slonims, V., Gliga, T., Jones, E., Cheung, C., Charman, T., & Johnson, M. (2017). Randomised trial of a parent-mediated intervention for infants at high risk for autism: Longitudinal outcomes to age 3 years. *Journal of Child Psychology and Psychiatry*, 58, 1330–1340.
- Greenspan, S. (1992). *Infancy and early childhood: The practice of clinical assessment and intervention with emotional and developmental challenges*. Madison: International Universities Press.
- Greenspan, S. (2004). *Greenspan social-emotional growth chart: A screening questionnaire for infants and young children*. Bulverde: The Psychological Corporation.
- Greenspan, S. I., & Lourie, R. S. (1981). Developmental structuralist approach to the classification of adaptive and pathologic personality organization: Application to infancy and early childhood. *The American Journal of Psychiatry*, 138, 725–735.
- Greenspan, S., & Shanker, S. (2004). *The first idea: How symbols, language and intelligence evolved from our primate ancestors to modern humans*. Reading: Perseus Books.
- Greenspan, S., & Wieder, S. (1997). Developmental patterns and outcomes in infants and children with disorders in relating and communicating: A chart review of 200 cases of children with autistic spectrum diagnoses. *The Journal of Developmental and Learning Disorders*, 1, 87–141.
- Greenspan, S., & Wieder, S. (1998). *The child with special needs: Encouraging intellectual and emotional growth*. Reading: Perseus Books.
- Greenspan, S., & Wieder, S. (2000). Principles of clinical practice for assessment and intervention. Developmentally appropriate interactions and practices. Developmentally based approach to the evaluation process. In *Interdisciplinary council on developmental and learning disorders clinical practice guidelines* (pp. 261–282). Bethesda: Interdisciplinary Council on Developmental and Learning Disorders.
- Greenspan, S., & Wieder, S. (2005). Can children with autism master the core deficits and become empathetic, creative and reflective? A ten to fifteen year follow-up of a subgroup of children with autism spectrum disorders (ASD) who received a comprehensive developmental, individual-difference, relationship-based (DIR) approach. *The Journal of Developmental and Learning Disorders*, 9, 39–61.
- Greenspan, S., & Wieder, S. (2006). *Engaging autism: The Floortime approach to helping children relate, communicate, and think*. Cambridge, MA: DaCapo Press/Perseus Books.
- Greenspan, S. I., & Wieder, S. (2011). Relationship-based early intervention approach to autistic spectrum disorders: The DIR model 1068. In D. G. Amaral, G. Dawson, & D. H. Geschwind (Eds.), *Autism spectrum disorders* (pp. 1068–1080). New York: Oxford University Press.

- Greenspan, S., Wieder, S., Lieberman, A., Nover, R., Lourie, R., & Robinson, M. (1987). *Infants in multirisk families: Case studies in preventive intervention. Clinical infant reports: No. 3*. New York: International Universities Press.
- Greenspan, S., DeGangi, G., & Wieder, S. (2001). *The functional emotional assessment scale (FEAS) for infancy and early childhood: Clinical and research applications*. Bethesda: Interdisciplinary Council on Developmental and Learning Disorders.
- Gutstein, S. (2005). Relationship development intervention: Developing a treatment program to address the unique social and emotional deficits of autism spectrum disorders. *Autism Spectrum Quarterly (Winter)*, 8–12.
- Gutstein, S. E., & Sheely, R. K. (2002). *Relationship development intervention with young children: Social and emotional developmental activities for Asperger syndrome, autism, PDD and NLD*. London: Jessica Kingsley.
- Jones, E. H., Dawson, G., Kelly, J., Estes, A., & Webb, S. J. (2017). Parent-delivered early intervention in infants at risk for ASD: Effects on electrophysiological and habituation measures of social attention. *Autism Research*, 00, 1–12.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in young children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry*, 47, 611–620.
- Kasari, C., Freeman, S. F. N., Paparella, T., & Jahromi, L. B. (2008a). Language outcome in autism: Randomized comparison of joint attention and play interventions. *The Journal of Clinical Psychiatry*, 76, 125–137.
- Kasari, C., Paparella, T., Freeman, S., & Jahromi, L. B. (2008b). Language outcome in autism: Randomized comparison of joint attention and play interventions. *Journal of Consulting and Clinical Psychology*, 76(1), 25–137.
- Koegel, R. I., Koegel, L. K., Harrower, J. K., & Carter, C. M. (1999). Pivotal response intervention1: Overview of approach. *Journal of the Association for Persons with Severe Handicaps*, 24, 174–185.
- Lewis, J. D., Evans, A.C., Pruitt, J.R., Botteron, K.N., McKinstry, R.C., Zwaigenbaum, L., Estes, A., Collins, D.L., Kostopoulos, P., Gerig, G., Dager, S.R., Paterson, S., Schultz, R.T., Styner, M.A., Hazlett, H.C., Piven, J., for The Infant Brain Imaging Study Network (2017). The emergence of network inefficiencies in infants with autism spectrum disorders. *Brain Psychiatry*, 82, 176–185.
- Lord, C., & McGee, J. (2001). *Educating children with autism*. Washington, DC: National Research Council National Academy Press.
- Mahoney, G., & Perales, F. (2005). A comparison of the impact of relationship-focused intervention on young children with pervasive developmental disorders and other disabilities. *Journal of Developmental and Behavioral Pediatrics*, 26, 77–85.
- Miller, L. J., Coll, J. R., & Schoen, S. A. (2007). A randomized controlled pilot study of the effectiveness of occupational therapy for children with sensory modulation disorder. *American Journal of Occupational Therapy*, 61, 228–238.
- Mostofsky, S., Dubai, P., Jerath, V., Jansiewicz, E., Goldberg, M., & Denckla, M. (2006). Developmental dyspraxia is not limited to imitation in children with autism spectrum disorders. *Journal of International Neuropsychological Society*, 12, 314–326.
- Mostofsky, S. H., Burgess, M. P., & Gidley Larson, J. C. (2007). Increased motor cortex white matter volume predicts motor impairment in autism. *Brain*, 130, 2117–2122.
- Mundy, P., Sigman, M., & Kasari, C. (1990). A longitudinal study of joint attention and language development in autistic children. *Journal of Autism and Developmental Disorders*, 20, 115–128.
- Oppenheim, D. (2009). Maternal insightfulness and resolution of diagnosis are associated with secure attachment in preschoolers with autism spectrum disorders. *Child Development*, 80, 519–527.
- Pfeiffer, B. A., Koenig, K., Kinnealey, M., Sheppard, M., & Henderson, L. (2011). Research scholars initiative – effectiveness of sensory integration interventions in children with autism spectrum disorders: A pilot study. *American Journal of Occupational Therapy*, 65, 76–85.
- Pickles, A., Le Couteur, A., Leadbitter, K., Salomne, E., Cole-Fletcher, R., Tobin, H., Gammer, I., Lowry, J., Vamvakas, G., Byford, S., Aldred, C., Slonims, V., McConachie, H., Howlin, P., Parr, J., Charman, T., & Green, J. (2016). Parent-mediated social communication therapy for young children (PACT): Long term follow up of a randomized controlled trial. Parent mediated communication-focused treatment. *Lancet*, 388, 2501–2508.
- Prizant, B., Wetherby, A., Rubin, E., & Laurent, A. (2003). The SCERTS model: A transactional, family-centered approach to enhancing communication and socio-emotional abilities of children with autism spectrum disorder. *Infants and Young Children*, 16, 296–316.
- Profectum Parent Toolbox, 2017. www.Profectum.org.
- Rogers, S. J., & Dawson, G. (2010). *Early start Denver model for young children with autism*. New York: Guilford.
- Rogers, S., Herbison, J., Lewis, H., Pantone, J., & Reis, K. (1986). An approach for enhancing the symbolic, communicative, and interpersonal functioning of young children with autism and severe emotional handicaps. *Journal of the Division for Early Childhood*, 10, 135–148.
- Rogers, S. J., Ozonoff, S., & Maslin-Cole, C. (1993). Developmental aspects of attachment behavior in young children with pervasive developmental

- disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 1274–1282.
- Schreibman, L., & Koegel, R. L. (2005). Training for parents of children with autism: Pivotal responses, generalization, and individualization of intervention. In E. D. Hibbs & P. S. Jensen (Eds.), *Psychosocial treatment for child and adolescent disorders: Empirically based strategies for clinical practice* (pp. 605–631). Washington, DC: American Psychological Association.
- Seida, J., Ospina, M., Karkhaneh, M., Hartling, L., Smith, V., & Clark, B. (2009). Systematic reviews of psychosocial interventions for autism: An umbrella review. *Developmental Medicine and Child Neurology*, 51, 95–104.
- Siegel, D. (2001). Toward an interpersonal neurobiology of the developing mind: Attachment, “mindsight,” and neural integration. *Infant Mental Health Journal*, 22, 67–94.
- Siller, M., & Sigman, M. (2002). The behavior of parents of children with autism predict the subsequent development of their children’s communication. *Journal of Autism and Developmental Disorders*, 32, 77–89.
- Solomon, R., Necheles, J., Ferch, D., & Bruckman, D. (2007). Pilot study of a parent training program for young children with autism: The P.L.A.Y. project home consultation model. *Autism: The International Journal of Research and Practice*, 11, 205–224.
- Solomon, R., Van Egeren, L. A., Mahoney, G., Quon Huber, M., & Zimmerman, P. (2014). PLAY project home consultation intervention program for young children with autism spectrum disorders: A randomized controlled trial. *Journal of Developmental and Behavioral Pediatrics*, 35(8), 475–485.
- Spreckley, M., & Boyd, R. (2009). Efficacy of applied behavioral intervention in preschool children with autism for improving cognitive, language, and adaptive behavior: A systematic review and meta-analysis. *Journal of Pediatrics*, 154, 338–344.
- Torres, E. B., Smith, B., Mistry, S., Brinckner, M., & Whyatt, C. (2016). Neonatal diagnostics: Toward dynamic growth charts of Neuromotor control. *Journal Frontiers in Pediatrics*, 4, Article 21.
- Vismara, L., & Rogers, S. J. (2009). Can one hour per week of therapy lead to lasting changes in children with autism? *Autism*, 13(1), 93–115.
- Wallace, K. S., & Rogers, S. J. (2010). Intervening in infancy: Implications for autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 51(12), 1300–1320.
- Wetherby, A. M., Prizant, B. M., & Hutchinson, T. (1998). Communicative, social-affective and symbolic profiles of young children with autism and pervasive developmental disorders. *American Journal of Speech-Language Pathology*, 7, 79–91.
- Wieder, S. (1996). Climbing the “symbolic ladder”: Assessing young children’s symbolic and representational capacities through observation of free play interaction. In S. Meisels & E. Fenichel (Eds.), *New visions for the developmental assessment of infants and young children* (pp. 267–287). Washington, DC: Zero to Three.
- Wieder, S. (2011). DIR: Developmental, individual-difference, relationship-based model: A dynamic model for the 21st century. In D. Zager, M. Wehmeyer, & R. Simpson (Eds.), *Research-based principles and practices for educating students with autism* (pp. 82–98). New York: Routledge/Taylor & Francis.
- Wieder, S., & Greenspan, S. (2001). The DIR (developmental, individual-difference, relationship-based) approach to assessment and intervention planning. *Zero to Three*, 21, 11–19.
- Wieder, S., & Greenspan, S. (2003). Climbing the symbolic ladder in the DIR model through floortime/interactive play. *Autism*, 7, 425–436.
- Wieder, S., & Wachs, H. (2012). *Visual spatial portals to thinking, feeling and movement: Advancing competencies and emotional development in children with learning and autism spectrum disorders*. Mendham: Profectum Foundation.
- Williams, D. L., & Minshew, N. J. (2007). Understanding autism and related disorders: What has imaging taught us? *Neuroimaging Clinics of North America*, 17(IV), 495–509.
- Zwaigenbaum, L., Bryson, S., Lord, C., Rogers, S., Carter, A., Carver, L., et al. (2009). Clinical assessment and management of toddlers with suspected autism spectrum disorder: Insights from studies of high-risk infants. *Pediatrics*, 123, 1383–1391.

Direct Instruction

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Definition

Direct instruction is a general term used to describe the explicit teaching of a skill set and was developed by Siegfried Engelmann, Wesley Becker, and colleagues. It is a teaching model that focuses on systematically planned lessons and

clearly defined teaching procedures. It often involves breaking down instructional targets into smaller components and using a scaffolding approach to teach material. Direct instruction emphasizes the explicit teaching of skills and requires that students consistently demonstrate mastery before moving on to new material. Direct instruction requires that students actively participate in learning and necessarily involves meaningful teacher-student interaction.

See Also

► [Didactic Approaches](#)

References and Reading

- Goeke, J. (2008). *Explicit instruction: Strategies for meaningful direct teaching*. Boston: Allyn and Bacon.
- Marchand-Martella, N. E., Slocum, T. A., & Martella, R. C. (2003). *Introduction to direct instruction*. Boston: Allyn and Bacon.
- Przychodzin, A. M., Marchand-Martella, N. E., Martella, R. C., & Azim, D. (2004). Direct instruction mathematics programs: An overview and research summary. *Journal of Direct Instruction*, 4, 53–84.

Direct Observation

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Definition

Direct observation, also known as observational study, is a method of collecting evaluative information in which the evaluator watches the subject in his or her usual environment without altering that environment. Direct observation is used when other data collection procedures, such as surveys, questionnaires, etc., are not effective; when the goal is to evaluate an ongoing behavior process,

event, or situation; or when there are physical outcomes that can be readily seen.

Direct observation can be overt, when the subject and individuals in the environment know the purpose of the observation, or covert, when the subject and individuals in the environment are unaware of the purpose of the observation.

Structured direct observations are most appropriate when standardized information needs to be gathered, and result in quantitative data. Unstructured direct observation looks at natural occurrence and provides qualitative data, such as that used when administering the Childhood Autism Rating Scale (CARS), the Checklist for Autism in Toddlers (CHAT), and the American Psychiatric Association's Diagnostic and Statistical Manual, 4th Edition (DSM-IV).

Data recording for direct observation includes narrative notes, video or photographs, recording checklist (yes/no), observation guidelines (printed forms with space to write notes), and combinations of the above. Direct observation provides the highest degree of ecological validity but lowest degree of experimental control. The value of direct observation is directly related to the evaluator's ability to capture detail, determine what is important, and interpret what has been observed. Because autism is a disorder that is diagnosed and individuals are evaluated through behavioral observation, direct observation is a critical evaluative tool that affords an objective perspective of the individual's profile.

References and Reading

- Barnhill, G. P. (2002). Behavioral, social and emotional assessment of students with ASD. *Assessment for Effective Intervention*, 27(1–2), 47–55.
- Carr, E. G., Ladd, M. V., & Schulte, C. F. (2008). Validation of the contextual assessment inventory for problem behavior. *Journal of Positive Behavior Interventions*, 10, 91–104.
- Drury, C. G. (1995). Methods for direct observation of performance. In J. R. Wilson & E. N. Corlett (Eds.), *Evolution of human work; a practical ergonomics methodology* (2nd ed., pp. 45–68). Philadelphia: Taylor and Francis.

- Matson, J. L., & Wilkins, J. (2007). A critical review of assessment targets and methods for social skill excesses and deficits for children with autism spectrum disorders. *Research in Autism Spectrum Disorders, 1*, 28–37.
- Noterdaeme, M., Mildenerger, K., Sitter, S., & Amorosa, H. (2002). Parent information and direct observation in the diagnosis of pervasive and specific developmental disorders. *Autism, 6*(2), 159–168. Abstract retrieved from <http://www.online.sagepub.com>.

Direct Observation Scales

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Definition

Direct observation scales are structured instruments used to collect first-hand information regarding observable behaviors. They contrast to scales that provide indirect accounts, such as rating scales, report forms, or interviews with parents, caregivers, or teachers regarding behaviors of an individual, although both provide important information. Direct observation scales are critical in both diagnosis and intervention with children with autism spectrum disorders. While these scales can vary in format, they share common characteristics, including having a structure as to what is attended to in the observation and what is coded. Thus, they are not simply a description of what an individual is doing. For autism spectrum disorders, the behaviors central to the diagnosis are most often the target of direct observation scales (e.g., eye contact, social initiations, conversational turn-taking) and behaviors that interfere with functioning and are the targets of intervention (e.g., aggression or bolting). Direct observation scales can be more or less structured in how the observation situation is set up and how the data is collected. Some direct observation scales

provide a set of semi-structured activities and record and code observed target behavior within those activities, while others provide a structured means to record and code direct observations in the natural environment. A number of direct observation scales have been developed for diagnostic purposes including those that provide semi-structured activities in which to record and code observations, such as the widely used Autism Diagnostic Observational Schedule (ADOS; Lord et al. 2001) and the Autism Observation Scale for Infants (AOSI; Bryson et al. 2000). For assessment of interfering behaviors for the purpose of treatment planning, direct observation scales are often used during a functional behavioral assessment (FBA). These direct observations are most often conducted in the natural environment and data is collected with a very systematic methodology. Different types of data can be collected during an FBA including interval, frequency, duration, latency, and antecedent-behavior-consequence (ABC) data.

See Also

- [Autism Diagnostic Observation Schedule](#)
- [Direct Observation](#)
- [Functional Behavior Assessment](#)

References and Reading

- Bryson, S. E., McDermott, C., Rombough, V., Brian, J., & Zwaigenbaum, L. (2000). *The autism observation scale for infants*. Toronto: Unpublished Scale.
- Lord, C., Rutter, M., DiLavore, P., & Risi, S. (2001). *Autism Diagnostic Observation Schedule (ADOS) manual*. Los Angeles: Western Psychological Services.

Directive Play Therapy

- [Play Therapy](#)

Disability

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Synonyms

[Affliction](#); [Detriment in skill](#); [Exceptionality](#); [Incapacity](#); [Relative weakness in an area of functioning](#)

Definition

In common terms, it is the condition of being unable to perform a skill as a consequence of physical or mental incapacity. It may suggest impairments, limitations, or restrictions on performing certain activities. Autism is one category of disability under special education regulations (IDEA 2004). Other categories of disability include intellectual disability, a hearing impairment (including deafness), a speech or language impairment, a visual impairment (including blindness), a serious emotional disturbance (referred to in this part as emotional disturbance), an orthopedic impairment, traumatic brain injury, other health impairments, a specific learning disability, deaf-blindness, or multiple disabilities. A child with a disability under IDEA (2004) needs special education and related services. Additionally, the law provides for services for children with a disability aged 3–9, to include a child experiencing developmental delays as measured by appropriate diagnostic instruments and procedures, in one or more of the following areas: physical development; cognitive development; communication development; social or emotional development; or adaptive development; and by reason thereof, needs special education and related services. In some situations, children with a disability may be said to present a developmental delay if members

of the IEP team are not certain that the individual meets the criteria for autism.

See Also

- ▶ [Exceptionality](#)
- ▶ [Psychotic Disorder](#)

References and Reading

Assistance to States for the Education of Children with Disabilities and Preschool Grants for Children with Disabilities; Final Rule, 2004, 4000-01-U Department of Education 34 CFR Parts 300 and 301, Part II. Individuals with Disabilities Education Act of 2004, 20 U.S.C., et. seq.

DISCO

- ▶ [Diagnostic Interview for Social and Communication Disorders](#)

Discontinuous Data Collection

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Synonyms

[Discontinuous measurement](#); [Time sampling procedures](#)

Definition

Discontinuous data collection methods are those that capture only a sample of the behavior that is observed. During discrete trial teaching, discontinuous data collection may be used to record a

sample of the instructional trials such as the first trial or first few trials. When data are collected on the first instructional trial, the level of performance in the absence of immediately preceding learned trials or prompts is revealed. Discontinuous data collection may also be used to capture a sample of behavior during an observation by dividing the observation into predetermined duration intervals and recording either the occurrence or nonoccurrence of targeted behavior. Examples of this type of measurement include partial interval recording, whole interval recording, and momentary time sample. While continuous data collection may be more comprehensive, discontinuous data collection may be sufficient for decision-making and analysis if the sample represents a valid approximation of the true parameters of the behavior of interest.

See Also

- [Continuous Data Collection](#)
- [Time Sampling Procedures](#)

References and Reading

- Fiske, K., & Delmolino, L. (2012). Use of discontinuous methods of data collection in behavioral intervention: Guidelines for practitioners. *Behavior analysis in practice*, 5(2), 77–81.
- Lerman, D., Harper Dittlinger, L., Fentress, G., & Lanagan, T. (2011). A comparison of methods for collecting data on performance during discrete trial teaching. *Behavior analysis in practice*, 4(1), 53–62.
- Najdowski, A. C., Chilingaryan, V., Bergstrom, R., Granpeesheh, D., Balasanyan, S., Aguilar, B., Tarbox, J., & Roane, H. (2009). Comparison of data-collection methods in a behavioral intervention program for children with pervasive developmental disorders: A replication. *Journal of Applied Behavior Analysis*, 42, 827–832.

Discourse Management

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Synonyms

[Conversational discourse](#); [Pragmatic language](#); [Topic management](#); [Turn-taking](#)

Definition

Discourse management refers to the ability to organize topics and turns and to repair any communication breakdowns during conversation. Carrying on a conversation involves the appropriate use and coordination of a variety of skills including: initiating and maintaining topics, using eye contact, taking turns, being polite, and observing and responding appropriately to nonverbal behaviors. During discourse, individuals must monitor their own contributions while taking into account the explicit and implicit responses, intentions, and knowledge of their conversational partner (s). The existing literature on language use and conversational skills in individuals with ASD indicates that the ability to contribute new information to topics introduced by others, shift topics appropriately, provide turns for others within conversation, take turns appropriately, and provide repairs for conversational breakdowns frequently present challenges to many individuals with ASD.

Discontinuous Measurement

- [Discontinuous Data Collection](#)

See Also

- [Conversational Manner](#)
- [Pragmatics](#)

References and Reading

- Adams, C., Green, J., Gilchrist, A., & Cox, A. (2002). Conversational behaviour of children with Asperger syndrome and conduct disorder. *Journal of Child Psychology and Psychiatry*, 43, 679–690.
- Bauminger-Zviely, N., Karin, E., Kimhi, Y., & Agam-Ben-Artzi, G. (2014). Spontaneous peer conversation in preschoolers with high-functioning autism spectrum disorder versus typical development. *Journal of Child Psychology and Psychiatry*, 55(4), 363–373.
- Cole, C. L. (2015). Peer-mediated intervention for social communication difficulties in adolescents with autism: Literature review and research recommendations. *International Journal of Medical, Health, Biomedical, Bioengineering and Pharmaceutical Engineering*, 9, 347–350.
- Colle, L., Baron-Cohen, S., Wheelwright, S., & van der Lely, H. K. J. (2008). Narrative discourse in adults with high-functioning autism and Asperger syndrome. *Journal of Autism and Developmental Disorders*, 38(1), 28–40. <https://doi.org/10.1007/s10803-007-0357-5>.
- Fine, J., Bartolucci, G., Szatmari, P., & Ginsberg, G. (1994). Cohesive discourse in pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(3), 315–329.
- Geller, E. (1998). An investigation of communication breakdowns and repairs in verbal autistic children. *The British Journal of Development Disabilities*, 44(87), 71–85.
- Hutchins, T. L., & Brien, A. (2016). Conversational topic moderates social attention in autism spectrum disorder: Talking about emotions is like driving in a snowstorm. *Research in Autism Spectrum Disorders*, 26, 99–110.
- Paul, R. (2007). *Language disorders from infancy to adolescence* (3rd ed.). St. Louis: Elsevier.
- Theimann, K. S., & Goldstein, H. (2001). Social stories, written text cues, and video feedback: Effect on social communication of children with autism. *Journal of Applied Behavior Analysis*, 34(4), 425–446.
- Volden, J. (2004). Conversational repair in speakers with autism spectrum disorder. *International Journal of Language & Communication Disorders*, 39(2), 171–189.

Discretionary Trust

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Discretionary Trust

A discretionary trust is a common method of estate planning whereby the creator of the trust (the *settlor*) transfers the assets to another (the *trustee*), who then has a duty to hold and manage the assets for the benefit of a third party (the *beneficiary*). If the trust is established by a living settlor, rather than through a will, the trust acts as a will substitute and the transferred estate avoids probate and some estate taxes.

The terms of a discretionary trust delegate power to the trustee to decide the “time, purpose and amount of all distributions” to one or more beneficiaries (POMS SI §01120.200.B.10). The trustee may be given complete authority over distributions that the trustee considers advisable. For example, a settlor might delegate absolute discretion upon a trustee in order to protect the long-term interests of a financially irresponsible beneficiary, or to establish a supplemental needs trust for lifetime support of an orphaned or disabled child. The purpose of such trusts may be to protect a spendthrift beneficiary from poor financial decisions; provide funds that “supplement, but not supplant, sources of income including SSI or other government benefits” (POMS SI §01120.200B.13); and shield the trust against invasion by the beneficiary, or the beneficiary’s general creditors. The trustee may only be compelled to distribute money from the trust under very restricted circumstances.

Alternatively, the settlor may limit the trustee’s discretion by directing that distributions be used for specific purposes. Often, such an arrangement is made to provide for the long-term care and support of an incompetent beneficiary. For instance, a trustee’s discretion is limited to

Discrete Versus Continuous

► Dimensional Versus Categorical Classification

distributing money for the “comfortable support, education, health and maintenance” of the beneficiary (Leslie and Sterk 2006). Support trusts are often used to provide care for a surviving spouse, or an incompetent or elderly adult.

Depending on the intent and objectives of the trust, creating a discretionary trust is an effective estate planning tool that may extend the life and availability of estate assets, while offering flexibility for the trustee to deal with unanticipated events. However, problems may arise such as a trustee’s breach of duty to manage the trust in “good faith,” or “in accordance with its terms and purposes,” or in the “interests of the beneficiaries” (Uniform Trust Code §801 *et. seq.*, 2005). This may be demonstrated when the trustee abuses the assigned discretionary power, and the beneficiary is not competent to address the breach. Therefore, one must carefully evaluate the goals of the settlor, the competence and trustworthiness of the trustee, and the long-term needs of the beneficiary in order to determine if a discretionary trust is the best estate planning option.

See Also

- [Beneficiary](#)
- [Trust](#)

References and Reading

Sources

- Andersen, R. (2003). *Understanding trusts and estates*. Newark: Matthew Bender & Company.
- Averill, L. (2005). *Wills, trusts, and future interests*. St. Paul: Thomson/West.
- Leslie, M., & Sterk, S. (2006). *Trusts and estates*. New York: Foundation Press.
- Programs Operations Manual System (POMS): Trusts §SI 01120.200A *et. seq.* Retrieved March 8, 2011., from <https://secure.ssa.gov/apps10/poms.nsf/lnx/0501120200>
- Rios v. Astrue, 159 Soc. Sec. Rep. Service 604 (N.D. Ill. 2010). Retrieved March 7, 2011, from Lexis-Nexis Database: 2010 U.S. Dist. LEXIS 121256
- Social Security Act (SSA), §42 U.S.C. §1396p
- Uniform Trust Code §801 (2005) *et. seq.*

Discrimination

- [Stimulus Control](#)

Discrimination Training

- [Stimulus Control](#)

Discriminative Stimulus

- [Stimulus Control](#)

Disfluency

- [Fluency and Fluency Disorders](#)
- [Stuttering](#)

Disguised Mands

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Definition

As described in the mand section of this encyclopedia, mands (a term derived from command, demand, counterdemand) are typically emitted from one person to another and yield a specific consequence (Skinner 1957, p. 53). For example, if a person said, “I want a cookie” to another person, and it led to the delivery of a cookie,

such exchange may be considered a mand. Unlike this example, when a mand does not directly state the specific intended consequence, the mand is called a disguised mand. Disguised mands are “responses that are under the control of an establishing operation (e.g., deprivation from cookies) and a discriminative stimulus (e.g., the presence of a listener) but the response does not specify the reinforcing consequence (e.g., access to cookies)” (Najdowski et al. 2017). In short, statements from one person to another, such as “I feel hot” or “I’m thirsty,” may be considered disguised mands because they do not directly indicate a specific desired consequence. Because disguised mands are indirect requests, initiating and responding to disguised mands may be challenging for individuals with social communication deficits, especially individuals with autism spectrum disorder (ASD; American Psychiatric Association 2013). Often, individuals with ASD have difficulty responding appropriately to sarcasm, metaphors, idioms, and other forms of language where the intended message is not directly stated (Najdowski et al. 2017). Because disguised mands are common parlance, responding in a socially acceptable manner is likely to improve social interactions but may require direct intervention. At the time of this publication, there are two studies that have conducted behavioral interventions to teach individuals with ASD to respond to disguised mands. Najdowski et al. (2017) used a combination of rule setting, role-play, and multiple exemplar training to teach three individuals with ASD to respond to disguised mands. First, the participants were provided with a rule that emphasized the importance of responding to people’s hints by helping them “because doing so makes them feel good and it will help [them] make and keep friends.” Then, the participants completed a role-play activity that involved responding to disguised mands, such as providing a pillow to an investigator that emitted the mand “I’m not very comfortable with my head just lying on the floor.” Last, the participants were provided with various examples and opportunities to respond to disguised mands, such as “I’m hot,” “I’m cold,” “I’m hungry,” etc. A replication and extension of Najdowski et al.’s study was

conducted by Erhard (2019) to include the training of nonvocal disguised mands as well. For example, in Erhard’s study, the participants were also taught to respond to disguised mands, such as when another person fans themselves with their hand when they are hot, wraps their arms around their body when they are cold, etc. Both studies indicated that the combined use of rule setting, role-play, and multiple exemplar training is an effective teaching strategy for teaching individuals with ASD to respond to various modalities of disguised mands. Both studies stressed the importance of future research to evaluate the effect that these teaching strategies have on the participant’s social environment. For instance, future studies could focus on teaching individuals to respond to disguised mands emitted by their peers or their parents.

See Also

- Mands
- Multiple Exemplar Training
- Verbal Behavior Interventions

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association.
- Erhard, P. (2019). *Teaching children with autism to respond to disguised mands and body language cues*. Symposium on Addressing the Needs of Children with Autism Across Social, Academic, and Behavioral Domains in the Annual Association for Behavior Analysis International Conference, Chicago, IL, May 2019.
- Najdowski, A. C., Bergstrom, R., Tarbox, J., & Clair, M. S. (2017). Teaching children with autism to respond to disguised mands. *Journal of Applied Behavior Analysis*, 50, 733–743. <https://doi.org/10.1002/jaba.413>.
- Skinner, B. F. (1957). *Century psychology series. Verbal behavior*. East Norwalk: Appleton-Century-Crofts.

Disinhibited Attachment Disorder

- Attachment Disorder

Disinhibited Social Engagement Disorder

- [Attachment Disorder](#)

Disintegrative Disorder

- [Acquired Autism](#)

Disorder of Executive Dysfunction

- [Frontal Lobe Syndrome](#)

Disordered Attachment

- [Reactive Attachment Disorder](#)

Disparities Among African Americans with Autism

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Historical Background

Autism spectrum disorder (ASD) is an umbrella term that includes a variety of specific neurodevelopmental impairments. These include Asperger syndrome, autistic disorder, and more. It is called a “spectrum” disorder because there is no single homogenous set of symptoms that present themselves across all cases. More accurately, ASD presents a series of symptoms that tend to manifest themselves at often vastly varying degrees from person to person. ASD is prevalent across all

racess, social classes, and geographic regions. According to the Centers for Disease Control and Prevention (CDC), common symptoms of ASD include problems with social, emotional, and communication behaviors. People with ASD tend to exhibit a strong affinity for routine and a severe distaste for sudden change. They also have trouble expressing their physical and emotional needs by way of conventional vectors of communication.

Throughout history, ASD has been severely mischaracterized and mistreated. Traditional approaches to ASD have simplified and misidentified the causes of ASD to simply bad parenting. This view persisted into the 1970s, and the subsequent developed approaches yielded more damage than harm. It was in the 1960s that the neurological nature of ASD was considered by Victor Lotter, and this was ultimately the spark that lit the convergence into a truly scientific approach to ASD (Lotter 1966). Lotter noticed a similar sociological manifestation between autism and other neurodevelopmental disorders and subsequently suggested that they shared a similar etiology. Researchers subsequently began to develop a category of distinct, but related disorders which they termed ASD.

Although there is significant research on ASD, understanding of the disorder as it relates to specific racial and ethnic groups is severely limited (Becerra et al. 2014). While there is a general consensus upon the similar prevalence of ASD across all demographic groups, there are other moving parts that affect other facets of ASD manifestation. Racial, ethnic, and class disparities in health literacy are well documented by various studies. One such study found that there remains a statistically significant disparity in health literacy between African Americans and Caucasians, and these disparities were more related to education and age rather than gender (Shea et al. 2004). These findings are devastating in a multifaceted manner, potentially affecting the outcome and treatment for nearly every prolific and rare medical problem across African American communities, as well as lifespans in potentially fatal scenarios. With this in mind, it is no surprise that the rate of disparity in excess death from chronic

diseases and other medical conditions between African Americans and Caucasians has increased from the 90s into the new millennium (Airhihenbuwa and Liburd 2006). These disparities are, no doubt, caused by a multitude of factors, including the socioeconomic marginalization of African American communities and the persistence of ghettos across America's larger cities. Studies have attributed an emotional causative pathway to certain pathophysiologies which play a hand in the rising health disparities (Carlson and Chamberlain 2004). At the most basic level, however, the problem begins with the failure to diagnose disorders.

Estimates indicate that ASD affects approximately 1 in 500 births every year. Studies have shown that rates of diagnosis for ASD between African American and Caucasian groups are not significantly different. There is a difference, however, in the stages at which ASD is diagnosed. Among African American groups, ASD tends to be diagnosed much later when compared to Caucasian groups (Gourdine et al. 2011). The implications of this are several, including later treatment, longer and more intensive intervention plans, increasing severity of untreated symptoms, and further psychological damage as a result of the patient's inability to be recognized as in need of extra care. This also poses the risk that the patient may never be diagnosed as autistic due to health illiteracy and lack of proper access to resources, which may result in significant difficulty later in life with the lack of proper accommodations in the educational system as well as discrimination in the work place without checks and balances by laws pertaining to disabled peoples.

Failure to diagnose ASD among other disorders has been attributed not only to socioeconomic factors such as literacy and access, but also to a higher prevalence of affinity towards religiosity and spiritual remedy. Various surveys and studies have indicated that significantly more African Americans identify as religious when compared with Caucasian populations. The ratio of religiosity can often present as 2:1 or more (Taylor et al. 1996). Misattributions of medical disorders to supernatural etiologies such as

demonic possession are not alien to the world of ASD. Religious scripture and preachers have often attributed mental disorders to demonic possession (Greydanus and Toledo-Pereyra 2012). Within African American communities, there are several major groups of churches that propagate supernatural access into the spiritual world as a way towards physical healing (Baer 1981). These churches belong to a wide array of denominations, including mainstream Baptists, Methodists, and Pentecostal churches among others. It is not surprising how influential religion remains as a factor in African Americans' health care perceptions and habits, given how prominent religion has been in African American history and cultural practice.

Overview of Current Research

By the late 1990s, Autism Spectrum Disorder (ASD) was diagnosed, on average, at 6 years of age, according to the findings of Dr. Patricia Howlin (Howlin and Moore 1997). Howlin's study spanned 1200 families with children having been diagnosed with ASD, or those in the process of receiving a diagnosis. The study did not emphasize ethnic disparities within the sample size, limiting the applicability of these findings to minority populations. The average age at which parents first became concerned with the child's mental development was 1.69 years of age (sd: 1.43, r: 0-18). Approximately 93% of parents had concerns by the time the child reached 3 years of age. A mere 10% were worried by 6 months of age, and in less than 3% of cases did the parents first become concerned after 5 years of age. Further, the average age at which parents first proceeded with professional assistance regarding their children's development was 2.3 years (sd: 1.94, r: 1 month to 38 years). Despite this, the average time that passed between parental concern about child development and the decision to seek professional help was 6-7 months, with over 23% of parents waiting up to 12 months.

Contemporarily, clinicians have successfully been able to detect ASD with reliable accuracy in children aged as young as 2-3 years old. The development of streamlined diagnostic

apparatuses such as the Autism Diagnostic Interview-Revised (ADI-R) and the Autism Diagnostic Observation Schedule-Generic (ADOS-G) have made this possible, but despite the technology, most children are not diagnosed before reaching 4 years of age (Couteur et al. 1989). The diagnosis tends to occur approximately 2 years after professional consultation is sought due to concern regarding the development of a child (Howlin and Moore 1997).

Unfortunately, many of these findings cannot be extended to African American populations, as the majority of recent research surrounding ASD has been almost exclusively limited to Caucasian populations. A study has found that African American patients that fall on the spectrum display more significant impediments in language development but typically yield the same degree of the other main symptoms of ASD (Cuccaro et al. 2007). Further analysis indicates that a higher social class marker is associated with greater chance of ASD (Bhasin and Schendel 2007). As it is understood that ASD occurs at a similar rate across all demographics, this statistic is indicative that a higher social class is more likely to result in a diagnosis of autism simply due to reasons of greater access to healthcare and higher health literacy. A large proportion of studies pertaining to non-Caucasian ASD patients focus heavily on the country of origin of the patients, which does not provide much in terms of etiology and disparities (Fombonne 2009). The CDC has reported that there is a higher frequency of ASD among Caucasian children as opposed to African American children in the majority of their studies. Additionally, Hispanic individuals with ASD were prevalent at a substantially lower rate when compared to Caucasian sample groups as well. A multisite case-control study of ASD yielded results that further indicate a lower rate of diagnosis among populations with less access to healthcare. Of a randomized population of 2768 children with ASD, approximately 33% of households had incomes below the United States median household income threshold (DiGuiseppi et al. 2016). Statistically, assuming that there is an equal proportion of ASD cases across all demographics, 50% of households should have

incomes below the United States median household income.

While it is thought that further studies will uncover factors that increase risk for ASD, the current corpus of research does not present much in that line of thought. Estimations of the frequency of ASD have been significantly increasing since the disorder was first characterized (Elsabbagh et al. 2012). Despite this, understanding of the etiological nature of ASD and potential factors that may increase the likelihood of its development is significantly limited (Newschaffer et al. 2007). The extent of recognition is that there is likely a deeply complex system of interplay between genetic, epigenetic, and environmental risk factors. Although studies are typically of low sample size along with a host of other clinical and infrastructural limitations, links between parental age and frequency of ASD have been suggested (Hultman et al. 2010). Despite these findings, the baseline operational fulcrum of thought is that ASD tends to occur at similar proportions across all demographics. Until conclusive studies are produced otherwise, attention must be paid to the disparities in the rate of diagnosis and onset of treatment between African American and Caucasian populations.

Future Directions

While it is recognized that individuals diagnosed with ASD are found in all regions, ethnic groups, cultural backgrounds, and socioeconomic levels, there has been inadequate attention given to ethnic disparities. While it is conceivable that unknown risk factors factor into the etiology of ASD and potentially present differences in the prevalence of the disorder among various populations, there is currently more work to be done prior to fixating research upon this facet of study. It is imperative that studies which properly measure the prevalence of ASD are fortified by a more accurate sampling, and such an accurate sampling is achievable by way of establishing a more reliable infrastructural pathway towards the diagnosis and treatment of ASD among African Americans and other minority populations.

There is no doubt that there exists a variety of confounding factors in the statistics regarding the proportionality of African American individuals with ASD. Many of these factors are cultural, while others are infrastructural. To overcome the infrastructural barriers towards diagnosing African American individuals with ASD, government policies must pave the road towards more accessible pathways to proper treatment. This may be manifested as an increase in resources focused on providing pediatric healthcare in low income urban areas and providing social workers trained to identify such disorders at an early age. Not only should these social workers be available to minorities living in low-income neighborhoods, but it is important that they also be financially accessible – perhaps by way of government subsidization. Disparities in the academic performance of children with ASD exist across ethnic lines, and this is a reality that may be dealt with by way of policy.

Confounding factors to do with cultural realities among African American populations should be addressed by way of social initiatives towards health literacy. So as to curb perception of the undermining of institutions such as churches that tend to emphasize alternative healing attempts, NGOs, and government initiatives should go about establishing relationships with these institutions to provide adequate health literacy. Health literacy workshops, blood drives, and health screenings are not unheard of in such institutions, and this is a potentially effective way to begin dampening these confounding variables.

More comprehensive study of the sociocultural influences that may delay and even altogether curb the effort to diagnose and identify ASD among African American populations are necessary. Studies into the magnitude of effect presented by disparities in health literacy and access to health are an excellent first step towards accomplishing this goal. The critical teleology of these studies, however, should be to generate accurate statistics to form the basis of research into the etiology of ASD. Of course, the aforementioned sociocultural research would assist in narrowing down proposed genetic, epigenetic, and lifestyle factors in the study of ASD etiology. Diagnostic techniques based upon behavior and

cognitive development may only go so far in the study of ASD. In the case of discovering a genetic etiology, ASD could potentially be pinpointed prior to birth, allowing for immediate medical attention and in increased threshold for development. A more holistic fundamentally focused approach will yield the most accurate data.

References and Reading

- Airhihenbuwa, C. O., & Liburd, L. (2006). Eliminating health disparities in the African American population: The Interface of culture, gender, and power. *Health Education & Behavior*, 33(4), 488–501. <https://doi.org/10.1177/1090198106287731>.
- Baer, H. A. (1981). Prophets and advisors in black spiritual churches: Therapy, palliative, or opiate? *Culture, Medicine and Psychiatry*, 5(2), 145–170. <https://doi.org/10.1007/bf00055418>.
- Becerra, T. A., et al. (2014). Autism Spectrum disorders and race, ethnicity, and nativity: A population-based study. *Pediatrics*, 134(1). <https://doi.org/10.1542/peds.2013-3928>.
- Bhasin, T. K., & Schendel, D. (2007). Sociodemographic risk factors for autism in a US metropolitan area. *Journal of Autism and Developmental Disorders*, 37(4), 667–677. <https://doi.org/10.1007/s10803-006-0194-y>.
- Carlson, E. D., & Chamberlain, R. M. (2004). The black-white perception gap and health disparities research. *Public Health Nursing*, 21(4), 372–379. <https://doi.org/10.1111/j.0737-1209.2004.21411.x>.
- Cuccaro, M. L., et al. (2007). Autism in African American families: Clinical-phenotypic findings. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*. <https://doi.org/10.1002/ajmg.b.30535>.
- Diguiseppi, C. G., et al. (2016). Demographic profile of families and children in the study to explore early development (SEED): Case-control study of autism Spectrum disorder. *Disability and Health Journal*, 9(3), 544–551. <https://doi.org/10.1016/j.dhjo.2016.01.005>.
- Elsabbagh, M., et al. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research*, 5(3), 160–179. <https://doi.org/10.1002/aur.239>.
- Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. *Pediatric Research*, 65(6), 591–598. <https://doi.org/10.1203/pdr.0b013e31819e7203>.
- Gourdine, R. M., et al. (2011). Autism and the African American community. *Social Work in Public Health*, 26(4), 454–470. <https://doi.org/10.1080/19371918.2011.579499>.
- Greydanus, D. E., & Toledo-Pereyra, L. H. (2012). Historical perspectives on autism: Its past record of discovery and its present state of solipsism, skepticism, and

- sorrowful suspicion. *Pediatric Clinics of North America*, 59(1), 1–11. <https://doi.org/10.1016/j.pcl.2011.10.004>.
- Howlin, P., & Moore, A. (1997). Diagnosis in Autism. *Autism*, 1(2), 135–162. <https://doi.org/10.1177/1362361397012003>.
- Hultman, C. M., et al. (2010). Advancing paternal age and risk of autism: New evidence from a population-based study and a meta-analysis of epidemiological studies. *Molecular Psychiatry*, 16(12), 1203–1212. <https://doi.org/10.1038/mp.2010.121>.
- Le Couteur, A., et al. (1989). Autism diagnostic interview: A standardized investigator-based instrument. *Journal of Autism and Developmental Disorders*, 19(3), 363–387. <https://doi.org/10.1007/bf02212936>.
- Lotter, V. (1966). Epidemiology of autistic conditions in young children. *Social Psychiatry*, 1(3), 124–135. <https://doi.org/10.1007/bf00584048>.
- Newschaffer, C. J., et al. (2007). The epidemiology of autism Spectrum disorders. *Annual Review of Public Health*, 28(1), 235–258. <https://doi.org/10.1146/annurev.publhealth.28.021406.144007>.
- Shea, J. A., et al. (2004). Assessing health literacy in African American and Caucasian adults: Disparities in rapid estimate of adult literacy in medicine (REALM) scores. *Family Medicine*, 36(8), 575–581.
- Taylor, R. J., et al. (1996). Black and white differences in religious participation: A multisample comparison. *Journal for the Scientific Study of Religion*, 35(4), 403. <https://doi.org/10.2307/1386415>.

Disparities in ASD Diagnosis

► Parental Action and Referral Patterns in Spatial Clusters of Childhood Autism Spectrum Disorders

Dispute Resolution Procedures

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Definition

A dispute resolution procedure is a method of resolving a conflict between parties. Historically,

dispute resolution was judicial in nature; however, in recent years the number of disputes resulting in trial has decreased significantly due in part to the advent of alternative dispute resolution (ADR) procedures such as arbitration, mediation, and negotiation. Today, most references to a dispute resolution procedure are to these alternatives to litigation (Yarn 1999, p. 154). These methods of dispute resolution along with other types of ADR may be utilized by parties trying to minimize costs and avoid the adversarial nature of litigation.

Types of Alternative Dispute Resolution Procedures

Modern alternative dispute resolution procedures were developed primarily as a response to the rising levels of litigation in the United States throughout the twentieth century. In the mid-1970s, Harvard Professor Frank Sander articulated his vision of a system where, instead of leading directly to litigation, disputes could be directed to various alternative methods to resolve the dispute without resorting to a trial. He described this multifaceted system of the judiciary working alongside other forms of conflict resolution as the “multidoor” courthouse. (Menkel-Meadow 2005, p. 19). The implementation of such alternatives to litigation has greatly reduced the number of cases going to trial even as the number of complaints filed in courthouses has greatly increased (Stipnowich 2004, p. 844).

Arbitration

Arbitration is a dispute resolution process in which the disputing parties present their cases to a third party intermediary who makes what is usually a binding decision for the parties. Arbitration is generally not as formal as in-court adjudication, and the procedural rules can be structured to meet the necessities of the particular situation. As in court-based adjudication, the outcome of an arbitration proceeding will typically result in a clear winner and loser. Although the arbitrator may recommend a solution that clearly benefits

both parties, arbitrators are not expected to develop ideas for meeting the interests of both sides or to help the parties see areas of agreement and reconciliation.

Mediation

Mediation is a process in which the parties attempt to resolve a conflict with the assistance of a neutral third party (mediator) (Yarn 1999, p. 272). Mediation is similar to negotiation in that the parties to the dispute are in control. However, to assist the process of negotiation, the mediator is present to help reach a mutually agreeable solution to their differences. Although ultimate control and decision-making authority remain with the parties, the mediator has significant power throughout the process to direct the negotiations, identify areas of agreement, and encourage the parties to accept concessions (Goldberg et al. 2007, p. 107).

Negotiation

Negotiation is a process where the parties, with the aid of a mediator, attempt to come to a mutually acceptable agreement about issues on which they disagree (Nieuwmeijer 1988, p. 9). The parties use bargaining and open communication to reach a consensus over their outstanding issues. Negotiation works best in situations where all parties involved in the process have a mutual interest in resolving their dispute and are willing to each make concessions in order to reach an acceptable resolution.

See Also

► [Eligibility \(for Services Under IDEA/ADA, etc.\)](#)

References and Reading

Garner, B. A. (Ed.). (2004). *Black's law dictionary*. Tampa: West Group Publishing.

- Goldberg, S. B., Sander, F. E. A., Rogers, N. H., & Cole, S. R. (2007). *Dispute resolution: Negotiation, mediation, and other processes* (5th ed.). New York: Aspen.
- Menkel-Meadow. (2005). Roots and inspirations: A brief history of the foundations of dispute resolution. In M. Moffitt & R. Bordone (Eds.), *The handbook of dispute resolution* (pp. 13–33). San Francisco: Jossey-Bass.
- Nieuwmeijer, L. (1988). *Negotiation: Methodology and training*. Preoria: HSRC.
- Stipnowich, T. J. (2004). ADR and the “Vanishing Trial”: The growth and impact of “alternative dispute resolution”. *Journal of Empirical Legal Studies*, 1(3), 843–912.
- Yarn, D. H. (1999). *Dictionary of conflict resolution*. San Francisco: Jossey-Bass.

Disruptive Behavior

► [Conduct Disorder](#)

Dissocial Behavior

► [Conduct Disorder](#)

Distributed Practice

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Definition

Distributed practice is a technique commonly used with students who are learning material or studying for a test. It involves the student creating a schedule of study sessions that are short in duration. Distributed practice can be contrasted to massed practice or cramming, where the student spends fewer studying sessions but for longer periods of time. This technique has proven to be beneficial for maintaining newly learned skills. Distributed practice aids students in prioritizing learning material.

References and Reading

http://psychology.wikia.com/wiki/distributed_practice
<http://web.ics.purdue.edu/~rallrich/learn/dist.html>

Murray, S. R., & Udermann, B. E. (2003). Massed versus distributed practice: Which is better? *CAHPERD Journal*, 28, 19–22.

Distribution-Free Statistics

► [Nonparametric Statistics](#)

Divalproex

► [Depakene](#)

Divalproex Sodium (Depakote, Depakote ER/CP)

► [Valproate](#)

Dizygotic (DZ) Twins

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Synonyms

[Fraternal twins](#)

Definition

Twins are two individuals who are the result of the same pregnancy. Dizygotic twins are nonidentical or fraternal twins. This is in contrast to identical or monozygotic twins. Unlike the case in monozygotic twins, the genetic information or deoxyribonucleic acid (DNA) carried by each of

the individual twins in a pair of dizygotic twins is different. This genetic information in the form of DNA is the material inherited from each of the parents that contains all the instructions for the creation and subsequent operation of an individual. Errors in this genetic information can lead directly to disease or make individuals more susceptible to disease.

In singleton pregnancies, a sperm from the father fuses with an egg from the mother, and together, they form a single cell called a zygote, the earliest stage of an embryo. In the case of dizygotic twins, two separate eggs in the mother are released at one time. Each of these is then fertilized by a separate sperm from the father and forms a distinct zygote and embryo. Since each sperm and egg carry distinct genetic material, each of the two embryos will, thus, carry distinct genetic material. On average, dizygotic twins' genetic material is only about 50% identical, as compared to 100% for monozygotic twins. Since each dizygotic twin carries distinct genetic material, his or her physical appearance will be distinct. Dizygotic twins may be of the same sex. However, they might also be of different sexes.

Monozygotic twins, in contrast, form as follows. A sperm from the father fuses with an egg from the mother and initially forms a single zygote. Further cell divisions occur during embryonic development. At an early point in this development, the embryo in the case of monozygotic twins splits into two separate embryos, the cells of each having originated from the initial zygote. Each of these embryos goes on to develop into a separate and complete individual. Since each originated from the same initial cell, each individual is identical in his or her genetic composition. Barring rare occurrences (see ► [Monozygotic \(MZ\) Twins](#)), since monozygotic twins share the same genetic material, their physical appearance will be identical, and they will be of the same sex.

Dizygotic twinning is more common than monozygotic twinning. Dizygotic twins comprise approximately two thirds of all twins. Monozygotic twins comprise one third. While the rate of monozygotic twinning in pregnancies that are

unassisted by fertility treatments is relatively stable across world populations at a rate of approximately 4 in every 1,000 live births, there is evidence that the rate of dizygotic twinning in the absence of fertility treatments varies from population to population. Studies estimate that dizygotic twinning rates are the lowest in East Asian countries fewer than 8 per 1,000 live births. Dizygotic twinning rates are intermediate in Europe, the United States, and India with a rate of approximately 9–16 per 1,000 births. They are highest in some African countries where they can be 18 or greater per 1,000 births.

The rate of both monozygotic and dizygotic twins has increased worldwide since the 1970s. It is thought that the majority of the increase has resulted from the increase in dizygotic twins born as a result of fertility treatments. Twin pregnancies of either type increase pregnancy risk and especially the risk of preterm delivery and low birth weight. Approximately 51% of twins are born preterm compared to 9.4% of singletons.

See Also

- [Genetics](#)
- [Monozygotic \(MZ\) Twins](#)
- [Twin Studies in Autism](#)

References and Reading

- Alexander, G. R., Kogan, M., et al. (1998). What are the fetal growth patterns of singletons, twins, and triplets in the United States? *Clinical Obstetrics and Gynecology*, 41(1), 115.
- Bortolus, R., Parazzini, F., et al. (1999). The epidemiology of multiple births. *Human Reproduction Update*, 5(2), 179.
- Gilbert, S. F. (1994). *Developmental biology*. Sunderland: Sinauer Associates.
- Hall, J. G. (2003). Twinning. *The Lancet*, 362(9385), 735–743.
- Machin, G. A. (1996). Some causes of genotypic and phenotypic discordance in monozygotic twin pairs. *American Journal of Medical Genetics*, 61(3), 216–228.
- Reeve, E. C. R., & Black, I. (2001). *Encyclopedia of genetics*. London: Routledge.
- Smits, J., & Monden, C. (2011). Twinning across the developing world. *PLoS One*, 6(9), e25239.
- Strachan, T., & Read, A. P. (2004). *Human molecular genetics*. New York: Garland Press.
- Vitthala, S., Gelbaya, T. A., et al. (2009). The risk of monozygotic twins after assisted reproductive technology: A systematic review and meta-analysis. *Human Reproduction Update*, 15(1), 45.

D-KEFS

- [Introduction to the Delis Kaplan Executive Function System](#)

DMG

- [Dimethylglycine](#)

DNA

- [Deoxyribonucleic Acid](#)

Domenic Cicchetti

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Biography

Dr. Domenic Vincent Cicchetti, PhD (1937–2019), was a biostatistician most notably known for the development of the Vineland Adaptive Behavior Scales (VABS) together with his late wife Sara Sparrow, a renowned clinical psychologist at the Yale Child Study Center.

Dr. Cicchetti obtained his BA, MA, and PhD between 1955 and 1965 in social psychology and

biostatistics at the University of Connecticut. He later obtained positions as a Senior Research Scientist at the Yale School of Medicine in the Child Study Center and Department of Psychiatry and School of Epidemiology and Public Health. He was an adjunct professor in the Department of Psychology at the University of Windsor in Ontario, Canada, and served as a Visiting Professor in the Division of Neuroscience & Psychological Medicine, Department of Public Mental Health, Imperial College of Science, Technology, and Medicine, St. Mary's Campus, London, England (Øien et al. 2019).

As mentioned, Dr. Cicchetti was most notably known for his work together with his wife Sara Sparrow in the development of the VABS and its later editions (Sparrow et al. 2005). But in addition to the development of the Vineland, Dr. Cicchetti authored or co-authored over 200 research publications in behavioral and biomedical research, computer science, and biostatistics that have been widely cited and been essential to the field of developmental psychology and disabilities. The *Vineland* would become the most widely used adaptive behavior assessment instrument in the world. For the development and publication of the *Vineland*, Dr. Cicchetti received the first Connecticut Psychological Association's Award for Distinguished Effort by a Connecticut Psychologist in 1984 together with his wife Sara Sparrow. Dom was a fellow in Division 5 of the American Statistical Association. Dr. Cicchetti was widely known for his development of methods to assess the psychometric properties and work on statistical methodologies in regard to validity and reliability. Dr. Cicchetti has, per May 2020, still scientific contributions in the peer review process.

To summarize, Dr. Domenic Vincent Cicchetti was a prolific scientist in the field of autism and related disabilities, developing instruments and methods that ultimately have led to the fact that an enormous amount of individuals globally have received services for their disability. Dr. Cicchetti also contributed massively to the understanding of statistical versus clinical meaningful results (Nordahl-hansen et al. 2018). Dr. Cicchetti deceased on June 30, 2019.

See Also

► Sparrow, Sara

References and Reading

- Nordahl-Hansen, A., Øien, R., Volkmar, F., Shic, F., & Cicchetti, D. (2018). Enhancing the understanding of clinically meaningful results: A clinical research perspective. *Psychiatry Research*, 270, 801–806. <https://doi.org/10.1016/j.psychres.2018.10.069>. *Psychological Bulletin*, 66 1966.
- Øien, R. A., Klin, A., Saulnier, C., et al. (2019). In Memoriam: Domenic V. Cicchetti, PhD. 1937–2019. *Journal of Autism and Developmental Disorders*, 49, 3475–3476. <https://doi.org/10.1007/s10803-019-04143-5>.
- Sparrow, S. S., Cicchetti, D., & Balla, D. A. (1984). *Vineland adaptive behavior scales*.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland Adaptive Behavior Scales Vineland-II: Survey Forms Manual*. Minneapolis, MN: Pearson.

Dominance, Cerebral

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Synonyms

Hemispheric dominance; Hemispheric lateralization; Hemispheric specialization

Definition

Cerebral dominance refers to the dominance of one cerebral hemisphere (commonly referred to as the left or right side of the brain) over the other in the control of particular cerebral functions. After decades of study in the fields of behavioral neurology, systems neuroscience, neuroimaging, and neuropsychology, it is clear that each hemisphere of the brain is dominant for specific behavioral and cognitive functions. For example, in most right-handed individuals, portions of the right hemisphere temporal lobe are specialized

for processing faces, and similar regions of the left hemisphere temporal lobe are specialized for processing letters. Similarly, in approximately 95% of right-handers and 65% of left-handers, the left side of the brain is dominant for language.

See Also

- [Cerebral Cortex](#)
- [Neuroscience](#)

References and Reading

Toga, A. W., & Thompson, P. M. (2003). Mapping brain asymmetry. *Nature Reviews Neuroscience*, 4(1), 37–48.

DONEP

- [Donepezil: Definition](#)

Donepezil: Definition

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Synonyms

[Aricept](#); [DONEP](#)

Definition

Donepezil is a central nervous system selective, reversible acetylcholinesterase inhibitor. Acetylcholinesterase is an enzyme that degrades acetylcholine (ACh), a neurotransmitter involved in the

pathogenesis of autism-related behavioral deficits such as inattention, decreased cognitive flexibility, lack of social and communication abilities, and increased stereotypical behaviors. Thus, donepezil serves to increase levels and action duration of ACh in the hopes of reversing abnormal phenotypes caused by deficits in ACh production and/or receptor function. Currently, donepezil is most widely used in the treatment of Alzheimer's disease. Its effectiveness has also been demonstrated in vascular and Parkinson's-associated dementia. In ASD, donepezil has demonstrated improvements mainly in expressive language but also in terms of general behavior, i.e., irritability, hyperactivity, eye contact, and inappropriate speech. Evidence also suggests donepezil's potential use in improving REM sleep duration in ASD patients. However, with all these observations further, substantial, larger-scale validation is required.

See Also

- [Anticholinesterase Inhibitors](#)

References and Reading

- Chez, M. G., Buchanan, T. M., Becker, M., Kessler, J., Aimonovitch, M. C., & Mrazek, S. R. (2003). Donepezil hydrochloride: A double-blind study in autistic children. *Journal of Pediatric Neurology*, 1(2), 83–88.
- Doyle, R. L., Frazier, J., Spencer, T. J., Geller, D., Biederman, J., & Wilens, T. (2006). Donepezil in the treatment of ADHD-Like symptoms in youths with pervasive developmental disorder a case series. *Journal of Attention Disorders*, 9(3), 543–549.
- Handen, B. L., Johnson, C. R., McAuliffe-Bellin, S., Murray, P. J., & Hardan, A. Y. (2011). Safety and efficacy of donepezil in children and adolescents with autism: Neuropsychological measures. *Journal of Child and Adolescent Psychopharmacology*, 21(1), 43–50.
- Hardan, A. Y., & Handen, B. L. (2002). A retrospective open trial of adjunctive donepezil in children and adolescents with autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, 12(3), 237–241.
- Yoo, J. H., Valdovinos, M. G., & Williams, D. C. (2007). Relevance of donepezil in enhancing learning and memory in special populations: A review of the literature. *Journal of Autism and Developmental Disorders*, 37(10), 1883–1901.

Dopamine

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Definition

Dopamine is a neurotransmitter that is implicated in the pathophysiology of many psychiatric and neurologic disorders. Its most notable psychiatric role is in the pathophysiology of psychosis and schizophrenia, particularly the presence of hallucinations and delusions. However, among a complicated network of neural pathways, dopamine is also believed to influence mood states, anxiety, cognition, and the presence of repetitive symptoms experienced in conditions like autism spectrum disorders (ASD), Tourette's disorder, and obsessive-compulsive disorder. For these reasons, dopamine is the target of research attempting to uncover etiologies and treatments for such diseases. Understanding dopamine's relationship to ASD may offer much insight into the pathophysiology of its symptoms.

Dopamine is synthesized in specialized neurons using the amino acid precursor tyrosine (Stahl 2008). Tyrosine is first pumped from the extracellular space into dopaminergic neurons by a tyrosine transporter. Within the neuron, tyrosine then passes through the rate-limiting enzyme tyrosine hydroxylase, followed by the enzyme dopa decarboxylase, to become dopamine. (Dopamine can also be converted to the neurotransmitter norepinephrine via the enzyme dopamine beta-hydroxylase.) Dopamine is packaged into vesicles by a vesicular monoamine transporter (VMAT2) for storage until later use. When a neuron receives the

appropriate signal, dopamine is released from synaptic vesicles to travel across the cleft between the presynaptic and postsynaptic axon terminals. Once in the cleft, dopamine is free to attach to dopamine receptors on the postsynaptic axon terminal. It can also be taken up by dopamine transporters in the presynaptic axon terminal to be repackaged for later use or degraded. One of the most notable receptors is the dopamine-2 (D₂) receptor, which is located on postsynaptic axon terminals, presynaptic axon terminals, and somatodendritic areas. When dopamine attaches to D₂ receptors on the presynaptic axon terminal or somatodendritic areas, D₂ receptors provide negative feedback that slows or further prevents the release of dopamine from the presynaptic terminal. Excess dopamine is degraded within the neuron by the enzymes monoamine oxidase (MAO)-A or MAO-B and outside the neuron by the enzyme catechol-O-methyltransferase (COMT). In some areas in the brain, such as the frontal cortex, there are fewer dopamine transporters to take up excess dopamine remaining in the cleft, so these alternative routes of degradation function to regulate dopamine concentration.

There are five key dopamine pathways in the brain. The first is the mesolimbic pathway, which projects from the dopaminergic cell bodies of the ventral tegmental area of the brainstem to the nucleus accumbens in the ventral striatum. Increased dopamine activity in this pathway is thought to generate psychosis, also known as the "positive symptoms" of schizophrenia, which include hallucinations and delusions. Stimulant drugs, like amphetamine and cocaine, produce increased dopaminergic activity and subsequent psychotic symptoms, whereas first- and second-generation antipsychotic medications, which antagonize dopamine in this pathway, cause reduced psychotic symptoms. The mesolimbic pathway is also known to regulate emotions, motivation, pleasure, and reward. Dysfunction in this area may result in symptoms such as avolition and anhedonia, accounting for some of the "negative symptoms" of schizophrenia. The second pathway is the mesocortical pathway, which projects from the dopaminergic cell bodies of the ventral

tegmental area to the prefrontal cortex. Branches from this pathway are believed to regulate cognition and executive function, as well as emotion and affect. Unlike the mesolimbic pathway, where an excess of dopamine is hypothesized to produce symptoms of psychosis, a deficit of dopamine in the mesocortical pathway is thought to cause more negative symptoms observed in schizophrenia, such as flat affect, reduced cognition, and impaired executive function. The model of increased or decreased dopaminergic activity in different pathways is hypothetical and is likely an oversimplification of a more complex system yet to be understood. The third pathway is the nigrostriatal pathway, which projects from the dopaminergic cell bodies in the brainstem substantia nigra to the basal ganglia or striatum. This area regulates motor movements and is part of the extrapyramidal nervous system. Hypoactivity of dopamine in this pathway produces parkinsonian symptoms of rigidity, akinesia or bradykinesia, and tremor. Hypoactivity in the basal ganglia specifically can result in dystonia or akathisia. Hyperactivity of dopamine in this pathway results in hyperkinetic movements, such as tics, chorea, and dyskinesia. Longer term blockade of the D₂ receptors via antipsychotics can produce a hyperkinetic disorder known as tardive dyskinesia. The fourth pathway is the tuberoinfundibular pathway, which projects from the hypothalamus to the anterior pituitary. Dopamine typically inhibits prolactin, a hormone that results in lactation. When dopaminergic activity is blocked in this pathway, prolactin levels rise as it is no longer inhibited. Elevated prolactin can cause galactorrhea (breast secretions), amenorrhea (loss of ovulation and menstruation), and possibly sexual side effects. This can occur with the use of antipsychotic medication, which blocks D₂ receptors. The fifth pathway is the lesser-known thalamic dopamine pathway, which innervates the thalamus. It is thought to originate in multiple sites, including the periaqueductal gray matter, ventral mesencephalon, hypothalamic nuclei, and lateral parabrachial nucleus. The function of this pathway may involve regulation of sleep and arousal.

Historical Background

In the late 1950s, a Swedish pharmacologist named Arvid Carlsson was the first person to discover dopamine as a distinct neurotransmitter, and in the year 2000, he won the shared Nobel Prize in Physiology and Medicine for this very significant contribution. As outlined in Abbott's article in *Nature* (2007), this monumental discovery occurred while experimenting with reserpine, the first antipsychotic to be used in the treatment of schizophrenia. Treatment with reserpine was observed to cause a catatonic state in experimental rabbits, but the mechanism by which this happened was unknown. Using a spectrophotofluorimeter, a machine used to measure the amount of neurotransmitter synthesized from fluorescently tagged precursors, Dr. Carlsson determined that reserpine somehow drained stores of brain neurotransmitters. Because the known neurotransmitters serotonin and norepinephrine were not able to cross the blood-brain barrier, Dr. Carlsson hypothesized that their precursors could be injected and cross over the blood-brain barrier to be converted to the needed neurotransmitters, hopefully restoring movement. He extracted serotonin and norepinephrine precursors, one of which was l-dopa (levodopa), and injected them into the catatonic rabbits. l-dopa restored movement in the animals, and Dr. Carlsson determined dopamine to be a separate neurotransmitter while examining the rabbits' postmortem brains.

With the help of his graduate students, Dr. Carlsson went on to discover dopamine concentrated in areas of the brain associated with movement, like the basal ganglia. Given the similarities between reserpine's side effects and Parkinson's disease, he hypothesized that the disease must be caused by a deficiency of dopamine. He brought these ideas to various symposia but received a mixed reception; the favored thinking at that time was that nerve conduction in the brain occurred via electrical impulses, with little emphasis on chemical transmission. Slowly, others began uncovering similar results, publishing studies showing an absence of dopamine in the basal ganglia in patients with Parkinson's

disease (Ehringer and Hornykiewicz 1960) and that healthy basal ganglia neurons contain high levels of dopamine (Birkmayer and Hornykiewicz 1961; Dahlstrom and Fuxe 1964). The drug is not without imperfection, and unwanted side effects such as nausea and emesis can occur. At times, the drug can lose its therapeutic effect in some patients. Nonetheless, l-dopa continues to be the first-line treatment for Parkinson's disease. It also likely garnered increased public awareness after being featured in the 1973 book *Awakenings* by British neurologist Oliver Sacks. This memoir recounts Dr. Sacks' use of l-dopa in 1969 to treat patients with catatonia who survived the 1917–1928 outbreak of *encephalitis lethargica*, otherwise known as sleeping sickness. The book was transformed into a 1990 film with the same name starring American actors Robin Williams and Robert De Niro. In 1961, l-dopa was injected into the first Parkinson's patients with dramatic effect, providing relief for their rigidity and immobility.

Not long after the discovery of dopamine's relationship to Parkinson's disease, neurologists observed that treatment with l-dopa resulted in psychosis, leading to the discovery that the pathophysiology of schizophrenia may be related to dopamine. The observation that antipsychotic drugs caused movement disorders similar to that observed in Parkinson's disease leads Dr. Carlsson to reason that antipsychotics blocked dopamine receptors, resulting in a feedback mechanism by which neurons released more compensatory dopamine. These conclusions have led Dr. Carlsson to become a pivotal figure in the discovery of dopamine as a distinct neurotransmitter, as well as someone who uncovered fundamental mechanisms in neurotransmission that continue to be employed today.

With time, neural pathways controlling dopamine were thought to influence motivation and reward, exemplified by the tendency of patients treated with l-dopa to gamble excessively. Research into addiction and drugs of abuse has also implicated brain regions and neural pathways primarily governed by dopamine. Dopamine's widespread effect in the brain has led autism researchers to investigate it in the

pathophysiology of ASD. The relationship between dopamine and ASD is explored in the “Current Knowledge” section.

Current Knowledge

Due to its extensive innervation of the brain, dopamine has been hypothesized to be involved in the pathophysiology of ASD. According to Baskerville and Douglas (2010), neurologic behavioral disorders caused by profound disruption of the key dopaminergic pathways in the brain are known to adversely affect prosocial behavior. These pathways likely involve complex interactions with multiple other neurotransmitters and neuropeptides, and it is hypothesized that dysfunctional interactions result in the aberrant social behavior observed in ASD. One of the proposed interactions is between dopamine and oxytocin, a neuropeptide with physiologic and behavioral influences. Oxytocin is an endocrine hormone that regulates parturition and milk ejection, but is also involved in regulating social behaviors such as social bonding, parental behavior, and sexual behavior (Lee et al. 2009). It is also thought to regulate nonsocial behaviors such as stress, anxiety, and aggression, which all appear in ASD, and has been thought to be influenced by dopamine pathways. Given their shared roles, dopamine and oxytocin are hypothesized to jointly contribute to aberrant social behaviors, anxiety, and aggression in ASD. The results of studies linking oxytocin to the pathophysiology of ASD have been mixed, however, but treatments involving intranasal oxytocin have resulted in improvements in communication and secure relationship attachment (Heinrichs et al. 2009; Kosfeld et al. 2005). Unfortunately, revealing a definitive, symbiotic relationship between oxytocin and dopamine has yet to occur, but research in this area continues. There has been stronger evidence linking dopamine dysfunction with autism-like disorders, particularly involving the dopamine transporter and D₄ receptor genes (Baskerville and Douglas 2010). Nakamura et al. (2010) used positron emission tomography (PET) to study the binding of dopamine and serotonin

transporters in the brains of autistic male adults compared to age- and IQ-matched controls. They found that dopamine transporter binding was significantly higher in the orbitofrontal cortex of the autistic individuals and that this was inversely correlated with serotonin binding, which was lower throughout the brain of autistic individuals. They concluded that dysfunction in dopamine and serotonin systems likely contributes to the pathophysiology of autism.

Research has attempted to link symptoms of ASD to specific genetic polymorphisms and neuroanatomical regions of the brain. For example, the presence of repetitive, stereotyped behaviors in ASD has been associated with the basal ganglia and frontal lobe circuitry (Langen et al. 2011), which is believed to be regulated by the dopamine system. Housed in the basal ganglia is the caudate nucleus, a brain region associated with behavioral rigidity. The caudate nucleus has been found to be enlarged in ASD, representing one of the best replicated neuroimaging findings in ASD research (Langen et al. 2009). The basal ganglia, particularly the caudate nucleus, exhibit high expression of the dopamine-3 receptor gene (DRD3), a gene that appears to be associated with ASD. Genetic studies have revealed an association between the SNP (single nucleotide polymorphism) rs16777 on DRD3 and ASD (de Krom et al. 2009). This polymorphism is also related to risperidone-induced extrapyramidal symptoms (Gasso et al. 2009), which is significant because risperidone and aripiprazole (a partial agonist at the D₂ receptor) are the only FDA-approved drugs for the treatment of symptoms associated with ASD (Staal et al. 2011). The DRD3 receptor site is also an action site for atypical antipsychotic drugs, and another of its polymorphisms is a predictor for improvement with risperidone therapy (Correia et al. 2010). All of this data supports a positive association between the presence of the DRD3 gene and ASD; however, a small study performed by Staal et al. (2011) revealed that the presence of SNP rs16777 on DRD3 paradoxically decreased the risk for “insistence on sameness,” a form of stereotyped behavior observed in ASD. Given the breadth of findings, some which are

conflicting, there is a need for continued research into DRD3 and its relationship to symptoms of ASD.

Other genetic biomarkers of interest include dopamine transporter gene (DAT1) and dopamine D4 receptor gene (DRD4). In a study by Gadow et al. (2010), the DAT1 and DRD4 genotypes approached significance for teachers’ ratings of oppositional behavior and mothers’ ratings of tics. The researchers proposed that variation in the alleles for DRD4 may serve as biomarkers predicting challenging behaviors in children with ASD, but the study was small and would require replication with larger samples.

Despite these attempts at localizing specific genes implicated in the pathophysiology of autism, some research indicates that it may not be so simple. A study genotyping 28 SNPs of 14 prominent dopamine pathway candidate genes concluded that the evidence was not strong in favor of linkage or association to any specific gene or combination of genes within the pathway (Anderson et al. 2008). The role of genes within the dopamine pathway, if any, was considered mild to moderate in the pathogenesis of autism.

Inattention and hyperactivity, symptoms that commonly occur in autism, also appear to result from dysfunction of the dopamine system. Gadow et al. (2008) found that a variable number tandem repeat (VNTR) in a region of the dopamine transporter gene (DAT1, SLC6A3) was associated with the severity of ADHD, anxiety, and tics in children with ASD.

The association between dopamine and autism is also evident via the observed effects of antipsychotics medication on the treatment of irritability in ASD. Approximately 30% of children and adolescents with ASD suffer from moderate-to-severe irritability (Lecavalier 2006). Irritability can include aggressive acts towards the self (self-injurious behavior) or others and severe tantrums. Currently, the most effective drugs used to treat symptoms of irritability are the antipsychotics, which are believed to antagonize postsynaptic D₂ receptors in the brain, although some may also have serotonin receptor antagonism. At present, there are only two atypical antipsychotics

that are FDA-approved for the treatment of irritability in autistic disorder: risperidone (Risperdal) and aripiprazole (Abilify). Risperidone is FDA-approved for the treatment of irritability in patients with autistic disorder aged 5–16 years, whereas aripiprazole is approved for the treatment of patients with autistic disorder aged 6–17 years. Risperidone is a potent D_2 antagonist, whereas aripiprazole has partial D_2 receptor agonists, meaning it detaches from the D_2 receptor more readily than risperidone and typical antipsychotics. These traits may contribute to these drugs' relative success in managing symptoms observed in ASD. Other antipsychotics that have been studied include the “typicals,” such as haloperidol, pimozide, chlorpromazine, trifluoperazine, thiothixene, trifluoperidol, fluphenazine, and molindone. Other atypical antipsychotics include clozapine, olanzapine, quetiapine, and ziprasidone. In addition to irritability, some studies have noted reductions in other symptom domains such as repetitive behavior and inattention, hyperactivity, and impulsivity. However, the direct relationship between dopamine and these symptom outcomes is not always apparent, so future research is needed to explicate this.

Future Directions

Future directions for research into the relationship between dopamine and autism will likely involve genetic studies, neuroendocrine research, neuroimaging, and pharmacologic treatment development. Genetic research has focused on identifying polymorphisms that may or may not be associated with symptoms of ASD. This type of research has yielded mixed results but will likely continue to examine the association between ASD and dopamine receptor genes like *DRD3* and *DRD4*. Attempting to link dopamine and oxytocin has also not yielded strong results, but their shared roles with regard to social behavior, stress, anxiety, and aggression will likely spur continued research attempting to find an association. Given the consistent finding of an enlarged caudate nucleus in patients with ASD, a dopamine-rich

area of the brain, neuroimaging may continue to yield useful information about other brain regions that utilize dopamine and how they relate to symptoms of ASD. It may also reveal more about dopamine binding in such areas. Lastly, new pharmacological treatments for symptoms of ASD should be investigated via randomized, double-blind, placebo-controlled studies to ensure a variety of safe and efficacious treatments are available to patients. The atypical antipsychotic paliperidone, which is the active metabolite of risperidone, is an evident choice since risperidone has shown efficacy in treating children and adolescents with autistic disorder (Stigler et al. 2010).

See Also

- ▶ [Antipsychotics: Drugs](#)
- ▶ [Aripiprazole](#)
- ▶ [Atypical Antipsychotics](#)
- ▶ [Caudate Nucleus](#)
- ▶ [Clozapine](#)
- ▶ [Obsessive-Compulsive Disorder \(OCD\)](#)
- ▶ [Olanzapine](#)
- ▶ [Pimozide](#)
- ▶ [Psychosis](#)
- ▶ [Quetiapine](#)
- ▶ [Risperidone](#)
- ▶ [Tourette Syndrome](#)
- ▶ [Ziprasidone](#)

References and Reading

- Abbott, A. (2007). Neuroscience: The molecular wake-up call. *Nature*, 447(7143), 368–370.
- Anderson, B. M., Schnetz-Boutaud, N., et al. (2008). Examination of association to autism of common genetic variation in genes related to dopamine. *Autism Research*, 1(6), 364–369.
- Baskerville, T. A., & Douglas, A. J. (2010). Dopamine and oxytocin interactions underlying behaviors: Potential contributions to behavioral disorders. *CNS Neuroscience and Therapeutics*, 16(3), e92–e123.
- Birkmayer, W., & Hornykiewicz, O. (1961). The L-3,4-dioxyphenylalanine (DOPA)-effect in Parkinson-akinesia. *Wiener Klinische Wochenschrift*, 73, 787–788.

- Correia, C. T., Almeida, J. P., et al. (2010). Pharmacogenetics of risperidone therapy in autism: Association analysis of eight candidate genes with drug efficacy and adverse drug reactions. *The Pharmacogenomics Journal*, 10(5), 418–430.
- Dahlstrom, A., & Fuxe, K. (1964). Localization of monoamines in the lower brain stem. *Experientia*, 20(7), 398–399.
- de Krom, M., Staal, W. G., et al. (2009). A common variant in DRD3 receptor is associated with autism spectrum disorder. *Biological Psychiatry*, 65(7), 625–630.
- Ehringer, H., & Hornykiewicz, O. (1960). Distribution of noradrenaline and dopamine (3-hydroxytyramine) in the human brain and their behavior in diseases of the extrapyramidal system. *Klinische Wochenschrift*, 38, 1236–1239.
- Emanuele, E., Boso, M., et al. (2010). Increased dopamine DRD4 receptor mRNA expression in lymphocytes of musicians and autistic individuals: Bridging the music-autism connection. *Neuro Endocrinology Letters*, 31(1), 122–125.
- Gadow, K. D., Roohi, J., et al. (2008). Association of ADHD, tics, and anxiety with dopamine transporter (DAT1) genotype in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 49(12), 1331–1338.
- Gadow, K. D., Devincent, C. J., et al. (2010). Association of DRD4 polymorphism with severity of oppositional defiant disorder, separation anxiety disorder and repetitive behaviors in children with autism spectrum disorder. *European Journal of Neuroscience*, 32(6), 1058–1065.
- Gasso, P., Mas, S., et al. (2009). A common variant in DRD3 gene is associated with risperidone-induced extrapyramidal symptoms. *The Pharmacogenomics Journal*, 9(6), 404–410.
- Heinrichs, M., von Dawans, B., et al. (2009). Oxytocin, vasopressin, and human social behavior. *Frontiers in Neuroendocrinology*, 30(4), 548–557.
- Kosfeld, M., Heinrichs, M., et al. (2005). Oxytocin increases trust in humans. *Nature*, 435(7042), 673–676.
- Langen, M., Schnack, H. G., et al. (2009). Changes in the developmental trajectories of striatum in autism. *Biological Psychiatry*, 66(4), 327–333.
- Langen, M., Leemans, A., Johnston, P., Ecker, C., Daly, E., Murphy, C. M., et al. (2011). Fronto-striatal circuitry and inhibitory control in autism: Findings from diffusion tensor imaging tractography. *Cortex*, 48, 183–193.
- Lecavalier, L. (2006). Behavioral and emotional problems in young people with pervasive developmental disorders: Relative prevalence, effects of subject characteristics, and empirical classification. *Journal of Autism and Developmental Disorders*, 36(8), 1101–1114.
- Lee, H. J., Macbeth, A. H., et al. (2009). Oxytocin: The great facilitator of life. *Progress in Neurobiology*, 88(2), 127–151.
- Nakamura, K., Sekine, Y., et al. (2010). Brain serotonin and dopamine transporter bindings in adults with high-functioning autism. *Archives of General Psychiatry*, 67(1), 59–68.
- Neuhaus, E., Beauchaine, T. P., et al. (2010). Neurobiological correlates of social functioning in autism. *Clinical Psychology Review*, 30(6), 733–748.
- Sacks, O. Awakenings (E.P. Dutton, 1973).
- Staal, W. G., de Krom, M., & de Jonge, M. V. (2011). Brief report: The dopamine-3-receptor gene (DRD3) is associated with specific repetitive behavior in autism spectrum disorder (ASD). *The Journal of Autism and Developmental Disorders*, 42(5), 885–888.
- Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd ed., pp. 102–105) (pp. 266–279). New York: Cambridge University Press.
- Stigler, K. A., Erickson, C. A., et al. (2010). Paliperidone for irritability in autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, 20(1), 75–78.

Dormicum

► Midazolam

Dorsal Medial-Frontal Cortex

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Definition

Dorsomedial frontal cortex (DMFC) is a region of the prefrontal cortex associated with interpreting social situations, specifically the ability to comprehend other's thoughts and intentions known as Theory of Mind. Functional imaging studies have identified reduced activity in DMFC during social decision tasks in ASD patients compared to neurotypical controls. Neuroanatomical studies have identified changes in organization of neurons in the DMFC in ASD patients, specifically Brodmann's area 9.

See Also

- ▶ Prefrontal Cortex
- ▶ Social Cognition
- ▶ Theory of Mind

Reference and Reading

- Amodio, D. M., & Frith, C. D. (2006). Meeting of minds: the medial frontal cortex and social cognition. *Nature Reviews. Neuroscience*, 7, 268–277.
- Ferrari, C., Lega, C., Vernice, M., Tamietto, M., Mende-Siedlecki, P., Vecchi, T., Todorov, A., & Cattaneo, Z. (2016). The dorsomedial prefrontal cortex plays a causal role in integrating social impressions from faces and verbal descriptions. *Cerebral Cortex*, 26, 156–165.
- Martin, A. K., Dzaic, I., Ramdave, S., & Meinzer, M. (2017). Causal evidence for task-specific involvement of the dorsomedial prefrontal cortex in human social cognition. *Social Cognitive and Affective Neuroscience*, 12, 1209–1218.
- Watanabe, T., Yahata, N., Abe, O., Kuwabara, H., Inoue, H., Takano, Y., ... & Yamasue, H. (2012). Diminished medial prefrontal activity behind autistic social judgments of incongruent information. *PLoS One*, 7, e39561.

Double Empathy

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Definition

The double empathy problem (DEP) refers to a “disjuncture in reciprocity between two differently disposed social actors” who hold different norms and expectations of each other, such as is common in autistic to non-autistic social interactions (Milton 2012, p. 884). With

different dispositional outlooks and personal conceptual understandings, interactions involving autistic and non-autistic people are susceptible to frequent misunderstandings. It is a “double problem” as both people experience it, and so it is not a singular problem located in any one person. However, “the disjuncture may be more severe for the non-autistic disposition as it is experienced as unusual, while for the ‘autistic person’ it is a common experience” (Milton 2012, p. 885).

In principal, the DEP becomes more marked the wider the disjuncture in dispositional perceptions of what constitutes the social context. It is suggested that “social subtext is never fully given as a set of *a priori* circumstances, but is actively constructed by social agents” (Milton 2012, p. 884). Thus, as interactions unfold, an initial gap in mutual understanding due to a dispositional difference can readily become a critical gap in mutual understanding which potentially terminates the interaction.

The explanatory scope of the double empathy problem is broad because it considers both individual outlooks of multiple social actors and the social context in which interactions takes place, such as cultural norms and stereotypes. Features of the double empathy problem that are characteristic of misunderstandings for non-autistic social actors include difficulties in reading autistic facial expressions and interpreting autistic perspectives, overgeneralizing attribution of blame, reduced tendency to critically reflect on one’s own role in contributing to misunderstandings, and underestimating autistic social ability because it may manifest unpredictably. Features of the double empathy problem experienced by autistic social actors include increased anxiety about interactional outcomes, increased frustration, and lower self-esteem, which in turn can have a potentially cascading effect on future mental health, economic prospects, and accessing supports and services.

The difficulty autistic people have in understanding non-autistic people has been extensively researched, although arguably not adequately from an autistic point of view (Milton and Bracher 2013). We therefore focus on research which

highlights the relatively ignored difficulties that non-autistic people have in understanding autistic perspectives.

Historical Background

The DEP theory originated from autistic scholar Damian Milton through discussions when advising parents in the late 2000s. Prior to diagnosis of his son and himself as autistic, Milton had referred to the DEP concept as “conditioned relativism” and “dispositional diversity” in the 1990s (Milton 2014a). Upon learning of his diagnosis, Milton began to explore autism theory including Theory of Mind. The DEP was Milton’s reinterpretation of ToM which re-situated perspective-taking and empathy as a two-way interactional process. It first appeared as a concept in conference presentations from 2010 and was first published in 2012 (Milton 2012). The theory was soon developed further looking at the concept of “interactional expertise” (Collins and Evans 2007; Milton 2014b) using Iris Marion-Young’s concept of “Asymmetrical Symmetry” (Milton 2016a).

The DEP has a number of theoretical antecedents reaching back to George Herbert Mead and his conceptualization of a “social act” (Mead 1934). For Mead social interaction did not comprise of functionally separate stimulus and response elements, because the categorization of such elements depends on one’s position within the social field. Instead a reciprocal relationship exists between stimulus and response whereby the response of one person is simultaneously the stimulus for another person. In this manner, people coregulate each other’s behavior through interaction. A two-way understanding of human social relations wherein people have the power to mutually reinforce each other’s behavior is the foundation of sociological works by Erving Goffman (1958) and Harold Garfinkel (1964) and underpins later work on “intersubjectivity” (Schegloff 1992).

The DEP shares commonalities with other theories explaining autistic social interaction. Ian Hacking’s research on “the looping effect”

explores the two-way effects of empathy that exists between society and individuals (Hacking 1995), wherein knowledge about an autistic diagnosis shapes the way autistic people behave and how others orientate to them, in turn “looping back” to reinforce societal expectations. Similarities can also be found in the work of Beardon (2017) in regard to what he refers to as “cross-neurological theory of mind,” and in the work of Chown (2014) who examined the double empathy problem through the use of Wittgenstein’s criteriological view of mind. Jaswal and Akhtar (2019) have argued that some influential accounts of autism may be based on unfavorable interpretation of autistic social motivation by non-autistic scientists, and that a more accurate and humane approach would consist of giving greater emphasis to autistic testimony. Bolis et al. (2017) have also examined the DEP in terms of a “dialectal misattunement hypothesis” which relates to disruptions in the dynamic and reciprocal nature of interactions across time, resulting in prediction errors and divergent interaction styles.

Current Knowledge

A growing body of experimental research is consistent with the notion that non-autistic people perceive autistic people differently and are prone to misperceiving autistic people.

Studies of Mindreading

Several studies have investigated whether non-autistic people find facial expressions of autistic people more difficult to read than those of non-autistic people. The majority of these studies asked groups of autistic and non-autistic participants to pose a series of facial expressions of emotion. Photos of these expressions were then shown to a separate group of raters who were blind to the diagnostic status of the participants and were asked to judge the emotion. Some studies have found the expressions of autistic participants were recognized more poorly than those posed by non-autistic comparison participants (Macdonald et al. 1989; Brewer et al.

2016), although others have found little difference (Volker et al. 2009).

Some studies have attempted to capture emotional expressions in more naturalistic ways, closer to the circumstances under which they may be observed in everyday life. Grossman et al. (2013) used a story retelling task and found that non-autistic adults were equally able to use facial expressions to identify the emotional content of a story told by autistic and non-autistic participants. Faso et al. (2015) elicited emotions in autistic and non-autistic participants by narrating autobiographical memories to them, finding that the facial expressions of autistic participants were recognized just as accurately as those of non-autistic participants, and in fact, anger was recognized more accurately for autistic participants.

Non-autistic people may also have difficulty interpreting other aspects of autistic people's behavior. Sheppard et al. (2016) investigated non-autistic participants' ability to interpret the behavioral reactions of autistic people in naturalistic social interactions. Autistic and non-autistic participants were covertly filmed reacting to a seemingly incidental but actually scripted aspect of the researcher's behavior. While briefing the participant, she either told them a joke, paid them some compliments, told them about the difficult day she was having, or kept them waiting while doing irrelevant activities. Non-autistic participants who viewed the recorded videos were less able to guess which event the video participant had experienced for autistic than non-autistic participants, apart from for reactions to the joke. Edey et al. (2016) asked autistic and non-autistic participants to manipulate two triangles to create animations depicting mental state interactions such as "coaxing" or "mocking." Non-autistic observers who viewed the animations were better at identifying the mental state depicted for animations created by other non-autistic participants than autistic participants.

In summary, research in this area suggests that while non-autistic people may sometimes be able to identify facial expressions of autistic people, they have difficulty making sense of autistic people's behavior in context which might negatively

impact on social interactions between autistic and non-autistic people.

Studies of Forming First Impressions

Research has also asked a more general question of how autistic people are perceived by non-autistic others. If autistic people are perceived less favorably, then this could result in avoidance and social exclusion, contributing to the social difficulties they experience. Stagg et al. (2014) found that non-autistic adults rated autistic children as less expressive and less attractive than the non-autistic children based on brief videos of them. Meanwhile, children rated them lower on a variety of evaluative dimensions. In a study using a much larger sample of adult participants, Sasson et al. (2017) carried out three studies in which they showed that non-autistic adults rated autistic adults and children less favorably than non-autistic adults and children on a wide variety of evaluative dimensions, as well as indicating reduced intentions to engage with them.

Further research by Sasson and Morrison (2017) examined the impact of providing diagnostic labelling information on the impressions formed. They compared non-autistic participants' judgments of video participants displayed with either no label, or the correct diagnostic label, or the alternative label (e.g., labelling the autistic person as having no diagnosis). Autistic and non-autistic participants were rated more positively when labelled as autistic than when no label or the alternative label was provided, although this did not completely eradicate the tendency to form more negative impressions of the autistic participants. Moreover, raters with higher levels of autism knowledge gave more favorable ratings to correctly labelled autistic participants. Taken together, these results suggest that diagnostic disclosure might reduce negative first impressions of autistic people, especially for people with greater knowledge about autism.

Studies of Metaperception

Some researchers have combined elements of mindreading and impression formation by examining metaperception, which is the ability

to form an impression of what others think about us. Sasson et al. (2018) investigated metaperception using the same videos from Sasson et al. (2017) and Sasson and Morrison (2017b). Video participants were asked to estimate how they thought others would perceive them on a wide range of personality traits, and then observers judged them on the same traits after viewing their video. They found that autistic participants were less accurate than non-autistic participants in judging how they would be perceived as others, because they overestimated how positively they would be perceived.

While Sasson et al. (2018) study asked participants about how they come across to others in general, Usher et al. (2018) studied impressions formed by dyads of adolescents where one member of the dyad was autistic and one was not, who engaged in a 5-min conversation. Autistic participants were found to be more accurate in judging whether the non-autistic partner liked them than non-autistic participants were. This is consistent with non-autistic people having difficulty interpreting autistic people and suggests that autistic people may be adept in using social feedback from a specific person to gauge how they are perceived by that particular individual.

Metaperception has also been investigated between dyads of autistic and non-autistic people who know each other well. Heasman and Gillespie (2018) used the Interpersonal Perception Methodology (IPM) to investigate perceptions and misperceptions for dyads of autistic individuals and their family members. Both groups predicted that the other would rate them differently than they had themselves on a number of characteristics, evidencing an ability to take a perspective distinct from their own. Moreover, there were few differences for either group between predicted ratings of the other and actual ratings made by the other, such that both groups were fairly accurate in estimating others' perceptions. When asked about reasons for misunderstandings, family members tended to cite an extreme impairment in social understanding of the autistic person, while autistic participants themselves reflected

on both the self and other as causes of misunderstandings.

A further double empathy barrier regarding metaperception may also relate to how autistic and non-autistic people see themselves relative to each other. Research conducted by Gernsbacher et al. (2017) explored the effect of manipulating the context (e.g., with whom) and reference (e.g., according to whom) on questionnaire items of the Broad Autism Phenotype Questionnaire (designed to measure autistic traits). In their first experiment, autistic and non-autistic participants completed the questionnaire where the context specified their outgroup, e.g., for autistic participants an item would read "I like being around non-autistic people." Both groups of participants self-reported more autistic traits when the context group was specified to the participants' out-group. Yet autistic participants reported having fewer autistic traits when the reference-group was specified to their in-group, e.g., for autistic participants an item would read "according to autistic people I have unusual eye contact." These findings highlight that a disjuncture between autistic and non-autistic people exists for both parties in how they view themselves relative to each other, and that autistic people report fewer autistic traits when the reference group was other autistic people.

Heasman and Gillespie (2019b) examined metaperception through a video game where typically developing participants were led to believe they were interacting with another online player to complete a maze when in truth all players interacted with an AI that was programmed to behave the same way. The diagnostic status of the AI was manipulated across three groups of participants to be autistic, dyslexic, or having no diagnostic status at all. The findings highlighted that when the AI was believed to be autistic, they were seen as significantly more intelligent; however, they were also seen as less helpful. Moreover, participants believed they were more helpful when the AI was believed to be autistic, despite there being no behavioral evidence of this. Such disparity between perceptions of being helpful and actual

helping behaviors towards autistic people may further contribute to DEP effects, alongside stereotyped beliefs associated with autistic intelligence and sociability (Heasman 2018).

Overall, studies of metaperception suggest that autistic people are quite good at estimating how specific others perceive them but may have some difficulty judging how they come across in general. Consistent with the DEP, non-autistic people demonstrate difficulty working out how they are perceived by familiar and unfamiliar autistic people.

Neurodiverse Interactions

It has been observed that autistic people appear to have a greater affinity with other autistic people than non-autistic people generally do (Chown 2014). This raises the possibility that autistic people may show improved, if not superior, understanding of other autistic people and may consequently show few signs of “social impairment” in the company of their in-group. Heasman and Gillespie (2019a) examined 30 naturally occurring interactions between autistic adults playing video games, focusing on neurodivergent intersubjectivity, the process through which neurodivergent people develop shared understanding. Their research identified a particular pattern of social coordination that flourished between autistic interlocutors, where the tendency to describe in detail one’s own interest and experiences provided ample opportunity for other autistic interlocutors to discover points of interest and reciprocate with their own stories and ideas. Similarly, the tendency for autistic interlocutors to have a low expectation for tight coordination (an expectation often enforced in neurotypical interactions) resulted in flexibility for autistic participants to explore individual and cooperative ways of making sense of their environment. Specifically, these features of generously sharing one’s ideas and not enforcing an expectation that every exchange should result in reciprocation were complimentary, a pattern which is unlikely to flourish in autistic to non-autistic interaction which take place against the backdrop of normative interaction expectations (Heasman 2018).

Further support for double empathy observed within interactions was highlighted in research by Crompton et al. (2019) who examined information transfer between autistic adults, neurotypical adults, and mixed groups of autistic and neurotypical adults along a diffusion chain (groups of eight participants passing information from one person to the next). Their study observed a significantly steeper decline in the retention of detailed information in chains consisting of autistic and neurotypical participants. Chains only consisting of autistic participants showed similar levels of information retention compared to chains of neurotypical participants, suggesting that it is specifically the interface between autistic and neurotypical communication where problems arise. Furthermore, rapport ratings were lower in mixed chains than autistic- or neurotypical-only chains highlighting that perceptions of sociability are also framed differently depending on the interlocutors.

Morrison et al. (2019) examined unstructured conversations between dyads consisting only of autistic people, only of typically developing people, and mixed dyads of autistic and typically developing people. All participants completed a rating evaluation of their partner after the conversation. Their results showed that on the whole autistic people were rated as more awkward, less attractive, and less socially warm than typically developing people by both typically developing people and autistic partners. This suggests that some aspects of social interaction associated with autism are invariant to the disposition of the perceiver and target. However, their study also found that autistic people had better social affiliation with other autistic people, evidenced by sharing more about themselves and a greater interest in future interactions.

It should be noted that other studies have been less successful in demonstrating an in-group advantage in perception towards autistic people. For example, Brewer et al. (2016) found that both autistic and non-autistic viewers were poorer at identifying the emotions posed by autistic participants, suggesting that emotion expression in autism may be idiosyncratic to the individual. In Edey et al. (2016), no in-group advantage was

observed: autistic viewers were equally able to identify the mental state depicted in animations created by autistic and non-autistic participants. Nevertheless, more research is needed in this area as it remains possible that an in-group advantage in understanding may be more evident natural contexts.

Interventions Addressing the DEP

The DEP has been incorporated into a number of autism training and intervention programs. It is one of five elements of best practice in autism that form the National Autistic Society's (UK) SPELL framework, which incorporates measures to reduce the double empathy gap. Other autism interventions that target the social situation rather than solely the autistic person also have the potential to ameliorate DEP effects. For instance, ATLASS training by Studio3 focuses on acknowledging how the context (carer or service staff) influence the autistic person's behavior, mediated by levels of stress. AT-Autism also include elements of the DEP in their Synergy program which is for professionals working in schools and aims to develop their understanding of how various factors including aspects of the social environment affect the autistic child's experience of the world. Further research is needed to evaluate such interventions taking into account perspectives of both the autistic and non-autistic participants.

Future Directions

Expanding the current evidence base for the double empathy problem will help to improve understanding about the processes through which it occurs, its scale and impact across different contexts of social life, and possible interventions that can ameliorate its negative social effects for both autistic and non-autistic individuals.

Further research could explore the empirical link between being misunderstood or perceived negatively and measures of quality of life (e.g., mental health) (Milton and Sims 2016). A variety of factors could be investigated with respect to

this relationship. For example, the effects of a two-way breakdown in empathy and understanding may result from a difference between monotropic individuals, who have the tendency to localize attentional resources on a specific interest to the exclusion of other potential inputs, and polytropic individuals who are capable of spreading their attentional resources to multiple inputs simultaneously (Murray 1992; Murray et al. 2005). Further research on the link between the DEP and monotropism could shed light on the developmental origins of the DEP, particularly given that most research to date has focused on adults, but we might assume these difficulties arise as a consequence of a transactional, albeit socially situated, developmental process. Another feature to explore is the role of culture in amplifying misunderstandings. Milton (2014b) explored theoretically to what extent the DEP is culturally embedded, given the different representations and approaches to autism in popular culture and suggested that culture may contribute to some difficulties in "interactional expertise" (Collins and Evans 2007) between autistic and non-autistic people. Cultural misinterpretations are an area of interactional difficulty that are easier to change than one's dispositional nature, thus in addition to developing new, holistic interventions, the DEP may also have implications for updating existing interventions, which often place social normativity as an assumed improvement on quality of life, when this is not always the case (Milton 2016b).

The DEP may have important application to a number of different areas of social life, particularly for older autistic populations who experience rapid increases in the size and diversity of their social networks as they progress through adolescence and adulthood (White et al. 2009). For example, as mentioned above, the DEP may help to explain why so many autistic adults have such high comorbidity with mental health issues. Pressures for children to become independent in late adolescence can place an increasing strain on family relationships, which may be amplified by the DEP effects especially if autistic people are disproportionately held accountable for breakdowns in understanding (Heasman and Gillespie

2018). Breakdowns in family relations may consequently deny autistic people of the few social supports available and could detrimentally impact perceptions of autism acceptance (Cage et al. 2018). Future directions for research should examine the perspectives of autistic family members in addition to autistic people themselves to identify the supports required as they transition towards being an informal carer.

Finding and retaining employment for autistic people is another context in which the DEP is particularly salient. The social encounter of the job interview and the difficulty in managing professional relations (which are qualitatively different from all other relationships) are two environments governed by complex roles, norms, and expectations which can easily lead to misunderstandings (Hendricks 2010). Future research can examine employers' potential biases in social perception of autistic adults which may impede progress in job interviews and daily working tasks. This may help to identify the contributing factors towards the current autism employment gap observed in many countries.

The DEP may also help to shed light on the numerous encounters autistic people face as they progress through the justice system, such as providing a police statement or testimony in court. In such interactions, autistic people will be highly anxious potentially reducing their credibility as their behavior and intentions are susceptible to misinterpretation. In addition, research has shown that autistic people have difficulty in recalling events personally experienced (Maras and Bowler 2014), thus future directions for research can examine the perspectives of magistrates in interpreting and scaffolding such recall, as well as the impressions that jury members may take from such encounters.

Late diagnosis of autism can leave many autistic adolescents and adults facing a variety of neurotypical interactions as they attempt to access support and services for their disability. The process of assessing disability needs may be further complicated by masking and camouflaging (Dean et al. 2017) and anxiety about outcomes both in terms of financial support and impacts on one's identity (Kite et al. 2013). Moreover,

research on other disability assessment procedures have highlighted the difficulty in translating one's impairment into criteria on assessment forms since caregivers and care-receivers have divergent perspectives on the burden of care (Moore and Gillespie 2014). Further research should therefore examine the DEP in terms of the perspectives involved in the social encounters experienced throughout the diagnostic pathway, and the institutional barriers that exist between the disabilities experienced and the instruments used to measure the support needs of disabilities.

Sexuality, sexual health, and gendered self are important frontiers for future research on the DEP since one's sense of self is relationally formed (Dewinter et al. 2017; Yergeau 2017). In addition to misunderstanding autistic perspectives, the complex sensory needs many autistic people experience may further contribute to misalignment of perspective in sexual encounters. Autistic vulnerability in social understanding means there is great risk of potential harm or abuse that might result from DEP misunderstandings in intimate relationships. Further research should explore these risk factors to inform education and support provided.

Finally, the DEP also has epistemological implications in terms of participatory and emancipatory research. The two-way nature of misunderstandings that are observed in interpersonal relations also exist between researcher and participant. For example, in the UK, autistic adults report a mismatch between their priorities for research and the funding for autism research, which should focus more on how to make a difference to people's day-to-day lives (Pellicano et al. 2014). It is therefore important that research design and engagement benefit from autistic involvement (Milton and Bracher 2013; Milton 2014b) in order to have a positive impact on outcomes for autistic people (Fletcher-Watson et al. 2019).

See Also

- [Monotropism: An Interest-Based Account of Autism](#)

References and Reading

- Beardon, L. (2017). *Autism and Asperger syndrome in adults*. London: Sheldon Press.
- Bolis, D., Balsters, J., Wenderoth, N., Becchio, C., & Shilbach, L. (2017). Introducing the dialectical misattunement hypothesis and a Bayesian account of Intersubjectivity. *Psychopathology*, 50(6), 335–372.
- Brewer, R., Biotti, F., Catmur, C., Press, C., Happé, F., Cook, R., & Bird, G. (2016). Can neurotypical individuals read autistic facial expressions? Atypical production of emotional facial expressions in autism spectrum disorders. *Autism Research*, 9(2), 262–271.
- Cage, E., Di Monaco, J., & Newell, V. (2018). Experiences of autism acceptance and mental health in autistic adults. *Journal of Autism and Developmental Disorders*, 48(2), 473–484. <https://doi.org/10.1007/s10803-017-3342-7>.
- Chown, N. (2014). More on the ontological status of autism and double empathy. *Disability & Society*, 29(10), 1672–1676.
- Collins, H., & Evans, R. (2007). *Rethinking expertise*. Chicago: University of Chicago Press.
- Crompton, C., Ropar, D., Evans-Williams, C., Flynn, E., & Fletcher-Watson, S. (2019). Autistic peer to peer information transfer is highly effective. <https://doi.org/10.31219/osf.io/j4knx>
- Dean, M., Harwood, R., & Kasari, C. (2017). The art of camouflage: Gender differences in the social behaviors of girls and boys with autism spectrum disorder. *Autism*, 21(6), 678–689. <https://doi.org/10.1177/1362361316671845>.
- Dewinter, J., Van Parys, H., Vermeiren, R., & Van Nieuwenhuizen, C. (2017). Adolescent boys with an autism spectrum disorder and their experience of sexuality: An interpretative phenomenological analysis. *Autism*, 21(1), 75–82. <https://doi.org/10.1177/1362361315627134>.
- Edey, R., Cook, J., Brewer, R., Johnson, M. H., Bird, G., & Press, C. (2016). Interaction takes two: Typical adults exhibit mind-blindness towards those with autism spectrum disorder. *Journal of Abnormal Psychology*, 125(7), 879–885.
- Faso, D. J., Sasson, N. J., & Pinkham, A. E. (2015). Evaluating posed and evoked facial expressions of emotion from adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(1), 75–89.
- Fletcher-Watson, S., Adams, J., Brook, K., Charman, T., Crane, L., Cusack, J., Leekham, S., Milton, D., Parr, J. R., & Pellicano, E. (2019). Making the future together: Shaping autism research through meaningful participation. *Autism*, 23(4), 943–953.
- Garfinkel, H. (1964). Studies of the routine grounds of everyday activities. *Social Problems*, 11(3), 225–250.
- Gernsbacher, M. A., Stevenson, J. L., & Dern, S. (2017). Specificity, contexts, and reference groups matter when assessing autistic traits. *PLoS One*, 12(2), e0171931.
- Goffman, E. (1958). *The presentation of self in everyday life*. Edinburgh: Edinburgh University Press.
- Grossman, R. B., Edelson, L. R., & Tager-Flusberg, H. (2013). Emotional facial and vocal expressions during story retelling by children and adolescents with high-functioning autism. *Journal of Speech, Language, and Hearing Research*, 56(3), 1035–1044.
- Hacking, I. (1995). The looping effects of human kinds. In D. Sperber, D. Premack, & A. J. Premack (Eds.), *Symposia of the Fyssen Foundation. Causal cognition: A multidisciplinary debate* (pp. 351–394). New York: Clarendon Press/Oxford University Press. <https://doi.org/10.1093/acprof:oso/9780198524021.003.0012>.
- Heasman, B. (2018). Enabling autistic sociality: Unrealised potentials in two-sided interaction. *Autism*. PhD thesis, The London School of Economics and Political Science (LSE). <http://etheses.lse.ac.uk/3864/>
- Heasman, B., & Gillespie, A. (2018). Perspective-taking is two-sided: Misunderstandings between people with Asperger's syndrome and their family members. *Autism*, 22(6), 740–750. <https://doi.org/10.1177/1362361317708287>.
- Heasman, B., & Gillespie, A. (2019a). Neurodivergent intersubjectivity: Distinctive features of how autistic people create shared understanding. *Autism*, 23(4), 910–921. <https://doi.org/10.1177/1362361318785172>.
- Heasman, B., & Gillespie, A. (2019b). Participants overestimate how helpful they are in a two-player game scenario toward an artificial confederate that discloses a diagnosis of autism. *Frontiers in Psychology*, 10 (JUN), 1–15. <https://doi.org/10.3389/fpsyg.2019.01349>.
- Hendricks, D. (2010). Employment and adults with autism spectrum disorders: Challenges and strategies for success. *Journal of Vocational Rehabilitation*, 32(2), 125–134. <https://doi.org/10.3233/JVR-2010-0502>.
- Jaswal, V., & Akhtar, N. (2019). Being versus appearing socially uninterested: Challenging assumptions about social motivation in autism. *Behavioral and Brain Sciences*, 42, E82. <https://doi.org/10.1017/S0140525X18001826>.
- Kite, D. M., Gullifer, J., & Tyson, G. A. (2013). Views on the diagnostic labels of autism and Asperger's disorder and the proposed changes in the DSM. *Journal of Autism and Developmental Disorders*, 43(7), 1692–1700. <https://doi.org/10.1007/s10803-012-1718-2>.
- Macdonald, H., Rutter, M., Howlin, P., Rios, P., Contour, A. L., Evered, C., & Folstein, S. (1989). Recognition and expression of emotional cues by autistic and normal adults. *Journal of Child Psychology and Psychiatry*, 30(6), 865–877.
- Maras, K. L., & Bowler, D. M. (2014). Eyewitness testimony in autism spectrum disorder: A review. *Journal of Autism and Developmental Disorders*, 44(11), 2682–2697. <https://doi.org/10.1007/s10803-012-1502-3>.
- Mead, G. H. (1934). Thought, communication, and the significant symbol. In C. W. Morris (Ed.), *Mind, self,*

- and society from the standpoint of a social behaviorist (pp. 68–75). Chicago: University of Chicago Press.
- Milton, D. (2012). On the ontological status of autism: The “double empathy problem.” *Disability & Society*, 27(6), 883–887. <https://doi.org/10.1080/09687599.2012.710008>.
- Milton, D. (2014a). Embodied sociality and the conditioned relativism of dispositional diversity. *Autonomy, the Critical Journal of Interdisciplinary Autism Studies*, 1(3), 1–7. <http://www.larry-arnold.net/Autonomy/index.php/autonomy/article/view/AR10>.
- Milton, D. (2014b). Autistic expertise: A critical reflection on the production of knowledge in autism studies. *Autism*, 18(7), 794–802. <https://doi.org/10.1177/1362361314525281>.
- Milton, D. E. (2016a). Disposable dispositions: Reflections upon the work of Iris Marion young in relation to the social oppression of autistic people. *Disability & Society*, 31(10), 1403–1407.
- Milton, D. (2016b). *Educational discourse and the autistic student: A study using Q-sort methodology*. Birmingham: University of Birmingham.
- Milton, D., & Bracher, M. (2013). Autistics speak but are they heard. *Journal of the BSA MedSoc Group*, 7, 61–69.
- Milton, D., & Sims, T. (2016). How is a sense of well-being and belonging constructed in the accounts of autistic adults? *Disability and Society*, 31(4), 520–534. <https://doi.org/10.1080/09687599.2016.1186529>.
- Moore, H., & Gillespie, A. (2014). The caregiving bind: Concealing the demands of informal care can undermine the caregiving identity. *Social Science and Medicine*, 116, 102–109. <https://doi.org/10.1016/j.socscimed.2014.06.038>.
- Morrison, K. E., DeBrabander, K. M., Jones, D. R., Faso, D. J., Ackerman, R. A., & Sasson, N. J. (2019). Outcomes of real-world social interaction for autistic adults paired with autistic compared to typically developing partners. *Autism*. <https://doi.org/10.1177/1362361319892701>.
- Murray, D. (1992). Attention tunnelling and autism. In P. Shattock, & G. Linfoot (Eds.), *Living with autism: The individual, the family and the professional*. Durham Research Conference, April 1995, Sunderland. pp. 183–193.
- Murray, D., Lesser, M., & Lawson, W. (2005). Attention, monotropism and the diagnostic criteria for autism. *Autism*, 9(2), 136–156.
- Pellicano, E., Dinsmore, A., & Charman, T. (2014). What should autism research focus upon? Community views and priorities from the United Kingdom. *Autism*, 18(7), 756–770. <https://doi.org/10.1177/1362361314529627>.
- Sasson, N. J., & Morrison, K. E. (2017). First impressions of adults with autism improve with diagnostic disclosure and increased autism knowledge of peers. *Autism*. <https://doi.org/10.1177/1362361317729526>.
- Sasson, N. J., Faso, D. J., Nugent, J., Lovell, S., Kennedy, D. P., & Grossman, R. B. (2017). Neurotypical peers are less willing to interact with those with autism based on thin slice judgments. *Scientific Reports*, 7, 40700.
- Sasson, N. J., Morrison, K. E., Pinkham, A. E., Faso, D. J., & Chmielewski, M. (2018). Brief report: Adults with autism are less accurate at predicting how their personality traits are evaluated by unfamiliar observers. *Journal of Autism and Developmental Disorders*, 48(6), 2243–2248.
- Schegloff, E. (1992). Repair after next turn: The last structurally provided defense of intersubjectivity in conversation. *American Journal of Sociology*, 97(5), 1295–1345.
- Sheppard, E., Pillai, D., Wong, G. T. L., Ropar, D., & Mitchell, P. (2016). How easy is it to read the minds of people with autism spectrum disorder? *Journal of Autism and Developmental Disorders*, 46(4), 1247–1254.
- Stagg, S. D., Slavny, R., Hand, C., Cardoso, A., & Smith, P. (2014). Does facial expressivity count? How typically developing children respond initially to children with autism. *Autism*, 18(6), 704–711.
- Usher, L. V., Burrows, C. A., Messinger, D. S., & Henderson, H. A. (2018). Metaperception in adolescents with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(2), 533–548.
- Volker, M. A., Lopata, C., Smith, D. A., & Thomeer, M. L. (2009). Facial encoding of children with high-functioning autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 24(4), 195–204.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229. <https://doi.org/10.1016/j.cpr.2009.01.003>.
- Yergeau, M. (2017). *Authoring autism: On rhetoric and neurological queerness*. Durham: Duke University Press.

Double-Blind Study

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Definition

DOUBLE-BLIND TRIAL. The double-blind trial is a research method that attempts to reduce the

bias in research studies. In the classic double-blind trial, subjects are randomly assigned to receive an active medication or a placebo. The placebo is formulated to look and perhaps even taste like the active medication – but the placebo contains no active ingredients. We use the term “double-blind” to indicate that investigators and patients (and parents) do not know whether the patient is getting the active medication or the placebo. The treatment mask is intended to reduce bias and expectation.

When a new medication is being introduced, there may be a lot of interest and hope for the new medication. In the absence of placebo control, this interest and hope could lead to false impressions about the benefits of the medication. Indeed, high expectations can also contribute to the so-called “placebo effect.” In several recent studies in children with autism spectrum disorders, as many as one third of the subjects on placebo were classified as much improved or very much improved. For example, in the citalopram study (► [Citalopram](#)), 34% of the subjects randomly assigned to placebo showed a positive response rated by clinician who was blind to treatment assignment.

Several elements are essential in the conduct of a double-blind, placebo-controlled trial. First and perhaps most important is random assignment. Random assignment is essential to ensure that the two treatment groups are similar. Second, there should be a match between the entry criteria and the study treatment. For example, in the risperidone trial conducted by the RUPP Autism Network (► [Citalopram](#)), subjects were required to have serious behavioral problems. This ensures that there is room for improvement on the target clinical symptoms. This is important for ethical and statistical reasons. It is fair to compare a new medication to placebo when it is unknown whether the new treatment is effective. In most situations, however, it seems unfair to enroll subjects into a medication study if it was known that the active treatment has a low chance of conferring benefit. In statistics, investigators are interested in finding out if the new treatment is superior to placebo. Subjects who have low severity on the clinical target have little room for improvement, which will make it difficult to detect change.

Finally, there is the issue of sample size. A trial that is too small cannot answer the question whether the new medication is superior to placebo. This could be unfortunate if a beneficial treatment is abandoned too soon because it failed to show efficacy in a small trial. On the other hand, treatment trials are expensive.

Moreover, we are asking subjects and families to consider randomization to placebo. A trial should only be as large as needed to test whether the new treatment is superior to placebo. Investigators have to determine the minimum magnitude of benefit that would be considered clinically meaningful and then calculate the sample size needed. The “minimum clinically meaningful benefit” depends on the treatment target. For example, self-injurious behavior is a serious problem. Even a modest level of benefit might be considered meaningful. Repetitive behavior such as rocking or watching the same video over and over can be problematic – but not as severe as self-injury. For a less severe behavior, a higher level of benefit might be demanded of a new treatment. In general, the smaller the difference between medication and placebo, the larger the trial has to be.

References and Reading

- Vitiello, B., & Scahill, L. (2011). Clinical trials methodology and design. In A. Martin, L. Scahill, & C. Kratochvil (Eds.), *Pediatric psychopharmacology: Principles and practice* (pp. 711–724). New York: Oxford University Press.

Douglass Developmental Disabilities Center

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Major Areas or Mission Statement

The Douglass Developmental Disabilities Center (DDDC) is a unit of the Graduate School of

Applied and Professional Psychology at Rutgers, the State University of New Jersey, and is located in New Brunswick, New Jersey, on the Rutgers campus. The DDDC opened in 1972 to serve children with autism. Because of the university affiliation, the Rutgers Board of Governors had to officially approve its establishment. In the beginning, there were nine children, two teachers, and several graduate students as well as a small cohort of undergraduates from the university.

Landmark Contributions

In the decades since its modest start, the DDDC has steadily expanded its services and refined its mission. The current tripart functions of the DDDC are to (1) serve people on the autism spectrum and their families, (2) educate undergraduate and doctoral students in the latest methods of treating people with autism spectrum disorders (ASD), and (3) do research on questions of importance in the treatment of people with autism and on meeting the needs of their families. From its inception, the DDDC has relied on the principles of applied behavior analysis (ABA) to guide services. As the teaching strategies derived from ABA have grown more elegant and precise, so too have the teaching methods at the Center. Methods that were once at the heart of practice in the 1970s have evolved steady into the more effective and extensively studied techniques in use in the twenty-first century.

Major Activities

Direct Service to People with Autism

Two units at the DDDC are devoted to center-based direct instruction of people with autism. These are the Douglass School and Adult Services.

The Douglass School is approved by the New Jersey Department of Education as a “college-operated program” to serve children and adolescents from 3 to 21 years of age with autism spectrum disorders (ASD) who need a specialized setting to address their educational needs and behavior intervention services to address

inappropriate behaviors. Because of a very intense staff to student ratio, the Douglass School has sufficient staff members to address the unique needs of each learner. Both skill acquisition and behavior reduction programs for all learners regardless of age are based on the science of applied behavior analysis. Specific strategies vary based on the needs of the learner and the most current empirically validated and least intrusive strategies that meet the needs of each individual. Families are urged to be active in the education of their children and are provided with training in ABA teaching methods as well as being invited to do regular observations at the Center. In addition to work in the classroom and at home, every effort is made to bring students into the community so they can use their skills in the settings where they will be most appropriate.

The DDDC’s Small Wonders Preschool is an integrated classroom that has both children on the autism spectrum and typically developing peers who serve as role models for age appropriate behavior. This classroom model, which was opened in the early 1980s, has been adopted by other programs in the public and private sector. Over the years, approximately half of the target children served in this classroom have left the Center for a regular education classroom in a public or private school.

The adult services program serves adults with autistic disorder and intellectual disability who are 21 years of age or older. These adults either continue to need a very intense adult to client ratio and/or have other significant challenges that make it difficult for them to be in a less specialized adult program. The Center’s objective for every person in the adult program is to integrate them as fully as possible into the community. As of 2011, a little under a quarter of the adults spend 5 days a week in community vocational settings, the majority of the other adults spend 3–4 days a week in a community vocational setting, and it is a rare for an adult client to have no vocational activities outside of the Center. These vocational placements include janitorial work at local restaurants, yard work both on and off of campus, house cleaning, and doing basic clerical work in offices including filing, copying, and other support tasks. In addition, among those adults who are not

engaged in vocational tasks 5 days a week, all of them take part in community-based recreational activities. The parent of one adult at the center created a private entity called “Men with Mops” that bills private individuals and companies for the services the adults provide and issues paychecks to the workers.

Consultation to Schools and Families

In addition to direct service to people with autism, the Center also provides extensive consultation services to public and private schools in the New Jersey, New York, and Pennsylvania area. These services are provided by staff members working for the DDDC’s Outreach Services. Schools sign contracts with Outreach Services to provide in-class consultation to teachers who have children with ASD in their classrooms or for support in establishing an in-district applied behavior analysis program. These consultations vary from once or twice a month to several days a week depending on the needs and request of the school district. Some districts contract for a brief period and others draw on these consultation services for many years to ensure that their teachers continue using state-of-the-art ABA techniques as those methods evolve.

Outreach Services also provide two kinds of home-based services. One of these is early intervention for children under the age of 3 years and the other is home-based services on a full-day basis or after the child’s school day has ended.

The early intervention program (EI) serves infants and toddlers younger than 3 years of age in their own homes. In addition to direct services to the child by the home consultant, parents are also taught the ABA intervention techniques so they can use them in their daily interactions with their child. Among the older children who receive home-based services about 30% have full day/4 or 5 days a week intervention. The rest of the families receive services after school or on a part-time basis during the day. Again, these services are based on the principles of ABA and typically involve direct instruction to the child as well as helping parents master the techniques so they can apply the ABA methods on their own.

Other Services to Families

In addition to educational/treatment services through Douglass Outreach, the DDDC provides assessment and diagnostic services for families and schools. This includes diagnostic assessments, intelligence testing, speech and language assessments, and learning evaluations. The Center has a group of full-time staff and part-time consultants who do these evaluations and make treatment recommendations. Douglass Outreach Services have an NJ Department of Education–approved child study team for providing second opinions at the request of families and/or schools. Outreach Services staff members also do functional assessments of problematic behaviors for schools and families and make detailed treatment recommendations based on these assessments.

Educating Undergraduate and Graduate Students

Educating undergraduate students about autism and behavioral intervention strategies has been at the core of the DDDC’s mission since its inception. Junior and senior undergraduate students at Rutgers University can enroll in fieldwork in psychology. Through the field work course, 40–50 undergraduates per semester participate in one day per week of clinical work in a classroom for students or adults with autism. Their hands-on clinical training is supplemented by didactic training and lectures by the DDDC’s teachers, graduate teaching assistants, and faculty. Fieldwork training covers topics such as behavioral intervention, applied behavior analysis teaching strategies, assessment, curriculum, and characteristics of autism. Undergraduates are able to take a second semester of fieldwork and participate in an advanced seminar while continuing their clinical experience.

Undergraduates are also able to enroll in a research methods class focusing on single-case design and applied behavior analysis research methodology. A small number of students enrolled in the research course each semester spend 10 h per week participating in ongoing DDDC research projects and activities such as running experimental sessions, integrity and reliability data collection, literature review and

critique, and data coding and compilation. Students are also active in a weekly seminar led by a senior graduate student.

Graduate training at the DDDC takes place in one of three ways. Primary graduate training experiences are available to the full-time doctoral program in clinical psychology through the Graduate School in New Brunswick and the Graduate School of Applied and Professional Psychology at Rutgers, the State University of New Jersey. Graduate students are offered practicum positions or graduate assistantships at the DDDC. Graduate students serve as behavioral consultation staff and support in the assessment and treatment of challenging behavior, while also supporting case management and behavior analytic research. Advanced doctoral students also support the DDDC's research mission while conducting independent theses and dissertations. Graduate students gain experience in teaching by coordinating the undergraduate courses in fieldwork and research.

Other graduate training at the DDDC takes place through the University's Center for Applied Psychology and Continuing Education program. Graduate students from other university departments and professionals from the general community can enroll in a series of graduate courses taught by DDDC faculty. These courses are designed to fulfill the academic requirements for becoming board-certified behavior analysts.

The Research Mission of the DDDC

The research mission of the DDDC is to explore best practice behavior analytic treatments for autism and contribute to the dissemination of research to support their use. Research activity at the DDDC is driven by the clinical needs of clients at the Center and the needs of the general and scientific communities to which we belong. As such, the focus of the DDDC's clinical research shifts according to the presenting needs of the students and the status of the science in the field of behavioral autism treatment.

The DDDC also works collaboratively with researchers across different disciplines at the University and at other University settings, by supporting recruitment, methodology consultation,

and providing autism expertise to projects by multidisciplinary research teams.

Current and ongoing research themes in the DDDC research plan are the evaluation of behavior analytic teaching strategies, methods for assessing and intervening with challenging behavior, impact of autism on families, and methods for assessing and predicting treatment outcome and progress in behavioral treatment.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Early Intensive Behavioral Intervention \(EIBI\)](#)

Down Syndrome

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Synonyms

[Down's syndrome](#); [Trisomy 21](#)

Definition

This condition, first described by Langdon Down in the 1860s, is caused by the presence of three copies of chromosome 21 (trisomy 21). The trisomy can reflect an entire extra copy of chromosome 21 or a partial one (the latter due to translocation). At one time, a very common genetically caused form of intellectual disability, the frequency has decreased given the potential for diagnosis early in the pregnancy. Both cognitive difficulties and characteristic features and medical problems are present.

Overall, cognitive level is typically in the mild to moderate range of impairment (average IQ 50).

Individuals with mosaic Down syndrome (where only some cell lines exhibit the trisomy 21) may have higher IQs. Physical problems include slow physical growth and characteristic features such as unusual facial appearance (due to a round face, epicanthal folds, small chin, and large tongue). Cardiac defects are frequent. Other medical problems can include seizures, ear infections, thyroid problems, as well as leukemia and higher rates of Alzheimer's disorder. Educational interventions focus on fostering overall development and adaptive skills to achieve the highest possible functional outcomes in adults.

Interest in Down syndrome relative to autism arises for several reasons. It has frequently been the case that individuals with Down syndrome have been used as control or comparison groups in studies of autism. There has also been an impression that Down syndrome individuals are typically more social than might otherwise be expected given their cognitive level although several case reports suggest that Down syndrome and autism co-occur. Howlin et al. (1995) described four such cases and emphasized the importance of correct diagnosis of both conditions to be able to provide appropriate services.

See Also

► [Intellectual Disability](#)

References and Reading

- Dykens, E. M., Shah, B., Sagun, J., Beck, T., & King, B. H. (2002). Maladaptive behaviour in children and adolescents with Down's syndrome. *Journal of Intellectual Disability Research*, 46(Pt. 6), 484–492.
- Ghaziuddin, M., Tsai, L. Y., & Ghaziuddin, N. (1992). Autism in Down's syndrome: Presentation and diagnosis. *Journal of Intellectual Disability Research*, 36 (Pt. 5), 449–456.
- Howlin, P., Wing, L., & Gould, J. (1995). The recognition of autism in children with Down syndrome: Implications for intervention and some speculations about pathology. *Developmental Medicine and Child Neurology*, 37(5), 406–414.
- Kasari, C., Freeman, S. F., Bauminiger, N., & Alkin, M. C. (1999). Parental perspectives on inclusion: Effects of autism and Down. *Journal of Autism and Developmental Disorders*, 29(4), 297–305.
- Yirmiya, N., Pilowsky, T., Solomonica-Levi, D., & Shulman, C. (1999). Brief report: Gaze behavior and theory of mind abilities in with autism, down syndrome, and mental retardation of unknown. *Journal of Autism and Developmental Disorders*, 29(4), 333–341.

Down's Syndrome

► [Down Syndrome](#)

Doxepin

► [Adapin](#)
 ► [Sinequan](#)

Drama and Autism

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Definition

I regard the theatre as the greatest of all art forms, the most immediate way in which a human being can share with another the sense of what it is to be a human being.

Oscar Wilde (as cited in Corbett et al. 2011)

Drama interventions attempt to provide creative, enjoyable, and engaging opportunities for people with autism spectrum disorders (ASD) to practice a wide range of social skills in the safety and protection of a workshop environment. Ranging from whole group to one-to-one settings, drama interventions operate on the basis of the creation of a fictional context (i.e., a pretend situation), which playfully captures the attention of the participants and encourages interaction and communication with others. Operating on a continuum, there are several different approaches to using

drama as an intervention with people with ASD, varying from involvement in theater performance and working on play scripts at one end to improvisation and simulation at the other. Underpinning all forms of drama interventions is an intention to actively involve the participant in exploring and making sense of the world in which they live and working creatively with them to understand their place and their relationship with others in that environment. Drama interventions are structured, arts-based, educational mediations, involving a facilitator, teacher, or therapist who draws from a range of creative and fun teaching and learning strategies to actively involve the participant in learning for increased social awareness, communication, and understanding. Typically, both participants and facilitators take on roles ranging from simple two-dimensional roles, such as pretending to be a shopkeeper, dentist, or grandmother, to highly developed roles such as pretending to be a superhero going on a mission to defeat Lord Taylor and save the world.

“Going into role” is an important defining characteristic of drama interventions and often involves a facilitator “in role,” improvising an everyday situation with the participants who may take on a different role and respond/react to the situation as it unfolds in a workshop setting (commonly referred to as role-playing; see O’Sullivan 2011). These roles can be swapped and developed, adding in new complications or changes each time a scene is replayed. Alternatively, if participants are socially and cognitively able for it and in particular if they are able to differentiate between fiction and reality, both facilitator and participants can “go into role” and engage in exciting, action-filled, improvised drama, which facilitates the practice and exploration of a wide range of personal and social skills. During such interventions, both participants and facilitator together try to unravel and solve various fictional mysteries and problems (this activity is typically referred to as drama in education or process drama; see Howell and Heap 2013; Edmiston 2014). The former approach is often used in drama therapy settings on a one-to-one basis or with small groups with explicit therapeutic objectives in a clinical setting, and the latter is

most often associated with inclusive classroom practice in schools or in extracurricular drama classes with specialist drama teachers working with groups of between 8 and 14 participants.

Theater as an intervention is typically associated with working from existing scripts or scripts which the group devise/create from their own experiences/interests and which participants rehearse and then perform in front of an audience. In contrast, drama in education places much less emphasis on the performance aspect and highlights the process and value of engaging with fictional scenarios, where there is no script but participants react to the fictional situation in an improvised manner. Drama in education is also commonly referred to as “process drama.” Drama therapy (written as dramatherapy in the UK and several other European countries) is closely related to process drama but is defined as the use of drama as a therapeutic method. Similarly, psychodrama is active and experiential and encourages the spontaneity and creativity of clients for therapeutic purposes.

Historical Background

The use of drama as a social and educational intervention began in the nineteenth century, but drama has been valued for thousands of years for its ability to entertain, educate, and heal (Brockett and Hildy 2007). References to the cathartic and therapeutic effects of both observing and participating in drama and theater performances are replete in the literature, from Sophocles’ *Antigone* in the fifth century BC to the dramatic works of Shakespeare, Middleton, Fletcher, and Ford in the seventeenth century and numerous references to the value of building theaters in psychiatric hospitals in countries such as Italy, France, Germany, and the USA in the eighteenth and nineteenth centuries (Casson 2007). Similarly, the use of drama in young people’s education has long been documented, stretching back to Athenian education and reappearing again in the Renaissance period, when “By the late sixteenth century, almost all schools used drama” (Courtney 1968, p. 19). Harriet Finlay-Johnson (1912/2008) and

Henry Caldwell Cook (1917) began using dramatic play in schools as a specific teaching and learning method in England at the turn of the twentieth century, with evidence of similar practices in use in schools in Croatia, Romania, Poland, and eastern Europe from the mid-1800s. The development of drama in education by pioneers such as Winifred Ward, Peter Slade, Viola Spolin, Brian Way, Nellie McCaslin, Dorothy Heathcote, Gavin Bolton, Betty Jane Wagner, Cecily O'Neill, David Davis, and Jonothan Neelands, throughout the twentieth century, firmly established the role of drama as an effective and creative approach to teaching and learning in formal and nonformal educational contexts. Recognizing that creativity and spontaneity are the propelling forces in human progress, Jacob Levy Moreno was similarly exploring the use of drama with children in Vienna [1908], before later developing its application in therapeutic procedures known as psychodrama and sociodrama (Moreno 1939). Peter Slade (1954), a pioneer in the field of theater for children and whose child drama philosophy was influential in the development of drama therapy, was also an expert in using drama as an intervention with children with special needs, which Winifred Ward had earlier started to develop in the USA in the 1920s.

From the 1960s onward, a parallel approach was developed to working with children and people with special educational needs (SEN). Some practitioners started to specialize in the emerging field of drama therapy, notably led by Sue Jennings (1982, 1987), and others began to work in this area using drama interventions developed by Dorothy Heathcote (Johnson and O'Neill 1984). Both traditions use similar techniques (dramatic play, improvisation, role-play, movement, mime, drama games, use of masks and puppets, and working with scripts, myths, and stories), often with similar equipment, such as dress-up materials, a box of clothes, art supplies, stories, books, and musical instruments. Both share interweaving influences from the areas of drama, theater, and psychology and aim to work on similar skills: expressing and exploring feelings, developing spontaneity, imagination and creativity, improving self-image and self-

confidence, and developing social relationships. However, they have very distinct purposes. Drama therapists use elements of the performing arts in their work and apply it in a clinical setting. It has been defined by Sue Jennings (1982) as "the specific application of theatre structures and drama processes with a declared intention that it is therapy," while Moreno (1983) described its forerunner psychodrama as "the science which explores the truth by dramatic methods. It deals with inter-personal relations and private worlds." An explicit emphasis on using drama as a therapeutic method in a clinical setting differentiates these practices from other forms of drama interventions, which focus on more general development and practice of personal and social skills and thus feature more prominently in the literature about interventions for people with ASD.

Despite the fact that Viktorine Zak, a nurse at the Vienna Children's Hospital where Hans Asperger worked, developed drama programs to teach social skills to children with ASD in the 1940s (Asperger, [1944], as cited in Attwood 2007), there has been little sustained attention to its use in the intervening period and even fewer published studies discussing the use of drama and theater interventions with people with ASD. Passing mention of drama is made when discussing functional or symbolic play as a social skills intervention. References to structured social and sociodramatic play (Conn 2007) and role-play (Nelson 2010) predominate in the field (the former particularly in early years settings), with limited reference to participants engaging in theater performance (Schneider 2007; Vickers 2005) or drama interventions (Sherratt and Peter 2002; O'Sullivan et al. 2009; 2010). However, despite its poor presence in the scholarly literature, online information is available about several recently established programs, associations, and professional publications and magazines dedicated to the use of theater and drama interventions with children and young people with ASD. Notable examples include Andrew Nelson and Parasuram Ramamoorthi's "Applied Theater Research and Autism Network" (ArTRAN) and its associated "Velvi Theater Heals" organization based in India; Stanley Greenspan's "Floortime," which inspired

Elaine Hall to establish the well-known “Miracle Project” in California leading to the release of “Autism: The Musical”; the Autism Theater Network (part of the Applied Theater Center); “Social Drama” being developed by Carmel O’Sullivan in association with ASPIRE (the Asperger Syndrome Association Of Ireland) in Dublin, Ireland; Theater Horizon; Face Place Theater Project in Indiana; Social Emotional NeuroScience Endocrinology (SENSE) Theater in California; People’s Light & Theater in Pennsylvania; and ArtStream in Washington, D.C. in addition, and several professional theater companies and playhouses have established programs involving what they describe as the use of occupational therapy and therapeutic interactive performances for and with young people with ASD, such as at the Florida Repertory Theater Company, Des Moines Playhouse in Iowa, and the Red Kite Project at Chicago Children’s Theater. The first autism-friendly performance on Broadway took place in 2011 (*The Lion King*) and has become one of the fastest growing services for people with communication barriers (Mandell 2013).

Rationale or Underlying Theory

ASD is characterized by differences in social communication and restricted interests and repetitive behaviors. Although individuals with a diagnosis of ASD have uneven cognitive and social skills profiles, they are all likely to experience a degree of challenge in the areas of pragmatics (the social use of language) and social cognition (being able to see things from another person’s perspective). It is acknowledged that teaching such skills should best be attempted using concrete materials and opportunities to make abstract concepts meaningful and tangible. However, they are often taught in isolation of real world, authentic contexts, which limits generalizability to other situations beyond the confines of the treatment room or classroom (O’Sullivan et al. 2010). In contrast, all drama interventions, whether theater, therapy, or process drama oriented, place participants in fictional roles and situations, many of which mirror or represent real life and which

require social communication, problem-solving, working in pairs/small groups and in whole group activities, being flexible and receptive to other’s ideas, constructively building on their suggestions, negotiating, collaborating, cooperating, and exercising global and social observation skills rather than local and physical processing (Russell-Smith et al. 2012; O’Sullivan et al. 2012). Drama interventions engage participants experientially, introducing them to fictional situations and stories, and somewhat larger than life, colorful characters who attract their curiosity and attention on a number of levels: emotionally, cognitively, personally, and socially. The intention is to engage participants in an exploration of that character’s life and to actively participate in following that journey, working collaboratively with peers and supporting teachers/facilitators to resolve various exciting and challenging situations as they arise (O’Sullivan et al. 2010).

As emotional education of people with ASD is an important component of their development (Sappok et al. 2014), drama interventions operate on the premise that emotions are rational and cognitive in kind and therefore can be educated (Best 1992). In drama interventions, the techniques used typically focus on communication skills and building social relationships rather than emphasizing only the dramatic or theatrical art form, as would be the case in professional actor training. Drama interventions are the medium through which people with ASD can encounter a range of human experiences and are offered the possibility of considering ideas from different angles and perspectives while expanding their conceptual horizons, deepening their understanding of human behavior, and, in so doing, appropriately educating their emotions and increasing their empathy with others.

Drama interventions draw from the traditions of dramatic play, process drama, theater, and developmental psychology, particularly:

- The “as-if” mode of thinking necessary for spontaneous make-believe play (Taylor and Warner 2006)
- Theater (Brook’s *Empty Space*, 1968, and Boal’s spect-actor, 2002)

- Education (Dewey's progressivism, 1938, and Vygotsky's social constructivism, 1978)
- Anthropology (ritual and myth, Schrader 2012)
- Social, developmental, and clinical psychology (object relations, symbolic interaction theories, and personal construct psychology, Langley 2006)

Drama theorists have developed strategies to create a safe and structured environment for participants. These include such techniques as a "drama contract" to diminish potential behavior management issues and distancing conventions so that participants are removed in time and space from the characters they are exploring or the roles they are playing (Jones 2007; Chasen 2011). This facilitates an important objective distancing from the participants' own lives, but experiences are gently resonated back to the individual through periods of planned reflection in each session. Techniques such as "protection into role" and "protection into emotion" (Davis 2014; O'Sullivan 2011) gently ease participants into role without placing social and communication demands on them for which they may not yet be ready.

It is in the domain of what are traditionally called "soft skills" that the use of drama interventions can play a role, pushing the boundaries further than what can be achieved through social skills interventions alone. Drama's relevance as an intervention with people with ASD is through its ability to create stories in which participants come face to face with the world: recognizing their world and their relationship to it (Davis 2014). Heathcote (Johnson and O'Neill 1984) developed a series of drama conventions, which allow participants to engage with the world from different perspectives, to see things from different points of view. This approach fits well with the theory of mind. Taking on a role in drama in education allows the participant to "stand in the shoes" of another person and respond to the unfolding fictional story "as if" they were that person. The key difference between this approach to drama and more formal theater approaches is that in the latter, the participant

often takes on the full set of attributes of a *character* (rather than a role), imitating how they walk, talk, and behave, which many people with ASD are good at and enjoy doing but which reduces the educational potential of the intervention, as the participant metaphorically "steps out of *character*" as soon as the activity is over and leaves much of the learning entailed in the experience behind in the rehearsal room or workshop. In drama education and drama therapy approaches, the participant takes on only a few of the characteristics of the role they are playing, and thus they are able to self-spectate, i.e., observe themselves playing that role and learn from the experience through reflection both during the drama and afterward (O'Sullivan 2011). It increases the potential for transferability of social communication skills, imagination, and flexibility into other settings beyond the immediate drama context.

Goals and Objectives

Drama interventions are closely related to social, symbolic, cooperative, and pretend play, social interventions, and social skills interventions. However, in drama the work is generally developed more in the domain of exploration, experiential learning, and reflection rather than focusing on the teaching and practice of social communication skills out of context. Drama interventions are intended to facilitate the exploration of pragmatics, social cognition, social imagination, social interaction, social emotional reciprocity, and personal development in contextualized and enjoyable "as-if" situations. Participants are gently eased into a make-believe scenario through the use of character and role and occasionally using props and costume, where they are placed into positions which require them to behave "as if" they are someone else encountering that particular situation and interacting with others, drawing on their personal and social skills repertoire to address and/or resolve the fictional scenario. The overriding objective of using drama interventions with people with ASD is to assist in the development of theory of mind, perspective taking, and

executive functioning [encoding, processing, and integrating social information] (Minne and Semrud-Clikeman 2012; Guli et al. 2013) while simultaneously using pragmatic language skills in a holistic experience where social skills are integrated in real-time experiences rather than being taught in isolation. Drama interventions contextualize social skills in incremental episodes according to the cognitive and social functioning abilities of participants and facilitate exploration and examination of identity (Hodermarska 2013; Goodley and Runswick-Cole 2011), autonomy (Carter et al. 2013), independence, empathy, emotional development, and developmentally appropriate relationships (see Goldstein and Naglieri 2013).

Treatment Participants

Drama interventions are used with children (typically aged 6 and older), with adolescents, and to a much lesser degree with adults, with a wide range of abilities and symptom severity across the autism spectrum. The most common participants are school-age children in extracurricular settings such as social skills and theater groups, although there is some evidence that process drama interventions are being increasingly used in mainstream inclusive educational settings and special schools (Schneider 2007; Sherratt and Peter 2002). Research has indicated that drama interventions are likely to be more successful for children with higher social interaction and communication skills which are often prerequisites for engagement in drama interventions (Chang et al. 2014).

Treatment Procedures

Individuals involved in drama interventions typically participate in one of three broad areas of treatment:

1. Theater and performance
2. Drama in education and process drama
3. Drama therapy and psychodrama

The first two categories do not require referral, and participants (often with support from parents, teachers, or other caregivers and professionals) self-select to attend such activities. In the case of attendance at drama therapy and psychodrama, referral is made to a specialist therapist who assesses the individual's needs and establishes treatment goals and objectives as part of an overall treatment plan, often working within a multidisciplinary team (Silverman 2006). Owing to its explicit therapeutic objectives, drama therapy and psychodrama adhere to strict regulatory guidelines relating to optimum group size, ranging from individual sessions to small group (3–5 people) and large group sessions (more than 6 people), and comply with formal requirements for evaluation and reporting mechanisms (Bailey 2010). Depending on the needs of individuals, the duration of drama therapy may range from 30 to 50 min sessions, one to three times weekly over a period of 10–16 weeks, although longer term therapy can be extended over a period of 2 or 3 years (Chasen 2011).

Participation in drama and theater in education activities typically involves groups of 8 or more subjects, in weekly sessions of 1–3 h duration, over a period of 6 months to a year. Where individuals enjoy participation in these drama interventions, their attendance may continue for a much longer period of time (O'Sullivan 2014). Formal and systematic assessment and evaluation methodologies are rarely used in such practices, where the emphasis is on participating in a highly charged social situation which requires participants to interact in a meaningful way and build social relationships with others in the group (Nelson 2010).

In all three treatment categories, social learning and experience is facilitated through active engagement with the art form of drama and theater which is used as a vehicle to explore:

- Psychosocial difficulties (including cognitive learning difficulties; difficulties related to defining and expressing feelings, interacting with others, planning and decision-making, and understanding the intentions of others;

emotional and behavioral problems; and socially inappropriate behavior)

- Neurodevelopmental difficulties (including difficulties in concentrating, motor coordination, dyspraxia, hyperactivity)
- Mental health/psychological difficulties (including agitation, anxiety, mood swings, loneliness, depression, phobias)
- Behavioral difficulties (including aggressive physical and verbal behavior, conduct disorders, oppositional defiant behavior) (Andersen-Warren 2013)

Each drama intervention session typically involves an opening and a closing activity to mark the beginning and end of the class, where participants are seated in a circle and greet one another, share any stories or “news” they have, and play some warm-up games and drama exercises to nurture the group identity. Opportunities for reflection are usually built in to the structure of each session to allow participants to relate what they have experienced in the drama or theater workshop to their own lives.

Efficacy Information

Despite the fact that social communication skills are core challenges associated with ASD, there is still relatively little attention paid to interventions in this area in the literature, and areas such as sociodramatic play, imaginative play, symbolic play, creative dramatics, arts therapy, play therapy, psychodrama, role-playing, and psychotherapy social skills training are significantly underrepresented in peer-reviewed publications. Not unexpectedly, drama interventions fare even worse and rarely feature in the scholarly literature. This may be related to challenges associated with accurately defining and delineating the scope of practice in this field but also possibly to the nature of engagement with the art form, which until relatively recently did not have a strong tradition in empirical research (Donovan 2011). However, drama interventions have recently begun to appear in the professional literature related to ASD (i.e., online websites, blogs, teaching

manuals, magazines, and videos), which are designed to impact upon and disseminate practice.

Notwithstanding criticisms about research in the area of social skills interventions, which has been beset by a lack of randomized control tests and claims that many studies lack rigorous research designs to assess the effectiveness of interventions, generalization effects, and maintenance (Kaat and Lecavalier 2014), there has been a dearth of systematic research on drama interventions. Apart from a few small-scale case studies involving single subject or small-N design (Kempe and Tissot 2012; Corbett et al. 2011; Wilmer-Barbrook 2013; Pimpas 2013; Dunne 2009), the literature has been largely silent in this regard. This despite increased interest in drama-based treatment programs to ameliorate the symptoms of ASD in recent years and with them, the demand to use evidence-based interventions (EBI) (Livanis et al. 2013).

However, what has emerged positively from those studies undertaken in recent years is the degree of enjoyment, empowerment, and independence, which participants and their parents/carers have reported as a result of being part of social skills and drama interventions (Mandelberg et al. 2014; Loyd 2013a; Guli et al. 2013; Ramamoorthi and Nelson 2011). Notwithstanding the challenges associated with accurately validating changes and progress and indeed accessing experiences of this nature (Shaughnessy 2013), participants themselves have self-reported such changes and developments in their engagement with peers, their self-confidence, and self-esteem, reduction in anxiety, etc. (Trimingham 2013). Participant voice is not a new area in the field of inclusive education (Oliver 1992), and first-person perspectives of people with ASD are beginning to bring fresh insights to this area of research (Kirby et al. 2014). For example, Loyd’s (2013b) study of 10 young people with autism in a drama education intervention program argues for the active involvement of people with autism in research about them.

In a study involving a drama in education intervention with two girls with ASD in a special school setting with other students with moderate intellectual disabilities, Kempe and Tissot (2012)

reported positive gains in participation in group work, language use, development of imagination and humor, and sustained engagement with a fictional character which led the participants into the practice of social skills that had relevance beyond the confines of the classroom. Trowsdale and Hayhow (2013) report similar progress in their case study of one boy with ASD (in a larger sample of participants with learning disabilities) who demonstrated marked improvements in identifying and regulating his emotions, developing a playful relationship with another child, and enhancing his imaginative and creative abilities. Using mimetics and theater methodology, they record reductions in ritualized activities and positive developments of the case study on the child's personal and social identity. Employing a similar methodology, researchers from Butler University (Loer 2012) engaged in a 10-week collaborative study with the Pathway School and People's Light & Theater Company, involving eight adolescents in rehearsals of Mark Twain's *The Adventures of Tom Sawyer*, in which repetition and learning lines, mimicry, and exploration of emotions were key elements. Further eight students from the same school served as a control group. Participating students exhibited significant improvement in four behaviors: displaying appropriate emotions, offering help without prompting, controlling temper, and acknowledging the perspective of others.

Corbett et al. (2011) identify close links between theater and modeling techniques, and in a 3-month pilot project involving the pairing of peer actors with participants with ASD, researchers noted some promising potential for social skills training in a nurturing and fun environment. Using a unique performance-based social skills curriculum called SDARI (Sociodramatic Affective-Relational Intervention) and employing affectively engaging improvisation games and dramatic training adapted for people with ASD, Lerner et al. (2011) suggest that increased social assertion, improved ability to detect emotions in adult voices, and decreased social problems may be encouraged by participating in this drama-based intervention. In a recent meta-analysis of social skills interventions, Kaat and Lecavalier (2014) refer to the potential of

further investigating performance-based (specifically drama-based) interventions, such as those described above by Corbett et al. (2011) and Lerner's SDARI (Lerner and Mikami 2012; Lerner et al. 2011).

In a counterpoint approach to mimesis, Shaughnessy (2013) describes a research project with 22 children called "Imagining Autism" which explored the potential of drama as an intervention to challenge the stereotypes surrounding ASD. The approach adopts a nonmimetic scenography requiring new modes of theatrical perception and demanding an active form of participatory spectatorship. In "Imagining Autism" (www.imaginingautism.org), participants recognized that they were performers and authors and at liberty to improvise and play within a process-based contemporary performance.

The studies cited above all relate to a work carried out with children and adolescents, but similar gains have been reported in projects with adults. In a study of 18 adults with ASD, participants referred to their attraction to theater-based interventions, such as improvisational theater classes, the writing of dramatic scripts, participation in role-playing games and voice workshops, which most respondents described as allowing them an opportunity to practice social skills and reduce social anxiety. In the words of one respondent, "by doing [improvisational theater], you realize that it's actually possible to be spontaneous, to just go with an impulse (laugh)" (Müller et al. 2008).

Preliminary evidence exists to support some degree of generalization of skills to settings outside of the drama intervention group, but a major limitation of the studies conducted is the small number of participants, which limits the generalizability of results.

Outcome Measurement

Increasingly, drama therapists and drama facilitators are being asked to demonstrate the effectiveness of their work through evidence-based practice (EBP) and practiced-based evidence (PBE) (Dokter and Winn 2010) and to produce

outcome measurements for their practice. Descriptive measures are no longer considered satisfactory on their own, and Jennings (2011) advocates the use of such assessment and evaluation measures as the PASAA technique (Play and Story Attachment Assessment), among others. The big challenge facing those who engage in drama interventions is to consolidate the relationship between research, impact, and evidence (Jones 2012). Despite the wider debate about the efficacy of double-blind randomized controlled trials (RCTs) which are regarded as the gold standard in the generation of knowledge, Zeisel (2011) argues that they are only one way of representing knowledge and that there are other methodologies which contribute substantially to our knowledge of non-pharmacological interventions which need to be taken seriously. He includes in this list music, visual arts, film, drama, counseling, and storytelling and advocates that the most appropriate methodology is the one which best fits the research question. The challenge then for the field of drama interventions is to grow its practice-based research and increase capacity in different ways, as is occurring in other creative interventions, such as music, arts, and dance therapy (Jones 2012).

A significant stumbling block may be the lack of assessment instruments capable of adequately measuring social skills across a variety of contexts. Standardized tests are typically inadequate in sensitivity measures to assess these skills. Casson (2004) notes that while clients and participants may evaluate a session as being fun or enjoyable, clinical effectiveness demands measurement in terms of behavioral change and adjustments to societal norms (Jones 2012). While it is undeniable that more rigorous research designs and targeted evaluation of social skills are needed to determine the efficacy of drama interventions, a number of studies have relied on qualitative methodologies from the field of social sciences, which have yielded satisfactory results (Kempe and Tissot 2012; Trowsdale and Haythorne 2013). Most use instruments from play therapy and social communication interventions (Wolfberg et al. 2012; Lerner et al. 2011; Corbett et al. 2011), with the most popular measures in

drama therapy being the Strengths and Difficulties Questionnaire (SDQs), the CORE system (Clinical Outcomes for Routine Evaluation), Behavioral Summarized Evaluation, and Therapy Outcomes. More recently, PSYCHLOPS and PSYCHLOPS Kids have been successfully used with children with ASD as a reliable and highly sensitive measure of change during psychotherapeutic drama interventions (Godfrey and Haythorne 2013).

Qualifications of Treatment Providers

While there is currently no requirement for people who use drama interventions with people with ASD to be certified, the available evidence suggests that most are either trained actors or qualified elementary or high school teachers with experience of working in inclusive educational settings. Many have graduate qualifications in developmental psychology, drama, or theater in education, special education, autism studies, film and media studies, occupational therapy, speech and language therapy, and music and arts therapy. Guli et al. (2008) advocate for training not only in working with youth with ASD but also in understanding the specialized skills needed to facilitate drama interventions to achieve specific group and individual goals.

In contrast, drama therapy is a registered and licensed profession in the USA and UK. However, there is no state regulation currently in respect of drama or other arts therapies in many countries (see <http://www.dramatherapy.net> and <http://www.ecarte.info>). Drama therapy is a masters-level profession, with applicants having successfully pursued a bachelors degree in drama or a psychological health-related subject, with appropriate experience of working, paid or voluntarily, with people with specific needs, for example, mental ill health, learning disabilities, nursing assistant, support worker, and drama or theater work with people with specific needs. Course content typically covers such areas as psychology; drama, theater, and performance work; knowledge of related therapies such as art, music, dance/movement, and play; supervised drama

therapy practice; personal therapy parallel with training; supervised internship; and work experience. Registered drama therapists follow a code of ethics and abide by the professional body's regulations on fitness to practice, continuing professional development, and standards of proficiency.

See Also

- [Integrated Play Groups \(IPG\) Model](#)
- [Music Therapy](#)
- [Play Therapy](#)
- [Social Skill Interventions](#)

References and Reading

- Andersen-Warren, M. (2013). Drama therapy with children and young people who have autistic spectrum disorders: An examination of dramatherapists' practices. *Drama Therapy, 35*(1), 3–19.
- Attwood, T. (2007). *The complete guide to Asperger's syndrome*. London: Jessica Kingsley.
- Bailey, S. (2010). Drama therapy. In K. Siri & T. Lyons (Eds.), *Cutting edge therapies for Autism 2010–2011*. New York: Skyhorse Publishing.
- Best, D. (1992). *The rationality of feeling*. Oxford: The Falmer Press.
- Boal, A. (2002). *Games for actors and non-actors* (2nd ed.) (trans: Jackson, A.). New York: Routledge.
- Bowell, P., & Heap, B. (2013). *Planning process drama: Enriching teaching and learning* (2nd ed.). Oxon: Routledge.
- Brockett, O. G., & Hildy, F. J. (2007). *History of the theatre* (10th ed.). London: Pearson.
- Brook, P. (1968). *The empty space*. London: Penguin.
- Caldwell Cook, H. (1917). *The play way*. London: Heinemann.
- Carter, I., Munro, S., & Matin, S. (2013). Exploring autonomy in group work practice with persons with intellectual disabilities. *Social Work With Groups, 36*(2–3), 236–248.
- Casson, J. (2004). *Drama, psychotherapy and psychosis, dramatherapy and psychodrama with people who hear voices*. East Sussex: Brunner-Routledge.
- Casson, J. (2007). 17th century theatre therapy Shakespeare, Fletcher, Massinger, Middleton and Ford: Five Jacobean Healing Dramas. *Dramatherapy, 29*(1), 3–9.
- Chang, Y. C., Laugeson, E. A., Gantman, A., Ellingsen, R., Frankel, F., & Dillon, A. R. (2014). Predicting treatment success in social skills training for adolescents with autism spectrum disorders: The UCLA Program for the Education and Enrichment of Relational Skills. *Autism, 18*(4), 467–470.
- Chasen, L. (2011). *Social skills, emotional growth and drama therapy: Inspiring connection on the autism spectrum*. Philadelphia: Jessica Kingsley.
- Conn, C. (2007). *Using drama with children on the Autism spectrum*. Brackley: Speechmark.
- Corbett, B. A., Gunther, J. R., Comins, D., Price, J., Ryan, N., Simon, D., Schupp, C. W., & Rios, T. (2011). Brief report: Theatre as therapy for children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 41*(4), 505–511.
- Courtney, R. (1968). *Play, drama & thought: The intellectual background to dramatic education*. London: Cassell.
- Davis, D. (2014). *Imagining the real: Towards a new theory of drama in education*. Stoke on Trent: Trentham Books.
- Dewey, J. (1938). *Experience and education*. New York: Kappa Delta Pi.
- Dokter, D., & Winn, L. (2010). Evaluating dramatherapy. EBP and PBE: A research project. *Dramatherapy, 31*(1), 3–9.
- Donovan, C. (2011). State of the art in assessing research impact. *Research Evaluation, 20*(3), 175–179.
- Dunne, L. (2009). Playing for real: Drama therapy, autism and an eight year old boy. In S. L. Brooke (Ed.), *The use of creative therapies with autism spectrum disorders*. Springfield: Charles C. Thomas.
- Edmiston, B. (2014). *Transforming teaching and learning with active and dramatic approaches*. New York: Routledge.
- Finlay-Johnson, H. (1912/2008). *The dramatic method of teaching*. London: Kessinger Publishing.
- Godfrey, E., & Haythorne, D. (2013). Benefits of dramatherapy for autism spectrum disorder: A qualitative analysis of feedback from parents and teachers of clients attending Roundabout dramatherapy sessions in schools. *Dramatherapy, 35*(1), 20–28.
- Goldstein, S., & Naglieri, J. A. (Eds.). (2013). *Interventions for autism spectrum disorders: Translating science into practice*. New York: Springer.
- Goodley, D., & Runswick-Cole, K. (2011). Something in the air? Creativity, culture and community. *Research in Drama Education: The Journal of Applied Theatre and Performance, 16*(1), 75–91.
- Guli, L. A., Wilkinson, A. D., & Semrud-Clikeman, M. (2008). *Social competence intervention program: A drama-based intervention for youth on the autism spectrum*. Champaign: Research Press.
- Guli, L. A., Semrud-Clikeman, M., Lerner, M. D., & Britton, N. (2013). Social Competence Intervention Program (SCIP): A pilot study of a creative drama program for youth with social difficulties. *Arts in Psychotherapy, 40*(1), 37–44.
- Hodermarska, M. (2013). Autism as performance. *Drama Therapy and Autism Spectrum (Special Issue), 35*(1), 64–76.
- Jennings, S. (1982). *Remedial drama*. London: Adam and Charles Black.

- Jennings, S. (1987). *Creative therapy*. Banbury: Kemble Press.
- Jennings, S. (2011). Play and story attachment assessment (PASAA). *Drama Therapy*, 33(1), 45–57.
- Johnson, L., & O'Neill, C. (1984). *Dorothy heathcote: Collected writings on education and drama*. London: Hutchinson.
- Jones, P. (2007). *Drama as therapy: Theory practice and research*. Oxon: Routledge.
- Jones, P. (2012). Approaches to the futures of research Part 2: Measure for measures, researching, re-viewing and re-framing drama therapy in practice. *Dramatherapy*, 34(3), 116–138.
- Kaat, A. J., & Lecavalier, L. (2014). Group-based social skills treatment: A methodological review. *Research in Autism Spectrum Disorders*, 8, 15–24.
- Kempe, A., & Tissot, C. (2012). The use of drama to teach social skills in a special school setting for students with autism. *British Journal of Learning Support*, 27(3), 97–102.
- Kirby, A. V., Dickie, V. A., & Baranek, G. T. (2014). Sensory experiences of children with autism spectrum disorder: In their own words. *Autism*, 19, 316.
- Langley, D. (2006). *An introduction to drama therapy*. London: Sage.
- Lerner, M. D., & Mikami, A. Y. (2012). A preliminary randomized controlled trial of two social skills interventions for youth with high-functioning autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 27, 147–157.
- Lerner, M. D., Mikami, A. Y., & Levine, K. (2011). Socio-dramatic affective-relational intervention for adolescents with Asperger syndrome and high functioning autism: Pilot study. *Autism*, 15(1), 21–42.
- Livanis, A., Benvenuto, S., Mertturk, A., & Hanthorn, C. A. (2013). Treatment integrity in autism spectrum disorder interventions. In S. Goldstein & J. A. Naglieri (Eds.), *Interventions for autism spectrum disorders: Translating science into practice* (pp. 19–38). New York: Springer.
- Loer, J. M. (2012, Apr 17). Theatre and therapy? The Twain Meet (Via Twain). *American Theatre*.
- Loyd, D. (2013a). Gaining views from pupils with Autism about their participation in drama classes. *British Journal of Learning Disabilities*. <https://doi.org/10.1111/bld.12078>.
- Loyd, D. (2013b). Obtaining consent from young people with autism to participate in research. *British Journal of Learning Disabilities*, 41(2), 133–140.
- Mandelberg, J., Frankel, F., Cunningham, T., Gorospe, C., & Laugeson, E. A. (2014). Long-term outcomes of parent-assisted social skills intervention for high-functioning children with autism spectrum disorders. *Autism*, 18(3), 255–263.
- Mandell, J. (2013, May/June). The circle of inclusion. *American Theater*, 66–69.
- Moreno, J. L. (1939). Psychodramatic shock therapy: A sociometric approach to the problem of mental disorders. *Sociometry*, 2(1), 1–30.
- Moreno, J. L. (1983). *The theatre of spontaneity*. Ambler: Beacon House Inc.
- Müller, E., Schuler, A., & Yates, G. B. (2008). Social challenges and supports from the perspective of individuals with Asperger syndrome and other autism spectrum disabilities. *Autism*, 12(2), 173–190.
- Nelson, A. (2010). *Foundation role plays for autism*. Philadelphia: Jessica Kingsley Publishers.
- O'Sullivan, C. (2011). Role-playing. In L. Cohen, L. Manion, & K. Morrison (Eds.), *Research methods in education* (7th ed., pp. 510–527). London: Routledge.
- O'Sullivan, C. (2014). The Day that Shrek was almost rescued: Doing process drama with children with an autism spectrum disorder. In P. Duffy (Ed.), *What was I thinking: A reflective practitioner's guide to (Mis) adventures in drama education*. New York: Peter Lang.
- O'Sullivan, C., McKernan, D., O'Halloran, S., & Rowland, J. (2009). *Asperger syndrome: A practical guide for parents, teachers, young people and other professionals*. [2 hour DVD]. Dublin: Aspire.
- O'Sullivan, C., McNulty, U., Conroy, L., Walsh, A., & McKernan, D. (2010). Asperger syndrome and social skills education through creative drama. In D. Lyons (Ed.), *Creative studies for the caring professions* (pp. 178–189). Dublin: Gill and McMillan.
- O'Sullivan, C., Boran, L., & Delany, D. (2012). A study of the role of drama in developing social versus physical attribution abilities in children on the autistic spectrum. In *Borders and translations: Towards new paradigms and languages in drama education*, IDIERI (the International Drama in Education Research Institute), University of Limerick, 10–15 July.
- Oliver, M. (1992). Changing the social relations of research production. *Disability, Handicap and Society*, 7(2), 101–114.
- Pimpas, I. (2013). A psychological perspective to dramatic reality: A path for emotional awareness in autism. *Dramatherapy*, 35(1), 57–63.
- Portman Minne, E., & Semrud-Clikeman, M. (2012). A social competence intervention for young children with high functioning autism and Asperger syndrome: A pilot study. *Autism*, 16(6), 586–602.
- Ramamoorthi, P., & Nelson, A. (2011). Drama education for individuals on the autism spectrum. In S. Schonmann (Ed.), *Key concepts in theatre/drama education* (pp. 177–181). Rotterdam: Sense Publishers.
- Russell-Smith, S., Murray, M., Bayliss, D., & Sng, A. (2012). Support for a link between the local processing bias and social deficits in autism: An investigation of embedded figures test performance in non-clinical individuals. *Journal of Autism & Developmental Disorders*, 42(11), 2420–2430.
- Sappok, T., Budczies, J., Dziobek, I., Bölte, S., Dosen, A., & Diefenbacher, A. (2014). The missing link: Delayed emotional development predicts challenging behavior in adults with intellectual disability. *Journal of Autism & Developmental Disorders*, 44(4), 786–800.

- Schneider, C. B. (2007). *Acting antics. A theatrical approach to teaching social understanding to kids and teens with asperger syndrome*. Philadelphia: Jessica Kingsley.
- Schrader, C. (2012). *Ritual theatre, the power of dramatic ritual in personal development groups and clinical practice*. Philadelphia: Jessica Kingsley.
- Shaughnessy, N. (2013). Imagining otherwise: Autism, neuroaesthetics and contemporary performance. *Interdisciplinary Science Reviews*, 38(4), 321–334.
- Sherratt, D., & Peter, M. (2002). *Developing play and drama in children with autistic spectrum disorders*. London: David Fulton.
- Silverman, T. (2006). Drama therapy theoretical perspectives. In S. L. Brooke (Ed.), *Creative arts therapies manual* (pp. 223–231). Springfield: Charles C. Thomas.
- Slade, P. (1954). *Child drama*. London: Hodder & Stoughton.
- Taylor, P., & Warner, C. (2006). *Structure and spontaneity: The process drama of Cecily O'Neill*. Stoke on Trent: Trentham Books.
- Trimingham, M. (2013). Touched by meaning: Haptic effect in autism. In N. Shaughnessy (Ed.), *Affective performance and cognitive science: Body, brain and being*. London: Methuen.
- Trowsdale, J., & Hayhow, R. (2013). Can mimetics, a theatre-based practice, open possibilities for young people with learning disabilities? A capability approach. *British Journal of Special Education*, 40(2), 72–79.
- Vickers, S. (2005). *Drama scripts for people with special needs: Inclusive drama for PMLD, autistic spectrum and special needs group*. London: Speechmark.
- Vygotsky, L. V. (1978). *Mind in Society: The development of higher psychological processes*. Cambridge, MA: Harvard University Press.
- Wilmer-Barbrook, C. (2013). Adolescence, Asperger's and acting: Can drama therapy improve social and communication skills for young people with Asperger's syndrome? *Dramatherapy*, 35(1), 43–56.
- Wolfberg, P., Bottema-Beutel, K., & DeWitt, M. (2012). Including children with autism in social and imaginary play with typical peers. Integrated play groups model. *American Journal of Play*, 5(1), 55–80.
- Zeisel, J. (2011). Letter and petition to President Obama, 'I'm still here'. Retrieved Apr 2014 from www.huffingtonpost.com/john-zeisel-phd/dementia-obama_b_933599.html

Driving Hazard Perception in ASD

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Synonyms

Situation awareness

Definition

Driving hazard perception is the ability to identify potentially dangerous events on the roads. It is typically tested by presenting participants with a series of video clips containing driving hazards filmed from the point of view of a driver. Participants are asked to press a button whenever they see a hazard developing and their accuracy and reaction time in doing so is measured. Hazard perception testing is based on the premise that the earlier a developing hazard is perceived, the longer the driver has for making a response, such as steering out of the way or slowing down; and consequently, the less likely an accident will occur. In line with this, hazard perception reaction time as measured in video-based hazard perception tests has been found to predict drivers' on-road accident involvement. In light of this, a hazard perception test of this nature is used as part of driver licensing procedures in addition to the on-road test in several countries including the UK, the Netherlands, and Australia.

Successful hazard perception frequently involves predicting the behavior of other road users. However, behavioral prediction has been reported as problematic for people with ASD (Zalla et al. 2010), raising the question of whether individuals with ASD have difficulty with hazard perception. Some research addressing this question has focused on individuals who have not yet started learning to drive to assess baseline levels of hazard perception skill,

Draw-a-Person Intellectual Ability Test for Children, Adolescents, and Adults

► Human Figure Drawing Tests

using the video-based testing methodology (Sheppard et al. 2010). Participants with ASD tended to be slower at responding to hazards than comparison individuals, and this appears to be due to those with ASD being slower in visually orienting to hazards within the driving scenes, perhaps due to less efficient search strategies (Sheppard et al. 2017). Non-drivers with ASD may also have poorer hazard perception accuracy (i.e., detect fewer hazards overall in such tests) than non-drivers without the condition, particularly when the hazard in question arises from the actions of a clearly visible person (Sheppard et al. 2010). This may relate to the well-documented difficulties attending to and processing dynamic social stimuli in ASD (e.g., Speer et al. 2007).

Research has also investigated hazard perception skill in experienced drivers with ASD using simulator driving, rather than a video-based hazard perception test (Bishop et al. 2017). In this context, the participant was not specifically asked to search for hazards but rather to complete a single simulator drive, during which several hazardous events were pre-programmed to occur at specific points. Hazard perception ability was indexed by measures of how the participant responded when the hazardous events occurred – such as by braking or steering. Drivers without ASD responded faster to hazards that involved a clearly visible person than hazards which did not, while drivers with ASD did not show this effect. However, overall there were few differences between the groups in how they responded to the hazardous events, implying that those with ASD who do drive may have similar hazard perception abilities to drivers without ASD.

While current drivers with ASD demonstrate hazard perception skills commensurate with comparison individuals, the differences in hazard perception skill in non-drivers with ASD might indicate additional challenges in learning to drive for individuals with ASD – especially in countries where hazard perception testing forms a mandatory part of driver licensing. Future research should examine

hazard perception in current learners with ASD to shed light on how hazard perception ability develops in this population during driver tuition.

See Also

- [Action Prediction in Autism](#)
- [Social Cognition](#)

References and Reading

- Bishop, H. J., Biasini, F. J., & Stavrinou, D. (2017). Social and non-social hazard response in drivers with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(4), 905–917.
- Sheppard, E., Ropar, D., Underwood, G., & van Loon, E. (2010). Brief report: Driving hazard perception in autism. *Journal of Autism and Developmental Disorders*, 40(4), 504–508.
- Sheppard, E., van Loon, E., Underwood, G., & Ropar, D. (2017). Attentional differences in a driving hazard perception task in adults with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 47(2), 405–414.
- Speer, L. L., Cook, A. E., McMahon, W. M., & Clark, E. (2007). Face processing in children with autism: Effects of stimulus contents and type. *Autism*, 11(3), 265–277.
- Zalla, T., Labruyère, N., Clément, A., & Georgieff, N. (2010). Predicting ensuing actions in children and adolescents with autism spectrum disorders. *Experimental Brain Research*, 201(4), 809–819.

DSM-5

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Synonyms

[Diagnosis](#)

Definition

The Diagnostic and Statistical Manual (DSM), published by the American Psychiatric Association (APA), is widely used to diagnose psychopathology. The fifth edition of the DSM was published in May of 2013. The DSM-5 differs from its predecessor in that the manual is organized developmentally, beginning with disorders that are present early in life to disorders that are present later in life, rather than a multiaxial system. Autism spectrum disorder (ASD) is in the category of neurodevelopmental disorders. In the previous version of the DSM, ASD was an umbrella term used to house five distinct disorders: autistic disorder, Asperger's syndrome, childhood degenerative disorders, Rett's disorder, and pervasive developmental disorder not otherwise specified. In the current addition, Rett's disorder was completely removed, and the four remaining disorders collapsed into one ASD diagnosis. The rationale behind this change was that one single spectrum better exemplifies the symptoms, course, and treatment of the disorder. Additional changes included combining the socialization domain and the communication domain into one domain; the age of onset criterion was expanded to include early childhood in general, and the number of symptoms required to be met for diagnosis was made stricter.

To meet criteria for ASD, three of three criteria must be met in the social and communication deficits domain (i.e., nonverbal communication, peer relationships, and social reciprocity); two of four criteria must be met in the restricted and repetitive patterns of behavior, interest, and activities domain (i.e., stereotyped or repetitive movements, insistence on sameness, restricted or fixated interests, and hyper- or hyporeactivity to sensory input); symptoms must present early in the developmental period; and symptoms must cause clinically significant impairments. Diagnosis severity is indicated with levels: level one, "requiring support"; level two, "requiring substantial support"; and level three "requiring very substantial support." Medical conditions, intellectual impairments, and language impairments are

recorded as specifiers accompanying the disorder. Comorbid diagnoses which are not part of the ASD diagnostic criteria may be given along with the ASD diagnosis (e.g., ADHD, anxiety disorder, depression; APA 2013).

The changes to the ASD category of DSM-5 have been controversial and met with much resistance. Major concerns have been that the stricter criteria requirements will impact prevalence rates. Some researchers have estimated approximately 30–45 % of individuals currently diagnosed with an ASD will no longer meet criteria under the DSM-5 diagnosis, with those diagnosed with PDD-NOS most likely affected (Matson et al. 2012a, b; McPartland et al. 2012). Loss of diagnosis due to the diagnostic criteria changes will have a significant impact on services and eligibility. To address this concern, DSM-5 makes note that any individuals with an existing diagnosis will not lose existing services if they do not meet the new diagnostic criteria.

See Also

- ▶ [American Psychiatric Association](#)
- ▶ [Diagnosis and Classification](#)
- ▶ [Diagnostic Process](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: APA Press.
- Matson, J. L., Belva, B. C., Horovitz, M., & Bamburg, J. (2012a). Comparing symptoms of autism spectrum disorders in a developmentally disabled adult population using the current DSM-IV-TR diagnostic criteria and the proposed DSM-5 diagnostic criteria. *Journal of Developmental and Physical Disabilities*, 24, 403–414.
- Matson, J. L., Kozlowski, A. M., Hattier, M. A., Horovitz, M., & Sipes, M. (2012b). DSM-IV vs DSM-5 diagnostic criteria for toddlers with autism. *Developmental Neuropsychology*, 15(3), 185–190.
- McPartland, J. C., Reichow, B., & Volkmar, F. R. (2012). Sensitivity and specificity of proposed DSM-5 diagnostic criteria for autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51, 368–383.

DSM-5 and Autism Spectrum Disorder

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Definition

The Diagnostic and Statistical Manual of Mental Disorders, fifth edition, outlines diagnostic criteria for accepted mental disorders and is used in the United States to provide diagnostic codes for treatment. With regard to autism, the current edition of the DSM states that ASD is characterized by persistent deficits in social communication and social interaction across multiple contexts as well as the presence of restricted and repetitive patterns of behavior. Social communication deficits include deficits in social-emotional reciprocity, deficits in nonverbal communicative behaviors, and deficits in the development and maintenance of social relationships. Restricted and repetitive behaviors may include stereotyped motor movements, insistence on sameness, highly restricted interests, or hyper- or hyposensitivity to sensory input. The abovementioned symptoms must be present in the early developmental period in order to merit a diagnosis of ASD.

Historical Background

The first descriptions of the symptoms now collectively known as autism spectrum disorder (ASD) were provided by Kanner (1943) and Asperger (1944). Kanner reported on 11 children who demonstrated an “inability to relate themselves” to others and an “extreme autistic aloneness,” as well as resistance to environmental change (p. 242). One year later, Asperger published a similar description of social isolation, though in a sample of individuals with

substantially greater verbal abilities and interests in highly specialized topics (1944). Subsequent research identified additional individuals with similar constellations of symptoms, separated autism from established disorders such as schizophrenia, and identified a strong genetic component to the disorder due in part to high rates of seizures in individuals with ASD.

As the constellation of symptoms associated with autism became increasingly distinct, researchers began to define its symptoms more clearly. Preliminary efforts to provide specific diagnostic criteria for what is now termed ASD included a definition provided by the National Society for Autistic Children in 1977. These criteria included disturbances in rate of development, abnormal responses to sensations, delayed speech and language, and abnormal ways of relating to people, objects, or events (Ritvo and Freeman 1977). Autism was first included in the DSM with the publication of its third edition, which focused its criteria on “classic autism” and individuals with more severe impairments. Additionally, in order to qualify for a diagnosis, an individual was required to meet all specified criteria (APA 1980). Subsequent revisions introduced more developmentally oriented criteria and became more inclusive for individuals without cognitive deficits.

Autism spectrum disorders changed again with the publication of DSM-IV-TR in 2000. These new criteria were instrumental in facilitating an explosion of research focused on autism spectrum disorders. Despite this virtue, however, these criteria were subject to some notable shortcomings. The category of pervasive developmental disorders included autistic disorder, Asperger’s disorder, and pervasive developmental disorder—not otherwise specified (APA 2000). These three diagnoses allowed for useful distinctions among individuals with varying levels of impairment but also raised questions regarding differential diagnosis and whether “high-functioning autism,” Asperger’s disorder, and PDD-NOS were phenotypically distinct (South et al. 2005). Additionally, some evidence indicated that clinicians used the “not otherwise specified” diagnosis (PDD-NOS)

too broadly and that this diagnosis became a catch-all term for symptoms approaching, but not sufficient for, a diagnosis of Asperger's disorder or autistic disorder (Norbury 2014).

Current Knowledge

The DSM-5 neurodevelopmental disorders workgroup aimed to address many of the noted shortcomings of DSM-IV-TR criteria (APA 2013). The most salient change eliminated the previous category of pervasive developmental disorders and eliminated diagnoses of Asperger's disorder and PDD-NOS in favor of subsuming all autism diagnoses under the ASD label. Another notable difference was the reduction in symptom categories from three domains (i.e., social impairments, communication deficits, and restricted and repetitive behaviors) to two (i.e., social communication and restricted and repetitive behaviors). This change reflected acknowledgment by the workgroup that the distinction between the social and communication symptom domains was somewhat arbitrary and that both were reflective of core deficits attributable to autism (Lord and Jones 2012). Reducing the number of domains, and requiring that all three criteria within the social communication domain were met, greatly reduced the number of symptom combinations that could result in a diagnosis (Volkmar and Reichow 2013). Within the restricted and repetitive behaviors domain, an additional criterion regarding hyper- or hypo-reactivity to sensory stimuli was added. The final substantial change consisted of the addition of a severity specifier, in which individuals are classified as requiring very substantial support, requiring substantial support, or requiring support. These specifiers may provide a means of describing an individual and their needs with a greater level of detail given that new criteria render it possible for individuals of widely varying levels of symptoms and cognitive ability to receive the same diagnosis. The extent to which these severity specifiers will affect research or clinical work remains unclear. Members of the neurodevelopmental disorders workgroup

conducted a detailed analysis of sensitivity and specificity of the finalized new criteria and found compelling evidence to suggest that the vast majority of individuals with DSM-IV-TR PDD diagnoses would retain them under the new criteria and that specificity improved markedly (Huerta et al. 2012).

As published, DSM-5 criteria offered advantages to the old classification system and accomplished stated goals of the neurodevelopmental disorders workgroup. However, a series of studies conducted in the period leading up to and immediately following publication suggested that the new criteria may be problematic in a number of ways. Among the most prominent of these examinations consisted of reevaluation of data collected on nearly 1000 individuals during the field trial for development of DSM-IV criteria. Clinicians reevaluated each case according to the proposed DSM-5 criteria; results suggested that only 60% of individuals classified with ASD under DSM-IV-TR criteria would receive diagnosis when assessed according to proposed DSM-5 criteria (McPartland et al. 2012). Reported specificity, however (i.e., correct identification of individuals who do *not* have ASD), was excellent (95%). A number of additional studies were published demonstrating similar effects of the proposed new criteria. Mattila et al. (2011) found a similarly low sensitivity of new criteria in conducting an epidemiological study of children in Finland.

Results of both of these studies also suggested that certain subsets of the ASD population would be disproportionately affected by these changes. Individuals diagnosed with Asperger's disorder or PDD-NOS under DSM-IV-TR criteria as well as individuals without intellectual disability (i.e., IQ >70) were more likely to fail to meet diagnostic criteria per DSM-5 than those with autistic disorder or intellectual disability. Other studies found similar reductions in the number of overall individuals diagnosed using the new criteria (Matson et al. 2012b). Analyses of samples of individuals meeting versus not meeting DSM-5 criteria suggested that individuals meeting DSM-5

criteria represented a more impaired population with higher levels of core symptoms than those meeting DSM-IV-TR, but not DSM-5, criteria (Matson et al. 2012a).

Reviews and meta-analyses of studies exploring changes in rates of ASD diagnoses based on DSM-5 criteria presented substantial evidence that considerable numbers of individuals would fail to qualify for diagnoses under new criteria and that individuals with high IQs and those previously diagnosed with Asperger's or PDD-NOS would be disproportionately affected (Kulage et al. 2014; Sturmey and Dalfern 2014; Smith et al. 2015). This evidence, however, must be considered along with a number of notable limitations. The majority of the studies described above relied on retrospective data, collected prior to the development and proposal of DSM-5 criteria. As a result, available data did not allow for comprehensive assessment by new criteria, potentially affecting reported sensitivity and specificity.

Publication of the studies described above resulted in considerable concern within the ASD research community. Changes in the set of symptoms considered under the ASD label created a number of potential difficulties with the application of criteria, both in research and clinical practice. If the population of individuals characterized as having ASD under DSM-5 differed substantially from those diagnosed with a disorder under the ASD umbrella in DSM-IV-TR, prior research conducted on the latter group might have limited application to individuals with a different constellation of symptoms (i.e., generally more severely affected). Questions regarding application of the new criteria also arose with regard to clinical practice and access to services. Prior to official publication, the proposed criteria made it theoretically possible for an individual diagnosed with autistic disorder, Asperger's, or PDD-NOS to fail to qualify for a diagnosis under the new criteria, potentially limiting that individual's access to treatment and support services. As a result, a stipulation was added to DSM-5 criteria stating that any individual qualifying for a diagnosis under DSM-IV-TR would automatically be

assigned the label of ASD under DSM-5, regardless of whether or not the new criteria were met.

While providing a single label for all autism spectrum disorders clarified distinctions among diagnostic subtypes and increased parsimony in the ASD research community, the effects of these terminology changes on individuals with ASD and their families may be considerable. First, families who have not kept abreast of changes within the research community were likely unaware that the label assigned to a given individual may have changed with the publication of DSM-5 (e.g., an individual with Asperger's now technically meriting a diagnosis of ASD). This offers the potential for confusion when families seek assessment or treatment, as providers are likely to have shifted in their usage of terminology with the publication of the new criteria, while families continue to use DSM-IV-TR labels. Second, and perhaps more importantly, new terminology may be unwelcome or even offensive to communities of individuals who identified strongly with previously used labels. Specifically, some individuals with Asperger's have demonstrated a strong affiliation with that term and its variants. This term and others have been used by the neurodiversity movement, which advocates against pathologizing ASD, especially by the scientific community; instead, they aim to characterize ASD as a different manner of thinking and not as a "disorder" *per se* (Robertson and Ne'eman 2008).

Another substantial change to the neurodevelopmental disorders section of the DSM in its fifth edition was the addition of social (Pragmatic) communication disorder (SCD). SCD is closely related to ASD in that its diagnosis requires deficits in the use of communication for social purposes, including social pragmatics and turn-taking (APA 2013). However, notably absent from diagnostic criteria is the presence of restricted and repetitive behaviors. The SCD diagnosis should be considered here as a new, independent neurodevelopmental disorder with a unique phenotype and associated symptoms.

However, this new diagnosis should also be viewed within the context of the debate surrounding new DSM criteria. Some researchers have

suggested that SCD, with a symptom set more limited than ASD, will occupy the same role in diagnosis that PDD-NOS did in DSM-IV-TR (i.e., essentially a milder form of autism). In this manner, the SCD diagnosis would constitute somewhat of a catch-all, and its validity could be called into question. Others have proposed that SCD should be considered in relation to the substantial number of individuals who may “lose” diagnoses in the transition from DSM-IV-TR to DSM-5. The possibility exists that individuals no longer meeting full criteria for ASD would be found not to meet criteria only in the restricted and repetitive behavior domain, and thus these individuals would be diagnosed with SCD. A final point to be made regarding SCD is that there exists some question regarding whether or not SCD can be considered a distinct phenotype, that is, whether it is possible that social pragmatic communication deficits appear in the absence of accompanying restricted and repetitive behaviors (Norbury 2014). As this diagnosis becomes more solidified with their application in research and clinical practice, the field will approach answers to these questions, as well as begin to identify appropriate services and interventions for individuals in this population.

Future Directions

Despite the controversy and limitations described above, DSM-5 criteria for ASD are, and will remain, the current valid and accepted criteria for ASD. Despite the increasing period of time that has passed since the publication of the new criteria, researchers have continued to examine the application of the criteria in new or different settings and question their utility (e.g., Bent et al. 2017). Although there exists substantial evidence to support modification of the current criteria to remedy some of the documented shortcomings of DSM-5 criteria, changes appear unlikely. Criteria published in previous versions of the DSM have also resulted in disagreement among the scientific community, and disputes arising from their publication did not serve to limit progress in ASD

research in any substantial way; DSM-5 criteria are not likely to represent a unique case.

Although future versions of the DSM may improve upon current criteria and allow for more accurate, sensitive, and specific diagnoses of ASD at earlier ages, the inherent limitations of behavioral criteria in diagnosing a neurodevelopmental disorder suggest that the ultimate goal must be a reliable and valid biomarker that can be used to diagnose ASD, even prior to the emergence of behavioral symptoms. A number of potential biological indicators predictive of diagnosis of ASD have been explored. Identification of biomarkers would be beneficial as a means of objective diagnosis but also in the ability of researchers to objectively measure treatment response. Neuroimaging techniques such as fMRI have also been considered as a potential means of making more objective diagnoses. The use of biomarkers is an emerging research area, with ongoing multisite trials contributing to significant advances. Recent evidence suggests that hyperexpansion of the cortical surface area in infants between 6 and 12 months of age was associated ASD diagnoses later in life (Hazlett et al. 2017). This and other emerging evidence suggests that the use of biomarkers for diagnosis offers considerable advantages over current behavioral criteria. Despite these advances, identification of clear and objective biomarkers that could be easily applied (i.e., low-cost and widely available) is likely too distant to consider as the next step in diagnosis of ASD. Future revisions of DSM-5 will likely update diagnostic criteria to remain current with advances in research providing a clearer conceptualization of symptoms that constitute ASD.

See Also

- ▶ [Alternative Diagnostic Concepts](#)
- ▶ [Asperger Syndrome](#)
- ▶ [Autism Diagnostic Interview-Revised](#)
- ▶ [Autism Diagnostic Observation Schedule](#)
- ▶ [Autism Diagnostic Observation Schedule \(ADOS\): Toddler Module](#)
- ▶ [DSM-5](#)

- ▶ DSM-IV Field Trial
- ▶ High-Functioning Autism (HFA)
- ▶ Spectrum/Continuum of Autism

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders*. Washington, DC: APA Press.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: APA Press.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: APA Press.
- Asperger, H. (1944). Die “autistischen Psychopathen” im Kindesalter. *Archiv Fur Psychiatrie Und Nervenkrankheiten*, 117, 76–136.
- Bent, C. A., Barbaro, J., & Dissanayake, C. (2017). Change in autism diagnoses prior to and following the introduction of DSM-5. *Journal of Autism and Developmental Disorders*, 47, 1–9. <https://doi.org/10.1007/s10803-016-2942-y>.
- Hazlett, H. C., Gu, H., Munsell, B. C., Kim, S. H., Synter, M., Wolff, J. J., & IBIS Network. (2017). Early brain development in infants at high risk for autism spectrum disorder. *Nature*, 542, 348–351.
- Huerta, M., Bishop, S. L., Duncan, A., Hus, V., & Lord, C. (2012). Application of DSM-5 criteria for autism spectrum disorder to three samples of children with DSM-IV diagnoses of pervasive developmental disorders. *American Journal of Psychiatry*, 169(10), 1056–1064. <https://doi.org/10.1176/appi.ajp.2012.12020276>.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kulage, K. M., Smaldone, A. M., & Cohn, E. G. (2014). How will DSM-5 affect autism diagnosis? A systematic literature review and meta-analysis. *Journal of Autism and Developmental Disorders*, 44(8), 1918–1932. <https://doi.org/10.1007/s10803-014-2065-2>.
- Lord, C., & Jones, R. M. (2012). Re-thinking the classification of autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 53(5), 490–509. <https://doi.org/10.1111/j.1469-7610.2012.02547.x>. Re-thinking.
- Matson, J. L., Belva, B. C., Horovitz, M., Kozlowski, A. M., & Bamburg, J. W. (2012a). Comparing symptoms of autism Spectrum disorders in a developmentally disabled adult population using the current DSM-IV-TR diagnostic criteria and the proposed DSM-5 diagnostic criteria. *Journal of Developmental and Physical Disabilities*, 24(4), 403–414. <https://doi.org/10.1007/s10882-012-9278-0>.
- Matson, J. L., Kozlowski, A. M., Hattier, M. A., Horovitz, M., & Sipes, M. (2012b). DSM-IV vs DSM-5 diagnostic criteria for toddlers with autism. *Developmental Neurorehabilitation*, 15(3), 185–190. <https://doi.org/10.3109/17518423.2012.672341>.
- Mattila, M. L., Kielinen, M., Linna, S. L., Jussila, K., Ebeling, H., Bloigu, R., et al. (2011). Autism spectrum disorders according to DSM-IV-TR and comparison with DSM-5 draft criteria: An epidemiological study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 50(6), 583–592. <https://doi.org/10.1016/j.jaac.2011.04.001>.
- McPartland, J. C., Reichow, B., & Volkmar, F. R. (2012). Sensitivity and specificity of proposed DSM-5 diagnostic criteria for autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(4), 368–383.
- Norbury, C. F. (2014). Practitioner review: Social (pragmatic) communication disorder conceptualization, evidence and clinical implications. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 55(3), 204–216. <https://doi.org/10.1111/jcpp.12154>.
- Ritvo, E. R., & Freeman, B. J. (1977). National society for autistic children definition of the syndrome of autism. *Journal of Pediatric Psychology*, 2(4), 146–148.
- Robertson, S. M., & Ne’eman, A. D. (2008). Autistic acceptance, the college campus, and technology: Growth of neurodiversity in society and academia. *Disability Studies Quarterly*, 28(4), 14.
- Smith, I. C., Reichow, B., & Volkmar, F. R. (2015). The effects of DSM-5 criteria on number of individuals diagnosed with autism Spectrum disorder: A systematic review. *Journal of Autism and Developmental Disorders*, 45(8), 2541–2552. <https://doi.org/10.1007/s10803-015-2423-8>.
- South, M., Ozonoff, S., & McMahon, W. M. (2005). Repetitive behavior profiles in asperger syndrome and high-functioning autism. *Journal of Autism and Developmental Disorders*, 35(2), 145–158. <https://doi.org/10.1007/s10803-004-1992-8>.
- Sturmey, P., & Dalfern, S. (2014). The effects of DSM5 autism diagnostic criteria on number of individuals diagnosed with autism spectrum disorders: A systematic review. *Review Journal of Autism and Developmental Disorders*, 1(4), 249–252. <https://doi.org/10.1007/s40489-014-0016-7>.
- Turygin, N., Matson, J. L., Beighley, J., & Adams, H. (2013). The effect of DSM-5 criteria on the developmental quotient in toddlers diagnosed with autism spectrum disorder. *Developmental Neurorehabilitation*, 16(1), 38–43. <https://doi.org/10.3109/17518423.2012.712065>.
- Volkmar, F. R., & Reichow, B. (2013). Autism in DSM-5: Progress and challenges. *Molecular Autism*, 4(1), 13. <https://doi.org/10.1186/2040-2392-4-13>.
- Volkmar, F. R., Reichow, B., & McPartland, J. (2012). Classification of autism and related conditions: Progress, challenges, and opportunities. *Dialogues in Clinical Neuroscience*, 14(3), 229–237.

DSM-III

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Definition

The 3rd edition of the *American Psychiatric Association's Diagnostic and Statistical Manual* (DSM-III) appeared in 1980 and proved to be a landmark in the development of psychiatric taxonomy.

Historical Background

Beginning in the 1800s, attempts had begun to be made in categorizing and studying rates of various disorders (including psychiatric disorders) in the USA and other countries. For psychiatry, an attempt was made to codify a specific approach in the early 1900s and was subsequently revised. These early attempts included only a small of categories. However, following World War II, a concern for providing mental health services in a more systematic fashion (and including to the many returning veterans) provided a stimulus for the first edition of DSM. This effort reflected both a growing awareness of the mental health issues in the military in the USA and the stimulus provided by a revision (the 6th edition) of the *International Statistical Classification of Diseases* (ICD) which recognized mental disorders for the first time. A second edition was undertaken in the 1960s, and DSM-II recognized over 180 disorders. Both the first and second editions of DSM adopted a specific theoretical (e.g., psychodynamic) framework. Developments more broadly in the field (particularly at Washington University in St. Louis and subsequently at Columbia University) led to a focus on less theoretical and more descriptive definitions useful for research. This research diagnostic criteria approach (Spitzer

et al. 1978) proved particularly helpful, and the decision was made for the third edition of DSM to move toward such an approach as well as greater convergence with the ICD (Spitzer et al. 1978).

Current Knowledge

Changes were made in DSM-III based on a series of consideration including issues of reliability and results of “field trials.” By the time it was published in the 1980s, DSM-III included over 260 categories and quickly proved invaluable in transforming research in psychiatry. Although not without controversy (e.g., around use of terms like neurosis and the approach to diagnosis of sexual orientation problems) for childhood-onset disorders, DSM-III had several important advantages. It recognized disorders like autism for the first time (previously the term childhood schizophrenia was the only “official” diagnosis available) based on a series of papers in the 1970s demonstrating the unique diagnostic features and course of children with autism (Kolvin 1971; Rutter 1972). Autism was included along with several other disorders in a newly class of conditions – the pervasive developmental disorders (PDD). Disorders recognized within this class included infantile autism, residual infantile autism (for individuals who once met full criteria for the infantile form of the disorder), childhood-onset pervasive developmental disorder (COPDD) (to encompass the rare individuals who developed autism or something very like it after several years of normal development), and a residual COPDD category along with a new subthreshold condition atypical PDD. The class name *pervasive developmental disorder* was used to refer to what today might more usually be termed autism spectrum disorders. The PDD term was meant to convey that a range of functions were impacted, although the term itself was widely debated (e.g., see Gillberg 1991; Volkmar and Cohen 1991b).

Apart from the explicit recognition of autism as a specific and valid diagnostic category, the DSM-III had several important advantages for childhood disorders in general including the use of multiple axes of diagnosis (psychiatric,

developmental, medical, and psychosocial stressors and overall adaptive functioning). This multiple axial approach proved especially helpful for child psychiatry (Rutter et al. 1975). The use of more detailed and specific definitions without a specific theoretical bias also enhanced reliability. The *DSM-III* system also had some disadvantages for autism. The proposed was based largely on Rutter's modification (Rutter 1978) of Kanner's (1943) original description, but in the attempt to make this more operational, the monothetic definition adopted focused on what might now be thought of as more prototypical ("classical") autism, i.e., more "infantile" autism (consistent with the name chosen). For example, the social criterion emphasized a pervasive lack of social responsiveness. This effectively meant that for many children who developed (to varying degrees) greater social skills, the clinician was technically forced to use the "residual" autism category. A problem with this lack of developmental orientation was the implicit implication that somehow their problems were less severe. Similarly, the rationale for COPDD as a category was to account for the small number of children who had developed an autistic-like disorder at a somewhat later point in early childhood (Kolvin 1971); COPDD was not, however, meant to be simply with Heller's syndrome (disintegrative psychosis) (Heller 1908, 1930) as it was assumed, probably incorrectly, that the latter was invariably a result of a general medical/neurological process (see Volkmar and Rutter 1995). Similarly, the term *atypical PDD* was used as a placeholder for the subthreshold condition ("autistic-like" or now "autism spectrum disorder") for difficulties that appeared to be best thought of as occurring within the overarching PDD class but meeting criteria for infantile autism or COPDD. Unfortunately, this term had its own prior history in that it was suggestive of Rank's earlier concept of atypical personality (Rank 1949; Rank and MacNaughton 1949). Another problem arose because of the recognition that autism was *not* a kind of schizophrenia and the adoption of an exclusionary rule for autism and schizophrenia; on the other hand, one might reasonably argue that adolescents and adults with autism are not necessarily protected

from this condition in later life – at rates presumably at least comparable to those of the general population (Volkmar and Cohen 1991b). Finally the multi-axial placement of autism and related disorders was somewhat controversial. Autism and mental retardation (intellectual disabilities) were by convention made axis I diagnosis, while the specific developmental disorders were placed on axis II of the multi-axial system. The problems with *DSM-III* were widely recognized, and a major revision was undertaken for *DSM-III-R* (American Psychiatric Association 1980, 1987).

See Also

- [DSM-III-R](#)
- [DSM-IV](#)
- [DSM-IV Field Trial](#)
- [ICD 10 Research Diagnostic Guidelines](#)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Gillberg, C. (1991). Debate and argument: Is autism a pervasive developmental disorder? *Journal of Child Psychology and Psychiatry*, 32(7), 1169–1170.
- Heller, T. (1908). Dementia infantilis. *Zeitschrift für die Erforschung und Behandlung des jugenlichen Schwachsinn*, 2, 141–165.
- Heller, T. (1930). Über dementia infantilis. *Zeitschrift für Kinderforschung*, 37, 661–667.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kolvin, I. (1971). Studies in the childhood psychoses. I. Diagnostic criteria and classification. *The British Journal of Psychiatry*, 118(545), 381–384.
- Rank, B. (1949). Adaptation of the psychoanalytic technique for the treatment of young children with atypical development. *The American Journal of Orthopsychiatry*, 19, 130–139. American Psychological Assn/Educational Publishing Foundation, US.
- Rank, B., & MacNaughton, D. (1949). A clinical contribution to early ego development. In A. Freud, H. Hartmann, et al. (Eds.), *The psychoanalytic study of the child* (Vol. 3/4, pp. 53–65). Oxford: International Universities Press.
- Rutter, M. (1972). Childhood schizophrenia reconsidered. *Journal of Autism and Childhood Schizophrenia*, 2(4), 315–337.

- Rutter, M. (1978). Diagnosis and definition of childhood autism. *Journal of Autism and Childhood Schizophrenia*, 8(2), 139–161.
- Rutter, M., Shaffer, D., & Shepherd, M. (1975). *A multi-axial classification of child psychiatric disorders: An evaluation of a proposal*. Albany: World Health Organization.
- Spitzer, R. L., Endicott, J. E., & Robins, E. (1978). Research diagnostic criteria. *Archives of General Psychiatry*, 35, 773–782.
- Volkmar, F. R., & Cohen, D. J. (1991a). Comorbid association of autism and schizophrenia. *The American Journal of Psychiatry*, 148(12), 1705–1707.
- Volkmar, F. R., & Cohen, D. J. (1991b). Debate and argument: The utility of the term pervasive developmental disorder. *Journal of Child Psychology and Psychiatry*, 32(7), 1171–1172.
- Volkmar, F. R., & Rutter, M. (1995). Childhood disintegrative disorder: Results of the DSM-IV autism field trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 34(8), 1092–1095.

DSM-III-R

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Definition

The successor to DSM-III (appearing in 1987) work on this edition began shortly after DSM-III (1980) had appeared. Originally viewed as a small-scale revision, major changes were made in several categories including autism and related conditions (see Waterhouse et al. 1993). The definition of autistic disorder (name changed from infantile autism in DSM-III) was more consistent with the views of Lorna Wing and her colleagues (e.g., see Wing 1981) who adopted a somewhat broader diagnostic view of the concept. This revision also put much greater weight on developmental changes discarding the earlier concept of “residual” infantile autism and replacing it with a single category with criteria applicable to the entire range of functioning over age and developmental level. Consistent with the previous

definition, the three major domains of dysfunction (social, communicative, and restricted interests/behaviors) were included, although early age of onset was no longer required. Final scoring rules were developed based on a field trial (Spitzer and Siegel 1990) with 16 criteria; a diagnosis of autism required that an individual exhibited at least 8 of these features (with a specified distribution over the 3 areas). The problematic earlier concept of childhood-onset pervasive developmental disorder (COPDD) was dropped, and throughout the manual, the earlier term “atypical” was replaced with “not otherwise specified” (in large part because of the potential confusion with an earlier diagnostic concept – atypical personality development; see (Volkmar and Klin 2005)).

The greater developmental orientation of the approach was welcomed but also appeared to come at a price. Several reports suggested high rates of false positives – particularly relative to more intellectually disabled individuals; this led to an apparent overdiagnosis of autism in more intellectually handicapped individuals while also diverting attention from more cognitively able persons. Additional problems included a complex and detailed criteria set with inclusion of examples within criteria (thus tending to “reify” the examples as features that should be present). The changes introduced complicated interpretation of previous research and were particularly acute relative to pending changes in the ICD (see Volkmar and Klin 2005; Volkmar et al. 1992).

See Also

- [DSM-III](#)
- [False Positive](#)
- [ICD 10 Research Diagnostic Guidelines](#)

References and Reading

- American Psychiatric Association. (1987). *Diagnostic and statistical manual* (3rd revised ed.). Washington, DC: APA Press.
- Spitzer, R. L., & Siegel, B. (1990). The DSM-III-R field trial of pervasive developmental disorders. *Journal of*

- the *American Academy of Child and Adolescent Psychiatry*, 29(6), 855–862.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 5–41). Hoboken: Wiley.
- Volkmar, F. R., Cicchetti, D. V., Bregman, J., & Cohen, D. J. (1992). Three diagnostic systems for autism: DSM-III, DSM-III-R, and ICD-10. *Journal of Autism and Developmental Disorders*, 22(4), 483–492.
- Waterhouse, L., Wing, L., Spitzer, R., & Siegel, B. (1993). Diagnosis by DSM-III-R versus ICD-10 criteria. *Journal of Autism and Developmental Disorders*, 23(3), 572–573.
- Wing, L. (1981). Language, social, and cognitive impairments in autism and severe mental retardation. *Journal of Autism and Developmental Disorders*, 11(1), 31–44.

DSM-IV

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Definition

The fourth edition of the *American Psychiatric Association's Diagnostic and Statistical Manual* was published in 1994 with a subsequent text revision published in 2000. The publication of DSM-IV followed several years of preparation. For autism and related disorders, the definitions proposed were, for the first time, convergent with the International Classification of Diseases 10th Edition (ICD-10). The fourth edition marked some important changes from its predecessors while maintaining much in the way of historical continuity.

Historical Background

Preparations for the fourth edition of DSM began shortly after DSM-III-R (APA 1987) due, in part, to the pending revision of ICD-10, and this edition

appeared partly due to the pending changes in the ICD-10. The intention was to base the revision on research and with consideration of relevant issues such as clinical utility, reliability, and descriptive validity of categories and the issue of coordination with ICD-10 (WHO 1994). As part of this process, a series of literature reviews were conducted with emphasis on categories “new” to DSM. On balance, these reviews suggested the potential usefulness of including categories in addition to autism within the overarching pervasive developmental disorder (PDD) group (see Volkmar and Klin (2005)) and also supported the desire for compatibility with ICD-10. As part of this process, a series of data reanalyses that were undertaken focused on autism, and these suggested the DSM-III-R definition to be overly broad (Volkmar et al. 1992). Other issues identified included the inclusion (or not) of early age of onset as an essential feature and the variability of sensitivity/specificity in relation to IQ and other variables, and to address these concerns, a large, international field trial was undertaken in conjunction with ICD-10.

Current Knowledge

The DSM-IV Field Trial

The final DSM-IV definition was based on the results of the DSM-IV field trial which included 21 sites from around the world, over 100 raters, and nearly 1,000 cases (Volkmar et al. 1994). Cases were sometimes rated more than once (for reliability), and other issues (e.g., rater experience) were addressed. In general, cases were rated as seen over the course of a year but with some supplementation of previously seen cases for certain issues (e.g., children with “late-onset” autism). Cases could be included if the clinician believed autism to reasonably be part of the differential diagnosis. Multiple sources of information (assessment, history) were available to the raters who judged the quality of data available to them good or excellent about 75% of the time. A standard coding system was used with information on the case and rater as well as ratings of various diagnostic criteria. The DSM-III approach

DSM-IV, Table 1 ICD-10 criteria for autism and related pervasive developmental disorders

Childhood autism (F84.0)	
(A) Abnormal or impaired development is evident before the age of 3 years in at least one of the following areas:	1. Receptive or expressive language as used in social communication
	2. The development of selective social attachments or of reciprocal social interaction
	3. Functional or symbolic play
(B) A total of at least six symptoms from (1), (2), and (3) must be present, with at least two from (1) and at least one from each of (2) and (3)	1. Qualitative impairments in social interaction are manifest in at least two of the following areas:
	(a) Failure adequately to use eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
	(b) Failure to develop (in a manner appropriate to mental age, and despite ample opportunities) peer relationships that involve a mutual sharing of interests, activities, and emotions
	(c) Lack of socio-emotional reciprocity as shown by an impaired or deviant response to other people's emotions; or lack of modulation of behavior according to social context; or a weak integration of social, emotional, and communicative behaviors
	(d) Lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., a lack of showing, bringing, or pointing out to other people objects of interest to the individual)
	2. Qualitative abnormalities communication as manifest in at least one of the following areas:
	(a) Delay in or total lack of development of spoken language that is not accompanied by an attempt to compensate through the use of gestures or mime as an alternative mode of communication (often preceded by a lack of communicative babbling)
	(b) Relative failure to initiate or sustain conversational interchange (at whatever level of language skill is present), in which there is reciprocal responsiveness to the communications of the other person
	(c) Stereotyped and repetitive use of language or idiosyncratic use of words or phrases
	(d) Lack of varied spontaneous make-believe play or (when young) social imitative play
	3. Restricted, repetitive, and stereotyped patterns of behavior, interests, and activities are manifested in at least one of the following:
	(a) An encompassing preoccupation with one or more stereotyped and restricted patterns of interest that are abnormal in content or focus, or one or more interests that are abnormal in their intensity and circumscribed nature though not in their content or focus
	(b) Apparently compulsive adherence to specific, nonfunctional routines or rituals
	(c) Stereotyped and repetitive motor mannerisms that involve either hand or finger flapping or twisting or complex whole-body movements
	(d) Preoccupations with part objects or nonfunctional elements of play materials (such as their odor, the feel of their surface, or the noise or vibration they generate)

(continued)

DSM-IV, Table 1 (continued)

(C) The clinical picture is not attributable to the other varieties of pervasive developmental disorders; specific development disorder of receptive language (F80.2) with secondary socio-emotional problems' reactive attachment disorder (F94.1) or disinhibited attachment disorder (F94.2); mental retardation (F70-F72) with some associated emotional or behavioral disorders; schizophrenia (F20) of unusually early onset; and Rett's syndrome (F84.12)	
F84.1 Atypical autism	
A pervasive developmental disorder that differs from autism in terms <i>either</i> of age of onset <i>or</i> of failure to fulfill all three sets of diagnostic criteria. Thus, abnormal and/or impaired development becomes manifest for the first time only after age 3 years, and/or there are insufficient demonstrable abnormalities in one or two of the three areas of psychopathology required for the diagnosis of autism (namely, reciprocal social interactions, communication, and restrictive, stereotyped, repetitive behavior) in spite of characteristic abnormalities in the other area(s). Atypical autism arises most often in profoundly retarded individuals whose very low level of functioning provides little scope for exhibition of the specific deviant behaviors required for the diagnosis of autism; it also occurs in individuals with a severe specific developmental disorder of receptive language. Atypical autism thus constitutes a meaningfully separate condition from autism and includes: Atypical childhood psychosis Mental retardation with autistic features	
F84.1 Atypical autism	(A) Abnormal or impaired development is evident at or after the age of 3 years (criteria as for autism except for age of manifestation) (B) There are qualitative abnormalities in reciprocal social interaction or in communication, or restricted, repetitive, and stereotyped patterns of behavior, interests, and activities (criteria as for autism except that it is unnecessary to meet the criteria for number of areas of abnormality) (C) The disorder does not meet the diagnostic criteria for autism (F84.0). Autism may be atypical in either age of onset (F84.10) or symptomatology (F84.11); the two types are differentiated with a fifth character for research purposes. Syndromes that are typical in both respects should be coded F84.12
F84.10 Atypicality in age of onset	(A) The disorder does not meet criterion A for autism (F84.0); that is, abnormal or impaired development is evident only at or after age 3 years (B) The disorder meets criteria B and C for autism (F84.0)
F84.11 Atypicality in symptomatology	(A) The disorder meets criterion A for autism (F84.0); that is, abnormal or impaired development is evident before age 3 years

(continued)

DSM-IV, Table 1 (continued)

	(B) There are qualitative abnormalities in reciprocal social interactions or in communication, or restricted, repetitive, and stereotyped patterns of behavior, interests, and activities (criteria as for autism except that it is unnecessary to meet the criteria for number of areas of abnormality)
	(C) The disorder meets criterion C for autism (F84.0)
	(D) The disorder does not fully meet criterion B for autism (F84.0)
F84.12 Atypicality in both age of onset and symptomatology	(A) The disorder does not meet criterion A for autism (F84.0); that is, abnormal or impaired development is evident only at or after age 3 years
	(B) There are qualitative abnormalities in reciprocal social interactions or in communication, or restricted, repetitive, and stereotyped patterns of behavior, interests, and activities (criteria as for autism except that it is unnecessary to meet the criteria for number of areas of abnormality)
	(C) The disorder meets criterion C for autism (F84.0)
	(D) The disorder does not fully meet criterion B for autism (F84.0)

Source: World Health Organization (2003)

was noted to be developmentally less sensitive than DSM-III-R, although that system appeared to overdiagnose autism in individuals with more severe intellectual handicap (i.e., relative to clinician judgment). The ICD-10 draft approach appeared more reasonable although overly detailed. Reliability of criteria was generally good with clinical diagnosis also noted to have excellent reliability for more experienced clinicians (Klin et al. 2000). A series of analyses suggested that a modification of the draft ICD-10 approach could be adopted with reasonable sensitivity and specificity and good coverage over the IQ range.

Although not primarily focused on disorders other than autism, the field trial also provided data regarding the inclusion and definition of Asperger’s disorder, Rett’s disorder, and childhood disintegrative disorder. The final DSM-IV definition had good sensitivity and specificity

over the IQ range. Diagnostic criteria adopted were essentially the same as in ICD-10 (see Table 1). At least six criteria had to be rated positive for a diagnosis of autism with at least two of these from the “social” category (this was consistent with Kenner’s original view of autism and also with a series of data analyses that confirmed the importance of social features). At least one feature must be present from the other two groups (impaired communication/play and restricted interests). Onset before age 3 was also specified.

The inclusion of various condition as well as autism and “subthreshold autism” was a major change from DSM-III-R. Although the substantive work on these other conditions was less advanced than that for autism, there appeared to be sufficient data for their inclusion; this further enhanced compatibility with ICD-10. Convergence of the final ICD-10 and DSM-IV definitions

of autism represented a major shift (i.e., with the same system being used around the world) and facilitated subsequent research and clinical work as reflected, in part, in the explosion of work in the area over the subsequent decade. These criteria are provided in Table 1.

Future Directions

At the time of this writing, preparation for DSM-V was underway with the two major preliminary proposals suggesting a change of the class of disorder to autism spectrum disorder and collapsing the various disorders currently listed in DSM-IV within one overarching disorder type.

See Also

- [DSM-III](#)
- [DSM-III-R](#)
- [ICD 10 Research Diagnostic Guidelines](#)

References and Reading

- American Psychiatric Association. (1987). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Klin, A., Lang, J., Cicchetti, D. V., & Volkmar, F. R. (2000). Brief report: Interrater reliability of clinical diagnosis and DSM-IV. *Journal of Autism and Developmental Disorders*, 30(2), 163–167.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 5–41). Hoboken: Wiley.
- Volkmar, F. R., Cicchetti, D. V., Cohen, D. J., & Bregman, J. (1992). Brief report: Developmental aspects of DSM-III – R criteria for autism. *Journal of Autism and Developmental Disorders*, 22(4), 657–662.
- Volkmar, F. R., Klin, A., Siegel, B., Szatmari, P., Lord, C., Campbell, M., et al. (1994). Field trial for autistic disorder in DSM-IV. *American Journal of Psychiatry*, 151(9), 1361–1367.
- World Health Organization. (1994). *Diagnostic criteria for research*. Geneva: Author.
- World Health Organization. (2003). *Diagnostic descriptions and criteria for autism and related pervasive developmental disorders from international classification of diseases* (10th ed.). Geneva: Author.

DSM-IV Field Trial

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Definition

The DSM-IV field trial for autism (Volkmar et al. 1994) was an international effort with the goal of establishing a definition with a reasonable balance of sensitivity and specificity that could be used to facilitate both clinical work and research. In contrast to previous DSM definitions of autism, this effort was done in conjunction with the ICD-10 revision 21 sites and 125 raters participated from around the world.

Historical Background

The DSM-IV field trial arose as a result of concerns about the definition of autism adopted in DSM-III and DSM-III-R. For the latter definition, a small field trial had been conducted but suffered from some deficiencies. Several published papers suggested that while the DSM-III-R definition was more developmental in nature, it also appeared to be more likely to give an autism diagnosis to individuals with greater levels of intellectual disability.

Current Knowledge

This field trial followed a period of considerable work including targeted literature reviews and data reanalyses (e.g., Frances et al. 1991; Mayes et al. 1993; Szatmari 1991, 1992a, b; Tsai 1992; Volkmar 1991, 1992; Volkmar et al. 1992a, b).

As part of the field trial, 21 sites and 125 raters participated. Each site had some clinical program for individuals with autism and raters with a range of experience and professional backgrounds;

DSM-IV Field Trial, Table 1 DSM-IV autistic disorder field trial group characteristics*

	Clinically autistic (<i>N</i> = 454)	Other PDDs (<i>N</i> = 240)	Non-PDD (<i>N</i> = 283)
Sex ratio (M/F)	4.49:1	3.71:1	2.29:1
% mute	54%	35%	33%
Age	8.99	9.68	9.72
IQ	58.1	77.2	66.9

*Note: Cases grouped by clinical diagnosis
Diagnoses of the “other PDD” cases included: Rett’s syndrome (13 cases), childhood disintegrative disorder (16 cases), Asperger syndrome (48 cases), PDD-NOS (116 cases), and atypical autism (47 cases)
Diagnoses of the non-PDD cases included mental retardation (132 cases), language disorder (88 cases), childhood schizophrenia (9 cases), and other disorders (54 cases)
Adapted from Volkmar et al. (1994). Reprinted from Volkmar et al. Issues in classification, Chapter in F. Volkmar, A. Klin, R. Paul, & D. Cohen (Eds.), *Handbook of Autism and Pervasive Developmental Disorders*, Vol I, page

about half the rates report relatively extensive experience allowing for comparison of issues of reliability and clinical utility in both more and less experienced clinicians. Over the course of a year, nearly 1,000 cases were submitted (with about 10% of cases rated by more than 1 evaluator). In general, the preference was that bases currently being seen be provided (i.e., rather than ratings based on past experience), and by design, 5 of the participating sites contributed about 100 consecutive cases. To be included, the case had to exhibit difficulties in which autism was a reasonable part of the differential diagnosis. Smaller groups of cases were specifically solicited to identify potential gaps relative to much less frequent conditions (e.g., Rett’s and childhood disintegrative disorders). However, consecutive cases constituted the bulk of the sample. Typically multiple sources of information were available (e.g., parents, past records, as well as current assessment), and raters indicated that in the majority (75%) of cases, the information available was good or excellent.

A standard system of coding was created for each deidentified case rating including information on the individual being examined (age, IQ, communicative ability, educational placement), basic information on the evaluator(s), and explicit ratings of diagnostic criteria both from previous (DSM-III, III-R) and potential new criteria. The evaluator was also asked to provide his/her best judgment of clinical diagnosis – the latter serving as the “gold standard” against which comparisons would then be made. The rating form also provided possible criteria for

Asperger’s syndrome, Rett’s syndrome, and childhood disintegrative disorder, based on the draft ICD-10 definitions.

Sample information is presented in Table 1. Clinicians’ primary diagnosis of the “nonautistic” PDD cases included Rett’s syndrome (13 cases), childhood disintegrative disorder (16 cases), Asperger’s syndrome (48 cases), and PDD-NOS (116 cases) or atypical autism (47 cases) (the latter group was included as possible clinical diagnosis given the ICD-10 draft inclusion of such a category which, essentially, referred to “subthreshold” autism or autism that was atypical in somewhat – cases that in US terminology would have been said to have PDD-NOS). In comparison (nomad), cases with primary clinical diagnoses included mental retardation (*N* = 132), language disorder (*N* = 88 cases), schizophrenia of childhood onset (*N* = 9), and other or mixed developmental disorders (*N* = 54).

A series of cases were addressed in a range of different analyses, and in addition to the main report of the field trial (Volkmar et al. 1994), many of these analyses were published in their own right. Consistent with previous reports, it appeared that the DSM-III approach was insufficiently developmental and overly stringent, whereas the DSM-III-R erred on the site of over-diagnosis of autism associated with more severe intellectual deficiency. An explicit goal for the development of final criteria set was that the final criteria for autistic disorder should work reasonably well over the entire range of IQ. A result is more consistent with the much more

DSM-IV Field Trial, Table 2 Sensitivity/specificity by IQ level – a comparison of DSM-III^a DSM-III-R ICD-10^b

Overall		Se	Sp	Se	Sp	Se	Sp
		0.82	0.8	0.86	83	0.79	89
By IQ level	N	Se	Sp	Se	Sp	Se	Sp
<25	64	0.90	0.76	0.84	0.39	0.74	0.88
25–39	148	0.88	0.76	0.90	0.60	0.88	0.92
40–54	191	0.79	0.76	0.93	0.74	0.84	0.83
55–69	167	0.86	0.78	0.84	0.77	0.78	0.89
70–85	152	0.79	0.81	0.88	0.81	0.74	0.96
>85	218	0.78	0.83	0.78	0.78	0.78	0.91

Table adapted, with permission, from Volkmar et al. (1994). Reprinted from Volkmar et al. Issues in classification, Chapter. In F. Volkmar, A. Klin, R. Paul, & D. Cohen (Eds.), *Handbook of Autism and Pervasive Developmental Disorders*. Vol I, page

^a“Lifetime” diagnosis (current IA or “residual” IA)

^bOriginal ICD-10 criteria and scoring

detailed ICD-10 (research) draft definition. These results are presented in Table 2.

In looking at other sources of diagnostic disagreement, it appeared that DSM-III-R’s failure to include age of onset as an essential feature contributed to its difficulties, while, on the other hand, its great developmental orientation and flexibility appeared to be a plus. Indeed, if onset of the condition by age 3 years was included as an essential feature, the sensitivity of that system was increased.

The available data also allowed for examination of clinician agreement on diagnosis based on various factors including clinician experience. Using chance-corrected statistics, the inter-rater reliability of individual diagnostic criteria was examined and in the good to excellent range. Typically more detailed criteria had greater reliability. More experienced raters also had excellent reliability both on the broader autism spectrum and narrower autistic disorder diagnoses; this was much less true for inexperienced raters (see Klin et al. 2000). In addition to inter-rater reliability, temporal stability was examined in a small number of cases and generally high; diagnosis stability was more problematic for very young children, those with lower IQ, and with the DSM-III-R system.

Before a final decision could be made about the definition of autistic disorder in DSM-IV, it was important to decide whether other disorders would be included in the PDD class (see Szatmari

1992a, b; Tsai 1992; Volkmar 1992; Volkmar et al. 1992a). While these disorders were not a primary focus of the field trial, some data relevant to their inclusion had been collected. Data from the field trial supported the inclusion of these conditions (Asperger’s disorder, Rett’s disorder, and childhood disintegrative disorder), and their inclusion had some advantages relative to compatibility with ICD-10 and for deriving a better diagnostic approach for autistic disorder. One area of difference between ICD-10 and DSM-IV was left unresolved; this had to do with the concept of atypical autism vs. PDD-NOS. ICD-10 allowed for more detailed coding of the “atypicality” of the presentation, e.g., failing to meet age or specific criteria cutoffs, while DSM-IV adopted a broader view of this as a “subthreshold” category that, today, would be equated with autism spectrum disorder.

The field trial data suggested that age of onset as an additional feature would have strengthened the DSM-III-R definition. A series of alternatives were considered, and in the end, a final definition was developed in coordination with IDD-10 (see “Appendix”). This definition balanced clinical and research needs, was reasonably concise and user-friendly, and had good coverage over both age and developmental level.

Several changes in proposal for other categories (notably Asperger’s disorder and PDD-NOS) were made in the final stages of the DSM process; these have raised other issues of concern to the

field (e.g., see Buitelaar et al. 1999; Miller and Ozonoff 1997). Some of these concerns were addressed in the DSM-IV text revision, which appeared in 2000; for Asperger's disorder, the text was very extensively revised, although no changes in the formal criteria were made.

The rather extensive field trial undertaken for DSM-IV contrasts markedly from that used in DSM-5 and may explain, in part, the various problems with the latter's approach (Smith et al. 2015).

See Also

- [Asperger's Disorder](#)
- [Autistic Disorder](#)
- [Childhood Disintegrative Disorder](#)
- [DSM-IV](#)
- [Rett's Disorder](#)

Appendix

ICD-10 Research Criteria*. Source: Diagnostic descriptions and criteria for autism and related pervasive developmental disorders from international classification of diseases, 10th Edition (World Health Organization, Geneva, Switzerland, 2003).

	<i>Pervasive developmental disorders</i>
	A group of disorders characterized by qualitative abnormalities in reciprocal social interactions and in patterns of communication and by a restricted, stereotyped, repetitive repertoire of interests and activities. These qualitative abnormalities are a pervasive feature of the individual's functioning in all situations.
	Use additional code, if desired, to identify any associated medical condition and mental retardation.
<i>F84.0</i>	<i>Childhood autism</i>
	A type of pervasive developmental disorder that is defined by (a) the presence of abnormal or impaired development that is manifest before the age of 3 years and (b) the characteristic type of abnormal functioning in all the three areas of psychopathology: reciprocal social interaction, communication, and restricted, stereotyped,

(continued)

	repetitive behavior. In addition to these specific diagnostic features, a range of other nonspecific problems are common, such as phobias, sleeping and eating disturbances, temper tantrums, and (self-directed) aggression.	
	Autistic disorder	
	Infantile	
	Autism	
	Psychosis	
	Kanner's syndrome	
	<i>Excludes:</i>	Autistic psychopathy (<i>F84.5</i>)
<i>F84.1</i>	<i>Atypical autism</i>	
	A type of pervasive developmental disorder that differs from childhood autism either in age of onset or in failing to fulfil all three sets of diagnostic criteria. This subcategory should be used when there is abnormal and impaired development that is present only after age 3 years, and a lack of sufficient demonstrable abnormalities in one or two of the three areas of psychopathology required for the diagnosis of autism (namely, reciprocal social interactions, communication, and restricted, stereotyped, repetitive behavior) in spite of characteristic abnormalities in the other area(s). Atypical autism arises most often in profoundly retarded individuals and in individuals with a severe specific developmental disorder of receptive language.	
	Atypical childhood psychosis	
	Mental retardation with autistic features	
	Use additional code (<i>F70-F79</i>), if desired, to identify mental retardation.	
<i>F84.2</i>	<i>Rett's syndrome</i>	
	A condition, so far, found only in girls, in which apparently normal early development is followed by partial or complete loss of speech and of skills in locomotion and use of hands, together with deceleration in head growth, usually with an onset between seven and 24 months of age. Loss of purposive hand movements, hand-wringing stereotypies, and hyperventilation are characteristic. Social and play development are arrested, but social interest tends to be maintained. Trunk ataxia and apraxia start to develop by age 4 years and choreoathetoid movements frequently follow. Severe mental retardation almost invariably results.	
<i>F84.3</i>	<i>Other childhood disintegrative disorder</i>	
	A type of pervasive developmental disorder that is defined by a period of entirely normal development before the onset of the disorder, followed by a definite loss of previously acquired skills in several areas of development over the course of a few months.	

(continued)

	Typically, this is accompanied by a general loss of interest in the environment, by stereotyped, repetitive motor mannerisms, and by autistic-like abnormalities in social interaction and communication. In some cases the disorder can be shown to be due to some associated encephalopathy, but the diagnosis should be made on the behavioral features.
	Dementia infantilis
	Disintegrative psychosis
	Heller's syndrome
	Symbiotic psychosis
	Use additional code, if desired, to identify any associated neurological condition.
	<i>Excludes:</i> Rett's syndrome (F84.2)
F84.4	<i>Overactive disorder associated with mental retardation and stereotyped movements</i>
	An ill-defined disorder of uncertain nosological validity. The category is designed to include a group of children with severe mental retardation (IQ below 35) who show major problems in hyperactivity and in attention, as well as stereotyped behaviors. They tend not to benefit from stimulant drugs (unlike those with an IQ in the normal range) and may exhibit a severe dysphoric reaction (sometimes with psychomotor retardation) when given stimulants. In adolescence, the overactivity tends to be replaced by underactivity (a pattern that is not usual in hyperkinetic children with normal intelligence). This syndrome is also often associated with a variety of developmental delays, either specific or global. The extent to which the behavioral pattern is a function of low IQ or of organic brain damage is not known.
F84.5	<i>Asperger's syndrome</i>
	A disorder of uncertain nosological validity characterized by the same type of qualitative abnormalities of reciprocal social interaction that typify autism, together with a restricted, stereotyped, repetitive repertoire of interests and activities. It differs from autism primarily in the fact that there is no general delay or retardation in language or in cognitive development. This disorder is often associated with marked clumsiness. There is a strong tendency for the abnormalities to persist into adolescence and adult life. Psychotic episodes occasionally occur in early adult life.
	Autistic psychopathy
	Schizoid disorder of childhood
F84.8	<i>Other pervasive developmental disorders</i>
F84.9	Pervasive developmental disorder, unspecified

References and Reading

- Buitelaar, J. K., Van der Gaag, R., Klin, A., & Volkmar, F. (1999). Exploring the boundaries of pervasive developmental disorder not otherwise specified: Analyses of data from the DSM-IV autistic field trial. *Journal of Autism and Developmental Disorders*, 29(1), 33–43.
- Frances, A., Davis, W. W., Kline, M., Pincus, H., First, M., & Widiger, T. (1991). The DSM-IV field trials: Moving towards an empirically derived classification. *European Psychiatry*, 6(6), 307–314.
- Klin, A., Lang, J., Cicchetti, D. V., & Volkmar, F. R. (2000). Brief report: Interrater reliability of clinical diagnosis and DSM-IV criteria for autistic disorder: Results of the DSM-IV autism field trial. *Journal of Autism and Developmental Disorders*, 30(2), 163–167.
- Mayes, L., Volkmar, F. R., Hooks, M., & Cicchetti, D. V. (1993). Differentiating pervasive developmental disorder not otherwise specified from autism and language disorders. *Journal of Autism and Developmental Disorders*, 23(1), 79–90.
- Miller, J. N., & Ozonoff, S. (1997). Did asperger's cases have asperger disorder? A research note. *Journal of Child Psychology and Psychiatry*, 38(2), 247–251.
- Smith, I. C., Reichow, B., & Volkmar, F. R. (2015). The effects of DSM-5 criteria on number of individuals diagnosed with autism spectrum disorder: A systematic review. *Journal of Autism and Developmental Disorders*, 45(8), 2541–2552.
- Szatmari, P. (1991). Asperger's syndrome: Diagnosis, treatment, and outcome. *The Psychiatric Clinics of North America*, 14(1), 81–93.
- Szatmari, P. (1992a). A review of the DSM-III-R criteria for autistic disorder. *Journal of Autism and Developmental Disorders*, 22(4), 507–523.
- Szatmari, P. (1992b). The validity of autistic spectrum disorders: A literature review. *Journal of Autism and Developmental Disorders*, 22(4), 583–600.
- Tsai, L. (1992). Is Rett syndrome a subtype of pervasive developmental disorder? *Journal of Autism and Developmental Disorders*, 22, 551–561.
- Volkmar, F. R. (1991). DSM-IV in progress. Autism and the pervasive developmental disorders. *Hospital & Community Psychiatry*, 42(1), 33–35.
- Volkmar, F. R. (1992). Childhood disintegrative disorder: Issues for DSM-IV. *Journal of Autism and Developmental Disorders*, 22(4), 625–642.
- Volkmar, F. R., Cicchetti, D. V., Bregman, J., & Cohen, D. J. (1992a). Three diagnostic systems for autism: DSM-III, DSM-III-R, and ICD-10. *Journal of Autism and Developmental Disorders*, 22(4), 483–492.
- Volkmar, F. R., Cicchetti, D. V., Cohen, D. J., & Bregman, J. (1992b). Brief report: Developmental aspects of DSM-III-R criteria for autism. *Journal of Autism and Developmental Disorders*, 22(4), 657–662.
- Volkmar, F. R., Klin, A., Siegel, B., Szatmari, P., Lord, C., Campbell, M., et al. (1994). Field trial for autistic disorder in DSM-IV. *The American Journal of Psychiatry*, 151(9), 1361–1367.

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Major Appointments (Institution, Location, Dates)

National Institute of Child Health and Human Development Washington, D.C., Director 1986–2009

Major Honors and Awards

His numerous awards included the USPHS Commendation Medal, a Meritorious Service Medal of Special Recognition, and the Surgeon General's Exemplary Service Medal. He also received the Arnold J. Capute Award from the American Academy of Pediatrics and the Dr. Nathan Davis Award for Outstanding Government Service from the American Medical Association. At the time of his retirement, he had achieved the rank of Assistant Surgeon General (Rear Admiral).

Landmark Clinical, Scientific, and Professional Contributions

During his directorship of NICHD, he undertook a number of initiatives including in the areas of amniocentesis, early screening for developmental disability, and sudden infant death. His work in

helping prevent the maternal-to-child transmission of HIV in the United States is particularly important as well as his work on syndromes like Fragile X and Rett. In the area of autism research, Dr. Alexander had a critically important role in the establishment of the Collaborative Programs of Excellence in Autism. He worked with program officer Dr. Marie Bristol Power in developing this highly innovative project to support research at ten institutions around the country. This work was central in facilitating the growth of multidisciplinary work in autism, and Dr. Alexander's support of work on Autism came at a time when federal supports was limited. His championship of the highest quality work in autism came at a time when autism appeared to be a disorder "without an agency." His visionary approach and leadership catapulted the science of autism forward, and many of the advances emerging today stem from the unique experiences afforded by the CPEA to young scholars, whose collaborative work with senior investigators and other junior investigators laid the foundation for innovations and breakthroughs that continue to benefit many individuals with autism spectrum disorder. The funding of the network ended in 2007 when the NIH consolidated its funding for autism research into new programs.

Short Biography

A tireless advocate for children with disabilities and retired leader of the National Institute of Child Health and Human Development, Dr. Alexander passed away on February 20, 2020. A native of Annapolis, MD, he graduated from Pennsylvania State University and received his medical education from Johns Hopkins University School of Medicine where he also completed his internship and residency in pediatrics. He joined NICHD in 1968 and also was a fellow in pediatrics focusing on developmental disabilities at the John F. Kennedy Institute for the Habilitation of the Mentally and Physically Handicapped Child (today known as the Kennedy Krieger Institute).

He served as the Director of NICHD from 1986 until his retirement. He served as medical officer in the Office of the Assistant Secretary for Health

in what is now the Department of Health and Human Services (HHS). During that time, he was also a physician on the staff of the National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research, whose recommendations form the basis of current HHS regulations that protect human subjects in research. He served as NICHD Deputy Director starting in 1978 and was acting director for a time before becoming NICHD Director in 1986. For more than a decade, he also served as the United States' observer on the Steering Committee on Bioethics for the Council of Europe.

Dr. Alexander became an assistant to the scientific director in 1971 and was also an Assistant Secretary for Health in what is now the Department of Health and Human Services. He was active in the area of protection of human subjects and became NICHD Deputy Director starting in 1978; he became the Director in 1986 and served in this role until 2009.



Photo credit: NICHD

Due Process

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Definition

The due process hearing and other procedural safeguards provide a system of checks and balances for schools and parents. The due process principle essentially aims to ensure that schools and parents are held accountable to each other for carrying out the student's rights as outlined in the Individuals with Disabilities Education Act (IDEA). A due process hearing may be requested by the parents or the school district if they are in disagreement about any of the following: identification, evaluation, placement, the IEP document, or the provision of a free and appropriate education (FAPE) to a child.

A due process complaint must be filed in written form and must contain the following specific information: the name of the child, the address of the residence of the child, the name of the school the child is attending, a description of the nature of the child's problem related to the proposed action, a statement of how requirements of part B of IDEA or its implementing regulations have been violated, the facts upon which the statement is based, and a proposed resolution of the problem. A copy of the due process complaint must be provided to the other party, and a copy must be forwarded to the state educational agency.

A due process hearing is like a mini-trial before an impartial, third-party, hearing officer and is similar to a regular courtroom trial. The hearing officer is responsible for listening to both sides of the dispute, examining all related issues, and settling the dispute. Parents have the right to be accompanied or advised by counsel, present evidence, cross-examine witnesses, and see the evidence presented by the school district 5 days

prior to the hearing. Parents also have the right to have the child present at the due process hearing, but it is not required. Only a small number of disagreements between parents and schools result in parents filing a due process petition. Even fewer cases actually proceed to a hearing. If either the parent or the school district is dissatisfied with the outcome of the hearing, they have the right to appeal the decision to state or federal courts.

The school district is responsible for scheduling a resolution or mediation meeting with the parents within 15 days of receiving a due process complaint. Under the IDEA provisions, the school district and parents have 30 days from the date that the due process complaint was filed to reach an agreement through the process of resolution or mediation. If the parties are not able to reach an agreement by the end of these 30 days, then a due process hearing must be held and final decision issued within the next 45 calendar days. In some cases, the parents or the school district may choose to expedite the due process hearing. If this is the case, then a due process hearing must be conducted within 20 school days after receiving the due process complaint and the decision must be issued within 10 school days after the hearing.

Historical Background

The due process provision has been a part of IDEA since its inception. Under the procedural safeguards provision in IDEA, parents and school districts have the right to a due process hearing and the rights that go along with those hearings. Procedural safeguards are the protections in IDEA that ensure that students with disabilities and their parents or guardians are meaningfully involved in all decisions related to the student's special education and that they have the right to seek a review of any decisions they think are appropriate. The procedural safeguards are grounded in the 5th and the 14th Amendments of the US Constitution, which guarantee that no person shall be deprived of

life, liberty, or property without due process of the law.

Current Knowledge

The recent 2004 amendments to IDEA emphasized the importance for parents and school districts to resolve disputes collaboratively and quickly. IDEA encourages parents and schools to utilize alternative methods for resolving their dispute prior to proceeding to a due process hearing. First, the IDEA recommends resolution and then mediation as methods for resolving agreements prior to proceeding to a due process hearing.

Under the current 2004 amendments to IDEA, parents and school districts have up to 2 years to file a due process complaint. Exceptions to the 2-year timeline may apply if the parent was prevented from filing the due process complaint due to misrepresentations by the school district or if the school district withheld information that was required to be provided to the parent under the statutes and regulations of IDEA. States do have the right to change the 2-year timeline as they desire. If a state decides to shorten or lengthen the timeline, this information must be included and explained in the procedural safeguards notice that the school district is required to provide to parents so that parents are fully informed of the timeline limitations within their individual state.

Key changes implemented under the amendments to the IDEA 2004 statutes and regulations include the following:

1. *Notification:* Parents and/or school districts filing a due process complaint must provide notice to the other party as well as the state educational agency. Further, school districts are required to provide parents with notice of their procedural safeguards and information about free or low-cost legal services in the area.
2. *Specific timelines for responding to a due process complaint:* If the school district has not already provided parents with written notice regarding their actions relating to the issue addressed in the due process complaint, the

school district must now respond within 10 days of receiving the due process complaint. The response must include an explanation of why the school district has proposed or refused to take the action addressed in the complaint. The response must include a description of (a) options that the IEP team considered and why those reasons were rejected; (b) the evaluation procedure, assessments administered, and reports reviewed by the IEP team; and (c) any additional factors related to the case.

3. *Specific timelines for conducting a resolution meeting:* The school district must convene a meeting with the parent within 15 days of receiving the due process complaint. The purpose of the meeting is to resolve the dispute if possible. This meeting may be waived if the parents and school district agree in writing to waive the meeting or if the parent and school district agree to use the mediation process as opposed to a resolution meeting. If the school district fails to respond to the due process complaint or fails to participate in a resolution meeting within 15 days of receipt of the complaint, the parent may contact the hearing officer to begin the due process hearing timeline.
4. *Request by the school district to dismiss the case:* If a school district has made reasonable, documented efforts to schedule a resolution meeting with the parent and the parent has been nonresponsive, the school district may request that the hearing officer dismiss the case after 30 days.
5. *Requirements for resolution meeting agreements:* In the case that the parents and school district are able to resolve the dispute during a resolution meeting, both parties are required to sign a legally binding document stating the agreement. This document may be voided by either party within 3 business days but otherwise is enforceable in any state or district court.
6. *Timelines for conducting the hearing and issuing a final decision:* After the 30-day resolution period, the due process hearing and final decision must be conducted within 45 days. By the end of the 45-day period, not only must the hearing be conducted but also a final decision issued by the hearing officer, and a copy of the

decision must be mailed to the parents and the school district. The IDEA amendments also allow states the opportunity to review the decision of the hearing officer. The state has 30 days to review the decision and provide a written copy of their review decision to the parents and school district.

7. *Hearing officer requirements and decisions:* The hearing officer cannot be an employee of the state or school district where the child attends school. The hearing officer must not only have knowledge of the provisions of the IDEA as well as federal and state regulations related to the IDEA statutes but also the ability to conduct hearings and document decisions according to standard legal practice. The decision of the hearing officer in the due process hearing must be based on substantive grounds. A hearing officer may determine that a child did not receive a free and appropriate education (FAPE) if the procedural inadequacies of the school district obstructed the parent's opportunity to participate in the identification, evaluation, and IEP development process; interfered with the child's right to a FAPE; or caused a deprivation of educational benefit.

See Also

- [Procedural Safeguards](#)

References and Reading

- Due process complaints, in detail*, (2010). National Dissemination Center for Children with Disabilities. Retrieved from <http://nichcy.org/schoolage/disputes/dueprocess/regs>
- Due process hearings*, Wrightslaw. Retrieved 10 June 2011, from <http://www.wrightslaw.com/info/dp.index.htm>
- Questions and answers on procedural safeguards and due process procedures for parents and children with disabilities. (2009). U.S. Department of Education. Retrieved 10 June from <http://idea.ed.gov/explore/view/p/root,dynamic,QaCorner,6>
- Topic: Procedural safeguards: Due process hearings*. US Department of Education. Retrieved 10 June 2011, from <http://idea.ed.gov/explore/view/p/root,dynamic,TopicalBrief,16>

Yell, M. L. (2006). *The law and special education* (2nd ed.). Upper Saddle River: Merrill/Prentice Hall.

Zirkel, P., & Scala, G. (2010). Due process hearing systems under the IDEA: A State-by-State survey. *Journal of Disability Policy Studies*, 21(1), 3–8. Retrieved 10 June 2011, from <http://www.directionservice.org/cadre/pdf/Due%20Process%20Hearing%20Systems.pdf>

Dulane

► Duloxetine: Definition

Duloxetine: Definition

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Synonyms

[Ariclaim](#); [Cymbalta](#); [Dulane](#); [Duzela](#); [Xeristar](#); [Yentreve](#)

Definition

Duloxetine is a selective serotonin and norepinephrine reuptake inhibitor. Serotonin is a mood-regulating neurotransmitter, while norepinephrine is mainly involved in attentiveness. By inhibiting the reuptake of these two neurotransmitters, duloxetine increases their levels and action durations in the neuronal synapse. While both serotonin and norepinephrine have been implicated in autism to various degrees, there is no substantial evidence of duloxetine's effectiveness in treating ASD. Duloxetine is approved for use as an antidepressant and anxiolytic. It is also used in the treatment of fibromyalgia and diabetes-associated

nerve damage. Side-effect profiles are highly varied including nausea, vomiting, heartburn, diarrhea, and many others but with an unknown incidence.

See Also

► Antidepressants

References and Reading

- Goldstein, D. J., Lu, Y., Detke, M. J., Lee, T. C., & Iyengar, S. (2005). Duloxetine vs. placebo in patients with painful diabetic neuropathy. *Pain*, 116(1), 109–118.
- Nemeroff, C. B., Schatzberg, A. F., Goldstein, D. J., Detke, M. J., Mallinckrodt, C., Lu, Y., & Tran, P. V. (2001). Duloxetine for the treatment of major depressive disorder. *Psychopharmacology Bulletin*, 36(4), 106–132.
- Niederhofer, H. (2011). Efficacy of duloxetine and agomelatine does not exceed that of other antidepressants in patients with autistic disorder: Preliminary results in 3 patients. *The Primary Care Companion to CNS Disorders*, 13(1), e1.

Duplic (Verbal Behavior)

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Skinner (1957) proposed a system of classifying verbal behavior, by function and form, resulting in discrete units of analysis termed *verbal operants*. The taxonomy is based on the forms of the antecedent controlling stimuli and response products and the specific consequences provided for each operant. Skinner's original categorization included the following operants: *echoic*, *mand*, *tact*, and *intraverbal*. However, these categories of behavior do not encompass all forms and functions of communication and language. Two additional categories have since been proposed: *codic* and *duplic* (Michael 1982).

The form of a verbal operant is said to have *point-to-point correspondence* when the individual components (e.g., letters, syllables) of the antecedent stimulus and response product forms match. And, when the sense mode of the stimulus and response is the same (e.g., auditory stimulus and vocal response), they exhibit *formal similarity*. The function of each verbal operant is determined by controlling consequences or motivating operations (*mand* only). Controlling consequences are changes in stimulus conditions that follow the occurrence of verbal operants (e.g., praise), whereas motivating operations are environmental variables that alter the effectiveness of some stimulus as a reinforcer and alter the frequency of behavior that has been reinforced by that stimulus (e.g., hunger).

A *duplic* is a verbal operant in which the antecedent stimulus and response product forms exhibit point-to-point correspondence and formal similarity, and that is reinforced by generalized conditioned reinforcement from a listener (usually a parent/guardian or teacher). Common examples of *duplic*s include saying words that one hears spoken (echoic), writing words that one sees written (copying a text), or signing a word that one sees signed by another person (mimetic). *Duplic*s are learned through both direct and incidental teaching strategies and are reinforced by the learner's verbal community with praise or some form of educational reinforcement.

References and Reading

- Michael, J. (1982). Skinner's elementary verbal relations: Some new categories. *The Analysis of Verbal Behavior*, 1(1), 1–3. <https://doi.org/10.1007/BF03392791>.
- Skinner, B. F. (1957). *Verbal behavior*. New York: Appleton-Century-Crofts.

Duplin

- [CHD8](#)

Durability of Treatment Effects

- [Maintenance of Treatment Effects](#)

Duzela

- [Duloxetine: Definition](#)

Dysarthria

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Definition

Dysarthria is a motor speech disorder caused by generalized weakness to the oral musculature that occurs as a result of damage to the central and/or peripheral nervous system (Duffy 1995; Freed 2000; Vinson 2007; Zemlin 1998). This damage may occur as a result of stroke, head injury, cerebral palsy, muscular dystrophy, or other brain injury (American Speech-Language-Hearing Association n.d.; Freed 2000). As a consequence of oral musculature weakness, the speech of individuals with dysarthria is slow and labored, and their articulation is imprecise (Freed 2000; Zemlin 1998). Other areas of speech may also be affected including respiration, voicing, and prosody (Duffy 1995).

See Also

- [Articulation Disorders](#)
- [Speech](#)

References and Reading

- ASHA. (n.d.) Dysarthria. In *American-speech-language-hearing-association speech disorders*. Retrieved 5 Jan 2011 from http://www.asha.org/public/speech/development/language_speech.htm
- Duffy, J. R. (1995). *Motor speech disorders: Substrates, differential diagnosis, and management*. St. Louis: Mosby.
- Freed, D. (2000). *Motor speech disorders: Diagnosis and treatment*. San Diego: Singular.
- Vinson, B. (2007). *Language disorders across the lifespan* (2nd ed.). Clifton Park: Thomson Delmar Learning.
- Zemlin, W. R. (1998). *Speech and hearing science: Anatomy and physiology* (4th ed.). Boston: Allyn and Bacon.

Dyscalculia

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Synonyms

[Developmental dyscalculia](#); [Mathematics disability](#)

Definition

Dyscalculia is a learning disability characterized by an inability to acquire the mathematical skills that would be expected for a given chronological age, cognitive ability, and educational level. Mathematics skills are typically broken down into mathematics calculation (i.e., computation) and mathematics reasoning (i.e., problem solving). Given the cumulative nature of mathematics, initial difficulty in learning and becoming fluent in basic numerical concepts impedes the later development of more complex arithmetic concepts (Butterworth 2008). For example, proficiency with whole numbers and fractions (including decimals, percent, and negative

fractions) as well as measurement and geometry has been identified as being foundational for later academic success (National Mathematics Advisory Panel 2008).

Specific characteristics of dyscalculia vary among individuals but commonly include one or more of the following: poor automatic recall of basic facts, such as multiplication tables; poor knowledge of or poor fluency in procedures for multidigit calculation, such as addition with regrouping; visuospatial weaknesses that result in place errors in written calculations and in difficulty with geometry; language deficits in areas requiring higher order thinking, thus interfering with carrying out word problems; and insufficient attention, impairing short-term memory necessary for mental arithmetic (e.g., Rapin 2010). Furthermore, those with dyscalculia not only struggle with mathematics in school but even have difficulty with numbers in everyday tasks, such as telling time and remembering phone numbers (Butterworth 2008). However, some individuals with dyscalculia often perform without difficulty in other academic areas, although there are individuals who struggle with reading and spelling, as well as mathematics.

Possible causes of dyscalculia are numerous; these include but are not necessarily limited to poor instruction, low intelligence, anxiety about numbers, behavioral and/or attentional problems, poor working memory, language-based learning disabilities, impaired visuospatial abilities, and motor skill deficits (e.g., Butterworth 2008; Zager and Donaldson 2010). Apart from environmental causes (e.g., poor teaching or behavioral issues), developmental dyscalculia may be brain-based, for example, associated with a familial-genetic predisposition (Shalev and Gross-Tsur 2001). Developmental dyscalculia also frequently occurs in neurologic disorders such as attention-deficit/hyperactivity disorder, developmental language disorder, and fragile X syndrome (Shalev and Gross-Tsur). Dyscalculia is also often associated with either a nonverbal learning disability, that is, performance is poor only in mathematics (but also reading and

spelling), or a comorbid condition of reading/spelling and mathematics disabilities.

Particular to autism spectrum disorder (ASD), studies have found that on mathematics assessments, approximately 25% of those students with high-functioning ASD perform at least one standard deviation below their vocabulary scores, thereby presenting with a mathematics disability (e.g., Mayes and Calhoun 2008; Zager and Donaldson 2010). The fact that nearly one-quarter of this population has a developmental dyscalculia contradicts the commonly held belief that persons with ASD have high mathematical ability levels (Williams et al. 2008).

Interventions for students with dyscalculia must address specific areas of difficulty using explicit and systematic teaching of skills. Practice and cumulative review are especially important. Often the use of visuals and/or manipulatives is of benefit in teaching and practicing mathematics concepts.

References and Reading

- Butterworth, B. (2008). Developmental dyscalculia. In J. Reed & J. Warner-Rogers (Eds.), *Child neuropsychology: Concepts, theory, and practice* (pp. 357–376). Hoboken: Wiley-Blackwell.
- Mayes, S. D., & Calhoun, S. L. (2008). WISC-IV and WIAT-II profiles in children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 38(3), 428–439.
- National Mathematics Advisory Panel. (2008). *Foundations for success: The final report of the national mathematics advisory panel*. Washington, DC: U.S. Department of Education.
- Rapin, I. (2010). Disorders of motor and mental development. In L. P. Rowland & T. A. Pedley (Eds.), *Merritt's neurology* (12th ed., pp. 568–575). Philadelphia: Lippincott Williams & Wilkins.
- Shalev, R. S., & Gross-Tsur, V. (2001). Developmental dyscalculia. *Pediatric Neurology*, 24, 337–342.
- Williams, D. L., Goldstein, G., Kojkowski, N., & Minshew, N. J. (2008). Do individuals with high-functioning autism have the IQ profile associated with nonverbal learning disability? *Research in Autism Spectrum Disorders*, 2, 353–361.
- Zager, D., & Donaldson, J. B. (2010). Mathematics interventions for students with high-functioning autism/Asperger's Syndrome. *Teaching Exceptional Children*, 42, 40–46.

Dyschezia

- [Constipation](#)

Dysexecutive Syndrome

- [Frontal Lobe Syndrome](#)

Dysfluency

- [Fluency and Fluency Disorders](#)

Dysgenesis (Malformation)

- [Agenesis of Corpus Callosum](#)

Dysgraphia

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Synonyms

[Handwriting difficulty](#)

Definition

Dysgraphia is a specific learning disability in which writing letters by hand is impaired, thereby affecting the acquisition and use of written language. Spelling difficulties may also be present in dysgraphia. Thus, an individual with dysgraphia may have problems with handwriting or spelling or both.

Importantly, handwriting (i.e., letter production) is not a simple motor act. Letter production requires visuospatial integration and motor planning for controlling the size of strokes and alignment of letters on paper, motor control and memory for hand movements involved in writing letters, and working memory for holding letters while motor acts are being planned and carried out (Berninger 2004). Similarly, spelling is not simply a visual skill. It requires both orthographic (i.e., letter-sound associations) and phonological processes (i.e., short-term memory for spoken information) (Berninger 2004).

The defining characteristic of dysgraphia is poor or slow handwriting, though additional characteristics are possible; thus, clinical subtypes of dysgraphia have been suggested: language-based or dyslexic dysgraphia, motor-executive dysgraphia, and visuospatial dysgraphia (e.g., O'Hare 1999). In dyslexic dysgraphia, writing difficulties arise from impairments in phonological processing (e.g., phonological awareness), semantics (e.g., word knowledge), and/or syntax (e.g., grammar). Writing is characterized by both poor legibility and weak spelling, though copying is typically relatively intact. Differently, a motor-executive dysgraphia is associated with a motor-planning weakness, that is, difficulty remembering the sequence of kinesthetic movements necessary to form letters correctly. As in dyslexic dysgraphia, handwriting is illegible; however, spelling is not affected and copying is impaired. Visuospatial dysgraphia results from impairment in understanding space. Characteristics are similar to motor-executive dysgraphia; however, in addition to letter formation, weaknesses also occur in margin consistency and spacing of letters. Further, along with handwriting difficulty, visuospatial dysgraphia also includes difficulty with drawing and motor-free visuospatial tasks (e.g., visually perceive [recognize] letters in the correct direction). Regardless of the clinical subtype, because those with dysgraphia focus more energy on handwriting, in order to shorten the handwriting process, they use as few words as possible in written communication.

The high prevalence of writing disabilities in individuals with autism spectrum disorders (ASD) has been well documented (e.g., Miyahara et al. 1997). Impairments in any or several domains that contribute to handwriting account for the frequency of dysgraphia in the population. These include but are not limited to problems with fine and gross motor functions that make precise manipulations of writing difficult; with proprioception, thus making it difficult to accurately develop fluid, automatic handwriting; and with vision that affects how letters are perceived and reproduced (Fuentes et al. 2009). Research has found that some children with ASD have problems forming letters, but not sizing, spacing, or aligning them (Fuentes et al. 2009).

It is important to address dysgraphia with appropriate treatment. Mechanics of writing, especially handwriting fluency, are predictive of both the fluency (rate) and quality of written compositions. Difficulties with the mechanics of writing (i.e., constructing letters and spelling) interfere with the same attentional resources necessary to generate meaningful composition (Berninger et al. 1997).

See Also

► [Agraphia](#)

References and Reading

- Berninger, V. W. (2004). Understanding the “graphia” in developmental dysgraphia. In D. Dewey & D. E. Tupper (Eds.), *Developmental motor disorders: A neuropsychological perspective* (pp. 328–350). New York: Guilford Press.
- Berninger, V., Vaughan, K., Abbott, R., Abbott, S., Rogan, L., Brooks, A., & Graham, S. (1997). Treatment of handwriting fluency problems in beginning writing: Transfer from handwriting to composition. *Journal of Educational Psychology*, 89, 652–666.
- Fuentes, C. T., Mostofsky, S. H., & Bastian, A. J. (2009). Children with autism show specific handwriting impairments. *Neurology*, 73(19), 1532–1537. <https://doi.org/10.1212/WNL.0b013e3181c0d48c>.
- Miyahara, M., Tsujii, M., Hori, M., Nakanishi, K., Kageyama, H., & Sugiyama, T. (1997). Brief report:

Motor incoordination in children with asperger syndrome and learning disabilities. *Journal of Autism and Developmental Disorders*, 27, 595–603.

O'Hare, A. (1999). Dysgraphia and dyscalculia. In W. Kingsley, H. Hart, & G. Willems (Eds.), *A neurodevelopmental approach to specific learning disorders* (pp. 96–118). London: Mac Keith Press.

Dyslexia

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Synonyms

[Developmental reading disorder](#); [Specific reading disability](#)

Short Description or Definition

The National Institute of Neurological Disorders and Stroke defines dyslexia as “a brain-based type of learning disability that specifically impairs a person's ability to read. These individuals typically read at levels significantly lower than expected despite having normal intelligence. Although the disorder varies from person to person, common characteristics among people with dyslexia are difficulty with spelling, phonological processing (the manipulation of sounds), and/or rapid visual-verbal responding. In adults, dyslexia usually occurs after a brain injury or in the context of dementia. It can also be inherited in some families and so on, and recent studies have identified a number of genes that may predispose an individual to developing dyslexia.”

Categorization

Learning disability
Neurological disorder

Epidemiology

Dyslexia is known as a neurological disorder in that there appear to be differences in brain functioning associated with language, learning, and reading in this population. Dyslexia is typically not acquired but related to congenital factors and frequently occurs in related family members. Current genetic research suggests that a number of genes exist that may predispose a child to developing dyslexia. Dyslexia is usually detected and diagnosed in early elementary years, when affected children demonstrate difficulties with classroom demands related to alphabet learning, word recognition, reading comprehension, spelling, and writing. It is thought to affect between 5% and 10% of the population although there have been no studies to indicate an accurate percentage. In adults, symptoms associated with dyslexia can occur following a brain injury/stroke, or as a result of developing dementia.

Natural History, Prognostic Factors, and Outcomes

Although typically not diagnosed until middle elementary grades, dyslexia is frequently associated with delayed language development during the preschool period. Although the language delay often appears to resolve in primary grades, learning to read and write is often slow and laborious. Children with dyslexia demonstrate difficulties in phonological awareness during primary school years; they have trouble identifying rhymes, recognizing words that start or end with the same letter, and breaking words down into component sounds (hit = /h/ + /I/ + /t/). Although in multisensory interventions children “experience” letters and words in various tactile modalities (such as feeling sandpaper letters or making letters in finger paint), the approach with the best evidence base is prolonged, intensified exposure to phonological awareness training, letter-sound correspondence, sound analysis (what sounds are in the word *cat*?), and sound-word synthesis (What word do these sounds make s-u-n?). With intensive instruction, many children with dyslexia

learn to read and write, although both may remain slow and labored. For students with severe dyslexia, educational practice requires alternative access to curricular texts by means of material to be read to the student, or recorded for listening. Computer applications that translate text to speech are also used. With appropriate compensations, individuals with dyslexia can achieve high levels of independence and achievement. However, many suffer from poor self-esteem if compensations and support are not provided.

Clinical Expression and Pathophysiology

The symptoms of dyslexia may vary, depending on the age of the individual and the severity of their difficulties. In young children, delayed language, trouble identifying and segmenting sounds, rhyming, and letter-sound matching are often seen. Children with dyslexia are commonly thought to simply “reverse” letters in their reading and writing; however, many typical children reverse letters when learning to read and write. In fact, a person with dyslexia may show no pattern of letter reversal at all. Although children and adults with dyslexia tend to have average to above-average cognitive skills, they may demonstrate associated difficulties with reading comprehension, writing, as well as math, as these areas also require connection and understanding of letters/graphics.

Evaluation and Differential Diagnosis

Thorough measures of neuropsychological assessment exist in order to detect and treat dyslexia in children from an early age. Assessment for a child showing delayed or impaired reading acquisition may include cognitive and academic achievement testing, as well as an evaluation of the critical underlying language skills closely linked to dyslexia (phonological awareness, letter naming, fluency). Developmental history, family history of dyslexia, and early development of speech and language should also be investigated. The presence of vision, auditory,

and attention impairments are unrelated to the reading and comprehension signs of dyslexia and should be ruled out and treated as separate disorders.

There are a number of underlying skills that comprise a person’s ability to read, including phonological awareness, alphabet decoding, automatic word recognition, and short-term memory. In young children learning to read, these skills develop along a continuum and work together for successful reading acquisition. For a child suspected of dyslexia, these skills should be carefully assessed in order to detect what areas are underdeveloped and/or causing the most difficulty. As typical children acquire these skills, their reading becomes more fluent and with a quicker pace. Pacing and fluency does not tend to come as naturally for children with dyslexia and remains a chronic difficulty for many adults with dyslexia.

Symptoms associated with dyslexia can also occur in children with other language and learning disabilities; therefore, a comprehensive assessment is necessary to evaluate areas of reading, writing, comprehension, and verbal expression. Many children with reading problems also have spoken language deficits.

Treatment

Depending on the varying degrees of difficulty and the areas most impaired in an individual with dyslexia (i.e., letter/word recognition, reading fluency, comprehension), treatment methods and intensity of approach will vary. For children, strategies and supports in developing phonological skills (e.g., letter-sound recognition, sound separation, segmentation, rhyming) will ultimately assist with reading and writing deficits. For school-aged children who have acquired reading but continue to struggle, teaching strategies should address pacing, processing, and comprehension. Regardless of student age, a team of professionals should consult and collaborate to develop an educational plan with specific teaching methods and accommodations to meet the needs of the child.

School professionals and clinicians, particularly reading specialists, special educators, and speech-language pathologists, are trained to develop interventions and treat dyslexia. Multi-sensory approaches that target auditory, visual, and tactile areas associated with reading and comprehension offer thorough methods to address difficulties caused by dyslexia. A thorough assessment and teacher report can also offer information related to individual learning styles. For example, individuals who benefit from visual versus purely auditory teaching methods.

Assessment can also reveal the specific reading skills that require remediation for a child or adult, including:

- Phonemic awareness: recognition of the letter sounds in words
- Phonological awareness: understanding that words can be broken down into phonemes and those phonemes can be manipulated, such as segmenting sounds in words, blending sounds, and rhyming (sounds are distinct from meaning)
- Reading fluency: spelling, speed, accuracy, and ease
- Oral reading: proper expression and fluency
- Reading comprehension: includes vocabulary knowledge and understanding the meaning of passages

For adults, individualized tutoring can provide both structured and tailored teaching to address problem areas and monitor progress. Counseling sessions with a trained clinician familiar with dyslexia can provide a supportive arena to develop and discuss strategies for home and career settings where reading is involved. Counseling is also beneficial for individuals with dyslexia who demonstrate fear or anxiety surrounding their reading difficulties.

References and Reading

American Speech, Language and Hearing Association, ASHA. (2010). Language based learning disabilities. Retrieved from <http://www.asha.org/public/speech/disorders/LBLD.htm>

- Clark, D. B., & Uhry, J. K. (1995). *Dyslexia: Theory & practice of remedial instruction*. Baltimore: York Press.
- Healey, J. M., & Aram, D. M. (1986). Hyperlexia and dyslexia: A family study. *Annals of Dyslexia*, 36(1), 237–252. <https://doi.org/10.1007/BF02648032>.
- International Dyslexia Association. (2007). Dyslexia basics. Retrieved from http://www.ldonline.org/article/Dyslexia_Basics
- McCardle, P., & Chhabra, V. (2004). *The voice of evidence in reading research*. Baltimore: Paul H. Brookes.
- Moats, L., & Dakin, K. (2008). *Basic facts about dyslexia and other reading problems*. Baltimore: The International Dyslexia Association.
- National Institute of Neurological Disorders and Stroke. (2010). Dyslexia information page.
- Rumsey, J. M., & Hamburger, S. (1990). Neuropsychological divergence of high-level autism and severe dyslexia. *Journal of Autism and Developmental Disorders*, 20(2), 155–168. <https://doi.org/10.1007/BF02284715>.
- Schaywitz, S. (2003). Early clues to dyslexia. In S. Schaywitz (Ed.), *Overcoming dyslexia* (pp. 93–101). New York: Random House.
- Shapiro, J., & Rich, R. (1999). *Facing learning disabilities in the adult years: Understanding dyslexia, ADHD, assessment, intervention and research*. New York: Oxford University Press.

Dysphasia

► [Global Aphasia](#)

Dysphoria

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Definition

A negative emotional state characterized by dissatisfaction, restlessness, anxiousness, irritability, and depression. Dysphoria is a symptom of various psychiatric disorders such as major depressive disorder, dysthymia, generalized anxiety disorder, body dysmorphic disorder, bipolar disorder, and premenstrual dysphoric disorder. Dysphoria is usually experienced during states of depression, but people with bipolar disorder may also experience dysphoria during manic or

hypomanic episodes. Dysphoria is the opposite of euphoria.

See Also

- [Depressive Disorder](#)
- [Mood Disorders](#)

References and Reading

- Blazer, D., & Williams, C. (1980). Epidemiology of dysphoria and depression in an elderly population. *The American Journal of Psychiatry*, 137, 439–444.
- Roberts, J., Gilboa, E., & Gotlib, I. (1998). Ruminative response style and vulnerability to episodes of dysphoria: Gender, neuroticism, and episode duration. *Cognitive Therapy and Research*, 22(4), 401–423.

Dyspraxia

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Synonyms

[Apraxia](#)

Definition

The term “dyspraxia” is often used to describe less severe forms of apraxia. Apraxia is the disruption in the ability to plan and execute volitional (purposeful) movements despite intact muscle strength and coordination. Involuntary movements remain intact. Importantly, apraxia is not associated with weakness, slowness, or incoordination. Apraxia is a motor disorder resulting from neurological damage. There are three types of apraxia: limb, oral, and verbal. In a limb apraxia, the volitional movements of the extremities are affected. In an oral apraxia, nonspeech

movements of the oral mechanism are affected. Verbal apraxia (or Apraxia of Speech) is a disorder in which an individual has difficulty positioning and sequencing muscles for the volitional production of speech.

See Also

- [Apraxia](#)
- [Developmental Coordination Disorder](#)
- [Developmental Dyspraxia](#)
- [Oral-Motor Apraxia](#)
- [Verbal Apraxia](#)
- [Verbal Dyspraxia](#)

References and Reading

- Darley, R. (1969, November). *Aphasia: Input and output disturbances in speech and language processing*. Presentation at the American Speech and Hearing Association, Chicago.
- Darley, F., Aronson, A., & Brown, J. (1975). *Motor speech disorders*. Philadelphia: W.B. Saunders.
- Kent, R., & Rosenbek, J. (1983). Acoustic patterns of apraxia of speech. *Journal of Speech and Hearing Research*, 26, 231–249.
- Liepmann, H. (1900). Das Krankheitsbild der Apraxie (motorischen Asymbolie) auf Grund eines falles von einseitiger Apraxie. *Monatsschrift für Psychiatrie und Neurologie*, 8, 15–40.
- Shriberg, L. D., Paul, R., Black, L. M., & van Santen, J. P. (2011). The hypothesis of apraxia of speech in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41, 405–426.

Dystaxia

- [Ataxia](#)

Dysthymia

- [Depressive Disorder](#)

Dystonia

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Definition

A movement disorder characterized by involuntary movements and prolonged muscle contraction, resulting in twisting body motions, repetitive movements, or abnormal posture (Fahn et al. 1998; Geyer and Bressman 2006). These movements may involve the entire body or only an isolated area. Dystonia can be hereditary or occur sporadically without any genetic pattern; it can occur as a result of birth related or other trauma or may be associated with medications, particularly neuroleptics, or diseases. The gene responsible for at least one form of dystonia has recently been identified. Some types of dystonia respond to dopamine or can be controlled with sedative-type medications or surgery. Dystonias may be classified by the age of onset, by the part (s) of the body affected, or by the cause (primary or secondary dystonia).

Primary dystonia presents with signs only related to the dystonia and is thought to be caused by pathology in parts of the brain that are concerned with movement (basal ganglia) and the GABA (gamma-aminobutyric acid) producing neurons. This dystonia occurs without additional neurologic, laboratory, or imaging abnormalities. The precise cause of primary dystonia is unknown. In many cases, it may involve some genetic predisposition towards the disorder combined with environmental conditions.

Secondary dystonia refers to dystonia associated with a known cause or additional neurologic findings. It usually arises as the result of a specific underlying condition such as insufficient oxygen at birth and exposure to medications that block dopamine receptors (Friedman and Standaert 2001).

Treatment of dystonias is based upon the symptomatology. There are no curative therapies, although in the case of medication-induced dystonia, discontinuation of the medication may result in a resolution of some dystonic symptoms. Management of dystonia includes medications such as levodopa, anticholinergics, tetrabenazine, clonazepam, and baclofen. Botulinum toxin injections and deep brain stimulation are also recognized treatments.

See Also

- [Anticholinergic](#)
- [Dopamine](#)

References and Reading

- Fahn, S., Bressman, S. B., & Marsden, C. D. (1998). Classification of dystonia. *Advanced Neurology*, 78, 1–10.
- Friedman, J., & Standaert, D. G. (2001). Dystonia and its disorders. *Neurologic Clinics*, 19(3), 681–705. vii.
- Geyer, H. L., & Bressman, S. B. (2006). The diagnosis of dystonia. *Lancet Neurology*, 5(9), 780–790.

Dytan™

- [Diphenhydramine](#)

Early Childhood Tutor

► [Itinerant Teacher](#)

Early Diagnosis

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Definition

Although autism spectrum disorders can be diagnosed at any age, the classification systems require onset prior to 3 years of age or in early childhood. The term “early diagnosis” refers to diagnosis in the second and third year of life.

Historical Background

Notwithstanding progress in understanding of the genetic and neurodevelopmental processes that lead to autism spectrum disorders (ASD), clinical diagnosis is reliant on the developmental and behavioral presentation. Until the 1990s, it was

rare for children to receive a diagnosis of autism until the age of 3 or 4 years, and sometimes considerably later (Howlin and Asgharian 1999). This was despite the fact that the DSM and ICD diagnostic systems (American Psychiatric Association [APA] 2000; World Health Organization [WHO] 1993) require that the symptoms be evident in the first 3 years of life, although in the proposed modifications in DSM-5, this requirement has been changed to “Symptoms must be present in early childhood (but may not become fully manifest until social demands exceed limited capacities).” However, nowadays many children are now first identified in the toddler and pre-school period (Charman and Baird 2002; Mandell et al. 2005; Manning et al. 2011), although others, in particular those with average or above average language and cognitive abilities, are not diagnosed until school age or older. Several factors have driven this change, including efforts to improve earlier identification with the recognition that earlier-delivered intervention may improve outcomes and prevent “secondary” neurodevelopmental disturbances (Dawson 2008; Mundy et al. 2009); the development of prospective screening instruments to identify possible cases of autism from the first few years of life (Barbaro and Dissanayake 2009); and the use of the genetic “high-risk” research design of prospectively studying younger siblings of children with a diagnosis of ASD from the first year of life (Rogers 2009; Yirmiya and Charman 2010).

Current Knowledge

One of the most significant challenges and concerns of this new era of prospectively studying children with autism spectrum disorders from the age of 2 and 3 years concerns diagnosis. Given the relatively lack of experience of applying the diagnostic criteria to children of this age, even among the relatively expert clinical teams conducting such research programs, one critical question was whether the diagnosis was possible, accurate, and stable when applied to toddlers at the age of 2 or 3? Over the past decade, a number of research teams have followed up children first seen at the age of 2 years into early, and more recently middle, childhood and provided an answer, to some extent, to this important question.

A number of studies have examined the stability and accuracy of diagnosis, both in samples of children referred for assessment at an early age and from screening studies. Over the past decade, these diagnostic outcome studies have followed cohorts of children from initial diagnostic assessments around the age of 2 years into the preschool years and in several of the more recent studies (Charman et al. 2005; Lord et al. 2006; Turner et al. 2006) into the school age years. The first series of studies (Cox et al. 1999; Lord 1995; Moore and Goodson 2003; Stone et al. 1999) all showed high stability of diagnosis in particular for “core” autism, with somewhat lower stability for broader autism spectrum disorder (ASD) and pervasive developmental disorder not otherwise specified (PDD-NOS). The movement across the ASD/PDD-NOS diagnostic category boundary was somewhat different in the different studies, with Stone et al. (1999) finding that 4 out of 12 children who met broader ASD criteria at the initial assessment did not meet criteria for an autism spectrum disorder at follow-up, whereas Cox et al. (1999) found that 7 from 31 children who did not receive an autism spectrum diagnosis at the initial assessment met criteria for broader ASD at follow-up. Several of the early studies (Cox et al. 1999; Lord 1995; Stone et al. 1999) concluded that for 2-year-olds, expert clinical judgment is more reliable than the standard diagnostic instruments. Several studies also found that

behaviors from the third symptom cluster that defines autism – restricted and repetitive behaviors and activities – were less evident at 2 years of age than at 3–5 years of age (Cox et al. 1999; Moore and Goodson 2003; Stone et al. 1999).

The more recent studies differ in a number of features, most notably in that some have considerably larger sample sizes ($N = 172$, Lord et al. 2006; $N = 89$, Chawarska et al. 2009; $N = 77$, Kleinman et al. 2008) and that the follow-up periods extend to age 7 years in the Charman et al. (2005) study and to age 9 years in the Lord et al. (2006) and Turner et al. (2006) studies. Broadly, the lessons are the same – that the diagnosis of autism is highly stable in these samples but that of broader ASD is less so see Rondeau et al. (2011); for a review). Lord et al. (2006) found that age 2 scores on measures of repetitive and restricted behaviors and activities predicted an autism diagnosis at age 9 years. In some of these more recent studies, there was greater movement from having an ASD diagnosis at age 2 years to a non-spectrum diagnosis at age 4 (Kleinman et al. 2008; Turner and Stone 2007). While the authors report the factors associated with these “good outcomes” – principally higher IQ and better language competency – it is important to remain cautious regarding predictors of poorer or better outcomes in children diagnosed at such a young age.

Future Directions

For clinicians, the lesson is to accept that autism is a developmental disorder and at a very young age, there may be less certainty regarding the pattern of behavior that a child is showing and the likelihood of them continuing to meet diagnostic criteria into the future. Charman and Baird (2002) discuss the importance of understanding the diagnostic process as an iterative process to be worked out between clinician teams and parents over time and that concepts such as a “working diagnosis” can be helpful. An important aspect of early diagnostic consultation is an open and straightforward approach to the negotiation of the diagnostic view

with parents over time. At the same time, clinical teams need to be aware of the need to provide sufficient certainty regarding the child's condition that they are not refused services following assessment.

Clinical work is often concerned with those children who do not clearly meet full criteria for childhood autism but who have apparently milder social problems or where there are mixed developmental difficulties. Clinical experience also suggests that some children who show definite features of autism earlier make remarkable developmental progress. Therefore, caution must be used, especially under 3 years, for those children with features of the broader autistic spectrum who may attract a PDD diagnosis. Experienced clinicians report that parents understand the difficulties of certainty in developmental assessment. Most appreciate honesty on professionals' part about the difficulty of reaching a precise prognosis on a very young child and can understand a frank discussion about the possible outcomes if accompanied by appropriate advice and help for intervention. Understanding why one's child behaves as he or she does is half way to doing something about it.

One other important clinical reminder is that while the trajectory of early emerging impairments in social and communication development accompanied by rigid and repetitive behaviors and interests characterizes many children on the autism spectrum, there is a subgroup of particularly verbal and able children who go on to receive a diagnosis of autism (sometimes called "high functioning autism") or Asperger syndrome who may not receive a diagnosis in the preschool years. There is also another group who might meet diagnostic criteria for an autism spectrum disorder who do not receive an explicit diagnosis – those individuals with moderate to severe intellectual disability or those with an already identified preexisting associated medical condition. In a recent epidemiological study, Baird et al. (2006) found that for cases meeting research diagnostic criteria for an ASD following in-depth assessment, low IQ predicted those who had not received a clinical diagnosis by local clinical services by age 10 years.

One final caveat is that the studies summarized have largely come from expert research clinical centers specifically studying young cohorts of children. In community settings in many countries, there is evidence including from recent studies that for many children and their families, a diagnosis is not confirmed until children are well into the school age years and this has implications for the training of community practitioners (Wiggins et al. 2006).

See Also

- [Diagnosis and Classification](#)
- [Diagnostic Process](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th Ed. – Text Revision) (DSM-IV-TR). Washington, DC: Author.
- Baird, G., Simonoff, E., Pickles, A., Chandler, S., Loucas, T., Meldrum, D., et al. (2006). Prevalence of disorders of the autism spectrum in a population cohort of children in South Thames: The Special Needs and Autism Project (SNAP). *Lancet*, 368, 210–215.
- Barbaro, J., & Dissanayake, C. (2009). Autism spectrum disorders in infancy and toddlerhood: A review of the evidence on early signs, early identification tools, and early diagnosis. *Journal of Developmental and Behavioral Pediatrics*, 30, 447–459.
- Charman, T., & Baird, G. (2002). Practitioner review: Diagnosis of autism spectrum disorder in 2- and 3-year-old children. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 43, 289–305.
- Charman, T., Taylor, E., Drew, A., Cockerill, H., Brown, J. A., & Baird, G. (2005). Outcome at 7 years of children diagnosed with autism at age 2: Predictive validity of assessments conducted at 2 and 3 years of age and pattern of symptom change over time. *Journal of Child Psychology and Psychiatry*, 46, 500–513.
- Chawarska, K., Klin, A., Paul, R., Macari, S., & Volkmar, F. (2009). A prospective study of toddlers with ASD: Short-term diagnostic and cognitive outcomes. *Journal of Child Psychology and Psychiatry*, 50, 1235–1245.
- Cox, A., Klein, K., Charman, T., Baird, G., Baron-Cohen, S., Swettenham, J., et al. (1999). Autism spectrum disorders at 20 and 42 months of age: Stability of clinical and ADI-R diagnosis. *Journal of Child Psychology and Psychiatry*, 40, 719–732.
- Dawson, G. (2008). Early behavioral intervention, brain plasticity, and the prevention of autism spectrum disorder. *Development and Psychopathology*, 20, 775–803.

- Howlin, P., & Asgharian, A. (1999). The diagnosis of autism and Asperger syndrome: Findings from a survey of 770 families. *Developmental Medicine and Child Neurology*, 41, 834–839.
- Johnson, C. P., Myers, S. M., & The American Academy of Pediatrics Council on Children with Disabilities. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120, 1183–1215.
- Kleinman, J. M., Ventola, P. E., Pandey, J., Verbalis, A. D., Barton, M., Hodgson, S., et al. (2008). Diagnostic stability in very young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38, 606–615.
- Lord, C. (1995). Follow-up of two-year-olds referred for possible autism. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 36, 1365–1382.
- Lord, C., Risi, S., DiLavore, P. S., Shulman, C., Thurm, A., & Pickles, A. (2006). Autism from 2 to 9 years of age. *Archives of General Psychiatry*, 63, 694–701.
- Mandell, D. S., Novak, M. M., & Zubritsky, C. D. (2005). Factors associated with age of diagnosis among children with autism spectrum disorders. *Pediatrics*, 116, 1480–1486.
- Manning, S. E., Davin, C. A., Barfield, W. D., Kotelchuck, M., Clements, K., Diop, H., et al. (2011). Early diagnoses of autism spectrum disorders in Massachusetts birth cohorts, 2001–2005. *Pediatrics*, 127, 1043–1051.
- Moore, V., & Goodson, S. (2003). How well does early diagnosis of autism stand the test of time? Follow-up study of children assessed for autism at age 2 and development of an early diagnostic service. *Autism*, 7, 47–63.
- Mundy, P., Sullivan, L., & Mastergeorge, A. M. (2009). A parallel and distributed- processing model of joint attention, social cognition and autism. *Autism Research*, 2, 2–21.
- Rogers, S. J. (2009). What are infant siblings teaching us about autism in infancy? *Autism Research*, 2, 125–137.
- Rondeau, E., Klein, L. S., Masse, A., Bodeau, N., Cohen, D., & Guile, J. M. (2011). Is pervasive developmental disorder not otherwise specified less stable than autistic disorder? A meta-analysis. *Journal of Autism and Developmental Disorders*, 41, 1267–1276.
- Stone, W. L., Lee, E. B., Ashford, L., Brissie, J., Hepburn, S. L., Coonrod, E. E., et al. (1999). Can autism be diagnosed accurately in children under 3 years? *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 40, 219–226.
- Turner, L. M., & Stone, W. L. (2007). Variability in outcome for children with an ASD diagnosis at age 2. *Journal of Child Psychology and Psychiatry*, 48, 793–802.
- Turner, L. M., Stone, W. L., Pozdol, S. L., & Coonard, E. E. (2006). Follow-up of children with autism spectrum disorders from age 2 to age 9. *Autism*, 10, 243–265.
- Volkmar, F. R., State, M., & Klin, A. (2009). Autism and autism spectrum disorders: Diagnostic issues for the coming decade. *Journal of Child Psychology and Psychiatry*, 50, 108–115.
- Wiggins, L. D., Baio, J., & Rice, C. (2006). Examination of the time between first evaluation and first autism spectrum diagnosis in a population-based sample. *Journal of Developmental and Behavioral Pediatrics*, 27, S79–S87.
- World Health Organisation. (1993). *Mental disorders: A glossary and guide to their classification in accordance with the 10th revision of the International Classification of Diseases: Research Diagnostic Criteria (ICD-10)*. Geneva: Author.
- Yirmiya, N., & Charman, T. (2010). The prodrome of autism: Early behavioral and biological signs, regression, peri- and post-natal development and genetics. *Journal of Child Psychology and Psychiatry*, 51, 432–458.

Early Intensive Behavioral Intervention (EIBI)

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Definition

Early intensive behavioral intervention (EIBI) is a treatment approach that is based upon the principles of applied behavior analysis (ABA) and the research of Ivar Lovaas and colleagues at the UCLA Young Autism Project. The EIBI approach has been extensively studied and actively debated in the scientific literature, popular media, and policy arena.

Historical Background

Developed by Lovaas and colleagues across several years of research and development at the University of California–Los Angeles, the EIBI approach has been extensively studied and actively debated in the scientific literature, popular media, and policy arena. Influenced by theories of learning and motivation, practitioners of EIBI refer to it as “the science of teaching.”

Rationale or Underlying Theory

Applied behavior analysis is the overarching philosophy underlying EIBI. Integrating principles from learning theory, operant conditioning, behavioral economics, and motivational theory, proponents of EIBI value the power of changing aspects of the teaching context in order to promote child gains. By providing systematic, direct instruction in an intensive manner (i.e., 30–40 h per week, 1:1 with an adult), proponents of EIBI suggest that young children with autism can improve significantly.

Treatment Participants

Early intensive behavioral intervention (EIBI) is a treatment approach that is thought to benefit children with autism who are younger than 5 years of age. Developers of the intervention emphasize the importance of beginning when a child is as young as possible, hopefully younger than 3 1/2 years.

There is some evidence that children who are more intellectually competent respond best to EIBI.

Treatment Procedures

Based upon the principles of applied behavior analysis (ABA), an EIBI program usually includes these characteristics: (1) active engagement of the child for 40+ h per week in (2) playful intervention, (3) delivered primarily in direct, 1:1 child-adult instruction (4) with specific individual goals, (5) carefully operationalized instructional objectives and procedures, and a (6) data collection system to promote objective observation and (7) analysis of a child's behavioral responses to different aspects of instruction. (8) Teaching strategies are then dynamically revised based upon this analysis, (9) implemented consistently across providers, and (10) evaluated again for effectiveness by monitoring the child's trajectory of skill acquisition. The built-in evaluation system enables families and providers to make dynamic decisions about how and where to modify

approaches, as the child progresses and/or faces new challenges. Young children participating in this treatment usually spend 6–8 h per day in treatment sessions, with breaks every 2–3 h. Often, the sessions happen in the child's home.

Efficacy Information

There is a debate between the proponents and critics of the effectiveness research documenting EIBI, as summarized below (see References and Reading).

Proponents' View

1. Meta-analytic methods (i.e., statistically analyzing all available data across several different studies) and comprehensive scientific reviews support the effectiveness of EIBI for some, but not all, children (Eldevik et al. 2011; Howlin et al. 2009; Matson and Smith 2007; Peters-Scheffer et al. 2011; Reichow and Wolery 2009).
2. Many of the teaching strategies used in EIBI are evidence-based (National Standards Project 2010), including discrete trial training, frequent reinforcement, and ongoing assessment of child behavior.

Critics' Views of EIBI

1. It can be difficult to implement an intensive treatment model for many families, both with regard to time, family stress, and financial resources (Gresham and MacMillan 1998; Schopler et al. 1989).
2. Some of the studies that are in the literature do not demonstrate a lot of improvement, and it is difficult to know which children will respond best to this particular model of intervention.
3. Some researchers are critical of features of the studies which are frequently cited as demonstrative of effectiveness of EIBI (i.e., Lovaas 1987; McEachin et al. 1993; Smith et al. 2000). For example, some early outcome studies reported a return to normal functioning for a substantial number of children (Lovaas 1987), which has been refuted by other scientists

(Shea 2004). Some researchers have expressed caution in interpreting the findings for effectiveness of EIBI, in part due to the ways the outcomes were measured and how decisions about group membership were made, issues of sample size, variability of characteristics of children across studies, lack of intervention fidelity across sites, and some inconsistent findings in studies conducted at replication sites (Bassett et al. 2000; Gresham and MacMillan 1997a, b; Howlin et al. 2009; Myers et al. 2007; Spreckley and Boyd 2008).

4. Developmentally oriented practitioners suggest that the curricula are not sequenced/implemented in a way that is consistent with principles of effective early childhood education.
5. Prioritizing 1:1 direct instruction with the child usually means foregoing instruction in group settings (i.e., preschool), and the child may lack opportunities to learn social and communication skills in real-life settings with other children.
6. Some children become passive learners, cooperative in instructional sessions but not able to spontaneously practice the targeted skill in natural settings.
7. Parent and family involvement is not necessarily a part of the intervention program.

Outcome Measurement

1. Many young children make significant gains in overall developmental functioning with this approach, with improvements in IQ scores ranging from 15 to 25 points after 2 years of intensive intervention during early childhood (see Fenske et al. 1985; Lovaas 1987; McEachin et al. 1993; Sheinkopf and Siegel 1998; Smith et al. 2000). There is some evidence that children who are more intellectually competent respond best to EIBI.
2. Several studies examined school placement and report that children receiving EIBI were likely to be fully included in general education classrooms at first grade (Sallows and Graupner 2005).

Qualifications of Treatment Providers

EIBI is most effective when delivered and supervised by a team of well-trained professionals (Bibby et al. 2001; Smith et al. 2000). EIBI teams tend to be multidisciplinary, with team members who are certified behavior analysts, speech therapists, educators, occupational therapists, and paraprofessionals.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Direct Instruction](#)
- [Lovaas Approach](#)
- [UCLA Young Autism Project](#)

References and Reading

Scientific Studies Examining Effectiveness

- Howard, J. S., Sparkman, C. R., Cohen, H. G., Green, G., & Stanislaw, H. (2005). A comparison of intensive behavior analytic and eclectic treatments for young children with autism. *Research in Developmental Disabilities, 26*, 359–383.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology, 55*, 3–9.
- Sallows, G. O., & Graupner, T. D. (2005). Intensive behavioral treatment for children with autism: Four-year outcome and predictors. *American Journal on Mental Retardation, 110*, 417–438.

Critiques of EIBI

- Shea, V. (2004).

Publications That Review EIBI and Other Early Intervention Programs

- Harris, S. L., & Handleman, J. S. (2001). *Preschool education programs for children with autism* (2nd ed., pp. 23–39). Austin: Pro-Ed.
- National Research Council, Committee on Educational Interventions for Children with Autism. (2008). *Educating children with autism*. Washington, DC: National Academy Press.
- Vismara, L. A. & Rogers, S. J. (2007). Early intervention: Teaching approaches with demonstrated success. *Autism Advocate, 48*, Autism Society of America.

Books for Parents

Maurice, C., Green, G., & Luce, S. L. (1996). *Behavioral intervention for young children with autism: A manual for parents and professionals*. Austin: Pro-Ed.

Websites

For a list of questions to ask about treatments: <http://www.nimh.nih.gov/health/publications/autism/treatment-options.shtml>

<http://www.autismspeaks.org/whattodo/index.php>

Early Intervention

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Synonyms

[Birth-to-three](#); [EI](#)

Definition

Early intervention (EI) refers to publicly funded programs available to infants and toddlers with disabilities and their families, through the Individuals with Disabilities Education Act (IDEA), first authorized by congress in 1986. EI programs offer specialized health, educational, and therapeutic services designed to meet the needs of children, from birth up until age 3, who have a developmental delay or disability, and their families. EI programs often have various names among different states. For example, the statewide EI program in Connecticut is often referred to by parents and professionals as "Birth-to-three," while those in Georgia use the program name "Babies Can't Wait." EI programs receive both federal and state funding, in order to offer services to families free of charge.

To be eligible for services, children must be less than 3 years of age and have a confirmed

disability or established developmental delay, as defined by their state of residence, in one or more of the following areas of development: cognitive, communication, social-emotional, motor skills, and/or adaptive skills.

Following developmental evaluation to determine eligibility, services are typically delivered to children in their home, or within community and center-based programs, or other natural environments. EI service providers include special educators, social workers, speech therapists, physical therapists, occupational therapists, nurses, psychologists, and nutrition specialists.

EI programming may also be provided to children who are considered to be at risk of developing substantial delays if services are not provided.

The following are considered overarching goals of EI: to reduce the likelihood of delays among at-risk children, support improved outcomes and independence among children with developmental disabilities throughout the lifespan, empower and educate families, and to provide intervention to children, regardless of race, ethnicity, or income.

See Also

- [Early Diagnosis](#)
- [First Words Project](#)

References and Reading

- American Speech-Language-Hearing Association. (2008). *Roles and responsibilities of speech-language pathologists in early intervention: Guidelines (Guidelines)*. Retrieved from www.asha.org/policy
- Matson, J. L., & Minshawi, N. F. (2006). *Early intervention for autism spectrum disorders: A critical analysis*. Oxford, UK: Elsevier.
- National Dissemination Center for Children with Disabilities. (n.d.). *State specific information*. Retrieved from: <http://www.nichcy.org/Pages/StateSpecificInfo.aspx>
- National Dissemination Center for Children with Disabilities, Help for Babies (0–3). (n.d.). Overview of early intervention. Retrieved from: <http://www.nichcy.org/babies/overview/Pages/default.aspx>

Early Language Milestone Scale

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Synonyms

[Early language milestone scale-2](#); [ELM scale-2](#)

Description

The Early Language Milestone Scale-2 (ELM Scale-2) was developed for use in pediatric clinical settings as a brief screening of the language abilities of children under the age of 3 years (Coplan 1993). Responses are obtained from a combination of parental/caregiver report, examiner observation, and direct testing. This assessment has three sections: auditory expressive, auditory receptive, and visual. It also provides screening for speech intelligibility (how understandable the child's speech is) at 3–4 years of age. The instrument is composed of 43 items and takes approximately 10 min to administer. The ELM Scale-2 is available in English only.

Historical Background

The ELM Scale-2 was published in 1993, authored by James Coplan, M.D.

Psychometric Data

The measure has a relatively small normed sample of under 200 children under 3 years of age with no breakdown by age within the sample. Eighty percent of the sample is classified as white and 20% is classified as "nonwhite." Eighty percent of the sample is classified as middle-class socioeconomic background and 20% from "lower" socioeconomic background groups. The manual reports higher

inter-rater reliability, but lower test-retest specificity. Concurrent validity data reported supports the use of the ELMS-2 as a screening instrument, to be completed prior to a more thorough communication assessment, if warranted. Although specificity is high, suggesting the measure is valid for identifying 24-month-old children who have normal language development, sensitivity is lower, so that it may be less useful for identifying children with the possibility of language delay.

Clinical Uses

This test is quick and easy to administer with little training. Aside from standardized, comprehensive language assessments administered by trained speech-language pathologist, the ELM Scale-2 can be administered by other medical and healthcare practitioners in pediatric clinical settings and early intervention settings.

The ELM Scale-2 contains scoring options, as it may be administered using a "pass/fail" or a point scoring method. The pass/fail method yields a global pass or fail rating for the test as a whole, whereas the point scoring yields percentile values, standard scores, and age equivalents for each area of language function mentioned above. Its properties suggest it is a viable screening measure; however, due to the small and limited normative sample, standard scores provided for the ELM Scale-2 must be used with caution.

See Also

- [Expressive Language](#)
- [Receptive Language](#)

References and Reading

- Coplan, J. (1993). *Early language milestone scale-2*. Austin: Pro-Ed.
- Paul, R., & Lewis, M. (2007). Assessing communication disorders. In A. Martin, F. Volkmar, & M. Lewis (Eds.), *Child and adolescent psychiatry* (pp. 371–376). New York: Guilford Press.

Early Language Milestone Scale-2

- ▶ [Early Language Milestone Scale](#)

Early Literacy

- ▶ [Emergent Literacy](#)
- ▶ [Emergent Literacy Skills of Preschoolers with Autism](#)

Early Multiword Utterances

- ▶ [Telegraphic Speech](#)

Early School Beginnings

- ▶ [Barriers to and Facilitators of Successful Early School Transitions for Children with Autism Spectrum Disorders](#)

Early Social Communication

- ▶ [Preverbal Communication](#)

Early Social-Communication Scales (ESCS)

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Synonyms

[ESCS](#)

Description

The Early Social Communication Scales is a structured assessment designed to provide measures of individual differences in nonverbal communication skills in children with mental ages between 8 and 30 months of age. The administration requires 15–25 min involving the presentation of approximately 17 tasks which provide opportunities for social communication. Tasks include the presentation of object spectacle toys (e.g., a wind-up toy), turn-taking tasks (e.g., ball play), social interaction (e.g., tickling), gaze following tasks, and opportunities to respond to an invitation to play. The child is typically seated across the table from the examiner, and may be either seated in a chair or seated in their parent’s lap.

The session is videotaped and from the recordings, observers classify children’s behaviors into the following mutually exclusive categories of early social-communication: joint attention behaviors (use of nonverbal behaviors to share experiences), behavioral requests (use of nonverbal behavior to obtain objects or events), and social interaction behaviors (ability to engage in playful turn-taking behavior). These behaviors are also classified based on whether or not they are child-initiated bids or child responses to the examiner.

Historical Background

The ESCS was first developed in 1982 and from a Piagetian stage-based understanding of early development, emphasizing the complexity of a child’s social communicative skills within the context of a child’s goals with a behavior (either communicative or interpersonal). In the previous version, a set of 25 semi-structured situations were utilized to elicit social communication, with approximately 110 possible occurrences of child behavior rated in terms of its complexity, goal, and degree of initiation. The current abridged version of the ESCS was designed to be utilized as a more practical research and clinical tool.

Psychometric Data

Several research studies have been conducted exploring the performance of typically developing children and children with developmental disabilities on the ESCS as well as the reliability of the instrument. In addition, preliminary normative information is available within the ESCS manual (Mundy et al. 2003). In typical populations, performance in responding to joint attention on the ESCS between 14 and 17 months was a significant predictor of subsequent receptive language development (Mundy and Gomes 1998). Research has been conducted on the ESCS across several clinical populations, including children with Down syndrome (Mundy et al. 1995, 1988) and also infants at risk (Sheinkopf et al. 2004). In terms of ASD, research suggests that children with ASD tended to demonstrate the greatest deficits in joint attention behaviors on the ESCS, although difficulties were noted across all areas of nonverbal communication for children on the spectrum (Mundy et al. 1994). Moreover, children with ASD displayed greater deficits in gestural joint attention skills, and these skills significantly predicted language development in children with ASD (Mundy et al. 1990).

Clinical Uses

The ESCS is largely utilized as a research tool (as it requires detailed offline scoring); however, it can also be used as a clinical tool by speech-language pathologists, early childhood specialists, and psychologists, as well as other professionals trained to administer the assessment in the context of a diagnostic or developmental evaluation.

See Also

- [Joint Attention](#)
- [Nonverbal Communication](#)

References and Reading

- Mundy, P., & Gomes, A. (1998). Individual differences in joint attention skill development in the second year. *Infant Behavior and Development*, 21, 469–482.
- Mundy, P., Sigman, M., Kasari, C., & Yirmiya, N. (1988). Nonverbal communication skills in Down syndrome children. *Child Development*, 59, 235–249.
- Mundy, P., Sigman, M., & Kasari, C. (1990). A longitudinal study of joint attention and language development in autistic children. *Journal of Autism and Developmental Disorders*, 20, 115–128.
- Mundy, P., Sigman, M., & Kasari, C. (1994). Joint attention, developmental level, and symptom presentation in young children with autism. *Development and Psychopathology*, 6, 389–401.
- Mundy, P., Kasari, C., Sigman, M., & Ruskin, E. (1995). Nonverbal communication and language development in children with Down syndrome and children with normal development. *Journal of Speech and Hearing Research*, 38, 1–11.
- Mundy, P., Delgado, C., Block, J., Venezia, M., Hogan, A., & Seibert, J. (2003). *A manual for the abridged early social communication scales*. Coral Gables: University of Miami.
- Sheinkopf, S., Mundy, P., Claussen, A., & Willoughby, J. (2004). Infant joint attention skill and preschool behavioral outcomes in at-risk children. *Development and Psychopathology*, 16, 273–291.

Early Stanford-Binet, Fifth Edition (Early SB5)

- [Stanford-Binet Intelligence Scales and Revised Versions](#)

Early Start Denver Model

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Definition

The Early Start Denver Model (ESDM) is a comprehensive early intervention for toddlers with autism ages 12–48 months. The model resulted

from the collaboration of Sally Rogers and Geraldine Dawson and their colleagues at the University of Washington Autism Center, with Rogers' colleagues, especially Laurie Vismara, at the University of California, Davis, and at JFK Partners, University of Colorado Health Sciences Center. The approach is manualized and described in detail by Rogers and Dawson (2010b).

The ESDM and the Denver Model (DM) that preceded it were developed to target the core deficits seen in toddlers and preschoolers with autism: social orientation, attention, affect sharing and attunement, imitation, joint attention, language development, and functional and symbolic play. The ESDM has an interactive communication- and relationship-based framework that fosters active experiential learning by supporting child spontaneity and initiative. It has a developmental curriculum which incorporates teaching techniques that have received empirical support for improving skill acquisition.

The ESDM is based on a fusion of the Denver Model, an affective and developmentally-based intervention for children (ages 2–5) with autism (Rogers 2000); the nature of the teaching interactions and the curricular priorities are influenced by Stern's model of infant interpersonal development (Stern 1985) and pivotal response training (PRT), developed by Laura Schreibman and Robert and Lynn Koegel (Koegel et al. 1989). PRT involves a naturalistic application of applied behavior analysis (ABA) to develop language and social skills.

The main differences between ESDM and DM involve (1) focus on toddlers ages 12–48 months in the ESDM; (2) fusion of practices and principles of PRT with those of the DM; (3) added concept and explicit terminology from applied behavior analysis; (4) more rigorous and defined measurement practices; and (5) a well-defined curriculum appropriate for children 12–48 months of age.

The ESDM and DM have been tested in classroom applications, in one-on-one delivery, in intensive delivery of 15–20 h a week, and via parent delivery. The approach is flexible and designed to be used (1) at home, embedded within typical play and caretaking activities, and in child

care and preschool settings; (2) in 1:1 treatment sessions including parent coaching provided by credentialed providers; and (3) in group programs that can provide individual support to a child.

Historical Background

Development of the Denver Model (DM) began at the University of Colorado Health Sciences Center in 1981, in response to demonstration preschool funding from the US Department of Education. The DM had a developmental and pragmatic approach to language acquisition and emphasis on learning through play and through positive, lively relationships. An interdisciplinary strategy, including a strong role of occupational therapy, positive behavior supports, and a central role of parents, has persisted. The curriculum tool was begun during this period and enhanced and extended for toddlers in the ESDM.

The DM expanded into an approach suitable for use as a 1:1 home-based program during the 1990s. Replications in publically funded sites demonstrated that the model could be implemented in community settings and that child development significantly accelerated with its use.

Significant enhancements of the model occurred in the past 10 years, including more rigorous definitions of delivery and measurement and data collection procedures when the University of Washington tested the approach in a randomized controlled trial with a focus on toddlers (Dawson et al. 2010).

Rationale or Underlying Theory

Three theoretical models provide the foundations for the ESDM curriculum and teaching practices. These include Rogers and Pennington's model of interpersonal development in autism (Rogers and Pennington 1991), Dawson and colleagues' model of autism as a disorder of social motivation (Dawson et al. 2004), and the approach to learning defined by PRT (Schreibman and Pierce 1993).

Rogers and Pennington (1991) hypothesized a developmental model of autism that began with biologically-based deficits in imitation abilities and related impairments in emotional sharing and nonverbal communication in the first year of life. The authors were influenced by Daniel Stern's 1985 model of interpersonal development in infancy. This model presents a theory concerning autism-specific impairments in three developmentally critical behaviors – imitation, emotion sharing, and joint attention. A main focus of the ESDM is to address these critical behaviors within affectively rich relationships with responsive, sensitive others.

The ESDM has been influenced by research on another core feature of autism: impaired social motivation. Dawson and colleagues (Dawson et al. 2004, 2005) have contributed to identifying this characteristic in infants who will develop autism, and they hypothesize that the biology of autism involves a deficiency in social motivation due to the infant's lack of sensitivity to social reward. This lack of sensitivity results in a failure to have a normal preference and active attention to social information, including others' faces, voices, gestures, and speech. This failure, combined with impairments in imitation, emotional sharing, and joint attention, is an obstacle to the child's development of socio-emotional and communicative skills. As a result, the child with autism becomes increasingly removed from the social world and all the learning experiences that exist inside that world. The child falls farther behind because he or she lacks the skills needed to access the social learning environment. Dawson and colleagues have suggested that this lack of engagement not only alters the course of behavioral development but also affects the way neural systems, underlying the perception and representation of social and linguistic information, are developed and organized (Dawson 2008). Several of the strategies utilized in the ESDM are designed to increase the salience of social rewards and enhance the child's attention and motivation for social interaction.

PRT involves a naturalistic use of applied behavior analysis to develop language and social skills. The approach is flexible and is designed to

be embedded within typical play and caretaking activities at home and in child care and preschool settings. PRT is an empirically supported practice, given its documentation of enhanced child motivation, spontaneity, and social initiation and of improved language, maintenance and response generalization, and for concomitant reductions in unwanted behaviors. Child motivation is optimized by the use of reinforcers related to the child's goals and responses and child choice, interspersing acquired tasks with acquisition tasks, therapist reinforcement of attempts to perform the desired behavior, and using activities that are highly motivating to the child. Therapists take turns with the child to share control of the interaction, to capture child attention, and to model behavior that may not be in the child's repertoire.

These three orientations have in common the view that autism impedes an infant's interpersonal experiences. In so doing, it creates barriers to social-communicative development, which lead to greater impairments due to the loss of social learning opportunities.

Goals and Objectives

Goals and Objectives

The goal of ESDM intervention is to increase child social-communicative and relational learning. The main intervention objectives are (1) to bring the child into coordinated, interactive social relations for most of his or her waking hours by supporting all caregivers and therapists to embed ESDM techniques into all daily activities; (2) to provide the child with social learning tools involving imitation, joint attention, language, and social play through teaching inside all daily activities; and (3) to embed a high frequency of specific dyadic learning opportunities in each activity of daily life and also into each intervention activity to "fill in" the learning deficits that have resulted from the past lack of social learning (Rogers 2000). These goals and objectives are accomplished with the following ESDM guiding principles:

- (a) A positive emotional exchange between children and key adults. ESDM intervention activities involve a series of play routines which facilitate the child's pleasure and social engagement and create many opportunities for shared affect and reciprocal interactions.
- (b) Joint activity routines (Ratner and Bruner 1978) are the primary vehicle for teaching, and the teaching episodes are carried out inside this joint activity frame. The play interactions are child-centered, in that children's choices and preferred activities and materials are featured. The adult shares control of the play by selecting what objects are available, what actions are modeled and reinforced, and how activities are sequenced. Joint activities involve objects and activities that are found in natural environments for children of this age. All developmental skills are taught in this way, including the development of a repertoire of sensory motor and constructive, functional, and symbolic play.
- (c) The ESDM is grounded in the science of learning and uses teaching strategies consistent with the principles of applied behavior analysis. ABA has received empirical support, altering the symptoms associated with both autism and general developmental delays (Koegel et al. 1988; Lovaas 1987). Core teaching techniques from PRT that are emphasized in the model include (1) obtaining child attention before delivering an instruction or model; (2) using a clear A-B-C format in teaching trials that are embedded in play activities (the antecedent stimulus precedes the behavior, which is followed by a consequence); (3) reinforcing target skills using intrinsic reinforcers where they exist, pairing nonsocial reinforcers with social attention, and delivering contingent consequences; (4) using shaping, chaining, prompting, fading, and error correction to develop the antecedent-behavior link and to shape a partial performance to a more accurate performance.
- (d) The language intervention approach comes from the science of communication development and recognizes that verbal language develops from nonverbal communication (Bates and Dick 2002; Bruner 1975). Both verbal language and nonverbal communication coordinate people's activities and allow partners to share mental states: intentions, desires, interests, thoughts, and feelings. In each intervention session, multiple and varied communicative opportunities are provided and many communications, both verbal and nonverbal, are elicited from the child. The range of communicative functions is developed so that a child not only requests an activity but also protests, greets familiar adults, shares attention, and comments, as well as other functions. Children's spontaneous communications exert much control over interactions and activities, thus demonstrating the power of communication and assuring its reinforcement.
- (e) The ESDM views autism as disrupting development in all domains (Goodman 1989), and it uses a multidisciplinary approach to address specific domains. Children's developmental skills are evaluated in each domain, and intervention objectives are written for all areas. Though developmental patterns in autism have been understudied, research in different domains has demonstrated that young children with autism follow fairly normal trajectories of development even in their affected areas (see Lifter et al. 1993 regarding symbolic play; Tager-Flusberg et al. 1990 regarding language development; and Ungerer and Sigman 1987 regarding early sensory motor development). The curriculum domain items were extracted from research in early child development in multiple domains: cognition, expressive and receptive language, social-emotional development, fine and gross motor development, self-care skills, play, and imitation. The curriculum was developed by a team of professionals with expertise in developmental and clinical psychology, applied behavior analysis, early childhood special education, speech and language pathology, and occupational therapy. Developmental and clinical psychologists contribute to the sequence of acquisition and the normative strategies for interaction, cognitive

development, play, and imitation. Applied behavior analysts contribute empirically derived strategies for effective teaching and use of functional assessment and analysis to develop approaches for unwanted behaviors and effective teaching practices. Early childhood special education contributes expertise on cognition and play and pre-academic development. Speech and language pathology informs the sequence of speech development, the varied functions of communication, and augmentative and alternative communication approaches. Occupational therapy informs the sequence and content of motor and self-care skills, personal independence, the use of activities to build developmental skills, and optimization of arousal and sensory responsivity to facilitate attention and engagement in learning. Developmental and behavioral pediatrics contributes knowledge of the health concerns of individual children which can interfere with children's ability to benefit from the intervention.

- (f) The ESDM team provides oversight and consultation regarding treatment design and delivery to the parent and primary therapist for each child. At this time, ESDM is primarily delivered by parents and trained therapists providing 1:1 teaching, though it can also be used in group situations, and several research studies are currently in progress concerning efficacy of group delivery. Direct delivery of the intervention is generally directed by one professional, the team leader, or primary therapist, working with parents and therapy assistants in a generalist model, with the multidisciplinary team supporting that primary provider. The generalist delivery approach is used to keep the intervention consistent across treatment sessions and as economical as possible. It also models what parents need to do: address all the child's needs. The full team is available as consultants to the primary therapist and family. When ESDM is delivered in a group preschool setting, the classroom teacher takes the generalist role. The team leader must be credentialed in his or her profession and certified as an ESDM therapist. The team leader is

responsible for a variety of tasks: working with the parents to learn ESDM at home and to assess children's strengths and needs; administering the Curriculum Checklist quarterly and writing 3-month objectives and breaking them down into teaching steps; delivering the intervention and supporting the parents to do so; training and supervising any noncredentialed person who is helping to deliver ESDM; coordinating care with other professionals; and keeping and analyzing daily data and adapting the intervention plan as needed to foster rapid child progress.

- (g) Parent learn to deliver ESDM at home from their team leader, since the embedding of ESDM techniques throughout daily care and play routines is fundamental to the model and necessary for optimal progress. A manual for parents is published by Rogers, Dawson, and Vismara, *An Early Start for Your Child with Autism: Using Everyday Activities to Help Kids Connect, Communicate, and Learn* (2012, Guilford Press).

Treatment Participants

The approach is developed for young children with autism, ages 12–48 months, and developmental quotients over 35 and has been demonstrated to be effective for children with a wide range of skill levels. However, it has only been tested with ambulatory children who have functional hand use and are interested in toys and other objects. It has not been tested with children who have additional diagnoses like Down syndrome or fragile X. In all trials of ESDM, parents have learned to use the methods at home. Effectiveness when parents have not learned to use the method and are not supported on a weekly basis has not been tested.

Treatment Procedures

Each child's learning plan is defined by short-term objectives that represent what is to be learned over a 12-week period and activities carried out daily to

teach the objectives. The objectives are derived from a curriculum assessment carried out each 12 weeks using the ESDM Curriculum Checklist (Rogers and Dawson 2010b). The ESDM Curriculum Checklist covers the following domains: receptive communication, expressive communication, social interaction, imitation skills, cognitive skills, play skills, fine motor skills, gross motor skills, independence/behavior, and joint attention. Each objective is broken down into 5–6 teaching steps that lay out the build-up of the skill. A daily data sheet allows the therapist to teach skills and track progress in very small steps.

Fourteen aspects of the teaching process are quantified in the ESDM fidelity tool. A primary characteristic involves child-initiated activities in which adults embed instruction into play and other daily functional activities, since this kind of teaching has demonstrated gains in spontaneity, motivation, maintenance, and generalization. Adults provide children with choices of materials and activities to support learning of the targeted objectives and to maximize attention and motivation, while considering the child's preferences, age, developmental skills, and learning style.

Joint activity routines (Ratner and Bruner 1978) are the vehicle for teaching. A joint activity routine involves a four-part play activity between child and adult that allows for an activity to be begun, developed, elaborated, and completed. Objectives from two or more different developmental domains are addressed in each 2–5-min joint activity routine. Learning opportunities occur approximately every 10–15 seconds during these activity routines. Transitions between activities are responsive to children's need for change and are accomplished in a fashion that fosters child independence, motivation, and choice. Adult emotion is expected to be warm, positive, and playful. Adults and children interact reciprocally with objects and in communication exchanges, taking turns and sharing leader-follower roles.

Another key aspect of the teaching process involves adult language. Adults use natural language fit to the child's ongoing activity and syntactically constructed using the "one up" rule,

which specifies that the adult's language should involve sentence length that is roughly one word longer than the child's typical sentence. Adults are to narrate, comment, and label actions, objects, and emotions; to model appropriate gestures and speech; and to expand child utterances rather than to question children or tell them what to say.

Children's communication begins with gestural development and multiple pragmatic intents, with a focus on phonetic development, development of intentional vocalization, and reciprocal and meaningful vocal exchanges.

Adult-child interactions are playful and follow children's interests and motivations, addressing short-term objectives from multiple domains. Adults are expected to provide clear learning opportunities, at a rate of several per minute.

A systematic decision process (response to intervention [RTI]) is used in ESDM to "tailor the treatment," by systematically altering teaching procedures if children are not progressing rapidly. This decision tree (Rogers and Dawson 2010b) is highly articulated and allows for a "toolbox" of empirically-based teaching practices for young children with autism to be used if needed, but it prescribes *how*, *when*, and *for how long*, to alter the basic ESDM teaching processes. While the basic ESDM teaching approach favors naturalistic teaching, warm and emotionally positive relationships, varied activities, intrinsic reinforcers, and shared control, no empirically supported teaching approach is "off limits". However, the quality of relationship and communication principles of ESDM is embedded regardless of what specific teaching practices are being used.

Parents are an integral part of ESDM, and their priorities, values, and child-rearing practices are incorporated into quarterly child objectives, teaching practices, and assessment of progress (Vismara et al. 2009a). Parents learn to incorporate the ESDM approach into caretaking and family routines as well as play activities. The transmission process involves a coaching relationship between provider and parents. The goal of parent coaching is to promote the child's social-communication and cognitive development and to promote parent-child communication and play, to foster parents' feelings of competence and

confidence in their interactions with their child, and to maximize child learning opportunities (Vismara et al. 2009b).

Efficacy Information

DM effects as a group preschool intervention were first examined in a series of papers examining pre-post test data (Rogers and DiLalla 1991; Rogers and Lewis 1989). Significant accelerations in developmental rates of young children with autism were found in cognition, language, reduction in autism symptoms, symbolic play, and social engagement. As a group, the children with autism doubled their developmental rates while in active treatment. Four independent replications of the model (Rogers et al. 2006) demonstrated significant accelerations of developmental rates within 6 months of implementation. These studies suggested that the DM has the capacity to affect development in many areas.

The first study of the DM as an individually delivered intervention used a single-subject design which randomized minimally verbal preschoolers to either the DM or the PROMPT treatment (Rogers et al. 2006). PROMPT is a method for treating apraxia of speech developed by Deborah Hayden (formerly Chumpelik) and her colleagues in the early 1980s. The technique was originally developed for adults suffering apraxia of speech secondary to brain injury (Chumpelik 1984). PROMPT involves physical stimulation of the speech motor system during intentional communicative vocalization or verbalization. The DM delivery involved 1 h of individual treatment and parent training weekly and daily 1-h home parent practice sessions for 12 weeks. Eighty percent of children in both approaches acquired functional speech at a frequency of 10–200 words per hour (Rogers et al. 2006).

The most important outcome study of ESDM used a randomized controlled design with 48 toddlers with autism, ages 18–30 months (Dawson et al. 2010). The ESDM group received 15 h per week of individual home treatment over a 24-month period, as well as 4 h per month of parent coaching. The comparison group received

a variety of community interventions for the same period of time. Over a 24-month period, the ESDM group demonstrated large and highly significant increases compared to the community intervention group in overall IQ gains, receptive and expressive language gains, and adaptive behavior functioning. These differences were not likely due to the number of intervention hours delivered, since the two groups' intervention hours were very similar. Follow-up studies of these children will determine the trajectory of their development 5 years after this study. A multisite replication of this study involving 100 12–24-month-olds is ongoing.

Outcome Measurement

Child learning objectives in all core developmental areas are written every 12 weeks. Child progress is monitored after every 15 min of treatment on “daily data sheets” developed from the child's short-term objectives. Measurable progress is expected on every objective within a 1–2-week period and mastery of all objectives is assessed every 12 weeks. New objectives are written and broken down into small teachable steps, and new data sheets are developed at these 12-week intervals. Annual progress is measured through a battery of standardized tests, involving development, adaptive behavior, vocabulary and gesture use, and the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 1999) to measure the number and severity of autism symptoms.

Qualifications of Treatment Providers

ESDM treatment providers have completed multiple levels of training and have received certification as ESDM therapists. Those who coach families to use ESDM at home have also been certified as ESDM parent coaches, and those who train others to use ESDM are certified as ESDM trainers. Training is available at multiple sites in the USA and abroad, and those seeking training can find information on the ESDM website. All ESDM published studies were

carried out by trained and credentialed therapists and staff. Professionals from a wide number of disciplines may be trained as ESDM therapists. Those professions include occupational therapy, physical therapy, speech and language pathology, early childhood education, early childhood special education, clinical child psychology, school psychology, and other professions that include background in early childhood development as part of the graduate curriculum. Those without a professional credential cannot be certified as an ESDM therapist, though they may work under the training and supervision of an ESDM-credentialed therapist.

The ESDM was developed to be provided by a team of childhood professionals with training and experience in early autism intervention from a variety of disciplines. The team leader is credentialed in their profession and as an ESDM provider and is responsible for a variety of tasks: working with the parent to assess the child's needs and strengths; developing short-term objectives and breaking them down into teaching steps; delivering the intervention and supporting the parent to do so; training and supervising any non-licensed persons who are helping deliver ESDM; and keeping and analyzing data to assure child progress or to adapt the intervention methods to improve progress.

Parents learn to deliver ESDM at home from their team leader, since the embedding of ESDM techniques in play and caretaking routines is necessary for optimal progress. Materials are available to help parents adopt the techniques, and a manual is currently being published (Rogers et al. 2012).

For children who are receiving many hours of ESDM weekly, well-trained paraprofessionals may deliver the intervention under the ongoing supervision of the team leader. Paraprofessionals learn to deliver the model at appropriate levels of fidelity of intervention under the supervision and guidance of trained ESDM professionals. Fidelity should be checked frequently when multiple adults are delivering ESDM. Team meetings should occur at least every 2 weeks to review child progress, discuss the intervention plan, and assure intervention consistency.

While printed ESDM materials are available to the public, it is usually necessary to attend specific training to master the ESDM. Those wanting to learn ESDM can access training materials through the MIND Institute website. Information about training workshops in various cities and countries is also posted.

<http://www.ucdmc.ucdavis.edu/mindinstitute/research/esdm/certification.html>

See Also

- ▶ [ABA](#)
- ▶ [Imitation](#)
- ▶ [Joint Attention](#)
- ▶ [Occupational Therapy \(OT\)](#)

References and Reading

- Bates, E., & Dick, F. (2002). Language, gesture, and the developing brain. *Developmental Psychobiology*, 40(3), 293–310.
- Bruner, J. (1975). The ontogenesis of speech acts. *Journal of Child Language*, 2, 1–19.
- Chumpelik, D. (1984). The PROMPT system of therapy: Theoretical framework and applications for developmental apraxia of speech. *Seminars in Speech and Language*, 5, 139–156.
- Dawson, G. (2008). Early behavioral intervention, brain plasticity, and the prevention of autism spectrum disorder. *Development and Psychopathology*, 20, 775–803.
- Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., & Liaw, J. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology*, 40(2), 271–283.
- Dawson, G., Webb, S. J., & McPartland, J. (2005). Understanding the nature of face processing impairment in autism: Insights from behavioral and electrophysiological studies. *Developmental Neuropsychology*, 27(3), 403–424.
- Dawson, G., Rogers, S., Munson, J., Smith, M., Jamie, W., Greenson, J., et al. (2010). Randomized controlled trial of the Early Start Denver Model: A developmental behavioral intervention for toddlers with autism: Effects on IQ, adaptive behavior, and autism diagnosis. *Pediatrics*. <https://doi.org/10.1542/peds.2009-0958>.
- Goodman, R. (1989). Infantile autism: A syndrome of multiple primary deficits. *Journal of Autism and Developmental Disorders*, 19(3), 409–424.
- Koegel, R. L., O'Dell, M. C., & Dunlap, G. (1988). Producing speech use in nonverbal autistic children by

- reinforcing attempts. *Journal of Autism and Developmental Disorders*, 18, 525–538.
- Koegel, R. L., Schreibman, L., Good, A., Cerniglia, L., Murphy, C., & Koegel, L. (1989). *How to teach pivotal behaviors to children with autism: A training manual*. Santa Barbara: University of California.
- Koegel, L. K., Koegel, R. L., Hurley, C., & Frea, W. D. (1992). Improving social skills and disruptive behavior in children with autism through self-management. *Journal of Applied Behavior Analysis*, 25, 341–353.
- Lifter, K., Sulzer-Azaroff, B., Anderson, S. R., Coyle, J. T., & Cowdery, G. E. (1993). Teaching play activities to preschool children with disabilities: The importance of developmental considerations. *Journal of Early Intervention*, 17, 139–159.
- Lord, C., Rutter, M., DiLavore, P., & Risi, S. (1999). *Autism diagnostic observation schedule: Manual*. Los Angeles: Western Psychological Services.
- Lovaas, O. I. (1987). Behavioral treatment and normal education and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- Ratner, N., & Bruner, J. (1978). Games, social exchange, and the acquisition of language. *Journal of Child Language*, 5, 391–402.
- Rogers, S. J. (2000). Interventions that facilitate socialization for children with autism. *Journal of Autism and Developmental Disorders*, 30(5), 399–410.
- Rogers, S. J., & Dawson, G. (2010a). *Early Start Denver Model for young children with autism: Promoting language, learning, and engagement*. New York: Guilford.
- Rogers, S. J., & Dawson, G. (2010b). *Early Start Denver Model curriculum checklist for young children with autism*. New York: Guilford.
- Rogers, S. J., & DiLalla, D. L. (1991). A comparative study of the effects of a developmentally based preschool curriculum on young children with autism and young children with other disorders of behavior and development. *Topics in Early Childhood Special Education*, 11(2), 29–47.
- Rogers, S. J., & Lewis, H. (1989). An effective day treatment model for young children with pervasive developmental disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28, 207–214.
- Rogers, S. J., & Pennington, B. F. (1991). A theoretical approach to the deficits in infantile autism. *Development and Psychopathology*, 3(2), 137–163.
- Rogers, S. J., Hall, T., Osaki, D., Reaven, J., & Herbison, J. (2000). The Denver model: A comprehensive, integrated, educational approach to young children with autism and their families. In S. Harris & J. Handleman (Eds.), *Preschool education programs for children with autism* (2nd ed.). Austin: Pro-Ed.
- Rogers, S. J., Hayden, D., Hepburn, S., Charlifue-Smith, R., Hall, T., & Hayes, A. (2006). Teaching young nonverbal children with autism useful speech: A pilot study of the Denver Model and PROMPT interventions. *Journal of Autism and Developmental Disorders*, 36(8), 1007–1024.
- Rogers, S. J., Dawson, G., & Vismara, L. A. (2012). *An early start for your child with autism: Using everyday activities to help kids connect, communicate, and learn*. New York: Guilford.
- Schreibman, L., & Pierce, K. (1993). Achieving greater generalization of treatment effects in children with autism: Pivotal response training and self-management. *Clinical Psychology*, 46, 184–191.
- Stern, D. N. (1985). *The interpersonal world of the infant: A view from psychoanalysis and developmental psychology*. New York: Basic Books.
- Tager-Flusberg, H., Calkins, S., Nolin, T., Baumberger, T., Anderson, M., & Chadwick-Dias, A. (1990). A longitudinal study of language acquisition in autistic and Down syndrome children. *Journal of Autism and Developmental Disorders*, 20(1), 1–21.
- Ungerer, J. A., & Sigman, M. (1987). Categorization skills and receptive language development in autistic children. *Journal of Autism and Developmental Disorders*, 17(1), 3–16.
- Vismara, L. A., Colombi, C., & Rogers, S. J. (2009a). Can 1 hour per week of therapy lead to lasting changes in young children with autism? *Autism: An International Journal*, 13(1), 93–115.
- Vismara, L. A., Young, G. S., Stahmer, A. C., Griffith, E. M., & Rogers, S. J. (2009b). Dissemination of evidence-based practice: Can we train therapists from a distance? *Journal of Autism and Developmental Disabilities*, 39(12), 1636–1651.

Early-Life Exposure to Antibiotics and Autism Spectrum Disorders

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Definition

Autism spectrum disorders (ASD) encompass a range of neurodevelopmental disorders, which include autism and Asperger syndrome. Early-life exposure to antibiotics includes their use by pregnant mothers (prenatal) and children during early childhood. The gut microbiota refers to the many species of microorganisms living in our intestine.

Historical Background

One of the key subjects in the field of human microbiota research is its connection to neurodevelopmental and psychiatric conditions. A number of studies have confirmed that individuals with autism have different intestinal microbiome compositions compared to those of healthy individuals (Kraneveld et al. 2016). Interactions between the gastrointestinal tract and the central nervous system, which are explained by the so-called microbiome-gut-brain axis model (Cryan and Dinan 2012), form the rationale for investigating the gut microbiota as a possible contributor to the pathogenesis of autism. According to the aforementioned model, disturbed immune response and release of cytokines triggered by microbiota alterations may affect function of the central nervous system. Antibiotics are proposed as a potential causative factor for this phenomenon, since they are known to disturb the composition of microbiota in infancy (Yassour et al. 2016).

The first reports that linked autism symptoms with antibiotic-associated microbiota alterations were published in the early 2000s. In 2000, Sandler et al. described a group of 11 autistic children who developed ASD symptoms and chronic diarrhea shortly after use of broad-spectrum antimicrobials. Subsequently, the children underwent an 8-week course of oral vancomycin therapy, after which a transient improvement was observed in their behavior and communication skills (Sandler et al. 2000). A case series published 5 years later described 206 children who reportedly received multiple courses (mean number per child: ~12) of antibiotics for multiple bouts of otitis media before the occurrence of autism symptoms (Fallon 2005). The aforementioned reports lacked control groups – or, in case of the 2005 study, any statistical analysis – therefore, their findings were insufficient to allow clear conclusions to be drawn. However, after these first claims of an association between early-life antibiotic exposure and the occurrence of autism, a number of larger observational studies with analytical design were performed.

Current Knowledge

The largest studies on the subject performed to date derived data from Danish and Canadian population-based cohorts. A study of more than 95,000 Danish children (Atladdottir et al. 2012) reported a borderline significant increase in the risk of ASD after use of any antibiotics during pregnancy. A slightly stronger association of prenatal antibiotic exposure with the risk of ASD was reported for the use of sulfonamides, whereas macrolides were associated with a higher risk of infantile autism. On the other hand, the authors pointed out that multiple testing could be a potential limitation of the study. In total, more than 100 comparisons were made; therefore, the few statistically significant associations could be chance findings.

Early childhood antibiotic exposure as a risk factor for ASD was investigated in a study of more than 200,000 Canadian children (Hamad et al. 2018). There was a trend toward a reduced risk of ASD in children exposed to any antibiotics during the first year of life; however, no such association was found in an analysis among siblings. In another study of more than 650,000 Danish children, a slightly increased risk of autism was observed in children exposed postnatally to penicillin or to broader-spectrum antibiotics compared to children unexposed to antibiotics (Axelsson et al. 2019). In this study as well, a significant association was no longer observed in the sibling model. Another cohort study of more than 780,000 Danish children reported a slightly increased risk of autism after use of broad- and moderate-spectrum antibiotics (Wimberley et al. 2018). However, the population of this study overlapped with the population of the previous one. Moreover, the findings were not explored in sibling analysis. Data from the aforementioned cohort studies do not support a clinically significant association between early childhood antibiotic exposure and the development of ASD. Authors of pointed out that the borderline significance of the results and lack of confirmation of findings in sibling analyses contribute to ambiguity of the results.

Additionally, the association between ASD and early-life antibiotic exposure has been evaluated in a number of smaller case-control studies. In some of them, no significant associations were found (George et al. 2014; Guisso et al. 2018; Isaksson et al. 2017). In others, a slightly increased risk of ASD was reported after use of various antibiotics (Bittker and Bell 2018; Grossi et al. 2018; Niehus and Lord 2006).

In 2019, a systematic review summarized the available evidence on pre- and postnatal antibiotic exposure and subsequent risk of ASD (Lukasik et al. 2019). This review included 11 observational studies, consisting of cohort studies and case-control studies. The results of the included studies were conflicting, and the reported significant associations were usually of borderline significance or were derived from case-control studies with a considerable risk of bias. Moreover, a meta-analysis performed by the authors did not yield any statistically significant results. The authors of the review concluded that the included articles do not provide strong support for the hypothesis that early-life antibiotic exposure is associated with subsequent development of ASD.

Future Directions

As of now, there are not enough available data to conclusively rule out or confirm the notion that antibiotic use is a risk factor for ASD. It needs to be emphasized that this field of research faces a number of significant difficulties. Firstly, it is hard to discern whether the observed associations are due to the antibiotics themselves or are due to the underlying infections. Antibiotics are usually used to treat an infection, so controlling for this confounding factor creates a serious challenge for the investigators. This obstacle could be partially overcome by identifying patterns of microbiota disturbance associated with ASD. However, even though some characteristic microbiota alterations have been identified (Rosenfeld 2015), it is unknown if a definable pattern of “ASD microbiome” exists. Moreover, even if

such a pattern is discovered, it would be difficult to link it to the impact of a particular antibiotic, as effects of antibiotics on microbiota are not specific (Yassour et al. 2016). To acquire a definitive answer to the question as to whether an antibiotic-ASD association exists, more studies are needed – both in epidemiology and in the field of the human microbiome.

See Also

- [Environmental Risk Factors for Autism](#)
- [Gastrointestinal Disorders and Autism](#)

References and Reading

- Atladdottir, H. O., Henriksen, T. B., Schendel, D. E., & Parner, E. T. (2012). Autism after infection, febrile episodes, and antibiotic use during pregnancy: An exploratory study. *Pediatrics*, 130(6), e1447–e1454. <https://doi.org/10.1542/peds.2012-1107>.
- Axelsson, P. B., Clausen, T. D., Petersen, A. H., Hageman, I., Pinborg, A., Kessing, L. V., et al. (2019). Relation between infant microbiota and autism?: Results from a National cohort sibling design study. *Epidemiology*, 30(1), 52–60. <https://doi.org/10.1097/ede.0000000000000928>.
- Bittker, S. S., & Bell, K. R. (2018). Acetaminophen, antibiotics, ear infection, breastfeeding, vitamin D drops, and autism: An epidemiological study. *Neuropsychiatric Disease and Treatment*, 14, 1399–1414. <https://doi.org/10.2147/ndt.s158811>.
- Cryan, J. F., & Dinan, T. G. (2012). Mind-altering microorganisms: The impact of the gut microbiota on brain and behaviour. *Nature Reviews. Neuroscience*, 13(10), 701–712. <https://doi.org/10.1038/nrn3346>.
- Fallon, J. (2005). Could one of the most widely prescribed antibiotics amoxicillin/clavulanate “augmentin™” be a risk factor for autism? *Medical Hypotheses*, 64(2), 312–315. <https://doi.org/10.1016/j.mehy.2004.06.023>.
- George, B., Padmam, M. S. R., Nair, M. K. C., Leena, M. L., & Russell, P. S. S. (2014). CDC Kerala 13: Antenatal, natal and postnatal factors among children (2–6 y) with autism – A case control study. *Indian Journal of Pediatrics*, 81(2), 133–137. <https://doi.org/10.1007/s12098-014-1594-1>.
- Grossi, E., Migliore, L., & Muratori, F. (2018). Pregnancy risk factors related to autism: An Italian case-control study in mothers of children with autism spectrum disorders (ASD), their siblings and of typically developing children. *Journal of Developmental Origins of*

- Health and Disease*, 9(4), 442–449. <https://doi.org/10.1017/s2040174418000211>.
- Guisso, D. R., Saadeh, F. S., Saab, D., El Deek, J., Chamseddine, S., El Hassan, H. A., et al. (2018). Association of autism with maternal infections, perinatal and other risk factors: A case-control study. *Journal of Autism and Developmental Disorders*, 48(6), 2010–2021. <https://doi.org/10.1007/s10803-017-3449-x>.
- Hamad, A. F., Alessi-Severini, S., Mahmud, S. M., Brownell, M., & Kuo, I. F. (2018). Early childhood antibiotics use and autism spectrum disorders: A population-based cohort study. *International Journal of Epidemiology*, 47(5), 1497–1506. <https://doi.org/10.1093/ije/dyy162>.
- Isaksson, J., Pettersson, E., Kostrzewa, E., Diaz Heijtz, R., & Bolte, S. (2017). Brief report: Association between autism spectrum disorder, gastrointestinal problems and perinatal risk factors within sibling pairs. *Journal of Autism and Developmental Disorders*, 47(8), 2621–2627. <https://doi.org/10.1007/s10803-017-3169-2>.
- Kraneveld, A. D., Szklany, K., de Theije, C. G. M., & Garssen, J. (2016). Gut-to-brain axis in autism spectrum disorders: Central role for the microbiome. *International Review of Neurobiology*, 131, 263–287.
- Lukasik, J., Patro-Golab, B., Horvath, A., Baron, R., & Szajewska, H. (2019). Early life exposure to antibiotics and autism spectrum disorders: A systematic review. *Journal of Autism and Developmental Disorders*, 49(9), 3866–3876. <https://doi.org/10.1007/s10803-019-04093-y>.
- Niehuis, R., & Lord, C. (2006). Early medical history of children with autism spectrum disorders. *Journal of Developmental and Behavioral Pediatrics*, 27(2 Suppl), S120–S127.
- Rosenfeld, C. S. (2015). Microbiome disturbances and autism spectrum disorders. *Drug Metabolism and Disposition*, 43(10), 1557–1571. <https://doi.org/10.1124/dmd.115.063826>.
- Sandler, R. H., Finegold, S. M., Bolte, E. R., Buchanan, C. P., Maxwell, A. P., Vaisanen, M. L., et al. (2000). Short-term benefit from oral vancomycin treatment of regressive-onset autism. *Journal of Child Neurology*, 15(7), 429–435. <https://doi.org/10.1177/088307380001500701>.
- Wimberley, T., Agerbo, E., Pedersen, C. B., Dalsgaard, S., Horsdal, H. T., Mortensen, P. B., et al. (2018). Otitis media, antibiotics, and risk of autism spectrum disorder. *Autism Research*, 11(10), 1432–1440. <https://doi.org/10.1002/aur.2015>.
- Yassour, M., Vatanen, T., Siljander, H., Hamalainen, A. M., Harkonen, T., Ryhanen, S. J., et al. (2016). Natural history of the infant gut microbiome and impact of antibiotic treatment on bacterial strain diversity and stability. *Science Translational Medicine*, 8(343), 343ra381. <https://doi.org/10.1126/scitranslmed.aad0917>.

Eating Disorders

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Synonyms

Anorexia; Bulimia nervosa; Pica; Rumination disorder

Short Description or Definition

Eating disorders like anorexia and bulimia nervosa (AN/BN) are complex psychosomatic disorders of unknown etiology, primarily affecting adolescent girls and young women. They are characterized by aberrant patterns of eating behavior and weight regulation which in AN result in weight loss and endocrine abnormalities such as amenorrhea, disturbances in attitude and perception about weight and shape, and an intense fear of gaining weight. Patients are classified AN according to the Diagnostic and Statistical Manual 4th edition (DSM-IV, American Psychiatric Association 1994) when they are incapable to maintain a body weight above a minimal normal level ($\text{BMI} < 17.5 \text{ kg/m}^2$ for adults), demonstrate an intense fear of becoming fat, have disturbed perceptions of body shape and size, and (after menarche) show amenorrhea. Two subgroups are classified, the restricting type in which weight loss is the result of dietary restriction and the binge/purge type in which periods of bingeing or purging and dietary restriction coexist. When compared to other psychiatric disorders, AN has the highest mortality rate: 10–15%. BN shares many features with AN but for the weight loss.

Accompanying symptoms, such as perfectionism, obsessive-compulsive behavior, and social

anxiety, are observed in many but not all patients. This characteristic cluster of personality and temperamental traits often persists after recovery and has been described preceding the onset of disease as well. In addition, a large proportion of AN patients displays abnormally high physical activity levels and over exercises, although estimations vary from 31% to 80% because of definition difference.

Although the etiology of AN is as yet unclear, a combination of cultural, social, psychological, genetic, and biological factors is implicated: “Genes load the gun, environment pulls the trigger” (Bulik and Tozzi 2004).

Other problems sometimes observed include pica (eating of nonnutritive substances) and rumination (repeated regurgitation and rechewing of food).

Categorization

The combination of eating disorders with autism spectrum disorders is uncommon and little researched. Most studies focus on disturbed eating behavior in autistic children. Some studies find that the behaviors themselves also occur in normally developing children, but much less often. Normally developing children seem to outgrow these behaviors while children with a disorder in the autistic spectrum do not. Two-thirds of parents of children with autistic spectrum disorder describe picky eating in their children. The most frequently described eating disturbances are food neophobia and selective eating; other disturbances such as pica and rumination seem to be more common in autism associated with mental retardation.

Problems with eating – including particular food refusal, food fads, pica, hoarding, overeating, and various degrees of anorectic behaviors, including complete food refusal and compulsive ordering of food on the plate – are extremely common in autism. Pica and rumination may also be seen.

Epidemiology

The total incidence of AN in the Netherlands is relatively stable. In the period of 1995–1999, the

age and sex adjusted incidence rate was 7.7/100,000. Interestingly, the incidence in female 15–19-year-olds (as well as 10–14-year-olds) almost doubled when compared to 10 years before, indicating that the onset of the disease is currently taking place at a younger age (Van Son 2006). Average prevalence rates of AN in Europe appear stable at 0.29% (Hoek and van Hoeken 2003).

In a controlled study of representative cases of anorexia nervosa, 18% had an autism spectrum disorder (4% autistic disorder, 6% Asperger syndrome, and 8% atypical autism), both at the time of onset of the eating disorder (around 15 years of age) and 5 and 10 years later.

Natural History, Prognostic Factors, and Outcomes

Recovery of anorexia nervosa in general takes a long time; it has been reported that stable physical recovery is reached after on average 4.7 years and psychosocial recovery after 6.6 years. Final outcome figures of AN leave room for improvement. According to Steinhausen (2002), 20.8% (0–79%) of AN patients remain chronically ill, and 5.3% (0–22%) die as a consequence of starvation or by suicide. Chances for recovery range from 0% to 92%, averaging at 46.5%. Data in younger patient groups are more optimistic, reaching about 60% for complete recovery.

Clinical Expression and Pathophysiology

A large number of studies indicate that there may be a propensity for underweight or comorbid eating disorders. One-fourth of male cases with autism of Asperger syndrome had a BMI in the 5th percentile or below, though no eating disorder diagnoses were justified. No association was found between autistic behavior and BMI, but there was a small effect of hyperactivity.

Disturbed Eating Behaviors in ASD

Rumination, the repetitive regurgitation of recently ingested food into the mouth with

subsequent spitting or remastication and swallowing, has been described in adolescents and adults with autism. It can lead to tooth erosion caused by gastric acid, as well as malnutrition and esophageal abnormalities.

Pica, or eating inedible nonfood substances, seems to be a problem especially in institutionalized cases of mental retardation associated with autism. When present, it is potentially dangerous and difficult to treat. Pica may result in gastrointestinal problems or poisoning with heavy metals. Overeating.

Eating disturbances in Asperger, teenage boys: selective eating and hyperactivity resulting in low body weight. In adults with ASD and normal intelligence, health reasons are often given for special diets.

Psychogenic polydipsia (excessive water intake) presents a problem in autism with mental retardation.

Food neophobia is the avoidance of all new foods. This has been linked to selective eating but should be treated separately.

Selective eating (eating only ten foods or fewer, sometimes food of a special color, texture, or brand, avoidance or refusal of new foods)

Proposed Underlying Mechanisms in Disturbed Eating Behaviors in ASD

Sensory abnormalities: Hyper/hyposensitivity to auditory, visual, tactile stimuli and to smell and taste may play a large part in mealtime behaviors.

Interests: Restricted and intense ideas and interests, for example, having strict ideas about the appropriate amount of food required without paying attention to feeling of hunger or satiety, fixating on ideas of which food are healthy etc.

Routines: Ritualized eating routines.

Social interaction and other skills involved in dining: Eating with other people is an ordeal for many persons with autism because of doing several tasks simultaneously, leading to unusual table manners in some cases.

Motor functioning: Motor problems are common in ASD, which can make the use of knife and fork difficult.

Gastrointestinal problems: A high rate of gastrointestinal symptoms is reported in children with

ASD compared to peers, in most cases without known medical causes.

Anorexia nervosa: It is suggested that ASD and AN share some features, namely, obsessiveness, insistence on sameness, and social impairment. It is suggested that a subgroup of AN has a cognitive style very similar to ASD.

Gender issues: AN and Asperger are both considered to be specific to one gender; perhaps if the criteria of AN would be reconsidered with the male population in mind, and the Asperger criteria would include more behaviors and attitudes more appropriate to females, much more overlap would occur.

Evaluation and Differential Diagnosis

Evaluation and assessment vary depending on the nature of the eating problem. In all cases, a careful search for possible contributory factors (environmental, associated medical conditions, and so forth) is indicated. Weight loss and eating problems can be observed in various disorders (e.g., depression), and sometimes unusual beliefs about food or eating are seen in other psychiatric conditions (e.g., schizophrenia). Diagnosis can be more complicated in individuals with autism and with cognitive disability.

Treatment

Treatments vary depending on the specific of the situation and the nature of the difficulty. Some syndromes of intellectual disability are associated with eating problems. For pica and rumination disorder, various behavioral procedures have been used. In younger children, it is important to be sure that other medical problems or environmental factors do not complicate the situation. In anorexia nervosa and bulimia nervosa, treatment is typically multifaceted with outpatient treatment preferred if possible. Pharmacological treatments may be combined with cognitive behavior therapy approaches depending on the specific situation.

See Also

- ▶ [Asperger Syndrome](#)
- ▶ [Cognitive Flexibility](#)
- ▶ [Extreme Male Brain \(EMB\) Theory](#)
- ▶ [Gender Differences](#)
- ▶ [Pica](#)
- ▶ [Rumination](#)
- ▶ [Weak Central Coherence](#)

References and Reading

- Bölte, S., Özkara, N., & Poutska, F. (2002). Autism spectrum disorders and low body weight: Is there really a systematic association? *International Journal of Eating Disorders*, 31, 349–351.
- Råstam, M. (2008). Eating disturbances in autism spectrum disorders with focus on adolescent and adult years. *Clinical Neuropsychiatry*, 5, 31–42.
- Schreck, K. A., Williams, K., & Smith, A. F. (2004). A comparison of eating behaviors between children with and without autism. *Journal of Autism and Developmental Disorders*, 34, 433–434.
- Volkmar, F. R., & Martin, A. (2011). *Essentials of child and adolescent psychiatry*. Philadelphia: Lippincott, Williams, and Wilkins.
- Zucker, N. L., Losh, M., Bulik, C. M., LaBar, K. S., Piven, J., & Pelphry, K. A. (2007). Anorexia nervosa and autism spectrum disorders: Guided investigation of social cognitive endophenotypes. *Psychological Bulletin*, 133, 976–1006.

ECHO Autism STAT

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Definition

ECHO Autism STAT is a training program for primary care providers (PCPs) that combines hands-on training in autism diagnosis with ongoing virtual mentorship using the Extension for Community Healthcare Outcomes (Project ECHO) framework.

Historical Background

Early identification and diagnosis of autism spectrum disorder (ASD) are essential for facilitating access to intervention and services. Younger age at entry to intervention is associated with greater improvements across developmental domains for children with ASD (MacDonald et al. 2014; Perry et al. 2011; Smith et al. 2015; Vivanti et al. 2016). However, many children in the United States experience significant diagnostic delays, waiting an average of 3–6 years between first concerns and ultimate diagnosis (Baio et al. 2018; Sheldrick et al. 2017; Oswald et al. 2017). This has resulted in large-scale federal and professional efforts to prioritize early identification of ASD (Filipek et al. 2000; Healthy People 2020; Interagency Autism Coordinating Committee 2017; Myers et al. 2007).

Despite these efforts, the average age at ASD diagnosis remains between 4 and 7 years for children in the United States (Baio et al. 2018). Shortages of healthcare providers with training in autism have contributed to these delays, with many local communities having no access to autism specialists. Lack of local autism expertise, long wait lists at specialty centers, and high costs of services have rendered best-practice care inaccessible for many families. Underserved children and families are disproportionately affected by these barriers. Children from families with low income or socioeconomic status, those living in rural areas, and those from minority racial and ethnic groups face even greater diagnostic delays (Fountain et al. 2011; Mandell et al. 2010; Mandell et al. 2002; Rosenberg et al. 2011).

In 2007, the American Academy of Pediatrics (AAP) published guidelines recommending that autism screening be conducted at regular well-child visits in order to help expedite early diagnosis for all children (Myers et al. 2007). However, physician compliance with these guidelines has remained remarkably poor (Arunyanart et al. 2012; Keil et al. 2014; Self et al. 2015). Pediatricians and other primary care physicians report receiving very little formal training on autism during medical school or residency training (Fenikilé et al. 2015; Finke et al. 2010), and a

lack of knowledge about autism and unfamiliarity with appropriate screening tools has been identified by PCPs as primary barriers to screening implementation in their practices (Fenikilé et al. 2015; Self et al. 2015). Even when children are appropriately screened and referred for further evaluation, significant wait times at specialty centers contribute to additional diagnostic delays. Given the importance of early intervention for young children with ASD, new models are needed to reduce diagnostic bottlenecks and to improve access to early diagnosis in local communities.

Building local expertise among community-based providers who serve young children and families has the potential to reduce diagnostic delays and disparities for underserved families. Primary care providers (PCPs) offer accessible and integrated healthcare services and are tasked with conducting developmental surveillance and screening for all children. As such, they are perfectly positioned to help close the gap between first concerns and diagnosis, particularly if provided with additional knowledge and tools for ASD assessment.

Current Knowledge

The ECHO Autism STAT program was developed to reduce diagnostic delays by training community-based PCPs in underserved areas in screening and diagnosis of young children at highest risk for autism (Mazurek et al. 2019). The program combines hands-on training in standardized tools with ongoing virtual mentorship and practice, thus facilitating both timely diagnosis and appropriate referral for comprehensive assessment when necessary. The program builds upon and extends the technology-based Project Extension for Community Healthcare Outcomes (Project ECHO) framework. Project ECHO was originally developed to increase access to treatment of hepatitis C virus infection by training community-based PCPs (Arora et al. 2010; Arora et al. 2011) and was more recently adapted for autism (Mazurek et al. 2017). Project ECHO's technology-based mentorship model reduces geographic barriers to specialty knowledge and is

based on adult learning principles that maximize practical application and knowledge retention.

The ECHO Autism STAT model (Mazurek et al. 2019) follows a multitiered approach to diagnosis in which the assessment method is determined by the child's symptom presentation and complexity. The program was designed to train PCPs in diagnosis of young children whose symptoms are unambiguous and in appropriate referral for complex cases that require more comprehensive assessment. The program begins with a 1.5-day face-to-face training focused on autism assessment and administration of the Screening Tool for Autism in Toddlers and Young Children (STAT), a standardized play-based observational autism assessment tool for very young children (Stone et al. 2000). After the in-person training, PCPs join bimonthly 90-min ECHO Autism STAT sessions for 12 months, during which participants are connected by videoconference with one another and with an interdisciplinary team of autism experts. The expert team includes a pediatrician, a psychologist, a child and adolescent psychiatrist, a social worker, a dietician, and a parent of a child with autism. Each session includes a brief didactic presentation, case-based learning (during which PCPs present their own cases for discussion and co-management), and collaborative guided practice. The curriculum focuses on best-practice guidelines for diagnosis and treatment of ASD, with specific emphasis on symptom identification, screening tools, diagnostic interviewing, differential diagnosis, parent support, community resources, and management of co-occurring symptoms.

Participants in the program receive in-depth training in administration and interpretation of both Level 1 and Level 2 autism screening and assessment tools. Strategies for implementation of general developmental and autism-specific screening tools are discussed throughout the 12-month program, and participants also engage in quality improvement efforts to track their success in implementation of these tools in their practices. For example, Level 1 tools include the Ages and Stages Questionnaire, third Edition (ASQ-3); the Parents' Evaluation of Developmental Status: Developmental Milestones (PEDS-

DM); and the Modified Checklist for Autism in Toddlers, Revised (M-CHAT-R), with Follow-up Interview. Following the in-person training on the STAT (a Level 2 tool), participants are required to achieve reliability in STAT coding and administration (including submission of videotaped recordings of their own administrations). Participants also receive training on diagnostic interviewing for autism and on providing feedback and guidance to families. Written and online guidelines, resources, and toolkits are made available to PCPs through a secure online portal.

In addition to formal didactic presentations, a large portion of the teaching and mentorship provided during ECHO sessions occurs within the context of case-based learning. The majority of time within each ECHO Autism STAT session is dedicated to discussion of de-identified cases presented by PCPs using a standard case presentation format. During the first 6 months of the program, PCPs discuss cases for which this is a concern of autism (screening) or cases for which there is a concern about symptom management. The expert team and all participants have opportunities to ask clarifying questions to learn more about the case and the presenter's approach to date. Following this discussion, the expert team provides evidence-based recommendations and guidance to the PCP, building knowledge and expertise among all participants. PCPs may then re-present cases during later sessions to receive feedback and ongoing coaching. Over time, participants form a collaborative learning community in which expertise is shared and developed. In contrast to traditional telemedicine approaches, the ECHO model enables PCPs to retain responsibility for managing their own patients with guidance from the specialist team. Through this process, PCPs acquire knowledge, skills, and self-efficacy that is directly applicable to their own practice.

During the second 6 months of the ECHO Autism STAT program, PCPs who have achieved reliability on the STAT are eligible to present cases for diagnostic consideration. For these cases, PCPs follow a specific ECHO Autism STAT diagnostic algorithm to assess young children who are determined to be at risk for ASD following routine

surveillance and/or Level 1 screening. Rather than referring the child to a specialty center for diagnostic evaluation, PCPs administer the STAT and a semi-structured diagnostic interview for autism in their own practices. PCPs then present the results of the evaluation during an ECHO Autism STAT session. Cases are discussed with the expert team and all participants and are determined to either (1) clearly meet criteria of a diagnosis of ASD or (2) require further evaluation due to subtle or complex symptom presentation. This risk-stratified process for diagnostic determination facilitates rapid access to diagnosis and intervention for children at highest risk for autism while fostering access to more comprehensive assessments when warranted.

Future Directions

The results of the initial ECHO Autism STAT pilot study indicated improvements in PCP practice and self-efficacy and demonstrated that the model is feasible for enhancing local access to early diagnosis for children at greatest risk for ASD (Mazurek et al. 2019). Future directions for the evaluation of this program should include validation of PCP diagnostic determination as compared to comprehensive assessment with gold-standard autism diagnostic instruments. Additional work is needed to develop and validate a process for verification of PCP mastery, competence, and readiness to complete evaluations independently of the ECHO Autism STAT program. An examination of effectiveness of this model in reducing time to intervention, increasing access to care, and reducing cost and burden for families of children with ASD will also be important.

The ECHO Autism STAT program represents one approach for reducing healthcare disparities for underserved children with or at risk for ASD by increasing local diagnostic expertise. Future work should continue to explore novel and flexible models that leverage technology to infuse autism expertise into local communities. Although building capacity for early identification and diagnosis in primary care is one important avenue, additional large-scale efforts are also

needed to support community-based practitioners in caring for children with and at risk for ASD across settings and service systems.

See Also

- [Clinical Assessment](#)
- [Medical Education on Autism](#)
- [Screening Instruments for ASD](#)
- [Screening Measures](#)
- [Screening Tool for Autism in Two-Year-Olds \(STAT\)](#)

References and Reading

- Arora, S., Kalishman, S., Thornton, K., Dion, D., Murata, G., Deming, P., . . . Collieran, K. (2010). Expanding access to hepatitis C virus treatment – Extension for community healthcare outcomes (ECHO) project: Disruptive innovation in specialty care. *Hepatology*, 52(3), 1124–1133.
- Arora, S., Thornton, K., Murata, G., Deming, P., Kalishman, S., Dion, D., . . . Dunkelberg, J. (2011). Outcomes of treatment for hepatitis C virus infection by primary care providers. *New England Journal of Medicine*, 364(23), 2199–2207.
- Arunyanart, W., Fenick, A., Ukritchon, S., Imjaijit, W., Northrup, V., & Weitzman, C. (2012). Developmental and autism screening: A survey across six states. *Infants & Young Children*, 25(3), 175–187.
- Baio, J., Wiggins, L., Christensen, D. L., et al. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and Developmental Disabilities Monitoring Network, 11 sites, United States, 2014. *Morbidity and Mortality Weekly Report Surveillance Summaries*, 67(6), 1–23.
- Fenikilé, T. S., Ellerbeck, K., Filippi, M. K., & Daley, C. M. (2015). Barriers to autism screening in family medicine practice: A qualitative study. *Primary Health Care Research & Development*, 16(4), 356–366.
- Filipek, P. A., Accardo, P. J., Ashwal, S., Baranek, G. T., Cook, E. H., Jr, Dawson, G., . . . Volkmar, F. R. (2000). Practice parameter: Screening and diagnosis of autism: Report of the quality standards subcommittee of the American academy of neurology and the child neurology society. *Neurology*, 55(4), 468–479.
- Finke, E. H., Drager, K. D. R., & Ash, S. (2010). Pediatricians' perspectives on identification and diagnosis of autism spectrum disorders. *Journal of Early Childhood Research*, 8(3), 254–268.
- Fountain, C., King, M. D., & Bearman, P. S. (2011). Age of diagnosis for autism: Individual and community factors across 10 birth cohorts. *Journal of Epidemiology and Community Health*, 65(6), 503–510.
- Healthy People. (2020). Washington, DC: U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion. Retrieved from <https://www.healthypeople.gov/2020/topics-objectives/topic/maternal-infant-and-child-health/objectives>
- Interagency Autism Coordinating Committee (IACC). (2017). *2016–2017 interagency autism coordinating committee strategic plan for autism Spectrum disorder*. U.S. Department of Health and Human Services Interagency Autism Coordinating Committee. Retrieved from <https://iacc.hhs.gov/publications/strategic-plan/2017/>.
- Keil, A., Breunig, C., Fleischfresser, S., & Oftedahl, E. (2014). Promoting routine use of developmental and autism-specific screening tools by pediatric primary care clinicians. *WMJ*, 113(6), 227–231.
- MacDonald, R., Parry-Cruwys, D., Dupere, S., & Ahearn, W. (2014). Assessing progress and outcome of early intensive behavioral intervention for toddlers with autism. *Research in Developmental Disabilities*, 35(12), 3632–3644.
- Mandell, D. S., Listerud, J., Levy, S. E., & Pinto-Martin, J. A. (2002). Race differences in the age at diagnosis among Medicaid-eligible children with autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(12), 1447–1453.
- Mandell, D. S., Morales, K. H., Xie, M., Lawer, L. J., Stahmer, A. C., & Marcus, S. C. (2010). Age of diagnosis among Medicaid-enrolled children with autism, 2001–2004. *Psychiatric Services*, 61(8), 822–829.
- Mazurek, M. O., Brown, R., Curran, A., & Sohl, K. (2017). ECHO autism: A new model for training primary care providers in best-practice care for children with autism. *Clinical Pediatrics*, 56(3), 247–256.
- Mazurek, M. O., Curran, A., Burnette, C., & Sohl, K. (2019). ECHO autism STAT: Accelerating early access to autism diagnosis. *Journal of Autism and Developmental Disorders*, 49(1), 127–137.
- Myers, S. M., Johnson, C. P., & American Academy of Pediatrics Council on Children with Disabilities. (2007). Management of children with autism spectrum disorders. *Pediatrics*, 120(5), 1162–1182.
- Oswald, D. P., Haworth, S. M., Mackenzie, B. K., & Willis, J. H. (2017). Parental report of the diagnostic process and outcome: ASD compared with other developmental disabilities. *Focus on Autism and Other Developmental Disabilities*, 32(2), 152–160.
- Perry, A., Cummings, A., Geier, J. D., Freeman, N. L., Hughes, S., Managhan, T., . . . Williams, J. (2011). Predictors of outcome for children receiving intensive behavioral intervention in a large, community-based program. *Research in Autism Spectrum Disorders*, 5(1), 592–603.
- Rosenberg, R. E., Landa, R., Law, J. K., Stuart, E. A., & Law, P. A. (2011). Factors affecting age at initial autism spectrum disorder diagnosis in a national survey. *Autism Research and Treatment*. <https://doi.org/10.1155/2011/874619>.

- Self, T. L., Parham, D. F., & Rajagopalan, J. (2015). Autism spectrum disorder early screening practices: A survey of physicians. *Communication Disorders Quarterly*, 36(4), 195–207.
- Sheldrick, R. C., Maye, M. P., & Carter, A. S. (2017). Age at first identification of autism spectrum disorder: An analysis of two US surveys. *Journal of the American Academy of Child and Adolescent Psychiatry*, 56(4), 313–320.
- Smith, T., Klorman, R., & Mruzek, D. W. (2015). Predicting outcome of community-based early intensive behavioral intervention for children with autism. *Journal of Abnormal Child Psychology*, 43(7), 1271–1282.
- Stone, W. L., Coonrod, E. E., & Ousley, O. Y. (2000). Brief report: Screening tool for autism in two-year-olds (STAT): Development and preliminary data. *Journal of Autism and Developmental Disorders*, 30(6), 607–612.
- Vivanti, G., Dissanayake, C., & Victorian ASELCC Team. (2016). Outcome for children receiving the early start Denver model before and after 48 months. *Journal of Autism and Developmental Disorders*, 46(7), 2441–2449.

Echolalia

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Synonyms

[Delayed echolalia](#); [Imitative speech](#); [Immediate echolalia](#); [Scripting](#)

Definition

Echolalia, either immediate or delayed, is the repetition of sounds, words, phrases, or larger chunks of language. It is a repetitive pattern of language that does not necessarily contain meaning, nor is it directed to others for a specific purpose. Echolalia can be seen in individuals with various developmental disorders, including autism, Tourette's syndrome, aphasia, and schizophrenia.

Typical children who are developing language may demonstrate echoed speech between the ages

of 12–30 months of age. Lovaas (1981) reported that in typically developing children, echolalia peaks at approximately 30 months. This pattern tends to fade as a child's vocabulary and variety of language forms develop. For children with autism, however, the pattern can persist beyond 30 months and their repetitive talk may often be related to areas of special interest, media, or other environmental factors. Children may echo language used by people in their environment, or language heard within TV or songs.

Research (Prizant and Duchan 1981) has shown that children and adults with autism may use echolalia for a variety of communicative purposes, such as a way to process language and improve their comprehension, as a means to initiate a conversation, or to form a reply. Echolalia is also thought to be a way for some children to cope with and “tune out” stressful situations.

Immediate echolalia is the repetition of a word or phrase directly following its utterance by another. For example, a child may repeat a question mother has just asked (Mother: “Do you want juice?” Child replies, “Do you want juice?”). Delayed echolalia is the repetition of words and phrases heard at some previous time, for example, a child may repeat an announcement heard at school the previous day (“Today we will be having a fire drill”). As spontaneous language improves in children with autism, echolalia tends to decrease, as it does among typically developing children.

See Also

► [Movie Talk](#)

References and Reading

- Paul, R. (2007). Special considerations for special populations. In R. Paul (Ed.), *Language disorders from infancy to adolescence* (3rd ed., p. 138). St. Louis: Mosby Elsevier.
- Prizant, B., & Duchan, J. (1981). The functions of immediate echolalia in autistic children. *Journal of Speech and Hearing Disorders*, 46, 241–249.

Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 346–347). Hoboken: Wiley.

Ecological Model of Autism

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Ecological Inventory

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Definition

An approach to identify functional, age-appropriate curriculum content a student needs in specific environments within the following curriculum domains: personal management, community, vocational, leisure, and school. The process involves five phases: (1) identify the curriculum domains, (2) identify current and future natural environments within each curriculum domain, (3) identify the sub-environments within the natural environments, (4) inventory the relevant tasks within each sub-environment, and (5) analyze each task into component skills needed to perform the task from start to finish.

References and Reading

Brown, L., Branston, M. B., Hamre-Nietupski, S., Pumpian, I., Certo, N., & Gruenewald, L. (1979). A strategy for developing chronological age appropriate and functional curricular content for severely handicapped adolescents and young adults. *Journal of Special Education*, 13(1), 81–90.

Definition

The ecological model of autism studies the behavior of individuals with autism within the context of many levels of environmental influence and assumes bidirectional influences between (a) the person and these environmental influences and (b) the many environmental levels. Context refers to the wide range of system levels that influence individuals with autism, including the immediate responses of caregivers and family, school, local community, as well as broader cultural, economic, and political practices. Some variables have a direct immediate impact, and some more distal variables have an indirect impact. With the ecological model, the appropriate unit of study is the interaction of the environment and individual, the organism-environment system; “autism is not a static condition existing within a person, but a developmental process that can only be understood as taking place through the interaction of person and environment” (Loveland 2001, p. 22). An essential element of the ecological model of autism “is that every level of the ecological system is interconnected and thus can influence all the other subsystems. These influences are reciprocal rather than unidirectional” (Weiss et al. 2010, p. xxv). Ecological models attempt to explain not only the immediate impact of caretaker/client interaction on the behavior of the individual with autism but also the impact of social and economic policies and the impact of those variables on one another.

Ecological Model

► [Monotropism: An Interest-Based Account of Autism](#)

Historical Background

The ecological model of autism is a component of the broader interactional model that assumes many variables interconnect to produce an

outcome. The transactional model is one type of interactional model used to study development. The primary assumption is that development is the outcome of dynamic interactions between the individual and many layers of the environment. The context of the environment ranges from variables near the individual to those that are quite distant (Wicks-Nelson and Israel 2009). One type of transactional model is the ecological model. The comprehensive ecological systems theory emphasizes the importance of the context in understanding a person's behavior (Bronfenbrenner 1979; Gibson 1979). Early behavioral models emphasize contextual variables such as discriminative stimuli and setting events, but the ecological model of autism analyzes a broader range of environmental variables, from microsystems that include physical space, lighting, and noise in the teaching environment to broader cultural and historical events and the interactions between these variables.

Current Knowledge

The ecological approach to treatment of autism requires a multidisciplinary treatment team to tailor services to the specific needs of the individual and their caretakers, so treatment is not limited to the individual and their family. Intervention extends to any ecological or contextual variable that affects the individual with autism.

The ecological theory of autism may be conceptualized as a multidimensional series of concentric circles having the individual with autism in the middle (Zigler and Stevenson 1993). Many investigators consider the critical autistic feature the inability of the individual to understand what others think or feel, a weakness in theory of mind. Broader developmental characteristics include observable operant behavior (both desirable and undesirable), cognition, neurological conditions, emotional behavior and mood, social behavior, communication skills, motor behavior, medical conditions, and sensory deficits. Molecular behavior includes the form of the response, duration, latency between antecedent and behavior, the

time between responses, and the intensity of the response. The individual's personal goals, preferences, and strengths are important considerations. The ultimate criterion "emphasizes that assessment efforts and intervention objectives specifically target behavior that will be functional for the individual with a disability in real environments" (Powers 2005, p. 818).

The *microsystem* is the first level of the environment. This includes the minute-by-minute and day-to-day interactions between the individual with autism and caregivers (parents and teachers), peers, and siblings as well as the daily settings and physical structure of home, school, and community. Within these systems, it is critical to recognize the bidirectional effects of behavior; caretakers influence people with autism, but the stress of autistic behavior also influences caretakers, and stressful caretaker reactions can in turn affect the autistic behavior. For example, in response to unusual social and emotional behavior, caretakers of children with autism are more directive and may even smile less toward the children. Loveland (2001) contends the socially inappropriate behavior of individuals with autism is a product of both neurobiological impairment and the abnormal reactions of significant others to the unusual social behavior. Furthermore, the abnormal feedback of others may affect neurological development (e.g., synaptic pruning) in the young child whose behavior then becomes more atypical, with the cycle revolving. At this level, the ecological unit of analysis may also examine coercive processes (Binnendyk et al. 2009; Patterson 1982). Such analyses may evaluate whether inappropriate behavior is the outcome of positive reinforcement by attention, negative reinforcement by caretaker acquiescence, or exacerbated by repeated commands or reprimands for trivial behavior.

The determination of target behavior strengths and weaknesses selected for treatment is an important component, as practitioners must also consider whether the selected targets are meaningful to the family, day-to-day caretakers, and the community (Kazdin 1977). Behavior analysis of the microsystem would include operational

definitions of selected target behaviors, distal setting events, antecedents that immediately precede the behavior (discriminative stimuli), and consequences that immediately follow the behavior (reinforcers and punishers). Ecological validity, or ecological assessment (Powers 1997), identifies whether the selected prosocial target behaviors are functional in the person's day-to-day environment. For generalization of behavior across time and settings, selected target behaviors must be naturally functional (i.e., reinforced by the natural community) within the microsystem, and care is taken to identify obstacles in the natural environment that may punish the selected behavior.

The physical structure of home, school, and community takes into account characteristics of the general setting such as large crowds, noise, and predictable schedules versus chaotic routines that may influence autistic behavior. For example, in the context of challenges presented to individuals with autism in the form of schedules and routines, Binnendyk et al. (2009) conceptualize the essential unit of analysis as the quality of parent-child interactions embedded within routines of everyday life. The quality of family interactions within these routines may have a "profound" impact on the child's development. Family-selected routines that have served as the basis for routine-specific treatment plans include bathroom routines, restaurant routines, dining at home, dining at a restaurant and fast food establishments, grocery shopping, family television time, and bedtime.

Other units of analysis go beyond the behavior of the individual person and require myriad sources of data from larger social systems. The next level of the environment is the *mesosystem*, representing the interactions across the individual's microsystem. Variables that influence the individual with autism include parents' marital strain, extended family relationships, the reciprocal relationships between parents and teachers, parents and siblings, teacher and school, etc. For example, interpersonal conflict between parents and teachers or parents and siblings may have an effect on the behavior of the individual with

autism, and if this were determined to be the case, an ecological approach will consider that conflict a variable that needs to be addressed as part of treatment. Sometimes, the highly sophisticated practices of treatment workers contrast with parent training intellectually limited family members at home. If these contrasting styles prevent adequate application of services in the home setting, then an ecological approach will consider that disconnect a variable that needs to be addressed as part of treatment.

Contexts that influence the child indirectly compose the *exosystem* of the ecological system. The exosystem influences the child indirectly by effecting institutions and people in the individual's microsystem. For example, a parent may lose a job or be transferred to employment in another state. The stress of parental unemployment or moving to another state with concomitant schedule and routine changes may influence the behavior of the individual with autism, especially an individual whose repertoire includes resistance to change or well-established rituals. Structural changes within social service agencies that provide counseling and family support may lead to new treatment workers who are required to relearn details of the family's goals and objectives and establish new relationships, all of which can affect the individual with autism. Other exosystems that do not include the person with autism but can influence them include parent social networks, socioeconomic status of the family and neighborhood, the availability of mental health services in the community, the availability of professional training in empirically validated practices for service providers, the availability of materials and human resources required to meet the needs of the child (Lovaas 2003), and the availability of follow-up care and respite as needed.

In recognition of the growing diversity of the family unit, an ecological analysis also includes a subjective understanding of a family's values and beliefs. This allows treatment plans to consider culturally sensitive interventions when collaborating with families, sometimes referred to as cultural competence (Lynch and Hanson 2004).

The broadest level of influence composed of the culture's values, the economy, political trends, and social policy is the *macrosystem*. Resources for the family and school, tolerance of individual differences, and general opportunities afforded individuals with autism are greatly influenced by the macrosystem. State and federal law has mandated a number of recent social policy trends. In 1990, the US Congress added the word "autism" to laws that guarantee special education services. The Individuals with Disabilities Education Act, the federal law pertaining to special education, was reauthorized in 1997 (National Dissemination Center for Children with Disabilities), and both of these acts have resulted in more educational/treatment services to children with autism and more inclusion in less segregated (and less restrictive) general school settings. At the same time, economic conditions in the 2010s may lead to reduced local school budgets and reduced educational services.

Many states in the USA have passed laws requiring state-regulated group health plans to include coverage for autism, and federal law prohibits insurance companies from refusing to issue or renew coverage for children because of pre-existing conditions including autism. The outcome for families of children with autism includes monetary saving and less emotional stress, with children eligible for a wide array of services.

The chronosystem signifies time both within the lifetime of the individual of interest and the historical context within which the person lives (Weiss et al. 2010). Over the course of time, the child and family's growth and learning can affect each of the above-described systems. Furthermore, the historical context within which the person lives influences the services available to that person. Compared with individuals living prior to 1980, much has changed for individuals with autism. One critical difference is how the definition of autism presented in the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM, American Psychiatric Association [APA], 1980, 1994, 2000) has changed. Although Kanner identified autism in the 1940s, it was not until 1980 that

criteria for autism were defined in the DSM. Since then, the phrasing to describe autism has become far less restrictive. The 1980 DSM required six mandatory criteria with phrasing that limited the number of people diagnosed with autism (e.g., "A pervasive lack of responsiveness to other people" in the 1980 DSM is replaced by "Lack of spontaneous seeking to share... achievements with other people"). In conjunction with the 1994 inclusion of Asperger's disorder in the DSM, the acknowledgment that autism can exist among people across the entire range of intelligence, and recent efforts to take advantage of neural plasticity by identifying and providing comprehensive treatment among the youngest children with autism, the number of people on the autism spectrum eligible for services has expanded greatly. Perhaps three fourths of those diagnosed with autism are accounted for by those diagnosed with milder variants (Chakrabarti and Fombonne 2001), and only 35 years ago, many of these people would not have been eligible for services.

Ultimately, ecologically sensitive interventions synthesize data from multidisciplinary sources to develop a holistic intervention that addresses the individual with autism, their caretakers, the physical structure of important settings, and cultural variables.

See Also

- [Advocacy](#)
- [Classroom Structure](#)
- [Clinical Assessment](#)
- [Ecological Inventory](#)
- [Ecological Validity](#)
- [Family Therapy](#)
- [Functional Ecological Approach](#)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (3rd ed. Rev.). Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. Text Rev.). Washington, DC: Author.
- Binnendyk, L., Fossett, B., Cheremshynski, C., Lohrmann, S., Elkinson, L., & Miller, L. (2009). Toward an ecological unit of analysis in behavioral assessment and intervention with families of children with developmental disabilities. In W. Sailor, G. Dunlap, G. Sugai, & R. Horner (Eds.), *Handbook of positive behavior support*. New York: Springer.
- Bronfenbrenner, U. (1979). Contexts of childrearing: Problems and prospects. *American Psychologist*, 34, 844–850.
- Chakrabarti, S., & Fombonne, E. (2001). Pervasive developmental disorders in preschool children. *Journal of the American Medical Association*, 285, 3093–3099.
- Gibson, J. J. (1979). *The ecological approach to VISUAL perception*. Boston: Houghton-Mifflin.
- Kazdin, A. E. (1977). Assessing the clinical or applied importance of behavior change through social validation. *Behavior Modification*, 1, 427–451.
- Lovaas, O. I. (2003). *Teaching individuals with developmental delays*. Austin: Pro-Ed.
- Loveland, K. A. (2001). Toward an ecological theory of autism. In J. A. Burack, T. Charman, N. Yirmiya, & P. R. Zelazo (Eds.), *The development of autism: Perspectives from theory and research*. Mahwah: Lawrence Erlbaum.
- Lynch, E. W., & Hanson, M. J. (2004). *Developing cross-cultural competence: A guide for working with children and their families* (3rd ed.). Baltimore: Brookes.
- National Dissemination Center for Children with Disabilities. Retrieved from <http://nichcy.org/laws/idea>
- Patterson, G. R. (1982). *A social learning approach* (Coercive family process, Vol. 3). Eugene: Castalia.
- Powers, M. D. (1997). Behavioral assessment of autism. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (2nd ed., pp. 448–459). New York: Wiley.
- Powers, M. D. (2005). Behavioral assessment of individuals with autism: A functional ecological approach. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders, assessment, interventions, and policy* (Vol. 2, pp. 817–830). New York: Wiley.
- Weiss, H. B., Kreider, H., Lopez, M. E., & Chatman-Nelson, C. (2010). *Preparing educators to engage families: Case studies using an ecological systems framework*. Los Angeles: Sage.
- Wicks-Nelson, R., & Israel, A. C. (2009). *Abnormal child and adolescent psychology*. Upper Saddle River: Pearson/Prentice Hall.
- Zigler, E. F., & Stevenson, M. F. (1993). *Children in a changing world: Development and social issues*. Pacific Grove: Brookes/Cole.

Ecological Validity

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Synonyms

External validity

Definition

Ecological validity is the degree to which experimental measures (i.e., settings, treatment agents, materials, behaviors) occur naturally and are representative of events that occur in everyday life or in the settings in which the skill or behavior is expected to be performed (Brewer 2000; Schlosser 2003). The results of an ecologically valid assessment are predictive of appropriate behavior in a free environment (Franzen 2000). Ecological validity is important for descriptive or demonstrative research (Brewer 2000) and is an aspect of external validity that is closely related to, but not to be confused with, social validity (Schlosser 2003).

See Also

► External Validity

References and Reading

- Brewer, M. B. (2000). Research design and issues of validity. In H. T. Reis & C. M. Judd (Eds.), *Handbook of research methods in social and personality psychology* (pp. 3–16). Cambridge, UK: Cambridge University Press.
- Franzen, M. D. (2000). Validity as applied to neuropsychological assessment. In *Reliability and validity in neuropsychological assessment* (pp. 33–54). New York: Springer.

- Sbordone, R. J., & Long, C. J. (Eds.). (1996). *Ecological validity of neuropsychological testing*. Boca Raton: CRC Press.
- Schlosser, R. W. (2003). Validity. In R. W. Schlosser (Ed.), *The efficacy of augmentative and alternative communication* (pp. 27–41). San Diego: Academic Press.

ECT

- [Electroconvulsive Therapy \(ECT\)](#)

EDI

- [Emotion Dysregulation Inventory](#)

Edinburgh Handedness Inventory

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Synonyms

[EHI](#)

Description

The Edinburgh Handedness Inventory (Oldfield 1971) is a measurement scale which is employed to establish hand dominance. The 20-item inventory contains a list of instructions to be carried out by the individual being assessed. Items are rated by direct observation of the individual's behavior or by self-report of everyday behavior. Oldfield developed a second, shorter version, namely, a 10-item inventory, and it is this form which has been used most often.

This brief quantitative measure has proved to be of use in neuropsychological, general, clinical,

and research fields. However, where the question of establishing laterality is critical, such as in research, the author has reported that this scale used on its own is not sufficient to determine cerebral laterality and is effectively best employed as a screening measure. Given its simplicity and brevity, it would be particularly useful where large populations are being assessed. The measure benefits from properties which provide a useful standard of comparison in the neuropsychological field. The EHI is the most widely used instrument to measure handedness, and this appears to have increased in recent years (Fazio et al. 2011; Fazio and Cantor 2015). Furthermore, the measure has been translated into a number of languages, and the psychometric properties of translated versions have been established (Albayay et al. 2019; Espirito-Santo et al. 2017; Yang et al. 2018).

Oldfield (1971) strongly urges clinicians and researchers to follow the instructions closely in order to ensure limitation of misinterpretation and to ensure the form is completed correctly. However, Fazio et al. (2011) found that the original instructions were in fact problematic and only 47.3% of individuals in their study were able to follow the instructions completely. By contrast, 88.2% of these individuals could successfully follow the instructions on a different measure with Likert-style presentation. The authors note that the level of education and the handedness of the participants significantly predicted their ability to adhere to the instructions which had clear implications for the use of the Edinburgh Handedness Inventory.

Where individuals are being required to complete the form (self-reporting rather than being observed in a clinical setting), they are presented with a typed sheet and instructed to provide information regarding age and biological sex. The original inventory asks whether they have ever had a tendency to left-handedness.

Individuals are then presented with a list of ten items to score. They are instructed to consider their hand preference for each activity, indicating with one plus (+) if the preference is for the left or right hand. If they are *indifferent*, a plus is placed in both L and R columns. "Where the preference is so strong that you would never try to use the other

hand unless absolutely forced to, put ++” (Oldfield 1971). Since some tasks require two hands (e.g., striking a match), they are asked to indicate the hand preference for the key object, e.g., the match. All items are scored unless the individual has no experience of a task or object at all. A laterality quotient (LQ) is obtained by

$$(R - L)/(R + L) \times 100$$

where R = number of ticks in the *right* column and L = number of ticks in the *left* column.

Interpretation of results:

Left-handed <40

Ambidextrous 40 to +40

Right-handed > +40

While the core instrument has remained in use, various authors appear to have made adjustments to the instructions and presentation of the form. In addition, some authors have added further questions, e.g., holding a computer mouse (Cohen 2008; Dragovic 2004b), holding a cup for drinking, and using a key to unlock a door (Cohen 2008). Williams (1986) proposed a revision of the EHI resulting in changes to the number of items tested – reducing these from ten to seven – and consistent with Dragovic’s suggestion (2004b), adding the item computer mouse. He argues that two items (opening a box and using a broom) have been discarded since new factor-analytic studies found these items to be low outliers when loaded onto the “handedness” factor. The computer mouse is viewed as a significantly “unimanual activity in today’s world” (Williams 1986).

The EHI (revised) proposes the adoption of a Likert-style format, thus reducing the wording in the instructions to “Please mark the box that best describes which hand you use for the activity in question.” For each item, the respondent hence selects from five possible options, namely, *always left*, *usually left*, *no preference*, *usually right*, and *always right*. The procedure for arriving at the laterality quotient (LQ) is by necessity adjusted and can be used to identify those individuals who are mixed-handed (LQ between +50 and 50). Scoring is as follows: “- 50” *always left*, “- 25”

usually left, “0” *no preference*, “25” *usually right*, and “50” *always right*.

Scores on the eight rows are summed to provide a score LQ* of -400 to +400. If the LQ* score is then divided by 4, an LQ of -100 to +100 is achieved, i.e., complete left-handedness to complete right-handedness as per the original EHI. With the revised scoring method, -200 to +200 becomes mixed-handedness. Williams describes a clinical observation that the mixed-handedness group may further divide into two, namely, clumsy or coordinated. He anticipates that the clumsy group may have an LQ* score of -200 to -1 and the coordinated group an LQ* of 0–200.

Historical Background

When first published in 1971, the Edinburgh Handedness Inventory was initially named the Edinburgh Inventory (Oldfield). Previous assessment of handedness relied on either (a) observation of the individual carrying out tasks (seminovel) with left or right hands separately or (b) posing a number of questions about day-to-day activities and the hand use in these. In the former, the individual’s performance may be rated on time and/or error, enabling the calculation of an index of handedness.

The EHI has been used extensively in research (Boucher 1977; Dragovic 2004a, b; Cohen 2008; Edlin et al. 2015; Escalante-Mead et al. 2003; Fazio et al. 2011; Fazio and Cantor 2015; Williams 1991). Oldfield developed a 20-item and a 10-item version of the EHI. When the 10-item version was compared to Annett’s 12-item scale (1970), Williams found that, owing to the instructions, the EHI produced more “either hand” responses when usually the “left hand” would be the response (1991). He suggested that where neuropsychological researchers were looking to detect any degree of left-handedness, the questionnaire by Annett might be preferable.

While the EHI has remained a well-used instrument to establish handedness, it has been adapted by a number of authors, primarily adjusting the

presentation of the recording system and simplifying the instructions which have been deemed too complicated and potentially confusing for testees (Dragovic 2004b; Fazio et al. 2011; Cohen 2008). However, Edlin et al. (2015) conducted a review of 899 articles (1998 to 2012) citing use of the EHI and found highly variable usage of the measure and ad hoc adaptations rendering replication of research challenging.

Lateralization in autism has been of interest to scientists for many years, and the EHI has been employed in studies researching cerebral dominance and language development in individuals with autistic conditions (Boucher 1977; Froehlich et al. 2011; Williams 1986, 1991).

Psychometric Data

The Edinburgh Handedness Inventory has been refined since the original 20-item questionnaire in 1971. Oldfield discarded those items found to be redundant or inappropriate, given the purpose of devising a measure that would have universal application. He conducted a study of the 20-item inventory on 1,100 students. Analysis of items on the basis of biological sex, socioeconomic, and cultural factors resulted in ten items being discarded, leaving the inventory in its current form. On analysis of 1,027 responses (360 males and 667 females) where a score < 0 denotes left-handedness, 10% of males and 5.92% females scored < 0 (chi-squared = 6.21, $p < 0.02$). The authors noted the difficulties conducting statistical analyses on the data owing to the nature of the frequency distribution (a U form with the left side only containing about 10% of the total population). Treating the sections separately with the LQ cut at zero, percentage cumulative curves could be represented as the “right-handed” and “left-handed” sections, respectively. While the “right-handed” section could be represented in decile values, the “left-handed” section was less regular. The author notes that larger numbers will be needed in future studies to establish larger frequency values for the left-handed group. Hence, decile values remain provisional.

In a study of hand preference in the parents of autistic children, a similar pattern of results was found with 5.9% males and 0% females using the left hand more than the right (Boucher 1977). In Oldfield's student sample, 10% of those who predominantly used their right hand showed a very marked (LQ = 95–100) or almost exclusive preference. In Boucher's study, 69.1% of males and females who preferred the right hand used this almost exclusively. “Pure” right-handedness was significantly different between the groups at the $p < 0.001$ level on Guilford's test of the difference between two proportions. These unexpected results were nonetheless confounded by concerns regarding the reliability of the EHI and the possibility that some participants did not fully follow the instructions. Evaluation of handedness in the autistic children of these parents, on the other hand, indicated a persistent, but slightly increased tendency of autistic children to use the left hand.

McMeekan and Lishman's study (1975) compared the EHI (10-item) with the 12-item Annett questionnaire. The test-retest reliability results were unexpectedly moderate (0.80), but Williams notes (1991) a number of potentially significant methodological factors which most likely affected the results. On retesting with the EHI, participants often changed the classification from strong to weak. Williams does state that the EHI has one particular difficulty in the procedure employed for deriving the laterality quotient (LQ). Employing the equation, $(R - L) / (R + L)$, it is possible to achieve an LQ score of 100 in two ways: if there are 10–20 ticks in the R column, but none in the L column. In other words, there is no ability to discriminate between degrees of right-handedness. The issue of weighting of inventory items is also of some concern with no justification for equal weighting of some items and double weighting of items scored with two ticks. Williams (1991) notes that Annett's designation of “primary” and “secondary” items facilitates more sophisticated statistical discrimination.

Fazio and Cantor (2015) compared the factor structure of the EHI with the Fazio Laterality Inventory (Fazio et al. 2013). They found the FLI to provide a more accurate measurement of

handedness. When tested in a population of atypical handedness, factor analysis of the EHI demonstrated a single factor loading, while factor analysis of the FLI elicited two underlying factors (eigenvalues >1 ; 5.74 and 1.39). The authors describe the factors as fine motor/ballistic movements and expressive/instrumental movements. They argue that while both the FLI and EHI have good face and construct validity in typical handedness populations, the former is more helpful in measuring handedness in populations that are associated with atypical handedness.

Clinical Uses

The Edinburgh Handedness Inventory (Oldfield 1971) is used to assess hand dominance in everyday activities. The measure may be rated by an observer or by the individual's self-reporting. The author viewed the latter as less reliable since individuals tend to overestimate the number of tasks carried out using the dominant hand. Nonetheless, for ease of use, clinicians and researchers predominantly employ a questionnaire-based assessment of handedness over performance-based measures (Williams 1991). Such questionnaires have demonstrated themselves to be highly correlated with behavioral measures (Chapman and Chapman 1987).

Establishing hand preference may shed light on how the brain matures. Laterality has been demonstrated to be of importance in autism spectrum conditions, language, mathematical, and musical ability.

For instance, in a study of early language disturbance and lateral preference (on the Edinburgh Handedness Inventory), researchers found that autistic individuals who had early language disturbance displayed more atypical cerebral dominance than neurotypical controls and autistic individuals who had had normal language development (Escalante-Mead et al. 2003). Results indicated that there was not in fact a greater incidence of left-handedness in autism, but rather a disturbance in the developmental process of establishing lateral dominance. An atypical pattern or delay in establishing cerebral dominance

may have implications for language development in autism.

The Edinburgh Handedness Inventory remains a valuable tool in clinical neuropsychology. Additional measures may contribute to further understanding of handedness in populations known to be neurologically atypical.

References and Reading

- Albayay, J., Villarroel-Gruner, P., Bascour-Sandoval, C., Parma, V., & Galvez-Garcia, G. (2019). Psychometric properties of the Spanish version of the Edinburgh Handedness Inventory in a sample of Chilean undergraduates. *Brain & Cognition*, 137, 103618.
- Boucher, J. (1977). Hand preference in autistic children and their parents. *Journal of Autism and Childhood Schizophrenia*, 7(2), 177–187.
- Chapman, L. J., & Chapman, J. P. (1987). The measurement of handedness. *Brain and Cognition*, 6, 175–183.
- Cohen, M. S. (2008). Handedness questionnaire. Retrieved from www.brainmapping.org
- Dragovic, M. (2004a). Categorization and validation of handedness using latent class analysis. *Acta Neuropsychiatrica*, 16, 212–218.
- Dragovic, M. (2004b). Towards an improved measure of the Edinburgh handedness inventory: A one factor congeneric measurement model using confirmatory factor analysis. *Laterality*, 9, 411–419.
- Edlin, J. M., Leppanen, M. L., Fain, R. J., Hacklander, R. P., Hanaver-Torrez, S. D., & Lyle, K. B. (2015). On the use (and misuse?) of the Edinburgh Handedness Inventory. *Brain & Cognition*, 94, 44–51.
- Escalante-Mead, P. R., Minshew, N. J., & Sweeney, J. A. (2003). Abnormal brain lateralization in high-functioning autism. *Journal of Autism and Developmental Disorders*, 33(5), 539–543.
- Espirito-Santo, H., Pires, C. F., Garcia, I. Q., Daniel, F., Silva, A. G., & Fazio, R. L. (2017). Preliminary validation of the Portuguese Edinburgh Handedness Inventory in an adult sample. *Applied Neuropsychology. Adult*, 24, 275–287.
- Fazio, R. L., & Cantor, J. M. (2015). Factor structure of the Edinburgh Handedness Inventory versus the Fazio Laterality Inventory in a population with established atypical handedness. *Applied Neuropsychology. Adult*, 22, 156–160.
- Fazio, R., Coenen, C., & Denney, R. L. (2011). The original instructions for the Edinburgh Handedness Inventory are misunderstood by a majority of participants. *Laterality: Asymmetries of Body, Brain and Cognition*, 9, 1–8.
- Fazio, R., Dunham, K. J., Griswold, S., & Denney, R. L. (2013). An improved measure of handedness: The Fazio laterality inventory. *Applied Neuropsychology. Adult*, 20(3), 197–202.

- Frøehlich A., Anderson, J. S., Lange N., Zielinski, B. A., Dubray M. B., Nielsen J. A., et al. (2011). *Relationship between handedness and language lateralization in autism*. International Meeting for Autism Research, San Diego.
- McMeekan, E. R. L., & Lishman, W. A. (1975). Retest reliabilities and inter-relationship of the Annett hand preference questionnaire and the Edinburgh Handedness Inventory. *British Journal of Psychology*, 66, 53–59.
- Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia*, 9, 97–113.
- Soper, H. V., Satz, P., Orsini, D. L., Rolando, R. H., Zvi, J. C., & Schulman, M. (1986). Handedness patterns in autism suggest subtypes. *Journal of Autism and Developmental Disorders*, 16(2), 155–167.
- Williams, S. M. (1986). Factor analysis of the Edinburgh handedness inventory. *Cortex*, 22, 325–326.
- Williams, S. M. (1991). Handedness inventories: Edinburgh versus Annett. *Neuropsychology*, 5(1), 43–48.
- Yang, N., Waddington, G., Adams, R., & Han, J. (2018). Translation, cultural adaption, and test-retest reliability of Chinese versions of the Edinburgh Handedness Inventory and Waterloo Footedness Questionnaire. *Laterality*, 23, 255–273.

proper support. To ensure that children with ASD have access to this support, federal legislation requires that public schools provide eligible children with ASD an education that is adapted to meet their individual needs (Steedman 2007). Since students with ASD have different strengths and deficits, their educational programs may look very different. Researchers and professionals have used research, legislation, and professional experience to develop ASD program quality indicators (Alquraini 2011; Crimmins et al. 2001; Ruble et al. 2010). These indicators include comprehensive student assessment, ongoing progress monitoring, assessment-based and detailed individualized education plans (IEPs), individualized and appropriate curricula and instructional methods and activities, procedures for assessing and replacing challenging behaviors, qualified and appropriate school personnel, and high levels of family involvement (please see the section “Current Knowledge” for more information).

Education

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Definition

Autism spectrum disorder (ASD) is characterized by communicative, social, and behavioral impairments. Specifically, the two major categories of deficits are in social communication and repetitive behaviors (American Psychiatric Association 2013). These issues can make it difficult for children with ASD to succeed in traditional educational settings. However, many children can progress and even excel with the

Historical Background

In the past, students with ASD and other disabilities were often denied access to a public education. It was not until the 1960s and 1970s that the states and Congress passed legislation intended to protect children with disabilities right to a free and appropriate public education (Yell et al. 1998). Unfortunately, many children with disabilities continued to receive none or inappropriate educational services after these legislative changes (Steedman 2007).

In an effort to better serve children with disabilities, the federal government enacted the Education for All Handicapped Children Act of 1975 (EAHCA; Yell et al. 1998). This act was designed to provide all children with disabilities more extensive educational rights. In 1990, this act was amended and renamed the Individuals with Disabilities Education Act (IDEA). IDEA continues to undergo revisions designed to better protect these rights and facilitate the success of children with disabilities. It was most recently amended in 2004, with final regulations published in 2006 and 2011 (US Department of Education

2017). IDEA 2004 promises all children an appropriate education, but there continues to be debate concerning the extent and quality of services that schools are required to supply. Further, many schools report that they lack the funds and resources required to provide students with appropriate services (Block and Block 2007; please see the section “[Current Knowledge](#)” for more information).

Several Supreme Court cases have also been instrumental in further clarifying and adapting elements of IDEA 2004. One Supreme Court ruling in particular, *Winkelman v. Panama School District* (2007), has been identified as one of the most important Supreme Court rulings impacting the United States’ special education system (Yell et al. 2009). In their ruling of this case, the Supreme Court expanded the definition of free and appropriate public education (FAPE) under the IDEA 2004 to allow parents the right to be involved in all aspects of their child’s education, including the IEP process (Yell et al. 2009).

Current Knowledge

The Individuals with Disabilities Education Act (IDEA) of 2004 requires school districts to provide eligible individuals with ASD and other disabilities ages 3–21 with a free and appropriate education. This education should be designed around the students’ individual strengths and deficits. Each student’s program should include the special education and other services the student needs to make progress and should be delivered in the least restrictive environment possible. The program should target the skills that the student needs to achieve independent functioning, continue his or her education, and succeed in the workforce. Each of these main components is briefly described below.

Individualized and appropriate education. Although children with ASD share common areas of deficit, there is considerable variation in individual students’ abilities and impairments. Because of this heterogeneity, no single educational program will benefit all students with

ASD. IDEA recognizes this and requires that each student receive an education designed to build upon his or her strengths and address his or her deficits. Each student’s educational program must be detailed in an individual education plan (IEP). This plan should be developed by a multidisciplinary team including school personnel, relevant professionals (e.g., an ASD specialist, speech and language pathologist, or occupational therapist), and the student’s parents. The IEP should describe the student’s current abilities, his or her goals for the next year, and the specific services and accommodations that the child will receive to facilitate his or her progress. The IEP team must meet to review and update the IEP at least once a year. Parents or school staff can request additional IEP meetings any time that they feel the plan is not meeting the student’s needs.

Special education and other services. IDEA requires that eligible students with ASD receive the special education and related services that they need to benefit from their educational program. Special education refers to any curricular or instructional modifications that the child needs to progress. Related services refer to any additional support that will help the child to succeed. For children with ASD, speech therapy, occupational therapy, and transportation to and from school are common related services (Steedman 2007). When developing these special education and other services, the schools are required to use evidence-based practices (Odom et al. 2010). In other words, the school should use instructional practices or interventions that have received empirical support. Using educational strategies that have been found to benefit at least some children with ASD is believed to increase the likelihood that this intervention will benefit the current student with ASD (Morris and Mather 2008). Considerable debate remains over the extent and quality of services that schools are required to provide. The Supreme Court ruled that schools may not deny a child services based on the cost. However, the Supreme Court has also declared that schools do not have to offer children with disabilities the *best* education possible. Instead, services must allow children to “benefit” from their education (Steedman 2007).

In addition, the State Education Agencies' definition and corresponding evaluation of ASD has become problematic in recent years (Pennington et al. 2014). These definitions have differed by state, impacting which individuals qualify for special education and other services. Pennington et al. (2014) suggests that state and federal policy changes should be made to improve the definition of and the evaluation procedures used to assess ASD as an educational disability.

Least restrictive environment. IDEA states that each student's educational program should be implemented in the least restrictive environment possible (Alquraini 2011). Whenever possible, the student's special education and other services should be delivered in general education classrooms with typically developing peers. However, the IEP team must determine which type of placement best facilitates the academic, social, and behavioral progress of the student. Placement options include inclusion, special education, and mainstreaming. Inclusion is the least restrictive environment possible. Inclusion is generally defined as placement in a general education classroom with neurotypical peers, while providing additional services as needed (Alquraini 2013). These services can be provided within the general education classroom (i.e., push-in services) or outside of the classroom (i.e., pull-out services). Advocates of inclusion argue that this environment provides students with ASD more rigorous academic instruction and increased access to appropriate peer models and interactions. However, others contend that general education classrooms do not meet the special learning needs of students with ASD. Placement in a special education classroom is considered a more restrictive environment. Special education classrooms are populated by students with disabilities. They tend to contain fewer students and have a higher adult to child ratio. Teachers typically tailor their instruction to the special needs of their students. These classrooms may provide a more supportive environment for the students, but are often believed to be less academically rigorous and provide fewer opportunities for typical peer models and interactions. Students with ASD may also be mainstreamed, spending part of their day

in a special education classroom and part of their day in a general education classroom (Mesibov and Shea 1996). This placement allows children to receive more individualized special education instruction while also exposing them to typical curricula and peer models. However, the desired benefits to simply exposing children with ASD to neurotypical peers is unclear.

Program Quality Indicators

As previously noted, it is not possible to develop a single "best" program for students with ASD. However, researchers and professionals have used existing research and their professional experience to develop a number of program quality indicators or components of effective programs. Several of these components are described below; however, this list is not exhaustive. For full lists and comprehensive descriptions of program quality indicators, please see Colorado's Autism Program Quality Indicators (Colorado Department of Education 2016) and the cited references. The National Autism Center (2009, 2015) also provides information on the quality of ASD programs currently being implemented in schools.

Child assessment. Assessments allow educators to identify the strengths and deficits of children with ASD. Assessments should be comprehensive, with measures of the child's social and language skills, academic skills, adaptive and maladaptive behaviors, and cognitive abilities across settings. The selected assessments should provide information that can be used to inform the student's educational program (Crimmins et al. 2001).

Individualized education programs. The IEP should state the student's current level of functioning across communicative, social, academic, and developmental domains in school, home, and community settings. This information should be used to formulate new, measurable goals that will facilitate the child's success and independence across skill domains and environments. The IEP should specify the special education and related services that will be used to promote student progress (Steedman 2007). These may include parent services or training that will help the parents facilitate child progress in home and community

settings. These services and accommodations should be detailed so that all parties (e.g., parents, school staff) understand their roles and responsibilities (New Jersey State Department of Education 2004). For added clarity, Ruble et al. (2010) developed an evaluation tool to assess whether IEP's are consistent with the requirements put forth by IDEA and the National Research Council for children with ASD.

Progress monitoring. Effective programs should include ongoing and systematic measurement of individual student progress towards set IEP goals. The information gained from progress monitoring should be used to make appropriate educational decisions. When a student meets a set goal, the educational team should develop new goals to further facilitate child progress. When a student does not progress towards meeting a goal, the educational team should consider adjusting the method or intensity of the student's current program to better meet his or her needs (Crimmins et al. 2001; New Jersey State Department of Education 2004). Precision teaching is one approach to measuring a student's progress (Please see the ► [“Precision Teaching”](#) chapter for more information).

Curriculum. Each student's educational program should focus on the skills that he or she needs to achieve independent functioning, continue his or her education, and succeed in the workforce. Therefore, educational programs should address a wide range of skills, including communication and social skills, adaptive skills, academic skills, and, for older students, vocational skills (Please see the ► [“Curriculum”](#) chapter for more information). The specific content of each student's educational program should be based on his or her current functioning level and be adjusted as needed. Whenever appropriate, the curriculum should integrate age-appropriate state standards (New Jersey State Department of Education 2004; Noland et al. 2007).

Instructional methods and activities. Whenever possible, instructional methods and activities should be research-based. Methods and activities should be adapted to the strengths of the child with ASD. Skills should be targeted within appropriate instructional formats (e.g., individual,

small-group, or large-group instruction). When appropriate, skills should be targeted via naturally occurring opportunities and reinforcers. Programs should include components designed to promote independent skill use, generalization across people and settings, and maintenance (Crimmins et al. 2001; New Jersey State Department of Education 2004).

Challenging behaviors. Children with ASD often demonstrate stereotypic, disruptive, or self-injurious behaviors (Lopez et al. 2007). Programs should have a process for systematically assessing the function of these behaviors (e.g., functional assessments). This information should be used to develop a plan to eliminate the challenging behaviors and replace them with more adaptive behaviors (Crimmins et al. 2001; New Jersey State Department of Education 2004). A focus in recent years has involved implementing more positive behavioral interventions and services (PBIS) in schools, in an effort to reduce students' challenging behaviors (Kauffman 2015).

School personnel. School staff (e.g., teachers, other service providers, paraprofessionals) should be educated on the needs and strengths of students with ASD, effective educational practices, methods of individualizing educational programs to meet each student's needs, and behavior management and naturalistic teaching strategies. All staff should attend professional development workshops. Paraprofessionals, who tend to have less knowledge and experience, should receive continuous supervision and instruction from classroom teachers and other personnel. The program should have enough personnel (teachers, aides, therapists, etc.) to meet the needs of the students. Personnel should be familiar with each student's IEP and be aware of their role in its implementation (Crimmins et al. 2001; New Jersey State Department of Education 2004; Noland et al. 2007).

Family involvement. Legally, schools are required to include parents in the development of the student's educational program (Yell et al. 2009). Involving parents and family members in the development, implementation, and revision of the student's program is believed to increase program effectiveness. Therefore, the school should

make an effort to inform or refer parents to information on available services and their student's program and progress. Parents should also be given support and training that allows them to better meet their student's needs in home and community settings. Student's programs should be developed with consideration of the family's beliefs and values (Crimmins et al. 2001; New Jersey State Department of Education 2004).

Sample Schedule and Activities

A sample special education classroom schedule, activity descriptions, and recommendations are provided below. The activities can be adjusted to meet the individual needs of each student.

Consulting activity schedules. Activity schedules are a great way to prepare the student for the day's activities and transitions and to increase independence (Koyama and Wang 2011). Using visual activity schedules with students on the spectrum is considered an evidence-based practice (Knight et al. 2015). Each classroom should have a clearly visible general schedule, and each student should have an individual schedule detailing his or her specific activities for the day. Depending on the student's level of functioning, a schedule could be a written list or involve pictures.

Opening circle. Opening circle is a good time to target communication skills and attending in a large-group setting. Opening circle activities may include calendar time, singing songs, or sharing about a given topic (e.g., what students did over the weekend or favorite ice cream flavor). This type of activity can be implemented with young children with ASD during the preschool and elementary school years. It is not commonly implemented in middle or high school settings.

Individual work time. Individual work time should focus on each student's IEP goals. The following steps should be taken to maximize on-task behavior and correct responding.

- Structuring the work environment. Individuals with ASD benefit from a structured work environment (Bennett et al. 2011). Structuring the

work environment may include a work area with minimal distractions, clearly presenting the demand, visual instructions, and immediately reinforcing a correct response with praise, tokens, or tangible reinforcers (Ganz 2007).

- Brief stimulus assessment task. To ensure that each student is motivated, a brief stimulus assessment should be conducted at the beginning of each work session to identify items that can be used as reinforcers. In a brief stimulus assessment task, students are presented with a number of potential reinforcers, and the first items they choose are considered the preferred items. These items should then be used during the following work session (Paramore and Higbee 2005).
- Varying maintenance and acquisition tasks. During individual work time, the one-to-one aide should present the student with a mix of maintenance and acquisition tasks (Benavides and Poulson 2009; Charlop et al. 1992). For example, if a student is learning expressive language and categories but has already mastered receptive body parts, a work session should include trials on all three tasks.
- Using an appropriate level of prompting. As students acquire a given task, they will need different levels of prompting or support. Teachers and aides should give the student the minimal prompt necessary to help them succeed at the given task.
- Using a variety of preferred reinforcers. To make sure that the student stays motivated and does not grow bored with their reinforcer, the student should be reinforced with different preferred items throughout the session (Milo et al. 2010; Polín and Pérez 2017).
- Incorporating naturalistic teaching opportunities. The classroom provides a number of opportunities for naturalistic teaching strategies, such as incidental learning. Preferred toys may be placed on high shelves or in clear, hard to open bins so that the student must ask for them. Using naturalistic teaching strategies in the classroom promotes generalization and spontaneous use (Charlop 2017).

Lunch time. Meals are ideal times to provide natural learning opportunities. For example, instead of automatically providing a student with his or her food, the teacher or aide may wait for the student to request that the food be passed to them.

Small-group time. Small-group time is the perfect time to practice communication and social skills in a fun, motivating environment. Teachers or classroom aides can facilitate peer interactions during a variety of desired activities. These activities may include board or card games, arts and crafts, or conversations about preferred topics.

Physical education/outside play. Physical activity has been shown to decrease stereotypic behavior in individuals with ASD (Petrus et al. 2008). There is also research evidence supporting the assertion that for this population, exercise can help increase on-task behavior during subsequent academic instruction (Mahar et al. 2006). In addition, having students with ASD participate in small-group or team games provides opportunities to facilitate communicative and social interactions. During outside play, students may participate in activities such as kickball, tag, or relay races (Miltenberger and Charlop 2014).

Closing circle. Closing circle is a good time to practice communication skills and attending in a large-group setting. Closing circle activities may include reviewing the day's activities, asking students to share their favorite part of the day, or having students share a favorite item from home with the rest of the group.

Token systems. Tokens can be used to reinforce correct responding and appropriate behavior throughout the day (Tarbox et al. 2006). Using objects of obsession (e.g., a favorite TV character) as the tokens may be especially effective (Carnett et al. 2014; Charlop-Christy and Haymes 1998). At the end of the day, students should be given the opportunity to spend any tokens they have earned on preferred items. If students choose, they should be allowed to save their tokens until they have earned enough for a bigger item.

Future Directions

Over the past several decades, there have been tremendous gains in the education of students with ASD (Parsons et al. 2011; Yell et al. 2009). However, the education of children with ASD is far from perfect. While IDEA guarantees children with ASD and other disabilities an appropriate education, its implementation is often less than ideal (Block and Block 2007; Pennington et al. 2014). Schools report that they lack the funds and resources required to meet the extensive and diverse needs of the increasingly large population of students with ASD and other disabilities (Block and Block 2007). In addition, some students in need of these services may not qualify for them because of the vagueness of the State Education Agencies' definitions of ASD and the variability with how it is evaluated in different states (Pennington et al. 2014).

Two ways to address these issues are to provide research to develop the highest quality definition of and evaluation procedures used to assess ASD as an educational disability in schools and to continue to research effective educational practices for children with ASD. IDEA recognizes the importance of research-based practices (Steedman 2007), but the necessary body of research is yet to develop. Although many strategies have been shown to be effective when working with children with ASD in tightly controlled settings, much less is known about the outcomes of these strategies when implemented at school. There is a particular need for research addressing effective educational interventions for older and more impaired students with ASD (Noland et al. 2007). Researching interventions and identifying effective practices will provide educational teams with information that can be used to develop efficient and effective educational programs (Morris and Mather 2008).

See Also

- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Special Education](#)

References and Reading

- Alquraini, T. A. (2011). An analysis of legal issues relating to the least restrictive environment standards. *Journal of Research in Special Education Needs*, 13, 152–158.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- Benavides, C. A., & Poulson, C. L. (2009). Task interspersal and performance of matching tasks by preschoolers with ASD. *Research in Autism Spectrum Disorders*, 3, 619–629.
- Bennett, K., Reichow, B., & Wolery, M. (2011). Effects of structured teaching on the behavior of young children with disabilities. *Focus on Autism and Other Developmental Disorders*, 26, 143–152.
- Block, A. W., & Block, S. R. (2007). Family resources during the school-age years. In R. L. Gabriel & D. E. Hill (Eds.), *Growing up with autism* (pp. 164–182). New York: The Guilford Press.
- Carnett, A., Raulston, T., Lang, R., Tostanoski, A., Lee, A., Sigafos, J., & Machalicek, W. (2014). Effects of a perseverative interest-based token economy on challenging and on-task behavior in a child with autism. *Journal of Behavioral Education*, 23, 368–377.
- Charlop, M.H. (2017). *Naturalistic teaching strategies focusing on incidental teaching with children with autism spectrum disorder*. Austin: Pro-ed.
- Charlop, M. H., Kurtz, P. F., & Milstein, J. P. (1992). Too much reinforcement, too little behavior: Assessing task interspersal procedures in conjunction with different reinforcement schedules with autistic children. *Journal of Applied Behavioral Analysis*, 25, 795–808.
- Charlop-Christy, M. H., & Haymes, L. K. (1998). Using objects as tokens reinforcers for children with autism. *Journal of Autism and Developmental Disorders*, 28, 189–198.
- Colorado Department of Education. (2016). *Autism program quality indicators*. Retrieved from www.cde.state.co.us/cdesped/autismqualityindicators.
- Crimmins, D. B., Durand, V. M., Theurer-Kaufman, K., & Everett, J. (2001). *Autism program quality indicators: A self-review and quality improvement guide for schools and programs serving students with autism spectrum disorders*. Retrieved 12 May 2011 from <http://www.p12.nysed.gov/specialed/autism/apqi.htm>
- Ganz, J. B. (2007). Classroom structuring methods and strategies for children and youth with autism spectrum disorders. *Exceptionality*, 15, 249–260.
- Kauffman, J. M. (2015). Opinion on recent developments and the future of special education. *Remedial and Special Education*, 36, 9–13.
- Knight, V., Sartini, E., & Spriggs, A. D. (2015). Evaluating visual activity schedules as evidence-based practice for individuals with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45, 157–178.
- Koyama, T., & Wang, H.-T. (2011). Use of activity schedule to promote independent performance of individuals with autism and other intellectual disabilities: A review. *Research in Developmental Disabilities*, 32, 2235–2242.
- Lopez, B. R., Hill, D. E., Shaw, S., & Gabriels, R. L. (2007). School consultation and interventions for middle school and high school students with autism. In R. L. Gabriel & D. E. Hill (Eds.), *Growing up with autism* (pp. 247–271). New York: The Guilford Press.
- Mahar, M. T., Murphy, S. K., Rowe, D. A., Golden, J., Shields, A. T., & Raedeker, T. D. (2006). Effects of a classroom-based program on physical activity and on-task behavior. *Medicine & Science in Sports & Exercise*, 38, 2086–2094.
- Mesibov, G. B., & Shea, V. (1996). Full inclusion of students with autism. *Journal of Autism and Developmental Disorders*, 26(3), 337–346.
- Milo, J.-S., Mace, F. C., & Nevin, J. A. (2010). The effects of constant versus varied reinforcers on preference and resistance to change. *Journal of the Experimental Analysis of Behavior*, 93, 385–394.
- Miltenberger, C. A., & Charlop, M. H. (2014). Increasing the athletic group play of children with autism. *Journal of Autism and Developmental Disorders*, 44, 41–54.
- Morris, R. J., & Mather, N. (2008). *Evidence-based interventions for students with learning and behavioral challenges*. New York: Routledge.
- National Autism Center (2009, 2015). National Standards Report. Randolph: National Autism Center.
- Noland, R., Cason, N., & Lincoln, A. (2007). Building a foundation for successful school transitions and educational placement. In R. L. Gabriel & D. E. Hill (Eds.), *Growing up with autism* (pp. 205–227). New York: The Guilford Press.
- Odom, S. L., Collet-Klingenberg, L., Rogers, S. J., & Hatton, D. D. (2010). Evidence-based practices in interventions for children and youth with autism spectrum disorders. *Preventing School Failure*, 54, 275–282.
- Paramore, N. W., & Higbee, T. S. (2005). An evaluation of a brief multiple-stimulus preference assessment with adolescents with emotional-behavioral disorders in an educational setting. *Journal of Applied Behavior Analysis*, 38, 399–403.
- Parsons, S., Guldberg, K., MacLeod, A., Jones, G., Prunty, A., & Balfe, T. (2011). International review of the evidence on best practice in educational provision for children on the autism spectrum. *European Journal of Special Needs Education*, 26, 47–63.
- Pennington, M. L., Cullinan, D., & Southern, L. B. (2014). Defining autism: Variability in state education agency definitions of and evaluations for autism spectrum disorder. *ASD Research and Treatment*, 2014, 1–8.
- Petrus, C., Adamson, S. R., Block, L., Einarson, S. J., Sharifnejad, M., & Harris, S. R. (2008). Effects of exercise interventions on stereotypic behaviors in children with autism spectrum disorder. *Physiotherapy Canada*, 60, 134–145.
- Polin, E., & Pérez, V. (2017). The effect of varied reinforcement on acquisition and extinction speed. *Psicothema*, 29, 83–90.
- Roane, H. S., Vollmer, T. R., Ringdahl, J. E., & Marcus, B. A. (1998). Evaluation of brief stimulus

- assessment. *Journal of Applied Behavior Analysis*, 31, 605–620.
- Ruble, L. A., McGrew, J., Dalrymple, N., & Jung, L. A. (2010). Examining the quality of IEPs for young children with autism. *Journal of Autism and Developmental Disorders*, 40, 1459–1470.
- Steedman, W. (2007). Advocating for services: Legal issues confronting parents and guardians. In R. L. Gabriel & D. E. Hill (Eds.), *Growing up with Autism* (pp. 145–163). New York: The Guilford Press.
- Tarbox, R. S., Ghezzi, P. M., & Wilson, G. (2006). The effects of token reinforcement on attending in a young child with autism. *Behavioral Interventions*, 21(3), 155–164.
- US Department of Education. (2017). *Building the legacy: IDEA 2004*. Retrieved from <http://idea.ed.gov/>.
- Yell, M. L., Rogers, D., & Rogers, E. L. (1998). The legal history of special education: What a long, strange trip it's been! *Remedial and Special Education*, 19, 219–228.
- Yell, M. L., Ryan, J. B., Rozalski, M. E., & Katsiyannis, A. (2009). The U.S. supreme court and special education: 2005 to 2007. *Teaching Exceptional Children*, 41, 68–75.

It was the first law to mandate special education in all states. It has been reauthorized several times, most recently in 2004 as the Individuals with Disabilities Education Act (IDEA).

See Also

- [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- IDEA Partnerships. www.fape.org
- Nation Dissemination Center for Children with Disabilities. www.nichcy.org
- Turnbull, A., Wehmeyer, M. L., & Turnbull, R. (2009). *Exceptional lives: Special education in today's schools* (pp. 8–21). Prentice-Hall.
- U.S. Department of Education, Office of Special Education Programs. www.ed.gov/ose
- Vaughn, S., Bos, C., & Schumm, J. S. (2010). *Teaching exceptional, diverse, and at-risk students* (pp. 2–17). Prentice-Hall.

Education as Therapy

- [Educational Therapy](#)

Education for All Handicapped Children Act of 1975 (PL94-14 L)

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Synonyms

[PL94-142](#)

Definition

P.L.94-142, passed in 1975, was the public law that ensured that all children with disabilities would receive a free and appropriate education.

Educational Assistant

- [Para-educator](#)

Educational Interventions

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Definition

The role of education for all children is to provide the experiences needed to learn skills that will lead to “personal independence and social responsibility” (National Research Council [NRC], 2001, p. 40). Educational interventions provide students with the support needed to acquire the skills being taught by the educational system and

should address functional skills, academic, cognitive, behavioral, and social skills that directly affect the child's ability access an education. For students with autism spectrum disorders, the interventions often address some of the core deficits in the areas of communication, social skills, and behavioral differences. The interventions should be aimed at skills that need to be acquired or that need to be performed more often to lead to successful results. For children with autism, these educational interventions must be specific and targeted to address the deficits and lead to generalization and maintenance.

Educational interventions vary widely depending on the needs and age of the student, resources available, background of the team members, philosophy of the program, parental requests and beliefs, and student progress. In addition, the interventions used are based on the child's profile which should include thorough assessments and reports detailing strengths, weaknesses, learning style, preferences, past successes, etc., and should lead to the most successful outcome. Data to monitor and assess the interventions should be reviewed a regular basis to determine effectiveness and to link progress or lack of progress specifically to the intervention. Prescribed interventions change over time as new research is conducted, as new theories are developed, and as new technology is designed.

See Also

- [Academic Supports](#)
- [Activity-Based Instruction](#)
- [Early Intervention](#)
- [Education](#)

References and Reading

- Carter, E. W., Sisco, L. G., Yun-Ching, C., & Stanton-Chapman, T. L. (2010). Peer interactions of students with intellectual disabilities and/or autism: A map of the intervention literature. *Research and Practice for Persons with Severe Disabilities*, 35(3/4), 63–79.
- Hume, K., Loftin, R., & Lantz, J. (2009). Increasing independence in autism spectrum disorders: A review of

three focused interventions. *Journal of Autism and Developmental Disorders*, 39, 1329–1338.

- Lynch, S. L., & Irvine, A. N. (2009). Inclusive education and best practice for children with autism spectrum disorder: An integrated approach. *International Journal of Inclusive Education*, 13(8), 845–859.

National Research Council. (2001). *Educating children with autism*. Committee on Educational interventions for Children with Autism (Catherine Lord & James P. McGee, Eds.). Division of Behavioral and Social Sciences and Education. Washington, DC: National Academy Press.

Odom, S. L., Collet-Klingenberg, L., Rogers, S. J., & Hatton, D. D. (2010). Evidence-based practices in interventions for children and youth with autism spectrum disorders. *Preventing School Failure*, 54(4), 275–282.

Sherer, M. R., & Schreibman, L. (2005). Individual behavioral profiles and predictors of treatment effectiveness for children with autism. *Journal of Consulting and Clinical Psychology*, 73(3), 525–538.

Educational Psychology

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Definition

Educational psychology is the study of the relationship between learning and our physical and social environments. Educational psychologists study the psychological processes involved in learning and develop strategies for enhancing the learning process. Educational development focuses on the cognitive development, and what a teacher can do impacts the development of the child. It teaches about how children learn and how they can be taught for them to be successful. Educational psychologists “study what people think and do as they teach and learn a particular curriculum in a particular environment where education and training are intended to take place.” Two fundamental assumptions that underlie formal educational systems are that students (a) retain knowledge and skills they acquire in school and (b) can apply them in situations

outside the classroom. Aspirations of the discipline rested on the application of the scientific methods of observation and experimentation to educational questions. Ultimately, the goal of those working in the field of educational psychology is to understand the processes and conditions under which human beings, both in childhood and throughout their lifespan, learn to become mature human beings who fulfill their individual potential, live in service to their community, and exercise loving stewardship over the environment.

It is likely that educational psychologists will continue to contribute to education as they learn more about the brain and how learning occurs; the development of intellect, affect, personality, character, and motivation; ways of assessing learning; and the creation of multifaceted learning environments.

The *Journal of Educational Psychology* is a peer-reviewed academic journal that was established in 1910 and covers educational psychology. It is published by the American Psychological Association.

See Also

► [School Psychologist](#)

References and Reading

- Alexander, P. A. (1996). The past, present, and future of knowledge research: A reexamination of the role of knowledge in learning and instruction. *Educational Psychologist*, 31, 89–92.
- Berliner, D. C. (1993). The 100-year journey of educational psychology: From interest, to disdain, to respect for practice. In T. K. Fagan & G. R. Vanden-Bos (Eds.), *Exploring applied psychology: Origins and critical analyses*. Washington, DC: American Psychological Association.
- Mayer, R. E. (1992). Cognition and instruction: Their historic meeting within educational psychology. *Journal of Educational Psychology*, 84, 405–412.
- Woolfolk Hoy, A. (2001). *Educational psychology* (8th ed.). Boston: Allyn and Bacon.
- Zimmerman, B. J., & Schunk, D. H. (Eds.). (2002). *Educational psychology: A century of contributions*. Mahwah: Erlbaum.

Educational Psychotherapy

► [Educational Therapy](#)

Educational Testing

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E

Synonyms

[Academic testing](#); [Achievement testing](#)

Definition

Educational testing refers to the formal assessment of academic achievement. This can take the form of group or individually administered tests which to formally evaluate performance in the areas of reading, decoding, and comprehension; writing, spelling, mathematics, computation, and problem solving; and, on occasion, other subject areas such as science. Test scores compare the individual's performance to age-based or grade-based normative groups. These scores are frequently used as part of the assessment academic progress and in the diagnosis and monitoring of specific learning disabilities or learning disorders. Commonly used individually administered educational testing instruments include the Wechsler Individual Achievement Test 3rd Edition, the Woodcock-Johnson Achievement Battery, and the Kaufman Achievement Battery for Children Edition II.

The Achievement Test Desk Reference: A Guide to Learning Disability Identification by Flanagan et al. (2006).

References and Reading

- Flanagan, D. P., Ortiz, S. O., Alfonso, V. C., & Mascolo, J. T. (2006). *The achievement test desk reference: A guide to learning disability identification*. Hoboken: Wiley.

Educational Therapy

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Synonyms

[Education as therapy](#); [Educational psychotherapy](#);
[Therapeutic education](#)

Definition

Educational therapy (also known as educational psychotherapy) has roots in early psychoanalytical models of child development but is now more concerned with remediation of learning and emotional difficulties based on neuropsychological models. In the UK, there is a master's qualification in educational psychotherapy but no official role for educational therapists (ETs) within the educational system and most work is undertaken privately. In the USA, the work of ETs is also private but certified by professional organizations such as the Association of Educational Therapists (AET). The focus of the work of ETs is on those with specific learning difficulties (such as dyslexia) and emotional and behavioral difficulties, rather than ASD. Their goal is to improve academic success (Ficksman and Adelizzi 2010) by addressing psychological factors that may be impeding learning. The AET approves training courses in educational therapy, which are at postgraduate to master's level. Entry requirements include qualified teacher status, educational psychology, or qualified therapist status with some experience of special education. Other routes may be approved by the AET. ETs may work with individuals within the autism spectrum, but the efficacy of such work has not been established. They focus on changing, or building, underlying psychological processes which are involved in auditory processing, reading, or on positively influencing self-esteem and optimism, which are known to be related to academic success. Evidence of efficacy, even in cases without an ASD, is largely based on case study or

pre-post designs (Maslow and Ungerleider 2007), which are not scientifically robust.

Jordan (2005) used the term “educational therapy” to refer to aspects of special education in autism spectrum disorders (ASD) that address the core symptoms; it is included as a concept in specialist autism training for professionals in the UK and features in the UK and European guidelines for good practice in ASD. It is intended to be an adjunct to academic education, but individuals with ASD will also need compensatory techniques to help them access the culturally valued skills, knowledge, and understanding to which everyone is entitled (i.e., the usual curriculum). Educational therapy is intended to characterize the work of all who educate and care for those on the spectrum; it is not confined to therapists. It is based on the recognition that explicit specialist education can help remediate many problems in ASD, but specific attempts to do so require evaluation. Golan et al. (2010) (a matched controlled study without random allocation to groups) and Herrera et al. (2008) (a small case study) are typical of evaluations in this area, where there is still need for more robust scientific evaluation.

Neither “education as therapy,” as used by Jordan, nor the work of ETs is meant to replace the entitlement of children with ASD to a full and broad education. Therapists, including ETs, may have a role in ASD in addressing specific difficulties or in meeting emotional and mental health needs. ETs may also address some of the comorbid specific learning difficulties that may accompany ASD. However, such roles are aids to development and to access educational entitlement, rather than alternatives to education. Likewise, Jordan's view of educational therapy expresses the need to extend education in ASD to include specific teaching of aspects of development that occur naturally in those not on the autism spectrum, not to limit education to those aspects.

See Also

- ▶ [Cognitive Behavioral Therapy \(CBT\)](#)
- ▶ [Cognitive Enhancement Therapy](#)

References and Reading

- Ficksman, M., & Adelizzi, J. U. (Eds.). (2010). *The clinical practice of educational therapy*. London: Taylor & Francis.
- Golan, O., Ashwin, E., Granader, Y., McClintock, S., Day, K., Leggett, V., et al. (2010). Enhancing emotional recognition in children with autism spectrum conditions: An intervention using animated vehicles with real emotional faces. *Journal of Autism and Developmental Disorders*, 40, 269–279.
- Herrera, G., Alcantua, F., Jordan, R., Blanquer, A., Labajo, G., & De Pablo, C. (2008). Development of symbolic play through the use of virtual reality tools in children with autistic spectrum disorders: Two case studies. *Autism: The International Journal of Research and Practice*, 12(2), 143–158.
- Jordan, R. R. (2005). Managing autism and Asperger's syndrome in current educational provision. *Paediatric Rehabilitation*, 8, 104–112.
- Maslow, P., & Ungerleider, D. (2007). The efficacy of educational therapy Pt.2. *The Educational Therapist*, 28(3), 16–21.

EEG Abnormalities as a Biomarker of Severity in ASD

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Synonyms

EEG anomalies; EEG discharges; Epileptiform abnormalities

Definition

Compared with the healthy population, patients with autism spectrum disorder (ASD) have an increased incidence of epilepsy (2–3% vs. 5–46%, respectively). Studies show a higher risk of autism in patients suffering from specific forms of epilepsy such as infantile spasms and Lennox-Gastaut syndrome. EEG abnormalities in subjects with ASD, without epilepsy, are detected with prevalence ranging from 6.7% to

61% and associated with more severe behavioral problems, thus indicating a distinct neuroelectrophysiological signature. The EEG findings include non-epileptiform abnormalities, such as slow activities or asymmetries in background rhythm, and, more frequently, epileptiform abnormalities. Commonly, the epileptiform abnormalities are morphologically classified as follows: spikes or sharp waves, slow waves, generalized wave-spike, or generalized polyspikes. Also, epileptiform abnormalities include interictal discharges (generalized, multifocal or focal, unilateral or bilateral), affecting different brain areas. Although there is no consensus among researchers, it seems that the EEG abnormalities occur more frequently in temporal regions in ASD population. The EEG abnormalities are most commonly encountered during sleep recording, suggesting the importance of performing the EEG in siesta status and prolonging the EEG recording in order to grasp all the sleep phases in management of ASD patients. Several studies reported a significantly higher incidence of epileptiform abnormalities in patients with ASD when compared with subjects with Asperger, highlighting that the increasing severity of autistic symptoms might be associated with higher likelihood of epileptiform abnormalities. In particular, EEG abnormalities in children with ASD were correlated with a higher incidence of autism severity, hyperactivity, anger outbursts, aggression, negative or destructive behavior, motor stereotypies, intellectual disability, language impairment, and self-harm. Whereas the association between epilepsy and cognitive disability is well-known, the potential role of isolated EEG abnormalities on cognitive functioning in subjects with ASD is uncertain. A well-known example of how EEG abnormalities can be associated with speech disorders is Landau-Kleffner's syndrome, an acquired epileptic aphasia in which bilateral and independent continuous sleep waves, predominant on temporal lobes, are associated with severe language deficits and, often, regressive behaviors. Rolandic epilepsy has been also associated with cognitive impairment, particularly in the presence of frequent epileptic discharges. In this type of epilepsy, written and oral language deficits are also documented. These data suggest that EEG

abnormalities could be considered as a neurophysiological biomarker of severity of cognitive and behavioral problems associated with ASD. However, whether the epileptiform abnormalities are considered as an expression of an underlying brain dysfunction, how these functional abnormalities are related to the clinical phenotype is still unclear. The literature data highlighting a clear association between epileptiform abnormalities and language disorders, hyperactivity, and intellectual disability in subjects with ASD are in line with a modern interpretation of neurodevelopment disorders, considered as a continuum of disease rather than distinct nosological categories. In fact, the presence of EEG abnormalities in different disorders, both in autistic and non-autistic subjects, suggests that structural and/or functional alterations of the cortex could be considered as a common thread among different pathologies. Therefore, the presence of EEG abnormalities in subjects with ASD could be useful for a more correct selection of patients for gene sequencing studies and in the identification of patients that may have a better outcome on anti-convulsants. Although “treating the EEG” is based on the hypothesis that discharges may contribute to the behavioral, language, or cognitive disturbances, to date, there are only few favorable case reports of EEG treatment in patients with ASD. Specifically, the literature suggests that only a small proportion of ASD population with EEG abnormalities improve with antiepileptic drug treatment. In conclusion, epileptiform abnormalities, even in the absence of epilepsy, are common in subjects with ASD and correlate with the severity of cognitive and behavioral problems associated with ASD. Therefore, it is reasonable to look at EEG abnormalities as a neurophysiological biomarker of ASD severity.

References and Reading

- Hagerman, R. J. (2013). Epilepsy drives autism in neurodevelopmental disorders. *Developmental Medicine & Child Neurology*, 55(2), 101–102. <https://doi.org/10.1111/dmcn.12071>. PMID: 23320573.
- Mulligan, C. K., & Trauner, D. A. (2014). Incidence and behavioral correlates of epileptiform abnormalities in

- autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(2), 452–458. <https://doi.org/10.1007/s10803-013-1888-6>. PMID: 23872941.
- Nicotera, A. G., Hagerman, R. J., Catania, M. V., Buono, S., Di Nuovo, S., Liprino, E. M., Stracuzzi, E., Giusto, S., Di Vita, G., & Musumeci, S. A. (2019). EEG abnormalities as a neurophysiological biomarker of severity in autism spectrum disorder: A pilot cohort study. *Journal of Autism and Developmental Disorder*, 49(6), 2337–2347. <https://doi.org/10.1007/s10803-019-03908-2>. PMID: 30726535.
- Rossi, P. G., Parmeggiani, A., Bach, V., Santucci, M., & Visconti, P. (1995). EEG features and epilepsy in patients with autism. *Brain & Development*, 17(3), 169–174. [https://doi.org/10.1016/0387-7604\(95\)00019-8](https://doi.org/10.1016/0387-7604(95)00019-8). PMID: 7573755.
- Trauner, D. A. (2015). Behavioral correlates of epileptiform abnormalities in autism. *Epilepsy & Behavior*, 47, 163–166. <https://doi.org/10.1016/j.yebeh.2014.10.020>. PMID: 25453621.
- Veerappan, V. D., Sweetha, B., Kavitha, H. R., Sivalingam, B., Nambi, S., & Pauline, L. (2018). Two-year follow-up of isolated epileptiform discharges in autism: An endophenotypic biomarker? *Indian Journal of Psychological Medicine*, 40, 219–224. https://doi.org/10.4103/IJPSYM.IJPSYM_555_17. PMID: 29875528.
- Volkmar, F. R., & Nelson, D. S. (1990). Seizure disorders in autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 29(1), 127–129. <https://doi.org/10.1097/00004583-199001000-00020>. PMID: 2295565.

EEG Anomalies

- [EEG Abnormalities as a Biomarker of Severity in ASD](#)

EEG Connectivity During Social Engagement in Autism

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Definition

The magnitude of coupling between two distinct electroencephalogram (EEG) signals measured during social engagement in individuals with autism.

Historical Background

Several theories of autism spectrum disorder (ASD) propose that a disturbance in neural organization during early development results in altered brain connectivity and trajectories of impaired social and communicative development (e.g., Belmonte et al. 2004; Courchesne and Pierce 2005; Just et al. 2004, 2007). Altered brain connectivity in ASD has been posited to result from excessive brain growth and aberrant neural pruning during early neurodevelopment (Courchesne and Pierce 2005). Theoretically, this can have consequences for the developing *social brain* – a functional network of brain structures that are involved in mediating social functioning (Adolphs 2009). For example, altered brain connectivity can result in inefficient integration among neural networks that support social cognitive function. Children with ASD may therefore develop with limited neurocognitive resources to effectively deal with the information processing demands of a dynamic social world. This may in turn degrade the affected child's social experiences during critical periods of experience-dependent neurodevelopment, thereby exacerbating social deficits. Thus, altered brain connectivity – particularly in the social brain – may be a potential nexus of social impairment in ASD.

However, characterizing an exact connectivity pattern that is specific to all of the dynamic brains of individuals with ASD is unlikely. This is evidenced by a large brain connectivity literature in ASD that presents mixed findings indicating reduced functional connectivity at rest and during a variety of tasks (e.g., Cherkassky et al. 2006; Coben et al. 2008; Darmala et al. 2010; Jones et al. 2010; Just et al. 2004, 2007; Kana et al. 2006; Koshino et al. 2005, 2008; Lazarev et al. 2013; Lynch et al. 2013; Villalobos et al. 2005; Wicker et al. 2008) as well as evidence of increased resting and task-based functional connectivity in ASD (e.g., Lynch et al. 2013; Shih et al. 2010; Uddin et al. 2013a). There are also findings in which both increased and decreased functional connectivity is observed in individuals with ASD depending on the frequency of neural

oscillations and distance between neural networks (e.g., Kitzbichler et al. 2015). Other factors that make delineating functional connectivity in ASD difficult are the use of different tasks, age groups, and measurement techniques (e.g., Kana et al. 2014; Uddin et al. 2013b).

Current Knowledge

The measurement of brain connectivity involving electroencephalography (EEG connectivity) is derived from biophysical processes in the brain that differ from the biophysical processes used in other neuroimaging approaches. Brain connectivity measures derived from functional magnetic resonance imaging (fMRI), for example, use the hemodynamic response – the rapid blood flow to active neuronal populations – to examine coupled activity between defined regions in the brain. EEG connectivity measures use oscillating electrical activity captured by sensors on the scalp (Nunez and Srinivasan 2006). These electrical fields on the scalp's surface are generated from the synchronous synaptic activity of millions of cortical neurons. They are represented in the EEG as oscillating waveforms that fluctuate in frequency and amplitude over time. The human EEG typically fluctuates in amplitude between 10 and 100 microvolts and between 1 and 100 Hz in frequency. Furthermore, EEG frequencies are clustered into *bands* that have been associated with different brain states and/or mental activities. For example, medium to high frequency bands are associated with attention, perception, and cognitive processing and lower frequency bands with a relaxed state or deep sleep. The EEG frequency bands are as follows: 1–4 Hz (delta), 4–7.5 Hz (theta), 8–12 Hz (alpha), 12.5–30 Hz (beta), and 30–100 Hz (gamma). EEG connectivity metrics can measure coupled activity both within and between frequency bands (Cohen 2014).

The use of EEG connectivity measures has advantages and disadvantages relative to fMRI. For example, EEG connectivity measures have better temporal resolution. That is, because the time course of electrical potentials in the brain is faster (in the order of milliseconds) than the

hemodynamic response, EEG connectivity measures are presumed to be more closely linked to perceptual and cognitive processes. Another advantage is that EEG acquisition does not require the participants to be placed inside a magnetic scanner; therefore, EEG can be more readily acquired in children and infants. One disadvantage of EEG connectivity is that it cannot inform about connectivity between specific brain structures. This poor spatial resolution of EEG connectivity occurs because the electric fields produced from neural generators within the brain instantly spread through biological tissue as they move up toward the sensors on the scalp, a process referred to as volume conduction (Nunez and Srinivasan 2006). As a result, coupled activity between two EEG sensors can reflect activity from a single rather than distinct neural generators. Volume conduction can therefore lead to spurious EEG connectivity results and is a confound that can plague studies involving EEG connectivity. There are however a number of computational approaches that can mitigate – but not completely remove – the effects of volume conduction in EEG connectivity research (Cohen 2014). In addition, the use of advanced quantitative methods and high-density EEG nets (100 + sensors) can be used to localize sources in the brain where the neural signal is generated (Michel and He 2019). One commonly used EEG connectivity metric is *coherence*. Coherence is a statistical estimate of the amount of phase stability between two EEG signals and represents the degree of synchronous activity between distinct neural networks in the brain (Nunez and Srinivasan 2006). Since coherence reflects that stability of phase differences between two EEG oscillating signals, the use of the coherence metric can reduce the volume conduction confound (Bastos and Schoffelen 2016).

Despite a large number of EEG connectivity studies in children and adults with ASD during rest, sleep, and the performance of nonsocial tasks, relatively few ASD studies have examined EEG connectivity during tasks involving social engagement (see O'Reilly et al. 2017; Schwartz et al. 2017 for a review on EEG connectivity studies in ASD). Given that one of the core

deficits of ASD is social functioning, there is knowledge to be gained from EEG connectivity studies that examine the ASD-affected brain while engaged within a social context. Prior fMRI research has explored the socially engaged brain in ASD by scanning subjects during live person-to-person social interaction via linked video feeds. This work has demonstrated that adults with ASD have reduced activity in brain areas that support social cognition relative to non-ASD controls (Redcay et al. 2013). However, a potential issue with fMRI work is that participants must remain still as movements can distort the signal and affect the interpretation of the results. These movement constraints can create conditions that may feel too artificial as well as present methodological difficulties when implemented with children with ASD.

Studies that have examined EEG connectivity during social engagement thus far have mainly involved participants passively observing social stimuli such as still images of faces or videos engendering joint social attention. Consistent with the broader literature on functional connectivity in ASD, EEG connectivity studies during social engagement report variable results such as reduced and/or increased coherence across different frequency bands, between sensors located on the left and right side of the head (interhemispheric coherence), and between sensors located within the same side of the head (intra-hemispheric coherence). For example, in a study that examined coherence during the perception of static faces in infants who later received a diagnosis of ASD, there was increased left intra-hemispheric coherence in the faster gamma frequency band (Keehn et al. 2015). Another study examined coherence in infants who had an older sibling with ASD (at-risk infants) as they observed social videos of a woman playing the game “peek-a-boo” (Orekhova et al. 2014). In this study, the at-risk infant group demonstrated increased interhemispheric coherence between frontal sites in the alpha frequency band relative to low-risk infants. Recently, Haartsen et al. (2019) demonstrated that alpha band EEG connectivity in 14-month-old infants who later met

ASD criteria at 36 months was positively associated with the severity of restricted and repetitive behaviors as measured by the Autism Diagnostic Observation Schedule (ADOS-2; Gotham et al. 2007). Taken together, this pattern of results suggests that infants who are more likely to display symptoms of ASD in later development may initially have overconnected brains.

Altered EEG connectivity during social engagement has also been reported in children already diagnosed with ASD. Carson et al. (2014) measured coherence while children watched videos of either a familiar or unfamiliar person reading a story. They also measured coherence during a rest condition. Results revealed that across both story conditions, there was decreased interhemispheric coherence in the alpha band in children with ASD relative to the typically developing (control) group. However, there was decreased alpha coherence in the rest condition as well. Jaime et al. (2016) examined coherence in adolescent children with ASD while they observed a series of 30-second videos that engendered joint attention. Joint attention involves the coordination of visual attention to share a point of view with another person and is recognized as one of the central impairments in early nonverbal social communication in ASD (Mundy 2016). These joint attention videos consisted of a red dot appearing at one of the four corners of the screen. As the red dot appeared in each corner, a person positioned in the center of the screen subsequently gazed (with the eyes and head) at one of the corners. In the congruent condition, the person gazed at the corner in which the red dot appeared. In the incongruent condition, the model gazed away from the corner in which the red dot appeared. The congruency of the joint attention videos was manipulated because prior research has demonstrated that when adult participants observe inconsistencies between the direction of gaze and a target, the superior temporal sulcus (STS) – a brain structure associated with social cognition – shows an increase in activity in typically developing adults but not in adults with ASD (Pelphrey et al. 2005). Thus, Jaime et al. (2016) hypothesized that the incongruent joint attention

condition would result in an increase in coherence for typically developing adolescents but adolescents with ASD would not show a change in coherence in response to the incongruency of joint attention.

The Jaime et al. (2016) study reported that the ASD group had reduced coherence in the alpha frequency band between the right temporal and central (i.e., midline of the scalp) EEG sensors. These are sensors that are located topographically near the STS region. There was reduced temporal–central alpha coherence only when coherence was collapsed across congruent and incongruent joint attention conditions and a separate condition in which adolescents fixated to a static dot. Thus, inconsistent with Pelphrey et al. (2005), the effect of incongruency did not appear to affect functional EEG coherence in either diagnostic group. The authors speculated that the instructions provided to participants, which was to “follow the dot,” may have precluded participants from inferring the actor’s intentions during the incongruent condition because participants were likely focused on performing the task correctly. Thus, a procedure in which participants would be allowed to visually explore the joint attention videos freely and without instruction might have revealed an interaction between congruent and incongruent joint attention and diagnostic status. The authors also reported that right temporal–central alpha coherence during the congruent and incongruent joint attention conditions correlated positively with performance on a test of social cognition in typically developing adolescents, but not in adolescents with ASD. Finally, two studies have examined EEG connectivity during social engagement in samples with high-functioning adults with ASD. Catarino et al. (2013) reported reduced interhemispheric alpha coherence in the ASD group relative to typically developing controls during a task requiring face recognition. However, these diagnostic group differences were only observed between one pair of temporal EEG sensors. Another study with high-functioning adults with ASD examined phase coupling across frequency bands during a task involving the identification of emotional

expressions in photographs of faces (Tseng et al. 2015). Results revealed reduced delta–theta phase coupling in the ASD group between central and parietal EEG sensors.

Due to the limited number of studies and variability in methodology, it is difficult to delineate the nature of functional connectivity during social engagement in individuals diagnosed with ASD. Thus far these studies do reveal a trend of reduced alpha band EEG connectivity both within and between hemispheres during social engagement tasks in children and adults with ASD. However, more research is needed, particularly studies that use standardized social interaction tasks such as the ADOS-2 in order to better characterize the nature of functional connectivity in the socially engaged brains of individuals with ASD.

Future Directions

Efforts to identify early biomarkers of ASD remain a key clinical goal for improvements in diagnosis. One research area that is garnering attention, but is still in its infancy, is the merging of advanced computational, data-driven techniques with EEG for the objective detection of ASD. There is a rapidly growing literature in the computer science field indicating that artificial learning systems can be powerful tools for detecting idiosyncrasies in the spectral patterns of EEG and can therefore be trained to accurately classify EEG records that indicate ASD from those that do not. There are recent findings that are in line with this hypothesis (e.g., Bosi et al. 2018), and data is rapidly emerging suggesting that machine learning algorithms that use EEG connectivity measures acquired during live social interaction in their feature set can achieve classification accuracies above 90% (Jayarathna et al. 2019). However, more studies with rigorous methodology and peer review will be needed for validation. If proven to be true, however, the integration of EEG connectivity during social engagement with machine learning approaches may significantly change the diagnostic process in the foreseeable future.

References and Reading

- Adolphs, R. (2009). The social brain: The neural basis of social knowledge. *Annual Reviews of Psychology*, 60, 693–716.
- Bastos, A. M., & Schoffelen, J.-M. (2016). A tutorial review of function connectivity analysis methods and their interpretational pitfalls. *Frontiers in Systems Neuroscience*, 9, 1–23.
- Belmonte, M. K., Allen, G., Beekel-Mitchener, A., Boulanger, L. M., Carper, R. A., & Webb, S. J. (2004). Autism and abnormal development of brain connectivity. *Journal of Neuroscience*, 24, 9228–9231.
- Bosi, W. J., Tager-Flusberg, H., & Nelson, C. A. (2018). EEG analytics for early detection of autism spectrum disorder: A data-driven approach. *Scientific Reports*, 8(1), 6828.
- Carson, A. M., Salowitz, N. M. G., Scheidt, R. A., Dolan, B. K., & Van Hecke, A. V. (2014). Electroencephalogram coherence in children with and without autism spectrum disorders: Decreased interhemispheric connectivity in autism. *Autism Research*, 7, 334–343.
- Catarino, A., Andrade, A., Churches, O., Wagner, A. P., Baron-Cohen, S., & Ring, H. (2013). Task-related functional connectivity in autism spectrum conditions: An EEG study using wavelet transform coherence. *Molecular Autism*, 4(1), 1.
- Cherkassky, V. L., Kana, R. K., Keller, T. A., & Just, M. A. (2006). Functional connectivity in a baseline resting-state network in autism. *Brain Imaging*, 17, 1687–1690.
- Coben, R., Clarke, A. R., Hudspeth, W., & Barry, R. J. (2008). EEG power and coherence in autistic spectrum disorder. *Clinical Neurophysiology*, 119, 1002–1009.
- Cohen, M. X. (2014). *Analyzing neural time series data: Theory and practice*. Cambridge, MA: The MIT Press.
- Courchesne, E., & Pierce, K. (2005). Brain overgrowth in autism during a critical time in development: Implications for frontal pyramidal neuron and interneuron development and connectivity. *International Journal of Developmental Neuroscience*, 23, 153–173.
- Darmala, S. R., Keller, T. A., Kana, R. K., Cherkassky, V. L., Williams, D. L., Minshew, N. J., & Just, M. A. (2010). Cortical underconnectivity coupled with preserved visuospatial cognition in autism: Evidence from an fMRI study of an embedded figures task. *Autism Research*, 3, 273–279.
- Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The autism diagnostic observation schedule: Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders*, 37, 613–627.
- Haartsen, R., Jones, E. J. H., Orekhova, E. V., Charman, T., Johnson, M. H., & The BASIS team. (2019). Functional EEG connectivity in infants associates with later restricted and repetitive behaviours in autism; a replication study. *Translational Psychiatry*, 9, 66.
- Jaime, M., McMahon, C. M., Davidson, B. C., Newell, L. C., Mundy, P. C., & Henderson, H. A. (2016). Reduced temporal-central EEG alpha

- coherence during joint attention perception in adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46, 1477–1489.
- Jayarathna, S., Jayawardana, Y., Jaime, M., & Thapaliya, S. (2019). Electroencephalogram (EEG) for delineating objective measure of autism spectrum disorder. In C. Chen & S. S. Cheung (Eds.), *Computational models for biomedical reasoning and problem solving* (pp. 34–65). Hershey: IGI Global.
- Jones, T., Bandettini, P. A., Kenworthy, L., Case, L. K., Milleville, S. C., Martin, A., & Birn, R. M. (2010). Sources of group differences in functional connectivity: An investigation applied to autism spectrum disorder. *NeuroImage*, 49, 401–414.
- Just, M. A., Cherkassky, V. L., Keller, T., & Minshew, N. J. (2004). Cortical activation and synchronization during sentence comprehension in high-functioning autism: Evidence of underconnectivity. *Brain*, 127, 1811–1821.
- Just, M. A., Cherkassky, V. L., Keller, T., Rajesh, K. K., & Minshew, N. J. (2007). Functional and anatomical cortical underconnectivity in autism: Evidence from an fMRI study of an executive function task and corpus callosum morphometry. *Cerebral Cortex*, 17, 951–961.
- Kana, R. K., Keller, T. A., Cherkassky, V. L., Minshew, N. J., & Just, M. A. (2006). Sentence comprehension in autism: Thinking in pictures with decreased functional connectivity. *Brain*, 129, 2484–2493.
- Kana, R. K., Uddin, L. Q., Kenet, T., Chugani, D., & Müller, R.-A. (2014). Brain connectivity in autism. *Frontiers in Human Neuroscience*, 8, 349.
- Keehn, B., Vogel-Farley, V., Tager-Flusberg, H., & Nelson, C. A. (2015). Atypical hemispheric specialization for faces in infants at risk for autism spectrum disorder. *Autism Research*, 8, 187–198.
- Kitzbichler, M. G., Khan, S., Ganesan, S., Vangel, M. G., Herbert, M. R., Hamalainen, M. S., & Kenet, T. (2015). Altered development and multifaceted band-specific abnormalities of resting state networks in autism. *Biological Psychiatry*, 77, 794–804.
- Koshino, H., Carpenter, P. A., Minshew, N. J., Cherkassky, V. L., Keller, T. A., & Just, M. A. (2005). Functional connectivity in an fMRI working memory task in high functioning autism. *NeuroImage*, 24, 810–821.
- Koshino, H., Kana, R. K., Keller, T. A., Cherkassky, V. L., Minshew, N. J., & Just, M. A. (2008). fMRI investigation of working memory for faces in autism: visual coding and underconnectivity with frontal areas. *Cerebral Cortex*, 18, 289–300.
- Lazarev, V. V., Pontes, A., Mitrofanov, A. A., & deAzevedo, L. C. (2013). Reduced interhemispheric connectivity in childhood autism detected by electroencephalographic photic driving coherence. *Journal of Autism and Developmental Disorders*, 45, 537–547.
- Lynch, C. J., Uddin, L. Q., Supekar, K., Khouzam, A., Phillips, J., & Menon, V. (2013). Default mode network in childhood autism: Posteromedial cortex heterogeneity and relationship with social deficits. *Biological Psychiatry*, 74, 212–219.
- Michel, C. M., & He, B. (2019). EEG source localization. In K. Levin & P. Chauvel (Eds.), *Handbook of clinical neurology* (pp. 85–101). Elsevier. <https://doi.org/10.1016/B978-0-444-64032-1.00006-0>
- Mundy, P. C. (2016). *Autism and joint attention: Development, neuroscience, and clinical fundamentals*. New York: Guilford Press.
- Nunez, P. L., & Srinivasan, R. (2006). *Electric fields of the brain: The neurophysics of EEG*. New York: Oxford University Press.
- O'Reilly, C., Lewis, J. D., & Elsabbagh, M. (2017). Is functional connectivity atypical in autism? A systematic review of EEG and MEG studies. *PLoS One*, 12(5), e0175870.
- Orehova, E. V., Elsabbagh, M., Jones, E. J., Dawson, G., Charman, T., & Johnson, M. H. (2014). EEG hyper-connectivity in high-risk infants is associated with later autism. *Journal of Neurodevelopmental Disorders*, 6, 40.
- Pelphrey, K. A., Morris, J. P., & McCarthy, G. (2005). Neural basis of eye gaze processing in autism. *Brain*, 128, 1038–1048.
- Redcay, E., Dodell-Feder, D., Pearrow, M. J., Mavros, P. L., Kleiner, M., Gabrieli, J. D. E., & Saxe, R. (2010). Live face-to-face interaction during fMRI: A new tool for social cognitive neuroscience. *NeuroImage*, 50, 1639–1647.
- Redcay, E., Dodell-Feder, D., Mavros, P. L., Kleiner, M., Pearrow, M. J., Triantafyllou, C., Gabrieli, J. D., & Saxe, R. (2013). Atypical brain activation patterns during a face-to-face joint attention game in adults with autism Spectrum disorder. *Human Brain Mapping*, 34, 2511–2523.
- Schwartz, S., Kessler, R., Gaughan, T., & Buckley, A. W. (2017). EEG coherence patterns in autism: An updated review. *Pediatric Neurology*, 67, 7–22.
- Shih, P., Shen, M., Ottl, B., Keehn, B., Gaffrey, M. S., & Muller, R. A. (2010). Atypical network connectivity for imitation in autism spectrum disorder. *Neuropsychologia*, 48, 2931–2939.
- Tseng, Y.-L., Yang, H. H., Savostyanov, A. N., Chien, V. S. C., & Liou, M. (2015). Voluntary attention in Asperger's syndrome: Brain electrical oscillation and phase-synchronization during facial emotion recognition. *Research in Autism Spectrum Disorders*, 13–14, 32–51.
- Uddin, L. Q., Supekar, K., Lynch, C. J., Khouzam, A., Phillips, J., Feinstein, C... Menon, V. (2013a). Salience network-based classification and prediction of symptom severity in children with autism. *JAMA Psychiatry*, 70, 869–879.
- Uddin, L. Q., Supekar, K., & Menon, V. (2013b). Reconceptualizing functional brain connectivity in autism from a developmental perspective. *Frontiers in Human Neuroscience*, 7, 458.
- Villalobos, M. E., Mizuno, A., Dahl, B. C., Kemmotsu, N., & Muller, R. A. (2005). Reduced functional

connectivity between V1 and inferior frontal cortex associated with visuomotor performance in autism. *NeuroImage*, 25, 916–925.

Wicker, B., Fonlupt, P., Hubert, B., Tardif, C., Gepner, B., & Deruelle, C. (2008). Abnormal cerebral effective connectivity during explicit emotional processing in adults with autism spectrum disorder. *Social Cognitive and Affective Neuroscience*, 3, 135–143.

EEG Discharges

► [EEG Abnormalities as a Biomarker of Severity in ASD](#)

Effect of Visual Information on Postural Control in Adults with Autism Spectrum Disorder

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Definition

Postural control is the ability to keep an upright posture of the body within the base of support. Its purpose is to provide humans with stability and to orientate their bodies to perform a variety of tasks, such as standing, walking, and reaching (Horak 2006; Massion 1994). Postural control relies on a postural control system that is dependent on many aspects of the body (Horak 2006). All aspects of the body, such as the feet size, joint movements, and sensory processing within the brain, provide relevant information to the central nervous system for the

control of posture. The context of the surrounding also plays an important role in postural control. Information about the surrounding is picked up by the senses of vision, touch, and hearing and sent to the central nervous system. The central nervous system combines all of the information to make sense of the body position in the context of the surrounding. Any change in the surrounding can affect one's posture. Therefore, all information must be organized accurately so that the correct motor commands can be sent out to different parts of the body. The motor commands guide the body to move in the direction of front/back or left/right to keep the body in a stable posture (Winter 1995).

Postural control can be measured in several ways using standardized clinical tests or measuring instruments. It is mostly assessed while standing because standing is known to be inherently unstable (Hwang et al. 2016). Standardized clinical tests are usually used to assess postural control in clinics as they can be done in a short time (Mancini and Horak 2010). The tests help clinicians identify postural control problems in their patients and then use the information to guide treatments. Examples of these tests include the Peabody Developmental Motor Scales, Gross Motor Function Measure, Timed “Up and Go” test, and Berg Functional Balance Scale (Mancini and Horak 2010; Westcott et al. 1997). Instrumented measures of postural control are usually used to provide an objective assessment of postural control for research and sports (Mancini and Horak 2010). These instruments typically record ground reaction force exerted on the surface of the instruments when the person stands on it. The force signals are later quantified into the center of pressure measurements for further analysis. An example of these instruments includes the force platform (Mancini and Horak 2010; Paillard and Noe 2015).

Historical Background

People with autism spectrum disorder (ASD) have often been documented in research literature to have problems with postural control. A large

amount of research on postural control in ASD involves children with ASD. Using comparisons of postures between children with ASD and typically developing children, those with ASD have been shown to have unusual postures (Kohen-Raz et al. 1992). Unlike typically developing children, children with ASD tend to hyper-extend their backs and have low muscle tone and an unusual walking style (Paquet et al. 2016). Assessments of postural control using measuring instruments also show that children with ASD tend to have a less stable posture than typically developing children when there are changes in their surrounding (Kohen-Raz et al. 1992; Molloy et al. 2003). Overall, the research shows consistent findings of postural control problems in children with ASD.

Current Knowledge

While much is known about postural control in children with ASD, there is limited research evidence with regard to adults with ASD. Understanding postural control in both children and adults with ASD is essential to identify problem areas of postural control and to appreciate the postural control system and its development in those with ASD across the lifetime (Minschew et al. 2004). Over the past 15 years, only a small quantity of studies involving adults with ASD has been added to the literature (Lim et al. 2017). Therefore, little is known if postural instability in childhood ASD is normalized by adulthood.

Of the limited research about postural control in adults with ASD, there is poor consistency in the findings. Among the few studies, most of them examined if changes in visual information could affect postural control in adults with ASD. These usually involve participants opening and closing their eyes or looking at moving scenes while standing. Under these testing conditions, some studies have found that adults with ASD show similar control of posture as typically developed adults (Greffou et al. 2012; Travers et al. 2013). In contrast, other studies have reported a more unstable posture in those with ASD than

that in typical adults when they used visual information during postural control (Doumas et al. 2016; Morris et al. 2015). Two recent studies have also found that adults with ASD responded differently from typical adults in visual environments controlled by immersive virtual technology (Lim et al. 2018a, b). Adults with ASD showed a more unstable posture when they had to focus on a central target and when exposed to a busy, incoherent, and unfamiliar visual environment while standing (Lim et al. 2018a, b). In addition, adults with ASD appeared to invest a larger amount of attention in postural control than typical adults when visual information was used during postural control (Lim et al. 2018b). Although some of the studies have indicated an important influence of visual environment on postural control in ASD, further research is still needed to ascertain these findings.

There have been several proposed reasons for postural control problems in adults ASD. One reason concerns the aspect of sensory information organization in the central nervous system. Sensory processing problems are commonly reported in people with ASD outside of motor behavior (American Psychiatric Association 2015). Mechanisms that have been proposed to explain sensory problems in ASD include an increased excitation-to-inhibition ratio (Rubenstein and Merzenich 2003); increased endogenous noise (Simmons et al. 2009); and altered neural network connectivity (Markram and Markram 2010). Problems related to any of these mechanisms could result in a series of inaccurate information relayed within the system. Thus, this would diminish the capacity for generating appropriate motor commands for postural control. Additionally, problems with an inaccurate representation of the body in space as a result of any process would result in the delivery of inaccurate motor commands for postural control (Van de Cruys et al. 2014).

Problems with postural control could affect a person's independence, and thus further understanding of postural control in adults with ASD is essential. Problems with postural control could mean that adults with ASD face difficulty completing tasks that require a stable posture,

such as lifting and carrying items and overhead reaching. The ability to perform these tasks is necessary for adults who want to work and live on their own (Happé and Charlton 2012). In addition, older adults with ASD could face more problems with postural control due to age-related body changes. A combination of body changes related to old age and pre-existing postural control problems could reduce a person's capacity to complete tasks on their own and could increase the risk of fall in older adults with ASD (Happé and Charlton 2012). Besides the need to know more about postural control in adults with ASD, further understanding of postural control across people of different age groups in ASD is needed. Knowing how postural control develops with age could contribute to the advancement and development of diagnostic tools and treatments for people with ASD in all age groups.

Future Directions

There is scope for future research to expand the way postural control is assessed in adults with ASD. Postural control is influenced by different types of sensory information in the environment (Horak 2006). However, little is known about the impact of somatosensory and vestibular information on postural control in adults with ASD. An understanding of how different types of sensory information affect the control of posture would provide more insights into the postural control system in adults with ASD.

Future studies may also want to understand how the brain responds during a postural control task in adults with ASD. Currently, there is limited knowledge about the neural mechanisms responsible for postural control problems in ASD. Knowing how the brain responds during a postural control task could help to identify areas in the brain contributing to postural control problem in ASD.

There may be benefits to investigate the association between postural control and the level of functional independence in adults with ASD. Functional independence is important for people who work and live on their own. However, it

is unclear if and how postural control problems in adults with ASD affect their functional independence. Understanding the physical needs of adults with ASD could guide the planning of resources to help them cope with daily tasks at home and in the community.

It is also important for future research to involve people with ASD from different age groups. A comprehensive understanding of the postural control system in ASD relies on knowing how postural control is presented across all ages. This also includes people in age groups that are not commonly assessed in the research literature, such as adolescents and older adults with ASD. Findings from future studies that include people of different age groups will be useful to understand how the postural control system changes with age and to come up with ways to treat postural control problems in ASD.

See Also

- [Deficits in Attention, Motor Control, and Perception](#)
- [Gross Motor Skills](#)
- [Motor Control](#)
- [Motor Planning](#)
- [Posturography](#)
- [Visual-Motor Function](#)

References and Reading

- American Psychiatric Association. (2015). *DSM-5 Classification* (5th ed.). Arlington: American Psychiatric Association Publishing.
- Doumas, M., McKenna, R., & Murphy, B. (2016). Postural control deficits in autism spectrum disorder: The role of sensory integration. *Journal of Autism and Developmental Disorders*, 46(3), 853–861. <https://doi.org/10.1007/s10803-015-2621-4>.
- Greffou, S., Bertone, A., Hahler, E. M., Hanssens, J. M., Mottron, L., & Faubert, J. (2012). Postural hypo-reactivity in autism is contingent on development and visual environment: A fully immersive virtual reality study. *Journal of Autism and Developmental Disorders*, 42(6), 961–970. <https://doi.org/10.1007/s10803-011-1326-6>.
- Happé, F., & Charlton, R. A. (2012). Aging in autism spectrum disorders: A mini-review. *Gerontology*, 58(1), 70–78. <https://doi.org/10.1159/000329720>.

- Horak, F. B. (2006). Postural orientation and equilibrium: What do we need to know about neural control of balance to prevent falls? *Age and Ageing*, 35(Suppl 2), ii7–ii11. <https://doi.org/10.1093/ageing/afii077>.
- Hwang, S., Agada, P., Kiemel, T., & Jeka, J. J. (2016). Identification of the unstable human postural control system. *Frontiers in Systems Neuroscience*, 10, 22–22. <https://doi.org/10.3389/fnsys.2016.00022>.
- Kohen-Raz, R., Volkmar, F. R., & Cohen, D. J. (1992). Postural control in children with autism. *Journal of Autism and Developmental Disorders*, 22(3), 419–432.
- Lim, Y. H., Partridge, K., Girdler, S., & Morris, S. L. (2017). Standing postural control in individuals with autism spectrum disorder: Systematic review and meta-analysis. *Journal of Autism and Developmental Disorders*, 47(7), 2238–2253. <https://doi.org/10.1007/s10803-017-3144-y>.
- Lim, Y. H., Lee, H. C., Falkmer, T., Allison, G. T., Tan, T., Lee, W. L., & Morris, S. L. (2018a). Effect of optic flow on postural control in children and adults with autism spectrum disorder. *Neuroscience*, 393, 138–149. <https://doi.org/10.1016/j.neuroscience.2018.09.047>.
- Lim, Y. H., Lee, H. C., Falkmer, T., Allison, G. T., Tan, T., Lee, W. L., & Morris, S. L. (2018b). Effect of visual information on postural control in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-018-3634-6>.
- Mancini, M., & Horak, F. B. (2010). The relevance of clinical balance assessment tools to differentiate balance deficits. *European Journal of Physical and Rehabilitation Medicine*, 46(2), 239–248.
- Markram, K., & Markram, H. (2010). The intense world theory – A unifying theory of the neurobiology of autism. *Frontiers in Human Neuroscience*, 4(224), 1–29. <https://doi.org/10.3389/fnhum.2010.00224>.
- Massion, J. (1994). Postural control system. *Current Opinion in Neurobiology*, 4(6), 877–887.
- Minshew, N. J., Sung, K., Jones, B. L., & Furman, J. M. (2004). Underdevelopment of the postural control system in autism. *Neurology*, 63(11), 2056–2061. <https://doi.org/10.1212/01.wnl.0000145771.98657.62>.
- Molloy, C. A., Dietrich, K. N., & Bhattacharya, A. (2003). Postural stability in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 33(6), 643–652.
- Morris, S. L., Foster, C. J., Parsons, R., Falkmer, M., Falkmer, T., & Rosalie, S. M. (2015). Differences in the use of vision and proprioception for postural control in autism spectrum disorder. *Neuroscience*, 307, 273–280. <https://doi.org/10.1016/j.neuroscience.2015.08.040>.
- Paillard, T., & Noe, F. (2015). Techniques and methods for testing the postural function in healthy and pathological subjects. *BioMed Research International*, 2015, 891390. <https://doi.org/10.1155/2015/891390>.
- Paquet, A., Olliac, B., Golse, B., & Vaivre-Douret, L. (2016). Current knowledge on motor disorders in children with autism spectrum disorder (ASD). *Child Neuropsychology*, 22(7), 763–794. <https://doi.org/10.1080/09297049.2015.1085501>.
- Rubenstein, J. L., & Merzenich, M. M. (2003). Model of autism: Increased ratio of excitation/inhibition in key neural systems. *Genes, Brain, and Behavior*, 2(5), 255–267.
- Simmons, D. R., Robertson, A. E., McKay, L. S., Toal, E., McAleer, P., & Pollick, F. E. (2009). Vision in autism spectrum disorders. *Vision Research*, 49(22), 2705–2739. <https://doi.org/10.1016/j.visres.2009.08.005>.
- Travers, B. G., Powell, P. S., Klinger, L. G., & Klinger, M. R. (2013). Motor difficulties in autism spectrum disorder: Linking symptom severity and postural stability. *Journal of Autism and Developmental Disorders*, 43(7), 1568–1583. <https://doi.org/10.1007/s10803-012-1702-x>.
- Van de Cruys, S., Evers, K., Van der Hallen, R., Van Eylen, L., Boets, B., de-Wit, L., & Wagemans, J. (2014). Precise minds in uncertain worlds: Predictive coding in autism. *Psychological Review*, 121(4), 649–675. <https://doi.org/10.1037/a0037665>.
- Westcott, S. L., Lowes, L. P., & Richardson, P. K. (1997). Evaluation of postural stability in children: Current theories and assessment tools. *Physical Therapy*, 77(6), 629–645.
- Winter, D. A. (1995). Human balance and posture control during standing and walking. *Gait & Posture*, 3(4), 193–214.

Effectiveness of a SCERTS Intervention

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Definition

The SCERTS (social communication, emotional regulation, and transactional support) model is an evidence-based multidisciplinary educational approach developed by a team of professionals trained in different disciplines (Prizant et al. 2003). The model focuses on addressing the core challenges faced by children with autism spectrum disorders (ASD) and their families by

enhancing competence in social communication, emotional regulation, and transactional support.

Historical Background

The SCERTS model was derived from abundant empirical research and clinical investigation into both conventional and unconventional social communication skills in ASD over two decades. Having recognized the core deficits of ASD in social interaction and emotional regulation, and the need for a comprehensive educational model that can address these impairments, a team of researchers and practitioners developed the SCERTS model. In developing this model, they made use of scholarly literature concerned with communication and with socioemotional development in children, as well as clinical experiences of ASD and other developmental disabilities. While SCERTS is intended to address common deficits in ASD, the model takes “into account critical individual differences across families in reference to their priorities, and their involvement in critical programmatic decision-making” (Prizant et al. 2003, p. 298). It can thus be implemented in an individualized manner and is considered family-centered.

The SCERTS model has two notable features. Firstly, the model focuses on social communication and emotional regulation, which resonate with the most important priorities in helping children with ASD to develop life skills (Handleman and Harris 2006; National Research Council [NRC, USA] 2001). Building competence in areas of socioemotional and communication skills in children with ASD helps them to achieve better long-term outcomes, and this model echoes the evidence-based practice recommended by researchers and practitioners working in the field of ASD and related developmental disabilities (Prizant et al. 2003). Secondly, the SCERTS model highlights the implementation of “transactional support” and the collective input by different social partners, which unites people surrounding the child to work together as a multidisciplinary team (O’Neill et al. 2010). Such a partnership among family, peers, teachers,

and professionals facilitates the implementation of individualized service in different settings, which helps children with ASD and their families to achieve more positive outcomes. The unique emphasis on multidisciplinary teamwork also makes the model flexible enough to incorporate other practices from approaches such as the Treatment and Education of Autistic and Communication Handicapped Children (TEACCH) (Schopler et al. 1995) and the Developmental, Individual-difference, and Relationship-based model (DIR) (Wieder and Greenspan 2005; Greenspan and Wieder 1997).

The SCERTS model provides a comprehensive framework that guides the development and implementation of different educational programs for children with ASD and for supporting families. It has been widely adopted in various intervention and education programs across the world (Molteni et al. 2013; Walworth 2007; Walworth et al. 2009). Researchers reported that programs based on the SCERTS model can have positive impacts not only for participating children with ASD but also for others involved, such as parents, special education teachers, and therapists (Prizant et al. 2003, 2005). Although the current research evidences vary in terms of methodology (e.g., randomized control trial, single-case experimental designs and case control, cross-sectional or longitudinal descriptive group research designs, etc.), they generally support the treatment impact in one or more domains focused on in the SCERTS model (Morgan et al. 2018; O’Neill et al. 2010; Walworth et al. 2009).

Current Knowledge

Evaluation study is a valuable tool for collecting, analyzing, and using information to answer basic questions about the effectiveness of intervention models, which helps researchers find out “what works” and “what does not work.” Limited evaluation studies have been conducted to systematically examine the effectiveness of the SCERTS model (Arick et al. 2003).

In the United Kingdom, researchers carried out a case study that investigated the impact of the

SCERTS model-based intervention on four elementary school students in a special education school. The results showed that, after 12 months of intervention, the participating students demonstrated significant improvement in the areas of joint attention, symbol use, mutual regulation, and self-regulation. The students also became more focused on tasks and ready to learn. Self-awareness of one's own needs and the use of regulations as required were also found to be improved in the four participants. It was further revealed that apart from the impact on children, the SCERTS model facilitated the development of positive multiagency cooperation in the school and supported collaboration among different school workers. The SCERTS model served as an effective way of working with children with ASD. This encouraged educators to think about their school curriculum and altered the culture of the school and the teaching style of adults (O'Neill et al. 2010).

Another reported case study was conducted in New Zealand. This examined the efficacy of a program that integrated the SCERTS model with music therapy for a 3-year-old boy with ASD. After 1 year of intervention, the boy showed positive changes in the areas of active learning, social membership, independence, cooperation, and appropriateness of behaviors (Ayson 2011). In the United States, researchers investigated the effectiveness of a SCERTS model-based parent-implemented intervention, the Early Social Interaction (ESI) Project, with 82 children diagnosed with ASD at 16–20 months (Wetherby et al. 2014). The intervention focused on teaching parents how to use strategies to support children's communication in everyday activities. Based on a randomized controlled trial (RCT), the effects of individual intervention (parents were trained at home or in the community) and group intervention (parents were trained in a clinic) were examined and compared in terms of participating children's social communication skills, adaptive behaviors, and developmental level. The results demonstrate the effectiveness of both individual and group intervention, while children in the individual home coaching group showed greater improvement on the social components of

communication, receptive language, and core social deficits. The researchers concluded that the SCERTS model-based parent-implemented intervention was effective, and little professional time was needed.

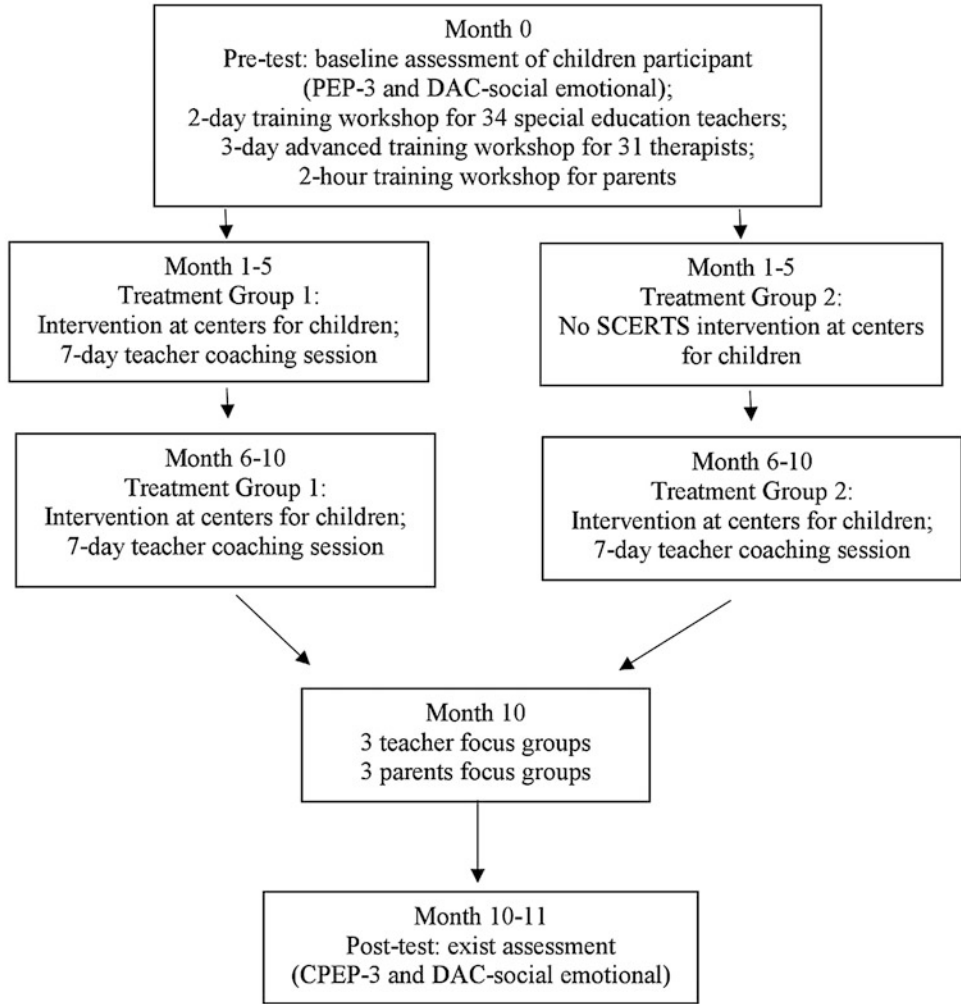
More recently, researchers reported a cluster randomized trial (CRT) that examined the effects of a SCERTS model-based intervention by classroom teachers in an elementary school setting in the United States (Morgan et al. 2018). A total of 60 elementary schools with 197 students with ASD and 129 teachers were randomly assigned to either a classroom SCERTS model-based intervention group or a normal school-based education group with autism training modules group. Standardized assessments, teachers' and parents' reports, and direct observation of student behaviors were employed to measure the intervention effect across various domains. Baseline and end-of-treatment assessments involved measures of active engagement (e.g., emotion regulation, productivity, social connectedness, directed communication, generative language production, and academic independence), adaptive behavior, executive functioning, and social outcomes. The results showed that the classroom SCERTS model-based intervention group had significantly better outcomes than the usual school-based education group with autism training modules group. Significantly better outcomes were found in the SCERTS group students' social participation, adaptive communication, social skills, problem behaviors, and executive functioning. The effect sizes were from small to medium. These findings supported the efficacy of a classroom-based, teacher-implemented, SCERTS model-based intervention.

In contrast with the widespread use of the SCERTS model in the West, its application in China has been limited. Consequently, there has been a lack of evaluation studies on the effectiveness of the SCERTS model in helping Chinese children with ASD. A recent research project in Hong Kong launched by Heep Hong Society has contributed to filling this gap. The project involves (1) a training program on the SCERTS model for professionals and parents; (2) the implementation of the SCERTS model-based

intervention; and (3) an evaluation study that examined the effectiveness of the SCERTS model-based intervention in training preschool children with ASD. The project’s procedures are depicted in Fig. 1. The following paragraphs report details of the training program, the SCERTS model-based intervention, and the evaluation findings.

The first part of the program offered training to educators from 10 special childcare centers in Hong Kong where interests in learning and applying the SCERTS model had been indicated.

A total of 65 professionals, including special education teachers, physiotherapists, speech therapists, and occupational therapists attended the training. The training program consisted of two major components. The first component was a 2-day training workshop on the SCERTS model for all special education teachers ($N = 34$). The second component was a 3-day advanced training workshop on the SCERTS model for all the therapists (11 physiotherapists, 10 speech therapists, and 10 occupational therapists). In addition, a 2-h training workshop was provided to parents of the



Effectiveness of a SCERTS Intervention, Fig. 1 Procedure of the project. (Reprinted from *Effectiveness of a SCERTS Model-Based Intervention for Children with Autism Spectrum Disorder (ASD) in Hong Kong:*

A Pilot Study, by L. Yu and X. Zhu, 2018, *Journal of Autism and Developmental Disorders*, 48, p. 3797). Copyright 2018 by Journal of Autism and Developmental Disorders

children in the 10 centers in order to help the parents understand the rationale of the SCERTS model, the basic ideas underlying it, and its application in the daily lives of children with ASD. After the training, all participating professionals implemented the SCERTS model-based intervention in their respective centers (Yu and Zhu 2018).

With regard to the implementation of the SCERTS model-based intervention, the 10 centers were randomly divided into two groups. Professionals in Group 1 implemented a 10-month intervention with 35 h of training per week; Group 2 implemented a 5-month intervention with the same number of hours of training per week. The training in both groups included (1) the special education teachers' daily teaching and (2) individual therapy and group treatment sessions provided by therapists on a weekly basis. In each center, a special education teacher taught five to seven children, while the size of the group treatment session was four to six children. The 5-month intervention (Group 2) was implemented 5 months after the commencement of the 10-month intervention (Group 1) (Yu and Zhu 2018).

Two measures were taken during the intervention period to ensure and monitor the quality of the program's implementation. Firstly, a 2-week coaching session on the application of the SCERTS model in the intervention practices with 6 h of observation and 2 h of case discussion each day were provided for special education teachers in the 10-month and 5-month groups, respectively. Secondly, three times during the intervention period, a total of eight trained professionals observed the implementation of the SCERTS model-based intervention at each center. A quality indicator scale was used to rate the extent to which each indicator was met in the areas of program planning, implementation, monitoring, adjustment, and transactional support including both learning and interpersonal support. Each item was rated on a 4-point scale: 0 = no or minimal evidence that this is happening; 1 = this is happening some of the time (less than 50%); 2 = this is happening most of the time (more than 50%); and 3 = this is always happening (more than 90% of the time) (Yu and Zhu 2018). These scores were used to provide feedback and

suggestions to the program implementers and to help them to address any problems which had been identified. In this study, good intervention quality was observed in both groups.

At the same time, a mixed-method evaluation study was carried out (1) to examine the impact of the SCERTS model-based intervention on the participating children in different developmental areas, especially in the domains of social communication and emotional regulation; (2) to investigate the relationship between the duration of an intervention and its effectiveness; and (3) to understand the opinions of the intervention implementers and the parents of participating children. The first two aims were addressed in the quantitative part of the study, based on a quasi-experimental pre- and posttest design focusing on participating children. The third aim was addressed in the qualitative part of the study focusing on the intervention implementers and the parents of participating children.

For the quantitative part, 122 children (age = 53.43 ± 9.05 months) were successfully recruited from the 10 special childcare centers with the following inclusion criterion: (1) children had a clinical diagnosis of ASD; (2) children age between 3 and 6.5; and (3) their primary caregivers gave informed consent. The children were rated by a group of trained professionals who were blind to the study using standardized assessment tools. The assessment was conducted before the implementation of the 10-month intervention and after the completion of all intervention sessions. For the qualitative part, six focus group interviews were conducted after the interventions with 19 parents (three groups) and 20 professionals (another three groups) by researchers with knowledge and experiences of the SCERTS model. The interview sessions focused on what parents and professionals thought about the SCERTS model, the changes they observed in the children after participating in the program, and the impacts of the model on their own knowledge, skills, and attitude towards children with ASD.

The findings showed that children in both the 10-month SCERTS model-based intervention group and the 5-month intervention group performed significantly better in social

communication and emotional behaviors than they did before the intervention. Apart from improvements in the core deficits of ASD, participating children also demonstrated significant development in other areas, such as fine motor skills, gross motor skills, visual-motor imitation, as well as improved personal self-care, as reported by caregivers. It has been proposed that these additional child outcomes of the intervention may be attributed to the enhanced transactional support, which serves as another key element of the SCERTS model. The study also found that duration of intervention has little influence on the effectiveness of the SCERTS model-based intervention. The 5-month intervention had similar impacts to those of the 10-month intervention on the participating children's development, suggesting that the effects of the SCERTS model-based intervention can be observed in a relatively short period of time. This may further encourage parents and professionals to continue to adopt the SCERTS model-based approach in their daily practices. These findings provide empirical support for the effectiveness of the SCERTS model-based intervention in improving children's development in multiple domains, among a sample of preschool children with ASD in Hong Kong. Given the limited evidence on the effects of comprehensive treatment models on child outcomes (Odom et al. 2010), particularly in a non-Western cultural context, these findings are uniquely important.

The results of the focus group interviews reveal that both program implementers (i.e., teachers and therapists) and parents expressed positive views about the SCERTS model, both reporting significant improvements in the participating children in different developmental areas. The training and implementation of the SCERTS model also had a positive impact on their own attitudes and behaviors when working with children with ASD. Specifically, both parents and professionals considered the SCERTS model to be a well-designed training model with clear objectives, assessment measures, intervention procedures, and evaluation tools. Based on the model, children's individual developmental needs can be identified, and this helped different social partners

to design tailor-made support for the children. Teacher participants also reported that the emotional regulation and transactional support concepts were very useful and helped them to better understand the children's needs. Consistent with the findings of the quantitative part, professionals and parents observed improvements in the children's social communication skills and emotional regulation, such as improved understanding of basic emotion, and the ability to use behavioral strategies to express their feelings more frequently. Parents also reported an increased confidence in children's expression and communication. Some children became more fluent in their speech and used a clearer tone of voice in communication after implementation of the SCERTS model-based intervention.

Moreover, parents and professionals reported that the SCERTS model-based intervention project shifted the way they think about and work with children with ASD. The project encouraged them to reflect on how to better work with the children and how to meet children's developmental needs. They displayed a higher sense of self-efficacy, more acceptance about children's challenging behaviors, and adopted a more positive approach to providing the necessary support to children with ASD. Educators reported that their roles changed from being highly directive to being more facilitative when they were provided with the SCERTS model-based intervention. More choices and shared control between educators and children encouraged a better understanding of their role in learning, improved the structure of lessons, and led to less problematic behaviors. The learning atmosphere became more relaxed with improved teacher-child relationships, enabling professionals to pay more attention to the promotion of children's social communication skills and emotional regulation abilities. Overall, both educators and parents reacted positively towards the SCERTS model-based intervention. Both were guided to respond to the needs and interests of children with ASD, to modify the environment, and to use different approaches to facilitate children's learning. At the same time, emotional and educational support provided to parents and educators further

improved their self-efficacy and confidence in coping with the difficulties that they encountered and in promoting improvements in children's key deficit areas as well as overall development. The qualitative findings verified the quantitative results and further demonstrated that the incorporation of the SCERTS model in training children with ASD will benefit the children and the related partners around the child.

The positive findings and successful experience of this research project have several practical implications. Professionals and researchers who are working with children with ASD may consider further integrating the model into their existing programs and investigating the impact of the program. Furthermore, regular training on the SCERTS model for parents of children with ASD would be beneficial. Such training empowers parents to apply the model in their daily life in order to facilitate their child's development. Finally, the model can be used to help different partners to collaborate closely with each other when working with children with ASD. These collaborative practices not only benefit the children and their families; they also have the potential to bring about positive changes in the practices of professionals and other treatment partners.

Future Directions

As a comprehensive treatment model for children with ASD, the SCERTS model has been well operationalized but has as yet yielded limited evidence on treatment outcomes (Lin et al. 2016; Odom et al. 2010). While the extant studies have provided preliminary empirical evidence for the effectiveness of the model, there remains a pressing need to systematically examine the implementation and impact of other types of SCERTS model-based intervention with rigorously designed research. Important future research directions might include performing randomized control trials (RCTs) to examine how the SCERTS model-based intervention program could contribute to positive changes in children with ASD in different age groups (such as

preschool, school-aged, adolescents), after excluding the effects of maturation. RCTs also need to be undertaken for the purpose of further investigating the relationship between the duration of intervention and its effectiveness. Additional research is needed to complement existing findings and to examine the effect of the SCERTS model-based intervention on the basis of a more representative sample of children with ASD. The long-term effects of the SCERTS model-based intervention furthermore will be followed up in longitudinal studies. Last, but not least, parents play an important role in enhancing the effectiveness of the SCERTS model-based intervention program, and future study should also examine the mediation effect of parental practices on children's outcomes.

In sum, intervention based on the SCERTS model recognizes the broader context of a child's development. It has a positive impact not only on children with ASD but also on their parents, teachers, and therapists. The SCERTS model provides a useful framework of intervention that can be potentially applied in a variety of educational and treatment settings to help children with ASD.

See Also

- [Developmental Intervention Model](#)
- [Early Intervention](#)
- [Family-Centered Care, Second Edition](#)
- [Multidisciplinary Evaluation](#)
- [Transactional Support \(SCERTS\) Model](#)

References and Reading

- Arick, J. R., Young, H. E., Falco, R. A., Loos, L. M., Krug, D. A., Gense, M. H., & Johnson, S. B. (2003). Designing an outcome study to monitor the progress of students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 18(2), 75–87.
- Ayson, C. (2011). The use of music therapy to support the SCERTS model objectives for a three year old boy with autism spectrum disorder in New Zealand. *New Zealand Journal of Music Therapy*, 9, 7.
- Greenspan, S. I., & Wieder, S. (1997). Developmental patterns and outcomes in infants and children with

- disorders in relating and communicating: A chart review of 200 cases of children with autistic spectrum diagnoses. *Journal of Developmental and Learning disorders*, 1, 87–142.
- Handleman, J. S., & Harris, S. L. (Eds.). (2006). *School-age education programs for children with autism*. Austin: Pro-Ed.
- Lin, T. L., Chiang, C. H., & Wu, H. C. (2016). Comprehensive early intervention research for children with autism spectrum disorder: A narrative overview. *Formosa Journal of Mental Health*, 29(1), 1–46.
- Linstead, E., Dixon, D. R., Hong, E., Burns, C. O., French, R., Novack, M. N., & Granpeesheh, D. (2017). An evaluation of the effects of intensity and duration on outcomes across treatment domains for children with autism spectrum disorder. *Translational Psychiatry*, 7(9), e1234.
- Molteni, P., Guldberg, K., & Logan, N. (2013). Autism and multidisciplinary teamwork through the SCERTS model. *British Journal of Special Education*, 40(3), 137–145.
- Morgan, L., Hooker, J. L., Sparapani, N., Reinhardt, V. P., Schatschneider, C., & Wetherby, A. M. (2018). Cluster randomized trial of the classroom SCERTS intervention for elementary students with autism spectrum disorder. *Journal of Consulting and Clinical Psychology*, 86(7), 631.
- National Research Council [NRC; USA]. (2001). *Educating children with autism*. Washington, DC: National Academic Press.
- O'Neill, J., Bergstrand, L., Bowman, K., Elliott, K., Mavin, L., Stephenson, S., & Wayman, C. (2010). The SCERTS model: Implementation and evaluation in a primary special school. *Good Autism Practice*, 11(1), 7–15.
- Odom, S. L., Boyd, B. A., Hall, L. J., & Hume, K. (2010). Evaluation of comprehensive treatment models for individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(4), 425–436.
- Prizant, B. M., & Rubin, E. (1999). Contemporary issues in interventions for autism spectrum disorders: A commentary. *Journal of the Association for Persons with Severe Handicaps*, 24(3), 199–208.
- Prizant, B. M., Wetherby, A. M., Rubin, E., & Laurent, A. C. (2003). The SCERTS model: A transactional, family-centered approach to enhancing communication and socioemotional abilities of children with Autism Spectrum Disorder. *Infants & Young Children*, 16(4), 296–316.
- Prizant, B. M., Wetherby, A. M., Rubin, E., Laurent, A. C., & Rydell, P. J. (2005). *The SCERTS model: A comprehensive educational approach for children with autism spectrum disorders*. Baltimore: Brookes.
- Rubin, E., Prizant, B. M., Laurent, A. C., & Wetherby, A. M. (2013). Social communication, emotional regulation, and transactional support (SCERTS). In *Interventions for Autism Spectrum Disorders* (pp. 107–127). New York: Springer.
- Schopler, E., Mesibov, G. B., & Hearsey, K. (1995). Structured teaching in the TEACCH system. In E. Schopler & G. B. Mesibov (Eds.), *Learning and cognition in autism* (pp. 243–268). New York: Springer.
- Walworth, D. D. (2007). The use of music therapy within the SCERTS model for children with Autism Spectrum Disorder. *Journal of Music Therapy*, 44(1), 2–22. <https://doi.org/10.1093/jmt/44.1.2>.
- Walworth, D. D., Register, D., & Engel, J. N. (2009). Using the SCERTS model assessment tool to identify music therapy goals for clients with Autism Spectrum Disorder. *Journal of Music Therapy*, 46(3), 204–216. <https://doi.org/10.1093/jmt/46.3.204>.
- Wetherby, A. M., Guthrie, W., Woods, J., Schatschneider, C., Holland, R. D., Morgan, L., & Lord, C. (2014). Parent-implemented social intervention for toddlers with autism: An RCT. *Pediatrics*, 134(6), 1084–1093.
- Wieder, S., & Greenspan, S. I. (2005). Developmental pathways to mental health: The DIR model for comprehensive approaches to assessment and intervention. In K. M. Finello (Ed.), *The handbook of training and practice in infant and preschool mental health* (pp. 377–401). San Francisco: Wiley.
- Yu, L., & Zhu, X. (2018). Effectiveness of a SCERTS model-based intervention for children with Autism Spectrum Disorder (ASD) in Hong Kong: A pilot study. *Journal of Autism and Developmental Disorders*, 48(11), 3794–3807.

Effectiveness of Parent-Child Interaction Therapy for Children With and Without Autism Spectrum Disorder

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Definition

Research and clinical experience suggest that a significant number of young children with autism spectrum disorder (ASD) present with coexisting clinically significant behavior problems. Children with ASD are often severely impacted in the areas of socialization, communication, repetitive behaviors, and restricted interests. The

impairment in communication and socialization may increase the risk for problem behaviors, making children with ASD more likely to display problem behaviors that warrant treatment. For children with ASD, an important social context for the development of both maladaptive, problem behaviors and adaptive, pro-social behaviors is the parent-child relationship, including parent-child interactions. Interventions that train parents to modify their interaction patterns often lead to positive behavioral changes in children with and without developmental disabilities. One intervention that has demonstrated effectiveness in typically developing young children and has a significantly growing research base for children with ASD for improving parent-child relationships, reducing problem behavior, and increasing child compliance is Parent-Child Interaction Therapy (PCIT). PCIT has been established as an effective, evidenced-based treatment for typically developing young children ages 2–7 with behavioral difficulties through highly specified, step-by-step, lived-coached sessions with both the caregiver and the child. Parent acquisition of skills taught in PCIT is gauged through the Dyadic Parent-Child Interaction Coding System (DPICS), and child behavior is typically assessed through parent report using the Eyberg Child Behavior Inventory (ECBI).

Historical Background

Behavioral Parent Training (BPT) programs are the most used and well-established treatments for children with externalizing problem behaviors. BPT programs train parents how to alter their child's behavior at home through behavior analytic techniques and principles. BPT programs have been shown to help alleviate symptoms of many disruptive behavior disorders and have additionally been shown to alleviate aggression and increase compliance in typically developing children. PCIT is one specific BPT program and is based on principles from attachment theory and social learning to promote an authoritative parenting style. Attachment theory informs the focus on a warm and responsive parent-child relationship

in order to develop a secure parent-child relationship. Social learning theory informs the behavioral techniques implemented within PCIT focused on consistent contingencies to alter dysfunctional interactions and patterns within the parent-child relationship. Theoretically, PCIT focuses initially on relationship enhancement (attachment) first as a necessary foundation for a later focus on effective limit setting, contingencies, and consistency in discipline. In PCIT parents learn to attend to positive behavior, ignore inappropriate behavior, and implement time-out for noncompliance and aggression.

Several treatment approaches have been used to improve the behavior, language, and social skills of children with autism, each with varied levels of empirical support. The more common and empirically supported include interventions based on applied behavior analysis, incidental teaching, errorless compliance training, and parent training (McDiarmid and Bagner 2005). PCIT incorporates a number of techniques that are traditionally seen in therapies addressing behavioral difficulties in children with ASD. Similar to Floortime and TEACCH, PCIT places a high degree of importance on consistent and high-quality parent-child interactions (Masse et al. 2007; Petrenko 2013). PCIT also shares similarities with pivotal response training in its emphasis of using familiar play objects, conducting training in an environment similar to the home, and a focus on generalization of skills (Masse et al. 2007). A common theme inherent within many interventions for children with ASD and what is often thought of as best practices for treatment of ASD (Smith and Iadarola 2015) is to take a comprehensive approach by allowing parents to play an integral part in therapy. By increasing parental involvement, skills learned within a clinic are then generalized to other settings such as the home and public environments. PCIT views the parent as the agent of change in a child's life. PCIT not only stresses the importance of relationship building through enriching and rewarding parent-child interactions but also contains an intensive compliance training component similar to the ABA discrete trial training.

The PCIT therapy protocol is structured in two phases, with the first focused on relationship enhancement (Child-Directed Interaction; CDI) and the second focused on parental consistency and discipline (Parent-Directed Interaction; PDI). Following a didactic parent-only session, parents are live-coached in the relationship building skills of **P**raise, **R**eflection, **I**mitation, **D**escription, and **E**njoyment (PRIDE). Coaching is continued until the parent reaches a specific level of mastery criteria in the observed skill use. Upon mastery of CDI skills, the therapist engages the caregiver in a didactic PDI teaching session followed by several PDI coaching sessions focused on the provision of effective commands, appropriate responses for child noncompliance, and strategies to increase compliance. PCIT employs a structured time-out technique as the primary discipline strategy. The taught time-out procedures can be individualized to the child and serve the purpose of eliminating a child's ability to escape a command before progressing with the parent-child interactions. The compliance training begins with the use of simple "play" commands prior to moving to more real-life instructions. Throughout PCIT parents review their progress on specific behaviors that are coded and graphed, are assigned homework activities, and are provided opportunities for independent problem-solving. Following therapy there is a graduation session where any additional resources are provided, and both the caregiver and the child receive certificates of completion.

Current Knowledge

PCIT is a widely recognized evidenced-based intervention for the reduction of child externalizing behaviors of typically developing children with over 25 years of research. Multiple meta-analyses of the effectiveness of PCIT for typically developing children support decreases in parental stress (Cooley et al. 2014) and in reduction in child behavior problem intensity (Cooley et al. 2014; Ward et al. 2016). Thomas et al. (2017) provided an updated comprehensive meta-analysis including both typically developing and

a variety of clinical populations such as children with mild to moderate intellectual impairment and developmental disabilities, preterm children, foster children, children with ADHD, children in Head Start families, and children who have experienced or are at risk for experiencing maltreatment. Findings suggest that PCIT is an effective means of reducing parenting stress and child problem behavior across a variety of child populations (Thomas et al. 2017).

Several single-subject design and small sample studies have examined the effectiveness of PCIT for children with ASD. All have reported a reduction in child problem behavior and an increase in positive parenting skills. Beyond child problem behavior and positive parenting skills, PCIT in the ASD population has been found to reduce maternal psychopathology (i.e., stress, depression, and anxiety), to increase child vocalization frequency, to decrease some ASD-related symptomology (e.g., atypicality and withdrawal), and to increase some ASD-related deficits (e.g., functional communication). Larger sample studies have decreases in child problem behavior with the ECBI and increases in positive parenting skills following PCIT for children with ASD (Furukawa et al. 2018; Parladé et al. 2019; Ros and Graziano 2019; Zlomke and Jeter 2019; Zlomke et al. 2017).

Future Directions

Although empirical evidence now supports the use of PCIT for children with ASD and coexisting disruptive behavior, several questions remain unanswered. Each study varies in their inclusion criteria, and by definition ASD is a heterogeneous diagnostic label. Therefore, future research should identify appropriateness of children with ASD for PCIT. It may be that certain language parameters, presence of self-injury, or other child characteristics may preclude the successful participation in PCIT.

Clinician tailoring of PCIT to a presenting parent-child dyad is standard practice. Given the unique presenting problems and symptoms present in children with ASD, variations in

implementation strategies, treatment targets, and techniques should be investigated. A proposed addition of a social-directed interaction (SDI) phase to the PCIT protocol would focus on communication abilities and social skills. A communication-focused SDI would incorporate command training and “when/then” statements. Communication targets could also include eye contact and greetings like “hello” and “good-bye.” PCIT with children with well-developed verbal abilities would possibly social skills targets (e.g., how to ask a question or get someone’s attention). In addition to the SDI phase, it has been recommended to add a time-out readiness phase after CDI and before PDI. The time-out readiness phase would allow for the specific teaching and practicing of compliance prior to the implementation of time-out. This phase would allow the child to gain access to expectations prior to time-out and allows the caregivers to practice their PDI skills prior to the implementation of time-out. During this time-out readiness phase, caregivers would also be taught physical prompting to achieve compliance without the use of time-out while they practice their skills, and the child experiences the compliance contingencies prior to experiencing time-out. Although these two suggested phases (social-directed interaction phase and time-out readiness phase) are currently untested, they may increase PCIT effectiveness and remain in the spirit of the theoretically based protocol.

References and Reading

- Cooley, M. E., Veldorale-Griffin, A., Petren, R. E., & Mullis, A. K. (2014). Parent-child interaction therapy: A meta-analysis of child behavior outcomes and parent stress. *Journal of Family Social Work, 17*, 191–208. <https://doi.org/10.1080/10522158.2014.888696>.
- Furukawa, K., Okuno, H., Mohri, I., Nakanishi, M., Eyberg, S. M., & Sakai, S. (2018). Effectiveness of child-directed interaction training for young Japanese children with autism spectrum disorders. *Child and Family Behavior Therapy, 40*(2), 166–186. <https://doi.org/10.1080/07317107.2018.1477344>.
- Ginn, N. C., Clionsky, L. N., Eyberg, S. M., Warner-Metzger, C., & Abner, J.-P. (2017). Child-directed interaction training for young children with autism spectrum disorders: Parent and child outcomes. *Journal of Clinical Child and Adolescent Psychology, 46*(1), 101–109. <https://doi.org/10.1080/15374416.2015.1015135>.
- Masse, J. J., McNeil, C. B., Wagner, S. M., & Chorney, D. B. (2007). Parent-child interaction therapy and high functioning autism: A conceptual overview. *Journal of Early and Intensive Behavior Intervention, 4*(4), 714–735. <https://doi.org/10.1037/h0100402>.
- McDiarmid, M. D., & Bagner, D. M. (2005). Parent child interaction therapy for children with disruptive behavior and developmental disabilities. *Education and Treatment of Children, 28*(2), 130–141. Retrieved from <https://search.ebscohost.com/login.aspx?direct=true&db=psyh&AN=2006-06834-003&site=ehost-live&scope=site>
- Parladé, M. V., Weinstein, A., Garcia, D., Rowley, A. M., Ginn, N. C., & Jent, J. F. (2019). Parent-child interaction therapy for children with autism spectrum disorder and a matched case-control sample. *Autism, 1*–17. <https://doi.org/10.1177/1362361319855851>.
- Petrenko, C. L. (2013). A review of intervention programs to prevent and treat behavioral problems in young children with developmental disabilities. *Journal of Developmental and Physical Disabilities, 25*, 651–679. <https://doi.org/10.1007/s10882-013-9336-2>.
- Ros, R., & Graziano, P. A. (2019). Group PCIT for preschoolers with autism spectrum disorder and externalizing behavior problems. *Journal of Child and Family Studies, 28*, 1294. <https://doi.org/10.1007/s10826-019-01358-z>.
- Smith, T., & Iadarola, S. (2015). Evidence base update for autism spectrum disorder. *Journal of Clinical Child & Adolescent Psychology, 44*, 897–922. <https://doi.org/10.1080/15374416.2015.1077448>.
- Solomon, M., Ono, M., Timmer, S., & Goodlin-Jones, B. (2008). The effectiveness of parent-child interaction therapy for families of children on the autism spectrum. *Journal of Autism and Developmental Disorders, 38*(9), 1767–1776. <https://doi.org/10.1007/s10803-008-0567-5>.
- Thomas, R., Abell, B., Webb, H. J., Avdagic, E., & Zimmer-Gembeck, M. J. (2017). Parent-child interaction therapy: A meta-analysis. *Pediatrics, 140*, 1–15. <https://doi.org/10.1542/peds.2017-0352>.
- Ward, M. A., Theule, J., & Cheung, K. (2016). Parent-child interaction therapy for child disruptive behaviour disorders: A meta-analysis. *Child & Youth Care Forum, 45*, 675–690. <https://doi.org/10.1007/s10566-016-9350-5>.
- Zlomke, K. R., & Jeter, K. (2019). Comparative effectiveness of parent-child interaction therapy for children with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders. https://doi.org/10.1007/s10803-019-03960-y*
- Zlomke, K. R., Jeter, K., & Murphy, J. (2017). Open-trial pilot of parent-child interaction therapy for children with Autism Spectrum Disorder. *Child and Family Behavior Therapy, 39*(1), 1–18. <https://doi.org/10.1080/07317107.2016.1267999>.

Effects of Social Stimuli on Postural Responses

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Definition of Autism Disorders and Postural Control

Autism spectrum disorders (ASD) refer to a range of conditions that are characterized by pervasive problems in social interactions and atypical motor behaviors (American Psychiatric Association 2013). Children with ASD usually have sensory deficits affecting fine and gross motor behaviors (Fournier et al. 2010). One of the key element governed by sensory motor processes is keeping the posture upright (Doumas et al. 2016; Kohen-Raz et al. 1992). Successful postural control requires integration of vestibular, somatosensory, and visual input (Doumas et al. 2016). Abnormal postures such as toe walking, hyperextension of the neck, or arching the back have been reported among children with ASD (Kohen-Raz et al. 1992). These atypical postural behaviors play a detrimental role in daily life and negatively affect independence of children on the spectrum. This can further exacerbate core symptoms of ASD by restricting social interactions and limiting opportunities to acquire perceptual-motor skills (Memari, et al. 2014; Travers et al. 2013).

Keeping posture upright is a fundamental skill, which has been the focus of attention in motor research. Postural control is the ability to maintain center of mass (COM) in the base of support (Memari et al. 2013). Position of the exerted ground reaction forces to confront body weight is considered as center of pressure (COP) (Shumway-Cook and Woollacott 2001). COP as the well-known index of postural control is an indicative of neuromuscular reactions to the

motion of body mass which retains the stability. Previous studies have shown that as children grow up, they gain more skills in keeping posture upright and as a result postural sway decreases (Hytönen et al. 1993). This might be considered in clinical diagnosis to portray a cardinal feature of postural control immaturity for children with neurodevelopmental disorders.

Historical Background of Postural Control

It has been shown that children with ASD exhibit higher postural sway and show atypical patterns compared with their typically developing (TD) peers (Memari et al. 2013; Minshew et al. 2004). Contrary to TD children, those with ASD have higher instability in medio-lateral direction than antero-posterior direction, suggesting postural immaturity, or an asynchrony between muscle stabilizers, or an alternative mechanism for generating postural control (Fournier et al. 2010; Memari et al. 2013). Postural instability among children with ASD was more pronounced when the somatosensory input either vestibular or visual or both were disrupted (Goulème et al. 2017; Minshew et al. 2004). Increase in sensory information demand or task difficulty will result in more postural sway in both groups of ASD and TD. However, because children with ASD have problems in multimodal sensory integration, the difference is significantly accentuated in children with ASD (Doumas et al. 2016; Minshew et al. 2004; Stins et al. 2015).

Because in real-life situations postural control is usually accompanied by a cognitive secondary task such as an audio or visual task, investigating the dual task paradigm can help to uncover mechanisms of postural control. It has been shown that children with developmental disorders, Tourette syndrome, or ASD will show more postural sway in dual task conditions (Laufer et al. 2008; Lemay et al. 2010; Memari et al. 2014); however, findings in the TD population are mixed (Pellecchia 2003; Riley et al. 2005). This might be due to the difficulty of the task and cognitive load which is involved. When two tasks are involved, they will

compete to allocate more attentional resources within the limited attentional capacity. Therefore, children with developmental disorders such as ASD with restricted attentional resources show higher postural sway when the attention is divided during a concurrent task (Memari et al. 2014; Pellecchia 2003).

Considering different types of tasks, children with ASD exhibit higher postural sway in inspection task than visual searching task (Chang et al. 2010). They also showed higher postural instability in visual searching than audio digit span task (Memari et al. 2014). This might be an indicative of higher visual reliance among children with ASD to maintain balance (Molloy et al. 2003; Stins et al. 2015). Thus, executing simultaneous visual tasks or any manipulation on visual input during postural control will decrease visual attentional resources dedicated to postural control. This will lead to higher postural sway among children with ASD to a greater extent than among their TD peers.

Current Knowledge on Postural Control and Social Cues

Recently, uncovering effects of social stimuli on postural control has been an upsurge of interest. It seems that there is an interconnection between motor control system and affective system. Previous studies have shown that social cues can alter arousal level and affect postural control (Gea et al. 2014; Ghanouni et al. 2017; Hillman et al. 2004; Stins and Beek 2007). For example, TD children showed larger postural sway when viewing happy and sad faces compared with angry or neutral ones (Goulème et al. 2017). Also, viewing unpleasant pictures can decelerate heart rate, decrease postural sway, and induce muscle stiffness (Azevedo et al. 2005; Roelofs et al. 2010; Stins and Beek 2007). These physiological responses resemble the freezing-like maneuvers seen in threatening situations. Coarse and fast processing of threatful stimuli is rooted in survival and evolution. Therefore, increased backward postural sway as opposed to forward sway when viewing unpleasant pictures will show approach-avoidance motor responses to affective stimuli (Gea et al. 2014;

Lopes et al. 2009). However, the extents of this connection among individuals with developmental disorders such as ASD are yet to be explored.

Previous study has shown that children with ASD, compared with their TD peers, exhibit higher postural sway when viewing social stimuli such as faces versus objects (Ghanouni et al. 2017). Authors also found that the extent of postural sway is moderated by the severity of the autistic disorder. This is an interesting finding as children with ASD have problems in perceiving social stimuli and may experience higher arousal and anxiety in social contexts (Dalton et al. 2005). This might be the reason that children with ASD usually avert their gaze from facial stimuli. When children with ASD view social versus object stimuli, induced higher anxiety and arousal will lead to higher postural sway (Ghanouni et al. 2017; Horslen and Carpenter 2011).

According to the weak central coherence theory, individuals with ASD have a tendency and bias towards attending to details rather than looking at the picture as a whole (Happé 2013). Facial stimuli require configural or global processing, a task which children with ASD have problems in doing it (Behrmann et al. 2006). Therefore, it can be assumed that the high-cognitive task of facial processing will recruit more attentional resources among children with ASD compared with their TD counterparts. Due to the limited attentional capacity, the residual attentional resources will contribute to the postural control, which in return accentuates the postural instability observed among children with ASD (Karatekin 2004; Pellecchia 2003). However, further investigations on the underlying mechanisms of postural control among population with ASD are warranted. This might be a potential avenue for preparing a new diagnosis tool or a model to detect early signs of developmental disorders such as ASD.

Future Directions

Comparing mechanisms of development and impairments in postural control among individuals with and without ASD would help to

distinguish patterns of maturation, compensation, and abnormality. Currently, there is little information on how postural control is maintained or affected among populations with ASD. Using various environmental perturbations, cognitive tasks, and difficulty levels would be helpful to uncover mechanisms involved in the postural control among individuals with ASD. Difficulties in postural control among populations with ASD may be associated with the presence of other comorbid conditions such as attention deficit hyperactivity disorders. However, there is a dearth of literature on how comorbidity, age, intellectual functioning, gender, and autistic traits will modulate postural responses in various contexts. Future studies should consider evaluation of postural control in conjunction with psychological responses such as pupillary dilation, eye gaze, heart rate, and electromyography of muscles among populations with ASD to portray more robust pictures of these phenomena.

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. Arlington: American Psychiatric Publishing.
- Azevedo, T. M., Volchan, E., Imbiriba, L. A., Rodrigues, E. C., Oliveira, J. M., Oliveira, L. F., et al. (2005). A freezing-like posture to pictures of mutilation. *Psychophysiology*, 42(3), 255–260.
- Behrmann, M., Avidan, G., Leonard, G. L., Kimchi, R., Luna, B., Humphreys, K., & Minshew, N. (2006). Configural processing in autism and its relationship to face processing. *Neuropsychologia*, 44(1), 110–129.
- Chang, C.-H., Wade, M. G., Stoffregen, T. A., Hsu, C.-Y., & Pan, C.-Y. (2010). Visual tasks and postural sway in children with and without autism spectrum disorders. *Research in Developmental Disabilities*, 31(6), 1536–1542.
- Dalton, K. M., Nacewicz, B. M., Johnstone, T., Schaefer, H. S., Gernsbacher, M. A., Goldsmith, H. H., et al. (2005). Gaze fixation and the neural circuitry of face processing in autism. *Nature Neuroscience*, 8(4), 519–526.
- Doumas, M., McKenna, R., & Murphy, B. (2016). Postural control deficits in autism spectrum disorder: The role of sensory integration. *Journal of Autism and Developmental Disorders*, 46(3), 853.
- Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010a). Motor coordination in autism spectrum disorders: A synthesis and meta-analysis. *Journal of Autism and Developmental Disorders*, 40(10), 1227–1240.
- Fournier, K. A., Kimberg, C. I., Radonovich, K. J., Tillman, M. D., Chow, J. W., Lewis, M. H., et al. (2010b). Decreased static and dynamic postural control in children with autism spectrum disorders. *Gait & Posture*, 32(1), 6–9.
- Gea, J., Muñoz, M. A., Costa, I., Ciria, L. F., Miranda, J. G., & Montoya, P. (2014). Viewing pain and happy faces elicited similar changes in postural body sway. *PLoS One*, 9(8), e104381.
- Ghanouni, P., Memari, A.-H., Gharibzadeh, S., Eghlidi, J., & Moshayedi, P. (2017). Effect of social stimuli on postural responses in individuals with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 5(47), 1305–1313.
- Goulème, N., Scheid, I., Peyre, H., Maruani, A., Clarke, J., Delorme, R., & Bucci, M. P. (2017). Spatial and temporal analysis of postural control in children with high functioning autism spectrum disorder. *Research in Autism Spectrum Disorders*, 40, 13–23.
- Happé, F. (2013). *Weak central coherence. Encyclopedia of autism spectrum disorders* (pp. 3344–3346). New York: Springer.
- Hillman, C. H., Rosengren, K. S., & Smith, D. P. (2004). Emotion and motivated behavior: Postural adjustments to affective picture viewing. *Biological Psychology*, 66(1), 51–62.
- Horslen, B. C., & Carpenter, M. G. (2011). Arousal, valence and their relative effects on postural control. *Experimental Brain Research*, 215(1), 27–34.
- Hytönen, M., Pyykkö, I., Aalto, H., & Starck, J. (1993). Postural control and age. *Acta Oto-Laryngologica*, 113(2), 119–122.
- Karatekin, C. (2004). Development of attentional allocation in the dual task paradigm. *International Journal of Psychophysiology*, 52(1), 7–21.
- Kohen-Raz, R., Volkman, F. R., & Cohen, D. J. (1992). Postural control in children with autism. *Journal of Autism and Developmental Disorders*, 22(3), 419–432.
- Laufer, Y., Ashkenazi, T., & Josman, N. (2008). The effects of a concurrent cognitive task on the postural control of young children with and without developmental coordination disorder. *Gait & Posture*, 27(2), 347–351.
- Lemay, M., Lê, T.-T., Richer, F., & Group, M. T. S. (2010). Effects of a secondary task on postural control in children with Tourette syndrome. *Gait & Posture*, 31(3), 326–330.
- Lopes, F. L., Azevedo, T. M., Imbiriba, L. A., Freire, R. C., Valença, A. M., Caldirola, D., et al. (2009). Freezing reaction in panic disorder patients associated with anticipatory anxiety. *Depression and Anxiety*, 26(10), 917–921.

- Memari, A. H., Ghanouni, P., Gharibzadeh, S., Eghlidi, J., Ziaee, V., & Moshayedi, P. (2013). Postural sway patterns in children with autism spectrum disorder compared with typically developing children. *Research in Autism Spectrum Disorders*, 7(2), 325–332.
- Memari, A. H., Ghanouni, P., Shayestehfar, M., & Ghaheri, B. (2014a). Postural control impairments in individuals with autism spectrum disorder: A critical review of current literature. *Asian Journal of Sports Medicine*, 5(3), e22963.
- Memari, A. H., Ghanouni, P., Shayestehfar, M., Ziaee, V., & Moshayedi, P. (2014b). Effects of visual search vs. auditory tasks on postural control in children with autism spectrum disorder. *Gait & Posture*, 39(1), 229–234.
- Minschew, N. J., Sung, K., Jones, B. L., & Furman, J. M. (2004). Underdevelopment of the postural control system in autism. *Neurology*, 63(11), 2056–2061.
- Molloy, C. A., Dietrich, K. N., & Bhattacharya, A. (2003). Postural stability in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 33(6), 643–652.
- Pellecchia, G. L. (2003). Postural sway increases with attentional demands of concurrent cognitive task. *Gait & Posture*, 18(1), 29–34.
- Riley, M. A., Baker, A. A., Schmit, J. M., & Weaver, E. (2005). Effects of visual and auditory short-term memory tasks on the spatiotemporal dynamics and variability of postural sway. *Journal of Motor Behavior*, 37(4), 311–324.
- Roelofs, K., Hagenaaers, M. A., & Stins, J. (2010). Facing freeze: Social threat induces bodily freeze in humans. *Psychological Science*, 21(11), 1575–1581.
- Shumway-Cook, A., & Woollacott, M. H. (2001). *Motor control: Theory and practical applications*. Philadelphia: Williams & Wilkins.
- Stins, J. F., & Beek, P. J. (2007). Effects of affective picture viewing on postural control. *BMC Neuroscience*, 8(1), 83.
- Stins, J. F., Emck, C., de Vries, E. M., Doop, S., & Beek, P. J. (2015). Attentional and sensory contributions to postural sway in children with autism spectrum disorder. *Gait & Posture*, 42(2), 199–203.
- Travers, B. G., Powell, P. S., Klinger, L. G., & Klinger, M. R. (2013). Motor difficulties in autism spectrum disorder: Linking symptom severity and postural stability. *Journal of Autism and Developmental Disorders*, 43(7), 1–16.

Effexor

- [Venlafaxine](#)

Effexor XR

- [Venlafaxine](#)

Efficacy

- [Self-Advocacy](#)

Efficacy of Group-Based Organized Physical Activity Participation for Children with Autism Spectrum Disorder

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Synonyms

[Group-based organized sport](#)

Definition

Physical activity is well recognized as movements produced by the body's skeletal and muscular system that require the expenditure of energy (World Health Organisation 2019). One subset of physical activity is group-based organized physical activity (OPA). This has been defined as physical activities typically involving formal and structured training sessions that are supervised by an adult coach (Okely 1999). This idea that OPAs are structured and planned in nature is consistent across Australian, UK, and US definitions. The

group-based aspect of this term also requires activities to involve two or more individuals (aside from the coach) engaging in the activity at once. These programs can include competitive team sports such as soccer or football and basketball or non-competitive programs such as group horse-riding and karate. Australian definitions also incorporate the idea that such activities are required to be organized by sporting bodies or recreational associations, for example, clubs and community associations (see Howells et al. 2019).

Importantly, group-based OPA and physical activity are not synonymous terms. Aside from increasing physical activity, group-based OPA and organized sports have been associated with added psychosocial benefits (Vella et al. 2016) above what an individual physical activity program such as walking can provide. This is particularly important for children with neurodevelopmental disorders such as autism spectrum disorder (ASD), where the salient features lie in social and communicative difficulties and individuals are at a heightened risk of psychiatric conditions such as clinically significant anxiety. The benefit to psychosocial domains following OPA, which is a movement-based activity, is unsurprising, not only because of the opportunity for social interaction with peers during the program but also because of the integration of neural circuitry between various developmental brain regions (e.g., frontal regions and the cerebellum). Hence, programs that develop motor skills also have the potential to benefit broader developmental areas such as social, cognitive, and behavioral functioning in children with ASD. Despite potential benefits, Canadian research shows children with neurodevelopmental conditions more generally often spend less time engaged in OPA, in comparison with TD children (Arim et al. 2012). Given this, it is imperative that children with ASD are provided with the same opportunities to participate in group-based OPA programs as their neurotypical peers, and research continues to explore factors which could hinder or facilitate participation in OPA in this group, alongside the potential benefits to participation. An increase in research may aid in the journey that sees these types of programs run in conjunction

with current clinical interventions for children with ASD. Moreover, while the potential benefits associated with physical activity more generally are broad ranging, participation in group-based OPA programs also provide children with ASD an opportunity to engage with peers and integrate into community activities. This may benefit not only the individual child but also their families, providing a sense of normalcy, and aid in attitudinal shifts at the community level.

See Also

- Exercise
- Sports

References and Reading

- Arim, R. G., Findlay, L. C., & Kohen, D. E. (2012). Participation in physical activity for children with neurodevelopmental disorders. *International Journal of Pediatrics*, 2012, 9. <https://doi.org/10.1155/2012/460384>.
- Howells, K., Sivaratnam, C., May, T., Lindor, E., McGillivray, J., & Rinehart, N. (2019). Efficacy of group-based organised physical activity participation for social outcomes in children with autism spectrum disorder: A systematic review and meta-analysis. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-04050-9>.
- Okely, A. D. (1999). *The relationship of participation in organised sports and games, participation in non-organised physical activity, and cardiorespiratory endurance to fundamental motor skill ability among adolescents*. Doctor of Education, University of Wollongong.
- Rinehart, N. J., Jeste, S., & Wilson, R. B. (2018). Organized physical activity programs: Improving motor and non-motor symptoms in neurodevelopmental disorders. *Developmental Medicine and Child Neurology*, 60(9), 856–857.
- Vella, S. A., Schranz, N. K., Davern, M., Hardy, L. L., Hills, A. P., Morgan, P. J., . . . Tomkinson, G. (2016). The contribution of organised sports to physical activity in Australia: Results and directions from the Active Healthy Kids Australia 2014 Report Card on physical activity for children and young people. *Journal Of Science And Medicine In Sport*, 19(5), 407–412. <https://doi.org/10.1016/j.jsams.2015.04.011>
- World Health Organisation. (2019). Physical activity. Retrieved 19 Aug 2019, from www.who.int/topics/physical_activity/en/

Efficacy of Peer Network Interventions

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Definition

Peer network interventions are a type of peer-mediated instruction/intervention in which peers are selected to form a network around a student with disabilities, and regularly scheduled opportunities for the network to interact are provided during noninstructional times (e.g., lunch, between classes, before school) with ongoing support from an adult facilitator (Carter et al. 2013). Social outcomes are the primary goals of peer network interventions, including increasing opportunities for students to meet new people, supporting friendship development, and encouraging students to engage in social activities during and outside of school (Carter et al. 2013, 2014). To date, peer network interventions have been implemented primarily with students with autism spectrum disorder (ASD) in an effort to address social and communication challenges.

Two to six peers (i.e., peer partners) are selected to join the student with ASD, and together, they create the peer network. The desirable traits of a peer partner include a positive peer model (e.g., demonstrates age-appropriate social skills) who is excited and expresses an interest in developing a relationship with the student with ASD (Carter et al. 2014). Peer partners should also express interest in staying involved throughout the duration of the intervention. Facilitators can be any adult who has a positive relationship with the student with ASD including but not limited to, a general or special educator, paraprofessional, special area educator (e.g., physical education), speech-language pathologist, counselor, or extracurricular teacher/coach.

The components of peer network interventions are flexible (Carter et al. 2019) but typically include the following core components:

(a) orienting the students to the network, (b) scheduling regular opportunities for students to interact, and (c) providing adult facilitation (Carter et al. 2013). Before weekly scheduled meetings begin, an orientation is held where the peer partners and student with ASD meet, get to know one another, share schedules, identify areas of common interest, and learn about peer network goals and how the peer network works (e.g., Sreckovic et al. 2017). During weekly meetings, the facilitator checks in with students to identify if they are interacting outside of weekly meetings (e.g., walking to class together) and encourages students to connect outside of meetings (Carter et al. 2013). The majority of the meeting is centered on a shared activity selected by the students, such as playing a game. The facilitator guides the meeting at the beginning of the intervention but slowly allows the students to take ownership over the meetings while still providing guidance when needed. For example, the facilitator may model how to engage the student with ASD in the activity or model how to use aided augmentative and alternative communication (AAC).

Historical Background

Individuals with ASD by definition have difficulty with social interaction and communication (APA 2013). These challenges, combined with segregated service delivery, close proximity of paraprofessionals and/or special educators, and peer attitudes and lack of understanding of ASD, make developing and maintaining positive peer relationships difficult (Carter 2018). Indeed, research consistently documents limited and low-quality peer relationships among students with ASD and their peers (Petrina et al. 2014), even though students with ASD have expressed interest in developing friendships with peers (Daniel and Billingsley 2010). This is concerning considering positive peer relationships make important contributions to success in school and overall quality of life (Rubin et al. 2009). On the contrary, peer rejection can lead to internalizing and externalizing behavioral problems (Ladd 2006)

and a myriad of psychological problems including loneliness (Buhs and Ladd 2001).

Examining the efficacy of social interventions to improve social deficits of individuals with ASD has been a long-standing focus in intervention research (White et al. 2017). While interventions to teach concrete social skills (e.g., social skills training) and communication skills (e.g., picture exchange communication) have been well established in the literature (Wong et al. 2015), less is known about the efficacy of generalizing those skills to natural environments, as well as how to create opportunities for individuals with ASD to develop positive peer relationships. Increased social skills do not necessarily equate to friendship development and greater social involvement in the school culture. In fact, in a nationally representative longitudinal study of the involvement of adolescent students with disabilities, students with ASD were reported as the least likely to receive telephone calls from friends, frequently see friends outside of school, and get invited to another student's social event (Wagner et al. 2004). Further, only 30% of the students with ASD participated in an organized group activity at school within the previous year (Wagner et al. 2004). Therefore, students with ASD may not fully participate in or benefit as much as they could be from the social aspects of school, such as establishing friendships and participating in group activities.

Given the low quality and quantity of reciprocal friendships and high bullying victimization rates among youth with ASD, identifying interventions that support increased social interaction and the development of positive peer relationships are now at the forefront of research (Winchell et al. 2018). Peer-mediated instruction/interventions naturally increase physical proximity between students with ASD and their peers as teachers systemically teach peers strategies for interacting with and/or helping students with ASD learn new skills (Wong et al. 2015). While peer-mediated instruction/interventions have been identified as an evidence-based practice with empirical evidence supporting social (birth to age 22) and academic (age 6–22) outcomes of youth and young adults with ASD (Wong et al.

2015), less is known about the efficacy of specific forms of peer-mediated instruction/interventions, such as peer network interventions.

Current Knowledge

Peer network interventions have been implemented in elementary (e.g., Kamps et al. 2014;), middle (e.g., Haring and Breen 1992; Koegel et al. 2012), and high (e.g., Gardner et al. 2014) school settings as a stand-alone intervention (e.g., Sreckovic et al. 2017) and as part of a multicomponent packaged intervention (e.g., direct instruction, scripted practice, peer-mediated free play, review of performance, and reinforcement; Kamps et al. 2014). The efficacy of peer network interventions has been examined using single case (e.g., Hochman et al. 2015), randomized controlled trial (e.g., Asmus et al. 2017), and quasi-experimental (Schmidt et al. 2017) research designs.

Across K-12 settings, peer network interventions have been efficacious at improving social outcomes for students with ASD with varying support needs, including students with complex communication needs (Biggs et al. 2018), students with significant support needs (Asmus et al. 2017), and students with ASD without comorbid ID (Sreckovic et al. 2017). Research indicates they have been implemented with high school students with ASD with diverse characteristics (e.g., autism symptomology, adaptive behavior, IQ scores) and education pathways (i.e., standard diploma, alternative diploma; Carter et al. 2019). Educators have implemented peer network interventions with fidelity and rated the interventions favorably, identifying them as feasible to implement, useful, and effective for their students with ASD and ID (Carter et al. 2019).

Across studies available in the literature, the components of peer network interventions maintain relatively consistent with core components including orienting students (e.g., preparing peer partners to effectively communicate with focal students), regularly scheduling opportunities for students to engage in a shared activity, and

incorporating adult facilitation. Yet, intervention procedures have been implemented flexibly to meet the needs of students and facilitators. The frequency of network meetings and length of intervention have also differed across studies. For example, participants in the study conducted by Asmus et al. (2017) met approximately once per week at least six times throughout the semester (Asmus et al. 2017), and participants in a study conducted by Sreckovic et al. (2017) met twice per week for 4 weeks. The efficacy of peer network interventions has been investigated with several outcome variables, all under the social/communication domain. These outcome variables include initiations and responses to/from peers/focal student (Haring and Breen 1992; Kamps et al. 1997, 2014, 2015; Koegel et al. 2012, 2013; Sreckovic et al. 2017), duration of social interaction among students (Garrison-Harrell et al. 1997; Kamps et al. 1997; Koegel et al. 2012, 2013), communicative acts toward peers (Mason et al. 2014), appropriate social responding, extended social interactions (Haring and Breen 1992), social interactions, social engagement, social-related goal, adult facilitative strategies (Gardner et al. 2014; Hochman et al. 2015), support behaviors from peer partners (Gardner et al. 2014), interaction quality (Haring and Breen 1992; Gardner et al. 2014), social contacts, friendship gains, social skills, school activity involvement (Asmus et al. 2017), proximity to peer partners, peers, adults (Biggs et al. 2018; Gardner et al. 2014; Hochman et al. 2015) and proximity to AAC device (Biggs et al. 2018), use of augmentative communication system (Garrison-Harrell et al. 1997), total number of words spoken, number of different word roots, length of conversational turn in reciprocal conversation, ratio of different words to total words (Schmidt et al. 2017), interactions with peers (symbolic and nonsymbolic) and nonprompted, symbolic communicative acts (Biggs et al. 2018), and bullying victimization (Sreckovic et al. 2017).

In a seminal peer network study, Haring and Breen (1992) investigated the efficacy of peer network interventions implemented with two junior high students (one student with ASD and

one student with ID and language delay). During the intervention, each focal student was grouped with four to five typically developing peers to form a network. Peer partners walked with the focal students in between classes, ate lunch with the focal students, and once a week the facilitator and network met to talk about times to interact, assess the group's satisfaction, and casually interact. Using a multiple baseline across participants design, the researchers measured social interactions, appropriate social responding, and extended social interactions. During intervention, the frequency of appropriate social interactions increased between the focal students and their peers. Further, both focal students participated in non-prompted events outside of school with their peer networks. One focal student's mother noted her son had been invited to an event outside of school by a peer for the first time. After the intervention, 89% of the peer partners rated focal students as a friend, and 11% rated focal students as a best friend. Subsequent work has replicated this study with the addition of treatment fidelity and demonstration of experimental control. Illustrations of studies across the K-12 continuum are included below.

Supporting evidence in elementary settings.

Several studies have examined the impact of peer network interventions on the social outcomes of young children with ASD. Kamps et al. (2015) investigated the impact of peer network interventions implemented with 95 kindergarten and first grade students with ASD. Four to six peers were selected from each focal student's class or a class within one grade level to serve as peer partners. The peers rotated throughout the week; two peer partners participated per meeting. The intervention consisted of direct instruction on a social skill (e.g., sharing and requesting), child-adult practice, peer-mediated free play (10–15 min of play with toys and games), and review of performance and reinforcement. The researchers employed a block randomization procedure by class to examine the impact of the intervention on total communication acts (i.e., initiations and responses) to peers, language performance, adaptive communication, and teacher impressions of children's social

communication. Children in the comparison group received instruction as indicated in their IEP and were not receiving structured peer-mediated social skill instruction. When compared to the control group, the implementation of the intervention resulted in statistically significant growth over time for initiations to peers during nontreatment social probes and generalization. Children with ASD were more likely to engage in more communicative acts the longer they were in the peer network intervention. At the end of first grade, no statically significant differences were found for initiations or responses across groups. Language performance and adaptive communication skills improved for all students over time, but those in the peer network group tended to see greater gains compared to the control group. Teacher's impressions of children's social communication also improved as a result of the peer network intervention. Overall, treatment fidelity averaged 86% with a range of 15–100%.

Using a small sample of participants from Kamps et al. (2014, 2015), used a multiple baseline across participants design to investigate the effects of peer network interventions implemented with four students with ASD (ages 6–7) on communicative acts to peers. The same intervention procedures were used as Kamps et al. (2015). Treatment fidelity averaged 84% across participants. Results demonstrated clear increases from baseline to intervention for all four participants for total communicative acts (i.e., initiations and responses) to peers, and Tau results indicated a statistically significant increase from baseline to implementation for total communicative acts. Generalization probes were higher in the intervention phase compared to the baseline phase for all three participants (generalization data were not gathered for one participant). Intervention implementers noted they felt the intervention was feasible and indicated they noticed improvements in focal students' interactions with trained and non-trained peers as reported on social validity forms.

While Kamps et al. (2014, 2015) implemented peer networks with students with ASD who demonstrated functional verbal

communication, peer network interventions have also been implemented with students with ASD who used aided AAC. Biggs et al. (2018) investigated the effects of a peer network intervention and an aided AAC modeling component using a multiple probe across participants design with two intervention phases. The interventions were implemented with three students with ASD and one student with ID who had significant cognitive impairment and aided AAC. Participants were in third or fourth grade, and 13 peers participated as peer partners. The peer network intervention consisted of an orientation meeting for the peer partners and focal students and peer network meetings where students participated in a shared activity held twice a week during lunch for three participants and during indoor recess for one participant. During the second phase of the intervention, a peer-implemented aided AAC modeling component was added to the peer network intervention. This consisted of an initial training meeting to teach peers how to use aided AAC modeling and coaching support for peers during at least two network meetings. Dependent variables were interaction with peers (i.e., symbolic and nonsymbolic communicative acts), symbolic communicative acts that were nonprompted, frequency of peers' interactions, frequency of peer-aided AAC models, modes of symbolic and nonsymbolic communication, and focal student proximity to peers, adults, and AAC device. Treatment fidelity averaged 96.1% across sessions. Results indicated a functional relationship between the implementation of the peer network and increased interactions between peers and the focal students. A functional relationship did not exist between the implementation of the peer network and nonprompted, symbolic communication to peers. When the peer-implemented aided AAC modeling component was added, nonprompted, symbolic communication increased, except for one participant whose peers used aided AAC modeling during the first intervention phase. Generalization data indicated peer interaction was higher than during pre-baseline probes and baseline phase but lower than during intervention.

Supporting evidence in secondary settings.

Peer network interventions have been implemented in secondary settings, which is both timely and needed as the prevalence of students with ASD in secondary settings now matches that of younger cohorts (Blumberg et al. 2013), and the social demands and expectations in middle and high school become more complex. Hochman et al. (2015) used a multiple baseline across participants' design to examine the efficacy of peer network interventions on social interactions, social engagement, social-related goal, proximity to peers and adults, and facilitator support behaviors. Four male students (age 15–17) with ASD and ID participated in the study as focal students, and 11 students participated as peer partners (2–3 peers per network). The intervention consisted of an orientation meeting for the peer partners and focal students and weekly meetings in the cafeteria. During weekly meetings, students ate, participated in a shared activity, talked about a selected topic, reflected on how meetings were going, shared ideas for new activities, discussed upcoming events they could do together, and reported interactions outside of weekly meetings. The implementation of the peer network intervention resulted in increased social engagement and peer interactions for all students, substantial gains related to the social-related goal for one student, and increased proximity to peer partners and decreased proximity to students with disabilities. Generalization data for both social engagement and peer interactions returned to near baseline levels for three out of four focal students. All peer partners and focal students considered each other as friends after the intervention. Treatment fidelity data were collected from an observer, coach, and facilitator. Social validity data indicated the facilitator and peer partners wanted to participate again and felt focal students benefited socially. Focal students and parents responded positively, and focal students wanted to be involved in the future.

Sreckovic et al. (2017) replicated Hochman et al. (2015) using similar intervention procedures but implemented the intervention with three high school students with ASD without comorbid

ID. Using a multiple baseline across participants with probe assessments during baseline design, the following dependent variables were investigated: initiations and responses from students with ASD to peers and from peers to students with ASD. Bullying victimization was also examined as an exploratory variable using the Bully Victimization Scale (Reynolds 2003) at four time points. Peer networks consisted of one student with ASD and three to six peer partners. All students participated in an orientation meeting (description of what a peer network is) and introduction meeting (a time for students to meet, exchange schedules, and identify common interests). Each network met approximately twice per week during lunch in an empty classroom or conference room. During weekly meetings, the facilitator encouraged students to connect outside of meetings, informally assessed student satisfaction, and provided guidance as needed as students played a game of their choice. A functional relationship was demonstrated between the implementation of the peer network intervention and total social interaction (i.e., initiations and responses from students with ASD to peers and from peers to students with ASD). Maintenance data were similar to intervention for all participants, and mean responses to and from students with ASD during generalization were greater than baseline for all three participants. Bullying victimization decreased for two participants, although changes were minimal for one student. Treatment fidelity data for the facilitator averaged 100% across sessions, and 98% across sessions for peer partners. All peer partners, parents, and school staff rated the intervention favorably, and two focal students rated intervention favorably.

Using a randomized controlled trial design, Asmus et al. (2017) examined the efficacy of peer network interventions implemented with 95 high school students with severe disabilities and 192 peer partners (3–6 partners per network). Adolescents with severe disabilities qualified for the state's alternate assessment and/or received special education supports under the category of ID or ASD and were educated for at least one class in general education settings. Twenty-one high

schools across two states were included in the study. Intervention procedures were similar to those in Hochman et al. (2015) and Sreckovic et al. (2017); however, a peer partner training was held without the focal students. Networks met approximately once per week and participated in a shared activity (e.g., meal, game) and planned times throughout the week to connect. In the comparison condition, students were included in at least one general education class with support from a paraprofessional or special educator and received services as stated in their individualized educational programs. Social contacts and friendship gains, social skills, and school activity involvement were examined. Treatment fidelity data averaged 95.9%. Results indicated students in the intervention group reported meeting outside of weekly meetings with the focal student an average of 11.4 times. Students who participated in the intervention had significantly more social contacts throughout the day and gained significantly more friends without disabilities by the end of the semester compared to students in the comparison group. No significant effects were found on parent-reported measures of out of school social connections. During the following semester, 33% of the peer partners reported a social contact with the focal student, and 41% reported they were friends. Teacher reported friendships and social contacts between students with severe disabilities, and those without disabilities at one semester follow-up were significantly higher for students in the intervention condition compared to the comparison condition. Further, students in the peer network condition had an average of 1.51 more social contacts and an average of 1.35 more friends than their counterparts in the comparison condition. No significant differences were found at two semesters follow-up. No significant differences were found for parent-reported social contacts and friendships at one or two semesters follow-up or for school activity involvement. Based on the evidence at both the elementary and secondary levels, peer network interventions are empirically supported as efficacious in improving social and communication outcomes.

Future Directions

All students, with or without ASD, should benefit from both the academic and social opportunities embedded in authentic educational settings. Implementing interventions to facilitate social interactions and friendship development is imperative to help students with ASD succeed socially and benefit from the positive consequences associated with supportive peer relationships (e.g., Rubin et al. 2009). A review of the literature highlights the positive social outcomes (e.g., increased social interaction and friendships with peers without disabilities) that result from implementing peer network interventions and areas of future research. As the efficacy base continues to expand, future research teams are encouraged to consider the following research recommendations.

First, peer network interventions consist of several components that are flexible to meet the needs of the focal student. Research teams are encouraged to conduct component analyses to examine the extent to which each component is necessary to yield desired outcomes. Further, intervention length ranged across studies (e.g., 1 month, one semester; one meeting per week, three meetings per week). Research is needed to determine optimal intervention intensity and dosage (e.g., recommended length of intervention and optimal meeting contexts) that result in desirable and generalizable outcomes.

Second, while several studies included generalization probes, these findings varied. More research is needed on how to maintain impact over time. Further, more research is needed on the long-term outcomes of peer network interventions. Longitudinal studies are needed to determine effects at 1-year follow-up. Similarly, more research is needed on friendship development both within and outside of the peer network. Do students with ASD develop reciprocal friendships with their peer partners and with other peers and does their social network centrality change after the peer network intervention is introduced?

Finally, research teams are encouraged to examine the impact of peer network interventions

beyond the K-12 education setting. Peer networks may be efficacious in work settings to increase sustainability in employment, in post-secondary education settings to increase participation in campus life, and in community settings to increase community engagement and overall quality of life.

In summary, the field has greatly expanded with regard to peer network interventions for youth and adolescents with ASD. The intervention has proven to be flexible, feasible to implement, and efficacious at improving social/communication outcomes for students with ASD across the K-12 continuum. Without targeted interventions and intentional efforts to foster meaningful relationships for students with ASD, students with ASD are unlikely to develop relationships (Carter et al. 2008). Therefore, implementing targeted interventions, such as peer network interventions, is imperative to support the social success of students with ASD.

See Also

- [Peer-Mediated Intervention](#)
- [Social Interventions](#)

References and Reading

- American Psychiatric Association. (2013). *American Psychiatric Association: diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association. Accessed 1 Aug 2019. dsm.psychiatryonline.org.
- Asmus, J. M., Carter, E. W., Moss, C. K., Biggs, E. E., Bolt, D., Born, T. L., . . . Wier, K. (2017). Efficacy and social validity of peer network interventions for high school students with severe disabilities. *American Journal on Intellectual and Developmental Disabilities*, 122, 118–137. <https://doi.org/10.1352/1944-7558-122.2.118>.
- Biggs, E. E., Carter, E. W., Bumble, J. L., Barnes, K., & Mazur, E. L. (2018). Enhancing peer network interventions for students with complex communication needs. *Exceptional Children*, 85(1), 66–85. <https://doi.org/10.1177/0014402918792899>.
- Blumberg, S. J., Bramlett, M. D., Kogan, M. D., Schieve, L. A., Jones, J. R., & Lu, M. C. (2013). Changes in prevalence of parent-reported autism spectrum disorder in school-aged U.S. children: 2007 to 2011–2012. *National Health Statistics Reports*, 65, 1–12.
- Buhs, E. S., & Ladd, G. W. (2001). Peer rejection as an antecedent of young children's school adjustment: An examination of mediating processes. *Developmental Psychology*, 37, 550–560. <https://doi.org/10.1037/0012-1649.37.4.550>.
- Carter, E. W. (2018). Supporting the social lives of secondary students with severe disabilities: Considerations for effective intervention. *Journal of Emotional and Behavioral Disorders*, 26(1), 52–61. <https://doi.org/10.1177/1063426617739253>.
- Carter, E. W., Sisco, L. G., Brown, L., Brickham, D., & Al-Khabbaz, Z. A. (2008). Peer interactions and academic engagement of youth with developmental disabilities in inclusive middle and high school classrooms. *American Journal on Mental Retardation*, 113, 479–494. <https://doi.org/10.1352/2008.113.479-494>.
- Carter, E. W., Asmus, J., Moss, C. K., Cooney, M., Weir, K., Vincent, L., . . . Fesperman, E. (2013). Peer network strategies to foster social connections among adolescents with and without severe disabilities. *Teaching Exceptional Children*, 46(2), 51–50. <https://doi.org/10.1177/004005991304600206>.
- Carter, E., Redding, J., Bottema-Beutel, K., Fan, H., Gardner, K., Harvey, M., . . . Stabel, A. (2014). *Peer network facilitator manual*. The Center on Secondary Education for Students with Autism.
- Carter, E. W., Steinbrenner, J. R. D., & Hall, L. J. (2019). Exploring feasibility and fit: Peer-mediated interventions for high school students with autism spectrum disorders. *School Psychology Review*, 48(2), 157–169. <https://doi.org/10.17105/SPR-2017-0112.V48-2>.
- Daniel, L. S., & Billingsley, B. S. (2010). What boys with an autism spectrum disorder say about establishing and maintaining friendships. *Focus on Autism and Other Developmental Disabilities*, 25, 220–229. <https://doi.org/10.1177/1088357610378290>.
- Gardner, K. F., Carter, E. W., Gustafson, J. R., Hochman, J. M., Harvey, M. N., Mullins, T. S., & Fan, H. (2014). Effects of peer networks on the social interactions of high school students with autism spectrum disorders. *Research and Practice for Persons with Severe Disabilities*, 39(2), 100–118. <https://doi.org/10.1177/1540796914544550>.
- Garrison-Harrell, L., Kamps, D., & Kravits, T. (1997). The effects of peer networks on social-communicative behaviors for students with autism. *Focus on Autism and Other Developmental Disabilities*, 12, 241–254. <https://doi.org/10.1177/108835769701200406>.
- Haring, T. G., & Breen, C. G. (1992). A peer-mediated social network intervention to enhance the social integration of persons with moderate and severe disabilities. *Journal of Applied Behavior Analysis*, 25, 319–333. <https://doi.org/10.1901/jaba.1992.25-319>.
- Hochman, J. M., Carter, E. W., Bottema-Beutel, K., Harvey, M. N., & Gustafson, J. R. (2015). Efficacy of peer networks to increase social connections among high school students with and without autism.

- Exceptional Children*, 82, 96–116. <https://doi.org/10.1177/0014402915585482>.
- Kamps, D. M., Potucek, J., Lopez, A. G., Kravits, T., & Kemmerer, K. (1997). The use of peer networks across multiple settings to improve social interaction for students with autism. *Journal of Behavioral Education*, 7(3), 335–357. <https://doi.org/10.1023/A:1022879607019>.
- Kamps, D., Mason, R., Thiemann-Bourque, K., Feldmiller, S., Turcotte, A., & Miller, T. (2014). The use of peer networks to increase communicative acts of students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 29, 230–245. <https://doi.org/10.1177/1088357614539832>.
- Kamps, D., Thiemann-Bourque, K., Heitzman-Powell, L., Schwartz, I., Rosenberg, N., Mason, R., & Cox, S. (2015). A comprehensive peer network intervention to improve social communication of children with autism spectrum disorders: A randomized trial in kindergarten and first grade. *Journal of Autism and Developmental Disorders*, 45, 1809–1824. <https://doi.org/10.1080/17489539.2015.1113648>.
- Koegel, R. L., Fredeen, R., Kim, S., Danial, J., Rubinstein, D., & Koegel, L. (2012). Using perseverative interests to improve interactions between adolescents with autism and their typical peers in school settings. *Journal of Positive Behavior Interventions*, 14(3), 133–141. <https://doi.org/10.1177/1098300712437043>.
- Koegel, R., Kim, S., Koegel, L., & Schwartzman, B. (2013). Improving socialization for high school students with ASD by using their preferred interests. *Journal of Autism and Developmental Disorders*, 43(9), 2121–2134. <https://doi.org/10.1007/s10803-013-1765-3>.
- Ladd, G. W. (2006). Peer rejection, aggressive or withdrawn behavior, and psychological maladjustment from ages 5 to 12: An examination of four predictive models. *Child Development*, 77(4), 822–846. <https://doi.org/10.1111/j.1467-8624.2006.00905.x>.
- Mason, R., Kamps, D., Turcotte, A., Cox, S., Feldmiller, S., & Miller, T. (2014). Peer mediation to increase communication and interaction at recess for students with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 8, 334–344. <https://doi.org/10.1016/j.rasd.2013.12.014>.
- Petrina, N., Carter, M., & Stephenson, J. (2014). The nature of friendship in children with autism spectrum disorders: A systematic review. *Research in Autism Spectrum Disorders*, 8(2), 111–126. <https://doi.org/10.1016/j.rasd.2013.10.016>.
- Reynolds, W. M. (2003). *Reynolds bully-victimization scales for schools*. San Antonio: The Psychological Corporation.
- Rubin, K. H., Bukowski, W. M., & Laursen, B. (Eds.). (2009). *Handbook of peer interactions, relationships, and groups*. New York: Guilford Press.
- Schmidt, C., Schmidt, M., Kamps, D., Thiemann-Bourque, K., & Mason, R. (2017). Pilot investigation of language development of children with autism receiving peer networks intervention. *Journal on Developmental Disabilities*, 23(1), 3–17. https://doi.org/10.1007/978-1-4419-1698-3_500.
- Sreckovic, M. A., Hume, K., & Able, H. (2017). Examining the efficacy of peer network interventions on the social interactions of high school students with Autism Spectrum disorder. *Journal of Autism and Developmental Disabilities*, 47(8), 2556–2574. <https://doi.org/10.1007/s10803-017-3171-8>.
- Wagner, M., Cadwallader, T. W., Garza, N., & Cameto, R. (2004). Social activities of youth with disabilities. *NLTS2 Data Brief*, 3(1), 1–4.
- White, S. W., Wieckowski, A. T., & Maddox, B. B. (2017). Social interventions. In: *Encyclopedia of Autism Spectrum Disorders* (pp. 1–5). https://doi.org/10.1007/978-1-4614-6435-8_109-3.
- Winchell, B. N., Sreckovic, M. A., & Schultz, T. R. (2018). Preventing bullying and promoting friendship for students with ASD: Looking back to move forward. *Education and Training in Autism and Developmental Disabilities*, 53(3), 243–252.
- Wong, C., Odom, S. L., Hume, K., Cox, A. W., Fettig, A., Kucharczyk, S., . . . Schultz, T. R. (2015). *Evidence-based practices for children, youth, and young adults with Autism Spectrum Disorder*. Chapel Hill: The University of North Carolina, Frank Porter Graham Child Development Institute, Autism Evidence-Based Practice Review Group.

Efficiency (Behavioral Assessment)

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Definition

Efficiency refers to the principle that responses which require less effort and produce more reinforcement will occur more frequently than responses that require more effort and produce the same or less reinforcement. Responses that take less effort and produce a similar level of reinforcement as more effortful responses are considered to be more efficient. To increase the likelihood that a desired behavior will occur, the environment or other antecedents can be

manipulated to increase the efficiency of the response (decrease the response effort), therefore making the response more likely. To decrease the likelihood that an undesirable behavior will occur, the efficiency of that response should be decreased (increase response effort), therefore making it more likely that the individual will engage in competing behaviors that require less effort.

See Also

► [Response Effort](#)

References and Reading

Miltonberger, R. G. (2004). *Behavior modification principles and procedures* (3rd ed.). Belmont: Wadsworth/Thomson Learning.

Effort (Behavioral Assessment)

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Synonyms

[Response effort](#)

Definition

Response effort is the amount of effort that a person must put forth to successfully complete a specific behavior. The amount of effort required to complete the behavior has an impact on the frequency at which that behavior will be emitted. If two responses result in fairly equal reinforcement, the behavior that requires less response effort is more likely to occur than the behavior that requires more response effort.

References and Reading

Miltonberger, R. G. (2004). *Behavior modification principles and procedures* (3rd ed.). Belmont: Wadsworth/Thomson Learning. Retrieved from <http://www.olemiss.k12.in.us/intervention/behavior/respeffort.pdf>

Egypt and Autism

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Historical Background

Egypt is located in North Africa, bordering the Mediterranean Sea, between Libya and the Gaza Strip. Genetic diseases in Egyptians have been known since the days of the pharaohs as recorded in the ancient history. Gene flow to Egyptian population occurred from Greco-Roman, Arab, Turkish, French, and English settlers. For reasons of custom, tradition, culture, and socioeconomic benefit, consanguineous marriages occur in approximately half, and first-cousin marriages occur in *one third of Egyptians* (Yamamah 2012). The population of the region is characterized by large family size, high maternal and paternal age, and a high level of inbreeding with consanguinity rates. Women are continuing to bear children until menopause increasing the predisposition to inherited disorders.

Developing countries are facing an alarming gap between innovations in childhood learning and developmental disabilities (LDD) research and their delivery to communities. Important problems include illiteracy (especially among women), lack of job opportunities (especially for young people), and slow economic growth because of loss of traditional economies, low productivity, and lack of innovation and competitiveness.

There is a critical gap in autistic spectrum disorder (ASD) research with respect to manifestations of the condition in developing countries.

This gap is now attaining public health significance. On one hand, they prioritize the control and eradication of infectious diseases and reproductive maternal health. On the other hand, LDD research receives scant attention and almost no funding from donor agencies that specialize in addressing the problems of developing nations.

While Egypt has shown considerable progress in the prevention and combating of infectious diseases, genetic disorders remain a major health problem. There are no comprehensive data on disabilities currently available. The importance of medical genetics in pediatric departments of Egyptian universities was well appreciated in the early 1960s at Cairo and Ain Shams Universities in Egypt. At 1966, Prof. Samia and her colleagues started the specialty of human genetics at the National Research Centre (NRC) followed by molecular genetics at 1985 (El Awady and his colleagues at the NRC). In 1967, medical genetics unit (Prof. Suzan Rushdy and her colleagues) was established at the Medical Research Institute in Alexandria. This was followed by the initiation of medical genetics units in other universities such as El-Mansoura (Prof. Mohamed Hafez). Mubarak City of Scientific Research encompasses centers for frontier sciences including genetic engineering and biotechnology. Human genetic courses are now introduced in the curriculum of medical students in most universities. Specialized postgraduate degrees in the field of medical human genetics are offered to graduates from medical schools in Egypt at Ain Shams and Alexandria Universities. Training programs given by specialized geneticists from different institutions are offered to physicians from the Ministry of Health and Population.

On the other hand, Egypt has 19 medical schools; all of them have psychiatric departments, some of which are departments of neuropsychiatry. They have offered a master's degree in psychiatry for the past 25 years and a doctorate for the past 20 years. In general, there are few psychiatrists specializing in childhood problems (Okasha 2004). Nowadays, there are psychiatrists and psychologists specializing in the mental health needs of people with mental retardation. This is the case in Egypt where people with mental retardation

and mental disorders are seen by general psychiatrists, pediatricians, and other professionals without specialist knowledge of their problems. Prevalence of autism spectrum disorders among children with developmental disorders in Egypt was documented to be 33.6 % (Seif eldin et al. 2008). This preliminary finding needs to be corroborated and extended, in conjunction with better descriptions of normal early childhood development. There is a need for both community and school-based studies on epidemiology of autism spectrum disorders in Egypt.

The pioneer works on autism spectrum disorders in Africa had been by Longe and Asuni (Longe and Asuni 1972) and Lotter (1978, 1980) about three decades after the first report of autism spectrum disorder by Kanner in 1943 (Kanner 1943). With the rise in the prevalence of autism spectrum disorders (ASD) in the Arab countries, the development of an Arabic tool for early diagnosis and intervention was sought as part of an effort to better understand the prevalence of this disorder. The Modified Checklist for Autism in Toddlers (M-CHAT) was chosen (Seif Eldin et al. 2008).

Many factors may contribute to the substantial underdiagnosis of ASD cases in developing countries. One of the factors is the difficulty in obtaining a diagnosis, as the pediatricians are often inexperienced in the diagnosis and management of these disorders. The lack of awareness among parents regarding ASD, including a failure to recognize symptoms and seek diagnosis and treatment, is also likely to be a factor, especially in cases of children with mild forms of the disorder (Al-Farsi et al. 2010). Most of the children diagnosed with autism belonged to families of low socioeconomic standards with unsatisfactory income, in Egypt and Jordan, along with a lower paternal education level (Mostafa et al. 2012).

Legal Issues, Mandates for Service

The condemning of women who are obligated to continue her pregnancy with disabled outcome is indicative of our society's prejudice against disabled people and women. Egyptian women were

expected to stay at home, take care of the children, and let their husband bring home the paycheck. Most of them were dominated by the man of the house, providing whatever the man dictated, food on the table when they come home, house cleaned, kids taken care of, and anything else handled. Egyptian women suffer from various forms of stigma manifested during marital and social events, schooling, and employment. The stigma linked with being mothers of a disabled child is so deeply rooted that it plays a central role in many aspects of their life. Many mothers felt they had “lost” aspects of their personal identity, with the role of motherhood dominating how they felt about themselves and how other people viewed them. One of the most influential factors in those cases is the socioeconomic constraint. When both parents have a good socioeconomic state, they have a different level of thinking and can rationalize the fact it is nobody’s fault their child was born with special needs, and they believe there are several ways to help him/her and support him/her, and most probably, they do have few means to help supporting him/her and start to isolate themselves away from their friends and other family members. The cases where the father decides to leave mostly show (in all the cases I’ve seen) that the parents are not highly educated and some have a rather low financial status. The father then can easily blame his wife for this child and simply leave them to start a new life elsewhere with new wife, leaving them with no financial support and certainly no emotional support.

As a challenge to the family, autism must rank among the most stressful of childhood developmental disabilities. The autistic disorders require the practitioner to work with different types of subjects, including the patient, the parents and other caregivers, those working in the school system, and other professionals involved in mental health. In our current understanding of the phenomena of identical gene copies for children of first-cousin marriages, will inherit identical gene from each parent in 1/16 of their gene loci ($F = 0.0625$), giving birth to disabled children. However, the prevailing culture and ignorance lead the society and husbands to blame the women who

bear disabled children because of this. The condemning of women who are obligated to continue her pregnancy with disabled outcome is indicative of our society’s prejudice against disabled people and women. Also, the burden of care is a matter of socio-structural constraints rather than only emotional distress.

In developing countries, basic needs such as nutrition, water, and sanitation often overshadow mental health concerns. In many developing countries, up to half of the population is children (Hong et al. 2004; Robertson et al. 2004). The difficult circumstances found in many countries violate the basic rights of children (Robertson et al. 2004).

The government of Egypt places a high priority on disability, with governmental and non-governmental organizations working together to solve disability issues. However, current services cover only 10% of the total number of persons with disabilities. Services in Egypt are still limited in spite of the new policy of deinstitutionalization and the provision of community care. Community care in the form of hostels, day centers, rehabilitation centers, and health visitors is only available in major cities. Mental health legislation was introduced in Egypt in 1944, in advance of most other Arab and African countries (Okasha 2004).

The chronic nature of autism can be draining to some parents, and there is a risk of parents becoming exhausted. Attitudes of the two parents toward the female were more negative. According to the 1975 Rehabilitation of Disabled Persons Act, any type of business that has over 50 employees must include at least 5% of people with physical or mental disabilities as part of their total workforce. Although on paper people with autism are supposed to be included, but much like with all other mental and physical disabilities, the reality is from what the law stipulates.

The Ministry of Education issued a minstrel decision in April of 2009 to include 10% of children with special needs in mainstream schools, with no more than four of those children in each class. Those 10% are around 152, 800 children out of an estimated seven million people born with disabilities in Egypt. However, there are not enough trained professionals to deal with

people with disabilities in general, let alone with a complex disorder such as autism. Launching our own awareness campaign against the stigma associated with mental illness should be addressed.

Special schools for intellectually disabled: These schools are free of charge and are situated in the major cities in the greater Cairo area. They accept students with mild intellectual disability. Also, some private schools have opened classrooms in their schools to serve intellectually disabled children. Those schools charge high fees and usually accept mild cases. The high prices of most of the services cannot be afforded by many families. However, there is an unfair geographical distribution of services provided to intellectually disabled individuals. Most of the services are situated in Cairo, although 64 % of the mentally challenged live in rural areas and countryside. We have shortage of professional manpower such as specialized doctors, nurses, psychiatrists, psychotherapists, teachers, etc. which is a major barrier against the spread of the rehabilitation programs in Egypt.

Parents having an autistic child will pass through several coping stages, namely, denial, anger, and guilt, bargaining, and finally acceptance; they must deal with the grief they feel over absence of healthy imagined child (King et al. 2006). Coping is a process of readjusting attitude, feeling, perception, and beliefs about self and others, while coping style is a particular coping mechanism used repeatedly by a person in different situations. Among Egyptians, maladaptive coping strategies are mechanisms used to reduce a person's emotional response to stressful circumstances in a short term, but lead to greater difficulties in the long term; moreover, the treatment services available for late adolescents and adults with autism are quite limited.

Epidemiological data are important for the development of public policy and programs to improve children's mental health. Epidemiological research answers the following questions (Leibson et al. 2001): How many children in the community have mental health problems? How many children make use of mental health services? What is the distribution of mental health

problems and services across age, sex, and ethnic group? Are there historical trends in the frequency of child mental health problems? What is the developmental course of mental health problems from childhood to adulthood? What etiological factors can be identified to inform the design of prevention and treatment programs? How cost-effective are child mental health services? What are the outcomes for children who receive services? The answers to these questions provide a rational basis for service design and implementation.

Overview of Current Treatments and Centers

Epidemiological surveys are viewed as a priority that extends beyond the need for objective and robust estimates of prevalence. These provide additional valuable benefits as they often result in systematic information regarding existing services and may help in assessing the needs and priorities for Egyptian community. Epidemiological studies themselves may also eventually provide revealing findings regarding the manifestation of PDD across cultures.

A review of global [prevalence](#) of ADS by Elsabbagh et al. (2012) reported a median of 62 cases per 10,000 people with average 4.3:1 male-to-female ratio. There is a lack of evidence from low- and middle-income countries. We could not identify published data on population-based estimates of prevalence of PDD from Egypt.

Level of knowledge and awareness about autism spectrum disorder is low among the general population and health care workers in Egypt. There is need for community education of the general population and continuous medical education for health care workers on issues relating to autism spectrum disorder. This would enhance early recognition and interventions and in turn improve prognosis. The mothers of autistic children are vulnerable to psychological distress; families have been burdened by financial concerns, worries about health of autistic children (Elbahnasawy and Girgis 2011).

Cohen and Volkmar (2005) reported that parents typically are active partners in their children's education to ensure that skills are learned in the educational program; they transfer to the home setting and teach their children many behaviors that are best mastered in the home and community. Regional variation in parental education may still determine their help seeking for autism (Seif Eldin et al. 2008) and that it is predominantly severe cases that are currently being identified in developing countries like Egypt and more specifically in poor communities. We recommend that continuous health education and counseling programs are necessary to improve mothers' coping patterns toward care of their autistic children. Feeling skilled and informed is needed to mother's sense of competence, their ability to make the right decisions, and their ability to support their child's well-being and development to lessen their stigma.

There are more than 50 Egyptian Association of Autism – Egyptian schools (egyptian-schools.com/en/schools/egyptian-association-of-autism/). Examples are Autism Solutions (www.autismaspirations.com/), Autism: Preschool (readingroom.mindspec.org/), and Autism Is Treatable (www.tacanow.org/).

The purpose of these schools is to provide helpful information about autism and tools and strategies to achieve positive interactions and increase learning for all members of the school community. They tried to provide valuable information for general education and administrative school staff, aides, office staff, bus drivers, nurses, custodians, classmates, and family members who interact with students with autism. Most of the successful schools and associations are nongovernmental and of expensive costs, so in fact services are often “paid out of pocket.” The Ministry of Health is urged to launch a comprehensive program to improve the status of autistic children in Egypt. Recently, teachers and support personnel in schools sporadic in the Egyptian community have been recognized and increased (Abdel et al. 2012). However, most of them are not qualified, some are mothers of autistic children, and others attended training workshops supervised by unqualified

nonspecific personnel, which are more a myth than a reality. School-based consultation services for child mental health do not operate regularly to the extent required in developing countries. This gap leads to a failure to prevent school dropout and other significant consequences.

Overview of Research Directions

Of the 84.6 million people living in Egypt, 29 % are children under age 15. Until recently, identification and treatment of child mental health problems have not been a high priority in Middle East countries.

Although autism occurs in all cultures and countries, most of the published researches have come from Western countries. Little is known about its clinical correlates and comorbidity in Middle Eastern and Arab countries (Al-Salehi et al. 2009). Publications on child psychiatry, in particular, on topics such as autism were found to be underrepresented among Egyptian population. Very low research is yet to investigate the **effects of culture on autism**; effect of culture among Egyptian and Saudi population suffering autism on the age of diagnosis and seeking for intervention of autism was studied by Hussein et al. (2011). They concluded that the age at diagnosis and at the commencement of intervention was lower in the Egyptian group. The Saudi group showed a higher percentage of missing examinations, older birth order, and significantly higher preference to drug treatment, while the Egyptian group showed a high preference to behavioral and phoniatric therapies, higher paternal and maternal education, higher employment among parents, and higher family concern. They highlight that cultural context may significantly influence the age of detection and the age of starting the intervention.

There are few studies investigating the parents' attitudes toward their autistic child (Obaid 2012). The medical community in the Middle East needs to redouble its efforts to ensure early diagnosis and the best intervention in children with autism. In Egypt and North Africa, there has been a

fivefold decrease in child mortality in recent decades. This success has also come with a push to improve the quality of life for children and foster their development during the early years of life. This includes recognition of autism, a condition long hidden in much of the Arab world.

Hussein et al. (2012) reported that most parents first sought the advice of pediatricians for their child's mental health problem, and a substantial number consulted traditional healers. They recommend that awareness programs targeting pediatricians and elementary school teachers are urgently needed in Egypt to shorten the duration of undiagnosed illness among children.

The Arab world lacks centers to collect and organize Arab medical documents and to disseminate the available information.

A study on increasing people awareness with the aid of health team professionals in prompting tolerance and understanding of autism was done by Elbahnasawy and Girgis (2011). On the other hand, studying the environmental risk factors of autism was reviewed by El Baz et al. (2011). They reported that low birth weight and delivery by caesarian section were significantly higher among cases than controls. In addition to hypoxia, resuscitation and history of jaundice were considered significantly risk factors for autism.

Nutritional assessment and complementary intervention in autistic children represent one aspect for researchers in Egypt. A pilot study done by Allam et al. (2008) was done investigating the effect of scalp acupuncture and language in autistic children. They find out some improvement in expressive language when combined with language therapy. Meguid et al. (2010a) reported an article investigating the potential role of vitamin D in autism through serum level assessment. They concluded that serum values of 25(OH)D in the children with autism of this study could classify them as being "vitamin D inadequate," which lends support to the hypothesis that autism is a vitamin D deficiency disorder. Another study by Ibrahim and El-Sayed (2012) discussed the role of dietary imbalances in the incidence of developmental disorders mainly conditions considered as leading

causes for abnormal nervous system development and altered function. They emphasized the need of dietary supplements. Iron metabolism alteration as a risk factor for several neurodegenerative disorders was investigated by Gebril and Meguid (2011). They reported on a pilot study for the association of HFE polymorphisms and autism. They did not find association of autism with the genes they worked on and recommended to include other iron metabolism genes.

While many aspects of autism remain poorly understood, major advances have been made in terms of **highlighting the genetic part** in advanced countries (Abrahams and Geschwind 2008) and few scanty publications from Egypt (Novarino et al. 2012).

Till date, research in autism in Egypt is sporadic regarding different issues:

Different studies on **the brain of autistic children** were done; a study to assess white matter integrity of autistic preschool children with diffusion-weighted MR imaging was done by Abdel Razek et al. (2013). The study was carried out on 19 autistic preschool children. They reported that the apparent diffusion coefficient value is helpful in the assessment of the integrity of the white matter in autistic preschool children and well correlated with CARS score, social age, and language age. Another study was done by Mostafa et al. (2008) where they proposed that autoimmunity to brain could play an etiopathogenic role in autistic patients. The frequency of serum anti-myelin-associated glycoprotein antibodies, as an index for autoimmunity to brain, and their relation to family history of autoimmunity were investigated in 32 autistic and 32 healthy matched children. They shed light on the etiopathogenic role of anti-myelin-associated glycoprotein antibodies and the role of immunotherapy in autism. The third Egyptian study regarding brain morphology of autistic children was done by Meguid et al. (2010b). Fragile X syndrome shares most of the behavioral phenotypic similarities with autism. How are these similarities reflected in

brain morphology? A total of ten children with autism and seven with fragile X syndrome underwent morphological (T1) 1.5-T magnetic resonance imaging (MRI). The authors found no significant difference in total brain volumes, regional volumes, gyrification index, sulcal depth, and cerebral cortical thickness. However, children with autism showed significant decrease in the medial prefrontal bilaterally and the left anterior cingulate cortices. Regression analysis revealed positive correlation between the medial prefrontal cortical thickness and the social IQ. The authors suggest that the difference between the two groups in the medial prefrontal and anterior cingulate cortices thickness may entail an altered social cognitive style. Functional MRI studies directly differentiating between social indifference (autism) and social avoidance (fragile X syndrome) are needed to further characterize the spectrum of social abnormalities between these two groups. Future functional MRI studies, with their noninvasive in vivo capacity in measuring the social behavior phenotypes and in correlation with the FMR1 gene, are needed to advance the efforts to elucidate the genetic and neural mechanisms underlying social behavior disturbances in fragile X syndrome and autism.

Meguid et al. (2007) found that the prevalence of fragile X syndrome among Egyptian males is 0.9:1,000, constituting for 6.4% among mentally subnormal males.

The prevalence of ADS among Eastern Mediterranean is as follows: 29/10,000 in the United Arab Emirates (Eapen et al. 2007), 1.4/10,000 in Oman (Al-Farsi et al. 2010), and 6.3/10,000 in Iran (Samadi and McConkey 2011).

The relatively low prevalence in the Omani and Iranian studies was attributed to prevalence caused by underdiagnosis and underreporting of cases resulting from limited access to service centers (Al-Farsi et al. 2010).

Defective antioxidant enzymes and increased lipid peroxidation in Egyptian children with autism have been studied and reported by Meguid et al. (2011).

Neuroimmunological Aspect of Autism

Immunological disorders are one of several contributing factors that have been suggested to cause autism. Kawashti et al. (2006) detected circulating IgA and IgG autoantibodies to casein and gluten dietary proteins done by enzyme immunoassays (EIA). Circulating IgG antibodies to measles, mumps, and rubella vaccine (M.M.R) and cytomegalovirus were also investigated by EIA in a sample of 30 autistic Egyptian children. They concluded that autoimmune response to dietary proteins and deficient immune response to measles, mumps, and rubella vaccine antigens might be associated with autism, as a leading cause or a resulting event. Further research is needed to confirm these findings.

Mostafa and Kitchener (2009) studied the frequency of antinuclear antibodies in 80 Egyptian autistic children and their relationship to a family history of autoimmunity. They found out that antinuclear antibody seropositivity had significant positive associations with disease severity, mental retardation, and electroencephalogram abnormalities. The study recommended that autoimmunity may play a role in a subgroup of children with autism. Another study by Mostafa and Shehab (2010) investigates the link of C4B null allele to autism and to a family history of autoimmunity in Egyptian autistic children. They concluded the possible link of C4B null allele to autism and to a family history of autoimmunity highlighting the possible contributing role to autoimmunity in autism.

Recently, the link between some alleles on human leukocyte antigen system and autistic children was investigated by Mostafa et al. (2013). They highlighted the probable protective association of HLA-DRB1*03 allele with autism. It warrants a replication study of a larger sample to validate the HLA-DRB1 genetic association with autism. This is important to determine whether therapeutic modulations of the immune function are legitimate avenues for novel therapy in selected cases of autism.

Social Policy and Training

Local authority training programs which help to spread knowledge and understanding should aim at extending expertise at different skill levels and to different sectors and should be open to all staff, not restricted for teachers only. In addition, recording of conferences and other sessions should be made available using high technology as webcasts so that they can be more accessible to a larger audience. Schools training policy should include an audit of existing skills, autism-specific qualifications, and training needs and should provide planning to meet those needs. Staff should be funded to participate in accredited courses in autism.

See Also

- [Autism Diagnostic Interview-Revised](#)
- [Autism Science Foundation](#)
- [Autism Speaks](#)
- [ICD 10 Research Diagnostic Guidelines](#)

References and Reading

- Abdel Razek, A., Mazroa, J., & Baz, H. (2013). Assessment of white matter integrity of autistic preschool children with diffusion weighted MR imaging. *Brain and Development*, 36(1), 28–34.
- Abdel, H., Ramadan, E., & Elshair, I. (2012). The effect of educational program on caregivers (knowledge and practicing) toward their autistic children. *Journal of American Science*, 8(1), 1–6. <http://www.jofamericanscience.org>
- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in ASD genetics: On the threshold of a new neurobiology. *Nature Reviews. Genetics*, 9, 341–355.
- Allam, H., ElDine, N. G., & Helmy, G. (2008). Scalp acupuncture effect on language development in children with autism: A pilot study. *Journal of Alternative and Complementary Medicine*, 14(2), 109–114.
- Al-Farsi, Y. M., Al-Sharbati, M. M., Al-Farsi, O. M., Al-Shafae, M. S., Brooks, D. R., Waly, M. I. (2010). Brief report: prevalence of autistic spectrum disorders in the sultanate of oman. *Journal of Autism and Developmental Disorders*, 41(6), 821–825.
- Al-Salehi, S. M., Al-Hifthy, E. H., & Ghaziuddin, M. (2009). Autism in Saudi Arabia: Presentation, clinical correlates and comorbidity. *Transcultural Psychiatry*, 46(2), 340–347.
- Cohen, N. J., & Volkmar, F. R. (2005). *Handbook of autism and pervasive developmental disorders* (2nd ed.). New York: Wiley.
- Eapen, V., MabroukAA Zoubeidi, T., Yunis, F. (2007). Prevalence of pervasive developmental disorders in preschool children in the UAE. *Journal of Tropical Pediatrics*, 53(3), 202–205.
- El Baz, F., Ismael, N., & Nour El-Din, S. (2011). Risk factors for autism: An Egyptian study. *The Egyptian Journal of Medical Human Genetics*, 12, 31–38.
- Elbahnasawy, H., & Girgis, M. (2011). Counseling for mothers to cope with their autistic children. *Journal of American Science*, 7(7), 183–192.
- Elsabbagh, M., Divan, G., Koh, Y.-J., Kim, Y. S., Kauchali, S., Marcin, C., Montiel-Nava, C., Pate, V., Paula, C. S., Wang, C., Yasamy, M. T., & FombonneGlobal, E. (2012). Prevalence of autism and other pervasive developmental disorders. *Autism Research*, 5(3), 160–179.
- Gebril, O., & Meguid, N. (2011). HFE gene polymorphisms and the risk for autism in Egyptian children and impact on the effect of oxidative stress. *Disease Markers*, 31(5), 289–294.
- Helping Egyptian children with autism – The Egyptian Gazette 213.158.162.45/~egyptian/index.php?action=news&id=18159...May 14, 2011 – Dr Shawkia S. Abdel-Halim of the Nutritional Institute
- Hong, K. M., Yamazaki, K., & Banaag, C. G. (2004). Systems of care in Asia. In H. Remschmidt, M. Belfer, & I. Goodyer (Eds.), *Facilitating pathways: Care, treatment and prevention in Child and Adolescent Mental Health* (pp. 58–70). Berlin: Springer.
- Hussein, H., Taha, G. H., & Almanasef, A. (2011). Characteristics of autism spectrum disorders in a sample of Egyptian and Saudi patients: Transcultural cross sectional study. *Child and Adolescent Psychiatry and Mental Health*, 5, 34.
- Hussein, H., Shaker, N., El-Sheikh, M., & Ramy, H. A. (2012). Pathways to child mental health services among patients in an urban clinical setting in Egypt. *Psychiatric Services*, 63(12), 1225–1230.
- Ibrahim, K. S., & El-Sayed, E. M. (2012). Proposed remedies for some developmental disorders. *Toxicology and Industrial Health*, 29(4), 367–384.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217.
- Kawashti, M. I., Amin, O. R., & Rowehey, N. G. (2006). Possible immunological disorders in autism: Concomitant autoimmunity and immune tolerance. *Egyptian Journal of Immunology*, 13(1), 99–104.
- King, G., Zwaigenbaum, L., King, S., & Baxter, D. (2006). A qualitative investigation of changes in the belief systems of families of children with autism or Down syndrome. *Child Care, Health & Development*, 3, 353–368.
- Leibson, C. L., Katusic, S. K., Barbaresi, W. J., et al. (2001). Use and costs of medical care for children and adolescents with and without attention-deficit-hyperactivity disorder. *JAMA*, 285, 60–66.

- Longe, C. I., & Asuni, T. (1972). Four cases of Infantile autism in Nigerian children. Paper presented at Third Pan African Psychiatric Conference, Khartoum, 1972.
- Lotter, V. (1978). Childhood autism in Africa. *Journal of Child Psychology and Psychiatry*, 19(3), 231–244.
- Lotter, V. (1980). Cross cultural perspectives on childhood autism. *Journal of Tropical Pediatrics*, 26(4), 131–133.
- Meguid, N. A., Dardir, A. A., El-Sayed E. M., Ahmed H. H., Hashish A. F., Ezzat A. (2010). Homocysteine and oxidative stress in Egyptian children with Down syndrome. *Clin Biochem*. 43(12), 963–967.
- Meguid, N. A., Hashish, A. F., Anwar, M., & Sidhom, G. (2010a). Assessment of 25-Hydroxy and 1, 25-Dihydroxy vitamin D in Egyptian children with autism. *Journal of Alternative and Complementary Medicine*, 16(6), 641–645.
- Meguid, N. A., Fahim, C., Uicheul, Y., Nashaat, N., Samir, A., Mancini-Marie, A., Brandner, C., & Evans, A. C. (2010b). Brian morphology in autism and Fragile X syndrome correlates with Social IQ. *Journal of Child Neurology*, 25(5), 599–608.
- Meguid, N. A., Dardir, A. A., Abdel-Raouf, E. R., & Hashish, A. (2011). Evaluation of oxidative stress in autism: Defective antioxidant enzymes and increased lipid peroxidation. *Biological Trace Element Research*, 143(1), 58–65.
- Meguid, N. A., Ehab R Abdel-Raouf, Ahmed A Dardir, Mostafa K. (2007): Prevalence of fragile X syndrome among school-age Egyptian males. *World J Pediatr*. 3 (4):271–275.
- Mostafa, G. A., & Kitchener, N. (2009). Serum anti-nuclear antibodies as a marker of autoimmunity in Egyptian autistic children. *Pediatric Neurology*, 40(2), 107–112.
- Mostafa, G. A., El-Sayed, Z. A., El-Aziz, M. M., & El-Sayed, M. F. (2008). Serum anti-myelin-associated glycoprotein antibodies in Egyptian autistic children. *Journal of Child Neurology*, 23(12), 1413–1418.
- Mostafa, G. A., Shehab, A. A. (2010): The link of C4B null allele to autism and to a family history of autoimmunity in Egyptian autistic children. *Journal of Neuroimmunology*, 223(1–2):115–9.
- Mostafa, G. A., Shehab, A. A., Al-Ayadhi, L. Y. (2012). The link between some alleles on human leukocyte antigen system and autism in children. *Journal of Neuroimmunology*, 255(1–2):70–4.
- Mostafa, G. A., Shehab, A. A., & Al-Ayadhi, L. Y. (2013). The link between some alleles on human leukocyte antigen system and autism in children. *Journal of Neuroimmunology*, 255(1–2), 70–74.
- Novarino, G., El-Fishawy, P., Kayserili, H., Meguid, N. A., Scott, E. M., Schroth, J., Silhavy, J. L., Kara, M., Khalil, R. O., Ben-Omran, T., Ercan-Sencicek, A. G., Hashish, A. F., Sanders, S. J., Gupta, A. R., Hashem, H. S., Matern, D., Gabriel, S., Sweetman, L., Rahimi, Y., Harris, R. A., State, M. W., & Gleeson, J. G. (2012). Mutations in BCKD-kinase lead to a potentially treatable form of autism with epilepsy. *Science*, 338, 394–397.
- Obaid, M. (2012). Parental attitudes towards autistic child. *European Journal of Social Sciences*, 31(1), 103–114.
- Okasha, A. (2004). Focus on psychiatry in Egypt. *British Journal of Psychiatry*, 185, 266–272.
- Robertson, B., Mandlhate, C., & Seif El-Din, A. (2004). Systems of care in Africa. In H. Remschmidt, M. Belfer, & I. Goodyer (Eds.), *Facilitating pathways: Care, treatment and prevention in child and adolescent mental health* (pp. 71–88). Berlin: Springer.
- Samadi, S. A., & McConkey, R. (2011). Autism in Developing Countries: Lessons from Iran. *Autism Research and Treatment*, 1–11.
- Seif Eldin, A., Habib, D., Noufal, A., Farrag, S., Bazaid, K., Al-Sharbati, M., Badr, H., Moussa, S., Essali, A., & Gaddour, N. (2008). Use of M-CHAT for a multinational screening of young children with autism in the Arab countries. *International Review of Psychiatry*, 20(3), 281–289.
- Yamamah, G., Abdel-Raouf, E., Talaat, A., Saad-Hussein, A., Hamamy, H., Meguid, N. A. (2012). Prevalence of consanguineous marriages in south Sinai, Egypt. *J Biosoc Sci*, 14, 1–9.

EHI

- [Edinburgh Handedness Inventory](#)

EI

- [Early Intervention](#)

Eisenberg, Leon

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Name and Degrees

Leon Eisenberg received both his BA and MD degrees from the University of Pennsylvania. He completed an internship at Mt. Sinai before

teaching briefly at the University of Pennsylvania. Following work as a psychiatrist in the US Army, he became a resident at Sheppard Pratt Hospital before beginning child psychiatry training at Johns Hopkins.

Major Appointments (Institution, Location, Dates)

- Johns Hopkins University: Baltimore, MD, 1953–1967, Instructor, Associate Professor, and Professor and Chief of Child Psychiatry.
- Harvard University, Cambridge, MA, 1967–1993, Professor of Psychiatry, Chair Department of Social Medicine and Health Policy, Maude and Lillian Presley Professor of Social Medicine, Emeritus status in 1993.

Major Honors and Awards

- Leon Eisenberg was the recipient of numerous honorary degrees and awards; some of the major awards include the Richmond award from the American Academy of Pediatrics, the Thomas W. Salmon Medal from the New York Academy of Medicine, and Juan José López Ibor Award from the World Congress of Psychiatry. In addition, several awards have been named in his honor. In 2009, the Leon Eisenberg Chair of Child Psychiatry at Children's Hospital Boston was named in his honor.

Landmark Clinical, Scientific, and Professional Contributions

Dr. Eisenberg conducted the first follow-up study (with Leo Kanner) of children with autism. Other accomplishments included his role in the first randomized control study of psychopharmacology in children and the first randomized controlled trial of stimulants in adolescents and of brief psychotherapy. He had long-standing interests in the social brain and in social medicine including an active role in working with Physicians for Human Rights.

Short Biography

A child psychiatrist and medical educator, Dr. Eisenberg was an original contributor to a series of innovations in the field of child psychiatry. These included, in collaboration with his mentor Leo Kanner, MD, some of the first follow-up studies of children with autism, in development of randomized clinical trials (RCTs), evidence-based medicine, and social medicine and international advocacy including for human rights.

Dr. Eisenberg followed Leo Kanner as the leader of child psychiatry at Johns Hopkins until his retirement there in 1967. At that time, he moved to Harvard University where he assumed leadership of the child psychiatry program. In 1974, he was appointed the Maude and Lillian Presley Professor of Psychiatry and, in 1980, became chair of the Department of Social Medicine and Health Policy until his retirement in 1993 when he became emeritus. He remained an active researcher and advocate until shortly before his death.

See Also

- [Kanner, Leo](#)

References and Reading

- Conners, C. K., Rothschild, G., Eisenberg, L., Schwartz, L. S., & Robinson, E. (1969). Dextroamphetamine sulfate in children with learning disorders. *Archives of General Psychiatry*, 21, 182–190.
- Eisenberg, L. (1956). The autistic child in adolescence. *American Journal of Psychiatry*, 112, 607–612. Reprinted in Alexander et al. (Eds.). (1959). *Psychopathology*. Cambridge: Harvard University Press.
- Eisenberg, L. (1957a). The fathers of autistic children. *American Journal of Orthopsychiatry*, 27, 715–724.
- Eisenberg, L. (1957b). The course of childhood schizophrenia. *Archives of Neurology and Psychiatry*, 78, 69–83.
- Eisenberg, L. (1957c). Progress in neuropsychiatry. *Journal of Pediatrics*, 51, 334–349.
- Eisenberg, L. (1957d). Psychiatric implications of brain damage in children. *Psychiatric Quarterly*, 31, 72–92.
- Eisenberg, L. (1968). The social development of human intelligence. *Harvard Medical Alumni Bulletin*, 43,

- 2–7; Reprinted in Freeman, H. (Ed.). (1969). *Progress in mental health*. Churchill.
- Eisenberg, L. (1998). Nature, niche and nurture: The role of social experience in transforming genotype into phenotype. *Academic Psychiatry*, 22, 213–222.
- Eisenberg, L. (2004). Social psychiatry and the human genome: Contextualizing heritability. *British Journal of Psychiatry*, 184, 101–103.
- Glaser, K., & Eisenberg, L. (1956). Maternal deprivation. *Pediatrics*, 18, 626–642.
- Kanner, L., & Eisenberg, L. (1956a). Child psychiatry; mental deficiency. *American Journal of Psychiatry*, 112, 531–534.
- Kanner, L., & Eisenberg, L. (1956b). Early infantile autism 1943–1955. *American Journal of Orthopsychiatry*, 26, 55–65. Reprinted in: Alexander et al. (Eds.). Op. cit. Reprinted in *Psychiatric Research Report 1957* (April), American Psychiatric Association, pp. 55–65.
- Kanner, L., & Eisenberg, L. (1957). Child psychiatry: Mental deficiency. *American Journal of Psychiatry*, 113, 617.
- Kanner, L., & Eisenberg, L. (1958). Child psychiatry; mental deficiency. *American Journal of Psychiatry*, 114, 609–615.
- Rutter, M., Lebovici, S., Eisenberg, L., Sneznevskij, A. V., Sadoun, R., Brooke, E., et al. (1969). A triaxial classification of mental disorders in childhood. *Journal of Child Psychology and Psychiatry*, 10, 41–61.

Elated, Euphoric and Grandiose

► Mania

Elavil (Amitriptyline)

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Synonyms

Amitriptyline; Endep; Levate

Definition

Elavil is a tricyclic antidepressant (TCA). By blocking their reuptake by presynaptic neurons, TCAs increase the synaptic levels and action durations of noradrenaline and serotonin. Serotonin is an inhibitory neurotransmitter that is heavily involved in mood and emotion regulation and has been found to be elevated in the whole blood and platelets of ASD patients. In addition to their reuptake-inhibiting properties, TCAs also exert antagonistic effects on histamine H₂, serotonin 5-HT₂, α₁-adrenergic, and muscarinic acetylcholine receptors. While many of these neurotransmission systems have been implicated in autism pathogenesis, the generally low receptor specificity results in a high toxicity profile. Side effects include nausea, drowsiness, weakness, dry mouth, changes in appetite or weight gain, constipation, blurred vision, increased sweating, and decreased sexual drive. Low doses of this drug do show promise in the treatment of hyperactivity, impulsivity, aggression, and self-injury in ASD patients. However, further substantial validation is still required. A Cochrane review in 2012 did not currently recommend the use of tricyclic antidepressants for the treatment of individuals with autism spectrum disorder.

See Also

- Antidepressants
- Serotonin Reuptake Inhibitors (SRIs)
- Tricyclic Antidepressants (TCAs)

References and Reading

- Barsa, J. A., & Saunders, J. C. (1961). Amitriptyline (Elavil), a new antidepressant. *American Journal of Psychiatry*, 117(8), 739–740.
- Bhatti, I., Thome, A., Smith, P. O., Cook-Wiens, G., Yeh, H. W., Gaffney, G. R., & Hellings, J. A. (2013). A retrospective study of amitriptyline in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 1–11.
- Hurwitz, R., Blackmore, R., Hazell, P., Williams, K., & Woolfenden, S. (2012). Tricyclic antidepressants for autism spectrum disorders (ASD) in children and

adolescents. *Cochrane Database of Systematic Reviews*, 3, CD008372.

Warren, R. P., & Singh, V. K. (1996). Elevated serotonin levels in autism: Association with the major histocompatibility complex. *Neuropsychobiology*, 34(2), 72–75.

Elderly with Autism Spectrum Disorders

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Definition

Kanner's sentinel article *Autistic Disturbances of Affective Contact* focused on children, and this autism research and inquiry has been focused on children and adolescents on the autism spectrum (Jang et al. 2014; Kanner 1943; Perkins and Berkman 2012; Wise et al. 2017). However, as our knowledge about the autism spectrum has increased, more research about adults and elderly people with this condition is emerging. This entry begins with an overview of the proportion of age cohorts which are examined in autism research. It will then summarize the small amount of published research about elderly people on the autism spectrum. This will be followed by an explanation for why this population is rarely researched and areas where more research about this age cohort could be useful in meeting individual needs as well as wider public health goals.

Historical Background

Since the publication of Kanner's writings about autism, there has been a systematic and ongoing effort to examine children with autism, resulting in a dearth of research about other cohorts. In 2014, Jang and colleagues published a study where they calculated the proportion of age cohorts in autism articles which were published from 1994 to 2004. They collected and analyzed

2857 articles from 7 journals which specialized in publishing articles about the autism spectrum. They excluded letters to the editor, meta-analysis studies, and systematic literature reviews. Participants were classified and assigned to one of the four age categories: infants/toddlers (0–3 years), children (4–9 years), adolescents (10–19 years), and adults (>20 years, with further breakdown for those over 60). Jang et al. (2014) found that 94% of all studies ($n = 2688$) which they collected and analyzed examined infants, toddlers, children, and adolescents (between 0 and 19 years of age). In contrast, 3% of studies ($n = 75$) examined adults on the autism spectrum aged 60 years or older. Jang et al. (2014) concluded that most of our knowledge about the autism spectrum has been obtained from infants, children, and adolescents. Furthermore, based on this finding, Jang et al. (2014) proposed that more research should be conducted on the adults since most of a person's life occurs in adulthood.

Current Knowledge

There are publications which have reviewed the types of research published about elderly people on the autism spectrum (Bennett 2016; Happé and Charlton 2012; Patra 2016; van Heijst and Geurts 2015; van Niekerk et al. 2011; Wright et al. 2013). Bennett (2016) cited five studies about elderly people on the autism spectrum which examined executive functions and memory, diagnostic instruments used to identify Asperger syndrome in seniors, and diagnosing depression in elderly people with Asperger syndrome. Happé and Charlton (2012) published a literature review focused on articles which explored elderly people on the autism spectrum. The studies which they collected were assigned to one of three categories: (1) case studies of elderly people on the autism spectrum, (2) discussion papers which outlined the challenges of diagnosing autism in seniors, and (3) articles explaining empirical findings from surveys of studies about elderly people with autism. Patra (2016) published a systematic literature review of studies

about seniors on the autism spectrum stored on the PubMed electronic citation database. They identified 12 English language studies which met their inclusion criteria. The oldest study which they retrieved was published in 2013, and the most recent study was published in June 2016. The studies which they examined explored the physical health, psychological well-being, and cognitive or social functioning of elderly people with autism. Finally, van Niekerk et al. (2011) published a systematic review of studies about the instruments used to diagnose the autism spectrum in elderly people. They searched for relevant articles on PubMed, PsychLIT, and EMBASE which were published from 1996 to December 2009. They identified three studies which met their inclusion criteria. These studies examined all showed the importance of identifying the autism spectrum in the elderly and explained the long-term consequences of not identifying this condition within the elderly. In summary, there is a paucity of literature about elderly on the autism spectrum.

Aside from systematic literature reviews, studies have also examined the executive functions and memory abilities of elderly people with autism (Geurts and Vissers 2012). Geurts and Vissers's (2012) study compared the neuropsychological profile of elderly adults on and not on the autism spectrum. They sampled 23 elderly adults on the autism spectrum (18 males, 5 females; mean age = 63.6 years, SD = 7.5 years) and 23 elderly adults not on the autism spectrum (18 males, 5 females; mean age = 63.7 years, SD = 8.1 years). In addition, out of 23 elderly adults on the autism spectrum, 16 had Asperger syndrome, 2 had autism, 1 had a diagnosis of pervasive developmental disorder-not otherwise specified, 1 had an autism spectrum disorder diagnosis, and the remaining 3 had autistic tendencies. In comparison to those not on the autism spectrum, Geurts and Vissers (2012) found that elderly people on the autism spectrum had deficits with their working memory and had different cognitive profiles and trajectories of cognitive development.

Jang et al. (2014) posited that most studies about the autism spectrum focused on children

rather than adults or elderly. It is possible that a barrier to research around elderly people is less likely to have been diagnosed with an autism spectrum disorder. According to Bennett (2016), current assessment processes and instruments used to diagnose autism have not been tailored to identify autism in the elderly. Often an autism diagnosis is based on a mixture of clinical tests, clinical observations of the patient, and the patient's family providing the clinician with a description of their relative's behaviors. For patients who are elderly, however, such an approach might not be effective in diagnosing autism. During old age, elderly people often live alone or in nursing homes where regular contact with immediate or extended family members is limited (Bennett 2016). Additionally, James et al. (2006) explain that the questions which clinicians use in autism diagnostic assessments for children might not be appropriate for seniors. For example, while a clinician might be able to diagnose a child with autism by asking him/her questions about his/her friendships, such questions might not be applicable for seniors because their social network is smaller and they might have less social contacts. Furthermore, Geurts and Vissers (2012) explain that the clinical tests used to diagnose children on the autism spectrum are difficult to translate and apply to elderly people on the autism spectrum. Finally, Mukaetova-Ladinska et al. (2012) explain that, aside for developing tests to diagnose the autism spectrum in the elderly, there is also a need to develop cognitive and behavioral assessment tools for elderly people who are on the autism spectrum.

Future Directions

Despite the small amount of literature about elderly people on the autism spectrum systematically increasing each year, there are still many areas of research to be explored (Bennett 2016; Happé and Charlton 2012; Hategan et al. 2017; Totsika et al. 2010). Bennett (2016) proposed that the impact of nursing home care programs on elderly people on the autism spectrum is an area yet to be researched. In addition, Happé and

Charlton (2012) explained that social isolation often has a detrimental impact on elderly people not on the autism spectrum. However, it is unknown if seniors on the autism spectrum who engage in solitary hobbies and interests, which they find enjoyable and motivating, can somehow shield themselves from the negative impacts of social isolation. Michael, a person on the autism spectrum, has also voiced views about the importance of researching elderly people on the autism spectrum (Michael 2016). According to Michael (2016), very little is known about how elderly people on the autism spectrum manage medical conditions which occur during middle or older age, such as arthritis, menopause, type 2 diabetes, and some types of cancer. Similarly, Kats et al. (2013) published a study about clinical problems which older adults with autism and an intellectual disability exhibited. They found that there was an urgent need for research about the nature and treatment of behavioral problems in elderly on the autism spectrum. Finally, Perkins and Berkman's (2012) article showed that people on the autism spectrum have a lower life expectancy than those not on the autism spectrum. However, it remains unknown why there is a life expectancy discrepancy.

While increasing our knowledge and understanding about elderly people on the autism spectrum is a sensible goal, it cannot, however, be perused without support. Researchers may also struggle to work with elderly adults on the autism spectrum to gain an insight into their lived experience which may impact on their willingness to research in this area. Communication and emotional difficulties can be exacerbated in old age, and there has been little impetus to look at the well-being or economic costs of this growing population so far.

Piven and Rabins (2011) explained that a prolonged, integrated, and multipronged approach is required to study seniors on the autism spectrum from a psychological, sociological, and economic perspective. Piven and Rabins (2011) also propose that clinical and research training programs should develop ways to attract and retain early career researchers, from a diverse range of disciplines, to study elderly people on the autism

spectrum. Similarly, Bennett (2016) suggested that governments should increase funding for projects which can evaluate healthcare systems and services for seniors on the autism spectrum. Support services which are effective will be of benefit to both elderly on the autism spectrum and the wider community in terms of improving social outcomes and increasing economic efficiency.

Although there have been many recent developments in the study of elderly on the autism spectrum (Wise et al. 2017), there remains many areas where additional research can be conducted (Amanullah and Rajeh 2019). For example, despite literature showing that the elderly are often financially (Wajnberg et al. 2020) and physically abused (Yon et al. 2020), there is a dearth of literature about these topics for elderly on the autism spectrum. In the future, researchers should explore if senior citizens on the autism spectrum encounter greater rates of elder abuse than those who are not on the autism spectrum. They might possibly experience greater rates of elder abuse since their social difficulties might leave them with greater vulnerabilities (Flynn and Healy 2012).

As Bennett (2016) has explained, most of the children currently living with an autism spectrum diagnosis, at current the focus of research, will inevitably become elderly. If the amount of research about elderly people on the autism spectrum does not increase, then today's children on the autism spectrum may be disadvantaged in their old age. Furthermore, if there is a lack of research about elderly on the autism spectrum in the future, then the needs of elderly people with this condition will become the subject of speculation. To avoid this speculation altogether, and to help future generations of elderly people on the autism spectrum, it is crucial that research about the autism spectrum in old age continues to increase.

References and Reading

- Amanullah, S., & Rajeh, A. (2019). An overview of autism in the elderly. *Asian Journal of Psychiatry*, 101, 897. <https://doi.org/10.1016/j.ajp.2019.101897>.

- Bennett, M. (2016). "What is life like in the twilight years?" A letter about the scant amount of literature on the elderly with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46(5), 1883–1884.
- Flynn, L., & Healy, O. (2012). A review of treatments for deficits in social skills and self-help skills in autism spectrum disorder. *Research in Autism Spectrum Disorders*, 6(1), 431–441. <https://doi.org/10.1016/j.rasd.2011.06.016>.
- Geurts, H. M., & Vissers, M. E. (2012). Elderly with autism: Executive functions and memory. *Journal of Autism and Developmental Disorders*, 42(5), 665–675.
- Happé, F., & Charlton, R. A. (2012). Aging in autism spectrum disorders: A mini-review. *Gerontology*, 58(1), 70–78.
- Hategan, A., Bourgeois, J. A., & Goldberg, J. (2017). Aging with autism spectrum disorder: An emerging public health problem. *International Psychogeriatrics*, 29(4), 695–697.
- James, I. A., Mukaetova-Ladinska, E., Reichelt, F. K., Briel, R., & Scully, A. (2006). Diagnosing Aspergers syndrome in the elderly: A series of case presentations. *International Journal of Geriatric Psychiatry*, 21(10), 951–960.
- Jang, J., Matson, J. L., Adams, H. L., Konst, M. J., Cervantes, P. E., & Goldin, R. L. (2014). What are the ages of persons studied in autism research: A 20-year review. *Research in Autism Spectrum Disorders*, 8(12), 1756–1760.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2(3), 217–250.
- Kats, D., Payne, L., Parlier, M., & Piven, J. (2013). Prevalence of selected clinical problems in older adults with autism and intellectual disability. *Journal of Neurodevelopmental Disorders*, 5(1), 27.
- Michael, C. (2016). Why we need research about autism and ageing. *Autism*, 20(5), 515–516.
- Mukaetova-Ladinska, E. B., Perry, E., Baron, M., & Povey, C. (2012). Ageing in people with autistic spectrum disorder. *International Journal of Geriatric Psychiatry*, 27(2), 109–118.
- Patra, S. (2016). Autism spectrum disorder in the elderly: A review of healthcare issues and challenges. *Journal of Geriatric Care and Research*, 3(1), 3–6.
- Perkins, E. A., & Berkman, K. A. (2012). Into the unknown: Aging with autism spectrum disorders. *American Journal on Intellectual and Developmental Disabilities*, 117(6), 478–496.
- Piven, J., & Rabins, P. (2011). Autism spectrum disorders in older adults: Toward defining a research agenda. *Journal of the American Geriatrics Society*, 59(11), 2151–2155.
- Totsika, V., Felce, D., Kerr, M., & Hastings, R. P. (2010). Behavior problems, psychiatric symptoms, and quality of life for older adults with intellectual disability with and without autism. *Journal of Autism and Developmental Disorders*, 40(10), 1171–1178.
- van Heijst, B. F., & Geurts, H. M. (2015). Quality of life in autism across the lifespan: A meta-analysis. *Autism*, 19(2), 158–167.
- van Niekerk, M. E., Groen, W., Vissers, C. T. W., van Driel-de Jong, D., Kan, C. C., & Voshaar, R. C. O. (2011). Diagnosing autism spectrum disorders in elderly people. *International Psychogeriatrics*, 23(5), 700–710.
- Wajnberg, A., Koppel, S., & Kahan, F. (2020). Elder abuse and neglect. In *Home-based medical care for older adults* (pp. 41–47). Cham: Springer. https://doi.org/10.1007/978-3-030-23483-6_7.
- Wise, E. A., Smith, M. D., & Rabins, P. V. (2017). Aging and autism Spectrum disorder: A naturalistic, longitudinal study of the comorbidities and behavioral and neuropsychiatric symptoms in adults with ASD. *Journal of Autism and Developmental Disorders*, 47(6), 1708–1715.
- Wright, S. D., Brooks, D. E., & Grandin, T. (2013). The challenge and promise of autism spectrum disorders in adulthood and aging: A systematic review of the literature (1990–2013). *Autism Insights*, 5, 21.
- Yon, Y., Lam, J., Passmore, J., Huber, M., & Sethi, D. (2020). The public health approach to elder abuse prevention in europe: progress and challenges. In *Advances in elder abuse research* (pp. 223–237). Cham: Springer. https://doi.org/10.1007/978-3-030-25093-5_15.

Elective Mutism

► Selective Mutism

Electroconvulsive Therapy (ECT)

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Synonyms

ECT

Definition

Electroconvulsive Therapy (ECT) is a medical procedure in which a seizure (convulsion) is

induced via the external application of brief, focused electrical current in order to effect alterations in the electrophysiology of the brain with the goal of mitigating severe psychiatric symptoms. Although the exact mechanism of action remains unclear, evidence suggests that ECT may produce changes across multiple domains, including neuroendocrine systems, neurotransmitter tone and activity, and neuroplasticity. For example, studies have provided evidence that ECT potentiates the effects of the neurotransmitters serotonin, norepinephrine, and dopamine and increases cortical Gamma-Aminobutyric Acid (GABA) concentration. ECT has also been shown to be a strong inducer of dendritic arborization and new cell formation in the hippocampus, as well as reducer of dendritic arborization and excitatory synapses in the amygdala. These findings are consistent with the clinical effects observed with ECT: improvements in depressive and manic symptoms and suicidality, mitigation of catatonia, and decreased severity of some Parkinsonian symptoms. More recent studies have also reported decreased frequency of self-injurious behaviors after ECT.

Per guidelines from the American Psychiatric Association and the American Academy of Child and Adolescent Psychiatry, ECT may be indicated for the treatment of adolescents and adults with persistent, significantly disabling or life-threatening mood disorders such as depression or mania, psychosis, or catatonia refractory to other treatment modalities. ECT can also be used to treat neuroleptic malignant syndrome, a rare but potentially fatal condition that can occur in patients taking neuroleptic medications. Inadequate evidence exists regarding the use of ECT in preadolescents. However, multiple studies across decades of research in adolescents and adults have shown ECT to have efficacy superior to other treatment modalities for indicated conditions, with response rates for severe, intractable mood disorders, and catatonia as high as 80%.

More recently, researchers have reported success in using ECT to decrease severe, repetitive

self-injurious behavior in both typically developing patients with psychiatric comorbidities and patients with Autism Spectrum Disorder (ASD). As noted in other sections, repetitive, restricted, and stereotyped behavior is a primary feature of ASD. In up to 25% of individuals with ASD, such behavior is manifested as self-injurious behavior (SIB), which can range from occasional, mild episodes to frequent, unrelenting, and severe behaviors, leading to potentially serious injuries. Stereotyped movements also form part of the diagnostic criteria for catatonia, and some researchers have posited that catatonia has been historically underdiagnosed and inadequately treated in individuals with ASD. In fact, several large studies have estimated that 12–18% of autistic adolescents and young adults exhibit some symptoms of catatonia. While first-line treatments for SIB and catatonia include medication and (for SIB) behavioral interventions, some individuals present with severe and disabling symptoms refractory to these treatments. Although more research is needed, ECT may be considered as an option in patients with such refractory symptoms in order to decrease patient distress and avoid potentially catastrophic outcomes.

There are no absolute medical or neurological contraindications to the use of ECT, though certain CNS tumors, active chest infections or cardiac conditions are relative contraindications. Likewise, there are no psychiatric comorbidities or developmental disabilities which would be contraindications to the procedure when it is otherwise indicated. ECT has been shown to be a relatively safe procedure in modern use, well-tolerated by most patients receiving treatment. Modern ECT is performed under brief general anesthesia, and the reported mortality rate of up to four deaths per 100,000 treatments is believed to be primarily due to the risks associated with the general anesthesia. Of note, the mortality rate of ECT remains significantly lower than mortality rates for the intractable depression, mania, and catatonia for which ECT is indicated. Common side effects after ECT sessions include transient headache, nausea, fatigue, confusion, difficulties

in learning, and memory loss. Of these, memory loss has often been reported to be the most troubling to patients, and although it is most often transient or self-limited, there have been credible reports of persistent or even permanent memory loss, though usually limited to the time period around treatment. A course of ECT for acute psychiatric symptoms usually includes from 6 to 20 treatments, with the number of treatments generally determined by each patient's response to treatment and tolerance of the procedure. Some patients also appear to benefit from maintenance ECT to provide ongoing relief and prevent recurrence of symptoms.

References and Reading

- American Academy of Child and Adolescent Psychiatry, Ghaziuddin, N., et al. (2004). Practice parameter for use of electroconvulsive therapy with adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43(12), 1521–1539.
- American Psychiatric Association; Committee on Electroconvulsive Therapy, Weiner, R. D. (chairperson), et al. (2001). *The practice of electroconvulsive therapy: recommendations for treatment, training and privileging* (2nd ed.). Washington, DC: American Psychiatric Publishing.
- D'Agati, D., Chang, A. D., Wachtel, L. E., & Reti, I. M. (2017). Treatment of severe self-injurious behavior in autism Spectrum disorder by neuromodulation. *Journal of ECT*, 33(1), 7–11.
- Mazzone, L., Postorino, V., Valeri, G., & Vicari, S. (2014). Catatonia in patients with autism: Prevalence and management. *CNS Drugs*, 28, 205–215.
- McCall, W. V., Andrade, C., & Sienart, P. (2014). Searching for the mechanism(s) of ECT's therapeutic effect. *Journal of ECT*, 30(2), 87–89.
- Reti, I. M. (2015). How does electroconvulsive therapy work? In I. M. Reti (Ed.), *Brain stimulation: Methodologies and interventions* (1st ed., pp. 107–122). Hoboken: Wiley Blackwell.
- Sadock, B. J., & Sadock, V. A. (2007). Brain stimulation methods. In B. J. Sadock & V. A. Sadock (Eds.), *Kaplan and Sadock's synopsis of psychiatry: Behavioral sciences/clinical psychiatry* (10th ed., pp. 1117–1124). Philadelphia: Wolters Kluwer.
- Wachtel, L. E., Dhossche, D. M., & Kellner, C. H. (2011). When is electroconvulsive therapy appropriate for children and adolescents? *Medical Hypotheses*, 76, 395–399.

Electroencephalogram (EEG)

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Synonyms

[Electroencephalography](#)

Definition

An electroencephalogram is a graphical representation of the brain's electrical activity. In human assessment, electrodes are placed at various locations on the scalp. As neurons in the brain fire, these electrodes capture the resulting voltage, which is then amplified and recorded. This electrical activity oscillates quite rapidly. The number of oscillations over a phase of time can be measured and are termed “frequency.” Variations in frequency have been historically quantified in terms of oscillations per 1-s period as hertz (Hz). Common categorizations of frequencies include alpha (8–13 Hz), beta (13–30 Hz), gamma (30–600 Hz), delta (≤ 4 Hz), theta (4–8 Hz), and mu (8–13 Hz).

A subtype of electroencephalography examines changes in brain activity time-locked to particular events, termed event-related potentials (ERPs). ERPs are generally assessed in small windows of time, in the tens and hundreds of milliseconds.

See Also

- ▶ [Electroencephalography](#)
- ▶ [Event-Related Potential](#)
- ▶ [Evoked Potentials](#)
- ▶ [Mu Rhythm](#)

References and Reading

- Andreassi, J. L. (2007a). *Psychophysiology: Human behavior & physiological response* (5th ed.). Mahwah: Lawrence Erlbaum Associates.
- Andreassi, J. L. (2007b). The nervous system and measurement of its activity. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 85–119). Cambridge, UK: Cambridge University Press.
- Herrmann, C. S., Grigutsch, M., & Busch, N. (2005). EEG oscillations and wavelet analysis. In T. C. Handy (Ed.), *Event-related potentials: A methods handbook* (pp. 229–259). Cambridge, MA: MIT Press.

windows of time, in the tens and hundreds of milliseconds.

See Also

- [Electroencephalogram \(EEG\)](#)
- [Event-Related Potential](#)
- [Evoked Potentials](#)
- [Mu Rhythm](#)

References and Reading

- Andreassi, J. L. (2007a). *Psychophysiology: Human behavior & physiological response* (5th ed.). Mahwah: Lawrence Erlbaum Associates.
- Andreassi, J. L. (2007b). The nervous system and measurement of its activity. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 85–119). Cambridge, UK: Cambridge University Press.
- Herrmann, C. S., Grigutsch, M., & Busch, N. (2005). EEG oscillations and wavelet analysis. In T. C. Handy (Ed.), *Event-related potentials: A methods handbook* (pp. 229–259). Cambridge, MA: MIT Press.

Electroencephalography

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Synonyms

[Electroencephalogram \(EEG\)](#)

Definition

Electroencephalography (EEG) is the recording of brain's electrical activity. In human assessment, electrodes are placed at various locations on the scalp. As neurons in the brain fire, these electrodes capture the resulting voltage, which is then amplified and recorded. This electrical activity oscillates quite rapidly. The number of oscillations over a phase of time can be measured and are termed "frequency." Variations in frequency have been historically quantified in terms of oscillations per 1-s period as hertz (Hz). Common categorizations of frequencies include alpha (8–13 Hz), beta (13–30 Hz), gamma (30–600 Hz), delta (≤ 4 Hz), theta (4–8 Hz), and mu (8–13 Hz).

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Electrophysiology

- [Neurophysiology](#)

Elementary and Secondary Education Act (No Child Left Behind)

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Definition

The No Child Left Behind (NCLB) Act of 2001 is the most recent reauthorization of the

Elementary and Secondary Education Act (ESEA). It was signed into law on January 8, 2002, and received an overwhelming bipartisan majority in both the House and the Senate. NCLB represents the most significant expansion of the federal government into education in our history as it has dramatically increased federal mandates and requirements on states, school districts, and public schools. The Obama administration released its blueprint for revising the Elementary and Secondary Education Act (ESEA) on March 13, 2010.

The primary goals of NCLB are to increase achievement of all students in American public schools; reduce the achievement gap based on race, ethnicity, language, socioeconomic status, and disability; and ensure that students in every public school achieve important learning goals while being educated in safe classrooms by well-prepared teachers.

NCLB is based on the following key principles:

1. *Accountability for results:* By the 2005–2006 school year, each state must test students annually in grades 3 through 8 in reading and math and students in grades 10–12 at least once in reading and math. Every student will be assessed according to each state’s standard for proficiency in reading/language arts, math, and science by the end of the 2013–2014 school year. Student test scores must be made accessible to parents and communities. Test score data must be disaggregated by identified subgroups such as race/ethnicity, socioeconomic status, and disability. Schools that do not demonstrate “adequate yearly progress” (AYP) in student proficiency levels at large and within each of the subgroups must provide students with supplemental services such as free tutoring or after-school assistance.
2. *Parental choice:* Schools must provide parents with a report about the overall achievement of students in the school their child attends, the qualifications of their child’s teacher, and school safety. Parents of children in schools that are not making AYP for at least two consecutive years have the option of transferring their child to an alternate school within the school district. Parents also have the right to transfer their student if the school their child is attending is considered persistently dangerous or if their child has been a victim of a violent crime.
3. *Teacher quality:* Each state must develop a plan to ensure that all teachers of core academic subjects will be highly qualified by the end of the 2005–2006 school year. Highly qualified means that each teacher has full certification and a bachelor’s degree and has passed a state-administered test on core academic subject knowledge.
4. *Scientifically based methods of teaching:* There is an emphasis on the use of scientifically based methods of teaching under NCLB. Schools that fail to meet adequate student achievement goals are required to use scientifically based instructional methods in order to remain open.
5. *Local flexibility:* NCLB grants states and school districts with unprecedented flexibility in how they use federal education funds under NCLB in order to permit school districts to respond to specific local problems.

References and Reading

- ESEA reauthorization: A blueprint for reform.* U.S. Department of Education. Retrieved 6 June 2011, from <http://www2.ed.gov/policy/elsec/leg/blueprint/index.html>.
- Overview and introduction to the No Child Left Behind Act.* U.S. Department of Education. Retrieved 7 June 2001, from <http://www2.ed.gov/nclb/landing.jhtml>.
- The No Child Left Behind Act of 2001.* U.S. Department of Education. Retrieved 6 June 2011, from <http://www2ed.gov/policy/elsec/leg/esea02/index.html>.
- Yell, M. L., & Drasgow, E. (2005). *No child left behind act: A guide for professionals*. Upper Saddle River: Merrill/Prentice Hall.

Elevated, Expansive, or Irritable Mood

► Mania

Elgar, Sybil

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Major Appointments (Institution, Location, Dates)

Sybil Elgar was a pioneer in the education and care of both children and adults with autism both in the United Kingdom and internationally. As Lorna Wing has written, Elgar was “the UK’s first autism-specific teacher and an inspiration to those who knew her, at a time when autism was still ill-defined and widely misunderstood.” In 1964, Sybil Elgar and the newly formed Society for Autistic Children (now the National Autistic Society) managed to purchase premises to open the world’s first residential school for children with autism, in Ealing, West London. Elgar was a pioneer twice over, as she also founded Somerset Court, the first residential community for adults with autism, in Brent Knoll, Somerset, in 1974.

Major Honors and Awards

Sybil Elgar was a founding member of the British National Autistic Society. She was awarded member of the Order of the British Empire (MBE) in 1975.

Landmark Clinical, Scientific, and Professional Contributions

There is some evidence to suggest that the structured approach which forms the basis of TEACCH – one of the main educational

approaches to autism used worldwide today – was inspired, at least in part, by Sybil Elgar’s pioneering work in the United Kingdom. Dr. Eric Schopler, who founded TEACCH in North Carolina in the 1970s, visited the Sybil Elgar School in the United Kingdom in the previous decade and was very impressed by what he saw.

Short Biography

Sybil Lilian Elgar was born in Willesden, North West London, on June 10, 1914. After leaving school, she became a town clerk’s assistant before getting a job as a school secretary. She had no money to go to college, and staying at home to be with her then widowed and unwell mother, she took a Montessori teaching diploma as a correspondence course. Her interest in helping children was first sparked during her Montessori training when a visit to a hospital in London for “severely emotionally disturbed children” in 1958 left her deeply shocked. Elgar revisited the hospital in 1960 and, seeing that nothing had changed, decided to set up her own school, initially in the basement of her home. She developed a structured approach to teaching, giving her pupils clear and simple instructions and visual aids to ensure they understood what was required of them. Her methods ran counter to mainstream educational thought at the time. However, all the children in her class made substantial progress. Demand for her teaching rapidly grew, and in 1964, Elgar bought the premises to open the world’s first residential school for children with autism, in Ealing, West London. She also founded Somerset Court, the first residential community for adults with autism, in Brent Knoll, Somerset, in 1974. Elgar died on January 8, 2007, at the age of 92.

References and Reading

Feinstein, A. (2010). *A history of autism: Conversations with the pioneers*. Oxford, England: Wiley-Blackwell.

Eligibility (for Services Under IDEA/ADA, etc.)

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Definition

“Eligibility” in the context of this encyclopedia refers to qualification to receive reimbursement for or benefits from a variety of private and public programs.

Historical Background

Eligibility issues regarding ASD patients may present in private health care insurance, public health care insurance, education, disability benefits, and antidiscrimination contexts.

Current Knowledge

Private Health Care Insurance

Treatment for ASD can be costly, with annual expenses exceeding \$50,000 (NCSL) and mean lifetime care expenses totaling \$3.2 million (Ganz 2006). As a result, eligibility for private health insurance coverage for ASD-related treatment can be important to people with ASD and their families.

Insurance coverage is especially difficult to obtain for behavioral therapies such as Applied Behavioral Analysis therapy (ABA). Kaiser Permanente, for example, classifies ABA therapy as educational rather than clinical treatment and, thus, denies insurance reimbursement for its cost (Reinke 2008).

In response to insurance company reluctance to cover treatment for ASD, state legislatures have begun to address the issue. At this writing, the legislatures of 35 states and the District of

Columbia have enacted laws mandating some types of insurance coverage for the treatment of ASD. Statutes in 23 of those states – Arizona, Colorado, Connecticut, Florida, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maine, Massachusetts, Missouri, Montana, Nevada, New Hampshire, New Jersey, New Mexico, Pennsylvania, South Carolina, Texas, Vermont, and Wisconsin – make specific reference to treatment for ASD or autism. The remaining dozen mandate coverage for mental health conditions that may include ASD. The types of coverage mandated under these laws vary from diagnostic testing to a variety of therapies and treatment options. Some laws prohibit insurers from setting premium or coinsurance rates based solely on an ASD diagnosis.

State laws, however, are not applicable to all insurance policies. The federal Employee Retirement Income Security Act (ERISA) exempts self-funded, employer-provided health insurance plans from state coverage mandates (ERISA). As a result, in 2009, the United States Congress considered enacting the Autism Treatment Acceleration Act. But, the bill failed to pass either in the house of representatives or the senate. As of this writing, there is no federal mandate that insurers cover treatments or therapies for ASD.

Public Health Insurance: Medicaid

Conventional Medicaid Eligibility

Medicaid is a national health care program funded jointly by the federal and state governments. Adopted in 49 states and the District of Columbia and represented in the fiftieth state, Arizona, by a similar program, Medicaid provides services to the poor. Eligibility is largely limited to those with incomes no higher than the federal poverty level, with slightly higher incomes allowed for women, children, and other groups. Those who meet the income and other resource prerequisites qualify for a wide range of health care benefits, including treatment for ASD.

Services available to those with ASD vary by state. Medicaid insurers in some states cover a wide range of services, including Applied Behavioral Analysis (ABA). In other states, insurers are

more reluctant to cover ABA and other treatment modalities for ASD. In 2003, the Centers for Medicare and Medicaid Services issued a directive that characterized ABA as habilitative rather than rehabilitative and, thus, not mandatory coverage. In response, many state Medicaid agencies have ceased providing reimbursement for ABA (Mauch et al. 2011).

Medicaid "Waiver" Eligibility

All 49 states, the District of Columbia, and Arizona also participate in the Medicaid waiver program. Under these programs, parental financial resource limitations are "waived" for certain services for people under the age of 21 with developmental disabilities or long-term illnesses. These waiver programs cover home- and community-based services (HCBS). A person qualifies for HCBS services if he or she otherwise "would require the level of care provided in a hospital or a nursing facility or intermediate care facility for the mentally retarded" (42 U.S.C. § 1396n(c)).

Thirty states have ASD-specific waiver programs that delineate the specific services (Autism NOW). Connecticut's waiver, for example, covers "residential habilitation, personal supports, respite, clinical behavioral supports, supported employment, job coaching, community transition services, life skills coaching, community transition services or short term crisis stabilization to remain in their own home, family home or other community home."

Patient Protection and Accountable Care Act (PPACA)

By 2014, the PPACA, which congress enacted in 2010, will expand the Medicaid program to cover nearly all people with incomes up to 133% of the federal poverty level. The PPACA will also preclude Medicaid from considering in an eligibility determination resources other than income.

Disability Benefits

The Federal Social Security Program offers two types of disability benefits for which children with ASD may qualify.

Supplemental Security Income (SSI)

The SSI program provides monthly payments to children from birth to age 18. Children whose parents' income and other financial resources are within the statutory limits are eligible for benefits if they are not "engaged in substantial gainful activity" and are diagnosed with an impairment that meets SSI's disability definition for children. Listed impairments include "mental disorders," which encompasses ASD.

At SSI's enactment in 1973, the Social Security Administration (SSA) required for eligibility for benefits that a child's impairment be "comparable in severity" to an impairment that would qualify an adult for benefits. In 1996, congress modified the standard by enacting the Personal Responsibility and Work Opportunity Reconciliation Act of 1996 (PRA). This Act replaced the "comparable severity" standard with a test to determine whether a child suffers a "marked and severe functional limitation." Eligibility for benefits requires two findings. First, the child's impairment must be "medically determinable." Second, the impairment must "meet, medically equal, or functionally equal" a listed impairment (20 CFR §416.92 and §416.926a).

SSA regulations establish six "domains" to determine functional equivalence to a listed impairment: "acquiring and using information," "attending and completing tasks," "interacting and relating with others," "moving about and manipulating objects," "caring for yourself," and "health and physical well-being." A child must exhibit a "marked" limitation in at least two of these categories or an "extreme" limitation in one. Factors relevant to these determinations include "the child's age; the effects of treatment and/or medication; the effect of structured settings; the need for assistive devices or adaptations; school functioning, including attendance; time spent in therapy; the effects of chronic illness; and the combined effects of multiple impairments." An "extreme" limitation exists when the impairment or impairments "very seriously" interfere with the ability to initiate, sustain, or complete activities independently. This status is consistent with a

score three standard deviations below the mean on an applicable standardized test. A “marked” limitation exists when the impairment or impairments “seriously” interfere with those same skills. This status is consistent with a score of at least two but fewer than three standard deviations below the mean on an applicable standardized test.

The first step for determining eligibility for SSI benefits based on ASD, the “medically determinable” inquiry, requires a finding of deficiencies in “reciprocal social interaction and in verbal and nonverbal communication and imaginative activity, and a markedly restricted repertoire of activities and interests” (Ruskell). The second step typically finds “functional equivalence” to the “mental disorders” listing and resolves not on a finding of “extreme” limitation in a single domain but on a “marked” limitation in two domains, most commonly in “attending and completing tasks,” “interacting and relating with others,” “caring for yourself,” and “health and physical well-being.”

Finally, the ASD limitation must have existed for at least 12 months.

Social Security Disability Insurance (SSDI)

SSDI provides benefits to adults who have a disability that began before they reached the age of 22 years. Because they are based on the Social Security earning record of the beneficiary’s parents, the social Security Administration considers SSDI to be a “child’s” benefit. A disabled adult is entitled to SSDI benefits if one of his or her parents is currently receiving Social Security retirement or disability benefits or died after working long enough to qualify for some form of Social Security benefit. In addition, a “child” is eligible for SSDI benefits if he or she received dependent benefits on a parent’s Social Security earnings record prior to reaching the age of 18.

SSDI eligibility differs from SSI eligibility in that the recipient must meet the impairment definition for adults. The adult disability impairments include “autistic disorder and other pervasive developmental disorders.”

Finally the “child” must not be engaged any substantial work.

Education: The Individuals with Disabilities Education Act (IDEA)

IDEA is a federal law that mandates the availability of a Free Appropriate Public Education (FAPE) for all eligible children with disabilities. IDEA defines “disability” as a person “(1) with mental retardation, hearing impairments . . . speech or language impairments, visual impairments . . . serious emotional disturbance . . . orthopedic impairments, *autism*, traumatic brain injury, other health impairments, or specific learning disabilities . . . (2) who needs special education and related services because of his or her disability or disabilities” (IDEA § 802, emphasis supplied). Thus, children with ASD are eligible for IDEA-related services.

IDEA’s regulations define autism as “a developmental disability significantly affecting verbal and nonverbal communication and social interaction, generally evident before age three that adversely affects a child’s educational performance. Other characteristics often associated with autism are engagement in repetitive activities and stereotyped movements, resistance to environmental change or change in daily routines, and unusual responses to sensory experiences.”

A child with ASD is not eligible for IDEA-related services if the “child’s educational performance is adversely affected primarily because the child has an emotional disturbance.” An “emotional disturbance” is “a condition exhibiting one or more of the following characteristics over a long period of time and to a marked degree that adversely affects a child’s educational performance”:

- “An inability to learn that cannot be explained by intellectual, sensory, or health factors”
- “An inability to build or maintain satisfactory interpersonal relationships with peers and teachers”
- “Inappropriate types of behavior or feelings under normal circumstances”
- “A general pervasive mood of unhappiness or depression”

- “A tendency to develop physical symptoms or fears associated with personal or school problems”

IDEA’s regulations also classify schizophrenia as an emotional disturbance.

For those children diagnosed with ASD and not an emotional disturbance, Part C of the IDEA, Infants and Toddlers with Disabilities, provides for developmental services for children from birth to 3 years of age. Part B, Assistance for Education of All Children with Disabilities, mandates educational services for children from ages 3 to 21. The educational requirements of Part B include an individualized education program (IEP) for each child and demand the placement of each child in the least restrictive environment (LRE) possible.

An “appropriate” education must address a child’s specific educational needs. Determining what is appropriate entails several steps. The responsible state actor must conduct an individualized assessment to ascertain a student’s strengths and weaknesses. Next, an IEP team, comprising representative of the school district, a teacher, the child’s parents, and, if appropriate, the child, must identify appropriate goals and objectives for the student and construct an IEP designed to aid the student in meeting the goals and objectives. Finally, the IEP team is charged with identifying the aids and services necessary for the child to succeed in the IEP.

These services consist of “transportation and such developmental, corrective, and other supportive services as are required to assist a child with a disability to benefit from special education.” Specifically, they may include:

- Audiology
- Counseling services
- Early identification and assessment of disabilities in children
- Medical services for diagnosis or evaluation
- Occupational therapy
- Parent counseling and training
- Physical therapy

- Psychological services
- Recreation
- Rehabilitation counseling
- School health services
- Social work services
- Speech pathology
- Transportation

An IEP must include:

- “A statement of the child’s present levels of academic achievement and functional”
- “A statement of measurable annual goals including academic and functional”
- “A description of how the child’s progress toward meeting the annual goals will be measured”
- “A statement of the special education and related services and supplementary aids and services . . . to be provided”
- “An explanation of the extent, if any, to which the child will not participate with nondisabled children in the regular class and in extracurricular and other nonacademic activities”
- “A statement of any individual appropriate accommodations that are necessary to measure the academic achievement and functional performance of the child”
- “The projected date for the beginning of the services and program modifications and the anticipated frequency, location, and duration of those services and modifications”
- “Beginning not later than the first IEP to be in effect when the child is 16 and updated annually thereafter: appropriate measurable post-secondary goals”

IDEA conditions state receipt of federal funding on meeting its minimum requirements. States may not provide fewer services but may provide greater services.

The needs of children vary, and, as a result, children with ASD will be eligible for varying IEPs and services. Their needs and the services to which they are entitled will also likely change over time.

Antidiscrimination: The Americans with Disabilities Act (ADA)

The ADA prohibits discrimination on the basis of disability in employment, state and local governmental services, public accommodations, transportation, and telecommunications. A person is “disabled” if he or she has a physical or mental impairment that substantially limits one or more major life activities, has a history or record of impairment, or is perceived by others as having an impairment.

The ADA does not enumerate the impairments that are “substantially limiting,” leaving that question to the regulators and courts. In the decade and a half following the ADA’s enactment in 1990, the federal courts, including the United States Supreme Court, issued a number of decisions construing the statutory phrase very narrowly. This resulted in the denial of many ADA claims, including claims of those with ASD. In response, congress enacted the Americans with Disabilities Act Amendments Act of 2008 (ADAAA). The ADAAA’s central feature was a command that “the definition of disability in this Act shall be construed in favor of broad coverage of individuals under this Act, to the maximum extent permitted under the terms of the Act.”

Specifically, the ADAAA expanded the definition of “major life activities” by articulating a non-exhaustive list of the activities: “caring for oneself, performing manual tasks, seeing, hearing, eating, sleeping, walking, standing, lifting, bending, speaking, breathing, learning, reading, concentrating, thinking, communicating, and working.” In addition, the Act included “major bodily functions” within “major life activities” and presented a non-exhaustive list of these functions: “functions of the immune system, normal cell growth, digestive, bowel, bladder, neurological, brain, respiratory, circulatory, endocrine, and reproductive functions.”

The ADAAA also mandated that, with the exception of eyeglasses and contact lenses, the determination that impairment substantially limits a major life activity “be made without regard to the ameliorative effects of mitigating measures” such as medication and other “assistive

technology.” Most importantly for people with ASD, the determination must be made without regard to “learned behavioral or adaptive neurological modifications.” A temporary impairment, or one that is “episodic or in remission” qualifies as a disability even when inactive “if it would substantially limit a major life activity when active.” Examples would include posttraumatic stress disorder, epilepsy, or cancer.

On May 24, 2011, new regulations designed to implement the ADAAA and issued by the Equal Employment Opportunity Commission (EEOC), the agency charged with enforcing the ADA, went into effect. Echoing the ADAAA, the regulations provide that “[t]he definition of disability . . . shall be construed broadly, to the maximum extent permitted by the terms of the ADA.” More importantly, the regulations announce that the ADAAA shifts the focus of an ADA claim from whether a disability exists to “whether discrimination occurred.” Furthermore, the regulations provide that the question whether an individual is “substantially limited” in a major life activity “should not demand extensive analysis” and “usually will not require scientific, medical, or statistical analysis.”

Most importantly for those with ASD, the regulations provide the first explicit recognition that autism constitutes an ADA-recognized impairment. Indeed, Section 1630.2(j) (3) (iii) provides that “in virtually all cases,” a number of conditions, including autism, meet the definition of disability. According to the EEOC, “autism substantially limits brain function” and thus “will, at a minimum, substantially limit . . . major life activities.”

As a result of the ADAAA and its accompanying regulations, those diagnosed with ASD are assured eligibility for remedies should they experience discrimination in employment, governmental services, public transportation, public accommodations, or in telecommunications. In the employment context, the ADA prohibits discrimination in recruitment, hiring, promotions, training, pay, social activities, and other employment-related privileges. Governmental services include public education, employment,

transportation, recreation, health care, social services, courts, voting, and town meetings. Public transportation includes city busses, subways, trains, and the like. Public accommodations include businesses and nonprofit service providers that are open to the public. These enterprises include privately operated educational facilities, transportation services, and commercial facilities such as restaurants, retail stores, hotels, movie theaters, private schools, convention centers, doctors' offices, homeless shelters, transportation depots, zoos, funeral homes, day care centers, and recreation facilities including sports stadiums and fitness clubs.

Accommodations accorded those with ASD vary according to a person's impairments and the context. In the educational context (also covered by the Individuals with Disabilities Education Act (IDEA)), accommodations include special education teachers, teacher aides, more frequent feedback from teachers, and help with learning strategies or study skills. Other accommodations include the use of technological aids such as calculators, computer software, and audiobooks.

In the employment context, ASD-related accommodations include the articulation of clear job expectations, mandates that superiors and colleagues communicate in a direct manner, minimization of multitask assignments, and the use of instructional visuals.

Future Directions

Eligibility for private and public health insurance will be subject in the future to the fortunes of the Patient Protection and Affordable Care Act (PPACA). Eligibility for social security, educational, and antidiscrimination benefits will be subject to the priorities and directions of federal and state legislatures.

See Also

- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [PL94-142](#)

References and Reading

- 20C.F.R. (2012). Ch. III, Pt. 404, Sbpt. P., Appen. 1, §12.10.
- 29 Code of Federal Regulations (2012). §1630.2.
- Administration, S. S. (2009). SSR 09-1p: Title XVI: Determining childhood disability under the functional equivalence rule – The “whole child” approach. *Federal Register*, 74(30), 7527. <http://www.ssa.gov/OP-Home/rulings/ssi/02/SSR2009-01-ssi-02.html>.
- Americans with Disabilities Act (2008). 42 U.S.C. §§12101 et seq.
- Autism Now. <http://autismnow.org/funding-and-public-policy/funding-and-public-policy-introduction/>
- Autism Treatment Acceleration Act of 2009, S. 819, H.R. 2413 (2008).
- Barner, A. (2009). Unlocking access to insurance coverage for autism treatment. *Journal of Law, Economics & Policy*, 6, 107–135.
- Bates, M. W. (1994). Free appropriate public education under the individuals with disabilities education act: Requirements, issues, and suggestions. *Brigham Young University Education and Law Journal*, 215–222.
- Breslin, M. A. (2009). No child left behind and the inherent conflict with the individuals with disabilities education act: Leaving special education students further behind. *Albany Government Law Review*, 2, 653–676.
- Caruso, D. (2010). Autism in the U.S.: Social movement and legal change. *American Journal of Law & Medicine*, 36, 483–539.
- Department of Social Services, Department of Developmental Services, Notice of Intent to Seek Three Medicaid Waivers for Individuals with Autism Spectrum Disorders Who Do Not Also Have a Diagnosis of Mental Retardation. (2011). Retrieved July 5, 2012, from <http://www.ct.gov/dds/cwp/view.asp?a=2730&Q=476378>
- Employee Retirement Income Security Act (ERISA) (2012). 29 USC §1001, et. Seq.
- Ganz, M. (2006). The costs of autism. In S. Moldin & J. Rubenstein (Eds.), *Understanding autism: From basic neuroscience to treatment*. New York: CRC Press.
- Hinson, C. (2009). A supreme paradox: Autism spectrum disorder and Rowley misapplication of judicial relic to an unprecedented social epidemic. *Florida A & M University Law Review*, 5, 87–105.
- Holland, C. D. (2010). Autism, insurance, and the IDEA: Providing a comprehensive legal framework. *Cornell Law Review*, 95, 1253–1282.
- IDEA Regulations (2010). § 300.8 Child with a disability. Individuals with Disabilities Education Act (IDEA) (2006). 20 U.S.C. §§ 1400, et seq.
- Mauch, D., Pfefferle, S., Booker, C., Pustell, M., & Levin, J. (2011). *Report on state services to individuals with autism spectrum disorders (ASD), Centers for Medicare & Medicaid Services (CMS) ASD Services Project 2011*. Cambridge, MA: ABT Associates.

- National Conference of State Legislators. (2010). *Insurance coverage for autism*. Washington, DC: NCSL Press.
- Patient Protection and Affordable Care Act (2012). Pub. L. No. 111–148, § 2001(a)(1), 124 Stat. 272–75.
- Regulations to Implement the Equal Employment Provisions of the Americans With Disabilities Act, as Amended. (2011). Retrieved July 5, 2012, from <http://www.federalregister.gov/articles/2011/03/25/2011-6056/regulations-to-implement-the-equal-employment-provisions-of-the-americans-with-disabilities-act-as#p-326>
- Reinke, T. (2008). States increasingly mandate special autism services. *Managed Care*, 17(8), 35–36.
- Ruskell, R. C. (2010). *Social Security Disability Claims Handbook*. §2:33.
- Social Security Act of 1965 (2012). 42 U.S.C. §§ 1396–1396W-5.
- Social Security Administration. (2008). *Disability Evaluation Under Social Security, §12.00 Mental Disorders – Adult*. Retrieved July 5, 2012, from <http://www.ssa.gov/disability/professionals/bluebook/12.00-MentalDisorders-Adult.htm>

ELM Scale-2

► Early Language Milestone Scale

Elopement

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Definition

Elopement behavior, sometimes termed “wandering,” is defined as the leaving of a supervised, safe space by a dependent person, such as a person with Autism Spectrum Disorder (ASD). It refers to incidents lasting longer than a brief period of

running away, which is developmentally normal in toddlers but is rare in the typically developing population, particularly in those over 5 years of age (Anderson et al. 2012).

Historical Background

Historically, elopement was grouped with other “challenging behaviors” commonly encountered among individuals with ASD and/or Intellectual Disability (ID), such as aggressive and disruptive behavior, and received little specific attention (Dekker et al. 2002; Doehring et al. 2014; Matson and Nebel-Schwalm 2007; Matson and Rivet 2008; Murphy et al. 2005). The majority of research specifically focused on elopement behavior has been conducted on populations of individuals with dementia rather than ASD, potentially limiting applicability of findings to the ASD population (Algase et al. 2009; Cipriani et al. 2014; Lai and Arthur 2003).

Within ASD-related literature, the small body of published studies on elopement consist largely of case reports or small case series reporting on efforts to manage the problem using behavior analytic approaches, particularly Applied Behavioral Analysis (ABA), and the use of related interventions such as functional communication training, differential reinforcement of other behaviors, and noncontingent reinforcement (Call et al. 2011; Falcomata et al. 2010; Lang et al. 2010; Perrin et al. 2008; Piazza et al. 1997; Stevenson et al. 2016; Tarbox et al. 2003).

Current Knowledge

Interest in the problem of elopement has increased in the last decade as a result of increasing awareness of its potentially deadly consequences for individuals with ASD. High profile incidents including drownings and traffic accidents prompted calls for more research focused on understanding and preventing elopement (The Autism Wandering Awareness Alerts Response and Education (AWAARE) Collaboration; McIlwain and Fournier 2012). In parallel, research on the causes

of elevated mortality among individuals with ASD (Gillberg et al. 2010; Guan and Li 2017; Mouridsen 2013; Schendel et al. 2016), including one study documenting a 40-fold increased risk of drowning for these individuals, (Guan and Li 2017), pointed toward elopement as a potentially important modifiable risk factor for individuals with ASD.

Increased awareness of the risks of elopement and advocacy by parents and ASD organizations (McIlwain and Fournier 2010) led to the first large-scale study of elopement among individuals with ASD. Surveying over 1200 caregivers of children with ASD recruited via the Interactive Autism Network (IAN), an online research database and autism registry, Anderson and colleagues (2012) reported that 49% of children with ASD over the age of 4 had reported attempts of elopement at least once after the age of 4. Rates in children with ASD were dramatically higher than in their unaffected siblings, particularly among older children. Among survey respondents, 26% reported their children had been missing long enough to cause concern, with 24% of them reported in danger of drowning and 65% in danger of a traffic injury. Elopement risk was correlated with greater ASD severity, with an increased risk of 9% on average for each 10-point increase in Social Responsiveness Scale (SRS) T score. Families reported high levels of stress and lost opportunities to participate in activities outside the home due to elopement, yet only 50% of families reported receiving any advice or guidance on addressing the behavior. Unfortunately, as noted by the authors, the study was not able to provide an estimate on the number of fatalities specifically due to elopement due to inclusion of only living children with ASD.

Subsequent to the study of Anderson and colleagues, two other large-scale epidemiological studies published in 2016 provided population-based estimates of elopement prevalence and correlates of the behavior. Utilizing the Centers for Disease Control (CDC) "Pathways" Survey data, consisting of telephone survey responses from over 4000 parents of children with a developmental disability, including ASD and Intellectual Disability (ID) or Developmental Delay (DD), Rice

and colleagues (2016) reported on rates of elopement behavior occurring within the year prior to parental survey participation, with the highest rate (37.7%) seen among children with ASD and ID, followed by children with ASD but not ID (32.7%) and those with only ID (23.7%). Rice and colleagues also reported an elevated risk of elopement in male children with ASD versus female children with ASD (OR 2.79, CI 1.03–7.57; $P < 0.05$). Children with ASD and ID had the highest use of prevention measures for elopement (40.8%), compared with approximately 1 in 4 children with ASD without ID, or ID without ASD. However, Rice and colleagues noted that despite the high rate of use of prevention measures, details on the use of specific measures and their effectiveness were lacking, as was information on the circumstances and motivations for elopement in different individuals with ASD.

Analyzing the same CDC "Pathways" dataset, Kiely and colleagues (2016) reported that 26.7% of children with ASD, ID/Developmental Disability (DD), or both had eloped within the previous year, with the highest rates among children with ASD and ID (34.6%), followed by ASD only (32.7%) and ID/DD only (22.7%), consistent with the analysis of Rice and colleagues. Kiely and colleagues similarly found that greater ASD severity, as indicated by the Children's Social Behavior Questionnaire (CSBQ), was correlated with elopement behavior, consistent with the findings of Anderson and colleagues (2012).

Despite increasing awareness of the prevalence of EB and associated risks, little evidence has been available to guide families and caregivers in managing the behavior (McIlwain and Fournier 2010). Responding to this knowledge gap, advocacy organizations have provided resources for caregivers such as the National Autism Association's "Big Red Safety Toolkit" (National Autism Association 2014), which includes recommendations for preventative interventions such as door alarms, shoe ID tags, and visual prompts, as well as a recommendation for swimming lessons in order to reduce drowning risk.

At the same time, the use of tracking technologies such as GPS-enabled bracelets has continued to receive interest as a means of alerting

caregivers when individuals with ASD elope (Hayward et al. 2016). In particular, following the tragic 2013 death of Avonte Oquendo during an elopement incident, a 14-year-old boy from Queens, New York, with ASD, New York Senator Charles Schumer pledged federal funding for tracking devices, reflecting hope that widespread use of these devices could be an effective means of locating missing children. Despite this interest, adoption of tracking devices has been limited thus far, as reflected by Rice and colleagues who found only about 3% of parents using them (Rice et al. 2016), potentially due to associated costs as well as limited evidence on their effectiveness (Hayward et al. 2016).

Research into behavioral treatments for elopement also continues to be an area of active investigation, (Boyle et al. 2019; Leahy et al. 2013) with one recent study reporting a substantial reduction in elopement occurrences (Cohen's $d = 1.18$) in a sample of 11 subjects treated at an intensive day treatment clinic for an average of 37 sessions (Call et al. 2017). Overall, however, as reported in a 2017 review of functional analysis and function-based treatments for elopement by Boyle and colleagues (2017), the body of evidence supporting behavioral therapies for elopement is limited, with Boyle and colleagues finding only 12 studies totaling 20 subjects that met criteria for review and noting that "conclusions regarding effectiveness of treatments should be drawn tenuously." Boyle and colleagues noted inherent challenges in the study of elopement from a functional behavioral perspective, in particular that the consequences of the behavior are confounded by the requirement that the clinician retrieves the child before harm occurs and that the behavior itself may be infrequent and therefore unlikely to occur during the period of observation.

To address the knowledge gap in real-world effectiveness of interventions for elopement behavior such as those noted above, Andersen and colleagues (2019) reported on caregiver responses to elopement and elopement patterns, using a sample of over 500 caregivers of children with ASD recruited via IAN. Most respondents reported using multiple types of interventions to prevent elopement, finding this strategy in general

to be effective but burdensome. Tracking devices were used infrequently and not rated as highly effective, whereas some environmental interventions such as fencing and window locks were rated as highly effective. The use of behavioral specialists to address the behavior was also common and rated as effective. Given high rates of off-label psychotropic medication administration reported among individuals with ASD with challenging behaviors (Matson and Neal 2009), the frequency and effectiveness of medications used for elopement was also surveyed. Although off-label use of psychotropic medications to reduce elopement was reported by 16% of respondents, most commonly antipsychotics (29%), no medications were rated as effective in preventing the behavior, and most were associated with high rates of side effects.

In reporting on elopement patterns in this study, the most common locations for the behavior were the home (71%) and parks or outdoor spaces (49%). The most commonly associated motivations included anxious situations (43%) or running or exploring for enjoyment (41%). Similar to prior reports, the frequency of elopement behavior was associated with greater levels of ASD severity, as indicated by higher SRS scores, comorbid ID, Language Disorder (LD), and other challenging behaviors including aggression, self-injury, and disruptive behavior. Interestingly, children with ASD and comorbid ADHD were found to be significantly more likely to elope due to having "too much energy" ($P < 0.001$), whereas those with comorbid Anxiety Disorder, NOS were significantly more likely to elope from "stressful" environments ($P < 0.001$), supporting the hypothesis that motivations underlying elopement behavior vary and that different approaches to preventing the behavior may be required depending on the individual.

Future Directions

High priorities for preventing elopement and related injury or death include improving understanding of its causes and the identification of effective measures to prevent the behavior.

Significant advances have been made in the last decade in our understanding elopement's prevalence and correlates, while evidence on the relative effectiveness of different interventions for elopement remains significantly limited. Available research suggests that caregivers have responded to this lack of evidence by employing whatever measures are available to them to prevent their loved ones with ASD from suffering elopement-related death or injury, but that these efforts come at a substantial cost in terms of daily burden and stress.

Going forward, more effective tools to identify high risk individuals and prevent or respond to elopement among individuals with ASD are greatly needed. In addition, efforts to overcome barriers to implementing interventions that have some evidence of effectiveness such as fencing and window locks, may reduce the risks of injury and death among this population whose families already shoulder significant economic burdens associated with the disorder (Lavelle et al. 2014). Thankfully, with the addition of an ICD billing code for "wandering in diseases classified elsewhere" (Z91.83), reimbursement for elopement behavior in individuals with ASD, ID, and dementia may be possible, offering hope that families will be able to meet some of the expense associated with preventing and treating the behavior. Improved awareness of the prevalence and risks of elopement on the part of clinicians will also be essential in recognizing the behavior and offering families guidance and treatment strategies.

At the public health level, efforts to mobilize existing resources and develop new strategies to protect children who elope will continue to be high priorities. Community agencies such as local police departments and school districts should continue to work with families to identify individuals at high risk for elopement, develop preventative strategies, and respond appropriately when elopement occurs. Finally, continued efforts by organizations such as the National Autism Association (2014) and the National Center for Missing and Exploited Children (2019) to both provide families with resources on managing elopement and advocate for more research on

this important clinical problem will be essential to creating a future where these vulnerable individuals are safe from harm.

References and Reading

- Algase, D. L., Antonakos, C., Beattie, E. R., Beel-Bates, C. A., & Yao, L. (2009). Empirical derivation and validation of a wandering typology. *Journal of the American Geriatrics Society*, 57(11), 2037–2045.
- Andersen, A. M., Law, J. K., Marvin, A. R., & Lipkin, P. H. (2019). Elopement patterns and caregiver strategies. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03961-x>.
- Anderson, C., Law, J. K., Daniels, A., Rice, C., Mandell, D. S., Hagopian, L., & Law, P. A. (2012). Occurrence and family impact of elopement in children with autism spectrum disorders. *Pediatrics*, 130(5), 870–877. <https://doi.org/10.1542/peds.2012-0762>.
- Boyle, M. A., & Adamson, R. M. (2017). Systematic review of functional analysis and treatment of elopement (2000–2015). *Behavior Analysis in Practice*, 10(4), 375–385.
- Boyle, M. A., Keenan, G., Forck, K. L., & Curtis, K. S. (2019). Treatment of elopement without blocking with a child with autism. *Behavior Modification*, 43(1), 132–145.
- Call, N. A., Pabico, R. S., Findley, A. J., & Valentino, A. L. (2011). Differential reinforcement with and without blocking as treatment for elopement. *Journal of Applied Behavior Analysis*, 44(4), 903–907.
- Call, N. A., Alvarez, J. P., Simmons, C. A., Lomas Mevers, J. E., & Scheithauer, M. C. (2017). Clinical outcomes of behavioral treatments for elopement in individuals with autism spectrum disorder and other developmental disabilities. *Autism*, 21(3), 375–379. <https://doi.org/10.1177/1362361316644732>.
- Cipriani, G., Lucetti, C., Nuti, A., & Danti, S. (2014). Wandering and dementia. *Psychogeriatrics*, 14(2), 135–142.
- Dekker, M. C., Koot, H. M., Ende, J. v. d., & Verhulst, F. C. (2002). Emotional and behavioral problems in children and adolescents with and without intellectual disability. *Journal of Child Psychology and Psychiatry*, 43(8), 1087–1098.
- Doehring, P., Reichow, B., Palka, T., Phillips, C., & Hagopian, L. (2014). Behavioral approaches to managing severe problem behaviors in children with autism spectrum and related developmental disorders: A descriptive analysis. *Child and Adolescent Psychiatric Clinics of North America*, 23(1), 25–40. <https://doi.org/10.1016/j.chc.2013.08.001>.
- Falcomata, T. S., Roane, H. S., Feeney, B. J., & Stephenson, K. M. (2010). Assessment and treatment of elopement maintained by access to stereotypy. *Journal of Applied Behavior Analysis*, 43(3), 513–517.

- Gillberg, C., Billstedt, E., Sundh, V., & Gillberg, I. C. (2010). Mortality in autism: A prospective longitudinal community-based study. *Journal of Autism and Developmental Disorders*, 40(3), 352–357. <https://doi.org/10.1007/s10803-009-0883-4>.
- Guan, J., & Li, G. (2017). Injury mortality in individuals with autism. *American Journal of Public Health*, 107(5), 791–793. <https://doi.org/10.2105/AJPH.2017.303696>.
- Hayward, B., Ransley, F., & Memery, R. (2016). GPS devices for elopement of people with autism and other developmental disabilities: A review of the published literature. *Journal of Policy and Practice in Intellectual Disabilities*, 13(1), 69–74. <https://doi.org/10.1111/jppi.12148>.
- Kiely, B., Migdal, T. R., Vettam, S., & Adesman, A. (2016). Prevalence and correlates of elopement in a nationally representative sample of children with developmental disabilities in the United States. *PLoS One*, 11(2), e0148337. <https://doi.org/10.1371/journal.pone.0148337>.
- Lai, C. K., & Arthur, D. G. (2003). Wandering behaviour in people with dementia. *Journal of Advanced Nursing*, 44(2), 173–182.
- Lang, R., Davis, T., O'Reilly, M., Machalicek, W., Rispoli, M., Sigafoos, J., . . . Regester, A. (2010). Functional analysis and treatment of elopement across two school settings. *Journal of Applied Behavior Analysis*, 43(1), 113–118.
- Lavelle, T. A., Weinstein, M. C., Newhouse, J. P., Munir, K., Kuhlthau, K. A., & Prosser, L. A. (2014). Economic burden of childhood autism spectrum disorders. *Pediatrics*, 133(3), e520–e529. <https://doi.org/10.1542/peds.2013-0763>.
- Lehardy, R. K., Lerman, D. C., Evans, L. M., O'Connor, A., & Lesage, D. L. (2013). A simplified methodology for identifying the function of elopement. *Journal of Applied Behavior Analysis*, 46(1), 256–270. <https://doi.org/10.1002/jaba.22>.
- Matson, J. L., & Neal, D. (2009). Psychotropic medication use for challenging behaviors in persons with intellectual disabilities: An overview. *Research in Developmental Disabilities*, 30(3), 572–586. <https://doi.org/10.1016/j.ridd.2008.08.007>.
- Matson, J. L., & Nebel-Schwalm, M. (2007). Assessing challenging behaviors in children with autism spectrum disorders: A review. *Research in Developmental Disabilities*, 28(6), 567–579.
- Matson, J. L., & Rivet, T. T. (2008). Characteristics of challenging behaviours in adults with autistic disorder, PDD-NOS, and intellectual disability. *Journal of Intellectual & Developmental Disability*, 33(4), 323–329. <https://doi.org/10.1080/13668250802492600>.
- McIlwain, L., & Fournier, W. (2010). Wandering and autism: The need for data and resources. *National Autism Association*. Retrieved from https://iacc.hhs.gov/meetings/iacc-meetings/2010/full-committee-meeting/october22/slides_fournier_mcilwain_102210.pdf
- McIlwain, L., & Fournier, W. (2012). Lethal outcomes in autism spectrum disorders (ASD) wandering/elopement. *National Autism Association*, January. Retrieved from http://nationalautismassociation.org/wp-content/uploads/2012/01/Lethal-Outcomes-In-Autism-Spectrum-Disorders_2012.pdf
- Mouridsen, S.-E. (2013). Mortality and factors associated with death in autism spectrum disorders: a review. *American Journal of Autism*, 1, 17–25.
- Murphy, G. H., Beadle-Brown, J., Wing, L., Gould, J., Shah, A., & Holmes, N. (2005). Chronicity of challenging behaviours in people with severe intellectual disabilities and/or autism: A total population sample. *Journal of Autism and Developmental Disorders*, 35(4), 405–418.
- National Autism Association. (2014). Big red safety toolkit. Retrieved from <http://nationalautismassociation.org/docs/BigRedSafetyToolkit.pdf>
- National Center for Missing and Exploited Children. (2019). Autism wandering tips. Retrieved from <http://www.missingkids.com/theissues/autism>
- Perrin, C. J., Perrin, S. H., Hill, E. A., & DiNovi, K. (2008). Brief functional analysis and treatment of elopement in preschoolers with autism. *Behavioral Interventions: Theory & Practice in Residential & Community-Based Clinical Programs*, 23(2), 87–95.
- Piazza, C. C., Hanley, G. P., Bowman, L. G., Ruyter, J. M., Lindauer, S. E., & Saiontz, D. M. (1997). Functional analysis and treatment of elopement. *Journal of Applied Behavior Analysis*, 30(4), 653–672. <https://doi.org/10.1901/jaba.1997.30-653>.
- Rice, C. E., Zablotzky, B., Avila, R. M., Colpe, L. J., Schieve, L. A., Pringle, B., & Blumberg, S. J. (2016). Reported wandering behavior among children with autism spectrum disorder and/or intellectual disability. *The Journal of Pediatrics*, 174, 232–239.
- Schendel, D. E., Overgaard, M., Christensen, J., Hjort, L., Jorgensen, M., Vestergaard, M., & Parner, E. T. (2016). Association of psychiatric and neurologic comorbidity with mortality among persons with autism spectrum disorder in a Danish population. *JAMA Pediatrics*, 170(3), 243–250. <https://doi.org/10.1001/jamapediatrics.2015.3935>.
- Stevenson, M. T., Ghezzi, P. M., & Valenton, K. G. (2016). FCT and delay fading for elopement with a child with autism. *Behavior Analysis in Practice*, 9(2), 169–173. <https://doi.org/10.1007/s40617-015-0089-5>.
- Tarbox, R. S., Wallace, M. D., & Williams, L. (2003). Assessment and treatment of elopement: A replication and extension. *Journal of Applied Behavior Analysis*, 36(2), 239–244. <https://doi.org/10.1901/jaba.2003.36-239>.
- The Autism Wandering Awareness Alerts Response and Education (AWAARE) Collaboration. Retrieved from <https://awaare.nationalautismassociation.org/>

Weblinks

Autism Society Safe and Sound Initiative. <https://www.autism-society.org/living-with-autism/how-the-autism-society-can-help/safe-and-sound/>

Autism Speaks Safety Products. <https://www.autismspeaks.org/safety-products-and-services>

National Center for Missing and Exploited Children's. Autism wandering tips. http://www.missingkids.com/content/dam/missingkids/pdfs/Autism%20Wandering%20Tips_2017.pdf

The Autism Wandering Awareness Alerts Response and Education (AWAARE) Collaboration. <http://awaare.nationalautismassociation.org/>

The National Autism Association's. Big red safety toolkit. <http://nationalautismassociation.org/docs/BigRedSafetyToolkit.pdf>

ELS

► Test of Early Language Skill

ELS Checklist

► Test of Early Language Skill

Emancipatory Autism Studies

► Critical Autism Studies

Embedded Figures Test (EFT)

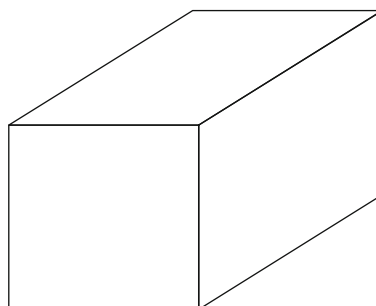
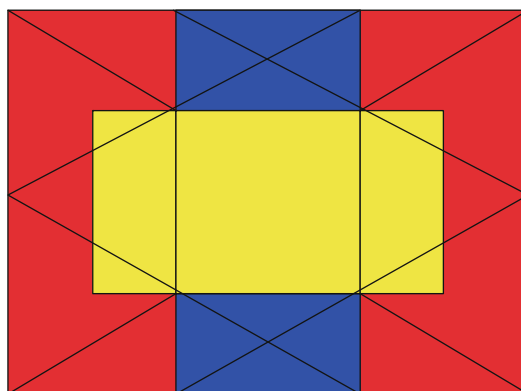
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Description

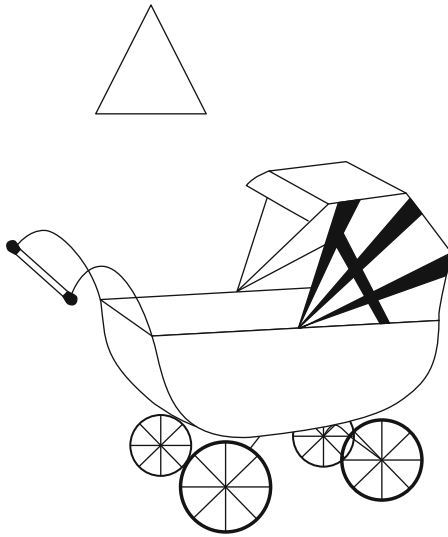
The Embedded Figures Test (EFT) was designed by Witkin in 1971 to assess his concept of “field dependence – independence” (e.g., Witkin and Goodenough 1981). Good performance on the EFT was taken as a marker of field independence, the ability to disembed information from context

or surrounding gestalt. The test requires the participant to spot a simple form within a more complex figure; the color and form of the latter create a gestalt within which the part is hidden (see Fig. 1). In the Children's EFT, the complex figure is also meaningful (e.g., a pram, within which the triangle to be found is hidden in the hood). Group-administered and short versions are also available.

People with ASD are often extremely good at the EFT, as first demonstrated by Shah and Frith (1983). Fast and accurate performance on this test is thought to reflect the ability to see parts within wholes, to ignore the distracting effect of the gestalt, and to focus on details. Indeed, superior performance on the EFT (compared to age- and IQ-matched comparison group) was one of the first demonstrations of so-called weak central coherence in ASD and remains one of the best replicated findings (see Happé and Frith 2006 for review) (Fig. 2).



Embedded Figures Test (EFT), Fig. 1 The participant's task is to find the simple shape within the complex and camouflaging gestalt



Embedded Figures Test (EFT), Fig. 2 In the children's version of this task, the complex figure within which the target is hidden, is identifiable, adding meaning to the gestalt-to-be-ignored

Historical Background

Witkin (1916–1979) was a founder of the notion of cognitive and learning styles. He proposed the idea that personality could be measured in part by how people perceived their environment. In particular, he attempted to create objective tests (in contrast to questionnaire methods), such as the Rod-and-Frame test, to measure individual differences in reliance on external versus internal frames of reference. The Embedded Figures Test was created by Witkin as a more portable and convenient test designed to measure these same facets of field dependence or independence.

Shah and Frith (1983) were the first to use the Embedded Figures Test with individuals with autism, showing superiority compared to matched comparison groups. This was one of the key findings that led Uta Frith to propose her theory of “weak central coherence” in autism.

See Also

- [Executive Function \(EF\)](#)
- [Global Versus Local Processing](#)
- [Theory of Mind](#)

References and Reading

- Happé, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36, 5–25.
- Shah, A., & Frith, U. (1983). An islet of ability in autistic children: A research note. *Journal of Child Psychology and Psychiatry*, 24, 613–620.
- Witkin, H. A., & Goodenough, D. R. (1981). *Cognitive styles: Essence and origins*. New York: International University Press.
- Witkin, H. A., Oltman, P. K., Raskin, E., & Karp, S. (1971). *A manual for the embedded figures test*. Palo Alto: Consulting Psychologists Press.

Emergency Department Utilization and Autism

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Definition

People with autism spectrum disorder (ASD) (American Psychiatric Association 2000) are susceptible to big emotional swings, and sometimes violent actions toward themselves and those around them, leading to frequent crises. Those with mental breakdowns and/or with acute, severe injuries often were often rushed to emergency departments (ED) for immediate treatments. Researchers have found a steady increase in ED visits by children and adolescents with ASD in the 2000s for both non-psychiatric and psychiatric referrals (McCraig and Burt 2005; Mahajan et al. 2009). This trend has also been documented more recently (Kalb et al. 2018; Liu et al. 2017, 2019) for adolescents and young adults with ASD who had higher documented ED visits compared to their same-age peers without ASD. Obsession with strict routines, stereotypy, self-injurious behavior (SIB) (Sterling et al. 2005), and plus deficits in social communication

make ASD patients prone to emotional instability ever since very early childhood. Physical maturation and the accompanying hormonal surge, among other triggers of various nature, further amplify the adverse consequence from those outbursts among young ASD patients. In addition, other mental and physical health comorbidities coupled with core ASD deficits which create barriers to accessing health care services for individuals with ASD often make those with ASD prone to acute adverse health events requiring treatment in the emergency department (ED) (Liu et al. 2019). These mental and physical comorbidities also lead to high rates of healthcare utilization and contribute to a growing cost for healthcare needs for children and adolescents with ASD. In fact, overall cost of medical care is greater for children with ASD compared to children without ASD (Croen et al. 2006; Leslie and Martin 2007; Peacock et al. 2012).

Historical Background

Non-psychiatric and psychiatric referrals have been increasing at a higher rate when compared to individuals without ASD (McCraig and Burt 2005; Gurney et al. 2006; Mahajan et al. 2009; Liu et al. 2017; Kalb et al. 2018). Deavenport-Saman et al. (2016) found that children and adolescents with ASD had 0.26 more documented visits to the ED when compared to individuals without ASD and that older children with ASD (i.e., school-age compared to preschool-age) were utilizing the ED more. A recent examination of ED utilization in adults with ASD found a similar trend in high utilization of the ED (Nicoliadis et al. 2013). Middle to late adolescence and residing in rural areas appeared to reflect higher rates of ED utilization in comparison to early adolescence and residing in urban areas. In addition, higher ED utilization was observed in females as compared to males with ASD, as well as a significant increase in behavioral health visits in the ED over time. Adolescents with ASD do not appear to be adequately supported through the transition to adulthood as they experience more social and communication difficulties often while dealing

with new-onset mental health conditions. This may be particularly true for older youth, those residing in rural settings, and for adolescent females with ASD.

Current Knowledge

Population Vulnerability

ASD patients typically have deficits in social communication, sensory sensitivities, stereotypical and self-injurious behaviors (SIB), as well as other well-known ASD signature behaviors. These ASD hallmark characteristics contribute to emotional dysregulation for ASD patients starting in early childhood (Samson et al. 2014) and consequently subject them more susceptible to emotional breakdown and physical injuries that often need emergency medical attentions, such as visits to emergency departments. Stereotypy refers to repetitive but typically not goal-directed behaviors (Sterling et al. 2005). Some stereotypical behaviors even not appear to be violent, are often associated with SIB. For example, reparative licking often cause skin damages. Young ASD children seldom cause server bodily-damage to themselves. In contrast, it was often acute flaring of lingering medical problems that leads them to ED.

There have been reports that those with have lingering comorbid physical health concerns, such as epilepsy (Tuchman and Cuccaro 2011; Berg and Plioplys 2012) and gastrointestinal (GI) illnesses (Chaidez et al. 2014; Dinan and Cryan 2017) which often lead to increased ED utilization. In addition, medical conditions such as respiratory problems (Kohane et al. 2012; Ming et al. 2016) and other miscellaneous injuries (Jain et al. 2014; Summers et al. 2017) have high prevalence of ED utilization among people diagnosed with ASD. A majority of children and adolescents with ASD are also diagnosed with multiple comorbid diagnoses, including anxiety, depression, and disruptive behavior disorders (Gotham et al. 2015; Gordon-Lipkin et al. 2018). For some children with ASD, physical maturation and the accompanying hormonal changes in adolescence, among other stressors associated with

adolescence, amplify the adverse consequence from depleted coping abilities among young ASD patients as they grow to face up the rules, disciplines, as well as social pressures from peers as well as teachers and school administrators at schools, even parents at home. The transition to adolescence is a particularly stressful time for individuals with ASD and their caregivers as social and societal demands increase, physical and sexual maturity advances, and parental emotional stress often peaks.

Another vulnerability faced by those with ASD is polypharmacy, generally defined as medications prescribed to individuals which are simultaneously filled across two or more classes of medications (Spencer et al. 2013). Given that the majority of children and adolescents with ASD present with comorbid mental and physical health issues, it is not uncommon for physicians to prescribe multiple medications both within and across drug classes, even though the majority of this practice is “off-label.” In particular, prescription of psychotropic medications, especially multiple medications often put those with ASD in great danger of having acute adverse events, often leading to visits to the emergency departments.

Many of the medical conditions among those with ASD may not require care in the ED if managed properly with adequate and regular access to health care services. As with psychiatric care, there are likely several barriers to routine healthcare, management of chronic health concerns, and accessing urgent care for adolescents with ASD. These barriers may include lack of access to health care services, socioeconomic barriers, deficits in communication and social interaction skills, distress or fear responses particularly in novel settings, and sensory sensitivities related to the healthcare setting (Gurney et al. 2006). Additionally, families who have children with ASD are also more likely to experience unmet health care needs, more likely to delay or forgo health care, and more likely to report a high dissatisfaction with care (Kogan et al. 2008; Chiri and Warfield 2012). Taken together, these barriers may contribute to a higher utilization of the ED to identify and manage acute healthcare needs in adolescents with autism.

Non-psychiatric and Psychiatric ED Visits

There has been a steady increase in utilization of the ED for both non-psychiatric and psychiatric referrals by children and adolescents over time (McCraig and Burt 2005; Mahajan et al. 2009). This trend has also been documented in children and adolescents with ASD and has been found to be increasing at a higher rate when compared to individuals without ASD (Gurney et al. 2006).

The most common non-psychiatric problems presenting at ED visits include epilepsy seizures and/or neurological symptoms (9–15%); gastrointestinal disturbances (15%) such as nausea, vomiting, diarrhea, abdominal pain, and constipation (Tuchman et al. 2010; Cohen-Silver et al. 2014; Deavenport-Saman et al. 2016); upper respiratory infections (7%) and viral infections (7%); otitis media (5%); and head or dental injuries (7%) (Deavenport-Saman et al. 2016). Epilepsy has been long found to be highly comorbid with ASD. Studies reported a pooled ASD prevalence of 6.3% among those with epilepsy and prevalence of epilepsy among those with ASD range from 2.7% to 44.4% (Bolton et al. 2011), while the prevalence of epilepsy in the general population is only approximately 0.5–2% (Nguyen and Tellez Zenteno 2009). GI problems has been widely studied and reported in association with ASD, despite the fact that a causal link between these two remains to be established (Chaidez et al. 2014; Dinan and Cryan 2017). Nevertheless, the prevalence of various GI issues has been reported ranging anywhere from 23% to 70% (Chaidez et al. 2014).

As ASD children grow up, those who visited ED more often involve mental health emergencies. While psychiatric-related ED visits have been found to be increasing at a swifter rate than non-psychiatric reasons for referral to the ED (Liu et al. 2017). It is reported that psychiatric-related ED visits among ASD patients have out-paced those of non-ASD population; most recently during year 2013 with about 20% of all ED visits among ages 12–21 years, ASD patients involve psychiatric/behavior health service (Liu et al. 2017). Kalb et al. (2012) found that ED visits for children and adolescents with ASD were more likely to be related to a psychiatric concern than

non-psychiatric concern. In regard to common psychiatric reasons for referral to the ED, Iannuzzi et al. (2014) found that as children with ASD entered elementary, school behavioral issues became a common reason for referral to the ED. However, around the time of entry into middle school, in addition to behavioral issues, mood symptoms, self-injurious behavior, and more significant aggression entered into the top reasons for referral to the ED and remained stable into young adulthood. Wharff et al. (2011) found that children with developmental disabilities, including ASD, were 2.5 times more likely to utilize the ED while waiting for an opening on a psychiatric inpatient facility.

Approximately one-fourth of children and adolescents with ASD have been found to make repeated visits to the ED, and of this 25%, half of the individuals had been to the ED in the 2 weeks prior to the current visit (Cohen-Silver et al. 2014). Of the children and adolescents with ASD who present to the ED, almost 20% have been subsequently admitted to the hospital compared to a rate of 10% in children and adolescents without ASD (Cohen-Silver et al. 2014). Deavenport-Saman et al. (2016) found that children and adolescents with ASD were less likely to be admitted to the hospital from the ED if they arrived at the ED during weekday daytime or evening hours, were female, were English-speaking, and had no insurance. In the same sample, being 6 years old or older, being non-Hispanic, and traveling a greater distance to the ED led to high admittance rates.

Risk Factor and Predictors of ED Visits

A variety of factors, acting independently, or interacting with each other, contribute to the mental and/or physical crises among young ASD patients, leading to higher ED utilization. It is known that a wide spectrum of ASD patients may have different comorbid mental illnesses. Kalb et al. (2012) reported that externalizing mental health illnesses were associated with ED visits and subsequent hospitalizations. On the other hand, Liu et al. (2017, 2019) found that female

adolescents and young adults with more internalizing comorbid illnesses, such as anxiety disorders, mood disorders, and depression, were more likely to visit ED compared to those with comorbid externalizing mental health diagnoses (e.g., ADHD, ODD, psychosis). Females with ASD are more often to have comorbid internalizing disorders but are less likely to be referred to the specialists for assessment than males ((Rucklidge 2010). However, if left undiagnosed, or undertreated, symptoms of these illnesses may exacerbate to the level of level of crises and medical emergencies and ED visits.

Entering high school (age 12–14) and exiting high school (age 18–21), times of transition often put tremendous pressure on female adolescents and young adults with ASD because often during which when new social relationships are to be established and new skills mastered. They struggle with establishing and maintaining appropriate peer relationships (Holtmann et al. 2007). This new layer of difficulty is particularly distressing for female adolescents with ASD as they may have had some success with peers when younger due to shared interests which cannot be sustained secondary to growing social communication deficits (Van Wijngaarden-Cremers et al. 2014).

Many researchers have proposed that increased ED utilization for children and adolescents with ASD may be related to deficits in first-line, community-based, outpatient care (Green et al. 2001; Leichtman et al. 2001). Caregivers of typically developing children and adolescents who reported that their PCP stresses family centeredness, timeliness, and coordinated care have been found to report decreased numbers of visits to the ED (Brousseau et al. 2009). Similarly, caregivers of children and adolescents with ASD report they are more likely to access the ED for healthcare when they perceived their healthcare providers do not listen to their concerns, display cultural insensitivity, do not supply needed information, and do not involve caregivers in decision-making. Therefore, it is less likely that caregivers of youth with ASD access their PCPs for routine visits that are for minor issues as a result of having less assurance for help with acute, emergent,

and/or complex behavioral or health issues (Kogan et al. 2008).

Finally, those living in rural areas have been found to have less access to regular and specialty medical and mental healthcare and therefore may be more likely to present at the ED for mental healthcare. Approximately half of families of children with ASD living in a rural community reported that they experienced problems with the services they received, most often as a result of lack of availability of well-trained providers. Caregivers of individuals with ASD living outside of metropolitan centers in Australia noted a lack of ASD expertise in healthcare providers in rural and remote areas of the country. Thomas and colleagues found that caregivers of children with ASD who lived in rural communities in the United States reported significantly less access to special summer camps and respite care. Another indicator of a paucity of services in rural communities is the documented older mean age of diagnosis of ASD for individuals living in a rural community when compared to those in suburban or urban settings (Mandell et al. 2005). Recent research has begun to examine the efficacy of telehealth remote technology, group-based parent interventions, and training models for PCPs to meet the needs of individuals with ASD and their families in rural settings.

Future Directions

Not only are ASD youth utilizing the ED at disproportionate rates, they are accessing the ED for behavioral health concerns at increasing rates outpacing non-ASD youth. By 2013, over 20% of ASD youth were accessing ED services for a readily identified behavioral health concern (Liu et al. 2017, 2019; Kalb et al. 2018). This appears to be a significant factor as the changes in behavioral health presenting concerns mirror the overall increased ED utilization. Adolescents with ASD who present for emergent psychiatric care appear to have difficulty accessing community-based mental health services. Children and adolescents with ASD have been documented as having significant difficulty accessing specialty medical and

mental health care when compared with other youth with disability. Better access to behavioral health services designed for adolescents with ASD, such as social skills training and specialized care coordination, appears to be a critical need. Greater understanding of service utilization before and after ED visits for youth with ASD is needed to craft more impactful interventions for individuals at risk.

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.). Washington, DC: APA Press.
- Berg, A. T., & Plioplys, S. (2012). Epilepsy and autism: Is there a special relationship? *Epilepsy & Behavior*, 23(3), 193–198. <https://doi.org/10.1016/j.yebeh.2012.01.015>.
- Bolton, P. F., Carcani-Rathwell, I., Hutton, J., Goode, S., Howlin, P., & Rutter, M. (2011). Epilepsy in autism: Features and correlates. *The British Journal of Psychiatry*, 198(4), 289–294.
- Brousseau, D. C., Gorelick, M. H., Hoffmann, R. G., Flores, G., & Nattinger, A. B. (2009). Primary care quality and subsequent emergency department utilization for children in Wisconsin Medicaid. *Academic Pediatrics*, 9, 33–39.
- Chaidez, V., Hansen, R. L., & Hertz-Picciotto, I. (2014). Gastrointestinal problems in children with autism, developmental delays or typical development. *Journal of Autism and Developmental Disorders*, 44(5), 1117–1127.
- Chiri, G., & Warfield, M. E. (2012). Unmet need and problems accessing core health care services for children with autism spectrum disorder. *Maternal and Child Health Journal*, 16(5), 1081–1091.
- Cohen-Silver, J. H., Muskat, B., & Ratnapalan, S. (2014). Autism in the emergency department. *Clinical Pediatrics*, 53(12), 1134–1138.
- Deavenport-Saman, A., Lu, Y., Smith, K., & Yin, L. (2016). Do children with autism overutilize the emergency department? Examining visit urgency and subsequent hospital admissions. *Maternal and Child Health Journal*, 20, 306–314.
- Dinan, T. G., & Cryan, J. F. (2017). Gut-brain axis in 2016: Brain-gut-microbiota axis – Mood, metabolism and behaviour. *Nature Reviews. Gastroenterology & Hepatology*, 14(2), 69–70. <https://doi.org/10.1038/nrgastro.2016.200>.
- Gordon-Lipkin, E., Marvin, A. R., Law, J. K., & Lipkin, P. H. (2018). Anxiety and mood disorder in children with autism spectrum disorder and ADHD. *Pediatrics*, 141(4). <https://doi.org/10.1542/peds.2017-1377>.

- Gotham, K., Brunwasser, S. M., & Lord, C. (2015). Depressive and anxiety symptom trajectories from school age through young adulthood in samples with autism spectrum disorder and developmental delay. *Journal of the American Academy of Child and Adolescent Psychiatry*, 54(5), 369–376. e363.
- Green, J., Kroll, L., Imrie, D., Frances, F. M., Begum, K., Harrison, L., & Anson, R. (2001). Health gain and outcome predictors during inpatient and related day treatment in child and adolescent psychiatry. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40(3), 325–332.
- Gurney, J. G., McPheeters, M. L., & Davis, M. M. (2006). Parental report of health conditions and health care use among children with and without autism. *Archives of Pediatrics and Adolescent Medicine*, 160, 825–830.
- Holtmann, M., Bolte, S., & Poustka, F. (2007). Autism spectrum disorders: Sex differences in autistic behaviour domains and coexisting psychopathology. *Developmental Medicine and Child Neurology*, 49(5), 361–366.
- Iannuzzi, D. A., Cheng, E. R., Broder-Fingert, S., & Bauman, M. L. (2014). Brief report: Emergency department utilization by individuals with autism. *Journal of Autism and Developmental Disorders*, 45, 1096–1102.
- Jain, A., Spencer, D., Yang, W., Kelly, J. P., Newschaffer, C. J., Johnson, J., ... Dennen, T. (2014). Injuries among children with autism spectrum disorder. *Academic Pediatrics*, 14(4), 390–397. <https://doi.org/10.1016/j.acap.2014.03.012>.
- Kalb, L. G., Stuart, E. A., Freedman, B., Zablotzky, B., & Vasa, R. (2012). Psychiatric-related emergency department visits among children with an autism spectrum disorder. *Pediatric Emergency Care*, 28, 1269–1276.
- Kalb, L. G., Stuart, E. A., & Vasa, R. A. (2018). Characteristics of psychiatric emergency department use among privately insured adolescents with autism spectrum disorder. *Autism*, 1362361317749951.
- Kogan, M. D., Strickland, B. B., Blumberg, S. J., Singh, G. K., Perrin, J. M., & van Dyck, P. C. (2008). A national profile of the health care experiences and family impact of autism spectrum disorder among children in the United States, 2005–2006. *Pediatrics*, 122(6), e1149–e1158.
- Kohane, I. S., McMurry, A., Weber, G., MacFadden, D., Rappaport, L., Kunkel, L., ... Churchill, S. (2012). The co-morbidity burden of children and young adults with autism spectrum disorders. *PLoS One*, 7(4), e33224. <https://doi.org/10.1371/journal.pone.0033224>.
- Leichtman, M., Leichtman, M. L., Barber, C. C., & Neese, D. T. (2001). Effectiveness of intensive short-term residential treatment with severely disturbed adolescents. *The American Journal of Orthopsychiatry*, 72(2), 227–235.
- Liu, G., Pearl, A. M., Kong, L., Brown, S. L., Ba, D., Leslie, D. L., & Murray, M. J. (2017). A profile on emergency department utilization in adolescents and young adults with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 47(2), 347–358. PMID: 31414259.
- Liu, G., Pearl, A. M., Kong, L., Leslie, D. L., & Murray, M. J. (2019). Risk factors for emergency department utilization among adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49(11), 4455–4467. PMID: 27844247.
- Mahajan, P., Alpern, E. R., Grupp-Phelan, J., Chamberlain, J., Dong, L., Holubkov, R., et al. (2009). Epidemiology of psychiatric-related visits to emergency departments in a multicenter collaborative research pediatric network. *Pediatric Emergency Care*, 25(11), 715–720.
- Mandell, D. S., Novak, M. M., & Zubritsky, C. D. (2005). Factors associated with age of diagnosis among children with autism spectrum disorders. *Pediatrics*, 116(6), 1480–1486.
- Marcus, L., Kuncie, L. J., & Schloper, E. (1997). Working with families. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and developmental disorders* (2nd ed., pp. 631–649). New York: Wiley.
- McCraig, L. F., & Burt, C. W. (2005). *National hospital ambulatory medical care survey: 2003 emergency department summary* (Vol. 358). Hyattsville: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics.
- Ming, X., Patel, R., Kang, V., Chokroverty, S., & Julu, P. O. (2016). Respiratory and autonomic dysfunction in children with autism spectrum disorders. *Brain Dev*, 38(2), 225–232. <https://doi.org/10.1016/j.braindev.2015.07.003>.
- Nicoliadis, C., Raymaker, D., McDonald, K., Dern, S., Boisclair, W. C., Ashkenazy, E., & Baggs, A. (2013). Comparison of healthcare experiences in autistic and non-autistic adults: A cross-sectional online survey facilitated by an academic-community partnership. *Journal of General Internal Medicine*, 28(6), 761–769.
- Rucklidge, J. J. (2010). Gender differences in attention-deficit/hyperactivity disorder. *The Psychiatric Clinics of North America*, 33(2), 357–373.
- Samson, A. C., Phillips, J. M., Parker, K. J., Shah, S., Gross, J. J., & Hardan, A. Y. (2014). Emotion dysregulation and the core features of autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(7), 1766–1772.
- Spencer, D., Marshall, J., Post, B., Kulakodlu, M., Newschaffer, C., Dennen, T., Azocar, F., & Jain, A. (2013). Psychotropic medication use and polypharmacy in children with autism spectrum disorders. *Pediatrics*, 132(5), 833–840.
- Sterling, L., McLaughlin, A., & King, B. H. (2005). Stereotypy and self-injury. In D. G. Amaral, G. Dawson, & D. H. Geschwind (Eds.), *Autism spectrum disorders* (Vol. I, pp. 640–649). New York: Oxford University Press.
- Summers, J., Shahrami, A., Cali, S., D'Mello, C., Kako, M., Palikucin-Reljin, A., ... Lunskey, Y. (2017). Self-injury in autism spectrum disorder and intellectual disability: Exploring the role of reactivity to pain and sensory input. *Brain Sciences*, 7(11) 140.

- Tuchman, R., & Cuccaro, M. (2011). Epilepsy and autism: Neurodevelopmental perspective. *Current Neurology and Neuroscience Reports*, 11(4), 428–434.
- Van Wijngaarden-Cremers, P. J., van Eeten, E., Groen, W. B., Van Deurzen, P. A., Oosterling, I. J., & Van der Gaag, R. J. (2014). Gender and age differences in the core triad of impairments in autism spectrum disorders: A systematic review and meta-analysis. *Journal of Autism and Developmental Disorders*, 44(3), 627–635.
- Wharff, E. A., Ginnis, K. B., Ross, A. M., & Blood, E. A. (2011). Predictors of psychiatric boarding in the pediatric emergency department. *Pediatric Emergency Care*, 27, 483–489.

Emergent Literacy

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Synonyms

[Early literacy](#); [Preliteracy](#)

Definition

Emergent literacy refers to the many literacy-rich activities children participate in prior to formal reading and writing instruction from birth to about 5 years of age. Specifically, emergent literacy has been defined as “the reading and writing behaviors of young children before they become readers and writers in the conventional sense” (Justice 2006, p. 3). Examples of emergent literacy activities include engaging in shared storybook reading, pretending to write or draw, incorporating literacy themes into play, and engaging in oral wordplay such as rhyming. Shared storybook reading is arguably the most common emergent literacy activity for many children. Parents read to children who are very young before they can verbally participate. Parents often engage in scaffolding or supportive behaviors during emergent literacy activities. Through scaffolding, parents adapt the experience to match the child’s growing abilities, thus supporting the child’s participation. For typically developing

children, emergent literacy instruction is most effective when it is targeted authentically instead of during contrived instructional “skill and drill” activities (Price and Ruscher 2006). Emergent literacy is associated with later literacy achievement and the development of other important skills. For example, children who experience more high-quality reading experiences early in childhood have larger vocabularies than children with fewer experiences (see Temple and Snow 2003 for a review). Emergent literacy activities provide a rich context for learning and practicing joint attention skills, which are particularly vulnerable in children with autism spectrum disorders. Historically, emergent literacy activities were withheld from some children who did not demonstrate signs of readiness, including oral language abilities and behavioral precursors. However, there is no evidence to suggest that any child should be excluded from emergent literacy activities. Because children with autism spectrum disorders are at risk for reduced opportunity for literacy achievement, providing rich and diverse emergent literacy activities is one way to support the literacy access for this population.

See Also

- [Literacy](#)
- [Reading](#)
- [Shared Storybook Reading](#)

References and Reading

- Justice, L. M. (Ed.). (2006). *Clinical approaches to emergent literacy intervention*. San Diego: Plural Publishing.
- Price, L. H., & Ruscher, K. Y. (2006). Fostering phonological awareness using shared book reading and an embedded explicit approach. In A. van Kleeck (Ed.), *Sharing books and stories to promote language and literacy* (pp. 15–76). San Diego: Plural Publishing.
- Temple, J. D., & Snow, C. E. (2003). Learning words from books. In A. van Kleeck, S. Stahl, & E. Bauer (Eds.), *On Reading books to children: Parents and teachers*. Mahwah: Lawrence Erlbaum.
- van Kleeck, A. (Ed.). (2006). *Sharing books and stories to promote language and literacy*. San Diego: Plural Publishing.
- van Kleeck, A., Stahl, S. A., & Bauer, E. B. (Eds.). (2003). *On reading books to children: Parents and teachers*. Mahwah: Lawrence Erlbaum Associates.

Emergent Literacy Skills of Preschoolers with Autism

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Synonyms

Early literacy; Reading

Definition

Emergent literacy is the stage of reading development that precedes conventional reading. Children are in the emergent literacy stage from birth to about 6 years old. During this stage, children are being exposed to experiences that will increase their knowledge and abilities in emergent literacy skills. Emergent literacy skills include alphabet knowledge, print-concept knowledge, phonological awareness, and narrative skills. For children who are typically developing, researchers have found that emergent literacy skills are strong predictors of later success with reading. There is an emerging body of research on emergent literacy and its role in the acquisition of conventional literacy skills for children with autism. Several common findings have emerged across the literature. First, as a group, children with autism tend to have a discrepant profile of strengths in code-related skills (i.e., alphabet knowledge) but meaning-related skills that seem to lag behind their peers. However, there is significant variance in emergent literacy skills for children with autism. The heterogeneity in emergent literacy has been found to be related to autism severity, oral language, and non-verbal cognition.

See Also

- Academic Skills
- Literacy

References and Reading

- Dynia, J. M., Brock, M. E., Logan, J. A. R., Justice, L. M., & Kaderavek, J. N. (2016). Comparing children with ASD and their peers' growth in print knowledge. *Journal of Autism and Developmental Disorders*, 46(7), 2490–2500. <https://doi.org/10.1007/s10803-016-2790-9>.
- Dynia, J. M., Brock, M. E., Justice, L. M., & Kaderavek, J. N. (2017). Predictors of decoding for children with autism spectrum disorder in comparison to their peers. *Research in Autism Spectrum Disorders*, 37, 41–48. <https://doi.org/10.1016/j.rasd.2017.02.003>.
- National Early Literacy Panel. (2008). *Developing early literacy*. Washington, D.C.: National Institute for Literacy.
- Westerveld, M. F., Trembath, D., Shellshear, L., & Paynter, J. (2016). A systematic review of the literature on emergent literacy skills of preschool children with autism spectrum disorder. *The Journal of Special Education*, 50(1), 37–48. <https://doi.org/10.1177/0022466915613593>.
- Whitehurst, G. J., & Lonigan, C. J. (1998). Child development and emergent literacy. *Child Development*, 69(3), 848–872. <https://doi.org/10.1111/j.1467-8624.1998.tb06247.x>.

Emotion and Decision-Making

- Frontal Lobe Findings in Autism

Emotion Awareness and Skills Enhancement (EASE) Program

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Definition

The Emotion Awareness and Skills Enhancement (EASE) Program is an individual therapy

intervention that was designed to improve emotion regulation skills and overall well-being in adolescents and adults with autism spectrum disorder (ASD).

Historical Background

Many people with ASD have interfering problems with emotional and behavioral control that lead to the use of psychiatric services including therapy, medications, and even inpatient hospitalization. Intense and sudden negative reactions, anxiety and worry, aggression and hostility, and persistently high negative affect are some examples of these emotional and behavioral problems, which are unfortunately very common in ASD, substantially impede capacity to learn and function across all life settings, and affect quality of life for the affected individual and family members. Despite the prevalence of emotional and behavioral concerns in ASD and widespread use of psychiatric services to address these concerns, there are few treatments that impact these behavioral and emotional concerns in ASD with proven effectiveness.

Psychotherapeutic intervention approaches in ASD have predominantly focused on the use of cognitive-behavioral therapy (CBT). Although applied primarily to anxiety in ASD, CBT has been proven effective for a range of concerns in non-ASD populations (Epp and Dobson 2010). CBT is considered an effective treatment for co-occurring anxiety in individuals with ASD who do not have intellectual disability (Shaker-Naeeni et al. 2014). However, CBT protocols for ASD have been developed primarily to treat specific anxiety disorders and, as such, generally do not address many common problems such as explosive behavior, irritability, and depression. In addition, not all individuals respond to CBT, and the improvements tend to be less substantial than observed in non-ASD populations (Kreslins et al. 2015). Given the multiple co-occurring problems seen in this population and concerns about applicability of CBT, a more efficient, and perhaps effective, approach is to target the core processes underlying the clinical phenomena.

Treatments that target a process that is believed to be shared or common across multiple disorders or problems are often referred to as transdiagnostic treatments. Transdiagnostic treatment models appear to be at least as effective as well-established, disorder-specific treatments in reducing symptomatic impairment (in non-ASD samples) (e.g., Barlow et al. 2010). They also have the advantage of not requiring therapists to be familiar with different treatment manuals for every specific disorder and are easier to apply to youth with complex presentations (e.g., youth who present with multiple types of concerns). The possibility that transdiagnostic approaches may have wider application than disorder-specific treatments suggests that these approaches could promote broader dissemination and adoption.

Emotion regulation is a strong candidate to target as a transdiagnostic treatment in ASD. Emotion regulation involves the modulation of arousal and emotional state to promote goal-directed behavior (Thompson 1991). Emotion regulation impairment may underlie many of the social, behavioral, and emotional problems presented by individuals with ASD (Mazefsky et al. 2012, 2013). Individuals with ASD may fail to employ adaptive emotion regulation strategies and instead react impulsively to emotional stimuli, with aggression, extreme social withdrawal, or self-injury (Sofronoff et al. 2007). Such behaviors are often interpreted as deliberate or defiant but may be more accurately understood as reduced capacity to manage emotional states. Furthermore, emotion regulation impairment is thought to underlie internalizing problems in non-ASD populations (Aldao et al. 2010), and it is quite likely that emotion regulation impairment plays a role in the commonly observed anxiety (White et al. 2009), suicidal behavior (Maddox et al. 2017), irritability, and depression (Magnuson and Constantino 2011) in ASD as well. This would suggest that successful treatment of emotion regulation impairment may produce improvement across many different manifestations of emotion regulation impairment in individuals with ASD.

The best approach to address emotion regulation in ASD may include mindfulness.

Mindfulness, taken from Buddhist tradition and defined in a multitude of ways, generally focuses on meditative and present-moment and nonjudgmental awareness of thoughts, emotions, and actions (Baer 2003). These techniques have been integrated into existing therapies, such as CBT, and into the development of new therapies in which mindfulness principles are the primary focus, such as mindfulness-based cognitive therapy (MBCT; Baer 2003). Additionally, mindfulness-based interventions have been tailored toward clinical, community, health, and nonclinical populations for the improvement of symptoms such as anxiety, depression, anger, overall well-being and quality of life, and stress, among other outcomes (see Gotink et al. 2015, for review).

Therefore, the development of EASE was based on existing theories and approaches related to both mindfulness-based and CBT interventions. Dr. Carla Mazefsky, Dr. Susan White, Dr. Kelly Beck, and Dr. Caitlin Conner jointly drafted a new intervention manual, called Emotion Awareness and Skills Enhancement (EASE), in order to apply a blend of these strategies in a manner that would be most effective for ASD. EASE was refined through an iterative process that involved input from key stakeholders, including individuals with ASD, parents of individuals with ASD, and clinicians.

Rationale or Underlying Theory

EASE is based on the premise that many of the behavioral and emotional problems experienced by individuals with ASD result from impaired emotion regulation. Although informed by effective interventions for emotion regulation in non-ASD populations, EASE was developed to address ASD-specific obstacles to effective emotion regulation (see Mazefsky et al. 2013; Mazefsky and White 2014). One of the primary considerations is that individuals with ASD often “react to emotional stimuli intensely without clear goal-directedness...and this may be compounded by poor emotional insight and self-monitoring” (Mazefsky et al. 2013, pp. 683–684). Individuals

with ASD without co-occurring Intellectual Disability also report greater use of less adaptive emotion regulation strategies such as suppression and resignation, which function to avoid emotion and generally tend to be ineffective at reducing distress (e.g., Jahromi et al. 2012; Weiss 2014). Therefore, EASE attempts to address these obstacles with a mindfulness-based approach in which awareness of emotion is cultivated, emotions (whether negative or positive) are acknowledged, and behavior is regulated in the face of intense emotion with mindfulness and/or cognitive strategies.

EASE uses both “bottom-up” [mindful awareness] strategies (e.g., emotion cuing, present awareness), which primarily address affective under-control, cognitive rigidity, and poor insight, and “top-down” strategies drawn from CBT (e.g., cognitive reframing, skills practice).

Given that emotion regulation impairment often manifests in the context of social situations in individuals with ASD, EASE’s treatment targets are addressed therapeutically in social situations. In other words, the social milieu is embedded in the treatment. For example, in learning about how to build tolerance for distress, the youth might practice this first in session with just the therapist and then gradually use the skills in increasingly social settings (e.g., with parents, then at school). Additionally, EASE frequently requires evocation of the emotion regulation impairment in the context of treatment. Social interaction may be used, therefore, to evoke difficulty as well as practice skills.

Goals and Objectives

The overall goal of EASE is to develop skills which allow the individual to manage distress and act in ways that are more adaptive (i.e., goal-consistent) rather than avoiding or reducing the experience of negative emotion. EASE has three primary treatment targets:

(1) Mindfulness and emotion awareness – This includes noticing emotions and body sensations and building the ability to identify changes in emotional intensity by learning the physical cues of intensifying emotions.

(2) Recognition of when emotion becomes dysregulated – This includes understanding the relationship between thoughts, feelings, sensations, and behaviors, as well as identifying the influence on and effect of social challenges.

(3) Providing “tools to calm the chaos” – This includes building tolerance for distress and practicing different strategies for emotion management (which is referred to as “ABCD”: awareness, breathe, change thoughts, temporary distraction).

Treatment Participants

EASE was developed for adolescents and adults who are cognitively able and verbal (i.e., no presence of intellectual disability and verbal IQ of at least 80), because of the cognitive and verbal demands of the therapy. An adaptation of EASE for individuals with ASD and co-occurring Intellectual Disability (EASE-ID) is currently in development. EASE is for individuals who have emotion regulation impairment of any type. This could include problems with depression, anxiety, anger, frustration tolerance, general stress management, or frequent outbursts/meltdowns. It was developed specifically to address the challenges and learning style of individuals with ASD, and thus far EASE has only been applied to individuals with ASD. However, the approaches and content of EASE likely also apply to other populations characterized by emotion regulation impairment. For example, while EASE was developed for individual outpatient therapy settings, a group-based school version of EASE which will include any student with emotion regulation impairment (not solely students with ASD) is also in development.

Treatment Procedures

The EASE program includes 16 50–60-minute sessions over the course of approximately 16 weeks. The treatment is delivered individually by a therapist and supported by parent involvement and online content. Each session begins with a mindful

check-in, review of the weekly practice, agenda setting, skill teaching, skill practice, a mindfulness exercise, and planning for post-session practice. Parents are brought into each session before the mindfulness practice and homework assignment.

There is an online platform to support EASE called emotionCoach or “eCoach.” eCoach is available to both the youth and parent. The hope is that having materials from session, as well as additional information to support the concepts introduced in session, available online at any time will allow individuals to learn and process information at their own pace. The parent is encouraged to use eCoach to learn strategies alongside the child. eCoach includes written summaries of the content for each session, all handouts used during EASE, written mindfulness exercises from EASE, and an audio recording of each mindfulness exercise. There are also some additional tips and frequently asked questions on eCoach. Families are encouraged to preview material on eCoach ahead of sessions if they wish. Ideally, parents are asked to review the eCoach content while their child is in session, to aid synchronous learning.

A key treatment component of EASE involves community sessions that were designed to provide an opportunity to practice the skills being learned in a more natural environment. Community sessions are structured so as to evoke sufficient emotion/distress in the client and support from the therapist for use of regulatory skills while distressed. Inclusion of the community sessions reinforces learning of new skills and improves generalization outside of the clinic. Often the community sessions literally involve leaving the office and going somewhere where social and environmental challenges will be more likely to naturally occur. Based on feedback from families, we believe these sessions are most impactful when they can take place in situations that are highly challenging and relevant to the client’s daily life.

Efficacy Information

Primary data on EASE is from an open trial, indicating that they are feasible, acceptable, and

can be administered to fidelity across sites (Conner et al. 2019). Over 70% of the participants were treatment-responders via clinician-naïve report. Parent report of emotion regulation impairment, irritability/aggression, anxiety, and depression symptoms decreased during treatment with medium-large effect sizes (Conner et al. 2019). An ongoing randomized controlled trial, comparing participants who receive EASE to those who receive an individual supportive therapy, will provide more information on efficacy (ClinicalTrials.gov: NCT03432832).

Outcome Measurement

EASE was developed to improve overall emotional reactivity and regulation as measured by the Emotion Dysregulation Inventory (Mazefsky et al. 2018a, b). Secondary outcomes include changes in specific problematic symptoms such as depression, anxiety, or aggression.

Qualifications of Treatment Providers

EASE therapists should have a background in a field that provides mental health intervention services (clinical psychology, advanced education degrees, rehabilitating sciences, social work, etc.). Therapists who participated in the initial trial of EASE included those with the following backgrounds: clinical psychology doctoral students, licensed psychologists (PsyD and PhD), licensed professional counselor, and master of education. EASE therapists ideally should have some prior experience working with clients with ASD and some familiarity with mindfulness-based interventions and cognitive-behavioral therapy. It is recommended that new EASE therapists receive supervision of their first EASE case and ongoing group supervision or consultation thereafter. EASE includes a variety of recommended readings on emotion regulation in ASD and mindfulness approaches.

See Also

- [Cognitive Behavioral Therapy \(CBT\)](#)
- [Emotion Dysregulation Inventory](#)
- [Emotional Regulation](#)
- [Mindfulness Therapy for Individuals with Autism Spectrum Disorder](#)

References and Reading

- Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30* (2), 217–237. <https://doi.org/10.1016/j.cpr.2009.11.004>.
- Baer, R. A. (2003). Mindfulness training as a clinical intervention: A conceptual and empirical review. *Clinical Psychology: Science and Practice, 10*(1998), 125–143. <https://doi.org/10.1093/clipsy/bpg015>.
- Barlow, D. H., Farchione, T. J., Fairholme, C. P., Ellard, K. K., Boisseau, C. L., Allen, L. B., & May, J. T. E. (2010). *Unified protocol for transdiagnostic treatment of emotional disorders: Therapist guide*. New York: Oxford University Press.
- Conner, C. M., White, S. W., Beck, K. B., Golt, J., Smith, I. C., & Mazefsky, C. A. (2019). Improving emotion regulation ability in autism: The emotional awareness and skills enhancement (EASE) program. *Autism, 23*(5), 1273–1287. <https://doi.org/10.1177/1362361318810709>.
- Epp, A., & Dobson, K. (2010). The evidence base for cognitive-behavioral therapy. In K. Dobson (Ed.), *Handbook of cognitive-behavioral therapies* (3rd ed., pp. 39–73). New York: Guilford Press.
- Gotink, R. A., Chu, P., Busschbach, J. J. V., Benson, H., Fricchione, G. L., & Hunink, M. G. M. (2015). Standardised mindfulness-based interventions in healthcare: An overview of systematic reviews and meta-analyses of RCTs. *PLoS One, 10*(4), 1–17. <https://doi.org/10.1371/journal.pone.0124344>.
- Jahromi, L. B., Meek, S. E., & Ober-Reynolds, S. (2012). Emotion regulation in the context of frustration in children with high functioning autism and their typical peers. *Journal of Child Psychology and Psychiatry, 53*(12), 1250–1258.
- Konstantareas, M. M., & Stewart, K. (2006). Affect regulation and temperament in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 36*(2), 143–154. <https://doi.org/10.1007/s10803-005-0051-4>.
- Kreslins, A., Robertson, A. E., & Melville, C. (2015). The effectiveness of psychosocial interventions for anxiety in children and adolescents with autism spectrum disorder: A systematic review and meta-analysis. *Child and Adolescent Psychiatry and Mental Health, 9*(1), 22. <https://doi.org/10.1186/s13034-015-0054-7>.
- Maddox, B. B., Trubanova, A., & White, S. W. (2017). Untended wounds: Non-suicidal self-injury in adults

- with autism spectrum disorder. *Autism*, 21(4), 412–422.
- Magnuson, K. M., & Constantino, J. N. (2011). Characterization of depression in children with autism spectrum disorders. *Journal of Developmental and Behavioral Pediatrics*, 32(4), 332–340. <https://doi.org/10.1097/DBP.0b013e318213f56c>.
- Mazefsky, C. A., & White, S. W. (2014). Emotion regulation: Concepts & practice in autism spectrum disorder. *Child and Adolescent Psychiatric Clinics of North America*, 23(1), 15–24.
- Mazefsky, C. A., Pelphrey, K. A., & Dahl, R. E. (2012). The need for a broader approach to emotion regulation research in autism. *Child Development Perspectives*, 6(1), 92–97. <https://doi.org/10.1111/j.1750-8606.2011.00229.x>.
- Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(7), 679–688. <https://doi.org/10.1016/j.jaac.2013.05.006>.
- Mazefsky, C. A., Day, T. N., Siegel, M., White, S. W., Yu, L., Pilkonis, P. A., & for the ADDIRC. (2018a). Development of the emotion dysregulation inventory: A PROMIS[®] ing method for creating sensitive and unbiased questionnaires for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(11), 3736–3746. <https://doi.org/10.1007/s10803-016-2907-1>.
- Mazefsky, C. A., Yu, L., White, S. W., Siegel, M., & Pilkonis, P. A. (2018b). The emotion dysregulation inventory: Psychometric properties and item response theory calibration in an autism spectrum disorder sample. *Autism Research*, 11(6), 928–941. <https://doi.org/10.1002/aur.1947>.
- Shaker-Naeni, H., Govender, T., & Chowdhury, U. (2014). Cognitive behavioral therapy for anxiety in children and adolescents with autism spectrum disorder. *British Journal of Medical Practitioners*, 7, a723–a731.
- Sofronoff, K., Attwood, T., Hinton, S., & Levin, I. (2007). A randomized controlled trial of a cognitive behavioural intervention for anger management in children diagnosed with Asperger syndrome. *Journal of Autism and Developmental Disorders*, 37(7), 1203–1214. <https://doi.org/10.1007/s10803-006-0262-3>.
- Thompson, R. A. (1991). Emotional regulation and emotional development. *Educational Psychology Review*, 3(4), 269–307. <https://doi.org/10.1007/BF01319934>.
- Weiss, J. A. (2014). Transdiagnostic case conceptualization of emotional problems in youth with ASD: An emotion regulation approach. *Clinical Psychology: Science and Practice*, 21(4), 331–350.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229. <https://doi.org/10.1016/j.cpr.2009.01.003>.
- White, S. W., Mazefsky, C. A., Dichter, G. S., Chiu, P. H., Richey, J. a., & Ollendick, T. H. (2014). Social-cognitive, physiological, and neural mechanisms underlying emotion regulation impairments: Understanding anxiety in autism spectrum disorder. *International Journal of Developmental Neuroscience*, 39, 22–36. <https://doi.org/10.1016/j.ijdevneu.2014.05.012>.

Emotion Coregulation Processes Between Parents and Their Children with ASD

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Definition

Children's emotion regulation capabilities are strategies that are used to manage (inhibit, enhance, or maintain) emotional experiences (Thompson 1994) and develop within the context of social interactions. Parents facilitate the advancement of their children's emotion regulation through *emotion coregulation* processes. Conceptually, emotion coregulation involves transactional exchanges between parent-child dyads; however, there is a lack of consensus regarding the optimal definition of emotion coregulation. Numerous related terms and definitions have been used to describe dyadic emotion processes between parents and their children. For example, synchrony refers to parents and children matching their behaviors and affect states to regulate emotional arousal (Feldman 2003); parent-driven coregulation involves behaviors that parents engage in to motivate or scaffold children's emotion regulation strategies (Gulsrud et al. 2010; Ting and Weiss 2017), and shared affect describes matched emotion expression between parents and children. In our program of research (Guo et al. 2017a; Valentovich et al.

2018), emotion coregulation is defined as a dynamic process of mutual construction of affective states and regulation of arousal between parents and children (see also Butler and Randall 2013; Tronick 1989).

Emotion coregulation with parents provides children the foundation for the development of their own emotion regulation capabilities. The ability to self-regulate emotional states and behavioral actions begins to emerge in infancy and continues to develop throughout childhood through interactions with important others (Kopp 1982, 1989), with parent-child interactions playing a critical role. During the early years, children's growing self-regulation capabilities require additional support to manage emotional arousal. Parents demonstrate emotion regulation strategies and provide external regulation for young children (Kopp 1989). Children initially depend on their parents to assist in modulating their emotional experiences (e.g., parent physically soothes child by holding her) given young children's limited self-regulation skills. During social interactions, parents and children jointly regulate their emotional experiences through emotion coregulation (Cole et al. 2004; Feldman 2003). Repeated dyadic interactions allow for children to practice effective emotion regulation strategies and for parents to facilitate (e.g., scaffold) children's strategies. As children become older, they gain a wider range of emotion regulation strategies and learn to increasingly rely on their own self-regulation strategies (Calkins and Hill 2007; Kopp 1989).

An important aspect to consider when studying emotion coregulation is the emotional content of parent-child interactions. The emotional content of dyadic interactions, which may include mutual positivity, mutual negativity, or mismatched emotion engagement (e.g., child in negative state and mother in positive state), has been the focus of many studies on neurotypical (NT) children and children with behavioral problems. For example, one study examined parent-NT child shared emotion expression during a semi-structured play activity and children's behavioral outcomes (Lindsey et al. 2013). Results showed that mutual positivity between

these young children and their mothers predicted fewer aggressive behaviors with peers, whereas mutual negativity predicted more aggressive behaviors with peers 8 months later. For parents and their children with behavioral problems, the emotional content of emotion coregulation differs from those of NT dyads. In one study, mother-child dyads of preschool-aged children with behavior problems engaged in more mutual negativity and mismatched emotion engagements relative to dyads of NT children (Cole et al. 2003). Increased mutual positivity and decreased mutual negativity in dyads over time were also found to be related to improvements in behavioral problems. These findings suggest that emotional content is an important aspect of emotion coregulation and children's adaptive functioning. It may be particularly important to include a focus on emotional content when examining emotion coregulation processes in children with developmental challenges.

Emotion coregulation processes between parents and their children with developmental disorders, such as Autism Spectrum Disorder (ASD), require unique consideration. Children with ASD have deficits in social interactions that impact parent-child social exchanges, such as a tendency to focus on objects and impaired joint attention on a shared task (American Psychiatric Association 2015; Dawson et al. 2004). Moreover, children with ASD tend to have impaired emotion regulation capabilities. The role that parents of children with ASD have with respect to teaching key emotion regulation strategies may be particularly challenging, as these children tend to lack adaptive emotion regulation strategies and may instead react with intense emotional responses and poor emotional control (Mazefsky et al. 2013). Children with ASD may not respond to parents' attempts at effective coregulation strategies in a manner analogous to NT children. Thus it is important to gain insight into early emotion coregulation processes between parents and their children with ASD as children are developing their self-regulation abilities. Such knowledge may in turn inform parent-child interventions targeting children's emotion regulation and adaptive functioning.

Historical Background

ASD is a lifelong, neurodevelopmental disorder that is characterized by challenges in social communication and restricted, repetitive behaviors (American Psychiatric Association 2015). In addition to core symptoms, children with ASD also commonly exhibit impairments in behavioral and emotional functioning. Prevalence rates for at least one co-occurring behavioral problem, such as internalizing symptoms (e.g., anxiety and depression) or externalizing symptoms (e.g., aggression and impulsivity), range from approximately 70% to 86% for children with ASD (Chandler et al. 2016; Ooi et al. 2011; Simonoff et al. 2008). Children with ASD also tend to have difficulties with aspects of emotional functioning, such as emotion expression, recognition, and regulation (Konstantareas and Stewart 2006; Uljarevic and Hamilton 2013). Importantly, impairments in children's ability to regulate their reactions to emotionally arousing situations may represent one key factor in the mechanisms that underlie the behavioral problems that children with ASD display (Mazefsky and White 2014).

The ability to regulate emotional experiences across contexts is a significant goal in early child development and is associated with concurrent and overall adaptive functioning (Denham et al. 2003; Eisenberg et al. 1993). Effective emotion regulation strategies (e.g., problem-solving and cognitive reappraisal) are linked to adaptive functioning, whereas ineffective strategies (e.g., physical aggression and avoidance) are associated with a host of emotional, behavioral, and social problems throughout the life span (Aldao et al. 2010; Southam-Gerow and Kendall 2002). Prior research on the emotional challenges that children with ASD face has primarily focused on children's emotion expression and recognition (e.g., Rump et al. 2009; Sasson 2006; Yirmiya et al. 1989); findings show difficulties in both domains. Over the past two decades, emotion regulation in children with ASD has begun to receive more attention (Cai et al. 2018; Mazefsky et al. 2013). Although the body of work investigating emotion regulation capabilities of children with ASD is relatively small, such work

represents important advances in our understanding of emotional functioning in children with ASD.

Research on emotion regulation has examined diagnostic group differences between children with ASD and NT children as well as the relationship between emotion regulation and socioemotional and behavioral outcomes in children with ASD. Overall, children with ASD have greater impairments in emotion processes relative to their NT peers. Specifically, in terms of emotion regulation skills, studies using various methodologies, including behavioral observations, child reports, and parent reports, have demonstrated that children and adolescents with ASD use more ineffective strategies and fewer effective strategies than NT peers across a variety of contexts (e.g., positive and negative situations) (Jahromi et al. 2012; Konstantareas and Stewart 2006; Samson et al. 2015). For example, observations of children during frustrating tasks demonstrated that preschool- and school-aged children with ASD used fewer efficient strategies (e.g., distraction) and more maladaptive strategies (e.g., physical objection) compared to NT peers (Jahromi et al. 2012; Konstantareas and Stewart 2006). Similarly, another study using parent report found that relative to NT peers, school-aged children and adolescents with ASD engaged in increased maladaptive strategies (e.g., repetitive behaviors) and decreased adaptive emotion regulation strategies (e.g., cognitive reappraisal) across positive and negative emotion states (Samson et al. 2015). Moreover, these children were less effective at utilizing strategies such as problem-solving, social support, and distraction.

In addition to the increased challenges that children with ASD encounter in regulating their emotional experiences relative to NT peers, emotional difficulties are also associated with socioemotional and behavioral problems. For example, in one study, parent ratings of emotion regulation skills of their preschool-aged children with ASD were related to children's socioemotional functioning, such that ineffective emotion regulation was associated with decreases in social skills and increases in internalizing and externalizing behaviors 10 months later

(Berkovits et al. 2017). Similarly, in another study, 9- to 12-year-old children with ASD who self-reported using maladaptive emotion regulation strategies, such as self-blame, also had higher levels of depressive symptoms and anxiety (Rieffe et al. 2011).

For children with ASD, early parent-child relationships may be particularly important for the development of efficient emotion self-regulation and for improving maladaptive behaviors. However, parent-child relationships within the context of emotion coregulation have only recently been explored in a handful of empirical studies (e.g., Gulsrud et al. 2010; Hirschler-Guttenberg et al. 2015a; Ting and Weiss 2017). Additionally, fathers are less often included in such research relative to mothers, and few studies have investigated emotion coregulation processes between fathers and their children with ASD (e.g., Hirschler-Guttenberg et al. 2015b). Prior studies on NT dyads suggest that fathers play an important role in children's socioemotional development (e.g., Cabrera et al. 2007; Lindsey et al. 2013). The inclusion of fathers in research on emotion coregulation in dyads of children with ASD may be important in order to expand our understanding of their unique contribution to the development of children's emotion regulation capabilities and adaptive functioning.

Prior work has demonstrated the importance of emotion coregulation, yet our understanding of these dyadic emotion processes for children with ASD and how they relate to their functioning is limited. Thus, the objectives of our program of research were to (a) compare patterns of emotion coregulation in mother-child dyads of children with ASD and NT children (Guo et al. 2017a), (b) examine associations between aspects of emotion coregulation and children's behavioral functioning in mother-child dyads of children with and without ASD (Valentovich et al. 2018), and (c) investigate emotion coregulation processes and children's behavioral functioning in father-child dyads of children with ASD (Guo et al. 2017b). Grounded in dynamic systems theory, our studies used a novel application of the State Space Grid (SSG) method (Lewis et al. 1999), a dyadic micro-level approach, to analyze

parent-child emotion engagements during a semi-structured play activity at home. Advantages of using a dyadic micro-level method are that parent and child behaviors can be analyzed simultaneously, rather than individually, and that momentary shifts between parent-child behaviors and affect states, rather than global behaviors, can be captured.

Current Knowledge

Past and current research indicates that parents shape children's emotion regulation capabilities during dyadic social exchanges by modeling appropriate behaviors, providing emotional coaching (e.g., labeling and validating emotions), teaching specific regulation strategies (e.g., redirecting attention and complex cognitive strategies), and monitoring and responding to children's cues, among others (Morris et al. 2007; Thompson and Meyer 2007). Parents of children with and without ASD engage in similar types of emotion coregulation behaviors; however, they differ in the frequency of behaviors. Parents of children with ASD, for example, use more direct, active strategies (e.g., redirecting and prompting) and engage in more physical behaviors (e.g., physical soothing) relative to parents of NT children (Doussard-Roosevelt et al. 2003; Gulsrud et al. 2010; Hirschler-Guttenberg et al. 2015b). Parents of children with ASD are sensitive to children's cues, and the differences in the frequency of specific strategies used may suggest an awareness of the child's developmental needs. Additionally, parent emotion coregulation behaviors are associated with behavioral outcomes for children with ASD. For example, greater parental scaffolding (e.g., sensitive responses to child) during discussions of negative past events between parents and their school-aged children with ASD was found to be associated with fewer child externalizing behaviors (Ting and Weiss 2017).

Recently, the SSG approach, based on dynamic systems theory, has provided a unique method for examining emotion coregulation in parent-child dyads. The SSG has been used to analyze fine-grained parent and child emotion processes

simultaneously to capture the transactional nature of dyadic interactions, rather than a global examination (Hollenstein 2007; Hollenstein and Lewis 2006). Moreover, the SSG allows for operationalizing emotion coregulation in terms of emotional content and structure of dyadic interactions. Emotional content refers to the ability of dyads to initiate and maintain emotion engagement (i.e., mutual positive, mutual negative, or mismatched interactions). Structure refers to the emotional variability of interactions (i.e., the range of emotional interactions, the shifts of emotions, and perseveration in an emotion state) (Granic et al. 2007; Hollenstein et al. 2004).

Research utilizing the SSG has demonstrated that both the emotional content and structure of emotion coregulation are associated with children's behavioral outcomes. For example, Hollenstein et al. (2004) analyzed interactions between parents and their high-risk children during a series of activities, including discussions of problems that cause conflict. Results indicated that decreased dyadic emotional variability (structure) when children were in kindergarten predicted higher levels of children's externalizing behaviors in first grade. Lunkenheimer et al. (2011) assessed parent-child emotion coregulation in a challenging block design task at home when children were 3 years old. The results showed that the interaction between dyadic parent-child emotional variability (structure) and positive affect predicted lower externalizing problems in children at age 5.5 years.

This method previously has been used to examine dyads of NT children and children with behavioral problems. Our lab is the first to use the SSG to examine emotion coregulation processes in families raising children with ASD (Guo et al. 2017a; Valentovich et al. 2018). In our recent studies, the emotional content and structure of emotion coregulation were examined in dyads of children with ASD and dyads of NT children utilizing the SSG approach. Mothers and their preschool-aged children completed the Three Boxes procedure (Tamis-LeMonda et al. 2004; Vandell 1979), a 10-min semi-structured play activity, in their home. During the activity, mothers and their children were given three

numbered boxes that contained toys, and mothers were asked to open the boxes in sequential order. This procedure reflects activities that mothers and their children typically engage in and reliably elicits maternal and child behaviors. Play sessions were videotaped for later coding of emotion engagement states of mothers and children. Children's internalizing and externalizing behaviors were also assessed using mother-reported Vineland Adaptive Behavior Scales (Sparrow et al. 2005) scores.

A novel coding scheme was developed to capture emotion engagements, which were predicated on behaviors, body postures, attention, facial expressions, and vocalizations, in both families of NT children and children with ASD. Mothers and children were coded separately for low, medium, and high levels of positive engagement, negative engagement, and disengagement states; children were also coded for object engagement (for detailed information on the coding schemes, see Guo et al. 2017a). Mangold International's INTERACT 9.47 (Mangold 2007) software program was used to code engagement states in 5-second intervals. The separate codes were merged for each dyad to create dyadic engagement states: *mutual positive engagement* (mother and child in any positive engagement state), *mutual negative engagement* (mother and child in any negative engagement or disengagement state), *mismatched engagement* (mother in any negative engagement or disengagement state and child in any positive engagement state; mother in any positive engagement state and child in any negative engagement or disengagement state), and *child-object* (mother in any engagement state and child in object engagement). The dyadic engagement codes were exported to the SSG software (State Space Grid GridWare 1.1.; Lamey et al. 2004) to obtain the emotional content (i.e., initiating and sustaining dyadic engagement states) and the structure (i.e., range of emotional interactions, the shifts of emotions, and perseveration in an emotion state) of the interactions.

The first goal of our research was to compare the emotional structure and content of interactions between mother-child dyads of children with ASD and dyads of NT children (Guo et al.

2017a). Results indicated that in terms of the structure, dyads of children with ASD engaged in greater emotional variability (i.e., a wider range of emotional interactions, greater shifts of emotions, and less time spent in an emotion state) relative to dyads of NT children. In terms of emotional content, dyads of children with ASD initiated mutual positive, mismatched, and child-object engagements more frequently and spent more time in mismatched and child-object engagements relative to NT dyads, whereas dyads of NT children spent more time in mutual positive engagements relative to dyads of children with ASD. Results suggest that high emotional variability in a low-stress context may not imply adaptive interactions for children with ASD but rather suggests difficulty maintaining positive interactions with their mothers.

The second goal of our research was to investigate whether interactions between emotional variability (structure) and mutual positive/negative engagements (content) of emotion coregulation were associated with maladaptive behaviors in children with ASD (Valentovich et al. 2018). Results indicated that for dyads of children with ASD, greater emotional variability (structure) and more frequent initiation of mutual positive engagements (but not negative engagements) were associated with fewer maladaptive behaviors (i.e., internalizing and externalizing behaviors) for children with ASD. These findings suggest that both flexible interactions and shared positive interactions may play an important role in preventing behavioral problems in children with ASD.

New research by Guo, Goldberg, Valentovich, and Garfin includes fathers and their children with and without ASD in research designed to parallel the mother-child studies. Semi-structured play activities were videotaped and later coded for father and child emotion engagement states using the coding schemes described above. Fathers also completed the Vineland Adaptive Behavior Scales (Sparrow et al. 2005) to assess their children's adaptive and maladaptive behaviors. This is the first study to examine the association between emotion coregulation in father-child dyads of children with ASD and children's

adaptive functioning utilizing the SSG method (Guo et al. 2017b). In general, we found that more time spent in positive engagements (content) and less time spent in mutual negative engagement (content) were associated with higher levels of adaptive behaviors in children with ASD. These results point to the importance of father-child positive interactions in social and emotional development in children with ASD.

Future Directions

Studies conducted to date on aspects of emotion coregulation between parents and their children with ASD demonstrate its importance for children's adaptive functioning; however, this area of inquiry is underresearched. Several key areas should be considered in future studies.

Emotion Coregulation Across Contexts

In our research, parents and their children were observed during a low-stress play task. The type of play (e.g., toy play, physically orientated play) that parents and children engage in as well as different emotion-eliciting contexts (e.g., low stress vs. high stress) may impact the process of emotion coregulation (Hirschler-Guttenberg et al. 2015b; Jahromi et al. 2012; Warreyn et al. 2005). For example, in past research, mothers' emotional availability toward their children with ASD varied across free play, structured play, and social play contexts (Dolev et al. 2009). In a mildly frustrating context, children with ASD used a greater range of emotion regulation strategies but were less effective compared to NT children (Konstantareas and Stewart 2006). Hirschler-Guttenberg and colleagues (2015b) showed that children with ASD displayed more negative emotions and behaviors with fathers in a fearful context, but not in a joyful context compared to NT peers. Although children with ASD are more vulnerable to negative emotional states, more studies are needed to clarify how both parents and children respond to different social contexts (Mazefsky et al. 2012). Furthermore, additional research should examine how emotion coregulation processes will unfold in terms of both

emotional structure and content when parent-child behaviors are observed in contexts other than play, such as during high-stress tasks.

Longitudinal Design

Much of the research on emotion coregulation to date has been cross-sectional. Age effects of emotion regulation on developmental outcomes would be removed in a longitudinal design. Future work with a longitudinal study design would help clarify relationships between emotion coregulation and developmental outcomes in children with ASD. In this way, a mechanism of emotion coregulation for psychiatric comorbidity and social competencies in children with ASD would be better understood (Mazefsky et al. 2013). Specifically, future research could expand emotion coregulation to include not only micro-real-time measurement but also context-to-context emotional variability and developmental changes as described in Hollenstein's Flex3 model (Hollenstein 2015).

Dyadic Repair

Disruptions that affect positive parent-child interactions occur routinely but can be managed by a process of dyadic repair (Beeghly and Tronick 2011). The ability of parents and children to repair ruptures, or to recover from temporarily mismatched or negative interactions by returning to positive states, could be crucial for children's adaptive functioning (Beeghly and Tronick 2011; DiCorcia and Tronick 2011). For example, NT preschool-aged children who engaged in higher rates of repair processes with their mothers during a challenging puzzle task also demonstrated the ability to use appropriate regulation strategies and had fewer externalizing behaviors 4 months later (Kemp et al. 2016). Ruptures in positive interactions and attempts to repair such interactions may provide an opportunity for children to internalize efficient strategies for managing challenges and learn that they do not need to remain in negative situations (Tronick and Beeghly 2011). Future work should examine repair processes in dyads of children with ASD across low- and high-stress contexts (e.g., free play in the home environment

vs. challenging laboratory task) as well as explore how dyadic repair processes are related to children's adaptive functioning.

Clinical Implications

Past research has emphasized the negative aspects of emotion regulation among children with ASD. Our studies expand prior findings by demonstrating that dyads of parents and their children with ASD can initiate positive interactions, but they have a diminished capacity for sustaining positive interactions relative to NT dyads in a low-stress, home environment. These results could guide future family-systems-based interventions that focus on improving the ability of parent-child dyads to maintain longer positive interactions, which may be particularly important for families raising children with ASD. In particular, children may benefit from interventions that focus on helping parents assist their children in engaging in dyadic positive interactions to reduce children's behavioral problems and improve adaptive functioning. Recent studies have generated evidence that a variety of techniques could plausibly be used to extend positive interactions in dyads with children with ASD; these include mentalization-based interventions (Slade 2005), relational savoring interventions (Burkhart et al. 2015), and mindfulness-based interventions (Cachia et al. 2016). From our research, we recommend that future studies incorporate micro-analytic indicators of different aspects of emotion coregulation when evaluating the effects of interventions.

See Also

- [Emotion Regulation](#)
- [Emotion Regulation Strategies in Preschoolers with Autism](#)
- [Internalization of Emotion Co-regulatory Support in Children with ASD](#)
- [Maladaptive Behavior](#)
- [Mutual Regulation](#)
- [Social Behaviors and Social Impairment](#)
- [Vineland Adaptive Behavior Scales \(VABS\)](#)

References and Reading

- Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review*, 30, 217–237. <https://doi.org/10.1016/j.cpr.2009.11.004>.
- American Psychiatric Association. (2015). What is autism spectrum disorder? Retrieved from <http://www.psychiatry.org/patients-families/autism/what-is-autism-spectrum-disorder>
- Beeghly, M., & Tronick, E. (2011). Early resilience in the context of parent–infant relationships: A social developmental perspective. *Current Problems in Pediatric and Adolescent Health Care*, 41, 197–201.
- Berkovits, L., Eisenhower, A., & Blacher, J. (2017). Emotion regulation in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 47, 68–79. <https://doi.org/10.1007/s10803-016-2922-2>.
- Burkhart, M. L., Borelli, J. L., Rasmussen, H. F., & Sbarra, D. A. (2015). Cherish the good times: Relational savoring in parents of infants and toddlers. *Personal Relationships*, 22, 692–711. <https://doi.org/10.1111/per.12104>.
- Butler, E. A., & Randall, A. K. (2013). Emotional coregulation in close relationships. *Emotion Review*, 5, 202–210. <https://doi.org/10.1177/1754073912451630>.
- Cabrera, N. J., Shannon, J. D., & Tamis-LeMonda, C. (2007). Fathers' influence on their children's cognitive and emotional development: From toddlers to pre-K. *Applied Development Science*, 11, 208–213. <https://doi.org/10.1080/10888690701762100>.
- Cachia, R. L., Anderson, A., & Moore, D. W. (2016). Mindfulness, stress and well-being in parents of children with autism spectrum disorder: A systematic review. *Journal of Child and Family Studies*, 25, 1–14. <https://doi.org/10.1007/s10826-015-0193-8>.
- Cai, R. Y., Richdale, A. L., Uljarević, M., Dissanayake, C., & Samson, A. C. (2018). Emotion regulation in autism spectrum disorder: Where we are and where we need to go. *Autism Research*, 11, 962–978. <https://doi.org/10.1002/aur.1968>.
- Calkins, S., & Hill, A. (2007). Caregiver influences on emerging emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 229–248). New York: The Guilford Press.
- Chandler, S., Howlin, P., Simonoff, E., O'Sullivan, T., Tseng, E., Kennedy, J., . . . Baird, G. (2016). Emotional and behavioural problems in young children with autism spectrum disorder. *Developmental Medicine & Child Neurology*, 58, 202–208. <https://doi.org/10.1111/dmcn.12830>.
- Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development*, 75, 317–333. <https://doi.org/10.1111/j.1467-8624.2004.00673.x>.
- Cole, P. M., Teti, L. O., & Zahn-Waxler, C. (2003). Mutual emotion regulation and the stability of conduct problems between preschool and early school age. *Development and Psychopathology*, 15, 1–18. <https://doi.org/10.1017/S0954579403000014>.
- Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., & Liaw, J. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology*, 40, 271. <https://doi.org/10.1037/0012-1649.40.2.271>.
- Denham, S. A., Blair, K. A., DeMulder, E., Levitas, J., Sawyer, K., Auerbach-Major, S., & Queenan, P. (2003). Preschool emotional competence: Pathway to social competence? *Child Development*, 74, 238–256. <https://doi.org/10.1111/1467-8624.00533>.
- DiCorcia, J. A., & Tronick, E. D. (2011). Quotidian resilience: Exploring mechanisms that drive resilience from a perspective of everyday stress and coping. *Neuroscience and Biobehavioral Reviews*, 35(7), 1593–1602. <https://doi.org/10.1016/j.neubiorev.2011.04.008>.
- Dolev, S., Oppenheim, D., Koren-Karie, N., & Yirmiya, N. (2009). Emotional availability in mother-child interaction: The case of children with autism spectrum disorders. *Parenting: Science and Practice*, 9, 183–197. <https://doi.org/10.1080/15295190902844332>.
- Doussard-Roosevelt, J. A., Joe, C. M., Bazhaenova, O. V., & Porges, S. W. (2003). Mother-child interaction in autistic and nonautistic children: Characteristics of maternal approach behaviors and child social responses. *Development and Psychopathology*, 15, 277–295. <https://doi.org/10.1017/S0954579403000154>.
- Eisenberg, N., Fabes, R. A., Bernzweig, J., Karbon, M., Poulin, R., & Hanish, L. (1993). The relations of emotionality and regulation to preschoolers' social skills and sociometric status. *Child Development*, 64, 1418–1438. <https://doi.org/10.1111/j.1467-8624.1993.tb02961.x>.
- Feldman, R. (2003). Infant–mother and infant–father synchrony: The coregulation of positive arousal. *Infant Mental Health Journal*, 24, 1–23. <https://doi.org/10.1002/imhj.10041>.
- Granic, I., O'Hara, A., Pepler, D., & Lewis, M. (2007). A dynamic systems analysis of parent-child changes associated with successful “real-world” interventions for aggressive children. *Journal of Abnormal Child Psychology*, 35, 845–857. <https://doi.org/10.1007/s10802-007-9133-4>.
- Gulsrud, A. C., Jahromi, L. B., & Kasari, C. (2010). The co-regulation of emotions between mothers and their children with autism. *Journal of Autism and Developmental Disorders*, 40, 227–237. <https://doi.org/10.1007/s10803-009-0861-x>.
- Guo, Y., Garfin, D. R., Ly, A., & Goldberg, W. A. (2017a). Emotion coregulation in mother-child dyads: A dynamic systems analysis of children with and without autism spectrum disorder. *Journal of Abnormal Child Psychology*, 45, 1369–1383. <https://doi.org/10.1007/s10802-016-0234-9>.

- Guo, Y., Garfin, D. R., Ly, A., & Goldberg, W. A. (2017b). Comparing mother-child and father-child emotion co-regulation processes in relation to adaptive functioning in children with ASD. *Proceedings of the annual meeting of the international society for autism research*.
- Hirschler-Guttenberg, Y., Feldman, R., Ostfeld-Etzion, S., Laor, N., & Golan, O. (2015a). Self-and co-regulation of anger and fear in preschoolers with autism spectrum disorders: The role of maternal parenting style and temperament. *Journal of Autism and Developmental Disorders*, 45, 3004–3014. <https://doi.org/10.1007/s10803-015-2464-z>.
- Hirschler-Guttenberg, Y., Golan, O., Ostfeld-Etzion, S., & Feldman, R. (2015b). Mothering, fathering, and the regulation of negative and positive emotions in high-functioning preschoolers with autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 56, 530–539. <https://doi.org/10.1111/jcpp.12311>.
- Hollenstein, T. (2007). State space grids: Analyzing dynamics across development. *International Journal of Behavioral Development*, 31, 384–396. <https://doi.org/10.1177/0165025407077765>.
- Hollenstein, T. (2015). This time, it's real: Affective flexibility, time scales, feedback loops, and the regulation of emotion. *Emotion Review*, 7, 355–361. <https://doi.org/10.1177/1754073915590621>.
- Hollenstein, T., Granic, I., Stoolmiller, M., & Snyder, J. (2004). Rigidity in parent-child interactions and the development of externalizing and internalizing behavior in early childhood. *Journal of Abnormal Child Psychology*, 32, 595–607. <https://doi.org/10.1023/B:JACP.0000047209.37650.41>.
- Hollenstein, T., & Lewis, M. D. (2006). A state space analysis of emotion and flexibility in parent-child interactions. *Emotion*, 6, 656–662. <https://doi.org/10.1037/1528-3542.6.4.656>.
- Jahromi, L. B., Meek, S. E., & Ober-Reynolds, S. (2012). Emotion regulation in the context of frustration in children with high functioning autism and their typical peers. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 53, 1250–1258. <https://doi.org/10.1111/j.1469-7610.2012.02560.x>.
- Kasari, C., Sigman, M., Mundy, P., & Yirmiya, N. (1990). Affective sharing in the context of joint attention interactions of normal, autistic, and mentally retarded children. *Journal of Autism and Developmental Disorders*, 20, 87–100. <https://doi.org/10.1007/BF02206859>.
- Kemp, C. J., Lunkenheimer, E., Albrecht, E. C., & Chen, D. (2016). Can we fix this? Parent-child repair processes and preschoolers' regulatory skills. *Family Relations*, 65, 576–590. <https://doi.org/10.1111/fare.12213>.
- Konstantareas, M. M., & Stewart, K. (2006). Affect regulation and temperament in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 36, 143–154. <https://doi.org/10.1007/s10803-005-0051-4>.
- Kopp, C. B. (1982). Antecedents of self-regulation: A developmental perspective. *Developmental Psychology*, 18, 199–214. <https://doi.org/10.1037/0012-1649.18.2.199>.
- Kopp, C. B. (1989). Regulation of distress and negative emotions: A developmental view. *Developmental Psychology*, 25, 343–354. <https://doi.org/10.1037/0012-1649.25.3.343>.
- Lamey, A., Hollenstein, T., Lewis, M. D., & Granic, I. (2004). GridWare (version 1.1) [Computer software]. Retrieved from <http://www.statespacegrids.org>
- Lewis, M. D., Lamey, A. V., & Douglas, L. (1999). A new dynamic systems method for the analysis of early socioemotional development. *Developmental Science*, 2, 457–475. <https://doi.org/10.1111/1467-7687.00090>.
- Lindsey, E. W., Caldera, Y. M., & Rivera, M. (2013). Mother-child and father-child emotional expressiveness in Mexican-American families and toddlers' peer interactions. *Early Child Development and Care*, 183, 378–393. <https://doi.org/10.1080/03004430.2012.711589>.
- Lunkenheimer, E. S., Olson, S. L., Hollenstein, T., Sameroff, A. J., & Winter, C. (2011). Dyadic flexibility and positive affect in parent-child coregulation and the development of child behavior problems. *Development and Psychopathology*, 23, 577–591. <https://doi.org/10.1017/S095457941100006X>.
- Mangold, P. (2007). *INTERACT (version 9.07) [Computer software]*. Arnstorf: Mangold International GmbH.
- Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52, 679–688. <https://doi.org/10.1016/j.jaac.2013.05.006>.
- Mazefsky, C. A., Pelphrey, K. A., & Dahl, R. E. (2012). The need for a broader approach to emotion regulation research in autism. *Child Development Perspectives*, 6, 92–97. <https://doi.org/10.1111/j.1750-8606.2011.00229.x>.
- Mazefsky, C. A., & White, S. W. (2014). Emotion regulation: Concepts & practice in autism spectrum disorder. *Child and Adolescent Psychiatric Clinics of North America*, 23, 15–24. <https://doi.org/10.1016/j.chc.2013.07.002>.
- Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Social Development*, 16, 361–388. <https://doi.org/10.1111/j.1467-9507.2007.00389.x>.
- Ooi, Y. P., Tan, Z. J., Lim, C. X., Goh, T. J., & Sung, M. (2011). Prevalence of behavioural and emotional problems in children with high-functioning autism spectrum disorders. *The Australian and New Zealand Journal of Psychiatry*, 45, 370–375. <https://doi.org/10.3109/00048674.2010.534071>.
- Rieffe, C., Oosterveld, P., Terwogt, M. M., Mootz, S., Van Leeuwen, E., & Stockmann, L. (2011). Emotion regulation and internalizing symptoms in children with

- autism spectrum disorders. *Autism: The International Journal of Research and Practice*, 15, 655–670. <https://doi.org/10.1177/1362361310366571>.
- Rump, K. M., Giovannelli, J. L., Minshew, N. J., & Strauss, M. S. (2009). The development of emotion recognition in individuals with autism. *Child Development*, 80, 1434–1447. <https://doi.org/10.1111/j.1467-8624.2009.01343.x>.
- Samson, A. C., Wells, W. M., Phillips, J. M., Hardan, A. Y., & Gross, J. J. (2015). Emotion regulation in autism spectrum disorder: Evidence from parent interviews and children's daily diaries. *Journal of Child Psychology and Psychiatry*, 56, 903–913. <https://doi.org/10.1111/jcpp.12370>.
- Sasson, N. J. (2006). The development of face processing in autism. *Journal of Autism and Developmental Disorders*, 36, 381–394. <https://doi.org/10.1007/s10803-006-0076-3>.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, 47, 921–929. <https://doi.org/10.1097/CHI.0b013e318179964f>.
- Slade, A. (2005). Parental reflective functioning: An introduction. *Attachment and Human Development*, 7, 269–281. <https://doi.org/10.1080/14616730500245906>.
- Southam-Gerow, M. A., & Kendall, P. C. (2002). Emotion regulation and understanding: Implications for child psychopathology and therapy. *Clinical Psychology Review*, 22, 189–222. [https://doi.org/10.1016/S0272-7358\(01\)00087-3](https://doi.org/10.1016/S0272-7358(01)00087-3).
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales: (Vineland II), survey interview form/caregiver rating form*. Livonia: Pearson Assessments.
- Tamis-LeMonda, C. S., Shannon, J. D., Cabrera, N. J., & Lamb, M. E. (2004). Fathers and mothers at play with their 2-and 3-year-olds: Contributions to language and cognitive development. *Child Development*, 75, 1806–1820. <https://doi.org/10.1111/j.1467-8624.2004.00818.x>.
- Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development*, 59, 25–52. <https://doi.org/10.1111/j.1540-5834.1994.tb01276.x>.
- Thompson, R. A., & Meyer, S. (2007). Socialization of emotion regulation in the family. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 249–268). New York: The Guilford Press.
- Ting, V., & Weiss, J. A. (2017). Emotion regulation and parent co-regulation in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47, 680–689. <https://doi.org/10.1007/s10803-016-3009-9>.
- Tronick, E. Z. (1989). Emotions and emotional communication in infants. *American Psychologist*, 44, 112–119. <https://doi.org/10.1037//0003-066x.44.2.112>.
- Tronick, E., & Beeghly, M. (2011). Infants' meaning-making and the development of mental health problems. *American Psychologist*, 66, 107–119.
- Uljarevic, M., & Hamilton, A. (2013). Recognition of emotions in autism: A formal meta-analysis. *Journal of Autism and Developmental Disorders*, 43, 1517–1526. <https://doi.org/10.1007/s10803-012-1695-5>.
- Valentovich, V., Goldberg, W. A., Garfin, D. R., & Guo, Y. (2018). Emotion coregulation processes between mothers and their children with and without autism spectrum disorder: Associations with children's maladaptive behaviors. *Journal of Autism and Developmental Disorders*, 48, 1235–1248. <https://doi.org/10.1007/s10803-017-3375-y>.
- Vandell, D. L. (1979). Effects of a playgroup experience on mother-son and father-son interaction. *Developmental Psychology*, 15, 379–385. <https://doi.org/10.1037/0012-1649.15.4.379>.
- Warreyn, P., Roeyers, H., & De Groote, I. (2005). Early social communicative behaviours of preschoolers with autism spectrum disorder during interaction with their mothers. *Autism*, 9, 342–361. <https://doi.org/10.1177/1362361305056076>.
- Yirmiya, N., Kasari, C., Sigman, M., & Mundy, P. (1989). Facial expressions of affect in autistic, mentally retarded and normal children. *Journal of Child Psychology and Psychiatry*, 30, 725–735. <https://doi.org/10.1111/j.1469-7610.1989.tb00785.x>.

Emotion Dysregulation Inventory

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Synonyms

EDI

Description

The Emotion Dysregulation Inventory (EDI) is an informant questionnaire that assesses poor emotion regulation and is validated for general community and clinical populations as well as youth with autism spectrum disorder (ASD). It was created using methods developed by the Patient-Reported Outcomes Measurement Information

System (PROMIS®). The EDI items were developed based on an iterative process that included caregiver input. The EDI response options ask respondents to rate each item on a 5-point Likert scale from “not at all” to “very severe” for observed functioning over the past 7 days. Caregivers of 1755 youth with ASD completed 66 candidate EDI items, and the final 30 items were selected based on classical test theory and item response theory (IRT) analyses. The analyses identified two factors: (1) Reactivity, characterized by intense, rapidly escalating, sustained, and poorly modulated negative emotional reactions, and (2) Dysphoria, characterized by anhedonia, sadness, and nervousness. The final items did not show differential item functioning (DIF) based on gender, age, intellectual ability, or verbal ability. Because the final items were calibrated using IRT, even a small number of items offers high precision, minimizing respondent burden. IRT co-calibration of the EDI with other commonly used measures demonstrated its superiority in assessing the severity of emotion dysregulation with as few as seven items. Validity of the EDI was supported by expert review, its association with related constructs (e.g., anxiety and depression symptoms, aggression), and higher scores in psychiatric inpatients with ASD compared to a community ASD sample. Analyses in a general sample of 1000 youth matched to the US Census on age, gender, race/ethnicity, years of education, and region supported the EDI’s original factor structure and psychometrics, including its sensitivity, reliability, and validity. The general youth sample data was also used to generate normative cutoffs to determine clinical significance. In sum, the EDI provides an efficient and sensitive method to measure emotion dysregulation for clinical assessment, monitoring, and research in individuals of any level of cognitive or verbal ability across populations.

The EDI is available for use (requests can be made at: www.reaact.pitt.edu by completing the “EDI Inquiry Form”). The EDI is available in English, Spanish, French, Dutch, German, Danish, traditional Chinese, and Turkish. Requests for other translations can be made through the inquiry form. Theta score and t-score conversions are

available based on either the autism norms or general population norms. Examples of Reactivity items include: “Has explosive outbursts,” “Has trouble calming him/herself down,” “Emotions go from 0 to 100 instantly,” and “Cries or stays angry for 5 minutes or longer.” Examples of Dysphoria items include: “Very little makes him/her happy,” “Seems sad or unhappy,” “Not responsive to praise or good things happening,” and “Refuses to leave the house or go to school unless forced.”

Historical Background

Emotion dysregulation, or difficulty modulating emotion in the service of one’s goals, is common in individuals with ASD and has significant implications for treatment needs and outcome (Mazefsky 2015; Weiss et al. 2017). However, the lack of sensitive measures suitable for use across the range of functioning in autism spectrum disorder (ASD) is a barrier to treatment development and monitoring. Most emotion regulation measures were developed for the general population and excluded youth with ASD when assessing their psychometric properties. In addition, emotion regulation measures generally rely on self-report or use wording such as “complains about” or “worries about,” which limits their utility for youth with minimal verbal ability. Having a dimensional measure of emotion dysregulation that could be applied across the autism spectrum would enable a more complete understanding of the role of emotion dysregulation in ASD and facilitate treatment planning and monitoring.

The EDI was developed to meet this need. As such, it was designed to capture the full range of manifestations of poor emotion regulation in ASD. Another primary goal was for it to be applicable across the autism spectrum, with items that could be rated regardless of the individual’s verbal ability. Therefore, the emphasis is on observable signs of poor emotion regulation, and the format is informant questionnaire. Informant report is an ideal form of assessment in situations when the individual is unable to report due to cognitive or other limitations, making it an appropriate method for assessing minimally verbal youth with ASD.

In addition, informant perspectives on emotion dysregulation are important because limited emotional insight and awareness is common in ASD (Griffin et al. 2016).

The EDI was created using guidelines from an NIH Roadmap Initiative focused on developing sensitive outcome measures, PROMIS® (www.nihpromis.org). The multiple-site PROMIS Research Network (2012) produced scientific standards for developing measures as well as a comprehensive battery of effective and sensitive outcome measures. The EDI was the first measure for ASD developed based on PROMIS principles.

The EDI item development process was described in detail in Mazefsky et al. (2018a). First, a comprehensive review of emotion regulation and existing measures of related constructs was completed to inform development of a conceptual model. The conceptual model was structured with a primary domain (emotion dysregulation), subdomains (affective experience and affective control), factors (negative mood, anxiety, reactivity, and emotional inflexibility), and facets (increased negative affect, decreased positive affect, disturbed behavior, decreased vitality, nervousness and fear, hyperarousal, rapid escalation, intensity, lability, sustained reactions, and poor modulation of emotion). The actual dimensional structure was tested empirically at a later step, but the conceptual model structure was utilized to develop an item hierarchy, which involved assignment of draft items to each facet to ensure adequate coverage of key constructs. The draft of items was reviewed by experts in ASD and measure development.

Once the initial item pool was developed, interviews were completed with 19 parents of children and adults with ASD who varied in verbal and intellectual ability and age to assess their understanding of the items and their decision-making processes when selecting their responses. Information generated from these interviews, along with input from a panel of experts in measure development and emotion dysregulation in ASD, was utilized to revise the items, directions, and response options and arrive at the final 66 candidate items that were used for psychometric analyses and calibration.

The complete EDI item bank, directions, and response options had a Flesch Reading Ease of 66.3 (scale of 0–100 with higher scores indicating easier to read) and a Flesch-Kincaid Grade Level of 7.6, which are both considered adequate. Without the instructions, which include some longer passages with guidance on how to interpret behavior, the items and response options had a Flesch Reading Ease of 100 and a Flesch-Kincaid Grade Level of 2.1.

Psychometric Data

The complete psychometrics of the EDI in an ASD sample are described in Mazefsky, Yu, White, Siegel, and Palkonis (2018b). The EDI psychometrics were established utilizing a sample that was representative of the full spectrum of severity of ASD (Interactive Autism Network; IAN) while also being enriched with the most extreme forms of emotion dysregulation in ASD (Autism Inpatient Collection; AIC). IAN uses the Social Communication Questionnaire Lifetime for confirmation of parental report of professional diagnosis of ASD. Parental report of professional diagnosis of ASD has been verified by medical records. A prior study documented the validity of the ASD diagnoses in IAN through standardized clinical assessments and independent clinician verification (see Lee et al. 2010 for full details). The AIC is a six-site study of children, adolescents, and young adults admitted to specialized inpatient psychiatric units for youth with ASD and other developmental disorders. The full methods of the AIC have been published previously (Siegel et al. 2015). Participants in the AIC had ASD confirmed by the Autism Diagnostic Observation Schedule-2 and expert opinion.

A total of 1755 caregivers of children with ASD from IAN ($n = 1323$) and AIC ($n = 432$) combined completed the EDI for psychometric analysis. The mean age in both samples was 12 with a standard deviation of 3, though the range for IAN's sample was 6–17.9, whereas the range for the AIC was 4–20. In the combined sample, 27.8% had intellectual disability, 55.3% were fluently verbal, and 20.9% were female. The

AIC had a higher proportion of youth with intellectual disability and limited verbal ability, and the gender composition was equal in both sources. The proportion of the combined sample with co-occurring intellectual disability and percent female was consistent with national autism surveillance statistics (Baio et al. 2018).

First, the dimensionality of the EDI item bank was explored. In order to do so, the sample was split in half for exploratory factor analysis (EFA) and confirmatory factor analysis (CFA). Given differences in IQ and verbal ability for the IAN and AIC samples, EFA was performed separately, as well as for the combined sample. The eigenvalues and factor structures were similar across the three samples; therefore, the combined sample was used for all additional analysis. The 1- and 2-factor solutions emerged as the most meaningful, given the scree plots, the magnitude of eigenvalues, and clinical interpretation. In the combined sample, the eigenvalues were 29.48 and 4.71 for the first two factors. Factor 1 was characterized by items capturing rapidly escalating, intense, and labile negative affect as well as difficulty downregulating that affect (sustained reactions and trouble calming down). Factor 2 included items that reflect common definitions of general negative affect (sadness, unease, and anxiety) as well as low motivation or anhedonia. The second round of EFA on the combined sample retained 53 items with the same general content, there were no items that loaded >0.45 loading on both factors, and the correlation between the two factors was 0.51.

Next, single-factor CFAs were completed on the reduced item pool, using the second half of the sample ($n = 885$). For F1, all factor loadings were greater than 0.50, and several fit indices were uniformly strong: RMSEA = 0.09, CFI = 0.96, TLI = 0.96. For F2, all factor loadings were larger than 0.45, and fit indices revealed less homogeneity in CFA terms (reflected primarily in a larger RMSEA: RMSEA = 0.12, CFI = 0.86, TLI = 0.84).

Based on the EFA and CFA analyses, the original 66 items were trimmed to a pool of 53 items distributed across two factors (Reactivity and Dysphoria). Next, the two corresponding item banks were calibrated separately using two-

parameter graded item response theory response model (GRM) for polytomous items (i.e., items with three or more ordinal response categories). The items were individually evaluated based on their slope parameter which measures item discrimination (i.e., how well the item differentiates higher versus lower levels of severity or Θ in IRT terms). Useful items have larger slope parameters. In addition, threshold parameters which measure item difficulty (i.e., the ease versus difficulty of endorsing different response options for an item) were explored. Local dependency (i.e., residual correlations) in the IRT models was also assessed using the LD chi-square. Finally, item analyses included investigation of differential item functioning (DIF) by gender, age, verbal ability, and IQ flagged no items by both DIF methods, and no further items were eliminated for this reason. Item information curves were also examined to eliminate items with limited information, i.e., less than 0.50.

Thus, the final calibrated item bank after completing all of these steps included 30 items: 24 items for factor 1 (Reactivity) and 6 items for factor 2 (Dysphoria). Overall, Reactivity was more robust than Dysphoria, with more items, better IRT parameters, and smaller standard errors. For example, 20 items had item information values over 2.0 for Reactivity, with the following items all having total item information values over 3.0: has explosive outbursts, hard to calm him/her down when mad or upset, has extreme or intense emotional reactions, has trouble calming him/herself down, has emotions go from 0 to 100 instantly, and cries or stays angry for 5 min or longer. For Dysphoria, only three items had item information values over 2.0, and only “very little makes him/her happy” had an item information value over 3.0. The correlation between the two EDI factors was 0.63 in the combined sample, 0.50 for AIC, and 0.59 for IAN, all significant at $p < 0.001$.

To be able to provide a static short form for Reactivity, items were rank ordered on four criteria: discrimination parameters, the percentage of times the item would have been selected in a simulated CAT using the calibration sample, expected information under the standard normal

distribution with a mean of 0 and SD of 1, and expected information under a normal distribution with a larger SD, i.e., a mean of 0 and SD of 1.5. Seven items were selected for the short form based on the convergence of the four psychometric criteria, the content of candidate items, and location parameters. The theta correlation between the short form and the full bank was 0.98.

Next, concurrent calibration, which refers to estimating item parameters across multiple measures on a single computer run, was completed to evaluate how well the EDI captures variability in emotion dysregulation in comparison to other measures. The EDI was compared to the Aberrant Behavior Checklist Irritability Scale (ABC-I; 15 items), Child Behavior Checklist (CBCL) Stress Problems Scale (14 items), and an Emotion Dysregulation Index from the CBCL (CBCL-EDI) that included 18 items that experts in emotion regulation agreed captured the construct (Samson et al. 2014). The full EDI Reactivity scale (24 items) provided more than double the test information than any other comparison measure. The EDI Reactivity Short Form, with just seven items, also provided about the same amount of information as the ABC-I and more information than the CBCL-EDI and CBCL-SPS across the same range of theta (-2 to $+2$ SDs).

Initial evidence for construct validity was based on expert review (see Mazefsky et al. 2018a). In addition, correlations with legacy measures revealed expected patterns. In particular, both EDI factors (Reactivity and Dysphoria) were positively and moderately to strongly correlated with measures of similar constructs [(CBCL Stress Problems; $r(1754) = 0.562, p < 0.001$) and Samson CBCL-Emotion Dysregulation Index; $r(1754) = 0.748, p < 0.001$]. In addition, they were correlated as expected with scores for other behavioral and emotional problems, based on prior literature demonstrating an association between emotion regulation and these constructs. Finally, the AIC group had significantly higher EDI Reactivity (mean = 0.91, SD = 0.80) and EDI Dysphoria theta scores (mean = 0.55, SD = 0.83) than the IAN sample (EDI Reactivity: mean = -0.30 , SD = 0.85, $t = 25.8, df = 1753, p < 0.001$; EDI Dysphoria: mean = -0.18 ,

SD = 0.87, $t = 15.4, df = 1753, p < 0.001$), as well as a higher proportion of the sample with items rated as moderate or higher. These findings suggest that EDI scores differ in expected ways between known groups, in support of criterion validity.

The EDI's psychometrics were also established in a general sample of youth in order to enable its use across populations and to generate clinical cutoffs (Mazefsky et al. 2020). The sample included parents of 1000 6- to 17-year-old youth, matched to the US Census on gender, age, race/ethnicity, years of education, and region. The sample was recruited through YouGov, a global public opinion polling company. Confirmatory factor analyses were conducted to determine whether the two-factor structure of the EDI derived in the autism sample still held. Because the highest response options (severe and very severe) were endorsed less frequently in the YouGov sample, IRT was also completed with these response options combined to determine if that would yield any benefit, in addition to completing IRT analyses with the original five response options. The same methods utilized to generate the EDI Reactivity Short Form were completed again, to evaluate whether the same or different items should be used in the short form for more general use outside of ASD. IRT co-calibration was completed to compare the EDI to existing measures of related constructs, as described previously in the autism sample. Finally, validity was assessed by examining the EDI's correlations with measures of related constructs, comparing means between groups that would be expected to differ, and receiver operating characteristics which provide an index of sensitivity to identify different groups.

The results in the general US sample provided strong support for the EDI and were consistent with conclusions from the prior work in the autism clinical sample. The factor structure with separate Reactivity and Dysphoria scores was upheld, with strong evidence for unidimensionality (factor loadings from 0.72 to 0.91 and strong fit statistics). Reactivity and Dysphoria were correlated 0.71, $p < 0.001$. IRT results with four versus five response options were roughly equivalent, so the five response option

version was retained to maintain consistency with the earlier autism work. The IRT parameters were strong and comparable to the original IRT parameters in the autism sample (slope parameters ranging from 2.13 to 3.62 for Reactivity and from 1.81 to 3.64 for Dysphoria). Seven items had slope parameters above 3.0 for Reactivity and three had slope parameters above 3.0 for Dysphoria. The reliable range of measurement was -0.75 to $+4$ SDs for Reactivity and 0 to $+3.75$ SDs for Dysphoria. The computerized adaptive testing results to identify the best short form items resulted in six of the same seven original items; so the full short form was retained as originally developed for consistency. Even with fewer items, both EDI scales provided more information, across a wider range of severity, than all existing measures of related constructs that were compared via IRT co-calibration. Both Reactivity and Dysphoria were correlated as expected with existing measures, in support of concurrent validity. Finally, EDI scores were significantly higher for both Reactivity and Dysphoria scales for those who had been psychiatrically hospitalized, who had a crisis evaluation within the past 2 months, who were taking psychotropic medications or in therapy, and who had current psychiatric diagnoses, all $p < 0.001$. These findings were consistent with the results of the receiver operating characteristics.

Clinical Uses

The EDI has strong evidence to support its use in ASD, as well as general community and clinical samples. Overall, it has enhanced precision compared to previously existing measures, and it is brief and efficient. It may be useful for screening in initial clinical assessments as well as progress and outcome monitoring. The EDI provides norms both based on an autism sample and based on a general sample of youth, with clinical cutoff guidelines based on the general population norms. The autism norms may be most useful in situations when clients or samples include more severe forms of emotion dysregulation. The most psychometric evidence is for youth aged 6–17.9.

Several projects are underway to develop a self-report version for adolescents and adults, norms for informant report for adults, and an informant report for 2- to 5-year-old children.

See Also

- [Emotion Awareness and Skills Enhancement \(EASE\) Program](#)
- [Emotion Regulation](#)
- [Emotion Regulation Strategies in Preschoolers with Autism](#)

References and Reading

- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., . . . Dowling, N. F. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summary*, 67, 1–23.
- Griffin, C., Lombardo, M. V., & Auyeung, B. (2016). Alexithymia in children with and without autism spectrum disorders. *Autism Research*, 9(7), 773–780.
- Lee, H., Marvin, A. R., Watson, T., Piggot, J., Law, J. K., Law, P. A., Constantino, J. N., & Nelson, S. F. (2010). Accuracy of phenotyping of autistic children based on internet implemented parent report. *American Journal of Medical Genetics, Part B*, 153B, 1119–1126.
- Mazefsky, C. A. (2015). Emotion regulation and emotional distress in autism spectrum disorder: Foundations and considerations for future research. *Journal of Autism and Developmental Disorders*, 45, 3405–3408.
- Mazefsky, C. A., Day, T. N., Siegel, M., White, S. W., Yu, L., & Pilkonis, P. A. (2018a). Development of the emotion dysregulation inventory: A PROMISing method for creating sensitive and unbiased questionnaires for autism Spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(11), 3736–3746. <https://doi.org/10.1007/s10803-016-2907-1>.
- Mazefsky, C. A., Yu, L., White, S. W., Siegel, M., & Pilkonis, P. A. (2018b). The emotion dysregulation inventory: Psychometric properties and item response theory calibration in an autism spectrum disorder sample. *Autism Research*, 11(6), 928–941. <https://doi.org/10.1080/15374416.2019.1703710>.
- Mazefsky, C. A., Yu, L., & Pilkonis, P. A. (2020). Psychometric properties of the emotion dysregulation inventory in a nationally representative sample of youth. *Journal of Clinical Child & Adolescent Psychology*. <https://doi.org/10.1080/15374416.2019.1703710>. Online in Advance of Print.
- PROMIS® Validity Standards Committee on behalf of the PROMIS® Network. (2012). *PROMIS instrument development and psychometric evaluation scientific*

- standards (pp. 1–72). Retrieved from <http://www.nihpromis.org/science/methodology?AspxAutoDetectCookieSupport=1>
- Samson, A. C., Phillips, J. M., Parker, K. J., Shah, S., Gross, J. J., & Hardan, A. Y. (2014). Emotion dysregulation and the core features of autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(7), 1766–1772.
- Siegel, M., Smith, K. A., Mazefsky, C., Gabriels, R. L., Erickson, C., Kaplan, D., et al. (2015). The autism inpatient collection: Methods and preliminary sample description. *Molecular Autism*, 6, 61–71.
- Weiss, J. A., Riosa, P. B., Mazefsky, C. A., & Beaumont, R. (2017). Emotion regulation in autism spectrum disorder. In C. A. Essau, S. S. LeBlanc, & T. H. Ollendick (Eds.), *Emotion regulation and psychopathology in children and adolescents* (pp. 235–258). Oxford: Oxford University Press.

one's emotions to fit the context or meet one's goals" (Thompson 1994). We all carry a toolbox of strategies to regulate our emotions (Calkins 1994; John and Gross 2007), which can be driven by ourselves, or driven by others, and can be active or passive (Gross 1998; Gyurak and Etkin 2014). Children with autism spectrum disorder (ASD) often have difficulty with regulating their emotions, which has been found to impact their behavioral and mental health. An emerging field of research has documented the strategies that children with ASD use to regulate their emotion and how these strategies overlap and differ from their typically developing peers across ages. Of particular importance is the emergence of emotion regulation strategies early in life.

Emotion Recognition

► Social Cognitive Interventions

Emotion Regulation

► Mutual Regulation

Emotion Regulation Strategies in Preschoolers with Autism

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Definition

At the core of mental health and well-being lies the ability to manage negative emotions. Emotion regulation is “the processes related to modifying

Historical Background

Emotion regulation skills have been shown to predict school success (Graziano et al. 2007; Gumora and Arsenio 2002; Howse et al. 2003), and conversely, difficulties with emotion regulation predict problem behavior and mental health problems (Eisenberg et al. 2000, 2001; Graziano et al. 2007; Hill et al. 2006; Rubin et al. 1995; Silk et al. 2003). Children with ASD are at a higher risk for episodes of emotional dysregulation, compared to their typically developing peers (Mazefsky et al. 2013; Samson et al. 2014); one study showed 74% of children with ASD have emotion regulation difficulties compared to 18% of typically developing children and 42% of children with intellectual disability (Totsika et al. 2011).

The features of emotional dysregulation in children with ASD include emotional lability (Samson et al. 2012), negative mood and irritability (Samson et al. 2014; Zwaigenbaum et al. 2005), internalizing symptoms such as anxiety and depression (Berthoz and Hill 2005), and externalizing behaviors, such as aggression and hyperactivity (Ashburner et al. 2010; Bauminger et al. 2010). Common ASD symptoms have been found to be associated with poorer emotional regulation (Mazefsky et al. 2013), including sensory abnormalities and restricted/repetitive behaviors

(Samson et al. 2014), social/communication deficits (Pouw et al. 2013a), and executive function difficulties (Jahromi et al. 2013). Additionally, as emotion regulation skills are built upon foundational joint attention (Morales et al. 2005; Raver 1996) and language (Cohen and Mendez 2009; Gallagher 1999; Gottman 1986) skills, individuals with ASD are significantly disadvantaged in their ability to deploy emotion regulation compared to typically developing peers due to significant difficulties in these core areas of ASD. The developmental trajectory of emotion co-regulation to self-driven regulation is built on a foundation of emotional expression (to signal the need for regulation), and here too, individuals with ASD are at a disadvantage due to difficulty unambiguously expressing emotional states and in identifying and describing their own emotions (Berthoz and Hill 2005; Nuske et al. 2013; Samson et al. 2012).

Most of the research to date examining emotion regulation strategies in individuals with ASD has focused on adolescents or adults, finding increased use of maladaptive forms of emotion regulation, including rumination, shutting down, suppression (with later outbursts), blaming others, avoidance, isolation, self-harm, and use of drugs (Bruggink et al. 2016; Mazefsky et al. 2014; Patel et al. 2017; Samson et al. 2012, 2015a, b; Santomauro et al. 2017). Findings also suggest less success with adaptative strategies, such as problem-solving and cognitive reappraisal (Bruggink et al. 2016; Samson et al. 2015b), along with successful use of other strategies including special interests, relaxation, music listening, and online communities (e.g., gaming communities; Santomauro et al. 2017).

Numerous research groups have found that maladaptive or avoidant strategies in adolescents and adults with ASD are associated with higher depression, anxiety and problem behavior, and lower well-being and social functioning (Bruggink et al. 2016; Cai et al. 2019; Goldsmith and Kelley 2018; e.g. Khor et al. 2014; Patel et al. 2017; Pouw et al. 2013b; Rieffe et al. 2011; Samson et al. 2015a).

Less work has focused on the emotion regulation strategies of children with ASD, though

initial findings are consistent with the adolescent and adult data. For example, Rieffe et al. (2011) found that some strategies, such as positive coping, may be less effective for 9- to 12-year-old children with ASD compared to typically developing children. Emotion dysregulation is particularly troublesome in school settings (Ashburner et al. 2008), where problem behaviors, which have been causally linked to maladaptive emotion regulation (Samson et al. 2015a), have been found to directly predict teacher burnout (Hastings and Bham 2003; Hastings and Brown 2002). Interestingly, Konstantareas and Stewart (2006) found 3- to 10-year-old children with ASD used a greater range of strategies than typically developing children, suggesting a trial-and-error (vs. social learning) approach, which is more common in ASD than in typical development (Vivanti et al. 2016; Vivanti and Rogers 2014).

Current Knowledge

In particular, studying emotion regulation strategies during the preschooler period is of great interest as this may help to identify key intervention targets in children who generally have easier access to 1:1 intervention, prior to school entry. Emotion regulation difficulties start early in children with ASD; by 12 months, 86% of parents of children with ASD reported regulatory difficulties (Gomez and Baird 2005). By preschooler age, typically developing children use multiple strategies that may be classified as co-regulated via others/other-directed (e.g., social negotiation, hugs) vs. self-directed (e.g., self-talk, self-soothing, deep exhalation) and passive (e.g., gaze aversion, task disengagement) vs. active (e.g., leaving one task for another) (Buss and Goldsmith 1998; Grolnick et al. 1996; Kopp 1989). There is also a shift from regulating through caregivers to regulating through other adults, such as teachers or even unfamiliar adults (Zimmer-Gembeck and Skinner 2011). Therefore, the typical emotion regulation strategy trajectory for preschoolers is shaped by parallel developments in autonomy and growing awareness of one's agency.

The few studies that have focused on emotion regulation strategies in preschoolers with ASD show that findings from the older population are already in place by this age, with increased maladaptive strategies, such more avoidance, resignation, and venting or tension release (Gulsrud et al. 2010; Jahromi et al. 2012; Zantinge et al. 2017). Results by Zantinge et al. (2017) found that use of such strategies was related to language impairments in the preschoolers with ASD, suggesting that developmental delays are driving the use of less optimal strategies relative to their typically developing peers. Consistent with this interpretation, Nuske et al. (2017) found that preschoolers with ASD used strategies that are characteristic of younger typically developing children, such as reliance on their family members for physical and communicative soothing, whereas the typically developing children relied on people outside of their family for help regulating their emotion. Moreover, authors found that positive self-development (self-recognition, self-evaluation, and autonomy) was related to more adaptive emotion regulation strategies in the preschoolers with ASD (Nuske et al. 2017).

Findings by Jahromi et al. (2012) suggest the pattern of less effective employment of strategies that are adaptive in typically developing individuals is also present in preschoolers with ASD; they found social support strategies (orienting and verbalizing to the experimenter) were ineffective for children with ASD. Therefore, findings to date suggest both developmental delays in emotion regulation strategies in preschoolers with ASD, particularly for strategies facilitating the shift from co-regulation to self-regulation during early childhood (e.g., Cole et al. 1994), and a qualitative difference in the types of strategies that work for preschoolers with ASD compared to typically developing preschoolers.

Children learn how to process and manage their emotions via parental modelling and guidance (Eisenberg et al. 1998). Indeed, recent findings suggest that parent-facilitated emotion regulation, specifically strategies that encouraged children to self-regulate their emotions, in preschoolers with ASD predicts positive self-development and school engagement 2 years

later (Nelson et al. [under review](#)). Consistently, Gulsrud et al. (2010) found that higher use of maternal strategies with toddlers with ASD that aimed to manage the child's stress *for them* (prompting, physical comfort, and redirection) was related to more frequent externalizing behaviors. This could suggest the need to put a stronger emphasis on supporting *self*-regulation in ASD versus externally supported regulation. Higher use of maternal vocal strategies (vocal comfort and reassurance), on the other hand, was related to less child stress (Gulsrud et al. 2010). However, in older children with ASD, higher parental scaffolding has been found to predict less child dysregulation in a dyadic context (Fenning et al. 2018) and less externalizing (but not internalizing) problems during anger/anxiety episodes, after controlling for child age and IQ (Ting and Weiss 2017). Children's own use of certain types of strategies also may impact those around them; Nuske et al. (2018) found that preschoolers with ASD that used two passive emotion regulation strategies (self-soothing, deep exhalation) more frequently were in families that reported a lower family quality of life.

The findings reported above highlight, in particular, the importance of supporting self-regulation and independence in children with ASD. Based on the research in this area, appropriate intervention strategies to support self-development in children with ASD, via targeting self-other mapping and self-awareness, may include imitation of the child, use of child names to refer to their body movements (e.g., walking), states (e.g., tired), permanent relations of possession (e.g., clothing), and photographs of themselves, modelling of personal pronouns during self-propelled actions on objects, turn-taking, and visuospatial perspective-taking games and the creation of stories where the child is the main protagonist (Charney 1980; DeSocio 2005; Hobson 1990, 2010; Loveland 1984; McLean et al. 2007). Given that agency and internal motivation are recognized as integral to self-development (Deci and Ryan 1980; Ryan and Deci 2000), interventions that offer children choices and capitalize on their interests as teaching props and reinforcers (e.g., naturalistic

developmental behavioral interventions; Schreibman et al. 2015) will also incidentally support self-development.

Future Directions

Although there has been a marked increase in emotion regulation research in ASD, there are still several gaps and important areas for future research. First, as argued here, the preschool period is critical for emotion regulation development and requires more attention. The majority of research thus far has focused on characterizing strategy use, and treatment development efforts could be supported by the identification of mechanisms or processes that may contribute to delayed or atypical emotion regulation development.

Many studies in the area did not report information on the actual experienced emotion of the children (e.g., coding of child emotional reactivity, physiological markers of stress). An important next step for future research is to examine how emotion regulation strategy use impacts, and is impacted by, the experienced emotion in children with ASD. Particularly in the preschool age range, it will be essential to also consider how the expressed emotion may impact the bidirectional relationship between the parent or other significant adults in the child's life and the child, as it related to emotion regulation development. Beyond emotion expression, it may further be helpful to consider how other aspects of emotional development, such as emotion recognition and emotion awareness, impact emotion self-regulation.

As this field moves forward, it would also benefit from enhanced methodological rigor, especially the use of multimethod assessment approaches, preferably incorporating measures validated in ASD, and longitudinal studies. Longitudinal research may help inform which aspects of emotion regulation increase risk for later problems. Importantly, there is great variability in emotion regulation development in ASD, and it will be important to understand factors that are associated with more positive trajectories. Along these same lines, the emotion regulation

research has emphasized the regulation of negative emotion, but it is equally important to understand how preschoolers with ASD learn to regulate positive emotion. Since neuroimaging work suggests emotion regulation is central to self-development (Pfeifer and Peake 2012), future research is also needed to delineate the developmental bidirectional relationship between emotion regulation and broader self-development.

Finally, further research on the strategies to support emotional self-regulation in ASD is needed. There are some small studies suggesting that emotion regulation can be improved with intervention in older preschool-aged children ASD (e.g., Scarpa and Reyes 2011), but emotion regulation has generally not been measured of an endpoint in any clinical trials with younger preschool children. Even among older children, adolescents, and adults, the treatment literature on emotion regulation is markedly limited, with only recent developments in this area (e.g., Conner et al. 2019). Given the accumulating evidence linking poor emotion regulation to negative outcomes, greater focus on treatment development and testing to improve emotion regulation is a priority. It will be important to consider the best means to deliver such interventions during the preschool period, such as embedded within broader treatment programs, in school settings, or with parent-supported delivery to support generalization of effects.

See Also

- ▶ [Emotion Awareness and Skills Enhancement \(EASE\) Program](#)
- ▶ [Emotion Coregulation Processes Between Parents and Their Children with ASD](#)
- ▶ [Emotion Dysregulation Inventory](#)
- ▶ [Emotional Processing in ASD](#)
- ▶ [Emotional Regulation](#)
- ▶ [Internalization of Emotion Co-regulatory Support in Children with ASD](#)
- ▶ [Neural Mechanisms of Emotional Dysregulation](#)
- ▶ [Predictors of Emotion Regulation in Children with Autism Spectrum Disorder \(ASD\)](#)

References and Reading

- Ashburner, J., Ziviani, J., & Rodger, S. (2008). Sensory processing and classroom emotional, behavioral, and educational outcomes in children with autism spectrum disorder. *American Journal of Occupational Therapy*, 62(5), 564–573.
- Ashburner, J., Ziviani, J., & Rodger, S. (2010). Surviving in the mainstream: Capacity of children with autism spectrum disorders to perform academically and regulate their emotions and behavior at school. *Research in Autism Spectrum Disorders*, 4(1), 18–27. <https://doi.org/10.1016/j.rasd.2009.07.002>.
- Bauminger, N., Solomon, M., & Rogers, S. J. (2010). Externalizing and internalizing behaviors in ASD. *Autism Research*, 3(3), 101–112.
- Berthoz, S., & Hill, E. L. (2005). The validity of using self-reports to assess emotion regulation abilities in adults with autism spectrum disorder. *European Psychiatry*, 20(3), 291–298. <https://doi.org/10.1016/j.eurpsy.2004.06.013>.
- Bruggink, A., Huisman, S., Vuijk, R., Kraaij, V., & Garnefski, N. (2016). Cognitive emotion regulation, anxiety and depression in adults with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 22, 34–44.
- Buss, K. A., & Goldsmith, H. H. (1998). Fear and anger regulation in infancy: Effects on the temporal dynamics of affective expression. *Child Development*, 69(2), 359–374.
- Cai, R. Y., Richdale, A. L., Dissanayake, C., Trollor, J., & Uljarević, M. (2019). Emotion regulation in autism: Reappraisal and suppression interactions. *Autism*, 23(3), 737–749.
- Calkins, S. D. (1994). Origins and outcomes of individual differences in emotion regulation. *Monographs of the Society for Research in Child Development*, 59(2–3), 53–72. <https://doi.org/10.1111/j.1540-5834.1994.tb01277.x>.
- Charney, R. (1980). Speech roles and the development of personal pronouns. *Journal of Child Language*, 7(03), 509–528.
- Cohen, J. S., & Mendez, J. L. (2009). Emotion regulation, language ability, and the stability of preschool children's peer play behavior. *Early Education and Development*, 20(6), 1016–1037.
- Cole, P. M., Michel, M. K., & Teti, L. O. (1994). The development of emotion regulation and dysregulation: A clinical perspective. *Monographs of the Society for Research in Child Development*, 59(2–3), 73–102.
- Conner, C. M., White, S. W., Beck, K. B., Golt, J., Smith, I. C., & Mazefsky, C. A. (2019). Improving emotion regulation ability in autism: The Emotional Awareness and Skills Enhancement (EASE) program. *Autism*, 23(5), 1273–1287. <https://doi.org/10.1177/1362361318810709>.
- Deci, E. L., & Ryan, R. M. (1980). The empirical exploration of intrinsic motivational processes. *Advances in Experimental Social Psychology*, 13, 39–80.
- DeSocio, J. E. (2005). Accessing self-development through narrative approaches in child and adolescent psychotherapy. *Journal of Child and Adolescent Psychiatric Nursing: Official Publication of the Association of Child and Adolescent Psychiatric Nurses, Inc.*, 18(2), 53–61. <https://doi.org/10.1111/j.1744-6171.2005.00012.x>.
- Eisenberg, N., Cumberland, A., & Spinrad, T. L. (1998). Parental socialization of emotion. *Psychological Inquiry*, 9(4), 241–273.
- Eisenberg, N., Guthrie, I. K., Fabes, R. A., Shepard, S., Losoya, S., Murphy, B. C., ... Reiser, M. (2000). Prediction of elementary school children's externalizing problem behaviors from attentional and behavioral regulation and negative emotionality. *Child Development*, 71, 1367–1382.
- Eisenberg, N., Cumberland, A., Spinrad, T. L., Fabes, R. A., Shepard, S. A., Reiser, M., ... Guthrie, I. K. (2001). The relations of regulation and emotionality to children's externalizing and internalizing problem behavior. *Child Development*, 72(4), 1112–1134.
- Fenning, R. M., Baker, J. K., & Moffitt, J. (2018). Intrinsic and extrinsic predictors of emotion regulation in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(11), 3858–3870.
- Gallagher, T. M. (1999). Interrelationships among children's language, behavior, and emotional problems. *Topics in Language Disorders*, 19(2), 1–15.
- Goldsmith, S. F., & Kelley, E. (2018). Associations between emotion regulation and social impairment in children and adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(6), 2164–2173.
- Gomez, C. R., & Baird, S. (2005). Identifying early indicators for autism in self-regulation difficulties. *Focus on Autism and Other Developmental Disabilities*, 20(2), 106–116.
- Gottman, J. M. (1986). *The world of coordinated play: Same-and cross-sex friendship in young children*. Retrieved from <http://psycnet.apa.org/psycinfo/1987-97934-004>
- Graziano, P. A., Reavis, R. D., Keane, S. P., & Calkins, S. D. (2007). The role of emotion regulation in children's early academic success. *Journal of School Psychology*, 45(1), 3–19.
- Grolnick, W. S., Bridges, L. J., & Connell, J. P. (1996). Emotion regulation in two-year-olds: Strategies and emotional expression in four contexts. *Child Development*, 67(3), 928–941.
- Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology*, 2(3), 271.
- Gulsrud, A. C., Jahromi, L. B., & Kasari, C. (2010). The co-regulation of emotions between mothers and their children with autism. *Journal of Autism and Developmental Disorders*, 40(2), 227–237.
- Gumora, G., & Arsenio, W. F. (2002). Emotionality, emotion regulation, and school performance in middle school children. *Journal of School Psychology*, 40(5), 395–413.

- Gyurak, A., & Etkin, A. (2014). A neurobiological model of implicit and explicit emotion regulation. In *Handbook of emotional regulation* (2nd ed.). New York: Guilford, 20318.
- Hastings, R. P., & Bham, M. S. (2003). The relationship between student behaviour patterns and teacher burn-out. *School Psychology International*, 24(1), 115–127.
- Hastings, R. P., & Brown, T. (2002). Coping strategies and the impact of challenging behaviors on special educators' burnout. *Mental Retardation*, 40(2), 148–156.
- Hill, A. L., Degnan, K. A., Calkins, S. D., & Keane, S. P. (2006). Profiles of externalizing behavior problems for boys and girls across preschool: The roles of emotion regulation and inattention. *Developmental Psychology*, 42(5), 913.
- Hobson, R. P. (1990). On the origins of self and the case of autism. *Development and Psychopathology*, 2(2), 163–181.
- Hobson, R. P. (2010). Explaining autism: Ten reasons to focus on the developing self. *Autism*, 14(5), 391–407.
- Howse, R. B., Calkins, S. D., Anastopoulos, A. D., Keane, S. P., & Shelton, T. L. (2003). Regulatory contributors to children's kindergarten achievement. *Early Education and Development*, 14(1), 101–120.
- Jahromi, L. B., Meek, S. E., & Ober-Reynolds, S. (2012). Emotion regulation in the context of frustration in children with high functioning autism and their typical peers. *Journal of Child Psychology and Psychiatry*, 53(12), 1250–1258. <https://doi.org/10.1111/j.1469-7610.2012.02560.x>.
- Jahromi, L. B., Bryce, C. I., & Swanson, J. (2013). The importance of self-regulation for the school and peer engagement of children with high-functioning autism. *Research in Autism Spectrum Disorders*, 7(2), 235–246.
- John, O. P., & Gross, J. J. (2007). Individual differences in emotion regulation. In *Handbook of emotion regulation* (pp. 351–372).
- Khor, A. S., Melvin, G. A., Reid, S. C., & Gray, K. M. (2014). Coping, daily hassles and behavior and emotional problems in adolescents with high-functioning Autism/Asperger's disorder. *Journal of Autism and Developmental Disorders*, 44(3), 593–608.
- Konstantareas, M. M., & Stewart, K. (2006). Affect regulation and temperament in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 36(2), 143–154. <https://doi.org/10.1007/s10803-005-0051-4>.
- Kopp, C. B. (1989). Regulation of distress and negative emotions: A developmental view. *Developmental Psychology*, 25(3), 343.
- Loveland, K. A. (1984). Learning about points of view: Spatial perspective and the acquisition of 'I/you'. *Journal of Child Language*, 11(03), 535–556.
- Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 52(7), 679–688. <https://doi.org/10.1016/j.jaac.2013.05.006>.
- Mazefsky, C. A., Borue, X., Day, T. N., & Minshew, N. J. (2014). Emotion regulation patterns in adolescents with high-functioning autism spectrum disorder: Comparison to typically developing adolescents and association with psychiatric symptoms. *Autism Research*, 7(3), 344–354.
- McLean, K. C., Pasupathi, M., & Pals, J. L. (2007). Selves creating stories creating selves: A process model of self-development. *Personality and Social Psychology Review: An Official Journal of the Society for Personality and Social Psychology, Inc.*, 11(3), 262–278. <https://doi.org/10.1177/1088868307301034>.
- Morales, M., Mundy, P., Crowson, M. M., Neal, A. R., & Delgado, C. E. (2005). Individual differences in infant attention skills, joint attention, and emotion regulation behaviour. *International Journal of Behavioral Development*, 29(3), 259–263.
- Nelson, S., Korbut, S., Hedley, D., Dissanayake, C., & Nuske, H. (under review). *Parent-facilitated emotion regulation strategies predict positive school engagement and self-development in children with autism spectrum disorder*.
- Nuske, H. J., Vivanti, G., & Dissanayake, C. (2013). Are emotion impairments unique to, universal, or specific in autism spectrum disorder? A comprehensive review. *Cognition & Emotion*, 27(6), 1042–1061. <https://doi.org/10.1080/02699931.2012.762900>.
- Nuske, H. J., Hedley, D., Woollacott, A., Thomson, P., Macari, S., & Dissanayake, C. (2017). Developmental delays in emotion regulation strategies in preschoolers with autism. *Autism Research*, 10, 1808.
- Nuske, H. J., Hedley, D., Tseng, C. H., Begeer, S., & Dissanayake, C. (2018). Emotion regulation strategies in preschoolers with autism: Associations with parent quality of life and family functioning. *Journal of Autism and Developmental Disorders*, 48(4), 1287–1300.
- Patel, S., Day, T. N., Jones, N., & Mazefsky, C. A. (2017). Association between anger rumination and autism symptom severity, depression symptoms, aggression, and general dysregulation in adolescents with autism spectrum disorder. *Autism: The International Journal of Research and Practice*, 21(2), 181–189. <https://doi.org/10.1177/1362361316633566>.
- Pfeifer, J. H., & Peake, S. J. (2012). Self-development: Integrating cognitive, socioemotional, and neuroimaging perspectives. *Developmental Cognitive Neuroscience*, 2(1), 55–69. <https://doi.org/10.1016/j.dcn.2011.07.012>.
- Pouw, L. B., Rieffe, C., Oosterveld, P., Huskens, B., & Stockmann, L. (2013a). Reactive/proactive aggression and affective/cognitive empathy in children with ASD. *Research in Developmental Disabilities*, 34(4), 1256–1266.
- Pouw, L. B., Rieffe, C., Stockmann, L., & Gadow, K. D. (2013b). The link between emotion regulation, social functioning, and depression in boys with ASD. *Research in Autism Spectrum Disorders*, 7(4), 549–556.

- Raver, C. C. (1996). Relations between social contingency in mother-child interaction and 2-year-olds' social competence. *Developmental Psychology*, 32(5), 850.
- Rieffe, C., Oosterveld, P., Terwogt, M. M., Mootz, S., Van Leeuwen, E., & Stockmann, L. (2011). Emotion regulation and internalizing symptoms in children with autism spectrum disorders. *Autism*. <https://doi.org/10.1177/1362361310366571>.
- Rubin, K. H., Coplan, R. J., Fox, N. A., & Calkins, S. D. (1995). Emotionality, emotion regulation, and preschoolers' social adaptation. *Development and Psychopathology*, 7(01), 49–62.
- Ryan, R. M., & Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist*, 55(1), 68.
- Samson, A. C., Huber, O., & Gross, J. J. (2012). Emotion regulation in Asperger's syndrome and high-functioning autism. *Emotion*, 12(4), 659.
- Samson, A. C., Phillips, J. M., Parker, K. J., Shah, S., Gross, J. J., & Hardan, A. Y. (2014). Emotion dysregulation and the core features of autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(7), 1766–1772.
- Samson, A. C., Hardan, A. Y., Lee, I. A., Phillips, J. M., & Gross, J. J. (2015a). Maladaptive behavior in autism spectrum disorder: The role of emotion experience and emotion regulation. *Journal of Autism and Developmental Disorders*, 45(11), 3424–3432.
- Samson, A. C., Wells, W. M., Phillips, J. M., Hardan, A. Y., & Gross, J. J. (2015b). Emotion regulation in autism spectrum disorder: Evidence from parent interviews and children's daily diaries. *Journal of Child Psychology and Psychiatry*, 56(8), 903–913.
- Santomauro, D., Sheffield, J., & Sofronoff, K. (2017). Investigations into emotion regulation difficulties among adolescents and young adults with autism spectrum disorder: A qualitative study. *Journal of Intellectual & Developmental Disability*, 42(3), 275–284.
- Scarpa, A., & Reyes, N. M. (2011). Improving emotion regulation with CBT in young children with high functioning autism spectrum disorders: A pilot study. *Behavioural and Cognitive Psychotherapy*, 39(04), 495–500. <https://doi.org/10.1017/S1352465811000063>.
- Schreibman, L., Dawson, G., Stahmer, A. C., Landa, R., Rogers, S. J., McGee, G. G., ... others. (2015). Naturalistic developmental behavioral interventions: Empirically validated treatments for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45, 2411–2428.
- Silk, J. S., Steinberg, L., & Morris, A. S. (2003). Adolescents' emotion regulation in daily life: Links to depressive symptoms and problem behavior. *Child Development*, 74(6), 1869–1880.
- Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development*, 59(2–3), 25–52.
- Ting, V., & Weiss, J. A. (2017). Emotion regulation and parent co-regulation in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(3), 680–689. <https://doi.org/10.1007/s10803-016-3009-9>.
- Totsika, V., Hastings, R. P., Emerson, E., Lancaster, G. A., & Berridge, D. M. (2011). A population-based investigation of behavioural and emotional problems and maternal mental health: Associations with autism spectrum disorder and intellectual disability. *Journal of Child Psychology and Psychiatry*, 52(1), 91–99.
- Vivanti, G., & Rogers, S. J. (2014). Autism and the mirror neuron system: Insights from learning and teaching. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 369(1644), 20130184.
- Vivanti, G., Hocking, D. R., Fanning, P., & Dissanayake, C. (2016). Social affiliation motives modulate spontaneous learning in Williams syndrome but not in autism. *Molecular Autism*, 7(1), 40.
- Zantinge, G., van Rijn, S., Stockmann, L., & Swaab, H. (2017). Physiological arousal and emotion regulation strategies in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 47(9), 2648–2657.
- Zimmer-Gembeck, M. J., & Skinner, E. A. (2011). Review: The development of coping across childhood and adolescence: An integrative review and critique of research. *International Journal of Behavioral Development*, 35(1), 1–17.
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, 23(2), 143–152.

Emotional Intelligence

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Definition

Emotional intelligence is defined as “the capacity to reason about emotions, and of emotions to enhance thinking. It includes the ability to accurately perceive emotions, to access and generate emotions so as to assist thought, to understand emotions and emotional knowledge, and to reflectively regulate emotions so as to promote

emotional and intellectual growth” (Mayer et al. 2004, p. 197).

Historical Background

Mayer and colleagues (Mayer et al. 2004; Mayer and Salovey 1993) view emotional intelligence as belonging to what they term “hot intelligences,” which essentially operate at the intersection of emotion processing and higher-level cognition, that is, between thinking and feeling. This conceptualization is reflected in their “four-branch ability model” in which they propose the following components of emotional intelligence: (1) perceiving emotion, (2) using emotion to assist thought, (3) understanding emotions, and (4) managing emotions in oneself and in others.

Current Knowledge

The concept of emotional intelligence (or “EQ”) has captured the imagination of non-psychologists, who have broadened the term to include not only the ability to understand and use emotional information but also to describe personality attributes and the capacity to relate to others in social situations (e.g., Goleman 1998). The concept is not without controversy, however. Academic scholars have been less willing to adopt the term “emotional intelligence” in scientific parlance, and the construct as described above has not been the subject of sustained, systematic investigation. Indeed, it has been described as an “elusive concept” (Davies et al. 1998, p. 989), one which “appears more myth than science...” (Matthews et al. 2002, p. 547).

Emotional Intelligence in Autism

Perhaps unsurprisingly, the term “emotional intelligence” has not been embraced by researchers in the field of autism. In fact, a PubMed search for articles from January 1970 until August 2011 with the terms “emotional intelligence” AND “autistic disorder” OR “autism” yielded 17 articles of which only one directly investigated emotional intelligence in autism (Petrides et al. 2011).

These authors reported lower scores on a trait emotional intelligence measure by adults with Asperger syndrome relative to typical adults.

This is not to say that the majority of the “processes” in Mayer et al.’s “branches” have not been systematically studied in autism, albeit under different – and arguably better validated – guises.

First, there is an extensive body of work investigating how people with autism perceive and recognize emotion in faces, voices, and other non-verbal cues (Branch 1). Second, the critical role that emotion plays in decision making and executive functions more broadly has been well documented in the burgeoning social neuroscience literature (e.g., Damasio 1994; Pessoa 2008). There have been several recent investigations of social decision making – problems that involve the regulation of affect and motivation – in autism (using the Iowa gambling task, for example), although there have been comparatively fewer investigations of the development of these processes either under the headings of “self-regulation” (Posner and Rothbart 2000) or “hot executive function” (Carlson et al. 2005; Happaney et al. 2004) (Branches 2 and 4). Finally, Baron-Cohen’s (2002) “empathizing” construct covers many of the abilities described in Branch 3 – those that are important for both identifying the thoughts and feelings of others *and* using that information to respond in appropriate ways – which may overlap with other heavily researched constructs like “theory of mind.” Indeed, Blair (2002) considers whether theory of mind might be a necessary prerequisite for the development of emotional intelligence.

Future Directions

Despite the limited empirical focus on the nature of the construct of emotional intelligence in autism, references to autistic individuals’ “lack of emotional intelligence” abound in the popular press and on the Internet. Caution is warranted, however, on the use of these and other extreme statements (e.g., “zero degrees of empathy”; Baron-Cohen (2011), etc.) (see Pellicano and

Stears 2011): They are often considered disparaging to the individuals concerned and often accurately reflect neither the empirical data nor the inner emotional lives of many people with autism (Cohen-Rottenberg 2011). Future research will need to take a more nuanced approach.

See Also

- ▶ Affective Development
- ▶ Emotional Regulation
- ▶ Empathizing-Systemizing Theory
- ▶ Empathy
- ▶ Executive Function (EF)
- ▶ Motivation
- ▶ Social Cognition
- ▶ Theory of Mind

References and Reading

- Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences*, 6, 248–254.
- Baron-Cohen, S. (2011). *Zero degrees of empathy: A new theory of human cruelty*. London: Penguin Books.
- Blair, R. J. R. (2002). Theory of mind, autism and emotional intelligence. In L. F. Barrett & P. Salovey (Eds.), *The wisdom in feeling: Psychological processes in emotional intelligence* (pp. 406–434). New York: The Guilford Press.
- Carlson, S. M., Davis, A. C., & Leach, J. G. (2005). Less is more: Executive function and symbolic representation in preschool children. *Psychological Science*, 16, 609–616.
- Cohen-Rottenberg, R. (2011). *The empathy issue is a human rights issue*. Accessed September 8, 2011, from <http://www.journeyswithautism.com/2011/09/05/the-empathy-issue/>
- Damasio, A. R. (1994). *Descartes' error*. New York: Putnam.
- Davies, M., Stankov, L., & Roberts, R. D. (1998). Emotional intelligence: In search of an elusive construct. *Journal of Personality and Social Psychology*, 75, 989–1015.
- Goleman, D. (1998). *Working with emotional intelligence*. New York: Bantam Books.
- Happaney, K. R., Zelazo, P. D., & Stuss, D. T. (2004). Development of orbitofrontal function: Current themes and future directions. *Brain and Cognition*, 55, 1–10.
- Matthews, G., Zeidner, M., & Roberts, R. D. (2002). *Emotional intelligence: Science and myth*. Cambridge, MA: MIT Press.
- Mayer, J. D., & Salovey, P. (1993). The intelligence of emotional intelligence. *Intelligence*, 17, 433–442.
- Mayer, J. D., Salovey, P., & Caruso, D. R. (2004). Emotional intelligence: Theory, findings, and implications. *Psychological Inquiry*, 15, 197–215.
- Pellicano, E., & Stears, M. (2011). Bridging autism, science and society: Moving towards an ethically-informed approach to autism research. *Autism Research*, 4, 271–282.
- Pessoa, L. (2008). On the relationship between emotion and cognition. *Nature Reviews Neuroscience*, 9, 148–158.
- Petrides, K. V., Hudry, K., Michalaria, G., Swami, V., & Sevdalis, N. (2011). A comparison of the trait emotional intelligence profiles of individuals with and without Asperger syndrome. *Autism*, 15, 671–682.
- Posner, M. I., & Rothbart, M. K. (2000). Developing mechanisms of self-regulation. *Development and Psychopathology*, 12, 427–441.

Emotional Processing in ASD

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Definition

Emotional processing refers to the ability to evaluate one's own and others' feelings, using information from current cues and prior knowledge about social relationships and contexts. Relevant emotional information includes visual cues, such as facial expressions, body posture, and gestures, and auditory cues such as vocal expressions (e.g., sighs and exclamations), prosody, and tone of voice. Emotional information may also be present in written text and spoken conversation. Being able to process emotional information is a developmental skill that increases in competence and complexity as children learn to identify people's feelings in social situations, attribute reasons for the emotions, and regulate their own behaviors and emotional responses to achieve social goals (Lerner and Arsenio 2000).

Emotional processing can be indexed by an individual's ability to recognize and label

emotions, describe emotions, and reason about possible causes of emotions. These tasks involve skills in information-processing and social cognition, including deriving emotional meaning from single or multiple cues, applying learned information about emotional responses and social norms to current situations, taking others' perspectives, and inferring others' internal states and intentions. The ability to make use of relevant background knowledge is particularly important for evaluating emotions that are socially based in interpersonal relationships or situations (e.g., *jealousy*, *pride*, *shame*), whereas basic or nonsocial emotions can sometimes be inferred from surface cues without reference to other people or the environment (e.g., *happiness*, *fear*). Social cognitive and emotional processes appear to be highly interconnected in neurotypical individuals (Olsson and Ochsner 2008), but these processes may not be as well integrated in individuals with ASD (Happé and Frith 2014).

In general, emotional processing in individuals with ASD tends to be impaired when compared to neurotypicals, including deficits in recognizing others' emotions, using emotional language, describing emotional concepts, and attributing causes for emotions. Further, processing of social emotions tends to be more impaired than of nonsocial emotions (Williams and Happé 2010). However, variability in abilities exists, and performance seems to be associated with cognitive, linguistic, and theory of mind abilities. The development of language-based empirical methods to investigate emotional processing in ASD has also facilitated an emerging view that emotions may be conceptualized, and/or experienced, differently in individuals with ASD (Losh and Capps 2006).

Historical Background

Kanner's (1943) seminal paper entitled *Autistic Disturbances of Affective Contact* documented multiple features suggesting innate emotional abnormalities in autistic children, including a "profound aloneness" (p. 248). He noted the extreme lack of interest in interpersonal relations which contrasted starkly with their ability to

maintain a meaningful relation to objects. Supporting Kanner's position, early experiments on emotional recognition reported that children with autism spectrum disorders (ASD) were less able to discriminate and recognize people's emotions from facial expressions, vocal expressions, and gestures, than non-autistic children matched on age, cognitive functioning, and/or language development (e.g., Hobson 1986a, b). Such findings contributed toward a prevailing view that there existed a "basic" social-affective impairment in children with ASD which reduced their performance in emotion recognition tasks above and beyond any deficits in domain-general cognitive processing and/or language abilities (Fein et al. 1986; Hermelin and O'Connor 1985). However, results from subsequent research were more equivocal and included reports of near-normative performance in recognizing at least one emotion, *happiness*, by individuals with ASD (see review by Uljarevic and Hamilton 2013). This variability in findings may be partially attributable to differences in sample characteristics such as chronological age, cognitive, and language abilities (Begeer et al. 2008; Lartseva et al. 2014). Crucially, methods using expressive language paradigms have allowed researchers to observe difficulties or differences in the conceptualization of emotions in individuals with ASD, compared to typically developing controls. Hence, instead of a universal emotional processing deficit in ASD, recent reviews point to the individual nature of the pattern of impairments (Leekam 2016; Nuske et al. 2013). The next section will provide a summary of the current knowledge arising from the variety of empirical methods that have been developed to characterize the patterns of skills and deficits in emotional processing in individuals with ASD.

Current Knowledge

Emotional processing has been studied both in terms of emotional recognition and emotional expression in individuals with ASD. Emotional recognition paradigms facilitate an examination of how individuals with ASD perceive and

evaluate different types of emotional cues. By comparison, emotional expression paradigms allow researchers to study the extent to which individuals with ASD conceptualize and reason about emotions in themselves and others. Findings from the different paradigms will be discussed in turn below, followed by a brief consideration of theoretical models of ASD that may explain the emotional processing deficits.

Emotional Recognition

Recognition from visual and auditory stimuli.

Early studies sought to examine ability to discriminate and recognize emotions in other people. The classic emotional processing experiment is where participants are presented with stimuli and asked to match or label the emotions depicted. Emotional stimuli include face photographs (e.g., Tantam et al. 1989), video clips where a person portrays different emotions in isolation or in contextualized scenarios (e.g., Hobson 1986a), video clips showing bodily expressions of emotions with the actor's face obscured (Hobson 1986b), and audio clips of emotionally expressive voices (e.g., Hobson et al. 1988). In these studies, emotional processing involves evaluating facial expressions, prosodic cues, "body language," and/or contextual cues. Multiple early studies reported that children with ASD were less accurate at recognizing emotions from faces and voices than comparison groups matched on one or more variables of chronological age, cognitive functioning, and verbal abilities (e.g., Hobson 1986a, b; Hobson et al. 1988).

However, later studies did not always find significant group differences in abilities (e.g., Prior et al. 1990). Uljarevic and Hamilton's (2013) meta-analysis of 48 emotion recognition studies using picture matching/labeling experiments revealed that there was a large overall emotion recognition deficit (Cohen's $d = 0.80$) in children and adults with ASD compared to control groups matched on age, IQ, and/or verbal ability. The emotions tested were one or more of the six basic or nonsocial emotions: *happiness*, *sadness*, *anger*, *fear*, *disgust*, and *surprise* (see Emotion). Basic emotions are typically recognized relatively early in development (Izard 1977), can often be

identified from surface cues such as smiles (for *happiness*) and frowns (for *anger*), and are non-social in the sense that they can be felt without reference to other people (Heerey et al. 2003). Despite reporting an overall mean emotion recognition deficit in ASD, Uljarevic and Hamilton noted the large variability in outcomes across studies and the lack of a significant correlation with age or IQ. Additionally, participants with ASD showed near-normal recognition of one emotion, *happiness*, which suggests that processing is not equally impaired for all types of emotions. Interestingly, studies comparing recognition of negative and positive emotions reported that adults with ASD made more errors on negative emotions, particularly *fear*, than on positive emotions, whereas no such valence effect was seen in typically developing adults (e.g., Ashwin et al. 2006; Philip et al. 2010). This suggested that valence may moderate emotional recognition abilities in ASD.

Recognition in text. Besides facial stimuli, researchers have investigated the ability of individuals with ASD to recognize emotions in spoken and/or written text, at the level of words (e.g., Järvinen-Pasley et al. 2008), sentences (e.g., Grossman et al. 2010), and stories (e.g., Hillier and Allinson 2002). Participants are usually asked to identify emotions explicitly stated in the stimuli (e.g., *happy* emotion: "I am feeling happy today"), to infer emotions from the text (e.g., *sad* emotion: "John cried when he lost his dog"), or, in the case of spoken stimuli, to infer emotions from its prosodic intonation. There are mixed findings from these text paradigms. Some researchers have found poorer accuracy of emotional judgment in participants with ASD than in typically developing controls matched on age, IQ, and/or verbal ability (e.g., Järvinen-Pasley et al. 2008), while others have reported no group differences in abilities (e.g., Grossman et al. 2010).

Compensatory strategies for emotional recognition. One caveat arising from findings with visual, auditory, and text stimuli is that individuals with ASD who have higher cognitive functioning and/or language abilities may be able to use alternative strategies to decode emotions. For example, they could analyze or draw upon

lexical knowledge for emotional words. A review by Begeer et al. (2008) reported that children with ASD with average or above-average IQ can recognize basic emotions in pictures on a par with age-matched typically developing children, whereas children with ASD with a lower IQ may have difficulty even with basic emotions. Lindner and Rosén (2006) compared ability to recognize emotions from video and audio stimuli, with and without verbal content, and found that verbal content facilitated emotional understanding in children with ASD (Asperger Syndrome), i.e., linguistic information might have been used as a compensatory strategy for deficits in emotional processing of facial expressions and prosody.

Hence researchers have argued that laboratory tasks on emotional recognition in individuals with ASD requiring explicit emotional judgments do not necessarily measure emotional processing abilities alone. Moreover, other studies using implicit emotional processing paradigms have shown that emotionality significantly affects response time and/or accuracy in typically developing controls (e.g., in memory or word repetition tasks), but it has limited or no influence on performance in individuals with ASD (Deruelle et al. 2008; Singh and Harrow 2014). Taken together, the results suggest a selective impairment in spontaneous processing and integration of emotional cues in individuals with ASD, which may reduce their ability to evaluate and learn about emotions in real-life experiences. To better measure spontaneous emotional processing and gain insight into the conceptual understanding of emotions in individuals with ASD, emotional expression paradigms may be used.

Emotional Expression

Emotional language use. The quantity and appropriateness of emotional labels produced in language samples is one way to compare spontaneous emotional processing in individuals with and without ASD. Researchers have collected language samples by eliciting story narratives using sequenced pictures (e.g., Canfield et al. 2016), picture books (e.g., Rumpf et al. 2012), or contextualized social and/or emotional pictures

(e.g., Teh et al. 2018). Other researchers have recorded language samples from participants during conversations with their family members (e.g., Tager-Flusberg 1992) or with the experimenter on selected emotional and nonemotional topics (e.g., Bang et al. 2013). Some researchers have reported fewer emotional terms produced by children with ASD compared to matched typically developing children (Canfield et al. 2016; Teh et al. 2018) or to age and IQ/verbal IQ-matched controls in other clinical groups (e.g., Tager-Flusberg (1992) on children with ASD and children with Down's syndrome). In contrast, others have reported no significant differences in production of emotional terms between groups with and without ASD (Bang et al. 2013; Rumpf et al. 2012). Two studies have reported a greater use of negative than positive emotional language by both children with ASD and typically developing children (Rumpf et al. 2012; Teh et al. 2018), but little is currently known about valence effects in this area. However, the quantity of internal state language (including emotions) is associated with age and theory of mind skills (Siller et al. 2014) and cognitive ability (Capps et al. 2000), so variability in sample characteristics could partially explain the mixed findings in the literature. It should also be highlighted that most studies included emotional language production as part of investigations into broader discourse skills, such as narrative organization and conversational skills, which are often impaired in children with ASD (Canfield et al. 2016; Bang et al. 2013). Thus, further studies designed to look specifically at emotional processing abilities are needed, with well-characterized samples, before these findings can be integrated productively.

Reasoning about emotions. Contrasting with the mixed picture on use of emotional language above, children with ASD have been consistently reported to show challenges with reasoning about emotions. In story narratives, children with ASD reportedly make fewer causal attributions for emotions than matched typically developing children, and the causes they suggest tend to refer to characters' actions or visually salient emotion cues, rather than the characters' intentions or desires (Canfield et al. 2016; Capps

et al. 2000). These findings are consistent with reported theory of mind deficits in ASD that reduce their ability to appreciate or evaluate others' mental states (Baron-Cohen et al. 1985; see ► [Theory of Mind](#)). Additionally, when talking about their own emotional experiences, children with ASD tend to attribute their emotions to events and actions, whereas typically developing children commonly contextualize their emotions within a personal or social perspective (e.g., Losh and Capps 2006; Rieffe et al. 2007). Such group differences in reasoning about emotions raises the question of whether conceptualization of emotions may differ in children with ASD and typically developing children.

Emotional concepts. Data from emotion interviews reveal that emotional concepts may be underdeveloped in children with ASD. In emotion interviews, participants are asked to define named emotions, describe situations that exemplify those emotions, and/or share personal experiences of the named emotions. Researchers have commented that descriptions and personal accounts of emotions by children with ASD tend to be less coherent and less detailed than those of typically developing controls (e.g., Ben-Itzhak et al. 2016; Rieffe et al. 2007). The result is descriptions that sometimes appear inappropriate, although not completely wrong, for the named emotions (e.g., feeling *proud* upon receiving a present; Losh and Capps 2006). Moreover, Yirmiya et al. (1992) reported that performance correlated positively with cognitive functioning in ASD, a relationship which was not present in typically developing control children, again suggesting that emotional processing in ASD may be mediated by other developmental skills. Additionally, ability to process negative emotions may be more impaired than positive emotions in children with ASD, as evidenced by fewer reports and poorer descriptions of negative than positive emotions by children with ASD compared to typically developing children (e.g., Ben-Itzhak et al. 2016; Rieffe et al. 2007). Furthermore, impairments with emotional concepts may be influenced by challenges in relating social contexts and relationships to emotional outcomes, as discussed next.

Moderating effect of social context.

Emotional processing is often closely related to processing of social information. However, for people with ASD, social cognitive studies have generally revealed deficits in the ability to perceive and/or interpret social cues, link socially relevant stimuli with retrieval of social knowledge, and evaluate others' social intentions (e.g., Flood et al. 2011). Teh et al. (2018) demonstrated that increasing social engagement information in pictures reduced the frequency of emotional language produced by children with ASD, but increased production of emotional language by typically developing children, particularly in positively valenced conditions. Perhaps social contexts require complex processing skills due to multiple sources of information, including current cues and prior knowledge, that need to be integrated to support emotional processing (Minshew and Goldstein 1998). Presumably, the need to process and integrate multiple cues explains why socially based emotions, such as *embarrassment*, *pride*, or *shame*, are reportedly more challenging for children with ASD to recognize (Williams and Happé 2010) and describe (Heerey et al. 2003; Losh and Capps 2006) than nonsocial emotions, since socially based emotions require an ability to consider others' perspectives and have an awareness of social norms and their violations. The processing imbalance between social and nonsocial emotions has also been observed in children with learning disabilities (Williams and Happé 2010). Hence, recent reviewers have debated whether and how emotional processing in ASD may be associated with broader deficits in ► [“Social Cognition”](#) characteristic of ASD and/or other domain-general information-processing deficits (Leekam 2016).

Theories Underlying Emotional Processing Deficits in ASD

There are at least four theories explaining emotional processing deficits in ASD: (1) reduced social motivation in ASD (see review by Chevallier et al. 2012), which partially explains lack of attention to social stimuli; (2) cognitive attention biases and rigidity or selective complex processing impairments (Minshew and Goldstein 1998) that

reduce the ability of people with ASD to combine and interpret multiple contextual cues and use relevant background knowledge to understand emotions; (3) theory of mind impairments that limit ability to attribute mental states to others (Baron-Cohen et al. 1985); and (4) ► “Weak Central Coherence”, which helps to explain difficulties in holistically processing relevant cues from the face, voice, body posture, and/or situational contexts to understand emotions (Happé and Frith 2006). However, no one theory is yet able to adequately explain the range of emotional processing skills and deficits reported in ASD. Furthermore, age, language, and cognitive skills may be positively associated with theory of mind development, social motivation, or even global processing abilities (Begeer et al. 2008; Steele et al. 2003), but the uneven profile of these skills in ASD has made these relationships difficult to ascertain.

Summary

Taken together, the extant research on recognition and expression of emotions by individuals with ASD has revealed quantitative and qualitative variation in the pattern and extent of emotional processing skills and impairments in ASD, across a wide range of experimental paradigms. While there is a general consensus in the literature that emotional processing is impaired in ASD compared to typically developing controls, there are contrary findings in studies that may be partially attributable to participant sampling. More recent evidence from self-report studies suggest that emotional concepts are underdeveloped in children with ASD (Rieffe et al. 2007). There is also emerging evidence that the pattern of emotional processing deficits may be uneven across conditions of valence and social engagement. Given that the range of skills and deficits is so varied, more than one mechanism appears to underlie emotional processing deficits in ASD. Further research is needed to clarify all these points.

Future Directions

After decades of research on emotional processing skills in ASD, the question remains unanswered

about whether emotional processing deficits are core in ASD symptomatology, as originally proposed by Kanner (1943), or whether deficits arise as a downstream effect of social cognitive deficits or other domain-general processing difficulties. Longitudinal studies may be useful to observe development of skills with age in children with ASD and may eventually enable researchers to unravel cause-and-effect relationships among social/emotional learning and domain-general cognitive, linguistic, and other skills development. Alternatively, more cohort studies are needed, using well-characterized samples on variables such as age, cognitive functioning, language, and theory of mind development, because the heterogeneous nature of ASD has made it difficult so far to integrate findings across studies and systematically explore relationships between participant characteristics and emotional processing.

Questions also remain over how processing of basic (nonsocial) and complex (social) emotions is different in individuals with ASD compared to other groups. A few studies have demonstrated valence effects on emotional recognition and expression in ASD, but research remains sparse in this area, and little is known about the processing of emotionally neutral situations in ASD (Uljarevic and Hamilton 2013). Hence, it is important for future researchers to systematically manipulate valence and social variables in experimental stimuli in order to examine their separate or combined/interactive effects on emotional processing. Lastly, emotional language use by individuals with ASD is an area that has received limited attention in the literature to date (Lartseva et al. 2014). Studies using this measure have revealed quantitative and qualitative differences in the way people with ASD process and respond to emotional stimuli compared to typically developing controls. Emerging findings from children with ASD also suggest that differences exist in the way emotions are conceptualized from young, in terms of how they reason about and/or experience emotions. Thus emotional language research shows potential to shed light on many questions regarding emotional processing skills in ASD, and further work in this area will be useful.

See Also

- ▶ Affective Development
- ▶ Social Cognition
- ▶ Theory of Mind
- ▶ Weak Central Coherence

References and Reading

- Ashwin, C., Chapman, E., Colle, L., & Baron-Cohen, S. (2006). Impaired recognition of negative basic emotions in autism: A test of the amygdala theory. *Social Neuroscience, 1*, 349–363.
- Bang, J., Burns, J., & Nadig, A. (2013). Brief report: Conveying subjective experience in conversation: Production of mental state terms and personal narratives in individuals with high functioning autism. *Journal of Autism and Developmental Disorders, 43*, 1732–1740.
- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a “theory of mind?”. *Cognition, 21*, 37–46.
- Begeer, S., Koot, H. M., Rieffe, C., Meerum Terwogt, M., & Stegge, H. (2008). Emotional competence in children with autism: Diagnostic criteria and empirical evidence. *Developmental Review, 28*, 342–369.
- Ben-Itzhak, E., Abutbul, S., Bela, H., Shai, T., & Zachor, D. A. (2016). Understanding one’s own emotions in cognitively-able preadolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 46*, 2363–2371.
- Canfield, A. R., Eigsti, I. M., de Marchena, A., & Fein, D. (2016). Story goodness in adolescents with autism spectrum disorder (ASD) and in optimal outcomes from ASD. *Journal of Speech, Language, and Hearing Research, 59*, 1–13.
- Capps, L., Losh, M., & Thurber, C. (2000). “The frog ate the bug and made his mouth sad”: Narrative competence in children with autism. *Journal of Abnormal Child Psychology, 28*, 193–204.
- Chevallier, C., Kohls, G., Troiani, V., Brodtkin, E. S., & Schultz, R. T. (2012). The social motivation theory of autism. *Trends in Cognitive Sciences, 16*, 231–239.
- Deruelle, C., Hubert, B., Santos, A., & Wicker, B. (2008). Negative emotion does not enhance recall skills in adults with autistic spectrum disorders. *Autism Research, 1*, 91–96.
- Fein, D., Pennington, B., Markowitz, P., Braverman, M., & Waterhouse, L. (1986). Toward a neuropsychological model of infantile autism: Are the social deficits primary? *Journal of the American Academy of Child Psychiatry, 25*, 198–212.
- Flood, A. M., Hare, D. J., & Wallis, P. (2011). An investigation into social information processing in young people with Asperger syndrome. *Autism, 15*, 601–624.
- Grossman, R. B., Bemis, R. H., Skwerer, D. P., & Tager-Flusberg, H. (2010). Lexical and affective prosody in children with high-functioning autism. *Journal of Speech, Language, and Hearing Research, 53*, 778–793.
- Happé, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders, 36*, 5–25.
- Happé, F., & Frith, U. (2014). Annual research review: Towards a developmental neuroscience of atypical social cognition. *Journal of Child Psychology and Psychiatry, 55*, 553–577.
- Heerey, E. A., Keltner, D., & Capps, L. M. (2003). Making sense of self-conscious emotion: Linking theory of mind and emotion in children with autism. *Emotion, 3*, 394–400.
- Hermelin, B., & O’Connor, N. (1985). Logico-affective states and nonverbal language. In E. Schopler & G. B. Mesibov (Eds.), *Communication problems in autism* (pp. 283–310). Plenum Press: New York.
- Hillier, A., & Allinson, L. (2002). Understanding embarrassment among those with autism: breaking down the complex emotion of embarrassment among those with autism. *Journal of Autism and Developmental Disorders, 32*, 583–592.
- Hobson, R. P. (1986a). The autistic child’s appraisal of expressions of emotions. *Journal of Child Psychology and Psychiatry, 27*, 321–342.
- Hobson, R. P. (1986b). The autistic child’s appraisal of expressions of emotion: A further study. *Journal of Child Psychology and Psychiatry, 27*, 671–680.
- Hobson, R. P., Ouston, J., & Lee, A. (1988). Emotion recognition in autism: Coordinating faces and voices. *Psychological Medicine, 18*, 911–923.
- Izard, C. E. (1977). Human emotions. New York: Plenum Press.
- Järvinen-Pasley, A., Peppé, S., King-Smith, G., & Heaton, P. (2008). The relationship between form and function level receptive prosodic abilities in autism. *Journal of Autism and Developmental Disorders, 38*, 1328–1340.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child, 2*, 217–250.
- Lartseva, A., Dijkstra, T., & Buitelaar, J. K. (2014). Emotional language processing in autism spectrum disorders: A systematic review. *Frontiers in Human Neuroscience, 8*, 991.
- Leekam, S. (2016). Social cognitive impairment and autism: What are we trying to explain? *Philosophical Transactions of the Royal Society B, 371*(1686).
- Lemerise, E. A., & Arsenio, W. F. (2000). An integrated model of emotion processes and cognition in social information processing. *Child Development, 71*, 107–118.
- Lindner, J. L., & Rosén, L. A. (2006). Decoding of emotion through facial expression, prosody and verbal content in children and adolescents with Asperger’s syndrome. *Journal of Autism and Developmental Disorders, 36*, 769–777.

- Losh, M., & Capps, L. (2006). Understanding of emotional experience in autism: Insights from the personal accounts of high-functioning children with autism. *Developmental Psychology, 42*, 809–818.
- Minschew, N. J., & Goldstein, G. (1998). Autism as a disorder of complex information processing. *Mental Retardation and Developmental Disabilities Research Reviews, 4*, 129–135.
- Nuske, H. J., Vivanti, G., & Dissonayake, C. (2013). Are emotion impairments unique to, universal, or specific in autism spectrum disorder? A comprehensive review. *Cognition and Emotion, 27*, 1042–1061.
- Olsson, A., & Ochsner, K. N. (2008). The role of social cognition in emotion. *Trends in Cognitive Sciences, 12*, 65–71.
- Philip, R. C., Whalley, H. C., Stanfield, A. C., Sprengelmeyer, R., Santos, I. M., Young, A. W., ... Hall, J. (2010). Deficits in facial, body movement and vocal emotional processing in autism spectrum disorders. *Psychological Medicine, 40*, 1919–1929.
- Prior, M., Dahlstrom, B., & Squires, T. (1990). Autistic children's knowledge of thinking and feeling states in other people. *Journal of Child Psychology and Psychiatry, 31*, 587–601.
- Rieffe, C., Meerum Terwogt, M., & Kotronopoulou, K. (2007). Awareness of single and multiple emotions in high-functioning children with autism. *Journal of Autism and Developmental Disorders, 37*, 455–465.
- Rumpf, A. L., Kamp-Becker, I., Becker, K., & Kauschke, C. (2012). Narrative competence and internal state language of children with Asperger Syndrome and ADHD. *Research in Developmental Disabilities, 33*, 1395–1407.
- Siller, M., Swanson, M. R., Serlin, G., & Teachworth, A. G. (2014). Internal state language in the storybook narratives of children with and without autism spectrum disorder: Investigating relations to theory of mind abilities. *Research in Autism Spectrum Disorders, 8*, 589–596.
- Singh, L., & Harrow, M. S. (2014). Influences of semantic and prosodic cues on word repetition and categorization in autism. *Journal of Speech, Language, and Hearing Research, 57*, 1764–1778.
- Steele, S., Joseph, R. M., & Tager-Flusberg, H. (2003). Brief report: Developmental change in theory of mind abilities in children with autism. *Journal of Autism and Developmental Disorders, 33*, 461–467.
- Tager-Flusberg, H. (1992). Autistic children's talk about psychological states: Deficits in the early acquisition of a theory of mind. *Child Development, 63*, 161–172.
- Tantam, D., Monaghan, L., Nicholson, H., & Stirling, J. (1989). Autistic children's ability to interpret faces: A research note. *Journal of Child Psychology and Psychiatry, 30*, 623–630.
- Teh, E. J., Yap, M. J., & Rickard Liow, S. J. (2018). Emotional processing in autism spectrum disorders: Effects of age, emotional valence, and social engagement on emotional language use. *Journal of Autism and Developmental Disorders, 48*, 4138–4154.
- Uljarevic, M., & Hamilton, A. (2013). Recognition of emotions in autism: A formal meta-analysis. *Journal of Autism and Developmental Disorders, 43*, 1517–1526.
- Williams, D., & Happé, F. (2010). Recognising 'social' and 'non-social' emotions in self and others: A study of autism. *Autism, 14*, 285–304.
- Yirmiya, N., Sigman, M. D., Kasari, C., & Mundy, P. (1992). Empathy and cognition in high-functioning children with autism. *Child Development, 63*, 150–160.

Emotional Regulation

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Synonyms

[Affective regulation](#); [ER](#)

Definition

Emotional regulation (ER, also known as affective regulation) is an area of research that has experienced significant growth in interest during the last two decades. In brief, ER is a person's attempt to regulate or influence the type and degree of emotional response he or she has to a stimulus, the timing of that emotional response, how that emotional response is experienced, or how that emotional response is expressed to others (Gross 2007). ER capacity is a person's ability to regulate their emotions toward adaptive ends.

ER is an aspect of self-regulation. All living organisms engage in processes of self-regulation in order to adapt to environmental demands. In humans, self-regulation is a developmental process, by which individuals gain greater control over their own physiological, emotional, cognitive/attentional, and behavioral responses to their environments. These response domains and their regulation are interrelated. When the environment poses manageable challenges to which a person

can draw upon his or her personal resources to meet these challenges, self-regulatory capacities are strengthened and adaptive development occurs. When the environment poses challenges beyond the capacities of the individual to adapt, maladaptive or disordered patterns of behavior may become evident (National Research Council & Institute of Medicine 2000).

Emotions play an important role in social relationships, both as a way to respond to others and as a way to elicit social connections from others. During typical development, emotional expression develops from generalized arousal states (e.g., a “fussy” baby) to more differentiated emotional responses (e.g., anger, sadness, frustration, fear), while the development of cognitive capacities helps the person to better understand the environmental antecedents of their emotional responses, identify emotional states in others, and develop self-coping strategies. These self-coping strategies are often mediated by language (self-talk), and evidence has long suggested that delayed or impaired language development may have a negative impact on the development of ER.

Aspects of ER capacity include the ability to identify one’s own feelings, to identify feelings in others and empathize, and to manage one’s own feelings toward constructive or adaptive ends. As in other areas of self-regulation, the development of ER capacities evolves from mostly external regulation (e.g., reliance on caregivers to soothe and modulate emotional response) to increasingly internal regulation (e.g., self-soothing and self-modulation of emotional expression). In most young children, external regulation of emotion can be easily seen during frequent social referencing – the tendency to look at a trusted caregiver for cues as to the appropriate emotional response. Temperament (an individual’s typical responses to environmental demands and novel stimuli and ability to regulate emotional and arousal responses to these stimuli) is a related construct.

Self-inhibitory skill (also described as effortful control) is central to the development of ER. This is the ability to behaviorally inhibit a natural emotional response in order to obtain a more socially

desirable outcome (e.g., not hitting someone when angry). Individuals who are poor at effortful control often experience difficulty with social relationships with peers, caregivers, and authority. ER, however, is not about the suppression of emotion, but rather about understanding emotions within social contexts and how to modulate emotional expression in adaptive ways. A greater capacity for ER helps one to be able to better regulate strong emotions, deal with fearful and frustrating events, and form and maintain meaningful and beneficial social relationships.

ER capacity is influenced by a person’s temperament, diagnosable disorders that may impact a person’s ability to attend to socially salient information, the degree of demands placed upon the individual by his or her environment, and executive functioning skills (e.g., attending to important environmental details, forming a symbolic understanding of the environment and how aspects of the environment interrelate, planning behavioral responses, sustaining and shifting attention, inhibiting, and self-reflecting). Cultural values also exert a strong influence by influencing caregiver responses to emotions and preferred methods for dealing with strong emotion.

Efforts to facilitate ER capacities have included teaching children to identify and label emotional states in themselves and others, to choose between a variety of potential emotional and behavioral responses, to delay gratification, and to use self-relaxation techniques.

See Also

- [Emotional Intelligence](#)
- [Empathy](#)
- [Executive Function \(EF\)](#)
- [Temperament](#)

References and Reading

- Gross, J. J. (2007). Emotion regulation. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds.), *Handbook of emotions* (3rd ed., pp. 497–512). New York: Guilford Press.

National Research Council, & Institute of Medicine. (2000). From neurons to neighborhoods: The science of early childhood development. Committee on integrating the science of early childhood development. In J. P. Shonkoff & D. A. Phillips (Eds.), *Board on children, youth, and families, commission on behavioral and social sciences and education*. Washington, DC: National Academy Press.

Emotional Synchrony

► Mutual Regulation

Emotionally Withdrawn/ Inhibited Attachment Disorder

► Attachment Disorder

Emotion-Focused Therapy for Autism Spectrum

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Definition

Emotion-Focused Therapy for Autism Spectrum (EFT-AS) is a small group version of Emotion-Focused Therapy (EFT) that provides a client-led framework within a process-guiding, task-oriented structure (Robinson and Elliott 2017). EFT-AS is a humanistic-experiential psychotherapy (HEP), which focuses on accessing and transforming core painful feelings often as a result of continued relational injuries through interpersonal encounters. EFT-AS uses the therapist relational stance, evocative process-guiding tasks, group member exchanges, and video Interpersonal Process Recall (IPR) to address core aspects of

emotion regulation, including cognitive-affective empathy to enhance intra- and interpersonal understanding.

Historical Background

Emotion-Focused Therapy (EFT) (Elliott et al. 2004; Greenberg et al. 1993) was developed as a treatment using broadly universally applicable principles to various client presentations. EFT has been used as a treatment for major depression (Greenberg and Watson 1998, 2006; Goldman et al. 2006), with clients suffering from abuse (Paivio and Greenberg 1995), trauma (Elliott et al. 1998), complex trauma (Paivio and Pascual-Leone 2010), and eating difficulties (Dolhanty and Greenberg 2007), and more recently as a treatment for social anxiety (Elliott 2013; Shahar 2014) and generalized anxiety (Timulak 2015). EFT has been further adapted to meet the specific emotional processing differences of clients on the autism spectrum (Robinson and Elliott 2017).

Rationale or Underlying Theory

Emotion-Focused Therapy (EFT) is an evidence-based humanistic-experiential psychotherapy (HEP). EFT combines relational principles from the person-centered approach (Rogers 1951) with more directive evocative interventions from Gestalt and other experiential therapies (Greenberg et al. 1993). As a humanistic therapy, it is grounded in values of authenticity, growth, self-determination, creativity, and pluralism, with the therapeutic relationship seen as a key curative element. It integrates active approaches of psychodrama (Moreno and Moreno 1959) and process-guiding methods from Gestalt therapy (Perls et al. 1951; Perls 1969) and experiential therapy, specifically Gendlin's Focusing (1981). Emotion-Focused Therapy for Autism Spectrum (EFT-AS) is an integrative group psychotherapy approach grounded in humanistic values and EFT theory and practice and uses video Interpersonal Process Recall (IPR; Kagan 1984) as a process-guiding method making experiential and self-

other relational processing more accessible and concrete.

EFT aims at helping clients to better deal with their emotions; to attend to, accept, symbolize, and make sense of them, for emotion regulation; and, ultimately, to restructure maladaptive emotion schemes that underlie client symptoms (Greenberg et al. 1993; Greenberg and Watson 2006). EFT uses several sets of concepts in its theory of function and dysfunction, including emotion schemes, emotion response types, and emotion regulation. According to EFT, emotions have an innately adaptive potential, which, if activated, can help clients change problematic emotional states or unwanted self-experiences. Therapeutic change in EFT is perceived as resulting from restructuring the cognitive-affective schemes that underlie presenting symptoms.

EFT involves changing emotion with emotion by accessing and transforming maladaptive emotions schemes (e.g., shame, fear, and lonely abandonment) underlying presenting symptoms. As clients on the autism spectrum often have difficulty accessing their emotions (Mazefsky et al. 2013) and episodic memory recall (McDonnell et al. 2017), EFT-AS uses video Interpersonal Process Recall as a clinical process-guiding method to facilitate client awareness and arousal. Specifically, to scaffold areas of difficulty related to affective empathy for self (emotional self-attunement or processing own emotions) and for other (interpersonal attunement or empathic relating) and also areas of difficulty related to cognitive empathy for self (self-understanding, reflection, and conception or theory of own mind (Williams 2010)) and for other or theory of your mind/mental representation (Baron-Cohen 1997). People on the autism spectrum are vulnerable to trauma-related experiences due to a diminished sense of self and lack of self-agency within interpersonal engagement (Robinson 2018). In EFT-AS, a main therapeutic task is repairing emotional hurt resulting from continued relational injuries through interpersonal conflict encounters. Clients on the autism spectrum work in a group setting to evoke and transform painful emotions, both directly and vicariously.

Goals and Objectives

Several goals have been addressed using Emotion-Focused Therapy for Autism Spectrum (EFT-AS). The main focus of EFT is to help clients to access maladaptive emotions and to transform these to useful adaptive emotions. Individuals on the autism spectrum can have difficulties in emotion processing, including recognizing their own and others' emotions, leading to problems in emotion regulation and interpersonal relating. The main focus in EFT-AS is working with emotional injuries to better aid emotion regulation, including cognitive-affective empathy to enhance intra- and interpersonal understanding. The therapeutic tasks evoke internal bodily focus on emotions, accessing not fully processed trauma-related experiences and compassion responses to self and to other while encoding and symbolizing new adaptive emotions.

The published research to date presents clinical case conceptualizations of clients working through different aspects of intra- and interpersonal distress. EFT-AS is marker driven and uses intra- and interpersonal markers as indicators of client readiness for engagement in therapeutic tasks. As a group therapy, it offers dynamic opportunities for clients to work on their own presenting issues, interpersonal opportunities between group members and opportunities for attachment patterns to be played out within the microcosm of the group.

One of the key presenting triggers bringing clients on the autism spectrum to therapy is interpersonal/relational issues, and these have been successfully worked with using a misempathy task (Robinson and Elliott 2017). A second presenting trigger bringing clients to therapy is social withdrawal, isolation, and loneliness. The group dynamic can reduce immediate feelings of social isolation, and it has been successfully used with clients working through emotional injuries from trauma-related experiences (Robinson 2018). A third presenting trigger bringing clients on the autism spectrum to seek therapy is feelings of extreme alienation and a sense of otherness, and this has been worked with successfully through a model of interpersonal rupture and repair (Robinson 2019).

Treatment Participants

EFT-AS is aimed at providing therapeutic support to adolescents and adults on the autism spectrum with no additional intellectual disability and complex communication needs: specifically, for people who are seeking psychological support due to emotional distress and who are struggling to understand themselves and their current and previous life experiences; for those who find others, particularly their typical development (TD) peers, difficult to understand; further, for those who are isolated and withdrawn who may have a co-occurring depression or anxiety disorder (social anxiety, generalized anxiety); and those who have experienced previous trauma-related difficulties through chronic peer victimization and bullying, social rejection, and loneliness through lack of social opportunity and friendship.

Treatment Procedures

Emotion-Focused Therapy for clients on the Autism Spectrum offers a humanistic client-led interpersonal framework within an experiential process-guiding, task-oriented structure. EFT-AS follows a 3-step model consisting of two kinds of alternating sessions: a group therapy session and a video-assisted Interpersonal Process Recall (IPR) session. It is a brief group treatment lasting a minimum of 9 weeks but can be extended in two-weekly cycles of therapy and IPR-assisted sessions, with an ending session in the final week. It is a small group format with between three and six clients and co-facilitation with two therapists. All sessions are video recorded, and the therapist analyses each therapy session (session 1, 3, 5, 7, and so on) to identify therapeutic task markers. The therapist edits three short clips that contain the task marker, which may be multiple markers for multiple members. The therapist plays these edited clips in the following IPR sessions (session 2, 4, 6, 8, and so on).

The therapist offers empathic attunement with client emotions, helping clients by focusing

attention on bodily feelings and by offering experiments in directed awareness and emotion stimulation using imagery and psycho-dramatic enactments, interpersonal exchanges, and video-IPR. Each session offers a familiar sequence, beginning with a transition task signaling the beginning of the therapy session. This is followed by a check-in which offers an opportunity to work toward what the client wants to accomplish and space to identify unfinished business from the previous session. Next, the therapist facilitates a guided focusing task aimed at helping clients focus attention to their inner bodily feelings.

As EFT is marker driven, the therapist looks for a task marker (emotion marker which is a behavior or discourse that indicates a client's readiness to engage in therapeutic work). In EFT-AS, the therapist uses these task markers in two ways. The first is in the moment to guide self- and other experiential processing tasks, and the second is through IPR clip selection to set up tasks in the following IPR session. Once the marker is identified, the therapist is guided by the emotion transformation model which provides a clinical strategy for emotional deepening that consists of several aspects within three phases. First, the therapist approaches emotions and explores client distress. At the beginning, the therapist works mostly with client global distress, that is, undifferentiated distress, to identify triggers (historical and current), negative self-treatment (negative self-talk), and avoidance behaviors (actions, thinking, and emotion). The therapist uses different emotion stimulation tasks to explore global distress and secondary emotions such as reactive anger, shame, or fear.

In the second phase, the therapist assists the client in moving toward painful and avoided emotions that have not been fully processed, staying with and deepening painful experience to reach core maladapted emotions. Once the client reaches the core emotion, the stuck painful feelings that hurt the most, the therapist helps the client to articulate the unmet need associated with that core pain.

In the third phase, the therapist uses emotion stimulation using imagery and psycho-dramatic

enactments to evoke new primary adaptive emotions and more adaptive experiences of self and others, such as self-compassion, self-soothing, and assertive anger. These are useful feelings that fit the immediate situation and provide a direction forward. The main tasks entail the use of imaginary chair dialogues such as two-chair dialogue for conflict split, two-chair dialogue for self-interruption, or empty chair dialogue for unfinished business (Elliott et al. 2004). EFT-AS uses a range of adapted therapeutic tasks to make this more accessible to the processing differences of those on the autism spectrum. The addition of video IPR can be used to scaffold retrieval of autobiographical memory recall for distanced trauma-related experiences evoking and activation of emotions associated with the event and experiential processing of these emotions. It can act as a visual concrete part of self (e.g., fragile self) for empty-chair work, to speak their truth to a significant other (e.g., a lost parent) or express their anger to a harmful other (e.g., abusive bully). Further, it can be used to evoke emotional responses to self, such as self-compassion and also to evoke interpersonal emotional responses, such as expressing and receiving compassion to and from group members. The emerging adaptive emotions facilitate mentalization of self (theory of own mind) and other (theory of your mind) that strengthens intrapersonal and interpersonal agency.

Efficacy Information

Emotion-Focused Therapy (EFT) is an empirically supported humanistic-experiential psychotherapy (HEP). Emotion-Focused Therapy in its individual form has been reported to be successfully applied for the treatment of depression (Greenberg and Watson 2006), adult survivors of childhood sexual abuse and complex trauma (Paivio and Pascual-Leone 2010), social anxiety disorder (Elliott and Shahar 2017), and generalized anxiety disorder (Timulak and McElvaney 2015). There is also growing evidence of its application with couples for interpersonal/relational problems (Johnson and Greenberg 1987) and Emotion-Focused Family Therapy (EFFT) with

patients suffering from anorexia and eating difficulties (Dolhanty and Greenberg 2007). To date, positive preliminary findings for EFT-AS have been reported (Robinson and Elliott 2016) with a number of case conceptualization from clinical data of clients working through specific presenting problems, such as misempathy (Robinson and Elliott 2017), trauma-related experiences (Robinson 2018), and relational rupture and repair (Robinson 2019).

Outcome Measurement

CEPS-AS: Client Emotional Processing Scale for Autism Spectrum (Robinson and Elliott 2016)

CORE-10: Clinical Outcomes in Routine Evaluation

QCAE: Questionnaire of Cognitive and Affective Empathy (Reniers et al. 2011)

EQ: Empathy Quotient (Baron-Cohen and Wheelwright 2004)

HAT: Helpful Aspects of Therapy Form (Llewelyn et al. 1988).

TAS-20: Toronto Alexithymia Scale (Parker et al. 2003)

OHQ: The Oxford Happiness Questionnaire (Hills and Argyle 2002)

Qualifications of Treatment Providers

EFT-AS can be provided by (a) experienced autism practitioners with a master's degree in Autism Studies and successful completion of Counseling Skills in Autism Spectrum module and (b) qualified person-centered counselors/therapists or clinical psychologists with advanced Empathy Training and successful completion of a Autism Spectrum module. This provides basic skills training in the person-centered approach and theory of autism processing differences. Additional EFT-AS training provides basic teaching on setting up and running groups, which consists of learning how to analyze and edit video recordings, how to present IPR clips, and how to recognize task markers to introduce therapeutic deepening and enactment tasks.

See Also

- [Client Emotional Processing Scale for Autism Spectrum](#)
- [Empathy](#)

References and Reading

- Baron-Cohen, S. (1997). *Mindblindness: An essay on autism and theory of mind*. Palatino: MIT Press.
- Baron-Cohen, S., & Wheelwright, S. (2004). The empathy quotient: An investigation of adults with Asperger syndrome or high functioning autism, and normal sex differences. *Journal Autism Developmental Disorder*, 34, 163–175.
- Elliott, R. (2013). Person-centered-experiential psychotherapy for anxiety difficulties: Theory, research and practice. *Person-Centered and Experiential Psychotherapies*, 12, 14–30.
- Elliott, R., Davis, K., & Slatick, E. (1998). Process-experiential therapy for post-traumatic stress difficulties. In L. Greenberg, G. Lietaer, & J. Watson (Eds.), *Handbook of experiential psychotherapy* (pp. 249–271). New York: Guilford.
- Elliott, R., & Shahar, B. (2017). Emotion-focused therapy for social anxiety (EFT-SA). Person-Centered and Experiential Psychotherapies, advance online publication. <https://doi.org/10.1080/14779757.2017.1330701>.
- Elliott, R., Watson, J. C., Goldman, R. N., & Greenberg, L. S. (2004). *Learning emotion-focused therapy: The process-experiential approach to change*. Washington, DC: APA.
- Elliott, R., Watson, J., Greenberg, L. S., Timulak, L., & Freire, E. (2013). Research on humanistic-experiential psychotherapies. In M. J. Lambert (Ed.), *Bergin & Garfield's handbook of psychotherapy and behavior change* (6th ed., pp. 495–538). New York: Wiley.
- Dolhanty, J., & Greenberg, L. (2007). Emotion-focused therapy in the treatment of eating disorders. *European Psychotherapy*, 7(1), 97–116.
- Gendlin, E. T. (1981). *Focusing* (2nd ed.). New York: Bantam Books.
- Greenberg, L. S. (2010). *Emotion-focused therapy*. Washington, DC: American Psychological Association.
- Greenberg, L. S. (2011). *Emotion focused therapy: Theories of psychotherapy series*. Washington, DC: American Psychological Association.
- Greenberg, L., Rice, L., & Elliott, R. (1993). *Facilitating emotional change: The moment by moment process*. New York: Guilford.
- Greenberg, L. S., & Watson, J. (1998). Experiential therapy of depression: Differential effects of client-centered relationship conditions and process experiential interventions. *Psychotherapy Research*, 8, 210–224.
- Greenberg, L., & Watson, J. (2006). *Emotion-focused therapy for depression*. Washington, DC: American Psychological Association.
- Goldman, R. N., Greenberg, L. S., & Angus, L. (2006). The effects of adding emotion-focused interventions to the client-centered relationship conditions in the treatment of depression. *Psychotherapy Research*, 16, 537–549.
- Hills, P., & Argyle, M. (2002). The Oxford happiness questionnaire: A compact scale for the measurement of psychological well-being. *Personality and Individual Differences*, 33, 1073–1082.
- Johnson, S. M., & Greenberg, L. (1987). Emotionally focused marital therapy: An overview. *Psychotherapy: Theory, Research, Practice, Training*, 24(3S), 552–560. <https://doi.org/10.1037/h0085753>.
- Kagan, N. (1984). Interpersonal process recall: Basic methods and recent research. In D. Larson (Ed.), *Teaching psychological skills* (pp. 229–244). Monterey: Brooks Cole.
- Llewelyn, S. P., Elliott, R., Shapiro, D. A., Firth, J., & Hardy, G. (1988). Client perceptions of significant events in prescriptive and exploratory periods of individual therapy. *British Journal of Clinical Psychology*, 27, 105–114.
- McDonnell, C. G., Valentino, K., & Diehl, J. J. (2017). A develop- mental psychopathology perspective on autobiographical memory in autism spectrum disorder. *Developmental Review*, 44, 59–81.
- Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52, 679–688.
- Moreno, J. L., & Moreno, Z. T. (1959). *Foundations of psychotherapy*. Beacon: Beacon House.
- Paivio, S. C., & Greenberg, L. S. (1995). Resolving “unfinished business”: Efficacy of experiential therapy using empty chair dialogue. *Journal of Consulting and Clinical Psychology*, 63, 419–425.
- Paivio, S. C., & Pascual-Leone, A. (2010). *Emotion-focused therapy for complex trauma: An integrative approach*. Washington, DC: American Psychological Association.
- Parker, J. D., Taylor, G. J., & Bagby, R. M. (2003). The 20-Item Toronto Alexithymia Scale. III. Reliability and factorial validity in a community population. *J Psychosom Res* 55(3):269–275. [https://doi.org/10.1016/s0022-3999\(02\)00578-0](https://doi.org/10.1016/s0022-3999(02)00578-0)
- Taylor, G. J., Bagby, R. M., & Parker, J. D. (2003). The 20-item Toronto Alexithymia Scale IV. Reliability and factorial validity in different languages and cultures. *Journal of Psychosomatic Research*, 55, 277–283. [https://doi.org/10.1016/S0022-3999\(02\)00601-3](https://doi.org/10.1016/S0022-3999(02)00601-3).
- Perls, F. S. (1969). *Gestalt therapy verbatim*. Lafayette: Real People Press.
- Perls, F. S., Hefferline, R. E., & Goodman, P. (1951). *Gestalt therapy: Excitement and growth in the human personality*. New York: Dell.
- Reniers, R. L., Corcoran, R., Drake, R., Shryane, N. M., & Völlm, B. A. (2011). The QCAE: a Questionnaire of Cognitive and Affective Empathy. *J Pers Assess* 93 (1):84–95. <https://doi.org/10.1080/00223891.2010.528484>
- Robinson, A., & Elliott, R. (2016). Brief report: An observational measure of empathy for autism spectrum:

- A preliminary study of the development and reliability of the client emotional processing scale. *Journal of Autism Developmental Disorders*, 46(6), 2240–2250. <https://doi.org/10.1007/s10803-016-2727-3>.
- Robinson, A., & Elliott, R. (2017). Emotion-focused therapy for clients with autistic process. *Person-Centered and Experiential Psychotherapies*, 16(3), 215–235. <https://doi.org/10.1080/14779757.2017.1330700>
- Robinson, A. (2018). Emotion-focused therapy for autism spectrum disorder: A case conceptualization model for trauma-related experiences. *Journal of Contemporary Psychotherapy*, 48(3), 133–143. <https://doi.org/10.1007/s10879-018-9383-1>.
- Robinson, A. (2019). Enhancing empathy in emotion-focused group therapy for adolescents with autism spectrum disorder: A case conceptualization model for interpersonal rupture and repair. *Journal of Contemporary Psychotherapy*. <https://doi.org/10.1007/s10879-019-09443-6>.
- Rogers, C. (1951). *Client-centered therapy: Its current practice, implications and theory*. London: Constable.
- Shahar, B. (2014). Emotion-focused therapy for the treatment of social anxiety: An overview of the model and a case description. *Clinical Psychology & Psychotherapy*, 21, 536–547.
- Timulak, L. (2015). *Transforming emotional pain in psychotherapy an emotion-focused approach*. London: Routledge.
- Timulak, L., & McElvaney, J. (2015). Emotion-focused therapy for generalized anxiety disorder: An overview of the model. *Journal of Contemporary Psychotherapy* 46(1). <https://doi.org/10.1007/s10879-015-9310-7>.
- Williams, D. M. (2010). Theory of own mind in autism. Evidence of a specific deficit in self-awareness? *Autism*, 14, 474–494.

Empathizing-Systemizing Theory

► Systems Intervention

Empathy

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Definition

Empathy, the capacity to share the feelings of others, is considered a significant core ability in the development of social and emotional

functioning. In general, individuals with autism experience difficulties in their capacity to empathize, thus failing to successfully engage in social interactions (Charman et al. 1997; Jones et al. 2010; Yirmiya et al. 1992).

In everyday language, as well as in the research literature, the term empathy is used synonymously for both mental and emotional states. However, cognitive empathy pertains to the capacity to represent others people's thoughts, beliefs, and intentions, whereas emotional empathy is defined as experiencing what it feels like for another person to experience a certain emotion, including a bodily sensation. Both indices tap overlapping processes and are considered interrelated aspects of the same complex construct of empathy (Singer 2006). Yet, there are quantitative differences. For example, sharing a close friend's feelings of grief is felt much differently than the recognition or comprehension of this person's thoughts and feelings in this painful situation. Understanding of these differences has led to the establishment of a more multidimensional approach to the definition of empathy, acknowledging that all these aspects are separate but related constructs of the integral concept of empathy (Eisenberg et al. 2006).

Sharing feelings with others may be manifested in facial and/or vocal expressions or in body movements and gestures. People can feel empathy for others in a range of emotional contexts, from basic sensations such as sadness or joy to more complex emotions such as love and remorse. Empathy has a variety of definitions such as the ability to place oneself in another's place ("put oneself in another's shoes"), the experiencing of another's affective or psychological states, or one's emotional responses to the emotional displays of others (Eisenberg and Fabes 1990; Moore 1990; Zahn-Waxler et al. 1992). Additional definitions describe empathy as an affective response more appropriate to someone else's situation than to one's own situation, or when another person's emotion evokes an analogous affective experience in oneself (Eisenberg et al. 2006). It is important to note that although in empathizing one may "feel the same" as another, the affective states of the self and other are nevertheless distinguishable. That

is, the emotion that was induced in the self by the perception of the other's emotional state can be distinguished from the same feeling originating in oneself. A related concept to empathy is sympathy, which is similarly described as an emotional response of sorrow or concern toward someone in distress. Sympathy usually stems from empathy, yet both empathy and sympathy differ from personal distress, which is a self-focused and somewhat aversive reaction of anxiety in response to a similar emotional state of distress in another (Eisenberg et al.).

The concept of empathy plays a significant role in developmental social cognition theories such as theory of mind (ToM) and the empathizing-systemizing theory and is associated with concepts such as mind reading, mentalizing, and perspective taking (Baron-Cohen 1995, 2002; Frith 1994). These theories suggest that the characteristic deficits in mentalizing skills that are associated with poor interpersonal relationships hinder the ability of individuals with autism to understand and engage in interpersonal relationships. In order to engage in reciprocal social interactions and to successfully navigate social exchanges, one should possess the capacity to recognize and accurately interpret and predict what the other is thinking and feeling and how he or she might respond.

Historical Background

In their initial writings, Leo Kanner as well as Hans Asperger described autism as affective impairment pertaining to difficulties in social and communicative functions as well as to difficulties in empathy. Kanner described "children's inability to relate themselves in the ordinary way to people and situations from the beginning of life." He noted that "It is not a 'withdrawal' from formerly existing participation. There is from the start an *extreme autistic aloneness* that whenever possible, disregards, ignores, shuts out anything that comes to the child from the outside" (Asperger 1944; Kanner 1943; Wing 1981). In his description, Asperger specifically addressed "autistic psychopathy" of children who manifested "severely disturbed and considerably

limited interaction. . . . and characteristic difficulties of social integration" including patterns of lack of empathy and difficulties in "reading between the lines."

Empathy is long considered an important process known to facilitate interpersonal relationships, particularly prosocial behavior. Religious, philosophical, and psychological theories have long debated whether humans are basically good, empathic, and altruistic in nature or whether all prosocial actions essentially derive from underlying egoism and self-interest. Twentieth century psychological theories such as Freud's psychoanalytic theory viewed human development mainly as an interplay between two innate trends, "egoistic" and "altruistic," although later psychoanalytic theories acknowledged prosocial actions as defense mechanisms used to deal with irrational demands such as feelings of guilt, self-destructive tendencies, and sexual strivings – all believed to underlie altruism and empathy. These theories also put forward the importance of the early mother-child relationship for the development of empathy, through mental mechanisms of identification and internalization. Furthermore, contemporary psychoanalytic theories consider empathy as crucial to the development of insight, for people may come to see themselves through eyes of others via empathy. Empathy is also considered an important condition for effective therapeutic intervention in psychotherapy. Piaget's theory on cognitive development emphasized the transition from infants' egocentric orientation to the more social orientations characteristic of later periods, through the acquisition of perspective-taking abilities during the preschool years; thus, prosocial behaviors were assumed to emerge at the beginning of the school years (Eisenberg et al. 2006).

Current Knowledge

Empathy in Individuals with Autism

A considerable body of research investigated difficulties in empathy among individuals with autism, who are characterized by qualitative impairments in social interaction and communication (Baron-Cohen and Wheelwright 2004;

Charman et al. 1997; Jones et al. 2010; Kasari et al. 2001; Rogers et al. 2007; Shamay-Tsoory et al. 2002; Yirmiya et al. 1992). It was suggested that individuals with autism exhibit deficits in their capacity to mentalize and empathize and that these core deficits hinder them in developing adequate reciprocal interactions. Individuals with autism reveal difficulties in interpreting social cues and in “mind reading” of others’ emotions; hence, it is more difficult for them to achieve a clear and accurate understanding of what other individuals think, feel, and experience.

Clinical observations suggested that children with autism do not respond to other people’s affect in the same way as children with typical development or children with mental retardation (Sigman and Capps 1997). The empathic response to negative affect (distress of other) was investigated in children with autism in comparison to children with typical development and children with mental retardation, matched on mental age (Kasari et al. 1993). It was found that more children with autism ignore, or did not notice, the negative affect of the adults and appeared less concerned than the other children in response to distressed adult. It was suggested that children with autism lack the cognitive and affective prerequisites that are necessary for being attentive and empathic to the emotion of others. Other studies investigated difficulties in the capacity to empathize in high-functioning children with autism and adolescents compared to typically developing children at the same age (Yirmiya et al. 1992). Children’s ability to discriminate between different emotional states, perspective-taking abilities of emotional states, and emotional responsiveness were compared. The strong associations between cognitive abilities and empathy that was found in the autism group compared to the typical development group may have suggested that children with autism use their cognitive abilities and cognitive strategies to successfully understand and engage in social situations more than is necessary for children with typical development (Sigman and Capps 1997).

Individuals with autism present difficulties in understanding the feelings and perspectives of

others, labeling the emotions felt by others, and responding with empathy. They also react unexpectedly to interpersonal situations. It is important to note that although individuals with autism manifest difficulties in empathy, this does not mean that their behavior in real-life situations is cold or uncaring or that their lack of empathy is related to antisocial behavior (Jones et al. 2010; Rogers et al. 2007). Many individuals with autism appear to show as much warmth, compassion, caring, and concern for others as typically developing individuals, especially when the information is presented in a way that allows them to better understand the feelings and perspectives of others. An alternative explanation for the lower empathy levels reported in autism than in typical development may be that these research findings could reflect difficulties in articulating emotions rather than in feeling them. Evidence for this possibility may be that individuals with autism who exhibit greater self-awareness as well as higher cognitive and verbal abilities are able to report their feelings more accurately and thus to appear more empathic than individuals with autism with lower cognitive and verbal abilities who exhibit lower levels of self-awareness and who show difficulties articulating their emotions (Travis et al. 2001; Yirmiya et al. 1992). Furthermore, comparing boys with autism and boys with psychopathic tendencies revealed that the latter had significant impairments in emotional empathy with intact perspective-taking abilities, whereas the boys with autism had a reverse pattern – significant impairments in cognitive perspective-taking abilities and intact emotional empathy (Jones et al. 2010).

Recently, Simon Baron-Cohen introduced the extreme male brain (EMB) theory of autism (Baron-Cohen 2002), suggesting that individuals with autism may manifest an exaggerated male brain psychometric profile, demonstrating extreme properties in two psychological dimensions: extremely low empathizing (defined as the drive to identify another’s mental state and to respond with an appropriate emotion) and extremely high systemizing (defined as mechanistic thinking; the drive to analyze, explore, and construct a system in terms of its underlying regularities).

The Development of Empathy

It is well known that infants and toddlers are sensitive and responsive to emotional cues or signals of others, thus yielding behaviors that resemble empathic expressiveness. An infant's reflexive crying or arousal-containing sucking behavior in response to the crying of another infant are considered early precursors of empathic reactivity (Ungerer et al. 1990; Zahn-Waxler et al. 1992). This emotional contagion, or emotional resonance, refers to the infant's ability to imitate another's emotional distress, without awareness of the other, and therefore is not considered "true" empathy. This kind of emotional involvement, which is strongly linked to infants' early developmental and cognitive abilities, diminishes after the first year as differentiation between self and other increases and toddlers come to perceive themselves as separate beings. Thus, empathy emerges throughout the second year of life, as well as sympathetic concern and comforting behaviors in response to others' distress (Eisenberg and Fabes 1990; Zahn-Waxler and Radkeyarrow 1990; Zahn-Waxler et al. 1992).

Longitudinal studies revealed that empathic behaviors increase and become more sophisticated and explicit during the first years, and this increase is associated with the developmental changes that are prerequisites for empathy. These cognitive and emotional prerequisites are self-other differentiation, perspective-taking ability, and emotion regulation, as well linguistic ability and social engagement skills. All these skills are necessary for the child to empathize, comfort others, inquire about others' feelings, and provide others with adequate support (Knafo et al. 2008; Zahn-Waxler et al. 1992). Furthermore, secure attachment relationships and early parent-infant interactions contribute to later development of empathy, through the synchronized exchange of shared emotions ("the dance") between the parents and infant in the first year. In the second year, emotion regulation pertains to the capacity for role-taking and the emergence of higher-order self-conscious emotions such as guilt or shame as well as empathy. Socialization and environmental influences also play an important role in the second year, when parents already expect their

children's behavior to be socially and interpersonally appropriate (Eisenberg et al. 2006).

Although the observation of early empathic manifestations in infants and toddlers is not straightforward due to children's limited verbal skills, some researchers demonstrated the presence of different early manifestations of empathic behaviors (Zahn-Waxler and Radkeyarrow 1990; Zahn-Waxler et al. 1992). For example, toddlers older than 1 year who observed a peer or adult in distress revealed varying behaviors: Some toddlers became quiet and observed the situation with a concerned facial expression; others expressed their discomfort by disquiet and agitated behaviors; and others ignored the distressed person and continued with their own activities. The earliest empathic reactions are mostly physical in nature, such as hugs and pats. After age 2 years, toddlers reveal more explicit empathic and prosocial behaviors such as verbally comforting, reaching for someone to get help, giving a victim toys, or trying to find ways to cheer someone up. Some of these behaviors may be still egocentric in nature; young children may try to comfort the other with things that make them feel better. With age, as ToM abilities become more developed (regarding the differentiation between one's own and others' states of mind) and as affective arousal becomes more modulated, children's empathic and prosocial behaviors become more attuned to the other person's needs (Eisenberg et al. 2006). There is some evidence regarding sex differences in empathy in the preschool years as well as in adulthood. Girls usually reveal more empathic reactions than boys, such as concern and joining into the emotional experiences of others, and these sex differences are present as early as 14 months. It may be assumed that these differences reflect both biologically based predispositions as well as socialization influences for females to be responsive to the physical and emotional needs of others (Eisenberg et al.).

Not many studies have investigated children with autism in terms of the emergence of empathy in the first years and the early manifestations of sympathetic concern and comforting behaviors in response to others' distress (Yirmiya et al. 1992),

as well as developmental trajectories for the ability to empathize in children with autism. Twenty-month-old toddlers with autism had difficulties looking to other person's face and showing expressed facial concern in response to feigned distress compared to control groups of 20-month-old toddlers with developmental delays and typical development (Charman et al. 1997). In a recent prospective study of young siblings of children with autism – a group considered at higher risk for autism – infants later diagnosed with autism paid less attention and showed less affective response to the examiner's display of a distress reaction as early as 12 months of age compared to infants who were not later diagnosed with autism (Hutman et al. 2010). Furthermore, it was found that atypical response to another person's distress differentiated infants subsequently diagnosed with autism from those infants who were not later diagnosed with autism spectrum disorder at 36 months. Thus, it was suggested that low levels of responsiveness to another person's distress at 12 and 18 months are indicative of elevated risk for autism spectrum disorder at 36 months. These findings need to be replicated in other low-risk and high-risk infant populations.

Measuring Empathy

Several research instruments have been used to measure empathy in children and adults in general and in individuals with autism. The Feshbach and Powell Audiovisual Test for Empathy (Feshbach 1982) was designed to measure children's empathy and emotional responsiveness. It consists of 10 videotaped segments presenting short stories about children experiencing different events and emotions such as happiness, anger, or sadness (e.g., a boy is sad because he lost his dog). After watching each story, the child is asked to report how he or she feels. In some adaptations of this procedure (Yirmiya et al. 1992), usually when examining children with autism, participants are first asked about the nature of the protagonists' feelings (labeling the emotion) in addition to the nature of their own emotional response to the video vignette (empathic reaction). This adaptation prevents the examiner from identifying children as failing to empathize in cases when the

child recognizes and reports a different emotion from the target emotion yet reveals the capacity to refer to the protagonist's emotional state.

Marian Sigman and her colleagues designed specific research procedures for measuring empathy among young children with autism (Sigman et al. 1992). The researchers recorded and then coded the child's reactions to a distress situation that was enacted by the mother. In this enactment, after a short period of play with a wooden pounding toy and hammer, the mother pretended to hurt herself by hitting her finger with the hammer, and she displayed facial and vocal expressions of distress without using words. Children with autism tended to ignore or not notice adults showing these negative affects more often than children with mental retardation or children with typical development.

Carolyn Zahn-Waxler and her colleagues (Zahn-Waxler et al. 1992), who investigated the early development of empathy in children and twins, used similar procedures where both an experimenter and the mother presented simulated situations of distress such as bumping into a chair. In addition, they used mothers as systematic observers, training them to collect data regarding their child's responses to the emotions of others, emotions both caused and witnessed by their children. This methodology may be of interest to investigators to employ in future studies of empathy in children with autism.

Children's limited verbal ability precludes the use of self-report measures, which are more common in the examination of empathy in adolescents and adults. The Interpersonal Reactivity Index (IRI; Davis 1983) is a self-report measure of empathy comprising four subscales: perspective-taking, empathic concern, personal distress, and fantasy. Although some of these subscales may not directly assess emotional empathy, indices such as imagination or emotional self-control correlate with empathy. A more recent self-report instrument for measuring empathy is the 60-item Empathy Quotient questionnaire (EQ; Baron-Cohen and Wheelwright 2004). This scale includes items like: "It is hard for me to see why some things upset people so much" or "I find it easy to put myself in somebody else's shoes" and

thus may be measuring prosocial behaviors and social skills in addition to empathy. Adults with Asperger's syndrome or high-functioning autism scored significantly lower on the EQ than individuals with typical development matched for age and sex; in addition, in the typical development group, women score significantly higher than men. Assessing individuals' beliefs about their own empathy might differ from their actual empathic reactions to emotional situations. Indeed, it is an important methodology strength of developmental research over the report measures used with older individuals.

Current Knowledge

Recent longitudinal and twin studies have identified continuity in empathic development over time and the relationship between empathy and prosocial behavior (Knafo et al. 2008). It was suggested that empathy is a relatively stable disposition across both time and different contexts, with heritability that tends to increase with age. Yet, environmental factors such as parenting practices, peer and sibling relationships, and educational programs contribute to empathic abilities (Knafo and Uzefovsky 2012). Several biological and genetic mechanisms were proposed as relevant to the development of empathy. Empathy was found to be associated with temperamental characteristics: For example, inhibited and unreactive temperament features in the first year were negatively associated with empathy at the age of 2 years (Young et al. 1999). Other studies have focused on the neuronal correlates and neural mechanisms underlying the ability to understand the mental and emotional states of others (Blair 1999; Singer 2006). Different neural circuitries were associated with the cognitive and emotional aspects of empathy: Perspective-taking abilities were found to involve structures of the superior temporal sulcus and the medial prefrontal cortex, whereas emotional empathy involved sensory-motor cortices as well as limbic and paralimbic structures (the "social" or "emotional" brain). Moreover, it was found that the limbic structures develop earlier than the temporal lobe and prefrontal structures – in line with developmental studies that demonstrated the early

precursors of emotional empathy during the first year as well as the later emergence of cognitive perspective-taking abilities (Decety 2010; Singer 2006).

Some speculations were raised about the associations between empathy and autism and the network of visuomotor cells known as mirror neurons (MNs). The MNs are activated and discharged when the individual performs a particular motor action as well as when observing somebody else perform a similar action. Thus, it was suggested that the MNs as an execution/observation matching system may be connected to social and emotional functioning such as imitation and empathy, which are considered as impaired in autism (Perkins et al. 2010). Finally, molecular genetics studies have demonstrated the associations between social behavior and two non-a peptides of arginine: vasopressin and oxytocin (Israel et al. 2008; Yirmiya et al. 2006). It was suggested that the arginine vasopressin receptor 1a (AVPR1a) gene and the oxytocin receptor (OXTR) gene are possible contributors to altruistic behavior. Furthermore, family studies of individuals with autism and their relatives revealed the role of the AVPR1a gene in mediating the association of socialization skills in autism. Other genetic studies examined candidate genes in a group of individuals with Asperger's syndrome and a population sample. Nineteen genes showed nominally significant associations with self-report measures of empathy (Empathy Quotient questionnaire) and autistic traits (the Autism Quotient questionnaire) (Chakrabarti et al. 2009).

Future Directions

Although difficulties in empathy are detectable among individuals with autism, not much is yet known about the developmental trajectories of empathy in infants or toddlers who are later diagnosed with autism (Hutman et al. 2010). The search for empathy precursors in autism is closely linked to the investigation of early manifestations of autism in the first year/s of life. Future research would do well to track infants who are underresponsive to emotional cues such as others'

distress and to explore these infants' developmental trajectories and later diagnoses. It will be of great importance to know whether early underresponsiveness during the first year predicts future diagnoses of autism or other developmental difficulties. Furthermore, within the field of autism, treatment studies should be initiated to explore strategies to improve the capacity to empathize and thus to facilitate the various aspects of social interactions and relationships.

See Also

- ▶ Affective Development
- ▶ Client Emotional Processing Scale for Autism Spectrum
- ▶ Empathizing-Systemizing Theory
- ▶ Extreme Male Brain (EMB) Theory
- ▶ Face Perception
- ▶ Face Recognition
- ▶ Friendships
- ▶ Interpersonal Skills
- ▶ Mindblindness
- ▶ Social Cognition

References and Reading

- Asperger, H. (1944). Die "Autistischen Psychopathen" im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Baron-Cohen, S. (1995). *Mind blindness: An essay on autism and theory of mind*. Cambridge: MIT Press.
- Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences*, 6(6), 248–254.
- Baron-Cohen, S., & Wheelwright, S. (2004). The empathy quotient: An investigation of adults with Asperger syndrome or high functioning autism, and normal sex differences. *Journal of Autism and Developmental Disorders*, 34(2), 163–175.
- Blair, R. J. R. (1999). Psychophysiological responsiveness to the distress of others in children with autism. *Personality and Individual Differences*, 26(3), 477–485.
- Chakrabarti, B., Dudbridge, F., Kent, L., Wheelwright, S., Hill-Cawthorne, G., Allison, C., et al. (2009). Genes related to sex steroids, neural growth, and social-emotional behavior are associated with autistic traits, empathy, and Asperger syndrome. *Autism Research*, 2(3), 157–177.
- Charman, T., Swettenham, J., Baron-Cohen, S., Cox, A., Baird, G., & Drew, A. (1997). Infants with autism: An investigation of empathy, pretend play, joint attention, and imitation. *Developmental Psychology*, 33(5), 781–789.
- Davis, M. H. (1983). Measuring individual differences in empathy: evidence for a multidimensional approach. *Journal of Personality and Social Psychology*, 44, 113–126.
- Decety, J. (2010). The neurodevelopment of empathy in humans. *Developmental Neuroscience*, 32(4), 257–267.
- Eisenberg, N., & Fabes, R. A. (1990). Empathy: Conceptualization, measurement, and relation to prosocial behavior. *Motivation and Emotion*, 14(2), 131–149.
- Eisenberg, N., Fabes, R. A., & Spinrad, T. L. (2006). Prosocial development. In W. Damon, R. M. Lerner, & N. Eisenberg (Eds.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 646–718). New York: Wiley.
- Feshbach, N. D. (1982). Sex differences in empathy and social behavior in children. In N. Eisenberg (Ed.), *The development of prosocial behavior* (pp. 315–338). New York: Academic Press.
- Frith, U. (1994). Autism and theory of mind in everyday life. *Social Development*, 3(2), 108–124.
- Hutman, T., Rozga, A., DeLaurentis, A. D., Barnwell, J. M., Sugar, C. A., & Sigman, M. (2010). Response to distress in infants at risk for autism: A prospective longitudinal study. *Journal of Child Psychology and Psychiatry*, 51(9), 1010–1020.
- Israel, S., Lerer, E., Shalev, I., Uzefovsky, F., Reibold, M., Bachner-Melman, R., et al. (2008). Molecular genetic studies of the arginine vasopressin 1a receptor (AVPR1a) and the oxytocin receptor (OXTR) in human behaviour: From autism to altruism with some notes in between. *Progress in Brain Research*, 170, 435–449.
- Jones, A. P., Happe, F. G. E., Gilbert, F., Burnett, S., & Viding, E. (2010). Feeling, caring, knowing: Different types of empathy deficit in boys with psychopathic tendencies and autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 51(11), 1188–1197.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kasari, C., Sigman, M., & Yirmiya, N. (1993). Focused and social attention of autistic-children in interactions with familiar and unfamiliar adults – A comparison of autistic, mentally-retarded, and normal-children. *Development and Psychopathology*, 5(3), 403–414.
- Kasari, C., Chamberlain, B., & Bauminger, N. (2001). Social emotions and social relationships in autism: Can children with autism compensate? In J. Burack, T. Charman, N. Yirmiya, & P. Zelazo (Eds.), *Perspectives on development in autism* (pp. 309–323). Hillsdale: Erlbaum.
- Knafo, A., & Uzefovsky, F. (2012). Variation in empathy: The interplay of genetic and environmental factors. In M. Legerstee, D. W. Haley, & M. H. Bornstein (Eds.),

- The developing infant mind: Integrating biology and experience.* Guilford Press.
- Knafo, A., Zahn-Waxler, C., Van Hulle, C., Robinson, J. L., & Rhee, S. H. (2008). The developmental origins of a disposition toward empathy: Genetic and environmental contributions. *Emotion, 8*(6), 737–752.
- Moore, B. S. (1990). The origins and development of empathy. *Motivation and Emotion, 14*(2), 75–80.
- Perkins, T., Stokes, M., McGillivray, J., & Bittar, R. (2010). Mirror neuron dysfunction in autism spectrum disorders. *Journal of Clinical Neuroscience, 17*(10), 1239–1243.
- Rogers, K., Dziobek, I., Hassenstab, J., Wolf, O. T., & Convit, A. (2007). Who cares? Revisiting empathy in Asperger syndrome. *Journal of Autism and Developmental Disorders, 37*(4), 709–715.
- Shamay-Tsoory, S. G., Tomer, R., Yaniv, S., & Aharon-Peretz, J. (2002). Empathy deficits in Asperger syndrome: A cognitive profile. *Neurocase, 8*(3), 245–252.
- Sigman, M., & Capps, L. (1997). *Children with autism: A developmental perspective (Development of social and emotional understanding)* (pp. 34–60). Cambridge, MA: Harvard University Press.
- Sigman, M. D., Kasari, C., Kwon, J. H., & Yirmiya, N. (1992). Responses to the negative emotions of others by autistic, mentally-retarded, and normal-children. *Child Development, 63*(4), 796–807.
- Singer, T. (2006). The neuronal basis and ontogeny of empathy and mind reading: Review of literature and implications for future research. *Neuroscience and Biobehavioral Reviews, 30*(6), 855–863.
- Travis, L., Sigman, M., & Ruskin, E. (2001). Links between social understanding and social behavior in verbally able children with autism. *Journal of Autism and Developmental Disorders, 31*(2), 119–130.
- Ungerer, J. A., Dolby, R., Waters, B., Barnett, B., Kelk, N., & Lewin, V. (1990). The early development of empathy: Self-regulation and individual differences in the first year. *Motivation and Emotion, 14*(2), 93–106.
- Wing, L. (1981). Asperger syndrome: A clinical account. *Psychological Medicine, 11*, 115–130.
- Yirmiya, N., Sigman, M. D., Kasari, C., & Mundy, P. (1992). Empathy and cognition in high-functioning children with autism. *Child Development, 63*(1), 150–160.
- Yirmiya, N., Rosenberg, C., Levi, S., Salomon, S., Shulman, C., Nemanov, L., et al. (2006). Association between the arginine vasopressin 1a receptor (AVPR1a) gene and autism in a family-based study: Mediation by socialization skills. *Molecular Psychiatry, 11*(5), 488–494.
- Young, S. K., Fox, N. A., & Zahn-Waxler, C. (1999). The relations between temperament and empathy in 2-year-olds. *Developmental Psychology, 35*(5), 1189–1197.
- Zahn-Waxler, C., & Radkeyarrow, M. (1990). The origins of empathic concern. *Motivation and Emotion, 14*(2), 107–130.
- Zahn-Waxler, C., Radkeyarrow, M., Wagner, E., & Chapman, M. (1992). Development of concern for others. *Developmental Psychology, 28*(1), 126–136.

Empirically Supported Treatments

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Definition

Evidence-based practice (EBP) refers to a multi-step process used to make decisions that involves the appraisal of current evidence, reference to clinical expertise, and consideration of patient values and choice (Sackett et al. 1996).

Historical Background

The conceptualization, ideals, and guidelines that have become evidence-based practice (EBP) emerged first in the medical field as evidence-based medicine. EBP is a multistep process that involves the appraisal of current evidence, reference to clinical expertise, and consideration of patient values and choice (Sackett et al. 1996). Since moving to the social sciences, EBP, albeit under various names (e.g., empirically supported treatment, scientifically based research), has been further refined and expanded to match the ideologies and traditions of the various disciplines within the social sciences. Most organizations representing individuals practicing in the social sciences now have a definition of EBP, although the amount and type of evidence needed for a practice to be recognized as evidence-based often differ across organizations. Given the

multitude of disciplines working in the field of autism, this likely hindered early efforts at identifying EBP.

Current Knowledge

Recently, a number of practices and treatments for individuals with ASDs have been identified EBP. Two recent books (e.g., *Evidence-Based Practices and Treatments for Children with Autism* (Reichow et al. 2011) and *Effective Practices for Children with Autism* (Luiselli et al. 2008)) have shown that many autism treatments can now be considered evidence-based (see also *Educating Children with Autism*; National Research Council 2001). Evidence of EBP in autism in peer-reviewed publications has also emerged. In 2000, Filipek and colleagues applied the American Academy of Neurology standards to screening and diagnostic procedures, and Rogers and Vismara (2008) outlined the evidence for autism treatments for the 10-year follow-up of APA Division 53 assessment of EBP. The new report suggested early intensive behavioral intervention based on applied behavior analysis, pivotal response treatment, and parent education could now be considered evidence-based. Shortly thereafter, two independent comprehensive reviews by the National Professional Development Center on ASD (2008) and the National Autism Center (2009) identified dozens of focal treatments that are evidence-based, including, but not limited to, visual schedules, social narratives, prompting, and reinforcement (complete lists available at websites listed below). Updates to both of these guides is currently underway which should further our knowledge about the most effective treatments for children with ASD. Government-led health agencies (e.g., Agency for Healthcare Research and Quality 2011; National Institute for Health and Clinical Excellence 2011; Scottish Intercollegiate Guidelines Network 2007) have also issued guidelines, often with accompanying parent-friendly guides, thus helping ensure consumers are able to access this critical information necessary to be an active participant in the EBP process.

Future Directions

The amount of research on ASD continues to expand. Thereby, continued identification of EBP for individuals with ASDs will be needed. Currently, little is known a priori concerning which EBP will be the most successful treatment for specific individuals. Future EBP guides should strive to help identify the individual characteristics of the client, therapist, and/or setting that will help maximize outcomes. Given the research to practice gap, further dissemination of EBPs are also needed, especially in formats that are accessible to practitioners and parents. As society continues to expand its use and emphasis of technology, it will also be important to begin to understand how the World Wide Web and social media (e.g., Facebook, Twitter) might be utilized to disseminate EBP to parents and practitioners. Research has shown that parents utilize the World Wide Web more than any other source to gain information on ASD, but recent analyses of the quality of the information on autism websites suggest caution and vigilance are needed as consumers are likely to access sites with mixed or poor quality (Reichow et al. 2012).

See Also

- Practice Guidelines in Autism
- Treatment Integrity

References and Reading

- Agency for Healthcare Research and Quality. (2011). *Therapies for children with autism spectrum disorders (Comparative effectiveness review number 26)*. Rockville: Author.
- Filipek, P. A., Accardo, P. J., Ashwal, S., Baranek, G. T., Cook, E. H., Jr., Dawson, G., et al. (2000). Practice parameter: Screening and diagnosis of autism. *Neurology*, 55, 468–479.
- Luiselli, J. K., Russo, D. C., Christian, W. P., & Wilczynski, S. M. (2008). *Effective practices for children with autism: Educational and behavior support interventions that work*. New York: Oxford University Press.
- National Autism Center. (2009). *National standards report*. Retrieved January 6, 2010, from <http://www.nacautismcenter.org/standards-report>

nationalautismcenter.org/pdf/NAC%20Standards%20Report.pdf

- National Institute for Health and Clinical Excellence. (2011). *Autism: Recognition, referral and diagnosis of children and young people on the autism spectrum (CG128)*. London: Author.
- National Research Council. (2001). *Educating young children with autism*. Washington, DC: National Academy Press.
- Reichow, B., Doehring, P., Cicchetti, D. V., & Volkmar, F. R. (Eds.). (2011). *Evidence-based practices and treatments for children with autism*. New York: Springer.
- Reichow, B., Halpern, J., Steinhoff, T., Letsinger, N., Naples, A., & Volkmar, F. R. (2012). Characteristics and quality of autism websites. *Journal of Autism and Developmental Disorders*, 42(6), 1263–1274.
- Rogers, S. J. (1998). Empirically supported comprehensive treatments for young children with autism. *Journal of Clinical Child Psychology*, 27, 168–179.
- Rogers, S. J., & Vismara, L. A. (2008). Evidence-based comprehensive treatments for early autism. *Journal of Clinical Child and Adolescent Psychology*, 37, 8–38.
- Sackett, D. L., Rosenberg, W. M. C., Gray, J. A. M., Haynes, R. B., & Richardson, W. S. (1996). Evidence based medicine: What it is and what it isn't. *British Medical Journal*, 312, 71–72.
- Scottish Intercollegiate Guidelines Network. (2007). *Assessment, diagnosis and clinical interventions for children and young people with autism spectrum disorders: A national clinical guideline*. Retrieved January 5, 2010, from <http://www.sign.ac.uk/pdf/sign98.pdf>

Employ

- [Employment](#)

Employee Development

- [Feedback on Provider Work Performance](#)

Employee Performance

- [Feedback on Provider Work Performance](#)

Employment

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Synonyms

[Employ](#); [Pay](#); [Service](#); [Working for pay](#)

Definition

An agreement between a person and a representative of a business who has hiring authority (i.e., owner, manager, personnel director, etc.) to exchange labor for a wage that is in alignment with others who are performing comparable work in the workplace and at least meets the federal minimum wage standard.

An arrangement where a person engages in providing a service and/or producing a product for another entity (person, the public, business, etc.) in exchange for monetary payment to earn a livable income.

See Also

- [Supported Employment](#)
- [Transition Planning](#)
- [Vocational Rehabilitation Act of 1973](#)

References and Reading

- Inge, K., & Moon, S. (2011). Preparing students with low incidence disabilities to work in the community. In J. M. Kauffman & D. P. Hallahan (Eds.), *Handbook of special education*. New York/London: Routledge.
- Wehman, P. (2012). *Life beyond the classroom: Transition strategies for young people with disabilities*. Baltimore: Paul H. Brookes.
- Wehman, P., Smith, M., & Schall, C. (2009). *Transition from school to adulthood for youth and young adults with autism: Growing up in the real world*. Baltimore: Paul Brookes.

Employment Consultant

► Employment Specialist

Employment in Adult Life

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Definition

There is limited research outlining the specific services and supports required by individuals with ASD. However, some examples of a supported employment approach have been described (e.g., Hillier et al. 2007a, b; Howlin et al. 2005; Keel et al. 1997; Lawer et al. 2009; Nesbitt 2000; Smith 1994; Schaller and Yang 2005; Wehman et al. 2009). Using this advocacy level service, a vocational rehabilitation professional known as an employment specialist or job coach provides and/or facilitates a unique mix of supports that vary in type, level, and intensity uniquely designed to assist the person with gaining and maintaining employment (Wehman et al. 2009).

Additionally, case studies conducted with adults with ASD provide tremendous insight regarding vocational needs (Hurlbutt and Chalmers 2002, 2004; Müller et al. 2003; Wehman et al. 2009). From this research, recommendations for vocational supports can be ascertained and grouped into four major themes: (1) job placement (Schaller and Yang 2005) and match (Howlin et al. 2005; Keel et al. 1997; Mawhood and Howlin 1999; Müller et al. 2003), (2) supervisors and coworkers (Bolman 2008; Burt et al. 1991; Hagner and Cooney 2005; Hillier et al. 2007a, b; Howlin et al. 2005; Keel et al. 1997; Mawhood and Howlin 1999; Müller et al. 2003; Nesbitt 2000), (3) on-the-job provisions (Burt et al. 1991; Fast 2004; Hagner and Cooney

2005; Hillier et al. 2007a, b; Lawer et al. 2009; Nuehring and Sitlington 2003; Smith et al. 1995), (4) work place modifications (Fast 2004; Hagner and Cooney 2005; Müller et al. 2003; Nuehring and Sitlington 2003) and predictability (Burt et al. 1991; Foley and Staples 2003; Hagner and Cooney 2005; Hume and Odom 2007), and (5) long-term support (Hillier et al. 2007a, b; Howlin et al. 2005; Keel et al. 1997; Müller et al. 2003; Smith and Philippen 1999; Smith et al. 1995).

While the small body of existing research gives shape to this topic, there are limitations. The number of peer-reviewed articles on this topic is scarce and hampered by limited sample sizes and restricted population ranges. Existing research covers an array of topics, yielding few solid conclusions in any given area. However, to some degree, the evidence does seem to endorse supported employment as a promising practice to assist individuals with ASD with employment and career advancement. If this supported employment is going to become a viable option for individuals with ASD, then key stakeholders including individuals with ASD, family members, educators, vocational rehabilitation service providers, and others will need to learn how to individualize an array of specific supports at work designed to meet each person's unique needs.

It will also require education of employers to understand how employing an individual with ASD can bring value to business. In summary, to date, few solid conclusions have been reached, and more research is needed.

Historical Background

There is limited research outlining the specific services and supports required by individuals with ASD. However, some examples of a supported employment approach have been described (e.g., Hillier et al. 2007a, b; Howlin et al. 2005; Keel et al. 1997; Lawer et al. 2009; Nesbitt 2000; Smith 1994; Schaller and Yang 2005; Wehman et al. 2009). Using this advocacy level service, a vocational rehabilitation professional known as an employment specialist or job

coach provides and/or facilitates a unique mix of supports that vary in type, level, and intensity uniquely designed to assist the person with gaining and maintaining employment (Wehman et al. 2009).

Additionally, case studies conducted with adults with ASD provide tremendous insight regarding vocational needs (Hurlbutt and Chalmers 2002, 2004; Müller et al. 2003; Wehman et al. 2009). From this research, recommendations for vocational supports can be ascertained and grouped into major themes: (1) job placement (Schaller and Yang 2005) and match (Howlin et al. 2005; Keel et al. 1997; Mawhood and Howlin 1999; Müller et al. 2003), (2) supervisors and coworkers (Bolman 2008; Burt et al. 1991; Hagner and Cooney 2005; Hillier et al. 2007a, b; Howlin et al. 2005; Keel et al. 1997; Mawhood and Howlin 1999; Müller et al. 2003; Nesbitt 2000), (3) on-the-job provisions (Burt et al. 1991; Fast 2004; Hagner and Cooney 2005; Hillier et al. 2007a, b; Lawer et al. 2009; Nuehring and Sitlington 2003; Smith et al. 1995), (4) work place modifications (Fast 2004; Hagner and Cooney 2005; Müller et al. 2003; Nuehring and Sitlington 2003) and predictability (Burt et al. 1991; Foley and Staples 2003; Hagner and Cooney 2005; Hume and Odom 2007), and (5) long-term support (Hillier et al. 2007a, b; Howlin et al. 2005; Keel et al. 1997; Müller et al. 2003; Smith and Philippen 1999; Smith et al. 1995).

While the small body of existing research gives shape to this topic, there are limitations. The number of peer-reviewed articles on this topic is scarce and hampered by limited sample sizes and restricted population ranges. Existing research covers an array of topics, yielding few solid conclusions in any given area. However, to some degree, the evidence does seem to endorse supported employment as a promising practice to assist individuals with ASD with employment and career advancement. If this supported employment is going to become a viable option for individuals with ASD, then key stakeholders including individuals with ASD, family members, educators, vocational rehabilitation service providers, and others will need to learn how to

individualize an array of specific supports at work designed to meet each person's unique needs.

It will also require education of employers to understand how employing an individual with ASD can bring value to business. In summary, to date, few solid conclusions have been reached, and more research is needed.

Current Knowledge

Predicting outcome and planning for adult service needs for children with autism spectrum disorders (ASD) is limited by gaps in current knowledge. The best quality information about autism in adulthood comes from a few population-based longitudinal studies that estimate the full picture of outcomes. However, changes in diagnostic criteria in the 1990s from a comparatively narrow definition to the broader current criteria for ASD means that studies of adults originally diagnosed as children with historical criteria have limited application to later generations. In addition, longitudinal research that depends on information from aging caregivers has inherent challenges. For example, recall of symptoms from earlier life may be compromised by memory problems and health problems of the aging informant. An alternative research design using cross-sectional samples of adults diagnosed with current, broader criteria can provide data relevant to the future of children being diagnosed today. Such cross-sectional studies are useful adjuncts to population-based, longitudinal research as they give a more comprehensive understanding of ASD in adulthood and can bring focus to specific issues. For example, the prevalence and variety of behaviors that lead to encounters with law enforcement have been described by Allen et al. (2009).

Two useful prognostic factors for adult outcome in ASD are childhood intellectual ability and onset of communicative speech. Like other people with intellectual disabilities (ID), people with ASD and ID generally achieve a limited range of independence and "success" in adult life as defined in developed Western societies

inclusive of gainful employment, a household independent of their parents, and a circle of reciprocal friendships and romantic relationships. Those with average-range intellectual abilities (i.e., ≥ 70) have widely varying adult outcomes. Several longitudinal studies have demonstrated that communicative phrase speech before age 6 and an average-range childhood intelligence quotient (IQ) are necessary for a chance at adult independence but in no way guarantee it (Billstedt et al. 2005; Farley et al. 2009; Howlin et al. 2004; Kobayashi et al. 1992). When assessed in adulthood, barriers to independence in people with ASD and average-range intellectual abilities appear to include co-occurring psychiatric conditions, difficulty with initiation of goal-oriented activities, and poor social skills. There may also be specific genetic variations, developmental processes, educational opportunities, ecological factors, and specialized adult supports that influence levels of independence in adulthood.

Natural Course

While ASD is a lifetime diagnosis, several longitudinal studies have shown improvements in autistic symptoms over the lifespan (Billstedt et al. 2005; Cederlund et al. 2008; Piven et al. 1996; Rumsey et al. 1985; Seltzer et al. 2004). The trend is toward improvement in symptom severity in participants as a group, with the greatest amount of behavioral improvement in individuals who had the highest IQs and the least severe symptom presentation at the initial evaluation. These studies also show that a small proportion of affected individuals no longer meet full diagnostic criteria in adulthood. Importantly, most of these individuals retain subtle impairments that continue to present daily challenges to fully independent functioning.

There also appears to be a small subgroup that experiences significant deterioration in cognitive or behavioral functioning in adolescence (Ballaban-Gil et al. 1996; Kobayashi et al. 1992; Venter et al. 1992). Causes for this deterioration are unknown as yet, but appear unrelated to

adolescent seizure onset that occurs in some individuals with ASD.

Mortality

Studies of mortality in autism have identified a higher rate of mortality for populations with ASD than in the general population, owing largely to complications related to epilepsy and other medical conditions and to accidental deaths that may be associated with ID. Standardized mortality ratios (i.e., the ratio of observed deaths in a specific sample to expected mortality in the general population matched on variables such as age, gender, and length of follow-up period) range from 1.9 to 2.4, approximately twice the expected rate for the general population (Isager et al. 1999; Pickett et al. 2006; Shavelle et al. 2001). Females have had higher mortality rates than males in studied populations, probably associated with a higher rate of ID.

Selected Longitudinal Outcome Studies

A number of authors have categorized outcomes of adults with AD using broad social and educational or occupational criteria (Howlin et al. 2004). Outcome classifications usually include five nodes and range from Very Poor (i.e., the person cannot function independently in any way) to Very Good (i.e., achieving great independence, having friends and a job). Findings from outcome studies are quite disparate, in spite of considerable similarities between outcome criteria and samples. A consistent finding from published outcome studies is that outcome for a majority (approximately 60%) of individuals with ASD was Fair, Poor, or Very Poor (Billstedt et al. 2005; Eaves and Ho 2008; Howlin et al. 2004).

Gillberg and Steffenburg (1987) studied outcome for a population-based sample of 23 people with ASD. As children, one-third obtained IQ scores in the mildly mentally retarded range, and 26% achieved scores in the normal or near normal ranges. Eight (35%) had communicative speech at age 6. These 23 participants were aged

16–23 years at the time of the follow-up. One person (4% of the sample) obtained a “Good” outcome. Thirty-five percent experienced the “Fair, but restricted outcome” (i.e., characteristics of “poor” outcome status, but accepted by and included in some social community). Thirteen percent had a “Fair” outcome, and 44% had “Poor” or “Very Poor” outcomes. Childhood IQ and use of communicative speech at age 6 were useful predictors of outcome status. Epileptic seizures were present in one-third of the population, often associated with severe mental retardation and pubertal symptom aggravation.

Kobayashi et al. (1992) conducted a follow-up investigation of 201 adults identified with ASD in childhood through clinical services in Japan. Four of the people had died. The mean age for the remaining 197 young adults was 21 years and 8 months ($SD = 3.6$). About one-fourth of the sample had an IQ score of 70 or better at age 6, and about 20% were able to speak without echolalia at that age. An additional 31% used communicative language at age 6 but also used echolalic speech. Forty percent of the sample began school in a general education class, but only 27% remained in general education at the age of 12. At follow-up, 43 (21%) were employed and 11 (6%) were enrolled in higher education or vocational training programs. Outcome adjustment was “Good” or “Very Good” for 47%, was “Fair” for 32%, and was “Poor” or “Very Poor” for 46%. Childhood IQ was the only strong predictor of outcome in this investigation. Although there were similarities between the sample in this study and others reported, the outcome for these participants was strikingly better, overall. The authors provided some possible explanations including sociodemographic factors in Japan, advances in public education standards for people with disabilities, intensive intervention histories, and a high proportion of people with ASD and average-range IQ scores at baseline.

Howlin et al. (2004) studied adult outcome for 68 people with ASD who also had a childhood nonverbal IQ score of 50 or better. The mean age at the initial evaluation was 7.24 ($SD = 3.10$) and at follow-up was 29.33 ($SD = 7.97$). Nonverbal IQ scores averaged 80.21 ($SD = 19.28$). At

follow-up, the average nonverbal IQ was 75 ($SD = 21.52$). Almost all of the subjects were known to have attended compulsory schooling; however, only 22% left school having achieved formal qualifications. At the time of the follow-up investigation, 23 people were employed. Eight worked in regular, independent jobs, one was self-employed as an artist but was unable to earn a living wage, and 14 worked in sheltered or supported employment. Twenty-seven people were occupied in general work/leisure programs at day centers for adults with disabilities. Outcome adjustment ratings for the sample were that 22% had “Good” or “Very Good” outcomes, 19% had “Fair” outcomes, and 58% had “Poor” or “Very Poor” outcomes. Analyses of the assessment results revealed that childhood IQ was a useful predictor of adult adjustment in that those with childhood nonverbal IQ scores of 70 or more were more likely to do well than those with scores below 70. Furthermore, a score of 100 or better did not increase the likelihood that a person would do well in adulthood. For those who were capable of completing a childhood verbal IQ measure, the combination of verbal and nonverbal IQ scores in childhood provided a more precise indication of outcome classification, with scores above 70 in both domains yielding the greatest likelihood of a “Fair” outcome or better. Specifically, among those with childhood nonverbal IQ scores of 70 or more, 7 had a “Very Good” outcome, 7 had a “Good” outcome, 10 obtained a “Fair” outcome, and 20 had “Poor” or “Very Poor” outcomes. Language level at age 5 was useful in predicting overall outcome and residential status, but none of the other outcome variables studied demonstrated predictive utility.

Eaves and Ho (2008) followed 48 individuals with autism spectrum disorders from childhood (mean age = 6.8) to adulthood (mean age = 24) in Canada. Fifty-seven percent of this sample had autistic disorder, while the remainder had less severe variants of ASD. Eight of the participants had a childhood IQ score above 70. All participants received special education support during their compulsory schooling years, and 30% engaged in some kind of post-secondary educational program. Overall outcome adjustment

ratings were that 21% had “Good” or “Very Good” outcomes, 32% had “Fair” outcomes, and 46% had “Poor” outcomes. No participants fell within the “Very Poor” outcome categorization. Sixty percent of the sample resided at home with their parents, 19% lived in group homes, and 13% lived in foster care. Almost 80% received a government disability pension and used the services of social workers. In this sample, childhood verbal IQ was most predictive of outcome status. However, the proportion of individuals who were capable of completing an assessment of verbal IQ was not reported.

Also in Cederlund et al. (2008), Cederlund et al. released their study of outcome for 70 adults with autism and 70 adults with Asperger syndrome, after 5 or more years time elapsed from original diagnosis. This research team used the same outcome categorization scheme as Gillberg and Steffenburg (1987), with categories of “Good,” “Fair,” “Restricted,” “Poor,” and “Very Poor.” Twenty-seven percent ($n = 19$) of this sample obtained an outcome categorization of “Good,” and 47% ($n = 33$) were categorized as having a “Fair” outcome. Sixteen people, or 23%, obtained “Restricted” outcome status, and 2 people, or 3%, fell within the “Poor” category. There were no participants with “Very Poor” outcome ratings.

Farley et al. (2009) studied 41 adults who had been identified through a population-based study of ASD in Utah in the 1980s. All of these individuals had previous IQ scores of 70 or greater. Mean age at the first assessment was 7.2 years ($SD = 4.1$) and in adulthood was 32.5 years ($SD = 5.7$). Outcome adjustment was somewhat better for this sample than previous samples, with 48% in the “Very Good” and “Good” categories, 34% in the “Fair” category, and 17% in the “Poor” category. No participants fit within the “Very Poor” category of outcome categorization. Six participants did not meet diagnostic criteria for current ASD using gold-standard diagnostic procedures, but five of these still retained significant social difficulties reported by themselves or significant others. Half were employed on a full- or part-time basis, and 39% had attended some kind of formal post-secondary education. Over half of the

sample (56%) continued to live with their parents, and almost 25% lived in supported living arrangements including a state residential center for people with significant disabilities. Almost 60% of the sample reported co-occurring psychiatric diagnoses. Reported chronic medical conditions were those commonly seen in the general population (e.g., seasonal allergies, gout, high blood pressure).

Cognitive Function

Evidence to date reflects uneven development of cognitive abilities across people with ASD. Initial evaluations during childhood often indicate better nonverbal than verbal abilities. However, many studies show evidence increases in verbal ability and decreases in nonverbal ability during adolescence and adulthood. Group results for individuals with ASD and average-range IQ scores demonstrate consistency in the distribution of subtest scores on Wechsler scales. However, some individuals who have relatively high IQs in childhood demonstrate significant increases in overall ability at follow-up. Disparities among findings may have several causes. Selection of tests at initial evaluation and follow-up for their appropriateness to the research question and participants’ behavior may influence results. Furthermore, tests may not be sufficiently parallel for comparison so that some of the variance is attributable to inequality across measures. Variation of tests from the initial evaluation to follow-up further obscures results since within-group variation on measures may be considerable (Howlin et al. 2004). Age at initial IQ also appears to be an important factor, with nonverbal abilities varying more among children initially tested before age 5 (Howlin et al. 2004).

Associated Co-occurring Conditions

Many of the outcome studies concerning adults with AD provide information concerning co-occurring medical and psychiatric conditions. Few have analyzed the specific contributions

these disorders make to restrictions in overall outcome (Danielsson et al. 2005). One of the clearest indicators of the presence of significant co-occurring psychiatric and medical diagnoses is the proportion of individuals who are prescribed anticonvulsant and psychotropic medications. Eaves and Ho (2008) reported that 40% of their sample was prescribed medication for behavioral difficulties. Similarly, 40% of the participants in the population-based study by Billstedt et al. (2005) were prescribed medication for psychiatric disorders, and 40% of the adolescents and adults in another study were prescribed psychotropic medications to control behavior (Ballaban-Gil et al. 1996). Thirty-seven percent of those studied by Farley et al. (2009) were described as taking prescription medications aimed at managing behavioral difficulties.

Epilepsy is a chronic condition involving recurring seizures and is more common in individuals with ASD than in the general population, with an average prevalence rate of 16.8% across epidemiological studies of ASD (Fombonne 2005). This disorder occurs more frequently in individuals with ASD and ID. The onset of seizures typically occurs early in childhood (i.e., before age 2) or in adolescence (Danielsson et al. 2005; Kobayashi et al. 1992). Seizures remit in a fraction of those afflicted (Danielsson et al. 2005). Kobayashi et al. (1992) reported that 19% of their sample, representing the full range of functioning within ASD, had epilepsy, and all took anti-epileptic medication. Nine percent of a sample of adults with ASD and average-range IQ scores who were taking antiepileptics (Howlin et al. 2004).

Affective disorders challenge a person's capacity to regulate mood and include depression, mania, and bipolar disorder. It is estimated that over 60% of people with AD suffer from a co-occurring affective disorder. In a study of 35 individuals with Asperger syndrome, Ghaziuddin et al. (1998) found that affective disorders were the most common type of psychiatric condition co-occurring in adults, affecting over half of their sample. Figures from outcome studies with adult samples range from 1% to 30% (Billstedt et al. 2005; Farley et al. 2009).

Results of several outcome studies demonstrate that anxiety disorders are present in a large proportion of adults with AD. Rumsey et al. (1985) determined that 50% of their sample was suffering from chronic, generalized anxiety which they suggested could account for the attention difficulties observed in one fifth of the sample. Another study of adults with ASD and average-range IQ scores concluded that 40% of their sample had OCD or chronic anxiety. Figures from other outcome studies are much smaller; however, these results may be confounded by the presence of ritualistic characteristics and hyperactivity commonly associated with ASD (Ghaziuddin et al. 1998).

Hyperactivity and short attention span are common in people with ASD. These have been most commonly noted in children, yet some adults present with behavioral characteristics of attention deficit-hyperactivity disorder (ADHD) as well (Ghaziuddin et al. 1998). Forty (33%) of the adults in the study by Billstedt et al. (2005) presented with hyperactivity.

Psychiatric conditions evident in a small number of people with ASD include tic disorders, psychotic features, and catatonia. Almost 20% of the sample examined by Billstedt et al. (2005) demonstrated tics, and 10% of the adults studied by Eaves and Ho (2008) had Tourette's disorder. One of the 15 adults in another investigation presented with Tourette's disorder (Ghaziuddin et al. 1998). A small number of individuals with ASD genuinely have co-occurring psychotic conditions. Eight percent of the sample in the study of adults with ASD conducted by Billstedt et al. (2005) and 38% of those examined by Szatmari et al. had characteristics of psychosis. Catatonia is another type of psychiatric disturbance that is rarely observed, but notable, in ASD. One of the 15 adults studied by Howlin (2000) had a sudden-onset catatonic episode during puberty. Billstedt et al. (2005) reported a much higher percentage (12%) in their sample of 120 adults.

While not psychiatric disorders in their own right, maladaptive behaviors are significant deviations from expected behavior for a person's developmental level. They are often disruptive and sometimes dangerous. Maladaptive behaviors

are frequently observed in people with ASD of all levels of ability and developmental age. In general terms, maladaptive behaviors have been reported in up to 69% of adults with ASD with no overall difference in frequency between males and females (Ballaban-Gil et al. 1996; Eaves and Ho 2008). Maladaptive behaviors may be relatively infrequent in adults with ASD and average-range IQ scores, but odd or severe enough to preclude acceptance into general social settings over time (Rumsey et al. 1985). Self-injurious behaviors were reported to have occurred in 50% of the sample studied by Billstedt et al. (2005) and have been reported to be more common in females than in males (Ballaban-Gil et al. 1996). Difficulties with toileting and feeding appear to persist in lower-functioning individuals, but difficulties with compulsive rituals may develop around these tasks in higher-functioning adults as well. Aggression among adults is rarely designed to harm others, but property damage or harm to self may occur intermittently, sometimes in response to unimportant changes or problems in the environment (Rumsey et al. 1985).

Social Relationships

Few adults with ASD develop significant relationships outside of the family of origin in spite of common increases in interest in developing social relationships as individuals with AD age (Rumsey et al. 1985). Almost 75% of family members interviewed in the study by Eaves and Ho (2008) reported that they enjoyed good to excellent relationships with their affected relative; however, only one-third of the sample of affected adults had one or more friendships outside of the family. Similar results have been found in other studies of adults with ASD (Howlin et al. 2004). Females have reportedly experienced greater success with peer relationships than males (Piven et al. 1996). Ten percent of adults in the study by Eaves and Ho (2008) had a romantic relationship at some time in the past, but none of the participants were romantically involved at the time of the investigation. Nineteen percent of the men with Asperger syndrome disorder in the Cederlund et al. (2008)

study and 3% of the men with autistic disorder were or had been in long-term romantic relationships. Thirty-two percent of those studied by Farley et al. (2009) had dated, and 20% were involved in a serious relationship at the time of the study. In general, very few adults with ASD have been reported to have successful, long-term romantic relationships (Howlin et al. 2004).

Education and Employment

Approximately 15% of adults with ASD studied in outcome research attend post-secondary education programs (Ballaban-Gil et al. 1996; Farley et al. 2009; Kobayashi et al. 1992; Rumsey et al. 1985; Venter et al. 1992). In general, gainful employment for adults with ASD is rare, as is sheltered employment, occupying less than 40% of adults with AD (Howlin et al. 2004). While outcome studies of autism into adulthood conducted since 1992 reflect some steady improvements in employment rates, with 22–54% of participants reporting gainful employment on a full- or part-time basis (Ballaban-Gil et al. 1996; Farley et al. 2009; Howlin 1994; Kobayashi et al. 1992; Venter et al. 1992), many of these individuals are underemployed based on their cognitive abilities and academic credentials.

Forensic Problems

Involvement with police officers and other law enforcement agents has been recognized as a major concern for parents of adolescents and adults with ASD. A study of offending behavior in 33 individuals with Asperger syndrome (Allen et al. 2009) revealed that most engaged in violent or threatening behavior that was related to interpersonal problems including social or sexual rejection, bullying, or family conflict. Investigators have suggested such offending behavior was likely to result from coercion by others, misinterpretation of social situations, or obsessional interests, while many with ASD may be protected from these by their tendency to adhere strictly to rules. Allen et al. (2009) found evidence of this insight

in that the least common offenses identified among their population of offenders with Asperger syndrome were drug offenses, theft, fraud, sexual offending, and motor offenses. Cederlund et al. (2008) found that 10% ($n = 7$) of their sample with Asperger syndrome had been involved with law enforcement officers, but the remainder was described as very law abiding. None of the individuals in their lower-functioning sample with autistic disorder had committed legal offenses. In the study by Farley et al. (2009), 29% of the sample was involved with law enforcement offices for infractions after childhood, but these were related exclusively to “suspicious” behaviors deriving from special interests, participants being coerced to engage in antisocial behavior by peers, and social misunderstandings.

Future Directions

The prognosis for a majority of adults with ASD based on studies conducted to date is guarded. Future studies are needed to further define the subtypes of ASD and the factors that influence adult outcome. Studies of genetics, brain imaging, and responses to interventions are likely to yield important information.

See Also

- Adulthood, Transition to
- Advocacy
- Asperger Syndrome Follow-Up Studies
- Benhaven Residential Services
- Community Services
- Community-Integrated Residential Services for Adults with Autism
- Competitive Employment
- Comprehensive Transition Program
- Course of Development
- Employment
- Employment Specialist
- Functional Life Skills
- Guardianship
- Independent Living
- Individualized Plan for Employment (IPE)
- Job Carving
- Job Coach
- Law Enforcement Agencies and Autism
- Legal Competency
- Living Arrangements in Adulthood
- Secure Employment
- Self-Advocacy
- Sexuality in Autism
- Sheltered Employment
- Sheltered Workshops
- Support Trust
- Supported Employment
- Transition Planning
- Transitional Living
- Travel Training
- Trust
- Violent/Criminal Behavior in Autism
- Vocational Evaluator
- Vocational Rehabilitation Act of 1973
- Vocational Training

References and Reading

- Allen, D., Lowe, K., Brophy, S., & Moore, K. (2009). Predictors of restrictive reactive strategy use in people with challenging behaviour. *Journal of Applied Research in Intellectual Disabilities*, 22(2), 159–168. Special Issue: Restrictive Behavioural Practices. <http://onlinelibrary.wiley.com/doi/10.1111/jar.2009.22.issue-2/issuetoc>
- Ballaban-Gil, K. M., Rapin, I., Tuchman, R., & Shinnar, S. (1996). Longitudinal examination of the behavioral, language, and social changes in a population of adolescents and young adults with autistic disorder. *Pediatric Neurology*, 15(3), 217–223.
- Berkman, K. A., & Meyer, L. H. (1988). Alternative strategies and multiple outcomes in the remediation of severe self-injury: Going “all out” nonaversively. *Journal of the Association for Persons with Severe Handicaps*, 13(2), 76–86.
- Billstedt, E., Gillberg, C., & Gillberg, C. (2005). Autism after adolescence: Population-based 13- to 22-year follow-up study of 120 individuals with autism diagnosed in childhood. *Journal of Autism and Developmental Disorders*, 35(3), 351–360.
- Bolman, W. M. (2008). Brief report: 25-year follow-up of a high-functioning autistic child. *Journal of Autism and Developmental Disorders*, 38, 181–183.
- Burt, D. B., Fuller, S. P., & Lewis, K. R. (1991). Competitive employment of adults with autism. *Journal of Autism and Developmental Disorders*, 21(2), 237–242.
- Camarena, P. M., & Sarigiani, P. A. (2009). Postsecondary educational aspirations of high-functioning adolescents

- with autism spectrum disorders and their parents. *Focus on Autism and Other Developmental Disabilities*, 24(2), 115–128. Cameto 2004.
- Cederlund, M., Hagberg, B., Billstedt, E., Gillberg, I. C., & Gillberg, C. (2008). Asperger syndrome and autism – A comparative longitudinal follow-up study more than 5 years after original diagnosis. *Journal of Autism and Developmental Disorders*, 38, 72–85.
- Cimera, R. E., & Cowan, R. J. (2009). The costs of services and employment outcomes achieved by adults with autism in the US. *Autism*, 13(3), 285–302.
- Danielsson, S., Gillberg, I. C., Billstedt, E., Gillberg, C., & Olsson, I. (2005). Epilepsy in young adults with autism: A prospective population-based follow-up study of 120 individuals diagnosed in childhood. *Epilepsia*, 46, 918–923.
- Eaves, L. C., & Ho, H. (2008). Young adult outcome of autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38, 739–747.
- Farley, M., McMahon, W., Fombonne, E., Jenson, W., Miller, J., Gardner, M., et al. (2009). Twenty-year outcome for individuals with autism and average or near-average cognitive abilities. *Autism Research*, 2, 109–118.
- Fast, Y. (2004). *Employment for individuals with Asperger syndrome or non verbal learning disability: Stories and strategies*. London/Philadelphia: Jessica Kingsley.
- Foley, B. E., & Staples, A. H. (2003). Developing augmentative and alternative communication (AAC) and literacy interventions in a supported employment setting. *Topics in Language Disorders*, 23(4), 325–343.
- Fombonne, E. (2005). The changing epidemiology of autism. *Journal of Applied Research in Intellectual Disabilities*, 2005, 281–294.
- Ghaziuddin, M., Weidmer-mikhail, E., & Ghaziuddin, N. (1998). Comorbidity of Asperger syndrome: A preliminary report. *Journal of Intellectual Disability Research*, 42(4), 279–283.
- Gillberg, C., & Steffenburg, S. (1987). Outcome and prognostic factors in infantile autism and similar conditions: A population-based study of 46 cases followed through puberty. *Journal of Autism and Developmental Disorders*, 17, 273–287.
- Hagner, D., & Cooney, B. F. (2005). “I do that for everybody”: Supervising employees with autism. *Focus on Autism and other Developmental Disabilities*, 20(2), 91–97.
- Hillier, A., Campbell, H., Mastriana, K., Izzo, M., Kool-Tucker, A., Cherry, L., et al. (2007a). Two-year evaluation of a vocational support program for adults on the autism spectrum. *Career Development for Exceptional Individuals*, 30(1), 35–47.
- Hillier, A., Fish, T., Cloppert, P., & Beversdorf, D. Q. (2007b). Outcomes of a social and vocational skills support group for adolescents and young adults on the autism spectrum. *Focus on Autism and Other Developmental Disabilities*, 22(2), 107–115.
- Howlin, P. (1994). Facilitated communication: are the claims for success justified? *Communication*, 28, 10–12.
- Howlin, P. (2000). Outcome in adult life for more able individuals with ASD or Asperger syndrome. *ASD*, 4(1), 63–83.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45(2), 212–229.
- Howlin, P., Alcock, J., & Burkin, C. (2005). An 8 year follow-up of a specialist supported employment service for high-ability adults with autism or Asperger syndrome. *Autism: The International Journal of Research & Practice*, 9(5), 533–549.
- Hume, K., & Odom, S. (2007). Effects of an individual work system on the independent functioning of students with autism. *Journal of Autism and Developmental Disorders*, 37, 1166–1180.
- Hurlbutt, K., & Chalmers, L. (2002). Adults with autism speak out: Perceptions of their life experiences. *Focus on Autism and other Developmental Disabilities*, 17(2), 103–111.
- Hurlbutt, K., & Chalmers, L. (2004). Employment and adults with Asperger syndrome. *Focus on Autism and other Developmental Disabilities*, 19(4), 215–222.
- Isager, T., Mouridsen, S. E., & Rich, B. (1999). Mortality and causes of death in pervasive developmental disorders. *Autism*, 3, 7–16.
- Keel, J. H., Mesibov, G. B., & Woods, A. (1997). TEACCH-supported employment program. *Journal of Autism and Developmental Disorders*, 27, 3–9.
- Kobayashi, R., & Murata, T. (1998). Setback phenomenon in autism and long-term prognosis. *Acta Psychiatrica Scandinavica*, (4), 296–303.
- Kobayashi, R., Murata, T., & Yoshinaga, K. (1992). A follow-up study of 201 children with autism in Kyushu and Yamaguchi areas, Japan. *Journal of Autism and Developmental Disorders*, 22, 395–411.
- Landa, R. J., & Goldberg, M. C. (2005). Language, social, and executive functions in high functioning autism: A continuum of performance. *Journal of Autism and Developmental Disorders*, 35, 557–573.
- Lawer, L., Brusilovskiy, E., Salzer, M. S., & Mandell, D. S. (2009). Use of vocational rehabilitative services among adults with autism. *Journal of Autism and Developmental Disorders*, 39, 487–494.
- Lopez, B. R., Lincoln, A. J., Ozonoff, S., & Lai, Z. (2005). Examining the relationship between executive functions and restricted, repetitive symptoms of autistic disorder. *Journal of Autism and Developmental Disorders*, 35, 445–460.
- Marks, S. U., Schrader, C., Longaker, T., & Levine, M. (2000). Portraits of three adolescent student's with Asperger's syndrome: Personal stories and how they can inform practice. *The Journal of the Association for Persons With Severe Handicaps*, 25(1), 3–17.
- Mawhood, L., & Howlin, P. (1999). The outcome of a supported employment scheme for high functioning adults with autism or Asperger syndrome. *Autism*, 3, 229–254.
- Mawhood, L., Howlin, P., & Rutter, M. (2000). Autism and developmental receptive language disorder-A comparative follow-up in early adult life. I: Cognitive and

- language outcomes. *Journal of Child Psychology and Psychiatry*, 41(5), 547–559.
- McEvoy, R. E., Rogers, S. J., & Pennington, B. F. (1993). Executive function and social communication deficits in young autistic children. *Journal of Child Psychology and Psychiatry*, 34, 563–578.
- Müller, E., Schuler, A., Burton, B. A., & Yates, G. B. (2003). Meeting the vocational support needs of individuals with Asperger syndrome and other autism spectrum disabilities. *Journal of Vocational Rehabilitation*, 18(3), 163–175.
- National Organization on Disability. (2004). *N.O.D./Harris survey of Americans with Disabilities: Landmark survey finds pervasive disadvantages*. Washington, DC: Author. Retrieved October 22, 2006, from <http://www.nod.org/content.cfm?id=1537>
- Nesbitt, S. (2000). Why and why not? Factors influencing employment for individuals with Asperger syndrome. *Autism*, 4(4), 357–369.
- Njardvik, U., Matson, J. L., & Cherry, K. E. (1999). A comparison of social skills in adults with autistic disorder, pervasive developmental disorder not otherwise specified, and mental retardation. *Journal of Autism and Developmental Disorders*, 29(4), 287–295.
- Nuehring, M. L., & Sitlington, P. L. (2003). Transition as a vehicle: Moving from high school to an adult vocational service provider. *Journal of Disability Policy Studies*, 14(1), 23–35.
- Patterson, A., & Rafferty, A. (2001). Making it to work: Towards employment for the young adult with autism. *International Journal of Language and Communication*, 36, 475–480.
- Pickett, J. A., Paculdo, D. R., Shavelle, R. M., & Strauss, D. J. (2006). 1998–2002 update on “Causes of Death in Autism.” *Journal of Autism and Developmental Disorders*, 36, 287–288.
- Piven, J., Harper, J., Palmer, P., & Arndt, S. (1996). Course of behavioral change in autism: a retrospective study of high-IQ adolescents and adults. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35(4), 523–529.
- Ruef, M. B., & Turnbull, A. P. (2002). The perspectives of individuals with cognitive disabilities and/or autism on their lives and their problem behavior. *Journal of the Association for Persons with Severe Handicaps*, 27(2), 125–140.
- Rumsey, J. M., & Hamburger, S. D. (1988). Neuropsychological findings in high-functioning men with infantile autism, residual state. *Journal of Clinical and Experimental Neuropsychology*, 10, 201–221.
- Rumsey, J. M., Rapoport, J. L., & Sceery, W. R. (1985). Autistic children as adults: psychiatric, social, and behavioral outcomes. *Journal of the American Academy of Child and Adolescent Psychiatry*, 24, 465–473.
- Schaller, J., & Yang, N. K. (2005). Competitive employment for people with autism: Correlates of successful closure in competitive and supported employment. *Rehabilitation Counseling Bulletin*, 49(1), 4–16.
- Seltzer, M. M., Krauss, M. W., Shattuck, P. T., Orsmond, G., Swe, A., & Lord, C. (2003). The symptoms of autism spectrum disorders in adolescence and adulthood. *Journal of Autism and Developmental Disorders*, 33, 565–581.
- Seltzer, M. M., Shattuck, P., Abbeduto, L., & Greenberg, J. S. (2004). Trajectory of development in adolescents and adults with autism. *Mental Retardation and Developmental Disabilities Research Reviews*, 20(4), 234–247.
- Shavelle, R. M., Strauss, D. J., & Pickett, J. (2001). Causes of death in autism. *Journal of Autism and Developmental Disorders*, 31, 6.
- Smith, M. (1986). Managing the aggressive and self-injurious behaviors of adults disabled by autism in the community. *Journal of the Association for Persons with Severe Handicaps*, 10, 228–232.
- Smith, M. (1987). Treatment of pica in an adult disabled by autism by differential reinforcement of incompatible behavior. *Journal of Behavior Therapy and Experimental Psychiatry*, 18(3), 285–288.
- Smith, M. (1990). *Autism and life in the community: Successful interventions for behavioral challenges*. Baltimore: Paul H. Brookes.
- Smith, M. (1994). Increasing work productivity of employees disabled by autism. *Journal of Vocational Rehabilitation*, 4(1), 60–65.
- Smith, M., & Belcher, R. (1985). Teaching life skills to adults disabled by autism. *Journal of Autism and Developmental Disorders*, 15, 163–175.
- Smith, M., & Coleman, D. (1986). Managing the behavior of adults with autism in the job setting. *Journal of Autism and Developmental Disorders*, 16, 145–154.
- Smith, M. D., & Philippen, L. R. (1999). Community integration and supported employment. In D. E. Berkell (Ed.), *Autism: Identification, education, and treatment* (pp. 253–271). Mahwah: Erlbaum.
- Smith, M., Belcher, R. G., & Juhrs, P. D. (1995). *A guide to successful employment for individuals with autism*. Baltimore: Paul H. Brookes.
- Sperry, L. A., & Mesibov, G. B. (2005). Perceptions of social challenges of adults with autism spectrum disorder. *Autism*, 9(4), 362–376.
- Targett, P., & Wehman, P. (2009). Integrated employment. In P. Wehman, M. Datlow Smith, & C. Schall (Eds.), *Autism and the transition to adulthood: Success beyond the classroom*. Baltimore: Paul H. Brookes.
- Twachtman-Cullen, D. (2000). More able children with autism spectrum disorders. In A. M. Wetherby & B. M. Prizant (Eds.), *Autism spectrum disorders* (pp. 225–249). Baltimore: Paul H. Brookes.
- Venter, A., Lord, C., & Schopler, E. (1992). A follow-up study of high-functioning autistic children. *Journal of Child Psychology and Psychiatry*, 33, 489–507.
- Wagner, M., Newman, L., Cameto, R., Garza, N., & Levine, P. (2005). *After high school: A first look at the post school experiences of youth with disabilities*. A report from the National Longitudinal Transition Study-2 (NLTS-2). SRI International, Menlo Park.
- Wehman, P., Datlow Smith, M., & Schall, C. (2009). *Autism and the transition to adulthood: Success beyond the classroom*. Baltimore: Paul H. Brookes.

Employment of Adults with ASD: A Motivational Perspective

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Definition

Work has a central role in adult life. Employment integration can fulfill a variety of needs, from basic subsistence, through social inclusion, to an expression of personal interests, abilities, self-concepts and values (Blustein 2013). Although employment of adults diagnosed with autism spectrum disorder (ASD) has received growing attention and effort, outcomes in this field remain unsatisfactory. In aim of taking the field foreword, new theoretical perspectives can shed more light on labor market integration of adults with ASD. We suggest that the self-determination theory (SDT) of motivation (Deci and Ryan 2000) has the potential to expand our knowledge and inform vocational rehabilitation practice.

Historical Background

In view of the growing prevalence of ASD, more and more diagnosed individuals face the transition from childhood and adolescence into adulthood, which presents new, complicated challenges. Concurrently, research looking into adult outcomes is growing in number and in breadth. The last decades have seen transition in the conceptualization of successful adult outcomes for individuals with ASD, with outcomes becoming more specific and measures more valid and reliable. As part of this change, employment has become a specific outcome criterion in adulthood (Henninger and Taylor 2013). Hence, the history

of autism and employment in research and practice is relatively young, and the road to successful employment is still considerably unpaved.

Looking at the literature up to the present time, three main research directions are evident: evaluations of employment outcomes; conceptualizations of strengths and barriers to work; and assessments of interventions, aiming to point out what elements promote success.

Many studies quantify employment outcomes of adults with ASD, in comparison to the general population and to other clinical groups. Overall, results show high rates of unemployment, unfavorable job conditions for those who do work, and a relatively high prevalence of “overeducation,” representing the gap between educational attainments and actual jobs (Baldwin et al. 2014). Outcomes are unfavorable not only in comparison to the general population but also to other disabilities (Chen et al. 2015).

A second body of knowledge is directed at defining common work-related strengths and challenges. Many studies have been devoted to this objective, examining perspectives of employees, employers, and other stakeholders involved, such as parents and vocational rehabilitation practitioners. The picture known to us today is quite detailed and consistent across studies. Challenges that frequently arise when facing employment are related to communication and social deficits, cognitive functions (specifically impairments in executive functions), and difficulties modulating sensory stimuli. These various difficulties can be a constant source of stress and anxiety, which also pose a central obstacle for individuals with ASD who seek employment (Chen et al. 2015; Hendricks 2010).

Alongside the challenges, there are also unique characteristics that may give individuals with ASD an advantage in the work domain. Research lists advantages such as enhanced visual search abilities, a preference for detail-oriented tasks, and a systematic working style (Chen et al. 2015; Hendricks 2010). Characteristic “special interests” have been also suggested to lead to expertise that can form an advantage in certain fields (Winter-Messiers 2007). Moreover, employers of workers with ASD reported

favorable personal characteristics such as punctuality, devotion, and consistency, which are perceived as contributing to work performance (Scott et al. 2017).

Last, in an attempt to overcome barriers to employment, interventions aimed at promoting employment integration were developed. Programs answering the needs of adults with ASD have been documented for over a decade. These target the common obstacles described above and apply different strategies for success. Commonly, the curriculum includes improving social and executive skills. Interventions offer work placement along with personalized supports and accommodations (Hedley et al. 2017). These practices seem promising, as research results show good outcomes. However, there is still a long way to go in means of the quality of research being done and the ability to generalize and disseminate research findings for the benefit of successful inclusion of the wide population of adults with ASD in the vocational world.

Current Knowledge

Two notable trends are prominent in the literature in recent years. The first widens the scope beyond the personal characteristics of the individuals seeking employment and suggests environmental accommodations are also obligatory. A focus on finding a fit between the person, the job, and the occupational surrounding is becoming more and more prominent. Success is no longer considered solely the responsibility of the individual, and the environment is also expected to adjust (Scott et al. 2019; Waisman-Nitzan et al. 2018). In addition, it is becoming clear that the key to success involves cooperation of multiple stakeholders (Nicholas et al. 2018).

The second trend, aligned with the neurodiversity movement, encourages organizations to accept workers with ASD and benefit from the unique characteristics and skills they may bring to the job. This strength-based approach emphasizes the need to highlight ASD-related strengths that can be relevant to the labor market. A common expression of this approach

can be found in the suggestion that characteristic “special interests” can be used to promote labor market integration (Winter-Messiers 2007; Kirchner and Dziobek 2014). This approach has been complimented by cultural trends, portrayed in the media and through anecdotal autobiographical accounts of individuals with autism. These cases demonstrate how a special interest in a specific field, matched with high abilities, can lead to a successful career (Grandin 2006). This potential match has been discussed in the field but has not been systematically studied (Harrop et al. 2019). The feasibility of using this approach in a wider population of adults with ASD seeking employment has been questioned (Goldfarb et al. 2019) and requires further investigation.

Without underestimating the importance of promoting workplace diversity, the reality of the labor market holds expectations for performance, productiveness, and profitability. Hence, a creative and pragmatic approach is needed to integrate these possibly contradicting approaches.

Overall, knowledge in the field of employment of adults with ASD is relatively young and still facing major development. Comparatively, research of employment within the general population has a rich history rooted in various disciplines. The use of well-established theories as a lens for further understanding of employment of adults with ASD is therefore warranted. Among the various theories of work, motivation has been widely researched within different disciplines (e.g., career psychology, organizational research, human resources, occupational therapy) and with relation to different contexts, including work, education, and health. Work motivation holds the potential of promoting action and perseverance. Since work can be a source of stress, with workers expected to carry out tasks in which they do not find interest or that do not match their level of training, motivation is important for maintaining long-term stability, an important goal for adults with ASD.

Common theories cluster work motivation into two basic categories: extrinsic motivation and intrinsic motivation. Extrinsic motivation to work is aimed at receiving benefits apart from the work itself, such as income or praise (Gagné

and Deci 2005). Studies show that it was found to be associated with less favorable outcomes such as lower job satisfaction, higher emotional exhaustion, impatience, lower ability to solve problems, less creativity, and more turnover intention.

Intrinsic motivation is defined as an engagement in work primarily for its own sake, because the work itself is interesting. Unlike extrinsic motivation, studies suggest that being intrinsically motivated to work has beneficial effects such as a higher sense of autonomy and higher levels of work performance and adaptation. Therefore, an intrinsic interest, achieved through matching between personal interests and environment, is one of the core theoretical foundations in vocational theories (Blustein 2013). In relation to ASD, it has been suggested that intrinsic motivation can be prompted by matching between special interests and jobs (Kirchner and Dziobek 2014; Winter-Messiers 2007).

Despite the clear benefits of intrinsic motivation, an intrinsically interesting job is not always a possibility, especially for populations faced with barriers and disabling conditions. In order to deal with this discrepancy, SDT has been suggested as a powerful lens through which work motivation can be addressed (Gagné and Deci 2005). The theory is distinct from other theories of motivation by two main conceptualizations: (a) it goes beyond the extrinsic vs. intrinsic motivation dichotomy and defines different categories of extrinsic motivation and (b) the theory addresses personal needs that have the potential of facilitating more adaptive forms of extrinsic motivation, thus “fueling” the motivational process.

With regard to the first distinction, SDT places different levels of motivation on a continuum. At one end is intrinsic motivation, considered to present the highest level of self-determined behavior, and at the other end is amotivation, which reflects no intention to act. In between, different levels of extrinsic motivation are described: On the lower end, external motivation describes an action aiming for an external desired outcome (such as earning money or getting approval), or avoiding a negative one (such as being fired); interjected motivation describes a form of motivation in

which the individual accepts the action as his own, relating to concepts such as ego involvement and self-worth (such as feeling proud or avoiding shame). This kind of motivation is considered internalized, but is still relatively controlled because it involves pressure to behave in a certain way; identified motivation accompanies actions that are congruent with personal goals and identities. Even when these activities are not intrinsically interesting or even unpleasant, they are viewed as important and hold personal significance. The higher the motivation is on the continuum, the more it involves acting with a sense of desire and having an experience of choice and self-determination (Gagné and Deci 2005).

Another aspect distinguishing SDT from other motivation theories is the conception that self-determined behavior can be achieved only when three psychological needs are satisfied: competence, relatedness, and autonomy (Gagné and Deci 2005). The feeling of autonomy provides a sense of authenticity, choice, volition, and self-regulation; Social relatedness is the feeling of being connected to others in a meaningful way; finally, the need for competence is related to a person's basic strive to experience success and a feeling of mastery. Accordingly, SDT suggests that frustration of these three basic needs can lead to a decrease in motivation.

Coming back to what we know about ASD and employment, inferences can be suggested on the basis of current knowledge: The need for *autonomy* may also involve dealing with uncertainty, which may be a source of stress for individuals with ASD. However, autonomy was identified as one of the factors leading to job satisfaction in adults with ASD (Pfeiffer et al. 2018). Furthermore, in the general population, studies did show that the need for autonomy can also be fulfilled in a structured environment (Jang et al. 2010), i.e., that autonomy is not necessarily tied with ambiguity.

Work provides an important opportunity for social interaction in daily life and a chance to experience *social relatedness*. This opportunity is especially important for adults with ASD, for whom work is sometimes the only setting for meeting other people. In contrast to the

common stereotype that individuals with ASD prefer to be alone, there is clear evidence to support their need for social engagement (Jaswal and Akhtar 2019). In the work arena, individuals with ASD mention social inclusion as a major factor leading to employment satisfaction and success (Krieger et al. 2012; Pfeiffer et al. 2018). Hence, the contribution of social relatedness experience to work satisfaction can help maintain motivation over time, without having to depend on an ongoing interest in the tasks performed.

Turning to the need for *competence*, it has been found that workers with ASD, as well as other populations, want to see themselves as good workers and take pride in their skills (Müller et al. 2003). Lorenz and Heinitz (2014) found a positive relationship between occupational self-efficacy and proactiveness, which can be considered a form of self-determined behavior.

To conclude, the basic needs portrayed by SDT hold relevance for individuals with ASD seeking stability and satisfaction in the workplace. The unsatisfactory occupational outcomes mentioned above, along with common gaps between the intellectual potential and employment demands, may detain success. To address this gap, motivational anchors like autonomy, relatedness, and competence can promote satisfaction and stability.

Future Directions

Employment of adults with ASD is a relatively new field of research, which requires more input. As current knowledge is limited in scope and depth, new research directions have the potential to spark fresh insights regarding employment of adults with ASD. Theories of motivation such as SDT can uncover the underlying mechanisms that enhance work motivation and promote other desirable employment outcomes. The emphasis on the work environment, which can be shaped and adjusted in order to respond to the workers' needs and support them, contributes to the promising notion that motivation is not just a given state, but can rather be facilitated toward a feeling of autonomy and volition. Future studies that

substantiate empirical validation of the theory in relation to adults with ASD can take us a step further in achieving this goal.

The theoretical perspective of SDT can also inform vocational practice. When facing a career choice, STD can help prioritize different considerations that should be taken into account: manifestation of interest areas, probability of success, characteristics of the social environment, work stress, and environmental factors. When an intrinsically motivating job is not available at a given point in time, other motives to work might be established. Interventions aimed at promoting employment integration can potentially prompt work motivation by focusing on fulfilling the needs for autonomy, relatedness, and competence, (Silva et al. 2014). For example, autonomy support can be enhanced by providing the option to choose between job alternatives; support for competence can be achieved by choosing gradual and realistic employment goals and offer practical guidance and support in achieving them; support for relatedness can be facilitated by enhancing relationships in the workplace, leading to a feeling of belonging. Satisfaction of these needs can be monitored by the employer or by a vocational coach through an open channel of communication. With this approach, motivational setbacks can be addressed, and needs can be answered to maintain stability and prevent attrition.

Future studies examining motivation and other work-related theories should also aim to understand workers with ASD in comparison to fellow neurotypical workers. If we aspire to create a work environment that is beneficial for all workers, motivations and needs of diverse populations should be assessed, considering their similarities and differences. Taking into account the high heterogeneity in ASD, these understandings can promote interventions that are more sensitive to individual differences and lead to a better understanding of "what works for whom". Future studies that address the issues raised in this chapter, and apply more fundamental theories of work to adults with ASD, can advance the field and pave new roads for employment success.

See Also

- ▶ [Competitive Employment](#)
- ▶ [Employment in Adult Life](#)
- ▶ [Employment Trends for People with ASD](#)
- ▶ [Restricted Interest](#)

References and Reading

- Baldwin, S., Costley, D., & Warren, A. (2014). Employment activities and experiences of adults with high-functioning autism and Asperger's disorder. *Journal of Autism and Developmental Disorders*, 44(10), 2440–2449.
- Blustein, D. (2013). *The psychology of working: A new perspective for career development, counseling, and public policy*. New York: Routledge.
- Chen, J. L., Leader, G., Sung, C., & Leahy, M. (2015). Trends in employment for individuals with autism spectrum disorder: A review of the research literature. *Review Journal of Autism and Developmental Disorders*, 2(2), 115–127.
- Deci, E. L., & Ryan, R. M. (2000). The “what” and “why” of goal pursuits: Human needs and the self-determination of behavior. *Psychological Inquiry*, 11(4), 227–268.
- Gagné, M., & Deci, E. L. (2005). Self-determination theory and work motivation. *Journal of Organizational Behavior*, 26(4), 331–362.
- Goldfarb, Y., Gal, E., & Golan, O. (2019). A conflict of interests: A motivational perspective on special interests and employment success of adults with ASD. *Journal of Autism and Developmental Disorders*, 49(9), 3915–3923.
- Grandin, T. (2006). *Thinking in pictures: And other reports from my life with autism*. New York: Vintage.
- Harrop, C., Amsbary, J., Towner-Wright, S., Reichow, B., & Boyd, B. A. (2019). That's what I like: The use of circumscribed interests within interventions for individuals with autism spectrum disorder. A systematic review. *Research in Autism Spectrum Disorders*, 57, 63–86.
- Hedley, D., Uljarević, M., Cameron, L., Halder, S., Richdale, A., & Dissanayake, C. (2017). Employment programs and interventions targeting adults with autism spectrum disorder: A systematic review of the literature. *Autism*, 21(8), 929–941.
- Hendricks, D. (2010). Employment and adults with autism spectrum disorders: Challenges and strategies for success. *Journal of Vocational Rehabilitation*, 32(2), 125–134.
- Henninger, N. A., & Taylor, J. L. (2013). Outcomes in adults with autism spectrum disorders: A historical perspective. *Autism*, 17(1), 103–116.
- Jang, H., Reeve, J., & Deci, E. L. (2010). Engaging students in learning activities: It is not autonomy support or structure but autonomy support and structure. *Journal of Educational Psychology*, 102(3), 588.
- Jaswal, V. K., & Akhtar, N. (2019). Being versus appearing socially uninterested: Challenging assumptions about social motivation in autism. *Behavioral and Brain Sciences*, 42(e82), 1–73.
- Kirchner, J. C., & Dziobek, I. (2014). Towards successful employment of adults with autism: A first analysis of special interests and factors deemed important for vocational performance. *Scandinavian Journal of Child and Adolescent Psychiatry and Psychology*, 2(2), 77–85.
- Krieger, B., Kinebanian, A., Prodinger, B., & Heigl, F. (2012). Becoming a member of the work force: Perceptions of adults with Asperger syndrome. *Work*, 43, 141–157.
- Lorenz, T., & Heinitz, K. (2014). Aspergers—different, not less: Occupational strengths and job interests of individuals with Asperger's syndrome. *PLoS One*, 9(6), e100358.
- Müller, E., Schuler, A., Burton, B. A., & Yates, G. B. (2003). Meeting the vocational support needs of individuals with Asperger syndrome and other autism spectrum disabilities. *Journal of Vocational Rehabilitation*, 18(3), 163–175.
- Nicholas, D. B., Mitchell, W., Dudley, C., Clarke, M., & Zulla, R. (2018). An ecosystem approach to employment and autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(1), 264–275.
- Pfeiffer, B., Brusilovskiy, E., Davidson, A., & Persch, A. (2018). Impact of person-environment fit on job satisfaction for working adults with autism spectrum disorders. *Journal of Vocational Rehabilitation*, 48(1), 49–57.
- Scott, M., Jacob, A., Hendrie, D., Parsons, R., Girdler, S., Falkmer, T., & Falkmer, M. (2017). Employers' perception of the costs and the benefits of hiring individuals with autism spectrum disorder in open employment in Australia. *PLoS One*, 12(5), e0177607.
- Scott, M., Milbourn, B., Falkmer, M., Black, M., Bölte, S., Halladay, A., et al. (2019). Factors impacting employment for people with autism spectrum disorder: A scoping review. *Autism*, 23(4), 869–901.
- Silva, M. N., Marques, M. M., & Teixeira, P. J. (2014). Testing theory in practice: The example of self-determination theory-based interventions. *The European Health Psychologist*, 16(5), 171–180.
- Waisman-Nitzan, M., Gal, E., & Schreuer, N. (2019). Employers' perspectives regarding reasonable accommodations for employees with autism spectrum disorder. *Journal of Management & Organization*, 25(4), 481–498.
- Winter-Messiers, M. A. (2007). From tarantulas to toilet brushes: Understanding the special interest areas of children and youth with Asperger syndrome. *Remedial and Special Education*, 28(3), 140–152.

Employment Readiness Skills

- [Preemployment Skills for People with ASD](#)

Employment Services

- [Department of Vocational Rehabilitation](#)

Employment Specialist

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Synonyms

[Employment consultant](#); [Job coach](#); [Vocational rehabilitation specialist](#)

Definition

An employment specialist is a vocational rehabilitation professional, who assists individuals with the most severe disabilities with gaining and maintaining employment in the community by providing supported employment services. The employment specialist who may also be known as a job coach or employment consultant provides individualized services to an individual with a severe disability with all phases of the employment process which include (a) preemployment, (b) employment, and (c) postemployment. Although the type of support service provided is individualized typically the employment specialist is engaged in the following activities.

During phase one, the employment specialist conducts preemployment activities. This requires spending time setting up and implementing a variety of functional assessment activities. These

practical experiences offer both the job seeker and employment specialist with opportunities to learn more about his or her (the job seeker's) abilities, interests, work preferences, and potential on the job support needs. Then with a vision of the jobseeker in mind, the employment specialist spends time meeting with potential employers to learn more about their business needs and positions him or herself as someone who has services to meet a range of business needs including the referral of potential employees with disabilities. During this time, the employment specialist explores ways a particular job seeker may be able to make a contribution to the workplace. An informational interview and workplace analysis take place to provide the employment specialist with insight into whether or not the job seeker may be able to fill an existing position or possible ways to restructure or create a job for the job seeker. At this time, there is also a review of what type, level, and intensity of both on and off the job supports the job seeker may require if hired. If an employer seems willing to move forward with a proposition on hiring the person, then the possibilities for employment are discussed further with the job seeker and or his or her family. In particular, the pros and cons associated with the potential job offer are reviewed. If the job seeker is interested in exploring it, then an introductory meeting is arranged which depending on the circumstances may be an introductory meeting and/or a job interview.

Once hired, or during the employment phase, the employment specialist provides and facilitates a variety of on or off the job supports specifically designed to assist the new hire with learning how to do the job to meet the employer's expectations, including nonwork-related tasks such as following procedures (i.e., upon arrival and departure, taking breaks, requesting time off, etc.) and working with others (i.e., getting to know coworkers, asking for help if needed, etc.) This support often involves providing the new hire with intensive on the job site skills training (including positive behavior support) that commences after both the new hire and employment specialist participate in

the employer's new employee orientation and training program. At this time, the employment specialist may also help the new hire complete his or her job responsibilities. This work completion guarantee is designed to keep an employer satisfied while the new hire is learning how to do the job. The employment specialist collects data to evaluate what the new hire has learned and the effectiveness of the training as well as design a schedule to fade his or her presence away from the job site. Overtime, as the new hire learns the job and job-related tasks the employment specialist is there less and less time. Eventually, the job seeker is working all or most of the time on his or her own. The employment specialist remains in touch with the new hire and employer and if indicated offers additional on the job skills training or support to the worker. In a holistic approach to implementing supported employment, the employment specialist is involved in all three phases of employment. In a partitioned approach, different employment specialists may only provide a support during a specific phase or phases of employment. Among other traits, an employment specialist should possess a 4-year degree in business, education, or related area and a valid driver's license and have excellent communication skills and the ability to independently solve problems.

See Also

- [Job Coach](#)
- [School to Work Transition Process](#)
- [Supported Employment](#)

References and Reading

- Targett, P. S., & Wehman, P. (2009). Integrated employment. In P. Wehman, M. D. Smith, & C. Schall (Eds.), *Autism and the transition to adulthood: Success beyond the classroom* (pp. 163–188). Baltimore: Paul H. Brookes Publishing.
- Wehman, P. (2012). *Life beyond the classroom*. Baltimore: Paul H. Brookes Publishing.
- Wehman, P., Inge, K. J., Revell, W. G., & Brooke, V. (2007a). *Real work for real pay: Inclusive employment for people with disabilities*. Baltimore: Paul H. Brookes Publishing.

- Wehman, P., Targett, P., & Young, C. (2007b). Off to work for individuals with Autism: A supported employment approach. *Autism Advocate*, 46(1), 54058.

Employment Trends for People with ASD

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Definition

In general terms, employment refers to the condition of being paid for work. It can also refer to someone's trade or profession. According to the US Department of Labor employment by definition is a little more complex. The US DoL collects *Current Employment Statistics (CES)*:

CES employment is an estimate of the number of nonfarm, payroll jobs in the U.S. economy. Employment is the total number of persons on establishment payrolls employed full- or part-time who received pay (whether they worked or not) for any part of the pay period that includes the 12th day of the month. Temporary and intermittent employees are included, as are any employees who are on paid sick leave, on paid holiday, or who work during only part of the specified pay period. A striking employee who only works a small portion of the survey period, and is paid, would be included as employed under the CES definitions. Persons on the payroll of more than one establishment are counted in each establishment. Data exclude proprietors, self-employed, unpaid family or volunteer workers, farm workers, and household workers. Persons on layoff the entire pay period, on leave without pay, on strike for the entire period, or who have a pending job but have not yet reported for work are not counted as employed. Government employment covers only civilian employees; it excludes uniformed members of the armed services. (For more information about CES employment, see the CES Technical Notes at www.bls.gov/web/empst/cestn.htm#section3a. (U.S. DoL 2017a))

The US Department of Labor Bureau of Labor Statistics (BLS) conducts the *Current Population*

Survey (CPS). Here they define who is counted as employed:

People are considered employed if they did any work at all for pay or profit during the survey reference week. This includes all part-time and temporary work, as well as regular full-time, year-round employment. Individuals also are counted as employed if they have a job at which they did not work during the survey week, whether they were paid or not, because they were:

- On vacation
- Ill
- Experiencing childcare problems
- On maternity or paternity leave
- Taking care of some other family or personal obligation
- Involved in a labor dispute
- Prevented from working by bad weather

These people are counted among the employed and tabulated separately as *with a job but not at work*, because they have a specific job to which they will return (U.S. DoL BLS 2015).

If these are the governmental definitions of employment, then what is a trend? A trend is a general direction toward which a phenomenon is moving. It may be thought of as a tendency, inclination, or drift (Merriam Webster 2017). Employment trends therefore are the general direction towards which the labor market is moving. Employment trend growth refers to "...the monthly rate of payroll job gains required to hold the unemployment rate constant at a level that is broadly consistent with the Federal Reserve's maximum employment mandate, also known as the natural rate of unemployment" (Bidder et al. 2016).

Unemployment by contrast is defined as the number of people looking for paid work that are unable to obtain a paid job. Who and how people are counted as unemployed by the federal government is important. According to the US DoL BLS:

People are classified as unemployed if they do not have a job, have actively looked for, work in the prior 4 weeks, and are currently available for work. Actively looking for work may consist of any of the following activities:

- Contacting:
 - An employer directly or having a job interview
 - A public or private employment agency

- Friends or relatives
- A school or university employment center
- Submitting resumes or filling out applications
- Placing or answering job advertisements
- Checking union or professional registers
- Some other means of active job search

Passive methods of job search do not have the potential to connect job seekers with potential employers and therefore do not qualify as active job search methods. Examples of passive methods include attending a job training program or course, or merely reading about job openings that are posted in newspapers or on the Internet. (U.S. Department of Labor Bureau of Labor Statistics 2015b)

Why are these definitions important? They are important to people on the autism spectrum because they help mask or underreport the issue of employment for people on the autism spectrum. No entity, not even the federal government, collects epidemiologically based survey data on individuals with autism. At best, the federal government collects data on individuals with disabilities through its *Current Population Survey*. This lumps together a wide assortment of disabilities varying in nature from physical disability to cognitive, intellectual, developmental, or mental illness disabilities. No recognition is given to the wide variability in the employability of individuals with a variety of conditions. Furthermore, it does not include individuals who are institutionalized, incarcerated, self-employed, or who have simply given up looking for work. Countless individuals on the autism spectrum live in congregate care situations and are not counted among individuals in the labor market. Another unknown number are the number of individuals on the autism spectrum who populate our jails and prisons in the United States. They too are not counted among the unemployed. According to the most recent government estimates:

Persons who are neither employed nor unemployed are not in the labor force. A larger proportion of persons with a disability – about 8 in 10 – were not in the labor force in 2016, compared with about 3 in 10 of those with no disability. (U.S. Department of Labor Bureau of Labor Statistics 2017a)

Not including self-employed individuals among labor trends underreports the number of individuals who are working for pay. Individuals with

disabilities are more likely than their nondisabled peers to be employed. In fact, people with disabilities are more likely to be self-employed than their nondisabled peers (10.6% vs. 6.2%) (U.S. Department of Labor Bureau of Labor Statistics 2015a). In 2015, 15 million people were self-employed which made up 10.1% of all US workers. However, self-employment has trended downward in the past two decades. This downward trend is attributed to less individuals working as self-employed in the agricultural sector of the economy (U.S. Department of Labor Bureau of Labor Statistics 2015b).

Historical Background

The history of Federal legislation in reference to employment extends as far back as the Smith Hughes Act of 1917 which provided matching monies for vocational educational programs. In 1918 World War I was the impetus for the Soldier's Rehabilitation Act which sought to train disabled veterans for employment and created programs to assist veterans only (History and Regulations –IN.gov 2017). No thought was given or monies provided to individuals who were either born disabled or became disabled through any other means.

It is only a relatively recent development that we have begun to include children with autism in our education system. This system is a training ground used by society to prepare young people to be the future workers in our economy. With the passage of the Education for All Handicapped Children Act (P.L. 94-142) in 1974, the federal government established that education was a right for all children. It also established core concepts such as the right of a child with a handicap to participate in a Free and Appropriate Public Education (FAPE) in the Least Restrictive Environment (LRE). Subsequent reauthorizations of P.L. 94-142 shifted the focus to person's first language with the passage of the Individuals with Disabilities Education Act (IDEA) and created concepts such as transition goals and plans with an emphasis upon employment for all children with disabilities. According to Carter et al.

(2012) “high quality transition services and supports have evolved over the last 25 years and that finding meaningful work after high school or college has become an essential outcome of education in the United States” (p. 50). In fact:

...the Individuals With [sic] Disabilities Education Improvement Act (IDEA) of 2004, which states that a central purpose of special education is to ‘prepare [students with disabilities] for further education, employment, and independent living’ as part of a national policy aimed at ‘ensuring equality of opportunity, full participation, independent living, and economic self-sufficiency for individuals with disabilities’ (Public Law 108-442). (Carter et al. 2012, p. 50)

In the 1970s Congress also passed the Rehabilitation Act of 1973 which amended the Vocational Rehabilitation Act of 1965. The Vocational Rehabilitation Act of 1965 provided extended evaluations to determine if “more severely handicapped individuals might benefit from vocational rehabilitation services.” The Rehabilitation Act was enacted to address the issue of discrimination in the workplace against people with disabilities across the board. The most salient aspect of this act was the creation of Title V which was a major piece of civil rights legislation for people with disabilities. Under Title V four sections were created that dealt with various aspects of disability and discrimination. Section 501 required nondiscrimination in hiring of handicapped individuals in federal government and required affirmative action plans for the hiring, placement, and promotion of such individuals. Section 502 has probably the most significant impact upon our physical environment by establishing the Architectural Barriers and Compliance Board. Section 503 prohibited discrimination against handicapped individuals in employment of federal contractor or subcontractors receiving \$2,500 or more in federal monies. This section also required affirmative action plans. Section 504 prohibits discrimination against handicapped persons in ANY federally supported or sponsored program which extended the laws reach to hospitals, schools, colleges, universities, and state welfare offices or any other organization that received federal funds (History and Regulations –IN.gov 2017).

The Higher Education Opportunity Act of 2008 (HEOA) changed the rules governing Title IV or the Federal Student Aid programs. Acknowledging the importance of post-secondary education and training in the acquisition of jobs, this act helped to create Comprehensive Transition and Post-secondary (CTP) programs at colleges and universities which already have approved Title IV programs. Students with Intellectual Disabilities (ID) and other significant cognitive impairments, including those on the autism spectrum, can now access Federal Student Aid by completing the Free Application for Federal Student Aid (FAFSA). The law, as currently written, only allows students with ID to receive grants if they are enrolled in an approved CTP (FinAid 2016; Finkel et al. 2010; VanBergeijk and Cavanagh 2012).

The Workforce Innovation Opportunity Act (WIOA) of 2012 amended several employment-related laws. Most notably this law directed state offices of vocational and rehabilitative services to allocate 15% of their budgets to transition aged youth who were at risk. This included youth with disabilities and extends the notion of youth and service provision beyond the age of 21, as is the case under IDEA, to age 24 under WIOA. Under this Act VR offices can fund programs that provide pre-employment skills that were previously not funded. Individuals with autism often desperately need pre-employment skills training in soft skills such as interviewing, how to dress appropriately for work, financial literacy, how to talk to supervisors, timeliness, travel training, etc. WIOA also emphasizes employment in integrated competitive employment settings and discourages employment in sheltered workshops for sub-minimum wage.

Current Knowledge

As of October 2017, the national unemployment rate for all Americans hovered around 4.2%. This is in stark contrast to the most recent employment data for individuals with disabilities. In 2016 the unemployment rate for individuals with disabilities across the board was 10.5% or more than

double the employment rate of the nondisabled (US Department of Labor, Bureau of Labor Statistics 2017b). The contrast becomes more apparent when one looks at the employment rates between these two groups. Only 17.9% of individuals with disabilities were employed as compared to 65.3% of nondisabled individuals. Individuals with disabilities are employed at a rate that is almost four times less than nondisabled persons. The jobless rate for individuals with disabilities was consistently higher than persons with no disability regardless of their educational attainment. In fact, according to the CPS:

Persons with a disability are less likely to have completed a bachelor's degree or higher than those with no disability. Among both groups, those who had attained higher levels of education were more likely to be employed than those with less education. Across all levels of education in 2016, persons with a disability were much less likely to be employed than were their counterparts with no disability. (U.S. Department of Labor Bureau of Labor Statistics 2017b)

Not only are individuals with disabilities more likely to be unemployed, they are also more likely to be underemployed. Individuals with disabilities are more likely to be employed part-time than their nondisabled peers. This precludes these individuals from making a living wage and from receiving full-time benefits such as health insurance and paid vacation. "Among workers with a disability, 34 percent usually worked part time in 2016, compared with 18 percent of those without a disability. The proportion of workers who were employed part time for economic reasons continued to be slightly higher among those with a disability than among those without a disability (6 percent versus 4 percent). These individuals were working part time because their hours had been cut back or because they were not able to find a full-time job" (U.S. Department of Labor Bureau of Labor Statistics 2017b).

The service sector of the economy is the most likely place an individual with a disability will find employment. In 2016, 21.3% of employed individuals with disabilities worked in the service sector compared to 17.6% of nondisabled persons. "Workers with a disability were more likely than those with no disability to work in production,

transportation, and material moving occupations (14.6%, compared with 11.6%). Persons with a disability were less likely to work in management, professional, and related occupations than those without a disability (31.7%, compared with 39.5%) (U.S. Department of Labor Bureau of Labor Statistics 2017b).

The one area in the economy where individuals with disabilities and nondisabled individuals were not statistically different was in reference to government employment. People with disabilities were employed in the same proportion than nondisabled employees (14.0% vs. 13.6%). “However, a smaller share of workers with a disability were employed as private wage and salary workers (75.4%), compared with those with no disability (80.1%), and a larger share were self-employed than were those with no disability (10.6% vs. 6.2%)” (U.S. Department of Labor Bureau of Labor Statistics 2017b).

In terms of autism-specific employment trends, Shattuck et al. 2012 found in their sample, “For youth with an ASD, 34.7% had attended college and 55.1% had held paid employment during the first 6 years after high school. More than 50% of youth who had left high school in the past 2 years had no participation in employment or education. Youth with an ASD had the lowest rates of participation in employment and the highest rates of no participation compared with youth in other disability categories.”

According to data analyzed from Wave 5 of the National Longitudinal Transition Study-2 (NLTS2) by Roux et al. (2013), “. . . Approximately one-half (53.4%) of young adults with an ASD had ever worked for pay outside the home since leaving high school, the lowest rate among disability groups. Young adults with an ASD earned an average of \$8.10 per h, significantly lower than average wages for young adults in the comparison groups, and held jobs that clustered within fewer occupational types. Odds of ever having had a paid job were higher for those who were older, from higher-income households, and with better conversational abilities or functional skills. . . Findings of worse employment outcomes for young adults with an ASD suggest that this population is experiencing particular difficulty

in successfully transitioning into employment” (Roux et al. 2013).

Other analyses using the NTL-2 data revealed that “The proportion of young adults with autism who had a job was comparable to that for young adults with deaf-blindness or with multiple disabilities, and far below the proportion of those with blindness, learning disabilities, or traumatic brain injuries” (Standifer 2012). This finding suggests that autism is a far more debilitating and complex disability than most of what the public imagines. This impacts even the highest functioning and most seemingly able individuals on the spectrum. For example, according to Standifer’s analysis, “On average, young adults with autism with a job earned 86% as much per hour as all young adults with disabilities with a job (\$8.90/h vs. \$10.40/h)” (Standifer 2012). Further, Standifer (2012) found, “Nearly half of employed young adults with autism earned less than \$7.25 an hour, twice the rate for all employed young adults with disabilities (44% vs. 22%).” His analysis also concluded that, “Nearly half of employed young adults with autism worked less than 20 hours a week, four times the rate for all employed young adults with disabilities (42% vs. 11%).” Even the “Average hours worked per week by young adults with autism was 36% lower than that for all young adults with disabilities (23.2 hrs. vs. 35.8 hrs.),” according to Standifer (2012). The final two facts of Standifer’s analysis really underscore the plight of individuals on the autism spectrum when it comes to employment: “The proportion of employed young adults with autism who were working full time (35 h or more per week) was one third that for all young adults with disabilities who had a job (26% vs. 71%),” and “The proportion of employed young adults with autism who worked in a sheltered workshop environment (only working with other people with disabilities) was seven times that for all employed young adults with disabilities (34% vs. 5%)” (2012). This makes it far more difficult for an individual on the spectrum to earn a living wage in order to be able to support themselves. This comparison, again, is in relation to peers with other types of disabilities and not nondisabled peers.

No blanket statement or trend can be made in reference to employment of individuals with an ASD. The manifestation of ASD is unique and individual. Consequently, each person on the spectrum needs to carefully consider his or her unique strengths, interests, and deficits. A key factor will be to realistically ascertain the level of education and or training the individual can successfully complete. After exploring each of these domains, individuals on the spectrum and their family members should explore the US Department of Labor Bureau of Labor Statistics publication known as the *Occupational Outlook Handbook*. This publication is updated on a quarterly basis and has the most up to date labor projections for the next 10 years. One feature of the *Handbook* that is particularly helpful is the Occupation Finder. This feature has an A–Z listing of the 818 federally recognized occupational titles. Readers can scroll down through the document to find a job title of interest or something close to their vocational interest by clicking on the job title itself; it will link the reader to similar job titles. In addition to the occupational titles, readers will learn the entry level educational requirements for the position, any on-the-job training requirements, projected number of new jobs, projected growth rate, and the previous year’s median pay.

In reviewing the *Occupational Outlook Handbook* some general statements can be made about employment trends over the next 10 years. The service industry is where the majority of jobs can be found. Specifically, the healthcare/eldercare will have the greatest amount of growth and the largest number of actual job openings. Working in some of these positions can be especially challenging for individuals on the autism spectrum because this directly abuts their difficulties in social communication, and dealing with variable working conditions. From Table 1, we can see that there are a variety of occupations that are in the health-care field and require various levels of education or training. Under the occupational title system, generally any position in the health-care field that has the word aide attached to it requires the lowest level of education or training. Frequently, this is a vocational certificate and/or

Employment Trends for People with ASD,
Table 1 Twenty fastest growing occupations with greatest growth rate 2014–2024

Occupation	Growth rate 2014–2024 (%)	2016 median pay (per year)
Wind turbine service technicians	108	\$52,260
Occupational therapy assistants	43	\$59,010
Physical therapist assistants	41	\$56,610
Physical therapist aides	39	\$25,680
Home health aides	38	\$22,600
Commercial divers	37	\$49,090
Nurse practitioners	35	\$100,910
Physical therapists	34	\$85,400
Statisticians	34	\$80,500
Ambulance drivers and attendants, except emergency medical technicians	33	\$23,850
Occupational therapy aides	31	\$28,330
Physician assistants	30	\$101,480
Operations research analysts	30	\$79,200
Personal financial advisors	30	\$90,530
Cartographers and photogrammetrists	29	\$62,750
Genetic counselors	29	\$74,120
Interpreters and translators	29	\$46,120
Audiologists	29	\$75,980
Hearing aid specialists	27	\$50,250

Source: Bureau of Labor Statistics, U.S. Department of Labor, *Occupational Outlook Handbook, 2016-17 Edition*, Fastest Growing Occupations, on the Internet at <https://www.bls.gov/ooh/fastest-growing.htm> (visited October 15, 2017)

completion of high school. Those positions in the health-care field, such as occupational or physical therapy assistants, require an associate degree level of education. Outside of the health-care field, cartographers and photogrammetrists are occupations that will have a 29% growth rate in the coming decade with a 2016 median annual

salary of \$62,750. This position, however, requires a bachelor degree level of training. For those individuals on the autism spectrum who are visually oriented and have a talent for pattern recognition, this occupation may be a suitable profession that pays a living wage. Some individuals on the spectrum have been employed by intelligence and military agencies in helping interpret satellite image data. For those individuals on the spectrum who are more quantitatively oriented and can complete either a bachelor's, master's or even Ph.D. level of education, operations research analysts' positions may provide meaningful and lucrative employment. Persons in this position use mathematical and analytical models and methods to help organizations identify and solve complex problems facing the organization in which they work.

What we know is that individuals on the autism spectrum who leave high school are more likely to be employed when several factors were taken into consideration beyond personal demographics. Graduating from high school, whether the student ever received career counseling in high school and whether the high school contacted postsecondary vocational training programs or potential employers are significant factors in participating in employment with this population (Chiang et al. 2013). Ohl et al. (2017) in their study of 254 adults with ASDs, found that participants who disclosed their ASD diagnosis were three times more to be employed when compared to individuals who did not disclose their diagnosis. They also found that education level was a significant predictor of employment status with higher levels of education being associated with employment. Pillay and Brownlow (2017) conducted a systematic literature review of 297 articles concerning postsecondary employment of young adults with ASDs. Of the 297, articles only eight studies met their full inclusion criteria. Pillay and Brownlow (2017) established four predictors of employment, namely – supported workplace intervention, ASD traits and behavioral intervention, functional independence intervention, and family advocacy intervention. The odds of gaining employment were greater for youth who received job placement services from their local

Vocational Rehabilitation Program; however, only 48% of youth with ASDs received such services according to Migliore et al. (2012). This group of researchers also found that postsecondary college services were the strongest predictors of better earnings, and yet, only 10% of their Vocational Rehabilitative Services data set received college-based services. Although not focused solely on autism per se, Carter et al. (Carter et al. 2012) found that: "Having held a paid, community-based job while still in high school was strongly correlated with post-school employment success. In addition, being male and having more independence in self-care, higher social skills, more household responsibilities during adolescence, and higher parent expectations related to future work were all associated with increased odds of employment after school for young adults with severe disabilities" (p. 50). A long-standing finding from Wehman (2001) contends that for students with disabilities even completing one college course significantly increases the odds that they will be employed. Furthermore, those students with a disability, broadly defined, who are able to complete a bachelor's degree are hired at virtually the same rate as their nondisabled peers. More recently, Wehman et al. (2013) conducted the first randomized clinical trial of vocational training as an intervention for transition aged youth on the autism spectrum. The control group was "business as usual condition" which meant that the students worked with their local school district and vocational rehabilitation office in an effort to find employment. Transition aged youth randomly assigned to the business as usual condition were employed at a rate of 6%. By contrast transition aged youth who received vocational training were employed at an 87.5% rate. These jobs were in a hospital setting and provided a living wage. The results suggest that for youth on the spectrum who are unable or not interested in pursuing a college degree, postsecondary employment training is an effective intervention in helping them gain employment. Moore and Schelling (2015) examining National Longitudinal Transition-2 study data found that students with intellectual disabilities, which included some students on the autism

spectrum, who attended postsecondary education programs, had positive employment outcomes when compared to individuals who did not attend such programs.

Future Directions

In order to fully understand the light of individuals on the autism spectrum, we need to collect epidemiologically based data in reference to employment. The Federal government by asking generic questions concerning disability is unable to determine how many individuals with ASDs are employed, underemployed, unemployed, or not even in the labor force. Small samples of nonrandomly selected participants in research studies give us a wide range of unemployment rates that may not represent the larger population.

A second area of study is one that examines the impact of Federal legislation upon the employment rates of people with ASDs. Namely, we need to examine how IDEA and the Individuals with Disabilities Education Improvement Act of 2004 (IDEIA), with its emphasis upon empirically based intervention techniques, transition goals, transition plans, and employment outcomes, have possibly improved the employment picture for the ASD population. Likewise, how has the Higher Education Opportunity Act of 2008 and the creation of Comprehensive Transition and Postsecondary (CTP) programs, (which provided access to Federal Financial Student Aid in the form of grants), affected the employment outlook for individuals on the spectrum and how this has affected the financial health of their families? The Workforce Innovation Opportunity Act (WIOA) of 2012 is the final piece of federal legislation we must evaluate in terms of its impact upon the employability of individuals on the spectrum.

A third area of study involves the efficacy of various postsecondary college-based or college affiliated transition program models. This future research must examine which program components are most effective in helping transition aged youth find and obtain gainful employment.

The research must examine not only academic course content and employment training but also assistance with social skills and social communication as well as independent living skills such as financial literacy skills, travel training, cooking, self-care, apartment management, and laundry.

A final area of future study should examine how professionals can capitalize upon the distinct but varied learning styles of individuals with ASDs. Grandin and Duffy (2004) identified these styles as “visual thinking (or thinking in pictures), music or higher math brain types, and verbal lists and language translator brains.” The challenge will be how professionals can first identify these learning styles and then match those styles to existing jobs in our economy in both the present and in the future.

See Also

Comprehensive Transition and Postsecondary (CTP) Programs
Federal Student Aid; <https://studentaid.ed.gov/sa/eligibility/intellectual-disabilities>
Free and Appropriate Public Education (FAPE)
Free Application for Federal Student Aid (FAFSA); <https://fafsa.ed.gov/>
Individuals with Disabilities Education Act (IDEA)
Individuals with Disabilities Education Improvement Act (IDEIA) of 2004
Occupational Outlook Handbook (OOH); <https://www.bls.gov/ooh/>
Workforce Innovation Opportunity Act (WIOA)

References and Reading

- Bidder, R., Mahedy, T., & Valletta, R. (2016). *FRBSF Economic Letter*: Trend Job Growth: Where's Normal? Federal Reserve Bank of San Francisco.
- Carter, E. W., Austin, D., & Trainor, A. A. (2012). Predictors of Postschool employment outcomes for young adults with severe disabilities. *Journal of Disability Policy Studies*, 23(1), 50–63.
- Chiang, H. M., Cheung, Y. K., Li, H., & Tsai, L. Y. (2013). Factors associate with participation in employment for high school leavers with autism. *Journal of Autism and Developmental Disorders*, 43, 1832–1842.

- FinAid. (2016). The history of Federal Student Aid. Retrieved from <http://www.finaid.org/educators/history.phtml>. 10 Oct 2016.
- Finkel, J., Anderson, H., & Shanley, J. L. (2010). *Eligibility of students with Intellectual Disabilities to receive Federal Student Aid: The FAFSA and more!* U.S. Department of Education: State of the Art Conference. Washington, DC. 28 Oct 2010.
- Grandin, T., & Duffy, K. (2004). *Developing talents: Careers for individuals with Asperger syndrome and high functioning autism*. Shawnee Mission: Autism Asperger Publishing Company.
- Higher Education Opportunities Act of 2008. (P.L. 110–315).
- History & Regulations –IN.gov. Legislative History of the American State-Federal Vocational Rehabilitative (VR) Program. Retrieved from: https://www.in.gov/fssa/files/History_and_Regulations.pdf. 17 Oct 2017.
- Merriam Webster. (2017). Trend. Definition of Trend. Retrieved from <https://www.merriam-webster.com/dictionary/trend>. 9 Oct 2017.
- Migliore, A., Timmons, J., Butterworth, J., & Lugas, J. (2012). Predictors of employment and postsecondary education of youth with autism. *Rehabilitation Counseling Bulletin*, 55(3), 176–184. <https://doi.org/10.1177/0034355212438943>.
- Moore, E. J., & Schelling, A. (2015). Postsecondary inclusion for individuals with intellectual disabilities and its effects on employment. *Journal of Intellectual Disabilities*, 19, 130.
- Ohl, A., Grice Sheff, M., Small, S., Nguyen, J., Paskor, K., & Zanjirian, A. (2017). Predictors of employment status among adults with autism Spectrum disorder. *Work*, 56(2), 345–355. <https://doi.org/10.3233/WOR-172492>.
- Pillay, Y., & Brownlow, C. (2017). Predictors of successful employment outcomes for adolescents with autism Spectrum disorders: A systematic literature review. *Journal of Autism and Developmental Disorders*, 47(1), 1–11.
- Roux, A. M., Shattuck, P. T., Cooper, B. P., Anderson, K. A., & Narendorf, S. C. (2013). Postsecondary employment experiences among young adults with an autism Spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 52(9), 931–939.
- Shattuck, P. T., Narendorf, S. C., Cooper, B., Sterzing, P. R., Wagner, M., & Lounds Taylor, J. (2012). Postsecondary education and employment among youth with an autism Spectrum disorder. *Pediatrics*, 129, 1042–1049. <https://doi.org/10.1542/peds.2011-2864>.
- Standifer, S. (2012). *Fact Sheet on Autism Employment*. Autism Works. The National Conference on Autism Employment. Retrieved from <http://autismhandbook.org/images/5/5d/AutismFactSheet2011.pdf>. 15 Oct 2017.
- The Workforce Innovation and Opportunity Act (WIOA). (H.R. 803).
- U.S. Department of Labor Bureau of Labor Statistics. (2015a). *Self-employment in the United States: Spotlight on statistics*. <https://www.bls.gov/spotlight/2016/self-employment-in-the-united-states/home.htm>.
- U.S. Department of Labor Bureau of Labor Statistics. (2015b). *Labor Force Statistics from the Current Population Survey*. Who Is Counted as Employed? Retrieved from: <https://www.bls.gov/cps/faq.htm#Ques4>. 9 Oct 2017.
- U.S. Department of Labor Bureau of Labor Statistics. (2017a). *Current Employment Statistics (CES)*. How Does the CES define employment? Retrieved from <https://www.bls.gov/web/empstat/cesfaq.htm>. 9 Oct 2017.
- U.S. Department of Labor Bureau of Labor Statistics. (2017b). *Economic news release*. Persons with a disability: Labor force characteristics summary – 2016. Retrieved from: <https://www.bls.gov/news.release/disabl.nr0.htm>. 9 Oct 2017.
- VanBergeijk, E. O., & Cavanagh, P. K. (2012). Federal Student aid for students with intellectual disabilities: A well-kept secret? *Parenting Special Needs Magazine* (pp. 64–65). <http://www.mydigitalpublication.com/publication/?i=111307&l=&m=&p=8&id=5779>.
- Wehman, P. H. (2001). *Life beyond the classroom: Transition strategies for young people with disabilities* (3rd ed.). Baltimore: Paul H. Brookes Publishing.
- Wehman, P. H., et al. (2013). Competitive employment for youth with autism Spectrum disorders: Early results from a randomized clinical trial. *Journal of Autism and Developmental Disorders*, 44, 487. <https://doi.org/10.1007/s10803-013-1892-x>.

Enactive Mind

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Synonyms

Enactive mind hypothesis

Definition

This theoretical model for understanding social difficulties in autism was inspired by the major gap typically observed in performance of

cognitively able individuals with ASD in their ability to correctly respond to explicit tasks of social reasoning and their typical major inability to perform in naturalistic social situations (Bowler 1992; Dahlgren and Trillingsgaard 1996). This inability is reflected pervasively in daily life and is a source of major disability (Klin et al. 2000). It is reflected in standard measures of social competence, e.g., in adaptive behavior as assessed by the Vineland (Volkmar et al. 1987).

This discrepancy has significant impact on activities of work and daily living. One solution for such individuals is often an overreliance on routine and structure avoiding novelty and, as much as possible, the inherent unpredictability of social life. This solution often leads to significant social isolation and loneliness for even the most cognitively able persons inspired by a series of studies focusing on naturalistic social situations (e.g., Klin et al. 2003). Klin and colleagues proposed that the discrepancy in performance between naturalistic and highly structured situations may be even greater than what was originally believed. Their work with eye tracking revealed major and profound differences in observation of social situations with critical elements of social scenes frequently missed entirely. The inability to encompass the quickly changing social panorama and rapidly select the most salient features of a scene before responding was dramatic and illustrated the need for new conceptualizations of these difficulties.

In their model Klin and colleagues (2003) provided an alternative to computational models of social-cognitive development. The enactive mind (EM) approach was based on emerging embodied cognitive neuroscience. In the EM model, cognition is seen as embedded in experience that results from the body's action on the relevant and salient aspects of the environment. Thus, social cognition is viewed as embedded in social interaction. These experiences become tools for social adaptation that can also be abstracted symbolically and used for explicit reassigning about social phenomena.

In autism, the EM approach suggests that these preceding processes are derailed given that the typical primary salience of social stimuli

is not present and is replaced, de facto, by an overreliance on the physical world. This pattern thus shapes phenomena like joint attention, incidental learning, social communication, and so forth. Put another way it leads young children with autism to become experts on things rather than on people. Thus, even for the most cognitively able individual the lack of reliance on embedded personal experience resulting in major difficulties in ever-changing naturalistic social situations. The EM approach contends that without the set of embodied social-cognitive tools interaction in such situations becomes halting, slow, and inefficient.

This approach emphasizes that individuals with ASD can learn about people but in a way that is not the normative model of social learning. As a result, their knowledge can be highly sophisticated in many respects in the abstract but highly limited in realistic interactions. This approach suggests the importance of studying compensatory pathways and approaches that move beyond highly structured laboratory situations into more naturalistic ones.

See Also

- [Brain Connectivity Theories of Autism](#)
- [Theory of Mind](#)
- [Weak Central Coherence](#)

References and Reading

- Bowler, D. M. (1992). Theory of mind "in Asperger's syndrome". *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 33(5), 877–893.
- Dahlgren, S. O., & Trillingsgaard, A. (1996). Theory of mind in non-retarded children with autism and Asperger's syndrome. A research note. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 37(6), 759–763.
- Klin, A., Schultz, R., & Cohen, D. J. (2000). *Theory of mind in action: Developmental perspectives on social neuroscience. Understanding other minds: Perspectives from developmental cognitive neuroscience* (pp. 357–388). New York: Oxford University Press.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., Cohen, D., Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002a). Defining and quantifying the social

phenotype in autism. [see comment]. *American Journal of Psychiatry*, 159(6), 895–908.

Klin, A., Jones, W., Schultz, R., Volkmar, F., Cohen, D., Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002b). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809–816.

Klin, A., Jones, W., Schultz, R., Volkmar, F., Klin, A., Jones, W., Schultz, R., & Volkmar, F. (2003). The enactive mind, or from actions to cognition: Lessons from autism. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 358(1430), 345–360.

Volkmar, F. R., Sparrow, S. S., Goudreau, D., Cicchetti, D. V., et al. (1987). Social deficits in autism: An operational approach using the Vineland Adaptive Behavior Scales. *Journal of the American Academy of Child & Adolescent Psychiatry*, 26(2), 156–161.

Enactive Mind Hypothesis

► [Enactive Mind](#)

Encephalography

► [Pneumoencephalography](#)

Enclave

► [Mobile Work Crew Model](#)

Encopresis

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Synonyms

[Fecal incontinence](#); [Soiling](#)

Short Description or Definition

Encopresis, as defined by the DSM-IV-TR (American Psychiatric Association [APA] 2000), consists in the repeated passage of feces in inappropriate places. Although soiling is usually involuntary, it may be intentional in some cases. Soiling must occur once a month for at least 3 months and the chronological age of the child must be at least 4 years. In the case of children with developmental delays, a mental age of at least 4 years is required in order to characterize for a diagnosis of encopresis. Any physical disorder, explaining incontinence, must be ruled out and the soiling should not be due to the direct effects of a substance (e.g., laxatives).

Categorization

Encopresis can be categorized as primary or secondary. In the case of primary encopresis, the fecal continence has never been obtained; and in secondary encopresis, there has been a period of fecal continence (defined as having reached continence for at least a period of 12 months) preceding the recurrence of soiling.

The DSM-IV-TR describes two subtypes of encopresis:

- Encopresis with constipation and overflow incontinence, which has been referred in the literature as “retentive encopresis.” On physical examination there is evidence of large stool mass on abdominal or rectal examination and a history of stool frequency of less than three per week. Usually, there are periods of retention which last several days, followed by a painful expulsion, and then the period of retention starts again.
- Encopresis without constipation and overflow incontinence, which has been referred in the literature as “non-retentive encopresis.” Here, there is no evidence of constipation on physical examination or by history, and the feces are likely to be of normal consistency. Here the main characteristic is the lack of psychological, physiologic, or combined control of the expulsion.

Epidemiology

It is estimated that 1% of 5-year-old children have encopresis, and the disorder is more frequent in males (APA 2000).

There are different studies involving children of different ages, with a prevalence varying between 1.5% and 4.1% and a male-female ratio of 3:1. The prevalence of encopresis is higher among younger children (Bellman 1996; Mikkelsen 2007; Van Der Wall et al. 2005).

Special Characteristics of Encopresis in Children with Autism

The figures reported before pertain to the general population. In children with autism, there are just a few studies to review. Data collected from these studies suggest that constipation in this population has a behavioral etiology, rather than a primary organic gastrointestinal etiology. The overall incidence of gastrointestinal symptoms among children with autism did not differ from children without the disorder (Ibrahim et al. 2009). Children with autism have an increased incidence of difficulties, regarding not only constipation but also feeding behavior and food selectivity, which are understood as related to behavioral problems, rather than to organic gastrointestinal pathology (Ibrahim et al. 2009).

Natural History, Prognostic Factors, and Outcomes

There have been several studies (Mikkelsen 2007; Loening-Baucke 1987; Loening-Baucke et al. 1987) to find out the basis for the encopresis. It seems clear that retentive encopresis has a direct relationship with abnormal contractions and an inability to relax the external sphincter. Even more, this problem very often generates a constipation problem, with painful defecation, which leads again to retention, making the problem bigger and bigger. The consistency of the stool becomes harder, making the process more painful and difficult. And the child avoids defecation, until leakage occurs.

In the non-retentive encopresis, psychological issues such as oppositional defiant or conduct disorder can be identified. Also, in the case of developmental disabilities, one can suspect that proper toileting behavior has never been mastered.

Clinical Expression and Pathophysiology

Constipation and functional fecal retention (FFR) have been included as causes leading to the development of retentive encopresis. It should include a period of a minimum of 12 weeks of constipation history, large stools, abdominal pain, soiling, and the need to use laxatives or enemas to treat the problem.

It seems clear that constipation and painful defecation are associated with stool toileting refusal (STR), and in most cases, hard and/or painful bowel movements occur before the onset of STR. Therefore, an effective treatment for constipation may be an option to decrease the duration and/or the incidence of STR (Blum et al. 2004). Constipation is a frequent and significant problem that happens in children suffering from autism. It is difficult to recognize its impact and how it functionally affects the rectosigmoid colon. It is often associated with megarectum. Since there is no significant correlation between the degree of fecal impaction and the clinical history, the importance of an abdominal radiography during the assessment, to evaluate the degree of constipation, is essential (Afzal et al. 2003).

Behavioral problems may be the primary or sole symptom of the underlying medical condition (Buie et al. 2010).

Evaluation and Differential Diagnosis

Soiling can be an original problem or a symptom of a different cause. Therefore, it is important to rule out possible physical or psychological causes. In typically developed children, there may be times when soiling and/or wetting accidents can occur, and if these are repetitive, the clinician must identify possible coincidental

stresses. Usually the problem remits when the stressor is removed. Other times, there may be physical or medical causes such as Hirschsprung disease, stenosis of the rectum, endocrine abnormalities, and other medical conditions that must be ruled out. Also, for a variety of different reasons, other problems such as attention deficit hyperactive disorder (ADHD), oppositional defiant disorder (ODD), and, again, developmental disorders like mental retardation or autistic spectrum disorders (ASD) can be associated with encopresis.

Soiling in children suffering from an autistic spectrum disorder (ASD) can include all the physical and psychological causes that must be considered in typically developing children. Peculiar aspects playing a role in ASD may include:

- Rigidity that leads where and when they defecate, leading to constipation
- Refusal to seat on the toilet
- Absence of appropriate learning of the toilet use
- Diet problems, leading to constipation
- Soiling challenging behavior, such as smearing feces and playing with them
- Social cognition limitations hampering their understanding about using toilets and when and how the toilet should be used
- Sensory anomalies involving olfactory, tactile sensations, or even the noise produced by the toilet that can generate refusal among ASD children

It is important to find out what are the possible one or more involved factors and include them into a coherent treatment plan.

Treatment

Once medical problems have been ruled out, we should check for psychological/behavioral explanation of the problem and treat accordingly.

Treating Anxiety Symptoms

We should keep a very close attention to anxiety symptoms that could lead to suffering and

avoidance of toilet use. We should encourage the use of the toilet in a positive and progressive way, coupling the toilet situation with rewarding and pleasant stimuli. Since changing routines is difficult for these children, they will need more time and effort to adapt to this challenging task.

Minimizing Understanding Difficulties

When giving tips or messages regarding the use of the toilet, it is important to ensure proper understanding. Therefore, the use of augmentative communication systems, using, for example, gestures, objects, pictograms, or pictures, may be quite helpful.

Treating Physical Causes

If poor diet is the problem leading to constipation, the bowel may be blocked with hard stools, painful to pass, and that may even cause fissures in the perianal area. The child, therefore, will try to avoid passing them because it is painful and the stools will become harder and may even further challenge passage through the bowel. There is usually leaking through the passage that stains the underpants. In children suffering from ASD, the fact that their clothes get stained may not be an issue for them, due to social unawareness, but there will be negative feedback obtained from peers or non-informed adults.

Measures to resolve this problem may be increasing high-fiber foods such as bran, whole wheat products, vegetables, and fruits or even increasing the liquids in the diet adding water or juices. Also the reduction in constipating foods like bananas, dairy, and peanuts can help. Most people react with a gastrocolic reflex, by feeling the need to defecate just after having a meal; and this can be used as a simple mechanism to favor appropriate defecation.

These general dieting advices may be important when diapers are removed for the first time and training is attempted.

Treating Sensory Problems

The physical structure of the toilet is very important and involves “isolating” the place (avoiding distracters) and the behavior (sits on the toilet to defecate or urinate only). It is not easy to assign a half-bath in every house, but if possible it can be

very helpful, to avoid distractions and confusing issues. Creating a secure and not overstimulating environment is important, to make the child more relaxed and comfortable. Therefore, think of the plumbing noises, echoes, and lights. Even playing soft music can help.

Treating Rigidity Problems

Establishing a visually supported routine can be crucial. We must create a visual sequence of steps that help the child to complete the goal. Since children presenting ASD have preferences for routines or rituals, we can create new ones to make a bridge between the old and the new ones. We could start with a transition object (a photograph, a picture, or an object of the toilet) that serves to initiate the bathroom routine. Once the transition to the toilet has been made, it is important to continue with the visual aids to visually support each step of the toileting routine. Those aids can be placed in a ziplock bag or glued shut.

Treating Refusal to sit on the Toilet

When resisting to sit on the toilet is an issue, we must break the target into as many steps as needed. Sometimes these children have motor planning difficulties, and although they may know the next step, they seem unable to physically move themselves. Therefore, we should physically help them to initiate the movement. Other times we may use desensitization, a behavioral technique useful to help the child decrease the fears over different situations such as sitting on a toilet and the noise of flushing it, step by step. For example, tips like sitting on the toilet without removing the clothes, using timers, using the length of one song on the CD player, entertaining the child, taking turns sitting on the toilet, and using dolls for modeling can be helpful. The adult must recognize that it may take time for the child to extinguish his or her fears. We also should be aware that they have difficulties imitating and generalizing behaviors.

If flushing the toilet is an issue because the child is afraid of it, one can try starting the flush while the child is away from the toilet and gradually help him or her to get closer. Give warning in advance. Show him how to do it and make a

“game” of it. If the problem is the opposite, and flushing is an intense interest, use visual sequence to show the child when to flush, and what follows afterward (washing hands, lights off, leave toilet), to help him to get distracted from this interest.

Treating Social Difficulties

The use of social stories about the use of the toilet can also help. By them, children with ASD can understand when and how to use a toilet, who can support them if they need help, how to ask if they need using the toilet, and where to locate one when they are not at home.

Treating Other Difficulties

There may be other difficulties such as playing with the toilet paper or even resisting being cleaned that need to be tackled. In those cases one can try using Kleenex, wet wipes, or a sponge. At the same time, we should reinforce all the desired behaviors such as spending more time sitting on the toilet, flushing it without fear, or doing so for the appropriate length of time.

When planning a structured program for potty training in children with ASD, three basic components should be covered: *order*, *predictability*, and *routine*. Each program has to be individualized, and we should not forget that potty training is a process, not an event. It should include visual supports, social stories, and very important individualized personal potty stories.

For parents it is also important, before beginning with the plan, to write the reasons that have caused them anxiety over potty training and understand why training has not been successful in the past. It is useful for them to anticipate any obstacles that will be found and consider ways to overcome them. Also it should help to write the strategies to be followed and think about the benefits generated when potty training is achieved.

It is mandatory to keep track of the progress (and difficulties) and to share them with the clinician or educator. First, a baseline observation time recording the time or times in the day where the “accident” takes place, and then, most people find that it is useful to consider this “likely times” and proceed before they happened, using the previously mentioned tips.

The investment in this training sooner or later pays back, and this achievement helps the dignity, autonomy, health, and social status of the child with previous encopresis.

See Also

- [Constipation](#)
- [Toilet Training](#)

References and Reading

- Afzal, N., Murch, S., Thirrupathy, K., Berger, L., Fagbemi, A., & Heuschkel, R. (2003). Constipation with acquired megarectum in children with autism. *Pediatrics*, 112, 939–942.
- American Psychiatric Association. (2000). Elimination disorders. In American Psychiatric Association (Ed.), *DSM-IV-TR diagnostic and statistical manual of mental disorders*. Text revision (4th ed., pp. 116–118). Washington, DC: Author.
- Batts, B. (2010). *Ready, set potty! Training for children with autism and other developmental disorders*. London/Philadelphia: Jessica Kingsley.
- Bellman, M. (1996). Studies on encopresis. *Acta Paediatrica Scandinavica*, 56(Suppl 170), 1–151.
- Blum, N. J., Taubman, B., & Nemeth, N. (2004). During toilet training, constipation occurs before stool toileting refusal. *Pediatrics*, 113, e520–e522.
- Buie, T., Campbell, D. B., Fuchs, G. J., Furuta, G. T., Levy, J., VandeWater, J., et al. (2010). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125(Suppl), S1–S18. <https://doi.org/10.1542/peds.2009-1878C>.
- Gray, C., & White, A. L. (2002). *My social stories book*. London/New York: Jessica Kingsley.
- Ibrahim, S. H., Voigt, R. G., Katusic, S. K., Barbaresi, W. J., & Weaver, A. L. (2009). Incidence of gastrointestinal symptoms in children with autism: A population based study. *Pediatrics*, 124, 680–686. <https://doi.org/10.1542/peds.2008-2933>.
- Loening-Baucke, V. A. (1987). Factors responsible for persistence of childhood constipation. *Journal of Pediatric Gastroenterology and Nutrition*, 6, 915–922.
- Loening-Baucke, V., Cruikshank, B., & Savage, C. (1987). Defecation dynamics and behavior profiles in encopretic children. *Pediatrics*, 80, 672–679.
- Mikkelsen, E. J. (2007). Elimination disorders: Enuresis and encopresis. In A. Martin & F. R. Volkmar (Eds.), *Lewis child and adolescent psychiatry. A comprehensive textbook* (4th ed., pp. 662–665). Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins.
- Parenting and Child Health. <http://www.cyh.com/HealthTopics/HealthTopicdetails.aspx>. Last revision 14 Mar 2011.
- Strategies for Dealing with Soiling (Encopresis). <http://www.autism-help.org/behavior-soiling-encopresis.htm>. Last revision 14 Mar 2011.
- Van Der Wall, M. F., Benninga, M. A., & Hirasing, R. A. (2005). The prevalence of encopresis in a multicultural population. *Journal of Pediatric Gastroenterology and Nutrition*, 40(Suppl 3), 345–348.
- Wheeler, M., & Kranowitz, C. S. (2007). *Toilet training for individuals with autism or other developmental disorders*. Arlington: Future Horizons.

Endep

- [Elavil \(Amitriptyline\)](#)

Endep (Amitriptyline)

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Synonyms

[Amitriptyline hydrochloride](#)

Definition

Endep is an antidepressant. The drug works on the brain by increasing the level of neurotransmitter chemicals, norepinephrine and serotonin, and blocking acetylcholine. While there is some evidence of its effectiveness with symptoms accompanied by hyperactivity, impulsivity, aggression, and self-injury of treatment-resistant youth with ASD at low doses, further research is required. Endep can be used for bed-wetting, provided that there are no primary bladder issues. Side effects may include weight gain, dry mouth, nausea, and constipation. Clinicians must perform quarterly EKG to evaluate heart irregularities.

See Also

► [Antidepressants](#)

References and Reading

- Bhatti, I., Thome, A., Smith, P. O., Cook-Wiens, G., Yeh, H. W., Gaffney, G. R., & Hellings, J. A. (2013). A retrospective study of amitriptyline in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(5), 1017–1027.
- Hurwitz, R., Blackmore, R., Hazell, P., Williams, K., & Woolfenden, S. (2012). Tricyclic antidepressants for autism spectrum disorders (ASD) in children and adolescents. *The Cochrane Database of Systematic Reviews* 3(3), CD008372.

Endocrine

► [Oxytocin](#)

Endophenotypes

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Definition

A phenotype is a characteristic of an organism or individual that can be observed. In psychiatry, the term is often used to refer to a set of behaviors that constitute a categorical diagnosis. An endophenotype is a measureable characteristic that exists somewhere along the biological pathway from gene risk to the overall phenotype or categorical diagnosis. It is a characteristic that may reflect an underlying or component feature of a disorder, such as autism. The characteristic can be of many types, including biochemical, neurophysiological, neuroanatomical, and cognitive (Viding and Blakemore 2007). It can be a subset of the clinical symptoms but not the full syndrome itself. For example, “age at first word”

has been proposed as an autism endophenotype (Alarcón et al. 2002). While it clearly relates to language disability, it is not the entire autism phenotype. Some terms have similar meanings but do not focus on the genetic connection. These include “intermediate phenotype,” “biological marker,” “subclinical trait,” and “vulnerability marker” (Gottesman and Gould 2003).

Historical Background

The term endophenotype was introduced into the psychiatric lexicon in a 1972 book on the genetics of schizophrenia by Irving Gottesman and James Shields (1972). They borrowed the term from a paper by John and Lewis who had used it to describe concepts in evolution and insect biology (Lewis 1966). Gottesman and Shields proposed the endophenotype concept in an effort to speed discovery of genes that contributed to neuropsychiatric disorders, especially Schizophrenia (Gottesman and Gould 2003).

The idea was that the complexity of neuropsychiatric behavioral phenotypes would likely result from the dysfunction of a greater number of genes than would less complex endophenotypes of these disorders (Gottesman and Gould 2003). The discovery of genes that contributed to endophenotypes, therefore, would help speed gene discovery in neuropsychiatric disorders both because the genes that contributed to endophenotypes would presumably also contribute to the disorder and because the genetic research and analysis involved in the discovery of genes resulting in an endophenotype (with fewer genes contributing) would be easier than the discovery of genes resulting in a full-blown neuropsychiatric phenotype (with multiple genes contributing).

In 2003, Gottesman and Gould published a paper updating and further elucidating the endophenotype concept in psychiatry (Gottesman and Gould 2003). They proposed the following five principles, some of which were adapted from those suggested by others, that they state make an endophenotype useful for genetic research:

1. The endophenotype is associated with illness in the population.
2. The endophenotype is heritable.
3. The endophenotype is primarily state-independent (manifests in an individual whether or not illness is active).
4. Within families, endophenotype and illness co-segregate, (meaning that those with the endophenotype have the illness and those without it do not have the illness).
5. The endophenotype found in affected family members is found in nonaffected family members at a higher rate than in the general population (Gottesman and Gould 2003).

As pointed out by Gottesman and Gould, the endophenotype concept lay dormant and largely unutilized from the time of their introduction of the concept to the psychiatric literature in the 1970s until the start of the twenty-first century (Gottesman and Gould 2003). However, in the new century, researchers began to utilize the concept significantly more frequently. Gottesman and Gould attribute this change to the lack of success of numerous genetic studies utilizing phenotypes as opposed to endophenotypes in discovering genes that contributed to neuropsychiatric disorders despite significant investments and large-scale projects (Gottesman & Gould). The slowness of the progress was certainly evident in research into the genetics of autism (El-Fishawy and State 2010). Regardless of the reason, the use of endophenotypes proliferated substantially after the year 2000 in neuropsychiatric research in general and in genetic research in autism in particular, as described below (Gottesman and Gould 2003).

Current Knowledge

The endophenotype concept has been used in multiple studies seeking to identify genes contributing to autism spectrum disorders. For example, a study by Alarcon et al. linked a region on chromosome 7 to an autism endophenotype, “age at first word” (Alarcón et al. 2002). Subsequently, this group found evidence for association of this

endophenotype to a specific gene in this interval called contactin-associated protein-like 2 (CNTNAP2). A series of subsequent studies have pointed to the involvement of CNTNAP2 in language development and autism (Abrahams and Geschwind 2010).

Multiple other endophenotypes have been explored with regard to autism genetics. For example, there has been a long-standing interest in the observation of increased platelet serotonin levels in individuals with ASD (Anderson et al. 1987) and whether this might serve as a useful endophenotype for gene discovery (Freitag 2006). Similarly, enlarged head size, or macrocephaly, has been demonstrated in a subset of autistic children (Sacco et al. 2007). In this regard, rare cases of mutations in the gene *PTEN* (*phosphatase and tensin homolog*) have been found in individuals with macrocephaly and autism. These findings have been robust enough to warrant the recommendation that individuals with these features should have a genetic evaluation including sequencing of this gene.

Quantitative scales have also been developed focusing on identifying potential endophenotypes in autism. For instance, scores on the Social Responsiveness Scale (Constantino and Todd 2005), aimed at measuring the severity of social impairment, have been shown to be heritable and continuously distributed in the population, with parents of autistic children showing significant shifts toward pathological scores (Constantino and Todd).

Future Directions

Several avenues of autism research have characterized traits that could potentially serve as novel endophenotypes. For example, eye-tracking experiments and functional neuroimaging have identified potential signatures of autism risk. Simultaneous advances in genomic technologies will increasingly allow these to be explored.

Importantly, as specific risk genes and regions have been identified based on the categorical diagnosis of ASD, it has become clear that these

genetic factors also increase the liability for a wide range of disorders that were previously conceptualized as being distinct. For example, duplications of a section on the short arm of chromosome 16, 16p11.2, have been shown to increase the risk for both autism and schizophrenia. Studies are just now being undertaken examining whether these and similar findings may, in part, reflect shared endophenotypes among disparate clinical syndromes (State and Levitt 2011).

See Also

- ▶ Biological Motion
- ▶ Candidate Genes in Autism
- ▶ DNA
- ▶ Event-Related Functional Magnetic Resonance Imaging (MRI)
- ▶ Genetics
- ▶ Social Responsiveness Scale

References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2010). Connecting genes to brain in autism spectrum disorders. *Archives of Neurology*, 67(4), 395–399.
- Alarcón, M., Cantor, R. M., et al. (2002). Evidence for a language quantitative trait locus on chromosome 7q in multiplex autism families. *American Journal of Human Genetics*, 70(1), 60.
- Alarcon, M., Yonan, A. L., et al. (2005). Quantitative genome scan and ordered-subsets analysis of autism endophenotypes support language QTLs. *Molecular Psychiatry*, 10(8), 747.
- Alarcón, M., Abrahams, B. S., et al. (2008). Linkage, association, and gene-expression analyses identify CNTNAP2 as an autism-susceptibility gene. *American Journal of Human Genetics*, 82(1), 150.
- Anderson, G. M., Freedman, D. X., et al. (1987). Whole blood serotonin in autistic and normal subjects. *Journal of Child Psychology and Psychiatry*, 28(6), 885–900.
- Arolt, V., Lencer, R., et al. (1996). Eye tracking dysfunction is a putative phenotypic susceptibility marker of schizophrenia and maps to a locus on chromosome 6p in families with multiple occurrence of the disease. *American Journal of Medical Genetics*, 67(6), 564–579.
- Butler, M. G., Dasouki, M. J., et al. (2005). Subset of individuals with autism spectrum disorders and extreme macrocephaly associated with germline PTEN tumour suppressor gene mutations. *Journal of Medical Genetics*, 42(4), 318.
- Conciatori, M., Stodgell, C. J., et al. (2004). Association between the HOXA1 A218G polymorphism and increased head circumference in patients with autism. *Biological Psychiatry*, 55(4), 413–419.
- Constantino, J. N., & Todd, R. D. (2005). Intergenerational transmission of subthreshold autistic traits in the general population. *Biological Psychiatry*, 57(6), 655–660.
- Duvall, J. A., Lu, A., et al. (2007). A quantitative trait locus analysis of social responsiveness in multiplex autism families. *The American Journal of Psychiatry*, 164(4), 656.
- El-Fishawy, P., & State, M. W. (2010). The genetics of autism: Key issues, recent findings, and clinical implications. *The Psychiatric Clinics of North America*, 33(1), 83–105.
- Freitag, C. M. (2006). The genetics of autistic disorders and its clinical relevance: A review of the literature. *Molecular Psychiatry*, 12(1), 2.
- Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *The American Journal of Psychiatry*, 160(4), 636–645.
- Gottesman, I., & Shields, J. (1972). *Schizophrenia and genetics: A twin study vantage point*. New York: Academic.
- Kaiser, M. D., Hudac, C. M., et al. (2010). Neural signatures of autism. *Proceedings of the National Academy of Sciences*, 107(49), 21223.
- Keating, M. T., & Sanguinetti, M. C. (2001). Molecular and cellular mechanisms review of cardiac arrhythmias. *Cell*, 104, 569–580.
- Keating, M., Atkinson, D., et al. (1991). Linkage of a cardiac arrhythmia, the long QT syndrome, and the Harvey ras-1 gene. *Science*, 252(5006), 704.
- Klin, A., Lin, D. J., et al. (2009). Two-year-olds with autism orient to non-social contingencies rather than biological motion. *Nature*, 459(7244), 257–261.
- Lewis, J. (1966). Chromosome variability and geographical distribution in insects: Chromosome rather than gene variation provide the key to differences among populations. *Science*, 152, 711–772.
- Matthysse, S., Holzman, P. S., et al. (2004). Linkage of eye movement dysfunction to chromosome 6p in schizophrenia: Additional evidence. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 128(1), 30–36.
- Sacco, R., Militeri, R., et al. (2007). Clinical, morphological, and biochemical correlates of head circumference in autism. *Biological Psychiatry*, 62(9), 1038–1047.
- State, M. W., & Levitt, P. (2011). The conundrums of 109 understanding genetic risks for autism spectrum disorders. *Nature Neuroscience*, 14(12), 1499–1506.
- Viding, E., & Blakemore, S. J. (2007). Endophenotype approach to developmental psychopathology: Implications for autism research. *Behavior Genetics*, 37(1), 51–60.

England and Autism

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The history of Autism is a discourse (Waltz 2013), a journey through a disputed landscape, whose territories are alternatively staked by Politics, Education, Society, and Culture. It is diachronic in nature, as the knowledge of the present is built upon the past, but a diachronic that has progressed differently in different states, at different rates as each impact upon each other. Essentially its origins are lost in myth (Frith 1992) but its presence has always been felt in one way or another, even before the concept of autism was framed in the Western psychiatric narrative.

It is important for the continued understanding of the position that autism occupies as a “phenomenon” in England to restore the emphasis of the narrative to the cultural landscape as this is the day-to-day experience of people living and working with this contested construct and category of humanity. Although the discourse of autism has been framed by professionals and academics, it is in reality only one side of the triangle, whose base is the people who have been ever increasingly subsumed into the category, while on the other side of the professionals are the parents who have applied political pressure and undertaken practical projects to improve the lives of their children. In the middle is everything else, media representation, popular fictions, and alas a cloud of misunderstanding. However, without autistic people themselves, there would be no discourse at all. It is then a landscape of neurological

difference emerging through the subjective experience, upon which political and cultural divisions have been drawn.

Early History

Although Autism as a term was not coined until 1912, or formally delineated until the 1940s, its phenotype can be found in England at the beginning of the psychiatric narrative through a new understanding of the writings of John Langdon Down (Waltz 2013). It can also be found in literature in the equally astute observations of authors whose fictional characters are based on their perspicacity. Sherlock Holmes springs instantly to mind, but there are examples to be found in Dickens, Hardy, and so on, going all the way back to Shakespeare, whose professional clown Robert Armin wrote “foole on foole” both a history of his profession and a narrative of the “natural,” among which descriptions you can be certain to find the traits of autism as it is now conceived (Mc Donagh 2008).

Current understandings of autism emerged mostly after the Second World War, with the predominant psychoanalytical school holding sway with the notion of the “refrigerator mother” precipitating her offspring into the form of the unreachable child afflicted with some kind of severe psychosis, to use Bettelheim’s metaphor of the “empty fortress.” In cultural terms, it has become an othering narrative (Said 1977) whose effect on the popular perception of the autistic can still be seen today in popular fiction. For example, Stuart Murray instances (Murray 2008) an Observer Sunday supplement article of the mid-1960s dealing with the “pioneering” efforts of the Lindens school to “reach” autistic children and prepare them for mainstream schooling.

It is important to remember that Education in England since the 1944 education act was predicated on separation of children into categories one of which was Educationally Subnormal (ESN) further subdivided into moderate and severe. Children in the severe category were deemed as “ineducable” and only capable of training. Most children who would then have been identified as

autistic (if at all) would have fallen into one of the ESN categories. This was a state of affairs that was perhaps first challenged in 1962 by a group of London-Based parents (including Lorna Wing q. v.) who decided to take matters into their own hands forming the Autistic Children's Aid Society of North London to establish a school for the education of their autistic children. The Sybil Elgar school was opened in 1965 and the Helen Allison school in 1968. As with many societies who had been established in a similar fashion by parents of children with a range of impairments, there were some with purely local and others with a regional or national scope.

In time the North London group assumed a national focus, and with the realization that autistic children grew up to be autistic adults, dropped the "children" from the title becoming what we now know as the National Autistic Society (NAS). This was a pattern reflected by other impairment groups who had focused originally on children.

Research

One of the earliest researchers to take an interest in the phenomenon of Autism was Sir Michael Rutter who has been associated with the Institute of Psychiatry Kings College London since 1965, firstly as a consultant psychiatrist at the Maudsley hospital and later as the first chair of child psychiatry. He currently holds the position of professor of Developmental Psychopathology. Although Rutter can best be characterized as "old school" in terms of current developments in the study of autism (particularly in terms of gender qv), he did contribute much to the field of epidemiology and helped to establish autism as a probably genetic condition in reaction to the former psychoanalytical and behaviorist perspectives, thus helping to put the study of autism on a firm scientific footing.

The 1970s saw changes in education, leading to the establishment of special schools and following on the publication of the influential Warnock Report this was crystallized into the 1981 Education act, with the widespread use of

"special needs" as a category replacing "subnormal" a broader term encompassing a range of impairments and disabilities including autism, with an emphasis on mainstream schooling with appropriate provision for those needs with the role of the Special Educational Needs Co-ordinator (SENCO) being prominent. The 1970s were also a time for the broadening of the phenotype of autism to include a larger population of autistic people hitherto unrecognized.

Lorna Wing in particular, with her NAS colleague Judy Gould, has arguably made one of the greatest contributions to the understanding of Autism through the introduction of the concept of an "autistic spectrum" (Wing and Gould 1979) to refer to this broader conception of the term "autism" which came about after one of the first widespread epidemiological surveys in Camberwell identifying a larger number of people with the traits of autism than those previously recognized as having the condition. Along with Uta Frith, Wing also helped to introduce the work of Austrian Paediatrician Hans Asperger to the English-speaking world. At the time she saw the use of the term Asperger's (or Asperger) syndrome as less stigmatizing to autistic people whose intelligence fell within and beyond the normal range.

Uta Frith, who completed her PhD in Autism in 1968 at University College London, has also been an influential writer and researcher bringing the study of autism as a neurocognitive phenomenon into greater prominence. She developed the weak central coherence explanation of autism which in common with Asperger's paper, which she was the first to translate into English, recognized strengths as well as deficits in autistic cognition. As author of "Autism Exploring the Enigma," she also strayed into cultural territory giving an overview of what Autism might have looked like in historical times. She has supervised a number of researchers who have gone on to prominence including Francesca Happé who has further developed the central coherence theory (Happé 1999), and Frith also supervised Simon Baron-Cohen (qv).

The seventies and on into the eighties also saw other developments in the perceptions of

disability which would have a subsequent impact on the landscape of Autism.

With growing social awareness and paralleling the civil rights movement of the USA and anti-apartheid movements in South Africa, segregation became to be seen by groups of radicalized disabled people as an experience not unique to race and something accomplished by the educational system, institutionalized care and prejudicial treatment of disabled people in the workforce. Out of this grew, the social model of disability which posited that disability arose from societal attitudes and barriers, and impairment was not its cause (Oliver 1990; Finkelstein 1975).

In terms of legislation, the 1970s brought in the Chronically Sick and Disabled Persons Act, which began to confer new rights to individuals (autistic people and the parents of autistic children among them) and new duties upon local authorities and health providers, and ushered in Social Services departments.

Another force for social change was the growth of an “antipsychiatry” movement which questioned the basis of contemporary psychiatry as more of a means of containment and control than directed toward recovery, drawing on the works of Foucault and Goffman and exemplified by RD Laing as a radical psychiatrist in England. Autism research would thus far seem to be untouched by this movement except so far as to become finally detached from its initial consideration as a psychosis and possible form of childhood schizophrenia through its inclusion in the Diagnostic and Statistical Manual, third edition (DSM-III) of the American Psychiatric Association in 1980 (APA 1980).

Simon Baron Cohen, based at the Autism Research Centre Cambridge University (founded in 1998), did much to popularize the “Theory of Mind” model which he had developed in 1985 (Baron-Cohen et al. 1985). He has subsequently gone on to more controversial research positing the Extreme Male Brain Theory (Baron-Cohen 2002), which has been challenged by Autistic scholars such as Melanie Yergau and others including the authors of this article (Yergau 2013).

Dinah Murray developed the interest theory of Autism (readers may be more familiar with the terms single attention and monotropism) with Mike Lesser (Murray et al. 2005). Although this has not become a mainstream model of autism to the extent of Theory of Mind or Central Coherence, it has nevertheless found favor with Autistic scholars as a fairer means of describing the neurological mechanisms of autism than any specific model which posits deficit. Murray argues that Autistic people’s strengths and special interests can play an important role in their education and provide a dividend for the employment of autistic people as adults as instanced by the work of Specialisterne, who have been elsewhere in the UK employing Autistic people in testing software.

Research Autism was established by the NAS in 2004 to evaluate and raise funds for research into interventions in Autism as well as providing authoritative guidance for professionals and the public on the bewildering variety of interventions that exist. It is currently winding down with its functions being reabsorbed into the work of the NAS.

Current Centers for Research, Education, and Publishing

While most of the research into autism has been conducted within the medical framework, there has been a significant focus on Education with certain programs attracting an increasing number of autistic students and researchers such as the Autism Centre for Educational Research at the University of Birmingham and the Sheffield Hallam University Autism Centre’s Post Graduate Certificate run in conjunction with the NAS, both of which have included autistic people in providing course materials, teaching, and tutoring.

Other prominent research arenas include the Centre for Research into Autism and Education (CRAE) at University College London (UCL), whose former director, Liz Pellicano, has led the way in involving autistic people in setting a research agenda through the shaping autism

research series of seminars. Other autistic-led research initiatives worthy of mention include the Participatory Autism Research Collective (PARC) – whose aim is to build a community of those who wish to see more significant involvement of autistic people in autism research – and the team of primarily autistic researchers responsible for recent surveys of support for autistic students at university and in colleges of further education in England.

Besides the Kings College Institute of Psychiatry, Kings College has been involved in examining the ethical basis of autism research, hosting an ethics discussion group including autistic activists, ethicists, and philosophers as well as autism researchers in an endeavor which led to the conference Autism, Ethics, and Society held at UCL in 2010.

The Autism Research Group (ARG) at City University, London, has also led medical research in attempting to establish the neurobiological and genetic underpinnings of the cognitive and psychological differences in Autism.

The open access academic journal “Autonomy” (the Critical Journal of Interdisciplinary Autism Studies) was first published in 2012 with Larry Arnold as Editor. Autonomy has probably published more contributions by autistic scholars than any other individual academic journal since it was founded (Arnold, Editorial, 2012).

Advocacy

The 1990s saw the beginnings of a backlash against the framing of autism without input from autistic people and with the growth of access to the internet during this period, this took on an international dimension as the ideas of Sinclair (2005) and others became available to autistic people in England. Significant in England at the time was the online facilities offered by Martijn Dekker (see ► [“Personal perspectives on Autism”](#)) which allowed groups of autistic people to discuss a variety of topics regarding self-help

and advocacy. From those beginnings, individuals who had initially met online started to meet face-to-face to achieve other aims. These movements drew upon the notion of “neurodiversity,” a term first coined by Judy Singer in 1998 to refer to the natural variation in neurotype. In parallel to Dekker’s mailing list, there were others centered upon other forms of neurodiversity such as dyspraxia. Mary Colley (a dyspraxic adult) was a significant figure in this discourse and instrumental in forming the Developmental Adult Neurodiversity Association (DANDA) in 2003 following dissatisfaction with the parent-led focus of the Dyspraxia Foundation charity, dyspraxia as a condition having strong overlaps with Autism. Many of the early members of this organization were also Autistic and went on to play significant roles in other autistic and neurodivergent movements including membership of the Disability Rights Commission neurodiversity action group established in 2005.

In 1999, the NAS saw its first autistic Council member (Richard Exley) and in 2001 saw its first Autistic Board member (Larry Arnold), whose appointment led to significant changes within the organization including constitutional amendment to ensure equal rights and greater participation for autistic people within the organization and significant changes to their public presentation and campaigning.

In 2005, a group of Autistic people were asking why there was no equivalent in Europe of Autreat (see ► [“Personal perspectives on Autism”](#)) (a retreat-style conference run by and for autistic people), a question which led to the introduction of a similar event in England known as Autscope. Initially with assistance and advice from Autism Network International (ANI) members Jim Sinclair and Patty Clarke, in its first year, it managed to attract an international attendance, from the USA, Israel, and with a strong presence from the Nordic Countries and the Netherlands. Autscope emphasized the importance of autistic people as an international community.

In 2007 a group of politically motivated individuals met together at Autscope which led to the

formation of the London Autistic Rights movement, launched at a meeting in City Hall London where the key speakers were Dinah Murray, Larry Arnold, and David Morris, the London Mayors senior disability policy advisor (Arnold, Provisional Front Page for Autreach, 2007). The group initially had a specific focus on the capital, with access to the Westminster Parliament; however, this led to a UK-wide focus and the formation of Autistic UK which is the only national campaigning organization in the UK run by autistic people.

Autistica was founded in 2004 as Autism Speaks UK by Dame Stephanie “Steve” Shirley. In 2009, it decided to end its links with the US Autism Speaks. Its aims are to *promote and fund* “medical research to understand the causes of autism, improve diagnosis, and develop new treatments and interventions.”

In 2008 the founders of US-based Autism Speaks toured to promote the organization in the UK. This was vigorously opposed by autism rights campaigners who saw the organization as erasing autistic identity and rights. Of significant import was the Tree House (currently Ambitious about Autism) annual lecture. Tree House had inadvertently invited Bob and Suzanne Wright, the founders of Autism Speaks. In order to balance the expression of opinions, they commissioned Autreach to make a video to be shown at the event. The video called “Something About Us” (Murray and Benstock 2007) consisted of a collection of autistic people – not all of them verbal – presenting individual segments about the positive aspects of autism, to counter the negative stereotypes and pity invoking narratives of hard-line organizations committed to the elimination of autism.

An example of the progress resulting from such dialogues was the National Autism Project established in 2015 with the aim of providing “authoritative recommendations on those aspects of autism research and practice that have demonstrable effectiveness in benefiting autistic people and their communities.” Dinah Murray’s own advisory team of autistic people subsequently became the autistic advisory panel for the whole project, marking a significant breakthrough in

terms of the input of autistic people to a national autism initiative.

Current Issues and Controversies

Gender

Autism was once thought of as a predominantly male phenomenon with an oft-quoted ratio of four males to one female (and higher in the so-called “low-functioning” individuals). This was defended on the basis that these were the research findings which has led to the Extreme Male Brain Theory of autism (op cit). This has been much criticized from a feminist perspective (Yergau op cit) as stereotyping the so-called traits of maleness.

Behavioural Approaches to Autism

For the most part, the UK has adopted an eclectic approach to “intervention” and education, including the NAS’ SPELL (Structure, Positive, Low arousal, Links) TEACH (Teaching, Expanding, Appreciating, Collaborating and Cooperating, Holistic), Social Stories™ and counseling, in contrast to the predominance of ABA (Applied Behavioral Analysis) in the USA. ABA, however, has found its strong proponents in the Treehouse School (op cit). Mentoring has been found to be of value in higher education and employment and an autistic mentoring project (the Cygnet Project) was developed by London South Bank University led by Nicki Martin and Damian Milton.

Pseudoscience and Cures

Ever since the psychodynamic approach to autism was challenged by Bernard Rimland (1964), there has been an increasingly biomedical focus on Autism in some quarters prompted much by Rimland’s later work at the Autism Research Institute (USA), whose findings have been met with some skepticism elsewhere. Undoubtedly the most damaging controversy to emerge initially in England was the Measles, Mumps, and Rubella (MMR) vaccine scare which was bolstered by the since retracted and discredited study of Andrew Wakefield (Flaherty 2011). Since then there has

been an explosion in alternative medicine and the promotion of nonvalidated and potentially dangerous “cures.”

Employment

Historically, it is likely that autistic people found it easier to get work, but in an increasingly demanding environment this is no longer the case. Notwithstanding the potential problem of successful autistic people being underdiagnosed, there is now substantial evidence of high unemployment of autistic people even among those who are highly qualified. A great deal of this can be attributable to the structural exclusion of autistic people through advertisements and selection processes, which generally reflect those traits that autistic people are less likely to have.

There have been numerous attempts, such as the NAS prospects service, to provide employment support for individuals to help integrate them into the workforce and to keep positions in a highly socialized and rapidly changing environment. Likewise, there have been approaches concentrating upon the strengths of autistic people in recruiting for example software engineers and testers. Economic cases have also been made out for the costs of excluding autistic skills.

Recommendations for successful employment involve creating less sensorially stressful working environments and job mentoring through the social aspects of employment.

Legal Perspectives on Autism

Discrimination on the grounds of race and gender in England had been outlawed by UK legislation in 1965 and 1975, respectively. However, it was not until the Disability Discrimination Act (DDA) 1995 that discrimination on the grounds of disability was made illegal (in respect of employment and the provision of goods and services). The DDA introduced the concept of “reasonable adjustments” for those with disabilities. The Special Educational Needs and Disability Act of 2001 extended the ban on disability discrimination and introduction of reasonable adjustments to the

education sector. In 2010, the Equality Act subsumed all previous UK disability discrimination legislation.

Although it took three decades for the law in England to treat disability on an equal footing to race and gender, this is only the beginning. Despite race discrimination legislation having been in place for many years, the Metropolitan Police Service was described as “institutionally racist” by the Macpherson Report of 1999. Institutional racism is likely to be endemic despite the efforts of antiracism campaigners and legislation. Even if disability discrimination is not endemic, and there are those who argue that it is, the legal requirement to provide reasonable adjustments is not fully complied with in many areas of society including higher education despite the legislative requirement being in place for more than 15 years. The first diagnostic acknowledgment of autism (in the DSM-III) was only 1 year before the legislative objective to achieve inclusive education was enacted and the introduction of the process around SEN. Although both covered autism, there was likely to be a delay in achieving both objectives in comparison to well-established disability categories.

The Autism Act 2009 is the first autism-specific law in the world and first law specific to a particular disability in England. This Act makes imposes certain requirements on local authorities in relation to provision for autistic adults and required the Government to produce an adult autism strategy. The first Adult Autism Strategy was produced in 2010 and updated in 2014. Unfortunately, despite the efforts of those pushing for autism legislation, the Act does not cover children and young people which might have helped autism “catch up” with other SEN categories in education. And as with previous legislation (e.g., the Education Act 1981), no additional funding was forthcoming from Government for implementing the Autism Act provisions.

The Community Care Act (1990) modernised the pioneering Chronically Sick and Disabled Persons Act 1970 which had ushered in modern social service provision. This currently includes provision for services for autistic people including direct payments and personal budgets. Its

workings are however currently much restrained by the climate of austerity.

References and Reading

- APA. (1980). *Diagnostic and statistical manual of mental disorders 3rd edition*. (Various ed.). Washington DC: American Psychiatric Association.
- Arnold, L. (2007). Provisional front page for autreach. Retrieved from Autreach: <http://www.autreach.eu>.
- Arnold, L. (2012). Editorial. *Autonomy, The Critical Journal of Interdisciplinary Autism Studies*, 1(1). Retrieved from <http://www.larry-arnold.net/Autonomy/index.php/autonomy/article/view/ED1/html>.
- Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences*, 6(6), 248–254.
- Baron-Cohen, S., Frith, U., & Leslie, A. (1985). Does the autistic child have a “theory of mind”? *Cognition*, 21, 37–46.
- Finkelstein, V. (1975). To deny or not to deny disability – What is disability? *Magic Carpet*, 27(1), 31–38.
- Flaherty, D. K. (2011). The vaccine-autism connection: A public health crisis caused by unethical medical practices and fraudulent science. *Annals of Pharmacology*, 45(10), 1302–1304.
- Frith, U. (1992). *Autism, explaining the enigma*. Oxford: Blackwell.
- Happé, F. (1999). Understanding assets and deficits in autism – Why success is more interesting than failure. *The Psychologist*, 12(11), 540.
- Mc Donagh, P. (2008). *A cultural history of idiocy*. Liverpool: Liverpool University Press.
- Murray, S. (2008). *Representing autism*. Liverpool: Liverpool University Press.
- Murray, D., & Benstock, J. (Directors). (2007). *Something About Us* [Motion Picture].
- Murray, D., Lesser, M., & Lawson, W. (2005). Attention, monotropism and the diagnostic criteria for autism. *Autism*, 9(2), 139–156.
- Oliver, M. (1990). *The individual and social models of disability paper presented at joint workshop of the Living Options Group and the Research Unit of the Royal College of Physicians. On People with established locomotor disabilities in hospitals July 1990*. Retrieved 10 May 2010, from <http://www.leeds.ac.uk/disability-studies/archiveuk/Oliver/in%20soc%20dis.pdf>.
- Rimland, B. (1964). *Infantile autism: The syndrome and its implications for a neural theory of behavior*. New York: Appleton-Century-Crofts.
- Said, E. (1977). *Orientalism*. London: Penguin.
- Sinclair, J. (2005). *Autism network international: The development of a community and it's culture*. Retrieved 16 June 2010, from Autreat.com: http://www.autreat.com/History_of_ANI.html.
- Waltz, M. (2013). *Autism a social and medical history*. Basingstoke: Palgrave Macmillan.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9(1), 11–29.
- Yergau, M. (2013). Clinically significant disturbance: On theorists who theorize theory of mind. *Disability Studies Quarterly*, 33(4).

Enhanced Perceptual Functioning

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Synonyms

EPF model

Definition

The enhanced perceptual functioning (EPF) model of autism proposes that superior function and increased independence of auditory and visual perceptual processes are responsible for the distinct pattern of cognitive, behavioral, and neural performance observed in autism. The EPF model emphasizes the primacy of perceptual processes, rather than social or higher order cognitive processes, in giving rise to the autistic phenotype.

Initially proposed by L. Motttron and colleagues as an alternative to the weak central coherence (WCC) model of perceptual functioning, the EPF outlines eight principles of perception in autism:

1. More locally oriented perception is the default for individuals in autistic populations relative to typical populations.

2. Neural complexity is inversely related to performance in low-level perceptual tasks.
3. Early atypical behaviors regulate perceptual input.
4. In autism, primary and associative brain regions involved in perception are atypically activated during social and nonsocial tasks.
5. Higher order processing is variable in autism and mandatory in typical development.
6. Perceptual expertise underlies savant syndrome.
7. Savant syndrome is an autistic model for subtyping within pervasive developmental disorder (PDD).
8. Enhanced functioning of perceptual brain regions may contribute to abnormalities in perception in autism.

Evidence in support of this model stems from the demonstration of enhanced visual functioning across multiple tasks (Caron et al. 2006) and enhanced brain activity in brain regions implicated in pattern processing in autism (Samson et al. 2011).

See Also

- Auditory Processing
- Autistic Savants
- Perception
- Perceptual Development
- Social Cognition
- Visual Processing
- Weak Central Coherence

References and Reading

- Caron, M. J., Mottron, L., Berthiaume, C., & Dawson, M. (2006). Cognitive mechanisms, specificity and neural underpinnings of the block design peak in autism. *Brain*, 129, 1789–1802.
- Mottron, L., & Burack, J. A. (2001). Enhanced perceptual functioning in the development of autism. In J. A. Burack, T. Charman, N. Yirmiya, & P. R. Zelazo (Eds.), *The development of autism: Perspectives from theory and research* (pp. 131–148). Mahwah: Erlbaum.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. A. (2006). Enhanced perceptual functioning in autism: An updated model and eight principles of

autistic perception. *Journal of Autism and Developmental Disorders*, 36(1), 27–43.

- Samson, F., Mottron, L., Soulières, I., & Zeffiro, T. A. (2011). Enhanced visual functioning in autism: An ALE meta-analysis. *Human Brain Mapping*. <https://doi.org/10.1002/hbm.21307>.

Entrepreneurial Model

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Synonyms

Entrepreneurial supports; Supported employment; Supported work

Definition

The term Entrepreneurial Model refers to creation of a new business entity as a means of providing work for an individual, or a group of individuals, with a diagnosis of a developmental disability. In this model, the skills and interests of the individual, or a group of individuals, with an autism spectrum disorder are used as a basis for the creation of a new business. The goal of the endeavor is to create enough revenue from the business to both pay the workers with a disability and support staff who assist them. An advantage of the Entrepreneurial Model is that the work involved is specifically chosen to match with the skills and interests of the individual (or individuals) with an autism spectrum disorder. The biggest challenge for an Entrepreneurial Model is that it faces the same start-up costs and difficulties in reaching profitability of any new business endeavor.

An Entrepreneurial Model is one work option along a continuum of supported employment for individuals with a disability. In addition to the Entrepreneurial Model, the most common types of supported employment models are an individual placement model, an enclave placement model, and a mobile work crew.

See Also

- ▶ Entrepreneurial Supports
- ▶ Mobile Work Crew Model
- ▶ Supported Employment

References and Reading

- Advancing Futures for Adults with Autism, an advocacy organization. <http://www.affaa-us.org/site/c.111Y1kNZJuE/b.5063863/k.BE3C/Home.htm>
- Autism Transition Handbook, an on-line resources supported by Devereux, Inc. http://www.autismhandbook.org/index.php/Main_Page
- Gehardt, P. F. (2009). The current state of services for adults with autism. *Organization for Autism Research*. Retrieved 29 May 2011 from http://www.mo-feat.org/files/oar_survey_11309.pdf
- James, E., & Young, D. (2007). Fee income and commercial ventures. In D. Young (Ed.), *Financing nonprofits*. Lanham: AltaMira Press.
- McDonnell, J., & Hardman, M. L. (2010). *Successful transition programs: Pathways for students with intellectual and developmental disabilities*. Thousand Oaks: Sage.
- Skloot, E. (1988). *The nonprofit entrepreneur: Creating ventures to earn income*. New York: The Foundation Center.
- Wehman, P., Brooke, V., & West, D. (2010). Vocational placements and careers: Toward inclusive employment. In P. Wehman (Ed.), *Life beyond the classroom: Transition strategies for young people with disabilities*. Baltimore: Brookes.

Entrepreneurial Supports

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Synonyms

Self-directed support corporation supports; Self-employment supports

Definition

Entrepreneurial supports are supports that would enable individuals working under an

entrepreneurial model to become successful. Success would be measured by the self-directed business ability to generate enough revenue not only for salaries of the individuals on the autism spectrum in the corporation but also for salaries of the staff hired to help them with this endeavor.

Like any other business, support takes many forms. It can include help in developing a business plan, securing loans or funding for start-up costs, accounting support in terms of paying taxes and other payroll issues, as well as taking advantage of employer initiatives and tax incentives such as Work Opportunity Tax Credits, Small Business Tax Credits, and Architectural/Transportation Tax Deduction: IRS Code Section 190, Barrier Removal (Gerhardt 2009). Support can also be in terms of renting office space, buying furniture and equipment, and advertising the product or service. Job coaching may also be a necessary support for individuals on the spectrum under this model.

Prevocational supports may also be necessary for individuals who are working under an entrepreneurial model. Prevocational supports may include travel training on mass transit in order to reach work or business meetings. It may also include how appropriate dress and hygiene for the type of business the individual is engaged in. Independent living skills might include money management and budgeting skills for the individual (e.g., grocery shopping, bill paying, paying for lunch) on top of money management skills or supports for the business.

“Many people with disabilities, particularly those in rural areas where jobs are often scarce, have already created opportunities for themselves through entrepreneurship. In fact, according to the U.S. Census Bureau, people with disabilities are nearly twice as likely to be self-employed as the general population, 14.7 percent compared to 8 percent” (US Department of Labor and Office of Disability Employment Policy 2011). Small businesses account for 60–80% of the new jobs annually (US Department of Labor and Office of Disability Employment Policy).

Government supports for individuals with disabilities who are engaged in the entrepreneurial model often begin with Vocational Rehabilitation (VR) services and programs:

- The Social Security Administration's (SSA) Plan for Achieving Self-Support (PASS) program allows people with disabilities receiving SSI benefits to set aside money and resources to help achieve a particular work goal, including self-employment.
- The Ticket to Work program connects SSI and SSDI beneficiaries with Employment Networks (EN) for training and other support services needed to achieve their employment goals, including self-employment.
- More than 1,100 Small Business Development Centers (SBDC) offer free or low-cost counseling, training, and technical assistance to individuals seeking to start their own business in communities across the nation.
- The Service Corps of Retired Executives (SCORE), comprising more than 10,000 counselors at 389 offices nationwide, provides free small business start-up advice through one-on-one counseling, group workshops, and online resources.
- Local One-Stop Career Centers funded through the US Department of Labor's (DOL) Employment and Training Administration (ETA) assist people in training for and obtaining employment, including self-employment (US Department of Labor and Office of Disability Employment Policy 2011).

See Also

- [Entrepreneurial Model](#)

References and Reading

- Brown, R. I. (1997). *Quality of life for people with developmental disabilities: Models, research, and practice* (2nd ed.). Cheltenham: Stanley Thorne.
- Center for Self-Determination. (2011). <http://www.centerforself-determination.com/>
- Edmonds, G., & Beardon, L. (Eds.). (2008). *Asperger syndrome and employment: Adults speak out about asperger syndrome*. London: Jessica Kingsley.
- Gerhardt, P. (2009). *The current state of affairs for adults with autism* (draft). Advancing futures for adults with autism: Think Tank. New York, January 21, 2009.
- Grandin, T., & Duffy, K. (2008). *Developing talents: Careers for individuals with Asperger syndrome and*

high functioning autism. Overland Park: Autism Asperger Publishing.

- Rizzo, D. C. (2002). With a little help from my friends: Supported self-employment for people with severe disabilities. *Journal of Vocational Rehabilitation*, 17(2), 97–105.

U.S. Department of Labor, Office of Disability Employment Policy. (2011). *Entrepreneurship: A flexible route to economic independence for people with disabilities*. Retrieved 28 July 2011, from <http://www.dol.gov/odep/pubs/misc/entrepre.htm>

Enuresis

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Synonyms

[Bed-wetting](#)

Short Description or Definition

It is typical in developed countries that toilet training begins in the toddler period and usually is completed by age 3; there is some variation in this age (and in toilet training) across cultures. Various factors can interfere with the process including motor difficulties, cognitive and social problems, and lack of motivation (any combination of which may be present in younger children with ASDs). When toilet training is not achieved or was once achieved and then lost, a diagnosis of enuresis is made.

Categorization

A distinction is made between primary enuresis (the child has never been fully trained) and secondary enuresis (the child had been trained for a prolonged period – usually at least a year). Night-time or nocturnal enuresis is much more common than the daytime form. Isolated and very occasional

episodes of bed-wetting, particularly in younger boys, may not merit either a diagnosis or treatment since there is an expectation that the problem be sufficiently severe as to merit attention (either because of frequency or because it serves as a source of distress). The diagnosis is not made in very young children and cannot be made if the voiding is due to medical problems.

Epidemiology

Longitudinal studies have shown that boys are more likely than girls to exhibit the problem. By age 6 years, the vast majority of children (90%) are dry at night, and this number continues to rise so that in adolescence (age 15) only about 1% of boys and 0.5% of girls have episodes of enuresis on at least a weekly basis. There is some suggestion of familial risk and association with both stress and psychosocial adversity.

Natural History, Prognostic Factors, and Outcomes

Spontaneous remission of bed-wetting is common and often occurs by age 7. It is typical in longitudinal studies that during a given year, about 15% of children will have their symptoms remit. Behavioral treatments are typically regarded as a first line of intervention. The presence of multiple risk factors in the child, stress, and psychosocial adversity can complicate intervention. Behavioral psychologists can be involved in situations where developmental delays are present to help both in an assessment of the child's potential for understanding and participating in a treatment program and in a broader analysis of any associated behavioral difficulties.

Clinical Expression and Pathophysiology

A range of theories have been proposed – from those that focus on anatomic abnormality, psychological or neurological immaturity, to psychodynamic explanations. Developmental delay is a risk factor, and some data suggest complex interactions of factors in pathogenesis. A genetic component can be present.

Evaluation and Differential Diagnosis

Laboratory analysis of urine (to rule out infection) is a first step. There is not a high yield from more medically invasive studies in the absence of an indication for the same. Those children who have both day- and nighttime difficulties are more likely to have anatomical or other physical problems. Sometimes enuresis can be associated with other medical conditions or medications. Urinalysis is an obvious first step in evaluation of enuresis, for example, to rule out urinary tract infection as a cause. In general invasive laboratory studies do not have a particularly high yield and would not be indicated unless other indications were present. Children who have problems in both night- and daytime may be more likely to exhibit structural or other problems of the urinary tract. Ultrasound evaluation is less invasive than past procedures.

At times enuresis may arise following other medical problems, for example, hyperthyroidism, although this is infrequent. A physical examination should look for potentially treatable underlying conditions. Associations to other factors, for example, nocturnal enuresis that follows administration of a new medication, should be explored as relevant.

Treatment

Two approaches have been used for treatment: behavioral and pharmacological. Behavioral treatments have long been used and combine aspects of classical and operant condition in the “bell and pad” approach. The child sleeps on a pad that, if it becomes wet, closes an electrical circuit and rings a bell, thus awakening the child. A majority of children do respond, and the response is frequently maintained after the treatment is discontinued. Other behavioral approaches include use of a timer to awaken the child periodically to void.

Drug treatments have included several agents. The tricyclic antidepressant imipramine has been used for many decades. An ECG is obtained at baseline and the dose of the medicine gradually increased. Many children respond positively. In

such cases periodic attempts to discontinue it should be made. The mechanism of action remains unclear.

A more recent pharmacological treatment has used desmopressin acetate (DDAVP) (an analogue of a pituitary hormone involved in kidney function). The agent frequently is associated with a positive response, but symptoms do tend to turn once the intervention is discontinued and various side effects can be noted.

In the absence of other indications, psychotherapy is not usually indicated.

See Also

► Toilet Training

References and Reading

- Robson, W. L. M. (2009). Clinical practice. Evaluation and management of enuresis. *New England Journal of Medicine*, 360(14), 1429–1436.
- Volkmar, F. R., & Martin, A. (2011). *Essentials of child and adolescent psychiatry*. Philadelphia: Lippincott, Williams, and Wilkins.
- Ward-Smith, P., & Barry, D. (2006). The challenge of treating enuresis. *Urologic Nursing*, 26(3), 222–224.
- Weaver, A., & Dobson, P. (2007). Nocturnal enuresis in children. *Journal of Family Health Care*, 17(5), 159–161.

Environmental Engineering/Modifications

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Definition

Environmental engineering/modification strategies, also known as *antecedent interventions*, are defined as changes to the immediate environment that allow the individual with ASD to process

stimuli, predict future events, and respond appropriately. The purpose of environmental engineering strategies is to provide additional information about the appropriate and expected response in any given situation (Neitzel 2009; Quill 1995). They are considered proactive strategies to prevent problem behavior and increase appropriate behavior before correcting strategies are needed (Bregman et al. 2005). Since they are preventative in nature, antecedent interventions are often considered very socially acceptable interventions (Radley and Dart 2016).

There are a myriad of interventions that fall under the heading of environmental engineering. Such interventions include, but are not limited to, social stories and video modeling, functional communication training, consistent scheduling, choice, and organization, and visual supports. These interventions are often used to prevent problem behavior, but can also be used to increase adaptive behavior and as academic supports for learners of all ages. It has also been suggested that environmental engineering may increase independence and generalization of learned skills (Quill 1995). The majority of studies on antecedent interventions, though, focus upon their use to decrease problem behavior, such as stereotypy or self-injurious behaviors (Neitzel 2009). Although used widely by educators and considered to be effective and easy to implement, there is comparatively little research on the effects of antecedent manipulations on the behavior of children with ASD.

Historical Background

Antecedent interventions have, in some way, been an important part of interventions for individuals with ASD throughout the short history of intervention for the disorder. Historically, researchers have focused on consequences when assessing and intervening for problem behavior, however (Wacker et al. 2006). Division TEACCH, considered by some to be the first program to provide structured interventions for individuals with ASD, has been incorporating environmental engineering strategies since the 1960s (Mesibov et al. 2005). Some antecedents found to influence

learning and appropriate behavior include how instructions are delivered, timing and scheduling of learning opportunities, and medical and physical issues.

In applied behavior analysis, the antecedents which evoke both problem behavior and appropriate responding are considered to be critical to intervention. There are two different types of antecedents to consider – those that signal responding will result in a reinforcer (discriminative stimuli), and those that make reinforcement more or less potent (Motivating Operations; Wacker et al. 2006). Tools such as antecedent-behavior-consequence (ABC) data collection and scatterplot data determine what types of environmental events evoke problem behavior and what environmental changes could prevent reoccurrence of these types of behavior (Bijou et al. 1968; Touchette et al. 1985).

Current Knowledge

Whatever form of environmental engineering is used, it should be universally accepted by the members of the individual's community and implemented consistently. Those strategies that are most socially valid are more likely to be implemented consistently. Therefore, any strategy should take into account the child's environment, preferences, and community at large (Quill 1995). Considering antecedents, as well as consequences, allows for more precise assessments and better tailored interventions overall (Wacker et al. 2006).

It is also very important that any strategy selected is age-appropriate, especially for older learners with more severe impairments. For example, a 16-year-old with a visual system that includes pictures of preschool cartoon characters may serve to stigmatize the individual and decrease opportunities for social interaction. A more socially appropriate alternative might be using pictures of the adolescent themselves or other individuals in the class (Wehman 2006).

Before making environmental modifications, a thorough functional behavioral assessment (FBA) should be conducted to determine what environmental variables reliably evoke problematic

behavior for the individual in question (Bregman et al. 2005; Umbreit et al. 2007). In FBA, detailed data are taken to determine both the environmental events and the consequences that might be maintaining problem behavior. In more difficult cases, a functional analysis might be conducted. Functional analysis consists of a series of short, tightly controlled sessions where situations which are hypothesized to evoke the problem behavior are recreated. Data are taken on the antecedents, problem behavior, and consequences. If specific situations, when compared to control conditions, do, in fact, evoke problem behavior, they are considered to be the function (Iwata et al. 1994). Hurl et al. (2016) conducted a meta-analysis comparing interventions derived from functional assessment and analysis to those developed without assessment. Interventions developed from functional assessment, and especially functional analysis, had much higher treatment effects than those without. FBA, including functional analysis, allows the practitioner to tailor the intervention to the function of the behavior being assessed.

Structural analysis (SA; Stichter et al. 2009) may also be of help, and implementation is very similar to a functional analysis. In SA, the consequences for problem behavior are held constant and the antecedents are systematically manipulated. For example, an interventionist might be interested in whether pictures or verbal prompts are more effective in increasing on-task behavior. In one condition, verbal prompts are presented to the individual and on-task behavior is reinforced by verbal praise. In the second condition, the same task is presented, but this time visual cues are used to prompt responding. Reinforcement stays constant for on-task behavior. The strategy that produces more on-task behavior across several presentations is considered to be the most effective of the two interventions being evaluated.

There are several types of environmental engineering strategies available to those who work with individuals with autism. For the sake of brevity, these will be broken into the following categories: (1) priming strategies, (2) physical environmental modifications, and (3) activity-based strategies. What follows should not be considered to be an exhaustive list of strategies that

utilize environmental engineering; instead, it should be considered to be a survey of the myriad of environmental engineering interventions that are available to the interested reader.

Priming Strategies

Priming strategies consist of instruction on how to behave appropriately in or preexposure to new or stressful situations. They are designed to provide advanced knowledge to the individual with ASD that allows him or her to predict what will happen in the future. Some examples of priming strategies include video modeling, Social Stories™, and functional communication training.

In a traditional priming session, an individual visits an unfamiliar situation (e.g., school) prior to entering the situation. For example, a student with ASD might visit the first-grade classroom he or she will attend before the first day of school. This allows the individual to become comfortable with the environment so that navigating that environment is less overwhelming. An example is provided by Koegel et al. (2003). Students experiencing difficulty in preacademic and academic situations were exposed to the day's lesson the night or day before the lesson was presented in class. This reduced problem behaviors such as stereotypic behavior, repeated requests for the bathroom, inappropriate verbalizations, and disruptive behavior. Many of the strategies that follow are used in conjunction with traditional priming to increase appropriate behavior and decrease stress. Orellana et al. (2014) found that a component package of priming interventions increased compliance with routine dental exams. Intervention included familiarizing participants with common dental tools, video models of dental exams, and explaining the dental procedure (Orellana et al. 2014).

High-Probability Request Sequences (Hi-P). A High-Probability Request Sequence, or Hi-P, is typically implemented when quick compliance with a less preferred task is required. With Hi-P, a series of requests with a high probability of compliance are presented in quick succession, with the low-probability request immediately following. The momentum built by engaging in high-probability tasks increases the likelihood of

engagement in the low-probability task (Mace et al. 1997). For example, a teacher may tell a student to do a series of easy responses such as touch their nose, clap their hands, and give them a high five. Once these easy responses are complete, the targeted instruction is delivered. In order for Hi-P to be effective, the interval between requests must be only a few seconds. Using strongly preferred items as reinforcers for compliance with a low-probability request is also critical to success (Mace et al. 1997; Radley and Dart 2016).

Video modeling uses a simple video of a social situation or task to teach the student how to complete the task or behave appropriately in a particular situation. The student watches this video several times and can pause, rewind, or otherwise manipulate the video model. The video model provides a concrete set of parameters for responding that can be viewed repeatedly to rehearse a social situation (Cihak 2011). Portable video modeling has recently been shown to be effective in a few studies, using iPad, iPhone, and iTouch technology and provides a unique opportunity for greater flexibility and modeling in the authentic environment (Carnahan et al. 2012). Recent research suggests that video models are effective in a variety of environments, with a variety of individual and across a wide variety of skills including social, communication, play, joint attention, academic, motor, and vocational (Acar and Diken 2012). It is considered to be an evidence-based practice according to the National Professional Development Center on Autism Spectrum Disorders (Franzone and Collet-Klingenberg 2008).

Social Stories™ are simple stories that are read by the individual prior to entering a social situation. The story highlights at a minimum what the individual should try to say or do and how their behavior affects others. According to Carol Gray, the developer of Social Stories™, the following minimum elements must be present: (1) descriptive statements, which present factual information about the situation; (2) perspective statements, which provide information about the emotions and behavior of others; and (3) directive statements, which guide the student regarding strategies to try in the situation (Gray 2000; Kokina and Kern 2010). These stories are always written as

suggestions for the student in order to encourage flexibility and generalized responding. Social Stories™ are reviewed by the individual before the challenging situation presents itself in a calm and nonthreatening environment (e.g., the social story is best read to the child right before lunch if the cafeteria is a problematic environment). Social Stories™ have been recognized by The National Autism Center as an evidence-based practice (Leaf et al. 2016).

Teachers and other practitioners report that Social Stories™ are an easy, cost-effective way to increase appropriate behavior and teach social skills, especially skills which require taking the perspective of others. Research on Social Stories™ is limited, and in most research, Social Stories™ have been most commonly used as one element of an intervention package; however, that which does exist suggests that Social Stories™ are best as brief interventions. According to Kokina and Kern (2010), those developed using functional behavioral assessment (FBA) data and addressing problem behavior versus complex social skills seemed to be most effective. However, Karkhaneh et al. (2010) found in their review of the literature that Social Stories™ increased behaviors such as game playing, reading comprehension, and reading and comprehending social situations. Individuals with moderate social skill problems and moderate reading ability seemed to benefit more from Social Stories™ than those with low social skills and lower reading ability (Kokina and Kern 2010). No evidence is available at present regarding the generalization and long-term maintenance of the effects of Social Stories™. The evidence base for Social Stories™ is at present rather small; however, its use as a brief intervention for individuals with higher communication and social skills is promising and Social Stories™ are being used in a wide variety of intervention settings (Kassardjian et al. 2014).

Functional communication training (or FCT) is a strategy in which the individual with ASD is taught to request preferred items or activities. These items are often identified as those requested through problem behavior in the past. Based upon the hypothesis that the majority of problem behavior is communicative in nature, FCT provides for a

more socially appropriate way to communicate wants and needs for individuals who may have limited expressive skills. In most cases, some sort of alternative or augmentative communication system, such as paper-based picture symbols or voice output device, is selected for functional communication training. The individual is then taught to manipulate the communication system to request preferred items that in the past triggered problem behavior. Research supports that replacing problem behavior with appropriate requesting skills can decrease the amount of problem behavior in individuals with ASD (Mancil 2006). For example, teaching a child to request a break rather than engage in self-injury can result in a decrease in the problem behavior and an increase in the likelihood that the child will request a break appropriately (Carr and Durand 1985).

Even when problem behavior is not a concern, *augmentative and alternative communication devices* (AAC) may be used for individuals without spoken speech. These devices range from low-technology (such as the picture exchange communication system or PECS; Frost and Bondy 2002) to sophisticated high-tech devices. As with all accommodations and modifications, the system is based upon a thorough evaluation of the child's needs. A qualified speech-language pathologist often evaluates the child and determines the best intervention method for that individual.

The Picture Exchange Communication System (PECS) is a common and well-known system for communication by individuals with ASD, although its use is not limited to just individuals with ASD. In PECS, individuals are systematically instructed to (1) hand their communicative partner a picture to request items, (2) to recruit attention if the communicative partner is otherwise engaged, (3) discriminate between cards, (4) increase complexity and combinations of cards, and (5) use picture cards to comment on the environment (Frost and Bondy 2002). A 2009 review of the literature returned over 30 articles showing the effectiveness of PECS. Although all presented positive results, many abandoned the protocol in the early phases, leaving the later and more sophisticated aspects of PECS untested. The authors suggested a number of research questions,

including research into generalization of the effects of PECS, using PECS with high-tech communication devices, and investigations into what elements of PECS affect the development of spoken speech for those who acquire it (Sulzer-Azaroff et al. 2009). PECS also offers users an exchange that closely resembles the interactive manner of a speech-based conversation; as the user hands the selected PECS icon to the receiver, they have the opportunity for reinforcement and engagement, which can be highly motivating (Bondy 2012).

One concern that researchers and practitioners previously expressed is that the use of PECS will preclude or delay the use of speech in users; this seems to have been resolved. The evidence supports that AAC enhances rather than suppresses spoken speech. Adding a qualified Speech-Language Pathologist to the treatment team increases the probability of success (Bondy 2012; Lerna et al. 2012). In this manner, PECS should be thought of as both an alternative and augmentative system. PECS has also been shown to be effective in addressing deficits in joint attention, imitation, and play skills in young children with autism (Lerna et al. 2014).

At present, there is a debate as to whether visually based communication systems (such as PECS and picture symbol-based devices, also sometimes called aided devices; Mirenda 2003) are better for individuals with ASD than more transient devices and systems such as signed exact english (SEE; also called unaided devices). The rationale is that picture-based symbols use the visual channel (considered to be a strength of individuals with ASD) are more static (i.e., they do not disappear) and resemble their referents. Recent research suggests that neither system is best for teaching functional communication; rather, choosing a system is an individually based clinical decision. Limited evidence suggests that for children who have more advanced fine motor imitation skills, sign may have the advantage of being easier to learn and generalized. However, these conclusions are based upon small studies with identified methodological difficulties (Tincani 2004).

Voice output communication devices (VOCA; Mirenda 2003) are an often chosen modality for

individuals on the autism spectrum. A VOCA produces either a synthesized or recorded vocalization when an individual presses a button. These buttons usually have some sort of graphic display. For example, pushing the button with a picture of a drink may produce a sound file where a voice says, "I want a drink." According to Mirenda (2003), VOCAs have the potential to encourage more social interactions. Individuals may respond more readily to an auditory bid for interaction than a sign or pictorial one. There is some evidence that VOCA devices may assist the functional communication of individuals with ASD in school or clinic settings. However, the research on VOCA for children with ASD is somewhat sparse, especially in the area of generalization to home and community (Mirenda 2003). Still et al. (2014) reviewed the literature on Speech Generating Devices (SGDs) and found few studies on the use of tablets and apps. Most studies focused on a small number of functional responses such as requesting preferred items or attention. Therefore, it is unknown whether SGDs have the capability to promote large vocabularies in individuals with autism. It is important to understand the variables that predict success with more high-tech communication devices as well as what variables affect the selection of a functional communication system.

The continually evolving nature of technology requires a careful eye on the research to support it. Further research is critical in this age of iPads, iPods, and apps that increase communication for children with ASD. Tablet computers such as iPads have gained popularity in the past few years. They are readily available and used by people with and without disabilities, thereby reducing the stigma of having an assistive device. At least initially, parents and family members prefer tablets to PECS and other portable devices (Allen et al. 2016). The multifunctional nature of tablets allows for one small machine to deliver instructions, visual cues, video models and serve as a SGD (e.g., Vandermeer et al. 2013). It is important to note that just buying a tablet for a child with autism isn't enough to increase communication and other skills. But paired with individualized instruction and evidence-based practices for

teaching, the tablet can be a tool that enhances learning and social interaction (Allen et al. 2016).

Whatever functional communication system is used, the *representational competence* of a learner must be taken into consideration. Representational competence refers to the ability of a learner to infer meaning from some sort of symbol (Mineo Mollica 2003). Although line drawings are a popular way to create schedules, they are in fact very advanced and abstract representations. While individuals with autism have demonstrated the ability to derive meaning from abstract line drawings, they seem to rely more on the resemblance of that drawing to concrete items to derive meaning (Hartley and Allen 2014). For individuals who do not respond to typically used picture symbols, it might be better to use a more concrete representation such as legible photographs or, in some cases, the actual object (Mirenda and Locke 1989).

Setting Modifications

Setting modifications, such as work systems, visual schedules, and organization and structure, refer to arrangements of the physical settings encountered by learners with ASD so that salient features of the environment are visually clear and so that the individual with ASD clearly knows what to do in each environment. The majority of setting modifications are visual; however, some are auditory in nature or a combination of both (e.g., timers, transition music). Division TEACCH uses many setting modifications, such as color coding, stations, and visual cues, to reduce the amount of verbal interaction (Mesibov et al. 2005). The rationale for using visual setting modifications is that most individuals with ASD tend to more easily process stimuli visually rather than verbally.

Visual schedules function in much the same way as a “to-do list.” In a visual schedule, the events of a person’s day (or in most cases, a subset of the person’s day) are arranged on a schedule either horizontally or vertically (Hume 2008). Visual schedules may also be created for specific tasks. For example, the task of hand washing may be broken into its component parts and a visual of each part may be placed in the bathroom to prompt the appropriate hand-washing sequence.

It allows the individual with ASD to process and organize his or her time. In most cases, pictures are used to represent each of the activities; however, words can be used for higher-functioning individuals and actual items are sometimes used for individuals with less processing and representational skill. As each item is completed, the picture is removed and put away, signaling its completion.

When creating a visual schedule, it is important to consider the needs of the child. For example, a younger or less experienced learner may need to have a simple schedule that consists solely of what is happening now and what will happen next. A more advanced learner may be able to process a full day’s schedule. In the case of a task-based visual schedule, a less experienced learner might need a more detailed analysis, whereas the more advanced learner might be able to anticipate and chunk information into a single visual cue. Examples of how to create and use appropriate visuals are available (see Hodgdon 1998). Representational competence, described earlier in this article, should be considered as well.

Although visual cues are often associated with schedules and/or task sequences, photographs and other types of picture symbols can be used to prompt appropriate behavior in a number of ways. Many individuals with ASD respond well when there are visual cues in the environment that can help them understand how to respond and when. Labels with picture symbols may also be used to describe expectations in the environment. For example, a child’s place at the dinner table might be signaled with their picture on a placemat. The child is then instructed to find their picture and sit at that chair. West (2008) found that pictorial cues were more effective than verbal prompts in teaching a variety of skills to individuals with ASD. These skills both generalized to novel skills and maintained over time (West 2008). Knight et al. (2015) reviewed the literature on visual schedules and used the criteria established by Horner et al. (2005) to determine whether visual schedules should be considered an evidence-based practice to increase, maintain, and generalize skills for individuals with autism. Their findings support the consideration of visual schedules as an evidence-based practice for

individuals with autism and suggest that they can be used to teach transition behaviors, improve latency from task direction to task initiation, and decrease prompts required for transitions.

Visual cues seem to be effective for individuals with autism across the lifespan (Hume 2008) and for teaching a variety of skills. For example, Ganz et al. (2008) used visual social scripts and cues to “be quiet” to increase appropriate social statements and reduce perseverative statements in three elementary children with ASD. Modest generalization of appropriate untrained social statements was observed as well.

Spriggs et al. (2015) examined the use of iPad technology with visual schedules to support four students with autism as they transitioned between activities. They found that the portable technology motivated the students, was age appropriate, socially acceptable, was readily available, and afforded the opportunity to embed video modeling within the visual schedules. While two of the students became independent with their tasks using the visual schedules with embedded video modeling, two required the additional support of video chunking. All four students high levels of generalization when the video models were faded from the visual schedules.

Organization of elements in the environment is also considered to be an important part of environmental engineering for individuals with autism. Some advocate for more ambient lighting, reduced distractions, and less stimulating environments. These changes are introduced to reduce the amount of stimulating sensory input and allow the child with autism to focus upon those important parts of the environment (Kluth 2003).

Organizing the environment by activity and reducing clutter is another element of environmental engineering. For example, a teacher might use bookcases to cordon off the reading area and surround the area with red tape. The activity area may include active toys and may be surrounded by blue tape on the floor. These supports are designed to give the individual with autism more information about what is expected in certain areas of the classroom or home. These types of visual supports are considered to be critical to the structured teaching strategies developed by division TEACCH

(Mesibov et al. 2005). Some interventionists have called eliminating clutter and extraneous stimuli *sterilizing the environment* (Carbone 2003).

As with all interventions, these supports should be individualized; some individuals will respond better to quiet environments while others work better with background noise (Scheuermann and Hall 2008). Clear and consistent scheduling is also considered to be a critical environmental support. Many individuals with autism have difficulty processing open-ended instructions or activities; therefore, supporting the understanding of the beginning or end of an activity can help to increase appropriate behavior. Visual schedules can assist with this task, as well as timers and cues such as music during transitions (Scheuermann and Hall 2008).

Any organizational strategy must be flexible; that is, the individual with ASD should be prepared in advance for changes or out-of-the-ordinary events. For example, a birthday party might be put on the calendar, or a surprise icon might be placed on the visual schedule to signal an unexpected event. While providing clear visual cues and eliminating extraneous stimuli from the environment are considered to be best for individuals with ASD, there is little research to support effectiveness at this time. Several literature searches with terms such as “classroom organization and autism,” “environment and autism,” and “environmental organization and autism” returned no empirical articles. Studies that investigate the effects of a carefully organized physical environment need to be conducted.

Activity-Based Strategies

Activity-based strategies are those that are embedded into everyday activities and provide modifications or accommodations for individuals with ASD. Some examples include supporting academic, modifying task, incorporating preference and choice, and prompting techniques.

A well-known intervention from TEACCH is the *work system*. In the work system, a task is broken down into its component parts and visuals are created that communicate to the learner with ASD (1) what is to be done, (2) how much work needs to be done, (3) when he or she is finished, and (4) what the person should do once the work

is complete. The goal of the work system is to increase independence in children with ASD and decrease prompts. It has been shown to be effective with elementary and middle school students and can be used to teach a myriad of tasks including vocational, self-help, and transitions (Hume and Carnahan 2008). Boyd et al. (2014) found that preschool-aged children with autism made gains or reductions in autism characteristics using the TEACCH system. An interesting finding in this study was that students with lower cognitive abilities demonstrated the most significant gains.

In the first study to examine the effects of the TEACCH work system on task accuracy and the use of newly acquired skills, Hume et al. (2012) found that the TEACCH work system increased student accuracy and reduced prompt dependency for three first grade students with autism. These results generalized across settings including the general education setting, where the TEACCH systems were never used. This finding offers promising evidence that skills could be taught in a special education setting using the TEACCH work systems and would generalize to inclusive, general education settings.

Academic supports and task modifications are those supports that allow an individual to be successful at a task. Some of the core characteristics of ASD (e.g., communication, restricted, and repetitive interests) can directly affect academic achievement (Fleury et al. 2014). These supports might include visual cues, such as reducing the amount of visual stimuli to isolate visual tasks (such as folding a piece of paper to cover up additional questions on a test). Other visual supports include highlighting pertinent information, color coding salient information, etc. Other modifications may include frequent proactive breaks (i.e., breaks set at preset intervals as opposed to in response to agitation) or using a task analysis, where a task is broken into its component parts and taught in a systematic fashion. Considerations for creating academic and task modifications are available in Vaughn and Bos (2012). Visual supports such as the visual schedules described above can also be used to modify tasks.

More students identified as having ASD are continuing their education beyond K-12.

Accommodations for individuals with ASD mirror those offered to others with disabilities in higher education, such as extended time and alternative formats of assignments (Gelbar et al. 2014). This points to an increased importance of teaching to generalization in postsecondary environments. Many of the antecedent interventions presented throughout this article, such as priming, clear expectations, and visual supports, are beneficial with older learners with ASD (Fleury et al. 2014). More research is needed, however, on how to best support learners with ASD in secondary and post-secondary settings.

Incorporating preference and choice can increase the success of an individual with ASD. Those items that a child is more likely to choose are typically the most reinforcing (Mason and Egel 1995) yet individuals with autism are making fewer daily choices than those without autism (Mehling and Tasse 2015). Using preferred items for teaching may also increase the likelihood of attention. Giving individuals simple choices, such as the order in which to complete a task, can decrease problem behavior and increase more socially appropriate behavior (Smeltzer et al. 2009; Ulke-Kurkcuoglu and Kircaali-Iftar 2010) and improve social outcomes such as community inclusion and interpersonal relationships (Mehling and Tasse 2015). Rispoli et al. (2013) found that although rates of challenging behavior were lower for all choice conditions than for no choice conditions, offering students with ASD choices across activities (i.e., the choice of a math or reading task) resulted in a greater decrease in challenging behaviors than offering choice within activities (i.e., the choice of using a pen or a pencil).

There are many different types of assessments of preference. Often, teachers and caregivers will give families a checklist or open-ended questionnaire to complete. Professionals, however, should be aware of respondent bias when questionnaires are used. Free-operant preference assessments consist of giving a child free access to a limited number of items. Those manipulated or consumed most frequently are considered to be the most preferred. In a forced-choice preference assessment, items are presented in pairs and the child is asked to choose which one they would like to manipulate or

consume. It is important to note that different types of preference assessments may yield different results; therefore, multiple modalities of preference assessment may be in order when finding preferred items proves difficult (Kodak et al. 2009).

Related to choice is the notion of environmental enrichment. In environmental enrichment, additional items are provided that allow the child to be appropriately engaged in activities as opposed to engaging in repetitive, stereotypic behavior (Neitzel 2009). Visual cues, items to hold, and timers may also be effective interventions for difficulties with transitions and wait time. Ringdahl et al. (1997) found that systematically providing environmental enrichment in the form of preferred items to manipulate reduced self-injurious behavior in three participants with developmental disabilities. A recent review of the literature found environmental enrichment to be an effective strategy for reducing stereotypic behavior in individuals with developmental delays (Lancioni et al. 2009).

Prompts are additional instructions provided to a child with autism that allow them to be successful in activities. The prompts and prompting sequence is tailored to the learner with ASD. Common strategies include the least-to-most prompting sequence, where prompts are introduced in order of intrusiveness (typically from verbal to hand-over-hand guidance) until the child is successful. Most-to-least prompting reverses the sequence and systematically fades prompts as the learner needs less and less assistance to be successful. In time delay, the teacher gradually increases the time between the instruction and the prompt until the learner anticipates the prompt (Alberto and Troutman 2009). The evidence base supports the use of prompting for most behaviors and individuals of all ages (Neitzel and Wolery 2009).

Future Directions

Anecdotal and empirical evidence support the use of environmental engineering for individuals with ASD; however, additional empirical evidence is needed for specific environmental engineering strategies. Antecedent-based interventions are

popular among practitioners because they are easy to implement, considered to be cost-effective and are perceived to be effective. Although there is much evidence that antecedent-based interventions are being utilized with individuals on the autism spectrum, much more research should be devoted to investigating why and how these interventions are effective.

The best-researched environmental engineering interventions seem to be those surrounding functional communication training, especially PECS, and priming. Many interventions have a rapidly growing evidence base, such as Social Stories and video modeling. Others, such as environmental arrangements and academic supports, have little evidence to support them. There is a marked paucity of literature on supporting adolescents and adults, especially those choosing postsecondary education. It seems as if, in many ways, “the devil is in the details.” At present, which interventions work best for specific students and the details of implementation seem to remain the domain of clinical judgment. Evidence-based guidance for clinicians may assist them in making more efficient, individualized decisions for intervention. Considering environmental engineering is deemed important in many respected books and training manuals, and more evidence is needed to support its use.

See Also

- ▶ [Developmental Continuum \(Principles of TEACCH\)](#)
- ▶ [Functional Analysis](#)
- ▶ [Functional Assessment and Curriculum for Teaching Everyday Routines](#)
- ▶ [Functional Communication Training](#)
- ▶ [Video Modeling/Video Self-Modeling](#)
- ▶ [Visual Supports](#)

References and Reading

- Acar, C., & Diken, I. H. (2012). Reviewing instructional studies conducted using video modeling to children with autism. *Educational Sciences: Theory and Practice*, 12(4), 2731–2735.

- Alberto, P. A., & Troutman, A. C. (2009). *Applied behavior analysis for teachers* (8th ed.). Upper Saddle River: Merrill-Prentice-Hall.
- Allen, M. L., Hartley, C., & Cain, K. (2016). iPads and the use of "apps" by children with autism spectrum disorder: Do they promote learning? *Frontiers in Psychology*, 7, 3–7.
- Bijou, S. W., Petersen, R. F., & Ault, M. H. (1968). A method to integrate descriptive and experimental field studies at the level of data and empirical concepts. *Journal of Applied Behavior Analysis*, 1, 175–191.
- Bondy, A. (2012). The unusual suspects: Myths and misconceptions associated with PECS. *The Psychological Record*, 62, 789–816.
- Boyd, B. A., Hume, K., McBee, M. T., Allesandri, M., Gutierrez, A., Johnson, L., Sperry, L., & Odom, S. L. (2014). Comparative efficacy of LEAF, TEACCH, and non-model-specific special education programs for preschoolers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44, 366–380.
- Bregman, J. D., Zager, D., & Gerdtz, J. (2005). Behavioral interventions. In F. R. Volkmar, R. Paul, A. Klin, & A. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.
- Carbone, V. J. (2003). *Teaching verbal behavior to children with autism and related disabilities*. Unpublished Workshop Manual.
- Carnahan, C. R., Basham, J. D., Christman, J., & Hollingshead, A. (2012). Overcoming challenges: Going mobile with your own video models. *Teaching Exceptional Children*, 45(2), 50–59.
- Carr, E. G., & Durand, V. M. (1985). Reducing behavior problems through functional communication training. *Journal of Applied Behavior Analysis*, 18, 111–126.
- Cihak, D. F. (2011). Comparing pictorial and video modeling activity schedules during transitions for students with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5(1), 433–441.
- Fleury, V. P., Hedges, S., Humer, K., Browder, D. M., Thompson, J. L., Fallin, K., El Zein, F., Reutbush, C. K., & Vaughn, S. (2014). Addressing the academic needs of adolescents with autism spectrum disorder in secondary education. *Remedial and Special Education*, 35, 68–79.
- Franzone, E., & Collet-Klingenberg, L. (2008). *Overview of video modeling*. Chapel Hill: The National Professional Development Center on Autism Spectrum Disorders, Frank Porter Graham Child Development Institute, The University of North Carolina.
- Frost, L. A., & Bondy, A. S. (2002). *The picture exchange communication system training manual* (2nd ed.). Newark: Pyramid Educational Consultants.
- Ganz, J. B., Kaylor, M., Bourgeois, B., & Hadden, K. (2008). The impact of social scripts and visual cues on verbal communication in three children with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 23(2), 79–94.
- Gelbar, N. W., Smith, I., & Reichow, B. (2014). Systematic review of articles describing supports of individuals with autism enrolled in college and university programs. *Journal of Autism and Developmental Disorders*, 44, 2593–2601.
- Gray, C. (2000). *The new social stories book*. Arlington: Future Horizons.
- Hartley, C., & Allen, M. L. (2014). Intentions versus resemblance: Understanding pictures in typical development and autism. *Cognition*, 131, 44–59.
- Hodgdon, L. A. (1998). *Visual strategies for improving communication volume 1: Practical supports for school and home*. Troy: QuirkRoberts.
- Horner, R. H., Carr, E. G., Halle, J., McGee, G., Odom, S., & Wolery, M. (2005). The use of single-subject research to identify evidence-based practice in special education. *Exceptional Children*, 71, 165–179.
- Hume, K. (2008). *Overview of visual supports*. Chapel Hill: The National Professional Development Center on Autism Spectrum Disorders, Frank Porter Graham Child Development Institute, The University of North Carolina.
- Hume, K., & Carnahan, C. R. (2008). *Overview of structured work systems*. Chapel Hill: The National Professional Development Center on Autism Spectrum Disorders, Frank Porter Graham Child Development Institute, The University of North Carolina.
- Hume, K., Plavnick, J., & Odom, S. L. (2012). Promoting task accuracy and independence in students with autism across educational setting through the use of individual work systems. *Journal of Autism and Developmental Disorders*, 42, 2084–2099.
- Hurl, K., Wightman, J., Haynes, S. N., & Virues-Ortega, J. (2016). Does a pre-intervention functional assessment increase intervention effectiveness? A meta-analysis of within-subject interrupted time series studies. *Clinical Psychology Review*, 47, 71–84.
- Iwata, B. A., Dorsey, M. F., Slifer, K. J., Bauman, K. E., & Richman, G. S. (1994). Toward a functional analysis of self-injury. *Journal of Applied Behavior Analysis*, 27, 197–209.
- Karkhaneh, M., Clark, B., Ospina, M. B., Seida, J. C., Smith, V., & Hartling, L. (2010). Social stories [TM] to improve social skills in children with autism spectrum disorder: A systematic review. *Autism: The International Journal of Research and Practice*, 14(6), 641–662.
- Kassardjian, A., Leaf, J. B., Ravid, D., Leaf, J. A., Alcalay, A., Dale, S., Tsuji, K., Taubman, M., Leaf, R., McEachin, J., & Oppenheim-Leaf, M. L. (2014). Comparing the teaching interaction procedure to social stories™: A replication study. *Journal of Autism and Developmental Disorders*, 44, 2329–2340.
- Kluth, P. (2003). *You're going to love this kid! Teaching students with autism in the inclusive classroom*. Baltimore: Paul H. Brookes.
- Knight, V., Sartini, E., & Spriggs, A. D. (2015). Evaluating visual activity schedules as evidence-based practice for individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 45, 157–178.
- Kodak, T., Fisher, W. W., Kelley, M. E., & Kisamore, A. (2009). Comparing preference assessments: Selection-versus duration-based preference assessment

- procedures. *Research in Developmental Disabilities: A Multidisciplinary Journal*, 30(5), 1068–1077.
- Koegel, L. K., Koegel, R. L., Frea, W., & Green-Hopkins, I. (2003). Priming as a method of coordinating educational services for students with autism. *Language, Speech, and Hearing Services in Schools*, 34(3), 228–235.
- Kokina, A., & Kern, L. (2010). Social story[TM] interventions for students with autism spectrum disorders: A meta-analysis. *Journal of Autism and Developmental Disorders*, 40(7), 812–826.
- Lancioni, G. E., Singh, N. N., O'Reilly, M. F., & Sigafoos, J. (2009). An overview of behavioral strategies for reducing hand-related stereotypes of persons with severe to profound intellectual and multiple disabilities: 1995–2007. *Research in Developmental Disabilities: A Multidisciplinary Journal*, 30(1), 20–43.
- Leaf, J. B., Mitchell, E., Townley-Cochran, D., McEachin, J., Taubman, M., & Leaf, R. (2016). Comparing social stories™ to cool versus not cool. *Education and Treatment of Children*, 39(2), 173–186.
- Lerna, A., Esposito, D., Conson, M., Russo, L., & Massagli, A. (2012). Social-communicative effects of the picture exchange communication system (PECS) in autism Spectrum disorders. *International Journal of Language & Communication Disorders*, 47(5), 609–617.
- Lerna, A., Esposito, D., Conson, M., & Massagli, A. (2014). Long-term effects of PECS on social-communicative skills of children with autism spectrum disorders. *International Journal of Language & Communication Disorders*, 49(4), 478–485.
- Mace, F. C., Mauro, B. C., Boyajian, A. E., & Eckert, T. L. (1997). Effects of reinforcer quality on behavioral momentum: Coordinated applied and basic research. *Journal of Applied Behavior Analysis*, 30, 1–20.
- Mancil, G. R. (2006). Functional communication training: A review of the literature related to children with autism. *Education and Training in Developmental Disabilities*, 41(3), 213–224.
- Mason, S. A., & Egel, A. L. (1995). What does Amy like? Using a mini-reinforcer assessment to increase student participation in instructional activities. *Teaching Exceptional Children*, 28(1), 42–45.
- Mehling, M. H., & Tasse, M. J. (2015). Impact of choice on social outcomes of adults with ASD. *Journal of Autism and Developmental Disorders*, 45, 1588–1602.
- Mesibov, G. B., Shea, V., & Schopler, E. (2005). *The TEACCH approach to autism spectrum disorders*. New York: Kluwer Academic/Plenum.
- Mineo Mollica, B. (2003). Representational competence. In J. Light, D. Beukelman, & J. Reichle (Eds.), *Communicative competence for individuals who use AAC* (pp. 107–146). Baltimore: Paul H. Brookes.
- Mirenda, P. (2003). Toward functional augmentative and alternative communication for students with autism: Manual signs, graphic symbols, and voice output communication aids. *Language, Speech, and Hearing Services in Schools*, 34, 203–216.
- Mirenda, P., & Locke, P. A. (1989). A comparison of symbol transparency on nonspeaking persons with intellectual disabilities. *The Journal of Speech and Hearing Disorders*, 54, 131–140.
- Neitzel, J. (2009). *Overview of antecedent-based interventions*. Chapel Hill: The National Professional Development Center on Autism Spectrum Disorders, Frank Porter Graham Child Development Institute, The University of North Carolina.
- Neitzel, J., & Wolery, M. (2009). *Overview of prompting*. Chapel Hill: The National Professional Development Center on Autism Spectrum Disorders, Frank Porter Graham Child Development Institute, The University of North Carolina.
- Orellana, L. H., Martinez-Sanchiz, S., & Silvestre, F. J. (2014). Training adults and children with an autism spectrum disorder to be compliant with a clinical dental assessment using a TEACCH-based approach. *Journal of Autism and Developmental Disorders*, 44, 776–785.
- Quill, K. (1995). *Teaching children with autism: Strategies to enhance communication and socialization*. Albany: Thomson.
- Radley, K. C., & Dart, E. H. (2016). Antecedent strategies to promote child and adolescent compliance with adult requests: A review of the literature. *Clinical Child Family Psychology Review*, 19, 39–54.
- Ringdahl, J. E., Vollmer, T. R., Marcus, B. A., & Roane, H. S. (1997). An analogue evaluation of environmental enrichment: The role of stimulus preference. *Journal of Applied Behavior Analysis*, 30(2), 203–216.
- Rispoli, M., Lang, R., Neely, L., Camargo, S., Hutchins, N., Davenport, K., & Goodwyn, F. (2013). A comparison of within- and across- activity choices for reducing challenging behavior in children with autism spectrum disorders. *Journal of Behavioral Education*, 22, 66–83.
- Scheuermann, B. K., & Hall, J. A. (2008). *Positive behavioral supports for the classroom*. Upper Saddle River: Pearson Merrill Prentice Hall.
- Smeltzer, S. S., Graff, R. B., Ahearn, W. H., & Libby, M. E. (2009). Effect of choice of task sequence on responding. *Research in Autism Spectrum Disorders*, 3(3), 734–742.
- Spriggs, A. D., Knight, V., & Sherrow, L. (2015). Talking picture schedules: Embedding video models into visual activity schedules to increase independence for students with ASD. *Journal of Autism and Developmental Disabilities*, 45, 3846–3861.
- Stichter, J. P., Randolph, J. K., & Kay, D. (2009). The use of structural analysis to develop antecedent-based interventions for students with autism. *Journal of Autism and Developmental Disorders*, 39, 883–896.
- Still, K., Rehfeldt, R. A., Whelan, R., May, R., & Dymond, S. (2014). Facilitating requesting skills using high-tech augmentative and alternative communication devices with individuals with autism spectrum disorders: A systematic review. *Research in Autism Spectrum Disorders*, 8, 1184–1199.
- Sulzer-Azaroff, B., Hoffman, A. O., Horton, C. B., Bondy, A., & Frost, L. (2009). The picture exchange communication system (PECS): What do the data say? *Focus on Autism and Other Developmental Disabilities*, 24, 89–103.

- Tincani, M. (2004). Comparing the picture exchange communication system and sign language training for children with autism. *Focus on Autism and Other Developmental Disabilities*, 19(3), 152–163.
- Touchette, P. E., McDonald, R. F., & Langer, S. N. (1985). A scatter plot for identifying stimulus control of problem behavior. *Journal of Applied Behavior Analysis*, 18, 343–351.
- Ulke-Kurkcuoglu, B., & Kircaali-Iftar, G. (2010). A comparison of the effects of providing activity and material choice to children with autism spectrum disorders. *Journal of Applied Behavior Analysis*, 43(4), 717–721.
- Umbreit, J., Ferro, J., Liaupsin, C. J., & Lane, K. L. (2007). *Functional behavioral assessment and function-based intervention: An effective, practical approach*. Upper Saddle River: Pearson Merrill Prentice Hall.
- Vandermeer, J., Beamish, W., Milford, T., & Lang, W. (2013). iPad presented social stories for young children with autism. *Developmental Rehabilitation*, 1751–8423, 1–7.
- Vaughn, S., & Bos, C. (2012). *Strategies for teaching students with learning and behavior problems* (8th ed.). Upper Saddle River: Merrill.
- Wacker, D. P., Berg, W. K., & Harding, J. W. (2006). The evolution of antecedent-based interventions. In J. K. Luiselli (Ed.), *Antecedent assessment and intervention: Supporting children and adults with developmental disabilities in community settings*. Baltimore: Brookes.
- Wehman, P. (2006). *Life beyond the classroom: Transition strategies for young people with disabilities* (4th ed.). Baltimore: Paul H. Brookes.
- West, E. A. (2008). Effects of verbal cues versus pictorial cues on the transfer of stimulus control for children with autism. *Focus on Autism and Other Developmental Disabilities*, 23(4), 229–241.

Environmental Risk Factors for Autism

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Definition

Environmental risk factors for autism may be broadly defined as any nongenetic contributor

to the risk of autism and include reproductive-, dietary-, chemical-, and demographic-related exposures.

Historical Background and Evidence for a Role of the Environment

Environmental factors have been explored as contributing to the development of autism spectrum disorders (ASD) since the condition was first characterized in 1943 by Dr. Leo Kanner, who stated that children with autism had “very few really warm-hearted fathers and mothers” (Kanner 1943). Soon after, the term “refrigerator mother” was coined, which blamed the child’s behaviors on emotionless parenting (Bettelheim 1972). This explanation has since been disproved, and though the focus shifted from environmental factors for some time, research from other areas has clearly demonstrated a role of the environment in ASD.

Early twin studies in the 1970s and 1980s demonstrating non-concordance of diagnosis in monozygotic twins provided support that the environment, in addition to genetics, plays a key part in the etiology of ASD (Folstein and Rutter 1977; Steffenburg et al. 1989). Over time, estimates of environmental contribution to ASD have increased, with current estimates indicating that the environment is responsible for between 50% to 55% of ASD genetic variance (Hallmayer et al. 2011; Sandin et al. 2014).

Evidence of structural and functional brain differences in individuals with ASD point to the prenatal and perinatal developmental period as a key vulnerable window for risk of ASD. The critical stages of neurodevelopment within early gestation provide a period of increased susceptibility to environmental exposures which may confer risk of ASD. Two historical examples have demonstrated the role of environmental prenatal exposure on ASD risk. The use of thalidomide for morning sickness in the 1950s demonstrated a marked increase in the risk of ASD, far greater than estimates from that decade (Lotter 1967). Prenatal use of valproic acid, an antiepileptic drug, has also been shown to increase ASD risk, in children whose mothers used anticonvulsants

during pregnancy, as reported in multiple studies (Moore et al. 2000; Rasalam et al. 2005). In addition to these historical examples, the increasing prevalence of ASD lends support for continued investigation into environmental risk factors. While improved awareness, social acceptance, and shifts in diagnostic criteria explain part of this increase, analyses have suggested steep rises in incidence that cannot be fully explained by these artifacts of societal changes (Hertz-Picciotto and Delwiche 2009; King and Bearman 2009). One study found that up to 40% of the rise in prevalence in Denmark could be due to yet unknown environmental causes (Hansen et al. 2015).

Current Knowledge

Prenatal and Perinatal Factors

Maternal Environmental Markers

One well-studied and consistently replicated risk factor is parental age. Increased maternal and paternal age are each independently associated with ASD risk. Maternal age greater than 35 is often associated with a substantial increase in risk compared to women younger than 25 according to a meta-analysis (Sandin et al. 2012). Paternal age greater than 40 is also associated with increased risk of ASD in offspring (Durkin et al. 2008). In addition to being a proxy for age-related genetic changes in germ cells, environmental explanations for this risk range from increasing toxin loads in both mother and father to increased risk of autoimmune disorders and rates of pregnancy complications in mothers (Shelton et al. 2010).

A few environmental factors may represent markers or proxies, as opposed to true risk factors. These include seasonality, or season of conception or birth, which has been associated with ASD risk (Zerbo et al. 2011), with some variation in findings. This may be due to seasonal exposures like pesticide use or infections. Inter-pregnancy interval (IPI), or the time between the live birth of one child and the conception of another pregnancy, may also represent a proxy measure. Several population-based investigations reported

that shorter IPIs of less than 12 months had odds ratios ranging from 1.44 to 3.39 compared to IPIs greater than 36 months (Cheslack-Postava et al. 2011, 2014; Durkin et al. 2015). This association may be a result of the depletion of essential maternal nutrients, such as folate, which can take up to a year postpartum to replenish (O'Rourke et al. 2000). Other mechanisms potentially responsible for this association include maternal stress and inflammation; however, no studies to date have examined these factors as potential mediators of the IPI-ASD association. Long IPIs of greater than 120 months have also been associated with ASD risk (Cheslack-Postava et al. 2014; Durkin et al. 2015), but overall findings remain inconsistent.

Immune Factors

A growing body of evidence points toward maternal immune system response during the prenatal period as an etiologic area of interest. One study reports that maternal hospitalizations due to infection are associated with a 30% increase in ASD risk, regardless of which trimester the hospitalization occurred (Lee et al. 2015). Other epidemiological investigations support these findings (Zerbo et al. 2015). However, evidence distinguishing infection type (viral vs. bacterial) and timing remains inconsistent.

A recent study examined maternal influenza and ASD in a population of 196,929 children and found that there was no significant association between ASD and physician visits for influenza or an influenza vaccination (Zerbo et al. 2017). The CHARGE (CHildhood Autism Risks from Genetics and the Environments) study (Hertz-Picciotto et al. 2006) also found no association with maternal influenza but did find an increase in odds of ASD with maternal fever in the second trimester (Zerbo et al. 2013). Consistent with this finding, a large prospective Norwegian study observed more than a tripling of risk associated with three or more fever episodes after 12 weeks' gestation (Hornig et al. 2018).

Regardless of infection type, there are several studies which describe altered immune marker concentrations during gestation with increased risk of ASD. Women with elevated levels of circulating cytokines and chemokines, such as

interleukin-6 (IL-6), during pregnancy were more likely to have a child with both ASD and intellectual disability (Jones et al. 2017). IL-6 is secreted within the body to stimulate immune response and is known to aid in fighting infection; unlike most interleukins, it readily crosses the placenta into the growing fetus's circulation (Zaretsky et al. 2004). Investigators also found that high concentrations of C-reactive protein, an inflammatory biomarker, within maternal serum during pregnancy increased the risk of ASD by 43% in a sample of nearly 1.2 million mother-child pairs (Brown et al. 2014).

Medication Usage

In addition to valproic acid, recent studies suggest that prenatal use of other medications may also influence ASD risk. Selective serotonin reuptake inhibitors (SSRIs), commonly used to treat depression, have been associated with ASD in several studies. Mothers taking SSRIs during early pregnancy had between a 1.4- and 1.8-fold increase in risk of having offspring with ASD (Brown et al. 2017). It is possible that maternal depression and mental illness, both associated with increased ASD risk in the child and indicated for use of SSRIs, are responsible for this finding (Brown et al. 2017) (a situation known as "confounding by indication" where the condition rather than the treatment represents the true risk factor). However, further research is needed to clarify the associations between antidepressants, maternal mental illness, and ASD.

An important consideration in studies of medication use and ASD is weighing the potential risks versus the benefits, as untreated conditions may also influence risk, and public health messages need to be carefully presented to pregnant women only after research has consistently demonstrated associations in separately replicated studies.

Recently, prenatal acetaminophen use has also been examined in association with ASD. Two large studies observed that higher maternal use of acetaminophen during pregnancy was associated with increased risk of ASD or autism-like symptoms, as well as attention deficits and hyperactivity (Avella-Garcia et al. 2016; Liew et al.

2016). Both studies found that earlier and more frequent use during pregnancy was associated with higher risk. However, these findings require replication and further study, careful selection of comparison group, and clearer details on dosage and frequency of use.

Pregnancy Complications

Several pregnancy complications have been repeatedly associated with increased risk of ASD. Metabolic complications, like gestational diabetes, have been associated with a consistent increase in risk of ASD in several studies (Lyll et al. 2012; Xiang et al. 2015; Li et al. 2016; Connolly et al. 2016), as have preeclampsia and maternal obesity (Walker et al. 2015; Sanchez et al. 2017). Numerous studies have implicated complications in the perinatal period, with risk of ASD. Delivery complications such as fetal distress, birth asphyxia, and placental insufficiency are all potentially associated with increased ASD risk (Hultman et al. 2002; Gardener et al. 2011), though associations with individual complications are less consistent (Getahun et al. 2017). For neonatal complications, a range of factors have also been examined in association with ASD, with greater consistency for increased risk associated with low birth weight (Mann et al. 2010), preterm birth (Buchmayer et al. 2009), and potentially, complications relating to hypoxia (Gardener et al. 2011). Evidence suggests a combination of several of these factors in the prenatal and perinatal periods, leading to suboptimal birth, could additively be responsible for a significant association with ASD (Lyll et al. 2012). Impaired fetal development assessed at birth may reflect prenatal maternal obstetric complications, and both of these may be related to further upstream etiologic factors, including environmental insults.

Folic Acid and Related Nutrients

The beneficial effects of folic acid supplementation in reducing neural tube defects are well-established, but the relationship of pre- and perinatal folic acid and multivitamin supplementation on ASD outcome has been more recently suggested. Most studies evaluating the association

between periconceptional intake of folic acid and ASD reported a reduced risk of ASD or autistic traits. Folic acid supplementation was shown to confer a greater risk reduction among mothers and children with genetic susceptibility for less efficient folate metabolism (Schmidt et al. 2012). Additionally, recent studies report that high folic acid intake, particularly during the first trimester, reduced the risk of ASD due to air pollution and prenatal pesticide exposure (Schmidt et al. 2017; Goodrich et al. 2018). The evidence based on measured folate levels during pregnancy and ASD risk is still unclear since studies have reported nonsignificant associations but concurrent significant associations with folic acid supplementation (Braun et al. 2014a; Steenweg-de Graaff et al. 2014). A potential explanation for this lack of association with the measured levels could be that high folate levels later in pregnancy offer no protection against ASD, as suggested by three studies reporting protective effects only from higher folic acid intake and supplementation within the window of a few months before to about 6 weeks after conception (Schmidt et al. 2012; Suren et al. 2013; Bjork et al. 2017). Thus, there is a need for additional studies to identify the critical windows of exposure that link biologically measured folate to ASD.

Other Prenatal Nutrients

Limited research has investigated other maternal dietary factors in association with ASD, though the importance of maternal diet in neurodevelopment is well-established. There is increasing interest in the role of vitamin D deficiency in the pathogenesis of ASD, given the role of vitamin D in immune system functioning and neurodevelopment. Two studies from Nordic countries, one that assessed clinical vitamin D deficiency in pregnancy and another which assessed the level of 25[OH]D₃, a biomarker of vitamin D exposure, in midpregnancy reported increased association with ASD and Social Responsiveness Scale (SRS) scores, respectively (Magnusson et al. 2016; Vinkhuyzen et al. 2018). Additionally, studies that measured 25[OH]D₂ in maternal blood at the first and second trimesters, and in neonatal blood spots, reported increased

odds of ASD diagnosis with low prenatal vitamin D levels (Chen et al. 2016; Vinkhuyzen et al. 2017; Fernell et al. 2015). Two studies examining prenatal iron intake reported a reduced risk of ASD with iron supplementation (Schmidt et al. 2014); however, reduction was also seen for multivitamin intake with or without iron or folic acid (DeVilbiss et al. 2017). Maternal dietary intake of polyunsaturated fatty acids (PUFAs) has been associated with reduced risk of ASD in one US cohort (Lyll et al. 2013), while a cohort study from Spain found that maternal seafood (including ocean fish) consumption, a key source for these fatty acids, was associated with fewer ASD symptoms as measured by the Childhood Asperger Syndrome Test (Julvez et al. 2016). However, an analysis of prenatal plasma levels of major classes of PUFAs, omega 6 and omega 3, found an increase in autistic traits as measured by a shortened version of the SRS for the former and no association for the latter (Steenweg-de Graaff et al. 2016). Prenatal fish consumption or fish oil supplement use was not associated with ASD in another large Norwegian cohort study (Suren et al. 2013), demonstrating conflicting findings across the available studies.

Continued research into these and other dietary factors may be fruitful in identifying modifiable risk factors for ASD, as well as factors that interact with other ASD risk factors (Schmidt et al. 2017; Goodrich et al. 2018).

Alcohol and Smoking

Both alcohol and tobacco smoke are known risk factors of adverse perinatal and neonatal outcomes. The current evidence shows a lack of association between prenatal exposure to tobacco and ASD. Analysis using national-level ASD monitoring data in the US found no overall associations and a suggestive increased relationship with higher-functioning ASD (Kalkbrenner et al. 2012), but a more recent meta-analysis using studies from multiple countries showed null associations (Rosen et al. 2015). There are fewer studies assessing the effect of prenatal alcohol exposure on ASD, but the most recent reports based on US and Danish data show null associations (Eliassen et al. 2010; Singer et al. 2017). For these lifestyle

factors, underreporting is likely a major problem; furthermore, many of the studies have not adequately adjusted for confounders. Studies with biomarkers and proper control for confounding are needed to fully resolve the potential impact of these substances on ASD.

Environmental Chemicals

The Toxic Substances Control Act's (TSCA) registry of chemicals created by the US Environmental Protection Agency (EPA) currently lists ~85,000 chemicals registered for commercial use, with new alternatives for problematic substances added each year. Despite historical evidence of teratogenic and neurodevelopmental problems due to mercury and lead, only a fraction of these chemicals have been tested for adverse effects on neurodevelopment. Some of these chemicals can cause downstream effects on fetal neurodevelopment through maternal hormonal or immune system-mediated pathways, whereas others can directly cross the placental barrier to affect the growing fetus. An increasing number of studies are focusing on exposure to these chemicals in association with ASD, given suggestive findings for other related outcomes.

Air Pollution

Prenatal exposure to air pollution has emerged as a risk factor for ASD over the last decade. Several epidemiological studies have evaluated the risk from estimated levels of hazardous air pollutants (HAPs), criteria air pollutants (nitrogen dioxide (NO₂), ozone, particulate matter less than 2.5 μ (PM_{2.5}), 10 μ in diameter (PM₁₀), metals such as lead and cadmium), and exposure to traffic-related pollution on ASD. The majority of recent studies have reported increases in risk of ASD with higher levels of estimated air pollution or proxies for it (such as distance to highways), though evidence regarding which specific component(s) of pollution are responsible for the observed association is far less consistent. In addition, recent studies have also started to explore modification of these effects by genetic variation.

The current epidemiological studies of HAPs and ASD have found moderately increased risk for ASD with industrial solvents. Maternal

prenatal exposure to chlorinated solvents and some heavy metals (mercury, lead, chromium, manganese, nickel) are associated with increased odds of ASD (Windham et al. 2006; Kalkbrenner et al. 2010; Roberts et al. 2013; von Ehrenstein et al. 2014; Talbott et al. 2015). These reports also implicate several toxicants as potential risk factors for ASD, but only few (styrene, solvents, diesel particulates, mercury, and lead) have been replicated. The most recent research on air toxins reported elevated associations with vehicular emission-related air toxins and industrial solvents (Kalkbrenner et al. 2018). Recent studies have used complex statistical techniques and adjusted for co-pollutants in analysis but utilize exposure assessment generated by the US National Air Toxics Assessment, limiting the spatial and temporal interpretability of results.

Most US-based studies that assessed PM_{2.5} have reported that the levels of fine particulate matter are associated with a moderate increase in the risk of ASD. Higher levels of PM₁₀, NO₂, and ozone are also implicated, but results are less consistent across studies. Within the CHARGE study, the presence of a genetic variant near the MET gene locus, coupled with high NO₂ exposure (Volk et al. 2014), increased the risk of ASD. In addition, increased copy number variation (CNV) burden, combined with elevated exposure to ozone, also resulted in much higher risk of ASD (Kim et al. 2017). Research using samples from other states have shown similar elevations in the risk for PM_{2.5}, and PM₁₀, ozone, and nitrogen oxides (Raz et al. 2015; Talbott et al. 2015). These studies used EPA air monitoring data-based dispersion models, traffic density, distance to roadways, and estimates based on residential history as well as land-use regression models.

In contrast to US-based studies, the evidence is more limited from other countries. A recent twin study that assessed nitrogen oxides and PM₁₀ based on road traffic estimated dispersion modeling did not find any significant associations with ASD (Gong et al. 2014). Similarly, one large study based on four European cohorts that measured these toxicants and PM_{2.5} based on land-use regression models (Guxens et al. 2016) also found no significant association with autistic traits.

A recent study in Israel that assessed NO₂ based on an optimized dispersion model found increased ASD risk for postnatal exposure but decreased risk for early prenatal exposure (Raz et al. 2018). The variation between US and international studies may stem from differences in outcome assessment, residual confounding in the US due to social factors, and the different mixtures of pollutants between nations. A recent review and commentary concluded that though there was potential for residual and unmeasured confounding in studies of air pollution and ASD, the likelihood of a true increase in risk associated with this factor was strong (Weisskopf et al. 2015). However, the focus of this discussion was primarily on confounding factors that tend not to change much over time intervals when air pollution changes. Further investigation of confounding is warranted.

Researchers have suggested that immune activation, neuroinflammation, and oxidative stress could be potential pathways for air pollution and its components to affect the developing brain and ultimately impact ASD risk. Although there is explosive growth in the quantity and quality of air pollution-related research, further work is needed. Future epidemiological studies need to consider exposure measurement limitations and residual confounding from SES, and factors that may vary in concert with air pollution including meteorologic and other variables develop methods to model pollutant mixtures, and identify critical windows of exposure relevant to the outcome, as well as further consider potential genetic interactions.

Pesticides

The toxic effects to the human body from acute pesticide exposures are well known, and work has also demonstrated adverse effects on neurodevelopment. Prenatal exposure to organophosphate pesticides (OP), a class of insecticides with wide-ranging agricultural uses, has been associated with abnormal reflexes (Engel et al. 2007), poorer mental development (Eskenazi et al. 2007) and a range of behavioral, cognitive, and neurologic deficits across many cohorts and study populations (Munoz-Quezada et al. 2013;

Gonzalez-Alzaga et al. 2014; Rauh et al. 2015). These exposures can be measured as individual metabolites in maternal biosamples, or through residential proximity to agricultural areas, which has been validated using air monitoring of pesticide applications (Wofford et al. 2014). A recent study used both exposure sources to assess associations with ASD traits and found that only prenatal urine total dialkyl phosphates (an OP metabolite) were associated with an increase in ASD traits as measured by the SRS (Sagiv et al. 2018). However, two other studies showed no associations with total OP metabolites and SRS scores (Furlong et al. 2014; Millenson et al. 2017). Only a few studies have examined associations with clinically diagnosed ASD: one, using prenatal measurements of OP metabolites, found no statistically significant associations in a very small study reporting preliminary findings (Philippat et al. 2018), although nonsignificant sex differences, with a higher risk among girls, but not boys, were noted. The first study to examine ASD in association with OPs and other agricultural pesticides using residential proximity reported significantly elevated odds of ASD associated with exposures in the top quartile for bifenthrin, a pyrethroid pesticide, and organophosphates (Roberts et al. 2007). Another study using proximity to agricultural pesticide applications found elevated risk for ASD, which was clinically confirmed by standardized diagnostic instruments. A significantly elevated risk was also noted for third trimester exposure to pyrethroids and second-trimester exposure to chlorpyrifos, the most commonly used agricultural OP pesticide (Shelton et al. 2014). Chlorpyrifos has been banned of neurodevelopmental harm, and recently the U.S. EPA proposed to revoke tolerances for residues in food, which would amount to a ban on agricultural uses, but subsequently reversed its position and announced there would be no such cancellation. In August 2018, EPA was ordered to finalize the ban by a U.S. Circuit Court of Appeals. In the meantime, chlorpyrifos is still being used in agriculture. Moreover, a recent study reported an interaction between exposure to these pesticides in pregnancy and folic acid, with the OP-related increased risk confined to those having low folic

acid intake (Schmidt et al. 2017); this interaction suggests that high folic acid may protect against the neurodevelopmental toxicity of pesticides, though replication of the finding is needed.

Another class of pesticides, organochlorines (OC), has also been examined in association with ASD. In the Roberts et al study based on residential proximity to agricultural pesticides (mentioned above), several OC pesticides were significantly associated with ASD (Roberts et al. 2007). In two studies using prenatal biomarkers of OC pesticides, results have been conflicting. Maternal prenatal serum concentration of one OC pesticide, trans-nonachlor, was associated with more ASD symptoms in a Cincinnati-based cohort study (Braun et al. 2014b), but a California-based study examining risk of diagnosed ASD (Lyll et al. 2017) found no association.

Considering that toxicity of pesticides has been known for a long time, surprisingly few studies have evaluated this exposure with ASD. Additional research is needed to clarify and replicate existing results and should consider the potential for exposure misclassification due to short half-lives of some OP pesticide metabolites, use sensitive and objective exposure measurement proxies, continue to explore time windows of sensitivity, and consider the potential effect modification of pesticides from other environmental and genetic factors.

Endocrine-Disrupting Chemicals

Endocrine-disrupting chemicals (EDCs) represent a class of substances (both persistent and nonpersistent chemicals) that interfere with the human endocrine system. Though some of these chemicals especially persistent EDCs are banned or have regulated use, the persistent nature of certain classes means that exposure is still relevant. As they are ubiquitously present in the environment, these substances have been studied for their effects on multiple neurodevelopmental outcomes over the last two decades.

OC pesticides, described above, are also known for their endocrine disrupting properties. Other EDC compounds examined in association with ASD include polychlorinated biphenyls

(PCBs), polybrominated diphenyl ethers (PBDEs), phthalates and bisphenol A (BPA). Two US and two international studies have evaluated the risk of ASD or measures of ASD symptoms in association with prenatal exposure to PCBs, but findings are contradictory. In a small pilot study, the sum of PCBs evaluated (Cheslack-Postava et al. 2013) was associated with an increased risk of ASD. Another study that measured 24 PCBs along with other EDCs in maternal blood reported inverse association with PCB-178 and SRS scores (Braun et al. 2014b). A German cohort study that summarized PCB congeners using the World Health Organization's defined toxic equivalency factors reported that PCB levels were associated with lower autistic mannerisms in their overall sample, and specifically among girls (Nowack et al. 2015). The most recent report measuring PCBs in second-trimester serum samples reported elevated risk of ASD with two congeners (PCB 153 and PCB 138/158) (Lyll et al. 2017), but further clarification of the role of PCBs in ASD is needed.

Though evidence links PBDE exposure to other adverse neurodevelopmental outcomes (Eskenazi et al. 2013; Chen et al. 2014), findings for ASD are limited. Only two studies have examined prenatal levels of PBDE in association with ASD, with conflicting findings. One study showed a marginally significant positive relationship with ASD symptoms as measured by the SRS (Braun et al. 2014b), and another found that elevated levels of certain PBDE congeners (PBDE-100, PBDE-153) were inversely associated with ASD diagnosis (Lyll et al. 2017; Traglia et al. 2017). In the latter study, there was also evidence of sexual dimorphism, with suggested, though not statistically significant, increases in risk with higher PBDE levels for girls and effects in the inverse direction for boys. It is possible that the inverse associations may be due to maternal and/or fetal genetic factors related to PBDE metabolism, as suggested in a follow-up to this study (Traglia et al. 2017).

The first study suggesting a link between phthalates and ASD was based on presence of PVC flooring in parents' bedroom (Larsson et al. 2009). However, in a study using measurements

of phthalates in household dust, no association was found with ASD, but associations were observed with several co-morbidities, including hyperactivity-impulsivity and inattention (Philippat et al. 2015). Only two studies have evaluated prenatal phthalate concentrations in biomarkers in association with ASD phenotype; one reported increased risk of SRS-measured ASD symptoms with increasing concentrations of low-molecular-weight phthalate metabolite concentrations in third-trimester urine (Miodovnik et al. 2011), and the other found no associations (Braun et al. 2014b). Few studies have examined BPA or perfluorinated compounds in association with ASD, and the results are inconsistent. Of note, phthalates and BPA are short-lived compounds, in comparison with PBDEs, PCBs, perfluorinated compounds and OC pesticides, all of which are persistent compounds with half-lives in the human body of months or years. Capturing exposures through the use of biologic markers is a greater challenge in the studies of compounds that are quickly excreted (phthalates, BPA, and the OP pesticides).

Other Environmental Chemicals

Like lead, methylmercury is a well-established neurotoxin with the primary source of human exposure through seafood consumption, but no associations with ASD have been described (van Wijngaarden et al. 2013; McKean et al. 2015). Several studies have suggested that mercury in air pollution is associated with an increased risk of ASD (Windham et al. 2006; Roberts et al. 2013; Lam et al. 2016). Although there are many reports on the differences in heavy metal levels between existing ASD cases and neurotypical controls, the primary hypothesis tested in these studies involves metabolic clearance of the toxicant rather than a causal mechanism of ASD (e.g., measurement of prenatal exposure), limiting interpretations. Innovations in this area, including the potential use of shed deciduous teeth or other novel biomatrices, may aid measurement of low-level prenatal exposures (Arora et al. 2017), but continued work is required.

Gene Environment Interactions

ASD is a complex neurodevelopmental condition, and evidence suggests significant environmental and genetic contributions to its etiology. However, because of the high degree of heterogeneity in both the environmental and genetic factors, it is possible that the combined effect of multiple genes and environmental factors contribute to the etiology over and beyond the main effects of individual genes or specific environmental factors. Over the last decade, there is a growing interest in evaluating gene-by-environment (GxE) interaction in ASD. Only a few studies have investigated GxE interaction in ASD to date. The first study reported significantly higher risk of ASD for children of mothers who did not take prenatal vitamins and had functional genetic variants involving inefficient conversion to folate or transmethylation in one-carbon metabolism (Schmidt et al. 2012). As mentioned before, mothers with the CC variant of the MET gene and high air pollution exposure had a higher risk of ASD compared to those with low pollution exposure and both CG and GG genotypes (Volk et al. 2014). Another study reported that children with ASD-associated CNVs and mothers with a history of infection during pregnancy had elevated ASD symptom severity compared to those without these factors (Mazina et al. 2015). CNV duplication and higher total CNV burden have also been shown to interact with ozone exposure in pregnancy until the second year of life to increase the risk of ASD (Kim et al. 2017). ASD-associated CNVs in children have been linked with other non-pollutant prenatal exogenous stressors as well. A recent study observed that exposure to first-trimester diagnostic ultrasound interacted with the presence of CNVs in autistic male children, increasing ASD symptom severity as compared to autistic male children with CNVs and no first-trimester ultrasound exposure (Webb et al. 2017).

Though of interest, the study of GxE in ASD is hampered by the need for both genetic and environmental data in the same study. In addition, as the potential mechanisms and possible interactions increase, large sample sizes are necessary to accurately estimate associations. Future research

harnessing collaborations across projects and linking datasets is expected to address these issues and expand the consideration of interactions in ASD (Hertz-Picciotto et al. 2018).

Conclusions and Future Directions

The complex and substantial heterogeneity in ASD phenotype and etiology makes studying suspected environmental factors challenging. Because ASD is a rare condition in the general population, few large-scale, prospective, population-based studies investigating environmental factors from preconception to diagnosis have been conducted. Replication of results for specific risk factors, clarification of mechanisms, and consideration of interactions are all key areas that studies of environmental risk factors for ASD will need to address in the future. However, considerable progress has been made in the past decade in our understanding of the role of the environment in ASD. This work has highlighted air pollution, parental age, the maternal intrauterine environment, various immune-related factors, and metabolic conditions as highly replicated risk factors for ASD and has provided suggestive findings warranting further consideration of other factors.

Several of the environmental risk factors discussed here are also associated with other adverse neurodevelopmental outcomes. Many substances cause multiple pathophysiological effects, and a better understanding of how common exposures can lead to diverse outcomes is needed. Though risk factors are often examined independently, exposures do not act in isolation. Considering the combined effects of multiple exposures, including not only GxE but also environment x environment interactions (to include nutrition) and exposure to complex chemical mixtures, may prove fruitful in gaining a better understanding of pathways. Additionally, incorporating objective biomarkers of exposure during biologically relevant time windows of neurodevelopment may serve this aim of clarifying mechanisms. Gold-standard biological measures of exposures, especially those that are not transient, could

supplement survey measures and improve the validity and precision of findings.

Another consideration for future work includes careful attention to timing. The prenatal period represents a key window of neurodevelopmental susceptibility, and some exposures may have even narrower windows of effect. Longitudinal pregnancy cohorts yield some of the richest sources of viable data, though these have been rare in ASD due to practical issues in assembling large enough study samples. One approach to mitigate this has been the use of high-risk pregnancy cohorts, which follow mothers who have already given birth to a child with ASD through a subsequent pregnancy (Newschaffer et al. 2012) – these studies have been critical in allowing for prospective investigation of a wide range of environmental risk factors for ASD and incorporation of biomarkers. However, sample sizes tend to be small, and the extent to which risk factors may differ in these families with perhaps increased genetic loading is unclear.

ASD clearly has a complex etiology, with multiple risk factors playing significant roles. Further investigations, using prospective design, large sample sizes, biological measures, and considering combined exposures, will be key to furthering our understanding of etiology and underlying mechanisms.

References and Reading

- Arora, M., Reichenberg, A., Willfors, C., Austin, C., Gennings, C., Berggren, S., Lichtenstein, P., Anckarsater, H., Tammimies, K., & Bolte, S. (2017). Fetal and postnatal metal dysregulation in autism. *Nature Communications*, 8, 15493.
- Avella-Garcia, C. B., Julvez, J., Fortuny, J., Rebordosa, C., García-Esteban, R., Galán, I. R., Tardón, A., Rodríguez-Bernal, C. L., Iñiguez, C., & Andiarena, A. (2016). Acetaminophen use in pregnancy and neurodevelopment: Attention function and autism spectrum symptoms. *International Journal of Epidemiology*, 45(6), 1987–1996.
- Bettelheim, B. (1972). *Empty fortress*. Simon and Schuster, New York.
- Bjork, M., Riedel, B., Spigset, O., Veiby, G., Kolstad, E., Daltveit, A. K., & Gilhus, N. E. (2017). Association of folic acid supplementation during pregnancy with the risk of autistic traits in children exposed to antiepileptic drugs in utero. *JAMA Neurology*, 75(2): 160–168.

- Braun, J. M., Froehlich, T., Kalkbrenner, A., Pfeiffer, C. M., Fazili, Z., Yolton, K., & Lanphear, B. P. (2014a). Brief report: Are autistic-behaviors in children related to prenatal vitamin use and maternal whole blood folate concentrations? *Journal of Autism and Developmental Disorders*, 44(10), 2602–2607.
- Braun, J. M., Kalkbrenner, A. E., Just, A. C., Yolton, K., Calafat, A. M., Sjodin, A., Hauser, R., Webster, G. M., Chen, A., & Lanphear, B. P. (2014b). Gestational exposure to endocrine-disrupting chemicals and reciprocal social, repetitive, and stereotypic behaviors in 4- and 5-year-old children: The HOME study. *Environmental Health Perspectives*, 122(5), 513–520.
- Brown, A. S., Sourander, A., Hinkka-Yli-Salomäki, S., McKeague, I. W., Sundvall, J., & Surcel, H.-M. (2014). Elevated maternal C-reactive protein and autism in a national birth cohort. *Molecular Psychiatry*, 19(2), 259.
- Brown, H. K., Hussain-Shamsy, N., Lunsy, Y., Dennis, C., & Vigod, S. N. (2017). The association between antenatal exposure to selective serotonin reuptake inhibitors and autism: A systematic review and meta-analysis. *The Journal of Clinical Psychiatry*, 78, e48.
- Buchmayer, S., Johansson, S., Johansson, A., Hultman, C. M., Sparén, P., & Cnattingius, S. (2009). Can association between preterm birth and autism be explained by maternal or neonatal morbidity? *Pediatrics*, 124(5), e817–e825.
- Chen, A., Yolton, K., Rauch, S. A., Webster, G. M., Hornung, R., Sjodin, A., Dietrich, K. N., & Lanphear, B. P. (2014). Prenatal polybrominated diphenyl ether exposures and neurodevelopment in U.S. children through 5 years of age: The HOME study. *Environmental Health Perspectives*, 122(8), 856–862.
- Chen, J., & Xin, K. et al. (2016). Lower maternal serum 25 (OH) D in first trimester associated with higher autism risk in Chinese offspring. *J Psychosom Res*, 89, 98–101.
- Cheslack-Postava, K., Liu, K., & Bearman, P. S. (2011). Closely spaced pregnancies are associated with increased odds of autism in California sibling births. *Pediatrics*, 127(2), 246–253.
- Cheslack-Postava, K., Rantakokko, P. V., Hinkka-Yli-Salomäki, S., Surcel, H. M., McKeague, I. W., Kiviranta, H. A., Sourander, A., & Brown, A. S. (2013). Maternal serum persistent organic pollutants in the Finnish Prenatal Study of Autism: A pilot study. *Neurotoxicology and Teratology*, 38, 1–5.
- Cheslack-Postava, K., Suominen, A., Jokiranta, E., Lehti, V., McKeague, I. W., Sourander, A., & Brown, A. S. (2014). Increased risk of autism spectrum disorders at short and long interpregnancy intervals in Finland. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(10), 1074–1081.e1074.
- Connolly, N., Anixt, J., Manning, P., Ping, I. L. D., Marsolo, K. A., & Bowers, K. (2016). Maternal metabolic risk factors for autism spectrum disorder—an analysis of electronic medical records and linked birth data. *Autism Res* 9:829–837.
- DeVilbiss, E. A., Magnusson, C., Gardner, R. M., Rai, D., Newschaffer, C. J., Lyall, K., Dalman, C., & Lee, B. K. (2017). Antenatal nutritional supplementation and autism spectrum disorders in the Stockholm youth cohort: Population based cohort study. *BMJ*, 359, j4273.
- Durkin, M. S., Maenner, M. J., Newschaffer, C. J., Lee, L.-C., Cunniff, C. M., Daniels, J. L., Kirby, R. S., Leavitt, L., Miller, L., & Zahorodny, W. (2008). Advanced parental age and the risk of autism spectrum disorder. *American Journal of Epidemiology*, 168(11), 1268–1276.
- Durkin, M. S., DuBois, L. A., & Maenner, M. J. (2015). Inter-pregnancy intervals and the risk of autism spectrum disorder: Results of a population-based study. *Journal of Autism and Developmental Disorders*, 45(7), 2056–2066.
- Eliassen, M., Tolstrup, J. S., Nybo Andersen, A. M., Gronbaek, M., Olsen, J., & Strandberg-Larsen, K. (2010). Prenatal alcohol exposure and autistic spectrum disorders – A population-based prospective study of 80,552 children and their mothers. *International Journal of Epidemiology*, 39(4), 1074–1081.
- Engel, S. M., Berkowitz, G. S., Barr, D. B., Teitelbaum, S. L., Siskind, J., Meisel, S. J., Wetmur, J. G., & Wolff, M. S. (2007). Prenatal organophosphate metabolite and organochlorine levels and performance on the Brazelton Neonatal Behavioral Assessment Scale in a multiethnic pregnancy cohort. *American Journal of Epidemiology*, 165(12), 1397–1404.
- Eskenazi, B., Marks, A. R., Bradman, A., Harley, K., Barr, D. B., Johnson, C., Morga, N., & Jewell, N. P. (2007). Organophosphate pesticide exposure and neurodevelopment in young Mexican-American children. *Environmental Health Perspectives*, 115(5), 792–798.
- Eskenazi, B., Chevrier, J., Rauch, S. A., Kogut, K., Harley, K. G., Johnson, C., Trujillo, C., Sjodin, A., & Bradman, A. (2013). In utero and childhood polybrominated diphenyl ether (PBDE) exposures and neurodevelopment in the CHAMACOS study. *Environmental Health Perspectives*, 121(2), 257–262.
- Fernell, E., & Bejerot, S. et al. (2015). Autism spectrum disorder and low vitamin D at birth: a sibling control study. *Mol Autism*, vol 6, p.3.
- Folstein, S., & Rutter, M. (1977). Infantile autism: A genetic study of 21 twin pairs. *Journal of Child Psychology and Psychiatry*, 18(4), 297–321.
- Furlong, M. A., Engel, S. M., Barr, D. B., & Wolff, M. S. (2014). Prenatal exposure to organophosphate pesticides and reciprocal social behavior in childhood. *Environment International*, 70, 125–131.
- Gardener, H., Spiegelman, D., & Buka, S. L. (2009). Prenatal risk factors for autism: Comprehensive meta-analysis. *The British Journal of Psychiatry*, 195(1), 7–14.
- Gardener, H., Spiegelman, D., & Buka, S. L. (2011). Perinatal and neonatal risk factors for autism:

- A comprehensive meta-analysis. *Pediatrics*, 128(2), 344–355.
- Getahun, D., Fassett, M. J., Peltier, M. R., Wing, D. A., Xiang, A. H., Chiu, V., & Jacobsen, S. J. (2017). Association of perinatal risk factors with autism spectrum disorder. *American Journal of Perinatology*, 7(03), 295–304.
- Gong, T., Almqvist, C., Bölte, S., Lichtenstein, P., Anckarsäter, H., Lind, T., Lundholm, C., & Pershagen, G. (2014). Exposure to air pollution from traffic and neurodevelopmental disorders in Swedish twins. *Twin Research and Human Genetics*, 17(6), 553–562.
- Gonzalez-Alzaga, B., Lacasana, M., Aguilar-Garduno, C., Rodriguez-Barranco, M., Ballester, F., Rebagliato, M., et al. (2014). A systematic review of neurodevelopmental effects of prenatal and postnatal organophosphate pesticide exposure. *Toxicol Lett*, 230(2), 104–21. <https://doi.org/10.1016/j.toxlet.2013.11.019>. PubMed PMID: 24291036.
- Goodrich, A. J., Volk, H. E., Tancredi, D. J., McConnell, R., Lurmann, F. W., Hansen, R. L., & Schmidt, R. J. (2018). Joint effects of prenatal air pollutant exposure and maternal folic acid supplementation on risk of autism spectrum disorder. *Autism Research*, 11(1), 69–80.
- Guxens, M., Ghassabian, A., Gong, T., Garcia-Esteban, R., Porta, D., Giorgis-Allemand, L., Almqvist, C., Aranbarri, A., Beelen, R., & Badaloni, C. (2016). Air pollution exposure during pregnancy and childhood autistic traits in four European population-based cohort studies: The ESCAPE project. *Environmental Health Perspectives*, 124(1), 133.
- Hallmayer, J., Cleveland, S., Torres, A., Phillips, J., Cohen, B., Torigoe, T., Miller, J., Fedele, A., Collins, J., Smith, K., Lotspeich, L., Croen, L. A., Ozonoff, S., Lajonchere, C., Grether, J. K., & Risch, N. (2011). Genetic heritability and shared environmental factors among twin pairs with autism. *Archives of General Psychiatry*, 68, 1095. Jul 4. [Epub ahead of print].
- Hansen, S. N., Schendel, D. E., & Parner, E. T. (2015). Explaining the increase in the prevalence of autism spectrum disorders: The proportion attributable to changes in reporting practices. *JAMA Pediatrics*, 169(1), 56–62.
- Hertz-Picciotto, I., & Delwiche, L. (2009). The rise in autism and the role of age at diagnosis. *Epidemiology*, 20(1), 84–90.
- Hertz-Picciotto, I., Croen, L. A., Hansen, R., Jones, C. R., Van de Water, J., & Pessah, N. (2006). The charge study: an epidemiological investigation of genetic and environmental factors contributing to autism. *Environmental Health Perspectives*, 114(7), 1119–25.
- Hertz-Picciotto, I., Schmidt, R. J., & Krakowiak, P. (2018). Understanding environmental contributions to autism: Causal concepts and the state of science. *Autism Research*, 11, 554.
- Hornig, M., Bresnahan, M., Che, X., Schultz, A., Ukaigwe, J., Eddy, M., Hirtz, D., Gunnes, N., Lie, K. K., & Magnus, P. (2018). Prenatal fever and autism risk. *Molecular Psychiatry*, 23(3), 759.
- Hultman, C. M., Sparen, P., & Cnattingius, S. (2002). Perinatal risk factors for infantile autism. *Epidemiology*, 13(4), 417–423.
- Jones, K. L., Croen, L. A., Yoshida, C. K., Heuer, L., Hansen, R., Zerbo, O., DeLorenze, G. N., Kharrazi, M., Yolken, R., & Ashwood, P. (2017). Autism with intellectual disability is associated with increased levels of maternal cytokines and chemokines during gestation. *Molecular Psychiatry*, 22(2), 273.
- Julvez, J., Mendez, M., Fernandez-Barres, S., Romaguera, D., Vioque, J., Llop, S., Ibarluzea, J., Guxens, M., Avella-Garcia, C., Tardon, A., Riano, I., Andiaarena, A., Robinson, O., Arijia, V., Esnaola, M., Ballester, F., & Sunyer, J. (2016). Maternal consumption of seafood in pregnancy and child neuropsychological development: A longitudinal study based on a population with high consumption levels. *American Journal of Epidemiology*, 183(3), 169–182.
- Kalkbrenner, A. E., Daniels, J. L., Chen, J. C., Poole, C., Emch, M., & Morrissey, J. (2010). Perinatal exposure to hazardous air pollutants and autism spectrum disorders at age 8. *Epidemiology*, 21(5), 631–641.
- Kalkbrenner, A. E., Braun, J. M., Durkin, M. S., Maenner, M. J., Cunniff, C., Lee, L. C., Pettygrove, S., Nicholas, J. S., & Daniels, J. L. (2012). Maternal smoking during pregnancy and the prevalence of autism spectrum disorders, using data from the autism and developmental disabilities monitoring network. *Environmental Health Perspectives*, 120(7), 1042–1048.
- Kalkbrenner, A. E., Windham, G. C., Zheng, C., McConnell, R., Lee, N. L., Schauer, J. J., Thayer, B., Pandey, J., & Volk, H. E. (2018). Air toxics in relation to autism diagnosis, phenotype, and severity in a US Family-Based Study. *Environmental Health Perspectives*, 126(3), 037004–037004.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2(3), 217–250.
- Kim, D., Volk, H., Girirajan, S., Pendergrass, S., Hall, M. A., Verma, S. S., Schmidt, R. J., Hansen, R. L., Ghosh, D., & Ludena-Rodriguez, Y. (2017). The joint effect of air pollution exposure and copy number variation on risk for autism. *Autism Research*, 10(9), 1470–1480.
- King, M., & Bearman, P. (2009). Diagnostic change and the increased prevalence of autism. *International Journal of Epidemiology*, 38(5), 1224–1234.
- Lam, J., Sutton, P., Kalkbrenner, A., Windham, G., Halladay, A., Koustas, E., ... & Woodruff, T. (2016). A systematic review and meta-analysis of multiple airborne pollutants and autism spectrum disorder. *PLoS One*, 11(9), e0161851.
- Larsson, M., Weiss, B., Janson, S., Sundell, J., & Bornehag, C.-G. (2009). Associations between indoor environmental factors and parental-reported autistic spectrum disorders in children 6–8 years of age. *Neurotoxicology*, 30(5), 822–831.
- Lee, B. K., Magnusson, C., Gardner, R. M., Blomström, Å., Newschaffer, C. J., Burstyn, I.,

- Karlsson, H., & Dalman, C. (2015). Maternal hospitalization with infection during pregnancy and risk of autism spectrum disorders. *Brain, Behavior, and Immunity*, 44, 100–105.
- Li, M., Fallin, M. D., Riley, A., Landa, R., Walker, S. O., & Silverstein, M., et al. (2016). The association of maternal obesity and diabetes with autism and other developmental disabilities. *Pediatrics* 137:1–10.
- Liew, Z., Ritz, B., Virk, J., & Olsen, J. (2016). Maternal use of acetaminophen during pregnancy and risk of autism spectrum disorders in childhood: A Danish national birth cohort study. *Autism Research*, 9(9), 951–958.
- Lotter, V. (1967). Epidemiology of autistic conditions in young children. *Social Psychiatry*, 1(4), 163–173.
- Lyall, K., Pauls, D. L., Spiegelman, D., Ascherio, A., & Santangelo, S. L. (2012). Pregnancy complications and obstetric suboptimality in association with autism spectrum disorders in children of the Nurses' Health Study II. *Autism Research*, 5(1), 21–30.
- Lyall, K., Munger, K. L., O'Reilly, E. J., Santangelo, S. L., & Ascherio, A. (2013). Maternal dietary fat intake in association with autism spectrum disorders. *American Journal of Epidemiology*, 178(2), 209–220.
- Lyall, K., Croen, L. A., Sjödin, A., Yoshida, C. K., Zerbo, O., Kharrazi, M., & Windham, G. C. (2017). Polychlorinated biphenyl and organochlorine pesticide concentrations in maternal mid-pregnancy serum samples: Association with autism spectrum disorder and intellectual disability. *Environmental Health Perspectives*, 125(3), 474.
- Magnusson, C., Lundberg, M., Lee, B. K., Rai, D., Karlsson, H., Gardner, R., Kosidou, K., Arver, S., & Dalman, C. (2016). Maternal vitamin D deficiency and the risk of autism spectrum disorders: Population-based study. *British Journal of Psychiatry Open*, 2(2), 170–172.
- Mann, J. R., McDermott, S., Bao, H., Hardin, J., Gregg, A. (2010). Pre-eclampsia, birth weight, and autism spectrum disorders. *J Autism Dev Disord*, 40 (5), 548–554.
- Mazina, V., Gerdts, J., Trinh, S., Ankenman, K., Ward, T., Dennis, M. Y., Girirajan, S., Eichler, E. E., & Bernier, R. (2015). Epigenetics of autism-related impairment: Copy number variation and maternal infection. *Journal of Developmental and Behavioral Pediatrics*, 36(2), 61–67.
- McKean, S. J., Bartell, S. M., Hansen, R. L., Barfod, G. H., Green, P. G., & Hertz-Picciotto, I. (2015). Prenatal mercury exposure, autism, and developmental delay, using pharmacokinetic combination of newborn blood concentrations and questionnaire data: A case control study. *Environmental Health*, 14(1), 62.
- Millenson, M. E., Braun, J. M., Calafat, A. M., Barr, D. B., Huang, Y.-T., Chen, A., Lanphear, B. P., & Yoltson, K. (2017). Urinary organophosphate insecticide metabolite concentrations during pregnancy and children's interpersonal, communication, repetitive, and stereotypic behaviors at 8 years of age: The home study. *Environmental Research*, 157, 9–16.
- Miodovnik, A., Engel, S. M., Zhu, C., Ye, X., Soorya, L. V., Silva, M. J., Calafat, A. M., & Wolff, M. S. (2011). Endocrine disruptors and childhood social impairment. *Neurotoxicology*, 32(2), 261–267.
- Moore, S., Turnpenny, P., Quinn, A., Glover, S., Lloyd, D., Montgomery, T., & Dean, J. (2000). A clinical study of 57 children with fetal anticonvulsant syndromes. *Journal of Medical Genetics*, 37(7), 489–497.
- Munoz-Quezada, M. T., Lucero, B. A., Barr, D. B., Steenland, K., Levy, K., Ryan, P. B., et al. (2013). Neurodevelopmental effects in children associated with exposure to organophosphate pesticides: a systematic review. *Neurotoxicology*, 39, 158–68. <https://doi.org/10.1016/j.neuro.2013.09.003>. PubMed PMID: 24121005; PubMed Central PMCID: PMC3899350.
- Newschaffer, C. J., Croen, L. A., Fallin, M. D., Hertz-Picciotto, I., Nguyen, D. V., Lee, N. L., Berry, C. A., Farzadegan, H., Hess, H. N., Landa, R. J., Levy, S. E., Massolo, M. L., Meyerer, S. C., Mohammed, S. M., Oliver, M. C., Ozonoff, S., Pandey, J., Schroeder, A., & Shedd-Wise, K. M. (2012). Infant siblings and the investigation of autism risk factors. *Journal of Neurodevelopmental Disorders*, 4, 7.
- Nowack, N., Wittsiepe, J., Kasper-Sonnenberg, M., Wilhelm, M., & Schölmerich, A. (2015). Influence of low-level prenatal exposure to PCDD/Fs and PCBs on empathizing, systemizing and autistic traits: Results from the Duisburg birth cohort study. *PLoS One*, 10(6), e0129906.
- O'Rourke, K. M., Redlinger, T. E., & Waller, D. K. (2000). Declining levels of erythrocyte folate during the postpartum period among Hispanic women living on the Texas-Mexico border. *Journal of Women's Health & Gender-Based Medicine*, 9(4), 397–403.
- Philippat, C., Bennet, D. H., Krakowiak, P., Rose, M., Hwang, H.-M., & Hertz-Picciotto, I. (2015). Phthalate concentrations in house dust in relation to autism spectrum disorder and developmental delay in the Childhood Autism Risks from Genetics and the Environment (CHARGE) study. *Environmental Health*, 14, 56.
- Philippat, C., Barkoski, J., Tancredi, D. J., Elms, B., Barr, D. B., Ozonoff, S., Bennett, D. H., & Hertz-Picciotto, I. (2018). Prenatal exposure to organophosphate pesticides and risk of autism spectrum disorders and other non-typical development at 3 years in a high-risk cohort. *International Journal of Hygiene and Environmental Health*, 221(3), 548–555.
- Rasalam, A., Hailey, H., Williams, J. H. G., Moore, S., Turnpenny, P., Lloyd, D. J., & Dean, J. C. (2005). Characteristics of fetal anticonvulsant syndrome associated autistic disorder. *Developmental Medicine and Child Neurology*, 47(8), 551–555.
- Rauh, V. A., Garcia, W. E., Whyatt, R. M., Horton, M. K., Barr, D. B., & Louis, E. D. (2015). Prenatal exposure to the organophosphate pesticide chlorpyrifos and childhood tremor. *Neurotoxicology*, 51, 80–6. <https://doi.org/10.1016/j.neuro.2015.09.004>. PubMed PMID: 26385760.
- Raz, R., Roberts, A. L., Lyall, K., Hart, J. E., Just, A. C., Laden, F., & Weiskopf, M. G. (2015). Autism spectrum disorder and particulate matter air pollution before, during, and after pregnancy: A nested case-

- control analysis within the Nurses' Health Study II Cohort. *Environmental Health Perspectives*, 123(3), 264–270.
- Raz, R., Levine, H., Pinto, O., Broday, D. M., & Weisskopf, M. G. (2018). Traffic related air pollution and autism spectrum disorder: A population based nested case-control study in Israel. *American Journal of Epidemiology*, 187(4), 717–725.
- Roberts, E. M., English, P. B., Grether, J. K., Windham, G. C., Somberg, L., & Wolff, C. (2007). Maternal residence near agricultural pesticide applications and autism spectrum disorders among children in the California Central Valley. *Environmental Health Perspectives*, 115(10), 1482–1489.
- Roberts, A., Lyall, K., Hart, J. E., Laden, F., Just, A. C., Bobb, J. F., Koenen, K. C., Ascherio, A., & Weisskopf, M. G. (2013). Perinatal air pollutant exposures and autism spectrum disorder in the children of Nurses' Health Study II participants. *Environmental Health Perspectives*, 121(8), 978–984.
- Rosen, B. N., Lee, B. K., Lee, N. L., Yang, Y., & Burstyn, I. (2015). Maternal smoking and autism spectrum disorder: A meta-analysis. *Journal of Autism and Developmental Disorders*, 45(6), 1689–1698.
- Sagiv, S. K., Harris, M. H., Gunier, R. B., Kogut, K. R., Harley, K. G., Deardorff, J., Bradman, A., Holland, N., & Eskenazi, B. (2018). Prenatal organophosphate pesticide exposure and traits related to autism spectrum disorders in a population living in proximity to agriculture. *Environmental Health Perspectives*, 126(4), 047012.
- Sanchez, C., Barry, C., Sabhlok, A., Russell, K., Majors, A., Kollins, S., & Fuemmeler, B. (2017). Maternal pre-pregnancy obesity and child neurodevelopmental outcomes: A meta-analysis. *Obesity Reviews*, 19, 464.
- Sandin, S., Hultman, C. M., Klevzon, A., Gross, R., MacCabe, J. H., & Reichenberg, A. (2012). Advancing maternal age is associated with increasing risk for autism: A review and meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(5), 477–486.e471.
- Sandin, S., Lichtenstein, P., Kuja-Halkola, R., Larsson, H., Hultman, C. M., & Reichenberg, A. (2014). The familial risk of autism. *JAMA*, 311(17), 1770–1777.
- Schmidt, R. J., Tancredi, D. J., Ozonoff, S., Hansen, R. L., Hartiala, J., Allayee, H., Schmidt, L. C., Tassone, F., & Hertz-Picciotto, I. (2012). Maternal periconceptional folic acid intake and risk of autism spectrum disorders and developmental delay in the CHARGE (CHildhood Autism Risks from Genetics and Environment) case-control study. *The American Journal of Clinical Nutrition*, 96(1), 80–89.
- Schmidt, R. J., Tancredi, D. J., Krakowiak, P., Hansen, R. L., & Ozonoff, S. (2014). Maternal intake of supplemental iron and risk of autism spectrum disorder. *American Journal of Epidemiology*, 180(9), 890–900.
- Schmidt, R. J., Kogan, V., Shelton, J. F., Delwiche, L., Hansen, R. L., Ozonoff, S., Ma, C. C., McCanlies, E. C., Bennett, D. H., Hertz-Picciotto, I., Tancredi, D. J., & Volk, H. E. (2017). Combined prenatal pesticide exposure and folic acid intake in relation to autism spectrum disorder. *Environmental Health Perspectives*, 125(9), 097007.
- Shelton, J., Tancredi, D., & Hertz-Picciotto, I. (2010). Independent and dependent contributions of advanced maternal and paternal ages for autism risk. *Autism Res* 3 (1), 30–39.
- Shelton, J. F., Geraghty, E. M., Tancredi, D. J., Delwiche, L. D., Schmidt, R. J., Ritz, B., Hansen, R. L., & Hertz-Picciotto, I. (2014). Neurodevelopmental disorders and prenatal residential proximity to agricultural pesticides: The CHARGE study. *Environmental Health Perspectives*, 122(10), 1103–1109.
- Singer, A. B., Aylsworth, A. S., Cordero, C., Croen, L. A., DiGuiseppi, C., Fallin, M. D., Herring, A. H., Hooper, S. R., Pretzel, R. E., & Schieve, L. A. (2017). Prenatal alcohol exposure in relation to autism spectrum disorder: Findings from the Study to Explore Early Development (SEED). *Paediatric and Perinatal Epidemiology*, 31(6), 573–582.
- Steenweg-de Graaff, J., Ghassabian, A., Jaddoe, V. W., Tiemeier, H., & Roza, S. J. (2014). Folate concentrations during pregnancy and autistic traits in the offspring. The Generation R Study. *The European Journal of Public Health*, 25(3), 431–433.
- Steenweg-de Graaff, J., Tiemeier, H., Ghassabian, A., Rijlaarsdam, J., Jaddoe, V. W., Verhulst, F. C., & Roza, S. J. (2016). Maternal fatty acid status during pregnancy and child autistic traits: The Generation R Study. *American Journal of Epidemiology*, 183(9), 792–799.
- Steffenburg, S., Gillberg, C., Hellgren, L., Andersson, L., Gillberg, I. C., Jakobsson, G., & Bohman, M. (1989). A twin study of autism in Denmark, Finland, Iceland, Norway and Sweden. *Journal of Child Psychology and Psychiatry*, 30(3), 405–416.
- Suren, P., Susser, E., & Stoltenberg, C. (2013). Maternal folic acid supplementation and risk of autism – Reply. *JAMA*, 309(21), 2208.
- Talbott, E. O., Marshall, L. P., Rager, J. R., Arena, V. C., Sharma, R. K., & Stacy, S. L. (2015). Air toxics and the risk of autism spectrum disorder: The results of a population based case-control study in southwestern Pennsylvania. *Environmental Health*, 14(1), 80.
- Traglia, M., Croen, L. A., Lyall, K., Windham, G. C., Kharrazi, M., DeLorenze, G. N., Torres, A. R., & Weiss, L. A. (2017). Independent maternal and fetal genetic effects on midgestational circulating levels of environmental pollutants. *G3 (Bethesda)*, 7(4), 1287–1299.
- van Wijngaarden, E., Davidson, P. W., Smith, T. H., Evans, K., Yost, K., Love, T., Thurston, S. W., Watson, G. E., Zareba, G., & Burns, C. M. (2013). Autism spectrum disorder phenotypes and prenatal exposure to methylmercury. *Epidemiology (Cambridge, Mass.)*, 24(5), 651.
- Vinkhuyzen, A. A. E., & Eyles, D. W. et al. (2017). Gestational vitamin D deficiency and autism spectrum disorder." *BJPsych Open*, 3(2), 85–90.

- Vinkhuyzen, A., Eyles, D., Burne, T., Blanken, L., Kruithof, C., Verhulst, F., Jaddoe, V., Tiemeier, H., & McGrath, J. (2018). Gestational vitamin D deficiency and autism-related traits: The Generation R Study. *Molecular Psychiatry*, 23, 240.
- Volk, H. E., Kerin, T., Lurmann, F., Hertz-Picciotto, I., McConnell, R., & Campbell, D. B. (2014). Autism spectrum disorder: Interaction of air pollution with the MET receptor tyrosine kinase gene. *Epidemiology*, 25(1), 44–47.
- von Ehrenstein, O. S., Aralis, H., Cockburn, M., & Ritz, B. (2014). In utero exposure to toxic air pollutants and risk of childhood autism. *Epidemiology*, 25(6), 851–858.
- Walker, C. K., Krakowiak, P., Baker, A., Hansen, R. L., Ozonoff, S., & Hertz-Picciotto, I. (2015). Preeclampsia, placental insufficiency, and autism spectrum disorder or developmental delay. *JAMA Pediatrics*, 169(2), 154–162.
- Webb, S. J., Garrison, M. M., Bernier, R., McClintic, A. M., King, B. H., & Mourad, P. D. (2017). Severity of ASD symptoms and their correlation with the presence of copy number variations and exposure to first trimester ultrasound. *Autism Res. Mar*, 10(3), 472–484.
- Weisskopf, M. G., Kioumourtoglou, M. A., & Roberts, A. L. (2015). Air pollution and autism spectrum disorders: Causal or confounded? *Current Environmental Health Reports*, 2(4), 430–439.
- Windham, G. C., Zhang, L., Gunier, R., Croen, L. A., & Grether, J. K. (2006). Autism spectrum disorders in relation to distribution of hazardous air pollutants in the San Francisco bay area. *Environmental Health Perspectives*, 114(9), 1438–1444.
- Wofford, P., Segawa, R., Schreider, J., Federighi, V., Neal, R., & Brattesani, M. (2014). Community air monitoring for pesticides. Part 3: Using health-based screening levels to evaluate results collected for a year. *Environmental Monitoring and Assessment*, 186(3), 1355–1370.
- Xiang, A. H., Wang, X., Martinez, M. P., Walthall, J. C., Curry, E. S., Page, K., Buchanan, T. A., Coleman, K. J., & Getahun, D. (2015). Association of maternal diabetes with autism in offspring. *JAMA*, 313(14), 1425–1434.
- Zaretsky, M. V., Alexander, J. M., Byrd, W., & Bawdon, R. E. (2004). Transfer of inflammatory cytokines across the placenta. *Obstetrics and Gynecology*, 103(3), 546–550.
- Zerbo, O., Iosif, A. M., Delwiche, L., Walker, C., & Hertz-Picciotto, I. (2011). Month of conception and risk of autism. *Epidemiology*, 22(4), 469–475.
- Zerbo, O., Iosif, A. M., Walker, C., Ozonoff, S., Hansen, R. L., & Hertz-Picciotto, I. (2013). Is maternal influenza or fever during pregnancy associated with autism or developmental delays? Results from the CHARGE (CHildhood Autism Risks from Genetics and Environment) study. *Journal of Autism and Developmental Disorders*, 43(1), 25–33.
- Zerbo, O., Qian, Y., Yoshida, C., Grether, J. K., Van de Water, J., & Croen, L. A. (2015). Maternal infection during pregnancy and autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 45(12), 4015–4025.
- Zerbo, O., Qian, Y., Yoshida, C., Fireman, B. H., Klein, N. P., & Croen, L. A. (2017). Association between influenza infection and vaccination during pregnancy and risk of autism spectrum disorder. *JAMA Pediatrics*, 171(1), e163609.

EPF Model

► Enhanced Perceptual Functioning

Epicanthal Fold

► Epicanthic Fold

Epicanthic Fold

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Synonyms

[Epicanthal fold](#); [Epicanthus](#); [Palpebronasal fold](#); [Plica palpebronasalis](#)

Definition

A skin fold of the upper eyelid that covers the inner corner (canthus) of the eye. It is seen normally in infants before the bridge of the nose develops and in East Asian people. It can also be a characteristic feature of some medical conditions, such as Down syndrome, fetal alcohol syndrome, Turner syndrome, Williams syndrome, and Noonan syndrome.

See Also

- [Down Syndrome](#)
- [Fetal Alcohol Syndrome](#)
- [Noonan and Ras/Mapk Pathway Syndromes](#)
- [Turner Syndrome](#)

References and Reading

- Burn, J. (1986). Williams syndrome. *Journal of Medical Genetics*, 23, 389–395.
- Delabar, J. M., Theophile, D., Rahmani, Z., Chettouh, Z., Blouin, J. L., Prieur, M., et al. (1993). Molecular mapping of twenty-four features of Down syndrome on chromosome 21. *European Journal of Human Genetics*, 1, 114–124.
- Kaneshiro, N. K. (2011). Epicanthal folds. *MedlinePlus*. Retrieved from <http://www.nlm.nih.gov/medlineplus/ency/article/003030.htm>
- Randolph, J. C., Sokol, J. A., Lee, H. B., & Nunery, W. R. (2011). Orbital manifestations of Noonan syndrome. *Ophthalmic Plastic and Reconstructive Surgery*, 27(6), e160–e163.
- Riley, E. P., Infante, M. A., & Warren, K. R. (2011). Fetal alcohol spectrum disorders: An overview. *Neuropsychology Review*, 21(2), 73–80.

Epicanthus

► Epicanthic Fold

Epidemiology

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Definition

Epidemiology is a multidisciplinary field of study that focuses on describing, investigating, and preventing disease in populations. Studying diseases on the population level creates a level of complexity not seen on the individual level. In order to understand the movement and patterns of disease in a population, epidemiologists often need to be well versed in biology (disease transmission, symptoms, treatment), social sciences (population dynamics, behavior), statistics (analysis of population level data, visualizing trends and patterns), and rigorous scientific methods (study design, causal pathways). Epidemiology employs a scientific, evidence-based

approach to investigations of health and disease and thus is sometimes referred to as the scientific arm of public health.

Historical Background

The term “epidemiology” is of Greek origin and means “the study of what is upon the people.” One of the first epidemiologists was thought to be the Greek physician Hippocrates. Besides coining the terms “epidemic” and “endemic” in approximately 400 B.C., he also noted the association between yellow fever and malaria and swamps and advocated their drainage.

Several other notable epidemiologists included John Graunt (1620–1674), who was the first to use life tables to support and refute theories on certain diseases; James Lind (1716–1794), who used an experimental study design to determine that citrus could cure scurvy in sailors; and Ignaz Semmelweis (1818–1865), who cut maternal mortality rates by more than ninefold over a 6-year time period by requiring that physicians in his ward wash their hands with chlorinated lime between patient examinations (Merrill and Timmreck 2006).

Perhaps the most notable epidemiologist, though, is John Snow (1813–1858), whose breakthrough work on cholera in London helped develop modern epidemiology as it is known today. Snow gathered data on the incubation period of cholera and time from infection to death and even plotted mortality events on maps. Not only did he study the disease extensively, he also identified a potential cause of illness – contaminated water. He found that a brewery with its own water source was protected from cholera, while approximately 500 people died over a 10-day span within close proximity to the Broad Street water pump. This led to the famous removal of the Broad Street pump handle. Years later, John Snow conducted one of the first major epidemiologic studies in a randomized setting. Two companies were competing to supply water to individuals in London. The first company, Lambeth Water Company, took water from the Thames River from a relatively unpolluted upstream

source. The second company, Southwark and Vauxhall Water Company, drew water contaminated with the city's effluent. Both companies ran water pipes to the same areas, and thus citizens randomly chose one company or the other to obtain water (some citizens did not even recall which company they had chosen). Snow was able to obtain the addresses of those who died of cholera and then determine what water company supplied water to those households. In this way, he was able to demonstrate that water from the Lambeth Water Company was far safer than the fecally contaminated water provided by the Southwark and Vauxhall Water Company. This seminal work, conducted in the mid-1800s, was produced at a time when most physicians still believed in the same miasma, or bad air, that Hippocrates had believed in over 2,000 years prior (Snow 1855).

Since Snow's time, modern epidemiology has blossomed. In 1905, a school of tropical medicine was created at the University of London, and in 1916, Johns Hopkins University created the first school of public health. The Centers for Disease Control was created in 1946, and the National Institutes of Health was created soon after in 1948 Breslow and Cengage (2006). Several large epidemiologic studies were subsequently conducted that helped solidify the reputation of epidemiology as a rigorous science. One of the most definitive set of studies conducted in the later half of the twentieth century were those conducted by Doll and Hill (1950) and Hammond and Horn (1954) which established smoking as a strong risk factor for lung cancer.

The epidemiologic study of autism spectrum disorders (ASDs) began in 1943 with Kanner's first description of autism in 11 children. Since then, many hypotheses have been developed regarding the potential causes of autism. Early hypotheses suggested that cold detached parents were a cause of autism. In 1964, an epidemic of rubella infected both mothers and their unborn or newly born infants, causing 20,000–30,000 congenital malformations. In a study conducted by Stella Chess in 1971, out of 243 preschool children who had experienced congenital infection with rubella, ten were found to have autism

(as described by Kanner) and eight demonstrated some autistic behavior. This finding, along with others, began to suggest that autism could be a disease rooted in biology and not in parenting (Amaral et al. 2011).

Current Knowledge

The current practice of epidemiology employs a variety of study designs to accurately determine the relationship between exposures and health outcomes. The tools of epidemiology have been used to address a wide variety of research questions, ranging from genetic epidemiology to outbreak epidemiology, the epidemiology of aging, and the epidemiology of childhood developmental disorders such as autism.

Study designs can be categorized into one of two headings: experimental or observational. Experimental study designs include randomized controlled trials, or clinical trials, where participants follow well-defined protocols. These types of studies are quite powerful because perfect randomization allows investigators to examine only the causal effect of exposure on the outcome of interest and prevents other factors from influencing the relationship between the exposure and disease. These factors which influence both exposure and disease are often referred to as confounders, and presence of confounders may bias the relationship between exposure and disease. However, there are several limitations to the randomized controlled design. Studies like these seek to mimic laboratory type conditions, and more control requires more time and more resources. Thus, randomized designs are often quite expensive and difficult to conduct. In addition, many ethical issues can arise. For example, individuals with illnesses that might benefit from treatment cannot be expected to take placebos.

Observational studies include many designs; the most common being cohort studies, case-control studies, and cross-sectional studies. The goal of such observational studies is to determine the causal relationships between exposures and disease, though accomplishing this task with observational study designs is much more difficult

than with a randomized controlled trial due to potential confounding.

Case-control studies involve finding individuals who are diseased, cases, and then identifying a group of nondiseased individuals, controls, who come from the same population that generated the cases. The past exposure status for all study participants is then determined, and a relationship between exposure and disease can then be calculated. Case-control studies have the advantage of being much faster and cheaper to conduct than randomized trials or cohort studies. Case-control studies are especially effective when the disease of interest is rare. However, identifying an appropriate control population may be difficult. Another limitation is that unless all confounders are identified and accounted for, the relationship between exposure and disease cannot be described as causal. Thus, many case-control studies and observational studies in general report associations between exposure and disease, not causal relationships.

Cohort studies differ from case-control studies in that the opposite approach is taken. Rather than identify diseased and nondiseased individuals to begin with, cohort studies begin by identifying at-risk exposed and unexposed individuals. These nondiseased groups are then followed until the end of the study. Illness in the exposed and nonexposed groups is then compared. Cohort studies are often more expensive than case-control studies, but are much less expensive than randomized trials. Like case-control studies, they suffer from the limitation of potential unmeasured confounders. However, one critical advantage of cohort studies is that exposure is known to precede disease, a critical criteria for a causal relationship. In addition, investigators do not have to rely on study participants to remember prior exposure status, as in a case-control study.

Cross-sectional studies are similar to taking a picture of the population at one point in time. Information on exposure and disease is often collected at the same time, and temporal ordering may or may not be recorded. They differ from case-control studies in that cross-sectional studies often attempt to collect data on the entire population of interest. They are often even less expensive

to conduct than case-control studies. An example of a cross-sectional design is mailing a questionnaire to all employees of a company, inquiring about current exposure and health status. Another example involves use of census data to determine the relationship between an exposure and disease. However, one major limitation to cross-sectional studies that use “high-level” data such as census data is the ecological fallacy. The ecological fallacy exists when a relationship is seen at a population level that does not exist at the level of individual persons. An example of this is measuring the average IQ score for a population. While the average is appropriate to use at the population level, the IQ of any individual taken from that population could be higher or lower than the average. It would be a fallacy to assume that each individual had exactly the average IQ measured for the entire population.

All of the above study designs are subject to a variety of biases that can distort the relationship between the exposure and disease. One such bias is random error, which can arise when data tend to be randomly biased in one direction, and thus bias final results. A solution to this type of bias is to either measure variables more precisely in order to minimize random noise or to increase the number of participants or samples to the point where small random fluctuations are unlikely to alter the final results significantly.

Another type of bias is selection bias, which occurs when exposure or disease status influence participation in a study. For example, if researchers want to recruit children with ASD into a study, and advertise on TV and the Internet for participants, they may likely get a biased sample. More educated, financially well-off individuals may be more likely to see those advertisements and participate. Results from that study may differ greatly than a study conducted using a random sample from a comprehensive registry of children diagnosed with ASD.

Confounding is another bias and may arise when there are systematic differences between exposed and unexposed groups. These differences may lead investigators to falsely conclude that exposure alone caused differences in health outcomes. Confounders are typically factors that

cause exposure as well as disease. A simple example of this would be a study that investigated whether or not use of a mood-stabilizing medication among men with Asperger's syndrome could cause autism in their child. A naïve analysis examining the relationship between medication and the child's ASD status could find a strong association, but it is likely that much if not all of the associations could be explained by the genetic history of the father. Attributing all cases of ASD to the medication would be a biased conclusion, one that is often referred to as "confounded."

Another important source of bias is information bias. Information bias generally refers to the circumstance when data collected on an individual is not completely accurate. If a study participant reported having a relative with autism when that relative actually was diagnosed with obsessive compulsive disorder, that would be an example of information bias. Information bias is often broken into two categories: differential and nondifferential. Nondifferential bias refers to inaccurate measurement of either exposure or disease status, but the inaccuracy is the same in both the control and comparison groups. For instance, in a case-control study examining ASD as an outcome and maternal flu vaccination as an exposure, if mothers of children with ASD incorrectly reported their vaccination history in a similar fashion to mothers of children without ASD, then that "recall bias" would be referred to as nondifferential. However, if mothers of children without ASD underreported their flu vaccination history while mothers of children with ASD overreported their flu vaccination history, then that inaccurate measurement of exposure would be called differential (different recall in the case and comparison groups). Nondifferential misclassification is less concerning than differential misclassification. While both forms of bias typically give answers that differ from the truth, nondifferential misclassification gives answers that are biased consistently toward the null (no association), while differential misclassification can bias results either toward or away from the null. This unpredictability is undesirable and makes it difficult to interpret the results with any confidence.

Future Directions

Historically, epidemiology has been used to discover associations between exposures and disease. In fact, according to US law, epidemiology cannot be used by itself to prove causative relationships between exposure and disease in a particular individual; rather, it can only make a probabilistic statement about whether an exposure *could* have caused an outcome in an individual (National Research Council 2011). However, the science of epidemiology has always attempted to explore and identify causal relationships. With the advent of computers, developments in genetics, and availability of sophisticated measurement tools, epidemiologists have been able to better explore causal relationships between various exposures and diseases. Modern technology, including sophisticated statistical tools and electronic recording of data, has greatly improved the feasibility of conducting large, rigorous studies of health and analyzing large quantities of data. As the field of epidemiology continues to develop, there is great hope that previously untreatable diseases and health conditions will be better understood and that each successive generation will enjoy better health than those that came before.

See Also

- Incidence
- Prevalence

References and Reading

- Amaral, D., Geschwind, D., & Dawson, G. (2011). *Autism spectrum disorders* (1st ed.). Oxford: Oxford University Press.
- Breslow, L., & Cengage, G. (Eds.). (2006). *Encyclopedia of public health*. eNotes.com. Retrieved from <http://www.enotes.com/public-health-encyclopedia/history-public-health>
- Chess, S. (1971). Autism in children with congenital rubella. *Journal of Autism and Childhood Schizophrenia*, 1(1), 33–47. <https://doi.org/10.1007/BF01537741>.
- Doll, R., & Hill, A. B. (1950). Smoking and carcinoma of the lung: Preliminary report. *British Medical Journal*, 2(4682), 739–748.

- Doll, R., & Hill, A. B. (1956). Lung cancer and other causes of death in relation to smoking: A second report on the mortality of British doctors. *British Medical Journal*, 2(5001), 1071–1081.
- Friedman, L., Furberg, C., & DeMets, D. (2010). *Fundamentals of clinical trials* (4th ed.). New York: Springer.
- Hammond, E. C., & Horn, D. (1954). The relationship between human smoking habits and death rates. *Journal of the American Medical Association*, 155(15), 1316–1328. <https://doi.org/10.1001/jama.1954.03690330020006>.
- Jadad, A. R., & Enkin, M. W. (2007). *Randomised controlled trials: Questions, answers and musings* (2nd ed.). London: BMJ Books.
- Koepsell, T. D., & Weiss, N. S. (2003). *Epidemiologic methods: Studying the occurrence of illness* (1st ed.). Oxford: Oxford University Press.
- Merrill, R. M., & Timmreck, T. C. (2006). *Introduction to epidemiology*. Sudbury: Jones & Bartlett Learning.
- National Research Council. (2011). *Reference manual on scientific evidence* (3rd ed.). Washington, DC: National Academies Press.
- Rothman, K. J., Greenland, S., & Lash, T. L. (2008). *Modern epidemiology* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
- Snow, J. (1855). *On the mode of communication of cholera*. London: John Churchill.
- Szklo, M., & Nieto, J. (2006). *Epidemiology: Beyond the basics* (2nd ed.). Sudbury: Jones and Bartlett.

biologist, geneticist, and philosopher who established foundations for systems biology. In 1942, predating knowledge of the physical nature of genes and their role in heredity, he used the term to refer to the study of the “causal mechanisms” by which “the genes of the genotype bring about phenotypic effects” (Waddington 1942). Early on, “epigenetics” was a vaguely understood idea that was often invoked to explain phenomena when basic genetic principles fell short. Despite an absence of direct scientific evidence, Holliday and Pugh (1975) and Riggs (1975) independently proposed the idea that *methylation* was a heritable epigenetic process that played an important role in regulating gene expression. Subsequently, a large amount of direct evidence accumulated which validated these ideas, and epigenetics began to evolve into a field of science whose molecular underpinnings were clarified. Holliday’s review of “the inheritance of epigenetic defects” (Holliday 1987) initiated widespread use of the term “epigenetic” during the 1990s. Today, molecular biologists define epigenetics as “the study of mitotically and/or meiotically heritable changes in gene function that cannot be explained by changes in DNA sequence” (Riggs 1996).

Epigenetic Mechanisms

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Definition

Means by which gene expression is regulated without altering the underlying DNA sequence. Epigenetic mechanisms result in heritable annotations or modifications to DNA and associated histones which include the following: methylation, acetylation, ubiquitylation, phosphorylation, and sumoylation.

Historical Background

Coining of the term “epigenetics” is often attributed to Conrad H. Waddington, a developmental

Current Knowledge

Overview

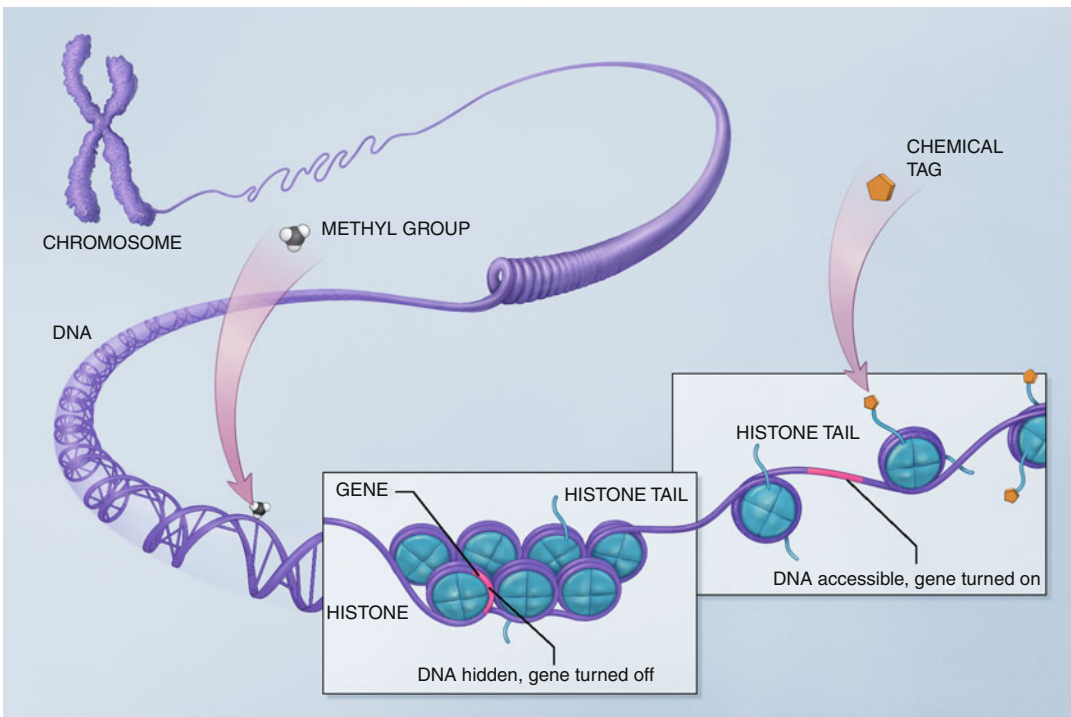
Two predominant mechanisms provide the molecular basis for epigenetic regulation of gene expression: DNA methylation and histone modifications (which include methylation, acetylation, ubiquitylation, phosphorylation, and sumoylation of histone tails). These alterations are thought to alter the shape of chromatin, the complex of DNA wrapped around an octamer structure of histone proteins in the cell nucleus, and thereby affect gene transcription. Epigenetic mechanisms are active in both the germ line and somatic tissues; they are influenced by both genetic and environmental factors. The resulting epigenetic alterations can be conceptualized as comprising an “epigenotype” that is heritable between mother and daughter cells (meiotic inheritance) and between generations (mitotic inheritance).

Epigenetic modifications yielding differing epigenotypes are one explanation for how cells or organisms with otherwise identical DNA sequence may display different phenotypes. With regard to autism spectrum disorders (ASD), epigenetic modifications in several genes and genomic regions have been reported in both syndromic and nonsyndromic forms of ASD and may represent an important pathway in pathogenesis. Understanding the role of epigenetic changes in ASD is of particular interest, as their reversal may represent a potential target of novel therapeutic strategies.

DNA Methylation

Methylation is a well-characterized epigenetic modification that involves the covalent addition of a methyl group to cytosine bases at the carbon-5 position of a CG dinucleotide sequence of DNA (CpG dinucleotide) (Fig. 1). Regions of

the genome rich in CG dinucleotide sequence are known as CpG islands and exist in the promoter regions of approximately half of all known genes. Methylation occurs on both strands of a CpG dinucleotide via DNA methyltransferase (DNMT) enzymes and the methyl donor S-adenosyl-l-methionine (SAM). There is no known enzyme with DNA demethylating activity. Highly methylated regions are associated with a closed chromatin conformation state (heterochromatin), displaying less transcriptional activity and therefore less gene protein expression. DNA methylation may inhibit gene expression by direct interaction with factors that repress transcription, or indirectly through recruitment of methyl-CpG-binding proteins complexed with enzymes that modify histone proteins, transforming chromatin from an active to a repressed state. Alternatively, lack of methylation is associated with an open chromatin conformation state



Epigenetic Mechanisms, Fig. 1 *Epigenomic marks.* The epigenome can mark DNA in two ways, both of which play a role in turning genes off or on. The first occurs when certain chemical tags called methyl groups attach to the backbone of a DNA molecule. The second occurs when

a variety of chemical tags attach to the tails of histones, which are spool-like proteins that package DNA neatly into chromosomes. This action affects how tightly DNA is wound around the histones. (Courtesy of National Human Genome Research Institute)

(euchromatin), leading to more transcriptional activity and gene expression. Tumor growth has been associated with a state of hypomethylation, increasing the rate of mitotic recombinations from baseline and leading to genomic instability (e.g., structural rearrangements).

Histone Modifications

Another type of epigenetic mechanism which has the potential to modify gene expression involves chromatin remodeling via reversible covalent posttranslational modifications to histone proteins (Fig. 1). Two copies of four histone proteins (H2A, H2B, H3, and H4) form an octamer structure around which DNA is wrapped. The flexible N-terminus tails of histone proteins, H3 and H4, are most likely to carry numerous modifications of amino acids, including acetylation, methylation, phosphorylation, ubiquitylation, and sumoylation. Histone acetylation (via histone acetyltransferase enzymes) and phosphorylation is associated with an open or relaxed chromatin conformation state (euchromatin), facilitating transcriptional activity and gene protein expression. Conversely, histone deacetylation (via histone deacetylase enzymes) or sumoylation is associated with a closed or compact chromatin conformation state (heterochromatin), repressing transcription and gene protein expression. Other histone modifications may facilitate or repress transcription, depending on which amino acid residue is altered.

Epigenetic Syndromes and ASD

With respect to ASD, there are several examples of known epigenetic syndromes associated with increased risk. With the important caveat that intellectual disability in these syndromes may account for some of the observed phenotypic overlap in ASD, it is also possible that shared genes and epigenetic mechanisms may confer risk for ASD as well.

Rett Syndrome and Methyl-CpG-Binding Proteins

Many features of Rett syndrome (language impairments, stereotypic behaviors, seizures, sleep abnormalities, age of onset) are shared in common with autism, and both disorders are

classified as pervasive developmental disorders in DSM-IV-TR. Approximately 80% of Rett syndrome patients (almost exclusively females) and a small number of patients with autism (without Rett syndrome) have been reported with rare and common mutations in the *MECP2* gene on the X chromosome. This transcript codes for a methyl-CpG-binding protein with functional domains for methyl binding and transcription repression via inhibition of transcription factor binding; it may also facilitate histone modifications. Studies of *MECP2* in autism have shown decreased brain expression relative to matched controls, and expression levels have been correlated with rare variants and hypermethylation in the gene's promoter (Nagarajan et al. 2006).

Fragile X Syndrome

Fragile X syndrome (FXS) is linked to the expansion of a CGG repeat sequence in the 5' untranslated region of the *FMR1* gene on chromosome Xq27. These repeat expansions lead to increased methylation, decreased acetylation, and therefore decreased expression of *FMR1*, a gene whose protein product is involved with RNA processing. Such epigenetic modifications appear to be crucial to the development of FXS, as a normal phenotype in the presence of the CGG repeat allele is observed if hypermethylation is absent. ASD risk for full-mutation (>200 CGG repeats) males and females range from 60% to 67% and 10% to 23%, respectively, based on studies using the Autism Diagnostic Interview (ADI) and Autism Diagnostic Observation Schedule (ADOS) (Clifford et al. 2007). Shared ASD features include repetitive behaviors, language impairments, and eye-gaze aversion.

Prader-Willi and Angelman Syndromes

Both Prader-Willi syndrome (PWS) and Angelman syndrome (AS) have an increased risk for ASD (19–37% for PWS and 42–100% for AS) (Hogart et al. 2010). These syndromes result from a specialized epigenetic mechanism called imprinting, a phenomenon by which each parent contributes different “epigenotypes” via their germ cells for specific genomic loci. DNA methylation or histone acetylation marks one

allele of an imprinted gene and prevents its transcription/expression. This yields monoallelic expression of an imprinted gene. PWS and AS are caused by epigenetic errors or chromosomal rearrangements at an imprinting cluster on chromosome 15q11-13 (e.g., AS results from loss of *UBE3A* expression, a gene that is normally expressed exclusively from the maternal chromosome) (Nicholls and Knepper 2001). Increased risk for ASD in PWS and AS and an increased risk for ASD with maternal duplications of 15q11-13 (with and without PWS and AS) suggest that a loss of imprinting may increase susceptibility to ASD (Veltman et al. 2005).

Epigenetic Modifications in ASD Candidate Genes

In addition to one report of ASD association with increased *MECP2* methylation, as mentioned above, there is some evidence that other (nonimprinted) ASD candidate genes may be epigenetically regulated. One study found increased DNA methylation in the promoter of the oxytocin receptor gene (*OXTR*) in blood and temporal cortex (Gregory et al. 2009). Similarly, the long allele of the reelin gene (*RELN*) has been associated with ASD and appears to cause decreased gene expression via epigenetic mechanisms (Persico et al. 2006). Serotonin transporter genes and *BDNF* (*brain-derived neurotrophic factor*) are included among other candidate genes which are being investigated for evidence of epigenetic regulation (Rumajogee et al. 2002). While these genes have not been definitively demonstrated to play a role in ASD, they are one line of evidence that epigenetic mechanisms could contribute ASD pathogenesis.

Environmental Influences on Epigenetic Modifications

There is some evidence that certain environmental exposures may cause epigenetic changes which can lead to increased risk for ASD. One example is prenatal exposure to valproate. The risk of ASD to children of mothers taking this drug during pregnancy has been estimated up to

15 times the general population risk (Rasalam et al. 2005). Two possible mechanisms of valproate teratogenicity include interference with folate metabolism, which can modify gene expression in certain metabolic pathways, and inhibition of histone deacetylases, which can result in increased expression of multiple genes (Phiel et al. 2001). Furthermore, decreased expression of an autism candidate gene, *NLGN3*, has been reported in mice treated with valproate (Kolozsi et al. 2009).

Future Directions

Epigenetic mechanisms that regulate gene expression have emerged as a fundamental mechanism in developmental biology and the pathogenesis of disease. New technologies allowing high-throughput and whole-genome screening for DNA methylation and chromatin modifications hold great promise for the identification of epigenetic determinants in ASD. Given that epigenetic modifications to DNA are potentially influenced by environmental factors, elucidating the epigenetics of ASD could lead to the development of a new era of therapeutics.

See Also

- [Angelman/Prader-Willi Syndromes](#)
- [Course of Social Avoidance in Fragile X Syndrome, The](#)
- [Deoxyribonucleic Acid](#)

References and Reading

- Clifford, S., Dissanayake, C., Bui, Q. M., Huggins, R., Taylor, A. K., & Loesch, D. Z. (2007). Autism spectrum phenotype in males and females with fragile X full mutation and premutation. *Journal of Autism and Developmental Disorders*, 37(4), 738–747.
- Grafodatskaya, D., Chung, B., Szatmari, P., & Weksberg, R. (2010). Autism spectrum disorders and epigenetics. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(8), 794–809.
- Gregory, S. G., Connelly, J. J., Towers, A. J., Johnson, J., Biscocho, D., Markunas, C. A., et al. (2009). Genomic

- and epigenetic evidence for oxytocin receptor deficiency in autism. *BMC Medicine*, 7, 62.
- Hamilton, J. P. (2011). Epigenetics: Principles and practice. *Digestive Diseases*, 29(2), 130–135.
- Hogart, A., Wu, D., LaSalle, J. M., & Schanen, N. C. (2010). The comorbidity of autism with the genomic disorders of chromosome 15q11.2-q13. *Neurobiology of Disease*, 38(2), 181–191.
- Holliday, R. (1987). The inheritance of epigenetic defects. *Science*, 238(4824), 163–170.
- Holliday, R., & Pugh, J. E. (1975). DNA modification mechanisms and gene activity during development. *Science*, 187(4173), 226–232.
- Kolozsi, E., Mackenzie, R. N., Rouillet, F. I., deCatanzaro, D., & Foster, J. A. (2009). Prenatal exposure to valproic acid leads to reduced expression of synaptic adhesion molecule neuroligin 3 in mice. *Neuroscience*, 163(4), 1201–1210.
- Nagarajan, R. P., Hogart, A. R., Gwyne, Y., Martin, M. R., & LaSalle, J. M. (2006). Reduced MeCP2 expression is frequent in autism frontal cortex and correlates with aberrant MECP2 promoter methylation. *Epigenetics*, 1(4), e1–e11.
- Nicholls, R. D., & Knepper, J. L. (2001). Genome organization, function, and imprinting in Prader-Willi and Angelman syndromes. *Annual Review of Genomics and Human Genetics*, 2, 153–175.
- Persico, A. M., Levitt, P., & Pimenta, A. F. (2006). Polymorphic GGC repeat differentially regulates human reelin gene expression levels. *Journal of Neural Transmission*, 113(10), 1373–1382.
- Phiel, C. J., Zhang, F., Huang, E. Y., Guenther, M. G., Lazar, M. A., & Klein, P. S. (2001). Histone deacetylase is a direct target of valproic acid, a potent anticonvulsant, mood stabilizer, and teratogen. *The Journal of Biological Chemistry*, 276(39), 36734–36741.
- Rasalam, A. D., Hailey, H., Williams, J. H., Moore, S. J., Turnpenny, P. D., Lloyd, D. J., et al. (2005). Characteristics of fetal anticonvulsant syndrome associated autistic disorder. *Developmental Medicine and Child Neurology*, 47(8), 551–555.
- Riggs, A. D. (1975). X inactivation, differentiation, and DNA methylation. *Cytogenetics and Cell Genetics*, 14(1), 9–25.
- Riggs, A. D. (1996). Introduction. In V. E. A. Russo (Ed.), *Epigenetic mechanisms of gene regulation* (pp. 1–4). Cold Spring Harbor: Cold Spring Harbor Laboratory Press.
- Rumajogee, P., Madeira, A., Vergé, D., Hamon, M., & Miquel, M. C. (2002). Up-regulation of the neuronal serotonergic phenotype in vitro: BDNF and cAMP share Trk B-dependent mechanisms. *Journal of Neurochemistry*, 83(6), 1525–1528.
- Veltman, M. W., Craig, E. E., & Bolton, P. F. (2005). Autism spectrum disorders in Prader-Willi and Angelman syndromes: A systematic review. *Psychiatric Genetics*, 15(4), 243–254.
- Waddington, C. H. (1942). The epigenotype. *Endeavour*, 1, 18–20.

Epilepsy

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Synonyms

Seizure disorder

Short Description or Definition

Epilepsy is operationally defined as more than one unprovoked seizure of any type. Recent definitions of epilepsy have emphasized the neurologic, cognitive, psychological, and social consequences of this group of disorders (Fisher et al. 2005). Epilepsy, like autism, is not one disorder and is best conceptualized as the epilepsies.

Seizures are clinical events characterized by paroxysmal, stereotyped, relatively brief interruptions of ongoing behavior, associated with electrographic seizure patterns (ILAE 1981). The term “subclinical or nonconvulsive seizure” is used to refer to electrographic patterns without clinically recognizable cognitive, behavioral, or motor functions or apparent impairment of consciousness and requires concurrent electroencephalogram (EEG) during behavioral testing. Seizures are differentiated into those that are provoked secondary to an acute event such as fever, infection, trauma, or metabolic illness and those that are unprovoked (i.e., seizures that are likely to be genetic). There are *two peaksof seizureonset* in autism. The early peak of seizure onset in autism occurs in the first 5 years of life and is commonly associated with epileptic encephalopathies (see below). The second peak of seizure onset starts in adolescence and continues into adulthood. This secondary peak of seizure onset may be more

common in individuals with ASD than early onset seizures and as such may represent a different distribution of seizure onset than in the general population (Bolton et al. 2011).

Interictal epileptiform discharges and epileptiform activity are terms used interchangeably to describe abnormal electroencephalogram (EEG) activity, specifically referring to spikes alone or accompanied by a slow wave, occurring either singly or in bursts and lasting at most 1 or 2 s. The association of epileptiform activity with an epileptic disorder is variable. The term “subclinical or nonconvulsive seizure” has also been used to refer to electrographic patterns without clinically recognizable cognitive, behavioral, or motor functions or any apparent impairment of consciousness. Studies on the prevalence of epileptiform activity in individuals with ASD and no clinical history of seizures range from 6% to 31% (Kagan-Kushnir et al. 2005). There is significant controversy regarding the specificity of these findings to the ASD phenotype, and it is likely that the high prevalence of epileptiform activity in ASD is secondary to pathophysiological processes common to autism and other neurodevelopmental disorders. The treatment of interictal epileptiform discharges in children with autism remains controversial with little evidence that suppression of spikes accounts for behavioral improvement, except in rare clinical scenarios (see below under epileptic encephalopathies) (Tuchman et al. 2010).

Epidemiology

The prevalence of epilepsy in ASD is highly variable and depends on the cohort studied, with rates ranging from 5% to 46% (Spence and Schneider 2009). The reported rates of epilepsy in ASD are several fold higher than the 0.5–1% prevalence of epilepsy in the general population but are similar to the prevalence of epilepsy in a population of children with intellectual disability, with the highest rates of epilepsy in those with severe cognitive impairments (Tuchman et al. 2010).

Natural History, Prognostic Factors, and Outcomes

A consistent finding across multiple studies of autism and epilepsy is that severity of intellectual disability is a significant risk factor for the development of epilepsy in children with ASD (Amiet et al. 2008). Individuals with ASD and epilepsy are, as a group, significantly impaired as young adults (Danielsson et al. 2005), and children with autism and epilepsy have worse cognitive (lower IQ), adaptive, behavioral, and social outcomes than children with autism without epilepsy (Hara 2007; Turk et al. 2009).

Clinical Expression and Pathophysiology

Clinical Expression

Epilepsy syndromes are differentiated based on clinical findings, etiology, and patterns of EEG abnormalities. The most common epilepsy syndromes associated with autism spectrum disorders, especially those in which the epilepsy begins in the first 3 years of life, are the *epileptic encephalopathies*.

Epileptic encephalopathy is defined as “a condition in which the epileptiform abnormalities themselves are believed to contribute to the progressive disturbance in cerebral function” (Berg et al. 2010; Engel 2001). As a group, the epileptic encephalopathies are associated with regression or slowing of cognitive, language, or behavioral development; the hypothesis is that the seizures or the interictal epileptiform activity are responsible for the deterioration (Dulac 2001; Nabbout and Dulac 2003, 2008). Among these epileptic encephalopathies, *West syndrome* or *infantile spasms (IS)*, *Landau-Kleffner syndrome (LKS)*, and *epilepsy with continuous spikewaves during slow-wave sleep (CSWS)* are most commonly associated with an ASD phenotype (Ballaban-Gil and Tuchman 2000). However, it should be noted that the ASD phenotype might not be exclusive to these three encephalopathies; the ASD phenotype may be under-recognized or under-investigated in other epileptic encephalopathies such as *Dravet syndrome* (Wolff et al. 2006).

Infantile spasms are associated with multiple etiologies and represent a distinct subgroup of epilepsies with poor cognitive and social outcomes. Infantile spasms is an age-specific epilepsy syndrome that occurs between 3 and 18 months with a peak age of presentation between 4 and 8 months of age (Zupanc 2009). The seizure consists of a sudden flexion or extension of the proximal and truncal muscles which lasts for approximately 0.2–2 s. They are more prolonged than a myoclonic jerk (<100 ms) but less sustained than a tonic seizure (few seconds to minutes). Frequently, they are associated with high-voltage abnormalities on the interictal EEG that have been called hypsarrhythmia. Coincident intellectual retardation or regression of neurodevelopment in infantile spasms is common. The risk of developing autism after having infantile spasms is approximately 46% but can be as high as 69% in those with infantile spasms and significant associated brain lesions.

Dravet syndrome is a genetically determined infantile epileptic encephalopathy mainly caused by de novo mutations in the *SCN1A* gene (Scheffer et al. 2009). Progressive decline or plateau in development occurs by 1–4 years of age with intellectual disability and an autism phenotype commonly present especially in those with greater than five seizures per month (Wolff et al. 2006). There is emerging evidence that vaccine encephalopathy, characterized by the appearance of seizures and regression in infants following vaccination, may be secondary to SCNA1 gene mutations in these infants, suggesting that vaccine encephalopathy could, in fact, be a genetically determined epileptic encephalopathy (Berkovic et al. 2006).

A controversial example of an epileptic encephalopathy that has been linked to ASD is *Landau-Kleffner syndrome (LKS)*, an acquired aphasia in association with an epileptiform EEG with spikes, sharp waves, or spike and wave discharges that are usually bilateral and occur predominantly over the temporal regions (Landau and Kleffner 1998). On a continuum with Landau-Kleffner syndrome is *continuous spike waves during slow-wave sleep (CSWS)*, an epileptic encephalopathy associated with the

EEG pattern of *electrical status epilepticus during slow-wave sleep (ESES)*, various seizure types, and cognitive, motor, and behavioral disturbances (Tassinari et al. 2009). Continuous spike waves during slow-wave sleep and Landau-Kleffner syndrome are *sleep-related epileptic encephalopathies* with common clinical features including seizures, regression, and epileptiform abnormalities that are activated by sleep (Nickels and Wirrell 2008). In continuous spike waves during slow-wave sleep, there is a regression in global skills, while in Landau-Kleffner syndrome, the primary clinical manifestation is a regression of language. *Autistic regression with an epileptiform EEG* is the terminology used to describe the association of an epileptiform EEG in children with autistic regression, e.g., those in whom both language and social skills are lost. This group of children should be differentiated from those with LKS. In this group of children, the language and social skill loss occurs earlier, before the age of 2, as compared to those with LKS in which it occurs usually after age 3 years. In addition, the children with autistic regression and an epileptiform EEG usually do not have frequent spikes on the EEG and rarely have the ESES EEG pattern common in LKS. The importance of differentiating children with autism, regression in language and social skills, and epileptiform EEG from those with LKS is that there is no evidence that the treatments used in LKS, such as the use of steroids or surgical interventions, are effective interventions for this group of children (Tuchman 2009).

Pathophysiology

The word “epileptogenesis” in pediatrics refers to dynamic processes which constitute the appearance and natural history of epilepsy. The appearance of autism in epilepsy patients and epilepsy in autism patients suggests that a final common set of neural pathways and molecular processes are shared by both autism patients and epilepsy patients. The understanding of the development of epilepsy, progression of epilepsy, and interaction between epileptic seizures and cerebral maturation will be critical to our knowledge of epileptogenesis in autism epilepsy patients.

Mechanisms responsible for epileptogenesis of partial seizures may be shared among autism and nonautism patients. Models of partial epilepsy reveal a characteristic course in the development of isolated epileptogenic lesions (Thom et al. 2010). Children under 10–11 years of age tend to have epileptiform discharges focally in the centrottemporal or centroparietal regions (Chez et al. 2006). Thus, mechanisms responsible for focal epilepsy may be applicable to younger children with autism/epilepsy. First, a localized structural or molecular change occurs in the region of brain tissue which in itself may not cause seizures. Reorganization of synaptic inputs with altered neural integration ensues, which, in many cases, change the regional balance to excessive excitation and pathologic synchronization of action potential firing. Localized interictal EEG spikes occur in this region, and later, in most cases, spontaneous seizures are seen emanating from the site.

The *kindling model of epilepsy* demonstrates the process of partial epileptogenesis. Kindling is a process by which initially subconvulsive electrical stimulus to a brain region, such as the amygdala, is repeated once a day for 21 days. An initial subconvulsive electrical stimulus may only evoke an electrographic discharge with little change in the animal's behavior (Racine et al. 1975). The repeated electrical stimulus will evoke first a behavior arrest (class I seizure) and then stepwise at day 21 evoke a secondarily generalized tonic-clonic seizure (class V seizure). An animal is considered fully kindled when the same stimulus evokes a secondarily generalized tonic-clonic seizure over three consecutive days. Electrophysiologic and structural analyses of kindled animals suggest permanent alterations to rodent limbic cortex which parallels changes seen in human temporal lobe epilepsy (Morrell and de Toledo-Morrell 1999). The genetic program activated over a period of time includes three phases: (1) expression of immediate early genes and apoptosis genes, (2) expression of secondary transcriptional regulatory proteins, and (3) expression of tertiary or quaternary target genes (neurotrophins, guidance cue genes, etc.) in response to transcriptional regulatory proteins

and electrical activity. The end result of this genetic reprogramming is synaptic reorganization and epileptogenesis. The final common pathway here seems to be recurrent excitatory networks. Kindling causes epileptogenesis by a variety of possible mechanisms including (1) reorganization of axons/synaptic connections, (2) alterations in AMPA and NMDA receptors, (3) enhanced release of glutamate by neurosecretory mechanisms, and (4) alteration of inhibition via kainic acid receptor activation (Wang et al. 2010).

Shared Molecular Pathophysiology Between Autism and Epilepsy

Genetic Components

The standard hypothesis to explain the overlap of autism and epilepsy is that an altered balance between excitation and inhibition is one biological mechanism for both diseases; thus, the disturbance in the development of either cortical projection neurons or interneuron maturation would tip over this delicate balance. Giving the fact that autism co-occurs with numerous neurodevelopmental disorders and the complexity of synaptogenesis and function, it is not surprising that there are multiple molecules and signaling pathways associated with the pathogenesis of autism. Subtle perturbations or alterations in any of these molecules critical in neurodevelopment, synaptogenesis, and synaptic function may cause defects in the downstream common biological pathways within brain circuits contributing to ASD pathogenesis (Geschwind and Levitt 2007).

The genetic architecture of autism includes at least two distinct genetic mechanisms: rare, private (de novo) single-gene mutations that may have a large effect in causing ASD and inherited, common functional variants of a combination of genes, each having a small to moderate effect in increasing ASD risk (Abrahams and Geschwind 2008; O'Roak and State 2008; Veenstra-VanderWeele and Cook 2004; Weiss et al. 2009). Rare point mutations range from ASD risk genes encoding numerous synaptic proteins (such as contactin-associated protein-like 2, CNTNAP2; SH3 and multiple ankyrin repeat domains 3, SHANK3; and neuroligin-3, NLGN3) to gains

or losses of DNA segments, termed copy number variation (e.g., 16p11.2 and 15q11-q13), and to gross chromosomal rearrangements that are estimated to occur in about 7% of autism cases (Abrahams and Geschwind 2008).

In epilepsy, genetic advances have identified ion channel genes as the major category of epilepsy susceptibility genes, although nonion channel genes have also been identified to be associated with epilepsy (Suzuki et al. 2004). These ion channels include both voltage-gated and ligand-gated ion channels. The voltage-gated ion channels include sodium channels like SCN1A, SCN2A, and SCN1B; potassium channels like KCNQ2 and KCNQ3; and calcium channels like CACNA1A (Glasscock et al. 2007) and CACNB4 (Escayg et al. 2000). The ligand-gated ion channels include GABA_A receptors (Kang and Macdonald 2009) and nicotinic acetylcholine receptors (Steinlein and Bertrand 2010). Mutations of these ion channel genes that either directly or indirectly enhance excitatory neurotransmission or reduce inhibitory neurotransmission increase brain hyperexcitability and thereby predispose patients to seizures. Rare copy number variations, already implicated in ASD (Weiss 2009), have been noted in patients with idiopathic generalized epilepsy (IGE) (Carmona-Mora and Walz 2010; Sisodiya and Mefford 2011). IGEs are a well-defined group of epilepsies, accounting for a third of all cases of epilepsy in the general population and an even higher proportion in children (1). IGEs usually begin in childhood, are genetically determined, and have no structural or anatomic cause (Benbadis 2005). However, the prevalence of this genetic mechanism in IGEs remains to be established.

There are a number of mutations or variants in GABA_A receptors that have been associated with epilepsies of various phenotypes (Kang and Macdonald 2009; Macdonald et al. 2010). These mutations/variants associated with epilepsies have been reported in $\alpha 1$, $\beta 3$, $\gamma 2$, and δ subunits. Most of the mutations have autosomal dominant inheritance and have been associated with epilepsy syndromes comprised of pure febrile

seizures (FS), mixed afebrile and febrile seizures such as CAE and FS, generalized epilepsy with febrile seizures plus (GEFS+) syndrome, Dravet syndrome, and juvenile myoclonic epilepsy (JME). These mutations include missense, nonsense, and intronic splice donor site mutations. Most of the mutations are associated with fairly mild phenotypes like simple febrile seizures and CAE which are outgrown with age. In summary, defects in multiple genes and molecules may lead to alterations in the final common neural pathways involved in the pathogenesis of epilepsy and autism. Among all these genetic and molecular pathways, impaired GABAergic signaling is a prominent pathology underlying the pathogenesis of both autism and epilepsy.

Abnormal Brain Development

Both autism and epilepsy have abnormal brain development. But there are many more studies on brain development of autism than on epilepsy. Cross-sectional magnetic resonance imaging (MRI) studies have long hypothesized that the brain in children with autism undergoes an abnormal growth trajectory that includes a period of early overgrowth. The abnormalities in autism could be broad including frontal and temporal lobes, amygdala, basal ganglia, corpus callosum, parietal lobe, and cerebellum (Allen and Courchesne 2003). In addition to the identification of brain overgrowth in autistic brains (Courchesne 2002; Courchesne et al. 2003), Courchesne and his group also demonstrated the first direct evidence with MRI study that anatomic abnormalities within the limbic system exist from the earliest years of autism and change throughout development and up through middle age. It is worth noting that cerebellar abnormality is repeatedly reported in most specimens examined, thus challenging the view that the function of cerebellum is exclusively motor coordination (Allen and Courchesne 2003). Interestingly, gene mutations in GABRA6 (Dibbens et al. 2009) were also associated with epilepsy. The transcript of $\alpha 6$ subunit was reported to be confined to the postnatal

cerebellum (Laurie et al. 1992). This may suggest that this subunit may exist in other unknown brain areas or argue against the role of cerebellum simply being motor coordination.

There are shared neuropathologies underlying ASD and epilepsy (Taylor et al. 1999; Wegiel et al. 2010). SPECT/PET scans of autism/epilepsy patients and pediatric patients with medication-resistant focal epilepsy caused by focal cortical dysplasia show similar areas of abnormalities in the frontal or temporal cortex (Sasaki et al. 2010). The discrete focal areas of hypometabolism suggest similar cellular abnormalities in the cortices of both types of patients (Sasaki et al. 2010; Taylor et al. 1999; Wegiel et al. 2010). The initial overgrowth in autism patients is associated with focal developmental abnormalities. The focal abnormalities include subependymal nodular dysplasias, subcortical and periventricular heterotopias, dysplasias including either cell loss or increased numbers of poorly differentiated neurons with disturbed lamination in neocortex, archicortex, dentate gyrus, cornu ammonis, and cerebellar cortex (Taylor et al. 1999; Wegiel et al. 2010). In autism brain, tuber-like structures as seen in tuberous sclerosis are detected in cortical and subcortical regions such as the basal ganglia (Numis et al. 2011). Older autopsy specimens tend to be associated with cell loss and disturbed cortical minicolumns. Posterior regions of the corpus callosum are also reduced in size in autism in a study of 3- to 42-year-olds (Schumann et al. 2010). MRI studies of older ASD children suggested that altered volumes of caudate nuclei, hippocampi, amygdale, and corpus callosum are associated with higher rates of epilepsy and lower sensory response rates (Bloss and Courchesne 2007; Brambilla et al. 2003; Schumann et al. 2004, 2010). In conclusion, these recent MRI and neuropathology observations suggest abnormal regulation of brain growth in autism: overgrowth early in life followed by abnormally slowed growth in some regions, but premature arrest of growth/poor differentiation followed by cell loss in others.

The role of pathologic neural activity such as interictal epileptiform discharges (IEDs) and

seizures in the developing brain of children with ASD is less clear. Evidence from human epilepsy and ASD patients indicates that there is abnormal and equivalent histology in human mesial temporal sclerosis and ASD hippocampi (Blumcke et al. 2009). These changes include substantial granule cell loss and architectural abnormalities like granule cell dispersion, ectopic neurons or clusters of neurons in the molecular layer, or bi-lamination (Blumcke et al. 2009). However, the contribution of abnormal brain development and neural activity in IGEs has just started to draw attention. For example, the knock-in mice harboring GABRG2 (R43Q) is a mutation associated with febrile seizures and childhood absence epilepsy. Activation of the mutant allele GABRG2 (R43Q) during early development increased the seizure susceptibility, and inactivation of the mutant allele would decrease the seizure susceptibility. These data suggest that disruption of the physiological effects of GABA_A receptors during the sensitive developmental epochs in fetal and neonatal/infantile life may compromise the developmental processes that are crucial for normal brain development and facilitate the development of epilepsy (Chiu et al. 2008). This may underlie why patients carrying GABA_A receptor subunit mutations have mental compromise ranging from mild learning difficulty to mental retardation in addition to seizures and autism such as those seen in Dravet syndrome (Li et al. 2011).

Abnormal GABA Receptor and Excitatory Receptors in Autism

The balance of excitatory and GABA neurotransmission throughout brain development determines the final trajectory of neural pathways responsible for cognitive and behavioral output in autism and epilepsy patients. Although it is unknown of how exactly GABA_A receptor function affects neurodevelopment, a recent study demonstrated that activation of GABA_A receptors leads to hyperpolarization, increased cell volume, and accumulation of stem cells in S phase, thereby causing a rapid decrease in cell proliferation (Andang et al. 2008). Any subtle change in stem cells may have profound impact

in neuronal function and leave imprint on later brain network activity. In addition to altered interneuron and excitatory neuronal numbers, studies on brain specimen of both autism and epilepsy from multiple investigators have consistently demonstrated that the expression of GABA_A receptors and ionotropic glutamate receptors is altered in both disorders (Fatemi et al. 2010; Jansen et al. 2010).

In the case of GABA, the alterations extend to multiple GABA_A receptor subunits instead of single GABA_A receptor subunit. This aberrant profile of GABA_A receptors is consistent with the complex GABA_A receptor assembly and dynamic expression patterns in the brain. In normal brain, expression of GABA_A receptor subunits varies regionally and temporally. For example, $\alpha 1$ and $\alpha 2$ subunits have low expression in early brain but increase over development and then stabilize through adolescence and adulthood, whereas $\alpha 4$ subunit expression is higher in infants than in older children. But based on a recent study in epilepsy patient brains, the normal expression pattern of GABA_A receptor subunit is absent in those with focal cortical dysplasia and in those with gliosis (Jansen et al. 2010). Brain specimens of autism patients have demonstrated that systematic changes in GABA_A subunit expression (Fatemi et al. 2010; Fatemi et al. 2009). Oblak demonstrated reduction of GABA_A receptors and benzodiazepine binding sites in both the anterior and posterior cingulate cortices and fusiform gyrus in autism (Oblak et al. 2009, 2011). Given the fact that GABA_A receptor traffics and functions as pentamer at the cell surface and synapse and the receptor stoichiometry of $2\alpha(x)2\beta(x)1\gamma$, $2\alpha(x)2\beta(x)1\delta$ or $2\alpha(x)3\beta(x)/3\alpha(x)2\beta(x)$, it is not surprising that multiple GABA_A receptor subunits are reduced. The brain regions that displayed different distribution include superior frontal cortex, parietal cortex, and cerebellum of subjects with autism (Fatemi et al. 2009, 2010). The fact that multiple regions are affected suggests the comprehensiveness of neurodevelopment abnormalities. There are also multiple mechanisms underlying the GABA_A receptor subunit protein reduction: (1) altered GABA_A receptor mRNA levels or stability, (2) altered posttranslational modifications

which reduce subunit maturation and receptor forward trafficking, (3) reduced neuronal numbers due to cell death of presynaptic interneurons, and (4) altered epigenetic regulations of GABA_A receptor subunits (Samaco et al. 2005).

Further research is needed to more fully understand how glutamate receptor and transporter biology is affected in autism and how it may contribute to epilepsy as a comorbidity in ASD. The core features of autistic brain development suggest that alterations of excitatory neuronal numbers may, in themselves, account for changes in excitatory receptor concentrations. In addition to abnormal minicolumnar structure in the frontal, temporal, and anterior cingulate cortices, Courchesne et al. (2011) recently reported that neuronal counts in the dorsolateral prefrontal cortex and the medial prefrontal cortex in autistic brains were increased by 79% and 29%, respectively, compared to similar young age-matched males (Casanova 2006). The idea of increased proliferation or reduced apoptosis in autistic brains leading to excessive production of excitatory neurons may explain the increases in many axonal tracts of autistic brains and increased local functional connectivity (Allen and Courchesne 2003). Studies of the blood have shown either increases or decreases in blood glutamate or blood glutamine (Lam et al. 2006). Gene expression studies have shown upregulation of several glutamate receptors or linked genes including EAAT1, GluR1, GluR2, and GluR3 mRNAs in the cerebellum and hippocampus of autistic individuals (Purcell et al. 2001). Surprisingly, few actual glutamate receptors or transporters are among identified ASD-associated genes including kainate receptor GluR6, metabotropic GluR8 (GRM8), NMDA receptor GRIN2A, and AMPA receptor-associated protein GRIP1 (Barnby et al. 2005; Choudhury et al. 2012; Mejias et al. 2011). Many of these associations are significant since these proteins control interneuron excitability (GluR6, GRIN2A), gamma oscillations from parvalbumin interneurons, critical periods (GRIN2A), and AMPA/GABA receptor expression (GRIP1 variants) in synapses (Endele et al. 2010; Fisahn et al. 2004; Kocsis 2011; Mejias et al. 2011; Zhang and Sun 2011).

Abnormal Neurotransmitter Signaling in Neurodevelopmental Disorders with Autism and Epilepsy as a Phenotype

Epilepsy and autism are comorbid in several neurodevelopmental disorders which, at their core, have impaired synaptic homeostasis. These disorders include, but are not limited to, Dravet syndrome, tuberous sclerosis, Angelman syndrome, fragile X syndrome, and Rett syndrome. GABA signaling could participate in neurodevelopmental disorders because GABA occupies a central role in development. Patients with these syndromes often suffer from autism, intellectual disabilities, and epilepsy. GABAergic function impairment may be a common pathway for many neurodevelopmental disorders including autism and epilepsy. It is not surprising given the essential role of GABA signaling in brain development. The exact mechanism of the concurrent epilepsy and autism in these neurodevelopmental disorders remains unclear. However, from both animal and genetic studies, altered GABAergic signaling is consistently observed in these disorders. In Angelman syndrome, a maternally inherited deletion of 15q11-13 locus is identified in the majority of cases. The deletion includes the UBE3A gene (ubiquitin-protein ligases E3A) as well as three GABA_A receptor subunit genes: the GABRB3, GABRA5, and GABRG3 (Hogart et al. 2007; Knoll et al. 1989; Wagstaff et al. 1991). UBE3A signaling controls EphB-EphrinB regulation of glutamate synapse development (Margolis et al. 2010). Conversely, increased dosage of UBE3A results in mice with autistic-like behavior and decreased glutamate neurotransmission (Smith et al. 2011).

In fragile X syndrome, GABA_A receptor subunits are downregulated, possibly due to lack of translational regulation by FMRP1 (D'Hulst et al. 2006; Fatemi et al. 2011). Studies on fragile X syndrome indicate that the increased activation of metabotropic glutamate receptor 5 (mGluR5), a member of the group I mGluR family, contributes to a predisposition for the development of epilepsy, autism, and other neuropsychiatric disorders (Bianchi et al. 2009). The major neuropsychiatric features of fragile X syndrome are caused by unchecked activation of mGluR5

(Bear et al. 2004), and downregulation of postsynaptic mGluR5 signaling can correct developmental disorders in the disease (Dolen et al. 2007). Depolarized firing states of neurons are driven by recurrent local excitation and inhibition in a given cortical region which underlies the so-called slow oscillation neocortical rhythm (<1 Hz). Aberrant postsynaptic mGluR5 signaling in neocortex of FXR1 KO mice alters this slow oscillation and inhibitory dendritic currents from somatostatin interneurons (Hays et al. 2011; Paluszkiwicz et al. 2011). Additionally, transient local hyperconnectivity of prefrontal cortex during development and abnormal presynaptic short-term plasticity contributes to slower synaptic responses and integration of informational processing in the FXR KO mice (Deng et al. 2011; Testa-Silva et al. 2011). All these mechanisms contribute to the aberrant maturation and function of synchronized cortical rhythmic activity needed for behavioral and cognitive output of neural networks.

In FXR KO mice, increased mTOR signaling and increased protein synthesis is associated with the above described synaptic pathology and seizures (Carson et al. 2012). In contrast, recent advances in TSC KO mouse studies show increased synaptic mTOR signaling, and *decreased* protein synthesis is associated with reductions in glutamate and GABA neurotransmission (Auerbach et al. 2011). In tuberous sclerosis (TSC), there is altered transcription of genes encoding glutamatergic and gamma-aminobutyric acid (GABA)-ergic receptors. GABRA1 and GABRA2 mRNA levels were reduced in both dysplastic neurons and giant cells compared to control neurons (White et al. 2001). In Rett syndrome, the recent study by Zoghbi et al. (2010) demonstrated that MeCP2 is critical for normal function of GABA-releasing neurons and that subtle dysfunction of GABAergic neurons alone can recapitulate the numerous neuropsychiatric phenotypes and epilepsy common to Rett syndrome patients (Chao et al. 2010). In addition to this comprehensive list of neurodevelopmental disorders, impaired GABAergic signaling is frequently identified in other ASD- and epilepsy-associated genes too. For example, genetic disruption of the autism spectrum disorder risk genes

MET, PLAUR, and neuropilin-2 induces the alteration of GABA_A receptor subunits and defects in GABAergic and excitatory circuitry which lead to autistic-like traits and epilepsy (Brooks-Kayal 2010; Eagleson et al. 2010; Gant et al. 2009; Powell et al. 2003).

In summary, the phenotypical and genomic heterogeneity of both autism and epilepsy still remains a challenge. These findings suggest that the genetic heterogeneity of both autism and epilepsy may produce similar deficits by bidirectional deviation from normal synaptic homeostasis at GABAergic or excitatory synapses. Future work using human neuropathology specimens and models especially those associated with neurodevelopmental abnormalities will further elucidate the molecular pathophysiological mechanisms of aberrant neurotransmitter receptors and their effects on signaling in autism alone, epilepsy alone, or patients with the autism and epilepsy phenotype.

Treatment

See ► [Neurologist](#)

See Also

► [Seizure](#)

References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, 9(5), 341–355.
- Allen, G., & Courchesne, E. (2003). Differential effects of developmental cerebellar abnormality on cognitive and motor functions in the cerebellum: An fMRI study of autism. *The American Journal of Psychiatry*, 160(2), 262–273.
- Amiet, C., Gourfinkel-An, I., Bouzamondo, A., Tordjman, S., Baulac, M., Lechat, P., et al. (2008). Epilepsy in autism is associated with intellectual disability and gender: Evidence from a meta-analysis. *Biological Psychiatry*, 64(7), 577–582.
- Andang, M., Hjerling-Leffler, J., Moliner, A., Lundgren, T. K., Castelo-Branco, G., Nanou, E., et al. (2008). Histone H2AX-dependent GABA(A) receptor regulation of stem cell proliferation. *Nature*, 451(7177), 460–464.
- Auerbach, B. D., Osterweil, E. K., & Bear, M. F. (2011). Mutations causing syndromic autism define an axis of synaptic pathophysiology. *Nature*, 480(7375), 63–68.
- Ballaban-Gil, K., & Tuchman, R. (2000). Epilepsy and epileptiform EEG: Association with autism and language disorders. *Mental Retardation and Developmental Disabilities Research Reviews*, 6(4), 300–308.
- Barnby, G., Abbott, A., Sykes, N., Morris, A., Weeks, D. E., Mott, R., et al. (2005). Candidate-gene screening and association analysis at the autism-susceptibility locus on chromosome 16p: Evidence of association at GRIN2A and ABAT. *American Journal of Human Genetics*, 76(6), 950–966.
- Behar, T. N., Schaffner, A. E., Scott, C. A., Greene, C. L., & Barker, J. L. (2000). GABA receptor antagonists modulate postmitotic cell migration in slice cultures of embryonic rat cortex. *Cerebral Cortex*, 10(9), 899–909.
- Benbadis, S. R. (2005). Practical management issues for idiopathic generalized epilepsies [Review]. *Epilepsia*, 46(Suppl. 9), 125–132.
- Berg, A. T., Berkovic, S. F., Brodie, M. J., Buchhalter, J., Cross, J. H., van Emde Boas, W., et al. (2010). Revised terminology and concepts for organization of seizures and epilepsies: Report of the ILAE commission on classification and terminology, 2005–2009. *Epilepsia*, 51(4), 676–685.
- Berg, A. T., Plioplys, S., & Tuchman, R. (2011). Risk and correlates of autism spectrum disorders in children with epilepsy: A community-based study. *Journal of Child Neurology*, 26(5), 540–547.
- Berkovic, S. F., Harkin, L., McMahon, J. M., Pelekanos, J. T., Zuberi, S. M., Wirrell, E. C., et al. (2006). De-novo mutations of the sodium channel gene *SCN1A* in alleged vaccine encephalopathy: A retrospective study. *Lancet Neurology*, 5(6), 488–492.
- Bloss, C. S., & Courchesne, E. (2007). MRI neuroanatomy in young girls with autism: A preliminary study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46(4), 515–523.
- Blumcke, I., Kistner, I., Clusmann, H., Schramm, J., Becker, A. J., Elger, C. E., et al. (2009). Towards a clinico-pathological classification of granule cell dispersion in human mesial temporal lobe epilepsies. *Acta Neuropathologica*, 117(5), 535–544.
- Bolton, P. F., Carcani-Rathwell, I., Hutton, J., Goode, S., Howlin, P., & Rutter, M. (2011). Epilepsy in autism: Features and correlates. *The British Journal of Psychiatry*, 198(4), 289–294.
- Brambilla, P., Hardan, A., di Nemi, S. U., Perez, J., Soares, J. C., & Barale, F. (2003). Brain anatomy and development in autism: Review of structural MRI studies. *Brain Research Bulletin*, 61(6), 557–569.
- Brooks-Kayal, A. (2010). Epilepsy and autism spectrum disorders: Are there common developmental mechanisms? *Brain & Development*, 32(9), 731–738.

- Carmona-Mora, P., & Walz, K. (2010). Retinoic acid induced 1, RAI1: A dosage sensitive gene related to neurobehavioral alterations including autistic behavior. *Current Genomics*, 11(8), 607–617.
- Carson, R. P., Van Nielen, D. L., Winzenburger, P. A., & Ess, K. C. (2012). Neuronal and glia abnormalities in Tsc1-deficient forebrain and partial rescue by rapamycin. *Neurobiology of Disease*, 45(1), 369–380.
- Casanova, M. F. (2006). Neuropathological and genetic findings in autism: The significance of a putative mini-columnopathy [Review]. *The Neuroscientist*, 12(5), 435–441.
- Chao, H. T., Chen, H., Samaco, R. C., Xue, M., Chahrouh, M., Yoo, J., et al. (2010). Dysfunction in GABA signalling mediates autism-like stereotypies and Rett syndrome phenotypes. *Nature*, 468(7321), 263–269.
- Chez, M. G., Chang, M., Krasne, V., Coughlan, C., Kominsky, M., & Schwartz, A. (2006). Frequency of epileptiform EEG abnormalities in a sequential screening of autistic patients with no known clinical epilepsy from 1996 to 2005. *Epilepsy & Behavior: E&B*, 8(1), 267–271. Epub January 5, 2006.
- Chiu, C., Reid, C. A., Tan, H. O., Davies, P. J., Single, F. N., Koukoulas, I., et al. (2008). Developmental impact of a familial GABAA receptor epilepsy mutation. *Annals of Neurology*, 64(3), 284–293.
- Choudhury, P. R., Lahiri, S., & Rajamma, U. (2012). Glutamate mediated signaling in the pathophysiology of autism spectrum disorders. *Pharmacology Biochemistry and Behavior*, 100(4), 841–849. Epub July 5, 2011.
- Courchesne, E. (2002). Abnormal early brain development in autism. *Molecular Psychiatry*, 7(Suppl. 2), S21–S23.
- Courchesne, E., Carper, R., & Akshoomoff, N. (2003). Evidence of brain overgrowth in the first year of life in autism. *Journal of the American Medical Association*, 290(3), 337–344.
- Courchesne, E., Mouton, P. R., Calhoun, M. E., Semendeferi, K., Ahrens-Barbeau, C., Hallet, M. J., et al. (2011). Neuron number and size in prefrontal cortex of children with autism. *Journal of the American Medical Association*, 306(18), 2001–2010.
- Cuccaro, M. L., Tuchman, R. F., Hamilton, K. L., Wright, H. H., Abramson, R. K., Haines, J. L., et al. (2011, November 22). Exploring the relationship between autism spectrum disorder and epilepsy using latent class cluster analysis. *Journal of Autism and Developmental Disorders*. [Epub ahead of print].
- D'Hulst, C., De Geest, N., Reeve, S. P., Van Dam, D., De Deyn, P. P., Hassan, B. A., et al. (2006). Decreased expression of the GABAA receptor in fragile X syndrome. *Brain Research*, 1121(1), 238–245.
- Danielsson, S., Gillberg, I. C., Billstedt, E., Gillberg, C., & Olsson, I. (2005). Epilepsy in young adults with autism: A prospective population-based follow-up study of 120 individuals diagnosed in childhood. *Epilepsia*, 46(6), 918–923.
- Delahanty, R. J., Kang, J. Q., Brune, C. W., Kistner, E. O., Courchesne, E., Cox, N. J., et al. (2011). Maternal transmission of a rare GABRB3 signal peptide variant is associated with autism. *Molecular Psychiatry*, 16(1), 86–96.
- Deng, P. Y., Sojka, D., & Klyachko, V. A. (2011). Abnormal presynaptic short-term plasticity and information processing in a mouse model of fragile X syndrome. *Journal of Neuroscience*, 31(30), 10971–10982.
- Dibbens, L. M., Harkin, L. A., Richards, M., Hodgson, B. L., Clarke, A. L., Petrou, S., et al. (2009). The role of neuronal GABA(A) receptor subunit mutations in idiopathic generalized epilepsies. *Neuroscience Letters*, 453(3), 162–165.
- Dulac, O. (2001). Epileptic encephalopathy. *Epilepsia*, 42 (Suppl. 3), 23–26.
- Eagleson, K. L., Gravielle, M. C., Schlueter McFadyen-Ketchum, L. J., Russek, S. J., Farb, D. H., & Levitt, P. (2010). Genetic disruption of the autism spectrum disorder risk gene PLAUR induces GABAA receptor subunit changes. *Neuroscience*, 168(3), 797–810.
- Eagleson, K. L., Campbell, D. B., Thompson, B. L., Bergman, M. Y., & Levitt, P. (2011). The autism risk genes MET and PLAUR differentially impact cortical development. *Autism Research*, 4(1), 68–83.
- Endele, S., Rosenberger, G., Geider, K., Popp, B., Tamer, C., Stefanova, I., et al. (2010). Mutations in GRIN2A and GRIN2B encoding regulatory subunits of NMDA receptors cause variable neurodevelopmental phenotypes. *Nature Genetics*, 42(11), 1021–1026.
- Engel, J., Jr. (2001). A proposed diagnostic scheme for people with epileptic seizures and with epilepsy: Report of the ILAE Task Force on classification and terminology. *Epilepsia*, 42(6), 796–803.
- Escayg, A., De Waard, M., Lee, D. D., Bichet, D., Wolf, P., Mayer, T., et al. (2000). Coding and noncoding variation of the human calcium-channel beta4-subunit gene *CACNB4* in patients with idiopathic generalized epilepsy and episodic ataxia. *American Journal of Human Genetics*, 66(5), 1531–1539.
- Fatemi, S. H., Reutiman, T. J., Folsom, T. D., & Thuras, P. D. (2009). GABA_A receptor downregulation in brains of subjects with autism. *Journal of Autism and Developmental Disorders*, 39(2), 223–230.
- Fatemi, S. H., Reutiman, T. J., Folsom, T. D., Rooney, R. J., Patel, D. H., & Thuras, P. D. (2010). mRNA and protein levels for GABA_Aα4, α5, β1 and GABA_BR1 receptors are altered in brains from subjects with autism. *Journal of Autism and Developmental Disorders*, 40(6), 743–750.
- Fatemi, S. H., Folsom, T. D., Kneeland, R. E., & Liesch, S. B. (2011). Metabotropic glutamate receptor 5 upregulation in children with autism is associated with underexpression of both Fragile X mental retardation protein and GABAA receptor beta 3 in adults with autism. *The Anatomical Record (Hoboken)*, 294(10), 1635–1645.
- Fisahn, A., Contractor, A., Traub, R. D., Buhl, E. H., Heinemann, S. F., & McBain, C. J. (2004). Distinct roles for the kainate receptor subunits GluR5 and GluR6 in kainate-induced hippocampal gamma oscillations. *Journal of Neuroscience*, 24(43), 9658–9668.

- Fisher, R. S., van Emde Boas, W., Blume, W., Elger, C., Genton, P., Lee, P., et al. (2005). Epileptic seizures and epilepsy: Definitions proposed by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE). *Epilepsia*, 46(4), 470–472.
- Gant, J. C., Thibault, O., Blalock, E. M., Yang, J., Bachstetter, A., Kotick, J., et al. (2009). Decreased number of interneurons and increased seizures in neuropilin 2 deficient mice: Implications for autism and epilepsy. *Epilepsia*, 50(4), 629–645.
- Geschwind, D. H., & Levitt, P. (2007). Autism spectrum disorders: Developmental disconnection syndromes. *Current Opinion in Neurobiology*, 17(1), 103–111.
- Glasscock, E., Qian, J., Yoo, J. W., & Noebels, J. L. (2007). Masking epilepsy by combining two epilepsy genes. *Nature Neuroscience*, 10(12), 1554–1558.
- Hara, H. (2007). Autism and epilepsy: A retrospective follow-up study. *Brain & Development*, 29(8), 486–490.
- Hays, S. A., Huber, K. M., & Gibson, J. R. (2011). Altered neocortical rhythmic activity states in Fmr1 KO mice are due to enhanced mGluR5 signaling and involve changes in excitatory circuitry. *Journal of Neuroscience*, 31(40), 14223–14234.
- Hogart, A., Nagarajan, R. P., Patzel, K. A., Yasui, D. H., & Lasalle, J. M. (2007). 15q11-13 GABAA receptor genes are normally biallelically expressed in brain yet are subject to epigenetic dysregulation in autism-spectrum disorders. *Human Molecular Genetics*, 16, 691–703.
- ILAE. (1981). ILAE commission on classification and terminology of the international league against epilepsy: Proposal for revised clinical and electroencephalographic classification of epileptic seizures. *Epilepsia*, 22, 489–501.
- Jansen, L. A., Peugh, L. D., Roden, W. H., & Ojemann, J. G. (2010). Impaired maturation of cortical GABA (A) receptor expression in pediatric epilepsy. *Epilepsia*, 51(8), 1456–1467.
- Kagan-Kushnir, T., Roberts, S. W., & Snead, O. C., 3rd. (2005). Screening electroencephalograms in autism spectrum disorders: Evidence-based guideline. *Journal of Child Neurology*, 20(3), 197–206.
- Kang, J. Q., & Macdonald, R. L. (2009). Making sense of nonsense GABA(A) receptor mutations associated with genetic epilepsies. *Trends in Molecular Medicine*, 15(9), 430–438.
- Knoll, J. H., Nicholls, R. D., Magenis, R. E., Graham, J. M., Jr., Lalande, M., & Latt, S. A. (1989). Angelman and Prader-Willi syndromes share a common chromosome 15 deletion but differ in parental origin of the deletion. *American Journal of Medical Genetics*, 32, 285–290.
- Kocsis, B. (2011, November 2). Differential role of NR2A and NR2B subunits in *N*-methyl-D-aspartate receptor antagonist-induced aberrant cortical gamma oscillations. *Biological Psychiatry*. [Epub ahead of print].
- Lam, K. S., Aman, M. G., & Arnold, L. E. (2006). Neurochemical correlates of autistic disorder: A review of the literature [Review]. *Research in Developmental Disabilities*, 27(3), 254–289. Epub July 5, 2005.
- Landau, W. M., & Kleffner, F. R. (1998). Syndrome of acquired aphasia with convulsive disorder in children 1957. *Neurology*, 51(5), 1241, 1248 pages following 1241.
- Li, B. M., Liu, X. R., Yi, Y. H., Deng, Y. H., Su, T., Zou, X., et al. (2011). Autism in Dravet syndrome: Prevalence, features, and relationship to the clinical characteristics of epilepsy and mental retardation. *Epilepsy & Behavior*, 21(3), 291–295.
- Ma, D. Q., Whitehead, P. L., Menold, M. M., Martin, E. R., Ashley-Koch, A. E., Mei, H., et al. (2005). Identification of significant association and gene-gene interaction of GABA receptor subunit genes in autism. *American Journal of Human Genetics*, 77(3), 377–388.
- Macdonald, R. L., Kang, J. Q., & Gallagher, M. J. (2010). Mutations in GABAA receptor subunits associated with genetic epilepsies. *The Journal of Physiology*, 588(Pt. 11), 1861–1869.
- Margolis, S. S., Salogiannis, J., Lipton, D. M., Mandel-Brehm, C., Wills, Z. P., Mardinly, A. R., et al. (2010). EphB-mediated degradation of the RhoA GEF Ephexin5 relieves a developmental brake on excitatory synapse formation. *Cell*, 143(3), 442–455.
- Mejias, R., Adamczyk, A., Anggono, V., Niranjana, T., Thomas, G. M., Sharma, K., et al. (2011). Gain-of-function glutamate receptor interacting protein 1 variants alter GluA2 recycling and surface distribution in patients with autism. *Proceedings of the National Academy of Sciences of the United States of America*, 108(12), 4920–4925.
- Morrell, F., & de Toledo-Morrell, L. (1999). From mirror focus to secondary epileptogenesis in man: An historical review [Review]. *Advances in Neurology*, 81, 11–23. No abstract available.
- Nabbout, R., & Dulac, O. (2003). Epileptic encephalopathies: A brief overview. *Journal of Clinical Neurophysiology*, 20(6), 393–397.
- Nabbout, R., & Dulac, O. (2008). Epileptic syndromes in infancy and childhood. *Current Opinion in Neurology*, 21(2), 161–166.
- Nickels, K., & Wirrell, E. (2008). Electrical status epilepticus in sleep. *Seminars in Pediatric Neurology*, 15(2), 50–60.
- Numis, A. L., Major, P., Montenegro, M. A., Muzykewicz, D. A., Pulsifer, M. B., & Thiele, E. A. (2011). Identification of risk factors for autism spectrum disorders in tuberous sclerosis complex. *Neurology*, 76(11), 981–987.
- O’Roak, B. J., & State, M. W. (2008). Autism genetics: Strategies, challenges, and opportunities. *Autism Research*, 1(1), 4–17.
- Oblak, A., Gibbs, T. T., & Blatt, G. J. (2009). Decreased GABAA receptors and benzodiazepine binding sites in the anterior cingulate cortex in autism. *Autism Research*, 2, 205–219.
- Oblak, A. L., Gibbs, T. T., & Blatt, G. J. (2011). Reduced GABA(A) receptors and benzodiazepine binding sites

- in the posterior cingulate cortex and fusiform gyrus in autism. *Brain Research*, 1380, 218–228. Epub September 19, 2010.
- Paluszkievicz, S. M., Olmos-Serrano, J. L., Corbin, J. G., & Huntsman, M. M. (2011). Impaired inhibitory control of cortical synchronization in fragile X syndrome. *Journal of Neurophysiology*, 106(5), 2264–2272.
- Powell, E. M., Campbell, D. B., Stanwood, G. D., Davis, C., Noebels, J. L., & Levitt, P. (2003). Genetic disruption of cortical interneuron development causes region- and GABA cell type-specific deficits, epilepsy, and behavioral dysfunction. *Journal of Neuroscience*, 23(2), 622–631.
- Purcell, A. E., Jeon, O. H., Zimmerman, A. W., Blue, M. E., & Pevsner, J. (2001). Postmortem brain abnormalities of the glutamate neurotransmitter system in autism. *Neurology*, 57(9), 1618.
- Racine, R., Tuff, L., & Zaide, J. (1975). Kindling, unit discharge patterns and neural plasticity. *Canadian Journal of Neurological Sciences*, 2(4), 395–405.
- Samaco, R. C., Hogart, A., & LaSalle, J. M. (2005). Epigenetic overlap in autism-spectrum neurodevelopmental disorders: MECP2 deficiency causes reduced expression of UBE3A and GABRB3. *Human Molecular Genetics*, 14(4), 483–492.
- Sasaki, M., Nakagawa, E., Sugai, K., Shimizu, Y., Hattori, A., Nonoda, Y., et al. (2010). Brain perfusion SPECT and EEG findings in children with autism spectrum disorders and medically intractable epilepsy. *Brain & Development*, 32(9), 776–782.
- Scheffer, I. E., Zhang, Y. H., Jansen, F. E., & Dibbens, L. (2009). Dravet syndrome or genetic (generalized) epilepsy with febrile seizures plus? *Brain & Development*, 31(5), 394–400.
- Schumann, C. M., Hamstra, J., Goodlin-Jones, B. L., Lotspeich, L. J., Kwon, H., Buonocore, M. H., et al. (2004). The amygdala is enlarged in children but not adolescents with autism; the hippocampus is enlarged at all ages. *Journal of Neuroscience*, 24(28), 6392–6401.
- Schumann, C. M., Bloss, C. S., Carter Barnes, C., Wideman, G. M., Carper, R. A., Akshoomoff, N., et al. (2010). Longitudinal magnetic resonance imaging study of cortical development through early childhood in autism. *Journal of Neuroscience*, 30(12), 4419–4427.
- Sisodiya, S. M., & Mefford, H. C. (2011). Genetic contribution to common epilepsies. *Current Opinion in Neurology*, 24(2), 140–145.
- Smith, S. E., Zhou, Y. D., Zhang, G., Jin, Z., Stoppel, D. C., & Anderson, M. P. (2011). Increased gene dosage of Ube3a results in autism traits and decreased glutamate synaptic transmission in mice. *Science Translational Medicine*, 3(103), 103–197.
- Spence, S. J., & Schneider, M. T. (2009). The role of epilepsy and epileptiform EEGs in autism spectrum disorders. *Pediatric Research*, 65(6), 599–606.
- Steinlein, O. K., & Bertrand, D. (2010). Nicotinic receptor channelopathies and epilepsy. *Pflügers Archiv*, 460(2), 495–503.
- Suzuki, T., Delgado-Escueta, A. V., Aguan, K., Alonso, M. E., Shi, J., Hara, Y., et al. (2004). Mutations in EFHC1 cause juvenile myoclonic epilepsy. *Nature Genetics*, 36(8), 842–849.
- Suzuki, T., Inoue, I., Yamagata, T., Morita, N., Furuichi, T., & Yamakawa, K. (2008). Sequential expression of Efhc1/myoclonin1 in choroid plexus and ependymal cell cilia. *Biochemical and Biophysical Research Communications*, 367(1), 226–233.
- Tassinari, C. A., Cantalupo, G., Rios-Pohl, L., Giustina, E. D., Rubboli, G., et al. (2009). Encephalopathy with status epilepticus during slow sleep: “The Penelope syndrome”. *Epilepsia*, 50(Suppl. 7), 4–8.
- Taylor, D. C., Neville, B. G., & Cross, J. H. (1999). Autistic spectrum disorders in childhood epilepsy surgery candidates. *European Child & Adolescent Psychiatry*, 8(3), 189–192.
- Testa-Silva, G., Loebel, A., Giugliano, M., de Kock, C. P., Mansvelder, H. D., & Meredith, R. M. (2011, August 19). Hyperconnectivity and slow synapses during early development of medial prefrontal cortex in a mouse model for mental retardation and autism. *Cerebral Cortex*. [Epub ahead of print].
- Thom, M., Mathern, G. W., Cross, J. H., & Bertram, E. H. (2010). Mesial temporal lobe epilepsy: How do we improve surgical outcome? [Review]. *Annals of Neurology*, 68(4), 424–434.
- Tuchman, R. (2009). CSWS-related autistic regression versus autistic regression without CSWS. *Epilepsia*, 50(Suppl. 7), 18–20.
- Tuchman, R., Alessandri, M., & Cuccaro, M. (2010a). Autism spectrum disorders and epilepsy: Moving towards a comprehensive approach to treatment. *Brain & Development*, 32(9), 719–730.
- Tuchman, R., Cuccaro, M., & Alessandri, M. (2010b). Autism and epilepsy: Historical perspective. *Brain & Development*, 32(9), 709–718.
- Turk, J., Bax, M., Williams, C., Amin, P., Eriksson, M., & Gillberg, C. (2009). Autism spectrum disorder in children with and without epilepsy: Impact on social functioning and communication. *Acta Paediatrica*, 98(4), 675–681.
- Veenstra-VanderWeele, J., & Cook, E. H., Jr. (2004). Molecular genetics of autism spectrum disorder. *Molecular Psychiatry*, 9(9), 819–832.
- Vincent, J. B., Horike, S. I., Choufani, S., Paterson, A. D., Roberts, W., Szatmari, P., et al. (2006). An inversion inv(4)(p12-p15.3) in autistic siblings implicates the 4p GABA receptor gene cluster. *Journal of Medical Genetics*, 43(5), 429–434.
- Wagstaff, J., Chaillet, J. R., & Lalonde, M. (1991). The GABAA receptor beta 3 subunit gene: Characterization of a human cDNA from chromosome 15q11q13 and mapping to a region of conserved synteny on mouse chromosome 7. *Genomics*, 11(4), 1071–1078.
- Wagstaff, J., Knoll, J. H., Glatt, K. A., Shugart, Y. Y., Sommer, A., & Lalonde, M. (1992). Maternal but not paternal transmission of 15q11-13-linked nondelation

- Angelman syndrome leads to phenotypic expression. *Nature Genetics*, 1(4), 291–294.
- Wang, Y. Y., Smith, P., Murphy, M., & Cook, M. (2010). Global expression profiling in epileptogenesis: Does it add to the confusion? [Review]. *Brain Pathology*, 20(1), 1–16. Epub February 24, 2009.
- Wegiel, J., Kuchna, I., Nowicki, K., Imaki, H., Wegiel, J., Marchi, E., et al. (2010). The neuropathology of autism: Defects of neurogenesis and neuronal migration, and dysplastic changes. *Acta Neuropathologica*, 119(6), 755–770.
- Weiss, L. A. (2009). Autism genetics: Emerging data from genome-wide copy-number and single nucleotide polymorphism scans. *Expert Review of Molecular Diagnostics*, 9(8), 795–803.
- Weiss, L. A., Arking, D. E., Daly, M. J., Chakravarti, A., & Gene Discovery Project of Johns Hopkins & the Autism Consortium. (2009). A genome-wide linkage and association scan reveals novel loci for autism. *Nature*, 461(7265), 802–808.
- White, R., Hua, Y., Scheithauer, B., Lynch, D. R., Henske, E. P., & Crino, P. B. (2001). Selective alterations in glutamate and GABA receptor subunit mRNA expression in dysplastic neurons and giant cells of cortical tubers. *Annals of Neurology*, 49(1), 67–78.
- Wolff, M., Casse-Perrot, C., & Dravet, C. (2006). Severe myoclonic epilepsy of infants (Dravet syndrome): Natural history and neuropsychological findings. *Epilepsia*, 47(Suppl. 2), 45–48.
- Zhang, Z., & Sun, Q. Q. (2011). Development of NMDA NR2 subunits and their roles in critical period maturation of neocortical GABAergic interneurons. *Developmental Neurobiology*, 71(3), 221–245.
- Zoghbi, H. Y., et al. (2010). Dysfunction in GABA signaling mediates autism-like stereotypies and Rett syndrome phenotypes. *Nature*, 468(7321), 263–269.
- Zupanc, M. L. (2009). Clinical evaluation and diagnosis of severe epilepsy syndromes of early childhood. *Journal of Child Neurology*, 24(Suppl. 8), 6S–14S.

Epilepsy and Autism

► Autism and Epilepsy

Epileptiform Abnormalities

► EEG Abnormalities as a Biomarker of Severity in ASD

Epinephrine

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Synonyms

Adrenaline; L-Adrenalin; L-Epinephrine; L-Epinephrine; Levo-Methylaminoethanol-catechol; Levoreninum; Lyodrin

Definition

Epinephrine is a catecholamine transmitter and hormone, generated from the amino acid tyrosine (or phenylalanine) in a common biosynthetic pathway with dopamine and norepinephrine. The enzyme tyrosine hydroxylase converts tyrosine to l-dihydroxyphenylalanine (l-DOPA). l-DOPA is then decarboxylated to dopamine by DOPA decarboxylase (DDC). Dopamine β -hydroxylase converts dopamine to norepinephrine. Finally, epinephrine is generated by N-methylation of norepinephrine, a process catalyzed by phenylethanolamine N-methyltransferase (E.C. 2.1.1.28, PNMT; Axelrod, 1962), utilizing S-adenosylmethionine as the methyl donor.

The adrenal medulla is the major site of peripheral epinephrine production. Once released in the blood, it causes systemic vasoconstriction and gastrointestinal relaxation, stimulates the heart, and dilates bronchi and cerebral vessels.

In the brain, epinephrine-producing (adrenergic) neurons were identified by their expression of PNMT. Immunohistochemical studies in animals have established several groups of adrenergic neurons (PNMT+) in the medulla oblongata: ventrolateral A1 and C1 cell groups, dorsomedial A2 and C2 groups, a rostral midline C3 group, and an additional unnumbered compact group in the dorsolateral portion of the nucleus tractus solitarii (nTS). PNMT-positive

neurons have also been mapped in the human medulla.

Epinephrine exerts its actions via three distinct types of adrenergic receptors: $\alpha 1$, $\alpha 2$, β . The $\alpha 1$ receptor class ($\alpha 1A$, $\alpha 1B$, and $\alpha 1D$ receptors) is coupled to Gq-type G-proteins. The $\alpha 2$ class ($\alpha 2A$, $\alpha 2B$, and $\alpha 2C$) of receptors is coupled to Gi-type G-proteins. The β class of receptors ($\beta 1$, $\beta 2$, and $\beta 3$) couples to Gs-type G-proteins.

Epinephrine is catabolized to the inactive compound L-metanephrine through the action of catecholamine-O-methyltransferase (COMT).

See Also

- Catecholamine System
- Catechol-O-Methyltransferase

References and Reading

- Gai, W. P., et al. (1993). Loss of C1 and C3 epinephrine-synthesizing neurons in the medulla oblongata in Parkinson's disease. *Annals of Neurology*, 33, 357–367.
- IUPHAR database. <http://www.iuphar-db.org/DATABASE/LigandDisplayForward?ligandId=509>
- Wong, D. L. (2006). Epinephrine biosynthesis: Hormonal and neural control during stress. *Cellular and Molecular Neurobiology*, 26(4–6), 891–900.
- Wurtman, R. J., & Axelrod, J. (1965). Adrenaline synthesis: Control by the pituitary gland and adrenal glucocorticoids. *Science*, 150, 1464–1465.

Episodic Memory

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Synonyms

[Autobiographical memory](#)

Definition

A subtype of the declarative memory system which stores personally experienced events (e.g., a memory of a specific place or time). Episodic memory is often associated with autobiographical context (e.g., remembering what happened on one's tenth birthday). This specific type of memory is assessed using a free recall task.

Research has shown that individuals with Autism Spectrum Disorder can have impairments in episodic memory.

See Also

- Declarative Memory
- Explicit Memory
- Free Recall
- Memory
- Memory Assessment
- Memory Development
- Recognition Memory
- Retrieval of Information
- Rote Memory
- Semantic Memory
- Short-Term Memory

References and Reading

- Lind, S. E., & Bowler, D. M. (2010). Episodic memory and episodic future thinking in adults with autism. *Journal of Abnormal Psychology*, 119, 896–905.
- Toichi, M., & Kamio, Y. (2003). Long-term memory in high-functioning autism: Controversy on episodic memory in autism reconsidered. *Journal of Autism and Developmental Disorders*, 33, 151–161.
- Tulving, E. (1983). *Elements of episodic memory*. Oxford, UK: Clarendon Press.
- Tulving, E. (1985). How many memory systems are there? *American Psychologist*, 40, 385–398.
- Wheeler, M. A., Stuss, D. T., & Tulving, E. (1997). Toward a theory of episodic memory: The frontal lobes and autoneoetic consciousness. *Psychological Bulletin*, 3, 331–354.

Epistasis

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Definition

In genetics, epistasis refers to the interaction between two or more genes, such that the phenotype differs from what would be expected if the genes exert their effects independently. The importance of epistasis has been increasingly recognized in human genetics in recent years, since it may be partially responsible for the “missing heritability” observed for complex diseases, that is, the aggregation of the marginal effects from genetic variants associated with a disease do not completely explain the heritability of the disease.

Historical Background

The term “epistasis” was first used by the English geneticist William Bateson in 1909 in his book “Mendel’s Principles of Heredity.” It was used to describe deviations from Mendelian inheritance patterns due to one gene masking the effects of another gene, that is, a particular variant in one locus prevents a variant at another locus from manifesting its effects. The book also popularized Mendel’s Law of Segregation and Law of Independent Assortment, originally conceived by Gregor Mendel in the eighteenth century and rediscovered in 1900. Bateson was also the first person to use the word “genetics” to describe the study of inheritance and the science of variation, and he codiscovered genetic linkage with Reginald Punnett.

There are many well-known examples of epistasis in genetics. One of the oldest examples

described in humans is on the expression of the ABO blood group system. Some individuals have a rare condition where they lack a protein called the H antigen. The H antigen is used to form A and B antigens, so these individuals cannot make A antigen or B antigen, and this scenario is often referred to as the “Bombay phenotype.” Individuals with Bombay phenotype can only be transfused with blood from other Bombay phenotype individuals. Even though these individuals with Bombay phenotype may have A or B genes, they cannot express the A or B blood group, and instead they appear to be blood group O. In this example, the H antigen masks the effect of the A or B genes, resulting in epistasis.

Another well-known example of epistasis is on genetics of coat color in mice. Two coat-color loci are involved in the control of coat color. At the first locus, having color (allele A) is dominant over lack of color (allele a). At the second locus, having the coat color agouti (allele B) is dominant over black (allele b). Consider a mouse that has homozygous aa alleles in the first locus: it will show no coat color, regardless of its genotype at the second locus (BB or Bb or bb). Thus, the variants at the first locus mask the effect of the second locus, and there is epistasis between the two loci.

It is important to emphasize that Bateson’s simple form of epistasis, as illustrated by the two well-known examples above, is merely a limited form of epistasis that works on Mendelian trait and is intuitively appealing to most biologists. However, a broader form of epistasis – from a population genetics perspective – involves more sophisticated statistical modeling and is more relevant for complex traits genetics. For example, two alleles at two different loci may each increase adult height by 1 cm on average, but when these two alleles occur together in the same individual, they may increase the height by 4 cm, which is more than what would be expected if the two alleles exert their effects independently. This broader form of epistasis is what typically emerges from current literature and is one of the hotly researched areas in genetics of complex diseases and traits.

Current Knowledge

Contribution of Epistasis to “Missing Heritability”

Genome-wide association studies (GWASs) have been very successful in identifying and replicating disease susceptibility loci for common and complex human diseases. These studies assay whole-genome SNP markers (typically 500,000 or more) for individuals grouped by different phenotypes (such as patients and control subjects) then test the statistical association between each marker and the phenotype. Top findings showing strong evidence of statistical significance are then replicated in another set of samples. As of December 2010, over 1,200 human GWASs have examined over 200 diseases and traits and found almost 4,000 SNP associations.

A question that often arises in GWAS is “where is the missing heritability?” The question refers to the fact that the collection of variants discovered in GWAS only explains a minor fraction of the heritability when simply added together, even for diseases or traits that are highly heritable. Multiple reasons have been proposed to explain the missing heritability, including (1) rare variants at known loci that act independent of common causal variants, (2) rare mutations in many thousands of loci, (3) copy number variants, (4) gene-gene interactions, (5) hidden environmental factors and epigenetic changes, and (6) wrong diagnosis of “common diseases.” Among these possible explanations, epistasis or gene-gene interactions could play a major role in contributing to “missing heritability,” since if the effect of one locus is masked by effects at another locus, power to detect the first locus is likely to be reduced and elucidation of the joint effects at the two loci will be hindered. If more than two loci are involved, complex multiway interactions may be present, further complicating the situation. For complex traits, given the known involvement of many contributing loci detected in GWAS, it is quite likely that epistasis will have to play a role. Therefore, it is reasonable to suspect that epistasis is a ubiquitous component of the genetic architecture of complex traits and diseases, such as neuropsychiatric and neurodevelopmental disorders.

Epistasis in Autism

Although several syndromes with single-gene causes, such as Rett syndrome and fragile X syndrome, share some phenotypic features with autism, it is widely recognized that autism is not a single-gene disease. Instead, the molecular pathophysiology of autism perhaps requires many genes in multiple biological pathways. However, the exact underlying genetic architecture for autism is still not well understood, and less is known on how different genetic components interact with each other. It is possible that rare, highly penetrant variants play a major role in each individual patient (oligogenic model), or it is also possible that thousands of variants, each with moderate effect sizes, contribute to autism pathogenesis. The first argument is supported by the observation that many highly penetrant mutations, especially large-scale deletions, have been enriched in patients with autism, although each patient may have different combinations of rare variants. The second argument is supported by the observation that sophisticated statistical approaches that utilize information from whole-genome markers, each with small effect sizes, can explain a large fraction of schizophrenia susceptibility. Besides these standing hypotheses, it is also possible that epistatic interactions play a major role in autism pathogenesis and that genetic variants with moderate effects sizes, when present in particular combination, will lead to much higher effect sizes that ultimately results in autism pathogenesis.

There are sporadic reports in literature on observed epistasis in autism susceptibility. For example, Coutinho et al. have tested for epistasis between loci with marginal effects in autism. In addition to the significant independent effects, evidence for interaction between SLC6A4 and ITGB3 markers was also found. The overall results implicate SLC6A4 and ITGB3 gene interactions in both autism etiology and in serotonin level determination, providing evidence for a common underlying genetic mechanism and a molecular explanation for the association of platelet hyperserotonemia with autism. With the wider application of GWAS and copy number variation studies, more epistasis may be observed in autism in the future.

Physical Interaction Versus Statistical Interaction

The word “interaction” is sometimes used to infer epistasis; however, there are two types of interactions in the contexts of genetics: physical interaction versus statistical interaction. Proteomics techniques, such as yeast two-hybrid systems, are now available to locate proteins that interact with one another in a protein complex. In these cases, proteins bind to each other, resulting in molecular functions that cannot be performed by each components of the protein complex. In contrast, statistical interaction summarizes genotype-phenotype relationships using population-level data rather than proteomic data. In fact, genes with statistical interactions do not have to physically bind to each other.

Statistical Methods to Detect Epistasis from Genetic Data

Mathematically, the quantitative genetic concept of epistasis may be represented for two loci by the linear model

$$y = u + a_1x_1 + a_2x_2 + d_1z_1 + d_2z_2 + i_{aa}x_1x_2 + i_{ad}x_1z_2 + i_{da}z_1x_2 + i_{dd}z_1z_2$$

where y is a quantitative phenotype and x_i and z_i are dummy variables related to the underlying genotype at locus i . The coefficients μ , a_1 , d_1 , a_2 , and d_2 represent genetic parameters that may be estimated corresponding to the mean effect, additive effect, and dominance effects at the two loci; i_{aa} , i_{da} , i_{da} , and i_{dd} correspond to epistatic interaction effects. Lack of epistasis in this model implies that all interaction coefficients are zero. In this case, the resulting model is

$$y = u + a_1x_1 + a_2x_2 + d_1z_1 + d_2z_2$$

where each allele exerts their effect independently with the effect size of a_1 , a_2 , d_1 , and d_2 , respectively.

In practice, it is more typical to assume a simple additive model where the effect sizes of a

homozygote doubles that of a heterozygote. Therefore, it is convenient to use a single variable x or z to denote the number of copies of the allele 2 present at the locus. The linear model can then be simplified as

$$y = u + ax + dx + i_{ad}xz$$

In this case, lack of epistasis in this model implies that i_{ad} is zero, that is, model will simply be

$$y = u + ax + dz$$

For binary traits, a logit function is typically used to model the response variable:

$$\begin{aligned} \log \text{it}(p) &= \log(p/(1-p)) \\ &= u + ax + dz + i_{ad}xz \end{aligned}$$

where the outcome of interest (such as probability of disease) is defined to be p .

It is important to note that besides these simple parametric linear models, other forms of modeling strategies also exist. There are unique advantages for using linear models as it is intuitively simple to interpret and it is relatively easy to implement. However, they may have limitations for detecting nonlinear patterns of interactions. For example, when many genes are involved in explaining complex diseases or traits, the number of predictor variables (genotype combinations) grow exponentially as each SNP is added to the model. Estimation of parameters in such linear models can be unstable and may easily result in overfitting the data. Additionally, linear models generally assume that genetic factors involved in interaction also exhibit independent marginal effects, but this may not be the case in reality (although it is not known how common it is for interaction to be present in the absence of any marginal effects). Nonparametric approaches, especially those involving modern machine-learning approaches, may have their unique advantages in specifically addressing these issues (see below).

Future Directions

The Role of Epistasis in Genetic Susceptibility to Autism

Despite the recent discovery of multiple common and rare genetic variants that confer autism susceptibility with different effect sizes, whether and how these genetic factors interact with each other is currently not known. Furthermore, the form of epistasis involved in autism pathogenesis may include both gene-gene and gene-environment interactions. Many environmental factors, such as air pollution and paternal age, have been linked to autism susceptibility in recent years, but whether these environmental risk factors function independently of genetic risk factors is an unexplored area of research. On the other hand, it is well known that certain environmental factors, including radiation, smoking, and older age, can increase the possibility of de novo genetic alterations, such as de novo copy number variants (CNVs). Given the known involvement of de novo variants in autism, it is highly reasonable to suspect that there may be gene-environment interactions that play a role in increasing genetic susceptibility to autism. This hypothesis needs to be tested in future larger scale genetic studies that also collect various aspects of environmental variables.

Testing Epistasis in Genome-Wide Level

In recent years, a large collection of methods have been developed to detect the presence of epistasis from population-level genetic data. By allowing for epistatic interactions between candidate disease loci, it may be possible to succeed in identifying genetic variants with weak marginal effects that may otherwise evade detection. In addition, some statistical or computational models for modeling genome-wide epistasis (such as support vector machine approaches and penalized regression approaches) can be easily adapted for prediction of phenotypes from genotypes, enabling the implementation of personalized disease risk predictions.

Although the importance of epistasis has been increasingly recognized, one may argue that it has

never really received the attention that it deserves in genetic association studies, especially in GWAS. The major reason is not the lack of methods to test epistasis per se, but the need to adjust the enormous burden of multiple testing, and the lack of appropriate biological interpretation to significant results. In typical GWAS with 500,000 SNP markers, testing genome-wide epistasis implies the need to test for approximately 250 billion hypotheses, even when considering only two-way interactions. The situation becomes worse when three-way or multiway interactions are considered. With the availability of massively parallel computational platform, this may no longer be a computational issue, but how to appropriately calculate the effective number of independent tests and accordingly adjust multiple testing is a problem without consensus on solution.

The Development of Bioinformatic Approaches

Although sophisticated approaches based on various forms of linear models have been used to detect epistasis in genome-wide scale, they may have some intrinsic limitations as discussed above. Therefore, novel approaches from the computational science arena, including machine-learning approaches such as random forests (RFs) and multifactor dimensionality reduction (MDR), have been developed to address these issues. Some of these approaches represent “black-box” modeling strategies: rather than fitting the data into a prespecified statistical model that the investigators believes in, these methods aim to let the data tell us what the appropriate model is. This is a rapidly growing area for methods development, and several recent reviews highlighted the advantage of these methods.

Biological Interpretation of Epistasis

When significant epistasis is found, the next natural question is how to interpret the results and

how to leverage this information to better understand the genetic architecture of complex diseases. Unfortunately, there is no easy answer to these questions. Many factors may blur the seemingly straightforward interpretation of the epistatic effects. For example, many modern genetic association studies use SNP markers, which rely on linkage disequilibrium to detect indirect associations. Since the marker allele most likely may not be the true disease allele, the detected epistatic effects could be due to the artifact of the imperfect tagging of the disease allele. Even worse, if the marker allele, being a common allele, is merely a proxy for multiple rare functional alleles (so-called synthetic association), then it will not be possible to tell which rare allele has epistasis with variants at another loci or whether the observed epistasis merely reflects a “synthetic epistasis” on multiple rare alleles. Therefore, significant statistical results may not always lead to a biologically sound explanation, and it is important not to overinterpret the statistical results.

In summary, direct biological inference from the results of analysis on epistasis can be very difficult. Compared to marginal effects of individual markers in association tests, the biological knowledge gained from investigation of epistasis, even in the presence of strong statistical evidence, can be quite limited. The most effective interpretation of epistasis probably depends on some levels of prior biological knowledge on the genes under investigation, as well as additional molecular evidence, rather than a pure statistical exercise on P values and effect sizes.

See Also

- [Genetics](#)
- [Genome-Wide Association](#)
- [Modifier Genes and Autism Susceptibility](#)

References and Reading

- Bateson, W. (1909). *Mendel's principles of heredity*. Cambridge: Cambridge University Press.
- Carlborg, O., & Haley, C. S. (2004). Epistasis: Too often neglected in complex trait studies? *Nature Reviews Genetics*, 5(8), 618–625.

- Cordell, H. J. (2002). Epistasis: What it means, what it doesn't mean, and statistical methods to detect it in humans. *Human Molecular Genetics*, 11(20), 2463–2468.
- Coutinho, A. M., Sousa, I., Martins, M., Correia, C., Morgadinho, T., Bento, C., Marques, C., Ataíde, A., Miguel, T. S., Moore, J. H., Oliveira, G., & Vicente, A. M. (2007). Evidence for epistasis between SLC6A4 and ITGB3 in autism etiology and in the determination of platelet serotonin levels. *Human Genetics*, 121(2), 243–256.
- Moore, J. H., & Williams, S. M. (2009). Epistasis and its implications for personal genetics. *American Journal of Human Genetics*, 85(3), 309–320.
- Moore, J. H., Asselbergs, F. W., & Williams, S. M. (2010). Bioinformatics challenges for genome-wide association studies. *Bioinformatics*, 26, 445–455.
- Phillips, P. C. (2008). Epistasis—the essential role of gene interactions in the structure and evolution of genetic systems. *Nature Reviews Genetics*, 9(11), 855–867.
- Wolf, J. B., Brodie, E. D., & Wade, M. J. (2000). *Epistasis and the evolutionary process* (1st ed.). New York: Oxford University Press.

Equilibrium

- [Vestibular Function in Children with Autism](#)

Equinus Gait

- [Toe Walking](#)

Equivalence-Based Instruction (EBI)

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Definition

Equivalence-Based Instruction (EBI) refers to procedures based on the behavioral principle of

stimulus equivalence (Sidman 1994). Stimulus equivalence classes are formed when stimuli that share no physical similarities become functionally equivalent to (i.e., substitutable for) each other (Green and Saunders 1998; Sidman 1994; Sidman and Tailby 1982). The term equivalence was borrowed from the field of mathematics (if $A = B$ and $B = C$ then $A = C$) to describe the emergent relational responding emitted by learners that was observed by early researchers.

An equivalence class consisting of three member stimuli (A, B, C), for example, forms when a learner emits the following nine responses of which only three are directly trained (Table 1 and Fig. 1): when presented with sample A (or B or C), the learner selects the identical stimulus (reflexivity or identity matching); after being

trained to select B when presented with A, the learner selects A when presented with B (symmetry); after being trained to select C when presented with A, the learner selects A when presented with C (symmetry); selecting B when presented with C (equivalence) and selecting C when presented with B (equivalence). Three or more classes of at least three stimuli are generally taught simultaneously (e.g., numbers, categories, types, etc.) (Table 2 and Fig. 2). For typically developing learners, these responses tend to emerge without the need for direct training. However, for learners with an Autism Spectrum Disorder (ASD), direct training is often necessary.

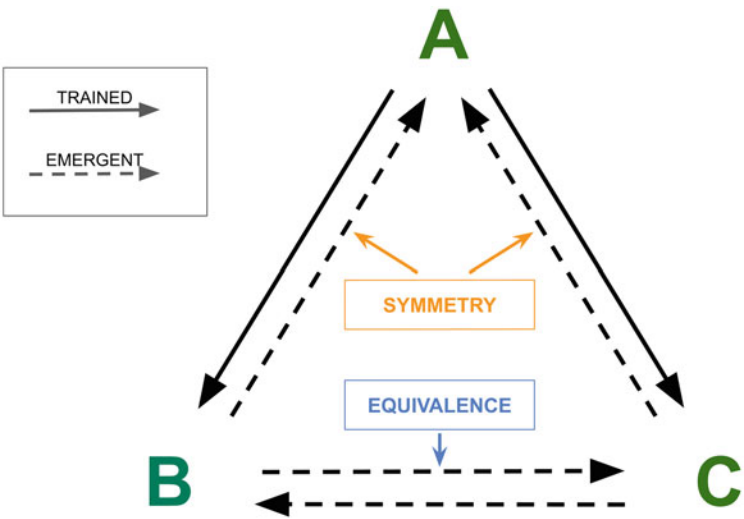
EBI procedures use a variety of specific training methods to teach a small number of skills

E

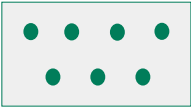
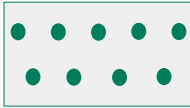
Equivalence-Based Instruction (EBI), Table 1 Alphanumeric labels of stimuli and responses and types of relations in a 3-member (A, B, C) equivalence class

Alphanumeric label	Sample stimulus	Selection response	Name of relation	Trained/tested
A1 = A1	A1	A1	Reflexivity	Trained or tested
B1 = B1	B1	B1	Reflexivity	Trained or tested
C1 = C1	C1	C1	Reflexivity	Trained or tested
A1 = B1	A1	B1	Trained	Trained
A1 = C1	A1	C1	Trained	Trained
B1 = A1	B1	A1	Symmetry	Tested
C1 = A1	C1	A1	Symmetry	Tested
B1 = C1	B1	C1	Equivalence	Tested
C1 = B1	C1	B1	Equivalence	Tested

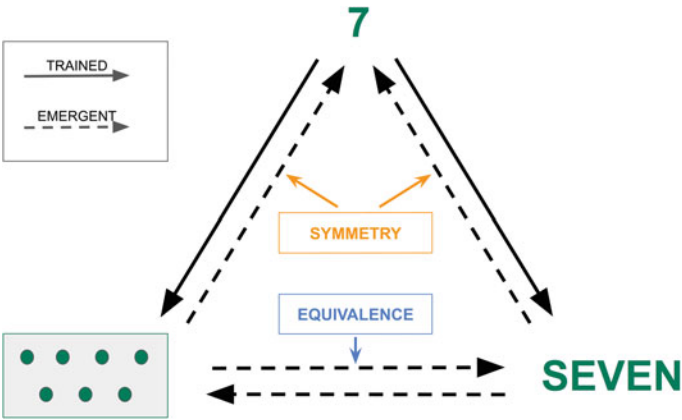
Equivalence-Based Instruction (EBI), Fig. 1 3-member equivalence class diagram



Equivalence-Based Instruction (EBI), Table 2 Example stimulus classes and members in an EBI training program

		Class		
Member		1	2	3
A	Printed numeral	7	8	9
B	Printed quantity			
C	Printed word	Seven	Eight	Nine

Equivalence-Based Instruction (EBI), Fig. 2 Example equivalence class diagram for the number/quantity “seven”



(typically matching or verbal responses) that result in the generation of an even larger number of emergent (i.e., untrained) skills. Training and testing methods may consist of conditional discrimination training (e.g., match-to-sample (MTS)), sorting, categorization, or verbal behavior training). EBI generally uses physical stimuli (e.g., laminated cards, objects, spoken, etc.); however, the instruction may also be delivered via a computer or mobile device.

Unlike stimulus generalization, in which a person’s responses occur in stimulus conditions that are similar to, but not the same as the conditions present during training, stimulus equivalence describes emergent, or derived relational responding. With stimulus generalization, a child might call every adult male “dad” after being taught that his father should be called “dad.” However, with stimulus equivalence, a child might learn that the written numeral “7” is functionally equivalent to a card with seven dots and also to the written word “seven,” and the

relations among all three members become substitutable (Fig. 2). Similar to stimulus generalization, these relations emerge as a result of the direct training of specific discriminations; however, unlike stimulus generalization, a person will emit the same responses when presented with stimuli that share no physical similarities, and this discriminative responding emerges without being directly taught.

Historical Background

Investigations and applications of EBI began in the 1990s and have continued across research sites, populations, settings, skills, and training methodologies (Rehfeldt 2011). EBI procedures are generally based on the principle of stimulus equivalence first identified by Sidman (1994). EBI research has sought to clarify some of the methodological questions that have naturally

arisen as it has been used with an increasing number of learners and skills. In addition, the use of technology (e.g., computer-based training and mobile applications) in the delivery of EBI systems has been an area of interest for researchers and educators.

There is some disagreement in the field of Applied Behavior Analysis (ABA) regarding the term EBI and its use to describe a number of similar instructional procedures; however, EBI is the most commonly used term to describe procedures where the explicit goal of instruction is the emergence of derived equivalence relations. The term has gained sufficient academic and clinical usage that the Behavior Analyst Certification Board (BACB) has included it in the 5th Edition of the Board Certified Behavior Analyst Task List (Behavior Analyst Certification Board 2017) as a required competency for practicing applied behavior analysts.

Rationale or Underlying Theory

There are several theoretical frameworks that attempt to account for the emergent responding seen in EBI. Sidman (1994) proposed that the observed emergent responding was a direct result of the contingencies of reinforcement and that no other behavioral principle was necessary to account for the untrained responding. However, others have argued that this explanation is insufficient to support the formation of equivalence classes. Hayes et al. (2001) argued for a new conceptualization of these emergent responses. The authors suggested that equivalence relations are merely one example of a vast array of derived relational responses (e.g., more, closer, taller, older, etc.). A third explanation for the emergence of derived responding is naming (Horne and Lowe 1996). The authors posit that a form of mediating (usually covert) verbal behavior is in some way responsible for the emergence of equivalence classes. However, for teachers and other practitioners, these different interpretations of the research are somewhat academic and do not significantly affect applications or the development of curricula.

Since the experimental identification of equivalence relations occurred in the 1970s, dozens of applied and translational studies have been conducted and published that replicated and extended the early laboratory observations of the formation of equivalence relations. However, like any established intervention or treatment, further investigation into specific learner characteristics, prompting procedures, training and testing parameters, fluency, generalization, maintenance, and application across settings continues.

Goals and Objectives

When compared to other teaching methods, the main goal of EBI is to teach skills in a more efficient way. With traditional methods, skills are usually taught to mastery, discretely, and then generalization and maintenance of the skills are tested. However, with EBI, a small set of skills is taught and the emergence of several other skills is then tested (including the maintenance and generalization of those emergent skills). For example, where a teacher might have previously used a stimulus fading procedure to establish relations between two stimuli, a teacher might now use EBI to establish an equivalence class. The underlying objective of EBI is thus to reduce the overall time required to teach skills by relying on the emergence of untrained skills.

Treatment Participants

EBI has been shown to be effective across a range of participants with a wide array of ages, functioning levels, and with children with developmental disabilities, specifically Autism Spectrum Disorders (ASDs). Published research has demonstrated the efficacy of EBI procedures for people with ASDs of all ages; however, the bulk of the research has been conducted with children. Research has been conducted on basic principles, protocols, and factors affecting mastery and efficacy of the procedures in well-controlled experiments using arbitrary stimuli in addition to applied skills and in clinical settings. Particularly, EBI has been shown to be effective in teaching

children with an ASD more efficiently, and EBI results in generative responding unlike with more traditional approaches to instruction (McLay et al. 2013).

Treatment Procedures

EBI is a general term that is used to refer to a number of component training and testing protocols, procedures, and structures. The most common method of training and testing is conditional discrimination training, specifically MTS. Other methods of training and testing have been shown to be effective including sorting, categorization, and verbal behavior (Jennings and Miguel 2017; Ma et al. 2016; Miguel et al. 2008). However, these procedures tend to be more difficult to prepare and implement in applied settings.

During MTS training, a sample stimulus and an array of comparison stimuli are presented and a selection response is prompted and reinforced, and during testing the response is generally not prompted or reinforced. Prior to training, pretests (i.e., baseline tests) are conducted to determine the learner's prior history with the stimuli. Then, certain discriminations (e.g., AB and AC) are trained and tests are conducted after training to determine if the derived responses (BA, CA, BC, CB) emerged. If a learner fails a test, remedial training for the trained relations is typically conducted. Posttests are then conducted to determine if an equivalence class has formed (i.e., all stimuli have become functionally equivalent).

Every sensory modality can be used with EBI training. Stimulus forms can include auditory (e.g., spoken), visual (e.g., pictures or written text), and tactile (e.g., physical objects). Similarly, several response forms can be trained including selection-based (e.g., selecting from an array of comparison stimuli, multiple choice questions) and topographical (e.g., vocal product, written, typed, drawn, etc.). It is recommended to use auditory stimuli as samples as it is impossible to simultaneously present multiple auditory stimuli as comparisons.

Green (2001) and MacDonald and Langer (2018) listed a number of best practices regarding conditional discrimination training and these

practices should be followed for MTS in EBI as well. The suggested methodological guidelines include:

- Fast-paced trial presentation
- Immediate reinforcement.
- Present a different sample stimulus every trial
- Have at least three comparison stimuli in an array
- Counterbalance the presentation order and positions of sample and comparison stimuli
- Require an observing response
- Auditory stimuli should be used as sample stimuli
- Prepare trials out of view of the learner
- Teach basic learner and readiness skills prior to using MTS
- Use errorless teaching or prompt-fading methods

Basic and applied research has also established best practices regarding EBI specifically. These include using a simple-to-complex training protocol and a one-to-many (OTM) training structure (Arntzen 2012; Saunders and Green 1999). A simple-to-complex training protocol describes a procedure where the AB and AC discriminations are taught followed by testing for the emergent relations. An OTM training structure is the term used to describe the method of specifically training the AB and AC discriminations as opposed to other combinations (e.g., AB then BC or AC then BC). Given that training and testing is usually selection-based, it is also recommended to test for the emergence of topographical responding (e.g., vocal tact, written response, etc.) where appropriate. And like all other behavior-analytic interventions, it is important to assess generalization (stimuli and settings) and maintenance over time.

Efficacy Information

EBI has been shown to be effective for a variety of skills in numerous settings with hundreds of learners in controlled experiments. In addition, dozens of published articles have established the

efficacy of EBI across a range of learners, ages, settings, ability levels, diagnoses, training procedures, stimulus parameters, and response topographies (Dixon et al. 2018; McLay et al. 2013; McKeel et al. 2015; Rehfeldt 2011). Given several published standards of the criteria for the identification of an evidence-based practice EBP (National Autism Center 2015; Wong et al. 2015), and given that EBI procedures are comprised of component instructional methods (e.g., discrimination training) that are well established as EBPs, EBI would be considered to be an EBP that can be used for the development of individualized curriculum for learners with ASDs.

Outcome Measurement

The defining feature of EBI is the establishment of emergent relational responding. This skill is easily observed and data can be collected in a variety of ways, including direct observation and electronically. In addition, procedural integrity assessments can be conducted, and interobserver agreement (IOA) and reliability data can be easily collected to ensure the validity of EBI procedures and the accuracy of accuracy and reliability of data collected from direct observations. Standard MTS data sheets may be used and data is commonly presented as a percentage of correct responding per sessions (either training or testing). The mastery criteria for the establishment of an equivalence class are generally between 90% and 100% correct for all trained and tested relations. Remedial training may be necessary in some cases. In addition to the establishment of equivalence classes comprised of stimuli used during training, generalization to novel stimuli that are also substitutable can be tested. Finally, test probes can be conducted periodically to determine if the skills maintain over time.

Qualifications of Treatment Providers

The underlying teaching protocols for most EBI programs are based on conditional discrimination training and MTS procedures. Most practicing

Board Certified Behavior Analysts (BCBAs) and/or Licensed Behavior Analysts receive academic and experiential training on conditional discrimination preparations and curriculum design. However, not all practicing BCBAs routinely use systematic and structured conditional discrimination training. Therefore, it is recommended that in addition to a BCBA or a License, that practitioners who wish to use EBI procedures gain some postcertification/licensure training (e.g., conference workshops, coursework, mentoring, etc.) before implementing EBI with their clients. That being said, the BACB's Task List - 5th Edition (Behavior Analyst Certification Board 2017), which will be the standard in 2022, includes EBI as a required skill that practicing BCBAs should be fluent with. Therefore, over the coming years, all graduate programs will include coursework specifically on EBI, and many supervised experiences will include direct training of EBI procedures. In addition, given the right amount of training, supervision, and support, teachers, parents, paraprofessionals, and caretakers should be able to implement basic protocols based on EBI.

See Also

- Academic Skills
- Applied Behavior Analysis (ABA)
- Early Intensive Behavioral Intervention (EIBI)
- Generalization and Maintenance
- Operant Conditioning
- Prompt Fading
- Reinforcement
- Stimulus
- Trial
- Verbal Behavior

References and Reading

- Arntzen, E. (2012). Training and testing parameters in formation of stimulus equivalence: Methodological issues. *European Journal of Behavior Analysis*, 13(1), 123–135. <https://doi.org/10.1080/15021149.2012.11434412>.
- Behavior Analyst Certification Board. (2017). *BCBA/BCaBA task list* (5th ed.). Littleton: Author.

- Dixon, M. R., Wiggins, S. H., & Belisle, J. (2018). The effectiveness of the peak relational training system and corresponding changes on the VB-MAPP for young adults with autism. *Journal of Applied Behavior Analysis*, 51(2), 321–334.
- Green, G. (2001). Behavior analytic instruction for learners with autism: Advances in stimulus control technology. *Focus on Autism and Other Developmental Disabilities*, 16(2), 72–85. <https://doi.org/10.1177/108835760101600203>.
- Green, G., & Saunders, R. R. (1998). Stimulus equivalence. In K. A. Lattal & M. Perone (Eds.), *Handbook of research methods in human operant behavior* (pp. 229–262). New York: Plenum Press.
- Hayes, S. C., Barnes-Holmes, D., & Roche, B. (2001). *Relational frame theory: A post-Skinnerian account of human language and cognition*. New York: Springer Science & Business Media.
- Horne, P. J., & Lowe, C. F. (1996). On the origins of naming and other symbolic behavior. *Journal of the Experimental Analysis of Behavior*, 65(1), 185–241.
- Jennings, A. M., & Miguel, C. F. (2017). Training intraverbal bidirectional naming to establish generalized equivalence class performances. *Journal of the Experimental Analysis of Behavior*, 108(2), 269–289.
- Ma, M. L., Miguel, C. F., & Jennings, A. M. (2016). Training intraverbal naming to establish equivalence class performances. *Journal of the Experimental Analysis of Behavior*, 105(3), 409–426.
- MacDonald, R., & Langer, S. (2018). *Teaching essential discrimination skills to children with autism*. Bethesda: Woodbine House.
- McKeel, A. N., Dixon, M. R., Daar, J. H., Rowsey, K. E., & Szekely, S. (2015). Evaluating the efficacy of the PEAK relational training system using a randomized controlled trial of children with autism. *Journal of Behavioral Education*, 24(2), 230–241.
- McLay, L. K., Sutherland, D., Church, J., & Tyler-Merrick, G. (2013). The formation of equivalence classes in individuals with autism spectrum disorder: A review of the literature. *Research in Autism Spectrum Disorders*, 7(2), 418–431.
- Miguel, C. F., Petursdottir, A. I., Carr, J. E., & Michael, J. (2008). The role of naming in stimulus categorization by preschool children. *Journal of the Experimental Analysis of Behavior*, 89(3), 383–405.
- National Autism Center. (2015). *Findings and conclusions: National standards project, phase 2*. Randolph: Author.
- Rehfeldt, R. A. (2011). Toward a technology of derived stimulus relations: An analysis of articles published in the journal of applied behavior analysis, 1992–2009. *Journal of Applied Behavior Analysis*, 44(1), 109–119.
- Saunders, R. R., & Green, G. (1999). A discrimination analysis of training-structure effects on stimulus equivalence outcomes. *Journal of the Experimental Analysis of Behavior*, 72(1), 117–137. <https://doi.org/10.1901/jeab.1999.72-117>.
- Sidman, M. (1971). Reading and auditory-visual equivalences. *Journal of Speech, Language, and Hearing Research*, 14(1), 5–13.
- Sidman, M. (1994). *Equivalence relations and behavior: A research story*. Boston: Authors Cooperative.
- Sidman, M., & Tailby, W. (1982). Conditional discrimination vs. matching to sample: An expansion of the testing paradigm. *Journal of the Experimental Analysis of Behavior*, 37(1), 5–22.
- Wong, C., Odom, S. L., Hume, K. A., Cox, A. W., Fettig, A., Kucharczyk, S., ... & Schultz, T. R. (2015). Evidence-based practices for children, youth, and young adults with autism spectrum disorder: A comprehensive review. *Journal of Autism and Developmental Disorders*, 45(7), 1951–1966.

ER

► Emotional Regulation

ERN

► Error-Related Negativity

Error Correction

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Definition

In the applied autism literature, error correction is a procedure that details what a trainer or program implementer does when the learner engages in an incorrect response during a teaching opportunity. Error correction aims to enhance learning by teaching the learner the appropriate response and increasing the learner's contact with reinforcement contingencies rather than simply extinguishing errors. This procedure is intended

to help learners acquire skills much faster and with less frustration than simply allowing trial and error. In other words, it teaches the learner what *to do* instead of just allowing them to make mistakes and try to determine the correct response on their own.

There are three types of procedures for error correction. All three types are presented after the learner engages in a defined incorrect response (including no response within a specific amount of time) and are combined with a differential reinforcement procedure. Each of the three is defined independently below:

1. A procedure consisting of a series of response prompts following a learner's error to increase the probability of the learner immediately engaging in the target response.
2. A series of repetitions of the target response contingent on a learner's error, typically combined with a differential reinforcement procedure (Worsdell et al. 2005).
3. Manipulation of the stimulus or addition of a stimulus contingent on the learner engaging in an error, typically combined with a differential reinforcement procedure (Catania 2007).

The type of error correction in teaching approaches can vary substantially. For instance, some approaches use an “errorless” approach, in which they provide the correction (in the form of prompts) before or as soon as the incorrect response occurs. Other approaches utilize the “no-no prompting” correction in which they gradually increase the amount of error correction. Often these are chosen and tailored on the basis of the learner's skill level and learning goals.

See Also

- [Differential Reinforcement](#)
- [Hand-Over-Hand Assistance](#)
- [Modeling](#)
- [Prompt Hierarchy](#)
- [Prompt System](#)
- [Prompting](#)

References and Reading

- Catania, C. (2007). *Learning*. New York: Sloan.
- Gena, A., Couloura, S., & Kymissis, E. (2005). Modifying the perspectives of preschoolers with autism using in-vivo or video modeling and reinforcement contingencies. *Journal of Autism and Developmental Disorders*, 35, 545–556.
- Rodgers, T. A., & Iwata, B. A. (1991). An analysis of error-correction procedures during discrimination training. *Journal of Applied Behavior Analysis*, 24, 775–781.

Error of Measurement

- [Measurement Error](#)

Error-Related Negativity

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Synonyms

[ERN](#)

Definition

The error-related negativity, or ERN, is an electrical brain signal measured with an electroencephalogram. Detectable at the scalp via the event-related potential (ERP), the ERN occurs when an individual makes a behavioral error. The ERN is typically evoked with simple cognitive tasks when an individual responds incorrectly or responds when a response should be withheld. The ERN manifests as a negative deflection in the ERP at approximately 80–150 ms following error commission, time-locked to an individual's response. The ERN is largest at central to frontal-central scalp regions. The most likely neural generator of the ERN is the anterior cingulate cortex, with converging evidence coming from

fMRI (Ito et al. 2003), EEG source modeling (Luu et al. 2003), and brain lesion research (Stemmer et al. 2004).

See Also

- Anterior Cingulate
- Cingulate Cortex
- Feedback-Related Negativity

References and Reading

- Ito, S., Stuphorn, V., Brown, J. W., & Schall, J. D. (2003). Performance monitoring by the anterior cingulate cortex during saccade countermanding. *Science*, 302(5642), 120–122.
- Luu, P., Tucker, D. M., Derryberry, D., Reed, M., & Poulsen, C. (2003). Electrophysiological responses to errors and feedback in the process of action regulation. *Psychological Science*, 14, 47–53.
- Stemmer, B., Segalowitz, S. J., Witzke, W., & Schönle, P. W. (2004). Error detection in patients with lesions to the medial prefrontal cortex: An ERP study. *Neuropsychologia*, 42(1), 118–130.

Erythrocyte Glutathione Peroxidase

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Synonyms

GSH-Px1 and Glutathione hydrogen-peroxide oxido-reductase

Definition

Free radicals remain an integral component of numerous physiological and pathophysiological activities within cellular systems. Free radicals result from electronic excitations leading to the creation of unevenly paired electrons which

react with biomolecules in three primary stages: initiation, propagation, and termination. In initiation, free radicals are produced through high-energy radiation, hemolytic cleavage, or the addition of excited electrons. Through propagation, free radicals produce additional free radicals through a process reminiscent of nuclear chain reactions. In the termination step, the propagation of free radicals may be eliminated when free radical species react with one another or are scavenged by antioxidant molecules. In biological systems, reactive oxygen species (ROS), such as $\bullet\text{OH}$ and $\text{O}_2\bullet^-$ are the most prominent free radical species produced from environmental exposure or cellular respiration (Devasagayam et al. 2004). Despite their destructive potential, ROS modulate numerous physiological activities, including the immune response, enzymatic activity, cellular signaling, and apoptosis (Devasagayam et al. 2004). Thus, the production of ROS is a highly regulated process requiring antioxidants to prevent, intercept, and repair free radical damage to lipids, proteins, and DNA (Devasagayam et al. 2004).

Free radical damage has been linked oxidative stress to numerous psychiatric conditions, including schizophrenia, major depressive disorder, anxiety, and obsessive-compulsive disorders (OCD) (Chauhan and Chauhan 2006). Increasing evidence suggests reduced antioxidant capacity from prolonged oxidative stress may underly ASD neuro-cognitive abnormalities (Parellada et al. 2012). Furthermore, proper neuronal differentiation and development requires a delicate balance and coordination between antioxidant and pro-oxidant cellular process (Fantel and Person 2002). As a result, researchers have investigated the correlation between ASD and several biomarkers for antioxidant status. In this regard, erythrocyte glutathione peroxidase (GSH-Px1) has shown significant promise (Kondolot et al. 2016; László et al. 2013; Paşca et al. 2006; Söğüt et al. 2003; Yorbik et al. 2002).

GSH-Px1 is an essential antioxidant which eliminates hydrogen peroxide to water using glutathione (GSH) and reduced NADPH as cofactors (Devasagayam et al. 2004). In a recent study, a negative correlation between homocysteine levels

and erythrocyte glutathione peroxidase activity was discovered among ASD patients (Paşca et al. 2006). The authors argued this trend matched previous studies showing elevations in homocysteine down-regulates GSH-Px1 and thus contributes to the pathogenesis of ASD (Paşca et al. 2006). In addition, several clinical studies showed a significant decrease in erythrocyte GSH-Px1 in ASD patients (Kondolot et al. 2016; László et al. 2013; Parellada et al. 2012; Yorbik et al. 2002). However, one clinical study showed an increase in erythrocyte GSH-Px1 activity resulting from previous cellular oxidative stress or compensatory mechanisms to decreased antioxidant defenses (Söğüt et al. 2003). Overall, ASD patients showed a consistent decrease in antioxidant status and free radical scavenging. Given these results, further investigation into antioxidant biomarkers may provide additional insight in the pathogenesis and treatment of ASD.

See Also

- Metallothionein
- Thioridazine
- Thiothixene

References and Reading

- Chauhan, A., & Chauhan, V. (2006). Oxidative stress in autism. *Pathophysiology*, 13(3), 171–181. <https://doi.org/10.1016/j.pathophys.2006.05.007>.
- Devasagayam, T., Tilak, J., Bloor, K., Sane, K. S., Ghaskadbi, S. S., & Lele, R. (2004). Free radicals and antioxidants in human health: Current status and future prospects. *The Journal of the Association of Physicians of India*, 52, 794–804.
- Fantel, A. G., & Person, R. E. (2002). Involvement of mitochondria and other free radical sources in normal and abnormal fetal development. *Annals of the New York Academy of Sciences*, 959(1), 424–433. <https://doi.org/10.1111/j.1749-6632.2002.tb02112.x>.
- Kondolot, M., Ozmert, E. N., Asci, A., Erkekoglu, P., Oztop, D. B., Gumus, H., ... & Yurdakok, K. (2016). Plasma phthalate and bisphenol a levels and oxidant-antioxidant status in autistic children. *Environmental Toxicology and Pharmacology*, 43, 149–158. <https://doi.org/10.1016/j.etap.2016.03.006>.
- László, A., Novák, Z., Szöllösi-Varga, I., du, Q. H., Vetró, Á., & Kovács, A. (2013). Investigation of antioxidant enzymes in children with autistic disorder. *Ideggyógyászati Szemle*, 30(66), 23–28.
- Parellada, M., Moreno, C., Mac-Dowell, K., Leza, J. C., Giraldez, M., Bailón, C., ... & Arango, C. (2012). Plasma antioxidant capacity is reduced in Asperger syndrome. *Journal of Psychiatric Research*, 46(3), 394–401. <https://doi.org/10.1016/j.jpsychires.2011.10.004>.
- Paşca, S. P., Nemeş, B., Vlase, L., Gagyi, C. E., Dronca, E., Miu, A. C., & Dronca, M. (2006). High levels of homocysteine and low serum paraoxonase 1 arylesterase activity in children with autism. *Life Sciences*, 78(19), 2244–2248. <https://doi.org/10.1016/j.lfs.2005.09.040>.
- Söğüt, S., Zoroğlu, S. S., Özyurt, H., Ramazan Yılmaz, H., Özüğurlu, F., Sivaslı, E., ... & Akyol, Ö. (2003). Changes in nitric oxide levels and antioxidant enzyme activities may have a role in the pathophysiological mechanisms involved in autism. *Clinica Chimica Acta*, 331(1–2), 111–117. [https://doi.org/10.1016/s0009-8981\(03\)00119-0](https://doi.org/10.1016/s0009-8981(03)00119-0).
- Yorbik, O., Sayal, A., Akay, C., Akbiyik, D. I., & Sohmen, T. (2002). Investigation of antioxidant enzymes in children with autistic disorder. *Prostaglandins, Leukotrienes and Essential Fatty Acids*, 67(5), 341–343. <https://doi.org/10.1054/plef.2002.0439>.

Escape Extinction

- Escape Training

Escape Training

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Synonyms

Escape extinction

Definition

Escape training/extinction is a behavioral procedure that is generally used to treat escape or avoidance maintained behaviors. Utilization of

escape extinction procedures includes discontinuing the escape contingency upon the occurrence of the behavior. That is, when a behavior that is reinforced by negative reinforcement (removal of a stimulus contingent on a response that leads to an increase in that behavior), preventing escape contingent on that behavior will lead to a reduction in that behavior. Escape extinction has been demonstrated as being effective alone and as part of a treatment package for a variety of escape maintained behaviors. One area of emphasis in the research literature where escape extinction has been widely used is feeding interventions for individuals with selective feeding or food refusal. Escape extinction in the form of non-removal of the spoon (i.e., presenting the bolus of food within close proximity to the mouth of the participant until the food is accepted) is one example of escape extinction. Extinction procedures have several potential side effects including the extinction burst – an immediate increase in the frequency or intensity of behavior following the discontinuation of reinforcement – extinction-induced aggression—an escalation in aggressive behavior and spontaneous recovery, that is, a return to higher levels of the target behavior after a behavior has been reduced to low levels. In order to address the potential side effects of extinction, use of reinforcement-based procedures (e.g., reinforcement of food acceptance in the above example) is strongly recommended. Behaviors can demonstrate resistance to extinction, meaning that the behavior will persist even when the reinforcement for that behavior has been removed. There are several factors that may contribute to this resistance including the level of motivation for the reinforcer (i.e., the higher the value of the reinforcer, the more resistant it is to extinction), the schedule of reinforcement, the alternative sources of reinforcement for the same behavior, the amount of effort required to emit the behavior (i.e., the higher the effort the less resistant to extinction), and whether the behavior has been under extinction before with rapid decreases with each application of extinction. Use of extinction is not recommended when the individual engages in high-intensity behavior such as dangerous aggressive and self-injurious behavior as

an increase in the intensity of behavior would lead to risk of injury to the individual or others.

See Also

- [Differential Reinforcement](#)
- [Negative Reinforcement](#)

References and Reading

- Ahearn, W. H., Kerwin, M. L., Shantz, J., & Swearingin, W. (1996). An alternating treatments comparison of two intensive interventions for food refusal. *Journal of Applied Behavior Analysis*, 29, 321–332.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson Education.
- Lalli, J. S., Casey, S., Goh, H., & Merlino, J. (1994). Treatment of escape-maintained aberrant behavior with escape extinction and predictable routines. *Journal of Applied Behavior Analysis*, 27, 705–714.
- Piazza, C. C., Patel, M. R., Gulotta, C. S., Sevin, B. M., & Layer, S. A. (2003). On the relative contributions of positive reinforcement and escape extinction in the treatment of food refusal. *Journal of Applied Behavior Analysis*, 36(3), 309–324.
- Zarcone, J. R., Iwata, B. A., Hughes, C. E., & Vollmer, T. R. (1993). Momentum versus extinction effects in the treatment of self-injurious escape behavior. *Journal of Applied Behavior Analysis*, 26, 135–136.

Escitalopram

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Synonyms

Brand names: [Anxiset E \(India\)](#); [Cipralext \(Canada\)](#); [Lexam](#); [Lexamil](#); [Lexapro](#); [Seroplex](#)

Indications

According to the official prescribing information, escitalopram is FDA-approved to treat major depressive disorder (MDD) in adults and in adolescents aged 12–17 years old, both acutely and as maintenance therapy (Forest Pharmaceuticals 2011). It is also approved for the acute treatment of generalized anxiety disorder (GAD) in adults. It is not approved for children under the age of 12 years.

Escitalopram is not approved for the treatment of autism spectrum disorders (ASDs). ASDs include the DSM-IV-TR diagnoses of autistic disorder, Asperger's disorder, and pervasive developmental disorder, not otherwise specified (PDD-NOS). The decision to use escitalopram in the treatment of established clinical indications that may co-occur with ASDs, as mentioned above, or for commonly observed symptoms of ASDs, such as hyperactivity, inattention, irritability, aggression, repetitive behaviors, and social impairment, can be made on an individual basis by the treating practitioner. This will be discussed in more detail in the “Clinical Uses” section.

Mechanisms of Action

Escitalopram is a selective serotonin reuptake inhibitor (SSRI). Normally, serotonin is released from the presynaptic axon terminal to travel across the synaptic cleft and attach to the postsynaptic axon terminal. Any excess serotonin remaining in the cleft is picked up by the serotonin reuptake transporter in the presynaptic axon to be degraded and recycled. It is hypothesized that a depletion of serotonin available to act on the postsynaptic axon results in symptoms experienced in some mood and anxiety disorders, including sadness, nervousness, guilt, loneliness, apathy, and avolition. Escitalopram attaches itself to the serotonin reuptake transporter and prevents whatever serotonin remains in the cleft from being taken back up in the presynaptic neuron. The excess serotonin is free to attach to postsynaptic receptors and exert its intended effects.

Escitalopram begins acting immediately to inhibit serotonin reuptake, but like other SSRIs, its antidepressant and anxiolytic effects are often not experienced for up to 6–8 weeks. This is thought to be due to gradual changes SSRIs make on serotonin receptor sensitivity. It is believed that the decreased amounts of serotonin found in mood and anxiety disorders cause the postsynaptic axon to upregulate the number of postsynaptic axonal receptors expressed. In other words, a decreased amount of serotonin in the synaptic cleft results in an increased amount of receptors at the ready. When escitalopram enters the picture, the level of serotonin in the cleft rises. Postsynaptic receptors respond to the serotonin increase and send messages to the axon nucleus about the changes in the synaptic cleft. The nucleus begins downregulating the amount of serotonin receptors ready to receive serotonin. These changes are gradual and are believed to take 6–8 weeks before being fully completed, reflecting the amount of time before many patients begin feeling relief from their symptoms.

Like other SSRIs, escitalopram's antagonism of the postsynaptic 5-HT_{2C} receptor results in some of its unique therapeutic effects. When serotonin attaches to the 5-HT_{2C} receptor, it blocks the release of neurotransmitters norepinephrine and dopamine in the brain. When escitalopram blocks the postsynaptic 5-HT_{2C} receptor, norepinephrine and dopamine are instead released and exert their effect in the prefrontal cortex. The effect is activating, often leading patients to feel more energized and less fatigued, with improved concentration and attention. Escitalopram's effects can also be described as anxiolytic, and it is currently FDA-approved for the treatment of GAD. Escitalopram is often considered the “quintessential SSRI” as it only contains the S enantiomer, unlike the related compound citalopram (Celexa), whose racemic makeup includes both R and S enantiomers. (Enantiomers are a pair of chemical structures that exist as mirror images and subsequently exert different effects.) Escitalopram's lone S enantiomer yields a “purer” serotonin reuptake inhibition response and removes some side effects that exist in the racemic (mixed) compound. This unique property allows for escitalopram to be more

efficacious at lower doses given the absence of a potentially interfering R enantiomer. The lack of CYP450 enzyme interactions makes escitalopram one of the best tolerated of the SSRIs, although it is not yet generic and therefore can be expensive.

Specific Compounds and Properties

(S)-1-[3-(dimethylamino)propyl]-1-(4-fluorophenyl)-1,3-dihydroisobenzofuran-5-carbonitrile.

Clinical Use (Including Side Effects)

FDA-Approved Clinical Uses

In adults, escitalopram is FDA-approved to treat MDD, both acutely and as maintenance therapy, and for the acute treatment of GAD (Forest Pharmaceuticals 2011). Per the official prescribing information, escitalopram is typically started at 10 mg once daily for the treatment of MDD with a recommended maximum of 20 mg daily. If the dose is increased to 20 mg once daily, it should occur after a minimum of 1 week. For GAD in adults, the recommended starting and target dose is 10 mg once daily. The goal of treatment is complete remission of symptoms and prevention of future relapses, so escitalopram is taken both throughout and in between clinical relapses (Stahl 2009). Although escitalopram can eliminate symptoms while being taken, it does not cure MDD or GAD, and symptoms can reoccur after the medicine has been stopped. If escitalopram is to be discontinued, a gradual dose reduction is recommended (Forest Pharmaceuticals 2011). After the first episode of depression in the treatment of adults with MDD, escitalopram should be taken for 1 year following symptom relief (Stahl 2009). After the second or any subsequent episodes of depression, treatment with escitalopram may be indefinite to avoid relapse of symptoms. Treatment of GAD may also require indefinite treatment.

Regarding the pediatric population, escitalopram is FDA-approved for the treatment of MDD in adolescents aged 12–17 years (Forest

Pharmaceuticals 2011). There are no clinically approved uses of escitalopram for children under the age of 12 years. In the treatment of adolescent MDD, escitalopram is typically started at 10 mg once daily with a recommended maximum of 20 mg daily (Forest Pharmaceuticals 2011). Per the official prescribing information, if the dose is increased to 20 mg once daily, it should occur after a minimum of 3 weeks.

Clinical Uses of Escitalopram in Autism Spectrum Disorders

Regarding pediatric populations, there is minimal evidence to suggest the effectiveness of escitalopram in children and adolescents with autism. Interestingly, antidepressants have been the most commonly prescribed psychotropic medications in treating symptoms associated with ASDs (Aman et al. 2005). Given the frequent use of SSRIs in autism, it is imperative that prescribing clinicians balance the benefit of such medications in lieu of their side effects. In 2004, the FDA released safety warnings about the increased risk of suicide-related behaviors in children and adolescents taking SSRIs, which subsequently curtailed the prescribing of SSRIs to this population (Nemeroff et al. 2007). The decision to use escitalopram in the treatment of established clinical indications that may co-occur with autism should therefore be made on an individual basis by the treating practitioner after careful consideration of the available data.

There are currently no published randomized, placebo-controlled trials examining the use of escitalopram in treating symptoms associated with ASDs. Therefore, minimal conclusions can be drawn about the efficacy of escitalopram in treating both adults and children with ASDs. Among the available research, a study by Owley et al. (2005) attempted to shed light on this subject. This prospective, open-label study included 28 children (25 males, 3 females) diagnosed with an ASD (71% with autistic disorder) between the ages of 6 and 17 years. They received escitalopram over the course of 10 weeks, which was increased to a maximum dose of 20 mg/day as tolerated. This study concluded that escitalopram

was “useful in treating some difficulties associated with pervasive developmental disorders (PDDs)” as evidenced by a 61% response rate, improvement in the areas of irritability (the most improvement), hyperactivity, lethargy, stereotypy, and inappropriate speech, and “significant improvement” via the Clinical Global Impressions scale. Again, a prescriber should be cautious in generalizing these results to the larger population of patients with ASDs given the study’s design and small sample size which limit its interpretation.

Side Effects

Escitalopram is often considered the most tolerated of the SSRIs due to its simplified chemical structure, which potentially results in “purer” inhibition of the serotonin transporter and fewer cytochrome P450 enzyme-mediated drug interactions (Stahl 2008). Stimulation of serotonin receptor subtypes (5-HT_{2A}, 5-HT_{2C}, 5-HT₃, and 5-HT₄) in various parts of the brain likely causes many of the SSRIs’ observed side effects. A small amount of increased synaptic serotonin shortly after initiating therapy is often enough to cause some side effects, even if the clinical benefit is not yet apparent to the patient. Therefore, it is possible that side effects may be experienced earlier than symptom relief when first starting treatment with escitalopram (Stahl 2008). Side effects experienced may also be dose dependent (i.e., they increase as the dose increases) or time dependent (i.e., they start right after taking the medication but diminish with time) (Stahl 2009).

Patients who are treated for MDD are at increased risk for experiencing suicidal thinking and behavior. Antidepressants as a class have been shown to increase the risk of suicidal thinking and behavior in children, adolescents, and young adults (ages 18–24) with MDD and other psychiatric disorders (Forest Pharmaceuticals 2011). Such risk should be carefully considered when prescribing escitalopram in the pediatric and young adult population. Escitalopram is currently not approved for use in children younger than age 12 years. According to Stahl (2009),

gastrointestinal side effects are common and can include decreased appetite, nausea, diarrhea, constipation, or dry mouth. Sexual dysfunction is a common side effect in both males and females and is due to the effect of increased serotonin in both the brain and the region of the spinal cord regulating sexual response. In males, this includes delayed ejaculation, erectile dysfunction, and decreased sexual desire. In women, this includes decreased sexual desire and anorgasmia. Central nervous system side effects include insomnia or sedation, agitation, tremors, headache, and dizziness or lightheadedness. Patients with undiagnosed bipolar or psychotic disorders may be vulnerable to the activating properties of escitalopram, resulting in hypomania, mania, or psychosis. Patients may also experience vasodilation, dry mouth, diaphoresis, or abnormal vision. Increased serotonin can lead to diminished dopamine release, which may lead to emotional flattening, apathy, and cognitive slowing in some patients. Escitalopram has not been shown to result in clinically important body weight changes (Forest Pharmaceuticals 2011).

Of note, SSRIs have been found to yield side effects in people with ASDs, particularly agitation, hyperactivity, aggression, and insomnia (Posey et al. 2006). Extrapyramidal symptoms (Sokolski et al. 2004) and hypomania (Damore et al. 1998) have also been observed in case reports. These results are limited by a lack of placebo-controlled studies, although there is some evidence to suggest that children with ASDs may be more likely to develop such side effects relative to adults with ASDs (Posey et al. 2006).

See Also

- Antidepressant Medications
- Anxiolytic Drugs
- Anxiolytics
- Citalopram
- Depressive Disorder
- Dopamine
- Generalized Anxiety Disorder
- Norepinephrine

- Repetitive Behavior
- Serotonin
- Serotonin Reuptake Inhibitors (SRIs)
- Stereotypic Behavior

References and Reading

- Aman, M. G., Lam, K. S., et al. (2005). Medication patterns in patients with autism: Temporal, regional, and demographic influences. *Journal of Child and Adolescent Psychopharmacology*, 15(1), 116–126.
- Damore, J., Stine, J., et al. (1998). Medication-induced hypomania in Asperger's disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 37(3), 248–249.
- Forest Pharmaceuticals prescribing information. (2011). Retrieved from http://www.frx.com/pi/lexapro_pi.pdf.
- Nemeroff, C. B., Kalali, A., et al. (2007). Impact of publicity concerning pediatric suicidality data on physician practice patterns in the United States. *Archives of General Psychiatry*, 64(4), 466–472.
- Owley, T., Walton, L., et al. (2005). An open-label trial of escitalopram in pervasive developmental disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44(4), 343–348.
- Posey, D. J., Erickson, C. A., et al. (2006). The use of selective serotonin reuptake inhibitors in autism and related disorders. *Journal of Child and Adolescent Psychopharmacology*, 16(1–2), 181–186.
- Sokolski, K. N., Chicz-Demet, A., et al. (2004). Selective serotonin reuptake inhibitor-related extrapyramidal symptoms in autistic children: A case series. *Journal of Child and Adolescent Psychopharmacology*, 14(1), 143–147.
- Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd ed., pp. 522–539). New York: Cambridge University Press.
- Stahl, S. M. (2009). *Stahl's essential psychopharmacology: The prescriber's guide* (3rd ed., pp. 171–179). New Delhi: Cambridge University Press.

ESCS

- Early Social-Communication Scales (ESCS)

ESP

- Evaluation of Sensory Processing

Establishing Operations

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Definition

Establishing operations (EOs) are “events that alter the value of a reinforcer” (Michael 1982). EOs are sometimes called motivating operations (see MOs) (Vargas 2009). EOs can be conditioned or unconditioned. They may exist in many forms and often are reliant on states of deprivation; however, it is important to note that just as deprivation is experienced throughout the day, aversive stimulation is also common in our environment. Vargas (2009) noted, “Aversive situations are establishing operations for avoidance/escape behaviors.” Another form of establishing operation is known as “value altering.” In this situation, a stimulus or set of stimuli function to make the reinforcer more reinforcing. For example, effective advertising increases the desire to have the product being advertised; advertising is not reliant on a state of deprivation, and in a sense, it may create an aversive situation if everyone else has the item that is advertised and the person who does not have the item stands out because they are “different.” Key components of EOs are that an establishing operation “precedes the response it is functionally related to and it increases the effectiveness of a particular stimulus change as reinforcement” (Peterson 1978). In sum, an establishing operation determines what a person wants at any given time, and it is dynamic in nature because establishing operations is always changing (Cooper et al. 2007).

Historical Background

Historically, Skinner (1953) introduced the concept of EOs in his first publications when he wrote about a “third variable”; however, it was Keller and Schoenfeld (1950) who first coined the

term “establishing operations” (Vargas 2009). Establishing operations have been discussed extensively in the literature on verbal behavior and are independent variables that have been present in the study of applied behavior analysis since its inception. Experimental analysis of behavior and applied behavior analysis have both been concerned with complex ways of arranging reinforcers to change behavior (Bailey and Burch 2002).

Rationale or Underlying Theory

As stated earlier, EOs change the value of a reinforcer. In a classroom situation, this is especially important as we want learners to acquire new skills. Ensuring specific states of deprivation, or altering the value of a reward such that it functions as a powerful reinforcer, enhances the potential for positive learning outcomes. In the field of autism, establishing operations play an important part for increasing verbal behavior, especially when teaching manding (requesting). When there is a strong establishing operation in place, there is an increased likelihood that the target behavior will be demonstrated in order to access the reinforcer. Accessing the reinforcer, in turn, strengthens the response and increases the likelihood that it will occur again under the same or similar conditions.

Goals and Objectives

As noted previously, the goal of using EOs is to alter the value of a reinforcer such that the target response will be more likely to occur. Use of EOs increases the effectiveness of a particular stimulus change as reinforcement. EOs play an important role in behavior change.

Treatment Participants

Studies that examine the effects of EOs have been executed and replicated for years and have focused on many different populations in the research from animals to human beings. The examination of EOs has been particularly

prevalent in the research conducted on verbal behavior and, of late, has included studies focused on teaching persons with autism spectrum disorders to request desired items. Research has also focused on aberrant behaviors of persons with severe disabilities who engage in self-injury, aggression, and pica (McGill 1999).

Treatment Procedures

EOs have been included in such treatment procedures such as (a) extinction, (b) noncontingent reinforcement, (c) multicomponent treatment packages, (d) social attention (behavioral momentum), (e) functional communication training, (f) naturalistic/incidental language training, and (g) social-positive, social-negative, and automatic reinforcement (McGill 1999).

Efficacy Information

Large quantities of research exist to point to the efficacy of EOs within the context of reinforcer delivery. Researchers have demonstrated the salience of student choice in increasing the value of a reinforcer (Mason and Egel 1995; Mason et al. 1989; Lerman et al. 1997), varying the presentation of the reinforcer (Egel 1980, 1981), states of deprivation versus satiation (Volmer and Iwata 1991), properties of sensory stimuli as reinforcers (Ferrari and Harris 1981; Rincover and Newsom 1985), attention as an establishing operation and reinforcer during functional analysis (Fisher et al. 1997), teaching students with autism to mand for social information (Shillinsburg et al. 2016; Gutierrez et al. 2007), and effects of conditioned establishing effects on stereotypy (Lanovaz et al. 2014). Each of these studies has demonstrated that EOs are intimately linked to what a person wants at any given time, and in so doing, establishing operations affect the potency of the reinforcer.

Outcome Measurement

Current outcome measures for EOs support their importance across a variety of research

applications. Quantitative data collected during research regarding EOs has repeatedly proven that EOs have an impact on behavior change. EOs have been examined in the context of teaching language and communication to students with autism as well as other disabilities. They also have been examined within the context of other studies of verbal behavior as well as reduction of aberrant behaviors such as self-injury, pica, and aggression. Although the bulk of research that has been conducted involves single-subject research design, the quantity of replication across behaviors, subjects, and settings speaks to the validity of EOs in the context of examining reinforcers and reinforcement.

Qualifications of Treatment Providers

Although not explicitly stated in the research, persons who provide reinforcers in the context of teaching and/or behavior change projects should be knowledgeable in the field of applied behavior analysis and/or experimental analysis of behavior. Careful examination of the effects of any behavior change procedure should be closely monitored through data collection and analysis; modifications should be implemented according to data analysis. As such, qualified behavior analysts are providers that can use EOs within the context of educational procedures and behavior change procedures. Special education and general education teachers and paraprofessionals are capable of implementing reinforcement procedures under the guidance of a qualified behavior analyst.

References and Reading

- Bailey, J. S., & Burch, M. R. (2002). *Research methods in applied behavior analysis* (p. 182). Thousand Oaks: Sage Publications.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). Positive reinforcement. In *Applied behavior analysis* (2nd ed., pp. 263–365). Upper Saddle River: Pearson/Merrill Prentice Hall.
- Egel, A. L. (1980). The effects of constant vs varied reinforcer presentation on responding by autistic children. *Journal of Experimental Child Psychology*, 30, 455–463.
- Egel, A. L. (1981). Reinforcer variation: Implications for motivating developmentally disabled children. *Journal of Applied Behavior Analysis*, 14, 345–350.
- Ferrari, M., & Harris, S. L. (1981). The limits and motivating potential of sensory stimuli as reinforcers for autistic children. *Journal of Applied Behavior Analysis*, 14, 339–343.
- Fisher, S. M., Iwata, B. A., & Worsdell, A. S. (1997). Attention as an establishing operation and as reinforcement during functional analysis. *Journal of Applied Behavior Analysis*, 30, 335–338.
- Gutierrez Jr., A., Vollmer, T. R., Dozier, C. L., Borrero, J. C., Bourret, J. C., & Gadaire, D. (2007). Manipulating establishing operations to verify and establish stimulus control during mand training. *Journal of Applied Behavior Analysis*, 40, 645–658.
- Keller, F. S., & Schoenfeld, W. N. (1950). *Principles of psychology*. New York: Appleton.
- Lanovaz, M. J., Rapp, J. T., Long, E. S., Richling, S. M., & Carroll, R. A. (2014). Preliminary effects of conditioned establishing operations on stereotypy. *Psychological Record*, 64, 209–216. <https://doi.org/10.1007/s40732-014-0027-x>.
- Lerman, D. C., Iwata, B. A., Rainville, B., Adelinis, J. D., Crosland, K., & Kogan, J. (1997). Effects of reinforcement choice on task responding in individuals with developmental disabilities. *Journal of Applied Behavior Analysis*, 30, 411–422.
- Mason, S. A., & Egel, A. L. (1995). What does Amy like?: Using a mini reinforcer assessment to increase student participation in instructional activities. *Teaching Exceptional Children*, 28, 42–44.
- Mason, S. A., McGee, G. G., Farmer-Dougan, V., & Risley, T. R. (1989). Client selected rewards: A practical strategy for ongoing assessment of stimulus preference in the classroom. *Journal of Applied Behavior Analysis*, 22, 171–179.
- McGill, P. (1999). Establishing operations: Implications for the assessment, treatment, and prevention of problem behavior. *Journal of Applied Behavior Analysis*, 32, 393–418.
- Michael, J. (1982). Distinguishing between discriminative and motivational functions of stimuli. *Journal of the Experimental Analysis of Behavior*, 37, 149–155.
- Peterson, N. (1978). Establishing operations. In *An introduction to verbal behavior* (pp. 19–21). Michigan: Behavior Associates.
- Rincover, A., & Newsom, C. D. (1985). The relative motivational properties of sensory reinforcement with psychotic children. *Journal of Experimental Child Psychology*, 24, 312–323.
- Shillinsburg, M. A., Frampton, S. E., Wymer, S. C., & Bartlett, B. (2016). A preliminary procedure for teaching children with autism to mand for social information. *Behavior Analysis in Practice*, 1(1), 1–5. <https://doi.org/10.1007/s40617-016-0163-7>.
- Skinner, B. F. (1953). *Science and human behavior*. New York: Macmillan.

- Vargas, J. S. (2009). Verbal behavior. In *Behavior analysis for effective teaching* (pp. 245–258). New York: Routledge.
- Volmer, T. R., & Iwata, B. A. (1991). Establishing operations and reinforcement effects. *Journal of Applied Behavior Analysis*, 24, 279–291.
- Wilder, D. A., & Carr, J. E. (1998). Recent advances in the modification of establishing operations to reduce aberrant behavior. *Behavioral Interventions*, 13, 43–59.

Estate Planning

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Definition

In General

“Estate planning” refers to one’s instructions for the distribution of assets after one’s death, and includes the establishment of a will and/or trusts, and the gifting of property. Estate planning also includes preparing a plan for the care of a child who will need assistance after the parents or other caregivers have died.

Planning for Children with Disabilities, Generally

Thoughtful estate planning for children with disabilities utilizes all resources, private and public, to optimize the child’s security and quality of life. Actors involved in the planning process should include the parents and other caregivers, social workers, disability law lawyers, financial planners, and others who possess relevant skills.

Subjects of the estate plan should include economic resources, living arrangements, caregiver selection, and estate management.

Planning with Private Assets

Private assets include all property, both real and personal, and life insurance. Private assets can be managed through the creation of trusts and the

designation of a manager or conservator of the funds. The trusts instruments can include living trusts, created while the parents and/or caregivers are alive, or testamentary trusts, created upon death. One can direct distribution of assets by will.

Planning with Public Assets

Public assets include Medicaid, which covers health care, and Supplemental Security Income (SSI), which provides “a minimum income” for qualifying individuals, including the disabled. Federal law defines “disabled” for purposes of SSI eligibility as, “the inability to engage in any substantial gainful activity by reason of any medically determinable physical or mental impairment which can be expected to result in death or which has lasted or can be expected to last for a continuous period of not less than twelve months.”

Social Security also provides benefits relevant to estate planning for one’s disabled child. The program provides benefits to an adult child “when that parent retires, becomes disabled, or dies.” The child must have a disability that manifested before the age of 22, be unmarried, and have been dependent on the person who earned the Social Security benefits.

References and Reading

- Black’s Law Dictionary. (2017). Retrieved from <http://thelawdictionary.org/estate-planning/>
- Eichstadt, G. C. (2000). Using trusts to provide for the needs of an adult child with a disability: An introduction to family concerns for lawyers and a primer on Trust for Parents. *S.D.L. Rev.*, 21, 45–74.
- Hoyt, P. R., & Pollock, P. M. (2003). *Special people, special planning: Creating a safe legal haven for families with special needs*. Orlando: Legacy Planning Partners.
- Kirby-McLemore, J. M. (2016). What are aging parents caring for adult children with disabilities to do?: A comprehensive framework for a healthy, stable, financially sound future. *Roger Williams U. L. Rev.*, 21, 45–73.
- Krooks, B. A., & Hook, A. (2005). *What attorneys need to know about special needs trusts*. Philadelphia: ALI-ABA. Retrieved from http://files.ali-aba.org/thumbs/datastorage/lacidoirep/articles/EPCM_J0510-KROOK_thumb.pdf.
- Frolik, L., & Brown, M. (2015). *Advising the Elderly or Disabled Client*. New York, Thompson Reuters.

- Medicaid Statute: 42 U.S.C. §1396, et seq. (2017).
- Medicaid: a primer. (1999). Washington, DC: The Kaiser Commission on Medicaid and the Uninsured. *Medicaid regulations*: 42 C.F.R. §430, et seq. (2017).
- Moore, R. J. & Landsman R. M. (2000, Supp. 2003). *Planning for disability*. Arlington: The Bureau of National Affairs.
- Russell, L. M., & Grant, A. E. (1995). *Planning for the future: Providing a meaningful life for a child with a disability after your death* (3rd ed.). Palantine: American Publishing Company.
- Russell, L. M. (1983). *Alternatives, a family guide to legal and financial planning for the disabled*. Lasalle: First Publication, Inc.
- Schneider, A. (2002). *The Medicaid source book*. Washington, DC: The Kaiser Commission on Medicaid and the Uninsured.
- Supplemental Security Income Regulations: 42 U.S.C. §1383c, et seq. (2017).
- Trapp, G. D. (1995). Estate planning: What parents of children with disabilities should know. *Future Reflections*, 14(3.) Retrieved from <https://nfb.org/images/nfb/publications/fr/fr14/issue3/fr140311.html>.

Estrogens (Female Sex Hormones)

► Sex Hormones

Ethics

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Definition

Broadly speaking, ethics is the philosophical study of how we ought to live and what kinds of people and societies we ought to be or become. While the term sometimes refers to the systematic study of reasoning about how we ought to act often called moral philosophy, ethics is also concerned with matters of the most fundamental practical import. Central among its questions are those to do with how, as individuals and societies,

we ought to treat others, why we ought to treat them in that way, how we should evaluate the motives underlying actions, and our responsibility for the predictable consequences of our actions and policies. The various answers to questions of this sort often depend on different general viewpoints drawn from moral theories such as deontology or consequentialism or from various religious perspectives, all with their different metaethical and ontological assumptions. In addition to this philosophical input, reflection on many ethical issues, such as those raised by autism spectrum disorders, should be informed to a large extent by empirical research in order to identify and clarify what the relevant issues are.

However, a continuing lack of knowledge about the biological causes of autism and its neurobiological bases means that there is a corresponding uncertainty about the identification and resolution of the moral issues specific to autism and about how much illumination can be drawn from more general and established ethical discourses such as those concerning disability rights or reproductive ethics. Given the current state of knowledge about autism, there are actual and emergent ethical issues relating to its diagnosis and categorization, its prevention and cure, its treatment, and its associated research practices and agenda. All of these ethical concerns tend to have both conceptual and empirical aspects.

Historical Background

As a neurobiological developmental condition, what we now call autism has almost certainly always existed in the human population. However, it was only identified as a distinct condition in the mid-twentieth century. Hence the concept of autism has a relatively short history. From the 1940s until the 1980s, autism was understood as a categorical condition affecting a relatively small number of people and characterized by a distinctive set of behavioral features such as severe delays in language, impaired cognitive skills, and a profound lack of emotional contact with others. The goal of therapy at that time was largely to “free” the child from the shell of

isolation imposed by autism. Later, this psychological account was replaced by the idea of autism as a biologically based problem. Treatment and research strategies changed accordingly while preserving the goal, assumed to be ethically straightforward, of its remediation. In the last three decades, a new body of knowledge has emerged from studies documenting the complex pattern of strengths as well as weaknesses present in autism and from autobiographical writings by people with autism, such as Temple Grandin. The introduction in the 1980s of the idea that autism is best understood as a spectrum condition was an attempt to capture the heterogeneity of autism and the many ways it is experienced by those affected and remains hugely influential in thinking about autism (Wing 1981). Currently a growing appreciation by researchers of the sheer heterogeneity of autism and the speculative nature of much of the science in this area has led some to suggest that rather than one unique phenomenon, there may be different “autisms,” with different underlying biological processes and developmental pathways (Happé et al. 2006; Volkmar et al. 2009). Some even suggest that “autism” could be understood as an umbrella term for numerous different conditions (Coleman and Gillberg 2012). Clearly, then, while autism is not a new condition (or range of conditions), the understanding and concept of autism are relatively new, still evolving, and currently quite contested – all of which reservations enter into the ethical debate about autism.

Current Knowledge

One of the major difficulties in identifying and clarifying the ethical issues raised by autism is the sheer range of ways in which the condition manifests itself. Unfortunately, the general public remains largely unaware of this level of complexity. An increasing current cultural fascination with autism is fed by representations of it established by books like *The Curious Incident of the Dog in the Night-Time* and iconic films like *Rainman*, where the savantism of the main autistic character has led many people to believe that people with

autism generally possess similarly unusual talents. In reality, the majority of diagnosed individuals sit at different points along the spectrum at different times and in different contexts, rather than occupying a static position at or near its extreme ends. Among the possibilities the idea of a spectrum attempts to capture is the autistic individual who is profoundly intellectually impaired and nonverbal, the one who is extremely disabled but has unique strengths in an area such as art, music, or mathematics, or the one who is able to work effectively in solitude but is overcome by serious social difficulties when asked to work in direct contact with others. Moreover it seems the position of an individual on the spectrum is not necessarily fixed through the course of their life, because the condition is developmental and because individuals may learn social and adjustment skills that may minimize their symptoms. Adding to this complicated picture, it seems that in any given period, an individual with autism may fit behavioral descriptions of both “high” and “low” functioning, in different respects and even in different settings. In comparison with other recognized disabilities then, autism seems unique in the sheer range of its symptoms and behaviors, in the relatively plastic nature of its symptoms (especially early in life), and in the possibility of movement along a wide spectrum through the course of life. Given this level of complexity, it is not surprising that the idea of a spectrum cannot capture all the variabilities in the manifestation of autism symptoms and the differences in health, developmental difficulties, and sensory problems that contribute to the different profiles of people affected by autism.

Compounding the difficulties for the spectrum idea is the problematic high-functioning/low-functioning distinction it encourages. In the diagnostic context, for instance, people with autism are often viewed as occupying a point somewhere on a broad spectrum from low to high functioning, reflected by, for example, the range in IQ from severe intellectual impairment to high IQ, or in communication styles from no speech or language use to high, often eccentric articulacy, associated with the condition. Though this distinction has passed into current popular understanding and

descriptions of autism, no such categories are mentioned in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), and there seems to be little scientific evidence to justify categorizing autistic people along these lines. The fact that most individuals with autism will move between these categories depending on context, circumstances, and the expectations placed on them means the distinction fails to capture an important reality about autism and therefore is doing little useful explanatory work. To some extent, the latest edition of the DSM (DSM-V 2013) sidesteps some of these difficulties by introducing levels of severity of autism (mild, moderate, and severe) based on social communication impairments and restricted repetitive patterns of behavior with three levels – individuals with autism may (1) require support or (2) require substantial support or (3) require very substantial support. Hence how an individual is categorized depends on his or her specific abilities to function across a broad range of contexts. Difficulties remain, however, to do with the assumption that autistic individuals function at the same level across all areas (ASAN 2017). Presumably the heterogeneity of autism described means that individuals will move between these categories of severity and necessary level of support, just as they were said to move between high and low levels of functioning.

The fact of heterogeneity presents obvious difficulties for identifying appropriate medical and educational interventions, but it also hugely complicates ethical questions about how we should regard those affected by autism and how we should respond to and intervene in their lives. Hence it is to be expected that any appropriate ethical judgment about autism will tend to be cautious, provisional, and open to much qualification.

One example of this is provided by the process of diagnosis. Initial diagnosis may provide the occasion, whereby individuals and their families come to understand for the first time the reason for the problems they have been experiencing, and importantly, it may serve as the mechanism through which they can access the range of interventions and supports that are available. On the other hand, it currently confirms someone as

a person with a neurologically based disorder, with all the implications of that for how the individuals view themselves, how others view them, and how they are viewed by services, teachers, employers, and so on. The clear moral seriousness of this is compounded by difficulties in making the diagnosis secure. First, in not a few cases, there will be the difficulty, implicit in the notion of a spectrum, of establishing a nonarbitrary cut-off point between behavior considered “normal” or “mildly eccentric” and behavior considered “disordered.” Second, in many more cases, the general difficulty of applying the accepted criteria with confident objectivity will be felt. The diagnosis of autism currently involves assessing an individual’s developmental history and patterns of behavior against purely behavioral criteria as set out in the internationally recognized diagnostic manuals, the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-V 2013) and the *International Classification of Diseases* (ICD-10 2010). Though minimal, these criteria are arrayed in complex ways such that there is no behavior or set of behaviors that unequivocally denote autism. Because the genetic and neurobiological bases of autism are as yet largely unknown, no more objective physical tests are available to the diagnostician. That lack has led to an intensive search for biological markers, which, in addition to shedding light on the causes of autism, might also be clinically useful in complementing and improving its behavioral diagnosis and in enabling its earlier detection. To date, comparatively little has been achieved in identifying biomarkers with clinical utility – though *if* they are found, they may well pose new ethical challenges in the field of autism (Walsh et al. 2011). It is clearly morally important that diagnostic dialogues should acknowledge these various difficulties and uncertainties to everyone concerned.

Asking whether those dialogues should not also include references to the positive aspects of autism brings a fundamental and profound question into focus: what value should be placed on autism as a condition, and on different aspects of the condition? Is it enough, or even correct, to characterize autism solely as a disability and, as such, something to be prevented or cured where

medically and ethically possible? The challenge here is to the prevailing “deficit model” of autism propagated by the exclusively negative character of the criteria in the diagnostic manuals. The “impairment” in question is characterized by examples of “failure,” “lack,” and “stereotyped,” “repetitive,” and “abnormal” behaviors for instance. While it can be argued that is required in order to secure services, nonetheless the wholly negative characterization of autistic behavior can be seen as implicitly evaluative and question-begging, rather than scientific. Unfortunately it has enormous influence on clinicians, researchers and research agenda alike. The challenge to the deficit model also extends to the many forms of treatment and intervention that, following the reasoning that guides diagnosis, focus strongly on counteracting behaviors taken to be undesirable. Whether in diagnosis or in treatment, there seems to be a good case for understanding, assessing and describing aspects of autism more positively: instead of deficits we find instead strong persistent interests, attention to detail, and unusual memory, as well as heightened skills that are associated with some forms of autism, such as great visual acuity, perfect musical pitch, and remarkable mathematical prowess. (Walsh 2010; Baron-Cohen et al. 2009).

Nevertheless, the idea that autistic behavior can be most helpfully understood in terms of a “triad of impairments” in communication, imagination, and social interaction, which emerged in the late 1970s, has been the impetus for many influential theoretical developments (Wing and Gould 1979). Psychologists have attempted to explain each of these fundamental deficits, with the aim of clarifying the forms of treatment that could ameliorate and compensate for them. According to one theory, people with autism lack a “theory of mind” in being unable to understand the perspectives and points of view of other people (Baron-Cohen 1995). A second “executive function” theory explains some aspects of the repetitive behaviors seen in autism as an inability to plan sequences and actions (McGregor et al. 2008). A third “weak central coherence” theory is based on the idea that people with autism cannot process differences in a manner that makes them

cohere into a general pattern (Frith 1989). Each of these deficit views of autism has moral as well as scientific and practical implications. Some philosophers, for instance, have taken the first of these theories in particular – the view that people with autism are “mindblind” – as posing a legitimate query as to whether they have the full moral status of “persons” (Barnbaum 2008). The argument draws on a putative distinction between the concepts “human being” and “person” which gained currency in bioethical debates about the moral permissibility of abortion in the 1970s. Being genetically human, it was (and is) argued, is a morally neutral fact, while the term “person” identifies those *morally* important features of beings, including human beings, that should guide our estimate of their moral status and, thereby, our thinking and decision-making in their regard. Features of persons said to provide reasons for regarding them as subjects of moral concern and bearers of the fundamental protection of rights vary but centrally include consciousness, self-consciousness, a capacity for rational decision-making, and a capacity to communicate. In possessing none of these characteristics, it is argued, for example, that a human fetus is not a person in this sense and that abortion may therefore be morally permissible. The same distinction has since been sometimes considered in relation to people in a persistent vegetative state and the severely demented and their treatment. In regard to autism, it is suggested that the lack of a functioning theory of mind means people with autism are unable to recognize that others have beliefs, preferences, emotions, and desires independent of their own. The resulting purported lack of empathy leads to an inability to relate to others as persons and to enter into meaningful reciprocal relationships with them. Even more fundamentally, being mindblind may mean they are not able to reflect on their own mental states. On this view of the matter, at least some autistic people fail on two of the criteria of personhood. Though undoubtedly conscious, they may not be conscious of other people in the meaningful ways central to human relationships, and they may not be self-conscious in the ways necessary to qualify for moral personhood.

The more general question of the value to place on autism is also seen as having to engage with philosophical discussions of the conditions necessary for a good or flourishing human life. Most accounts of these conditions give a central place to the emotions, including particularly having personal relationships and a sense of affiliation with other people (Nussbaum 2006). Many of us (including many autistic people) would agree that friendship, for example, is an essential contributor to our well-being and recognize that autistic people have particular difficulties in this area. However, it is sometimes argued that individuals who lack a theory of mind, like those with autism, simply cannot live a full flourishing human life because their inability to empathize compromises their friendships and their sense of affiliation with others.

What might be said in response to these suggestions of the severe limits on the value of autistic life, said to follow from the deficit account of “mindblindness”? First, it is extremely important to remember that “mindblindness” is a *metaphor*, not a statement of fact, and that metaphors can be harmful and also incorrect. The most we can infer using this metaphor is that autistic people sometimes behave *as if* they were mindblind (Smukler 2005). Second, if this deficit hypothesis is not valid – not correct or only partly correct – none of the ethical implications described would follow. And in fact there are many problems about the precise status of the mindblindness hypothesis, to do with its explanatory power and specificity to the case of autism, how it interacts with the other two “fundamental deficit” theories, and, crucially, whether it can incorporate and reflect the idea of a spectrum of *developmental* abilities and impairments (Wellman et al. 2001). At present, it may be best viewed as a metaphorical description rather than an explanation of autistic behavior or as a step on the way to a full and coherent psychological explanation of autism. But even if the “mindblindness” theory is correct, neither of the claims said to follow, about the moral status of people with autism and the possibility of a flourishing autistic life, would necessarily follow. It is important to remember that the notion of a moral person at play here is highly

stipulative, a matter of moral argument and decision rather than an empirical discovery of facts about human beings. Indeed, the long history of the idea that those who are “different from us” should thereby be placed outside the circle of our moral concern or assigned a diminished level of moral status is an ignominious one. Again, accounts of a flourishing human life that assign so central a place to friendship and the emotional life may be seen as simply begging the question when it comes to people with autism. Even for non-autistic people, the need for friendship and socialization fluctuates and may be quite minimal. To assume that socialization with others is so important that we cannot have well-being without it may be to buy far too much into a contemporary culture which values extrovert behavior. Furthermore, it can be seen as dangerous, for the future if not the present. Arguing that autistic people alive today have a less flourishing life than the rest of us may be a problem for them, but its implications may be much more fateful for a future context in which emergent genetic technologies will play a major role in reproductive decisions about whether to prevent the birth of disabled individuals.

The advent of a “neurodiversity” movement led by, and giving a voice to, people on the autistic spectrum and their families and supporters has brought a sharp political edge to this ethical question of how we should value autism. Drawing on a wider political debate about disabilities and disability rights and paralleling campaigns by other disadvantaged groups to change societal perspectives of their condition, the neurodiversity movement forces attention onto the complexities and contested nature of the concept “normality,” the positive aspects of autistic spectrum conditions, and the advantages as well as the justice of respect for cognitive differences. Autism is claimed to be simply a neurologically atypical, but not abnormal, human variation. A distinction between “neurodiverse” and “neurotypical” functioning displaces the conceptually and morally problematic distinction between “normal” and “abnormal” functioning, with the implication that we should no longer think of autism as a condition requiring fundamental treatment,

correction, or prevention. Support for this stance is sometimes drawn from the list of famous people – including Isaac Newton, Albert Einstein, Carl Jung, Immanuel Kant, Vincent Van Gogh, Bill Gates, and Andy Warhol, among others – who are deemed to have had, or to have, autistic traits. Whatever their case to case legitimacy – and this may be doubted (Verhoeff 2012) – these attributions have been sufficiently plausible to contribute to a shift in the perception and representation of autism, from outright “disability” toward “difference,” while also encouraging those with autism to celebrate the distinctive strengths of their historical community. On the other hand, the neurodiversity movement’s drive to construct a specific autistic identity faces some possible social and philosophical challenges: for example, the claim that autistic people are specifically neurologically different – they are *neurodiverse* – is contentious, while there is little in the way of consensus about the etiology of autism (Ortega 2009). Similarly, the ontological claim that individuals are identical with and can be defined by their neurological traits is a claim which is currently highly contested and possibly philosophically incoherent. In any case, there is a risk to be guarded against; that emphasis on a biological basis of “difference” – a still unidentified but presumed to be “deep” basis – may inadvertently reinforce queries regarding the moral status of people with autism. There is, perhaps, such a thing as being “too different.”

On the other side of the debate about how to value autism, an equally vocal lobby regards autism as a serious disability and supports funding of the kind of scientific research that might lead to prevention and cure. These objectives are shared by many carers of autistic people and many autistic people themselves, who claim that the neurodiversity movement underestimates the grave effect autism has on the lives of everyone involved. It does not, and cannot, represent those so severely disabled by autism that they are incapable of speaking for themselves or the families and carers of those with severe cognitive impairments or even those high-functioning individuals who often experience acute isolation and loneliness. The disagreement between this group and

the neurodiversity movement is somewhat reminiscent of the conflict within feminism in the 1960s and 1970s when there was a grassroots challenge to a feminism perceived as the expression of the needs and concerns of middle class white women alone and which ignored the different difficulties faced by some women due to race, class, nationality, poverty, level of education, and religion (Crenshaw 1991). Similarly, it might be said that while an identity-based politics such as that advocated by the neurodiversity movement may be a great source of strength and community, it also runs the risk of conflating and ignoring important intragroup differences and the obstacles to be overcome because those difficulties interact in complex ways with other possible forms of oppression.

The autism research agenda is a major focus of the conflict between advocates of neurodiversity and those who view autism as a catastrophic disorder. Despite the growing influence of the neurodiversity movement, early detection, prevention, and cure still seem to be the main impetus and the overriding goals of biomedical research into autism. The National Institute of Mental Health in the United States, for example, proclaims its vision to be “a world in which mental illnesses are prevented and cured” and names autism as one of a group of “serious, often life-threatening illnesses for which we need reliable diagnostic tests, new treatments, and effective strategies for prevention” (National Institute of Mental Health [NIMH] 2008). One of the largest autism charities in the world, Autism Speaks, declares itself “dedicated to funding research into the causes, prevention, treatment and a cure for autism” and has donated more than \$160 million to such research since its inception in 2005 (Autism Speaks 2012). The official mission statements of such research agencies also include research support for those who must live with autism – and they and their supporters are not necessarily opposed to respecting cognitive difference. It remains, however, that these seem not to be their priorities. One of the main bones of contention in this area is about whether research funding should be directed toward improving the lot of autistic people living now rather than

be for the benefit of future generations of those with autism: proponents of the neurodiversity approach have little trouble suggesting research projects that would have objective practical benefit for those who live with autism, if only funds were more equitably allocated.

However, the more basic criticism from the neurodiversity perspective is that the goals of prevention, radical treatment, and cure are fundamentally misguided. The value issues here are complex. They include, but go well beyond, interpretations of the key terms. A “cure,” to start with it, ought to be rather more than something that would ameliorate the negative impacts of autism on affected individuals. But taken to mean something that “gets rid” of autism, it faces two distinguishable challenges. The first, relating to identity, is captured in a website statement of a view that is often expressed by those in the neurodiversity movement: “To ‘cure’ someone of autism would be to take away the person they are, and replace them with someone else” (Aspies for Freedom 2010). The obliteration of the condition is seen here as amounting to the destruction of an identity. That this view is persuasive owes much to two features of autism: first that by its cognitive nature, it has a pervasive impact on a person’s life and second that it is an early onset condition assumed to be a pre-experiential and pre-social “given,” though not manifesting itself in the very earliest years. In combination, these features imply that autism by its nature is truly deep set in those who have it and live with it. Whether it is constitutive of a person’s identity in the way suggested is an open and very difficult philosophical question. However, it seems pertinent to observe that identity, in one important sense, is something that is acquired, built up, and defined progressively. So, while curing, as opposed to ameliorating, an adult’s autism may be a destructive project, if indeed it is not an incoherent one, the case is less clear where infants are the likely recipients of the cure.

“Prevention,” to come to it, does not have to mean “eradication” inasmuch as its primary reference may be symptoms rather than the condition itself. So, a major goal of research programs, whether biological or psychological, may

be to identify interventions that “prevent” the more severe cognitive, behavioral, and social challenges that are often associated with autism and impact negatively on autistic individuals and their families. This prevention may be by early treatment that forestalls the significant development of these challenges or by interventions that reduce or limit their impact when they have already developed, by restoring function as much as possible. Regarding the latter, the neurodiversity lobby object to specific research and interventions aimed at “normalizing” certain autistic behaviors, such as prolonging eye contact or reducing “stimming,” where that is done for no more objectively important reason than to make the person with autism less visible by encouraging them to fit in with contemporary cultural norms.

The Future

All discussions of autism and the ethical problems it throws up suffer from the undermining effect of uncertainty about how well autism is established as a cohesive syndrome. The Medical Research Council of Great Britain puts this uncertainty this way: “Whilst it is well known that autism involves impaired social communication, language impairment, and repetitive, stereotyped interests, it is still unknown whether this constellation of impairments is inherent in a cohesive syndrome (usually called Autism Spectrum Disorder or ASD), or whether it is an artefact of diagnostic practice” (MRC 2010). Some scientists have suggested that autism is not a single large spectrum of disease but that what we call autism is in fact a group of conditions with multiple etiologies (Coleman and Gillberg 2012). Yet others question whether there is such a thing as autism in the natural biological world (Timimi et al. 2011) or draw attention to the way autism has emerged and developed in close relation to historical and social ideas about what constitutes acceptable and abnormal behavior (Nadeson 2005). The lack of a clear scientific, medical, and public consensus on the definition, causes, diagnosis, and treatment of autism means that many ethical issues pertaining to autism are probably

still to emerge. No doubt this process cannot be hurried given that it depends on future scientific and medical developments, on an array of social, cultural, philosophical, and psychological issues and crucially on the involvement of the autistic community. Two issues of current ethical importance are identified here.

The first concerns the area of intelligence testing in autism. The history of intelligence testing is, on the whole, an ignominious one in which many historical examples illustrate the fact that IQ tests merely confirm the existence of well-known social disadvantage (Gould 1996; Hayman 2000). It is said that most children with autism exhibit mild to moderate cognitive deficits with a characteristically uneven cognitive profile, though the rates of intellectual disability in autism range widely between 40% and 63% (Matson et al. 2008). One question that arises then concerns the precise relationship between autism and IQ levels: does level of IQ affect the range and expression of autism symptoms? Of more basic concern is the effect of mainstream deficit approaches to autism on our understanding and conceptualization of autistic intelligence. We have seen how these approaches may be criticized for the personal and social harm they effect. But is it also possible that they impede scientific and philosophical progress in understanding intelligence in autism? In general, IQ testing in autism is carried out in a way that shows little evidence of the much wider social debate about the general validity of IQ testing and what it tests, the equation of intelligence with IQ, and the existence of different sorts of intelligence (Gardner 1983). This narrow frame of reference combined with a deficit approach to autism not only determines what is researched but may also affect test results. For example, it seems many cognitive scientists fail to appreciate how the behavioral and communication difficulties associated with autism negatively affect test performance and need not denote lower intelligence despite low scores. And in viewing autism purely as a disabling condition, they may also not recognize that to describe and measure a possibly different cognitive style with tests designed to test what is

“normal” cognition will yield results of questionable validity (Eagle 2002). The interpretation of results may also display a research bias introduced by the deficit model – when intelligence and talent are recognized, they are usually pathologized as “splinter skills” or “islets of ability,” and it has been shown that in tests where an autistic group outperforms the non-clinical control group, the difference is often explained not by superior ability in that area but by reference to some cognitive impairment associated with autism. It is clear that how we represent people to ourselves and to others often determines the outcomes of our ethical judgments about them. But the consequences of a purely deficit account of autism may also be serious and far-reaching for social justice and possibly for science itself.

A second issue probably causes most consternation in many parts of the autism community, as well as being a concern to others. It is the prospect of a future range of genetic technologies capable of indicating levels of risk of autism for the use in the context of reproductive decision-making. If and when they are developed, the availability of such technologies, it is feared, will lead to an avoidance of having children by those identified as at risk of conceiving autistic offspring and, beyond that, to large-scale elective abortion of fetuses deemed to be at some – perhaps even quite small – level of risk.

Supporting the fears of proponents of neurodiversity about an agenda to eradicate autism, there is evidence to suggest that preventing the birth of individuals with disabilities that can be diagnosed prenatally seems to many an obvious step to take and is encouraged by medical professionals and bioethicists alike. Some bioethicists have even argued that there is a moral obligation to prevent the births of disabled individuals where possible and regardless of the level and kind of disability (Savulescu and Kahane 2009). As their decision-making is likely to be influenced by available genetic information, it will be important to offer parents counseling about genetic and non-genetic risk factors; potential social, educational, and developmental outcomes; and treatment options (Walsh et al. 2011). It will also be crucial

precisely how genetic counselors communicate the probabilistic and uncertain picture derived from genetics as well as the complex and contested nature of the autistic spectrum itself, since this is also likely to have a huge impact on what parents decide. From a moral point of view, this is an extremely important issue for genetic counselors and society at large, for it has been suggested that even if individual parental decisions in this area are not eugenic, *the choices on offer* may be regarded as such (Sparrow 2008).

Professional guidelines for genetic counselors display a strong commitment to patient autonomy particularly in regard to reproductive decisions because of a belief that parents are in the best position to know what they should do and have a right to choose (and also, no doubt, to distance the practice of genetic counseling from questions of eugenics and the promotion of abortion). Many would agree that the final moral decision appropriately lies with the choosers among the available options – that is, the parents of the actual or potential child at risk of autism – with the hope that important ethical considerations regarding, for example, the morality of abortion or how we should think about disability and disabled lives would continue to weigh heavily in the choices people make. Even this seemingly moderate point of view may be challenged however. It could be argued that just as our autonomous decisions affecting others including our children after birth are restricted by considerations of harm to others, so should they be similarly restricted before birth. Should parents decide to abort a fetus identified as at risk for autism (if and when that becomes possible), their choice, it may be claimed, harms the fetus in that from the fetus' point of view, it is better to be born autistic than not born at all. Hence the harm criterion suggests abortion in such cases is wrong (Barnbaum 2008). It is unclear whether proponents of this view think abortion is wrong in all circumstances, wrong in all cases of disability, or wrong because it is the abortion of people with autism. However, if living an autistic life is less good than living a non-autistic life, considerations of harm would not serve to restrict those parental choices aimed at preventing the existence of autistic children (e.g.,

through preimplantation genetic diagnosis). What this brief discussion of the complexity of reproductive decision-making as it pertains to autism again indicates is how necessary it has become to think about how autism is and should be characterized: it seems winning the battle of words about the value of autism is not only important for setting and reorienting the research agenda but is likely to be crucial in life and death decision-making in the context of future reproductive choices.

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed., text rev.). Arlington: American Psychiatric Association.
- Aspies for Freedom. (2010). Retrieved from <http://www.aspiesforfreedom.com>
- Autism Speaks. (2012). Retrieved from <http://www.autismspeaks.org>
- Barnbaum, D. R. (2008). *The ethics of autism: Among them but not of them*. Bloomington: Indiana University Press.
- Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory of mind*. Cambridge: MIT Press.
- Baron-Cohen, S., Ashwin, E., Ashwin, C., Tavassoli, T., & Chakrabarti, B. (2009). Talent in autism: Hyper-systemizing, hyper-attention to detail and sensory hypersensitivity. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 364, 1377–1383.
- Coleman, M., & Gillberg, C. (2012). *The autisms*. Oxford: Oxford University Press.
- Crenshaw, K. (1991). Mapping the margins: Intersectionality, identity politics and violence against women of color. *Stanford Law Review*, 43, 1241–1299.
- Eagle, R. S. (2002). Accessing and assessing intelligence in individuals with low functioning autism. *Journal on Developmental Disabilities*, 9, 45.
- Frith, U. (1989). *Autism: Explaining the enigma*. Oxford: Blackwell.
- Gardner, H. (1983). *Frames of mind: The theory of multiple intelligences*. New York: Basic Books.
- Gould, S. J. (1996). *The mismeasure of man*. New York: W.W. Norton.
- Happé, F., Ronald, A., & Plomin, R. (2006). Time to give up on a single explanation for autism. *Nature Neuroscience*, 9, 1218–1220.
- Hayman, R. L. (2000). *The smart culture: Society, intelligence and law*. New York: New York University Press.
- Matson, J., Dempsey, T., LoVullo, S., & Wilkins, J. (2008). The effects of intellectual functioning on the range of core symptoms of autism spectrum disorder. *Research in Developmental Disorders*, 29, 341–350.

- McGregor, E., Nunez, M., Cebula, K., & Gomez, J.-C. (Eds.). (2008). *Autism: An integrated view from neurocognitive, clinical and intervention research*. Oxford: Blackwell Publishing/E. L. Hill.
- Medical Research Council of Great Britain. (2010). *MRC autism forward look and review*. Retrieved from <http://www.mrc.ac.uk/Utilities/Documentrecord/index.htm?d=MRC007354>
- Nadeson, M. H. (2005). *Constructing Autism: Unravelling the 'Truth' and Understanding the Social*. Routledge.
- National Institute of Mental Health. (2008). *NIMH strategic plan*. Retrieved from <http://www.nimh.gov/about/strategic-planning-reports/index.shtml>
- Nussbaum, M. (2006). *Frontiers of justice: Disability, nationality, species membership*. Cambridge: Harvard University Press.
- Ortega, F. (2009). The cerebral subject and the challenge of neurodiversity. *BioSocieties*, 4, 425–445.
- Savulescu, J., & Kahane, G. (2009). The moral obligation to create children with the best chance of the best life. *Bioethics*, 23, 274–290.
- Smukler, D. (2005). Unauthorised minds: How “theory of mind” theory misrepresents autism. *Mental Retardation*, 43, 11–24.
- Sparrow, R. (2008). Genes, identity and the expressivist critique. In L. Skene & J. Thompson (Eds.), *The sorting society*. Cambridge: Cambridge University Press.
- The Autistic Self Advocacy Network (ASAN) Position Statements. (2017). Retrieved from <http://www.autisticadvocacy.org>
- Timimi, S., Gardner, N., & McCabe, B. (2011). *The myth of autism*. London: Palgrave Macmillan.
- Verhoeff, B. (2012). What is this thing called autism? A critical analysis of the tenacious search for autism's essence. *BioSocieties*, 7, 410–432.
- Volkmar, F. R., State, M., & Klin, A. (2009). Autism and autism spectrum disorders: Diagnostic issues for the coming decade. *Journal of Child Psychology and Psychiatry*, 50, 108–115.
- Walsh, P. (2010). Asperger syndrome and the supposed obligation not to bring disabled lives into the world. *Journal of Medical Ethics*, 36, 521–524.
- Walsh, P., Elsabbagh, M., Bolton, P., & Singh, I. (2011). In search of biomarkers for autism: Scientific, social and ethical challenges. *Nature Reviews Neuroscience*, 12, 603–612.
- Wellman, H. R., Cross, D., & Watson, J. (2001). Meta-analysis of theory of mind development: The truth about false belief. *Child Development*, 72, 655–684.
- Wing, L. (1981). Language, social and cognitive impairments in autism and severe mental retardation. *Journal of Autism and Developmental Disorders*, 11(1), 31–44.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11–29.
- World Health Organisation. (2010). *International classification of diseases (ICD-10)*. Geneva: World Health Organisation.

Ethiopia and Autism

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Historical Background

Historic, Economic, and Cultural History of Ethiopia

Ethiopia is a country located in the Horn of Africa rich in archeological, historical, and cultural heritage. Archeological findings have mapped Ethiopia as the birthplace for humans, *Homo sapiens* (McDougall et al. 2005). Lucy, a hominin who lived 3.2 million years ago was discovered in 1974 in the Awash Valley, Ethiopia (Johanson and White 1979). Ethiopia also has the highest number of UNESCO World Heritage Sites in Africa reflecting the role the region played in ancient and modern civilizations. Axum (monolithic obelisks), Fasil Ghebbi (palaces of Ethiopian sixteenth and seventeenth century emperors), Harar Jugol (walled historic city), and Konso cultural landscape and Lalibela (rock-hewn churches) (Ethiopia 2016a) are a few of the many sites. Ethiopia is also home to natural wonders like the Ethiopian Highlands, the Roof of Africa that form the largest continuous mountain through which the Blue Nile courses (Henze 2000). These mountains are inhabited by endemic animals like the gelada baboon, walia ibex, and the Ethiopian Simien fox. On the south-eastern part of the country in the Oromia region lies the Sof Omar Cave, which is the largest system of caves in Africa (Robson 1967).

In addition to its historical heritage, Ethiopia is notable for its strong resistance against Italian occupation that was used as a model for anti-colonial resistance across the continent. Ethiopia was in the forefront during the formation of Organization of African Unity that is now African Union (El-Ayouty and Zartman 1984). Addis Ababa, Ethiopia's capital city continues to be the center for African peace and development efforts as it serves as the headquarters of United Nations

Economic Commission for Africa, the African Union, and the Pan African Chamber of Commerce and Industry.

Ethiopia has one of the fastest growing economy in Africa with a GDP per capita of \$1800 (Ethiopia 2016b). Ethiopia has a population of approximately 100 million people (Ethiopia 2016b). Eighty percent of the population lives in rural areas primarily engaged in agriculture. Ethiopia's very diverse population comprises of more than 80 ethnic groups. There are about 88 individual languages spoken in Ethiopia. Amharic is the official national language and has its own unique script that grew out of the Ge'ez, ancient liturgical language (Roncovic 2005). Oromo, most widely spoken language in Ethiopia, Somali, Tigrigna, and Afar are regional official working languages (Ethiopia 2016b). English is the major foreign language taught in schools and universities (Ethiopia 2016b). Several sign languages that are derived from signed Amharic and English languages are primarily used in schools for those with hearing disabilities throughout the nation (Morgan 2009).

Education in Ethiopia

Traditional education and literacy associated with the Coptic Church and mosques in Ethiopia dates back over 1000 years (Ridley and Bridges 1998). Western style education was introduced to Ethiopia in 1908 (Tessema 2007). Despite a long history of education in the country, the literacy rate in Ethiopia is still low at 49.1% as of July 2016 (Ethiopia 2016b). The low literacy is partially due strain on the availability and access to resources by the rapid rise in population and increasing burden of those under the age of 15 which currently accounts for about 40% of the total population (Ethiopia 2016b). In fact, in many areas, the student to teacher ratio can be greater than 40:1. In addition to the teacher shortage, another obstacle for early education in Ethiopia is the lack of training in education; indeed, only 48% of primary school teachers are trained (Education for All: Global Monitoring Report 2014).

Furthermore, not only is primary education sparse and of poor quality, but special needs

education is also practically nonexistent. Out of 285 schools with special needs classes, none were autism specific and they were evaluated and deemed between very poor and satisfactory by the Federal Ministry of Education (Burton 2016). There are no school programs specifically designed for children with autism within public schools. Hence, children with developmental disorders and disabilities are often confined to staying at home. In fact, it is estimated that only 4% of children with special needs receive primary schooling (Burton 2016).

Mental Health in Ethiopia

Research about and services available for mental health in Ethiopia is very scarce. Only 1.7% of the national health expenditure in Ethiopia was spent on mental health (Russo 2007). Amanuel Hospital is the only psychiatric hospital in Ethiopia. Mental health services and human rights issues are not regulated by external reviewing committees and Amanuel Hospital itself serves as both a service provider and a regulator of its services. There are 53 outpatient and 6 inpatient psychiatric facilities around the country, and they lack specific programs for children and adolescents (WHO-AIMS Report on Mental Health System in Ethiopia 2006). As of 2007, there were only 17 psychiatrists (all of whom work in the capital city, Addis Ababa) and 190 psychiatric nurses in the entire country (Russo 2007).

During medical training education in Ethiopia, medical students spend only 2% of their curriculum learning about mental health issues and how to manage them (WHO-AIMS Report on Mental Health System in Ethiopia 2006). In terms of physicians, all primary care physicians are able to prescribe psychotropic medicines without restrictions in Ethiopia (WHO-AIMS Report on Mental Health System in Ethiopia 2006).

Autism in Ethiopia

Autism and other neurodevelopmental disorders in Ethiopia are largely viewed as a curse. Children with these disorders are often hidden from the society. In addition to the lack of special services for them, their isolation often lead to their poor

health and nutrition. It is estimated that about 500,000 children with autism live in Ethiopia (Burton 2016). Research and services for mental health have focused on severe mental disorder disorders so far (Burton 2016). National Mental Health Strategy has started an initiative to educate primary health-care workers about mental health disorders but neurodevelopmental disorders have yet to be included in the training (Burton 2016). In the two child and adolescent psychiatric clinics in Ethiopia, neurodevelopmental disorders are the second leading cases after epilepsy (Burton 2016).

Legal Issues, Mandates for Service

Various national and international initiatives have been underway in the past two decades to improve the services for those with mental health problems and disabilities. In accordance with the UN Convention on the Rights of Persons with Disabilities, the National Plan of Action for Inclusion of Persons with Disabilities 2010–2020 was designed and finalized in 2011 to grant equal rights to people with disability and ameliorate their standard of living in Ethiopia (WHO-AIMS Report on Mental Health System in Ethiopia 2006). In addition, to improve the quality and the availability of primary education in Ethiopia, United Nations Educational, Scientific and Cultural Organization (UNESCO) has made a five-year plan to train about 6000 well-qualified teachers, 155 of which would be trained in special needs education (WHO-AIMS Report on Mental Health System in Ethiopia 2006). World Health Organization Mental Health Gap Action Program (WHO mhGAP) is also preparing a parent training program for those with children with neurodevelopmental disorders to address the misunderstandings about the disorders and help them interact with and manage challenging behaviors of their children (Burton 2016).

Health Education and Training + (HEAT+) project that was launched in 2012 is primarily aimed at helping health workers to be able to identify neurodevelopmental disorders in the field (Burton 2016). Health extension workers,

who provide health education about topics like hygiene and contraception, are being trained to detect and support those with mental health conditions by the Ethiopian Federal Ministries of health and Education and the Open University (UK) (Burton 2016).

Overview of Current Treatments and Centers

The Nia Foundation's Joy Center was founded by Zemi Yenus in 2003. She opened the school after seeing how schools were unwilling to accept her son with autism. Now the center provides services for about 80 children and offers diagnosis and assessment for autism (Burton 2016). Ms. Yenus has been in the forefront of campaigns to spread awareness about autism.

Nehemiah Autism Center was established in 2010 by ReachAnother Foundation and Rahel Abayneh in Addis Ababa. ReachAnother Foundation, founded by Dr. Marinus Koning, is a US based, nonprofit organization that aims to improve healthcare and special education services in underserved populations in Ethiopia. Ms. Abayneh like Ms. Yenus was determined to open an autism center because she could not find services for her son with autism. When the center was first opened, its teachers were previous kindergarten teachers, parent volunteers of children with autism, and psychology graduates from Addis Ababa University. The center primarily uses principles of Applied Behavior Analysis (ABA) on the children attending the center. Currently, 60 children receive services at Nehemiah.

The Bethel Adama Center for Autism was established in 2015 in Adama in the Oromiya region by the ReachAnother Foundation. It is the first center established outside the capital city and it currently has four teachers who provide services to 20 children.

The Bright Autism Center was recently established in May of 2016 in Hawassa, in the Southern Nations, Nationalities and Peoples' region of Ethiopia also by ReachAnother Foundation. The center closely collaborates with the

Hawassa University Psychology Department and University of Dilla Special Education Department to train teachers and spread awareness about autism in the local community.

Overview of Research Directions

Given that autism has only recently garnered attention, not much research has been conducted about autism in Ethiopia. In fact, the first autism-related research project was conducted as part of the Health Education and Training + (HEAT+) project. The study showed the limited amount of diagnostic and education services for children with autism in Ethiopia (Tekola et al. 2016). The prevalence of neurodevelopmental disorders, the clinical manifestation of autism in this subset of population, and the effectiveness and the application of autism diagnosis and treatment methods within the context of Ethiopia are yet to be studied.

Overview of Training

The four autism centers have not only been sources of services for children with autism and other neurodevelopmental disorders but also have been critical in autism training in Ethiopia. Professionals through the ReachAnother Foundation and Addis Ababa University Psychology Departments make visits to these centers and train the teachers in speech pathology, occupation therapy, and ABA therapy.

See Also

► [Autism Science Foundation](#)

References and Reading

Burton, A. (2016). Ethiopia: Educating everyone about autism. *Lancet Neurology*, 15, 1307–1308. [https://doi.org/10.1016/S1474-4422\(16\)30297-6](https://doi.org/10.1016/S1474-4422(16)30297-6).

- Education for All: Global Monitoring Report. (2014). UNESCO.
- El-Ayouty, Y., & Zartman, I. W. (1984). The OAU after twenty years. *David Lubin Memorial Library, Food and Agriculture Organization of the U. N.*
- Ethiopia. (2016a). *UNESCO World Heritage Centre*.
- Ethiopia. (2016b). *The World Factbook. Central Intelligence Agency*.
- Henze, P. B. (2000). *Layers of time: A history of Ethiopia*. London: C. Hurst & Publishers.
- Johanson, D. C., & White, T. D. (1979). A systematic assessment of early African hominids. *Science*, 203(4378), 321–330. <https://doi.org/10.1126/science.104384>.
- McDougall, I., Brown, F. H., & Fleagle, J. G. (2005). Stratigraphic placement and age of modern humans from Kibish, Ethiopia. *Nature*, 433(7027), 733–736. <https://doi.org/10.1038/nature03258>.
- Morgan, M. (2009). Complexities of Ethiopian sign language contact phenomena and implications for AAU. Ethiopia: French Centre for Ethiopian Studies, National Centre for Scientific Research in France. https://www.academia.edu/1230482/Complexities_of_Ethiopian_Sign_Language_Contact_Phenomena_and_Implications_for_AAU.
- Ridley, B., & Bridges, D. (1998). Secondary teacher education in Ethiopia: Ancient traditions and modern tensions. Norwich: School of Education and Professional Development University of East Anglia
- Robson, G. E. (1967). The caves of Sof Omar. *The Geographical Journal*, 133(3), 344–349.
- Roncevic, M. (2005). Ethnologue: Languages of the world. *Library Journal*, 130(14), 180–180.
- Russo, K. (2007). Psychiatrists see big need in Ethiopia. *The Boston Globe*. Bosten: Globe Newspaper Company. http://archive.boston.com/yourlife/health/men tal/articles/2007/04/23/psychiatrists_see_big_need_in_ethiopia/?page=1.
- Tekola, B., Baheretibeb, Y., Roth, I., Tilahun, D., Fekadu, A., Hanlon, C., & Hoekstra, R. A. (2016). Challenges and opportunities to improve autism services in low-income countries: Lessons from a situational analysis in Ethiopia. *Global Mental Health*, 3. <https://doi.org/10.1017/gmh.2016.17>.
- Tessema, K. A. (2007). Clinging to the managerial approach in implementing teacher education ‘reform’ tasks in Ethiopia. *International Journal of Progressive Education*, 3(3).
- WHO-AIMS Report on Mental Health System in Ethiopia. (2006). *WHO and ministry of health*. Ethiopia: Addis Ababa.

Ethnography

► [Qualitative Versus Quantitative Approaches](#)

Evaluation of a Transition to University Program for Students with Autism Spectrum Disorder

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Definition

The University of Bath Autism Summer School (UBASS) is a university transition program that introduces autistic students to life at university through the lens of work, rest, and play. The program aims to help students make a more informed decision when selecting and applying to universities in the UK.

Historical Background

Autism at University

In recent years, there has been an increase in autistic students' participation in postsecondary education. For example, in the UK, the widening participation agenda set up by the government aims to increase accessibility to postsecondary education for students from more disadvantaged backgrounds, as well as students with physical disabilities and other forms of developmental and specific learning disabilities, and has helped increase participation rate in higher education for autistic students from 7.4% in 2009/2010 to 10.7% in 2014/2015 (Connell-Smith and Hubble 2018). However, despite an overall increased participation rate, the transition to university can be an especially turbulent process for many autistic students.

Previous studies that have investigated students' mental health and well-being at university have found that up to 71% of autistic students reported symptoms of anxiety, 53% reported

loneliness, and 47% reported experiencing symptoms of depression (Gelbar et al. 2014), as well as increased rates of suicidal ideation and attempts (Jackson et al. 2018a). In the USA, it is estimated that only 35% of autistic students can complete their postsecondary education, which is lower than 38% of graduation rate for students with other disabilities and 51% of typically developing peers (Fisher and Hood 1987). Similarly, in the UK, fewer autistic students graduated from university with 2:1 or first-class honors degree (62.8%) compared to students with other forms of disabilities (66%) and typically developing peers (68.1%) (Lucas and James 2018). Furthermore, for many autistic students, university does not necessarily guarantee a safe passageway to employment following graduation. In the UK, within 6 months of completing their degree, autistic students are more than twice (18.5%) as likely to be unemployed compared to students with other disabilities (7.2%) and almost four times as likely to be unemployed compared to their typically developing peers (5.1%) (AGCAS Disability Task Group 2014).

With a growing number of autistic students facing the potential transition to postsecondary education, it is important for university stakeholders and transition services to provide informational, practical, and emotional support during the transition process to both the students and their family/caregivers and help make an informed decision as to whether or not university may be the right choice for each young person in question. In addition, the poor retention, graduation, and employment prospects for autistic students transitioning to university have called for more research to better understand the challenges associated with transitioning to postsecondary education (Jackson et al. 2018b), in order to facilitate universities and transition services to develop support plans and interventions to address students' unique needs and vulnerabilities when transitioning to university.

Understanding Students' Strengths and Vulnerabilities

Compared to their peers, autistic students often struggle in the complex university social setting. Although having poor social communication

skills is one of the core diagnostic criteria of autism spectrum disorder (ASD) (American Psychiatric Association 2013), many autistic adults and young people also experience co-occurring anxiety, with a lifetime prevalence rate for any disorders being between 27% and 79% (Hollocks et al. 2019; Kent and Simonoff 2017), as well as elevated rates of social anxiety, marked by having a persistent fear of being in one or more social situations that can lead to increased social avoidance (American Psychiatric Association 2013). Therefore, many autistic students may be less successful in developing a new social network at university and making friends, due to compounded social communication difficulties as a result of both autism and social anxiety (Adreon and Durocher 2007; Dipeolu et al. 2014).

Many autistic students also have restricted interests and preference for routine that make them less capable of adapting to changes in schedule when transitioning to university, resulting in additional distress (Dipeolu et al. 2014). In addition, many autistic individuals experience deficits in executive functioning skills (such as planning, organization, and working memory), which is associated with poor adaptive functioning, accounting for one's ability to initiate a range of activities essential for independent living ranging from socialization and communication skills such as initiating social contact with others to daily practical living such as cooking, self-care, and time and finance management (Pugliese et al. 2015; Sparrow et al. 2005). Furthermore, many students also experience sensory difficulties such as hypersensitivity to light and noise at university (Longtin 2014), and around 31% of autistic students have reported that their transition plans and accommodations at university have failed to address their sensory needs sufficiently (Sarrett 2018).

Another issue for many autistic students to consider when transitioning to university is related to diagnosis disclosure (Adreon and Durocher 2007). As students leave their supportive family environment when transitioning to university, learning to self-advocate can be very important for securing necessary resources and support services on campus to facilitate their independent living on campus (Giarelli and Fisher 2013). In addition, typically developing students

often show increased acceptance and perceived many socially awkward behaviors displayed by autistic students more positively when made aware of the other's autism diagnosis (Matthews et al. 2015), further highlighting possible social benefits of diagnosis disclosure among peers.

Finally, in contrast to many challenges in the social and daily living domain faced by autistic students at university, academic studies can often be an area of relative strength (Anderson et al. 2018; Geller and Greenberg 2009; Jackson et al. 2018a). University often provides autistic students with an opportunity to focus on their area of special interest, for which they often display a greater depth of knowledge compared to their typically developing peers (Geller and Greenberg 2009). Autistic students often also demonstrate more systematic way of thinking that is more logical and rational compared to their typically developing peers (Baron-Cohen et al. 2009), as well as paying more attention to detail (Happé 1997), which can be advantageous in subjects in the fields of science, technology, engineering, and mathematics (STEM). Helping autistic students to better recognize and utilize their strengths at university can help them become highly skilled professionals in these disciplines, which are particularly attractive for many employers in finance, engineering, and scientific industries and thus help increase their employment rate following graduation (Shattuck et al. 2012).

Interventions Supporting Student Transition to University

To date, interventions aimed at supporting autistic students to transition to university have largely included contacts (e.g., Shmulsky et al. 2015) and campus-based events that help provide information for students considering and seeking to apply to university (e.g., Retherford and Schreiber 2015) and peer support during early stages of the actual transition (e.g., Littlefield 2010). The content for the majority of pre-transitional programs are developed to address autistic students' concerns identified through qualitative studies, such as meeting coursework requirements, disability awareness on campus, social skills and social relationships, availability of practical and emotional support, organizational difficulties such as

time management and problem-solving skills, sensory challenges, and mental health concerns (Cai and Richdale 2016; Camarena and Sarigiani 2009; Mitchell and Beresford 2014; Van Hees et al. 2015; White et al. 2016).

Participation rate in postsecondary education for students who have either actively taken part in transition planning (61%), as well as those who have received instruction specifically focused on transition planning (74%), is almost two to three times greater compared to students who did not participate in transition planning (36%) or receive transition-planning instructions (24%) (Chiang et al. 2012; Wei et al. 2016), thus highlighting the importance of pre-transitional planning to increase higher education accessibility among autistic students.

In addition to increasing participation rates, developing a well-informed and useful pre-transition package to aid autistic students transitioning to university should also (1) serve as a platform for offering insights into university life to help autistic students make a more informed decision regarding university and (2) help students recognize their support needs which can be communicated to the relevant stakeholders and transition services and to help develop more personalized support plan for transitioning to university. The University of Bath Autism Summer School (UBASS) is an annual pre-transitional residential program running since 2012 that emphasizes on helping autistic students experience firsthand what university life is like, through various sessions structured around the themes of “work,” “rest,” and “play.” A summary of the UBASS program, as well as students’ feedback and evaluation from the first 5 years (2013–2017), has been published (Lei et al. 2018) and is summarized below, with future directions for research and practice highlighted.

Current Knowledge

University of Bath Autism Summer School (UBASS) Program

The Autism Summer School program is delivered across 3 days on a campus-based university, including two overnight stays in student

accommodation on campus. Students aged 16+ who hold a diagnosis of autism from a clinical professional seeking to learn more about university are eligible to apply using the online application system. Each year, around 20–30 students attend the Autism Summer School. Prior to arrival at the summer school, students are contacted and asked to complete some questionnaires surrounding their worries and concerns associated with transitioning to university, self-report levels of social anxiety, as well as self-reports on their social communication skills and level of autism identity. Parents are also contacted regarding students’ medical information, as well as additional dietary and other requirements. The questionnaires collected inform the program administrators to make reasonable adjustments for the students prior to their arrival. The summer school curriculum is developed to introduce students to sample different aspects of typical university life through the lens of “work,” “rest,” and “play” and to help promote self-care and well-being when at university. All sessions are delivered face-to-face in a group setting, with varying group sizes depending on nature of the session. A more detailed description of the sessions offered under each theme is shown in Table 1.

With the exception of two, all remaining sessions are prepared and delivered by academic and clinical staff in the Department of Psychology or staff from the disability and careers service at the campus university, who all have extensive experience working with autistic students at university. The two remaining sessions include (1) employment session delivered by an external employer who have an autism at work program and informs students about potential internship and career development opportunities alongside their studies, as well as what adjustments students can request to facilitate their application process, and (2) experience at university session delivered by autistic students who are either currently at or have recently graduated from either undergraduate or graduate studies. Clinical psychologists (both licensed and in training) delivered the anxiety-related psychoeducation sessions.

Beyond the structured timetabled sessions, many structured and unstructured social opportunities were also embedded into the program, to

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Table 1 Description of sessions offered at University of Bath Autism Summer School

Session	Description
Work	
Sample lecture	Experience teaching in lecture style, delivered in a lecture theater
Social and cultural aspects of student life	Learning more individual differences at university and adapting to a more socially and culturally diverse environment
Employment after university	Delivered by employer – focusing on how to navigate internship and seeking adjustments during application process. Careers service offers guidance on careers support on campus
Playing to the academic strengths associated with ASD	Understanding personal strengths associated with autism and how to best use them to improve academic studies and beyond
Rest	
Managing stress and anxiety	Psychoeducation session on how to identify stress and anxiety, adaptive coping mechanisms, and how to seek support
Social relationships	Addendum to psychoeducation session on managing stress and anxiety, with a more specific focus on managing social anxiety
Talking about your diagnosis	Workshop on understanding the pros and cons associated with disclosing diagnosis on campus, focusing on when best to disclose, who to disclose to, and how to disclose
General living and eating on campus (throughout the 3-day experience)	Experience firsthand what student accommodation is like and how to find and order food on campus
Play	
Speakers with ASD: An insider's view. Student support at university and beyond	Current autistic students and/or recent graduates sharing their personal experience of university life, as well as tips and advice
Clubs and societies at university	Showcasing how to enrich social life on campus through joining/starting a club or society of relevant interest
Sports and activities	Trialling out sport facilities on campus for leisure use
Visit town center and historical site of interest	Exploring life outside of university campus, informal socializing opportunity, and practice travelling to and from campus

help students overcome some of their social challenges and to help facilitate students to both initiate and take part in socializing with both typically developing students (the student ambassadors), other autistic peers, as well as faculty staff. To ensure students fully experience living on campus, during the summer school, all students stay in student accommodation on campus, which are single en suite bedrooms that are housed together in small flats, with six to seven people staying in each flat, which has a shared communal and kitchen area. Students also have catering and dine on campus for all of their meals, including ordering food for dinner from the student canteen. Throughout the summer school, students are also supported by “student ambassadors” who are current students at University of Bath trained to support widening participation and inclusion

activities on campus. In addition, a group of overnight supervisors who are either undergraduate or graduate students also stay in student accommodation along with students every evening and help address any issues that might emerge overnight. All student ambassadors and overnight supervisors have received a specific training session prior to the summer school to become more familiar with the needs of autistic students.

Feedback and Evaluation

Every year, students are asked to provide feedback evaluating their experience at the summer school, to help administrators review the sessions offered and amend the curriculum to best address students' needs based on feedback. Students' evaluation across the first 5 years of the program (2013–2017) was collected using mixed methods,

which focused on (1) changes in short-term outcomes in respect to worries and concerns about attending university; (2) student satisfaction ratings with the overall program; and (3) qualitative written feedback about the summer school. Overall, a total of 122 participants aged 16–30 years (mean age = 17.76 years) took part in the summer school from 2013 to 2017, 91 students were male (74%) and largely Caucasian (90.1%).

Following completion of the summer school program, students from 2013 to 2017 reported significant reductions in their worries and concerns associated with various aspects of transitioning to university, including navigating the social world, coping with leaving home, and meeting academic demands, daily living skills, and time management. The reduction in worries and concerns associated with a broad range of topics highlights that the Autism Summer School program was effective in improving students' understanding and perception of different aspects of university life. Students also rated the summer school to be very enjoyable and helpful, and the majority also viewed going to university more positively. Students were also asked to give qualitative feedback on what they found to be the most helpful at the Autism Summer School, as well as what they were most looking forward to about going to university.

Students found that both the content and delivery format of many of the lecture sessions were very helpful, including a range of topics such as psychoeducation about managing stress and anxiety, how to best utilize their strengths at university, hearing experiences of past autistic students, and the importance of diagnosis disclosure and accessing support at university, as well as learning more about clubs and societies. Some students also highlighted the usefulness of finding out more about university life firsthand, such as staying in university accommodation, eating in canteens, and visiting the university campus as well as seeing different departments related to their subject of interest. Finally, some students also commented on enjoying the chance to meet and socialize with other autistic students, as well as student ambassadors, which have helped to relieve some of their anxiety

around socializing with same-aged peers in a university setting.

The majority of students also gave a more positive outlook when asked about what they were most looking forward to about going to university, especially about having new social opportunities and meeting similar-minded people through their academic studies, accommodation, and joining new clubs and societies. A few students also commented on looking forward to meeting other autistic students at university, as well as making friends with other students during the summer school who will be attending the same university as themselves. Students were looking forward to the many new experiences that university life will bring, and that the summer school has helped eased some of their transition worries, though not all. Some students were particularly looking forward to the prospect of pursuing their academic subject at university to greater depth and their desire to perform well academically. Some students commented on looking forward to having greater independence at university and becoming more confident with greater range of social experiences, as well as looking forward to having their own routine and personal space at university. A few students commented on how the summer school has helped them recognize how to access support at university and from whom and the need to actively seek out help when needed. Finally, a few students commented on how the summer school has helped them make a more informed decision about whether university may be the right choice for them, and that they will be rethinking their university application, and possibly delaying entry until they felt ready to begin.

Future Directions

The University of Bath Autism Summer School (UBASS) provides an example of a pre-transitional program that helps autistic students to gain more first-person insights into life at university and has been shown to help students feel less worried and have a more positive outlook about starting university upon completion. The

social, nonsocial, and anxiety-related concerns raised by autistic students prior to transitioning to university can thus be addressed in a manner that is perceived to be helpful and enjoyable and serve as an important stepping stone for those who are seeking to apply, or transition, to university. However, one key limitation of the evaluation is that only short-term (i.e., immediately after completing the summer school) outcomes were evaluated, and no long-term impacts have been assessed, such as students' participation rate in postsecondary education following the summer school and transition outcomes. Using a longitudinal design with continued evaluation of transition to university experience for autistic students who have attended the summer school (or another pre-transition program) versus those who did not can further help evaluation of the summer school program and ask students to reflect in hindsight what they would have liked the summer school to include to facilitate their transition.

Overall, university stakeholders and transition services are recommended to offer more tailored support to autistic students who are transitioning into, through, and out of university life. Using the UBASS example, offering a structured pre-transition program that embed key components relating to daily life on campus such as staying in university accommodation as well as ordering food from the cafeteria can alleviate some of the worries autistic students have prior to applying to university (Lambe et al. 2018). Actively including the autistic students, as well as having clear communications between stakeholders and caregivers and family members to begin the university transition planning early, can give students the opportunity to have enough time to reflect on what their strengths and weaknesses are and to identify their personal support needs. It is also important that families and students are made aware of other alternatives beyond that of university studies and postsecondary education, as well as the possible career prospects following university studies, so that an informed decision can be made in the best interest of the young person in question. To support autistic students transitioning through university, ongoing support in the domains of socialization (e.g., autism social groups), as well

as daily living and academic studies (e.g., such as having a designated peer mentor or support buddy), should be carefully considered, and students should be made aware of the resources available to them. Furthermore, increasing autism awareness and training for both school teachers and university staff can also be important. Ensuring that school teachers are made aware of the support needs of autistic students and the importance of helping students decide when and how to disclose their diagnosis to seek the support they need when applying to university can help secure resources earlier on in the application process. Helping university staff to better understand autistic students' strengths and weaknesses, and in particular on how to tailor teaching and coursework assignments to help autistic students better utilize their strengths, can further help students realize their full potential at university. Finally, helping autistic students to start preparing for transitioning out of university and seeking employment earlier on during their university studies can also improve their postgraduation prospects. Educating students about the employment application process, such as how to seek adaptations from employers based on their needs and having more tailored interview skills training, can help students ease some of their worries associated with job seeking and help students better use resources available to them on campus to secure future employment.

See Also

- ▶ [College Transitional Programs](#)
- ▶ [Interventions to Support Transition to Adulthood for Individuals with Autism Spectrum Disorder](#)
- ▶ [Quality of Life for Transition-Age Youth with ASD](#)

References and Reading

- Adreon, D., & Durocher, J. S. (2007). Evaluating the college transition needs of individuals with high-functioning autism spectrum disorders. *Intervention in*

- School and Clinic*, 42(5), 271–279. <https://doi.org/10.1177/10534512070420050201>.
- AGCAS Disability Task Group. (2014). *What happens next? A report on the first destinations of 2014 disabled graduates*. Sheffield: AGCAS Disability Task Group.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. Arlington: American Psychiatric Association.
- Anderson, K. A., Sosnowy, C., Kuo, A. A., & Shattuck, P. T. (2018). Transition of individuals with autism to adulthood: A review of qualitative studies. *Pediatrics*, 141(Supplement 4), S318–S327. <https://doi.org/10.1542/peds.2016-43001>.
- Baron-Cohen, S., Ashwin, E., Ashwin, C., Tavassoli, T., & Chakrabarti, B. (2009). Talent in autism: Hyper-systemizing, hyper-attention to detail and sensory hypersensitivity. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 364(1522), 1377–1383. <https://doi.org/10.1098/rstb.2008.0337>.
- Cai, R. Y., & Richdale, A. L. (2016). Educational experiences and needs of higher education students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(1), 31–41. <https://doi.org/10.1007/s10803-015-2535-1>.
- Camarena, P. M., & Sarigiani, P. A. (2009). Postsecondary educational aspirations of high-functioning adolescents with autism spectrum disorders and their parents. *Focus on Autism and Other Developmental Disabilities*, 24(2), 115–128. <https://doi.org/10.1177/1088357609332675>.
- Chiang, H.-M., Cheung, Y. K., Hickson, L., Xiang, R., & Tsai, L. Y. (2012). Predictive factors of participation in postsecondary education for high school leavers with autism. *Journal of Autism and Developmental Disorders*, 42(5), 685–696. <https://doi.org/10.1007/s10803-011-1297-7>.
- Connell-Smith, A., & Hubble, S. (2018). *Widening participation strategy in higher education in England*. Briefing paper House of Commons Library. Retrieved from www.parliament.uk/commons-library
- Dipeolu, A. O., Storlie, C., & Johnson, C. (2014). Transition to college and students with high functioning autism spectrum disorder: Strategy considerations for school counselors. *Journal of School Counseling*, 12(11). Retrieved from <https://eric.ed.gov/?id=EJ1034736>.
- Fisher, S., & Hood, B. (1987). The stress of the transition to university: A longitudinal study of psychological disturbance, absent-mindedness and vulnerability to homesickness. *British Journal of Psychology*, 78(4), 425–441. <https://doi.org/10.1111/j.2044-8295.1987.tb02260.x>
- Gelbar, N. W., Smith, I., & Reichow, B. (2014). Systematic review of articles describing experience and supports of individuals with autism enrolled in college and university programs. *Journal of Autism and Developmental Disorders*, 44(10), 2593–2601. <https://doi.org/10.1007/s10803-014-2135-5>.
- Geller, L. L., & Greenberg, M. (2009). Managing the transition process from high school to college and beyond: Challenges for individuals, families, and society. *Social Work in Mental Health*, 8(1), 92–116. <https://doi.org/10.1080/15332980902932466>.
- Giarelli, E., & Fisher, K. (2013). Transition to community by adolescents with Asperger syndrome: Staying afloat in a sea change. *Disability and Health Journal*, 6(3), 227–235. <https://doi.org/10.1016/j.dhjo.2013.01.010>.
- Happé, F. G. E. (1997). Central coherence and theory of mind in autism: Reading homographs in context. *British Journal of Developmental Psychology*, 15(1), 1–12. <https://doi.org/10.1111/j.2044-835X.1997.tb00721.x>.
- Hollocks, M. J., Lerh, J. W., Magiati, I., Meiser-Stedman, R., & Brugha, T. S. (2019). Anxiety and depression in adults with autism spectrum disorder: A systematic review and meta-analysis. *Psychological Medicine*, 49(4), 559–572. <https://doi.org/10.1017/S0033291718002283>.
- Jackson, S. L. J., Hart, L., Brown, J. T., & Volkmar, F. R. (2018a). Brief report: Self-reported academic, social, and mental health experiences of post-secondary students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(3), 643–650. <https://doi.org/10.1007/s10803-017-3315-x>.
- Jackson, S. L. J., Hart, L., & Volkmar, F. R. (2018b). Preface: Special issue – College experiences for students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(3), 639–642. <https://doi.org/10.1007/s10803-018-3463-7>.
- Kent, R., & Simonoff, E. (2017). Chapter 2: Prevalence of anxiety in autism spectrum disorders. In C. M. Kerns, P. Renno, E. A. Storch, P. C. Kendall, & J. J. Wood (Eds.), *Anxiety in children and adolescents with autism spectrum disorder* (pp. 5–32). <https://doi.org/10.1016/B978-0-12-805122-1.00002-8>.
- Lambe, S., Russell, A., Butler, C., Fletcher, S., Ashwin, C., & Brosnan, M. (2018). Autism and the transition to university from the student perspective. *Autism*, 23, 1531–1541. <https://doi.org/10.1177/1362361318803935>.
- Lei, J., Calley, S., Brosnan, M., Ashwin, C., & Russell, A. (2018). Evaluation of a transition to university programme for students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-018-3776-6>.
- Littlefield, C. (2010). *Students with Asperger's syndrome transitioning to postsecondary education: What are the common issues?* Rochester: Rochester Institute of Technology.
- Longtin, S. E. (2014). Using the college infrastructure to support students on the autism spectrum. *Journal of Postsecondary Education and Disability*, 27(1), 63–72.
- Lucas, R., & James, A. I. (2018). An evaluation of specialist mentoring for university students with autism spectrum disorders and mental health conditions. *Journal of Autism and Developmental Disorders*, 48(3), 694–707. <https://doi.org/10.1007/s10803-017-3303-1>.
- Matthews, N. L., Ly, A. R., & Goldberg, W. A. (2015). College students' perceptions of peers with autism spectrum disorder. *Journal of Autism and*

- Developmental Disorders*, 45(1), 90–99. <https://doi.org/10.1007/s10803-014-2195-6>.
- Mitchell, W., & Beresford, B. (2014). Young people with high-functioning autism and Asperger's syndrome planning for and anticipating the move to college: What supports a positive transition? *British Journal of Special Education*, 41(2), 151–171. <https://doi.org/10.1111/1467-8578.12064>.
- Pugliese, C. E., Anthony, L., Strang, J. F., Dudley, K., Wallace, G. L., & Kenworthy, L. (2015). Increasing adaptive behavior skill deficits from childhood to adolescence in autism spectrum disorder: Role of executive function. *Journal of Autism and Developmental Disorders*, 45(6), 1579–1587. <https://doi.org/10.1007/s10803-014-2309-1>.
- Retherford, K. S., & Schreiber, L. R. (2015). Camp campus: College preparation for adolescents and young adults with high-functioning autism, Asperger syndrome, and other social communication disorders. *Topics in Language Disorders*, 35(4), 362–385. <https://doi.org/10.1097/TLD.0000000000000070>.
- Sarrett, J. C. (2018). Autism and accommodations in higher education: Insights from the autism community. *Journal of Autism and Developmental Disorders*, 48(3), 679–693. <https://doi.org/10.1007/s10803-017-3353-4>.
- Shattuck, P. T., Narendorf, S. C., Cooper, B., Sterzing, P. R., Wagner, M., & Taylor, J. L. (2012). Postsecondary education and employment among youth with an autism spectrum disorder. *Pediatrics*, 129(6), 1042–1049. <https://doi.org/10.1542/peds.2011-2864>.
- Shmulsky, S., Gobbo, K., & Donahue, A. (2015). Groundwork for success: A college transition program for students with ASD. *Journal of Postsecondary Education and Disability*, 28(2), 235–241.
- Sparrow, S. S., Ballard, D. H., Cicchetti, D. V., Harris, P. L., & Doll, E. A. (2005). *Vineland adaptive behavior scales* (2nd ed.). Circle Pines: American Guidance Service.
- Van Hees, V., Moyson, T., & Roeyers, H. (2015). Higher education experiences of students with autism spectrum disorder: Challenges, benefits and support needs. *Journal of Autism and Developmental Disorders*, 45(6), 1673–1688. <https://doi.org/10.1007/s10803-014-2324-2>.
- Wei, X., Wagner, M., Hudson, L., Yu, J. W., & Javitz, H. (2016). The effect of transition planning participation and goal-setting on college enrollment among youth with autism spectrum disorders. *Remedial and Special Education*, 37(1), 3–14. <https://doi.org/10.1177/0741932515581495>.
- White, S. W., Elias, R., Salinas, C. E., Capriola, N., Conner, C. M., Asselin, S. B., ... Getzel, E. E. (2016). Students with autism spectrum disorder in college: Results from a preliminary mixed methods needs analysis. *Research in Developmental Disabilities*, 56, 29–40. <https://doi.org/10.1016/j.ridd.2016.05.010>

Evaluation of Sensory Processing

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Synonyms

ESP

Definition

In 2000, Diane Parham and Cheryl Ecker formally shared their work called the *Evaluation of Sensory Processing* (ESP). This parent questionnaire, whose development was originally initiated by LaCroix in 1993, was intended to identify behaviors thought to be indicative of sensory processing problems. The unpublished test, produced at the University of Southern California, was ultimately included in the 2002 (Parham and Ecker 2002) text titled *Sensory Integration: Theory and Practice* (2nd ed.) for occupational therapy clinician's to utilize in practice. After many years of research on the utility of the tool, the authors signed an agreement with Western Psychological Services (WPS) to complete the standardization and normative sampling. Simultaneously, WPS was also working with a group of researchers currently undertaking the task of developing a similar testing instrument for use within the school environment called the *School Assessment of Sensory Integration* (SASI; Miller Kuhaneck, Henry, & Glennon, unpublished). The two assessment tools, one for parents and one for educational staff, were ultimately combined for standardization to create the *Sensory Processing Measure* (Parham et al. 2007) and the *Sensory Processing Measure - Preschool* (Miller Kuhaneck et al. 2010).

See Also

► [Sensory Processing Measure](#)

References and Reading

- LaCroix, J. E. (1993). *A study of content validity using the sensory history questionnaire*. Unpublished master's thesis, University of Southern California, Los Angeles.
- Miller Kuhaneck, H., Ecker, C. E., Parham, L. D., Henry, D. A., & Glennon, T. J. (2010). *Sensory processing measure-preschool (SPM-P): Manual*. Los Angeles: Western Psychological Services.
- Parham, L. D., & Ecker, C. (2000). *Evaluation of sensory processing*. Unpublished test, University of Southern California, Los Angeles.
- Parham, L. D., & Ecker, C. (2002). Evaluation of sensory processing. In A. Bundy, S. Lane, & E. Murray's (Eds.), *Sensory integration: Theory and practice* (2nd ed.). Philadelphia: F A. Davis.
- Parham, L. D., Ecker, C., Miller Kuhaneck, H., Henry, D. A., & Glennon, T. J. (2007). *Sensory processing measure (SPM): Manual*. Los Angeles: Western Psychological Services.

Event-Related Functional Magnetic Resonance Imaging (fMRI)

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Synonyms

[Blood-oxygen-level-dependent \(BOLD\) contrast; fMRI; Functional MRI](#)

Definition

Functional magnetic resonance imaging (fMRI) is a brain imaging technique that uses a standard magnetic resonance imaging scanner, a high rate of image acquisition, and specialized pulse sequences to measure localized brain activity. In

most cases, fMRI takes advantage of the endogenous blood-oxygen-level-dependent (BOLD) contrast to image brain activity without the use of ionizing radiation. As such, this technique is ideal for studying the developing brain in children. The technique is now the dominant technique for the study of brain function and its disruption in autism.

See Also

► [Functional MRI](#)

References and Reading

- Malisza, K. L., Clancy, C., Shiloff, D., Foreman, D., Holden, J., Jones, C., et al. (2011). Functional evaluation of hidden figures object analysis in children with autistic disorder. *Journal of Autism and Developmental Disorders*, 41(1), 13–22.
- Pitskel, N. B., Bolling, D. Z., Hudac, C. M., Lantz, S. D., Minshew, N. J., & Pelphrey, K. A. (2011). Brain mechanisms for processing direct and averted gaze in individuals with autism. *Journal of Autism and Developmental Disorders*, 41(12), 1686–1693.

Event-Related Potential

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Synonyms

[Evoked potentials](#)

Definition

An event-related potential is an electrical brain response, recorded via electroencephalography,

time-locked to a particular stimulus or event. External visual and auditory stimuli are commonly used in ERP research. In order to isolate the response to the particular stimulus being studied, multiple trials of responses to similar stimuli are averaged together to produce a composite waveform, ultimately reducing the potential influence of extraneous factors.

See Also

- [Electroencephalography](#)
- [Evoked Potentials](#)
- [Mismatch Negativity](#)

References and Reading

- Coles, M. G. H., & Rugg, M. D. (1996). Event-related brain potentials: An introduction. *Electrophysiology of Mind*, 1(6), 1–27.
- Fabiani, M., Gratton, G., & Federmeier, K. D. (2007). Event-related brain potentials: Methods, theory, and applications. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 85–119). Cambridge, UK: Cambridge University Press.
- Luck, S. J. (2005). *An introduction to the event-related potential technique*. Cambridge, MA: MIT Press.
- Otten, L. J., & Rugg, M. D. (2005). Interpreting event-related brain potentials. In T. C. Handy (Ed.), *Event-related potentials: A methods handbook* (pp. 229–259). Cambridge, MA: MIT Press.

Event-Related Potential (ERP)

- [Evoked Potentials](#)

Everyday Memory

- [Prospective Memory in Autism Spectrum Disorder](#)

Evoked Potentials

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Synonyms

[Brainstem Evoked Response \(BER\)](#); [Event-Related Potential \(ERP\)](#); [Evoked Response](#); [Visual Evoked Potential \(VEP\)](#)

Definition

Evoked potentials refer to electrical activity generated by a biological system in response to an event. Events triggering electrical potentials classically include responses in the visual system, auditory system, motor system, and nervous system.

See Also

- [Electroencephalography](#)
- [Event-Related Potential](#)

References and Reading

- Andreassi, J. L. (2007). *Psychophysiology: Human behavior & physiological response* (5th ed.). Mahwah: Lawrence Erlbaum Associates.
- Coles, M. G. H., & Rugg, M. D. (1996). Event-related brain potentials: An introduction. *Electrophysiology of Mind*, 1(6), 1–27.
- Fabiani, M., Gratton, G., & Federmeier, K. D. (2007). Event-related brain potentials: Methods, theory, and applications. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 85–119). Cambridge, UK: Cambridge University Press.
- Otten, L. J., & Rugg, M. D. (2005). Interpreting event-related brain potentials. In T. C. Handy (Ed.), *Event-related potentials: A methods handbook* (pp. 229–259). Cambridge, MA: MIT Press.

Evoked Response

- ▶ [Evoked Potentials](#)

EVT-2

- ▶ [Expressive Vocabulary Test II](#)

Exactingness

- ▶ [Perfectionism](#)

Example, Demonstration

- ▶ [Modeling](#)

Exceptionality

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Synonyms

[Abnormality](#); [Atypical](#); [Disability](#); [Extraordinary](#)

Definition

Having some degree of difference from an established norm in a specific skill area. Exceptionalities may include characteristics including disabilities as well as special gifts and talents. An individual with a noted exceptionality may present a need for special education or supports in relation to a disabling condition or due to special talents or abilities such as mental giftedness. Individuals with autism spectrum disorder may present

exceptionalities that are below what would be normally expected in some areas of function and may also present exceptional abilities above the norm in others. The term “exceptionality” is often used interchangeably with the terms “disability” and “special need.” Many state statutes use the term “exceptionality” to denote categories of disability. Autism is one example of a category of exceptionality.

See Also

- ▶ [Disability](#)
- ▶ [Giftedness](#)
- ▶ [Special Needs](#)

References and Reading

Assistance to States for the Education of Children with Disabilities and Preschool Grants for Children with Disabilities; Final Rule, 2004, 4000-01-U Department of Education 34 CFR Parts 300 and 301, Part II.
Individuals with Disabilities Education Act of 2004, 20 U.S. C., et. seq.

Executive Function (EF)

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Definition

“Executive functions” is a broad term used to describe the set of cognitive processes required to prepare for and execute goal-directed behaviors. Various theoretical models propose that executive functions are executed by slightly different component processes. However, in general terms, these components are thought to include:

- Goal (or rule) representation, which also may be referred to as working memory
- Inhibition

- Cognitive flexibility, which also may be referred to as set shifting or task switching
- Planning

Some also consider “higher level” cognitive processes like problem solving and abstract reasoning to be forms of executive functioning. Other terms such as controlled (versus automatic) processing; selective attention; and supervisory attention, which originate in different theoretical models, may be used to describe the properties of executive functions.

Historical Background

Impairments in executive functions are among the most consistently reported deficits in individuals with autism spectrum disorders or “ASD” (see Hill 2004; Ozonoff et al. 2006; Pennington and Ozonoff 1996). Many early studies of executive functions in autism used the Wisconsin Card Sorting Test (WCST), which requires participants to figure out a sorting rule, derived from one of the cards’ three dimensions (color, quantity, or design), based on minimal feedback. Once the participant has demonstrated that they understand the sorting rule by executing it properly multiple times, the rule unexpectedly changes. Studies using the WCST find that persons with autism tend to perseverate on incorrect responses (i.e., are unable to stop sorting using the old rule when it is no longer correct) more than matched controls. Interestingly, these impairments appear to be reduced when the task is administered on a computer.

Given that patients with focal frontal lobe lesions have difficulty in planning future actions and in inhibiting habitual responses, executive functions traditionally are associated with the pre-frontal cortex (PFC). Many clinical neuropsychology assessment batteries and tasks like the WCST are premised on this assumption and are designed to test what are believed to be the specific components of executive functioning based on studies of patients with brain lesions. However, more contemporary research suggests that executive

functions cannot be localized to one brain region or process.

Executive functions deficits are not specific to autism spectrum disorders. They are present in many other forms of developmental psychopathology including attention deficit hyperactivity disorder (ADHD). However, it is important to study executive functions in persons with ASD because longitudinal studies show that executive functions deficits have a parallel influence across social, language, and other domains of functioning. Furthermore, they are related to cognitive abilities that have a very strong relationship with long-term outcomes, and can serve as a treatment target.

Current Knowledge

Consistent with findings about perseveration on the WCST in persons with ASD, the most common perspective on executive functions deficits is that they are related to cognitive flexibility. More specifically, many studies have shown that individuals with ASDs have difficulty in shifting their frame of reference in thinking about problems (shifting cognitive set); in shifting how they conceptualize options that are not presented in the immediate environment (extra-dimensional set shifting); in shifting the focus of their visual attention; in shifting their attention between sensory modalities, like seeing and hearing; and in shifting between sets of rules they have learned.

Another component of executive function that is consistently found to be impaired is planning. This commonly is measured using the Tower of Hanoi or the Tower of London tasks which require participants to move disks of various sizes across three pegs. They are given a starting position and a picture of an ending position and must reproduce the ending position using the fewest moves. The task grows progressively harder over trials, and task rules prevent self-correction. The Tower task requires that the participants think through (or plan) disk moves in their heads before executing them. This is a relatively difficult task, and some have criticized the use of this type of task because it is unclear exactly what it assesses.

Several executive functions are thought to be intact in ASDs. The first of these is inhibition, which once was thought to differentiate persons with ASDs from those with ADHD. However, a growing body of research suggests that individuals with ASDs also have difficulty inhibiting prepotent response tendencies. It is unclear why there has been this shift in the literature. Some have suggested that there have been changes in diagnostic conventions since the early 1990s and that individuals formerly diagnosed with ADHD now are diagnosed with ASDs – a trend referred to as diagnostic substitution. Another possibility is that tasks that have been used to assess inhibition are not directly comparable and that generally harder tasks produce greater deficits. A second executive function traditionally thought to be intact is working memory. Here too, results have become less clear as various versions of the task have been used.

Following one study that showed that high functioning persons with autism perform better on the intelligence tests that involve more visuospatial versus verbal reasoning – the Raven's Progressive Matrices test (Dawson et al. 2007), there now have been several that show that aspects of conditional and analogical reasoning are relatively preserved in persons with ASDs.

As all parents and clinicians know, the contention that executive functions deficits create problems for persons with ASDs in everyday life would appear to be true. Individuals with ASDs have difficulty maintaining reciprocal social relationships, having two-sided conversations, transitioning between activities, and disengaging from idiosyncratic interests. They also have difficulty inhibiting old behaviors in favor of new, more appropriate ones. At school, they struggle to stay organized. They lose or forget to turn in homework. They have difficulty reading for meaning and writing clearly. They may be unable to organize information well enough to correctly answer math story problems. However, despite the fact that deficits in executive functions would appear to map directly related to these inflexible everyday behaviors, it has been challenging to demonstrate this link empirically using clinical neuropsychology measures (see Geurts et al.

2009). It has been suggested that we need better measures that tap “real-world” inflexible behaviors.

Another focus of current research is on how executive functions develop. Adolescent development of executive functions has become an area of active scrutiny given that profound brain changes occur during normative adolescent development, leading to maturation, and providing a window of opportunity for intervention. During adolescence, reductions in cortical gray matter, and increases in white matter volume, result in the refined calibration of the excitatory-inhibitory balance in the PFC, and the strengthening of networks governing cognitive processing. In typical development, most executive functions mature by age 15, although reaction times and performance on more complex tasks and combinations of tasks improve into the 20s.

Future Directions

Fortunately, the advent of new technologies including functional magnetic resonance imaging is helping to advance what we know about brain functioning beyond what could be learned by lesion models. “Cognitive control” is a term evolving in the field of cognitive neuroscience to describe the cognitive processes that traditionally have been thought of as executive functions (Miller and Cohen 2001). The newer cognitive control-based model of PFC function suggests that (1) the PFC is specialized for the representation and maintenance of context information; (2) that context information is maintained in the PFC as a pattern of neural activity; and (3) that context representations mediate cognitive control through interactions that modulate the flow of information in other brain systems that more directly support task performance. Proper functioning of this system is required for (a) effective allocation of attention, (b) inhibition of irrelevant responses, (c) appropriate shifting of frame of reference, (d) relating information appropriately over time and space, and (e) adjusting behavior in relation to the evolving environment. For example, when Americans visit London and attempt to

cross the street, cognitive control must be engaged to avoid the customary practice of looking to the left for oncoming traffic, in favor of looking to the right. In sum, cognitive control must be engaged when overcoming habitual responses, ignoring irrelevant stimuli, or transforming representations. Cognitive control is not required to perform simple or automatic behaviors, but must be engaged to guide action in novel, difficult, or rapidly changing conditions. This is especially important when there is strong competition between the potential responses (such as choosing between the ingrained habit of looking left versus facing the prospect of being run over!).

Specific brain regions thought to be involved in the application of cognitive control include the dorsolateral prefrontal cortex (DLPFC), medial frontal cortex (including the anterior cingulate cortex), and parietal cortex. In the cognitive control model, the DLPFC is believed to maintain appropriate context for action. The anterior cingulate cortex is thought to function as part of a “control loop.” It detects response conflict and signals the DLPFC to allocate more control-related resources. The parietal cortex is activated when it is necessary to switch attentional focus. It also is thought to act as a repository of learned stimulus-response associations from which the DLPFC “selects” the appropriate response. In another emerging model, a hierarchy of brain regions is thought to implement cognitive control. The representation of the highest conceptual level of a task is thought to rely on the anterior or rostralateral PFC.

In conclusion, it is an exciting time for executive functions research, and more work in this area will help to advance the understanding of ASDs. Directions for future executive functions research include:

1. The development of models and measures that better capture what happens in the brain when we are engaged in goal-directed activities. This will include measures of connectivity between different brain regions and neural circuits.
2. Studies that compare these models and measures across other neurodevelopmental disorders. This type of strategy may help us better

understand the genes involved in autism (and other disorders involving similar problems).

3. Studies of executive functions/cognitive control in animals including rodents and non-human primates. These studies can help us tease apart the pathophysiology of disorders. Animal models also can be used to test promising drug treatments.
4. More ecologically valid measures might help in forging associations between the observed day-to-day behavior and models and measures of goal-directed behavior.
5. Most experimental research has used data analytic models that focus on mean group differences. We need studies that consider the role of individual differences in areas such as temperament and personality, stress responsiveness, and motivation in studies of executive functions.
6. Clinical trials of interventions that help us to remediate executive functions deficits.
7. Studies that examine the development of executive functions/cognitive control through the life span.

See Also

- [Attention](#)
- [Memory](#)

References and Reading

- Dawson, M., Soulières, I., Gernsbacher, M. A., & Mottron, L. (2007). The level and nature of autistic intelligence. *Psychological Science*, 18, 657–662.
- Geurts, H., Corbett, B., & Solomon, M. (2009). The paradox of cognitive flexibility in autism spectrum disorders. *Trends in Cognitive Science*, 13(2), 74–82.
- Hill, E. L. (2004). Executive dysfunction in autism. *Trends in Cognitive Science*, 8(1), 26–32.
- Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, 24, 167–202.
- Ozonoff, S. J., Pennington, B. F., & Solomon, M. (2006). Neuropsychological perspectives on developmental psychopathology. In D. Cicchetti (Ed.), *Developmental psychopathology* (2nd ed.). New York: Wiley.
- Pennington, B. F., & Ozonoff, S. J. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 37(1), 51–87.

Executive Functioning and Martial Arts Training in Children with Autism Spectrum Disorder

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Definition

Executive functions (EFs) are a host of interrelated, cognitive processes that drive goal-directed cognitive, emotional, and behavioral functioning. The umbrella term of EFs includes the three core processes of behavioral inhibition (e.g., impulse control), working memory (e.g., holding information in mind while mentally manipulating it), and cognitive flexibility (e.g., transitioning from thinking about one concept to another). Higher-order EFs include fluency, planning, and problem solving/reasoning. Challenges in EFs are common in children with neurodevelopmental disorders such as Autism Spectrum Disorder (ASD). Compared to their typically-developing (TD) peers, children with ASD demonstrate poorer EFs, particularly poorer planning abilities and cognitive flexibility (Hill 2004; Kenworthy et al. 2008).

Different EFs emerge and improve as children age. For example, the capacity to engage in cognitive switching during multidimensional tasks emerges in middle childhood and continues to improve thereafter (Anderson 2002). EFs also appear to be malleable through effortful practice and interventions. A variety of physical and cognitive activities has been proposed to improve EFs in both typical and neurodiverse samples. One of these activities is the practice of martial arts. Martial arts also is an umbrella term and is defined as systemized traditions of combat that are expressed through movement and used for self-

defense, sport/competition, entertainment/recreation, preservation of cultural traditions, and for physical fitness and mental development. Martial arts styles vary greatly in their historical/contemporary contexts, theoretical/practical application, use of weaponry, degree of physical contact, and potential health and fitness benefits.

Our recent research was directed at testing a particular form of martial arts as an intervention to help improve EFs among middle-childhood-aged children with ASD (Phung and Goldberg 2019). In our study, a traditional approach, focused on character development in addition to physical growth, to Mixed Martial Arts (MMA) was implemented as a potential executive functioning intervention for children with ASD. Children were randomly assigned either to attend a set of 26 MMA sessions over 13 weeks (i.e., the intervention) or to be in a waitlist control (WLC) group. Adult instructors and TD peer buddies were trained in how to interact with children with ASD to help deliver the intervention. The MMA intervention included physical movements (e.g., punches and kicks) that increased in complexity (e.g., number of steps) over the course of the program. At each session, the main activity was practiced back-and-forth with TD peer buddies. At the end of the intervention, the MMA group demonstrated improved objective and parent-reported EFs, whereas the WLC group did not show EF improvements between pre- and post-test assessments. Thus, support was found for the effectiveness of a structured, peer-mediated, mixed-martial arts intervention for middle-childhood-aged children with ASD. Our intervention study was the first to demonstrate the benefits of MMA for EFs in children with ASD (Phung 2017; Phung and Goldberg 2019). The authors called for replication with a larger sample and greater balance of gender between the MMA and WLC groups.

Historical Background

Deficits in executive functions (EFs) are common among individuals with ASD and other neurodevelopmental disorders (Diamond 2013;

Hill 2004; Ozonoff and Jensen 1999). Boys appear especially vulnerable to executive functioning problems compared to girls (Diamond 2002), which may be related to the higher incidence of ASD among males compared to females (Baio et al. 2018).

Executive dysfunctions are common in children with ASD but are not part of the core symptomology of ASD. However, EFs seem to be associated with the presence of restricted and repetitive patterns of behavior (RRBs), which are fundamental to the diagnostic criteria of ASD (American Psychiatric Association 2013). RRBs are broad patterns of behaviors, actions, and thoughts that may include intense preoccupations with interests (e.g., overly specific knowledge about a movie that makes it difficult to talk about nonpreferred topics), rigid and non-functional routines (e.g., completing sequences in a specific order), repetitive motor movements (e.g., hand flapping), and nonfunctional use or close examination of parts of objects (e.g., repeated spinning the wheels of a toy car) (Kim and Lord 2010). Severe RRBs hinder adaptive functioning as they cause perseveration on nonfunctional details and limit the ability to generalize or mentally shift previous knowledge to accommodate new information.

Research concerning the consistency of the connection between RRBs and EFs is mixed and the mechanisms linking these phenomena are unclear. For example, one study found that RRBs were associated with the three core EFs of behavioral inhibition, working memory, and cognitive flexibility (Lopez et al. 2005); another study reported that RRBs were only associated with behavioral inhibition (Boyd et al. 2009); and yet another study observed the link between RRBs and EFs only for some measures of RRBs (South et al. 2007). Despite evidence that suggests that executive functioning deficits are linked in part to the diagnostic criteria of RRBs, RRBs in children with ASD manifest at early ages (Richler et al. 2007; Wolff et al. 2014) and EFs are slow to develop in all children. In fact, EFs are the last of the cognitive abilities to reach full maturity, continuing to develop throughout adolescence and into early adulthood. Given its slow

developmental trajectory, researchers have suggested that EFs are malleable and can be improved with effortful practice. An important developmental period in the growth and plasticity of EFs is middle childhood (Best and Miller 2010).

EF abilities appear to worsen with age in ASD samples: Older children with ASD show greater EF problems than do younger children with ASD when compared to typically-developing (TD) peers (Rosenthal et al. 2013). Age has thus emerged as a salient factor when considering interventions to improve EFs. To date, studies examining EFs in middle childhood have focused on interventions to help boost EFs in TD and ASD groups. For example, one study using martial arts training with TD children found that fourth- and fifth-graders benefited more from the EF intervention in terms of improved inhibitory control and working memory compared to the kindergarten and first-graders, who did not show any improvement after receiving the intervention (Lakes and Hoyt 2004). Similarly, a computer-training study with 4- and 6-year-old TD children found no improvements in inhibitory control and working memory; the authors suggested that the children could have been too young (Thorell et al. 2009) (see Diamond and Lee 2011 for a review of EF intervention studies). Thus, middle childhood seems to be an optimal period for these sorts of interventions.

Because children with ASD face difficulties with executive functioning and given that studies have been conducted using martial arts to improve EFs in children with other vulnerable developmental profiles (e.g., low SES; Lakes et al. 2013), we thought that children with ASD might benefit from martial arts training as well. Until recently, few studies had examined the potential of martial arts to benefit EFs in children with ASD. The initial ventures were conducted by Bahrami and colleagues (Bahrami et al. 2012), which focused on the martial art of Kata, and Chan et al. (2013), which focused on the martial art of Nei Yang Gong. The results of these studies will be discussed in the next section.

Our recent research was the first to select a Mixed Martial Arts (MMA) style of martial arts

to implement as an EF intervention to help improve EFs in children with ASD (Phung and Goldberg 2019). A MMA approach was selected for the following reasons, all of which have the potential to specifically target EFs in children with ASD. First, MMA grew in part from Jeet Kune Do, a Chinese martial art that was developed by consolidating elements and techniques from over 20 different martial arts. The goal of Jeet Kune Do was to have different tools that can be used to adapt to different contexts (Lee 1975). Because children with ASD fall widely on the autism spectrum in terms of diverse strengths and weaknesses, an adapted MMA intervention could accommodate to each individual child's abilities by providing various "tools" for the child to use. Second, an adapted MMA approach could help children learn to use these tools across different contexts, which provide the opportunity for the generalization of the skills, thus practicing the EF of cognitive flexibility. Third, compared to some other martial arts styles that are high in intensity and pace, MMA is a relatively low- to moderate-intensity exercise, which past research has indicated could reduce stereotypic behaviors (a form of RRBs) in children with ASD. Specifically, research suggests that low-intensity exercise may help with improving the EF of behavioral inhibition for stereotypic behaviors (Schmitz et al. 2017). Fourth, the MMA style, when taught in a "traditional" rather than "modern" approach, emphasizes self-control of behaviors and emotions rather than physical conditioning and competition (Trulson 1986), which are both important elements of EFs. Lastly, the MMA style involves repeated practice and was designed to increasingly challenge EFs over the course of time, both of which are important elements of effective EF interventions as recommended by Diamond (2012). Considering these reasons together, we selected a traditional approach to the MMA style as our EF intervention for children with ASD given its ability to accommodate to the diversity of skills among children with ASD, the increased likelihood of skill generalization across contexts, its low- to moderate-intensity (which has been recommended for children with ASD), its use of a traditional approach that emphasizes important

elements of EFs, and the learning of techniques that increase complexity over time, thus further challenging and strengthening EFs.

Current Knowledge

In a review paper that compared different exercise interventions (e.g., yoga/dance, swimming, jogging), martial arts training emerged as one of the most effective activities to improve behavioral outcomes in children with ASD (Bremer et al. 2016). To date, only three known studies (including ours) have used martial arts to address EF challenges in children with ASD. In one study, Bahrami et al. (2012) indirectly examined EFs by asking for caregiver reports of stereotypic behaviors (e.g., RRBs) before and after the implementation of a traditional Kata (Japanese forms) program with children and adolescents with ASD. Compared to a no-exercise control group that showed no improvement, the participants who received the Kata training had reduced stereotypic behaviors at post-test, and the benefits extended to caregiver reports at one-month follow-up as well. Because this study found lessened stereotypic behaviors following a martial arts intervention and given the partially supported links between RRBs and EFs, we thought a martial arts intervention might benefit EFs as well.

Another study directly examined one aspect of EFs, self-control (i.e., behavioral inhibition), before and after one month of training in Nei Yang Gong (NYG), a Chinese martial art (Chan et al. 2013). Compared to a control group of children and adolescents with ASD that received Progressive Muscle Relaxation (PMR) training, the NYG group had higher parent-reported self-control and lower daily behavior problems. Furthermore, the NYG group demonstrated increased EEG brain activity during a lab-based EF task compared to baseline, whereas the PMR control group showed no difference. Although participants only received 8 h of martial arts training over the course of one month, the parent-reported and objective EEG findings demonstrated that martial arts could be effective

in improving the self-control aspect of EFs; we wanted to examine whether a martial arts intervention could yield benefits for the other EFs as well.

As mentioned above, our recent study implemented a Mixed Martial Arts (MMA) intervention with middle-childhood-aged children with ASD (aged 8–11 years; 82.4% boys). At pre- and post-test, parents completed the Behavior Rating Inventory of Executive Functions – 2nd edition (BRIEF-2; Gioia et al. 2000) to report on their child's executive functioning. The BRIEF-2 has three indexes of executive functioning: behavior regulation index, emotion regulation index, and cognitive regulation index. Together, these three indexes yield the Global Executive Composite.

During each of the two laboratory visits, children completed a computerized EF task, the Hearts & Flowers test, which used a touch-screen laptop computer. The Hearts & Flowers test measured the three core EFs: behavioral inhibition, working memory, and cognitive flexibility (Davidson et al. 2006; Diamond et al. 2007). The Hearts & Flowers test consisted of one block of same-side stimuli (congruent block), one block of opposite-side stimuli (incongruent block), and one block of trials with both same- and opposite side stimuli (mixed block) (for description of the blocks, see Wright and Diamond 2014). Executive functioning performance on the Hearts & Flowers test was measured for two dependent variables, accuracy and response time (in milliseconds).

After completing the pre-test assessments, children were then randomly assigned to either begin the martial arts intervention in the MMA group, or to not receive any martial arts training in the WLC group. Children in the MMA group then began a 26-session (2 times per week), 13-week MMA intervention, which took place at a local martial arts studio. The lead author collaborated with the studio's instructors to develop the content of the curriculum, which increased in complexity over the course of the 26-session program. For example, in the first third of the program (Weeks #1 through 4) the instructors taught movements (striking, i.e., punches and kicks) that were made

up of 2- to 3-step sequences. In the second third of the program (Weeks #5 through 8), the instructors taught movements (grappling) that were now 4- to 9-step sequences. In the final third of the program (Weeks #9 through 13), instructors taught movements (striking and grappling) that consisted of 4- to 13-step sequences. Over the course of the 26-session program, the movements not only grew in complexity, but children were also taught to generalize previously learned skills to build upon the new material. Unique to the program was the inclusion of peer buddies, typically-developing (TD) children who were students in other classes at the studio. Peer buddies volunteered to assist in the program and acted as training partners with the children with ASD.

In terms of the structure of each class, each session began with the children lining up and bowing-in to pay respects to the instructor. Next, the instructor led children in a brief meditation and breathing activity, focusing on the goals of the day. Warm-up activities that included activities like jogging, footwork, stretching, and jumping were followed by the main activity of the specific class session. During the main activity, the instructor taught the martial arts movements (e.g., striking, grappling) and children with ASD were paired with TD peer buddies to then practice the taught movements. As mentioned previously, the curriculum for the main activity increased in complexity over the course of the 26-session program. Each class ended with a social game and paying respects to the instructor and classmates. At the end of the intervention, children in both the MMA and WLC groups returned to the lab testing space for post-test assessments using the parent-reported BRIEF-2 (Gioia et al. 2000) and child performance on the computer-based Hearts & Flowers test (Davidson et al. 2006).

In general, results from the study found that both parent-reported and computer-based assessments of EFs improved with the MMA group, but not with the WLC group. Specifically, for parent-reported child EF on the BRIEF-2 questionnaire, children in the MMA group had improved EFs on the behavior regulation and emotion regulation indexes but not on the

cognitive regulation index, compared to the reports from parents of children in the WLC group. Parents of children in the MMA group also reported improved overall EF on the Global Executive Composite. In terms of computer-based assessment of EF, the MMA group demonstrated improvements on the accuracy variable on the congruent and mixed blocks but not on the incongruent block. The WLC group did not improve in their accuracy on any of the blocks. Neither the MMA nor WLC groups demonstrated performance differences on the response time variable.

The results of this study demonstrated that a traditional approach to MMA helped improve both parent-reported and objectively-measured EF in middle-childhood-aged children with ASD. Limitations of our study include gender distributions in the two groups and variability in attendance. We still seek to understand why some EF domains showed improvement but not others.

The few studies on executive functioning and martial arts in children with ASD share some similar characteristics for study design and design of the intervention. In terms of study design, all three studies (Bahrami et al. 2012; Chan et al. 2013; Phung and Goldberg 2019) randomly assigned participants to either the martial arts or control group. Whereas Bahrami et al. (2012) and Phung and Goldberg (2019) included a no-martial arts control group in which participants were passively awaiting the post-test session, Chan et al. (2013) had their control group actively participate in the muscle relaxation training program at the same time that the martial arts group received their intervention program. All three studies included parent-reported assessments of child executive functioning; only Bahrami et al. (2012) included multiple informants (e.g., parents, caregivers, and teachers). Chan et al. (2013) and Phung and Goldberg (2019) both included objective measures of child executive functioning to supplement the parent report. In terms of number of test sessions, all three studies conducted pre- and post-test assessments, but only Bahrami et al. (2012) conducted an additional one-month follow-up after the intervention concluded. Finally, of the three studies, Phung and Goldberg (2019) had the most discrete age range (ages 8- to 11-years).

The other two studies included participants whose ages ranged from childhood (ages 5–6 years) through adolescence (ages 16–17 years).

Intervention design for the three studies also varied widely. First, the three studies executed different styles of martial arts: Bahrami et al. (2012) implemented Kata, Chan et al. (2013) implemented Nei Yang Gong, and Phung and Goldberg (2019) implemented Mixed Martial Arts. Although the three martial arts styles varied, they were all taught using a traditional approach, which emphasized character development in addition to physical conditioning rather than a modern approach that typically focuses on sport and competition (Trulson 1986). The intervention designs also varied in their duration (i.e., how long the program lasted from start to finish). The Bahrami et al. (2012) intervention was the most time intensive and took place four times per week for 14 weeks (56 sessions), followed by Phung and Goldberg (2019), which took place two times per week for 13 weeks (26 sessions), and lastly, Chan et al. (2013), which took place two times per week for four weeks (8 sessions). A similar pattern followed for intervention dosage or intensity (i.e., how much of the intervention to be delivered to participants). Again, martial arts participants in the Bahrami et al. (2012) study received the highest total dosage of martial arts intervention at approximately 3120 min. Martial arts participants in the Phung and Goldberg (2019) study received the second highest total dosage of martial arts intervention at approximately 1170 min. Lastly, martial arts participants in the Chan et al. (2013) study received the smallest total dosage of martial arts intervention at approximately 480 min. (Note that these were the expected dosages as reported by the researchers.)

Future Directions

Children with ASD often face difficulties with tasks that require the use of executive functions (EFs). Fortunately, EFs are malleable, especially during middle childhood compared to earlier developmental stages. There are a number of

activities that have been studied to improve EFs. Considering the recent martial arts intervention studies reviewed here, training in martial arts appears to improve EFs in children with ASD, compared to those in control groups that do not participate in martial arts. However, the field is comparatively new, and more research is needed.

Following up on our recent study (Phung and Goldberg 2019), future work should explore why some EFs improved but not others with the Mixed Martial Arts (MMA) intervention. Replication is also needed with a larger sample and a greater balance of genders between the MMA and WLC groups. Completion of the expected number of sessions was challenged in part by the many different types of obligations juggled by parents raising children with ASD (Myers et al. 2009). Future MMA intervention studies could offer more MMA classes throughout the week and weekend to accommodate to families' busy schedules.

Martial arts training includes mindfulness, physical activity, and cognitive complexity, but research to date has not yet isolated these three components. Future studies should include a mindfulness or yoga comparison group to determine if improvements are possibly due to the meditative component of the martial arts since mindfulness training has been linked to improved EFs in TD children (Flook et al. 2010). Although Chan et al. (2013) had a comparison group of children with ASD who participated in a meditation-type of activity, the martial arts group showed better overall self-control than the control group after the respective interventions. Additional research is needed to expand EFs in addition to self-control (e.g., working memory and cognitive flexibility).

Furthermore, to address the question of which physical activity matters most, future research should include a comparison group of children with ASD enrolled in non-martial arts sports teams (e.g., basketball, soccer, baseball, etc.) or individual aerobic exercise (e.g., jogging, swimming, etc.). As evidenced by prior studies, physical exercise has been linked to a decrease in stereotypy in children with ASD (e.g., Schmitz et al. 2017). Therefore, the inclusion of a sport

or aerobic exercise comparison group may be useful in determining if the physical activity drives the improvements in EFs, or if there is something unique about training martial arts in addition to its physical benefits.

In future research, the variability of dosage should be taken into consideration and streamlined across studies to allow for comparability. In a systematic review of behavioral outcomes following exercise interventions for children with ASD, Bremer et al. (2016) commented that a "more is better" approach is often taken with behavioral interventions, but this may not hold true regarding exercise and outcomes. For example, a ceiling effect could occur where observed benefits appear to plateau (Bremer et al. 2016). Alternatively, excessive or too much of an exercise intervention could result in the regression of progress. For example, Schmitz et al. (2017) found in their study that the most exhaustive exercise condition led to increased stereotypic behaviors in children with ASD compared to the less exhaustive exercise conditions. Fidelity checks are recommended to confirm that the intervention is in fact implemented as it was initially intended (Dumas et al. 2001).

Finally, future work should also consider the cognitive complexity component that is part of martial arts training. Because martial arts training includes mindfulness, physical activity, and cognitive complexity, future research should isolate these three components to determine if they each uniquely contribute to improving EFs, or whether the observed improvements are due to their interaction when implemented as part of martial arts training.

Martial arts training not only contributes to improving executive functioning, but could also contribute to improving other domains that are challenged in children with ASD, such as social dysfunction and behavior problems (e.g., Movahedi et al. 2013). Martial arts training is a feasible and ecologically valid means to improve EFs in children with ASD, and might also improve other areas of development. Benefits conferred from improvements in executive functioning likely extend beyond the cognitive and behavioral domains to include better mental,

physical, and social health, and overall improved quality of life in children with ASD. Indeed, the current state of knowledge is promising, and the future of this work is wide and has the potential for expansion.

See Also

- [BRIEF \(Behavior Rating Inventory of Executive Functions\)](#)
- [Coaching Students with Autism](#)
- [Exercise in Autism](#)
- [Inclusion](#)
- [Sports](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual* (5th ed.). Washington, DC: Author.
- Anderson, P. (2002). Assessment and development of executive function (EF) during childhood. *Child Neuropsychology*, 8(2), 71–82.
- Bahrani, F., Movahedi, A., Marandi, S. M., & Abedi, A. (2012). Kata techniques training consistently decreases stereotypy in children with Autism Spectrum Disorder. *Research in Developmental Disabilities*, 33, 1183–1193.
- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., et al. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summaries*, 67(6), 1–23.
- Best, J. R., & Miller, P. H. (2010). A developmental perspective on executive function. *Child Development*, 81(6), 1641–1660.
- Boyd, B. A., McBee, M., Holtzclaw, T., Baranek, G. T., & Bodfish, J. W. (2009). Relationships among repetitive behaviors, sensory features, and executive functions in high functioning autism. *Research in Autism Spectrum Disorders*, 3, 959–966.
- Bremer, E., Crozier, M., & Lloyd, M. (2016). A systematic review of the behavioral outcomes following exercise interventions for children and youth with Autism Spectrum Disorder. *Autism*, 20, 899–915.
- Chan, A. S., Sze, S. L., Siu, N. Y., Lau, E. M., & Cheung, M. C. (2013). A Chinese mind-body exercise improves self-control of children with autism: A randomized controlled trial. *PLoS One*, 8, e68184.
- Davidson, M. C., Amso, D., Anderson, L. C., & Diamond, A. (2006). Development of cognitive control and executive functions from 4 to 13 years: Evidence from manipulations of memory, inhibition, and task switching. *Neuropsychologia*, 44, 2037–2078.
- Diamond, A. (2002). Normal development of prefrontal cortex from birth to young adulthood: Cognitive functions, anatomy and biochemistry. In *Principles of frontal lobe function* (pp. 466–503). London: Oxford University Press.
- Diamond, A. (2012). Activities and programs that improve children's executive functions. *Current Directions in Psychological Science*, 21, 335–341.
- Diamond, A. (2013). Executive functions. *Annual Review of Psychology*, 64, 135–168.
- Diamond, A., & Lee, K. (2011). Interventions shown to aid executive function development in children 4 to 12 years old. *Science*, 333, 959–964.
- Diamond, A., Barnett, W. S., Thomas, J., & Munro, S. (2007). Preschool program improves cognitive control. *Science*, 318, 1387–1388.
- Dumas, J. E., Lynch, A. M., Laughlin, J. E., Smith, E. P., & Prinz, R. J. (2001). Promoting intervention fidelity: Conceptual issues, methods, and preliminary results from the Early Alliance prevention trial. *American Journal of Preventive Medicine*, 20, 38–47.
- Flook, L., Smalley, S. L., Kiti, J. M., Galla, B. M., Kaiser-Greenland, S., Locke, J., & Kasari, C. (2010). Effects of mindful awareness practices on executive functions in elementary school children. *Journal of Applied School Psychology*, 26, 70–95.
- Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2000). Test review behavior rating inventory of executive function. *Child Neuropsychology*, 6, 235–238.
- Hill, E. (2004). Executive dysfunction in autism. *Cognitive Science*, 8, 26–32.
- Kenworthy, L., Yerys, B. E., Anthony, L. G., & Wallace, G. L. (2008). Understanding executive control in autism spectrum disorders in the lab and in the real world. *Neuropsychology Review*, 18(4), 320–338.
- Kim, S. H., & Lord, C. (2010). Restricted and repetitive behaviors in toddlers and preschoolers with autism spectrum disorders based on the autism diagnostic observation schedule (ADOS). *Autism Research*, 3, 162–173.
- Lakes, K. D., & Hoyt, W. (2004). Promoting self-regulation through school-based martial arts training. *Applied Developmental Psychology*, 25, 283–302.
- Lakes, K. D., Bryars, T., Sirisinahal, S., Salim, N., Arastoo, S., Emmerson, N., . . . & Kang, C. J. (2013). The Healthy for Life Taekwondo pilot study: A preliminary evaluation of effects on executive function and BMI, feasibility, and acceptability. *Mental Health and Physical Activity*, 6, 181–188.
- Lee, B. (1975). *Tao of Jeet Kune do*. Burbank: Ohara Publications.
- Lopez, B., Lincoln, A. J., Ozonoff, S., & Lai, Z. (2005). Examining the relationship between executive functions and restricted, repetitive symptoms of autistic disorder. *Journal of Autism and Developmental Disorders*, 35, 445–460.

- Movahedi, A., Bahrami, F., Marandi, S. M., & Abedi, A. (2013). Improvement in social dysfunction of children with Autism Spectrum Disorder following long term kata techniques training. *Research in Autism Spectrum Disorders*, 7, 1054–1061.
- Myers, B. J., Mackintosh, V. H., & Goin-Kochel, R. P. (2009). “My greatest joy and my greatest heart ache:” Parents’ own words on how having a child in the autism spectrum has affected their lives and their families’ lives. *Research in Autism Spectrum Disorders*, 3, 670–684.
- Ozonoff, S., & Jensen, J. (1999). Specific executive function profiles in three neurodevelopmental disorders. *Journal of Autism and Developmental Disorders*, 29, 171–177.
- Phung, J. N. (2017). *Mixed martial arts as a means to improve social communication and executive functioning in children with Autism Spectrum Disorder* (Doctoral dissertation). UC Irvine.
- Phung, J. N., & Goldberg, W. A. (2019). Promoting executive functioning in children with Autism Spectrum Disorder through community-based mixed martial arts training. *Journal of Autism and Developmental Disabilities*, 49(9), 3669–3684.
- Richler, J., Bishop, S. L., Kleinke, J. R., & Lord, C. (2007). Restricted and repetitive behaviors in young children with Autism Spectrum Disorders. *Journal of Autism and Developmental Disorders*, 37(1), 73–85.
- Rosenthal, M., Wallace, G. L., Lawson, R., Wills, M. C., Dixon, E., Yerys, B. E., & Kenworthy, L. (2013). Impairments in real-world executive function increase from childhood to adolescence in autism spectrum disorders. *Neuropsychology*, 27(1), 13–18.
- Schmitz, O. S., Mcfadden, B. A., Golem, D. L., Pellegrino, J. K., Walker, A. J., Sanders, D. J., & Arent, S. M. (2017). The effects of exercise dose on stereotypical behavior in children with autism. *Medicine and Science in Sports and Exercise*, 49, 983–990.
- South, M., Ozonoff, S., & McMahon, W. M. (2007). The relationship between executive functioning, central coherence, and repetitive behaviors in the high-functioning autism spectrum. *Autism*, 11, 437–451.
- Thorell, L. B., Lindqvist, S., Bergman, N. S., Bohlin, G., & Klingberg, T. (2009). Training and transfer effects of executive functions in preschool children. *Developmental Science*, 12, 106–113.
- Trulson, M. E. (1986). Martial arts training: A novel “cure” for juvenile delinquency. *Human Relations*, 39, 1131–1140.
- Wolff, J. J., Botteron, K. N., Dager, S. R., Elison, J. T., Estes, A. M., Gu, H., . . . & Zwaigenbaum, L. (2014). Longitudinal patterns of repetitive behavior in toddlers with autism. *Journal of Child Psychology and Psychiatry*, 55(8), 945–953.
- Wright, A., & Diamond, A. (2014). An effect of inhibitory load in children while keeping working memory load constant. *Frontiers in Psychology*, 5, 1–9.

Executive Functions

► Frontal Lobe Findings in Autism

Executive Functions, Social Impairment, Friendship Quality, and Adjustment in HFA with ASD

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Definition

Social impairment is a hallmark feature of autism spectrum disorder (ASD). Research has shown the impacts of social deficits, particularly with high functioning ASD, including challenges with friendship development, loneliness, and depressive symptoms. Factors are also being identified that influence social skills, including executive functions (EF). It is important for the research and clinical community to understand the direct and indirect influence that these constructs have on each other as this has treatment implications. To support a child with ASD as they are experiencing social isolation or depression, it may be necessary to recognize and help build other skill areas such as cognitive flexibility, emotion regulation, inhibition, etc., which may assist in improving their socio-emotional outcomes.

Current Knowledge

Research has consistently shown comorbid depressive symptoms in youth with autism spectrum disorder (ASD; e.g., Ghaziuddin et al. 2002; Volkmar and Klin 2005; Klin et al. 2005; Lopata et al. 2010). Although studies vary widely in their reports of prevalence rates (see DeFilippis 2018 for a review), many comorbidity rates are as high as 53–54%, compared to the recent National

Institute of Mental Health statistic of 9.4% in the general adolescent population (Kim et al. 2000; Mayes et al. 2011; Ghaziuddin et al. 1998; Solomon et al. 2012; Thapar et al. 2012; National Institute of Mental Health 2019). Higher functioning youth with ASD are often aware of their differences in relation to same-aged peers (Volkmar and Klin 2005) and can be frustrated with social difficulties (Klin et al. 2005), which can lead to higher rates of anxiety and depressive symptoms than typically developing youth (e.g., Kim et al. 2000). This population exhibits greater levels of loneliness compared to neurotypical peers (e.g., Bauminger and Kasari 2000; Ghaziuddin et al. 2002; Lasgaard et al. 2009) and reports more social loneliness (e.g., Bauminger et al. 2003), suggesting they are not only aware of the concept of loneliness, but recognize it in themselves. Even in early elementary school, children with ASD are already reporting feeling symptoms of loneliness and being out of place at school (Zeedyk et al. 2016).

A direct association has been established between social impairment and adjustment (e.g., Barnhill 2001; Vickerstaff et al. 2007; White and Roberson-Nay 2009). The quality of social interactions in youth with ASD are often different, including fewer social initiations, shorter social interactions, isolated or more parallel play, and challenging or repetitive behaviors that may interfere with successful social interactions (see McConnell 2002 for a literature review). In addition, children in general often gravitate toward others with common interests; therefore, if a child with ASD has a focused interest on an uncommon topic (e.g., Titanic, fire extinguishers, box fans, the War of 1812), this can impact social experiences and friendships. Not surprisingly, youth with ASD report having fewer friends and spending less time interacting with friends. A review of 235 adults and adolescents with ASD found that only 8.1% reported interacting with same-aged friends on a weekly basis outside of an organized activity and 46.6% of the sample reported having no same-aged friends (Orsmond et al. 2004). Zeedyk et al. (2016) found almost 40% of an early elementary school ASD sample reported challenges with developing friendships

at school. This is despite youth with ASD having similar expectations for friendships compared to typically developing peers (Bottema-Beutel et al. 2019). Furthermore, Pouw et al. (2013) found that poorer friendship quality was associated with increased self-reported depressive symptoms in boys with ASD. While some reviews of the literature find that increased reports of loneliness are primarily from samples of adolescents with ASD, other studies find similar reports of loneliness in pre-adolescents (see Deckers et al. 2017 for an example). Further, Deckers et al. (2017) found significantly higher levels of loneliness compared not only to a non-clinical control group but a clinical-ADHD group.

Executive function (EF) impairments likely affect one's ability to successfully interact socially with others (e.g., Joseph 1999; Solomon et al. 2004). EFs are a set of cognitive behaviors that allow for higher level planning and organization (Wong et al. 2006). Research shows that youth with ASD demonstrate EF impairment compared to neuro-typical peers (e.g., Russo et al. 2007; Verte et al. 2006; Akshoomoff 2005; Semrud-Clikeman et al. 2014) and even those with ADHD (e.g., Semrud-Clikeman et al. 2010; Hughes 2011) although with the allowance of the dual diagnosis of ASD and ADHD in the DSM-5, recent research has suggested a more nuanced EF differentiation between the two diagnoses (e.g., Berenguer et al. 2018). Domains impacted include set shifting and cognitive flexibility (Ozonoff and Jensen 1999; Joseph 1999; Russo et al. 2007; Verte et al. 2006; Semrud-Clikeman et al. 2010), working memory (e.g., Russo et al. 2007; Rogers and Bennetto 2000; Verte et al. 2006), emotional control (Konstantareas and Stewart 2006; Semrud-Clikeman et al. 2010), and planning (e.g., Semrud-Clikeman et al. 2010). Results suggesting deficits in inhibition are less conclusive (Rogers and Bennetto 2000; Semrud-Clikeman et al. 2010; Verte et al. 2006; Narzisi et al. 2013). Inconsistencies in results may relate to differences in how studies address moderators and what measures of EF are used (see Demetriou et al. 2018 for further discussion).

There is further evidence demonstrating the impact of EF deficits on many areas of ASD

including social impairment, friendship quality, and even adjustment. Carrington et al. (2003) documented cognitive inflexibility when making decisions about friendships in five youth with ASD. Leung et al. (2016) found that parent-reported metacognitive EF deficits (working memory, organization, initiation, etc.) predicted increased greater social impairment in youth with ASD but not typically developing controls. They linked this to challenges with theory of mind, which requires one to think “metacognitively.” Interestingly, their study found that both ASD and control groups had increased social impairment as parent-reports of behavior dysregulation increased. Furthermore, the impact of social impairment on adjustment in youth with ASD may be impacted by poorer EF, making processing and utilizing social information even more challenging. Lieb and Bohnert (2017) assessed parent report of EF and social impairment and parent and self-reported friendship quality, loneliness, and depressive symptoms in a sample of high functioning ASD youth. Not only did they find direct relations between poorer EF and more adjustment difficulties, but structural equation modelling found good fitting models, whereby social impairment mediated domains of EF (inhibition, shifting, emotional control, working memory). Thus, greater EF impairment was linked to greater social impairment and subsequently poorer adjustment. Interestingly, there was less evidence to support the mediating relation of friendship quality with the exception of its impact on the relation between emotional control and loneliness.

Future Directions

Longitudinal studies examining the direct and indirect effects of EF, social impairment, and friendship quality are necessary. While it is possible that having stronger EF skills leads to better friendship quality or social skills, it is unknown whether those with better abilities to make and keep friends may more easily be able to improve upon and develop their EF skills over time. Furthermore, understanding whether the relations between these domains

differs in females compared to males is important, as emerging research has continued to demonstrate the subtle yet important differences in the behavioral presentation of ASD in females and specific gender differences in social experiences including friendship quality and social motivation for youth both with and without ASD (e.g., Sedgewick et al. 2016). Additionally, developing assessment measures with excellent norms for populations across the autism spectrum will allow for more accurate parent- and self-report. Relatedly, it is important to evaluate and possibly refine our operational definitions of these constructs. For example, Donaldson et al. (2018) propose that nuanced differences in the friendship literature may be impacted by how neuro-typical youth and those with ASD define friendships and social relationships, which can impact how we approach both research and intervention. Finally, it is important to further explore other influential factors, including cognitive and language abilities, and co-morbid medical and/or mental health diagnoses to understand the differential impact that belonging to “multiple groups” may have on a person’s outcomes. For example, Berenguer et al. (2018) found that children with comorbid ASD and ADHD had great EF deficits than those with ASD only and that the ASD and ADHD group had more behavioral challenges than either the ASD only or ADHD only groups. Better understanding the complexities of the whole person can help clinicians, teachers, and even families and caregivers address challenges more effectively.

See Also

► [Friendships](#)

References and Reading

Sample References

- Akshoomoff, N. (2005). The neuropsychology of autistic spectrum disorders. *Developmental Neuropsychology*, 27, 307–310. https://doi.org/10.1207/s15326942dn2703_1.
- Barnhill, G. P. (2001). Social attributions and depression in adolescents with Asperger syndrome. *Focus on Autism*

- and Other Developmental Disabilities, 16, 46–53. <https://doi.org/10.1177/108835760101600112>.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development, 71*, 447–456. <https://doi.org/10.1111/1467-8624.00156>.
- Bauminger, N., Shulman, C., & Agam, G. (2003). Peer interaction and loneliness in high-functioning children with autism. *Journal of Autism and Developmental Disorders, 5*, 489–507. <https://doi.org/10.1023/a:1025827427901>.
- Berenguer, C., Rosello, B., Colomer, C., Baixauli, I., & Miranda, A. (2018). Children with autism and attention deficit hyperactivity disorder: Relationships between symptoms and executive function, theory of mind, and behavioral problems. *Research in Developmental Disabilities, 83*, 260–269. <https://doi.org/10.1016/j.ridd.2018.10.001>.
- Bottema-Beutel, K., Malloy, C. Cuda, J., Kim, S. Y., & MacEvoy, J. P. (2019). Friendship expectations may be similar for mental age-matched children with autism spectrum disorder and typically developing children. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-04141-7>.
- Carrington, S., Templeton, E., & Papinczak, T. (2003). Adolescents with Asperger syndrome and perceptions of friendship. *Focus on Autism and Other Developmental Disabilities, 18*, 211–218. <https://doi.org/10.1177/10883576030180040201>.
- Deckers, A., Muris, P., & Roelofs, J. (2017). Being on your own or feeling lonely? Loneliness and other social variables in youths with autism spectrum disorders. *Child Psychiatry and Human Development, 48*, 828–839. <https://doi.org/10.1007/s10578-016-0707-7>.
- DeFilippis, M. (2018). Depression in children and adolescents with autism spectrum disorder. *Children, 5*. <https://doi.org/10.3390/children5090112>.
- Demetriou, E. A., Lampit, A., Quintana, D. S., Maismith, S. L., Song, Y. J. C., Pye, J. E., Hickie, I., & Guastella, A. J. (2018). Autism spectrum disorders: A meta-analysis of executive function. *Molecular Psychiatry, 23*, 1198–1204.
- Donaldson, A. L., Nolfo, M., & Montejano, M. (2018). Relationships, friendships, and successful social Communication: Addressing disability. *Seminars in Speech and Language, 36*, 166–177. <https://doi.org/10.1055/s-0038-1628368>.
- Ghaziuddin, M., Weidmer-Mikhail, E., & Ghaziuddin, N. (1998). Comorbidity of Asperger syndrome: A preliminary report. *Journal of Intellectual Disability Research, 42*, 279–283. <https://doi.org/10.1111/j.1365-2788.1998.tb01647.x>.
- Ghaziuddin, M., Ghaziuddin, N., & Greden, J. (2002). Depression in persons with autism: Implications for research and clinical care. *Journal of Autism and Developmental Disorders, 32*, 299–306. <https://doi.org/10.1023/A:1016330802348>.
- Hughes, C. (2011). Changes and challenges in 20 years of research into the development of executive functions. *Infant and Child Development, 20*, 251–271. <https://doi.org/10.1002/icd.736>.
- Joseph, R. M. (1999). Neuropsychological frameworks for understanding autism. *International Review of Psychiatry, 11*, 309–325. <https://doi.org/10.1080/09540269974195>.
- Kim, J. A., Szatmari, P., Bryson, S. E., Streiner, D. L., & Wilson, F. J. (2000). The prevalence of anxiety and mood problems among children with autism and Asperger syndrome. *Autism, 4*, 117–132. <https://doi.org/10.1177/1362361300004002002>.
- Klin, A., McPartland, J., & Volkmar, F. R. (2005). Asperger syndrome. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 88–125). Hoboken: Wiley.
- Konstantareas, M. M., & Stewart, K. (2006). Affect regulation and temperament in children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 36*, 143–154. <https://doi.org/10.1007/s10803-005-0051-4>.
- Lasgaard, M., Nielse, A., Eriksen, M. E., & Goossens, L. (2009). Loneliness and social support in adolescent boys with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 40*, 218–226. <https://doi.org/10.1007/s10803-009-0851-z>.
- Leung, R. C., Vogan, V. M., Powell, T. L., Anagnostou, E., & Taylor, M. J. (2016). The role of executive functions in social impairment in autism spectrum disorder. *Child Neuropsychology, 22*, 336–344. <https://doi.org/10.1080/09297049.2015.1005066>.
- Lieb, R. L., & Bohnert, A. (2017). Relations between executive functions, social impairment, and friendship quality on adjustment among high functioning youth with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 47*, 2861–2872. <https://doi.org/10.1007/s10803-017-3205-2>.
- Lopata, C., Toomey, J. A., Fox, J. D., Volker, M. A., Chow, S. Y., Thomeer, M. L., ... Smerbeck, A. M. (2010). Anxiety and depression in children with HFASD: Symptom levels and source differences. *Journal of Abnormal Child Psychology, 38*, 765–776. <https://doi.org/10.1007/s10802-010-9406-1>.
- Mayes, S., Calhoun, S., Murray, M., & Zahid, J. (2011). Variables associated with anxiety and depression in children with Autism. *Journal of Developmental and Physical Disabilities, 23*, 325–337. <https://doi.org/10.1007/s10882-011-9231-7>.
- McConnell, S. R. (2002). Interventions to facilitate social interaction for young children with autism: Review of available research and recommendations for educational intervention and future research. *Journal of Autism and Developmental Disorders, 32*, 351–372. <https://doi.org/10.1023/A:1020537805154>.
- National Institute of Mental Health. (2019, February). Major depression. Retrieved August 20, 2019, from https://www.nimh.nih.gov/health/statistics/major-depression.shtml#part_155033.
- Orsmond, G. I., Krauss, M. W., & Seltzer, M. M. (2004). Peer relationships and social and recreational activities among adolescents and adults with autism. *Journal of Autism and Developmental Disorders, 34*, 245–256. <https://doi.org/10.1023/B:JADD.0000029547.96610.df>.

- Ozonoff, S., & Jensen, J. (1999). Brief report: Specific executive function profiles in three neurodevelopmental disorders. *Journal of Autism and Developmental Disorders*, 29, 171–177. <https://doi.org/10.1023/A:1023052913110>.
- Pouw, L. B. C., Rieffe, C., Stockmann, L., & Gadow, K. D. (2013). The link between emotion regulation, social functioning, and depression in boys with ASD. *Research in Autism Spectrum Disorders*, 7, 549–556. <https://doi.org/10.1016/j.rasd.2013.01.002>.
- Rogers, S. J., & Bennetto, L. (2000). Intersubjectivity in autism. In A. M. Wetherby & B. M. Prizant (Eds.), *Autism spectrum disorders: A transactional developmental perspective* (1st ed., pp. 79–107). Baltimore: Paul H. Brooks Publishing Co.
- Russo, N., Flanagan, T., Iarocci, G., Berringer, D., Zelazo, P. D., & Burack, J. A. (2007). Deconstructing executive deficits among persons with autism: Implications for cognitive neuroscience. *Brain and Cognition*, 65, 77–86. <https://doi.org/10.1016/j.bandc.2006.04.007>.
- Sedgewick, F., Hill, V., Yates, R., Pickering, L., & Pellicano, E. (2016). Gender differences in the social motivation and friendship experiences of autistic and non-autistic adolescents. *Journal of Autism and Developmental Disorders*, 46, 1297–1306. <https://doi.org/10.1007/s10803-015-2669-1>.
- Semrud-Clikeman, M., Walkowiak, J., Wilkinson, A., & Butcher, B. (2010). Executive functioning in children with Asperger syndrome, ADHD-combined type, ADHD-predominately inattentive type and controls. *Journal of Autism and Developmental Disorders*, 40, 1017–1027. <https://doi.org/10.1007/s10803-010-0951-9>.
- Solomon, M., Goodlin-Jones, B. L., & Anders, T. F. (2004). A social adjustment enhancement intervention for high functioning autism, Asperger's syndrome, and pervasive developmental disorder NOS. *Journal of Autism and Developmental Disorders*, 34, 649–668. <https://doi.org/10.1007/s10803-004-5286-y>.
- Solomon, M., Miller, M., Taylor, S. L., Hinshaw, S. P., & Carter, C. S. (2012). Autism symptoms and internalizing psychopathology in girls and boys with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42, 48–59. <https://doi.org/10.1007/s10803-011-1215-z>.
- Thapar, A., Collishaw, S., Pine, D. S., & Thapar, A. K. (2012). Depression in adolescence. *The Lancet*, 379, 1056–1067. [https://doi.org/10.1016/S0140-6736\(11\)60871-4](https://doi.org/10.1016/S0140-6736(11)60871-4).
- Verte, S., Geurts, H. M., Roeyers, H., Oosterlaan, J., & Sergeant, J. A. (2006). Executive functioning in children with an autism spectrum disorder: Can we differentiate within the spectrum? *Journal of Autism and Developmental Disorders*, 36, 351–372. <https://doi.org/10.1007/s10803-006-0074-5>.
- Vickerstaff, S., Heriot, S., Wong, M., Lopes, A., & Dossetor, D. (2007). Intellectual ability, self-perceived social competence, and depressive symptomatology in children with high-functioning autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 1647–1664. <https://doi.org/10.1007/s10803-006-0292-x>.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 5–41). Hoboken: Wiley.
- White, S. W., & Roberson-Nay, R. (2009). Anxiety, social deficits, and loneliness in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 1006–1013. <https://doi.org/10.1007/s10803-009-0713-8>.
- Wong, D., Maybery, M., Bishop, D. V. M., Maley, A., & Hallmayer, J. (2006). Profiles of executive function in parents and siblings of individuals with autism spectrum disorders. *Genes, Brain, & Behavior*, 5, 561–576. <https://doi.org/10.1111/j.1601-183X.2005.00199.x>.
- Zeedyk, S. M., Cohen, S. R., Eisenhower, A., & Blacher, J. (2016). Perceived social competence and loneliness among young children with ASD: Child, parent, and teacher reports. *Journal of Autism and Developmental Disorders*, 46, 436–449. <https://doi.org/10.1007/s10803-015-2575-6>.

Exercise

- Inclusion of Adolescents with ASD in Community Sporting Clubs
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Exercise in Autism

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Definition

Physical activity (exercise) can be defined as the expenditure of energy through body movements produced by skeletal muscles (World Health Organization [WHO] 2017). Physical activity

may be structured (organized sporting events) or unstructured (use of playground equipment) and may involve commonly recognized physical exercises (e.g., running and lifting weights) as well as activities such as doing household chores and gardening (Ayvazoglu 2009; Biddle 1994; Obrusnikova and Miccinello 2012). The National Heart, Lung, and Blood Institute (NHLBI 2016) defines four types of physical activity: (a) aerobic exercises involving the sustained movement of larger muscles, as in jogging and cycling; (b) muscle-strengthening exercises such as lifting weights, push-ups, and sit-ups designed to increase muscle strength and endurance; (c) bone-strengthening exercises that require supporting one's own body weight (e.g., rope climbing and gymnastics); and (d) stretching, which improves flexibility by extending muscle groups through a range of body motions. Physical activity may be further classified according to intensity and physical exertion (i.e., heart and breathing rate) as either low, moderate, or vigorous. For example, in most cases light walking slightly increases heart rate and respiration and could be considered low intensity. Jogging might be counted as a moderate-intensity exercise because it results in a higher heart rate, but individuals can still perform for a relatively long period of time without fatigue, while sprinting would be classified as vigorous intensity, if it substantially increases heart rate and breathing.

Individuals with autism spectrum disorder (ASD) experience a number of deficits and challenges in areas related to physical activity and exercise (Fournier et al. 2010; Hocking and Caeyenberghs 2017). For example, people with ASD are reported to display difficulties on measures of balance, postural stability, muscle strength, and gait when compared to individuals without ASD (Bhat et al. 2011; Liu and Breslin 2013; Provost et al. 2007; Staples and Reid 2010). Furthermore, people with ASD are less likely to engage in moderate to vigorous physical activity and participate in organized sports. They tend to live sedentary lifestyles as compared to others without ASD (Bandini et al. 2013; Obrusnikova and Cavalier 2011; Pan 2009; Pan and Frey 2006; Pan et al. 2017; Pan et al. 2011;

Rimmer et al. 2004; Rimmer and Rowland 2008; Rimmer et al. 2007; Todd and Reid 2006). Regular exercise participation results in a number of physical and psychological benefits (e.g., reduces risks of cardiovascular disease, certain cancers, diabetes, and obesity) and may facilitate a reduction in stress as well as provide opportunities for community involvement, recreation, and socialization (Ensel and Lin 2004; Hillier et al. 2011; USDHHS 2002). Given these health and wellness benefits, research has examined the use of exercise in the education and treatment of people with ASD (Bremer et al. 2016; Lang et al. 2010; Tan et al. 2016).

Historical Background

In general, the health and wellness benefits of physical activity and exercise have been widely recognized for centuries and the positive effects have been well documented (Janssen and Leblanc 2010; National Association for Sport and Physical Education [NASPE] 2002). However, research on the potential benefits of exercise for people with ASD began to appear more regularly in the 1980s (Lang et al. 2010). In addition to improved physical fitness, several early studies investigating the effect of exercise in people with ASD reported benefits specifically related to ASD characteristics. For example, Watters and Watters (1980) reported a 32.7% mean decrease in the self-stimulation in children with ASD following physical exercise. Allen (1980) found that jogging reduced the frequency of problem behaviors among children with ASD; similarly, Kern et al. (1982) reported that jogging decreased self-stimulation and increased appropriate play and academic responding. These findings have been replicated by different research teams using a variety of research designs (Bachman and Fuqua 1983; Gordon et al. 1986; Kern et al. 1984; Levinson and Reid 1993; Reid et al. 1988). Overall, early research studies suggest that exercise can reduce stereotypic behaviors and improve adaptive responses in children with ASD.

Since the 1980s, research has progressed to studying the relative benefits of low, moderate, and vigorous exercise involving diverse types of physical activities on various dependent variables and target behaviors. Additionally, research has continued to examine the physical activity and exercise benefits and has started to investigate various potential biological and behavioral mechanisms that may account for improvements in nonphysical domains (e.g., how/why does exercise reduce stereotypy). Although clear mechanisms of action have not yet emerged from the literature and studies investigating various intensity levels have been inconsistent, the current corpus of research literature includes a sufficient number of rigorous studies to identify a few well-established findings involving ASD and physical activity.

Current Knowledge

During early childhood, physical activity levels of young children with and without ASD are similar (Bandini et al. 2013; Pan 2009). However, in the absence of intervention, school-aged children with ASD are likely to spend less time engaging in moderate physical activity and participating in fewer after-school physical activities than their typically developing peers (Bandini et al. 2013; Liu and Breslin 2013; Liu et al. 2016; Provost et al. 2007). Differences in physical activity levels may be due to lack of social interaction and motor skill competence, obesity, and reduced opportunity to participate (MacDonald et al. 2011; McCoy et al. 2016).

Increased physical activity and exercise has been shown to result in improved cardiorespiratory endurance, flexibility, balance, muscle strength, and fitness similar to children without ASD. For example, Fragala-Pinkham et al. (2008) reported improved cardiorespiratory endurance and muscle strength in children with ASD following an aquatic physical activity participation. Although a few studies have reported differences in physiological responses to exercise between groups of children with and without ASD (e.g., lower heart rate in ASD group relative to control;

Pace and Bricout 2015), there appears to be some consensus that exercise improves the health of people with ASD similar to that of people without ASD.

Increased physical activity and exercise-based interventions have also been linked to improvements in the cognitive, social, and behavioral characteristics often associated with ASD (Tan et al. 2016). A reduction in stereotypy for a brief period of time following exercise is among the most commonly reported beneficial outcomes in the current literature (Bremer et al. 2016; Lang et al. 2010; Neely et al. 2015; Petrus et al. 2008). Unfortunately, the reduction in stereotypy appears to be temporary and stereotypy tends to return to baseline levels within 2 h post exercise (Liu et al. 2016).

The evidence-base supporting improved cognitive and social performance in people with ASD following exercise is not as well established but should still be considered promising. Tan et al. (2016) conducted a systematic review of 22 studies involving a combined total of 579 participants with ASD between the ages of three and 25 years old and reported a small-to-medium effect of exercise on cognitive performance. In addition, Anderson-Hanley et al. (2011) found significant improvements in the measures of sustained attention and working memory following physical exercise. Similarly, improved academic performance (e.g., accuracy and efficiency in completing school tasks) and school-readiness behavior (compliance and on-task behavior) have been reported in multiple studies (ElGarhy and Liu 2016; Neely et al. 2015; Rosenthal-Malek and Mitchell 1997).

For clinicians and those involved in designing programs for people with ASD, the bottom line is that increasing physical activity in children with ASD appears to be an increasingly common treatment goal, and the current research base clearly indicates that opportunities for exercise, sports, and other physical activity should be embedded within school and residential programs serving people with ASD (Bremer et al. 2016; ElGarhy and Liu 2016; Liu and Breslin 2013; McCoy et al. 2016; Sowa and Meulenbroek 2012; Tan et al. 2016).

Future Directions

Recent systematic reviews and meta-analyses of the peer-reviewed research literature related to physical activity and exercise for people with ASD have identified several directions for future research, including (a) evaluating long-term outcomes of physical activity; (b) elucidating the physiological or psychological mechanisms that cause, contribute, or impede beneficial outcomes; and (c) determining efficient and effective approaches to embedding exercise into routines and existing treatment protocols (Bremer et al. 2016; Lang et al. 2010; Lewis et al. 2017; Tan et al. 2016). This future research should focus on evidence-based components and consider incorporating technology. For example, video games that require physical activity and software applications (apps) that prompt and reinforce physical activity may facilitate inclusion of exercise into daily routines while also providing motivation by making physical activity easier and more enjoyable (Hamilton et al. 2010).

Some of the more promising outcomes of increased physical activity for people with ASD require additional replication and extension. For example, improved cognitive and social functioning have been reported in a number of studies, but the wide range of cognitive and social dependent variables measured in those studies as well as the variety of exercise and physical activities that have been investigated limit the certainty and clarity of this corpus of research. Cognitive performance has been considered in terms of retention (memory), academic performance, among other measures of executive functioning following aerobic, muscle-strengthening, bone-strengthening, and stretching exercises with inconsistent results both within and across studies (Tan et al. 2016). Similar issues apply to the purported social functioning effects of exercise, and it has been suggested that improved approaches to measuring social performance are necessary. Additionally, the relative effects of low, moderate, and vigorous activity are not yet clear. For example, Olin et al. (2017) reported that high-intensity (vigorous) exercise exacerbated stereotypy, while low- to moderate-intensity exercise

occasioned large significant reductions; however, other studies have conversely reported vigorous-intensity exercise to have a more pronounced effect than low- and moderate-intensity (Kern et al. 1984). Currently, it is not clear if such discrepancies across studies are due to uncontrolled differences in participants, types of exercise, criterion used to classify intensity, or other similar variables. Finally, it may be fruitful to consider whether physical activity conducted immediately prior to other forms of therapy (e.g., language training) enhances the desirable outcomes of existing evidence-based interventions for children with ASD (Pan et al. 2017; Tan et al. 2013).

See Also

- Motor Planning
- Sensorimotor Development
- Stereotyped Movement Disorder

References and Reading

- Allen, J. I. (1980). Jogging can modify disruptive behaviors. *The Council Exceptional Children*, 66–70. Retrieved from <http://journals.sagepub.com/doi/pdf/10.1177/004005998001200207>.
- Anderson-Hanley, C., Tureck, K., & Schneiderman, R. (2011). Autism and exergaming: Effects on repetitive behaviors and cognition. *Psychology Research and Behavior Management*, 4, 129–137.
- Ayvazoglu, N. (2009). *Physical activity determinants in youth with high functioning autism*. ProQuest Dissertations Publishing, Indiana University.
- Bachman, J., & Fuqua, R. (1983). Management of inappropriate behaviors of trainable mentally impaired students using antecedent exercise. *Journal of Applied Behavior Analysis*, 16, 477–484.
- Bandini, L. G., Gleason, J., Curtin, C., Lividini, K., Anderson, S. E., Cermak, S. A., . . . , & Must, A. (2013). Comparison of physical activity between children with autism spectrum disorders and typically developing children. *Autism: The International Journal of Research and Practice*, 17, 44–54.
- Bhat, A. N., Landa, R. J., & Galloway, J. C. (2011). Current perspectives on motor functioning in infants, children, and adults with autism spectrum disorders. *Physical Therapy*, 91, 1116–1129.
- Biddle, S. (1994). What helps and hinders people becoming more physically active? In A. Killoran, P. Fentem,

- & C. Caspersen (Eds.), *Moving on: International perspectives on promoting physical activity* (pp. 110–126). London: Health Education Authority.
- Bremer, E., Crozier, M., & Lloyd, M. (2016). A systematic review of the behavioral outcomes following exercise interventions for children and youth with autism spectrum disorders. *Autism, 20*, 899–915.
- ElGarhy, S., & Liu, T. (2016). Effects of psychomotor intervention program on students with autism spectrum disorder. *School Psychology Quarterly, 31*, 491–506.
- Ensel, W., & Lin, N. (2004). Physical fitness and the stress process. *Journal of Community Psychology, 32*, 81–101. <https://doi.org/10.1002/jcop.10079>.
- Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism spectrum disorders: A synthesis and Meta-analysis. *Journal of Autism and Developmental Disorders, 40*, 1227–1240.
- Fragala-Pinkham, M., Haley, S. M., & O'Neil, M. E. (2008). Group aquatic aerobic exercise for children with disabilities. *Developmental Medicine & Child Neurology, 50*, 822–827.
- Gordon, R., Handleman, J. S., & Harris, S. L. (1986). The effects of contingent versus noncontingent running on the out-of-seat behavior of an autistic boy. *Child and Family Behavior Therapy, 8*, 37–44.
- Hamilton, M., Healy, G., Dunstan, G., Zderic, T., & Owen, N. (2010). Too little exercise and too much sitting: Inactivity physiology and the need for new recommendations for sedentary behavior. *Current Cardiovascular Risk Reports, 2*, 292–298.
- Hillier, A., Murphy, D., & Ferrara, C. (2011). A pilot study: Short-term reduction in salivary cortisol following low level physical exercise and relaxation among adolescents and young adults on the autism spectrum. *Journal of the International Society for the Investigation of Stress, 27*, 395–402. <https://doi.org/10.1002/smi.1391>.
- Hocking, D. R., & Caeyenberghs, K. (2017). What is the nature of motor impairments in autism, are they diagnostically useful, and what are the implications for intervention? *Current Developmental Disorders Report, 4*, 19. <https://doi.org/10.1107/s40474-017-01090-y>.
- Janssen, I., & Leblanc, A. (2010). Systematic review of the health benefits of physical activity and fitness in school-aged children and youth. *International Journal of Behavioral Nutrition & Physical Activity, 7*, 40–55. <https://doi.org/10.1186/1479-5868-7-40>.
- Kern, L., Koegel, R., Dyer, K., Blew, P., & Fenton, L. (1982). The effects of physical exercise on self-stimulation and appropriate responding in autistic children. *Journal of Autism and Developmental Disorders, 12*, 399–419.
- Kern, L., Koegel, R. L., & Dunlap, G. (1984). The influence of vigorous versus mild exercise on autistic stereotyped behaviors. *Journal of Autism and Developmental Disorders, 14*, 57–67.
- Lang, R., Koegel, K. L., Ashbaugh, K., Regeister, A., Ence, W., & Smith, W. (2010). Physical exercise and individuals with autism spectrum disorders: A systematic review. *Research in Autism Spectrum Disorders, 4*, 565–576.
- Levinson, L., & Reid, G. (1993). The effects of exercise intensity on the stereotypic behaviors of individuals with autism. *Adapted Physical Activity Quarterly, 10*, 255–268.
- Lewis, B., Napolitano, M., Buman, M., Williams, D., & Nigg, C. (2017). Future directions in physical activity intervention research: Expanding our focus to sedentary behaviors, technology, and dissemination. *Journal of Behavioral Medicine, 40*, 112–126. <https://doi.org/10.1007/s10865-016-9797-8>.
- Liu, T., & Breslin, C. M. (2013). Fine and gross motor performance of the MABC-2 by children with autism spectrum disorder and typically developing children. *Research in Autism Spectrum Disorders, 7*, 1244–1249.
- Liu, T., Fedak, A. T., & Hamilton, M. (2016). The effects of physical activity on the stereotypic behaviors of children with autism spectrum disorder. *International Journal of School Health, 3*(1), e28674. <https://doi.org/10.17795/intjsh-28674>.
- MacDonald, M., Esposito, P., & Ulrich, D. (2011). The physical activity patterns of children with autism. *BMC Research Notes, 4*, 422.
- McCoy, S. M., Jakicic, J. M., & Gibbs, B. B. (2016). Comparison of obesity, physical activity, and sedentary behaviors between adolescents with autism spectrum disorders and without. *Journal of Autism and Developmental Disorders, 46*, 2317–2326.
- National Association for Sport and Physical Education [NASPE], an association of the American Alliance for Health, Physical Education, Recreation and Dance. (2002). *Active start: A statement of physical activity guidelines for children birth to five years* (pp. 5–11). Reston: National Association for Sport and Physical Education.
- National Heart, Lung and Blood Institute. (2016). Physical activity types. U.S. Department of Health & Human Services. Retrieved from <https://www.nhlbi.nih.gov/health/health-topics/topics/phys/types>.
- Neely, L., Rispoli, M., Gerow, S., & Ninci, J. (2015). Effects of antecedent exercise on academic engagement and stereotypy during instruction. *Behavior Modification, 39*, 98–116.
- Obrusnikova, I., & Cavalier, A. (2011). Perceived barriers and facilitators of participation in after-school physical activity by children with autism spectrum disorders. *Journal of Developmental & Physical Disabilities, 23*, 195–211.
- Obrusnikova, I., & Miccinello, D. (2012). Parent perceptions of factors influencing after-school physical activity of children with autism spectrum disorders. *Adapted Physical Activity Quarterly, 29*, 63–80.
- Olin, S. S., McFadden, B. A., Golem, D. L., Pellegrino, J. K., Walker, A. J., Sanders, D. J., & Arent, S. M. (2017). The effects of exercise dose on stereotypic

- behavior in children with autism. *Medicine and Science in Sports and Exercise*, 49, 983–990.
- Pace, M., & Bricout, V. (2015). Low heart rate response of children with autism spectrum disorders in comparison to controls during physical exercise. *Physiology & Behavior*, 141, 63–68.
- Pan, C. (2009). Age, social engagement, and physical activity in children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 3, 22–31. <https://doi.org/10.1016/j.rasd.2008.03.002>.
- Pan, C., & Frey, G. (2006). Physical activity patterns in youth with autism spectrum disorders. *Journal of Autism Developmental Disorders*, 36, 597–606. <https://doi.org/10.1007/s10803-006-0101-6>.
- Pan, C., Tsai, C., Hsieh, K., Chu, C., Li, Y., & Huang, S. (2011). Accelerometer-determined physical activity among elementary school-aged children with autism spectrum disorders in Taiwan. *Research in Autism Spectrum Disorders*, 5, 1042–1052. <https://doi.org/10.1016/j.rasd.2010.11.010>.
- Pan, C., Chu, C., Tsai, C., Sung, M., Huang, C., & Ma, W. (2017). The impacts of physical activity intervention on physical and cognitive outcomes in children with autism spectrum disorder. *Autism*, 21, 190–202.
- Petrus, C., Adamson, S. R., Block, L. R., Einarson, S. J., Sharifnejad, M., & Harris, S. R. (2008). Effects of exercise interventions on stereotypic behaviours in children with autism spectrum disorder. *Physiotherapy Canada*, 60, 134–245.
- Provost, B., Heimerl, S., & Lopez, B. R. (2007). Levels of gross and fine motor development in young children with autism spectrum disorder. *Physical & Occupational Therapy in Pediatrics*, 27, 21–36. https://doi.org/10.1300/J006v27n03_03.
- Reid, P. R., Factor, D. C., Freeman, N. L., & Sherman, J. (1988). The effects of physical exercise on three autistic and developmentally disordered adolescents. *Therapeutic Recreation Journal*, 22, 47–56.
- Rimmer, J. H., & Rowland, J. L. (2008). Health promotion for people with disabilities: Implications for empowering the person and promoting disability-friendly environments. *American Journal of Lifestyle Medicine*, 2, 409–420.
- Rimmer, J. H., Riley, B., Wang, E., Rauworth, A., & Jurkowski, J. (2004). Physical activity participation among persons with disabilities: Barriers and facilitators. *American Journal of Preventive Medicine*, 26, 419–425.
- Rimmer, J. H., Rowland, J. L., & Yamaki, K. (2007). Obesity and secondary conditions in adolescents with disabilities: Addressing the needs of an underserved population. *Journal of Adolescent Health*, 41, 224–229.
- Rosenthal-Malek, A., & Mitchell, S. (1997). Brief report: The effects of exercise on the on the self-stimulatory behaviors and positive responding of adolescents with autism. *Journal of Autism and Developmental Disorders*, 27, 193–202.
- Sowa, M., & Meulenbroek, R. (2012). Effects of physical exercise on autism spectrum disorders: A meta-analysis. *Research in Autism Spectrum Disorders*, 6, 46–57.
- Staples, K. L., & Reid, G. (2010). Fundamental movement skills and autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40, 209–217.
- Tan, B., Cohen, L., & Pooley, J. (2013). Physical activity: its implication on attention span and quality of life in children with autism spectrum disorders. *GSTF International Journal of Law and Social Sciences*, 2, 107–116.
- Tan, B. W. Z., Pooley, J. A., & Speelman, C. P. (2016). A meta-analytic review of the efficacy of physical exercise on cognition in individuals with autism spectrum disorder and ADHD. *Journal of Autism and Developmental Disorders*, 46, 3126–3143.
- Todd, T., & Reid, G. (2006). Increasing Physical Activity in Individuals with Autism. *Focus on Autism and Other Developmental Disabilities*, 21, 167–176.
- U. S. Department of Health and Human Services. (2002). Healthy people 2010: Physical activity and fitness. Retrieved from <http://www.health.gov/healthypeople/Document/HTML/Volume2/22Physical.htm>.
- Watters, R., & Watters, W. (1980). Decreasing self-stimulatory behavior with physical exercise in a group of autistic boys. *Journal of Autism and Developmental Disorders*, 10, 379–387.
- World Health Organization (WHO). (2017). Hot topics, physical activity. Retrieved from http://www.who.int/topics/physical_activity/en/

Experimental Research

► Qualitative Versus Quantitative Approaches

Explicit Memory

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Synonyms

Declarative memory; Fact memory

Definition

A specific type of memory that involves intentional, conscious recollection. Explicit memory

is often assessed by using tasks that require recognition and recall. Research has shown that individuals with Autism Spectrum Disorder (ASD) have intact explicit memory, although they use a different memory retrieval process. Individuals with ASD and intellectual disability show greater impairments in explicit memory compared to high-functioning individuals with ASD.

See Also

- [Declarative Memory](#)
- [Explicit Memory](#)
- [Free Recall](#)
- [Memory](#)
- [Memory Assessment](#)
- [Memory Development](#)
- [Recognition Memory](#)
- [Retrieval of Information](#)
- [Rote Memory](#)
- [Semantic Memory](#)
- [Short-Term Memory](#)

References and Reading

- Anooshian, L. J. (1997). Distinctions between implicit and explicit memory: Significance for understanding cognitive development. *International Journal of Behavioral Development*, 21, 453–478.
- Murphy, K., McKone, E., & Slee, J. (2003). Dissociations between implicit and explicit memory in children: The role of strategic processing and the knowledge base. *Journal of Experimental Child Psychology*, 84, 124–165.
- Renner, P., Klinger, L. G., & Klinger, M. R. (2000). Implicit and explicit memory in autism: Is autism an amnesic disorder? *Journal of Autism and Developmental Disorders*, 30, 3–14.
- Rovee-Collier, C. K., Hayne, H., & Columbo, M. (2001). *The development of implicit and explicit memory: Advances in consciousness research*. Philadelphia: John Benjamins Publishing Company.
- Squire, L. R. (1987). *Memory and brain*. New York: Oxford University Press.

Exploratory Play

- [Play](#)

Expressive Communication

- [Expressive Language](#)

Expressive Dysphasia

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Synonyms

[Developmental language disorder](#); [Expressive language disorder](#); [Specific language impairment](#)

Definition

In neurological contexts, language impairment is identified as either acquired (aphasia) or developmental (dysphasia). An expressive dysphasia is a language disorder linked to deviation or an early injury to areas of the brain that are specialized for language functions. In expressive dysphasia, children have difficulty with verbal expression but have adequate language comprehension skills. Difficulties can be noted in putting words into coherent sentences, using appropriate grammar, and recalling words. Expressive dysphasia is often observed in children with autism spectrum disorders (ASD) and can be noted across the spectrum regardless of severity. Issues noted specifically in children with ASD include difficulties with word order in sentences (syntax), difficulties using appropriate word morphology (word endings) and grammar (especially pronouns), as well as noted difficulties in semantic organization and word finding.

Exploratory Factor Analysis

- [Latent Variable Modeling](#)

See Also

- [Broca's Aphasia](#)
- [Childhood Aphasia](#)
- [Expressive Language](#)
- [Expressive Language Disorder](#)

References and Reading

- De Hirsch, K. (1982). Differences in language acquisition: Dysphasia-autism. *Annals of Dyslexia*, 32(1), 305–320.
- Martos, J., & Ayuda, R. (2002). Communication and language in the autistic spectrum: Autism and dysphasia. *Revista de Neurologia*, 34(1), 58–63.
- Resnick, T., Allen, D., & Rapin, I. (1984). Disorders of language development: Diagnosis and intervention. *Pediatrics in Review*, 6, 85–92.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. I, pp. 335–364). Hoboken: Wiley.
- Tuchman, R., Rapin, I., & Shinnar, S. (1991). Autistic and dysphasic children. I: Clinical characteristics. *Pediatrics*, 88(6), 1211–1218.

Expressive Language

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Synonyms

[Expressive communication](#); [Oral language](#); [Spoken language](#)

Definition

Expressive language is a cognitive process that is involved in the transmission of oral, symbolic, or written language and allows for communicating

one's ideas, desires, or intentions to others. Expressive language is composed of form (grammar and syntax), content (semantics or meaning), and use (communicative function and intent). Expressive language can take many forms including verbalizations, sign language, gestures, pictures, written words, and, in some cases, voice output communication devices. Individuals with autism spectrum disorders (ASD) can have difficulty with expressive language in any of the aforementioned areas, especially in the area of language use.

See Also

- [Communicative Functions](#)
- [Expressive Language Disorder](#)
- [Language](#)
- [Speech Morphology](#)

References and Reading

- Geurts, M., & Embrechts, M. (2008). Language profiles in ASD, SLI, and ADHD. *Journal of Autism and Developmental Disorders*, 38(10), 1931–1943.
- Jarrold, C., Boucher, J., & Russell, J. (1997). Language profiles in children with autism: Theoretical and methodological implications. *Autism*, 1, 57–76.
- Rescorla, L., & Schwartz, E. (1990). Outcomes of toddlers with specific expressive language delay. *Applied Psycholinguistics*, 11, 393–407.
- Tager-Flusberg, H., & Caronna, E. (2007). Language disorders: Autism and other pervasive developmental disorders. *Pediatric Clinics of North America*, 54(3), 469–481.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. I, pp. 335–364). Hoboken: Wiley.

Expressive Language Delay

- [Expressive Language Disorder](#)

Expressive Language Disorder

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Synonyms

[Developmental language delay/disorder](#); [Expressive language delay](#); [Specific language impairment](#)

Definition

Expressive language disorder indicates an individual is likely to understand more than what he/she is able to communicate. This means a person's receptive language (comprehension) is better than his/her expressive language (use of language). This disorder is often associated with a developmental language delay. Expressive language disorders can also be acquired (as a result of brain injury/damage), as in aphasia. A developmental delay in expressive language is more common in children and can occur in a child with normal intelligence or co-occur with cognitive impairment or other developmental disorders a child might experience. Expressive language disorder can be observed in both verbal and written communication forms. Children with autism spectrum disorders (ASD) often present with expressive language disorders. Many children with ASD begin speaking later than typically developing children and often demonstrate a significantly slower development of expressive language through the preschool years. In particular, children with ASD present with differences in the social use of language (pragmatics).

See Also

► [Communication Disorder/Communication Impairment](#)

- [Expressive Dysphasia](#)
- [Speech Delay](#)
- [Speech Impairment](#)
- [Speech/Communication Disabilities](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed.). Washington, DC: APA Press. Text Rev.
- Geurts, M., & Embrechts, M. (2008). Language profiles in ASD, SLI, and ADHD. *Journal of Autism and Developmental Disorders*, 38(10), 1931–1943.
- Tager-Flusberg, H., & Caronna, E. (2007). Language disorders: Autism and other pervasive developmental disorders. *Pediatric Clinics of North America*, 54(3), 469–481.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. I, pp. 335–364). Hoboken: Wiley.
- Toth, K., Munson, J., Meltzoff, A., & Dawson, G. (2006). Early predictors of communication development in young children with autism spectrum disorder: Joint attention, imitation, and toy play. *Journal of Autism and Developmental Disorders*, 36(8), 993–1005.

Expressive Vocabulary Test II

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Synonyms

[EVT-2](#)

Description

The *Expressive Vocabulary Test, Second Edition* (EVT-2) is a quick, norm-referenced assessment designed to measure expressive vocabulary and word retrieval for Standard American English. Norms are provided for a wide age range (i.e., 2 years and 6 months to beyond 90 years of age). Items require examinees to provide either

labels or synonyms. For each item, examinees are shown a colored picture and prompted by the examiner to provide a one-word response (e.g., “What is this animal?” or “Tell me another word for jacket.”). The EVT-2 is co-normed with the *Peabody Picture Vocabulary Test, Fourth Edition* (PPVT-4; Dunn and Dunn 2007), a measure of receptive vocabulary (i.e., understanding of spoken words). When both the PPVT-4 and EVT-2 are administered, direct comparisons can be made between a client’s receptive and expressive vocabulary. The EVT-2 has two parallel test forms (A and B) so it may be used for progress monitoring. EVT-2 materials include an examiner’s manual, test easel (either form A or B), and 25 record forms (additional forms can be purchased separately). Users can choose to purchase ASSIST, a software program for electronic scoring, which provides various types of group and individual reports. Examiners administering the EVT-2 must be trained and experienced in test administration and scoring. Only qualified educational and psychological professionals should interpret the test results.

The EVT-2 manual provides clear guidelines for administration and scoring. Each form (A or B) contains 190 test items presented in order of increasing difficulty. In addition, two practice items are presented to ensure the client understands the procedure. The EVT-2 is administered individually, and testing time is estimated to be 10–20 min, depending on age and ability (e.g., individuals with larger vocabularies tend to require more testing time). Start points are based on the client’s chronological age. The basal rule is five consecutive correct responses, and the ceiling rule is five consecutive incorrect responses. A stimulus prompt and all possible correct answers are provided for each item on the score form. The most common incorrect answers are also listed. Some incorrect answers are tagged with a “P” indicating that the examiner should provide an additional prompt to encourage a correct response from the examinee. The manual does not provide an explanation as to why some incorrect answers are prompted, while others are not. Although the test is officially untimed, the manual suggests a 10^{-s} limit for responding to

each item, especially if the EVT-2 is to be used as a measure of word retrieval. Items are scored as incorrect (“0”) or correct (“1”) on the score form during test administration. Space is provided for examiners to write responses that are not listed on the score form for later scoring.

The EVT-2 provides numerous options for converting the data and guidelines for interpreting the results. First, raw scores are calculated by taking the highest item in the client’s ceiling and subtracting errors for all items administered. Items below the basal are considered to be correct. Raw scores are converted to standard scores (mean = 100, standard deviation = 15) based on either age or grade (fall or spring). A raw score can also be converted to a growth scale value (GSV) which is helpful for tracking a client’s vocabulary development over time. The GSV is an indicator of absolute level of performance rather than a norm-referenced score. If a client’s score increases on the EVT-2 upon subsequent administrations, then his or her GSV will also increase (versus norm-referenced scores which may decrease over time if a client’s growth is below average compared to his or her peers). Tables are also provided for converting standard scores to percentile ranks, normal curve equivalents, and stanine scores. If the PPVT-4 was also administered, tables are provided to determine whether the absolute difference between the EVT-2 and PPVT-4 scores is statistically significant.

The EVT-2 also provides tools for conducting various qualitative interpretations of a client’s performance. These analyses include expressive versus receptive vocabulary, home versus school vocabulary, vocabulary by part of speech (i.e., noun, verb, attribute), vocabulary by tier using a three-tier model (i.e., tier 1 – vocabulary common in conversation, tier 2 – vocabulary common in school curricula, tier 3 – technical vocabulary; Beck et al. 2002), and crossover vocabulary sampling between the EVT-2 and PPVT-4 (i.e., comparing items common to both assessments). Worksheets are provided in the test manual to complete these qualitative analyses by hand, or the ASSIST software will compute them upon entering a client’s performance item by item.

Historical Background

The previous edition of this assessment was the *Expressive Vocabulary Test* (EVT; Williams 1997). The first step in the 5-year revision process was to survey and interview current users of the EVT to gather ideas for improvements. In addition, a panel of ten consultants representing the perspectives of several ethnic minorities was formed to examine the test for cultural and linguistic biases. Offensive items and items that were potentially biased were eliminated. Several updates were subsequently made to the EVT: two parallel test forms were developed, more words representing everyday vocabulary were included, more words found in oral directions given in the classroom were included, labeling-type items were included throughout the range of difficulty (versus just synonym types at the higher age ranges), a specific stimulus question was included for each item on the score form, several items were dropped, all of the stimulus pictures were updated, and the EVT-2 was co-normed with the PPVT-4.

In order to create a parallel test form, many new words needed to be added. Two guidelines were followed for choosing which words were included in the initial tryout pool. The first was to choose words that represented Standard American English vocabulary encountered in typical life experiences. The second guideline was to choose words that occurred with relatively high frequency (in order to measure word retrieval). Several published lists of word frequencies were consulted for the selection of new words (see the test manual for the specific references). A set of 400 items were eventually chosen for the two national tryout studies. Guidelines for scoring were based on the results of the first tryout study of 1,451 participants and also based on several reference materials (e.g., dictionaries and thesauruses). Specific details on choosing correct and incorrect responses were not provided in the manual. Items were subsequently analyzed in terms of item difficulty, item discrimination (i.e., ability to differentiate examinees with lower and higher vocabulary levels), and item bias (i.e., examining if an item was more difficult for one group of examinees than another). The items were reviewed

by a second panel of 15 individuals representing various ethnic backgrounds to examine cultural sensitivity and fairness with inappropriate items subsequently eliminated. Next, a second national tryout with 852 participants was conducted, and similar item analyses were completed. Based on the two tryout studies, the EVT-2 standardization versions were created and norms were developed.

Psychometric Data

The sample used to standardize the EVT-2 and create the test norms consisted of 3,540 individuals at 320 sites nationwide ranging in age from 2 years and 6 months to over 90 years of age. The demographic characteristics of the participants (i.e., gender, ethnicity, socioeconomic status, geographic region, special education status) closely resembled the larger population of the United States according to the 2004 US Census. The sample was divided into 28 age groups, with 60 to 200 individuals within each group (all but the two oldest groups contained at least 100 individuals). Grade norms are based on a subset of 2003 cases with 100 to 233 students per grade. Approximately half of the participants were administered form A of the EVT-2 and half completed form B. A subset of 507 participants were administered both forms. Mean scores across age groups indicate no floor or ceiling effects for either form. In addition, the test manual provides evidence suggesting that the two forms are of similar difficulty.

Reliability of the EVT-2 was assessed through analyses measuring internal consistency (i.e., split-half and alpha reliabilities), alternate-form reliability, and test-retest reliability. Measures of internal consistency were calculated for the 28 age groups and for both EVT-2 forms (A and B). Split-half reliability coefficients were high, ranging from 0.88 to 0.97, and alpha coefficients were also high, ranging from 0.93 to 0.98, indicating a high degree of internal consistency. Alternate-form reliability resulted in correlations ranging from 0.83 to 0.91 for the subset of participants who were given both forms A and B, indicating that the two forms provide similar results. Another

subgroup of 348 examinees across five age groups was given the EVT-2 twice with an interval of 2 to 6 weeks between administrations. The resulting test-retest reliability was high, with coefficients ranging from 0.94 to 0.97, suggesting excellent stability of scores. Inter-rater reliability was not reported.

The validity of the EVT-2 was assessed in terms of content validity, its relationship to age, and correlations to other tests (i.e., criterion-related validity). In addition, its sensitivity to measuring differences in various special populations was examined. The author refers to the process of item selection (discussed above) as evidence for content validity. As stated earlier, items were chosen if they were of moderate to high frequency according to various published resources and if they represented everyday experiences. In addition, items were reviewed by content specialists and panels examining cultural and linguistic biases. No more detail on the processes of item generation or selection is provided. Also, the manual does not report mean scores by race or ethnicity which would provide additional evidence for the cultural and linguistic fairness of the test. EVT-2 mean test scores increase rapidly in childhood, while growth is gradual in adolescence and young adulthood. Scores plateau through middle adulthood and then decline gradually at older ages. This pattern of results is what would be expected given what is known about cognitive and linguistic development. Therefore, the EVT-2 appears to be sensitive to age-related changes in expressive vocabulary skill. In terms of criterion-related validity, the relationships between the EVT-2 and other measures of language and reading were examined using subsets of the norming sample (consisting mostly of children and adolescents), including the PPVT-4, the *Comprehensive Assessment of Spoken Language* (CASL; Carrow-Woolfolk 1999), the *Clinical Evaluation of Language Fundamentals, Fourth Edition* (CELF-4), the *Group Reading Assessment and Diagnostic Evaluation* (GRADE; Williams 2001), and the EVT. Results indicated that the EVT-2 was moderately to highly correlated with these other measures, providing evidence that it is a valid measure of language skill.

The performance of 12 special populations on the EVT-2 was also reported. These populations included children or adults with speech impairment, young children with language delay, children or adults with language disorder, children with hearing impairment (divided into those with and without cochlear implants), and children with either learning disability (reading), mental retardation, emotional/behavioral disturbance, attention deficit disorder, or giftedness. Each group's mean score fell above, at, or below the mean as would be expected based on group membership, suggesting that the EVT-2 is a sensitive measure of vocabulary skill in special populations. The manual does not provide sensitivity or specificity data for diagnostic classification, which is considered to be the gold standard when using assessment tools for diagnostic purposes (Spaulding et al. 2006). Moreover, the average score for children with language delay or disorder fell within one standard deviation of the mean. Therefore, the results of the EVT-2 alone should not be used for making diagnostic decisions about language delay or disorder.

Clinical Uses

The EVT-2 is a quick assessment of expressive vocabulary skill and word retrieval for children and adults who speak Standard American English. In addition, the EVT-2 is sensitive to age-related changes in vocabulary skill and discriminates among special populations. Several qualitative interpretations are also available (although no evidence for the validity of these interpretations is provided). The EVT-2 was co-normed with the PPVT-4, so comparisons between expressive and receptive vocabulary can be made if both assessments are administered. The optional software program, ASSIST, provides group and individual reports in addition to evidence-based intervention ideas. Overall, the EVT-2 appears to be a psychometrically sound assessment of expressive vocabulary. However, given that data on classification accuracy for children with language delay or disorder were not provided, caution should be taken in using the EVT-2 for diagnostic classification.

The EVT-2 included 4 individuals with autism in its norming sample, or 0.2%, which was reflective of the prevalence of autism in the 2004 US population. It is unknown if individuals included in the other special population groups (e.g., language disorder, mental retardation) may have been on the autism spectrum. If both the PPVT-4 and EVT-2 are administered to a client, a higher expressive than receptive vocabulary score (i.e., EVT-2 > PPVT-4) could be an indicator of ASD (Paul et al. 2008).

See Also

- ▶ [Communication Assessment](#)
- ▶ [Expressive Language](#)
- ▶ [Expressive Language Disorder](#)
- ▶ [Language Tests](#)
- ▶ [Norm-Referenced Testing](#)
- ▶ [Peabody Picture Vocabulary Test](#)
- ▶ [Standardized Tests](#)

References and Reading

- Beck, I. L., McKeown, M. G., & Kucan, L. (2002). *Bringing words to life: Robust vocabulary instruction*. New York: Guilford Press.
- Carrow-Woolfolk, E. (1999). *Comprehensive assessment of spoken language*. Circle Pines: American Guidance Service.
- Dunn, L. M., & Dunn, D. M. (2007). *Peabody picture vocabulary test* (4th ed.). Bloomington: NCS Pearson.
- Graham, T. (2010). Review of the expressive vocabulary test, second edition. In R. A. Spies, J. F. Carlson, & K. F. Geisinger (Eds.), *The eighteenth mental measurements yearbook* (pp. 223–226). Lincoln: Buros Institute of Mental Measurements.
- Paul, R., Chawarska, K., & Volkmar, F. (2008). Differentiating ASD from DLD in toddlers. *Perspectives on Language Learning and Education*, 15, 101–111.
- Rathvon, N. (2010). Review of the expressive vocabulary test, second edition. In R. A. Spies, J. F. Carlson, & K. F. Geisinger (Eds.), *The eighteenth mental measurements yearbook* (pp. 226–229). Lincoln: Buros Institute of Mental Measurements.
- Semel, E., Wiig, E. H., & Secord, W. A. (2003). *Clinical evaluation of language fundamentals* (4th ed.). San Antonio: Psychological Corporation.
- Spaulding, T., Plante, E., & Farinella, K. (2006). Eligibility criteria for language impairment: Is the low end of normal always appropriate? *Language, Speech, and Hearing Services in Schools*, 37, 61–72.
- Williams, K. T. (1997). *Expressive vocabulary test*. Circle Pines: American Guidance Service.
- Williams, K. T. (2001). *Group reading assessment and diagnostic evaluation*. Circle Pines: American Guidance Service.
- Williams, K. T. (2007). *Expressive vocabulary test* (2nd ed.). Minneapolis: NCS Pearson.

Extended School Year

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The traditional (in the USA) 9-month academic year can present challenges to children needing ongoing support. In the past, parents frequently had to arrange a patchwork of supports over the summer months. However, for children with disabilities that qualify them for an IEP who may be in demonstrable need of ongoing services, **extended school year (ESY)** services are available. These services are designed to support a student with a disability as documented under the Individuals with Disabilities Education Act (IDEA) to maintain the academic, social/behavioral, communication, or other skills that they have learned as part of their Individualized Education Program (IEP) or Section 504 accommodation plan. These services are outlined in the IEP and are provided at no charge to parents. For children on the autism spectrum, these services often include ongoing speech-communication, special educational supports, occupational and physical therapy, and so forth.

It is emphasized that in order for a student to receive ESY services, the student must have evidenced substantial regression and recoupment issues during the previous IEP year and/or there is evidence of emerging skills, which are often

referred to as “breakthrough” skills. The focus of the services provided to the student as part of an ESY program is generally not upon learning new skills or “catching up” to grade level, but rather to provide practice to maintain previously acquired or learned skills. In some cases, ESY is focused on continuing education for students whose rate of progress is insufficient to enable effective progress during the regular school year. If a student has received ESY services in previous years, the student may not be eligible in future years as determinations for eligibility of ESY services are made annually by the IEP or 504 plan (which includes the parent and student of age 16 or older. *Note:* The mandatory age at which a student must be included varies by State but the Federal law states no later than age 16.).

Some children with autism may lose skills that they have learned (e.g., regress) if not provided with continuous programming (i.e., they regress over the long traditional summer vacation from school). Extended school year services (often referred to simply as ESY) can include both classroom-based services and related services such as occupational or speech therapy. These special services are not simply the same as summer school or summer enrichment programs and indeed are not solely limited to summer, e.g., other periods might also be appropriate. Any student receiving special education may be eligible for such services although the need, for a specific child, is determined by his or her IEP team (which includes the parents).

It should also be noted that the simple provision of a “summer program” or “summer school” is not a simple way to meet the requirements for an extended school year. That is, ESY are individualized and based on the specific programmatic needs of the individual child, whereas general summer enrichment or remedial programs are not typically individualized.

In the USA, under the IDEA mandate, these services must meet acceptable state standards and be individualized to meet each child’s specific needs. Many of the mandates/guidelines in the area have evolved based on legal decisions. Criteria frequently used to determine need include, but are not limited to, the child’s current

rate of progress, interfering behavioral concerns, opportunities to master emerging skills, the likelihood of regression, and the time that it might take the child to regain skills that would be lost.

It is sometimes the case that schools (often private special needs school) provide a full year of service with an IEP in place for the entire period. In such cases, ESY services would not be required.

See Also

- [Education](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Regression](#)

References and Reading

- National Research Council. (2001). *Educating young children with autism*. Washington, DC: National Academy Press.
- Volkmar, F., & Wiesner, L. (2009). *A practical guide to autism*. Hoboken: Wiley.
- Wrisht, P. W., & Wright, P. D. (2007). *Wrightslaw: Special education law* (2nd ed.). Deltaville: Harbor House Law Press.
- Yell, M. L. (2011). *The law and special education* (3rd ed.). Boston: Pearson.

External Validity

- [Ecological Validity](#)

Extinction Procedures

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Definition

Extinction refers to the process of discontinuing the reinforcer that historically follows a behavior (Cooper et al. 2007).

Historical Background

In operant psychology, behavior is influenced by both the stimuli that occur prior to and following it. The stimuli that follow behaviors are consequences. Consequences influence behavior in three ways – consequences can increase (strength rate, frequency, etc.), decrease, or have no influence on strength. A reinforcing consequence results in an increased strength (or frequency or rate) of that behavior. Similar to the presentation of a reinforcer strengthening a behavior, removal of the reinforcer (or preventing it from occurring) that follows a behavior will have a weakening effect on that behavior; it will reduce in strength (or frequency or rate).

Historically, the treatment of challenging behaviors that interfere with developing independence or adaptive skills has focused on the application of punitive consequences that reduce or eliminate the target behavior. It has always been true that extinction is also relevant in this context. Over the past 15 years, the use of extinction has been explored as an alternative to aversive treatments and has been shown to effectively reduce behavior under certain conditions.

Current Knowledge

Extinction is withholding of reinforcement that typically follows a behavior and that served to maintain the behavior. However, the actual form that an extinction procedure will follow depends upon the form of the reinforcement that will be withheld. The reinforcers that maintain aberrant behaviors are not uniform or singular; behaviors serve different functions and are maintained by multiple potential effects. Sometimes, a behavior will lead to the acquisition of desirable consequences, such as access to attention, desired objects, preferred activities, or internal, physiological (e.g., sensory) stimulation. Consider a child who acts out in class to obtain teacher attention. The child engages in disruptive behavior in order to garner teacher attention. (In fact, teachers must often attend to such disruptions to ensure minimal intrusion into the flow of instruction.)

In this case, an extinction procedure would involve the teacher ignoring the behavior or in a similar case, consider a child who aggresses in order to obtain a toy or other preferred object. The child may aggress against a sibling and then gain access to a preferred toy. In this case, extinction would consist of preventing the child from obtaining that object contingent on aggression. Another example would be a child who acts out in order to get a preferred activity. For example, consider a child who is on the playground and, when told by the teacher that recess is ended and the child must go back inside, throws tantrums and cries. Historically, it could be the case that this behavior leads to more time on the playground. For example, the teacher might cajole the child, offer 5 more minutes, give several transition warnings, etc. An extinction procedure would consist of the teacher preventing the child from remaining on the playground. Instead, the child would be made to come inside.

Some behaviors exhibited by persons with autism are labeled as “self-stimulatory,” due to the belief that these behaviors are reinforced immediately by specific sensory input. For example, a child might wave her hand in front of her face because the visual movement is highly desired. These behaviors are also termed as “automatically reinforcing” behaviors because of their inherent sensory consequences that are presumably maintaining the behavior. Extinction in this type of example would require that the sensory input that the individual finds highly desirable is identified and prevented from happening. For example, Rincover et al. (1979) used extinction to stop the self-stimulatory behaviors of young children. One boy engaged in spinning plates on a table and enjoyed listening to the sound they made. The extinction procedure consisted of carpeting the surface on which he spun the plate. The carpet masked the sound, and thus plate spinning stopped. Another child engaged in flipping a light switch on and off, presumably for the visual feedback of the light. The extinction procedure for this behavior consisted of disconnecting the switch from the electrical source. From that point on, the behavior of flipping the switch produced no visual consequence, and the behavior eventually

stopped. In such sensory extinction procedures, the feedback is either reduced or eliminated, and the behavior diminishes.

With other situations, the reinforcement for a behavior could be the escape or avoidance of an aversive situation. Consider a child who throws tantrums when presented a difficult task. Historically, the tantrum is reinforced by the removal of the task. (A teacher may react to the tantrum by removing the task, changing the task, providing a break, etc.) Thus, in this particular example, the extinction procedure would involve the prevention of the termination of the task; that is, extinction in this case would consist of the teacher continuing to present the task, so that the child learns that throws tantrums will not be reinforced by task removal. An example of the well-documented use of escape extinction is in food refusal and food selectivity interventions (Dawson et al. 2003; LaRue et al. 2011; Patel et al. 2002; Piazza et al. 2003).

As noted above, if the behavior is followed by the *presentation* of a reinforcer, such as attention, preferred activities, or objects, then extinction is implemented by preventing the delivery of those reinforcing consequences or terminating them as soon as possible. On the other hand, if the behavior is reinforced by the cessation of something unpleasant (consider a child asked to do a difficult work task who has learned that a tantrum will cease the work demand), then extinction requires the continued presentation of the unpleasant stimulus. In the work demand case, extinction would require that the teacher continually present the work demand and make sure that no escape or avoidance of the task is possible. The teacher would be sensitive to even small ways in which escape could be reinforced. For example, a delay in the representation of the instruction or a brief pause in instruction could reinforce escape-motivated behavior.

Thus, it is important to understand that extinction is a procedure that prevents the reinforcement – that has typically followed a behavior – from occurring. However, since there are different types of reinforcement for behavior, the actual procedures for implementing extinction may vary.

Research on extinction has identified several effects of this process. One effect, which has been shown to generalize across response classes and settings, is a gradual decrease in rate, frequency, or intensity of behavior. Although the behavior that is extinguished will eventually return to baseline (i.e., pretreatment) levels or be eliminated altogether, the rate of decrease is slow. If a behavior is extremely severe and immediate cessation is imperative, extinction may not be the best procedure to select.

Concomitant with this gradual decrease is what is termed an “extinction burst,” a sudden increase in strength (frequency, rate, and/or intensity) of that behavior. Problem behaviors could worsen before they reduce. For example, with a young child who is crying at bedtime, parents reentering the room to comfort the child and then exiting often intensify the screaming. It may lead to escalations of the behavior and new forms of behavior during the burst that evening, and future nights may be characterized by even higher levels of the behavior. Essentially, the parents going back into the bedroom to comfort the child may reinforce screaming when being put to bed at night. Once the parents stop reentering the room, the crying may increase in both frequency and intensity for a short while. When the burst occurs, it is likely a confirmation that the controlling reinforcer has in fact been identified and successfully withheld following the occurrence of the response. If the extinction plan stays in effect, the behavior will once again reduce in strength, but some variations of an extinction burst may occur for several sessions once an extinction plan is put into place. In other words, there could be several days with some variability before the behavior is extinguished.

A phenomenon commonly associated with extinction is that of “spontaneous recovery.” The behavior is likely to recur the next day after the first day of using extinction even though the reinforcement for that behavior is no longer occurring. When this happens, the typical response is to maintain the implementation of extinction. Each day that goes by, the spontaneous recovery will be a little less, and eventually the behavior will no longer occur. This can also

happen after a behavior has been successfully extinguished, even in the absence of new reinforcement for the behavior. This can be very confusing for parents and teachers. Generally, however, spontaneous recovery is short-lived and consistent implementation of extinction will successfully address the issue.

To implement extinction, the following conditions must be met. First, the behavior must be clearly defined so that all caregivers and interventionists know when it is occurring and when it is not. Second, there should be some preextinction data on the level of the behavior, against which postextinction data can be compared, to determine if the extinction program is having the reductive effect that is intended. Third, all reinforcers that maintain the behavior must be clearly identified. This will likely require conducting a functional assessment or preferably a functional analysis (i.e., systematic manipulation of potential consequences). Fourth, a plan for either terminating the reinforcer when it occurs, or preventing it from occurring in the first place, must be developed. This is often the most difficult part of implementing extinction. Often the reinforcer is identified but with little ability to prevent it from occurring. For example, consider a child who teases other peers in his classroom for attention. The teachers may correctly identify the reinforcer as attention of the other children, but there may be a huge difficulty (and ethical considerations) in preventing all of the children in the classroom from attending to the misbehavior. If alternate sources of reinforcement remain available for the target behavior, extinction is not likely to be successful. Fifth, the people who will implement the extinction plan must consider whether they will be able to continue the extinction plan when the extinction burst occurs. Consider the following scenario – a child with autism has a history of throwing tantrums violently when a difficult work task is presented. His teacher is confident that the escape from the work demands is reinforcing the tantrum. The teacher develops a plan for continually presenting the work (i.e., making sure the work demands do not cease). However, the teacher also considers how much more violent the throws tantrums may become during the

extinction burst. For extinction to work effectively, the plan must be put into place upon the occurrence of the target behavior, regardless of the severity of the extinction burst. The teacher and other implementers must plan on how to react to the possible increase in frequency and intensity. If a child is likely to engage in topographies of behavior that are unacceptable and not ignorable (e.g., throwing desks, self-injury), extinction is not an appropriate choice.

Finally, the implementers must consistently withhold the reinforcers each time the target behavior occurs. This is one of the most difficult parts of implementing extinction, and it is one of the more critical aspects of the procedure. Failure to implement extinction contingent upon each occurrence will result of the target behavior being reinforced occasionally, which is intermittent reinforcement. Research has shown that behaviors that are intermittently reinforced are much more difficult to extinguish than behaviors on a continuous schedule. So, when implementing extinction, failure to withhold reinforcement for each behavioral occurrence could likely result in the behavior becoming even stronger in the person's repertoire. If some reinforcement is likely to occasionally occur, extinction may not be the best method to select.

Good practice suggests that extinction be implemented in combination with other procedures, such as differential reinforcement of other behaviors, noncontingent reinforcement, or differential reinforcement of alternative behaviors. Research suggests that when used in combination, extinction may increase the effectiveness of other procedures. Additionally, the use of reinforcement procedures to strengthen appropriate behaviors is part of best clinical practice.

Future Directions

Extinction is considered a treatment for problem behaviors that is not aversive and acceptable to most staff. It is not as regulated as other more intrusive procedures, such as time-out or over-correction. Although researchers have investigated the role of clinical treatment with and without implementing extinction procedures, the

results remain ambiguous. Identification of behavior classes and/or environmental conditions under which extinction is necessary or optimal in clinical application would be helpful. Clinically, it is important to match the procedure of extinction to certain behavioral characteristics and learners. Clinicians must be mindful of controlling all sources of reinforcement and of monitoring behavioral escalations in bursts.

See Also

- [Functional Analysis](#)
- [Functional Assessment](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Dawson, J. E., Piazza, C. C., Sevin, B. M., Gulotta, C. S., Lerman, D., & Kelley, M. L. (2003). Use of the high-probability instructional sequence and escape extinction in a child with food refusal. *Journal of Applied Behavior Analysis*, 36(1), 105–108.
- LaRue, R. H., Stewart, V., Piazza, C. C., Volkert, V. M., Patel, M. R., & Zeleny, J. (2011). Escape as reinforcement and escape extinction in the treatment of feeding problems. *Journal of Applied Behavior Analysis*, 44(4), 719–735.
- Patel, M. R., Piazza, C. C., Martinez, C. J., Volkert, V. M., & Santana, C. M. (2002). An evaluation of two differential reinforcement procedures with escape extinction to treat food refusal. *Journal of Applied Behavior Analysis*, 35(4), 363–374.
- Piazza, C. C., Patel, M. R., Gulotta, C. S., Sevin, B. M., & Layer, S. A. (2003). On the relative contributions of positive reinforcement and escape extinction in the treatment of food refusal. *Journal of Applied Behavior Analysis*, 36(3), 309–324.
- Rincover, A., Cook, R., Peoples, A., & Packard, D. (1979). Sensory extinction and sensory reinforcement principles for programming multiple adaptive behavior change. *Journal of Applied Behavior Analysis*, 12(2), 221–233.

Extraordinary

- [Exceptionality](#)

Extreme Demand Avoidance

- [Pathological Demand Avoidance \(PDA\)](#)

Extreme Male Brain (EMB) Theory

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Definition

Autism spectrum conditions (ASC) have been described as an extreme manifestation of certain traits or as a consequence of an “extreme male brain.”

Historical Background

The link between ASC and “maleness” was first proposed by Hans Asperger in his 1944 clinical account where he states, “The autistic personality is an extreme variant of male intelligence. . . In the autistic individual the male pattern is exaggerated to the extreme.” More recent evidence has led to the proposal that ASC may be an exaggeration of certain male-typical characteristics (Baron-Cohen 2002; Baron-Cohen et al. 2005).

Current Knowledge

Autism spectrum conditions (ASC) are characterized by impairments in social interaction and communication, alongside unusually restricted, repetitive, stereotyped patterns of behavior, interests, and activities. The American Psychiatric Association uses the term ASD for autism spectrum disorders. The term ASC will be used as those at the higher-functioning end of the autistic

spectrum do not necessarily see themselves as having a “disorder,” and the profile of strengths and difficulties in ASC can be conceptualized as atypical but not necessarily disordered. ASC remains a medical diagnosis, hence the use of the term “condition,” which signals that such individuals need support. Use of the term ASC recognizes that the profile in question does not fit a simple “disease” model but includes areas of strength (e.g., in attention to detail) as well as areas of difficulty and does not identify the individual purely in terms of the latter.

Approximately 1% of children have a diagnosis of ASC. These conditions have a strong neurobiological and genetic component. There is also a clear male to female ratio in the incidence of ASC, estimated at 4:1 for classic autism and over 10:1 in individuals with Asperger syndrome. The cause of the male bias in ASC is not fully understood. Many clinical conditions occur in males more often than females, including autism, dyslexia, specific language impairment, attention-deficit/hyperactivity disorder (ADHD), and early-onset persistent antisocial behavior. Depression, anorexia, and the anxiety disorders show a female bias in sex ratio, raising the question of whether there are sex-linked or sex-limiting factors involved in the etiology of conditions that exhibit a male bias.

ASC in particular have been described as an extreme manifestation of certain sexually dimorphic traits or as a consequence of an “extreme male brain” (EMB) (Baron-Cohen 2002; Baron-Cohen et al. 2005). Individuals with ASC have been shown to be impaired in empathy (the drive to identify another person’s emotions and thoughts, and to respond to these with an appropriate emotion and an area where females show an advantage), while being average or even superior in systemizing (the drive to analyze, explore, and construct a system and an area males show an advantage).

Studies of Empathy and Systemizing

It is widely accepted that males and females show significant differences in their neuroanatomy,

cognition, and behavior from an early age. Sex differences in the precursors of empathy are seen from birth, with female babies on average showing a stronger preference for looking at social stimuli (faces) from 24 h after birth (Connellan et al. 2000). Girls have also been found to make more eye contact immediately after birth (Hittelman and Dickes 1979), at 12 months of age (Lutchmaya et al. 2002a) and at 2 and 4 years of age (Podrouzek and Furrow 1988). Girls on average also exhibit more comforting, sad expressions or more sympathetic vocalizations when witnessing another’s distress (Hoffman 1977). Girls on average also show better quality of social relationships at 48 months, as measured by a subscale of the Children’s Communication Checklist. Similar patterns have been observed in adults, with women on average being more likely to report more intimate relationships, having a confidant and receiving social support and visits from friends and family (Baron-Cohen and Wheelwright 2003).

Using measures that directly assess aspects of empathy, girls are on average better than boys at evaluating the feelings and intentions of characters in a story (Bosacki and Astington 1999) and differentiating between the appearance and reality of emotion (Banerjee 1997). There is also a female superiority on the “faux pas” test of social sensitivity (Baron-Cohen et al. 1999) which measures the recognition of someone saying something that might be hurtful. Sex differences in empathy remain evident in adulthood: for example, women on average score higher than men on the “Reading the Mind in the Eyes” test, which examines subtle mental state and emotion recognition (Baron-Cohen et al. 2001).

In general, studies have shown that individuals with ASC are also impaired on empathy-related tasks that normally give rise to female superiority, such as the “Social Stories Questionnaire” (Lawson et al. 2004), the “Reading the Mind in the Eyes” task (Baron-Cohen et al. 2001), and the recognition of “faux pas” in short stories (Baron-Cohen et al. 1999). Adults with ASC score lower on the Friendship and Relationship Questionnaire, which assesses empathic styles of relationships (Baron-Cohen and Wheelwright 2003).

Children with autism perform less well than controls on the “Feshbach and Powell Audiovisual Test for Empathy,” a measure of empathy and emotional responsiveness (Yirmiya et al. 1992). Children with ASC also show more difficulties passing “theory of mind” tests compared to typically developing children (Happé 1995).

Studies examining play preferences point toward more interest in mechanical and constructional play in boys, demonstrated by a preference to play with toy vehicles or construction sets, while girls are more likely to choose to play with dolls or toy animals (Berenbaum and Hines 1992). Males on average also score higher on tasks that require systemizing such as using directional cues in map reading and map making (Kimura 1999), intuitive physics (Lawson et al. 2004), and the SAT Math Test (Benbow and Stanley 1983). They are also more accurate on measures of spatial ability such as mental rotation and spatial visualization (Voyer et al. 1995). Finally, males on average score higher on the Embedded Figures Test (EFT) (Witkin et al. 1962), which measures attention to detail and field independence – both prerequisites for systemizing (Baron-Cohen 2002).

Experimental evidence supporting the EMB theory of autism includes findings that individuals with ASC tend to show superior performance compared to typical controls on tasks that involve systemizing and on certain visuospatial tasks that normally give rise to male superiority, such as figure disembedding (Falter et al. 2008; Jolliffe and Baron-Cohen 1997; Ropar and Mitchell 2001; Shah and Frith 1983), block design (Ropar and Mitchell 2001; Shah and Frith 1993), and mental rotation (Brosnan et al. 2009; Falter et al. 2008).

Brosnan et al. (2009) suggest that mental rotation tasks can be separated into rotational and non-rotational components and observed a significant correlation between systemizing and the non-rotational components of the mental rotation task but not the rotational component of the task (Brosnan et al. 2009). Individuals with high-functioning autism (and therefore intact IQ) have also been observed to demonstrate superior accuracy and shorter learning times in tasks that involve maps (Caron et al. 2004).

A recent study by Pierce et al. (2010) found that toddlers with an ASD as young as 14 months spent significantly more time fixating on dynamic geometric (“systemizable”) images, whereas typically developing toddlers showed longer looking times at social stimuli. Further, if a toddler spent more than 69% of his or her time fixating on geometric patterns, then the positive predictive value for accurately classifying that toddler as having an ASD was 100% (Pierce et al. 2010). These findings suggest that these early looking preferences can be found very early in life and used to differentiate toddlers with ASC from typically developing toddlers.

However, the EMB theory has not been shown to apply to all measures showing a male advantage. For example, Falter et al. (2008) found that children with autism do not show superior performance on a measure of targeting ability compared to typically developing boys. It is possible that problems with motor coordination (dyspraxia) in the ASC group may have affected performance on this task. It is worth emphasizing that the EMB theory predicts intact or superior performance on measures of systemizing in ASC and that the EMB theory does not focus on systemizing alone, but on the discrepancy between an individual’s empathy and systemizing abilities.

The Empathy Quotient (EQ) and Systemizing Quotient (SQ)

The Empathy Quotient (EQ) and Systemizing Quotient (SQ) were developed in order to examine trends in gender-typical behavior in adults. The EQ and SQ are self-report questionnaires with a Likert format and contain a list of statements about real-life situations, experiences, and interests where empathizing or systemizing skills are required. Findings from the EQ in adults revealed a significant sex difference, with women scoring significantly higher than men. Results from the SQ indicate that men score significantly higher than women (Carroll and Chiew 2006; Wheelwright et al. 2006). A parent-report version of the EQ and SQ for children between 4 and 11 years of age has also shown similar results with girls scoring significantly higher on the EQ and boys scoring significantly higher on

the SQ (Auyeung et al. 2009c), suggesting that these patterns are present from an early age.

In adults, EQ and SQ scores have also been shown to be better predictors than sex for career choice in science and engineering, or in degree choice (e.g., science vs. humanities), suggesting that typical sex differences in interests or aptitudes may reflect the individual’s cognitive style, independent of their sex.

In order to compare an individual’s empathizing and systemizing, Goldenfeld et al. (2005) examined standardized (normalized) scores on the EQ and SQ. The differences between standardized scores demonstrated strong sex differences and led to the definition of empirical “brain types.” The five “brain types” describe whether an individual is “balanced” (Type B), better at empathizing (Type E), or better at systemizing (Type S). “Extreme” empathizing (Extreme E) or systemizing (Extreme S) types were also assigned where an individual showed a significant discrepancy in different directions (Goldenfeld et al. 2005; Wheelwright et al. 2006). The assignment of “brain types” based on relative EQ and SQ scores in both children and adults appears to be a useful method of describing differences in sex-typical behavior, with the majority of females toward Type E and the majority of males toward Type S (Auyeung et al. 2009c; Goldenfeld et al. 2005; Wheelwright et al. 2006).

Findings using the EQ and SQ questionnaires also provide further evidence for the EMB theory of ASC. When the scores obtained from the EQ and SQ are standardized using the method suggested by Goldenfeld et al. (2005), the vast majority of children and adults with high-functioning autism or Asperger syndrome are found to show the Type S or Extreme S “brain types” (Auyeung et al. 2009c; Goldenfeld et al. 2005; Wheelwright et al. 2006). See Table 1 for a summary of psychological evidence that – irrespective of the direction of sex difference – people with autism show an extreme of the male profile Table 2.

In addition to the evidence at the behavioral level, it has been suggested that characteristics of neurodevelopment in autism such as larger overall brain volumes and greater growth of the amygdala during childhood may also represent an exaggeration of typical sex differences in brain development (Baron-Cohen et al. 2005). Studies using fMRI indicate that typical females show increased activity in the extrastriate cortex during the Embedded Figures Test and increased activity bilaterally in the inferior frontal cortex during the “Reading the Mind in the Eyes” task. Parents of children with ASC also tend to show hyper-masculinization of brain activity, suggesting that hyper-masculinization may be part of the broader autism phenotype.

Extreme Male Brain (EMB) Theory, Table 1 A summary of the psychological evidence for the extreme male brain (EMB) theory

Psychological measure	Autism > male > female	Female > male > autism
Adult systemizing quotient (SQ)	✓	
Child SQ	✓	
Embedded figures test	✓	
Intuitive physics test	✓	
Adult autism spectrum Quotient (AQ)	✓	
Adolescent AQ	✓	
Child AQ	✓	
Childhood autism spectrum Test (CAST)	✓	
Quantitative checklist for autism in toddlers (Q-CHAT)	✓	
Reading the mind in the eyes		✓
adult empathy quotient (EQ)		✓
Child EQ		✓
Faux pas test		✓

Extreme Male Brain (EMB) Theory, Table 2 A summary of the evidence consistent with the EMB theory at the neural level

Brain region	Autism > male > female	Female > male > autism
Total brain volume	✓	
Head circumference	✓	
Grey and white matter	✓	
Amgydala	✓	
Corpus callosum		✓
Perisylvian language areas (Heschl’s gyrus/planum temporale)		✓
L > R asymmetry in planum temporale		✓
Lateral frontoparietal cortex		✓

The Role of Social Factors in the Development of Sex Differences

Social interactions undoubtedly play an important role in the development of gender-typical play and toy choices. Gender-based expectations may cause parents, teachers or caregivers to elicit and reinforce expected behavior from children (Stern and Karraker 1989), thus shaping the child’s behavior. It has been shown that infant gender labeling as male or female often elicits sex-stereotypic responses from adults and children (Stern and Karraker 1989). It has also been suggested that girls are encouraged to be more sensitive and caring toward others than boys (Gilligan 1982). Findings from studies examining play preferences have indicated that boys are encouraged by parents to play with masculine-typical toys and discouraged from playing with feminine-typical toys (Fagot and Hagan 1991). Girls, on the other hand, are also encouraged to play with feminine-typical toys but not necessarily discouraged from playing with masculine-typical toys (Fagot and Hagan 1991). While these factors might influence the behavior exhibited by typically developing children, studies examining eye contact and preference for social stimuli in newborn children provide convincing evidence for a biological basis for some sex differences. It is also not clear how such social factors might apply to the ASC group.

The Role of Prenatal Hormones in the Development of Sex Differences

Though genetic sex is determined at conception, it is the gonadal hormones (i.e., androgens, estrogens, and progestins) that are responsible for differentiation of the male and female phenotypes in the developing human fetus. It is thought that behaviors showing large sex differences are the best candidates for studying effects of hormones on later development (Hines 2004). The direct sampling of fetal serum or manipulation of fetal hormone levels would be highly dangerous. As a result, researchers have employed indirect methods of measuring prenatal hormone exposure to study effects on later development.

One such indirect measure is the ratio between the length of the 2nd and 4th digit (2D, 4D) of the hand. This ratio has been found to be sexually dimorphic, being lower in males than in females. 2D:4D ratio is thought to be fixed by week 14 of fetal life and has been found to reflect fetal exposure to prenatal sex hormones in early gestation (Lutchmaya et al. 2004; Manning 2002). Results from studies of 2D:4D ratios as proxies for fetal testosterone (fT) levels show that children with ASC have more masculinized digit ratios compared to typically developing boys. These patterns have also been observed in the siblings and parents of children with ASC, indicating the possibility of a link between genetically based elevated fT levels and the development of ASC (Manning et al. 2001).

The medical condition of Congenital Adrenal Hyperplasia (CAH) leads to abnormally high prenatal and neonatal androgen levels and has provided researchers with an indirect method of examining the effects of elevated androgen exposure. Girls with CAH have more autistic traits (measured using the adult AQ) compared to their unaffected sisters (Knickmeyer et al. 2006a). Given that this condition is usually treated following birth, this suggests their higher AQ scores reflect elevated prenatal androgen levels. These findings should be interpreted with caution, however, since CAH carries a number of related problems (as well as extensive treatment) which may affect the atypical cognitive profiles found in this population.

Some studies have also compared measurements of testosterone in umbilical cord blood with postnatal development. A recent study using umbilical cord blood testosterone measures examined pragmatic language ability in girls followed-up at 10 years of age. Results showed that the higher a girl’s free testosterone level at birth, the higher the scores on a pragmatic language difficulties questionnaire (Whitehouse et al. 2010). However, levels of fT are typically at very low levels from about week 24 of gestation, umbilical cord samples can contain blood from the mother as well as the fetus, and hormone levels may vary due to labor itself, so umbilical cord blood testosterone does not allow one to test if outcomes reflect fT per se.

Currently the best method to examine the effect of fT is to sample the amniotic fluid surrounding the fetus via amniocentesis. An advantage of amniotic fluid samples is that amniocentesis is often performed for routine clinical purposes within a relatively narrow time period which coincides with the hypothesized critical period for human sexual differentiation between weeks 8 and 24 of gestation (Hines 2004). This is also more direct than the 2D:4D method as the hormones themselves can be assayed, rather than relying on a proxy for these.

A number of studies have linked elevated levels of fT in the amniotic fluid with the masculinization of certain behaviors, beginning shortly after birth. Elevated fT has been linked to reduced

eye contact in infants (Lutchmaya et al. 2002a), smaller vocabulary in toddlers (Lutchmaya et al. 2002b), narrower interests and poorer quality of social relationships at 4 years of age (Knickmeyer et al. 2005), less empathy at 4 and 8 years (Chapman et al. 2006; Knickmeyer et al. 2006b), more male-typical play behavior (Auyeung et al. 2009a), better performance on the Children’s Embedded Figures Test (Auyeung et al., in press), and increased systemizing at 8 years (Auyeung et al. 2006). In addition, fT levels have been found to be positively correlated with number of autistic traits (measured using the Quantitative Checklist for Autism in Toddlers (Allison et al. 2008)) in toddlers between 18 and 24 months of age (Auyeung et al. 2010), as well as in older children (ages 6–10 years old), using two independent dimensional measures of autistic traits (the child version of the AQ and the Childhood Autism Spectrum Test (Auyeung et al. 2009b)). Evidence from typically developing children for effects of fT is summarized in Table 3.

The use of amniotic fluid to measure prenatal hormonal exposure has several limitations. Ideally, it would be best to make direct measurements of testosterone at regular intervals throughout

Extreme Male Brain (EMB) Theory, Table 3 Evidence from typically developing children for effects of fT

Evidence from typical children	Key references
<i>Eye contact</i> is inversely related to fT	Lutchmaya et al. (2002b)
<i>Social skills</i> are inversely related to fT	Knickmeyer et al. (2005)
<i>Vocabulary</i> size is inversely related to fT	Lutchmaya et al. (2002b)
<i>Empathy</i> is inversely related to fT	Chapman et al. (2006), Knickmeyer et al. (2005)
<i>Autistic traits</i> are positively associated with fT	Auyeung et al. (2009b, 2010)
<i>Restricted interests</i> are fT is positively associated	Knickmeyer et al. (2005)
<i>Systemizing</i> is positively associated with fT	Auyeung et al. (2006)
<i>Rightward asymmetry</i> in the isthmus of the corpus callosum is positively associated with fT	Chura et al. (2010)

gestation and into postnatal life. However, it would be extremely hazardous to attempt direct measurements from the fetus itself for purely research purposes. It is not possible to obtain repeated samples of fT because amniocentesis itself carries a risk of causing miscarriage (of about 1%). As a result, obtaining amniotic fT measures are opportunistic, when the procedure is being carried out for clinical reasons, with never more than a single measurement of fT at one time-point although it is known that hormones fluctuate during the day and between days, even in fetuses. The representativeness of a single sample of fT thus remains unclear, but would be difficult to explore in an ethical manner.

In addition, given the reported time course of testosterone secretion, the most promising time to measure fT is probably at prenatal weeks 8–24 (Smail et al. 1981), but this is still a relatively wide range. In addition, research in nonhuman primates has shown that androgens masculinize different behaviors at different times during gestation, suggesting different behaviors may also have different sensitive periods for development. For all these reasons, the inferences that can be drawn about a single measurement of fT are therefore limited. However, where a significant correlation between amniotic fT and a behavior is observed, this should represent a very conservative estimate of the correlation between overall fT levels and that behavior.

Evidence Implicating Testosterone in the Etiology of Autism

Genetic influences are undoubtedly involved with other factors (such as prenatal hormone levels) which lead to the development of ASC. Evidence of a genetic link to ASC is provided by a recent study which shows that genes regulating sex steroids are associated with autistic traits, as measured by scores on the Autism Spectrum Quotient (AQ), in a typical adult sample (Chakrabarti et al. 2009). A parallel study also showed that genes regulating sex steroids are associated with a diagnosis of Asperger syndrome in a case-control sample (Chakrabarti et al. 2009).

Extreme Male Brain (EMB) Theory, Table 4 Evidence for testosterone effects in people with ASC

Evidence from people with ASC	Key references
<i>10 sex steroid genes</i> associated with AS or AQ or empathy HSD11B1, LHCGR, CYP17A1, CYP19A1, SCP2, CYP11B1*, ESR1, ESR2, HSD17B4, HSD17B2*	Chakrabarti et al. (2009)
<i>Timing of puberty</i> Boys with ASC enter puberty earlier, girls with ASC enter puberty later	Ingudomnukul et al. (2007), Knickmeyer et al. (2006c), Tordjman et al. (1997)
<i>Testosterone related medical conditions</i> in women with ASC and their mothers (e.g., PCOS, breast and ovarian cancers, acne)	Ingudomnukul et al. (2007)
<i>Testosterone related characteristics</i> in women with ASC and their mothers	Ingudomnukul et al. (2007), Knickmeyer et al. (2008)
<i>Lower 2D:4D ratio</i> in ASC, and parents	Manning et al. (2001), Milne et al. (2006), Noipayak (2009)
<i>SRD5A1, and AR genes</i> associated with ASC	Henningssson et al. (2009), Hu et al. (2009)

Other lines of evidence implicating testosterone in the etiology of ASC are summarized in Table 4:

Conclusions

There is a significant body of evidence connecting the characteristic behaviors of ASC to extremes of certain male-typical behaviors. Evidence includes superior performance on a range of tasks where males typically outperform females but impairment compared to typical males on tasks showing a female advantage. This observation has led to the development of the “extreme male brain” theory of autism. Support for this theory can also be found very early in life, and also in some primate studies suggesting the development of sex-typical behaviors is at least partly biological.

Research using direct measures of potential biological factors such as prenatal hormones as well as multiple measures of empathizing and systemizing, including both observational and behavioral measures are needed to explore the link between these factors in greater detail. Although the findings presented in this chapter lend support to the “extreme male brain” theory of ASC and its link to fT, a thorough evaluation of this theory will require testing not just for associations between fT and autistic traits, but between fT and clinically diagnosed ASC. This remains an active area of research.

Future Directions

Future studies of empathizing and systemizing in children could examine whether the relationships between fT levels (and other biological factors) remain consistent using multiple measures of empathy and systemizing (e.g., observational, experimental). In addition, the replication of the findings related to fT levels in larger sample sizes would assist in identifying any factors that are linked with levels in the extreme ranges. Future studies could assess whether relationships between fT levels and the development of autistic traits are consistent for individuals with a clinical diagnosis of ASC since the current samples only included typically developing children. Considering the current support for a role for fT in the development of autistic traits, it would also be beneficial for future studies to examine the relationships between fT levels, genetic variation, and the development of autistic traits.

The EMB theory of autism has been developed from studies in typically developing and high-functioning individuals with ASC. It would be interesting to extend the scope of this theory with an examination of individuals with more severe forms of ASC. It is also important to assess the validity of “empathizing” and “systemizing” measures by correlating these with performance and everyday measures of functioning, and future studies could further explore how these domains develop and also how they correlate with neural structure and function.

See Also

- [Broader Autism Phenotype](#)
- [Cognitive Skills](#)
- [Empathy](#)
- [Face Recognition](#)
- [Friendships](#)
- [Gender Differences](#)
- [Social Behaviors and Social Impairment](#)
- [Social Cognition](#)
- [Systemizing](#)
- [Theory of Mind](#)

References and Reading

- Alexander, G. M., & Hines, M. (1994). Gender labels and play styles: Their relative contribution to children's selection of playmates. *Child Development*, 65, 869–879.
- Allison, C., Baron-Cohen, S., Wheelwright, S., Charman, T., Richler, J., Pasco, G., et al. (2008). The Q-CHAT (Quantitative Checklist for Autism in Toddlers): A normally distributed quantitative measure of autistic traits at 18–24 months of age: Preliminary report. *Journal of Autism and Developmental Disorders*, 38, 1414–1425.
- Auyeung, B., Baron-Cohen, S., Chapman, E., Knickmeyer, R., Taylor, K., & Hackett, G. (2006). Foetal testosterone and the child Systemizing Quotient. *European Journal of Endocrinology*, 155.(Suppl. 1, S123–S130.
- Auyeung, B., Baron-Cohen, S., Ashwin, E., Knickmeyer, R., Taylor, K., Hackett, G., et al. (2009a). Fetal testosterone predicts sexually differentiated childhood behavior in girls and in boys. *Psychological Science*, 20, 144–148.
- Auyeung, B., Baron-Cohen, S., Chapman, E., Knickmeyer, R., Taylor, K., & Hackett, G. (2009b). Fetal testosterone and autistic traits. *British Journal of Psychology*, 100, 1–22.
- Auyeung, B., Baron-Cohen, S., Wheelwright, S., Samarawickrema, N., & Atkinson, M. (2009c). The children's Empathy Quotient (EQ-C) and Systemizing Quotient (SQ-C): Sex differences in typical development and of autism spectrum conditions. *Journal of Autism and Developmental Disorders*, 39, 1509–1521.
- Auyeung, B., Taylor, K., Hackett, G., & Baron-Cohen, S. (2010). Foetal testosterone and autistic traits in 18 to 24-month-old children. *Molecular Autism*, 1, 11.
- Auyeung, B., Knickmeyer, R., Ashwin, E., Taylor, K., Hackett, G., & Baron-Cohen, S. (2012). Effects of fetal testosterone on visuospatial ability. *Archives of Sexual Behavior*, 41, 571–581.
- Banerjee, M. (1997). Hidden emotions: Preschoolers' knowledge of appearance-reality and emotion display rules. *Social Cognition*, 15(2), 107–132.

- Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences*, 6(6), 248–254.
- Baron-Cohen, S., & Wheelwright, S. (2003). The Friendship Questionnaire: An investigation of adults with Asperger syndrome or high-functioning autism, and normal sex differences. *Journal of Autism and Developmental Disorders*, 33(5), 509–517.
- Baron-Cohen, S., O'Riordan, M., Stone, V., Jones, R., & Plaisted, K. (1999). Recognition of faux pas by normally developing children and children with Asperger syndrome or high-functioning autism. *Journal of Autism and Developmental Disorders*, 29(5), 407–418.
- Baron-Cohen, S., Wheelwright, S., & Hill, J. (2001). The "Reading the mind in the eyes" test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry*, 42, 241–252.
- Baron-Cohen, S., Knickmeyer, R., & Belmonte, M. K. (2005). Sex differences in the brain: Implications for explaining autism. *Science*, 310, 819–823.
- Benbow, C. P., & Stanley, J. C. (1983). Sex differences in mathematical reasoning ability: More facts. *Science*, 222(4627), 1029–1031.
- Berenbaum, S. A., & Hines, M. (1992). Early androgens are related to childhood sex-typed toy preferences. *Psychological Science*, 3, 203–206.
- Bosacki, S., & Astington, J. W. (1999). Theory of mind in preadolescence: Relations between social understanding and social competence. *Social Development*, 8(2), 237–255.
- Brosnan, M., Daggar, R., & Collomosse, J. (2009). The relationship between systemising and mental rotation and the implications for the extreme male brain theory of autism. *Journal of Autism and Developmental Disorders*, 40, 1–7.
- Caron, M. J., Mottron, L., Rainville, C., & Chouinard, S. (2004). Do high functioning persons with autism present superior spatial abilities? *Neuropsychologia*, 42(4), 467–481.
- Carroll, J. M., & Chiew, K. Y. (2006). Sex and discipline differences in empathising, systemising and autistic symptomatology: Evidence from a student population. *Journal of Autism and Developmental Disorders*, 36(7), 949–957.
- Chakrabarti, B., Dudbridge, F., Kent, L., Wheelwright, S., Hill-Cawthorne, G., Allison, C., et al. (2009). Genes related to sex steroids, neural growth, and social-emotional behavior are associated with autistic traits, empathy, and Asperger syndrome. *Autism Research*, 2(3), 157–177.
- Chapman, E., Baron-Cohen, S., Auyeung, B., Knickmeyer, R., Taylor, K., & Hackett, G. (2006). Fetal testosterone and empathy: Evidence from the empathy quotient (EQ) and the "reading the mind in the eyes" test. *Social Neuroscience*, 1, 135–148.
- Chura, L. R., Lombardo, M. V., Ashwin, E., Auyeung, B., Chakrabarti, B., Bullmore, E. T., et al. (2010). Organizational effects of fetal testosterone on human corpus callosum size and asymmetry. *Psychoneuroendocrinology*, 35, 122–132.
- Connellan, J., Baron-Cohen, S., Wheelwright, S., Batki, A., & Ahluwalia, J. (2000). Sex differences in human neonatal social perception. *Infant Behavior & Development*, 23(1), 113–118.
- Fagot, B. I., & Hagan, R. (1991). Observations of parent reactions to sex-stereotyped behaviors: Age and sex effects. *Child Development*, 62(3), 617–628.
- Falter, C. M., Plaisted, K. C., & Davis, G. (2008). Visuo-spatial processing in autism-testing the predictions of extreme male brain theory. *Journal of Autism and Developmental Disorders*, 38(3), 507–515.
- Gilligan, C. (1982). *In a different voice: Psychological theory and women's development*. Cambridge, MA: Harvard University Press.
- Goldenfeld, N., Baron-Cohen, S., & Wheelwright, S. (2005). Empathizing and systemizing in males, females and autism. *International Journal of Clinical Neuropsychology*, 2, 338–345.
- Happe, F. (1995). The role of age and verbal ability in the theory of mind task performance of subjects with autism. *Child Development*, 66, 843–855.
- Henningsson, S., Jonsson, L., Ljunggren, E., Westberg, L., Gillberg, C., Rastam, M., et al. (2009). Possible association between the androgen receptor gene and autism spectrum disorder. *Psychoneuroendocrinology*, 34(5), 752–761.
- Hines, M. (2004). *Brain gender*. New York: Oxford University Press.
- Hittelman, J. H., & Dicks, R. (1979). Sex differences in neonatal eye contact time. *Merrill-Palmer Quarterly*, 25, 171–184.
- Hoffman, M. L. (1977). Sex differences in empathy and related behaviors. *Psychological Bulletin*, 84(4), 712–722.
- Hu, V. W., Sarachana, T., Kim, K. S., Nguyen, A., Kulkarni, S., Steinberg, M. E., et al. (2009). Gene expression profiling differentiates autism case-controls and phenotypic variants of autism spectrum disorders: Evidence for circadian rhythm dysfunction in severe autism. *Autism Research*, 2(2), 78–97.
- Ingudomnukul, E., Baron-Cohen, S., Wheelwright, S., & Knickmeyer, R. (2007). Elevated rates of testosterone-related disorders in women with autism spectrum conditions. *Hormones and Behavior*, 51(5), 597–604.
- Jolliffe, T., & Baron-Cohen, S. (1997). Are people with autism and Asperger syndrome faster than normal on the embedded figures test? *Journal of Child Psychology and Psychiatry*, 38(5), 527–534.
- Kimura, D. (1999). *Sex and cognition*. Cambridge, MA: The MIT Press.
- Knickmeyer, R., Baron-Cohen, S., Raggatt, P., & Taylor, K. (2005). Foetal testosterone, social relationships, and restricted interests in children. *Journal of Child Psychology and Psychiatry*, 46(2), 198–210.
- Knickmeyer, R., Baron-Cohen, S., Fane, B. A., Wheelwright, S., Mathews, G. A., Conway, G. S., et al.

- (2006a). Androgens and autistic traits: A study of individuals with congenital adrenal hyperplasia. *Hormones and Behavior*, 50(1), 148–153.
- Knickmeyer, R., Baron-Cohen, S., Raggatt, P., Taylor, K., & Hackett, G. (2006b). Fetal testosterone and empathy. *Hormones and Behavior*, 49, 282–292.
- Knickmeyer, R. C., Wheelwright, S., Hoekstra, R., & Baron-Cohen, S. (2006c). Age of menarche in females with autism spectrum conditions. *Developmental Medicine and Child Neurology*, 48(12), 1007–1008.
- Knickmeyer, R. C., Wheelwright, S., & Baron-Cohen, S. B. (2008). Sex-typical play: Masculinization/defeminization in girls with an autism spectrum condition. *Journal of Autism and Developmental Disorders*, 38(6), 1028–1035.
- Lawson, J., Baron-Cohen, S., & Wheelwright, S. (2004). Empathising and systemising in adults with and without Asperger syndrome. *Journal of Autism and Developmental Disorders*, 34(3), 301–310.
- Lutchmaya, S., Baron-Cohen, S., & Raggatt, P. (2002a). Foetal testosterone and eye contact in 12 month old infants. *Infant Behavior & Development*, 25, 327–335.
- Lutchmaya, S., Baron-Cohen, S., & Raggatt, P. (2002b). Foetal testosterone and vocabulary size in 18- and 24-month-old infants. *Infant Behavior & Development*, 24(4), 418–424.
- Lutchmaya, S., Baron-Cohen, S., Raggatt, P., Knickmeyer, R., & Manning, J. T. (2004). 2nd to 4th digit ratios, fetal testosterone and estradiol. *Early Human Development*, 77, 23–28.
- Manning, J. T. (2002). *Digit ratio: A pointer to fertility, behavior and health*. New Brunswick: Rutgers University Press.
- Manning, J. T., Baron-Cohen, S., Wheelwright, S., & Sanders, G. (2001). The 2nd to 4th digit ratio and autism. *Developmental Medicine and Child Neurology*, 43(3), 160–164.
- Milne, E., White, S., Campbell, R., Swettenham, J., Hansen, P., & Ramus, F. (2006). Motion and form coherence detection in autistic spectrum disorder: Relationship to motor control and 2:4 digit ratio. *Journal of Autism and Developmental Disorders*, 36(2), 225–237.
- Noipayak, P. (2009). The ratio of 2nd and 4th digit length in autistic children. *Journal of the Medical Association of Thailand*, 92(8), 1040–1045.
- Pierce, K., Conant, D., Hazin, R., Stoner, R., & Desmond, J. (2010). Preference for geometric patterns early in life as a risk factor for autism. *Archives of General Psychiatry*.
- Podrouzek, W., & Furrow, D. (1988). Preschoolers' use of eye contact while speaking: The influence of sex, age, and conversational partner. *Journal of Psycholinguistic Research*, 17(2), 89–98.
- Ropar, D., & Mitchell, P. (2001). Susceptibility to illusions and performance on visuospatial tasks in individuals with autism. *Journal of Child Psychology and Psychiatry*, 42(4), 539–549.
- Servin, A., Bohlin, G., & Berlin, D. (1999). Sex differences in 1-, 3-, and 5-year-olds' toy-choice in a structured play session. *Scandinavian Journal of Psychology*, 40, 43–48.
- Shah, A., & Frith, U. (1983). An islet of ability in autistic children: A research note. *Journal of Child Psychology and Psychiatry*, 24(4), 613–620.
- Shah, A., & Frith, C. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry*, 34, 1351–1364.
- Smail, P. J., Reyes, F. I., Winter, J. S. D., & Faïman, C. (1981). The fetal hormonal environment and its effect on the morphogenesis of the genital system. In S. J. Kogan & E. S. E. Hafez (Eds.), *Pediatric andrology* (pp. 9–19). Boston: Martinus Nijhoff.
- Stern, M., & Karraker, K. H. (1989). Sex stereotyping of infants: A review of gender labeling studies. *Sex Roles*, 20, 501–522.
- Tordjman, S., Ferrari, P., Sulmont, V., Duyme, M., & Roubertoux, P. (1997). Androgenic activity in autism. *The American Journal of Psychiatry*, 154(11), 1626–1627.
- Voyer, D., Voyer, S., & Bryden, M. P. (1995). Magnitude of sex differences in spatial abilities: A meta-analysis and consideration of critical variables. *Psychological Bulletin*, 117(2), 250–270.
- Wheelwright, S., Baron-Cohen, S., Goldenfeld, N., Delaney, J., Fine, D., Smith, R., et al. (2006). Predicting autism spectrum quotient (AQ) from the systemizing quotient-revised (SQ-R) and empathy quotient (EQ). *Brain Research*, 1079(1), 47–56.
- Whitehouse, A. J., Maybery, M. T., Hart, R., Mattes, E., Newnham, J. P., Sloboda, D. M., et al. (2010). Fetal androgen exposure and pragmatic language ability of girls in middle adulthood: Implications for the extreme male-brain theory of autism. *Psychoneuroendocrinology*, 35(8), 1259–1264.
- Witkin, H. A., Dyk, R. B., Fattuson, H. F., Goodenough, D. R., & Karp, S. A. (1962). *Psychological differentiation: Studies of development* (p. 418). Oxford, UK: Wiley.
- Yirmiya, N., Sigman, M. D., Kasari, C., & Mundy, P. (1992). Empathy and cognition in high-functioning children with autism. *Child Development*, 63(1), 150–160.

Eye Contact

► Mutual Gaze

Eye Gaze

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Definition

The term “eye gaze” may refer to either of the two distinctive, but related, topics. Firstly, it refers to the perception of other persons’ eye gaze, which can be inferred from the relative position of the iris within the eyelid, in conjunction with the orientation of the head and the body. Secondly, it also refers to the production of eye gaze by a patient or a participant, such as the control of saccades and fixations. This entry mainly covers the former topic. Perception of others’ eye gaze is crucial for social interaction and communication and its impairment in ASD because eye gaze signals the direction of others’ attention and intention.

Current Knowledge

Fixations on Another Person’s Eyes

Atypical pattern of mutual gaze behavior, or eye contact, is among the most distinguishable manifestation of the qualitative impairment in social interaction in ASD. Since Kanner’s first report (Kanner 1943), such atypical pattern of eye contact has been reported and discussed in many clinical and experimental settings, including recent studies using eye-tracking methods (Senju and Johnson 2009a). Based on this clinical significance, eye contact is currently included in standardized diagnostic criteria such as DSM and ICD. In DSM-IV-TR, it is defined as a “marked impairment in the use of multiple nonverbal behaviors (e.g., eye-to-eye gaze) to regulate social interaction and communication” (American Psychiatric Association [APA] 2000, p. 70). Retrospective home video analyses of infants who were

later diagnosed with ASD have revealed that atypical patterns of eye contact can be observed within the first year of life, well before the age of diagnosis (Maestro et al. 2005). Some researchers and clinicians once proposed that such atypical eye contact behavior results from “gaze avoidance,” active avoidance of others’ eye gaze due to negatively valenced overarousal (e.g., Hutt and Ounsted 1966). However, follow-up studies reported mixed results, suggesting that the gaze avoidance may be present in some individuals with ASD, but it may not be universal or predominant in this population (Buitelaar 1995).

Some of eye-tracking studies have revealed that individuals with ASD fixate others’ eyes less than typically developing individuals do, but other studies failed to replicate or reported mixed results (Senju and Johnson 2009a). Such inconsistencies may result from the differences in task demands and/or the characteristics of stimuli used. In general, reduced fixations on the eyes are most prominent with complex and cognitively demanding face stimuli and/or by using dynamic videotape stimuli, including conversations. Moreover, individual differences in the fixations on the eyes within ASD correlate with the volume and the face sensitivity of amygdala (e.g., Dalton et al. 2005), as well as the degree of self-reported social anxiety (Corden et al. 2008). However, it does not correlate with autistic symptoms measured with ADOS or AQ (Corden et al. 2008).

Perception of Direct Gaze

Direct gaze signals that the person is looking at the perceiver. It also leads to the establishment of mutual gaze or eye contact, which signals the initiation of communication. In typically developed population, perceived direct gaze, or eye contact, modulates concurrent and/or immediately following cognitive processing and/or behavioral responses, a phenomenon called the eye contact effect (Senju and Johnson 2009b). For example, perceived eye contact facilitates the performance of face-related tasks such as gender discrimination, recognition of face identity, and detection of gaze direction. Results from neuroimaging studies

also indicate that perceived eye contact modulates the activation of social brain network (defined as the cortical and subcortical structures specialized for the processing of social information, such as fusiform gyrus, superior temporal sulcus, medial prefrontal and orbitofrontal cortex, and amygdala). Sensitivity to the direction of another person's eye gaze appears very early in typical development (Johnson et al. 2009). For example, newborns preferentially look longer at the faces with direct gaze than those with averted gaze. Perceived eye contact also facilitates the processing of face identity and communicative facial expression during the first half year of life.

A series of studies have demonstrated atypical processing of direct gaze in ASD (Senju and Johnson 2009a). For example, Senju, Yaguchi, Tojo, and Hasegawa (2003) used an oddball procedure in which children were asked to detect a rare stimulus presented occasionally within the context of a frequently reoccurring stimulus. The rare stimuli were either faces with direct gaze or those with laterally averted eye gaze (i.e., faces with eyes looking leftward or rightward). The frequent stimuli were faces with eyes looking downward. Thirteen children with autism (mean age: 12 years) were compared with typically developing children of the same range of the age and nonverbal intelligence. Typically developing children showed the eye contact effect, that they were better at detecting direct gaze than averted gaze. By contrast, although children with autism are equally good at detecting averted gaze as typically developing children, they were less skilled at detecting faces with direct gaze, and they failed to show the eye contact effect or the facilitation of performance caused by the perceived direct gaze. Similarly, Pellicano and Macrae (2009) assessed whether direct gaze facilitates the person categorization in children with autism (mean age: 10 years) and age- and IQ-matched typically developing children. Results revealed that typically developing children were faster to categorize faces by sex when they were with direct gaze than with averted gaze. By contrast, although children with autism were as fast as typically developing children to categorize faces, their performance were unaffected by the gaze direction of the faces. These studies

demonstrate that unlike typically developing individuals, individuals with ASD do not show the eye contact effect or the facilitation of face and gaze processing caused by the perceived direct gaze.

Several studies have recorded field potentials on the scalp with either EEG or MEG to assess the cortical response to direct and averted gazes in individuals with ASD, but results are mixed. Two studies reported that 3- to 7-year-old children (mean age: 5 years, Grice et al. 2005) as well as 7- to 12-year-old children (mean age: 10 years old, Kylliäinen et al. 2006) with ASD showed larger event-related potential (ERP) or event-related field (ERF) response to direct gaze than averted gaze, but control children did not. By contrast, Senju, Tojo, Yaguchi, and Hasegawa (2005) reported that typically developing children (9- to 14-year-olds, mean age: 12 years) showed a larger ERP amplitude for direct than for averted gaze, but ERPs of children with ASD were not modulated by the presence of eye contact. Moreover, Elsabbagh et al. (2009) recorded EEG from high-risk infants (see also ► [“Early Diagnosis”](#)) as well as from low-risk control infants while they watched faces with either direct or averted eye gaze and conducted ERP and time-frequency analysis (TFA). Results showed that a late ERP component (P400), which is known to relate to face processing, has a longer latency in response to direct gaze in high-risk infants. Secondly, TFA analysis revealed clearly distinguished and temporally sustained high-frequency oscillatory activity in the gamma-band frequency for direct gaze compared to averted gaze in control infants. In contrast, high-frequency oscillatory activity in gamma band for direct gaze compared to averted gaze in high-risk infants was delayed and less persistent. These results suggest that atypical eye contact processing in high-risk infants relates to the top-down modulation (as indicated by the slower P400 latency) and task-relevant synchronization of brain activations (as indicated by the lack of differential gamma-band activation in response to eye contact).

Two studies have assessed skin conductance response (SCR), an index of physiological arousal, while individuals with ASD observe faces with either direct or averted eye gaze.

However, the results are again mixed – one study found larger SCR in response to direct gaze in 10-year-old children with ASD (Kylliäinen and Hietanen 2006), but the other study did not replicate this findings in 12-year-old children (Joseph et al. 2008). Such inconsistency between physiological studies may result from the different age ranges of the participants and/or the different experimental tasks used for EEG/MEG/SCR recordings.

To summarize, behavioral studies have consistently found that direct gaze facilitates cognitive processing of faces in typically developing children, but the face and gaze processing of individuals with ASD are unaffected by the presence of direct gaze (Senju and Johnson 2009a). Inconsistencies in the physiological studies make it difficult to understand the neural basis of such atypical processing of direct gaze in individuals with ASD.

Perception of Averted Gaze

Averted gaze, by contrast, signals that the person is looking at something in the environment. Thus, following another person's gaze helps enable the perceiver to infer his or her attention, goal, and/or intention (see also ► “Gaze”). In typical development, perceived averted gaze reflexively shifts the attention of the perceiver to the corresponding direction, which is often called gaze cueing effect (Frischen et al. 2007). The gaze cueing effect can be observed from 3 months of age, which corresponds to the age range when a rudimentary form of gaze following starts to appear.

Individuals with ASD do not have an overall impairment in detecting the direction of others' eye gaze. For example, Leekam, Baron-Cohen, Perrett, Milders, and Brown (1997) demonstrated that children with autism are as good as typically developing children at discerning the subtle differences in gaze direction. Moreover, majority of studies demonstrated that individuals with autism across the wide age range, from 2-year-old toddlers to adults, show apparently typical gaze cueing effect (Nation and Penny 2008). In these studies, participants are presented with a picture of a face with gaze averted to the left or right followed by a target stimuli presented either to the left or right of the face. Individuals with ASD, as well as typically developing individuals,

are faster to detect the target when the face was looking at the location than when the face was looking at the opposite direction. These studies contrast with the impairment in gaze following behavior in individuals with ASD (see also ► “Gaze”).

However, several studies also suggest the qualitative differences in the gaze cueing effect between individuals with ASD and typically developing individuals. For example, Senju, Tojo, Dairoku, and Hasegawa (2004) compared the cueing effect of eye gaze and an arrowhead and found that children with autism do not show preferential sensitivity to eye gaze unlike typically developing children. Other studies (e.g., Ristic et al. 2005) used a schematic face, instead of a photographic image of a face, and found that only typically developing individuals show the gaze cueing effect in response to the schematic eyes. By contrast, individuals with ASD do not show gaze cueing effect in response to these stimuli. These studies suggest that the gaze cueing effect in individuals with ASD is not as robust as in typically developing individuals and perhaps is not processed in a functionally specialized cognitive mechanism.

Individuals with ASD also show difficulties in inferring mentalistic and communicative significance of the eyes. For example, Baron-Cohen, Baldwin, and Crowson (1997) presented two novel objects to 7- to 12-year-old children with ASD as well as typically developing children matched by the mental age and verbally labelled one of these objects. Typically developing children correctly associated the label with the object being looked at by the experimenter, even when the child was looking at the other object when the experimenter uttered the label. By contrast, children with autism tended to apply the label to objects in their own focus rather than the one the experimenter was looking at. An adult neuroimaging study also demonstrated that individuals with ASD, unlike typically developing individuals, do not show differential cortical activation in response to the referential gaze (Pelphrey et al. 2005).

To summarize, individuals with ASD have relatively good capacity to encode the direction of others' eye gaze and reflexively shift their

attention to the corresponding direction. However, the gaze processing in individuals with ASD may not be as robust or specialized as those of typically developing individuals, which may be related to the difficulty in using others' eye gaze in the context of social learning.

Future Directions

Further studies will be required to clarify the neurodevelopmental basis of eye gaze processing in ASD and its relationship with other clinical symptoms. Firstly, it is not clear why there are such inconsistencies between different neurophysiological studies contrasting direct and averted gaze processing in ASD, as well as the different eye-tracking studies investigating the pattern of face scanning in ASD (Senju and Johnson 2009a). Thus, further studies will be required to systematically control the background of participants (e.g., age, gender, and/or comorbidity) as well as the properties of the stimuli and the tasks used (e.g., dynamic vs. static, active task vs. passive viewing, size and/or facial expression) to reveal the neurophysiological basis of direct gaze processing in ASD. Secondly, it will be important to investigate the relationship between atypical cortical response to eye gaze in early infancy (e.g., Elsabbagh et al. 2009) and the later manifestation of clinical symptoms.

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Rev.), DSM-IV-TR. Washington, DC: Author.
- Baron-Cohen, S., Baldwin, D. A., & Crowson, M. (1997). Do children with autism use the speaker's direction of gaze strategy to crack the code of language? *Child Development*, 68(1), 48–57.
- Buitelaar, J. K. (1995). Attachment and social withdrawal in autism – Hypotheses and findings. *Behaviour*, 132, 319–350.
- Corden, B., Chilvers, R., & Skuse, D. (2008). Avoidance of emotionally arousing stimuli predicts social-perceptual impairment in Asperger's syndrome. *Neuropsychologia*, 46(1), 137–147.
- Dalton, K. M., Nacewicz, B. M., Johnstone, T., Schaefer, H. S., Gernsbacher, M. A., Goldsmith, H. H., et al. (2005). Gaze fixation and the neural circuitry of face processing in autism. *Nature Neuroscience*, 8(4), 519–526.
- Elsabbagh, M., Volein, A., Csibra, G., Holmboe, K., Garwood, H., Tucker, L., et al. (2009). Neural correlates of eye gaze processing in the infant broader autism phenotype. *Biological Psychiatry*, 65(1), 31–38.
- Frischen, A., Bayliss, A. P., & Tipper, S. P. (2007). Gaze cueing of attention: Visual attention, social cognition, and individual differences. *Psychological Bulletin*, 133(4), 694–724.
- Grice, S. J., Halit, H., Farroni, T., Baron-Cohen, S., Bolton, P., & Johnson, M. H. (2005). Neural correlates of eye-gaze detection in young children with autism. *Cortex*, 41(3), 342–353.
- Hutt, C., & Ounsted, C. (1966). The biological significance of gaze aversion with particular reference to the syndrome of infantile autism. *Behavioral Science*, 11(5), 346–356.
- Johnson, M. H., Grossmann, T., & Kadosh, K. C. (2009). Mapping functional brain development: Building a social brain through interactive specialization. *Developmental Psychology*, 45(1), 151–159.
- Joseph, R. M., Ehrman, K., McNally, R., & Keehn, B. (2008). Affective response to eye contact and face recognition ability in children with ASD. *Journal of International Neuropsychological Society*, 14(6), 947–955.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kylliäinen, A., & Hietanen, J. K. (2006). Skin conductance responses to another person's gaze in children with autism. *Journal of Autism and Developmental Disorders*, 36(4), 517–525.
- Kylliäinen, A., Braeutigam, S., Hietanen, J. K., Swithenby, S. J., & Bailey, A. J. (2006). Face and gaze processing in normally developing children: A magnetoencephalographic study. *European Journal of Neuroscience*, 23(3), 801–810.
- Leekam, S., Baron-Cohen, S., Perrett, D., Milders, M., & Brown, S. D. (1997). Eye-direction detection: A dissociation between geometric and joint attention skills in autism. *British Journal of Developmental Psychology*, 15(1), 77–95.
- Maestro, S., Muratori, F., Cavallaro, M. C., Pecini, C., Cesari, A., Paziente, A., et al. (2005). How young children treat objects and people: An empirical study of the first year of life in autism. *Child Psychiatry and Human Development*, 35, 383–396.
- Nation, K., & Penny, S. (2008). Sensitivity to eye gaze in autism: Is it normal? Is it automatic? Is it social? *Development and Psychopathology*, 20(01), 79–97.
- Pellicano, E., & Macrae, C. N. (2009). Mutual eye gaze facilitates person categorization for typically developing children, but not for children with autism. *Psychonomic Bulletin and Review*, 16(6), 1094–1099.
- Pelphrey, K. A., Morris, J. P., & McCarthy, G. (2005). Neural basis of eye gaze processing deficits in autism. *Brain*, 128(Pt 5), 1038–1048.
- Ristic, J., Mottron, L., Friesen, C. K., Iarocci, G., Burack, J. A., & Kingstone, A. (2005). Eyes are special but not

- for everyone: The case of autism. *Cognitive Brain Research*, 24(3), 715–718.
- Senju, A., & Johnson, M. H. (2009a). Atypical eye contact in autism: Models, mechanisms and development. *Neuroscience and Biobehavioral Reviews*, 33(8), 1204–1214.
- Senju, A., & Johnson, M. H. (2009b). The eye contact effect: Mechanisms and development. *Trends in Cognitive Sciences*, 13(3), 127–134.
- Senju, A., Yaguchi, K., Tojo, Y., & Hasegawa, T. (2003). Eye contact does not facilitate detection in children with autism. *Cognition*, 89(1), B43–B51.
- Senju, A., Tojo, Y., Dairoku, H., & Hasegawa, T. (2004). Reflexive orienting in response to eye gaze and an arrow in children with and without autism. *Journal of Child Psychology and Psychiatry*, 45(3), 445–458.
- Senju, A., Tojo, Y., Yaguchi, K., & Hasegawa, T. (2005). Deviant gaze processing in children with autism: An ERP study. *Neuropsychologia*, 43(9), 1297–1306.

Eye Movement Desensitization and Reprocessing (EMDR) Therapy in Children and Adults with Autism

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Definition

Eye movement desensitization and reprocessing (EMDR) therapy is a first-choice psychological treatment for posttraumatic stress disorder

(PTSD) in the general population (World Health Organization 2013; ISTSS 2019). Although there is growing evidence that the scope of EMDR therapy is broader than treatment of full blown PTSD, little is known about the feasibility and potential effectiveness of EMDR in children and adults with autism spectrum disorder (ASD) who suffer from the consequences of exposure to adverse events and trauma. There are indications that these consequences often are overlooked and misinterpreted as autistic features and therefore remain untreated. This entry provides an overview of the current knowledge about the feasibility and effectiveness of EMDR therapy in individuals with ASD and trauma-related symptoms and its clinical and scientific implications.

EMDR therapy is a protocolized treatment, aimed to resolve symptoms resulting from disturbing and unprocessed life experiences. EMDR therapy is an evidence-based treatment for PTSD and may be supportive in the treatment of a wide variety of other mental health problems like anxiety disorders, depressive disorders, and obsessive-compulsive disorders (Shapiro 2018; Valiente-Gómez et al. 2017). EMDR therapy starts with history taking and a case conceptualization. Therapy continues with a short introduction about how EMDR therapy works, shortly after which the client focusses on the traumatic memory. The therapist then asks the client to bring up the memory and to focus on the most distressing image, eliciting the dysfunctional negative cognition (NC) of oneself in relation to the image, as well as the accompanying emotions and the body disturbance that go along with it. Next, the therapist moves his or her fingers back and forth in front of the client's eyes as fast as the client can follow. Repeatedly, the client is asked to report about emotional, cognitive, somatic, and/or imagistic experiences that arise. A new set of eye movements follows, and this procedure is repeated until the disturbance related to the memory reaches a SUD (Subjective Unit of Disturbances scale) of zero out of ten and an adaptive and positive statement about oneself (PC, Positive Cognition) is rated as fully believable on a VoC (Validity of Cognition) scale. The end of the session is dedicated to closing down the session positively and preparing the

client for the interim in between sessions. A core feature of the procedure is carrying out a sufficient demanding bilateral working-memory-task which is accomplished by the rapid eye movements (De Jongh et al. 2013).

Historical Background

Children and adults with ASD are at *elevated risk* of experiencing a history of adverse events and revictimization (Hoover 2015; Kildahl et al. 2019). It has been argued that exposure to adverse events inhibits the ability to detect violations and exacerbates already impaired emotion regulation problems in youth with ASD (Kerns et al. 2015). These factors may negatively influence the ability to cope with future stressors and elevate the risk of revictimization. PTSD in children and adolescents with ASD co-occurs at a similar or greater rate compared to general population estimates (Rumball 2019).

Individuals with ASD are known to have specific difficulties in resolving unprocessed life experiences, making them *susceptible to psycho-social consequences of exposure* to adverse events (e.g., Roberts et al. 2015). ASD can be characterized as a different way of sense-making and as a problem with self-regulation, which is reflected in problems in social communication and interaction and restricted and repetitive patterns of behavior or interests. Adverse events that are considered to be mildly annoying for individuals without ASD may be perceived as distressing or even traumatic by individuals with ASD and vice versa (Taylor and Gotham 2016). Wood and Gadow (2010) hypothesized that ASD-related sensory hyper-reactivity to daily stimuli, social confusion, incomprehension, and rejection by others may lead to clinically significant anxiety, which affects resilience to cope with stressors.

Several authors reported a risk of *overlooking* a history of exposure to adverse events and symptoms of PTSD in persons with ASD. There may be several reasons for this phenomenon. First, the different sense-making may prevent them from recognizing risky circumstances and

communicating about their experiences (Kerns et al. 2015). The risk of overlooking is even stronger in case of persons with ASD and an intellectual disability (Kildahl et al. 2019). Second, symptoms attributed to ASD might in fact be stress reactions to adverse events or trauma, a phenomenon termed *diagnostic overshadowing*. For example, hyper-arousal and numbing (symptoms of PTSD) overlap with ASD related hyper- and hypo-reactivity to sensory stimuli. Feelings of detachment of others – a symptom of PTSD – partly overlap with deficits in social-emotional reciprocity. A reduced ability to mentalize and recognize emotions is seen in both people with PTSD and in people with ASD. Also, there is an overlap between the ASD-related symptoms of perseveration and rumination and the criterion *negative cognitions and mood* due to PTSD (DSM-5 2013). Especially people with severe PTSD symptoms not fulfilling PTSD DSM-5 criteria (e.g., because they do not meet DSM-5 criterion A for PTSD) are at risk to be excluded for trauma treatment. Hence, both trauma and trauma-related symptoms can be overlooked or overshadowed by autistic features and therefore remain untreated.

Current Knowledge

Little is known about the feasibility and potential effectiveness of EMDR therapy in individuals with ASD and trauma-related symptoms. This may partly be caused by the problem of overlooking and overshadowing as mentioned above. Another impeding factor may be that individuals with ASD are often excluded from participating in research (Spinazzola et al. 2005). This entry gives a short overview of what is known about EMDR in autistic children and adults with and without intellectual disabilities.

EMDR Therapy in Children with ASD

Preliminary findings from a few case reports suggest EMDR therapy for PTSD to be feasible and potentially effective for children with ASD (e.g., Ipci et al. 2017) also in case of comorbid intellectual disabilities (Mevisen et al. 2011). Controlled studies are lacking.

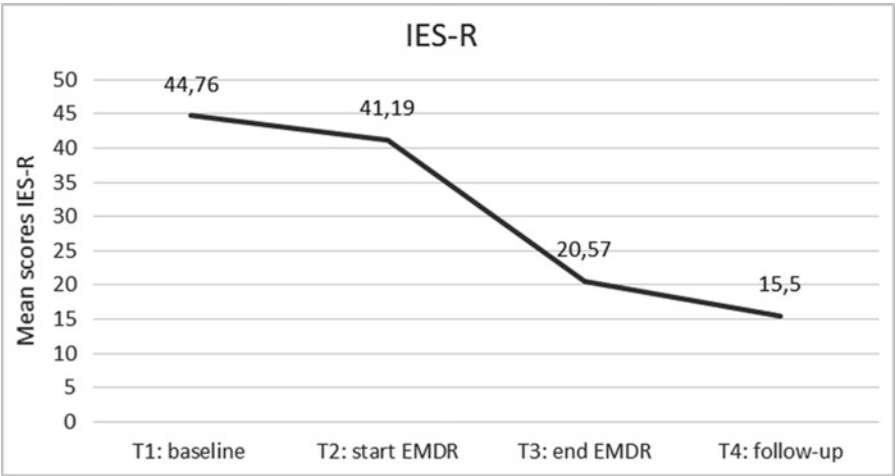
EMDR Therapy in Adults with ASD

Several case reports have been published describing the treatment of trauma with EMDR therapy in adults with ASD (Kosatka and Ona 2014) and in adults with ASD and intellectual disabilities (Barol and Seubert 2010; Mevissen et al. 2011), with promising results.

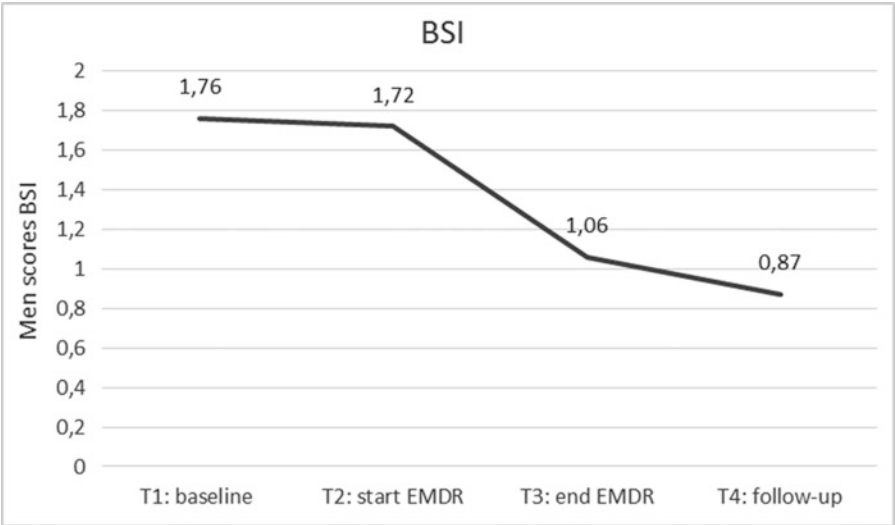
Until now, one controlled study has been published describing the feasibility and effectiveness of EMDR therapy for PTSD symptoms in adults with ASD (Lobregt-van Buuren et al. 2019). The study investigated whether EMDR is a feasible therapy for adults with ASD and a history of adverse events and whether it is associated with reductions in symptoms of PTSD, psychological distress, and autism. The study had a non-randomized add-on design consisting of three phases, in which participants were their own controls. In the first phase participants received treatment as usual (TAU) during 6–8 weeks while on the waiting list for EMDR therapy. The second phase consisted of up to 8 sessions EMDR in addition to TAU. The third phase comprised of a follow-up period with the TAU only condition. TAU consisted of the most common treatments for adults with ASD aimed at coping with ASD, psychological distress, and comorbid problems. TAU included pharmacotherapy, psychoeducation, supportive counseling, job

coaching, support with housekeeping, and so-called case management. Results showed a significant reduction of symptoms of post-traumatic stress (measured with the Impact of Event Scale-Revised: $d=1.16$; see Fig. 1), psychological distress (measured with the Brief Symptom Inventory: $d=0.93$; see Fig. 2), and autistic features (Social Responsiveness Scale-Adult version: $d=0.39$; see Fig. 3). Moreover, the participants experienced a significantly lower level of daily life impairment related to the traumatic events following EMDR therapy (thermometer card of the Adapted Anxiety Disorders Inventory Scale for children (ADIS-C) section PTSD: $d=1.81$; see Fig. 4). The positive results were maintained at follow-up. The results suggest EMDR therapy added to TAU to be a feasible and potentially effective treatment for individuals with ASD who suffer from the consequences of exposure to distressing events. It should be noted that the study was based upon a small sample of participants ($n=21$).

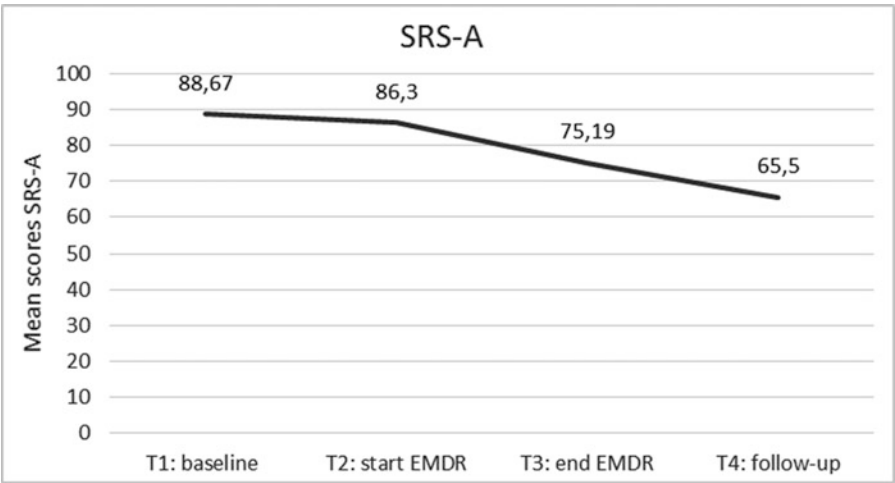
The significant reduction of autistic features concerning social motivation and communication following EMDR therapy, and at follow-up, albeit with a small effect size, is remarkable. A possible explanation for this finding might be that the clinical manifestation of autistic symptoms decreases when individuals with ASD experience



Eye Movement Desensitization and Reprocessing (EMDR) Therapy in Children and Adults with Autism, Fig. 1 Mean scores IES-R at four moments in time



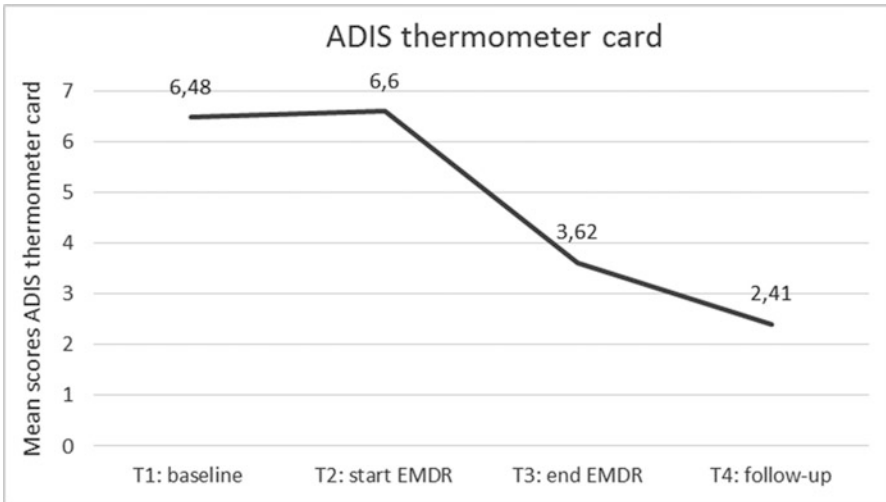
Eye Movement Desensitization and Reprocessing (EMDR) Therapy in Children and Adults with Autism, Fig. 2 Mean scores BSI at four moments in time



Eye Movement Desensitization and Reprocessing (EMDR) Therapy in Children and Adults with Autism, Fig. 3 Mean scores SRS-A at four moments in time

less trauma-related stress and psychological distress, such as somatization, depression, and obsessive-compulsive symptoms. Hence, it is conceivable that trauma-related symptoms are a moderator for the severity of ASD symptoms, such that exposure to trauma and other adverse events exacerbate autistic core features like deficits in social-emotional reciprocity (e.g., reduced

sharing of interests, emotions, or affect; Wood and Gadow 2010). Another possible explanation for the observed reduction of autistic features in the study is that symptoms ascribed to phenotypic features of ASD may in fact be symptoms of PTSD, the phenomenon termed *diagnostic overshadowing*. For example, hyperarousal as a consequence of exposure to trauma may be



Eye Movement Desensitization and Reprocessing (EMDR) Therapy in Children and Adults with Autism, Fig. 4 Mean scores ADIS thermometer card at four moments in time

interpreted as an autistic feature similar to hyper-reactivity to sensory stimuli. Also, trauma-related avoidance of social situations can be confused with autistic features similar to problems in social communication. In other words, symptoms of PTSD can be masked by symptoms of autism, therefore symptoms of PTSD, previously seen as features of ASD, might have declined as a result of EMDR therapy.

Future Directions

Clinical Implications

The following practical recommendations based on current knowledge can be made for future clinical practice.

- Awareness of the impact of adverse events and trauma in individuals with ASD is important to prevent undertreatment.
- There is a high prevalence of being bullied in individuals with ASD (Hoover 2015; Maïano et al. 2016). Exposure to bullying is associated with severe psychiatric outcomes in adulthood. Bullying does not comply with the (i.e., A-criterion) definition of trauma in the PTSD section of *DSM-5*. When there is no formal

PTSD-classification, there is an extra risk of undertreatment of the trauma-related symptoms. Disturbing memories of being bullied (e.g., social situations where participants felt excluded and intimidated) can be reprocessed with EMDR therapy.

- Trauma history and associated trauma-related symptoms should be routinely assessed in individuals with ASD who present to clinical services, taking into account the social communication impairments. What is not explicitly asked for often remains undisclosed, because of inability to spontaneously share relevant information. This issue can be addressed by making use of the concrete, visualized, and structured way in which potentially traumatic events and trauma-related symptoms are probed by the Diagnostic Interview Trauma and Stressors-Intellectual Disability (DITS-ID; Mevissen et al. 2016, 2017, 2018, in press), such that also in children and adults with ASD unprocessed memories can be identified. The DITS-ID is the latest DSM-5 based version of the Adapted Anxiety Disorders Inventory Scale for children (ADIS-C) section PTSD. All documents (only in Dutch language) belonging to the DITS are available and can be downloaded for free. See <https://www.accare.nl/childstuddycenter/opleidingen/bijbscholing/dits-lvb/aanvragen-documenten-dits-lvb>.

- Trauma-related symptoms and PTSD in individuals with ASD can be treated successfully with EMDR therapy, taking into account aspects of their specific information processing. The EMDR procedure for children can be used, when the language of the EMDR protocol for adults is too abstract for people with ASD (Dutch version, De Roos et al. 2014). Individuals with ASD seem to need more time to become familiar with the therapist and the procedures and to fill out questionnaires. In case of obstacles when administering EMDR therapy to individuals with ASD, see the link to the following document: *Guidelines and tips for EMDR treatment for Autism Spectrum Disorders* (Special Interest Group EMDR – ASD November 2013, <https://psycho-trauma.nl/wp-content/uploads/2014/01/GuidelinesEMDRASD-1.pdf>). These preliminary recommendations are based on well-established and evidence-based practices for working with this population.
- Based on clinical observations, EMDR therapy seems difficult to perform in individuals with ASD who have problems with activating the traumatic memories because of fear of affects, in combination with severe problems in self- and emotional regulation, a cognitive, rigid, and extremely negative mindset. These individuals are often exposed to prolonged and repeated trauma such as childhood sexual abuse and domestic violence (complex PTSD) and/or are exposed to prolonged and repeated overload and relational mismatches between the needs of the individual and the possibilities of the environment. The iatrogenic effects of inadequate treatments due to delayed diagnostics or inadequate facilities should not be underestimated in these subgroups.
- EMDR embedded in treatment as usual seems appropriate and necessary given the comorbid psychiatric conditions and/or structural problems at home, school, or work in individuals with ASD.

Scientific Implications

Controlled trials with sufficient power to detect differences are greatly needed to confirm the

present findings and to test the hypothesis that EMDR therapy is more effective than treatment as usual in reducing trauma- and stressor-related symptoms. Investigation and experimentation into other evidence-based trauma treatments like cognitive behavioral therapy with a trauma focus (CBT-T), narrative exposure therapy for individuals with ASD, and trauma-related symptoms are strongly recommended. More generally, the issue as to how PTSD and other trauma-related disorders manifest or may be masked by symptoms of ASD is an intriguing one which should be explored in further research.

See Also

- Diagnostic Overshadowing
- Emotional Regulation
- Posttraumatic Stress Disorder

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders, DSM-5* (5th ed.). Washington, DC: American Psychiatric Association.
- Barol, B. I., & Seubert, A. (2010). *Journal of EMDR Practice and Research*, 4(4), 156–169. <https://doi.org/10.1891/1933-3196.4.4.156>.
- De Jongh, A., Ernst, R., Marques, L., & Hornsveld, H. (2013). The impact of eye movements and tones on disturbing memories involving PTSD and other mental disorders. *Journal of Behavior Therapy and Experimental Psychiatry*, 44, 477–483.
- De Roos, C., Beer, R., De Jongh, A., & Ten Broeke, E. (2014). EMDR-protocol voor kinderen tot 18 jaar. In E. Ten Broeke, A. De Jongh, & H. J. Oppenheim (Eds.), *Praktijkboek EMDR: Casusconceptualisatie en specifieke patiëntengroepen [bijlage V]*. Amsterdam: Harcourt.
- Hoover, D. W. (2015). The effects of psychological trauma on children with autism spectrum disorders: A research review. *Review Journal of Autism and Developmental Disorders*, 2, 287–299. <https://doi.org/10.1007/s40489-015-0052-y>.
- International Society for Traumatic Stress Studies. (2019). Posttraumatic stress disorder prevention and treatment guidelines. Methodology and recommendations. Retrieved from: <http://www.istss.org/treating-trauma/new-istss-prevention-and-treatment-guidelines.aspx>
- Ipci, M., Inci, S. B., Ardic, U. A., & Ercan, E. S. (2017). A case of Asperger syndrome with comorbidity of

- posttraumatic stress disorder and selective mutism: Significant remission with the combination of aripiprazole and eye movement desensitization and reprocessing. *Journal of Clinical Psychopharmacology*, 37(1), 109–110.
- Kerns, C. M., Newschaffer, C. J., & Berkowitz, S. J. (2015). Traumatic childhood events and autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45, 3475–3486. <https://doi.org/10.1007/s10803-015-2392-y>.
- Kildahl, A. N., Bakken, T. L., Iversen, T. E., & Helvershou, S. B. (2019). Identification of posttraumatic stress disorder in individuals with autism spectrum disorder and intellectual disability: A systematic review. *Journal of Mental Health Research in Intellectual Disabilities*, 12, 1–2. <https://doi.org/10.1080/19315864.2019.1595233>.
- Kosatka, D., & Ona, C. (2014). Eye movement desensitization and reprocessing in a patient with Asperger's Disorder: case report. *Journal of EMDR Practice and Research*, 8(1), 13–18.
- Lobregt-van Buuren, E., Sizoo, B., Mevissen, L., & De Jongh, A. (2019). Eye movement desensitization and reprocessing (EMDR) therapy as a feasible and potential effective treatment for adults with Autism Spectrum Disorder (ASD) and a history of adverse events. *Journal of Autism and Developmental Disorders*, 49(1), 51–164. <https://doi.org/10.1007/s10803-018-3687-6>.
- Maïano, C., Normand, C. L., Salvas, M. C., Moullec, G., & Aimé, A. (2016). Prevalence of school bullying among youth with autism spectrum disorders: a systematic review and meta-analysis. *Autism Research*, 9, 601–615.
- Mevissen, L., Lievegoed, R., & De Jongh, A. (2011). EMDR treatment in people with mild ID and PTSD: 4 cases. *Psychiatric Quarterly*, 82, 43–57.
- Mevissen, L., Didden, R., Korzilius, H., & De Jongh, A. (2016). Assessment of PTSD in children with mild to borderline intellectual disabilities. *European Journal of Psychotraumatology*, 7, 29786. <https://doi.org/10.3402/ejpt.v7.29786>.
- Mevissen, L., Didden, R., Korzilius, H., & De Jongh, A. (2017). Eye movement desensitisation and reprocessing therapy for posttraumatic stress disorder in a child and an adolescent with mild to borderline intellectual disability: A multiple baseline across subjects study. *Journal of applied research in intellectual disabilities*, 00, 1–8. <https://doi.org/10.1111/jar.12335>.
- Mevissen, L., Didden, R., & De Jongh, A. (2018). *DITS-LVB Handleiding. Diagnostisch Interview Trauma en Stressoren – Licht Verstandelijke Beperking [DITS-MID Manual. Diagnostic interview trauma and stressors – Mild intellectual disability]*. Accare Child Study Center. <https://www.accare.nl/childstudycenter/opleidingen/bijtscholing/dits-lvb/aanvragen-documenten-dits-lvb>
- Mevissen, L., Didden, R., de Jongh, A., & Korzilius, H. (in press). Assessing posttraumatic stress disorder in adults with mild intellectual disabilities or borderline intellectual functioning. *Journal of Mental Health Research in Intellectual Disabilities*.
- Roberts, L. A., Koenen, K. C., Lyall, K., Robinson, E. B., & Weisskopf, M. G. (2015). Association of autistic traits in adulthood with childhood abuse, interpersonal victimization and posttraumatic stress. *Child abuse & neglect*, 45, 135–142.
- Rumball, F. (2019). A systematic review of the assessment and treatment of posttraumatic stress disorder in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 6, 294–324. <https://doi.org/10.1007/s40489-018-0133-9>.
- Shapiro, F. (2018). *Eye movement desensitization and reprocessing (EMDR) therapy: Basic principles protocols, and procedures*. New York: Guilford Press.
- Spinazzola, J., Blaustein, M., & Van der Kolk, B. A. (2005). Posttraumatic stress disorder treatment outcome research: the study of unrepresentative samples? *Journal of Traumatic Stress*, 18(5), 425–436.
- Taylor, J. L., & Gotham, K. O. (2016). Cumulative life events, traumatic experiences, and psychiatric symptomatology in transition-aged youth with autism spectrum disorder. *Journal of Neurodevelopmental Disorders*, 8, 28. <https://doi.org/10.1186/s11689-016-9160-y>.
- Valiente-Gómez, A., Moreno-Alcázar, A., Treen, D., Cedrón, C., Colom, F., Pérez, V., & Amann, B. L. (2017). EMDR beyond PTSD: A systematic literature review. *Frontiers in Psychology*, 8, 1668. <https://doi.org/10.3389/fpsyg.2017.01668>.
- Wood, J. J., & Gadow, K. D. (2010). Exploring the nature and function of anxiety in youth with autism spectrum disorders. *Clinical Psychology: Science and Practice*, 17(4), 281–292.
- World Health Organization. (2013). *Guidelines for the management of conditions specifically related to stress*. Geneva: WHO. Retrieved from http://www.who.int/mental_health/emergencies/stress_guidelines/en/.

Eyeblink Reflexes

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Synonyms

Corneal reflex; Startle response

Definition

The first and most reliable component of the startle reflex in humans. The blink reflex is an involuntary blinking of the eyelids elicited when the cornea is stimulated by touch, bright light, loud sounds, or other peripheral stimuli. The evolutionary purpose of this involuntary response is believed to be a survival-related function, to protect the eyes from potentially harmful stimuli (i.e., threat). When a stimulus is presented to the cornea of the eye, sensory information is carried to the trigeminal nucleus and relayed to the accessory abducens and abducens motor nuclei, initiating a motor response. The eyeblink consists of a rapid contraction of the orbicularis oculi muscle surrounding the eye; this is the most sensitive component of the blink. Strength of the response can be measured by electromyographic activity (EMG) of the orbicularis muscle during the eyeblink, or blink magnitude. Because EMG is quantifiable, it is the behavioral variable often used in startle response experiments designed to investigate neurological indices of fear and anxiety (i.e., amygdala activation).

See Also

► [Startle Reflex](#)

References and Reading

- Blumenthal, T. D., Cuthbert, B. N., Filion, D. L., Hackley, S., Lipp, O. V., & van Boxtel, A. (2005). Committee report: Guidelines for human startle eyeblink electromyographic studies. *Psychophysiology*, 42, 1–15.
- Dichter, G. S., Benning, S. D., Holtzclaw, T. N., & Bodfish, J. W. (2010). Affective modulation of the startle eyeblink and postauricular reflexes in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 40, 858–869.

Eyes Test

► [Reading the Mind in Eyes Test](#)

Eye-to-Eye Gaze

► [Mutual Gaze](#)

Eye-Tracking

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Definition

Eye-tracking is a technique in which one or more of a subject's eyes are tracked with the intent of inferring, in a moment-by-moment fashion, what the individual is attending to in his or her visual world.

Historical Background

Eye-tracking has had a long history in psychological research and vision science. Some of the earliest reported work using eye-tracking appeared in the late 1800s and early 1900s, focusing primarily on understanding and optimizing visual processes during reading (e.g., the work of Huey, 1898, 1910). Later work, by Guy Buswell (1935), Alfred Yarbus (1967), and others, would use eye-tracking to study more natural visual scanning processes during the viewing of complex scenes such as those found in paintings or photographs. From these early experiments, it was noted that typical individuals, both young and old, when presented visually with scenes with people, and unless given instructions to the contrary, would spend much of their time looking at the people portrayed. Furthermore, it was noted that given a face to view, subjects would largely attend to the eyes and mouth of the face, despite the fact that these static stimuli never changed. Yarbus noted, in his treatise on eye movements: "The observer's attention is frequently drawn to

elements which do not give important information but which, in his opinion, may do so.” This early work, which advanced the primary of social information processing in natural viewing for typically developing individuals, set the stage for modern investigations of the visual strategies employed by individuals with autism spectrum disorders (ASD).

Current Knowledge

The use of eye-tracking for understanding neuropsychiatric conditions and special populations in general is attractive for a number of reasons. First, at its essence, eye-tracking is a method for seeing how others see, providing a robust, quantitative measure of where someone is looking. Since where someone is looking is highly correlated to where that person’s attention is deployed, eye-tracking can be used to understand temporally changing cognitive processes as they unfold (with caveats; see section “[Limitations of Eye-Tracking](#)”). Second, eye-tracking is today a relatively noninvasive and easily tolerated experimental technology. As compared to the original systems of the early nineteenth century, which were often so uncomfortable that the eye had to be anesthetized with cocaine, modern desktop-mounted video oculography eye-tracking systems (see section “[Types of Eye-Trackers](#)”) can obtain precise measurements of the eye without ever touching any part of the participant. Third, the oculomotor system is early maturing and highly preserved neurophysiological control system. This means that eye-tracking can be an applicable and appropriate technique across the vast cognitive, behavioral, and physiological heterogeneity found in many neuropsychiatric conditions.

The earliest published work using eye-tracking to study individuals with ASD appeared in 2002 (Klin et al. 2002; Pelphrey et al. 2002; van der Geest et al. 2002a; van der Geest et al. 2002b). The general pattern of this work suggested that in many cases, the visual scanning strategies of individuals with ASD are significantly different than that of controls. These initial reports found, for example, that older adolescents and adults with

ASD tended to look more at the mouth and less at the eyes in comparison to control individuals. They also highlighted relationships between communicative ability and scanning patterns, with the somewhat surprising finding that individuals with ASD who looked more at the mouth were actually more communicatively capable than those who looked less. Additional studies have largely confirmed the atypical nature of visual scanning strategies in individuals with ASD, especially under conditions where choices in attentional selection must be made between social and nonsocial stimuli. Results of several studies show, for instance, decreased attention toward social actors, people in general, and faces, and increased attention toward nonsocial background elements of scenes (e.g., Jones et al. 2008; Klin et al. 2002; Pierce et al. 2011; Riby and Hancock 2009; Shic et al. 2011). Other studies also highlight decreased sensitivity in individuals with ASD to communicative and social aspects of interactions, such as gaze cues and shared activities (Freeth et al. 2009; Riby and Doherty 2011; Shic et al. 2011). Finally, eye-tracking studies have related inefficient social information-gathering strategies to poor performance in face recognition tasks in both adults (Spezio et al. 2007) and toddlers and children with ASD (Chawarska and Shic, 2009). Taken together, these studies provide evidence for atypical visual exploratory behaviors in individuals with ASD and promote the use of eye-tracking technology in efforts toward understanding the mechanisms underlying atypically developing social skills in individuals with ASD.

However, it is important to note that the use of eye-tracking in studying ASDs is a developing and emerging field. While originally there was a great deal of enthusiasm regarding atypical distributions of attention toward specific elements of the face (e.g., proportions of mouth and eye scanning), recent reports have highlighted the complex interrelationships between cognitive, social, and behavioral phenotypes and visual exploratory behavior (Chawarska and Shic 2009; Norbury et al. 2009). Indeed, several studies did not replicate atypical patterns of looking at faces or social scenes, even as early as 2002 (van der Geest et al. 2002a, b). The reasons for these disagreements are

currently active areas of exploration. However, there are likely several factors involved, such as heterogeneity of sample populations, variety of experimental paradigms and contexts, and inconsistencies across research sites in use of pre-processing methodology and analytical techniques. Nevertheless, eye-tracking has been and continues to be a powerful methodology for understanding the nature of ASD; as more research using eye-tracking is conducted, its application will likely extend further toward questions regarding the heterogeneity of behavioral and visual exploratory activities in ASD.

Types of Eye-Trackers

There are several types of eye-tracking technologies still in widespread use (Duchowski 2007). However, recently, psychological and behavioral studies of individuals with ASD and other neuropsychiatric conditions have largely begun to rely exclusively on the use of remote video oculography (VOG) methods. In modern video oculography, a video camera or multiple video cameras are positioned so as to be able to obtain a clear image of the participant's eye. These cameras, typically linked up to either specialized hardware or a computer, employ sophisticated computational algorithms in order to extract from the video the position of the participant's pupil(s). In the most common methodology, infrared filters are used to remove contaminating light sources which otherwise complicate signal analysis extraction of the pupil location. In these infrared systems, additional infrared light sources, which are largely invisible to the human eye, are used in order to generate "glints" or corneal reflections which serve as a reference coordinate system for the pupil that is more invariant to participant motion. Additionally, the eye and face of the participants can be lit by on-camera-axis lighting or off-camera-axis lighting. On-camera-axis lighting generates a form of infrared video oculography called "bright-pupil" eye-tracking. In bright-pupil eye-tracking, illumination of the eye causes a "red-eye effect" (similar to what occurs when a flash is reflected off someone's eye in a camera photograph). This makes the pupil appear bright. In off-camera-axis lighting,

the pupil remains dark because infrared light is not reflected, resulting in a "dark pupil." Though some manufacturers suggest one form of lighting is superior for certain individuals (e.g., for those with light blue eyes as compared to those with dark brown eyes), there have been no independent scientific studies showing one method is actually superior in practical applications and modern use.

Another form of eye-tracking which has been in many ways superseded by video oculography methods, at least in behavioral studies of individuals with neuropsychiatric disorders, is electrooculography (EOG). Electrooculography uses conductive electrodes placed nasally and temporally to the eye, together with a reference electrode, in order to measure the potential difference between the front of the eye and the back of the eye (the corneofundal potential; see Brown et al. 2006; Marmor and Zrenner 1993). This method of eye-tracking typically is noisier than commercial video oculography systems, and the head must remain relatively immobilized, as compared to video oculography techniques which can accommodate relatively larger amounts of head movements. However, EOG systems are relatively inexpensive compared to VOG eye-tracking systems, making their use a viable alternative in situations where precision is not at a premium and participants are very compliant to instructions and immobilization.

Finally, a form of eye-tracking used primarily in primate and animal research employs scleral coils. With scleral coils, a conducting ring is placed directly on the sclera of the eye and an enclosing alternating magnetic field induced around the head of the participant. As the eye changes position, voltages generated within the coil give the position of the eye, giving extremely precise recordings of eye position. The main drawback of this technique is the physical discomfort associated with inserting the coil directly into the eye and the risk of corneal abrasion. Still, recent work has identified the lead from the coil to the measuring device as a major source of discomfort for scleral coils, and strides have been made using wireless scleral coils which may make the technology more usable for fine-

grained detection of eye movements in the future (Roberts et al. 2008).

Mechanics of Eye Movements and Common Eye-Tracking Outcome Variables

Primary classifications of eye movements include saccades, fixations, and smooth pursuit. Saccades, a term coined by French psychologist Louis Javal in 1879, are rapid, ballistic movements of the eye. Research has shown that awareness of the visual world is suppressed during saccades, an effect neatly summarized by Dodge (1900). Dodge's observation was that when an individual stands in front of a mirror, looking at different positions on his or her own eyes, that individual does not detect any movement of the eyes. However, an observer standing immediately to the side of the person in the mirror will see the eyes moving rapidly. This effect, known as saccadic suppression, is associated with decreased conscious awareness of the visual world during saccades. Fixations are the complement of saccades and represent periods in which the pupil is focused relatively stably and steadily on a particular location. By contrast, smooth pursuits are indicated by slower, sweeping motions of the eye and are found when the eye is used employed to visually track a moving target. Interestingly, smooth pursuits cannot be elicited voluntarily in the absence of a target and require a slow-moving target to manifest.

Fixation/saccade identification algorithms have been advanced in order to segregate saccades from fixations in eye-tracking data. However, it is unclear if these algorithms are effective in drawing clear distinctions between saccades and fixations at modern eye-tracking speeds (60 Hz) and resolutions (Shic et al. 2008a, b). Nevertheless, these fixation identification algorithms are in widespread use and are supported by most eye-tracking manufacturers. By comparison, smooth pursuit recognition, which would affect the interpretation of studies employing dynamic stimuli, is currently not a standard analytical technique.

Perhaps the most common technique used in analysis of eye-tracking data is region-of-interest (ROI) analysis. In ROI analysis, the scene presented to the participant is divided into a series of

not necessarily (but usually) nonoverlapping regions, and the amount of time spent by the participant looking at each of these regions is calculated. As studies of natural activities have shown that people tend to be looking for information they need, it is inferred that more examined areas correspond to areas found by observers to be more important. Typically, for comparability purposes, looking times are expressed as a proportion of the trial time. Some researchers choose to exclude from analyses eye-tracking data acquired during saccades due to diminished awareness of the scene by the participant, but others choose to incorporate all data into proportion data and/or looking time reports.

Additional measures employed in eye-tracking research are extensive (Jacob and Karn 2003), and there exists a large body of research on sophisticated techniques for processing eye-tracking data (see Duchowski 2007; see Holmqvist et al. 2011a for reference). While many of these techniques are theoretically and conceptually interesting, it is also important to note that, in many cases, the relationships between derived eye-tracking variables and physiological or behavioral constructs is unclear. A notable exception is the detection of blinks. The inhibition of blinks has recently been shown to correspond to decreased attribution of salience to social scenes in toddlers with ASD (Shultz et al. 2011). Concerted and rigorous explorations of the space of possible eye-tracking measures may thus more firmly ground our understanding of outcome eye-tracking measures, from psychological, neurophysiological, and behavioral perspectives, in the years to come.

Limitations of Eye-Tracking

Interpretations of the results of eye-tracking experiments often involve assumptions regarding the concurrent relationship between the position of eye and the allocation of attention. However, attention and eye movements are not always coupled; indeed, research suggests that the movement of attention toward a particular point in the world precedes an eye movement toward that location by 100–120 ms. In addition, attention can be moved *covertly*, i.e., without an explicit, overt eye movement. Research has shown,

however, that when a movement of the eye is made, it must be preceded by focal attention to that location. Thus, the occurrence of an eye movement suggests that attention has already been deployed to the target of that eye movement; on the other hand, when the eye is relatively still, standard interpretations of eye-tracking data rely on an assumption that the point of fixation is the same as the point of attention. For practical purposes, this is often the case (Duchowski 2003; Holmqvist et al. 2011; Kowler 1990).

Future Directions

Keith Rayner, one of the pioneers of eye-tracking research, especially in studies of reading, notes that there have been four main eras of eye-tracking research (Rayner 2009). In the first era (early 1900s), many of the basic properties about eye movements were discovered. In the second era (1930s to 1950s), experimental work in eye-tracking research took a behaviorist position, classifying and quantifying, in context, the myriad behaviors comprising eye movements. In the third era (1970s to 1990s), the cognitivist revolution brought new perspectives for delving deeper into the mechanisms and cognitive processes underlying eye movements. Rayner notes that we are now in the fourth era of eye-tracking research, one where quantifiable and predictive models of eye movements, indexing underlying cognitive substrates, can be developed.

While Rayner's summary mainly refers to studies of reading, the same classification of eras can be applied to eye-tracking research in social visual information processing, with the understanding that this field lags research in reading by about a decade. Currently, there is a great deal of interest in using eye-tracking technology for understanding autism, and much of this effort is aimed at understanding the specific individual behavioral and cognitive characteristics that affect visual scanning of social information in autism. Once this heterogeneity of behavior can be decoded, it is hoped that eye-tracking research in autism will also enter into its fourth era, where the accurate models of eye movements can help us

both understand individual variability as well as predict those forms of intervention that will be most efficacious.

The fourth era of eye-tracking research also corresponds with the proliferation and technological development of eye-tracking technology as a whole. In many ways, eye-tracking is now appearing as an almost requisite adjunct methodology for social information processing paradigms that directly target underlying neural substrates, such as functional magnetic resonance imaging and electroencephalography. Combining these multiple sources of information will provide a more complete picture of the multifaceted complexities in ASDs. Given the wealth of information, an increased focus on computational and statistical approaches toward integrating and unifying eye-tracking data with other sources of information will be critical to future research.

Finally, while atypical scanning of social scenes in individuals with ASD has been found by several research groups employing eye-tracking, there is still much work left to do in terms of pinpointing specific behavioral markers of ASD. It is yet unknown whether atypical gaze patterns in ASD are a secondary manifestation of social perceptual disadvantages and to what extent the compounding effects of the atypical experience reflected by these gaze patterns diminish prototypical social development. Current and future work with eye-tracking will thus continue to isolate autism-specific developmental processes and deepen our appreciation of the roles of typical and atypical gaze patterns in shaping the behavioral phenotype of individuals with ASD.

See Also

- [Eye Gaze](#)
- [Visual Processing](#)

References and Reading

- Brown, M., Marmor, M., Vaegan, Zrenner, E., Brigell, M., & Bach, M. (2006). ISCEV standard for clinical electro-oculography (EOG) 2006. *Documenta*

- Ophthalmologica*, 113(3), 205–212. <https://doi.org/10.1007/s10633-006-9030-0>.
- Buswell, G. T. (1935). *How people look at pictures: A study of the psychology of perception in art*. Chicago: The University of Chicago Press.
- Chawarska, K., & Shic, F. (2009). Looking but not seeing: Atypical visual scanning and recognition of faces in 2 and 4-year-old children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 39(12), 1663–1672. <https://doi.org/10.1007/s10803-009-0803-7>.
- Dodge, R. (1900). Visual perception during eye movement. *Psychological Review*, 7(5), 454–465. <https://doi.org/10.1037/h0067215>.
- Duchowski, A. T. (2003). *Eye tracking methodology: Theory and practice* (1st ed.). New York: Springer.
- Duchowski, A. T. (2007). *Eye tracking methodology: Theory and practice*. New York: Springer.
- Freeth, M., Chapman, N., Ropar, D., & Mitchell, P. (2009). Do gaze cues in complex scenes capture and direct the attention of high functioning adolescents with ASD? Evidence from eye-tracking. *Journal of Autism and Developmental Disorders*, 40(5), 534–547.
- Holmqvist, K., Nyström, M., Andersson, R., Jarodzka, H., & van de Weijer, J. (2011a). *Eye-tracking data and dependent variables*. Oxford: Oxford University Press.
- Holmqvist, K., Nyström, M., Andersson, R., Dewhurst, R., Jarodzka, H., & Van de Weijer, J. (2011b). *Eye tracking: A comprehensive guide to methods and measures*. New York: Oxford University Press Inc.
- Huey, E. B. (1898). Preliminary experiments in the physiology and psychology of reading. *The American Journal of Psychology*, 9(4), 575–586. Retrieved July 21, 2008.
- Huey, E. B. (1910). *The psychology and pedagogy of reading: With a review of the history of reading and writing and methods, texts, and hygiene in reading*. New York: Macmillan.
- Jacob, R. J. K., & Karn, K. S. (2003). Eye tracking in human-computer interaction and usability research: Ready to deliver the promises (section commentary). In J. Hyona, R. Radach, & H. Deubel (Eds.), *The mind's eye: Cognitive and applied aspects of eye movement research* (pp. 573–605). Oxford, UK: Elsevier Science.
- Jones, W., Carr, K., & Klin, A. (2008). Absence of preferential looking to the eyes of approaching adults predicts level of social disability in 2-year-old toddlers with autism spectrum disorder. *Archives of General Psychiatry*, 65(8), 946–954.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809.
- Kowler, E. (1990). The role of visual and cognitive processes in the control of eye movement. *Reviews of Oculomotor Research*, 4, 1–70.
- Marmor, M. F., & Zrenner, E. (1993). Standard for clinical electro-oculography. *Documenta Ophthalmologica*, 85(2), 115–124. <https://doi.org/10.1007/BF01371127>.
- Norbury, C. F., Brock, J., Cragg, L., Einav, S., Griffiths, H., & Nation, K. (2009). Eye-movement patterns are associated with communicative competence in autistic spectrum disorders. *Journal of Child Psychology and Psychiatry*, 50(7), 834–842. <https://doi.org/10.1111/j.1469-7610.2009.02073.x>.
- Pelphrey, K. A., Sasson, N. J., Reznick, J. S., Paul, G., Goldman, B. D., & Piven, J. (2002). Visual scanning of faces in autism. *Journal of Autism and Developmental Disorders*, 32(4), 249–261. <https://doi.org/10.1023/A:1016374617369>.
- Pierce, K., Conant, D., Hazin, R., Stoner, R., & Desmond, J. (2011). Preference for geometric patterns early in life as a risk factor for autism. *Archives of General Psychiatry*, 68(1), 101.
- Rayner, K. (2009). Eye movements in reading: Models and data. *Journal of Eye Movement Research*, 2(5), 1–10.
- Riby, D. M., & Doherty, M. J. (2011). Tracking eye movements proves informative for the study of gaze direction detection in autism. *Research in Autism Spectrum Disorders*, 3(3), 723–733. <https://doi.org/10.1016/j.rasd.2009.02.001>.
- Riby, D., & Hancock, P. J. B. (2009). Looking at movies and cartoons: Eye-tracking evidence from Williams syndrome and autism. *Journal of Intellectual Disability Research*, 53(2), 169.
- Roberts, D., Shelhamer, M., & Wong, A. (2008). A new wireless search-coil system. In *Proceedings of the 2008 Symposium on Eye Tracking Research & Applications* (pp. 197–204). ACM.
- Shic, F., Chawarska, K., & Scassellati, B. (2008a). The incomplete fixation measure. In *Proceedings of the 2008 Symposium on Eye Tracking Research & Applications* (pp. 111–114). Savannah, Georgia: ACM. <https://doi.org/10.1145/1344471.1344500>.
- Shic, F., Chawarska, K., & Scassellati, B. (2008b). The amorphous fixation measure revisited: With applications to autism. In *30th Annual Meeting of the Cognitive Science Society*. Washington, DC: Presented at the 30th Annual Meeting of the Cognitive Science Society.
- Shic, F., Bradshaw, J., Klin, A., Scassellati, B., & Chawarska, K. (2011). Limited activity monitoring in toddlers with autism spectrum disorder. *Brain Research*, 1380, 246–254. <https://doi.org/10.1016/j.brainres.2010.11.074>.
- Shultz, S., Klin, A., & Jones, W. (2011). Inhibition of eye blinking reveals subjective perceptions of stimulus salience. *Proceedings of the National Academy of Sciences*, 108(52), 21270–21275. <https://doi.org/10.1073/pnas.1109304108>.
- Spezio, M., Adolphs, R., Hurley, R., & Piven, J. (2007). Abnormal use of facial information in high-functioning autism. *Journal of Autism and Developmental Disorders*, 37(5), 929–939. <https://doi.org/10.1007/s10803-006-0232-9>.

- van der Geest, J. N., Kemner, C., Camfferman, G., Verbaten, M. N., & Van Engeland, H. (2002a). Looking at images with human figures: Comparison between autistic and normal children. *Journal of Autism and Developmental Disorders*, 32(2), 69–75.
- van der Geest, J. N., Kemner, C., Verbaten, M. N., & Van Engeland, H. (2002b). Gaze behavior of children with pervasive developmental disorder toward human faces: A fixation time study. *Journal of Child Psychology and Psychiatry*, 43(5), 669–678. <https://doi.org/10.1111/1469-7610.00055>.
- Yarbus, A. L. (1967). *Eye movements and vision*. New York: Plenum Press.

F

Face Blindness

► Pareidolic Faces

Face Perception

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Definition

Facial perception refers to the ability to rapidly recognize and understand information from faces. The ability to perceive faces and to use that information to guide and direct behavior plays a critical role in interpreting and forming representations from the social world and in the acquisition and understanding of reciprocal social interaction. Early facial recognition research suggests that humans innately attend to faces and are able to rapidly recognize faces remarkably early in life (Goren et al. 1975). Within 9 min after birth, infants turn their head to a 90° angle to preferentially attend to faces or face-like images. At 42 min after birth, infants

can imitate facial expressions; and by 2 days old, infants can recognize the face of their mother. One-month-old infants show greater pupillary dilation to faces than to nonsocial stimuli suggesting greater interest and arousal in infants when looking at faces. By the end of the first year of life, infants are able to respond to directional gaze, emotional expression, and facial gestures. These early infant preferential face-directed behaviors mark the beginning of a highly protracted development in which repertoires for the face processing system become more sophisticated over time. The examination of facial perception in typical and atypical development, such as in autism spectrum disorders (ASD), has elucidated face perception processes across behavioral, anatomical, and neurophysiological domains and revealed differences in functioning in individuals with ASD and typical development in face perception.

Historical Background

Research with children with ASD has consistently revealed deficits in facial perception surfacing within the first 3 years of life. Children with ASD show impairments in social behaviors related to the perception and processing of information from faces. These impairments include reduced eye contact, joint attention, and social orienting, deficits in the imitation of faces and in

face recognition, and attenuated responses to emotional displays of others (Dawson et al. 2005).

The retrospective examination of joint attention and gaze monitoring of toddlers through home videos of first birthday parties has provided insight into facial perception in ASD. In novel and uncertain situations, typically developing young children will seek affective cues from others' faces. Given that a child's first birthday can be a highly social, yet novel event, the setting provides the opportunity for a range of observable social behavior. Osterling and Dawson collected videotapes of 1st year birthday parties from children who were later diagnosed with ASD and from children with typical development. The tapes were rated by coders blind to the child's subsequent diagnostic status. The authors found that the children later diagnosed with ASD looked at and attended to faces significantly less than the typically developing children. The authors determined that the single most critical factor in recognizing early signs of ASD was a failure to look at others and attend to faces in a typical manner, at or before 12 months old (1994).

Current Knowledge

The dynamic processes related to facial perception can be examined from behavioral, neuroanatomical, and neurophysiological perspectives. Much of our understanding of facial perception has been derived from clinical work and research with individuals with ASD. These impressions suggest that individuals with autism, compared to typical peers, perform worse on face discrimination, recognition, and memory tasks when behaviorally assessed. Additionally, individuals with ASD display atypical development and anatomical activation of the brain structures involved in face processing and exhibit delayed and differential neurophysiological responses to faces.

Behavioral Perspectives on Face Perception

Very early in life, typically developing individuals show an attentional preference for faces and are equipped for the specialization of face processing.

These specialized abilities involve recognizing upright faces better than inverted faces and interpreting complex interactions such as direction of gaze, emotional expressions, and facial gestures. Into middle childhood, the specialization for facial perception grows in complexity. Typically, individuals show better memory for faces than objects. They also derive meaningful social-emotional information from faces through preferential attention toward the eyes and perceive faces as a holistic unit rather than isolating individual facial features (Joseph and Tanaka 2003). In contrast, individuals with ASD attend to inverted faces for the same amount of time as upright faces and recognize inverted faces better than typical individuals by focusing on isolated features of the face rather than the face as a whole (van der Geest et al. 2002). Further, when viewing faces, individuals with ASD fixate on the mouth region rather than the eyes. Accordingly, they exhibit better performance memory for the lower portion of the face than the upper portion of the face but show a decrease in attention to the entire internal region of the face. As a result, individuals with ASD perform in the below average range on tasks of overall face memory ability. Further, individuals with ASD show impairments in the ability to use information from faces to guide behavior, such as failing to monitor a gaze shift and follow a gaze to share attention.

Studies of facial discrimination and recognition using eye tracking suggest that individuals with ASD employ atypical strategies during facial perception tasks. First, individuals with ASD attend to other objects in the environment more often than the face. For example, a pioneering study using eye tracking found that when watching a videotape of the movie *Who's Afraid of Virginia Woolf*, individuals with ASD attended two times more to the actors' mouth, bodies, and objects in the scenes than to the actors' eyes (Klin et al. 2002). This suggests that individuals with ASD do not seek social information and cues from naturalistic exchanges in the way that typical individuals seek social information. Subsequent studies have shown that individuals with ASD utilize atypical spatial distribution fixation strategies exemplified by fixating to, or avoiding specific

regions of the face, focusing on the mouth, rather than the typical direction toward the eyes.

A second atypical face perception strategy that individuals with ASD appear to use involves processing faces utilizing a piecemeal (i.e., feature-based) strategy (Chawarska and Shic 2009). In contrast, typical individuals employ the piecemeal processing strategy when looking at objects but not faces. Typical individuals attend to faces holistically by utilizing a complex-looking strategy called configural processing (Dawson et al. 2005). Configural processing is the perception and encoding of faces as a single holistic unit by incorporating the major features (i.e., eyes, nose, mouth), in respect to other features throughout the face.

To explore the limitations of the configural strategy employed by the typical population, researchers alter the configuration of faces by inverting and partially obscuring the images.

Typical individuals are adept at distinguishing similarities between faces that are upright; however, when the face is inverted, it is more difficult to discern facial feature differences. Inversion appears to alter the perception of the face by challenging the relational facial features (i.e., eye, nose, mouth), which contests the configural processing looking strategy producing what is called the inversion effect. While inverting faces results in the inversion effect, altering a face by forcing one to focus on individual facial features results in the decomposition effect. Even though typical individuals are adept at perceiving faces, once the face is altered from its original composition (e.g., inverted or presented in sections), the accuracy in which the face is identified is challenged. By challenging the configural processing strategy in these two ways, typical individuals have to abandon a configural processing strategy when looking at faces and adopt a piecemeal strategy as if they were looking at an object. This concept provides support that face perception is a highly specialized and sensitive processing system, which when challenged, forces individuals to compensate using differential processing strategies and neuroanatomical mechanisms that are not specialized for face perception tasks.

From studies exploring the configural processing strategy, the decomposition effect and the inversion effect, researchers have found that typical individuals attend to inverted faces for longer lengths of time, are better at recognizing parts of the face when presented in the context of the entire face, and are better at recognition of the eyes compared to the mouth than their peers with ASD (van der Geest et al. 2002). When presented with the inversion face perception task, individuals with autism spend an equal amount of time looking at upright and inverted faces. This is similar to the way typical individuals attend to upright and inverted objects. By employing the object specific, piecemeal processing strategy, individuals with ASD are better at perceiving specific features of the face or partially exposed sections of the face with emphasis on local detail rather than global detail. In fact, individuals with ASD are in some cases better at feature-based matching, especially around the mouth, because they are not inhibited by the implementation of decomposition effect.

These behavioral-based face perception studies suggest that typically developing individuals use the configural processing strategy to perceive faces and the piecemeal processing to perceive objects while individuals with ASD appear to apply the piecemeal strategy to process both faces and objects. The benefits of configural processing strategies are reflected in better performance on face discrimination and recognition tasks and better memory for faces relative to objects for typical individuals. In contrast, children with ASD perform equally to typical peers and in some cases better on nonface memory recognition tasks that include objects such as buildings, animals, houses, and bicycles (Blair et al. 2002).

Neuroanatomical Perspectives on Face Perception

Much of our understanding of the neuroanatomical structures related to face perception has been driven by research with individuals with impairments in face perception. Studies of the brain in individuals with prosopagnosia, a disorder characterized by intact object recognition ability, but

impairment in the ability to recognize faces, provide a model for face perception that can be applied to ASD. Neuroanatomical findings in individuals with prosopagnosia, who exhibit behavioral face perception impairments similar to those found in ASD, indicate damage to particular cortical regions, such as the fusiform gyrus, indicating a critical role in face perception for this neural region (De Renzi et al. 1994). By utilizing functional magnetic resonance imaging (fMRI), structural magnetic resonance imaging (MRI), and positron-emission tomography (PET), neuroimaging research has provided further evidence that occipitotemporal cortical and other neural structures are functionally abnormal in individuals with ASD during facial perception. The regions identified as playing a primary role in face processing include the fusiform gyrus, the posterior superior temporal sulcus, and the amygdala. This dynamic human face processing system that is linked to the structural encoding of facial features, processing emotional expressions, and interpreting biological movement is critical to understanding the fundamental neural circuitry of the “social brain” (Pelphrey et al. 2007).

Fusiform Gyrus

Research over the last decade has verified a region of the fusiform gyrus that activates more strongly to faces than any other visual stimuli and appears to be specialized for face processing and facial expression discrimination. The fusiform face area (FFA), located in the medial-lateral region of the fusiform gyrus, responds almost twice as strongly to faces than wide varieties of nonface objects (Kanwisher 2000). However, additional research adds complexity to the role of the FFA region. After finding that the FFA is activated while viewing objects that fall in an individual’s area of expertise (e.g., car experts display FFA activation when viewing images of automobiles), Gauthier, Tarr, Anderson, and Skudlarski proposed that the FFA region is not only responsible for face perception but is responsible for the mediation of specific visual expertise. Gauthier and colleagues supported this conclusion by showing that training a group of college students to identify

computer generated 3-D objects (called Greebles) results in an increase of activity of the FFA (1999).

Rather than increased activity in the fusiform gyrus when looking at faces as seen in typical development, neuroimaging findings suggest that individuals with ASD display reduced activation of the fusiform gyrus while looking at faces (Schultz et al. 2000). Further, individuals with ASD exhibit an increase in activation in the inferior temporal gyri, a surrounding anatomical region specialized for object recognition. While for typical individuals, viewing objects of expertise, such as faces, results in FFA activation, individuals with ASD show FFA activation only when looking at images of their specialized interest. While the fusiform gyrus is an area linked to facial perception in typical individuals, for individuals with ASD, this “social brain region” fails to activate to faces in the same manner.

Amygdala

While there is strong research to support the FFA role in the perception of faces, the amygdala plays a significant role in detecting emotional expressions on faces. The amygdala is a fast-responding structure that plays a critical role in notifying other brain regions of emotional arousal through a mediated reward system. This structure quickly and reliably reacts to salient environmental stimuli, especially fear, and plays a role in assigning significance and constructing social judgments, such as inferring personality characteristics from pictures of the face or facial features (LeDoux 1996).

The allure of the amygdala in ASD research was initially recognized in postmortem cases of individuals with ASD who appeared to have limited dendritic tree development resulting in an amygdala that was small and densely packed compared to typical individuals (Bauman and Kemper 1985). Adolphs found that individuals with ASD demonstrated similar impairments to those with focal lesions to limbic structures, including the amygdala. While individuals with ASD are able to form representations of faces and understand basic knowledge of emotions, they are unable to link the perception of faces to social judgments that are often revealed by facial expressions.

Neuroimaging studies of individuals with ASD have reported hypoactivation of the amygdala when viewing images of emotional and mental states of another individual. For example, Pelphrey and colleagues demonstrated that when emotional faces were presented naturalistically through a dynamic video display, all three components of the broader social brain – the amygdala, the superior temporal sulcus, and the fusiform gyrus – were hypoactive (2007). A study examining the perception of emotionally expressive faces in individuals with amygdala and hippocampal lesions found that there was a strong correlation between the extent of amygdala lesions and hypoactivation of the FFA, suggesting that the amygdala plays a direct role on the FFA when interpreting emotionally expressive face (Vuilleumier et al. 2004).

Superior Temporal Sulcus (STS)

The STS region of the brain is responsible for the detection and processing of biological motion (Pelphrey et al. 2007). In synchrony with the amygdala and fusiform gyrus, this neural network is important for linking structural encoding of facial features and emotions to biological motion. Perception of biological motion is thought to underlie impairments in attention and imitation, skills that are strongly linked to language and social development.

Since past research examined the face processing system through the display of emotional expressions using still image stimuli, Pelphrey et al. (2007) were interested in how presenting dynamic facial expressions affects the face processing system. They found that individuals with ASD display reduced activation in the STS, fusiform, and amygdala when faces contain dynamic information, such as movement, relative to static snapshots of the same emotional expressive face. These results were specific to the social brain and were not observed outside of the human face processing system (i.e., STS, fusiform gyrus, and amygdala).

The STS also plays a role in comprehending intentions and detecting “errors” in social situations (Pelphrey et al. 2007). This region modulates tasks involving the attribution of intention

in the context of dynamic social situations. During situations in which an individual’s actions do not fulfill the expectation (e.g., a character in a story is not looking in the direction that the viewer would think he/she should), there was an increase in blood flow to the STS in a typical observer.

However, for individuals with ASD, there was reduced activity in the STS region. The lack of observed activity in the STS during attribution tasks in ASD provides further evidence that the STS region plays a strong role in the social dysfunction in ASD.

Neurophysiological Perspectives on Facial Perception

By utilizing neurophysiological measures such as event-related potentials (ERPs), scientific studies have provided insight into the neuropsychological processes underlying face perception and face perception impairment in ASD. While fMRI studies have allowed researchers to efficiently investigate abnormal regional brain activity during facial performance tasks, electroencephalogram (EEG) and event-related potentials (ERPs) have allowed researchers to examine the temporal characteristics and strength of neural activity by assessing oscillatory activity in response to viewing faces. Unlike fMRIs, which cannot determine the timing of neuronal firing, EEG/ERPs are able to monitor the timing of the summation of a collection of neuronal postsynaptic potentials firing at the resolution of milliseconds. Most studies utilizing EEG data concentrate on the amplitude, scalp topography, and latency. Amplitude is considered a representation of the amount of neuronal resources recruited for a particular process. Scalp topography distribution differences are thought to represent the location of the originally generated neuronal activity, and the timing of information processing speed is represented through the wave-like display of troughs and peaks, known as latency. Because simply passive viewing of stimuli is needed for EEG/ERP studies, valuable information can be gathered from children with significant social communication impairments through the use of EEG/ERP to effectively and noninvasively address fundamental questions

about the neural basis of face processing in individuals with ASD.

Electrophysiology studies are particularly salient in face processing research because of the discovery of a face-sensitive component that occurs with a negative slope at approximately 170 milliseconds poststimuli exposure. The N170 represents one stage in a series of stages of face processing; the P100, a positive going peak around 100 milliseconds, reflects neural activity in basic visual processing; the N170 reflects structural encoding of the face; and the N250 reflects higher order recognition such as affect or identity recognition. The N170 is recorded over the posterior temporal lobe and is greater in the right hemisphere than the left hemisphere. The N170 responds quicker to face and eye stimuli alone, rather than objects or inverted faces. Slight changes in face composition or inversion alter the latency and amplitude of this component. In typical children, the N170 undergoes a prolonged period of development and does not reach full maturation until late adolescence. Between 4 and 15 years of age, the precursor to the N170, identified as the prN170, is measured from the same electrode configuration as the N170 in adults but is more positive (does not extend beyond the baseline) than the fully matured N170 component in adults. The prN170 in children ages 3–6 years is shown to have a faster response to faces. Similar to adults, this component is shown to have preferential responses to faces than objects.

McPartland, Dawson, Webb, and Panagiotides documented the first report of an altered N170 pattern in adolescents and adults with ASD (2004). Individuals with ASD showed a slower latency when looking at faces than furniture and failed to show the face inversion effect. Additionally, researchers observed a slower speed of information processing which disrupts early structural encoding. These differences in the timing of cortical processing suggest that individuals with ASD have abnormal circuitry or delayed neural processes that modulate the face processing system.

Children with ASD also show disruptions to this physiological measure of face processing.

They demonstrate longer prN170 responses to faces than objects. By the age of 6, compared to typically developing peers who continue to show faster responses to faces over objects, children with ASD show no latency differences to faces versus objects (Webb et al. 2006). In addition, research indicates that children with ASD display structural encoding of faces that is disrupted and slowed. While typically developing children exhibit a faster prN170 response to faces than objects, individuals with ASD exhibit a slower prN170 response to faces than objects.

Differential activation patterns have been observed using ERP studies in typical children comparing familiar and unfamiliar stimuli (i.e., caregivers to strangers, familiar objects to unfamiliar objects) as early as 6 months. This suggests early preferential neurological responses to familiar over unfamiliar faces and objects (de Haan and Nelson 1999). Typically developing infants and young children display ERP differences when viewing familiar and unfamiliar faces and objects at the P400 component recorded from posterior electrodes. Children with ASD, however, fail to show a differential P400 component when looking at familiar versus unfamiliar faces. Instead, children with ASD display an enhancement in the P400 component when looking at favorite objects versus unfamiliar objects. These results provide evidence that children with ASD have specific impairment in processing faces, but not objects.

Children with ASD also show abnormal electrophysiological responses when viewing emotional expressions. Compared to typical peers, children with ASD show a slower and more positive N300 wave in response to fear and exhibit abnormal scalp topography. This slower information processing for faces in the N300 for emotional stimuli is associated with greater severity in social domains such as joint attention and social orienting (Dawson et al. 2004).

Neurophysiological studies in early childhood provide insight into the structural and neurological mechanisms that are involved in the development of face processing. Face processing impairments examined at the neurophysiological level provide insight into social brain functioning

and the face processing system in individuals with ASD from infancy and throughout development.

Future Directions

Faces hold special significance and convey valuable social information early in life. Face perception provides a foundation for the development of social cognitive skills such as sharing attention and understanding emotions. Given the prominent role of face perception in social cognition, the study of ASDs has provided insight into the behavioral, neuroanatomical, and neurophysiological components of face perception.

Behavioral studies of face perception in individuals with ASD have yielded insight into visual scanning, face processing strategies, and the role of expertise of processing information. Imaging studies have identified structures related to the processing of faces, such as the fusiform gyrus, superior temporal sulcus, and amygdala, and demonstrated that the structures of the “social brain system” involved in face perception appear to be functioning differently in ASD. Neurophysiological studies have elucidated face-specific brain waves and revealed temporal delays in the processing of faces in ASD.

The ability to process and gather information from facial expressions is crucial for successful social interactions. This unique ability allows humans to understand affective cues in uncertain situations, such as situations that may be dangerous and important for survival, and to accurately read the emotions of another individual. This ability provides attentional and directive focus through gaze monitoring of others and provides a catalyst to overtly and covertly mimic the facial expressions and affective states of others, such as states that offer the opportunity for bonding or shared reciprocal experience (Chawarska and Shic 2009). While face perception provides immediate insight and interpretation of the social world, over time, and development, it is clear that early developing face processing skills set the foundation for the maintenance of exchanges and social relationships.

References and Reading

- Adolphs, R., Sears, L., & Piven, J. (2001). Abnormal processing of social information from faces in autism. *Journal of Cognitive Neuroscience*, 13(2), 232–240.
- Bauman, M., & Kemper, T. L. (1985). Histoanatomic observations of the brain in early infantile autism. *Neurology*, 35(6), 866–874.
- Blair, R., Frith, U., Smith, N., Abell, F., & Cipolotti, L. (2002). Fractionation of visual memory: Agency detection and its impairment in autism. *Neuropsychologia*, 40, 108–118.
- Chawarska, K., & Shic, F. (2009). Looking but not seeing: Atypical visual scanning and recognition of faces in 2 and 4-year-old children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 39, 1663–1672.
- Dawson, G., Webb, S., Carver, L., Panagiotides, H., & McPartland, J. (2004). Young children with autism show atypical brain responses to fearful versus neutral facial expressions. *Developmental Science*, 7, 340–359.
- Dawson, G., Webb, S. J., & McPartland, J. (2005). Understanding the nature of face processing impairment in autism: Insights from behavioral and electrophysiological studies. *Developmental Neuropsychology*, 27(3), 403–424.
- de Haan, M., & Nelson, C. A. (1999). Brain activity differentiates face and object processing in 6-month-old infants. *Developmental Psychology*, 35, 1113–1121.
- De Renzi, E., Perani, D., Carlesimo, G. A., Silveri, M. C., & Fazio, F. (1994). Prosopagnosia can be associated with damage confined to the right hemisphere – An MRI and PET study and review of the literature. *Neuropsychologia*, 32(8), 893–902.
- Gauthier, I., Tarr, M. J., Anderson, A. W., Skudlarski, P., & Gore, J. C. (1999). Activation of the middle fusiform “face area” increases with expertise in recognizing novel objects. *Nature Neuroscience*, 2, 568–573.
- Goren, C., Sarty, M., & Wu, P. (1975). Visual following and pattern discrimination of face-like stimuli by newborn infants. *Pediatrics*, 56, 544–549.
- Joseph, R. M., & Tanaka, J. (2003). Holistic and part-based face recognition in children with autism. *Journal of Child Psychology and Psychiatry*, 44, 529–542.
- Kanwisher, N. (2000). Domain specificity in face perception. *Nature Neuroscience*, 3, 759–763.
- Klin, A., Jones, W., Schultz, R., Volkmar, F. R., & Cohen, D. J. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809–816.
- LeDoux, J. (1996). *The emotional brain*. New York: Simons & Schuster.
- McPartland, J., Dawson, G., Webb, S., Panagiotides, H., & Carver, L. (2004). Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 45, 1235–1245.

- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders*, 24, 247–257.
- Pelphrey, K., Morris, J., McCarthy, G., & LaBar, K. (2007). Perception of dynamic changes of facial affect and identity in autism. *Social Cognitive and Affective Neuroscience*, 2, 140–149.
- Schultz, R., Gauthier, I., Klin, A., Fulbright, R., Anderson, A., Volkmar, F., et al. (2000). Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and asperger syndrome. *Archives of General Psychiatry*, 57, 331–340.
- van der Geest, J. N., Kemner, C., Verbaten, M. N., & van Engeland, H. (2002). Gaze behavior of children with pervasive developmental disorder toward human faces: A fixation time study. *Journal of Child Psychology and Psychiatry*, 43, 669–678.
- Vuilleumier, P., Armony, J. L., Driver, J., & Dolan, R. J. (2004). Distant influences of the amygdala lesion on visual cortical activation during emotional face processing. *Nature Neuroscience*, 7(11), 1271–1278.
- Webb, S. J., Dawson, G., Bernier, R., & Panagiotides, H. (2006). ERP evidence of atypical face processing in young children with autism. *Journal of Autism and Developmental Disorders*, 36(7), 881–890.

Face Processing in Autism: Active Avoidance of the Eyes Versus Passive Indifference

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Definition

A person's face is the source of their identity, a reflection of their internal emotions, and the means by which they communicate thoughts, feelings, and intentions to others. Most people are "expert" face perceivers – able to identify a familiar face, interpret a facial expression, and follow a person's gaze, instantaneously, effortlessly, and without conscious forethought. However, many individuals on the autism spectrum experience

difficulties with face processing and struggle to understand the perceptual and social cues conveyed by the human face.

Current Knowledge

If individuals on the autism spectrum have difficulties recognizing faces and understanding the emotional and social cues in a face, this could contribute to problems in the navigation of everyday interpersonal interactions. In this article, we will discuss the face processing strategies employed by people on autism spectrum and their problems with recognizing face identities and expressions, gaze following, and eye contact. We reason that impairments in face processing abilities have cascading effects on the social and emotional functioning of people on the autism spectrum that further exacerbate their difficulties with social communication and interaction. We propose that the pattern of face impairment in autism is best explained by a failure to attend to the eye information in a face. To account for this eye deficit, we will compare two accounts: the "Eye Avoidance" hypothesis, in which it is proposed that people on the autism spectrum actively avoid the eye region, and the "Eye Indifference" hypothesis, which claims that people on the autism spectrum do not show preferential attention to the socially-relevant information conveyed by the eyes.

Deficits in Facial Identity

The human face is the physical manifestation of one's identity and the primary source of how people individuate one another as conspecifics, each with a distinct personality and personal history. Although it is not a defining characteristic of the disorder, many people on the autism spectrum are impaired in their ability to perceive and recognize facial identity. Research has shown that persons on the autism spectrum have difficulties with the perceptual discrimination of different facial identities (Behrmann et al. 2006; Tantam et al. 1989; Wallace et al. 2008), are impaired in their recognition of familiar faces (Boucher and Lewis 1992), and have problems with learning to

recognize unfamiliar faces (Blair et al. 2002; Boucher and Lewis 1992; Gepner et al. 1996; Hauck et al. 1998; Klin et al. 1999). These impairments do not reflect a breakdown in general perceptual processing, because individuals on the autism spectrum perform equivalently or even sometimes better than control participants on tasks that involve the perception and recognition of nonface objects, such as cars and houses (Wallace et al. 2008; Wolf et al. 2008; Weigelt et al. 2013).

However, not all individuals on the autism spectrum have difficulties with face recognition. In their extensive review of the literature, Weigelt et al. (2012) found that about half of the reviewed studies ($N = 46$) supported differential impaired face processing in autism, whereas the other half ($N = 44$) showed no difference between ASD and non-ASD control groups. Nevertheless, in the studies demonstrating face deficits, systematic breakdowns in face processing were identified. Face deficits in autism are most pronounced when the task involves either immediate or long-term memory for faces. Furthermore, when identifying unfamiliar faces, children on the autism spectrum process eye information differently than typically developing children. On a part/whole task intended to measure holistic strategies, children on the autism spectrum relied less on the eye features than neurotypical children (Wolf et al. 2008). On a series of face matching tasks, participants with autism performed more poorly than neurotypically developing children and children diagnosed with Williams Syndrome on items that tested the upper eye region of the face, but performed on par with comparison groups on items that tested the lower mouth region (Riby et al. 2009). One implication of the converging research evidence is that individuals on the autism spectrum might not have a basic impairment in face recognition, but that they employ face recognition strategies that are different from neurotypical individuals.

Deficits in Emotion

Facial expressions play a key role in our everyday interactions with others because they are our moment-to-moment manifestations of our internal emotions or the emotions that we wish to project

to others (Ekman and Friesen 1971). Therefore, success in social interactions relies on the capacity to recognize and interpret facial emotions in a social context. In typically developing children, the ability to discriminate basic facial expressions, such as anger, fear, sadness, happiness, and surprise, appears by approximately 4 months (Walker-Andrews 1998) and between 8 and 10 months, infants begin to use the emotional expressions of parents or caregivers to form their responses to events in their environment (Camras and Shutter 2010). If individuals on the autism spectrum struggle in their ability to perceive and interpret facial expressions, this would invariably lead to difficulties in everyday social exchanges.

Although there is a clear connection between the accurate recognition of facial expressions and everyday social competence, there is inconsistent empirical evidence concerning whether individuals with autism are differentially impaired in their ability to perceive and interpret facial emotions. On the one hand, there are studies providing results consistent with impaired emotion perception and recognition in autism populations (Corbett et al. 2009; Davies et al. 1994; Loveland et al. 2008; Tantam et al. 1989; Tanaka et al. 2012). Some studies show that individuals on autism spectrum perform worse than control participants on tasks requiring the labeling of primary basic emotions, such as anger, disgust, and sadness (Bölte and Poustka 2003; Boraston et al. 2007). On the other hand, many studies report no differences between typical and autism population participants in their perception and recognition of facial expressions (Baron-Cohen et al. 1997; Castelli 2005; Jones et al. 2011; Lacroix et al. 2009; Ozonoff et al. 1990; Spezio et al. 2007). However, when the expression studies were combined in a recent meta-analysis of 48 papers, Uljarevic and Hamilton (2013) found that people on the autism spectrum performed below control participants, with a calculated mean effect size of 0.80, and an effect size of 0.41 after correction for publication bias. Relative to neurotypical groups, recognition of happiness was only marginally impaired in autism. Individuals on the autism spectrum performed most poorly on their recognition of fear, and the

difference between their recognition of fear and happiness was marginally reliable. The relative sparing of the happy expression and the selective impairment of the fear expression is informative, because whereas the perceptual cues for happiness are centered on the mouth region (i.e., upturned smile), fear requires attention to cues contained in the upper eye region (i.e., widened eye and arched eyebrows) (Dimberg and Petterson 2000; Dimberg and Thunberg 1998; Smith et al. 2005). Thus, evidence from the identity and expression recognition studies suggests that individuals on the autism spectrum might be predisposed to ignore information in the upper eye region.

Cognitive Strategies in Face Processing and Autism

What is the source of the face processing deficits in autism? A likely candidate is the failure to process information in the eye region of the face. The eye region is a perceptually rich area of the face that provides critical cues for recognizing identity (Schyns et al. 2002; Vinette et al. 2004) and expression (Calder and Jansen 2007; Smith et al. 2005). If individuals on the autism spectrum fail to attend to eye information, it follows that they would be impaired in face identity and expression recognition tasks and have difficulty interpreting social cues in their daily lives.

Eye tracking is a powerful technique for revealing cognitive strategies and it provides insights into the perceptual strategies guiding face processing in autism (Black et al. 2017; Boraston and Blakemore 2007). When a photograph of a face is presented to neurotypical participants, the majority of their looking behaviors (70–80%) are devoted to the eye region of the face (Vinette et al. 2004; Walker-Smith et al. 1977). In contrast, when adults on the autism spectrum look at a face, they look less at the eye, nose, and mouth features of the face and more at the external features (hair, chin, clothing) (Pelphrey et al. 2002). Critically, when individuals with autism do attend to the facial features, their gaze patterns are directed more to the mouth area; they spend less time inspecting the eyes (Dalton et al. 2005; Pelphrey et al. 2002; Spezio et al. 2007).

In the real world, faces are not static objects, but are dynamic entities that constantly move and change in reaction to social events in the environment. To capture dynamic social situations, eye movements were recorded while individuals with autism and control participants viewed a film depicting two actors engaged in a highly emotional conversation. Whereas neurotypicals spent the majority of time fixated on the eyes of the actors, individuals on the autism spectrum spent more time looking at the mouths of the actors and unrelated objects in the scene and less time on actors' eyes (Klin et al. 2002). In a replication study, Speer et al. (2007) also found that individuals on the autism spectrum spent significantly less time looking at the eyes than did their typically developing peers. Interestingly, a link was found between social competence as measured by the Social Rating Scale (SRS) and scanning behaviors, such that as social competence decreased, the participant's fixation time on the eyes also decreased. The authors suggested that the amount of time an individual spends fixating on others' eyes in social interactions may reflect the degree to which they are able to interpret social information as conveyed through facial cues.

Developmentally, eye neglect in autism emerges early and provides a reliable predictor of social impairment for toddlers who are later diagnosed with autism (Jones et al. 2008; Klin and Jones 2008). However, the precise age at which these eye contact differences first emerge is unclear. A prospective study using dynamic, but relatively still, face stimuli suggests that infants with autism could be less likely to look at the eyes as early as 3 months of age (Rutherford et al. 2015), but another prospective study using dynamic videos of an individual interacting with viewers found that infants later diagnosed with autism showed normative patterns of eye looking at 2 months of age, with fixation on the eyes declining gradually over subsequent observations (Jones and Klin 2013).

Eye Avoidance or Eye Indifference?

There are two accounts addressing why individuals on the autism spectrum might not show a

preference for the eyes. The “Eye Avoidance” hypothesis asserts that people on the autism spectrum actively divert their attention away from the eyes as a strategy to reduce and regulate social interactions. In contrast, the “Eye Indifference” hypothesis claims that individuals on the autism spectrum are insensitive to the social information conveyed by the eyes, which therefore are perceived to be a region of the face that is not particularly informative, engaging, or adaptively useful. Whereas the “Eye Avoidance” account regards the eyes as aversive and as having a negative valence, the “Eye Indifference” account regards the eyes as a nonthreatening and neutral stimulus. In this section, we will discuss the research relevant to the “Eye Avoidance” and “Eye Indifference” accounts and provide an explanation that attempts to reconcile these perspectives.

Eye Avoidance Hypothesis

Despite the importance of the eyes for recognition of identity and expression, the eyes present the most threatening area of the face to many individuals on the autism spectrum. Eye contact conveys a social message of approach and can be interpreted as an invitation for social engagement and intimacy (Kleinke 1986). In everyday face-to-face encounters, social communication is initiated and regulated through the “language of our eyes.” Therefore, for individuals with autism, avoiding eye contact may be an adaptive strategy for deflecting, discouraging, or coping with stressful social interactions. Indeed, people with autism anecdotally comment that looking into another person’s eyes is an unpleasant, even painful experience (Robison 2007). These anecdotal observations have been validated by the self-reports of older children and adults indicating the threat and anxiety associated with looking at the eyes (Corden et al. 2008; Tottenham et al. 2014).

Physiological measures provide an indirect measure of emotional reactivity. It is well established that increased skin conduction is a measure of increased emotional arousal, and therefore, serves as a good biomarker for examining a possible link between eye gaze and emotion in autism (Bradley et al. 2001). Consistent with an Eye Avoidance account, children with autism

exhibit a higher skin reactivity to direct gaze faces than averted gaze faces, suggesting the direct eye contact is perceived as more threatening by participants. In contrast, the skin reactivity of neurotypical children does not change across direct and averted gaze conditions (Kylliäinen and Hietanen 2006). In an important ecological extension of this finding, Kaartinen et al. (2012) measured the skin conductance response of participants on the autism spectrum and neurotypical control participants using live models. Participants viewed the models displaying either an eyes closed, gaze averted, or direct gaze pose through a liquid crystal shutter. Although no group differences were found in skin reactivity between the participants on the autism spectrum and neurotypical participants, the magnitude of arousal for children on the autism spectrum was reliably associated with their social skill abilities (e.g., language, social communication skills, non-verbal play) in the direct gaze condition, but not in the eyes closed or averted gaze conditions, suggesting that engaging in eye contact promotes social competence. Gaze may interfere with face recognition ability in autism, insofar as skin reactivity to direct gaze faces was negatively correlated with face recognition performance; that is, high skin reactivity to direct gaze faces was correlated with poor recognition (Joseph et al. 2008). Although researchers have not always found a reliable link between skin reactivity and eye gaze in autism (Louwerse et al. 2013), null findings might be related to variations in stimulus type (static, videos, live models), task demands, age, and level of functioning of the participants. As a whole, the bulk of the skin conductance evidence indicates that individuals on the autism spectrum are more emotionally aroused when they look at the eyes of faces and this response is accentuated when the eyes of the faces are looking back at them.

At the neuroanatomical level, a network of cortical and subcortical brain structures that includes the fusiform gyrus, the amygdala, and the superior temporal sulcus is central to social and emotional processing of faces. Whereas the fusiform gyrus is selectively sensitive to faces compared to nonface objects (Kanwisher et al. 1997; Puce et al. 1995)

and to familiar faces compared to unfamiliar faces (Lehmann et al. 2004; Rotshtein et al. 2005), the amygdala is sensitive to emotional information in a face (Kawashima et al. 1999; Morris et al. 2002; Whalen et al. 2004). In autism, activation of amygdala is strongly and positively correlated with the time spent fixating on the eyes, relative to neurotypical participants (Dalton et al. 2005; Hadjikhani et al. 2004; Hadjikhani et al. 2017; Kliemann et al. 2012). Although they are not predisposed to look at the eyes, when individuals on the autism spectrum are cued to the eye region, they demonstrate greater bilateral amygdala activation than neurotypical individuals (Hadjikhani et al. 2004). Tottenham et al. (2014) showed that amygdala activity could be increased through an experimental manipulation to the eyes and that those participants with autism who made the fewest natural eye movements toward the eyes exhibited the highest amygdala response when their gaze was experimentally driven toward the eyes. The researchers hypothesized that those individuals on the autism spectrum who found direct gaze to be the most aversive may have modulated their amygdala-mediated arousal by averting their gaze away from direct eye contact.

For many people on the autism spectrum, looking into the eyes of another person is a threatening experience, one that provokes feelings of anxiety and trepidation. As the foregoing research indicates, subjective feelings of discomfort are manifested in physiological responses that include heightened skin conductance and increased amygdala brain activity and these responses are accentuated by direct eye contact. According to the Eye Avoidance hypothesis, choosing not to look at someone's eyes is an adaptive strategy that allows people on the autism spectrum to regulate their social interactions and manage potential feelings of discomfort that these encounters might elicit.

Gaze Indifference Account

In contrast to the Eye Avoidance hypothesis, the Gaze Indifference hypothesis maintains that individuals on the autism spectrum are insensitive to social cues and, consequently, are indifferent to the role that the eyes play in social

communication. Whereas the Eye Avoidance hypothesis assumes an awareness on the part of people on the autism spectrum to the potential social threat of the eyes, the Gaze Indifference hypothesis claims that they have no awareness of the social meaning or significance of eye information. According to this account, insensitivity to eye gaze is not specific to face processing, but reflects a general lack of interest in the social behaviors of others.

Consistent with the Gaze Indifference account, neuroimaging studies have demonstrated that individuals on the autism spectrum show either hypoactivation or nonselective activation in the amygdala to social stimuli (Pelphrey et al. 2005), to faces (Pierce et al. 2001), and to the eyes (Pelphrey et al. 2005). Thus, in contrast to previously discussed findings (Dalton et al. 2005; Tottenham et al. 2014), these studies indicate that for people on the autism spectrum, the affective valence of a stimulus has little effect on the brain structures that are most closely associated with emotional processing.

To support the Gaze Indifference hypothesis, Moriuchi et al. (2017) monitored the eye movements of 2-year-old toddlers on the autism spectrum while they watched a video of an actress speaking into the camera. When cued to the eyes of the actress, the 2-year-olds with autism did not shift their gaze away from the eyes as predicted by the Eye Avoidance account. Unlike the toddlers from the control group, the eye movements of toddlers on the autism spectrum were unaffected by the social cues of the actress, such as when she made eye contact and spoke directly into the camera. Hence, while the toddlers on the autism spectrum did not find the actress' eyes particularly noxious or aversive, they did not respond to the social cues conveyed through her eyes. The results are consistent with a Gaze Indifference account in which the 2-year-olds on the autism spectrum displayed a passive insensitivity to the social cues conveyed by the eyes of others.

Although the foregoing results are consistent with the Gaze Indifference interpretation, the findings are not necessary incompatible with the Eye Avoidance account. Moriuchi and colleagues

speculated that whether an individual on the autism spectrum responds passively or aversively to the eyes of others might depend on factors related to the age and social and cognitive development of the child. Previous eye gaze reports of atypical amygdala activation (Dalton et al. 2005; Tottenham et al. 2014), heightened anxiety (Corden et al. 2008), and atypical skin reactivity (Kaartinen et al. 2012; Kylliäinen and Hietanen 2006) have examined the responses in older children and adults on the autism spectrum, in contrast to the eye-tracking study by Moriuchi et al., which was conducted with young, 2-year-old toddlers. If children on the autism spectrum learn that attending to the eyes is associated with a potential social threat, then the eye contact-social threat connection would require time, experience, and awareness. The frequency of anxiety symptoms in autism increases with age (van Steensel et al. 2011) and is moderated by cognitive functioning (White et al. 2009). It is not surprising then that the eye contact-social threat contingency is not fully established at 2 years of age. One model proposes that people on the autism spectrum could develop anxiety as a result of stressful experiences, including the stressful experience of navigating unpredictable social interactions (Wood and Gadow 2010). Hence, Eye Avoidance behaviors would not be expected to appear until later childhood and early adulthood, when the connection between social anxiety and eye threat is more firmly established.

Future Directions

Summary

In this article, we discussed the face processing abilities and strategies of individuals on the autism spectrum. Although impairments in the recognition of face identities and expressions are not defining characteristics of the autism spectrum, the converging evidence indicates that many individuals on the autism spectrum have difficulty in these areas. Because their recognition abilities for nonface objects are on par with or superior to those of neurotypical individuals, the deficits cannot be attributed to low-level or perceptual visual impairments but are selective to

face processing. Cognitive and eye-tracking experiments indicate that in contrast to neurotypical individuals, individuals on the autism spectrum fail to attend to and encode the information in the eyes – a region of the face that is critical for recognizing identity and expression.

Two hypotheses have been advanced to explain why individuals on the autism spectrum fail to look at the eyes of another person. According to the “Eye Avoidance” hypothesis, individuals on the autism spectrum divert their attention away from the eyes as a strategy to regulate the social threat of eye contact. In contrast, the “Gaze Indifference” hypothesis posits that individuals on the autism spectrum regard the eyes as a socially neutral stimulus that neither attracts nor repels their attention. Although the autonomic, neuroimaging, and eye-tracking results are inconclusive, it is plausible that the indifference and avoidance responses reflect behaviors at different developmental time points. Gaze indifference behavior might be evident in early childhood (e.g., 2 years of age) when the young child on the autism spectrum is relatively insensitive to the social cues in their world. Eye avoidance behaviors may appear later in childhood (e.g., school age) as a result of increased social interaction and a developing sense of self-awareness. As a test of this developmental account, longitudinal studies could be conducted to monitor for a possible “indifference-to-aversion” shift in eye-tracking behaviors.

If the “eyes are windows into the soul,” then it is little surprise that individuals on the autism spectrum should struggle with understanding the emotions and intentions of other people. Although there might be good reasons why people on the autism spectrum choose not to attend to the eyes of others, understanding the source of this strategy is a rich area for future research and potential interventions.

See Also

- ▶ [Gaze](#)
- ▶ [Joint attention](#)
- ▶ [Social Language Development Test](#)

References and Reading

- Baron-Cohen, S., Jolliffe, T., Mortimore, C., & Robertson, M. (1997). Another advanced test of theory of mind: Evidence from very high functioning adults with autism or Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 38(7), 813–822. <https://doi.org/10.1111/j.1469-7610.1997.tb01599.x>.
- Behrmann, M., Avidan, G., Leonard, G. L., Kimchi, R., Luna, B., Humphreys, K., & Minshew, N. (2006). Configural processing in autism and its relationship to face processing. *Neuropsychologia*, 44(1), 110–129. <https://doi.org/10.1016/j.neuropsychologia.2005.04.002>.
- Black, M. H., Chen, N. T. M., Iyer, K. K., Lipp, O. V., Bölte, S., Falkmer, M., et al. (2017). Mechanisms of facial emotion recognition in autism spectrum disorders: Insights from eye tracking and electroencephalography. *Neuroscience & Biobehavioral Reviews*, 80(September), 488–515. <https://doi.org/10.1016/j.neubiorev.2017.06.016>.
- Blair, R. J. R., Frith, U., Smith, N., Abell, F., & Cipolotti, L. (2002). Fractionation of visual memory: Agency detection and its impairment in autism. *Neuropsychologia*, 40(1), 108–118. [https://doi.org/10.1016/S0028-3932\(01\)00069-0](https://doi.org/10.1016/S0028-3932(01)00069-0).
- Bölte, S., & Poustka, F. (2003). The recognition of facial affect in autistic and schizophrenic subjects and their first-degree relatives. *Psychological Medicine*, 33(5), 907–915. <https://doi.org/10.1017/S0033291703007438>.
- Boraston, Z., & Blakemore, S.-J. (2007). The application of eye-tracking technology in the study of autism. *The Journal of Physiology*, 581(3), 893–898. <https://doi.org/10.1113/jphysiol.2007.133587>.
- Boraston, Z., Blakemore, S.-J., Chilvers, R., & Skuse, D. (2007). Impaired sadness recognition is linked to social interaction deficit in autism. *Neuropsychologia*, 45(7), 1501–1510. <https://doi.org/10.1016/j.neuropsychologia.2006.11.010>.
- Boucher, J., & Lewis, V. (1992). Unfamiliar face recognition in relatively able autistic children. *Journal of Child Psychology and Psychiatry*, 33(5), 843–859. <https://doi.org/10.1111/j.1469-7610.1992.tb01960.x>.
- Bradley, M. M., Codispoti, M., Cuthbert, B. N., & Lang, P. J. (2001). Emotion and motivation I: Defensive and appetitive reactions in picture processing. *Emotion*, 1(3), 276–298. <https://doi.org/10.1037/1528-3542.1.3.276>.
- Calder, A. J., & Jansen, J. (2007). Configural coding of facial expressions: The impact of inversion and photographic negative. *Visual Cognition*, 12(3), 495–518. <https://doi.org/10.1080/13506280440000418>.
- Camras, L. A., & Shutter, J. M. (2010). Emotional facial expressions in infancy. *Emotion Review*, 2(2), 120–129. <https://doi.org/10.1177/1754073909352529>.
- Castelli, F. (2005). Understanding emotions from standardized facial expressions in autism and normal development. *Autism*, 9(4), 428–449. <https://doi.org/10.1177/1362361305056082>.
- Corbett, B. A., Carmean, V., Ravizza, S., Wendelken, C., Henry, M. L., Carter, C., & Rivera, S. M. (2009). A functional and structural study of emotion and face processing in children with autism. *Psychiatry Research: Neuroimaging*, 173(3), 196–205. <https://doi.org/10.1016/j.psychres.2008.08.005>.
- Corden, B., Chilvers, R., & Skuse, D. (2008). Avoidance of emotionally arousing stimuli predicts social-perceptual impairment in Asperger's syndrome. *Neuropsychologia*, 46(1), 137–147. <https://doi.org/10.1016/j.neuropsychologia.2007.08.005>.
- Dalton, K. M., Nacewicz, B. M., Johnstone, T., Schaefer, H. S., Gernsbacher, M. A., Goldsmith, H. H., et al. (2005). Gaze fixation and the neural circuitry of face processing in autism. *Nature Neuroscience*, 8(4), 519–526. <https://doi.org/10.1038/nn1421>.
- Davies, S., Bishop, D., Manstead, A. S. R., & Tantam, D. (1994). Face perception in children with autism and Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 35(6), 1033–1057. <https://doi.org/10.1111/j.1469-7610.1994.tb01808.x>.
- Dimberg, U., & Petterson, M. (2000). Facial reactions to happy and angry facial expressions: Evidence for right hemispheric dominance. *Psychophysiology*, 37(5), 693–696. <https://doi.org/10.1017/S0048577200990759>.
- Dimberg, U., & Thunberg, M. (1998). Rapid facial reactions to emotional facial expressions. *Scandinavian Journal of Psychology*, 39(1), 39–45. <https://doi.org/10.1111/1467-9450.00054>.
- Ekman, P., & Friesen, W. V. (1971). Constants across cultures in the face and emotion. *Journal of Personality and Social Psychology*, 17(2), 124–129. <https://doi.org/10.1037/h0030377>.
- Gepner, B., de Gelder, B., & de Schonen, S. (1996). Face processing in autistics: Evidence for a generalized deficit? *Child Neuropsychology*, 2(2), 123–139. <https://doi.org/10.1080/09297049608401357>.
- Hadjikhani, N., Joseph, R. M., Snyder, J., Chabris, C. F., Clark, J., Steele, S., et al. (2004). Activation of the fusiform gyrus when individuals with autism spectrum disorder view faces. *Neuroimage*, 22(3), 1141–1150. Retrieved from http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=15219586.
- Hadjikhani, N., Asberg Johnels, J., Zurcher, N. R., Lassalle, A., Guillon, Q., Hippolyte, L., et al. (2017). Look me in the eyes: constraining gaze in the eye-region provokes abnormally high subcortical activation in autism. *Scientific Reports*, 7(October 2016), 3163. <https://doi.org/10.1038/s41598-017-03378-5>.
- Hauck, M., Fein, D., Maltby, N., Waterhouse, L., & Feinstein, C. (1998). Memory for faces in children with autism. *Child Neuropsychology*, 4(3), 187–198. <https://doi.org/10.1076/chin.4.3.187.3174>.
- Jones, W., & Klin, A. (2013). Attention to the eyes is present but in decline in 2–6-month-old infants later diagnosed with autism. *Nature*, 504, 427–431. <https://doi.org/10.1038/nature12715>.

- Jones, W., Carr, K., & Klin, A. (2008). Absence of preferential looking to the eyes of approaching adults predicts level of social disability in 2-year-old toddlers with autism spectrum disorder. *Archives of General Psychiatry*, 65(8), 946–954. <https://doi.org/10.1001/archpsyc.65.8.946>.
- Jones, C. R. G., Pickles, A., Falcato, M., Marsden, A. J. S., Happé, F., Scott, S. K., et al. (2011). A multimodal approach to emotion recognition ability in autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 52(3), 275–285. <https://doi.org/10.1111/j.1469-7610.2010.02328.x>.
- Joseph, R. M., Ehrman, K., McNally, R., & Keehn, B. (2008). Affective response to eye contact and face recognition ability in children with ASD. *Journal of the International Neuropsychological Society*, 14(6), 947–955. <https://doi.org/10.1017/S1355617708081344>.
- Kaartinen, M., Puura, K., Mäkelä, T., Rannisto, M., Lemponen, R., Helminen, M., et al. (2012). Autonomic arousal to direct gaze correlates with social impairments among children with ASD. *Journal of Autism and Developmental Disorders*, 42(9), 1917–1927. <https://doi.org/10.1007/s10803-011-1435-2>.
- Kanwisher, N., McDermott, J., & Chun, M. M. (1997). The fusiform face area: A module in human extrastriate cortex specialized for face perception. *Journal of Neuroscience*, 17(11), 4302–4311. Retrieved from <http://www.jneurosci.org/content/17/11/4302>.
- Kawashima, R., Sugiura, M., Kato, T., Nakamura, A., Hatano, K., Ito, K., et al. (1999). The human amygdala plays an important role in gaze monitoring: A PET study. *Brain*, 122(4), 779–783. <https://doi.org/10.1093/brain/122.4.779>.
- Kleinke, C. L. (1986). Gaze and eye contact: A research review. *Psychological Bulletin*, 100(1), 78–100. <http://dx.doi.org/10.1037/0033-2909.100.1.78>.
- Kliemann, D., Dziobek, I., Hatri, A., Baudewig, J., & Heekeren, H. R. (2012). The role of the amygdala in atypical gaze on emotional faces in autism spectrum disorders. *Journal of Neuroscience*, 32(28), 9469–9476. <https://doi.org/10.1523/JNEUROSCI.5294-11.2012>.
- Klin, A., & Jones, W. (2008). Altered face scanning and impaired recognition of biological motion in a 15-month-old infant with autism. *Developmental Science*, 11(1), 40–46. <https://doi.org/10.1111/j.1467-7687.2007.00608.x>.
- Klin, A., Sparrow, S., de Bildt, A., Cicchetti, D. V., Cohen, D. J., & Volkmar, F. R. (1999). A normed study of face recognition in autism and related disorders. *Journal of Autism and Developmental Disorders*, 29(6), 499–508. <https://doi.org/10.1023/A:1022299920240>.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809–816. Retrieved from <http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&listuids=12215080>.
- Kylliäinen, A., & Hietanen, J. K. (2006). Skin conductance responses to another person's gaze in children with autism. *Journal of Autism and Developmental Disorders*, 36(4), 517–525. <https://doi.org/10.1007/s10803-006-0091-4>.
- Lacroix, A., Guidetti, M., Rogé, B., & Reilly, J. (2009). Recognition of emotional and nonemotional facial expressions: A comparison between Williams syndrome and autism. *Research in Developmental Disabilities*, 30(5), 976–985. <https://doi.org/10.1016/j.ridd.2009.02.002>.
- Lehmann, C., Mueller, T., Federspiel, A., Hubl, D., Schroth, G., Huber, O., et al. (2004). Dissociation between overt and unconscious face processing in fusiform face area. *NeuroImage*, 21(1), 75–83. <https://doi.org/10.1016/j.neuroimage.2003.08.038>.
- Louwerse, A., Van Der Geest, J. N., Tulen, J. H. M., Van Der Ende, J., Van Gool, A. R., Verhulst, F. C., & Greaves-Lord, K. (2013). Effects of eye gaze directions of facial images on looking behavior and autonomic responses in adolescents with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 7(9), 1043–1053. <https://doi.org/10.1016/j.rasd.2013.04.013>.
- Loveland, K. A., Bachevalier, J., Pearson, D. A., & Lane, D. M. (2008). Fronto-limbic functioning in children and adolescents with and without autism. *Neuropsychologia*, 46(1), 49–62. <https://doi.org/10.1016/j.neuropsychologia.2007.08.017>.
- Moriuchi, J. M., Klin, A., & Jones, W. (2017). Mechanisms of diminished attention to eyes in Autism. *American Journal of Psychiatry*, 174(1), 26–35. <https://doi.org/10.1176/appi.ajp.2016.15091222>.
- Morris, J. S., deBonis, M., & Dolan, R. J. (2002). Human amygdala responses to fearful eyes. *NeuroImage*, 17(1), 214–222. <https://doi.org/10.1006/nimg.2002.1220>.
- Ozonoff, S., Pennington, B. F., & Rogers, S. J. (1990). Are there emotion perception deficits in young autistic children? *Journal of Child Psychology and Psychiatry*, 31(3), 343–361. <https://doi.org/10.1111/j.1469-7610.1990.tb01574.x>.
- Pelphrey, K. A., Sasson, N. J., Reznick, J. S., Paul, G., Goldman, B. D., & Piven, J. (2002). Visual scanning of faces in autism. *Journal of Autism and Developmental Disorders*, 32(4), 249–261. <https://doi.org/10.1023/A:1016374617369>.
- Pelphrey, K. A., Morris, J. P., & McCarthy, G. (2005). Neural basis of eye gaze processing deficits in autism. *Brain: A Journal of Neurology*, 128(Pt 5), 1038–1048. <https://doi.org/10.1093/brain/awh404>.
- Pierce, K., Muller, R. A., Ambrose, J., Allen, G., & Corchesne, E. (2001). Face processing occurs outside the fusiform “face area” in autism: evidence from functional MRI. *Brain*, 124, 2059–2073.
- Puce, A., Allison, T., Gore, J. C., & McCarthy, G. (1995). Face-sensitive regions in human extrastriate cortex studied by functional MRI. *Journal of Neurophysiology*, 74(3), 1192–1199. Retrieved from <http://jn.physiology.org/content/74/3/1192>.

- Riby, D. M., Doherty-Sneddon, G., & Bruce, V. (2009). The eyes or the mouth? Feature salience and unfamiliar face processing in Williams syndrome and autism. *Quarterly Journal of Experimental Psychology*, 62, 189–203.
- Robison, J. E. (2007). *Look me in the eye: My life with Asperger's*. New York: Crown Publishers.
- Rotshtein, P., Henson, R. N. A., Treves, A., Driver, J., & Dolan, R. J. (2005). Morphing Marilyn into Maggie dissociates physical and identity face representations in the brain. *Nature Neuroscience*, 8(1), 107–113. <https://doi.org/10.1038/nn1370>.
- Rutherford, M. D., Walsh, J. A., & Lee, V. (2015). Brief report: Infants developing with ASD show a unique developmental pattern of facial feature scanning. *Journal of Autism and Developmental Disorders*, 45(8), 2618–2623. <https://doi.org/10.1007/s10803-015-2396-7>.
- Schyns, P. G., Bonnar, L., & Gosselin, F. (2002). Show me the features! Understanding recognition from the use of visual information. *Psychological Science*, 13(5), 402–409. Retrieved from http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=12219805.
- Smith, M. L., Cottrell, G. W., Gosselin, F., & Schyns, P. G. (2005). Transmitting and decoding facial expressions. *Psychological Science*, 16(3), 184–189. <https://doi.org/10.1111/j.0956-7976.2005.00801.x>.
- Speer, L. L., Cook, A. E., McMahon, W. M., & Clark, E. (2007). Face processing in children with autism: effects of stimulus contents and type. *Autism*, 11(3), 265–277. Retrieved from http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=17478579.
- Spezio, M. L., Adolphs, R., Hurley, R. S. E., & Piven, J. (2007). Abnormal use of facial information in high-functioning autism. *Journal of Autism and Developmental Disorders*, 37(5), 929–939. <https://doi.org/10.1007/s10803-006-0232-9>.
- Tanaka, J. W., Wolf, J. M., Klaiman, C., Koenig, K., Cockburn, J., Herlihy, L., et al. (2012). The perception and identification of facial emotions in individuals with autism spectrum disorders using the *Let's Face It!* emotion skills battery. *Journal of Child Psychology and Psychiatry*, 53(12), 1259–1267. <https://doi.org/10.1111/j.1469-7610.2012.02571.x>.
- Tantam, D., Monaghan, L., Nicholson, H., & Stirling, J. (1989). Autistic children's ability to interpret faces: A research note. *Journal of Child Psychology and Psychiatry*, 30(4), 623–630. <https://doi.org/10.1111/j.1469-7610.1989.tb00274.x>.
- Tottenham, N., Hertzog, M. E., Gillespie-Lynch, K., Gilhooly, T., Millner, A. J., & Casey, B. J. (2014). Elevated amygdala response to faces and gaze aversion in autism spectrum disorder. *Social Cognitive and Affective Neuroscience*, 9(1), 106–117. <https://doi.org/10.1093/scan/nst050>.
- Uljarevic, M., & Hamilton, A. (2013). Recognition of emotions in autism: A formal meta-analysis. *Journal of Autism and Developmental Disorders*, 43(7), 1517–1526. <https://doi.org/10.1007/s10803-012-1695-5>.
- van Steensel, F. J. A., Bögels, S. M., & Perrin, S. (2011). Anxiety Disorders in Children and Adolescents with Autistic Spectrum Disorders: A Meta-Analysis. *Clinical Child and Family Psychology Review*, 14(3), 302–317. <https://doi.org/10.1007/s10567-011-0097-0>.
- Vinette, C., Gosselin, F., & Schyns, P. G. (2004). Spatio-temporal dynamics of face recognition in a flash: It's in the eyes. *Cognitive Science*, 28(2), 289–301. <https://doi.org/10.1016/j.cogsci.2004.01.002>.
- Walker-Andrews, A. S. (1998). Emotions and social development: Infants' recognition of emotions in others. *Pediatrics*, 102(E1), 1268–1271. Retrieved from http://pediatrics.aappublications.org/content/102/Supplement_E1/1268.
- Walker-Smith, G. J., Gale, A. G., & Findlay, J. M. (1977). Eye movement strategies involved in face perception. *Perception*, 6(3), 313–326. <https://doi.org/10.1068/p060313n>.
- Wallace, S., Coleman, M., & Bailey, A. (2008). Face and object processing in autism spectrum disorders. *Autism Research*, 1(1), 43–51. <https://doi.org/10.1002/aur.7>.
- Weigelt, S., Koldewyn, K., & Kanwisher, N. (2012). Face identity recognition in autism spectrum disorders: A review of behavioral studies. *Neuroscience and Biobehavioral Reviews*, 36(3), 1060–1084. <https://doi.org/10.1016/j.neubiorev.2011.12.008>.
- Weigelt, S., Koldewyn, K., & Kanwisher, N. (2013). Face recognition deficits in autism are both domain specific and process specific. *PLoS One*, 8(9), e74541. <https://doi.org/10.1371/journal.pone.0074541>.
- Whalen, P. J., Kagan, J., Cook, R. G., Davis, F. C., Kim, H., Polis, S., et al. (2004). Human amygdala responsivity to masked fearful eye whites. *Science*, 306(5704), 2061. <https://doi.org/10.1126/science.1103617>.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229. <https://doi.org/10.1016/j.cpr.2009.01.003>.
- Wolf, J. M., Tanaka, J. W., Klaiman, C., Cockburn, J., Herlihy, L., Brown, C., et al. (2008). Specific impairment of face-processing abilities in children with autism spectrum disorder using the *Let's Face It!* skills battery. *Autism Res*, 1(6), 329–340. <https://doi.org/10.1002/aur.56>.
- Wood, J. J., & Gadow, K. D. (2010). Exploring the nature and function of anxiety in youth with autism spectrum disorders. *Clinical Psychology: Science and Practice*, 17(4), 281–292. <https://doi.org/10.1111/j.1468-2850.2010.01220.x>.

Face Processing Tests

► Facial Recognition Tests

Face Recognition

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Definition

Face recognition is an important dimension of face processing in which specific information about identity is derived. The capacity to recognize individual faces is critical to socialization throughout development and adulthood, providing important social cues that guide interactions. While no scientific consensus exists on whether autistic populations show behavioral impairment in the ability to recognize faces, substantial evidence indicates that individuals with autism may use atypical visual and neural mechanisms to determine facial identity. By disrupting the extraction of important social information from the face, these abnormalities in processing facial recognition may contribute to the social deficits that characterize autism spectrum disorders (ASD).

Historical Background

Faces are highly relevant visual stimuli, conveying information critical to social understanding and effective communication. The ability to accurately recognize others' faces, which allows for quick identification of others and retrieval of information associated with them, is particularly critical to social function. Developmental studies indicate that face recognition facilitates engagement with others from infancy; from the first days of life, infants demonstrate the capacity to recognize their mothers' faces among others (Pascalis et al. 1995). Further studies of face recognition ability in children have revealed that the basic capacity to differentiate faces matures over the course of development, following a

developmental trajectory distinct from general pattern recognition or memory (Carey et al. 1980).

Neuropathological findings have emphasized the importance of face recognition over the lifespan, suggesting that circumscribed regions of the brain are specialized for differentiating faces. Patients with prosopagnosia, a disorder of face perception, exhibit normal recognition of common objects in spite of severe impairment in recognizing faces (Whiteley and Warrington 1977), while individuals demonstrating severe impairment in recognizing common objects retain normal face recognition (Moscovitch and Winocur 1997). Together, these findings suggest a double dissociation between face and object recognition, i.e., that face and object recognition rely upon distinct neural networks in the brain. Subsequent neuroimaging studies have indicated that the region of the temporal lobe known as the fusiform gyrus is activated preferentially during face recognition tasks, suggesting the specialization of this brain region for face processing (e.g., Kanwisher et al. 1997). However, recent studies reveal that nonface objects with which an individual has expertise and novel face-like objects also animate this brain region, calling this interpretation into question (Grelotti et al. 2005).

In addition, early research into the effects of inversion upon recognition accuracy has indicated that face and object recognition rely upon distinct processing strategies. Yin (1969) discovered that inversion of faces impairs recognition in typical adults to a greater degree than does inversion of common objects; this robust phenomenon, known as the "inversion effect," suggests that adults use a different visual strategy to identify faces versus objects. Subsequent research indicates that, whereas object recognition depends upon discrimination of individual features, accurate face recognition relies more heavily upon the configuration of the face, which is disrupted during inversion. This processing strategy, however, may not be specific to faces but more generally applied to stimuli that an individual is expert at recognizing; individuals with expertise in nonface objects demonstrate an inversion effect for objects in their domain of expertise comparable to that for faces (Diamond and Carey 1986).

The first experimental investigations of face recognition in autism were carried out by Langdell (1978) to assess whether disruption of the typical visual mechanisms underlying face recognition might contribute to the social impairment observed in children with autism; this research indicated that children and young adults with autism are proficient in recognizing faces but employ atypical visual strategies in order to do so. While children with autism did not exhibit behavioral deficits in recognizing photographs of their peers, they demonstrated a greater reliance on features located in the lower parts of the face (i.e., the mouth region) than did typical peers. In contrast, typically developing children relied more heavily on the upper part of the faces (i.e., the eye region) to recognize peers, replicating previous findings. Langdell (1978) also found that older children with autism failed to show an inversion effect for faces, while typically developing children did. Similarly, Hobson, Ouston, and Lee (Hobson et al. 1988) found that adolescents and young adults with autism were significantly better than typical peers at recognizing inverted faces, which they were able to identify as well as upright faces, suggesting that individuals with autism employ different processing strategies to determine facial identity than do typically developing individuals.

Current Knowledge

Initial studies of face recognition ability in autism indicated that children and adolescents with autism demonstrate behavioral proficiency in recognizing faces but atypical patterns of visual processing. Current research continues to explore the question of whether individuals with autism are impaired in face recognition. However, the focus of this line of investigation has largely shifted from reporting behavioral performance to clarifying the visual mechanisms and neural networks that underlie the distinct processing strategies for face recognition in ASD.

To date, no consensus exists as to whether individuals with autism demonstrate significant deficits in face recognition relative to typically

developing peers matched on cognitive and verbal ability. While several recent studies report behavioral impairment in face recognition in children with ASD that is independent of overall functioning, many have failed to report such impairment (*see* Klin et al. 1999 *for a review*). Although most research to date indicates that recognition deficits in autism are specific to faces, some studies have reported more general impairment in processing both face and nonface stimuli (e.g., Davies et al. 1994). Klin et al. (1999) suggest that these inconsistencies in the literature are attributable to differences in age and functioning in the subject populations, control group criteria, and experimental tasks employed across studies of face recognition in ASD; in addition, Klin et al. (1999) hypothesize that abnormalities in the way facial identity is determined in autism may not always translate into impaired performance because children with autism may develop compensatory strategies for face processing to overcome difficulties in the critical domain of face recognition.

While controversy exists as to whether individuals with autism demonstrate behavioral impairment in the specific domain of face recognition, an emerging body of research supports the hypothesis that atypical visual mechanisms underlie face recognition processing in ASD. Recent studies have provided direct evidence that the facial inversion effect observed in typical adults reflects the use of a configural visual strategy (i.e., one that relies upon the spatial relationship between individual features) for face recognition that is distinct from the feature-based strategies employed in object recognition (Freire et al. 2000). Given the lack of inversion effect in autism, this suggests that facial recognition in autism relies less upon the configural strategies and more upon the feature-based strategies characteristic of object recognition in typical development. However, subsequent work has indicated that high-functioning children with ASD do employ holistic strategies for face recognition (i.e., evaluating the face as a whole rather than part by part) in instances where accurate face recognition depends upon the mouth region of the face (Joseph and Tanaka 2003); this suggests

that atypical patterns of face recognition in autism cannot be fully explained by impairment in configural processing of faces.

Current research confirms that individuals with autism rely upon different regions of the face to determine facial identity. While typically developing children are more proficient in recognizing faces based upon the eyes compared to the mouth, high-functioning children with ASD demonstrate the reverse pattern (Joseph and Tanaka 2003). Eye-tracking studies have provided further evidence that, while observing and encoding faces, individuals with autism may preferentially attend to different face regions than do typically developing peers. Prior research has indicated that adults with high-functioning autism (HFA) spend more time scanning the external features of the face than core internal features (Pelphrey et al. 2002). Similarly, adults with HFA have been found to spend more time viewing mouths, bodies, and objects than eyes while viewing video clips of dynamic social interactions (Klin et al. 2002). In comparison, typically developing subjects spend more time scanning the eye region of faces, indicating that abnormal patterns of viewing faces in autism, characterized by reduced attention to the socially informative eye region of faces and enhanced attention to mouths, may underlie atypical face recognition in ASD (Klin et al. 2002).

Neuroimaging techniques have recently been applied to shed light on the neural mechanisms that support the distinct processing of facial identity in autism. While brain imaging studies of typically developing individuals have consistently revealed activation in the fusiform face area (FFA) to viewing faces (e.g., Kanwisher et al. 1997), individuals with autism demonstrate atypical patterns of neural activation during face processing (Pierce et al. 2001; Schultz et al. 2000). Work in this field has revealed evidence that face processing can occur outside the FFA, at individual-specific sites, in ASD (Pierce et al. 2001). This difference in where face processing occurs in the brain may be associated with distinct strategies for face recognition in this population. Furthermore, individuals with autism and Asperger disorder have shown weak activation

in the FFA and heightened activation of the inferior temporal gyrus during a face discrimination task, the same pattern of brain activity observed when typically developing individuals performed a nonface object discrimination task (Schultz et al. 2000). This supports the idea that individuals with ASD may use feature-based processing strategies to discriminate among both faces and objects, showing a lack of typical specialization of face processing strategies in this population.

Further studies have revealed key differences in how the brain response to faces is mediated in real time in typical and atypical development, suggesting that children with autism show impaired neural processing of faces at multiple stages of face recognition. Two distinct temporal markers of brain activity (the N400 and Pc) have been found to differentiate between familiar and unfamiliar faces and familiar and unfamiliar objects in typically developing 3- and 4-year-olds; in contrast, same-age peers with autism show differentiation between familiar and unfamiliar objects but not between familiar and unfamiliar faces at these two markers, suggesting that impairment in processing facial recognition emerges early in life (Dawson et al. 2002). Disruption of typical mechanisms for discriminating faces at this early age may derail subsequent brain and behavioral development by affecting how children with autism attend to faces in the world around them and how effective they are at gathering critical social information from faces (Dawson et al. 2002). Additional research has revealed that individuals with autism also show significant delay at a key temporal marker of face processing, the N170, compared to typically developing individuals, reflecting slowed neural speed of face processing in autism only 170 ms after viewing a face (McPartland et al. 2004). Slowed speed of processing has been correlated with poorer performance on tests of facial recognition in autism; this suggests that greater delays in neural processing of faces may contribute to difficulties in face discrimination. While the N170 is delayed in typical development to inverted versus upright faces, this inversion effect is not observed in ASD, providing further

support for the idea that individuals with autism may rely upon configural rather than typical holistic strategies for processing faces (McPartland et al. 2004).

Future Directions

While an emerging body of eye-tracking and neuroscientific research has provided evidence that individuals with autism employ atypical visual and neural mechanisms for discriminating faces, the exact nature of the relationship between these divergent mechanisms and social impairment in autism remains unknown. Atypical mechanisms for recognizing faces may serve as both a cause and an effect of social impairment in autism. For example, reduced attention to faces early in development may disrupt typical specialization of visual strategies and neural circuitry for discriminating faces; the resulting abnormalities in processing facial recognition may further deprive individuals of critical social inputs necessary for the development of typical patterns of social behavior (Dawson et al. 2002). Additional work is needed to clarify the connection between divergent patterns of visual and neural processing of facial identity and the social difficulties that characterize autism in order to design effective interventions.

See Also

- Face Perception
- Fusiform Gyrus (FG)

References and Reading

- Carey, S., Diamond, R., & Woods, B. (1980). Development of face recognition: A maturational component? *Developmental Psychology*, 16(4), 257–269. <https://doi.org/10.1037/0012-1649.16.4.257>.
- Davies, S., Bishop, D., Manstead, A. S. R., & Tantam, D. (1994). Face perception in children with autism and Asperger's syndrome. *Journal of Child Psychology and Psychiatry*, 35(6), 1033–1057. <https://doi.org/10.1111/j.1469-7610.1994.tb01808.x>.
- Dawson, G., Carver, L., Meltzoff, A. N., Panagiotides, H., McPartland, J., & Webb, S. J. (2002). Neural correlates of face and object recognition in young children with autism spectrum disorder, developmental delay, and typical development. *Child Development*, 73(3), 700–717. <https://doi.org/10.1111/1467-8624.00433>.
- Diamond, R., & Carey, S. (1986). Why faces are and are not special: An effect of expertise. *Journal of Experimental Psychology: General*, 115(2), 107.
- Freire, A., Lee, K., & Symons, L. A. (2000). The face-inversion effect as a deficit in the encoding of configural information: Direct evidence. *Perception*, 29(2), 159–170. <https://doi.org/10.1068/p3012>.
- Grelotti, D. J., Klin, A. J., Gauthier, I., Skudlarski, P., Cohen, D. J., Gore, J. C., et al. (2005). fMRI activation of the fusiform gyrus and amygdala to cartoon characters but not to faces in a boy with autism. *Neuropsychologia*, 43(3), 373–385. <https://doi.org/10.1016/j.neuropsychologia.2004.06.015>.
- Hobson, R. P., Ouston, J., & Lee, A. (1988). What's in a face? The case of autism. *British Journal of Psychology*, 79(4), 441–453. <https://doi.org/10.1111/j.2044-8295.1988.tb02745.x>.
- Joseph, R. M., & Tanaka, J. (2003). Holistic and part-based face recognition in children with autism. *Journal of Child Psychology and Psychiatry*, 44(4), 529–542. <https://doi.org/10.1111/1469-7610.00142>.
- Kanwisher, N., McDermott, J., & Chun, M. M. (1997). The fusiform face area: A module in human extrastriate cortex specialized for face perception. *Journal of Neuroscience*, 17(11), 4302–4311.
- Klin, A., Sparrow, S. S., de Bildt, A., Cicchetti, D. V., Cohen, D. J., & Volkmar, F. R. (1999). A normed study of face recognition in autism and related disorders. *Journal of Autism and Developmental Disorders*, 29(6), 499–508. <https://doi.org/10.1023/a:1022299920240>.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809–816. <https://doi.org/10.1001/archpsyc.59.9.809>.
- Langdell, T. (1978). Recognition of faces: An approach to the study of autism. *Journal of Child Psychology and Psychiatry*, 19(3), 255–268. <https://doi.org/10.1111/j.1469-7610.1978.tb00468.x>.
- McPartland, J., Dawson, G., Webb, S. J., Panagiotides, H., & Carver, L. J. (2004). Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 45(7), 1235–1245. <https://doi.org/10.1111/j.1469-7610.2004.00318.x>.
- Moscovitch, M., & Winocur, G. (1997). What is special about face recognition? Nineteen experiments on a person with visual object. *Journal of Cognitive Neuroscience*, 9(5), 555.
- Pascalis, O., de Schonen, S., Morton, J., Deruelle, C., & Fabre-Grenet, M. (1995). Mother's face recognition by

- neonates: A replication and an extension. *Infant Behavior & Development*, 18(1), 79–85. [https://doi.org/10.1016/0163-6383\(95\)90009-8](https://doi.org/10.1016/0163-6383(95)90009-8).
- Pelphrey, K. A., Sasson, N. J., Reznick, J. S., Paul, G., Goldman, B. D., & Piven, J. (2002). Visual scanning of faces in autism. *Journal of Autism and Developmental Disorders*, 32(4), 249–261. <https://doi.org/10.1023/a:1016374617369>.
- Pierce, K., Muller, R. A., Ambrose, J., Allen, G., & Courchesne, E. (2001). Face processing occurs outside the fusiform ‘face area’ in autism: Evidence from functional MRI. *Brain*, 124(10), 2059–2073. <https://doi.org/10.1093/brain/124.10.2059>.
- Schultz, R. T., Gauthier, I., Klin, A., Fulbright, R. K., Anderson, A. W., Volkmar, F., et al. (2000). Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and Asperger syndrome. *Archives of General Psychiatry*, 57(4), 331–340. <https://doi.org/10.1001/archpsyc.57.4.331>.
- Whiteley, A. M., & Warrington, E. K. (1977). Prosopagnosia: A clinical, psychological, and anatomical study of three patients. *Journal of Neurology, Neurosurgery & Psychiatry*, 40(4), 395–403. <https://doi.org/10.1136/jnnp.40.4.395>.
- Yin, R. K. (1969). Looking at upside-down faces. *Journal of Experimental Psychology*, 81(1), 141–145. <https://doi.org/10.1037/h0027474>.

Face Tests

► Facial Recognition Tests

Face Validity

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Definition

Face validity refers to the extent to which a test appears to measure what it is intended to measure. A test in which most people would agree that the test items appear to measure what the test is intended to measure would have strong face validity. For example, a mathematical test consisting of

problems in which the test taker has to add and subtract numbers may be considered to have strong face validity. The test items appear, at face value, to measure what one is seeking to measure.

See Also

► Validity

References and Reading

- Cicchetti, D. V. (2008). From Bayes to the just noticeable difference to effect sizes: A note to understanding the clinical and statistical significance of oenologic research findings. *Journal of Wine Economics*, 3, 185–193.
- Cicchetti, D. V. (2011). On the reliability and accuracy of the evaluative method for identifying evidence-based practices in autism, Chapter 3. In B. Reichow, P. Doehring, D. V. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-based practices and treatments for children with autism*. New York: Springer.
- Cicchetti, D. V., & Rourke, B. P. (2004). *Methodological and biostatistical foundations of clinical neuropsychology and medical and health disciplines* (2nd ed.). London: Psychology Press/Taylor & Francis.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale: Lawrence Erlbaum Associates.
- Tsatsanis, K. D., Saulnier, C., Sparrow, S. S., & Cicchetti, D. (2011). The role of adaptive behavior in evidence-based practices for ASD: Translating intervention into functional success, Chapter 11. In B. Reichow, P. Doehring, D. V. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-based practices and treatments for children with autism*. New York: Springer.

Facial Imitation

► Oral-Facial Imitation

Facial Inversion Effect

► Upright/Inverted Figures

Facial Recognition Tests

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Abbreviations

ADT	Age differentiation test
ASD	Autism spectrum disorder
BTFR	Benton test of facial recognition
CFMT	Cambridge face memory test
CFMT-K	Cambridge face memory test-kids
CFPT	Cambridge face perception test
CMS	Children's memory scales
CNB	Penn computerized neurocognitive battery
FME	Facial memory test
K-ABC-II, FR	Kaufman Assessment Battery for Children-II, Face Recognition Subtest
LFI	Let's Face It!
RMT	Warrington recognition memory test
WMS	Wechsler memory scales

Synonyms

[Face processing tests](#); [Face tests](#)

Description

A variety of tests have been developed to measure the ability to recognize faces. In order to recognize a face, one must have the basic perceptual ability to distinguish that face from others, often over time and from multiple vantage points. Tests that measure this ability provide information about the accuracy with which the face is represented, recognized, and distinguished from others. A range of skills are critical for face recognition including the ability to analyze the face and its features, understand the relationship of these features within the overall configuration of the face,

perceive the identity of the face across changes in viewpoint and expression, and explicitly recognize the faces of familiar people.

Recognition of faces is critical throughout social development, because faces are used to identify others in social situations more than any other physical characteristic. The face provides a unique way to identify individuals and comes to serve as a representation of that person and their personal characteristics. Infants distinguish faces of familiar individuals shortly after they are born. Without the early ability to recognize and remember faces or to make use of the nonverbal information they contain, many other subsequent learning processes may be derailed, including the development of coordinated eye contact, joint referencing, and emotional reciprocity.

Impaired face recognition in individuals with autism spectrum disorder (ASD) has been demonstrated with a wide range of experimental tests. On average, groups with ASD are less accurate in their ability to recognize the faces of others. This is true for discrimination of facial identities, recognition of familiar faces, and immediate recognition of newly learned faces. Impairment in face recognition appears to be somewhat specific because individuals with ASD are typically unimpaired in their recognition of objects. On an individual level, there appears to be variability in the ability of people with ASD to recognize faces. There is evidence of an association between the degree of severity of face processing impairments and the severity of more general social symptoms. For this reason, tests of facial recognition are important in classifying the degree of difficulty individuals experience in this perceptual domain in clinical and research settings. Standardized face recognition tests are also useful when describing group characteristics of a research sample, because they provide a common means for comparison across studies and for interpreting experimental measures of face processing.

Historical Background

Early reports of individuals who experienced difficulty with recognizing faces were made in the

late 1800s, though these difficulties were usually associated with other visual impairments. Bodamer (1947) coined the term prosopagnosia (i.e., “loss of knowledge of faces”) to describe individuals who had greater impairments with faces than objects. In order to evaluate patients with frank impairments in face recognition, tests of facial recognition were developed. For example, in the 1960s, Benton and Van Allen developed a face-matching test and De Renzi and colleagues developed a same-different face test. The former became the Benton Facial Recognition Test (Benton et al. 1983). Warrington (1984) developed a test that involved learning a set of new faces followed by a series of target-distracter face pairs to test recognition memory for faces, although this measure has not been widely used with autism and nonface information is contained in the pictures that may aid in the discrimination of targets. In more recent years, similar face recognition tests have been included with standardized batteries of memory and neuropsychological functioning. The Wechsler Memory Scales-III (Wechsler 1997) included the Faces subtest, which is appropriate for adults. A very similar task is included in the Children’s Memory Scales (Cohen 1997), and is appropriate for children. Also appropriate for younger children are the Face Recognition subtest of the Kaufman Assessment Battery for Children (K-ABC-2; Kaufman and Kaufman 2004) and the Memory for Faces subtest of the NEPSY-II (Korkman et al. 2007). In recent years, a new set of facial recognition tests has been developed to increase the sensitivity, validity, and reliability of available measures including the Cambridge, Caledonian, Penn, and Philadelphia facial tests. Most of these tests, along with experimental tasks, have been used to assess face recognition in ASD.

The face processing of individuals with ASD has been studied extensively since Langdell (1978) first offered evidence of atypical strategies in the way young children with autism use information to identify familiar faces. Langdell proposed that face processing deficits may explain the social symptoms associated with autism including impaired eye contact and reduced social reciprocity. Throughout the 1980s and 1990s, a

series of studies followed that revealed impaired or unusual patterns of face recognition in children, adolescents, and adults with autism. In many cases, these studies did not use standardized measures of face recognition, and instead used tasks developed for the particular study being conducted.

Most investigations of face recognition have also been limited by small sample size with a few exceptions, thus limiting the ability to generalize conclusions about the integrity of the face recognition system and determine the expected range of function for individuals with ASD. In an effort to address this problem, the Face Recognition subtest of the K-ABC was administered to over 100 children with ICD-10 defined autistic disorder, pervasive developmental disorder-not otherwise specified, and a comparison group without ASD (Klin et al. 1999). Children with autistic disorder (i.e., the most severe cases) recognized fewer faces than the other two groups, even when matched on verbal and nonverbal mental age. Based on this investigation, it was concluded that children with autism exhibited impairments in face recognition that could not be accounted for by more generalized cognitive impairment. Further, this study suggests face recognition difficulty corresponds with autism severity.

Most studies have been also limited by use of a single measure of face processing or recognition. As a result, it has been difficult to compare results across studies, and the ability to determine the nature and relative contribution of various processing deficits is limited. Additionally, while standardized measures provide a means of comparison across studies, they are limited in their sensitivity to the specific challenges with face processing exhibited by some individuals with ASD (i.e., a focus on features within the face rather than the face as a whole, decreased visual attention to the eye region of the face, and reduced disruption in performance when faces are presented upside down). To address these limitations, Tanaka and Schultz developed the *Let’s Face It!* (LFI) Skills Battery of computerized face recognition tasks aimed at children that includes measures of short-term memory, feature processing and configural perception, and holistic processing

of faces. Many LFI tasks require children to recognize and match individual faces across changes in orientation, different emotional expressions, and with certain regions of the face obscured. LFI also includes tasks that measure processing and short-term memory for objects. Compared with a group without ASD, the LFI Skills Battery is sensitive to the specific ASD-related impairments in face memory and matching, a processing advantage for the mouth region of the face, and unimpaired object processing.

Psychometric Data

All tests presented are individually administered, use still photographs of unfamiliar faces, and are thought to measure facial recognition.

The *Benton Test of Facial Recognition* (BTFR; Benton et al. 1983) is a measure of the ability to perceive and match unfamiliar faces without a memory component. Normative samples span a wide range of ages (6–74 years). Black and white photos are presented, with the target at the top of the page and six test faces at the bottom of the page. Photos vary based on orientation and lighting condition across three conditions: (1) an identical photograph, (2) the same face with different lighting, and (3) three-quarter views of the same face. Initially, there is only one target face, but in the latter portion of the task, three of the six faces have the same identity (i.e., there are three correct responses). Administration time typically ranges from 5–15 min, although the items are untimed. The BTFR has a short and long form (i.e., a subset of the first 13 items versus the full set of items). Scores are obtained by a simple summation of correct responses. Normative data is available for 286 adults and 229 children (Benton et al. 1994).

The psychometric properties of the long form are adequate at best, and the short form has poor psychometric properties. In particular, evidence challenging the construct validity comes from work by Duchaine and colleagues who found adults with typical development were able to score in the normal range when administered a version of the BTFR that presented only the

eyebrows and hairline of faces. These authors concluded that the simultaneous presentation of targets and test faces permits the use of feature-based strategies. The same group showed individuals with developmental prosopagnosia could score in the normal range – a challenge to the BTFR's predictive validity.

The *Cambridge Face Memory Test* (CFMT; Duchaine and Nakayama 2006; Bowles et al. 2009) is a standardized assessment of face processing, recognition, and memory of unfamiliar faces with good psychometric properties and sensitivity to face impairments. The CFMT first establishes familiarity with six study images of male faces from three angles (left, frontal, and right) for 3 s each, then tests recognition from multiple viewpoints, different poses and lighting conditions, and with visual noise from a set of forced-choice groupings. Administration lasts between 10–15 min. There are four phases of the task (Practice with Cartoons, Introduction with the Same Images, Novel Images, and Novel Images with Noise). During the Same Images phase, three test items that are identical to the study images are presented for each face. In the Novel Images phase, a brief review of the study images is followed by presentation of 30 test items that vary by lighting or pose. Finally, in the Novel Images with Noise phase, study images are again briefly reviewed followed by 24 test items with different levels of Gaussian noise added to each image. The CFMT also assesses the effect of face inversion, which is thought to disrupt specialized face processing mechanisms. The CFMT is appropriate for adults, and was developed and initially tested with 50 college students and 9 older adults. A long form with 30 additional difficult trials was developed to identify individuals for whom face recognition and memory is a particular strength (Russell et al. 2009). Initial data support the validity of the CFMT, and normative data and reliability measurements are available from 240 healthy adults (Bowles et al. 2009) including measures of acceptable reliability between blocks and good internal consistency based on data from 124 individuals. Of note, normative data demonstrate that norms must be derived from participants with similar race and ethnicity as the stimuli.

A *Cambridge Face Memory Test-Kids* (CFMT-K) has also been developed for children (Croydon et al. 2014). The CFMT-K stimuli are images of children, and it was validated with 401 children between 5 and 12 years of age to demonstrate age-related developmental sensitivity for upright and inverted face recognition and memory.

The *Cambridge Face Perception Test* (CFPT; Duchaine et al. 2007; Russell et al. 2012) does not require face memory. Rather it requires participants to rank order a set of six faces according to their similarity to a target face. There are eight upright trials and eight inverted trials, with 1 min allowed per set. The target face is presented with a three-fourth view and the test faces are morphed to include 88, 76, 64, 52, 40, and 28% of the original target face. Scoring accounts for the magnitude of the error (i.e., distance from correct sort position). Norms are available, but there is significant variability in performance of the initial sample. Adequate internal consistency was reported for a different sample of 126 adults, as well as a high level of correlation with the CFMT suggesting they assess largely overlapping but partially separable abilities (Bowles et al. 2009). Cambridge face measures (CFMT, CFMT-K, CFPT) are available for research.

The *Caledonian face test* (Logan et al. 2016) requires participants to determine the odd-one-out from a group of four faces. That is, they must select the one face that is different from the three distracters. The task is untimed and presented on a computer. Completion time is 3–4 min. Stimuli are synthetic faces with predictable differences between targets and distracters. The face with the mean value across dimensions occurs in every trial and is the target for half of the trials, whereas the other face is selected from a pool of 80 faces, which allows the task to be readministered without learning effects. The difference between faces on each trial has a varying magnitude that is individually controlled for each participant using a QUEST algorithm. The psychometrics of the Caledonian test were determined in a sample of 80 young adults. An inversion effect was present and face discrimination thresholds had comparable sensitivity to

other lab-based measures, both confirmations of the validity of the more efficient Caledonian test. The measure has good sensitivity including detection of individual differences in performance among nonclinical adults. It was also more sensitive to the impairments of an individual with developmental prosopagnosia, with sensitivity almost threefold greater than two other measures of face processing. Finally test-retest on the same day had good reliability. The Caledonian test is available for research.

The *Children's Memory Scales (CMS) Faces subtest* (Cohen 1997) measures face recognition and memory with unfamiliar faces. The test is appropriate for children aged 5–16 years and can be administered in 10 min plus a 25- to 35-min delay. A series of color photographs of unfamiliar faces are presented one at a time for 2 s (either 12 or 16 faces depending on age). Immediately a series of test faces are presented one at a time and the child is asked to respond to each by indicating whether the face was viewed in the learning phase (or not). After a delay, a second series of test images is presented and children are asked to respond in the same manner. Correct responses for the Immediate and Delayed Faces subtests are totaled to attain two scaled scores. Normative data were obtained from a representative sample of children in the United States during the 1990s. Stability of the assessment across administrations is adequate.

The *Glasgow Face Matching Test* (Burton et al. 2010) requires matching in the same view but stimuli are taken with different cameras, which introduce variability in the images, and is particularly relevant in forensic or security identification tasks. The test contains 168 pairs of adult faces with neutral expressions and takes about 15 min to complete. Half the pairs are same-face trials. The test was normed on 300 adults aged 18–80. Split-half reliability is high and performance related to face processing ability on other common manipulations such as recognition memory for faces supports the validity of the test as a measure of face processing ability. A short version with 40 of the most difficult face pairs was also examined using 194 new adult volunteers aged 18–46 and takes about 3–4 min to complete. Short and long versions are available for research.

The *Kaufman Assessment Battery for Children-II (K-ABC-II) Face Recognition Subtest* (Kaufman and Kaufman 2004) is standardized for ages 3–18; however, psychometric data for the Face Recognition test is available only for ages 3–5 years. The test involves viewing and attending one or two photographs of faces with a brief exposure. Then, children are asked to select the correct face or faces from a group. The task requires children to recognize the target face(s) across changes in pose. In the revision of the K-ABC, new items were added to the subtest and distracting features in the background of photos were removed. The K-ABC-II Face Recognition subtest is standardized on a national sample of children and has excellent psychometric properties.

The *Let's Face It! Skills Battery* (Wolfe et al. 2008) is a battery of tasks designed to measure specific aspects of face recognition and memory that are particularly relevant for children with ASD. Administration of the battery occurred over 2 days in the initial validation and requires more time for completion than other individual tests described in this section. The battery contains five facial identity tests, three emotion processing tests, and two object processing tests. Face recognition subtests include: Matching Identity Across Expression, Matching Identity Across Masked Features, Featural and Configural Face Dimensions, Parts/Whole Identity, and Immediate Memory for Faces. The LFI Skills Battery is not standardized, but has been administered to a relatively large sample of children, adolescents, and young adults with ASD (85 total, with >60 responding to any given subtest) and 130 with typical development. The age range of participants with ASD was 5.1–20.1 years (mean age = 11.58) and cognitive functioning was high (mean full scale IQ of 99.74). Effect sizes for individual subtests were moderate to large. The largest effect sizes were found for the Matching Identity across Expression subtest (effect size = 1.00) and for the Immediate Memory for Faces subtest (effect size = 1.00). Moderate effect sizes were also reported for task conditions in which the eye region was tested (Parts/whole identity, Face dimensions).

The *NEPSY-II Memory for Faces Subtest* (Korkman et al. 2007) is thought to measure encoding, discrimination, and recognition of unfamiliar faces. Children are first presented with a series of photos of faces, and the child is asked to identify the gender of each face as a way to increase attention to the stimuli. Immediately after presentation, test trials require children to select the target face from a group of three faces (a target and two distracters). Fifteen to twenty minutes later, children are asked to select the same set of targets from a new set of three faces. The longer delay assesses long-term facial memory. The subtest yields three scores: Memory for Faces Total Score (immediate recognition), Memory for Faces Delayed Total Score (delayed memory for faces), and Memory for Faces vs. Memory for Faces Delayed Contrast Scaled Score (contrast between the first two). The task is standardized for ages 5–16 years. Normative data were collected between 2004 and 2006 from across the United States, and included 1200 children aged 3–16 years. The revision process also included clinical studies of children with ASD. The validity of the subtest is supported by moderate correlations between the Memory and Delayed Memory scores, between the NEPSY-II and NEPSY, between the NEPSY-II Memory for Faces and CMS Faces subtests, and by diminished performance by children with ASD. Concurrent validity data suggest that processing speed and visual discrimination are also involved, and inattention, impulsivity, and repetitive behaviors may influence performance. Internal consistency ranges from adequate to high and has adequate stability over time. Of interest, the NEPSY-II battery also includes other measures of social function including Affect Recognition and Theory of Mind subtests.

The *Penn Computerized Neurocognitive Battery (CNB) Facial Memory Test (FME) and Age Differentiation Test (ADT)* (Moore et al. 2015) are brief web-based computerized measures of functioning validated with functional neuroimaging. Scores for accuracy as well as reaction time for correct responses are available. Good reliability is reported for FME and ADT response speed, adequate reliability for ADT accuracy, and

questionable reliability for FME accuracy. The FME begins with 20 images of faces presented for 1 sec that participants are told they will be asked to remember. Then, a series of 40 test faces is presented including 20 targets and 20 distracters matched on age, ethnicity, and gender. Participants must decide if the face is one that has been viewed previously using a four-point scale (definitely not, probably not, probably yes, or definitely yes). The test takes 4 min to administer. The FME is administered immediately and again after a delay. The ADT takes 3 min to administer and simultaneously presents neutral face pairs; participants must determine which face is older. Stimuli were generated from faces morphed in age in order to control the level of difficulty. The psychometric properties and validity of the CNB have been evaluated with thousands of adults and children in the USA and the Netherlands (Gur et al. 2010; Moore et al. 2015; Swagerman et al. 2016). It is available online for research.

The *Philadelphia Face Perception Battery* (Thomas et al. 2008) measures matching based on similarity, evaluation of attractiveness, gender classification, and identification of older faces. Stimuli were created in software allowing modification of 114 parameters and refined via pilot studies. To develop the tests, 124 adults participated. A target face is presented for matching with two forced choice options in the Similarity test (more/less similar faces) and the Gender test (male/female labels). Two faces are presented with a question for the Beauty test (who is more attractive?) and Age test (who is older?). Test-retest was adequate only for the Similarity subtest based on the performance of 19 participants. A patient with prosopagnosia performed two standard deviations below the healthy adult sample. This test is available for research.

The *Warrington Recognition Memory Test* (Warrington 1984) tests memory of faces after a short delay. It begins by asking participants to view a set of faces and words and judge whether each is pleasant or unpleasant. Then, after a delay, participants must decide which face or word in a target-distracter pair they previously saw. Originally standardized with 300 adults, additional

psychometric testing was conducted with 72 patients with traumatic brain injuries. Internal consistency was adequate; however, as with other face memory tests, the Recognition Memory Test has been criticized because poor performance could be due to general memory impairment.

The *Wechsler Memory Scales-III (WMS-III) Faces subtest* (Wechsler 1997) is a measure of face memory and recognition for use with individuals aged 16–89 years. As with the CMS, this subtest may be administered in 10 min plus the 25- to 35-min delay. Twenty-four photographs of unfamiliar faces are presented one at a time for 2 sec, followed immediately by a test phase of 48 faces. Adults are asked to respond to each item by indicating whether the face was viewed in the learning phase. After a delay, a second test series is presented in the same way. Correct responses for the Immediate and Delayed faces are totaled to arrive at two scaled scores (immediate and delayed). The scales were normed on a representative sample of over 1000 individuals throughout the United States during the 1990s, with good reliability. The Faces subtest was not included in the most recent edition (WMS-IV) of the battery.

Clinical Uses

Measurement of face recognition in a clinical setting may provide useful information about social function as well as memory for social versus nonsocial information. Subtests within larger batteries provide a means for such comparisons and offer standardized scores for comparison with the population. For preschoolers, the K-ABC-II Face Recognition subtest provides a useful measure. Older children may be assessed with tests such as the NEPSY-II Memory for Faces subtest or Children's Memory Scales Faces subtest. The Wechsler Memory Scales-III Faces subtest provides a similar measure for adults. These tests provide a more global assessment of face recognition, but may be influenced by motivation, attention, and repetitive or impulsive responding. As well, they do not provide insight into why face recognition is disrupted. An emerging research

literature with tests such as the *Let's Face It! Skills Battery* and *Cambridge Face Memory Test*, which provide more focused assessment of face recognition and perception, and current psychometric investigations of biological measures of facial recognition, suggests that such tests may ultimately be available.

See Also

- [Face Perception](#)
- [Face Recognition](#)
- [Memory Assessment](#)

References and Reading

- Benton, A. L., Sivan, A. B., Varney, N. R., & Spreen, O. (1983). *Facial recognition: Stimulus and multiple choice pictures*. New York: Oxford University Press.
- Benton, A. L., Sivan, A. B., Hamsher, K. de. S., Varney, N. R., & Spreen, O. (1994). Facial recognition. In A. L. Benton (Ed.), *Contributions to neuropsychological assessment: A clinical manual* (2nd ed., pp. 517–552). New York: Oxford University Press.
- Bodamer, J. (1947). Die Prosopagnosie. *Archiv fur Psychiatrie und Zeitschrift fur Neurologie*, 179, 6–54.
- Bowles, D. C., McKone, E., Dawel, A., Duchaine, B., Palermo, R., Schmalzl, L., et al. (2009). Diagnosing prosopagnosia: Effects of ageing, sex, and participant–stimulus ethnic match on the Cambridge face memory test and Cambridge face perception test. *Cognitive Neuropsychology*, 26, 423–455.
- Burton, A. M., White, D., & McNeill, A. (2010). The Glasgow face matching test. *Behavior Research Methods*, 42, 286–291.
- Cohen, M. J. (1997). *Children's memory scale*. San Antonio: The Psychological Corporation.
- Croydon, A., Pimperton, H., Ewing, L., Duchaine, B. C., & Pellicano, E. (2014). The Cambridge face memory test for children (CFMT-C): A new tool for measuring face recognition skills in childhood. *Neuropsychologia*, 62, 60–67.
- Duchaine, B., Germine, L., & Nakayama, K. (2007). Family resemblance: Ten family members with prosopagnosia and within-class object agnosia. *Cognitive Neuropsychology*, 24, 419–430.
- Duchaine, B., & Nakayama, K. (2006). The Cambridge face memory test: Results for neurologically intact individuals and an investigation of its validity using inverted face stimuli and prosopagnosic participants. *Neuropsychologia*, 44, 576–585.
- Gur, R. C., Richard, J., Huggett, P., Calkins, M. E., Macy, L., Bilker, W. B., et al. (2010). A cognitive neuroscience-based computerized battery for efficient measurement of individual differences: Standardization and initial construct validation. *Journal of Neuroscience Methods*, 187, 254–262.
- Kaufman, A. S., & Kaufman, N. L. (2004). *Kaufman assessment battery for children* (2nd ed.). Circle Pines: American Guidance Service.
- Klin, A., Sparrow, S. S., de Bildt, A., Cicchetti, D. V., Cohen, D. J., & Volkmar, F. R. (1999). A normed study of face recognition in autism and related disorders. *Journal of Autism and Developmental Disorders*, 29, 499–508.
- Korkman, M., Kirk, U., & Kemp, S. L. (2007). *NEPSY II. Clinical and interpretative manual*. San Antonio: Psychological Corporation.
- Langdell, T. (1978). Recognition of faces: An approach to the study of autism. *Journal of Child Psychology and Psychiatry*, 19, 255–268.
- Logan, A. J., Wilkinson, F., Wilson, H. R., Gordon, G. E., & Loffler, G. (2016). The Caledonian face test: A new test of face discrimination. *Vision Research*, 119, 29–41.
- Moore, T. M., Reise, S. P., Gur, R. E., Hakonarson, H., & Gur, R. C. (2015). Psychometric properties of the Penn computerized neurocognitive battery. *Neuropsychology*, 29, 235–246.
- Russell, R., Duchaine, B., & Nakayama, K. (2009). Super-recognisers: People with extraordinary face recognition ability. *Psychonomic Bulletin & Review*, 16, 252–257.
- Russell, R., Chatterjee, G., & Nakayama, K. (2012). Developmental prosopagnosia and super-recognition: no special role for surface reflectance processing. *Neuropsychologia*, 50, 334–340.
- Swagerman, S. C., de Geus, E. J. C., Kan, K. J., van Bergen, E., Nieuwboer, H. A., Koenis, M. M., et al. (2016). The computerized neurocognitive battery: Validation, aging effects, and heritability across cognitive domains. *Neuropsychology*, 30, 53–64.
- Tanaka, J., Lincoln, S., & Hegg, L. (2003). A framework for the study and treatment of face processing deficits in autism. In H. Leder & G. Switzer (Eds.), *The development of face processing*. Berlin: Hogrefe Publishers.
- Thomas, A., Lawler, K., Olson, I. R., & Aguirre, G. K. (2008). The Philadelphia face perception battery. *Archives of Clinical Neuropsychology*, 23, 175–187.
- Volkmar, F. R., Sparrow, S. S., Rende, R. D., & Cohen, D. J. (1989). Facial perception in autism. *Journal of Child Psychology & Psychiatry*, 30, 591–598.
- Warrington, E. K. (1984). *Recognition memory test*. Windsor: NFER-Nelson.
- Wechsler, D. (1997). *Wechsler memory scale* (3rd ed.). San Antonio: The Psychological Corporation.
- Wolfe, J. M., Tanaka, J. W., Klaiman, C., Cockburn, J., Herlihy, L., Brown, C., South, M., McPartland, J., Kaiser, M. D., Phillips, R., & Schultz, R. T. (2008). Specific impairment of face-processing abilities in children with autism spectrum disorder using the *Let's Face It! Skills* battery. *Autism Research*, 1, 329–340.

Facilitated Communication

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Definition

Facilitated communication (FC) is an intervention for individuals with a disability that involves pointing to letters or other materials to communicate using the support of a partner. The supporting partner, or facilitator, may provide emotional, physical, or communicative support as needed by the person with a disability (“FC user”) to produce messages. Although FC can be used with different targets, messages are most often generated by typing on a keyboard.

Historical Background

Facilitated communication was first recognized in the 1970s in Australia with the work of Rosemary Crossley. Crossley began to use physical support to help individuals with significant communication and motor impairments to type (Biklen 1990). In 1990 Douglas Biklen published his experience with facilitated communication while at the Dignity through Education and Learning Communication (DEAL) Center in Australia. He shared his changed perspective about the assumptions of the cognitive abilities of individuals with autism and the possibility that their speech challenges were a function of motor expression (Biklen 1990). In 1992 Biklen helped to establish the Facilitation Communication Institute (FCI) at Syracuse University (Syracuse University Institute for Community Inclusion). In the years that followed, FC use expanded, as did the professional community’s concerns about this unproven intervention. In 1992 Cummins et al. published a critical response

to Biklen’s article, challenging many of Biklen’s claims. They concluded their commentary by saying “to imply in the absence of empirical validation that this technique can break through the severe communication difficulties of children with autistic or other severe developmental disabilities raises serious ethical concerns” (Cummins and Prior 1992, p. 240). Even as the use of FC was growing in the 1990s, it continued to be surrounded by controversy related to its validity. Over the next nearly 30 years, numerous studies demonstrated the influence of the facilitator on the messages produced (Hemsley et al. 2018a). Researchers showed again and again that the messages were being authored by facilitator and not the FC user and were therefore denying the person their right to communicate (Hemsley et al. 2018a). By 2020 many professional organizations had officially denounced FC as a valid communication intervention (American Speech-Language-Hearing Association n.d.). FC, or typing to communicate, continues to be supported by the Institute for Community Inclusion (ICI, formerly FCI) and still practiced by some individuals.

Rationale or Underlying Theory

In her 1994 book on facilitated communication, Rosemary Crossley explained that “a partner (facilitator) helps the communication aid user overcome physical problems and develop functional movement patterns” (p. 3).

Goals and Objectives

The training materials associated with FC (Institute for Community Inclusion Training Standards Manual 2000) indicate that the goal of FC is independent typing, which may include speech or reading of the typed message aloud. The Standards state that FC user should be able to demonstrate that they are the source of the typed messages, so that there is no suspicion that it is the facilitator who is influencing the content. Some of the ways described in the Manual include

having the FC user type a message not known to the facilitator or completing some typing tasks independently. The FC user and facilitator are expected to regularly practice activities to build independence. A successful, independent communicator would need to access their communication support, initiate communication, and produce accurate messages without the support of the facilitator (ICITSM 2000).

Treatment Participants

According to information from the Institute for Community Inclusion (ICI) at Syracuse University, people who have significant communication impairment resulting in limited or impaired speech and who are not able to point reliably without support would be potential candidates for FC. The individuals who use this method are referred to as FC user, typer, or communication aid user. Information from the ICI Training Standards Manual (2000) recommends that consideration for the use of FC begin with an assessment which looks at present and past communication, movement, and support strategies. An experienced facilitator trained in assessment would help determine the benefit of FC and possibly develop recommendations for its use (ICI Training Standards 2000).

Treatment Procedures

According to the Institute for Community Inclusion's website entry on supported typing, FC involves the use of a facilitator to help the person with a disability to communicate by pointing at a target. The target can be an object or picture, but most often it is a letter. The letters are accessed from a paper-based or electronic communication display. The ICI Training Standards Manual (ICI) recommends that multiple people be trained as facilitators to increase the validity of the process.

The person who provides the support to the person typing is known as the facilitator. Facilitators are expected to receive training on their role, learning about the specific methods of

assistance that are part of FC. The Institute for Community Inclusion's Training Standards Manual provides the information below regarding the individualized physical, emotional, or communicative support provided by facilitators. The facilitator's physical support is usually provided at the hand, wrist, arm, or shoulder. The facilitator provides resistance, stability, and movement to increase the accuracy of the FC user's point. Emotional support includes encouragement, motivation, and sharing an expectation of competence. It also promotes focus and attention. Communicative support by the facilitator includes monitoring and providing prompts and feedback to make sure the user's selections and message are clear. A wide range of support is acceptable with FC, and ultimately the specific format of assistance is based on the FC user's motor and emotional needs. Depending on the nature and severity of the persons' motor and emotional needs, it may take considerable time for FC to be successful even with the supports described.

Efficacy Information

Questions about the validity of facilitated communication have focused primarily on authorship, determining whether the typed messages are produced by the FC user or if they are a product of the facilitator (Hemsley et al. 2018a). In a review of the FC research, Hemsley et al. (2018a) noted that the primary method for testing the authenticity of the supported typing process is to see if the person typing can share information that is unknown to the facilitator. In 2014 Schlosser et al. published the results of their comprehensive review of peer-reviewed research establishing the source of messages generated through facilitated communication. The review was conducted by a committee of members of the International Society of Augmentative and Alternative Communication (ISAAC) in order to inform the organization's Position Statement on Facilitated Communication (Schlosser et al. 2014).

Relevant materials for the review were initially identified for the use of the term facilitated

communication or equivalent (e.g., supported typing, assisted typing) (Schlosser et al. 2014). Materials were then compared to a checklist of features to identify those that met the committee's established criteria for inclusion in the review. Many of the materials originally identified did not focus on FC or were not published in a peer-reviewed journal and therefore were excluded (Schlosser et al. 2014). In addition, papers needed to include conditions that allowed for the identification of the source of the typed messages. They had to be able to establish authorship of the communication to be included in the review. A total of seven products, both systematic reviews and individual studies, met the checklist criteria and were included in the analysis. Schlosser et al. reported that the results of every one of the reviewed sources established that the facilitator was the source of the typed messages. The messages generated were influenced by the facilitator and did not reflect the communication of the person with a disability. Considered together, the reviewed materials contained strong evidence for the conclusion that the facilitator, and not the FC user, was the author of the messages (Hemsley et al. 2018a). The results of this review were used as the basis of ISAAC's Facilitated Communication Position Statement of 2014. This paper reads "ISAAC does not support FC as a valid form of AAC... The weight of evidence does not support FC and therefore it cannot be recommended for use in clinical practice" (ISAAC 2014, p. 358).

In 2017–2018 the American Speech Language Hearing Association (ASHA) created an Ad Hoc Committee to review the research related to authorship to inform an updated Position Paper on Facilitated Communication. The Committee found that since the work of Schlosser et al. in 2014, no new research had been conducted that included conditions for establishing authorship using facilitated communication (Hemsley et al. 2018a). Schlosser et al. had stated that "Results indicated unequivocal evidence for facilitator control: messages generated through FC are authored by the facilitators rather than the individuals with disabilities. Hence, FC is a technique that has no validity" (Schlosser et al. 2014, p. 2). In 2018 ASHA updated its 1995 Position Paper on

Facilitated Communication to reflect a stronger statement. The 2018 Position Paper ASHA stated:

Facilitated Communication (FC) is a discredited technique that should not be used. There is no scientific evidence of the validity of FC, and there is extensive scientific evidence—produced over several decades and across several countries—that messages are authored by the “facilitator” rather than the person with a disability. (ASHA 2018)

In addition, ASHA's Position Paper (2018) expressed concerns over the use of FC and identified sources of potential harm. When FC is inaccurately used to represent the person's communication they are denied the right to communicate their own thoughts. In addition, focus on FC can reduce the time and financial resources available to pursue other AAC tools and interventions which have been proven effective. ASHA cautions that SLPs have a responsibility to share the issues related to FC and share other AAC options (ASHA 2018).

A number of other professional organizations and advocacy groups have officially denounced the practice of FC based on lack of research to support its claims of authorship. These include American Academy of Pediatrics, American Psychological Association, Association for Behavior Analysis, Association for Science in Autism Treatment, Speech-Language and Audiology Canada, and American Association on Intellectual and Developmental Disabilities (ASHA n.d.)

Outcome Measurement

In the ICI Training Standards Manual (2000), the ICI indicates a range of factors which can affect the FC user's progress toward independence, including those related to the user (e.g., motor skills), the facilitator (their skill, experience, availability), access to the right tools and environments, and practice opportunities.

The ICI Training Standards Manual (2000) offers the following suggestions for documentation as indicators of valid application of FC:

similar patterns of spelling and typographical errors across facilitators; typing of similar topics and

themes across facilitators; consistent style of typing across facilitators; instances of independent and/or initiated communication; self-correction of mistakes; effective use of protest strategies; sharing of information not known to the facilitator; successful participation in message-passing exercises; behavior or actions that confirm typed communication; speech that correlates with typed communication; consistent physical style of typing across facilitators, and physical attention to the typing (e.g., eye contact with the communication device). (p. 13)

Qualifications of Treatment Providers

Facilitators are expected to receive training on the proper way to provide support without influencing the FC user. “All facilitators should participate in a supervised training process so that they learn the appropriate and correct skills for supporting a person in his or her communication” (ICI Training Standards Manual 2000, p. 11). The ICI Training Standards Manual (2000) provides information for facilitators on best practices and methods of moving toward independence.

See Also

- [Augmentative and Assistive Technology](#)
- [Nonverbal Communication](#)
- [Visual Supports](#)

References and Reading

- American Association on Intellectual and Developmental Disabilities. (2019). *Facilitated communication and rapid prompting method: Position statement of the AAIDD board of directors*. <https://www.aaid.org/news-policy/policy/position-statements/facilitated-communication-and-rapid-prompting-method>
- American Speech-Language-Hearing Association. (2018). Facilitated communication [Position statement]. www.asha.org/policy/
- American Speech-Language-Hearing Association. (n.d.). *A number of organizations caution against use of FC and RPM*. <https://www.asha.org/SLP/Cautions-Against-Use-of-FC-and-RPM-Widely-Shared/>
- Biklen, D. (1990). Communication unbound: Autism and praxis. *Harvard Educational Review*, 60(3), 291–314. ProQuest Central.
- Biklen, D., Winston Morton, M., Gold, D., Berrigan, C., & Swaminathan, S. (1992). Facilitated communication: Implications for individuals with autism. *Topics in Language Disorders*, 12(4), 1–28.
- Crossley, R. (1994). *Facilitated communication training*. New York: Teachers College Press.
- Cummins, R. A., & Prior, M. P. (1992). Autism and assisted communication: A response to Biklen. *Harvard Educational Review*, 62(2), 228–241. <https://doi.org/10.17763/haer.62.2.p86j205360177322>.
- Hemsley, B., Bryant, L., Schlosser, R. W., Shane, H. C., Lang, R., Paul, D., & Ireland, M. (2018a). Systematic review of facilitated communication 2014–2018 finds no new evidence that messages delivered using facilitated communication are authored by the person with disability. *Autism & Developmental Language Impairments*. <https://doi.org/10.1177/2396941518821570>.
- Hemsley, B., Shane, H. C., Todd, J. T., Schlosser, R. W., & Lang, R. (2018b). *It's time to stop exposing people to the dangers of facilitated communication*. <https://theconversation.com/its-time-to-stop-exposing-people-to-the-dangers-of-facilitated-communication-95942>
- Institute on Community Inclusion. (2000). ICI training standards manual. Retrieved January 21, 2020, from Syracuse University's website: https://ici.syr.edu/wp-content/uploads/2018/09/Training_Standards_Manual.pdf
- Institute on Community Inclusion. (n.d.). <https://ici.syr.edu/>
- International Society for Augmentative and Alternative Communication. (2014). ISAAC position statement on facilitated communication. *Augmentative and Alternative Communication*, 30(4), 357–358. <https://doi.org/10.3109/07434618.2014.971492>.
- Schlosser, R., Balandin, S., Hemsley, B., Iacono, T., Probst, P., & von Tetzchner, S. (2014). Facilitated communication and authorship: A systematic review. *Augmentative and Alternative Communication*, 30(4), 359–368.
- Singer, G., Horner, R., Dunlap, G., & Wang, M. (2015). Standards of proof: TASH, facilitated communication, and the science-based practices movement. *Research and Practice for Persons with Severe Disabilities*, 39, 178–188. <https://doi.org/10.1177/1540796914558831>.

Faciogenital Dysplasia

- [Aarskog Syndrome](#)

Fact Memory

- [Explicit Memory](#)

Factor Analysis

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Definition

Factor analysis refers to statistical methods that attempt to represent a set of correlated observed variables (questionnaire items, autism symptoms, etc.) using a smaller number of unobserved (latent) variables, also called factors or dimensions. The ultimate goal of this technique is to determine the number and composition of factors or dimensions that can parsimoniously represent a larger set of variables.

Historical Background

Factor analytic methods began with Charles Spearman's observation that specific measures of children's academic performance and cognitive skills were correlated. He developed a theory that the correlations between different measures of ability were due to a single underlying common factor or dimension that he called "g" or general intelligence (Spearman 1904). Subsequent uses of factor analysis examined whether multiple components of intelligence can be identified, such as verbal and nonverbal abilities. As factor analytic techniques became more popular, their use expanded to other sets of variables, including psychiatric symptoms. Factor analytic studies of psychiatric symptoms have been helpful in identifying the "boundaries" of psychiatric conditions and separating important core markers and components from peripheral or associated symptoms.

One of the earliest uses of factor analysis in autism was a study by Freeman et al. (1980)

examining the factor structure of a behavior observation scale. Since then, numerous studies have used factor analysis methods in samples of individuals with autism. A search of the terms *autism and factor analysis* in PubMed yields more than 800 hits. The majority of studies examine autism and/or other behavioral symptoms, often using specific measures designed to assist in screening, diagnosing, or tracking of symptoms in individuals with autism.

Existing literature suggests that the primary use of factor analysis has been to better understand which autism symptoms group/cluster together to form broader symptom domains. The other major use has been to evaluate how many dimensions are captured by a specific instrument, typically a measure of autism symptoms. Most instruments do not include all autism symptoms or include some items that are not considered core and/or unique aspects of autism (e.g., self-injurious behavior). Thus, results of factor analytic studies examining specific instruments are mainly useful for scoring the instrument rather than understanding the core symptom domains in autism. The exceptions are factor analyses of measures which comprehensively cover autism symptoms, such as the Autism Diagnostic Interview-Revised (Lord et al. 1994).

There are two general categories of factor analyses: exploratory and confirmatory factor analyses. The main difference in these categories is that, in exploratory analyses, variables are used with no preconceived notion or hypothesis of which variables will measure which factor. In confirmatory factor analysis, the researcher specifies, a priori, which variables will measure which factors and whether factors will be correlated. Researchers can be even more specific by explicitly "fixing" how an item or symptom will load on a given factor. Thus, confirmatory methods are more rigorous in that a researcher must have a hypothesis prior to conducting the analysis. Confirmatory methods are also useful in that multiple models that correspond to different hypotheses can be specified and tested. Direct comparisons of the fit of these models to the data provide evidence of the relative plausibility of the hypotheses. However, because not all possible

models can be tested in confirmatory factor analyses, the better fitting model is not viewed as the “truth” but rather a better approximation of reality.

Both exploratory and confirmatory approaches consist of multiple methods for estimating the factor structure, including estimating model fit to the observed variables. Norris and Lecavalier (2010) provide an excellent overview of exploratory factor analytic methods and recommendations for their use in autism and other developmental disorders. Tabachnick and Fidell (2016) provide a very accessible introduction to both exploratory and confirmatory methods. Although principal components analysis (PCA) is not strictly a factor analysis method, results are often highly similar, and this method is included when discussing previous factor analytic studies of autism. Conceptual differences are that PCA involves analyzing all the variance in the indicators whereas factor analysis involves analyzing the common variance (covariance) in the indicators. Differences between the two methodologies are discussed in detail in Tabachnick and Fidell (2016).

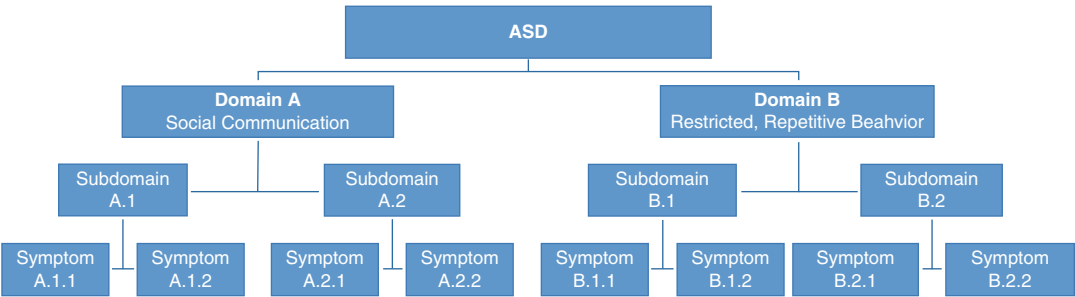
Current Knowledge

Defining the Autism Symptom Phenotype

Factor analytic methods have helped to clarify the nature and underlying structure of autism

symptoms. Although there is not perfect agreement across studies, a consensus has emerged as to the number and composition of autism symptom dimensions. Based on accumulating evidence from factor analytic studies (Frazier et al. 2008; Snow et al. 2009; van Lang et al. 2006), the new diagnostic criteria for ASD include two domains (DSM-5) instead of three domains (DSM IV) – a combined social communication domain and a restricted, repetitive behavior domain (American Psychiatric Association 2013). Several investigations have since used factor analytic techniques to evaluate the underlying structure of the new criteria (2-factor model; see Fig. 1). Since these analyses were based on a priori hypotheses, confirmatory factor analysis was used in most studies (Guthrie et al. 2013; Sipes and Matson 2014; Mandy et al. 2012).

Although these types of studies can be informative, results are mostly driven by the samples used; and in the absence of new samples assembled using the DSM-5 criteria, these results need to be interpreted with caution and replicated in future studies. As Kim et al. (2017) note, instead of continuing to debate the optimal number of factors at a given level of the ASD phenotype, perhaps there is value in examining the underlying structural hierarchy in its symptomatology. Such a hierarchical approach would inform our understanding on how features from differing levels (core diagnostic domains, subdomains, related and/or comorbid phenotypes) come together to



Factor Analysis, Fig. 1 Schematic of the ASD factor structure with diagnosis at the top level, followed by the two primary diagnostic symptom domains (as per DSM-5) and subdomains and individual symptoms. Due to the lack

of agreement across studies regarding the exact number and composition of subdomains and symptoms, those are not specified/named in this figure

make up the complex clinical profiles seen in individuals with ASD.

Assessment Tools Used in Autism

As previously noted, factor analysis has also been used in studies evaluating the number and composition of factors/dimensions captured by a specific instrument, typically a measure of autism symptoms (Constantino et al. 2004; Slappendel et al. 2016).

There are hundreds of published studies describing the use of factor analytic methods to evaluate the structure of different tools measuring autism-related constructs. Since this section is not meant to serve as a review of the literature, we highlight here an example of a tool – the Social Responsiveness Scale (SRS) – that has triggered constructive debate among researchers over the past decade or so. Several studies examining the SRS, a quantitative trait measure of autism symptoms, have identified only a single autism symptom factor with predominantly social difficulties represented (Cheon et al. 2016; Constantino et al. 2004; Constantino and Gruber 2005). This discrepancy between a single symptom severity model and the 2-factor model in the DSM-5 (also see Fig. 1) is most likely due to the predominance of social indicators examined on the Social Responsiveness Scale and the focus on population samples rather than mixed clinical or autism-only samples. Specifically, the discrepancy between a single autism factor and separate social communication and restricted/repetitive dimensions is at least partly due to the fact that, in population samples, the majority of healthy individuals will show less variation in symptom levels, while a small minority of mostly affected individuals will show very large variations in symptom levels. This pattern exaggerates correlations among social communication and restricted/repetitive behavior symptoms, resulting in a single symptom severity factor.

In a more recent study of the SRS-2, Frazier et al. (2014) showed that both a two-factor structure (as per DSM-5) and a three-factor structure (DSM-5 but with restricted, repetitive behavior

factor breaking into two factors, insistence on sameness and repetitive mannerisms; see also Georgiades et al. 2007) provided acceptable model fits using confirmatory factor analysis. Of note were the intercorrelations among symptom factors/domains, something that suggests that understanding their co-emergence remains a high priority in conceptualizing common clinical profiles and causal mechanisms underlying ASD (Frazier et al. 2014).

Grouping Variables and Individuals

One of the recent and promising methodological advances in the field has been the use of novel techniques that simultaneously examine categorical and dimensional models of ASD. By using factor mixture modeling, a method that includes factor analysis and latent class analysis, researchers are able to move away from the simplistic debate of “categories versus dimensions” and examine phenotypic models of ASD that integrate the two. By doing so, empirical methods can be used to begin to disentangle the observed heterogeneity in ASD and examine how both, variables and individuals, come together to form more homogeneous dimensions and subgroups within the autism spectrum (Frazier et al. 2010; Georgiades et al. 2013).

Future Directions

In an era where Big Data is becoming more and more prevalent, factor analytic studies may prove to be very useful in “reducing” information without losing the necessary components that can describe the nature and structure of the ASD phenotype. Such an approach, combined with new methods used for the identification of homogeneous subgroups (Frazier et al. 2010; Georgiades et al. 2013) within the spectrum, has the potential to inform the development of more precise diagnostics and interventions.

To truly understand the causes, diagnosis, and prognosis of ASD, prospective factor analytic studies are needed to examine the hierarchical structure of the revised DSM-5 criteria in relation

to time (Georgiades et al. 2017). Such studies can also examine the associations and potential interactions between the core autism domains and symptoms (see Fig. 1) and the “clinical specifiers” (e.g., intellectual and language impairment, comorbid disorders, etc.) introduced in the DSM-5. Factor analytic methods could also be expanded to better understand underlying neurobiological characteristics. For example, different methods may be useful for reducing genetic variants and/or imaging signals variants to a smaller set of dimensions that may correspond with specific symptom and/or behavior profiles.

Future work using factor analyses and related methods would benefit from using empirically supported approaches for determining the number and composition of dimensions. It is clear that a large number of previous studies did not use empirically recommended approaches to conducting analyses. In future work, it will be important that researchers follow general recommendations for implementing both exploratory and confirmatory methods (McCutcheon 2002; Muthén 2001; Norris and Lecavalier 2010; Ruscio et al. 2006).

See Also

- Latent Variable Modeling
- Statistical Approaches to Subtyping

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: APA Press.
- Cheon, K. A., Park, J. I., Koh, Y. J., Song, J., Hong, H. J., Kim, Y. K., . . . Paik, K. C. (2016). The social responsiveness scale in relation to DSM IV and DSM5 ASD in Korean children. *Autism Research*, 9(9), 970–980.
- Constantino, J. N., & Gruber, C. P. (2005). *Social responsiveness scale: Manual*. Los Angeles: Western Psychological Services.
- Constantino, J. N., Gruber, C. P., Davis, S., Hayes, S., Passanante, N., & Przybeck, T. (2004). The factor structure of autistic traits. *Journal of Child Psychology and Psychiatry*, 45(4), 719–726.
- Frazier, T. W., Youngstrom, E. A., Kubu, C. S., Sinclair, L., & Rezaei, A. (2008). Exploratory and confirmatory factor analysis of the autism diagnostic interview- revised. *Journal of Autism and Developmental Disorders*, 38(3), 474–480.
- Frazier, T. W., Youngstrom, E. A., Sinclair, L., Kubu, C. S., Law, P., & Rezaei, A. (2010). Autism spectrum disorders as a qualitatively distinct category from typical behavior in a large, clinically ascertained sample. *Assessment*, 17(3), 308–320.
- Frazier, T. W., Ratliff, K. R., Gruber, C., Zhang, Y., Law, P. A., & Constantino, J. N. (2014). Confirmatory factor analytic structure and measurement invariance of quantitative autistic traits measured by the Social Responsiveness Scale-2. *Autism*, 18(1), 31–44.
- Freeman, B. J., Schroth, P., Ritvo, E., Guthrie, D., & Wake, L. (1980). The behavior observation scale for autism (BOS): Initial results of factor analysis. *Journal of Autism and Developmental Disorders*, 10(3), 343–346.
- Georgiades, S., Szatmari, P., Zwaigenbaum, L., Duku, E., Bryson, S., Roberts, W., . . . Mahoney, W. (2007). Structure of the autism symptom phenotype: A proposed multidimensional model. *Journal of the American Academy of Child & Adolescent Psychiatry*, 46(2), 188–196.
- Georgiades, S., Szatmari, P., Boyle, M., Hanna, S., Duku, E., Zwaigenbaum, L., . . . Smith, I. (2013). Investigating phenotypic heterogeneity in children with autism spectrum disorder: A factor mixture modeling approach. *Journal of Child Psychology and Psychiatry*, 54(2), 206–215.
- Georgiades, S., Bishop, S. L., & Frazier, T. (2017). Editorial perspective: Longitudinal research in autism—introducing the concept of ‘chronogeneity’. *Journal of Child Psychology and Psychiatry*, 58(5), 634–636.
- Guthrie, W., Swineford, L. B., Wetherby, A. M., & Lord, C. (2013). Comparison of DSM-IV and DSM-5 factor structure models for toddlers with autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 52(8), 797–805.
- Kim, H., Keifer, C. M., Rodriguez-Seijas, C., Eaton, N. R., Lerner, M. D., & Gadow, K. D. (2017). Structural hierarchy of autism spectrum disorder symptoms: an integrative framework. *Journal of Child Psychology and Psychiatry*. <https://doi.org/10.1111/jcpp.12698>
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism Diagnostic Interview-Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(5), 659–685.
- Mandy, W. P., Charman, T., & Skuse, D. H. (2012). Testing the construct validity of proposed criteria for DSM-5 autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(1), 41–50.
- McCutcheon, A. L. (2002). Basic concepts and procedures in single- and multiple-group latent class analysis.

- In J. A. Hagenaars & A. L. McCutcheon (Eds.), *Applied latent class analysis* (pp. 56–85). Cambridge, UK: Cambridge University Press.
- Muthén, B. O. (2001). Second-generation structural equation modeling with a combination of categorical and continuous latent variables: New opportunities for latent class/latent growth modeling. In A. Sayer & Collins (Eds.), *New methods for the analysis of change* (pp. 291–322). Washington, DC: American Psychological Association.
- Norris, M., & Lecavalier, L. (2010). Evaluating the use of exploratory factor analysis in developmental disability psychological research. *Journal of Autism and Developmental Disorders*, 40(1), 8–20.
- Ruscio, J., Haslam, N., & Ruscio, A. M. (2006). *An introduction to the taxometric method: A practical guide*. Mahwah: Lawrence Erlbaum Associates.
- Sipes, M., & Matson, J. L. (2014). Factor structure for autism spectrum disorders with toddlers using DSM-IV and DSM-5 criteria. *Journal of Autism and Developmental Disorders*, 44(3), 636–647.
- Slappendel, G., Mandy, W., van der Ende, J., Verhulst, F. C., van der Sijde, A., Duvekot, J., ... Greaves-Lord, K. (2016). Utility of the 3Di short version for the diagnostic assessment of autism spectrum disorder and compatibility with DSM-5. *Journal of Autism and Developmental Disorders*, 46(5), 1834–1846.
- Snow, A. V., Lecavalier, L., & Houts, C. (2009). The structure of the autism diagnostic interview-revised: Diagnostic and phenotypic implications. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 50(6), 734–742.
- Spearman, C. (1904). “General intelligence,” objectively determined and measured. *The American Journal of Psychology*, 15(2), 201–292.
- Tabachnick, B. G., & Fidell, L. S. (2016). *Using multivariate statistics* (6th ed.). Boston: Pearson/Allyn & Bacon.
- van Lang, N. D. J., Boomsma, A., Sytema, S., Bildt, A. A., Kraijer, D. W., & Ketelaars, C. (2006). Structural equation analysis of a hypothesized symptom model in the autism spectrum. *Journal of Child Psychology and Psychiatry*, 47, 37–44.

Factor Mixture Modeling

► Latent Variable Modeling

Factors Affecting Outcome

► Course of Development

Factors Affecting Outcomes

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Definition

The outcome for individuals with autism is very variable and depends not only on the characteristics of the individual but on many family and environmental factors and external support systems. Childhood factors that are associated with later outcome include early language development, IQ, severity of autism symptoms, and access to appropriate autism-specific intervention. In adolescence and adulthood, the onset of comorbid mental health problems and the lack of adequate support and interventions are associated with poorer outcomes.

Historical Background

It has been evident since the very earliest follow-up studies of Eisenberg (1956), Rutter et al. (1967), Kanner (1973), and others that outcome in autism is highly variable. Many individuals remain highly dependent on support throughout their lives; others are able to live independently and some go on to make significant achievements. Both Asperger (1944) and Kanner (1971) were struck by the wide heterogeneity in outcome among individuals with autism. Kanner (1971), for example, noted that “The results of the follow-up after about 30 years . . . invite serious curiosities about the departures from the initial likeness, ranging all the way from complete deterioration to a combination of occupational adequacy with limited, though superficially smooth social adjustment.” Similarly, Asperger (1944) commented that although the social integration of individuals with autism might be expected to be extremely difficult, if not impossible, this was the case only for a minority and “almost exclusively in

those people with considerable intellectual retardation in addition to autism . . . This is not so with intellectually intact autistic individuals.” Indeed, he suggested that “able autistic individuals can rise to eminent positions and perform with such outstanding success that one may even conclude that *only* such people are capable of certain achievements.”

The factors affecting outcome, and the reasons why some individuals are able to make very good progress while others continue to have significant problems throughout their lives, has been the focus of considerable research over the years.

Current Knowledge

Although many follow-up studies have indicated a relatively poor outcome for individuals with autism, the results of such research cannot be interpreted without detailed information on the characteristics of the individuals involved. Kanner (1943) and Asperger (1944) were among the first to note a possible association between higher intellectual ability and greater achievements in adulthood. Subsequent follow-up studies have confirmed that childhood IQ is highly correlated with adult outcome. For example, in a series of studies conducted in the UK in the late 1960s and early 1970s, Rutter and colleagues (Lockyer and Rutter 1969; Rutter et al. 1967) found that individuals with an IQ below 60 were far more likely to have a “poor” or “very poor” outcome than those with an IQ above 60. Individuals with an IQ of at least 70 (i.e., at the lower end of the normal range) tended to be rated as having a “fair” outcome, while those with a “good outcome” had an average IQ above 80. More recent studies, involving individuals across the IQ range (i.e., from severe cognitive impairment to superior intelligence), have also noted a strong association between IQ and outcome, with very few individuals with a childhood IQ below 75 living independently as adults (see Howlin and Magiati 2017; Magiati et al. 2014; Steinhausen et al. 2016 for reviews). However, childhood IQ alone is not the only factor affecting outcome (Bal et al. 2015; Lord et al. 2015), and many individuals

with a high IQ in childhood still do poorly in adulthood. Thus, it seems that while *only* individuals with a childhood IQ in the average range or above do well in adulthood, even among those with an IQ above 70, outcome can still vary markedly (Steinhausen et al. 2016). The *range* of early cognitive abilities may also be important, in that children who are able to score only on nonverbal tests, or on a very limited range of subtests – even if their scores on these assessments are within the normal range – tend to have a poorer prognosis than those able to score on both verbal and nonverbal tests in early childhood (Howlin et al. 2014).

Another crucial indicator of later outcome is early language development and it is now well established that there is a strong link between early language abilities and subsequent development (Magiati et al. 2014). Thus, the majority of individuals who go on to do well as adults have usually developed at least some useful speech by the age of 5 years. However, there are exceptions to this, and some children with significant early delays in language later catch up and may make good progress more generally. Overall, however, language impairments, particularly if coupled with low IQ, are the factors most strongly associated with a poorer adult outcome. In adulthood, individuals with good verbal comprehension, good spoken language, and a verbal IQ in the normal range are significantly more likely to be functioning well socially than those who are impaired in these areas (Howlin et al. 2014).

There also appears to be an association between the severity of early autistic symptomatology and later outcome, although findings here are inconsistent. Indeed, predicting later adult outcomes from early child characteristics remains highly challenging (Howlin and Magiati 2017; Lord et al. 2015). There is a need to focus more research on different patterns of developmental trajectories in autism, as such knowledge may be important for identifying different genetic aetiologies as well as having implications for more individually tailored interventions (Lord et al. 2015).

Although many follow-up studies suggest that the severity of autistic symptoms tends to reduce

with age, the overall level of autistic symptomatology in adulthood continues to affect social and economic independence (Gillberg et al. 2016; Howlin et al. 2013; Lord et al. 2015). The presence of mental health problems can also have a very negative impact on outcome, as well as proving difficult to treat effectively (Howlin and Magiati 2017). Although estimates of the frequency of mental health problems in autism vary widely, it is estimated that at least half of all individuals with autism suffer from significant emotional and psychiatric difficulties (Croen et al. 2015; Russell et al. 2016; Moss et al. 2015). The most frequent problems relate to stress, anxiety, and depression, but attention deficit disorders and obsessive compulsive disorders are also common. Physical disorders, including epilepsy, are more frequent than in the general population (Croen et al. 2015) and these too can have a negative impact on adult functioning.

Data on the effects of ageing remain limited. Although overall severity of autism symptoms tends to reduce between early childhood and early adulthood, there are few studies of trajectories of change in older individuals. Nevertheless, some small-scale studies suggest that certain cognitive skills (notably visual and working memory) may be less prone to decline in elderly people with autism than in the general population (Lever and Geurts 2016). There is also some indication that quality of life in ASD is less affected by age than in the general population (van Heijst and Geurts 2015), but to date, studies on ageing in ASD are small scale and involve few participants aged over 60.

The role of gender remains uncertain since most follow-up studies have involved so few females. There are some suggestions that women with autism have poorer social outcomes, especially with respect to employment (Taylor et al. 2015) and quality of life (Bishop-Fitzpatrick et al. 2016). However, other studies (Kirby et al. 2016; Woodman et al. 2015) have identified no significant impact of gender.

The role of external, environmental factors on outcome is yet another area in need of much greater research. Although, to date, there is no evidence that early, highly intensive behavioral

or other interventions make a significant difference to functioning in adulthood, it is likely that the quality of early intervention, access to appropriate education programs, and support for families will have an important impact on later life. There are indications, too, that mental health difficulties in adulthood may be associated with environmental disturbance, such as the transition from school, difficulties in coping with college work or employment, family disruption and loss, and change and unpredictability in daily life. The paucity of appropriate adult services, especially for individuals of average IQ, is also likely to be another reason why adults with ASD are more economically, educationally, and socially disadvantaged than adults with other developmental or intellectual disabilities (Roux et al. 2013). Recent studies also suggest the importance of a wide range of other child and adult factors associated with social functioning and autism symptom severity in adulthood. These include bullying, extent of inclusion in school, maternal warmth/quality of mother-child relationships, physical health, independence in daily living skills, executive function, stress, physical health, and adaptive behavior skills (see Howlin and Magiati 2017 for review).

Future Directions

Long-term follow-up studies over the past 50 years have taught us a great deal about the factors that are associated with outcome in adulthood. However, many issues still remain to be resolved. Thus, although there is good evidence that language, IQ, and severity of autism are all linked to longer-term prognosis, we still know relatively little about how these factors interact, or how far environmental, genetic, family, social, educational, racial, or other variables may ameliorate or exacerbate their impact. The role of temperament and personality is almost unexplored, and almost nothing is known about factors that influence functioning in the later years of life.

Research into effective treatments has led to major improvements in early intervention for

many children. However, how long the effects of such interventions endure remains unknown, and much longer-term follow-up studies are needed if we are to judge the true benefit of these (often very costly) programs.

In summary, although research into children with autism has made significant strides forward over recent years, leading to major advances in diagnosis, understanding of causes, and treatment, research into adulthood still has a long way to go. Follow-up studies of individuals identified and fully assessed as children and followed up periodically from decade to decade are essential if we are to understand the factors that influence prognosis and how these factors exert their effects.

See Also

- [Adulthood, Transition To](#)
- [Asperger Syndrome Follow-Up Studies](#)
- [Employment](#)
- [Employment in Adult Life](#)
- [Factors Affecting Outcome](#)
- [Living Arrangements in Adulthood](#)
- [Psychotic Disorder](#)

References and Reading

- Asperger, H. (1944). "Autistic psychopathy" in childhood (U. Frith, Trans., and annotated in Autism and Asperger syndrome (1991)). In U. Frith (Ed.), *Autism and asperger syndrome* (1st ed., pp. 37–92). Cambridge: Cambridge University Press.
- Bal, V. H., Kim, S. H., Cheong, D., & Lord, C. (2015). Daily living skills in individuals with autism spectrum disorder from 2 to 21 years of age. *Autism, 19*(7), 774–784.
- Bishop-Fitzpatrick, L., Hong, J., Smith, L. E., et al. (2016). Characterizing objective quality of life and normative outcomes in adults with autism spectrum disorder: An exploratory latent class analysis. *Journal of Autism and Developmental Disorders, 46*(8), 707–719.
- Croen, L. A., Zerbo, O., Qian, Y., et al. (2015). The health status of adults on the autism spectrum. *Autism, 19*(7), 814–823.
- Eisenberg, L. (1956). The autistic child in adolescence. *The American Journal of Psychiatry, 112*, 607–612.
- Gillberg, I. C., Helles, A., Billstedt, E., & Gillberg, C. (2016). Boys with Asperger syndrome grow up: Psychiatric and neurodevelopmental disorders 20 years after initial diagnosis. *Journal of Autism and Developmental Disorders, 46*(1), 74–82.
- Howlin, P., & Magiati, I. (2017). Autism spectrum disorder: Outcomes in adulthood. *Current Opinion in Psychiatry, 30*(2), 69–76.
- Howlin, P., Moss, P., Savage, S., & Rutter, M. (2013). Social outcomes in mid to later adulthood among individuals diagnosed with autism and average non-verbal IQ as children. *Journal of the American Academy of Child and Adolescent Psychiatry, 52*(6), 572–581.
- Howlin, P., Moss, P., Savage, S., Tempier, A., & Rutter, M. (2014). Cognitive and language skills in adults with autism: A 40 year follow-up. *Journal of Child Psychology and Psychiatry, 55*(1), 49–58.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child, 2*, 217–250.
- Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of Autism and Childhood Schizophrenia, 1*(2), 119–145.
- Kanner, L. (1973). How far can autistic children go in matters of social adaptation? In L. Kanner (Ed.), *Childhood psychosis: Initial studies and new insights* (pp. 189–213). New York: V. H. Winston.
- Kirby, A. V., Baranek, G. T., & Fox, L. (2016). Longitudinal predictors of outcomes for adults with autism spectrum disorder systematic review. *OTJR, 36*(2), 55–64.
- Lever, A. G., & Geurts, H. M. (2016). Age-related differences in cognition across the adult lifespan in autism spectrum disorder. *Autism Research, 9*, 666–676.
- Lockyer, L., & Rutter, M. (1969). A five- to fifteen-year follow-up study of infantile psychosis. III. Psychological aspects. *The British Journal of Psychiatry, 115*, 865–882.
- Lord, C., Bishop, S., & Anderson, D. (2015). Developmental trajectories as autism phenotypes. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics, 169*(2), 198–208.
- Magiati, I., Tay, X. W., & Howlin, P. (2014). Cognitive, language, social and behavioural outcomes in adults with autism spectrum disorders: A systematic review of longitudinal follow-up studies in adulthood. *Clinical Psychology Review, 34*, 73–86.
- Moss, P., Howlin, P., Savage, S., Bolton, P., & Rutter, M. (2015). Self and informant reports of mental health difficulties among adults with autism: Findings from a long-term follow-up study. *Autism, 19*(7), 832–841.
- Roux, A. M., Shattuck, P. T., Cooper, B. P., et al. (2013). Postsecondary employment experiences among young adults with an autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 52*(9), 931–939.
- Russell, A. J., Murphy, C. M., Wilson, E., et al. (2016). The mental health of individuals referred for assessment of autism spectrum disorder in adulthood: A clinic report. *Autism, 20*(5), 623–627.
- Rutter, M., Greenfield, D., & Lockyer, L. (1967). A five to fifteen year follow-up study of infantile psychosis.

- II. Social and behavioural outcome. *The British Journal of Psychiatry*, 113, 1183–1199.
- Steinhausen, H. C., Mohr Jensen, C., & Lauritsen, M. B. (2016). A systematic review and meta-analysis of the long-term overall outcome of autism spectrum disorders in adolescence and adulthood. *Acta Psychiatrica Scandinavica*, 133(6), 445–452.
- Taylor, J. L., Henninger, N. A., & Mailick, M. R. (2015). Longitudinal patterns of employment and post-secondary education for adults with autism and average-range IQ. *Autism*, 19(7), 785–793.
- van Heijst, B. F., & Geurts, H. M. (2015). Quality of life in autism across the lifespan: A meta-analysis. *Autism*, 19(2), 158–167.
- Woodman, A. C., Smith, L. E., Greenberg, J. S., & Mailick, M. R. (2015). Change in autism symptoms and maladaptive behaviors in adolescence and adulthood: The role of positive family processes. *Journal of Autism and Developmental Disorders*, 45(1), 111–126.

Fading

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Synonyms

Prompt fading; Stimulus fading

Definition

Fading is the process of reducing assistance (i.e., cues, prompts, supports) until no longer needed so that the skill or response being taught is exhibited independently. In behavioral instruction, fading generally refers to *gradually* removing supports put in place during training programs so that the target behavior eventually occurs independently. For example, when teaching a child with autism how to spell his name, the teacher starts with stating each letter out loud and having the child

repeat them until he does so without error. The adult then instructs the child to spell his name and starts by saying only the first letter out loud and then only “mouthing” (silently moving lips) the remaining letters using no voice. Eventually, the teacher asks the child to spell his name, and he does so without additional prompts. The removal of the verbal prompts illustrates the process of fading.

See Also

- Prompt Fading
- Prompts

References and Reading

- Harper, C. B., Symon, J. B. G., & Frea, W. D. (2008). Recess is time-in: Using peers to improve social skills of children with autism. *Journal of Autism and Developmental Disorders*, 38(5), 815–826.
- Krantz, P. J., & McClannahan, L. E. (1998). Social interaction skills for children with autism: A script-fading procedure for beginning readers. *Journal of Applied Behavior Analysis*, 31, 191–202.
- Miltenberger, R. (2001). *Behavior modification: Principles and procedures* (2nd ed.). Belmont: Wadsworth.
- Patel, M., Piazza, C., Kelly, M., Ochsner, C. A., & Santana, C. (2001). Using a fading procedure to increase fluid consumption in a child with feeding problems. *Journal of Applied Behavior Analysis*, 34, 357–360.

FAFSA

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Synonyms

Financial aid; Grants; Scholarships

Definition

FAFSA is an acronym which stands for Free Application for Federal Student Aid. FAFSA is the mechanism by which the government distributes grants, loans, and student work study funds. More specifically, it is the gateway by which college students and their families access Federal Pell Grants, Stafford and Perkins Loans, Federal Supplemental Education Opportunity Grants, and Federal Work-Study program funds. By completing the FAFSA form, families learn what their expected family contribution (EFC) will be. The EFC is the amount of money the family is expected to contribute to the student's college education. Prior to 2008, only students enrolled full time in a degree-bearing program at an Institution of Higher Education (IHE) were eligible to complete the FAFSA application.

With the passage of the Higher Education Opportunity Act (HEOA) of 2008 (P.L. 110-315), the regulations governing financial aid and the FAFSA process changed to make it easier for students with intellectual disabilities (ID) to gain access to postsecondary education. The term ID is broadly defined and can include students with autism spectrum disorders or cognitive impairments. According to HEOA, a student with an intellectual disability is one who has mental retardation or a significant cognitive impairment *and* is or was eligible for a Free and Appropriate Public Education (FAPE) under the Individuals with Disabilities Education Act (IDEA). This can include students who were homeschooled or attended private schools. The college is responsible for the determination and supporting documentation of the student's ID (Bergeron et al. 2010).

In order for a student with an ID to be eligible, he or she must be enrolled in an approved Comprehensive Transition and Postsecondary Program (CTP). He or she also must meet all of the general student eligibility requirements *except* the following: (1) he or she does *not* have to be enrolled for the purpose of obtaining a degree or certificate; (2) he or she is *not* required to have a high school diploma or have passed an ability-to-benefit test; and (3) he or she must maintain

satisfactory academic progress under school's policy for students in the CTP (Finkel et al. 2010).

After a public commentary period, the US Department of Education began accepting applications from IHE, for approval of their Comprehensive Transition Programs (CTP) in the latter part of 2010. An eligible CTP must be offered by a college that already has an established financial aid program for its general student body. It also must be designed to support students with intellectual disabilities (ID) and include an advising and curriculum structure. The CTP must require students with ID to participate in courses and activities with students without disabilities (Bergeron et al. 2010).

Students with ID will be eligible for Federal Pell Grants, Federal Supplemental Education Opportunity Grants, and Federal Work-Study programs funds only (Finkel et al. 2010). It is important to note that students with ID currently are not eligible for Perkins or Stafford loans.

US DOE-approved CTPs are listed at the National Coordinating Center and include the Consortium for Postsecondary Education for Individuals with Developmental Disabilities and the Center for Postsecondary Education for Individuals with Intellectual Disabilities.

See Also

- Comprehensive Transition Program
- Free Appropriate Public Education
- Individuals with Disabilities Education Act (IDEA)
- Transition Planning

References and Reading

- Bergeron, D., Murray, B., Shanley, J. L., & Daley, R. (2010). *U.S. Department of Education state of the art conference on postsecondary education for students with intellectual disabilities. Comprehensive transition and postsecondary (CTP) program: The process and lessons learned*. Fairfax: George Mason University.
- College Preparation Checklist: <http://studentaid.ed.gov/PORTALSWebApp/students/english/checklist.jsp>.

Federal student aid: Funding education beyond high school: The guide to federal student aid. http://studentaid.ed.gov/students/publications/student_guide/index.html.

Finkel, J., Anderson, H., & Shanley, J. L. (2010). *U.S. Department of Education state of the art conference on postsecondary education for students with intellectual disabilities. Eligibility of students with intellectual disabilities to receive federal student aid: The FAFSA and more!* Fairfax: George Mason University.

Free Application for Federal Student Aid. <http://www.fafsa.gov>

Heath Resource Center: Online clearinghouse on post secondary education for individuals with disabilities. New postsecondary programs for students with intellectual disabilities.

Student Aid on the Web (<http://www.studentaid.ed.gov>, www.studentaid.ed.gov) – The U.S. Department of Education's federal student aid information website.

<http://www.heath.gwu.edu/>.

References and Reading

- Denham, S. A., Wyatt, T. M., Bassett, H. H., Echeverria, D., et al. (2009). Assessing social-emotional development in children from a longitudinal perspective. *Journal of Epidemiology and Community Health*, 63(Suppl 1), i37–i52.
- Johnson, S., Hollis, C., Hennessy, E., Kochhar, P., et al. (2011). Screening for autism in preterm children: Diagnostic utility of the Social Communication Questionnaire. *Archives of Disease in Childhood*, 96, 73–77.
- Neyman, J., & Pearson, E. S. (1967) [1928]. On the use and interpretation of certain test criteria for purposes of statistical inference, Part I. *Joint Statistical Papers*. Cambridge University Press, pp. 1–66.
- Posserud, M.-B., Lundervold, A. J., & Gillberg, C. (2009). Validation of the autism spectrum screening questionnaire in a total population sample. *Journal of Autism and Developmental Disorders*, 39(1), 126–134.

False Positive

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Synonyms

Alpha (α) error; Type I error; Wrong decision

Definition

A false positive occurs when the null hypothesis is true and rejected erroneously. That is, a difference is observed when there is no true difference. An individual may be diagnosed with a condition, for example, that he does not have. A test that produces a substantial number of false positives (i.e., Type 1 Errors) is unable to systematically rule out disorders and diagnoses, and consequently has poor specificity.

See Also

► [Sensitivity and Specificity](#)

False-Belief Task

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Definition

False-belief task is based on false-belief understanding which is the understanding that an individual's belief or representation about the world may contrast with reality. False-belief task is a frequently used methodology to examine theory of mind (i.e., child's ability to construct people in terms of internal mental states such as their beliefs, Wellman 1993). It is considered as litmus test of theory of mind, in that in such cases, it becomes possible to distinguish unambiguously between the child's (true) belief and the child's awareness of someone else's different (false) belief (Dennett 1978). First-order false-belief tasks involve attribution about other's false belief with regard to real events; whereas, second-order false-belief tasks are related with what people think about other people's thoughts. In second-order false-belief tasks, the child is required to attribute the false belief of one person based on the thoughts of another (Perner and Wimmer 1985). An example of a commonly used first-order false-belief task is the "Smarties," in which the child is required to predict another child's perception about the content of a box

of candies (that actually includes a pencil) (Gopnik and Astington 1988). A commonly used second-order false-belief task is the Perner and Wimmer (1985) “ice-cream van story” (or John and Marry tasks). In this story, Marry is asked to predict John’s thoughts about the location of the ice-cream man. Only Marry knows the actual location (in the school), but John knows about his former intention to stay in the park. Thus, Marry needs to take into consideration John’s thoughts about the location of the ice-cream man, based on the ice-cream man’s original intentions.

See Also

► [Theory of Mind](#)

References and Reading

- Dennett, D. (1978). *Brainstorms: Philosophical essay on mind and psychology*. Montgomery: Harvester Press.
- Gopnik, A., & Astington, J. W. (1988). Children’s understanding of changes in their mental states. *Child Development*, 62, 98–110.
- Perner, J., & Wimmer, H. (1985). John thinks that Mary thinks that: Attribution of second-order beliefs by 5-to 10-year-old children. *Journal of Experimental Child Psychology*, 60, 689–700.
- Wellman, H. M. (1993). Early understanding of mind: The normal case. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds.), *Understanding of other minds: Perspectives from autism* (pp. 10–39). Oxford: Oxford University Press.

Family Accommodation in Autism Spectrum Disorder

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Definition

Ways in which family members, mostly parents, modify their behavior to help a child avoid or

alleviate distress and negative affect associated with mental health problems (Lebowitz and Bloch 2012; Lebowitz et al. 2014).

Historical Background

A sizable body of literature indicates that family accommodation is common among families of children with obsessive-compulsive disorders (OCD) and anxiety disorders. Such accommodation is associated with both proximal and distal negative outcomes. High levels of family accommodation in these populations are associated with increased severity of anxiety or OCD symptoms, poorer psychosocial functioning, and elevated parental distress (Caporino et al. 2012; Lebowitz and Bloch 2012; Lebowitz et al. 2013, 2014; Storch et al. 2007). Additionally, higher levels of family accommodation are predictive of poorer subsequent response to treatment in children with OCD and anxiety disorders (Lebowitz et al. 2016; Kagan et al. 2016). These findings have already yielded clinical benefit through the development of an efficacious parent-based intervention that targets the reduction of family accommodation (Lebowitz et al. *in press*). This work raises the possibility that the study of family accommodation in children with other disorders could yield similar contributions.

Several findings point to a partial phenotypic overlap between autism spectrum disorders (ASD), OCD, and anxiety disorders, in addition to a possible partial overlap in their etiology. The phenotypic overlap exists with regard to the restricted and repetitive behaviors (RRBs) inherent to a diagnosis of ASD. RRBs are defined in the *Diagnostic and Statistical Manual of Mental Disorders 5* (DSM5) (APA 2013) as “restricted, repetitive patterns of behavior, interests, or activities...” Within OCD, compulsions are defined as “repetitive behaviors or mental acts.” While not synonymous, these two concepts bear resemblance to one another (Jacob et al. 2009; Wood and Gadow 2010). In addition to the partial phenotypic overlap, high comorbidity between these diagnostic constructs has been noted, with greater prevalence of anxiety disorders and OCD

in children with ASD than in typically developing children (van Steensel et al. 2011). Children of parents with OCD or ASD are at greater risk of ASD or OCD, respectively (Meier et al. 2015), and the likelihood that an individual will be diagnosed with OCD or anxiety disorders is elevated in relatives of children with ASD (Jacob et al. 2009), pointing to some degree of shared heritability.

Given the above-noted links between OCD, anxiety disorders, and the RRBs of children with ASD, as well as the potential biological links between the disorders, an examination of family accommodation in the context of autism, specifically regarding RRBs, was undertaken.

Current Knowledge

Building on the data related to family accommodation in OCD and anxiety disorders, an examination of family accommodation in the context of RRBs in ASD was deemed worthwhile (Feldman et al. 2019). Applying the concept of accommodation to RRBs, however, is complicated by several factors. Research has suggested the existence of different sub-groups of RRBs, including repetitive motor and sensory behavior, insistence on sameness, ritualistic behavior, compulsive behavior, circumscribed interests, and self-injurious behavior (Honey et al. 2012; Leekam et al. 2011). Notably, different types of RRBs have been linked to different psychiatric symptoms (e.g., anxiety, depression, and oppositional-defiant symptoms; Lidstone et al. 2014; Stratis and Lecavalier 2013). These and other findings (e.g., Leekam et al. 2011) suggest that the various types of RRBs, and perhaps even different specific RRBs within a single sub-group, may differ in both their etiologies and functions.

Furthermore, RRBs may also vary in function and in degree of adaptiveness, indicating that accommodation of these RRBs may be either potentially helpful or detrimental to the child and family system. Certain RRBs may allow the individual to occupy themselves, regulate hyper- or hypo-sensory arousal, or reduce anxiety (Leekam et al. 2011). In some cases, these behaviors can

even provide a source of income and employment (Attwood 2003; Howlin 2003). Other RRBs, however, such as self-injurious behaviors, are unambiguously harmful. Studies link certain types of RRBs, including preoccupation with object parts, sensory interests, and stereotyped motor behaviors, to poorer reasoning skills, lower adaptive functioning at a later age, and increased caregiver stress (Harrop et al. 2016; Troyb et al. 2016). Further complicating the matter, findings suggest that the same RRBs may have differing etiologies and serve different functions for different children (Leekam et al. 2011; Stratis and Lecavalier 2013).

Particularly relevant to the issue of family accommodation in the context of ASD is the role of RRBs in regulating arousal and in alleviating anxiety and distress, similar to the role of compulsive behaviors in OCD (Leekam et al. 2011; Lidstone et al. 2014). It has been proposed that family accommodation of children's OCD symptoms increases the severity of these symptoms over time by enabling avoidance of distress-inducing stimuli and hampering the development of more adaptive strategies for independent regulation of negative arousal (Lebowitz 2013; Storch et al. 2007). To the extent that RRBs might serve to alleviate distress in children with ASD, family accommodation of RRBs may function in a similar manner and could potentially lead to decreased self-regulation.

Prior to Feldman et al. (2019), limited work had been done examining the role of family accommodation in ASD. Russel et al. (2013) examined a small group of adolescents and adults ($n = 23$) with comorbid ASD and OCD who received cognitive behavioral therapy for OCD. In that study, higher reported family accommodation prior to treatment was associated with poorer response to the treatment. Storch et al. (2015) examined the prevalence and correlates of family accommodation of anxiety symptoms in 40 children with ASD and comorbid anxiety disorders. In line with previous findings on family accommodation in anxiety disorders, family accommodation of anxiety symptoms in the context of ASD was found to be highly prevalent and was correlated with the severity of the children's

anxiety symptoms. The same study also found that family accommodation decreased following cognitive behavioral therapy, with the reduction in family accommodation associated with the improvement in the children's anxiety symptoms. Of note, both of the aforementioned studies focused on accommodation of the anxiety or OCD symptoms and did not investigate accommodation of RRBs.

The first study to examine family accommodation of core ASD symptoms, specifically RRBs, piloted the use of the Family Accommodation Scale for Restricted and Repetitive Behaviors (FAS-RRB; Feldman et al. 2019). This study found that accommodation of RRBs is highly prevalent, with 80% of parents engaging in family accommodation at least once a month and 55% reporting daily accommodation. These rates of accommodation are high, but lower than those reported in pediatric OCD (Lebowitz et al. 2016) and anxiety disorders (Lebowitz et al. 2013, 2014, 2016; Thompson-Hollands et al. 2014), as well as those reported for anxiety symptoms in children with ASD (Storch et al. 2015). The most common accommodations of RRBs reported by parents of children with ASD were (1) providing symptom-related items, (2) participating in symptom-related actions, and (3) assisting in the avoidance of symptom-related stimuli. In this study, higher levels of accommodation were strongly associated with higher RRB severity ($r = 0.820, p < 0.001$). Additionally, higher levels of accommodation were associated with poorer communication ($r = -0.258, p = 0.029$) and daily living skills ($r = -0.407, p < 0.001$) in the children, and a majority of parents report feeling distress due to the accommodation. Most parents also reported their children responding aggressively when parents do not accommodate. These findings parallel previous work focused on children with OCD and anxiety disorders (Lebowitz et al. 2013, 2016; Thompson-Hollands et al. 2014).

While certain similarities exist between family accommodation of RRBs in ASD and accommodation of OCD and anxiety disorders, there

are also significant differences. Every child with ASD presents with some RRBs. However, the frequency, severity, and nature of these behaviors vary widely. Some children with ASD experience difficulty primarily in the area of social communication and interaction while presenting with relatively few RRBs. In such cases, parents may have fewer RRBs to accommodate compared with cases in which the RRBs are a more prominent clinical feature. In the context of OCD and anxiety, the accommodation can relate to almost any clinical feature of the disorder. This may partially explain the lower rates of accommodation reported by parents of children with ASD relative to those reported in OCD and anxiety disorders. Additionally, in light of the heterogeneity in form and function of RRBs, it is yet to be determined whether a reduction of accommodation of RRBs would be invariably beneficial or whether clinical recommendations must be informed by a functional assessment of the child's symptoms.

Future Directions

Further research on the directions of associations and the causal links that may exist between accommodation and severity of ASD is required. RRB severity and lower adaptive functioning may lead to increased family accommodation. It is alternatively plausible that, as in OCD and anxiety disorders, family accommodation leads to more severe child symptomatology and to greater functional impairment in the children. A third possibility is that the relationship is bidirectional or moderated by variables not yet taken known. Longitudinal research is critically needed in order to address these questions.

An additional area worthy of attention is the motivation of parents engaging in family accommodation of RRBs. When asked to provide a qualitative description of their child's RRBs, some parents used language expressing a belief that these behaviors were not optional, such as "must" or "have to," in describing both the RRBs

and their own accommodations. As such, parents of children with ASD may be providing accommodations due to their belief that no other alternatives exist. Children's aggressive responses to not being accommodated, reported by a majority of parents, are another likely contributor to the maintenance of such parental accommodations.

Family accommodation may offer a useful target for clinical interventions for individuals with ASD and their parents or families. Future work elucidating the role of accommodation should aim to determine how best to support parents in this context. In both OCD and anxiety disorders, current interventions emphasize the supportive reduction of family accommodation as a path to increasing independent coping in the child (Lebowitz 2015; Kagan et al. 2016; Lebowitz et al. 2018a, b; Salloum et al. 2018). Recent work supports the efficacy of SPACE (Supportive Parenting for Anxious Childhood Emotions), a parent-based intervention that reduces family accommodation of childhood anxiety and OCD. In a recent randomized controlled trial, SPACE was as efficacious as CBT in treating child anxiety disorders, and it led to greater reduction in family accommodation (Lebowitz et al. 2019). A similar approach may be beneficial in cases of ASD. The development and evaluation of such an intervention program targeting the accommodation of RRBs would be novel and could represent an important step forward in autism-specific interventions.

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub.
- Attwood, T. (2003). Understanding and managing circumscribed interests. In M. R. Prior (Ed.), *Learning and behavior problems in Asperger syndrome* (pp. 126–147). New York: Guilford Press.
- Caporino, N. E., Morgan, J., Beckstead, J., Phares, V., Murphy, T. K., & Storch, E. A. (2012). A structural equation analysis of family accommodation in pediatric obsessive-compulsive disorder. *Journal of Abnormal Child Psychology*, 40(1), 133–143.
- Feldman, I., Koller, J., Lebowitz, E. R., Shulman, C., Itzhak, E. B., & Zachor, D. A. (2019). Family accommodation in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49, 1–9.
- Harrop, C., McBee, M., & Boyd, B. A. (2016). How are child restricted and repetitive behaviors associated with caregiver stress over time? A parallel process multi-level growth model. *Journal of Autism and Developmental Disorders*, 46(5), 1773–1783.
- Honey, E., Rodgers, J., & McConachie, H. (2012). Measurement of restricted and repetitive behaviour in children with autism spectrum disorder: Selecting a questionnaire or interview. *Research in Autism Spectrum Disorders*, 6(2), 757–776.
- Howlin, P. (2003). Longer-term educational and employment outcomes. In M. R. Prior (Ed.), *Learning and behavior problems in Asperger syndrome* (pp. 269–293). New York: Guilford Press.
- Jacob, S., Landeros-Weisenberger, A., & Leckman, J. F. (2009). Autism spectrum and obsessive-compulsive disorders: OC behaviors, phenotypes and genetics. *Autism Research*, 2(6), 293–311.
- Kagan, E. R., Peterman, J. S., Carper, M. M., & Kendall, P. C. (2016). Accommodation and treatment of anxious youth. *Depression and Anxiety*, 33(9), 840–847.
- Lebowitz, E. R. (2013). Parent-based treatment for childhood and adolescent OCD. *Journal of Obsessive-Compulsive and Related Disorders*, 2, 425–431.
- Lebowitz, E. R. (2016). Treatment of extreme family accommodation in a youth with obsessive-compulsive disorder. In E. A. Storch, & A. B. Lewin (Eds.), *Clinical handbook of obsessive-compulsive and related disorders* (pp. 321–335). Cham: Springer.
- Lebowitz, E. R., & Bloch, M. (2012). Family accommodation in pediatric obsessive-compulsive disorder. *Expert Review of Neurotherapeutics*, 12(2), 229–238.
- Lebowitz, E. R., Scharfstein, L. A., & Jones, J. (2014). Comparing family accommodation in pediatric obsessive-compulsive disorder, anxiety disorders, and nonanxious children. *Depression and Anxiety*, 31(12), 1018–1025.
- Lebowitz, E. R., Panza, K. E., & Bloch, M. H. (2016). Family accommodation in obsessive-compulsive and anxiety disorders: A five-year update. *Expert Review of Neurotherapeutics*, 16(1), 45–53.
- Lebowitz, E. R., Gee, D. G., Pine, D. S., & Silverman, W. K. (2018a). Implications of the Research Domain Criteria project for childhood anxiety and its disorders. *Clinical Psychology Review*, 64, 99–109.
- Lebowitz, E. R., King, R. A., & Silverman, W. K. (2018b). Anxiety disorders of childhood and adolescence. In M. Ebert, J. F. Leckman, & I. Petrakis (Eds.), *Current diagnosis and treatment psychiatry* (3rd ed, pp. 554–564). New York: McGraw-Hill Education.
- Lebowitz, E. R., Marin, C., Martino, A., Shimshoni, Y., & Silverman, W. K. (2019). Parent-based treatment as efficacious as cognitive-behavioral therapy for

childhood anxiety: A randomized noninferiority study of supportive parenting for anxious childhood emotions. *Journal of the American Academy of Child and Adolescent Psychiatry*.

- Leekam, S. R., Prior, M. R., & Uljarevic, M. (2011). Restricted and repetitive behaviors in autism spectrum disorders: A review of research in the last decade. *Psychological Bulletin*, 137(4), 562.
- Lidstone, J., Uljarević, M., Sullivan, J., Rodgers, J., McConachie, H., Freeston, M., Le Couteur, A., Prior, M., & Leekam, S. (2014). Relations among restricted and repetitive behaviors, anxiety and sensory features in children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 8(2), 82–92.
- Meier, S. M., Petersen, L., Schendel, D. E., Mattheisen, M., Mortensen, P. B., & Mors, O. (2015). Obsessive-compulsive disorder and autism spectrum disorders: Longitudinal and offspring risk. *PloS One*, 10(11), e0141703.
- Salloum, A., Andel, R., Lewin, A. B., Johnco, C., McBride, N. M., & Storch, E. A. (2018). Family accommodation as a predictor of cognitive-behavioral treatment outcome for childhood anxiety. *Families in Society*, 99(1), 45–55.
- Storch, E. A., Geffken, G. R., Merlo, L. J., Jacob, M. L., Murphy, T. K., Goodman, W. K., Larson, M. J., Fernandez, M., & Grabill, K. (2007). Family accommodation in pediatric obsessive-compulsive disorder. *Journal of Clinical Child and Adolescent Psychology*, 36(2), 207–216.
- Storch, E. A., Zavrou, S., Collier, A. B., Ung, D., Arnold, E. B., Mutch, P. J., Lewin, A. B., & Murphy, T. K. (2015). Preliminary study of family accommodation in youth with autism spectrum disorders and anxiety: Incidence, clinical correlates, and behavioral treatment response. *Journal of Anxiety Disorders*, 34, 94–99.
- Stratis, E. A., & Lecavalier, L. (2013). Restricted and repetitive behaviors and psychiatric symptoms in youth with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 7(6), 757–766.
- Thompson-Hollands, J., Kerns, C. E., Pincus, D. B., & Comer, J. S. (2014). Parental accommodation of child anxiety and related symptoms: Range, impact, and correlates. *Journal of Anxiety Disorders*, 28(8), 765–773.
- Troyb, E., Knoch, K., Herlihy, L., Stevens, M. C., Chen, C. M., Barton, M., Treadwell, K., & Fein, D. (2016). Restricted and repetitive behaviors as predictors of outcome in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46(4), 1282–1296.
- Van Steensel, F. J., Bögels, S. M., & Perrin, S. (2011). Anxiety disorders in children and adolescents with autistic spectrum disorders: A meta-analysis. *Clinical Child and Family Psychology Review*, 14(3), 302–317.
- Wood, J. J., & Gadow, K. D. (2010). Exploring the nature and function of anxiety in youth with autism spectrum disorders. *Clinical Psychology: Science and Practice*, 17(4), 281–292.

Family Burden

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Definition

The physical, emotional, and economic impact directly associated with the responsibility of caring for a person with ASD, including the secondary impact on all family members. These can include: (a) the economic cost of care (Montes and Halterman 2008), including lost opportunities for income (Jabrink 2007); (b) quality of life (Lee et al. 2008; Khanna et al. 2010); and (c) the impact on siblings (Harris and Glasberg 2003). Unlike the typical costs associated with child rearing, this burden often extends – and may increase – into adulthood, for those families who elect guardianship. It is reasonable to expect that this burden may vary as a function of the family's race, ethnicity, income, and education, given other findings suggesting disparities in accessing health services, and cultural differences in attitudes toward disability. It is generally believed that various types of supports, including parent training, respite care, and wraparound services, can mitigate some aspects of the family burden.

See Also

- ▶ [Family-Centered Care, Second Edition](#)
- ▶ [Guardianship](#)
- ▶ [Health Disparities](#)
- ▶ [Parent Training](#)
- ▶ [Respite Care](#)
- ▶ [Wraparound Services](#)

References and Reading

- Harris, S. L., & Glasberg, B. A. (2003). *Siblings of children with autism: A guide for families* (2nd ed.). Bethesda: Woodbine House.

- Jabrink, K. (2007). The economic consequences of autistic spectrum disorder among children in a Swedish municipality. *Autism, 11*, 453–463.
- Khanna, R., Madhavan, S. S., Smith, M. J., Patrick, J. H., Tworek, C., & Becker-Cottrill, B. (2010). Assessment of health-related quality of life among primary caregivers of children with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 41*(9), 1214–1227.
- Lee, L. C., Harrington, R., Louie, B., & Newschaffer, C. (2008). Children with autism: Quality of life and parental concerns. *Journal of Autism and Developmental Disorders, 38*(6), 1147–1160.
- Montes, G., & Halterman, J. S. (2008). Association of childhood autism spectrum disorders and loss of family income. *Pediatrics, 121*, e821–e826.

Family Therapy

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Definition

Family-based treatments attempt to decrease interactions between family members that contribute to psychiatric disorders and to increase interactions that protect them from these problems. The psychoeducational model is most commonly used in conjunction with psychiatric disorders. Family life is all about relationships and communication. Autism spectrum disorders are about communication challenges, misunderstanding of social cues, and lack of emotional understanding, thus affecting relationships in families. The family burden for parents and siblings is very high in families with a member on the spectrum, and the divorce rate is also high in families with children who have autism. In marriage, if one of the partners is on the spectrum, there will be more problems than just the normal marriage conflicts.

Collaborative family work is directed to meet the needs of people with autism and their families and should address problems in each developmental phase of the family life cycle. Parents of a young autistic child are perplexed when they realize that the child is not developing properly and

may feel rejected or unimportant when the child does not seek their attention. Getting a diagnosis and services may be stressful, as well as the process of adaptation: accepting the diagnosis and informing other family members and friends. At school age, the family has to adapt in many ways: confrontation with outside world, dealing with aversive reactions of peers, specific ways of rearing, arranging for child care, and dealing with professionals. In adolescence, families have to cope with the chronicity of the child's disability, with issues of aggression, odd behavior, sexuality, and addiction. Parents have to delay the developmental phase of launching and moving on and are confronted with the perspective of endless care.

Marriages with an autistic member have a serious risk of running into trouble. The spouse with ASD misses relational skills, there will be an asymmetry in the relation, and the non-ASD spouse will feel lonely and neglected.

A diagnosis of an autism spectrum disorder is likely to be experienced as ambiguous loss in a family, because resolution of the situation is not possible and the outcome is not predictable, especially when the individual with ASD may give an outward appearance of health, thus raising too high expectations for his functioning.

Sometimes, families have several autistic members, for instance, one or two siblings, or a father and a son both have the condition, complicating family function and rearing practices even more.

Family interventions have been less effective in reducing core ASD symptoms, yet they do contribute to reducing the comorbid family and behavior problems associated with the disorder in children and adolescents. A biopsychosocial approach with a treatment package (e.g., family treatment, behavioral therapy, and pharmacology) is common in most clinical settings. Some models and schools of family therapy should not be applied in families with an autistic member, because they focus primarily on context, meaning, and mentalizing processes. Moreover, family and couples' therapists should adapt their style, taking into account that family members may have problems with imagination, generalizing, and they need basic knowledge about ASD in order to work effectively with these families.

Historical Background

For decades following Kanner's description of the disorder, it was wrongly believed that the parents, especially the mother, were responsible for causing the disorder by emotional neglect. Family psychoeducation started in the 1970s of last century in the treatment of schizophrenia. It was based on the premise that the patient has a brain disorder and that families need to be supported in their care of the mentally ill person. The emphasis on the biological aspect of the illness is intended to correct that families somehow cause the illness.

Family psychoeducation nowadays is commonly used in various chronic childhood and adult illnesses.

Current Knowledge

Nowadays, the association of genetic factors in the etiology of autism spectrum disorders is well established. Problems in parenting and family dysfunction may be a response to the child and adolescent psychopathology. A psychoeducational approach in families is the treatment of choice in clinical practice. It is defined as the systematic administration of information to both patient and family (significant others) about symptoms, diagnosis, treatment, and prognosis. Its aim is behavioral change and not just teaching for the sake of increasing knowledge. It must be given in doses, as the recipients are able to hear it and given repetitively over time. Other goals are improving family skills, positive communication development, and increased social involvement for the family. Family psychoeducation includes the provision of emotional support and resources during periods of transition and crisis.

Future Directions

Early diagnosis and treatment of the child and its family may have positive effects for some children. There is no cure for ASD, yet various treatments are used to treat the effects of the disorder.

See Also

► [Family Burden](#)

References and Reading

- Aston, M. (2004). *Asperger in love: Couples relationships and family affairs*. London: Jessica Kingsley.
- Baker-Ericzén, M. J., Brookman-Frazee, L., & Stahmer, A. (2005). Stress levels and adaptability in parents of toddlers with and without autism spectrum disorders. *Research and Practice for Persons with Severe Disabilities*, 30, 194–204.
- Bolman, W. (2006). *The autistic family life cycle: Family stress and divorce*. Retrieved 15, July 2006 from <http://Asa.confex.com/asa/2006/tehprograms/s1940.htm>
- Carr, A. (Ed.). (2002). *Prevention: What works with children and adolescents?* Sussex: Brunner-Routledge.
- Diamond, G., & Josephson, A. (2005). Family-based treatment research: A 10-year update. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44(9), 872–887.
- Hastings, R. P., Kovshoff, H., Ward, N. J., Degli Espinosa, F., Brown, T., & Remington, B. (2005). Systems analysis of stress and positive perceptions in mothers and fathers of pre-school children with autism. *Journal of Autism and Developmental Disorders*, 35(5), 635–644.
- Keitner, G. I., Heru, A. M., & Glick, I. D. (2010). *Clinical manual of couples and family therapy*. Washington, DC: American Psychiatric Publishing.
- McGoldrick, M., & Carter, B. (2003). The family life cycle. In F. Walsh (Ed.), *Normal family processes* (pp. 375–399). New York: The Guilford Press.
- O'Brien, M. (2007). Ambiguous loss in families of children with autism spectrum disorders. *Family Relations*, 56(2), 135–146.
- Rolland, J. S. (2003). Mastering family challenges in illness and disability. In F. Walsh (Ed.), *Normal family processes*. New York: The Guilford Press.
- Seligman, M., & Darling, B. (2007). *Ordinary families, special children: A systems approach to childhood disability*. New York: The Guilford Press.
- Sicile-Kira, C. (2008). The affects of Autism in Families and in Partner Relationships. *Family Therapy Magazine*, pp. 19–23.
- Solomon, M., Ono, M., Timmer, S., & Goodlin-Jones, B. (2008). The effectiveness of parent–child interaction therapy for families of children on the autism spectrum. *Journal of Autism and Developmental Disorders*, 38(9), 1767–1776.
- Van Voorst, A. J. P. (2008). Relatieproblemen bij personen met een autisme spectrum stoornissen (couple therapy with one partner having an autism spectrum disorder). *Systeemtherapie*, 20(3), 152–164.
- Van Voorst, A. J. P., Jansma, H. B. M., van de Wiel, N. H. M., & Matthys, W. (2005). Kinderpsychiatrische stoornissen en gezinsbelasting (Psychiatric disorders in children and family stress). *Kind en Adolescent*, 25(3), 181–244.

Family-Centered Care, Second Edition

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Synonyms

Family-centered practice; Patient- and family-centered care

Definition

Family-centered care is an approach to care that is based on the understanding that the family is a child's primary source of strength and support (American Academy of Pediatrics 2012). As such, the family is recognized as an integral part of the team in collaboration with service providers to best support the care of the child. Service providers view the family as a unit that includes the child, and this unit is the primary focus of care (Coyne et al. 2018). The family unit may include parents, extended family, and/or other caregivers who are determined to be most meaningful and supportive to the child in time of need (Baas 2012).

Core attributes of family-centered care include collaboration, evidenced by empowering families to participate in care by building partnerships that provide dignity and respect; communication, evidenced by honest and open information-sharing, providing choices, and including families in shared decision-making; negotiation, evidenced by educating and building family confidence in care, sharing expectations of roles and responsibilities with family, and assessing families' needs to provide care that is holistic and individualized; and support, evidenced by providing care that is appropriate to the cultural and social context of the family, including formal and informal support (Coyne et al. 2018).

Family-centered care is mutually beneficial for the child, family, and service providers. Research

has shown that family-centered care leads to improved health outcomes, decreased health-care costs, more effective allocation of resources, and increased child, family, and staff satisfaction (American Academy of Pediatrics 2012). Youth on the autism spectrum who receive family-centered care are also more likely to experience successful transition from pediatric to adult primary care (Rast et al. 2018). Families experience increased coping, adjustment, confidence, and competence as caregivers and decision-makers when family-centered care is utilized. Inclusion of the child as developmentally appropriate encourages the child to take responsibility for his or her own health and assist with transition to adult services (American Academy of Pediatrics 2012; Christon and Myers 2015).

In the care of children on the autism spectrum, families are constant in the child's life and therefore can provide a unique perspective and information to service providers who transition in and out of a child's life when planning, delivering, and evaluating interventions. Including families in creating goals of care and incorporating family-centeredness throughout care is a particularly effective tool in managing medical concerns for children on the autism spectrum.

See Also

- Culture and Autism
- Interdisciplinary Team

References and Reading

- American Academy of Pediatrics Committee on Hospital Care and Institute for Patient- and Family-Centered Care. (2012). Policy statement: Patient- and family-centered care and the pediatrician's role. *Pediatrics*, 129(2), 394–404.
- Baas, L. S. (2012). Patient- and family-centered care. *Heart & Lung: The Journal of Acute and Critical Care*, 41(6), 534–535. <https://doi.org/10.1016/j.hrtlng.2012.08.001>.
- Christon, L. M., & Myers, B. J. (2015). Family-centered care practices in a multidisciplinary sample of pediatric professionals providing autism spectrum disorder services in the United States. *Research in Autism Spectrum*

Disorders, 20, 47–57. <https://doi.org/10.1016/j.rasd.2015.08.004>.

Coyne, I., Holmström, I., & Söderbäck, M. (2018). Centeredness in healthcare: A concept synthesis of family-centered care, person-centered care and child-centered care. *Journal of Pediatric Nursing*, 42, 45–56. <https://doi.org/10.1016/j.pedn.2018.07.001>.

Rast, J. E., Shattuck, P. T., Roux, A. M., Anderson, K. A., & Kuo, A. (2018). The medical home and health care transition for youth with autism. *Pediatrics*, 141(Supplement 4), S328–S334. <https://doi.org/10.1542/peds.2016-4300J>.

Family-Centered Practice

- [Family-Centered Care, Second Edition](#)

Family-Centered Programming

- [Parent-Professional Partnership](#)

Fanatrex

- [Gabapentin](#)

FAST (Functional Assessment Screening Tool)

- [Functional Analysis Screening Tool](#)
- [Functional Assessment Screening Tool \(FAST\)](#)

FAS-Test

- [Verbal Fluency](#)

FazaClo

- [Clozapine](#)

FBA

- [Functional Behavior Assessment](#)

Fear

- [Anxiety](#)

Febrile Convulsions

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Synonyms

[Febrile seizures](#)

Definition

Febrile convulsions are a common type of seizure seen in children between the ages of 6 months and 6 years. These seizures are triggered by fever, (temperature > 38 °C) but with no evidence of encephalitis or meningitis or prior history of afebrile seizures. Children who have febrile seizures typically have an acute intercurrent viral or bacterial illness such as an ear infection or sinus infection. The seizures are not associated with any systemic metabolic abnormalities such as hypoglycemia.

Febrile seizures are commonly categorized as simple or complex. Simple febrile seizures last less than 15 min (typically only last a minute or two) and have no focal features. Two or more simple febrile seizures can occur in succession, but the total duration of the seizures should be less than 30 min. A “complex” febrile seizure is one that has focal features, may last more than 15 min, or, when they occur in a series, may last more than 30 min. Rarely, febrile seizures can present with status epilepticus.

Febrile seizures are a type of “provoked seizure.” Seizures can also be “provoked” or “triggered” by head injury or withdrawal from medications.

See Also

- ▶ [Electroencephalogram \(EEG\)](#)
- ▶ [Epilepsy](#)
- ▶ [Seizures](#)

References and Reading

Patel, N., Ram, D., Swiderska, N., Mewasingh, L. D., Newton, R. W., & Offringa, M. (2015). Febrile seizures. *BMJ*, 351:h4240.

Febrile Seizures

- ▶ [Febrile Convulsions](#)

Fecal Impaction

- ▶ [Constipation](#)

Fecal Incontinence

- ▶ [Encopresis](#)

Federal Rules of Evidence

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Definition

In General

The Federal Rules of Evidence are the rules governing the admissibility of evidence at civil

and criminal trials in federal courts. The Federal Rules of Evidence generally comprise a set of restraints that the courts place on lawyers in an attempt to manage the risks associated with the adversarial process in a trial setting. The Federal Rules of Evidence were adopted in 1975, and as of 2009, 42 states had adopted codes based on the federal model.

Admission of Relevant Evidence

Federal Rule of Evidence 402 provides for the admissibility of relevant evidence unless the rule provides otherwise. Federal Rule of Evidence 403 gives the trial judge discretion to exclude relevant evidence where its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury, or by considerations of undue delay, waste of time, or needless presentation of cumulative evidence. Because exclusion occurs only where the probative value is “substantially outweighed,” the rule favors admissibility.

Witness

Federal Rule of Evidence 601 serves the purpose of removing a considerable number of the common law grounds for disqualifying witnesses. In particular, a section of Federal Rule of Evidence 601 states that “[e]very person is competent to be a witness except as otherwise provided in these rules.” Federal Rule of Evidence 602 requires that before a witness can testify, evidence must support that the witness has personal knowledge and memory of the matter. Thus, one may not testify to knowledge obtained from what others told them.

Expert Witnesses

Under Federal Rule of Evidence 702, only experts may testify on matters that are scientific, technical, or specialized in nature. In *Daubert v. Merrell Down Pharmaceuticals*, the Supreme Court held that (1) “general acceptance” is not necessary precondition to admissibility of scientific evidence under Federal Rule of Evidence and (2) under the Federal Rules of Evidence, the trial judge is assigned the task of ensuring that any and all scientific testimony or evidence admitted is not only relevant but reliable. In *Daubert* and similar

cases, the United States Supreme Court set forth a number of factors to consider in determining whether to admit expert testimony under Federal Rule of Evidence 702. These factors are neither exclusive nor dispositive. For instance, the US Supreme Court stated in *Daubert* that courts may consider whether the theory or technique employed by the expert is generally accepted in the scientific community, whether it has been subjected to peer review and publication, whether it can be and has been tested, whether the known or potential rate of error is acceptable, and the existence and maintenance of standards and controls.

Sources

Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993).

Federal Rules of Evidence (As amended to December 1, 2011)

Federal Rule of Evidence 402. General Admissibility of Relevant Evidence

Relevant evidence is admissible unless any of the following provides otherwise

- the United States Constitution
- a federal statute
- these rules; or
- other rules
- prescribed by the Supreme Court.

Federal Rule of Evidence 403. Excluding Relevant Evidence for Prejudice, Confusion, Waste of Time, or Other Reasons

The court may exclude relevant evidence if its probative value is substantially outweighed by the danger of one or more of the following: unfair prejudice, confusing the issues, misleading the jury, undue delay, wasting time, or needlessly presenting of cumulative evidence.

Federal Rule of Evidence 601. Competency to Testify in General

Every person is competent to be a witness unless these rules provide otherwise. But in a civil case, state law governs the witness's competency regarding a claim or defense for which state law supplies the rule of decision.

Federal Rule of Evidence 602. Need for Personal Knowledge

A witness may testify to a matter only if evidence is introduced sufficient to support a finding that the witness has personal knowledge of the matter. Evidence to prove personal knowledge may, consist of the witness' own testimony. This rules does not apply to a witness's expert testimony under rule 703

Federal Rule of Evidence 702. Testimony by Expert Witnesses

A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise, if: (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue (b) the testimony is based on sufficient facts or data; (c) the testimony is the product of reliable principles and methods; and (d) the expert has reasonably applied the principles and methods to the facts of the case.

References and Reading

- Garner, B. A. (2009). *Black's law dictionary* (9th ed.). St. Paul: West Publishing Company.
- Mueller, C. B., & Patrick, L. C. K. (2009). *Evidence* (9th ed.). New York: Aspen Publishers.

Feedback Error-Related Negativity (fERN)

► [Feedback-Related Negativity](#)

Feedback on Provider Work Performance

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Synonyms

[Employee development](#); [Employee performance](#); [Performance reviews for direct care staff](#); [Staff training and development](#)

Definition

Refers to the various methods used to encourage personnel working with individuals with autism spectrum disorders to improve the strategies they use in their everyday interactions, teachings, and supports they provide. Though various methods have been tested (Brown et al. 1981; Quilitch 1975), it was found that the most successful method for increasing work performance was a combination of feedback and praise based upon the ability of the staff member to assist the individual under their care meet their specific goals/outcomes (Kreitner et al. 1977; Montegar et al. 1977).

See Also

- [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- Brown, K. M., Willis, B. S., & Reid, D. H. (1981). Differential effects of supervisor verbal feedback and feedback plus approval on institutional staff performance. *Journal of Organizational Behavior Management*, 3, 57–68.
- Kreitner, R., Reif, W. E., & Morris, M. (1977). Measuring the impact of feedback on the performance of mental health technicians. *Journal of Organizational Behavior Management*, 1, 105–109.
- Montegar, C. A., Reid, D. H., Madsen, C. H., & Ewell, M. D. (1977). Increasing institutional staff-to-resident interactions through in-service training and supervision approval. *Behavior Therapy*, 8, 533–540.
- Quilitch, H. R. (1975). A comparison of three staff management procedures. *Journal of Applied Behavior Analysis*, 7, 217–221.

Feedback-Related Negativity

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Synonyms

[Feedback error-related negativity \(fERN\)](#); [Medial frontal negativity \(MFN\)](#)

Definition

The feedback-related negativity or FRN is an electrical brain signal measured with an electroencephalogram. Detectable at the scalp via the event-related potential (ERP), the FRN occurs when an individual receives external feedback (visual, auditory) indicating that performance is worse than expected in a given context. Such contexts include monetary loss and feedback about performance in simple games. The FRN occurs approximately 250 ms after feedback and is typically observed at central to frontal-central scalp regions. The most likely neural generator of the FRN is the anterior cingulate cortex (Gehring and Willoughby 2002; Luu et al. 2003).

See Also

- [Anterior Cingulate](#)
- [Cingulate Cortex](#)
- [ERN](#)
- [Error-Related Negativity](#)

References and Reading

- Gehring, W. J., & Willoughby, A. R. (2002). Medial prefrontal cortex and rapid processing of monetary gains and losses. *Science*, 295, 2279–2282.
- Luu, P., Tucker, D. M., Derryberry, D., Reed, M., & Poulsen, C. (2003). Electrophysiological responses to errors and feedback in the process of action regulation. *Psychological Science*, 14, 47–53.
- Nieuwenhuis, S., Holroyd, C. B., Mol, N., & Coles, M. G. (2004). Reinforcement-related brain potentials from medial frontal cortex: Origins and functional significance. *Neuroscience and Biobehavioral Reviews*, 28(4), 441–448.

Feeding Disorder

- [Pica](#)

Feeding Problems

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Definition

Feeding problems are delays and/or disorders in the development of eating and drinking skills including disordered placement of food in the mouth, difficulty in appropriately manipulating food when it is in the mouth, difficulty chewing, and/or difficulty swallowing (American Speech-Language-Hearing Association 2001). Feeding problems also include deficits in “any aspect of taking nutritional elements that result in under nutrition, poor growth or stressful mealtimes for children and their caregivers”.

Historical Background

In the early 1900s, doctors were cognizant of the fact that behaviors such as vomiting could be a symptom of feeding problems including overfeeding or too frequent feeding, medical conditions such as pyloric obstruction, and feeding of improper foods (Lowenburg 1912). By 1935, clinicians were addressing feeding problems that occurred as the result of structural anomalies with equipment such as the modified Asepto syringe and medicine dropper. In fact, the Brecht feeder was specifically developed to assist in feeding infants with structural abnormalities and/or physical and mental handicaps (Hess 1946). Feeding problems began to appear in physicians' descriptions of developmental disabilities, such as cerebral palsy in the 1950s (Ingram 1954; Perlstein and Barnett 1952). During this time, it was suggested that feeding problems be classified into one of two groups: organic feeding problems and feeding problems in children who are emotionally disturbed and show tension and anxiety (Shwartz 1958).

By the 1970s, reports suggested that feeding problems occurred frequently in preschool

handicapped children and were a “significant clinical entity” (Palmer et al. 1975). However, no system was consistently being used to classify these difficulties; a problem that continues to exist today. Feeding problems during this time were attributed to neuromotor dysfunction or physical abnormalities. In cases where neuromotor dysfunction and physical abnormalities were ruled out, the feeding problems were determined to have a behavioral cause (Thompson and Palmer 1974). Interventions to address feeding problems included techniques designed to treat feeding problems caused by mechanical difficulties (as cited by Thompson & Palmer). These techniques often included positioning recommendations aimed at facilitating movement and/or prescribed exercises to normalize tone and oral sensitivity. Treatment of behavioral feeding problems often included psychotherapy, using hunger as a means to motivate the child to eat a specific food and gradually increasing texture as a means to introduce solid foods into a child's diet, and/or behavioral modification strategies (Benotvin 1970; Bernal 1972; Jones 1982; Thompson and Palmer 1974; Palmer et al. 1975).

Diagnosis and treatment of feeding problems is still in its infancy. The treatment strategies used during the 1960s and 1970s are similar to those used now (for a review of early strategies, see Palmer et al. 1975). One of the most significant changes in treatment of feeding problems since the 1970s is awareness that feeding is a give-and-take experience that relies on both the child and caregiver. Thus, treatment today often includes consideration of child-caregiver interactions during mealtimes.

Current Knowledge

Successful feeding involves (1) acceptance of a wide variety of developmentally appropriate foods and (2) development of motor skills that enable efficient and safe sucking, chewing, propelling, and swallowing. However, normal feeding development is not restricted to the physical act of eating. It also includes the successful integration of a range of physical functions and interpersonal relationships. Both physical factors

(e.g., integration of swallowing and respiration, hand-eye coordination, normal posture, and tone development) and social factors (e.g., cultural patterns and social factors within a family) influence feeding development (Arvedson and Brodsky 2002). Disruption in one or more of these areas can result in a feeding problem (Bryant-Waugh et al. 2010).

Feeding problems are defined as delays and/or disorders in the development of eating and drinking skills. This includes the motor aspects of eating (e.g., difficulty chewing and difficulty swallowing) as well as deficits in taking nutritional elements, which causes poor growth or stressful mealtimes (American Speech-Language-Hearing Association 2001). Feeding problems manifest themselves in a variety of ways, ranging from an inability to swallow without choking to restricted diets. Approximately 25–40% of infants and toddlers are reported to have feeding problems (Reau et al. 1996). Many early feeding problems are transient and resolve without significant clinical intervention (Bryant-Waugh et al. 2010). However, others fail to resolve without intervention. These feeding disorders often have multifactorial causes, which may include structural abnormalities, and a substantial behavioral component (Arvedson and Brodsky 2002; Bernard-Bonnin 2006).

A consistent classification system is not yet used for feeding problems. The Diagnostic and Statistical Manual of Mental Disorder-4th Edition (DSM-IV) classifies feeding disorders into three diagnostic categories: feeding disorders, pica, and rumination disorders. However, a number of eating disorders commonly seen in early childhood have no place in the DSM-IV classification system (Bryant-Waugh et al. 2010). The organic and nonorganic dichotomy has also been used to classify feeding problems. Organic feeding problems occur as a result of structural anomalies, neuromuscular issues, or other known physiological causes (e.g., gastroesophageal reflux), whereas nonorganic feeding problems occur due to disruptive environments or have emotional underpinnings. While this dichotomy is helpful, feeding problems often have more than one cause (for a review, see Burklow et al. 1998). The American Speech-Language-Hearing Association has

adopted a more in-depth classification system (American Speech-Language-Hearing Association 2007). While this classification may prove to be more useful than the organic/nonorganic dichotomy, it does not address the issue that most children with feeding problems often have multiple diagnoses (Arvedson and Brodsky 2002) and, as such, may fall under more than one category. For example, organic factors may disrupt the typical development of eating which results in the development of maladaptive behavioral patterns by the child and/or the caregiver (Burklow et al. 1998).

Given the complexity of many feeding problems, an interdisciplinary approach to addressing these problems is recommended. Interdisciplinary feeding teams may include a speech-language pathologists, developmental pediatrician, otolaryngologist, gastroenterologist, nutritionist/dietitian, occupational therapist, and social worker or nurse. Speech-language pathologists conduct feeding and swallowing evaluations and develop feeding plans. Developmental pediatricians provide diagnoses of pediatric and neurodevelopmental disabilities and manage overall patient care. Otolaryngologists examine the integrity of the structural mechanisms of swallowing and manage medical and surgical treatment of issues related to drooling, aspiration, and gastroesophageal reflux. Gastroenterologists conduct gastrointestinal evaluations and manage gastrointestinal diseases. The nutritionist/dietitian assesses and manages the child's nutritional needs, and the occupational therapist evaluates the child's posture, tone, and sensory system. Social workers or nurses coordinate home care and other appointments (Arvedson and Brodsky 2002).

Regardless of who is on the team, diagnosis and management of feeding problems follow the same steps: (1) define the feeding and/or swallowing problem, (2) identify etiology(ies), (3) determine appropriate diagnostic tests, (4) plan approach to patient/family, (5) teach about problem, (6) implement treatment, monitor progress, and (7) evaluate progress (Arvedson and Brodsky 2002). Determining the underlying etiology is a critical aspect of any successful intervention program because disturbances in feeding and eating behavior with similar clinical presentations

may differ in their underlying etiologies and require different interventions (Bryant-Waugh et al. 2010). Certain medical conditions and developmental disabilities are associated with specific feeding problems. Neurological conditions and anatomical anomalies are most often associated with skills deficits such as oral motor delays, whereas medical conditions such as gastroesophageal reflux are most often associated with food refusal among children with Down syndrome, autism, and cerebral palsy (Field et al. 2003; Williams et al. 2005).

Feeding programs often have multiple components. For example, programs may address oral sensorimotor issues and posture and positioning during feeding. Oral sensorimotor programs are designed to address deficits in the structures that support feeding and swallowing. Goals of these programs may include improving jaw, lip, and/or tongue movements; developing coordinated movements of the mouth; and normalizing sensory oral experiences during mealtimes. Therapy may also be aimed at developing transitional feeding skills including spoon-feeding, cup-drinking, straw-drinking, and chewing. In addition to targeting skills that directly support feeding, interventionists may also address positioning and posture. Positioning is especially important for children with abnormal muscle tone. Management of behavioral feeding problems may focus on weight gain, initiation of oral feedings, weaning from tube feeds, increased oral intake, improved cooperation and reduced stress during mealtimes, and/or acceptance of a wider variety of flavors and textures (Arvedson and Brodsky 2002). Because mealtimes offer the opportunity for parent-child interactions, it is important that management of feeding and swallowing problems occurs within the context of social and communicative activities associated with mealtime for a parent and child (Arvedson and Brodsky 2002).

Future Directions

The field of feeding problems is still in its infancy, especially with regard to feeding and swallowing

problems in children (Arvedson and Brodsky 2002). Although research in the area of feeding and typical swallowing and swallowing disorders has increased, continued research is needed. In the field of feeding problems, there are still inconsistencies in terminology and the detail of description of a number of eating problems, which hampers research. Thus, it is critical that an internationally recognized and accepted classification is established to inform clinical interventions for particular disorders and move the field forward (Bryant-Waugh et al. 2010).

To date, few research studies have attempted to examine prognosis, course, outcome, and treatment response in feeding disorders using a formal, widely accepted classification system. There is little evidence to guide clinical determination of what constitutes a clinically significant feeding problem and to help clinicians distinguish feeding problems that are more serious from those that are likely to be short lived. In order to better assess prognosis and outcome, researchers should employ a set of consistently used standardized assessment measures (Bryant-Waugh et al. 2010). In addition to directly examining feeding problems, research should continue to examine the outcomes of feeding assessment and intervention in schools and the overall impact on education. Finally, there needs to be an expansion of education programs focused on delivery of feeding and swallowing services in the school (American Speech-Language-Hearing Association 2007).

See Also

- ▶ [Gastrointestinal Disorders and Autism](#)
- ▶ [Nutritional Interventions](#)
- ▶ [PICA](#)

References and Reading

- American Speech-Language-Hearing Association. (2007). *Guidelines for speech-language pathologists providing swallowing and feeding services in schools [Guidelines]*. Available from <http://www.asha.org/policy>
- American-Speech-Hearing-Association. (2001). *Roles of speech-language pathologists in swallowing and feeding disorders: Technical report*. Retrieved 4 Mar 2011

from <http://www.asha.org/docs/html/TR2001-00150.html>

- Arvedson, J. C., & Brodsky, L. (2002). *Pediatric swallowing and feeding: Assessment and management second edition*. Clifton Park: Thompson Delmar Learning.
- Benotvin, A. (1970). A clinical approach to feeding disorders of childhood. *Journal of Psychosomatic Research*, 14, 267–276.
- Bernal, M. E. (1972). Behavioral treatment of a child's eating problem. *Journal of Behavior Therapy and Experimental Psychiatry*, 3, 45–50.
- Bernard-Bonnin, A. (2006). Feeding problems in infants and toddlers. *Canadian Family Physician*, 52, 1247–1251.
- Bonafede, J. P., Lavertu, P., Wood, B. G., & Eliachar, I. (1997). Surgical outcome in 87 patients with Zenker's diverticulum. *Laryngoscope*, 107, 720–725.
- Bryant-Waugh, R., Markham, L., Kreipe, R. E., & Walsh, T. B. (2010). Feeding and eating disorders in childhood. *International Journal of Eating Disorders*, 43, 98–111.
- Burklow, K. A., Phelps, A. N., Schultz, J. R., McConnell, K., & Rudolph, C. (1998). Classifying complex pediatric feeding disorders. *Journal of Pediatric Gastroenterology and Nutrition*, 27, 143–147.
- Field, D., Garland, M., & Williams, K. (2003). Correlates of specific childhood feeding problems. *Journal of Pediatrics and Child Health*, 39, 299–304.
- Hess, J. G. (1946). A modification of the Brecht feeder. *The Journal of Pediatrics*, 29, 233–234.
- Ingram, T. T. S. (1954). A study of cerebral palsy in the childhood population of Edinburgh. *Archives of Diseases in Childhood*, 30, 85–98.
- Jones, W. (1982). Treatment of behavior-related eating problems in retarded students: A review of the literature. *Monographs of the American Association on Mental Deficiency*, 5, 3–26.
- Lowenburg, H. (1912). The significance and treatment of vomiting in infants and young children. *Journal of American Medical Association*, 3, 180–183.
- Palmer, S., Thompson, R. J., & Linscheid, T. R. (1975). Applied behavior analysis in treatment of childhood feeding problems. *Developmental Medicine and Child Neurology*, 17, 333–339.
- Perlstein, M. A., & Barnett, H. E. (1952). Nature and recognition of cerebral palsy in infancy. *Journal of the American Medical Association*, 148, 1389–1397.
- Reau, N. R., Senturia, Y. D., Lebailly, S. A., & Christoffel, K. K. (1996). Infant and toddler feeding patterns and problems: Normative data and a new direction. *Journal of Developmental and Behavioral Pediatrics*, 17, 149–153.
- Shwartz, S. (1958). Eating problems. *Pediatric Clinics of North America*, 5, 596–611.
- Thompson, R. J., & Palmer, S. (1974). Treatment of feeding problems – A behavioral approach. *Journal of Nutrition Education*, 6, 63–36.
- Williams, K. E., Bridget, B. G., & Schreck, K. A. (2005). Comparing selective eaters with and without developmental disabilities. *Journal of Developmental and Physical Disabilities*, 17, 299–309.

Feingold Diet

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Definition

Feingold diet is a dietary intervention popularized for symptoms of inattention, impulsivity, and hyperactivity by Benjamin Feingold, M.D., a pediatric allergist who clinically observed improvement in behavior in his patients for whom he prescribed dietary restrictions for management of symptoms of allergy in the 1960s. It calls for the elimination of synthetic food colorings and synthetic preservatives made from petroleum-based products, synthetic sweeteners, and foods containing high levels of natural salicylates (related to the chemical compound in aspirin).

The Feingold diet eliminates:

- Synthetic food coloring made from petroleum products (e.g., Red #40; Yellow #5)
- Synthetic food preservatives made from petroleum products (e.g., BHT-butylated hydroxytoluene)
- Foods that may be high in natural salicylates (e.g., apples, almonds, berries, cucumbers, grapes, and oranges, among other fruits and vegetables)
- Artificial sweeteners (e.g., aspartame)

Foods could be reintroduced as tolerated. Later modifications to the diet included removal of the preservatives Sodium Benzoate and BHA (butylated hydroxyanisole).

While other dietary approaches to hyperactivity have become popular since the initial dissemination of the Feingold diet, the additional assertions that sugar, yeast, milk, or other foods result in behavioral change were not part of the Feingold diet.

Historical Background

It was initially reported in the 1940s that some foods high in natural salicylates and FD and C Yellow #5 (a food coloring approved for food, drug, and cosmetics) produced similar allergic symptoms. Dr. Feingold was the chief of pediatrics of Cedar Sinai Medical Center in Los Angeles and subsequently the Chief of Allergy for Kaiser Permanente Medical Center in San Francisco. In 1965, Dr. Feingold prescribed a diet low in foods containing salicylates to a patient with hives whose psychiatric symptoms subsequently improved (Feingold 1985). While he initially called the regimen for management of hyperactivity the K-P Program standing for Kaiser Permanente, it became known in the press as the Feingold diet or program. He clinically observed that the dietary regimen of removal of foods he believed contained high levels of natural salicylates and contained synthetic food dyes and sweeteners resulted in subjective behavioral improvement in up to 30–50% of his patients. By 1979, he further recommended elimination of synthetic preservatives with subjective reports of improvement in 60–70% of his patients. While Dr. Feingold recommended limiting sweets, he at no time recommended elimination of sugar in the diet. His concern was about the additives in processed foods and recommended that families make treats and sweets from scratch that should be provided as part of a healthy diet.

He presented his observations regarding food and food additives and behavior to the American Medical Association in 1973. Much controversy ensued, with small clinical trials both reporting improvement in children on the restricted diet and other studies not being able to confirm these findings (Mattes and Gittelmann 1981). The National Institutes of Health convened a consensus development conference in 1983 to examine the data concerning defined diets and hyperactivity. The expert panel concluded that the data were insufficient to recommend defined diets for the treatment of hyperactivity but allowed that a diet trial could be considered by families after appropriate neurobehavioral evaluation and diagnosis took place with consideration of evidence-based

options (National Institutes of Health Consensus Development Panel 1983). Since that time, additional studies have been undertaken examining the behavioral effects of component parts of the Feingold diet. Wolraich et al. demonstrated that the artificial sweetener aspartame (Wolraich et al. 1994) does not impact attention. Studies on the effects of synthetic food coloring have been more controversial (reviewed in Schab and Trinh 2004). Synthetic food coloring use is restricted in European countries, but the scientific data have been interpreted to allow for continued use in the USA (Advisory Committee (March 30–31, 2011)).

Dr. Feingold's book – *Why is Your Child Hyperactive?* – was last published in 1985. The Feingold Foundation maintains an active society to support families who elect to pursue this dietary intervention (www.feingold.org).

Rationale or Underlying Theory

The original theory proposed for behavioral improvement with the Feingold diet was related to the elimination of allergens, leading to improved attention, learning, and behavior. Proponents point toward the introduction of increasing amounts of artificial flavors and colors in the diets of children as paralleling the increase in diagnosis of Attention Deficit Hyperactivity Disorder (ADHD). A hypothesis related to inhibition of neurotransmitter uptake of dopamine by FDC Red #3 was proposed but not confirmed in subsequent animal and human studies (National Institutes of Health Consensus Development Panel 1983). The lack of documentation of benefit in clinical trials and biologic mechanisms to explain clinical observations impacted the FDA decision to continue to allow synthetic food coloring (Advisory Committee (March 30–31, 2011)).

Goals and Objectives

The Feingold diet eliminates specific types of foods in the diet reported to be high in natural salicylates and processed foods containing

synthetic food colorings, sweeteners, and preservatives to promote behavioral improvement in attention, hyperactivity, irritability, and learning. Dr. Feingold managed over 600 of his patients with this approach and reported that up to two-thirds of the children improved. He reported that many children could be taken off of stimulant medications used to treat symptoms of ADHD once the diet was initiated. Dr. Feingold advised Connors et al. (1976) in the first clinical trial testing his hypotheses. Prospective studies attempted to examine the behavioral effects of the Feingold diet, artificial sweeteners, and/or food dyes and preservatives on the behavior of children in the learning laboratory, the classroom, and the home. Studies were designed to examine both behavioral change with implementation of the diet and the response to double-blind placebo-controlled challenges of specific agents. The studies are difficult to compare because of varied design features and the different goals and outcome measures of the individual studies. Some address only components of the diet (e.g., aspartame, specific food dyes). There is no consistency to the dosage of exposure of synthetic food coloring across studies.

Treatment Participants

None of the published studies specifically evaluates the utility of the Feingold diet for children with ASD. The studies evaluate children with ADHD, children with symptoms of hyperactivity, and/or community samples. There are several different sets of studies that examined components of the Feingold program that all approached identification of the study population in different ways. Many selectively recruited children whose families made the observation that their child improved behaviorally on the dietary intervention (Schab and Trinh 2004). Because of the difficulty in maintaining a restricted diet and cost of clinical trials, the double-blind placebo-controlled studies were all of relatively small size.

Studies that set out to evaluate the effect of the Feingold diet including all four components included the original trial by Connors et al.

(1976) that compared 15 children with ADHD to themselves in a 1-month crossover design. A crossover design was used to assess 36 children with hyperactivity without medication by Harley et al. (1978). While not the original Feingold diet, oligoantigenic diets where a few nonprocessed foods are initially permitted then other foods are sequentially added back have been studied more recently (Pelser et al. 2010) in children with behavioral symptoms.

Other studies examined the effects of sucrose or artificial sweeteners like aspartame on behavior. Wolraich et al. (1994) demonstrated no effect on cognition or attention from double-blind placebo-controlled challenges of aspartame in 25 typical preschool children and 23 school-aged children who were reported to have behavioral effects with sugar. In another report, Wolraich et al. (1995) summarized studies examining the effect of sugar on attention that included a total of 535 children. No significant effect was sustained across studies.

A third set of studies had as their goal the assessment of the behavioral effects of food dyes and preservatives on children. Mattes and Gittelman (1981) reviewed the literature and reported on 11 children with symptoms of ADHD who were recruited as responders to the Feingold diet. A meta-analysis of studies including 136 participants in randomized double-blind placebo-controlled studies that examined the effects of artificial food coloring was published by Schab and Trinh (2004). McCann et al. (2007) examined the behavioral response to two different concentrations of synthetic food dyes and sodium benzoate in two groups of children: 153 3-year-olds and 144 8- and 3-year-olds drawn from a diverse community sample and not selected for hyperactivity. Lok et al. (2013) compared the effect on attention in 130 8-year-old children in Hong Kong randomized to food coloring, sodium benzoate, or placebo delivered by capsule. There was no difference between groups studied.

A fourth type of study investigating the effect of increasing the nutritional value of foods provided a large population of public school students that included provision of foods with fewer synthetic ingredients (Schoenthaler et al. 1986).

Treatment Procedures

The study procedures are varied.

Diet introduction and maintenance: Most studies introduced the diet as part of the study or evaluated children already on the diet. Harley et al. (1978) provided all the foods to the families for the child under study to ensure that the diet was stringently followed. Some studies restricted other medications or supplements the children ingested. None formally analyzed and reported on the dietary sufficiency of the diet consumed or looked for other nutritional effects on behavior, although several commented on the overall sufficiency of the diet.

Dietary exposure to challenges: The studies that used a double-blind placebo-controlled approach to examine the behavioral effect of exposures typically provided a snack or beverage that contained synthetic food coloring, preservative, and/or aspartame at a standard time or times through the day. Some provided for repeat daily exposure for a week or more; others provided single challenges. At least one study examined the effect of time of day. Allowance for washout was not typically addressed and might impact the results of otherwise carefully done trials (Schab and Trinh 2004).

Measurement of change in behavior: Outcome measures will be discussed below. The study designs included subjective rating scales from parents, teachers, and research team members; laboratory tests of attention and learning; and objective measures of behavior largely in class settings.

Efficacy Information

The report of the NIH consensus development panel in 1982 was an accurate summary of the evidence available at that time and remains an appropriate summary of the efficacy information accrued over the subsequent 35 years:

- There are clinical reports of children whose parents observe that dietary restriction improved symptoms of hyperactivity.
- The scientific literature overall does not demonstrate a statistically significant improvement

in attention or hyperactivity as reported by laboratory tasks of inattention and impulsivity, clinician ratings, or teacher ratings with the defined diets that eliminate food dyes and food preservatives or alter the fruit and vegetable composition of the diet.

- There may be an impact on behavior in parent rating scales related to food dye exposure. It is unknown if this may be related to specific food dyes and preservatives, exposure, and/or age.
- Aspartame does not impact attention or hyperactivity.
- A defined diet should only be considered after diagnostic evaluation for ADHD and the implementation of educational and behavioral interventions. Families considering a trial of dietary intervention should have adequate information to weigh the potential benefit from conventional medications and should discuss their plan with their health-care provider. Data collection regarding change in target behaviors will help families determine if their behavioral goals are met by this intervention.

No controlled trials on the effects of the Feingold Diet have been completed in children with autism spectrum disorders. A recent review of dietary treatments by Millichap and Yee (2012) reiterates that dietary intervention may be preferred by some families but does not have the scientific support to endorse general use.

Outcome Measurement

The response to dietary interventions across studies is difficult to compare because different measures were used that capture different aspects of behavioral response.

Subjective response as reported by parents: Studies used a variety of parent report measures designed to capture the core symptoms of ADHD: impulsivity, inattention, and motor hyperactivity. The Conners scales have been validated and widely used by researchers studying hyperactivity (Schab and Trinh 2004). Other investigators compromised the interpretation of their studies

by developing their own measures that were not well validated (reviewed in Schab & Trinh).

Subjective response as reported by teachers: Teacher reports using the Conner's scales were common to many studies. This questionnaire is well validated and was widely used in the time period that many of these studies were completed. Schab and Trinh (2004) point out that teachers often rate children as more hyperactive than parents because of the tasks required during the school day.

Laboratory measures of attention and activity: Laboratory measures of vigilance and attention were used in several studies. Tests such as the Conner's Continuous Performance Test II were used by investigators to provide data on sustained attention and response inhibition on standard tasks that are impacted by inattention, impulsivity, and hyperactivity in children with ADHD.

Combination of outcome measures: While most investigators reported on individual measures by examiner, McCann et al. (2007) created a Global Hyperactivity Index to capture the results of the laboratory attention task, classroom observation and rating, and parent rating in a single score. The participants in their study were not selected for symptoms of hyperactivity and represented a community sample.

Measurement of behaviors other than ADHD: Few studies examined behaviors other than those related to attention and hyperactivity. Rowe and Rowe (1994) attempted to capture parent rating of irritability and sleep difficulties which were elevated in their report on exposure to food coloring. When parent report data are separated from the data collected by schools and clinicians, exposure to synthetic food coloring may be associated with increased behavioral symptoms (McCann et al. 2007; Schab and Trinh 2004). It may be that behavioral effects other than ADHD core symptoms are captured in the parent report data.

Qualifications of Treatment Providers

The advocates of this diet suggest that there is no harm in the removal of synthetic food colorings and preservatives and artificial sweeteners by the provision of a diet containing fewer processed foods.

The Feingold Foundation publishes a list of common products that meet their specifications, so implementation of the diet should be neither expensive nor onerous. Removal of a large number of the fruits and vegetables consumed in the American diet might have a nutritional impact if the necessary nutrients are not consumed in other forms. This has not been investigated. The diet recommends substitution of other foods such as bananas, cashews, and pears that the Feingold Foundation suggests are lower in natural salicylates. Reading food labels for processed foods for the presence of food dyes and preservatives may be challenging. Families who are considering a trial of dietary intervention should consult with their primary health-care provider. It may be helpful for families to be referred to a Registered Dietitian, a licensed professional with training that helps them understand the nutritional content of foods, for counseling regarding diet change and how to provide an adequate diet if supplemented, processed foods are eliminated. Not all fruits and vegetables are nutritionally equal, so children with food selectivity might require targeted supplementation.

A family considering a dietary intervention might also consult with their child's educational or behavioral providers to collect data on the behaviors they wish to change. This may be very helpful in determining if there is a behavioral effect that is clinically significant enough to continue intervention. Since the dietary intervention targets symptoms of ADHD, formal rating scales for ADHD in use in the school setting could be considered.

See Also

- Aberrant Behavior Checklist
- Gluten-Free Diet

References and Reading

Advisory Committee. (March 30–31, 2011). Background document for food advisory committee: Certified color additives in food and possible association with attention deficit hyperactivity disorder in children. <http://www.fda.gov/downloads/AdvisoryCommittees/CommitteesMeetingMaterials/FoodAdvisoryCommittee/UCM248549.pdf>.

- Conners, C. K., Goyette, C. H., Southwick, D. A., Lees, J. M., & Andralous, P. A. (1976). Food additives and hyperkinesis: A controlled double blind experiment. *Pediatrics*, 58(2), 154–166.
- Feingold, B. F. (1985). *Why is your child hyperactive?* New York: Random House. ISBN 0-394-73426-2.
- Harley, J. P., Ray, R. S., Tomasi, L., Eichman, P. L., Matthews, C. G., Crun, R., Cleeland, C. S., & Transmar, E. (1978). Hyperkineses and food activities: Testing the Feingold hypothesis. *Pediatrics*, 61(6), 18–28.
- <http://www.feingold.org/overview.php>
- Lok, K. Y., Chan, R. S., Lee, V. W., Leung, P. W., Leung, C., Leung, J., & Woo, J. (2013). Food additives and behavior in 8- to 9-year-old children in Hong Kong: A randomized, double-blind, placebo-controlled trial. *Journal of Developmental and Behavioral Pediatrics*, 34(9), 642–650. PMID: 24217026.
- Mattes, J. A., & Gittelman, R. (1981). Effects of artificial food colorings in children with hyperactive symptoms. *Archives of General Psychiatry*, 38, 714–718.
- McCann, D., Barrett, A., Cooper, A., Grumpler, D., Daler, L., Grimshaw, K., Kitanin, E., Lok, K., Porteaes, L., Prince, E., Sonuga-Barke, E., Warner, J. O., & Stevenson, J. (2007). Food additives and hyperactive behavior in 3 year old and 8/9 year old children in the community: A randomized, double blinded, placebo controlled trial. *Lancet*, 5, 5.
- Millichap, J. G., & Yee, M. M. (2012). The diet factor in attention deficit hyperactivity disorder. *Pediatrics*, 129(2), 330–337. <https://doi.org/10.1542/peds.2011-2199>. Epub 2012 Jan 9. PMID: 22232312.
- National Institutes of Health Consensus Development Panel. (1983). National Institutes of Health consensus development conference statement: Defined diets and childhood hyperactivity. *American Journal of Clinical Nutrition*, 37, 161–165.
- Pelser, L. M., Frankena, K., Buitelaar, J. K., & Rommelse, N. N. (2010). Effects of food on physical and sleep complaints in children with ADHD: A randomized controlled pilot study. *European Journal of Pediatrics*, 169, 1129–1138.
- Rowe, K. S., & Rowe, K. J. (1994). Synthetic food coloring and behavior: A dose response effect in a double blind placebo controlled repeated measures study. *Journal of Pediatrics*, 125(50+1), 691–698.
- Schab, D. W., & Trinh, N. D. (2004). Do artificial food colors promote hyperactivity in children with hyperactive syndromes? A meta analysis of double blind placebo controlled trials. *Journal of Developmental and Behavioral Pediatrics*, 25, 423–443.
- Schoenthaler, S. J., Doraz, W. E., & Wakefield, J. A. (1986). The impact of a low food additive and sucrose diet on academic performance in 803 New York City public schools. *International Journal of Biosocial Research*, 8(2), 185–195.
- Weber, W., & Newmark, S. (2007). Complementary and alternative medical therapies for attention deficit/hyperactivity disorder and autism. *Pediatric Clinics of North America*, 54, 987–1006.
- Wender, E. H. (1986). The food additive free diet in the treatment of behavior disorders: A review. *Journal of Developmental and Behavioral Pediatrics*, 7(1), 35–42.
- Wolraich, M. L., Lindgren, S., Stumbo, P., Stegink, L. D., Applebaun, I., & Kiritsy, M. L. (1994). Effects of diets high in sucrose or aspartame on the behavior and cognitive performance of children. *The New England Journal of Medicine*, 330, 301–307.
- Wolraich, M. L., Wilson, D. B., & White, J. W. (1995). The effect of sugar and behavior or cognition in children. *Journal of the American Medical Association*, 274(20), 1617–1621.

Fenfluramine

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Synonyms

[Pondimin](#)

Definition

Fenfluramine is a drug with an incompletely understood mechanism of action. It was originally introduced for the treatment of obesity. It promotes the release of serotonin acutely but may have a serotonin-depleting effect over time. Given the replicated finding that at least a subgroup of children with autism are *hyperserotonergic*, investigators considered fenfluramine for the treatment of autism. Side effects were noteworthy. A randomized trial conducted in the mid-1980s showed that fenfluramine was no better than placebo. Since then, fenfluramine was removed from the market in 1997 when there were reports of pulmonary hypertension and damage to heart valves.

See Also

► [Serotonin](#)

References and Reading

- Ritvo, E. R., Freeman, B. J., Yuwiler, A., Geller, E., Schroth, P., Yokota, A., et al. (1986). Review group(s): Cochrane child health field. *Psychopharmacology Bulletin*, 22(1), 133–140.
- Volkmar, F. R., Paul, R., et al. (1983). Irritability in autistic children treated with fenfluramine. *New England Journal of Medicine*, 309(3), 187.

Feral Children

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Definition

The term “feral” children is used to refer to children who are known, or are believed, to have been deprived of contact with other human beings or to have been neglected or treated inhumanely by a human being since infancy. They have had little or no experience of human love or care and no experience of human communication through spoken language. Some are thought to have been reared by animals. Accounts of such children can be found in myths, legends, fiction, and in stories believed to be true, though some have turned out to be hoaxes. The relevance for autism spectrum conditions lies in the inability to understand and speak and the odd behavior of many of the children and young adults, who are known or thought to have been deprived of human love and communication in their early years.

Historical Background

The accounts of such children in myths, legends, and fiction do not, however, have any link with

autism. The legend of Romulus and Remus is one example. They were twin brothers, sons of the god Mars. When very young, they were abandoned by the banks of the river Tiber, but were found by a she-wolf who fed them with her milk and sustained them until they were able to feed themselves. Later, they were found and given a home and care by a shepherd, and they grew up to be strong and clever. They decided to build a city on the place where the shepherd had found them – the legendary origin of the city of Rome. After it was built, they quarreled over who should be in charge, and Romulus killed Remus. Romulus became King of Rome, which he named after himself.

The most famous “feral” characters in fiction are Mowgli and Tarzan. Mowgli was a character in stories written by Rudyard Kipling. Mowgli’s parents were killed by a tiger in the Indian jungle when Mowgli was still a baby. He was found and reared by a mother and father wolf in a pack of wolves. He developed remarkable skills in hunting and tracking. He had close relationships with a black panther and a brown bear and was able to communicate with them. In the end, he returned to human society and had no problems communicating in human speech.

Tarzan was the fictional character invented by Edgar Rice Burroughs (although it has been suggested that he was influenced by the Mowgli stories). Tarzan’s parents died when he was a year old, and he was adopted by a group of great apes, the mangani, which is a species unknown to science! Tarzan’s jungle upbringing gave him remarkable abilities, beyond those acquired by most human beings. He was strong, agile, and well able to wrestle with all kinds of animals. He learned languages rapidly, including the language of the apes who reared him and other jungle animals and English, French, German, Latin, and ancient Greek, among others. He met and fell in love with a young American woman, Jane, whom he eventually married. They tried living together in England but found civilization hypocritical, so they returned to live in Africa.

The stories of Mowgli and Tarzan have been and are enormously popular and have led to many more books, as well as films, DVDs, television

programs, and even Disney cartoons. There seems to be something about these stories that people all over the world find endlessly fascinating. However, the fictional pictures of highly intelligent, gifted individuals who have learned languages and can communicate with animals and with human beings stand in stark contrast to the known facts concerning real “feral” children. Those in fiction bear no relationship to autism, unlike many of those, who, in real life, in their early years, were deprived of care and love from other human beings (Frith 2003a).

Current Knowledge

Another inappropriate use of the term “feral” children is to refer to those who live on the streets of cities. They are also known as “street” children. Such children have been deprived of family care because of neglect, or abuse, or parental death, or abandonment by poor parents who cannot afford to care for them. Large numbers of such children can be found in great cities throughout the world. Professor Martin Patt (2010) has created a website on which it is possible to find details of street children in different countries throughout the world. Patt emphasizes that such children can fall under the control of traffickers, who may disfigure the children in order to increase the money they can make by begging. The street children are also vulnerable to sexual abuse.

The suggestion of a link with autism is not appropriate especially for those street children who do not fall under the control of criminal adults. Typically, such children form their own gangs, with their own social rules and hierarchies to support each other and survive through various forms of criminal behavior. (See note on Romanian street children, Patt (2010) below.) They appear in fiction, for example, as the young pickpockets organized by Fagin in Charles Dickens’ book “*Oliver Twist*” or the street gangs, the “Baker Street Irregulars” who carried out various tasks for Sherlock Holmes in the stories by Sir Arthur Conan Doyle. They are quite different from the isolated child deprived of any human concern, care, and communication.

The children sometimes referred to as “feral,” whose history and behaviors are most relevant for any discussion of autism spectrum conditions, are those who have been deprived of human companionship and loving care for all or most of their infancy and childhood. There are accounts of children who are known or believed to have been kept in solitary confinement from very early in life, perhaps from infancy. These children have been given minimal basic feeding and minimal care by a human being who does not show them any love and companionship and does not communicate beyond simple orders. One of these unfortunate children was Kaspar Hauser, who appeared in Nuremberg in 1828, at the age of 16–17 years. A detailed account of Kaspar and his behavior was written by Anselm von Feuerbach (Feuerbach and Von Ritter 1833), then President of the Bavarian Court of Appeal at Anspael near Nuremberg. It seems that, during his first 16 years, Kaspar had been kept in a dark cellar. He had been fed on bread and water and had never seen his keeper. His only companion was a toy horse. When found in Nuremberg, he was able to say only a few phrases and fragments of speech. He preferred darkness to daylight and liked to sit on the ground with his legs stretched out in front of him. He had problems with distance vision, which could be linked to the fact that his vision had been limited by the darkness and walls of the cellar he had lived in.

His story is one of those discussed by Uta Frith (2003b). She notes that Kaspar rapidly learned to spell. He also showed social interest and affection towards children and adults who became close to him. He loved the toy horse he was given to replace the one he used to have in the cellar, and offered it a share of his food. He continued to have an odd gait, and he showed a marked preference for cleanliness and order for his possessions. Despite these continuing oddities, Professor Frith concluded that Kaspar was certainly not autistic – his social attachments and rapid improvement being strong indicators that he was not affected by this developmental disorder. Kaspar survived one attempt by a stranger to kill him. However, he was assassinated 5 years after his arrival in Nuremberg. The mysteries of his

origin, why he was kept in a cellar, why he was assassinated, and who killed him have never been solved, though there were unverified rumors that he was the unwanted child of a royal person.

A much more recent account of a child deprived of contact with loving human care and the outside world from early childhood is that of the girl known as Genie (not her real name). The psycholinguist who studied her, Susan Curtis (1977), referred to her as a modern-day “wild child.” Genie was born in the USA, the daughter of a violent, difficult father and a sad, frightened mother. She was the fourth and last child in the family, and was delivered by cesarean section. The first had died in infancy because his father objected to his crying and so kept him in the unheated garage where he developed pneumonia, which caused his death. The second child also died. The third, a boy, at 5 years, was taken into the care of his paternal grandmother, where his development, previously retarded, improved markedly, and he finally returned to his parents. At the age of 14 months, Genie developed pneumonia and was seen by a pediatrician, who suggested that she showed signs of retardation, though the pediatrician could not be sure because Genie had a high fever.

When Genie was 20 months old, her paternal grandmother died in a road accident. Genie’s father was intensely disturbed by this. He moved the family into his mother’s house and isolated them from the outside world. Genie was kept in a small bedroom, tied by a harness to a potty chair. At night, she was placed in a sleeping bag, which severely restricted her movements. Genie was kept secluded and had no toys to play with or radio to listen to. Her father hated any sort of noise, so conversation in the home was kept to a minimum and at a low volume. Genie’s father did not speak to her but barked and growled at her, and then beat her if she made any noises. She was fed baby foods that were stuffed into her mouth. This pattern of life for Genie lasted until she was 13½ years old, when her parents had a violent argument and Genie’s mother left her husband and home, taking Genie with her. After spending 3 weeks with the maternal grandmother, Genie was seen by a social worker, who sensed that

there were serious problems. Genie was admitted to hospital with severe malnutrition. The police were involved and charges brought against the parents. On the day of the trial, the father killed himself.

For 5 years from November 1970, when Genie was released from virtual captivity and isolation, her skills and disabilities were studied in detail by Curtiss and other colleagues. At first, she was very underweight and undersized and had major problems of self-care, movement, and distance vision. She was silent, even when sobbing, apart from a high-pitched whimpering and a few words. To quote from Susan Curtis (1977), “. . . she salivated copiously, spitting onto anything at hand. Genie was unsocialized, primitive, hardly human.”

Susan Curtiss analyzed in detail aspects of Genie’s ability with understanding and using language. Among other problems, Genie consistently confused *you* and *me*. She used entire phrases as labels, she failed to acknowledge questions, requests, etc., addressed to her. These are features also seen in children with autism spectrum disorders. Despite all this, Curtiss reports that Genie was alert and curious, avidly exploring her new surroundings. She was intensely eager for human contact and made good eye contact. These aspects of her behavior counteract any suggestion that she had an autism spectrum disorder. Susan Curtiss concluded that Genie appeared to be a “right hemisphere thinker.” She suggested that this was due to the deprivation of language experienced in her childhood.

Many questions relevant for a possible diagnosis of autism remain unanswerable. Was the pediatrician right in thinking that Genie was retarded when he or she saw her at 14 months, before her isolation? Did Genie show any of the odd hand and arm movements associated with autism, as well as her odd gait and posture? What was the underlying cause of Genie’s father’s bizarre behavior to his family – did he have a developmental disorder? There is no way of knowing.

Very relevant to the subject of children deprived of loving personal care and human communication from infancy or early childhood are the stories of the Romanian orphans. Nicolae Ceausescu was president of Romania from 1974

to 1989. During his presidency, birth control was made illegal in order to boost the population. The result was that many families had more children than they could possibly afford to care for, so these children were placed in state-run institutions. The care provided was minimal and often appallingly bad, and the children were kept in cots, deprived of human love, social interaction, and communication as well as lacking adequate nutrition and other health needs. In addition, during the Ceausescu regime, children who did not pass the state-organized physical and psychological assessments carried out on preschool children were also placed in institutions.

After the fall of Ceausescu and his regime, the sad life of the children in such institutions was publicized outside Romania. Some children from Romanian institutions were adopted by non-Romanian families, including some in the UK. A sample of 144 children who had experienced institutional care in Romania and who were later adopted by UK parents were assessed at ages 4, 6, and 11 years by Professor Michael Rutter et al. (2007; O'Connor et al. 2000; Rutter et al. 1999) They were compared with 52 non-deprived UK children adopted in the UK and placed before 6 months of age.

The children from Romania, when they arrived in England, showed marked signs of physical and psychological deprivation. They had been kept in their cots with no social or intellectual stimulation of any kind. The majority of the group showed remarkable catch-up by the time they were seen at 4 years of age. However, 16 children (11.1% of the sample of 144 children) showed "quasi-autism." Three of the 16 had IQs below 50, the rest having IQs above this level. These 13 children were studied in detail.

Their pattern of symptoms was called "quasi-autism" for specific reasons. While their scores on the ADI-R assessment at age 4 years were similar to a group of children with typical autism studied using the Autism Diagnostic Observation Schedule (Lord et al. 1994), their scores at age 6 years were significantly lower (less autistic) than those of the typically autistic children. There was also a tendency for the IQ scores to rise. They showed more flexibility in communication. Some showed

substantial social approaches, though of an abnormal kind, some being indiscriminately friendly. Those with "quasi-autism" had a much greater degree of cognitive impairment than the remainder of the adoptees from Romania. The sex ratio in the "quasi-autistic" children was approximately equal, compared with an excess of boys in the typically autistic children. Unlike the typically autistic children, the head circumference was not raised in those with "quasi-autism." The follow-up at 12 years showed that one quarter of the affected children had lost their autistic-like features by age 11 years.

Rutter and his team considered the possible causes of the "quasi-autistic" behavior, particularly in those (the majority) who did not have IQs below 50. They were significantly older than the rest when leaving institutional care to come to the UK. To quote from Rutter et al. (1999), "The quasi-autistic pattern seems to be associated with a prolonged experience of perceptual and experiential privation, with a lack of opportunity to develop attachment relationships and with cognitive impairment." The strongest predictor was the children's age of entry to the UK. Those adopted before 6 months showed almost complete physical and cognitive catch-up by 4 years. Those adopted between 6 and 24 months had lower scores on cognitive tests than those adopted before 6 months. Those adopted between 24 and 42 months had the lowest scores of all and a general developmental impairment. Rutter and his colleagues concluded that children adopted before 2 years of age were likely to show considerable resistance and to catch-up in cognitive and physical development.

The research team also found another 12 children who had some autistic-like features but not enough to qualify for "quasi-autism."

Future Directions

Dr. Sue Sheppard, a chartered specialist educational psychologist who is working in the field of developmental disorders and is a consultant to the NAS Diagnostic Centre, went to Romania in 1992 as one of the group of professionals whose

aim was to help the children in the state institutions. Among the children in the institutions, Sue Sheppard (personal communication, in conversation with the author in December 2010) found a small minority who quite clearly had autism spectrum disorders. This is not surprising, since parents who sent or abandoned children to institutions would be likely to select those whose behavior was challenging or whose development was clearly abnormal. Furthermore, state-implemented preschool screening tests would identify those with developmental disorders. In addition to those who were clearly in the autism spectrum, there was another minority who had some autistic traits, especially odd movements and other repetitive behaviors, and who were not socially responsive, though they did not present the full picture of an autism disorder. They closely resembled the children Rutter and colleagues described and called “quasi-autistic” (see above). Unlike those with clearly diagnosable autism, these children showed definite improvement over a period of time when given loving care and help by people who understood their problems, who came from relief agencies from other countries, sent in response to the problems children were having in Romania. These children’s physical health and intelligence levels also improved markedly. The majority of the institutionalized children did not show either of the above abnormal behavior patterns, seeming to be highly resistant despite the deprivation suffered. This was most likely to be the case if, as Rutter and his team also found, the children were under 2 years old when their living conditions were improved.

Sue Sheppard also observed another group of children, often described as “wild” or as “bandits.” These were children who lived in gangs on the streets and were sociable according to the values of the group they were with. They had become “street children” for various reasons, including having run away from abusive parents or institutions. They could smile sweetly and act in a charming way in order to get near enough to someone to steal from them. Although their behavior was antisocial from the point of view of society as a whole, as a group, they had their

own social system, which certainly does not fit with a diagnosis of autism spectrum disorder.

The last group of “feral” children to be discussed here are those who are given the name “feral” because they are thought to have been reared by animals in their early childhood years. Stories of such individuals found wandering, captured, and reintroduced into human society can be traced back to the 1300s. Lucien Malson (1972) gives a list of 53 “feral” children recorded from 1344 up to 1961. Wikipedia adds another 10 in the years from 1961 to 1999 and 8 from 2000 to 2009. The animals alleged to have cared for the children include wolves, cows, pigs, sheep, leopards, bears, chimpanzees, monkeys, dogs, goats, and gazelles. A few children were actually found with animals (most such accounts are of children found with packs of dogs), but there is no direct evidence of them being suckled by the animals in their infancy. Several of the stories are known or thought to be fictitious, including the two that described children reared by gazelles. Where sufficient contemporary information is available, it is clear that those thought to have been reared by animals lacked basic social skills. The great majority of those reported did not learn to speak or else managed only a few words; they had problems learning to walk upright and to use a toilet and had no interest in other human beings or human activity.

The one “feral” child concerning whom we have a considerable amount of information is the “wild boy of Aveyron.” This boy was found in 1798 wandering naked and wild in the woods in south-central France. He was captured and eventually given into the care of the physician Dr. Jean Marc Gaspard Itard and his house keeper, Madam Guerin. Itard wrote two detailed reports in 1801 and 1806 (see English translations, Itard 1962) describing the boy’s behavior and his progress in learning the skills that Itard tried to teach him.

When captured, Victor (the name given him by Itard) was thought to be about 12 years old, though small for his age. He had many scars on his body and limbs including one across his throat. He had been glimpsed in the forest on occasions for 2 years before his capture, so it seemed that he had lived in the wild at least from around age 10 up to 12 years.

In her book on autism, Uta Frith (2003c) includes a detailed discussion of the evidence concerning the possibility that Victor had autism. The relevant aspects of his behavior, as described by Itard, are as follows.

Victor showed no marked social attachment to anyone. He showed some attachment to his caretakers, Dr. Itard and Madame Guerin, which seemed to be due to the fact that they provided his food. He showed no sympathy and no evidence that he had any concept of other people's ideas, wishes, or feelings. He had no ability to conform to any social rules.

Victor made various noises but did not learn to speak. He did learn that the word "lait" referred to milk, but never used it to request milk, only as a label when he was given milk. He used limited nonverbal ways of communicating the few messages he wished to communicate. For instance, he enjoyed having Dr. Itard touch and stroke his head and would take Itard's hand and place it on his head to indicate what he wanted. He never showed any evidence of pretend play or imaginative skills.

As is typical in autism, Victor did respond in unusual ways to various kinds of sensory input (Leekam et al. 2007). He ignored most sounds, however sudden and loud, but turned at once to the sound of a nut cracking (nuts being a favorite food), however faint the sound. He seemed oblivious of heat, cold, or pain. This was one of the possible reasons why he could exist for at least 2 years in the wild.

He would wake early in the morning, wrap his head and body in his blanket and rock himself back and forth. He would lie down from time to time and then rock again until it was time for breakfast. He was fascinated by the moonlight, and would sit and watch the moon while rocking himself.

Despite lack of language skills, Victor was very efficient at shelling and preparing beans for cooking, including picking out any that were bad. He learned to carry out some other practical domestic tasks that did not require language to perform.

These behaviors indicate impairment of social interaction, communication, and imagination, and a preference for repetitive routines (rocking),

which are diagnostic of an autism spectrum disorder. Uta Frith considers that it is highly likely that Victor had such a disorder. The present writer agrees with her, though, from such a distance in time, there can be no absolute certainty.

Harlan Lane (1979), professor of social psychology, does not accept that Victor could have had an autism spectrum disorder. However, the aspects of Victor's behavior he lists as evidence that would exclude a diagnosis of autism could be seen in many children with this diagnosis. Lane notes that Victor was not totally aloof and indifferent to others. For example, as already mentioned above, Victor did show some signs of attachment to Dr. Itard and to Madame Guerin, who were his careers. There is an account of him weeping when reproached by Itard. But many children with typical autism spectrum disorders are not totally aloof – their social behavior is limited and can be inappropriate. Lane appears to be aware only of the most severe manifestations of autism and not the wide range of behavior across the whole autism spectrum and the changes that can occur with increasing age and the positive effects of education and of loving care.

While it is highly likely that Victor was in the autism spectrum and a few other "feral" children were clearly not autistic, for the great majority of cases of "feral" children, a diagnosis of autism or at least autistic traits appears to be a possibility, but there is rarely enough evidence to be certain.

A question that inevitably arises in any discussion of "feral" children is whether the autism spectrum disorder is due to biological causes and was present from birth (Feinstein 2010; Frith 2003d), leading to the child's loss or abandonment, or whether the lack of loving human care was the cause (Bethelheim 1967). The available information on "feral" children cannot provide an answer to this question. The known cases of "feral" children are just a minute fraction of all those with autism spectrum disorders. The latest prevalence studies suggest that around 1% or more of children and adults have some form of this disorder (Gillberg and Wing 1999; Wing 2004; Wing and Potter 2002). Research into the nature and origins of autism has shown that autism spectrum disorders are most likely to be due to

genetic or other biological causes (Feinstein 2010; Frith 2003d). The amount of progress possible for those with autism has been shown to be linked to their innate level of ability, as measured by IQ tests, though the prognosis even for those with IQ over 70 is very variable (Beadle-Brown et al. 2006; Howlin et al. 2004).

Another question that has not been answered concerns whether there is a “critical” or “sensitive” period for the acquisition of language. The critical period hypothesis holds that there is a limited time in early childhood when language can be acquired in response to exposure to facilitative speech presented by caretakers. If a child is deprived of this essential experience during the appropriate age period, he or she will not learn to understand and speak (Lenneberg 1967). It has been suggested that the histories of “feral” children support this theory. The findings of Rutter et al. concerning the importance of removal to a supportive environment before 2 years, described above, do provide some support. The problem is that there is no way of knowing whether or not the “feral” children who do not speak suffered from autism, or a developmental language disorder, or intellectual disability, as the reason why they did not speak and did not learn language when education was provided.

The sad truth is that the stories of “feral” children in all their variation, in most cases, present fascinating but insoluble mysteries. The questions they raise can only be solved (if at all) by appropriate modern research.

See Also

- Atypical Autism
- Child Abuse in Autism
- Factors Affecting Outcomes
- Romanian Adoptive Children

References and Reading

- Beadle-Brown, J., Murphy, G., & Wing, L. (2006). The Camberwell Cohort, 25 years on; characteristics and changes in skills over time. *Applied Research in Intellectual Disabilities, 19*, 317–329.
- Bethelheim, B. (1967). *The empty fortress*. New York: The Free Press.
- Candland, D. K. (1993). *Feral children and clever animals: Reflections on human nature*. Oxford: Oxford University Press.
- Curtis, S. (1977). *Genie: A psycholinguistic study of a modern-day wild child*. New York: Academic Press.
- Feinstein, A. (2010). *A history of autism: Conversations with the pioneers*. Chichester: Wiley-Blackwell.
- Feuerbach, A. R., & Von Ritter, P. (1833). *Kaspar Hauser: An account of an individual kept in a dungeon separated from all communication with the world from early childhood until about the age of 17*. London: Simpkin & Marshall.
- Frith, U. (2003a). Lessons from history. In U. Frith (Ed.), *Autism explaining the enigma* (2nd ed., pp. 34–57). Oxford: Blackwell.
- Frith, U. (2003b). The case of Kaspar Hauser: Lessons from history. In U. Frith (Ed.), *Autism explaining the enigma* (2nd ed., pp. 43–49). Oxford: Blackwell.
- Frith, U. (2003c). The case of the wild boy of Aveyron: Lessons from history. In U. Frith (Ed.), *Autism explaining the enigma* (2nd ed., pp. 35–43). Oxford: Blackwell.
- Frith, U. (2003d). Seeing the brain through a scanner: Lessons from history. In U. Frith (Ed.), *Autism explaining the enigma* (2nd ed., pp. 1820–1824). Oxford: Blackwell.
- Gillberg, C., & Wing, L. (1999). Review: Autism is not an extremely rare disorder. *Acta Psychiatrica Scandinavica, 99*, 399–406.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry, 45*(2), 212–229.
- Itard, J. M. G. (1962). *The wild boy of Aveyron*. New York: Appleton. Reports published in French in 1801 and 1807, translated into English by G. Humphrey & M. Humphrey.
- Lane, H. (1977). *The wild boy of Aveyron: The dramatic account of a wild boy of nature and a young French doctor who shaped the modern education of retarded, deaf and pre-school children* (pp. 176–178). London: George Alan & Unwin.
- Lane, H. (1979). *The wild boy of Aveyron*. Cambridge, MA: Harvard University Press.
- Leekam, S. R., Nieto, C., Libby, S., Wing, L., & Gould, J. (2007). Describing the sensory abnormalities of individuals with autism. *Journal of Autism and Developmental Disorders, 37*, 894–910.
- Lenneberg, E. H. (1967). *Biological foundations of language*. Chichester: Wiley.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 24*(5), 659–685.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2001). *Autism diagnostic observation schedule manual*. Los Angeles: Western Psychological Services.

- Malson, L. (1972). *Wolf children*. New York: Monthly Review Press.
- O'Connor, T. G., Rutter, M., Beckett, C., Keaveney, L., Kreppner, J. M., & The English & Romanian Adoptees Study Team. (2000). The effectiveness of global severe privation on cognitive competence: Extension and longitudinal follow-up. *Child Development*, 71(2), 376–390.
- Patt, M. (2010). *Street children: The prevalence, abuse & exploitation of street children*. Internet address: <http://gvnet.com/streetchildren/>. Accessed 25 June 2012.
- Rutter, M., Wood, L. A., Beckett, C., Bredenkamp, D., Castle, J., Groothues, C., et al. (1999). Quasi-autistic patterns following severe early global privation. *Journal of Child Psychology and Psychiatry*, 40(4), 537–549.
- Rutter, M., Kreppner, J., Croft, L., Murin, M., Colvert, E., Beckett, C., et al. (2007). Early adolescent outcomes of institutionally deprived and non-deprived adoptees III Quasi-autism. *Journal of Child Psychology and Psychiatry*, 48(12), 1200–1207.
- Wing, L. (2004). The spectrum of autistic disorders. *Hospital Medicine*, 65, 542–545.
- Wing, L., & Potter, D. (2002). The epidemiology of autistic spectrum disorders: Is the prevalence rising? *Mental Retardation and Developmental Disabilities Research Reviews*, 8, 151–161.

Ferster, C.B.

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Name and Degrees



Charles Ferster

Ferster, Charles Bohris (Nov. 1, 1922–Feb. 3, 1981).

B.S. (Rutgers University, 1947), M.A. (Columbia University, 1948), Ph.D. (Columbia University, 1950).

Major Appointments (Institution, Location, Dates)

- US Army Air Force, 1943–1946
- Research Fellow under B. F. Skinner, Harvard University, 1950–1955
- Postdoctoral Research, Yerkes Laboratory, Emory University, 1955–1957
- Postdoctoral Research, Indiana University Medical Center, 1957–1962
- Institute for Behavioral Research (Silver Spring, MD), 1962–1968, as Executive Director (1962–1963), Associate Director (1963–1965), Senior Research Associate (1965–1968)
- Professor of Psychology, Georgetown University (Washington, DC), 1967–1968
- Professor of Psychology, American University (Washington, DC), 1969–1981 (Department Chair, 1970–1973)

Major Honors and Awards

- First Editor and Executive Editor of the *Journal of the Experimental Analysis of Behavior*, 1958–1961.

Landmark Clinical, Scientific, and Professional Contributions

With B. F. Skinner, developed the experimental methods of the experimental analysis of behavior, including the design of apparatus and procedures, especially with regard to schedules of reinforcement.

Participated in the founding of the first major journal in the field, the *Journal of the Experimental Analysis of Behavior*.

Extended the basic principles of behavior analysis to clinical problems and to education, in areas

such as clinical depression, psychotherapy, and individual instruction.

Distinguished between the natural consequences of behavior and the arbitrary consequences that must be arranged when for some reason the natural consequences are not effective.

Opened the way to the treatment of autism by demonstrating that the behavior of the autistic child is sensitive to its consequences.

Short Biography

Charles B. Ferster was a pioneer in the experimental analysis of behavior and its extension to areas of application. His undergraduate career at Rutgers University was interrupted by service in the US Army Air Force during World War II. Upon completing his Rutgers B.S. in 1947, he entered the graduate program in Psychology at Columbia University. There Fred S. Keller, with W. N. Schoenfeld, had established a curriculum based on B. F. Skinner's analysis of behavior (Keller and Schoenfeld 1949, 1950). Keller referred Ferster to Skinner at Harvard University, and Ferster, completing his Ph.D. in 1950, moved to Harvard as Skinner's research assistant. Once there, he developed many innovations in methodology and in the automation of Skinner's pigeon laboratory, summarized in a journal article that became a guide for many other researchers (Ferster 1953). By 1957, he and Skinner had published a seminal book, *"Schedules of Reinforcement"* (Ferster and Skinner 1957), that detailed the properties and the results of these novel procedures in the analysis of behavior. Skinner was sufficiently impressed by Ferster's contributions to the research enterprise that he gave Ferster first authorship.

Behavior is maintained by its consequences, as when a pigeon's key peck or a rat's lever press occurs frequently because it produces access to food. This property of behavior is called reinforcement; reinforcement is not a theory but rather is simply a name for a ubiquitous phenomenon. What Skinner discovered about reinforcement in his earlier research (Skinner 1938), and what he and Ferster explored in depth, was that the maintenance of behavior by reinforcement depends

crucially on the contingencies arranged for responses, their schedules of reinforcement. In the real world as well as in the laboratory, not every response is reinforced. In what is called a ratio schedule, for example, only some fraction of responses is reinforced, as when some percentage of bets pays off; one must play to win, but one will not win every time. In what is called an interval schedule, however, whether a response is reinforced depends on when the response occurs rather than on how many responses have occurred, as when one finds mail in one's mailbox only if checking it after the mail has been delivered; checking more often will not affect when the mail comes. A full account of reinforcement schedules, which include both fixed and variable versions of ratio and interval schedules as well as other categories of contingencies, is beyond the scope of this article. For the present purposes, the crucial point is that different schedules produce and maintain very different patterns of behavior. For example, ratio schedules typically maintain substantially higher rates of behavior than do interval schedules, and details of the schedules determine the patterning of behavior over time (e.g., some schedules produce relatively constant responding, others produce gradual increases, and still others produce abrupt shifts from not responding to high rates of responding). Schedules of reinforcement soon became commonplace procedures for the study of a broad range of topics, such as the analysis of sensory processes, effects of psychopharmacological agents, and motivational operations.

One procedure studied by Ferster (1954) involved using a blackout, turning off the lights in the pigeon chamber and therefore eliminating the pigeon's opportunity to produce food. The name was later changed to time-out, and it was discovered that time-outs were sometimes useful because they could be used to reduce behavior, as when errors in some visual task produced time-outs from positive reinforcement. The use of time-out in some behavioral programs and eventually by some parents and teachers evolved from this early pigeon research.

In those early days of operant research, extending the basic results from the animal

laboratory to significant human problems had just begun. Examples were Fuller's (1949) demonstration that reinforcers could be effective even on the behavior of a vegetative human, work by Lindsley (1956) showing the effects of schedules of reinforcement on the behavior of adult psychotic patients, and Bijou's (1957) extensions to the study of child development. After moving to a postdoctoral position at the Medical Center at Indiana University in 1957, Ferster undertook an extension of his work with schedules of reinforcement to the behavior of autistic children. In those days, diagnostic criteria were highly variable, and such children were often described in terms of early-stage schizophrenia. More important, the classification of autism implied individuals who were virtually confined within themselves, impervious to events in their environments. Ferster, in collaboration with DeMyers (Ferster and DeMyer 1961, 1962), arranged laboratory settings functionally similar to those used in the analyses of reinforcement schedules and found that when appropriately arranged food and candy and trinkets could become effective reinforcers of the behavior of these children. Other studies examined ways in which these reinforcers might eventually provide a foundation for building social reinforcers, which are typically ineffective with these children (DeMyer and Ferster 1962). The research also showed that food could be used to make coins effective as conditioned reinforcers and that reinforcers could be arranged so as to enhance the attention of these children to some of the stimuli around them.

Beyond the fundamental observation that the behavior of these children could be reached through reinforcement schedules, albeit more slowly than with children without these deficits, this research provided the precedent for extensions that were soon to follow, including work by Lovaas and others (e.g., Lovaas et al. 1965, 1966). In his basic research, Ferster had shown that relatively simple and small changes in the contingencies arranged by reinforcement schedules could produce profound changes in ongoing behavior, and he brought these observations to bear in his subsequent work, which moved to analyses of other clinical deficits and pathologies,

such as clinical depression (Ferster 1965, 1972, 1973). Some additional contributions Ferster has made to the analysis of behavior have been described by Keller (1981) and by Skinner (1981).

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavior Analysis](#)
- [Contingencies of Reinforcement](#)
- [Operant Behavior](#)
- [Schedule of Reinforcement](#)

References and Reading

- Bijou, S. W. (1957). Methodology for an experimental analysis of child behavior. *Psychological Reports*, 3, 243–250.
- DeMyer, M. K., & Ferster, C. B. (1962). Teaching new social behavior to schizophrenic children. *Journal of the American Academy of Child Psychology*, 1, 443–461.
- Ferster, C. B. (1953). The use of the free operant in the analysis of behavior. *Psychological Bulletin*, 50, 263–274.
- Ferster, C. B. (1954). Use of the blackout in the investigation of temporal discrimination in fixed-interval reinforcement. *Journal of Experimental Psychology*, 47, 69–74.
- Ferster, C. B. (1961). Positive reinforcement and behavioral deficits of autistic children. *Child Development*, 32, 437–456.
- Ferster, C. B. (1965). Classification of behavioral pathology. In L. Krasner & L. P. Ullman (Eds.), *Research in behavior modification* (pp. 6–26). New York: Holt, Rinehart & Winston.
- Ferster, C. B. (1970). Schedules of reinforcement with Skinner. In P. B. Dews (Ed.), *Festschrift for B. F. Skinner* (pp. 37–46). New York: Appleton-Century-Crofts.
- Ferster, C. B. (1972). The experimental analysis of clinical phenomena. *Psychological Record*, 22, 1–16.
- Ferster, C. B. (1973). A functional analysis of depression. *American Psychologist*, 28, 857–870.
- Ferster, C. B., & DeMyer, M. K. (1961). The development of performances in autistic children in an automatically controlled environment. *Journal of Chronic Diseases*, 13, 312–345.
- Ferster, C. B., & DeMyer, M. K. (1962). A method for the experimental analysis of the behavior of autistic children. *Journal of Orthopsychiatry*, 32, 89–98.
- Ferster, C. B., & Skinner, B. F. (1957). *Schedules of reinforcement*. New York: Appleton-Century-Crofts.
- Fuller, P. R. (1949). Operant conditioning of a vegetative human organism. *American Journal of Psychology*, 62, 587–590.

Keller, F. S. (1981). Charles Bohris Ferster (1922–1981), An appreciation. *Journal of the Experimental Analysis of Behavior*, 36, 299–301.

Keller, F. S., & Schoenfeld, W. N. (1949). The psychology curriculum at Columbia College. *American Psychologist*, 4, 165–172.

Keller, F. S., & Schoenfeld, W. N. (1950). *Principles of psychology*. New York: Appleton-Century-Crofts.

Lindsley, O. R. (1956). Operant conditioning methods applied to research in chronic schizophrenia. *Psychiatric Research Reports*, 6, 118–139.

Lovaas, O. I., Freitag, G., Kinder, M. I., Rubinstein, B. D., Schaeffer, B., & Simmons, J. Q. (1965). Establishment of social reinforcers in two schizophrenic children on the basis of food. *Journal of Experimental Child Psychology*, 4, 109–125.

Lovaas, O. I., Berberich, J. P., Perloff, B. F., & Schaeffer, B. (1966). *Science*, 151, 705–707.

Skinner, B. F. (1938). *The behavior of organisms: An experimental analysis*. New York: Appleton-Century-Crofts.

Skinner, B. F. (1981). Charles B. Ferster-A personal memoir. *Journal of the Experimental Analysis of Behavior*, 35, 259–261.

Fetal Alcohol Effects

► [Fetal Alcohol Spectrum Disorder](#)

Fetal Alcohol Spectrum Disorder

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Synonyms

[Alcohol-related neurodevelopmental disorder](#);
[Fetal alcohol effects](#); [Fetal alcohol syndrome](#)

Short Description or Definition

Used to refer to the range of negative outcomes associated with prenatal ethyl-alcohol exposure,

fetal alcohol spectrum disorders (FASD) is an umbrella term that describes a heterogeneous set of permanent birth defects. When an FASD includes growth deficiency, a unique cluster of three minor facial anomalies (i.e., small eyes, a thin upper lip, and a smooth philtrum), and evidence of central nervous system (CNS) abnormalities, it is referred to as *fetal alcohol syndrome* (FAS). Other FASD can include any combination of these impairments in the context of confirmed prenatal alcohol exposure. CNS abnormalities are the most common impairment found in individuals with an FASD. FASD may also include ophthalmologic, otologic, cardiac, renal, and orthopedic abnormalities.

Categorization

Diagnostic categories
FAS, alcohol exposed
FAS, alcohol exposure unconfirmed
Partial FAS, alcohol exposed
Sentinel physical finding(s) with static encephalopathy, alcohol exposed
Static encephalopathy, alcohol exposed
Sentinel physical finding(s) with neurobehavioral disorder, alcohol exposed
Neurobehavioral disorder, alcohol exposed
Sentinel physical finding(s), alcohol exposed

Epidemiology

Although the impact of maternal drinking on the pre- and postnatal development of children was examined as early as the late nineteenth century (Sullivan 1899), teratogenic effects of prenatal ethyl-alcohol exposure were not characterized until the late 1960s (Lamache 1967) and not widely recognized until the work by Smith and colleagues in 1973 (Jones et al. 1973). The *fetal alcohol syndrome* (FAS) they described is now recognized internationally as a permanent birth defect syndrome resulting from prenatal alcohol exposure and is understood to include growth deficiency, a unique cluster of three minor facial anomalies (i.e., small eyes, a thin upper lip, and a

smooth philtrum), and evidence of central nervous system (CNS) abnormalities. Subsequent work has shown that significant impairments are frequently found in children with prenatal alcohol exposure even in the absence of the full set of symptoms required for a diagnosis of FAS. Over time, the term fetal alcohol spectrum disorders (FASD) has come to be used to describe the full range of impairments associated with prenatal alcohol exposure. FAS is the most easily recognized FASD, largely because the distinctive FAS facial phenotype provides a specific diagnostic marker of prenatal alcohol exposure (Astley 2004a). With prevalence of FAS estimated at 1–5 cases per 1,000 live births, it is the leading known preventable cause of developmental and intellectual disabilities. Given lifetime costs for FAS estimated at two million dollars per case (Lupton et al. 2004), FAS is estimated to have an annual cost in the United States in excess of five billion dollars (Riley and McGee 2005). FASD that lack the telltale facial features of FAS are more difficult to diagnose, but share a similar range and severity of central nervous system impairments and social costs. With numbers approaching 1% of all children, other FASD are many times more prevalent than FAS (Bailey and Sokol 2008).

Natural History, Prognostic Factors, and Outcomes

The impairments associated with prenatal ethyl-alcohol exposure are permanent birth defects that effect on the developing system throughout the life course of individuals with fetal alcohol spectrum disorders (FASD). The degree and extent of damage caused by prenatal alcohol exposure is both dose and timing dependent, and the central nervous system is the most vulnerable body system. Individuals with FASD are at risk for learning, behavioral, and mental health impairments throughout their lives. Protective factors that reduced the chances of negative outcomes in individuals with prenatal alcohol exposure and/or FASD include early diagnosis, stable home placement with quality care, protection from violence,

absence of substance abuse in the home, and the presence of appropriate social services. Early intervention in a coordinated system of care that maximizes these protective factors may reduce the chances that children with FASD will experience negative outcomes and should support their chances of success as they grow to adulthood (Streissguth et al. 2004).

Clinical Expression and Pathophysiology

The degree and extent of damage caused by prenatal ethyl-alcohol exposure is both dose and timing dependent. The central nervous system (CNS) is the most vulnerable body system, and various regions, developmental processes, and cell types in the CNS are differentially vulnerable to prenatal alcohol exposure at different stages of development. Prenatal alcohol exposure changes many ontogenetic processes in the developing CNS—disrupting or distorting neurulation and neural tube development, neuronal proliferation and migration, synaptogenesis, neuronal differentiation, and apoptosis. Prenatal alcohol exposure alters the hormone balance in the system as well as astroglial development and gene expression. Alcohol interferes with gliogenesis and development, delays myelination, and causes changes in the production of important neurotrophic factors (e.g., cell adhesion molecules such as the integrins and laminins) and neurotransmitters such as glutamate, NDMA, and serotonin.

Neurological Abnormalities

As expected, given its impact on developmental processes, prenatal alcohol exposure is associated with structural abnormalities throughout the CNS. These can include microcephaly, microgyria, gray and white matter defects, heterotopias, and regional hypoplasia. Imaging studies of the population with FASD are in their early stages, but some trends are emerging in the findings. Studies using functional magnetic resonance imaging and electroencephalography consistently find atypical results suggesting abnormal processing and development in individuals with prenatal alcohol exposure (e.g., D'Angiulli et al. 2006). The few

magnetic resonance spectroscopy studies conducted have all found differences in neurochemical profiles associated with prenatal alcohol exposure (e.g., reduced choline; Astley et al. 2009). Prenatal alcohol exposure can result in reduced frontal lobe volume relative to overall brain volume particularly in the presence of FAS facial features (Astley et al. 2009). There is also emerging evidence that temporal and parietal lobes may also be disproportionately vulnerable to prenatal alcohol exposure with exposure resulting in reduced volume of these structures. The caudate nucleus of the basal ganglia seems particularly sensitive to prenatal alcohol exposure (Astley et al.), while the putamen, amygdala, and hippocampus seem relatively resistant to damage from prenatal alcohol exposure (Spadoni et al. 2007).

Findings related to the volume of the cerebellar vermis (CV) are equivocal (e.g., Astley et al. 2009), making it unclear whether findings of reduced CV in children with FASD result from smaller overall brain size or not. However, when found in children with FASD, reduced volume of the CV is associated with deficits in verbal learning tasks. The effects of prenatal alcohol exposure on the corpus callosum (CC) are also not clear, but there is a consistent finding across studies of shorter CC, with some regions of the CC apparently more sensitive to prenatal alcohol exposure than others (Astley et al.). When they are found, CC abnormalities are associated with functional deficits.

Neuropsychological Findings

Neuropsychological impairments have been documented across a variety of domains in children with FASD. Deficits may include impairments of attention, motor and choice reaction time, visual spatial learning, fine/gross motor control and balance, response conditioning, executive function, working memory, mathematical reasoning, nonverbal inductive reasoning, and information processing (Kodituwakku 2007). Communication deficits are among the most frequently reported in FASD literature, and peripheral and central hearing impairments are common in FAS. Differences in IQ between children with

prenatal alcohol exposure and their unexposed peers are well documented. However, in the FASD population, IQ scores cover the range from profoundly handicapped to above average intelligence. Although lower IQ scores have been shown to be associated with the presence of the face of FAS and increased prenatal alcohol exposure levels, even among children with FAS, approximately 75% have IQ scores above 70 (Riley and McGee 2005).

Executive Functioning, Attention, and Behavioral Regulation: The caudate nucleus of the basal ganglia and the frontal lobes, structures which support executive functioning, seem particularly vulnerable to PAE, and impairments of executive function appear widespread in FASD. The most commonly reported concern is inattention. Deficits in planning, set shifting, problem solving, verbal and nonverbal fluency, and working memory are also commonly described. Deficits in social skills and adaptive functioning are widely reported, as are clinically significant problems maintaining appropriate behaviors (Kodituwakku 2007; Riley and McGee 2005).

Communication: Communication deficits have been documented for children with FASD in all areas of language and communication. These include impairments in auditory processing, phonology, morphosyntax, semantics, and integrative language/pragmatics. Later-developing discourse-level communication skills such as narrative storytelling may be particularly vulnerable to prenatal alcohol exposure (e.g., Coggins et al. 2007).

Sensory-Motor Development: Children with FASD frequently have deficits in fine motor, gross motor, and visual-motor abilities (Riley and McGee 2005). These can include delayed achievement of early motor milestones, tone abnormalities, tremulousness, and oral-motor (e.g., suck-swallow) difficulties. Older children with FASD may experience impaired balance, reduced fine motor control, visual spatial problems, and motor immaturities. The poor sensory modulation commonly described among clinic-referred children with FASD may play a role in motor and postural deficits and poor behavioral regulation (Jirikowic et al. 2008).

Mental Health: Mental health and psychiatric problems are found at high rates among clinical samples of individuals with prenatal alcohol exposure. Indeed, even low levels of prenatal alcohol exposure have been shown to increase risk of clinically significant mental health issues in young girls. Other adverse pre-/postnatal risk factors commonly associated with prenatal alcohol exposure (e.g., polydrug exposure, abuse/neglect) may exacerbate early biologic vulnerability and increase the risk for secondary disabilities later in life (Streissguth et al. 2004).

Evaluation and Differential Diagnosis

Fetal alcohol spectrum disorder (FASD) diagnoses are based on the degree of presentation of (1) prenatal and/or postnatal growth deficiency, (2) the fetal alcohol syndrome (FAS) facial phenotype, and (3) central nervous system (CNS) abnormality, in the context of (4) prenatal alcohol exposure. There are several competing diagnostic systems used in the diagnosis of FASD. This discussion of FASD diagnosis will be structured around the most established system, the FASD *4-Digit Diagnostic Code* (3rd edition, Astley 2004a: diagnostic guidelines, Lip-Philtrum Guides, and facial measurement software available at www.fasdpn.org). The other major diagnostic systems, the *Canadian Guidelines for FASD Diagnosis* (Chudley et al. 2005) and the *Guidelines for Identifying FAS* from the Centers for Disease Control (CDC; Bertrand et al. 2005), incorporate methods/tools from the *4-Digit Diagnostic Code* but use different diagnostic criteria.

In individuals being assessed for an FASD, as the presentation of key features of FAS move from not characteristic to highly characteristic of FAS, the *4-Digit Diagnostic Code* quantifies the expression of these features using a set of four 4-point Likert ranks that move from 1 to 4. The 4-digit code is created by combining these four Likert ranks from left to right in the order GROWTH, FACE, CNS, and PRENATAL ALCOHOL EXPOSURE (PAE). This numeric code is then used to assign an individual the appropriate

FASD diagnosis. A brief summary of how each Likert rank is determined is presented below.

Growth

In the *4-Digit Diagnostic Code*, a GROWTH rank of 1 is given when both prenatal and postnatal height and weight are above the 10th percentile; a GROWTH rank of 2 indicates that either prenatal or postnatal height, weight, or both are above the 3rd percentile, but less than or equal to the 10th percentile; a GROWTH rank of 3 indicates that either prenatal or postnatal height or weight is less than or equal to the 3rd percentile; and a GROWTH rank of 4, severe growth deficiency, is given when both height and weight are less than or equal to the 3rd percentile during either the prenatal or postnatal period.

The FAS Facial Phenotype

Three key facial features are considered diagnostic of FAS: (1) small eyes with palpebral fissure length (PFL) more than 2 standard deviations (SD) below the mean for age; (2) smooth philtrum consistent with a rank 4 or 5 on the University of Washington Lip-Philtrum Guide; and (3) thin upper lip consistent with a rank 4 or 5 on the University of Washington Lip-Philtrum Guide. *At these values*, this constellation of features has high specificity for prenatal ethyl-alcohol exposure, and their presence can serve as a proxy indicator of prenatal alcohol exposure when exposure is unknown (Astley 2006). In the *4-Digit Diagnostic Code*, a FACE rank of 4 indicates that all three facial features are present; a FACE rank of 3 indicates that deficits have been measured in all three features, but that one is only moderately impacted (e.g., PFL between -1 and -2 SD or a lip/philtrum Likert of 3); a FACE rank of 2 indicates that at least one feature is present; and a FACE rank of 1 indicates that there are no features present. Increased FACE rank is associated with increased risk/severity of CNS abnormality (Astley et al. 2009).

Central Nervous System (CNS) Integrity

The *4-Digit Diagnostic Code* includes a nested ranking of CNS abnormalities. This ranking quantifies both evidence of structural abnormality and

severity of functional impairment. A CNS rank of 4 indicates that there is direct structural/neurological evidence (e.g., microcephaly, seizures) of a “definite” abnormality in CNS structure; a CNS rank of 3 indicates that there is a “significant” functional impairment in three or more domains of brain function (i.e., a deficit >2 SD on a standardized test); a CNS rank of 2 indicates mild to severe impairment in some domain of functioning; and a CNS rank of 1 indicates that the individual does not meet criteria for CNS ranks of 2 through 4.

Documenting Prenatal Ethyl-Alcohol Exposure (PAE)

The *4-Digit Diagnostic Code* includes 4 exposure ranks. A PAE rank of 4 indicates a “confirmed exposure to high levels of alcohol.” Blood alcohol concentration greater than 100 mg/dL present on a weekly basis is given as a general guideline for defining “high levels” of exposure (Astley 2004a, p. 43). A PAE rank of 3 indicates “confirmed exposure” of either unknown quantity or of an amount that falls short of a PAE rank of 4. A PAE rank of 2, “unknown exposure,” indicates that exposure cannot be confirmed present nor absent. A PAE rank of 1 indicates a “confirmed absence of exposure from conception to birth.”

Diagnostic Categories

When combined, ranks for GROWTH, FACE, CNS, and PAE provide a *4-Digit Diagnostic Code*. This code can then be converted into 1 of 22 possible diagnoses. Eight of these fall under the umbrella of FASD. Importantly, only a diagnosis of FAS implies a causal role for prenatal alcohol exposure in producing the impairments identified during assessment. This causal inference is grounded in the specificity of the FAS facial phenotype to prenatal ethyl-alcohol exposure. For this reason, both FAS diagnoses in the *4-Digit Diagnostic Code* require a FACE rank of 4 (FAS = GROWTH 2–4, FACE 4, CNS 3–4, PAE 2–4). The other 6 FASD diagnostic categories simply describe the degree and type of impairment and document prenatal alcohol exposure. The eight FASD categories from the *4-Digit Diagnostic Code* are:

Diagnostic category	4-Digit code criteria
FAS, alcohol exposed	GROWTH 2–4, FACE 4, CNS 3–4, PAE 3–4
FAS, alcohol exposure unconfirmed	GROWTH 2–4, FACE 4, CNS 3–4, PAE 2
Partial FAS, alcohol exposed	GROWTH 1–4, FACE 3, CNS 3–4, PAE 3–4
Sentinel physical finding(s) with static encephalopathy, alcohol exposed	GROWTH 3–4, FACE 1–2, CNS 3–4, PAE 3–4
Static encephalopathy, alcohol exposed	GROWTH 1–2, FACE 1–2, CNS 3–4, PAE 3–4
Sentinel physical finding(s) with neurobehavioral disorder, alcohol exposed	GROWTH and/or FACE 3–4, CNS 2, PAE 3–4
Neurobehavioral disorder, alcohol exposed	GROWTH 1–2, FACE 1–2, CNS 2, PAE 3–4
Sentinel physical finding(s), alcohol exposed	GROWTH and/or FACE 3–4, CNS 1, PAE 3–4

In the *Canadian Guidelines for FASD Diagnosis*, there are four FASD diagnoses. These diagnoses are based on the *4-Digit Diagnostic Code* and include two FAS categories: FAS, alcohol exposed; and FAS, alcohol exposure unconfirmed. The *Canadian Guidelines* use loosened facial criteria for FAS compared to the *4-Digit Diagnostic Code*. In the Canadian system, FAS can be diagnosed with the following ranks: GROWTH 2–4, FACE 3–4, CNS 3–4, and PAE 2–4. The *Canadian Guidelines* diagnosis of Partial FAS is also a broader category than in the *4-Digit Diagnostic Code* and includes a FACE rank of 2. The final diagnostic category in the Canadian system is alcohol-related neurodevelopmental disorder (i.e., ARND), which corresponds to the *4-Digit Diagnostic Code* diagnoses of static encephalopathy with or without sentinel physical findings.

The Centers for Disease Control’s *Guidelines for Identifying FAS* has the most liberal FAS diagnostic criteria. CDC guidelines relax requirements for eye size to include PFL as large as 10th percentile. The CDC guidelines also relax criteria for CNS impairment and only require mild functional impairments (i.e., one standard deviation from the mean below the 16th percentile) in three domains of functioning for a diagnosis of FAS.

Treatment

Evidence-based interventions for fetal alcohol spectrum disorders (FASD) are emerging in the literature (Premji et al. 2007). Currently, primary prevention and direct individualized instruction to remediate specific learning and/or behavior problems are the primary tools available for treating individuals with FASD. Because children with FASD commonly face disruptive challenges from their environment as well as complex neurophysiologic deficits (Coggins et al. 2007), successful treatment for this population demands multimodal services. These services should incorporate the knowledge and expertise of multiple disciplines (e.g., medicine, psychology, social work, special education, speech-language pathology, audiology, occupational therapy) in order to remediate both environmental barriers to success and central nervous system (CNS) dysfunction.

Primary Prevention: FASD are entirely preventable, and simple, low-cost informative counseling directed at women who use alcohol and are pregnant or thinking about pregnancy can decrease FASD risk (Bailey and Sokol 2008). More intensive interventions for women at high risk who have already given birth to one child with prenatal alcohol exposure are also important. For example, the *Parent–Child Assistance Program* (Grant et al. 2005) provides participants with a paraprofessional advocate throughout their pregnancy and for 3 years after their child is born. The program helps mothers to decrease or eliminate their use of alcohol and/or to use family planning to prevent pregnancies when using alcohol. The program also promotes addiction recovery, supports economic and personal self-sufficiency, and helps participants connect to community resources.

Screening: Screening in high-risk populations has also been demonstrated to be effective in reducing FASD (e.g., Astley 2004b). The state of Washington, for example, developed an effective program for screening children in the foster care system by using a review of medical records and facial photograph. In the program, those children with a positive screen for the FAS facial phenotype or for CNS damage (e.g., microcephaly) are

invited for a diagnostic evaluation. Benefits from this kind of program include early diagnosis, earlier access to community supports and services, and increased caregiver knowledge regarding signs and symptoms of FASD.

Medical Interventions: Medical providers play an important role in FASD prevention, screening, diagnosis, and treatment. Early referrals to appropriate service providers are of particular importance and should always include formal vision and audiological screenings. Because efficacy and safety data on psychotropic medications are not generally available for the FASD population (Premji et al. 2007), there is little guidance for providers on how to target specific psychiatric or behavioral symptoms associated with FASD. Children with FASD may be at risk for paradoxical reactions and can be more sensitive to dosing. While research using animal models has revealed a number of promising medical approaches for protecting against or even reversing the long-term impacts of alcohol exposure, a more rigorous evidence base needs to be established before these approaches can play a major role in FASD treatment. Avenues being pursued include perinatal choline supplementation, prenatal folic acid, exercise training, and postnatal aniracetam treatment.

Educational and Behavioral Interventions: Because the impairments associated with prenatal ethyl-alcohol exposure are timing and dose dependent, the FASD population is extremely heterogeneous. As a result, no single behavioral or educational intervention approach will meet the needs of all those in the FASD population. Interventions need to be designed to target the specific needs of a particular individual with FASD in order to be most effective. Indeed, targeted and multimodal interventions are reported to make important changes across behavioral and learning domains for children with FASD while also improving caregiver outcomes (Kalberg and Buckley 2007). Effectiveness may be maximized when interventions include a functional behavioral analysis to identify problem behaviors and antecedent events leading to those behaviors. This analysis can then be paired with caregiver and teacher education to “reframe” problem behaviors from a neurodevelopmental

perspective. Accommodations and environmental modifications to compensate for specific neurological deficits are also important pieces of any effective intervention program (c.f., Olson et al. 2005). Social skills training can be another important element in any comprehensive intervention plan and has been shown to be effective with school-age children with FASD. Targeted interventions for specific academic deficits are effective for children with FASD, and approaches that work with typically developing populations and children with other similar developmental disabilities should be effective interventions for individuals with FASD.

References and Reading

- Astley, S. J. (2004a). *Diagnostic guide for fetal alcohol spectrum disorders: The four-digit diagnostic code* (3rd ed.). Seattle: FAS Diagnostic and Prevention Network, University of Washington, electronic version available from <http://fasdpn.org>
- Astley, S. J. (2004b). Fetal alcohol syndrome prevention in Washington State: Evidence of success. *Paediatrics & Perinatal Epidemiology*, 18(5), 344–351.
- Astley, S. J. (2006). Comparison of the 4-digit diagnostic code and the hoyme diagnostic guidelines for fetal alcohol spectrum disorders. *Pediatrics*, 118(4), 1532–1545.
- Astley, S. J., Aylward, E. H., Olson, H. C., Kerns, K., Brooks, A., Coggins, T. E., et al. (2009a). Magnetic resonance imaging outcomes from a comprehensive magnetic resonance study of children with fetal alcohol spectrum disorders. *Alcoholism, Clinical and Experimental Research*, 33(10), 1671–1689.
- Astley, S. J., Richards, T., Aylward, E. H., Olson, H. C., Kerns, K., Brooks, A., et al. (2009b). Magnetic resonance spectroscopy outcomes from a comprehensive magnetic resonance study of children with fetal alcohol spectrum disorders. *Magnetic Resonance Imaging*, 27(6), 760–778.
- Bailey, B. A., & Sokol, R. J. (2008). Pregnancy and alcohol use: Evidence and recommendations for prenatal care. *Clinical Obstetrics and Gynecology*, 51(2), 436–444.
- Bertrand, J., Floyd, R., Weber, K., O'Connor, M., Riley, E., Johnson, K., & Cohen, D. (2005). *National task force on fetal alcohol syndrome and fetal alcohol effect. Fetal alcohol syndrome: Guide-lines for referral and diagnosis*. Atlanta: Centers for Disease Control and Prevention.
- Coggins, T. E., Timler, G. R., & Olswang, L. B. (2007). A state of double jeopardy: Impact of prenatal alcohol exposure and adverse environments on the social communicative abilities of school-age children with fetal alcohol spectrum disorder. *Language, Speech, and Hearing Services in Schools*, 38(2), 117–127.
- Chudley, A. E., Conry, J., Cook, J. L., Looock, C., Rosales, T., & LeBlanc, N. (2005). Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis. *Journal of Canadian Medical Association*, 172, Suppl. 5).
- D'Angiulli, A., Grunau, P., Maggi, S., & Herdman, A. (2006). Electroencephalographic correlates of prenatal exposure to alcohol in infants and children: A review of findings and implications for neurocognitive development. *Alcohol*, 40(2), 127–133.
- Grant, T. M., Ernst, C. C., Streissguth, A. P., & Stark, K. (2005). Preventing alcohol and drug exposed births in Washington state: Intervention findings from three parent-child assistance program sites. *The American Journal of Drug and Alcohol Abuse*, 31(3), 471–490.
- Jirikowic, T., Carmichael-Olson, H., & Kartin, D. (2008). Sensory processing, school performance, and adaptive behavior among young school-aged children with FASD. *Physical & Occupational Therapy in Pediatrics*, 28(2), 117–136.
- Jones, K., Smith, D., Ulleland, C., & Streissguth, A. (1973). Pattern of malformation in offspring of chronic alcoholic mothers. *Lancet*, 301(7815), 1267–1271.
- Kalberg, W. O., & Buckley, D. (2007). FASD: What types of intervention and rehabilitation are useful? *Neuroscience and Biobehavioral Reviews*, 31(2), 278–285.
- Kodituwakku, P. W. (2007). Defining the behavioral phenotype in children with fetal alcohol spectrum disorders: A review. *Neuroscience and Biobehavioral Reviews*, 31(2), 192–201.
- Lamache, A. (1967). The progeny of alcoholics article in French. *Bulletin de l'Académie Nationale de Médecine*, 151(25), 517–521.
- Lupton, C., Burd, L., & Harwood, R. (2004). Cost of fetal alcohol spectrum disorders. *American Journal of Medical Genetics. Part C, Seminars in Medical Genetics*, 127(1), 42–50.
- Olson, H. C., Quamma, J., Brooks, A., Lehman, K., Ranna, M., & Astley, S. J. (2005). Efficacy of a new model of behavioral consultation for families raising school-aged children with FASD and behavior problems. *Alcoholism, Clinical and Experimental Research*, 29(5), 247A. Abstract 243.
- Premji, S., Benzie, K., Serrett, K., & Hayden, K. A. (2007). Research-based interventions for children and youth with a fetal alcohol spectrum disorder: Revealing the gap. *Child: Care, Health and Development*, 33(4), 389–397. discussion 398–400.
- Riley, E. P., & McGee, C. L. (2005). Fetal alcohol spectrum disorders: An overview with emphasis on changes in brain and behavior. *Experimental Biology and Medicine (Maywood, N.J.)*, 230(6), 357–365.
- Sullivan, W. C. (1899). A note on the influence of maternal inebriety on the offspring. *Journal of Mental Sciences*, 45, 489–503.
- Spadoni, A. D., McGee, C. L., Fryer, S. L., & Riley, E. P. (2007). Neuroimaging and fetal alcohol spectrum

disorders. *Neuroscience and Biobehavioral Reviews*, 31(2), 239–245.

Streissguth, A. P., Bookstein, F. L., Barr, H. M., Sampson, P. D., O'Malley, K., & Young, J. K. (2004). Risk factors for adverse life outcomes in fetal alcohol syndrome and fetal alcohol effects. *Journal of Developmental and Behavioral Pediatrics*, 25(4), 228–238.

Fetal Alcohol Syndrome

► Fetal Alcohol Spectrum Disorder

Fetal Anticonvulsant Syndrome

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Synonyms

Fetal antiepileptic drug syndrome (fetal AED syndrome); Fetal carbamazepine syndrome; Fetal phenytoin syndrome; Fetal valproate syndrome (FVS)

Short Description or Definition

“Fetal anticonvulsant syndrome” or “FACS” is the term used to describe the adverse effects of in utero exposure to anticonvulsant medication (e.g., phenobarbitone, phenytoin, carbamazepine, valproate, lamotrigine, etc.) and may manifest as dysmorphic facial features, major and minor structural anomalies, learning difficulties, and/or behavioral problems. Specific health and developmental problems are noted following exposure to specific antiepileptic drugs, e.g., phenytoin, valproate; the terms fetal phenytoin syndrome and fetal valproate syndrome are then used.

Epidemiology

Epilepsy is a common neurological condition affecting 0.6–1% of the population, and about a third of these are women of reproductive age. The majority of these women will be treated with antiepileptic drugs. Hence, approximately 1 in 160–250 pregnancies are exposed to antiepileptic drugs.

Phenobarbital was introduced for use as an antiepileptic drug (AED) in 1912, phenytoin in 1938, carbamazepine in 1961, and sodium valproate in 1964. In the early 1960s, there were reports from Germany (Janz and Fuchs 1964), followed shortly later by reports from England, describing a pattern of major and minor anomalies affecting orofacial, cardiac, and skeletal development in children with prenatal exposure to AEDs. Gradually, reports of cognitive deficits and behavioral problems emerged.

The exact incidence of fetal anticonvulsant syndrome (FACS) is not clear, as not all individuals with in utero exposure to AEDs show the adverse effects. Also, variability in the manifestation of the adverse effects is noted even between individuals who have been exposed to the same dose of the same AED. Some AEDs like valproate may be used in the treatment of neuropsychiatric disorders such as bipolar disorders. Offspring of these women are also at a risk of developing the adverse effects, if exposed in utero.

Natural History, Prognostic Factors, and Outcomes

Natural history: The diagnosis of FACS is first suspected when ultrasound scan abnormalities are identified in a pregnant woman taking AEDs. However, the absence of scan abnormalities is not reassuring, as minor structural abnormalities may not always be picked up on scan and also because the scans do not usually provide any clues to the neurodevelopmental problems that may develop later on in life.

The rate of obstetric complications in women with epilepsy is thought to be increased, but no single complication has been identified as a

Fetal Anticonvulsant Syndrome, Table 1 Congenital malformations associated with FACS

Systems involved	Abnormalities	Additional information
Neural tube defects	Spina bifida, anencephaly	1–2% risk of spina bifida with valproate, 0.5–1% with carbamazepine exposure
Congenital heart defects	Ventricular septal defects, atrial septal defects, patent ductus arteriosus	Seen with exposure to any AED
Orofacial clefts	Cleft lip and palate, cleft palate	Cleft palate has been reported with valproate exposure, but not cleft lip
Limb defects	Radial ray abnormalities, split hand malformation, polydactyly, tibial hypoplasia, camptodactyly	Seen with exposure to valproate; camptodactyly may be seen with other AED exposure as well
Genitourinary defects	Hypospadias, renal hypoplasia, hydronephrosis	More common with valproate exposure
Brain abnormalities	Agenesis of corpus callosum, porencephaly, hydranencephaly	Not frequently seen but have been reported
Eye abnormalities	Cataracts, optic nerve hypoplasia, iris defects	Iris defects have been reported with exposure to valproate only
Abdominal wall defects	Omphalocele	
Respiratory tract abnormalities	Laryngeal hypoplasia, abnormal lobation of lung, lung hypoplasia, tracheomalacia	More common with valproate exposure
Craniosynostosis	Trigonocephaly	Seen with valproate exposure only and is caused by premature fusion of the metopic suture; it may need surgical intervention

F

common finding, and in most studies, the increased rate of obstetric complications has not been found to be statistically significant. An increased incidence of stillbirths has been reported but not proven to be significant statistically. The birth weight is usually average. There is an increased rate of admission to special care baby units due to withdrawal symptoms such as lethargy, irritability, poor feeding, jitteriness, and due to the presence of major malformations such as neural tube defects, heart defects, kidney abnormalities, etc.

Congenital malformations: There is a 6–7% risk of structural abnormalities at birth compared to 2–3% in the general population. This risk may be as high as 9% in children exposed to valproate (Table 1).

Minor anomalies: Inguinal hernia, joint laxity, club feet, capillary hemangiomas (birth marks), strabismus (squint), nail hypoplasia, digital hypoplasia, clinodactyly, and overlapping toes are more frequently seen compared to the general population.

Facial dysmorphism: There are subtle facial features that are described with exposure to different AEDs. There is an overlap in the facial features described with exposure to different drugs. Of these, the facial gestalt of valproate exposure is the most characteristic and is formed by the following features: thin arched eyebrows with medial deficiency, epicanthic folds, infraorbital grooves, broad nasal bridge, short anteverted nose, smooth long philtrum, and a thin upper lip (Kini et al. 2006).

Childhood medical problems: Visual and hearing problems are more frequently seen. Myopia (short-sightedness) and squints are more common; hearing problems include recurrent otitis media with effusion.

Developmental delay and learning difficulties: Global developmental delay may be seen, but speech delay alone is more common. Joint laxity and poor muscle tone contribute to poor coordination which may affect daily activities such as writing, dressing and undressing, riding a bike, swimming, etc. A large proportion of children

have additional educational needs, and some of them may need one-to-one help much of the time (Adab et al. 2001). Many studies have shown that children exposed to AEDs such as carbamazepine, phenytoin, and even the newer drug, lamotrigine, have an IQ in the normal range (Gaily et al. 2004; Mawer et al. 2002; Dean et al. 2002; Meador et al. 2011). However, the verbal IQ of individuals with valproate exposure appears to be significantly lower (Adab et al. 2004).

Behavioral problems: Autistic spectrum disorders have been reported more frequently with valproate exposure (Rasalam et al. 2005). Difficulty in developing peer relationships, sharing interests and enjoyment, using language in social communication, symbolic or imaginative play, changes to routine, and lack of emotional or social reciprocity have been reported commonly. Poor concentration and hyperactivity are other behavioral issues reported.

Prognostic factors: Several factors influence the severity of FACS: (1) Type of AED – Valproate exposure causes the most severe adverse effects. (2) Number of AEDs – Polytherapy (exposure to more than one AED) has a worse outcome than exposure to monotherapy (one AED). (3) Dose of the drug – Although the adverse effects have not been found to be dose related with exposure to other AEDs, exposure to $\geq 1,000$ mg/day of valproate has been associated with more severe problems. Specific dose-related features of valproate are neural tube defects, radial ray abnormalities, obviously dysmorphic face, and a reduction in verbal IQ (Kini 2006). (4) Time of exposure – First trimester exposure is associated with structural abnormalities. It is possible however that AED exposure through the second and third trimesters may have an effect on neurodevelopment as the brain continues to grow throughout the pregnancy. (5) Parental and environmental factors – The child's IQ is known to strongly correlate with the parental IQ and also with the socioeconomic status. Women with epilepsy may themselves have learning difficulties, contributing further to the phenotype. (6) Genetic factors – The teratogenic effects of AEDs differ between fetuses that have been exposed to the same dose of the same drug suggesting that

maternal genetic factors may contribute. Studies have shown the contribution of genetic factors such as MTHFR (Kini et al. 2007), epoxide hydrolase genes, etc.

Clinical Expression and Pathophysiology

The clinical expression of FACS is very variable. The full spectrum of problems, i.e., dysmorphic facial features, major and minor malformations, developmental delay, childhood medical problems, learning difficulties, and behavioral problems, is rarely seen in a single individual with prenatal AED exposure. The presence of specific dysmorphic facial features may be a clue to early diagnosis. However, the facial gestalt is usually obvious in the most severely affected patients only. In fact, the facial features with exposure to some AEDs such as carbamazepine are very mild, adding to the difficulty of making a conclusive diagnosis. In addition, some individuals may present with subtle dysmorphic features and neurodevelopmental problems only. In these individuals, a diagnosis of “fetal anticonvulsant effects” should be considered.

Mechanisms of Teratogenicity

The exact mechanism by which AEDs cause their teratogenic effects is not known. Several plausible theories have been proposed:

1. Folic acid deficiency: AEDs such as phenytoin, phenobarbitone, and carbamazepine interfere with the intestinal absorption of folic acid, while valproate acts on its metabolism. Embryonic folic acid deficiency may disrupt gene expression, increase oxidative stress, and cause changes in protein synthesis (Wegner and Nau 1992). In animal studies, the rate of neural tube defects was reduced in mice exposed to valproate, when high doses of folic acid and folinic acid were given. Alteration in expression of folate pathway genes has been demonstrated in mice following valproate exposure (Finnell et al. 1997). Certain genetic

- polymorphisms in the folate pathway (*C677T* in the *MTHFR* gene) have been shown to be associated with a higher rate of malformations in AED-exposed children (Kini et al. 2007).
2. Oxidative stress: Intermediate metabolites of AEDs may increase oxidative stress in the developing embryo, whose antioxidant defense mechanism is immature. This may damage the developing organs of the embryo. Embryonic oxidative stress due to free radical formation following hypoxia from phenytoin-induced embryonic bradycardia has been demonstrated in animal studies.
 3. Arene oxide intermediates: Reactive intermediate metabolites of AEDs such as phenytoin and carbamazepine in the form of arene oxides may bind cell macromolecules affecting cell function and causing cell death. Arene oxides are detoxified by epoxide hydrolases; genetic variability in the activity of these enzymes may influence the susceptibility of the fetus to the adverse effects.
 4. Histone deacetylase inhibition: Valproate is a potent histone deacetylase inhibitor. Histone deacetylases are enzymes that induce chromatin changes to enable transcription of genes. Histone deacetylase inhibition therefore leads to interruption in the cell cycle, growth arrest, and apoptosis (cell death). Valproate also causes demethylation of DNA which results in changes in gene expression and hence in congenital malformations.

Evaluation and Differential Diagnosis

FACS is a difficult diagnosis to make as it is a diagnosis of exclusion. There are no specific tests by which the diagnosis of FACS can be confirmed. Hence, basic tests such as karyotyping, fragile X syndrome, and urine metabolic tests should be offered to the patients to rule out other causes. Other fetal insults such as exposure to alcohol, toluene, and maternal diabetes may cause similar features as FACS and should be kept in mind while taking the medical history. Malformations related to specific drug exposure such as radial ray defects and trigonocephaly with

valproate exposure may give a more definitive diagnosis, but other dysmorphic syndromes such as Baller-Gerold syndrome which have similar features need to be ruled out. This differentiation is important as it will impact on the recurrence risk given to these families. Referral to a clinical geneticist is therefore recommended. In those with a history of AED exposure, who present mainly with behavioral problems such as autistic spectrum disorder, evaluation for other syndromes such as Rett syndrome, tuberous sclerosis, and fragile X syndrome should be considered.

Treatment

Management

The management of FACS is symptomatic. Specific structural abnormalities, such as heart defects, kidney abnormalities, etc., may need monitoring, medication, or surgical intervention. Children with developmental delay may need help in the form of physiotherapy, speech and language therapy, and occupational therapy. Surveillance for childhood medical problems such as myopia and recurrent otitis media is recommended. Assessment for special educational needs should be carried out early and appropriate help instituted early to allow the child to develop to his or her full potential. Behavioral therapy and sometimes medication may be helpful in dealing with behavioral issues such as autistic spectrum disorder and attention deficit/hyperactivity disorder.

Prevention

For women with epilepsy, prepregnancy counseling should be provided so as to reduce the risk of FACS. Every attempt should be made to reduce the dose (e.g., valproate should be reduced to <1,000 mg/day) and number of AEDs to the minimal level required to control the epilepsy, and this should be done in liaison with the neurologist. Monotherapy is preferable to polytherapy. Valproate should be avoided wherever possible as this has been deemed to be the most teratogenic AED (Mawer et al. 2002). The weaning of the AED should be done well in advance of the pregnancy as seizures during

pregnancy may have a detrimental effect on the health of the mother and baby.

High-dose folic acid (4–5 mg/day) is recommended during pregnancy, starting from at least 6 weeks prior to conception. While intake of folic acid in the first trimester may help prevent congenital malformations, intake in the last two trimesters may help prevent neurodevelopmental problems due to continued development of the fetal brain throughout pregnancy. Antenatal scans should be offered at about 12 weeks (gross structural abnormalities such as anencephaly can be identified) and a detailed anomaly scan should be offered at 18–20 weeks. Malformation such as isolated cleft palate is likely to be missed on antenatal scans.

See Also

► [Intellectual Disability](#)

References and Reading

- Adab, N., Jacoby, A., Smith, D., & Chadwick, D. (2001). Additional educational needs in children born to mothers with epilepsy. *Journal of Neurology, Neurosurgery & Psychiatry*, 70(1), 15–21.
- Adab, N., Kini, U., Vinten, J., Ayres, J., Baker, G., Clayton-Smith, J., et al. (2004). The longer term outcome of children born to mothers with epilepsy. *Journal of Neurology, Neurosurgery & Psychiatry*, 75(11), 1575–1583.
- Dean, J. C., Hailey, H., Moore, S. J., Lloyd, D. J., & Turnpenny, P. D. (2002). Long term health and neurodevelopment in children exposed to antiepileptic drugs before birth. *Journal of Medical Genetics*, 39(4), 251–259.
- Finnell, R. H., Wlodarczyk, B. C., Craig, J. C., Piedrahita, J. A., & Bennett, G. D. (1997). Strain-dependent alterations in the expression of folate pathway genes following teratogenic exposure to valproic acid in a mouse model. *American Journal of Medical Genetics*, 70(3), 303–311.
- Gaily, E., Kantola-Sorsa, E., Hiilesmaa, V., Isoaho, M., Matila, R., Kotila, M., et al. (2004). Normal intelligence in children with prenatal exposure to carbamazepine. *Neurology*, 62(1), 28–32.
- Janz, D., & Fuchs, U. (1964). Are antiepileptic drugs harmful during pregnancy? *Deutsche Medizinische Wochenschrift*, 89, 241–248.
- Kini, U. (2006). Fetal valproate syndrome: A review. *Paediatric and Perinatal Drug Therapy*, 7(3), 123–130.
- Kini, U., Adab, N., Vinten, J., Fryer, A., Clayton-Smith, J., & Liverpool Manchester Neurodevelopmental Study Group. (2006). Dysmorphic features: An important clue to the diagnosis and severity of fetal anticonvulsant syndromes. *Archives of Disease in Childhood: Fetal and Neonatal*, 91(2), F90–F95.
- Kini, U., Lee, R., Jones, A., Ramsden, A., Fryer, A., Clayton-Smith, J., & Liverpool Manchester Neurodevelopmental Study Group. (2007). Influence of MTHFR genotype on the rate of malformations in children with antiepileptic drug exposure in utero. *European Journal of Medical Genetics*, 50(6), 411–420.
- Mawer, G., Clayton-Smith, J., Coyle, H., & Kini, U. (2002). Outcome of pregnancy in women attending an outpatient epilepsy clinic: Adverse features associated with higher doses of sodium valproate. *Seizure*, 11(8), 512–518.
- Meador, K. J., Baker, G. A., Browning, N., Cohen, M. J., Clayton-Smith, J., Kalayjian, L. A., Kanner, A., Liporace, J. D., Penell, B., Privitera, M., Loring, D. W., & NEAD Study Group. (2011). Foetal antiepileptic drug exposure and verbal versus non-verbal abilities at three years of age. *Brain*, 134(2), 396–404.
- Rasalam, A. D., Hailey, H., Williams, J. H. G., Moore, S. J., Turnpenny, P. D., & Lloyd, D. J. (2005). Characteristics of fetal anticonvulsant syndrome associated with autistic disorder. *Developmental Medicine and Child Neurology*, 47, 551–555.
- Wegner, C., & Nau, H. (1992). Alteration of embryonic folate metabolism by valproic acid during organogenesis: Implications for mechanism of teratogenesis. *Neurology*, 42(4 Suppl 5), 17–24.

Fetal Antiepileptic Drug Syndrome (Fetal AED Syndrome)

► [Fetal Anticonvulsant Syndrome](#)

Fetal Carbamazepine Syndrome

► [Fetal Anticonvulsant Syndrome](#)

Fetal Phenytoin Syndrome

► [Fetal Anticonvulsant Syndrome](#)

Fetal Valproate Syndrome (FVS)

► [Fetal Anticonvulsant Syndrome](#)

Fidelity of Implementation

► [Procedural Fidelity](#)
 ► [Treatment Fidelity](#)

Figurative Language

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Synonyms

[Figures of speech](#); [Metaphoric language](#); [Non-literal language](#)

Definition

Figurative language is language that is used in nonliteral ways in order to achieve a special effect or meaning. Similes, metaphors, idioms, proverbs, and slang are examples of figurative language. The ability to comprehend and produce nonliteral language is an important development in school-age children. Figurative language is commonly used in a variety of communicative contexts, including casual conversation, teacher talk, and written texts.

Individuals with autism spectrum disorders (ASD) often have difficulty comprehending figurative language and may interpret figurative statements literally. Upon hearing the expression “It’s raining cats and dogs,” an individual with ASD may think that the speaker means that cats and dogs are literally falling from the sky. The meaning

and appropriate use of figurative language may need to be explicitly taught to individuals with ASD. Difficulties with figurative language are common in individuals with a variety of disorders (e.g., Specific Language Impairment, Attention Deficit Hyperactivity Disorder).

See Also

► [Definition: Metaphor](#)
 ► [Metaphoric Language](#)

References and Reading

- Hedge, M. N., & Maul, C. (2006). *Language disorders in children: An evidence-based approach to assessment and treatment*. Boston: Pearson Education.
- Paul, R. (2007). *Language disorders from infancy through adolescence: Assessment and intervention* (3rd ed.). St. Louis: Mosby.

Figure-Ground Discrimination

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Definition

Figure-ground discrimination or perception refers to the ability to separate the elements of a visual image on the basis of contrast (e.g., light, dark), to perceive an object (figure) against a background (ground). A classic illustration of figure-ground perception is the Rubin vase, a simple black and white image which can be seen as two dark faces against a white background, or a white vase against a dark background. As well as simple properties, such as light and dark, more complex gestalt properties help separate the important objects in a scene from the background. The ability to discriminate

figure from ground can also refer to the more general capacity to tell foreground from background information, or identify what is important versus what is less salient.

In ASD, superior ability is seen in some tasks requiring identification of parts within wholes, such as the Embedded Figures Task (see entry). This has been interpreted within theories postulating a detail-focused cognitive style in ASD, such as the “weak central coherence” account. Ambiguous figures (e.g., duck/rabbit), of which the Rubin vase may be considered an example, have been used in autism research to investigate flexibility (see ► [“Behavior Observation Scale”](#)) and also self-awareness. Findings are mixed; whether people with ASD show neurotypical patterns of processing ambiguous figures depends in part on whether this is tapped through explicit or implicit tasks (e.g., Allen and Chambers 2011). Links have also been found between ASD-like *traits* and the perception of multiple interpretations of ambiguous figures, perhaps reflecting figure-ground discrimination processes (Best et al. 2008).

See Also

► [Global Versus Local Processing](#)

References and Reading

- Allen, M. L., & Chambers, A. (2011). Implicit and explicit understanding of ambiguous figures by adolescents with autism spectrum disorder. *Autism, 15*(4), 457–472.
- Best, C. S., Moffat, V. J., Power, M. J., Owens, D. G. C., & Johnstone, E. C. (2008). The boundaries of the cognitive phenotype of autism: Theory of Mind, Central Coherence and ambiguous figure perception in young people with autistic traits. *Journal of Autism and Developmental Disorders, 38*, 840–847.

Figures of Speech

► [Figurative Language](#)

Financial Aid

► [FAFSA](#)

Fine Motor Development

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Definition

Fine motor skills are also termed hand skills, fine motor coordination, object manipulation, or dexterity. Components of fine motor development include reach, grasp, release, in-hand manipulation, and bimanual coordination (Exner 2010). Early development of these skills requires integration of the visual motor and somatosensory systems. Fine motor skills initially develop through the infant’s sensory motor exploration, e.g., the infant’s reach and grasp of objects. They are refined as the young child develops functional and pretend play skills that include manipulation, eye-hand coordination, and bimanual skill. In older children and adults, fine motor development involves learning the precise, complex sequences of movement required to perform writing, keyboarding, activities of daily living, leisure pursuits, and vocational tasks. Delays or disorders in fine motor development can relate to low muscle tone or poor strength; impairment in visual motor skills, bimanual coordination, or precision of manipulation (dexterity); and motor learning deficits or dyspraxia (motor planning problems).

Historical Background

In the past, researchers and practitioners did not broadly recognize that children with ASD have impaired fine motor development. The few studies that included motor development noted that motor

skills were a strength in some children, particularly when compared to cognitive and communication skills (Gillberg et al. 1990; Mayes and Calhoun 2003). Using parent report of motor development, Mayes and Calhoun found that 67% of their sample with ASD had normal motor milestones. Early research highlighted the exceptional manipulation skills of certain individuals with ASD, e.g., savants (Hermelin et al. 1994).

Contributing to the confusion about whether or not children with ASD have fine motor impairment, a number of studies found that the motor development of children with ASD was similar to that of children with general developmental delay (Provost et al. 2007), learning disabilities (Miyahara et al. 1997), or language delays (Landa and Garrett-Mayer 2006). One interpretation of these findings is that fine motor delays may reflect general developmental disability rather than define a core feature of ASD. More recent literature confirms that fine motor impairments are not only prevalent in ASD but are long lasting and persistent, affecting function throughout the lifespan (Vanvuchelen et al. 2011).

Current Knowledge

Prevalence

Recent studies and systematic reviews have estimated the extent and prevalence of fine motor delay and motor coordination impairment in children with ASD. Ming, Brimacombe, and Wagner (2007) found high prevalence of motor impairment in a cohort of 154 children with ASD. In young children 2–6 years, 63% exhibited hypotonia, and in children 7–18 years, 38% exhibited hypotonia. Motor planning problems, i.e., Dyspraxia were common (34%) in this cohort.

When prevalence was examined using clinical data from standardized motor development scales, 63–68% of a sample of children with ASD had significant motor delays of more than 25% (Provost et al. 2007). Eighty-four percent of the children demonstrated at moderate fine motor delays. Although the fine motor skills of the young children in this sample were significantly

lower than the typical children and the normative sample, their performance was similar to a comparison sample of children with developmental disabilities. This finding that motor skill development of children with ASD is similar to children with other developmental disabilities is consistent across studies (e.g., Bhat et al. 2011; Landa and Garrett-Mayer 2006).

Using rigorous meta-analysis techniques with 83 studies of motor coordination in ASD, Fournier, Hass, Naik, Lodha, and Cauraugh (2010) examined 51 comparisons of children with ASD to typically developing peers. The standardized mean difference in motor coordination was substantial with an average effect size of 1.2. Given these robust findings, Fournier et al. (2010) concluded that motor coordination deficits are a cardinal feature of ASD.

Infants and Young Children

Beginning in the late 1990s, researchers who analyzed videotapes of infants later diagnosed with ASD reported that the infants showed fine motor differences and stereotypic arm movements (Adrien et al. 1993; Baranek 1999). These retrospective studies revealed that at young ages, children exhibit deficits in specific movement patterns, hypotonia, hypoactivity, and aberrant motor patterns. These retrospective studies motivated researchers to complete prospective studies to examine fine motor skills in young children at risk for ASD (Bryson et al. 2007; Gernsbacher et al. 2008; Landa and Garrett-Mayer 2006).

Prospective studies of infants and toddlers at risk for ASD suggest that motor characteristics may provide a reliable early sign of ASD. When compared to the manual skills of a typically developing sample, those of children later diagnosed with ASD were significantly delayed (Gernsbacher et al. 2008). At 6 months, the infants were less skilled in reaching and grasping of objects, and at 12 months, they were significantly less skilled in stacking, scribbling, and banging. At 18 and 24 months, the toddlers were deficit in the fine motor skills important to communication (pointing) and play (using puzzles and blocks). Although this study identified motor delays in the youngest age groups (6 and 12 months), other

studies have reported that significant differences in fine motor skills among children with ASD do not emerge until 14 months (Landa and Garrett-Mayer 2006). As an early sign of ASD, motor delays or atypical movement patterns can contribute to its early identification.

In preschool age children, 3–4 years, Jasmin et al. (2009) found that 53% had fine motor delays. The mean fine motor composite score and mean scaled scores for the children with ASD were below the normal range, suggesting moderate fine motor impairment. These studies conclude that young children with ASD exhibit mild to moderate delays in fine motor skill and estimate that between 53% and 84% of all children with ASD have fine motor delays or problems.

Based on the assumption that movements (e.g., gestures, oral motor skills, motor imitation) are fundamental to social interaction, researchers (Bhat et al. 2011) suggest that motor impairments may negatively influence children's acquisition of social and communication skills. The correlation of fine motor skills to development of social interaction and communication proficiency needs to be further researched to confirm or refute this hypothesis.

Older Children and Adolescents

Older children and adults with ASD, 7–32 years, have shown impairment in visual motor and manual dexterity tasks (Bhat et al. 2011; Green et al. 2002). When fine motor skills of older children with ASD were compared to children with learning disabilities (LD), they were lower in manual dexterity such as manipulation, drawing, and cutting (Miyahara et al. 1997). Manjiviona and Prior (1995) found that children, 7–17 years with Asperger's syndrome (AS) and high-functioning autism (HFA), demonstrated motor impairment in manual dexterity, including speed and accuracy of hand movement, eye-hand coordination, and bimanual coordination. They also demonstrated more movement errors. Hilton et al. (2007) found that children with Asperger's syndrome scored below the mean on a standardized assessment of motor skill. These findings are consistent with others who found a slower reaction time in

skilled motor tasks (Mostofsky et al. 2000). They suggest that children with AS and HFA have difficulty with motor learning and motor planning.

Children with ASD often demonstrate poor handwriting legibility. When children with ASD were compared to typically developing children on a standardized handwriting assessment, the children with ASD had significantly lower legibility. Specifically, they showed impairments in forming letters, leading to lower overall quality and legibility (Fuentes et al. 2009). One contributing factor to learning legible handwriting is using a consistent preferred hand to write. Frequently, children with ASD are delayed in establishing hand dominance and show mixed laterality or left-hand dominance. Hauck and Dewey (2001) found that children with ASD often do not establish hand dominance and that this lack of preference was not related to cognitive level or lack of motor skills. Studies from the 1980s found that 22–36% of children with ASD do not show a hand preference at ages that hand dominance should be established.

These studies suggest that precision of manipulation, quality of movement, and movement speed are lower in older children with ASD, and these differences can occur in children across the spectrum. The research literature suggests that the majority of children with ASD have significant motor delays or deficits and that these deficits can limit daily living skills, play, communication, and peer interaction.

Motor Imitation

Motor imitation is fundamental to learning and is important to both social and cognitive development (McDuffie et al. 2007). Motor skills are also learned, in part, through a child's imitation of others, i.e., attending to another person's movement and then repeating that movement. The child's goal may be social interaction or may be self-motivated imitation and mastery. Children with ASD have impairments in imitation that are believed to be core to their social impairment (Rogers et al. 2003; Stone et al. 1997). Because these children imitate less, they may have less practice of age-appropriate motor skills. On the

other hand, fine motor delays may be a reason that children exhibit limited motor imitation. When a sample of children with Asperger's syndrome (AS) were compared to a matched sample of children with developmental motor delays, the children with AS had difficulty imitating gestures, particularly when the gesture required symbolic representation of an object (Green et al. 2002). Motor performance using a standardized test accounted for 45% of the variance in imitation performance.

In further investigation of motor imitation, Vanvuchelen, Roeyers, and deWeerd (2011) reported that boys with ASD had problems in motor imitation, whether or not they had cognitive impairment. They concluded that the motor imitation problems were more related to general motor competence than executive function. McDuffie et al. (2007) examined the correlates of motor imitation in children with ASD. They investigated the relationship of attention-following and nonimitative fine motor ability to motor imitation. Both fine motor ability and attention-following accounted for unique variance in motor imitation performance. Whether impaired imitation is a cause or result of lower fine motor skill, it likely relates to and influences fine motor development. Mostofsky et al. (2006) further clarified that motor imitation appears to be delayed but not disordered in children with ASD. Therefore, the delay in acquisition can catch up during late childhood. Previously reported cross-sectional studies of motor development noted that motor delays are common in young children with ASD and less prevalent in older children.

Motor Planning

Motor planning or praxis is the ability to plan and sequence novel or unpracticed movements to reach a goal. A number of studies have identified apraxia or motor planning problems in children with ASD. Using kinematic analysis for reaching, Forti et al. (2011) found that children with ASD were slower in planning their movements and needed to reorient their hand as they reached toward a target. They required more self corrections to accurately reach the target, indicating less automaticity and slower processing. These

findings concur with Rinehart et al. (2001) who found that children with ASD are slow to prepare movement and have impaired anticipatory guidance.

When children with ASD were tested specifically on praxis, e.g., using gestures and tools, they made significantly more errors than children who were typically developing (Mostofsky et al. 2006). This study confirmed earlier findings that high-functioning children with ASD have impaired performance of gestures and problems in motor planning of precise movements (Rogers et al. 1986). Motor planning deficits appear to relate to perception of kinesthetic/spatial aspects of movement (i.e., sensory feedback) and planning of goal-directed actions (Mostofsky et al. 2006). These authors concluded that children with ASD can have deficits in conceptual planning or learning of movement.

Praxis problems or dyspraxia implies that a child has a distinct impairment in planning movement that cannot be accounted for by impaired motor execution or a general movement disorder. To analyze if children with ASD have basic motor skill deficits or dyspraxia, Dziuk et al. (2007) measured performance on a praxis test and on a basic motor skill assessment in a sample of children with ASD, aged 8–14 years. They found that basic motor skill had a significant effect on praxis performance. Although basic motor skills and dyspraxia in ASD were related, the dyspraxia could not be entirely accounted for by basic motor skill deficits. These authors concur with Mostofsky et al. (2006) that praxis deficits in ASD relate to children's perception of sensory feedback from movement, spatial representation of movement, and ability to learn movement.

Stereotypic Movements

In addition to motor delays and motor planning problems, children with ASD may exhibit abnormal movement or motor stereotypies. These include arm flapping or finger flicking and are characterized as repetitive, seemingly non-purposeful movements (Bhat et al. 2011). Retrospective studies of children with ASD have found that toddlers exhibit atypical hand and finger movements and more stereotypical object play,

such as spinning objects. These repetitive movements may interfere with motor development or may create a propensity for stereotypic movements through adulthood (Matson et al. 2009). These motor stereotypies are believed to relate to sensory processing and may serve to lower or raise arousal.

Repetitive movements are also associated with lower levels of overall functioning. Cuccaro et al. (2003) found that children use stereotypic movements for sensory stimulation or as an adaptive function to cope with transitions or changes in the environment. In general, stereotypic movements appear to decrease with age. When useful to support a person's arousal level or ability to cope, stereotypic or repetitive movements may serve an adaptive, functional purpose.

Summary

Fine motor skills are fundamental to social skills and communication and are an important consideration when remediating the core deficits of ASD. A majority of children with ASD have delays in fine motor development, and motor impairments tend to continue through adulthood (Fournier et al. 2010). Fine motor delays are more apparent in young children than in older children, suggesting that motor learning is a factor. Although the severity and prevalence of fine motor deficits appear to improve as children with ASD reach adulthood, motor coordination and motor planning impairments persist.

Fine motor development is among the variables that significantly influence function in children with ASD. Underlying motor delays can impair a child's ability to gesture, imitate, manipulate toys, and participate in social play. In older children, motor planning and motor coordination problems can relate to poor handwriting, school difficulties, and limited participation in sports and recreation. In adults, the fine motor deficits can restrict vocation or leisure pursuits. Researchers recommend that comprehensive intervention programs for persons with ASD include specific strategies to improve fine motor skills (Bhat et al. 2011; Fournier et al. 2010).

Future Directions

Recent studies of children and adolescents with ASD suggest that they present with motor learning and motor planning impairments. It is not clear how the fine motor and motor coordination impairments relate to the cognitive features of ASD. Research is needed to better understand motor skill development and to further analyze how imitation, praxis, and motor learning influence fine motor performance.

With increased understanding of the basis for fine motor deficits, specific interventions can be designed. Efficacy research on motor skill interventions for ASD is scant (see Baranek 2002), and rigorous trials are needed.

See Also

- [Motor Planning](#)
- [Sensory Processing](#)

References and Reading

- Adrien, J. L., Lenoir, P., Martineau, J., Perrot, A., Hameury, L., Larmande, C., et al. (1993). Blind rating of early symptoms of autism based upon family home movies. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 617–626.
- Baranek, G. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders*, 29, 213–224.
- Baranek, G. (2002). Efficacy of sensory and motor interventions for children with autism. *Journal of Autism and Developmental Disorders*, 29, 213–224.
- Bhat, A. N., Landa, R. J., & Galloway, J. C. (2011). Current perspectives on motor functioning in infants, children and adults with autism spectrum disorders. *Physical Therapy*, 91(7), 1–13.
- Bryson, S. E., Zwaigenbaum, L., Brian, J., Roberts, W., Szatmari, P., Rombough, V., & McDermott, C. (2007). A prospective case series of high-risk infants who developed autism. *Journal of Autism and Developmental Disorders*, 37, 12–24.
- Cuccaro, M., Shao, Y., Grubber, J., Stifer, M., Wolpert, C., Donnelly, W., et al. (2003). Factor analysis of restricted and repetitive behavior in autism using the Autism Diagnostic Interview-R. *Child Psychiatry and Human Development*, 34, 3–17.

- Dziuk, M. A., Gidley Larson, J. C., Apostu, A., Mahone, E. M., Denckla, M. B., & Mostofsky, S. H. (2007). Dyspraxia in autism: Association with motor social and communicative deficits. *Developmental Medicine and Child Neurology*, 49, 734–739.
- Exner, C. E. (2010). Evaluation and interventions to develop hand skills. In J. Case-Smith & J. Obrien (Eds.), *Occupational therapy for children* (6th ed., pp. 275–324). St. Louis: Mosby/Elsevier.
- Forti, S., Valli, A., Perego, P., Nobile, M., Crippa, A., & Molteni, M. (2011). Motor planning and control in autism. A kinematic analysis of preschool children. *Research in Autism Spectrum Disorders*, 5, 834–842.
- Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism spectrum disorders: A synthesis and meta-analysis. *Journal of Autism and Developmental Disorders*, 40, 1237–1240.
- Fuentes, C. T., Mostofsky, S. H., & Bastian, A. J. (2009). Children with autism show specific handwriting impairments. *Motor Control*, 10, 244–264.
- Gernsbacher, M. A., Sauer, E. A., Geye, H. M., Schweigert, E. D., & Goldsmith, H. H. (2008). Infant and toddler oral- and manual-motor skills predict later speech fluency in autism. *Journal of Child Psychology and Psychiatry*, 49, 43–50.
- Gillberg, C., Ehlers, S., Schaumann, H., Jakobsson, G., Dahlgren, S. O., Lindblom, R., et al. (1990). Autism under age 3 years: A clinical study of 28 cases referred for autistic symptoms in infancy. *Journal of Child Psychology and Psychiatry*, 31, 921–934.
- Green, D., Baird, G., Barnett, A. L., Henderson, L., Huber, J., & Henderson, S. E. (2002). The severity and nature of motor impairment in Asperger's syndrome: A comparison with specific developmental disorder of motor function. *Journal of Child Psychology and Psychiatry*, 43, 655–668.
- Hauck, J. A., & Dewey, D. (2001). Hand preference and motor functioning in children with autism. *Journal of Autism and Developmental Disorders*, 31, 265–277.
- Hermelin, B., Pring, L., & Heavey, L. (1994). Visual and motor functions in graphically gifted savants. *Psychological Medicine*, 24, 673–680.
- Hilton, C., Wentea, L., LaVerer, P., Itoc, M., Reed, C., & Herzberg, G. (2007). Relationship between motor skill impairment and severity in children with Asperger syndrome. *Research in Autism Spectrum Disorders*, 1, 339–349.
- Jansiewicz, E. M., Goldberg, M. C., Newschaffer, C. J., Denckla, M. B., Landa, R., & Mostofsky, S. (2006). Motor signs distinguish children with high functioning autism and Asperger's syndrome from controls. *Journal of Autism and Developmental Disorders*, 36, 613–621.
- Jasmin, E., Couture, M., McKinley, P., Reid, G., Fombonne, E., & Gisel, E. (2009). Sensorimotor and daily living skills of preschool children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 39, 231–241.
- Johnson, D. P., & Myers, S. M. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120, 1183–1191.
- Landa, R., & Garrett-Mayer, E. (2006). Development in infants with autism spectrum disorders: A prospective study. *Journal of Child Psychology and Psychiatry*, 47, 629–638.
- Manjiviona, J., & Prior, M. (1995). Comparison of Asperger syndrome and high-functioning autistic children on a test of motor impairment. *Journal of Autism and Developmental Disorders*, 25, 23–39.
- Matson, J., Dempsey, T., & Fodstad, J. (2009). Stereotypies and repetitive/restrictive behaviors in infants with autism and pervasive developmental disorder. *Developmental Neurorehabilitation*, 12, 122–127.
- Mayes, S. D., & Calhoun, S. L. (2003). Ability profiles in children with autism: Influence of age and IQ. *Autism*, 6, 65–80.
- McDuffie, A., Turner, L., Stone, W., Yoder, P., Wolery, M., & Ulman, T. (2007). Developmental correlates of different types of motor imitation in young children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47, 401–412.
- Ming, X., Brimacombe, M., & Wagner, G. C. (2007). Prevalence of motor impairment in autism spectrum disorders. *Brain & Development*, 29, 565–570.
- Miyahara, M., Tsujii, M., Hori, M., Nakanishi, K., Kageyama, H., Sugiyama, T., et al. (1997). Brief report: Motor incoordination in children with Asperger's syndrome and learning disabilities. *Journal of Autism and Developmental Disorders*, 27, 595–603.
- Mostofsky, S. H., Goldberg, M. C., Landa, R. J., & Denckla, M. B. (2000). Evidence for a deficit in procedural learning in children and adolescents with autism: Implications for cerebellar contribution. *Journal of the International Neuropsychological Society*, 6, 752–759.
- Mostofsky, S. H., Dubey, P., Jerath, V. K., Jansiewicz, E. M., Goldberg, M. C., & Denckla, M. B. (2006). Developmental dyspraxia is not limited to imitation in children with autism spectrum disorder. *Journal of the International Neuropsychological Society*, 13, 314–326.
- Provost, B., Lopez, B. R., & Heimerl, S. (2007). A comparison of motor delays in young children: Autism spectrum disorder, developmental delay, and developmental concerns. *Journal of Autism and Developmental Disorders*, 37, 321–328.
- Rinehart, N. J., Bradshaw, J. L., Brereton, A. V., & Tonge, B. M. (2001). Movement preparation in high-functioning autism and Asperger disorder; A serial choice reaction time task involving motor reprogramming. *Journal of Autism and Developmental Disorders*, 31, 79–88.
- Rogers, S. J., Herbison, J. M., Lewis, H. C., Pantone, J., & Reis, K. (1986). An approach for enhancing the symbolic communicative, and interpersonal functioning of young children with autism or severe emotional handicaps. *Journal of the Division for Early Childhood*, 10(2), 135–148.

- Rogers, S. J., Hepburn, S. L., Stackhouse, T., & Wehner, E. (2003). Imitation performance in toddlers with autism and those with other developmental disorders. *Journal of Child Psychology and Psychiatry*, 44, 763–781.
- Stackhouse, T. M. (2010). Motor differences in the autism spectrum disorders. In H. Miller-Kuhaneck & R. Watling (Eds.), *Autism: A comprehensive occupational therapy approach* (3rd ed.). Bethesda: American Occupational Therapy Association.
- Stone, W. L., Ousley, O. Y., & Littleford, C. D. (1997). Motor imitation in young children with autism: What's the object? *Journal of Abnormal Child Psychology*, 25, 475–485.
- Vanvuchelen, M., Roeyers, H., & DeWeerd, W. D. (2011). Do imitation problems reflect a core characteristic in autism? Evidence from a literature review. *Research in Autism Spectrum Disorders*, 5, 89–95.

Finger Oscillation Test

► Finger-Tapping Test

Finger-Tapping Test

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Synonyms

[Finger oscillation test](#); [FOT](#); [FTT](#)

Definition

The finger-tapping test (FTT) is a neuropsychological test that examines motor functioning, specifically, motor speed and lateralized coordination. During administration, the subject's palm should be immobile and flat on the board, with fingers extended, and the index finger placed on the counting device. One hand at a time, subjects tap their index finger on the lever as quickly as possible within a 10-s time interval, in order to

increase the number on the counting device with each tap. The original procedure calls for five consecutive trials within a 5-point range for each hand, but variations include a total of six trials, in two sets of three. Results from FTT can be compared to age and gender normative data and may indicate motor impairment or lateralized brain dysfunction. The FTT is included in the Halstead-Reitan neuropsychological test battery.

The FTT may be included as part of a comprehensive neuropsychological assessment for children with Autism Spectrum Disorder. Additionally, the FTT has been used in multiple research protocols as a standardized method for assessing motor speed as it relates to brain functioning and anatomy.

See Also

► [Halstead-Reitan Neuropsychological Test Battery](#)

References and Reading

- Golden, C. J., Espe-Pfeifer, P., & Wachsler-Felder, J. (2000). *Critical issues in neuropsychology: Neuropsychological interpretations of objective psychological tests*. New York: Kluwer/Plenum.
- Hardan, A. Y., Kilpatrick, M., Keshavan, M. S., & Minshew, N. J. (2003). Motor performance and anatomic magnetic resonance imaging (MRI) of the basal ganglia in autism. *Journal of Child Neurology*, 18, 317–324.
- Reitan, R. M., & Wolfson, D. (1985). *Neuroanatomy and neuropathology*. Tucson: Neuropsychology Press.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). Oxford: Oxford University Press.
- Takarae, Y., Minshew, N. J., Luna, B., Krisky, C. M., & Sweeney, J. A. (2004). Pursuit eye movement deficits in autism. *Brain*, 127, 2584–2594.

Finite Mixture Models

► [Latent Variable Modeling](#)

First “Period”

► [Menarche](#)

First Responders and Autism

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Definition

This entry is focused on providing: (1) background on contact between autistic individuals and the criminal justice system and (2) guidance to first responders on issues that may arise in a first contact situation involving an autistic person to enhance their ability to act in an autism-friendly manner.

Historical Background

Although research in this area is limited, there is some evidence to suggest that autistic individuals come into contact with the criminal justice system significantly more often than predominant neurotype (PNT; nonautistic) individuals (King and Murphy 2014). Although autistic people are statistically less disposed to criminality (Mouridsen et al. 2007), they are seven times more likely to be arrested (Curry et al. 1993), sometimes unlawfully. The longer they remain (innocently) in the criminal justice system, the greater the injustice and suffering inflicted upon them. There are also potential negative impacts on the police such as a greater risk of litigation, waste of resources, and negative public relations. There is a clear need for the police, and, in particular,

officers initially on the scene of an incident (first responders), to have a sufficient understanding of autism, how it may present in an individual, and how best to handle incidents involving an autistic person.

So what is autism? Richard Howlin (2003) calls it social dyslexia. In the same way that dyslexia involves difficulty understanding words, the term “social dyslexia” captures the difficulty that all autistic individuals have, to a greater or lesser degree, in understanding social situations. As virtually all situations people find themselves in are social, not fully understanding social behavior can give rise to problems when interacting with other people including first responders. Autism is highly complex for various reasons. Firstly, there is its heterogeneity and extreme diversity whereby one autistic individual is unable to live independently or work whereas another highly intelligent individual is married and working as a university professor. Although the variation in presentation in autism is very wide, all autistic people will have difficulties in understanding PNT social behavior. Autism may also involve what are known as repetitive behaviors (beneficial to the person by introducing some stability and/or being comforting) and “special interests” (fewer, more intense interests than is usually the case for the PNT). Many autistic individuals are hyper- and/or hyposensitive to sensory stimuli (Bogdashina 2016). Many individuals also experience debilitating anxiety (Bejerot et al. 2014). Autism is made even more complex as the majority of autistic individuals have other conditions as well as autism (comorbidities). These may include pathological anxiety, clinical depression, attention-deficit hyperactivity disorder, and obsessive-compulsive disorder (psychiatric comorbidities), and allergies, gastrointestinal problems, migraines, and seizures (medical comorbidities).

Yet another complexity in autism is the ever-changing diagnostic criteria. For many years to come, any of the following diagnoses may come to the attention of first responders: Asperger’s disorder, Asperger’s syndrome, atypical autism, autism spectrum disorder (ASD), “classical” autism, and pervasive developmental disorder not otherwise specified. All first responders need

to know is that these are all attempts by clinicians to diagnose autism so if they are informed that a person they are involved with has one of these diagnoses they know that autism is involved and must respond accordingly.

If an officer is advised by their despatcher that an individual is autistic, or the individual themselves discloses their autism, any interaction requires sensitive handling based on an understanding of autism. In the introduction to his field response tips for first responders encountering a person with autism, Debbaudt writes: "Law enforcement professionals may unexpectedly encounter or be asked to find a person with autism. Recognizing the behavior symptoms and knowing contact approaches can minimize situations of risk - risk or victimization of the person with autism, and risk to the officer" (<http://www.autismriskmanagement.com/autism-articles>). The role of police officers and other emergency service personnel in a first response situation is to avoid a situation where lack of knowledge of autism causes a situation to escalate beyond what would be expected because of a failure to understand autism. The training of all first responders in autism (and other neurodiverse conditions) is therefore essential.

Current Knowledge

Autistic people are in contact with the police as victims, witnesses, and perpetrators. In this respect, they are no different from people without autism. The differences could arise in various ways. Firstly, while contact with law enforcement can be a stressful thing for anyone, people with autism – who tend to experience greater levels of anxiety, and have lower levels of "global stability" (strategies to cope with the "ups and downs" of life reasonably easily) than their PNT peers – are likely to find such contact especially stressful (Beardon 2008; Chown 2010; Debbaudt and Rothman 2001; North et al. 2008). Secondly, there is an ongoing debate as to whether or not persons with autism are less likely to commit crime than those without autism because of the general tendency in autism to comply with rules

and/or are particularly prone to committing certain types of crime (e.g., stalking) because of social difficulties and less well-developed theory of mind (the means by which people make sense of the behavior of others by attributing beliefs, desires, and feelings that motivate actions) than the PNT (King and Murphy 2014).

On the issue of whether autistic people are more or less likely to commit crimes than their PNT peers, Mouridsen (2012) concluded that:

Currently, there is still no body of evidence to suppose that people with ASD are more prone to commit offences than anyone else. However, a small number of serious crimes can be linked to the core features of ASD. Co-morbid psychiatric disorders are important risk factors for offending in people with ASD. (Mouridsen 2012, p. 79)

Although Mouridsen is careful to clarify that there is no valid evidence regarding types of offending that may be associated with autism, he discusses certain types of offending that the limited studies undertaken in this area indicate *may* be linked with autism. The serious crimes he referred to are arson and sexual offending with and without violence. He concluded by repeating that "available findings still indicate that people with ASD are not more prone to committing offences than anyone else" (ibid., p. 85).

Since Mouridsen's review, Sevillever et al. (2013) have reported on sexual abuse and offending in autism. They have been unable to identify any evidence of a greater risk of sexual offending conduct in autistic individuals. They include a useful discussion of factors that may lead to an increased or reduced risk of sexual offending in autism (summarized in the following table) that may be of more general application.

Factors that may increase the risk

Difficulty with social-emotional reciprocity/reduced levels of empathy (PNT individuals who sexually offend also demonstrate lack of empathy for their victims)

Naiveté (interpersonal/exploitation by others)

Sexual frustration and preoccupations/limited intimate relationships

Comorbid intellectual disability (in which case it may be the latter that increases the risk of offending)

Individuals with autism may be more likely to offend against younger children because, given poor social

(continued)

skills, younger children may be easier to interact with than older children or adults

Tendency to engage in private sexual behaviors in public places

Factors that may decrease the risk

The greater supervision of some autistic individuals reducing opportunities to offend

A limited ability to deceive others which may reduce precursor behaviors to sexual offending

A tendency for autistic individuals, especially higher functioning individuals, to be rule-governed could inhibit such offending

Extracted from Sevelever et al. (2013), pp. 192 and 194–195

Where there is a comorbid psychiatric disorder in addition to autism, one has to bear in mind that it may be the comorbidity that is leading to the offending acts, not the autism per se. We know of no research suggesting that an autistic person with any particular comorbid psychiatric disorder is any more likely to commit a crime than a PNT individual with the same psychiatric disorder. Based on their systematic review of the literature, King and Murphy (2014) reported that there was “little” evidence to support the often-heard contention that people with autism are overrepresented in relation to certain crime types. Allen et al. (2008, p. 748) expressed the view that “The apparent association with offending has been in part generated by sensationalised ... media reports.” As some autistic individuals who commit a crime do so without criminal intent, there may be a lower tendency to *knowingly* commit crimes in autism (Beardon 2008).

Autistic individuals are well known to have uneven ability and skills profiles. While a person may be extremely adept in one area, they may present as extremely poor in another. This can lead to erroneous assumptions being made by others – either of ability above and beyond actual capability or incorrectly inferring a skill set is below par. Having an “autism profile” that identifies how autism impacts on an individual on a day-to-day basis can be an invaluable tool to help understand experience from that autistic person’s perspective. It is extremely common for people to make judgments based on behavior, and it is equally common for those judgements to be based on years of ingrained PNT concepts; however, the

application of those judgements onto an autistic person whose behavior results from different cognition is likely to lead to false premises. A good autism profile must include a full sensory profile, i.e., a thorough identification of how the sensory environment impacts on the individual.

Anxiety and poor stress management are common in children with autism. Anxiety levels may increase during adolescence, as young people face an increasingly complicated social environment and often become more aware of their difficulties interacting with others (White et al. 2009). Anxiety levels exhibited by adolescents with autism are significantly higher than those in the general population. Anxiety remains a serious issue for many adults with autism. Gillott and Standen (2007) demonstrated that adults with autism were almost three times more anxious than their comparison group of adults with intellectual disabilities. They wrote that “stress was found to correlate with high anxiety levels for the autism group, particularly the ability to cope with *change, anticipation, sensory stimuli and unpleasant events*” (ibid., 2007, p. 359, *our italics*). While a first response situation, or other contact with the criminal justice system, is likely to be stressful for most people, individuals with autism will often face higher levels of anxiety and stress than their PNT peers. Steps should be taken, as far as possible, to reduce anxiety and stress; this will be of benefit to first responders as well as to the autistic individuals. We have set out various steps that can be taken in a variety of situations to achieve this. However, the most important action an organization can take is to train its officers, and those staff who come into contact with the public, to have an understanding of autism.

The behavior of an autistic individual at times of duress is highly likely to differ from that of a PNT individual. If that behavior is only understood within a PNT context, it is very probable that it will be misinterpreted. At times of increased anxiety, the individual may well present behaviors necessary to avoid a “meltdown.” Many autistic people experience meltdowns. The public often finds it hard to tell meltdowns and temper tantrums apart, but they are different things. A meltdown is an intense response to a situation

an autistic person finds overwhelming. To cope with such a situation, the autistic person may need to engage in repetitive body movements (stimming). Interrupting this may increase levels of anxiety exacerbating the situation. Some possible scenarios that could lead to incorrect initial perceptions on the part of first responders include:

1. *Refusal to engage* – Sometimes, when under duress, the autistic response is essentially to shut off from external stimuli as much as is possible. To the “untrained eye,” the person may appear to be refusing to respond, or excessively rude, or even having something physically wrong with them. But it may be a coping mechanism to reduce their anxiety.
2. *Seemingly excessive reaction to proximity and/or touch* – At times of high anxiety, sensory sensitivities may be greatly increased. This means that if someone is tactile sensitive, for example, then any touch (including very light touch) may be processed as intense pain. In some cases, a “natural” reaction to a perceived assault for some people would be to defend themselves. Even well-intentioned contact meant as reassurance might be perceived as painful. Sometimes an autistic individual can move from a state of anxiety to a state of panic simply because someone such as a first responder is too close to them.
3. *Already in a state of panic* – A single-crewed officer received a call about a man with a knife. She followed the man in her car to allow backup to arrive but soon realized that she knew the man to be autistic and with intellectual learning difficulties and that he was not dangerous. A group of young men had approached him and sent him into a panic. The officer knew that firearms officers and dogs were on the way to the scene, so she knew she had to act quickly and get the knife off him. She explained that other officers would soon arrive who did not know him like she did and was able to get him to throw the knife away. She also agreed not to handcuff him, as she knew it would hurt him, and suggested that they sat in the back of the police car together. The situation had been “defused.”
4. *Eye contact issues* – Social practice assumes a good way of gaining attention is to insist on eye contact and that eye contact reciprocity will lead to a greater sense of trust. Although some autistic people do not have difficulty looking other people in the eye, many do and this difficulty can be exacerbated at times of stress. A first responding police officer may easily misinterpret this as deliberate evasion, a guilty reaction, or disrespect. Of course, as with any other individual, an autistic person can deliberately evade, react guiltily, or show disrespect, but it is far more likely that not looking the officer in the eye is an aspect of their autism.
5. *Flight risk* – A person with autism may try and run away from a situation involving a first responder. Unless an officer has an understanding of autism, they will assume that this is a guilty reaction. It could be a guilty reaction of course but the officer should not assume this as it may be an autistic reaction. The autistic person may have great difficulty with social interaction and experience high levels of anxiety. Their natural response may be to get away from a highly stressful situation without thought as to the impression this creates, nor to the potential consequences of their own actions.

There are other aspects of autism that may lead to misinterpretation of behavior by first responders irrespective of an autistic individual’s level of anxiety. For example:

6. *Sensory processing* – Autistic individuals may only be able to process sensory information through one sensory channel at a time. This is known as single attention (Murray et al. 2005). They may be unable to process auditory information, for example, because they are focusing on visual information. A lack of ability to hear, and subsequently respond to, verbal requests may come across as defiance and/or noncompliance.
7. *Shutdown* – A shutdown is caused by the same factors that cause a meltdown but instead of losing behavioral control the individual stops

reacting to the situation as a means of avoiding it. An autistic former member of the police staff of a major United Kingdom police force found herself in the reception area of an office building when police suspected a terrorist incident was in play. An officer shouted to the people there to get out of the building. This autistic individual “froze.” This was misinterpreted by officers and a difficult situation arose.

8. *Lack of or “odd” facial expression/nonverbal communication* – Some autistic people demonstrate either no facial expression (i.e., the “same” face irrespective of the situation) or a facial expression seemingly at odds with the situation (e.g., smiling when in distress). The misinterpretation of facial expressions could put the autistic individual at risk. First responders should be aware that facial expressions, “body language,” and other nonverbal cues are not always an indication of an emotional state or intent.
9. *Expressive versus receptive language* – For most people, expressive language skills are a reasonable indicator of receptive skills (comprehension). In other words, there might be an assumption that an eloquent person will have equally good comprehension skills. This may not be the case with an autistic person who may display very good expressive language skills but have huge problems with understanding what is being said to them; conversely, they may not speak but have a good understanding when being spoken to.
10. *Feeling obliged to respond* – Remaining with the poor comprehension theme, some individuals will have learnt (usually by being directly taught) that a rule in verbal exchanges is that they *must* respond if asked a question. This places the individual at a huge disadvantage if they feel obliged to respond despite not understanding the question. In an interview situation, this clearly could create numerous problems for an autistic individual.
11. *Difficulty with repeated questions* – An autistic individual may consider that once they have responded to a question that should be the end of that particular matter as they have said what they wanted to say in response and should not be expected to answer the same question again. This may present a problem for officers in a police interview situation where standard interviewing technique may require repetition of certain questions. Officers should be aware that refusal to answer a repeated question may not reflect lack of cooperation.
12. *Multiple concepts or questions at one time* – Many autistic people can get confused when they are asked to deliberate over multiple concepts at the same time or when more than one question is asked at the same time. It is good practice when communicating with an autistic person to ask questions that are clear and “one dimensional” and to do so one at a time.
13. *Literal interpretation* – An autistic person often has a very specific way of understanding language. They may assume a direct correlation between the words being spoken and their meaning (sometimes referred to as literal interpretation). However, many people do not use language in this way, even if they might think they do. Language is full of contradictions and ambiguities and some people’s use of language is more accurate than others. For example, the instructions: “Freeze! Don’t move! Take your hands out your pockets and turn around slowly...” have a clear meaning for most people. But to an autistic person in a stressful situation, it might appear that the person is quite literally contradicting himself or herself and they will not know how to react to what they hear as a confusing set of commands.
14. *Processing language* – Processing time may be considerably longer for autistic individuals than for PNT individuals. This means that after each sentence, for example, an individual might need a few seconds to “digest” what has been said in order to understand it. If this additional processing time is not allowed,

either the person will fall further and further behind the communication – or they will simply miss chunks of it out in order to keep up.

15. *Prosody* – this refers to the tone of voice, inflection, and stressors placed on words – which is often either misunderstood or not taken into account at all by an autistic person. In some cases, the same sentence can have very different meanings dependent on the prosodic expression – in written language grammar along with italics provides the equivalent information. It is important for anyone communicating with an autistic person to take into account that their language (including prosody) needs to be as ambiguity-free as possible. For example, asking “*what* do you think happened” may mean something very different to “what do *you* think happened,” but the prosodic inflection may not be processed by the autistic person.

Some autistic people may break the law as a direct result of the way in which autism impacts upon them, as opposed to having criminal intent (Beardon 2008). Having an understanding of what caused an autistic person to behave in a criminal manner would enable better decisions. For example, an autistic adult who is both socially naïve and desperate for friendship might be persuaded to hoard stolen goods in return for the promise of being included in a social group. Lack of understanding of cause and effect, consequences of actions, difficulty with understanding the intentions of others, naïve levels of trust, difficulty in reading social situations, and accepting language at “face value” are all common characteristics of being autistic and may increase the risk of engaging in activity without knowing that it is criminal activity.

Future Directions

In this section, we discuss the importance of disclosure of a diagnosis of autism, include tips for first responders in a “first contact” situation (courtesy of the autism specialist, Dennis Debbaudt) and for communicating with persons

with autism (including in an interview situation), make some general comments on anxiety levels and sensory sensitivities in autism, and include recommendations for future practice.

Police officers and other first responders come into contact with all types of neurodiversity, not just autism. While clinicians often require hours to diagnose a neurodiverse condition, first responders may have very little time in which to “size up” an individual with whom they are in contact. Even if all first responders received effective training in neurodiverse conditions, they cannot be expected to identify any one of these conditions in a first response situation. This emphasizes the importance of disclosure in such situations. It is appreciated that there are various reasons why an individual may not wish to disclose a diagnosis outside family and friends. However, we consider it important that police forces, other first response organizations, and those advocating for autism and other neurodiverse conditions do all they can to encourage disclosure in first response situations (even by those individuals who otherwise would not disclose) as first responders cannot reasonably be expected to react appropriately in every situation they may face in the absence of disclosure.

Major autism charities in the United Kingdom, like the National Autistic Society, have produced information cards that autistic people can carry and share with first responders. These cards explain that the person they are speaking to is autistic. They also highlight the potential of communication difficulties the autistic person may have during unfamiliar situations. Debbaudt (2002) has developed some guidelines to assist first responders when an individual is known to be autistic.

- Approach in a quiet, nonthreatening manner.
- Turn sirens and flashing lights off.
- Talk calmly in a moderated voice.
- Do not interpret limited eye contact as deceit or disrespect.
- Avoid metaphorical questions that cause confusion when taken literally.
- Avoid body language that can cause confusion. Be alert to a person modelling your body language.

- Understand the need to repeat and rephrase questions.
- Understand that communications will take longer to establish.
- Use simple and direct instructions and allow for delayed responses to questions, directions and commands (Debbaudt 2002).

We would add the following additional guidance to Debbaudt's suggestions:

- Where possible avoid touch as even a light touch may cause pain where a person is hypersensitive. Touching may cause a "flight or fright" reaction; don't assume that means the person is guilty of an offence.
- The cold metal part of handcuffs may cause pain so avoid where possible.

When a person is arrested they are being deprived of their liberty and an officer needs to explain why, in the form of a police caution. The police caution can be complicated to understand so instead of asking a suspect if they understand which may produce a "yes" or "no" response, ask them to explain in their own words what they understood; this could help to identify vulnerable suspects. It is worth noting that in the UK, the Police and Criminal Evidence Act, 1984 (PACE), states that a person should be treated as vulnerable when a custody officer has any doubt about their mental state. Easy read custody sheets that help bridge the gap between a custody officer and a suspect whilst they await an appropriate adult have been piloted in a UK force with positive outcomes for autistic people. Moreover, they take away the uncertainty of what will happen while they are in custody (Parsons and Sherwood 2016).

Attwood (2008) suggests that a number of autistic people find that sensory sensitivity causes them more issues in daily life than the social aspects connected with autism. Hence, understanding what may cause these sensory distractions will help to create a less stressful environment for an interview to take place. For example, the noise of a clock in an interview room may be distracting or cause pain for someone

hypersensitive to noise so consider moving objects out of the interview room that will cause distractions. When there are sensitivities, a holding cell or an interview room accompanied by an officer maybe more appropriate than placing a suspect with autism in a police cell alone. Although some guidance for first responders recommends attempting to move a person in the midst of a meltdown, this should not be attempted; the person can be moved to a quiet area after the meltdown has subsided.

Debbaudt (2002) highlights susceptibility in autistic people during interviews; in particular, when leading questions are asked. As a result of the susceptibility of some autistic people, they may feel that they do not need legal advice and confess to an offence that they did not commit. This is especially true if the questions were not responded to correctly due to misinterpretation. It is good practice to summarize from time to time during an interview to check understanding. If the person is quiet or confused, consider asking them to write their responses down. Allow them extra time to process information. Additionally, direct support from another person, either a family member or an appropriate adult should be considered a reasonable adjustment. An autistic person may also agree to receiving a police caution for a crime they have not committed due to anxiety of going to court and being cross-examined. It is also paramount that the person understands that accepting a police caution results in a criminal record and will affect any criminal conviction checks in the future. It is also noteworthy that in the UK, you can only appeal against your conviction, sentence, or both at a magistrates' court if you pleaded not guilty at your trial (<https://www.gov.uk/appeal-against-sentence-conviction/magistrates-court-verdict>). Therefore, a custody officer's risk assessment for an autistic person should include assigning an appropriate adult to ensure procedures are followed and understood by the detainee. An appropriate adult should be additional to legal representation and should be present during the interview. However, it is only when vulnerabilities are suspected or declared that such provision will be arranged emphasizing the importance of disclosure when in police

custody. Officers should consider taking statements from an autistic victim of a crime in surroundings familiar to them. Explain what being a witness entails and offer support if a case goes to trial.

We have explained that individuals with autism will often face higher levels of anxiety and stress than their PNT peers. Steps should be taken, as far as possible, to reduce their anxiety and stress; this will be of benefit to first responders as well as to the autistic individuals they interact with. We have set out various steps that can be taken in a variety of situations to achieve this. However, the most important action an organization can take is to train its officers (and those staff who come into contact with the public) to have an understanding of neurodiverse conditions since knowledge enhances interaction.

Future operational first responder practice *must* involve adequate training of officers and staff if interactions between first responders and persons with autism are to improve. Hence we conclude this entry with some comments on existing autism awareness training and a set of recommendations. In the United Kingdom, there have been only two studies of autism training provision by first responders – the police service in both cases – by Artingstall (2007) and Chown (2010). However, we have analyzed responses from 34 of the 43 UK police forces to a questionnaire submitted as Freedom of Information Act requests in July 2016, including a question asking for details of autism awareness training. Our data shows that there is no consistency in training provision for autism across the police service in the UK. Only 16 of the 34 respondents provide, or had provided, some form of autism training. The training provided varies greatly and may only be a PowerPoint presentation on a force website, online learning, or a 90 minute in-person session. Only five forces had involved their local autism organization in developing their training. Another force had provided four separate autism courses – each of a full day – in which they had trained almost 1200 officers and staff although almost all this training had been undertaken 4 years ago or more. In the USA, even in a situation where training is mandated by the state legislature

(New Jersey in this case), and the training is delivered on a “minimalist” online basis, a “survey indicated that a significant percentage of emergency service personnel have not completed the state mandated training” (Kelly and Hassett-Walker 2015, p. 9).

The following recommendations are made regarding future first responder autism awareness training (Chown 2010; Debbaudt 2002; Kelly and Hassett-Walker 2015):

1. While online training may have its place, in-person training is arguably far superior, especially where autistic individuals are involved in the delivery.
2. Individuals with experience of first response situations in a professional capacity *and* of autism bring a personal bond and validity to autism training. Officers and staff in first responder organizations with an immediate family relationship with a person with autism represent a potential training resource for their organizations.
3. Studies suggest that lengthier and more diverse approaches to training are often desired and covering all neurodiverse conditions rather than “just” autism.
4. Training should also be conducted jointly between the police, fire, and ambulance services, including discussion groups and role play scenarios, so that each service can contribute their own perspective and learn from the others.

See Also

- [Interpersonal Skills](#)
- [Reasonable Accommodation](#)
- [Social Communication](#)

References and Reading

- Allen, D., Evans, C., Hider, A., Hawkins, S., Peckett, H., & Morgan, H. (2008). Offending behaviour in adults with Asperger syndrome. *Journal of Autism and Developmental Disorders*, 38(4), 748–758.

- Artingstall, S. (2007). What do the police know and understand about ASD? *Good Autism Practice (GAP)*, 8(2), 21–30.
- Attwood, T. (2008). *The complete guide to Asperger's syndrome*. London: Jessica Kingsley Publishers.
- Beardon, L. (2008). *Asperger syndrome and perceived offending conduct: a qualitative study* unpublished doctoral dissertation. Sheffield: Sheffield Hallam University. Last accessed 27 Sept 2016 at: http://shura.shu.ac.uk/7155/1/Beardon_aspergers_-_full.pdf
- Bejerot, S., Eriksson, J. M., & Mörtberg, E. (2014). Social anxiety in adult autism spectrum disorder. *Psychiatry Research*, 220(1), 705–707.
- Bogdashina, O. (2016). *Sensory perceptual issues in autism and Asperger syndrome: Different sensory experiences-different perceptual worlds*. London: Jessica Kingsley Publishers.
- Brugha, T., Cooper, S. A., McManus, S., Purdon, S., Smith, J., Scott, F. J., et al. (2012). *Estimating the prevalence of autism spectrum conditions in adults: Extending the 2007 adult psychiatric morbidity survey*. Leeds: NHS Information Centre for Health and Social Care.
- Chown, N. (2010). 'Do you have any difficulties that I may not be aware of?': A study of auneurotism awareness and understanding in the UK police service. *International Journal of Police Science & Management*, 12(2), 256–273.
- Curry, K. L., Posluszny, M. P., & Kraska, S. L. (1993). Training criminal justice personnel to recognize offenders with disabilities. *OSERS News in Print*, 5(3), 4–8.
- Debbaudt, D. (2002). *Autism, advocates, and law enforcement professionals. Recognising and reducing risk situations for people with autism spectrum disorders*. Jessica Kingsley: London.
- Debbaudt, D., & Rothman, D. (2001). Contact with individuals with autism: effective resolutions. *FBI L. Enforcement Bull.*, 70, 20.
- Gillott, A., & Standen, P. J. (2007). Levels of anxiety and sources of stress in adults with autism. *Journal of Intellectual Disabilities*, 11(4), 359–370.
- Howlin, R. (2003). *Asperger syndrome: Social dyslexia*. Asperger Society of Michigan. Last accessed online 27 July 2016 at: http://www.aspergersmichigan.com/assets/article-Social_Dyslexia.pdf
- Kelly, E., & Hassett-Walker, C. (2015). The training of New Jersey emergency service first responders in autism awareness. *Police Practice and Research*, 1–12.
- King, C., & Murphy, G. H. (2014). A systematic review of people with autism spectrum disorder and the criminal justice system. *Journal of Autism and Developmental Disorders*, 44(11), 2717–2733.
- Mouridsen, S. E. (2012). Current status of research on autism spectrum disorders and offending. *Research in Autism Spectrum Disorders*, 6(1), 79–86.
- Mouridsen, S. E., Rich, B., Isager, T., & Nedergaard, N. J. (2007). Pervasive developmental disorders and criminal behaviour: A case control study. *International Journal of Offender Therapy and Comparative Criminology*, 52(2), 196–205.
- Murray, D., Lesser, M., & Lawson, W. (2005). Attention, monotropism and the diagnostic criteria for autism. *Autism*, 9(2), 139–156.
- North, A. S., Russell, A. J., & Gudjonsson, G. H. (2008). High functioning autism spectrum disorders: An investigation of psychological vulnerabilities during interrogative interview. *The Journal of Forensic Psychiatry & Psychology*, 19(3), 323–334.
- Parsons, S., & Sherwood, G. (2016). Vulnerability in custody: Perceptions and practices of police officers and criminal justice professionals in meeting the communication needs of offenders with learning disabilities and learning difficulties. *Disability & Society*, 31(4), 553–572.
- Sevlever, M., Roth, M. E., & Gillis, J. M. (2013). Sexual abuse and offending in autism spectrum disorders. *Sexuality and Disability*, 31(2), 189–200.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229.

First Words Project

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Definition

The First Words Project is a clinical and research program, staffed by speech pathologists, researchers, and interdisciplinary consultants, aiming to investigate developmental communication disorders while providing services for young children with communication delays and their families.

An overarching goal of the First Words Project is to emphasize the importance of early screening and diagnostic methods to detect social-communication delays and disabilities in young children and to provide assessment and intervention to children and families of toddlers with delayed development who otherwise may have not had access to knowledge of the need for services.

Historical Background

Led by Amy Wetherby, Ph.D., The First Words Project conducts longitudinal research in the Autism Institute in the College of Medicine at Florida State University. The goals of the project are to identify early red flags of autism spectrum disorders and other developmental disorders in children under 2 years of age, to develop and improve early screening tools and early detection of communication disorders, and to provide training and support to families of children with ASD and other developmental disorders affecting communication.

A number of early intervention screening and assessment recommendations have been developed based on the research and work with children and families since the beginning of the First Words Project, including the use of the Communication and Symbolic Behavior Scales (Wetherby & Prizant, 2001) to identify children 6–24 months of age at risk for developmental disabilities, including autism.

The First Words evaluation model follows two-step process designed to involve participating families, thereby reducing the need for clinicians and in-home providers. The goal here is twofold, providing parents and caregivers with training and resources for developing communication and social skills in the home and reducing costs surrounding early intervention home services.

Current Knowledge

First Words Project receives national and private funding to evaluate the communication development of children from 6 to 24 months of age. For children who are delayed in communication, intervention services are offered at no cost to families or service providers, based on availability of grant funds.

Future Directions

First Words is also developing educational materials and assessment tools for training of

healthcare and childcare providers to improve early detection of communication problems in young children.

The First Words Project continues efforts to disseminate research, clinical tools, and information to all those involved in serving children with developmental disabilities and autism, including parents and caregivers. Online educational tools such as the *ASD Video Glossary*, an innovative web-based video application, are available to help parents and professionals learn more about the early red flags of autism spectrum disorders.

This ASD Video Glossary is available free of charge through the First Words Project website as well as the Autism Speaks website. The ASD Video Glossary contains over 100 video clips. Each video highlights diagnostic areas important to assess and consider when diagnosing ASD and/or differentiating ASD from other common childhood developmental delays. Each section of clips contains both child examples and short tutorials to help viewers detect subtle differences between typical and delayed development in young children and to spot the early red flags for ASD. As per the First Words Project website, all of the children featured in the *ASD Video Glossary* as having red flags for ASD are, in fact, diagnosed with ASD.

See Also

- [Communication and Symbolic Behavior Scale](#)
- [Early Diagnosis](#)
- [Early Intervention](#)

References and Reading

Wetherby, A. M., & Prizant, B. M. (2001). *Communication and symbolic behavior scales developmental profile/infant toddler checklist*. Paul H. Brookes Publishing Co.

Project Homepage

First Words Project. (2007). Early red flags of autism spectrum disorders. Retrieved from <http://firstwords.fsu.edu/>

Resource for Clinicians, Parents & Students: ASD Video Glossary (Free of Charge)

ASD video glossary also available at the Autism Speaks website: <http://www.autismspeaks.org/video/glossary.php>

ASD video glossary. <http://firstwords.fsu.edu/ASDglossary/ASDabout.html>

Fixed Interval

► Fixed Ratio

Fixed Ratio

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Synonyms

[Fixed interval](#)

Definition

Fixed ratio is a schedule of reinforcement. In this schedule, reinforcement is delivered after the completion of a number of responses. The required number of responses remains constant. The schedule is denoted as FR-#, with the number specifying the number of responses that must be produced to attain reinforcement. In an FR-3 schedule, 3 responses must be produced in order to obtain reinforcement. In an FR-15 schedule, 15 responses must be emitted before reinforcement is delivered. This ratio requirement (number of responses to produce reinforcement) is conceptualized as a response unit. In other words, it is the response unit (not the last response) that leads to the reinforcer (Cooper et al. 2007; Skinner 1938).

Applications of FR schedules can be found in business and in education. Some tasks are paid on an FR schedule (e.g., piecework). Students might

receive a token after the completion of ten spelling words.

FR schedules are associated with a particular pattern of responding. After the first response, there is generally little hesitation between responses and the required responses are completed. After the ratio requirement is met, reinforcement is delivered. This is followed by a postreinforcement pause, in which the participant does not respond for a period of time following reinforcement. The size of the ratio influences the length of the pause. Duration of the postreinforcement pause is greater for large ratio requirements. Shorter pauses occur with small ratio requirements. The FR schedule is visually depicted as a stepwise pattern.

FR schedules produce high rates of responding. It is associated with rapid response speed, since completion of the ratio requirement leads to reinforcement. In general, higher ratio requirements produce higher rates of responding (to gain more reinforcement). If the ratio requirements are too stringent, however, the rate of response can decrease. The maximum ratio must be individually determined and continually monitored. The determination of the maximal value is influenced by motivating operations, the individual's reinforcement history, and the quality of the reinforcer.

See Also

► [Schedule of Reinforcement](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Skinner, B. F. (1938). *The behavior of organisms*. New York: Appleton.

Flat Prosody

► [Monotone](#)

Flexibility

► [Treatment for Higher-Order Restricted, Repetitive Behaviors](#)

See Also

- [Attention](#)
- [Linguistic Idiosyncrasies and Neologisms](#)
- [Mania](#)

Flight of Ideas

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Synonyms

[Derailment](#); [Loose associations](#); [Tangentially](#)

Definition

Flight of ideas is a formal thought disorder. It refers to the expression of rapidly shifting thoughts in an individual. Thoughts are expressed through language. In individuals with “flight of ideas,” thoughts are expressed in a highly associative manner. These associations may be linked to cue in the surrounding environment, elicited by associations stemming from the topic or merely by words. The individual’s speech becomes incomprehensible, because he does not tune into the listener’s needs by providing the listener with information that prepares him for changes of topic of conversation, thus making these better understandable.

Flight of ideas may occur in the course of a manic episode, during a psychosis, but is not uncommon in individuals with an autistic disorder of multiple complex developmental disorder. In the latter individuals, the question is raised whether this is really a thought disorder or a communicative deviance more related to expressive language disorder in these individuals. Others (Raymond Lake 2008) consider thought disorder as an expression of mood disorder.

References and Reading

- Caplan, R. (1994). Thought disorder in childhood. *Journal of the American Academy of Child and Adolescent Psychiatry*, 33(5), 605–615.
- Kowatch, R. A., Youngstrom, E. A., Danielyan, A., & Findling, R. L. (2005). Review and meta-analysis of the phenomenology and clinical characteristics of mania in children and adolescents. *Bipolar Disorders*, 7(6), 483–496.
- Raymond Lake, C. (2008). Disorders of thought are severe mood disorders: The selective attention defect in mania challenges the Kraepelinian dichotomy a review. *Schizophrenia Bulletin*, 34(1), 109–117.
- Solomon, M., Ozonoff, S., Carter, C., & Caplan, R. (2008). Formal thought disorder and the autism spectrum: Relationship with symptoms, executive control, and anxiety. *Journal of Autism and Developmental Disorders*, 38(8), 1474–1484.
- Van der Gaag, R. J., Caplan, R., van Engeland, H., Loman, F., & Buitelaar, J. K. (2005). A controlled study of formal thought disorder in children with autism and multiple complex developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 15(3), 465–476.

Floor Effect

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Definition

The floor effect is a statistical phenomenon in which most data points fall in the very low range of possible values (“bottom out” on the “floor” of the measure). The floor effect is often seen in assessment when a test is too challenging for a given target population. In turn, many subjects obtain scores that are clustered together at the bottom of a measure, with very few extending

across the possible range of scores. This results in a skewed distribution with very limited variability. Floor effects greatly limit the clinical utility of measures. For example, if there are floor effects on a measure prior to and following an intervention designed for individuals with ASD, it will not be possible to observe any actual benefits that might have occurred for participants.

See Also

► Ceiling Effect

References and Reading

- Everitt, B. S. (2006). *The Cambridge dictionary of statistics* (3rd ed.). Cambridge, UK: Cambridge University Press.
- Jackson, S. L. (2011). *Research methods and statistics: A critical thinking approach* (4th ed.). Belmont: Wadsworth.

Floor Time/Circle of Communication

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Definition

Floortime is one component of the Developmental, Individual-Difference, Relationship-based (DIR[®]) approach for engaging children with autism spectrum and related socioemotional disorders. The DIR[®] model provides an interdisciplinary approach for practitioners, educators, and families to comprehensively assess and provide intervention according to the unique developmental profile of the child. Floortime focuses on creating opportunities of connection between the care-provider and student, fostering learning through emotionally engaging and meaningful interactions while encouraging mastery of six

foundational stages of social, emotional, and intellectual development (Greenspan and Wieder 2006). Previous implementation suggested the ideal targeted population to be in the early toddler years; however, more recently the authors have expanded the target population to include older children and adolescents who have developmental differences. The initial goal of Floortime is to discover individual differences in sensory processing, language, motor, visual, and intellectual capacities, as well as identify the child's view of the world by attending to the child's interests, sensory modulation and processing, motor planning, and symbolic formulation and use. The practitioner then enters into the child's world by imitating or following the child's lead, attempting to draw the child into a shared interaction (Davis et al. 2014). Once an initial affective interaction is established, the clinician encourages a response, creating a *circle of communication* marked by an increase in intentional or purposeful two-way communication.

When the foundational skills of opening (initiating) and closing (responding to) circles of communication have been established, more complex areas of social, emotional, and intellectual capacities are fostered by creating small obstacles or variations during Floortime (Greenspan and Wieder 1997). The DIR[®] approach, of which Floortime is a part, emphasizes individualized early intervention to treat the developmental differences often observed in autism (Landa et al. 2007) in consideration of the child's unique presentation of social, cognitive, and processing deficits.

Historical Background

Stanley I. Greenspan first developed and presented the DIR[®]/Floortime approach for treating children with developmental and emotional disorders in 1992. Greenspan and colleagues, including, Serena Wieder, drew from interventions across disciplines to hone and perfect a comprehensive and child-led approach for intervening with nonprogressive developmental disorders, such as autism spectrum disorders (ASD), from a

functional developmental framework instead of treating individual symptoms (Greenspan and Wieder 1999).

The development of the DIR[®]/Floortime model has as its foundation the most influential research in the field of developmental psychology (ICDL 2000) dating back to the early work of Jean Piaget (1896–1980) and Lev Vygotsky (1896–1934). Piaget hypothesized that there was a typical progression through human development whereby babies construct or build on innate “schemas” about the world as they progress through four stages of cognitive development. This theory, which became known as the constructivist view of learning, suggested that children have an innate or intrinsic motivation to interact with others and play, gathering and constructing knowledge about the world, through which they form the foundation for understanding abstract and symbolic representations of the world (Huitt and Hummel 2003; Prelock 2006). The significance of play in learning and development was also an idea shared by Vygotsky; however, he suggested a more sociocultural view of cognitive development (Prelock 2006), as he posited that it was through the continued interaction with others that developmental changes occurred in individuals (Cole and Scribner 1978).

In 2004, Greenspan and Shanker suggested that while play was important for general development, emotions and, more specifically, affective signaling between communicative partners during play directly allowed for individuals to develop their “first idea,” a mental construct or representation of something intangible. They hypothesized that through the development of emotional understanding and reciprocal signaling, play and social interaction lead to symbolic reasoning and higher-order cognition. Drawing from the literature and research on the core deficits defining ASD, they developed a model of functional developmental capacities that when absent or reduced led to decreased formation of the foundations for relating, communicating, and thinking.

Historically, treatment approaches for ASD follow two models for intervention: behavioral approaches for reducing maladaptive or inefficient behaviors and cognitive approaches for

training individual skills according to the absence or presence of expected abilities at a given age (Greenspan and Wieder 2006). In the development of the DIR[®]/Floortime model, the authors defined the core goal areas based on relevant areas of developmental functioning that may or may not have been impaired in a given disorder. They reasoned that the very foundation of a comprehensive treatment program was to build upon a core set of skills that are requisite for the development of more broad and higher-cognitive skills. Using the authors’ experience in emotional and social development, and their focus on treating a multifaceted neurodevelopmental disorder with interventions across several disciplines, they suggested the DIR[®] model, which included all relevant and critical areas of development, including systems responsible for emotional, social, language, motor planning, and sensory processing and modulation.

Rationale or Underlying Theory

The development and expansion of play skills has gained recognition as serving an integral role in the acquisition of cognition, language, emotional intelligence, and social skills in children (National Research Council [NRC] 2001; Prelock 2006). Further, it has been suggested that a connection with an adult, during play and social routines, scaffolds and informs the child’s ongoing development of meaning, beliefs, and values for the social context, thereby influencing the child’s ability to expand his/her knowledge through experiences with objects, actions, and events (Greenspan and Shanker 2004; Greenspan and Wieder 2006; NRC 2001; Prelock 2006). As intervention techniques begin to broaden and include more intensive and early intervention programs, some suggest a developmental and relationship-based approach may be more effective for treating children with ASD. *Development*, *Individual Differences*, and *Relationships* are three integral aspects of the Floortime approach, which purport to form a strong foundation from which several strategies can be used to create attuned relationships, which thereby foster continued brain

development. *Development* refers specifically to a symbolic ladder, corresponding to a typical developmental trajectory of social emotional capacities, which successively lead to the development of intimacy, mutuality, intentionality, empathy, creativity and resilience. *Individual Differences* reflect the importance of the acknowledgment, examination, and integration of differences across how individuals functionally engage in sensory registration, modulation, and interpretation; and language and motor processing; as well as interpret related concepts. *Relationships* are defined as the development of warm, flexible, sensitive, and open adult-child connections leading to finely attuned relationships, which provide the firm foundation to support neural integration, socio-emotional, and sociocognitive growth (Davis et al. 2014).

Limited ability to relate and emotionally connect within the social context is inherent in the core deficits often observed in ASD and related sociocommunicative disorders. This limitation, coupled with a reduced or lack of initiation and/or attention toward others, restricts the child's ability to form ideas and expand their knowledge of reciprocal and symbolic play (Greenspan and Wieder 2006; Mundy et al. 1990; Prelock 2006). Greenspan and Shanker (2004) refer to the early development of a "first idea" as a primitive construct that leads to the integration of symbolic, linguistic, and intellectual knowledge. This, in turn, allows for the child to appreciate the ever-changing social context and navigate the dynamics of play leading to increased interpersonal communication.

In 1997, Greenspan and Wieder presented a paradigm of Functional Developmental Levels (FDLs) that reflected expected capacities in relating, emotional understanding, and symbolic representation throughout six stages of typical development. They posited that typical development progresses through successive mastery of each stage and is essential for normal development of emotional reasoning, a matured sense of self as well as higher-level cognitive development. Later these stages were expanded to include some higher-level aspects of emotional understanding, affective reasoning, and cognition, and

are now referred to as Functional Emotional Developmental Capacities (FEDCs) (Greenspan and Wieder 2006). Consequently, for the purpose of clarity, they will be referred to as FEDCs and are described in more detail below.

Stage one of the FEDCs is the capacity for *mutual or shared attention* or the ability to regulate attention and behavior while monitoring and processing sensory information from the environment and one's own body. Self-regulation is one component of this stage whereby a child learns the association between a physical state and an emotion. Another component is the emergence of joint attention, typically occurring between 6 and 9 months of age, although children begin processing sensory information shortly after birth. The capacity for joint attention is also viewed as a prerequisite skill for later language and communicative development (Mundy et al. 1990). Successful joint attention between caregiver and child begins to foster the child's knowledge for discriminating pleasurable from unpleasurable experiences (Greenspan and Wieder 2006). It is through initial sensory processing paired with increased attention to others during mutually shared experiences that a child's emotional understanding begins to deepen.

Stage two typically emerges between 2 and 5 months of age and is marked by *engagement and relating with others*. As a child's ability to engage in emotional interactions increases, so does the ability to discriminate between an interaction with another social being and an inanimate object. Through this discrimination, the child recognizes patterns about his/her world and demonstrates an emerging ability to assign meaning to various acts of communication and symbolic representation. During this stage, a child's genuine desire for engaging and relating begins to emerge, without signs of distress or withdrawal, affording more opportunities for increased reciprocal communication.

The third stage, *intentionality and two-way communication*, is marked by the child beginning to interpret emotions as "signals" of communication. By 6 months of age, caregivers and babies begin to engage in intentional or purposeful communication where caregivers read and respond to

children's emotions and, in turn, require them to read and respond to theirs. The result is a back-and-forth flow of emotional signaling, which is referred to as a "circle of communication." As the child approaches 8 months of age, circles of communication are mastered and become longer and more frequent, expanding the child's understanding of interactions as following a causal and logical pattern, mimicking the turn-taking pattern of more complex and later developing skills such as in maintaining a conversation.

Between 9 and 18 months of age, children begin to master the fourth stage of the FEDCs, *social problem solving, mood regulation, and formation of a sense of self*. In this stage, two-way reciprocal communication is used to solve problems and achieve a desired goal (e.g., pointing to direct others' attention or to obtain a desired object). Developing emotional maturity at this stage allows for an increase in *shared social problem solving*, where a child considers not only his/her own thoughts and beliefs but also the thoughts and desires of another individual and engages in negotiations during play and communication. Further, the emergence of skills in *regulating mood and behavior* allows a child to rapidly recognize and adjust his/her emotional signals. It is during this stage that children begin to develop an understanding of cause and effect and become more aware of complex patterns of communication leading to an early *sense of self*. They begin to refer to themselves as "I" or "me" and others as representing "you." This sense of self and others leads to early empathetic reasoning or a theory of mind.

By one and half years of age, the child's development progresses into the fifth stage of FEDCs, *creating symbols and using words and ideas*. Through joint attention and complex emotional signaling the child learns to separate perceptions from actions and begins to pair mental constructs, or images, with a linguistic or gestural symbol. Communication becomes meaningful and pretend play becomes more imaginative (e.g., a block as a car or a pan as a hat). A better understanding of the function of words and symbols emerges, and a child learns that words and actions communicate ideas, convey mental and physical states, and signal intent.

As a child progresses through the second year of life, at approximately two and a half years of age, he/she enters into the sixth stage of FEDCs, *emotional thinking, logic, and a sense of reality*. The child begins to connect symbols and ideas in a logical manner. The ability to link an internal to an external idea emerges as well as a child's sense of events across time. As the child's logical thinking expands, higher-cognitive skills emerge such as inventing characters or games and following the rules of another's game.

Assessment of the child's functional capacity at each of the aforementioned stages forms the core of the DIR[®] model and further informs treatment at each stage through child-centered, interactive, and guided Floortime sessions. At the heart of the intervention is the premise that through maturing emotional development, a child learns to construct his/her knowledge of the world, learning to construe multiple representations or symbols for events, eventually leading to reciprocal social competence through an understanding of the self and others (Greenspan and Wieder 2006).

Goals and Objectives

Two common goals are at the heart of the Floortime approach: (1) following the child's lead, identifying that child's unique processing style and/or interests in the environment, and (2) bringing the child into a shared world, using two-way intentional and reciprocal interactions (Greenspan and Wieder 2006). In addition, three cornerstones have been identified as essential to participation in the program. First, relationships are formed through emotional and meaningful interactions and are the context for learning that leads to the development of language, cognition, and social skills. Second, motor and sensory processing capacities are varied in children, and these variations affect the emotional processing that goes along with interpreting and modulating sensory information. In essence, children vary in the way they process information, thereby making their availability for learning and relating individually unique. Finally, progress across all developmental areas should be viewed as interrelated.

Considering these three cornerstones the main goal of the Floortime approach is to increase a child's capacity for thinking of themselves as intentional and interactive individuals as they progress through mastery of the six FEDCs described above (Greenspan and Wieder 2006). The initial step is to administer a comprehensive interdisciplinary assessment of the child's developmental capacities within the context of his/her unique sensory processing profile, the dynamic of his/her family relationships, as well as his/her interactive patterns (Greenspan and Wieder 1999). A functional developmental profile is derived from clinical observations of interactions with both clinicians and parents, chart reviews, interviews with other care providers, assessments across disciplines (speech-language pathology, occupational therapy, physical therapy, etc.), a developmental history, a review of current programs, as well as a biomedical evaluation.

Following the generation of a functional developmental profile, a comprehensive DIR[®] intervention program is developed with goals that are individualized to the unique profile of the child while considering functioning across the six areas of functional emotional development. The foundational goal is meeting the child's need for a connection with others by providing stable and secure relationships with providers. Upon the foundation of ongoing emotional interactions with consistent care providers, individualized therapeutic goals can be designed to specifically address the child's functional developmental level, his/her individual differences in sensory processing, motor planning, and sequencing, as well as specific skills in the areas of speech and language and general education. Lastly, the authors highlight the importance of continual team and family consultation as well as ongoing assessment of core deficits as well as the functional developmental capacities, which further informs the progression and predicted outcome of an individualized DIR[®] program.

Treatment Participants

The DIR[®]/Floortime approach was designed for children and adolescents with developmental

differences, specifically, socioemotional deficits such as those often observed in ASD. It is appropriate for individuals who are verbal or nonverbal and with a diagnosis of ASD (Ingersoll et al. 2005) or related sociocommunicative disorders. While there are no prerequisite skills necessary to begin a DIR[®] program, researchers have found that individuals who benefit most from this treatment tend to present as having a specific profile, quite possibly allowing them to be more capable of learning to relate and interact with intentionality and symbolically. The children who have a more positive prognosis with the DIR[®] approach are those that have demonstrated some capacity for joint attention and an emerging ability for using gestures in an affective interaction. In addition, they typically demonstrate some form of functional language use or some degree of pretend play (Greenspan and Wieder 1997). Lastly, while this approach has typically been implemented with toddlers, recognizing early intervention leads to better outcomes for children who are identified early as having developmental delay (Landa et al. 2007), it can also be used with older adolescents with autism who have some functional capacity at each of the six FEDCs, but continue to struggle with higher-level thinking. For older children, this model is typically implemented within a school program or in community environments that purport to foster and facilitate social interaction (Greenspan and Wieder 2006).

Treatment Procedures

A comprehensive DIR[®] program typically involves a home-based program, an educational setting-based program, as well as the addition of specialized therapies such as speech and language, occupational, physical, and/or certain biomedical therapies, depending on the needs of the child (Greenspan and Wieder 2006). The mean age for entry into a DIR[®] program is typically 36 months, ideally beginning as early as 22 months (NRC 2001) or immediately following identification of key strengths and challenges in affective reasoning, sensory modulation and

processing, symbol formulation, and motor planning through a comprehensive, interdisciplinary assessment. However, this approach is also used for older children and adolescents who demonstrate developmental differences, particularly in the area of social emotional relatedness. Floortime training and mentoring can be implemented in the home or clinical setting by a trained professional in the discipline of speech-language pathology, psychology, or a person with training and expertise in the application of the Floortime approach. Eventually parents are trained to implement these techniques as well. Duration of intervention ranges from 10 to 25 h per week (NRC 2001). One pilot study has suggested that implementation of the approach by parents for as little as 15 h a week can potentially produce both positive outcomes and remain cost-effective for families (Solomon et al. 2007). However, one limitation noted that, for children whose parents spent less than the prescribed time implementing the program, there was less progress in the functional developmental level observed, supporting the importance of duration and frequency of treatment, as suggested by the NRC (2001).

The most common technique applied in the DIR[®] model is Floortime interactions, which occur both at home between clinician and child as well as between parent and child. These sessions are designed to be approximately 20 minutes per session and are ideally spontaneously interspersed throughout the day for a frequency of at least eight sessions. Once the child has mastered the initial functional developmental capacities required for interaction (e.g., joint or shared attention and can complete multiple circles of communication), peer play sessions can be introduced to facilitate shared problem solving, play negotiation, and the exchange of symbols, gestures, or words. Additional structured and semi-structured sessions are implemented to address specific skills in the areas of motor, sensory and spatial skills, balance and coordination, and visuospatial functioning.

Several strategies specific to DIR Floortime have been identified as techniques for promoting therapeutic relationships which in turn foster continued functional social emotional and

sociocognitive development. These are outlined in *Floortime Strategies to Promote Development in Children and Teens* (Davis et al. 2014). The highlighted strategies include, but are not limited to, intervention techniques to support: the attunement of relationships, securing connections, responding and expanding to reciprocal bids for communication, fostering pretend and symbolic play, and challenging and reflecting upon interactions to foster more complex thinking.

There is a clear interdisciplinary and collaborative effort inherent in the DIR[®] approach, allowing a plan to be individualized to each child's unique functional developmental profile. Specialized therapeutic services, such as speech-language pathology, occupational, and/or physical therapy, are implemented, as needed, in addition to Floortime sessions. Several components of the DIR[®] model, however, are implemented or facilitated by parents. The affect-based curriculum is one such component, which is specifically designed to include highly motivating semi-structured, structured, and spontaneous activities that are based on a common goal of sharing affect and attention while simultaneously focusing on the foundations of language, such as phonology, syntax, grammar, and semantics (Greenspan and Wieder 2006).

Efficacy Information

Efficacy of the DIR[®] model has been measured using the *Functional Emotional Assessment Scale* (FEAS) for the child and the caregiver, by clinical measurement of the Functional Emotional Development Level (FEDL), or the child's functioning according to the six developmental levels suggested by Greenspan and Wieder (1997) as well as in overall presentation and severity of symptoms in autism. In a retrospective chart review of 200 cases conducted by Greenspan and Wieder (1997), it was reported that children's progress could be organized into three outcome areas of the intervention. Fifty-eight percent of the cases were interpreted as having *good to outstanding* outcomes, 25% of the cases had *medium outcomes*, and 17% demonstrated *ongoing*

difficulties. The conclusions of this review suggest that a small subgroup made progress in learning to relate to others, communicate, think creatively and abstractly, and showed an increase in emotional understanding and empathy, while other cases, measured as having more severe symptoms, continued to have difficulties despite intervention. Limitations noted in this chart review included variability in the assessment procedures used for the 200 children, the limitations associated with a case study design, and a small sample size, therefore suggesting that these results could not be definitively generalized to the greater population with ASD.

Later, a 10–15-year follow-up of a subgroup of children with ASD who received the DIR[®] intervention program was conducted by the same authors. This study revisited 16 in the subgroup of children who were reported to have *good to outstanding* outcomes from the earlier chart review of 200 cases (Greenspan and Wieder 1997). Conclusions from the follow-up study indicated that most individuals from that subgroup maintained the progress in the areas of social relatedness, creative thinking, and empathy, suggesting that early implementation of the DIR[®] approach may have effects later into adolescence. Limitations included questions about the generalizability to the larger population of children with ASD, the sample size, and extraneous factors that could not be controlled for, such as other interventions implemented across the years (Wieder and Greenspan 2005).

In 2005, Ingersoll and colleagues examined the effects of the Developmental Social-Pragmatic model (DSP) on the use of spontaneous functional communication of three children with ASD between the ages of 2 and 3 years of age. Through a single subject-multiple baseline design, trained therapists implemented a DSP model of treatment, which included the DIR[®]/Floortime approach, Hanen program, SCERTS model, and responsive teaching approaches. The children received treatment twice weekly for 10 weeks, followed by a 1-month follow-up to assess generalization. The authors found that two of the three children demonstrated gains in spontaneous language use after treatment began that was maintained and

generalized across new treatment settings. The third child exhibited gains in spontaneous communication; however, he also demonstrated an increase in his baseline, thereby confounding the interpretation that his gains could be solely attributed to the DSP intervention. The conclusions of this study speak to the strength in the application of this research model and support evidence for DSP models implemented by specialists in the field of speech and language as well as the use of spontaneous functional communication as a preferred outcome measure. In addition, progress in spontaneous communication following the use of the DSP model for both verbal and nonverbal children with ASD was found. Limitations included the absence of a follow-up session for one child, a failure to explore generalization to more naturalistic settings, as well as an inability to control for the therapists being the sole providers of the DSP approach throughout the duration of treatment.

Further support for the DIR[®]/Floortime approach was found by Solomon et al. (2007) who conducted a pilot study that focused on training and consultation for parents implementing the Floortime approach across an 8- to 12-month program. Their study found significant gains for 45.5% of the children enrolled as indicated by an increase in their overall FEAS score. In addition, no significant changes in the caregivers FEAS scores were observed, suggesting that parent's participation was appropriate for implementing the relationship-focused intervention. Mahoney and Perales (2005) also reported that the parent implementation of the relationship-based intervention model for children with pervasive developmental disorder (PDD) was effective, encouraging the caregivers to provide more responsive interactions with their child, which in turn led to more than two thirds of the children demonstrating an increase in their targeted developmental behaviors. The research published on the DIR[®] model suggests that parent education, training, and provider roles are factors that positively influence the outcome of changing pivotal behaviors in children treated with this approach (Mahoney and Perales 2005; Solomon et al. 2007). Another factor affecting the outcome of

treatment is the amount of time spent with the child implementing Floortime (NRC 2001; Solomon et al. 2007).

By 2010 there was some research supporting positive outcomes for a subgroup of children participating in early DIR[®] intervention, with most research including case studies and chart reviews, which indicated the need for further and more stringent research models. Based on the National Autism Center's first phase of the National Standards Report (2009), developmental and relationship-based approaches were considered to have emerging evidence, further highlighting the need for more research examining the outcomes of such programs for addressing the individuals with ASD. In 2015, Phase Two of the National Standards Report (2015) was published providing further confirmation that the DIR/Floortime model has emerging evidence, suggesting that approximately seven studies indicate beneficial intervention effects for a set of interventions categorized as developmental and relationship-based approaches; however, more rigorous research is still needed to draw firm conclusions regarding the overall efficacy of this model.

In 2011, a pilot randomized controlled trial, conducted by Pajareya and Kopmaneejumrulers, focused on parent training and confirmed that the addition of home-based, parent-led Floortime sessions at an average of 15.2 h per week across 3 months led to clinically significant gains in social emotional functioning. Additionally, since 2011 one study has also demonstrated the effectiveness of training the caregiver in the implementation of DIR/Floortime strategies. This study examined the efficacy of implementation of the home-based Play and Language for Autistic Youngsters (PLAY) Project and demonstrated that there were significant gains in functional development reflected in the diagnostic categories on the Autism Diagnostic Observation Schedule (ADOS) as well as large treatment effects for parent-child interactional behaviors (Solomon et al. 2014). One additional study identified significant gains in discrete social skills, including: turn taking; reciprocal, or two-way,

communication; understanding cause and effect; and emotional thinking following intensive early intervention using the Floortime approach (Lal and Chhabria 2013).

Outcome Measurement

The DIR[®] model uses two outcome measures, the *Functional Emotional Assessment Scale* (FEAS) and the *Greenspan Social Emotional Growth Chart* (SEGC), to assess baseline levels at the initial evaluation, measure progress at follow-up sessions, and establish outcomes of the therapy (NRC 2001). The initial evaluation of a child should include developing a profile of that child's individual functioning when compared to the six stages of emotional development. Visual-spatial and motor planning should also be assessed to determine the child's ability to process information (Greenspan and Wieder 2006).

The FEAS is a criterion-referenced tool for measuring functional emotional capacities in children ages 7 months through 4 years of age. More specifically, the FEAS systematically assesses the child's ability to negotiate play activities in relation to people or objects, to self-regulate mood and attention, to form attachments with the caregiver, to use emotional and communicative reciprocity, and to represent emotions, ideas, and emotional thinking within the context of play interactions from videotaped samples. A caregivers' capacity to support development in the aforementioned areas of emotional development is also assessed (Greenspan and DeGangi 2001). In addition to the two measures noted above, continued assessment of the child's functioning in each of the stages of FEDCs informs the direction of treatment as well as progress across developmental areas (Greenspan and Wieder 2006).

Qualifications of Treatment Providers

The DIR[®]/Floortime model is considered to be a comprehensive and individualized approach

that can be developed and adjusted according to the child's individual differences, family dynamics, and level of development. The development of the program requires participation of several disciplines: speech-language pathologists, occupational or physical therapists, early educators and/or special educators, and a therapist trained in intensive Floortime work (Greenspan 1992a as cited in Greenspan and Wieder 1997).

Although multiple care providers with expertise in several disciplines are required for the development and implementation of a comprehensive DIR[®] program, parents play an essential role as providers of spontaneous Floortime sessions throughout the day (Greenspan and Wieder 2006). Additionally, while several specialists with relevant training in his/her area of expertise may cooperate in implementing the Floortime approach, the application of a comprehensive DIR[®] approach should be managed and overseen by a specialist from a relevant discipline, who has training in applying DIR[®] theory. The Interdisciplinary Council on Developmental and Learning Disorders (ICDL) provides a few training opportunities for licensed professionals from several disciplines, such as speech-language pathology, occupational and physical therapy, as well as social work, to name a few. Among the training opportunities offered, there is a DIR[®] introductory level course, a DIR[®] beginning course, as well as an intensive DIR[®] certification program. Information regarding further information and training opportunities on the DIR[®] model can be found at www.icdl.com.

See Also

- [Affective Development](#)
- [Emotion Regulation](#)
- [Relationship Development Intervention \(RDI\) Model](#)
- [Sensory Processing](#)

References and Reading

- Cole, M., & Scribner, S. (1978). Introduction. In M. Cole, V. John-Steiner, S. Scribner, & E. Souberman (Eds.), *Mind in society: The development of higher psychological processes* (pp. 1–14). Cambridge, MA: Harvard University Press.
- Davis, A., Isaacson, L., & Harwell, M. (2014). *Floortime strategies to promote development in children and teens: A user's guide to the DIR[®] model*. Baltimore: Paul H Brookes Publishing.
- Greenspan, S. I., & DeGangi, G. (2001). Research on the FEAS: Test development, reliability, and validity studies. In S. Greenspan, G. DeGangi, & S. Wieder (Eds.), *The functional emotional assessment scale (FEAS) for infancy and early childhood: Clinical and research applications* (pp. 167–247). Bethesda: Interdisciplinary council on developmental and learning disorders (ICDL). <http://www.icdl.com/dirFloortime/research/FunctionalEmotionalAssessmentScale.shtml>
- Greenspan, S. I., & Shanker, S. G. (2004). Origin of symbols. In S. I. Greenspan & S. G. Shanker (Eds.), *The first ideas: How symbols, language, and intelligence evolved from our primate ancestors to modern humans* (pp. 1–40). Cambridge, MA: De Capo Press.
- Greenspan, S. I., & Wieder, S. (1997). Developmental patterns and outcomes in infants and children with disorders in relating and communicating: A chart review of 200 cases of children with autism spectrum diagnoses. *Journal of Developmental and Learning Disorders, 1*, 87–141.
- Greenspan, S. I., & Wieder, S. (1999). A functional developmental approach to autism spectrum disorders. *Journal of the Association for Persons with Severe Handicaps, 24*(3), 147–161.
- Greenspan, S. I., & Wieder, S. (2006). *Engaging autism: Using the floor time approach to help children relate, communicate and think*. Cambridge, MA: First Da Capo Press.
- Huitt, W., & Hummel, J. (2003). Piaget's theory of cognitive development. *Educational Psychology Interactive*. Valdosta: Valdosta State University. Retrieved December 4, 2010, from <http://www.edpsycinteractive.org/topics/cogsys/piaget.html>
- ICDL (2000). ICDL: DIR[®]/Floortime and autism spectrum disorders: Overview and summary of scientific and public support. *The Interdisciplinary Council on Developmental and Learning Disorders*, Bethesda. Retrieved November 3, 2010, from www.icdl.com
- Ingersoll, B., Dvortcsak, A., Whalen, C., & Sikora, D. (2005). The effects of a developmental, social-pragmatic language intervention on rate of expressive language production in young children with autistic spectrum disorders. *Focus on Autism and Other Developmental Disorders, 20*(4), 213–222.
- Lal, R., & Chhabria, R. (2013). Early intervention of autism: A case for floor time approach. *Recent Advances in Autism Spectrum Disorders, 1*. <https://doi.org/10.5772/54378>.

- Landa, R. J., Holman, K. C., & Garrett-Mayer, E. (2007). Social and communication development in toddlers with early and later diagnosis of autism spectrum disorders. *Archives of General Psychiatry*, 64(7), 853–864.
- Mahoney, G., & Perales, F. (2005). Relationship-focused early intervention with children with pervasive developmental disorders and other disabilities: A comparative study. *Developmental and Behavioral Pediatrics*, 26(2), 77–85.
- Mundy, P., Sigman, M., & Kasari, C. (1990). A longitudinal study of joint attention and language development in autistic children. *Journal of Autism and Developmental Disorders*, 20, 115–128.
- National Autism Center (2009). *National Standards Project, Phase 1: Addressing the need for evidence-based practice guidelines for ASD*. www.nationalautismcenter.org
- National Autism Center (2015). *National Standards Project, Phase 2: Addressing the need for evidence-based practice guidelines for ASD*. www.nationalautismcenter.org
- National Research Council. (2001). Comprehensive programs. In C. Lord & J. P. McGee (Eds.), *Educating children with autism* (pp. 140–172). Committee on educational interventions for children with autism. Division of behavioral and social sciences and education.). Washington, DC: National Academy Press.
- Pajareya, K., & Nopmaneejumrulers, K. (2011). A pilot randomized control trial of DIR-Floortime™ parent training intervention for pre-school children with autism spectrum disorders. *Autism*, 15(5), 1–15.
- Prelock, P. (2006). Interventions to support the social-emotional needs of children with ASD. In P. A. Prelock (Ed.), *Autism spectrum disorders: Issues in assessment and intervention* (pp. 479–539). Austin: Pro-Ed.
- Solomon, R., Necheles, J., Ferch, C., & Bruckman, D. (2007). Pilot study of a parent training program for young children with autism: The PLAY project home consultation program. *Autism*, 11(3), 205–224.
- Solomon, R., Van Egeren, L. A., Mahoney, G., Quon Huber, M. S., & Zimmerman, P. (2014). PLAY project home consultation intervention program for young children with autism spectrum disorders: A randomized controlled trial. *The Journal of Developmental and Behavioral Pediatrics*, 35(8), 475–485.
- Wieder, S., & Greenspan, S. I. (2005). “Can children with autism master the core deficits and become empathetic, creative, and reflective?” A ten to fifteen year follow-up of a subgroup of children with autism spectrum disorders (ASD) who received a comprehensive developmental, individual-difference, relationship based (DIR®) approach. *The Journal of Developmental and Learning Disorders*, 9, 39–61.

Fluency and Fluency Disorders

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Clinical Issues in Speech-Language Pathology,
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Synonyms

Cluttering; Disfluency; Dysfluency; Nonfluency; Stammering; Stuttering

Definition

Fluency is the aspect of speech production that refers to continuity, smoothness, rate, and effort (American Speech-Language-Hearing Association [ASHA] [n.d.](#)). *Stuttering* is the most common fluency disorder. Stuttering is an interruption in the forward flow of speech and is characterized by repetitions (sounds, syllables, words, phrases), sound prolongations, blocks, interjections, and revisions, which may affect the rate and rhythm of speech. These disfluencies may be accompanied by physical tension, negative reactions, secondary behaviors, and avoidance of sounds, words, or speaking situations (ASHA [1993](#); Yaruss [2007](#)).

Incidence and Prevalence

The lifetime incidence of stuttering is approximately 5% or higher (Mansson [2000](#); Yairi and Ambrose [2013](#)). Among children, the incidence reaches a rate of 8.5% by 3 years and 11% by 4 years (Reilly et al. [2013](#)). Stuttering is more common among males than females. Among elementary school-age children, it is estimated that boys are three to four times more likely to stutter than girls (Craig et al. [2002](#)). The estimated prevalence rate for stuttering across ages is 0.72% (Craig et al. [2002](#)).

Fludac

► Prozac™ (Fluoxetine)

Types of Disfluencies

All speakers have disfluencies, which may include hesitations for language formulation including silent pauses as well as insertion of word fillers (e.g., “The book is *like* new”) and nonword fillers (sometimes called *interjections*, e.g., “My friend is *uh* driving”); other examples also may include whole-word repetitions (e.g., “Go-go get that bottle”) and phrase repetitions or revisions (e.g., “*Don’t tell me-* don’t tell me again”). These may be considered “more typical,” nonstuttering disfluencies when there are a small number.

Less typical, more stuttering-like disfluencies include *part-word* or *sound/syllable repetitions* (e.g., “Give me the *p-p-p* pencil”), *prolongations* (e.g., “*Sssssss*stay here with me”), and *blocks* (i.e., silent fixations or inability to initiate sounds). In addition, stuttering-like disfluencies are usually accompanied by decidedly greater than average duration, effort, tension, or struggle than are typical disfluencies.

Developmental Progression

Stuttering most often begins during childhood. Some young children go through a period of excessive disfluency. However, they go on to display typical fluency skills as their language abilities develop. A large majority of early stuttering remits without clinical intervention, although recovery estimates vary greatly from 6.3% (Reilly et al. 2013) to 89% (Yairi and Ambrose 1999).

Impact of Stuttering

Stuttering can greatly interfere with school, work, or social interactions. Children and adults who stutter may report fear or anxiety about speaking and frustration or embarrassment with the time and effort required to speak. Despite popular beliefs, emotional problems or emotional trauma do not cause stuttering. However, dealing with stuttering can result in significant emotional reactions and avoidance behavior. Stuttering is often more

severe when there is increased pressure to communicate (e.g., giving a presentation, interviewing for a job).

Causes

The cause of stuttering is presently unknown but may reflect subtle underlying differences in brain structure and function, particularly when an individual is processing and producing language. Stuttering may have a genetic component and the risk of stuttering is elevated in families having other members with persistent stuttering. Stuttering can also occur following neurological trauma (e.g., brain injury) and following psychological trauma (e.g., posttraumatic stress disorder).

Cooccurrence

Stuttering can cooccur with other disorders. For example, although there is little systematic evidence describing disfluency in autism spectrum disorder (ASD), increasing numbers of case reports indicate atypical, stuttering-like behaviors that are additionally distinguished by unusual features, such as repetition of final segments of words (Paul et al. 2005; Shriberg et al. 2001; Sisskin and Wasilus 2014). Children who stutter may show expressive language problems because of a tendency to avoid speaking. Less talking and reduced linguistic complexity may result (Silverman and Bernstein Ratner 2002).

Terminology

The term disfluency (sometimes spelled dysfluency) often is used synonymously with stuttering. However, the term disfluency refers to typical and atypical breaks in the forward flow of speech, while stuttering refers to a fluency disorder, characterized by a high number of less typical disfluencies and usually a greater effort to speak. *Stammering* is synonymous with *stuttering* and is the common term for the disorder in Great Britain. In North

America, the term *stammering* is rarely used. *Secondary behaviors* are also known as *accessory behaviors*, *secondary mannerisms*, *secondaries*, *concomitant behaviors*, or *extraneous behaviors*. For example, a person may tap his foot or nod her head while talking in a misplaced effort to speak fluently. Secondary behaviors usually start coincidentally and then become incorporated when the person notices an improvement in fluency. If a behavior appears to help, it may persist, even though it loses its effectiveness.

Cluttering

Cluttering is another fluency disorder. Cluttering is characterized by a rapid and/or irregular speech rate, and excessive disfluencies, which are usually of the more typical type (revisions and overuse of filler words, such as “um”). The speech of a person who clutters often contains pauses in unusual places and unusual prosody (St. Louis and Schulte 2011). Other symptoms may include language or phonological errors. Cluttering has been documented in children with autism (Scaler Scott 2011).

Speech-Language Pathologists

Speech-language pathologists (SLPs) are the professionals who perform fluency assessments and intervention individually or in groups. SLPs also serve as members of collaborative teams that may include the individual, family/caregivers, educators, and other relevant persons.

See Also

- [Cluttering](#)
- [Disfluency](#)
- [Dysfluency](#)
- [Nonfluency](#)
- [Speech-Language Pathologist \(SLP\)](#)
- [Stammering](#)
- [Stuttering](#)

References and Reading

- American Speech-Language-Hearing Association. (1993). *Definitions of communication disorders and variations*. Retrieved from www.asha.org/policy
- American Speech-Language-Hearing Association. (n.d.). *Fluency disorders in childhood (Practice portal)*. Available from www.asha.org/Practice-Portal/Clinical-Topics/Childhood-Fluency-Disorders
- Craig, A., Hancock, K., Tran, Y., Craig, M., & Peters, K. (2002). Epidemiology of stuttering in the community across the entire life span. *Journal of Speech, Language, and Hearing Research*, 45, 1097–1105.
- Mansson, H. (2000). Childhood stuttering: Incidence and development. *Journal of Fluency Disorders*, 25, 47–57.
- Paul, R., Shriberg, L. D., McSweeney, J., Cicchetti, D., Klin, A., & Volkmar, F. (2005). Brief report: Relations between prosody performance and communication and socialization ratings in high functioning speakers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 35, 861–869.
- Reilly, S., Onslow, M., Packman, A., Cini, E., Conway, L., Obioha, C., . . . Wake, M. (2013). Natural history of stuttering to 4 years of age: A prospective community-based study. *Pediatrics*, 132, 460–467.
- Scaler Scott, K. (2011). Cluttering and autism spectrum disorders. In D. Ward & K. Scaler Scott (Eds.), *Cluttering: Research, intervention and education* (pp. 115–134). East Sussex: Psychology Press.
- Shriberg, L. D., Paul, R., McSweeney, J. L., Klin, A., Cohen, D. J., & Volkmar, F. R. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning autism and Asperger syndrome. *Journal of Speech, Language, and Hearing Research*, 44, 1097–1115.
- Silverman, S., & Bernstein Ratner, N. (2002). Measuring lexical diversity in children who stutter: Application of *vocd*. *Journal of Fluency Disorders*, 27(4), 289–304.
- Sisskin, V., & Wasilus, S. (2014). Lost in the literature, but not the caseload: Working with atypical disfluency from theory to practice. *Seminars in Speech and Language*, 35(2), 144–152.
- St. Louis, K. O., & Schulte, K. (2011). Defining cluttering: The lowest common denominator. In D. Ward & K. Scaler Scott (Eds.), *Cluttering: Research, intervention and education* (pp. 233–253). East Sussex: Psychology Press.
- Yairi, E., & Ambrose, N. G. (1999). Early childhood stuttering I: Persistency and recovery rates. *Journal of Speech, Language, and Hearing Research*, 42(5), 1097–1112.
- Yairi, E., & Ambrose, N. (2013). Epidemiology of stuttering: 21st century advances. *Journal of Fluency Disorders*, 38(2), 66–87.
- Yaruss, J. S. (2007). Application of the ICF in fluency disorders. *Seminars in Speech and Language*, 28(4), 312–322.

Fluent Aphasia

- [Wernicke's Aphasia](#)

Fluent Speech

- [Verbal Communication](#)

Fluid Intelligence

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Synonyms

[Fluid reasoning](#)

Definition

Fluid intelligence (abbreviated Gf) is the ability to reason quickly, think abstractly, and problem-solve, independent of acquired knowledge. Cattell (1971) proposed two factors underlying general intelligence: fluid and crystallized intelligence. Tests of fluid intelligence tap current reasoning ability and are considered to be more “culture-fair,” being less affected by differences in learning experience or test familiarity. Raven's Progressive Matrices, and other tasks with novel stimuli, are thought to tap fluid intelligence.

People with ASD often do well on certain tests thought to measure fluid intelligence, such as Raven's Matrices (Hayashi et al. 2008) and Block Design (Shah and Frith 1993). Interpretation of these peaks in an uneven intelligence test profile has varied: many fluid intelligence tests

tap visuospatial problem solving, which may be superior to verbal reasoning in many people with autism. Alternatively, Dawson et al. (2007) have suggested that good Raven's Matrices scores reveal the true level of intelligence in autism, underestimated by standard IQ tests. Scheuffgen et al. (2000) suggested that people with autism may have good potential processing efficiency (as seen in fluid intelligence tests), not reflected in standard IQ test performance because of failure to acquire skills and knowledge through socially mediated learning. Fluid intelligence is thought to include several processes, only some of which may be superior (relative to own profile or others') in autism; visualization and pattern recognition, for example, may be areas of skill, while ideational fluency or generativity (see ► [“Behavior Observation Scale”](#)) may not.

See Also

- [Behavior Observation Scale](#)
- [Crystallized Intelligence](#)

References and Reading

- Cattell, R. B. (1971). *Abilities: Their structure, growth, and action*. New York: Houghton Mifflin.
- Dawson, M., Soulières, I., Gernsbacher, M. A., & Mottron, L. (2007). The level and nature of autistic intelligence. *Psychological Science*, 18, 657–662.
- Hayashi, M., Kato, M., Igarashi, K., & Kashima, H. (2008). Superior fluid intelligence in children with Asperger's disorder. *Brain and Cognition*, 66, 306–310.
- Scheuffgen, K., Happé, F., Anderson, M., & Frith, U. (2000). High “Intelligence”, low “IQ”? Speed of processing and measured IQ in children with autism. *Development and Psychopathology*, 12, 83–90.
- Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry*, 34, 1351–1364.

Fluid Reasoning

- [Fluid Intelligence](#)

Fluoxetine

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Synonyms

Brand names: [Prozac](#); [Prozac Weekly \(once-weekly dosing\)](#); [Rapiflux](#); [RECONCILE](#); [Sarafem](#); [Selfemra](#)

Indications

Fluoxetine is FDA-approved to treat major depressive disorder (MDD) in both adults and in children aged 8–18 years old, obsessive-compulsive disorder (OCD) in both adults and children aged 7–17 years old, and bulimia nervosa in adult patients. In these disorders, fluoxetine can be used both acutely and as maintenance therapy. It is approved for adults only in the acute treatment of panic disorder with and without agoraphobia. In combination with olanzapine, marketed as Symbyax, it is approved for the acute treatment of depressive episodes associated with bipolar I disorder and treatment-resistant depression in adults only (Lilly 2009).

Fluoxetine is not approved for the treatment of autism spectrum disorders (ASDs), which include the DSM-IV-TR diagnoses of autistic disorder, Asperger's disorder, and pervasive developmental disorder, not otherwise specified (PDD-NOS). The decision to use fluoxetine in the treatment of established clinical indications that may co-occur with ASDs, as mentioned above, or for commonly observed symptoms of ASDs, such as hyperactivity, inattention, irritability, aggression, repetitive behaviors, and social impairment, can be made on an individual basis by the treating practitioner.

This will be discussed in more detail in the “[Clinical Uses](#)” section.

Mechanisms of Action

Fluoxetine is a selective serotonin reuptake inhibitor (SSRI). Normally, serotonin is released from the presynaptic axon terminal to travel across the synaptic cleft and attach to the postsynaptic axon terminal. Any excess serotonin remaining in the cleft is picked up by the serotonin reuptake transporter in the presynaptic axon to be degraded and recycled. It is hypothesized that a depletion of serotonin available to act on the postsynaptic axon results in symptoms experienced in mood and anxiety disorders, like sadness, nervousness, guilt, loneliness, apathy, and avolition. Fluoxetine attaches itself to the serotonin reuptake transporter and prevents whatever serotonin remains in the cleft from being degraded. The excess serotonin is free to attach to postsynaptic receptors and exert its intended effects (Stahl 2008).

Although fluoxetine begins acting immediately to inhibit serotonin reuptake, its antidepressant and anxiolytic effects are often not experienced for up to 6–8 weeks. This is thought to be due to gradual changes fluoxetine makes on serotonin receptor sensitivity. It is believed that the decreased amounts of serotonin found in mood and anxiety disorders cause the postsynaptic axon to upregulate the number of postsynaptic axonal receptors expressed. In other words, a decreased amount of serotonin in the synaptic cleft results in an increased amount of receptors at the ready. When fluoxetine enters the picture, the level of serotonin in the cleft rises. Postsynaptic receptors recognize the serotonin increase and send messages to the axon nucleus about the changes in the synaptic cleft. The nucleus begins downregulating the amount of serotonin receptors ready to receive serotonin. These changes are gradual and are believed to take 6–8 weeks before fully completed, reflecting the amount of time before many patients begin feeling relief from their symptoms (Stahl 2008).

Fluoxetine's antagonism of the postsynaptic 5-HT_{2C} receptor results in some of its unique therapeutic effects. When serotonin attaches to

the 5-HT_{2C} receptor, it blocks the release of neurotransmitters norepinephrine and dopamine in the brain. When fluoxetine blocks the postsynaptic 5-HT_{2C} receptor, norepinephrine and dopamine are instead released and exert their effect in the prefrontal cortex. The effect is activating, often leading patients to feel more energized, less fatigued, and to have improved concentration and attention. Fluoxetine is therefore considered for patients whose constellation of symptoms includes fatigue, apathy, and avolition as opposed to nervousness, insomnia, and agitation. Other therapeutic effects from postsynaptic 5-HT_{2C} antagonism include anorexia and anti-bulimia, particularly at higher doses of fluoxetine, and relief from bipolar depression when combined with olanzapine, a second-generation antipsychotic. Of note, neither olanzapine nor fluoxetine alone is approved for the treatment of bipolar depression. However, they both antagonize the 5-HT_{2C} receptor. This combined antagonism likely boosts norepinephrine and dopamine release in the cortex and may be what drives the antidepressant effect seen in the treatment of bipolar depression. The combination of fluoxetine and olanzapine is marketed as Symbyax with a fixed-dose ratio of fluoxetine to olanzapine, although both can be prescribed separately.

Fluoxetine also weakly attaches to the norepinephrine transporter and prevents norepinephrine's reuptake from the synaptic cleft. This may contribute to its clinical effects at higher doses.

Fluoxetine inhibits the cytochrome P450 CYP 2D6 and 3A4 enzymes via the parent compound and the active metabolite. The half-life of the parent compound is 2–3 days and that of the active metabolite is 2 weeks (Stahl 2008).

Specific Compounds and Properties

N-Methyl-γ-[4-(trifluoromethyl)phenoxy]benzenepropanamine hydrochloride.

Clinical Use (Including Side Effects)

FDA-Approved Clinical Uses

Fluoxetine is FDA-approved for the treatment of some mood and anxiety disorders. Mood

disorders include major depressive disorder (MDD), premenstrual dysphoric disorder (PMDD), and bipolar depression. Anxiety disorders include OCD and panic disorder, with or without agoraphobia (Lilly 2009).

In the treatment of mood and anxiety disorders, fluoxetine is usually started between 10 and 20 mg once daily and gradually increased to 20–80 mg daily in adults (Lilly 2009). The goal of treatment is complete remission of symptoms and prevention of future relapses, so fluoxetine is taken both throughout and in between clinical relapses (Stahl 2009). Fluoxetine does not cure mood or anxiety disorders, and symptoms can reoccur after the medicine has been stopped.

In adults with MDD, fluoxetine is used to treat acute depressive episodes and as maintenance therapy between episodes (Lilly 2009). After the first episode of depression, fluoxetine should be taken for 1 year (Stahl 2009). After the second or any subsequent episodes of depression, treatment with fluoxetine may be indefinite to avoid relapse of symptoms. Fluoxetine has also been helpful in the short-term treatment of acute depressive episodes associated with bipolar I disorder when used in combination with olanzapine (Lilly 2009). Combination therapy with set doses of fluoxetine and olanzapine is manufactured as Symbyax by Eli Lilly, although these two medications can be prescribed separate from each other. Separate dosing may allow for more flexibility on the part of the prescribing clinician. Fluoxetine should not be used as monotherapy for treatment of depression associated with bipolar I disorder. The combination of fluoxetine and olanzapine is also indicated for treatment-resistant depression, referring to depression that does not respond to two separate trials of different antidepressants of adequate doses and duration within the current episode. A once-weekly dosing of fluoxetine is marketed as Prozac Weekly for the treatment of MDD.

In adults with OCD, fluoxetine is given to treat acute episodes and as maintenance therapy between episodes. Fluoxetine is also used in the acute treatment of adults with panic disorder, with or without agoraphobia. Fluoxetine can be helpful in the treatment of bulimia nervosa, an eating

disorder. Moderate to severe bulimia nervosa, which is defined as three bulimic episodes per week within 6 months, has been shown to benefit from treatment with fluoxetine. For bulimia, fluoxetine is dosed between 60 and 80 mg daily and patients may require indefinite treatment (Stahl 2009).

Regarding children and adolescents, fluoxetine is FDA-approved for the treatment of MDD in pediatric populations aged 8–18 years and OCD in those aged 7–17 years (Lilly 2009). In MDD, fluoxetine is given for acute depressive episodes and as maintenance therapy between episodes. The starting dose is typically between 10 mg and 20 mg/day. Due to higher plasma levels in lower weight children, the target dose may not exceed the initial dosage or much beyond. In OCD, fluoxetine is given for acute episodes and as maintenance therapy between episodes. The starting dose is typically 10 mg/day, increased to as high as 60 mg/day in adolescents. Lower weight children may only tolerate a lower target dose, such as 20 mg/day.

Clinical Uses of Fluoxetine in Autism Spectrum Disorders

Regarding pediatric populations, there is limited evidence to suggest the effectiveness of fluoxetine in children and adolescents with autism. Interestingly, antidepressants have been the most commonly prescribed psychotropic medications in treating symptoms associated with ASDs (Aman et al. 2005). Given the frequent use of SSRIs in autism, it is imperative that prescribing clinicians balance the benefit of such medications in lieu of their side effects. In 2004, the FDA released safety warnings about the increased risk of suicide-related behaviors in children and adolescents taking SSRIs, which subsequently curtailed the prescribing of SSRIs to this population (Nemeroff et al. 2007). The decision to use fluoxetine in the treatment of established clinical indications that may co-occur with autism should therefore be made on an individual basis by the treating practitioner after careful consideration of the available data.

In the only published double-blind, placebo-controlled trial using fluoxetine to treat symptoms

associated with ASDs in children and adolescents, fluoxetine was found to be significantly better than placebo in reducing repetitive behaviors (Hollander et al. 2005). There was no improvement on measures of speech or social interaction, and side effects were not significantly different between fluoxetine and placebo. This study used low doses of liquid fluoxetine in a population of 45 children between the ages of 5 and 16 years (mean age, 8.2 years) who met criteria for autism, Asperger's disorder, or PDD-NOS. The starting dose began at 2.5 mg/day and was increased weekly to the target dose of 0.8 mg/kg/day, although the highest dose given was 0.4 mg/kg/day. Unfortunately, the remaining research in this area has been open label or retrospective, limiting further conclusions about the effectiveness of fluoxetine in treating children and adolescents with ASDs. There is no data regarding the effect of fluoxetine on anxiety, depression, hyperactivity, inattention, irritability, or aggression in children with ASDs.

In adult populations, there is minimal data supporting the use of fluoxetine in the treatment of ASDs as evidenced by only one small, placebo-controlled pilot study. This trial focused on six adult patients, five of whom had autism and one with Asperger's disorder (Buchsbaum et al. 2001). The patients showed significant improvement of anxiety and obsessive-compulsive symptoms while on fluoxetine. There was no significant improvement in depression. The starting dose was 10 mg daily, which was gradually titrated up to 40 mg daily in most of the patients. One should be cautious in applying these findings to the greater population of adults with autism as the small number of patients included in the study limits its interpretation. Unfortunately, there is no data regarding the effect of fluoxetine on hyperactivity, inattention, irritability, aggression, or social impairment in adults with ASDs.

It should be noted that the repetitive thoughts and behaviors observed in individuals with ASDs are qualitatively different from the symptoms of OCD (McDougle et al. 1995). For example, individuals with ASDs are more likely to experience compulsions of ordering, hoarding, telling or

asking, and touching, tapping, or rubbing. They are less likely to experience obsessions of contamination, sexual, religious, symmetry, or somatic content, unlike individuals with OCD. In the study of fluoxetine cited above, participants were rated on symptoms of OCD but no distinction was made regarding which repetitive behaviors improved.

Side Effects

Fluoxetine's side effect profile results from the stimulation of serotonin receptor subtypes as well as lesser actions on other neurotransmitters and enzymes. Stimulation of serotonin receptor subtypes (5-HT_{2A}, 5-HT_{2C}, 5-HT₃, and 5-HT₄) in various parts of the brain likely causes many of SSRIs' observed side effects (Stahl 2008). A small amount of increased synaptic serotonin shortly after initiating therapy is often enough to mediate side effects, even if the clinical benefit is not yet apparent to the patient. Therefore, it is possible that side effects may be experienced earlier than symptom relief when first starting treatment with fluoxetine (Stahl 2008). Side effects experienced may also be dose dependent (i.e., they increase as the dose increases) or time dependent (i.e., they start right after taking the medication but diminish with time) (Stahl 2009).

Patients who are treated for MDD are at increased risk for experiencing suicidal thinking and behavior. Antidepressants as a class have been shown to increase the risk of suicidal thinking and behavior in children, adolescents, and young adults (ages 18–24) with MDD and other psychiatric disorders. Such risk should be carefully considered when prescribing fluoxetine in the pediatric and young adult population. According to Stahl (2009), gastrointestinal side effects are common and can include decreased appetite, nausea, diarrhea, constipation, or dry mouth. Sexual dysfunction is a common side effect in both men and women and is due to the effect of increased serotonin in both the brain and the region of the spinal cord regulating sexual response. In men, this includes delayed ejaculation, erectile dysfunction, and decreased sexual desire. In women, this includes decreased sexual

desire and anorgasmia. Central nervous system side effects include insomnia or sedation, agitation, tremors, headache, and dizziness or lightheadedness. Patients may experience vasodilatation, dry mouth, diaphoresis, or abnormal vision. Increased serotonin can lead to diminished dopamine release, which may lead to emotional flattening, apathy, and cognitive slowing in some patients. Anorexia and weight loss have been reported when using fluoxetine, so it should be used with caution in underweight patients or those who are prone to eating disorders (Lilly 2009).

Of note, SSRIs have been found to yield side effects in people with ASDs, particularly agitation, hyperactivity, aggression, and insomnia (Posey et al. 2006). Extrapyramidal symptoms (Sokolski et al. 2004) and hypomania (Damore et al. 1998) have also been observed in case reports. These results are limited by a lack of placebo-controlled studies, although there is some evidence to suggest that children with ASDs may be more likely to develop such side effects relative to adults with ASDs (Posey et al. 2006).

See Also

- ▶ Antidepressant Medications
- ▶ Depressive Disorder
- ▶ Dopamine
- ▶ Norepinephrine
- ▶ Obsessive-Compulsive Disorder (OCD)
- ▶ Repetitive Behavior
- ▶ Serotonin
- ▶ Serotonin Reuptake Inhibitors (SRIs)
- ▶ Stereotypic Behavior

References and Reading

- Aman, M. G., Lam, K. S., et al. (2005). Medication patterns in patients with autism: Temporal, regional, and demographic influences. *Journal of Child and Adolescent Psychopharmacology*, 15(1), 116–126.
- Buchsbaum, M. S., Hollander, E., et al. (2001). Effect of fluoxetine on regional cerebral metabolism in

- autistic spectrum disorders: A pilot study. *The International Journal of Neuropsychopharmacology*, 4(2), 119–125.
- Damore, J., Stine, J., et al. (1998). Medication-induced hypomania in Asperger's disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 37(3), 248–249.
- Hollander, E., Phillips, A., et al. (2005). A placebo controlled crossover trial of liquid fluoxetine on repetitive behaviors in childhood and adolescent autism. *Neuropsychopharmacology*, 30(3), 582–589.
- Lilly, E. & Company prescribing information. (2009). Retrieved from <http://pi.lilly.com/prozac.pdf>.
- McDougle, C. J., Fleischmann, R. L., et al. (1995). Risperidone addition in fluvoxamine-refractory obsessive-compulsive disorder: Three cases. *The Journal of Clinical Psychiatry*, 56(11), 526–528.
- Nemeroff, C. B., Kalali, A., et al. (2007). Impact of publicity concerning pediatric suicidality data on physician practice patterns in the United States. *Archives of General Psychiatry*, 64(4), 466–472.
- Posey, D. J., Erickson, C. A., et al. (2006). The use of selective serotonin reuptake inhibitors in autism and related disorders. *Journal of Child and Adolescent Psychopharmacology*, 16(1–2), 181–186.
- Romano, S. J., Halmi, K. A., et al. (2002). A placebo-controlled study of fluoxetine in continued treatment of bulimia nervosa after successful acute fluoxetine treatment. *The American Journal of Psychiatry*, 159, 96–102.
- Sokolski, K. N., Chicz-Demet, A., et al. (2004). Selective serotonin reuptake inhibitor-related extrapyramidal symptoms in autistic children: A case series. *Journal of Child and Adolescent Psychopharmacology*, 14(1), 143–147.
- Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd ed., pp. 522–535). New York: Cambridge University Press.
- Stahl, S. M. (2009). *Stahl's essential psychopharmacology: The prescriber's guide* (3rd ed., pp. 193–198). New Delhi: Cambridge University Press.

Fluoxin

- **Prozac™ (Fluoxetine)**

Flutine

- **Prozac™ (Fluoxetine)**

Fluvoxamine

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Synonyms

Fluvoxamine maleate; Luvox; Luvox CR

Indications

Fluvoxamine is FDA-approved for the treatment of obsessive-compulsive disorder (OCD).

Mechanisms of Action

Fluvoxamine is a selective serotonin (5-HT) reuptake inhibitor (SSRI). Its efficacy in the treatment of OCD is thought to result from inhibition of the serotonin reuptake process and increased serotonergic neurotransmission (Stahl 2008).

Fluvoxamine primarily inhibits the oxidative drug-metabolizing enzyme CYP1A2 and also significantly inhibits CYP3A4, CYP2C9, and CYP2C19. It is a weak inhibitor of CYP2D6 (Irons 2005; Kuzma and Black 2008). Additionally, it is a sigma-1 receptor agonist (Ordacgi et al. 2009). Sigma-1 receptors regulate the release of glutamate, dopamine, serotonin, norepinephrine, and acetylcholine. Fluvoxamine's high affinity for these receptors may account for some of its effects on depression and anxiety.

Specific Compounds and Properties

Fluvoxamine maleate has the International Union of Pure and Applied Chemistry (IUPAC) name (E)-5-methoxy-1-[4-(trifluoromethyl)phenyl]pentan-1-one *O*-2-aminoethyl oxime maleate and the empirical formula $C_{15}H_{21}O_2N_2F_3 \cdot C_4H_4O_4$ (Fig. 1).

Fluvoxamine is metabolized by the liver into at least 11 products, all of which are pharmacologically inactive. It is excreted primarily as metabolites, with less than 4% of the original compound remaining. The precise CYP isoenzymes involved in its metabolism are not known. There is evidence that fluvoxamine has nonlinear steady-state pharmacokinetics so that plasma concentrations are disproportionately higher when the dosage is increased. It has a mean plasma half-life of 12–22 h, which is increased by 30–50% at steady state. It has been demonstrated to be eliminated from the brain at a slower rate than from the plasma (2.4:1 ratio; Ordacgi et al. 2009).

In vitro studies have found it to be a stronger inhibitor of serotonin reuptake than fluoxetine, but weaker than paroxetine, sertraline, citalopram, and escitalopram. It is also a weak inhibitor of norepinephrine and dopamine reuptake when compared to other serotonin reuptake inhibitors (Ordacgi et al. 2009).

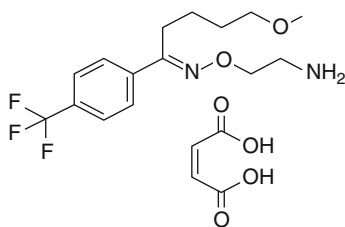
Clinical Use (Including Side Effects)

Luvox is the brand name of fluvoxamine maleate in the United States. It is available in both

immediate-release (IR) and extended-release (controlled-release; CR) tablets and is FDA-approved for the treatment of obsessions and compulsions in individuals with OCD. The IR form is approved for use in patients 8 years and older, while the lowest dosage of the CR form may not be appropriate for pediatric patients (U.S. Food and Drug Administration 2011a, b). Its efficacy in the treatment of OCD has been demonstrated in numerous randomized, double-blind, controlled studies. Fluvoxamine has repeatedly been shown to be more efficacious than placebo when measured by the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), National Institute of Mental Health-Obsessive Compulsive (NIMH-OC) Scale, and Clinical Global Impression (CGI) scales (Ordacgi et al. 2009). Studies have also compared fluvoxamine to the tricyclic antidepressants clomipramine and desipramine. Fluvoxamine was found to be equally efficacious as clomipramine and more efficacious than desipramine in the treatment of OCD (Irons 2005). It has additionally been shown to be efficacious in the treatment of OCD in children and adolescents aged 8–17 years (Irons).

Luvox CR was initially approved for the treatment of social anxiety disorder, but this indication was removed from its label in 2011. However, a number of double-blind, placebo-controlled studies have shown promise for the treatment of social anxiety disorder with fluvoxamine. The results of several studies, two involving over 250 patients each, have demonstrated significantly greater improvements with fluvoxamine than with placebo, using measures such as the Liebowitz Social Anxiety Scale, CGI scales, and the Hamilton Anxiety Scale. Furthermore, fluvoxamine has been demonstrated to be significantly more effective than placebo in a study of 128 children and adolescents aged 6–17 years with social anxiety disorder, separation anxiety disorder, or generalized anxiety disorder (GAD) (Irons 2005).

Fluvoxamine may also be an effective treatment for some of the behaviors associated with autism. In a randomized, double-blind study with 30 adults, 53% of the patients treated with



Fluvoxamine, Fig. 1 Chemical structure of fluvoxamine maleate

fluvoxamine were classified as responders. There was a significant reduction in repetitive thoughts and behaviors, maladaptive behavior, and repetitive language usage in the fluvoxamine group compared to the placebo group (McDougle et al. 1996).

There have been mixed results in studies reporting the use of fluvoxamine for the treatment of autism in children. In one double-blind cross-over study of fluvoxamine and placebo, 10 out of 18 children were classified as responders, while in another, improvements were shown in behaviors such as eye contact and language use (Fukuda et al. 2001; Sugie et al. 2005). However, an open-label trial and a double-blind, parallel groups comparison with placebo, which both administered fluvoxamine to 18 subjects, classified only 3 and 1 subjects, respectively, as responders (Martin et al. 2003; McDougle et al. 2000).

Numerous studies of adults have shown benefits for the treatment of depression, panic disorder, GAD, and posttraumatic stress disorder (PTSD), for which fluvoxamine is also commonly prescribed (Irons 2005; Stahl 2008). Additionally, several randomized, double-blind studies have demonstrated fluvoxamine's efficacy in the reduction of binge eating, relapse of bulimia nervosa, compulsive buying, and pathological gambling (Irons).

The recommended starting dose of fluvoxamine is 25 mg in pediatric patients and 50 mg in adults, taken before bed. The dose should be increased every 4–7 days in 25-mg increments in children and 50-mg increments in adults until the maximum benefit is reached. The dose should not exceed 200 mg/day in children up to age 11 years and 300 mg/day in adolescents and adults. If the dose is more than 50 mg for the pediatric population or 100 mg for adults, it is recommended for it to be divided into two doses. With uneven doses, the larger one should be taken before bed (U.S. Food and Drug Administration 2011b).

Side Effects

The most common adverse reactions in trials with adults with OCD and depression include nausea, somnolence, insomnia, asthenia, nervousness,

dyspepsia, abnormal ejaculation, sweating, anorexia, tremor, and vomiting. Less frequent side effects include anorgasmia, decreased libido, dry mouth, rhinitis, taste perversion, and urinary frequency in individuals with OCD and agitation, depression, dysmenorrhea, flatulence, hyperkinesia, and rash in pediatric patients with OCD (U.S. Food and Drug Administration 2011b).

As with other antidepressants, fluvoxamine may result in the worsening of depressive symptoms and an increased suicide risk. It should not be used in combination with monoamine oxidase inhibitors or within 14 days of ending treatment with one. Additionally, tizanidine, thioridazine, alosetron, and pimozide should not be coadministered with fluvoxamine. Serotonin syndrome and neuroleptic malignant syndrome have also been reported with SSRIs, including fluvoxamine. There is evidence that the CR form has a lower maximum plasma concentration, reducing the risk of adverse effects (Ordacgi et al. 2009).

See Also

- ▶ Antidepressants
- ▶ Selective Serotonin Reuptake Inhibitors (SSRIs)

References and Reading

- Fukuda, T., Sugie, H., Ito, M., & Sugie, Y. (2001). Clinical evaluation of treatment with Luvox, a selective serotonin reuptake inhibitor in children with autistic disorder. *No to Hattatsu*, 33(4), 314–318.
- Irons, J. (2005). Luvox in the treatment of anxiety disorders. *Neuropsychiatric Disease and Treatment*, 1(4), 289–299.
- Kuzma, J. M., & Black, D. W. (2008). Extended-release Luvox for social anxiety disorder and OCD. *Current Psychiatry*, 7(7), 54–66.
- Martin, A., Koenig, K., Anderson, G., & Scahill, L. (2003). Low dose Luvox treatment of children and adolescents with pervasive developmental disorders: A prospective, open-label study. *Journal of Autism and Developmental Disorders*, 33(1), 77–85.
- McDougle, C. J., Naylor, S. T., Cohen, D. J., Volkmar, F. R., Heninger, G. R., & Price, L. H. (1996). A double-blind, placebo-controlled study of Luvox in adults with autistic disorder. *Archives of General Psychiatry*, 53, 1001–1008.

- McDougle, C. J., Kresch, L. E., & Posey, D. J. (2000). Repetitive thoughts and behavior in pervasive developmental disorders: Treatment with serotonin reuptake inhibitors. *Journal of Autism and Developmental Disorders*, 30(5), 427–435.
- Ordacgi, L., Mendlowicz, M. W., & Fontenelle, L. F. (2009). Management of obsessive-compulsive disorder with Luvox extended release. *Neuropsychiatric Disease and Treatment*, 2009(5), 301–308.
- Stahl, S. M. (2008). *Essential psychopharmacology online*. Retrieved July 15, 2011, from http://stahlonline.com/bridge.org/prescribers_drug.jsf?page=0521683505c36_p205-210.html.therapeutics&name=Luvox&title=Therapeutics.
- Sugie, Y., Sugie, H., Fukuda, T., Ito, M., Sasada, Y., Nakabayashi, M., et al. (2005). Clinical efficacy of Luvox and functional polymorphism in a serotonin transporter gene on childhood autism. *Journal of Autism and Developmental Disorders*, 35(3), 377–385.
- U.S. Food and Drug Administration. (2011a). *Luvox CR label information*. Retrieved from http://www.accessdata.fda.gov/drugsatfda_docs/label/2011/022033s003lbl.pdf.
- U.S. Food and Drug Administration. (2011b). *Luvox label information*. Retrieved from http://www.accessdata.fda.gov/drugsatfda_docs/label/2011/021519s002lbl.pdf.

Fluvoxamine Maleate

► Fluvoxamine

Fluxil

► Prozac™ (Fluoxetine)

fMRI

► Event-Related Functional Magnetic Resonance Imaging (fMRI)

Fontex

► Prozac™ (Fluoxetine)

Food Intolerance

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Definition

Food intolerance refers to the occurrence of physical symptoms relative to ingestion of foods that are not the result of conventional IgE-mediated food allergy. IgE is the subclass of immunoglobulins that cause an individual to make antibodies to food or other compounds that result in allergic symptoms. Food intolerance may be more common than true food allergy. The physical symptoms of food intolerance may impact the skin and the gastrointestinal and respiratory tracts. Central nervous system symptoms like migraines may occur. Food intolerance may manifest in a delayed fashion, unlike IgE-mediated allergy. Food intolerance is attributed to the following mechanisms:

1. Absence of an enzyme necessary for metabolism of an ingested food: An example of this is lactose intolerance. Approximately 10% of Americans are intolerant of the milk sugar, lactose, because of an absence of the enzyme necessary for digestion with resultant symptoms of diarrhea, bloating, and intestinal gas.
2. Symptomatic response to a food or food ingredient based on genetic susceptibility: An example of this is intolerance of natural salicylates. Salicylic acid is the active ingredient in aspirin. Approximately 2.5% of Europeans are intolerant of natural salicylates found in various fruits and vegetables. Other chemicals in food that are associated with intolerance include food additives like monosodium glutamate, nitrites, and artificial food colors.
3. Symptomatic response to a chemical compound in food based on a co-occurring condition: An example of this is development of

physical symptoms relative to histamine ingestion when the enzymatic degradation of this vasoactive amine is impaired by an imbalance of intestinal flora or a medication prescribed for another condition.

4. Non-IgE-mediated immune response: The utility of measuring IgG to foods is controversial and at present does not guide therapy.

Food intolerance needs to be clinically distinguished from IgE-mediated food allergy. This is done by history and physical examination, allergy testing, elimination diets, and double-blind food challenges. Unlike food allergy, many people with food intolerance do not have symptoms with small and infrequent exposures. Dietary avoidance is the treatment.

Families of children in Norway later diagnosed with autism spectrum disorders report more food allergies and intolerance in the first 3 years of life (Bresnahan et al. 2015).

See Also

- [Food Intolerance](#)
- [Gastrointestinal Disorders and Autism](#)

References and Reading

- Bresnahan, M., Hornig, M., Schultz, A. F., Gunnes, N., Hirtz, D., Lie, K. K., Magnus, P., Reichborn-Kjennerud, T., Roth, C., Schjølberg, S., Stoltenberg, C., Surén, P., Susser, E., & Lipkin, W. I. (2015). Association of maternal report of infant and toddler gastrointestinal symptoms with autism: evidence from a prospective birth cohort. *JAMA Psychiatry*, 72(5), 466–474. PMID: 25806498.
<http://www.webmd.com/allergies/guide/food-allergy-intolerances>. Accessed 24 May 2012, WebMD.
- Mullin, G. E., Swift, K. M., Lipski, L., Turnbull, L. K., & Rampertab, S. D. (2010). Testing for food reactions: The good, the bad and the ugly. *Nutrition in Clinical Practice*, 25, 192–198.
- Ortolani, C., & Pastorello, E. A. (2006). Food allergy and food intolerances. *Best Practice and Research Clinical Gastroenterology*, 20(3), 467–483.
- Zopf, Y., et al. (2009). The differential diagnosis of food intolerance. *Deutsches Ärzteblatt International*, 106(21), 359–370. <http://www.ncbi.nlm.nih.gov.ezpmminer.urochester.edu/pmc/articles/PMC2695393/?tool=pubmed>.

Forced-Choice Preference Assessment

- [Preference and Reinforcer Assessment](#)

Formal Complaint

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Definition

Formal Complaint

In General

A formal complaint is an initial filing that starts a civil lawsuit and usually states the basis for a court's jurisdiction, the basis for a plaintiff's claim, and a demand for relief.

Individuals with Disabilities Education Act

Individuals (typically parents) may submit a formal complaint to a child's school district and/or state education agency alleging violation of the Individuals with Disabilities Education Act.

Any party has an opportunity to present a formal complaint regarding the identification, evaluation or educational placement (IEP) of a child, or the provision of a free appropriate public education (FAPE) to such child. The complaint must be filed within 2 years of the date when the parent or public agency knew or should have known about the alleged action that forms the basis of the complaint.

When filing a complaint alleging that a local educational agency has violated a requirement of the Individuals with Disabilities Education Act, the complaint must be written, signed, and must cite the facts upon which the allegation is made and the specific statutory requirement that was violated. The state educational agency must resolve the issues of the complaint within 60 days after it is filed.

Americans with Disabilities Act

If an individual believes that he or any other person has suffered discrimination, he may file a complaint with the Department of Justice. Such a complaint should include contact information, the name of the business, organization, institution, or person that perpetrated the alleged discrimination and a description of the discrimination.

See Also

- [Eligibility \(for Services Under IDEA/ADA, etc.\)](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- Americans with Disabilities Act (ADA), U.S.C. § 12101
 Department of Justice. (2010). *Frequently Asked Questions about Filing an ADA Complaint with the U.S. Department of Justice*. Retrieved from http://www.ada.gov/fact_on_complaint.htm#1
- Garner, B. A. (Ed.). (2009). *Black's law dictionary* (9th ed.). St. Paul: West Publishing.

FOT

- [Finger-Tapping Test](#)

France and Autism

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Historical Background

The first approach to autism in France was based on the psychoanalytic model, in terms of etiology

as well as in treatments. Regarding etiology, the parents' role in the pathology's development was emphasized leading to treatments based on that interpretation. Therefore, in the extent that the child's autism was supposed to be due to relational difficulties, psychotherapy prevailed for many years. There were mostly institutional interventions, implemented in day hospitals. They were initiated by psychiatrists such as Roger Misès and Serge Lebovici. Despite the assertions that psychoanalysts used a multifactorial approach, the treatments were essentially psychological leaving little room for educational aspects. Above all, they preferred a nondirective attitude saying that it was better to wait for "the emergence of desire" rather than to provide teaching. The parents were not directly involved in their child's treatment and had to personally undergo psychotherapy. Only a few facilities took the different dimensions of autism into account and carried out more complete medical explorations. However, in most cases this did not have a real impact on treatment. At the time, parents were not well informed on autism and when the first parent association, ASITP, was founded in the 1960s, it mainly focused on establishing new day hospitals in which this type of approach was implemented.

In France, Gilbert Lelord, who passed away early in the year 2017, was the great pioneer of the scientific approach to autism. He was surrounded by a team of clinicians and researchers among who Catherine Barthélémy, his most brilliant collaborator, gave a quality extension to his initial work. This team largely contributed to changing the image of autism in France by defending the biological cause of this condition. Numerous studies carried out by the INSERM 316 unit have demonstrated that childhood autism is not a psychological pathology but linked to a developmental disorder of the nervous system. Clinicians gathered extensive patient information while providing support to families. Collaboration with the research team resulted in creating the first standardized tools for assessment of children with autism (Barthélémy and Gilbert 1991; Rogé 2017). The different research protocols helped to better understand the

neurophysiological functioning and to determine subgroups of patients. All of this work and research underpinned the reflection on a new therapeutic approach, which culminated with the development of exchange and development therapy (Barthélémy et al. 1995). Parent associations have also significantly contributed to the improvement of people with autism's situation in France even though they initially opposed one another, some continuing to defend the psychiatric approach, while others, very militant, demanded an adapted education like those advocated in most other countries.

In the 1990s, things began to change. Government authorities finally took parents' repeated requests into account. Various commissions were organized. From then on, the French classification of Mental Diseases CFTMEA (1993), which still classified autism in infantile psychoses, was abandoned by the Ministry of Health. The World Health Organization's CIM10 became the new classification reference. At that time, three major diagnostic centers were recognized, one in Tours, one in Toulouse, and one in Paris. Subsequently resource centers were created and later developed throughout France. The specific way in which individuals with autism function was finally acknowledged and new units delivering behavioral and developmental programs were created. In 1995, Simone Veil, Minister of Social Affairs, Health and the City, adopted a 5 year, 100 million francs (approximately seven million dollars) plan of action for autism. The problem of the exclusion of individuals with autism was brought before the Council of Europe. In 1996, the Chossy Act was voted, which assigns a disability status to autism and guarantees these individuals the right to access care in a multidisciplinary approach. In 2003, Deputy Chossy reported to the government denouncing the situation of individuals with autism and called for a plan of action to enforce their rights. Despite all these efforts, France remained behind. Parents of "Autism France" took up action before the European Court of Justice which condemned France on several occasions for failing to respect the right to education of individuals with autism. This will be

followed by recommendations for diagnosis, the 2005 Disability Act, which guarantees equal rights and opportunities, the referral to the National Ethics Advisory Committee in 2007, which highlights the lack of care and the resulting distress in families, recommendations for a scientifically validated intervention, and three autism action plans that allowed new funding to be allocated to developing the first Resource Centers and specific interventions. In 2012, autism was declared Great National Priority, leading to various awareness-raising campaigns and strong media coverage. Recommendations on intervention for adults are currently being developed and an international scientific committee has been set up to determine what the main axes of the fourth autism action plan should be.

Legal Issues, Mandates for Service

The disabilities Act of 11 February 2005 and Decree No 2005-223 of 11 March 2005 defines the goals of support services in social and medical institutions. This Act aims to improve inclusion of individuals with disabilities. Based on the principle of equality, the act recognizes that a disabled person's right to compensation is a societal duty and is not dependent on appeal to pity or charity. It aims to restore equal opportunities for all citizens. Among other things, this Act resulted in the increase of requirements regarding accessibility to urban areas and access to employment for disabled individuals. A National Solidarity Fund for Autonomy (CNSA) was established to finance this Disabilities Act, but it is not specifically dedicated to autism.

After the enactment of the Chossy Law in 1996, various parent association actions led the government to define a new policy in favor of autism through successive autism action plans.

During the first plan of action from 2005 to 2007, Autism resource centers are created. Their main tasks are to inform and guide the affected people and their families. They must organize diagnoses and multidisciplinary assessments of children and adults, develop individual

evaluations in order to establish a personalized project for each person, facilitate contact and information within professional circles, and develop local documentation centers. Improvement in parent and professional training is planned. Adapted services are developed but still extremely insufficient. The plan of action also advocates better schooling for children with autism in mainstream classrooms.

The second autism plan of action between 2008 and 2010 provided 4100 openings in services for children and adults with autism. The High Authority for Health (HAS) had the task of creating an international, scientific, and multi-disciplinary database which would serve as a reference. Together with the National Agency for the Evaluation and the Quality of Social and Medical Social Establishments and Services (ANESM), they must define guidelines on good practice in the field of autism.

The third autism plan of action organizes early diagnosis and intervention. It stresses the importance of lifelong support with schooling for children and social and professional inclusion for adults. Better support for families is advocated and the resource centers are responsible for organizing it. The action plan also calls for continued efforts to support research on the origins of autism, the mechanisms involved, indicators facilitating early diagnosis, evidence-based treatments, and actions promoting social inclusion.

These three Autism Action Plans have helped improve common knowledge on autism and have attempted to change practices. They made it possible to affirm that psychoanalysis was not a treatment for autism and promote scientifically validated treatments. Resources have been allocated to developing quality services for patients and their families as well as to research. Nevertheless, the situation remains critical as not all the measures planned by the government have been implemented. Many families are still struggling to find suitable services. Despite government recommendations, the services provided in each region vary greatly. While many professionals have turned to scientifically validated practices, others remain on old positions and continue to apply methods that

families no longer want. The Autism Action Plans generated substantial funds yet they are still insufficient to adequately assist people with autism and their families in France.

A fourth Autism Action Plan is being considered. The main aim should be to reinforce the progress of the previous action plans. The current minister organized a committee of international experts to support her approach. A new government was elected in 2017. It is committed to continuing efforts for people with autism. The 4th autism plan should focus on continuity of care at all ages and develop new facilities for adults with autism. Real progress can only be achieved through ground breaking decisions such as allocating financial aid based on compliance with recommendations and the application of scientifically validated practices.

Overview of Current Treatments and Centers

Due to the long history of psychiatric medicalization in France, services for individuals with autism primarily depend on the Ministry of Social affairs and Health. This Ministry delegates the organization of services to a ministry under its supervision: Ministry of disabled people and the fight against exclusion. The ministries of education, labor, cities, youth, and sports are secondarily involved, usually for specific actions or for creating partnerships.

Therefore, the Ministry of disabled people and the fight against exclusion plays the primary role in organizing services. A national solidarity fund was established to finance care services, but it is not specific to autism. It covers all disabilities as well as assistance to the elderly.

Two national agencies, ANESM (National Agency for the Evaluation and the Quality of Social and Medical Social Establishments and Services) and HAS (High authority of Health) are responsible for monitoring the quality of services and have initiated a process of reflection leading to recommendations on good practice in cooperation with parent associations and experts in autism. This has helped the situation in France

evolve. There is a better understanding of autism and new services correspond more accurately to the needs of individuals with autism. Nevertheless, widely disseminating this knowledge is necessary to change the outdated services and develop new options for people with ASD.

On a regional level, local health agencies publish calls for projects based on the development policy launched by the Ministry. They open up the competition to project promoters which can either be associations or hospital services. They receive funds from the CNSA (National Solidarity Fund for Autonomy) and attribute these funds to the selected projects. They then monitor the use of allocated resources.

The MDPH (Departmental Office of disabilities) manages orientations. Its teams assess and determine the degree of disability and orient towards available services. The disability rate opens rights to financial compensation allowances (AEEH = disabled child education allowance and AAH = adult disability allowance) for disabled people and a possible compensatory allowance (PCH = Disability Compensation Benefit). The commission that allocates financial aid (Committee on rights and autonomy of persons with disabilities = CDAPH) consists of only professionals but service users are represented by parent associations. Resource centers inform families and professionals about services.

Services for Children

For children and adolescents with disabilities, the right to education is associated to an obligation of schooling or appropriate support. It can be implemented within the schools' "ordinary" classrooms. Enrollment in the mainstream is favored with adaptations (teacher's aid, teacher, and staff training). Schooling can also be implemented in a collective arrangement School Integration Classes – CLIS – or Localized Units for Inclusive Education – ULIS – in higher education. Enrollment can be full-time or part-time with the support of a specialized institution if needed. Self-contained classrooms for children from 3 to 6 years of age were recently created in preschool. These classes are opened to a small number of children (maximum seven) who could not be

enrolled despite the help of a teachers aid. In these specialized classrooms, a multidisciplinary team applies a personalized program for each child. They also benefit from inclusion in regular classrooms (mainstreaming) therefore attending school full-time.

Different Specialized Structures Can Take Care of Children with Autism

For young children with sensory, motor, or mental deficits up to the age of 6, CAMSP (Early medico-social centers) are involved in the early detection of disability, outpatient treatment, rehabilitation, and family-directed services. They offer consultation, rehabilitation, as well as individual or group activities promoting child development and social integration. With parental consent, concerted actions can be carried out in the child's natural environment (day care centers, school, or recreation center).

For children able to attend school, different outpatient services are available: CMP (Medico-psychological centers) are consultation services for people in need of psychological support. There are infant-juvenile CMPs for children and adolescents up to 16 years of age. The care is free and multidisciplinary. CMPP (medical psychoeducational centers) are in charge of diagnosis and treatment in outpatient care for children aged 3–18 or 20 years, suffering from neuropsychiatric disorders or behavioral disorders. Treatments are individual psychotherapy, psychotherapy, family therapy, speech and language rehabilitation, and psychomotor therapy. The child or adolescent stays in his family, school, and social environment. A SESSAD (Special Education and Home Care Services) aims to enable children with various disabilities within their natural environment allowing them to continue developing in their family and social environment. The multidisciplinary team's main goal is the early management of the child, family-directed services, academic support, and self-help skills. Services can be provided in the child or adolescents natural environment. Early intervention SESSAD programs have recently been established. They can accommodate children starting from 18 months who can benefit from programs like the ESDM.

Day hospital institutions take care of patients on a full-time or part-time basis during the day. A team composed of psychiatrists, psychologists, speech therapists, occupational therapist, educators, and social workers care for the patient. Usually special education classrooms are implemented in the day hospital but are not a priority over treatment. Some day-hospitals include mainstreaming.

The other structures care for children and adolescents with intellectual disabilities on a full-time basis regardless of the degree of their disability. IME (Medical-Educational Institutes) regroup several categories of structures: "Medical-Pedagogical Institutes" (IMP) provide full residency, semi-boardings, temporary reception or day schooling for children aged 3–6 to 14 years old. Medical-Professional Institutes (IMPRO) accept children and adolescents ranging from 14 to 20 years old. Here they receive health and educational care through a therapeutic and pedagogical approach supposed to promote the development of the child or adolescent. The acquisition of autonomy skills (transport, management of a budget, etc.) and preprofessional training are also important goals. IMPROs provide vocational training (which may lead to a Diploma) for integration into the protected or ordinary work environment. ITEP (Therapeutic, educational, and pedagogical institutes) are medical-social structures which take in children and adolescents with intact intellectual and cognitive potential but who present psychological difficulties and behavioral disorders which severely disrupt socialization and access to learning. The different autism action plans favored the creation of experimental structures in which behavioral and developmental models are applied in accordance with the recommendations of the High Authority of Health. Parents hope that this type of structure will continue to develop.

The structures caring for children with disabilities are therefore numerous and diverse, but they are often inadequate for children with autism. Therapeutic approaches are often unspecific due to the lack of professional training. This situation is changing through the different autism action plans, but many parents are still unhappy with

what is being offered and availabilities in the structures applying the recommendations of the HAS are still insufficient. As a result, many children are on waiting lists with no options. Parents, who have no other alternative or who do not agree the institutions approach, can call on psychologists and educators who work in private practice. However, this represents a significant cost, sometimes preventing the child to access sufficient treatment, whereas the treatments in institutions are paid for by the state.

Services for Adults

Services available to adults are either in the health or in the medical-social field. In the psychiatric services of the health sector, people with autism are often mixed with patients having various psychiatric pathologies; therefore, their specific needs are not met. They may also suffer from the violence of other psychiatric patients. Some professionals have become aware of this serious problem and have organized smaller units for people with autism. However, generally these adults are often left on their own, without specific support causing many behavioral disorders.

In the medical-social sector (with or without identified specific services for autism), there are different structures. MAS (Specialized Care Homes) receive disabled adults who have acquired little autonomy and whose condition requires medical supervision and constant care. These specialized care homes or MAS are nursing homes. They can accommodate people with multiple disabilities, usually with a severe mental disability or people who acquired disabilities (brain injury). Some MAS also welcome people with autistic syndromes but do not provide specialized treatment. Generally, MAS provide year-round accommodation, day care services, or temporary accommodation. Those being cared for in a MAS lack autonomy and are unable to care for themselves in daily life responsibilities. Assistance is needed for activities of daily living and for health monitoring. In general, people accommodated in MAS have less autonomy than those in other health and welfare services for disabled adults (foster home or medicalized foster home), but MAS residents

are not within the psychiatric sector. A person admitted to this structure may end his days there. The MAS are financed by health insurance funds, therefore the services of the regional health agencies (ARS).

Adult Foster Homes (FAM) are nursing homes. They welcome people with multiple disabilities, severely disabled adults unfit for any professional activity who need assistance with daily living tasks or medical monitoring. They can also accommodate seniors with disabilities. Generally, FAM residents have more autonomy than those in the specialized care homes (MAS). Medicalized FAMs are funded in part by health insurance, for care and medical staff, in part by the departmental council, for accommodation and entertainment. That is why the FAM were also called “dual pricing home.”

There are other forms of accommodation which depend on a person's level of autonomy. Living spaces or homes are very small structures providing a personalized support. Legally, these are not social institutions or services. However, they are obligated to implement individual life projects, taking into account the resident's expectations and needs.

Group homes for disabled adults provide accommodation and care for disabled individuals who work in either an ordinary environment, an institution, a support service through work (ESAT), or an adapted enterprise during the day. Foster homes offer accommodation, care, and medical assistance for physically or mentally disabled working adults. A SAMSAH (Medical and medical-Social support services for Disabled Adults) tries to maintain people with disabilities in the mainstream, by helping restore social ties (family, school, business, etc.) and promoting access to services offered by the community. In addition to these benefits, they provide medical assistance. Temporary Homes for disabled adults have several objectives: first of all, they allow the disabled person to change his or her living environment and if necessary, escape from time to time to avoid the risk of confinement; secondly, they allow “caregivers” to benefit from respite time. Temporary stay is organized for a limited period of up to 90 days per year, full or part time,

with or without accommodation, including day care. It can be arranged in a sequential mode, that is, to say by programmed periods throughout the year.

For the most autonomous, services are organized to allow access to employment.

Support services through work (ESAT), formerly called CAT (Centre for assistance through work), provide services to disabled adults with a working capacity of less than one third of the normal capacity to take up employment in an ordinary environment. Vocational Rehabilitation Centers provide training for disabled adults to facilitate integration or reintegration into either an ordinary or protected working environment. A SAVS (Accompanying Service for accessing society) is a structure that assists disabled adults in fulfilling their life project by helping maintain or restore family, social, academic, or professional ties and by facilitating access to community services and events.

Experimental Facilities for disabled adults are aiming to promote new types of support, organize a more rational way to access to the care system facilities, a better coordination of this support, as well as training and prevention for behavior disorders and health issues.

There are therefore many structures in France for adults with disabilities. Unfortunately, they are rarely suitable for people with autism. The different autism action plans have partially corrected this situation, but there is still a lot to be done in order to provide all adults with the assistance that suits their specific needs. Too many people are still left with no options in France. These individuals live in their parents' home. This is an important issue because aging parents, struggling to handle the situation, are concerned about the future of their child and what will happen when they are no longer around.

Overview of Research Directions

In France, a majority of the research units belong to large organizations such as the CNRS (National Center for Scientific Research) and INSERM (National Institute for Health and

Medical Research or Institut Pasteur). These units are located in university hospitals, universities, or specific institutes. Other laboratories are part of universities.

Thomas Bourgeron's team at Pasteur Institute works on the genetics of autism and sleep disorders related to melatonin production. This team found several genes involved in autism, such as SHANK1, SHANK2, SHANK3, and NRXN1, which also play a role in the transmission of neuronal information. These researchers also proposed a murine model of autism (Jamain et al. 2003; Durand et al. 2007; Delorme et al. 2013; Bourgeron 2015).

The Fundamental Foundation supports research in psychiatry, including research in the field of high-functioning autism. The teams of the Asperger Experts Centers of Créteil and Robert Debré recently published a study comparing high-level autism and schizophrenia in Multimodal MRI. The results reveal the coexistence of common and opposite susceptibility factors in these two pathologies (Katz et al. 2016). InFoR-Autism is a new project whose objective is to study patients' clinical and cognitive profiles to look for biomarkers (clinical, neuroanatomic, immunological, and biochemical) and to identify genetic factors involved in autism. The establishment of a 2-year cohort follow-up of patients, healthy subjects, and relatives (parents, brothers, or sisters of the patients included in the study) is planned.

An INSERM team in Tours pursues research on the evaluation tools and the neurophysiological mechanisms involved in autism. Their main research uses MRI, EEG, and eye tracking. These technologies are used to study the maturation of human face exploration (Aguillon et al. 2016), the differences between ASD and Mental deficiency (Bonnet-Brilhault et al. 2016), and atypical brain mechanisms in ASD (Thillay et al. 2016). Currently, the team is engaged in a new project "Emotional Attention and Attention Related to Change in Autism."

Other examples of research carried out by university teams are eye-tracking research (Guillon et al. 2014, 2015, 2016), research on the effect of slowing down movement of what will be

perceived (Tardif et al. 2007), and research on screening (Baduel et al. 2016). New research directions include treatment evaluation and cost-effectiveness studies.

It is impossible to cite all the research teams here and these are merely examples, but although France has lagged behind in the implementation of adapted treatments, there are some interesting achievements in the field of research. Public funds for research are, however, rather limited and researchers must solicit private foundations and contributors. Involvement in international programs, particularly in Europe (e.g., Salomone et al. 2015), reinforces French research. Recently INSERM created the Autism Research Network that will bring together the efforts of different teams and strengthen their resources. This initiative will hopefully increase knowledge and enhance practices on autism.

Overview of Training

The training of professionals in France is very inadequate. In physicians' training, only a small part of the curriculum focuses on autism, except for university centers where specialists and researchers work in neurodevelopmental disorders. In psychology departments, curriculums vary greatly from one university to another and some professors still teach old models of autism highlighting psychoanalysis. Fortunately, some researchers teach courses that are up to date and correctly train their students. The same problem exists in the various institutes that train educators, social workers, speech therapists, and occupational therapists. In order to compensate their lack of specific training, continuing education qualifications have been created. After graduation they can receive an up-to-date training. Associations have also created training courses to complement professional training.

Social Policy and Current Controversies

The current government intends to maintain its efforts to develop adapted services in the next

autism action plan. Progress is still needed in early diagnosis and intervention as well as in appropriate services for school-aged children, adolescents, and adults, and parents are fighting for this. However, France is still lagging behind and budgets are limited. In addition, some teams still refuse to follow HAS recommendations. Training needs to be profoundly modified so that all medical professionals have solid knowledge on autism and are able to diagnose it as early as possible. Other professionals should be able to implement appropriate treatments. It would also be important for the general public to be better informed in order to help encourage the inclusion of people with autism in society.

See Also

<http://social-sante.gouv.fr/grands-dossiers/l-autisme/>
<http://www.autisme-france.fr/>
<http://www.sesame-autisme.com/>
<http://www.arapi-autisme.fr/>
<http://www.fondationorange.com>

References and Reading

- Aguillon-Hernandez, N., Roché, L., Bonnet-Brilhault, F., Roux, S., Barthélémy, C., & Martineau, J. (2016). Eye movement monitoring and maturation of human faces exploration. *Medical Principles and Practice*. <https://doi.org/10.1159/000447971>.
- Baduel, S., Guillon, Q., Afzali, M. H., Foulon, N., Kruck, J., & Rogé, B. (2016). The French version of the modified-checklist for autism in toddlers (M-CHAT): A validation study on a French sample of 24 month-old children. *Journal of Autism and Developmental Disorders*, 1–8. <https://doi.org/10.1007/s10803-016-2950-y>.
- Barthélémy, C., & Gilbert, L. (1991). *Les échelles d'évaluation clinique en psychiatrie de l'enfant*. Paris: Expansion scientifique française.
- Barthélémy, C., Hameury, L., & Lelord, G. (1995). *L'autisme de l'enfant: la thérapie d'échange et de développement*. Paris: Expansion scientifique française.
- Bonnet-Brilhault, F., Alirol, S., Blanc, R., Bazaud, S., Marouillat, S., Thépault, R. A., Andres, C. R., Lemonnier, É., Barthélémy, C., Raynaud, M., Toutain, A., Gomot, M., & Laumonnier, F. (2016). GABA/glutamate synaptic pathways targeted by integrative genomic and electrophysiological explorations distinguish autism from intellectual disability. *Molecular Psychiatry*, 21(3), 411–418.
- Bourgeron, T. (2015). From the genetic architecture to synaptic plasticity in autism spectrum disorders. *Nature Reviews Neuroscience*, 16, 551–563.
- Delorme, R., Ey, E., Toro, R., Leboyer, M., Gillberg, C., & Bourgeron, T. (2013). Progress towards treatments for synaptic defects in autism. *Nature Medicine*, 19, 685–694.
- Derguy, C., M'Bailara, K., Michel, G., Roux, S., & Bouvard, M. (2016). The need for an ecological approach to parental stress in autism spectrum disorders: The combined role of individual and environmental factors. *Journal of Autism and Developmental Disorders*, 46(6), 1895–1905. <https://doi.org/10.1007/s10803-016-2719-3>.
- Durand, C., Betancur, C., Boeckers, T. M., Bockmann, J., Chaste, P., Fauchereau, F., Nygren, G., Rastam, M., Gillberg, I. C., Anckarsäter, H., Sponheim, E., Goubran-Botros, H., Delorme, R., Chabane, N., Mouren-Simeoni, M. C., de Mas, P., Bieth, E., Rogé, B., Héron, D., Burglen, L., Gillberg, C., Leboyer, M., & Bourgeron, T. (2007). Mutations of the synaptic scaffolding protein SHANK3 are associated with autism spectrum disorders. *Nature Genetics*, 39, 25–27.
- Guillon, Q., Hadjikhani, N., Baduel, S., Kruck, J., Arnaud, M., & Rogé, B. (2014). Both dog and human faces are explored abnormally by young children with autism spectrum disorders. *Neuroreport*. <https://doi.org/10.1097/WNR.0000000000000257>.
- Guillon, Q., Afzali, M. H., Rogé, B., Baduel, S., Kruck, J., & Hadjikhani, N. (2015). The importance of networking in autism gaze analysis. *PLoS One*, 10(10), e0141191. <https://doi.org/10.1371/journal.pone.0141191>.
- Guillon, Q., Rogé, B., Afzali, K., Baduel, S., Kruck, J., & Hadjikhani, N. (2016). Intact perception but abnormal orientation towards face like objects in young children with ASD. *Scientific Reports*, 6, 22119. <https://doi.org/10.1038/Srep22119>.
- Jamain, S., Quach, H., Betancur, C., Råstam, M., Colineaux, C., Gillberg, I. C., Soderstrom, H., Giros, B., Leboyer, M., Gillberg, C., & Bourgeron, T. (2003). Mutations of the X-linked neurologins NLGN3 and NLGN4 are associated with autism. *Nature Genetics*, 34, 27–29.
- Katz, J., d'Albis, M. A., Boisgontier, J., Poupon, C., Mangin, J. F., Guevara, P., Duclap, D., Hamdani, N., Petit, J., Monnet, D., Le Corvoisier, P., Leboyer, M., Delorme, R., & Houenou, J. (2016). Similar white matter but opposite grey matter changes in schizophrenia and high-functioning autism. *Acta Psychiatrica Scandinavica*, 134(1), 31–39. <https://doi.org/10.1111/acps.12579>. Epub 2016 Apr 22.
- Rogé, B. (2017). Behaviour summarized evaluation – Revised (BSE-R). In F. R. Volkmar (Ed.), *Encyclopedia*

of autism spectrum disorders (2nd ed.). New York: Springer.

- Salomone, E., Beranová, S., Bonnet-Brilhault, F., Briciet, L. M., Budisteanu, M., Buitelaar, J., Canal-Bedia, R., Felhosi, G., Fletcher-Watson, S., Freitag, C., Fuentes, J., Gallagher, L., Garcia, P. P., Gliga, F., Gomot, M., Green, J., Heimann, M., Loa, J. S., Kaale, A., Kawa, R., Kylliainen, A., Lemcke, S., Markovska-Simoska, S., Marschik, P. B., McConachie, H., Moilanen, I., Muratori, F., Narzisi, A., Noterdaeme, M., Oliveira, G., Oosterling, I., Pijl, M., Pop-Jordanova, N., Poustka, L., Roeyers, H., Rogé, B., Sinzig, J., Vicente, A., Warreyn, P., & Charman, T. (2015). Use of early intervention for young children with autism spectrum disorder across Europe. *Autism*, 1–17. <https://doi.org/10.1177/1362361315577218>.
- Tardif, C., Lainé, F., Rodriguez, M., & Gepner, B. (2007). Slowing down presentation of facial movements and vocal sounds enhances facial expression recognition and induces facial-vocal imitation in children with autism. *Journal of Autism and Developmental Disorders*, 37(8), 1469–1484.
- Thillay, A., Lemaire, M., Roux, S., Houy-Durand, E., Barthélémy, C., Knight, R. T., Bidet-Caulet, A., & Bonnet-Brilhault, F. (2016). Atypical brain mechanisms of prediction according to uncertainty in autism. *Frontiers in Neuroscience*. <https://doi.org/10.3389/fnins.2016.00317>.

Fraternal Twins

► Dizygotic (DZ) Twins

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Butler Hospital, Brown University, Providence, RI, USA

Name and Degrees: Volkmar, Fred R.

- B.S., University of Illinois, with high honors (1972)
- M.A. (psychology), Stanford University (1976)

- M.D., Stanford University School of Medicine (1976)
- M.A. (Hon), Yale University (1999)
- D.Sc. (Hon), University of Illinois (2013)

Major Appointments

- Director, Yale University Child Study Center, New Haven, CT (2006–2014)
- Chief of Child Psychiatry, Yale New Haven Hospital, New Haven, CT (2006–2014)
- Irving B. Harris Professor of Child Psychiatry, Psychiatry, Pediatrics, and Psychology, Yale University, New Haven, CT
- Director, Autism Program, Yale University Child Study Center, New Haven, CT (1983–2006)

Major Honors and Awards

- International society for Autism Research Lifetime Achievement Award, Atlanta, GA, 2014
- Distinguished Contributor to the Field Award, Autism Services & Resources Connecticut, 2014
- Frank J. Menolascino Award for Work in Developmental Disabilities, American Psychiatric Association, Philadelphia, 2013
- Honorary Doctor of Science, University of Illinois, 2013
- Lauretta Bender Award, New York State Psychiatric Institute, 2012
- George Tarjan Award for Research in Developmental Disabilities, American Academy of Child and Adolescent Psychiatry, 2007
- Emily and Frank M Puzio Award, Eden Institute Foundation and Princeton Lecture Series, 2007
- Blanche F. Ittleson Award, American Psychiatric Association, 1997

Landmark Clinical, Scientific, and Professional Contributions

- Over \$10 million in grants from the NIH, including for the projects “The Social

Neuroscience of Autism and Related Disorders” and the “Neurobiology and Genetics of Autism and Related Conditions”

- Over 200 articles in peer reviewed journals, including the seminal papers “Seizure Disorders in Autism (1990)” and “Field trial for autistic disorder in DMS-IV”
- Over 150 book chapters and editor of multiple books on autism spectrum disorders
- Extensive service on local, national, and international committees including the APA Child and Adolescent Disorders Work Group for DSM-IV, the Committee on Disorders of Infancy and Early Childhood for DSM-IV (chair), and the APA Disorders of Infancy Work Group for DSM-5. On advisory board for Autism Genome Project since 2006 and reviewer for multiple organizations including the NIMH, Wellcome Trust, Autism Speaks, and the Medical Research Foundation of the UK
- Editor of the Journal of Autism and Developmental Disorders, the Encyclopedia of Autism, and the definitive “Child and Adolescent Psychiatry: A Comprehensive Text”

Biography

Fred Robert Volkmar was born in Illinois in 1950 and received an undergraduate degree in psychology from the University of Illinois in 1972. He then attended Stanford University where he graduated with both an M.D. and a master’s degree in psychology. During his time at Stanford, he was engaged in basic science research studying dendritic branching in rats, with the first publication of his career appearing in the journal *Science*. Dr. Volkmar then completed a psychiatry residency at Stanford before coming to the Yale Child Study Center for a fellowship in Child and Adolescent Psychiatry. He remained at Yale for the duration of his career, where he founded the Developmental Disabilities Clinic in 1982 and engaged in landmark research which spanned from efforts at

understanding the basic neuroscience of ASD to clinical research exploring the implications of changes in the definition of autism and related disorders over time. Dr. Volkmar led the DSM-IV field trial to develop the diagnostic criteria for autism and Asperger’s syndrome – this work was critical in bringing the diagnosis of Asperger’s into the public consciousness. Dr. Volkmar cofounded an undergraduate course on autism that was the first of its kind in the world and continues to be offered at Yale every year. Dr. Volkmar was the director of the Yale Child Study Center for 9 years, following which he returned to clinical work, teaching and research in the department as the Irving B Harris professor of Child Psychiatry, Psychology, and Pediatrics. Dr. Volkmar has an extremely impressive legacy of mentorship, having mentored many now-famous ASD scholars such as Ami Klin, Christopher McDougle, and James McPartland. Dr. Volkmar is married to Lisa Weisner and has two daughters.

See Also

http://childstudycenter.yale.edu/faculty_people/fred_volkmar.profile

https://en.wikipedia.org/wiki/Fred_R._Volkmar

References and Reading

- Volkmar, F. R., & Greenough, W. T. (1972). Rearing complexity affects branching of dendrites in the visual cortex of the rat. *Science*, 176(4042), 1445–1447.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 5–41). Hoboken: Wiley.
- Volkmar, F. R., & Nelson, D. S. (1990). Seizure disorders in autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 29(1), 127–129.
- Volkmar, F. R., et al. (2004). Autism and pervasive developmental disorders. *Journal of Child Psychology and Psychiatry*, 45(1), 135–170.

Free Appropriate Public Education

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Definition

Free appropriate public education (FAPE) is a term that is used to describe the federal mandate that all school-aged special education students be provided with special education and related services at public expense. The word “free” means that the individual is entitled to daily schooling at no extra cost to the family. The word “appropriate” means that the student’s education must be individualized to the unique learner’s needs as decided upon during the development of the student’s individualized education program (IEP). The word “public” refers to who will be paying for, supervising, and directing the learner’s education. The word “education” means that children who qualify for special education are entitled to schooling like their same-aged, typical peers. The services provided under FAPE must meet the standards of the local state education agency in addition to being provided at public expense to school-aged children from preschool to high school in accordance with the IEP.

For children with a diagnosis of autism, this means that the majority of their weekdays from the time they are 3 years old will be spent in a public school classroom. An individualized family service plan or an IEP is created for a student when they enter the school system. The IFSP or IEP team consists of several members whose input is critical to the development of an IFSP/IEP: parents, school personnel, and anyone else that the family feels should be a part of the team. Together, the team will decide on the least restrictive environment and the most appropriate setting for the individual with autism. For individual students, this means different settings. An appropriate education for one student may be to be included with same-aged peers in his or her home school. An appropriate education

for another student may be a self-contained classroom for children with autism within a comprehensive school. When the IEP team has come to an agreement on what is appropriate for a student, the student’s FAPE can begin.

See Also

► [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- Snell, M. E., & Brown, F. (2006). *Instruction of students with severe disabilities* (6th ed.). Upper Saddle River: Pearson Educational.
- Wright, P. W. D., & Wright, P. D. (2007). *Wrightslaw: Special education law* (2nd ed.). Virginia: Harbor House Law Press.

Free Recall

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Definition

Free recall is a paradigm used in memory testing. It involves the presentation of information, such as a list of words or images, to the examinee, followed by a period of either immediate or delayed recall, during which the examinee is asked to produce as much of the presented information as possible. This recall can be oral or written. The primacy effect and recency effect, presented in a classic study by Murdock (1962), both affect free recall: “primacy effect” is the ability to recall information presented first more easily, and “recency effect” is the ability to recall information presented last more easily. Free recall of verbal stimuli tends to be an area of relative weakness for individuals with autism spectrum disorders because they may be less able than

individuals without ASD to make subjective connections across words that, in turn, assist with their recall (Gaigg et al. 2008). Children with ASD have been found to perform more compared to children without ASD when recall is cued or assisted, not free (Tsatsanis 2005).

See Also

- [Memory](#)
- [Wechsler Memory Scale \(All Versions\)](#)

References and Reading

- Gaigg, S. B., Gardiner, J. M., & Bowler, D. M. (2008). Free recall in autism spectrum disorder: The role of relational and item-specific encoding. *Neuropsychologia*, 46(4), 983–992.
- Hasher, L. (1973). Position effects in free recall. *American Journal of Psychology*, 86(2), 389–397.
- Murdock, B. B. (1962). The serial position effect of free recall. *Journal of Experimental Psychology*, 64(5), 482–488.
- Tsatsanis, K. D. (2005). Neuropsychological characteristics of autism and related conditions. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 365–381). Hoboken: Wiley.

Free-Operant Preference Assessment

- [Preference and Reinforcer Assessment](#)

Friendship Satisfaction in Children with ASD

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Albert J. Solnit Children's Center, Middletown, CT, USA

Definition

Friendship satisfaction is the overall quality that individuals perceive their relationships with friends.

Historical Background

Childhood friendship is a close connection between children based on companionship capabilities, reciprocity, and stable social interactions with a peer for greater than 6 months (Freeman and Kasari 1998). The importance of friendships was highlighted since the 1950s. Literature revealed in the 1980s that children's expectations for friendships were to improve social-emotional statuses. In addition, friendship was shown to function as a protective factor against bullying (Hodges et al. 1999). The positive effects such as enhanced self-worth, protection, and emotional growth warranted the creation of friendship scales to evaluate other possible qualifications and quantifications of relationships. For example, the Friendship Qualities Scale is a devised instrument used to assess the quality of children's and early adolescents' friendships according to five domains including companionship, conflict, help/aid, security, and closeness (Bukowski et al. 1994).

Perceived conflict in friendships was associated with higher levels of school loneliness and avoidance and lower levels of overall school engagement in boys. For both boys and girls, validation through friendships positively predicted improvements in children's school attitudes and identification of support. However, perceived exclusivity in friendships was associated with lower levels of school achievement (Ladd et al. 1996).

Companionship, intimacy-trust, and affection are distinguishable factors for friends vs. non-friends among older children during preadolescence and adolescence (Buhrmester et al. 1990). Additionally, research indicates children with secure attachments (intact trust and intimacy) with their caretakers are able to form more positive and responsive friendships compared to children with insecure parent attachments, yielding securely attached children to seek similar qualities with their companionships (Lieberman et al. 1999).

Friendship intensity, defined by the frequency that individuals saw their friends, and quality, measured by the satisfaction with friendships were found to be directly correlated to life satisfaction (Amati et al. 2018). The formation and

maintenance of friendship satisfaction is achieved through positive interpersonal and social-cognitive skills, for example, the ability to form intimate bonds and the ability to empathize (Schneider et al. 2001). The friendships of children with autism spectrum disorder (ASD) differ in quality and quantity from typical developing children due to their social-emotional limitations and deficits with their ability to understand and embrace different perspectives (e.g., theory of mind) (Bauminger et al. 2007). The logico-affective hypothesis postulates that individuals with ASD use alternative cognitive strategies to hurdle over socio-emotional difficulties that otherwise come naturally to non-autistic individuals (Bauminger et al. 2009).

Current Knowledge

Empirical evidence commonly suggests problems with reciprocal friendships with same-age peers in children with ASD. Parents' reports have also demonstrated that friendship is uncommon in children with low-functioning ASD compared to those with typical development. Conversely, high-functioning children with autism spectrum disorder (HFASD) in middle childhood and adolescence were often identified to have at least one friend, who usually was less socially impaired (Bauminger et al. 2008).

Exposure to typical peers appears to have significant effects on friendship behaviors for ASD children. Participants in mixed (ASD and typical developing peers) friendship dyads showed increased responsiveness, stronger receptive language skills, and greater positive social assimilation with closer physical proximity, communication, appropriate smiling, and eye contact and demonstrated more complex coordinated play than in the non-mixed dyads (Bauminger et al. 2007). Intellectual functioning and language abilities appear to be important potential mechanisms for supporting friendship capacities in ASD.

Studies show fewer associations have been found between loneliness and friendship for children with ASD compared to their typically developing counterparts, suggesting less understanding

of the relation between loneliness and friendship. Implications of these results are discussed for conceptualizing the social deficits in autism. Albeit having reports of having at least one friend, children with ASD may suggest poorer quality of their friendships in terms of companionship, security, and help (Bauminger and Kasari 2000; Petrina et al. 2016, 2017).

Conversely, other studies elucidate that children with HFASD and their friends perceived friendship qualities similarly, suggesting that pre-adolescents with HFASD have capacities for interpersonal awareness which positively contribute to friendship satisfaction. Current studies which examine the level of friendship satisfaction of children with ASD and their friends with and without diagnosis of ASD show relatively high levels of friendship satisfaction among all groups without significant differences (Bauminger et al. 2008).

Research has shown that friendship impacts the overall experience of mainstream school for autistic children. Furthermore, autistic children's social motivation likely determines both the nature and extent of their friendships. Adults can play an active and integral role in supporting their ASD children's friendships, but this can be conflictual to the kids' wants sometimes. These findings not only underscore the need to ascertain the perspectives of young people with autism on their friendships but also for adult caretakers to be more socially and ethically considerate of the timing and quality of intervention (Calder et al. 2012).

Future Directions

Future studies embrace the potential of identifying particular differences in social-emotional functioning and friendship building capacity specifically within the ASD population. These uncoverings can yield more clarity about friendship development and maintenance by comparing children within the spectrum who form friendships with other children with ASD in comparison to children with ASD who do not have friends. Additionally, exploring the differences of the nature of friendship within the same child with ASD while playing with a friend with a disability versus

playing with a friend with typical development can provide better understanding of the factors that contribute to a satisfactory friendship for an ASD child.

The notion that typically developing peers enhance and somewhat scaffold the social development of preadolescent children with HFASD embrace the postulation that the HFASD child uses the typical peer as a role model for learning normative social behaviors. More studies are indicated to explore the quality of friendships longitudinally through adolescence and adulthood to assess to what extent the social-emotional skills of ASD are consolidated through later years.

In other studies which explore ASD and typical development peer dyads, there was less opportunity for the child with autism to take a leadership role in the activities. This dissymmetry of power in the mixed friendship dyads indicates a need for applying intervention in inclusive settings to provide opportunities for leadership building for ASD children and, consequently, expand the possibilities for these children to have more balanced options for relationship building.

Another implication for intervention is elucidated amid the findings that ASD children in mixed friendships have stronger receptive language abilities, suggesting a relationship between language ability and peer competence (Bauminger et al. 2007). This warrants the need for more approaches that target language skill building, yielding more possibilities for educational and social success and consequently friendship building.

Parents and teachers of children with ASD play an integral role in supporting and facilitating their friendships. Thus, the direction for better understanding and contribution to friendship development implores early interventions and teacher and parent training on the development of friendships in preschool children with ASD within school and community settings.

Security attachment may, in a compensatory fashion, contribute positively to the quality of friendships of children with ASD with their typically developing and fellow ASD peers amid social-cognitive difficulties. Thus, it is mandatory in the future for more resources and training opportunities that support the fostering of responsivity

and sensitivity between parents and their children with ASD with the lens of aiding in the development of clear communicative cues. Parents also can be coached to verbally mediate the social world for their HFASD children during structured play, therefore helping them develop schemas for this form of dyadic interaction which consequently provide better opportunities for friendship development and overall friendship satisfaction (Solomon et al. 2004).

Further research is needed to examine the effectiveness of various interventions for children with ASD that help them form successful friendships with their peers. In the future, program facilitators which support friendship development with ASD and typically developing dyads through interactive activities can be more beneficial by actively serving as exemplars for teaching children with ASD how to explore their peer's different perspectives during these allotted times of engagement.

In sum, friendships are indeed obtainable for children with ASD. However, there is more understanding needed about the process of establishing, developing, and consolidating peer relationships with ASD children in order to be able to more effectively provide interventions that can yield increased friendship satisfaction. The socio-emotional encumbrances that are usually experienced with these children call for the support of parents, educators, and clinicians to foster opportunities for responsivity, reciprocity, communication, and empathy. Nonetheless, more playtime with typical developing peers can also possibly serve as useful scaffolding for improving social repertoire which can contribute to more satisfactory friendship states for children with ASD.

See Also

► [Friendship Satisfaction in Children with ASD](#)

References and Reading

- Amati, V., Meggiolaro, S., Rivellini, G., & Zaccarin, S. (2018). Social relations and life satisfaction: The role of friends. *Genus*, 74(1), 7.

- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development, 71*, 447–456.
- Bauminger, N., Solomon, M., Aviezer, A., Heung, K., Brown, J., & Rogers, S. J. (2007). Friendship in high-functioning children with autism spectrum disorder: Mixed and non-mixed dyads. *Journal of Autism and Developmental Disorders, 38*(7), 1211–1229.
- Bauminger, N., Solomon, M., Aviezer, A., Heung, K., Gazit, L., Brown, J., & Rogers, S. J. (2008). Children with autism and their friends: A multidimensional study of friendship in high-functioning autism spectrum disorder. *Journal of Abnormal Child Psychology, 36*(2), 135–150.
- Bauminger, N., Solomon, M., & Rogers, S. J. (2009). Predicting friendship quality in autism spectrum disorders and typical development. *Journal of Autism and Developmental Disorders, 40*(6), 751–761.
- Buhrmester, D. (1990). Intimacy of friendships, interpersonal competence and adjustment during preadolescence and adolescence. *Child Development, 61*, 1101–1111.
- Bukowski, W. M., Hoza, B., & Boivin, M. (1994). Measuring friendship quality during pre-and early adolescence: The development and psychometric properties of the friendship qualities scale. *Journal of Social and Personal Relationships, 11*, 471–484.
- Calder, L., Hill, V., & Pellicano, E. (2012). ‘Sometimes I want to play by myself’: Understanding what friendship means to children with autism in mainstream primary schools. *Autism, 17*(3), 296–316.
- Freeman, S. F., & Kasari, C. (1998). Friendships in children with development disabilities. *Early Education and Development, 9*, 341–355.
- Hodges, E., Boivin, M., Vitaro, F., & Bukowski, W. M. (1999). The power of friendship: Protection against an escalating cycle of peer victimization. *Developmental Psychology, 35*, 94–101.
- Ladd, G. W., Kochenderfer, B. J., & Coleman, C. C. (1996). Friendship quality as a predictor of young children’s early school adjustment. *Child Development, 67*(3), 1103–1118.
- Lieberman, M., Doyle, A. B., & Markiewicz, D. (1999). Developmental patterns in security of attachment to mother and father in late childhood and early adolescence: Associations with peer relations. *Child Development, 70*(1), 202–213.
- Petrina, N., Carter, M., Stephenson, J., & Sweller, N. (2016). Perceived friendship quality of children with autism spectrum disorder as compared to their peers in mixed and non-mixed dyads. *Journal of Autism and Developmental Disorders, 46*(4), 1334–1343.
- Petrina, N., Carter, M., Stephenson, J., & Sweller, N. (2017). Friendship satisfaction in children with autism spectrum disorder and nominated friends. *Journal of Autism and Developmental Disorders, 47*(2), 384–392.
- Schneider, B. H., Atkinson, L., & Tardiff, C. (2001). Child-parent attachment and children’s peer relations:

A quantitative review. *Developmental Psychology, 37*, 68–100.

- Solomon, M., Goodlin-Jones, B. L., & Anders, T. F. (2004). A social adjustment enhancement intervention for high functioning autism, asperger’s syndrome, and pervasive developmental disorder NOS. *Journal of Autism and Developmental Disorders, 34*, 649–668.

Friendships

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Definition

Friendship is a type of social relationship appearing throughout the life span, from early childhood to old age. It is conceptualized under the *social relationships approach* (Hinde 1976), according to which relationships are developed through continuous dyadic interactions over long periods of time with a specific partner (a minimum of 6 months to denote friendship; Howes 1996), to extract a relationship model that goes beyond the influence of each member’s characteristics (Dunn 1993). Through friendship formation, children are “meshed” with each other to create a whole that is greater than the sum of its parts, and they share the properties and histories of their mutual interaction (Dunn 1993). The essence of friendship as a relationship is a mutual liking, whereby children reciprocate an affectionate bond of emotional closeness. Having friends is cardinal to children’s well-being and protects them from depression and loneliness, since friendship provides the child with a sense of belonging and self-worth (e.g., Asher et al. 1996; Vitaro et al. 2009).

The reciprocal-dyadic and stable nature of friendship is a challenge of child development and a necessary component in the formation of fundamental pro-socio-emotional capabilities (e.g., mutual caring, companionship, empathy, mutual regulation) as well as socio-cognitive capacities (e.g., awareness and responsiveness to

another's emotions, desires, intentions, and thoughts; conflict resolution; complex information processing processes; Asher et al. 1996). Affective closeness, which is a marker of friendship, underscores the essence of friendship as an affective bond reflecting the strength of the child's attachment to the friend. In addition to mutual liking, closeness includes the sense of "specialness" to one another, manifested in verbal and nonverbal expressions of affect and caring (Bukowski et al. 1994; Howes 1996). Intimacy (i.e., making one's innermost thoughts and feelings known to a significant other) is another important dimension of friendship (Cassidy 2001). Intimacy includes the belief that friends can be relied upon to provide support and help in times of need. Intimacy is also related to the sense of stability and reciprocity in friendship and to the belief that the friendship is strong enough to overcome negative events such as quarrels (e.g., Bukowski et al. 1994). Markers of intimacy include sharing capabilities and pro-social resources such as providing help (e.g., Bukowski et al. 1994). Friendship should also serve the need for enjoyable companionship. Companionship is usually perceived as a child's ability to spend time with another child and to have fun (e.g., "playing together," "hanging out," "doing things together"; Howes 1996). Markers of companionship include children's cooperative skills during shared work or play, as well as their social conversational skills and positive affect (e.g., shared fun) (Asher et al. 1996).

In sum, friendship is characterized by stable, frequent, and interconnected affective interactions, manifested by certain classes of behavioral markers (e.g., sharing, play, and conversational skills) that facilitate the functions of companionship, intimacy, and closeness. A satisfying friendship is an interpersonal achievement that both develops and builds upon a fundamental capacity for affective relationships and social cognition.

Historical Background

In his first description of the syndrome, Kanner (1943) historically conceptualized autism as a

disorder of affective contact, "these children have come into the world with an innate inability to form the usual, biologically provided affective contact with people" (p. 250). Kanner's description of Elaine (a 7-year-old girl with ASD) supports this conceptualization:

She soon learned the names of all the children (in her school), knew the color of their eyes, the bed in which each slept, and many other details about them, but never entered into any relationship with them. . . . She has no relation to children, has never talked to them, to be friendly with them, or to play with them. She moves among them like a strange being, as one moves between the pieces of furniture of a room. (Kanner 1943, pp. 240–241).

To this day, abnormalities in interpersonal relations are considered a defining characteristic of ASD (DSM-IV-TR, 2000). Friendship is a direct reflection of the child's capacity for interpersonal relationships, and for this reason, both the formation and the understanding of friendship in children with ASD are considered a major theoretical challenge. Contemporary perceptions of the disorder identify difficulties in friendship formation based on these children's inability to experience relationship-based emotions and on their deficit in intersubjective sharing. The results of such impairments are more impersonal and superficial rather than interpersonal friendship relationships; thus, intimacy and affective closeness, according to this view, are unattainable for ASD children (e.g., Hobson 2005). Another view is provided by theorists that highlight ASD children's difficulty in understanding that other people have different thoughts, desires, and feelings than their own (i.e., the theory of mind hypothesis). Theory of mind also predicts crucial difficulties in reciprocity and empathic pro-social behaviors (e.g., comforting, caring, complimenting, listening), which are key defining characteristics of friendship (Tager-Flusberg 2001). These two views, affective and social-cognitive, have led to a general consensus that friendship constitutes a major area of difficulty for children with ASD. In light of these theoretical assumptions, it is not surprising that friendship in ASD has recently been receiving more attention, but it is still an overlooked area of

empirical investigation, despite its significance to the understanding of the social deficit in ASD.

Current Knowledge

Friendship Formation

Studies screening for the prevalence of friendship among individuals on the ASD spectrum have presented a fairly pessimistic picture, with significant percentages found to be outside of this meaningful social experience (e.g., Howlin et al. 2004; Koning and Magill-Evans 2001; Orsmond et al. 2004). For example, half of 69 adults with ASD examined were said to have no friends with whom they share activities (Howlin et al. 2004). In another study, Orsmond et al. (2004) found that of 235 adults and adolescents, 29% had at least one friendship that involved some activities outside prearranged settings and one-quarter (24%) had peer relationships only in prearranged settings. Orsmond et al. also reported a significantly higher number of friends for younger participants (adolescents rather than adults) and for individuals with less impaired social interaction skills. Similarly, in Koning and Magill-Evans' (2001) study, 44% of their adolescent participants with Asperger syndrome had one close friend. Findings based on a slightly different paradigm, examining children and adolescents' social network and social involvement, presented a similar picture, with children on the ASD spectrum more often peripheral in their classroom and forming less reciprocal friendships compared with children of typical development (e.g., Chamberlain et al. 2007; Kasari et al. 2011; Locke et al. 2010; Rotheram-Fuller et al. 2010). The difference is more dramatic in later elementary school and during adolescence (e.g., Locke et al. 2010; Rotheram-Fuller et al. 2010). Recently, studies have begun to specifically examine friendship during preschool with regard to ASD (e.g., Bauminger-Zviely and Agam-Ben-Artzi 2014; Bauminger-Zviely et al. 2014; Chang et al. 2016; Freeman et al. 2015). Information about the number of identified friends for ASD children is provided only by Chang et al. (2016). The number of friends reported by parents (42%,

$n = 13$) and teachers (54%, $n = 17$) exceeded the number of children who were identified as having friends according to observational data (20%, $n = 7$), following an adaptation of Howes' (1983) criteria (e.g., at least 50% of social initiation attempts with responses, at least one unit of joint engagement or games, and one positive affective exchange during the interaction). Nine (29%) sets of parents and teachers identified the same friends; however, parents reported friends from other settings in addition to school, such as family friends ($n = 13$), outside activities ($n = 6$), and the neighborhood ($n = 6$). Further examination of the number of friends for ASD children in preschool is still needed to clarify the gap between parents/teachers and observational reports. Also Bauminger-Zviely and Agam-Ben-Artzi (2014) identified 30 high-functioning preschoolers with ASD who enjoyed at least one reciprocal friendship, based on mother's and teacher's reports and verified by an observation in the child's preschool.

Other studies focusing specifically on friendship in HFASD (high-functioning ASD) have demonstrated the presence of at least one reciprocal friendship in these children (e.g., Bauminger and Kasari 2000; Bauminger and Shulman 2003; Bauminger et al. 2008b; Daniel and Billingsley 2010). Based on these different findings, we may conclude that friendship, though not frequent, is an attainable experience at least for certain subgroups of ASD children, probably for those with higher cognitive and language skills. The two periods with somewhat higher percentages of friendship are middle childhood and early adolescence, with adults showing less frequent experiences of friendship (e.g., Mazurek 2014) and with as-yet unclear numbers regarding young children with ASD.

Very few studies evaluated the characteristics of children who make friends. Chang et al. (2016) have examined spontaneous peer interactions and play compared preschoolers with friends to preschoolers who do not have friends. Children with friends were more likely than children without friends to be jointly engaged with their peers during free play and used higher joint attention skills. In line with these findings, Freeman et al. (2015) examined the influence of joint attention

and play on the quality of friendship among 40 children as measured 5 years later via parent and child reports. The authors found that children who had better joint attention skills at the age of three reported having more closeness and less conflict in their friendships at the age of eight. Children who had better play abilities in general, and specifically greater variability and flexibility in symbolic play, conveyed higher levels of helpfulness in their friendships. These are important results that link core deficits to later friendship development in ASD.

Friendship characteristics. Variables of friendship such as the friend's attributes (age, gender, type of disability), the duration of the friendship, and the frequency of play dates have rarely been explored in children with ASD. Based on the few findings presented in the literature (e.g., Bauminger-Zviely and Agam-Ben-Artzi 2014; Bauminger and Shulman 2003; Bauminger et al. 2008b; Howlin et al. 2004; Kasari et al. 2011; Orsmond et al. 2004; Locke et al. 2010; Petrina et al. 2015), we may conclude with caution that both mixed and nonmixed friendships (i.e., friendship with a child of typical development and friendship with a child with a disability, most likely ASD, respectively) have been found in children with ASD, although the percentages of children who form each type of friendship are not yet clear.

Petrina et al. (2015) assessed school-age friendships with regard to reciprocity and mutuality included 37 boys and 8 girls between the ages of 6.4 and 10.4 years (mean, 8.5; SD, 0.9). Forty-nine percent ($n = 22$) of the ASD group were in a nonmixed friendship dyad, and 51% ($n = 23$) were in a mixed friendship dyad. The level of friendship quality was assessed by the Friendship Quality Questionnaire (FQQ; Parker and Asher 1993), and both friends from each dyad answered the questionnaire using a three-point response scale. According to the answers, the friendships were classified as a best, regular, or nonfriendship. The results showed that 89% of the friendships (i.e., 42 out of 47) across both mixed and nonmixed dyads were reciprocated. The reciprocation rate was 19% for best friends, 15% in the mixed dyads and 24% in the nonmixed, and 55% for

regular friendship, 54% in the mixed dyad and 62% in the nonmixed dyad. The majority of friendships consist of same-age and same-gender couples. Surprisingly, the duration of the friendship was found to be relatively long, ranging from about 1–4 years, but the frequency of play dates outside the school was reported to be about once a week, which is lower than for typically developing children. As mentioned, these somewhat optimistic findings should be further examined, especially with regard to the quality of the relationship. In a meta-analytic study that examined the friendships of school-aged boys with ASD Mendelson et al. (2016) found that boys with ASD do form reciprocal friendships in which they engage in affective sharing, although it is of lower quality. In typical development, friendship functions as an important source of emotional support and as a marker for social adjustment, but the consequences of friendship for children's psychosocial development are highly dependent on its quality (e.g., Vitaro et al. 2009).

Friendship quality and associated behaviors. The quality of the friendship relationship is probably the most debated topic in ASD. Even if researchers agree that some children with ASD do have friends, they will still question the quality of this relationship. More specifically, difficulties in experiencing intimacy and closeness are expected in these children. One source of information on the quality of friendship is children and adolescents' self-reports. Several studies have used Bukowski et al.'s (1994) Friendship Qualities Scale (FQS), which assesses children's reports about their relationship with a close friend in the following dimensions: companionship, intimacy-security, closeness, help, and conflict (e.g., Bauminger and Kasari 2000; Bauminger et al. 2004; Bauminger et al. 2008b; Chamberlain et al. 2007; Kasari et al. 2011; Locke et al. 2010). In these studies, the friendship quality in ASD children was found to be poorer compared with friendship in children of typical development, especially for intimacy, help, and companionship (e.g., Bauminger and Kasari 2000; Bauminger et al. 2004, 2008b; Chamberlain et al. 2007). Differences on the dimension of closeness between the two groups were reported

by Bauminger et al. (2008b) (preadolescence, age range 8.6–12.6) and Kasari et al. (2011) (middle childhood, age range 6–11). Interestingly, the FQS did not show friendships of children with ASD to be more conflictual than those of typically developing children.

In another study, this time examining adults, Baron-Cohen and Wheelwright (2003) provided support for lower friendship quality, revealing less close, empathic, and supportive friendships, in adults with ASD compared with typical controls. Jobe and Williams-White (2007) obtained similar results for adults with severer autism symptoms. Altogether, self-reported friendship quality of HFASD children and adults portrayed difficulties in the formation of quality friendship with peers. This finding has clinical significance: it is interesting that individual with such limited interpersonal awareness and knowledge (e.g., Carrington et al. 2003; Hobson 2005) can reveal a realistic picture of the quality of their friendship, implying that they are aware of their difficulties in interpersonal engagement. In Daniel and Billingsley's (2010) study, 10- to 14-year-old boys described the establishment of friendship as the most difficult aspect in their social life. Higher ratings of loneliness were found in children with HFASD compared with typically developing children (e.g., Bauminger and Kasari 2000; Locke et al. 2010), a finding which may also support the finding of children's awareness of their social difficulties in forming relationships. Such awareness has significant clinical implications, especially in light of accumulating evidence showing high rates of affective disorders (65%; Attwood 2004) such as anxiety, depression, and social isolation, in high-functioning ASD adolescents and adults compared with typical controls (e.g., Attwood 2004). Lack of friends may be one of the reasons for the increase in affective disorders during adolescence and adulthood.

Further support for the notion of lower friendship quality in children with ASD has been provided by observations on children's social interactions with a friend (e.g., Bauminger et al. 2008b) and comparisons between interactions with friends and nonfriends (Bauminger-Zviely and Agam-Ben-Artzi 2014). In Bauminger

et al.'s study, HFASD and typical children's social interactions and their dyadic qualities were coded during an encounter with a friend in two different, noncompetitive play activities: building with blocks and drawing. Results demonstrated significant differences between the two groups in cooperative skills and positive affect on both the building and the drawing activities; differences in sharing were found only in building, and differences in nonverbal behavior were found only while drawing. The play scale, coded only for the building activity, showed a lower frequency of coordinated play and higher frequency of parallel play in the HFASD group than in the typical group. Pairs in which at least one child had HFASD also differed in conversational flow and social conversation. The HFASD group revealed a more rigid conversational style and engaged in less social conversation compared with the typical children. Group differences emerged in all dimensions of the dyadic relationship Q-set that evaluate dyadic quality of interaction between friends (Park and Waters 1989) (positive social orientation, cohesiveness, harmony, responsiveness, coordinated play, and control), except control, with HFASD pairs demonstrating poorer dyadic quality of friendship than typical pairs. A global evaluation of affective closeness and shared fun found that friendships between children with HFASD were lower on these two dimensions compared with typical friendships. It is important to note that children with higher receptive language abilities enjoyed better friendship quality.

Thus, it is safe to conclude that the quality of friendships among children with HFASD is poorer than among typical children. The picture is not complete, however, until a comparison is made between HFASD children's interactions with friends and with nonfriends, for only such a comparison can demonstrate the differential role of a friend to the enhancement of these children's social capabilities. In a recent study (Bauminger-Zviely and Agam-Ben-Artzi 2014), the author and colleagues explored friendship in preschoolers with HFASD. Each child was videotaped interacting with a friend and with a nonfriend while building with blocks, drawing, and during snack time. Results indicated that HFASD

children showed more positive affect and higher degrees of closeness, shared fun, and reciprocity when interacting with a friend compared with a nonfriend. Furthermore, pragmatic skills such as reciprocal conversation, responsiveness to the interlocutor, co-regulation of the conversation, appropriate reference to the other's emotions, appropriate use of facial expressions and eye contact were more intact during interactions with friends versus nonfriends (Bauminger-Zviely et al. 2014), as well as greater conversational adequacy, more initiations of conversational exchanges, and more responsiveness to information evoked by the partner during interaction with friends versus nonfriends dyads (Bauminger-Zviely and Agam-Ben-Artzi 2014). Bauminger-Zviely et al. (2017) showed better use of speech acts-SA (SAs; the primary illocutionary values conventionally conveyed by utterances as acts), during interaction with a friend versus a non-friend. Thus, friendship may serve an important function in the enhancement of social-communicative skills in HFASD.

To complete the discussion on friendship quality, it is important to discuss the partner's contribution to the friendship, namely, whether differences can be found between mixed and non-mixed friendships. In Bauminger et al. (2008a), the author and colleagues examined differences between mixed (HFASD and typical child), nonmixed (HFASD and child with disability, mainly with HFASD), and typical friendships (both children typical). Findings showed that mixed dyads were similar to typical friendship dyads in many of the observational measures collected. More specifically, mixed typical and HFASD dyads differed significantly from non-mixed dyads but not from typical dyads in the amount of engagement in goal-directed activity, sharing, positive affect, and parallel play. Mixed dyads were found to be more responsive and cohesive and to be higher in positive social orientation and to show more complex levels of play (coordinated play), compared with nonmixed dyads. This finding points to the possible benefits to the HFASD child of having a typical child as a friend. Further, according to this study, HFASD children in mixed friendships differed from

HFASD children in nonmixed friendships only in receptive language, demonstrating higher language skills. Other differences were not significant: HFASD children in both friendship types (mixed and nonmixed) showed high social-emotional characteristics including theory of mind (ToM), affective recognition, and attachment to parents.

On the other hand, an important advantage of nonmixed friendship (i.e., friendship between two disabled children) was the element of equality in the exchange: children in such dyads maintained a balance in the degree to which each partner assumed dominant or subordinate roles such as leader and follower, whereas children in mixed friendships had fewer leadership opportunities. Thus, it appears that interactions with a similarly disabled friend (most likely another child with HFASD) are also very important. Based on the studies described, a complete understanding of friendship quality in HFASD requires consideration of the characteristics of the friend, with seeming benefits from participation in both mixed and nonmixed friendships. Further studies should investigate the different benefits of participation in each of the friendship types for the child with HFASD.

Understanding of Friendship

Although interpersonal awareness and understanding are considered to be severely deficient in ASD (Hobson 2005), few studies have focused on ASD children's ability to understand the concept of friendship. One example is Carrington et al. (2003) study, which examined descriptions obtained through semi-structured interviews with five adolescents with Asperger syndrome aged 14–18, of what a friend is, as well as what a friend is not. The adolescents demonstrated difficulties explaining what a friend is: explanations were usually superficial and focused on sharing activities (e.g., computer games such as *Dungeons and Dragons*) and other areas of interest, without mentioning more emotional aspects of friendship, such as liking each other. Bauminger et al. (2004) provided additional support for these children's major difficulties in capturing the more affective dimensions of friendship. In their study, children

(8–17 years) were shown a picture depicting two children in an intimate exchange, looking at each other, smiling, and appearing to be telling each other a secret. Participants were asked to suggest a title and make up a story to go with the picture and to say whether the children in the picture were friends. Only 50% of the HFASD compared to 81.3% of the typical control group named the picture in a way that referred to friendship. Among those who did, only one child with autism referred to the intersubjective sharing and closeness aspect of the friendship (the child suggested the title “soul mates”), compared with many of the typical children (e.g., “best friends,” “secret friends,” “brotherhood”). In Bauminger and Kasari (2000), HFASD children showed difficulty in all three defining aspects of friendship, namely, closeness, companionship, and intimacy. In sum, it is clear that the pragmatic understanding of friendship is lacking in these children, as well as the use of language to describe it. However, it is important to emphasize that lack of understanding may not necessarily preclude the experience of friendship. These children may have specific difficulties in reflecting about their interpersonal experience.

Future Directions

There is little doubt that ASD is a disorder involving severe deficits both in experiencing and understanding friendship. However, friendship is not an all-or-nothing concept, and for these children, some of whom do have friends and whose friendships are of great importance to them, even more limited forms of friendship can be fairly durable. Although consistent difficulties have been demonstrated across studies examining the understanding and experience of friendship in ASD, a subgroup of ASD children has nevertheless emerged, who show higher interpersonal awareness and a better quality of friendship. The phenomenon of friendship in children with ASD would probably best be described as a continuum of interpersonal resources, with some children able to develop relationships or even meaningful and enduring friendships, others capable of more superficial attachments in which the friend is

merely a playmate rather than an intimate “soul mate,” and still others being unable to develop any type of stable relationship with a friend or even having not yet developed the desire to have a peer as a friend. This continuum should be further explored in order to identify what characterizes each of these subgroups, and such exploration is indeed under way in the field of typical friendship research, which is beginning to examine various types and functions of friendships (see, e.g., Kerns 2000, for preschool friendship, and Shulman 1993, for adolescents).

Just as the child with ASD clearly plays a significant role in determining the quality of the friendship, so does the friend, with typically developing friends probably helping to promote more complex interactions, and friends with a disability contributing more to aspects of equality in the friendship. Language skills have also been found important, with children of higher language skill appearing to develop higher quality friendships. In this respect, it is important to note that children with ASD in both mixed and nonmixed friendships reveal high social-emotional abilities such as ToM, security of attachment, and affective recognition. Perhaps, within the spectrum of ASD, these children with higher social, cognitive, and emotional resources do develop friendship. What helps children develop a friendship with a typical peer, beyond better language skills, has yet to be explored.

Another reason why friendship is an uncommon experience in ASD may be because social interventions tend to neglect focusing on friendship. Theoretical assumptions of friendship as a nontangible experience for children within the spectrum may limit professionals’ attempts to discover ways to support, encourage, and build up ASD children’s ability to form friendships when planning social interventions. Only rarely is friendship a target of such interventions for this population, and very few social intervention programs (specifically at younger ages) are aimed at developing and maintaining friendships (e.g., Frankel and Myat 2003). In adolescence, for example, children with ASD consistently perceive themselves as lonelier than typically developing children (e.g., Bauminger and Kasari 2000). For these reasons, friendship should be considered an aim of ASD social intervention across the

lifespan, and future studies need to further explore the necessary skills leading to the formation of friendship in ASD. Friendship in young children particularly merits thorough investigation due to its preventive value. Another important direction for research is the identification of early markers of higher interpersonal resources and capacities for richer friendships in children within the ASD spectrum. Also, since friendship has not been carefully explored in children with lower cognitive and language skills and little is known about these children's ability to form friendship, further research is warranted. Companionship skills seem to be easier to teach, but the real struggle is helping these children share their innermost feelings and thoughts and develop intimacy and affective closeness with a friend. To conclude, we are presently at the most initial stages of untangling the intricacies of interpersonal engagement with friends in ASD. Longitudinal studies exploring the phenomenon are needed, alongside the planning of interventional goals to enhance friendship in children with ASD.

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders-text revision* (4th ed.). Washington, DC: American Psychiatric Association.
- Asher, S. R., Parker, J. G., & Walker, D. L. (1996). Distinguishing friendship from acceptance: Implications for intervention and assessment. In W. M. Bukowski, A. F. Newcomb, & W. W. Hartup (Eds.), *The company they keep: Friendships in childhood and adolescence* (pp. 366–406). Cambridge: Cambridge University Press.
- Attwood, T. (2004). Cognitive behaviour therapy for children and adults with Asperger's syndrome. *Behaviour Change*, 21, 147–161.
- Baron-Cohen, S., & Wheelwright, S. (2003). The friendship questionnaire: An investigation of adults with Asperger syndrome or high-functioning autism, and normal sex differences. *Journal of Autism and Developmental Disorders*, 33, 509–517.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development*, 71, 447–456.
- Bauminger, N., & Shulman, C. (2003). The development and maintenance of friendship in high-functioning children with autism: Maternal perception. *Autism, the International Journal of Research and Practice*, 7, 81–97.
- Bauminger, N., Shulman, C., & Agam, G. (2004). The link between the perception of self and of social relationships in high-functioning children with autism. *Journal of Development and Physical Disabilities*, 16, 193–214.
- Bauminger, N., Solomon, M., Aviezer, A., Heung, K., Brown, J., & Rogers, S. (2008a). Friendship in high-functioning children with ASD: Mixed and non-mixed dyads. *Journal of Autism and Developmental Disorders*, 38, 1121–1229.
- Bauminger, N., Solomon, M., Aviezer, A., Heung, K., Gazit, L., Brown, J., & Rogers, S. (2008b). Friendship manifestations, dyadic qualities of friendship, and friendship perception in high-functioning preadolescents with autism spectrum disorder. *Journal of Abnormal Child Psychology*, 36, 135–150.
- Bauminger-Zviely, N., & Agam-Ben-Artzi, G. (2014). Young friendship in HFASD and typical development: Friend versus non-friend comparisons. *Journal of Autism and Developmental Disorders*, 44, 1733–1748.
- Bauminger-Zviely, N., Karin, E., Kimhi, Y., & Agam Ben Artzi, G. (2014). Spontaneous peer conversation in preschoolers with high-functioning autism spectrum disorder versus typical development. *Journal of Child Psychology and Psychiatry*, 55, 363–373.
- Bauminger-Zviely, N., Golan-Itzhaky, A., & Tubul-Lavy, G. (2017). Speech acts during spontaneous peer conversation in preschoolers with high-functioning autism spectrum disorder versus typical development. *Journal of Autism and Developmental Disorders*, 47, 1380–1390.
- Bukowski, W. M., Boivin, M., & Hoza, B. (1994). Measuring friendship quality during pre- and early adolescence: The development and psychometric properties of the friendship qualities scale. *Journal of Social and Personal Relationships*, 11, 471–484.
- Carrington, S., Templeton, E., & Papinczak, T. (2003). Adolescents with Asperger syndrome and perceptions of friendship. *Focus on Autism and Other Developmental Disabilities*, 18, 211–218.
- Cassidy, J. (2001). Truth, lies, and intimacy: An attachment perspective. *Attachment & Human Development*, 3, 121–155.
- Chamberlain, B., Kasari, C., & Rotheram-Fuller, E. (2007). Involvement or isolation? The social network of children with autism in regular classrooms. *Journal of Autism and Developmental Disorders*, 37, 230–242.
- Chang, Y. C., Shih, W., & Kasari, C. (2016). Friendships in preschool children with autism spectrum disorder: What holds them back, child characteristics or teacher behavior? *Autism: The International Journal of Research and Practice*, 20(1), 65–74.
- Church, C., Alinsanski, S., & Amanullah, S. (2000). The social, behavioural, and academic experiences of children with Asperger syndrome. *Focus on Autism and Other Developmental Disabilities*, 15, 12–20.
- Daniel, L. S., & Billingsley, B. S. (2010). What boys with an Autism spectrum disorder say about establishing and

- maintaining friendships. *Focus on Autism and Other Developmental Disabilities*, 25, 220–229.
- Dunn, J. (1993). *Young children's close relationships: Beyond attachment*. London: SAGE.
- Frankel, F., & Myat, R. (2003). *Children's friendship training*. New York: Taylor & Francis Books.
- Freeman, S. F., Gulsrud, A., & Kasari, C. (2015). Brief report: Linking early joint attention and play abilities to later reports of friendships for children with ASD. *Journal of Autism and Developmental Disorders*, 45(7), 2259–2266.
- Hinde, R. A. (1976). On describing relationships. *Journal of Child Psychology and Psychiatry*, 17, 1–19.
- Hobson, P. (2005). Autism and emotion. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 406–422). Hoboken: Wiley.
- Howes, C. (1983). Patterns of friendship. *Child Development*, 54, 1041–1053.
- Howes, C. (1996). The earliest friendships. In W. M. Bukowski, A. F. Newcomb, & W. W. Hartup (Eds.), *The company they keep: Friendship in childhood and adolescence* (pp. 66–86). Cambridge, UK: Cambridge University Press.
- Howlin, P., Good, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45, 212–229.
- Jobe, L. E., & Williams-White, S. (2007). Loneliness, social relationships, and a broader autism phenotype in college students. *Personality and Individual Differences*, 42, 1479–1489.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kasari, C., Locke, J., Gulsrud, A., & Rotheram-Fuller, E. (2011). Social networks and friendships at school: Comparing children with and without ASD. *Journal of Autism and Developmental Disorders*, 41(5), 533–544.
- Kerns, K. A. (2000). Types of preschool friendships. *Personal Relationships*, 7, 311–324.
- Koning, C., & Magill-Evans, J. (2001). Social and language skills in adolescent boys with Asperger syndrome. *Autism*, 5, 23–36.
- Locke, J., Ishijima, E., Kasari, C., & London, N. (2010). Loneliness, friendship quality and the social networks of adolescents with high-functioning autism in an inclusive school setting. *Journal of Research in Special Educational Needs*, 10, 74–81.
- Mazurek, M. O. (2014). Loneliness, friendship and well-being in adults with autism spectrum disorders. *Autism*, 18(3), 223–232.
- Mendelson, J. L., Gates, J. A., & Lerner, M. D. (2016). Friendship in school-age boys with autism spectrum disorders: A meta-analytic summary and developmental, process-based model. *Psychological Bulletin*, 142(6), 601–622.
- Ormond, G., Krauss, M., & Seltzer, M. (2004). Peer relationships and social and recreational activities among adolescents and adults with autism. *Journal of Autism and Developmental Disabilities*, 34, 245–256.
- Park, K. A., & Waters, E. (1989). Security of attachment and preschool friendships. *Child Development*, 60, 1076–1081.
- Parker, J. G., & Asher, S. R. (1993). Friendship and friendship quality in middle childhood: Links with peer group acceptance and feelings of loneliness and social dissatisfaction. *Developmental Psychology*, 29, 611–621.
- Petrina, N., Carter, M., & Stephenson, J. (2015). Parental perception of the importance of friendship and other outcome priorities in children with autism spectrum disorder. *European Journal of Special Needs Education*, 30(1), 61–74.
- Rotheram-Fuller, E., Kasari, C., Chamberlain, B., & Locke, J. (2010). Social involvement of children with autism spectrum disorders in elementary school classrooms. *Journal of Child Psychology and Psychiatry*, 51, 1227–1234.
- Shulman, S. (1993). Close friendships in early and middle adolescence: Typology and friendship reasoning. In B. Laursen (Ed.), *Close friendships in adolescence* (pp. 55–71). San Francisco: Jossey-Bass.
- Tager-Flusberg, H. (2001). A reexamination of the theory of mind hypothesis of autism. In J. Burack, T. Charman, N. Yirmiya, & P. Zelazo (Eds.), *Development and autism: Perspectives from theory and research* (pp. 173–193). Hillsdale: Erlbaum Press.
- Vitaro, F., Boivin, M., & Bukowski, W. M. (2009). The role of friendship in child and adolescents psychosocial development. In K. H. Rubin, W. M. Bukowski, & B. Laursen (Eds.), *Handbook of peer interactions, relationships, and groups*. New York: Guilford Press.

Froidir

► Clozapine

Frontal Lobe Findings in Autism

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Synonyms

Emotion and decision-making; Executive functions; Frontal lobe functions; Mirror mechanisms; Social cognition

Structure

Frontal Lobes: Anatomy and Function

The frontal lobes constitute the largest part of the human brain. Anatomically, they represent those areas of the cortex anterior to the central sulcus. They are the last to mature during development, and only reach full maturity by the mid-20s.

Function

The frontal cortex can roughly be divided into the following parts with corresponding functions:

- *Frontal operculum*: language, mirror mechanisms
- *Precentral*: motor
- *Dorsolateral prefrontal*: executive functions
- *Orbitofrontal/ventromedial*: emotion, decision-making, response inhibition
- *Anterior cingulate*: choice selection, reward, social emotion, empathy, response monitoring
- *Medial prefrontal*: social cognition

Lesions of the frontal lobes are characterized by profound personality changes, described in the famous case of Phineas Gage (Damasio 1994). Other elements of frontal dysfunction may include perseveration, aphasia, lack of spontaneous activity, indifference, or disinhibition. Some of the effects resulting from frontal lesions are due to the fact that the orbitofrontal cortex is extensively connected to the amygdala and subcortical structures and can be considered as one of the elements of the limbic system.

In order to tap into their different functions, including language production, motor, and executive functions, neuropsychological testing of frontal lobes is performed with a variety of tasks. A vast number of tests are available to test language production, including the Boston Naming Test. Neurological examinations are used to test for motor functions. Executive functions (EF) include skills required for action planning and execution, inhibition, organization, self-monitoring, cognitive flexibility, and set-shifting. EF tests include Go/No-Go task, Trail Making

Test (TMT), Lexical Fluency, attention and concentration tests (e.g., serial 7), digit span, alternative sequence task, Tower of Hanoi (or of London), and Wisconsin Card Sorting Test (WCST). In addition, a computerized battery for testing EF is available: the Cambridge Neuropsychological Test Automated Battery (CANTAB) (Robbins et al. 1994).

Below, we describe the findings of neuropsychological, anatomical, and functional studies in ASD.

Pathophysiology

Neuropsychological Studies

Executive functions: Abnormalities in executive functions are very common in autism, although their relationship with autistic symptomatology is complex (Joseph and Tager-Flusberg 2004; Kenworthy et al. 2008). In their review, Kenworthy et al. (2008) note that while there is a consensus about difficulties in various aspects of executive functions in daily life of individuals with autism, a number of questions remain in terms of their performance during neuropsychological testing. In particular, this review underlines the fact that while abnormal performance has been observed in tasks that required human administration and hence adherence to socially presented rules and interactions (such as the WCST or the Tower tasks), less impairments are reported in ASD when tests are administered using CANTAB, a computerized battery. A more recent review (Demetriou et al. 2019) suggests that the heterogeneity of EF difficulties needs to be quantified and that a RDoC approach may be needed to better understand potential ASD subtypes based on EF functions.

Other confounding factors in EF studies in ASD are IQ levels and age, with differential maturation trajectories of components of EF during adolescence. A robust finding, however, is the presence of spatial working-memory deficits. Ecologically valid EF control questionnaires (e.g., BRIEF) reveal that both children and adults with ASD have difficulties on unstructured tasks, but the potential confound with social demands on these measures still need to be better understood.

Mirror mechanisms: Mirror mechanisms were first described in primates and rapidly thereafter in humans by the group of Giacomo Rizzolatti (Gallese et al. 1996; Rizzolatti et al. 1996). The caudal part of the inferior frontal gyrus (corresponding to Broca's area on the left hemisphere) is part of the action mirror network. Mirror mechanisms translate sensory information during observation of an action done by others into that same action coded in the observer's motor system. The result of this sensory-motor transformation depends on the location of the mirror neurons: for neurons in the motor system, mirror mechanisms will make possible the understanding of action and imitation; for neurons located in the insula and the cingulate, mirror mechanisms will support emotional understanding and empathy. Language is very likely to have evolved from mirror mechanisms (Rizzolatti and Arbib 1998).

The results of studies addressing imitation deficits in autism are sometimes contradictory (for review, see Seveler and Gillis (2010)), probably partly due to the fact that the term "imitation" covers several categories of behaviors, including emulation, mimicry, and true imitation, and that imitation tasks used in different studies are extremely variable.

Imitation performance in ASD depends on task type (Rogers et al. 2003). Children with autism seem to be capable of elicited imitation, but do not show a tendency to spontaneously imitate (Ingersoll 2008; Whiten and Brown 1999; but see Bird et al. 2007). One common finding in ASD is the lack of automatic mimicry and spontaneous imitation of facial expressions (reviewed in Seveler and Gillis (2010)). The EP-M model (emulation and planning-mimicry; Hamilton 2008) suggests that while the EP route is intact, the M route, responsible for automatic mimicry and spontaneous imitation, may be impaired in children with ASD.

Training of motor imitation can reduce other symptoms (e.g., improve language) in ASD (Ingersoll and Lalonde 2010), and neurofeedback training during imitation and observation seem to result in behavioral improvement in ASD (Datko et al. 2018).

Joint attention (JA), theory-of-mind (ToM): Deficits in joint attention are one of the first symptoms to appear autism (for review, see Mundy (2003)). Capacity for joint attention normally emerges at 6 months of age and is a prerequisite for social learning. ToM is the capacity to attribute mental states to oneself and others, and deficits in ToM have been a prevalent theory in autism (Baron-Cohen et al. 1985). However, a fair amount of empirical evidence seems to fail supporting the claim of an impairment of ToM in autism, as recently reviewed in Gernsbacher and Yergeau (2019), who show that tasks used in the past to evaluate ToM failed to account for autistic traits, social interaction, and empathy. In particular, it appears that many tasks rely on language comprehension (e.g., False Belief task, Strange Stories task, Reading-the-Mind-in-the-Eye task); therefore, it is not a surprise that autistic individuals with communication impairments would perform less well in these tasks.

Anatomical Studies

Brain Volume and Cortical Thickness

Anatomical differences in ASD brain are reviewed in Amaral, Schumann, and Nordahl (Amaral et al. 2008). A common theory posits that ASD brains undergo a period of precocious growth during early postnatal life, followed by a deceleration (for review, see Courchesne et al. (2004)) related to a disproportionate increase in white matter (Herbert et al. 2003). Greatest increases in volume have been observed in the dorsolateral prefrontal and medial frontal cortices (Carper et al. 2002; Herbert et al. 2004), whereas no differences have been reported in the orbitofrontal cortex. A longitudinal study reports that by age 2.5, both cerebral gray and white matter are significantly enlarged in toddlers with ASD, with the most severe enlargement occurring in frontal, temporal, and cingulate cortices (Schumann et al. 2010). Abnormalities in gyrification of the inferior frontal gyrus, as well as cortical thickness decreases in the inferior frontal cortex, have been described (Ecker et al. 2010; Hadjikhani et al. 2006). In the dorsolateral

prefrontal cortex, Casanova et al. have observed abnormal minicolumns (Casanova et al. 2002). A more recent microstructural study has revealed focal patches of abnormal laminar cytoarchitecture and cortical disorganization in the prefrontal cortex of children with autism (Stoner et al. 2014).

Diffusion Tensor Imaging (DTI) Studies

Multiple studies have described white matter alteration in the frontal lobes in ASD. Most DTI studies report reduced fractional anisotropy (FA) in frontal white matter in adolescents and adults with ASD (Alexander et al. 2007; Barnea-Goraly et al. 2004; Keller et al. 2007; Thakkar et al. 2008) as well as in children (Kumar et al. 2010; Sundaram et al. 2008).

Opposite findings (increased FA in the frontal lobe) are reported in children, suggesting the role of a developmental component, with an early and accelerated abnormal maturation of the white matter (Ben Bashat et al. 2007). Increased myelination of the frontal lobe was also described in a study using T2-weighted imaging in young children with ASD (Carmody and Lewis 2010).

Because ASD most probably is the consequence of several possible pre-, peri- and postnatal pathogenic events that affect neural proliferation and migration (see above) as well as myelination, pruning, axonal development, and connectivity, it is to be expected that differences will be present in the white matter. A recent review nicely illustrates all the findings related to structural neuroimaging in ASD (Girault and Piven 2020).

Functional Imaging

Activation Studies

Activation studies have addressed different functions of the frontal lobes.

Language: Semantic processing is associated with stronger activation in Broca's area in adolescents with ASD, who also exhibit less laterality for this area (in the context of similar behavioral performance) (Knaus et al. 2008). In adults, other studies have shown reduced activation in Broca's

area during semantic processing; however, behavioral performance was not equal between groups, making the interpretation of the data difficult. A recent meta-analysis of 22 published studies (Herringshaw et al. 2016) reported increased left middle frontal and right inferior frontal gyrus activity in ASD during language processing, in particular in tasks with poorer performance accuracy for the right IFG.

Motor functions: Very few functional studies have examined the imaging correlates of motor dysfunctions in autism. Mostofsky et al. (2009) describe greater activation of the supplementary motor area (SMA) during finger tapping task in children with autism, compared to controls who tend to engage the cerebellum for this type of task. In addition, a recent study has demonstrated atypical lateralization in circuits subserving motor functions (Floris et al. 2016).

Executive functions: Four main caveats need to be underlined in functional imaging of EF in ASD. The first critical issue is the necessity to compare brain activation in tasks where behavioral performance is similar between the groups being compared. A second issue is that the fact that age may play a role in the synchronization of frontal and fronto-striatal brain regions involved in response inhibition and that in consequence age groups need to be strictly defined and homogeneous. A fourth issue is the very common comorbidity of ADHD and ASD, which would be de facto associated with EF difficulties. Finally, one has to bear in mind that an abnormal developmental trajectory may also be the basis of the differences observed in children and adolescents with ASD.

The majority of functional magnetic resonance imaging (fMRI) studies of EF in ASD have demonstrated diminished brain activation compared to controls, with some exceptions (reviewed in Solomon et al. (2009)).

Mirror mechanisms: Several functional studies using different imaging techniques including fMRI, electroencephalography (EEG), and magnetoencephalography (MEG) have reported abnormalities in mirror mechanisms in ASD. In neurotypicals, action observation is typically associated with a suppression of the mu rhythm in the EEG signal – a phenomenon that seems

absent in ASD (Oberman et al. 2008). Using transcranial magnetic stimulation (TMS), Theoret et al. (2005) have demonstrated that TMS applied over the primary motor cortex did not modulate the excitability of the primary motor cortex in children with ASD, contrary to controls.

fMRI studies in children and in adults have reported weaker activation of the frontal operculum during face or body emotion expression perception (Dapretto et al. 2006; Hadjikhani et al. 2007; Hadjikhani et al. 2009).

However, recent studies report normal (Dinstein et al. 2010) or even enhanced (Martineau et al. 2010) activation of mirror system areas during action observation, or during perception of facial expression of pain (Hadjikhani et al. 2014), leaving the question of atypical activity of the MNS in autism open.

Reward processing: One of the current hypotheses about social impairments in autism is a lack of social motivation, due to a decreased reward value for social stimuli. A recent systematic review and meta-analysis (Clements et al. 2018), including 30 studies, points to hypoactivation of the anterior cingulate in ASD during social rewards, nonsocial rewards, and restricted interests.

Social emotion, joint attention: Normal activation has been reported in middle prefrontal cortex during social-emotional tasks (Schulte-Ruther et al. 2010). However, hypoactivation has been observed during tasks of joint attention (Zürcher et al. 2013; Oberwelland et al. 2017).

Response monitoring: Abnormal (increased) anterior cingulate activation has been shown in response to correct trials during response monitoring in ASD (Thakkar et al. 2008).

In summary, functional activation studies remain controversial, and a better understanding of frontal functions in autism will require further investigation.

Functional Connectivity Studies

A common finding of functional connectivity studies has been the presence of underconnectivity in ASD between large-scale networks and over-connectivity in small-scale

networks. In most studies specifically addressing connectivity of the motor system (Mostofsky et al. 2009) and of the EF system (Just et al. 2004; Kana et al. 2007; Koshino et al. 2005; Solomon et al. 2009; Thakkar et al. 2008), underconnectivity was reported. However, other groups show no differences (Lee et al. 2009) or even increased (Shih et al. 2010) connectivity in the frontal regions in ASD (superior frontal and anterior cingulate) – concluding to atypical connectivity of the imitation network with an enhanced role of the dorsal prefrontal cortex. The different findings of resting-state functional connectivity in autism have recently been reviewed by Hull et al. (Hull et al. 2016).

One major caveat needs to be kept in mind for functional connectivity studies: we know that that slightly different motion magnitudes may induce spurious differences between groups, and this factor needs to be specifically addressed in studies (Van Dijk et al. 2012).

Perspectives

Because of the developmental trajectory of executive functions and of the frontal lobes in general, future studies will need to have larger number of participants, with homogeneous age groups and cognitive abilities (IQ). In addition, functional studies will need to be designed so that behavioral performance is equal between groups, if brain activations need to be compared for a specific task.

See Also

- [Executive Function \(EF\)](#)
- [Mirror Neuron System](#)

References and Reading

- Alexander, A. L., Lee, J. E., Lazar, M., Boudos, R., DuBray, M. B., Oakes, T. R., et al. (2007). Diffusion tensor imaging of the corpus callosum in autism. *NeuroImage*, 34(1), 61–73.

- Amaral, D. G., Schumann, C. M., & Nordahl, C. W. (2008). Neuroanatomy of autism. *Trends in Neurosciences*, 31(3), 137–145.
- Barnea-Goraly, N., Kwon, H., Menon, V., Eliez, S., Lotspeich, L., & Reiss, A. L. (2004). White matter structure in autism: Preliminary evidence from diffusion tensor imaging. *Biological Psychiatry*, 55(3), 323–326.
- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a “theory of mind”? *Cognition*, 21(1), 37–46.
- Ben Bashat, D., Kronfeld-Duenias, V., Zachor, D. A., Ekstein, P. M., Hendler, T., Tarrasch, R., et al. (2007). Accelerated maturation of white matter in young children with autism: A high b value DWI study. *NeuroImage*, 37(1), 40–47.
- Bird, G., Leighton, J., Press, C., & Heyes, C. (2007). Intact automatic imitation of human and robot actions in autism spectrum disorders. *Proceedings of the Biological Sciences*, 274(1628), 3027–3031.
- Carmody, D. P., & Lewis, M. (2010). Regional white matter development in children with autism spectrum disorders. *Developmental Psychobiology*, 52(8), 755–763.
- Carper, R. A., Moses, P., Tigue, Z. D., & Courchesne, E. (2002). Cerebral lobes in autism: Early hyperplasia and abnormal age effects. *NeuroImage*, 16(4), 1038–1051.
- Casanova, M. F., Buxhoeveden, D. P., Switala, A. E., & Roy, E. (2002). Minicolumnar pathology in autism. *Neurology*, 58(3), 428–432.
- Clements, C. C., Zolowski, A. R., Yankowitz, L. D., Yerys, B. E., Schultz, R. T., & Herrington, J. D. (2018). Evaluation of the social motivation hypothesis of autism: A systematic review and meta-analysis. *JAMA Psychiatry*, 75(8), 797–808.
- Courchesne, E., Redcay, E., & Kennedy, D. P. (2004). The autistic brain: Birth through adulthood. *Current Opinion in Neurology*, 17(4), 489–496.
- Damasio, A. R. (1994). *Descartes' error*. New York: G.P. Putnam's Sons.
- Dapretto, M., Davies, M. S., Pfeifer, J. H., Scott, A. A., Sigman, M., Bookheimer, S. Y., et al. (2006). Understanding emotions in others: Mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neuroscience*, 9(1), 28–30.
- Datko, M., Pineda, J. A., & Muller, R. A. (2018). Positive effects of neurofeedback on autism symptoms correlate with brain activation during imitation and observation. *The European Journal of Neuroscience*, 47(6), 579–591.
- Demetriou, E. A., DeMayo, M. M., & Guastella, A. J. (2019). Executive function in autism spectrum disorder: History, theoretical models, empirical findings, and potential as an endophenotype. *Frontiers in Psychiatry*, 10, 753.
- Dichter, G. S., Felder, J. N., Green, S. R., Rittenberg, A. M., Sasson, N. J., & Bodfish, J. W. (2012). Reward circuitry function in autism spectrum disorders. *Social Cognitive and Affective Neuroscience*, 7(2), 160–172.
- Dinstein, I., Thomas, C., Humphreys, K., Minshew, N., Behrmann, M., & Heeger, D. J. (2010). Normal movement selectivity in autism. *Neuron*, 66(3), 461–469.
- Ecker, C., Marquand, A., Mourao-Miranda, J., Johnston, P., Daly, E. M., Brammer, M. J., et al. (2010). Describing the brain in autism in five dimensions-magnetic resonance imaging-assisted diagnosis of autism spectrum disorder using a multiparameter classification approach. *Journal of Neuroscience*, 30(32), 10612–10623.
- Floris, D. L., Barber, A. D., Nebel, M. B., Martinelli, M., Lai, M. C., Crocetti, D., et al. (2016). Atypical lateralization of motor circuit functional connectivity in children with autism is associated with motor deficits. *Molecular Autism*, 7, 35.
- Gallese, V., Fadiga, L., Fogassi, L., & Rizzolatti, G. (1996). Action recognition in the premotor cortex. *Brain*, 119 (Pt 2), 593–609.
- Gernsbacher, M. A., & Yergeau, M. (2019). Empirical failures of the claim that autistic people lack a theory of mind. *Archives of Scientific Psychology*, 7(1), 102–118.
- Girault, J. B., & Piven, J. (2020). The neurodevelopment of autism from infancy through toddlerhood. *Neuroimaging Clinics of North America*, 30(1), 97–114.
- Hadjikhani, N., Joseph, R. M., Snyder, J., & Tager-Flusberg, H. (2006). Anatomical differences in the mirror neuron system and social cognition network in autism. *Cerebral Cortex*, 16(9), 1276–1282.
- Hadjikhani, N., Joseph, R. M., Snyder, J., & Tager-Flusberg, H. (2007). Abnormal activation of the social brain during face perception in autism. *Human Brain Mapping*, 28(5), 441–449.
- Hadjikhani, N., Joseph, R. M., Manoach, D., Naik, P., Snyder, J., Dominick, K., et al. (2009). Body expressions of emotion do not trigger fear contagion in autism spectrum disorder. *Social Cognitive and Affective Neuroscience*, 4(1), 70–78.
- Hadjikhani, N., Zurcher, N. R., Rogier, O., Hippolyte, L., Lemonnier, E., Ruest, T., et al. (2014). Emotional contagion for pain is intact in autism spectrum disorders. *Translational Psychiatry*, 4, e343.
- Hamilton, A. F. (2008). Emulation and mimicry for social interaction: A theoretical approach to imitation in autism. *Quarterly Journal of Experimental Psychology (Colchester)*, 61(1), 101–115.
- Herbert, M. R., Ziegler, D. A., Deutsch, C. K., O'Brien, L. M., Lange, N., Bakardjiev, A., et al. (2003). Dissociations of cerebral cortex, subcortical and cerebral white matter volumes in autistic boys. *Brain*, 126 (Pt 5), 1182–1192.
- Herbert, M. R., Ziegler, D. A., Makris, N., Filipek, P. A., Kemper, T. L., Normandin, J. J., et al. (2004). Localization of white matter volume increase in autism and developmental language disorder. *Annals of Neurology*, 55(4), 530–540.
- Herringshaw, A. J., Ammons, C. J., DeRamus, T. P., & Kana, R. K. (2016). Hemispheric differences in language processing in autism spectrum disorders:

- A meta-analysis of neuroimaging studies. *Autism Research*, 9(10), 1046–1057.
- Hull, J. V., Dokovna, L. B., Jacokes, Z. J., Torgerson, C. M., Irimia, A., & Van Horn, J. D. (2016). Resting-state functional connectivity in autism spectrum disorders: A review. *Frontiers in Psychiatry*, 7, 205.
- Ingersoll, B. (2008). The effect of context on imitation skills in children with autism. *Research in Autism Spectrum Disorders*, 2, 332–340.
- Ingersoll, B., & Lalonde, K. (2010). The impact of object and gesture imitation training on language use in children with autism spectrum disorder. *Journal of Speech, Language, and Hearing Research*, 53(4), 1040–1051.
- Joseph, R. M., & Tager-Flusberg, H. (2004). The relationship of theory of mind and executive functions to symptom type and severity in children with autism. *Development and Psychopathology*, 16(1), 137–155.
- Just, M. A., Cherkassky, V. L., Keller, T. A., & Minshew, N. J. (2004). Cortical activation and synchronization during sentence comprehension in high-functioning autism: Evidence of underconnectivity. *Brain*, 127 (Pt 8), 1811–1821.
- Kana, R. K., Keller, T. A., Minshew, N. J., & Just, M. A. (2007). Inhibitory control in high-functioning autism: Decreased activation and underconnectivity in inhibition networks. *Biological Psychiatry*, 62(3), 198–206.
- Keller, T. A., Kana, R. K., & Just, M. A. (2007). A developmental study of the structural integrity of white matter in autism. *Neuroreport*, 18(1), 23–27.
- Kenworthy, L., Yerys, B. E., Anthony, L. G., & Wallace, G. L. (2008). Understanding executive control in autism spectrum disorders in the lab and in the real world. *Neuropsychology Review*, 18(4), 320–338.
- Knaus, T. A., Silver, A. M., Lindgren, K. A., Hadjikhani, N., & Tager-Flusberg, H. (2008). fMRI activation during a language task in adolescents with ASD. *Journal of International Neuropsychological Society*, 14(6), 967–979.
- Koshino, H., Carpenter, P. A., Minshew, N. J., Cherkassky, V. L., Keller, T. A., & Just, M. A. (2005). Functional connectivity in an fMRI working memory task in high-functioning autism. *NeuroImage*, 24(3), 810–821.
- Kumar, A., Sundaram, S. K., Sivaswamy, L., Behen, M. E., Makki, M. I., Ager, J., et al. (2010). Alterations in frontal lobe tracts and corpus callosum in young children with autism spectrum disorder. *Cerebral Cortex*, 20(9), 2103–2113.
- Lee, P. S., Yerys, B. E., Della Rosa, A., Foss-Feig, J., Barnes, K. A., James, J. D., et al. (2009). Functional connectivity of the inferior frontal cortex changes with age in children with autism spectrum disorders: A fcMRI study of response inhibition. *Cerebral Cortex*, 19(8), 1787–1794.
- Martineau, J., Andersson, F., Barthelemy, C., Cottier, J. P., & Destrieux, C. (2010). Atypical activation of the mirror neuron system during perception of hand motion in autism. *Brain Research*, 1320, 168–175.
- Mostofsky, S. H., Powell, S. K., Simmonds, D. J., Goldberg, M. C., Caffo, B., & Pekar, J. J. (2009). Decreased connectivity and cerebellar activity in autism during motor task performance. *Brain*, 132(Pt 9), 2413–2425.
- Mundy, P. (2003). Annotation: The neural basis of social impairments in autism: The role of the dorsal medial-frontal cortex and anterior cingulate system. *Journal of Child Psychology and Psychiatry*, 44(6), 793–809.
- Oberman, L. M., Ramachandran, V. S., & Pineda, J. A. (2008). Modulation of mu suppression in children with autism spectrum disorders in response to familiar or unfamiliar stimuli: The mirror neuron hypothesis. *Neuropsychologia*, 46(5), 1558–1565.
- Oberwelland, E., Schilbach, L., Barisic, I., Krall, S. C., Vogeley, K., Fink, G. R., et al. (2017). Young adolescents with autism show abnormal joint attention network: A gaze contingent fMRI study. *NeuroImage: Clinical*, 14, 112–121.
- Rizzolatti, G., & Arbib, M. A. (1998). Language within our grasp. *Trends in Neurosciences*, 21(5), 188–194.
- Rizzolatti, G., Fadiga, L., Matelli, M., Bettinardi, V., Paulesu, E., Perani, D., et al. (1996). Localization of grasp representations in humans by PET: 1. Observation versus execution. *Experimental Brain Research*, 111(2), 246–252.
- Robbins, T. W., James, M., Owen, A. M., Sahakian, B. J., McInnes, L., & Rabbitt, P. (1994). Cambridge Neuropsychological Test Automated Battery (CANTAB): A factor analytic study of a large sample of normal elderly volunteers. *Dementia*, 5(5), 266–281.
- Rogers, S. J., Hepburn, S. L., Stackhouse, T., & Wehner, E. (2003). Imitation performance in toddlers with autism and those with other developmental disorders. *Journal of Child Psychology and Psychiatry*, 44(5), 763–781.
- Schulte-Ruther, M., Greimel, E., Markowitsch, H. J., Kamp-Becker, I., Remschmidt, H., Fink, G. R., et al. (2010). Dysfunctions in brain networks supporting empathy: An fMRI study in adults with autism spectrum disorders. *Social Neuroscience*, 6, 1–21.
- Schumann, C. M., Bloss, C. S., Barnes, C. C., Wideman, G. M., Carper, R. A., Akshoomoff, N., et al. (2010). Longitudinal magnetic resonance imaging study of cortical development through early childhood in autism. *Journal of Neuroscience*, 30(12), 4419–4427.
- Sevlever, M., & Gillis, J. M. (2010). An examination of the state of imitation research in children with autism: Issues of definition and methodology. *Research in Developmental Disabilities*, 31(5), 976–984.
- Shih, P., Shen, M., Ottl, B., Keehn, B., Gaffrey, M. S., & Muller, R. A. (2010). Atypical network connectivity for imitation in autism spectrum disorder. *Neuropsychologia*, 48(10), 2931–2939.
- Solomon, M., Ozonoff, S. J., Ursu, S., Ravizza, S., Cummings, N., Ly, S., et al. (2009). The neural substrates of cognitive control deficits in autism spectrum disorders. *Neuropsychologia*, 47(12), 2515–2526.
- Stoner, R., Chow, M. L., Boyle, M. P., Sunkin, S. M., Mouton, P. R., Roy, S., et al. (2014). Patches of disorganization in the neocortex of children with autism. *The New England Journal of Medicine*, 370(13), 1209–1219.

- Sundaram, S. K., Kumar, A., Makki, M. I., Behen, M. E., Chugani, H. T., & Chugani, D. C. (2008). Diffusion tensor imaging of frontal lobe in autism spectrum disorder. *Cerebral Cortex*, 18(11), 2659–2665.
- Thakkar, K. N., Polli, F. E., Joseph, R. M., Tuch, D. S., Hadjikhani, N., Barton, J. J., et al. (2008). Response monitoring, repetitive behaviour and anterior cingulate abnormalities in autism spectrum disorders (ASD). *Brain*, 131(Pt 9), 2464–2478.
- Theoret, H., Halligan, E., Kobayashi, M., Fregni, F., Tager-Flusberg, H., & Pascual-Leone, A. (2005). Impaired motor facilitation during action observation in individuals with autism spectrum disorder. *Current Biology*, 15(3), R84–R85.
- Van Dijk, K. R., Sabuncu, M. R., & Buckner, R. L. (2012). The influence of head motion on intrinsic functional connectivity MRI. *NeuroImage*, 59(1), 431–438.
- Whiten, A., & Brown, J. (1999). Imitation and the reading of other mind: Perspective from the study of autism, normal children and non-human primates. In S. Braten (Ed.), *Intersubjective communication and emotion in ontogeny: A sourcebook* (pp. 260–280). Cambridge: Cambridge University Press.
- Zürcher, N. R., Rogier, O., Boshyan, J., Hippolyte, L., Russo, B., Gillberg, N., et al. (2013). Perception of social cues of danger in autism spectrum disorders. *PLoS One*, 8(12), e81206.

Frontal Lobe Functions

► Frontal Lobe Findings in Autism

Frontal Lobe Syndrome

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Synonyms

Disorder of executive dysfunction; Dysexecutive syndrome; Frontostriatal disorder

Short Description or Definition

The frontal lobes are the slowest regions of the central nervous system to fully develop, rendering them more susceptible to neurodevelopmental disorders, such as autism spectrum disorder, than any other region (Bradshaw 2001). The frontal lobes form part of key brain regulatory circuitry where multiple cortico-striato-thalamo-cortical loops connect the frontal lobes to the rest of the brain. Frontal lobe syndromes have in common abnormalities in structure and chemical balance in the frontal lobes, basal ganglia, and thalamus. Abnormalities in these structures and chemical imbalances are proposed to lead to deficiencies in the domains of cognition, emotions, and motor control. A mixture of these deficiencies can explain the symptomatology observed in classic frontal syndromes of childhood, for example, autism, Asperger's disorder (henceforth to be referred to as "autism spectrum disorder"), and attention deficit hyperactivity disorder (ADHD), and neurodegenerative frontal disorders, for example, Parkinson's and Huntington's disease. The common basis accounts for why there is high comorbidity within neurodevelopmental and neurodegenerative sub-groupings of frontal syndromes; for example, it is common for children with autism to also have clinical manifestations of ADHD. Such comorbidity may also point to common heritability factors across syndromes.

Categorization

Disorders which arise from damage to the frontal lobes are often referred to under the umbrella term of "frontal lobe syndrome." Frontal lobe syndrome can result as the consequence of: (1) developmental problems with the frontal lobe, as is the case with autism spectrum disorder, ADHD, obsessive compulsive disorder (OCD), Tourette's disorder (TD) schizophrenia, and depression, (2) degeneration of the frontal lobes, for example, Parkinson's disease, Alzheimer's disease, and Huntington's disease, or (3) Acquired brain injury, for example, stroke or traumatic brain injury.

While autism is defined in DSM-IV-TR (American Psychiatric Association 2000) as a psychiatric disorder, it is commonly referred to as a disorder which crosses the psychiatry-neurology divide, involving both “psychiatric” and “neurological” symptoms. When we consider autism under the umbrella of a “frontal lobe syndrome,” we group autism with other disorders where vital brain pathways which connect the frontal lobes to the rest of the brain are disrupted, either through genetic abnormalities, or environmental insult. While some of these disorders fall within the categorization of “neurological” disorders, for example, Parkinson’s disease, others fall under the categorization of psychiatric disorders, for example, autism and ADHD. It has been argued that the division between neurology and psychiatry is in many ways arbitrary; in the case of autism, frontal-related symptoms such as “repetitive motor mannerisms” could be considered as a neurological “motor disorder” or as a psychiatric “perseverative” phenomenon.

The question remains whether the presence of a particular frontal lobe syndrome is a necessary precursor for the development of autism and AD, given that some autistic behaviors (e.g., stereotyped or ritualistic activities) are present in other childhood psychiatric disorders, such as TD, OCD, and ADHD. It may be considered that autism, TS, OCD, and ADHD are five clusters of behaviors that represent disorders in several discrete dimensions, such as communication, social skills, or stereotyped/ritualistic/obsessive behavior. Autism and AD may be thought of as occurring at the same end of the social ability continuum, but as occurring at different ends of the communication ability continuum, while OCD and TS may lie toward the less-impaired ends of these dimensions. Alternatively, on the stereotyped movements-obsessive behaviors dimension, autism and TS may be thought of as occurring at the “stereotyped movement” end of the continuum, with OCD occurring at the “obsessive behaviors” end. These diagnostically distinct disorders may simply reflect different combinations of a finite set of behavioral disturbances (Bradshaw 2001).

Epidemiology

ASDs are highly prevalent with the incidence estimated at between 60 and 90 per 10,000 individuals (Autism and Developmental Disabilities Monitoring Network Surveillance Year, Principal Investigators, Centers for Disease Control and Prevention 2009; Fombonne 2009), although a recent study of students aged between 5 and 9 years in the United Kingdom sets this estimate to approximately 160 in 10,000 (Baron-Cohen et al. 2009). It is likely that the apparent increase in prevalence is due to better diagnosis and awareness in the general public. The apparent “epidemic” of ASD is similar to the increased ascertainment of ADHD in the 1990s. For this reason, “ASD” is sometimes referred to in the context of being the “new ADHD.”

Although the precise cause of executive dysfunction in ASD is not known, researchers have used neuroimaging and electrophysiological techniques to demonstrate atypical activation across a range of frontostriatal cortical and subcortical structures. This includes, for example, hypoactivation of inferior frontal gyrus, orbitofrontal cortex, and ventral medial prefrontal cortex, and hyperactivation of supplementary motor area, dorsal anterior cingulate, and middle frontal gyrus. This is presumably underpinned by various neurochemical abnormalities that have demonstrated relevance to both executive function and ASD, including abnormal serotonergic, dopaminergic, glutamatergic, and GABAergic function. More recent evidence also points to the role of impaired neural connectivity within frontostriatal circuitry, which includes reduced integrity of white matter pathways. There are, however, no consistent results in ASD demonstrating structural abnormalities (e.g., lesions) that reflect what is typically seen in frontal lobe syndrome (FLS) associated with acquired or neurodegenerative presentations.

Natural History, Prognostic Factors, Outcomes

While autism manifests in the first 3 years of life, the full extent of the disorder unfolds as the slowly

developing frontal lobes reach maturity; this in part contributes to the changing clinical manifestation of autism throughout childhood and adolescence (see “Clinical Presentation” below).

The term most commonly used to describe the impairments which arise from disruption to the frontal lobes is “executive dysfunction.” Executive dysfunction can be thought of as a loss or disruption to skills which we require to interact in a flexible way with the environment, solve problems, plan, engage efficiently in goal-directed behavior, and defer or inhibit responses. Overall, frontal lobe syndrome results in a deficit in maintaining appropriate problem-solving set for the attainment of future goals (Bradshaw 2001).

Pennington et al. (1997) proposed the “executive dysfunction hypothesis” of autism spectrum disorder on the basis that there is severe, early disruption to the frontal areas, disrupting the ability to plan complex behaviors. Working memory deficits are also noted as being important to this theory. Poor performance on tests of executive functioning is common in children with autism.

Neuropsychological research has shown that two core aspects of frontal lobe function are disturbed in autism: (1) reduced cognitive flexibility, which refers to the ability to shift one’s focus of attention from one feature, or cognitive set, to another and (2) perseveration, which refers to a tendency to repeat a verbal or nonverbal behavior beyond what is required. These frontal syndrome deficits underpin the daily challenges a child with autism experiences with planning and organizing tasks required for everyday life. The ability to sustain attention and inhibit responses has been shown to be generally intact in individuals with autism using standardized neuropsychological tests (see “Evaluation and Differential Diagnosis” below). Using more novel experimental paradigms, there is some evidence to suggest that one aspect of inhibitory function may be impaired in autism, verbal inhibition (see Rinehart et al. 2006). Such deficits are also seen in patients with neurodegenerative “frontal lobe” syndrome, Parkinson’s and Alzheimer’s disease, and neurodevelopmental disorders such as schizophrenia.

The difficulties an individual with autism has with cognitive flexibility persist into adult life. In

contrast to children with autism, adults with autism can develop better skills for managing executive dysfunction, either through further acquirement and practice of techniques to scaffold executive difficulties (see “Treatment” below), via greater insight into their own inflexibility, or through greater desire to follow the rules and behaviors necessary for successful adult life.

There is some evidence that children with autism experience a “second wave” of executive deficits around puberty due to a divergence in the developmental trajectory of the frontal lobes (Minshew and Williams 2007). At around this time children with autism are also likely to suffer from depression; this may manifest as mood disturbance and irritability, sleep and appetite disturbance, and thoughts of suicide (Tonge and Rinehart 2006). Organizational and planning deficits can become more pronounced when children are depressed or anxious. In addition to pubertal brain development, particularly impacting on the frontal lobes, the increased vulnerability to depression during adolescence may be associated with increased insight into the disorder. Graduation from Primary School, or other change in educational placement, which tends to coincide with puberty, is often a major stress for children with autism. When individuals with autism exhibit self-injury and aggression, in addition to executive dysfunction, as often occurs with individuals who have associated intellectual disability, this can result in socially inappropriate and “disinhibited” behavioral patterns which can reduce successful participation in the school and community settings, and may require more restricted care plans.

Comorbidity with other neurodevelopmental disorders, which fall under the umbrella term of “frontal lobe syndromes,” may also impact on quality of life. This comorbidity may be genetically determined, as is likely the case with ADHD, a stress-induced vulnerability, or both, as is the case for Tourette’s disorder. For example, clinically significant symptoms consistent with ADHD are reported to be highly prevalent in children with autism spectrum disorder. (Note that despite this overlap in diagnostic symptoms, our major diagnostic classification system

(DSM-IV-TR) currently precludes a dual-diagnosis of autism and ADHD.). There is some empirical evidence that children who present with autism and clinically significant levels of ADHD have higher levels of emotional-behavioral disturbances (Gargaro et al. 2010). In the case of Tourette's disorder, a frontal syndrome vulnerability, together with environmental stress, possibly related to biological or situational changes (see above), may trigger the comorbid presentation of Tic disorder or full-blown Tourette's disorder.

Clinical Expression and Pathophysiology

Executive dysfunction, in part, gives way to one of the most striking paradoxes of autism, the contrasting and uneven development of skills, sometimes characterized by peaks in certain areas of intellect, artistic, or numerical ability, which occur in the context of an impoverished ability to manage and organize the tasks necessary for daily life. In general, the impact of executive dysfunction on an individual with autism varies according to the individuals' intellectual ability, capacity for independent functioning, and provision of social, emotional, financial, and later, occupational support. It has been suggested that individuals with associated intellectual disability and epilepsy are at greater risk for deterioration during early adulthood, while there may be some improvement in functioning in normally intelligent young adults with ASDs.

Frontal lobe syndrome or executive dysfunction is commonly linked to the repetitive, stereotyped behavioral patterns which are core diagnostic features of autism (Turner 1999). The repetitive behavioral patterns may be due to a difficulty inhibiting previously relevant response patterns, or may be due to a problem with generating new ideas and responses (Turner 1999). Common repetitive, restricted, and stereotyped behavioral patterns include: lining up toys or objects, being preoccupied with special objects such as stones, repeating play sequences, obsessions with train timetables or dinosaurs. Individuals with autism may have a number of rituals

associated with daily life, such as a fixed order for bathing and dressing, or an insistence on wearing the same clothes or taking the same route to a familiar place. These rituals go beyond what is normally seen in typically developing children. There are usually some motor mannerisms, such as hand flapping, or tiptoe walking and gait abnormality.

The symptoms of frontal lobe syndrome in autism cannot be neatly separated out from the broader network of behavior-brain disruptions which characterize autism. For example, children with autism may study the detail in a picture book or closely observe spinning wheels, or a ceiling fan; this may be due to problems with disengaging from interests (executive dysfunction), or it might be due to an overly developed visual-perceptual system which enhances the experience of looking at small objects (Plaisted et al. 1999). Similarly, a child with autism may have difficulty interacting with the peer group, not primarily because of social impairments related to "theory of mind" deficits, but perhaps because of an inability to shift attentional set from his or her own topics of interest. Therefore, a child with autism may engage in a monologue of speech about his favorite interests rather than a reciprocal dialogue with his peers either due to executive-based inflexibility, or due to a poor understanding that "turn taking" is required in productive social conversations (theory of mind deficit).

The clinical manifestation of frontal lobe syndrome may differ in girls with autism in a way which is consistent with gender-related differences in the typically developing population of male and female children. For example, while boys may have an intense preoccupation with vehicles, such as trains and cars, girls may develop their special interests in areas such as teddy-bear or doll collections, and spend unusual amounts of time on craft and art to the exclusion of other types of play. Gender-neutral activities, such as "drawing," may form the "special interest" for either males or females with autism. It has been suggested that females tend to have fewer special interests than males with autism (Gillberg and Coleman 2000), although this may relate to male interests being more circumscribed

(e.g., trains, cars) than female interests (e.g., craft, doll play), and thus more clinically salient.

Pathophysiology

Development of the frontal lobes is guided by regulatory genes which lay down and prune brain pathways (referred to as apoptosis), and set the timing of how the brain will be “wired” (referred to as synaptic connectivity). There is a disturbance in these regulatory genes in autism which results in atypically developing frontal regions. The current literature points to the developmental time course of brain development being the salient disturbance, rather than the “final product” of brain development in autism (Amaral et al. 2008, p. 137). The unfolding neurobiological disruptions which characterize autism may contribute to the changes in core symptoms of autism we see over time, such as stagnation and regression in social-communicative symptoms, and changes in the focus of obsessions and nature of repetitive behavioral patterns.

Bradshaw’s (2001) frontostriatal theory provides the most comprehensive review of the pathophysiology of “frontal lobe” syndrome in the context of neurodevelopmental disorders. According to this theory, the frontostriatal system involved in neurodevelopmental disorders comprises the dorsolateral prefrontal cortex, the lateral orbitofrontal cortex, the anterior cingulate, the supplementary motor area, and the basal ganglia. These areas are involved in a number of functions, such as executive functions (e.g., functions such as self-monitoring, planning, organization, flexibility of thinking, and inhibition), motivation, control of complex behaviors, and sequencing movements. A number of neurotransmitters are implicated in the frontostriatal system, including dopamine, nor-adrenaline, acetylcholine, GABA (inhibitory), glutamate (excitatory), and serotonin.

Evaluation and Differential Diagnosis

Frontal lobe syndrome (FLS) resulting from acquired brain injury involves impairment in

cognitive and/or mood domains that are presumed to be largely governed by neural circuitry involving the frontal lobe. This may include difficulties with planning, organization, daily living skills, attention, and social cognition. Individuals with FLS may be disinhibited and impulsive, and this can include aggression or inappropriate sexual, financial, or other behaviors. Depression, anxiety, and apathy are often also present. FLS in the purest sense is thought to arise from a lesion, be it from disease or trauma. This lesion can occur anywhere within fronto-striatal-thalamic circuitry, and the nature and degree of impairment is related to the site of injury. As noted above, FLS can also relate to neurodevelopmental, e.g., ASD and neurodegenerative disorders, e.g., Parkinson’s disease.

In addition to deficits in executive function, FLS syndrome can also involve impaired social functioning, including poor emotion recognition and theory of mind, and perseverative and repetitive behaviors. Individuals with FLS can also be quite rigid and concrete in their thinking. For these reasons, ASD is often referred to under the FLS umbrella. In addition, there is much evidence for executive deficits among individuals diagnosed with an ASD, and this can include problems with planning, time estimation, attentional set-shifting, and response inhibition.

Obtaining a thorough and careful developmental history is critical for differential diagnosis. Symptoms of FLS related to lesions typically have a relatively rapid onset, and are recognized because they are uncharacteristic of the individual. ASD, on the other hand, generally must first present within certain time periods, and there is emerging evidence to suggest that some symptoms are present in infancy. When assessing individuals later in life (e.g., adolescence onward), it is crucial to involve family members in the diagnostic process, and to conduct a structural magnetic resonance imaging (MRI) brain scan.

Assessment for autism spectrum disorder (ASD) should include neuropsychological testing, which at a minimum would involve an age-appropriate, standardized cognitive assessment, but perhaps also tests of executive function. This might include inhibitory controls (e.g., word-color Stroop test), attentional set-shifting

(e.g., Wisconsin Card Sorting Test), and planning/organization (e.g., Tower of Hanoi, Trail making test). Verbal fluency assessment may also be used (e.g., FAS). This will characterize an individual's strengths and weaknesses, and allow clinicians to devise an appropriate treatment plan that meets the individual's needs. For very young children, or those for whom standardized assessment is not possible, parent and teacher report can provide insights into relative strengths and weaknesses, including problematic behaviors.

Treatment

The best outcomes for managing clinical symptoms associated with executive impairment in autism are produced by a combination of psychological, educational, and medical approaches if indicated. Treatment should be designed to target the specific needs of an individual child and also to meet the needs of parents and schools (Tonge and Rinehart 2006).

Psychosocial

Training in problem solving and organizational skills can be useful for targeting difficulties with generating new or novel behaviors and moving the individual away from rigid thinking styles. Interventions which capitalize on a child's special interests, for example, technology, may improve compliance. Reminders, schedules, visual prompting (e.g., when I finish playing trains, I can choose from a list of these activities to do), and executive-function "scaffolding" by care-givers, (e.g., breaking down tasks into smaller components, helping individuals to structure daily events to prevent becoming submerged in one activity during the day, assisting with school-based tasks) are often helpful.

Pharmacological

Drugs might be prescribed where psychological and behavioral interventions have not been

successful in the management of disturbed emotions and behaviors, in particular, repetitive self-injurious behaviors which place children at greater psychological and physical risk. There is some evidence that neuroleptic medication (e.g., haloperidol, risperidone) reduces repetitive and stereotyped behaviors; however, it may produce side effects including sedation, dystonic reactions, and increased weight gain. Unlike the original neuroleptic medications, new atypical drugs are not associated with the risk of irreversible tardive dyskinesia. Tricyclic antidepressants (e.g., imipramine, clomipramine) are effective anxiolytics, and may also reduce repetitive behaviors, but may have potential cardiotoxic effects (Tonge and Rinehart 2006). Parents may be stressed by the uncertainty of potential long-term effects of placing their child on medication. Currently there is a lack of good long-term evidence for the effectiveness of stimulant medication on learning but they may decrease the risk of later substance abuse (Tonge and Rinehart 2006).

See Also

- [Frontal Lobe Findings in Autism](#)
- [Prefrontal Cortex](#)

References and Reading

- Amaral, D. G., Schumann, C. M., & Nordahl, C. W. (2008). Neuroanatomy of autism. *Trends in Neurosciences*, 31(3), 137–145.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders IV-TR* (4th ed.). Washington, DC: American Psychiatric Association Press.
- Autism and Developmental Disabilities Monitoring Network Surveillance Year Principal Investigators, Centers for Disease Control and Prevention. (2009). Prevalence of autism spectrum disorders – Autism and developmental disabilities monitoring network, United States, 2006. *Morbidity and Mortality Weekly Report. Surveillance Summaries*, 58(10), 1–20.
- Baron-Cohen, S., Scott, F. J., Allison, C., Williams, J., Bolton, P., Matthews, F. E., et al. (2009). Prevalence of autism-spectrum conditions: UK school-based population study. *The British Journal of Psychiatry*, 194(6), 500–509.

- Bradshaw, J. L. (2001). *Developmental disorders of the frontostriatal system: Neuropsychological, neuropsychiatric and evolutionary perspectives*. Hove, East Sussex: Psychology Press.
- Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. *Pediatric Research*, 65(6), 591–598.
- Gargaro, B., Rinehart, N., Bradshaw, J., Tonge, B. J., & Sheppard, D. (2010). Autism and ADHD: How far have we come in the comorbidity debate? *Neuroscience and Biobehavioural Reviews*, 35(5), 1081–1088.
- Gillberg, C., & Coleman, M. (2000). *The biology of the autistic syndromes* (3rd ed.). London: Mac Keith Press.
- Minshew, N., & Williams, D. L. (2007). The new neurobiology of autism: Cortex, connectivity, and neuronal organization. *Archives of Neurology*, 64(7), 945–950.
- Pennington, B. F., Rogers, S. J., Bennetto, L., McMahon, E., Reed, D. T., & Shyu, V. (1997). Validity tests of executive dysfunction in autism and other disorders. In J. Russell (Ed.), *Autism as an executive disorder*. New York: Oxford Press.
- Plaisted, K., Swettenham, J., & Rees, L. (1999). Children with autism show local precedence in a divided attention task and global precedence in a selective attention task. *Journal of Child Psychology and Psychiatry*, 40(5), 733–742.
- Rinehart, N. J., Bradshaw, J. L., Moss, S. A., Brereton, A., & Tonge, B. (2006). Pseudo-random number generation in children with high-functioning autism and Asperger's disorder: Further evidence for a dissociation in executive functioning. *Autism*, 10(1), 70–85.
- Tonge, B., & Rinehart, N. J. (2006). Autism and attention deficit hyperactivity. In A. H. V. Schapira et al. (Eds.), *Neurology- basic and clinical neuroscience*. Philadelphia: Elsevier.
- Turner, M. (1999). Annotation: Repetitive behavior in autism: A review of psychological research. *Journal of Child Psychology and Psychiatry*, 40, 839–849.

Frontostriatal Disorder

► Frontal Lobe Syndrome

FTT

► Finger-Tapping Test

Full Physical Prompt

► Hand-Over-Hand Assistance

Functional Analysis

► Analog Condition Functional Analysis

Functional Analysis Screening Tool

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Synonyms

FAST (Functional Assessment Screening Tool)

Description

The FAST is a type of indirect functional assessment method. It is comprised of a self-report checklist designed to identify whether maladaptive behavior is maintained via attention and tangibles, escape, sensory stimulation, or pain attenuation.

Prior to the rating scale section of the instrument, the FAST has two information sections: Informant-Client Relationship and Problem Behavior Information. The Information-Client Relationship section contains four questions regarding the informant-client relationship (e.g., relationship to the client, how often and in what situations they interact). The Problem Behavior Information section contains eight questions regarding the topography, frequency, and severity of the target behavior.

The checklist section contains 16 items. Each question is marked as either “yes,” “no,” or “not applicable.” Items endorsed with a “yes” are then categorized into one of the four possible sources of reinforcers for problem behavior: attention/preferred items (social positive reinforcement), escape (social negative reinforcement), sensory stimulation (automatic positive

reinforcement), and pain attenuation (automatic negative reinforcement). Behavioral function is inferred by calculating a summary score for the four possible maintaining variables. The FAST interview process is 10–15 min in length.

Historical Background

The FAST was originally developed in 1995, as an adjunct to functional analysis procedures. The scale underwent several revisions before the most recent version, which was released in 2005. The initial scale included 34 items. After field-testing the FAST with over 300 individuals, the item pool was reduced to the present 16 items.

Psychometric Data

Psychometric data for the FAST are generally sparse. Zaja et al. (2010) evaluated and compared the psychometric properties of the Questions About Behavioral Function (QABF) scale (Matson and Vollmer 1995), the Functional Assessment for Multiple Causality (FACT) scale (Matson et al. 2003), and the FAST for the indirect assessment of self-injurious, stereotypic, and aggressive/destructive behavior. The FAST subscales generally showed the lowest inter-rater agreement with correlations ranging from poor to good relative to the QABF and FACT. Zaja et al. also found that the FAST had unacceptably low internal consistency especially for the social attention and social escape subscales. The authors indicated that the weaker psychometrics properties (reliability and validity) for the FAST were likely related to its length, limited response format (“Yes”/“No”/“NA”), and restricted range of subscales (4 instead of 5).

Iwata et al. (2013) evaluated the reliability and validity of the FAST. In the first study, the authors assessed the interrater reliability of the FAST with 196 problem behaviors through independent administration of the assessment to pairs of raters. The average item-by-item agreement between pairs of raters was 71.5%, with a range of 53.3–84.5%. In the second part of the

investigation, the authors compared the results of the FAST to the results of 69 functional analyses. The authors found that the FAST predicted the primary function of challenging behavior 63.8% of the time. The authors note that, while the results are comparable to other indirect assessment tools, they are not an adequate substitute for functional analysis procedures.

Clinical Uses

The FAST is an indirect functional assessment procedure. The purpose of the instrument is to provide preliminary information regarding the cause (of function) of maladaptive behavior. The information gathered from the FAST can be incorporated into direct assessment methods. Given the questionable reliability of most indirect assessment measures (such as interviews and rating scales), it is often recommended that these methods be employed in conjunction with direct observation and/or functional analysis procedures and not as the sole means to determining behavioral function (Zarcone et al. 1991).

See Also

► [Functional Behavior Assessment](#)

References and Reading

- Iwata, B., & DeLeon, I. (2005). *The functional analysis screening tool*. Gainesville: The Florida Center on Self-Injury, University of Florida.
- Iwata, B. A., DeLeon, I. G., & Roscoe, E. M. (2013). Reliability and validity of the functional analysis screening tool. *Journal of Applied Behavior Analysis*, 46(1), 271–284.
- Matson, J. L., & Vollmer, T. R. (1995). *The questions about behavioral function (QABF) user's guide*. Baton Rouge: Scientific Publishers.
- Matson, J. L., Kuhn, D. E., Dixon, D. R., Mayville, S. B., Laud, R. B., Cooper, C. L., Malone, C. J., Minshawi, N. F., Singh, A. V., Luke, M. A., Lott, J. D., & Matson, M. L. (2003). The development and factor structure of the functional assessment for multiple causality (FACT). *Research in Developmental Disabilities*, 24, 485–495.

- Zaja, R. H., Moore, L., van Ingen, D. J., & Rojahn, J. (2010). Psychometric comparison of the functional assessment instruments QABF, FACT and FAST for self-injurious, stereotypic and aggressive/destructive behaviour. *Journal of Applied Research in Intellectual Disabilities*, 24(1), 18–28.
- Zarcone, J. R., Rodgers, T. A., Iwata, B. A., Rourke, D. A., Dorsey, M. F. (1991). Reliability analysis of the motivation assessment scale: A failure to replicate. *Research in Developmental Disabilities*, 12(4), 349–360.

Functional Analysis: Assessment Procedures for Complex Challenging Behavior

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Definition

Assessing behavior through data-based collection procedures is a necessary component of applied behavior analysis (Cooper et al. 2007). The primary goal is to establish a reliable relationship between the treatment and behavior change. This concept forms the basis for the assessment of challenging behavior in applied behavior analysis. Functional analysis is the procedure by which environmental conditions are manipulated to reliably evoke a target behavior (Carr and Durand 1985; Cooper and Harding 1993; Iwata et al. 1982/1994a, 1990; O'Reilloy et al. 2009; Roscoe et al. 2010). Based upon the results of these assessments, maladaptive behavior is conceptualized as being motivated by a particular function, thus allowing appropriate interventions to be developed. An important component of functional analytic methodology is that behavior must be understood by its consequences within the environment and not solely by its topography or form (Cooper and Harding 1993). The majority of functional analysis procedures currently used are based upon the seminal work of Iwata et al. (1982/1994a).

Functional analysis methodology has been applied to a variety of treatment settings. The original research on these procedures was conducted in highly controlled treatment settings (Carr and Durand 1985; Cooper and Harding 1993; Iwata et al. 1982/1994a, 1990). In these environments, experimenters have exposed participants to repeated treatment conditions in order to establish a reliable relationship between environmental contingencies and the occurrence of a target behavior (Carr and Durand 1985; Iwata et al. 1982/1994a, 1990). Research attention has expanded toward evaluating the use of functional analytic procedures in outpatient and community-based settings with less precise and sustained control (Cooper and Harding 1993). Further, functional analytic models have evolved to include a variety of brief methodologies which have gained substantial evidence-based support (e.g., LaRue et al. 2010; Thomason-Sassi et al. 2011). The ongoing research on functional analysis continues to provide a great deal of support for its methods to represent the best practice means of assessing challenging behavior (e.g., Cooper et al. 1992; Derby et al. 1992, 1994; Ruiz and Kubina 2017).

Historical Background

Carr (1977) established the conceptual foundation for functional analysis. The model for clinical practice is associated with the work of Iwata et al. (1982/1994a) and Carr and Durand (1985). The methodology developed in these studies has since been widely examined and used in the applied behavior analysis literature (Hanley et al. 2003). The initial study by Iwata et al. (1982/1994a) utilized four assessment conditions to evaluate self-injurious behavior in nine individuals diagnosed with developmental disability, ranging in age from 19 months to 17 years and 2 months. The conditions included in the functional analysis were social disapproval, academic demand, unstructured play, and alone. During the assessments, conditions lasted for 15 min and were randomly ordered for each participant. The functional analysis continued until stable levels of responding were observed in each condition, or until 12 days of assessment were completed.

The social disapproval condition was designed to replicate contingencies for positive reinforcement in the form of attention for engaging in self-injury. The participant was instructed to play with toys while the experimenter worked. If the participant engaged in self-injury, the experimenter provided physical and vocal attention. The academic demand condition tested for the presence of negative reinforcement contingencies in the form of escape from work for engaging in self-injury. The experimenter ran academic programs appropriate to each participant's ability level. Social praise was delivered after each response whether or not the response was correct. If the participant engaged in self-injurious behavior, the experimenter turned away and terminated the learning trial. The alone condition was designed to assess for self-injurious behavior maintained by automatic reinforcement. The participant was left alone without access to attention or tangible items. The experimenter did not provide a consequence for an occurrence of self-injury. Finally, the unstructured play condition served as a control condition for the functional analysis. In this condition, the experimenter provided noncontingent attention and access to toys and gave no demands. Again, no consequence was provided contingent upon an occurrence of self-injury.

Operational definitions were created for each participants' topography of self-injury to address individual differences. Interobserver agreement was calculated to ensure that all observers were able to reliably identify all the topographies of challenging behavior. The results of the Iwata et al. (1982/1994a) study demonstrated that similar topographies of behavior can serve different functions. In their study, the level of responding varied from individual to individual across the assessment conditions. As a result of these data, Iwata et al. (1982/1994a) supported functional analysis as a means of systematically evaluating the stimuli maintaining behavior and subsequently the use of individualized assessment and intervention procedures for self-injurious behavior.

Carr and Durand (1985) echoed the Iwata et al. (1982/1994a) results, showing that similar forms of challenging behavior can be maintained by

different contingencies in each individual. The study evaluated a number of topographies of challenging behavior experienced by four participants, ages 7–14 years, with either developmental disabilities or brain damage. The functional analysis conditions were designed to assess escape and attention motivations for each target behavior. The "easy 100" condition served as the control condition for the analysis. In this condition, the experimenter provided easy demands and attention during 100% of the condition's intervals. In the "easy 33" condition, the experimenter again utilized easy demands but only provided attention during 33% of the intervals. This condition was used to assess for an attention motivation for each target behavior. During the "difficult 100" condition, the participants were given challenging demands and attention during 100% of the condition's intervals. It was expected that this condition would assess for an escape function maintaining any target behavior.

Consequences were provided for all topographies of behavior in the same manner during each condition. All behavior except darting and responses that risked physical injury were placed on extinction (Carr and Durand 1985). If the participant darted from work and did not return in 10 s, she or he was physically guided back to the table. In cases where physical risk was a concern, the participant's hands were restrained for 5–10 s while the experimenter followed through with the work demands. The results of the functional analyses suggested that the various forms of challenging behavior of the participants were maintained by different environmental contingencies. The data from the Carr and Durand study supported those obtained by Iwata et al. (1982/1994a). That is, functional analysis was endorsed as a means for evaluating challenging behavior.

Carr and Durand (1985) further supported functional analysis as a means of assessing and treating challenging behavior by implementing functional communication training for each of the participants. The target of the training was requests for attention and help as a replacement for challenging behavior reinforced by attention and escape, respectively. By providing consistent reinforcement for appropriate requests for

attention and help, all participants' challenging behavior decreased. Functionally equivalent interventions were thereby supported as the optimal treatment for challenging behavior.

Contemporary Considerations

A great deal of research has been conducted on functional analysis methodology since the seminal studies by Carr and Durand (1985) and Iwata et al. (1982/1994a). Systematic reviews of the literature on functional analysis have consistently supported the procedure's efficacy in both identifying the function of challenging behavior and determining effective replacement skills (e.g., Hanley et al. 2003, 2014; Iwata et al. 1994b; LaRue et al. 2010; O'Reilloy et al. 2009; Thomason-Sassi et al. 2011). Hanley et al. (2014) reviewed 575 functional analysis studies, 96% of which rendered usable outcomes. While the functional analysis procedures utilized in typical studies are rarely identical, the basic premise of controlled antecedents and consequences as defined by environmental manipulations has aided in the development of functionally equivalent interventions that have shown a decrease in problem behavior and an increase in targeted replacement skills (Hanley et al. 2014; Iwata et al. 1994b).

Hanley et al. (2003) reviewed the literature to identify trends for best practices in functional analysis methodology. Their review supported the use of functional analysis to study many topographies of challenging behavior in individuals with disabilities of varying severities. While a substantial portion of the literature has focused on learners with developmental disabilities or intellectual disability, it is important to note that a variety of other mental disorders and mild behavior problems have been included in analyses (e.g., Cooper et al. 1990; Doggett et al. 2001).

The experimental conditions most prevalent in the literature for analogue functional analyses are developed based upon those used by Iwata et al. (1982/1994a). These conditions, social positive reinforcement (attention), social negative reinforcement (escape), automatic reinforcement

(alone), and control, appear as they were described above. In addition, a tangible condition has been applied to in a number of studies (e.g., Fisher et al. 2000; Moore et al. 2002a; Mueller et al. 2001; Shirley et al. 1999). In this condition, the individual is given access to a highly preferred item for 1 min at which point the experimenter removes the item and places it out of reach (Mueller et al. 2001). The participant is told that the target item is unavailable and directed toward other low-preference items. Upon the occurrence of the target behavior, the experimenter grants access to the high-preference item. These variables have continued to be incorporated into the conditions of subsequent functional analysis models (e.g., trial-based or response latency; LaRue et al. 2010).

Several concerns are common in developing the conditions for a functional analysis. These concerns often focus on the presence of confounds in the assessment conditions (Hanley et al. 2003; Moore et al. 2002a; Shirley et al. 1999) as well as the challenges of repeatedly evoking challenging behavior (e.g., Thomason-Sassi et al. 2011). Hanley et al. (2003) noted that while there are not universal firm rules for functional analysis, certain components have been identified that can be considered among best practices. Included here are factors such as limiting assessment to a manageable number of responses, considering the influence of establishing operations on the contingencies active in each condition, relatively short sessions, brief designs that can be expanded on an individual basis, and programming for consequences (Hanley et al. 2003).

Concerns regarding session confounds are common. For example, such challenges were illustrated by Moore et al. (2002a) regarding the influence that attention can play during a tangible condition. A functional analysis was conducted on a child's self-injurious behavior (SIB). The results of the analysis suggested that SIB was a multi-operant behavior maintained by positive reinforcement in the form of attention and access to preferred items (Moore et al. 2002a). In a follow-up analysis, the level of attention provided during the tangible condition was evaluated. By reducing the amount of attention paired with the

presentation of the tangible, the rate of SIB was decreased. Moore et al. (2002a) suggested that the attention inadvertently delivered during the tangible condition was acting as a confound and evoking SIB. Weakening the contingency between the target behavior and access to attention (e.g., delivering attention noncontingently) may serve to control for the influence of confounds (Moore et al. 2002a). If the contingent presentation of attention does confound the tangible condition, it stands to reason that all independent variables should be carefully controlled during the development of functional analysis sessions.

Another similar methodological concern was identified by Shirley et al. (1999) in a study on incidental maintenance in the tangible condition. A functional analysis conducted on an individual's hand mouthing suggested that the behavior was maintained by automatic reinforcement and access to tangible items. Observations of the behavior indicated that the preferred items used in the assessment were almost never provided as a natural consequence. Therefore, the functional analysis may have identified a tangible function that was not actually maintaining challenging behavior but rather could have maintained behavior if it were presented contingently (Shirley et al. 1999). Shirley et al. suggested caution when using the results of a preference assessment without collecting some form of data on the natural environment.

Application across diverse settings and brief session durations have gained increased attention in recent years as pressing topics requiring investigation in the area of functional analysis methodology. There is now a growing literature base demonstrating the use of functional analysis in less controlled settings such as schools and outpatient clinics (e.g., Cooper et al. 1992; Cooper and Harding 1993; Iwata et al. 2000; LaRue et al. 2010; Moore et al. 2002b; Ruiz and Kubina 2017; Thomason-Sassi et al. 2011; Umbreit 1995). To be useful within a diverse array of settings, considerations related to the efficient application of functional analysis procedures must be addressed.

A study by Wallace and Iwata (1999) considered the influence of session duration on determining function. Forty-six individuals

participated in functional analyses based on the model described by Iwata et al. (1982/1994a). Tangible conditions were also run for those individuals whose indirect assessment suggested that access to tangible items might evoke the target behavior. The sessions were videotaped, and three sets of data were prepared for each participant, by using the first 5, 10, and 15 min of the sessions. Trained independent raters evaluated data from each video. The results rendered strong agreement between the 15- and 10-min sessions and only three disagreements between 15- and 5-min sessions. As a result, shorter session duration was supported as a means for increasing the practical application of functional analysis methodology (Wallace and Iwata 1999). Similarly, LaRue et al. (2010) and Thomason-Sassi et al. (2011) both identified efficiencies in models applying limited session durations while maintain a high degree of correspondence with traditional analogue findings.

Another concern regarding functional analysis conducted in controlled clinical settings where naturally occurring environmental events are much less likely to influence assessment conditions is that the functional analysis may suggest a relationship that does not exist in the natural environment (Hanley et al. 2003). This phenomenon may compromise the ecological validity of the findings. In addition, a long-standing factor in treatment is that most individuals referred for treatment are not admitted directly to inpatient facilities. Typically intervention attempts on an outpatient basis constitute the first stage of treatment (Cooper et al. 1990). By developing a model compatible with an outpatient treatment facility and using parents during a functional analysis, Cooper et al. (1990) were able to identify the functions maintaining different topographies of challenging behavior and develop successful treatment interventions. This research has continued extensively, and a growing body of research has demonstrated the use of functional analysis procedures in a variety of treatment settings such as outpatient clinics, schools, and homes (e.g., Cooper and Harding 1993; Cooper et al. 1990; Doggett et al. 2001; LaRue et al. 2010; Thomason-Sassi et al. 2011; Umbreit 1995).

Doggett et al. (2001) and Umbreit (1995) tested the application of functional analysis methodology in classroom environments. Their studies focused on developing a process in which the conceptual foundations of applied behavior analysis were incorporated with an efficient use of classroom resources. Similar to Cooper et al. (1992) and Cooper and Harding (1993), Doggett et al. and Umbreit suggested the use of indirect data collection procedures, as well as descriptive analyses and observations, to aid in the interpretation of functional analysis data; however, the use of such data has not been consistently shown to have high agreement with objective functional analysis outcomes (e.g., Paclawskyj et al. 2008; Zarccone et al. 1991). While correspondence between indirect methods and functional analysis conditions may be low, it has been suggested that such procedures may increase the ecological validity of the analysis (e.g., Fisher et al. 2016).

Another pressing issue in the development of functional analysis methods has been the ability to engage multiple participants in the implementation of conditions. This is particularly relevant for those analyses conducted outside of highly controlled clinical settings. In the Doggett et al. (2001) study, behavioral consultants assisted general education classroom teachers in conducting an entire functional assessment. The functional analysis component of the assessment was implemented during periods of general classroom instruction. Behavioral consultants trained and supervised the entire assessment procedure, ensuring that the teachers played a primary role in hypothesis development and data analysis. Similarly, in Umbreit (1995), a teacher was supported in the implementation of a functional analysis that proved successful in identifying a function of the student's challenging classroom behavior. In a review of trial-based functional analysis models, Ruiz and Kubina (2017) found that teachers and paraprofessionals were able to implement conditions and that the training for this could occur in short intervals. The successful implementation of functional analysis in a variety of settings and with individuals who only require brief training to implement conditions further demonstrates the technology's use outside of

controlled inpatient clinics (Cooper and Harding 1993; Doggett et al. 2001; Umbreit 1995; Ruiz and Kubina 2017).

Future Directions

In addition to modifications to session duration and implementor, changes to session structure have been introduced and are increasingly common (e.g., Bloom et al. 2011; Hanley et al. 2014; LaRue et al. 2010; Thomason-Sassi et al. 2011). Thomason-Sassi et al. found that in latency functional analyses where 5-min sessions were used and where the conditions were terminated immediately following implementation of a consequence, that nine of ten cases showed correspondence between this brief format and standard analyses. Similarly, multiple investigations of trial-based functional analysis models have found strong correspondence with transitional formats. Such models tend to deploy brief conditions that terminate after the first occurrence of a challenging behavior and are either preceded or followed by a condition-specific control phase (or motivating operation absent phase). LaRue et al. compared standard analyses with trial-based methods which implemented conditions of 1–2 min in length and found exact correspondence in four of five participants, with partial correspondence found in the fifth case. Similarly, Bloom et al. (2011) determined trial-based functional analysis to be a viable method, particularly for assessments conducted within natural routines and when more standard analysis would not be feasible. As trial-based methods have gained increasing attention in the literature, Ruiz and Kubina (2017), in an analysis of 17 trial-based studies, found a number of benefits to these models such as implementation within the typical day, few repetitions of severe behavior, shorter session durations, and ease of training implementors.

Ongoing efforts to further improve the efficiency and social validity of functional analyses also include investigations to incorporate functional assessment interviews and observations to directly inform the development of the functional analysis (Hanley et al. 2014). This suggests that

synthesizing multiple assessment and treatment elements within conditions can improve outcomes and the social validity of the analyses. However, Fisher et al. (2016) questioned the benefits of synthesized conditions, including any improvements achieved through use of open-ended interviews. Though they did note the potential for improved ecological validity from such efforts. This area of development in functional analysis remains actively debated and subject to ongoing research investigations.

Functional analysis is considered among the most efficient assessment technologies as it provides data on demonstrated relationships between environmental events and target behavior. This allows for the development of behavior intervention and teaching plans that are precisely crafted to the needs of the individual. Functional analytic methods enable investigators to control confounding environmental variables that make direct observation-based assessments often very difficult. For example, difficulties discriminating whether escaping a difficult demand or gaining access to direct teacher attention is the most relevant feature of a student's noncompliant behavior. Using functional analysis, these variables are precisely controlled. The demonstrated relationship between environmental triggers and behavior leads to therapeutic interventions that are functionally driven and evaluable. As methods have expanded to include brief and naturally embedded methods, the impact of functional analysis across both research and practice communities has grown substantially.

See Also

- [Analog Condition Functional Analysis](#)
- [Functional Behavior Assessment](#)

References and Reading

- Bloom, S. E., Iwata, B. A., Fritz, J. N., Roscoe, E. M., & Carreau, A. B. (2011). Classroom application of a trial-based functional analysis. *Journal of Applied Behavior Analysis*, 44, 19–31.

- Carr, E. G. (1977). The motivation of self-injurious behavior: A review of some hypotheses. *Psychological Bulletin*, 84, 800–816.
- Carr, E. G., & Durand, M. V. (1985). Reducing behavior problems through functional communication training. *Journal of Applied Behavior Analysis*, 18, 111–126.
- Cooper, L. J., & Harding, J. (1993). Extending functional analysis procedures to outpatient and classroom settings for children with mild disabilities. In S. F. Warren, & J. Reichle (series Eds.), J. Reichle, & D. P. Wacker (volume Eds.), *Communication and language series: Vol. 3. Communicative alternatives to challenging behavior: Integrating functional assessment and intervention strategies* (pp. 41–62). Baltimore: Paul H. Brookes.
- Cooper, L. J., Wacker, D. P., Sasso, G. M., Reimers, T. M., & Donn, L. K. (1990). Using parents as therapists to evaluate appropriate behavior of their children: Application to a tertiary diagnostic clinic. *Journal of Applied Behavior Analysis*, 23, 285–296.
- Cooper, L. J., Wacker, D. P., Thursby, D., Plagmann, L. A., Harding, J., Millard, T., et al. (1992). Analysis of the effects of task preferences, task demands, and adult attention on child behavior in outpatient and classroom settings. *Journal of Applied Behavior Analysis*, 25, 823–840.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis*. Upper Saddle River: Prentice Hall.
- Derby, K. M., Wacker, D. P., Sasso, G., Steege, M., Northup, J., Cigrand, K., et al. (1992). Brief functional assessment techniques to evaluate aberrant behavior in an outpatient setting: A summary of 79 cases. *Journal of Applied Behavior Analysis*, 25, 713–721.
- Derby, K. M., Wacker, D. P., Peck, S., Sasso, G., DeRaad, A., Berg, W., et al. (1994). Functional analysis of separate topographies of aberrant behavior. *Journal of Applied Behavior Analysis*, 27, 267–278.
- Doggett, R. A., Edwards, R. P., Moore, J. W., Tingstrom, D. H., & Wilczynski, S. M. (2001). An approach to functional assessment in general education classroom settings. *School Psychology Review*, 30, 313–328.
- Fisher, W. W., O'Connor, J. T., Kurtz, P. F., DeLeon, I. G., & Gotjen, D. L. (2000). The effects of noncontingent delivery of high and low-preference stimuli on attention-maintained destructive behavior. *Journal of Applied Behavior Analysis*, 33, 79–83.
- Fisher, W. W., Greer, B. D., Romani, P. W., Zangrillo, A. N., & Owen, T. M. (2016). Comparisons of synthesized and individual reinforcement contingencies during functional analysis. *Journal of Applied Behavior Analysis*, 49, 596–616.
- Hanley, G. P., Iwata, B. A., & McCord, B. E. (2003). Functional analysis of problem behavior: A review. *Journal of Applied Behavior Analysis*, 36, 147–185.
- Hanley, G. P., Jin, S. C., Vanselow, N. R., & Hanratty, L. A. (2014). Producing meaningful improvements in problem behavior of children with autism via synthesized analyses and treatments. *Journal of Applied Behavior Analysis*, 47, 16–36.

- Iwata, B. A., Pace, G. M., Kalsher, M. J., Cowdery, G. E., & Cataldo, M. F. (1990). Experimental analysis and extinction of self-injurious escape behavior. *Journal of Applied Behavior Analysis*, 23, 11–27.
- Iwata, B. A., Dorsey, M. F., Slifer, K. J., Bauman, K. E., & Richman, G. S. (1994a). Toward a functional analysis of self-injury. *Journal of Applied Behavior Analysis*, 27, 197–209, 215–240. (Reprinted from *Analysis and Intervention in Developmental Disabilities*, 2, 3–20, 1982).
- Iwata, B. A., Pace, G. M., Dorsey, M. F., Zarcone, J. R., Vollmer, T. R., Smith, R. G., et al. (1994b). The functions of self-injurious behavior: An experimental–epidemiological analysis. *Journal of Applied Behavior Analysis*, 27(2), 215–240.
- Iwata, B. A., Wallace, M. D., Kahng, S., Lindberg, J. S., Roscoe, E. M., Conners, J., et al. (2000). Skill acquisition in the implementation of functional analysis methodology. *Journal of Applied Behavior Analysis*, 33, 181–194.
- LaRue, R. H., Lenard, K., Weiss, M. J., Bamond, M., Palmieri, M., & Kelley, M. E. (2010). Comparison of traditional and trial-based methodologies for conducting functional analyses. *Research in Developmental Disabilities*, 31, 480–487.
- Moore, J. W., Mueller, M. M., Dubard, D., Roberts, D. S., & Sterling-Turner, H. E. (2002a). The influence of therapist attention on self-injury during a tangible condition. *Journal of Applied Behavior Analysis*, 35, 283–286.
- Moore, J. W., Edwards, R. P., Sterling-Turner, H. E., Riley, J., DuBard, M., & McGeorge, A. (2002b). Teacher acquisition of functional analysis methodology. *Journal of Applied Behavior Analysis*, 35, 73–77.
- Mueller, M. M., Wilczynski, S. M., Moore, J. W., Fusilier, I., & Trahan, D. (2001). Antecedent manipulations in a tangible condition: Effects of stimulus preference on aggression. *Journal of Applied Behavior Analysis*, 34, 237–240.
- O'Reilloy, M., Rispoli, M., Davis, T., Machalick, W., Lang, R., Sigafoos, J., et al. (2009). Functional analysis of challenging behavior in children with autism spectrum disorders: A summary of 10 cases. *Research in Autism Spectrum Disorders*, 4, 1–10.
- Paclawskyj, T. R., Matson, J. L., Rush, K. S., Smalls, Y., & Vollmer, T. R. (2008). Assessment of the convergent validity of the questions about behavior function scale with analogue functional analysis and the motivation assessment scale. *Journal of Intellectual Disability Research*, 45, 484–494.
- Roscoe, E. M., Kindle, A. E., & Pence, S. T. (2010). Functional analysis and treatment of aggression maintained by preferred conversational topics. *Journal of Applied Behavior Analysis*, 43, 723–727.
- Ruiz, S., & Kubina, R. M. (2017). Impact of trial-based functional analysis on challenging behavior and training: A review of the literature. *Behavior Analysis: Research and Practice*, 17, 347–356.
- Shirley, M. J., Iwata, B. A., & Kahng, S. (1999). False-positive maintenance of self-injurious behavior by access to tangible reinforcers. *Journal of Applied Behavior Analysis*, 32, 201–204.
- Thomason-Sassi, J. L., Iwata, B. A., Neidert, P. N., & Roscoe, E. M. (2011). Response latency as an index of response strength during functional analyses of problem behavior. *Journal of Applied Behavior Analysis*, 44, 51–67.
- Umbreit, J. (1995). Functional assessment and intervention in a regular classroom setting for the disruptive behavior of a student with attention deficit hyperactivity disorder. *Behavioral Disorders*, 20, 267–278.
- Wallace, M. D., & Iwata, B. A. (1999). Effects of session durations on functional analysis outcomes. *Journal of Applied Behavior Analysis*, 32, 175–183.
- Zarcone, J. R., Rodgers, T. A., Iwata, B. A., Rourke, D. A., & Dorsey, M. F. (1991). Reliability analysis of the motivation assessment scale: A failure to replicate. *Research in Developmental Disabilities*, 12, 349–360.

Functional Assessment

► Functional Behavior Assessment

Functional Assessment and Curriculum for Teaching Everyday Routines

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Definition

The Functional Assessment and Curriculum for Teaching Everyday Routines (FACTER) is a criterion-referenced assessment and curriculum for teaching daily routines and activities of daily living. It is designed to address the needs of individuals with developmental disabilities. FACTER provides task-analyzed skills that cover a variety of skills needed to navigate an individual's daily environment. There are two versions of FACTER, an elementary level and secondary level. Both consist of formative and summative assessments

of student learning and provide a framework for developing and tracking instruction across routines.

Historical Background

The history of FACTER is outlined in the Program Manual (Arick et al. 2000). FACTER was developed to meet the requirements of the Individuals with Disabilities Education Act Amendments of 1997. The first iteration, named the Extended Career and Life Role Assessment System (Arick et al. 2000), was designed to assess individuals who were not participating in statewide or district assessments and meet the state standards for career-related learning. There were several features of the Extended CLRAS that allowed the state of Oregon to meet IDEA mandates for reporting assessment results.

The first version of FACTER was an extension of the Extended CLRAS that added the instructional component. Individualized lesson plans were added to assist educators in developing instructional programs that complemented the assessments conducted for program planning and assessing progress on intervention goals and objectives. Progress monitoring was added to support ongoing instructional development and modification and track the effectiveness of interventions. Skills targeted for assessment and instruction were derived from special education standards and areas of functioning referenced in the literature and clinical practices as important for independent living.

Rationale or Underlying Theory

The primary rationale for FACTER is the development of skills that will maximize independent skill development, a primary goal of special education. Assessment of skills and instructional practices are developed to maximize student independence and allow for a complete assessment to goal development and instructional planning to implementation of instruction. The underlying

instructional theory is that of direct instruction utilizing task analytic approaches to instruction. Additional strategies utilized during instruction include visual supports, tactile systems, and social stories. Preteaching of routines and instruction during the routine are implemented during instruction. Criterion-referenced assessment is a critical feature of this program with skills that are broken down into smaller component skills that allow for analysis and progression through the sequence of behaviors necessary to independently perform the skill.

Goals and Objectives

The goals and objectives of FACTER are related to the instruction of routines and underlying skills related to those routines to enhance a student's independent living skills. Individualized goals and objectives are developed based on results obtained in the FACTER assessment phase. Subsequently, lesson plans based on these objectives are developed and implemented utilizing direct instructional approaches. Ongoing data collection is utilized to track individual's progress as well as report the results of larger groups of students. Individual student tracking allows for adjustment of instructional programming in response to student performance. Summative assessment utilizing criterion-referenced assessment allows for reporting on Individualized Educational Plans and for statewide and district-wide assessments.

Treatment Participants

FACTER is designed for individuals who require special education services with a particular focus on individuals in need of life skills training and those that are not participating in statewide or district-wide assessments. A particular focus is on developing skills that maximize student independence and align with state educational standards for post-secondary skills that support career, independent living, and involvement within society as a responsible citizen.

Treatment Procedures

There are three components outlined in the FACTER manual related to treatment procedures. These are:

Assessment Phase

This phase consists of utilizing the Program Manual of FACTER to assess the individual's ability across a variety of domains. These include Living Skills, Transition, Academic, Leisure, and Community at the elementary level with the addition of Career as a domain in the secondary level. During the assessment phase, the educator obtains baseline ratings of routines on a 5-point scale from 0 (does not complete with physical assistance) to 4 (completes independently). An N can be used to indicate Not Applicable which may be due to physical or medical limitations that prevent the student from engaging in the routine or indicates that the routine is deemed inappropriate by the educational team or is not supported by the school environment. Baseline ratings are completed by individuals who are familiar with the student being assessed.

Following the baseline ratings, routines are selected for performance assessment. Corresponding skills related to completion of the routine are also selected. Skill areas include expressive communication, receptive communication, problem solving, teamwork/social skills, motor skills, and functional academics. These are subsequently broken down from basic to complex. All selections in this step are made on the basis of IEP goals and objectives developed by the educational team. The criterion for selection is that if completed, these skills will lead to increased independence by the student. Data are collected via direct observation on these routines and skills and are charted in the student booklets included with the Program Manual.

Instruction Phase

This phase includes a variety of instructional strategies that can be utilized by educators to instruct students on independence of routines

selected in the assessment phase. Routines are typically taught in inclusive settings, with transition routines integrated between routines from other domains. That is, when a routine in the classroom involves an academic task, then a transition to group instruction, the educator strings these three routines (academic, transition, social) together. Taken as a whole, increasing independence on these routines allows the student to be better able to navigate a typical environment with as little assistance as possible. This includes strategies of prompting and prompt fading to increase independent functioning. Instruction before the routine is executed (pre-routine instruction) and instruction within the routine follows. Location of this instruction is relevant to the individual's ability to execute the skills in the natural environment.

Steps described by the authors include prioritizing instructional objectives, providing instruction and assessing progress, and conducting performance assessment of routines following instruction. During the first step, educators choose teaching strategies to utilize with the student with specific setup, and teaching suggestions are outlined in the Program Manual. Instructional strategies are grouped into categories such as creating cues, creating pictures, fine and gross motor skills practice, modeling, picture storybook, role playing, social stories, tactile systems, and visual systems. Ongoing assessment occurs with direct observation data collection which indicates what strategies were used and what outcomes were achieved by the student. Assessments are conducted after several teaching sessions in a probe format. The final step involves a performance assessment of the student's performance on the routine. This is the fourth assessment of the routines following instruction and ongoing performance assessment.

Evaluation Phase

This phase involves determining whether to continue instruction or return to the assessment phase. The Program Manual provides guidelines for determining which decision to make. Continuing

with the instruction phase is warranted if the student has not met the criteria of mastery in the instructional phase. There are several conditions that can be used to terminate current instruction. These involve mastery of the routines and meeting the instructional goals of the student's individualized education plan that may not be at the level of independent functioning. That is, if a student has progressed by way of decreased dependence on teacher support, this may meet the goals set forth by the educational team. Finally, instruction may be discontinued in several instances, including student's lack of progress and environmental limitations that do not support ongoing instruction.

Efficacy Information

There are no empirical studies to date that outline the efficacy of FACTER in educational settings, although the Program Manual references some unpublished research. The Program Manual does provide data that was collected during field testing of FACTER in the state of Oregon through the Department of Education. Interrater reliability and test-retest reliability were reported with high correlation coefficients reported for elementary and secondary levels. These were somewhat lower for secondary scores and ratings between teachers and assessment team raters.

Concurrent validity was assessed with the Vineland Adaptive Behavior Scales – Interview Edition and correlations between teacher ratings on FACTER and actual student performance. Correlation coefficients in both cases were adequate.

Outcome Measurement

Outcomes on FACTER are assessed for each individual on the routines that are the target of instruction. Assessments indicate level of independent performance. Please see the "Treatment Procedures" section for additional information on the assessment and evaluation procedures of FACTER.

Qualifications of Treatment Providers

FACTER is intended for use by special educators in a school setting, although no specific recommendations are made for individuals who may administer the FACTER or implement the teaching procedures. While the authors provide the necessary tools to utilize the curriculum, they do offer training and technical assistance to users.

See Also

- ▶ [Criterion-Referenced Assessment](#)
- ▶ [Prompts](#)
- ▶ [Social Stories](#)
- ▶ [Task Analysis](#)
- ▶ [Visual Supports](#)

References and Reading

- Arick, J. R., Nave, G., Hoffman, T., & Krug, D. A. (2000). *FACTER program manual*. Austin: PRO-Ed.
- Arick, J., Nave, G., & Hoffman, T. (2002). Extended career and life role assessment system. Oregon Department of Education: Office of Assessment and Evaluation. Retrieved from http://www.ode.state.or.us/teachlearn/testing/samples/2002_03/extclarasadminman.pdf
- Bennett, K., Reichow, B., & Wolery, M. (2011). Effects of structured teaching on the behavior of young children with disabilities. *Focus on Autism and Other Developmental Disabilities*, 26(3), 143–152. Retrieved from EBSCOhost.
- Mays, N., & Heflin, L. (2011). Increasing independence in self-care tasks for children with autism using self-operated auditory prompts. *Research in Autism Spectrum Disorders*, 5(4), 1351–1357. <https://doi.org/10.1016/j.rasd.2011.01.017>.
- Stokes, J. V., Cameron, M. J., Dorsey, M. F., & Fleming, E. (2004). Task Analysis, correspondence training, and general case instruction for teaching personal hygiene skills. *Behavioral Interventions*, 19(2), 121–135.
- Zisimopoulos, D., Sigafoos, J., & Koutromanos, G. (2011). Using video prompting and constant time delay to teach an Internet search basic skill to students with intellectual disabilities. *Education and Training in Autism and Developmental Disabilities*, 46(2), 238–250. Retrieved from EBSCOhost.

Functional Assessment Screening Tool (FAST)

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Synonyms

FAST (functional assessment screening tool)

Description

The FAST is a type of indirect functional assessment method. It is comprised of a self-report checklist designed to identify whether maladaptive behavior is maintained via attention and tangibles, escape, sensory stimulation, or pain attenuation.

In addition to the checklist, the FAST is comprised of two information sections: informant-client relationship and problem behavior information. The informant-client relationship section contains four questions regarding the informant-client relationship (e.g., relationship to the client, how often and in what situations they interact). The problem behavior information section contains eight questions regarding the topography, frequency, and severity of the target behavior.

The checklist section contains 16 items. Each question is marked as either “yes,” “no,” or “not applicable.” Items endorsed with a “yes” are then categorized into one of four possible sources of reinforcers for problem behavior: attention/preferred items (social positive reinforcement), escape (social negative reinforcement), sensory stimulation (automatic positive reinforcement), and pain attenuation (automatic negative reinforcement). Behavioral function is inferred by calculating a summary score for the four possible maintaining variables. The FAST interview process is 10–15 min in length.

Historical Background

The FAST was originally developed in 1995, as an adjunct to functional analysis procedures. The scale underwent several revisions before the most recent version, which was released in 2005. The initial scale included 34 items. After field-testing the FAST with over 300 individuals, the item pool was reduced to the present 16 items.

Psychometric Data

Psychometric data for the FAST are generally sparse. Zaja et al. (2010) evaluated and compared the psychometric properties of the Questions About Behavioral Function (QABF) scale (Matson and Vollmer 1995), the Functional Assessment for Multiple Causality (FACT) scale (Matson et al. 2003), and the FAST for the indirect assessment of self-injurious, stereotypic, and aggressive/destructive behavior. The FAST subscales generally showed the lowest inter-rater agreement with correlations ranging from poor to good relative to the QABF and FACT. Zaja et al. also found that the FAST had unacceptably low internal consistency especially for the social attention and social escape subscales. The authors indicated that the weaker psychometrics properties (reliability and validity) for the FAST were likely related to its length, limited response format (“yes”/“no”/“NA”), and restricted range of subscales (4 instead of 5).

The FAST was evaluated by Iwata et al. (2013) for reliability and validity. Interrater reliability was assessed for 196 problem behaviors through independent administration of the assessment to pairs of raters. The average item-by-item agreement between pairs of raters was 71.5%, with a range of 53.3% to 84.5%. The authors also compared the results of the FAST to the results of 69 functional analyses. They concluded that the FAST predicted the primary function of challenging behavior 63.8% of the time. The authors noted that, while the results are comparable to other indirect assessment tools, they are not an adequate substitute for functional analysis procedures.

Clinical Uses

The FAST is an indirect functional assessment procedure. The purpose of the instrument is to provide preliminary information regarding the cause (of function) of maladaptive behavior. The information gathered from the FAST can be incorporated into direct assessment methods. Given the questionable reliability of most indirect assessment measures (such as interviews and rating scales), it is often recommended that these methods be employed in conjunction with direct observation and/or functional analysis procedures and not as the sole means to determine behavioral function (Zarcone et al. 1991).

See Also

► [Functional Behavior Assessment](#)

References and Reading

- Iwata, B., & DeLeon, I. (2005). *The functional analysis screening tool*. Gainesville: The Florida Center on Self-Injury, University of Florida.
- Iwata, B. A., DeLeon, I. G., & Roscoe, E. M. (2013). Reliability and validity of the functional analysis screening tool. *Journal of Applied Behavior Analysis*, 46(1), 271–284.
- Matson, J. L., & Vollmer, T. R. (1995). *The questions about behavioral function (QABF) user's guide*. Baton Rouge: Scientific Publishers.
- Matson, J. L., Kuhn, D. E., Dixon, D. R., Mayville, S. B., Laud, R. B., Cooper, C. L., Malone, C. J., Minshawi, N. F., Singh, A. V., Luke, M. A., Lott, J. D., & Matson, M. L. (2003). The development and factor structure of the functional assessment for multiple causality (FACT). *Research in Developmental Disabilities*, 24, 485–495.
- Zaja, R. H., Moore, L., van Ingen, D. J., & Rojahn, J. (2010). Psychometric comparison of the functional assessment instruments QABF, FACT and FAST for self-injurious, stereotypic and aggressive/destructive behaviour. *Journal of Applied Research in Intellectual Disabilities*, 24(1), 18–28.
- Zarcone, J. R., Rodgers, T. A., Iwata, B. A., Rourke, D. A., Dorsey, M. F. (1991). Reliability analysis of the motivation assessment scale: A failure to replicate. *Research in Developmental Disabilities*, 12(4), 349–360.

Functional Behavior Assessment

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Synonyms

[FBA](#); [Functional assessment](#)

Definition

Functional behavior assessment (FBA) procedures are used to investigate the maintaining variables associated with a target response and identify adaptive responses to be taught in order to allow the individual to better meet his or her needs. There are a variety of assessment procedures associated with the completion of a comprehensive FBA. These may be categorized into indirect and direct measures. Indirect procedures will capture archival information about the patient and allow those who are familiar with the individual and the target response to offer reports. Completion of indirect interviews and questionnaires allows investigators to capture information from many sources in a generally time-efficient fashion. The information gathered through indirect methods enables investigators to develop a comprehensive conceptualization of the target behavior for the assessment, as well as to capture information relevant to understanding the patient's needs, life context, learning history, motivators, and interpersonal support system. This information allows investigators to gain insight into common environmental variables associated with the target behavior, specifically including both high- and low-probability contexts. Indirect data collection tools will address environmental variables such as locations, times of day, individuals present, engagement, demand levels, access to preferred materials, and characteristics (e.g., sensory variables) of different

environments. Direct data collection is a required feature of any FBA. The observations must be guided by an operational definition of the target response for the assessment, and all observers must be trained to levels of acceptable reliability on the target. Direct data should be captured across multiple contexts in order to capture a sufficiently comprehensive data set for analysis. This typically includes observations across multiple days, times of day, and settings (e.g., high- and low-demand environments and high-, low-, and divided-attention environments). Antecedent behavior consequence (ABC) data are among the most commonly collected direct observation data when completing an FBA. These data require the observer to record information on the general environmental context and the exact changes that occurred immediately before and after the occurrence of the target response. The data are analyzed in order to determine common classifications of antecedent and consequence events associated with the target behavior. This information is then reviewed with all other sources of information collected during the assessment in order to develop a functional hypothesis of the maintaining variables for the challenging behavior and to map replacement skills to teach during intervention phases.

See Also

- ▶ [Analog Condition Functional Analysis](#)
- ▶ [Functional Analysis](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis*. Upper Saddle River: Prentice Hall.
- Gresham, F. M., Watson, T. S., & Skinner, C. H. (2001). Functional behavior assessment: Principles, procedures, and future directions. *School Psychology Review*, 30, 156–172.
- Love, J. R., Carr, J. E., & LeBlanc, L. A. (2009). Functional assessment of problem behavior in children with autism spectrum disorders: A summary of 32 outpatient cases. *Journal of Autism and Developmental Disorders*, 39, 363–372.
- O'Neill, R. E., Horner, R. H., Albin, R. W., Sprague, J. R., Storey, K., & Newton, J. S. (1996). *Functional assessment and program development for problem behavior: A practical handbook*. Florence: Wadsworth Publishing.
- Reese, R. M., Richman, D. M., Zarcone, J., & Zarcone, T. (2003). Individualizing functional assessment for children with autism: The contribution of perseverative behavior and sensory disturbances to disruptive behavior. *Focus on Autism and Other Developmental Disabilities*, 18, 89–94.

Functional Behavior-Based Cognitive-Behavioral Therapy for Obsessive-Compulsive Behavior in Children with ASD

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Definition

Functional behavior-based cognitive-behavioral therapy (CBT) for obsessive-compulsive behavior (OCB) in the pediatric population is a blending of two disciplines: (a) CBT with cognitive, social, and linguistic adaptations tailored to the specific needs of children and youth with autism spectrum disorder (ASD) and (b) functional behavior assessment and intervention derived from applied behavior analysis (ABA) to address perceived maintaining variables of target compulsions (e.g., positive social attention) that are beyond anxiety reduction. Working in combination, the two disciplines of CBT and ABA may allow for a more comprehensive treatment of overt and covert behaviors that comprise OCBs in children and youth with ASD.

Historical Background

In comparison to social-communicative challenges characteristic of ASD (DSM-5; American

Psychiatric Association 2013), research on restrictive and repetitive behaviors (RRBs) is limited. Factor analytic studies reveal two broad subtypes, including lower-order RRBs (e.g., self-injury, echolalia, motor stereotypy) and higher-order RRBs (e.g., washing rituals, rigid routines, arranging, and ordering). This limited research is troubling given that these behaviors may have negative effects on educational and social outcomes for individuals with ASD, are linked to increased parental stress, and often negatively impact family functioning (Boyd et al. 2012). Studies have used the Repetitive Behavior Scale-Revised (RBS-R; Bodfish et al. 1999) which is a 43-item parent report measure of RRB frequency and severity with good psychometric properties for individuals with ASD and related disabilities. Studies reveal preliminary support for higher-order RRBs (compulsive, ritualistic, and insistence on sameness behaviors) in ASD loading on the same factor (Mirenda et al. 2010), with many higher-order behaviors (e.g., arranging and ordering) showing topographic similarities to symptoms that are characteristic of obsessive-compulsive disorder (OCD, DSM-5; APA 2013). Studies acknowledge overlapping symptom presentation in individuals with ASD and OCD, with common phenotypic and genetic factors in these disorders (Stone and Chen 2016). OCD is defined by the presence of persistent thoughts, urges, or images that elicit anxiety and the overt display of repetitive behaviors or mental acts (compulsions) that an individual feels the need to perform to mitigate anxiety or in response to a rigid rule (APA 2013).

When conducting a diagnostic assessment of OCD in a child or youth with ASD, differentiating between higher-order repetitive behaviors related to ASD and symptoms that are characteristic of OCD can often be problematic. Given challenges in the ability to engage in expressive communication with others as well as limited self-reflection and introspection, it may be difficult for children and youth with ASD to identify and describe their obsessions and compulsions that is an integral part of standardized assessments such as the Child Yale-Brown Obsessive-Compulsive Scale (CY-BOCS; Goodman et al. 1986). The

CY-BOCS is a semi-structured interview comprised of 10 items and is used to rate symptom severity on a five-point ordinal scale. This measure has good psychometric properties for individuals who are 6–17 years old and otherwise typically developing. However, assessment tools such as the CY-BOCS (Goodman et al. 1986) are not specifically normed for children and youth with an ASD diagnosis. Also, different from OCD in typically developing individuals, research indicates that compulsive behaviors in ASD may or may not be strongly associated with obsessions, and strength of association may vary for any given compulsion (Ruzzano et al. 2015). Therefore, when customizing therapy to a given individual, it is of utmost importance that, in addition to diagnostic assessment tools, consideration be given to other assessment methods such as functional behavior assessment in order to identify motivating factors and maintaining variables beyond anxiety reduction such as positive sensory stimulation (automatic reinforcement) and social attention from others (Kose et al. 2018; Neil and Sturmey 2014; Vause et al. 2018). Given the acknowledged difficulty in differentiating between OCD symptoms and higher-order RRBs, the term obsessive-compulsive behavior (OCB) has been used by researchers to represent both phenomena (e.g., Chok and Harper 2016; Vause et al. 2018).

Given the early focus and popularity of using CBT to treat externalizing behaviors such as impulsivity, CBT for internalizing issues such as anxiety is considered the “second generation for CBT” (Kendall and Choudhury 2003, p. 89). Although CBT consists of a multitude of components, keystone strategies typically involve (a) cognitive training with an emphasis on cognitive restructuring techniques; (b) using behavioral principles to foster skill development with use of reinforcement; and (c) exposure-based strategies (Scarpa and Lorenzi 2013). Regarding pediatric anxiety and OCD, the two first-line treatments include CBT with exposure-based therapy and pharmacological treatment including selective serotonin reuptake inhibitors [SSRIs] (Rosa-Alcázar et al. 2015; Sánchez-Meca et al. 2014; Schwartz et al. 2019; Torp et al. 2015). With

18 studies that met their inclusion criteria, a meta-analysis conducted by Sánchez-Meca et al. (2014) for pediatric OCD examined the differential efficacy of treatments and showed a reduction in symptoms with a large effect size for CBT alone and CBT with pharmacological treatment, with effect sizes being significantly higher than pharmacological treatment alone.

Regarding the pediatric ASD population, randomized controlled trials (RCTs) evaluating treatment of anxiety disorders in children and youth with ASD have established the efficacy of CBT with cognitive, linguistic, and ASD-specific adaptations (e.g., social skills modules; Wood et al. 2009) to meet the needs of this population (Sukhodolsky et al. 2013). However, these studies focused on anxiety disorders (DSM-5; APA 2013), including a very small sampling of individuals with a primary diagnosis of OCD (which is now part of obsessive-compulsive and related disorders in DSM-5, APA 2013). Promisingly, in the last 15 years, there has been increased interest in evaluating psychosocial treatments for OCB in ASD. Limited research still exists regarding the use of pharmacological treatment such as SSRIs for anxiety disorders and OCD in the ASD population, and preliminary evidence suggests possible side effects such as impulsivity (Postorino et al. 2017). The present summary will cover (i) the current state of knowledge on CBT interventions for OCB with an emphasis on adaptations for this unique population, (ii) behavior analytic interventions based on functional behavior assessment to address OCBs, and, finally, (iii) the blending of adapted CBT with function-based behavior assessment and behavior analytic components for children and youth with ASD. Future directions will be discussed.

Current Knowledge

A series of CBT case studies follow a general CBT protocol for pediatric OCD developed by March and Mulle (1998) and other CBT experts. The main ingredient is exposure and response prevention (E/RP), where a youth is exposed to the internal and external stimuli associated with

anxiety and refrains from engaging in the compulsion resulting in habituation in the presence of those stimuli. E/RP along with traditional components such as psychoeducation and “bossing back OCD,” creating a hierarchy of least-to-most anxiety-provoking OCD symptoms, and teaching cognitive and behavioral skills were largely adapted from the work of March and Mulle. Tailoring the treatment to children and youth with high-functioning ASD, a number of case studies (e.g., Elliott and Fitzsimons 2014; Lehmkuhl et al. 2008; Reaven and Hepburn 2003) demonstrated clinically significant reductions in reported symptoms. Varied modifications were used to tailor treatment to each child or youth to meet his/her idiosyncratic needs. Specifically, emphasis was placed on adaptations such as increased use of visual stimuli, individualized story metaphors, self-monitoring of behavior, use of positive reinforcement such as verbal praise, a simplified cognitive component, and increased parent involvement (Kose et al. 2018).

Two studies compared a standard CBT protocol for OCBs in children and youth with and without ASD. One study used an open trial to compare standard group CBT for youth with OCD alone ($n = 23$) versus OCD and ASD [$n = 15$] (Farrell et al. 2012). Using the CY-BOCS (Goodman et al. 1986), the standard group CBT protocol showed that the protocol was equally effective for participants with and without ASD. In contrast, using similar outcome measures, a subsequent trial involving a case-controlled comparison (n 's = 22) indicated that standard CBT treatment was less effective for youth with ASD and OCD in comparison to when OCD was the sole diagnosis (Murray et al. 2015). Treating both anxiety disorders and OCBs in children and youth with ASD suggests that several adaptations are likely necessary to maximize CBT treatment success for this underserved population.

In addition to CBT studies, selected studies have used single-case experimental designs to illustrate the success of interventions based on behavior analytic principles to reduce OCBs in children and youth with ASD, including functional behavior assessment procedures. In these

studies, all participants had a diagnosis of ASD with an accompanying intellectual disability (ID). Behavior analysis is predicated on the understanding of motivating factors and environmental contingencies that maintain these learned behaviors. To date, a wealth of research provides empirical support for the use of functional behavior assessment methods to guide intervention decisions regarding behavior reduction and adaptive skill development in individuals with ASD and related disabilities (Hanley et al. 2003; Matson and Williams 2014; Mulligan et al. 2014). Although individuals with ASD may engage in compulsive behaviors to eliminate anxiety and distress, there may be additional factors operating such as a deprivation of attention from others (especially given challenges in social-communicative skills), seeking sensory stimulation to modulate arousal, or a need to escape an undesired task (Martin and Pear 2019; Vause et al. 2017). By assessing all possible variables that may be maintaining a particular behavior, an individualized comprehensive treatment plan can be devised.

Regarding OCBs, a small number of studies have used a wide variety of antecedent and consequent interventions to treat compulsive behaviors in children and youth with ASD and ID including ordering and arranging (e.g., Chok and Harper 2016; Rodriguez et al. 2012); rigid routines (Rispoli et al. 2014); and throwing away non-trash stimuli (Kuhn et al. 2009). For example, in a study by Rodriguez et al. (2012), a functional analysis was employed for one ordering and arranging behavior in each of three youth (13 to 15 years) with ASD and ID and revealed automatic positive (sensory) reinforcement as the maintaining variable. Intervention included access to competing stimuli combined with response blocking and, in one case, product extinction (returning furniture to its original position) that led to a reduction of OCBs to near-zero levels. In a different study, using a latency functional analysis with heart rate monitoring, Chok and Harper (2016) indicated automatic positive reinforcement as the maintaining variable for arranging items in a 12-year-old female with ASD, febrile seizures, and limited communication skills. Using a multiple schedule design, a

discrimination training procedure effectively addressed this compulsive behavior. Using the Chambless and Hollon (1998) criterion for empirically supported treatment, based on four single-case research designs including six participants conducted by four independent research teams, Neil and Sturmey (2014) reported that behavior analysis and behavior modification met criteria for *probably efficacious treatment* for OCBs in children with ASD. Targeting a wide range of OCBs including need to touch rituals, symmetry and exactness, repetitive questioning, and bathroom-related rituals, behavior analytic treatment included procedures such as functional communication training, differential reinforcement, token economies, response blocking, fading, and extinction.

Overall, CBT case studies with varying adaptations for children and youth with high-functioning ASD showed promising results in reducing OCBs, and nonrandomized group studies employing a standard CBT protocol showed mixed results. Single-case experimental designs evaluating assessment and treatment derived from ABA (including functional behavior assessment) have been successful in reducing OCBs in children and youth with ASD and ID. With increasingly rigorous research designs, the *combination* of adapted CBT and ABA elements (e.g., positive reinforcement, functional communication training) derived from functional behavior assessment methods has been explored.

A small number of case studies and single-case experimental designs have evaluated this combined approach (e.g., Boyd et al. 2013; Chok and Koesler 2014; Neil et al. 2017; Vause et al. 2014) with children and youth with ASD. With an emphasis on E/RP and behavior procedures, Boyd et al. (2013) attempted to lessen OCBs in 5- to 11-year-old children with ASD and ID. Using exposure trials and redirection to alternative tasks, Boyd and colleagues showed mixed results with some participants showing a marked change in duration and latency to engage in a respective OCB. In a study by Chok and Koesler (2014), a functional analysis was used with a 14-year-old male with ASD and severe ID for excessive cleaning behavior. In this case, functional analysis

results showed differing measures of heart rate when access to wiping was allowed versus when it was restricted. It was reported that when E/RP with response blocking was implemented, rates of cleaning reduced to near-zero levels.

Using a multi-component CBT with inclusion of functional behavior assessment, a small pilot group design (Vause et al. 2017) tested a manualized group functional behavior-based CBT (Fb-CBT) entitled *I Believe in Me, Not OCB!* (Vause et al. 2013) with children with high-functioning ASD. This manual built on the work of March and Mulle (1998) and other CBT experts (Piacentini et al. 2007a). Fourteen children with ASD ranged in age from 8 to 12 years, with seven children randomly assigned to the experimental condition and seven children to treatment as usual (TAU). Participants assigned to TAU continued with any services that they were currently accessing. Two primary outcome measures including the RBS-R (Bodfish et al. 1999) and the five-item compulsion subscale of the CY-BOCS (Goodman et al. 1986) demonstrated that this multimodal Fb-CBT treatment (e.g., psychoeducation, cognitive and behavioral skills training, and E/RP) with ABA components (including functional behavior assessment) significantly decreased OCBs at posttreatment, and results were maintained at 5-month follow-up, with medium to large effect sizes.

Vause et al. (2018), the first known RCT to treat OCBs in children and youth with high-functioning ASD, included 37 children (7–13 years) who were randomly assigned to the experimental group ($n = 19$) or TAU ($n = 18$). For those in the treatment condition, based on the extent of interference and distress in the youth's life and impact on family functioning, several OCBs were chosen and treated per participant over a 9-week period. Fb-CBT consisted of group-based activities, work in parent-child dyads, and parent coaching. Children were treated in small groups consisting of three or four children (with at least one parent) and two experienced therapists. Similar to CBT studies tailored to ASD, to maintain interest in the program, sustain attention, and foster comprehension skills, several adaptations were embedded including a

predetermined program plan with frequent repetition, presentation of material in various modalities (e.g., visual stimuli paired with auditory; 3D vs. 2D materials), incorporation of immediate and delayed reinforcers, inclusion of circumscribed interests, and hierarchically determined social skills exercises (e.g., greetings with fellow participants which progressed to playing interactive games).

The treatment program was comprised of two phases. The first phase (2.5 sessions) involved psychoeducation and mapping of *all* OCBs. This general phase focused on rapport building, psychoeducation about "OCB," operationally defining compulsions (and related obsessions when present) as well as triggers, using a fear thermometer to visually map out compulsions and determine a child's perceived control in resisting OCB (or performance of the compulsion), and creating externalizing statements (e.g., "Buzz off, OCB!"). Extending beyond discussing OCBs, similar to Reaven and Hepburn (2003), various concepts were introduced such as interference caused by obsessions and performing compulsions, and how these behaviors may impact the quality of life for the youth and his/her family members.

Following this general framework, in session 3, *individualized* work began for each compulsion in a graded fashion or based on a least-to-most hierarchy (March and Mulle 1998). Beginning in session 3, behavior analytic-based individualized treatment for each OCB was initiated with a functional behavior assessment to identify singular and multiple perceived functions of the targeted compulsive behaviors. For each OCB, an indirect assessment called the Questions About Behavior Function (QABF; Matson and Vollmer 1995) was used. The QABF is a parent rating questionnaire that shows good psychometric properties for the ASD population (Matson et al. 2012) and attempts to address functions including attention, nonsocial, tangible, and escape as well as physical pain/discomfort as an antecedent event. This assessment measure was supplemented with descriptive data conducted by a researcher trained in behavior analysis to analyze antecedents and consequences of perceived functions pertaining to each OCB.

Using these assessment methods in combination, a function-based assessment and intervention plan (FBAI) was derived to accompany the CBT approach.

The FBAI helped to identify alternative replacement skills that served the same function as the OCB. For example, consider an identified perceived function (beyond anxiety reduction) of “needing to tell” on siblings as social attention. In replacement of attending to the “need to tell” behavior, a parent was coached to minimize conversation around the telling behavior and to teach alternative behaviors such as conversational skills with prompts, social stories, or social script fading. Throughout this phase, participants learned key behavioral skills (e.g., functional communication, relaxation) that were appropriate to the targeted OCB and could also be generalized to other compulsions and different settings. Cognitive training existed on a spectrum where cognitive restructuring was attempted with a variety of modalities (e.g., verbally reframing thoughts, gathering facts, using visual representations to explain concepts) but like previous research (e.g., Lehmkuhl et al. 2008) was modified as needed based on the cognitive, linguistic, and interpersonal needs of the child. If there was an absence of a thought/obsession, focus was on simplified statements such as “I’m OK” or “I can beat this” (with engagement in an alternative behavior such as choosing a preferred activity or engaging in social conversation). Utilization of CBT skills training was encouraged when engaging in graded E/RP both in session and at home with positive reinforcement such as verbal praise and tangibles. Each of the nine sessions consisted of 30 min of parent coaching where parents learned the key concepts and procedures. Following parent coaching, they attempted to work with their child as independently as possible, with a therapist present to assist as needed.

Regarding general outcomes for this RCT evaluating the Fb-CBT treatment package for children and youth, the two primary outcome measures including the RBS (Bodfish et al. 1999) and the CY-BOCS compulsion subscale (Goodman et al. 1986) showed statistically significant differences between the experimental group and TAU with

large effect sizes, and these results were maintained at 6 months (Vause et al. 2018). Of note is that multiple OCBs were treated for each child ranging from 3 to 9 compulsions ($M = 6$). Secondary measures also examined level of functional impairment (e.g., Child Obsessive-Compulsive Impact Scale-Revised; Piacentini et al. 2007b) with the experimental group faring significantly better than TAU, and parents were generally satisfied with the treatment which supports the social validity of Fb-CBT.

The summarized literature demonstrates that using adapted CBT shows promise in successfully treating OCBs in children and youth with ASD. Combining adapted CBT with the discipline of ABA and, specifically, inclusion of functional behavior assessment may provide a more comprehensive clinical picture that aids in maximizing the reduction of OCBs and improving quality of life for individuals and their families.

Future Directions

Regarding assessment and treatment of OCBs in children and youth with ASD, there are several areas that require further attention and exploration. First, standardized assessment tools that are normed for an ASD pediatric population need to be developed. Whether used diagnostically or as a means of determining severity and interference of OCBs, given the acknowledged lack of introspection and often limited vocal communication in children and youth with ASD, future research may examine simplifying existing standardized interviews or combining interview questions with direct and/or self-observation, where possible.

In the RCT study (Vause et al. 2018), an indirect assessment with analysis of descriptive observational (antecedent-behavior-consequence) data was collectively used to determine perceived functions of OCBs. Through experimental manipulation of variables, functional analysis targets behavioral functions (Cooper et al. 2019). However, when treating multiple OCBs, it may be impractical to conduct multiple functional analyses per participant, and given the covert nature

of obsessions, experimental conditions may be difficult to design and repeatedly implement. However, future research may examine brief functional analyses and the use of physiological measures such as heart rate monitoring (Chok and Koesler 2014).

Although studies report promising results, additional randomized controlled trials are sorely needed to firmly establish the efficacy of adapted CBT alone as well as the additive value of combining CBT with ABA-based procedures including functional behavior assessment for children and youth. Addressing *all* maintaining variables in a treatment package derived from function-based assessment may quicken treatment response for OCBs. Typically, CBT studies included high-functioning individuals, whereas ABA studies included children and youth with ASD and an ID. Future research is needed with children and youth who present with a wide range of cognitive and adaptive levels to evaluate ideal treatment modalities, including psychosocial and pharmacological treatment. Research is needed to tease out key treatment components that are evidence-based for varying populations. Studies involving component analyses may aid in determining active ingredients for a range of ASD presentations, including children with ASD alone as well as those presenting with comorbid conditions such as attention deficit hyperactivity disorder (ADHD), comorbid anxiety, and varying neurodevelopmental disorders (DSM-5, APA 2013; Postorino et al. 2017). Also, examination of sociocultural variables (e.g., socioeconomic status, cultural values) is crucial in designing treatment protocols that are person- and family-centered to meet the needs of youth and their families.

Evaluation of CBT and combined treatments is also needed across varying age ranges. For example, only a handful of case studies and one RCT (Russell et al. 2013) have evaluated CBT to treat OCBs in older adolescent and adult populations with ASD. In this study, 46 adolescents and adults with a mean age of 27 years were randomized to CBT versus an anxiety management condition, with both groups showing significant reductions in OCBs. Extension is also needed to younger populations, with an emphasis on early

intervention for children of preschool age presenting with ASD and other related disabilities (Guertin et al. 2019).

To date, published research shows promise, but as suggested above, additional research evaluating singular and dual treatment modalities including Fb-CBT, with consideration of variables including level of cognitive functioning, comorbid presentations, cultural factors, and various age ranges, is needed. A focus of future studies should be on the establishment of a strong evidence base for treating obsessive-compulsive behavior with manualized packages such as Fb-CBT that are tailored to the needs of individuals across the ASD spectrum.

See Also

- Anxiety
- Autism Spectrum Addendum to the Anxiety Disorders Interview Schedule-Parent Interview
- Applied Behavior Analysis (ABA)
- Cognitive Behavioral Therapy (CBT)
- DSM-5 and Autism Spectrum Disorder
- Functional Analysis
- Functional Behavior Assessment
- Habit Reversal
- Hoarding in Youth with Autism Spectrum Disorders
- Mental Health and ASD
- Obsessive-Compulsive Disorder (OCD)
- Repetitive Behavior
- Restricted Interest

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- Bodfish, J. W., Symons, F. J., & Lewis, M. H. (1999). *The repetitive behavior scale: Test manual*. Morganton: Western Carolina Center Research Reports.
- Boyd, B. A., McDonough, S. G., & Bodfish, J. W. (2012). Evidence-based behavioral interventions for repetitive behaviors in autism. *Journal of Autism and Developmental Disorders*, 42(6), 1236–1248. <https://doi.org/10.1007/s10803-011-1284-z>.
- Boyd, B. A., Woodard, C. R., & Bodfish, J. W. (2013). Feasibility of exposure response prevention to treat

- repetitive behaviors of children with autism and an intellectual disability: A brief report. *Autism*, 17(2), 196–204. <https://doi.org/10.1177/1362361311414066>.
- Chambless, D. L., & Hollon, S. D. (1998). Defining empirically supported therapies. *Journal of Consulting and Clinical Psychology*, 66(1), 7–18. <https://doi.org/10.1037/0022-006X.66.1.7>.
- Chok, J. T., & Harper, J. M. (2016). Heart rate assessment and use of a multiple schedule treatment for an individual with obsessive compulsive-like behavior. *Journal of Developmental and Physical Disabilities*, 28(6), 821–834. <https://doi.org/10.1007/s10882-016-9511-3>.
- Chok, J. T., & Koesler, B. (2014). Distinguishing obsessive-compulsive behavior from stereotypy. *Behavior Modification*, 38(3), 344–373. <https://doi.org/10.1177/0145445513509475>.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2019). *Applied behavior analysis* (3rd ed.). Hoboken: Pearson Education.
- Elliott, S. J., & Fitzsimons, L. (2014). Modified CBT for treatment of OCD in a 7-year-old boy with ASD: A case report. *Journal of Child and Adolescent Psychiatric Nursing*, 27(3), 156–159. <https://doi.org/10.1111/jcap.12081>.
- Farrell, L., Waters, A., Milliner, E., & Ollendick, T. (2012). Comorbidity and treatment response in pediatric obsessive-compulsive disorder: A pilot study of group cognitive-behavioral treatment. *Psychiatry Research*, 199(2), 115–123. <https://doi.org/10.1016/j.psychres.2012.04.035>.
- Goodman, W. K., Price, L. H., Rasmussen, S. A., Riddle, M. A., & Rapoport, J. L. (1986). *Children's Yale-Brown obsessive compulsive scale (CY-BOCS)*. Bethesda: National Institute of Mental Health.
- Guertin, E. L., Vause, T., Jaksic, H., Frijters, J. C., & Feldman, M. (2019). Treating obsessive compulsive behavior and enhancing peer engagement in a preschooler with intellectual disability. *Behavioral Interventions*, 34(1), 19–29. <https://doi.org/10.1002/bin.1646>.
- Hanley, G. P., Iwata, B. A., & McCord, B. E. (2003). Functional analysis of problem behavior: A review. *Journal of Applied Behavior Analysis*, 36(2), 147–185. <https://doi.org/10.1901/jaba.2003.36-147>.
- Kendall, P. C., & Choudhury, M. S. (2003). Children and adolescents in cognitive-behavioral therapy: Some past efforts and current advances, and the challenges in our future. *Cognitive Therapy and Research*, 27, 89. <https://doi.org/10.1023/A:1022542814822>.
- Kose, L. K., Fox, L., & Storch, E. A. (2018). Effectiveness of cognitive behavioral therapy for individuals with autism spectrum disorders and comorbid obsessive-compulsive disorder: A review of the research. *Journal of Developmental and Physical Disabilities*, 30(1), 69–87. <https://doi.org/10.1007/s10882-017-9559-8>.
- Kuhn, D. E., Hardesty, S. L., & Sweeney, N. M. (2009). Assessment and treatment of excessive straightening and destructive behavior in an adolescent diagnosed with autism. *Journal of Applied Behavior Analysis*, 42(2), 355–360. <https://doi.org/10.1901/jaba.2009.42-355>.
- Lehmkuhl, H. D., Storch, E. A., Bodfish, J. W., & Geffken, G. R. (2008). Brief report: Exposure and response prevention for obsessive compulsive disorder in a 12-year-old with autism. *Journal of Autism and Developmental Disorders*, 38(5), 977–981. <https://doi.org/10.1007/s10803-007-0457-2>.
- March, J. S., & Mulle, K. (1998). *OCD in children and adolescents: A cognitive-behavioral treatment manual*. New York: The Guilford Press.
- Martin, G., & Pear, J. J. (2019). *Behavior modification: What it is and how to do it* (11th ed.). New York: Routledge.
- Matson, J. L., & Vollmer, T. R. (1995). *User's guide: Questions about Behavioral function (QABF)*. Baton Rouge: Scientific Publishers.
- Matson, J. L., & Williams, L. W. (2014). Functional assessment of challenging behavior. *Current Developmental Disorders Reports*, 1(2), 58–66. <https://doi.org/10.1007/s40474-013-0006-y>.
- Matson, J. L., Tureck, K., & Rieske, R. (2012). The questions about Behavioral function (QABF): Current status as a method of functional assessment. *Research in Developmental Disabilities*, 33(2), 630–634. <https://doi.org/10.1016/j.ridd.2011.11.006>.
- Mirenda, P., Smith, I. M., Vaillancourt, T., Georgiades, S., Duku, E., Szatmari, P., . . . Zwaigenbaum, L. (2010). Validating the repetitive behavior scale-revised in young children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 40(12), 1521–1530. <https://doi.org/10.1007/s10803-010-1012-0>.
- Mulligan, S., Healy, O., Lydon, S., Moran, L., & Foody, C. (2014). An analysis of treatment efficacy for stereotyped and repetitive behaviors in autism. *Review Journal of Autism and Developmental Disorders*, 1(2), 143–164. <https://doi.org/10.1007/s40489-014-0015-8>.
- Murray, K., Jassi, A., Mataix-Cols, D., Barrow, F., & Krebs, G. (2015). Outcomes of cognitive behaviour therapy for obsessive-compulsive disorder in young people with and without autism spectrum disorders: A case controlled study. *Psychiatry Research*, 228(1), 8–13. <https://doi.org/10.1016/j.psychres.2015.03.012>.
- Neil, N., & Sturmey, P. (2014). Assessment and treatment of obsessions and compulsions in individuals with autism spectrum disorders: A systematic review. *Review Journal of Autism and Developmental Disorders*, 1(1), 62–79. <https://doi.org/10.1007/s40489-013-0006-1>.
- Neil, N., Vause, T., Jaksic, H., & Feldman, M. (2017). Effects of group functional behavior-based cognitive-behavioral therapy for obsessive-compulsive behavior in a youth with autism spectrum disorder. *Child & Family Behavior Therapy*, 39(3), 179–190. <https://doi.org/10.1080/07317107.2017.1338448>.
- Piacentini, J., Langley, A., & Roblek, T. (2007a). *Cognitive-behavioral treatment of childhood OCD*:

- It's only a false alarm, therapist guide. Programs that work.* New York: Oxford University Press.
- Piacentini, J., Peris, T. S., Bergman, R. L., Chang, S., & Jaffer, M. (2007b). BRIEF REPORT: Functional impairment in childhood OCD: Development and psychometrics properties of the child obsessive-compulsive impact scale-revised (COIS-R). *Journal of Clinical Child & Adolescent Psychology*, 36(4), 645–653. <https://doi.org/10.1080/15374410701662790>.
- Pistorino, V., Kerns, C. M., Vivanti, G., Bradshaw, J., Siracusano, M., & Mazzone, L. (2017). Anxiety disorders and obsessive-compulsive disorder in individuals with autism spectrum disorder. *Current Psychiatry Reports*, 19(12), 92. <https://doi.org/10.1007/s11920-017-0846-y>.
- Reaven, J., & Hepburn, S. (2003). Cognitive-behavioral treatment of obsessive-compulsive disorder in a child with Asperger syndrome. *Autism*, 7(2), 145–164. <https://doi.org/10.1177/1362361303007002003>.
- Rispoli, M., Camargo, S., Machalicek, W., Lang, R., & Sigafoos, J. (2014). Functional communication training in the treatment of problem behavior maintained by access to rituals. *Journal of Applied Behavior Analysis*, 47(3), 580–593. <https://doi.org/10.1002/jaba.130>.
- Rodriguez, N. M., Thompson, R. H., Schlichenmeyer, K., & Stocco, C. S. (2012). Functional analysis and treatment of arranging and ordering by individuals with autism spectrum disorder. *Journal of Applied Behavior Analysis*, 45(1), 1–22. <https://doi.org/10.1901/jaba.2012.45-1>.
- Rosa-Alcázar, A. I., Sánchez-Meca, J., Rosa-Alcázar, Á., Iniesta-Sepúlveda, M., Olivares-Rodríguez, J., & Parada-Navas, J. L. (2015). Psychological treatment of obsessive-compulsive disorder in children and adolescents: A meta-analysis. *The Spanish Journal of Psychology*, 18, E20. <https://doi.org/10.1017/sjp.2015.22>.
- Russell, A. J., Jassi, A., Fullana, M. A., Mack, H., Johnston, K., Heyman, I., . . . Mataix-Cols, D. (2013). Cognitive behavior therapy for comorbid obsessive-compulsive disorder in high-functioning autism spectrum disorders: A randomized controlled trial. *Depression and Anxiety*, 30(8), 697–708. <https://doi.org/10.1002/da.22053>.
- Ruzzano, L., Borsboom, D., & Geurts, H. M. (2015). Repetitive behaviors in autism and obsessive-compulsive disorder: New perspectives from a network analysis. *Journal of Autism and Developmental Disorders*, 45(1), 192–202. <https://doi.org/10.1007/s10803-014-2204-9>.
- Sánchez-Meca, J., Rosa-Alcázar, A. I., Iniesta-Sepúlveda, M., & Rosa-Alcázar, Á. (2014). Differential efficacy of cognitive-behavioral therapy and pharmacological treatments for pediatric obsessive-compulsive disorder: A meta-analysis. *Journal of Anxiety Disorders*, 28(1), 31–44. <https://doi.org/10.1016/j.janxdis.2013.10.007>.
- Scarpa, A., & Lorenzi, J. (2013). Cognitive-Behavioral therapy with children and adolescents: History and principles. In A. Scarpa, S. W. White, & T. Attwood (Eds.), *CBT for children and adolescents with high-functioning autism Spectrum disorders* (pp. 1–26). New York: Guilford Press.
- Schwartz, C., Barican, J. L., Yung, D., Zheng, Y., & Waddell, C. (2019). Six decades of preventing and treating childhood anxiety disorders: A systematic review and meta-analysis to inform policy and practice. *Evidence Based Mental Health*, 22(3), 103–110. <https://doi.org/10.1136/ebmental-2019-300096>.
- Stone, W. S., & Chen, G. (2016). Comorbidity of autism spectrum and obsessive-compulsive disorders. *North American Journal of Medicine and Science*, 8(3), 109–112. <https://doi.org/10.7156/naajms.2015.0803109>.
- Sukhodolsky, D. G., Bloch, M. H., Panza, K. E., & Reichow, B. (2013). Cognitive-behavioral therapy for anxiety in children with high-functioning autism: A meta-analysis. *Pediatrics*, 132(5), e1341–e1350. <https://doi.org/10.1542/peds.2013-1193>.
- Torp, N. C., Dahl, K., Skarphedinsson, G., Compton, S., Thomsen, P. H., Weidle, B., . . . Ivarsson, T. (2015). Predictors associated with improved cognitive-behavioral therapy outcome in pediatric obsessive-compulsive disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 54(3), 200–207.e1. <https://doi.org/10.1016/j.jaac.2014.12.007>.
- Vause, T., Neil, N., Jaksic, H., & Feldman, M. (2013). *I believe in me, not OCB!: Clinician's manual*. St. Catharines: Brock University.
- Vause, T., Hoekstra, S., & Feldman, M. (2014). Evaluation of individual function-based cognitive-behavioural therapy for obsessive compulsive behaviour in children with autism spectrum disorder. *Journal on Developmental Disabilities*, 20(3), 30–41. Retrieved from http://ovidsp.ovid.com/ovidweb.cgi?T=JS&CSC=Y&NEWS=N&PAGE=fulltext&D=psyc11&AN=2015-00447-003%5Cn; http://sfx.nottingham.ac.uk:80/sfx_local?genre=article&atitle=Evaluation+of+individual+function-based+cognitive-behavioural+therapy+for+obsessive+compulsive+b
- Vause, T., Neil, N., Jaksic, H., Jackiewicz, G., & Feldman, M. (2017). Preliminary randomized trial of function-based cognitive-behavioral therapy to treat obsessive compulsive behavior in children with autism spectrum disorder. *Focus on Autism and Other Developmental Disabilities*, 32(3), 218–228. <https://doi.org/10.1177/1088357615588517>.
- Vause, T., Jaksic, H., Neil, N., Frijters, J. C., Jackiewicz, G., & Feldman, M. (2018). Functional behavior-based cognitive-behavioral therapy for obsessive compulsive behavior in children with autism spectrum disorder: A randomized controlled trial. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-018-3772-x>.
- Wood, J., Drahota, A., Sze, K., Har, K., Chiv, A., & Langer, D. (2009). Cognitive behavioral therapy for anxiety in children with autism spectrum disorders: A randomized, controlled trial. *Journal of Child Psychology and Psychiatry*, 50, 224–234.

Functional Communication Training

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Definition

Functional communication training (FCT) is a systematic approach to intervention in which a more socially appropriate behavior is taught to replace a challenging behavior (Durand 1990; Durand and Merges 2008, 2009; Prelock et al. 2011). It is presumed that the challenging behavior is an attempt to communicate; therefore, vocalizations, manual signs and gestures, or graphic symbols are used to replace the less desirable behaviors. Notably, FCT is used in combination with other interventions (Durand 1999; Olive et al. 2008).

Historical Background

Challenging behavior is a frequent occurrence for individuals with ASD, particularly when they do not possess the speech and language skills required to communicate their wants and needs. These behaviors may include self-injury, tantrums, aggression, etc. Research suggests that individuals with disabilities, and particularly those with ASD, are more likely to exhibit problem behaviors that are frequent, severe, and sustained over time. These behaviors interfere with family life and make it difficult for individuals with ASD to be integrated in their community. Further, such behavior problems are a strong predictor of school failure (Feldman et al. 2007). Therefore, behavioral strategies have been established to address these behavior problems. Durand, Carr, Dunlap, and colleagues recognized that these challenging behaviors are often a result of an inability to communicate intent including an

individual's wants, needs, and desires (Carr and Durand 1985; Dunlap et al. 1991; Durand 1990; Durand and Merges 2008, 2009). FCT was designed as an intervention technique to address the reported challenging behaviors in individuals with ASD and other developmental disabilities by replacing the undesired behaviors with an alternative that is based on the perceived and assessed intent of the behavior. A variety of professionals (e.g., psychologists, speech-language pathologists, behavioral interventionists) and families use FCT across age, language skills, and developmental abilities in several contexts (Dunlap et al. 2006; Durand 1999; Mancil 2006; Petscher et al. 2009). FCT has strong support in the literature as an established skill-based intervention (Smith et al. 2007).

Rationale or Underlying Theory

There are several concepts that provide a theoretical framework for understanding why FCT is an effective intervention for reducing challenging behavior. Three concepts have been identified as crucial to understanding FCT and implementing it with a diverse population: functional equivalence, natural communities of reinforcement, and features of the alternative response (Carr 1988; Durand 1987, 1990, 2012; Durand and Merges 2009).

Functional equivalence is used to explain how and why behavior changes in FCT. Assumptions are made about why a behavior occurs such as being reinforced by getting the attention of another or escaping from doing something. If this is true, then these challenging behaviors could be adjusted by offering an alternative or new behavior that achieves the same function. Thus, FCT reduces problem behavior because it involves teaching and reinforcing a replacement behavior that serves the same function.

Using a communication strategy as a replacement for challenging behavior has an added advantage of being able to capitalize on "natural communities of reinforcement" (Durand 1990). This means an individual learns to ask for a reinforcer from a variety of communication partners,

and those partners do not require specific training from an interventionist.

A third component underlying the conceptual framework of FCT that is important to the success of this intervention is the opportunity for creating an alternative response (Durand and Merges 2009). An alternative response is one that is functionally equivalent to the challenging behavior and includes several features like how closely it matches the function of the behavior, how successful and efficient it is likely to be, how recognizable and acceptable it is as a response, and how it works in the natural environment (Durand 2012).

An important principle in understanding the approach and value of FCT is that behavior problems are not viewed as undesirable occurrences that must be eliminated. Instead, challenging behaviors are viewed as functionally equivalent to other modes of communication. This view recognizes that although the behaviors may be unacceptable, what is being communicated is not (e.g., getting attention, asking for help). This is a critical concept because the intervention goal changes. Instead of eliminating the behavior and providing no alternative for accessing what an individual needs or wants, an alternative and more acceptable communication behavior is taught so the desired reinforcer can be achieved.

Goals and Objectives

FCT is primarily designed to address a range of challenging behaviors that interfere with an individual's ability to fully participate in learning, and family and community life. In fact, parental stress is known to increase when parents are dealing with a child exhibiting challenging behaviors (Floyd and Gallagher 1997; Hastings 2002; Saloviita et al. 2003). Appropriate targets may include self-injury, aggression, tantruming, stereotypies, and inappropriate sexual behavior, to name a few (Fyffe et al. 2004).

Treatment Participants

FCT can be implemented for anyone who displays problematic behavior. It is an appropriate strategy

to address the behavior challenges reported for individuals with ASD as well as other disorders (e.g., ADHD, schizophrenia, traumatic brain injury) in which behavior problems are a frequent occurrence (Durand and Merges 2009). Research also suggests that FCT can be used to address the difficult behaviors of individuals of all ages with a range of language and cognitive abilities (Bird et al. 1989; Mancil 2006; Petscher et al. 2009).

Treatment Procedures

FCT requires the implementation of three primary steps: (1) assessing the function of the challenging behavior to be addressed, (2) selecting an appropriate alternative to the challenging behaviors, and (3) teaching the alternative response and fading prompts to use the alternative response in the environment.

To begin FCT, the interventionist identifies the antecedents (what comes before) and consequences (what follows) of a problem behavior in an effort to assess the function the behavior serves. When the function is determined, individuals are taught to make a request in a more acceptable manner. Selecting an alternative communication modality to make this request is a critical next step. Consideration is given to those modes of communication the individual has already demonstrated some skill (e.g., signing, graphic symbols, writing, verbalization).

Once an alternative mode of communication is identified, teaching situations are created. First, the environment is arranged to provide multiple opportunities for using the new communication strategy. Situations are established that are most likely to elicit the interest of the individual. Training opportunities are interspersed through the day in the environment where the individual is expected to communicate. This approach increases the likelihood of intervention effects that are generalized and maintained.

Several language training techniques are required to teach individuals to communicate their intent using an acceptable means as a replacement for their challenging behavior (Durand et al. 1999). Prompts are used to teach

the new response and then are faded as quickly as possible. Prompts may be full physical prompts or partial physical, gestural, or verbal prompts. Delayed prompting is also used to help fade the prompting and encourage spontaneous use of the desired communication behavior (Halle et al. 1981; Schwartz et al. 1989). Once an individual demonstrates success in using the new communicative behavior in three of five consecutive opportunities, prompt fading begins to eliminate prompt dependency.

The context for training occurs on a continuum from more artificial settings like a therapy room to more natural settings like the community. The context is largely dependent on where the individual is expected to use the new trained communication response. If training begins in a more restricted setting like a therapy room, once the response is learned, performance should be encouraged in the settings where the behavior is expected to occur to ensure generalization and maintenance.

Efficacy Information

There are more than 200 studies documenting the effectiveness of FCT for addressing challenging behaviors for a range of individuals (Durand and Merges 2008, 2009; Mancil 2006; Mirenda 1997; Petscher et al. 2009). Research also suggests that FCT successfully decreases problematic behavior and increases communication in individuals with ASD. Some of the communicative responses that have been successfully taught as a replacement to difficult behaviors include mands (O'Neill and Sweetland-Baker 2001) and augmentative and alternative communication (AAC) strategies such as signing, communication books, and voice output devices (Mirenda 1997). The use of these replacement behaviors has led to increased spontaneous and appropriate verbal communication (Mancil 2006) that is maintained and generalized to untrained activities (Olive et al. 2008; Wacker et al. 2005).

Petscher et al. (2009) reviewed over 80 studies describing FCT as a treatment to address problematic behaviors and found it met their

preestablished criteria for a well-established intervention. This supports the findings of the National Standards Project (2009) which also identified FCT as an established behavioral intervention that has successfully decreased problem behaviors, restricted repetitive behaviors, and increased academic, communication, and social behaviors in individuals with autistic disorder and pervasive developmental disorder – not otherwise specified ranging in age from very young to 21 years.

Although there is extensive research examining the effectiveness of FCT, most studies use single-subject designs with small numbers of participants. Some larger studies do exist (e.g., Durand and Carr 1992; Wacker et al. 1998), but no randomized clinical trials have been reported that compare treatments or outcomes to no treatment.

Durand and Rost (2005) suggest that there is some question about the representativeness of the literature examining behavioral interventions including FCT. They suggest it is unclear if the current intervention research systematically excludes certain individuals which would influence the results and applications to specific populations.

Outcome Measurement

Functional behavioral assessment (FBA) is a key element in the implementation of FCT (Durand 1990). FBA is used to determine the function of problematic behavior that eventually leads to selecting a more appropriate alternative communication behavior. The selection of the alternative behavior must serve the same function as the challenging behavior.

Informal observations, antecedent-behavior-consequence (ABC) charts, functional analyses, and a variety of rating scales are frequently used to determine the function of a behavior. Functional analysis is often identified as the best method to determine the function of a behavior. It is a process by which aspects of the environment are manipulated to assess behavior change (Hanley et al. 2003), although there are challenges in gaining accessibility to manipulate the

environment. There are also some influences that may not be conducive to manipulation.

Assessment typically begins with interviews of those who know the individual best as well as informal observations. Once this information is gathered, more formal assessments are completed to validate the impressions made from the informal assessment. Available instruments include the *Motivation Assessment Scale*, the *Motivation Analysis Rating Scale*, the *Functional Analysis Interview Form*, the *Functional Assessment Checklist*, the *Functional Analysis Checklist*, and the *Questions About Behavioral Function*.

Data collection requires establishing goals, analyzing behavior patterns, assessing environmental obstacles, and monitoring results (Durand and Hieneman 2008a, b). To establish goals, there needs to be sufficient information to define the problem and determine the desired responses across settings. Different forms of functional assessment are then employed to analyze behavior patterns. The learning environment is then examined to identify what alternative communicative responses could be supported. Those responsible for collecting data are then identified and trained to tally responses (Durand and Hieneman 2008a, b).

Qualifications of Treatment Providers

Individuals who implement FCT are usually highly trained in behavioral strategies and recognize the role of behavior in communication. FCT is best implemented using a team approach in collaboration with a psychologist who has experience with functional behavior assessment and a speech-language pathologist who can help to identify the function and appropriate replacements for expressing communicative intent considering an individual's cognitive and communication abilities. Children with ASD often require a behavioral plan, so once this is developed, training needs to occur for all those who come into contact with the individual who is exhibiting challenging behavior. Adequate training in behavioral principles is necessary to ensure that regression does not occur and behavior problems do not increase (Durand and Carr 1991, 1992).

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Communication Assessment](#)
- [Communication Interventions](#)
- [Functional Analysis](#)
- [Functional Behavior Assessment](#)
- [Functional Goals](#)

References and Reading

- Bird, F., Dores, P. A., Moniz, D., & Robinson, J. (1989). Reducing severe aggressive and self-injurious behaviors with functional communication training. *American Journal on Mental Retardation*, 94(1), 37–48.
- Carr, E. G. (1988). Functional equivalence as a mechanism of response generalization. In R. H. Horner, G. Dunlap, & R. L. Koegel (Eds.), *Generalization and maintenance: Lifestyle changes in applied settings* (pp. 194–219). Baltimore: Paul H. Brookes.
- Carr, E. G., & Durand, V. M. (1985). Reducing behavior problems through functional communication training. *Journal of Applied Behavior Analysis*, 18(2), 111–126.
- Dunlap, G., Kern-Dunlap, L., Clarke, S., & Robbins, F. R. (1991). Functional assessment, curricular revision, and severe behavior problems. *Journal of Applied Behavior Analysis*, 24, 387–397.
- Dunlap, G., Ester, T., Langhans, S., & Fox, L. (2006). Functional communication training with toddlers in home environments. *Journal of Early Intervention*, 28(2), 81–96.
- Durand, V. M. (1987). “Look homeward angel:” A call to return to our (functional) roots. *The Behavior Analyst*, 10, 299–302.
- Durand, V. M. (1990). *Severe behavior problems: A functional communication training approach*. New York: Guilford Press.
- Durand, V. M. (1999). Functional communication training using assistive devices: Recruiting natural communities of reinforcement. *Journal of Applied Behavior Analysis*, 32(3), 247–267.
- Durand, M. (2012). Functional communication training: Treating challenging behavior. In P. A. Prelock & R. J. McCauley (Eds.), *Treatment of autism spectrum disorders: Evidence-based intervention strategies for communication and social interaction* (pp. 107–138). Baltimore: Paul H Brookes.
- Durand, V. M., & Carr, E. G. (1991). Functional communication training to reduce challenging behavior: Maintenance and application in new settings. *Journal of Applied Behavior Analysis*, 24(2), 251–264.
- Durand, V. M., & Carr, E. G. (1992). An analysis of maintenance following functional communication training. *Journal of Applied Behavior Analysis*, 25(4), 777–794.

- Durand, V. M., & Hieneman, M. (2008a). *Helping parents with challenging children: Positive family intervention: Facilitator guide*. New York: Oxford University Press.
- Durand, V. M., & Hieneman, M. (2008b). *Helping parents with challenging children: Positive family intervention, workbook*. New York: Oxford University Press.
- Durand, V. M., & Merges, E. (2008). Functional communication training to treat challenging behavior. In W. O'Donohue & J. E. Fisher (Eds.), *Cognitive behavior therapy: Applying empirically supported techniques in your practice* (2nd ed., pp. 222–229). New York: Wiley.
- Durand, V. M., & Merges, E. (2009). Functional communication training to treat challenging behavior. In W. O'Donohue & J. E. Fisher (Eds.), *General principles and empirically supported techniques of cognitive behavior therapy* (pp. 320–327). New York: Wiley.
- Durand, V. M., & Rost, N. (2005). Does it matter who participates in our studies? A caution when interpreting the research on positive behavioral support. *Journal of Positive Behavior Interventions*, 7, 186–188.
- Durand, V. M., Mapstone, E., & Youngblade, L. (1999). The role of communicative partners. In J. Downing (Ed.), *Teaching communication skills to students with severe disabilities within general education classrooms* (pp. 139–155). Baltimore: Paul H. Brookes.
- Feldman, M., McDonald, L., Serbin, L., Stack, D., Secco, M. L., & Yu, C. T. (2007). Predictors of depressive symptoms in primary caregivers of young children with or at risk for developmental delay. *Journal of Intellectual Disability Research*, 51(8), 606–619.
- Floyd, F. J., & Gallagher, E. M. (1997). Parental stress, care demands and use of support services for school age children with disabilities and behavior problems. *Family Relations*, 46(4), 359–371.
- Fyffe, C. E., Kahng, S., Fittro, E., & Russell, D. (2004). Functional analysis and treatment of inappropriate sexual behavior. *Journal of Applied Behavior Analysis*, 37(3), 401–404.
- Halle, J. W., Baer, D., & Spradlin, J. (1981). Teachers' generalized use of delay as a stimulus control procedure to increase language use in handicapped children. *Journal of Applied Behavior Analysis*, 14, 389–409.
- Hanley, G. P., Iwata, B. A., & McCord, B. E. (2003). Functional analysis of problem behavior: A review. *Journal of Applied Behavior Analysis*, 36, 147–185.
- Hastings, R. P. (2002). Parental stress and behavior problems of children with developmental disability. *Journal of Intellectual and Developmental Disability*, 27(3), 149–160.
- Mancil, G. R. (2006). Functional communication training: A review of the literature related to children with autism. *Education and Training in Developmental Disabilities*, 41(3), 213–224.
- Miranda, P. (1997). Supporting individuals with challenging behavior through functional communication training and AAC: Research review. *Augmentative and Alternative Communication*, 13, 207–225.
- National Autism Center (2009). National Standards Project, Phase 1: Addressing the need for evidence-based practice guidelines for ASD. (www.nationalautismcenter.org).
- Olive, M. L., Lang, R. B., & Davis, T. N. (2008). An analysis of the effects of functional communication and a voice output communication aid for a child with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 2(2), 223–236.
- O'Neill, R. E., & Sweetland-Baker, M. (2001). An assessment of stimulus generalization and contingency effects in functional communication training. *Journal of Autism and Developmental Disorders*, 31, 235–240.
- Petscher, E. S., Rey, C., & Bailey, J. S. (2009). A review of empirical support for differential reinforcement of alternative behavior. *Research in Developmental Disabilities*, 30(3), 409–425.
- Prelock, P. A., Paul, R., & Allen, E. (2011). Evidence-based treatments in communication for children with autism spectrum disorders. In B. Reichow, P. Doehring, D. V. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-based treatments for children with autism* (pp. 93–169). New York: Springer.
- Saloviita, T., Itälä, M., & Leinonen, E. (2003). Explaining the parental stress of fathers and mothers caring for a child with intellectual disability: A double ABCX model. *Journal of Intellectual Disability Research*, 47(4/5), 300–312.
- Schwartz, I. S., Anderson, S. R., & Halle, J. W. (1989). Training teachers to use naturalistic time delay: Effects on teacher behavior and on the language use of students. *Journal of the Association for Persons with Severe Handicaps*, 14(1), 48–57.
- Smith, T., Scahill, L., Dawson, G., Guthrie, D., Lord, C., Odom, S., et al. (2007). Designing research studies on psychosocial interventions in autism. *Journal of Autism and Developmental Disorders*, 37(2), 354–366.
- Wacker, D. P., Steege, M. W., Northup, J., Sasso, G., Berg, W., Reimers, T., et al. (1990). A component analysis of functional communication training across 3 topographies of severe behavior problems. *Journal of Applied Behavior Analysis*, 23(4), 417–429.
- Wacker, D. P., Berg, W. K., Harding, J. W., Derby, K. M., Asmus, J. M., & Healy, A. (1998). Evaluation and long-term treatment of aberrant behavior displayed by young children with disabilities. *Journal of Developmental and Behavioral Pediatrics*, 19(4), 260–266.
- Wacker, D. P., Berg, W. K., Harding, J. W., Barretto, A., Rankin, B., & Ganzer, J. (2005). Treatment effectiveness, stimulus generalization, and parent acceptability of functional communication training. *Educational Psychology*, 25, 231–254.

Functional Connectivity

► Brain Connectivity Theories of Autism

Functional Ecological Approach

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Definition

The functional ecological approach is an approach to the assessment and intervention process that emphasizes environmental variables that may contribute to target behavior (Powers 2005, 1997). The model stands in contrast to earlier conceptualizations of behavioral assessment focusing on the topographical aspects of behavior (i.e., what the behavior looks like) rather than focusing on the underlying function (or cause) maintaining behavior. In contrast, the functional ecological emphasizes the need to evaluate the individual's behavior in a broad developmental context, accounting for relevant antecedent and consequent conditions affecting behavior.

The process emphasizes the context in which behavior occurs, thus increasing the likelihood that the assessment process, intervention strategies, and outcomes lead to benefits across environments. The approach leads to (1) the modification of the environment in which the target behavior occurs, (2) the manipulation of the controlling contingencies fort target behavior, (3) the development of functional equivalent replacement skills, and (4) the development of functional and socially valid skill repertoires. These gains exert their effects in multiple settings and are maintained in the natural environment.

See Also

► [Functional Behavior Assessment](#)

References and Reading

- Powers, M. D. (2005). Behavioural assessment of individuals with autism: A functional ecological approach. In F. Volkmar, R. Paul, A. Klin, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 817–830). New Jersey: Wiley.
- Powers, M. D. (1997). Behavioral assessment of autism. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (2nd ed., pp. 448–459). New York: Wiley.

Functional Goals

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Definition

A functional goal outlines a target skill to be acquired in measurable terms, while including a precise behavior to be accomplished and a specific criterion. It identifies the behavior or skill caregivers/instructors want the individual to learn or accomplish, the context in which the skill will be taught, and a quantifiable level of mastery. Functional goals should be developed so that they are implemented and practiced within the individual's daily routine.

Functional goals include three components: an acquisition statement, a normalized context, and a criterion. An acquisition statement describes the target behavior in terms that are observable and measurable. A normalized context identifies the activities or daily routines in which the behavior is expected. A criterion states the desired level of the behavior for mastery of the skill.

References and Reading

- Buyse, V., & Bailey, D. B. (1993). Behavioral and developmental outcomes in young children with disabilities in integrated and segregated settings: A review of comparative studies. *Journal of Special Education*, 26, 434–461.

- Haley, S. M., Baryza, M. J., & Blanchard, Y. (1993). Functional and naturalistic frameworks in assessing physical and motor disablement. In I. Wilhelm (Ed.), *Physical therapy assessment in early infancy* (pp. 225–255). New York: Churchill Livingstone.
- Heward, W. (2008). *Exceptional children* (9th ed.). Upper Saddle River: Merrill/Prentice Hall.
- McWilliam, R. A. (2009). *Goal functionality scale III*. Retrieved 8 Aug 2012 from The National Early Childhood Technical Assistance Center Website: http://www.nectac.org/~pdfs/topics/families/GoalFunctionalityScaleIII_2_.pdf
- Sandall, S., Hemmeter, M. L., Smith, B. J., & McLean, M. E. (2005). *DEC recommended practices: A comprehensive guide for practical application*. Longmont: Sopris West.

- If the student is unable to perform the skill himself or herself, it would need to be completed by another person.
- The skill will be needed throughout the person's life.

Historical Background

The philosophy behind teaching functional life skills is full inclusion in all activities of family and community life, making adaptations where necessary. There is one absolute purpose of education for all students, including those with autism spectrum disorder (ASD): Preparation for adulthood. The Individuals with Disabilities Education Act (IDEA 2004) was established to ensure all children with disabilities reach this collective goal and are provided opportunities for acquisition of knowledge and skills that lead to personal independence and social responsibility.

Research on the outcomes of adults with ASD yields sobering results. While some individuals with ASD are able to successfully achieve maximum independence and live self-fulfilling lives, many are faced with very significant obstacles in multiple areas. Even for those considered to be more skilled, they are unable to negotiate their way into college, work, community participation, and independent living. Experience has proven that individuals with all levels of ASD can work in a variety of businesses and industries (McDonough and Revell 2010; O'Brien and Daggett 2006). A spectrum of employment options is now available which includes segregated training centers, supported employment, and competitive employment. Despite research documenting the benefit of community-based employment, the vast majority of people with ASD continue to be unemployed or underemployed (National Organization on Disability 2004; Taylor and Seltzer 2011; Wagner et al. 2005). Additionally, participation in secondary education may provide a suitable alternative to employment; however, limited research in this area indicates participation is low (Howlin 2000). There is a continuum of living arrangements available to individuals with ASD which

Functional Integration

► Brain Connectivity Theories of Autism

Functional Life Skills

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Definition

Functional life skills are the variety of skills which are frequently demanded in natural domestic, vocational, and community environments. The skills involve those immediately applicable to daily life or may also include those which teach students to participate in future environments (Brown et al. 1979). Key qualities of functional skills include the following:

- It is performed within the context of a real activity.
- The activity is meaningful to the student.
- People without disabilities believe the activity serves a purpose.

includes living independently, living with family, partially supported living, and fully supported living. While an array of residential options is available, research on living outcomes demonstrate a small percentage of adults with ASD live alone, some reside in long stay hospitals or institutions, but most continue to live at home with their parents (Billstedt et al. 2005).

Such disappointing outcomes for adults with ASD have led to a shift in ensuring functional life skills are addressed during the educational years. Although it is important to focus on academic content and ensure students with ASD are provided the opportunity to benefit from the general education curriculum, it is also necessary to ensure they are taught skills needed to achieve independence and community integration. This should not be viewed as a lessening of goals but instead as an opportunity to engage all students in real-life application of skills.

Current Knowledge

Each student is an individual and requires a specialized set of learning objectives specifically designed to meet his or her needs. In many classrooms across the United States, students' individualized education plans (IEP) are virtually identical in content. This is providing an injustice to many students, as not all will require the same skills to achieve maximum independence. Considerations regarding functional life skills designed for current and future success will include different learning objectives based on the student. Such skills are individualized and focus on the unique qualities, dreams, and desires of the student. They require planning for the future, providing a positive road map regarding what it may hold, and systematically addressing the skills needed to achieve it. Through such visionary planning, the student learns what is needed for enhanced functioning in the natural community.

Determining skills to teach involves cooperation between students and teachers. Perhaps the most important factor when selecting functional life skills to teach is the value of student

and family preferences. Of crucial significance is whether the student is interested in the skill and has had a role in selecting the objective. If the student has been involved, he or she will be more motivated to learn it and is far more likely to use it outside of the school setting. Additionally, learning skills the student is vested in can lead to increasing self-concept and sense of accomplishment.

Another critical step to determining functional life skills requires examining a student's current and future environments (Brown et al. 1976). An ecologically oriented approach requires the student, teacher, and family to identify the environments and sub-environments where the student participates. For example, the student may participate in the school, home, synagogue, gym, and park. Next, the team discusses the student's high-priority activities for each of these major living environments. For example, if the student goes to a fast-food restaurant every Saturday, he may need to learn to take turns, wait, order food, and exchange money. There is no limit to the number of functional life skills identified using an ecological approach since any student with an autism spectrum disorder will have some deficits related to increasing independence that will need to be addressed (Schall et al. 2012).

Although targeting functional life skills is traditionally emphasized at the high school level, they should be carefully considered for elementary and middle school students as well. Certainly, as the student ages and needs to evolve, the skill requiring instruction will change. Acquisition becomes more essential as the student gets closer to graduation but should begin early for every student with ASD.

Although functional life skills will differ from individual to individual, there are key areas to be addressed. They include the following:

- Career education and work: Students need to learn skills that will allow them to identify a career path, obtain and hold a job, and understand the relationship between work responsibility, pay, and getting along with others (McDonough and Revell 2010; Wehman 2001).

- **Community participation:** Community-based skills encompass a breadth of skills related to mobility, accessibility, and interactions. Listing the number of skills related to community is nearly impossible because there are so many. However, skills may include using a sidewalk, crossing a street, soliciting a retailer, or waiting in line at a movie theatre.
- **Home living skills:** Domestic skills are essential for community living. The optimal living environment for the student must be identified and then the skills essential for success targeted. Examples of home living skills include preparing lunch, cleaning the bathroom, and making the bed.
- **Self-help:** A key component of independence is self-care. Self-help skills allow a person to meet his own needs and be less reliant on another person. Self-help skills include such skills as eating, meal preparation, dressing, grooming, and toileting. They are critical skills as some are needed to survive (e.g., eating), while others are needed to leave the house and participate in the community (e.g., getting dressed). Self-help skills can be a major factor in accessibility to certain environments. For example, a young child with ASD who is not toilet trained may not be allowed in a child care center, while an adult with ASD who cannot use a public restroom may not be able to maintain a job.
- **Transportation:** A major aspect of independence in the community is related to getting to and from various locations. Skills related to transportation include not only using various modes of transportation such as driving a car or using a bus but also include planning travel based on desire as well as need to get to determined locations.
- **Self-determination:** Self-determination prepares students for adulthood teaching them to take control over their lives, understand their rights, and self-advocate. Too often, students with ASD become highly dependent on caretakers. This reliance then inhibits independence. Some examples of skills related to self-determination include self-awareness, goal setting, choice making, decision-making, assertiveness, and problem solving (Wehmeyer and Lawrence 1995).
- **Personal health and safety:** Acting responsibly and promoting the well-being of one's self as well as others is a vital functional skill. This focuses on attending to one's own health needs and demonstrating behaviors that ensure safety and may include such skills as applying a Band-Aid, calling 911, or monitoring medication.
- **Financial planning and management:** Economic self-sufficiency is a goal for most individuals with or without a disability. Activities may range from basic skills such as managing money to more complex activities including investment planning. Skill related to financial planning involves knowing how to shop by comparing prices, budgeting, saving for a purpose, investing, as well as avoiding scams and unfair marketing practices.
- **Functional academics:** Most education programs focus on academic content. For some students, learning the general education academic curriculum may not be possible. Therefore, functional academics may be advantageous. This involves teaching academic content in a way that uses real-life application and moves the student toward independence. For example, a student working on reading may learn to identify sight words he or she will see in the community. A student working on math may learn to count money and add the cost of two items purchased. An essential way to determine whether to teach functional academics is to ask whether the student will be able to use the information currently or in the future or if the information is needed to obtain the student's educational goals. Increased learning of functional academics may impact the type of diploma received and may determine whether the student is able to pursue postsecondary education.
- **Socialization:** Appropriate social skills are essential for all people. Socialization deficits is the hallmark characteristic of ASD. Therefore, targeted instruction in this area is required for every student on the spectrum. The skills required for socialization are complex and are

arguably the most difficult for an individual with ASD to learn. However, unless social skills are taught, full community integration and social fulfillment remain a challenge (National Research Council 2001). Socialization skills include those related to peer interaction, friendships, and the range of relationships one may encounter. Additionally, socialization includes skills related to comprehending social stimuli, reciprocation, social rules, and affect and emotions.

- Sexuality: Sexuality is a natural part of the human experience, and it is a right for individuals with ASD to experience it. Skills related to understanding the facts regarding sexuality as well as the social rules surrounding its expression are typically required for a person on the spectrum (Stokes et al. 2007).
- Recreation and leisure: Students need to learn activities to perform in his or her free time. For many with ASD, leisure pursuits are rarely community based and are likely to be isolated activities such as video games and watching television (Jennes-Coussens et al. 2006; Wagner et al. 2005). Skills related to recreation and leisure may include teaching independent play skills to reduce stereotypy, interactive games, or team activities.

Future Directions

With the passage of No Child Left Behind (NCLB 2001) as well as the revision of IDEA (2004), access to the general education curriculum has become a more prominent focus for students with disabilities including those with ASD. However, as research has evolved and we have learned more about the autism spectrum, it has become increasingly clear that all students with this disorder require a focus on functional life skills in addition to other curricular content. Instruction is required related to community, employment, home, and leisure as well as academics related to the transition goals (Downing 2005; Nuehring and Sitlington 2003). Moreover, strategies for remedying communication,

socialization, and behavioral deficits which profoundly and perpetually impact the individual are critical (Iovannone et al. 2003). Future directions in this area include educating teachers on how to identify critical life skills students need to be successful as well as help them to integrate such skills into the general curriculum, so the student leaves school armed with the skills needed for adulthood.

See Also

- Adaptive Behavior
- Curriculum

References and Reading

- Billstedt, E., Gillberg, C., & Gillberg, C. (2005). Autism after adolescence: Population-based 13-to 22-year follow-up study of 120 individuals with autism diagnosed in childhood. *Journal of Autism and Developmental Disorders*, 35(3), 351–360.
- Brown, L., Nietupski, J., & Hamre-Nietupski, S. (1976). Criterion of ultimate functioning. In M. Thomas (Ed.), *Hey, don't forget about me!* (pp. 212–242). Reston: Council for Exceptional Children.
- Brown, L., Branstetter, M. B., Hamre-Nietupski, S., Pumpian, I., Certo, N., & Gruenewald, L. (1979). A strategy for developing chronological age appropriate and functional curricular content for severely handicapped adolescents and young adults. *Journal of Special Education*, 13(1), 81–90.
- Downing, J. E. (2005). *Teaching literacy to students with significant disabilities*. Thousand Oaks: Corwin Press.
- Howlin, P. (2000). Outcome in adult life for more able individuals with autism or asperger syndrome. *Autism*, 4(1), 63–83.
- Hurlbutt, K., & Chalmers, L. (2004). Employment and adults with Asperger syndrome. *Focus on Autism and Other Developmental Disabilities*, 19(4), 215–222.
- Individuals with Disabilities Education Act Amendments of 2004*, U.S.C. PL 105-17, 111 (2004).
- Iovannone, R., Dunlap, G., Huber, H., & Kincaid, D. (2003). Effective educational practices for students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 18, 150–165.
- Jennes-Coussens, M., Magill-Evans, J., & Koning, C. (2006). The quality of life of young men with Asperger syndrome: A brief report. *Autism*, 10(4), 403–414.
- McDonough, J., & Revell, G. (2010). Accessing employment supports in the adult system for transitioning

- youth with autism spectrum disorders. *Journal of Vocational Rehabilitation*, 32(2), 89–100.
- National Organization on Disability. (2004). *N.O.D./Harris Survey of Americans with Disabilities: Landmark survey finds pervasive disadvantages*. Washington, DC: National Organization on Disability. Retrieved 22 Oct 2010, from <http://www.nod.org/content.cfm?id=1537>
- National Research Council. (2001). In C. Lord & J. P. McGee (Eds.), *Educating children with autism*. Washington, DC: National Academy Press, National Research Council: Division of Behavioral and Social Sciences.
- No Child Left Behind Act of 2001*, 20 U.S.C. 70 6301 (2001).
- Nuehring, M. L., & Sitlington, P. L. (2003). Transition as a vehicle: Moving from high school to an adult vocational service provider. *Journal of Disability Policy Studies*, 14(1), 23–35.
- O'Brien, M., & Daggett, J. A. (2006). *Beyond the autism diagnosis: A professional's guide to helping families*. Baltimore: Paul H. Brookes.
- Schall, C., Doval, E. C., Targett, P. S., & Wehman, P. (2012). Applications for youth with autism spectrum disorders. In P. Wehman (Ed.), *Life beyond the classroom: Transition strategies for young people with disabilities* (5th ed., pp. 447–472). Baltimore: Paul H. Brookes.
- Stokes, M., Newton, N., & Kaur, A. (2007). Stalking, and social and romantic functioning among adolescents and adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37(10), 1969–1986.
- Taylor, J., & Seltzer, M. (2011). Employment and post-secondary educational activities for young adults with autism spectrum disorders during the transition to adulthood. *Journal of Autism and Developmental Disorders*, 41, 566–574.
- Wagner, M., Newman, L., Cameto, R., Garza, N., & Levine, P. (2005). *After high school: A first look at the post school experiences of youth with disabilities*. A report from the National Longitudinal Transition Study-2 (NLTS-2). Menlo Park: SRI International.
- Wehman, P. (2001). *Supported employment in business: Expanding the capacity of workers with disabilities*. St. Augustine: Training Resource Network.
- Wehmeyer, M. L., & Lawrence, M. (1995). Whose future is it anyway? Promoting student involvement in transition planning. *Career Development for Exceptional Individuals*, 18(2), 68–84.

Functional Magnetic Resonance Imaging

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Definition

Functional magnetic resonance imaging (fMRI) is a technique that examines brain activity by measuring blood oxygenation. When a region of the brain is in use, neuronal firing increases, and oxygen is supplied to that area by the blood. The oxygen-carrying protein in the blood, hemoglobin, is affected differently by magnetic fields based on whether or not the hemoglobin is oxygenated. This property allows us to index the relative amount of these types of hemoglobin in the blood, which is reflected in the **blood oxygen level-dependent (BOLD)** signal. A powerful magnet in the magnetic resonance imaging (MRI) scanner allows for the acquisition of data about where in the brain the BOLD signals are located. This signal tends to follow a particular activation pattern called the **hemodynamic response**. When neurons fire, oxygenated blood rushes to that area of the brain, displacing the deoxygenated blood about 2 s after firing, a response which peaks 4–6 s after the neural activation and then declines (see Fig. 1). This signal is an indirect measure of where in the brain activity is occurring, allowing for conclusions about which brain regions are active during certain cognitive processes.

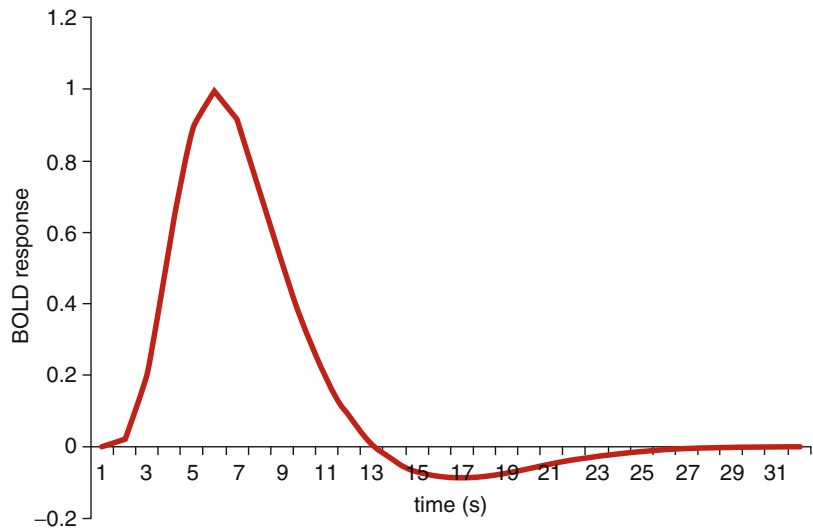
Patients undergoing an fMRI scan lie still inside a tube, called a bore, with a coil placed above their heads. The head coil emits radio frequencies that, upon return, are captured by the coil in a manner proportional to the strength of the magnetic field there. The amount of energy returned depends on the degree of blood oxygenation, and the BOLD signal varies accordingly. The combination of a static magnetic field and oscillating gradient fields is used to create a

Functional Living Skills

► Assessment of Functional Living Skills (AFLS)

Functional Magnetic Resonance Imaging,

Fig. 1 The hemodynamic response



two-dimensional grid upon which the BOLD signal is localized.

Functional MRI is noninvasive and does not require the injection of a radioactive tracer, as do some neuroimaging approaches like positron emission tomography (PET) and single photon emission computed tomography (SPECT). fMRI has superior **spatial resolution** compared to other functional neuroimaging techniques, with the capability of locating brain activation in nearly any region within a few millimeters. Moreover, it has been used successfully with awake children as young as four (both typically and atypically developing), as well as with sleeping infants. However, fMRI exhibits limited temporal resolution due to the necessity of waiting to capture the BOLD signal's change and the technological limitations of whole-brain image acquisition, which occurs approximately every two seconds. Moreover, the signal itself is only a correlate of neural activity, not a direct measurement. fMRI is also expensive, with a single machine costing around 1.2 million. In order to do fMRI research, a highly trained team and dedicated space for the equipment are required, which adds to the total production cost.

Despite these challenges, fMRI provides insight into the neural mechanisms underlying a range of cognitive processes. In addition, fMRI can offer information on how brain activity differs between typically developing individuals and those with

intellectual, emotional, or developmental disabilities. fMRI findings can be examined in conjunction with data from other neuroimaging methods, such as PET, event-related potentials (ERP), magnetoencephalography (MEG), and functional near-infrared spectroscopy (fNIRS), all of which measure brain activity in different ways. Along with these other neuroimaging methods, fMRI can help to identify specific neurobiological indicators of disease and dysfunction toward the goals of prevention, early diagnosis, and individualized treatment.

Historical Background

fMRI Technology

Localization of BOLD responses with functional MRI is carried out in conjunction with a high-resolution structural MRI scan. The first structural image collected with MRI (a pair of water-filled test tubes) was published in 1973 and was created using four gradients oriented at different angles. This method was inefficient and time-consuming, which inspired the proposition of **echo-planar imaging (EPI)** in 1976. Echo-planar imaging uses rapidly shifting magnetic field gradients and a series of singular electromagnetic pulses to record MR signals. This approach dramatically decreased the time necessary to acquire an image and is the foundation of MRI technology used

today. The first magnetic resonance image of the human body was acquired in 1977, and by the late 1970s, medical personnel were beginning to realize the clinical potential of MRI. However, this early MRI technology could only reveal biological structure and was not sensitive to oxygen levels in the blood.

Throughout the next two decades, 1.5 T scanners were the most commonly used in research and clinical practice. Standard magnets were soon replaced with superconducting magnets, which made possible increases in the strength of the magnetic field (as measured in Teslas). When the Food and Drug Administration (FDA) approved MRI scanners for clinical use in 1985, their installation in hospitals increased dramatically, which facilitated greater interest in MRI and paved the way for functional MRI to flourish. Most MRI scanners used today are 3.0 T scanners, which appeared in the mid-2000s.

Blood Flow and the Hemodynamic Response

Early measurements of blood flow across the scalp were conducted in the 1890s by Angelo Mosso, a physiologist. He recorded pulsation on the skull during mental activity and provided some of the first evidence of the coupling of blood flow to neuronal activity. Though brain activity and blood flow had been linked repeatedly by the 1930s, there was not widespread acceptance of this idea as a way to measure brain function. PET was the primary method of neuroimaging for decades, despite the discovery by Pauling and Coryell of the differential reactions of oxygen-rich and oxygen-poor blood to magnetic fields in 1936. However, in 1990, Seiji Ogawa performed the first functional MRI scan measuring the BOLD signal in a rodent, which inspired the three studies published in 1992 examining the BOLD signal in humans. Since then, the number of studies using fMRI to examine changes in blood oxygenation as an indicator of brain activity has skyrocketed.

Current Knowledge

fMRI has been used to study the neural mechanisms underlying nearly every psychological

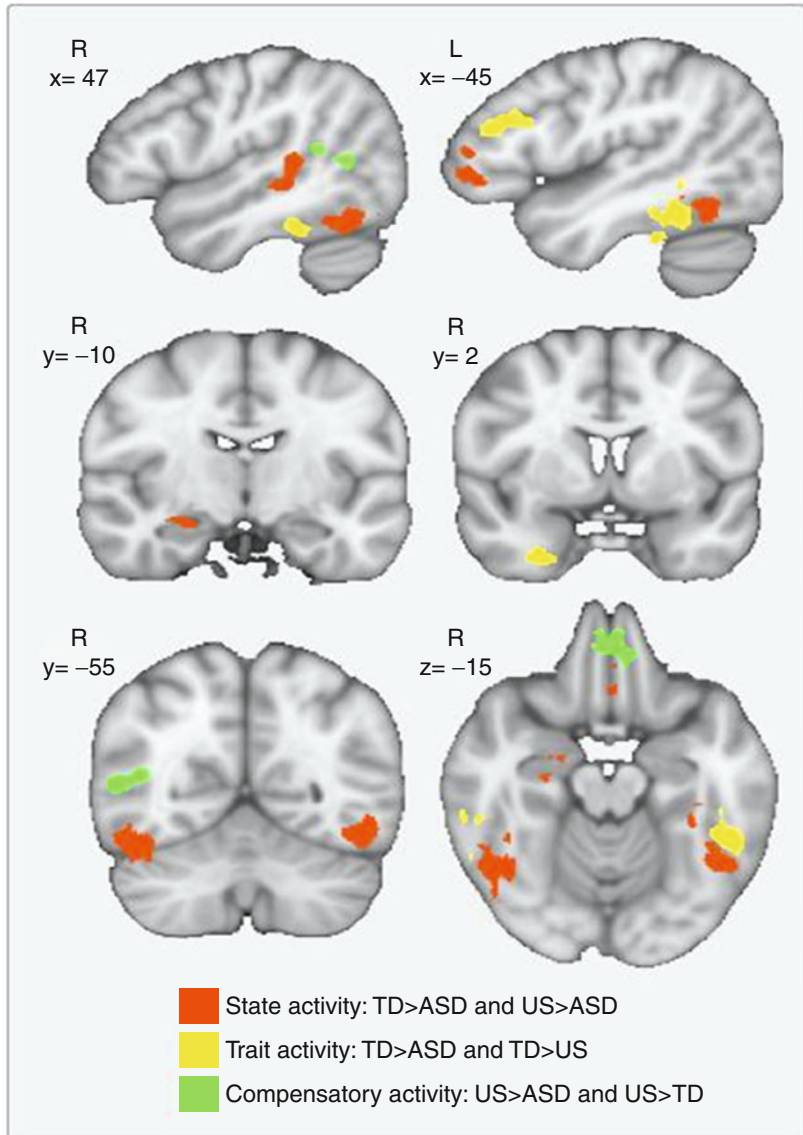
disorder, from depression and anxiety to schizophrenia and Alzheimer's disease, as well as many physical disorders. This broad use has resulted in a wealth of knowledge about the specific functionality of many areas of the human brain, information that can be used to augment our understanding of developmental disorders such as autism. The nature of fMRI is well suited to studying autism, as it can track brain structure and function during developmentally important periods. Current information points toward the causes of autism being, at least in part, neurobiological, making the examination of the structural and functional progression of the brain throughout disorder development crucial to understanding its etiology. Identification of where in the brain dysfunction is occurring also suggests potential areas for genetic or structural abnormalities, information that is unavailable from other imaging methods like ERP. Furthermore, unlike PET scans, which risk radioactive material accumulation and exposure when repeated too often, fMRI can be done repeatedly with minimal risk of ill effects. This allows for the longitudinal studies that are especially important for understanding the developmental trajectories of autism.

fMRI has been used to examine a number of processes thought to be relevant to autism-related behavioral symptomatology. One of the most highly studied networks of areas is the "social brain," which includes the orbitofrontal cortex (OFC), superior temporal sulcus (STS), amygdala, prefrontal cortex (PFC), and the fusiform gyrus (FG). These regions have been implicated in many socially related processes, such as face processing, detection and attention to biological motion, and theory of mind or thinking about the mental states of others.

For example, fMRI has revealed that individuals with autism tend to exhibit less activation in social brain regions when viewing biological motion, a phenomenon which is thought to be related to autistic deficits in attention to socially motivating stimuli such as faces. In support of this idea, adults with autism show significantly less activation in the FG, as well as activation in the superior temporal gyrus (STG), when viewing facial expressions. Moreover, adults with autism have been shown to utilize areas of the brain

Functional Magnetic Resonance Imaging,

Fig. 2 Localization of decreased activity in those with autism (state); decreased activity, reflecting genetic vulnerability to autism, in both children with autism and their unaffected siblings (trait); and compensatory activity unique to unaffected siblings (Figure reprinted with permission from the Proceedings of the National Academy of Sciences. Kaiser, M. D., Hudac, C. M., Shultz, S., Lee, S. M., Cheung, C., Berken, A. M., Pelphrey, K. A. (2010). Neural signatures of autism. Proceedings of the National Academy of Sciences, 107, 21223–21228)



associated with object feature processing (the inferior temporal gyrus) when processing faces, as opposed to using the FG as seen in typically developing controls. Taken together, these findings suggest that individuals with autism process faces differently than do typically developing individuals.

Research comparing the brain function of children with autism and their unaffected siblings against typically developing individuals is beginning to emerge. In one study, while viewing biological motion, children with autism showed

decreased activation in the PFC, STS, and FG as expected. However, their unaffected siblings, who were behaviorally indistinguishable from typically developing controls, also showed decreased activation in parts of the PFC and in the FG, likely reflecting the shared genetic vulnerability to developing autism. Unaffected siblings also exhibited unique patterns of activation in particular areas of the PFC and STS that were not activated in either affected or typically developing children (see Fig. 2). This activity could be a mechanism by which unaffected siblings

compensate for genetic vulnerability to autism or could reflect genetic factors that served a protective function against disorder development.

The existence of brain activation commonalities in children with autism and their siblings, and of compensatory activity in unaffected siblings, has been supported by other work. Decreased activation in the STS, FG, and PFC in response to happy faces was observed in both affected children and unaffected siblings compared to typically developing controls, indicating a potential common deficit in those at genetic risk for developing autism. Moreover, during a visual attention task, children with autism and their siblings both showed atypical, delayed activation in the frontocerebellar attention systems relative to controls, with the strongest activation in the unaffected siblings. This suggests that unaffected siblings are attempting to compensate for the delayed activation, which is likely a reflection of genetic vulnerability to autism.

Studies of this design offer a unique and powerful opportunity to identify **biomarkers**, neuroimaging findings exclusive to, or shared between, each subgroup (affected, unaffected, and typical). Accurate identification of these biomarkers can assist in developing an **endophenotype** of autism, which details the anatomical, neuropsychological, and physiological features that reflect genetic risk for developing the disorder. Current MRI work has highlighted potential anatomical biomarkers of autism risk, but the use of functional MRI to identify differences in brain activation is less prevalent. The identification of a reliable endophenotype of autism could provide important insight into the mechanisms underlying the disorder toward the goal of presymptomatic diagnosis, which could then facilitate earlier implementation of intervention.

Neural activation, as measured by fMRI, is starting to be utilized as a treatment outcome. Following 4 months of pivotal response treatment (PRT), a behavioral therapy designed to increase social motivation through naturalistic, play-based intervention, preschool and kindergarten-aged children with autism showed greater STS, FC, and PFC in response to biological motion, suggesting that treatment had increased (i.e., normalized)

activation in regions used by typically developing children during social perception. These findings are particularly interesting in that they demonstrate that the social brain areas may be malleable to treatment beyond the commonly accepted “critical period” for intervention (2–4 years). In addition, after 10 months of treatment with diuretic bumetanide, a drug that reinforces GABAergic inhibition, young adults with autism exhibited enhanced activation in the brain areas involved in social and emotional information processing from faces, highlighting the potential for drug intervention to change brain function. Though further study is needed, these preliminary findings suggest that fMRI may be a useful tool for quantifying treatment outcomes, with the goal of eventually identifying biomarkers of treatment response. Once these biomarkers are reliably established, treatment response may be able to be predicted based on neuroimaging findings, which would then help facilitate the customization of treatment plans to meet each individual’s specific needs.

While fMRI can be used to identify discrete areas of the brain that are active during certain cognitive processes, it can also be used to examine networks of brain regions involved in these processes, a method called functional connectivity analysis. For example, individuals with autism exhibited decreased synchronization between the inhibition network (anterior and middle cingulate gyrus and insula) and parts of the frontal and parietal regions relative to controls during an executive function task tapping response inhibition capabilities. Resting state functional connectivity has blossomed recently as a point of interest in the field. True to its name, resting state functional connectivity intends to locate neural networks in the absence of overt stimulation. This data is gathered during a “resting state” task, where the participant is asked to lie quietly and look at a blank screen. Resting state functional connectivity analyses have shown hypoconnectivity in many areas of the brain in children with autism, particularly in the temporal lobe and between hemispheres, as well as hyperconnectivity in subcortical areas such as the thalamus. Moreover, the mid- and posterior insula and posterior cingulate cortex have been identified as

areas of dysfunction in children with autism. Connectivity abnormalities seen in the default network, or the areas of the brain (such as the medial PFC, superior frontal gyrus, temporal lobe, and posterior cingulate cortex) that are active in the absence of overt stimulation, are associated with poorer social functioning and more severe restricted and repetitive behaviors. Functional connectivity analyses can inform understanding of how areas of the brain communicate with one another and how this communication may be disrupted in children with autism, which may further reveal biomarkers or potential targets for intervention.

Future Directions

Neuroimaging as a field, including the use and application of fMRI, is in its infancy relative to many other areas of study. The progress already made in advancing the knowledge of brain structure and function and the increasing complexity of the processes able to be studied highlights the incredible potential for impact in both research and clinical spheres. Scanning technology is improving constantly, from the gradient fields used to collect the images to the software that analyzes the data, underscoring the possibility of greater spatial resolution and a more precise delineation of neural activation. Methodological design in the field is also changing rapidly; it is now feasible to gather fMRI data and PET, ERP, MEG, or fNIRS data simultaneously. While this has been possible for nearly a decade, utilization costs have impeded widespread use until recently, particularly for combined fMRI-PET research, as these scans require a specially equipped MRI scanner. With the quality and capability of scanning technology increasing exponentially, it is likely that the use of these methods will flourish.

Recent advances in data acquisition and processing techniques have also allowed for the use of real-time fMRI neurofeedback as a method by which to encourage the acquisition of control over certain cortical processes, such as emotion regulation. Results from the relatively small number of studies that have utilized this method are positive,

highlighting the potential of this technique to inspire treatment progress. These advances may be particularly relevant to clinical practice, where the functionality of fMRI as a clinical tool has been slow to translate. The use of fMRI in diagnosing disorders before behavioral or physical symptoms develop, informing drug development and delivery, customizing treatment plans, and supporting treatment progress is bound to have a marked impact on the design and execution of clinical practice.

Furthermore, translational work that combines the study of neurobiology, genetics, and behavior is beginning to appear in world-class research facilities across the globe. For example, a current Autism Centers of Excellence (ACE) project focuses specifically on the etiology of autism in girls using a multisite collaboration design that spans the United States. This project examines the genetics of the disorder by gathering samples from typically developing children and children with autism, as well as the siblings and parents of both groups. In addition, fMRI and ERP are used to quantify neural activation to stimuli evoking the various cognitive processes thought to contribute to autism-related deficits. Finally, extensive behavioral measures, including cognitive, language, adaptive functioning, and autism symptom assessments, are collected from all children involved. Once completed, the study will be one of the most comprehensive ever done on the etiology of autism in general and specifically on what autism looks like in girls. The study will assist in elucidating the differences in the neurobiological, genetic, and behavioral profiles of girls and boys with autism, of children with autism and their unaffected siblings, and of those with autism and typically developing individuals. As recognition of the importance of research that crosses disciplinary and methodological boundaries grows, the number of studies employing translational principles is bound to increase markedly.

Functional magnetic resonance imaging is a powerful and widely applicable method for examining brain function. There is incredible potential for growth and progress within the field, and the possibility of widespread clinical use is in the foreseeable future. Specifically in autism, a better

understanding of the differences in brain function of children with autism is crucial to understanding key autism-related deficits, how to ameliorate them, and, perhaps most importantly, how to prevent the disorder's development altogether. Within the next few decades, more sophisticated technology and increased visibility of neuroimaging methods will hopefully inspire great strides in both research and clinical arenas.

See Also

- [Magnetic Resonance Imaging](#)
- [Neural Mechanisms in Autism](#)
- [Superior Temporal Sulcus Region](#)

References and Reading

- Ashwin, C., Baron-Cohen, S., Wheelwright, S., O'Riordan, M., & Bullmore, E. T. (2007). Differential activation of the amygdala and the 'social brain' during fearful face-processing in Asperger Syndrome. *Neuropsychologia*, 45, 2–14.
- Belmonte, M. K., Gomot, M., & Baron-Cohen, S. (2010). Visual attention in autism families: 'Unaffected' sibs share atypical frontal activation. *Journal of Child Psychology and Psychiatry*, 51, 259–276.
- Cherkassky, V. L., Kana, R. K., Keller, T. A., & Just, M. A. (2006). Functional connectivity in a baseline resting-state network in autism. *Neuroreport*, 17, 1687–1690.
- Di Martino, A., Yan, C. G., Li, Q., Denio, E., Castellanos, F. X., Alaerts, K., & Milham, M. P. (2013). The autism brain imaging data exchange: Towards a large-scale evaluation of the intrinsic brain architecture in autism. *Molecular Psychiatry*, 19, 659–667.
- Hadjikhani, N., Zürcher, N. R., Rogier, O., Ruest, T., Hippolyte, L., Ben-Ari, Y., & Lemonnier, E. (2013). Improving emotional face perception in autism with diuretic bumetanide: A proof-of-concept behavioral and functional brain imaging pilot study. *Autism*. <https://doi.org/10.1177/1232361313514141>.
- Huettel, S. A., Song, A. W., & McCarthy, G. (2009). *Functional magnetic resonance imaging* (Vol. 2). Sunderland: Sinauer Associates.
- Kaiser, M. D., Hudac, C. M., Shultz, S., Lee, S. M., Cheung, C., Berken, A. M., & Pelphrey, K. A. (2010). Neural signatures of autism. *Proceedings of the National Academy of Sciences*, 107, 21223–21228.
- Kana, R. K., Keller, T. A., Minshew, N. J., & Just, M. A. (2007). Inhibitory control in high-functioning autism: Decreased activation and underconnectivity in inhibition networks. *Biological Psychiatry*, 62, 198–206.
- Monk, C. S., Peltier, S. J., Wiggins, J. L., Weng, S. J., Carrasco, M., Risi, S., & Lord, C. (2009). Abnormalities of intrinsic functional connectivity in autism spectrum disorders. *NeuroImage*, 47, 764–772.
- Raichle, M. E. (1998). Behind the scenes of functional brain imaging: A historical and physiological perspective. *Proceedings of the National Academy of Sciences*, 95, 765–772.
- Savoy, R. L. (2001). History and future directions of human brain mapping and functional neuroimaging. *Acta Psychologica*, 107, 9–42.
- Schultz, R. T., Gauthier, I., Klin, A., Fulbright, R., Anderson, A., Volkmar, F., et al. (2000). Abnormal ventral temporal cortical activity among individuals with autism and Asperger syndrome during face discrimination. *Archives of General Psychiatry*, 57, 331–340.
- Spencer, M. D., Holt, R. J., Chura, L. R., Suckling, J., Calder, A. J., Bullmore, E. T., & Baron-Cohen, S. (2011). A novel functional brain imaging endophenotype of autism: The neural response to facial expression of emotion. *Translational Psychiatry*, 1, e19.
- Voos, A. C., Pelphrey, K. A., Tirrell, J., Bolling, D. Z., Vander Wyk, B., Kaiser, M. D., & Ventola, P. (2013). Neural mechanisms of improvements in social motivation after pivotal response treatment: Two case studies. *Journal of Autism and Developmental Disorders*, 43, 1–10. <https://doi.org/10.1007/s10803-012-1683-9>.

Functional MRI

- [Event-Related Functional Magnetic Resonance Imaging \(MRI\)](#)

Functional Play

- [Play](#)

Functional Routines (FR), Teaching

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Definition

Information-processing deficits in individuals with autism spectrum disorders (ASD) inhibit

their ability to accurately attend to instructions, effectively participate in learning and social activities, and easily adapt to novel activities and change. Routines are effective at diminishing the information-processing load (Shatz 1983) by providing an inherent organizational structure that reduces variation and supports individual's participation in them (Constable 1986). The teaching of functional routines can assist individuals with ASD to be more successful in engaging in and completing everyday activities. Routines have the benefit of being sequential (McClannahan and Krantz 1999; Siegel-Causey and Guess 1989; Yoder and Davies 1992) and predictable (Prizant et al. 2006; Snyder-McLean et al. 1984) and are found in the natural environment (Pretti-Frontczak and Bricker 2004; Sussman 1999). Functional routines are predictable events that involve a chain of behaviors that are broken down into teachable units. They provide a reliable context that allows the individual to more independently participate or complete a specified activity. Functional routines instruction involves providing systematic prompts and cues to teach the child to participate independently in common school or home-based (self-care) routines (Arick et al. 2004). For example, the behaviors of arriving to school, hanging one's coat up in the locker, traveling to desk, and taking out materials for the day are all integrated behaviors that must be completed to achieve the function of this routine. The routines will vary upon the setting in which they are taught. If it is during the school day, they could consist of transitioning and arrival, participating in circle-time or classroom activities, using the restroom, greeting a peer, eating lunch, etc. If the functional routines are being taught in the home environment, they may consist of improving daily living skills, such as independently dressing, eating meals, playing a social game with a caregiver, or making leisure choices.

Historical Background

Routines have been described as a "scaffold" for children (Bruner 1975). Bruner and his

colleagues focused their research on the development of joint-action routines which promote shared attention, cooperative activity, and language development, for example, peekaboo, book sharing, or object exchange (Ninio and Bruner 1978). This use of routines was later adapted by McLean and Snyder-McLean (1987) as part of a communication intervention that focused on highlighting the identifiable cycles and turn-taking opportunities that were present in everyday routines, such as feeding or bathing. The use of routines has historically been implicated in various language interventions (see Constable 1986; Nelson 1986).

Functional skills are described as those that will have a meaningful impact on the individual's life. The importance of explicitly teaching skills that are functional and using a routine as the platform for teaching these skills has been a critical intervention focus, particularly with individuals with severe disabilities (Brown et al. 1976; Snell 1983; Wilcox and Bellamy 1982). Historically, a task-analysis model was used to identify the skills and activities that are functional for a student and then further divide the activity or routine into teachable units of behavior. Brown et al. (1987) offered the component model of functional life routines, which provides a systematic structure for the specification of behaviors relevant to student competence. The model was proposed to provide a more comprehensive approach to the identification of behavioral sequences that will lead to a more successful and independent completion of routines by the individual with a disability.

Specific research related to the use of functional routines with individuals with ASD is scarce; however, the concept has evolved from both language interventions (as described above) and the documented need to provide predictability and routine (Dawson and Osterling 1997) to promote participation and independence for individuals with ASD. Further, the use of routines is often a component in functional communication training, which is a common intervention used to improve the language and behavior of individuals with ASD (see Mancil 2006).

Current Knowledge

Limited research is available regarding the sole use of functional routines in teaching individuals with ASD. Often, the use of functional routines is naturally embedded as part of a comprehensive intervention program or classroom model (Arick et al. 2003; Dawson et al. 2010; Landa et al. 2011). The use of functional routines varies on the setting in which they are being used. In the school or workplace setting, they can be used as a generalization strategy after specific behaviors have been taught in a highly structured context (such as one-on-one using discrete trial training), and now they might be chained together in a more natural context (such as circle time) to promote participation or engagement in the routine.

The STAR program, Strategies for Teaching Based on Autism Research, identifies functional routines as one of the three primary instructional strategies (the other two being discrete trial training and pivotal response training) that comprise the intervention. The STAR program utilizes functional routines to assist the young child with ASD to independently participate in common school (e.g., participation in circle-time routines) and self-care routines (e.g., using the bathroom). The use of functional routines in this program assists with creating a structured learning environment. Arick et al. (2003) researched the use of functional routines in a preliminary outcome study of the program and noted it to be one of the most common one-to-one instructional strategies utilized in the population studied. A study by Stahmer et al. (2015) looked at the fidelity of implementation of the evidence-based practices within the STAR program for teachers within an urban public school classroom. The researchers found that with consistent and ongoing coaching and support, teachers were able to implement functional routines with a high level of procedural integrity. In a follow up study investigated the fidelity of implementation of the individual components of the STAR program as associated with gains in cognitive ability, even though teachers once again demonstrated high fidelity implementing functional routines, this strategy was not directly related to gains in cognition (Pellecchia et al. 2015). However, as discussed

below, the type of gains expected from the use of functional routines would be related to participation in activities and independence in self-care or school-based routines rather than a direct increase in cognitive abilities.

Rationale or Underlying Theory

The underlying theory for teaching functional routines is grounded in the need for creating a context that is predictable, repeated often (offering multiple opportunities to practice and participation), and comprised of smaller segments of functional activities that can be broken down and taught part by part. The behaviors taught as part of the routine are functional to ensure more independence in the individual's life. The routine can be conceptualized as a script (Bruner 1975) for the individual with ASD and reduce the cognitive load and improve their successful and independent participation in or completion of the activity (Nelson 1986).

Goals and Objectives

When identifying the goals and objectives associated with a functional routine, one must consider the functionality of the skills targeted within the curriculum or those required for completion of important activities in the life of an individual with ASD. Focus should be on those skills that (a) are most likely to be useful in the student's life to control his or her environment, (b) will increase the student's independence and quality of life, and (c) will increase the student's competent performance (Iovannone et al. 2003). The goal will most likely be to independently and successfully complete or participate in the specified functional routine. Each functional routine can be task analyzed into a number of core steps or behaviors, and these behaviors can be identified as the objectives. Independent mastery of each objective in succession will lead to the functional and independent completion of the routine. The ASD individual's specific strengths and needs, the context, and the actual steps or components of the routine will determine the specific goals and objectives.

Treatment Participants

Traditionally, the teaching of functional routines has been used with individuals with cognitive impairments and those with ASD. They can be utilized with very young children, related to creating routines for caregivers and children (Kashinath et al. 2006; Kern et al. 2007) or with older individuals who need assistance completing functional life skills (Iovannone et al. 2003; Ogletree et al. 2007).

Treatment Procedures

There is an inherent cycle for teaching functional routines, which includes (a) identification of a routine which is meaningful for the individual with ASD to complete independently, (b) task analysis of the steps and skills needed for a particular routine, (c) assessment of the individual's independent completion of each step or use of each skill to successfully participate or complete the routine, (d) direct instruction of each behavior/skill required in the functional routine, and (e) data collection on independence in participation or completion of the routine postinstruction. Further explanation of this cycle is below:

1. Identification of a functional routine. Determine what routine is meaningful for the individual with ASD to learn to complete independently. This could be to successfully arrive at work and prepare for the day, taking a bath at home, or participating in a song-gesture routine at circle time. The specific routine will depend on the context and the individual needs of the person with ASD.
2. Task analyze the steps and skills needed for a particular routine. The adult who will be teaching the functional routine should break down the routine into specific sequential steps and skills and identify what behaviors need to be taught for successful participation or completion of the routine.
3. Assessment of the individual's independent completion of each step of the routine. Prior to teaching the functional routine, the adult

instructor should complete a baseline measurement of the routine and measure the ASD individual's independence in completing each of the identified steps and skills in the functional routine. After collecting this information, the adult instructor should develop a lesson plan identifying the specific objectives (behaviors/skills) that need to be taught and the instructional procedures and strategies that will be used to teach the behaviors/skills for the routine.

4. Direct instruction of each behavior or skill in the functional routine. Depending on the needs of the individual with ASD, one might need to preteach each specific behavior individually using direct instruction. Once each step and behavior is mastered, then during the day, when the functional routine occurs, the instructor should provide the needed instruction and support for the individual to participate and complete each step of the routine.
5. Progress monitoring. Consistently monitor the ASD individual's successful and independent completion of the functional routine. If the individual has mastered the routine/related skills, restart the process, and identify a new functional routine. If not, return to instruction and reevaluate the instructional strategies.

The teaching of functional routines is typically carried out one-to-one or in a caregiver-coaching model. The particular routine that is taught will be determined by the functional needs of the individual within the particular context (e.g., home, school, or community).

Efficacy Information

There is limited efficacy data available for teaching functional routines.

Outcome Measurement

Outcome measurement for functional routines is related to the specific behaviors that are needed to successfully complete or participate in the routine.

These behaviors have typically been identified through the task analysis completed prior to teaching the functional routine. See further information described in the section “[Treatment Procedures](#).”

Qualifications of Treatment Providers

There are no specific qualifications for treatment providers. Treatment providers can be teachers, speech-language pathologists, caregivers, counselors, behavior therapists, job coaches, or other significant adults who work and who are familiar with the individual with ASD.

See Also

- [Functional Assessment and Curriculum for Teaching Everyday Routines](#)
- [Functional Communication Training](#)

References and Reading

- Arick, J. R., Young, H. E., Falco, R. A., Loos, L. M., Krug, D. A., Gense, M. H., et al. (2003). Designing an outcome study to monitor the progress of students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 18, 75–87.
- Arick, J. R., Loos, L., Falco, R., & Krug, D. (2004). *The STAR program: Strategies for teaching based on autism research*. Austin: PRO-ED.
- Brown, L., Branston, M., Nietupski, S., Pumpian, I., Certo, N., & Gruenewald, L. (1976). A strategy for developing chronological-age-appropriate and functional curricular content for severely handicapped adolescents and young adults. *The Journal of Special Education*, 13, 81–90.
- Brown, F., Evans, I., Weed, K., & Owen, V. (1987). Delineating functional competencies: A component model. *Journal of the Association for Persons with Severe Handicaps*, 12(2), 117–124.
- Bruner, J. (1975). The ontogenesis of speech acts. *Journal of Child Language*, 2, 1–9.
- Constable, C. M. (1986). The application of scripts in the organization of language intervention contexts. In K. Nelson & A. van Kleeck (Eds.), *Children's language* (Vol. 6, pp. 44–63). Hillsdale: Erlbaum.
- Dawson, G., & Osterling, J. (1997). Early intervention in autism. In M. Guralnick (Ed.), *The effectiveness of early intervention* (pp. 307–326). Baltimore: Brookes.
- Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenon, J., et al. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The early start Denver model. *Pediatrics*, 125, e17–e23.
- Iovannone, R., Dunlap, G., Huber, H., & Kincaid, D. (2003). Effective educational practices for students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 18, 150–165.
- Kashinath, S., Woods, J., & Goldstein, H. (2006). Enhancing generalized teaching strategy use in daily routines by parents of children with autism. *Journal of Speech, Language, and Hearing Research*, 49, 466–485.
- Kern, P., Wolery, M., & Aldridge, D. (2007). Use of songs to promote independence in morning greeting routines for young children with autism. *Journal of Autism and Developmental Disorders*, 37, 1264–1271.
- Landa, R. J., Holman, K. C., O'Neill, A. H., & Stuart, E. A. (2011). Intervention targeting development of socially synchronous engagement in toddlers with autism spectrum disorder: A randomized controlled trial. *Journal of Child Psychology and Psychiatry*, 52, 13–21.
- Mancil, G. (2006). Functional communication training: A review of the literature related to children with autism. *Education and Training in Developmental Disabilities*, 41, 213–224.
- McClannahan, L. E., & Krantz, P. J. (1999). *Activity schedules for children with autism: Teaching independent behavior*. Bethesda: Woodbine House.
- McLean, J. E., & Snyder-McLean, L. (1987). Form and function of communicative behaviour among persons with severe developmental disabilities. *Australia and New Zealand Journal of Developmental Disabilities*, 13, 83–98.
- Nelson, K. (1986). *Event knowledge: Structure and function in development*. Hillsdale: Erlbaum.
- Ninio, A., & Bruner, J. (1978). The achievement and antecedents of labeling. *Journal of Child Language*, 5, 1–5.
- Ogletree, B. T., Oren, T., & Fischer, M. A. (2007). Examining effective intervention practices for communication impairment in autism spectrum disorder. *Exceptionality*, 15(4), 233–247.
- Pellecchia, M., Connell, J. E., Beidas, R. S., Xie, M., Marcus, S. C., & Mandell, D. S. (2015). Dismantling the active ingredients of an intervention for children with autism. *Journal of Autism and Developmental Disorders*, 45, 2917–2927.
- Pretti-Fontczak, K., & Bricker, D. (2004). *An activity-based approach to early intervention* (3rd ed.). Baltimore: Brookes.
- Prizant, B. M., Wetherby, A. M., Rubin, E., Laurent, A. C., & Rydell, P. J. (2006). *The SCERTS model: A comprehensive educational approach for children with autism spectrum disorders* (Programming planning and intervention, Vol. II). Baltimore: Brookes.
- Shatz, M. (1983). Communication. In P. H. Mussen (Ed.), *Handbook of child psychology* (Cognitive development, Vol. 3, pp. 841–889). New York: Wiley.
- Siegel-Causey, E., & Guess, D. (1989). *Enhancing non-symbolic communication interactions among learners with severe disabilities*. Baltimore: Brookes.

- Snell, M. E. (1983). Functional reading. In M. E. Snell (Ed.), *Systematic instruction of the moderately and severely handicapped* (pp. 445–487). Columbus: Merrill.
- Stahmer, A. C., Rieth, S., Lee, E., Reisinger, E. M., Mandell, D. S., & Connell, J. E. (2015). Training teachers to use evidence-based practices for autism: Examining procedural implementation fidelity. *Psychology in Schools*, 52, 181–195.
- Sussman, F. (1999). *More than words: A guide to helping parents promote communication and social skills*. Toronto: Hanen Centre.
- Snyder-McLean, L., Solomonson, B., McLean, J. E., & Sack, S. (1984). Structuring joint action routines: A strategy for facilitating communication and language development in the classroom. *Seminars in Speech and Language*, 5, 213–228.
- Wilcox, B., & Bellamy, G. T. (1982). *Design of high school programs for severely handicapped students*. Baltimore: Paul Brookes.
- Yoder, P. J., & Davies, B. (1992). Do children with developmental delays use more frequent and diverse language in verbal routines? *American Journal on Mental Retardation*, 97, 197–208.

Functional Speech

► Verbal Communication

Functionally Equivalent Alternative Behavior

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Synonyms

[Replacement behavior](#)

Definition

Functionally equivalent alternative behaviors, or functionally equivalent replacement behaviors, are desirable/acceptable behaviors that achieve the same outcome as a less desirable problem behavior. For example, an individual may shout

in order to gain his or her parent's attention when the parent is otherwise occupied. An alternative behavior that could serve the same function might be to say "Excuse me." In order to determine an appropriate alternative behavior, one must first understand the function of the problem behavior. This requires completion of a functional behavior assessment. Once the function of the problem behavior is clearly articulated, an appropriate alternative response, or repertoire of alternative responses, can be selected that are equal in function to the targeted challenging behavior. Alternative responses should be selected based on their social validity and should be more efficient than the challenging behavior. For example, saying "excuse me" and raising one's hand are both socially acceptable ways to gain attention and thus represent potential alternative behaviors. On the other hand, circling a person three times silently may be another way to gain attention without shouting but should not be selected because the behavior lacks social validity. Once an alternative behavior is selected, the individual should be specifically taught to engage in the behavior and then reinforced for doing so. In the current example, the child would be taught to say "excuse me" and then reinforced for doing so. In order to make the alternative behavior more efficient than the problem behavior, the problem behavior should not be reinforced or should be reinforced less often. In the example cited, the parent would consistently provide attention to the child when he or she says "excuse me" but ignore him or her when he or she shouts. In another example, a learner who engages in hitting others (problem behavior) in order to escape work demands (function) may be taught a functionally equivalent alternative behavior of requesting a break. The behavior of requesting a break allows the individual to meet his or her need (escaping the demand) without engaging in a socially inappropriate response. Approaches to intervention that seek to eliminate problem behaviors without teaching functionally equivalent alternative behaviors are ill advised as the individual will ultimately select his or her own ways to meet the needs that the extinguished problem behavior was previously serving. For example, a child who

shouts at his or her parent to gain attention could be taught to stop shouting for attention if the shouting is consistently ignored but over time he or she may begin hitting his or her parent in order to solicit attention if he or she is never taught what to do instead (e.g., say “excuse me”). Behavior support plans require explicit programming that addresses functionally equivalent alternative behaviors in order to best address individual needs. This includes specific teaching procedures for the alternative behaviors and plans to systematically reinforce the alternative behaviors when they occur.

See Also

- [Differential Reinforcement](#)
- [Functional Behavior Assessment](#)
- [Functional Communication Training](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Glasberg, B. A. (2008). *Stop that seemingly senseless behavior! FBA-based interventions for people with autism*. Bethesda: Woodbine House.
- Scott, T. M., et al. (2005). An examination of the relation between functional behavior assessment and selected intervention strategies with school-based teams. *Journal of Positive Behavior Interventions*, 7, 205–215.

Fusiform Gyrus (FG)

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Definition

The fusiform gyrus (FG) is a brain region consisting of occipitotemporal cortex (temporal lobe) that corresponds to Brodmann area 37. It is involved in high-level visual processing, specifically object recognition and category identification, of complex stimuli such as faces. Individuals

with ASD demonstrate deficits in certain cognitive abilities for which the fusiform gyrus has been implicated – most notably and socially relevant being face processing – as well as abnormalities in the fusiform gyrus itself.

Historical Background

The fusiform gyrus is a brain region involved in high-level visual processing. Visual processing is organized hierarchically and involves many brain regions working together to transform information provided by the external world into internal representations. Early low-level visual processing occurs milliseconds after light hits the retina and distinguishes basic, isolated features such as orientation, spatial frequency, and color. Later high-level visual processing combines information from lower levels of processing and culminates in identification, recognition, and categorization of objects and actions. Visual processing begins in occipital cortex and proceeds anteriorly via ventral and dorsal pathways; the highest levels of processing occur in temporal cortex. The ventral and dorsal pathways are hypothesized to represent the processing of “what” and “where/how” information, respectively, and are correspondingly associated with “perception” and “action” (Underleider and Mishkin 1982). The FG has been shown to be involved in numerous ventral stream processes, including high-level visual processes that entail semantic encoding.

The initial discoveries concerning specific functions of the FG arose from studies of primates (Van Essen et al. 1992) and patients with brain lesions or psychological disorders (Mesulam 1994), both of which have revealed the anatomy of the mammalian visual system. First, single-unit recordings from the inferotemporal cortex in macaques found populations of neurons that responded specifically to faces (Gross et al. 1972). Then, electrophysiological studies in humans suggested similar cortical specialization. Recordings from the same brain region in epilepsy patients with implanted subdural electrodes showed large N200 potentials elicited by faces but not scrambled faces, cars, or butterflies

(Allison et al. 1994). These studies were consistent with an older study of brain-injured patients that showed the same brain region seemed to be involved in recognizing faces but not houses (Yin 1970). Additional studies of patients with damage to occipitotemporal cortex of the right hemisphere, who had selectively lost the ability to recognize faces, further supported the notion that this region is involved in face processing (DeRenzi and Saetti 1997). At the same time, research in several areas, including cognitive psychology, neuropsychology, neurophysiology, and computational vision, converged to suggest the differentiation of face and object recognition as qualitatively different processes and perhaps even entailing distinct brain areas.

As research continued building an understanding of the function of the FG, a growing body of evidence indicated that face processing was not its only function. Neuroimaging became more popular as a technique to study brain function, and as a result, specific brain regions could be much more easily implicated in a wide range of cognitive processes, including processing shape and surface properties such as color and texture (Barrett et al. 2001), number and word recognition (Allison et al. 1994), and within-category identification (Gauthier et al. 2000). Yet while these studies showed that the FG is involved in myriad visual process, individuals with ASD do not characteristically demonstrate deficits in the lower level visual processes (e.g., shape and surface properties); rather, they often demonstrate difficulty integrating more basic features into configural representations and processing certain higher level visual categories, specifically the socially relevant category of faces. Early research investigating face processing in ASD showed that, unlike typically developing peers, children with ASD fail to show both a “face inversion effect” – a disproportionate difficulty recognizing upside down faces – and a face “decomposition effect” – reduced recognition of face components or partially obscured faces (Langdell 1978). That is, children with ASD do not show more difficulty recognizing upside down faces than upright faces, nor do they show more difficulty recognizing face components or partially obscured faces than

whole faces. These abnormalities in face processing suggest that, unlike typical counterparts, individuals with ASD do not process faces holistically (Freire et al. 2000), but instead, they may process faces using feature-based strategies similar to how typical people process objects.

Given that ASD is at its core a social disorder and that face perception is among the most vital of social functions, it is especially noteworthy that individuals with ASD were observed to process faces atypically. But it remained uncertain whether these face-processing abnormalities stem from a primary brain abnormality, a primary behavioral difference (i.e., lowered attention to faces), or some combination and subsequent interaction thereof. Studies found that individuals with ASD already show lower attention to faces as young as 1 year of age (Osterling and Dawson 1994); by childhood, they show more difficulty than typically developing children discriminating and recognizing (Boucher and Lewis 1992) faces, and through adolescence and adulthood, they continue to show poorer recognition of faces than typically developing counterparts (McPartland et al. 2004). In order to disentangle the possible origins of these behavioral deficits in face processing – whether they arise from primary brain abnormalities or primary behavioral differences – later studies used neuroimaging techniques to examine the brain bases of face processing in ASD. Functional magnetic resonance imaging (fMRI) studies showed that, when viewing neutral faces, individuals with ASD exhibit lowered fusiform gyrus activity compared to typical counterparts (Schultz et al. 2000). Furthermore, Schultz et al. (2000) suggested that individuals with ASD process faces more similarly to how typical counterparts process objects, with decreased FG activity and increased inferior temporal gyrus activity. Event-related potential (ERP) studies showed that children with ASD, but not typically developing children, fail to exhibit differential brain activity for familiar versus unfamiliar faces (Dawson et al. 2002); similarly, adolescents and adults with ASD, but not typical counterparts, fail to exhibit differential brain activity for upright versus inverted faces (McPartland et al. 2004). Moreover, McPartland et al. (2004) found that

individuals with ASD exhibit longer latencies to faces and that processing speed positively correlates with face recognition ability. These face-sensitive ERPs have been localized to the FG. Finally, one recent study found that individuals with ASD have smaller and fewer neurons in the FG (van Kooten et al. 2008).

Current Knowledge

Initial investigations of the FG revealed its involvement in face processing. While it has since been implicated in many additional roles, current investigations continue to focus on the role of the FG in face processing. There are presently three prevailing theories for the neural organization of face processing involving the FG. One is that the FG is specialized for face processing, which is modular and domain specific (Kanwisher et al. 1997). A second theory is that the FG is not specialized for faces per se but for objects with which one has experience or expertise; most humans have extensive experience with and are thus “experts” of faces, making the FG specialized for faces by default. This theory proposes that the FG is domain general, and it may also serve to differentiate between specific levels of categorization, particularly subordinate levels of identification (Gauthier et al. 2000). A third theory is that the FG and, more generally, inferotemporal cortex are neither specialized for face nor object processing but for face-like features. That is, the brain region containing and surrounding the FG is topographically arranged according to visual features of objects, and the FG “happens to be” where types of visual features associated with faces are processed (Haxby 1999).

It still remains uncertain as to which, if any, of these theories most accurately describes the role of the FG as it relates to face processing. Moreover, these theories do not comprehensively reflect face-processing research. Yet other studies suggest that face processing and the role of the FG are affected by the manner in which one conceives a visual percept. Specifically, thinking about a visual percept in a social context or imagining faces may be sufficient for FG activation. Schultz

et al. (2003) found that the FG activated for geometric shape stimuli behaving in social but not mechanistic ways, suggesting that FG activity is dependent upon a social framework. While this interpretation is inconsistent with any of the above three theories, it is consistent with findings from other studies. These unreconciled findings have important implications for the source of social dysfunction in ASD. A major question is whether the observed abnormalities in face processing in ASD are causes or effects of the diagnostic social deficits in ASD. While some studies argue the former (Trepagnier 1995), most evidence supports the latter; children with ASD may have experienced typical development with appropriate attention to faces early on followed by autistic regression, and individuals with ASD exhibit abnormalities in widely distributed brain systems, some of which are unrelated to face processing (Kemper and Bauman 1998). A larger body of research suggests that face-processing difficulties stem from developmental abnormalities in social interest and attention. Individuals with ASD may exhibit face-processing deficits because they do not acquire adequate experience with faces due to a lack of social motivation (Dawson et al. 2002). This theory is consistent with both the domain-specific and domain-general theories of face processing aforementioned. Another explanation of face-processing abnormalities in ASD that has not been empirically supported is that they stem from early developmental abnormalities in the FG or, more generally, inferotemporal cortex.

Schultz and colleagues (2005) have proposed that face perception and social cognitive skills are part of a network supported by a system comprised of the amygdala and FG and that abnormalities in this brain system, which in turn cause deficits in the social perceptual network, may play a large role in the etiology of autism. They argue that the best neuroimaging evidence for brain-based differences in the ASDs thus far involves FG hypoactivation, and they describe three possible interdependent factors that might moderate the degree of FG activity in ASD. First, they suggest an attentional factor: lowered attention to a visual stimulus reduces FG activity. Second, they suggest a perceptual skill factor:

chronically reduced attention to social stimuli, particularly faces, leads to reduced perceptual skill and reduced FG activity. Third, they suggest a social knowledge factor: the FG may encode social knowledge such that social judgment not necessarily involving faces may strongly activate the FG, and therefore, conversely, reduced social knowledge may reduce social judgments, which in turn may result in FG hypoactivation.

Two recent studies have provided a confound to this model. Dalton et al. (2005) found that when individuals with ASD view faces, amygdala and FG activation are strongly and positively correlated with time spent fixating the eyes. In addition, eye fixation is strongly and positively associated with amygdala activation, suggesting a heightened emotion response associated with eye fixation. Similarly, Morris, Pelphrey, and McCarthy (2007) found that when typically developing individuals view faces, the scanpath one follows alters FG activation such that greater focus on the eyes (typical scanpath) as opposed to other face regions (atypical scanpath) elicits greater FG activation. These findings indicate that, in addition to degree of attention, the specific features attended to within social stimuli (i.e., faces) affect FG activation. These findings therefore imply that controlling gaze fixation for individuals with ASD when viewing social stimuli may serve to alter the neural mechanisms underlying their social deficits.

Future Directions

Further research concerning FG function will continue to disentangle the various models of face processing and, concerning the etiology of ASD, whether FG abnormalities are primary or whether deficits in social motivation and ability are primary to deficits in social skills. In addition, future neuroimaging studies will seek to investigate the effects on FG activation of various scanpaths for social stimuli in individuals with ASD. These studies will contribute to a refining of the understanding of both the causes of and potential treatments for ASD.

See Also

- [Face Perception](#)
- [Face Recognition](#)
- [Occipital Lobe](#)

References and Reading

- Allison, T., McCarthy, G., Nobre, A., Puce, A., & Belger, A. (1994). Human extrastriate visual-cortex and the perception of faces, words, numbers, and colors. *Cerebral Cortex*, 4(5), 544–554.
- Barrett, N. A., Large, M. M., Smith, G. L., Michie, P. T., Karayanidis, F., Kavanagh, D. J., et al. (2001). Human cortical processing of colour and pattern. *Human Brain Mapping*, 13(4), 213–225.
- Boucher, J., & Lewis, V. (1992). Unfamiliar face recognition in relatively able autistic children. *Journal of Child Psychology and Psychiatry*, 33, 843–859.
- Dalton, K. M., Nacewicz, B. M., Johnstone, T., Schaefer, H. S., Gernsbacher, M. A., Goldsmith, H. H., et al. (2005). Gaze fixation and the neural circuitry of face processing in autism. *Nature Neuroscience*, 8(4), 519–526.
- Dawson, G., Carver, L., Meltzoff, A. N., Panagiotides, H., McPartland, J., & Webb, S. J. (2002). Neural correlates of face and object recognition in young children with autism spectrum disorder, developmental delay, and typical development. *Child Development*, 73(3), 700–717.
- DeRenzi, E., & Saetti, M. C. (1997). Associative agnosia and optic aphasia: Qualitative or quantitative difference? *Cortex*, 33(1), 115–130.
- Freire, A., Lee, K., & Symons, L. A. (2000). The face-inversion effect as a deficit in the encoding of configural information: Direct evidence. *Perception*, 29(2), 159–170.
- Gauthier, I., Tarr, M. J., Moylan, J., Skudlarski, P., Gore, J. C., & Anderson, A. W. (2000). The fusiform “face area” is part of a network that processes faces at the individual level. *Journal of Cognitive Neuroscience*, 12(3), 495–504.
- Gross, C. G., Rochamir, C. E., & Bender, D. B. (1972). Visual properties of neurons in inferotemporal cortex of macaque. *Journal of Neurophysiology*, 35(1), 96–111.
- Haxby, J. V. (1999). Human neural systems for face perception. *Biological Psychiatry*, 45(8S), 102S–102S.
- Kanwisher, N., McDermott, J., & Chun, M. M. (1997). The fusiform face area: A module in human extrastriate cortex specialized for face perception. *Journal of Neuroscience*, 17(11), 4302–4311.
- Kemper, T. L., & Bauman, M. (1998). Neuropathology of infantile autism. *Journal of Neuropathology and Experimental Neurology*, 57(7), 645–652.

- Langdell, T. (1978). Recognition of faces – Approach to study of autism. *Journal of Child Psychology and Psychiatry*, 19(3), 255–268.
- McPartland, J., Dawson, G., Webb, S. J., Panagiotides, H., & Carver, L. J. (2004). Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 45(7), 1235–1245.
- Mesulam, M. M. (1994). Higher visual functions of the cerebral cortex and their disruption in clinical practice. In D. M. Albert & F. A. Jakobiec (Eds.), *The principles and practice of ophthalmology: The Harvard system* (pp. 2640–2653). Philadelphia: W.B. Saunders.
- Morris, J. P., Pelphrey, K. A., & McCarthy, G. (2007). Controlled scanpath variation alters fusiform face activation. *Social Cognitive and Affective Neuroscience*, 2(1), 31–38.
- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism – A study of 1st birthday home videotapes. *Journal of Autism and Developmental Disorders*, 24(3), 247–257.
- Schultz, R. T. (2005). Developmental deficits in social perception in autism: the role of the amygdala and fusiform face area. *International Journal of Developmental Neuroscience*, 23(2–3), 125–141.
- Schultz, R. T., Gauthier, I., Klin, A., Fulbright, R. K., Anderson, A. W., Volkmar, F., et al. (2000). Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and Asperger syndrome. *Archives of General Psychiatry*, 57(4), 331–340.
- Schultz, R. T., Grelotti, D. J., Klin, A., Kleinman, J., Van der Gaag, C., Marois, R., et al. (2003). The role of the fusiform face area in social cognition: Implications for the pathobiology of autism. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, 358(1430), 415–427.
- Trepagnier, C. (1995). Infant social gaze in autism. In *Proceedings of the 1995 National Conference on Autism* (pp. 453–457), 493.
- Trepagnier, C. (1996). A possible origin for the social and communicative deficits of autism. *Focus on Autism and Other Developmental Disabilities Fall, 11*, 170–182.
- Underleider, L. G., & Mishkin, M. (1982). In D. J. Ingle, M. A. Goodale, & R. J. W. Mansfield (Eds.), *Analysis of visual behavior* (pp. 549–586). Cambridge, MA: MIT Press.
- Van Essen, D. C., Anderson, C. H., & Felleman, D. J. (1992). Information processing in the primate visual system, an integrated systems perspective. *Science*, 255, 419–423.
- van Kooten, I. A. J., Palmen, S. J. M. C., von Cappeln, P., Steinbusch, H. W. M., Korr, H., Heinsen, H., et al. (2008). Neurons in the fusiform gyrus are fewer and smaller in autism. *Brain*, 131, 987–999.
- Yin, R. K. (1970). Face recognition by brain-injured patients: A dissociable ability? *Neuropsychologia*, 8(4), 395–402.

G

Gabapentin

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Synonyms

Fanatrex; Gabarone; Horizant; Neurontin

Definition

Gabapentin is an anticonvulsant medication that is also used for the treatment of anxiety. It is also used for the treatment of bipolar illness. The mechanism of action of gabapentin is complicated and not completely understood. It appears to assist with the decreased excitability in the brain. However, it does not appear to directly affect the GABA system in the brain. Recent evidence suggests that it exerts its positive effects by turning down calcium channels. This would fit with its effect to decrease excitability in the brain.

There are no studies of gabapentin in children with autism spectrum disorders. However, it is used as an anticonvulsant in children.

References and Reading

Martin, A., Scahill, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.

Gabarone

► [Gabapentin](#)

GAD

► [General Anxiety](#)
► [Generalized Anxiety Disorder](#)

Games with Rules

► [Play](#)

Gaming Technologies in Rehabilitation of Individuals with Autism

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Children with autism spectrum disorder (ASD) have difficulties in social interactions, affecting their social participation and quality of life. There is a growing use of assistive technologies to support social participation of children with ASD. Despite potential improvements of social skills after using these programs, there are concerns on technology dependence and generalization of learned skills. It is still unclear to what extent virtual environments are effective and what populations on the autism spectrum are the suitable candidates to take advantage of using virtual gaming programs. This manuscript highlights trends in current literature and recommends ideas for future studies.

Definition and Historical Background

Autism spectrum disorder (ASD) is a childhood neurodevelopmental condition that is currently being diagnosed in about 1 in 59 children. Children with ASD may experience difficulties in communication and socio-emotional skills, which affect their daily functioning (American Psychiatric Association 2013; Baron-Cohen et al. 2001; Simonoff et al. 2008). Children with ASD have challenges with understanding and applying social rules and interpreting verbal and nonverbal social cues (Kelly et al. 2008; Ghanouni et al. 2019). All these factors can have repercussions for the child's ability to socialize, develop relationships, and make friends. As children get older and gain insights about these significant deficits, they demonstrate decreased self-

esteem in communicating with their peers (Lopata et al. 2008). This may act as a vicious cycle that precludes children from acquiring social skills to interact appropriately with peers.

The delivery of early and effective therapeutic services is crucial to enhance the outcomes and functioning of clients with ASD (Eikeseth 2009; Matson and Smith 2008). Gaming programs have been widely used in the fields of education and rehabilitation to address a diverse range of clients' needs. These programs provide an opportunity for practicing in a fun environment with gradual exposure to stimuli, providing consistency and stimulus control, giving real-time feedback, and augmenting attention to performance (Parsons and Cobb 2011). These programs can provide a simulated environment in which the intensity of training, level of difficulty, and amount of feedback can be adjusted. Virtual gaming programs can simulate real-life situations, which increases the probability of transferring learned skills to real-life contexts, and provide a safe and risk-free environment to practice skills that may be dangerous or risky to complete in real-life contexts (Bellani et al. 2011). This allows for the creation of an individualized program that keeps clients motivated to endure the program.

Current Knowledge

Recently, there have been rapid advancements in developing low-cost technologies in rehabilitation, including computer-based programs, virtual reality games, motion-tracking games, and video games (Anderson-Hanley et al. 2011; Parsons and Cobb 2011). However, developing an appropriate program for children with ASD is a complex task due to the heterogeneity of the disorder and co-occurring conditions (Wilczynski et al. 2007). Not every program can fit the broad range of needs that may be observed in individuals on the spectrum. It is necessary to tailor each program to address the symptoms and deficits that a child with ASD demonstrates. In addition to the importance of individualized programs, the involvement of clients in making decisions about the program to suit their family style and preferences

would maximize outcomes (Elwyn et al. 2000; Guadagnoli and Ward 1998).

Looking at the literature, there has been mixed findings on potential effects of using video game and virtual reality programs in individuals with ASD (Parsons and Cobb 2011). Some of the literature did not show significant results, whereas others demonstrated significant improvements in socio-emotional skills. Gaming programs were demonstrated to be effective for improving specific social skills, such as social initiation/interaction, verbal communication, and facial affect and emotion recognition for children diagnosed with ASD. Using humanoid or cartoon characters in computer-based intervention programs has been shown to potentially improve emotion recognition in individuals with ASD (Golan et al. 2010; Hopkins et al. 2011; Tanaka et al. 2010). Increased perspective-taking and a higher level of social interaction within short-term usage of the computer-based interventions, as complementary programs, hold great promise for individuals with ASD (Hughes 2014; Golan et al. 2010; Moore et al. 2000; Mesa-Gresa et al. 2018). The majority of the findings demonstrated positive trends for social skill development but lack the rigor and power to reach levels of significance.

Although there was no reported negative experience of using these gaming programs, there are concerns on the dosage of the program and the extent to which users of video games can generalize the learned skills to real life. If the task can only be performed in a virtual setting and in a controlled environment, the benefits of the program should be justified. Using the virtual settings, which include real-life scenarios, helps individuals with ASD relate to these scenarios when they face them in real life. Adjusting the level of difficulty of the task, as individuals with ASD progress over time, keeps them engaged in the activities. Modification of sensory, visual, and auditory modalities should also be considered when working with individuals with sensory problems. Given the interest of individuals with ASD to use virtual settings, screen time and technology dependence are other concerns (Montes 2015). The optimal goal is helping users to be independent, and not mentally reliant on technology that may affect the quality of daily

interactions. Although setting schedules might help monitor screen time, there is still a significant gap in the literature on how we can mitigate potential risks of using virtual settings and appropriate dosage of programs to facilitate learning new skills in individuals with ASD.

In addition to the content of virtual settings, another presumption is that not everyone can be an ideal candidate to use it. Being able to navigate through virtual environments, relate scenarios, and transfer learned skills to daily life requires high levels of practice, environmental support, as well as individual mental and functional capacity. Severity of disorder, selective and sustained attention, executive function, and IQ level might be factors that play significant roles for transition of learned skills from virtual settings to daily activities.

Future Direction

Identifying subgroups of individuals with ASD who have the potential to benefit from using these virtual settings is a recommendation for future research. Providing more comprehensive profiles, using brain imaging to examine areas that are activated in virtual settings as opposed to real settings, and seeking potential differences in brain functions provide additional insight in deciding which tool, when, and how they might be useful (Bohil et al. 2011; Weber et al. 2006). Given the heterogeneous nature of ASD and disperse behavioral studies, brain imaging techniques such as EEG can advance knowledge and provide additional understanding of the extent in which a simulated virtual environment is similar to in vivo settings. Following up with participants of the study after receiving gaming programs to examine the degree in which the learned skilled can be generalized to other settings and/or maintained after a period of time is also warranted. Additionally, future research will need to consider more rigorous studies using stronger study designs by increasing sample size, addressing co-intervention, comparing different types of gaming programs, and using outcome measures with strong psychometric properties. As a result, informed modifications can be made

to potentially optimize learning in virtual settings for individuals with ASD.

See Also

- ▶ [Augmentative and Assistive Technology](#)
- ▶ [Computer-Based Intervention Assistive Technology](#)
- ▶ [Video Games, Use of](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5* (5th ed.). Washington, DC: American Psychiatric Association.
- Anderson-Hanley, C., Tureck, K., & Schneiderman, R. L. (2011). Autism and exergaming: Effects on repetitive behaviors and cognition. *Psychology Research and Behavior Management*, 4, 129–137.
- Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The “Reading the mind in the eyes” test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry*, 42, 241–251.
- Bellani, M., Fornasari, L., Chittaro, L., & Brambilla, P. (2011). Virtual reality in autism: State of the art. *Epidemiology and Psychiatric Sciences*, 20, 235–238.
- Bohil, C. J., Alicea, B., & Biocca, F. A. (2011). Virtual reality in neuroscience research and therapy. *Nature Reviews. Neuroscience*, 12, 752–762.
- Eikeseth, S. (2009). Outcome of comprehensive psycho-educational interventions for young children with autism. *Research in Developmental Disabilities*, 30, 158–178.
- Elwyn, G., Edwards, A., Kinnersley, P., & Grol, R. (2000). Shared decision making and the concept of equipoise: The competences of involving patients in healthcare choices. *British Journal of General Practice*, 50, 892–899.
- Ghanouni, P., Jarus, T., Zwicker, J., Lucyshyn, G., Chauhan, J., & Moir, S. (2019). Perceived barriers and existing challenges in participation of children with autism Spectrum disorders: “He did not understand and no one Else seemed to understand him”. *Journal of Autism and Developmental Disorders*, 49, 3136–3145.
- Golan, O., Ashwin, E., Granader, Y., McClintock, S., Day, K., Leggett, V., & Baron-Cohen, S. (2010). Enhancing emotion recognition in children with autism spectrum conditions: An intervention using animated vehicles with real emotional faces. *Journal of Autism and Developmental Disorders*, 40, 269–279.
- Guadagnoli, E., & Ward, P. (1998). Patient participation in decision-making. *Social Science & Medicine*, 47, 329–339.
- Hopkins, I. M., Gower, M. W., Perez, T. A., Smith, D. S., Amthor, F. R., Wimsatt, F. C., & Biasini, F. J. (2011). Avatar assistant: Improving social skills in students with an ASD through a computer-based intervention. *Journal of Autism and Developmental Disorders*, 41, 1543–1555.
- Hughes, D. E. (2014). *The design and evaluation of a video game to help train perspective-taking and empathy in children with autism spectrum disorder*. Orlando: University of Central Florida.
- Kelly, A., Garnett, M., Attwood, T., & Peterson, C. (2008). Autism spectrum symptomology in children: The impact of family and peer relationships. *Journal of Abnormal Child Psychology*, 36, 1069–1108.
- Lopata, C., Volker, M. A., Putnam, S. K., Thomeer, M. L., & Nida, R. E. (2008). Effect of social familiarity on salivary cortisol and self-reports of social anxiety and stress in children with high functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38, 1866–1877.
- Matson, J. L., & Smith, K. R. (2008). Current status of intensive behavioral interventions for young children with autism and PDD-NOS. *Research in Autism Spectrum Disorders*, 2, 60–74.
- Mesa-Gresa, P., Gil-Gómez, H., Lozano-Quilis, J.-A., & Gil-Gómez, J.-A. (2018). Effectiveness of virtual reality for children and adolescents with autism Spectrum disorder: An evidence-based systematic review. *Sensors*, 18, 2486.
- Montes, G. (2015). Children with autism spectrum disorder and screen time: Results from a large, nationally representative US study. *Academic Pediatrics*, 16, 122–128.
- Moore, D., McGrath, P., & Thorpe, J. (2000). Computer-aided learning for people with autism—a framework for research and development. *Innovations in Education and Teaching International*, 37, 218–228.
- Parsons, S., & Cobb, S. (2011). State-of-the-art of virtual reality technologies for children on the autism spectrum. *European Journal of Special Needs Education*, 26, 355–366.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, 47, 921–929.
- Tanaka, J. W., Wolf, J. M., Klaiman, C., Koenig, K., Cockburn, J., Herlihy, L., . . . Schultz, R. T. (2010). Using computerized games to teach face recognition skills to children with autism spectrum disorder: The Let’s face it! Program. *Journal of Child Psychology and Psychiatry*, 51, 944–952.
- Wang, X., Laffey, J., Xing, W., Galyen, K., & Stitche, J. (2017). Fostering verbal and non-verbal social interactions in a 3D collaborative virtual learning

environment: a case study of youth with autism spectrum disorders learning social competence in iSocial. *Education Technology Research Development*, 65, 1015–1039.

Weber, R., Ritterfeld, U., & Mathiak, K. (2006). Does playing violent video games induce aggression? Empirical evidence of a functional magnetic resonance imaging study. *Media Psychology*, 8, 39–60.

Wilczynski, S. M., Menousek, K., Hunter, M., & Mudgal, D. (2007). Individualized education programs for youth with autism spectrum disorders. *Psychology in the Schools*, 44, 653–666.

Gamma-Aminobutyric Agonist

► [Zolpidem](#)

Ganglioside Lipidosis

► [Gangliosidoses](#)

Gangliosidoses

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Synonyms

[Autosomal recessive disorder](#); [Ganglioside lipidosis](#); [Lysosomal lipid storage disorder](#); [Sphingolipidoses](#)

Definition

Gangliosidosis contains different types of lipid storage disorders caused by the accumulation of lipids called gangliosides. Gangliosides are made and biodegraded rapidly in early life as the brain develops. The two main types of gangliosidosis are GM1 gangliosidosis and GM2 gangliosidosis, and these have two distinct genetic causes. Both

types are autosomal recessive, meaning that both copies of the gene in each cell have the mutation, and affect males and females equally. This disorder occurs in 1 in 100,000–200,000 newborns.

GM1 gangliosidosis is an inherited disorder caused by mutations in the GLB1 gene, which is a gene that provides instructions for making an enzyme called beta-galactosidase and is located in the lysosomes. Beta-galactosidase helps break down several molecules including the GM1 ganglioside and is important for normal functioning of nerve cells in the brain. Buildup of GM1 gangliosides leads to destruction of nerve cells in the brain and spinal cord. GM1 gangliosides are inherited, autosomal recessive sphingolipidoses, resulting from marked deficiency of acid beta-galactosidase. Diagnosis can be made by either enzyme analysis of the beta-galactosidase enzyme or by molecular genetic testing of the GLB1 gene. Enzyme activity may determine diagnosis, but it is not necessarily predictive of carrier status in the relatives of affected people. Prenatal diagnosis of GM1 gangliosidosis is possible by measurement of acid beta-galactosidase in cultured amniotic cells. There is no effective medical treatment, other than symptomatic and supportive treatments. Symptomatic treatment for neurological signs and symptoms, such as anticonvulsants for seizures, does not alter progression of the condition. Supportive treatments include proper nutrition and hydration. Additionally, there is potential benefit from physical and occupational therapy. There is active research in areas of enzyme replacement and gene therapy, but this has not yet been advanced to human trials.

The three types of GM1 from most to least severe are Early Infantile GM1, Late Infantile GM1, and Adult GM1. The onset of Early infantile GM1 occurs shortly after birth and usually becomes apparent by the age of 6 months. Symptoms include neurodegeneration, seizures, liver and spleen enlargement, coarsening of facial features, skeletal irregularities, joint stiffness, distended abdomen, muscle weakness, exaggerated startle response to sound, and problems with gait. Diagnosed children may be deaf and blind by age 1. Half of patients with

GM1 develop cherry-red spots in the eye. These children often die by age 3 from either cardiac complications or pneumonia. The onset of Late Infantile GM1 is usually between 1 and 3 years old. Neurological symptoms include ataxia, seizures, dementia, and difficulties with speech. These children also experience developmental regression, but no cherry-red spots, distinctive facial features, or enlarged organs as seen in Early Infantile GM1. The progression is slower than Early Infantile GM1 but still causes a shortened life expectancy. The onset of Adult GM1 is between 3 and 30 years old. Typical symptoms are muscle atrophy, neurological complications, corneal clouding, and dystonia. This is the mildest form of GM1 and life expectancy is quite variable.

GM2 gangliosidosis results from mutations in at least 1 of 3 recessive genes (HEXA, HEXB, GM2A). The products of these three genes are the alpha subunits of b-hexosaminidase A, the beta subunits of Hex A, and the GM2 activator protein. GM2 encompasses a group of three related genetic disorders that results from a deficiency of beta-hexosaminidase, an enzyme found in the lysosomes that catalyzes the biodegradation of gangliosides. In GM2, a harmful quantity of gangliosides accumulates in the brain's nerve cells, leading to premature death of cells. The signs and symptoms become apparent in infancy. Infants appear normal until 3–6 months of age when development slows and muscles used for movement weaken. Infants often develop exaggerated startle reaction to loud noises, as well as seizures, vision and hearing loss, intellectual disability, paralysis, and cherry-red spots in the eye as the disease progresses. All three GM2 disorders are extremely rare in the general population. All are associated with failure of the same metabolic pathway and have the same outcome, but each represents a distinct molecular point of failure in a subunit that is required for activation of the enzyme.

Tay-Sachs disease is the most common of the GM2 gangliosidoses. It is an autosomal recessive genetic disorder caused by a mutation in the HEXA gene and results in GM2 ganglioside storage in the central nervous system (CNS).

Tay-Sachs usually develops around 6 months of age and results in death by age 4. Sandhoff disease is an autosomal recessive metabolic disorder caused by mutations on chromosome 5 in the HEXB gene. The most common form is infantile Sandhoff disease, which is fatal in early childhood. Sandhoff disease is clinically indistinguishable from Tay-Sachs disease. The third type of GM2, AB Variant, is an autosomal recessive metabolic disorder caused by a mutation in the GM2A gene. The GM2A gene provides instructions for making a protein called the GM2 activator, which is a cofactor that is required for the normal function of beta-hexosaminidase A. All three types of GM2 are extremely rare and normally fatal by early childhood. There does not appear to be a correlation between clinical disease progression and survival in GM2 gangliosidosis. The main reasoning for this lack of correlation is most likely due to the impact of improved supportive care over time.

As gangliosidosis is a rare disorder, there is a deficiency of research on the relation between gangliosidosis and autism spectrum disorder (ASD). GM1 gangliosides in the cerebrospinal fluid (CSF) were found to be increased in individuals with autism compared to age-matched controls and those with non-progressive neurologic disorders. This increase of CSF gangliosides in the synaptic junctions indicates enhanced activity of the inhibitory synapses. Proper glycoprotein sialylation is necessary for synaptic function and a sialylation deficiency in the CNS is related to the occurrence of ASD.

References and Reading

- Barone, R., Sturiale, L., Fiumara, A., Palmigiano, A., Bua, R. O., Rizzo, R., ... & Garozzo, D. (2016). CSF N-glycan profile reveals sialylation deficiency in a patient with GM2 gangliosidosis presenting as childhood disintegrative disorder. *Autism Research*, 9(4), 423–428.
- Bley, A. E., Giannikopoulos, O. A., Hayden, D., Kubilus, K., Tift, C. J., & Eichler, F. S. (2011). Natural history of infantile GM2 gangliosidosis. *Pediatrics*, peds-2011.

- Breiden, B., & Sandhoff, K. (2018). Ganglioside metabolism and its inherited diseases. In *Gangliosides* (pp. 97–141). New York: Humana Press.
- Skjeldal, O. H., Sponheim, E., Lekman, A., & Svennerholm, L. (1994). Gangliosides in CSF in autistic children. *Pediatric Neurology*, 11(2), 174.
- Volk, B. W., Adachi, M., & Schneck, L. (1975). The gangliosidoses. *Human Pathology*, 6(5), 555–569.

GARS

- [Gilliam Autism Rating Scale \(GARS\)](#)

GARS-2

- [Gilliam Autism Rating Scale \(GARS\)](#)

Gastrointestinal Disorders and Autism

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Synonyms

[GI issues](#); [Nutritional issues](#)

Definition

Autism spectrum disorders (ASDs) are a group of developmental disorders characterized by impairments in social interaction; varying degrees of verbal and nonverbal communication deficits; and restricted, repetitive, and stereotyped patterns of behavior and interests. The term includes autistic disorder, Asperger disorder, and pervasive developmental disorder, not otherwise specified (PDD-NOS) (Johnson et al. 2007).

A meta-analysis representing the first rigorous evaluation of evidence regarding GI symptoms in children with ASD, quantifying the past 32 years of research, indicate greater risk of general GI symptoms among children with ASD than in those without ASD, indicating that this population may be more prone to specific symptoms of abdominal pain, constipation, and diarrhea by greater than two- to threefolds (McElhanon, 2014).

In these children, a variety of gastrointestinal dysfunctions and associated symptoms have been reported frequently. Gastrointestinal problems in individuals with ASDs can be challenging to evaluate. Clinical practice guidelines exist for the evaluation and management of ASDs by primary care and other physicians responsible for the care of individuals with ASDs but do not include routine consideration of potential gastrointestinal and other medical problems (Volkmar et al. 1999).

Many individuals with ASDs are nonverbal or minimally verbal and cannot express pain or discomfort through speech. As a result, they may not be able to communicate information about their symptoms. Even individuals with ASDs who acquire verbal communication skills may have difficulty describing subjective experiences or symptoms. In addition, the magnitude of the observed association combined with difficulties identifying and studying GI dysfunction in ASD warrants the adoption of a lower referral threshold by practitioners for evaluation and treatment by a gastroenterologist if an underlying problem is suspected.

Historical Background

Autism spectrum disorders (ASDs) are clinically heterogeneous neurodevelopmental disorders.

Gastrointestinal (GI) dysfunction is frequently cited among children with ASD, and many causal and therapeutic hypotheses of ASD involve the GI system. This includes the idea that there is a specific GI pathology associated with ASD, triggered by abnormal immune function or elevated intestinal permeability.

A great amount of controversy has surrounded this topic since publication and public awareness

in 1998 naming a new pathologic entity, “autistic enterocolitis,” (Wakefield et al. 1998) as responsible for developmental regression in 12 children after administration of the measles-mumps-rubella (MMR) vaccine. Ultimately, this research was retracted. Additional theories have posited that children with ASD are at greater risk for gluten sensitivity (Lau et al. 2013), lactase deficiencies (Williams et al. 2011), and gene variants (genetic evidence implicating genes in the METreceptor tyrosine kinase pathway in ASD) (Campbell et al. 2008). Although the presence of a unique GI pathophysiology specific to ASD’s has yet to be identified, elevated risk for GI symptoms in this population remains a critical issue in pediatric settings, because this population is significantly more likely to use GI agents and experience hospitalizations related to GI disturbance compared with peers (Croen et al. 2006).

In addition, key issues such as the prevalence and best treatment of these conditions are incompletely understood. A fundamental difficulty in recognizing and characterizing gastrointestinal dysfunction with ASDs is the communication difficulties experienced by many affected individuals. A multidisciplinary panel reviewed the medical literature with the aim of generating evidence-based recommendations for diagnostic evaluation and management of gastrointestinal problems in this patient population. The panel concluded that evidence-based recommendations are not yet available (Buie et al. 2010a). The consensus expert opinion of the panel was that individuals with ASDs deserve the same thoroughness and standard of care in the diagnostic workup and treatment of gastrointestinal concerns as should occur for patients without ASDs. Problem behaviors may be the symptoms of underlying gastrointestinal disorders. Insufficient data were available to determine whether GI symptoms often linked with an organic pathology. Questions also remain about the relative contribution of behavioral factors, such as toileting and feeding problems, to the observed association between diarrhea, constipation, and abdominal pain in ASD. An approach that identifies and treats medical problems simultaneously with behavioral symptoms may be necessary. Further research is

necessary to better characterize the nature of the gastrointestinal symptoms experienced by individuals with autism as well as the biological etiology.

Current Knowledge

Chronic Abdominal Pain

Chronic abdominal pain is defined as intermittent or constant abdominal pain that exceeds 1 or 2 months in duration. For children with ASDs, assessment may be challenging. Although underlying causes of chronic abdominal pain are frequently benign, parents are often worried that the child with an ASD is in gastrointestinal distress and that the clinician should be concerned about missing a serious disease. Generally, for children without ASDs between the ages of 4 and 18 years, functional abdominal pain can be diagnosed correctly by the primary care practitioner when alarm symptoms (e.g., involuntary weight loss, deceleration of linear growth, gastrointestinal blood loss, significant vomiting, chronic severe diarrhea, persistent right upper or right lower quadrant pain, unexplained fever, family history of inflammatory bowel disease, abnormal or unexplained physical findings) are absent, results of the physical examination are normal, and stool does not contain occult blood. There are no specific and reliable signs or symptoms that enable the health care provider to distinguish between organic and functional disorders. Children with ASD may be able to communicate distress with language, but many have atypical communication and may express abdominal pain with unusual behaviors (e.g., pressing on the abdomen and tapping on the areas of distress) or change in state of being (e.g., sleep disturbance, self-injurious behavior, and aggression) that may not be perceived as indicating the source of the discomfort (Horvath and Perman 2002). The presence of any alarm symptom should initiate an evaluation. Even in the absence of alarm symptoms, a diagnostic evaluation or empiric trial of a therapeutic intervention may be considered. Education is an important part of treatment. In the absence of alarm symptoms and after an unrevealing diagnostic evaluation and failure of empiric treatment to resolve the

symptom or behavior, it may be helpful to review with the family the child's symptoms and explain that although the pain is real, there is no evidence at present of a serious or chronic disease. The clinical picture can change over time, and individuals with ASDs should be reevaluated if their symptoms or signs change.

Constipation

Constipation is the occurrence for 2 weeks or so of a delay or difficulty in defecation. The causes of constipation are many and may be organic or nonorganic; medications can be a potential cause (e.g., opiates, phenobarbital, antacids, sucralfate, anticholinergics, antidepressants, sympathomimetics, antihypertensives) (Constipation Guidelines Committee of the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition 2006). Children with ASDs can have sensory processing abnormalities and develop stool-withholding behaviors or constipation related to altered pain responses. Even children with ASDs who have daily bowel movements may have retention of stool that is not evident to parents, teachers, or health care providers.

The evaluation of all children who present with constipation should include a thorough medical history and physical examination. Information from the history and physical examination usually enables the physician to determine if further evaluation is warranted or if functional constipation is present. Determination of what the family or child means when they use the term "constipation" is important. The history should delineate the frequency of bowel movements, the consistency and size of stools, and whether the child experiences abdominal pain. A history of stool-withholding behavior reduces the likelihood of a causative organic condition. For children with ASDs, the physical examination may not identify palpable stool in the left lower quadrant, and a careful rectal examination might not be feasible. Every attempt should be made to examine the rectum, although at times this cannot be accomplished. The rectal examination enables assessment of stool retention, anal tone, and occult mass, as well as the presence or absence of blood and helps to reassure the family that the child's anatomy is normal. It

need not be repeated on subsequent visits unless there is a change in the history or physical examination. A plain radiograph of the abdomen may reveal a rectal fecal mass not palpable on the abdominal examination. If large amounts of stool are found on rectal examination, an abdominal radiograph is not needed to establish the presence of fecal impaction (Constipation Guidelines Committee of the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition 2006). Diagnostic clues can help to identify some organic causes of constipation. Hirschsprung disease is not less common in children with ASDs, and a history of delayed passage of stool after birth should raise suspicion of aganglioneurosis. Anatomic abnormalities such as an anteriorly displaced anus, which is more common in girls than boys, can be diagnosed by careful inspection of the rectum. Children with altered intestinal motility may have underlying mitochondrial disease (Chitkara et al. 2003). Recent studies have suggested a potential association of ASDs and mitochondrial dysfunction (Oliveira et al. 2005).

Treatment of constipation includes mineral oil, magnesium hydroxide, lactulose, sorbitol, and polyethylene glycol (PEG), or a combination of lubricant (mineral oil) and laxative is recommended for the daily management of constipation in children.

Chronic Diarrhea

Chronic diarrhea occurs when loose stools persist for 2 weeks or longer, with or without an increase in stool frequency. Most episodes of acute diarrhea resolve within a week and are frequently caused by self-limited infections. In contrast, the causes of chronic diarrhea more frequently include noninfectious causes than for acute diarrhea. In the US general pediatric population, the most common causes of chronic diarrhea are functional disorders, malabsorption syndromes, inflammatory bowel disease (Crohn disease or ulcerative colitis), and chronic infections. The causes of diarrhea in children with ASD are likely the same as in children without ASDs, and the differential diagnosis should be approached with similar rigor. Chronic nonspecific diarrhea of

childhood can first present between 6 and 36 months of age and often resolves by 60 months of age. It is characterized by loose and sometimes frequent stools and, importantly, the absence of other abnormalities such as growth failure, abdominal pain, and difficulty in passing stool. If the latter signs or symptoms are present, other causes of chronic diarrhea should be considered. Guidelines for the diagnostic evaluation of chronic diarrhea have not yet been developed, but recommended approaches are available in standard pediatric gastroenterology texts (Guarino and De Marco 2008). These same approaches are relevant for the child with an ASD. A careful history and physical examination are important and include definition of the age at symptom onset and whether symptoms develop abruptly or gradually. Causes of chronic diarrhea in children include enteric infections, immunodeficiency, abnormal immune response, inflammatory bowel disease, congenital persistent diarrhea, hereditary lactase deficiency, chronic nonspecific diarrhea of childhood, and syndromic persistent diarrhea and malnutrition (in developing countries and worldwide). Family history of allergy or atopic disease may increase the likelihood of cow's milk allergy. It is important to assess a child's growth and nutrition. Poor weight gain may be a result of malabsorption or inadequate or inappropriate feeding; delayed growth may then be the result of a child being fed a diluted, hypocaloric formula or clear liquids in an effort to reduce diarrhea. In contrast, for infants or children who appear to be thriving or overweight while suffering from chronic diarrhea, a careful dietary record for 1 week may determine if a patient is being overfed or drinking excessive amounts of fruit juices that are known to induce diarrhea. A functional cause of chronic diarrhea is suggested by protracted symptoms (12 months) or lack of significant weight loss, nocturnal diarrhea, and straining with defecation. Loose stool in children with ASDs may be misdiagnosed as diarrhea. Constipation is a common, albeit somewhat paradoxical, cause of loose stool and may be difficult to confirm by history or physical examination. Stool might not be palpable in the left lower quadrant, and a rectal examination may be challenging and traumatic for the child. A plain abdominal radiograph may be useful. Frequently, an empiric trial with a

stool softener, such as PEG 3350 (MiraLAX) for 2–4 weeks, supports the diagnosis by causing a change in behavior. Lactose intolerance can be difficult to diagnose. A diagnostic trial of a strict lactose-free diet may be difficult to maintain, especially for a patient for whom food choices are limited. A lactose breath test (LBT), which requires an overnight fast and repeated collection of breath samples over 3 h, can be difficult to perform on some children. Similar information can be obtained at the time of an upper endoscopy via tissue analysis for disaccharidase-specific activity.

Food allergy, another but less frequent cause of diarrhea, is often challenging to diagnose because most instances of intestinal food allergy are cell mediated rather than mediated by immunoglobulin E (IgE). IgE-based tests may not identify individuals who do not have atopic or immediate reactions, and IgG-based tests are of no value in assessing intestinal food allergy. Referral to an allergist for skin testing may be appropriate. Assessment for celiac disease should be performed for any child with an ASD and gastrointestinal symptoms. Testing at a minimum should include a total IgA level, tissue transglutaminase IgA antibodies with or without endomysial IgA antibodies. Antigliadin antibodies are less reliable. Stool samples to test for enteric pathogens, ova/parasites, or occult blood can be obtained easily at the time of a lower endoscopy but otherwise may be difficult to collect. Biopsies of the colon and ileum are routinely obtained on endoscopy and determine whether there is acute or chronic mucosal inflammation. Therapeutic interventions vary depending on the cause of chronic diarrhea.

Gastroesophageal Reflux Disease

GER, the term for passage of gastric contents into the esophagus, can produce diverse symptoms and complications, called gastroesophageal reflux disease (GERD). During infancy, in contrast to nonpathologic reflux, GERD presents most frequently as an adverse effect of recurrent vomiting but also as apparent life-threatening events (ALTE). As a child matures, other symptoms and signs of GERD include chest pain or heartburn, esophagitis, food refusal, and extra-esophageal manifestations of the airway. As in

children without ASDs, the expression of disease related to GER can vary widely in children with ASDs. Certain children may express discomfort through problem behaviors, restriction of foods of specific texture, or simply pointing to their chest. Objective measures become important to elicit from the patient and family, such as the frequency and timing of regurgitation, the character and amount of material regurgitated, and the kinds of foods that are tolerated by the child. The presenting symptom or sign is a clue to the underlying cause. Children with ASDs who have obstruction caused by, for example, malrotation or antral web may regurgitate many times an hour, whereas those individuals who have typical GERD may experience symptoms when they lie down at night or after meals. The vomiting of undigested foods suggests a delay in gastric emptying; hematemesis suggests the presence of inflammation or ulceration. In young children, a preference for liquids or a refusal to eat textured foods or foods that require chewing should raise suspicion of esophagitis.

Diagnostic evaluation begins with a careful history and physical examination. In some children more than 2 years of age, recurrent regurgitation or vomiting disrupts their participation in childhood activities. As for children without ASDs, an empiric trial of acid suppression may be of diagnostic value, but then the clinician may want to order an upper gastrointestinal series to exclude an anatomic abnormality, as well as upper endoscopy with biopsy to look for inflammation associated with GERD, allergy, or eosinophilic esophagitis. Other warning signals for additional diagnostic testing include gastrointestinal bleeding, abdominal tenderness, and fever. A diagnostic trial of acid suppression, with an appropriate dose of a proton pump inhibitor (PPI), should be considered before invasive studies. Diagnosis of GERD may be delayed in individuals with ASD who do not localize pain or provide the classic history. PPI medications should be given 30 min before the first meal of the day, and if two doses are prescribed, 30 min before the evening meal. Assessment of response to a PPI trial is somewhat subjective in children with ASDs and might depend on changes in

behavior as perceived by care providers (parents, teachers, or health care providers). If further diagnostic testing is pursued, upper gastrointestinal radiographs can be challenging for many children who are unable to cooperate with drinking barium and lying down quietly for the procedure. Because of these potential barriers, it can be difficult to identify anatomic abnormalities, such as a malrotation, annular pancreas, or antral inflammation or narrowing, that may be the cause of GERD-like symptoms. Most children with ASDs are unlikely to tolerate placement of a transnasal pH probe for a prolonged period. A Bravo pH monitoring system (Given Imaging Ltd., Yoqneam, Israel), which is endoscopically attached to the distal esophagus, may be better tolerated. For such children, upper endoscopy under general anesthesia is often the diagnostic test initially used and might provide information about other gastrointestinal conditions such as carbohydrate malabsorption or food allergy or intolerance. Endoscopy is usually reserved for children who are unresponsive to a diagnostic trial of gastric acid suppression or when other clinical factors, such as hematemesis or food refusal, support the scheduling of invasive tests.

Treatment of GERD depends on the cause. Surgical therapy should be considered with a diagnosis of anatomic abnormality such as malrotation, whereas esophagitis may be treated best by prolonged acid suppression. Subsequent evaluation depends on the resolution and recurrence of an individual's symptoms. Upper endoscopy should be considered to follow children with long-standing GERD who may be at risk for developing complications such as esophageal stricture, Barrett esophagus, and esophageal cancer. As in children without ASDs, GERD in children with ASDs can be a chronic problem, with waxing and waning of symptoms. The management of such children often requires continuity with advancing age and an appreciation by the health care team of the natural history of disorders that underlie GERD (Vandenplas et al. 2009).

Sleep and Gastrointestinal Disturbance

Sleep disorders are among the many comorbid conditions present in ASDs (Lai et al. 2014). An

important point to consider is that sleep problems may be exacerbated by or even be the result of, other comorbid conditions. Other comorbid disorders in children with ASD are quite prevalent. In a population of over 160 children with ASD, Liu et al. observed attention deficit hyperactivity disorder (31%), epilepsy (11%), asthma (17%), allergies (50%), and gastrointestinal (GI) symptoms (21% with vomiting, reflux, spit-ups, 17% with chronic diarrhea, 18% with chronic constipation, 18% with abdominal pain, and 14% with intestinal bloating) (Liu et al. 2006). Although the mechanism of these sleep disturbances is difficult to ascertain, nighttime awakening with pain or abdominal discomfort is common with gastroesophageal reflux and reflux esophagitis in children. Autistic children with GI symptoms have been found to have a higher prevalence of sleep disturbances, compared with those in the general population who do not have GI symptoms and with their siblings. Furthermore, GI symptoms have been significantly correlated with insomnia and parasomnias. Taking into account that GI problems may be responsible for a great deal of sleep problems, one should consider that treatment focused on these conditions may bring relief or at least improvement in sleep problems symptoms. Lifestyle modifications, including smaller but more frequent meals or elevated head position during sleep should be considered as possible first-line treatments. Other approaches that should be taken into account whenever a child with ASD presents with sleep problems include, but are not limited to, proton pump inhibitors for gastroesophageal reflux or diet elimination/substitution therapies in food allergy such as cow milk protein allergy, lactose intolerance, or celiac disease. Naturally, of paramount importance is adhering to medical indications for introducing these treatments by way of proper diagnostic tests (Klukowski et al. 2015).

Future Directions

Medical disorders, including gastrointestinal problems, occur commonly in individuals with ASDs. Because the symptoms may be atypical,

these medical conditions may be undiagnosed. Whether the prevalence is higher than in the general population is not known with certainty. Prospective, well-controlled studies are necessary. Many parents and care providers have observed and reported improvements in problem behaviors with nutritional or medical interventions to address gastrointestinal symptoms and disorders. Some of these therapies are based on purely observational reports. The care of individuals who are nonverbal or have difficulties in communication or who display self-injurious or other problem behaviors presents special challenges. Nevertheless, the approach to evaluation and diagnosis of possible underlying medical conditions, in particular gastrointestinal disorders, should be no different from the standard of care for persons without ASDs. Management of gastrointestinal problems in individuals with ASDs usually begins with the primary care provider and may warrant consultation with a gastroenterologist and other specialists. Anecdotal reports that restricted diets may ameliorate symptoms of ASDs in some children have not been supported or refuted in the scientific literature, but these data do not address the possibility that there exists a subgroup of individuals who may respond to such diets. Professional supervision of restricted diets is recommended to prevent nutritional inadequacies (Buie et al. 2010a). Such reports help propagate interest in the use of dietary manipulation (e.g., gluten and casein-free diets) as an ASD-focused treatment. Dietary interventions, including the gluten- and casein-free diets, nutritional supplements, enzymes, and antimicrobial agents, have not been substantiated by empirical investigation, presenting a clear need to investigate how possible deviations in the GI tract in ASD relate to current dietary recommendations being promoted and implemented in the ASD community (McELhanon et al., 2014).

One should also consider possible deviations in the establishment and maintenance of the gut microbiome in ASD and the relative contribution of early feeding practices to overall gut health and acceptance of new feeding tastes and textures. Studies of fecal DNA extracts have found certain bacterial clusters overrepresented in children with

ASD and gastrointestinal complaints compared with children with similar GI complaints but typical neurobehavioral development (Mulle et al. 2013). Provisional evidence also suggests that children with ASD are at greater risk for suboptimal breastfeeding, including late initiation and shorter duration of exclusive breastfeeding (Al-Farsi et al. 2012). In addition to promoting optimal nutrition, breast milk assists in the development of the gastrointestinal tract, pancreas and endocrine system, and related mucosal defenses, and it is therefore possible that suboptimal breastfeeding may result in atypical colonization of the gut microbiome in ASD. In turn, changes in the gut microbiome may help explain anecdotal reports of improvement in behavioral functioning in response to dietary changes if such changes serve a probiotic function and improve symptoms of irritable bowel syndrome (e.g., bloating, abdominal pain, flatulence) among certain children with ASD (McElhanon et al., 2014).

Microbiota is an emerging topic that has attracted several researchers to look for the possible connection between the GI microflora and behavioral abnormalities. Earlier report of deficient disaccharidase enzymatic activity in ASD children and GI symptom prompted investigations looking for intestinal mucosal microbiota involved in carbohydrate metabolism (Horvath et al. 1999). Abnormal carbohydrate digestion and transport and mucosal dysbiosis (imbalance in the intestinal microbial ecosystem) was reported in the ASD children (Williams et al. 2011). Reduced level of fermenters has been found in the intestinal microflora of the ASD patients. The microbiota-gut-brain axis refers to the ability of gut microbiota to communicate with brain and regulate behavior. Fecal microbiota transplantation has been used in treating several GI disorders but increase knowledge and control trials are needed before it can be used broadly in clinic. Imbalance in gut microbiota population may render the intestinal mucosa susceptible to injuries, infections, inflammation, abnormal digestion, immune imbalance, immune reaction and cross reaction in other tissues including the brain. More research is emerging in this area (Samsam et al. 2014).

Future research is expected to clarify the role of metabolic disorders, allergic/toxic reactions, immune dysregulation, and inflammatory changes in the etiology of gastrointestinal disturbances in individuals with ASDs. Whether unique genetic, metabolic, or physiologic conditions exist and are specific to ASDs remains to be determined. New knowledge in these areas will advance our approach to the management of ASDs. Recognition that problem behaviors might indicate an underlying medical condition will facilitate diagnosis and treatment and ultimately improve the quality of life for many persons with ASDs. Well-designed trials are needed to develop an evidence base for optimal diagnostic and treatment strategies to manage gastrointestinal disorders in children with ASDs. Until then, application and, where necessary, adaptation of conventional recommendations for the general pediatric population are relevant to children with ASDs (Buie et al. 2010b).

See Also

- ▶ Allergies
- ▶ Constipation
- ▶ Gluten-Free Diet
- ▶ Intestinal Permeability Studies
- ▶ Leaky Gut Syndrome
- ▶ Medical Conditions Associated with Autism
- ▶ Metabolic Testing

References and Reading

- Buie, T., Campbell, D., Fuchs, G. J., III, Furuta, G. T., Levy, J., VandeWater, J., et al. (2010a). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125, S1–S18.
- McElhanon, B. O., McCracken, C., Karpen, S., & Sharp, W. G. (2014a). Gastrointestinal symptoms in autism spectrum disorder: A meta-analysis. *Pediatrics*, 133(5), 872–883.
- Buie, T., Fuchs, G. J., 3rd, Furuta, G. T., Kooros, K., Levy, J., Lewis, J. D., et al. (2010b). Recommendations for evaluation and treatment of common gastrointestinal problems in children with ASDs. *Pediatrics*, 125 (Suppl. 1), S19–S29.
- Chitkara, D. K., Nurko, S., Shoffner, J. M., Buie, T., & Flores, A. (2003). Abnormalities in gastrointestinal

- motility are associated with diseases of oxidative phosphorylation in children. *American Journal of Gastroenterology*, 98(4), 871–877.
- Constipation Guidelines Committee of the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition. (2006). Evaluation and treatment of constipation in infants and children: Recommendations of the North American society for pediatric gastroenterology, hepatology and nutrition. *Journal of Pediatric Gastroenterology Nutrition*, 43(3), e1–e13.
- Guarino, A., & De Marco, G. (2008). Persistent diarrhea. In R. E. Kleinman, I. R. Sanderson, O. Goulet, P. M. Sherman, G. Mieli-Vergani, & B. L. Shneider (Eds.), *Walker's pediatric gastrointestinal disease: Pathology, diagnosis, management* (5th ed., pp. 265–274). Hamilton: BC Decker.
- Horvath, K., & Perman, J. A. (2002). Autism and gastrointestinal symptoms. *Current Gastroenterology Reports*, 4(3), 251–258.
- Johnson, C. P., Myers, S. M., & American Academy of Pediatrics, Council on Children with Disabilities. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120(5), 1183–1215.
- Oliveira, G., Diogo, L., Grazina, M., Garcia, P., Ataide, A., Marques, C., et al. (2005). Mitochondrial dysfunction in autism spectrum disorders: A population-based study. *Developmental Medicine and Child Neurology*, 47(3), 185–189.
- Vandenplas, Y., Rudolph, C. D., Di Lorenzo, C., Hassall, E., Liptak, G., Mazur, L., et al. (2009). Pediatric gastroesophageal reflux clinical practice guidelines: Joint recommendations of the North American Society of Pediatric Gastroenterology, Hepatology, and Nutrition and the European Society of Pediatric Gastroenterology, Hepatology, and Nutrition. *Journal of Pediatric Gastroenterology Nutrition*, 49(4), 498–547.
- Volkmar, F., Cook, E. H., Jr., Pomeroy, J., Realmuto, G., & Tanguay, P. (1999). Practice parameters for the assessment and treatment of children, adolescents, and adults with autism and other pervasive developmental disorders. *Journal of the American Academy of Child & Adolescent Psychiatry*, 38(Suppl. 12), 32S–54S. American Academy of Child and Adolescent Psychiatry Working Group on Quality Issues (published correction appears in *J Am Acad Child Adolesc Psychiatry*. 2000; 39(7):938.
- Lai, M. C., Lombardo, M. V., & Baron-Cohen, S. (2014). Autism. *Lancet*, 383, 896–910.
- Liu, X., Hubbard, J. A., Fabes, R. A., et al. (2006). *Child Psychiatry and Human Development*, 37, 179.
- Klukowski, M., Wasilewska, J., & Lebensztejn, D. (2015). *Dev Period Med.*, 19(2), 157–161.
- McElhanon, B. O., McCracken, C., Karpen, S., et al. (2014b). Gastrointestinal symptoms in autism spectrum disorder: A meta-analysis. *Pediatrics*, 133, 872–883.
- Mulle, J. G., Sharp, W. G., & Cubells, J. F. (2013). The gut microbiome: A new frontier in autism research. *Current Psychiatry Reports*, 15(2), 337.
- Al-Farsi, Y. M., Al-Sharbaty, M. M., Waly, M. I., et al. (2012). Effect of suboptimal breast-feeding on occurrence of autism: A case-control study. *Nutrition*, 28(7–8), e27–e32.
- Horvath, K., Papadimitriou, J. C., Rabsztyrn, A., Drachenberg, C., & Tildon, J. T. (1999). Gastrointestinal abnormalities in children with autistic disorder. *The Journal of Pediatrics*, 135, 559–563.
- Williams, B. L., Hornig, M., Buie, T., Bauman, M. L., Cho Paik, M., Wick, I., Bennett, A., Jabado, O., Hirschberg, D. L., & Lipkin, W. I. (2011). Impaired carbohydrate digestion and transport and mucosal dysbiosis in the intestines of children with autism and gastrointestinal disturbances. *PLoS One*, 6, e24585.
- Samsam, M., Ahangari, R., & Naser, S. A. (2014). Pathophysiology of autism spectrum disorders: Revisiting gastrointestinal involvement and immune imbalance. *World Journal of Gastroenterology : WJG*, 20(29), 9942–9951.
- RETRACTED: Wakefield AJ, Murch SH, Anthony A, et al. (1998). Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *Lancet*; 351(9103):637–641.
- Lau, N. M., Green, P. H., Taylor, A. K., et al. (2013). Markers of celiac disease and gluten sensitivity in children with autism. *PLoS One*, 8(6), e66155.
- Campbell, D. B., Li, C., Sutcliffe, J. S., Persico, A. M., & Levitt, P. (2008). Genetic evidence implicating multiple genes in the MET receptor tyrosine kinase pathway in autism spectrum disorder. *Autism Research*, 1(3), 159–168.
- Croen, L. A., Najjar, D. V., Ray, G. T., Lotspeich, L., & Bernal, P. (2006). A comparison of health care utilization and costs of children with and without autism spectrum disorders in a large group-model health plan. *Pediatrics*, 118(4), e1203–e1211.

GAUTENA Autism Society

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Background and Context

GAUTENA is the acronym in Basque language of Gipuzkoa Autism Society www.gautena.org. This society of families of people with autism spectrum disorder (from now on referred to as ASD) was founded in 1978, in Gipuzkoa, one of the three provinces that constitute the Basque Country (Spain). In compliance with its Statute of Autonomy from 1979, the Basque Country has full

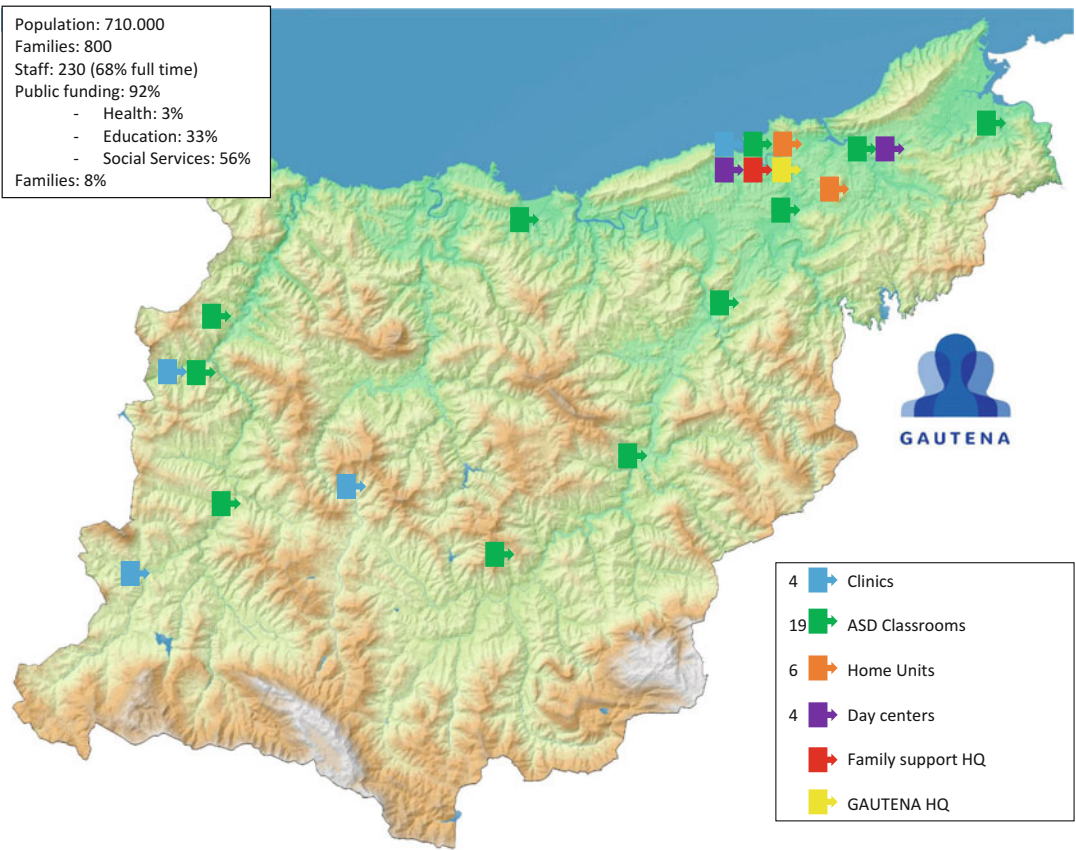
capacity and responsibility in areas such as Health, Education, and Social Services. This circumstance, together with the financial autonomy that the mentioned political regime confers to the Basque region, helps to understand GAUTENA’s development in favor of people with ASD in Gipuzkoa, a region of 1909 km² with a population of about 710,000 inhabitants.

The Basque Country, traditionally an industrial region and most recently focusing on the services sector, enjoyed in 2016 a per capita income of 31,805€ (10% higher than the average of the EU 28). The social protection expenditure per person, measured in purchasing power parity (PPP), was 8.341€, while the average of the UE 28 amounted to 7.903€. In terms of the UN Human Development Index that takes into account life expectancy, education, and wealth, the Basque Country is placed in the eight position in the world (0.91)

just next to the USA (0.92), although it is not fair to compare specific regions with full countries.

Landmark Contributions

Based on the previous work of other associations of families involved with disabilities and keeping a good relationship with them, GAUTENA began to work in 1978 with the purpose of achieving a community-based network of services for people with ASD within a society that pursues to be more inclusive every day. It currently provides support to 800 families and has a staff of about 230 members. Although the families pay some fees for the provided services, financing is mainly public (92%). In the last years, several laws at state and community level have reinforced the role of social services in the society.



Through different formal agreements with the Public Administration in the areas of Education, Health, and Social Services, GAUTENA encourages and manages a network of services that offer comprehensive support throughout the whole life cycle to people with ASD, such as diagnosis and treatment services, education, day services, sheltered housing, temporary stays, leisure, and support to families.

The key characteristics at the time of identifying GAUTENA's reality are the application of a suitable community-based model, the significant level of public support, the social sensitivity in our territory encouraging this kind of initiatives, and a nonprofit management. The organization is based on the pillars of quality-oriented practice, an ethical framework with empowering for human rights and personalization, and research and social innovation, in order to adjust the model to the changing needs of people with ASD. Based on these pillars, the main goal is the continuous fostering of a good quality of life for the users and their families and a more comprehensive and inclusive community for all.

Major Activities

Services Network

Through formal agreements with the Public Administration in areas of Education, Health, and Social Services, GAUTENA manages a service network that includes diagnosis and treatment, education, day services, housing, temporary stays, leisure and free-time activities, as well as support to families. More detailed information is given below.

Diagnosis and Treatment

The clinical team, which includes professionals of psychiatry and psychology, is in charge of the diagnosis and treatment of the people with ASD referred to GAUTENA from the Public Administration, mostly from the National Health System. In collaboration with the families, the GAUTENA team gives support through individual or group treatment sessions to more than 500 children and also offers specialized support for their schooling.

Education

The Public Administration policy in the Basque educational system strongly encourages and supports inclusion of students with special educational needs. For this reason, a good number of people with ASD – over 400 – supported by GAUTENA usually attend regular schools where they receive different levels of individualized support. Some of these students attend lessons in classrooms managed directly by GAUTENA, 19 classes at present, located in different districts of Gipuzkoa. In these classrooms, called “stable classrooms,” an average number of five students receive the support of two teachers. In such classrooms, the personalized mainstreaming of the students with ASD into the school is encouraged. At present, there are 98 students who attend these GAUTENA classrooms, located in regular schools (a special school we had was voluntarily closed years ago). The educational period extends from the age of 3 to the age of 20 with the possibility of an extraordinary extension up to 21.

Day Service

At the end of their school period, after they have reached the age of 20, people with ASD can access a well-developed sheltered employment organization, where GAUTENA sits on its Board, or try a supported job in the ordinary market. Up to the present time, it has been found that most of the people with ASD with from medium to severe affectation reaching adulthood turn toward the Day Service Centers managed by GAUTENA for day services. GAUTENA's four Day Centers offer a suitable combination of work on personal and vocational skills, together with a wide use of community-based resources that make possible to enjoy a good day program for about 112 adults with ASD in Gipuzkoa.

Housing Service

GAUTENA offers different types of housing to those who request this service, ranging from flats in blocks (Sheltered Flats) to small dwelling units (Group Homes) for people who need more structured support. This housing network includes one house for temporary stays extending the coverage

through different ways – weekends, short stays, critical periods, medium-period stays, etc. – to a significant group of families, children, teenagers, and adults. At the present time, 14 housing units provide permanent support to 78 people, and another house is used for temporary stays for around other 50 families.

Family Support Service

This service focuses directly on the families of people with ASD and offers them a wide range of support services, from family welcoming programs to the organization of support groups, training conferences and seminars, and leisure activities for families with their children with ASD. Also navigating the necessary processes to obtain economic subsidies, among other administrative benefits, is supported. This service involves and trains volunteers, usually university students, who may become staff persons later on. The Family Support Service organizes the leisure and free-time activities carried out by GAUTENA throughout the year and especially during the holiday periods, an initiative that involves more than 500 users.

Approach to Quality

GAUTENA is an organization inspired in community-based models, having paid since it's beginning a great deal of attention to the quality of services provided to the person with ASD. Based on technical criteria inspired in the most reputable international programs, since the middle of the 1990s, it has been working in compliance with the Quality Assurance criteria of ISO 9001 Standard, having received the pertinent accreditation from the Spanish Organization for Standardization (AENOR). In 2000, GAUTENA received a pilot accreditation from the National Autistic Society (UK) www.nas.org.uk. In 2008, GAUTENA received the Silver Q to Management from EUSKALIT (Basque Foundation for Excellence www.euskalit.net) for having obtained more than 400 points in the application of the EFQM model (European Foundation for Quality Management www.efqm.eu). Besides, GAUTENA enhances its work in favor of quality with other approaches such as that of “The Council”

www.thecouncil.org (USA) or that of Plena Inclusión/FEAPS www.feaps.org (Spanish Confederation in favor of People with Intellectual and Developmental Disabilities). In 2016 GAUTENA was granted by the European Parliament one of the European Citizen Awards for “having transformed the European values into support programs for the people with ASD.”

Looking at the Future

The Quality approach taken by GAUTENA starts from the response to the rights of people with ASD by guaranteeing a coherent ethical approach in a personalized plan, with the purpose of fostering quality of life for people with ASD and their families.

For that purpose, GAUTENA is developing instruments for the practical promotion of the rights of the people to whom support is given and especially for those that, because of their significant needs, almost whole responsibility is taken up by the staff. At the same time, an Ethical Adequacy Plan prepared in collaboration with the University of Deusto www.deusto.es (Bilbao and San Sebastian) is being applied, in order to embed ethics into the philosophy and practice of professionals and families in GAUTENA.

While developing rights, ethics, and quality of life, GAUTENA is giving as much support as possible to initiatives in research of ASD and has collaborated with important international entities. An agreement with the Research Foundation Policlínica Gipuzkoa Fundazioa, in San Sebastián, has been established, and it is taking part in research projects on genetics, phenotyping, immunology, epidemiology, and the use of augmentative communication devices www.policlinicagipuzkoa.com. In the frame of this agreement, GAUTENA is currently part of the ASDEU project, sponsored by the European Commission, involving over 10,500 children in its county.

In addition, social innovation, understood as the advance of innovative responses to the needs of people with disabilities, guides GAUTENA's steps and makes it to take part in general activities such as the one of the Basque Innovation Agency (Innobasque) www.innobasque.com. GAUTENA applies a policy of encouragement of

benchmarking and alliances, both in its most immediate area in Gipuzkoa and Basque Country, as well as in Spain and Europe, and it is an active member of several organizations, being a founding society of Autism Europe www.autismeurope.org.

See Also

- [Community Services](#)
- [Ethics](#)

References and Reading

- Fuentes, J., Barinaga, R., & Gallano, I. (2000). Applying TEACCH in developing autism services in Spain: The GAUTENA project. *International Journal of Mental Health*, 29(2), 78–88.

Gaze

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Definition

Following the eye gaze of other people is a crucial component of face-to-face social interactions from infancy to adulthood. The ability to follow where a person looks is usually called “gaze following” or “responding to joint attention.” This and other gaze behaviors are important because they are powerful learning mechanisms. Specifically, research has shown that gaze following and other gaze behaviors predict language development for children with and without autism (Brooks and Meltzoff 2008; Mundy et al. 2007; Toth et al. 2006; Wetherby et al. 2007). In addition, the patterns of use and perception of gaze may help identify which at-risk children develop autism spectrum disorders (Jones et al. 2014).

There are several eye gaze behaviors that are widely studied, including mutual gazing, cued gaze shifting, gaze alternating, and gaze following. Each of these gaze behaviors has potential functions in early development. Special attention is paid to relevant findings for young children with autism spectrum disorder (ASD), as well as for infants at risk for developing ASD because their sibling has ASD. This entry uses gaze following to illustrate developmental changes in infants’ understanding of eye gaze.

Gaze following is an interesting developmental phenomenon because it provides a window into infants’ social cognition. Adults typically understand a person’s gaze in a way that puts them in touch with the other person’s mind, informing them of the other’s desires, emotions, intentions, and perceptions. When adults notice where someone is looking, they do not simply track the movement of the head and eyes; rather, adults understand that people use their eyes to see things of interest in their surroundings. In that sense, developmental scientists consider gaze following an important front-end ability that feeds into the development of understanding other minds (see entry on “► [Theory of Mind](#)”). For example, a mother could look with a disgusted facial expression at an object while saying, “I don’t like that.” For adults viewing this act, they can follow her gaze to find out what has caught her attention and what she is talking about, as well as what disgusted her. Individuals who do not follow gaze would miss this important social and referential information. This is of particular concern for children with ASD because they have developmental deficits in following the gaze of others, and this may contribute to other downstream problems in social understanding (Mundy et al. 2009; Jones et al. 2014; Toth et al. 2006).

Current Knowledge

As soon as infants are born, they are introduced to social interactions. At first, parents and infants share face-to-face eye contact in the *dyadic* context. Later, the social interactions become *triadic* when the dyad includes a third entity,

such as when an infant shifts her gaze between a parent and an object. The questions for developmental psychologists are when do infants begin to follow the gaze of other people and how do infants interpret others' eye gaze.

Gaze behaviors could indicate that both adult and child are aware that they are sharing attention. Indeed, triadic social interactions are often referred to as joint attention because the adult and the infant attend to the same topic. However, it is unclear whether young infants and children with ASD are aware that they are sharing attention and intentions. This has led to an ongoing debate in the field about how to interpret early gaze behaviors, especially gaze following (Flom et al. 2007; Mundy et al. 2009). On one side of the debate, infants gaze follow because they already understand that people can make psychological contact with external objects, that people see something interesting. On the other side of the debate, gaze following occurs for lower-order physical reasons, simply because of the signal value of the other's head movement. According to this latter perspective, infants do not understand anything about the adult as a perceiver of objects, but simply process the movement shown by the head. This ongoing debate has fueled extensive research on the emergence and meaning of gaze following.

Another important issue is the developmental outcome for children who have little or no interest in attending to the gaze of other people. This is a common concern of parents of children with ASD. A significant question is whether children with ASD notice the gaze of others and whether they interpret it as having psychological meaning. In order to answer this question, it is important to make conceptual distinctions among different types of closely related behaviors that are sometimes confused in the literature about gaze processing. As described below, there are crucial logical, developmental, and clinical distinctions among them.

Interest in Eyes and Mutual Gaze. Mutual gaze – staring directly into the eyes of another person – is one of the earliest experiences people have. It is a context for sharing emotions and communicating. Social partners recognize that

they are looking at each other. The developmental question is whether infants recognize that others are looking at them. It is known that young infants have preferences for face-like patterns containing eyes or eyespots (Johnson et al. 2008). For example, newborns look longer at faces with eyes facing toward them than eyes averted to the side. By the age of 2 months, infants begin scanning the eye region more than the edges of the face. These early patterns could stem from perceptual biases and preferences, but they are not clear evidence for noticing the eyes or recognizing eyes as having social and communicative intent.

Detecting cues of mutual gaze may be difficult for individuals with ASD. As an interesting test, Elsabbagh and colleagues (2012) presented gaze cues that contrasted eyes shifting to the side versus looking straight ahead to simulate looking away versus making eye contact. When infants (6–10 months old) viewed such cues, the infants with a subsequent ASD diagnosis were less sensitive on neural measures to the gaze shifts than infants without a later ASD diagnosis. In addition to difficulties with detecting mutual gaze, children with ASD often scan faces and eyes differently than children with typical development (Nation and Penny 2008). Decreased scanning of eyes may emerge as early as 6 months of age (Jones and Klin 2013). Across ages, children and adults with ASD are often interested in the mouth, yet they do not completely ignore the eyes (Webb 2008). An overall concern would be that reduced attention to eyes could lead to less experience with the socially meaningful information that eyes convey.

Peripheral Cue with Gaze Shifts. Another line of eye-gaze research investigates whether gaze shifts influence the attention of observers. To address this issue, gaze shifts are used to cue the location of peripheral targets, such as a rectangle or an asterisk (Johnson et al. 2008; Nation and Penny 2008). The classic stimulus with this task is a digitized face with eyes that shift to one side before a target appears on the right or left. The face usually vanishes before the peripheral targets appear in this paradigm. Adults and infants typically look with shorter latencies to the target that has been cued. If the cue shifts to

the right side, they attend to a target that appears on the right faster than on the left.

These are fascinating effects, but they do not provide evidence about following gaze in biologically plausible settings. In the real world, when a mother looks at and labels an object (“There’s a ball!”), her face does not vanish to free up attentional resources for the child. Moreover, the peripheral targets do not pop into existence to attract attention as they do in the cueing paradigms. Rather, in everyday settings, the world stays stable and the parental gaze spotlights a preexisting object. Nonetheless, infants’ responses to directional shifts in cueing studies may be a precursor to actual gaze following.

For individuals with ASD, the key question is whether they respond to gaze cues. In many but not all studies, the direction of the gaze shifts cued the reactions of children and adults with ASD (Nation and Penny 2008). For example, at 2 years old, toddlers with ASD responded faster to locations cued by gaze shifts than non-cued locations, which was similar to typically developing toddlers (Chawarska et al. 2003). Of interest with this study, the 2-year-olds with ASD on average “passed” this test of cueing, but they failed to follow a person’s gaze in a real room. Thus, the cueing phenomenon is not necessarily correlated with what individuals do in ecologically valid settings, which suggests that calling this “gaze following” would be a misnomer.

Gaze Cues in Habituation Studies. A third body of work that addresses infants’ reactions to gaze cues is habituation studies. In the first phase of a prototypical habituation study, a person looks at one of two objects. The trials are repeated until there is a decline in infants’ looking at the presentation (i.e., habituation). In the second phase, infants’ looking times are measured to examine which of two types of presentations attracts renewed interest (i.e., dishabituation). For example, Woodward (2003) found that infants, who habituated to a person looking at a ball on the left, dishabituated more strongly to that person looking at a bear on the left (new object, same location) than to that person looking at the ball on the right (same object, new location). This suggests that infants encoded the relationship

between the looker and the object, and that they dishabituated when the link was broken. The findings also showed that there is a developmental change for encoding this looker-object link, as seen by 12-month-olds succeeding in the task and younger infants failing. A consideration is that these habituation-dishabituation studies chiefly document infants’ recognition abilities. Infants recognize (and encode) that the other person is looking, but they are not required to produce an action to follow that person’s gaze.

To date, there are no parallel studies of gaze with habituation for children with ASD. It would be interesting to examine whether children with ASD encode the looker-object link. Further, does a child’s ability to spontaneously follow a person’s looking behavior in a real situation influence the habituation test (or vice versa)?

Gaze Alternation and Joint Engagement. Joint engagement refers to when infants and caretakers attend to and play with the same toy. This also is called a triadic interaction because the child and the parent communicate about a third object, such as a toy. Adults are aware of shared or joint attention in the triad, but an interesting question is whether infants are aware of it. An argument has been that infants show their awareness of joint attention by initiating eye contact with the adult and shifting their gaze to the toy. This type of gaze alternation is called “coordinated joint engagement” (Adamson et al. 2009). From observations of parents and infants at play, in the majority of joint play, infants did not alternate their gaze between the parent and a shared toy. Nonetheless, there were some instances of gaze alternation by the infants. The general pattern is that coordinated joint engagement emerges at about 9 months when it is relatively infrequent and brief, but gradually becomes more frequent and lasts longer, by 15–18 months, for typically developing infants (Carpenter et al. 1998).

Because of the importance seen in these gaze behaviors, researchers have designed tasks to prompt and assess the capacity for alternating gaze (Mundy et al. 2007; Wetherby et al. 2007). In a typical version of the gaze-prompting tasks, an experimenter activates a mechanical toy that is not within reach of the child. In a longitudinal

study, Mundy et al. (2007) found that 9-month-old infants readily made eye contact with the experimenter and alternated gaze to toys in the context of gaze-prompting tasks. These gaze behaviors were key components of their measure of “initiation of joint attention” and did not significantly increase by 18 months of age. The findings with the gaze-prompting tasks suggest that infants are capable of alternating their own gaze in structured situations before they consistently do so during unstructured play. Of note, these tasks do not directly measure how well infants understand the gaze of others.

Studies show that children with ASD are less likely to alternate gaze in a triadic context than children with typical development. At 20 months, toddlers with ASD were less likely to shift their gaze from the adult to a toy (or vice versa) than comparison groups in gaze-prompting tasks (Wetherby et al. 2007). As seen in play observations, 30-month-old children with ASD had many opportunities for joint play with the support of their parents, yet they rarely had episodes of coordinated joint engagement (Adamson et al. 2009). Even at older ages, gaze alternation remains less frequent among children with ASD than their peers with typical development; this has been viewed as a fundamental problem for children with ASD (Mundy et al. 2009; Toth et al. 2006).

Following the Gaze of Others. A simple head turn takes on referential meaning when a person looks at something in the environment. Adults recognize this and turn to see what the other has seen. However, it is unclear whether young infants with typical development and children with ASD turn for that reason. In the typical gaze-following test, an adult looks at an object by turning and aligning his (or her) head and eyes with an object in the room, and in studies of “responding to joint attention,” the adult also points during the look (Mundy et al. 2007). Research has shown that infants turn in the direction of the adult’s head (and arm) movement, as early as 3–6 months of age under certain conditions, and that this following becomes more frequent between 12 and 18 months for typically developing infants (Flom et al. 2007; Jones et al. 2014; Mundy et al. 2007). The difficulty in

interpreting these findings is that as the adult turns toward a target, the adult’s large and salient head motion may draw infants in the correct direction without them processing the adult’s gaze at all. Infants may simply be following the head motion that they see in their visual field and find the target object by chance.

To test whether infants follow head motion or eye gaze, Brooks and Meltzoff (2002) designed a special test using a gaze-following paradigm. The key manipulation was that the adult turned toward an object with open eyes for one group and closed eyes for the other group. The test controlled for head motion because the adult made the same head motion for both groups – what differed was whether the adult’s eyes were open or shut. If infants relied on simple head motion, they would turn in the same direction as the adult in both cases. If, however, infants recognize that eyes are part of the referential act, they would turn to look at the target object in one situation but not the other. The results showed that 12- to 18-month-old infants were more likely to look at the adult’s target when she turned with open eyes than closed eyes. Infants did not simply follow head motion because they differentiated the head turns. Instead, infants followed the gaze of the adult to the target objects; infants from 12 to 18 months genuinely followed *gaze*.

The question remained as to when gaze following develops. To test for the emergence of gaze following, Brooks and Meltzoff (2005) used the same test of open versus closed eyes with 9-, 10-, and 11-month-olds. The findings suggested that genuine gaze following emerges at 10–11 months of age. At the two older ages, infants followed the adult’s head turns with open eyes more often than turns with closed eyes. In contrast, 9-month-olds looked at the target object in both cases, for eyes open *and* closed. The 9-month-olds “failed” to follow gaze – they followed the adult’s head turns even when her eyes were closed. These findings suggested that young infants were following head motion rather than eye gaze.

With their lack of interest in eyes, will children with ASD follow a person’s gaze? It is unknown whether children with ASD follow eyes versus head motion because this particular empirical

test has not yet been done. However, in other gaze-following protocols, infants at risk for ASD have deficits that are detectable after 12 months of age (Jones et al. 2014). By 20 months, toddlers with ASD were unlikely to follow the line of regard of an adult, even though the adult looked and pointed at a target; their scores were lower than those of toddlers with other types of developmental delay or with typical development (Wetherby et al. 2007). By about 4 years of age, children with ASD are more likely to follow the adult's line of regard than younger children with ASD, but many children with ASD are still less likely to follow an adult's actions than peers with typical development (Mundy et al. 2009; Nation and Penny 2008).

Future Directions

There are several provocative issues left to resolve for gaze behaviors. How do gaze behaviors relate to one another (e.g., detecting mutual gaze and gaze following)? Does improving one type of gaze behavior lead to global changes in other gaze behaviors? What are the developmental steps that help infants and children understand the referential nature of looking and seeing? Does the difficulty that children with ASD have with an elementary behavior such as gaze following contribute to and predict their later deficits in understanding others' minds (i.e., theory of mind)?

Researchers have shown that interventions can prompt children with ASD to follow others' line of regard, but they have not examined whether children with ASD are specifically following eye gaze (e.g., Kasari et al. 2006; Schreibman et al. 2015). Future research can continue to explore best practices to help children with ASD develop an interest in eyes and follow the eye gaze of others. One suggestion is that infants' own experiences may help them learn. Infants and children may use their own visual experiences as a framework for understanding that behavior in others. When infants open and close their own eyes, they immediately change whether they can (or cannot) see their surroundings. This

experience could help them recognize the same effect of eye closure for others. In an experimental study for infants with typical development, infants' own experiences with visual obstructions changed how they interpreted another person's line of sight (Meltzoff and Brooks 2008). The findings suggest that infants' visual experiences change what they understand about looking. Will interventions designed to vary the visual experiences of toddlers and children with ASD help them learn to follow the gaze of others? This and other interventions that actively involve the infant or child may greatly advance developmental progress for children with ASD (Schreibman et al. 2015). The overall goal to improve children's gaze following is important because gaze following predicts language development, which in turn can help children understand the minds of other people (Brooks and Meltzoff 2015; Mundy et al. 2009).

See Also

- Eye Gaze
- Joint Attention
- RJA/IJA (Initiating/Responding to Joint Attention)
- Theory of Mind

References and Reading

- Adamson, L. B., Bakeman, R., Deckner, D. F., & Ronski, M. A. (2009). Joint engagement and the emergence of language in children with autism and Down syndrome. *Journal of Autism and Developmental Disorders*, 39, 84–96.
- Brooks, R., & Meltzoff, A. N. (2002). The importance of eyes: How infants interpret adult looking behavior. *Developmental Psychology*, 38, 958–966.
- Brooks, R., & Meltzoff, A. N. (2005). The development of gaze following and its relation to language. *Developmental Science*, 8, 535–543.
- Brooks, R., & Meltzoff, A. N. (2008). Infant gaze following and pointing predict accelerated vocabulary growth through two years of age: A longitudinal, growth curve modeling study. *Journal of Child Language*, 35, 207–220.
- Brooks, R., & Meltzoff, A. N. (2015). Connecting the dots from infancy to childhood: A longitudinal study

- connecting gaze following, language, and explicit theory of mind. *Journal of Experimental Child Psychology*, 130, 67–78.
- Carpenter, M., Nagell, K., & Tomasello, M. (1998). Social cognition, joint attention, and communicative competence from 9 to 15 months of age. *Monographs of the Society for Research in Child Development*, 63(4, Serial No. 255), 1–174.
- Chawarska, K., Klin, A., & Volkmar, F. (2003). Automatic attention cueing through eye movement in 2-year-old children with autism. *Child Development*, 74, 1108–1122.
- Elsabbagh, M., Mercure, E., Hudry, K., Chandler, S., Pasco, G., Charman, T., . . . The BASIS Team. (2012). Infant neural sensitivity to dynamic eye gaze is associated with later emerging autism. *Current Biology*, 22, 338–342.
- Flom, R., Lee, K., & Muir, D. (Eds.). (2007). *Gaze-following: Its development and significance*. Mahwah: Lawrence Erlbaum.
- Johnson, M. H., Grossmann, T., & Farroni, T. (2008). The social cognitive neuroscience of infancy: Illuminating the early development of social brain functions. In R. V. Kail (Ed.), *Advances in child development and behavior* (Vol. 36, pp. 331–372). San Diego: Elsevier.
- Jones, W., & Klin, A. (2013). Attention to eyes is present but in decline in 2–6-month-old infants later diagnosed with autism. *Nature*, 504, 427–431.
- Jones, E. J. H., Gliga, T., Bedford, R., Charman, T., & Johnson, M. H. (2014). Developmental pathways to autism: A review of prospective studies of infants at risk. *Neuroscience and Biobehavioral Reviews*, 39, 1–33.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in young children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry*, 47, 611–620.
- Meltzoff, A. N., & Brooks, R. (2008). Self-experience as a mechanism for learning about others: A training study in social cognition. *Developmental Psychology*, 44, 1257–1265.
- Mundy, P., Block, J., Delgado, C., Pomares, Y., Van Hecke, A. V., & Parlade, M. V. (2007). Individual differences and the development of joint attention in infancy. *Child Development*, 78, 938–954.
- Mundy, P., Sullivan, L., & Mastergeorge, A. M. (2009). A parallel and distributed-processing model of joint attention, social cognition and autism. *Autism Research*, 2, 2–21.
- Nation, K., & Penny, S. (2008). Sensitivity to eye gaze in autism: Is it normal? Is it automatic? Is it social? *Development and Psychopathology*, 20, 79–97.
- Schreibman, L., Dawson, G., Stahmer, A. C., Landa, R., Rogers, S. J., McGee, G. G., . . . Halladay, A. (2015). Naturalistic developmental behavioral interventions: Empirically validated treatments for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45, 2411–2428.
- Toth, K., Munson, J., Meltzoff, A. N., & Dawson, G. (2006). Early predictors of communication development in young children with autism spectrum disorder: Joint attention, imitation, and toy play. *Journal of Autism and Developmental Disorders*, 36, 993–1005.
- Webb, S. J. (2008). Impairments in social memory in autism? Evidence from behaviour and neuroimaging. In J. Boucher & D. Bowler (Eds.), *Memory in autism* (pp. 188–209). New York: Cambridge University Press.
- Wetherby, A. M., Watt, N., Morgan, L., & Shumway, S. (2007). Social communication profiles of children with autism spectrum disorders late in the second year of life. *Journal of Autism and Developmental Disorders*, 37, 960–975.
- Woodward, A. L. (2003). Infants' developing understanding of the link between looker and object. *Developmental Science*, 6, 297–311.

GEC (Global Executive Composite)

► BRIEF (Behavior Rating Inventory of Executive Functions)

Gelotophobia and Autism

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Definition

Gelotophobia can be defined as the fear of being laughed at. It comes from the Greek word “gelos,” which means laughter, and the word “phobia” meaning fear (Ruch and Proyer 2008a; Titze 2009).

Historical Background

Gelotophobia was originally derived from clinical case studies and introduced as a clinical

condition by Titze (1996, 2009). However, gelotophobia is not a pathology but is experienced in the typically developing population also (Ruch et al. 2014). Gelotophobia is an interindividual differences variable. At its extreme end, it may lead to a pathological fear, but only in rare cases of gelotophobia (Ruch and Proyer 2008a, b). Given the implications and consequences associated with gelotophobia, such as social anxiety and social withdrawal (Titze 1996, 2009), this is an important area of study.

The phenomenon of gelotophobia has not only been investigated in the typically developing population. Previous research has found that 40% of individuals with eating disorders exceeded the threshold for a slight form of gelotophobia, 35.7% of individuals with personality disorders experienced gelotophobia (Forabosco et al. 2009), and 24.6% of shame-bound neurotics showed gelotophobic tendencies (Ruch and Proyer 2008b).

Gelotophobia is of importance to study in individuals with autism spectrum disorder (ASD). Due to the deficits in social communication and social cognition, individuals with ASD may experience difficulties with empathy. This is a result of their reduced theory of mind capacity which hinders their ability to distinguish between one's own mental state and that of others (Woods et al. 2013). Individuals with ASD may have greater difficulty understanding and compartmentalizing their own emotions and conveying these emotions to others (Woods et al. 2013). Individuals with ASD may have difficulty in understanding nonverbal cues, reciprocal interaction, and initiating appropriate nonverbal communication (Frith 2004; Wing 1981). Individuals with ASD may also have difficulty understanding humor. Weiss et al. (2012) compared whether children with ASD could appreciate slapstick comedy in comparison to children who were typically developing. While children with ASD could appreciate the material, they had greater difficulties in discriminating humorous from non-humorous material. Given these reasons, gelotophobia is of relevance to study in individuals with ASD.

Current Knowledge

Samson et al. (2011) conducted the first study that investigated gelotophobia in individuals with Asperger's syndrome. Samson et al. (2011) investigated gelotophobia and its relationship with recalled experiences of having been laughed at in the past in 40 individuals with Asperger's syndrome and in 83 neurotypically developing control group participants. It was found that in individuals with Asperger's syndrome, 45% exceeded the cut-off of having at least a slight fear of being laughed at. This was in comparison to only 6% of control participants who exceeded this cut-off. There were statistically significant higher levels of gelotophobia found in the Asperger's syndrome group, compared to the control group. It was found that 17.5% of individuals with Asperger's syndrome had a marked fear of being laughed at, with a further 7.5% endorsing a severe fear of being laughed at. High levels of gelotophobia were found in individuals with Asperger's syndrome in comparison to control group participants, when both groups had a high frequency of recalled experiences of being laughed at. Therefore, recalled experiences of being laughed at does not explain why individuals with Asperger's syndrome demonstrated high levels of gelotophobia.

In control group participants, who were measured on the Autism-Spectrum Quotient, gelotophobia was positively associated with autism spectrum level, which demonstrates a relationship between gelotophobia and Asperger's syndrome. The percentage of those with Asperger's syndrome who presented with gelotophobia (45%) was the highest percentage reported in the literature at that time.

Samson et al. (2011) outlined a number of reasons as to why gelotophobia may be more common in individuals with Asperger's syndrome. First, individuals with Asperger's syndrome may have a higher frequency of situations where they were laughed at in the past. Second, individuals with Asperger's syndrome often lack social awareness and therefore may have difficulties understanding how to deal with someone laughing at them. Third, individuals with

Asperger's syndrome may have difficulty reading social cues and may not be able to differentiate between good-natured teasing and bullying. Samson et al. (2011) provided a novel area of research for autism researchers to address.

Following Samson et al. (2011), there has been only a small number of studies that have investigated gelotophobia in individuals with ASD. Samson (2013) conducted a literature review on sense of humor in individuals with autism spectrum disorders (ASD). While focused on humor, the literature review provided a valuable summary of the literature on humor in ASD and briefly discussed gelotophobia. Wu et al. (2015) investigated the role of parental attachment in relation to gelotophobia in adolescents with ASD. Participants were 101 Taiwanese high school students with ASD with average intelligence and in 163 typically developing students. Adolescents with ASD showed significantly higher levels of gelotophobia than those without ASD. A negative relationship was found between attachment to fathers and gelotophobia in the ASD group. Therefore, as the quality of attachment to their fathers increased, gelotophobia decreased. However, no relationship was found between maternal attachment and gelotophobia. Therefore, by increasing the quality of father-child attachment in ASD, this may have implications for the occurrence of gelotophobia (Wu et al. 2015). The authors suggested that future research should investigate the relationship between attachment to peers and gelotophobia.

Tsai et al. (2018) investigated the relationship between gelotophobia and personality traits in 123 Taiwanese high school students with ASD and in 156 typically developing students. The ASD group demonstrated higher rates of gelotophobia than the control group. It was found that 42.3% of individuals with ASD had some form of gelotophobia, with 4.9% showing extreme gelotophobia, 4.9% with marked gelotophobia, and 35.8% with slight gelotophobia. The personality traits examined were the Big Five: Extraversion, Conscientiousness, Agreeableness, Openness, and Emotional Stability. Extraversion was found to be a mediator of gelotophobia. Therefore, individuals who

showed lower levels of extraversion experienced more gelotophobia, as these individuals were less able to engage in humor with others. The authors suggested that these results may be applicable to participants from an Asian cultural background, and therefore, the results of this research need to be replicated across different cultures.

Grennan et al. (2018) conducted a literature review on the link between gelotophobia and high-functioning ASD (hfASD). In this review, the characteristics of gelotophobia were discussed, including the conceptualization and measurement implications of gelotophobia, the etiology and consequences of gelotophobia, and the social competence of gelotophobes. The importance of studying gelotophobia in hfASD was discussed, as well as the relationships between hfASD and other variables relevant to gelotophobia, including comorbid psychopathology, quality of life, social functioning, perceived social support, past experiences of bullying, and shame-bound emotions. This review demonstrated the need for further research to be conducted in the area and discussed the importance of better understanding the relationship between gelotophobia and comorbid psychopathology in individuals with ASD.

Following this, Leader et al. (2018) conducted an empirical investigation specifically on individuals with hfASD, where gelotophobia was examined in 103 adults with hfASD and in 137 typically developing control participants. Moreover, it was the first study of its kind to explore the relationship between gelotophobia and other psychosocial constructs and the presence of psychopathological disorders in individuals with hfASD. The study examined gelotophobia in relation to a number of other variables, including social functioning, perceived social support, life satisfaction, quality of life, past experiences of bullying, and comorbid psychopathology. Individuals with hfASD presented with high rates of gelotophobia (87.4%) compared to 22.6% of control participants. This was a higher rate of gelotophobia, almost double, than what was found in the results of the Samson et al. (2011) study. Significantly higher levels of gelotophobia and higher past experiences of bullying were found in those with hfASD, in

comparison to typically developing control participants. The hfASD group showed lower quality of life, life satisfaction, social functioning, and perceived social support, in comparison to the control group. Of the 84.7% of participants with hfASD who presented with gelotophobia, 34% demonstrated a marked form of gelotophobia, 29.1% presented with an extreme form, and 24.3% presented with a slight form of gelotophobia.

Leader et al. (2018) found significant negative correlations between gelotophobia and quality of life, life satisfaction, perceived social support, and depression and anxiety. A significant positive correlation was found between gelotophobia and personal involvement in bullying and social functioning. The predictors of gelotophobia were examined in Leader et al. (2018). Social functioning, past experiences of bullying, anxiety, and life satisfaction were identified as predictors of gelotophobia. It was found that past experiences of verbal bullying were a stronger predictor of gelotophobia than physical and indirect forms of bullying.

Future Directions

Given the small number of studies that have focused on gelotophobia in individuals with ASD, there is a need for future research to focus on a number of specific directions. First, other methodologies should be used other than self-report. Self-report may lead to incidences of social desirability among participants. Where research requires individuals to report remembered situations of past experiences of bullying, self-report can be compromised by age-related memory loss. Future research could also include interviewing instead of questionnaires, field observation, and the use of peer and parental reports (Wu et al. 2015). Samson et al. (2011) outlined that peer-reports by teachers or parents could be used to investigate the influence of prior experiences of being laughed at, and whether this affects the development of gelotophobia later in life. Other peer report could come from other individuals in

a person's life, such as siblings or employers, in order to encapsulate the influence of bullying exposure on gelotophobia. Second, more research is needed to determine the relationship between gelotophobia and other fears and phobias. Samson et al. (2011) suggested that future research should focus on gelotophobia in relation to the development of social phobia. Leader et al. (2018) found that the presence of an anxiety disorder diagnosis was a predictor of gelotophobia. This possible link between gelotophobia and anxiety needs to be examined in future research. Third, research is needed on ASD populations other than individuals with hfASD. Gelotophobia needs to be investigated across the autism spectrum, from individuals with more severe symptoms of ASD to those with milder symptoms. Fourth, the relationship between gelotophobia and ASD severity needs to be better understood. Samson (2013) outlined that if the relationship between humor and ASD severity is investigated in future research studies, a model could be designed to explain the connections between symptom severity and the domains of humor in individuals with ASD.

Fifth, given the negative consequences of gelotophobia, research is needed to determine how gelotophobia can be treated. Gelotophobia must first receive further empirical investigative work in order to support its prevalence and further determine its consequences, symptomatology, and consequences in individuals with ASD. This will in the future lead to the development of specific intervention and preventative strategies for its treatment. While literature exists on strategies to reduce bullying in ASD, empirical evidence is needed on the intervention of and prevention of gelotophobia in individuals in ASD. This treatment may include learning to differentiate between teasing and mocking, and strategies which may help individuals with hfASD to disentangle harmless teasing from hostile bullying (Attwood 2004). Sixth, research is needed on the use of the Picture-Geloph in individuals with hfASD. The Picture-Geloph is a semi-projective tool, piloted by Ruch et al. (2009) which involves 20 cartoons depicting social situations which

involve laughter or potential laughter. Whether these cartoons are interpreted in the same way by individuals with hfASD as they are by individuals in the typically developing population is a question for future research to determine. Seventh, gelotophobia needs to be investigated with children with ASD. A child version of the PhoPhiKat (Ruch and Proyer 2009) has been developed, which measures gelotophobia, gelotophilia (i.e., the joy of being laughed at), and katagelasticism (i.e., the joy of laughing at others). The child version has been used in previous research with typically developing children (Proyer et al. 2012). Research is needed to determine the validity of this measure in children with ASD.

Finally, research is needed to determine the relationship between gelotophobia and alexitymanias in individuals with hfASD. Alexitymania is the inability to express emotions and has been identified as being an issue in individuals with ASD (Costa et al. 2017; Fitzgerald and Bellgrove 2006). A link between gelotophobia and alexitymania has been discovered (Boda-Ujlaki and Séra 2013), yet future research is needed to empirically explore these concepts in individuals with hfASD. In conclusion, the area of gelotophobia and ASD is a novel field of autism research, where much more research is needed to expand our understanding of the concept of gelotophobia in individuals with ASD.

See Also

- [Mental Health and ASD](#)
- [Social Behaviors and Social Impairment](#)

References and Reading

- Attwood, T. (2004). Strategies to reduce the bullying of young children with Asperger syndrome. *Australian Journal of Early Childhood*, 29(3), 15.
- Boda-Ujlaki, J. E., & Séra, L. (2013). Gelotophobia, alexithymia and emotional intelligence. *Mentálhigiéné és Pszichoszomatika*, 14, 297–321.
- Costa, A., Steffgen, G., & Samson, A. C. (2017). Expressive incoherence and alexithymia in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(6), 1659–1672.
- Fitzgerald, M., & Bellgrove, M. A. (2006). The overlap between alexithymia and Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 36(4), 573.
- Forabosco, G., Ruch, W., & Nucera, P. (2009). The fear of being laughed at among psychiatric patients. *Humor: International Journal of Humor Research*, 22(1), 233–251.
- Frith, U. (2004). Emanuel Miller lecture: Confusions and controversies about Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 45, 672–686.
- Grennan, S., Mannion, A., & Leader, G. (2018). Gelotophobia and high-functioning autism spectrum disorder. *Review Journal of Autism and Developmental Disorders*, 5(4), 349–359.
- Leader, G., Grennan, S., Chen, J. L., & Mannion, A. (2018). An investigation of gelotophobia in individuals with a diagnosis of high-functioning autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(12), 4155–4166.
- Proyer, R. T., Neukom, M., Platt, T., & Ruch, W. (2012). Assessing gelotophobia, gelotophilia, and katagelasticism in children: An initial study on how six to nine-year olds deal with laughter and ridicule and how this relates to bullying and victimization. *Child Indicators Research*, 5(2), 297–316.
- Ruch, W., & Proyer, R. T. (2008a). The fear of being laughed at: Individual and group differences in gelotophobia. *Humor: International Journal of Humor Research*, 21(1), 47–67.
- Ruch, W., & Proyer, R. T. (2008b). Who is gelotophobic? Assessment criteria for the fear of being laughed at. *Swiss Journal of Psychology*, 67, 19–27.
- Ruch, W., & Proyer, R. T. (2009). Extending the study of gelotophobia: On gelotophiles and katagelasticists. *Humor: International Journal of Humor Research*, 22(1–2), 183–212.
- Ruch, W., Altfreder, O., & Proyer, R. T. (2009). How do gelotophobes interpret laughter in ambiguous situations? An experimental validation of the concept. *Humor: International Journal of Humor Research*, 22(1–2), 63–89.
- Ruch, W., Hofmann, J., Platt, T., & Proyer, R. T. (2014). The state-of-the-art in gelotophobia research: A review and some theoretical extensions. *Humor: International Journal of Humor Research*, 27, 23–45.
- Samson, A. C. (2013). Humor(lessness) elucidated – Sense of humor in individuals with autism spectrum disorders: Review and introduction. *Humor: International Journal of Humor Research*, 26(3), 393–409.
- Samson, A. C., Huber, O., & Ruch, W. (2011). Teasing, ridiculing and the relation to the fear of being laughed at in individuals with Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 41, 475–483.

- Titze, M. (1996). The Pinocchio Complex: Overcoming the fear of laughter. *Humor & Health Journal*, 5, 1–11.
- Titze, M. (2009). Gelotophobia: The fear of being laughed at. *Humor: International Journal of Humor Research*, 22(1), 27–48.
- Tsai, M. N., Wu, C. L., Tseng, L. P., An, C. P., & Chen, H. C. (2018). Extraversion is a mediator of gelotophobia: A study of autism spectrum disorder and the Big Five. *Frontiers in Psychology*, 9, 150.
- Weiss, E. M., Schuler, G., Freudenthaler, H. H., Hofer, E., Pichler, N., & Papousek, I. (2012). Potential markers of aggressive behavior: The fear of other person's laughter and its overlaps with mental disorders. *PLoS One*, 7, e38088.
- Wing, L. (1981). Asperger syndrome: A clinical account. *Psychological Medicine*, 11, 115–129.
- Woods, A. G., Mahdavi, E., & Ryan, J. P. (2013). Treating clients with Asperger's syndrome and autism. *Child and Adolescent Psychiatry and Mental Health*, 7, 32–40.
- Wu, C. L., An, C. P., Tseng, L. P., Chen, H. C., Chan, Y. C., Cho, S. L., & Tsai, M. L. (2015). Fear of being laughed at with relation to parent attachment in individuals with autism. *Research in Autism Spectrum Disorders*, 10, 116–123.

referred to as “sex differences”) between the sexes.

Historical Background

Kanner's earliest writings about autism highlighted the now frequently documented gender difference in the prevalence of the disorder (1943). These early case studies, of eight boys and three girls, described few other gender differences within the sample; the male and female children were described similarly with respect to their limited social reciprocity and communication functioning and atypical interests and repetitive patterns of behavior. Nevertheless, since Kanner's time, there has been much speculation that gender differences exist in the clinical presentation of ASD. These concerns have been raised in no small part because much of the research on ASD has used clinic and research samples with a predominantly or exclusively male composition (Lai et al. 2014).

Genahist™ [OTC]

► Diphenhydramine

Gender Differences

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Definition

Gender differences in autism spectrum disorders (ASD) primarily refer to the behavioral phenotypic differences between affected males and females. Gender differences have also been examined with respect to the diagnosis and prevalence of ASD. Of note, the term “gender differences” is used here to describe socially defined as well as biologically defined differences (elsewhere

Current Knowledge

Researchers have proposed models of ASD that explain and predict gender differences such as Simon Baron-Cohen's “extreme male brain” theory which proposes that testosterone-driven differences in cognition are at the core of ASD (2005). Others propose a “female protective model”; this model states that females require larger genetic insults for ASD symptom expression and has found some support in genetic studies (Jacquemont et al. 2014). More recently, the concept of “camouflaging” has been used to explain the gender differences in ASD; the term is currently used in an interchangeable manner to describe a number of behaviors the use of compensatory strategies (Dean et al. 2017; Lai et al. 2017; Livingston and Happé 2017), the presence of an atypical cluster of symptoms (Kirkovski et al. 2013), as well as self-reported efforts to minimize or “hide” impairments (Bargiela et al. 2016;

Lai et al. 2017). However, despite the fact that there are very clear sex differences in the prevalence of ASD, the existing literature describes few differences in the behavioral and clinical presentation of ASD in males and females. The following are highlights of the work completed to date.

Current prevalence estimates place the overall male-to-female ratio of ASD at 3:1 (Loomes et al. 2017). In samples with moderate to severe intellectual disability, the male-to-female ratio decreases to 1.95:1, while in samples without cognitive impairment, the ratio is closer to 5.5:1 (see Fombonne 2005 for a review). There is also evidence that the male-to-female ratio in ASD is higher in “essential autism” (ASD with no significant congenital anomalies) than in “complex autism” or ASD associated with significant dysmorphology or microcephaly (Miles et al. 2005).

As with ASD prevalence, research studies have consistently found gender differences in cognitive functioning. Affected females, on average, have lower nonverbal and adaptive scores than males (Van Wijngaarden-Cremers et al. 2014; Frazier et al. 2014; Volkmar et al. 1993; Lord et al. 1982). Gender differences in language development merit further study. Overall, when compared to affected males, females demonstrate lower verbal ability and greater difficulties in the areas of language processing and comprehension (e.g.; Frazier et al. 2014; Konstantareas et al. 1989). In the area of pragmatic language skills, toddler girls demonstrate greater impairments than boys (Carter et al. 2007; Hartley and Sikora 2009); this gender difference is reversed in school-age samples (Conlon et al. 2019).

With respect to the core features of ASD, large-scale studies have been able to account for the effects of developmental factors (such as IQ and language level) and have found no gender differences in autism severity (Kaat et al. 2020; Tillmann et al. 2018). Similarly, gender differences have not been found in the social communication behavior of toddlers, children, and adults with autism (van Wijngaarden-Cremers et al. 2014). In contrast, studies that examine social

behaviors more generally find that female children demonstrate compensatory strategies (e.g., Dean et al. 2017) and present with relative strengths compared to males (Jamison et al. 2017; Head et al. 2014). Consistent with these findings, female children demonstrate fewer impairments than males on “higher-level” social communication items of the ADOS, behaviors that as a group are not specific to a diagnosis of ASD (Bishop et al. 2016). In this same study, no gender differences were found on items uniquely associated with ASD, those that assess basic social communication.

Various studies have identified gender differences in some aspects of restricted and repetitive behavior (RRBs). In samples of children, adolescents, and adults, but not toddlers, females with ASD demonstrate fewer RRB symptoms than males (Van Wijngaarden-Cremers et al. 2014). Recent studies reveal that this gender difference is found across all levels of functioning in RRB total scores of the ADI-R and RBS-R restricted interest scores (Frazier et al. 2014). Notably, RRBs described as “insistence on sameness” behaviors on the ADI-R do not appear to be predicted by gender (Hus et al. 2007; Richler et al. 2010). Qualitative examination of repetitive behavior in school-age children reveals further differences. Girls, as compared to boys, are less likely to engage in repetitive use of objects and more likely to demonstrate fixations, often on age-appropriate topics (Hiller et al. 2014).

In regard to the broader behavioral and psychological functioning of children and adults with ASD, results vary by developmental level and age. In a sample composed primarily of children and young adults with ASD and intellectual disability, males and females demonstrate similar levels of disruptive behavior, hyperactivity, and attentional difficulties (see, e.g., Brereton et al. 2006). In the Simons Simplex Study, comprised primarily of cognitively able children and adolescents with ASD, females demonstrate greater externalizing problems relative to males (Frazier et al. 2014) girls also show greater sleep problems, anxiety/mood, and emotional problems (Hartley

and Sikora 2009; Holtmann et al. 2007, Mandy et al. 2012). The findings with respect to attention problem and overactivity in children are mixed (Mandy et al. 2012; Frazier et al. 2014). In adolescence and adulthood, females diagnosed with ASD demonstrate greater increases in anxiety and depression (Gotham et al. 2015; Solomon et al. 2012). Self-report data also suggests such gender differences in mood disruptions, but not in anxiety (Lever and Geurts 2016).

Few studies have longitudinally examined gender differences in the core symptoms of ASD, and fewer still have included sufficient samples of females. Emerging research suggests that in early childhood, female children may demonstrate better trajectories than their male counterparts (Waizbard-Bartov et al. 2020). In adulthood, males and females present with similar clinical profiles (Howlin et al. 2004; Shattuck et al. 2007; Taylor and Seltzer 2010). However, when broader outcomes are examined (e.g., employment), there is evidence that adult females fare worse than males (Howlin et al. 2004; Taylor and Mailick 2014). Self-report from adult women with ASD further suggests that unique challenges arise when impairments associated with ASD intersect with cultural norms and expectations of women's behavior (Bargiela et al. 2016).

In sum, the existing body of research points to a clear gender difference in the prevalence ASD; the male-to-female ratio is estimated at 3:1, with a greater frequency of females at the lower end of cognitive ability. Taking together all of the currently available research, gender differences in the behavioral phenotype of ASD are subtle (e.g., qualitative differences in repetitive behavior) and reflect interactions among multiple factors (e.g., developmental level). At present, sex differences in social functioning are limited to broader areas of functioning that are nonspecific to ASD. Importantly, the current research on gender differences in ASD should be considered in light of the following: female children with ASD are less likely to have a formal diagnosis of ASD (Giarelli et al. 2010) and more likely to be diagnosed later than

their male peers (Begeer et al. 2013; McCormick et al. 2020).

Future Directions

While significant strides have been made in recent years to utilize large samples to examine differences between the male and female autism phenotype (see Kaat et al. 2020; Tillmann et al. 2018), further quantitative and qualitative examination of the gender differences in ASD in large and well-characterized samples of children and adults is needed. Important to this work will be our ability to identify other within-group differences in ASD (see Joseph et al. 2002; Miles et al. 2005; Lord et al. 2015; Stelios et al. 2017) and further refine our ability to assess the core symptoms of ASD across development and contexts (Anagnostou et al. 2015).

See Also

► [Subtyping Autism](#)

References and Reading

- Anagnostou, E., Jones, N., Huerta, M., Halladay, A. K., Wang, P., et al. (2015). Measuring social communication behaviors as a treatment endpoint in individuals with autism spectrum disorder. *Autism, 19*(5), 622–636.
- Banach, R., Thompson, A., Goldberg, J., Tuff, L., Zwaigenbaum, L., & Mahoney, W. (2009). Brief report: Relationship between non-verbal IQ and gender in autism. *Journal of Autism and Developmental Disorders, 39*(1), 188–193.
- Bargiela, S., Steward, R., & Mandy, W. (2016). The experiences of late-diagnosed women with autism spectrum conditions: An investigation of the female autism phenotype. *Journal of Autism and Developmental Disorders, 46*, 3281–3294.
- Baron-Cohen, S., Richler, J., Bisarya, D., Gurunathan, N., & Wheelwright, S. (2003). The systemizing quotient: An investigation of adults with Asperger syndrome or high functioning autism, and normal sex differences. *Philosophical Transactions of the Royal Society of London, 358*, 361–374.

- Begeer, S., Mandell, D., & Wijnker-Holmes, B. (2013). Sex differences in the timing of identification among children and adults with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43, 1151–1156.
- Bishop, S. L., Havdahl, K. A., Huerta, M., Lord, C. (2016). Subdimensions of social-communication impairment in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 57(8), 909–916. <https://doi.org/10.1111/jcpp.12510>
- Brereton, A. V., Tonge, B. J., & Einfeld, S. L. (2006). Psychopathology in children and adolescents with autism compared to young people with intellectual disability. *Journal of Autism and Developmental Disorders*, 36(7), 863–870.
- Carter, A. S., Black, D. O., Tewani, S., Connolly, C. E., Kadlec, M. B., & Tager-Flusberg, H. (2007). Sex differences in toddlers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(1), 86–97.
- Conlon, O., Volden, J., Smith, I. M., et al. (2019). Gender differences in pragmatic communication in school-aged children with autism spectrum disorder (ASD). *Journal of Autism and Developmental Disorders*, 49(5), 1937–1948. <https://doi.org/10.1007/s10803-018-03873-2>
- Dean, M., Harwood, R., & Kasari, C. (2017). The art of camouflage: Gender differences in the social behaviors of girls and boys with autism spectrum disorder. *Autism*, 21(6), 678–689. <https://doi.org/10.1177/1362361316671845>
- Fombonne, E. (2005). Epidemiology of autistic disorder and other pervasive developmental disorders. *The Journal of Clinical Psychiatry*, 66, 3–8.
- Frazier, T. W., Georgiades, S., Bishop, S. L., & Hardan, A. Y. (2014). Behavioral and cognitive characteristics of females and males with autism in the Simons Simplex Collection. *Journal of the American Academy of Child and Adolescent Psychiatry*, 53(3), 329–340.
- Giarelli, E., Wiggins, L. D., Rice, C. E., Levy, S. E., Kirby, R. S., Pinto-Martin, J., et al. (2010). Sex differences in the evaluation and diagnosis of autism spectrum disorders among children. *Disability and Health Journal*, 3(2), 107–116.
- Gotham, K., Brunwasser, S. M., Lord, C. (2015). Depressive and anxiety symptom trajectories from school age through young adulthood in samples with autism spectrum disorder and developmental delay. *Journal of the American Academy of Child and Adolescent Psychiatry*, 54(5), 369–76.e3. <https://doi.org/10.1016/j.jaac.2015.02.005>
- Hartley, S. L., & Sikora, D. M. (2009). Sex differences in autism spectrum disorder: An examination of developmental functioning, autistic symptoms, and coexisting behavior problems in toddlers. *Journal of Autism and Developmental Disorders*, 39, 1715–1722.
- Head, A. M., McGillivray, J. A., & Stokes, M. A. (2014). Gender differences in emotionality and sociability in children with autism spectrum disorders. *Molecular Autism*, 5, 19.
- Hiller, R. M., Young, R. L., & Weber, N. (2014). Sex differences in autism spectrum disorder based on DSM-5 criteria: Evidence from clinician and teacher reporting. *Journal of Abnormal Child Psychology*, 42, 1381–1393.
- Holtmann, M., Bolte, S., & Poustka, F. (2007). Autism spectrum disorders: Sex differences in autistic behaviour domains and coexisting psychopathology. *Developmental Medicine and Child Neurology*, 49(5), 361–366.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45, 212–229.
- Hus, V., Pickles, A., Cook, E. H., Risi, S., & Lord, C. (2007). Using the autism diagnostic interview-revised to increase phenotypic homogeneity in genetic studies of autism. *Biological Psychiatry*, 61(4), 438–448.
- Jacquemont, S., Coe, B. P., Hersch, M., et al. (2014). A higher mutational burden in females supports a “female protective model” in neurodevelopmental disorders. *American Journal of Human Genetics*, 94(3), 415–425. <https://doi.org/10.1016/j.ajhg.2014.02.001>
- Jamison, R., Bishop, S. L., Huerta, M., Halladay, A. K. (2017). The clinician perspective on sex differences in autism spectrum disorders. *Autism*, 21(6), 772–784. <https://doi.org/10.1177/1362361316681481>
- Joseph, R., Tager-Flusberg, H., & Lord, C. (2002). Cognitive profiles and social-communicative functioning in children with autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 43(6), 807–821.
- Kanner, L. (1943). Autistic disturbances of affective content. *The Nervous Child*, 2, 217–250.
- Kaat, A. J., Shui, A. M., Ghods, S. S., et al. (2020). Sex differences in scores on standardized measures of autism symptoms: a multisite integrative data analysis [published online ahead of print, 2020 Apr 20]. *Journal of Child Psychology and Psychiatry*. <https://doi.org/10.1111/jcpp.13242>
- Kim, S., & Lord, C. (2010). Restricted and repetitive behaviors in toddlers and preschoolers with autism spectrum disorders based on the autism diagnostic observation scheduled (ADOS). *Autism Research*, 3(4), 162–173.
- Kirkovski, M., Enticott, P. G., & Fitzgerald, P. B. (2013). A review of the role of female gender in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(11), 2584–2603. <https://doi.org/10.1007/s10803-013-1811-1>
- Klin, A., Saunier, C. A., Sparrow, S. S., Cicchetti, D. V., Volkmar, F. R., & Lord, C. (2007). Social and communication abilities and disabilities in higher functioning individuals with autism: The Vineland and the ADOS. *Journal of Autism and Developmental Disorders*, 37, 748–759.

- Konstantareas, M. M., Homatidis, S., & Busch, J. (1989). Cognitive, communication, and social differences between autistic boys and girls. *Journal of Applied Developmental Psychology, 10*(4), 411–424.
- Lai, M. C., Lombardo, M. V., & Auyeung, B. (2014). Sex/gender differences and autism: Setting the scene for future research. *Journal of the American Academy of Child and Adolescent Psychiatry, 54*, 11–24.
- Lai, M. C., Lombardo, M. V., Ruigrok, A. N., et al. (2017). Quantifying and exploring camouflaging in men and women with autism. *Autism, 21*(6), 690–702. <https://doi.org/10.1177/1362361316671012>
- Lever, A. G., Geurts, H. M. (2016). Psychiatric co-occurring symptoms and disorders in young, middle-aged, and older adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 46*, 1916–1930. <https://doi.org/10.1007/s10803-016-2722-8>
- Little, L. M., Wallisch, A., Salley, B., & Jamison, R. (2017). Do early caregiver concerns differ for girls with autism spectrum disorders? *Autism, 21*(6), 728–732.
- Livingston, L. A. & Happé, F. (2017). Conceptualising compensation in neurodevelopmental disorders: Reflections from autism spectrum disorder. *Neuroscience and Biobehavioral Reviews 80*: 729–42.
- Loomes, R., Hull, L., & Mandy, W. (2017). What is the male-to-female ratio in autism spectrum disorder? A systematic review and meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry, 56*(6), 466–474.
- Lord, C., Schopler, E., & Revicki, D. (1982). Sex differences in autism. *Journal of Autism and Developmental Disorders, 12*(4), 317–330.
- Lord, C., Bishop, S. L., & Anderson, D. (2015). Developmental trajectories as autism phenotypes. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics, 169*(2), 198–208.
- Luyster, R., Richler, J., Rise, S., Hsu, W., Dawson, G., Bernier, R., et al. (2005). Early regression in social communication in autism spectrum disorders: A CPEA study. *Developmental Neuropsychology, 27*(3), 311–336.
- Mandy, W., Chilvers, R., Chowdhury, U., Salter, G., Seigal, A., & Skuse, D. (2012). Sex differences in autism spectrum disorder: evidence from a large sample of children and adolescents. *Journal of Autism and Developmental Disorders, 42*(7), 1304–1313. <https://doi.org/10.1007/s10803-011-1356-0>
- McCormick, C. E. B., Kavanaugh, B. C., Sipsock, D., et al. (2020). Autism heterogeneity in a densely sampled U.S. population: Results from the first 1,000 participants in the RI-CART Study. *Autism Research, 13*(3):474–488. <https://doi.org/10.1002/aur.2261>
- McLennan, J. D., Lord, C., & Schopler, E. (1993). Sex differences in higher functioning people with autism. *Journal of Autism and Developmental Disorders, 23*(2), 217–227.
- Miles, J. H., Takahashi, T. N., Bagby, S., Sahota, P. K., Vaslow, D. F., Wang, C. H., et al. (2005). Essential versus complex autism: Definition of fundamental prognostic subtypes. *American Journal of Medical Genetics, 135*(A), 171–180.
- Richler, J., Huerta, M., Bishop, S., & Lord, C. (2010). Developmental trajectories of restrictive and repetitive behaviors and interests in children with autism spectrum disorders. *Development and Psychopathology, 22*(1), 55–69.
- Shattuck, P. T., Seltzer, M. M., Greenberg, J. S., Orsmond, G., Bolt, D., Kring, S., et al. (2007). Change in autism symptoms and maladaptive behaviors in adolescents and adults with an autism spectrum disorder. *Journal of Autism and Developmental Disorders, 37*(9), 1735–1747.
- Solomon, M., Miller, M., Taylor, S. L., Hinshaw, S. P., & Carter, C. (2012). Autism symptoms and internalizing psychopathology in girls and boys with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 42*(1), 48–59.
- Stelios, G., Bishop, S. L., & Frazier, T. (2017). Editorial perspective: Longitudinal research in autism- introducing the concept of ‘chronogeneity’. *Journal of Child Psychology and Psychiatry, 58*(5), 634–636.
- Taylor, J. L., Mailick, M. R. (2014). A longitudinal examination of 10-year change in vocational and educational activities for adults with autism spectrum disorders. *Developmental Psychology, 50*(3), 699–708. <https://doi.org/10.1037/a0034297>
- Taylor, J. L., & Seltzer, M. M. (2010). Changes in the autism behavioral phenotype during the transition to adulthood. *Journal of Autism and Developmental Disorders, 40*, 1431–1446.
- Tillmann, J., Ashwood, K., Absoud, M., Bolte, S., Bonnet-Brilhault, F., Buitelaar, J. K., et al. (2018). Evaluating sex and age differences in ADI-R and ADOS scores in a large European multi-site sample of individuals with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 48*(7), 2490–2505. Advance online publication.
- Van Wijngaarden-Cremers, P. J., van Eeten, E., Groen, W. B., Van Deurzen, P. A., Oosterling, I. J., & Van der Gaag, R. J. (2014). Gender and age differences in the core triad of impairments in autism spectrum disorders: A systematic review and meta-analysis. *Journal of Autism and Developmental Disorders, 44*(3), 627–635.
- Volkmar, F. R., Szatmari, P., & Sparrow, S. S. (1993). Sex differences in pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 23*(4), 579–591.
- Waizbard-Bartov, E., Ferrer, E., Young, G. S., et al. (2020). Trajectories of autism symptom severity change during early childhood [published online ahead of print, 2020 May 14]. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-020-04526-z>
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders, 9*(1), 11–29.

Gender Dysphoria and Autism Spectrum Disorders

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Definition

The term Gender Dysphoria (GD) is defined as a mismatch between the phenotypic sex of an individual and that person's perception of their biological gender (American Psychiatric Association 2013), and was previously defined as Gender Identity Disorder (American Psychiatric Association 2000; World Health Organization 1992). GD hereby used to describe Gender Identity Disorder (GID), and Gender Dysphoria (GD) is increasingly reported in ASD research.

Background

The term gender refers not to the sex one is born with but to the psychological identification of oneself as the traditional dichotomy female or male, but also as both, neither, or something else regardless of the physical attributes. The awareness of one's gender usually emerges around 18 months to 3 years of age. However, sometimes an individual's natal sex is not the same as their gender (George and Stokes 2017a). In DSM-5, the term "gender dysphoria" (GD) is used and van Schalkwyk et al. (2015) points out the difference in terminology from the DSM-IV,

where the diagnosis previously was named "gender identity disorder," which gave the impression that being transgender was a form of mental illness. There are still some concerns regarding the criteria in DSM-5 that the individual must experience a certain presence of distress to be diagnosed, which might result in individuals experiencing perhaps confusion, but not distress, not receiving proper help and guidance (van Schalkwyk et al. 2015). This process towards an acceptance of a more diverse view of gender identity and gender roles can be compared to the one of homosexuality, which was classified as a mental illness by the American psychiatric association until 1987 (Burton 2015). It is estimated that between 0.005–0.014% of all natal males and between 0.002–0.003% of all natal females would have been diagnosed with GD following current diagnostic criteria (George and Stokes 2017b). GD can, for instance, be expressed by a wish to dress in clothes typically worn by the opposite sex (defined by that individual's cultural norms), adapting to cross-gender norms, and typical thought patterns related to the opposite sex.

Current Knowledge

There has been increased attention towards individuals with ASD who also express gender-related issues in the past years, and both case reports and systematic studies have been published showing that it is apparent that there is some overlap between ASD and GD (George and Stokes 2017b). A Dutch study (De Vries et al. 2010) looked at the rates of ASD in 204 children and adolescents referred to a gender identity clinic, finding that 7.8% ($n = 16$) of them were thought to qualify as having ASD. Of these individuals, one in seven children had persistent GID (they used DSM-IV, which is more inclusive concerning criteria compared to ICD-10). Five of nine adolescents met criteria for GID, and the remaining met the criteria for Gender Identity Disorder Not Otherwise Specified (GID-NOS) or transvestic fetishism, leading the authors to conclude that ASD is more common in GID individuals (De Vries et al. 2010). van Schalkwyk et al.

(2015) points out that this conclusion might be more correctly rephrased as “ASD is more common in individuals with a broad range of gender-related concerns or questions” (van Schalkwyk et al. 2015). They also suggest that gender-formation in children can be seen as a process in development, where children reflect upon their identity through and by their gender and eventually forming an idea of this. However, due to large variations in cognitive and social development in children, this might be difficult to quantify by age-groups in research. This concept of gender-formation in childhood as a process with many possible outcomes is supported by research, where children with GD were followed up 10 years later, showing that only 27% of the individuals still qualified for a GD diagnosis. The majority of the non-GD individuals had instead developed a non-heterosexual identity. This indicates that gender identity and sexuality in some children are fluid concepts that later in puberty tend to crystallize into a more “final” form. ASD children display different patterns in social development, so this development of gender- and sexual identity might take on other forms or time perspectives, compared to TD children (van Schalkwyk et al. 2015). It is clear that co-occurrence of ASD and GD can lead to significant confusion, distress, and difficulties for the affected individual, primarily if the individual is not supported and guided. Therefore, it is essential for clinicians, health personnel, school teachers, and relatives to be informed and prepared to help and guide ASD individuals and to be aware of the increased possibility of confusion and uncertainty regarding their gender identity and sexuality already from an early age.

Pasterski et al. (2014) examined the co-occurrence of GD and autistic traits in an adult population with a confirmed diagnosis of transsexualism. To measure autistic traits, the Autism Spectrum Quotient (AQ) was administered. It is a self-administered questionnaire designed to measure where an individual lies on the spectrum, from normal to autistic. It uses a Likert scale comprised of five scales: social skills, attention switching, attention to detail, communication, and imagination. A person who scores high on the AQ scale are often not very skilled at social interaction, have an

unusual ability to focus, but struggles with switching attention, have an eye for details but lacks imagination, traits prevalent among individuals on the spectrum. Their results showed that 7.1% in Female to Male (FtM) and 4.8% in Male to Female (MtF) individuals scored above the estimated threshold for a potential diagnosis of autism, a clear average above the prevalence in the general population. The result that there were no significant differences in the co-occurrence of ASD and GD between natal females and males differs somewhat from other reports. Other studies have found that there’s a higher ratio of natal males in childhood who present with ASD and GD diagnoses, separately (Pasterski et al. 2014), and in the similar study by Jones et al. (2012) where they compared FtM and MtF transsexuals and found that the FtM group displayed a higher mean AQ than control groups of both sexes and the MtF group. They suggested that the Extreme Male Brain (EMB) theory might explain autistic traits as a part of a masculinized phenotype in FtM transsexuals.

A recent study examined the relationship between GD, ASD, and sexual orientation by looking for GD traits, and not the diagnosis, in a population of individuals diagnosed with ASD. They further hypothesized that GD traits would have a mediating effect between sexual orientation and ASD traits (George and Stokes 2017b). Their results concluded that the earlier seen association between GD traits and autistic traits also is true the reversed direction. Autistic individuals score higher on the GD scale than the typically developing control group, indicating that there is a bigger gender-variance in an autistic population. Autistic persons born as women only differed from those borne as men only on one of the subscales. Levels of GD traits were significantly higher among all other sexuality groups (homosexuality, bisexuality, asexuality, and other) than heterosexuality, and by heterosexuality, the authors refer to being attracted to the opposite of one’s natal sex. The EMB theory is also discussed here, with similar conclusions as mentioned earlier that high levels of fetal testosterone (fT) to some degree might explain the heightened rate of GD in ASD females, by shaping the individual in a masculine way. However, this reasoning does not explain the same heightened

rates of GD in males with ASD (George and Stokes 2017b).

The internet has made explicit sexual material available for the public, and there are countless of other sources that we are daily exposed to, providing different messages of relations, sexuality, norms, and ideals (George and Stokes 2017b). These changes have also sparked new life into the research on sexuality, and also on the topic of autism and sexuality. Flirting, courting, and relationships are activities that many typically developing (TD) individuals find challenging at times, and with an ASD where one of the most prominent traits concerns difficulties in the social domain, this can prove especially challenging. There are not a plethora of studies on autism and sexuality, and the few have been conducted on institutionalized, autistic male individuals with varying cognitive abilities living in group homes. These individuals were often reported engaging in more or less problematic sexual behavior, such as masturbating in public and inappropriate removal of clothes in front of others. Important to note though is that the studied subjects might have influenced the results from these studies and may not be representable for so-called “high functioning” autistic individuals living successfully as a part of society (George and Stokes 2017b). It is worth noting though that these most studies have had small- and gender-atypical male samples might have obscured more correct tendencies (Gilmour et al. 2012). The study by George and Stokes (2017b) found higher rates of asexuality in ASD individuals compared to TD but speculate that there might be several reasons for this. It is not uncommon for ASD individuals to experience sensory issues, hypersensitivity, tactile defensiveness, and other physical obstacles and might also find social interaction challenging. These factors might make lead to a decision of celibacy, even though it necessarily doesn't mean that the individual lacks feelings of sexual desire. Males and females with ASD also reported higher rates of bisexuality compared to their sex-matched TD group, supporting the finding by Gilmour et al. (2012). Several participants expressed that either heterosexual or homosexual sexual identity did not describe their identity correct but suggested others such as asexual, intersexual,

transsexual, and “other.” George and Stokes (2017b) discuss the EMB theory and points out that higher than usual levels of fT among males could be expected to lead to a hyper-masculinized individual resulting in heterosexuality. There is no convincing evidence for this connection. Some studies find this to be the case, and others have found opposite results where male ASD individuals have more homosexual tendencies, some found no relationship, and other studies again have found a relationship between lower fT and higher homosexuality. It is clear that there is no consensus on how fT affects the sexuality of males.

Future Directions

The field of autism spectrum disorders (ASD) and gender dysphoria (GD, here also includes GID) have seen a massive increase in research on this somewhat rare co-occurrence over the past decades. Notably, a considerable rise can be found in studies published in scientific journals over the past 3–4 years (Øien et al. 2018). This increased interest in gender dysphoria, trends, and patterns could explain sexuality and ASD we see elsewhere in the society, where sexual attitudes that previously were taboo is receiving increasing acceptance, and the empowering of the LGBT movement has also been seen. Furthermore, it is likely that the technologies such as social media and easier access to relevant information and internet communities have affected and inspired individuals to be open about their sexuality and provided them with a way of interacting and discussing their difficulties with someone in the same situation. While we do see a significant improvement in attitudes and acceptance for sexual minorities, there is still a long way to go before we meet a global improvement for all of these sexual minorities. Most studies are conducted in liberal Western countries, and little is currently known about the co-occurrence in more conservative countries where, e.g., homosexuality is still highly taboo.

Besides from the demand for more studies from other cultures, the most prominent difficulty for the field of GD and ASD research is

the fact that the co-occurrence is rare, and thus very few individuals are to be recruited to studies.

See Also

► [DSM-5](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (Vol. 1, 4th ed.). Arlington: American Psychiatric Publishing. <https://doi.org/10.1176/appi.books.9780890423349>.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. Washington, DC: American Psychiatric Publishing.
- Burton, N. (2015, 09.18.2015). *When homosexuality stopped being a mental disorder*. Retrieved 27 June 2018 from <https://www.psychologytoday.com/blog/hide-and-seek/201509/when-homosexuality-stopped-being-mental-disorder>.
- De Vries, A. L., Noens, I. L., Cohen-Kettenis, P. T., van Berckelaer-Onnes, I. A., & Doreleijers, T. A. (2010). Autism spectrum disorders in gender dysphoric children and adolescents. *Journal of Autism and Developmental Disorders*, 40(8), 930–936.
- George, R., & Stokes, M. (2017a). Sexual orientation in autism spectrum disorder. *Autism Research*, 11, 133–141.
- George, R., & Stokes, M. A. (2017b). Gender identity and sexual orientation in autism spectrum disorder. *Autism*. <https://doi.org/10.1177/1362361317714587>.
- Gilmour, L., Schalom, P. M., & Smith, V. (2012). Sexuality in a community based sample of adults with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 6(1), 313–318.
- Jones, R. M., Wheelwright, S., Farrell, K., Martin, E., Green, R., Di Ceglie, D., & Baron-Cohen, S. (2012). Brief report: Female-to-male transsexual people and autistic traits. *Journal of Autism and Developmental Disorders*, 42(2), 301–306.
- Øien, R. A., Cicchetti, D. V., & Nordahl-Hansen, A. (2018). Gender dysphoria, sexuality and autism spectrum disorders: a systematic map review. *Journal of Autism and Developmental Disorders*, 1–10.
- Pasterski, V., Gilligan, L., & Curtis, R. (2014). Traits of autism spectrum disorders in adults with gender dysphoria. *Archives of Sexual Behavior*, 43(2), 387–393.
- van Schalkwyk, G. I., Klingensmith, K., & Volkmar, F. R. (2015). Gender identity and autism spectrum disorders. *The Yale Journal of Biology and Medicine*, 88(1), 81.
- World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders*. Geneva: World Health Organization.

Gene Regulatory Networks in Autism

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Structure

Findings from genetic studies of autism spectrum disorder (ASD) reflect a central divergence as to whether the majority of genetic risk arises from common variation that is frequent but with low penetrance, or from rare variation with a high degree of penetrance and large effect. ASD is characterized by a high degree of both genetic and phenotypic heterogeneity. Despite long-standing evidence that ASD is a highly heritable, polygenic disorder, there still remains some uncertainty about the full allelic spectrum that underlies ASD.

With the advent of next-generation sequencing technologies, researchers have focused on the contribution of de novo variation through whole exome sequencing (WES) studies and genome-wide genotyping arrays to identify rare single nucleotide variants (SNVs), insertion-deletions (indels), and copy number variants (CNVs) associated with ASD. The identification of such variation in ASD-diagnosed individuals has resulted in the discovery of dozens of definitive ASD risk loci and genes over the past decade (De Rubeis et al. 2014; Dong et al. 2014; Iossifov et al. 2014; Iossifov et al. 2012; Neale et al. 2012; O’Roak et al. 2012; Pinto et al. 2014; Sanders et al. 2012), and confirmed the critical role of de novo germline mutation in the phenotypic presentation of ASD (Sanders et al. 2015).

Until recently, studies that attempted to investigate the role of common variation in ASD often proved insufficiently powered to reliably identify statistically significant findings. A population-based study resolved such limitations by analyzing the complex interplay of common, rare, inherited, and de novo variation across a Swedish epidemiological sample (Gaugler et al. 2014). The study concluded that while high-impact rare variation,

especially *de novo* mutations, may largely determine the individual liability for developing ASD, common variants account for the most substantial contribution to the narrow-sense heritability.

In the investigation of *de novo* variation implicated in ASD, the most common study design involves the study of simplex families, either classified as trios (affected proband and two unaffected parents) or quartets that also include at least one unaffected sibling. Across these studies, most estimates suggest that as many as 1000 genomic loci underlie risk for ASD. To assess the contribution of risk-conferring *de novo* mutations, researchers have assessed the recurrence of disruptive genetic events within the same gene or genomic loci as well as the significance of such recurrence, while burden analyses evaluated the presence or absence of significant enrichment among classes of mutations in cases compared to controls.

Across multiple studies of genetic variation in ASD, burden analyses consistently demonstrate a significant excess of *de novo* loss-of-function (LoF) or likely-gene-disrupting (LGD) mutations in probands that result in a premature truncation of the gene, shift in the protein reading frame, or canonical splice site variant across large cohorts (De Rubeis et al. 2014; Iossifov et al. 2012; O’Roak et al. 2011; Sanders et al. 2015). While statistical evidence for the contribution of *de novo* missense mutations in ASD appears less robustly replicated in the literature, some studies have demonstrated the importance of such variation in the development of ASD (Sanders et al. 2012), especially those missense variants predicted to be damaging by tools such as PolyPhen-2 (De Rubeis et al. 2014).

Multiple studies have confirmed the importance of rare CNVs as a source of risk in the genomic architecture of ASD (Bucan et al. 2009; Kumar et al. 2008; Marshall et al. 2008; Mefford et al. 2008; Moreno-De-Luca et al. 2013; Pinto et al. 2010, 2014; Sanders et al. 2011; Sanders et al. 2015; Sebat et al. 2007; Weiss et al. 2008). Indeed, two studies have further shown that *de novo* CNVs in individuals diagnosed with ASD demonstrated greater frequency, length, and intersection of disrupted genes when compared to

controls (Pinto et al. 2014; Sanders et al. 2015). Beyond such findings, one study hypothesized that separate classes of mutations may converge on a common set of genes associated with ASD.

Function

Bolstered by the development of advanced statistical modeling methods and software packages, researchers have sought to translate genetic findings of *de novo* variation associated with ASD into a broad understanding of the pathophysiological mechanisms that underlie ASD neurobiology. The central methodology of such translational efforts remains guided by two central principles: (1) recurrent *de novo* LGD or damaging missense mutations that occur within the same gene in unrelated individuals point toward genes that are likely to be associated with ASD, given the infrequency of such occurrences; and (2) risk loci and risk genes that share the same associated phenotype will converge in functional clusters that correlate to shared biological pathways, regulatory functions, developmental context, or coexpression networks.

Several seminal papers that investigated genetic regulatory networks implicated in ASD with a statistically robust framework led to a finding widely replicated throughout the literature: a convergence on genes that encode for critical components of networks involved in proper synaptic function, chromatin modification, and the regulation of transcription in ASD (De Rubeis et al. 2014; Pinto et al. 2014; Sanders et al. 2015). Among these genes, both *de novo* CNVs and *de novo* LGD mutations have been shown to play a role in establishing such convergence, further emphasizing the possibility that different classes of variation may target a common biological set of ASD risk genes.

These pathways, however, include a diverse variety of biological components, from voltage-gated ion channels to histone-modifying enzymes. Specifically, one report identified significant enrichment for histone-modifier genes, which encode histone-modifying enzymes (De Rubeis et al. 2014). These histone-modifying enzymes work in tandem with chromatin remodelers to

read and recognize posttranslational modifications and ultimately activate or repress transcription. Another report further hypothesized that genes involved in chromatin modification may in fact regulate the expression of synaptic genes (Sanders et al. 2015). Such a theory draws support from findings that show both direct and indirect regulatory targets of CHD8 are strongly enriched for genes both associated with ASD and implicated in neuronal and developmental pathways that control synapse formation (Cotney et al. 2015; Sugathan et al. 2014).

Indeed, while clear evidence exists for the role of synaptic proteins and chromatin remodelers in the pathophysiology of ASD, a polygenic, complex disorder such as ASD likely results from disruptions in multiple distinct biological pathways. Studies of genetic regulatory networks in ASD have demonstrated the potential for future diagnostic and therapeutic targets through the convergence of ASD-risk genes in biological pathways related to neuronal development, kinase signaling cascades, as well as brain-expressed genes (Geschwind and State 2015). Rare gene-disruptive events in ASD have further been shown to occur in genes found to interact with the neuronal RNA-binding protein known as fragile X mental retardation protein, or FMRP-associated genes (Darnell et al. 2011; Iossifov et al. 2012; Pinto et al. 2014). The significant enrichment of FMRP targets among ASD risk genes further emphasizes the role of proper human adaptation, neuroplasticity, and synaptic function in protecting against the development of ASD.

In keeping with the theory that ASD-associated genes may form functional networks or clusters, convergence may exist across diverse biological functions, but in a highly specific developmental context. Indeed, a large number of genes that gave rise to the significant signal for enrichment among chromatin and transcription regulators involved genes with a prenatal expression profile (Pinto et al. 2014). Using data from the BrainSpan atlas, one study demonstrated highly significant findings for the convergence of coexpression networks harboring ASD-associated genes underlying the proper function of deep-layer glutamatergic cortical projection neurons in

two overlapping periods of midfetal human development corresponding to 10–24 post-conception weeks (Willsey et al. 2013). Such a result highlights the promise that clues about the pathophysiological mechanisms implicated in ASD may exist not just in shared biological functions but also in shared cell types or developmental timeframes.

Path of Physiology

Narrow-sense heritability estimates for ASD range from 17% to 50%, with such wide discrepancies likely derived from differences in study design, cohort composition (e.g., simplex vs. multiplex families), sample size, and ascertainment bias. Though as previously discussed, a population-based study suggested that narrow-sense heritability is roughly 52% for ASD, with most of the total heritability explained by common variation and only a small fraction attributable to rare de novo mutations (Gaugler et al. 2014; Klei et al. 2012).

However, though these de novo events contribute modestly to the variance in liability for ASD, de novo carrier status determined affected status for ASD in 80% of de novo CNV carriers and 57% of de novo LGD carriers (Gaugler et al. 2014). A separate study of the genomic risk loci underlying ASD found that roughly 70% of de novo CNVs and 46% of de novo LGD mutations carried ASD risk, with both estimates higher among females than males (Sanders et al. 2015). Of individuals affected with ASD, multiple studies estimate the contribution of de novo CNVs and/or de novo SNVs in mediating ASD risk at roughly 10% (Iossifov et al. 2012; Sanders et al. 2015).

Phenotypically, deviations in head circumference in either direction, a history of seizures, and the presence of an unaffected sibling in the family all increased the mutational burden of a de novo event. While several studies have demonstrated a significant increase in ASD risk with increased paternal age (De Rubeis et al. 2014; Dong et al. 2014; Iossifov et al. 2012; O’Roak et al. 2012; Saha et al. 2009; Sanders et al. 2012), other studies did not observe such a difference with either a

higher paternal or maternal age (Sanders et al. 2015). Additionally, investigations of the genetic risk architecture of ASD, particularly the demonstrated convergence of genomic risk loci in regulatory networks associated with synaptic function, have shown that common genetic mechanisms may underlie frequently comorbid disorders such as intellectual disability (ID), schizophrenia, developmental delay, and epilepsy (De Rubeis et al. 2014; Sanders et al. 2015; Zoghbi and Bear 2012).

See Also

► Genetics

References and Reading

- Bucan, M., Abrahams, B. S., Wang, K., Glessner, J. T., Herman, E. I., Sonnenblick, L. I., et al. (2009). Genome-wide analyses of exonic copy number variants in a family-based study point to novel autism susceptibility genes. *PLoS Genetics*, 5(6), e1000536.
- Cotney, J., Muhle, R. A., Sanders, S. J., Liu, L., Willsey, A. J., Niu, W., et al. (2015). The autism-associated chromatin modifier CHD8 regulates other autism risk genes during human neurodevelopment. *Nature Communications*, 6, 6404.
- Darnell, J. C., Van Driesche, S. J., Zhang, C., Hung, K. Y., Mele, A., Fraser, C. E., et al. (2011). FMRP stalls ribosomal translocation on mRNAs linked to synaptic function and autism. *Cell*, 146(2), 247–261.
- De Rubeis, S., He, X., Goldberg, A. P., Poultney, C. S., Samocha, K., Cicek, A. E., et al. (2014). Synaptic, transcriptional and chromatin genes disrupted in autism. *Nature*, 515(7526), 209–215.
- Dong, S., Walker, M. F., Carriero, N. J., DiCola, M., Willsey, A. J., Ye, A. Y., et al. (2014). De novo insertions and deletions of predominantly paternal origin are associated with autism spectrum disorder. *Cell Reports*, 9(1), 16–23.
- Gaugler, T., Klei, L., Sanders, S. J., Bodea, C. A., Goldberg, A. P., Lee, A. B., et al. (2014). Most genetic risk for autism resides with common variation. *Nature Genetics*, 46(8), 881–885.
- Geschwind, D. H., & State, M. W. (2015). Gene hunting in autism spectrum disorder: on the path to precision medicine. *Lancet Neurology*, 14(11), 1109–1120.
- Iossifov, I., O’Roak, B. J., Sanders, S. J., Ronemus, M., Krumm, N., Levy, D., et al. (2014). The contribution of de novo coding mutations to autism spectrum disorder. *Nature*, 515(7526), 216–221.
- Iossifov, I., Ronemus, M., Levy, D., Wang, Z., Hakker, I., Rosenbaum, J., et al. (2012). De novo gene disruptions in children on the autistic spectrum. *Neuron*, 74(2), 285–299.
- Klei, L., Sanders, S. J., Murtha, M. T., Hus, V., Lowe, J. K., Willsey, A. J., et al. (2012). Common genetic variants, acting additively, are a major source of risk for autism. *Mol Autism*, 3(1), 9.
- Kumar, R. A., KaraMohamed, S., Sudi, J., Conrad, D. F., Brune, C., Badner, J. A., et al. (2008). Recurrent 16p11.2 microdeletions in autism. *Human Molecular Genetics*, 17(4), 628–638.
- Marshall, C. R., Noor, A., Vincent, J. B., Lionel, A. C., Feuk, L., Skaug, J., et al. (2008). Structural variation of chromosomes in autism spectrum disorder. *American Journal of Human Genetics*, 82(2), 477–488.
- Mefford, H. C., Sharp, A. J., Baker, C., Itsara, A., Jiang, Z., Buysse, K., et al. (2008). Recurrent rearrangements of chromosome 1q21.1 and variable pediatric phenotypes. *The New England Journal of Medicine*, 359(16), 1685–1699.
- Moreno-De-Luca, D., Sanders, S. J., Willsey, A. J., Mulle, J. G., Lowe, J. K., Geschwind, D. H., et al. (2013). Using large clinical data sets to infer pathogenicity for rare copy number variants in autism cohorts. *Molecular Psychiatry*, 18(10), 1090–1095.
- Neale, B. M., Kou, Y., Liu, L., Ma’ayan, A., Samocha, K. E., Sabo, A., et al. (2012). Patterns and rates of exonic de novo mutations in autism spectrum disorders. *Nature*, 485(7397), 242–245.
- O’Roak, B. J., Deriziotis, P., Lee, C., Vives, L., Schwartz, J. J., Girirajan, S., et al. (2011). Exome sequencing in sporadic autism spectrum disorders identifies severe de novo mutations. *Nature Genetics*, 43(6), 585–589.
- O’Roak, B. J., Vives, L., Girirajan, S., Karakoc, E., Krumm, N., Coe, B. P., et al. (2012). Sporadic autism exomes reveal a highly interconnected protein network of de novo mutations. *Nature*, 485(7397), 246–250.
- Pinto, D., Delaby, E., Merico, D., Barbosa, M., Merikangas, A., Klei, L., et al. (2014). Convergence of genes and cellular pathways dysregulated in autism spectrum disorders. *American Journal of Human Genetics*, 94(5), 677–694.
- Pinto, D., Pagnamenta, A. T., Klei, L., Anney, R., Merico, D., Regan, R., et al. (2010). Functional impact of global rare copy number variation in autism spectrum disorders. *Nature*, 466(7304), 368–372.
- Saha, S., Barnett, A. G., Foldi, C., Burne, T. H., Eyles, D. W., Buka, S. L., et al. (2009). Advanced paternal age is associated with impaired neurocognitive outcomes during infancy and childhood. *PLoS Medicine*, 6(3), e40.
- Sanders, S. J., Ercan-Sencicek, A. G., Hus, V., Luo, R., Murtha, M. T., Moreno-De-Luca, D., et al. (2011). Multiple recurrent de novo CNVs, including duplications of the 7q11.23 Williams syndrome region, are strongly associated with autism. *Neuron*, 70(5), 863–885.
- Sanders, S. J., He, X., Willsey, A. J., Ercan-Sencicek, A. G., Samocha, K. E., Cicek, A. E., et al. (2015). Insights into Autism Spectrum Disorder Genomic Architecture and Biology from 71 Risk Loci. *Neuron*, 87(6), 1215–1233.

- Sanders, S. J., Murtha, M. T., Gupta, A. R., Murdoch, J. D., Raubeson, M. J., Willsey, A. J., et al. (2012). De novo mutations revealed by whole-exome sequencing are strongly associated with autism. *Nature*, *485*(7397), 237–241.
- Sebat, J., Lakshmi, B., Malhotra, D., Troge, J., Lese-Martin, C., Walsh, T., et al. (2007). Strong association of de novo copy number mutations with autism. *Science*, *316*(5823), 445–449.
- Sugathan, A., Biagioli, M., Golzio, C., Erdin, S., Blumenthal, I., Manavalan, P., et al. (2014). CHD8 regulates neurodevelopmental pathways associated with autism spectrum disorder in neural progenitors. *Proceedings of the National Academy of Sciences of the United States of America*, *111*(42), E4468–E4477.
- Weiss, L. A., Shen, Y., Korn, J. M., Arking, D. E., Miller, D. T., Fossdal, R., et al. (2008). Association between microdeletion and microduplication at 16p11.2 and autism. *The New England Journal of Medicine*, *358*(7), 667–675.
- Willsey, A. J., Sanders, S. J., Li, M., Dong, S., Tebbenkamp, A. T., Muhle, R. A., et al. (2013). Coexpression networks implicate human midfetal deep cortical projection neurons in the pathogenesis of autism. *Cell*, *155*(5), 997–1007.
- Zoghbi, H. Y., & Bear, M. F. (2012). Synaptic dysfunction in neurodevelopmental disorders associated with autism and intellectual disabilities. *Cold Spring Harbor Perspectives in Biology*, *4*(3), a009886.

TR; American Psychiatric Association [APA] 2000). Individuals suffering from general anxiety commonly experience distress with accompanying disturbances of sleep, concentration, and social or occupational/academic functioning.

Categorization

Diagnostic criteria for GAD as defined by the DSM-IV-TR (APA 2000) are as follows:

- Excessive anxiety and worry that occur more days than not for at least 6 months, about a variety of events, circumstances, or activities (such as work or school performance).
- The worry is difficult for the person to control.
- The anxiety and worry are associated with three or more (in children, one or more) of these six symptoms (with at least some symptoms present for more days than not for the past 6 months): restlessness or feeling keyed up or on edge, being easily fatigued, difficulty concentrating or mind going blank, irritability, muscle tension, and sleep disturbance (difficulty falling or staying asleep, or restless unsatisfying sleep).

General Anxiety

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Synonyms

GAD

Short Description or Definition

General anxiety, or generalized anxiety disorder (GAD), is marked by excessive anxiety and worry occurring more days than not for at least 6 months, about a number of events or activities (DSM-IV-

The diagnostic criteria for GAD apply to individuals with ASD; however, it is important to consider that symptoms may be qualitatively different with variable behavioral manifestations within the context of an ASD (cf. Wood and Gadow 2010). For example, anxiety may be more associated with acting out behaviors in children with ASD than in typically developing children (White et al. 2009). Individuals with ASD often have limited insight or ability to express emotion (e.g., Hill et al. 2004), which may lead to frustration and consequent acting out behaviors. Careful assessment is needed to determine whether observed behaviors reflect ASD, anxiety, disruptive behavior symptoms, or a combination thereof.

Epidemiology

Anxiety disorders are prevalent among youth and adults with ASD. Specific phobias, social anxiety

(social phobia), and GAD are common subtypes. Epidemiological reports of large and/or national samples indicate that anxiety disorders in typical youth range from 10% to 20% (Achenbach et al. 1998; Shaffer et al. 1996). An early study indicated that approximately 4.9% of typical adolescents met criteria for GAD at some point in their lives (Whitaker et al. 1990).

Symptoms of anxiety may be the most common associated psychiatric concern among individuals with ASD (Skokauskas and Gallagher 2010) and occur so frequently that they are sometimes assumed to be a component of the disorder. In fact, in many cases, it is difficult to distinguish between symptoms of anxiety and ASD (see below: Part H. Evaluation and Differential Diagnosis). Comorbid anxiety disorders are reported in 11–84% of youth with ASD (e.g., Sukhodolsky et al. 2007), which is more common than in typically developing youth, youth with conduct disorder, and even some otherwise typical clinically anxious youth.

Natural History, Prognostic Factors, and Outcomes

Individuals with general anxiety tend to report a slow, gradual onset of chronic worry rather than a sudden manifestation of symptoms. Children who have general anxiety have reported that greater impairment is associated with intensity of worries, number of associated symptoms (e.g., restlessness, trouble concentrating), and number of worry domains (e.g., school, family); according to clinicians, greater impairment is associated with intensity of worries, number of associated symptoms, and number of comorbid DSM-IV disorders (Layne et al. 2009). If not treated properly, anxiety disorders in youth predict adult anxiety disorders; more than half of adults with anxiety and mood disorders had a history of a childhood anxiety disorder (Ferdinand and Verhulst 1995; Kessler et al. 1994). Some youth with anxiety disorders may experience fluctuation or a remission of symptoms, but more often, childhood anxiety disorders are chronic and precede anxiety or mood disorders later in life (Keeton et al. 2009). In

terms of vulnerability factors for developing anxiety, evidence suggests that being female, having high trait anxiety, and significant life events predict presence of chronic anxiety in adulthood (de Beurs et al. 2000).

Family liability, temperament, and environmental factors may also contribute to the development of GAD. In a large community sample, GAD in adulthood was associated with parental GAD, high behavioral inhibition, exposure to childhood separation events, and parental overprotection (Beesdo et al. 2010). High emotionality and shy temperament are prominent features in teens with anxiety as well as their parents and siblings (Masi et al. 2003). As well, offspring of anxious parents have a significantly greater chance of developing an anxiety disorder (Micco et al. 2009). Common traits in parents and siblings suggest a shared genetic liability for the development of anxiety disorders. In fact, a meta-analysis of available twin studies estimated a heritability rate of 0.32 for GAD (Hettema et al. 2001).

Similarly, it has been suggested that maternal mood symptoms, such as interpersonal sensitivity, hostility, phobic anxiety, depression, and anxiety, may be related to depression and anxiety in youth with ASD (Mazefsky et al. 2010). High rates of psychopathology in first-degree relatives of individuals with ASD may suggest a common genetic liability to develop ASD and disorders such as anxiety. In fact, it has been proposed that a multigenic form of inheritance (i.e., involving multiple genes) results in a “lesser variant” of ASD in some individuals, manifesting with anxious and obsessive traits rather than the full-blown disorder (Wilcox et al. 2003). The lesser variant has also been referred to as the “broad autism phenotype,” which comprises a constellation of subtle social and communication impairments, in addition to increased rates of psychopathology, in some relatives of individuals with ASD (e.g., Bolte et al. 2007). Higher rates of depression and anxiety in nonautistic relatives of individuals with ASD do not appear to be accounted for by the stress of raising a child (or children) with autism, providing further support for common genetic substrates (Piven and Palmer 1999).

Clinical Expression and Pathophysiology

Clinical features. Individuals with GAD are characterized by excessive fears and worries about a range of issues, such as natural disasters, illness, or the future. Common domains of worry among children include school performance, the health of significant others, minor daily events (e.g., saying the wrong thing), social status, family concerns, and natural phenomena (Layne et al. 2009; Masi et al. 2004). In addition to persistent worry, youth with GAD experience other difficulties including restlessness and trouble relaxing, trouble concentrating and attending, and trouble sleeping (Layne et al. 2009). It is common for youth to seek excessive reassurance, respond negatively to routine criticism and evaluation, have a negative self-image, experience irritability, and have physical complaints (DSM-IV-TR; Masi et al. 2004). Such difficulties can cause significant distress and may interfere with school performance, the development and maintenance of peer relationships, and family functioning (e.g., by leading to conflict).

Relation to gender. In a large prospective community cohort of youth followed from late childhood to late adolescence, girls were more likely to have all types of anxiety than boys (Van Oort et al. 2009). This is consistent with previous findings indicating that GAD is more commonly reported in girls, and the gender difference is present by late childhood. Despite the gender disparity, boys and girls with GAD appear to have similar numbers and types of symptoms (Masi et al. 2004).

Relation to age. Due to the gradual onset of chronic worry, it can sometimes be difficult to determine the exact age when symptoms develop. However, available evidence suggests GAD has an average age of onset of 8.5 years (Masi et al. 2004) and symptoms may increase in middle adolescence (Van Oort et al. 2009). It can be diagnosed as early as symptoms of worry can be verbalized by children or inferred with confidence by parents.

Though core ASD symptoms may improve as youth with ASD enter adolescence (Seltzer et al. 2004), some individuals with ASD experience greater risk for the development of psychopathology at this time. Compared to their typically

developing peers, increased rates of psychopathology, including anxiety and depression, have been reported by adolescents with ASD, as well as their teachers and parents (Hurtig et al. 2009). It may be that increased awareness of their own challenges and the complex social milieu of adolescence exacerbate anxiety in ASD (White et al. 2009).

Relation to cognitive functioning and verbal ability. Although anxiety symptoms are present among individuals with ASD who have a range of cognitive functioning, the type of anxiety may vary depending on cognitive and verbal ability. For example, in higher functioning youth, anxieties can be better articulated and may consist of persistent worrying about performance or that something bad might happen; in youth with less verbal capacity, anxiety may manifest more behaviorally, such as reluctance to change a routine in order to avoid a novel situation. Evidence does suggest that anxiety is more common in higher functioning youth, such as those with Asperger syndrome (e.g., Gadow et al. 2005); however, such findings may reflect better ability to verbally report and express psychiatric symptoms.

Relation to other problems. Pediatric anxiety disorders in typically developing youth predict other childhood sequelae such as substance abuse problems, suicide attempts, and hospitalization (e.g., Kendall et al. 2004). Anxiety disorders have also been associated with significant functional impairment, behavioral problems, and heightened symptomatology in youth with ASD. For example, higher parent-rated anxiety in youth with ASD was associated with greater impairment in social responsiveness (Sukhodolsky et al. 2007) and social skills deficits (Bellini 2004). Several large studies of children with ASD have found strong linkages between high anxiety and increased severity of ASD symptoms such as repetitive behaviors (e.g., Sukhodolsky et al. 2007), sensory symptoms (Ben-Sasson et al. 2008), and total ASD symptoms even when controlling for intellectual impairment, social maladjustment, and degree of speech impairment in a causal model (Kelly et al. 2008). Heightened fears and anxieties in youth with ASD have also been

associated with more externalizing behavioral problems, including conduct, impulsivity, and hyperactivity (Evans et al. 2005). In another study of the pediatric ASD population, high levels of parent-rated anxiety were strongly associated with more loose associations in a performance-based assessment of formal thought disorder (Solomon et al. 2008).

Pathophysiology. In terms of neuropathology, research indicates that the amygdala plays a primary function in the development and expression of anxiety, given its role in processing emotional cues (e.g., Phelps and LeDoux 2005), particularly fearful stimuli. Youth with general anxiety tend to exhibit increased activation in neural fear circuitry, including the amygdala, in response to fearful stimuli (e.g., McClure et al. 2007). Interestingly, abnormalities of the amygdala have also been implicated in the development and maintenance of social impairments (e.g., Baron-Cohen et al. 2000) and abnormal fears and high rates of anxiety in ASD (Amaral and Corbett 2003). In terms of neurobiological vulnerability, the serotonin transporter gene has been implicated in a number of anxiety disorders as well as in ASD.

Evaluation and Differential Diagnosis

Evaluation. As summarized by Keeton et al. (2009), three primary aspects of anxiety are evaluated during diagnostic assessment: anxious behaviors, thoughts, and physical symptoms (Keeton et al. 2009). In terms of behavior, clinicians observe and probe for avoidant behavior and its functional impact on daily activities. It is also important to identify thought patterns related to the child's anxiety. Youth with general anxiety may report more worries, ask more questions (e.g., reassurance seeking), and have more thoughts reflecting concerns about performance. Older children and adolescence may be able to articulate their thoughts and worries, whereas age-appropriate approaches (e.g., use of cartoons/pictures, thought bubbles) may be required to facilitate description of thoughts and feelings in younger children. Physical symptoms, including headaches, gastrointestinal problems, dizziness,

and restlessness, are common among youth with anxiety disorders. It can be helpful for a physician to rule out medical problems before assessing for an anxiety disorder. During the assessment for anxiety, clinicians should review ways in which physical symptoms may interfere with daily functioning, such as sleep, appetite, and school performance. A thorough clinical evaluation would include assessment of other psychiatric concerns as well as family mental health history, and discussion (and perhaps observation) of the child's functioning in different contexts (e.g., school, social, and home).

Anxiety is commonly assessed through clinical interview and rating scales from parents/caregivers, children, and teachers, following a multi-informant, multimethod approach. Semi-structured diagnostic interviews, such as the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS; Ambrosini 2000) and the Anxiety Disorder Interview Schedule (ADIS; Silverman and Albano 1996), have parent and child versions to allow for consensus scoring. Rating scales, such as the Multidimensional Anxiety Scale for Children (MASC), Screen for Child Anxiety Related Emotional Disorders (SCARED), the Revised Child Anxiety and Depression Scale (RCADS), and the Pediatric Anxiety Rating Scale (PARS), also have parent and child versions and correspond to DSM-IV criteria for anxiety disorders.

Because few measures to assess anxiety have been developed and validated for use with individuals with ASD (one exception: the parent-report Childhood Anxiety Sensitivity Index; CASI), clinicians and researchers tend to utilize the same measures as those used in the typical population. Despite the utility of these measures in documenting the presence of anxiety in youth with ASD, it is important to note that symptoms of anxiety may manifest differently in youth with ASD; moreover, dependence on child and parent report to evaluate anxiety can be problematic in ASD. Individuals with ASD are often characterized by diminished ability to think abstractly or communicate effectively through speech or facial expression and often lack awareness of internal states or motivation to report symptoms (Lainhart

and Folstein 1994). Research has shown that children with ASD provide less coherent representations of emotional experiences than their typical peers (Losh and Capps 2006). Children may not verbally express their feelings and worries to parents, and internalizing symptoms are not always easily observed by caretakers (Kuusikko et al. 2008). When evaluating possible anxiety symptoms in youth with ASD, it is important that a skilled clinician, familiar with both ASD and general anxiety, interpret child report, parent report, and observed behaviors with these considerations in mind.

Using a modified version of the Kiddie Schedule for Affective Disorders and Schizophrenia (Geller et al. 1996), Leyfer and colleagues developed and piloted the Autism Comorbidity Interview-Present and Lifetime Version (ACI-PL) (Leyfer et al. 2006). Based on the ACI-PL, 44% of children in the study had specific phobia, which is much higher than rates found in typically developing children and adolescents. Interestingly, generalized anxiety disorder occurred at low rates in the ASD group (2%; Leyfer et al. 2006). Higher rates of anxiety disorders have been reported in other studies (e.g., 84% using the Diagnostic Interview Schedule for Children; Muris et al. 1998), suggesting that different assessment approaches may yield variable rates.

Differential diagnosis. General anxiety rarely occurs in isolation; in a sample of 157 children and adolescents, another psychological disorder was present in 93% of the patients (Masi et al. 2004). GAD can have a similar clinical presentation as other anxiety disorders, including panic disorder, social phobia, separation anxiety disorder (SAD), and obsessive-compulsive disorder (OCD). Some symptoms also overlap with depression (as well as the basic pattern of ruminative thought), and the two disorders are highly comorbid in many samples. Symptoms of anxiety can also appear similar to symptoms and behaviors associated with autism spectrum disorders (e.g., perseverative thought and speech).

Other anxiety disorders: In both typical and ASD populations, it is important to consider how symptoms of general anxiety might overlap with other anxiety disorders. Masi et al. (2004)

reported that comorbid anxiety disorders were present in 75% of the youth in their sample of youth with GAD (Masi et al. 2004). It can be difficult to make the distinction between panic disorder and GAD, especially when individuals with general anxiety experience acute situational or anticipatory anxiety (i.e., heightened anxiety in certain situations); this can be particularly challenging in youth with ASD, who reportedly have more fears related to “specific situations” (Evans et al. 2005). One distinguishing factor is that panic attacks do not generally occur with preceding generalized anxiety (Rickels and Rynn 2001). General anxiety commonly overlaps with *social phobia* as well. Individuals with social phobia exhibit phobic avoidant behavior that is specific to a social situation, whereas someone diagnosed with GAD may have “free-floating” anxiety (i.e., not restricted to particular circumstances) that contributes to feelings of apprehension, causing a person to be shy or uncomfortable in public (Rickels and Rynn 2001). Among youth with GAD, SAD has a high rate of overlap (e.g., 31.8%; Masi et al. 2004) and tends to precede the onset of GAD. Youth who have both SAD and GAD tend to be younger, report more physical symptoms, and have a higher incidence of panic disorder than youth with GAD alone. It can also be difficult to discriminate between OCD and general anxiety, and the two disorders frequently co-occur (see Comer et al. 2004 for review). OCD is characterized by excessive recurrent thoughts (i.e., obsessions) and sometimes accompanied by repetitive and purposeful behavioral attempts to reduce the anxiety (i.e., compulsions; DSM-IV-TR). Such symptoms can be easily confused with GAD when it is unclear whether a child’s recurrent thoughts reflect true, irrational obsessions or more general worrying.

Depression: Anxiety and depression are highly comorbid, and depressive disorders may occur in over half of typical youth with GAD (Masi et al. 2004). The presence of depressive symptoms may cause increased interference with daily activities and more functional impairment and predict worse outcome in individuals with GAD. Evidence also suggests that anxiety disorders may precede and be a risk factor for the

development of depression later in life (Beesdo et al. 2010).

ASD: Symptoms of anxiety and ASD may have similar overt manifestations in some cases, making it difficult to interpret behaviors and determine whether they reflect symptoms of anxiety or ASD. For example, avoidance of unfamiliar situations is a commonly observed feature of ASD and general anxiety even though underlying symptom composition and motivating factors may differ (e.g., lack of interest vs. anxious apprehension). As a second example, many individuals with ASD are sensitive to sensory stimuli such as loud noises; similarly, individuals with general anxiety may have a heightened “startle response” or reaction to sudden loud sounds. Third, persistent, repetitive thought (and associated speech) characterizes both ASD and GAD for many individuals: in the former case, often taking the form of perseverative “special” interests, and in the latter case, worry and catastrophizing. Although the affective content often differs, the inability to shift attention away from specific thoughts is common to both ASD and GAD (Wood and Gadow 2010). It is important to consider the possibility that anxiety symptoms are present and are not accounted for by symptoms expected to occur as part of an ASD. “Diagnostic overshadowing” of psychiatric problems by developmental disabilities (Davis et al. 2008; Reiss 1982) may tend to obscure anxiety disorders in youth with ASD such that clinicians may conceptualize anxiety symptoms as falling under the umbrella of ASD criteria when in fact they represent a coexisting anxiety disorder. In recent years, mounting evidence has suggested that many individuals with ASD do have a coexisting anxiety disorder (e.g., Leyfer et al. 2006); however, more work needs to be done to develop accurate ways of assessing for anxiety in this population.

Treatment

Treatment for general anxiety in children should ideally involve parents as well as the child. Depending on the individual characteristics of the child and the treatments available to families,

children with general anxiety may benefit from behavioral intervention, medication, or a combination of both.

Behavioral treatments (CBT). There is an abundance of evidence indicating that cognitive-behavioral therapy (CBT) is effective (e.g., Walkup et al. 2008); it is considered the treatment of choice for pediatric anxiety. Numerous randomized controlled trials conducted with children have demonstrated that CBT is efficacious and significantly decreases remission rates (e.g., Cartwright-Hatton et al. 2004). Manualized CBT programs, such as the Coping Cat program (Kendall 2000), have been used extensively with anxious youth. The CBT approach assumes that pathological anxiety reflects the interaction between cognitive distortions, somatic or physiological arousal, and avoidance of fearful stimuli. As such, therapists help children identify beliefs and thought patterns that may be irrational and unrealistic and replace them with rational or realistic perspectives (i.e., cognitive restructuring). Other components of CBT usually include psychoeducation with children and parents, problem solving, relaxation training and techniques to manage physiological arousal, modeling, and imaginary or in vivo exposure to increasingly anxiety-provoking stimuli. Intervention should also include strategies to maintain treatment gains and prevent relapse.

Cognitive-behavioral therapy must be tailored to the developmental level of the child, including cognitive capacity (Sauter et al. 2009). This is especially relevant when treating symptoms of anxiety in youth with ASD. Recent evidence suggests modified CBT can effectively target symptoms of anxiety in children with ASD, particularly when a parent component is included (Chalfant et al. 2007; Reaven 2009; White et al. 2009; Wood et al. 2009). For example, Wood’s manualized CBT intervention for youth with comorbid ASD and anxiety (Wood et al. 2009) incorporates parents into each of the weekly treatment sessions; parents then implement strategies at home to facilitate generalization. Symptoms of anxiety are addressed using core components of CBT, such as hierarchical exposure therapy techniques. However, to address developmental level, social

challenges, and other unique characteristics associated with having an ASD, modifications are made to the intervention. For instance, therapeutic concepts are taught using multimodal stimuli (e.g., discussion scaffolded by drawing or writing), and concrete examples are provided rather than abstract descriptions of symptoms and cognitive processes. Children's special interests may be used as metaphors to maintain enthusiasm and motivation. In Wood's manualized intervention, youth and parents are also taught friendship skills, which are practiced at school, in the community (e.g., playgrounds), and on playdates and sleepovers. In addition to the amelioration of anxiety symptoms, improvement in parent-reported core autism symptoms has been reported in children receiving CBT for anxiety compared to children in a waitlist group (Wood et al. 2009). This suggests identification and appropriate treatment of associated symptoms of anxiety can impact core symptoms of autism, thereby improving global functioning.

Pharmacological treatments. Selective serotonin reuptake inhibitors (SSRIs) have been established as the most effective pharmacological approach for children with general anxiety. For example, in a randomized, double-blind comparison of an SSRI (fluvoxamine) and placebo in youth between the ages of 6–17, the SSRI was significantly more effective in reducing anxiety symptoms (Walkup et al. 2001). Nevertheless, there is limited information on the long-term safety of medication use in youth with anxiety, and this remains a continued focus of research. Available evidence does point toward the efficacy of both CBT and treatment with SSRI, with recent findings suggesting that combination of the two approaches provides maximum benefit (Walkup et al. 2008).

Although pharmacological treatments have not demonstrated consistent efficacy in treating core symptoms of ASD, it is possible that medications, including SSRIs, can ameliorate associated psychiatric symptoms (e.g., Kolevzon et al. 2006). The appropriate use of medications that help regulate emotion in ASD might theoretically facilitate a child's capacity to benefit from educational

and behavioral modification interventions. However, clinical trials need to be conducted to determine whether pharmacological approaches effectively target anxiety in this population.

See Also

- [Cognitive Behavioral Therapy \(CBT\)](#)
- [Separation Anxiety Disorder](#)

References and Reading

- Achenbach, T. M., Howell, C. T., McConaughty, S. H., & Stranger, C. (1998). Six-year predictors of problems in a national sample: IV. Young adult signs of disturbance. *Journal of the American Academy of Child and Adolescent Psychiatry*, 37, 718–727.
- Amaral, D. G., & Corbett, B. A. (2003). The amygdala, autism and anxiety. *Novartis Foundation Symposium*, 251, 177–187.
- Ambrosini, P. J. (2000). Historical development and present status of the schedule for affective disorders and schizophrenia for school-age children (K-SADS). *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 49–58.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author. Text Revision.
- Baron-Cohen, S., Ring, H. A., Bullmore, E. T., Wheelwright, S., Ashwin, C., & Williams, S. C. R. (2000). The amygdala theory of autism. *Neuroscience and Biobehavioral Reviews*, 24, 355–364.
- Beesdo, K., Pine, D. S., Lieb, R., & Wittchen, H. U. (2010). Incidence and risk patterns of anxiety and depressive disorders and categorization of generalized anxiety disorder. *Archives of General Psychiatry*, 67, 47–57.
- Bellini, S. (2004). Social skills deficits and anxiety in high-functioning adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 19, 78–86.
- Ben Shalom, D., Mostofsky, S. H., Hazlett, R. L., Goldberg, M. C., Landa, R. J., Faran, Y., et al. (2006). Normal physiological emotions but differences in expression of conscious feelings in children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 36, 395–400.
- Bolte, S., Knecht, S., & Poustka, F. (2007). A case-control study of personality style and psychopathology in parents of subjects with autism. *Journal of Autism and Developmental Disorders*, 37, 243–250.
- Cartwright-Hatton, S., Roberts, C., Chitsabesan, P., Fothergill, C., & Harrington, R. (2004). Systematic review of the efficacy of cognitive behaviour therapies

- for childhood and adolescent anxiety disorders. *The British Journal of Clinical Psychology*, 43, 421–436.
- Chalfant, A. M., Rapee, R., & Carroll, L. (2007). Treating anxiety disorders in children with high functioning autism spectrum disorders: A controlled trial. *Journal of Autism and Developmental Disabilities*, 37, 1842–1857.
- Comer, J. S., Kendall, P. C., Franklin, M. E., Hudson, J. L., & Pimental, S. S. (2004). Obsessing/worrying about the overlap between obsessive-compulsive disorder and generalized anxiety disorder in youth. *Clinical Psychology Review*, 24, 663–683.
- Davis, E., Saeed, S. A., & Antonacci, D. J. (2008). Anxiety disorders in persons with developmental disabilities: Empirically informed diagnosis and treatment. Reviews literature on anxiety disorders in DD population with practical take-home messages for the clinician. *The Psychiatric Quarterly*, 79, 249–263.
- de Beurs, E., Beekman, A. T., Deeg, G. J., Van Dyck, R., & van Tilburg, W. (2000). Predictors of change in anxiety symptoms of older persons: Results from the Longitudinal Study Amsterdam. *Psychological Medicine*, 30, 515–527.
- Evans, D. W., Canavera, K., Kleinpeter, F. L., Maccubbin, E., & Taga, K. (2005). The fears, phobias, and anxieties of children with autism spectrum disorder and down syndrome: comparisons with developmentally and chronologically age matched children. *Child Psychiatry and Human Development*, 36(1), 3–27.
- Ferdinand, R. F., & Verhulst, F. C. (1995). Psychopathology from adolescence into young adulthood: An 8-year follow-up study. *The American Journal of Psychiatry*, 152, 1586–1594.
- Gadow, K. D., DeVincent, C. J., Pomeroy, J., & Azizian, A. (2005). Comparison of DSM-IV symptoms in elementary school-age children with PDD versus clinic and community samples. *Autism*, 9, 392–415.
- Geller, B., Williams, M., Zimmerman, B., & Frazier, J. (1996). *Washington University in St. Louis Kiddie Schedule for Affective Disorders and Schizophrenia (WASH-U-KSADS)*. St. Louis: Washington University.
- Ghaziuddin, M. (2002). Asperger syndrome: Associated psychiatric and medical conditions. *Focus on Autism and Other Developmental Disabilities*, 17, 138–144.
- Hettema, J. M., Neale, M. C., & Kendler, K. S. (2001). A review and meta-analysis of the genetic epidemiology of anxiety disorders. *The American Journal of Psychiatry*, 158, 1568–1578.
- Hill, E., Berthoz, S., & Frith, U. (2004). Brief report: Cognitive processing of own emotions in individuals with autistic spectrum disorder and in their relatives. *Journal of Autism and Developmental Disorders*, 34, 229–235.
- Hurtig, T., Kuusikko, S., Mattila, M. L., Haapsamo, H., Ebeling, H., & Jussila, K. (2009). Multi-informant reports of psychiatric symptoms among high-functioning adolescents with Asperger syndrome or autism. *Autism*, 13, 583–598.
- Keeton, C. P., Kolos, A. C., & Walkup, J. T. (2009). Pediatric generalized anxiety disorder. *Paediatric Drugs*, 11, 171–183.
- Kelly, A. B., Garnett, M. S., Atwood, T., & Peterson, C. (2008). Autism spectrum symptomatology in children: The impact of family and peer relationships. *Journal of Abnormal Child Psychology*, 36, 1069–1081.
- Kendall, P. C. (1994). Treating anxiety disorders in children: Results of a randomized clinical trial. *Journal of Consulting and Clinical Psychology*, 62, 100–110.
- Kendall, P. C. (2000). *Cognitive-behavioral therapy for anxious children: Therapist manual* (2nd ed.). Ardmore: Workbook Publishing.
- Kendall, P. C., Safford, S., Flannery-Schroeder, E., & Webb, A. (2004). Child anxiety treatment: Outcomes in adolescence and impact on substance use and depression at 7.4-year follow-up. *Journal of Consulting and Clinical Psychology*, 72, 276–287.
- Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., et al. (1994). Lifetime and 12-month prevalence of DSM-III psychiatric disorders in the United States. Results from the National Comorbidity Study. *Archives of General Psychiatry*, 51, 8–19.
- Kolevzon, A., Mathewson, K. A., & Hollander, E. (2006). Selective serotonin reuptake inhibitors in autism: A review of efficacy and tolerability. *The Journal of Clinical Psychiatry*, 67, 407–414.
- Kuusikko, S., Pollock-Wurman, R., Jussila, K., Carter, A. S., Mattila, M. L., Ebeling, H., et al. (2008). Social anxiety in high-functioning children and adolescents with autism and Asperger syndrome. *Journal of Autism and Developmental Disorders*, 38, 1697–1709.
- Lainhart, J. E., & Folstein, S. E. (1994). Affective disorders in people with autism: A review of published cases. *Journal of Autism and Developmental Disorders*, 24, 587–601.
- Layne, A. E., Bernat, D. H., Victor, A. M., & Bernstein, G. A. (2009). Generalized anxiety disorder in a non-clinical sample of children: Symptom presentation and predictors of impairment. *Journal of Anxiety Disorders*, 23, 283–289.
- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., Tager-Flusberg, H., & Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36, 849–861.
- Losh, M., & Capps, L. (2006). Understanding of emotional experience in autism: Insights from the personal accounts of high-functioning children with autism. *Developmental Psychology*, 42, 809–818.
- Masi, G., Mucci, M., Favilla, L., Brovedani, P., Millepiedi, S., & Perugi, G. (2003). Temperament in adolescents with anxiety and depressive disorders and in their families. *Child Psychiatry and Human Development*, 33, 245–259.

- Masi, G., Millepiedi, S., Mucci, M., Poli, P., Bertini, N., & Milantoni, L. (2004). Generalized anxiety disorder in referred children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43, 752–760.
- Mazefsky, C. A., Conner, C. M., & Oswald, D. P. (2010). Association between depression and anxiety in high-functioning children with autism spectrum disorders and maternal mood symptoms. *Autism Research*, 3, 120–127.
- McClure, E. B., Monk, C. S., Nelson, E. E., Parrish, J. M., Adler, A., Blair, R. J., et al. (2007). Abnormal attention modulation of fear circuit function in pediatric generalized anxiety disorder. *Archives of General Psychiatry*, 64, 97–106.
- Micco, J. A., Henin, A., Mick, E., Kim, S., Hopkins, C. A., Biederman, J., et al. (2009). Anxiety and depressive disorders in offspring at high risk for anxiety: A meta-analysis. *Journal of Anxiety Disorders*, 23, 1158–1164.
- Muris, P., Steerneman, P., Merckelbach, H., Holdrinet, I., & Meesters, C. (1998). Comorbid anxiety symptoms in children with pervasive developmental disorders. *Journal of Anxiety Disorders*, 12, 397–393.
- Phelps, E. A., & LeDoux, J. E. (2005). Contributions of the amygdala to emotion processing: From animal models to human behavior. *Neuron*, 48, 175–187.
- Piven, J., & Palmer, P. (1999). Psychiatric disorder and the broad autism phenotype: Evidence from a family study of multiple incidence autism families. *American Journal of Psychiatry*, 156, 557–563.
- Reaven, J. (2009). Children with high-functioning autism spectrum disorders and co-occurring anxiety symptoms: Implications for assessment and treatment. *Journal for Specialists in Pediatric Nursing*, 14, 192–199.
- Reiss, S. (1982). Psychopathology and mental retardation: A survey of developmental disabilities mental health problems. *Mental Retardation*, 20, 128–132.
- Rickels, K., & Rynn, M. (2001). Overview and clinical presentation of generalized anxiety disorder. *The Psychiatric Clinics of North America*, 24, 1–17.
- Sauter, F. M., Heyne, D., & Westenberg, P. (2009). Cognitive behavior therapy for anxious adolescents: Developmental influences on treatment design and delivery. *Clinical Child and Family Psychology Review*, 12, 310–335.
- Seltzer, M. M., Shattuck, P., Abbeduto, L., & Greenberg, J. S. (2004). Trajectory of development in adolescents and adults with autism. *Mental Retardation and Developmental Disabilities Research*, 10, 234–247.
- Shaffer, D., Fisher, P., Dulcan, M. K., Davies, M., Piacentini, J., Schwab-Stone, et al. (1996). The NIMH Diagnostic Interview Schedule for Children Version 2.3 (DISC-2.3): Description, acceptability, prevalence rates, and performance on the MECA study. Methods for the Epidemiology of Child and Adolescent Disorders Study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35, 865–877.
- Silverman, W. K., & Albano, A. M. (1996). *The anxiety disorders interview schedule for DSM-IV – Child and parent versions*. San Antonio: Graywind.
- Skokauskas, N., & Gallagher, L. (2010). Psychosis, affective disorders and anxiety in autistic spectrum disorder: Prevalence and nosological considerations. *Psychopathology*, 43, 8–16.
- Solomon, M., Ozonoff, S., Carter, C., & Caplan, R. (2008). Formal thought disorder and the autism spectrum: Relationship with symptoms, executive control, and anxiety. *Journal of Autism and Developmental Disorders*, 38, 1474–1484.
- Sukhodolsky, D. G., Scahill, L., Gadow, K. D., Arnold, L. E., Aman, M. G., McDougle, C. J., et al. (2007). Parent-rated anxiety symptoms in children with pervasive developmental disorders: Frequency and association with core autism symptoms and cognitive functioning. *Journal of Abnormal Child Psychology*, 36, 117–128.
- Van Oort, F. V., Greaves-Lord, K., Verhulst, F. C., Ormel, J., & Huizink, A. C. (2009). The developmental course of anxiety symptoms during adolescence: The TRAILS study. *Journal of Child Psychology and Psychiatry*, 50, 1209–1217.
- Walkup, J. T., Labellarte, M. J., Riddle, M. A., Pine, D. S., Greenhill, L., Klein, R., et al. (2001). Fluvoxamine for the treatment of anxiety disorders in children and adolescents. *The New England Journal of Medicine*, 344, 1279–1285.
- Walkup, J. T., Albano, A. M., Piacentini, J., Birmaher, B., Compton, S. N., Sherrill, J. T., et al. (2008). Cognitive behavioral therapy, sertraline, or a combination in childhood anxiety. *The New England Journal of Medicine*, 359, 2753–2766.
- Whitaker, A., Johnson, J., Shaffer, D., Rapoport, J. L., Kalikow, K., Walsh, B. T., et al. (1990). Uncommon troubles in young people: Prevalence estimates of selected psychiatric disorders in a nonreferred adolescent population. *Archives of General Psychiatry*, 47, 487–496.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29, 216–229.
- Wilcox, J. A., Tsuang, M. T., Schnurr, T., & Baida-Fragoso, N. (2003). Case-control family study of lesser variant traits in autism. *Biological Psychiatry*, 47, 171–177.
- Wood, W. J. J., & Gadow, K. D. (2010). Exploring the nature and function of anxiety in youth with autism spectrum disorders. *Clinical Psychology: Research and Practice*, 17, 281–292.
- Wood, J. J., & McLeod, B. D. (2007). *Child anxiety disorders: A family-based treatment manual for practitioners*. New York: Norton.
- Wood, J. J., Drahota, A., Sze, K., Har, K., Chiu, A., & Langer, D. A. (2009). Cognitive behavioral therapy for anxiety in children with autism spectrum disorders: A randomized, controlled trial. *Journal of Child Psychology and Psychiatry*, 50(3), 224–234.

General Case Analysis

► General Case Programming (GCP)

General Case Model

► General Case Programming (GCP)

General Case Programming (GCP)

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Synonyms

General case analysis; General case model;
Training the general case

Definition

General case programming is an instructional process with the ultimate goal of increasing the likelihood that a learner will generalize a skill set (i.e., target behavior) learned during structured teaching situations to relevant real-world settings and situations in which the target behavior should be demonstrated.

General case programming can be broken into three phases: design, implementation, and assessment. The design phase is key to general case programming and begins with analysis of the target skill to be taught. The instructor must identify the essential behaviors and all variations of those behaviors that are necessary for successful performance of that skill in relevant situations. The instructor must also consider and identify the full range of stimuli that may be encountered when the

learner engages in the target skill in real-world settings. After analysis of the relevant behaviors and stimuli, the instructor can select teaching materials that encompass as many of the behavioral and stimulus variations as possible. To reduce the likelihood of overgeneralization (engaging in the target behavior in inappropriate situations), the instructor must also identify materials that share some, but not all, characteristics with the target skill materials and ensure the learner does not engage in the target skill inappropriately. During the implementation phase, the instructor exposes the learner to the selected materials and teaches the relevant behaviors using teaching strategies appropriate for the learner (see Horner et al. 1982, 1986; Horner and Albin 1988 for recommendations on presentation order of examples). Implementation continues until the learner demonstrates success with at least one set of materials. In the assessment phase, the learner is exposed to untaught examples to test for generalization. If necessary, additional sets of materials can be used during subsequent teaching sessions until the learner shows generalization or is taught the full variety of relevant responses including the full variety of materials.

In sum, general case programming is an instructional process that is designed to increase the efficiency of teaching and subsequent likelihood of generalization of new skills. General case programming is based on thorough analysis of target skills sets and selection of materials that include the full variations in behaviors and materials likely to be encountered in real-world situations.

See Also

► Generalization and Maintenance

References and Reading

- Horner, R. H., & Albin, R. W. (1988). Research on general-case procedures for learners with severe disabilities. *Education and Treatment of Children, 11*, 375–388.
- Horner, R. H., Sprague, J. R., & Wilcox, B. (1982). Constructing general case programs for community

activities. In B. Wilcox & G. T. Bellamy (Eds.), *Design of high school programs for severely handicapped students* (pp. 61–98). Baltimore: Paul H. Brookes.

Horner, R. H., McDonnell, J. J., & Bellamy, G. T. (1986). Teaching generalized behaviors: General case instruction in simulation and community settings. In R. H. Horner, L. H. Meyer, & H. D. B. Fredericks (Eds.), *Education of learners with severe handicaps: Exemplary service strategies* (pp. 289–315). Baltimore: Paul H. Brookes.

General Case Teaching, General Case Instruction

► [Multiple Exemplar Training](#)

General Health Questionnaire (GHQ-12)

► [Autism Family Experience Questionnaire \(AFEQ\)](#), [The](#)

General Health Questionnaire-12 (GHQ-12)

► [Autism Family Experience Questionnaire \(AFEQ\)](#)

Generalization and Maintenance

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Definition

The term generalization, defined most broadly (Stokes and Baer 1977), is used to describe

when skills learned in a training environment transfer to the natural environment after training has ended. Generalization, in its more narrow definition, is a behavioral term that is used to describe the spread of effect of a training procedure to untrained stimuli and responses, as well as the durability of treatment effects over time. Generalization includes three specific forms: Stimulus generalization, response generalization, and maintenance. Stimulus generalization involves the occurrence of a behavior in response to another similar stimulus. For example, a child who learns to say “ball” (behavior) in response to a picture of a ball (trained stimulus) shows stimulus generalization if he says “ball” in response to an actual ball (new stimulus). Response generalization involves the spread of an effect of a trained response to other similar responses. For example, a child who is taught to greet another person by saying “Hi,” also begins to greet people by waving. Maintenance involves the continued use of a trained behavior over time, after the direct intervention period has ended.

An important outcome of any intervention is that its effects generalize to similar stimuli outside of the treatment setting. Without generalization, the ability to use a given skill may be confined to a highly specific environment or stimulus, and thus is not particularly valuable for use in real-world contexts. For individuals with ASD, generalizing skills to new settings, new people, or in response to new events can be especially challenging. In addition, it is important that the effects of intervention lead to generalized responding, such that training in one behavior leads to the development of a variety of related behaviors without direct training; otherwise, training is inefficient and leads to an inflexible pattern of behavioral responses. Further, it is critical that gains made in treatment be maintained over time, after the intervention is complete. Individuals with ASD may show deficits in skill maintenance, in part, because the types and patterns of reinforcement in the real world are often divergent from those employed in treatment settings. As such, for individuals with ASD, newly acquired skills may extinguish rather quickly in extra-therapy contexts.

It has been suggested that without targeted intervention, generalization and maintenance of skill does not occur automatically for individuals with ASD (Koegel and Rincover 1977). Some have suggested that these difficulties may be related to an insistence of sameness, stimulus over-selectivity, and/or lack of motivation in individuals with ASD (Arnold-Saritepe et al. 2009). While the etiology of these deficits remain unclear, it is obvious that without the ability to translate knowledge to various contexts, an individual will continue to experience impairment in important areas of functioning. As such, it is critical that intervention programs incorporate specific components to help encourage behavioral change outside of the direct therapy environment.

Historical Background

Difficulty with treatment generalization and maintenance for children with autism was identified as early as 1973 by Lovaas and colleagues, who noted that children in their study of intensive behavioral intervention who returned to an institutional setting lost the majority of their treatment gains; however, children who returned to live with their parents who had been trained to carry out the intervention at home continued to make gains.

Historically, generalization was conceptualized as a passive process that was a natural consequence of the inability to effectively utilize stimulus discrimination. It was thought that if generalization did not occur, an individual had been successfully taught to discriminate among specific stimuli and responses. Stokes and Baer (1977) were some of the first to argue that generalization is an active process and that it should be directly targeted as a teaching outcome. In this way, “generalization may be claimed when no extratraining manipulations are needed for extratraining changes; or may be claimed when some extra manipulations are necessary, but their cost or extent is clearly less than that of the direct intervention” (Stokes and Baer 1977). In sum, an intervention can be considered effective when relevant targeted behaviors are observed in untrained environments (e.g., across

subjects, settings, people, behaviors, and/or time) without the same schedule of prompting and reinforcement utilized in training conditions (Stokes and Baer 1977).

Building off this idea of generalization as an active process, Stokes and Baer (1977) suggested that treatments should directly target generalization and maintenance by incorporating “generalization-promotion” techniques into intervention protocols. In an initial review of the generalization literature, these authors described several “generalization-promotion” techniques that were being utilized to track and target generalization. These techniques were categorized into nine groups:

1. **Train and Hope:** These procedures document the presence or absence of generalization but do not explicitly target the generalization process.
2. **Sequential Modification:** Such procedures involve the implementation of intervention techniques and the probing for generalization. If generalization is not observed, the procedures are systematically altered to determine and then use the techniques that are most effective for promoting generalization.
3. **Introduce to Natural Maintaining Contingencies:** This process relies on incorporating into the treatment program, circumstances and reinforcement that are natural to the behaviors being taught. In this way, the reinforcement and consequences that are used in treatment are the same, or very similar to, those which are available in real-world contexts.
4. **Train Sufficient Exemplars:** These procedures involve teaching with several different examples of the same lesson until an individual is able to generalize sufficiently across related conditions and responses.
5. **Train Loosely:** This process relies on incorporating variety into the teaching conditions, settings, stimuli, and responses in order to mirror real-world situations.
6. **Use Indiscriminable Contingencies:** This process depends on the use of intermittent reinforcement, such that situations in which reinforcement would be expected are

undistinguishable from those in which reinforcement would not be expected. In this way, target behaviors should eventually occur across contexts, regardless of whether or not reinforcement is available.

7. **Program Common Stimuli:** Such procedures involve making the treatment context similar to the generalization context. Thus, the settings and stimuli utilized in training are the same or similar to those available to an individual in real-world contexts.
8. **Mediate Generalization:** This process relies on the incorporation of a particular stimulus (e.g., an object or a person) that is present in the training conditions and generalization conditions. In this way, access to the mediating stimulus during generalization will prompt the individual to use the behaviors learned in the training condition.
9. **Train to Generalize:** These procedures involve giving an individual direct instruction to generalize, or reinforcing generalization as if it were a specific behavior.

Current Knowledge

Although considerable individual variation exists with regards to the development of generalization and maintenance of skill, there is a significant body of research to indicate that certain factors can be particularly beneficial in supporting these processes. Many of these factors are based on the incorporation of the techniques described by Stokes and Baer (1977) into intervention programs. Currently, the literature indicates that implementing intervention in natural settings, using parents as intervention providers, utilizing peer-mediated intervention, and self-management are especially effective in supporting the development of skill generalization and maintenance.

Naturalistic Interventions: Some of the first effective intervention programs developed for individuals with ASD utilized a traditional behavioral approach, discrete trial training, which relies on a highly structured and controlled learning environment. In such programs,

the therapist and child usually sit face-to-face at a table with the therapist prompting the child for a series of discrete skills presented in successive trials. Child behavior is typically reinforced with food or access to a preferred toy. Discrete trial training has been shown to be effective for teaching a range of skills (e.g., Lovaas et al. 1973). However, results of these studies suggested limited generalization of skill, especially in non-treating settings, with nontreatment therapists, and in the spontaneous use of the new skill. In fact, subsequent research has demonstrated that highly structured intervention environments and artificial reinforcers can limit the generalization and maintenance of newly acquired skill (Lovaas et al. 1973; Spradlin and Siegel 1982). As such, programs utilizing behavioral techniques directly in natural contexts have emerged in order to actively promote generalization during intervention. Naturalistic behavioral interventions use prompting and reinforcement but are implemented in natural settings (e.g., during play interactions or other daily routines) and utilize more natural reinforcement (e.g., praise or the natural consequence of behavior). Examples of such programs include pivotal response training (PRT), incidental teaching, and milieu teaching. A significant amount of research has demonstrated that naturalistic behavioral techniques are effective at increasing verbal and nonverbal communication, play, imitation, and joint attention (Schreibman et al. 2015). Importantly, research has also demonstrated that the use of these types of intervention techniques promote generalization of skill to untrained stimuli, settings, and interaction partners (e.g., Kaiser and Hester 1994). Several studies have compared naturalistic and traditional behavioral approaches for teaching specific language skills (e.g., preposition use, color adjectives) as well as broader language functioning. Across studies, naturalistic behavioral approaches have been found to be superior to traditional behavioral approaches in terms of producing skill generalization (Delprato 2001; Mohammadzahari et al. 2014). As such, the literature indicates that the implementation of intervention in natural settings with natural reinforcement seems to be an

especially effective way to increase the generalization and maintenance of skill for individuals with ASD.

Parents as Intervention Providers: A variation of implementing intervention in natural contexts is to train parents as intervention providers. By training parents in intervention techniques, children are provided with increased access to intervention in more of their natural settings with their natural interaction partners. A growing body of literature suggests that parents can be successfully trained in a variety of intervention approaches to improve the quality of parent-child interactions, to produce gains in language skills, imitation skills, joint attention/joint engagement, and appropriate play skills, and to decrease problem behavior in children with ASD (e.g., Bearss et al. 2015; Green et al. 2010; Kasari et al. 2010; Wetherby et al. 2014). Moreover, generalization and maintenance of skill was observed in the majority of these studies; the children were able to utilize variations of newly acquired skill in untrained environments, with untrained interaction partners, and in response to untrained stimuli. These results indicate that using parents as agents of change can lead to better generalization and maintenance of skill for individuals with ASD.

Peer-Mediated Intervention: Another way to promote generalization and maintenance in individuals with ASD is to utilize peers in intervention programs. In this way, individuals with ASD can receive training in the classroom, the playground, and during daily routine interactions with same-age individuals. A number of studies have shown that typically developing children can be successfully trained to directly implement, or support the implementation of, naturalistic intervention techniques to elicit changes in behavior from peers with ASD (e.g., Kasari et al. 2012; Kamps et al. 2015). Research has demonstrated that the utilization of multiple peer trainers can help promote generalization of skill, particularly social-communication skills, to untrained peers (Pierce and Schreibman 1997). Moreover, additional research has suggested that peer-mediated naturalistic interventions can enhance generalization of skill to untrained environments, untrained

stimuli, and untrained interaction partners, including siblings (e.g., Belchic and Harris 1994; Chang and Locke 2016).

Self-Management: Self-management, in which the individual is taught to monitor his own behavior and to provide self-reinforcement, has also been found to be successful for producing and maintaining improvements in appropriate social interaction and play skills in individuals with autism (Carr et al. 2014). With self-management, the individual's behavior becomes under their own control, and thus the presence of a treatment provider is not necessary. This helps individuals generalize skills to new environments and maintain behavioral improvements over time in the absence of treatment providers.

In order to utilize the techniques described above in an effective and ecologically valid way, one must first consider the individual receiving instruction. Different stimuli, responses, settings, and consequences are going to be relevant and reinforcing for a given individual. In order to implement intervention to promote generalization and maintenance of skill, a thorough understanding of the individual and his or her needs is imperative.

Future Directions

Although it has been identified that individuals with ASD experience difficulties with generalization and maintenance and that some teaching techniques are more effective than others for promoting these processes, the research on the application of these techniques continues to be limited. In a 2009 review of the intervention literature, Arnold-Saritepe and colleagues found that more than 40% of entries did not evaluate or discuss generalization and maintenance. Thus, as a field, our understanding of the most effective ways to encourage generalization and maintenance is still somewhat restricted. It is important for researchers and clinicians to continue to utilize, explore, and expand upon the techniques described above in order to actively target generalization and maintenance of skills in individuals with ASD.

See Also

- [Parent Training](#)
- [Peer-Mediated Intervention](#)

References and Reading

- Arnold-Saritepe, A. M., Phillips, K. J., Mudford, O. C., De Rozario, K. A., & Taylor, S. A. (2009). Generalization and maintenance. In J. L. Matson (Ed.), *Applied behavior analysis for children with autism spectrum disorders*. New York: Springer.
- Bearss, K., Johnson, C., Smith, T., Lecavalier, L., Swiezy, N., Aman, M., . . . & Sukhodolsky, D. G. (2015). Effect of parent training vs parent education on behavioral problems in children with autism spectrum disorder: A randomized clinical trial. *JAMA*, *313*, 1524–1533.
- Belchic, J. K., & Harris, S. L. (1994). The use of multiple peer exemplars to enhance the generalization of play skills to the siblings of children with autism. *Child & Family Behavior Therapy*, *16*, 1–25.
- Carr, M. E., Moore, D. W., & Anderson, A. (2014). Self-management interventions on students with autism: A meta-analysis of single-subject research. *Exceptional Children*, *81*, 28–44.
- Chang, Y. C., & Locke, J. (2016). A systematic review of peer-mediated interventions for children with autism spectrum disorder. *Research in Autism Spectrum Disorders*, *27*, 1–10.
- Delprato, D. J. (2001). Comparisons of discrete-trial and normalized behavioral intervention for young children with autism. *Journal of Autism and Developmental Disorders*, *31*, 315–325.
- Green, J., Charman, T., McConachie, H., Aldred, C., Slonims, V., Howlin, P., . . . & Barrett, B. (2010). Parent-mediated communication-focused treatment in children with autism (PACT): A randomised controlled trial. *The Lancet*, *375*, 2152–2160.
- Kaiser, A. P., & Hester, P. P. (1994). Generalized effects of enhanced milieu teaching. *Journal of Speech and Hearing Research*, *37*, 1320–1340.
- Kamps, D., Thiemann-Bourque, K., Heitzman-Powell, L., Schwartz, I., Rosenberg, N., Mason, R., & Cox, S. (2015). A comprehensive peer network intervention to improve social communication of children with autism spectrum disorders: A randomized trial in kindergarten and first grade. *Journal of Autism and Developmental Disorders*, *45*, 1809–1824.
- Kasari, C., Gulsrud, A. C., Wong, C., Kwon, S., & Locke, J. (2010). Randomized controlled caregiver mediated joint engagement intervention for toddlers with autism. *Journal of Autism and Developmental Disorders*, *40*, 1045–1056.
- Kasari, C., Rotheram-Fuller, E., Locke, J., & Gulsrud, A. (2012). Making the connection: Randomized controlled trial of social skills at school for children with autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, *53*, 431–439.
- Koegel, R. L., & Rincover, A. (1977). Research on the difference between generalization and maintenance in extra-therapy responding. *Journal of Applied Behavior Analysis*, *10*, 1–12.
- Lovaas, O. I., Koegel, R., Simmons, J. Q., & Long, J. S. (1973). Some generalization and follow up measures on autistic children in behavior therapy. *Journal of Applied Behavior Analysis*, *6*, 131–166.
- Mohammadzahari, F., Koegel, L. K., Rezaee, M., & Rafiee, S. M. (2014). A randomized clinical trial comparison between pivotal response treatment (PRT) and structured applied behavior analysis (ABA) intervention for children with autism. *Journal of Autism and Developmental Disorders*, *44*, 2769–2777.
- Pierce, K., & Schreibman, L. (1997). Multiple peer use of pivotal response training to increase social behaviors of classmates with autism: Results from trained and untrained peers. *Journal of Applied Behavior Analysis*, *30*, 157–160.
- Schreibman, L., Dawson, G., Stahmer, A. C., Landa, R., Rogers, S. J., McGee, G. G., . . . & Halliday, A. (2015). Naturalistic developmental behavioral interventions: Empirically validated treatments for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, *45*, 2411–2428.
- Spradlin, J. E., & Siegel, G. M. (1982). Language training in natural and clinical environments. *Journal of Speech and Hearing Disorders*, *47*, 2–6.
- Stokes, T. F., & Baer, D. M. (1977). An implicit technology of generalization. *Journal of Applied Behavior Analysis*, *10*, 349–367.
- Wetherby, A. M., Guthrie, W., Woods, J., Schatschneider, C., Holland, R. D., Morgan, L., & Lord, C. (2014). Parent-implemented social intervention for toddlers with autism: An RCT. *Pediatrics*, *134*, 1084–1093.

Generalized Anxiety Disorder

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Synonyms

GAD

Short Description or Definition

Generalized Anxiety Disorder does not go under any other denomination.

The DSM-IV-TR includes Overanxious Disorder of Childhood (DSM-III-R) in this category.

Generalized Anxiety Disorder (GAD) is characterized by the presence of *excessive and uncontrollable worries* about a number of life events such as weather, illnesses, natural disasters, risky situations, failure, or the future. These worries are accompanied by at least three of the six possible associated symptoms of negative affect/tension: restlessness, feeling keyed up, or on edge; easily fatigued; difficulty concentrating, mind going blank; irritability; muscle tension; and sleep disturbance. Because usually one or more situations provoke these symptoms, the patient tends to avoid them. *Avoidance behavior* is a cardinal symptom of all anxiety disorders, including GAD (Table 1).

Generalized Anxiety Disorder, Table 1 Diagnostic criteria for DSM-IV generalized anxiety disorder

(A) Excessive anxiety and worry (apprehensive expectation), occurring more days than not, for at least 6 months, about a <i>number of events</i> or activities (such as work or school performance)
(B) The person finds it <i>difficult to control</i> the worry
(C) The anxiety and worry are associated with <i>three (or more)</i> of the following six symptoms (with at least some symptoms present for more days than not, for the past 6 months). Note: <i>Only one item is required in children</i>
(1) Restlessness, or feeling keyed up or on edge
(2) Being easily fatigued
(3) Difficulty concentrating or mind going blank
(4) Irritability
(5) Muscle tension
(6) Sleep disturbance (difficulty falling or staying asleep, or restless unsatisfying sleep)
(D) The <i>focus</i> of the anxiety and worry is <i>not confined to features of an Axis I disorder</i> , for example, the anxiety or worry is <i>not</i> about: having a panic attack (as in panic disorder), being embarrassed in public (as in social phobia), being contaminated (as in obsessive-compulsive disorder), being away from home or close relatives (as in separation anxiety disorder), gaining weight (as in anorexia nervosa), having multiple physical complaints (as in somatization disorder), or having a serious illness (as in hypochondriasis), and the anxiety and worry do not occur exclusively during posttraumatic stress disorder
(E) The anxiety, worry, or physical symptoms cause <i>clinically significant distress or impairment</i> in social, occupational, or other important areas of functioning
(F) The disturbance is <i>not due</i> to the direct physiological effects of a <i>substance</i> (e.g., a drug of abuse, a medication) or a <i>general medical condition</i> (e.g., hyperthyroidism), and does not occur exclusively during a mood disorder, psychotic disorder, or a pervasive developmental disorder

Due to their *multiple and uncontrollable worries*, the patient has a constant level of arousal and a sense of impending doom but does not develop panic attacks. Patients with GAD frequently emphasize the negative aspects, have unrealistic expectancies about the future, ask too many questions, feel insecure about their competencies, seek constant reassurance, and respond negatively to ordinary criticism and evaluation (Keeton et al. 2009).

Physical symptoms (or somatic complaints) are common in patients with GAD: nausea, abdominal pain, dizziness, headache, tachycardia, sweating, etc. Many patients have previously visited other medical specialties to rule out medical problems.

All these symptoms often interfere, sometimes severely, in the patient’s sleep, concentration, and eating habits; and they can alter his/her capacity for normal relationships (with peers and family). To meet diagnostic criteria, *symptoms must provoke marked distress and interfere with social, emotional, or educational functioning* (American Psychiatric Association [APA] 2000).

GAD and Autism

Currently, anxiety is not considered a phenomenological characteristic of Autism spectrum disorders (ASD; White et al. 2009). It is logical to think that the social disability associated with ASD could provoke anxiety – especially in higher functioning youth who are conscious of their deficits (White et al. 2009). In children with ASD the rate of anxiety disorders range from 18% (Gadow et al. 2004) to 87% (Muris et al. 1998). deBruin et al. (2006) found that approximately 55% of the sample of ASD had at least one anxiety disorder. Simonoff et al. (2008) reported an overall anxiety disorder diagnosis rate of almost 42%. Gjevik et al. (2011) found that 41% of the 71 children and adolescents with ASD had an anxiety disorder. These studies suggest that anxiety disorders are more frequent in patients with ASD than in the general population.

However frequent, these are rarely diagnosed with ASD due to a clinical consensus that they are better explained by the ASD itself (White et al. 2009). Given the substantial symptom overlap and the lack of clarity in differential diagnosis, this topic remains controversial.



Some of the most frequently reported anxiety disorders and symptoms seen in children with ASD are simple phobias, generalized anxiety disorder, separation anxiety disorder, obsessive-compulsive disorder (OCD), and social phobia.

There is some evidence that the prevalence of anxiety may differ across the specific diagnoses. Children with Asperger Syndrome appear most likely to experience anxiety, followed by PDD-NOS, and then Autism Disorder. These group differences must be interpreted cautiously given that diagnosis is often related to other factors such as level of cognitive functioning, which may also impact psychiatric comorbidity (White et al. 2009).

Nonetheless, identification is difficult due to the patient's concurrent difficulties with communication, the presence of other behavior problems, the lack of standardized assessments specific to these patients, and the need for greater collateral sources of assessment information (Davis et al. 2008). This explains why there is limited systematic study of the diagnosis and treatment of anxiety disorders in patients with ASD.

Categorization

GAD is not classified into categories.

Epidemiology

Lifetime prevalence of GAD in the general population ranges from 2% to 15%. GAD occurs in up to 10% of the pediatric population, most of all adolescents. Childhood-onset GAD has an average age of onset of 8.5 years. There is a 2:1 female-to-male preponderance, at all ages. Boys and girls of all ages with GAD have similar number and types of symptoms (Keeton et al. 2009).

Natural History, Prognostic Factors, and Outcomes

DSM-III was the first to include the category of GAD (APA 1980), and it did so as a residual category. The diagnosis was permitted only if

the patient did not meet criteria for another Axis I disorder. The diagnostic criteria for GAD were revised substantially in DSM-III-R (APA 1987) and then again in DSM-IV (APA 1994).

Although GAD can start in childhood or adolescence, most frequently GAD begins in early adulthood. A large proportion of patients with GAD cannot report a specific moment of onset.

Typically a person starts with subclinical symptoms, and these worsen progressively. There are no consistent differences in the clinical presentation between early onset and late onset GAD. Once it is established, GAD tends to have a chronic and persistent course. Exceptionally patients can have periods of full remission, but usually only have periods of improvement and others of worsening. Fluctuations are common, mainly related to life stressors.

If left untreated GAD may persist, and even lead to another psychiatric disorder such as another type of anxiety disorder, a depressive disorder or a substance use disorder, in adolescence or adulthood. Treatment reduces the probability of persistence and of comorbidity, thus improving prognosis. The initial severity of symptoms is a negative prognostic factor. Another prognostic factor is the parents' reaction to the child's fears. Overprotective parents usually try to diminish the child's distress by letting him avoid a stress-provoking situation, but this can, in the long term, increase the child's fear and avoidance, and prevent the development of coping skills (Keeton et al. 2009).

Impairment/Outcome

GAD is associated with significant physical and psychological symptoms and impacts health, family relationships, academic performance, and employment.

Clinical Expression and Pathophysiology

The clinical features of GAD were already reviewed.

Approximately 90% of patients with GAD have at least one other psychiatric disorder *at some point of time in their lives* (Wittchen et al. 1994).

The National Comorbidity Survey (NCS) estimated that 65% of people with current GAD had at least one other psychiatric disorder *at the time of assessment*, mainly another anxiety disorder or mood disorder (Brawman-Mintzer et al. 1993).

In children, GAD is often diagnosed with another anxiety disorder (social phobia or separation anxiety disorder), depression or ADHD. In children with GAD, co-occurrence with social phobia or separation anxiety disorder is the rule, rather than the exception. Up to 60% of *anxious children* have at least two of the three, and 30% have the three of them (Keeton et al. 2009).

Pathophysiology

The etiology of anxiety is still under study, but *biological (including genetic) and psychosocial (environmental) factors* are involved. These factors cause anxiety by modulating the following:

1. The neural circuits underlying emotion process
2. Psychological processes
3. Behavioral tendencies, including temperament

In relation to genetics, heritability of GAD is around 20–30%. Genes that increase the risk for anxiety disorders, also increase the risk for depressive disorders.

Environmental factors, such as the death of a loved one, lack of family attachment, or conflicts with peers, exert the greatest influence in the emergence of anxiety disorders. An anxiety disorder can start with external cues, but also with how the person processes them.

Moreover, genetic and environmental factors can interact to cause an anxiety disorder in a child. For example, a child can inherit an anxiety disorder from a parent. On top of that, an anxious parent may also have distinctive parenting practices, and encourage a child to maladaptive patterns of responding to ambiguous situations, such as avoiding them (Keeley and Storch 2009).

In relation to neural circuits, fear is regulated mainly by *the prefrontal cortex (PFC) and the amygdala*.

Within the PFC there are two areas involved in anxiety: The *orbitofrontal cortex* makes a representation of negative and positive reinforcers. The

anterior cingulate cortex regulates the emotional response.

The amygdala registers the emotional significance of environmental stimuli and stores emotional memories.

When circuits connecting the PFC and the amygdala are altered, the amygdala is often activated by neutral harmless stimuli. When activated, the person perceives a stimulus as dangerous; and the amygdala starts a cascade reaction, activating other brain regions, leading to the following anxiety symptoms:

- The *HPA axis*, resulting in CNS activation. The hypothalamus secretes corticotrophin releasing factor (CRF) that induces the secretion of ACTH, which induces the secretion of cortisol and adrenaline from the adrenal gland. This causes hyperglycemia and tachycardia, so that the brain and muscles respond to danger.
- The dorsomedial nucleus of the *vagus nerve* and *nucleus ambiguus*, activating the parasympathetic nervous system.
- The *parabrachial nuclei* that increases respiratory frequency, resulting in dyspnea and hyperventilation.
- The *locus ceruleus* that also releases adrenaline, which raises blood pressure and heart rate, activates sweating, and induces tremor (Revised in Soutullo and Figueroa-Quintana 2010).

Among temperamental traits, *behavioral inhibition* is the most studied.

The neurotransmitters most implicated in anxiety are serotonin and *noradrenaline*. Experts believe that anxiety appears when there is an underactivation of the serotonergic system and an overactivation of the noradrenergic system. Disruption of the gamma-aminobutyric acid (GABA) system has also been implicated. The role of corticosteroid regulation in anxiety is presently under study.

Evaluation and Differential Diagnosis

Diagnostic Evaluation

Assessment requires a multi-informant, multi-method approach involving the child, the parents,

and schoolteachers. The clinician should perform a structured or a semi-structured clinical interview. Several semi-structured diagnostic interviews are available.

For children, the *Anxiety Disorder Interview Schedule for DSM-IV (ADIS-IV)* is designed for youth aged 6–17. It assesses DSM-IV anxiety, mood, externalizing, tic, substance use, and pervasive developmental disorders.

The *Kiddie Schedule of Affective Disorders and Schizophrenia for School age children, Present and Lifetime version (K-SADS-PL)* is used for children 6–18 years old, to assess all Axis I diagnosis, except pervasive developmental disorders.

Using these interviews, the clinician should assess the three primary groups of anxiety symptoms: *behaviors, thoughts, and physical symptoms*. The clinician should explicitly ask about their presence, their frequency and how they interfere with their daily functioning. Anxious children tend to report more physical symptoms, while their parents usually emphasize the avoidant behaviors.

The evaluation should also include past psychiatric history, family psychiatric history, and medical history. If possible, it is helpful that the clinician actually sees the patient in an anxiety-provoking natural situation (i.e., school).

Rating scales completed by the patient, caregivers, and teachers should never be used as diagnostic instruments. However, they provide valuable information for the diagnosis and the monitoring of symptoms. The clinician can use several scales, none of which are specific for GAD (Keeton et al. 2009).

The Multidimensional Anxiety Scale for Children (*MASC*) is a standardized validated 39-item self-report measure for youth aged 8–19 years. It yields four factors that assemble DSM-IV anxiety diagnoses: social anxiety, separation anxiety/panic, physical symptoms, and harm avoidance. It effectively discriminates between controls and youth with anxiety and depression. However, it does not discriminate among the different types of anxiety disorders.

The *SCARED* is a 41-item measure for youth aged 9–18 years, with factors matching DSM-IV

anxiety disorders, plus school phobia, such as somatic/panic, generalized anxiety, separation anxiety, and social phobia. It effectively differentiates between anxiety and non-anxiety, anxiety and depression, and anxiety and disruptive disorders. Brief versions of the *SCARED* are available for screening and can be helpful in primary care settings. The *SCARED-R* is a revised 66-item version that includes items for separation anxiety and specific phobias, obsessive-compulsive disorder and post-traumatic stress disorder.

Other scales that can be used in children are the Spence Children's Anxiety Scale (SCAS) and the Pediatric Anxiety Rating Scale (PARS). The Hamilton Anxiety Scale can be used in adults.

Differential Diagnosis

- Normal anxiety: in a stressful situation
- Organic cause: hyperthyroidism, Cushing syndrome, etc.
- Drug use: symptoms appear only when under the effect of the drug

GAD should also be differentiated from other psychiatric disorders as follows:

- Other anxiety disorders
- Obsessive-compulsive disorder
- Depression

Treatment

There are multiple treatment options for GAD. Treatment selection depends on factors related to *the disorder* (severity, duration, dysfunction), *the patient and his/her family* (developmental age, insight, treatment preferences, family motivation and availability, and financial resources), and the clinician (availability and experience).

The *therapeutic alliance* is an essential element, and is best developed in the context of *psychoeducation*, which is also fundamental in the treatment process. Educating the family and

the child (according to his or her developmental age) increases the insight and motivation for treatment. Psychoeducation should always include the concept of anxiety as a normal emotion, etiological and maintenance factors, the natural course of the disorder, treatment alternatives, and prognosis. It is also helpful to inform them on how to manage some symptoms such as avoidance behaviors or cognitive biases.

Two treatments, cognitive-behavioral therapy (CBT) and selective serotonin reuptake inhibitors (SSRIs), have demonstrated efficacy for the treatment of pediatric anxiety disorders including GAD (Keeton et al. 2009).

CBT

Many randomized clinical trials have demonstrated short-term and long-term effectiveness of CBT. Thus, CBT has become the initial treatment of choice, except when anxiety symptoms are too severe to work with the child (medication would be indicated in this case). CBT can be given in monotherapy if symptoms are only mild. Moreover, parents often perceive CBT as a more acceptable initial treatment, compared to medication. The targets of CBT are gaining consciousness about the symptoms, controlling worries, changing the persistent over-arousal state, and confronting fears without permitting avoidance.

There are several CBT-manualized programs, which are based on classical and operant principles, and social learning. CBT is composed of psychoeducation, cognitive restructuring (like reducing negative self-talk, addressing negative thoughts), problem solving, relaxation training (addressing physical symptoms), modeling, contingency management, exposure, and relapse prevention. The duration of the treatment depends on the disorder severity and treatment response. Because of the nature of the worries that characterize GAD, patients may need greater use of imaginary than in vivo exposures.

Medications

There are a number of placebo-controlled studies assessing short-term effectiveness of different

medications in children and adolescents with GAD. There is none studying long-term effectiveness.

SSRIs

SSRIs are the first-line option. In fact, SSRIs tend to be more effective in anxiety disorders than in OCD or depression (Bridge et al. 2007). Sertraline, fluvoxamine, and fluoxetine have US FDA pediatric indications for obsessive-compulsive disorder (OCD), and fluoxetine for pediatric major depression. However, there is sufficient evidence suggesting that these and other SSRIs (such as citalopram and escitalopram) are effective for GAD and other anxiety disorders, in children and adults. There is not a universal algorithm for choosing among the different SSRI, neither in adults nor children.

The clinician should start with a low dose of an approved SSRI and titrate it weekly, assessing clinical response and side effects. Dosing can begin with 25 mg/day of fluvoxamine, 10 mg/day of fluoxetine, or 25 mg/day of sertraline, or even lower in young children. Most SSRIs can be dosed daily in the morning. Evening dosing is possible if treatment does not disrupt sleep.

However, even when a patient significantly improves by weeks 8–12, he or she frequently remains symptomatic and dysfunctional. If the patient presents only partial response, the dose should be increased. To achieve maximum benefit children may need the same dose as adults.

Clinical benefit can be observed during the first week, but it is usually in the second to fourth week that the patient refers improvement. Maximum benefit can occur over 12–16 weeks, with additional benefit accruing over 6–12 months (additive long-term effect).

This, along with the fact that anxiety disorders tend to be chronic, suggest that the treatment should be maintained for an extended period. That way, medication can help consolidate initial gains (continuing exposure to anxiety-provoking situations in an anxiety-free state); help the patient to fully benefit from CBT and work on self-concept, avoidance, social relations, etc.; and prevent relapse. In the absence of evidence, experts

recommend continuing treatment at least *1 year after achieving full remission*.

Deciding when to stop treatment may be difficult. It is not advisable to discontinue medication at the start of school, while at camp, during final exams, or even during vacations. A discontinuation trial is more appropriate when the patient can be monitored closely (to immediately assess relapse), and is in a stable environment at school and at home. As with all medications, the dose of the SSRI should be tapered progressively, avoiding abrupt discontinuation to prevent withdrawal effects. If symptoms return, medication should be restarted.

During the treatment with an SSRI, if the patient does not show clear benefit by weeks 6–8, another SSRI should be tried.

Patients usually tolerate SSRIs well. Common side effects include drowsiness, stomach aches, headaches, restlessness, and behavioral activation. These are generally mild and usually dissipate after the first weeks of treatment. If these are marked or persistent the clinician should lower the dose. If they persist, the clinician should discontinue the treatment and try another SSRI.

Sometimes, improvement could be difficult to differentiate from behavioral activation, for example, when the child becomes less fearful and starts confronting previously feared situations, and even testing the limits of adults. Apathy is a less-known, late-onset side effect; clinicians often miss it as a side effect, and classify it as a symptom, which has treatment implications. Clinicians have informed anecdotal cases of new-onset easy bleeding or bruising. In most cases, coagulation tests and bleeding times are either delayed or normal.

Rarely, a patient may suffer a manic switch or episode, characterized by changes in mood (elevated, euphoric, or irritable) or behavior (grandiosity, fast speech, higher level of energy and activity), and possible psychotic symptoms. Should it appear, this usually starts later in the course of treatment, and is less likely to remit when lowering the dose. It may diminish when discontinuing the treatment, or may require starting a mood stabilizer or an

antipsychotic. Children 10–14 years of age are considered at greatest risk for this severe side effect.

Potential risks should always be considered in the context of potential benefits. With SSRIs for the treatment of anxiety disorders, the benefits clearly outweigh the risks, but systematic tracking of side effects is mandatory. Monitoring height, weight, and physical status is also prudent. There is no need to routinely run laboratory testing or ECG at baseline or during follow-up, if the patient is asymptomatic. Also, ongoing medical symptoms should be monitored by the pediatrician or the general practitioner.

A recently published comparative treatment trial examined the effectiveness of sertraline, CBT, combination therapy with sertraline and CBT, and placebo for 488 youth aged 7–17 years, who presented GAD, Separation anxiety disorder, and/or social phobia. Response rates were based on the Clinical Global Impressions-Improvement (CGI-I) following 12 weeks of treatment. All therapies were statistically superior to placebo, and combination therapy was superior to both monotherapies. Response rates were up to 80.7% for combination therapy, 59.7% for CBT, and 54.9% for sertraline. This was the first study to examine the combined effects of CBT and SSRI for children and adolescents with anxiety disorders, and confirmed that CBT alone and sertraline alone are effective short-term treatments, and that there is a clear clinical advantage in combining both (Walkup et al. 2008).

Second-Line Medications

Venlafaxine and Duloxetine

There are some studies suggesting that noradrenaline and serotonin inhibitors, such as venlafaxine and duloxetine, may be effective in GAD and other anxiety disorders.

Tricyclic Antidepressants

Tricyclic antidepressants have demonstrated efficacy in anxiety disorders. However, they are second-line treatments because they require

pre- and post-ECG controls, have more side effects than SSRIs, and may be lethal in overdose.

Benzodiazepines

Benzodiazepines (BDZ) are frequently used in adults with GAD. Their use is more limited in children and adolescents as they have not been extensively studied in this population. Their main disadvantage is their potential for physical dependence.

Atypical Antipsychotics

Atypical antipsychotics have been studied as adjunct therapy to standard GAD pharmacotherapy in refractory patients (Bloch et al. 2006).

Other Medications

There is little evidence supporting the use of *antihistamines*, *alpha-adrenoceptor agonists*, or *melatonin*, for the treatment of anxiety disorders. However, in practice, clinicians may prescribe these if the patient presents insomnia. *Bupirone* has shown controversial results in adults, and one study could not demonstrate its effectiveness in children.

See Also

- ▶ [Anxiety](#)
- ▶ [Cognitive Behavioral Therapy \(CBT\)](#)
- ▶ [Selective Serotonin Reuptake Inhibitors \(SSRIs\)](#)

References and Reading

- Brawman-Mintzer, O., Lydiard, R. B., Emmanuel, N., Payeur, R., Johnson, M., Roberts, J., Jarrell, M. P., & Ballenger, J. C. (1993). Psychiatric comorbidity in patients with generalized anxiety disorder. *American Journal of Psychiatry*, 150(8), 1216–1218.
- Brown, T. A., O’Leary, T. A., & Barlow, D. H. (2001). Generalized anxiety disorder. In D. H. Barlow (Ed.), *Clinical handbook of psychological disorders: A step-by-step treatment manual* (3rd ed.). New York: Guilford.
- Davis, E., Saeed, S. A., & Antonacci, D. J. (2008). Anxiety disorders in persons with developmental disabilities: Empirically informed diagnosis and treatment. Reviews literature on anxiety disorders in DD population with practical take-home messages for the clinician. *Psychiatric Quarterly*, 79(3), 249–263.
- Egger, H. L., & Angold, A. (2006). Common emotional and behavioral disorders in preschool children: Presentation, nosology, and epidemiology. *Journal of Child Psychology and Psychiatry*, 47(3–4), 313–337.
- Gadow, K. D., DeVincent, C. J., Pomeroy, J., & Azizian, A. (2004). Psychiatric symptoms in preschool children with PDD and clinic and comparison samples. *Journal of Autism and Developmental Disorders*, 34(4), 379–393.
- Gjevik, E., Eldevik, S., Fjæran-Granum, T., & Sponheim, E. (2011). Kiddie-SADS reveals high rates of DSM-IV disorders in children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 41(6), 761–769.
- Keeley, M. L., & Storch, E. A. (2009). Anxiety disorders in youth. *Journal of Pediatric Nursing*, 24(1), 26–40.
- Keeton, C. P., Kolos, A. C., & Walkup, J. T. (2009). Pediatric generalized anxiety disorder: Epidemiology, diagnosis, and management. *Pediatric Drugs*, 11(3), 171–183.
- McGee, R., Feehan, M., Williams, S., Partridge, F., Silva, P. A., & Kelly, J. (1990). DSM-III disorders in a large sample of adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry*, 29(4), 611–619.
- Muris, P., Steerneman, P., Merckelbach, H., Holdrinet, I., & Meesters, C. (1998). Comorbid anxiety symptoms in children with pervasive developmental disorders. *Journal of Anxiety Disorders*, 12(4), 387–393.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, 47(8), 921–929.
- Soutullo, C., & Figueroa-Quintana, A. (2010). *Convivir con niños y adolescentes con ansiedad [Living with children and adolescents with anxiety]*. Madrid: Editorial Médica Panamericana.
- Walkup, J. T., Albano, A. M., Piacentini, J., Birmaher, B., Compton, S. N., Sherrill, J. T., Ginsburg, G. S., Rynn, M. A., McCracken, J., Waslick, B., Iyengar, S., March, J. S., & Kendall, P. C. (2008). Cognitive behavioral therapy, sertraline, or a combination in childhood anxiety. *New England Journal of Medicine*, 359(26), 2753–2766.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229.
- Wittchen, H. U., Zhao, S., Kessler, R. C., & Eaton, W. W. (1994). DSM-III-R generalized anxiety disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, 51(5), 355–364.

Generative Complexity

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Definition

In his 1993 book, *Origins of Order: Self-Organization and Selection in Evolution*, Stuart Kauffman proposed generative complexity as a mechanism responsible for adaptive human skills. His model suggests that organisms and their neurobiological systems will self-generate with greater complexity. Generative complexity is thought to be a result of spontaneous variation driven by genetics. This unrestrained complexity is then shaped into an organized system of adaptive variations through a Darwinian selection process. Kauffman's generative complexity extends evolutionary theories to explain how human social behavior and other complex traits have evolved. Social behaviors such as bonding, imitation, and emotional expression may be related to distinct brain circuits. Impairment in these areas may be at the level of intermediate traits (those that are between genes and social behaviors). Applying the model of generative complexity, geneticists and other researchers are working to identify intermediate traits associated with the social behavioral deficits used to diagnose children with autism.

See Also

- [Candidate Genes in Autism](#)
- [Epigenetic Mechanisms](#)
- [Functional Connectivity](#)

References and Reading

Kauffman, S. (1993). *The origins of order: Self-organization and selection in evolution*. New York: Oxford University Press.

Waterhouse, L., Fein, D., & Nichols, E. G. W. (2007). Autism, social neuroscience, and endophenotypes. In F. R. Volkmar (Ed.), *Autism and pervasive developmental disorders* (2nd ed., pp. 307–335). New York: Cambridge University Press. Retrieved from http://ebookey.org/Fred-R-Volkmar-Autism-and-Pervasive-Developmental-Disorders_311848.html.

Generativity

- [Imagination](#)

Genetic Disorders

- [Medical Conditions Associated with Autism](#)

Genetics

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Definition

Genetics is a branch of biological science that focuses on the study of how the biological instructions for the development and functioning of living organisms are passed from generation to generation and how changes in these instructions lead to variation in the traits of organisms. In addition, genetics seeks to understand how these biological instructions operate within cells, the building blocks of living organisms.

Deoxyribonucleic acid (DNA) is the molecule that carries the instructions for the development and functioning of living organisms and that is passed down from generation to generation. The instructions are spelled out in a sequence or code of four chemical units called nucleotide bases. Certain segments of the DNA molecule called genes contain the code for creating the components of cells, most importantly, molecules called

proteins. On average, all humans possess genomes that are about 99% identical to each other. However, it is the genetic variation between individuals that is of particular interest to physicians and scientists searching for the underpinnings of human disease. It is presumed that if genetic factors increase the liability for certain conditions in a subset of the human population, the risks will be found among the small portion of the genome that varies from one person to the other.

Variations of all types, including rare mutations and common polymorphisms and sequence and structural variations, all contribute to the development of human disease. Some variations are passed from one generation to the next and give rise to inherited diseases. Mutations can also arise anew in germ cells during the creation of the zygote. These are called *de novo* mutations. Mutations can also arise in adult cell types. These are called somatic mutations.

The measure of the extent to which genetic factors contribute to a particular condition, syndrome, or disease is called heritability. Studies that compare how frequently pairs of monozygotic twins (identical twins) share a phenotype compared to how frequently pairs of dizygotic twins (nonidentical twins) share that phenotype are called twin studies. These types of studies show that autism has a high degree of heritability.

The identification of genetic factors contributing to ASD can have a number of benefits. Such knowledge can improve early diagnosis, help better predict the risk of an offspring developing autism, identify subgroups of individuals with differential responses to treatments, and contribute to a better understanding of the underlying biology of autism, which, in turn, can point to new strategies for treatment and prevention.

See Also

- ▶ [Dizygotic \(DZ\) Twins](#)
- ▶ [Monozygotic \(MZ\) Twins](#)
- ▶ [Recessive Genes](#)
- ▶ [Twin Studies in Autism](#)
- ▶ [Variable Expressivity of Genes](#)

References and Reading

- Alberts, B., Bray, D., et al. (2002). *The cell*. New York: Garland Science.
- Bailey, A., Le Couteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda, E., et al. (1995). Autism as a strongly genetic disorder: Evidence from a British twin study. *Psychological Medicine*, 25(1), 63.
- El-Fishawy, P., & State, M. W. (2010). The genetics of autism: Key issues, recent findings, and clinical implications. *The Psychiatric Clinics of North America*, 33(1), 83–105.
- Strachan, T., & Read, A. P. (2004). *Human molecular genetics*. New York: Garland Press.
- Watson, J. D., & Crick, F. H. C. (1953). Molecular structure of nucleic acids. *Nature*, 171(4356), 737–738.

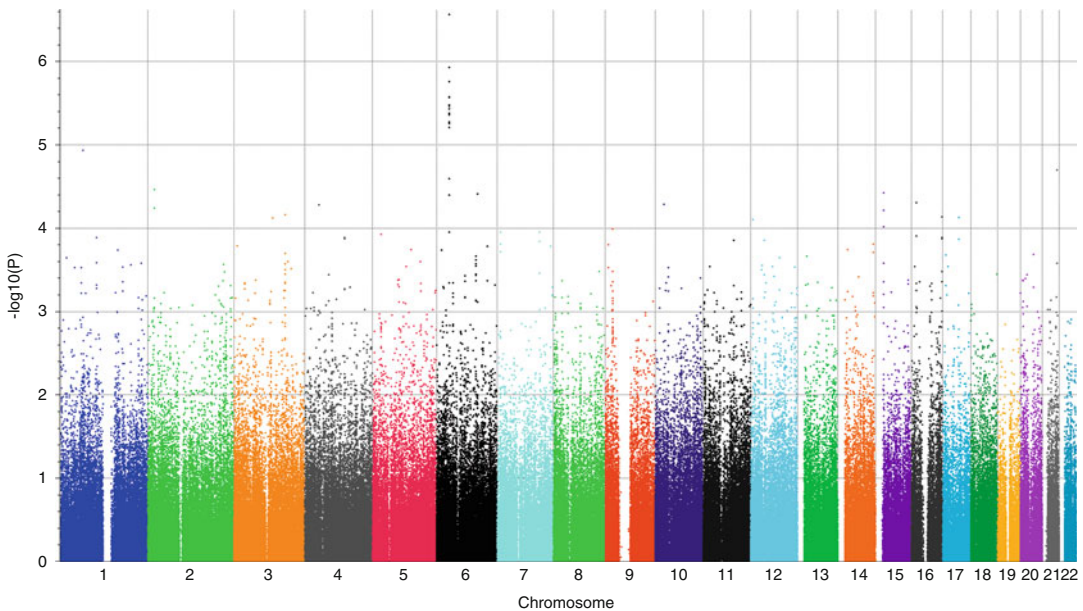
Genome-wide Association

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Definition

Genome-wide association (GWA) is an approach to studying a disease or trait whereby markers (usually common genetic variants, or polymorphisms) across the genomes of many individuals with (cases) and without (controls) the phenotype are scanned in order to find genetic variations associated with the disease or trait. The associated genetic variants, with allele frequencies that differ significantly between cases and controls, can serve as powerful indicators of regions of the human genome where the phenotype-causing events reside. The effect size of a variant associated with the phenotype in a GWA study is usually reported as an *odds ratio* (ratio of proportion of individuals in the case group carrying the risk allele to proportion of control individuals carrying the same allele). Statistical significance of the odds ratio (whether it is significantly different from 1) is typically calculated using a chi-squared test. In order to protect against false positive associations that may occur with the large number of statistical comparisons required for a GWA study,



Genome-wide Association, Fig. 1 *Manhattan plot.* A scatter plot typically used to summarize results of a GWA study. Genomic coordinates are displayed along the x-axis, and the negative logarithm of association p -value

for each marker polymorphism across the genome is displayed on the y-axis. The strongest associations have the smallest p -values and therefore the largest negative logarithms

the commonly accepted genome-wide significance threshold is $p < 5 \times 10^{-8}$. GWA study results are commonly summarized by a “Manhattan plot” (Fig. 1), which shows the negative logarithm of the p -value for each marker polymorphism across the genome.

Historical Background

Prior to the introduction of GWA studies (around 2000), the main approach to investigating genetic causes of human disease was *genetic linkage* studies in families. While linkage is useful for single gene Mendelian disorders, using this approach for common and complex diseases generally yields inconsistent results. *Genetic association* arose as an alternative to linkage for detecting weaker genetic effects and was initially performed by interrogating polymorphisms in individual candidate genes between cases and controls. The *common disease-common variant* hypothesis, that common polymorphisms ($>1\%$ allele frequency) contribute to susceptibility of common diseases,

provided the rationale for the use of GWA studies to map loci contributing to common disease. In order to test this hypothesis, several tools and resources were developed and converged to allow identification of common variants throughout the genome. The completion of the *Human Genome Project* in 2003 provided a human reference genome sequence, and the *International HapMap Project* in 2005 identified a majority of the common single nucleotide polymorphisms (SNPs) investigated in GWA studies and also identified *haplotypes* which indicated those SNPs describing most of the genetic variation.

The first GWA study, published in 2005, identified two SNPs within the complement factor H gene with significantly different allele frequencies between 96 cases with age-related macular degeneration and 50 controls (Klein et al. 2005). In 2007, the Wellcome Trust Case Control Consortium reported a landmark GWA study, including 14,000 cases of seven common diseases (including types 1 and 2 diabetes, coronary heart disease, hypertension, rheumatoid arthritis, Crohn’s disease, and bipolar disorder) and 3,000

controls (Welcome Trust Case Control Consortium 2007). This study suggested many unexpected genes underlying risk for the included diseases.

Current Knowledge

Current testing for GWA typically involves testing hundreds of thousands to millions of common SNP markers for association with disease in cases versus controls. Recent advances in microarray technologies, in which millions of SNP probes can be arranged on a single microscope slide, have allowed researchers to query these genetic markers, evenly spaced throughout the genome, in a single reaction and at relatively low cost. This allows for testing of association throughout the entire genome, without having to choose candidate loci in advance.

Many early GWA studies were plagued with difficulties which resulted in inconsistent findings. For example, overestimation of allelic effect sizes led to initial cohort sizes that were underpowered, and inadequate corrections for cryptic confounds such as relatedness and ancestry could lead to false positive findings. As these difficulties have become more appreciated and sufficiently addressed, GWA studies in several areas of medicine, including diabetes, intracranial aneurysms, and inflammatory bowel disease, have yielded reproducible results (Hindorff et al. 2011; Manolio 2010).

In autism spectrum disorders (ASD), three associated alleles, one each from three independent GWA studies, have been identified that meet accepted discovery criteria, as of December 2011: (1) an intragenic region on chromosome 5p14 between the genes *CDH9* and *CDH10* (Wang et al. 2009), (2) a region on 5p15.3 within 80 kb of *SEMA5A* (Weiss et al. 2009), and (3) an intragenic region on 20p12 within *MACROD2* (Anney et al. 2010). While each study demonstrated genome-wide significance and internal replication, none of the three studies replicate one another, and a combined analysis of data from all three studies appears to decrease evidence for association for all three alleles (Devlin et al.

2011). Despite this lack of convergence so far, it is believed that multiple common variants of very small effect remain to be identified in ASD, and increasing sample sizes may be required (State and Levitt 2011).

The current state of GWA studies in ASD illustrates an important lesson learned over the last decade for GWA studies across all of medicine: most of the variants found are associated with only a small increased risk of the disease (e.g., odds ratios of 1.1–1.5 per associated allele) and explain only a small amount of the heritability, as determined from twin studies. These modest effect sizes have little clinically useful predictive value and suggest that most GWA studies have low power to detect associations at typical sample sizes (Altshuler et al. 2008; Manolio 2010). Furthermore, the question of why so much heritability is unexplained by GWA studies has led to an increased focus on the contribution of rare and structural variants (poorly detected by currently available genotyping arrays), interactions between genes, interactions between genes and environmental factors, and studies of ancestrally diverse populations (Manolio 2010; Manolio et al. 2009).

Future Directions

With the application of rigorous methodology and large sample sizes to GWA studies in ASD, there is hope that definitive replication of common ASD alleles will emerge. This will lead to the additional challenge of translating such findings into potentially targetable neurobiological mechanisms. Even with replication, there remains the challenge of narrowing an associated locus to a single variant that is directly implicated in disease etiology, an achievement which has so far remained elusive for GWA studies throughout medicine.

Next-generation sequencing technologies are now allowing a genome-wide view of all nucleotides in the genome. As the 1,000 Genomes Project (1000 Genomes Project Consortium 2010) and other large-scale efforts aim to create an accurate map of all single nucleotide and structural variants in the genome, GWA studies may be extended to rare variants which could explain

some of the unmapped heritability in common polymorphism GWA studies.

See Also

► [Common Disease-Common Variant Hypothesis](#)

References and Reading

- 1000 Genomes Project Consortium. (2010). A map of human genome variation from population-scale sequencing. *Nature*, 467(7319), 1061–1073.
- Altshuler, D., Daly, M. J., & Lander, E. S. (2008). Genetic mapping in human disease. *Science*, 322(5903), 881–888.
- Anney, R., Klei, L., Pinto, D., Regan, R., Conroy, J., Magalhaes, T. R., et al. (2010). A genome-wide scan for common alleles affecting risk for autism. *Human Molecular Genetics*, 19(20), 4072–4082.
- Devlin, B., Melhem, N., & Roeder, K. (2011). Do common variants play a role in risk for autism? Evidence and theoretical musings. *Brain Research*, 1380, 78–84.
- Ge, D., Fellay, J., Thompson, A. J., Simon, J. S., Shianna, K. V., Urban, T. J., et al. (2009). Genetic variation in IL28B predicts hepatitis C treatment-induced viral clearance. *Nature*, 461(7262), 399–401.
- Hindorf, L., MacArthur, J., Wise, A., Hall, P., Klemm, A., & Manolio, T. A. (2011). Catalog of published genome-wide association studies. Retrieved December 15, 2011 from www.genome.gov/gwastudies
- Klein, R. J., Zeiss, C., Chew, E. Y., Tsai, J. Y., Sackler, R. S., Haynes, C., et al. (2005). Complement factor H polymorphism in age-related macular degeneration. *Science*, 308(5720), 385–389.
- Manolio, T. A. (2010). Genomewide association studies and assessment of the risk of disease. *The New England Journal of Medicine*, 363(2), 166–176.
- Manolio, T. A., Collins, F. S., Cox, N. J., Goldstein, D. B., Hindorf, L. A., Hunter, D. J., et al. (2009). Finding the missing heritability of complex diseases. *Nature*, 461(7265), 747–753.
- Pearson, T. A., & Manolio, T. A. (2008). How to interpret a genome-wide association study. *Journal of the American Medical Association*, 299(11), 1335–1344.
- State, M. W., & Levitt, P. (2011). The conundrums of understanding genetic risks for autism spectrum disorders. *Nature Neuroscience*, 14(12), 1499–1506.
- Wang, K., Zhang, H., Ma, D., Bucan, M., Glessner, J. T., Abrahams, B. S., et al. (2009). Common genetic variants on 5p14.1 associate with autism spectrum disorders. *Nature*, 459(7246), 528–533.
- Weiss, L. A., Arking, D. E., Gene Discovery Project of Johns Hopkins & the Autism Consortium, Daly, M. J., & Chakravarti, A. (2009). A genome-wide linkage and

association scan reveals novel loci for autism. *Nature*, 461(7265), 802–808.

Welcome Trust Case Control Consortium. (2007). Genome-wide association study of 14,000 cases of seven common diseases and 3,000 shared controls. *Nature*, 447(7145), 661–678.

Geodon

► [Ziprasidone](#)

Geodon (Ziprasidone)

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Synonyms

[Marketed as Geodon; Zeldox](#)

Definition

Ziprasidone is an atypical antipsychotic agent with a high affinity for dopamine, serotonin, and alpha-adrenergic receptors and a moderate affinity for histamine receptors. The mechanism of action of ziprasidone is unknown. Ziprasidone is a complex molecule with the chemical name 5-[2-[4-(1,2-benzisothiazol-3-yl)-1-piperazinyl]ethyl]-6-chloro-1,3-dihydro-2H-indol-2-one. Its action is thought to be caused by antagonizing dopamine receptors, especially D2. Antagonism of histaminic and alpha-adrenergic receptors may be related to adverse effects of ziprasidone, such as sedation and orthostasis.

Ziprasidone can be administered intramuscularly or orally with food. Steady-state plasma concentrations are achieved within 1–3 days with a mean half-life ranging from 2 to 5 h. Ziprasidone is largely metabolized by aldehyde oxidase and for a smaller portion by cytochrome P450 3A4

(CYP3A4). Ziprasidone has a number of adverse effects most notably including sedation, insomnia, orthostasis, life-threatening neuroleptic malignant syndrome, akathisia, tardive dyskinesia, chest pains, and impaired erectile function.

In the United States, ziprasidone is Food and Drug Administration (FDA) approved for the treatment of schizophrenia and for acute treatment of mania and mixed states associated with bipolar disorder.

See Also

- ▶ [Antipsychotics: Drugs](#)
- ▶ [Pyramidal System](#)

References and Reading

De Hert, M., Dobbelaere, M., Sheridan, E. M., Cohen, D., & Correll, C. U. (2011). Metabolic and endocrine adverse effects of second-generation antipsychotics in children and adolescents: A systematic review of randomized, placebo controlled trials and guidelines for clinical practice. *European Psychiatry*, 26(3), 144–158.

Komossa, K., Rummel-Kluge, C., Hunger, H., Schwarz, S., Bhoopathi, P. S., Kissling, W., et al. (2009). Ziprasidone versus other atypical antipsychotics for schizophrenia [Review]. *Cochrane Database of Systematic Reviews*, 4, CD006627.

Newcomer, J. W. (2005). Second-generation (atypical) antipsychotics and metabolic effects: A comprehensive literature review. *CNS Drugs*, 19 (Suppl. 1), 1–93.

Geri Dawson

Sara Jane Webb
Psychiatry and Behavioral Sciences and UW Autism, Seattle Children’s Research Institute, University of Washington, Seattle, WA, USA

Name and Degrees

Geraldine Dawson
Ph.D. Duke University, Durham NC, USA

B.S. University of Washington, Seattle WA, 1974
Ph.D. University of Washington, Seattle WA, 1979
Postdoctoral Fellow & Clinical Intern. UCLA, Los Angeles CA, 1979–1980

Major Appointments (Institution, Location, Dates)

Assistant Professor of Psychology, University of North Carolina, Chapel Hill NC	(1980–1985)
Director, Child Clinical Psychology Program University of Washington, Seattle WA	(1985–1991; 1999–2004)
Associate Professor, Psychology University of Washington, Seattle WA	(1985–1990)
Professor with tenure, Psychology University of Washington, Seattle WA	(1990–2007)
Founding Director, UW Autism Center, University of Washington, Seattle WA	(2000–2007)
Chief Science Officer, Autism Speaks	(2008–2013)
Research Professor, Psychiatry University of North Carolina at Chapel Hill, NC	(2008–2013)
Professor Emeritus, Psychology University of Washington, Seattle WA	(2008–)
Public Member, Interagency Autism Coordinating Committee, DHHS	(2012–2018)
Director, Duke Center for Autism and Brain Development Duke University, Durham NC	(2013–)
Faculty Affiliate, Duke Institute for Brain Sciences, Durham NC	(2013–)
Professor with tenure, Psychiatry & Behavioural Science Duke University, Durham NC	(2014–)
Professor, Psychology and Neuroscience, Duke University, Durham NC	(2014–)
Professor, Pediatrics Duke University, Durham NC	(2014–)
President, International Society for Autism Research	(2015–2018)
Faculty Affiliate, Center for Child and Family Policy Duke University, Durham NC	(2016–)



Major Honors and Awards

Fellow, American Psychological Association
Fellow, American Psychological Society

Since 2000

Autism Society of America Honoree for Research Contributions to the Autism Community (2004)
Autism Society of Washington Medical Professional of the Year (2004)
Autism Hero Award Cure Autism Now Foundation (2006)
NIH Top Science Advances in Autism Research Award - (2007, 2008, 2009, 2010, 2012, 2013, 2016)
Top Ten Science Advances of the Year Award - Autism Speaks (2008, 2009, 2010, 2012, 2013)
Geoffrey Beene Rock Star of Science Award (2010)
Univ. of Wash College of Arts & Science Timeless Outstanding Service & Achievement Award (2012)
Association for Psychological Science James McKeen Cattell Lifetime Achievement Award (2012)
The News and Observer Triangle and NC Tarheel of the Week (2014)
President, International Society for Autism Research (2015)
Duke School of Medicine Noteworthy Faculty Recognition Award (2016)

Landmark Clinical, Scientific, and Professional Contributions

Dr. Geraldine Dawson's research on autism spectrum disorders has focused on early detection and intervention and elucidating the nature of brain dysfunction and development. A former Professor at University of Washington and Chief Scientific Officer at Autism Speaks, Dr. Dawson is currently the Director of the Duke Center for Autism and Brain Development at Duke University, President of the International Society for Autism Research, and a Professor of Psychiatry and Behavioral

Sciences, Pediatrics, and Psychology and Neuroscience at Duke University. Dr. Dawson's work demonstrated how maternal depression influences early brain activity and stress responses in infants and children (Dawson et al. 1992). Dawson used home videotapes to demonstrate that autism symptoms emerge during the first year of postnatal life (Osterling and Dawson 1994) and empirically validate the phenomenon of autistic regression (Werner and Dawson 2005). She published the first case study of an infant with autism (Dawson et al. 2000). Dawson demonstrated that autism is associated with early deficits in social attention (Dawson et al. 1998; Dawson et al. 2004) and proposed that such attention deficits are related to reduced sensitivity to the reward value of social stimuli ("social motivation hypothesis") (Dawson et al. 2002). Dawson's laboratory pioneered the use of electrophysiological techniques to study brain function in autism, including during early childhood (Dawson et al. 1986; Dawson et al. 1988; Dawson et al. 2002). Research in her laboratory identified autism neural signatures (event-related brain potentials in response to faces) (Dawson et al. 2002) that were later shown to be associated with early risk identification (Jones et al. 2016) and endophenotypes in relatives (Dawson et al. 2005), and used for biomarkers in autism clinical trials (Dawson et al. 2012a, b). As part of the NIMH STAART (Studies To Advance Autism Research and Treatment), and in collaboration with Dr. Sally Rogers, Dr. Dawson co-developed and empirically validated the Early Start Denver Model (Rogers and Dawson 2010; Rogers et al. 2012), the first comprehensive early intervention program for very young children with autism (Dawson et al. 2010; Estes et al. 2015). ESDM has been translated into 14 languages and is used worldwide. Her research demonstrating that early intervention based on ESDM can normalize brain activity in children with autism was recognized by TIME magazine as one of the top 10 medical breakthroughs of 2012 (Dawson et al. 2012b). Dawson has also been a contributor to several autism studies on using structural and functional MRI (e.g., Sparks et al. 2002; Friedman et al. 2007) and genetic risk factors.

Short Biography

Dr. Dawson is a licensed, practicing Child Clinical Psychologist, obtaining her PhD from University of Washington and completing her postdoctoral training and internship at the UCLA Neuropsychiatric Institute. As a faculty at the University of North Carolina and University of Washington, Dr. Dawson has trained 23 doctoral students and 5 postdoctoral fellows who have gone on to careers as clinical psychologists and university faculty. She continues mentoring students at Duke, where from 2013 to 2016, she trained 1 doctoral student, 9 postdoctoral fellows, 1 clinical psychology intern, and 2 medical students. As faculty at UW, she received continuous funding for her research from 1980 to 2008 including serving as Director for multidisciplinary center grants from the NIH Collaborative Programs for Excellence in Autism, STAART, and Autism Center of Excellence scientific programs. These multidisciplinary autism research programs focused on genetics, neuroimaging, early diagnosis, and treatment. At UW, Dr. Dawson also founded and oversaw the UW Autism Center endowed treatment center for children with ASD. At Autism Speaks, she directed \$20–30 million in annual research funding, including funding for the Autism Treatment Network, the Autism Genetic Resource Exchange, the Autism Genome Project, and the Autism Tissue Program. Under her leadership at Autism Speaks, several new initiatives were launched, including the Translational Medicine Research Initiative, Postdoctoral Research Fellowship, the Trailblazer Award, the Autism 10K Genome Project, the Early Access to Care Initiative, the Global Autism Public Health Initiative, and DELSIA (Delivering Scientific Innovation for Autism). At Duke University, Dr. Dawson is the Founding Director of the Duke Center for Autism and Brain Development at Duke University, North Carolina, which includes an integrated interdisciplinary clinical care, research, and training facility. Her current research at Duke focuses on early detection and early behavioral intervention and clinical trials testing novel molecular and cellular therapies for

autism using eye-tracking, EEG, and MRI as biomarkers and outcome measures. Dr. Dawson has published over >270 articles and 10 books on early detection and treatment of autism and brain development. She has testified before the US Senate in support of legislation for autism research funding and insurance coverage for autism services.

See Also

- [Broader Autism Phenotype](#)
- [Early Intervention](#)
- [Early Start Denver Model](#)
- [Face Perception](#)
- [Retrospective Infant Video Analysis](#)
- [USA and Autism](#)

References and Reading

- Dawson, G., Finley, C., Phillips, S., & Galpert, L. (1986). Hemispheric specialization and the language abilities of autistic children. *Child Development*, 57, 1440–1453. PMID: 3802970.
- Dawson, G., Finley, C., Phillips, S., & Galpert, L. (1988). Reduced P3 amplitude of the event-related brain potential: Its relationship to language ability in autism. *Journal of Autism and Developmental Disorders*, 18, 493–504. PMID: 3215878.
- Dawson, G., Grofer, L., Hill, D., Panagiotides, H., & Speiker, S. (1992). Frontal lobe activity and affective behavior of infants of mothers with depressive symptoms. *Child Development*, 63, 725–737. PMID: 1600832.
- Dawson, G., Meltzoff, A., Osterling, J., & Brown, E. (1998). Children with autism fail to orient to social stimuli. *Journal of Autism and Developmental Disorders*, 28, 479–485. PMID: 9932234.
- Dawson, G., Osterling, J., Meltzoff, A. N., & Kuhl, P. (2000). Case study of the development of an infant with autism from birth to 2 years of age. *Journal of Applied Developmental Psychology*, 21, 299–313. PMID: 23667283.
- Dawson, G., Carver, L., Meltzoff, A. N., Panagiotides, H., & McPartland, J. (2002). Neural correlates of face recognition in young children with autism spectrum disorder, developmental delay, and typical development. *Child Development*, 73, 700–717. PMID: 12038546.
- Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., & Liaw, J. (2004). Defining the early social attention impairments in autism: Social orienting, joint

- attention, and responses to emotions. *Developmental Psychology*, 40(2), 271–283. PMID: 14979766.
- Dawson, G., Webb, S. J., Wijsman, E., Schellenberg, G., Estes, A., Munson, J., & Faja, S. (2005). Neurocognitive and electrophysiological evidence of altered face processing in parents of children with autism: Implications for a model of abnormal development of social brain circuitry in autism. *Development and Psychopathology*, 17, 679–697. PMID: 16262987.
- Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., Donaldson, A., & Varley, J. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The early start Denver model. *Pediatrics*, 125(1), e17–e23. PMCID: PMC4951085. Noted in the IACC summary of advances 2010.
- Dawson, G., Bernier, R., & Ring, R. H. (2012a). Social attention: A possible early indicator of efficacy in autism clinical trials. *Journal of Neurodevelopmental Disorders*, 4, 11. PMCID: PMC3436672.
- Dawson, G., Jones, E. J., Merkle, K., Venema, K., Lowy, R., Faja, S., Kamara, D., Murias, M., Greenson, J., Winter, J., Smith, M., Rogers, S., & Webb, S. J. (2012b). Early behavioral intervention is associated with normalized brain activity in young children with autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(11), 1150–1159. PMID: 23101741. Noted in the IACC Summary of Advances 2012; TIME Magazine's Top Ten Medical Breakthrough 2012.
- Estes, A., Munson, J., Rogers, S., Greenson, J., Winter, J., & Dawson, G. (2015). Long-term outcomes of early intervention in 6 year old children with autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 54(7), 580–587. <https://doi.org/10.1016/j.jaac.2015.04.005>.
- Friedman, S. D., Shaw, D. W., Artru, A. A., Dawson, G., Petropoulos, H., & Dager, S. R. (2007). Gray and white matter brain chemistry alterations in young children with autism. *Archives of General Psychiatry*, 63, 786–794. PMID: 16818868.
- Jones, E. J. H., Venema, K., Earl, R., Lowy, R., Barnes, K., Estes, A., Dawson, G., & Webb, S. J. (2016). Reduced engagement with social stimuli in 6-month-old infants with later autism spectrum disorder: A longitudinal prospective study of infants at high familial risk. *Journal of Neurodevelopmental Disorders*, 18(8), 7. PMID: 26981158.
- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders*, 24, 247–257. PMID: 8050980.
- Pinto, D., et al. (2010). Functional impact of global rare copy number variation in autism spectrum disorders. *Nature*, 466, 368–372. PMCID: PMC3021798.
- Rogers, S. J., & Dawson, G. (2010). *The early start Denver model for young children with autism: Promoting language, learning, and engagement*. New York: The Guilford Press. Translated into Japanese, Italian, Dutch, French, Spanish, Portuguese, Chinese, Arabic, Romanian, Korean, Polish, German, Russian, and Turkish.
- Rogers, S. J., Dawson, G., & Vismara, L. (2012). *An Early Start for your Child with Autism*. New York: Guilford Press. Awarded a 2012 American Journal of Nursing book of the year award – 1st place. Translated into Spanish, Dutch, Japanese, Portuguese, French, Chinese, Lithuanian, and Russian.
- Sparks, B. F., Friedman, S. D., Shaw, D. W., Aylward, E. H., Echelard, D., Artru, A. A., Maravilla, K. R., Giedd, J. N., Munson, J., Dawson, G., & Dager, S. R. (2002). Brain structural abnormalities in young children with autism Spectrum disorder. *Neurology*, 59, 184–192. PMID: 12136055.
- Werner, E., & Dawson, G. (2005). Validation of the phenomenon of autistic regression using home videotapes. *Archives of General Psychiatry*, 62, 889–895. PMID: 16061766.

German Measles

► Rubella

Germany and Autism

Sven Bölte

Center of Neurodevelopmental Disorders (KIND), Department of Women's and Children's Health and Child and Adolescent Psychiatry, Center for Psychiatry Research, Karolinska Institutet and Stockholm County Council, Stockholm, Sweden

Historical Background

Leo Kanner had studied medicine in Berlin between 1913 and 1919, got Prussian citizenship in 1920, worked at the Charité, and as a private practitioner in Berlin until 1924 (Neumärker 2003) before immigrating to the USA. His concept of autism (Kanner 1943) was introduced to Germany shortly after World War II by Hermann Stutte, a child and adolescent psychiatrist from Marburg, who had exchange with Arn Van

Krevelen, a Dutch child and adolescent psychiatrist from Leiden who was among the first Europeans to confirm the condition in the early 1950s (Van Krevelen 1952a, b). During the same time psychiatrist Gerhard Bosch described the first cases of autism in Frankfurt, particularly focusing on language development issues and the differentiation of autism and schizophrenia. His book “Infantile autism: A philosophical study of the languages of autism” was published in 1962 (in English 1970) and includes thoughtful and empathetic case descriptions together with many valuable novel insights such as a discussion of the differences and similarities between Kanner’s and Hans Asperger’s (1944) descriptions and a need to broaden the concept of autism, long before the term Asperger syndrome and autism spectrum were coined. Bölte and Bosch (2004, 2005) followed up on Bosch’s cases 40 years after initial diagnosis. In the 1970s, Doris Weber from Marburg studies autism in relation to Heller’s dementia infantilis (1908) [childhood disintegrative disorder], a diagnosis often given to children with autism at that time in Germany, while Gerhardt Nissen in Würzburg studied Kanner’s and Asperger’s description versus psychogenic and somatic forms of autism. The concept of Asperger syndrome was not used frequently in Germany before the early 1970s. While until the later 1970s, autism was still widely considered an infantile psychosis and psychoanalytic views were also still influential, it is today widely accepted among many professions that autism is a brain-based developmental disorder of multiple genetic and environmental origin. The understanding of autism being a spectrum disorder was well established before the release of DSM-5 (American Psychiatric Association 2013). Autism awareness and services in many parts of the society and among clinical professionals working with adults have gradually developed in recent years. National clinical guidelines for the assessment of autism spectrum disorder have recently been updated by the German Society of Child and Adolescent Psychiatry and Psychotherapy and the German Association for Psychiatry, Psychotherapy, and Psychosomatics (<http://www.awmf.org/leitlinien/detail/II/028-018.html>).

Legal Situation

The Social Security State Book came into effect in 2001, entailing rules for rehabilitation and participation for individuals with disabilities. The Federal Act of Equal Opportunities was released in 2002 aiming to achieve equal opportunities for people in public space (“accessibility”). The General Equal Treatment Act came into effect in 2006 and comprises among other things the equalization of people with disabilities in the area of the civil law. The United Nations convention on the rights of people with disabilities was ratified in 2006. For the state, counties and municipalities, administrations and courts, these guidelines are now applicable law, and all national laws need to be interpreted in the light of the UN convention. The degree of disablement and the entitlement are connected to the International Classification of Diseases (ICD-10, WHO 1992) codes for different forms of autism spectrum disorder (F84.0, F84.1, F84.5). Thus, an ICD-10 diagnosis is required before an assessment of the degree of disability which is a prerequisite for access to additional clinical and other services. The assessment of the degree of disability is complex and often lengthy and is reassessed with varying intervals.

The Social Security State Book and The General Equal Treatment Act include several support means for people classified as having different degrees of disability, for instance, additional autism-specific individual intervention in addition to medical treatment; speech, language, and occupational therapy; integration aids for kindergarten and school; and further education and employment. It also comprises costs for traveling, a study assistant for structuring and orientation, and assistive technologies. In case of a high degree of disability there are several options for sheltered or supported/integrated employment. In long-term full support residential care, the complete basic needs are covered, plus pocket money. In partly supported homes, people with autism cover the costs for food, clothing, and rent themselves by their salary or different forms of relief payments. Caregivers can apply for care insurance payments for supporting their child if they

compensate for the individual with autism's disabilities, instead of using public services. In this case, the medical service of the statutory nursing care insurance decides on the degree of an individual's disability based on physician and nurse assessments. Another option is the so-called personal budget that was released in 2008, where a social service may not only be given as a traditional benefit in kind, but a payment or voucher to a caregiver. This gives the possibility to caregivers to decide themselves which help they want to provide to their child. For parents of children with autism it is also recommended that they do a "disabled testament" in order to ensure that after their deaths the complete heritage is not transferred to the welfare agency, so that their offspring can still receive care beyond livelihood using the heritage. Currently, a new regulation, the Federal law of integration and participation, is under negotiation, planned to come into effect January 1, 2017. While it is aiming to improve the inclusion of individuals with functional disabilities, its draft has caused considerable concerns about weakened access to services for individuals with autism.

Development of Services

In the beginning, individuals diagnosed with autism were treated at university hospitals and then referred to institutions for long-term residential care, mostly designed for intellectual disability care. As autism was rarely diagnosed until the later 1960s, there was little apparent need or interest for specialized care for autism. The Max-Planck Institute of Psychiatry Research in Munich introduced behavior therapy to autism influenced by US American developments. Like in other countries, a major breakthrough for autism was achieved in Germany by an emerging engagement of parents trying to get adequate diagnostic assessment and treatment for their children with mostly severe forms of autism. In 1970, "Hilfe für das autistische Kind" (today renamed in "Autismus Deutschland"; www.autismus.de) was founded. During this time, many parents sought advice from Hans Kehrner, child and adolescent

psychiatrist in Münster, who also had published the first articles on behavior therapy for social and language difficulties in autism (Kehrner and Körber 1971; Kehrner and Budde 1972) and the first comprehensive text book on autism (Kehrner 1978). The parent society developed rapidly and started to establish autism therapy centers favoring family work, the upcoming TEACCH approach, and behavior modification, but also a variety of alternative forms of intervention. Today, more and more special school classes and even occasionally regular schools provide day care and inclusive teaching for children with autism. A range of academic and municipality hospitals have established autism centers, specialized day care units, or outpatient department for autism assessment and intervention aiming to follow evidence-based best practice principles. There is also a clear tendency to accomplish early diagnosis of autism and treatment in child and adolescent psychiatry. Even many private practitioners offer autism-specific services, some center-like organizations offering particular forms of intervention only, such as Applied Behavior Analysis, TEACCH, PECS, or social skills training. In the last decade, services for adults with autism spectrum disorder have started to improve in clinical settings, but also in other fields of society (e.g., employment, living). For instance, the psychiatry departments at the universities in Freiburg and Cologne run specialized departments for higher functioning autism. Most of this development is driven by increasing diagnoses rates of autism among adults and a previous lack of autism expertise among professionals in the adult mental health field, but also by the increasing voice of the neurodiversity and empowerment movement, for instance organized within the "Aspies e.V., Menschen im Autismus-spektrum" (www.aspies.de).

Research

Autism science has developed considerably in Germany in the last decade. For instance, while in the year 2005 only 19 original articles (cited in PubMed) on autism were published with a

German academic affiliation, the number was 171 in the year 2015. Many German clinics and labs are today involved in European and international autism research collaborations and projects, such as the European Collaboration in Science and Technology (COST) activity “Enhancing the Scientific Study of Early Autism” (www.cost-essea.com), EU-AIMS (www.eu-aims.eu), European Autism Interventions – A Multicentre Study for Developing New Medications – the largest single grant for autism in the world, and the largest for the study of any mental health disorder in Europe, or The Autism Genome Project (www.autismspeaks.org/science/initiatives/autism-genome-project). Universities that are particularly active in autism research are Aachen, Berlin, Cologne, Frankfurt, Freiburg, Mannheim, and Marburg. In 2008, the Scientific Society Autism Spectrum (WGAS) ([www.wgas-autismus.org](http://wgas-autismus.org)) was established. The WGAS is devoted to supporting basic and applied autism research primarily in Germany, Switzerland, and Austria. Among other things, the WGAS meets this goal through producing scientific publications, facilitating annual events such as the Scientific Meeting for Autism Spectrum Conditions (WTAS) (<http://wgas-autismus.org/meeting-wtas>), promoting networking among colleagues active within the autism field, and the formation of professional groups. WGAS also provides training and continuing education for researchers, support for junior scientists, and sponsorship of awards for outstanding research contributions.

Training

The majority of autism specific training in Germany is still conveyed via postgraduate courses, predominantly by autism therapy centers or private providers. Such education often focuses on applied intervention techniques, such as ABA, TEACCH, PECS, and social skills training but also and autism assessment and other preclinical and applied areas. Autism often only plays a minor role in the comprehensive postgraduate curricula to accomplish approximation for being a certified psychotherapist. Clinical ADOS training is offered by several

academic child and adolescent psychiatry departments. Some higher education departments have established autism-specific modules or curricula for students, such as the Department of Psychology and Pedagogics at Ludwig Maximilian University Munich, The Jacobs University in Bremen, and the University of Applied Science in Münster.

References and Reading

- American Psychiatric Association (APA). (2013). *Diagnostic and statistical manual of mental disorders, 5th edition text-revision (DSM-5)*. Washington, DC: APA Press.
- Asperger, H. (1944). Die autistischen Psychopathen im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Bölte, S., & Bosch, G. (2004). Bosch’s cases: A 40 years follow-up. *German Journal of Psychiatry*, 7, 10–13.
- Bölte, S., & Bosch, G. (2005). The long-term outcome in two females with autism spectrum disorder. *Psychopathology*, 38, 151–154.
- Bosch, G. (1962). *Der frühkindliche Autismus – eine klinische und phänomenologisch-anthropologische Untersuchung am Leitfaden der Sprache*. Berlin: Springer.
- Bosch, G. (1970). *Infantile autism: A clinical and phenomenological-anthropological approach taking language as the guide*. New York: Springer.
- Heller, T. (1908). Dementia infantilis. *Zeitschrift für die Erforschung und Behandlung des jugendlichen Schwachsinn*, 2, 141–165.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–253.
- Kehrer, H. E. (1978). *Kindlicher Autismus* (Bibliotheca psychiatrica, Vol. 157). Basel: S. Karger.
- Kehrer, H. E., & Budde, R. (1972). Verhaltenstherapie von Kontaktstörungen bei Autismus Infantum. *Acta Paedopsychiatrica*, 39, 253–263.
- Kehrer, H. E., & Körber, H. P. (1971). Sprachbehandlung durch Verhaltenstherapie bei autistischen-mutistischen Kindern. *Acta Paedopsychiatrica*, 38, 2–17.
- Neumärker, K. J. (2003). Leo Kanner: His years in Berlin, 1906–1924: The roots of autistic disorder. *Historical of Psychiatry*, 14, 205–218.
- Van Krevelen, A. (1952a). Early infantile autism. *Zeitschrift für Kinderpsychiatrie. Revue de psychiatrie infantile*, 19, 81–97.
- Van Krevelen, A. (1952b). A case of early infantile autism. *Nederlands Tijdschrift voor Geneeskunde*, 96, 202–206.
- World Health Organization (WHO). (1992). *The ICD-10 classification of mental and behavioural disorders. Clinical descriptions and guidelines*. Geneva: WHO.

Gesell, Arnold

W. Thornton N. Deegan
Yale Child Study Center, New Haven, CT, USA

Name and Degrees

- Gesell, Arnold Lucius (June 1, 1880–May 21, 1961).
- Arnold Gesell received his B.Ph. in 1903 from the University of Wisconsin and his Ph.D. in Education in 1906 from Clark University. In 1911, Gesell accepted an assistant professorship at Yale University, teaching graduate courses in education while working toward his M.D., which he received in 1915.

Major Appointments (Institution, Location, Dates)

- Stevens Point High School, Stevens Point, WI, 1899–1901, Teacher of US history, ancient history, German, accounting, and commercial geography.
- Chippewa Falls High School, Chippewa Falls, WI, 1903–1904, Principal.
- Los Angeles State Normal School, Los Angeles, CA, 1908–1910, Professor of Psychology.
- Yale University, New Haven, CT, 1911–1948, Assistant Professor of Education, Professor of Education, Professor of Child Hygiene, Founder and Director of the Yale Psycho-Clinic (later renamed Yale Clinic of Child Development, later renamed Yale Child Study Center), Professor Emeritus of Child Hygiene.
- Connecticut State Board of Education, Hartford, CT, 1915–1919, School Psychologist.
- Connecticut Commission on Child Welfare, Hartford, CT, 1919–1921, Member.
- American Psychological Association, Washington, D.C., 1922–1936, Director.
- American Child Health Association, 1928–1940, Director.

- New Haven Hospital, New Haven, CT, 1928–1948, Attending pediatrician.

Major Honors and Awards

Dr. Gesell received numerous honors and awards during his lifetime. Among them are honorary Doctor of Science degrees from Clark University (1930) and the University of Wisconsin (1953). The Gesell Institute of Child Development is named in his honor.

Landmark Clinical, Scientific, and Professional Contributions

Dr. Gesell is most renowned for his Developmental Schedules, which track the behavioral development of typical children through the first few years of life. Dr. Gesell was also known for his groundbreaking observational methods, pioneering the use of cinematography, still photography, and one-way vision screens to observe subjects without disrupting them. Cinematography especially provided a way to reproduce nuances of behavior faithfully and completely for later analysis. Through his professional contributions, Dr. Gesell helped establish child development as a scientific field, effectively bridging the gap between psychology and pediatrics.

Short Biography

Arnold Gesell was born on June 21, 1880, in the small town of Alma, located on the western edge of Wisconsin, on the eastern bank of the Mississippi River. Gesell's father, Gerhard, was a photographer with a studio in Alma and had a great interest in education, and Arnold's mother, Christine, was an elementary school teacher. Arnold was also the first of five children, with two brothers and two sisters. This family upbringing had a tremendous effect upon Arnold. Experiencing childhood with a family

background in education, in the presence of other children, led Arnold to be fascinated with childhood, education, and growing up, and more specifically with the experiences of those who deviated from what was considered normal. This in turn led him to study education and psychology, that he might better understand the mechanics of human development. Gesell attended the University of Wisconsin, receiving his B.Ph. in 1903, thereafter serving as principal at Chippewa Falls High School in Chippewa Falls, Wisconsin, for 1 year before attending Clark University in Worcester, Massachusetts. Here he met G. Stanley Hall, who advocated the investigation of the mental traits and abilities of children, which left a distinct impression on Gesell long after he received his Ph.D. in Education from Clark in 1906 and left to further his career. It was G. Stanley Hall's influence, perhaps more than any other, that ignited Gesell's interest in child development and which would last him the rest of his career.

In 1911, Gesell accepted an assistant professorship at Yale University, teaching courses in education at the graduate school while simultaneously enrolled at Yale Medical School. In the same year, he founded the Yale Psycho-Clinic, which is now the Yale Child Study Center. He received his M.D. in 1915, and in the years that followed began expanding his work at the Psycho-Clinic. Dr. Gesell's primary interest was intellectually disabled children; however, he realized that an understanding of what constituted "normal" was essential to understanding "abnormal." Dr. Gesell hypothesized that normal human behavior develops in a systematic, patterned way and can therefore be charted. Dr. Gesell, drawing on inspiration from his father, introduced cinematography as one of the main observational tools used for research at the Clinic. Using this and other groundbreaking tools and methods, Dr. Gesell produced the Gesell Developmental Schedules, which illustrated general patterns and stages of normal behavioral development. Arnold Gesell was also noteworthy for his extensive studies with twins and also for being vocal on issues related to education and family welfare. A prolific

writer, Dr. Gesell published numerous books and scientific articles on a variety of subjects, from social and family issues to developmental diagnosis. Dr. Gesell served as Director of the Yale Child Study Center until 1948, when he retired and became professor emeritus. Milton J.E. Senn, M. D, superseded him in his role as Director of the YCSC. He died in 1961.

See Also

► Object Permanence

References and Reading

- Ames, L. B. (1989a). *Arnold Gesell: Themes of his work*. New York: Human Sciences Press.
- Ames, L. B. (1989b). *The Arnold Gesell papers*. Washington, DC: Library of Congress.
- Gesell, A. (1930). *The guidance of mental growth in infant and child*. New York: Macmillan.
- Gesell, A. (1932). How science studies the child. *The Scientific Monthly*, 34(3), 265–267. <http://www.jstor.org/stable/15125>
- Gesell, A. (1934). *An atlas of infant behavior: A systematic delineation of the forms and early growth of human behavior patterns*. New Haven: Yale University Press.
- Gesell, A. (1940). *The first five years of life: A guide to the study of the preschool child*. New York: Harper & Brothers.
- Gesell, A. (1946). Cinematography and the study of child development. *The American Naturalist*, 80(793), 470–475. Accessed 22 Mar 2010. <http://www.jstor.org/stable/2458189>
- Gesell, A. (1952). In E. G. Boring et al. (Eds.), *A history of psychology in autobiography* (Vol. 4, pp. 123–142). Worcester: Clark University Press.
- Gesell, A., Thompson, H., & Amatruda, C. S. (1934). *Infant behavior: Its genesis and growth*. New York: McGraw-Hill.
- Gesell, A., Thompson, H., & Amatruda, C. S. (1938). *The psychology of early growth, including norms of infant behavior and a method of genetic analysis*. New York: Macmillan.
- Gesell, A., Ilg, F., & Ames, L. B. (1946). *The child from five to ten*. New York: Harper & Brothers.
- Herman, E. (2001). Families made by science: Arnold Gesell and the technologies of modern child adoption. *Isis*, 92, 684–715.
- Miles, W. R. (1964). *Arnold Lucius Gesell 1880–1961: A biographical memoir*. Washington, DC: National Academy of Sciences.

Gestural Imitation

► Body Movements, Imitation of

Gesture Form and Function in ASD

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Definition

Communicative co-speech gestures are spontaneously produced body movements (primarily hand movements) that occur during fluent speech and add both semantic and pragmatic content to communication.

Historical Background

Differences in co-speech gesture production have been clinically observed since Kanner first described autism in 1943 (► [Kanner, Leo](#)).

Current Knowledge

Impairments in nonverbal communication (including gesture) are required for a diagnosis of autism spectrum disorder (ASD), and the absence of early gestures is considered a red flag for ASD in toddlers. Despite their clear relevance to social-communication functioning, gestures produced during the context of developed, fluent speech have received limited empirical attention. This entry reviews what is known about both the *production* and *comprehension* of these *co-speech gestures* in verbally fluent children, adolescents, and adults with ASD.

Gesture Versus Speech: How Does Gesture Communicate?

Speech and gesture both convey meaning but in different, complementary formats. Speech conveys information in discrete/digital units (i.e., words) that are culturally defined and consistently used to mean the same thing across contexts (i.e., consistent semantics). Speech presents this information in a linear, sequential format (i.e., consistent syntax). In contrast, gestures are “global-synthetic: a representation that is not divided into parts but rather captures the whole of the referent” (Goldin-Meadow and Alibali 2013). Further, whereas the meaning of words is almost always arbitrarily related to their sound, the meaning of gesture is often directly related to its form (i.e., iconicity). As an example, imagine a child describing a roller coaster: “first it went up up up, then it slowed down, then we shot back down to the ground and went around and around!” In speech, this information must be conveyed one unit at a time and combined using the rules of syntax. Now picture the gestures that might accompany this description – they are likely portrayed continuously across the entire utterance, *showing* the roller coaster as a whole, and incorporating multiple features at once, including shape, speed, and direction. Note also that the child’s gestures are likely to portray information that may be absent or difficult to describe in speech. For example, in what way did the roller coaster go around and around? Was it a loop-de-loop, or was it a flat circle parallel to the ground? This information, while clunky and complicated to describe in speech, is likely to be automatically enacted in gesture as the child’s visual representation of the roller coaster spills out into their hands (i.e., Hostetter and Alibali’s “gesture as simulated action” framework). For an excellent review on the role of gesture in speaking and learning in typical development, see Goldin-Meadow and Alibali (2013).

Gesture benefits communication both by supporting and elaborating information conveyed in speech and also by engaging listeners’ attention and interest during discourse; thus, gestures alter and enhance the communicative signal, both by adding *content* to the discourse and by adding

social structure to the discourse. Although debated, most gesture theorists believe that gesture and speech are fundamentally part of the same system; thus, to truly understand gesture production and comprehension, it must be studied within the context of its integration with speech. Accordingly, the current entry reviews only research that includes both modalities, across both production and comprehension.

Gesture Production in ASD

There are at least three reasons why the field should care about understanding gesture production in ASD during developed, fluent speech: (1) co-speech gestures enhance the communicative signal, (2) co-speech gestures benefit a speakers' own cognitive processes, and (3) co-speech gestures may provide a means for elucidating qualitative differences in behavior in ASD that have been challenging to describe and quantify. These are reviewed below.

Co-speech Gestures Enhance the Communicative Signal

From the perspective of production, one way to look at how much content gesture adds to speech is simply to look at the *rate* of gestures produced over a discourse (i.e., gestures per minute/word/utterance). Gesture rate is often assumed to be reduced in ASD throughout the lifetime, because reductions in gesture are such a prominent sign of ASD in toddlers. In fact, gesture rate does *not* appear to be consistently reduced in verbally fluent children, adolescents, and adults on the autism spectrum. While a number of studies have demonstrated small reductions in gesture rate across different kinds of tasks (de Marchena and Eigsti 2014; Medeiros and Winsler 2014; Silverman et al. 2017; So et al. 2014), many studies have failed to demonstrate group differences in case-control studies using similar tasks (Attwood et al. 1988; Capps et al. 1998; de Marchena and Eigsti 2010; de Marchena et al. 2018; Hobson et al. 2010; Morett et al. 2016; So and Kit-Yi Wong 2016). Taken as a whole, the literature suggests that gesture rate may be slightly reduced in ASD, but not enough to account for the very noticeable differences in gesture production

that have long been reported by clinicians. So if rate does not adequately portray the picture of atypical gesture in ASD, let us now turn to *what* gesture communicates.

Gestures add *content* to verbal interactions in multiple ways. Gestures can add concrete visuo-spatial information to speech (e.g., imagine someone describing a tiny egg they found on a hike while holding their finger and thumb a half-inch apart). They can add emphasis or signal uncertainty. They also serve critical pragmatic functions that are often overlooked, such as linking the context of the physical environment to the contents of speech, thereby allowing listeners to make inferences about a speaker's intended meaning (e.g., imagine a speaker who says "wow, it's getting stuffy in here" while pointing at a window near you; Kelly et al. 1999).

The functions and meanings of gestures are typically quantified by categorizing gestures into distinct "types"; there are multiple systems for this. Categorizing gesture presents certain challenges, as gesture is a holistic, analog system that does not categorize easily. The first study to look at gesture production in ASD across different types of co-speech gestures (Attwood et al. 1988) found differences in the types of gestures produced; specifically, adolescents with ASD did not use *expressive* gestures (i.e., gestures making reference to emotions or other internal states), but were comparable to controls in their use of pointing and instrumental gestures. People with ASD may use more *iconic* gestures (i.e., gestures depicting physical aspects of a referent, such as shape, size, or motion; "descriptive" gestures on the ADOS) than controls (Morett et al. 2016; So and Kit-Yi Wong 2016), perhaps because enacting experiences is more accessible than verbalizing them (Capps et al. 1998). However, not all studies find an increased rate of iconic gesture production in ASD, and some studies find no difference in gesture types at all (de Marchena and Eigsti 2010; Silverman et al. 2017).

In addition to conveying specific content, gestures also add *social structure* to the discourse. For example, gestures may be used to cite another person's contribution to the conversation (e.g., an open palm moving toward an interlocutor could

be translated as, “like you said earlier”); to signal agreement, disagreement, hesitation, or emphasis; and to regulate turn-taking dynamics (e.g., “you go ahead,” or “I’m still talking”). Thus, gesture helps structure the interaction itself. Autistic adults have been found to use these “interactive gestures” (Bavelas et al. 1992) *more often* than controls, particularly to help regulate conversational turn-taking, suggesting again that gesture may be a particularly accessible communicative modality (de Marchena et al. 2018).

Gesture can also structure a conversational exchange through the use of *anaphor*, in which a physical area within the gesture space is used to consistently refer to a specific referent within a discourse (e.g., when talking about two friends, whenever I talk about *Sadie*, I always gesture toward my upper right, but when talking about *Oliver*, I always gesture toward my upper left). During an explanation task, children with ASD were found to be internally consistent in their own use of anaphor; however, when an examiner suggested specific locations for referents by using anaphor herself (e.g., the toothbrush is on the right, the toothpaste is on the left), children with ASD were less likely to adopt the spatial locations indicated by the examiner (So and Kit-Yi Wong 2016) relative to controls. This suggests weaknesses in attending to others’ gestures and subsequently incorporating that information into one’s own communication, which could easily lead to communication breakdowns. On the other hand, there is some evidence that individuals with ASD intend for at least some of their own gestures to be communicative. Like controls, adolescents with ASD produce more gestures when in the presence of a visible listener (Morett et al. 2016), suggesting that they recognize the communicative benefits of gesture and do not simply gesture to facilitate their own cognitive processes.

Co-speech Gestures Benefit a Speakers’ Own Cognitive Processes

In addition to its social-communicative power, gesture also confers a range of benefits for speakers themselves. Some of these benefits relate directly to communication: for example, gesturing

can help speakers plan and organize the contents of a story. The act of gesturing can also help facilitate word finding; indeed, when speakers are prevented from gesturing, they are less likely to recover from tip-of-the-tongue experiences. In addition to these communication-relevant benefits, gesture also facilitates cognitive processes that are relatively independent of communication, such as working memory and spatial reasoning. Strikingly, gesture can cause knowledge change – either directly (the act of gesturing can cause cognitive change, such as learning a new mathematical concept) or socially (gesture can convey information about a speaker’s readiness to learn and current knowledge state, allowing teachers or therapists to tailor their instruction in the moment). In the only study to probe gesture’s benefits on cognition in ASD, gestures produced by adolescents with ASD were measured during both a storytelling task and an executive function task. Teens with ASD produced fewer gestures than controls during the storytelling task (in which gestures serve a range of social-communication functions) but *more* gestures during the executive function task, in which gestures are more likely to support cognitive processes such as visual attention and concentration (de Marchena and Eigsti 2014). These findings suggest that gesture *does* convey cognitive benefits for speakers with ASD.

Co-speech Gestures May Elucidate Qualitative Differences in Behavior in ASD

Finally, the way in which co-speech hand gestures are produced by speakers may provide an assay for measuring qualitative features of behavior that have thus far proved difficult to quantify in ASD. Per DSM-5 (American Psychiatric Association 2013) criteria, nonverbal communication in ASD is defined both in terms of *quantity* (i.e., “total lack of...”) and *quality* (i.e., “abnormalities in...”). As reviewed above, by many concrete/objective measures, gesture production in ASD is quite similar to gesture production by controls: gestures are produced at a similar rate and for roughly the same purposes. Yet this similarity is quite inconsistent from clinical impressions, which often report large differences in how people

with ASD gesture, and, accordingly, with clinical measures. Quantity of nonverbal communication is relatively well-characterized on ASD questionnaires. The Autism Diagnostic Observation Schedule (► [Autism Diagnostic Observation Schedule](#)), a clinical observational tool, prompts clinicians to attend to both quality and quantity of different gesture types. However, the specific ways in which gestures might differ qualitatively in ASD have yet to be adequately characterized empirically.

Two studies have looked at how gesture production affects overall communication quality. de Marchena and Eigsti (2010) recorded adolescents with ASD telling the “Monkey” cartoon from the ADOS, in which participants were prompted to tell a story based on six black-and-white line drawings. They then played recordings of these adolescents to diagnosis-naïve college students either with or without video (i.e., audio only). Controls were rated as telling higher quality stories in the video condition (i.e., when raters could see them); in contrast, teens with ASD were rated as telling higher-quality stories in the audio-only condition. This suggests that something about the gestures, facial expressions, or general movements produced by autistic teens may have been distracting to observers, whereas these behaviors benefited communication in controls. Similarly, Morett and colleagues (2016) found that quality ratings for stories produced by teens with and without ASD differed when participants were speaking to a visible interlocutor, but did not differ when told to a hidden interlocutor (and participants were therefore using fewer non-verbals). Morett and colleagues further found that features of speech, gesture, and speech-gesture combinations predicted communication quality in controls, whereas *only speech* predicted communication quality in teens with ASD, suggesting once again that gesture does not benefit communication to the same extent in ASD.

As described above, gestures are often used at the same rate, and for similar communicative functions, in ASD. So why are they not benefiting communication to the same extent? Several studies have now looked at the quality of each individual gesture produced within a discourse and

consistently found large group differences in children, adolescents, and adults on the spectrum (de Marchena et al. 2018; Hobson et al. 2010; Hobson and Lee 1998; Silverman et al. 2017). Group differences have been identified in terms of (1) the physical form of the gesture (Hobson et al. 2010; Hobson and Lee 1998), (2) the clarity of the gesture as a communicative act (i.e., whether a movement was a gesture or not; Silverman et al. 2017), and (3) the intended meaning or purpose of the gesture (de Marchena et al. 2018; Silverman et al. 2017). Further supporting the claim that qualitative features of gesture may better describe ASD, Silverman and colleagues (2017) found that qualitative features of gesture predicted clinical measures (e.g., ADOS scores), whereas gesture rate did not. Further research is needed to understand how these differences may be related within individuals with ASD (e.g., are atypically formed gestures harder to identify as acts of communication)?

So what about gesture in ASD leads to reduced quality and, in turn, less communicative power? More research is needed to answer this question and to point to new possible targets for intervention. One possibility is that gestures are less well-integrated into speech, making it less clear that gesture and speech represent a unified package of meaning. In fact, gesture and speech are less temporally synchronous in ASD (de Marchena and Eigsti 2010; Morett et al. 2016), and the degree of asynchrony between speech and gesture predicts observers’ ratings of communication quality (de Marchena and Eigsti 2010). Observed differences in speech-gesture synchrony are consistent with the growing literature on difficulties with multimodal integration in ASD. Second, atypicalities in gesture may be secondary to motor skills differences in ASD that are now widely understood to be part of the behavioral phenotype. Specifically, difficulties with *praxis* (► [Praxis](#)), or the ability to perform skilled movements, are strongly theoretically linked to co-speech gesture production, particularly when considering gestures that involve motor enactment of visuospatial features, such as shape or motion. The relationship between gesture and praxis, or motor behavior in general, has not yet been directly tested.

Hobson and Lee (1998), who reported a decreased rate and quality of spontaneous greeting/farewell waves in ASD, informally described the waving as “ill-directed, perfunctory, or clumsy and limp” (Hobson and Lee 1998, p. 124), consistent with a wide range of possible social-motor differences and consistent with stakeholder reports that gestures in ASD are often produced in surprising or odd ways (e.g., pointing with the middle finger, which can run the risk of the occasional faux pas!).

Gesture Comprehension in ASD

For neurotypical individuals, gestures facilitate comprehension both by enhancing information already conveyed in speech and by communicating information that goes beyond speech. The small literature on gesture comprehension in ASD supports the conclusion that bimodal processing (i.e., comprehension of speech-gesture combinations) is slowed and inefficient in ASD, despite relatively intact accuracy on structured tasks. People with ASD thus may benefit less from gestures during spontaneous conversation, even if they are able to parse gestures themselves when given enough time. For example, when given instructions presented in both speech and gesture, both adolescents (Silverman et al. 2010) and adults (Canfield et al. [under review](#)) with ASD are slower to process these speech-gesture combinations compared to controls. This effect is particularly strong when the information presented in gesture is incongruent with the information in speech (Canfield et al. [under review](#)), suggesting that autistic adults may prioritize the verbal modality over the gestural modality. However, slowed processing is found even when speech and gesture present the same information, for example, saying “find the loop,” while tracing a loop with a finger. In fact, controls tend to be faster to respond when presented with semantically congruent information across speech and gesture, whereas teens with ASD respond more quickly when information is presented in the audio channel only. These findings suggest that individuals with ASD are slowed by the additional processing demands of incorporating gestural information (Silverman et al. 2010), even when

this information is semantically identical to speech. Neural processing of communicative co-speech gestures also appears to be atypical in ASD. In the only fMRI study of co-speech gesture in ASD (Hubbard et al. 2009), participants passively viewed videos of people talking either with or without accompanying beat gestures (i.e., gestures conveying emphasis, with little to no specific semantic content). Typical children showed increased activation of the superior temporal sulcus while viewing beat gestures, suggesting that they interpreted these movements as communicative; children with ASD, on the other hand, increased visual cortex activation, suggesting that while they viewed the movements as visually salient, they did not automatically interpret them as a signal relevant to communication.

These differences in gesture processing, which have been observed across measures of reaction time, eye tracking, and fMRI signal, are in sharp contrast to gesture comprehension *accuracy* in ASD on behavioral tasks, which is generally strong. Children with ASD are able to understand deictic, iconic/descriptive (Dimitrova et al. 2017), and emblems/conventional gestures (Attwood et al. 1988) on structured tasks. Likewise, adults with ASD are as accurate as controls at identifying whether an iconic/descriptive gesture matches a target action (e.g., chopping; Canfield et al. [under review](#)). Comprehension accuracy may be reduced when gestures are unexpected or atypical (e.g., nodding the head toward an object instead of pointing at it; Hobson et al. 2010). A major limitation of the comprehension literature thus far is that it has been conducted exclusively with relatively simple, trial-by-trial, highly structured tasks. To date, just one study provides a hint at what gesture comprehension in ASD looks like in a more naturalistic setting. Using a problem-solving task that parents and children completed together, Medeiros and Winsler (2014) found that in controls, parents gestured more when their kids weren’t as good at the task, but this relationship not observed in ASD. This suggests that parents of neurotypical children perceive that their kids benefit more from nonverbal communication support, which is not assumed by parents of children with ASD.

Future Directions

As reviewed above, by many concrete/objective measures, gesture production in ASD is quite similar to gesture production by controls: gestures are produced at a similar rate, and for roughly the same purposes. This is quite inconsistent from clinical impressions and established clinical instruments, which often report large differences in how people with ASD gesture. In contrast, qualitative differences in how gesture is performed or integrated into conversation are robust; understanding the nuance of these differences will lead to a richer understanding of communication in ASD. One potential starting point is testing the relationship between gesture production and motor skills in ASD.

With respect to comprehension, much more information is needed to truly understand how people with ASD process and understand gestures in the real world. Accuracy may be relatively high in ASD, when there is enough time – and when gestures are tagged by an experimenter as semantically relevant. However, the real world moves fast, and observed inefficiencies in processing suggest that gesture may often be left behind. Ultimately, we must understand and intervene at the level of the real world; thus, naturalistic research is needed to push the field forward and pave the way for interventions to improve non-verbal communication in verbally fluent individuals on the autism spectrum.

See Also

- [Autism Diagnostic Observation Schedule](#)
- [Kanner, Leo](#)
- [Nonverbal Communication](#)
- [Praxis](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. Arlington: American Psychiatric Publishing.
- Attwood, A., Frith, U., & Hermelin, B. (1988). The understanding and use of interpersonal gestures by autistic and Down's syndrome children. *Journal of Autism and Developmental Disorders*, 18, 241–257.
- Bavelas, J. B., Chovil, N., Lawrie, D. A., & Wade, A. (1992). Interactive gestures. *Discourse Processes*, 15(4), 469–489.
- Canfield, A., Castelluccio, B., & Eigsti, I. M. (under review). *Comprehension of gestures in adults with autism spectrum disorder: Gesture-speech incongruence*.
- Capps, L., Kehres, J., & Sigman, M. (1998). Conversational abilities among children with autism and children with developmental delays. *Autism*, 2, 325–344.
- de Marchena, A., & Eigsti, I. M. (2010). Conversational gestures in autism spectrum disorders: Asynchrony but not decreased frequency. *Autism Research*, 3(6), 311–322.
- de Marchena, A., & Eigsti, I. M. (2014). Context counts: The impact of social context on gesture rate in verbally fluent adolescents with autism spectrum disorder. *Gesture*, 14(3), 375–393.
- de Marchena, A., Kim, E. S., Bagdasarov, A., Parish-Morris, J., Maddox, B. B., Brodtkin, E. S., & Schultz, R. T. (2018). Atypicalities of gesture form and function in autistic adults. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-018-3829-x>.
- Dimitrova, N., Özçalışkan, Ş., & Adamson, L. B. (2017). Do verbal children with autism comprehend gesture as readily as typically developing children? *Journal of Autism and Developmental Disorders*, 47(10), 3267–3280. <https://doi.org/10.1007/s10803-017-3243-9>.
- Goldin-Meadow, S., & Alibali, M. W. (2013). Gesture's role in speaking, learning, and creating language. *Annual Review of Psychology*, 64(1), 257–283. <https://doi.org/10.1146/annurev-psych-113011-143802>.
- Hobson, R. P., & Lee, A. (1998). Hello and goodbye: A study of social engagement in autism. *Journal of Autism and Developmental Disorders*, 28, 117–127.
- Hobson, R. P., García-Pérez, R. M., & Lee, A. (2010). Person-centred (deictic) expressions and autism. *Journal of Autism and Developmental Disorders*, 40(4), 403–415. <https://doi.org/10.1007/s10803-009-0882-5>.
- Hubbard, A. L., Wilson, S. M., Callan, D. E., & Dapretto, M. (2009). Giving speech a hand: Gesture modulates activity in auditory cortex during speech perception. *Human Brain Mapping*, 30(3), 1028–1037.
- Kelly, S. D., Barr, D. J., Church, R. B., & Lynch, K. (1999). Offering a hand to pragmatic understanding: The role of speech and gesture in comprehension and memory. *Journal of Memory and Language*, 40(4), 577–592. <https://doi.org/10.1006/jmla.1999.2634>.
- Medeiros, K., & Winsler, A. (2014). Parent-child gesture use during problem solving in autistic spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(8), 1946–1958. <https://doi.org/10.1007/s10803-014-2069-y>.

- Morett, L. M., O'Hearn, K., Luna, B., & Ghuman, A. S. (2016). Altered gesture and speech production in ASD detract from in-person communicative quality. *Journal of Autism and Developmental Disorders*, 46(3), 998–1012. <https://doi.org/10.1007/s10803-015-2645-9>.
- Silverman, L. B., Bennetto, L., Campana, E., & Tanenhaus, M. K. (2010). Speech-and-gesture integration in high functioning autism. *Cognition*, 115(3), 380–393. <https://doi.org/10.1016/j.cognition.2010.01.002>.
- Silverman, L. B., Eigsti, I.-M., & Bennetto, L. (2017). I tawt i taw a puddy tat: Gestures in canary row narrations by high-functioning youth with autism spectrum disorder: Gesture production in ASD. *Autism Research*. <https://doi.org/10.1002/aur.1785>.
- So, W.-C., & Kit-Yi Wong, M. (2016). I use my space not yours: Use of gesture space for referential identification among children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 26, 33–47. <https://doi.org/10.1016/j.rasd.2016.03.005>.
- So, W.-C., Wong, M. K.-Y., Lui, M., & Yip, V. (2014). The development of co-speech gesture and its semantic integration with speech in 6- to 12-year-old children with autism spectrum disorders. *Autism*. <https://doi.org/10.1177/1362361314556783>.

Gestures

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Synonyms

[Nonsymbolic communication](#)

Definition

Gestures are actions produced with the intent to communicate. Gestures can involve fingers, hands, arms, facial features, or the entire body (Iverson and Thal 1998). When these actions are performed with the purpose of affecting another person's behavior, they are considered to be intentional communication acts (Bates 1976). Intentional use of gestures develops before speech and continues to develop as the child's speech emerges (Iverson and Thal 1998).

Gestures can be organized according to their purpose or communicative function. Bruner (1981) classified gestures according to three broad categories based on functions: social interaction, behavior regulation, and joint attention. Differences have been found in the gesture development of children later diagnosed with autism spectrum disorders (ASD).

Social interaction gestures are used to initiate or maintain the attention of another person in a social experience. These gestures might include waving "hi" or "bye," requesting games or routines, and shaking the head for "yes" or "no." In their sample of 9–12-month-old infants, Colgan et al. (2006) examined the emergent use of social interaction gestures in infants later diagnosed with autism. They found that types of gestures used were strongly associated with autism status, suggesting that the examination of the number of different gestures in a child's repertoire may be useful for detecting impairments in their language development.

Behavior regulation gestures manage the behavior of others, such as requesting an object, getting another person to do something, or getting another person to stop doing something. In studies of the communicative functions of gestures, children with autism show relative strengths in the use of gestures serving the function of behavior regulation at preschool age (Mundy et al. 1990) and at school age (Wetherby and Prutting 1984).

Gestures used to establish joint attention direct another person's attention to an object or event and usually include manual pointing or making eye contact with an object. Children with ASD demonstrate significant weaknesses in the use of gestures for the purpose of joint attention (directing another's attention or sharing interest with another person) both at preschool age (Mundy et al. 1990) and school age (Loveland and Landry 1986).

See Also

- [Augmentative and Alternative Communication \(AAC\) Device](#)
- [Nonverbal Communication](#)

References and Reading

- Acredolo, L. P., & Goodwyn, S. W. (1985). Symbolic gesturing in language development. *Human Development*, 28, 40–49.
- American Psychiatric Association. (2000). Pervasive developmental disorders. In *Diagnostic and statistical manual of mental disorders* (4th ed., pp. 69–70). Washington, DC: American Psychiatric Association. Text revision (DSM-IV-TR).
- Bates, E. (1976). *Language and context. The acquisition of pragmatics*. New York: Academic.
- Bruner, J. (1981). The social context of language acquisition. *Language and Communication*, 1(2/3), 155–178.
- Colgan, S. E., Lanter, E., McComish, C., Watson, L. R., Crais, E. R., & Baranek, G. T. (2006). Analysis of social interaction gestures in infants with autism. *Child Neuropsychology*, 12, 307–319.
- Crais, E. (2008). *Using the best available evidence to identify infants and toddlers with (or at risk for) communication deficits* (presentation). Retrieved on December 18, 2010 from Telability's website: <http://www.telability.org/search/search.pl>
- Crais, E., Douglas, D. D., & Campbell, C. C. (2004). The intersection of the development of gestures and intentionality. *Journal of Speech, Language, and Hearing Research*, 47, 678–694.
- Iverson, J. M., & Thal, D. J. (1998). Communicative transitions: There's more to the hand than meets the eye. In A. M. Wetherby, S. F. Warren, & J. Reichle (Eds.), *Transitions in prelinguistic communication* (pp. 59–86). Baltimore: Brookes.
- Landa, R. J., Holman, K. C., & Garrett-Mayer, E. (2007). Social and communication development in toddlers with early and later diagnosis of autism spectrum disorders. *Archives of General Psychiatry*, 64, 853–864.
- Loveland, K., & Landry, S. H. (1986). Joint attention and language in autism and developmental language delay. *Journal of Autism and Developmental Disorders*, 16, 335–349.
- McDuffie, A., Yoder, P., & Stone, W. (2005). Prelinguistic predictors of vocabulary in young children with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, 48, 1080–1097.
- Mundy, P., Sigman, M., & Kasari, C. (1990). A longitudinal study of joint attention and language development in autistic children. *Journal of Autism and Developmental Disorders*, 20, 115–128.
- Sigman, M., & Casari, C. (1995). Joint attention across contexts in normal and autistic children. In C. Moore & P. J. Dunham (Eds.), *Joint attention: Its origins and role in development* (pp. 189–204). Hillsdale: Laurence Erlbaum.
- Thal, D., & Tobias, S. (1992). Communicative gestures in children with delayed onset of oral expressive vocabulary. *Journal of Speech and Hearing Research*, 35, 1281–1289.
- Thal, D., & Tobias, S. (1994). Relationships between language and gesture in normal and late-talking toddlers. *Journal of Speech and Hearing Research*, 37, 157–171.
- Tomasello, M. (1995). Joint attention as social cognition. In C. Moore & P. J. Dunham (Eds.), *Joint attention: Its origins and role in development* (pp. 103–130). Hillsdale: Laurence Erlbaum.
- Werner, E., & Dawson, G. (2005). Validation of the phenomenon of autistic regression using home videotapes. *Archives of General Psychiatry*, 62, 889–895.
- Wetherby, A., & Prutting, C. (1984). Profiles of communicative and cognitive-social abilities in autistic children. *Journal of Speech and Hearing Research*, 27, 364–377.
- Wetherby, A. M., Watt, N., Morgan, L., & Shumway, S. (2007). Social communication profiles of children with autism spectrum disorders late in the second year of life. *Focus on Autism and Other Developmental Disorders*, 37, 960–975.
- Williams, J., Whiten, A., & Singh, T. (2004). A systematic review of action imitation in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 34, 285–299.

GI Issues

► Gastrointestinal Disorders and Autism

Giftedness

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Definition

Giftedness refers to high values in the distribution of skills fundamental to human accomplishments. Intelligence, talent, and creativity are neighboring but distinct notions. Whereas domain-general giftedness partially overlaps with the notion of *intelligence*, domain-specific giftedness calls its nature into question, and there is no single

intelligence-test-based threshold which consensually defines giftedness. *Talent* describes the outcome of giftedness in actual accomplishments, which depends on a combination of giftedness, training, and more or less optimal conditions for the acquisition of expertise. *Creativity* designates the capacity for innovation, which may or may not be associated with giftedness, and does not overlap with intelligence. Giftedness includes a genetic and/or innate component in the form of an overrepresentation of giftedness within some families or in the form of genetic variation favoring atypical information-processing abilities.

The association between giftedness and autism is multifaceted. First, giftedness has been associated with a certain number of psychological particularities, including an overattention and emotional reactivity to the surrounding world, a tendency to interact with older peers, indifference toward social conventions, etc. Some of these particularities overlap with the autistic phenotype and may be difficult to separate. Second, the distribution curve of autistic intelligence features higher variance than that of nonautistic individuals, such that some autistics present extreme values of general intelligence, or “double exceptionality.” Third, autistic peaks of ability, or domains where autistics as a group perform at a superior level than their IQ (as measured with certain instruments) would have predicted, represent a “group giftedness” in the form of an innate advantage for certain cognitive operations. Peaks of ability represent the main element which makes the giftedness versus autism distinction difficult in clinical settings. For example, an advance of 2 years in word-decoding skills may announce an autism diagnosis, giftedness without autism, or a combination of both. Finally, savant syndrome is strongly associated with autism and is defined by the presence of exceptional performance in neurodevelopmental variants. All possible combinations of giftedness, intelligence, talent, and autism may be manifested in savant syndrome. The range of tasks for which savants display giftedness is variable, with some savants possessing multiple areas of excellence, combined or not with deficits in other areas.

The degree, adaptive value, recognition by others, eventual outcomes, etc., of talent arising

from giftedness in autism all vary to the extreme.

See Also

- [Autistic Savants](#)
- [Enhanced Perceptual Functioning](#)
- [Psychological Assessment](#)

References and Reading

- Mackintosh, N. J. (2011). *IQ and human intelligence* (2nd ed.). New York: Oxford University Press.
- Miller, L. K. (1999). The savant syndrome: Intellectual impairment and exceptional skill. *Psychological Bulletin*, 125, 31–46.
- Mottron, L., Dawson, M., & Soulières, I. (2009). Enhanced perception in savant syndrome: Patterns, structure and creativity. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 364, 1385–1391.
- Nicpon, M. F., Allmon, A., Sieck, B., & Stinson, R. D. (2011). Empirical investigation of twice-exceptionality: Where have we been and where are we going? *Gifted Child Quarterly*, 55, 3–17.
- Obler, L. K., & Fein, D. (Eds.). (1988). *The exceptional brain: Neuropsychology of talent and special abilities*. New York: Guilford.

Gilles de la Tourette Syndrome

- [Tourette Syndrome](#)
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Gilliam Autism Rating Scale (GARS)

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Synonyms

[GARS](#); [GARS-2](#); [Gilliam Autism Rating Scale, Second Edition](#); [Gilliam Autism Rating Scale, Third Edition](#)

Description

The Gilliam Autism Rating Scale is a standardized instrument for the assessment and diagnosis of autism and other behavioral conditions (GARS). It relies on parental or teacher reports regarding the child's presentation and behavior and is a quick measure to administer. Furthermore, no significant training is necessary. Nonetheless, some studies suggest a tendency to "miss" children who would otherwise meet the diagnostic criteria for an autism spectrum disorder (Lecavalier 2005; South et al. 2002).

The scale was revised (GARS-2) in 2006 and based on the *DSM-IV-TR* (American Psychiatric Association 2000) and *Autism Society of America* definitions of autism. The core items (42) on the three subscales were retained in the revised version, although some minor changes in wording were made.

The GARS-2 relies on parental rating of directly observed behaviors and developmental history of delay or abnormality.

The instrument is comprised of three subscales, namely, *Stereotyped Behaviors*, *Communication*, and *Social Interaction*. Instructions are straightforward and require the clinician/parent to rate a number of items according to the frequency with which the behaviors occur. Ratings are presented in a Likert style, from 0 (never observed), 1 (seldom observed), 2 (sometimes observed), to 3 (frequently observed). Additional guidance/example is provided for each rating, i.e., sometimes observed – individual behaves in this manner 3–4 times per 6-h period. Behavior is rated under typical circumstances, for instance, in the presence of familiar people, in usual places, and during day-to-day activities. Scores are summed to yield a total raw score for *Stereotyped Behaviors*.

The *Communication Subscale* and *Social Interaction Subscale* are completed in the same manner, i.e., on the basis of behaviors observed by the parent/caregiver. However, if the individual does not have speech and does not sign or use other forms of communication, the former is omitted. A total raw score is derived by summing the ratings on each subscale (14 items each). Raw scores on individual subscales are converted to

standard scores and represented as percentiles. Total standard scores for all 42 items yield a sum of standard scores. This is represented as an Autism Index and corresponds to a given percentile. The response booklet allows the plotting of the individual's profile. Furthermore, interpretation of standard scores and the Autism Index yields three possible outcomes with respect to a diagnosis of autism: *very likely*, *possibly*, and *unlikely*.

Information from parental interviews relates specifically to *delays* and *abnormal functioning*. This data may be gathered at interview or be completed by a parent directly. Responses take the form of *yes* or *no*. The questions relate to delays in social interaction and language used in social communication, specifically during the first 3 years of life. Abnormal functioning within the first 3 years of life in social interaction, language used in social communication, and symbolic or pretend play is assessed in the subsequent section. A *no* response to any of these questions indicates that the individual meets the *DSM-IV-TR* criteria for *delays* or *abnormal functioning*.

Additional questions are presented in the response form for the benefit of the clinician when reflecting on the GARS-2 results and considering the diagnostic outcome.

The measure was revised again in 2013 (GARS-3) with items reflecting the updated autism diagnostic criteria (DSM-5) (American Psychiatric Association 2013). A further four items have been included, and the GARS-3 is comprised of six subscales (total of 58 items), viz., Restricted/Repetitive Behaviors (13), Social Interaction (14), Social Communication (9), Emotional Responses (8), Cognitive Style (7), and Maladaptive Speech (7). The examiner/practitioner can use the interpretation guide to score and conclude probability and severity of ASD. DSM-5 criteria are included in a diagnostic validation form to confirm that criteria are met.

Historical Background

A number of screening and diagnostic measures for autism spectrum disorders have been developed in the past two decades. The Gilliam Autism

Rating Scale (GARS, Gilliam 1995) was initially developed as a screening measure to identify individuals with autism in research and clinical settings. Although it was widely used, there was little empirical evaluation of the measure and its usefulness (Lecavalier 2005; Mazefsky and Oswald 2006; Pandolfi et al. 2010; Norris and Lecavalier 2010).

Gilliam 2006; Eaves et al. 2006; Schopler et al. 1980; and Ehlers and Gillberg 1993 revised the original instrument retaining most of the items on the three subscales but adding a section (25 items) relating to delays or abnormalities during the first 3 years of life. The scoring system was changed, and the total score, namely, the Autism Quotient (AQ), was replaced by an Autism Index (AI).

The purpose of the GARS-3 is to assist teachers, parents, and clinicians in identifying and diagnosing autism in individuals from 3 to 22 years of age. Furthermore, consistent with GARS-2, the revised tool aims to provide information regarding *severity* of the condition. The GARS-3 may also be used to rate change, and the manual provides examples of discreet target behaviors for all items which may be useful for research.

Psychometric Data

Normative data for the GARS-2 were presented by Gilliam (2006). The scale was tested on a sample of 1,107 autistic children and adolescents and compared with scores of children with intellectual disabilities and those without any disabilities. The author reported that the measure successfully screened for autism with the children in the autism group having higher Autism Index (AI) scores than the children in the other groups. A cutoff score of 85 was selected for the AI, and the author reports sensitivity, specificity, and positive predictive values of 0.85 in the intellectual disability group. Reported values were 0.84 for the multiple disability group. The sensitivity of 1.0, specificity of 0.87, and positive

predictive value of 0.85 were reported in the control group.

The author reported internal consistency for the subscales (Cronbach's alpha): Stereotyped Behaviors (0.84), Communication (0.86), Social Interaction (0.88), and 0.94 for the Autism Index. The author reported that concurrent and positive predictive validity had been established (Gilliam 2006).

However, independent studies of the GARS/GARS-2 have not replicated these findings and have expressed concern about the nature of the normative sample and the poor sensitivity of the measure, hence missing a number of children on the spectrum (Lecavalier 2005; Mazefsky and Oswald 2006; Sikora et al. 2008; South et al. 2002). While Eaves et al. (2006) established more positive results for the sensitivity (0.83) and specificity (0.68), these were still significantly lower than the results published by the author. Pandolfi et al. (2010) examined the validity of the GARS-2 and were unable to match the psychometric properties previously described by the GARS' authors.

The authors report good psychometric properties for the GARS-3 (2014) with the measure accurately discriminating autistic children from non-autistic children (sensitivity = 0.97, specificity = 0.97, ROC/AUC = 0.93). Subscale internal consistency reliability coefficients are reported to exceed 0.85 (content sampling), and test-retest reliability coefficients (time sampling) are reported to exceed 0.80 for the subscales and 0.90 for the Autism Indexes (AI). Furthermore, the authors report interrater reliability intraclass coefficients above 0.80 and 0.84 for the AI.

Karren (2017) reviewed the GARS-3 and described improved properties, including the DSM-5-based items and an instructional goals booklet to assist in planning for intervention. The author expresses concern around interpretation of results for the older group (20–22 years) and those from ethnic minority groups. Hence, further data are needed regarding its reliability and validity in broader clinical practice.

Clinical Uses

The GARS-2 is a revision of the GARS and aims to assist in the screening and diagnosis of autism in individuals from the age of 3 years to 22 years. It is a quick and inexpensive means of gathering information from parents, teachers, or caregivers taking between 5 and 10 min to complete. The revised version also aims to provide an indication of severity of the condition and provides the possibility of detecting change over time with respect to discreet behaviors.

There are currently a number of screening measures and rating scales for autism spectrum disorders. These may differ with respect to their focus, strengths, and weaknesses but with the common goal of quantifying autistic features and assisting in the diagnostic process (Lecavalier 2005; Thabtah and Peebles 2019). While some instruments are designed to be administered by a clinician or trained rater, such as the ADI-R (Lord et al. 1994), the ADOS-2 (Lord et al. 2000), and the Childhood Autism Rating Scale (CARS, Schopler et al. 1980), others rely on parental and teacher ratings of behaviors. The ADOS-2 and CARS-2 ratings are also based on direct observations. While widely used in clinics and research, these measures are time intensive for professionals.

Standardized, informant-rated scales, such as the Childhood Autism Spectrum Test (CAST, Scott et al. 2002), the Social Communication Questionnaire (SCQ, Rutter et al. 2003), and the Autism Spectrum Screening Questionnaire (ASSQ, Ehlers and Gillberg 1993), can provide an alternative method of gathering information in an easy and efficient manner. Screening measures also differ with respect to their respective levels. The SCQ, GARS, and GARS-2 are presented as level 2 instruments, i.e., they are used with individuals who are already identified as being at risk for some developmental disabilities and aim to differentiate those with a possible autism spectrum disorder and those with other kinds of

conditions. However, a number of studies have demonstrated concerns about the GARS and GARS-2 with respect to clinical utility owing to psychometric properties.

The GARS and GARS-2 may be useful as a general screening tool rather than a primary tool in the screening and assessment of autism spectrum disorder. The GARS-3 offers more robust information to assist in assessment of ASD but may need further examination regarding its utility in particular groups.

See Also

- ▶ [ADI-R](#)
- ▶ [Childhood Autism Rating Scale](#)
- ▶ [Clinical Assessment](#)
- ▶ [Diagnostic Process](#)
- ▶ [Screening Measures](#)

References and Reading

- Akshoomoff, N., Corsello, C., & Schmidt, H. (2006). The role of the autism diagnostic observation schedule in the assessment of autism spectrum conditions in school and community. *California School Psychologist*, 11, 7–19.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Eaves, R.C., Woods-Groves, S., Williams, Jr, T.O., & Fall, A. (2006). Reliability and validity of the Pervasive Developmental Disorders Ratings Scale and the Gilliam Autism Rating Scale. *Education and training in developmental disabilities*, 41, 300–309.
- Ehlers, S., & Gillberg, C. (1993). The epidemiology of Asperger Syndrome. A total population study. *Journal of child psychology and psychiatry*, 34, 1327–1350.
- Gilliam, J. E. (1995). *Gilliam autism rating scale*. Austin: PRO-ED.
- Gilliam, J. E. (2006). *Gilliam autism rating scale-2*. Austin: PRO-ED.
- Gilliam, J. E. (2014). *Gilliam autism rating scale* (3rd ed.). Austin: Pro-Ed.

- Howlin, P. (2000). Autism and intellectual disability: Diagnostic and treatment issues. *Journal of the Royal Society of Medicine*, 93, 351–355.
- Karren, B. C. (2017). A test review: Gilliam, J. E. (2014). Gilliam Autism Rating Scale—Third Edition (GARS-3). *Journal of Psychoeducational Assessment*, 35(3), 342–346. <https://doi.org/10.1177/0734282916635465>.
- Lecavalier, L. (2005). An evaluation of the Gilliam Autism Rating Scale. *Journal of Autism and Developmental Disorders*, 35, 795–805.
- Lord, C., & Rutter, M. (2012). *Autism diagnostic observation schedule* (2nd ed.). Los Angeles: Western Psychological Services.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24, 659–685.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (1999). *Autism diagnostic observation schedule*. Los Angeles: Western Psychological Services.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., DiLavore, P. C., et al. (2000). Autism diagnostic observation schedule – Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30, 205–223.
- Mazefsky, C. A., & Oswald, D. P. (2006). The discriminative ability and diagnostic utility of the ADOS-G, ADI-R, and GARS for children in a clinical setting. *Autism*, 10(6), 533–549.
- Norris, M., & Lecavalier, L. (2010). Screening accuracy of level 2 autism spectrum disorder rating scales. A review of selected instruments. *Autism*, 14(4), 263–284.
- Pandolfi, V., Magyar, C. I., & Dill, C. A. (2010). Constructs assessed by the GARS-2: Factor analysis of data from the standardization sample. *Journal of Autism and Developmental Disorders*, 40(9), 1118–1130.
- Rutter, M., Bailey, A., & Lord, C. (2003). *SCQ: Social communication questionnaire*. Los Angeles: Western Psychological Services.
- Schopler E., Reichler R. J., DeVellis R. F., & Daly K. (1980). Toward objective classification of childhood autism: Childhood Autism Rating Scale (CARS). *Journal of autism and developmental disorders*, 10, 91–103.
- Scott, F., Baron-Cohen, S., Bolton, P., & Brayne, C. (2002). The CAST (Childhood Asperger Syndrome Test): Preliminary development of a UK screen for mainstream primary-school age children. *Autism*, 6(1), 9–31.
- Sikora, D. M., Hall, T. A., Hartley, S. L., Gerrard-Morris, A. E., & Cagle, S. (2008). Does parent report of behavior differ across ADOS-G classifications: Analysis of scores from the CBCL and GARS. *Journal of Autism and Developmental Disorders*, 38(3), 440–448.
- South, M., Williams, B. J., McMahon, W. M., Owley, T., Filipek, P. A., et al. (2002). Utility of the Gilliam autism rating scale in research and clinical populations. *Journal of Autism and Developmental Disorders*, 32(6), 593–599.
- Thabtah, F., & Peebles, D. (2019). Early autism screening: A comprehensive review. *International Journal of Environmental Research and Public Health*, 16, 3502.

Gilliam Autism Rating Scale, Second Edition

► Gilliam Autism Rating Scale (GARS)

Gilliam Autism Rating Scale, Third Edition

► Gilliam Autism Rating Scale (GARS)

Girls Night Out Model, The

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Definition

Girls Night Out (herein referred to as *GNO*) is a social skills and self-care program designed to address the unique needs of adolescent females with ASD/DD with goals to improve their social-emotional health. GNO is a manualized intervention program that incorporates empirically based strategies and gathers data on program outcomes resulting in a meaningful, socially valid intervention for participants. The program is also unique in that it incorporates typically developing peers as mediators of the strategies, providing socially valid cues and reinforcement for use of skills. Sessions take place in community settings where social and self-care opportunities would naturally occur.

Historical Background

Females with ASD are currently a minority (approximately 25%) of those diagnosed with ASD, and comprise only about 15% of the subjects in published research literature (Watkins et al. 2014). This discrepancy in prevalence is likely related to multiple factors, including an increased genetic risk for males, females with ASD being more likely to be missed, or diagnosed later (Begeer et al. 2013) perhaps because of milder symptom expression (Lai et al. 2015), or because of compensatory skills developed by girls with ASD (Mandy et al. 2012). Researchers and practitioners are beginning to study more closely the unique symptomatology, experiences, and support needs of females with ASD (Lai et al. 2015).

For example, a study examining diagnostic criteria for higher functioning males and females (Hiller et al. 2014) found that not meeting criteria for “restricted or fixated interests...” was predictive of being female. Females were more likely to demonstrate partial impairment in nonverbal skills and no impairment within the “sharing of interests” category as compared to males. If sex differences within the general population suggest females use more nonverbal communication (i.e., eye gaze, gestures), this may explain findings as in Hiller’s study suggesting less or only partial impairment in nonverbal communication.

It is important to note that there are key differences in social interaction and friendship styles between males and females. For example, male friendships tend to be more activity-based, while female relationships include more emotion-focused, empathic connections and relationships (Hannah and Murachver 1999).

As research continues on the unique presentation or symptomatology for females with ASD, relatively little is known about potential differences regarding intervention approaches to promote social-emotional health in females with ASD. The *GNO* program, developed with a basis in empirical approaches, is the first published intervention study specifically targeted to females with ASD (Jamison and Schuttler 2017). The first

author developed *GNO* in 2008 in response to needs identified from clinical experience.

Rationale or Underlying Theory

Rationale

Although females with ASD, particularly high functioning, may demonstrate less impaired social-communication skills at some points in development (Van Wijngaarden-Cremers et al. 2014), they often experience increased social and communication impairments during adolescence as compared to males with ASD (Kirkovski et al. 2013; Koenig and Tsatsanis 2005). Some individuals with ASD demonstrate variable adaptive skills or skills well below the expected performance given their cognitive functioning (Ditterline and Oakland 2009). Thus, for adolescent girls with ASD who have self-care skills below expected norms and limited social interactions, the potential impact on self-perception is significant. Compared to males, typically developing adolescent females demonstrate an increased risk for developing internalizing symptoms such as anxiety and depression (Galambos et al. 2003). The elevated risk for internalizing symptoms and increased complexity in social interaction coupled with social impairment as a core feature in ASD result in adolescent girls with ASD experiencing a “double hit” (Solomon et al. 2012) or “double whammy” (Jamison and Schuttler 2015). In fact, adolescent girls with ASD exhibit significantly more internalizing symptoms (e.g., anxiety, depression) compared to typically developing girls (Jamison and Schuttler 2015) and to boys with ASD (Solomon, et al. 2012), suggesting this is a critical area for intervention. These potential sex differences in social behavior and coexisting social-emotional challenges reflect unique targets within interventions and programs aimed at improving social competence.

Underlying Theory

GNO aims to improve self-perception and self-confidence, anticipating that greater confidence

will result in the increased likelihood of engaging in social “entry” skills and social activities and, in turn, that improved social skills and increased competence will result in improved self-perception and positive social-emotional health. The curriculum targets socially valid behaviors (e.g., reciprocal conversation, shared interests, independence) related to social norms of adolescent females and the unique needs of females with ASD (i.e., improved social emotional competence and emotional health, independence). Instruction and practice occur within age-expected activities, providing a meaningful social experience to individuals who often report limited social opportunities or friendships (Bauminger and Kasari 2000). GNO’s framework is based on social learning theory, behavioral and cognitive-behavioral theories, while embracing concepts related to self-determination, developing social competence, empowering young women, and building inclusive communities.

The theory of change is rooted in Social Learning Theory (SLT) and related behavioral or cognitive-behavioral theories. SLT suggests that people learn new behaviors and/or improve skills through observation (Bandura 1971) and that behaviors are the result of reciprocal interactions between cognitive, behavioral, and environmental determinants. Behavioral theory describes learning skills or behaviors through positive reinforcement and environmental contingencies (Skinner 1988) while in cognitive-behavioral theory, skills are learned by understanding emotional and cognitive interpretations of events, with cognitive and behavioral rehearsal of appropriate behaviors (Kendall 2006).

Our model postulates that behavior changes or skills develop based on some combination of and interaction between observation, practice, and cognitive restructuring that occurs under specific conditions. These conditions include: planned observations for discrete behaviors or skills (live models, video models); repeated exposure and practice across multiple exemplars (skills, people, settings, activities); cognitive restructuring as a result of specific feedback (in vivo coaching and specific praise) and reinforcement of target skills

(differential attention, verbal feedback, token economy); and planned generalization through use of the desired skill within settings future performance is needed. GNO includes similar age girls without ASD/NDD that model skills and use of supports and help to craft opportunities for genuine practice across exemplars, creating frequent, yet natural opportunities to practice target skills. The premise is that experiencing success demonstrating these skills should increase the likelihood of engaging in the behavior in the future, which could be the result of reinforcement principles or cognitive appraisal (e.g., confidence, schema of skill, awareness, etc.), or likely some combination of both. Increased competence or self-perception of competence (confidence) leads to increased engagement in social experiences, which increase social competence and self-perception. While skill increase or enhancement is the immediate goal, a broader intention is to promote independence and positive social-emotional health by improving self-perception and confidence through individually identified goals rooted in the concept of self-determination. Conducting sessions in natural settings and providing training to community-based providers promote communities that are more inclusive.

Goals and Objectives

The goals of *GNO* are primarily twofold: (1) Promote social-emotional health, primarily related to improved social competence, self-perception, self-determination, and perceived quality of life and reduced internalizing symptoms and (2) greater independence and competency in self-care and social skills. Target skills reflect a developmental framework considering expected skills for adolescent females, the unique needs of females with ASD, and individual goals and areas of growth.

Improving Social-Emotional Health

GNO is designed specifically to address common symptoms or challenges females with ASD often encounter, including fewer peer interactions and

social experiences, variable social skills, limited self-confidence, and negative self-perception. By increasing competence and confidence in these social skill areas, the program aims to mitigate symptoms of social anxiety or depression.

Specific objectives related to this goal vary based on individual needs of participants, with core areas related to these broad skillsets: increased frequency and improved quality of conversation entry and reciprocity, more social experiences (e.g., group activities, getting together, phone/text, etc.), setting, monitoring, and achieving goals, and improved self- and caregiver-ratings of social emotional health and competence (self-confidence, self-perception, quality of life, social competence).

Greater Independence and Competency in Self-Care and Social Skills

GNO addresses challenges related to independence completing adaptive behaviors, particularly related to self-care and social skills. Program objectives include fostering greater independence and reducing overreliance on family or other caregivers for self-care and social skills, potentially leading to increases in social supports from peers. The goal is that improved competence within this arena will influence confidence and self-perception, promoting overall social-emotional health. Specific skillsets targeted and measured include: frequency and type of self-care and social activities, and level of independence navigating these activities and tasks.

Treatment Participants

Population

Developed for girls with ASD and other related neurodevelopmental disorders (NDD). Due to the high prevalence of internalizing symptoms in adolescent females with ASD, this intervention targets a broader concept of social-emotional health, and thus appropriate for girls with developmental disability and co-existing mental health disorders or symptoms related to anxiety, depression, and poor self-perception.

Age and Developmental Considerations

Designed for girls *about* 14–19 years old, with positive outcomes found for girls ranging from 8th grade, high school, and a few years past high school graduation (e.g., 18–21 special education program). Girls of a variety of abilities can participate in *GNO* and benefit from the intervention. Previous inclusion criteria for *research participation* required participants to read words at or above a 4th grade level in order to reference printed materials and supports and to demonstrate some conversational speech. However, curriculum and materials are adaptable for individuals of a variety of abilities. Previous adaptations include modified materials (e.g., lower reading level, large print for visual impairments) and inclusion of girls with limited expressive language but capabilities to make some comments contributing to individual or group conversations and receptive abilities necessary to follow directions and comprehend conversation topics and lesson content. Because participants include girls with and without ASD/NDD, groups regularly include girls with a range of cognitive, academic, and communication abilities and various social experiences and interests. The current program description focuses on the adolescent age range, with adaptations currently in progress to modify the curriculum for school-age girls and young adults.

Other Considerations

Independence and choice are valued and thus consent for participation is required from participants as well as caregivers. However, it is not uncommon for participants to express a hesitancy to begin the program and associated social activities. Parents or caregivers are coached at enrollment on how to present details of the program, and acknowledge feelings and concerns of their participant, while also maintaining focus on potential positives of the experience.

Our experience implementing the program over time suggests that frequently, while individuals may not express a desire for friendships or an initial interest in *GNO*, by committing to attending and attempting activities, they are often surprised by the positive effects they experience. These

impacts include increased social interests, desire for ongoing relationships, and positive experiences with the program and fellow participants/peers. Initial enthusiasm of the participant is not essential or predictive of outcomes or engagement in *GNO* activities. Post session survey data indicate high satisfaction regarding program participation for all participants and peer volunteers.

A primary aim in the delivery of this intervention is to tailor supports and strategies to meet the individual goals and needs of each participant, provide social interactions within familiar and unfamiliar activities or settings, promote independence in social and self-care skills, and to facilitate connections (or desire for connections) between girls with similar interests.

Group characteristics might also influence participant outcomes. Enrolment requires parents, peers, and participants to demonstrate availability for nearly all sessions and reliable transportation. Participants are most likely to benefit from this intervention when they attend all (or nearly all) sessions, practice skills, and utilize supports within and outside of weekly sessions.

Treatment Procedures

Program Structure

GNO entails weekly, 2-h sessions over the course of 12–15 weeks. Each session meeting targets one or more of the core curriculum areas and take place during age-typical self-care or leisure activities within community-based settings. Groups include 4–6 participants with a disability (ASD/NDD) and *at least* an equal number of trained peer volunteers. Peer training includes information about peer expectations and activities prior to beginning *GNO* as well as immediately prior to about half or more of subsequent sessions. Each session follows a similar format (see table for *GNO* Session Format), including multiple opportunities to practice targeted self-care or social skills with subsequent sessions designed to build on and reinforce skills from previous sessions. The program utilizes carefully selected empirically based strategies to teach and reinforce skills related to core curriculum concepts.

Targeted social conversation skills are practiced and reinforced in each session and supported by cue cards with tips for conversation (known as “conversation keyrings”), and video modeling clips depicting conversation skills based on the *relating to others* concept. Facilitators utilize differential reinforcement, including specific feedback paired with “*GNO* bucks” (token economy), to reinforce targeted curriculum skills and behaviors tailored to the individual. Participants access support materials housed in personalized *GNO Planners* with homework designed to promote practice and use of skills outside of sessions, including self-monitoring. Participants earn *GNO* bucks for bringing required materials, self-monitoring, and completing homework (Table 1).

GNO Curriculum

The curriculum is organized into three core areas: relationship building skills, promoting independence in self-care skills, and self-determination in social competence and self-perception. The

Girls Night Out Model, The, Table 1 *GNO* Session Format

• Facilitator meeting, preparation, community partner training
• Peer training
• <i>GNO</i> opening activities
- <i>GNO</i> “business” (distribute conversation keyring topics, pay people with <i>GNO</i> bucks for WIDTW sheets, planners, HW)
- Social time (review conversation topics, facilitators provide specific feedback paired with <i>GNO</i> bucks)
• Follow up on homework
• Planned activity or lesson (teach, practice, community partner consult)
• Practice during social or self-care activity with in vivo coaching, specific feedback, and <i>GNO</i> bucks to reinforce target skills
• Data collection
• Closing activities
- Shop at <i>GNO</i> store (token economy)
- Group picture and community partner thank you (if applicable)
- Homework: Assign my <i>GNO</i> friend, review new homework
• Facilitator debriefing
- Integrity checklist, participant notes, next session plans

table summarizes targeted skills within each of these three core areas, with strategies utilized to teach and reinforce these concepts throughout the intervention. (1) *Relationship building skills* address the heightened focus on conversation, relationship building, and shared interests that occur in adolescent female relationships (Hannah and Murachver 1999) and include conversation and friendship skills organized around relating to the person, the activity, and the relationship. Specific social conversation skills serve as “entry” tools to initiate conversation with other girls across contexts and the lifespan, with a focus on both conversation skills (i.e., asking questions/making comments, reciprocal exchanges, complimenting, etc.) and conversation content (i.e., exchange of person-related information, activity or setting related topics, etc.). Concepts to promote “relating to the relationship” include establishing common ground, making plans, and offering encouragement or emotional support. Establishing common ground is obtaining and organizing information about people in your life (personal and shared interests) to create social opportunities and establish relationships. Participants utilize the information about friends or potential friends to guide if, when, and how to initiate making plans and how to identify activities of interest or mechanisms to find social opportunities. Emotional support includes giving and receiving compliments and using verbal and non-verbal encouragers, with ample opportunities to practice and demonstrate these skills during a variety of activities and settings (e.g., fitness, hair salon, ropes course). (2) *Building independence in completing self-care skills* acknowledges difficulties some individuals with ASD experience with daily living skills and addresses the changes in required self-care resulting from biological changes during adolescence. Building independence in self-care skills contributes to improved self-perception and confidence, leading to increased competence in social relationships. The focus within the curriculum is on promoting independence in determining relevant self-care needs and activities, improving skills necessary to complete these tasks, and utilizing supports designed to promote independent use of skills.

Specific areas of focus within the curriculum include selection of clothing consistent with context (setting, weather, activity, etc.); body care (skin, hair); and health (fitness, exercise), tailoring these self-care priorities and activities to align with individual participant values, needs, and interests. Goal development, reinforcement methods, and practice opportunities support skill development while empowering participants to recognize their individual preferences. (3) GNO facilitators *promote self-determination in social competence and self-perception* through specific activities designed to engage participants in guiding their own social-emotional health outcomes. Activities include soliciting information from participants and their families about areas of interest and growth, facilitating individual goal setting and monitoring, and providing opportunities for a shift towards independence and positive peer support. Support targets relate to goal setting, use of visual supports to promote self-sufficiency, and accountability to themselves (self-monitoring, goal revision) and others within their support network (GNO facilitators and participants, caregivers, community partners). Program components include designing strategic opportunities to support the participant’s experience of autonomy, mastery, and control (Ward and Meyer 1999; Wehmeyer 1998) by crafting situations to practice and reinforce specific skills or actions. Community partnerships leverage shared expertise creating rich practice experiences based on knowledge and skills of partner (e.g., hair care and style expertise by salon) and strategies or supports provided by the program (e.g., visual supports for participants and facilitator feedback to community partners). Peer training includes techniques to elicit desired skills from participants or to set the occasion for target behaviors. For example, peers make “bids” in conversation to elicit questions (e.g., “I’m so excited I can hardly wait for this weekend”) and extend pauses before continuing in conversation to allow participants ample time to form questions. Participants receive specific feedback and reinforcement (*GNO bucks*) for use of targeted skills and experience successes within a safe, supportive, and socially valid context. Homework is designed to promote skill use

outside of session, including generalization of skills and goal attainment. Participants earn GNO bucks for self-monitoring and bringing materials to sessions and support fellow GNO participants in goal-related activities or skill practice outside of session as well.

Relating to others	Strategies
Conversation “entry” skills (person and activity topics, giving compliments, asking questions)	Social Learning Theory -peer mediated -video modeling (VM) -Modeling and role play -visual supports (task analysis, my GNO planner, individualized)
Finding common ground	
Making plans	
Emotional support	
Promoting independence in self-care skills	Cognitive and behavioral -reinforce target skills -differential attention -goal setting and monitoring -in-vivo coaching -specific feedback -token economy GNO bucks/GNO store) -planned generalization (natural—/community-based settings, multiple exemplars)
Selecting appropriate clothing	
Body care (hygiene)	
Skin care	
Hair care	
Health (fitness, nutrition)	
Self-determination in social competence and self-perception	
Identify personal strengths and areas of growth (<i>COP M</i>)	
Goal setting and monitoring	
Build self-determination	
Independence in activities/ skills	
Use of supports and technology	

Related Program Elements and Facilitator Roles

Parents or caregivers do not attend sessions with participants. However, a simultaneous *PNO* (*Parents Nigh Out*) component emerged based on experiences conducting groups and feedback from families. *PNO* includes GNO coordinated activities as well as impromptu or parent initiated gathering. Although variable, the goal is for about 50% of sessions to include *PNO* (formal or informal), with a focus on connecting families, sharing resources, and building infrastructure for continued social opportunities and relationships. A parent volunteer from a previous *GNO* group

often coordinates these activities based on feedback from families. Past activities included hosting a guest speaker (e.g., transition specialist), connecting families from current and past groups, or shared resources and support during informal social gatherings. Although *PNO* is optional and not systematically evaluated, families often report benefit from *PNO* events and seem more likely to participate in future *GNO* related activities designed to promote skill maintenance and ongoing social experiences.

An optimal ratio of approximately four facilitators for a group of 9–12 girls is suggested. GNO facilitators utilize a coaching model to give specific feedback about skill use during role-plays and naturalistic practice opportunities for social and self-care skills. Facilitators whisper specific praise or feedback when participants demonstrate target skills or goal-related behaviors. Facilitators utilize principles of differential attention (Pemberton et al. 2013) by focusing attention and feedback on positive behaviors and minimizing feedback about negative or inappropriate behaviors, gradually introducing *some* prompting of skills, still followed by specific praise for exhibiting the skill. Facilitator planning and debriefing provide opportunities for strategic feedback that is tailored to participants and peers based on individual goals and areas of need.

Unique Qualities of Treatment

GNO incorporates unique treatment components along with some common, evidence-based strategies often utilized within social skills programs. One obvious feature is the all-female group designed to address the unique needs of girls with ASD. Although one might argue a 4–5:1 male to female ratio suggests a lesser impact on females as a general population, this discrepancy in diagnosis actually has a residual impact on females with autism, potentially exacerbating social-communication impairments and risk for co-occurring mental health conditions.

The greater representation of males within autism-specific programs and special education services (~66% male; Retrieved 13 December

2017 at https://nces.ed.gov/programs/digest/d16/tables/dt16_204.50.asp) translates to less time girls with ASD spend with similar-aged female peers, narrowing the pool of potential friends and limiting opportunities to practice social interactions and establish relationships with other girls their age. Unlike many social skills programs or settings that target individuals with ASD, *GNO* is an all-female group that provides opportunities for social outings and interactions with other girls about the same age and for families to potentially connect around shared experiences. Families often report few girls in previous social programs, support groups, and classrooms and describe the value of participants and families connecting and relating to one another. Use of a coaching model, with peer-mediated strategies for teaching and practicing skills, provides authentic and genuine opportunities for practice within realistic social settings. The model and curriculum reaches beyond skill building and targets overall social-emotional health. Partnerships within the community promote shared expertise between local professionals and facilitators with disability-specific expertise and work towards more inclusive communities.

Efficacy Information

Clinical outcome data, based on a combined sample ($n = 34$) from five groups across the past 4 years, suggests *GNO* is desirable to families and shows promising results for improved conversation and social-emotional outcomes in adolescent girls with ASD (Jamison and Schuttler 2017). Sample sizes for outcome measures vary ($n = 14$ – 34) as evaluation procedures evolved over time. Results show statistically significant improvements following intervention for total social competence ($p = 0.035$, $ES = 0.53$) and related subscales, global self-perception ($p = 0.008$; $ES = 0.53$), and Quality of life ($p = 0.04$, $ES = 0.52$). Participants also reported a significant decrease in internalizing symptoms ($p = 0.04$, $ES = 0.47$). Although samples are small,

medium to large effect sizes suggest meaningful improvements. Consumer satisfaction ($n = 36$) is high (*satisfaction with program activities* = $4.5/5.0$; *positive changes in specific skills* = $3.5/5.0$) with ratings from parents, participants, and peers signifying the intervention is a service that families value and need.

Program activities and targets skills reflect an iterative process informed by the literature and ongoing involvement of participants, peers, and families. Program *activities* arose initially in part from group discussions with typically developing, adolescent females between the ages of 13–18 years old ($n = 10$; Caucasian and living in a suburban community in a large Midwestern city in 2008). Subsequent activities and target skills reflect feedback from previous groups and our research on girls with ASD. Examining factors that influence peer acceptance revealed *engagement* as a critical component in successful conversations (i.e., asking questions, looking and sounding interested, nonverbal encouragers; Jamison et al., poster presented at AUCD 2012), which informed and validated the core components of relationship building skills. Preliminary findings from our qualitative study (Schuttler, Jamison, & Edwards, poster presented at IMFAR 2014) provide evidence for the theoretical framework and curriculum components that foster independence and promote reliance on positive peer influence rather than adults. Typically developing females ($n = 46$; mean age = 16.4) reported increased social complexity, greater independence, and more reliance on peers compared to family; however, girls with ASD ($n = 14$; mean age = 15.57) relied significantly more on family members (e.g., parents) for social and self-care support.

Outcome Measurement

The program uses informal and formal methods of measurement for key outcome areas of social skills/competence, self-care/adaptive skills, self-perceptions, and social-emotional functioning (including both broad quality of life and more

specific internalizing/externalizing symptomatology). Current tools used for pre-, post-, and follow-up measurement of key skill areas include the Behavior Assessment System for Children, 3rd Edition (BASC-3), Parent and Self Report, the Social Skills Improvement System (SSIS), Social Responsiveness Scale-Second Edition (SRS-2), Self-Perception Profile for Adolescents, 2nd Edition (SPPA-2), Autism Social Skills Profile, Youth Quality of Life (research version), and a self-care checklist developed concurrent with the program. Interview and ratings using the Canadian Occupational Performance Measure (COPM) inform goal development and attainment.

Data is collected throughout the session for some skills. Participants and peers self-monitoring specific skills related to social competence, adaptive skills (self-care, hygiene), and individual goals throughout the session. A few previous groups included evaluation of specific conversation skills as part a research study using a conversation coding system. Satisfaction data is collected at the conclusion of the program from peers, participants, and parents using Likert scale ratings of activities and skill levels and as an opportunity for families to provide written feedback about their experience.

Qualifications of Treatment Providers

Sessions typically involve two kinds of facilitators: lead and support. The lead facilitator is responsible for guiding the session and supervising overall session activities. Support facilitators help with setup and follow up post session. Both types of facilitators are responsible for in vivo coaching and feedback, teaching and supporting skills within session. The lead facilitator (1) should have background in implementing social skills programming and have experience with individuals with developmental disabilities. Support facilitators (2–3) usually have at least a bachelor's degree related to education, behavioral sciences, psychology, or communication sciences. Experience with individuals with ASD and other

DD is very helpful. For more information on individualized training for local implementation of the program, readers are encouraged to contact the authors of this chapter.

See Also

- [Behaviorally Based Social Skill Groups](#)
- [Peer-Mediated Intervention](#)
- [Social Skill Interventions](#)

References and Reading

- Bandura, A. (1971). *Social learning theory*. New York: General Learning Corporation.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development*, 71(2), 447–456. <https://doi.org/10.1111/1467.8624.00156>
- Begeer, S., Mandell, D., Wijnker-Holmes, B., et al. (2013). Sex differences in the timing of identification among children and adults with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43, 1151–1156.
- Ditterline, J. & Oakland, T. (2009). Relationships between adaptive behavior and impairment. *Assessing Impairment*, 24(2), 31–48.
- Galambos, N. L., Barker, E. T., & Almeida, D. M. (2003). Parents do matter: Trajectories of change in externalizing and internalizing problems in early adolescence. *Child Development*, 74, 578–594.
- Hannah, A., & Murachver, T. (1999). Gender and conversational style as predictors of conversational behavior. *Journal of Language and Social Psychology*, 18(2), 153–174. <https://doi.org/10.1177/02691927X99018002002>
- Hiller, R. M., Young, R. L., & Weber, N. (2014). Sex differences in autism spectrum disorder based on DSM-5 criteria: Evidence from clinician and teacher reporting. *Journal of Abnormal Child Psychology*, 42, 1381–1393.
- Jamison, T. R., & Schuttler, J. O. (2017). Overview and preliminary evidence for a social skills and self-care curriculum for adolescent females with autism: The girls night out model. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-016-2939-6>
- Jamison, T. R., & Schuttler, J. O. (2015). Examining social competence, self-perception, quality of life, and internalizing and externalizing symptoms in adolescent females with and without autism spectrum disorder: A quantitative design including between-groups and correlational analyses. *Molecular Autism*, 6, 53.

- Kendall, P. C. (2006). Guiding theory for therapy with children and adolescents. In P. C. Kendall (Ed.), *Child and adolescent therapy: Cognitive-behavioral procedure* (3rd ed., pp. 3–30). New York: Guilford Press.
- Kirkovski, M., Enticott, P. G., & Fitzgerald, P. B. (2013). A review of the role of female gender in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(11), 2584–2603. <https://doi.org/10.1007/s10803-013-1811-1>
- Koenig, K., & Tsatsanis, K. D. (2005). Pervasive developmental disorders in girls. In D. J. Bell & S. L. Foster (Eds.), *Handbook of Behavioral and emotional problems* (pp. 211–237). New York: Kluwer Academic/Plenum Publishers.
- Lai, M. C., Baron-Cohen, S., & Buxbaum, J. D. (2015). Understanding autism in the light of sex/gender. *Mol Autism*, 6, 24.
- Mandy, W., Chilvers, R., Chowdhury, U., et al. (2012). Sex differences in autism spectrum disorder: Evidence from a large sample of children and adolescents. *Journal of Autism and Developmental Disorders*, 42, 1304–1313.
- Pemberton, J. R., Borrego, J., & Sherman, S. (2013). Differential attention as a mechanism of change in parent–child interaction therapy: Support from time-series analysis. *Journal of Psychopathology and Behavioral Assessment*, 35(1), 35–44.
- Skinner, B. F. (1988). Methods and theories in the experimental analysis of behavior. In A. Charles & S. Harnad (Eds.), *The selection of behavior: The operant behaviorism of B.F. Skinner: Comments and consequences* (pp. 77–149). New York: Cambridge University Press.
- Solomon, M., Miller, M., Taylor, S. L., Hinshaw, S. P., & Carter, C. S. (2012). Autism symptoms and internalizing psychopathology in girls and boys with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 48, 48–59. <https://doi.org/10.1007/s10803-011-1215-z>
- Van Wijngaarden-Cremers, P. J., van Eeten, E., Groen, W. B., et al. (2014). Gender and age differences in the core triad of impairments in autism spectrum disorders: A systematic review and meta-analysis. *Journal of Autism and Developmental Disorders*, 44, 627–635.
- Ward, M. J., & Meyer, R. N. (1999). Self-determination for people with developmental disabilities and autism: Two self-advocates' perspectives. *Focus on Autism and Other Developmental Disabilities*, 14, 133–139. <https://doi.org/10.1177/108835769901400302>
- Watkins, E. E., Zimmermann, Z. J., & Poling, A. (2014). The gender of participants in published research involving people with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 8(2). <https://doi.org/10.1016/j.rasd.2013.10.010>
- Wehmeyer, M. L. (1998). Self-determination and individuals with significant disabilities: Examining meanings and misinterpretations. *Research and Practice for Persons with Severe Disabilities*, 23(1), 5–16.

Glasgow Sensory Questionnaire (GSQ)

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Synonyms

Adult/Adolescent Sensory Profile (AASP); Inventory of Interpersonal Situations (IIS); Principal component analysis (PCA); Sensory Sensitivity Questionnaire (SSQ)

Description

The Glasgow Sensory Questionnaire is a 42-item measure, which captures self-rated hyper- and hypo-sensory sensitivity to stimuli across seven sensory domains (Robertson and Simmons 2013). It was initially constructed based on reports of common sensory signs and symptoms associated with ASD. The sensory domains represented in the measure are visual, auditory, gustatory, olfactory, tactile, vestibular, and proprioception. Items are equally distributed among sensory modalities, with three questions assessing reported hypersensitivity and three determining hyposensitivity. All questions ask how frequently certain sensory events were experienced, with participants responding using the scale: “Never–Rarely–Sometimes–Often–Always.” Responses are coded on a scale from 0 to 4, with possible scores ranging from 0 to 168.

Questions are phrased to ask if participants currently encounter the sensory events described. However, if the frequency with which they experience such events has changed over their lifetime, they are asked to choose the response that best matches their experiences over the preceding 12 months. Sample questions are included at the

start of the questionnaire in order to highlight these points.

The questionnaire has been translated into six languages, with the French (Sapey-Triomphe et al. 2018), Japanese (Takayama et al. 2014), and Dutch (Kuiper et al. 2019) versions being published thus far. Our key results have been replicated in a larger sample (e.g., Horder et al. 2014). The GSQ is a useful measure of sensory processing beyond autism and has, for example, been used by researchers exploring synesthesia (Ward et al. 2017, *Scientific Reports*) and traits in ADHD (Panagiotidi et al. 2018). Informal reports by users of the GSQ for research purposes suggest that this questionnaire provides a quick and useful characterization of sensory sensitivities in both autistic people and non-autistic individuals.

The original GSQ was designed as a self-report questionnaire for adults, but recently efforts have been made to validate a caregiver report version (Smees et al. 2019) and a self-report version for children (Simmons et al. 2016; Millington and Simmons 2019).

In addition to replications of the original study in different populations and cultures, the GSQ has come to be used as a useful instrument for characterizing sensory sensitivities in empirical studies, especially when the experiment has a sensory component, for example, Freyberg et al. (2015) and Poole et al. (2017), in their comparisons of binocular rivalry and temporal order judgments, respectively, between autistic and control groups.

Historical Background

The Glasgow Sensory Questionnaire was initially developed with 70 items (version 1.0); this was subsequently reduced to 42 items after a principal component analysis (PCA) (version 1.1). This is reported in Robertson and Simmons (2013). The reduction affected all modalities equally (the number of items for each modality was reduced from 10 to 6, with an even split between questions targeting hypersensitivity and hyposensitivity). Version 1.2 was released in 2018, correcting an error that existed in the coding sheet.

Psychometric Data

In the original study (Robertson and Simmons 2013), reliability analyses for the 42 items of the questionnaire were reported: Cronbach's alpha (0.935) and Guttman's split-half technique (0.929). These scores indicated acceptable levels of reliability. Principal component analysis was used to reduce the number of items from 70 to 42 and also explore the factor structure, with a single underlying factor being identified.

Ujie and Wakabayashi (2015) reported psychometric features of the Japanese version of the GSQ. Internal consistency using Cronbach's alpha was acceptable (0.84). A joint factor analysis of the GSQ and AQ showed a two-factor solution, with the GSQ loading onto the first factor and AQ (minus the "attention to detail" subscale) loading onto the second factor. The authors highlighted that there are some differences in performance between high and low AQ scorers, something that warrants further analysis.

Sapey-Triomphe et al. (2018) conducted a comprehensive psychometric analysis of the French version of the GSQ, comparing to the original version (Robertson and Simmons 2013). They found that the GSQ score was very similar between the two studies, with all but one of the items in the French version (number 39) being within 1 standard deviation of the original English version. Internal consistency was assessed using Cronbach's alpha (0.95 for all 42 items; 0.94 for 14 subscales). They used factor analysis to assess the GSQ and found evidence of two factors, one consisting mainly of hypersensitive items and the second of hyposensitive items. This was observed in both high AQ and low AQ groups, but there were some fine-grain differences between the groups (e.g., more variance was explained for the high AQ group than the low AQ group).

Kuiper et al. (2019) investigated the psychometrics of the Dutch version of the GSQ, comparing those with a diagnosis on the autistic spectrum and those without. They reported excellent levels of reliability in both groups (autistic, 0.91; non-autistic, 0.90) for the whole questionnaire and good levels for the hyper (autistic, 0.87;

non-autistic, 0.85)- and hyporesponsiveness (autistic, 0.85; non-autistic, 0.81) scales. Reliabilities for the individual modalities were mixed. Test-retest reliability was high for both groups (autistic, $r = 0.92$, $p < 0.001$; non-autistic, $r = 0.83$, $p < 0.001$). Convergent and divergent validity was also assessed, by comparing data from the GSQ to (a) established self-report measures of sensory responsiveness and (b) non-sensory measures, for example, those measuring social skills. To measure convergent validity, the Dutch version of the GSQ was compared to the Adolescent/Adult Sensory Profile (AASP; Brown and Dunn 2002) and Sensory Sensitivity Questionnaire (SSQ; Minshew and Hobson 2008). Between the GSQ and AASP, a strong correlation was reported for the autistic group ($r = 0.77$, $p < 0.001$), with a moderate correlation for the non-autistic group ($r = 0.66$, $p < 0.001$). A moderate correlation was observed when compared to the SSQ (autistic, $r = 0.51$, $p < 0.001$; non-autistic, $r = 0.56$, $p < 0.001$). To assess divergent validity, the GSQ was compared to the Inventory of Interpersonal Situations (IIS; Van Dam-Baggen and Kraaimaat 1999). A small nonsignificant correlation existed for the autistic group ($r = 0.03$, $p = 0.78$) and a small negative correlation with the non-autistic group ($r = 0.25$, $p = 0.04$).

Clinical Uses

At this time, widespread clinical use is not recommended since further validation studies are needed. However, clinicians have obtained the GSQ for using in a clinical setting. The main purpose is reportedly to help identify areas of sensory need. It is important to highlight that diagnostic or eligibility decisions should not be based upon GSQ scores. However, on a case-by-case basis, the GSQ might help indicate which individuals need more extensive evaluation and perhaps offer some ideas about intervention. Professionals experienced in treating sensory processing problems in adults with ASD or related conditions should contact the authors before using the GSQ to discuss its appropriateness.

See Also

- [Sensory Features as a Marker of Autism Spectrum Disorders](#)
- [Sensory Processing](#)
- [Sensory Stimuli](#)

References and Reading

- Brown, C. E., & Dunn, W. (2002). *Adolescent/adult sensory profile: User's manual*. San Antonio: Psychological Corporation.
- Freyberg, J., Robertson, C. E., & Baron-Cohen, S. (2015). Reduced perceptual exclusivity during object and grating rivalry in autism. *Journal of Vision*, 15(13), 1–12. <https://doi.org/10.1167/15.13.11>.
- Horder, J., Wilson, C. E., Mendez, M. A., & Murphy, D. G. (2014). Autistic traits and abnormal sensory experiences in adults. *Journal of Autism and Developmental Disorders*, 44(6), 1461–1469.
- Kuiper, M. W., Verhoeven, E. W., & Geurts, H. M. (2019). The Dutch Glasgow Sensory Questionnaire: Psychometric properties of an autism-specific sensory sensitivity measure. *Autism*, 23(4), 922–932.
- Millington, E., & Simmons, D. R. (2019). A comparison of the children and parent Glasgow Sensory Questionnaire. Open Science Framework. <https://osf.io/xu9ym/>
- Minshew, N. J., & Hobson, J. A. (2008). Sensory sensitivities and performance on sensory perceptual tasks in high-functioning individuals with autism. *Journal of Autism and Developmental Disorders*, 38(8), 1485–1498.
- Panagiotidi, M., Overton, P. G., & Stafford, T. (2018). The relationship between ADHD traits and sensory sensitivity in the general population. *Comprehensive Psychiatry*, 80, 179–185.
- Poole, D., Gowen, E., Warren, P. A., & Poliakoff, E. (2017). Brief report: Which came first? Exploring crossmodal temporal order judgements and their relationship with sensory reactivity in autism and neurotypicals. *Journal of Autism and Developmental Disorders*, 47, 215–223.
- Robertson, A. E., & Simmons, D. R. (2013). The relationship between sensory sensitivity and autistic traits in the general population. *Journal of Autism and Developmental Disorders*, 43(4), 775–784.
- Sapey-Triomphe, L. A., Moulin, A., Sonié, S., & Schmitz, C. (2018). The Glasgow Sensory Questionnaire: Validation of a French language version and refinement of sensory profiles of people with high autism-spectrum quotient. *Journal of Autism and Developmental Disorders*, 48(5), 1549–1565.
- Simmons, D. R., Robertson, A. E., & Brown, L. (2016). The P-GSQ: A new self-report sensory questionnaire for children. Poster presented at the International Meeting for Autism Research (IMFAR), Baltimore, MD, May 11–14, 2016.

- Smees, R., Rinaldi, L. J., Simmons, D. R., & Simner, J. (2019). Measuring sensory sensitivities in children: The parent-completed Glasgow Sensory Questionnaire (GDQ-P). (in preparation).
- Takayama, Y., Hashimoto, R., Tani, M., Kanai, C., Yamada, T., Watanabe, H., . . . Iwanami, A. (2014). Standardization of the Japanese version of the Glasgow Sensory Questionnaire (GSQ). *Research in Autism Spectrum Disorders*, 8(4), 347–353.
- Ujie, Y., & Wakabayashi, A. (2015). Psychometric properties and overlap of the GSQ and AQ among Japanese university students. *International Journal of Psychological Studies*, 7(2), 195.
- Van Dam-Baggen, C. M. J., & Kraaimaat, F. W. (1999). Assessing social anxiety: The Inventory of Interpersonal Situations (IIS). *European Journal of Psychological Assessment*, 15(1), 25–38.
- Ward, J., Hoadley, C., Hughes, J. E., Smith, P., Allison, C., Baron-Cohen, S., & Simner, J. (2017). Atypical sensory sensitivity as a shared feature between synaesthesia and autism. *Scientific Reports*, 7, 41155.

Global and Regional Asperger Syndrome Partnership (GRASP)

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“Global and Regional Asperger Syndrome Partnership (GRASP)” was created by a group of individuals with Asperger’s syndrome in New York City seeking to form an organization run by people with Asperger’s and autism spectrum disorder in order to help their peers. In 2003, GRASP was gifted a generous grant by the FAR fund and subsequently filed for 501c-3 status to become a nonprofit. Since then, the GRASP team has expanded the organization to include more programming specific to increasing the independence of autistics and individuals with autism spectrum disorder. GRASP’s team has established structured workshops, social events and groups, as well as increasing the visibility of ASD adults within society through collaborative work with high-profile regional, national, and international organizations.

GRASP’s mission is to improve and enrich the quality of life of autistic teens and adults and individuals on the autism spectrum and their

families through community advocacy and outreach, education, peer supports, programming and services, at no cost to our members.

GRASP’s vision is to actualize and work toward a world where all autistic individuals and individuals on the autism spectrum are self-determined, respected, valued, and fairly represented, where appropriate supports and services are readily available to those in need, where people on the spectrum are empowered to participate in personal decisions that affect their lives and to achieve their dreams.

With over 20,000 members – over 40 in-person groups, online support groups, and real-time chats with GRASP staff, and information and referral services for individuals and families – GRASP is the largest organization for individuals with ASD. GRASP has a strong presence on social media through Facebook, Twitter, LinkedIn, and through website information and support.

For more information, visit www.grasp.org or contact by email at info@grasp.org.

Global Aphasia

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Synonyms

[Dysphasia](#)

Definition

Global aphasia is a disorder of language characterized by a severe impairment in all aspects of spoken and written language across comprehension and production.

When considering types and categories that fall under the broader term “aphasia,” global aphasia is among the most severe of the nonfluent aphasias as both comprehension and production of language are involved.

Estimates of global aphasia in the larger population are largely unknown, though it has been estimated that 80,000 people develop aphasia in the United States each year.

The prognosis for individuals with global aphasia is largely dependent upon the extent of the damage to the brain and the severity of the impairments they encounter. For some individuals, an initial global aphasia resolves as swelling in the brain decreases and the patient is left with residual symptoms that are more consistent with Broca's or Wernicke's aphasia. For others, the impairments across comprehension and production persist. Factors including age of onset, health, education level, and how soon treatment takes place after brain damage have been shown to be predictive of recovery in aphasia.

Individuals with global aphasia have often experienced significant damage to the majority of the "language centers" of the brain, including the arcuate fasciculus, Broca's area, and Wernicke's areas of the left hemisphere. Language skills are typically impaired across comprehension and production, and individuals are often rendered without functional speech or produce repetitive/rote speech only.

Intervention for individuals with global aphasia largely depends on the severity of the symptoms experienced. As both comprehension and production are impaired, treatment often focuses on providing the individual with maximal support and compensatory strategies. In addition, family and caregiver training and supports are critical to the recovery and management of global aphasia.

Please also see ► ["Aphasia"](#) for a list of caregiver support strategies.

See Also

► [Aphasia](#)

References and Reading

American Speech-Language-Hearing Association (ASHA). (2008). Incidence and prevalence of speech, voice, and language disorders in adults in the United States. Retrieved 1 May 2011 from: www.asha.org/research/reports/speech_voice_language.htm

Barresi, B., Goodglass, H., & Kaplan, E. (2001). *The assessment of aphasia and related disorders*. Hagerstown: Lippincott Williams & Wilkins.

Chapey, R. (2008). *Language intervention strategies in aphasia and related neurogenic communication disorders*. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins.

Goodglass, H., Kaplan, E., & Barresi, B. (2000). *Boston diagnostic aphasia examination (BDAE-3)* (3rd ed.). Austin: Pro-Ed.

Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *The Boston naming test*. Philadelphia: Lea and Febiger.

Kent, R. D. (1994). *Reference manual for communicative sciences and disorders: Speech and language*. Austin: Pro-Ed.

Kertes, A. (2006). *Western aphasia battery- revised (WAB-R)*. Austin: Pro-Ed.

Lapointe, L. L. (2004). *Aphasia and related neurogenic language disorders*. New York: Thieme Medical Publishers.

National Institute on Deafness and Other Communication Disorders. (2008). *Aphasia*. Retrieved 1 May 2011 from <http://www.nidcd.nih.gov/health/voice/aphasia.htm>

Global Versus Local Processing

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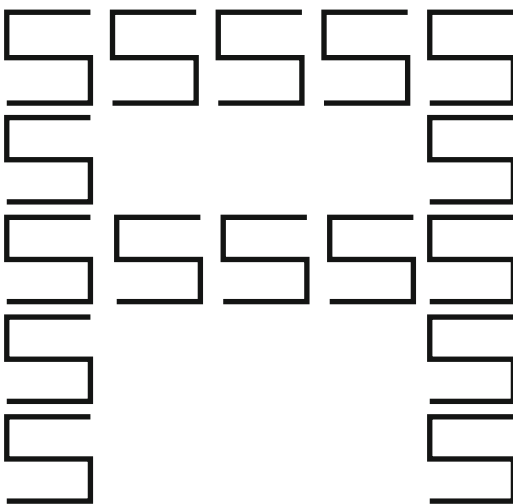
Definition

Local and global processing refer to hierarchical dimensions within perceptual patterns. Any spatially or temporally extended structure can be dichotomized in its embedding component or global level, and its embedded components or

local elements. Local and global dimensions are interdefined and are therefore only relative concepts: the same level can be considered as local relatively to its embedding structure and as global if it can itself be decomposed into embedded elements. The classic local–global tasks use “Navon-type” stimuli, which are large patterns (e.g., letters, geometrical figures) composed of small patterns of same or different nature (see Fig. 1). In the auditory modality, hierarchical stimuli can be implemented in melodies where local elements are single pitches or intervals, and global elements are melody or contour.

Global level has to be distinguished from holistic level, which is a pattern emerging only from the combination of local and global elements. For example, in a face, global level would be the face outline, parts would be eyes or mouth, but holistic (or gestalt) level would be the information that it is a face, which emerges only from the integration of parts and whole together. Whereas local versus global terminology is technically related to laboratory studies of pattern construction, whole and parts are akin to the gestalt tradition and refer, when empirically studied, to the relation between perception and understanding/recognition processes.

Differences in priority, speed, and accuracy of access to global versus local level information



Global Versus Local Processing, Fig. 1 Navon-type stimulus

define global or local *advantage*. The facilitating or detrimental effect that the processing of one level has on processing of the other is called *interference*. For example, dissimilarity between local and global level has typically more detrimental effect on the local than on the global processing: global-to-local interference is usually larger than local-to-global interference. Combined global advantage and superior global-to-local interference is called global *precedence* in cognitive literature (Robertson and Lamb 1991). The term *precedence* is usually replaced by *bias* in the autism literature.

Local and global levels differ not only in their logical and spatial relationships or hierarchical status but also in their respective psychophysical dimensions, neural representation, and relation with further recognition and labeling processes. For visually presented information, spatial frequencies are the average number of elements per unit of length. Global level is therefore composed of lower spatial frequencies and local level of higher spatial frequencies. In terms of neural representation, the local–global distinction is represented within the feed-forward flow of information of visual processing, in the form of a smaller versus larger amount of cells required to code the information (Grill-Spector and Malach 2004). Local elements require the integration of multiple psychophysical dimensions (contrast, luminance, and color). Local processing level is therefore anatomically and functionally more akin to low-level perception, level at which orientation or luminance discontinuities are coded. Conversely, global elements are anatomically and functionally closer to further levels of processing, as object recognition and labeling require the construction of a global representation (Grill-Spector and Malach 2004). Emotional value, for example, is mostly conveyed by global aspects of socially relevant material (Pourtois et al. 2005).

Historical Background

The local–global issue in autism goes back to Kanner’s initial description of various autistic behaviors apparently related to “irrelevant” or

“minute” aspects of their surroundings. Kanner’s first writings emphasized a dependence of the access to global dimension of an object or situation on *integrity* of all its parts. Thus, autistics would present an “inability to experience wholes *without full attention to the constituent parts*.” Kanner therefore did not postulate a *deficit* in the perception of global aspects of objects, but rather a *mandatory consideration of details* in the recognition/manipulation of entire objects or situations. Kanner’s reference to a perceptual hyperaccuracy and to superior performance of perceptual memory, “photographic or phonographic identity,” was explicit in this context (Kanner 1951). The detail-whole distinction, which may be considered as the ancestor of the “local–global” distinction in autism, had initially two meanings, which, although conceptually and ecologically interconnected, should be distinguished. First, *details* are opposed to *entire objects* and refer to a hierarchical relation of inclusion between levels within a perceptual pattern. This relation occurs within perception: local and global aspects are both perceptual features. This interpretation will be referred to as the within-perception meaning of the local–global distinction. Second, details refer to the entire domain of phenomenal appearance or perception, and whole refers to meaning, function, or use of a perceptually distinct pattern or object. In this latter case, “attention to details” refers to an atypical top-down relationship between perception and other elements of cognitive architecture involving higher-order processes. This constitutes the top-down meaning of the local–global distinction. Both meanings are contained in Kanner’s statements on the relation between sameness and detail, in that sameness is the preservation of detailed perceptual appearance of the world, as opposed to its meaning or function. In contrast, the default level of conscious cognitive functioning in typical individuals is that of meaning, which bypasses the detailed appearance (Kanner 1951). The ambiguity between a within-perception and a top-down approach of local–global issues in autistic perception has obscured empirical research on perception in autism for 50 years.

After Kanner, the top-down approach was the first to be developed. Creak (1963) described in autism an interest for object characteristics independently of their function. Hermelin and O’Connor (1970) expanded the idea that autistics have access to “raw processing” which, according to the dichotomy in use at that time, corresponds to the idea of uncategorized, perceptual material. The top-down approach is also the basis for the weak central coherence model, which emerged during the late 1960s and 1970s through studies from Hermelin’s student, Uta Frith, before its first formal description in 1989. In one of her landmark papers, Frith opposed *gestalt* to *perceptual detail* and to *raw material* (Shah and Frith 1983). The Embedded Figures Test used in this paper, which contains two embedded perceptual levels, bridges the top-down to the within-perception interpretations, though it was not exploited as such. The enhanced detection of embedded figures was attributed to autistics being “less captured by meaning.” The gestalt was not clearly considered as a perceptual impairment in autism since autistics could label global pictures.

The local–global conceptual distinction appears in these terms in 1993, simultaneously in Shah and Frith’s study on Block Design task, with a mostly top-down approach, and in Mottron and Belleville (1993) with a strictly perceptual approach. For Shah and Frith, “a relative preference for processing local as opposed to global features” is attributed to a deficit in global processing, understood in a top-down sense. Therefore, the hypothesized deficit is not primarily perceptual, but refers to diminished or absent top-down influence on the entire field of perception. Tasks used to support these assumptions were also not primarily perceptual (they involved response times of several tens of seconds) despite presenting a hierarchical component. In contrast, Mottron and Belleville (1993) investigated hierarchical effects within perception with the description of perceptual analysis in an autistic savant draftsman, with a mixture of strictly perceptual (hierarchical stimuli, perception of impossible figures) and visuoconstructive tasks (graphic construction). They reported that the hierarchical processing and graphic construction of visual

representation did not present the global precedence effects evident in typical individuals.

In the following years, this ambiguity remained, resulting in two partly overlapping, partly antagonist trends of research: one on perceptual laws, the enhanced perceptual functioning model, with a late incursion in top-down influence on perception; the other on top-down effects or weak central coherence, with a late incursion in perceptual laws. Both are unfortunately confounded in the “local–global” issue. Consequently, the question “is there a global deficit” and “is there a local bias” in autism received two empirically and theoretical answers, according to the within-perception or top-down approach of these terms. The cliché contrasting enhanced perceptual functioning and weak central coherence models, with the first emphasizing intact global processing and the other supporting impaired global processing, is misleading. The term global has a within-perception meaning in the enhanced perceptual functioning model and a mainly top-down meaning in the weak central coherence account.

Current Knowledge

Only the within-perception meaning of the local–global distinction will be presented here. Visual perceptual hierarchy has been studied in autism through perceptual hierarchical tasks, in which response times are in the range of hundreds of milliseconds, thereby more prone to tap specifically perceptual processes, and visuospatial hierarchical tasks, in which the answer requires several tens of seconds.

Perceptual hierarchical tasks include Navon-type hierarchical stimuli, part-whole relationships in patterns and faces, and attentional search tasks. A review by Wang et al. (2007) of nine studies using hierarchical stimuli in autism, for a total of 96 autistic spectrum individuals and 126 typical individuals, reveals that group differences, when found, involve superior local bias or local-to-global interference in autistics. Response times

to global level stimuli among autistics are more affected by incongruent stimuli at the local level than are the response times of typical individuals. The default setting of hierarchical autistic perception can also be assessed by prompting the participant to the likelihood of the occurrence of the stimulus at one level or the other. Autistics present a shorter response time for local element similarity than for global similarity, contrary to typical individuals, with equivalent accuracy in both groups. Another consistent result in this type of tasks is autistics’ typical performance level for various aspects of multi-features, global pattern dimensions when constrained to do so (except in specific cases that cannot be considered as representative of the autistic population). Typical holistic processing is manifested by standard effect of “good form” on visual target detection; typical global advantage under various visual angles; faster response to global as compared to local configurations; similar influence of structural global bias, although to a lesser extent than typically developing individuals; and slower response to local stimuli in global incongruence condition or global interference (see Happe and Frith 2006; Mottron et al. 2006, for reviews). A neighboring approach of the local–global distinction is represented by attentional visual search tasks, in which the participant has to disembody targets from surrounding task-irrelevant distracters which share one or several features with the targets. Autistics usually display faster target detection, more accurate discrimination of elementary stimuli differing at the feature level, and diminished influence of increasing number of distracters (Plaisted et al. 1998). For social material (e.g., faces), autistics display a preference for local information in identity matching, a superior priming effect of face parts, and a generally superior processing of high spatial frequencies in tasks involving faces. Integrity of global level analysis is demonstrated by typical gains from the addition of configural information, mostly typical inversion effect, and by superior recognition of an embedded facial target compared to an isolated one.

Visuospatial hierarchical tasks in the autism literature comprise mainly the Block Design subtest of the Wechsler scales of intelligence, graphic reproduction of possible and impossible figures, and the Embedded Figures Test. Autistics display a constant pattern of enhanced performance in these tasks. When the processing of a global aspect conflicts with a local analysis among typical individuals (e.g., perceptual cohesiveness in Block Design, impossibility of a figure in graphic reproduction, and visual context in embedded figures), autistics perform better than typical individuals. In contrast, when this conflict is diminished, for example, by segmentation to diminish the perceptual cohesiveness in Block Design or by copying possible instead of impossible figures, autistics are brought back to a level of performance equivalent to that of typical individuals. Conversely, autistics are not rigidly bound to a local strategy, as demonstrated by their capacity to switch to a global strategy when required by the task (Caron et al. 2006).

Analogues of Navon-type stimuli in the auditory modality allow investigating hierarchical effects in auditory modality, by using the single note versus melody or chord perception, with the same message of enhanced perception of local elements and intact perception of global auditory patterns, despite diminished detrimental effects of pitch and timing interference on judgments of pitch change. Even when considered in isolation, some psychophysical properties of auditory elements such as pitch are processed more accurately in autistics (Bonnell et al. 2010). The same applies to visual modality, in that lower thresholds for luminance-defined features are reported in autistics (Bertone et al. 2005).

Main Accounts

Despite the apparent homogeneity of autistic “local bias,” the large array of tasks grouped under the umbrella term of local–global tasks renders poorly plausible the notion of a unitary mechanism. Explanations are centered either on

the modification of within-perception hierarchy under the constraint of altered processing of elementary visual and auditory properties or on the modification of top-down influences on perception.

Within-perception accounts: Visual and auditory hierarchical tasks share the requirement to detect, identify, match, or reproduce a local target embedded in a larger probe. The enhanced perceptual functioning model proposes that an intrinsic superiority in pattern detection may have the effect of a local bias in comparison to typical individuals. This superiority can be traced back to an enhanced processing performance for the psychophysical properties of the patterns for which autistics display superiority. This is well demonstrated in the auditory modality (superior pitch perception), with some correspondent in the visual modality (enhanced perception of luminance-defined gratings) or in intermodal perception (enhanced perception of audiovisual synchrony). In some tasks like melody and verbal pattern discrimination, visual search, block design, and face perception, the local bias can be directly related to enhanced feed-forward processing of elementary psychophysical visual features. In support of this account, the same autistics who display the higher level of “local bias” also display superiority in low-level discrimination tasks as well as in a wide range of pattern identification tasks (Caron et al. 2006). Conversely, a multimodal local bias may be involved in atypical perception of social releasers. For example, the preference for high-spatial frequencies may modify face scanning as well as the route of emotion interpretation, as most of the emotional aspects of faces are conveyed by low spatial frequencies (Vlamings et al. 2010).

Top-down accounts: Another series of accounts attributes the local bias, evident in autistics, to a diminished global precedence within hierarchical patterns. The type of limitation of a top-down intervention favoring global over local processing varies according to accounts. The weak central coherence account, initially proposed for the Embedded Figures Test and the

Block Design task, hypothesizes a deficit in the typical tendency to favor a gestalt approach. For example, increasing the perceptual cohesiveness of the figure to be reproduced double the execution time in typical individuals, without influencing response times in autistics with a Block Design peak. The concomitant superiority of these autistics in a *global* task discounts the possibility that their remarkable performance on the Block Design task derived from a deficit in analyzing global aspects of a figure (Caron et al. 2006). Within the enhanced perceptual functioning account, M. Dawson's optionality model states that autistics are not obligated to use a global strategy when a global approach to the task is detrimental to performance (Soulières et al. 2007). For example, autistic persons are better able than typically developing persons to copy globally impossible figures, but as able to identify that these figures are impossible. Optionality may apply both to the competition between local and global level within pattern construction and between meaningless and meaningful processing of perceptual material.

Conclusion

Two findings clearly emerge from the now extensive investigation of local–global processing in autism. First, when the processing of the global aspect conflicts with a local analysis among typically developing persons, autistics either display superiority in local detection or present a diminished influence from the global level on the local one. Second, when the global level is tested in isolation, it is unremarkable; when the local level is tested in isolation, it is either unremarkable or superior. When it can be disentangled from other executive, intentional or motor components, the active ingredient in autistics' atypical performance is invariably of a perceptual nature. Local bias has two sources: an altered low-level input shared by most perceptual tasks in which autistics display superiority, and a diminished influence on local processing from either global perceptual

channel or top-down, nonperceptual processes. These findings appear consistent across visual and auditory modalities, for short as well as long exposure tasks. Local bias in autism is now consensual and represents one of the strongest arguments for an atypical perception in autism as well as a key path to the understanding of overall autistic cognition.

Future Directions

- Which low-level property is responsible of enhanced pattern detection, when observed?
- Are elementary grouping processes intact, atypical, or impaired?
- Lateral interaction and crowding as possible contour enhancers
- Role of altered spatial frequencies in face perception
- Role of pitch processing in language acquisition
- How do hierarchical atypicalities cluster in the same individuals?

See Also

- [Perception](#)
- [Weak Central Coherence](#)

References and Reading

- Bertone, A., Mottron, L., Jelenic, P., & Faubert, J. (2005). Enhanced and diminished visuo-spatial information processing in autism depends on stimulus complexity. *Brain*, 128(10), 2430–2441.
- Bonnel, A., McAdams, S., Smith, B., Berthiaume, C., Bertone, A., Ciocca, V., et al. (2010). Enhanced pure-tone pitch discrimination among persons with autism but not Asperger syndrome. *Neuropsychologia*, 48(9), 2465–2475.
- Caron, M. J., Mottron, L., Berthiaume, C., & Dawson, M. (2006). Cognitive mechanisms, specificity and neural underpinnings of visuospatial peaks in autism. *Brain*, 129(Pt 7), 1789–1802.
- Creak, M. (1963). Childhood psychosis: A review of 100 cases. *The British Journal of Psychiatry*, 109, 84–89.

- Frith, U. (1989). *Autism: Explaining the enigma*. Oxford, UK/Cambridge, MA: Basil Blackwell.
- Grill-Spector, K., & Malach, R. (2004). The human visual cortex. *Annual Review of Neuroscience*, 27, 649–677.
- Happe, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36(1), 5–25.
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. Oxford: Pergamon Press.
- Kanner, L. (1951). The conception of wholes and parts in early infantile autism. *The American Journal of Psychiatry*, 108(1), 23–26.
- Mottron, L., & Belleville, S. (1993). A study of perceptual analysis in a high-level autistic subject with exceptional graphic abilities. *Brain and Cognition*, 23(2), 279–309.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36(1), 27–43.
- Plaisted, K., O'Riordan, M., & Baron-Cohen, S. (1998). Enhanced visual search for a conjunctive target in autism: A research note. *Journal of Child Psychology and Psychiatry*, 39(5), 777–783.
- Pourtois, G., Dan, E. S., Grandjean, D., Sander, D., & Vuilleumier, P. (2005). Enhanced extrastriate visual response to band pass spatial frequency filtered fearful faces: Time course and topographic evoked-potentials mapping. *Human Brain Mapping*, 26(1), 65–79.
- Robertson, L. C., & Lamb, M. R. (1991). Neuropsychological contributions to theories of part/whole organization. *Cognitive Psychology*, 23(2), 299–330.
- Shah, A., & Frith, U. (1983). An islet of ability in autistic children: A research note. *Journal of Child Psychology and Psychiatry*, 24(4), 613–620.
- Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry*, 34(8), 1351–1364.
- Soulières, I., Mottron, L., Saumier, D., & Larochelle, S. (2007). Atypical categorical perception in autism: Autonomy of discrimination? *Journal of Autism and Developmental Disorders*, 37(3), 481–490.
- Vlamings, P. H., Jonkman, L. M., van Daalen, E., van der Gaag, R. J., & Kemner, C. (2010). Basic abnormalities in visual processing affect face processing at an early age in autism spectrum disorder. *Biological Psychiatry*, 68(12), 1107–1113.
- Wang, L., Mottron, L., Peng, D., Berthiaume, C., & Dawson, M. (2007). Local bias and local-to-global interference without global deficit: A robust finding in autism under various conditions of attention, exposure time, and visual angle. *Cognitive Neuropsychology*, 24(5), 550–574.

Globus Pallidus

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Synonyms

Paleostriatum

Definition

Its name derived from the Latin for “pale globe” and also termed the paleostriatum the globus pallidus (GP) is a subcortical structure of the brain and part of the basal ganglia. For some time it was associated with the putamen and referred to as the lentiform nucleus. Topographically, the globus pallidus it is part of the telencephalon and a major component of the extrapyramidal motor system. It connects with the substantia nigra, which is quite similar in neuronal architecture. The GP is divided into two parts – termed the internal and external GP – by the medial medullary lamina. These nuclei receive GABAergic axonal terminal branches from the striatum. Damage to the GP may result in stereotyped behavior and impaired extrapyramidal motor functioning. The GP is a target for deep brain stimulation therapy in Parkinson's disease.

References and Reading

- Bronstein, J. M., Tagliati, M., Alterman, R. L., Lozano, A. M., Volkmann, J., Stefani, A., et al. (2011). Deep brain stimulation for Parkinson disease: An expert consensus and review of key issues. *Archives of Neurology*, 68(2), 165. Epub October 11, 2010.
- Langen, M., Kas, M. J., Staal, W. G., van Engeland, H., & Durston, S. (2011). The neurobiology of repetitive behavior: Of mice. ... *Neuroscience and Biobehavioral Reviews*, 35(3), 345–355.
- Schuenke, M., Schulte, E., Ross, L. M., Lamperti, E. D., Schumacher, U., & Rude, J. (2010). *Head and neuroanatomy* (THIEME atlas of anatomy series) (pp. 211–214). Stuttgart: Thieme.

Glu

► Glutamate

Glutamate

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Synonyms

Glu; Glutamatergic

Definition

Glutamate (Glu) is a neurotransmitter that governs the excitatory synaptic transmission in the central nervous system. It plays a key role in brain development, such as migration, differentiation, survival, synaptic pruning, and plasticity. Glu activates ionotropic receptors including α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPA) and N-methyl-D-aspartate receptor (NMDAR), as well as metabotropic glutamate receptors (mGluR 1-7). Aberrant Glu system has been implicated in autism spectrum disorder (ASD). ASD patients show elevated serum levels of Glu, and increased brain expression of glutamate receptor and transporters. Increased densities of dendritic spine, a specialized compartment of glutamatergic synapse, correlate with decreased brain weight and severity of ASD symptomatology. In ASD-like animal models, abnormal pre-synaptic glutamate release, glutamate-mediated neurotransmission, and plasticity are found, and dysfunction of glutamate transporter leads to overproduction of glutamate and NMDAR-mediated excitotoxicity. At the genetic level, missense mutations of genes encoding Glu receptor are found in ASD patients,

while animals expressing these mutated genes show abnormal Glu channel properties and glutamatergic synapse morphology. Clinically, both agonists and antagonists of NMDAR have been reported to ameliorate ASD-related symptoms in human, suggesting that deviation of NMDAR activity in either direction may be implicated in ASD. Most significantly, alteration of glutamate synaptic transmission may lead to excitation-inhibition imbalance within the neuronal network, which has been postulated as pathophysiology of ASD.

See Also

► Shank 3

References and Reading

- Aida, T., Yoshida, J., Nomura, M., Tanimura, A., Iino, Y., Soma, M., & Tanaka, K. (2015). Astroglial glutamate transporter deficiency increases synaptic excitability and leads to pathological repetitive behaviors in mice. *Neuropsychopharmacology*, 40(7), 1569–1579.
- Hosenbocus, S., & Chahal, R. (2013). Memantine: A review of possible uses in child and adolescent psychiatry. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, 22(2), 166–171.
- Hutsler, J. J., & Zhang, H. (2010). Increased dendritic spine densities on cortical projection neurons in autism spectrum disorders. *Brain Research*, 1309, 83–94.
- O’Roak, B. J., Deriziotis, P., Lee, C., Vives, L., Schwartz, J. J., Girirajan, S., & Eichler, E. E. (2011). Exome sequencing in sporadic autism spectrum disorders identifies severe de novo mutations. *Nature Genetics*, 43(6), 585–589.
- Purcell, A. E., Jeon, O. H., Zimmerman, A. W., Blue, M. E., & Pevsner, J. (2001). Postmortem brain abnormalities of the glutamate neurotransmitter system in autism. *Neurology*, 57(9), 1618–1628.

Glutamatergic

► Glutamate

Gluten-Free Diet

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Definition

The gluten-free and casein-free diet (GFCF diet) is a popular dietary intervention used by families of people with autism spectrum disorders (ASD) in the effort to lessen core symptoms of ASD and/or gastrointestinal problems like bloating, diarrhea, and discomfort that may impact behavior. Gluten is a peptide (protein) found in grains such as barley, rye, and wheat, which provides elasticity to baked goods. Casein is a peptide found in all milk and milk products. The GFCF diet eliminates grain and dairy foods containing these peptides and processed food products containing them. Current use of a diet eliminating gluten and/or casein is reported by less than 20% of families participating in the Autism Treatment Network (Perrin et al. 2012).

Historical Background

Elimination diets have been investigated as means of altering behavior in people with developmental, behavioral, and medical diagnoses for many years. Case reports in the 1970s described individuals with symptoms of ASD whose behaviors improved with elimination of multiple foods including wheat products (Bird et al. 1977; O'Banion et al. 1978). In the 1970s and 1980s, the potential for dietary intervention to impact attention and hyperactivity was studied in clinical trials with largely negative results in the USA (see ► [Feingold Diet](#)). During this period, investigators in the UK and Norway observed that some

individuals with symptoms of ASD or schizophrenia seemed to improve with elimination of gluten and casein from their diets (Milward et al. 2008). To explain this clinical observation, they examined urinary excretion of peptides (proteins) that they thought were related to these foods. The GFCF diet gained popularity in the late 1990s through anecdotal reports of success, books for families in the popular press (Lewis 1998), and web-based organizations that supported families in implementing the GFCF diet.

Rationale or Underlying Theory

To explain the observation that some people with ASD seemed to have improvement in behavior or medical symptoms with elimination of gluten and/or casein, Shattock et al. (1990) and Reichelt et al. (1991) suggested the hypothesis that people with ASD had a "leaky" intestinal lining that allowed proteins to be absorbed from food that typically were not absorbed and that these substances subsequently acted like opiates in the brain. An additional hypothesis has been proposed that increased absorption of peptides related to casein and gluten may be due to altered gut peptidase activity (Christison and Ivany 2006).

Urine peptide measurement in people with autism on and off GFCF diets was published that suggested an increase in urine metabolites of gluten and casein that could be decreased with dietary intervention (reviewed in Christison and Ivany 2006). Measurement techniques for urinary peptide identification were less precise in the 1980s, so the compounds separated in the urine could not be specifically identified. Subsequent studies using newer and more accurate measurement techniques did not identify increased excretion of opiate-like peptides related to gluten and casein in children with ASD compared to controls (Cass et al. 2008; Hunter et al. 2003).

Although the original "opiate hypothesis" has not been supported by the more recent studies of urine peptides or current understanding of the neurobiology of ASD, it is plausible that the

empiric observation of behavioral improvement with dietary restriction may have an alternative explanation in subgroups of children with ASD. Celiac disease is an autoimmune disorder found in up to 1% of the population. Antibodies to gluten alter the intestinal lining in response to gluten exposure. This results in characteristic findings on blood testing for antibodies and on biopsy of the intestine performed during diagnostic endoscopy. Elimination of gluten from the diet in people with celiac disease results in improvement in gastrointestinal symptoms (e.g., pain, diarrhea) that might lead to irritability. Neurological symptoms associated with celiac disease include ataxia and peripheral neuropathy with anecdotal reports of impaired attention. Other hypotheses have been proposed regarding primary immunologic abnormalities in the intestine in people with ASD (Buie et al. 2010). Another potential explanation for improvement in behavioral symptoms in a subgroup of children with ASD is lactose intolerance. Lactose is an enzyme that allows the intestine to break down milk sugars found in cow's milk. The genetic predisposition to lactose intolerance is especially common in certain ethnic groups. Lactose intolerance may also occur after gastrointestinal infections from viruses or bacteria. If someone is lactose intolerant, elimination of milk sugar might decrease irritability from bloating and decrease diarrhea. Some people might benefit from an elimination diet if true allergy or dietary intolerance to milk protein or other foods. The most common food allergies include milk, wheat, eggs, tree nuts, and soy. It is also plausible that the elimination of processed foods containing gluten and casein changes the diet in other ways such as removal of food dyes and preservatives (see ► [Feingold Diet](#)). Alteration of the foods eaten may change the microbial environment in the gut which could potentially alter absorption of nutrients that are precursors to neurotransmitters (Dinan and Cryan 2017).

Goals and Objectives

The GFCF diet is typically trialed by families to determine if the symptoms of ASD or of gastrointestinal distress can be lessened with total

elimination of casein- and gluten-containing foods. Clinical studies to date have attempted to examine outcome criteria related to the core symptoms of ASD, language, and nutritional status.

Treatment Participants

The published studies in the scientific literature have used different criteria for characterizing the participants, controlling for behavioral and medical interventions that might impact change in symptoms over time, age of participants, the rigor with which the diet was monitored, and medical conditions which might impact outcome. Several case series (reviewed in Christison and Ivany 2006; Mulloy et al. 2010; Millward et al. 2009) and a single-blind study (Knivsberg et al. 2002) report on children with clinical diagnoses of ASD. The children in these studies ranged in age from 3 to 17 years. No attempt was made to control for other interventions. A larger single-blind trial (Whitely et al. 2010) reported on children whose diagnosis of ASD was supported by the Autism Diagnostic Observation Schedule. A smaller double-blind study supported the diagnosis with the Childhood Autism Rating Scale (CARS) (Elder et al. 2006). A small double blind, placebo-controlled challenge study studied 14 children between 2 and 5 years of age who all were getting similar Early Intense Behavioral Intervention (Hyman et al. 2016). Analysis of parent report data on a 90 item survey describing subjective response to the GFCF diet in 387 children included history of gastrointestinal problems (Pennesi and Klein 2012).

Treatment Procedures

The studies published in the scientific literature to date have used different procedures to educate families about implementing and maintaining the GFCF diet. The majority provided education-related implementation and ongoing support but did not describe dietary adherence. Elder et al. (2006) provided a complete diet to the families for the child under study. Hyman et al. (2016) had

a dietitian collect and examine 24-h diet diaries throughout the trial.

Study design also varied, making it difficult to compare data (Buie et al. 2010; Christison and Ivany 2006; Mulloy et al. 2009; Millward 2008). Several case series reported on open trials in which children were placed on GFCF diets (Pennesi and Klein 2012). Individual services varied, and medications and supplements that might affect behavior were permitted. Subjective and nonstandardized assessments further compromise interpretation. In two single-blind trials (Knivsberg et al. 2002; Whitely et al. 2010), the study evaluators were blinded to the group assignment, but the reporters of behavior were not. This has the potential for the parents and school personnel who know if the child is on the diet or not to report based on their bias. Double-blind, placebo-controlled approaches included a cross-over of 6 weeks on GFCF diet and 6 weeks off (Elder et al. 2006). Hyman et al. (2016) reported on double-blind placebo-controlled challenges of gluten, casein, and both and placebo in children maintained on a GFCF diet.

Efficacy Information

The efficacy of the GFCF diet was not consistent across studies. The utility of the diet varied between reported studies based on the outcome measures selected, study population, and interpretation of the data. The outcome measures are summarized in the section below.

While the popular press and Internet are replete with anecdotal endorsements of the GFCF diet, the scientific literature to date is not supportive of its use based on the clinical trials that have been published (Buie et al. 2010; Millward 2009). The studies typically are of small size. Negative studies usually need to be quite large to establish that a treatment is not useful. The largest study to date (Whitely et al. 2010) has over 50 participants but is single blind and does not control for other interventions that might have impacted change in behaviors. It did not document medical comorbidities like celiac disease or food allergies that might have impacted the response to dietary intervention. Since educational and behavioral

interventions may impact development and behavior as well (see ► [ABA](#)), interventions that are used for prolonged periods need to be evaluated in concert with other approaches that are also being used to impact symptoms.

For families who desire to implement an empiric trial of the GFCF diet, the scientific literature does not provide sufficient information to recommend a length of dietary trial or specific workup that would identify children with increased likelihood of response. The longer that a child is exposed to an intervention, the greater the likelihood that ongoing development and the other educational and behavioral interventions in place will also impact developmental gain. Many advocates of the diet recommend removal of one factor (e.g., casein) then removal of the other after an adjustment period. There is no evidence of an “addiction” to either casein or gluten to support a “withdrawal” effect. However, if an underlying intolerance to either component is the cause of irritability or GI distress, then a stepwise removal of casein and gluten might help a family determine the least restrictive diet necessary for symptomatic improvement.

If a family desires to undertake a trial of the GFCF diet, they should consult with their health-care provider regarding the potential for celiac disease, food allergy, or lactose intolerance. General allergy testing and measurement of urine peptides are not likely to be helpful. The family and therapy team can identify target behavioral data that can be collected during implementation of a trial of the GFCF diet to help determine if there is benefit. It can be as simple as structured collection of a Clinical Global Index, behavioral rating scales such as the Aberrant Behavior Checklist (see ► [ABC](#)), sleep diaries or rating scales, or formal language or cognitive testing. Some measures are not designed to measure change over short periods (e.g., ► [Autism Diagnostic Observation Schedule](#)) or cannot be administered at a frequent interval without introducing a practice effect (e.g., formal language and IQ tests). These measures may not be sensitive to subtle differences in behavior.

Outcome Measurement

The approach to measurement of behavioral effect varies between published studies, making comparison difficult. Core symptoms of ASD have been reported as subjective parent impression (Pennesi and Klein 2012) as well as through parent report using behavioral scales (GARS, Vineland) (Millward 2009; Whitely et al. 2010). Parent report captures important information; however, it also allows for significant bias if the parents are not blinded to the intervention. They, of course, knew that the diet was implemented in all but the double-blind, placebo-controlled study (Elder et al. 2006).

Core symptoms of ASD were addressed by varied approaches as well. While subjective ratings cannot be dismissed, they are not readily interpreted. Whitely et al. (2010) used the GARS to quantify the report of behaviors observed by the parents and the ADOS to document improvement over time. The ADOS as used was not developed to be used as a measure of change for symptoms of ASD (see ► [Autism Diagnostic Observation Schedule](#)). Elder et al. (2006) used the Childhood Autism Rating Scale to quantify improvement. While the families were blind as to dietary status, this instrument was not developed to measure improvement with repeat administration either (see ► [CARS](#)). The double-blind, placebo-controlled, randomized study did not identify exposure to gluten/casein as altering core symptoms of autism in the short run by the measures used.

Language and development was identified as an outcome measure in several studies. Knivsberg et al. (2002) suggested there was an improvement in formal language scores that did not reach statistical significance. Whitely et al. (2010) suggested developmental improvement over time on the Vineland during the first 6 months of intervention. Elder et al. (2006) found no change in language over the 6-week study interval.

Other behaviors that might be affected by diet were considered using several approaches. Elder et al. (2006) looked at global change without demonstrable effect.

Nutritional outcome of the GFCF diet has been examined as well. Cornish (2002) reviewed diet diaries collected by families recruited through a

postal survey. She suggested that children on restricted diets were not found to be at increased risk for nutritional deficiencies. The nutritional implications of the GFCF diet in community use over time are unknown. Hediger et al. (2008) noted decreased cortical bone density in boys with ASD that was further decreased in the presence of dietary limitation of dairy products. Stewart et al. (2015) did not identify nutritional compromise in children with ASD given a GFCF diet who were supplemented with calcium and vitamin D, but not all children on limited diets are appropriately supplemented (Marí-Bauset et al. 2016; Srinivasan et al. 2017). Ghalichi et al. (2016) used the Rome III criteria to document improvement in the gastrointestinal symptoms of children with ASD randomized to the GFCF diet. However, the report of behavioral improvement is difficult to interpret because the outcome measure was the GARS-2, a tool not designed to measure behavioral change.

Qualifications of Treatment Providers

Families considering a trial of the GFCF diet should discuss their plans with their health-care provider. Since dairy products are a major source of calcium and vitamin D for children, another source of these nutrients will need to be identified if milk and milk products are eliminated from the diet. Grain-containing products are typically supplemented with B vitamins and other nutrients and as such provide many essential nutrients in children's diets. Referral of families considering a trial of this diet to a registered dietitian for education may be helpful regarding education on foods containing gluten and casein, how to introduce new foods, nutritional monitoring, and advice regarding prudent supplementation as needed. An RD is a professional with graduate training in nutrition and state licensure. Health insurance may cover nutrition consultation depending on diagnosis.

In addition to counseling regarding nutritional adequacy of the foods their child will consume, families pursuing a GFCF diet need to be educated on reading food labels to determine which products contain gluten and casein. Gluten is present in foods labeled as containing wheat, barley, and rye. Other

ingredients containing gluten include malt, modified food starch, and others. Hidden sources of gluten may include spices dusted in flour to prevent clumping and foods fried in oil also used for gluten-containing products (e.g., oil in a restaurant used to cook both French fried potatoes and chicken nuggets with bread coating). All dairy products contain casein including milk, cheese, yoghurt, ice cream, sherbet, butter, and powdered milk. Milk proteins like casein and whey may be ingredients in packaged foods. Some vegan products like cheese substitute may contain casein. Lactose-free dairy products may contain casein or whey.

Clinicians who practice complementary, holistic, and integrative medicine may advocate for a trial of the GFCF diet with or without use of other nutritional supplements. The studies in the scientific literature do not report on combined interventions. The long-term health effects of high intake of supplements above recommended intake is unknown.

See Also

- ▶ [Autism Diagnostic Observation Schedule](#)
- ▶ [Childhood Autism Rating Scale](#)
- ▶ [Feingold Diet](#)
- ▶ [Gastrointestinal Disorders and Autism](#)

References and Reading

- Bird, B., Russo, D., & Cataldo, M. (1977). Considerations in the analysis and treatment of dietary effects on behavior: A case study. *Journal of Autism and Childhood Schizophrenia*, 7, 373–382.
- Buie, T., Campbell, D. B., Fuchs, G. J., Furuta, G. T., Levy, J., VandeWater, J., et al. (2010). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125S, S1–S18.
- Cass, H., Gringras, P., March, J., McKendrick, I., O'Hare, A. E., Owen, L., et al. (2008). Absence of urinary opioid peptides in children with autism. *Archives of Disease in Childhood*, 93, 745–750.
- Christison, G., & Ivany, K. (2006). Elimination diets in autism spectrum disorders: Any wheat amidst the chaff? *Journal of Developmental and Behavioral Pediatrics*, 27, S162–S171.
- Cornish, E. (2002). Gluten and casein free diets in autism: A study of the effects on food choice and nutrition. *Journal of Human Nutrition and Dietetics*, 15(4), 261–269.
- Dinan, T. G., & Cryan, J. F. (2017). The microbiome-gut-brain axis in health and disease. *Gastroenterology Clinics of North America*, 46(1), 77–89. PMID: 28164854.
- Elder, J. H., Shankar, M., Shuster, J., Theriaque, D., Burns, S., & Sherrill, L. (2006). The gluten-free, casein-free diet in autism: Results of a preliminary double-blind clinical trial. *Journal of Autism and Developmental Disorders*, 36, 413–420.
- Ghalichi, F., Ghaemmaghami, J., Malek, A., & Ostadrahimi, A. (2016). Effect of gluten free diet on gastrointestinal and behavioral indices for children with autism spectrum disorders: A randomized clinical trial. *World J of Pediatrics: WJP*, 12(4), 436–442. PMID: 27286693.
- Hediger, M., England, L., Molloy, C., Yu, K., Manning-Courtney, P., & Mills, J. (2008). Reduced bone cortical thickness in boys with autism or autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 38, 848–856.
- Hunter, L. C., O'Hare, A., Herron, W. J., Fisher, L. A., & Jones, G. E. (2003). Opioid peptides and dipeptidyl peptidase in autism. *Developmental Medicine and Child Neurology*, 45(2), 121.
- Hyman, S. L., Stewart, P. A., Foley, J., Cain, U., Peck, R., Morris, D. D., Wang, H., & Smith, T. (2016). The gluten-free/casein-free diet: A double-blind challenge trial in children with autism. *Journal of Autism and Developmental Disorders*, 46(1), 205–220. PMID: 26343026.
- Knivsberg, A., Reichelt, K., Høien, T., & Nodland, M. (2002). A randomized, controlled study of dietary intervention in autistic syndromes. *Nutritional Neuroscience*, 5, 251–261.
- Lewis, L. (1998). *Special diets for special kids*. Arlington: Future Horizons.
- Marí-Bauset, S., Llopis-González, A., Zazpe, I., Marí-Sanchis, A., & Suárez-Varela, M. M. (2016). Nutritional impact of a gluten-free casein-free diet in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(2), 673–684. PMID: 26428353.
- Milward, C., Ferriter, M., Calver, S. J., & Connell-Jones, G. G. (2008). Gluten-and casein-free diets for autistic spectrum disorder. *Cochrane Database of Systematic Reviews*, 1, 2–28.
- Mulloy, A., Lang, R., O'Reilly, M., Sigafos, J., Lancioni, G., & Rispoli, M. (2009). Gluten and casein free diets in the treatment of autism spectrum disorders: A systematic review. *Research in Autism Spectrum Disorders*, 4, 328–339.
- O'Banion, D., Armstrong, B., Cummings, R., & Stange, J. (1978). Disruptive behavior: A dietary approach. *Journal of Autism and Childhood Schizophrenia*, 8, 325–337.
- Pennesi, C. M., & Klein, L. C. (2012). Effectiveness of the gluten-free, casein-free diet for children diagnosed with autism spectrum disorder: Based on parental report.

- Nutritional Neuroscience*, 15(2), 85–91. PMID: 22564339.
- Perrin, J. M., Coury, D. L., Hyman, S. L., Cole, L., Reynolds, A. M., & Clemons, T. (2012). Complementary and alternative medicine use in a large pediatric autism sample. *Pediatrics*, 130(Suppl 2), S77–S82. PMID: 23118257.
- Reichelt, K., Knivsberg, A., Lind, G., & Nodland, M. (1991). Probable etiology and possible treatment of childhood autism. *Brain Dysfunction*, 4, 308–319.
- Seung, H., Rogalski, Y., Shankar, M., & Elder, J. (2007). The gluten- and casein-free diet and autism: Communication outcomes from a preliminary double-blind clinical trial. *Journal of Medical Speech-Language Pathology*, 15, 337–345.
- Shattock, P., Kennedy, A., Rowell, F., & Berney, T. (1990). Role of neuropeptides in autism and their relationships with classical neurotransmitters. *Brain Dysfunction*, 3, 328–345.
- Srinivasan, S., O'Rourke, J., Bersche Golas, S., Neumeyer, A., & Misra, M. (2017). Calcium and vitamin D supplement prescribing practices among providers caring for children with autism spectrum disorders: Are we addressing bone health? *Autism Research and Treatment*, 2016, 6763205. PMID: 27042348.
- Stewart, P. A., Hyman, S. L., Schmidt, B. L., Macklin, E. A., Reynolds, A., Johnson, C. R., James, S. J., & Manning-Courtney, P. (2015). Dietary supplementation in children with autism spectrum disorders: Common, insufficient, and excessive. *Journal of the Academy of Nutrition and Dietetics*, 115(8), 1237–1248. PMID: 26052041.
- Whitely, P., Haracopos, D., Knivsberg, A. M., Reichelt, K. L., Palar, S., Jacobsen, J., et al. (2010). The ScanBrit randomized, controlled, single blind study of a gluten and casein free dietary intervention for children with autism spectrum disorders. *Nutritional Neuroscience*, 13(2), 87–100.

Goal Attainment of Students with ASD Using COMPASS

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Definition

The Collaborative Model for Promoting Competence and Success (COMPASS) is an evidence-

based parent-teacher consultation and coaching intervention that roughly doubles the Special Education IEP goal attainment outcomes of children and youth with autism spectrum disorder (ASD). COMPASS brings together parents and teachers, with guidance and support from the COMPASS consultant, to create a shared understanding of the child with ASD that integrates the essential and critical information from home and school. This holistic and ecologically informed view leads to the identification of pivotal goals within the critical social, communication, and learning skill domains that leverage other areas of development. Following the identification of contextualized and socially validated goals, an intervention plan is developed based on the child's personal and environmental challenges and supports, consistent with the stipulations of the Evidence-Based Practice in Psychology (EBPP) tripartite framework. There is experimental evidence that COMPASS results in better education goal attainment outcomes for children and youth.

Historical Background

Developed by Ruble et al. (2012a), COMPASS is a framework for helping individuals with ASD achieve optimal outcomes. The model represents an accumulation of more than 80 years of combined experience of the authors in collaboration with parents, teachers, administrators, and other personnel in the field. Developed and refined over 25 years, COMPASS is a well-validated consultation intervention to systematically develop, implement, and monitor programs for students with ASD and remains one of the few consultation approaches experimentally tested in several randomized controlled studies evaluated by an independent assessment of student progress. The COMPASS consultation approach is based on educational research that shows that empirically validated professional development models that result in sustainable changes in teacher behavior and therapeutic instruction requires ongoing coaching support that occurs within the teacher's own instructional setting and is repeated over time.

The model was adapted originally from the work of August, Anderson, and Bloomquist and was first promulgated in 1992 under the title Minnesota Competence Enhancement Program. From 1978 until 1992, with both state and federal funding and under the leadership of Nancy Dalrymple at the Indiana University (Bloomington) University Affiliated Program (UAP), the model was utilized within the UAP-based residential programs for children and youth with autism and subsequently as part of a state-wide training initiative in Indiana. During this period, after extensive data gathering, the model was modified to include the concept of balancing risk factors with protective factors for developing competency. That concept was a key to the publication of “An alternative view of outcome” (Ruble and Dalrymple 1996), which advocated for new and different ways to measure outcome by focusing on the development of competence and quality of life as central outcomes and linking these to accommodations and social and family support networks. This work helped to reaffirm the evolving model’s emphasis on collaboration and building environmental and personal supports rather than emphasizing deficits.

Extensive field testing, including the development and validation of a COMPASS professional development training package, has continued from 1992 to the present time. In 1996 the model was used as the basis of the Autism Technical Assistance Manual for Kentucky Schools, which Dr. Ruble and Dalrymple authored. School systems throughout Kentucky had the opportunity to be trained with the manual, and the Kentucky Western Education Cooperative took the lead in incorporating the model in extensive training of all their school systems over several years. This training was always specific to individual students with autism, although the underlying framework can be readily applied to children with other types of developmental disabilities that could benefit from holistic approaches to assessment and intervention. The model was used for planning purposes, to address specific behavioral problems, and to help with transition planning from pre-school to elementary school, to middle school, to high school, and to postsecondary activities. In

1998, the model served as the consultation framework for TRIAD at Vanderbilt University as part of a contract to provide consultation across the state of Tennessee and was renamed the Collaborative Model for Promoting Competence and Success of Persons with Autism Spectrum Disorder (COMPASS).

Since 2004, federal funding from several National Institute of Health grants has enabled the authors to continue to evaluate the effectiveness of COMPASS for young children and for youth preparing for postsecondary activities. COMPASS has also been studied using web-based delivery. A professional development package for training COMPASS consultants is available to support its widespread use.

Rationale or Underlying Theory

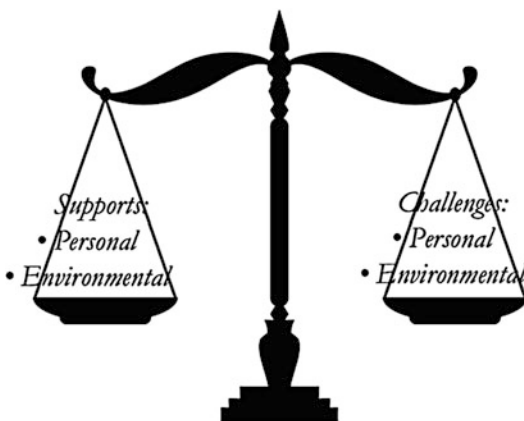
Unlike most ASD evidence-based practices that focus on a specific skill, the COMPASS framework for improved goal attainment outcomes is a comprehensive program that emphasizes interventions for the core underlying areas of social, communication, and learning skills instruction that can be targeted throughout the school day, over multiple activities and learning opportunities. Designed to bring together the people with the most frequent interactions with the student – parents, teachers, therapists, etc., COMPASS helps the team to jointly identify the key social, communication, and independent work/learning skills often missing in the educational programs of students with ASD yet have a pivotal impact on other areas of development. For example, a skill such as initiation impacts asking for help, starting a work task, and making social greetings. These pivotal goals must be identified and carefully crafted for the individual student and an evidenced-based intervention plan developed and modified based on the student’s needs, preferences, and strengths. Thus, COMPASS explicitly embraces and applies an Evidence-Based Practice in Psychology (EBPP) approach (McGrew et al. 2016), combining identification of the best empirically supported intervention with a consideration of the student’s needs and

preferences, together with the personnel and general resources available within the school, home, and community.

What differentiates COMPASS from traditional behavioral consultation is the focus on quality of life (QOL). Longitudinal outcome research in autism typically defines a good outcome when an individual achieves normal social development and independence. These outcomes, however, tend to misrepresent and underestimate the competencies and critical gains in ability that impact QOL. COMPASS is based on the developmental theory that competency is the result of reciprocal and dynamic interactions between individuals and their environments (i.e., transactional) (Ruble and Dalrymple 1996). Thus, by carefully examining and identifying environmental factors and introducing modifications to reduce individual risk factors and enhance protective factors, we can influence the development of important QOL skills (Fig. 1). Competence looks different across the lifespan and is person-specific. For example, transition to adulthood brings with it vocational decisions, as well as demands for more independent living skills and for establishing and creating lifelong supportive social connections. This requires specific instruction across social, communication, and emotional competencies to continue to be refined and utilized throughout adult life. Further, the extreme heterogeneity in ASD

requires a framework, like COMPASS, that can be helpful independent of the vast differences in language, cognitive, or social abilities that characterize the autism spectrum.

A consultation intervention is complex because it is a multilevel EBP. To conceptualize the levels, we have applied Dunst and Trivette's (2012) Framework for Evidence-Based Implementation and Intervention Practices (FEBIIP) as a model to understand COMPASS (Ruble and McGrew 2015). The FEBIIP includes three levels of assessment that differentiate between the three critical actors in consultation: the consultant, the consultees (parent, teacher, service provider), and the client (student). The first level focuses on quality assessment of the implementation practice or what the consultant does to impact the second level of the intervention practice or what the teacher, parent, and service provider does as a result of the consultant. This second level, the actual intervention, then impacts the practice outcome, indirectly in terms of what the student does as a result of the interventions delivered by the parent, teacher, and service provider, as well as the direct impacts of the three consultee actors on outcomes. These multilevel aspects account for the complexity within a consulting intervention such as COMPASS. For example, in prior research on COMPASS, we showed using serial mediation that COMPASS has an indirect impact on student IEP outcomes through changes in what the teacher does (i.e., teacher adherence) and in the identification of pivotal goals (i.e., IEP quality) (Wong et al. 2018) (Fig. 2).

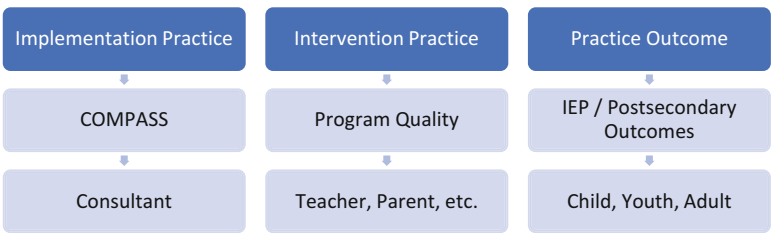


Goal Attainment of Students with ASD Using COMPASS, Fig. 1 Building competency by balancing challenges and supports

Goals and Objectives

The goal of COMPASS is to support teachers, parents, and other service providers with the implementation of high-quality and effective comprehensive interventions able to be provided in the community. As a consultation intervention, COMPASS helps spread expertise to where it is needed most so that children, youth, and adults achieve their optimal outcomes through the attainment of skills in the areas of social, communication, and learning.

Goal Attainment of Students with ASD Using COMPASS, Fig. 2 FEBIIP



Treatment Participants

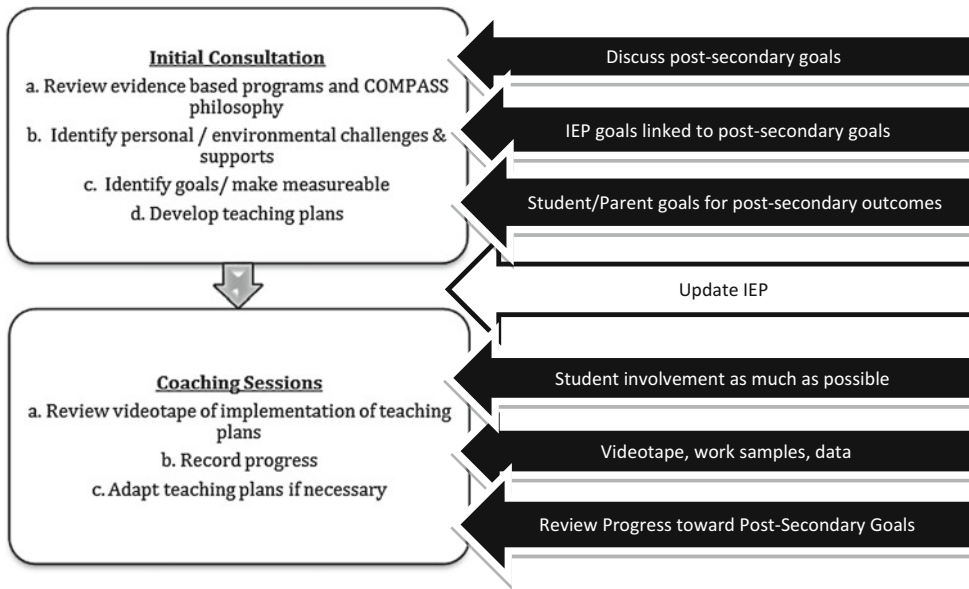
Everyone agrees that informed teachers and parents need support and access to ASD-specific research-based interventions that can be individualized for each student with ASD and result in meaningful outcomes. One means to this end is trained consultants, who can provide the “glue” that assembles the team to together. COMPASS assists parents and teachers in working together to create positive, meaningful, effective, and personalized programs for children and youth with ASD. The personalized approach of COMPASS supports the use of research-supported interventions. Effective implementation of COMPASS requires consultants who are competent about ASD and also able to provide effective consultation to successfully support parents and teachers and other service providers.

We have examined implementation factors associated with COMPASS outcomes at the teacher and student levels (Ruble and McGrew 2015). For example, teacher factors associated with greater goal attainment progress of children who receive COMPASS are fidelity of implementation of intervention plans, teaching quality, and decreased teacher burnout. With respect to student factors, for children with autism, child engagement, rather than IQ, language, ASD severity, or adaptive skills, accounted for goal attainment outcomes, suggesting COMPASS works equally for all children (Ruble and McGrew 2013). We also have evidence that COMPASS results in less child-related parent stress (Krakovich et al. 2016).

COMPASS is effective for improving educational outcomes for young children between 3 and 8 years of age and with high school-age students with ASD.

Treatment Procedures

The detailed procedures for implementing and evaluating COMPASS are manualized (Ruble et al. 2012a). COMPASS begins with an initial 3-hour joint session that allows for discussion between the team. Starting with the COMPASS Profile or joint summary form that summarizes independently obtained parent and teacher ratings on the student’s challenges and strengths related to social skills, adaptive/self-management, communication, problem behaviors, learning skills, and sensory avoidances and preferences, the consultant facilitates in-depth information sharing. This discussion helps pinpoint critical social, communication, and work behavior/learning goals and informs the intervention plans that are generated for each goal. Following this initial consultation are four 1-hour coaching sessions with the teacher that includes progress assessment and performance feedback monitoring. Each session is standardized and allows for assessment of student progress so that intervention modification and teacher self-reflection on the implementation of the intervention plans occurs. During coaching, teachers and students provide a video or artifact (grades, diaries) to determine student progress using psychometric equivalence tested goal attainment scaling (PET-GAS; Ruble et al. 2012b). PET-GAS represents an improvement over typical GAS, in that it controls for factors that may result in biased assessment of outcomes such as level of difficulty of the skill, measurability of the skill, and the distance between the benchmarks. After progress is determined, the implementation of the teaching plan and fidelity are reviewed. Supportive problem-solving occurs based on the performance feedback and fidelity



Goal Attainment of Students with ASD Using COMPASS, Fig. 3 COMPASS intervention

monitoring. Figure 3 shows the COMPASS activities in the white boxes for young children, and the black boxes show the adaptations for high school students.

Efficacy Information

COMPASS originally focused almost exclusively on the school setting and was developed and tested for young children with ASD. In two NIH-funded RCTs of COMPASS for young children (ages 3–8) with autism (Ruble et al. 2010; Ruble et al. 2013), COMPASS resulted in large effect sizes (Cohen's $d=1.5$; 1.1). Importantly, we were able to demonstrate success in extending COMPASS to geographically isolated areas through videoconferencing, addressing the need for service access for those in underserved areas. Next, we tested a parent-mediated version of COMPASS called **COMPASS for Hope (C-HOPE)**. C-HOPE was designed for parents of children with ASD and challenging behavior. C-HOPE was effective for decreasing child problem behavior ($p < 0.001$), increasing parent competency ($p = 0.02$), and decreasing parent stress

($p < 0.001$) (Kuravackel et al. 2018). The third intervention **COMPASS for Transition (COMPASS-T)** was tested in an RCT with high school students in their final year of school with a focus on improving IEP and postsecondary outcomes (Ruble et al. 2018). Students whose teachers received COMPASS made more progress on their IEP goals based on blinded goal attainment ratings, with a very high effect size (Cohen's $d = 2.1$) Consultants were able to deliver COMPASS-T with high fidelity, and similar to our studies of young children, teacher adherence to the implementation of teaching plans increased over time, meaning coaching sessions were essential for positive student outcomes. Further, parents and teachers reported high satisfaction; teachers reported COMPASS-T was acceptable, feasible, and not burdensome (Ruble et al. 2018).

Outcome Measurement

Data collection and progress monitoring are critical features of evidence-based interventions like COMPASS. But in special education, goals are individualized, and often the measurement

-2 Present Level of Performance	-1 Progress	0 Expected level of outcome (goal)	+1 Somewhat more than expected	+2 Much more than expected

Goal Attainment of Students with ASD Using COMPASS, Fig. 4 Example of a Goal Attainment Scale

approaches using standardized assessments are insensitive to interventions for school-age and older individuals (see Bacon et al. 2014) and are, therefore, not helpful for making data-informed decisions in practice. This is an issue that was recognized decades ago when special education researchers called for an alternative approach that allowed for assessment that could be repeated frequently and used for progress monitoring and instructional decision-making for a student with individualized goals (Carr 1979; Shuster et al. 1984).

When designing the randomized controlled trials to evaluate COMPASS, an outcome measurement tool was needed that not only had good psychometric properties (reliability, validity) when used to compare groups but also good sensitivity for detecting change and growth in skills as a result of personalized instructional plans developed from COMPASS (Ruble et al. 2019). Because of a need for a measurement approach that was broad, able to be used for any age, useful for goals that varied from child to child, meaningful, and socially valid for community-based interventions such as educational settings, goal attainment scaling (GAS) was selected. GAS is a common outcome assessment approach for intervention studies from both physical and mental health sciences and was described as a method for evaluating special education outcomes more than 40 years ago (Carr 1979). For consultation research GAS is the most frequently used method (Hurwitz et al. 2015; Sladeczek et al. 2001) for several types of research designs including single-case, quasi-experimental, and randomized controlled designs. For ASD research, GAS has been applied to several group design research

studies (c.f. Odom et al. 2013; Ruble et al. 2010; Ruble et al. 2013; Ruble et al. 2019). As mentioned earlier, we developed PET-GAS to resolve potential problems with measurement bias when using GAS when making between group comparisons.

A goal attainment scale traditionally has five benchmarks that range from −2 (present level of performance) to +2 (much more than expected). Details on how to create a GAS are available from Ruble et al. (2012a). After careful assessment of the student that is based on parent and teacher input, rating scales, observations, and other sources, a goal is developed that should be achievable by the end of the school year (0 level or expected level of outcome). After the goal is identified and developed, then benchmarks are written related to the goal (progress or −1; somewhat more than expected or +1; much more than expected or +2). Figure 4 shows an example of GAS.

Qualifications of Treatment Providers

Psychologists (including Rehabilitation, Clinical, Counseling, and School Psychologists); Speech and Language Pathologists; Occupational Therapists; Cognitive Rehabilitation Specialists; General and Special Educators; Vocational Rehabilitation Counselors and Life Care Planners

See Also

- COMPASS for Hope Training Program
- Education



- Family Burden
- Free Appropriate Public Education
- National Research Council
- Parent-Professional Partnership
- Social Interventions

References and Reading

- August, G. A., Anderson, D., & Bloomquist, M. L. (1992). Competence enhancement training for children: An integrated child, parent, and school approach. In S.-L. Christenson & J.-C. Conoley (Eds.), *Home-school collaboration: Enhancing children's academic and social competence* (pp. 175–192). Silver Spring: National Association of School Psychologists.
- Bacon, E. C., Dufek, S., Schreibman, L., Stahmer, A. C., Pierce, K., & Courchesne, E. (2014). Measuring outcome in an early intervention program for toddlers with autism spectrum disorder: Use of a curriculum-based assessment. *Autism Research and Treatment*, 2014, 964704. <https://doi.org/10.1155/2014/964704>.
- Carr, R. A. (1979). Goal attainment scaling as a useful tool for evaluating progress in special education. *Exceptional Children*, 46(2), 88–95.
- Dunst, C. J., & Trivette, C. M. (2012). Meta-analysis of implementation practice research. In B. Kelly & D. Perkins (Eds.), *Handbook of implementation science for psychology in education* (pp. 68–91). New York: Cambridge University Press.
- Hurwitz, J. T., Kratochwill, T. R., & Serlin, R. C. (2015). Size and consistency of problem-solving consultation outcomes: An empirical analysis. *Journal of School Psychology*, 53(2), 161–178. <https://doi.org/10.1016/j.jsp.2015.01.001>.
- Krakovich, T. M., McGrew, J. H., Yu, Y., & Ruble, L. A. (2016). Stress in parents of children with autism spectrum disorder: An exploration of demands and resources. *Journal of Autism and Developmental Disorders*, 46(6), 2042–2053. <https://doi.org/10.1007/s10803-016-2728-2>.
- Kuravackel, G. M., Ruble, L. A., Reese, R. J., Ables, A. P., Rodgers, A. D., & Toland, M. D. (2018). COMPASS for hope: Evaluating the effectiveness of a parent training and support program for children with ASD. *Journal of Autism and Developmental Disorders*, 48(2), 404–416. <https://doi.org/10.1007/s10803-017-3333-8>.
- McGrew, J., Ruble, L., & Smith, I. (2016). Autism spectrum disorder and evidence-based practice in psychology. *Clinical Psychology: Science and Practice*, 23(3), 239–255.
- Odom, S. L., Cox, A. W., & Brock, M. E. (2013). Implementation science, professional development, and autism spectrum disorders. *Exceptional Children*, 79(2), 233–251.
- Ruble, L.-A., & Dalrymple, N.-J. (1996). An alternative view of outcome in autism. *Focus on Autism and Other Developmental Disabilities*, 11(1), 3–14.
- Ruble, L., & McGrew, J. (2013). Teacher and child predictors of achieving IEP goals of children with autism. *Journal of Autism and Developmental Disorders*, 43, 2748–2763.
- Ruble, L., & McGrew, J. (2015). *COMPASS and implementation science: An evidence-based practice in psychology approach for improving educational outcomes of children with autism spectrum disorder*. New York: Springer.
- Ruble, L. A., Dalrymple, N. J., & McGrew, J. H. (2010). The effects of consultation on Individualized Education Program outcomes for young children with autism: The collaborative model for promoting competence and success. *Journal of Early Intervention*, 32(4), 286–301.
- Ruble, L., Dalrymple, N. J., & McGrew, J. H. (2012a). *Collaborative model for promoting competence and success for students with ASD*. New York: Springer.
- Ruble, L., McGrew, J. H., & Toland, M. D. (2012b). Goal attainment scaling as an outcome measure in randomized controlled trials of psychosocial interventions in autism. *Journal of Autism and Developmental Disorders*, 42(9), 1974–1983. <https://doi.org/10.1007/s10803-012-1446-7>.
- Ruble, L. A., McGrew, J. H., Toland, M. D., Dalrymple, N. J., & Jung, L. A. (2013). A randomized controlled trial of COMPASS web-based and face-to-face teacher coaching in autism. *Journal of Consulting and Clinical Psychology*, 81(3), 566–572. <https://doi.org/10.1037/a0032003>.
- Ruble, L. A., McGrew, J. H., Toland, M., Dalrymple, N., Adams, M., & Snell-Rood, C. (2018). Randomized control trial of COMPASS for improving transition outcomes of students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-018-3623-9>.
- Ruble, L., Wong, W. H., & McGrew, J. (2019). Data collection in education and measurement of progress. In R. Jordan, J. M. Roberts, & K. Hume (Eds.), *The SAGE handbook of autism and education* (pp. 578–592). Los Angeles: Sage.
- Shuster, S. K., Fitzgerald, N., Shelton, G., Barber, P., & Desch, S. (1984). Goal attainment scaling with moderately and severely handicapped preschool children. *Journal of Early Intervention*, 8(1), 26–37. <https://doi.org/10.1177/105381518400800104>.
- Sladeczek, I. E., Elliott, S. N., Kratochwill, T. R., Robertson Mjaanes, S., & Stoiber, K. C. (2001). Application of goal attainment scaling to a conjoint behavioral consultation case. *Journal of Educational and Psychological Consultation*, 12(1), 45–58.
- Wong, V., Ruble, L. A., McGrew, J. H., & Yu, Y. (2018). An empirical study of multidimensional fidelity of COMPASS consultation. *School Psychology Quarterly*, 33(2), 251–263. <https://doi.org/10.1037/spq0000217>.

Goldenhar Syndrome

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Synonyms

Craniofacial microsomia; Hemifacial microsomia; Oculoauriculovertebral (OAV) spectrum or dysplasia

Short Description or Definition

Goldenhar Syndrome is a congenital condition affecting the development of the eye, ear, and spine. In most cases, the syndrome affects only one side of the body, but both sides of the body can be affected. Individuals with Goldenhar Syndrome have facial asymmetry, underdeveloped or absent ear, benign growths in the eye called epibulbar dermoids or lipodermoids, and benign growths or skin tags on the ear, called preauricular appendages. Abnormalities of the spine can manifest in scoliosis, as well as vertebrae or ribs that are missing, partially formed, or abnormally fused. Problems of the heart, lungs, kidneys, and central nervous system are also common. The root cause of the syndrome is unknown, but it is associated with abnormal development of the first and second branchial arches of the fetus, which develop to form the neck and head.

Categorization

Goldenhar syndrome is part of a complex group of conditions characterized by underdeveloped jaw, facial asymmetry, benign growths or skin tags on the ear, and underdevelopment or absence of the ear (Heike et al. 2009). The relationship of these conditions to each other is not well defined; they may be distinct conditions or may describe different severities of the same

condition. Other conditions in this group include hemifacial microsomia and oculoauriculovertebral (OAV) spectrum. Although these terms can be used interchangeably, they can also refer to specific combinations of craniofacial abnormalities. Goldenhar Syndrome is characterized by these craniofacial abnormalities in addition to spinal malformations and benign growths of the eye (Gorlin et al. 2001).

Epidemiology

Goldenhar Syndrome is rare; therefore, it is difficult to estimate prevalence. Some have estimated the prevalence as high as 1 in 5600 births (Gorlin et al. 2001; Grabb 1965), while others place it as low as 1 in 45,000 (Morrison et al. 1992). Males are more likely to be affected, with the male to female ratio estimated at 3:2 (Gorlin et al. 2001). Most people with Goldenhar syndrome are the only person in their family to be affected, but there have been cases in which individuals have a chromosome abnormality, in which case the condition might be inherited. Cases of both autosomal dominance and recessive inheritance have been recorded (Heike et al. 2009).

Natural History, Prognostic Factors, Outcomes

In 1952, Maurice Goldenhar coined the term Goldenhar syndrome, including in its description the characteristic preauricular appendages, as well as epibulbar dermoids and mandibular hypoplasia, or underdevelopment of the jaw. Since then, additional symptoms have been recognized as representative of Goldenhar syndrome, namely, vertebral abnormalities and underdevelopment or absence of the ear (Feingold and Baum 1978; Gorlin et al. 1963). Current thinking varies on whether Goldenhar syndrome is a distinct syndrome or whether it is simply a severe phenotype on a spectrum of facial abnormalities. Some classify it as a phenotype included under the umbrella term craniofacial microsomia (Heike et al. 2009). Others use the term OAV spectrum to denote a

“symptom-complex” that includes Goldenhar syndrome as well as hemifacial microsomia (Gorlin et al. 1963, 2001).

Goldenhar syndrome can range from mild to severe. More mild cases may need no other treatment than careful monitoring. Children with Goldenhar syndrome have been seen to be shorter than their peers and may have delayed motor development or hearing loss (Bogusiak et al. 2017). Some speech disorders have been observed, as well as psychosocial problems and behaviors commonly associated with autism (Barton and Volkmar 1998; Bogusiak et al. 2017; Maris et al. 1999). Individuals with Goldenhar syndrome may also exhibit breathing problems, including tachypnea and sleep apnea, and feeding problems (Gorlin et al. 2001). Cleft lip, face, or palate is also commonly associated with Goldenhar syndrome. More severe expression of the disorder is associated with cerebral and cranial defects, which often are associated with intellectual disability and higher mortality (Gorlin et al. 2001; Setzer et al. 1981). Congenital heart disease is common in Goldenhar syndrome; tetralogy of fallow (TOF) and atrial and ventricular septal defects have been found to be the most common, but many types of heart problems have been seen (Digilio et al. 2008).

Clinical Expression and Pathophysiology of Goldenhar Syndrome

Clinical presentation of Goldenhar syndrome is highly variable and ranges from mild to severe. It is congenital, meaning it is present at birth. The literature is mixed and no clear diagnostic criteria have been defined for Goldenhar syndrome, but the general clinical expression is characterized by abnormalities in the eye, ear, jaw, and spine (Bogusiak et al. 2017; Feingold and Baum 1978; Gorlin et al. 2001; Heike et al. 2009). Goldenhar syndrome is set apart from other craniofacial conditions by the presence of epibulbar dermoids or lipodermoids in the eye. These are benign growths that are yellow or milky white in appearance and can range in size. Some individuals with

Goldenhar syndrome have also presented with underdevelopment or absence of an eye (Gorlin et al. 1963). Mandibular or maxillary hypoplasia or other developmental issues in the bone structure of the face are often present (Feingold and Baum 1978; Gorlin et al. 2001; Heike et al. 2009). Preauricular or auricular appendages, skin tags near or on the ear, are characteristic of Goldenhar syndrome, and malformation or absence of one or both ears is common (Gorlin et al. 2001; Heike et al. 2009). Additionally, some malformation of the inner or middle ear can be seen, with or without hearing loss. Facial asymmetry is a hallmark of Goldenhar syndrome and is often due to hypoplasia, or underdevelopment, of bones in one side of the face (Bogusiak et al. 2017). Often symptoms appear only on one side of the face, also contributing to facial asymmetry. In some cases, symptoms have appeared on both sides; in these cases, one side is often more severe (Gorlin et al. 2001). Finally, vertebral anomalies are characteristic of Goldenhar syndrome. Other symptoms not previously associated with Goldenhar syndrome, including central nervous system abnormalities, are beginning to be recognized as characteristic of the disorder, providing further evidence to support the idea of a spectrum (Gorlin et al. 2001). Goldenhar syndrome may become more severe as the individual grows older (Bogusiak et al. 2017).

Evaluation and Differential Diagnosis

Literature is mixed on the differentiation of Goldenhar syndrome from hemifacial or craniofacial microsomia, or whether Goldenhar syndrome is even distinct from these conditions. Some require the presence of epibulbar dermoids to differentiate Goldenhar syndrome from hemifacial microsomia (Gorlin et al. 1963). Some consider Goldenhar syndrome to be a phenotype under the umbrella term craniofacial microsomia. In this case, this condition is a diagnosis by exclusion, meaning it is diagnosed by ruling out disorders with similar symptoms. A common difference between Goldenhar

syndrome and other syndromes is that Goldenhar syndrome is characterized by facial asymmetry, while other craniofacial abnormalities are symmetric (Heike et al. 2009). Because of the lack of specified diagnostic criteria, many syndromes must be taken into consideration before an individual will receive a diagnosis of Goldenhar syndrome (for a comprehensive review, see Heike et al. 2009). Some examples of these syndromes are Treacher Collins syndrome, CHARGE association, and Branchio-oto-renal syndrome. Treacher Collins syndrome presents similarly to Goldenhar Syndrome, especially in underdevelopment of the ears but lacks the characteristic facial asymmetry. CHARGE association does present asymmetrically but lacks facial or preauricular appendages, as well as vertebral anomalies. Similarly, Branchio-oto-renal syndrome presents asymmetrically without vertebral anomalies; however, it can present with facial or preauricular appendages. It also usually will not include cardiac abnormalities (Heike et al. 2009). In cases such as Cat-eye syndrome and Townes-Brocks syndrome, the phenotype of the syndrome is exactly the same and so can only be differentiated through a genetic test (Heike et al. 2009).

Treatment

Because the presentation of Goldenhar syndrome is highly variable, treatment depends on the specific symptoms of the individual and often develops and changes as the individual grows older.

Due to craniofacial abnormalities, individuals with Goldenhar syndrome often have difficulty breathing and may suffer from breathing-related conditions such as tachypnea and sleep apnea, among others. A newborn diagnosed with Goldenhar syndrome or any form of craniofacial microsomia should be evaluated immediately for airway obstruction, and airway interventions will be performed in the first year of life. At the time of diagnosis, the individual should have a renal evaluation as well as a cardiac evaluation; some issues

may require immediate treatment, while some are less urgent (Heike et al. 2009).

Surgery may also be necessary early in life to correct cleft lip, cleft palate, or cleft face and to remove facial, preauricular, or auricular appendages. If cleft lip, cleft palate, or cleft face is present, the newborn will often have trouble feeding, so feeding interventions may also be performed (Bogusiak et al. 2017; Heike et al. 2009). The individual will need hearing evaluations and may require interventions such as hearing prosthetics or cochlear implants (Bogusiak et al. 2017).

More surgery may be required as the individual grows older to correct any spinal abnormalities or scoliosis, to correct dental issues, to rebuild ears or to create a prosthetic ear, and to remove epibulbar dermoids if they impair vision (Bogusiak et al. 2017). Finally, due to the higher likelihood of an individual with Goldenhar syndrome experiencing psychosocial difficulties, psychiatric or neurological evaluations and treatment may be necessary. The individual may also require speech therapy for any speech problems (Heike et al. 2009).

See Also

- ▶ [CATCH 22 \(Chromosome 22q11 Deletion Syndrome\)](#)
- ▶ [Developmental Delay](#)
- ▶ [Velocardiofacial Syndrome](#)

References and Reading

- Barton, M., & Volkmar, F. (1998). How commonly are known medical conditions associated with autism? *Journal of Autism and Developmental Disorders*, 28(4), 273–278.
- Bogusiak, K., Puch, A., & Arkuszewski, P. (2017). Goldenhar syndrome: current perspectives. *World Journal of Pediatrics*, 13(5), 405–415.
- Digilio, M. C., Calzolari, F., Capolino, R., Toscano, A., Sarkozy, A., de Zorzi, A., ... & Marino, B. (2008). Congenital heart defects in patients with oculo-auriculo-vertebral spectrum (Goldenhar syndrome). *American Journal of Medical Genetics Part A*, 146(14), 1815–1819.

- Feingold, M., & Baum, J. (1978). Goldenhar's syndrome. *American Journal of Diseases of Children*, 132(2), 136–138.
- Gorlin, R. J., Jue, K. L., Jacobsen, U., & Goldschmidt, E. (1963). Oculoauriculovertebral dysplasia. *The Journal of Pediatrics*, 63(5), 991–999.
- Gorlin, R. J., Cohen, M. M., Jr., & Hennekam, R. C. (2001). *Syndromes of the head and neck*. New York: Oxford University Press.
- Grabb, W. C. (1965). The first and second branchial arch syndrome. *Plastic and Reconstructive Surgery*, 36(5), 485–508.
- Heike, C. L., Luquetti, D. V., & Hing, A. V. (2009). Craniofacial Microsomia Overview. In M. P. Adam, H. H. Ardinger, R. A. Pagon, et al. (Eds.), *GeneReviews® [Internet]*. Seattle: University of Washington, Seattle; 1993–2018. Updated 9 Oct 2014.
- Maris, C. L., Endriga, M. C., Omnell, M. L., & Speltz, M. L. (1999). Psychosocial adjustment in twin pairs with and without hemifacial microsomia. *The Cleft Palate-Craniofacial Journal*, 36(1), 43–50.
- Morrison, J., Mulholland, H. C., Craig, B. G., & Nevin, N. C. (1992). Cardiovascular abnormalities in the oculo-auriculo-vertebral spectrum (Goldenhar syndrome). *American Journal of Medical Genetics Part A*, 44(4), 425–428.
- Setzer, E. S., Ruiz-Castaneda, N., Severn, C., Ryden, S., & Frias, J. L. (1981). Etiologic heterogeneity in the oculoauriculovertebral syndrome. *The Journal of Pediatrics*, 98(1), 88–90.

Grammar

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Definition

Grammar, the framework for a language, consists of *syntax* (the way words are combined to convey meaning) and *morphology* (the forms of words) of a particular language.

Morphology is the system that governs the structure of words and the construction of word forms (e.g., nouns, verbs, adjectives). Morphology includes how morphemes (the smallest unit of meaning) are used to develop the rules of word formation in English, such

as adding an *s* to indicate a plural (e.g., *books*) or an *'s* to indicate possessive (e.g., *mommy's*), the use of prefixes, the use of suffixes, and so forth.

Syntax is the system governing the order and combination of words to form sentences and the relationships among the elements within a sentence (e.g., subject, object).

Individuals with speech and language disorders may have difficulty – either singly or in combination – with grammar. Speech-language pathologists assess, diagnose, and treat communication and related problems, including grammar problems.

See Also

► [Syntax](#)

References and Reading

- American Speech-Language-Hearing Association. (1993). *Definitions of communication disorders and variations* [Relevant Paper]. Retrieved December 14, 2010, from www.asha.org/policy

Grammatical Morphemes

► [Speech Morphology](#)

Grand Mal Seizure

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Synonyms

[Tonic-clonic seizure](#)

Definition

Although this term was replaced by “tonic-clonic seizure” in 1981 (Commission on Classification and Terminology of the International League Against Epilepsy 1981), it is still sometimes used, perhaps by those who are not aware of the change in terminology. The term “tonic-clonic seizure” should be used not only because it is the current accepted terminology but also because it describes what happens. In this context, the word “tonic” means stiff and the word “clonic” means jerking. A tonic-clonic seizure is one in which the person is stiff and jerking. When the stiffening includes the respiratory muscles, a cry may be emitted at the beginning of the seizure and cyanosis may occur. The tongue is sometimes bitten. Incontinence of urine may occur. Injuries may be sustained if the patient falls. In the clonic phase, there may be copious salivation, sometimes with frothing at the mouth. If the tonic-clonic seizure is preceded by a simple partial seizure (consciousness fully retained) and/or a complex partial seizure (some impairment of consciousness), it is a secondarily generalized tonic-clonic seizure. In contrast, if there is no preceding partial seizure, it is a primary generalized tonic-clonic seizure. The sequence of events in a tonic-clonic seizure is that typically the stiffening occurs first and lasts for around 20 s, followed by rhythmical jerking, which lasts for around 45 s (Theodore et al. 1994). However, seizures can be very variable. In some people, both the stiffening and jerking appear to be present at the start of the seizure. The duration of tonic-clonic seizures can also vary greatly from one patient to another. The tonic-clonic seizure and indeed epilepsy are clinical diagnoses based on a clear description from someone who has seen the episodes. The EEG can support diagnosis but is no substitute for a good clinical history. The sequence of events in the EEG during a tonic-clonic seizure can vary from one person to another. The electroencephalogram (EEG) can also vary greatly from one individual to another. A typical situation would be that the EEG would be obscured by muscle artifact, but if the filters

allowed the underlying pattern to become evident, this would show repetitive spikes with increasing amplitude and with increasing frequency all over the brain during the tonic phase. In the clonic (jerking) phase, slow waves appear and the spikes become less frequent. After the seizure, the EEG may show nonspecific slow-wave activity in a generally suppressed record. Prolonged tonic-clonic seizures, lasting 20 min or longer (status epilepticus), are uncommon but constitute a medical emergency requiring prompt treatment to decrease the risk of permanent brain damage. The tonic-clonic seizure is one of the many types of seizures that can occur in people with autism. Estimates of the prevalence of epilepsy in people with autism vary from about 20% to 40%. The question of whether epilepsy is more common than in the general population in people with autism who do not also have learning disability (mental retardation) has been debated, but most of the evidence suggests that epilepsy primarily occurs in those who have both autism and learning disability. The relationships between epilepsy and autism continue to be debated (Besag 2009).

See Also

- ▶ Autism
- ▶ Electroencephalogram (EEG)
- ▶ Epilepsy
- ▶ Learning Disability

References and Reading

- Besag, F. M. (2009). The relationship between epilepsy and autism: A continuing debate. *Acta Paediatrica*, 98, 618–620.
- Commission on Classification and Terminology of the International League Against epilepsy. (1981). Proposal for revised clinical and electroencephalographic classification of epileptic seizures. *Epilepsia*, 22, 489–501.
- Theodore, W. H., Porter, R. J., Albert, P., Kelley, K., Bromfield, E., Devinsky, O., et al. (1994). The secondarily generalized tonic-clonic seizure: A videotape analysis. *Neurology*, 44, 1403–1407.

Grandin, Temple

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Name and Degrees

Temple Grandin

- 1966 B.A. Franklin Pierce College, Concord, New Hampshire
- 1975 M.A. Animal Science Arizona State University, Phoenix, Arizona
- 1989 Ph.D. Animal Science, University of Illinois, Urbana-Champaign

Major Appointments (Institution, Location, Dates)

- Professor of Animal Science, Colorado State University, Fort Collins, CO

Major Honors and Awards

- 2010 Honorary Doctoral Degree, Duke University, Durham North Carolina

Landmark Clinical, Scientific, and Professional Contributions

Temple Grandin, Ph.D., is an animal scientist and widely known for her writing and lectures on her experience of autism. Her book *Thinking in Pictures* provides a personal and highly illuminating view of the world from the perspective of a highly able person with autism. She has written vividly about the contribution of sensory experience in autism. In addition to her advocacy, she has had an interest in approaches to treatment that involve sensory experiences. She

has also been a prominent advocate for animal welfare.

Short Biography

Born in Boston, Temple Grandin was diagnosed with autism in 1950. At that time, treatment options were limited, but she received a structured school program and communication therapy. By age 5, she was talking but recalls experiences of feeling isolated. She attended a boarding school for gifted children before going on to do undergraduate in psychology and then graduate work in animal science. In addition to her work on animal management, she has written several volumes on autism including books concerned with visual thinking, the use of pressure sensation as an intervention technique, and an autobiography.

See Also

- [Occupational Therapy \(OT\)](#)

References and Reading

- Grandin, T. (1990). Needs of high functioning teenagers and adults with autism: Tips from a recovered autistic. *Focus on Autistic Behavior*, 5(1), 16 p.
- Grandin, T. (1992). Calming effects of deep touch pressure in patients with autistic disorder, college students, and animals. *Journal of Child & Adolescent Psychopharmacology*, 2(1), 63–72.
- Grandin, T. (1995). *Thinking in pictures: And other reports from my life with autism*. New York: Doubleday.
- Grandin, T. (2005). A personal perspective of autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, pp. 1276–1286). Hoboken: Wiley.
- Grandin, T., & Johnson, C. (2005). *Animals in translation: Using the mysteries of autism to decode animal behavior*. New York: Scribner/Simon & Schuster.
- Grandin, T., & Scariano, M. M. (1986). *Emergence: Labeled autistic*. New York: Warner Books.

Grandma's Rule

- [Premack Principle](#)

Grants

► [FAFSA](#)

Gray Matter

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Synonyms

[Gray substance](#); [Substantia grisea](#)

Definition

Gray matter refers to the portion of the central nervous system composed of neuronal cell bodies, neuropil, glial cells, and capillaries. This region is so named because of its gray color (in contrast to white matter). Gray matter can be found in the cerebral cortex as well as in the thalamus, basal ganglia, and cerebellum.

See Also

► [Cerebral Cortex](#)

References and Reading

- Palmen, S. J. M. C., Pol, H. E. H., Kemner, C., Schnack, H. G., Durston, S., Lahuis, B. E., et al. (2005). Increased gray-matter volume in medication-naïve high-functioning children with autism spectrum disorder. *Psychological Medicine*, 35, 561–570.
- Rojas, D. C., Peterson, E., Winterrowd, E., Reite, M. L., Rogers, S. L., & Tregellas, J. R. (2006). Regional gray matter volumetric changes in autism associated with social and repetitive behavior symptoms. *BMC Psychiatry*, 6, 56.

Gray Substance

► [Gray Matter](#)

Gross Motor Skills

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Definition

Gross motor abilities entail the use of large muscle groups that coordinate body movements to perform activities such as maintaining balance, walking, sitting upright, jumping, throwing objects, etc. Gross motor skills are acquired during infancy and early childhood as part of a child's motor development and continue to be refined throughout most of the individual's years of development into adulthood. Factors that contribute to the ability and the rate that children develop their gross motor skills include both genetic and environmental influences.

The development of gross motor abilities occurs in the motor cortex, the region of the cerebral cortex that controls voluntary muscle groups.

The development of motor autonomy is the infant's main task in the first year of life. The process can be described as a series of distinct stages, where mastering each stage prepares the infant's progression to the next one. At the beginning of each stage, movement is characterized by a persistent lack of symmetry. As the stage progresses, movement becomes increasingly symmetric. During each stage, complex reflex

patterns appear and are integrated with those already existing. By 2 years of age, almost all children are able to stand up, walk, jump, walk upstairs and downstairs, and run.

Difficulties in gross motor abilities are observed in a range of conditions and have been documented in individuals with autism spectrum disorders (ASDs; Dewey et al. 2007; Lloyd et al. 2011).

Historical Background

Although both Kanner (1943) and Asperger (1944/1991) reported signs of gross motor impairment in their seminal descriptions of patients with autism, the issue of motor difficulties in this population was overlooked, until recently, in favor of research on the social and communicative deficits in this population. After 1990, and for the successive two decades, there has been a growing recognition that children with autism do experience motor difficulties (Ghaziuddin and Butler 1998; Zingerevich et al. 2009). Furthermore, the development of gross motor skills in autism and its utility as precursory sign has increasingly gained attention (Jansiewicz et al. 2006). Currently, the examination of motor abnormalities is considered to be a valuable tool for understanding the neurological basis of autism, as motor signs are easily observable and quantifiable, and they can be examined in a manner that minimizes the potential confounds of social and communicative impairments (Wodka and Mostofsky 2011). Moreover, the importance of developing specific remediation strategies to target motor difficulties is increasingly acknowledged in clinical practice (Bhat et al. 2011). Recent research is focusing on motor development (Kim 2008), motor learning (Dowell et al. 2009), motor control (Rinehart et al. 2006), preambulatory movement (Esposito et al. 2009, 2011), and the relation between motor and social-communicative symptoms in autism (Gallese et al. 2009; Gernsbacher et al. 2008; LeBarton and Iverson 2016; Setoh et al. 2017).

Current Knowledge

A range of motor atypicalities have been documented in individuals with autism spectrum disorders, including delayed walking; abnormalities in gait (e.g., toe walking), posture, balance, coordination, tone, and difficulties in higher-level motor learning tasks involving imitation and pantomime of complex gestures (Dewey et al. 2007; Gowen and Hamilton 2013; Minshew et al. 2004; Staples and Reid 2010). While motor abnormalities are not considered as a crucial feature in the diagnostic process and are not a main focus of most intervention models, literature suggests that motor impairments might be observed in individuals with autism across the spectrum and across developmental levels and chronological ages (Fournier et al. 2010). Whether motor anomalies are a universal and/or specific symptom of autism is still debated (Ozonoff et al. 2008). Similarly, it is still debated whether motor abnormalities are present and identifiable in the first year of life (Esposito et al. 2009, 2011; Ozonoff et al. 2008). This issue has far-reaching relevance with regard to early diagnosis and identification of autism. Studies investigating gross motor abilities in very young children with autism indicated that delays and/or atypical patterns in gross motor development are present between 12 and 36 months and that these differences become more pronounced with age in preschool years (Landa and Garrett-Mayer 2006; Lloyd et al. 2011). A study by Suter and colleagues found that motor difficulties at age 2 years was the clearest distinguishing factor for identifying the children who continued to meet criteria for an ASD at age of 4 years (Suter et al. 2007). Additionally, early gross motor skills have been found to predict subsequent development of language in children with ASD and those at risk for ASD (Bedford et al. 2016; Leonard et al. 2015), and preliminary evidence suggests a link between early gross motor abnormalities and social communication development in ASD (Bhat et al. 2012; LeBarton and Iverson 2016;

see also Barbeau et al. 2015). Given the paucity of studies that investigated the development of motor abilities through childhood and adulthood, it is not clear whether motor difficulties present with different features at different ages and/or whether they improve over time. More empirical research is needed to identify the age of onset and the developmental pattern of motor difficulties in autism, as well as relationship between gross motor development and later social communication abilities in these children. Recent research suggested that motor abnormalities observed in autism might reflect a developmental dyspraxia, that is, a deficit in the execution of skilled movements caused by difficulties in forming the sensory representation of the motor plan that is necessary to execute the actions (Mostofsky and Ewen 2011; Dziuk et al. 2007). While little is known about the etiology of motor impairment in autism, functional and anatomical abnormalities, as well as abnormal connectivity in the brain systems underlying motor planning and execution, have been documented in autism (Mostofsky et al. 2009; Nayate et al. 2005; Nebel et al. 2014; Qiu et al. 2010; Mostofsky et al. 2007).

Future Directions

Current studies emphasizing the involvement of the motor system in action understanding and social cognition provide an exciting avenue for future research in the field (Rizzolatti and Sinigaglia 2007; Giorello and Sinigaglia 2007). Moreover, longitudinal studies involving infants at risk for autism is starting to provide crucial information on the nature of motor impairment in autism and its relationship with social and communicative core symptoms and other motor abnormalities such as stereotyped movements (Leonard et al. 2015; Van Waelvelde et al. 2010). The increasing recognition of the importance of motor abilities in autism is leading practitioners in the field to include increasingly refined motor assessments and programs in their clinical practice.

See Also

- ▶ Apraxia
- ▶ Dyspraxia
- ▶ Early Diagnosis
- ▶ Imitation
- ▶ Mirror Neuron System
- ▶ Motor Control
- ▶ Motor Planning
- ▶ Occupational Therapy (OT)
- ▶ Praxis
- ▶ Toe Walking

References and Reading

- Asperger, H. (1944). Die "Autistischen psychopathen" im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136. Translated by Frith, U. (Ed.). (1991). *Autism and Asperger's syndrome* (pp. 37–92). Cambridge: Cambridge University Press.
- Baranek, G. T. (2002). Efficacy of sensory and motor interventions for children with autism. *Journal of Autism and Developmental Disorders*, 32(5), 397–422.
- Barbeau, E. B., Meilleur, A. A. S., Zeffiro, T. A., & Mottron, L. (2015). Comparing motor skills in autism spectrum individuals with and without speech delay. *Autism Research*, 8(6), 682–693.
- Bedford, R., Pickles, A., & Lord, C. (2016). Early gross motor skills predict the subsequent development of language in children with autism spectrum disorder. *Autism Research*, 9, 993–1001.
- Bhat, A. N., Landa, R. J., & Galloway, J. C. (2011). Current perspectives on motor functioning in infants, children, and adults with autism spectrum disorders. *Physical Therapy*, 91, 1116–1129.
- Bhat, A. N., Galloway, J. C., & Landa, R. J. (2012). Relation between early motor delay and later communication delay in infants at risk for autism. *Infant Behavior and Development*, 35(4), 838–846.
- Bishop, S. L., Thurm, A., Farmer, C., & Lord, C. (2016). Autism spectrum disorder, intellectual disability, and delayed walking. *Pediatrics*, 137(3), e20152959.
- Dewey, D., Cantell, M., & Crawford, S. G. (2007). Motor and gestural performance in children with autism spectrum disorders, developmental coordination disorder, and/or attention deficit hyperactivity disorder. *Journal of International Neuropsychological Society*, 13(2), 246–256.
- Dowell, L. R., Mahone, E. M., & Mostofsky, S. H. (2009). Associations of postural knowledge and basic motor skill with dyspraxia in autism: Implication for abnormalities in distributed connectivity and motor learning. *Neuropsychology*, 23(5), 563–570.

- Dziuk, M. A., Gidley Larson, J. C., Apostu, A., Mahone, E. M., Denckla, M. B., & Mostofsky, S. H. (2007). Dyspraxia in autism: Association with motor, social, and communicative deficits. *Developmental Medicine and Child Neurology*, 49(10), 734–739.
- Esposito, G., Venuti, P., Maestro, S., & Muratori, F. (2009). An exploration of symmetry in early autism spectrum disorders: Analysis of lying. *Brain & Development*, 31(2), 131–138.
- Esposito, G., Venuti, P., Apicella, F., & Muratori, F. (2011). Analysis of unsupported gait in toddlers with autism. *Brain Development*, 33(5), 367–373.
- Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism spectrum disorders: A synthesis and meta-analysis. *Journal of Autism and Developmental Disorders*, 40(10), 1227–1240.
- Gallese, V., Rochat, M., Cossu, G., & Sinigaglia, C. (2009). Motor cognition and its role in the phylogeny and ontogeny of action understanding. *Developmental Psychology*, 45(1), 103–113.
- Gernsbacher, M. A., Sauer, E. A., Geye, H. M., Schweigert, E. K., & Hill Goldsmith, H. (2008). Infant and toddler oral- and manual-motor skills predict later speech fluency in autism. *Journal of Child Psychology and Psychiatry*, 49(1), 43–50.
- Ghaziuddin, M., & Butler, E. (1998). Clumsiness in autism and Asperger syndrome: A further report. *Journal of Intellectual Disability Research*, 42(Pt 1), 43–48.
- Giorello, G., & Sinigaglia, C. (2007). Perception in action. *Acta Bio-Medica*, 78(Suppl 1), 49–57.
- Gowen, E., & Hamilton, A. (2013). Motor abilities in autism: A review using a computational context. *Journal of Autism and Developmental Disorders*, 43(2), 323–344.
- Gutman, S. A., Raphael, E. I., Ceder, L. M., Khan, A., Timp, K. M., & Salvant, S. (2010). The effect of a motor-based, social skills intervention for adolescents with high-functioning autism: Two single-subject design cases. *Occupational Therapy International*, 17(4), 188–197.
- Jansiewicz, E. M., Goldberg, M. C., Newschaffer, C. J., Denckla, M. B., Landa, R., & Mostofsky, S. H. (2006). Motor signs distinguish children with high functioning autism and Asperger's syndrome from controls. *Journal of Autism and Developmental Disorders*, 36(5), 613–621.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kim, H. U. (2008). Development of early language and motor skills in preschool children with autism. *Perceptual and Motor Skills*, 107(2), 403–406.
- Landa, R., & Garrett-Mayer, E. (2006). Development in infants with autism spectrum disorders: A prospective study. *Journal of Child Psychology and Psychiatry*, 47(6), 629–638.
- LeBarton, E. S., & Iverson, J. M. (2016). Associations between gross motor and communicative development in at-risk infants. *Infant Behavior and Development*, 44, 59–67.
- Leonard, H. C., Bedford, R., Pickles, A., Hill, E. L., & BASIS Team. (2015). Predicting the rate of language development from early motor skills in at-risk infants who develop autism spectrum disorder. *Research in Autism Spectrum Disorders*, 13, 15–24.
- Lloyd, M., Macdonald, M., & Lord, C. (2011). Motor skills of toddlers with autism spectrum disorders. *Autism*. doi:10.1177/1362361311402230.
- Minshew, N. J., Sung, K., Jones, B., & Furman, J. (2004). Underdevelopment of the postural control system in autism. *Neurology*, 63(11), 2056–2061.
- Mostofsky, S. H., & Ewen, J. B. (2011). Altered connectivity and action model formation in autism is autism. *The Neuroscientist*, 17, 437–448.
- Mostofsky, S. H., Burgess, M. P., & Gidley Larson, J. C. (2007). Increased motor cortex white matter volume predicts motor impairment in autism. *Brain*, 130 (Pt 8), 2117–2122.
- Mostofsky, S. H., Powell, S. K., Simmonds, D. J., Goldberg, M. C., Caffo, B., & Pekar, J. J. (2009). Decreased connectivity and cerebellar activity in autism during motor task performance. *Brain*, 132 (Pt 9), 2413–2425.
- Nayate, A., Bradshaw, J. L., & Rinehart, N. J. (2005). Autism and Asperger's disorder: Are they movement disorders involving the cerebellum and/or basal ganglia? *Brain Research Bulletin*, 67(4), 327–334.
- Nebel, M. B., Joel, S. E., Muschelli, J., Barber, A. D., Caffo, B. S., Pekar, J. J., & Mostofsky, S. H. (2014). Disruption of functional organization within the primary motor cortex in children with autism. *Human Brain Mapping*, 35(2), 567–580.
- Ozonoff, S., Young, G. S., Goldring, S., Greiss-Hess, L., Herrera, A. M., Steele, J., et al. (2008). Gross motor development, movement abnormalities, and early identification of autism. *Journal of Autism and Developmental Disorders*, 38(4), 644–656.
- Qiu, A., Adler, M., Crocetti, D., Miller, M. I., & Mostofsky, S. H. (2010). Basal ganglia shapes predict social, communication, and motor dysfunctions in boys with autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(6), 539–551, e531–e534.
- Rinehart, N. J., Tonge, B. J., Iansek, R., McGinley, J., Brereton, A. V., Enticott, P. G., et al. (2006). Gait function in newly diagnosed children with autism: Cerebellar and basal ganglia related motor disorder. *Developmental Medicine and Child Neurology*, 48(10), 819–824.
- Rizzolatti, G., & Sinigaglia, C. (2007). Mirror neurons and motor intentionality. *Functional Neurology*, 22(4), 205–210.
- Setoh, P., Marschik, P. B., Einspieler, C., & Esposito, G. (2017). Autism spectrum disorder and early motor abnormalities: Connected or coincidental companions? *Research in Developmental Disabilities*, 60, 13–15.

- Staples, K. L., & Reid, G. (2010). Fundamental movement skills and autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(2), 209–217.
- Sutera, S., Pandey, J., Esser, E. L., Rosenthal, M. A., Wilson, L. B., Barton, M., et al. (2007). Predictors of optimal outcome in toddlers diagnosed with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(1), 98–107.
- Van Waelvelde, H., Oostra, A., Dewitte, G., Van Den Broeck, C., & Jongmans, M. J. (2010). Stability of motor problems in young children with or at risk of autism spectrum disorders, ADHD, and/or developmental coordination disorder. *Developmental Medicine and Child Neurology*, 52(8), e174–e178.
- Wodka, E., & Mostofsky, S. H. (2011). Motor development and its relation to social and behavioral manifestations. In D. Fein (Ed.), *The neuropsychology of autism*. New York: Oxford University Press.
- Zingerevich, C., Greiss-Hess, L., Lemons-Chitwood, K., Harris, S. W., Hessel, D., Cook, K., et al. (2009). Motor abilities of children diagnosed with fragile X syndrome with and without autism. *Journal of Intellectual Disability Research*, 53, 11–18.

Group Research

► Qualitative Versus Quantitative Approaches

Group Therapy

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Definition

A treatment modality that makes use of group, rather than individual, format to provide counseling/support/psychotherapy.

Historical Background

Group treatments for children have their origins in the attempts to provide rehabilitation services for

children, for example, those in the juvenile justice system or in some form of congregate care. As with work with adults, approaches to group treatment have become more specialized over time, for example, in autism focusing primarily on social skills or to help children with some common situation or problems. As with other approaches to psychotherapies, there has been an increased emphasis on use of common methods and specific methods and curricula.

Current Knowledge

Various approaches to group treatment have been developed (see Moss et al. 2007). Groups can be time limited or ongoing, and they may be focused on a special issue or problem.

The role of the leader(s) may also vary so that, for example, highly focused or short-term groups may need a much more active leadership process and be more didactic.

Groups can be guided by various theoretical perspectives, and such a perspective may have implications for group composition. Groups for children and adolescents must be cognizant of the ages and developmental levels of the members. Particularly for social skills-based groups, the inclusion of typically developing peers may provide important role models. The leaders provide overall structure and ongoing monitoring and can plan and implement a range of activities. For example, for a group of more cognitively able students with autism, explicit discussion of common tasks for the age group may include an experiential component to provide practice with opportunities for feedback. For the group leaders, awareness of the group's needs and levels of functioning can guide interventions and group process as well as issues of selection of new members. Group approaches are used in a range of settings (including clinics and schools). For children, it is common to have a standard structured format with some period for group discussion, an activity and snack, and then a brief review/wrap-up. Various techniques can be used to cope with behavioral difficulties including changes in group activities or structure, verbal interpretation, limit setting,

and so forth. Sometimes an individual will not be able to tolerate the demands of being in a group, and in such cases, removal of a group member should provide an opportunity for explanation as well as discussion. Group leaders can make use of a range of intervention strategies.

Time-limited groups can be readily used with less able individuals and provide opportunities to practice social skills in a supportive context. For more cognitively able individuals, extended groups can have a broader focus on a range of issues. If typical peers are included, some degree of (often relatively minimal) preparation is needed. Support groups for parents can also be useful.

As with other psychotherapies, considerations for great treatment include the nature of the difficulties and goals, the degree to which a group approach can be helpful, and so forth. Children who are highly disruptive may be difficult to incorporate in a group treatment approach. A small body of work on the efficacy of group treatment now exists with overall results suggesting some moderate level of benefit on average.

Future Directions

There has been an increased emphasis on implementation of specific behavioral and cognitive strategies in group treatment. New approaches focus on such CBT-based approaches. Although frequently used to teach social skills, the body of actual research on group treatments in autism is rather limited.

See Also

► [Psychotherapy](#)

References and Reading

Cappadocia, M., & Weiss, J. A. (2011). Review of social skills training groups for youth with Asperger syndrome and high functioning autism. *Research in Autism Spectrum Disorders*, 5(1), 70–78.

Dugan, E., Kamps, D., et al. (1995). Effects of cooperative learning groups during social studies for students with autism and fourth-grade peers. *Journal of Applied Behavior Analysis*, 28(2), 175–188.

Kohler, F. W., Strain, P. S., et al. (1990). Promoting positive and supportive interactions between preschoolers: An analysis of group-oriented contingencies. *Journal of Early Intervention*, 14(4), 327–341.

Moss, N. E., Racusin, G. R., & Moss-Racusin, C. (2007). Group therapy. In A. Martin & F. Volkmar (Eds.), *Lewis's child and adolescent psychiatry: A comprehensive textbook* (4th ed., pp. 842–854). Philadelphia: Lippincott Williams & Wilkins.

Williams, T. I. (1989). A social skills group for autistic children. *Journal of Autism and Developmental Disorders*, 19(1), 143–155. Philadelphia: Wolters Kluwer.

Group Work and Class Discussions

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Synonyms

[Cooperative groups](#)

Definition

Group work and class discussions are teaching strategies that can be found in a general education classroom. In order for students with ASD to be successful in these two learning environments, some accommodations may be needed. In group work, students are typically assigned a task to be completed by the group. Students may be assigned a specific role (e.g., notetaker) or the group may be directed to have a discussion on a specific topic or problem with the expectation that all students will equally contribute to the discussion. Students with ASD may find group work confusing unless the roles of the group members are clearly defined and written instructions for the task to be accomplished are provided. Often, step-by-step instructions for the task (task analysis) are

most helpful to the student with ASD because in addition to identifying specific steps that lead to task completion, the student sees the total process required to complete the task. In addition, the social behavior expected during the group work may need to be clearly defined and practiced through role playing prior to the inclusion of the student in a small work group. Finally, the student with ASD may need to practice discourse skills required for successful participation in a group discussion such as accepting suggestions, changing topics in a discussion, compromising, considering others opinions, negotiating, clarifying, and questioning.

Class discussions pose additional challenges for some individuals with ASD. It will be important for the teacher to make prior connections to the topic under discussion with all students, but this will be especially helpful for the student with ASD. In addition, the general topic and related vocabulary may need to be reviewed, pretaught, or “primed” with the student with ASD if they are to keep up and comprehend the discussion as it unfolds in the general classroom setting. During class discussions, the teacher can provide additional accommodations for the student with ASD by creating a visual web of information that highlights specific points and gives the student the entire picture of the topic being discussed.

See Also

- [Academic Supports](#)
- [Activity-Based Instruction](#)
- [Classroom Management](#)
- [Self-management Interventions](#)
- [Visual Supports](#)

References and Reading

- Mith, T. E. C., Polloway, E. A., Patton, J. R., & Dowdy, C. A. (2008). *Teaching students with special needs in inclusive settings* (5th ed.). Upper Saddle River: Pearson.
- Volkmar, F. R., Paul, R., Klin, A., & Cohen, D. (Eds.). (2005). *Handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.

Group-Based Early Start Denver Model (G-ESDM)

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Definition

The Group-Based Early Start Denver Model (G-ESDM; Vivanti et al. 2017) is an early intervention approach based on the adaptation of the Early Start Denver Model (ESDM; Rogers and Dawson 2010) for delivery within group settings. The G-ESDM emphasizes the importance of providing intensive teaching, drawing from evidence-based educational strategies, individualizing the program, and addressing multiple developmental domains within group-based cooperative learning experiences, with one adult delivering instruction to small groups of 2–3 children with ASD. Principles and strategies incorporate knowledge from developmental science, behavior analysis, and social-affective neuroscience to support learning and development in young children with autism. The group delivery format capitalizes on the feasibility, sustainability, and cost-effectiveness of providing treatment in small groups, the widespread availability of preschool programs in the community, the culturally universal tradition of educating young children in group settings, and the social and learning opportunities provided by peers.

Historical Background

The origins of G-ESDM date back to the early 1980s, when Sally Rogers and colleagues at the University of Colorado Health Sciences Center developed the Denver Model (Rogers et al. 1987), an autism intervention model informed by developmental models of autism and typical development (e.g., Rogers and Pennington 1991; Stern 1985). Subsequently, Sally Rogers and Geraldine Dawson created the “Early Start

Denver Model” (ESDM; Rogers and Dawson 2010), which involves the expansion and adaptation of the original Denver Model curriculum to address the developmental needs of toddlers, as well as the inclusion of a curriculum checklist (ESDM Curriculum Checklist; Rogers and Dawson 2010) to design individualized intervention targets. In 2010, Vivanti and colleagues at La Trobe University in Melbourne, Australia, developed the Group-Based Early Start Denver Model (G-ESDM), an approach for delivering ESDM within group-based settings, such as childcare and preschool programs. The G-ESDM was developed and implemented in the context of a partnership between the La Trobe University Olga Tennison Autism Research Center, the MIND Institute at University of California Davis, and the Victorian Autism Specific Early Learning and Care Centre, an early intervention program located at La Trobe University’s Community Children’s Centre.

Rationale or Underlying Theory

The G-ESDM, like the original ESDM, is based on cascade model of ASD (Pennington et al. 2006). According to this model, early emerging autism symptoms create a barrier to the development of the processes that facilitate bodily and emotional engagement during early interactions, such as imitation, reciprocal vocalization, and sharing of affect. Diminished participation in bodily routines that are social and playful in nature, in turn, disrupts the construction of neural specialization and behavioral knowledge in the social communication domain. As recently proposed by Dawson et al. (2005; Dawson 2008), such early disruptions might be linked to a biologically-based deficiency in experiencing social engagement as intrinsically rewarding. A corollary of this model is that targeting early social disruptions during early childhood might counteract the cascading effects of social impairments for individuals with ASD, conferring beneficial effects across different developmental domains. This is particularly important for younger children, as neural plasticity during early

developmental stages might allow for a deeper impact of social learning experiences on the developing brain.

Additionally, procedures of ESDM and G-ESDM are informed by the naturalistic application of applied behavior analysis (ABA), as articulated by Schreibman, Koegel et al. 1989 in their pioneering work on Pivotal Response Training (PRT), and more recently in the framework of Naturalistic Developmental Behavioral Intervention models of ASD (Schreibman et al. 2015).

Finally, the Group-ESDM draws heavily on the literature focused on the beneficial effects of early interaction with peers and preschool experiences for social-cognitive development. This literature indicates that the opportunity to practice complex social and cooperative behaviors during play routines with peers supports the development of social-cognitive and social emotional skills such as empathy and mentalizing (McAlister and Peterson 2007) and that involvement in high-quality child care environments that facilitate engagement in joint activities with peers has positive effects on social and communication development (National Institute of Child Health and Human Development Early Child Care Research Network 2000, 2003).

Goals and Objectives

The chief objective in the G-ESDM is to provide a sustainable evidence-based early intervention approach for young children with ASD that can be delivered in regular childcare and preschool settings. In the G-ESDM, children with ASD are provided with high-quality social learning experiences within group settings, so that the child can learn from others in their everyday experiences in their preschool program and in any other settings, and build a behavioral repertoire that support further learning and foster social-cognitive development.

While this overarching goal is relevant for all children with autism, specific treatment objectives are based on the distinctive profile of strength and weakness of each child, so that learning experiences can be individually tailored to

maximize learning progress. Individualized treatment objectives are developed from a careful assessment of the child's behavioral repertoire based on the ESDM Curriculum Checklist (Rogers and Dawson 2010), which evaluate each child's skills across multiple developmental domains including receptive communication, expressive communication, social skills, play skills, cognitive skills, fine motor skills, and adaptive skills.

Treatment Participants

The G-ESDM is designed for toddlers and preschoolers with ASD. Treatment goals are based on the ESDM curriculum, which addresses developmental skills in the 12–48 months range. The G-ESDM has been tested on children 12–60 months, with results showing better outcomes in children receiving the ESDM before 48 months of age. It has not been tested with children who have additional diagnoses like Down syndrome or fragile X.

Treatment Procedures

In the G-ESDM, individualized measurable learning objectives covering multiple developmental domains are generated every 12 weeks based on the curriculum checklist assessment. Objectives are designed to be addressed within the constraints and opportunities of group settings. Child progress is systematically recorded and mastery of all objectives is assessed every 12 weeks; new learning objectives are then generated based on the assessment results.

Individual goals are targeted within group routines. These are organized around clear, predictable, and shared goals, and designed to provide naturalistic learning opportunities, i.e., age-appropriate learning experiences involving routines and materials that children typically encounter in their everyday environments. Adults set up fun routines and play activities that naturally bring children together in the same physical space including art table activities, book activities,

“sensory” games such as games with water, sand, and shaving cream, group music and movement games such as Ring-around-a Rosie and parachute games as well as table games in which the children need to share and pass materials, or help each other. Children have duplicate objects and are face to face, so they can imitate one another, and the materials and their placement actively facilitate peer interaction. In this context, the adult organizes group-based joint activity routines, in which the children and adults in the small group are coconstructing activities that provide opportunities to do things together and learn from such experiences (Ratner and Bruner 1978). A joint activity routine is articulated in four stages, including (a) a set-up phase, in which the child gets to choose the activity; (b) a theme in which the child and the play-partners participate equally in the activity chosen by the child, creating a predictable and enjoyable routine; (c) a variation or elaboration that expands the theme; and (d) a closing phase, marking smooth ending for the current activity followed by a transition to the opening phase of the next activity. For example, a child might start to play with a piece of material (set-up phase), then the adult joins in, and the adult and child begin playing with material together, while other children are also encouraged to join in (the theme phase). As one child in the group starts engaging in a different action with the same material, the adult points that out and prompts other children to do the same (elaboration phase). When the activity starts to feel repetitive and/or children start to lose interest, the adult encourages children to close the activity and transition to the next activity (closing phase). The goal of joint activity routines is to address both the social difficulties (through the joint engagement component) and the flexibility difficulties (through the systematic introduction of variations on the theme) that characterize autism, while also providing opportunities to target multiple objectives across developmental domains.

The repeated engagement in meaningful and rewarding joint activity routines in close proximity to the peers under the guidance of the adult provide the framework for targeting and practicing expressive and receptive

communication, turn taking, imitation, sharing of affect, joint attention, functional and symbolic play, and motor skills, using evidence-based instructional techniques based on naturalistic developmental behavioral intervention, including the use of “Antecedent–Behavior–Consequence (ABC)” sequences, shaping, fading, prompting, chaining and error correction procedures, peer-mediated teaching, active management of affect, arousal, and motivation, and the use of warm, playful shared interactions as a context for learning (see Vivanti et al. (2017) for details).

A series of “decision trees” are used in the G-ESDM to readjust the program when data show that progress is slower than expected in one or more developmental areas. Modifications might include increasing the amount of teaching episodes delivered to the child during group activities, organizing 1:1 focused teaching sessions in a distraction-free environment, increasing reinforcers strength, and introducing augmentative communication tools.

The G-ESDM group activity fidelity tool (Vivanti et al. 2017) is used to determine whether the program is being delivered according to the G-ESDM implementation standards.

Efficacy Information

Preliminary evidence for effectiveness of the G-ESDM is documented in a controlled study involving 57 children with ASD ages 18–60 months, of whom 27 received 12 months of 15+ h per week of G-ESDM with staff ratios of one adult to three children (Vivanti et al. 2014). This group was compared to a carefully matched group of 30 children receiving 15+ h of early intervention for 12 months delivered in high-quality preschools that used a combination of empirically based intervention practices. Children in the G-ESDM group showed a significant advantage in overall developmental rate and receptive language scores. Additional research documented that children with younger chronological age and more advanced skills in object play, joint

attention, and imitation experienced the largest gains (Vivanti et al. 2013, 2016).

Outcome Measurement

Within the G-ESDM, there is a strong emphasis on daily tracking of a child’s progress against each learning objective. Consistent with the ESDM procedures detailed in Rogers and Dawson (2010), child’s response to each learning opportunity is recorded according to a time interval recording system, with adults taking data at between 10 and 15 min intervals depending on the frequency and intensity of teaching opportunities. These data are then summarized on each child’s “data summary sheet” on a daily basis. Measurable progress is expected on every objective within a 1–2-week period and mastery of all objectives is assessed every 12 weeks. Based on the assessment results, new objectives are written and broken down into small teachable steps, and new data sheets are developed. For research purposes, annual progress has been measured through standardized tests measuring cognitive functioning (Mullen Scales of Early Learning; Mullen 1995), adaptive behavior (Vineland Scales of Adaptive Behavior, Sparrow et al. 2005), and severity of autism symptoms (Autism Diagnostic Observation Schedule; Lord et al. 2012).

Qualifications of Treatment Providers

Professionals certified as ESDM therapists might deliver the G-ESDM following the manualized guidelines in Vivanti et al. 2017. Certification in ESDM involves completion of multiple levels of training according to the steps detailed here https://www.ucdmc.ucdavis.edu/mindinstitute/research/esdm/pdf/certification_steps.pdf.

Professionals from a wide number of disciplines are eligible to be trained as ESDM therapists, including occupational therapy, physical therapy, speech and language pathology, early

childhood education, early childhood special education, clinical child psychology, and school psychology.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Early Intervention](#)
- [Early Start Denver Model](#)

References and Reading

- Dawson, G. (2008). Early behavioral intervention, brain plasticity, and the prevention of autism spectrum disorder. *Development and Psychopathology*, 20, 775–803.
- Dawson, G., Webb, S., & McPartland, J. (2005). Understanding the nature of face processing impairment in autism: Insights from behavioral and electrophysiological studies. *Developmental Neuropsychology*, 27(3), 403–424.
- Koegel, R. L., Schreibman, L., Good, A. B., Cerniglia, L., Murphy, C., & Koegel, L. K. (1989). *How to teach pivotal behaviors to autistic children*. Santa Barbara: University of California.
- Lord, C., Rutter, M., DiLavore, P., Risi, S., Gotham, K., & Bishop, S. (2012). *Autism diagnostic observation schedule – 2nd edition (ADOS-2)*. Los Angeles: Western Psychological Corporation.
- McAlister, A., & Peterson, C. (2007). A longitudinal study of child siblings and theory of mind development. *Cognitive Development*, 22(2), 258–270.
- Mullen, E. M. (1995). *Mullen scales of early learning*. Circle Pines, MN: American Guidance Service.
- NICHD Early Child Care Research Network. (2000). The relation of child care to cognitive and language development. *Child Development*, 71(4), 960–980.
- NICHD Early Child Care Research Network. (2003). Does amount of time spent in child care predict socio-emotional adjustment during the transition to kindergarten? *Child Development*, 74(4), 976–1005.
- Pennington, B., Williams, J., & Rogers, S. (2006). Conclusions. In S. Rogers & S. Williams (Eds.), *Imitation and the social mind. Autism and typical development* (pp. 431–450). New York: Guilford Press.
- Ratner, N., & Bruner, J. (1978). Games, social exchange and the acquisition of language. *Journal of Child Language*, 5(03), 391–401.
- Rogers, S. J., & Dawson, G. (2010). *Early start Denver model for young children with autism: Promoting language, learning, and engagement: The curriculum checklist*. NY: Guilford Press.
- Rogers, S. J., & Pennington, B. F. (1991). A theoretical approach to the deficits in infantile autism. *Development and Psychopathology*, 3(2), 137–162.
- Rogers, S. J., Lewis, H. C., & Reis, K. (1987). An effective procedure for training early special education teams to implement a model program. *Journal of the Division of Early Childhood*, 11, 180–188.
- Schreibman, L., Dawson, G., Stahmer, A. C., Landa, R., Rogers, S. J., McGee, G. G., et al. (2015). Naturalistic developmental behavioral interventions: Empirically validated treatments for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(8), 2411–2428.
- Sparrow, S., Balla, D., & Cicchetti, D. (2005). *Vineland adaptive behavior scales* (2nd ed.). Circle Pines: American Guidance Service.
- Stern, D. N. (1985). *The interpersonal world of the infant*. New York: Basic Books.
- Vivanti, G., Dissanayake, C., Zierhut, C., Rogers, S. J., & Victorian ASELCC Team. (2013). Brief report: Predictors of outcomes in the early start Denver model delivered in a group setting. *Journal of Autism and Developmental Disorders*, 43(7), 1717–1724.
- Vivanti, G., Paynter, J., Duncan, E., Fothergill, H., Dissanayake, C., Rogers, S. J., & Victorian ASELCC Team. (2014). Effectiveness and feasibility of the early start Denver model implemented in a group-based community childcare setting. *Journal of Autism and Developmental Disorders*, 44(12), 3140–3153.
- Vivanti, G., Dissanayake, C., & Victorian ASELCC Team. (2016). Outcome for children receiving the early start Denver model before and after 48 months. *Journal of Autism and Developmental Disorders*, 46(7), 2441–2449.
- Vivanti, G., Duncan, E., Dawson, G., & Rogers, S. (2017). *Implementing the group-based early start Denver model for preschoolers with autism*. New York: Springer International Publishing.

Group-Based Organized Sport

- [Efficacy of Group-Based Organized Physical Activity Participation for Children with Autism Spectrum Disorder](#)

Growth Mixture Modeling

- [Latent Variable Modeling](#)

GSH-Px1 and Glutathione Hydrogen-Peroxide Oxido-reductase

► [Erythrocyte Glutathione Peroxidase](#)

Guanfacine

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Synonyms

[Intuniv](#); [Tenex](#)

Definition

Guanfacine is an alpha-2 agonist medication that has properties in common with clonidine. Early studies in adults with hypertension showed that it was less likely to cause sedation. In addition, it appeared to be less potent as antihypertensive medication. Studies done in animals showed that guanfacine also had other properties that are different from clonidine. These studies showed that guanfacine improved performance on delayed memory test in primates. In the early 1990s, studies of guanfacine in children with various conditions targeting hyperactivity and impulsive behavior began to emerge. These trials used the immediate-release product. Most trials were small and few were placebo-controlled. The immediate-release form of guanfacine has been examined in two studies in children with autism spectrum disorders. These studies suggest that the medication is helpful for reducing hyperactivity,

impulsiveness, and distractibility in these children. However, these studies were small and not definitive.

More recently, a long-acting formulation of guanfacine has been studied in two large-scale trials and also has been studied in a long-term extension trial. This new product is approved for the treatment of children with ADHD. The long-acting form of guanfacine is now approved for treatment of ADHD. Adverse effects of guanfacine are similar to clonidine. However, it does appear to be less likely to cause sedation that is often observed with clonidine. Midsleep awakening and irritability have been observed in studies of children with guanfacine. As with clonidine, guanfacine does not usually cause problems with hypotension. However, pulse and blood pressure are routinely monitored when children are treated with these compounds.

See Also

► [ADHD](#)

References and Reading

- Arnsten, A. F., Scahill, L., & Findling, R. L. (2007). Alpha2-Adrenergic receptor agonists for the treatment of attention-deficit/hyperactivity disorder: Emerging concepts from new data. *Journal of Child and Adolescent Psychopharmacology*, 17(4), 393–406.
- Biederman, J., Melmed, R., Patel, A., McBurnett, K., Konow, J., Lyne, A., et al. (2008). A randomized, double-blind, placebo-controlled study of guanfacine extended release in children and adolescents with attention-deficit/hyperactivity disorder. *Pediatrics*, 121(1), e73–e84. Retrieved from EBSCOhost.
- Sallee, F., McGough, J., Wigal, T., Donahue, J., Lyne, A., & Biederman, J. (2009). Guanfacine extended release in children and adolescents with attention-deficit/hyperactivity disorder: A placebo-controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(2), 155–165. Treatments for Children with Autism (pp. 231–243). Springer (2011). <https://doi.org/10.1097/CHI.0b013e318191769e>.
- Scahill, L., & Boorin, S. (2011). Psychopharmacology in children with PDD: Review of current evidence. In B. Reichow, P. Doering, D. Cicchetti, & F. Volkmar (Eds.), *Evidence-based practices*. New York: Springer.

Guardianship

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Synonyms

Conservatorship

Definition

Guardianship

A guardianship is the process whereby one person assumes the legal responsibility for protecting the personal and property interests of another person who is unable to adequately do so due to physical incapacity, mental incapacity, or immaturity of age (infancy). Guardianship effectively replaces the judgment of the legally incompetent or incapacitated person (the ward) with that of the capable person (the guardian). A guardian may be an actual person, professional or nonprofessional, or a “legal person” such as a state agency, a private nonprofit organization, or a private for-profit organization. Regardless of their specific identities, all guardians must scrupulously act in good faith to protect their wards’ interests. If they fail to do so, the courts can remove and replace them. State law controls guardianship regulations and procedures, with each state determining the conditions, provisions, and specific terminology of guardianships within its jurisdiction. Guardianship laws generally prefer that courts appoint qualified and willing family members as guardians but also allow them to appoint nonrelatives when doing so would better serve the needs of the wards.

Due to the loss of personal freedom that results from the appointment of a guardian, guardianship proceedings must follow a careful and orderly legal process that ensures that the ward’s legal rights will be fully protected. Guardianships are not always permanent: adult guardianships can be terminated if the adult wards can demonstrate that

they have regained or acquired the necessary competence to care for their own needs, and guardianships of children can be terminated once the children reach the age of legal maturity.

Some types of relevant guardianships include:

Guardianship of the person. The guardian is responsible only for the ward’s personal welfare.

Guardianship of the estate (or conservatorship). The guardian is responsible only for the ward’s property; such guardians may be called “conservators.” While complete mental incompetence of the property owner is not required, his or her incapacity to adequately manage his or her property must be demonstrated.

Plenary guardianship. The guardian is responsible for all aspects of the ward’s personal welfare and property.

Limited guardianship. The guardian assumes authority only for those specific areas in which the ward is unable to make his or her own competent decisions. Limited guardianships are rarely created.

Standby guardianship. A guardian is chosen in advance by a child’s parents to assume future responsibility for the child’s welfare only if the parents become unable to do so.

See Also

► Conservatorship

References and Reading

- Frolik, L. A., & Barnes, A. M. (2007). *Elder law: Cases and materials* (4th ed.). Newark: Lexis-Nexis, Matthew Bender.
- Garner, B. A. (Ed. in Chief). (2009). *Black's law dictionary* (9th Ed.). St. Paul: West-Thomson Reuters.
- National Guardianship Association, Inc. *Guardianship and developmentally disabled individuals*. Retrieved July 5, 2012 from <http://www.guardianship.org/pdf/developmentallyDisabled.pdf>
- National Guardianship Association, Inc. *Guardianship/conservatorship-an overview*. Retrieved July 5, 2012 from <http://www.guardianship.org/pdf/guardianshipConservatorship.pdf>.
- National Guardianship Association, Inc. *Questions and answers on guardianship issues*. Retrieved July 5, 2012 from <http://www.guardianship.org/pdf/question.pdf>

Guided Compliance

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Definition

Guided compliance procedures characteristically involve the systematic delivery of progressively more intrusive prompts. The prompts are delivered contingent upon an individual's non-compliance. The type of prompts used may include verbal prompting, model prompting, and/or physical guidance. These prompts are methodically increased following noncompliance with less-intrusive prompts, eventually culminating with full physical guidance when necessary.

Interventions using guided compliance have been successfully implemented to decrease noncompliance.

References and Reading

- Cote, C., Thompson, R. H., & McKerchar, P. M. (2005). The effects of antecedent interventions and extinction on toddlers' compliance during transitions. *Journal of Applied Behavior Analysis*, 38, 235–238.
- Horner, R. D., & Keilitz, I. (1975). Training mentally retarded adolescents to brush their teeth. *Journal of Applied Behavior Analysis*, 25, 491–498.
- Smith, M. R., & Lerman, D. C. (1999). A preliminary comparison of guided compliance and high-probability instructional sequences as treatment for noncompliance in children with developmental disabilities. *Research in Developmental Disabilities*, 20, 183–195.
- Wilder, D., & Atwell, J. (2006). Evaluation of a guided compliance procedure to reduce noncompliance among preschool children. *Behavioral Interventions*, 21, 265–272.

H

H₁ Receptor Antagonist

► [Antihistamines: Definition](#)

H₁ Receptor Inverse Agonist

► [Antihistamines: Definition](#)

Habit Reversal

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Definition

Habit reversal training (HRT) is a multi-component treatment package primarily used to treat tic disorders and body-focused repetitive behavior disorders such as skin picking, nail-biting, and hairpulling (trichotillomania; TTM). The first primary component of HRT is awareness training, in which the therapist works with the patient to identify instances of the target behavior, insuring the patient has, or develops, the ability to detect when the behavior occurs in real time.

After the patient is adequately aware of the behavior, competing response (CR) training

begins. In CR training, the patient and therapist identify a socially inconspicuous behavior that is physically incompatible with the target behavior, easy to perform in a variety of settings, and acceptable to the patient. The patient is taught to do CR for a prescribed period of time whenever the target behavior begins or is about to begin. The client practices using CR in session. The therapist provides praise for appropriate use of CR and prompts the patient when he or she does the targeted behavior but does not do CR. After the patient is trained to use CR in session, he or she is instructed to use it whenever the target behavior occurs or whenever he or she feels the urge to engage in the target behavior.

The final component of HRT is social support. In social support, a parent, spouse, or other individual who has a close relationship with the patient is taught to the use of HRT skills in the patient's day-to-day life. Social support ensures that HRT skills (especially the use of CR) generalize to settings beyond the therapy session. The support person is taught to praise the patient for using CR exercises and to provide gentle reminders to use CR when the target behavior occurs but CR does not.

Historical Background

The first evidence of HRT's efficacy emerged in a 1973 study by Nathan Azrin and Gregory Nunn. Throughout the 1970s and 1980s, a series of

small-n and group design studies built an initial evidence base suggesting that HRT was an efficacious treatment for tics and body-focused repetitive behaviors. More recently, larger controlled trials have lent support for HRT as a viable treatment option (either in place of or as an adjunct to drug therapy) to treat TTM, chronic tic disorders (e.g., Tourette syndrome), and other repetitive behavior problems. Within the field of tic disorders, a series of randomized controlled trials showed that patients who received HRT experienced greater symptom reduction than those on a wait list or those who received active control therapy (i.e., supportive psychotherapy). The largest of these trials showed that eight 1-h sessions of comprehensive behavioral intervention for tics – a manualized treatment centered on HRT’s core components – produced greater tic reductions than education and supportive psychotherapy and yielded gains comparable to those found with standard pharmacological treatments for tics.

Rationale or Underlying Theory

Across all problems for which the treatment is used, HRT is believed to increase awareness of behaviors that occur rapidly and often out of conscious awareness. In addition, it is believed that HRT may disrupt the underlying reinforcement processes serving to maintain a particular condition. For example, in persons with tics, aversive sensory experiences known as “premonitory urges” are known to build up immediately prior to a tic and are relieved immediately after the tic. This reduction in the aversive premonitory urge is believed to reinforce the tic. HRT physically prevents the tic from occurring and providing subsequent relief, which then allows the patient to habituate to the urge rather than eliminating it through ticcing. Over time, the urge becomes weaker with respect to eliciting tics. Similar reinforcement patterns are disrupted in the use of HRT to treat TTM. A large reinforcer for pulling involves the tactile stimulation that comes from the act of pulling. When the patient does CR at the beginning of the pulling act, the

tactile reinforcement that is usually received does not occur, and ultimately the pulling is decreased.

Goals and Objectives

HRT is intended to address tic disorders (e.g., Tourette syndrome) along with a variety of body-focused repetitive behavior disorders such as nail-biting, thumb-sucking, TTM, stereotypies, and skin picking. Like behavioral approaches to the treatment of other disorders, HRT takes a pragmatic, present-focused approach whose primary aim is reduction of the target behavior. When used as a treatment for neuropsychiatric conditions such as chronic tic disorders, HRT does not propose to “cure” the affected individual but rather teach skills that can effectively aid symptom management. HRT can be used either alone or in combination with pharmacotherapy or other treatments. As the severity of chronic tic disorders and TTM can wax and wane over time, the frequency with which a patient uses HRT skills at a given point in time may also vary. Evidence suggests that HRT skills are retained long after acute treatment ends (see section “[Efficacy Information](#)”) and therefore serve as a long-term tool for self-management of repetitive behavior disorders.

Treatment Participants

HRT is effective for both children and adults. Most studies evaluating HRT have been done with patients who have IQ > 80; however, clinical observation suggests that patient’s motivation for change, receptive language abilities, and instruction following skills are critical to the success of HRT. Also, research has shown that general deficits in response inhibition predict poor response to HRT for adults with tic disorders. Patients with deficits in these areas, either due to young biological age or delays in language and/or cognitive development, may struggle with traditional HRT. Nevertheless, the core components of HRT could be implemented in a fashion that does not rely as much on patient self-management skills. For

example, awareness training could consist of reinforcing a specific, subtle response such as raising a finger immediately after the occurrence of the behavior (a type of tacting) to indicate awareness. After this skill is mastered, chaining procedures could then be used to teach the patient to perform the competing response after the finger-raising response, thus mimicking the typical CRT requirement that patients perform the competing response after the occurrence of the targeted behavior.

To date, two published studies have demonstrated the potential effectiveness of modified HRT procedures as part of effective treatments for repetitive behaviors in individuals with intellectual disabilities and/or autism. The modification included physical prompting by the therapist during AT and the use of differential reinforcement and auditory feedback to augment the “self-management” style of HRT intervention. Although more research on the viability and efficacy of such adapted forms of HRT should be conducted, these studies serve to demonstrate the potential utility of HRT in individuals with autism spectrum disorders and intellectual disabilities who may have deficits in language and instruction following skills.

Treatment Procedures

HRT is delivered on a flexible outpatient basis, which is typically acceptable to consumers. Research has shown benefits resulting from as little as one or two HRT sessions, but typically HRT has been implemented across 8–10 weekly sessions lasting approximately 1 h each. Treatment is usually delivered in an individualized, face-to-face format, though research has shown that the treatment can be effective in groups and when implemented via remote teleconferencing equipment.

Since HRT skills involve a “self-management” repertoire employed by the individual himself or herself (i.e., are ultimately to be performed in the absence of prompts from others), it is important that the treatment be acceptable to the patient and able to be practically implemented in the patient’s

day-to-day environment. To ensure this, HRT employs open dialogue between the therapist, patient, and parent or others close to the patient. The core role of the HRT therapist is to teach HRT skills involved in AT and CR training, as well as to educate the support person, devise an appropriate reward system, and to work in concert with other treating professionals and parents to ensure smooth implementation of the intervention.

Efficacy Information

A large evidence base exists to support the use of HRT for body-focused repetitive behavior disorders. A series of small-n and uncontrolled studies throughout the 1970s–1990s showed that HRT tends to provide rapid reductions in symptom severity that are fairly durable following the cessation of treatment. Research has also shown that relapses do sometimes occur, but can most often be ameliorated by the inclusion of one or more posttreatment “booster sessions” in which HRT skills are briefly reviewed and practiced.

Throughout the past 20 years, the bulk of research on the efficacy of HRT has shifted to the study of Tourette syndrome and other chronic tic disorders. Researchers have conducted a series of randomized controlled trials, confirming that HRT is superior to wait list and active control treatments. In the largest of these studies, children with chronic tic disorders and Tourette syndrome received either an HRT-based intervention or psychoeducation plus supportive psychotherapy (as a control condition). Fifty-three percent of those receiving HRT showed clinically meaningful improvement following the acute phase of treatment (compared to 17% of those in the supportive psychotherapy condition), and 87% of these patients maintained treatment gains at the 6-month follow-up. The magnitude of the symptom reduction observed in the HRT group was comparable to the reduction of symptoms reported in studies evaluating pharmacological treatments for tics.

Although children with autism experience a higher prevalence of chronic tic disorders than typically developing children (Baron-Cohen

et al. 1999), stereotypies are by far the most common repetitive behavior problem seen in children with autism and related disorders. HRT has been far less studied as a treatment for stereotypy compared to chronic tic disorders, but the single published study investigating HRT for stereotypy in typically developing children found that HRT was an effective treatment. This finding, combined with the finding that HRT can be modified and taught to individuals with language deficits, sets the stage for future research investigating the application of HRT for tics and other repetitive behavior problems in children with autism spectrum disorders.

Outcome Measurement

The selection of tools to assess outcomes will depend on the age of the patient as well as the target behavior. For some behaviors, such as nail-biting and skin picking, permanent product measures such as the length of fingernails or the size of picking-induced wounds may be used to evaluate the severity of the behavior. However, other behaviors treated with HRT do not yield an obvious product, and other methods of evaluation must be used. Calculating the frequency with which the behavior occurs during a specific observation period has high face validity, but has significant limitations which preclude its use as a stand-alone measure of behavior. In addition, tics and many other repetitive behaviors often occur in the absence of others and are heavily influenced by minute-to-minute contextual variables such as physical setting, fatigue, and stress, thus making direct observation more difficult.

To overcome these difficulties, clinical researchers have developed interview-based, parent-report, and self-report assessment tools. Examples of tic assessments include the Yale Global Tic Severity Scale (YGTSS; validated for ages 5 and above), the Parent Tic Questionnaire (PTQ; validated for ages 7–16), and the Premonitory Urge for Tics Scale (PUTS; valid for ages 10 and above). The Massachusetts General Hairpulling Scale (valid for adults), the Milwaukee Inventory for Styles of Trichotillomania (MIST-A, adult version valid for ages 18 and

above; MIST-C, child version valid for ages 10–17) can be used to assess TTM, while the Skin Picking Scale (SPS; valid for adults) and the Milwaukee Inventory for the Dimensions of Adult Skin Picking (MIDAS; valid for adults) can be used to assess skin picking. The Stereotypy Severity Scale can be used to assess stereotypies. Due to the strengths and weaknesses of each type of assessment, clinicians should use multimodal assessment both at intake and throughout treatment to monitor symptom severity and the patient's response to the intervention.

Qualifications of Treatment Providers

Like any form of therapy, HRT should be implemented only by adequately trained behavioral health professionals, who are knowledgeable in the presenting problem. Although no formal certification process currently exists, it is recommended that HRT training involve didactic instruction through graduate courses or through one of the many workshops on HRT and related techniques that are regularly offered throughout the United States and other countries. In addition, it is recommended that practitioners interested in learning HRT be supervised for the first few cases by someone well versed in the procedure. HRT-based treatment manuals have been devised for many disorders, and health professionals trained in other forms of behavior therapy may be able to effectively administer HRT with the manuals as therapeutic guides.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Tics](#)
- [Tourette Syndrome](#)
- [Trichotillomania](#)

References and Reading

- Azrin, N. H., & Nunn, R. G. (1973). Habit-reversal: A method of eliminating nervous habits and tics. *Behavior Research and Therapy*, 11, 619–628.
- Baron-Cohen, S., Scahill, V. L., Izaguirre, J., Hornsey, H., & Robertson, M. M. (1999). The prevalence of Gilles

- de la Tourette syndrome in children and adolescents with autism: A large scale study. *Psychological Medicine*, 29, 1151–1159.
- Conelea, C. A., & Woods, D. W. (2008). Examining the impact of distraction on tic suppression in children and adolescents with Tourette syndrome. *Behavior Research and Therapy*, 46, 1193–1200.
- Cook, C. R., & Blacher, J. (2007). Evidence-based psychosocial treatments for tic disorders. *Clinical Psychology: Science and Practice*, 14(3), 252–267.
- Deckersbach, T., Rauch, S., Buhlmann, U., & Wilhelm, S. (2006). Habit reversal versus supportive psychotherapy in Tourette's disorder: A randomized controlled trial and predictors of treatment response. *Behavior Research and Therapy*, 44, 1079–1090.
- Himle, M. B., Olufs, E., Himle, J., Tucker, B. T. P., & Woods, D. W. (2010). Behavior therapy for tics via videoconference delivery: An initial pilot test in children. *Cognitive and Behavioral Practice*, 17, 329–337.
- Long, E. S., Miltenberger, R. G., Ellingson, S. A., & Ott, S. M. (1999). Augmenting simplified habit reversal in the treatment of oral-digital habits exhibited by individuals with mental retardation. *Journal of Applied Behavior Analysis*, 32, 35–365.
- Marcus, A., Sinnott, B., Bradley, S., & Grey, I. (2010). Treatment of idiopathic toe-walking in children with autism using GaitSpot auditory speakers and simplified habit reversal. *Research in Autism Spectrum Disorders*, 4, 260–267.
- Piacentini, J., Woods, D. W., Scahill, L., Wilhelm, S., Peterson, A. L., Chang, S., et al. (2010). Behavior therapy for children with Tourette disorder: A randomized controlled trial. *Journal of the American Medical Association*, 303, 1929–1937.

Hair Analysis

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Synonyms

[Commercial hair analysis](#); [Hair mineral analysis](#);
[Hair trace metal analysis](#)

Definition

Hair analysis is an approved method for determining trace element concentrations and exposure to heavy metals (World Health Organization 1990).

Scalp hair is removed and sent to a laboratory for analysis. The most common elements analyzed by laboratories in the United States, as determined by Seidel et al. (2001), include aluminum (Al), arsenic (As), calcium (Ca), cadmium (Cd), cobalt (Co), chromium (Cr), copper (Cu), iron (Fe), mercury (Hg), potassium (K), magnesium (Mg), manganese (Mn), molybdenum (Mo), sodium (Na), nickel (Ni), phosphorus (P), lead (Pb), selenium (Se), and zinc (Zn).

Many children with autism are selective eaters, leading to concerns about possible nutritional deficiencies. Additionally, trace metals are related to neurotransmitter synthesis and brain electrochemistry and therefore have the potential to affect neurological function. These concerns inspired several studies performed during the 1980s in which the hair of children with autism was analyzed and compared to controls for trace elements. The results of these studies did not show consistent differences between individuals with and without autism (Shearer et al. 1982; Wecker et al. 1985; Gentile et al. 1983), and hair analysis became less prevalent in the autism literature.

Hair analysis became relevant to autism again because of a proposed relationship between mercury exposure and the development of autism (Bernard et al. 2001). Hair analysis was adopted as one technique to test for unusual mercury levels, in addition to other elements. Holmes et al. (2003) determined that mercury levels were reduced in the results of hair analysis of children with autism compared to control children. The authors explained this observation by claiming that children with autism have a reduced ability to clear mercury from their bodies. In contrast, Fido and Al-Saad (2005) discovered elevated levels of mercury, lead, and uranium in the hair of children with autism. Other studies (Ip et al. 2004; Williams et al. 2008) have found no differences in hair mercury levels between children with autism and control children.

Results from commercial hair analysis should be interpreted with extreme caution. Barrett (1985) reported the lack of inter- and intra-laboratory reliability of 13 commercial hair analysis laboratories. In 2001, Seidel et al. confirmed the lack of interlaboratory reliability even after considerable technological improvements.

Similarly, laboratories used different techniques (e.g., atomic emission, mass spectroscopy), and without any existing proficiency testing programs, each laboratory was responsible for its own verification method and criteria for accuracy. At the time of publication, Seidel et al. reported that there were no regulation standards among commercial hair analysis laboratories.

Similarly, Steindel and Howanitz (2001) advised clinicians that no standard method for commercial hair analysis exists. However, they give several suggestions for choosing a good laboratory. The best laboratories will give proof of certification or accreditation, an explanation of the specific washing and digestion procedures, and information about recovery rates, minimum detection limits, and internal quality control. The World Health Organization (1990) recommends the neutron activation procedure as the most accurate and sensitive procedure. Lind et al. (1988) found good interlaboratory agreement for this method. Another widely used and accepted procedure is the selective atomic absorption (“Magos”) method.

- Lind, B., Bigras, L., Cernichiari, E., Clarkson, T. W., Friberg, L., Hellman, M., & Ohlin, B. (1988). Quality control of analyses of mercury in hair. *Fresenius Journal of Analytical Chemistry*, 332, 620–622.
- Seidel, S., Kreutzer, R., Smith, D., McNeel, S., & Gilliss, D. (2001). Assessment of commercial laboratories performing hair mineral analysis. *Journal of the American Medical Association*, 285, 67–72.
- Shearer, T. R., Larson, K., Neuschwander, J., & Gedney, B. (1982). Minerals in the hair and nutrient intake of autistic children. *Journal of Autism and Developmental Disorders*, 12, 25–34.
- Steindel, S. J., & Howanitz, P. J. (2001). The uncertainty of hair analysis for trace metals. *The Journal of the American Medical Association*, 285, 83–85.
- Wecker, L., Miller, S. B., Cochran, S. R., Dugger, D. L., & Johnson, W. D. (1985). Trace element concentration in hair from autistic children. *Journal of Mental Deficiency Research*, 29, 15–22.
- Williams, P. G., Hersh, J. H., Allard, A., & Sears, L. L. (2008). A controlled study of mercury levels in hair samples of children with autism as compared to their typically developing siblings. *Research in Autism Spectrum Disorders*, 2, 170–175.
- World Health Organization. (1990). Methylmercury. In *International programme on chemical safety* (Environmental health criteria 101). Retrieved from <http://www.inchem.org/documents/ehc/ehc101.htm>

See Also

- [Thimerosal](#)

References and Reading

- Barrett, S. (1985). Commercial hair analysis: Science or scam? *Journal of the American Medical Association*, 254, 1041–1045.
- Bernard, S., Enayati, A., Redwood, L., Roger, H., & Binstock, T. (2001). Autism: A novel form of mercury poisoning. *Medical Hypotheses*, 56, 462–471.
- Fido, A., & Al-Saad, S. (2005). Toxic trace elements in the hair of children with autism. *Autism*, 9, 290–298.
- Gentile, P. S., Trentalange, M. J., Zamichek, W., & Coleman, M. (1983). Brief report: Trace elements in the hair of autistic and control children. *Journal of Autism and Developmental Disorders*, 13, 205–206.
- Holmes, A. S., Blaxill, M. F., & Haley, B. E. (2003). Reduced levels of mercury in first baby haircuts of autistic children. *International Journal of Toxicology*, 22, 277–285.
- Ip, P., Wong, V., Ho, M., Lee, J., & Wong, W. (2004). Mercury exposure in children with autistic spectrum disorder: Case-control study. *Journal of Child Neurology*, 19, 431–434.

Hair Mineral Analysis

- [Hair Analysis](#)

Hair Trace Metal Analysis

- [Hair Analysis](#)

Halcion

- [Triazolam and Autism](#)

Haldol™

- [Haloperidol](#)

Haloperidol

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Synonyms

Alti-Haloperidol; Apo-Haloperidol; Haldol™;
Novo-Peridol; Peridol; Pms-Haloperidol; Ratio-
Haloperidol

Definition

The brand name of haloperidol in the United States is Haldol. Canadian brand names include Alti-Haloperidol, Apo-Haloperidol, Novo-Peridol, Peridol, Pms-Haloperidol, and Ratio-Haloperidol. It comes in three forms: a pill, an intramuscular injection, and a longacting depot decanoate form and is manufactured by Ortho-McNeil-Janssen Pharmaceuticals, Inc.

Indications

The oral and immediate-release injection forms of haloperidol are FDA-approved for the treatment of schizophrenia as well as for tics and vocal utterances associated with Tourette's disorder (Janssen 2005). These forms are also used as second-line treatment to manage hyperactivity in the pediatric population as well as for severe behavior problems, particularly combativeness, explosiveness, and hyperexcitability (Stahl 2009). The long-acting depot intramuscular decanoate form of haloperidol is approved for the treatment of schizophrenia in situations where prolonged parenteral antipsychotic therapy

is needed. Haloperidol is not approved for use in the treatment of autism or any other autism spectrum disorder (ASD).

Mechanisms of Action

Mechanism of Action, According to Stahl (2008).

Haloperidol is a conventional antipsychotic, also known as a “first-generation” or “typical” antipsychotic. The precise mechanism of action is not clearly established, but it is believed to antagonize postsynaptic dopamine-2 (D₂) receptors in the brain. One of haloperidol's most notable roles is D₂ receptor antagonism in the mesolimbic pathway is diminishing the symptoms of psychosis. The introduction of this drug class resulted in relief to individuals living with schizophrenia and associated psychotic illnesses by reducing perceptual disturbances, such as hallucinations and delusions. The downside is that the mesolimbic pathway is believed to moderate the brain's reward and reinforcement system. Blockade here can also result in decreased pleasure, anhedonia, apathy, and reduced social interest, often referred to as “secondary negative symptoms.” This is further exacerbated by dopamine blockade in the brain cortex and mesocortical pathways. Blockade of the dorsolateral prefrontal cortex can lead to worsening cognition, whereas blockade of the ventromedial prefrontal cortex can lead to worsening affective symptoms, like depression.

The benefit of D₂ receptor antagonism in the nigrostriatal dopamine pathway is reduction of tics and other symptoms of Tourette's disorder. However, disproportionate dopamine blockade in this area can mimic the pathophysiology of Parkinson's disease. When the amount of dopamine available to the postsynaptic receptor is excessively diminished, Parkinsonian-like side effects can occur including stiffness, cogwheeling, and tremor. Since the nigrostriatal pathway is part of the brain's extrapyramidal nervous system, these side effects are referred to as extrapyramidal symptoms or EPS. Continued, long-term blockade of nigrostriatal D₂ receptors can result in a hyperkinetic movement disorder

known as tardive dyskinesia, involving involuntary movements of the face, jaw, tongue, extremities, or torso. This may present like chewing, facial grimacing, jerking of the arms or legs, or even rhythmic, fluid dancing of the body known as chorea. These side effects may be reversible if the drug is stopped early enough, but there may be a point in chronic therapy when it is too late. It is hypothesized that chronic antagonism of D₂ receptors may cause them to permanently become hypersensitive or increase in number, leading to irreversible tardive dyskinesia.

D₂ receptor antagonism in the tuberoinfundibular pathway causes elevated plasma prolactin levels. This can result in amenorrhea (irregular menstruation) in women and galactorrhea (breast secretions) in either men or women. It may also interfere with fertility, particularly in women, as well as bone mineralization in postmenopausal women, raising their risk of osteoporosis.

Haloperidol is also selective in its affinity for the alpha-1 adrenergic receptors, which can lead to orthostatic hypotension, dizziness, and drowsiness. Unlike other conventional antipsychotics, it has minimal affinity for cholinergic or histaminic receptors and therefore puts the patient at risk for increased EPS.

Specific Compounds and Properties

Haloperidol generic and immediate-release injection: 4-[4-(*p*-chlorophenyl)-4-hydroxypiperidine]-4'-fluorobutyrophenone (Janssen 2005).

Haloperidol decanoate for long-acting injection: 4-(chlorophenyl)-1-[4-(4-fluorophenyl)-4-oxobutyl]-4-piperidinyl decanoate (Janssen 2005).

Clinical Use (Including Side Effects)

Haloperidol comes in a generic pill form, an immediate-release intramuscular injectable form called Haldol Injection, and a long-acting injectable form called Haldol Decanoate. Haloperidol can be sedating, so both the oral or intramuscular

forms are often used to stabilize acutely agitated patients with moderately severe to very severe symptoms (Janssen 2005). Haloperidol is used in the short- and long-term treatment of psychotic illnesses, particularly in diminishing symptoms of schizophrenia, like hallucinations and delusions (Stahl 2009). It is also used as management for tics and involuntary vocal utterances associated with Tourette's disorder. In the pediatric population, haloperidol can be used to manage hyperactivity and severe behavioral disturbance, particularly combativeness, explosiveness, and hyperexcitability. Safety and efficacy have not yet been established in children and adolescents, so haloperidol should be considered second-line treatment for these conditions behind the atypical, second-generation antipsychotics that are approved for use in the pediatric population. Oral and intramuscular haloperidol is commonly given to patients once or multiple times daily, depending on their clinical presentation and medication needs. The long-acting depot decanoate form is approved for the treatment of schizophrenia where prolonged parenteral antipsychotic therapy is needed and given as a monthly injection.

Caution should be used when administering haloperidol to individuals with cardiovascular disorder due to a risk of hypotension and/or precipitation of anginal pain (Janssen 2005). Haloperidol also decreases the seizure threshold and should therefore be used with caution in patients being treated with anticonvulsants. There are no well-controlled studies with haloperidol in pregnant women, so the effect on the fetus is unknown. Haloperidol is a pregnancy category C drug, meaning there are some animal studies demonstrating adverse effects but no controlled studies in humans. Haloperidol therefore should only be used in pregnancy (or in women likely to become pregnant) if the benefit clearly outweighs the risk.

Clinical Uses in Autism Spectrum Disorders

Haloperidol is the most critically studied antipsychotic medication for managing or reducing symptoms associated with autism (Malone and Waheed 2009). The earliest double-blind,

placebo-controlled studies examined the efficacy of haloperidol in treating symptoms associated with autism such as repetitive behavior, stereotypies, hyperactivity, mood lability, learning, language acquisition, and sociability in autistic children between the ages of 2 and 7.5 years (Anderson et al. 1984, 1989; Campbell et al. 1978; Cohen et al. 1980). Overall, these studies found haloperidol to be efficacious in alleviating many of these symptoms with minimal side effects, although these were short-term studies that did not extend beyond 4–14 weeks. Other studies have compared the safety and efficacy of haloperidol with other drugs in treating autistic disorder and were overall successful (Faretra et al. 1970; Miral et al. 2008; Naruse et al. 1982; Remington et al. 2001). These latter studies looked at broader age ranges and included older children, adolescents, and some adults. There has been one long-term placebo-controlled study showing the efficacy of discontinuous haloperidol treatment over the course of 6 months in children with autism, but the results are somewhat limited by the study population as it only included children who responded favorably to haloperidol in the past (Perry et al. 1989). The results are intriguing nonetheless and have been included in this summary of double-blinded, placebo-controlled studies examining haloperidol in the treatment of autism.

Short-Term Research (4–14 Weeks)

In a study by Campbell et al. (1978), haloperidol was found to be superior to placebo in reducing stereotypies and social withdrawal in 40 children diagnosed with autism aged 2–7 years over the course of 12 weeks. Participants received dosages between 0.5 and 4.0 mg/day with a mean of 1.65 mg/day. Interestingly, children who received both medication and behavioral treatment improved the most in acquiring imitative speech, making it one of the first reports showing that combined treatments produced superior results (Malone and Waheed 2009). Excessive, dose-related sedation was the most common side effect. Another study by Cohen et al. (1980) demonstrated decreased stereotypies in autistic children ages 2–7 using haloperidol over the course of

10 weeks. In both of these studies, acute dystonia was observed in a few participants as an adverse event. Another study by Anderson et al. (1984) showed that haloperidol resulted in significantly reduced behavioral symptoms, general clinical improvement, and greater facilitation and retention of discrimination learning in 40 autistic children ages 2–7 years over 14 weeks. There were no adverse effects noted at therapeutic doses, which ranged from 0.5 to 3.0 mg/day (0.019–0.217 mg/kg/day). Anderson et al. (1989) described haloperidol as a “powerful drug in reducing, both statistically and clinically, maladaptive behaviors” including decreased hyperactivity, temper tantrums, withdrawal, and stereotypies. The sample of 45 children in this study were inpatients whose symptoms ranged from moderate to severe and whose ages were between 2 and 7.5 years. Medication dosages ranged from 0.25 to 4.0 mg/day. During the 4-week course of treatment, no adverse side effects were noted.

Haloperidol has been compared to two other conventional antipsychotics in the management of autism: fluphenazine in 1970 by Faretra et al. and pimozide in 1982 by Naruse et al. Each of these drugs demonstrated efficacy, but in comparison, haloperidol was found to be more effective than fluphenazine in reducing withdrawal, aggression, and stereotypies. Acute dystonic reaction, akathisia, and sedation were observed side effects. A 2001 study by Remington et al. compared haloperidol to clomipramine, a tricyclic antidepressant with potent serotonin reuptake inhibiting (SSRI) properties, in managing withdrawal, stereotypy, hyperactivity, and affective lability in autistic disorder. This study examined 36 children and adults between the ages of 10 and 36 years over 7 weeks. Both drugs demonstrated comparable improvement in symptoms when compared to baseline, but unfortunately, a significant number of participants were unable to complete the study due to side effects and lack of efficacy with clomipramine. The study favored haloperidol over clomipramine in the treatment of autistic disorder, particularly for hyperactivity and irritability but also in global symptom severity. Clomipramine was not better tolerated nor was it superior to

haloperidol in the management of stereotypy, as was originally hypothesized. In 2008, Miral et al. compared the safety and efficacy of haloperidol to risperidone, a second-generation antipsychotic that is believed to antagonize dopamine-2 receptors, serotonin 2A receptors, and other serotonin receptors in the treatment of autistic disorder. This study examined 30 children aged 8–18 years for 12 weeks, with dosages ranging from 0.01 to 0.08 mg/kg/day. Although both drugs were found to be safe and well tolerated in the treatment of autistic disorder, risperidone was more effective in treating impulsivity, behavioral symptoms, language skills, and impaired sociability. Risperidone led to a greater increase in prolactin, whereas haloperidol resulted in increased alanine amino transferase (ALT).

Long-Term Research (6 Months or More)

In a 1989 study by Perry et al., haloperidol demonstrated efficacy when dosed continuously and discontinuously (5 days taking haloperidol and 2 days taking placebo) in children who had previously responded favorably to haloperidol. The children ranged in age from 2 to 8 years and showed a reduction in many maladaptive symptoms, with the most response in those with irritability, labile and angry affect, and uncooperativeness. Although the two groups were not matched to placebo, a subset of these children who were switched to placebo after the study has discontinued required additional haloperidol treatment for the recurrence of severe symptoms, supporting the drug's efficacy.

Side Effects, per Ortho-McNeil-Janssen Pharmaceuticals

Cardiovascular side effects include hypotension, tachycardia, and hypertension. QT prolongation and ventricular arrhythmias have been reported.

Central nervous system (CNS) side effects include Parkinsonian-like symptoms like tremor, rigidity, or cogwheeling; akathisia (inner restlessness); dystonias; or tardive dyskinesia. Dystonias are prolonged contractions of muscles and can include neck muscle spasms, tightening of the throat, dyspnea, or tongue protrusion. These can occur at low doses but are known to occur more frequently and with greater severity at higher

doses. They are also more often seen in males and younger age groups. Administering anticholinergic medication can limit or reduce these effects. Tardive dyskinesia is the potentially irreversible syndrome of involuntary, dyskinetic movements (see Mechanism of Action). Haloperidol reduces the seizure threshold and therefore increases the risk of seizures. Other CNS side effects can include insomnia, anxiety, euphoria, agitation, drowsiness, depression, headache, confusion, sedation, dizziness, and vertigo.

Neuroleptic malignant syndrome (NMS) is a potentially fatal adverse effect of using any antipsychotic medication. It presents as hyperpyrexia, muscle rigidity, altered mental status, and autonomic instability. This includes dysrhythmias, irregular blood pressure, tachycardia, or diaphoresis. It can lead to elevated creatinine phosphokinase, myoglobinuria from muscle breakdown, and acute renal failure.

Autonomic reactions can include dry mouth, blurred vision, urinary retention, diaphoresis, and priapism. Dermatologic side effects include hair loss, photosensitivity, and maculopapular or acne-like skin reactions. Visual disturbances can include cataracts or retinopathy. Effects on the gastrointestinal system can include constipation, diarrhea, nausea, vomiting, anorexia, dyspepsia, or hypersalivation. Endocrine effects can include breast engorgement, gynecomastia, menstrual irregularities, impotence, increased libido, hyperglycemia, hypoglycemia, or hyponatremia. Hematologic reactions may include agranulocytosis, anemia, leukocytosis, leucopenia, or lymphomonocytosis. Hepatic side effects can include jaundice or impaired liver function. Respiratory manifestations may include bronchospasm, laryngospasm, or increased depth of respiration. Since the release of haloperidol, there has been one case of hyperammonemia in a 5-year-old child with citrullinemia, an inherited disorder of ammonia excretion. Haldol Decanoate may produce a local tissue reaction at the injection site.

See Also

- [Antipsychotics: Drugs](#)
- [Dystonia](#)

- ▶ Tardive Dyskinesia
- ▶ Tics
- ▶ Tourette Syndrome

References and Reading

- Anderson, L. T., Campbell, M., et al. (1984). Haloperidol in the treatment of infantile autism: Effects on learning and behavioral symptoms. *The American Journal of Psychiatry*, 141(10), 1195–1202.
- Anderson, L. T., Campbell, M., et al. (1989). The effects of haloperidol on discrimination learning and behavioral symptoms in autistic children. *Journal of Autism and Developmental Disorders*, 19(2), 227–239.
- Campbell, M., Anderson, L. T., et al. (1978). A comparison of haloperidol and behavior therapy and their interaction in autistic children. *Journal of the American Academy of Child Psychiatry*, 17(4), 640–655.
- Cohen, I. L., Campbell, M., et al. (1980). Behavioral effects of haloperidol in young autistic children. An objective analysis using a within-subjects reversal design. *Journal of the American Academy of Child Psychiatry*, 19(4), 665–677.
- Faretra, G., Dooher, L., et al. (1970). Comparison of haloperidol and fluphenazine in disturbed children. *The American Journal of Psychiatry*, 126(11), 1670–1673.
- Janssen-Cilag Pty Limited. (2005). *Haldol product information*. Retrieved from http://www.janssen.com.au/files/Products/Haldol_PL.pdf.
- Malone, R. P., & Waheed, A. (2009). The role of antipsychotics in the management of behavioural symptoms in children and adolescents with autism. *Drugs*, 69(5), 535–548.
- Miral, S., Gencer, O., et al. (2008). Risperidone versus haloperidol in children and adolescents with AD: A randomized, controlled, double-blind trial. *European Child & Adolescent Psychiatry*, 17(1), 1–8.
- Naruse, H., Nagahata, M., et al. (1982). A multi-center double-blind trial of pimozide (Orap), haloperidol and placebo in children with behavioral disorders, using crossover design. *Acta Paedopsychiatrica*, 48(4), 173–184.
- Perry, R., Campbell, M., et al. (1989). Long-term efficacy of haloperidol in autistic children: Continuous versus discontinuous drug administration. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28(1), 87–92.
- Remington, G., Sloman, L., et al. (2001). Clomipramine versus haloperidol in the treatment of autistic disorder: A double-blind, placebo-controlled, crossover study. *Journal of Clinical Psychopharmacology*, 21(4), 440–444.
- Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd ed., pp. 328–342). New York: Cambridge University Press.
- Stahl, S. M. (2009). *Stahl's essential psychopharmacology: The prescriber's guide* (3rd ed., pp. 237–242). New Delhi: Cambridge University Press.

Halstead-Reitan Neuropsychological Test Battery

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Synonyms

HRB

Description

The HRB represents a fixed battery approach to neuropsychological assessment. The HRB is composed of three separate batteries: the adult battery (Halstead Neuropsychological Test Battery for Adults; ages 15+), an older children's battery (Halstead Neuropsychological Test Battery for Children; ages 9–14), and a younger children's battery (Reitan-Indiana Neuropsychological Test Battery for Children; ages 5–8). The adult battery is composed of the Category Test, Tactual Performance Test, Seashore Rhythm Test, Speech Sounds Perception Test, Finger Oscillation Test, Trail-Making Test, Aphasia Screening Test, Sensory-Perceptual Disturbances Test, Lateral Dominance Test, and Grip Strength Test. In creating the older children's battery, Reitan simplified and/or shortened the Category Test, Trail-Making Test, Tactual Performance Test, and Speech Sounds Perception Test (Reitan and Wolfson 1992a). Several of these tests were simplified by Reitan when he created the older children's battery (Horton 2008). Additional modifications were made for the younger children's battery, including addition of the Progressive Figures Test (a visual modification of the Trail-Making Test), use of an electronic finger tapper, and simplification of the Category Test, Tactual Performance Test, and Sensory-Perceptual Examination (Reitan and Wolfson 1992b). An age-appropriate Wechsler intelligence

measure, as well as a personality measure such as the Minnesota Multiphasic Personality Inventory, is also typically administered (Horton 2008).

Historical Background

Development of the HRB began in the 1940s when Dr. Ward Halstead aimed to develop a series of tests sensitive to the presence of brain damage, in contrast to the single-test assessment approaches which were prevalent at the time. In order to develop his test battery, Halstead observed brain-damaged patients in their everyday lives to discern which aspects of their behavior were different from the behavior of unimpaired individuals. Given the heterogeneity of their deficits, it was clear to Halstead that a single test would likely not be able to adequately identify and evaluate the nature of their deficits (Reitan and Wolfson 2009). Halstead ultimately developed 10 tests he considered to be sensitive to the presence of brain damage (Halstead 1947).

In a fact-driven validation procedure over the course of 15 years, Dr. Ralph Reitan utilized Halstead's original 10 tests to examine patients and blindly interpreted the results without knowledge of their histories or neurological findings. After a report was written, it was compared with an independent summary of the medical and neurological findings in order to examine inaccuracies in the neuropsychological test results. Tests were accepted or rejected solely on the basis of their accurate prediction of the presence of brain damage for thousands of patients with various types of injuries. Although Halstead's tests typically resulted in a correct conclusion about whether a patient had brain damage, conclusions regarding the nature of the damage (e.g., lateralization, type of lesion) could not be done in a highly accurate manner. Reitan experimentally evaluated additional tests for their ability to provide information about the nature of the brain damage. Three of Halstead's original 10 tests were ultimately excluded as a result of this procedure (Reitan and Wolfson 2009).

Psychometric Data

Interpretation of results from the HRB typically occurs along several different levels of analysis: level of performance, pathognomonic signs, score patterns, and right-left differences. Analysis at the level of performance involves the comparison of patients to normative standards. However, many other factors besides brain injury can impact test performance (e.g., limited education, mood disorders), and the patients' degree of decline from pre-morbid function is not taken into account. Use of composite indices generated from test results, as well as comparisons to performance on Wechsler intelligence measures, can occur at this level of interpretation. Interpretation at the level of pathognomonic signs involves examining for evidence of aphasia, agnosia, and apraxia. Limitations at this level of analysis include the reliance upon the examiner's judgment for the presence of signs, and depending on the location of their brain injury, individuals do not necessarily show signs. Score patterns are examined to determine the relative integrity of the two cerebral hemispheres and to allow for intraindividual comparisons. Finally, analysis of left-right differences involves the comparison of right and left hands on various sensory and motor tasks, although the utility of this can be limited in cases involving significant peripheral injury of the arms/hands (Sweeney et al. 2007; Horton 2008).

Screening tests, requiring only 30–45 min to administer, have also been developed in order to more quickly identify those individuals who would benefit from administration of the complete HRB battery. Results from young children (Reitan and Wolfson 2008a) indicate the brief screening tests have 92% sensitivity and 85% specificity in detecting individuals with brain damage. Results from older children (Reitan and Wolfson 2008b) indicate 92% sensitivity and 88% specificity in detecting individuals with brain damage. Reitan and Wolfson argue that many of the misclassified children had borderline scores, and recommendations to take into account additional sources of information were made. For adults, the screening tests had a sensitivity of 96% and a specificity of 82%, with

older adults more likely to be misclassified as brain injured (Reitan and Wolfson 2008c).

Clinical Uses

The HRB does have several limitations: It is unwieldy and difficult to transport, takes a relatively long time to administer and score, and is not suitable for thorough examination of patients with sensory or motor handicaps. It contains no formal memory tests, and supplemental language measures may also be needed. Few ecological validity studies of the HRB have been done, and adequate interpretation requires a high level of expertise and years of experience (Horton 2008). In addition, with modern neuroimaging methods, neuropsychologists are rarely called upon to identify the presence of brain injury and its location, although they may be asked to judge the integrity of localized cognitive functions (Lezak et al. 2004). Finally, the need to use demographic normative corrections with test scores has been raised as an issue, although Reitan and Wolfson (1995) argue the effects of brain damage may reduce/eliminate age and education effects. Most recently, Heaton et al. (2004) revised the demographic age, education, and gender-corrected norms to include normative data for Caucasians and African Americans. Finally, several authors have argued the greatest contribution of the HRB may be with its impact upon the practice of neuropsychological assessment more generally and its push to assess many different aspects of behavior and cognitive function during neuropsychological evaluations (Lezak et al. 2004; Horton 2008).

References and Reading

- Halstead, W. C. (1947). *Brain and intelligence: A quantitative study of the frontal lobes*. Chicago: University of Chicago Press.
- Heaton, R. K., Miller, S. W., Taylor, M. J., & Grant, I. (2004). *Revised comprehensive norms for an expanded Halstead-Reitan Battery*. Odessa: Psychological Assessment Resources.

- Horton, A. M. (2008). The Halstead-Reitan neuropsychological test battery: Past, present, and future. In A. M. Horton & D. Wedding (Eds.), *The neuropsychology handbook* (3rd ed.). New York: Springer.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment*. New York: Oxford University Press.
- Reitan, R. M., & Wolfson, D. (1992a). *Neuropsychological evaluation of older children*. Tucson: Neuropsychology Press.
- Reitan, R. M., & Wolfson, D. (1992b). *Neuropsychological evaluation of younger children*. Tucson: Neuropsychology Press.
- Reitan, R. M., & Wolfson, D. (1995). Influence of age and education on neuropsychological test results. *The Clinical Neuropsychologist*, 9, 151–158.
- Reitan, R. M., & Wolfson, D. (2008a). The use of serial testing to identify young children in need of comprehensive neuropsychological evaluation. *Applied Neuropsychology*, 15, 1–10.
- Reitan, R. M., & Wolfson, D. (2008b). Serial testing of older children as a basis for recommending comprehensive neuropsychological evaluation. *Applied Neuropsychology*, 15, 11–20.
- Reitan, R. M., & Wolfson, D. (2008c). The use of serial testing in evaluating the need for comprehensive neuropsychological testing of adults. *Applied Neuropsychology*, 15, 21–32.
- Reitan, R. M., & Wolfson, D. (2009). The Halstead-Reitan neuropsychological test battery for adults—theoretical, methodological, and validation bases. In I. Grant & K. M. Adams (Eds.), *Neuropsychological assessment of neuropsychiatric and neuromedical disorders* (3rd ed.). New York: Oxford University Press.
- Sweeny, J. E., Slade, H. P., Ivins, R. G., Nemeth, D. G., Ranks, D. M., & Sica, R. B. (2007). Scientific investigation of brain-behavior relationships using the Halstead-Reitan battery. *Applied Neuropsychology*, 14(2), 65–72.

Handicaps, Behavior and Skills Schedule (HBSS)

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Synonyms

HBSS

Description

The Handicaps, Behavior and Skills schedule (HBS, Wing and Gould 1978) is a semi-structured, investigator-based interview schedule. It is the precursor of the Diagnostic Interview for Social and Communication Disorders (DISCO) (Wing 1999). The HBS was developed to be used for assessing autism symptoms in children with mental retardation. It was designed to elicit a detailed description of the child's skills, impairments, and behavior, in order to assist clinicians in their judgment of the level of development and special needs of a child in different domains. The HBS measures skills and deficits as shown in daily life instead of during formal intelligence tests. It provides the clinician with a profile of the child's pattern of development and behavior and its strengths and difficulties which can be used for treatment planning.

The interview is completed with parents or other caregivers and inquires about specific, easily observable aspects of behavior during the preceding month. It takes from 45 min to 2.5 h to complete, depending on the informant and the complexity of the child's behavior. The schedule consists of items covering behavioral abilities, such as social skills, language and communication, imaginative play, self-care, domestic skills, visuospatial abilities, and school work. In addition, there are items covering challenging behavior often seen in autism, such as sleep disturbance, echolalia, or stereotypic movements. The items within the developmental sections are hierarchically ordered according to the steps in normal development. A higher total score represents a higher level of development. A high total score on the part of the schedule covering specific abnormalities signifies less severe behavioral difficulties.

Historical Background

The HBS was developed in the early 1970s, by Lorna Wing and Judith Gould. It was constructed

to be used in an epidemiological study of all children under 15 years of age with intellectual disability and/or autistic characteristics in the former London Borough of Camberwell. One of the aims of the Camberwell study was to identify children with features of autism and to see if any patterns could be discovered among the clinical pictures thus found. At that time, several checklists, questionnaires, and behavior observations scales were available for the assessment of autistic individuals, but none of those was suitable for this study. Most instruments were designed to identify autism, rather than to collect detailed information about skills and difficulties necessary to investigate patterns of behavior.

Therefore, the Handicaps, Behavior and Skills schedule was constructed. The developmental and self-care items were largely based on an unpublished research interview schedule by Williams and Kushlick and on the Vineland Social Maturity Scale (Doll 1965). The behavioral items were in part derived from a previous rating scale by Wing (1969) and based on the work of DeMyer (1971), Rutter et al. (1971), and Sheridan (1969).

The schedule enabled the researchers to delineate skills, challenging behaviors, and the three specific impairments that were found to co-occur: social problems, communication deficits and repetitive activities, and a lack of imaginative play (later known as the triad of impairments; Wing and Gould 1979).

In 1990, the Elliot House Centre for Social and Communication Disorders was set up by the National Autistic Society (UK). Both children and adults with a variety of developmental disorders and cognitive abilities, ranging from profound mental retardation to superior cognitive skills, were referred to this center. The HBS, which had been designed for children with intellectual disability, did not fulfill the requirements in clinical practice. It was adapted to cover all ages and levels of ability and to record not only current behavior, but also developmental history. The adapted schedule was renamed the Diagnostic Interview for Social and Communication Disorders (DISCO).

Psychometric Data

Very few data are available on the psychometric properties of the HBS. In the original study of Wing and Gould (1978), the inter-informant reliability was investigated. Both parents and nurses/teachers of 104 children were interviewed. The mean Spearman correlation coefficient was 0.8 (ranging from 0.2 to 0.9), and the mean kappa was 0.5 (ranging from 0.1 to 0.8). In general, parents and teachers were in agreement, except for a few behavioral features for which the correlations were weak, such as echolalia, intonation, or special skills.

Age and sex were not significantly associated with the level of agreement between parents and nurses/teachers. The severity of the mental retardation and type of disability were significantly associated with level of agreement. There was better agreement for children with lower developmental levels and fewer behavior problems.

Inter-rater reliability was calculated on 20 interviews. When raw scores were collapsed into a 3-point scale, there was maximum overall agreement between the raters on 30 of the 31 sections in all children.

Internal consistency was investigated in a Dutch study with 72 children (Van Berckelaer-Onnes and van Duijn 1993). Cronbach's alpha coefficients were considered satisfactory, ranging from 0.4 to 0.7, and slightly higher when only the autistic individuals were included, except for the imagination/social play subscale.

Clinical Uses

The HBS has been adapted and reconstructed to make it suitable for clinical use with individuals of all ages and levels of ability. In addition to present functioning, it now covers also developmental history. This revised interview is named the Diagnostic Interview for Social and Communication Disorders (DISCO) (see [DISCO](#)).

References and Reading

- Rutter, M., Bartak, L., & Newman, S. (1971). Autism – A central disorder of cognition and language? In M. Rutter (Ed.), *Infantile autism: Concepts, characteristics and treatment*. London: Churchill Livingstone.
- Sheridan, M. (1969). Playthings in the development of language. *Health Trends*, 1, 7–10.
- Van Berckelaer-Onnes, I., & van Duijn, G. (1993). A comparison between the handicaps behaviour and skills schedule and the psychoeducational profile. *Journal of Autism and Developmental Disorders*, 23(2), 263–272.
- Wing, L. (1969). The handicaps of autistic children: A comparative study. *Journal of Child Psychology and Psychiatry*, 10, 1–40.
- Wing, L. (1999). *Diagnostic interview for social and communication disorders. Manual*. Bromley: Centre for Social and Communication Disorders.
- Wing, L., & Gould, J. (1978). Systematic recording of behaviors and skills of retarded and psychotic children. *Journal of Autism and Childhood Schizophrenia*, 8(1), 79–97.

Hand-Over-Hand Assistance

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Synonyms

[Controlling prompt](#); [Full physical prompt](#)

Definition

Hand-over-hand assistance involves placing one's hands over an individual with autism's hands to help them complete a movement. When using hand-over-hand assistance, the adult is controlling the movements of the child's hands. For example, hand-over-hand assistance can be used to help teach a child how to wash their hands. In this

case, the adult would take the child's hands in their hands and complete all of the steps of hand washing using the child's hands; i.e., the adult would use the child's hands to turn on the water, pump the soap, scrub hands. . .dry hands. Hand-over-hand assistance, also referred to as a full physical prompt, is the most intrusive prompt that you can use, and care should be taken to ensure the adult is not exerting too much force on the child. Because it is the most restrictive prompt, it should be used sparingly when other options are not effective and systematically faded to less intrusive prompting techniques (e.g., partial physical prompts, models).

See Also

- [Prompt Hierarchy](#)
- [Prompting](#)

Handwriting Difficulty

- [Dysgraphia](#)

Hanen Approach

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Definition

The Hanen approach is an intervention approach in which parents and educators are taught to facilitate communication development in young children. The Hanen Centre has developed a number of programs designed to teach parents and educators of young children to promote communication, social skills, and literacy in everyday activities. Two specific approaches have been developed for parents of children with autism: More Than Words[®] – The Hanen Program[®] for Parents of Children on the Autism Spectrum and

TalkAbility[™] – The Hanen Program[®] for Parents of Verbal Children on the Autism Spectrum.

The Hanen approach includes seven programs, four for parents and three for early childhood educators. These programs are:

- It Takes Two to Talk[®] – The Hanen Program[®] for Parents of Children with Language Delays
- Target Word[®] – The Hanen Program[®] for Parents of Children who are Late Talkers
- More Than Words[®] – The Hanen Program[®] for Parents of Children on the Autism Spectrum
- TalkAbility[™] – The Hanen Program[®] for Parents of Verbal Children on the Autism Spectrum
- Learning Language and Loving It[™] – The Hanen Program[®] for Early Childhood Educators
- Teacher Talk[™] Training Series
- ABC and Beyond[™] – The Hanen Program[®] for Building Emergent Literacy

Historical Background

The Hanen Centre is a Canada-based nonprofit center. The first Hanen program was designed in 1975 to teach parents to take a primary role in helping their young children develop communication. This original program became *It Takes Two to Talk*. The Hanen approach now includes seven programs, four for parents and three for early childhood educators, designed to teach parents and educators of young children to promote communication, social skills, and literacy in everyday activities. Hanen programs are now offered in Canada, the United States, and other countries around the world.

Rationale or Underlying Theory

Hanen programs reflect a family-centered model of intervention based on a social-interactionist perspective of language development. The Hanen approach is based on the principles that parents should be involved in their child's intervention process and that communication should be facilitated in naturalistic contexts. Hanen

programs emphasize the critical role of parents, the importance of early intervention, and a focus on everyday routines and activities. The Hanen programs are designed to provide social support for parents in small-group interactions with other parents.

The strategies that parents are taught in *It Takes Two to Talk* are based on the responsivity hypothesis. According to this hypothesis, when parents are taught to respond to child's communication and focus on the child's interest, language input is more easily processed by the child.

The strategies that parents are taught in *More Than Words* are based on a social-pragmatic theory approach. Social communication is facilitated by structuring the environment to promote communication and by responding to the child's communication.

The strategies that parents are taught in the *TalkAbility* program are based on research on development of theory of mind. Specifically, the program emphasizes a connection between the development of language and the development of theory of mind.

Goals and Objectives

The goals of the Hanen approach are to teach parents and educators to promote communication with young children during daily routines and activities. *It Takes Two to Talk* is designed to teach parents to help their young children with language delays develop language skills. *More Than Words* is designed to teach parents to help their children with autism improve social skills, increase receptive language, and engage in interactions. *TalkAbility* is designed to teach parents to help their verbal children with autism improve social skills, participate in conversations, and understand the meaning of nonverbal cues like body language and facial expressions.

Treatment Participants

The Hanen approach is designed for parents, early childhood educators, and young children. *It Takes*

Two to Talk is designed for parents of children with a language delay who are 5 years and younger. *More Than Words* is designed for parents and their children with autism 5 years old and younger. *TalkAbility* is designed for parents and their verbal children with autism between 3 and 7 years old.

Treatment Procedures

The Hanen programs are led by Hanen-certified speech-language pathologists. Small groups of parents or educators participate in group teaching sessions and individual feedback sessions. Individual feedback sessions use videotapes of parent-child or educator-child interactions.

It Takes Two to Talk is a program for parents of children with language delay and is conducted by a Hanen-certified speech-language pathologist. The program includes a preprogram consultation, 6–8 small-group teaching sessions, and three individual visits in which the parent and the speech-language pathologist review videotapes of the parent and child interacting. *It Takes Two to Talk* is designed to teach parents to be responsive to their child. Parents are taught to respond promptly and positively when their child communicates. Parents are also taught to follow the child's lead by talking and paying attention to what the child is interested in. The focus of the program is on teaching practical strategies that parents can use in daily routines and activities. For example, parents are taught to *OWL*: observe, wait, and listen to follow their child's lead during daily routines such as meal time. *It Takes Two to Talk* also teaches parents to use strategies during play, when reading books, and with music. Parents are also taught to identify and respond to their child's individual communication ability. For example, parents of a child who is a *Communicator* learn to recognize and respond to their child's gestures and sounds.

More Than Words is an 11-week program for parents of children with autism and is conducted by a Hanen-certified speech-language pathologist. The program includes a preprogram consultation, eight small-group teaching sessions, and three

individual visits in which the parent and speech-language pathologist review videotapes of the parent and child interacting. The *More Than Words* program is designed to guide parents in adjusting the way they talk to their child with autism during daily activities. Materials include a guidebook and accompanying DVD. The 4-S acronym identifies the key elements of the approach: Say Less, Stress, Go Slow, and Show. Parents learn to simplify what they say to their children, to talk about what their child is paying attention to and stress key words, and to talk slowly and use pictures, gestures, and objects. The program is designed to teach parents to use strategies in daily routines (e.g., meals, bath time).

TalkAbility is conducted by a Hanen-certified speech-language pathologist. The program includes a preprogram consultation, small-group teaching sessions, and individual visits in which the parent and speech-language pathologist review videotapes of the parent and child interacting and videotapes of the child interacting with another child. The *TalkAbility* program is designed to teach parents to facilitate social communication and social skills with their verbal children with autism. Materials include a guidebook and accompanying DVD. The *TalkAbility* program emphasizes social communication skills like conversational turn-taking, storytelling and imaginative play, and peer relationships. In the program, parents are taught to identify their child's communication strengths and weaknesses. For example, the guidebook provides a *Conversation Checklists* for parents to use to identify the stages of conversations that their child struggles with. Parents are taught strategies to teach their child to have successful conversations. For example, for a child who has trouble initiating a conversation, a parent is taught to interpret the child's nonverbal messages (e.g., watching a peer play with a toy) and provide a verbal model (e.g., You want to play.).

Efficacy Information

Efficacy studies have been conducted on some of the Hanen programs, but empirical support is limited. In most of these studies, a quasi-experimental design was employed. Participants

in the treatment groups have been parents participating in a Hanen program. Participants in the control groups have been groups of parents on the wait list to participate in a Hanen program.

In a quasi-experimental study, parents who participated in *It Takes Two to Talk* demonstrated changes in interaction with their children including increases in responsivity and decrease in complexity of language directed at child. Children whose parents participated in *It Takes Two to Talk* demonstrated an increase in expressive language. Other studies have examined effectiveness of approaches that use procedures modified from the Hanen approach. In a quasi-experimental study, parents were taught a modified Hanen approach that emphasized target vocabulary words. Parents demonstrated a change in parental interaction and children demonstrated an increase in expressive vocabulary.

Case studies and quasi-experimental design have been used to examine the outcomes of parents and children who have participated in the *More Than Words* program. Case studies have reported an increase in parental interaction and child social interaction following participation in the *More Than Words* Program. In a quasi-experimental study, parents who participated in the *More Than Words* program had higher scores on measures of parental interaction and children had higher expressive vocabulary relative to the control group.

There are no published efficacy studies of *TalkAbility*.

Outcome Measurement

The Hanen Approach does not specify outcome measurement. Research studies that have examined *It Takes Two to Talk* have measured outcomes of parent interaction and child communication. Parent interaction has been measured using researcher-developed coding systems. Child communication has been measured using standardized measures (e.g., The Sequenced Inventory of Communication Development), researcher-developed tools, and analysis of language samples.

Research studies that have examined the *More Than Words* program have used the following tools to measure outcomes: the *Autism Diagnostic*

Observation Schedule, the *MacArthur-Bates Communicative Development Inventory*, the *Behavior Screening Questionnaire*, the *Questionnaire on Resources and Stress*, and the *Parent Feelings Questionnaire*. Researcher-developed measures have included the *Joy and Fun Assessment* and other parent report measures. Outcomes also have been measured using analysis of parent-child interactions, spontaneous language samples, researcher-child interactions, and child social initiations.

Qualifications of Treatment Providers

For the parent-teaching programs, including *It Takes Two to Talk*, *More Than Words*, and *TalkAbility* programs, qualified treatment providers are speech-language pathologists who have received certification from the Hanen Centre. Certification in *It Takes Two to Talk* requires participation in a 3-day workshop led by a Hanen Instructor. Certification in the *It Takes Two to Talk* program is a prerequisite for certification in all other programs. Certification in *More Than Words* requires participation in another 3-day workshop led by a Hanen Instructor. Certification in *TalkAbility* requires certification in both *It Takes Two to Talk* and *More Than Words* and participation in a 2-day workshop led by a Hanen Instructor.

Early childhood professionals can become qualified treatment providers for the Learning Language and Loving It and ABC and Beyond programs.

See Also

- [Language Interventions](#)
- [Parent Training](#)
- [Speech-Language Pathologist \(SLP\)](#)

References and Reading

Aldred, C., Green, J., & Adams, C. (2004). A new social communication intervention for children with autism: Pilot randomized controlled treatment study suggesting effectiveness. *Journal of Child Psychology and Psychiatry*, 40, 1–11.

- Awcock, C., & Habgood, N. (1998). Early intervention project: Evaluation of WILSTAAR, Hanen and specialist playgroup. *International Journal of Language & Communication Disorders*, 33(S1), 500–505.
- Bohannon, J., & Bonvillian, J. (1997). Theoretical approaches to language acquisition. In J. B. Gleason (Ed.), *The development of language* (4th ed., pp. 259–316). Boston: Allyn & Bacon.
- Brady, N., Warren, S. F., & Sterling, A. (2009). Interventions aimed at improving child language by improving maternal responsiveness. *International Review of Research in Mental Retardation*, 37, 333–357.
- Dominey, P. F., & Dodane, C. (2004). Indeterminacy in language acquisition: The role of child directed speech and joint attention. *Journal of Neurolinguistics*, 17, 121–145.
- Girolametto, L., Pearce, P., & Weitzman, E. (1996a). The effects of focused stimulation for promoting vocabulary in children with delays: A pilot study. *Journal of Childhood Communication Development*, 17, 39–49.
- Girolametto, L., Pearce, P., & Weitzman, E. (1996b). Interactive focused stimulation for toddlers with expressive vocabulary delays. *Journal of Speech and Hearing Research*, 39, 1274–1283.
- Girolametto, L. E. (1988). Improving the social-conversational skills of developmentally delayed children: An intervention study. *The Journal of Speech and Hearing Disorders*, 53, 156–167.
- Girolametto, L., & Weitzman, E. (2006). It takes two to talk® – The Hanen program® for parents: Early language intervention through caregiver training. In R. McCauley & M. Fey (Eds.), *Treatment of language disorders in children* (pp. 77–103). Baltimore: Paul H. Brookes.
- Girolametto, L., Tannock, R., & Siegel, L. (1993). Consumer-oriented evaluation of interactive language intervention. *American Journal of Speech-Language Pathology*, 2, 41–51.
- Girolametto, L., Verbey, M., & Tannock, R. (1994). Improving joint engagement in parent-child interaction. *Journal of Early Intervention*, 18, 155–167.
- Girolametto, L., Pearce, P., & Weitzman, E. (1997). Effects of lexical intervention on the phonology of late talkers. *Journal of Speech, Language, and Hearing Research*, 40, 338–348.
- Girolametto, L., Weitzman, E., & Greenberg, J. (2003). Training day care staff to facilitate children's language. *American Journal of Speech-Language Pathology*, 12, 299–311.
- Girolametto, L., Sussman, F., & Weitzman, E. (2007). Using case study methods to investigate the effects of interactive intervention for children with autism spectrum disorders. *Journal of Communication Disorders*, 40, 470–492.
- Ingersoll, B., & Dvortcsak, A. (2006). Including parent training in the early childhood special education curriculum for children with autism spectrum disorders. *Journal of Positive Behavior Interventions*, 8, 79–87.
- Ingersoll, B., Dvortcsak, A., Whalen, C., & Sikora, D. (2005). The effects of a developmental, social-pragmatic language intervention on rate of expressive

- language production in young children with autistic spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 20, 213–222.
- Manolson, A. (1992). *It takes two to talk: A parent's guide to helping children communicate* (2nd ed.). Toronto: Hanen Centre.
- McConachie, H., Randle, V., & Couteur, L. (2005). A controlled trial of a training course for parents of children with suspected autism spectrum disorder. *The Journal of Pediatrics*, 147, 335–340.
- Pepper, J., & Weitzman, E. (2004). *It takes two to talk®: A practical guide for parents of children with language delays* (2nd ed.). Toronto: Hanen Centre.
- Prelock, P. A., Paul, R., & Allen, E. M. (2011). Evidence-based treatments in communication for children with autism spectrum disorders. In B. Reichow, P. Doehring, D. Cicchetti, & F. Volkmar (Eds.), *Evidence-Based Practices and Treatments for Children with Autism* (pp. 93–169). New York: Springer.
- Prizant, B. M., Wetherby, A. M., & Rydell, P. J. (2000). Communication intervention issues for children with autism spectrum disorders. In A. M. Wetherby & B. M. Prizant (Eds.), *Autism spectrum disorders: A transactional developmental perspective* (pp. 193–224). Baltimore: Brookes.
- Sheldon, M. L., & Rush, D. D. (2001). The ten myths about providing early intervention services in natural environments. *Infants and Young Children*, 14, 1–13.
- Sussman, F. (1999). *More than words: The Hanen program for parents of children with autism spectrum disorder*. Toronto: Hanen Centre.
- Tannock, R., Girolametto, L., & Siegel, L. (1992). Language intervention with children who have developmental delays: Effects of an interactive approach. *American Journal of Mental Retardation*, 97, 145–160.
- Weitzman, E., Girolametto, L., & Greenberg, J. (2006). Adult responsiveness as a critical intervention mechanism for emergent literacy: Strategies for early childcare educators. In L. Justice (Ed.), *Clinical approaches to emergent literacy intervention*. San Diego: Plural.

internship at St. Elizabeths Hospital in Washington, DC, after which she accepted a position as assistant professor at Douglass College, Rutgers, The State University of New Jersey. Dr. Harris has been at Rutgers ever since, retiring in 2007.

Major Appointments (Institution, Location, Dates)

- Douglass College, Rutgers, The State University of New Jersey: 1969–1979. Assistant and associate professor
- Graduate School of Applied and Professional Psychology, Department of Clinical Psychology and Department of Psychology, Faculty of Arts and Sciences, Rutgers, The State University of New Jersey: 1979–2007. Professor I and professor II
- Graduate School of Applied and Professional Psychology, Rutgers, The State University of New Jersey: 1992–1993, acting dean; 1993–2001, dean
- Board of Governors Distinguished Service Professor, Rutgers, The State University of New Jersey: 2003–2007
- Board of Governors Distinguished Service Professor Emerita, Rutgers, The State University of New Jersey: 2007
- Douglass Developmental Disabilities Center, Rutgers, The State University of New Jersey 1972–2007: Founder and executive director

Major Honors and Awards

She is the recipient of numerous awards for distinguished service and contributions to science including the Daniel Gorenstein Memorial Award, the President's Award for Research in Service to New Jersey, the Anne Klein Advocate Award, and the Distinguished Alumni Award, SUNY Buffalo.

Harris, Sandra

Michael D. Powers
The Center for Children with Special Needs,
Glastonbury, CT, USA

Name and Degrees

Sandra L. Harris (1943–) received her BA from the University of Maryland and PhD in clinical psychology from the State University of New York at Buffalo. She completed her clinical

Landmark Clinical, Scientific, and Professional Contributions

Dr. Harris' scientific and clinical interests have been at the forefront of the evolution of

evidence-based treatment for those with autism and their families for over 40 years. From an early interest in teaching language and the generalizability of teaching strategies to families to treatment of core behavioral and social deficits of autism, through research on the effect of autism on parents, grandparents, and siblings, and to demonstrating the effectiveness and sustainability of intensive early behavioral intervention, her work with colleagues and students has been both robust and pragmatic, emphasizing the development of new science but also the training of new professionals in those endeavors. Dr. Harris established one of the first behavioral treatment centers for children with autism in the United States at the Douglass Developmental Disabilities Center and, in so doing, created both a nationally recognized center of excellence for educating individuals with autism across the life span and also a training facility for a generation of psychologists, teachers, and other health-care professionals working in the field.

Short Biography

As a clinical psychologist in the best tradition of the scientist-practitioner model and as an educator, Dr. Harris has mentored a generation of leaders in the field of evidence-based behavioral treatment of those with autism spectrum disorders. While initially devoting her efforts for many years to research and teaching in the graduate program in clinical psychology at Rutgers and simultaneously serving as executive director of the Douglass Developmental Disabilities Center, Dr. Harris became the third dean of the Graduate School of Applied and Professional Psychology (GSAPP), the nation's first university-based school of professional psychology, in the early 1990s. For a period of nearly 10 years, she oversaw a substantial expansion of GSAPP's training, clinical, and community-based programs, in the process of helping to reshape the mission of this flagship program. By honoring her as the Board of Governors distinguished service professor in 2003, the Rutgers community recognized her contributions to research and service

to the children and families of New Jersey and beyond. While she retired as professor emerita in 2007, Dr. Harris continued to serve as executive director of the Douglass Developmental Disabilities Center and to mentor new professionals for nearly 10 additional years, retiring recently.

References and Reading

- Ferraoli, S. J., & Harris, S. L. (2011). Treatments to increase social awareness and social skills. In B. Reichow, P. Doering, D. V. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-based practices and treatments for children with autism* (pp. 171–196). New York: Springer.
- Gill, M. J., & Harris, S. L. (1991). Hardiness and social support as predictors of psychological discomfort in mothers of children with autism. *Journal of Autism and Developmental Disorders*, 21, 407–416.
- Harris, S. L. (1975). Teaching language to nonverbal children-with emphasis on problems of generalization. *Psychological Bulletin*, 82, 564–580.
- Harris, S. L. (1983). *Families of the developmentally disabled: A guide to behavioral intervention*. Elmsford: Pergamon.
- Harris, S. L. (1984). Intervention planning for the family of the autistic child: A multilevel assessment of the family system. *Journal of Marital and Family Therapy*, 10, 157–166.
- Harris, S. L., & Glasberg, B. (2003). *Siblings of children with autism* (2nd ed.). Bethesda: Woodbine House.
- Harris, S. L., & Handleman, J. S. (2000). Age and IQ at intake as predictors of placement for young children with autism: A four to six year follow-up. *Journal of Autism and Developmental Disorders*, 30, 137–142.
- Harris, S. L., & Weiss, M. J. (2007). *Right from the start: Intensive behavioral intervention for young children with autism* (2nd ed.). Bethesda: Woodbine House.
- Harris, S. L., Wolchik, S., & Weitz, S. (1981). The acquisition of language skills by autistic children: Can parents do the job? *Journal of Autism and Developmental Disorders*, 11, 373–384.
- Harris, S. L., Handleman, J. S., & Palmer, C. (1985). Parents and grandparents view the autistic child. *Journal of Autism and Developmental Disorders*, 15, 127–137.
- Harris, S. L., Handleman, J. S., Kristoff, B., Bass, L., & Gordon, R. (1990). Changes in language development among autistic and peer children in segregated and integrated preschool settings. *Journal of Autism and Developmental Disorders*, 20, 23–31.
- Harris, S. L., Handleman, J. S., Gordon, R., Kristoff, B., & Fuentes, F. (1991). Changes in cognitive and language functioning of preschool children with autism. *Journal of Autism and Developmental Disorders*, 21, 281–290.

Hate Crimes/Victimization

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Definition

Many people with autism experience abuse, maltreatment, and other forms of victimization including hate crimes. Experiences of victimization can take many forms, including physical, sexual, emotional, medical, and financial abuse. Physical abuse can include hitting, slapping, and pushing; sexual abuse can involve unwanted touching, sexual contact, or rape; emotional abuse can include bullying, threatening, and intimidating a person; medical abuse can involve overmedicating a person or denying them appropriate medications; and financial abuse involves wrongfully using someone else's finances. These forms of abuse are sometimes called maltreatment, bullying, or neglect. As well as the forms of abuse listed above, neurodiverse people may be subjected to disability hate crimes. Disability hate crimes involve the deliberate targeting of an individual because of his or her disability.

Historical Background

Studies of abuse and victimization have been conducted for decades, consistently indicating that there is an unacceptably high rate of victimization of people across the autistic spectrum in terms of abuse, harassment, bullying, and other forms of victimization. However, the actual estimations of the overall rate differ greatly. These different prevalence rates possibly reflect different methodologies: some studies ask caregivers, others ask parents, and others ask neurodiverse people. The size of the sample (i.e., the number of people questioned in the survey) also is a major factor in terms of the generalizability of the study – smaller samples tend to mean that the

results cannot automatically be assumed to apply to different groups across a broad range of social and cultural contexts.

One study of the experiences of 156 children with autism asked caregivers to indicate whether their children with autism had been abused (Mandell et al. 2005). These caregivers reported that 18.5% of children with autism had been physically abused and 16.6% had been sexually abused. Such abuse frequently resulted in self-harming strategies, such as suicide attempts, by the victims of abuse. Other effects included sexually acting out, running away from home, and psychiatric hospitalization. A similar study, which asked parents of people with Asperger's syndrome whether their child had been abused, reported that 100% of the children had been victimized, on average 1.25 times per week, but for 23% of the children, such victimization occurred on average twice or three times per week (Konstantareas 2005).

In the United Kingdom, the National Autistic Society has reported that 56% of people with autistic spectrum disorder have been bullied or harassed as adults, a figure which rises to 70% for those people with autistic spectrum disorder who live alone (Rosenblatt 2008). This organization wrote a response to the UK Government's "Anti-social behavior: policy and procedure" document which contained one story about a person who was given the pseudonym of Matthew. The document explained that Matthew had experienced verbal abuse, graffiti on his home, and was hospitalized after being physically assaulted within his own home. Matthew's autistic spectrum disorder made it harder to communicate effectively with police. He lacked support workers or family who could help him in lodging such criminal complaints (The National Autistic Society 2009). This problem raises another issue: the importance of support programs that assist disabled people in accessing the criminal justice system. And there is obviously a need to raise awareness of the problems that such barriers exist in the first place.

People with Asperger's syndrome have been called "perfect targets" for bullying and

victimization (Heinrichs and Myles 2003). For instance, when students with Asperger's syndrome are constantly reminded that they do not "fit in" and that their social, academic, or communication skills make them somehow "abnormal," they become not only socially marked but socially isolated and victimized as well. When a potential target is seen as socially isolated and having little peer support, a bully might assume that there will be little or no consequences for their actions. Additionally, many people with Asperger's syndrome are unable to predict the social behavior of others, making it even more difficult for them to avoid potential setups for violence, intimidation, or bullying.

Current Knowledge

There have been some suggestions that certain behaviors associated with autism and with the processes used to interview victims of crimes reduce the likelihood of sexual abuse against an autistic person being recognized. For instance, one author suggests that "because of the use of lengthy, one-time interviews and the need for sustained reciprocity and verbal exchanges," autistic people may face extreme barriers in interviews with law enforcement personnel (Edelson 2010). Additionally, the behaviors which an autistic person might demonstrate in response to sexual abuse may be misattributed to autism rather than a sex crime. Nevertheless, psychological evaluations of people across the autistic spectrum who have experienced various types of trauma, abuse, and harassment consistently indicate that they have lower self-esteem and higher rates of depression and anxiety than those who have not been victimized (Brownlie et al. 2007; Mandell et al. 2005; Shtayermman 2007).

A number of studies suggest that the vast majority of perpetrators of abuse are male and are known to the victim (National Center for Injury Prevention and Control 1998). Perpetrators of abuse include caregivers, family members, other people with disabilities, health-care providers, and acquaintances. The fact that many

people with disabilities have a number of caregivers in their lives, whose work often involves rather intimate tasks, may be one of the factors which puts them at increased risk of abuse. Social and personal boundaries are often at risk of being blurred in the provision of personal assistance (Saxon et al. 2001; Saxton et al. 2006).

The following cases involving crimes against people with autism gained national attention in the media. They involve two US cases: threats to burn an autistic boy's home and the deliberate hitting of an autistic boy in a T-ball game. In Seattle in July 2008, Mark Joe Levison was charged with malicious harassment after threatening to burn the home of an autistic child. In July 2008, he is alleged to have told the child's mother "Keep your fucking son in the house or the backyard like a dog. If you don't I'll burn the (child's bedroom) down" (Jones 2008). The family had only moved into the house 2 weeks earlier and the child's mother, Acquinnette Engen, said "I was terrified." According to police, Levison, 48, also said "I pay \$1,000 a month rent and shouldn't have to see that idiot spinning around and staring at my house" (Pulkkinen 2008). Levison was again arrested a few months later for similar behavior. One report indicated that he had repeatedly threatened to burn down the home, had also taken to imitating the autistic child, and had challenged the child's father to a fight. He pleaded guilty in November 2008 to one count of malicious harassment, a felony, and one count of attempted malicious harassment, a misdemeanor (Sherry 2010). Levison was sentenced to nearly 4 months in jail, as well as a 1-year suspended sentence that could be imposed if he violated the conditions of his release during the next 2 years. Levison was also ordered to undergo substance abuse treatment and anger management classes; he was a self-described "alcoholic" and told the judge "When I'm sober, I would never say words like that" (Seattle Times Staff 2008).

The case of Mark Reed Downs, a 27-year-old T-ball coach from Philadelphia, illustrates the ways in which people with autism can be attacked in social situations. (T-ball is a children's version of baseball). Downs was coaching a children's

team in 2005 when he offered one of his players \$25 to hit Harry Bowers, Jr., an 11-year-old autistic boy on their own team, in the head with a baseball during warm-ups so that he would be unable to play that day. After speaking to the coach, the player threw a ball which bounced and then hit the disabled child in the groin with a pitch. Bowers ran off crying to his mother. She sent him back out to practice and the other child hit him again, this time in the ear. Bowers was unable to play in the game and was taken to the Uniontown Hospital. Bowers was targeted because his autism meant that he was not a great player, and every player is required to play three innings unless they are injured. The boy who threw the balls, Keith Reese, Jr., was one of the better pitchers in their team and the team was in the play-offs (finals) that day. He testified in court that after he had struck Bowers on the first occasion, "He (Downs) told me to go out there and hit him harder. So I went out there and hit him in the ear." Reese, Jr., testified that he did not receive the \$25 from Downs. Downs was convicted of criminal solicitation to commit simple assault and corruption of minors but was found not guilty of the more serious charge of criminal solicitation to commit aggravated assault, and the jury could not reach agreement on whether his actions constituted reckless endangerment. Downs was sentenced to 1–6 years in prison but was granted bond while appealing to the state Superior Court (Sherry 2010).

Awareness of disability hate crimes, a particular form of victimization, is growing internationally (Sherry 1999, 2000a, b, 2002, 2003, 2004, 2010). In these crimes, the perpetrator targets a person because of their disability identity. They are called "hate crimes" or "bias crimes," depending on the particular local legislation. Perpetrators often receive additional sentencing for the hate crime, as well as the original crime, because hate crimes not only affect the individual but also the whole community to which that person belongs. Hate crimes send a message of intolerance that might mean people in the group consider a particular place unsafe and a "no go zone," so the perpetrator is punished for that effect of their actions as well.

Future Directions

The future direction for dealing with and preventing abuse, maltreatment, and other forms of victimization involves the development of a multidimensional response. One author recommends six different areas which could be addressed to eliminate bullying of children with Asperger's syndrome: empowering victims, empowering bystanders, empowering teachers, understanding bullies, empowering parents, and empowering schools (Dublin 2007). Presumably, the responses for adults would be somewhat different, given that they are not in school. Some of the more general responses which have (Sherry 1999, 2000a, b, 2002, 2003, 2004, 2010) been developed to prevent abuse include training programs for both potential victims and caregivers to increase awareness of abuse issues; sex education programs which emphasize choice-making, personal rights, and assertiveness training; and staff screening programs involving improved reference and police checks to weed out convicted sex offenders from caregiving positions (Sobsey 1994). It is essential that child protection workers, law enforcement personnel, and educators (particularly in special education settings) be provided with sufficient training in order to appropriately respond to cases of disability abuse.

Unfortunately, many child protection workers lack knowledge about disability issues. This lack of confidence dealing with disability issues has led to the situation where disabled children are overrepresented among victims of abuse but underrepresented among the caseloads of child protection workers (Orellove et al. 2000). As a result, disabled victims of abuse often experience significant difficulty in accessing appropriate services. Many professionals also report a lack of training in dealing with abuse histories of male clients, which may compound these problems (Lab et al. 2000).

Another specific challenge for investigators may be the use of assisted communication by some people with autism. Assisted communication may involve the use of symbols, pointing devices, computer aids, a communication partner, word prediction software, and many other

individualized approaches. These assisted communication tools are necessary because up to 25% of people with autism have communication impairments (Howlin 2006). However, many law enforcement officers and court systems are unfamiliar with these communication techniques, creating an additional barrier for victims who seek redress through the judicial system.

Abuse, harassment, vilification, and maltreatment are unfortunately common experiences for people with autism. They can have devastating and lifelong effects on self-esteem and leave a person struggling with depression and anxiety. Awareness of this problem is growing within the community and within the criminal justice system, as more victims come forward and discuss their experiences. However, many cases are not reported, for fear of retaliation or for fear that the victim will not be believed. Also, there are challenges associated with limited awareness about assisted communication devices within the criminal justice system. There is a definite need for more advocacy, support, and awareness of this problem.

See Also

► Bullying

References and Reading

- Brownlie, E. B., Jabbar, A., Beitchman, J., Vida, R., & Atkinson, L. (2007). Language impairment and sexual assault of girls and women: Findings from a community sample. *Journal of Abnormal Child Psychology*, 35(4), 618–626.
- Dublin, N. (2007). *Asperger syndrome and bullying: Strategies and solutions*. London: Jessica Kingsley.
- Edelson, M. G. (2010). Sexual abuse of children with autism: Factors that increase risk and interfere with recognition of abuse (Electronic Version). *Disability Studies Quarterly*, 30. Retrieved May 15, 2011, from <http://www.ds-q-sds.org/article/view/1058/1228>
- Heinrichs, R., & Myles, B. S. (2003). *Perfect targets: Asperger syndrome and bullying – Practical solutions for surviving the social world*. Shawnee Mission: Autism Asperger.
- Howlin, P. (2006). Augmentative and alternative communication systems for children with autism. In T. Charman & W. Stone (Eds.), *Social and communication development in autism spectrum disorders: Early identification, diagnosis, and intervention* (pp. 236–266). New York: Guilford Press.
- Jones, L. A. (2008). South Seattle man accused of harassing autistic child, threatening arson. *The Seattle Times*.
- Konstantareas, M. (2005). Anxiety and depression in children and adolescents with Asperger syndrome. In K. Stoddart (Ed.), *Children, youth and adults with Asperger syndrome: Integrating multiple perspectives* (pp. 47–59). London: Jessica Kingsley.
- Lab, D. D., Feigenbaum, J. D., & De Silva, P. D. (2000). Mental health professionals' attitudes and practices towards male childhood sexual abuse. *Child Abuse & Neglect*, 24(3), 391–409.
- Mandell, D. S., Walrath, C. M., Manteuffel, B., Sgro, G., & Pinto-Martin, J. A. (2005). The prevalence and correlates of abuse among children with autism served in comprehensive community-based mental health settings. *Child Abuse & Neglect*, 29(12), 1359–1372.
- National Center for Injury Prevention and Control. (1998). *Sexual violence against people with disabilities*. Atlanta: Centers for Disease Control and Prevention, Division of Violence Prevention.
- Orellove, F. P., Hollahan, D. J., & Myles, K. T. (2000). Maltreatment of children with disabilities: Training needs for a collaborative response. *Child Abuse & Neglect*, 24(2), 185–194.
- Pulkkinen, L. (2008). Neighbor sentenced for directing drunken tirades at autistic boy. Retrieved August 1, (subsequently made a private post), 2009, from http://www.seattlepi.com/local/395505_levinson10.html
- Rosenblatt, M. (2008). *I Exist: The message from adults with autism in England*. Bristol: The National Autistic Society.
- Saxon, M., Curry, M. A., Powers, L. E., Maley, S., Eckels, K., & Gross, J. (2001). Bring my scooter so I can leave you. *Violence Against Women*, 7(4), 393–417.
- Saxton, M., McNeff, E., Powers, L., Limont, M., & Benson, J. (2006). "We're all little John Waynes": A study of disabled men's experiences of abuse by personal assistants. *Journal of Rehabilitation*, 72(4), 3–13.
- Seattle Times Staff. (2008, July 24). South Seattle man accused of harassing autistic child, threatening arson. *The Seattle Times*.
- Sherry, M. (1999). Hate crimes against people with disabilities. *Hate Crime Conference*, The University of Sydney.
- Sherry, M. (2000a). Hate crimes and disabled people. *Social Alternatives*, 19(4), 23–30.
- Sherry, M. (2000b, July 1). Talking about hate. Bent: A Journal of Crip Gay Voices Retrieved 5 May, 2011.
- Sherry, M. (2002). Don't ask, Tell or respond: Silent acceptance of disability hate crimes. Paper presented at the Ed Roberts Post doctoral fellowship in disability studies at the University of California at Berkeley Public Lecture Series 21 November.
- Sherry, M. (2003). Exploring disability hate crimes. Paper presented at the 16th Annual Meeting of the Society for Disability Studies.

- Sherry, M. (2004). Exploring disability hate crimes. *Review of Disability Studies: An International Journal*, 1(1), 51–60.
- Sherry, M. (2010). *Disability hate crimes: Does anyone really hate disabled people?* London: Ashgate.
- Shtayermman, O. (2007). Peer victimization in adolescents and young adults diagnosed with Asperger's syndrome: A link to depressive symptomatology, anxiety symptomatology and suicidal ideation. *Issues in Comprehensive Pediatric Nursing*, 30(3), 87–107.
- Sobsey, D. (1994). *Violence and abuse in the lives of people with disabilities: The end of silent acceptance?* Baltimore: Paul H Brookes.
- The National Autistic Society. (2009). A response from The National Autistic Society: NAS comments on the anti-social behaviour bill part 2 (Housing). Retrieved October 25, 2009.

Hatred of Sound

- [Misophonia](#)

Hay Fever

- [Allergies](#)

HBSS

- [Handicaps, Behavior and Skills Schedule \(HBSS\)](#)

HEA 2008

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Definition

The Higher Education Opportunity Act of 2008 is also known as Public Law 110–135. It is the latest reauthorization of the Higher Education Act of

1965 (Pub. L. No. 89–329), originally signed into law by President Lyndon Johnson on November 8, 1965. The Higher Education Opportunity Act of 2008 is important for individuals with a diagnosis of autism or an autism spectrum disorder because it creates new legislative requirements for access to postsecondary education for individuals with an intellectual disability.

Historical Background

The real importance of the passage of the Higher Education Opportunity Act of 2008 (HEOA) for individuals with a diagnosis on the autism spectrum is in its legislative mandates for higher educational opportunities for individuals with an intellectual disability. The original Higher Education Act (HEA) of 1965 does not specifically provide for educational opportunities for individuals with a disability. Nevertheless, the history of the Higher Education Act of 1965 and its subsequent amendments and reauthorizations provides a context for understanding the new legislative provisions in the HEOA of 2008. While the Education Act of 1965 and its subsequent revisions has provided for federal support for many aspects of higher education in the United States, the most extensive impact of the legislation has come from Title IV of the Act which provided for direct federal financial aid to students pursuing higher education as well as federally subsidized loans for these students. The limits on access to this financial aid is central to the new legislative mandates in HEOA of 2008 which support access to higher education for individuals with an intellectual disability. This section will provide a brief historical context to the original passing of the Higher Education Act and examine the major developments in the legislation in its nine subsequent reauthorizations since 1965.

In the United States, the establishment of institutes of higher education has always been a private or state responsibility. The United States has never established a national university. The federal role in higher education has been via certain federal laws and regulations and limited, targeted federal aid. The early aspect of direct federal aid to

higher education came via legislation that mandated that certain lands be set aside for the establishment of institutes of higher education. The first provision for this was as part of the Northwest Territories Act of 1787 and then similar provisions where the central purpose of the Morrill Acts of 1862 and 1890 creating the so-called Land Grant Colleges. Probably, the most significant foray of the federal government into support for higher education was the Servicemen's Readjustment Act of 1944, more commonly known as the "GI Bill." The GI Bill provided financial support to honorably discharged veterans for from 1 to 4 years of study in an institute of higher education. Prior to the passage of the Higher Education Act, congress passed the National Defense Education Act in 1958. This Act used a needs-based formula to provide low-interest loans to college students studying science, math, or foreign languages. Students who became teachers had an option to have their loan forgiven.

As part of President Lyndon Johnson's War on Poverty, he commissioned a taskforce on higher education and the role of the federal government in student aid. The recommendations from this taskforce ultimately become the major provisions of the Higher Education Act (HEA). The HEA was sponsored in congress by Wayne Morse in the senate and by Edith Green in the house, both representing the state of Oregon. The Act was signed into law by President Lyndon Johnson on November 8, 1965, at his alma mater, the Southwest Texas State College (now known as Texas State University). The Act had seven major sections identified as Title I–Title VII. Title I of the Act provided for grants to strengthen community service projects in particular those addressing such problems as poverty, housing, transportation, and youth opportunities. Title II provided grants for the expansion of college library collections as well as for an increase in the education and training of college librarians. Title III provided for aid to developing institutions with its aim primarily to support the growth of traditional African-American colleges. Technical and 2-year colleges could also receive support under Title III. Title IV is probably the best known aspect of the HEA and will be discussed in more detail below.

Title IV provided for both direct federal financial aid as well as federally insured loans for eligible college students enrolled full-time. Title V established a Teacher Corps for the development of better teacher education. Title V is later moved to a reauthorization of the Elementary and Secondary Education Act. Title VI established grants for the use of technology in the improvement of undergraduate education. Title VII afforded institutes of higher education with funding for new construction. Title VII amended and incorporated the Higher Education Facilities Act of 1963 into the HEA.

Title IV has been the most influential aspect of the HEA having substantially increased access to higher education for a large segment of American society. Title IV of the HEA of 1965 had four parts labeled A–D. Part A itself and two main provisions were the first created Educational Opportunity Grants, which are now known as Supplemental Educational Opportunity Grants. The Educational Opportunity Grants were funneled through the states to the institutes of higher education who could then distribute them to students in need based on their own criteria. The second provision in Part A of Title IV incorporated an existing program, Upward Bound, with a new program called Talent Search in order to encourage college attendance among disadvantaged youth. Part B of Title IV established Guaranteed Students Loans, loans that would be funded by private lenders but backed by federal guarantees. There have been numerous changes in this program over the years, but the essential features of the original legislation still exist: loans are made directly to student families from private lenders, but the lenders have the promise of payment from the federal government if the recipient defaulted on the loan. In addition, based on need, the loan recipient may have some of her or his interest subsidized while attending school.

A third aspect of Title IV was the incorporation of the Federal Work Study Program into the HEA, which was authorized in Part C of the Title. A Federal Work Study Program had already been started as part of the War on Poverty, but would now be moved to the Department of

Education from the Office of Economic Opportunity. Federal Work Study is funded by the institute of higher education with matching funds from the federal government. The final provision under Title IV of the HEA was to move the National Defense Student Loan (NDSL) Program from the Office of Economic Opportunity to Department of Education. The NDSL program is now known as the Perkins Student Loan program. It is a direct loan program and is funded by both the federal government and the individual institutions of higher education. It provides low-interest loans to financially needy undergraduate, graduate, and professional students.

Current Knowledge

The Higher Education Opportunity Act (HEOA) of 2008 (Public Law 110–135) is the ninth reauthorization of the original 1965 law (Pub. L. No. 89–329). The 2008 law was passed on July 31, 2008, and signed into law on August 14, 2008. The reauthorization was 5 years late and completed after congress passed 14 extensions of the statutory deadline. Originally, the 1998 reauthorization only extended until September 30, 2003.

While the reauthorization addresses literally hundreds of different provisions within the law, the essential aspects of the new law that have a new and unique impact for individuals with a diagnosis on the autism spectrum are the provisions related to postsecondary education for individuals with an intellectual disability. Thus, this section will primarily explicate the new rights and mandates concerning postsecondary education for individuals with an intellectual disability. The end of the section will briefly overview the other main provisions of the new law.

There are four main features of the HEOA of 2008 that address students with an intellectual disability. The law provides for a competitive federal grant program for institutes of higher education to create model comprehensive postsecondary transition programs for students with an intellectual disability. HEOA also provides authorization for the creation of two centers to support the development of transition programs. One initiative is to create a National Center for

Information and Technical Support for Postsecondary Students with Disabilities. The second is to create a Coordinating Center for institutes of higher learning offering postsecondary transition programs for individuals with an intellectual disability. And finally, the law opens the eligibility for receiving Pell Grants, Federal Supplemental Education Opportunity Grants, and Federal Work Study for students with an intellectual disability. Importantly, for students enrolled in an approved comprehensive postsecondary transition program, the law modifies the requirements for full-time matriculation in a degree-granting program and for demonstrating satisfactory academic progress.

The Higher Education Opportunity Act of 2008 expands access to higher education for students with an intellectual disability. Probably, the most substantial change in the law for students with an intellectual disability is modifications to Title IV of the law addressing student financial aid. Prior to this, it was difficult for a student with an intellectual disability to be eligible for any type of federal financial aid because they often lacked a regular high school diploma (or General Educational Development equivalent). Even if they did meet the diploma requirement, they often failed the Title IV “ability to benefit” test, based on satisfactory academic progress as a full-time student in a degree-granting program. Changes to Title IV give the secretary of education broad authority to waive restrictions on eligibility for federal financial aid for students with an intellectual disability if they meet two general criteria. The student must be enrolled in an approved comprehensive transition program at an institution of higher education, and they must maintain satisfactory progress as determined by the institution.

Title VII (Part D, Section 760) of the HEOA defines the criteria for a comprehensive transition and postsecondary program for students with intellectual disabilities as well as defining the term “a student with an intellectual disability.” The law identifies a comprehensive postsecondary transition program as a degree, certificate, or nondegree program offered by an institution of higher education that is designed to support students with an intellectual disability

who are seeking to continue academic, career and technical, and independent living instruction in order to prepare for gainful employment; the program includes an advising and curriculum structure and requires students to participate on not less than a half-time basis, as determined by the institution and focusing on academic components. The academic components must include regular involvement with nondisabled individuals via one or more of the following: regular enrollment in credit-bearing courses, auditing or participating in credit-bearing courses, enrollment in noncredit-bearing or nondegree courses, or participation in internships or work-based training.

The change in Title VII defines the meaning of the term a “student with an intellectual disability.” Specifically, the law indicates that such a student is a student with mental retardation or a cognitive impairment, characterized by significant limitation in intellectual and cognitive functioning, and adaptive behavior as expressed in conceptual, social, and practical adaptive skills. In addition, a student with an intellectual disability is a student who is currently or formally eligible for a free appropriate public education (FAPE) under the Individuals with Disabilities Education Act (IDEA).

Subpart of 2 of Part D of Title VII of the HEOA creates the authorization of model comprehensive transition and postsecondary programs for students with intellectual disabilities. On a competitive basis, institutes of higher education will be able to apply for federal matching grants to support comprehensive transition programs. The grants, once awarded, will last 5 years. The first set of grants was awarded in October of 2010 to 27 institutes of higher learning. The list of all grantees is available via the National Coordinating Center which is housed at the Institute for Community Inclusion at the University of Massachusetts Boston (<http://www.thinkcollege.net/tpsid-coordinating-center>). The main criteria for institutes of higher education receiving funding is as follows: The program must serve students with an intellectual disability; it must provide for the inclusion of students with an intellectual disability with the regular academic and extracurricular activities of the institution; it must use a person-centered planning approach; and it must provide a focus on academic enrichment, socialization,

independent living skills, and integrated work experiences that lead to gainful employment.

In October of 2010, an award was also made to establish a National Coordinating Center for institutions of higher education offering inclusive comprehensive postsecondary transition programs for students with intellectual disabilities. This National Coordinating Center is housed at the Institute for Community Inclusion at the University of Massachusetts Boston (<http://www.thinkcollege.net/tpsid-coordinating-center>). The HEOA stipulates several key responsibilities for the National Coordinating Center. First, it must provide technical assistance for the development, evaluation, and improvement of grantee transition programs. Secondly, it should assist in developing the standards and measurable competencies for the awarding of a meaningful credential by the comprehensive postsecondary transition programs. In establishing a meaningful credential, the Coordinating Center needs to work with the transition programs in identifying the necessary programmatic components for these programs, how student progress should be evaluated, and what should determine student eligibility.

The law also calls for the creation of a National Center for Information and Technical Support for Postsecondary Students with Disabilities (National Center). As of 2011, no award for the creation of such a center had been made. The law envisions the National Center as providing technical assistance to two primary constituencies: students and their families and institutions of higher education. The law indicates that the National Center would provide information and technical assistance to individuals with a disability and their families both while they are still receiving secondary education and while they are participating in some type of postsecondary education or transition program. Included in this is a mandate for support to Individualized Education Program Teams, who are mandated to provide transition planning for students in secondary education by the Individuals with Disabilities Education Act. The law calls for the National Center to provide assistance to administrators, staff, and faculty of institutes of higher education to better recruit and retain students with a disability. Specifically, the law encourages the center to collect and disseminate proven

best-practices models for transitioning and supporting students with a disability in postsecondary education. It also calls on the National Center to provide training modules and tutorials for administrators, staff, and faculty on the best practices of supporting and retaining students with disabilities in postsecondary education.

The reauthorization of the Higher Education and Opportunities Act of 2008 does provide numerous other changes in the law, in addition to those that directly address students with an intellectual disability. The variety of changes are too numerous to elaborate on all of them here. The most important changes are summarized briefly below.

One major area of changes in the HEOA of 2008 revolves around greater transparency and reporting requirements on college costs. There are several components of the law that create new requirements for institutes of higher learning to calculate, publish, and report to the department of education accurate estimates of the net cost of attending school at their institution. In addition to the financial aid changes specific to individuals with an intellectual disability described above, the law makes several other important changes to federal financial aid law. Concerning the Pell Grant, the maximum award is increased and it is made available to students year-round (in the past, it was not available for summer sessions) in order to encourage a shorter time to degree in more schools. A major change in student eligibility for financial aid is a provision that permits institutes of higher education to register students who are also dually (or concurrently) enrolled in secondary education. The provision may also provide significant new opportunities for students with an intellectual disability. In addition, the law calls for simplifying and streamlining the federal financial aid process, including fewer and more understandable questions on the Free Application for Federal Student Aid (FAFSA).

Future Directions

The Higher Education Opportunity Act (HEOA) of 2008 (PL 110–135) was signed into law by President George W. Bush on August 14, 2008. This reauthorization of the original 1965 law opened up

dramatically new opportunities for individuals with an intellectual disability to participate in higher education. As of October of 2008, the U.S. Department of Education had funded 27 model “comprehensive transition and postsecondary programs for individuals with an intellectual disability” at both 2- and 4-year institutes of higher education throughout the United States. In addition, the Institute for Community Inclusion at the University of Massachusetts Boston received funding to establish the National Coordinating Center to support the 27 initial model programs and any other programs that might develop.

The mandate for the National Coordinating Center involves the development of national standards for the provisions of education via the comprehensive transition programs, qualitative and quantitative measures for student outcomes on program strengths, and the criteria necessary for establishing a meaningful credential. It can be expected that as these requirements are defined and solidified, more institutions of higher education will become involved in the provision of educational services to individuals with an intellectual disability.

The law calls for the creation of a National Center for Information and Technical Support for Postsecondary Students with Disabilities. As of 2011, no grant had been awarded to create such a center. It is expected that the Department of Education will create such a center and that this center will become an important advocate to individuals with an intellectual disability, and their families, seeking to continue their education beyond secondary school. Then mandate for the National Center also calls for it to be a resource for Independent Education Program Teams doing IDEA-mandated transition planning for students in secondary schools. Such a national resource of information and technical assistance should greatly increase the incidence of postsecondary education being part of the formal IEP transition plans for students with an intellectual disability.

See Also

- [Comprehensive Transition Program](#)
- [Individual Education Plan](#)

- Individualized Transition Plan (ITP)
- Individuals with Disabilities Education Act (IDEA)
- Transition Planning
- Transitional Living

References and Reading

- Grigal, M., & Hart, D. (2010). *Think college: Post-secondary education options for students with intellectual disabilities*. Baltimore: Paul H. Brookes.
- Higher Education Opportunity Act of 2008, Pub. L. No. 110–31, USC.
- Higher Education Resource Hub. (n.d.). Higher Education Act of November 8, 1965; Retrieved May 29, 2011, from <http://www.highered.org/resources/HEA.htm>
- TG Research and Analytical Services. (2005). Higher Education Act: Forty years of opportunity. Retrieved May 29, 2011, from <http://www.tgslc.org/>: http://www.tgslc.org/pdf/HEA_History.pdf
- U.S. Department of Education Office of Civil Rights. (n.d.). Students with Disabilities Preparing for Post-secondary Education: Know Your Rights and Responsibilities. Retrieved July 23, 2010, from ED.Gov: <http://www2.ed.gov/about/offices/list/ocr/transition.html>

distribution of health may be considered unnecessary, avoidable, unjust, and unfair. Outside of the USA, such disparities are typically referred to as health inequalities or inequities.

References and Reading

- Graham, H. (2007). *Unequal lives: Health and socioeconomic inequalities*. Maidenhead: McGraw Hill.
- Healthy People 2020. Retrieved from <http://www.healthypeople.gov/2020/default.aspx>
- World Health Organization (2008). *Closing the gap in a generation: Health equity through action on the social determinants of health*. Final report of the Commission on the Social Determinants of Health. Geneva: Author.
- World Health Organization and the World Bank. (2011). *World report on disability*. Geneva: World Health Organization.

H

Health Inequalities

- Health Disparities

Health Disparities

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Synonyms

[Health inequalities](#); [Health inequity](#)

Definition

Differences in health status between different population groups. Some health disparities may be attributable to biological variations or free choice. Others may be attributable to environmental conditions beyond the control of individuals concerned. In these instances, the uneven

Health Inequity

- Health Disparities

Healthcare Transition for Individuals with Autism

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Definition

Healthcare transition for individuals with ASD is defined as the transition from pediatric to adult healthcare systems.

Historical Background

The access to and provision of quality healthcare for individuals with disabilities including autism spectrum disorder (ASD) has been identified as a significant public health issue (Bruder et al. 2012). During the period of transition from pediatric care to adult healthcare for young adults with ASD, the gap in access to high-quality patient-centered care widens further (Cooley 2013; Lotstein et al. 2008). Transition age is generally defined as occurring between the ages of 14 and 17 years, up to the age of 22 years (AAP et al. 2011). In addition, compared to youth with other disabilities, youth and young adults with ASD are more likely to require specialty care for common comorbidities such as epilepsy, gastrointestinal problems including inflammatory bowel disease (IBD), and anxiety and depression, as well as respiratory, food, and skin allergies (AAP et al. 2011).

Many obstacles can interfere with a smooth healthcare transition for TAY with ASD. The possible roadblocks include the attitudes of stakeholders; age limits in pediatric practices; complexity of health conditions; lack of insurance reimbursement for management of medical complexity often associated with transition age youth (TAY) with ASD; and, most importantly, the scarcity of competent educated healthcare providers prepared to meet the needs of this ever-growing patient demographic (Oswald et al. 2013).

Individuals with ASD have higher rates of healthcare utilization (Croen 2006; Kogan et al. 2008; Warfield and Gulley 2006; Liptak 2006), an increased burden of unmet healthcare needs (Newacheck and Kim 2005), and decreased satisfaction with the medical care received (Leslie and Martin 2007; Souders 2002). These data further support the imperative for improvement in access to quality healthcare services for individuals with ASD across the life span and especially during the period of transition from pediatric to adult healthcare (Iannuzzi et al. 2018). Many medical comorbidities can first present during the adolescent years, adding to the challenges of meeting the medical needs of this patient population (Bauman 2010).

In addition, access to developmentally appropriate quality healthcare for TAY with ASD is

extremely limited due to a critical shortage of primary care providers, including physicians and advanced practice nurses, willing to accept TAY with ASD into their adult primary care practices (Warfield et al. 2015). Studies addressing the scarcity of primary care providers have suggested that the lack of providers for this population may be a result of the lack of content, including an experiential component, focused on the healthcare needs of individuals with ASD and other developmental disabilities within health professional education programs (Bruder et al. 2012; Patel and O'Hare 2010).

Current Knowledge

Many TAY with ASD present with complex medical, behavioral, and sensory processing challenges. For example, some studies have described a prevalence of up to 80% for sleep disorders in individuals with ASD, as compared to the rate of 30% for individuals without ASD (Bauman 2010; Malow et al. 2012). Seizure disorders can occur in up to 35% of individuals with ASD, and about 60% of patients with ASD have abnormal electroencephalograms (EEGs) (Bauman 2010). The true prevalence of gastrointestinal disorders (GI) in individuals with ASD is not known; however, current estimates range anywhere from 9% to 70% (Buie et al. 2010).

Individuals with ASD can also have a hypersensitivity to auditory, visual, and tactile stimuli. These sensory challenges can be especially difficult to manage in a medical setting, where an individual may be exposed to novel smells, sounds, lighting, and medical staff that are unfamiliar (Scarpinato et al. 2010). Many individuals with ASD, including TAY, can have difficulties in overly stimulating environments like the environments found in most healthcare settings (Aylott 2010). These stimuli may result in hyper-arousal and maladaptive behavior, which can often be minimized by accommodating an individual's sensory profile and any specific sensory sensitivity they may have. Having some understanding and appreciation of the significance of these sensory processing challenges before a medical procedure or physical exam can greatly reduce the

level of distress for the patient, caregiver, and providers.

A healthcare professional may misinterpret maladaptive behaviors and not understand that these maladaptive behaviors can be a manifestation of pain. This misunderstanding can lead to serious errors in assessment and treatment (Iannuzzi et al. 2014). Assessment of discomfort can be particularly difficult in nonverbal patients with autism; pain is often communicated through maladaptive behavior (Kopecky et al. 2013). It is critical for clinicians to specifically ask the individual with ASD or their parent, guardian, or caretaker about sensory sensitivities and triggers, preferred communication methods, and, most importantly, how the individual communicates that they are in distress or pain. These are questions that most clinicians are not trained to ask and can be crucial in assessment and the development of a treatment plan that is individualized to the unique comprehensive needs of an individual with ASD presenting for care.

A clinician in the position of providing healthcare in either an emergent or primary care setting needs to understand that, at times, maladaptive behavior can also be a red flag, signaling the presence of an underlying medical issue. Lack of knowledge about the connection between aberrant behavior and pain is an issue that can lead to misdiagnosis and suboptimal medical care (Buie et al. 2010). Likewise, self-injurious or aggressive behavior exhibited by an individual with autism presenting for emergent care may represent an underlying medical issue. Thus, it is incumbent upon primary care and emergency clinicians to first investigate the possibility of an underlying medical condition that may explain aberrant behaviors (Iannuzzi et al. 2014). This is especially true for nonverbal individuals whose only means of communicating physical pain or distress is to exhibit maladaptive behaviors.

Future Directions

The current healthcare workforce, including physicians and advanced practice nurses, is simply not equipped or prepared to meet the medical

needs of adults with ASD (McDougle 2013). The need for a clinically competent healthcare workforce that is able to meet the healthcare needs of TAY with ASD will continue to increase as more young adults with ASD age out of pediatric healthcare practices. In order for healthcare professionals to develop competencies, a curriculum that is focused on the unique care needs of this patient population must be embedded into existing healthcare professional educational programs. Since individuals with ASD are growing older, are living longer, and develop many of the health issues and concerns of the typical aging population, their medical needs will increase in scope and severity (Drum et al. 2009; Henry et al. 2011; Smith et al. 2010). Because individuals with ASD across the life span can present in any healthcare setting, all healthcare providers, need to be prepared to meet the healthcare needs of this growing patient population (Smeltzer et al. 2014).

See Also

- ▶ [Autism Spectrum Disorder \(ASD\)](#)
- ▶ [Healthcare Transition for Individuals with Autism](#)
- ▶ [Quality of Life for Transition-Age Youth with ASD](#)

References and Reading

- American Academy of Pediatrics, American Academy of Family Physicians, American College of Physicians, & Transitions Clinical Report Authoring Group. (2011). Clinical report: Supporting the healthcare transition from adolescence to adulthood in the medical home. *Pediatrics*, 128(1), 182–200.
- Aylott, J. (2010). Improving access to health and social care for people with autism. *Nursing Standard*, 24(27), 47–56.
- Bauman, M. L. (2010). Medical comorbidities in autism: Challenges to diagnosis and treatment. *Neurotherapeutics*, 7(3), 320–327.
- Bruder, M. B., Kerins, G., Mazzeella, C., Sims, J., & Stein, N. (2012). Brief report: The medical care of adults with autism spectrum disorders: Identifying the needs. *Journal of Autism and Developmental Disorders*, 42(11), 2498–2504.
- Buie, T., Campbell, D., Fuchs, G., Furuta, G., Levy, J., Van de Water, J., . . . Winter, H. (2010). Evaluation,

- diagnosis and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125(Suppl 1), S1–S18.
- Cooley, W. C. (2013). Adolescent health care transition in transition. *Journal of American Medical Association (JAMA) Pediatrics*, 167(10), 897–899.
- Croen, L. A. (2006). A comparison of healthcare utilization costs of children with and without autism spectrum disorders in a large group model health plan. *Pediatrics*, 118(4), 1203–1211.
- Drum, C. E., Krahn, G. L., Peterson, J. J., Horner-Johnson, W., & Newton, K. (2009). Health of people with disabilities: Determinants and disparities. In C. E. Drum, G. L. Krahn, & H. Bersani (Eds.), *Disability and public health* (pp. 125–144). Washington, DC: American Association on Intellectual and Developmental Disabilities.
- Henry, A. D., Long-Bellil, L., Zhang, J., & Himmelstein, J. (2011). Unmet need for disability-related health care services and employment status among adults with disabilities in the Massachusetts Medicaid program. *Disability and Health Journal*, 4(4), 209–218. <https://doi.org/10.1016/j.dhjo.2011.05.003>.
- Iannuzzi, D. A., Cheng, E. R., Broder-Fingert, S., & Bauman, M. L. (2014). Brief report: Emergency department utilization by individuals with autism. *Journal of Autism and Developmental Disorders*, 45(4), 1096–1102. <https://doi.org/10.1007/s10803-014-2251-2>.
- Iannuzzi, D., Rismiller, P., Duty, S. M., Feency, S., Sullivan, M., Curtin, C. (2018). *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-018-3846-9>.
- Kogan, M. D., Strickland, B. B., Blumberg, S. J., Singh, G. K., Perrin, J. M., & van Dyck, P. C. (2008). A national profile of the health care experiences and family impact of autism spectrum disorder among children in the United States, 2005–2006. *Pediatrics*, 122(6), 1149–1158.
- Kopecky, K., Broder-Fingert, S., Iannuzzi, D., & Connors, S. (2013). The needs of hospitalized patients with autism spectrum disorders: A parent survey. *Clinical Pediatrics*, 52(7), 652–660. <https://doi.org/10.1177/0009922813485974>.
- Leslie, D. L., & Martin, A. (2007). Health care expenditures associated with autism spectrum disorders. *Archives of Pediatrics and Adolescent Medicine*, 161(4), 350–355.
- Liptak, G. S. (2006). Satisfaction with primary health care received by families of children with developmental disabilities. *Journal of Pediatric Health Care*, 20(4), 245–252.
- Lotstein, D. S., Inkelas, M., Hays, R. D., Halfron, N., & Brook, R. (2008). Access to care for youth with special health care needs in the transition to adulthood. *Journal of Adolescent Health*, 43(1), 23–29.
- Malow, B., Adkins, K. W., McGrew, S. G., Wang, L., Goldman, S. E., Fawkes, D., & Burnette, C. (2012). Melatonin for sleep in children with autism: A controlled clinical trial examining dose, tolerability and outcomes. *Journal of Autism and Developmental Disorders*, 42(8), 1729–1737. <https://doi.org/10.1007/s00431-011-1669-1>.
- McDougle, C. J. (2013). Sounding a wake-up call: Improving the lives of adults with autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(6), 566–568.
- Montes, G., Halterman, J. S., & Magyar, C. I. (2009). Access to and satisfaction with school and community health services for US children with ASD. *Pediatrics*, 124(Suppl 4), S407–S413. <https://doi.org/10.1542/peds2009-1255L>.
- Newacheck, P. W., & Kim, S. E. (2005). A national profile of health care utilization and expenditures for children with special health care needs. *Archives of Pediatrics and Adolescent Medicine*, 159(1), 10–17.
- Newschaffer, C. J., Croen, L. A., Daniels, J., et al. (2007). The epidemiology of autism spectrum disorders. *Annual Review of Public Health*, 28(1), 235–258.
- Oswald, D. P., Gilles, D. L., Cannady, M. S., Wenzel, D. B., Willis, J. H., & Bordurtha, J. N. (2013). Youth with special healthcare needs: Transition to adult health care services. *Maternal and Child Health Journal*, 17(10), 1744–1752.
- Patel, M. S., & O'Hare, K. (2010). Residency training in transition of youth with childhood-onset chronic disease. *Pediatrics*, 126(Suppl 3), S190–S193.
- Scarpinato, N., Bradley, J., Kurbjun, K., Bateman, X., Holtzer, B., & Ely, B. (2010). Caring for the child with an ASD in the acute care setting. *Journal for Specialists in Pediatric Nursing*, 15(3), 244–254.
- Smeltzer, S. C., Blunt, E., Marozsan, H., & Wetzel-Effinger, L. (2014). Inclusion of disability-related content in nurse practitioner curricula. *Journal of the American Association of Nurse Practitioners*, 27(4), 213–221.
- Smith, L. E., Hong, J., Seltzer, M. M., Greenberg, J. A., Almeida, D., & Bishop, S. L. (2010). Daily experiences among mothers of adolescents and adults with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(2), 167–178. <https://doi.org/10.1007/s10803-009-0844-y>.
- Souders, M. C. (2002). Caring for children and adolescents with autism who require challenging procedures. *Pediatric Nursing*, 28(6), 555–562.
- Souders, H. T. (2016). Preparing tomorrow's doctors to care for patients with autism spectrum disorder. *Intellectual and Developmental Disabilities*, 54(3), 202–216. <https://doi.org/10.1352/1934-9556-54.3.202>.
- Warfield, M. E., & Gulley, S. (2006). Unmet need and problems accessing specialty medical and related services among children with special health care needs. *Maternal and Child Health Journal*, 10(2), 201–216.
- Warfield, M. E., Crossman, M. K., Delahaye, J., Der Weerd, E., & Kuhlthau, K. A. (2015). Physician perspectives on providing primary medical care to adults with autism spectrum disorders (ASD). *Journal of Autism and Developmental Disorders*, 45(7), 2209–2217. <https://doi.org/10.1007/s1083-015-2386-9>.

Hearing

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Synonyms

[Audition](#)

Definition

Hearing is one of our five senses. The ability to hear is contingent upon the ability to perceive vibrations in air molecules that enter the auditory system through the outer ear. This energy is transmitted through the middle ear and inner ear, ending in the temporal lobe of the brain where it is ultimately perceived. The ability to hear is a critical component to the reception and comprehension of speech. Individuals with autism spectrum disorders can have impairment in their ability to hear, but more systematic research needs to be completed regarding hearing sensitivity in this population.

See Also

- ▶ [Auditory Acuity](#)
- ▶ [Auditory System](#)

References and Reading

- Justice, L. (2006). *Communication sciences and disorders: An introduction*. Columbus: Pearson.
- Rosenhall, U., Nordin, V., Sandstrom, M., Ahlsen, G., & Gillberg, C. (1999). Autism and hearing loss. *Journal of Autism and Developmental Disorders*, 29(5), 349–357.
- Smith, D. E. P., Miller, S. D., Stewart, M., Walter, T. L., & McConnell, J. V. (1988). Conductive hearing loss in autistic, learning-disabled, and normal children. *Journal of Autism and Developmental Disorders*, 18, 53–65.

Hearing Sensitivity

- ▶ [Auditory Acuity](#)

Hearing System

- ▶ [Auditory System](#)

Hearing Threshold

- ▶ [Auditory Acuity](#)

Heart Rate Variability (HRV)

- ▶ [Physiological Measures of Parental Stress](#)

Heart-Hand Syndrome

- ▶ [Timothy Syndrome](#)

Heller, Theodore

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Name and Degrees

Theodor Heller, Ph.D.

Landmark Clinical, Scientific, and Professional Contributions

Heller was a founding figure in special education. He also first described a disorder he termed

dementia infantilis, currently called childhood disintegrative disorder. In addition, he formed an institute for the treatment and education of children with special needs with international draw.

Short Biography

Theodor Heller was born June 9, 1869, in Vienna, Austria. His father, Simon Heller, was the director of an institute for the blind, and Heller spent much of his childhood at the institute. During school, he worked as a reader for blind people, including the philosopher Hitschmann, and through this became interested in philosophy. He went to University of Leipzig to study under Wilhelm Wundt, an important figure in modern psychology. Heller's area of concentration was the psychology of blindness, and his thesis, *Studien zur Blinden-Psychologie*, was submitted in 1894. He also explored a number of other topics including the psychology of acoustic and haptic perception. During this time, he attended lectures on medicine and the brain also and became very interested in the education of children with intellectual and developmental disabilities (special education). Shortly afterward, he started an institute for handicapped children together with his father and Richard Freiherr von Krafft-Ebing (a psychiatrist and author of *Psychopathia Sexualis*) at a property he had inherited at Grinzing in the Döbling district of Vienna. The institute started with two children as patients, but would ultimately serve 40 children at a time, many from outside of Austria. Many of the children came from wealthy families, but Heller used the proceeds to fund places for children from less privileged backgrounds. The institute included individualized treatment as well as social and academic education.

Heller was a pioneer of special education. He published works on the topic, including *Grundriß der Heilpädagogik* (*Groundwork for Special Education*) in 1904, *Pädagogische Therapie* (*Educational Therapy*) in 1914, and *Die Heilpädagogik in der Gegenwart und Zukunft* (*Special Education in the Present and Future*) in 1922. Heller also wrote about children who

underwent a sudden and catastrophic regression of their adaptive function, including sociability and language, coupled with the onset of stereotyped behaviors (all defining characteristics of autism) after a period of normal development. He termed this *dementia infantilis*, and its essential features are captured by the current diagnostic concept *childhood disintegrative disorder*, despite radical shifts in diagnostic frameworks and treatment approaches, marking the clarity of Heller's observation.

In his first publication on the topic, *Über Dementia Infantilis: Verblödungsprozess im Kindesalter* (*On Dementia Infantilis: Mental Regression in Childhood*) in 1908, Heller described six cases he had seen in Vienna, referred from as far away as Turkey (Heller 1908). The children had all been developing normally until their third or fourth year of life, and then had dramatic losses, ultimately ending up with little or no language, and strange stereotypes of behavior. One of Heller's case descriptions is excerpted below to illustrate the period of regression:

At the end of his third year of life he developed **states of anxiety**. He didn't want to stay in a dark room. He was **afraid** when he was left alone. He often cried without cause. **Later** his intelligence decreased rapidly. He wasn't interested in games he had previously enjoyed and no longer understood them. He made strange movements and held strange postures. He played with his saliva. His speech became worse and worse.

Although the term *autism* was used to describe the inward turning aspect of schizophrenia by Bleuler, a contemporary of Heller's, around the same time that Heller was writing about these cases, it was not until many years later that Kanner and Asperger would use the term to describe the combination of social disabilities and repetitive behaviors. However, Heller's vivid descriptions of *dementia infantilis* are without question also descriptions of autism. Heller led an extremely productive life. He founded organizations (Society for Special Education, Society for Pediatric Research, Austrian Society for Special Education), contributed significantly to other organizations (International Society for Special Education), and served as an editor (*Journal of*

Child Research). Heller gave his last public speech during March of 1938. Later that month, as a Jew, he was removed from his position as head of the institute by the Nazis. His temporary replacement was a butcher. In May, Heller attempted suicide. He died on December 12, 1938, as a result. The following year, his wife and daughter were taken to Riga and killed by the Nazis. His institute was disbanded in 1941.

See Also

- [Childhood Disintegrative Disorder](#)

References and Reading

- Heller, T. (1908). Über dementia infantilis: Verblödungsprozeß im kindesalter. *Zeitschrift für die Erforschung und Behandlung des Jugendlichen Schwachsinn*s, 2, 17–28.
http://de.wikipedia.org/wiki/Theodor_Heller
- Lotz, D. (2000). Theodor Heller (1869–1938). In M. Buchka, R. Grimm, & F. Klein (Eds.), *Lebensbilder bedeutender Heilpädagoginnen und Heilpädagogen im 20. Jahrhundert* (pp. 111–128). München/Basel: E. Reinhardt.
- Westphal, A., Schelinski, S., Volkmar, F., & Pelphey, K. (2013). Revisiting regression in autism: heller’s dementia infantilis. *Journal of Autism and Developmental Disorders*, 43, 265–271.

HELSNF1

- [CHD8](#)

Hemifacial Microsomia

- [Goldenhar Syndrome](#)

Hemispheric Dominance

- [Dominance, Cerebral](#)

Hemispheric Lateralization

- [Dominance, Cerebral](#)

Hemispheric Specialization

- [Dominance, Cerebral](#)

Hendrick Hudson Central School District v. Rowley (Provision of What Is an Appropriate Program)

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Definition

Bd. of Educ. of Hendrick Hudson Cent. Sch. Dist. v. Rowley

In General

In this case, the United States Supreme Court declared that the “free appropriate public education” (FAPE) requirement in the Education for All Handicapped Children Act (EAHCA) – the Individuals with Disabilities Education Act’s (IDEA) predecessor statute – required school districts merely to provide all disabled students with educational benefits rather than to maximize each disabled student’s potential. The plaintiffs, parents of a hearing-impaired student, brought suit against the defendant school district alleging that its refusal to provide their daughter with a sign language interpreter constituted a denial of FAPE. Noting the student’s successful completion of her grade level, the Court sided with the school district by finding the FAPE requirement satisfied regardless of whether she may have performed better with access to a sign language interpreter.

In reaching this conclusion, the Court found that the Act neither expressly defined FAPE, nor imposed upon the states any substantive standard prescribing the level of education to be accorded to disabled children. The Court thus prescribed a flexible two-part inquiry for examining educational programs, whereby courts should ask: (1) whether the state complied with the procedures set forth in the IDEA and (2) whether the student's individualized educational program (IEP) was reasonably calculated to enable the child to receive educational benefits. Pursuant to this decision, a disabled student's education must merely provide the student with some meaningful benefit rather than providing the best education possible. Thus, school districts must provide only a "basic floor of opportunity" to children with disabilities, even when it is arguable that students could perform better if granted additional services.

Implications for ASD Students

Autism spectrum disorders were added to the IDEA's list of disabilities in 1991. Since then, however, much debate surrounds the issue of defining FAPE for ASD students. The low threshold announced in *Rowley* made it difficult for parents to prove that their ASD student's IEP is inadequate. The preferred early intervention programs for ASD students, such as applied behavioral analysis (ABA), are very costly and require trained personnel. District programs are often-times school-based and do not provide the student with the one-on-one attention that some experts believe is necessary for ASD treatment. Some critics argue that the "equal floor of opportunity" requirement is too vague, particularly in light of the broad spectrum of autism disorders and the wide variety of treatment options for ASD.

Litigation Strategies

It is important to note that there is much room for disagreement with respect to defining appropriate educational programs for ASD students. These disagreements pose problems for parents and professionals attempting to prove that an ASD student's IEP is inadequate under the IDEA. In addition, parents must be aware before challenging their child's IEP that IDEA litigation is expensive. Although victorious parents may sometimes

collect attorneys' fees, they are unlikely to recover such fees if they reach a settlement agreement before trial. Since 2007, though, parents have a recognized right to bring suit under the IDEA directly, without legal representation, which helps reduce the costs associated with such litigation.

See Also

- ▶ [Dispute Resolution Procedures](#)
- ▶ [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- Bates, M. W. (1994). Free appropriate public education under the individuals with disabilities education act: Requirements, issues, and suggestions. *Brigham Young University Education and Law Journal*, 1, 215–222.
- Bd. of Ed. of Hendrick Hudson Cent. Sch. Dist. v. Rowley, 458 U.S. 176 (1982).
- Breslin, M. A. (2009). No Child left behind and the inherent conflict with the individuals with disabilities education act: Leaving special education students further behind. *Albany Government Law Review*, 2, 653–676.
- Caruso, D. (2010). Autism in the U.S.: Social movement and legal change. *American Journal of Law & Medicine*, 36, 483–539.
- Hinson, C. (2009). A supreme paradox: Autism spectrum disorder and Rowley misapplication of judicial relic to an unprecedented social epidemic. *Florida A & M University Law Review*, 5, 87–105.
- Holland, C. D. (2010). Autism, insurance, and the IDEA: Providing a comprehensive legal framework. *Cornell Law Review*, 95, 1253–1282.
- Individuals with Disabilities Education Act (IDEA). 20 U.S.C. §§ 1400, *et seq.* (2012).
- Kemper, K. A. (2006). Construction and application of individuals with disabilities act. *American Law Reports Federal 2d*, 13, 321.

Heritage Language Use for Intervention in Autism

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Definition

Heritage languages are those languages that are specific to an ethnic or racial group. For instance,

the heritage language of a Chinese person might be Mandarin. An individual's heritage language may be different from the majority language (i.e., the language that is spoken by most people in a region), like in the case of a Chinese person living in the United States. For individuals with autism spectrum disorder (ASD) whose families might speak a heritage language that is different from the majority language, heritage language usage during intervention might look like (a) delivering the entire intervention in the heritage language, (b) providing verbalizations in the heritage language, or (c) teaching using heritage and majority languages.

Historical Background

Because a core characteristic of ASD is the impairment of language abilities, some parents and practitioners believed that exposure to more than one language could exacerbate the already impaired language development of children with ASD (Hampton et al. 2017; Kay-Raining Bird et al. 2012; Kremer-Sadlik 2005; Ohashi et al. 2012). As Lim et al. (2018) pointed out, the arguments against dual-language exposure mirror those that surrounded the use of augmentative and alternative communication (AAC), which is now widely used to help children with ASD communicate. Studies that have compared the language abilities of bilingual and monolingual children with ASD have found no detrimental effects of bilingual exposure (Dai et al. 2018; Gonzalez-Barrero and Nadig 2017; Lund et al. 2017; Zhou et al. 2017). In particular, Dai et al. (2018) compared the expressive and receptive language abilities of bilingually exposed to monolingually exposed children with ASD before they received any intervention. Results demonstrated that there were no detrimental effects of bilingual exposure, even when cognitive functioning and socioeconomic status were controlled for. Additionally, some studies even suggest that bilingual children with ASD might have an advantage over monolingual children with ASD in terms of vocabulary size (Gonzalez-Barrero and Nadig 2017) and early social communication skills like gesturing (Valicenti-McDermott et al. 2013; Zhou

et al. 2017). Overall, it would seem that learning more than one language does not negatively impact the language development of children with ASD.

Current Knowledge

Because research regarding the effects of dual-language exposure in children with ASD is relatively new, few studies have investigated the effects heritage language use in interventions for individuals with ASD. Seung et al. (2006) reported on a case study whereby a Korean-American preschooler with ASD received speech-language intervention entirely in Korean, then bilingually in Korean and English, and finally entirely in English. Results indicated that conducting speech-language intervention in Korean, the participant's heritage language, helped to facilitate the participant's acquisition of English, the majority language. Specifically, authors noted that the participant acquired new English words as his Korean vocabulary increased.

In terms of providing verbalizations in heritage languages, preference assessments suggest that children with ASD may prefer listening to verbalizations such as praise in their primary home language, which may be the heritage or majority language depending on the household (Aguilar et al. 2016, 2017). Bilingually exposed children with ASD's language preferences may also depend on task difficulty levels such that they prefer their primary home language as tasks become more challenging (Aguilar et al. 2016). Additionally, the language that verbalizations are delivered in may also affect children with ASD's engagement in play behaviors (Lim and Charlop 2018). Specifically, children with ASD may demonstrate more play behaviors when play partners deliver verbalizations like instructions, comments, and praise in their heritage languages.

A recent meta-analysis of studies comparing the effects of teaching children with neurodevelopmental disorders in the heritage language versus the majority language identified five studies that included participants with ASD (Lim et al. 2019). One study did not disaggregate the data of

participants with ASD from those with intellectual disability (Durán and Heiry 1986). Thus, the specific effects of providing verbal prompts in English versus Spanish on the vocational skills of adults with ASD are unclear. Of the remaining four studies, one study found that conducting discrete trial training in the participant's heritage language, Spanish, led to increased accuracy and decreased instances of challenging behaviors as compared to the majority language (Lang et al. 2011). The other three studies found no effects of language of instruction in the context of functional communication training (Dalmau et al. 2011), tact training (León 2016), and pivotal response training (Vaughn 2014). Functional communication training in Spanish and English were equally effective at decreasing challenging behaviors and increasing task completion and requests for reinforcement (Dalmau et al. 2011). Conducting tact training in English and bilingually (i.e., English and Spanish) had similar effects on correct responding (León 2016). Pivotal response training in Spanish and English had comparable effects on participants' conversation skills (Vaughn 2014).

In sum, the current literature indicates that (a) delivering speech-language intervention in the heritage language of a child with ASD might facilitate the acquisition of the majority language, (b) children with ASD from bilingual households might demonstrate preferences for their primary home language and engage in more play behaviors when verbalizations are provided in their heritage languages, and (c) heritage and majority language-based treatments may be equally effective.

Future Directions

In general, more studies investigating the effects of using heritage languages during interventions for children with ASD are needed. With preference assessments indicating that bilingual children with ASD might prefer their primary home languages, researchers may want to pay special attention to reporting the amount of exposure that participants have with heritage and majority languages within their homes. A challenge for

those conducting research in this area may be finding interventionists who are fluent in participants' heritage languages. Because heritage languages are most often spoken within the context of family, researchers may want to look at training parents or siblings of children with ASD to deliver interventions in their heritage languages. Nonetheless, if the family member is not fluent in the majority language, then the same challenge of finding a trainer who speaks the heritage language will be present. Thus, another area for future research may be investigating how training can be conducted in the presence of language barriers.

References and Reading

- Aguilar, J. M., White, P. J., Fragale, C., & Chan, J. M. (2016). Preference for language of instruction of an English language learner with autism. *Developmental Neurorehabilitation*, 19, 207–210. <https://doi.org/10.3109/17518423.2015.1044133>.
- Aguilar, J. M., Chan, J. M., White, P. J., & Fragale, C. (2017). Assessment of the language preferences of five children with autism from Spanish-speaking homes. *Journal of Behavioral Education*, 1–14. <https://doi.org/10.1007/s10864-017-9280-9>.
- Dai, Y. G., Burke, J. D., Naigles, L., Eigsti, I.-M., & Fein, D. A. (2018). Language abilities in monolingual- and bilingual-exposed children with autism or other developmental disorders. *Research in Autism Spectrum Disorders*, 55, 38–49. <https://doi.org/10.1016/j.rasd.2018.08.001>.
- Dalmau, Y. C. P., Wacker, D. P., Harding, J. W., Berg, W. K., Schieltz, K. M., Lee, J. F., . . . Kramer, A. R. (2011). A preliminary evaluation of functional communication training effectiveness and language preference when Spanish and English are manipulated. *Journal of Behavioral Education*, 20, 233–251. <https://doi.org/10.1007/s10864-011-9131-z>.
- Durán, E., & Heiry, T. J. (1986). Comparison of Spanish only, Spanish and English, and English only cues with handicapped students. *Reading Improvement*, 23, 138–141.
- Gonzalez-Barrero, A. M., & Nadig, A. (2017). Verbal fluency in bilingual children with autism spectrum disorders. *Linguistic Approaches to Bilingualism*, 7, 460–475. <https://doi.org/10.1075/lab.15023.gon>.
- Hampton, S., Rabagliati, H., Sorace, A., & Fletcher-Watson, S. (2017). Autism and bilingualism: A qualitative interview study of parents' perspectives and experiences. *Journal of Speech, Language, and Hearing Research*, 60, 435–446. https://doi.org/10.1044/2016_JSLHR-L-15-0348.
- Kay-Raining Bird, E., Lamond, E., & Holden, J. (2012). Survey of bilingualism in autism spectrum disorders.

- International Journal of Language & Communication Disorders*, 47, 52–64. <https://doi.org/10.1111/j.1460-6984.2011.00071.x>.
- Kremer-Sadlik, T. (2005). To be or not to be bilingual: Autistic children from multilingual families. In J. Cohen, K. McAlister, K. Rolstad, & J. MacSwan (Eds.), *Proceedings of the 4th international symposium on bilingualism* (pp. 1225–1234). Somerville: Cascadilla Press.
- Lang, R., Rispoli, M., Sigafoos, J., Lancioni, G., Andrews, A., & Ortega, L. (2011). Effects of language of instruction on response accuracy and challenging behavior in a child with autism. *Journal of Behavioral Education*, 20, 252–259. <https://doi.org/10.1007/s10864-011-9130-0>.
- León, A. L. (2016). *Evaluating the effects of tact training in two languages on the acquisition of tacts and untrained listener responses* (Master's thesis). Available from ProQuest Dissertations & Theses Global. (Order No. 10289910).
- Lim, N., & Charlop, M. H. (2018). Effects of English versus heritage language on play in bilingually exposed children with autism spectrum disorder. *Behavioral Interventions*, 33, 339–351. <https://doi.org/10.1002/bin.1644>.
- Lim, N., O'Reilly, M. F., Sigafoos, J., & Lancioni, G. E. (2018). Understanding the linguistic needs of diverse individuals with autism spectrum disorder: Some comments on the research literature and suggestions for clinicians. *Journal of Autism and Developmental Disorders*, 48, 2890–2895. <https://doi.org/10.1007/s10803-018-3532-y>.
- Lim, N., O'Reilly, M. F., Sigafoos, J., Ledbetter-Cho, K., & Lancioni, G. E. (2019). Should heritage languages be incorporated into interventions for bilingual individuals with neurodevelopmental disorders? A systematic review. *Journal of Autism and Developmental Disorders*, 49, 887–912. <https://doi.org/10.1007/s10803-018-3790-8>.
- Lund, E. M., Kohlmeier, T. L., & Durán, L. K. (2017). Comparative language development in bilingual and monolingual children with autism spectrum disorder: A systematic review. *Journal of Early Intervention*, 39, 106–124. <https://doi.org/10.1177/1053815117690871>.
- Ohashi, J. K., Mirenda, P., Marinova-Todd, S., Hambly, C., Fombonne, E., Szatmari, P., . . . Thompson, A. (2012). Comparing early language development in monolingual- and bilingual- exposed young children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6, 890–897. <https://doi.org/10.1016/j.rasd.2011.12.002>.
- Seung, H., Siddiqi, S., & Elder, J. H. (2006). Intervention outcomes of a bilingual child with autism. *Journal of Medical Speech-Language Pathology*, 14, 53–63.
- Valicenti-McDermott, M., Tarshis, N., Schouls, M., Galdston, M., Hottinger, K., Seijo, R., . . . Shinnar, S. (2013). Language differences between monolingual English and bilingual English-Spanish young children with autism spectrum disorders. *Journal of Child Neurology*, 28, 945–948. <https://doi.org/10.1177/0883073812453204>.
- Vaughn, J. L. (2014). *An evaluation of English versus Spanish language choice during conversation training intervention for children with autism* (Doctoral dissertation). Available from ProQuest Dissertations & Theses Global. (Order No. 3612044).
- Zhou, V., Munson, J. A., Greenson, J., Hou, Y., Rogers, S., & Estes, A. M. (2017). An exploratory longitudinal study of social and language outcomes in children with autism in bilingual home environments. *Autism: The International Journal Of Research And Practice*, 23, 394–404. <https://doi.org/10.1177/1362361317743251>.

HFDT

► Human Figure Drawing Tests

Higher Education Opportunity Act of 2008 (HEOA)

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Definition

In 2008, Congress reauthorized the Higher Education Act as the Higher Education Opportunity Act (HEOA). This reauthorization brought about major changes to the eligibility requirements under Title IV which is the title that governs the Federal Student Aid (FSA). Now students with intellectual disabilities (ID) and developmental disabilities (DD) including some students on the autism spectrum may have an opportunity to attend postsecondary education as a result of these amendments and receive some limited forms of Federal Student Aid. This piece of legislation creates an avenue through which more students with autism spectrum disorders and other developmental disabilities can attend college.

The Higher Education Opportunity Act (HEOA) allows Institutions of Higher Education

(IHE) that already are approved to distribute Federal Student Aid under Title IV to apply to the US Department of Education (DOE) for approval of a comprehensive transition and post-secondary program (CTP). Colleges and universities would need to design a curriculum and advising structure specifically geared toward meeting the needs of students with intellectual disabilities. At least 51% of the students with ID's time would need to be spent with non-disabled peers. If the US DOE approved a college's CTP, then the student with an intellectual disability would be limited to only some forms of Federal Student Aid, namely, Federal Pell Grants, Federal Supplemental Educational Opportunity Grants (FSEOG), and Federal Work-Study monies. Although the student with ID enrolled in a US DOE-approved CTP does not have to pay back these grants, it does limit their ability to pay the high costs of post-secondary education. The bulk of Federal Student Aid is in the form of guaranteed student loans that are both subsidized and unsubsidized by the government.

An eligible student with an intellectual disability (ID) is defined in Section 760 of the HEOA (with slight modifications) and includes a student (A) with mental retardation or significant cognitive impairment and (B) who is/was eligible for FAPE under IDEA including students who were private and/or homeschooled. The student must be enrolled in an approved CTP and must meet all of the general student eligibility requirements under Section 668.32 **except:**

- Does *not* have to be enrolled for the purpose of obtaining a degree or certificate
- Is *not* required to have a high school diploma or have passed an ability-to-benefit test
- Must maintain satisfactory academic progress under school's policy for students in the CTP (Bergeron et al. 2010).

The student must also have a documentation demonstrating that he or she has an intellectual disability or significant cognitive impairment which can also include individuals with autism spectrum disorders (VanBergeijk and Cavanagh 2012).

An eligible CTP must be offered by a college that already has an established financial aid program for its general student body. It also must be specifically designed to support students with intellectual disabilities (ID) and include an advising and curriculum structure. The CTP must require students with ID to participate in courses and activities with students without disabilities. These activities can include vocational internships in addition to academic coursework (VanBergeijk and Cavanagh 2012).

Students with ID and/or significant cognitive impairments including those students on the autism spectrum can complete the Free Application for Federal Student Aid (FAFSA) and receive aid provided they are intending to enroll in a US DOE-approved CTP. The FAFSA form helps the college determine how much aid a student enrolled in a CTP is entitled to by calculating the Expected Family Contribution (EFC) (Finkel et al. 2010).

Historical Background

The Higher Education Opportunity Act can trace its roots to the Higher Education Act of 1965 (P.L. 89–329). This act authorized most federal student financial aid programs, including the Federal Supplemental Educational Opportunity Grant program and the guaranteed student loan program (FinAid 2016) (known as Title IV). At the time President Lyndon B. Johnson signed the act into law, the proportion of grants to loans is the mirror opposite of what they are today. In the 1960s, about 2/3 of the Federal Student Aid came in the form of grants. Now that proportion is inverted with 2/3 of today's Federal Student Aid being given to students in the form of loans.

Prior to the passage of HEOA, only students who were enrolled full time in a degree-bearing program were eligible for Federal Student Aid. Full-time students were able to complete the Free Application for Federal Student Aid (FAFSA). The student was eligible for both subsidized and unsubsidized guaranteed student loans as well as grants including Federal Pell Grants, Federal Supplemental Educational Opportunity Grants (FSEOG), and Federal Work-Study program funds. The Congress was amending the

act in the midst of the worst economic recession since the Great Depression. A driving force behind the recession was the provision of risky mortgage and business loans. Consequently, the Congress decided not to allow students with ID to be eligible. The thought was that the students with IDs' debit to income ratio would be too high for them to be able to pay off the debt. Therefore, this population of students was perceived as being yet another risky loan prospect. Interestingly, Wehman (2001) found that students with disabilities who did complete a college degree were employed at a rate that was not statistically different from their nondisabled peers. Many individuals on the autism spectrum have the capacity to complete college level work. It is their impairment in executive functioning, independent living, and social skills that make it difficult for them to complete a degree – not their intellectual ability. This blanket policy of excluding this population needs further examination.

Current Knowledge

Although the HEOA was passed in 2008, it was not until 2011 that the US DOE began taking applications from Institutions of Higher Education (IHE) to have their comprehensive transition and postsecondary programs (CTP) approved to distribute Federal Student Aid. Despite the fact that there are over 7600 IHE that are authorized to disperse Federal Student Aid under Title IV, only about colleges have a US DOE-approved CTP. This represents that less than of colleges and universities are serving this population through the CTP structure.

The reasons for the slow adoption of the CTP model by our nation's Institutions of Higher Education are unclear. The law was enacted with little fanfare considering this provides students with ID an entrée into postsecondary education. The actual implementation of the law took a number of years and appeared to be not well coordinated. There were high administrative costs incurred by the universities to undertake the creation of a CTP with little economic incentive to commit the organization's resources. Perhaps, if the legislation had also included the provision of student loans to students with ID enrolled in a CTP, then more

colleges of universities may have created these programs for students with ID.

What we know from current research is that postsecondary transition programs do make a difference in the employment outcomes of students with intellectual disabilities. Moore and Schelling (2015) found that nine out of ten students who graduated from a postsecondary program were employed within 2 years of their study. This is in contrast to the finding from the National Longitudinal Transition Study-2 that only about half of the students with intellectual disabilities were employed (as cited in Moore and Schelling 2015). Wehman et al. (2013) used postsecondary vocational training as an intervention in a randomized clinical trial. His team's finding was that 87.5% of the students on the autism spectrum who participated in the postsecondary vocational training were employed at the end of the study, whereas the control group, which consisted of students working with their high school and their state office of Vocational Rehabilitation Services, had an employment rate of 6.5%.

Future Directions

In order for the CTP model to gain traction, three things need to occur. First, research into the efficacy of this model in general needs to be conducted. We need to examine not only if these models improve the employment outcomes of individuals with ID/DD and autism but also whether or not these individuals are making a living wage, are able to live independently, and are engaged in their communities with meaningful connections such as friendships and affiliations with organizations including civic, recreational, and religious organizations. More sophisticated research should examine the structural differences in the CTP that promote positive outcomes with this population. Given the broad nature of the requirements of a CTP outlined by HEOA, there will be a great deal of variance in the programs' offerings and structure. Some CTP will have a residential component. Others will simply stand as a day program where the students commute daily to campus. The academic offerings will also vary widely with some colleges allowing

students with ID to simply audit credit-bearing courses. Others will encourage students to take coursework for credit. Another possible structure is that the CTP offer vocational training and pre-employment skills readiness courses.

With the passage of the Workforce Innovation and Opportunity Act (WIOA), colleges and universities need to learn that a substantial amount of government funding is available for programs and services directed to transitional age youth with disabilities. Institutions of Higher Education are eligible to be providers of preemployment services. The law not only specifically designated IHEs as potential partners but also mentions CTP as being potentially eligible for funding as well. Only when universities realize this potential funding stream is available will the CTP model gain traction.

The final change that needs to occur before the CTP model will become more widespread is amending the HEOA to allow students with ID/DD to obtain student loans. The rationale for not allowing this population of students to receive the federal student loans is that their ability to repay those loans is perceived as being low. The unintended consequence of this feature of the HEOA is that a number of families are turning to private lenders for continuing education loans in order to finance their sons' and daughters' attendance at a CTP. These loans are much more expensive for families to maintain and are more likely to result in a default in the loan than the government-provided subsidized and unsubsidized loans at lower rates of interest.

See Also

- [Free Appropriate Public Education](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Workforce Innovations Opportunity Act](#)

References and Reading

Bergeron, D., Murray, B., & Shanley, J. L. (2010). *Comprehensive transition and postsecondary (CTP) program: The process and lessons learned*. Washington, DC: U.S. Department of Education: State of the Art Conference.

FinAid (2016). The history of Federal Student Aid. Retrieved from <http://www.finaid.org/educators/history.phtml>. Accessed 10 Oct 2016.

Finkel, J., Anderson, H., & Shanley, J. L. (2010). *Eligibility of students with intellectual disabilities to receive Federal Student Aid: The FAFSA and more!* Washington, DC: U.S. Department of Education: State of the Art Conference.

Higher Education Opportunities Act of 2008. (P.L. 110–315).

Moore, E. J., & Schelling, A. (2015). Postsecondary inclusion for individuals with intellectual disabilities and its effects on employment. *Journal of Intellectual Disabilities*, 19(2), 130–148.

The Workforce Innovation and Opportunity Act (WIOA) (H.R. 803).

VanBergeijk, E.O., & Cavanagh, P.K. (2012, May/June). Federal Student Aid for students with intellectual disabilities: A well-kept secret? *Parenting Special Needs Magazine*, pp. 64–65. <http://www.mydigitalpublication.com/publication/?i=111307&l=&m=&p=8&id=5779>

Wehman, P. H. (2001). *Life beyond the classroom: Transition strategies for young people with disabilities* (3rd ed.). Baltimore: Paul H. Brookes Publishing.

Wehman, P. H., et al. (2013). Competitive employment for youth with autism spectrum disorders: Early results from a randomized clinical trial. *Journal of Autism and Developmental Disorders*, 44, 487–500. <https://doi.org/10.1007/s10803-013-1892-x>

High-Functioning Autism (HFA)

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Definition

High-functioning autism is a term used to refer to a subset of individuals on the autism spectrum who have cognitive and/or linguistic abilities

that are in the average to above-average range for their age. It is common for the acronym “HFA” to be used to describe individuals in this range of functioning.

Historical Background

In the first characterizations of the disorder, both Kanner and Asperger noted average to above-average intellectual capabilities in the individuals they observed, despite their significant social deficits. Kanner argued explicitly in several papers that social deficits were separable from intellectual disability and that most of the children he encountered were of average to above average intelligence. Interestingly, Asperger used the “special gifts” of the children he observed to advocate for their protection from the Nazi eugenics movement (Feinstein 2010).

Historically, testing IQ in children on the autism spectrum was difficult (Alpern 1967; Alpern and Kimberlin 1970). Although many of the children had discernible strengths (and sometimes savant skills) in areas of cognitive function, language and social deficits often interfered with standardized administration of tests. Moreover, even individuals on the autism spectrum who did not exhibit specific savant skills often showed pronounced relative strengths and weaknesses from subtest to subtest that made generating an overall IQ score difficult. In one of the earliest instances of the use of “high-functioning” as a term in reference to individuals with ASD in a research study, DeMeyer and colleagues suggested that children who currently were defined as “high-functioning psychotic children” should be reclassified as children with “high-functioning infantile autism” (DeMyer et al. 1971). They used the term “high-autistic children” to refer to a group of children with IQs within two standard deviations of the mean or higher.

The term “high-functioning autism” was used infrequently until the late 1980s, when Asperger syndrome grew in popularity as a distinct diagnosis. At that time, “High-functioning autism” was used to describe the subgroup of individuals with autism who had average or above average intelligence or language abilities to compare them to

individuals with Asperger syndrome (e.g., DeLong and Dwyer 1988). The use of the acronym “HFA” also began at around this time (e.g., Szatmari et al. 1989).

Recently, the use of the term “high-functioning” has become a common qualifier in research studies and for clinical diagnoses. In research, its use is common in part because a significant portion of published research is conducted on this subset of individuals with autism spectrum disorder (ASD). Individuals with average to above average IQs/language abilities are overrepresented in research because of the behavioral challenges in conducting research on individuals with ASD and intellectual disability.

The term HFA is sometimes used in clinical evaluations, even though it is not currently an established diagnostic category. In DSM-5 criteria for ASD, there is an item that categorizes “Severity Level,” but these categories do not include the term HFA. The categories are “Level 1 – Requiring Support,” “Level 2 – Requiring Substantial Support,” and “Level 3 – Requiring Very Substantial Support.” Each diagnostic criterion receives its own Severity Level score. It is likely that most individuals with an HFA label would fall under “Level 1,” although it is possible that somebody who is considered high-functioning could still require substantial support in one or more areas. Deficits at Level 1 are described as causing noticeable impairments without supports in place, whereas impairments in the higher levels (2 and 3) are noticeable even with appropriate supports in place.

Current Knowledge

“High-functioning” is a qualifier that is commonly used to describe a subgroup of individuals on the autism spectrum, but currently there is no recognized definition or criteria for inclusion in this group, either clinically or in research. The term high-functioning has been used: (a) to differentiate this subgroup of individuals on the autism spectrum from a group with intellectual disability (i.e., “low-functioning”), (b) to differentiate it from a group with an Asperger syndrome

diagnosis, and (c) to compare it a typically developing comparison group or to specify an individual's level of cognitive and/or language ability in the context of a research study.

In research, inclusion criteria can vary from study to study. Minimally, group membership is defined as having either an intelligence test or language test standard score above 70 (or within two standard deviations of the mean or above, if the test uses a different statistic). Some studies use more stringent criteria, such as requiring both language and cognitive standard scores to be above 70. Other studies will require one or both language/cognitive scores to be above 80, which would exclude individuals who have scores in the "borderline" range of intellectual functioning (standard 70–80). Still other studies will use criteria that exclude individuals with standard scores below 85 (i.e., one standard deviation below the mean) on standardized language and/or cognitive tests. These stringent criteria include only individuals that are at or above the typical range of functioning, as defined by standardized testing.

Even though the term "functioning" is included in the label, adaptive functioning level is generally not considered when using the terminology. "Functioning" is generally used to refer to cognitive and/or linguistic functioning. This is not consistent with the conceptualization of intellectual disability; the intellectual disability definition includes both cognitive and adaptive functioning in the criteria.

High-Functioning Autism Versus Asperger Syndrome

The increased frequency of the use of the term HFA has been due in part to the addition of Asperger syndrome as a diagnostic category. In the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV), individuals with Asperger's Disorder meet the criteria for social impairment and restricted/repetitive behavior necessary for a diagnosis of Autistic Disorder but are not required to meet requirements for impairments in communication and do not show

delays in early language milestones. In addition, individuals with an Asperger syndrome diagnosis must have shown no delays in cognitive development. It was known, however, that a subgroup of individuals with the autistic disorder diagnosis also showed no delays in cognitive development, which necessitated comparisons between the two groups to understand categorical differences.

There has been a considerable amount of research comparing individuals with the diagnosis of Asperger syndrome to individuals with HFA in order to determine if there are categorical differences between the autism and Asperger syndrome diagnoses. Some of the factors that have been proposed as differences between individuals with Asperger syndrome and HFA include (but are not limited to) presence/absence of delays in reaching language developmental milestones, motor skills/clumsiness, types of restricted/repetitive behavior displayed, and speaking style (including prosody), among other characteristics. Research has shown that DSM-IV criteria do not yield meaningful and categorical differences between Asperger syndrome and HFA. As a result, Asperger syndrome was removed from the newest version of the DSM, although this change generated considerable controversy. One argument against the change is that the problem in differentiating the groups might be an artifact of imprecise diagnoses, rather than the absence of categorical differences.

It is likely, however, that, even with the elimination of Asperger syndrome as a diagnosis in DSM-5, the use of the "high-functioning" qualifier (e.g., "high-functioning ASD") will continue. In the past several years, research studies have begun to refer to children with Asperger syndrome and HFA together as a group of individuals with "high-functioning ASD."

Future Directions

For clarity, it will be important to set clear criteria for what constitutes high-functioning autism, for both research and clinical purposes. To date, it has been difficult to make meaningful comparisons across research studies because of the wide

variety of ways group membership has been defined in research studies. For research studies, criteria should include *both* language level and cognitive level when defining a group as “high-functioning.” Moreover, groups defined as “high-functioning” should strongly consider excluding individuals with cognitive or language abilities below one standard deviation from the mean and above the cutoffs for intellectual disability or language impairment (i.e., individuals with IQ and Language standard scores between 70 and 85). These more stringent criteria are more consistent with the idea that the group is “high-functioning.” Moreover, these criteria create a more homogeneous group that is more easily matched with a typically developing comparison group, both on mean and standard deviation of scores.

Second, it will be important to consider adaptive functioning, in addition to cognitive and language level, when defining using the term “high-functioning.” The use of adaptive functioning in the high-functioning criteria will provide a definition that is more consistent with the definition of Intellectual Disability, which includes both intellectual and adaptive functioning. Moreover, the inclusion of adaptive functioning in the definition is a more valid representation of level of functioning, because individuals with ASD can have significant discrepancies between cognitive and adaptive functioning, and language/IQ scores may not be representative of level of functioning for some individuals.

In clinical settings, clear criteria would be important, but only if it is determined that there are clinically relevant benefits of using a “high-functioning” qualifier. It is important to investigate whether there are specific intervention recommendations that can be tailored to individuals with this qualifier.

Finally, it should also be investigated whether the addition of the “high-functioning” qualifier lessens the stigma of the diagnosis, and if the qualifier changes the way that parents, peers, and/or professionals interact with the child or the family. Research on other disabilities has shown that adding levels of ability in a label increases the stigma associated with individuals on the “lower” end of ability. It is possible that the high-

functioning qualifier reinforces stigma associated with portions of the population who are not considered high-functioning.

See Also

- Adaptive Behavior
- Asperger Syndrome
- DSM-IV
- Intelligence Norms
- Intelligence Quotient
- Language Tests
- Pervasive Developmental Disorder Not Otherwise Specified
- Psychological Assessment
- Spectrum/Continuum of Autism

References and Reading

- Alpern, G. D. (1967). Measurement of “untestable” autistic children. *Journal of Abnormal Psychology*, 72(6), 478–486.
- Alpern, G. D., & Kimberlin, C. C. (1970). Short intelligence test ranging from infancy levels through childhood levels for use with the retarded. *American Journal of Mental Deficiency*, 75, 65–71.
- Asperger, H. (1944). Die ‘autistischen psychopathen’ im kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Baron-Cohen, S. (2009). The short life of a diagnosis. *New York Times*, p. A35.
- DeLong, G. R., & Dwyer, J. T. (1988). Correlation of family history with specific autistic subgroups: Asperger syndrome and bipolar affective disease. *Journal of Autism and Developmental Disorders*, 18(4), 593–600.
- DeMyer, M. K., Churchill, D. W., Pontius, W., & Gilkey, K. M. (1971). A comparison of five diagnostic systems for childhood schizophrenia and infantile autism. *Journal of Autism and Childhood Schizophrenia*, 1, 175–189.
- DeMyer, M. K., Barton, S., Alpern, G. D., Kimberlin, C., Allen, J., Yang, E., et al. (1974). The measured intelligence of autistic children. *Journal of Autism and Developmental Disorders*, 4(1), 42–60.
- Diehl, J. J., Wolf, J., Herlihy, L., & Moller, A. C. (2011). Seeing red: Are colors a window into implicit societal conceptions about the autism spectrum? *Disability Studies Quarterly*, 31(3). <http://dsq-sds.org/article/view/1676/1595>.
- Feinstein, A. (2010). *A history of autism: Conversations with pioneers*. Hoboken: Wiley-Blackwell.

- Frith, U. (1991). *Autism and Asperger syndrome*. Cambridge: Cambridge University Press.
- Ghaziuddin, M., & Gerstein, L. (1996). Pedantic speaking style differentiates Asperger syndrome from high-functioning autism. *Journal of Autism and Developmental Disorders*, 26, 585–595.
- Howlin, P., & Goode, S. (1998). Outcome in adult life for people with autism and Asperger's syndrome. In F. R. Volkmar (Ed.), *Autism and pervasive developmental disorders* (pp. 209–241). New York: Cambridge University Press.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Miller, J. N., & Ozonoff, S. (1997). Did Asperger's cases have Asperger disorder? A research note. *Journal of Child Psychology and Psychiatry*, 38, 247–251.
- Miller, J. N., & Ozonoff, S. (2000). The external validity of Asperger disorder: Lack of evidence from the domain of neuropsychology. *Journal of Abnormal Psychology*, 109, 227–238.
- Szatmari, P. (2000). Perspectives on the classification of Asperger syndrome. In A. Klin & F. R. Volkmar (Eds.), *Asperger syndrome* (pp. 403–417). New York: The Guilford Press.
- Szatmari, P., Bartolucci, G., & Bremner, R. (1989). Asperger's syndrome and autism: Comparison of early history and outcome. *Developmental Medicine and Child Neurology*, 31(6), 709–720.
- Volkmar, F. R., Lord, C., Bailey, A., Schultz, R. T., & Klin, A. (2004). Autism and pervasive developmental disorders. *Journal of Child Psychology and Psychiatry*, 45, 135–170.
- Wing, L. (1981). Asperger's syndrome – A clinical account. *Psychological Medicine*, 11, 115–129.

High-Functioning Autism Diagnostic Interview

► Asperger Syndrome Diagnostic Interview

High-Probability Requests

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Definition

High-probability request sequence is an intervention used to increase compliance. It is a

nonaversive intervention commonly used to treat noncompliance by reducing escape-motivated behavior. It is also associated with and has been historically referred to as interspersed requests, pretask requests, and behavioral momentum. It is considered an antecedent intervention.

The basic procedure involves the teacher presenting three to five “high-probability requests,” which are easy and quick responses already mastered by the child and highly likely that the child will emit. The teacher presents these requests quickly, one after the other, providing brief verbal praise after each one. Immediately after receiving praise for the last of these high-probability requests, the teacher immediately presents the request to which the child has historically not complied. Reinforcement can also be added to the high-probability sequence to effectively increase compliance with low-p instructions (Wilder et al. 2015).

The use of the high-p sequence often prevents the noncompliance. Responding is facilitated (and momentum is built) through the high-p requests. Theoretically, the effect of the high-p sequence stems from the abative effects of an abolishing operation (AO). It reduces the value of reinforcement for noncompliance to the low-probability requests. The value of escape from demands is lowered. In application, behavior analysts must ensure that the high-p response request intervention is not done after the occurrence of a problem behavior. Such use could inadvertently strengthen escape-motivated noncompliance. In addition, attention must be paid to the quality of reinforcement delivered for compliance, especially if behaviors are severe (e.g., Mace and Belfiore 1990). Finally, the number of high-probability requests can be gradually reduced as compliance with low-probability requests improves.

See Also

- [Behavioral Momentum](#)
- [Establishing Operations](#)

References and Reading

- Mace, F. C., & Belfiore, P. (1990). Behavioral momentum in the treatment of escape-motivated stereotypy. *Journal of Applied Behavior Analysis*, 23, 507–514.
- Wilder, D. A., Majdalany, L., Sturkie, L., & Smeltz, L. (2015). Further evaluation of the high-probability instructional sequence with and without programmed reinforcement. *Journal of Applied Behavior Analysis*, 48(3), 511–522.

High-Throughput Sequencing

► Next-Generation Sequencing

Hippocampus

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Definition

The hippocampus is a bilateral incurved brain structure, a coiled elongated ridge located beneath the neocortex in the basal medial surface of the temporal lobes. Comprising a major component of the limbic system, the hippocampus is phylogenetically considered an ancient brain structure. It was first coined hippocampus by the sixteenth-century Italian anatomist Julius Caesar Aranzi who likened its shape to that of a seahorse (in Greek hippo = horse, kampe = curve). The hippocampus is mostly implicated in processes of memory formation and consolidation as it is critical for the formation of new factual and autobiographical memories. Hippocampal damage can result in anterograde amnesia: the loss of ability to form new memories. The hippocampus is also known for its involvement in the coding of spatial information, navigation, and orientation.

References and Reading

- Andersen, P., Morris, R., Amaral, D., Bliss, T., & O'Keefe, J. (2007). *The hippocampus book*. New York: Oxford University Press.
- Raymond, G., Bauman, M. L., & Kemper, T. L. (1989). The hippocampus in autism: Golgi analysis. *Acta Neuropathologica*, 91, 117–119.

Hippotherapy

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Definition

Hippotherapy is a treatment that uses the multi-dimensional movement of the horse. The term hippotherapy comes from the Greek word “hippos” which means horse. Specially trained physical, occupational, and speech therapists use this treatment for clients who have movement dysfunction and social deficits. The central tenant of the intervention is to use the movement the client experiences while on horseback to improve physical capabilities while developing a therapeutic relationship (All et al. 1999; Bass et al. 2009).

Historical Background

Pet ownership has been shown to be beneficial for individuals with a wide range of diagnoses (Voelker 1995). Pet or animal therapy is based on the assumption that interactions with animals can cause both physiological changes and psychological benefits (All et al. 1999). For example, Lehrman and Ross (2001) reported an increase in verbal sounds made by a 9-year-old girl with visual impairments when pet therapy was employed. In addition, children diagnosed with autism have been shown to make improvements in communication when animal-assisted therapy has been employed (Blue 1986). Social skills have

been taught to children with autism via the use of animal therapy (Law and Scott 1995; Turner 2005). Horseback riding has been shown to benefit children with communication disorders (Health and McKinney 1989). There have also been a few anecdotal reports that horseback riding and hippotherapy can have recreational and social benefits. Bass et al. (2009) evaluated the effects of horseback riding on the social functioning of children with autism. Utilizing a wait-list control design, those authors were able to demonstrate the efficacy of the interventions for increasing children's sensory seeking, sensory sensitivity, and social motivation. In addition, an inverse relationship was observed for problem behaviors such as inattentiveness and distractibility. Its use for children with autism spectrum disorders (ASD) is in its infancy, but the preliminary data are promising (Bass et al. 2009).

Rationale or Underlying Theory

At this time, the underlying theory for why hippotherapy has been beneficial appears to rely on both physiological and environmental factors (Macaulay and Gutierrez 2005). Hippotherapy is believed to help an individual develop an awareness of body movement, weight distribution, improved hand-eye coordination, improved speech, a wider tactile experience, and a wider experience of sounds (Macaulay and Gutierrez 2005). In a descriptive evaluation of therapist perceptions in Germany and Britain, Debuse et al. (2005) found that most practitioners of hippotherapy believed that hippotherapy improved the rejuvenation of muscle tone, improved postural control, and resulted in psychological benefits. However, these findings relied on the self-report of practitioners. Environmentally, the positive outcomes could be hypothesized to occur based on applied behavior analysis literature. For example, Carr and Durand (1985) demonstrated that through differential reinforcement, severe behaviors are reduced and a covariant increase in communicative behavior occurrences. Similarly, Derby et al. (1997) reported that if the child is provided an alternate

form of communication to obtain identified reinforcers (e.g., increased horse movement), appropriate social behavior increases. Thus, increased social behavior could be the function of a reinforcer/behavior contingency. Within the context of hippotherapy, the contingency would be between horse movement and social behavior.

Goals and Objectives

Physiologically, through the movement of the horse, the goal of hippotherapy is to help an individual develop an awareness of body movement, weight distribution, improved hand-eye coordination, improved speech, a wider tactile experience, and a wider experience of sounds (Macaulay and Gutierrez 2005). It is also used as a treatment tool by speech pathologists to increase the social motivation (Taylor et al. 2009), sensory seeking and sensitivity (Bass et al. 2009), and social behavior (Nelson et al. 2006).

Treatment Participants

Hippotherapy has been used as a therapy technique and is primarily used for persons with physical impairments (Benda et al. 2003) and speech impairments (Bass et al. 2009; Taylor et al. 2009).

Treatment Procedures

Occupational therapists, speech-language pathologists, and physical therapists typically perform the practice of hippotherapy. Even though every specialty derives its effectiveness from the unique movement of the horses, each enters the therapeutic environment with their own unique goals. Physical therapists target functional skills such as sitting, standing, and walking. Conversely, speech-language therapists use the therapy to remediate student communication. Each therapy modality relies on the movement of the horse to improve client function. Typical therapy sessions may last between 45 and 60 min. The therapy session is often broken down into distinct

segments, which may include putting on protective equipment such as a helmet, mounting the equine, engaging in activities while with horse, and dismounting the horse. For a safe and effective experience, a minimum of four trained professionals are required to complete a therapeutic session. A trained horse leader is always present who is responsible for prompting all horse movements. Two side-walkers are placed on either side of the equine to ensure client stability and safety. A lead therapist is assigned who is responsible for all therapeutic activities. Once mounting the equine activities is achieved, the procedures used focus on the individual needs of the client. The client may be seated on the equine a number of ways including sitting forward or sideways. Therapy is focused on the use of activities that are meaningful to the client and also meet treatment goals. For example, Benda et al. (2003) evaluate the effects of 8 min of a steady walking movement (4 min clockwise followed by 4 min counterclockwise) versus a stationary board control condition with children with spastic cerebral palsy. These authors found that following as little as 8 min of therapy, significant improvement in symmetry of muscle movements occurred. As an adjunctive speech intervention, Nelson et al. (2006) utilized a functional communication approach to increase the vocalizations of three children with autism spectrum disorder. In the Nelson et al. investigation, the children were required to sign or say "please" to prompt the horse handlers to start, stop, turn, and change the stride of the horse. The researchers found that the children's use of the functional communication and other social vocal responses increased. Nelson et al. hypothesized that the horse functioned as a reinforcer for appropriate communications. Similarly, Bass et al. (2009) exposed children with autism to 12, 1-h treatment sessions of hippotherapy across specific activities. Specifically, the therapist prompted the children to engage in a number of horse-related activities which included mounting/dismounting, riding skills, movement games (i.e., Simon says, catch and throw, etc.), and horse grooming. These investigators found that the children who participated in therapy exhibited increased sensory seeking, sensory sensitivity,

and social motivation. Regardless of the specific professionals conducting the therapy (i.e., occupational therapy, speech therapy, physical therapy), sessions are individualized in order to obtain functional goals for the client.

Efficacy Information

Overall, the literature found supports the efficacy of hippotherapy is in its infancy for increased social functioning (Bass et al. 2009). Conversely, the use of the horse as a therapeutic tool to increase posture, balance, and overall functioning of persons with motor dysfunction is better documented. For example, Benda et al. (2003) reported that hippotherapy could increase the symmetry of muscle activity when compared to sitting astride on a barrel using surface electromyography as an outcome measure. Positive effects have also been reported and have produced improved posture and balance for persons with cerebral palsy (Zadnikar and Kastrin 2011).

Outcome Measurement

Outcome measures vary depending on the focus of therapy. If therapy is focused on improving physical attributes, outcome measures range from the therapist using researcher-made objective scoring scales, weight bearing, and standardized measures such as the Gross Motor Function Measure (Russel et al. 2000) or the Pediatric Evaluation of Disability Inventory Scale (Feldman et al. 1990). In addition, surface electromyography has been used to measure muscle activity (Benda et al. 2003). If therapy is focused on social skill and motivation, measures typically involve the use of questionnaires and direct observational data collection systems. Bass, Duchowny, and Llabre documented sensory outcomes using the Sensory Profile (Dunn 1999) and increase social functioning and motivation using the Social Responsiveness Scale (Constantino 2002). Conversely, Nelson et al. (2006) measured increased social behaviors via in vivo measurement of child behaviors and vocalizations.

Qualifications of Treatment Providers

Licensed occupational therapists, physical therapists, and speech-language pathologists typically practice hippotherapy. A majority of the professionals that practice hippotherapy do not have a formal certificate beyond the qualifications for one of these professions. However, the American Hippotherapy Association has established a certificate process. Persons with these credentials need to have practiced their profession for 3 years, have 100 h of hippotherapy practice, and passed a multiple-choice examination.

References and Reading

- All, A. C., Loving, G. L., & Crane, L. L. (1999). Animals, horse riding, and implications for rehabilitation therapy. *Journal of Rehabilitation*, 65(3), 49–57.
- Bass, M. M., Duchowny, C. A., & Llabre, M. M. (2009). The ethics of therapeutic horseback riding on social functioning in children with autism. *Journal of Autism and Developmental Disorders*, 39, 1261–1267.
- Benda, W., McGibbon, W. H., Grant, K., & Davis, M. (2003). Improvement in muscle symmetry in children with cerebral palsy after equine-assisted therapy (hippotherapy). *Journal of Alternative and Complementary Medicine*, 9, 817–875.
- Blue, G. F. (1986). The value of pets in children's lives. *Childhood Education*, 62, 85–90.
- Carr, E., & Durand, V. M. (1985). Reducing behavior problems through functional communication training. *Journal of Applied Behavior Analysis*, 18, 111–126.
- Constantino, J. N. (2002). *The social responsiveness scale*. Los Angeles: Western Psychological Services.
- Debusse, D., Chandler, C., & Gibb, C. (2005). An exploration of German and British physiotherapists' views on the effects of hippotherapy and their measurement. *Physiotherapy Theory and Practice*, 21, 219–242.
- Derby, K. M., Wacker, D. P., Berg, W., DeRaad, A., Ulrich, S., Asmus, J., et al. (1997). The long-term effects of functional communication training in home settings. *Journal of Applied Behavior Analysis*, 30, 507–531.
- Dunn, W. (1999). *The sensory profile manual*. San Antonio: The Psychological Corporation.
- Feldman, A. B., Haley, S. M., & Coryell, J. (1990). Concurrent and construct validity of the pediatric evaluation of disability inventory. *Physical Therapy*, 70, 602–610.
- Health, T. D., & McKinney, P. C. (1989). Potential benefits of companion animals for self-care children. *Childhood Education*, 7, 311–314.
- Law, S., & Scott, S. (1995). Tips for practitioners: Pet care – A vehicle for learning. *Focus on Autistic Behavior*, 10(2), 17–18.
- Lehrman, J., & Ross, D. P. (2001). Therapeutic riding for a student with multiple disabilities and visual impairment: A case study. *Journal of Visual Impairment and Blindness*, 95, 108–109.
- Macaulay, B. L., & Gutierrez, K. M. (2005). The effectiveness of hippotherapy for children with language learning disabilities. *Communication Disorders Quarterly*, 25, 205–217.
- Nelson, K., Derby, K. M., Barretto, A., & Axtell, J. (2006). The use of equine therapy to increase social communicative behavior in children with autism. In D. Holmes (Chair) *Collaboration in Autism Intervention*. Paper presented at the annual conference of the Association for Behavior.
- Russel, D. J., Avery, L. M., Rosenbaum, P. L., Raina, P. S., Walter, S. D., & Palisano, R. J. (2000). Improved scaling of the gross motor function measure for children with cerebral palsy: Evidence of reliability and validity. *Physical Therapy*, 80, 873–885.
- Taylor, R. R., Kielhofner, G., Smith, C., Cahill, S. M., Ciaka, M. D., & Gehman, M. (2009). Volitional change in children with autism: A single case design study of the impact of hippotherapy on motivation. *Occupational Therapy in Mental Health*, 25, 192–200.
- Voelker, R. (1995). Puppy love can be therapeutic, too. *Journal of the American Medical Association*, 279, 1897–1899.
- Zadnikar, M., & Kastrin, A. (2011). Effects of hippotherapy and therapeutic horseback riding on postural control or balance in children with cerebral palsy: A meta-analysis. *Developmental Medicine and Child Neurology*, 53(8), 684–691.

Histaprin [OTC]

► Diphenhydramine

Histidinemia

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Definition

Histidinemia is an essential amino acid during infancy. In the 1960s, disorders of histidine metabolism were thought to be associated with

speech delay and intellectual impairment. There were also multiple case reports noting the co-occurrence of histidinemia and various disorders, including autism. Using newborn screening and dietary management of phenylketonuria as a model, New York and Massachusetts implemented newborn screening programs for histidinemia. By the late 1970s, histidinemia was understood as a benign metabolic variant, and enthusiasm for state-sponsored newborn screening for histidinemia has become a cautionary tale.

History of Autism as a Diagnostic Concept

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Historical Background

Among the many questions that surround autism, one likely never to get a solid, uncontroverted answer is the following:

How far back in human history does autism go?

It is obvious that at some stage in our species' development, some person was first to display the behaviors we today call "autistic." To date, however, we have no way to identify when or where that happened on the timeline of human evolution. There are no direct fossils for behavior. Despite that, it seems reasonable and intuitive to assume that the autism's defining behaviors, relative to our own time, are nothing new – and that a thousand years ago, there were people acting in ways which, if they were observed today, would prompt a modern-day diagnosis of autistic spectrum disorder (ASD).

That such individuals were *not* diagnosed during their own lifetimes, over most of those ten centuries, owes to a simple fact that autism, *as a diagnostic concept*, achieved recognition only remarkably recently, coming into existence, in the minds of medical observers, only around the time that World War II was getting started.

Before that, people exhibiting such behaviors, if they were pronounced enough, risked getting pinned with some other label inevitably suggesting either mental illness or intellectual disability. The sorts of hurtful-sounding terms used in the past – lunatic, psychotic, demented, idiot, imbecile, retarded, among many others – were actual clinical categories in their day, before lay society twisted them into slurs. They also had consequences. Starting in the nineteenth century, especially in the more "advanced" societies of Europe and North America, people so labelled were increasingly pushed out of mainstream society and shunted off to supposed "asylums" or "training schools." Initially launched with therapeutic and educational aspirations, these institutions all too often decayed into soul-destroying human warehouses, overcrowded, underfunded, and inhumane. Lost within their populations, where their cluster of "odd" behaviors were not recognized as distinctively meaningful in any way, were many people with today's ASD. They were lost, in part, because no one had ever heard of autism.

Current Knowledge

In 1943, a child psychiatrist named Leo Kanner published a groundbreaking description of 11 children, in which he made the case in vivid detail that their short histories merited a new kind of diagnosis. These eight boys and three girls were clearly not typical in their development – to the extent, Kanner wrote that "several... were introduced to us as idiots or imbeciles." Following the usual pattern, most of the children, none older than 11, had already been sent off to institutions, some given diagnoses of "feble-mindedness" or schizophrenia. To Kanner's eyes, however, there was something seriously wrong with these assessments. The example of the boy he designated "case 1" demonstrates why.

"Donald T" was 5 years old in 1938, the first time his concerned parents brought him from Mississippi to be seen by Kanner at his clinic at Johns Hopkins University. In describing Donald, Kanner relied on his own and his staff's

observations, then and over the next few years, while also quoting liberally from a lengthy description of his behaviors written by Donald's father: "It was observed at an early time that he got happiest when left alone. . . He paid no attention to persons around him." He had never cried for his mother and never noticed his father's arrivals home from work. Yet inanimate objects fascinated him, and he "developed a mania for spinning blocks and pans and other round objects." His speech was unusual too. Donald was using words by 13 months, but he reversed the pronouns "you" and "I" and spoke with a flat intonation. When asked a question, his answer was to recite the question, verbatim, followed by silence. Otherwise, his utterances consisted mainly of the same small set of phrases, such as "I could put a little comma or semicolon," which he had once heard someone else say and then taken to speaking aloud in a ritualistic manner.

Finally, Kanner noted, Donald hated change and cherished repetition.

Most of his actions were repetitions carried out in exactly the same way in which they had been performed originally. If he spun a block, he must always start with the same face uppermost. When he threaded buttons, he arranged them in a certain sequence that had no pattern to it but happened to be the order used by the father when he first had shown them to Donald. There were also innumerable verbal rituals recurring all day long. . . [which his mother had to comply with] or else he squealed, cried, and strained every muscle in his neck in tension.

But there was something else: Donald was clearly gifted in some areas. His memory was prodigious, and he was very quick at absorbing information.

At the age of 1 year 'he could hum and sing many tunes accurately.' Before he was 2 years old, he had 'an unusual memory for faces and names, knew the names of a great number of houses' in his home town. . . He knew the pictures of the presidents and knew most of the pictures of his ancestors and kinfolks on both sides of the house. He quickly learned the whole alphabet 'backward as well as forward' and to count to 100.

To Kanner, such talents ruled out intellectual disability as an explanation. Schizophrenia made no sense either, since its symptoms typically first manifest in adolescence and include

hallucinations, which were no part of Donald's presentation.

At the same time, Kanner did discern a few overarching patterns in Donald's behaviors and in those of the other ten children he described, which he judged significant. One was their distinct relationship to, and use, of language. Another was an "obsessive need for sameness." A third was what Kanner referred to as "autistic aloneness." Critically, too, Kanner concluded that these traits were "innate," "inborn," and present "from the beginning of life." He declared these as the key aspects of the syndrome, when he published his findings, in 1943, in a paper entitled "Autistic Disturbances of Affective Contact."

Kanner's choice of that adjective – *autistic* – proved consequential. It was not his invention. By 1943, it was already in use, and familiar to psychiatrists – along with the noun form *autism* – but in a context significantly different from the one Kanner was introducing. Till that time, adjective and noun alike had served to label exclusively a transient symptom in adults with schizophrenia: their tendency at times to disengage from contact with their external surroundings and the people in them. Such patients entered "a world of their own. . . encased with their desires and their wishes," wrote the Swiss psychiatrist Eugen Bleuler in 1911. Indeed, Bleuler himself came up with the term. "This detachment from reality and absolute predominance of the inner life, he wrote, "*we call autism*." He based it on the Greek word *αὐτός*, which means "self."

Kanner did not attempt to anoint his syndrome "autism" in 1943, when he used the noun form of the word only once in the 34 pages of his journal article. Instead, he made liberal use of Bleuler's adjective "autistic" as a descriptor – a way to get across *some* aspects of what his children's behaviors resembled. But, as Kanner saw it, the comparison went only so far, as he made clear that this rough resemblance to schizophrenic autism – what he referred to as "extreme aloneness" – was but part of the total story. "The total picture," he pointed out, included additional behaviors such as "obsessiveness, stereotypy and echolalia," which fell outside the boundaries of autism as the term's coiner defined it. Certainly, Kanner's

contemporaries grasped these nuances, and for years afterwards, when requiring a name for the syndrome Kanner had described, chose to call it, not “autism” but “Kanner’s syndrome.”

Notably, a similar borrowing of Bleuler’s concept took place, at nearly the same time, in Vienna, where a pediatrician named Hans Asperger had published work in German in 1938 and 1944, describing a number of Austrian boys struggling significantly in the domain of interpersonal relationships. Their challenges, as Asperger described them, resulted from a syndrome of obliviousness to social cues, inflexibility in behavior, and a tendency to develop obsessive interests in narrowly limited subjects. Overall, Asperger’s boys differed in significant ways from Kanner’s group, exhibiting greater capability for independence, creative output, and intellectual performance (above average in some cases) than the children Kanner wrote about. In short, Asperger highlighted children who were likely to struggle socially throughout life but were less likely to be institutionalized. And where Kanner considered his syndrome to be “rare enough,” Asperger saw his as “not at all” rare.

Like Kanner, however, Asperger used Bleuler’s autism as a reference point, and in a similar way, to suggest a rough resemblance to some aspects of schizophrenia’s social detachment. Again like Kanner, he did not call his syndrome “autism” (though, confusingly, the definitive English translation of his 1944 paper has him doing so, in more than 20 instances) but employed the adjective in describing his boys as “autistic psychopaths” and in the title of his major paper on the subject “Autistic Psychopathy in Childhood.” It should be noted that the term “psychopathy” in German best translates to English as “personality disorder,” without the negative connotations the word carries in English.

That both men made the same word choice has been put down by some to mere coincidence and by others to the normal cross-pollination of ideas that occur in science and medicine. In fact, Kanner employed a former colleague of Asperger’s, a Jew fleeing Vienna ahead of Nazi persecution whom also Jewish Kanner helped reach the United States

and with whom he could well have shared insights. As it happened, however, for the next 40 years, Asperger’s writing about boys he called “autistic” went largely overlooked outside the German-speaking world, while Kanner’s proved definitive everywhere else.

Why does autism’s etymological lineage merit focus at all? Because in it lie the origins of a major and enduring obstacle to studying, treating, and even talking about the disorder, one that remains operative to this day, namely, that from the beginning, well-intentioned voices have disagreed vigorously about what autism actually is, how to describe it, and where to draw its boundaries. This started with the terminology, a problem the astute British child psychiatrist and autism researcher Michael Rutter pointed to in 1978, when he lamented: “To begin with, there was the unfortunate choice of name.” As Rutter explained, this caused immediate confusion, owing to the unintended implication that these children had schizophrenia after all. Kanner played an unintentional part in that. In 1944, in an effort at clarity, he began referring to his syndrome as “early infantile autism.” He did this to reemphasize that the “extreme aloneness” – the “autism” which comprised but one part of the picture – was there from the start, from infancy. Notably, this was *his* first use of the noun form in this manner, and, with hindsight, it marks the moment where his descriptions of autistic-like behaviors began shaping up into a new “-ism” of their own. In the short term, however, calling his condition *autism* only muddled the waters for the many who knew the term solely by connection to schizophrenia.

A few years after Kanner’s initial paper, a remarkable sort of diagnostic melee ensued from the confusion. On the one hand, other researchers and clinicians began bringing forth new reports of children under their care similar in presentation to Kanner’s but under diagnoses they packaged according to slightly different parameters and under disparate names – for example, “symbiotic psychosis,” “prepubertal schizophrenia,” “infantile psychosis,” and “atypical child.” Again, Rutter put his finger on the problem: “It is by no means clear that all these authors are talking about

the same condition.” Then, too, it was by no means clear they weren’t.

On the other hand, and at the same time, there were many clinicians who positively embraced Kanner’s syndrome but who interpreted the diagnostic criteria far more loosely than he ever did. By the mid-1950s, Kanner was complaining about autism diagnoses being rendered merely for “one or another isolated symptom,” inflating the number of cases out of all proportion to reality. “Almost overnight,” he later said of this early period, “the country seemed to be populated by a multitude of autistic children.”

Such diagnostic disorder of course played havoc with the lives of people being given these labels, who in this period were entirely children. It also confounded attempts to conduct scientific research on autism. How, for example, were epidemiologists to determine a prevalence rate of autism when there was such profound disagreement on what constituted its necessary and sufficient symptoms?

This was exactly the issue faced by a London-based psychology graduate student named Victor Lotter, who in 1964 attempted the first-ever prevalence study of autism among the population of boys and girls aged eight to ten living in Middlesex County, England – a total of 78,000 children. When Lotter turned to the medical literature to construct a checklist of behaviors that were flags for autism, he discovered a definitional din: numerous competing portraits of the condition which, taken together, yielded little clarity but instead established – as he said with chagrin – “the lack of agreed behavioural criteria.” Pressing on, however, Lotter improvised by combining some of Kanner’s criteria with several from a list known as Creak’s nine points. With these as his starting point, Lotter in 1966 reported a prevalence rate of autism of 1 in 2500 among his cohort in Middlesex. But even as he did so, the intellectually honest student sent up a critical warning that his finding should not be considered dispositive. “‘True’ prevalence,” Lotter wrote near the end of his report, “may not be a useful concept in the case of a syndrome (or syndromes) so poorly defined.” Despite that, to this day, advocacy groups cite

Lotter’s 1 in 2500 result as representing a solid fact: what the *global* prevalence rate of autism “used to be.”

Efforts to standardize criteria for the diagnosis would preoccupy stakeholders for years to come. Continuing frustration over the issue appeared regularly in the scholarly literature, literally for decades. The following are some examples:

- In 1968, Michael Rutter was writing, not for the last time, about the “hopelessly confused state of affairs.”
- In 1978, Eric Schopler, of the University of North Carolina, was arguing that scientists working on autism were not understanding one another’s research “because criteria for diagnosis are different.”
- In 1988, Yale’s Fred Volkmar and Donald Cohen were voicing frustration over the “long and controversial history of the concepts surrounding autism.”
- In 1998, Yale’s Ami Klin, joined by Volkmar, was underscoring “the confusion and the plethora of diagnostic concepts.”

Schopler’s comment, in particular, illustrates but one ramification of this diagnostic confusion: disputes over criteria tended to confound research. Perhaps more insidious than mismatched symptom lists – for the sake of people getting diagnosed – was the clash over basic *concepts* of autism – the term used by Volkmar, Cohen, and Klin. A syndrome so prone to definitional malleability (largely because it offered no clear biomarkers but has been based on the interpretation of behaviors) also lent itself to rather disparate theories concerning its essential nature. Even as they came up short as universal explanations for autism, many of these represented coherent and insightful ideas while doing little harm. For a time, for example, one theory held that the core deficit, at least in more classic cases of autism, was a failure in the facility of language and that all other symptoms, including social challenges, flowed from that. Other ideas placed compromised cognitive functions at the center, with much attention given to the part played by

“theory of mind” and “weak central coherence.” Still others went down more biological paths, seeking to understand whether sensory issues, and even metabolic ones, were at the heart of the disorder.

And then there was the so-called psychogenic model of autism, which caused enormous harm to autistic individuals and their families. Better known as the “refrigerator mother theory” and dominant from the late 1940s at least into the mid-1970s, this concept of autism held that the disorder was a child’s defensive reaction to insufficient maternal love. Treatment, such as it existed, centered on intensive psychotherapy of the mother (and sometimes the father as well). Reflecting the sway held by traditional psychoanalytic thinking at mid-century, especially in American psychiatry, the psychogenic concept stole decades of meaningful psychological and biological research attention to the topic of autism, bringing enormous pain to families and no practical therapy or support for autistic people themselves.

The only good thing that can be said about the dominance of the “refrigerator mother” theory is that it finally lost currency, roughly around the time that steps were taken to codify a valid, science-and-data-based autism diagnosis in the American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorders* (DSM). In itself, however, this effort turned into yet another endless deliberation, spanning four decades (the late 1970s to the present), during which the successive working groups of experts recruited to validate autism’s definition from one DSM edition to the next repeatedly moved the boundary lines – sometimes jarringly.

To some extent, the zigzags reflected the fact that fresh data on autism was coming in all the time, shedding new light on the disorder, and legitimizing revisions. But each update (and they tended to be significant) reflected something else: a negotiation. By design, the expert working groups represented diverse points of view, whose members brought to the table their own distinctive frameworks for thinking about autism. These were shaped by their training (psychiatry or

psychology), by the professional milieus in which they worked (numerous regions of the world were represented), and especially by each person’s own interactions with autistic people either as researcher or clinician (itself an important distinction, though many of the recruited experts were both). Inevitably, though, diversity meant disagreement, and consensus rarely meant unanimity. To a degree that might surprise the lay observer, the evolving definition of autism – at least as it appeared in successive editions of the DSM – reflected a debate and a vote and an outcome influenced by which people in the room possessed the better powers of persuasion.

A case in point: The radical overhaul that autism’s diagnostic criteria underwent between 1980 and 1987.

DSM-III, which appeared in 1980, marked the first time autism was addressed in *any* DSM (3 years after it was listed in the World Health Organization’s International Classification of Diseases). DSM-III used a name that closely tracked what Kanner came up with 36 years earlier – “infantile autism” – placing it under the category “pervasive developmental disorders,” with the diagnostic code number 299.00. At a mere 70 words long, its tightly written criteria comprised three categories of behavior – lack of responsiveness to other people, deficits in communicative skills, and “bizarre responses to . . . the environment” – with a few sample behaviors listed for each category: for example, echolalia, in the communication category, and resistance to change, in the “bizarre responses” category.” No guidance was given on how to weigh these various behaviors against one another in deciding on a diagnosis.

The picture was dramatically different 7 years later, with the publication of an updated DSM-III-R (“R” for “revision”). Gone for good in 1987 was “infantile autism,” which was seen to cloud recognition that autism was lifelong. Also gone were a further four closely pervasive developmental disorders – for example, “infantile autism residual state,” which was supposed to account for individuals whose autistic behaviors lost some of their intensity over time. Now, DSM-III-R gave the

299.00 designation to an overhauled construct it dubbed “autistic disorder.” Designed to be both less restrictive than the old “infantile autism” and more reflective of developmental concepts (Szatmari 1992), it was framed around a diagnostic checklist that ran to more than 600 words. Again, symptomatic behaviors were grouped around three themes. This time, the categories were social interaction, communication (verbal and nonverbal), and a “restricted repertoire of activities and interest.” Also, a more deliberative diagnostic scheme was put in place. Now 16 specific behaviors were listed – such as “gross impairment in ability to make peer friendships” and “marked distress over changes in trivial aspects of environment, e.g., when a vase is moved from usual position.” These 16 behaviors were sorted among the 3 categories, and a rule was specified: diagnosis required a match with at least 8 of the 16 behaviors; at least 2 had to belong to the social interaction group; and at least 1 had to come from the other two groups.

This more operational approach to diagnosis was but one of that edition’s innovations in autism diagnosis. The other, even more sweeping one, was the creation of a controversial new “sister” diagnosis: “299.80, Pervasive Development Disorder Not Otherwise Specified.” The inelegant nomenclature belied a well-intentioned purpose – to provide a diagnostic landing spot to individuals who were near misses for the 299.00 diagnosis. These were defined as people displaying “qualitative impairment in the development of reciprocal interaction and of verbal and nonverbal communication skills,” but not to the extent, or in such combinations, that satisfied the checklist. Because this relatively vague definition made allowance for even quite mild symptoms, some would come to think of PDDNOS as a kind of “autism-lite” – especially compared to the “classic” autism Kanner had described. But this misunderstood the breadth and depth of impairment a diagnosis of PDDNOS might represent for many people who were given this diagnosis.

In the push and pull among experts, 1987’s *DSM* revision also represented a period of

ascendancy for one particularly compelling viewpoint on how to make sense of a phenomenon obvious to everyone: that autistic traits were remarkably variable in how differently they presented themselves in different people. This is the observation that gives rise to the oft-repeated aphorism, “when you’ve met one person with autism, you’ve met *one* person with autism.” In practice, the matter of how to interpret this heterogeneity has been an enduring source of tension in understanding what the word “autism” properly represents. One view holds that these differences likely indicate distinct and separate underlying syndromes – different autisms, so to speak – which should be delineated as such by splitting them off from one another. Both Kanner and Asperger, for example, stood among the so-called splitters, as both insisted adamantly that, despite some overlap, each had described a full syndrome distinct from the other’s. The splitter viewpoint was dominant in the 1980 *DSM*, where autistic traits were covered across those five different diagnoses.

But in the 1987 *DSM*, the “splitter” position lost ground to a competing perspective: that in nearly all cases, nearly all manifestations of autistic traits resulted from the same core mechanism or mechanisms, which happened to affect different people to different degrees. Those holding this view were often called the “lumpers,” for preferring to see past the differences and for preferring to “lump” together, to the maximum extent, the vast array of people displaying any measure of autistic behavior.

Prominent in this group was the British psychiatrist Lorna Wing, who played a singular role in crystalizing the concept she called the autism “spectrum,” the profoundly influential proposition that all autistic behavior – though representing a huge range of intensities, from profound to extremely mild, while operating independently of intelligence – is nevertheless rooted in the same syndrome of impairments, which she identified as a “triad of impairments.” This model, originally based on epidemiological studies Wing carried out with Judith Gould in the mid-1970s London, was not a dramatic departure from

previous formulations, specifying three areas of challenge: in social interaction, in the use of reciprocal language, and in what they called “social imagination,” where the ability to pretend play is an example of the last item.

Where she did break ground, however, was in the inclusiveness of her concept, which resisted the “splitters” tendency to want to draw sharp lines between, say, those who were called “higher” and “lower” functioning. Such distinctions, Wing wrote disapprovingly, have the “effect of excluding those who do not fit neatly into the categories.” It should come as no surprise that Wing served on the working group that developed the PDDNOS diagnosis in 1987, which was an excellent example of a “lumper”-type category and took a major step toward a “spectrum”-type conceptualization of autism. Wing, the mother of a daughter with autism, was motivated in part by a desire to justify support services for a far broader range of people than narrow diagnosis would allow. In time, the implementation of her ideas led to just that outcome.

But that is not to say that the diagnostic push and pull ever ended. Indeed, in 1994, it moved back toward “splitting” mode, slicing up the pervasive developmental disorders once again to include, in addition to autistic disorder, some of the previously “not otherwise specified” PDD conditions: Rett’s disorder, childhood disintegrative disorder, and, half a century after it was first described by its namesake, Asperger’s disorder.

It was Lorna Wing who had rediscovered Asperger’s work in the late 1970s and then popularized it in the 1980s. Wing’s involvement proved ironic, however, because her intention was never to see Asperger’s “autistic psychopathy” recognized as a standalone diagnosis. Rather, she brought attention to the Austrian’s work for the opposite purpose: she had wanted to suggest just how far the spectrum concept could be stretched, given that Asperger’s children were so different from Kanner’s, even as both employed “autistic” to describe some of their behaviors. The elevation of Asperger’s in 1994’s *DSM* was an outcome Wing neither desired nor anticipated, always

insisting there “no clear boundaries separating it from other autistic disorders (Wing 2005).”

Yet acknowledgment of Asperger’s syndrome by the *DSM* that year (and even earlier in the *ICD*) proved pivotal across multiple fronts. It quickly caught on with clinicians, becoming an increasingly assigned diagnosis, often employed, Wing herself believed, to spare parents the discomfort of hearing the word “autism.” The diagnosis also began to be given to adults – people who had grown up facing social challenges without the benefit of a diagnostic perspective in which to frame their difficulties. Finally, recognition of Asperger’s as “a form of autism” – a phrase the media often used – had important cultural impact, to some degree destigmatizing autism in general by association with often highly intelligent and articulate self-advocates who positively embraced their own diagnoses as central to their identities. Plays, books, and movies began to appear, centering sympathetically on the stories of people who more or less matched the diagnosis, while websites began appearing designed for, and later by, self-described “Aspies,” who expressed pride in the ways they were different from the population at large.

But then, in 2013, a new *DSM* appeared, and this one no longer included Asperger’s syndrome. Once again demonstrating the extreme malleability of such constructs, the team assigned to define the pervasive developmental disorders listened to the researchers and clinicians who had long complained that Asperger’s was actually a problematic concept, resistant to stringent criteria, and understood differently in different clinical settings – or, as one team member puts it, the diagnosis had proven “confusing and not terribly useful.” Asperger’s was dropped and so, too, was PDDNOS and all the other specified developmental disorders that had been in use for the previous two decades. Indeed, in vindication of Lorna Wing’s 35-year-old argument that autism represented a spectrum, a new formulation appeared in the latest *DSM*, using that term “299.00 Autistic Spectrum Disorder” (ASD).

Future Directions

ASD represented the apotheosis of “lumper” thinking, by aiming to eliminate all reference to sub-syndromes. In addition, the criteria aimed to be inclusive of nearly all individuals previously assigned any of the now-vanished diagnoses, as well as some who had earlier qualified for no diagnosis at all. As a result of this development, the number of individuals empowered to claim “I have autism” or “I’m autistic,” by reference to how professionals in the field defined the term, reached an all-time high and is estimated to account for roughly 1 percent of the human race.

At least, for now. It is by no means clear that the all-encompassing spectrum will remain the last word in how autism is perceived and addressed. The lack of certainty about the underlying drivers of autistic behaviors remains as persistent as ever, while the interplay of subjective and even competitive perspectives in the diagnostic process remains as problematic as ever. It is entirely possible that genetic and other biomedical research may, ultimately, discover the existence of multiple pathways to the same still rather amorphous grouping of the behaviors that are used to identify autism. This could once again revive interest in delineating multiple syndromes “hidden” within the spectrum, each with its own instigator, internal mechanisms, and treatments. If that were that to happen, the ever-evolving debate about what we all mean when we say “autism” would, without a doubt, enter yet another new round.

See Also

- ▶ [Asperger, Hans](#)
- ▶ [Broader Autism Phenotype](#)
- ▶ [DSM-5](#)
- ▶ [DSM-III](#)
- ▶ [DSM-III-R](#)
- ▶ [DSM-IV](#)
- ▶ [Kanner, Leo](#)

References and Reading

- Asperger, H. (1944). Die Autistischen Psychopathen im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Bettelheim, B. (1967). *The empty fortress: Infantile autism and the birth of the self*. New York: Free Press.
- Bleuler, E. (1950). *Dementia praecox, or the group of schizophrenias* (trans: J. Zinkin). New York: International Universities.
- Creak, M. (1961). Schizophrenic syndrome in childhood: Progress report of a working party (April 1961). *British Medical Journal*, 2, 889.
- Donvan, J., & Zucker, C. (2016). *In a different key: The story of autism*. New York: Crown Publishers/Random House.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kanner, L. (1944). Early infantile autism. *The Journal of Pediatrics*, 25(3), 211–217.
- Kanner, L. (1965). Infantile autism and the schizophrenias. *Behavioral Science*, 10(4), 412–420.
- Lotter, V. (1966). Epidemiology of autistic conditions in young children. *Social Psychiatry*, 1(3), 163–173.
- Rutter, M. (1968). Concepts of autism: A review of research. *Journal of Child Psychology and Psychiatry*, 9(1), 1.
- Rutter, M. (1978). Diagnosis and definition of childhood autism. *Journal of Autism and Childhood Schizophrenia*, 8(2), 139–161.
- Schopler, E. (1978). On confusion in the diagnosis of autism. *Journal of Autism and Childhood Schizophrenia*, 8(2), 137–138.
- Szatmari, P. (1992). A review of the DSM-III-R criteria for autistic disorder. *Journal of Autism and Developmental Disorders*, 29(4), 507.
- Volkmar, F. R., & Cohen, D. J. (1988). Classification and diagnosis of childhood autism. In E. Schopler & G. Mesibov (Eds.), *Diagnosis and assessment in autism* (p. 72). New York: Plenum Press.
- Volkmar, F. R., & Klin, A. (1998). Asperger syndrome and nonverbal learning disabilities. In E. Schopler, G. Mesibov, L. Kunc (Eds.) *Asperger Syndrome or High-Functioning Autism?* (pp 107–121). New York: Plenum Press.
- Wing, L. (1981a). Language, social, and cognitive impairments in autism and severe mental retardation. *Journal of Autism and Developmental Disorders*, 11(1), 31–44.
- Wing, L. (1981b). Asperger’s syndrome: A clinical account. *Psychological Medicine*, 11(1), 115–129.
- Wing, L. (2000). Past and future of research on Asperger syndrome. In A. Klin, R. R. Volkmar, & S. S. Sparrow (Eds.), *Asperger syndrome*. New York: Guilford Press.
- Wing, L. (2005). Problems of categorical classification systems. In F. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders, diagnosis, development neurobiology, and behavior*. Hoboken: Wiley.

Hoarding in Youth with Autism Spectrum Disorders

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Definition

Before publication of the DSM-5, hoarding was conceptualized as a symptom of obsessive-compulsive disorder (OCD). However, research findings has led to the reclassification of pathological hoarding, known as hoarding disorder (HD), into a separate diagnosis from OCD with primary diagnostic criteria encompassing (a) a persistent difficulty discarding possessions, (b) distress associated with discarding items, (c) the accumulation of clutter, and (d) clinically significant distress/impairment (American Psychiatric Association 2013). The age of onset of compulsive hoarding ranges from 11 to 15 years old, but symptoms may not become clinically significant until adulthood (Tolin et al. 2010). The overall prevalence rate of HD ranges from about 1.5 to 5.8%, though most research has studied adults (Nordsletten and Mataix-Cols 2012; Timpano et al. 2011).

Hoarding behaviors may be common among youth with autism spectrum disorders (ASD) for three reasons. First, executive functioning deficits (e.g., in planning, attention, task completion, organization) are associated with both childhood (and adult) HD and ASD and could contribute to hoarding behaviors. High rates of HD have been found with ADHD (Fullana et al. 2013; Hacker et al. 2016), which is comorbid in 28.2–30.6% of cases with ASD (Leyfer et al. 2006; Simonoff

et al. 2008). Executive functioning difficulties may be associated with impulsive collecting/acquisition, difficulty organizing and discarding possessions, and planning to throw away objects in the future (Storch 2011a). Second, the repetitive and restricted interests associated with ASD may influence incidence of hoarding behaviors (Storch et al. 2016). For instance, a child with ASD, who has a strong interest in geology, may attempt to acquire rocks and related items and have difficulty discarding those items. Third, the overlap between ASD and anxiety disorders in children and adolescents may increase the risk or severity of HD. Anxiety disorders are often maintained by safety cues (e.g., stuffed animals, music, caregivers) that children or adolescents use to mitigate the anxiety they experience from aversive stimuli (Storch et al. 2016). As a result, children and adolescents may develop personal attachments to these objects that lead to strong emotional reactions when having to discard or separate from them.

Assessment

In children and adolescents, behaviors meeting criteria for HD must first be differentiated from developmentally appropriate behaviors (i.e., hobbies, collections) as well as symptoms associated with ASD (e.g., the collection of objects due to restricted interests or repetitive behaviors). To aid in this differentiation, Storch et al. (2011b) note that children who hoard often collect objects such as boxes, paper clips, school papers, stickers, items from nature (i.e., rocks), or erasers. In addition, children and adolescents who hoard may develop strong emotional attachments to objects by assimilating them into their personal identities (Plimpton et al. 2009). These attachments are often strong enough to cause significant distress about hurting or damaging specific items, thereby preventing children or adolescents from discarding them. Another distinguishing feature is that among children and adolescents who hoard, removing or throwing away hoarded objects typically leads to negative emotional/behavioral reactions (Plimpton et al. 2009).

To assist with diagnoses and treatment, a multimodal, multi-method assessment that identifies hoarding symptoms and comorbidities is recommended for children with hoarding behaviors (Frost and Hristova 2011). An interview with a trained clinician who has experience in working with children with ASD is the cornerstone of assessment. Currently, the only developmentally appropriate measure is the Child Saving Inventory (CSI; Storch et al. 2011a), which is a 23-item, parent-rated scale assessing four domains: discarding, clutter, acquisition, and distress/impairment. Beyond this, broadband measures that include items that assess hoarding may be helpful (e.g., Achenbach 1991) as well as the Children's Yale-Brown Obsessive-Compulsive Scale (CY-BOCS; Scahill et al. 1997), which includes a single item assessing hoarding symptom presence, as well as overall severity.

Treatment

Few studies have been conducted with children and adolescents experiencing comorbid ASD and HD. Although not among children with ASD and HD, case studies have reported success in childhood HD when using a parent-focused behavioral intervention involving contingency management. For instance, Ale et al. (2014) demonstrated that parental involvement played an integral role in treatment to generalize skills to multiple settings, to increase treatment gains, and to provide encouragement. In his case study on a 10-year-old with OCD and hoarding behaviors, McKay (2016) first utilized an assessment battery containing the CY-BOCS and conducted a functional assessment to provide more detail about the patient's hoarding behaviors. When the functional assessment revealed hoarding that was narrowly focused on objects related to academics (e.g., schoolwork from years ago), he implemented cognitive-behavioral therapy with exposure and built a fear hierarchy to help the patient systematically address her hoarding behaviors. Similar to Ale et al. (2014), McKay (2016) also highlighted the need for parent-provider collaboration and reward systems to boost motivation and treatment compliance. Preliminary data are also available from a

study focused on cognitive-behavioral therapy (CBT) for anxiety among youth with ASD. Storch et al. (2016) conducted exposure-based CBT with 26 youth with ASD and a comorbid anxiety disorder over 16, 90-minute sessions. Although the treatment was focused on anxiety broadly, core hoarding symptoms across several domains (e.g., distress, clutter, difficulty discarding, and acquiring items) decreased following intervention.

Future Directions

There are several areas highlighted for future work. First, large epidemiological studies may elucidate the role of genetics and heritability in ASD and HD. As a result, more accurate prevalence rates will narrow comorbidity estimates and provide researchers and practitioners with more accurate base rates for their diagnostic decisions. Second, treatment protocols for HD in youth are needed, as well as how to adapt such interventions to be individualized to working with youth with ASD. Third, further development and evaluation of rating scales to supplement the CSI will improve assessment practices and increase diagnostic accuracy. Finally, the literature could benefit from studies investigating shared etiology in HD and ASD that could provide a developmental/prospective course of such symptoms.

See Also

- ▶ [Anxiety](#)
- ▶ [Anxiety Disorders](#)
- ▶ [Behavior Therapy](#)
- ▶ [Child Psychotherapy](#)
- ▶ [Cognitive Behavioral Therapy \(CBT\)](#)
- ▶ [Diagnosis and Classification](#)
- ▶ [Diagnostic Process](#)
- ▶ [DSM-5 and Autism Spectrum Disorder](#)
- ▶ [General Anxiety](#)
- ▶ [Onset](#)
- ▶ [Prevalence](#)
- ▶ [Psychotherapy](#)
- ▶ [Repetitive Behavior](#)
- ▶ [Stereotypic Behavior](#)
- ▶ [Study Design Impact on Prevalence](#)

References and Reading

- Achenbach, T. M. (1991). *Manual for the child behavior checklist/4-18 and 1991 profile*. Burlington: Department of Psychiatry, University of Vermont.
- Ale, C. M., Arnold, E. B., Whiteside, S. P., & Storch, E. A. (2014). Family-based behavioral treatment of pediatric compulsive hoarding: A case example. *Clinical Case Studies*, 13(1), 9–21.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Frost, R. O., & Hristova, V. (2011). Assessment of hoarding. *Journal of Clinical Psychology*, 67(5), 456–466.
- Fullana, M. A., Vilagut, G., Mataix-Cols, D., Adroher, N. D., Bruffaerts, R., Bunting, B., . . . & Alonso, J. (2013). Is ADHD in childhood associated with lifetime hoarding symptoms? An epidemiological study. *Depression and Anxiety*, 30(8), 741–748.
- Hacker, L. E., Park, J. M., Timpano, K. R., Cavitt, M. A., Alvaro, J. L., Lewin, A. B., . . . & Storch, E. A. (2016). Hoarding in children with ADHD. *Journal of Attention Disorders*, 20(7), 617–626.
- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., . . . & Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36(7), 849–861.
- McKay, D. (2016). Cognitive-behavioral treatment of hoarding in youth: A case illustration. *Journal of Clinical Psychology*, 72(11), 1209–1218.
- Nordsletten, A. E., & Mataix-Cols, D. (2012). Hoarding versus collecting: Where does pathology diverge from play? *Clinical Psychology Review*, 32(3), 165–176.
- Plimpton, E. H., Frost, R. O., Abbey, B. C., & Dorer, W. (2009). Compulsive hoarding in children: Six case studies. *International Journal of Cognitive Therapy*, 2(1), 88–104.
- Seahill, L., Riddle, M. A., McSwiggin-Hardin, M., Ort, S. I., King, R. A., Goodman, W. K., . . . Leckman, J. F. (1997). Children's Yale-Brown obsessive compulsive scale: Reliability and validity. *Journal of the American Academy of Child & Adolescent Psychiatry*, 36(6), 844–852.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, 47(8), 921–929.
- Storch, E. A., Lack, C. W., Merlo, L. J., Geffken, G. R., Jacob, M. L., Murphy, T. K., & Goodman, W. K. (2007). Clinical features of children and adolescents with obsessive-compulsive disorder and hoarding symptoms. *Comprehensive Psychiatry*, 48(4), 313–318.
- Storch, E. A., Muroff, J., Lewin, A. B., Geller, D., Ross, A., McCarthy, K., . . . Steketee, G. (2011a). Development and preliminary psychometric evaluation of the Children's saving inventory. *Child Psychiatry & Human Development*, 42(2), 166–182.
- Storch, E. A., Rahman, O., Park, J. M., Reid, J., Murphy, T. K., & Lewin, A. B. (2011b). Compulsive hoarding in children. *Journal of Clinical Psychology*, 67(5), 507–516.
- Storch, E. A., Nadeau, J. M., Johnco, C., Timpano, K., McBride, N., Mutch, P. J., . . . & Murphy, T. K. (2016). Hoarding in youth with autism spectrum disorders and anxiety: Incidence, clinical correlates, and behavioral treatment response. *Journal of Autism and Developmental Disorders*, 46(5), 1602–1612.
- Timpano, K. R., Exner, C., Glaesmer, H., Rief, W., Keshaviah, A., Brähler, E., & Wilhelm, S. (2011). The epidemiology of the proposed DSM-5 hoarding disorder: Exploration of the acquisition specifier, associated features, and distress. *Journal of Clinical Psychiatry*, 72(6), 780–786.
- Tolin, D. F., Meunier, S. A., Frost, R. O., & Steketee, G. (2010). Course of compulsive hoarding and its relationship to life events. *Depression and Anxiety*, 27(9), 829–838.

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Short Biography

Liane Holliday Willey, Ed.D. (born 1959), is a best-selling American author, educator and public speaker. She was diagnosed with Asperger's syndrome in 1996. She coined the term "Aspie" in her 1999 autobiography, *Pretending to be Normal: Living with Asperger Syndrome*. Dr. Willey has authored several other internationally best-selling books, all published by Jessica Kingsley Publishers, including *Asperger Syndrome in Adolescence: Living with the Ups, the Downs and Things in Between* and *Asperger Syndrome in the Family: Redefining Normal*. Her latest book is *Safety Skills for Asperger Women: How to Save a Perfectly Good Female Life* (2011).

Dr. Willey has contributed to many additional books and journals and is currently the senior editor for *Autism Spectrum Quarterly*. In addition to her numerous interviews on national and international television and radio, her life story was an

inspiration for the film *Normal Folk*, currently in preproduction, and the feature film *Adam*, as well as the focus of the video *Asperger Syndrome: Crossing the Bridge* with Dr. Tony Attwood.

Dr. Willey believes the Asperger's syndrome community is a wonderfully rich world that is populated by some of the most interesting and incredible individuals on the planet and she is exceptionally proud to be a part of it. She is the founder of the Asperger Society of Michigan.

References and Reading

- Willey, L. H. (1999). *Pretending to be normal: Living with Asperger's syndrome*. London: Jessica Kingsley.
- Willey, L. H. (2001). *Asperger syndrome in the family: Redefining normal*. London: Jessica Kingsley.
- Willey, L. H. (2003). *Asperger syndrome in the adolescent years: Living with the ups, the downs and things in between*. London: Jessica Kingsley.
- Willey, L. H. (2010). *Unwrapping the mysteries of Asperger's: The search for truth and discovery of solutions – Guide for girls and women with Asperger's syndrome*. AuthorHouse.
- Willey, L. H. (2011). *Safety skills for Asperger women: How to save a perfectly good female life*. London: Jessica Kingsley.

Home Living Skills

► Daily Living Skills

Home Schooling in Autism

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Definition

The 1990 Individuals with Disabilities Education Act (IDEA) promotes students being placed in the least restrictive environment for educational purposes, and that has encouraged the widespread

placement of students with autism spectrum disorder (ASD) in brick-and-mortar schools – public and private, general education and special education, and integrated and specialized. The heterogeneity of the ASD population complicates this task because while certain core communications deficits affect most individuals with ASD, the levels of intensity and interference vary widely, as do the particular constellations of symptoms that any person manifests. The characteristics of students with ASD, and the lack of preparedness of schools to deal with them, have had a negative impact on the experiences of many of these students in school settings, with problems encompassing poor academic performance, bullying, and strained social relationships. For many ASD students, homeschooling – where there are fewer conflicts and greater individualization – could provide a more appropriate and successful setting for learning.

Homeschooling is an option available to all students, and families choose this alternative educational setting for a variety of reasons, including religious, professional, financial, medical, social, and philosophical. Homeschooling programs can be taught by parents, by joining in cooperatives with other families, by computer, or by hiring professional pedagogues. In the case of students with ASD, those who function at a higher intellectual level can usually avail themselves of the full range of homeschool implementation choices. But students with more severe manifestations of ASD generally require trained, experienced clinicians, special education teachers, and therapists to carry out an effective individualized home-based education plan that fosters meaningful academic, social, and behavioral progress.

Historical Background

The Centers for Disease Control and Prevention (CDC) reports that 1 in 68 children in the United States has been diagnosed with ASD (Baio 2014), and all of those children are entitled to a Free and Appropriate Public Education (FAPE), as dictated by the 1990 Individuals with Disabilities Education Act. Prior to 1975, school districts could

decide that a child with a disability was too difficult, or too expensive, to educate. In 1970, for example, government statistics show that only 1 in 5 children with disabilities attended public schools, with school districts regularly refusing with impunity to embrace children who were, among other disorders, deaf, blind, and mentally retarded. Most children with special needs were educated in private schools paid for by their families, or they were institutionalized (United States Department of Education, Office of Special Education and Rehabilitative Services, pamphlet date unknown). In 1975, however, Congress passed the Education for All Handicapped Children Act, enshrining the rights of all children to a government-funded education, no matter their circumstances. The law was expanded and updated in 1990 with the passage of IDEA and further amended in 2004. Today, the vast majority of students with disabilities, including those with ASD, are taught in brick-and-mortar schools (Fleury et al. 2014; Chen and Schwartz 2012). The schools are public and private, special education, and mainstream. Some ASD students learn alongside typically developing peers, in some cases with assistance from special educators or paraprofessionals for all or part of their days.

IDEA demands that students be allowed to learn in the least restrictive environment, a rule intended to counteract the forced warehousing of students with disabilities that happened in decades past. The process for deciding the appropriate placement for any student is complex. What seems restrictive for one person might be more liberating and productive for another. For some students, even those with high IQs, being in a traditional school involves a level of sensory overstimulation, conformity to extensive rules, and continuous social interaction that could impede their ability to make meaningful educational progress. Indeed, current methods for dealing with ASD students show limited long-term success. People with ASD rank third from the bottom in college enrollment among 11 disability categories. Meanwhile, only 37 % of young adults with ASD are employed, and most of them only in part-time jobs without benefits (Fleury et al. 2014). Ironically, those with ASD who are not

intellectually compromised have triple the chance of being unemployed and unengaged in leisure activities with their peers, compared with young adults who have ASD coupled with intellectual disabilities (Fleury et al. 2014). This statistic is especially disconcerting because the overwhelming majority of people diagnosed with ASD has milder manifestations of the disorder (Van Bergeijk et al. 2008) and presumably should be able to attain greater success in the classroom and beyond. In general, minimal research has been done on this population as it moves past young adulthood; still, while these shards of data are limited, they offer a bleak window onto the prospects for ASD adults.

Current Knowledge

Academic Barriers to School Success for ASD Students

The characteristics that define a person as having ASD render school a treacherous system to navigate. In all its iterations, autism is primarily a communication disorder that undermines a person's capacity to interact with others appropriately (Van Bergeijk et al. 2008). People with ASD have, to varying degrees, a compromised ability to observe, imitate, and apply new skills – all essential components of acquiring knowledge. They typically exhibit rigid and repetitive behaviors that interfere with learning and dealing with other people, whether fellow students, teachers, or school staff. Their auditory and visual processing speed is often slow and difficult to manage (Fleury et al. 2014; Van Bergeijk et al. 2008). Their conversation skills are severely constrained – at the most affected edge of the spectrum in basic expressive and receptive language and even in quality of speech production, and at the higher functioning and Asperger syndrome (AS) edge, in their dexterity in reciprocating and empathizing with another in conversation (Fleury et al. 2014; Van Bergeijk et al. 2008; Volkmar and Klin 2001).

The list of scholastic roadblocks runs on. While many ASD students have good decoding skills in reading, they often have poor

comprehension, in part because of their difficulty in assuming the perspective of another (Fleury et al. 2014). In writing, poor fine motor skills interfere with the physical ability to produce words on paper or to type, a process further frustrated by weak visual motor speed and coordination. In terms of content, trouble with empathy and perspective gets in the way of composing creatively and with clarity (Fleury et al. 2014). And one of the most complex skills needed to succeed in a school environment is the organization born of executive function processes that most people with ASD lack (Rosenthal et al. 2013).

Over the last few decades, schools have relied more upon team learning and projects, a looming obstacle for ASD students compromised in their pragmatic and social language abilities, and their facility at reading social cues well enough to join forces for a group endeavor (Durkin and Conti-Ramsden 2007). Moreover, as focus shifts from individual students with ASD to the group, executive functioning deficits can seriously undermine their ability to retain information and contribute meaningfully to the collaborative work (Rosenthal et al. 2013).

People with ASD also have a high incidence of other psychiatric and neurological conditions that exacerbate the autism itself and can inhibit learning. Common comorbid disorders for people with ASD include attention deficit hyperactivity disorder (ADHD), anxiety, depression, grapho-motor deficits, seizures, and intellectual disabilities (Van Bergeijk et al. 2008). Indeed, Van Bergeijk et al. (2008) note that 65 % of people with ASD have anxiety and/or depression (p. 1361), conditions that on their own would impede any student's ability to cope in the fast-paced academic and social world in a school and that can overwhelm a person already reeling under the weight that ASD deficits impose.

Imagine for a minute the ASD student in a school building bustling with children racing to class, returning from a football game, or practicing for the annual musical. The aroma of tacos and pizza wafts from the cafeteria – a crowded room abuzz with laughter, conversation, and song. In addition to all the intellectual and organizational

skills he lacks, the ASD student is also sensitive to sudden noise and movement, keenly aware of sights, sounds, and smells he cannot control.

These deficits are obstacles to every facet of learning in a school filled with other pupils, whether those students share one's disabilities or are free of them. Since every person with ASD has a different cluster of symptoms, behaviors, challenges, strengths, and compensations, schools are stymied in their efforts to create comfortable environments for all. The heterogeneity of the population makes it impossible for schools to develop an easy, "off-the-rack" approach to deal with ASD students. Furthermore, behavioral problems can deluge the child and staff, interfering with the learning not only of the individual with ASD but of the entire class as well. Given the staggering demands, there are generally insufficient academic and behavioral support services to foster reliable success for ASD students in a school setting (Van Bergeijk et al. 2008; Catherine Lord, personal communication, June 25, 2014).

One significant measure of the subpar school options being offered to students with ASD is the rise in litigation by dissatisfied parents seeking reimbursement for nonpublic school programs for their children. Parents of children with ASD are 10 times more likely than parents of children with other disabilities to sue school districts to force fulfillment of FAPE, and they account for about a third of all cases filed overall (Zirkel 2001). This disproportionality is due in part to the complexity and severity of ASD but also speaks to the failure of school districts to offer the proper comprehensive services and environment necessary to educating a student with the disorder.

Social Barriers to School Success for ASD Students

By their nature, schools and classrooms are not just centers of scholarship; they are communal organizations that depend on the kind of compromise and cooperation that eludes most people with ASD because the interfering symptoms and behaviors are the root expression of the neurological condition itself. Poor social skills are a hallmark of people all along the autism spectrum

(Volkmar and Klin 2001; Durkin and Conti-Ramsden 2007; Van Bergeijk et al. 2008). When someone has poor social competence and lagging academic skills, the two deficiencies feed off one another in a way that produces negative outcomes in both spheres (Welsh et al. 2001). There is a “chicken-and-egg” debate about which is the primary determinant of the other: some research suggests that academic failure leads to students being ostracized socially, while other research points to low social status as a source of low academic achievement. Whether or not a definitive cause and effect can be established, it is clear that over time, the two influence one another and are mutually predictive. Welsh et al.’s (2001) longitudinal study found that “academic competence exerts a significant influence over social competence consistently,” with the negative correlate being true as well (pp. 477–478).

This finding bodes ill for students with ASD studying in traditional schools, where managing social relationships with teachers, fellow students, and staff is enmeshed in all daily activities. The need while in school to respect boundaries, to empathize with others, to be flexible, and to read social cues thwarts people with ASD, who lack such emotional intelligence when it comes to dealing with peers and with adults who work in schools. Even the most academically capable students with ASD, namely, those with Asperger syndrome, suffer from poor social skills, sensory issues, and pragmatic language deficits (e.g., lack of reciprocal conversation and perseverative topics) that compromise their overall performance and well-being in school (Church et al. 2000). The inability to communicate appropriately through language circumscribes a child’s ability to form close relationships. When combined with impaired social cue reading and inconsistent self-regulation, ASD students are limited in their ability to access the friendships theoretically available in a school setting (Durkin and Conti-Ramsden 2007). High functioning students with autism, although unburdened by comorbid intellectual disabilities, can be so rigid and rules-bound that they often, for example, tattle on their classmates, causing further social alienation (Church et al. 2000). As one parent lamented

about his son, “He doesn’t have weak social skills, his social skills are non-existent!” (Church et al. 2000, p. 18).

The opportunity to socialize with peers, to learn from their example how to behave appropriately, is often cited as a critical potential benefit for ASD students in attending schools. Reality, however, does not commonly live up to such expectations, according to the literature and interviews with experts in the field. For ASD students in specialized schools, peers often model maladaptive behaviors – such as tantrums and flapping – that should not be imitated but frequently are. For those in mainstream schools, acceptance is not easily forthcoming. In school, where one sits, the foods one eats, the clothes one wears, all send myriad silent signals that most ASD students cannot interpret. “The greatest lesson of childhood is not reading, writing or arithmetic,” said Dr. Orrin Devinsky, a neurologist and the director of NYU’s Comprehensive Epilepsy and Seizure Center, who has many patients with ASD since seizures are a common comorbid condition with autism (O. Devinsky, personal communication, June 26, 2012). “For most of human evolution, we acquired language without teachers, just older individuals who spoke. Socialization is the key of childhood. The lesson. But for ASD kids, they are with other ASD kids and the lesson of socialization is a bizarre and foreign one – the role models are skewed to watching kids with self-injurious behaviors, tantrums, lack of eye contact, etc. For those who get some mainstream time, there are great benefits (more normal social peers) but also cases where kids can be insensitive and cruel.”

Media stereotypes of people with autism coupled with ignorance reinforce the social sand traps that the disorder itself sets. Typical children generally reveal unfavorable attitudes toward peers with autism when they are shown pictures and film clips of them. Although some research has shown that these negative associations improve when the children are given explanations for the aberrant behavior – including the lack of eye contact and repetitive motions, such as arm flapping – other research does not (Campbell et al. 2005). This negative stereotyping not only influences students but also affects the views of their

parents, of teachers in the classroom, and of the general school staff. In some cases, students engaged as peer mentors can help bring an ASD classmate into the social fold. This intervention tends to work best when the mentors are so-called high-status children, whose leadership skills and popularity influence classroom social dynamics. But the research indicates that acceptance of the ASD student can be fleeting because it occurs due to a desire by the other students to please the mentors, not a genuine change in their attitudes. The mentors themselves often do not change their attitudes either, nor do they always act out of altruism; rather, they take on the role generally because they perceive that being an advocate for a disabled peer will enhance their own leadership ranking (Campbell et al. 2005).

Few typical students are willing or able to become a genuine protector to their ASD classmates. Most students simply ignore them. But some seize on the ASD students as easy prey for ostracizing and maltreatment. Having low status and diminished social skills leaves many students with ASD particularly vulnerable to bullying in school, as targets are often awkward, marginalized young people with poor peer relationships – characteristics common to ASD students (Cappadocia et al. 2011; Chen and Schwartz 2012). Parents, students, and teachers report high levels of victimization of students with ASD by fellow classmates. About 40 % of students with ASD report being bullied, according to Dr. Fred Volkmar, director of the Yale Child Study Center; for those with a comorbid condition, that number rises to roughly 75 % (F. Volkmar, personal communication, June 29, 2014). Research by Cappadocia et al. in 2011 put the percentage of ASD students who are victimized as possibly higher, with 77 % of parents interviewed reporting that their children with ASD had been bullied at school in the previous month (Cappadocia et al. 2011). Such victimization has serious negative consequences for the target from academic, behavioral, social, physical, and psychological perspectives (Chen and Schwartz 2012; Sterzing et al. 2012).

School bullying is difficult to root out, however, because it involves a complex web of social interactions, rather than a singular act of cruelty

(Cappadocia et al. 2011). Bullies are generally adept at choosing moments of attack that evade adult detection, and students with ASD often lack the verbal skills to report their own victimization. Even when they are able to identify an attacker, ASD students – and, indeed many typically developing students who are targets – overwhelmingly fail to report being abused because they are ashamed, fear retribution, or are too socially oblivious to realize they have been humiliated or shunned (Campbell et al. 2005; Chen and Schwartz 2012). Moreover, ASD students generally do not have the self-advocacy skills to confront their tormentors or a safety net of peers to look out for them and comfort them when they are victimized (van Roekel et al. 2010). So instead of learning from typical peer models how to socialize appropriately, ASD students generally pull back from social interactions, or worse: Sometimes they imitate the bullying behaviors and target others – either out of anger, or ignorance (Chen and Schwartz 2012). Indeed, many people with ASD are emotionally labile and even abusive (Church et al. 2000), characteristics that make them especially susceptible to the invidious lure of bullying others.

Even when ASD students avoid the perils of bullying – as both perpetrators and, more often, victims – the structure of the school environment underscores that they are different from their peers. They labor under labels that regularly remind them – and their classmates – that they stand apart. They are the ones with special educators or paraprofessionals shadowing them in the classrooms and on the playing fields, the ones who get extra time on tests, or have augmentative communication devices, the ones who get pulled out of class for therapy. Throughout the school day, the emphasis is on their differences, not their similarities – a dagger to the self-esteem of those special education students intellectually and emotionally aware enough to take note (Ensign 2000).

Future Directions

There is scant peer-reviewed or scholarly research and literature on homeschooling students with

ASD. There are some books on the subject, but they are almost entirely how-to manuals, with some incorporating personal stories. None contains any systematic research. As such, in order to hypothesize how a similar home learning environment might work for students with ASD, extrapolating from the professional literature that examines the homeschooling experiences of less compromised students, for example, those with learning disabilities or ADHD, is required. In a 9-year longitudinal study of homeschooled special education students, Ensign (2000) revealed that learning in a home environment freed such students of the stigmas and stereotypes that inhibited their education and socialization in traditional schools – mainstream and specialized. Ensign noted research that showed “the culture of school orchestrated labeling and disabling” students with special needs (p. 149), a plight common to ASD students educated in schools, while being at home minimized or eliminated those stressors.

Duvall led two different investigations into the homeschool experiences of children with other interfering disorders – one in 1997 of students with learning disabilities and one in 2004 of students with ADHD (Duvall et al. 1997, 2004). Both studies showed that students with special needs flourished in the calmer, more attentive home environment and that they engaged significantly more with their work than they had when they were in school, making greater reading and math gains than their peers with similar issues who attended the public schools. Although small in size, both studies indicated that homeschooling provides an advantage to students who need more intensive instruction.

In her study, Ensign (2000) found that the success emanated from the fact that parents were able to create an educational milieu at home that emphasized the strengths of special students, rather than their deficits, and that encouraged excellence based on individual aptitude and interests. Unbound by the dogma of most state rules and regulations, the parents (or their surrogate teachers) could give extra time for areas of struggle and encourage acceleration in areas of promise (Ensign 2000). Dr. Devinsky said he has observed

that for ASD students with heightened sensitivities and circumscribed capabilities, a homeschool program often offers the only appropriate environment for fostering progress and forestalling regression. “The greatest benefits of homeschooling are removing a child from a chaotic environment where their mind may be hyperstimulated and so overwhelmed they shut down or explode as their major behavioral patterns,” he noted (O. Devinsky, personal communication, June 26, 2014). Furthermore, he stated that homeschooling students with ASD “removes them from negative peer behaviors and models (they don’t learn to spit or hit their heads) and can remove them from a chaotic environment to a more nurturing one, and can allow a more relaxed and individually targeted program” (O. Devinsky, personal communication, June 26, 2014).

Creating a homeschool program for typically developing students is cumbersome; for ASD students with the need for highly specialized therapies and instruction, doing so is especially costly – financially and in the burden it places on the student’s family. It puts added pressure on the parents to earn enough money to cover the significant expenses such programs impose and burdens them with managing its details (F. Volkmar, personal communication, June 29, 2014). Since IDEA guarantees a free and appropriate public education to all children, when the school district fails to provide an ASD student with an education that is appropriate to his or her unique needs, parents often construct a program on their own and then litigate to receive reimbursement to fulfill the promise of “free” embedded in FAPE. Not all families have the intellectual, emotional, and financial resources to sue their school districts. The process can be long and success is not assured. The responsibility to ascertain what their ASD student needs educationally – and to arrange, monitor, and fund the program – falls on the shoulders of parents already exhausted by the responsibility of caring for a challenging child. “Most families do not have situations in which students could learn at home – they do not have enough space and often do not have someone at home who can coordinate various educational activities,” reported Dr. Catherine Lord, Director

of the Center for Autism and the Developing Brain in New York, and one of the creators of the Autism Diagnostic Observation Schedule (ADOS), a leading assessment tool for diagnosing ASD (C. Lord, personal communication, June 25, 2014). But faced with inadequate programs in their local schools, parents of students with ASD are increasingly crafting individualized homeschool programs for their children outside of the public or private school system, and they are increasingly suing for reimbursement of the substantial costs (Zerkel 2011).

For many students, the barriers to learning and socializing in brick-and-mortar schools can be overcome, and they can achieve success. But the literature that does exist and the experiences of experts in the field suggest that many students could do much better academically, behaviorally, socially, and psychologically in an individualized homeschool setting. At home there are no learning disabilities, there is only learning. As such, this approach should be studied in a comprehensive and methodical way to measure its potential for improved academic, post-academic, and social outcomes for students with ASD.

See Also

- [Bullying](#)
- [Communication Disorder/Communication Impairment](#)
- [Comorbidity](#)
- [Free Appropriate Public Education](#)
- [Home-Based Programs](#)
- [Individual Education Plan](#)
- [Sensory Impairment in Autism](#)
- [Social Behaviors and Social Impairment](#)

References and Reading

- Baio, J. (2014, March 28). Prevalence of autism spectrum disorder among children aged 8 Years — Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2010. Centers for Disease Control and Prevention. Retrieved July 2, 2014, from http://www.cdc.gov/mmwr/preview/mmwrhtml/ss6302a1.htm?s_cid=ss6302a1_w.
- Campbell, J. M., Ferguson, J. E., Herzinger, C. V., Jackson, J. N., & Marino, C. (2005). Peers' attitudes toward autism differ across sociometric groups: An exploratory investigation. *Journal of Developmental and Physical Disabilities, 17*, 281–298.
- Cappadocia, M. C., Weiss, J. A., & Pepler, D. (2011). Bullying experiences among children and youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 42*, 266–277.
- Chen, P. Y., & Schwartz, I. S. (2012). Bullying and victimization experiences of students with autism spectrum disorders in elementary schools. *Focus on Autism and Other Developmental Disabilities, 27*, 200–212.
- Church, C., Alisanski, A., & Amanullah, S. (2000). The social, behavioral, and academic experiences of children with asperger syndrome. *Focus on Autism and Other Developmental Disabilities, 15*, 12–20.
- Durkin, K., & Conti-Ramsden, G. (2007). Language, social behavior, and the quality of friendships in adolescents with and without a history of specific language impairment. *Child Development, 78*, 1441–1457.
- Duvall, S. F., Ward, D. L., Delquadri, J. C., & Greenwood, C. R. (1997). An exploratory study of home school instructional environments and their effects on the basic skills of students with learning disabilities. *Education and Treatment of Children, 20*, 150–172.
- Duvall, S. F., Delquadri, J. C., & Ward, D. L. (2004). A preliminary investigation of the effectiveness of homeschool instructional environments for students with attention-deficit/hyperactivity disorder. *School Psychology Review, 33*, 140–158.
- Ensign, J. (2000). Defying the stereotypes of special education: Home school students. *Peabody Journal of Education, 75*, 147–158.
- Fleury, V. P., Hedges, S., Hume, K., Browder, D. M., Thompson, J. L., Fallin, K., El Zein, F., Klein Reutebuch, C., & Vaughn, S. (2014). Addressing the academic needs of adolescents with autism spectrum disorder in secondary education. *Remedial and Special Education, 35*, 68–80.
- Giangreco, M. F., Broer, S. M., & Edelman, S. W. (2001). Teacher engagement with students with disabilities: Differences between paraprofessional service delivery models. *Journal of the Association for Persons with Severe Handicaps, 26*, 75–86.
- Koegel, R. L., Fredeen, R., Kim, S., Danial, J., Rubenstein, D., & Koegel, L. (2012). Using perseverative interests to improve interactions between adolescents with autism and their typical peers in school settings. *Journal of Positive Behavior Interventions, 14*, 133–141.
- Pennington, R. C., & Delano, M. E. (2012). Writing instruction for students with autism spectrum disorders: A review of literature. *Focus on Autism and Other Developmental Disabilities, 27*, 158–167.
- Prater, M. A. (2003). Learning disabilities in children's and adolescent literature: How are characters portrayed? *Learning Disability Quarterly, 26*, 47–62.
- Rogers, S. (2000). Interventions that facilitate socialization in children with autism. *Journal of Autism and Developmental Disorders, 30*, 399–409.

- Rosenthal, M., Wallace, G. L., Lawson, R., Wills, M. C., Dixon, E., Yerys, B. E., & Kenworthy, L. (2013). Impairments in real-world executive function increase from childhood to adolescence in autism spectrum disorders. *Neuropsychology*, 27, 13–18.
- Sterzing, P. R., Shattuck, P. T., Narendorf, S. C., Wagner, M., & Cooper, B. P. (2012). Bullying involvement and autism spectrum disorders: Prevalence and correlates of bullying involvement among adolescents with an autism spectrum disorder. *Archives of Pediatric and Adolescent Medicine*, 166, 1058–1064.
- Van Bergeijk, E., Klin, A., & Volkmar, F. (2008). Supporting more able students on the autism spectrum: College and beyond. *Journal of Autism and Developmental Disorders*, 38, 1359–1370.
- Van Roekel, E., Scholte, R. H. J., & Didden, R. (2010). Bullying among adolescents with autism spectrum disorders: Prevalence and perception. *Journal of Autism and Developmental Disorders*, 40, 63–73. doi: 10.1007/s10803-009-0832-2.
- Volkmar, F. R., & Klin, A. (2001). Asperger syndrome and higher functioning autism: Same or different? *International Review of Research in Mental Retardation*, 23, 83–110.
- Welsh, M., Park, R. D., Widaman, K., & O'Neil, R. (2001). Linkages between children's social and academic competence: A longitudinal analysis. *Journal of School Psychology*, 39, 463–481.
- Zirkel, P. A. (2001). Autism litigation under the IDEA: A new meaning of "disproportionality"? *Journal of Special Education Leadership*, 24, 92–103.

Home-Based Programs

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Definition

Home programs to provide treatment for young children with autism were pioneered by O. Ivar Lovaas in the 1970s and 1980s. A home program focuses on remediating the children's delays in communication and social and emotional skills and emphasizes the integration of these children with typical peers in mainstream community settings. Principles derived from laboratory and applied research on learning psychology are used to teach developmentally appropriate and functional skills.

As described by Green and colleagues, a home program has the following characteristics:

1. Treatment is individualized and comprehensive, addressing all skill domains.
2. Typical developmental sequences derived from data from developmental psychology guide the selection of intervention goals and short-term objectives.
3. Multiple behavior analytic procedures are used to teach new skills and to reduce interfering behavior (e.g., differential reinforcement, prompting, discrete-trial teaching, natural environment teaching, activity-embedded trials, task analysis, and others).
4. One or more professionals with advanced training in applied behavior analysis and advanced training in behavioral intervention with young children with autism direct and supervise the intervention.
5. Parents serve as active co-therapists for their children.
6. Intervention is initially delivered in one-to-one sessions, with gradual transitions to small-group and large-group formats as a child progresses.
7. Intervention begins in the home and is carried over into other environments (e.g., community settings), with gradual, systematic transitions to preschool, kindergarten, and elementary school classrooms when children develop the skills required to learn in those settings.
8. Intervention involves intensive, year-round teaching, including 20–40 h of structured sessions per week plus informal instruction and practice throughout most of the children's other waking hours.
9. In most cases, a child receives two or more years of intervention.
10. Most children start intervention in the pre-school years.

Home programs are usually associated with early intervention programs, especially Early and Intensive Behavioral Intervention (EIBI) for children with autism. The use of home programs to deliver EIBI originates from the Lovaas

approach (the UCLA Model) and the UCLA Young Autism Project.

Historical Background

Home programs have roots in applied behavior analytic (ABA) interventions that were first introduced for children with autism in the early 1960s. Home programs originated a decade later as a result of finding from the first long-term follow-up study of ABA interventions for children with autism (Lovaas et al. 1973). Twenty children with autism participated. After 12–14 months of behavioral treatment, stereotyped behaviors including echolalia decreased, and appropriate speech, appropriate play, and social nonverbal behaviors. Some of the children were discharged to a state hospital while others remained with their parents, who had received parent training. Follow-up assessments one to four years after the end of treatment showed that children whose parents were trained to carry out the behavioral treatment continued to improve, while children who were institutionalized regressed. After a brief reinstatement of the behavioral treatment, however, the institutionalized children recouped some of their original gains.

From the 1973 follow-up study, Lovaas made several important observations that led him to develop the home program. First, the youngest children in the 1973 study appeared to make the greatest gains; hence, he focused on providing treatment to toddlers and preschoolers with autism and regarded home as the most developmentally appropriate setting for children. Second, treatment effects in the 1973 study were situation specific. As a result, treatment was moved away from a hospital or clinic setting and into the children's everyday environments (including the home). Third, some parents became skilled teachers of their children and became the best allies in helping accelerate and maintain treatment gains. Provision of treatment at home maximized opportunities for parental involvement.

Following the 1973 study, a home program became an integral part of EIBI services, especially in the UCLA Young Autism Project. In 1987, Lovaas (1987) reported an evaluation of a group of 19 children with autism who participated in a home-based EIBI program for 40 h per week for a minimum of 2 years. This report indicated that, after treatment, the average IQ of children in home-based EIBI programs was more than 30 points higher than 40 children in comparison groups and that the children were much more likely to be included in general education settings. Subsequent reports also showed that children in home-based EIBI made large gains relative to comparison groups (e.g., Cohen et al. 2006; Sallows and Graupner 2005; Smith et al. 2000).

Since Lovaas's (1987) report, home programs have become increasingly accepted as an intervention for young children with autism. They are a common feature of EIBI services that emphasize ABA teaching methods. They are also a component of other treatment models such as the Developmental, Individual Differences, Relationship-Based Approach (DIR).

Rationale or Underlying Theory

In a home program, an underlying rationale is that the treatment environment should differ as little as possible from the typical learning environment in order to help the child with autism transfer skills to the typical environment and function more successfully in that setting. Also, active involvement of the parents and other family members is a crucial component of treatment success.

Goals and Objectives

A home program is usually set up for delivering an EIBI curriculum such as the curriculum described in the sections on the UCLA Young Autism Project and Lovaas approach. The curriculum is individualized based on each child's needs and stages of development that typically developing children go through.

Treatment Participants

The home program was developed primarily in EIBI models for children with autism who have mild to moderate intellectual disability and are under the age of 42 months at treatment onset. However, it has also been evaluated with children with more severe intellectual disability. Several studies have examined relations between intake variables and outcome measures. Meta-analyses have indicated that the most robust finding to date is a strong positive correlation between treatment intensity and outcome.

Treatment Procedures

Typically, 30 to 40 h per week of one-to-one instruction is provided in the child's home by the parents and trained therapists. If the child is younger than 3 years of age, 20 to 30 h per week of instruction is more common. Initially, most of the teaching is carried out in a discrete trial format, which is a highly structured teaching procedure used to establish a number of important skills, including cognitive skills, communication, play, social, and self-help skills. Also, time is devoted to play and other structured activities, in which the child has opportunities to use skills acquired during discrete trial teaching (e.g., labeling toys or taking turns with the tutor during a game) and learning new skills in situations where they naturally occur (e.g., learning names of new toys, acquiring motor skills while playing soccer, or practicing dressing before going out for community trips).

After the child has acquired important communication, play, and social skills, play dates with typical peers become a priority for the home program. The tutor facilitates mastered activities for the child and peer (e.g., conversation, pretend play with toys, or turn-taking games) and encourages the peer to engage the child.

After the child is able to sit still and attend to a teacher during small group instruction, and has learned prerequisite communication, preacademic (e.g., cutting, pasting, drawing, doing puzzles)

play, and social skills, the child is mainstreamed in a regular preschool. At that time, there is a gradual decrease in number of one-to-one hours at home and a gradual increase in time spent in school. Trained tutors accompany the child to school functioning as a classroom aide.

Efficacy Information

Since the original study by Lovaas (1987), there have been a number of other studies on home-based EIBI programs. Recently, a number of effect-size studies have been published, and several report large effect sizes for IQ and adaptive functioning.

While researchers generally agree that the results of studies have generally been positive, there remains controversy about the scientific quality of the studies, and many have called for additional research with improved research designs. In addition, direct comparisons between home programs and center-based or school-based programs have not been conducted. Thus, it remains unclear to what extent the provision of treatment at home contributes to overall outcomes.

Outcome Measurement

The most common outcome measures used are IQ, language, and adaptive functioning. The Bayley Scales of Infant Development and the Wechsler Intelligence Scales (WIPPSI or WISC) are the most common IQ tests.

The most widely used instrument to assess language is the Reynell Developmental Language Scales, but due to their severe delays in communication, many participants fail to achieve basal on the Reynell at intake.

To assess adaptive behavior, the Vineland Adaptive Behavior Scales is used.

Some studies have also assessed other domains, such as maladaptive behavior (using the maladaptive scale from Vineland Adaptive Behavior Scales, the Personality Inventory for

Children, and the Child Behavior Checklist), school performance (using the Woodcock-Johnson III Tests of Achievement), and changes in diagnosis (using the Autism Diagnostic Interview-Revised (ADI-R).

Qualifications of Treatment Providers

There are currently no established programs to certify professionals to become competent home-program therapists or home-program supervisors. Practitioners of the home program indicate that, to become a competent therapist, individuals should obtain a bachelor's degree in psychology, education, or social work, with coursework applied behavior analysis, developmental disorders, and child development. In addition, they require therapists to work at least three months hands-on with a child in an apprenticeship with a trained therapist. At the end of this training, the therapist must be able to apply the behavioral principles correctly during one-to-one teaching.

To become a supervisor, Lovaas and colleagues have required the practitioner to be an experienced home-program therapist and hold a master's degree that includes graduate courses in applied behavior analysis, developmental disorders, and child development. In addition, the supervisors must learn how to conduct staff training, develop and individualize the child's curriculum, and work with families. These skills can be learned through working in an apprenticeship with a competent home-program supervisor, and by studying available teaching manuals, evidenced-based parent training programs, and typical development. Finally, the supervisor should be supervised by a Ph.D. level clinical psychologist specializing in the home program.

See Also

- [Early Intensive Behavioral Intervention \(EIBI\)](#)
- [Early Intervention](#)
- [Lovaas, O. Ivar](#)
- [Psychological Assessment](#)
- [UCLA Young Autism Project](#)

References and Reading

- Bibby, P., Eikeseth, S., Martin, N. T., Mudford, O. C., & Reeves, D. (2002). Progress and outcomes for children with autism receiving parent-managed intensive interventions. *Research in Developmental Disabilities, 23*, 81–104.
- Cohen, H., Amerine-Dickens, M., & Smith, T. (2006). Early intensive behavioral treatment: Replication of the UCLA model in a community setting. *Developmental and Behavioral Paediatrics, 27*, 145–155.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). New Jersey: Prentice Hall.
- Davis, B. J., Smith, T., & Donahoe, P. (2002). Evaluating supervisors in the UCLA treatment model for children with autism: Validation of an assessment procedure. *Behavior Therapy, 33*, 601–614.
- Eikeseth, S. (2009). Outcome of comprehensive psycho-educational interventions for young children with autism. *Research in Developmental Disabilities, 30*, 158–178.
- Eikeseth, S. (2010). Examination of qualifications required of an EIBI professional. *European Journal of Behavior Analysis, 11*, 239–246.
- Eikeseth, S. (2011). Intensive early intervention. In J. L. Matson & P. Sturmey (Eds.), *International handbook of autism and pervasive developmental disorders* (pp. 321–338). New York: Springer.
- Eldevik, S., Hastings, R. P., Hughes, J. K., Jahr, E., Eikeseth, S., & Cross, S. (2009). Meta-analysis of early intensive behavioral intervention for children with autism. *Journal of Clinical Child and Adolescent Psychology, 38*, 439–450.
- Eldevik, S., Hastings, R. P., Hughes, J. K., Jahr, E., Eikeseth, S., & Cross, S. (2010). Analyzing outcome for children with autism receiving early intensive behavioral intervention: Secondary analysis of data from 457 children. *The American Journal on Intellectual and Developmental Disabilities, 34*, 16–34.
- Ferster, C. B., & DeMyer, M. K. (1961). The development of performances in autistic children in an automatically controlled environment. *Journal of Chronic Diseases, 13*, 312–345.
- Green, G., Brennan, L. C., & Fein, D. (2002). Intensive behavioral treatment for a toddler at high risk for autism. *Behavior Modification, 26*, 69–102.
- Hayward, D. W., Eikeseth, S., Gale, C., & Morgan, S. (2009). Assessing progress during treatment for young children with autism receiving intensive behavioural interventions. *Autism, 13*, 613–633.
- Leaf, R., & McEachin, J. (1999). *A work in progress: Behavior management strategies and a curriculum for intensive behavioral treatment of autism*. New York: DRL Books, L. L. C.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology, 55*, 3–9.

- Lovaas, O. I. (2003). *Teaching individuals with developmental delays: Basic intervention techniques*. Austin: Pro-Ed.
- Lovaas, O. I., & Smith, T. (1989). A comprehensive behavioral theory of autistic children: Paradigm for research and treatment. *Journal of Behavior Therapy and Experimental Psychiatry*, 20, 17–29.
- Lovaas, O. I., Koegel, R. L., Simmons, J. Q., & Long, J. (1973). Some generalization and follow-up measures on autistic children in behavior therapy. *Journal of Applied Behavior Analysis*, 6, 131–166.
- Lovaas, O. I., Ackerman, A., Alexander, D., Firestone, P., Perkins, M., & Young, D. B. (1981). *Teaching developmentally disabled children: The ME Book*. Austin: Pro-Ed.
- Maurice, C. (Ed.), Green, G., & Luce, S. (Co-Eds.). (1996). *Behavioral intervention for young children with autism: A manual for parents and professionals*. Austin: Pro-Ed.
- Maurice, C., Green, G., & Foxx, R. M. (Eds.). (2001). *Making a difference: Behavioral intervention for autism*. Austin: Pro-Ed.
- McEachin, J. J., Smith, T., & Lovaas, O. I. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. *American Journal on Mental Retardation*, 97, 359–372.
- Perry, A., Cummings, A., Dunn Geier, J., Freeman, N. L., Hughes, S., LaRose, L., et al. (2008, epub). Effectiveness of Intensive Behavioral Intervention in a large, community-based program. *Research on Autism Spectrum Disorders*.
- Reed, P., Osborne, L. A., & Corness, M. (2007a). The real-world effectiveness of early teaching interventions for children with autism spectrum disorder. *Exceptional Children*, 73, 417–433.
- Reed, P., Osborne, L. A., & Corness, M. (2007b). Brief report: Relative effectiveness of different home-based behavioral approaches to early teaching intervention. *Journal of Autism and Developmental Disorders*, 37, 1815–1821.
- Reichow, B. (2011). Overview of meta-analysis on early intensive behavioral intervention for young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-011-1218-9>.
- Remington, B., Hastings, R. P., Kovshoff, H., degli Espinosa, F., Jahr, W., Brown, T., et al. (2007). A field effectiveness study of early intensive behavioral intervention: Outcomes for children with autism and their parents after two years. *American Journal on Mental Retardation*, 112, 418–438.
- Sallows, G. O., & Graupner, T. D. (2005). Intensive behavioral treatment for children with autism: Four-year outcome and predictors. *American Journal on Mental Retardation*, 110, 417–438.
- Smith, T., Eikeseth, S., Klevstrand, M., & Lovaas, O. I. (1997). Intensive behavioral treatment for preschoolers with severe mental retardation and pervasive developmental disorder. *American Journal on Mental Retardation*, 102, 238–249.
- Smith, T., Groen, A. D., & Wynn, J. W. (2000). Randomized trial of intensive early intervention for children with pervasive developmental disorder. *American Journal on Mental Retardation*, 105, 269–285.
- Wolf, M. M., Risley, T., & Mees, H. (1964). Application of operant conditioning procedures to the behavior problems of an autistic child. *Behaviour Research and Therapy*, 1, 305–312.

Homework/Assignments, Modifications

► Homework/Assignments, Modifying

Homework/Assignments, Modifying

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Synonyms

[Homework/assignments, modifications](#)

Definition

Changes in the content, instructional level, or performance criteria provided for students with autism. Modifications to homework or in-class assignments are implemented to allow active participation in classroom activities while also ensuring that IEP goals are addressed and successfully accomplished. Common modifications for individuals with ASD include the use of picture symbols and/or visual supports such as social stories; audio and video recordings in place of/in addition to text; shorter or alternate assignments such as outlines or projects in place of lengthy essays or written reports; accessing technology such as calculators, personal digital assistants (PDA), or spell check programs during homework/assignments;

incorporating motivational procedures; alternative assessments; and adjusted grading standards.

See Also

- [Environmental Engineering/Modifications](#)
- [Individual Education Plan](#)
- [Pictorial Cues/Visual Supports \(CR\)](#)

References and Reading

- Adams, L., Gouvousis, A., VanLue, M., & Waldron, C. (2004). Social story intervention: Improving communication skills in a child with autism spectrum disorder. *Focus on Autism & Other Developmental Disabilities*, 19, 87–94.
- Koegel, R., Tran, Q., Mossman, A., & Koegel, L. (2006). Incorporating motivational procedures to improve homework performance. In R. L. Koegel & L. K. Koegel (Eds.), *Pivotal response treatments for autism*. Baltimore: Brookes.
- Myles, B., Ferguson, H., & Hagiwara, T. (2007). Using a personal digital assistant to improve the recording of homework assignments by an adolescent with Asperger syndrome. *Focus on Autism & Other Developmental Disabilities*, 22, 96–99.
- Newman, L. (2007). Secondary school experiences of students with autism. *National Center for Special Education Research Facts from NLTS2*. U.S. Department of Education NCSER, 2007–3005.

Horizant

- [Gabapentin](#)

Hormone

- [Oxytocin](#)

HRB

- [Halstead-Reitan Neuropsychological Test Battery](#)

Hug Machine

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Definition

I developed the hug machine, also called the squeeze machine, to calm my anxiety when I was a teenager. I am a person with autism, and when I entered puberty, I was in a constant state of anxiety and panic attacks. When I visited my aunt's ranch at the age of 15, I observed cattle being handled in a squeeze chute for their vaccination. A squeeze chute is a narrow metal stall that holds the cattle tightly between two metal side panels. Some of the cattle appeared to relax when pressure was applied by the squeeze sides. After I saw this, I tried the squeeze chute and the deep pressure calmed my anxiety. Then I built a squeeze chute-type device that I could get in (Grandin 2006; Grandin and Scariano 1986). It has padded sides that apply pressure on both sides of the body. I got in the squeeze machine on my hands and knees, and the sides applied pressure to the sides of my body. The effect was to have even, deep pressure over a large area of my body. Another feature of the squeeze machine was that I could control the amount of pressure that it applied. This is a really important feature. Even though the pressure was calming, it sometimes becomes overwhelming. When it became too intense, I could release it. At any time, I could stop the pressure.

Sensory Difference

Sensory differences in autism are highly variable. Practical experience and anecdotal evidence both show that some individuals are deep pressure seekers and others are not (Bestbier and Williams 2017). I was a deep pressure seeker. When I was a child, I liked my bed covers tucked in very tight, and I would get under the sofa cushions and ask

my sister to sit on top. The hug machine and other methods for applying deep pressure may have positive effects on some individuals and no effect on others. Further research is definitely needed. Subjects should be chosen to participate in studies depending on whether or not they are a deep pressure seeker. The hug machine and other deep pressure methods are most likely to have a positive effect on individuals who are “deep pressure seekers.”

Research on Deep Pressure

Our first studies on the effects of deep touch pressure assessed the reaction of college students to the hug machine (Grandin 1992). Many students were calmed and relaxed, but a few students felt claustrophobic. It was clear that some people reacted positively and others did not. Edelson et al. (1999) reported that 20 min in the hug machine reduced galvanic skin response in some individuals with autism. Other researches with weighted vests and weighted blankets have reported positive results in some individuals (Fertel-Daly et al. 2001; Mullen et al. 2008). Occupational therapists have found from clinical experiences that deep pressure is most effective if it is applied for 20 min and then taken off. When I used the squeeze machine, I found that the calming effect peaked at 20 min. People working with aggressive dogs have also found that deep pressure over wide areas of the body has a calming effect (Williams and Borchelt 2003). Further studies with dogs showed that a pressure garment reduced heart rate in dogs that were separated from other dogs (King et al. 2014). Studies on sensory-type interventions have been hindered by the vast heterogeneity of individuals with autism. Sensory reactions and sensory oversensitivity are highly variable.

In conclusion, the hug machine or other methods for applying deep pressure may be useful for some individuals and not useful for others. When large numbers of studies are analyzed, the effectiveness of deep pressure is not validated (Losinski et al. 2016). The problem is that deep pressure is only effective for a subgroup of individuals with autism. When all the individuals with an autism label are mixed

together, the pressure responders would be mixed with the pressure non-responders. Deep pressure may be most effective for individuals who actively seek it. Studies are needed that separate the subtypes of sensory problems in autism. The disadvantage of the hug machine is that it is expensive. A smaller sitting design has been developed (Lo and Huang, 2018). Occupational therapists have developed many simpler methods that are easy and economical to use on young children, such as weighted vests, rolling up in gym mats, or laying in tied-together inner tubes (Ayres 1979). The hug machine would be most useful with teenagers and adults because the other methods do not apply sufficient pressure.

References and Reading

- Ayres, J. A. (1979). *Sensory integration and the child*. Los Angeles: Western Psychological Services.
- Bestbier, L., & William, T. I. (2017). The immediate effects of deep pressure on young people with autism and severe intellectual difficulties – Demonstrating individual differences, *Occupational Therapy International* 2017–7534972.
- Edelson, S. M., Edelson, M. G., Kerr, D. C., & Grandin, T. (1999). Behavioral and physiological effects of deep pressure on children with autism: A pilot study evaluating the efficacy of Grandin’s hug machine. *American Journal of Occupational Therapy*, 53, 145–152.
- Fertel-Daly, D., Bedell, G., & Hinojosa, J. (2001). Effects of a weight vest on attention to task and self-stimulatory behaviors in preschoolers with developmental disorders. *American Journal of Occupational Therapy*, 55, 626–640.
- Grandin, T. (1992). Calming effects of deep touch pressure in patients with autistic disorders, college students and animals. *Journal of Child and Adolescent Psychopharmacology*, 2, 63–72.
- Grandin, T. (2006). *Thinking in pictures*. New York: Vintage.
- Grandin, T., & Scariano, M. (1986). *Emergence labeled autistic*. Novato: Arena Press.
- King, C., Buffington, L., Smith, T. J., & Grandin, T. (2014). The effect of pressure wrap (Thundershirt) on the heartrate and behavior of canines diagnosed with anxiety disorder. *Journal of Veterinary Behavior: Clinical Applications and Research*, 9, 215–221.
- Lo, J. S., & Huang, S. L. (2018). Creative design of sitting hug machine in the treatment of students with autism. *MATEC Web of Conferences*, 213, 01009.
- Losinski, M., Sanders, S. A., & Wiseman, N. M. (2016). Examining the use of deep touch pressure to improve

educational performance of students with disabilities. *Research and Practice for Persons with Severe Disabilities*, 4, 3–18.

Mullen, B., Champagne, T., Krishnamurty, S., Dickson, D., & Gao, R. (2008). Exploring the safety and therapeutic effects of deep pressure stimulation using a weighted blanket. *Occupational Therapy in Mental Health*, 24, 65–89.

Williams, N. G., & Borchelt, P. L. (2003). Full body restraint and rapid stimulus exposure as a treatment for dogs, with defensive aggressive behavior: Three core studies. *International Journal of Comparative Psychology*, 16, 226–236.

Human Figure Drawing Tests

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Synonyms

DAP:IQ; Draw-a-person intellectual ability test for children, adolescents, and adults; HFDT

Description

The Draw-A-Person Intellectual Ability Test for Children, Adolescents, and Adults (Reynolds and Hickman 2004) provides one of the most recently standardized scoring systems for calculating an IQ estimate based on human figure drawings. In addition to updated norms, the authors have expanded the age range beyond childhood to include adolescents and adults. The DAP:IQ uses a simple administration procedure and “provides an estimate of cognitive development that is at least an estimate of the lower bound of the person’s ability and does so with a task and scoring criteria that have less cultural specificity than most intelligence tests, verbal or nonverbal” (Examiner’s manual, p. vi).

The DAP:IQ manual states that this test is designed for ages 4 through 89 years and may be administered to individuals or groups. Most individuals complete the drawing in 5 min or less, and

the combination of both administration and scoring is estimated to take 10–12 min. Very few materials are required: the administration/scoring form, the drawing form, two sharpened pencils with erasers, and the Examiner’s manual which includes technical information, normative data, and scoring examples. The DAP:IQ is purported to be appropriate for a wide range of backgrounds and ability levels. While the authors are careful not to say it is “culture-free,” they describe it as “culture-reduced.” However, because it is a drawing task that relies on visual-motor skills, it is obviously not appropriate for individuals with severe motor or visual impairments.

Because the DAP:IQ was devised as a measure of cognitive ability, the authors recommend that examiners have formal training in assessment and knowledge about current theories of cognitive development and neuropsychology. Guidance about the testing environment includes the usual recommendations for freedom from noise and distractions but adds the unique caveat that there should be no large pictures or posters containing human figures in the room that an individual could copy while taking the DAP:IQ.

Task instructions are fairly brief but do require receptive language skills: *I want you to draw a picture of yourself. Be sure to draw your whole body, not just your head, and draw how you look from the front, not from the side. Do not draw a cartoon or stick figure. Draw the very best picture of yourself that you can. Take your time and work carefully. Go ahead* (Examiner’s manual, p. 5).

After the figure is drawn, the examiner assigns points for 23 scoring elements. Scores for each element range from 0 to 3 points. Raw score totals may be converted to a variety of scores: IQ scores ($M = 100$, $SD = 15$), T-scores ($M = 50$; $SD = 10$), z-scores, stanines, percentile ranks, age equivalents, or grade equivalents. The authors used a continuous norming procedure to determine the optimal breakdown of age groupings for the normative tables, with the size of the age groupings ranging from 6-month intervals at 4 through 8 years to 15-year intervals at 75 through 89 years.

The administration/scoring form appears to be well designed and easy to use. The first page

includes space for demographic information, test scores, description of testing conditions, and both individual and group directions. The second and third pages provide written and pictorial guidelines for the scoring criteria for each of the 23 scoring elements.

Historical Background

The DAP:IQ follows a long tradition of using human figure drawings as a means of obtaining information about mental or emotional development. Florence Goodenough (1926) is recognized as the first person to develop a drawing test to measure intelligence in children (Jolley 2010). For her Draw-A-Man Test, children were asked to draw their best picture of a man. This drawing was then scored based on 51 details that pertained to the number of body parts, their proportion, and their attachment to the figure. The points were summed, and the total score was converted to a mental age from which a ratio IQ was calculated (Harris and Pinder 1974). In emphasizing the Draw-A-Man Test's long history, Anastasi (1988) reported that it was "used without change from its original standardization in 1926 until 1963." It was then revised by Dale B. Harris and published as the Goodenough-Harris Drawing Test (1963). The age range was extended to include young adolescents, and three drawings were required rather than just one (i.e., children were asked to draw a picture of a man, a woman, and themselves). Scoring criteria were expanded, and separate norm tables were developed for boys and girls, though the self-drawing was not scored.

A few years after Harris' revision, Koppitz (1968) developed another HFDT based on drawings by nearly 2,000 children between 5 and 12 years of age. Children were only asked to draw one figure, and this was scored according to 30 developmental items derived from the Goodenough-Harris test. Items were grouped by expected, common, not unusual, and exceptional details, and the total score could be converted to an IQ score. In contrast to the previously described HFDTs, Koppitz (1968, 1984) viewed

her drawing test as both a developmental test *and* a projective test.

After another 20 years, Naglieri (1988) published the Draw-a-Person: A Quantitative Scoring System (DAP) which was reported to update and improve the prior scoring systems and expand the norm group. Consistent with the Harris revision, children drew three figures, but the self-drawing was scored, and the three scores were averaged. Rather than having separate norms for boys and girls, Naglieri presented only one conversion table for all three drawings. Consequently, Jolley (2010) characterized the Naglieri DAP as a shorter and simpler alternative to the Goodenough and Goodenough-Harris tests.

When the DAP:IQ appeared in 2004, the manual states that no norming on HFDTs had been done since the late 1980s. Like the original Draw-A-Man Test, examinees are asked to draw only one figure – but this time it is a self-drawing. Reynolds and Hickman (2004) emphasized the conceptual aspects of the drawing to estimate IQ. Their stated goals for the DAP:IQ were to provide updated norms, expand the age range to also include adults, and "reduce the influence of motor skills on the scoring of figure drawings" (Examiner's manual, p. 2).

Psychometric Data

The DAP:IQ was normed on a sample of 3,290 individuals from 46 states. The sample was decreased to 2,295 to closely match the U.S. Bureau of the Census (2001) demographic characteristics in terms of geographic area, gender, race, Hispanic origin, family income, educational attainment of parents, and disability status. A continuous norming procedure was used to develop standard scores for the DAP:IQ. Consequently, the size of the age groupings differs across the life span, and it should be noted that there is wide variability in the number of participants included within the specific age groupings (range: $N = 194$ at age 5 years to $N = 14$ at 75–89 years).

Chapter 5 of the Examiner's manual provides information about the reliability of the DAP:IQ

test scores in terms of content sampling, time sampling, and interscorer reliability. With regard to content sampling, internal consistency reliability of the scoring elements for the entire normative sample was calculated using Cronbach's coefficient alpha. The median alpha reliability was 0.82, with the lowest being 0.74 at age 4. The standard errors of measurement for all 22 age intervals are 4–5 standard score points. Coefficient alphas were also calculated for selected subgroups by gender, racial/ethnic origin, and self-identified handedness, with all of the coefficients falling above 0.73. Evidence for test-retest reliability was based on one small study of 45 individuals ranging from 6 through 57 years of age. They were tested and then retested after a 1-week interval, and the standard scores obtained from the two sessions correlated at 0.84. Interscorer reliability was established by two studies. In the first study, two PRO-ED staff members independently scored 31 drawings completed by individuals 11–75 years of age. In the second study, two PhD graduate students each scored 148 protocols for children from 6 to 11 years. The IQs obtained by these examiners correlated very highly – .95 for the first study and .91 for the second. In an independent study, Williams et al. (2006) supported the reliability results reported in the DAP:IQ manual. They administered the DAP:IQ to a sample of college students aged 19–29 years who were primarily Caucasian and female. Using scores from 110 undergraduate and graduate students to examine internal consistency, they obtained alpha coefficients of 0.82 – which matched the values reported in the DAP:IQ manual. With a smaller subsample of 31 students, the interscorer reliability coefficient was 0.83 which is somewhat lower than the correlation reported in the manual. The intrascorer reliability in this study was 0.92, but this calculation was not reported in the manual, so it is not possible to make a comparison.

Evidence for the validity of the DAP:IQ is provided from several different vantage points in the manual. The authors state that the DAP:IQ is “offered as a measure that estimates overall cognitive ability as represented in the drawing or construction of the human figure” (Examiner's

manual, p. 24). They support the validity of the DAP:IQ by citing historical evidence of the use of human figure drawings by clinicians and researchers for over 100 years. The scoring elements of this current test were generated based on knowledge of the literature “but without reference to any other scoring criteria” (Examiner's manual, p. 25). However, it is unclear how one could be familiar with the previous tests and not be influenced by their scoring systems. After the DAP:IQ scoring criteria were developed, Reynolds and Hickman had them reviewed by PhD-level psychologists, special educators, and measurement specialists affiliated with the PRO-ED research department. Two studies comparing DAP:IQ scores with scores from the Koppitz for children 4–11 years and one study comparing scores with the Goodenough-Harris for children 6–11 years yielded strong correlations of 0.85 to 0.86.

The DAP:IQ was also studied in relation to other frequently administered tests of intelligence, aptitude, and achievement. For example, a correlation of 0.46 was obtained between the DAP:IQ scores and the Full Scale IQ scores from the Wechsler Intelligence Scale for Children – Third Edition (WISC-III) administered to a sample of 211 children between 6 and 11 years. Because the correlation was higher between the DAP:IQ and the performance IQ (0.49) than the verbal IQ (0.33), this was interpreted as supporting the contention that the DAP:IQ is more closely associated with nonverbal as compared to verbal skills. Correlations between the DAP:IQ and other measures (e.g., Detroit Test of Learning Aptitude-Primary: Second Edition, Woodcock Johnson Revised: Tests of Achievement, and Wechsler Individual Achievement Test) were consistent with the WISC-III results in yielding moderate correlations.

Clinical Uses

Despite the time and effort devoted by the authors to design and validate this measure, questions remain about when it is appropriate to administer and for what purpose. The authors

state that the DAP:IQ is designed to “improve the pervasive practice of evaluating human figure drawings as a measure of cognitive ability” (Examiner’s manual, p. v) and further suggest that it is appropriate for large screening efforts, in part because it lacks significant language demands and cultural bias. However, because carefully constructed brief IQ measures that assess a wider array of skills have been developed (e.g., Kaufman Brief Intelligence Test – Second Edition, Reynolds Intellectual Screening Test, and Wechsler Abbreviated Scale of Intelligence) and because the DAP:IQ correlates only moderately with more comprehensive measures of intelligence, aptitude, and achievement, it does not seem advisable to use the DAP:IQ to assess cognitive ability or to make placement decisions. It still may be useful to clinicians who utilize drawings as a means of building rapport or facilitating conversation.

References and Reading

- Abell, S. C., Wood, W., & Liebman, S. J. (2001). Children’s human figure drawings as measures of intelligence: The comparative validity of three scoring systems. *Journal of Psychoeducational Assessment, 19*, 204–215.
- Anastasi, A. (1988). *Psychological testing* (6th ed.). New York: McMillan.
- Cox, M. V. (1993). *Children’s drawings of the human figure*. Hove: Lawrence Erlbaum.
- Goodenough, F. L. (1926). *Measurement of intelligence by drawings*. Yonkers-on-Hudson: World Book.
- Goodenough, F. L., & Harris, D. B. (1963). *The Goodenough-Harris drawing test*. New York: Harcourt, Brace, and World.
- Harris, D. B., & Pinder, G. D. (1974). *The Goodenough-Harris drawing test as a measure of mental maturity of youths*. Rockville: U.S. Department of Health, Education and Welfare.
- Hiltonsmith, R. W. (2010). Review of the draw-a-person intellectual ability test for children, adolescents, and adults. In K. F. Geisinger, R. A. Spies, J. F. Carlson, & B. S. Plake (Eds.), *The seventeenth mental measurements yearbook*. Lincoln: University of Nebraska Press.
- Jolley, R. P. (2010). *Children and pictures: Drawing and understanding*. Chichester: Wiley-Blackwell.
- Kaufman, A. S., & Kaufman, N. L. (2004). *Kaufman brief intelligence test-second edition (KBIT-2)*. Circle Pines: AGS Publishing.
- Koppitz, E. M. (1968). *Psychological evaluation of children’s human figure drawings*. New York: Grune & Stratton.
- Koppitz, E. M. (1984). *Psychological evaluation of human figure drawings by middle school pupils*. New York: Grune & Stratton.
- Naglieri, J. A. (1988). *Draw-a-person: Quantitative scoring system*. San Antonio: The Psychological Corporation.
- Reynolds, C. R., & Hickman, J. A. (2004). *Draw-a-person intellectual ability test for children, adolescents, and adults: Examiner’s manual*. Austin: PRO-ED.
- Reynolds, C. R., & Kamphaus, R. W. (2003). *Reynolds intellectual screening test (RIST)*. Odessa: Psychological Assessment Resources.
- Wechsler, D. (1999). *Wechsler abbreviated scale of intelligence (WASI)*. San Antonio: Harcourt Assessment.
- Williams, T. O., Jr., Fall, A. M., Eaves, R. C., & Woods-Groves, S. (2006). The reliability of scores for the draw-a-person intellectual ability test for children, adolescents, and adults. *Journal of Psychoeducational Assessment, 24*, 137–144.

Human or Animal Motion

► Biological Motion

Hydramine [OTC] [DSC]

► Diphenhydramine

Hydrocortisone

► Cortisol, Serum

Hyperactivity Syndrome

► Serotonin Syndrome

Hyperkinetic Disorders

► Attention Deficit/Hyperactivity Disorder

Hyperlexia

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Synonyms

Reading comprehension disorder

Short Description or Definition

“Hyperlexia” is a term used, most often, to describe skilled word reading, that is, decoding print to speech, in the absence of concomitant reading comprehension ability. Hyperlexia is not a formal diagnosis; thus, the term is used inconsistently to describe a heterogeneous group of individuals. These descriptions most often refer to individuals whose reading decoding skills exceed their reading comprehension skills, usually assessed via a discrepancy in standard scores. However, other descriptions include individuals with ASD who exhibit precocious reading ability across both decoding and comprehension. Consequently, hyperlexia can refer to individuals with above average reading comprehension and even further above average reading decoding skills as well as individuals with poor reading decoding skills and even poorer reading comprehension skills, for example, individuals who can successfully read aloud but may have no functional language and profound intellectual disability. There is no “gold standard” for defining hyperlexia. Nevertheless, the common depiction of an individual with hyperlexia is a child with an autism spectrum disorder, who begins reading words at a very early age without explicit instruction and without any apparent comprehension of the text.

Categorization

The current characterization of hyperlexia, and the operational definition most often used, is an

individual with an autism spectrum disorder who exhibits precocious word-reading skills. As these individuals develop, their comprehension skills almost invariably begin to even out with their decoding skills because, once mastered, word decoding does not get harder. Despite this operational characterization, the field does not have a single cohesive model for why hyperlexia emerges. Prior hyperlexia research has focused on characterizing comprehension deficits in these individuals, concluding that, although advanced, their word-decoding skills are no different from those of typically developing readers (Aram 1997) or that these skills simply represent preserved “cognitive modules” specialized for processing words (O’Conner and Hermelin 1994).

Epidemiology

There are very few papers that attempt to characterize the prevalence of hyperlexia. The most recent and thorough estimate comes from a sample of 80 children with developmental delays (Grigorenko et al. 2002). The paper employed a discrepancy criterion wherein children were categorized as hyperlexic if their reading-decoding scores were two or more standard deviations higher than their IQ scores. Using these criteria, 12 of the 80 children were characterized as hyperlexic. However, as this is only one study with a relatively small sample, further research is required to determine if these results are comparable across different age ranges and populations.

Natural History, Prognostic Factors, and Outcomes

The term “hyperlexia” was first used in the late 1960s by Silberberg and Silberberg (Silberberg 1967) to describe children in the classroom who were able to read printed words but failed to adequately comprehend text. This description distinguished between word decoding, the ability to decode a printed word to speech, and reading comprehension, the ability to garner meaning from printed text. It is important to note both features of this description: (1) Hyperlexia was

not characterized as a strength; these children were identified because of their reading comprehension difficulties. (2) The reading difficulties these children exhibited were not associated with decoding the printed word. As the subsequent 40 years of research showed, many children who struggle with reading do so at the word-decoding level, and these difficulties can, and often do, lead to difficulties in reading comprehension, but what was described in the hyperlexia literature was a reading difficulty at the level of comprehension in the presence of otherwise skilled decoding.

Subsequent hyperlexia research, primarily driven by small samples and case studies, described a heterogeneous group of children. Typically, these children have autism spectrum disorders (ASDs), are early readers, and exhibit difficulty with reading comprehension. Characterizations in the literature broadly range from individuals with ASDs who would otherwise not be expected to read due to age, or severe language or cognitive disability, and individuals with ASDs who exhibit a disparity between their ability to decode and comprehend text but would otherwise be expected to be able to learn to read (Graziani et al. 1983; Nation 1999; Sliberberg 1967). Importantly, the published literature is composed of primarily case reports and small samples from fewer than 100 papers in the last four decades; thus, it is difficult to make strong conclusions regarding the characterization of these individuals.

Successful reading relies on the development of spoken language. Because many individuals with ASD exhibit language delays or dysfunction, it would be expected that they would show similar difficulties with reading, and many do. However, some individuals with ASD and hyperlexia successfully read words despite profound language impairments. Printed words are symbols that represent the sounds of spoken words. Thus, reading develops upon the scaffold of spoken language. This dependence on spoken language highlights the inconsistency of precocious word decoding in children with ASDs, as language and communicative impairments are part of the diagnostic criteria of ASD. Furthermore, it is not the case that children without language delays exhibit hyperlexia; quite the contrary, there are children

with profound language impairments who nonetheless begin to read with no apparent instruction. It is unknown what leads to the development of successful reading in the presence of language dysfunction in some individuals with ASD. Hyperlexia in individuals with language impairments is inconsistent with the developmental trajectory of typical reading, and further research is required to elucidate our understanding of the phenomenon. It has been proposed that hyperlexia is the result of a circumscribed interest in letters and words. This explanation is in line with the clinical presentation of circumscribed interests in ASDs and the obsession with words and letters that some individuals with hyperlexia exhibit. Nevertheless, successful word decoding requires mastery of the speech sounds of the language; this mastery requires attention to spoken language. Thus, theories of hyperlexia as the result of a circumscribed interest must reconcile the mastery of speech sounds, a very social domain, with the clinical expression of ASDs, where social information is often avoided or ignored.

Clinical Expression and Pathophysiology

The clinical expression of hyperlexia is one of heterogeneity. However, many standardized measures for reading have allowed insight into the reading mechanism of these children. First and foremost is that these individuals are decoding and not memorizing words. It had been hypothesized that these children have a savant-like memory for words wherein they memorized entire words as pictures and then named them; however, research has shown this not to be the case. Several studies have shown that these individuals are able to successfully read pseudowords, for example, pronounceable but novel words like “loink” or “ugcrity” (Cardoso-Martins and Da Silva 2008; Newman et al. 2007). Furthermore, tests of memory do not show these individuals to perform better than their typically developing peers. Secondly, while these individuals do exhibit comprehension difficulties, these difficulties are in line with what would be predicted by their IQ and oral language skills; thus, it is not that their reading comprehension performance is unexpectedly low,

but rather, their word-decoding skills are unexpectedly high (Nation 1999; Nation et al. 2006; Nation and Norbury 2005; Pennington et al. 1987). Importantly, as these individuals get older, their age adjusted word-decoding standard scores will get lower and lower. This does not reflect a regression; instead, it reflects that word-decoding skills reach a ceiling of difficulty. As individuals get older, their peers are attaining mastery in word decoding.

For both the caretaker and clinician, it is important to appreciate just how scant the hyperlexia literature is, lest they be guided by out of date, underpowered, and unreplicated research. While the quality of research in hyperlexia is high, it is based on a very small group of children. Since the term was coined in 1967, there are fewer than 100 peer-reviewed articles focusing on hyperlexia. Of these articles, there are multiple review papers, case studies, and instances where the same individual is included in the sample of multiple papers. The latter issue is understandable as children are often seen over multiple occasions by the same clinicians or recruited for multiple studies at the same institution. However, it highlights just how little research there is to base clinical practice on. Finally, there are several papers with provocative titles, claiming that hyperlexia is a marker for positive outcomes in ASDs, or how to accurately define and diagnose hyperlexia, when there is very little data to base these claims on.

Evaluation and Differential Diagnosis

Again, as there is no formal diagnosis of hyperlexia, there is no standardized assessment procedure. However, it can be very useful to have a clear characterization of reading skills in children and individuals in academic settings, especially if they are having reading difficulties. In addition to a clinical characterization for autism spectrum disorders including both cognitive and language abilities, a comprehensive assessment of written language proficiency is warranted. This assessment would include, depending on the age and functioning of the individual, measures of reading decoding, single-word reading, reading

comprehension, reading fluency, phonological processing, and concepts of print. The expected profile from this assessment for an individual with hyperlexia would be one of very strong single-word-decoding skills contrasted by relatively poor reading comprehension skills. Given that individuals with ASDs exhibit a range of communicative and cognitive abilities, it is important that the assessments used during evaluation are chosen to provide an accurate estimate of skill; the particular assessments will vary based on the participant's age and cognitive ability, as well as their native language.

Treatment

Treatment recommendations for reading comprehension difficulties in individuals with hyperlexia combine best practices in reading comprehension interventions, for example, oral language training (Clarke et al. 2010; Snowling and Hulme 2011), and awareness of the specific needs of individuals with ASDs, for example, difficulty with inference, figurative language, and executive functioning. Specifically, direct instruction in reading comprehension strategies includes those used before, during, and after reading a text, including activating background knowledge, previewing vocabulary and content, comprehension monitoring (with embedded stimulus cues), and reviewing text. The use of graphic organizers matched to text types, for example, Venn diagram for cause and effect, is often important for organizing information and providing a visual support for language-based tasks. Finally, for many students with ASDs, a focus on the development of nonfiction reading comprehension and written expression will be important, as these are the text types that will constitute the majority of their academic classes moving forward.

See Also

- [Academic Skills](#)
- [Academic Supports](#)
- [Achievement Testing](#)
- [Case Study](#)
- [Ceiling Effect](#)

- ▶ [Decoding Skills](#)
- ▶ [Diagnostic Instruments in Autistic Spectrum Disorders](#)
- ▶ [Dyslexia](#)
- ▶ [Educational Testing](#)
- ▶ [Floor Effect](#)
- ▶ [High-Functioning Autism \(HFA\)](#)
- ▶ [Intellectual Disability](#)
- ▶ [Language](#)
- ▶ [Language Tests](#)
- ▶ [Literacy](#)
- ▶ [Phonemic Fluency](#)
- ▶ [Phonetics](#)
- ▶ [Phonological Deficits](#)
- ▶ [Phonological Development](#)
- ▶ [Phonological Disorders](#)
- ▶ [Phonology](#)

References and Reading

- Aram, D. (1997). Hyperlexia: Reading without meaning in young children. *Topics in Language Disorders*, 17(3), 1–13.
- Cardoso-Martins, C., & Da Silva, J. (2008). Cognitive and language correlates of hyperlexia: Evidence from children with autism spectrum disorders. *Reading and Writing*, 23, 1–17.
- Clarke, P. J., Snowling, M. J., Truelove, E., & Hulme, C. (2010). Ameliorating children's reading-comprehension difficulties: A randomized controlled trial. *Psychological Science*, 21(8), 1106–1116.
- Graziani, L. J., Brodsky, K., Mason, J. C., & Zager, R. P. (1983). Variability in IQ scores and prognosis of children with hyperlexia. *Journal of the American Academy of Child Psychiatry*, 22(5), 441–443.
- Grigorenko, E. L., Klin, A., Pauls, D. L., Senft, R., Hooper, C., & Volkmar, F. (2002). A descriptive study of hyperlexia in a clinically referred sample of children with developmental delays. *Journal of Autism and Developmental Disorders*, 32(1), 3–12.
- Nation, K. (1999). Reading skills in hyperlexia: A developmental perspective. *Psychological Bulletin*, 125(3), 338–355.
- Nation, K., & Norbury, C. (2005). Why reading comprehension fails: Insights from developmental disorders. *Topics in Language Disorders*, 25(1), 21–32.
- Nation, K., Clarke, P., Wright, B., & Williams, C. (2006). Patterns of reading ability in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 36(7), 911–919.
- Newman, T., Macomber, D., Naples, A., Babitz, T., Volkmar, F., & Grigorenko, E. L. (2007). Hyperlexia in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(4), 760–774.

- O'Connor, N., & Hermelin, B. (1994). 2 Autistic savant readers. *Journal of Autism and Developmental Disorders*, 24(4), 501–515.
- Pennington, B. F., Johnson, C., & Welsh, M. C. (1987). Unexpected reading precocity in a normal preschooler: Implications for hyperlexia. *Brain and Language*, 30(1), 165–180.
- Sliberberg, S. (1967). Hyperlexia—specific word recognition skills in young children. *Exceptional Children*, 34(1), 41–42.
- Snowling, M. J., & Hulme, C. (2011). Evidence-based interventions for reading and language difficulties: Creating a virtuous circle. *The British Journal of Educational Psychology*, 81(Pt 1), 1–23.

Hyperresponsiveness

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Definition

This term, used in the context of sensory processing, refers to the exaggerated responses demonstrated by a person following a sensory experience. The term overresponsiveness is also used in the literature. The nervous system is designed to receive and register incoming sensory information in a graded manner where stronger sensory information is registered at a more intense level. Individuals exhibiting hyperresponsiveness or overresponsiveness demonstrate a heightened state of arousal when seemingly non-noxious or subtle sensory stimuli are presented. An individual may be hyperresponsive to only one form of sensory information such as auditory or tactile or an individual may be hyperresponsive to several types of sensory experiences. An individual with hyperresponsiveness may actively avoid situations that include a specific form of sensory stimulus such as avoiding noisy rooms or an individual may demonstrate unusual behaviors when encountering overwhelming sensory experiences such as grimacing, crying, or becoming agitated as the sensory experience becomes increasingly intense. The hallmark of hyperresponsiveness is an overreaction to a seemingly

subtle or non-noxious sensory experience. Occupational therapy intervention addresses this hyperresponsiveness from several perspectives to allow the individual more opportunities to engage in everyday activities.

References and Reading

- Lane, S. J., Miller, L. J., & Hanft, B. E. (2000). Toward a consensus in terminology in sensory integration theory and practice: Part 2: Sensory integration patterns of function and dysfunction. *Sensory Integration Special Interest Section Quarterly*, 23, 1–3.
- Miller, L. J., Anzalone, M. E., Lane, S. J., Cermak, S. A., & Osten, E. T. (2007). Concept evolution in sensory integration: A proposed nosology for diagnosis. *American Journal of Occupational Therapy*, 61, 135–140.
- Smith Roley, S., Mailloux, Z., Miller-Kuhaneck, H., & Glennon, T. (2007). Understanding Ayres Sensory Integration®, OT Practice September 24 (17), CE-1-CE-8.
- Wilbarger, P., & Wilbarger, J. (1991). *Sensory defensiveness in children aged 2–12: An intervention guide for parents and other caretakers*. Santa Barbara: Avanti Educational Programs.

Hyperserotonemia

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Definition

Serotonin [5-hydroxytryptamine (5-HT)] is a neurotransmitter with effects in the central nervous system and some peripheral organs. It plays a key role as a growth factor in the immature mammalian brain, directing proliferation and maturation (Whitaker-Azmitia 1993). As a result, serotonin has long been implicated in the pathophysiology of autistic disorder (autism), with the first report of

elevated whole-blood serotonin (WBS) levels in patients with autism published in 1961 by Schain and Freedman. WBS levels have been a consistent finding in individuals with autism in a number of research studies, although it is unclear what role serotonin exactly plays, if any, in the pathophysiology of autism (McDougle et al. 2003).

The degree of elevated serotonin, referred to as “hyperserotonemia,” has been defined using various cutoffs. One 1987 paper by Anderson et al. compared the WBS levels of 40 children with autism to that of 87 normal children. The 95th percentile of WBS in the normal group was used to define “hyperserotonemia” (WBS greater than 200 ng/ml), and 38% of autistic individuals were found to be hyperserotonemic. The causal reason for this increase is unknown as these subjects did not appear to represent any identifiable subgroup. Other studies have attempted to study WBS or platelet 5-HT levels in children with autism with regard to age, pubertal development, ethnicity, cognitive impairment, or familial WBS or platelet 5-HT levels, with mixed results (McBride et al. 1998; Croonenberghs et al. 2000; Leboyer et al. 1999; Piven et al. 1991). Overall, many but not all investigations have found elevated WBS levels in prepubertal autistic subjects compared to controls, whereas most normal subjects show an age-related decline of WBS (McDougle et al. 2003). Studies that attempt to probe 5-HT function via endocrine challenges in patients with autism have suggested that 5-HT responsivity may be reduced in autistic subjects compared to controls, particularly in adults. These findings have led researchers to propose that an abnormal maturational process in the 5-HT system may contribute to the pathophysiology of autism. Studies of urinary excretion of 5-HIAA, whole-blood tryptophan concentrations, and baseline levels of cerebrospinal fluid (CSF) 5-HIAA have not found significant differences between autistic subjects and controls. Genetic studies of the 5-HT system in autism have also yielded inconsistent results. It is unclear if WBS levels will prove to be a useful measure in the search for genetic susceptibility to autism, but the concept of elevated WBS as a recurring phenomenon in autism continues to intrigue researchers.

See Also

► [Serotonin](#)

References and Reading

- Anderson, G. M., Freedman, D. X., et al. (1987). Whole blood serotonin in autistic and normal subjects. *Journal of Child Psychology and Psychiatry*, 28(6), 885–900.
- Croonenberghs, J., Delmeire, L., et al. (2000). Peripheral markers of serotonergic and noradrenergic function in post-pubertal, caucasian males with autistic disorder. *Neuropsychopharmacology*, 22(3), 275–283.
- Leboyer, M., Philippe, A., et al. (1999). Whole blood serotonin and plasma beta-endorphin in autistic probands and their first-degree relatives. *Biological Psychiatry*, 45(2), 158–163.
- McBride, P. A., Anderson, G. M., et al. (1998). Effects of diagnosis, race, and puberty on platelet serotonin levels in autism and mental retardation. *Journal of the American Academy of Child and Adolescent Psychiatry*, 37(7), 767–776.
- McDougle, C. J., Posey, D. J., & Potenza, M. N. (2003). Neurobiology of serotonin function in autism. In E. Hollander (Ed.), *Autism spectrum disorders* (pp. 199–220). New York: Marcel Dekker.
- Piven, J., Tsai, G. C., et al. (1991). Platelet serotonin, a possible marker for familial autism. *Journal of Autism and Developmental Disorders*, 21(1), 51–59.
- Schain, R. J., & Freedman, D. X. (1961). Studies on 5-hydroxyindole metabolism in autistic and other mentally retarded children. *The Journal of Pediatrics*, 58, 315–320.
- Whitaker-Azmitia, P. M. (1993). The role of serotonin and serotonin receptors in development of the mammalian nervous system. In I. S. Zagon & P. J. McLaughlin (Eds.), *Receptors in the developing nervous system* (Neurotransmitters) (Vol. 2, pp. 43–53). London: Chapman and Hall.

Hypo-arousal

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Synonyms

[Under-arousal](#)

Definition

Arousal refers to the physiological state of readiness or general state of excitation of one's nervous system. Arousal states lie on a continuum from low to high, and the ability to maintain optimal arousal levels is often required for adaptive interaction with one's environment. Hypo-arousal refers to an arousal state that lies of the low end of this continuum. Behaviorally, hypo-arousal may be observed as under-responsiveness to stimuli and one's environment, for example, as lethargy, inattention, apathy, or boredom.

Theories of hypo-arousal in autism originated in the 1960s, linked directly to the idea of autism-specific deficits in the arousal system per se, specifically implicating the reticular activating system (Rimland 1964). Although general arousal levels have since been found to be normal in persons with autism, there is empirical support for under-arousal in response to specific sensory stimuli in many persons with autism. For example, laboratory data has shown physiological under-responsiveness to auditory, tactile, visual, vestibular, and olfactory stimuli compared to typically developing peers matched on chronological age (Ben-Sasson et al. 2009). However, the evidence base for hypo-arousal and under-responsiveness in autism is limited, due to the paucity and methodological limitations of the available studies (Rogers and Ozonoff 2005). Moreover, results are often inconsistent across studies, possibly reflecting a genuine heterogeneity in patterns of sensory responsivity in individuals with autism.

See Also

- [Hypo-responsiveness](#)
► [Low Registration](#)
► [Sensation-Seeking](#)
► [Sensory Impairment in Autism](#)

References and Reading

- Ben-Sasson, A., Hen, L., Fluss, R., Cermak, S. A., Engel-Yeger, B., & Gal, E. (2009). A meta-analysis of sensory modulation symptoms in individuals with autism

spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 1–11.

- Huebner, R. A., & Dunn, W. (2001). Introduction and basic concepts. In R. A. Huebner (Ed.), *Autism: A sensorimotor approach to management* (pp. 3–40). Austin: Pro-ed.
- Rimland, B. (1964). *Infantile autism*. New York: Appleton.
- Rogers, S. J., & Ozonoff, S. (2005). Annotation: What do we know about sensory dysfunction in autism? A critical review of the empirical evidence. *Journal of Child Psychology and Psychiatry*, 46(12), 1255–1268.

Hypogenesis (Partial Formation)

- [Agenesis of Corpus Callosum](#)

Hypoplasia (Underdevelopment)

- [Agenesis of Corpus Callosum](#)

Hyporesponsiveness

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Definition

Hyporesponsiveness: This term, used in the context of sensory processing, refers to the limited responses demonstrated by a person following a sensory experience. The term underresponsiveness is also used in the literature. The nervous system is designed to receive and register incoming sensory information in a graded manner where stronger sensory information is registered at a more intense level. For individuals who have hyporesponsiveness or underresponsiveness, they may have difficulties detecting novel or pertinent sensory information such as watching a quickly approaching car when attempting to cross a street or controlling the speed of pouring milk from a

carton and spilling the milk by overfilling the cup. Individuals with hyporesponsiveness may appear disinterested in surrounding events due to a limited ability to respond effectively to those events. For individuals with hyporesponsiveness, occupational therapy services are provided to enhance the safety and ability of the individual to engage in everyday activities with better accuracy and efficiency.

References and Reading

- Lane, S. J., Miller, L. J., & Hanft, B. E. (2000). Toward a consensus in terminology in sensory integration theory and practice: Part 2: Sensory integration patterns of function and dysfunction. *Sensory Integration Special Interest Section Quarterly*, 23, 1–3.
- Miller, L. J., Anzalone, M. E., Lane, S. J., Cermak, S. A., & Osten, E. T. (2007). Concept evolution in sensory integration: A proposed nosology for diagnosis. *American Journal of Occupational Therapy*, 61, 135–140.
- Smith Roley, S, Mailloux, Z, Miller-Kuhaneck, H & Glennon, T. (2007). Understanding Ayres Sensory Integration®, OT Practice September 24 (17), CE-1-CE-8.

Hypothalamic-Pituitary-Adrenal Axis

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Definition

The hypothalamic-pituitary-adrenal axis (HPAA) is a chemical messenger system that generates and regulates glucocorticoids (GC). Cortisol, the principal GC in humans, is central for the maintenance of homeostasis, including basic body temperature, autonomic stability, sleep/wake cycles, feeding and thirst, as well as the functioning of higher-order systems (e.g., learning and memory). As such, the HPAA facilitates neurobiological response to all significant internal signals and external stimuli from the environment. It does so

through the sequential release of neurosubstances in the brain structures of the hypothalamus, pituitary, and adrenal glands.

Historical Background

Widely examined in animal and human research, the hypothalamic-pituitary-adrenal axis (HPAA) is best known as the regulator of the “fight-flight” response to threat, diverting energy resources needed for protective actions and promoting a subsequent return to homeostasis when the threat is no longer present. Such a system exists in all vertebrates (Lovejoy 2005). Thus, the HPAA has been a central focus of basic animal research on fear conditioning, general effects of environmental stress on the developing brain, “learned helplessness” models of depression, as well as human studies of anxiety and mood disorders more generally (Friedman et al. 1996).

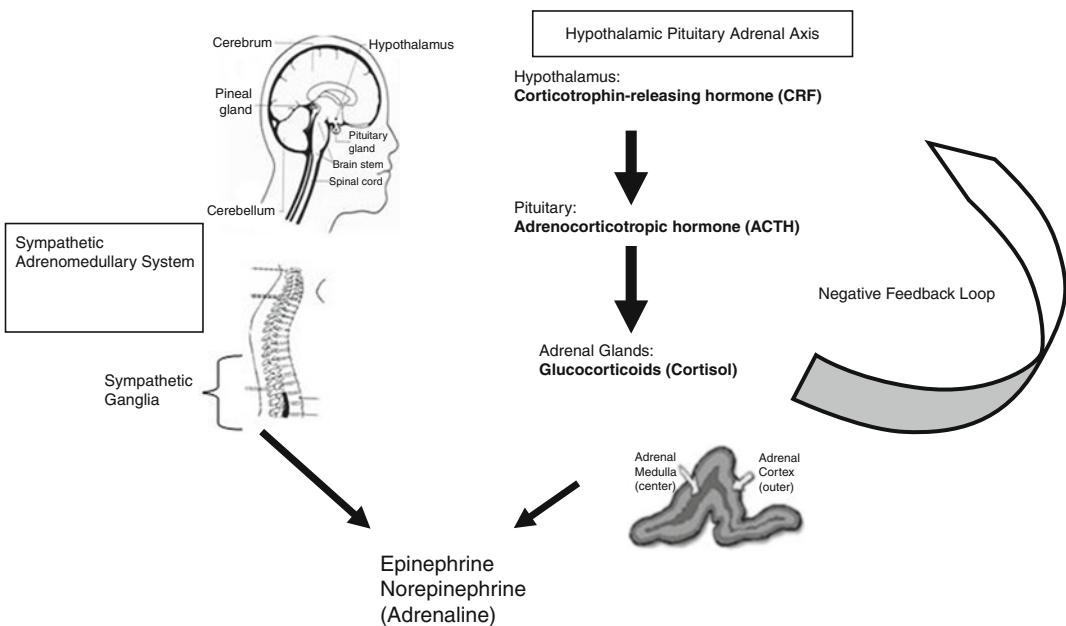
In humans, the perception and interpretation of stimuli occurs within the limbic system, consisting

mainly of the hippocampal formation, amygdala, and entorhinal cortex. When stimuli are identified as potential perturbations to the basic state of equilibrium (i.e., physical or emotional “stressors”), the Sympathetic Adrenomedullary (SAM) system and the HPAA are triggered.

The SAM system responds to threat with *immediate* release of norepinephrine and epinephrine through the sympathetic ganglia of the spinal cord, and the adrenal glands, which sit atop the kidneys.

Release of these catecholamines facilitates processes that increase the heart rate, blood pressure, and blood glucose levels in muscles and vital organs in order to allow the body to carry out defensive actions (Fig. 1).

In contrast, the HPAA responds in a much slower fashion (occurring over several minutes). The hypothalamus releases corticotrophin-releasing hormone (CRH/alternatively “factor” or CRF) to the anterior pituitary. The pituitary response to CRH is to release adrenocorticotrophic hormone (ACTH). In turn, ACTH stimulates



Together, the HPAA and SAM systems are responsible for elevations in catecholamine and cortisol levels in response to acute stress and the HPAA system ensures a return to resting levels through the negative feedback system.

Hypothalamic-Pituitary-Adrenal Axis, Fig. 1 Hypothalamic-Pituitary Adrenal axis

release of cortisol from the adrenal glands. Cortisol feeds back to the hypothalamus to inhibit further release of CRH. Thus, the HPAA represents a “negative feedback” loop by which cortisol release is triggered *and* subsequently inhibited.

Verbal reports and behavioral observations from family, caretakers, and service providers confirm frequent disruptive behaviors in children diagnosed with autism spectrum disorder (ASD). These may include unexplained fear or anxiety, agitation, tantrums, aggression, and resistance to seemingly innocuous directives and situations. Such behaviors are consistent with the HPAA regulated actions of “fight or flight” and are often precipitated by efforts to engage the child in social interactions, elicit communication, or thwart preferred stereotypic movements, activities and routines—all of which can be interpreted as a potential threat to a child with ASD.

However, the HPAA has not been viewed as etiologically relevant to core ASD abnormalities (i.e., social reciprocity, communication and restricted range of behaviors, interests and activities) including (a) repetitive stereotypic behaviors (e.g., hand-flapping and other vestibular movements or lining up objects); (b) reduced or inappropriate affect (including no response to common appetitive and aversive stimuli or a fearful response to certain innocuous sensory stimuli); and (c) preoccupation with a single toy or topic.

The absence of a theoretical model linking the HPAA to ASD may account for the limited number of studies examining HPAA functioning in ASD groups. There is some direct evidence for abnormalities specific to the limbic system which triggers HPAA activation (e.g., the size of the amygdala). There are also a few studies showing abnormal levels (higher *and* lower) of peripheral salivary, urinary or plasma cortisol, and plasma ACTH (Hamza et al. 2010), with greater peak elevations and slow recovery in cortisol reactivity to threat in ASD versus controls (Spratt et al. 2011). The “fractional autism triad” proposed by Happe and Ronald (2008), suggests the three symptom domains used to diagnose ASD may not share the same etiology. Hypothesized

etiologically mechanisms of ASD to date do not account equally well for established knowledge of ASD, including the strong genetic component, male predominance of the disorder, early onset (detectable by the age of two), and frequent overlap with epilepsy and other neurodevelopmental comorbidities. Data indicate none of the symptom domains are primary, universal across all ASD cases, or specific to ASD and not other developmental disorders. With such diversity of symptom patterns, it is perhaps not surprising that HPAA findings in ASD have largely been equivocal.

Current Knowledge

The basic functioning of the HPAA is now known to be considerably more complex than early “fight-flight” models indicated and this complexity may also lead to equivocal findings in ASD. Under normal circumstances, cortisol inhibitory feedback allows for a return to homeostasis after threatening stimuli are no longer present. However, when threat is very intense, frequent, or chronic, the effects of the HPAA on target cells and organs may be prolonged. Cortisol and the catecholamines interact with cell receptors to produce short-term changes in cell processes, as well as changes in gene expression that may have long-lasting consequences for cell function. Over time, these effects can lead to brain changes (structural atrophy, suppressed neurogenesis, and synaptic and dendritic remodeling), and ultimately, poor health outcomes across multiple systems, including cardiovascular, metabolic, and immune systems (McEwen and Gianaros 2011).

Such central neurobiological changes may lead to HPAA dysregulation that is opposite to expected patterns. A counterintuitive *low* level of urinary cortisol was first reported in men diagnosed with Posttraumatic Stress Disorder (PTSD) (Yehuda et al. 1990). PTSD is, by definition, a fear-based psychiatric condition. Diagnostic requirements include (a) exposure to a traumatic event involving perceived threat of death or harm to physical integrity of the self or another person and (b) an acute emotional reaction of fear, helplessness, or horror. Brain

imaging studies confirm distinct neurological areas are activated in PTSD patients who show heightened peripheral SAM system reactivity (e.g., increased heart rate) versus those showing blunted reactivity (Lanius et al. 2010) with an explanatory proposal that there are two forms of PTSD. These and subsequent studies in other psychiatric populations (e.g., Major Depressive Disorder: Ahrens et al. 2008; Panic Disorder: Petrowski et al. 2010) and otherwise healthy individuals experiencing chronic stress (e.g., Juster et al. 2011) demonstrate the bidirectional nature of HPA output at rest and/or in response to new threat. The general direction of HPA dysregulation (hyper- vs. hypo responsive) is thought to depend upon characteristics of the threat (e.g., the intensity, frequency, and chronicity), the timing of assessment relative to the threat (immediate or minutes/hours versus days or months), or to the developmental stage in which the threat occurs (McEwen 2010). Also, the directional impact of cortisol on other neurotransmitters and systems appears to depend upon the perceived anticipatory immediacy of new and recurring threat (Sapolsky et al. 2000). Of note, these are environmental, not genetic factors.

The experiential nature of threat effects on HPA reactivity is demonstrated in a study of monozygotic twins discordant for bullying (one with and one without a victimization history) (Ouellet-Morin et al. 2011). Bullied twins showed a blunted cortisol response to a psychosocial stress test, whereas their non-bullied siblings had a normal cortisol rise. There was no evidence that these group differences were due to genetic makeup, family environment, preexisting or concomitant individual factors, and the perception of stress or the level of subjective emotional responding to the stress test.

Given the heterogeneity of HPA findings among non-ASD populations and the considerable heterogeneity of symptoms in ASD patients specifically, assessment of HPA functioning by currently available measurement methods from peripheral urine, saliva, or blood may not yield clear data. Nonetheless, connections between the hypothalamus and pituitary that are proximal to

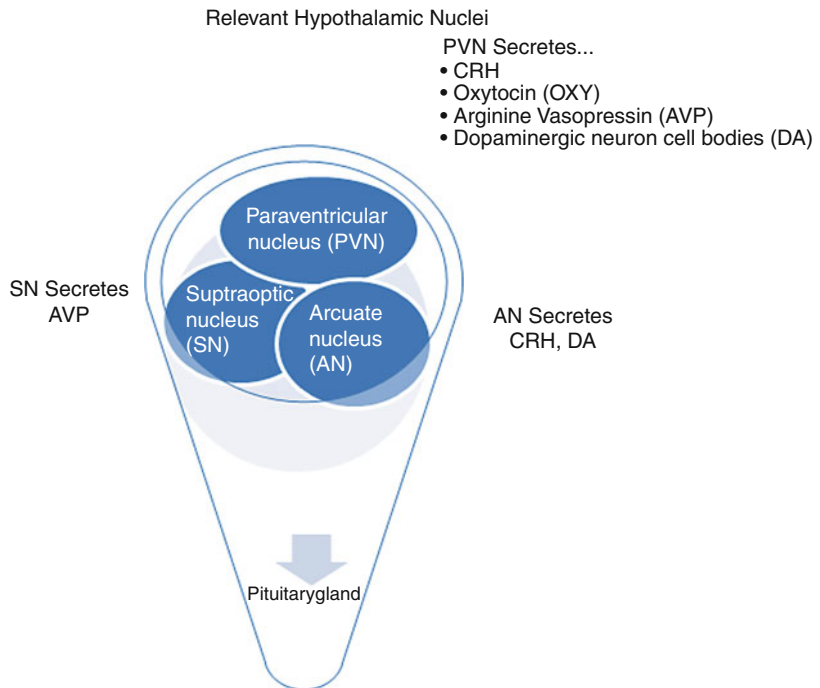
and interact with those of the HPA system (see Gardner and Shoback 2007) have been directly implicated in affiliative behavior relevant for ASD.

The hypothalamus is a cone-shaped brain structure that sits below the thalamus, projecting downward into the pituitary (infundibular) stalk which ends at the roundish-shaped pituitary gland (Afifi and Bergman 1998). CRH is secreted in the paraventricular nucleus (PVN) and the arcuate nucleus (AN) of the hypothalamus. CRH is transported to the anterior pituitary via a system of blood vessels called the *hypophyseal portal system*, where it stimulates release of ACTH. The PVN is also the primary originator of oxytocin (OXY) and one of two hypothalamic nucleus sources of arginine vasopressin (AVP) (known alternatively as vasopressin, argipressin, or antidiuretic hormone (ADH)). [The primary source of AVP is thought to be the supra-optic nucleus.] (Fig. 2).

Cell bodies of OXY and AVP neurons have direct distal axon terminals in the posterior pituitary, and as such, are not neurosecreted via blood vessels like CRH and other hormones traveling into the anterior pituitary. Despite this difference, mutual origination from the PVN and excitatory and inhibitory cross-talk between CRH, OXY and AVP suggest these may be more interrelated than previously thought. For example, recent data indicate AVP potentiates CRF stimulation of ACTH and OXY inhibits release of CRF (van den Burg and Neumann 2011). The role of OXY and AVP in affiliative behavior was recently reviewed by Insel (2010). Like CRH, these neuropeptides originate in the hypothalamus and are carried to the pituitary to facilitate further downstream changes. Stimuli perceived as significant may not only elicit agonistic or avoidance behavior (i.e., fight/flight) and thus CRH, but also approach and affiliation behaviors (“tend and befriend”), which are associated with OXY and AVP (Taylor et al. 2000). Building on this concept, Insel reviews evidence for the social effects of oxytocin and vasopressin within and across species, emphasizing complex interactions among, neuromodulators and species-specific behaviors, including mating, pair-bonding (monogamous or promiscuous),

Hypothalamic-Pituitary-Adrenal Axis,

Fig. 2 Relevant Hypothalamic Nuclei



parental involvement (maternal and paternal), and social structure (solitary or interactive). An interaction between oxytocin and dopaminergic neurotransmission has also been proposed to control social behaviors relevant for ASD (Baskerville and Douglas 2010). Dopaminergic (DA) neuron cell bodies, although not exclusive to this region, are found in the PVN and AN. DA cells project to the anterior pituitary via the tubero-infundibular tract. A recent study of the distribution and morphology of catecholamine elements in the hypothalamus showed them to be almost exclusively dopaminergic synapses (Dudas et al. 2010).

As with bidirectional dysregulation of cortisol in HPAA, behavioral effects of OXY and AVP do not appear to be directly related to levels per se. Instead, receptor location and expression patterns are suggested to be more strongly related to downstream effects (Insel 2010). Current clinical research measurement techniques from peripheral sources (e.g., urine, saliva, and blood) clearly limit the study of central mechanisms occurring within the brain. Nonetheless, the profound social and behavioral deficits seen in ASD may arise from abnormalities in these neuroanatomical areas and corresponding neuroendocrine systems.

Future Directions

Pharmacological and behavioral interventions are known to be effective in dampening the negative sequelae of disruptive behavioral symptoms. Yet, these are likely to be secondary to core domains that temporally precede and act as antecedents to HPAA actions. Whereas the HPAA is likely to play a central role in behavioral symptoms in response to perceived threat, the most parsimonious interpretation of evidence to date is that HPAA abnormalities are secondary consequences of ASD etiology arising from abnormalities in proximal or interrelated areas of the brain.

References and Reading

- Afifi, A. K., & Bergman, R. A. (1998). *Functional neuroanatomy*. New York: McGraw-Hill.
- Ahrens, T., Deuschle, M., Krumm, B., van der Pompe, G., den Boer, J. A., & Lederbogen, F. (2008). Pituitary-adrenal and sympathetic nervous system responses to stress in women remitted from recurrent major depression. *Psychosomatic Medicine*, 70(4), 461–467.
- Baskerville, T. A., & Douglas, A. J. (2010). Dopamine and oxytocin interactions underlying behaviors: Potential contributions to behavioral disorders. *CNS Neuroscience & Therapeutics*, 16, e92–e113.

- Dudas, B., Baker, M., Rotoli, G., Girgnol, G., Bohn, M. C., & Merchenthaler, I. (2010). Distribution and morphology of the catecholaminergic neural elements in the human hypothalamus. *Neuroscience*, 171, 187–195.
- Friedman, M. J., Charney, D. S., & Deutch, A. Y. (Eds.). (1996). *Neurobiological and clinical consequences of stress: From normal adaptation to post-traumatic stress disorder*. Philadelphia: Lippencott-Raven.
- Gardner, D. G., & Shoback, D. (Eds.). (2007). *Greenspan's basic and clinical endocrinology*. New York: McGraw-Hill Companies.
- Green, R. L., & Ostrander, R. L. (2009). *Neuroanatomy for students of behavioral disorders*. New York: W. W. Norton & Company.
- Hamza, R. T., Hewedi, D. H., & Ismail, M. A. (2010). Basal and adrenocorticotrophic hormone stimulated plasma cortisol levels among Egyptian autistic children: Relation to disease severity. *Italian Journal of Pediatrics*, 36(71), 1–6.
- Happé, F., & Ronald, A. (2008). The fractionable autism triad. A review of evidence from behavioural, genetic, cognitive, and neural research. *Neuropsychological Review*, 18, 287–304.
- Insel, T. R. (2010). The challenge of translation in social neuroscience: A review of oxytocin, vasopressin and affiliative behavior. *Neuron*, 65, 768–779.
- Juster, R. P., Marin, M. F., Sindi, S., Nair, N. P., Ng, Y. K., Pruessner, J. C., et al. (2011). A clinical allostatic load index is associated with burnout symptoms and hypocortisolemic profiles in healthy workers. *Psychoneuroendocrinology*, 36(6), 797–805.
- Lanius, R. A., Vermetten, E., Loewenstein, R. J., Brand, B., Schmahl, C., Bremner, J. D., et al. (2010). Emotion modulation in PTSD: Clinical and neurobiological evidence for a dissociative subtype. *American Journal of Psychiatry*, 167, 640–647.
- Lovejoy, D. A. (2005). *Neuroendocrinology: An integrated approach*. Chichester: Wiley.
- McEwen, B. S. (2010). Stress, sex, and neural adaptation to a changing environment: Mechanisms of neuronal remodeling. *Annals of the New York Academy of Science*, 1204(Suppl), E38–E59.
- McEwen, B. S., & Gianaros, P. J. (2011). Stress and allostasis-induced brain plasticity. *Annual Review of Medicine*, 62, 431–445.
- Ouellet-Morin, I., Danese, A., Bowes, L., Shakoob, S., Ambler, A., Pariante, C. M., et al. (2011). Discordant monozygotic twin design shows blunted cortisol reactivity among bullied children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 50(6), 574–582.
- Petrowski, K., Herold, U., Joraschky, P., Wittchen, H. U., & Kirschbaum, C. (2010). A striking pattern of cortisol non-responsiveness to psychosocial stress in patients with panic disorder with concurrent normal cortisol awakening responses. *Psychoneuroendocrinology*, 35(3), 414–421.
- Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory and preparative actions. *Endocrine Reviews*, 21(1), 55–89.
- Spratt, E. G., Nicholas, J. S., Brady, K. T., Carpenter, L. A., Hatcher, C. R., Meekins, K. A., et al. (2011). Enhanced cortisol response to stress in children in autism. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-011-1214-0>. Online 22 Mar 2011.
- Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. A., & Updegraff, J. A. (2000). Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review*, 107, 411–429.
- van den Burg, E. H., & Neumann, I. D. (2011). Bridging the gap between GPCR activation and behaviour: Oxytocin and prolactin signalling in the hypothalamus. *Journal of Molecular Neuroscience*, 43, 200–208.
- Yehuda, R., Southwick, S. M., Nussbaum, G., Waahby, V., Giller, E. L., & Mason, J. W. (1990). Low urinary cortisol excretion in patients with post-traumatic stress disorder. *Journal of Nervous and Mental Disorders*, 178, 366–369.

Hypotonia

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Definition

A state of reduced muscle tension or resistance to movement in a muscle. Hypotonia is not a disorder on its own but a sign or symptom of many different disorders. In infants, hypotonia may be accompanied by delay in motor skills, drooling and speech difficulties, and hyperflexibility of the joints and tendons.

See Also

► [Inborn Errors of Metabolism](#)

References and Reading

- Harris, S. R. (2008). Congenital hypotonia: Clinical and developmental assessment [Review]. *Developmental Medicine and Child Neurology*, 50(12), 889–892.
- Lisi, E. C., & Cohn, R. D. (2011). Genetic evaluation of the pediatric patient with hypotonia: Perspective from a hypotonia specialty clinic and review of the literature. *Developmental Medicine and Child Neurology*, 53(7), 586–599.

IBSE

► Behavior Summarized Evaluation-Revised (BSE-R)

ICD 10 Research Diagnostic Guidelines

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Definition

The International Classification of Mental and Behavioral Disorders – 10th Edition is used by over 70% of the psychiatrists in the world. Although the DSM-IV and ICD-10 definitions for the majority of disorders included in both classifications are quite similar, they are rarely identical, and for a substantial minority of disorders, there are significant differences.

Unlike the DSM-IV definitions of mental disorder which are meant to be used in all settings (i.e., clinical, research, administrative, educational), there are actually three versions of the ICD-10 mental disorders section. The “official” version, the one approved by the World Health Assembly and primarily used by administrators,

includes brief glossary definitions for each of the mental disorders. The ICD-10 Clinical Descriptions and Diagnostic Guidelines (CDDG), commonly known as the “blue book” (because of the color of its cover), is “intended for general clinical, educational, and service use” (ICD-10 CDDG, p. 1) and provides descriptions of the main clinical features for each disorder, accompanied in most cases by diagnostic guidelines, “indicating the number and balance of symptoms usually required before a confident diagnosis can be made” (CDDG, p. 1). The ICD-10 Diagnostic Criteria for Research (DCR), commonly known as the “green book,” is intended primarily for researchers and contains specific diagnostic criteria based on the definitions in the ICD-10 CDDG. These diagnostic criteria were designed to be “deliberately restrictive: their use allows the selection of groups of individuals whose symptoms and other characteristics resemble each other in clearly stated ways” (DCR, p. 1).

A comparison of the diagnostic guidelines and diagnostic criteria for research for a particular ICD-10 disorder usually reveals much greater precision and specificity in the DCR in terms of required minimum and maximum durations and required numbers of symptom features. For example, Table 1 shows the ICD-10 diagnostic guidelines for childhood autism, and Table 2 shows the corresponding diagnostic criteria for research. Although the diagnostic guidelines, like the diagnostic criteria for research, require qualitative impairments in both reciprocal social interaction

ICD 10 Research Diagnostic Guidelines, Table 1 ICD-10 Diagnostic guidelines for childhood autism (ICD-10 CDDG, pp. 253–254) Reprinted by permission of World Health Organization

Usually there is no prior period of unequivocally normal development but, if there is, abnormalities become apparent before the age of 3 years. There are always quantitative impairments in reciprocal social interaction. These take the form of an inadequate appreciation of socio-emotional cues, as shown by a lack of response to other people's emotions and/or a lack of modulation of behavior according to social contexts; poor use of social signals and a weak integration of social, emotional, and communicative behaviors, and especially, a lack of socio-emotional reciprocity. Similarly, quantitative impairments in communication are universal. These take the form of a lack of social usage of whatever language skills are present; impairment in make-believe and social imitative play; poor synchrony and lack of reciprocity in conversational interchange, poor flexibility in language expression and a relative lack of creativity and fantasy in thought processes; lack of emotional response to other people's verbal and nonverbal overtures; impaired use of variations in cadence or emphasis to reflect communicative modulation; and a similar lack of accompanying gesture to provide emphasis or aid meaning in spoken communication.

The condition is also characterized by restricted, repetitive, and stereotyped patterns of behavior, interests, and activities. These take the form of a tendency to impose rigidity and routine on a wide range of aspects of day to day functioning; this usually applies to novel activities as well as to familiar habits and play patterns. In early childhood particularly, there may be specific attachment to unusual, typically non-soft objects. The children may insist on the performance of particular routines in rituals of a nonfunctional character; there may be stereotyped preoccupations with interests such as dates, routes, or timetables; often, there are motor stereotypies; a specific interest in nonfunctional elements of objects (such as their smell or feel) is common; and there may be a resistance to changes in routine or in details of the personal environment (such as the movement of ornaments or furniture in the family home).

and communication, and by the presence of restricted, repetitive, and stereotyped patterns of behavior, interests, and activities, there is no indication about how many of the exemplary symptoms are required for the diagnosis; this judgment is left up to the clinician. In contrast, the diagnostic criteria for research specify that at least six autistic symptoms must be present, with at least two symptoms indicative of a qualitative abnormality in reciprocal social interaction, at least one

ICD 10 Research Diagnostic Guidelines, Table 2 ICD-10 Diagnostic Criteria for Research for F84.0 childhood autism (ICD-10 DCR, 147–149)

A. Abnormal or impaired development is evident before the age of 3 years in at least one of the following areas:

- (1) Receptive or expressive language as used to social communication
- (2) The development of selective attachments or of reciprocal social interactions
- (3) Functional or symbolic play

B. A total of at least six symptoms from (1), (2), and (3) must be present, with at least two from (1) and at least one from each of (2) and (3):

(1) Qualitative abnormalities in reciprocal social interaction are manifest in at least two of the following areas:

- (a) Failure adequately to use eye-to-eye gaze, facial expression, body posture, and gesture to regulate social interaction
- (b) Failure to develop (in a manner appropriate to mental age and despite ample opportunities) peer relationships that involve a mutual sharing of interests, activities, and emotions
- (c) Lack of socio-emotional reciprocity as shown by an impaired or deviant response to other people's emotions; or lack of modulation of behavior according to social context; or a weak integration of social, emotional, and communicative behaviors
- (d) Lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., a lack of showing, bringing, or pointing out to other people objects of interest to the individual)

(2) Qualitative abnormalities in communication are manifest in at least one of the following areas:

- (a) A delay in, or total lack of, development of spoken language that is not accompanied by an attempt to compensate through the use of gesture or mime as an alternative mode of communication (often preceded by a lack of communicative babbling)
- (b) Relative failure to initiate or sustain conversational interchange (at whatever level of language skills is present), in which there is reciprocal responsiveness to the communications of the other person
- (c) Stereotyped and repetitive use of language or idiosyncratic use of words or phrases
- (d) Lack of varied spontaneous make-believe or (when young) social imitative play

(3) Restricted, repetitive, and stereotyped patterns of behavior, interests, and activities are manifest in at least one of the following areas:

- (a) An encompassing preoccupation with one or more stereotyped and restricted patterns of interest that are abnormal in content or focus; or one or more interests that are abnormal in their intensity and circumscribed nature though not in their content or focus

(continued)

ICD 10 Research Diagnostic Guidelines, Table 2
(continued)

(b) Apparently compulsive adherence to specific, non-functional routines or rituals
(c) Stereotyped and repetitive motor mannerisms that involve either hand or finger flapping or twisting, or complex whole body movements
(d) Preoccupations with part-objects or non-functional elements of play materials (such as their odor, the feel of the surface, or the noise or vibration that they generate)
C. The clinical picture is not attributable to other varieties of pervasive developmental disorder, specific developmental disorder of receptive language (F80.2) with secondary socio-emotional problems; reactive attachment disorder (F94.1) or disinhibited attachment disorder (F94.2); mental retardation (F70-F492) with some associated emotional or behavioral disorder; schizophrenia (F20.-) of unusually early onset, and Rett's syndrome (F84.2)

symptom indicative of a qualitative abnormality in communication, and at least one symptom indicative of restricted, repetitive, and stereotyped patterns of behavior.

ICD-10 lists six specific disorders within its pervasive developmental disorders section (F84.0 Childhood autism, F84.1 Atypical autism, F84.2 Rett's syndrome, F84.3 Other childhood disintegrative disorder, F84.4 Overactive disorder associated with mental retardation and stereotyped movements, and F84.5 Asperger's syndrome), and one residual disorder (F84.9 Pervasive developmental disorder, unspecified) for cases that do not meet the criteria for any of the specific pervasive developmental disorders included in this section. Pervasive developmental disorders, according to the ICD-10 CDDG, are "characterized by qualitative abnormalities in reciprocal social interactions, and in patterns of communication, and by restricted, stereotyped, repetitive repertoire of interests and activities" (ICD-10, CDDG, p. 252). Because the category overactive disorder associated with mental retardation and stereotyped movements is noted in the ICD-10 to be "an ill-defined disorder with uncertain nosological validity" and because it resembles the other disorders in this section only by virtue of the presence of stereotyped movements,

it will not be discussed any further. Notably, while DSM-IV includes criteria sets for childhood autism (called "autistic disorder" in the DSM-IV), Rett's syndrome (called Rett's disorder), other childhood disintegrative disorder (called "childhood disintegrative disorder"), and Asperger's syndrome (called "Asperger's disorder"), it does not include a category for atypical autism. All cases of pervasive development disorder not meeting criteria for one of the specific disorders are classified as pervasive developmental disorder not otherwise specified, a category that subsumes the ICD-10 categories of atypical autism and pervasive developmental disorder, unspecified.

Childhood autism (Tables 1 and 2) is characterized by abnormalities in all three areas (i.e., social interaction, communication, range of interests) and are evident prior to age 3. Atypical autism differs from childhood autism in terms either of age of onset or of failure to manifest symptoms from all three of the characteristic areas of abnormalities in autism. Three subtypes are provided: F84.10 (atypicality in age of onset) for cases that meet the symptomatic requirements but for which abnormal or impaired development is evident only after the age of 3 years; F84.11 (atypicality in symptomatology) for cases in which abnormal development is evident before age 3 but for which there are abnormalities in social interactions, communication, or repertoire of interests and activities but not all three; and F84.12 (atypicality in both age of onset and symptomatology) for cases that fail both the age of onset and symptom requirements for autism. The remaining specific ICD-10 pervasive developmental disorders have distinguishing features in addition to abnormalities of social interaction, communication, or repertoire of interests and activities and preempt the diagnoses of childhood autism and atypical autism, i.e., if criteria are met for one of these disorders, childhood autism and atypical autism are not also diagnosed even if criteria were to be met for them.

Rett's syndrome (see Table 3), which appears only in girls, has a very characteristic onset, course, and symptomatology. Apparently normal development for the first 5 months is followed by

ICD 10 Research Diagnostic Guidelines, Table 3 ICD-10 Diagnostic Criteria for Research for F84.2 Rett's syndrome (ICD-10 DCR, p. 151)

A. There is an apparently normal prenatal and perinatal period and apparently normal psychomotor development through the first 5 months and normal head circumference at birth

B. There is deceleration of head growth between 5 months and 4 years and loss of acquired purposeful hand skills between 5 and 30 months of age that is associated with concurrent communication dysfunction and impaired social interaction and the appearance of poorly coordinated/unstable gait and/or trunk movements

C. There is severe impairment of expressive and receptive language, together with severe psychomotor retardation

D. There are stereotyped midline hand movements (such as hand wringing or "hand washing") with an onset at or after the time when purposeful hand movements are lost

deceleration of head growth and loss of previously acquired hand skills accompanied by communication dysfunction and impaired social interaction and the appearance of unstable gait and trunk movements and hand stereotypies. Childhood disintegrative disorder (see Table 4) is distinguished by its requirement that the child have evidence of normal development up to the age of at least 2 years followed by the definite loss of previously acquired skills accompanied by qualitatively abnormal social functioning. Asperger's syndrome (see Table 5) is characterized by the same kind of qualitative abnormalities of reciprocal social interaction and restricted stereotyped interests and activities that are typical of autism, but there is no general delay or retardation in language or cognitive development. Although a diagnosis of atypical autism with atypicality in symptomatology would also apply to such cases (i.e., abnormalities in two out of the three areas required in childhood autism), a diagnosis of atypical autism does not also apply because Asperger's syndrome is given precedence.

Historical Background

Among all the disorders in ICD-10 and DSM-IV, the criteria sets for the pervasive developmental

ICD 10 Research Diagnostic Guidelines, Table 4 ICD-10 Diagnostic Criteria for Research for F84.3 other childhood disintegrative disorder (ICD-10 DCR, pp. 151–152)

A. Development is apparently normal up to the age of at least 2 years. The presence of normal age-appropriate skills in communication, social relationships, play, and adaptive behavior at age 2 years or later is required for diagnosis

B. There is a definite loss of previously acquired skills at about the time of onset of the disorder. The diagnosis requires a clinically significant loss of skills (not just a failure to use them in certain situations) in at least two of the following areas:

(1) Expressive or receptive language

(2) Play

(3) Social skills or adaptive behavior

(4) Bowel or bladder control

(5) Motor skills

C. Qualitatively abnormal social functioning is manifest in at least two of the following areas:

(1) Qualitative abnormalities in reciprocal social interaction (of the type defined by autism)

(2) Qualitative abnormalities in communication (of the type defined for autism)

(3) Restricted, repetitive, and stereotyped patterns of behavior, interests, and activities, including motor stereotypies and mannerisms

(4) A general loss of interest in objects and in the environment

D. The disorder is not attributable to the other varieties of pervasive developmental disorder; acquired aphasia with epilepsy (F80.6); elective mutism (F94.0); Rett's syndrome (F84.2); or schizophrenia (F20.-)

disorders, especially the criteria for childhood autism, are the most closely harmonized. This is a direct result of the DSM-IV field trial for autistic disorders which tested various definitions of autism for potential inclusion into DSM-IV. The multisite field trial involved 125 clinicians with varied levels of experience assessing 977 patients (454 with autism, 240 with other pervasive developmental disorders, and 283 with conditions other than pervasive developmental disorders) using a coding system which compared the DSM-III definition, the DSM-III-R definition, the 1990 draft ICD-10 definition, and a proposed DSM-IV definition derived from the results of literature reviews and data reanalyses in terms of agreement with clinician diagnosis (used as a *ersatz* gold standard) and relative to a diagnostic standard

ICD 10 Research Diagnostic Guidelines, Table 5 ICD-10 Diagnostic Criteria for Research for F84.5 Asperger's syndrome (ICD-10 DCR, pp. 153–154)

- A. There is no clinically significant general delay in spoken or receptive language or cognitive development. Diagnosis requires that single words should have developed by 2 years of age or earlier and that communicative phrases be used by 3 years of age or earlier. Self-help skills, adaptive behavior, and curiosity about the environment during the first 3 years should be at a level consistent with normal intellectual development. However, motor milestones may be somewhat delayed and motor clumsiness is usual (although not a necessary diagnostic feature). Isolated special skills, often related to abnormal preoccupation, are common, but are not required for diagnosis
- B. There are quantitative abnormalities in reciprocal social interaction (criteria as for autism)
- C. The individual exhibits an usually intense, circumscribed interest or restricted, repetitive, and stereotyped patterns of behavior, interests, and activities (criteria as for autism; however, it would be less usual for these to include either motor mannerisms or preoccupations with part-objects or non-functional elements of play materials)
- D. The disorder is not attributable to other varieties of pervasive developmental disorder; simple schizophrenia (F20.6); schizotypal disorder (F21); obsessive-compulsive disorder (F42.-); anankastic personality disorder (F60.5); reactive and disinhibited attachment disorders of childhood (F94.1 and F94.2, respectively)

derived through latent class analysis. Based on these analyses, the draft ICD-10 diagnostic criteria had the highest agreement with clinician diagnoses and the best balance of sensitivity and specificity relative to the latent class standard (i.e., sensitivity = 0.87, specificity = 0.98). The ICD-10 diagnostic criteria also had the highest inter-rater reliability ($\kappa = 0.68$) as compared to DSM-III-R ($\kappa = 0.67$) and DSM-III ($\kappa = 0.45$).

Given the superiority of the ICD-10 diagnostic criteria, it was decided that the DSM-IV criteria should be based on these draft ICD-10 criteria. However, because the draft ICD-10 criteria were considered to be more detailed in terms of number of criteria than was desirable for DSM-IV, criteria were identified for elimination based on reliability and signal detection, resulting in the deletion of four criteria and a revision of the scoring algorithm. Because the ICD-10 criteria also were more

detailed and lengthy than desired for DSM-IV, a series of additional analyses were undertaken, resulting in briefer versions of some of the ICD-10 criteria.

Current Knowledge

In terms of number of items and scoring rules (i.e., requiring 6 items total, at least two from the social interaction group and at least one each from the communication group and restricted interests group), the final ICD-10 and DSM-IV criteria are identical. They differ primarily in the complexity of the wording of some of the items. For example, ICD-10 criterion BI3(c) requires “stereotyped and repetitive motor mannerisms that involve either hand or finger flapping or twisting, or complex whole body movements,” whereas the corresponding DSM-IV item requires simply “stereotyped and repetitive motor mannerisms” and provides as examples “hand or finger flapping or twisting or complex whole body movements.” While these two items may superficially appear to be identical, in actuality, the ICD-10 item is more narrow in that it restricts the types of stereotyped and repetitive motor mannerisms to hand and finger flapping or twisting or complex whole-body movements, whereas DSM-IV allows the clinician to count other stereotyped body movements as well. Comparing the final ICD-10 and DSM-IV criteria sets in a group of 123 subjects randomly selected from 16 of the original field trial sites, diagnostic agreement was excellent ($\kappa = 0.86$), suggesting the research studies using either the DSM-IV or ICD-10 criteria would likely yield the same results.

See Also

► [Comorbidity](#)

References and Reading

<http://www.who.int/classifications/icd/en/>

Iceland and Autism

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Historical Background

When autism was first recognized, when program became available, important historical figures and centers for autism research and service.

Iceland is a rather small island in the North Atlantic Ocean; total size is 103,000 km². Settlers, mainly from Norway, began to arrive in the ninth century (AD). Iceland got independent in 1944 and established a parliamentary democracy (63-members) with a directly elected president as head of state (Iceland 2018). The population grew slowly until the twentieth century (total of 78,000 in the year 1900) but has increased faster in the last 100 years and is at present around 340,000 inhabitants (Hagstofa Íslands 2017). The language spoken is called Icelandic and has its origin in the Old Norse (Iceland 2018). The term “*einhverfa*” is used in Icelandic for the diagnostic term “autism.” This term has been seen in the Icelandic sagas but was first seen related to a psychiatric term in 1939 (Sæmundsen and Guðmundsdóttir 2014). Special educational and intervention services started for a group of children with disabilities in Iceland in the 1970s, along with the development of deinstitutionalization (Bjarnason et al. 2016). A child psychiatric department was first established in 1970, at the National Hospital in Iceland, and around this time professionals were becoming more aware of autism diagnosis. A report of the first child recognized to be diagnosed with autism in Iceland was in 1968. Then there were two girls (2 and 10 years old) diagnosed in 1973 (Sæmundsen and Guðmundsdóttir 2014). At the child psychiatric unit, there were 57 children diagnosed with autism during 1970–1991. The educational services improved for children with disabilities following change in compulsory school act in 1974

and with the development of special educational departments within mainstream schools and with a development of special schools. The State Diagnostic and Counselling Center (SDCC) run by the state (Ministry of Social Affairs) was established in 1986 and in 1997; the center was appointed by the ministry to establish a specialized service around the assessment and intervention for children with autism (Sæmundsen and Guðmundsdóttir 2014). All children and adolescents in Iceland with suspicion of autism were referred to SDCC during the period of more than 10 years. A systematic approach was developed to train professionals to use assessment and diagnostic tools, including ADI-R and ADOS. The methods of early intervention were explored. Two main methods have developed in Iceland, TEACCH and behavioral intervention, based on applied behavior analysis (ABA) (Hjartardóttir and Sigurjónsdóttir 2014). For the last decade due to increased awareness, autism spectrum disorders are diagnosed by other centers in the country as well. The State Diagnostic and Counselling Center still plays a major role to give support and education to parents and professionals working in the area of autism.

Legal Issues, Mandates for Service

Issues related to legal recognition of autism, issues relative to eligibility for support, etc.

Primary health care is generally provided by the state in Iceland. Each region's health-care center provides developmental surveillance until 6 years of age. If there are concerns of developmental growth or symptoms of autism for a child, it might be found during a routine visit or the child might come for extra visits. At each visit the child sees a nurse (n) and for certain visits a doctor (d) as well. The nurse or a midwife meets the child few times and as needed during the post-neonatal period. The first visit at the health-care center is at 6 weeks (n+d). After this scheduled visits are at 9 weeks, (n) 3 (n+d), 5 (n), 6 (n), 8 (n), 10 (n+d), 12 (n), 18 (n+d), 30 (n), and 48 months (n). Parents are often the first to bring up the

worries for their child, so *Parent's Evaluation of Developmental Status* (PEDS) is applied for all children at 18, 30, and 48 months (Directorate of Health 2017a). The *BRIGANCE screen* is applied for all children at 30 and 48 months (Directorate of Health 2017b). In addition large proportion of children in Iceland start play school at a very young age, above 90% of children at 2 years of age (Þorbergsdóttir 2017). Often concerns for the child's developments are brought up by the day care staff. The majorities of play schools are run by the local authorities around the country. Specialist services and support structures should be organized by the local authorities according to special legislations (playschool act, 2008 & regulation 2010). Further assessments are done when the parents agree to do so. The referral for further assessment is usually to the special services provided by the municipality. The local authorities usually have access to clinical psychologists, special educational professional/interventionist, and sometimes a speech and language pathologist or occupational therapist within their team. The team has some resources to do preliminary developmental assessments and screening for autism spectrum symptoms. If the child then needs further diagnostic evaluation, then a referral to a tertiary center, the State Diagnostic and Counseling Center, is made. All referrals are accepted if the child is under 6 years of age with strong suspicion of autism or global developmental delay. All referrals are also accepted for children with significantly limited participation due to autism and/or intellectual disability above 6 years of age (6–18 years). Services for adults with autism might be more fragile. There are teams within the psychiatric units (outpatients) and some private professionals that are providing somewhat limited accessible services.

The directorate of health provides the health-care professionals with some guidelines within the autism. All coding within the health-care system is done with ICD-10 (*International Statistical Classification of Diseases and Health Related Problems, 10th revision*) published by WHO, and the classification of autistic diagnoses is done according to ICD-10 in Iceland (Directorate of Health 2017c). Any child

diagnosed with autism spectrum disorder is able to be supported within the disability services in Iceland (Act on the Affairs of Disabled People 1992).

Overview of Current Treatments and Centers

“Snapshot” of currently prominent and active programs of intervention; its treatment and diagnosis

When the child is diagnosed, the local authorities are obligated to give proper educational services within the mainstream schools (i.e., play schools, compulsory schools). If the child is young and starting play school, parents are encouraged to get involved in choosing the method of early intervention used for their child. As mentioned above ABA and TEACCH are the most prominent methods available in Iceland and used widely at play school level. Almost all municipalities have some experience or trained personal to provide appropriate intervention for the child, but due to increase in ASD diagnoses in recent years, it has become more difficult to meet all diagnosed children with proper quantity of good quality intervention in some areas. The main reason is lack of number of appropriately educated and trained staff at many of the play schools. When the child is at compulsory school age (6–16 years of age), then these specific methods tend not to be the main focus of intervention. The compulsory school intervention is more curriculum educationally driven (special educational needs), rather than able, social, or participation driven. There are no specific centralized intervention centers in Iceland regarding autism specifically. Higher education is widely available (Iceland 2018) in the country and is run directly by the state (Ministry of Education).

Overview of Research Directions

Most significant research programs

Most activities in autism research in Iceland have been done by the State Diagnostic and Counselling Center. Saemundsen's studies have

shown the increasing prevalence in the diagnosis of autism in Iceland in the last decades (Saemundsen et al. 2013). For example, the prevalence of autism in a 1974–1983 birth cohort was 3.8 per 10,000, while it was 8.6 per 10,000 for a younger birth cohort (1984–1993) (Magnússon and Saemundsen 2001). In a study published in 2013, the prevalence of childhood autism was 33.7 per 10,000 (120 per 10,000 for the whole spectrum) in an Icelandic 1994–1998 birth cohort, by the end of 2009 (Saemundsen et al. 2013). From a phenotypic descriptions and genetic analysis, there have been some publications from Icelandic data, some copy number variations (CNV), and some genetic mutations in relation to autism, schizophrenia, and other neurodevelopmental problems (Weiss et al. 2008; Autism Spectrum Disorders Working Group of The Psychiatric Genomics Consortium 2017). There have also been some research activities in cooperation with the University of Iceland, including that children with high-functioning autism rate their quality of life themselves better than their parents (Egilson et al. 2016).

Overview of Training

Ongoing training of providers (teachers, health-care professionals, and others)

The State Diagnostic and Counseling Center provides courses about autism and interventions, for professionals working in the field and for parents (SDCC website 2018).

Social Policy and Current Controversies

Attitudes or social movements with regard to autistic people; debates about autism

The diagnostic practices have been under some focus for few years. Due to increased awareness and observation of the importance of early intervention, there has been improvement in services in general. Many think that there might be some over diagnosis in the field of the autism spectrum disorder. Some think that a driving force is labelling to gain further access to services. This

labelling is also driven by how the funding system is built for the municipalities providing the services. Funding is distributed according to the diagnosis of a child, and the more severe the diagnosis is some more money is provided by a special fund (Jöfnunarsjóður). A recent research exploring this issue has suggested to reduce the emphasis on diagnostic pressure (labelling) and increase early inclusive intervention within the schools in Iceland (European Agency for Special Needs and Inclusive Education 2017).

References and Reading

- Act on the Affairs of Disabled People, No. 59. (1992). Retrieved: https://eng.velferðarraduneyti.is/media/acrobat-enskar_sidur/Act-on-the-Affairs-of-Disabled-People-No-59-1992-with-subsequent-amendments.pdf.
- Autism Spectrum Disorders Working Group of the Psychiatric Genomics Consortium. (2017). Meta-analysis of GWAS of over 16,000 individuals with autism spectrum disorder highlights a novel locus at 10q24.32 and a significant overlap with schizophrenia. *Molecular Autism*, 22(8), 21.
- Bjarnason, D.S., Gunnþórsdóttir, H., & Jónsson Ó.P. (2016). Skóli margbreytileikans. Menntun og manngildi í kjölfar Salamanca. Háskólaútgáfan.
- Directorate of Health. (2017a). Guidelines on child surveillance. PEDS – Mat foreldra á þroska barna. (Parents' Evaluation of Developmental Status). Retrieved from: <https://www.landlaeknir.is/servlet/file/store93/item32585/PEDS%20-%20Mat%20foreldra%20a%20throska%20bama.pdf>.
- Directorate of Health. (2017b). Brigrance þroskaskimun. Retrieved from: <https://www.landlaeknir.is/servlet/file/store93/item32584/Brigrance%20throskaskimun.pdf>.
- Directorate of Health. (2017c). Um flokkunarkerfi (about classification systems). Retrieved at: <https://www.landlaeknir.is/tolfraedi-og-rannsoknir/flokkunarkerfi/>.
- Egilson, S. T., Olafsdóttir, L. B., et al. (2016). Quality of life of high-functioning children and youth with autism spectrum disorder and typically developing peers: Self- and proxy-reports. *Autism*, 21(2), 133–141.
- European Agency for Special Needs and Inclusive Education. (2017). Education for all in Iceland. External audit of the Icelandic system for inclusive education. Retrieved from: https://www.stjornarradid.is/media/menntamalaraduneyti-media/media/frettatengt2016/Final-report_External-Audit-of-the-Icelandic-System-for-Inclusive-Education.pdf.
- Hagstofa Íslands. (2017). Population development 2016. *Statistical Series*, 102(13). Retrieved from <http://www.statice.is/publications/publication-detail?id=58377>.
- Hjartardóttir, S., & Sigurjónsdóttir, M. (2014). Heildstæðar aðferðir. In S. J. Jónsdóttir & E. Sæmundsen (Eds.),

- Litróf einhverfunnar* (pp. 249–264). Iceland: Háskólaútgáfan.
- Iceland. (2018). *Encyclopædia Britannica*. Retrieved from <http://academic.eb.com/levels/collegiate/article/Iceland/106235>.
- Magnússon, P., & Sæmundsen, E. (2001). Prevalence of autism in Iceland. *Journal of Autism and Developmental Disorders*, 31(2), 153–163.
- Play school Act. no. 90/2008 & regulation no. 584/2010. Retrieved from: <https://eng.menntamalaraduneyti.is/media/fretatengt2016/Preschool-Act-No-90-2008.pdf> & <https://www.reglugerd.is/reglugerdir/eftir-raduneytum/mennta-og-menningarmalaraduneyti/nr/16592>.
- Sæmundsen, E., & Guðmundsdóttir, A. K. (2014). *Einhverfa á Íslandi*. In S. J. Jónsdóttir & E. Sæmundsen (Eds.), *Litróf einhverfunnar* (pp. 33–55). Iceland: Háskólaútgáfan.
- Sæmundsen, E., Magnússon, P., et al. (2013). Prevalence of autism spectrum disorders in an Icelandic birth cohort. *BMJ Open*, 20(6), e002748.
- The compulsory school act, no. 63/1974.
- The State Diagnostic & Counseling Center. (2018). Courses and training. Retrieved from: <https://www.greining.is/is/fraedsla-og-namskeid>.
- Þorbergsdóttir, S.Y. (2017). Skýrsla BSRB um dagvistunarræði. Report on daycare services. Retrieved from: https://www.bsrb.is/static/files/Utgefid_efni/skyrsla-bsrb-um-dagvistunarurraedi-mai-2017.pdf.
- Weiss, L. A., Shen, Y., et al. (2008). Association between microdeletion and microduplication at 16p11.2 and autism. *The New England Journal of Medicine*, 14(7), 667–675.

icons has been evaluated relative to transparency and translucency. Transparency refers to how well an icon's referent can be guessed by an unfamiliar user. Translucency refers to the semantic, linguistic, or conceptual relation of icon and its referent. For example, a photo of an object is highly iconic because it is easily recognized and has a strong visual relation with its referent. An abstract line drawing is less iconic because it is less easily recognized by an unfamiliar user and has a more abstract relation with its referent. There is some evidence that icons may be most easily learned when the icon strongly resembles its referent; this is referred to as the iconicity hypothesis. There is evidence that use of icons can be an effective communication strategy for individuals with autism.

See Also

- Iconic Systems
- Picture Exchange Communication System
- Symbol Use
- Visual Schedule
- Visual Supports

Icon

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Synonyms

Symbol

Definition

An icon, or symbol, is a visual representation of a word or linguistic concept. Icons are used in selection-based iconic communication systems (e.g., Picture Exchange Communication Systems, Visual Schedules). The iconicity of

References and Reading

- Bloomberg, K., Karlan, G. R., & Lloyd, L. L. (1990). The comparative translucency of initial lexical items represented in five graphic symbol systems and sets. *Journal of Speech and Hearing Research*, 33, 717–725.
- Bondy, A., & Frost, L. (2002). *A picture's worth: PECS and other visual communication strategies in autism*. Bethesda: Woodbine House.
- Fuller, D. (1997). Initial study into the effects of translucency and complexity on the learning of Blissymbols by children and adults with normal cognitive abilities. *Augmentative and Alternative Communication*, 13, 30–39.
- Koul, R. K., Schlosser, R. W., & Sancibrian, S. (2001). Effects of symbol, referent, and instructional variables on the acquisition of aided and unaided symbols by individuals with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 16, 162–169.
- Mirenda, P., & Erickson, K. (2000). Augmentative communication and literacy. In A. Wetherby & B. Prizant

(Eds.), *Autism spectrum disorders: A transaction development perspective* (pp. 333–367). Baltimore: Paul H. Brookes.

Mizuko, M. (1987). Transparency and ease of learning of symbols represented by Blissymbols, PCS, and Picsyms. *Augmentative and Alternative Communication*, 3, 129–136.

Ogletree, B. T., & Harn, W. E. (2001). Augmentative and alternative communication for persons with Autism: History, issues, and unanswered questions. *Focus on Autism and Other Developmental Disabilities*, 16, 138–140.

Iconic Systems

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Definition

Iconic systems are systems of communication in which icons or symbols are used to represent words or linguistic concepts. Iconic systems are a type of augmentative communication. Iconic systems can be part of a picture exchange system (e.g., PECS) or used in augmentative communication devices. Iconic systems are hypothesized to facilitate communication by a recognition process (e.g., the user recognizes the icon as a picture of the object). In contrast, linguistic systems such as verbal speech involve a recall process (e.g., the user must recall the word that represents that object). Use of iconic systems can be an effective communication strategy for individuals with autism. The concrete and static nature of icons (e.g., a visual symbol has a specific, unchanging referent) and iconic systems (e.g., the system has a set of specific, unchanging rules) may be a reason that iconic systems are effective for individuals with autism. There is evidence that iconic systems can be useful for reducing challenging behaviors in individuals with autism particularly when implemented as part of a communication choice system.

See Also

- ▶ [Picture Exchange Communication System](#)
- ▶ [Symbol Use](#)
- ▶ [Visual Schedule](#)
- ▶ [Visual Supports](#)

References and Reading

- Angermeier, K., Schlosser, R. W., Luiselli, J. K., Harrington, C., & Carter, B. (2008). Effects of iconicity on requesting with the picture exchange communication system in children with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 2, 430–446.
- Beukelman, D. R., & Mirenda, P. (1992). *Augmentative and alternative communication: Management of severe communication disorders in children and adults*. Baltimore: Paul H. Brookes.
- Bloomberg, K., Karlan, G. R., & Lloyd, L. L. (1990). The comparative translucency of initial lexical items represented in five graphic symbol systems and sets. *Journal of Speech and Hearing Research*, 33, 717–725.
- Bondy, A., & Frost, L. (2002). *A picture's worth: PECS and other visual communication strategies in autism*. Bethesda: Woodbine House.
- Fuller, D. (1997). Initial study into the effects of translucency and complexity on the learning of Blissymbols by children and adults with normal cognitive abilities. *Augmentative and Alternative Communication*, 13, 30–39.
- Koegel, R. L., Frea, W. D., & Surratt, A. V. (1994). Self-management as a strategy for modifying problem behavior. In E. Schopler & G. Mesibov (Eds.), *Assessment and management of behavior problems in autism*. New York: Plenum Press.
- Koul, R. K., Schlosser, R. W., & Sancibrian, S. (2001). Effects of symbol, referent, and instructional variables on the acquisition of aided and unaided symbols by individuals with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 16, 162.
- Mirenda, P., & Erickson, K. (2000). Augmentative communication and literacy. In A. M. Weatherby & B. M. Prizant (Eds.), *Autism spectrum disorders: A transactional developmental perspective* (pp. 333–367). Baltimore: Brookes.
- Ogletree, B. T., & Harn, W. E. (2001). Augmentative and alternative communication for persons with autism: History, issues, and unanswered questions. *Focus on Autism and Other Developmental Disabilities*, 16, 138–140.
- Reichle, J. (1991). *Implementing augmentative and alternative communication: Strategies for learners with severe disabilities*. Baltimore: Paul H. Brookes.
- Vaughn, B., & Horner, R. (1995). Effects of concrete versus verbal choice systems on problem behavior. *Augmentative and Alternative Communication*, 11, 89–92.

Identical Twins

- ▶ [Monozygotic \(MZ\) Twins](#)

Idioglossia

- ▶ [Idiosyncratic Language](#)

Idiopathic Toe Walking

- ▶ [Toe Walking](#)

Idiosyncratic Language

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Synonyms

[Idioglossia](#)

Definition

Idiosyncratic language occurs when the child uses standard words or phrases in an unusual, but meaningful way (Volden and Lord 1991). It is a broad term that can refer to a number of speech characteristics that are errors in the pragmatics of communication. A common characteristic of speech in children with Autism Spectrum Disorder (ASD), idiosyncratic language is described as stereotypical and inappropriate word use. These unusual utterances include pedantic speech, in which the child uses overly specific details.

See Also

- ▶ [Linguistic Idiosyncrasies and Neologisms](#)
- ▶ [Metaphoric Language](#)
- ▶ [Pedantic Speech](#)
- ▶ [Pragmatic Communication](#)
- ▶ [Word Use](#)

References and Reading

Volden, J., & Lord, C. (1991). Neologisms and idiosyncratic language in autistic speakers. *Journal of Autism and Developmental Disorders*, 21(2), 109–130.

IEP Matrix: Manuscript

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An Individualized Education Program (IEP) provides the school team with clear and concise goals and objectives to be targeted across an individual student’s school day and week. It is important for all educators (including service providers) to clearly outline, for implementers of educational and behavioral instruction, the setting(s) in which teaching opportunities need to occur based on the student’s IEP. An IEP matrix can assist the school team (including parents and caregivers) in identifying the settings where specific goals and objectives across all educational domains will be targeted for instruction in a manner that is effective and efficient in order to promote learning and generalization of skills of new and/or acquired skills. Providing the implementers of instruction with an IEP matrix will assist in expediting instruction across settings that is informed and systematic. Further, it will assist the school team in identifying any staff training needs that are specific to executing instruction within a particular setting. This may include educators engaging

in the development and careful review of teaching protocols and lesson plans with implementer that is also paired with modeling and the delivery of feedback. In order for an IEP matrix to be effective, the school team must provide sufficient and adequate staff training to ensure that instruction is being implemented with fidelity.

An IEP matrix is often a document that outlines for the implementer(s) where and when a skill will be targeted for instruction. It will specifically identify times of the day, setting, teaching format, activities, and materials necessary for ensuring that instruction occurs in a systematic fashion to promote generalization of skills day long. This may also serve as a reminder to school team members for when and where instruction needs to occur in order to ensure that teaching opportunities are happening in these settings. The content within the IEP matrix will vary depending on the identified direct instruction (e.g., individual or small group instruction) and inclusion time (i.e., time with non-disabled peers) as well as the student's grade level. The matrix is individualized to meet the unique needs of the student with respect to the setting(s) where instruction will be delivered. Therefore, it may include information relative to the student's accommodations, instructional and prompting strategies, and data collection procedures. It is critical that instruction continues to occur across a variety of settings that are outside of the one-on-one setting particularly after the student has acquired a new skill. An IEP matrix is a useful tool for educators to use when programming for generalization of skills as it will help to identify any resources or supports necessary for successful instruction across the school day.

For a parent or caregiver, the IEP matrix provides a visual representation of where a student is receiving instruction specific to their individualized goals and objectives and also identifies the teaching methods used within each of the settings. It may also identify the implementers (e.g., related service providers, special education teacher, general education teacher, and direct-care staff) of instruction for each of the time intervals across the school day and week. The IEP matrix is intended to guide the school team in delivering instruction across settings and should be modified

or adjusted as necessary to include special school activities, changes in implementers of the IEP, and any new instructional strategies identified to be effective. Further, the IEP matrix is intended to help promote learning of skills not restrict learning to certain settings or situations. It is a working document that allows for modifications as the student continues to progress in all areas of their educational program.

IEP Signature Page

► [Notice of Recommended Educational Placement \(NOREP\)](#)

IJA/RJA Skills

► [Initiating/Responding to Joint Attention Skills \(IJA/RJA\)](#)

ILAUGH Model

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Definition

The ILAUGH model is the training component of the “social thinking” intervention framework designed by Michelle Garcia Winner (2000). It is defined as the multiple skills and concepts an individual must process to have successful social interactions and engage in effective social problem solving. The ILAUGH model was created by Winner to explain to parents and professionals the many components of social thinking or what constitutes good problem solving. In addition, the framework was developed to provide an overall model of social-cognitive deficits. The key concepts of the ILAUGH model include:

Initiation of communication
 Listening with your eyes and brain
 Abstract and Inferential Language/Communication
 Understanding perspective
 Gestalt Processing/Getting the big picture
 Humor and human relatedness

The model was specifically designed as an assessment and intervention approach to address the complex social deficits children with high-functioning autism, Asperger syndrome, and non-verbal learning disabilities struggle with during their day-to-day social interactions.

Historical Background

The ILAUGH model is part of Michelle Garcia Winner's social thinking curriculum, and it investigates traits that have been identified as areas of challenge in persons with social-relatedness disorders. Winner (2000) formulated the ILAUGH model as a result of her work in a school while assessing students' needs for social skills and specific aspects of their curriculum. From there, research was used to support the development of the six key concepts noted in the definition. Originally, the ILAUGH model was created to help students address their social communication needs in academic settings. The ILAUGH model provides an understanding of the relationship between problem solving, social interactions, and the ability to respond to specific areas of the academic curriculum (Winner and Crooke 2009). The ILAUGH model also came about as it was realized that a majority of students on the autism spectrum were weak listeners, failed to use eye contact or sustain conversations, and were challenged to understand the perspectives of another (Dunn Buron and Wolfberg 2008). Most recently, the ILAUGH model has been linked to the social thinking-social learning tree (Winner 2012) and serves primarily as the "trunk" of the tree, with the roots of the tree grounded in biological underpinnings of social thinking (joint attention, executive function, central coherence, theory of mind, emotional regulation and reciprocity) and the branches of the tree representing skills that emerge from the

"trunk's" development (reading comprehension, playing with peers, written expression, conversational skills, working in a group). The leaves of the tree's design are the strategies required to complete the branch, in the case of reading comprehension some skills needed are to summarize what one has read and to organize characters and events (Winner 2012). Currently, it is being developed as a research methodology, and a treatment efficacy study has been recently initiated. The ILAUGH model continues to evolve as an understanding of social cognition research expands.

Rationale or Underlying Theory

The key concepts of the ILAUGH model are grounded in research in the area of social relatedness. A summary of each concept and associated research is described below.

- I – Initiation of Communication* (Krantz and McClannahan 1993). Children with ASD have significant challenges in initiating conversations in stressful situations or when information is not easily understood. Even individuals on the autism spectrum with higher cognitive and linguistic abilities have difficulty with communication, in sharp contrast to the interest they display when talking about a favorite topic of interest.
- L – Listening with Your Eyes and Brain* (Jones and Carr 2004; Kuncie and Mesibov 1998; Mundy and Crowson 1997). Many individuals with ASD struggle with auditory comprehension. From a social perspective, however, listening requires much more than our ears; it includes listening with our eyes, which requires a mastery of joint attention. Once children begin to learn that we listen with our eyes, they can be shown the value of nonverbal cues. This can then expand into teaching children to listen with their whole body.
- A – Abstract and Inferential Language/Communication* (Minshew et al. 1992). Most of our communication is not intended for literal comprehension and, instead, is littered with metaphors and idioms – an area of particular

challenge for individuals with ASD. Accurate message interpretation requires an understanding of literal and figurative language.

U – Understanding Perspectives (Baron-Cohen et al. 1997b; Flavell 2004). To understand the perspectives of others, children must have a developed theory of mind (ToM). Whereas typically developing children acquire ToM between the ages of 4 and 6, children with ASD are often delayed in this acquisition, not fully developing ToM until the age of 10. Perspective taking or ToM considers the following abilities: actively considering and adjusting to the thoughts and emotions of one's self as well as the person(s) with whom one is communicating; actively considering and then comparing and contrasting beliefs (e.g., religious, political, and cultural) of one's self as well as the person(s) with whom one is communicating; actively considering and then adjusting one's message given the prior knowledge or experiences of one's communicative partner (s); and actively considering and then adjusting given the motives and intentions of one's self and/or their communicative partner. Winner (2004a) argues that there is a range of perspective taking skills across the autism spectrum.

G – Gestalt Processing/Getting the Bigger Picture (Fullerton et al. 1996; Shah and Frith 1993). Information is conveyed through conceptual processing, not just factual processing. When engaged in a discussion, conversationalists are constantly assessing the root of the conversation instead of assessing each piece as a separate entity. Conceptual processing is a key component in the understanding of academic and social information. Children who have difficulties with conceptual processing often have difficulties with organizational skills, written expression, and time management while being tangential in their expressive skills.

H – Humor and Human Relatedness (Greenspan and Wieder 2003; Gutstein 2001; Prizant et al. 2006a, b). Although many children on the spectrum have a good sense of humor, they struggle with anxiety secondary to missing

social cues that allow them to understand how to have successful interactions with others. Some children on the spectrum have inappropriate humor, and this must also be addressed during intervention. To support human relatedness, it is important to teach students how to relate to their own emotions as well as that of others while helping them understand and experience the happiness or joy in mutual sharing.

Winner (2002) created the following table to establish the rationale behind the ILAUGH framework and how social-cognitive deficits impact social interaction and classroom functioning.

Key concept	How it affects social interactions	How it affects classroom performance
I – poor initiation of communication or action	Student does not initiate appropriate social interactions	Student does not ask for help Student sits and does nothing when others are doing something In class group work, student may not participate or only knows how to direct others; weak negotiator
L – listening with eyes and brain	Student does not observe other's social cues	Student does not observe other's social cues in the classroom
	Student does not process the meaning of their message	Student does not process the meaning of the message in the academic environment
A – abstract and inferential	Student doesn't infer meaning from social cues and has difficulty deciphering meaning from spoken language	Student is limited in the ability to infer meaning from books and teacher's lectures Student literally interprets all materials

(continued)

Key concept	How it affects social interactions	How it affects classroom performance
U – understanding perspective	Student has difficulty recognizing and incorporating other person’s perspectives into self-regulation in social relationships	Student has difficulty understanding the perspective of characters in literature
		Student has difficulty regulating classroom behavior in consideration of others’ needs
G – gestalt processing; getting the big picture	Student has tangential language	Student attends to details but misses the underlying concept of assignments
	Student fails to track the underlying concept of a verbal interaction	Writing can be tangential; the point is often missed
		Student has difficulty staying with the concept of group work and cooperative learning
H – humor	Student usually has a great sense of humor, but may miss the subtleties of humor	Student responds well to a teacher who has a more relaxed, humorous style, but is still able to follow a fairly structured routine
	Student may not understand if they are being laughed at or laughed with	Student may use inappropriate humor in the class as an attempt to engage others

Goals and Objectives

The ILAUGH model is designed to help professionals/families make sense of the challenges that

are seen by those students with social-learning challenges and also to provide direction for professionals to build on a child’s strengths and needs in intervention. For children with social-relatedness disorders, goals include concepts that are within the realm of social cognition and apply directly to each of the key components of the ILAUGH model. The ILAUGH model was designed to explain ways to succeed at social interaction and engage in successful problem solving. A primary goal of the ILAUGH model is to teach children specific language-based concepts that can be used to explain social expectations and related social emotions and thoughts (Winner and Crooke 2009).

Treatment Participants

Participants should be those with social thinking challenges, including individuals with and without diagnostic labels, primarily those with autism spectrum disorders. In practice, Winner implements the model for children and adolescents with high-functioning autism, Asperger syndrome and those with nonverbal learning disabilities.

Treatment Procedures

The ILAUGH framework offers different components to use in the assessment of a child suspected of having deficits in social cognition. Winner (2002)suggests that there are different ways to assess a child using the ILAUGH framework: (a) observe the student with their peers, across different aspects of their environment, (b) relate with the student without facilitating their social success, (c) use informal assessment tools, (d) administer carefully considered standardized measures, and (e) interview teachers and parents about a student’s daily functioning.

As the ILAUGH model is part of Winner’s social cognition program, there are steps that interventionists take during the process. They must focus on making abstract things concrete,

provide a scaffold for language support, foster self-esteem and self-awareness, program in a progressive manner, and provide structured opportunities for generalization along with ongoing practice (Krasney et al. 2003; Winner and Crooke 2009). To address the components in the ILAUGH model, Winner (2000, 2002, 2005, 2007, 2008) recognized the need to create concrete means of teaching abstract social concepts, so she developed language-based concepts titled *social thinking vocabulary* (Winner and Crooke 2009). Social thinking vocabulary includes terms such as “thinking with your eyes,” expected/unexpected behavior, smart guess/wacky guess, and social fake (Winner and Crooke 2009). Other topics included during social cognition intervention include addressing what good social skills are and recognizing that social rules change with age (Winner and Crooke 2009).

Efficacy Information

There is no research currently available on the efficacy of ILAUGH as a treatment, and/or factors affecting treatment. However, there is research to support the efficacy of the conceptual frameworks used to develop the ILAUGH model (as described above). The skills that children develop to support their social skills interactions are the same skills that are needed for a child to work successfully as part of a group in the classroom (Winner 2002). Social skills deficits have been correlated with academic performance deficits, and thus, by improving social skills, academic skills can also improve (Winner 2004b). Strahan (2003) found that among other things, social skills were a significant predictor of GPA among college-level students.

Crooke, Hendrix, and Rachman (2008) examined the effectiveness of teaching social thinking concepts to students with Asperger syndrome and high-functioning autism (HFA). They found that teaching social concepts resulted in a decrease of unexpected nonverbal/verbal social behaviors.

Outcome Measurement

Currently there are no identified outcome measurement tools.

Qualifications of Treatment Providers

There are no suggested criteria for treatment providers; however, Winner states that assessments of individuals with ASD should be conducted by qualified treatment providers who can evaluate a child’s social, communicative, academic, and play needs.

See Also

► [Social Cognition](#)

References and Reading

- Baron-Cohen, S. (1995). *Mindblindness. An essay on autism and theory of mind*. Massachusetts: Bradford Book/MIT Press.
- Baron-Cohen, S. (2000). Theory of mind and autism: A fifteen year review. In S. Baron-Cohen, H. Tager-Flusberg, & D. Cohen (Eds.), *Understanding other minds: Perspectives from developmental cognitive neuroscience*. New York: Oxford University Press.
- Baron-Cohen, S., Baldwin, D. A., & Crowson, M. (1997a). Do children with autism use the speaker’s direction of gaze strategy to crack the code of language? *Child Development*, 68, 48–57.
- Baron-Cohen, S., Jolliffe, T., Mortimore, C., & Robertson, M. (1997b). Another advanced test of theory of mind: Evidence from very high functioning adults with Autism and Asperger Syndrome. *Journal of Child Psychiatry and Psychology*, 38(7), 813–822.
- Crooke, P., Hendrix, R., & Rachman, J. (2008). Measuring the effectiveness of teaching social thinking to children with autism spectrum disorder. *Journal of Autism & Developmental Disorders*, 38(3), 581–591.
- Dunn Buron, K., & Wolfberg, P. J. (Eds.). (2008). *Learners on the autism spectrum: Preparing highly qualified educators*. Shawnee Mission: Autism Asperger Publishing.
- Flavell, J. (2004). Theory-of-mind development: Retrospect and prospect. *Merrill Palmer Quarterly*, 50(3), 274–290.

- Fullerton, A., Stratton, J., Coyne, P., & Gray, C. (1996). *Higher functioning adolescents and young adults with autism*. Austin: Pro-ED.
- Greenspan, S., & Wieder, S. (2003). *Engaging autism: The floortime approach to helping children relate, communicate and think*. Jackson: Perseus Books.
- Gutstein, S. E. (2001). *Autism/Aspergers: Solving the relationship puzzle*. Arlington: Future Horizons.
- Jones, E., & Carr, E. G. (2004). Joint attention in children with autism: Theory and intervention. *Focus on Autism and Other Developmental Disabilities*, 19(1), 13–26.
- Krantz, P., & McClannahan, L. (1993). Teaching children with autism to initiate to peers: Effects of a script-fading procedure. *Journal of Applied Behavior Analysis*, 26, 121–132.
- Krasney, L., Williams, B., Provencal, S., & Ozonoff, S. (2003). Social skills interventions for the autism spectrum: Essential ingredients and a model curriculum. *Child & Adolescent Psychiatric Clinics*, 12, 107–122.
- Kunce, L., & Mesibov, G. (1998). Educational approaches to high-functioning autism and Asperger syndrome (Chapter 11). In E. Schopler, G. Mesibov, & L. Kunce (Eds.), *Asperger syndrome or high-functioning autism?* New York: Plenum Press.
- Minshew, N., Goldstein, G., Muenz, L., & Payton, J. (1992). Neuropsychological functioning in non-mentally retarded autistic individuals. *Journal of Clinical and Experimental Neuropsychology*, 14(5), 749–761.
- Mundy, P., & Crowson, M. (1997). Joint attention and early social communication: Implications for research on intervention with autism. *Journal of Autism and Developmental Disorders*, 27(6), 653–676.
- Prizant, B., Wetherby, A., & Rydell, P. (2000). Communication intervention issues for children with autism spectrum disorders. In A. Wetherby & B. Prizant (Eds.), *Autism spectrum disorders: A transactional developmental perspective* (Vol. 9). Baltimore: Paul H. Brookes.
- Prizant, B., Wetherby, A., Rubin, E., Laurent, A., & Rydell, P. (2006a). *The SCERTS™ model. A comprehensive educational approach for children with autism spectrum disorders* (Program planning and intervention) (Vol. II). Baltimore: Paul H. Brookes.
- Prizant, B., Wetherby, A., Rubin, E., Laurent, A., & Rydell, P. (2006b). *The SCERTS™ model. A comprehensive educational approach for children with autism spectrum disorders* (Assessment) (Vol. I). Baltimore: Paul H. Brookes.
- Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry*, 34(8), 1351–1364.
- Winner, M. (2000). *Inside out: What makes the person with social cognitive deficits tick?* San Jose: Think Social Publishing.
- Winner, M. (2002). Assessment of social skills for students with Asperger syndrome and high-functioning autism. *Assessment for Effective Intervention*, 27, 73–80.
- Winner, M. (2005). *Think social! A social thinking curriculum for school age students*. San Jose: Think Social Publishing.
- Winner, M. (2007). *Thinking about you thinking about me* (2nd ed.). San Jose: Think Social Publishing.
- Winner, M. (2008). The first step of communication, teaching thinking strategies. *Autism Asperger's Digest Magazine*, May–June 2008 issue.
- Winner, M. (2012). *Social thinking*.
- Winner, M., & Crooke, P. (2009). Social Thinking®: A developmental treatment approach for students with social learning/social pragmatic challenges. *Perspectives on Language Learning and Education*, 16(2), 62–69.

Illusion des sosies

► Capgras Syndrome

Imagination

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Synonyms

[Creativity](#); [Generativity](#); [Pretense](#)

Definition

The process of mentally representing concepts that are not immediately, perceptually evident. These concepts include those in the realm of fantasy as well as those in the realm of the non-present reality (e.g., the recent past and the historical past). Imagination skills are evident in symbolic play, perspective taking, problem solving, and other behaviors that require the flexible manipulation of mental representations (see Harris 2000).

In ASD research, imagination in the context of pretend play has received significant attention. Deficits in imaginative play have been useful as a diagnostic marker for ASD, although the interpretation of these deficits has been controversial. One prominent theoretical view holds that the lack of imaginative play indicates a symbolic deficit. From a Piagetian framework, pretend play requires operating from mental representations of previous experiences (Piaget 1962). Leslie (1987) argued that pretense requires the ability to simultaneously hold two representations in mind: the primary (veridical) representation and the imagined representation. This symbolic ability was proposed to be lacking in ASD. This perspective has been challenged by a robust finding of an autism-specific deficit only in the spontaneous generation of pretend play, not in the generation of pretend play in response to elicitation (Lewis and Boucher 1988, 1995). Children with autism generate fewer ideas for pretend acts, with and without props, than controls (Jarrold et al. 1996). However, they are no different from well-matched peers when prompted verbally to perform a pretend act, such as an object substitution, by an adult (Lewis and Boucher 1988). Further, they appear to understand the imagined consequences of an adult's novel, pretend actions (Kavanaugh and Harris 1994). Taken together, children with autism appear able to form and manipulate symbols when suggested by another person but demonstrate a production problem when left to generate pretend acts on their own. An interpretation of these findings is that the pretend play deficit reflects a performance deficit stemming from an autism-specific impairment in generating ideas for play (Jarrold 1997; Jarrold et al. 1996; Lewis and Boucher 1995).

Impairments in generativity may reflect an executive functioning problem that extends beyond the play context, to explain, for example, impairments generating word lists and original ideas for drawings (Lewis and Boucher 1991, 1995). Craig and Baron-Cohen (1999) report diminished performance among school-age children with autism, relative to controls, on relevant tasks that involve generating multiple drawings

from a standard baseline of parallel lines and squiggles, as well as generating ideas for improving a toy, and uses for novel 3-D objects. Additional studies are needed to further understand the interplay of social and cognitive factors underlying diminished imaginative behaviors in autism, in spontaneous pretend play, and in other contexts.

See Also

- Play
- Pretend Play
- Symbolic Play

References and Reading

- Craig, J., & Baron-Cohen, S. (1999). Creativity and imagination in autism and Asperger syndrome. *Journal of Autism and Developmental Disorders*, 29, 319–326.
- Harris, P. L. (2000). *The work of the imagination*. Oxford: Blackwell.
- Jarrold, C. (1997). Pretend play in autism: Executive explanations. In J. Russell (Ed.), *Autism as an executive disorder* (pp. 101–133). Oxford: Oxford University Press.
- Jarrold, C., Boucher, J., & Smith, P. K. (1993). Symbolic play in autism: A review. *Journal of Autism and Developmental Disorders*, 23, 281–307.
- Jarrold, C., Boucher, J., & Smith, P. K. (1996). Generativity deficits in pretend play in autism. *British Journal of Developmental Psychology*, 14, 275–300.
- Kavanaugh, R. D., & Harris, P. L. (1994). Imagining the outcome of pretend transformations: Assessing the competence of normal children and children with autism. *Developmental Psychology*, 30, 847–854.
- Leslie, A. M. (1987). Pretence and representation: The origins of 'theory of mind'. *Psychological Review*, 94, 412–426.
- Lewis, V., & Boucher, J. (1988). Spontaneous, instructed and elicited play in relatively able autistic children. *British Journal of Developmental Psychology*, 6, 325–339.
- Lewis, V., & Boucher, J. (1991). Skill, content and generative strategies in autistic children's drawings. *British Journal of Developmental Psychology*, 9, 393–416.
- Lewis, V., & Boucher, J. (1995). Generativity in the play of young people with autism. *Journal of Autism and Developmental Disorders*, 25, 105–121.
- Piaget, J. (1962). *Play, dreams and imitation in childhood*. London: Routledge & Kegan Paul.

Imipramine

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Synonyms

[Tofranil](#)

Indications

Imipramine is a tricyclic antidepressant. The term tricyclic refers to the three-ring structure of this class of antidepressant medications. These medications are not used as commonly as in the past as they have been largely replaced by the SSRIs. Imipramine has been used to treat both depression and anxiety.

Mechanisms of Action

While desipramine has highly selective norepinephrine reuptake inhibitor properties, and clomipramine is well known for its more selective serotonin reuptake inhibiting properties, imipramine is intermediate with both norepinephrine and serotonin reuptake inhibiting properties.

Clinical Use (Including Side Effects)

The tricyclic antidepressants have several adverse effects in common including dry mouth, urinary retention, constipation, nausea, increased heart rate, dizziness, and, at higher doses, confusion. The tricyclic antidepressants

also carry some risk of altering the electrical conduction in the heart. They are well known to be fatal on overdose due to their potential for causing cardiac arrhythmia. Because of their known toxicity at higher doses, treatment with tricyclic antidepressants requires blood-level monitoring and electrocardiogram monitoring as well. Finally, the tricyclic antidepressants are also vulnerable to drug-drug interaction. For example, some medications such as SSRIs or certain antibiotics may interfere with the breakdown of tricyclic antidepressant medications. The interference of metabolism of the tricyclic can cause a sharp increase in the blood levels of the tricyclic antidepressants and increase the vulnerability to toxic effects. The tricyclic medications have not been well studied in children or adults with autism.

References and Reading

- Martin, A., Scahill, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.
- McDougle, C., & Posey, D. J. (2011). Assessment and treatment of autistic and other pervasive developmental disorders. In A. Martin, L. Scahill, & C. Kratochvil (Eds.), *Pediatric psychopharmacology: Principles and practice* (pp. 547–560). New York: Oxford University Press.

Imitation

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Definition

Imitation is considered to be an integral component of young children's social development. The ability to imitate helps lay the foundation for quality social interactions. Empirical findings exploring early imitative abilities in children have found them to relate to an array of

early childhood skills including: social engagement, nonverbal communication, language development, social understanding, and cognitive skills (Young et al. 2011; Rose et al. 2009; Olineck and Poulin-Dubois 2009; Strid et al. 2006).

Historical Background

Although empirical evidence supports the notion that individuals with ASD, as a group, imitate others with less accuracy and less frequency than their typically developing peers (Vivanti and Hamilton 2014), studies reporting on imitation abilities in individuals with ASD report heterogeneity across individuals (Rogers and Dawson 2010; Salowitz et al. 2012; Vanvuchelen et al. 2011; Vivanti et al. 2011). These differences in imitation abilities seem to be related to differences in social and communicative abilities in this population (Young et al. 2011).

A number of studies have provided evidence suggesting a relationship between imitation and language in children with ASD (Ingersoll and Meyer 2011; McDuffie et al. 2007; Toth et al. 2007). In addition, a relationship also appears to exist between imitation and joint attention (Ingersoll and Schreibman 2006; Rogers et al. 2003), play (Vivanti et al. 2013), social reciprocity (McDuffie et al. 2007; Young et al. 2011), and severity of autism symptomatology (Ingersoll and Meyer 2011; Rogers et al. 2003).

While some studies suggest that imitation is a core deficit of autism spectrum disorder (Rogers and Pennington 1991; Rogers 1999), others contend that there is incomplete evidence to suggest that impairments in imitation are unique and specific to ASD (Williams 2008). As such, although there is empirical evidence to support that children with ASD exhibit deficits in their imitation capabilities, there is not a consensus in the field regarding this matter (e.g., Toth et al. 2006). Regardless of whether or not imitation is a core deficit of autism spectrum disorder, its relationship to other critical early childhood skills distinguish it as a worthy target of early intervention.

Current Knowledge

Intervening on Imitation

Due to its prominent role in the development of social and communication skill, there has been much work done on the development of interventions aimed at improving imitation skills in children with ASD. These interventions report promising findings suggesting improvements in object imitation (Ingersoll and Gergans 2007; Ingersoll and Schreibman 2006), motor imitation (Ingersoll et al. 2006), gesture imitation (Ingersoll et al. 2007), and overall imitation (Ozonoff and Cathcart 1998; Rogers et al. 2005). In addition to improvements in several aspects of imitative abilities, interventions targeting imitation have also been shown to have positive effects on various related skills including imitative language (Ingersoll et al. 2006), expressive language (Young et al. 2011), appropriate play schemes (Ingersoll and Schreibman 2006), joint attention (Ingersoll et al. 2011), combined gestures (Ingersoll et al. 2007), spontaneous gestures (Ingersoll et al. 2007), spontaneous language (Rogers et al. 2005), social communication behaviors (Rogers et al. 2005), as well as fine motor, gross motor, and cognitive performance (Dawson et al. 2010).

Imitation is also used as a pivotal component in early interventions that aim to effect change in core deficits of children with autism. One such example is an intervention that targets joint attention, symbolic play, engagement, and regulation (JASPER) in which adults frequently imitate young children's play acts in order to promote social communication (Kasari and Lawton 2010). Imitation is conceptualized as an important adult strategy for helping children achieve better social communication.

Future Directions

Work in the field still aims to determine the centrality of imitation as a deficit in individuals with ASD, and the ways in which imitation profiles differ between individuals on the spectrum. In addition, there continues to be work done to determine which imitation interventions are most

successful in improving imitation skills of young children with ASD, as well as the ways in which these imitation interventions affect other important social communication skills.

References and Reading

- Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., . . . Varley, J. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The Early Start Denver Model. *Pediatrics*, 125(1), e17–e23.
- Ingersoll, B., & Gergans, S. (2007). The effect of a parent-implemented imitation intervention on spontaneous imitation skills in young children with autism. *Research in Developmental Disabilities*, 28(2), 163–175.
- Ingersoll, B., & Meyer, K. (2011). Examination of correlates of different imitative functions in young children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5(3), 1078–1085.
- Ingersoll, B., & Schreibman, L. (2006). Teaching reciprocal imitation skills to young children with autism using a naturalistic behavioral approach: Effects on language, pretend play, and joint attention. *Journal of Autism and Developmental Disorders*, 36(4), 487.
- Ingersoll, B., Lewis, E., & Kroman, E. (2007). Teaching the imitation and spontaneous use of descriptive gestures in young children with autism using a naturalistic behavioral intervention. *Journal of Autism and Developmental Disorders*, 37(8), 1446–1456.
- Kasari, C., & Lawton, K. (2010). New directions in behavioral treatment of autism spectrum disorders. *Current Opinion in Neurology*, 23(2), 137.
- McDuffie, A., Turner, L., Stone, W., Yoder, P., Wolery, M., & Ulman, T. (2007). Developmental correlates of different types of motor imitation in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(3), 401–412.
- Mumford, K. H. (2014). *The relationship between vocabulary and gesture development in early childhood and infancy*. Doctoral dissertation, University of Birmingham.
- Ozonoff, S., & Cathcart, K. (1998). Effectiveness of a home program intervention for young children with autism. *Journal of Autism and Developmental Disorders*, 28(1), 25–32.
- Rogers, S. J. (1999). An examination of the imitation deficit in autism.
- Rogers, S. J., & Dawson, G. (2010). *Early Start Denver Model for young children with autism: Promoting language, learning, and engagement*. New York: Guilford Press.
- Rogers, S. J., & Pennington, B. F. (1991). A theoretical approach to the deficits in infantile autism. *Development and Psychopathology*, 3(2), 137–162.
- Rogers, S. J., Hepburn, S. L., Stackhouse, T., & Wehner, E. (2003). Imitation performance in toddlers with autism and those with other developmental disorders. *Journal of Child Psychology and Psychiatry*, 44(5), 763–781.
- Rogers, S. J., Cook, I., & Meryl, A. (2005). Imitation and play in autism. In *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 382–405).
- Rose, S. A., Feldman, J. F., & Jankowski, J. J. (2009). A cognitive approach to the development of early language. *Child Development*, 80(1), 134–150.
- Salowitz, N. M., Eccarius, P., Karst, J., Carson, A., Schohl, K., Stevens, S., . . . Scheidt, R. A. (2012). Brief report: Visuo-spatial guidance of movement during gesture imitation and mirror drawing in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(4), 985–995.
- Strid, K., Tjus, T., Smith, L., Meltzoff, A. N., & Heimann, M. (2006). Infant recall memory and communication predicts later cognitive development. *Infant Behavior and Development*, 29(4), 545–553.
- Toth, K., Munson, J. N., Meltzoff, A., & Dawson, G. (2006). Early predictors of communication development in young children with autism spectrum disorder: Joint attention, imitation, and toy play. *Journal of Autism and Developmental Disorders*, 36(8), 993–1005.
- Toth, K., Dawson, G., Meltzoff, A. N., Greenson, J., & Fein, D. (2007). Early social, imitation, play, and language abilities of young non-autistic siblings of children with autism. *Journal of Autism and Developmental Disorders*, 37(1), 145–157.
- Vanvuchelen, M., Roeyers, H., & De Weerd, W. (2011). Imitation assessment and its utility to the diagnosis of autism: Evidence from consecutive clinical preschool referrals for suspected. *Journal of Autism and Developmental Disorders*, 41(4), 484–496.
- Vivanti, G., & Hamilton, A. (2014). Imitation in autism spectrum disorders. In *Handbook of autism and pervasive developmental disorders* (4th ed.).
- Vivanti, G., McCormick, C., Young, G. S., Abucayan, F., Hatt, N., Nadig, A., . . . Rogers, S. J. (2011). Intact and impaired mechanisms of action understanding in autism. *Developmental Psychology*, 47(3), 841.
- Vivanti, G., Dissanayake, C., Zierhut, C., Rogers, S. J., & Victorian ASELCC Team. (2013). Brief report: Predictors of outcomes in the Early Start Denver Model delivered in a group setting. *Journal of Autism and Developmental Disorders*, 43(7), 1717–1724.
- Whalen, C., Schreibman, L., & Ingersoll, B. (2006). The collateral effects of joint attention training on social initiations, positive affect, imitation, and spontaneous speech for young children with autism. *Journal of Autism and Developmental Disorders*, 36(5), 655–664.
- Williams, J. H. (2008). Self–other relations in social development and autism: Multiple roles for mirror neurons and other brain bases. *Autism Research*, 1(2), 73–90.
- Young, G. S., Rogers, S. J., Hutman, T., Rozga, A., Sigman, M., & Ozonoff, S. (2011). Imitation from 12 to 24 months in autism and typical development: A longitudinal Rasch analysis. *Developmental Psychology*, 47(6), 1565.

Imitation of Intransitive Actions

- [Body Movements, Imitation of](#)

Imitation of Nonmeaningful Gestures

- [Body Movements, Imitation of](#)

Imitative Speech

- [Echolalia](#)

Immediate Echolalia

- [Echolalia](#)

Immigrant Status (Epidemiological Studies)

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Definition

An immigrant is a person who leaves his or her country of origin to reside in another country. A refugee is a specific type of immigrant who leaves his or her country of origin due to safety threats or out of fear of persecution.

Historical Background

Typically, studies of immigrant status and autism spectrum disorders (ASDs) have examined whether the occurrence of ASDs is the same for children born to a mother or father who was born in another country (immigrant) as compared to parent-child pairs who were born in the same country (nonimmigrant).

Current Knowledge

Most epidemiologic studies of ASDs have been conducted in Europe, North America, and areas of Asia; there is limited information on the occurrence and characteristics of ASDs in other areas of the world. A few studies have raised speculation about increased occurrence of ASDs among children who had at least one parent who immigrated to the country where the child was born (Gardener et al. 2009 for a review). These studies look at the occurrence of ASDs among subgroups within the country being studied and have not included comparisons of ASDs across countries. For example, a few studies in the UK found that Caribbean-born mothers and fathers residing in the UK were more likely to have a child with an ASD than UK-born parents (Goodman and Richards 1995; Keen et al. 2010; Wing 1980). In addition, mothers born in Africa and Asia were more likely to have children with ASDs in the UK; however, European-born immigrants to the UK showed no association (Keen et al. 2010). Studies from Sweden report increased prevalence of ASDs in the group where either parent was not Swedish-born (Gillberg and Gillberg 1996). They also found increased frequency of ASDs among immigrant groups only in urban, but not rural areas (Gillberg et al. 1987). Increased occurrence of ASDs among children born in Sweden was reported for mothers born outside of Sweden (Hultman et al. 2002), among mothers born in Uganda (Gillberg et al. 1995), and if either parent was of Somali descent (Barnevik-Olsson et al. 2010) as compared to Swedish-born parents. Concern about increased prevalence of ASDs among US-born children with parents of Somali descent has also been raised in the USA

(Minnesota Department of Health [MN] 2009). Among children with ASDs born in Denmark, increased association was found for offspring of mothers born outside of Europe or parents born in different countries (Lauritsen et al. 2005) with one study finding an association for mothers, but not fathers, born outside Denmark (Maimburg and Vaeth 2006). Increased frequency of ASDs in New South Wales was noted for children of mothers born outside of Australia, specifically from Asia (Williams et al. 2008). There has also been some indication that the level of impairment may be more severe among parental immigrant groups (Dealberto 2011). Contrary to the hypothesis that parental immigration increases the chances of having a child with an ASD in the new country of residence, a US study indicated that mothers who immigrated from Mexico to California were less likely to have a child with an ASD than Hispanic mothers born in the United States (Croen et al. 2002).

While many of these studies are suggestive of an association between parental immigration status and having a child with an ASD, small sample sizes limit firm conclusions. There are many challenges in evaluating immigrant status including the potential variation and bias in the way immigration status may be documented. This could impact the ability to classify a child as having an ASD among immigrant families. This could also decrease the validity of data on parental birth location and limit the available data on the total population of immigrants by specific countries of origin or ethnicities needed to make reliable prevalence estimates (MN 2009; Fombonne 2003). In addition, there are many reasons why an individual or family may immigrate to another country. Grouping all immigrants together as a homogeneous group may mask some important differences among subgroups. For example, there may be different sociodemographic, environmental, and possibly biological profiles among groups who immigrate for higher education or sponsored employment from those who immigrate based on refugee status or who are undocumented (i.e., not legally registered in the new country of residence). The larger sociopolitical context is also relevant. For example, administrative prevalence

estimates of ASDs among Hispanic children in California vary based on immigration policy changes (Fountain and Bearman 2011).

Several theories have been proposed to account for a difference in the occurrence of ASDs based on parental immigrant status. These explanations include cultural differences in how the features of ASDs are identified and viewed by parents and professionals among different ethnic groups, which may result in differential identification of ASDs among some groups. In addition, there may be disparities in how children are identified. However, contrary to the findings that minority groups tend to be underidentified with ASDs (CDC 2009; Kogan et al. 2009), higher frequency among select immigrant groups indicates the opposite pattern. For some immigrant groups, it could be that having unique status such as a member of a political refugee group enhances identification of ASDs through linkages to service systems available to this population (MN 2009). While several identification mechanisms have been proposed, it is important to consider whether there is a true etiologic difference caused by biologic factors, exposures, or a combination of the two across immigrant and non-immigrant groups. For example, differences in familial relatedness and differential genetic risk for ASDs or other developmental disabilities have been noted, but not well studied (Barnevik-Olsson et al. 2010; Morrow et al. 2008; Ropers 2010). Another possibility is that there is differential migration of people with ASD-like social deficits, resulting in an increased likelihood of their children having an ASD compared to the general population (Fombonne 2005; Gillberg et al. 1995). Hypotheses of differential environmental experiences among immigrant and nonimmigrant groups have also been raised as possible routes to increased risk for ASDs among these groups. These include increased maternal stress (Gardener et al. 2009), exposure to new infectious agents (Gillberg et al. 1995) or environmental neurotoxins such as pesticides (Fountain and Bearman 2011), and differential vitamin D deficiency among groups with skin or clothing that reduces exposure (Dealberto 2011; Fernell et al. 2010).

Future Directions

Multiple studies suggest an increased occurrence of ASDs among children with at least one immigrant parent as compared to nonimmigrant parents. Additional studies with larger samples in different locations would be helpful both to confirm and better understand this possible association. In addition, data on the prevalence of ASDs in the countries of origin (e.g., in Somalia or areas of the Caribbean) would provide useful comparison data. Further attention to the heterogeneity of immigrant populations and whether specific characteristics apply to a group (e.g., refugee status) would also be helpful. In addition, attention to the quality of the data to include case-finding methods that attempt to identify all individuals in a defined geographic area by relevant characteristics (age group, race/ethnicity, parent birth place, etc.) is important. Also needed is a reliable source for the population denominator (base population of people within that immigrant status group). Finally, more careful examination of biologic and exposure differences between immigrant and non-immigrant groups in association with status of having an ASD could be very useful in identifying unique risk factors for ASDs among these populations.

See Also

- [Epidemiology](#)
- [Incidence](#)
- [Obstetrical Complications/Risk Factors](#)
- [Prevalence](#)
- [Study Design Impact on Prevalence](#)

References and Reading

- Barnevik-Olsson, M., Gillberg, C., & Fernell, E. (2010). Prevalence of autism in children of Somali origin living in Stockholm: Brief report of an at-risk population. *Developmental Medicine and Child Neurology*, 52(12), 1167–1168. <https://doi.org/10.1111/j.1469-8749.2010.03812.x>. Epub 2010 Oct 21.
- Centers for Disease Control and Prevention. (2009). Prevalence of autism spectrum disorders – Autism and developmental disabilities monitoring network, united states, 2006. *Morbidity and Mortality Weekly Report Surveillance Summaries*, 58(10), 1–20.
- Croen, L., Grether, J., & Selvin, S. (2002). Descriptive epidemiology of autism in a California population: Who is at risk? *Journal of Autism and Developmental Disorders*, 32(3), 217–224.
- Dealberto, M. J. (2011). Prevalence of autism according to maternal immigrant status and ethnic origin. *Acta Psychiatrica Scandinavica*, 123(5), 339–348. <https://doi.org/10.1111/j.1600-0447.2010.01662.x>. Epub 2011 Jan 11.
- Durkin, M. S., Maenner, M. J., Meaney, F. J., Levy, S. E., DiGuseppi, C., Nicholas, J. S., et al. (2010). Socioeconomic inequality in the prevalence of autism spectrum disorder: Evidence from a U.S. cross-sectional study. *PLoS One*, 5(7), e11551.
- Fernell, E., Barnevik-Olsson, M., Bågenholm, G., Gillberg, C., Gustafsson, S., & Sääf, M. (2010). Serum levels of 25-hydroxyvitamin D in mothers of Swedish and of Somali origin who have children with and without autism. *Acta Paediatrica*, 99(5), 743–747. Epub 2010 Mar 5.
- Fombonne, E. (2003). Epidemiological surveys of autism and other pervasive developmental disorders: An update. *Journal of Autism and Developmental Disorders*, 33, 365–382.
- Fountain, C., & Bearman, P. (2011). Risk as social context: Immigration policy and autism in California. *Social Forum*, 26(2), 215–240.
- Gardener, H., Spiegelman, D., & Buka, S. L. (2009). Prenatal risk factors for autism: Comprehensive meta-analysis. *British Journal of Psychiatry*, 195(1), 7–14. Review.
- Gillberg, I. C., & Gillberg, C. (1996). Autism in immigrants: A population-based study from Swedish rural and urban areas. *Journal of Intellectual Disability Research*, 40(Pt 1), 24–31.
- Gillberg, C., Steffenburg, S., Börjesson, B., & Andersson, L. (1987). Infantile autism in children of immigrant parents. A population-based study from Göteborg, Sweden. *British Journal of Psychiatry*, 150, 856–858.
- Gillberg, C., Schaumann, H., & Gillberg, I. C. (1995). Autism in immigrants: Children born in Sweden to mothers born in Uganda. *Journal of Intellectual Disability Research*, 39(Pt 2), 141–144.
- Goodman, R., & Richards, H. (1995). Child and adolescent psychiatric presentations of second-generation Afro-Caribbeans in Britain. *The British Journal of Psychiatry*, 167(3), 362–369.
- Hultman, C. M., Sparén, P., & Cnattingius, S. (2002). Perinatal risk factors for infantile autism. *Epidemiology*, 13(4), 417–423.
- Keen, D. V., Reid, F. D., & Arnone, D. (2010). Autism, ethnicity and maternal immigration. *The British Journal of Psychiatry*, 196(4), 274–281.
- Kogan, M., Blumberg, S., Schieve, L., Boyle, C., Perrin, J., et al. (2009). Prevalence of parent-reported diagnosis of

- autism spectrum disorder among children in the U.S., 2007. *Pediatrics*, 124(5), 1395–1403.
- Lauritsen, M. B., Pedersen, C. B., & Mortensen, P. B. (2005). Effects of familial risk factors and place of birth on the risk of autism: A nationwide register-based study. *Journal of Child Psychology and Psychiatry*, 46(9), 963–971.
- Maimburg, R. D., & Vaeth, M. (2006). Perinatal risk factors and infantile autism. *Acta Psychiatrica Scandinavica*, 114(4), 257–264.
- Minnesota Department of Health. (2009). Autism spectrum disorders among preschool children participating in the Minneapolis Public Schools early childhood special education programs. <http://www.health.state.mn.us/ommh/projects/autism/index.cfm>
- Morrow, E. M., Yoo, S. Y., Flavell, S. W., Kim, T. K., Lin, Y., Hill, R. S., et al. (2008). Identifying autism loci and genes by tracing recent shared ancestry. *Science*, 321(5886), 218–223.
- Ropers, H. H. (2010). Genetics of early onset cognitive impairment. *Annual Review of Genomics and Human Genetics*, 11, 161–187.
- Williams, K., Helmer, M., Duncan, G. W., Peat, J. K., & Mellis, C. M. (2008). Perinatal and maternal risk factors for autism spectrum disorders in New South Wales, Australia. *Child: Care, Health and Development*, 34(2), 249–256.
- Wing, L. (1980). Childhood autism and social class: A question of selection. *The British Journal of Psychiatry*, 137(410), 417.

Impact of Race, Ethnicity, and Gender on Social and Behavioral Ratings Within the ADOS

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Definition

At present, diagnosing autism spectrum disorder (ASD) relies on the identification of symptoms by comparing clinician-observed and parent-reported behaviors to typical and atypical exemplars. Historically these examples reflect behaviors from the most highly researched groups – White, Western, and males (Hilton et al. 2010). Using reference behaviors based on

majority groups discounts the dynamic nature of cultural and social behavior and can inadvertently lead to culture-based biases in ASD measures assessing social constructs. Research has documented cross-cultural variability in a range of behaviors central to ASD diagnosis.

Distinct values about eye contact and varied interpretation guidelines among cultures (i.e., individualist and collectivist countries) result in different gaze topography. The extent of sustained eye contact and regularity of eye contact between adults and children also varies between racial groups in the USA (Fugita et al. 1974). Similarly, cross-cultural differences exist in the amount and type of play, particularly in child and adult dyads (Lancy 2007). Both internationally and among distinct US racial and gender groups, play differences reflect cultural variability in sociocultural gender roles (Lai et al. 2015) and in valuation of attributes like self-assertion (Kochman 1981).

Culture also impacts emotion expression and recognition. Cross-cultural variability in emotion recognition (Elfenbein et al. 2007) is attributable to using unique facial expressions to represent basic emotions across cultures and different facial viewing patterns (i.e., holistic patterns in collectivistic cultures and analytic patterns in individualistic cultures; Masuda and Nisbett 2001). Culture can also impact how different facial features are “weighted” during face processing (Yuki et al. 2007). Culture also impacts nonverbal facial cues used to express emotions, referred to as “nonverbal accents.” Thus, people demonstrate an in-group effect where they recognize emotions from their own culture more accurately (Marsh 2003). Different levels of expressivity between racial groups and gender in the USA (Vrana and Rollock 2002) might reflect distinct values placed on spontaneous expression of feelings (McClure 2000).

Aspects of language with cultural underpinnings, such as pragmatic use, nonverbal communication, and speech patterns are embedded in both social and repetitive/restrictive ASD symptom clusters. Cultures weight verbal and nonverbal messages differently depending on their reliance on context (Knapp et al. 2013) and have differing styles for proximity, volume, and other nonverbal conversational cues (Collett

1971). Cultural variability in pragmatic language impacts important aspects of conversing (i.e., turn-taking, interrupting, humor), as well as frequency of conversation between children and adults (Carter et al. 2005). Cultural variability is also documented in pronoun usage among bilingual individuals (Carter et al. 2005) and grammatical patterns between different US racial groups depending on the English dialect spoken at home (Hall and Freedle 1975).

A failure to account for known variability in these behaviors fundamental to ASD diagnostic assessment could result in meaningful under- or over-pathologizing of behavioral deviations from classic exemplars. One study examined a subset of items from one widely used observation-based ASD diagnostic tool to determine if racial, ethnic, or gender measurement biases existed. After holding overall symptom levels constant, results revealed modest biases in items assessing eye contact, stereotyped language use, and echolalia for racial and ethnic minority groups (Harrison et al. 2017). Although the amount of bias in the sample was small, results highlight the diminished accuracy that could arise through a reliance on narrow, culturally bound definitions of what constitutes typical behaviors and arbitrary clinical thresholds. The tendency to rely on one presentation of ASD is linked to inaccurate identification of autistic females (Kirkovski et al. 2013). Cultural variability in caregiver perception of behavior and personal values can also influence endorsement of problems and, thus, diagnostic outcomes. Taken together, these cultural factors could impact ASD prevalence. Increasing clinician awareness of cultural differences in child and parent behaviors and utilizing rigorous transadaptation strategies for assessments are critical strategies for buffering diagnostic measurement bias.

See Also

- Clinical Assessment
- Culture and Autism
- Diagnostic Process
- Measurement Error
- Test Bias

References and Reading

- Carter, J. A., Lees, J. A., Murira, G. M., Gona, J., Neville, B. G., & Newton, C. R. (2005). Issues in the development of cross-cultural assessments of speech and language for children. *International Journal of Language & Communication Disorders*, 40(4), 385–401. <https://doi.org/10.1080/13682820500057301>.
- Collett, P. (1971). Training Englishmen in the non-verbal behaviour of Arabs. *International Journal of Psychology*, 6(3), 209–215. <https://doi.org/10.1080/00207597108246684>.
- Elfenbein, H. A., Beaupré, M., Lévesque, M., & Hess, U. (2007). Toward a dialect theory: Cultural differences in the expression and recognition of posed facial expressions. *Emotion*, 7(1), 131. <https://doi.org/10.1037/1528-3542.7.1.131>.
- Fugita, S. S., Wexley, K. N., & Hillery, J. M. (1974). Black-White differences in nonverbal behavior in an interview setting. *Journal of Applied Social Psychology*, 4(4), 343–350. <https://doi.org/10.1111/j.1559-1816.1974.tb02606.x>.
- Hall, W. S., & Freedle, R. O. (1975). *Culture and language: The black American experience*. Washington, DC: Hemisphere Publishing.
- Harrison, A. J., Long, K. A., Tommet, D. C., & Jones, R. N. (2017). Examining the role of race, ethnicity, and gender on social and behavioral ratings within the Autism Diagnostic Observation Schedule. *Journal of Autism and Developmental Disorders*, 47(9), 2770–2782. <https://doi.org/10.1007/s10803-017-3176-3>.
- Hilton, C. L., Fitzgerald, R. T., Jackson, K. M., Maxim, R. A., Bosworth, C. C., Shattuck, P. T., ... Constantino, J. N. (2010). Brief report: Underrepresentation of African Americans in autism genetic research: A rationale for inclusion of subjects representing diverse family structures. *Journal of Autism and Developmental Disorders*, 40(5), 633–639. <https://doi.org/10.1007/s10803-009-0905-2>.
- Kirkovski, M., Enticott, P. G., & Fitzgerald, P. B. (2013). A review of the role of female gender in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(11), 2584–2603. <https://doi.org/10.1007/s10803-013-1811-1>.
- Knapp, M. L., Hall, J. A., & Horgan, T. G. (2013). *Nonverbal communication in human interaction*. Boston, MA: Wadsworth Cengage Learning.
- Kochman, T. (1981). *Black and white styles in conflict*. Chicago: University of Chicago Press.
- Lai, M.-C., Lombardo, M., Auyeung, B., Chakrabarti, B., & Baron-Cohen, S. (2015). Sex/gender differences and Autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 54(1), 11–24.
- Lancy, D. F. (2007). Accounting for variability in mother-child play. *American Anthropologist*, 109(2), 273–284. <https://doi.org/10.1525/aa.2007.109.2.273>.

- Marsh, A. (2003). Nonverbal “accents” cultural differences in facial expressions of emotion. *Psychological Science*, 14(4), 373–376.
- Masuda, T., & Nisbett, R. E. (2001). Attending holistically versus analytically: Comparing the context sensitivity of Japanese and Americans. *Journal of Personality and Social Psychology*, 8(5), 922–934. <https://doi.org/10.1037/0022-3514.81.5.922>.
- McClure, E. B. (2000). A meta-analytic review of sex differences in facial expression processing and their development in infants, children, and adolescents. *Psychological Bulletin*, 126(3), 424. <https://doi.org/10.1037/0033-2909.126.3.424>.
- Vrana, S. R., & Rollock, D. (2002). The role of ethnicity, gender, emotional content, and contextual differences in physiological, expressive, and self-reported emotional responses to imagery. *Cognition & Emotion*, 16(1), 165–192. <https://doi.org/10.1080/0269993014300018>.
- Yuki, M., Maddux, W. W., & Masuda, T. (2007). Are the windows to the soul the same in the East and West? Cultural differences in using the eyes and mouth as cues to recognize emotions in Japan and the United States. *Journal of Experimental Social Psychology*, 43(2), 303–311. <https://doi.org/10.1016/j.jesp.2006.02.004>.

Inactive

► Passive Group

Inborn Errors of Metabolism

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Synonyms

Congenital metabolic diseases; Inherited metabolic diseases

Definition

Inborn errors of metabolism are a very large group of genetic or inherited disorders, often due to single-gene defects, that impact the typical function of specific and identified biochemical processes. It is

estimated that they occur in 70/10,000 live births. Metabolism is the act of synthesizing or breaking down compounds and involves biochemical action. When there is a congenital absence of an enzyme or other biochemical impediments to breakdown of cellular by-products or creation of necessary molecules, the intended cellular functions cannot take place resulting in illness or cellular damage. Accumulation of toxic substance(s) may occur. An individual may be unable to create an essential compound. One example of an inborn error of metabolism is phenylketonuria or PKU. This is an autosomal recessive genetic disorder characterized by the absence of an enzyme (phenylalanine hydroxylase) that forms the amino acid phenylalanine ingested through the diet into tyrosine. If untreated, phenylalanine accumulates as a toxic compound and can cause brain damage. Prior to newborn screening programs to identify this disorder and allow for implementation of a special dietary treatment, this was one cause of symptoms of autism and intellectual disability. Inborn errors of metabolism may affect biochemical systems that are responsible for synthesis and/or removal of amino acids, cholesterol and bile acids, creatine, fatty aldehydes, lysosomes, organic acids, peroxisomes, pyrimidines, urea cycle intermediates, and vitamins/cofactors and glucose homeostasis and transport, among others. This class of disorders is often categorized into disorders that impact amino acid, carbohydrate, or organic acid metabolism in addition to lysosomal storage diseases. Newborn screening with mass spectroscopy makes it possible to identify many of these disorders early in infancy. At this time, research studies indicate that evaluation for inborn errors of metabolism in children with nonsyndromic autism is not indicated (Campistol et al. 2016). Measured metabolites suggesting metabolic dysfunction that are not associated with a known inborn error of metabolism could be causative of the symptoms of ASD or may be secondary to either treatments used for ASD or to associated medical conditions. Mutations of genes in the maternally inherited mitochondrial genome appear to cause increased oxidative stress and changes in energy metabolism in the cell. It has been suggested that there is an increased rate of mitochondrial disorders among individuals with ASD.

See Also

- [Medical Evaluation in Autism](#)
- [Metabolic Testing](#)

References and Reading

- Baieli, S., Pavone, L., Meli, C., Fiumara, A., & Coleman, M. (2003). Autism and phenylketonuria. *Journal of Autism and Developmental Disorders*, 33(2), 201–204.
- Campistol, J., Díez-Juan, M., Callejón, L., Fernandez-De Miguel, A., Casado, M., García Cazorla, A., Lozano, R., & Artuch, R. (2016). Inborn error metabolic screening in individuals with nonsyndromic autism spectrum disorders. *Dev Med Child Neurol*, 58(8):842–7. <https://doi.org/10.1111/dmcn.13114>. Epub 2016 Mar 31.
- Myers, S. M., & Johnson, C. P. (2007). Management of children with autism spectrum disorders. *Pediatrics*, 120, 1162–1182.
- Siddiqui, M. F., Elwell, C., & Johnson, M. H. (2016). Mitochondrial dysfunction in autism spectrum disorders. *Autism Open Access*, 6(5), pii: 1000190.

Incapacity

- [Disability](#)

Incidence

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Synonyms

[Cumulative incidence](#); [Cumulative risk](#); [Incidence density](#); [Incidence rate](#)

Definition

Broadly, incidence refers to newly occurring health events. In the context of public health, incidence may be used to describe new cases of disease, as opposed to chronic or preexisting

conditions. More specifically, incidence is expressed as the number of new conditions measured over a period of time divided by a denominator.

When this denominator is the number of persons “at risk” (free of the condition being studied) at the beginning of the specified period of time, this measure of incidence is called the incidence proportion, the cumulative risk, or the cumulative incidence. Cumulative incidence is often expressed as a percentage. When the denominator is the amount of “person-time” at risk, the measure of incidence is called the incidence density, incidence density rate, incidence rate, or person-time incidence rate. Person-time is a single number which represents the amount of time an entire population was at risk over the prespecified period of time.

See Also

- [Prevalence](#)

References and Reading

- Rothman, K. J., Greenland, S., & Lash, T. L. (2008). *Modern epidemiology* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
- Szklo, M., & Nieto, J. (2006). *Epidemiology: Beyond the basics* (2nd ed.). Boston: Jones and Bartlett.

Incidence Density

- [Incidence](#)

Incidence Rate

- [Incidence](#)

Incidental Emotional Processing

- [Visual Responses to Implicit Emotional Faces](#)

Incidental Teaching

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Definition

Incidental teaching is a naturalistic language intervention approach which targets the acquisition of spoken language within the context of naturally occurring adult-child interactions, including play. Incidental teaching requires that the child's environment be arranged to attract the child to interact with toys and activities. Child learning can then be structured and sequenced to take advantage of the child's interests and motivation within these play-based activities. Incidental teaching focuses on child-directed activities, that is, following the child's lead to encourage child motivation to initiate an interaction by requesting help from the adult. Because it is situated within the natural environment, incidental teaching supports the generalization of targeted skills. Teachers/clinicians and parents can use arrangement of the natural environment to indirectly prompt child initiation of teaching episodes which are based upon pre-determined intervention targets or educational objectives. Once the child shows an interest in the activity, by gesturing or requesting, the parent or clinician can prompt an elaborated version of the child's initiation. Natural consequences (in the form of access to desired materials, assistance, or adult attention) are provided consistently to reinforce the child's production of an elaborated communicative attempt. Incidental teaching differs from traditional discrete trial behavioral approaches in that teaching trials are child initiated and the reward for correct language production is directly related to the child's target behavior.

Historical Background

The motivation for the development of a naturalistic language teaching paradigm emerged from

the work of Don Baer at the University of Kansas in the 1970s. Baer's work highlighted the lack of skill generalization that could result from the implementation of highly structured, massed trials using behavior modification techniques. These operant techniques resulted in language behaviors that were under strict stimulus control and failed to generalize to spontaneous use in uncontrolled contexts. Incidental teaching was first introduced by Hart and Risley (1968, 1974, 1975) to teach labels, adjectives, and compound sentences to disadvantaged children within a preschool classroom in which the environment was carefully arranged to motive child initiations. In addition, incidental teaching provides many of the stimulus conditions necessary to support generalization. It is conducted on numerous occasions throughout the day within the setting conditions that maintain natural language use. Additionally, reinforcement contingencies are likely to be less discriminable under incidental teaching, than in one-to-one teaching, leading to enhanced generalization. Because the child controls the instances in which incidental teaching occurs (also known as "teachable moments") and specifies the reinforcers, incidental teaching constitutes "loose training" (training that involves many exemplars in an environment that is not tightly controlled).

Rationale or Underlying Theory

The rationale for the effectiveness of incidental teaching arises from both developmental and behavioral theories. According to behavioral theories, incidental teaching should be effective in teaching language targets in natural contexts because it incorporates the behavioral learning principles of imitation, modeling, shaping, loose teaching, multiple exemplars, and contingent reinforcement. According to developmental theories, incidental teaching should be effective in teaching language targets because it follows the child's lead and teaches to the child's interests and motivations. In addition, there is a close correspondence between the child's focus of attention to an event and the way this event is linguistically encoded by the adult. The selection of developmentally

progressive teaching targets is closely matched to the child's current abilities, and the child is able to observe that his communicative attempts have an effect on the environment by gaining him access to attention, assistance, and child-selected reinforcers.

Goals and Objectives

The goal of incidental teaching is to increase language skills within the natural environment by means of adult-child interactions that resemble those in typical development. The incidental teaching approach is child directed and uses natural consequences to reinforce child communicative attempts.

Treatment Participants

Over the years, incidental teaching has been used successfully with many groups of children who are language delayed. The earliest participants in studies of incidental teaching were children who were economically disadvantaged. The approach was then extended to children with intellectual impairments. More recently, incidental teaching strategies have been examined for use with children with autism (e.g., the Walden Toddler Program). The application of incidental teaching to children with autism was motivated by problems with prompt dependence and generalization that might emerge for children with autism following language teaching based on discrete trial training.

Treatment Procedures

Incidental teaching involves: (1) arranging the environment to increase the likelihood of child initiations (which set the occasion for teaching episodes), (2) selecting developmentally appropriate language targets for the child, (3) responding to child initiations with requests for elaborated language that resemble targeted forms, and (4) reinforcing child communication bids with attention and access to desired materials. Incidental teaching episodes are brief to maintain

child engagement. Incidental teaching differs from naturally occurring language learning by typically developing children in that language targets are preselected by the adult and a sequence of least to most prompts is used. Adapted versions of incidental teaching have introduced the use of time delay procedures and the mand-model technique. Time delay is a procedure in which a brief pause is introduced between a stimulus and a prompt in order to elicit a previously learned response from the child. In the mand-model procedure, non-yes-no questions as well as imitative prompts are used to increase the number of teaching episodes that can be implemented while using an incidental teaching approach. Another modification of incidental teaching is the training of parents to act as their child's interventionist.

Efficacy Information

Many published studies over the last 40 years have demonstrated the efficacy of various versions of incidental teaching procedures in teaching language skills to children with autism.

Outcome Measurement

Outcome measures for studies utilizing an incidental teaching approach typically consist of spontaneous and imitative spoken responses by the child. However, some studies of incidental teaching have also measured children's receptive identification of targeted vocabulary.

Qualifications of Treatment Providers

Incidental teaching has been found to be effective when used by teachers, clinicians, and parents.

See Also

- [Environmental Engineering/Modifications](#)
- [Milieu Teaching](#)
- [Natural Language Paradigm](#)

References and Reading

- Charlop-Christy, M. H., & Carpenter, M. H. (2000). Modified incidental teaching sessions: A procedure for parents to increase spontaneous speech in their children with autism. *Journal of Positive Behavior Interventions*, 2, 98–112.
- Goldstein, H. (2002). Communication intervention for children with autism: A review of treatment efficacy. *Journal of Autism and Developmental Disorders*, 32, 373–396.
- Hart, B., & Risley, T. R. (1968). Establishing use of descriptive adjectives in the spontaneous speech of disadvantaged preschool children. *Journal of Applied Behavior Analysis*, 1, 109–120.
- Hart, B., & Risley, T. R. (1974). Using preschool materials to modify the language of disadvantaged children. *Journal of Applied Behavior Analysis*, 7, 243–256.
- Hart, B., & Risley, T. R. (1975). Incidental teaching of language in the preschool. *Journal of Applied Behavior Analysis*, 8, 411–420.
- McGee, G. G., Krantz, P. J., & McClannahan, L. E. (1985). The facilitative effects of incidental teaching on preposition use by autistic children. *Journal of Applied Behavior Analysis*, 18, 17–31.
- McGee, G., Morrier, M., & Daly, T. (1999). An incidental teaching approach to early intervention for toddlers with autism. *Journal of the Association for Persons with Severe Handicaps*, 24, 133–146.
- Stokes, T., & Baer, D. (1977). An implicit technology of generalization. *Journal of Applied Behavior Analysis*, 10, 349–367.
- Warren, S., & Kaiser, A. (1986). Incidental language teaching: A critical review. *Journal of Speech and Hearing Disorders*, 51, 291–299.

Inclusion

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Definition

Inclusion means that children with autism (ASD) learn alongside typically developing children in general education classrooms. For young children with ASD, those classrooms may be community-based child care programs, Head Start, or prekindergarten programs. For school-aged children,

those programs are in their local elementary, middle, or high schools. When children with ASD are included, they have the opportunity to develop relationships and learn from and model the behavior of their typically developing peers (Fein and Dunn 2007). When children with ASD are fully included, they not only have the opportunity to learn academic and functional skills, they become accepted members of their community (Allen and Cowdery 2012).

Historical Background

The term “inclusion” began to appear in the 1990s (Odom and Diamond 1998). Before then, children who received instruction in general education classrooms were mainstreamed. Children who were mainstreamed were in general education classrooms part time. They visited the general education classroom for selected lessons and activities, but their primary placement was still in a special education classroom. The special education teacher was responsible for their educational progress (Mastropieri and Scruggs 2000). That changed in the 1990s with inclusion. Today, children who are included are now placed in general education classrooms, and the general education teacher has that responsibility.

The change from segregated placements to mainstreaming to inclusion came about through a series of laws and court cases with the most prominent being IDEA (Individuals with Disabilities Education Act), passed originally as PL 94–142: The Education for All Handicapped Children Act. This law and its subsequent amendments contain a number of provisions, which have provided the basis for educating children with ASD in inclusive environments. Among those provisions are zero reject and least restrictive environment. Zero reject sets forth the basic principle that children with disabilities cannot be excluded from receiving a public education. Least restrictive environment states that, to the maximum extent possible, children with disabilities should be educated with their peers who do not have a disability. Another law, the Americans with Disabilities Act (ADA), has also had an impact on

children being served in inclusive environments. The ADA has had a particular impact on young children because ADA stipulates that children cannot be excluded from child care programs unless their presence affects the health or safety of other children (Grisham-Brown et al. 2005).

There are additional arguments that support the education of children with disabilities in inclusive environments that include moral and rational reasons (Bailey et al. 1998). From a moral perspective, children with disabilities have the same right as other children to participate in programs and activities. Further, it is just the right thing to do. From a rational perspective, children with disabilities benefit when they interact with typically developing children because they can learn new skills from them. Typically developing children benefit as well because they learn about differences among people and may become more accepting of people who are different (Bailey et al. 1998).

Current Knowledge

According to Harrower and Dunlap (2001), there have been relatively few studies which have investigated inclusion as the independent variable, that is, few which ask the question if children with ASD perform better socially and academically in inclusive classrooms than they do in segregated classrooms. However, as Harrower and Dunlap suggested, the better question to ask is how can children with ASD receive the help they need to be successful in an inclusive classroom? Inclusion cannot be accomplished successfully if the adults who work with the children with ASD in their classroom are not prepared for that challenge.

The Role of Teachers in Inclusive Classrooms

There are a number of ways that general education and special education teachers can work together to provide instruction for children with ASD. One approach is coteaching (Friend and Cook 2007). In coteaching, a general education teacher and a special education teacher are classroom equals. They plan together and make decisions about

how learning can be optimized for all the children in the classroom. The general education teacher is the curriculum expert; the special education teacher knows how to differentiate instruction so that children with ASD can learn as well. Coteachers can use a number of approaches to ensure that all children are engaged during instruction. They include one teaching and one observing, station teaching, parallel teaching, alternative teaching, teaming, and one teaching and one assisting (Friend and Cook 2007). In all the approaches, it is important to be sure that the special education teacher does not get relegated to the role of a classroom assistant who is only there to help out.

Paraeducators

Another approach to fostering inclusion for children with ASD is to assign a paraeducator to them in the classroom. This approach is used frequently with children with ASD because they often have intensive needs. The paraeducator works under the supervision of the special education teacher who plans the specific instruction. The paraeducator's role might include providing alternative instruction within the general education classroom, modifying and simplifying the response that the child with ASD is expected to provide, providing reinforcement when the child with ASD makes an appropriate response, and collecting data on child progress (Friend and Cook 2007). It is important that the teachers in the classroom ensure that the paraeducator does not become a "Velcro" aide. If the paraeducator is always attached to the child with ASD, the benefits of being in an inclusive classroom disappear because the child does not have the opportunity to learn from other children.

General Approaches to Inclusion

Regardless of the age of the children with ASD, their inclusion into general education classrooms and activities can be facilitated if the curriculum incorporates elements of Universal Design for Learning (UDL). A UDL curriculum focuses on creating learning environments and adopting practices that give children a variety of formats for responding, demonstrating what they know, and expressing ideas, feelings, and preferences

(Center for Applied Special Technology [CAST] 2004). The three key principles of UDL are multiple means of representation, multiple means of engagement, and multiple means of expression (CAST 2004). Teachers who use *multiple means of representation* provide their instruction using a variety of formats for children who may need actual objects, pictures, or text to understand the content that the teacher presents. When a curriculum is designed using UDL principles, children are given the opportunity to use *multiple means of expression* for their responses. Some children may make written responses or respond using language. Others may use gestures or PECS or other assistive devices to demonstrate what they know. A curriculum that includes *multiple means of engagement* has activities and lessons that are interesting to all children; they are engaged and interested in participating and learning (Blackhurst et al. 1999; CAST 2004; Orkwis 2003). Having a curriculum that incorporates elements of UDL is particularly valuable for children with ASD who are included in general education classrooms.

Inclusion in Early Childhood Classrooms

The importance of including young children with disabilities in all early childhood classrooms has been supported by two major early childhood organizations, the Division for Early Childhood (DEC) and the National Association for the Education of Young Children (NAEYC). These organizations published a joint statement on inclusion in 2009. This position statement argues that children can be included successfully if they have access, can participate, and are provided with supports. Access comes through the design of the program and the environment; participation occurs when teachers use a variety of approaches including routine-based teaching and explicit interventions; and supports happen when the administration, teachers, related service providers, and families collaborate to ensure children's success (DEC/NAEYC 2009). More recently, in 2015, the U.S. Department of Health and Human Services and the U.S. Department of Education issued a Policy Statement on Inclusion of Children with Disabilities in Early Childhood Programs.

Included in this policy statement is: (1) the expectation that inclusion in early childhood programs be of high quality; (2) that there are legal and scientific reasons for inclusion; and (3) identification of free resources for States, LEAS, schools, and early childhood programs to support high quality inclusion (HHS/ED 2015, p. 1).

Young children with ASD are eligible to receive services as soon as they are identified. Children before the age of three are typically served in their homes, but they may receive services in other natural environments like a child care center. Once they turn three, however, most attend programs with other children and may be included in a variety of programs. Children with disabilities have been included in Head Start since 1972 with the passage of PL 92-424. Beginning in 1972, Head Start programs have been required to ensure that 10% of the children they serve have disabilities (Grisham-Brown et al. 2005). Although many Head Start programs serve children with more mild disabilities such as speech/language disabilities, some children with ASD are included. Young children with ASD may also attend state-funded prekindergarten programs and community-based child care programs.

In order for young children with ASD to learn and thrive in inclusive programs, the first step is to ensure that the program is of high quality. According to Sandall and Schwartz (2008), a high-quality program provides a consistent schedule that is divided into time segments that are appropriate for young children, has a combination of large group and small group activities as well as time for children to play and work alone, has activities that teachers lead and those that are child-directed, allows children time for outdoor play and routines such as toileting and eating, has both quiet times and active times, and finally, maximizes the time that teachers teach and minimizes the time that children wait.

Having a high-quality program is only the beginning. In order for children with ASD to learn both academically and socially, the teachers need to be ready to make modifications to the curriculum. In their Building Blocks model, Sandall and Schwartz (2008) described eight curriculum modifications: environmental support,

materials adaptation, activity simplification, child preferences, special equipment, adult support, peer support, and invisible support. Because young children with ASD are typically delayed in their language and social development, a number of these modifications can be particularly useful as teachers include them in the ongoing classroom routine.

Barton et al. (2011) described how curriculum modifications can help a child with ASD participate successfully in circle time, a large group activity that is standard in most early childhood classrooms. Circle time is a teacher-led activity that is used to teach early literacy activities through book reading, time concepts through reviewing the daily routine and calendar, as well as music and movement activities. Barton et al. suggested that circle time can be particularly challenging for children with ASD because during circle time, children are required to attend to a teacher and follow teacher directions, to be next to other children, and to use language to communicate. However, it can be a valuable activity because it provides an opportunity for children with ASD to address skills that they have most difficulty mastering. Barton et al. provided a number of examples of how the curriculum modifications can be used for children with ASD during the circle time activity. If children have difficulty making the transition from another activity to the circle, the teacher can use child preferences and allow the children to bring a favorite toy with them to the circle. Children can hold on to that toy throughout the circle activity. Once children are seated at the circle, the teacher can reduce the distractions that come from the shelves of toys that surround the circle area by covering those shelves with fabric that drop down during circle time. The teacher can adapt the materials used during circle by creating a daily schedule that incorporates words and pictures to symbolize parts of the daily schedule. In another adaptation, as the teacher reads a book, the children can have their own smaller version of the book to follow along with. Peers can be used to support the children with ASD in a variety of ways. Children with ASD can sit next to a favorite peer, the peer can go

first when the teacher asks a question so that the children with ASD have a language model to follow, and the peer can even prompt the children with ASD to make a response. These curriculum modifications can be used throughout the daily routine for other large group as well as small group activities.

In addition to curriculum modification, children's learning is enhanced in inclusive classrooms when teachers embed children's learning objectives within the context of ongoing classroom activities (Sandall and Schwartz 2008). If a child with ASD has an IEP objective to enhance expressive language by making requests that objective can be targeted during many activities throughout the day. During snack time, the child can be asked to request juice, cookies, and fruit. And children are naturally reinforced when given the food they asked for.

Inclusion for School-Aged Children

Sandall and Schwartz (2008) described a general approach to adapting instruction that can be used across curricula in early childhood classrooms to promote effective inclusion for young children with disabilities. Harrower and Dunlap (2001) similarly discussed general instructional strategies that have been shown to be effective in improving social and academic outcomes and that can be implemented in inclusive settings for school-aged children.

A number of the approaches that Harrower and Dunlap (2001) described involve peers. One approach is peer tutoring in which a typical peer provides assistance, instruction, and feedback to a child with ASD. Another approach, that involves larger groupings, is cooperative learning. In cooperative learning, small groups of children work together to learn information. Small groups allow children with ASD to participate more frequently than they could in a large group, teacher-led activity. More frequent participation leads to higher levels of engagement with the material. Cooperative learning also allows the children with ASD to engage in social interactions with their peers while learning academic content. Handleman et al. (2005) listed other

peer-focused interventions that have been used successfully in inclusive classrooms. These include having a peer buddy system for all children in the classroom and instituting social skills groups.

A second group of approaches that Harrower and Dunlap (2001) identified are self-management strategies. According to Harrower and Dunlap, these strategies help children with ASD become more independent which allows them to operate more effectively in an inclusive classroom. As children become more independent, teachers can spend more of their time providing instruction to the class. Harrower and Dunlap (2001) provided additional strategy categories including antecedent procedures and delayed contingencies. These approaches are designed to help children with ASD become less dependent on adults so that they can operate more effectively in inclusive classrooms where there are fewer adults and more children.

Because general education teachers are responsible for the instructional content in inclusive classrooms, they can foster the participation of children with ASD by ensuring that the content is meaningful to all of the children, by having a range of materials to teach the content, and by using a variety of lesson formats (Kluth 2010). These methods incorporate elements of UDL and make the content accessible to a wide range of learners.

Future Directions

Although children with ASD have been included in general education classrooms for over 20 years, there are a number of areas that need further investigation. These areas include:

- Effective strategies for middle and high school students with ASD in inclusive settings
- How best to train general education teachers to provide instruction for children with ASD
- Strategies to enhance collaboration between general and special educators who work with children with ASD

See Also

- [Integration](#)
- [Least Restrictive Environment \(LRE\)](#)
- [Natural Environment](#)

References and Reading

- Allen, K. E., & Cowdery, G. E. (2012). *The exceptional child: Inclusion in early childhood education* (7th ed.). Belmont: Wadsworth.
- Bailey, D. B., McWilliam, R. A., Buysse, V., & Wesley, P. W. (1998). Inclusion in the context of competing values in early childhood education. *Early Childhood Research Quarterly*, 13(1), 27–47.
- Barton, E. E., Reichow, B., Wolery, M., & Chen, C. (2011). We can all participate! Adapting circle time for children with autism. *Young Exceptional Children*, 14(2), 2–21.
- Blackhurst, E., Carnine, D., Cohen, L., Kameenui, E., Langone, J., Palley, D., et al. (1999, Fall). Research connections in special education: Universal design. Retrieved 7 June 2011, from <http://www.hoagiesgifted.org/eric/osep/recon5/rc5cov.html>
- Center for Applied Special Technology. (2004). Universal design for learning. Retrieved 7 June 2011, from <http://www.cast.org/udl>
- DEC/NAEYC. (2009). *Early childhood inclusion: A joint position statement of the division for early childhood (DEC) and the National Association for the education of young children (NAEYC)*. Chapel Hill: The University of North Carolina, FPG Child Development Institute.
- Fein, D., & Dunn, M. A. (2007). *Autism in your classroom: A general educator's guide to students with autism spectrum disorders*. Bethesda: Woodbine House.
- Friend, M., & Cook, L. (2007). *Interactions: Collaboration skills for school professionals* (5th ed.). Boston: Pearson.
- Grisham-Brown, J., Hemmeter, M. L., & Pretti-Fontczak, K. (2005). *Blended practices for teaching young children in inclusive settings*. Baltimore: Brookes.
- Handleman, J. S., Harris, S. L., & Martins, M. P. (2005). Helping children with autism enter the mainstream. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. II, pp. 1029–1042). Hoboken: Wiley.
- Harrower, J. K., & Dunlap, G. (2001). Including children with autism in general education classrooms: A review of effective strategies. *Behavior Modification*, 25(5), 762–784.
- Kluth, P. (2010). *You're going to love this kid!* (2nd ed.). Baltimore: Brookes.
- Mastropieri, M. A., & Scruggs, T. E. (2000). *The inclusive classroom: Strategies for effective instruction*. Upper Saddle River: Merrill.

- Odom, S. L., & Diamond, K. E. (1998). Inclusion of young children with special needs in early childhood education: The research base. *Early Childhood Research Quarterly*, 13(1), 3–25.
- Orkwis, R. (2003). Universally designed instruction. *ERIC/OSEP Digest* (ERIC document reproduction service No. ED 475386).
- Sandall, S. R., & Schwartz, I. S. (2008). *Building blocks for teaching preschoolers with special needs* (2nd ed.). Baltimore: Brookes.
- U.S. Department of Health and Human Services and U.S. Department of Education. (2015). Policy statement on inclusion of children with disabilities in early childhood programs.

Inclusion of Adolescents with ASD in Community Sporting Clubs

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Synonyms

Community sport; Exercise; Social inclusion

Definition

1.0% to 2.64% of children are estimated to have a diagnosis of autism spectrum disorder (ASD) with an increasing number of individuals receiving a diagnosis of ASD (ASD; Baio et al. 2018; Baron-Cohen et al. 2009; Boilson et al. 2016; Kim et al. 2011; Wing and Potter 2002). Given this increase, there is a growing demand on diagnostic and intervention services (Shattuck and Grosse 2007; Wise et al. 2010), and parents of children with ASD report that they are not receiving the disability services that they need (Jones et al. 2017). Furthermore families with a child with a diagnosis of ASD report feelings of social isolation and feeling cut off from the wider society (Byrne et al. 2018; Woodgate et al. 2008).

Given families with a child with ASD often face long wait lists for intervention in public disability services and report not receiving needed

services (Jones et al. 2017; Rivard et al. 2014), exercise interventions outside the clinic could be a worthwhile alternative. Moreover there is research to suggest that providing social skill promotional activities in a child's natural setting appears to be more effective than traditional clinical settings (Bellini and Peters 2008).

It is necessary to distinguish structured exercise from physical activity. While there are many definitions of exercise and physical activity, Caspersen et al.'s (1985) interpretation shall be employed for the purpose of this definition. Physical activity is defined in a broad term as any movement of the body, created by the body, that results in energy expenditure, (p. 126), which can be interpreted as movements that are required in everyday life such as climbing stairs, cleaning, etc. Caspersen et al. (1985) define exercise as a purposeful, planned activity that is repetitive in nature and has set goals pertaining to the improvement or maintenance of physical fitness (p. 128). Therefore, structured exercise has a clear start and finish time. Given individuals with autism spectrum disorder (ASD) have greater difficulties coping with unstructured time than their typically developing peers, there is benefit to be sought from structured exercise (Van Bourgondien et al. 2003).

The inclusion of disabilities within exercise is neither a new nor novel idea as Block and Obrusnikova (2007) reviewed 20 years of studies highlighting the success of inclusion of children with disabilities in a supported physical education environment. Furthermore, there is converging evidence to suggest that the benefits of structured exercise in children with ASD extend beyond general physical health and have a positive impact on their social development. Ohrberg (2013) described structured exercise in the lives of children with ASD as essential, emphasizing that it allows children to develop interpersonal skills through engaging in fun activities with peers in a very normal way (p. 53).

There is converging evidence to suggest that structured exercise and inclusive physical education classes improve social interaction and promote social development in students with ASD (Foti et al. 2014; Pan et al. 2011). Structured cycling, swimming, and martial arts programs

have reported improvements in self-efficacy and social interaction and a reduction in antisocial behaviors in children with ASD (Movahedi et al. 2013; Pan 2010; Todd et al. 2010).

However, the core characteristics of autism spectrum disorder (ASD), which include impairments in social communication and interaction, and the presentation of restricted, repetitive behaviors (American Psychiatric Association [APA] 2013) may conflict with the expectations of exercise (Jones 2003; Obrusnikova and Cavalier 2011; Pan 2014; Srinivasan et al. 2014). Such a conflict may explain why children with a diagnosis of ASD are not meeting the recommended exercise guidelines (Pan and Frey 2006). Furthermore, children with ASD are reported to be less active during breaktime and engage in few physical activities for shorter periods of time when compared with their typically developing peers (Bandini et al. 2013; Healy et al. 2013; Hilton et al. 2008; Pan 2008). Moreover, parents and teachers report being unaware of the many inclusive structured exercise programs for children with ASD (Srinivasan et al. 2014).

The social model of disability guides the rationale and success of inclusive sport programs. The social model welcomes diversity, including and valuing each adolescent and providing a liberating perspective on disability, relocating the disability into the structures of society and outside of the individual. The restrictions that people with impairments face in sport can be readily observed, and challenged, through the social model, from individual attitudinal and institutional prejudices to inaccessible sporting facilities, exclusionary policies, or unusable transport systems (Oliver 2004). Mike Oliver (2004) argued that the social model has the power to “transform consciousness” (p. 42) by connecting personal experience to professional practice.

Despite these benefits, sport is an area which is still governed by the medical model of disability (Townsend et al. 2016). The medical model of disability would suggest that people are disabled by their impairments or differences. The medical model looks at what is “wrong” with the person and not what the person needs, and it can create low expectations which can lead to people losing

independence, choice, and control in their own lives.

In light of the medical model of disability, the provision of inclusive opportunities still has a low priority in community recreation and sport in many countries (Storey 2004) causing individuals with additional needs to take part in sporting activities much less frequently than other people with disability and those who do not have a disability (Darcy and Dowse 2013).

A lack of coaching expertise specific to disability and adaptations has been identified as a barrier for the participation of adolescents with additional needs in sport (D. B. Jones 2003; Kozub and Porretta 1998; Tsai and Fung 2009). Coaching is a vital aspect of athletic development and success, and as such, coaches play an important role in facilitating, encouraging, and guiding participation (Erickson et al. 2008; Martin and Whalen 2014). Coaches report very little inclusion of information specific to disability in the accredited training courses for their specific sport (Wareham et al. 2018). Moreover, coaches report the necessity of familiarization with the biological and physiological effects of their athletes’ impairment(s), highlighting that it was left to them to source this information. There is some evidence to suggest that factors such as insufficient knowledge of, or experience with, disability are contributing factors to the reluctance by many coaches to make the transition to coaching athletes with additional needs (Martin and Whalen 2014).

There is a need to develop localized theoretical frameworks to inform initiatives going forward (Edwards 2015). Research suggests there is an absence of participant experiences and perspectives of inclusive sport (Levermore 2010; Rich et al. 2015) and in particular hearing the voices and viewpoints of people with disabilities (Wickman 2015).

Future research should focus on how adolescents with autism spectrum disorder (ASD) can be integrated and included within community sporting clubs with the potential focus sport to be rowing. Such research should obtain the perspective of the main stakeholders regarding the facilitation of an ASD inclusive community club.

These stakeholders would be community club coaches, parents, adolescents with ASD, and typically developing adolescents involved in the club.

Ohrberg (2013) advises on the development of a handbook to facilitate an autism-aware culture in sport. Therefore the input from the stakeholders would potentially be compiled to produce a handbook to facilitate the participation of adolescents with ASD (i.e., identifying potential barriers to participation and sport-specific coaching content to aid understanding of ASD and the subsequent and necessary adaptations to coaching style and training program). This handbook would be introduced and piloted with local clubs and adjusted based on subsequent feedback. This handbook would then be introduced into the national coaching training course as an independent module. The handbook would be edited one further time based on national feedback before being submitted to national sporting body. The proposed handbook would inform a sport-specific framework which encompasses the perspective of all relevant stakeholders, including the adolescent with ASD.

References and Reading

- American Psychiatric Association [APA]. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. Philadelphia: American Psychiatric Pub.
- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., et al. (2018). Prevalence of autism spectrum disorder among children aged 8 years—Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summaries*, 67(6), 1.
- Bandini, L. G., Gleason, J., Curtin, C., Lividini, K., Anderson, S. E., Cermak, S. A., et al. (2013). Comparison of physical activity between children with autism spectrum disorders and typically developing children. *Autism*, 17(1), 44–54.
- Baron-Cohen, S., Scott, F. J., Allison, C., Williams, J., Bolton, P., Matthews, F. E., & Brayne, C. (2009). Prevalence of autism-spectrum conditions: UK school-based population study. *The British Journal of Psychiatry*, 194(6), 500–509.
- Bellini, S., & Peters, J. K. (2008). Social skills training for youth with autism spectrum disorders. *Child and Adolescent Psychiatric Clinics of North America*, 17(4), 857–873.
- Block, M. E., & Obrusnikova, I. (2007). Inclusion in physical education: A review of the literature from 1995–2005. *Adapted Physical Activity Quarterly*, 24(2), 103–124.
- Boilson, A. M., Staines, A., Ramirez, A., Posada, M., & Sweeney, M. R. (2016). Operationalisation of the European protocol for autism prevalence (EPAP) for autism spectrum disorder prevalence measurement in Ireland. *Journal of Autism and Developmental Disorders*, 46(9), 3054–3067.
- Byrne, G., Sarma, K. M., Hendler, J., & O'Connell, A. (2018). On the spectrum, off the beaten path. A qualitative study of Irish parents' experiences of raising a child with autism spectrum conditions. *British Journal of Learning Disabilities*, 46(3), 182–192.
- Caspersen, C. J., Powell, K. E., & Christenson, G. M. (1985). Physical activity, exercise, and physical fitness: Definitions and distinctions for health-related research. *Public Health Reports*, 100(2), 126.
- Darcy, S., & Dowse, L. (2013). In search of a level playing field—the constraints and benefits of sport participation for people with intellectual disability. *Disability & Society*, 28(3), 393–407.
- Edwards, M. B. (2015). The role of sport in community capacity building: An examination of sport for development research and practice. *Sport Management Review*, 18(1), 6–19.
- Erickson, K., Bruner, M. W., MacDonald, D. J., & Côté, J. (2008). Gaining insight into actual and preferred sources of coaching knowledge. *International Journal of Sports Science & Coaching*, 3(4), 527–538.
- Foti, F., Mazzone, L., Menghini, D., De Peppo, L., Federico, F., Postorino, V., et al. (2014). Learning by observation in children with autism spectrum disorder. *Psychological Medicine*, 44(11), 2437–2447.
- Healy, S., Msetfi, R., & Gallagher, S. (2013). “Happy and a bit nervous”: The experiences of children with autism in physical education. *British Journal of Learning Disabilities*, 41(3), 222–228.
- Hilton, C. L., Crouch, M. C., & Israel, H. (2008). Out-of-school participation patterns in children with high-functioning autism spectrum disorders. *American Journal of Occupational Therapy*, 62(5), 554–563.
- Jones, D. B. (2003). “Denied from a lot of places” barriers to participation in community recreation programs encountered by children with disabilities in Maine: Perspectives of parents. *Leisure/Loisir*, 28(1–2), 49–69.
- Jones, S., Bremer, E., & Lloyd, M. (2017). Autism spectrum disorder: Family quality of life while waiting for intervention services. *Quality of Life Research*, 26(2), 331–342.
- Kim, Y. S., Leventhal, B. L., Koh, Y.-J., Fombonne, E., Laska, E., Lim, E.-C., et al. (2011). Prevalence of autism spectrum disorders in a total population sample. *American Journal of Psychiatry*, 168(9), 904–912.
- Kozub, F. M., & Porretta, D. L. (1998). Interscholastic coaches' attitudes toward integration of adolescents with disabilities. *Adapted Physical Activity Quarterly*, 15(4), 328–344.

- Levermore, R. (2010). CSR for development through sport: Examining its potential and limitations. *Third world quarterly*, 31(2), 223–241.
- Martin, J. J., & Whalen, L. (2014). Effective practices of coaching disability sport. *European Journal of Adapted Physical Activity*, 7(2), 13–23.
- Movahedi, A., Bahrami, F., Marandi, S. M., & Abedi, A. (2013). Improvement in social dysfunction of children with autism spectrum disorder following long term Kata techniques training. *Research in Autism Spectrum Disorders*, 7(9), 1054–1061.
- Obrusnikova, I., & Cavalier, A. R. (2011). Perceived barriers and facilitators of participation in after-school physical activity by children with autism spectrum disorders. *Journal of Developmental and Physical Disabilities*, 23(3), 195–211.
- Ohrberg, N. J. (2013). Autism Spectrum disorder and youth sports: The role of the sports manager and coach. *Journal of Physical Education, Recreation & Dance*, 84(9), 52–56.
- Oliver, M. (2004). The social model in action: If I had a hammer. *Implementing the social model of disability: Theory and research*, 18–31.
- Pan, C.-Y. (2008). Objectively measured physical activity between children with autism spectrum disorders and children without disabilities during inclusive recess settings in Taiwan. *Journal of Autism and Developmental Disorders*, 38(7), 1292.
- Pan, C.-Y. (2010). Effects of water exercise swimming program on aquatic skills and social behaviors in children with autism spectrum disorders. *Autism*, 14(1), 9–28.
- Pan, C.-Y. (2014). Motor proficiency and physical fitness in adolescent males with and without autism spectrum disorders. *Autism*, 18(2), 156–165.
- Pan, C.-Y., & Frey, G. C. (2006). Physical activity patterns in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36(5), 597.
- Pan, C.-Y., Tsai, C.-L., & Hsieh, K.-W. (2011). Physical activity correlates for children with autism spectrum disorders in middle school physical education. *Research Quarterly for Exercise and Sport*, 82(3), 491–498.
- Rich, K. A., Misener, L., & Dubeau, D. (2015). Community cup, we are a big family. Examining social inclusion and acculturation of newcomers to Canada through a participatory sport event. *Social Inclusion*, 3(3), 129–141.
- Rivard, M., Terroux, A., Parent-Boursier, C., & Mercier, C. (2014). Determinants of stress in parents of children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(7), 1609–1620.
- Shattuck, P. T., & Grosse, S. D. (2007). Issues related to the diagnosis and treatment of autism spectrum disorders. *Mental Retardation and Developmental Disabilities Research Reviews*, 13(2), 129–135.
- Srinivasan, S. M., Pescatello, L. S., & Bhat, A. N. (2014). Current perspectives on physical activity and exercise recommendations for children and adolescents with autism spectrum disorders. *Physical Therapy*, 94(6), 875–889.
- Storey, K. (2004). The case against the Special Olympics. *Journal of Disability Policy Studies*, 15(1), 35–42.
- Todd, T., Reid, G., & Butler-Kisber, L. (2010). Cycling for students with ASD: Self-regulation promotes sustained physical activity. *Adapted Physical Activity Quarterly*, 27(3), 226–241.
- Townsend, R. C., Smith, B., & Cushion, C. J. (2016). Disability sports coaching: Towards a critical understanding. *Sports Coaching Review*, 4(2), 80–98. <https://doi.org/10.1080/21640629.2016.1157324>.
- Tsai, E. H.-L., & Fung, L. (2009). Parents' experiences and decisions on inclusive sport participation of their children with intellectual disabilities. *Adapted Physical Activity Quarterly*, 26(2), 151–171.
- Van Bourgondien, M. E., Reichle, N. C., & Schopler, E. (2003). Effects of a model treatment approach on adults with autism. *Journal of Autism and Developmental Disorders*, 33(2), 131–140.
- Wareham, Y., Burkett, B., Innes, P., & Lovell, G. P. (2018). Sport coaches' education, training and professional development: The perceptions and preferences of coaches of elite athletes with disability in Australia. *Sport in Society*, 21(12), 2048–2067.
- Wickman, K. (2015). Experiences and perceptions of young adults with physical disabilities on sports. *Social Inclusion*, 3(3), 39–50.
- Wing, L., & Potter, D. (2002). The epidemiology of autistic spectrum disorders: Is the prevalence rising? *Mental Retardation and Developmental Disabilities Research Reviews*, 8(3), 151–161.
- Wise, M. D., Little, A. A., Holliman, J. B., Wise, P. H., & Wang, C. J. (2010). Can state early intervention programs meet the increased demand of children suspected of having autism spectrum disorders? *Journal of Developmental & Behavioral Pediatrics*, 31(6), 469–476.
- Woodgate, R. L., Ateah, C., & Secco, L. (2008). Living in a world of our own: The experience of parents who have a child with autism. *Qualitative Health Research*, 18(8), 1075–1083.

Inclusion Specialist

► Itinerant Teacher

Independence

► Self-Advocacy

Independent Living

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Synonyms

[Autonomous living](#)

Definition

Independent living consists of participating in day-to-day life with full capacity to successfully choose courses of action and make decisions about where to live and how to participate in society without the oversight of another individual. Independent living requires having the skills necessary to manage life tasks including, but not limited to, household maintenance, time and money management, and self-care.

Independent living is also a philosophical viewpoint within the disability culture that promotes self-determination in individuals with disabilities and equal rights to participate in and contribute to society (The National Council on Independent Living 2010).

See Also

- [Benhaven Residential Services](#)
- [Living Arrangements in Adulthood](#)
- [Self-Determination](#)
- [Self-Help Skills](#)

References and Reading

- National Council on Independent Living. (2010). About NCIL. Retrieved December, 2010, from <http://www.ncil.org/>
- Stancliffe, R. J., & Lakin, K. C. (2007). Independent living. In S. L. Odom, R. H. Horner, M. E. Snell, & J. Blacher (Eds.), *Handbook of developmental disabilities* (pp. 429–448). New York: Guilford Press [Chapter].

Independent Variable Measurement

- [Procedural Fidelity](#)

Index of Productive Syntax (IPSyn)

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Synonyms

[IPSyn](#)

Description

Index of Productive Syntax (IPSyn) is a method for evaluating and quantifying the grammatical complexity of young children's spontaneous language samples. IPSyn is based upon the grammatical categories and developmental scheme of Assigning Structural Stage (Miller 1981). Individual utterances in a sample are scored for the occurrence of 60 different syntactic forms categorized under four subscales: noun phrase, verb phrase, question/negation, and sentence structure forms. Although IPSyn was developed for use with mainstream English speakers, it has been found to be a valid measure for children who speak African American English (Oetting et al. 2010).

IPSyn scores are calculated from children's spontaneous language samples. After a sample is elicited and recorded, a corpus is formed by transcribing 100 successive, intelligible utterances, excluding imitations, self-repetitions, and routines (stereotyped language that does not represent productive language use). Following guidelines stated in the IPSyn coding manual (Scarborough 1990), the transcript is then scored for 60 syntactic forms belonging to phrases and sentences that are categorized under four

subscales. The first two different occurrences of each form are credited. No additional credit is given for further occurrences in the sample. Certain early-developing structures are automatically credited if there is evidence in the transcript that the child has mastered later-developing structures in the same category. For example, if a child produces a question containing a wh-pronoun and verb (Q4), he also receives credit for intonationally marked question (Q1) and routine do/go or existence/name question (Q2). The points earned for each subscale are summed across all utterances, then added together to yield the total IPSyn score. No examiner qualifications are explicitly stated for this measure; however, the user must be familiar with English grammatical structures.

Historical Background

IPSyn was developed to provide a time-efficient means for measuring the grammatical complexity of children's utterances from the beginning of oral language through the period of early literacy. Other measures of grammatical complexity, such as Mean Length of Utterance (MLU, Brown 1973; Miller 1981) and Developmental Sentence Scoring (DSS, Lee 1974) had been and continue to be used for that purpose, but these other measures were criticized for their lack of scope and insensitivity to many important developmental language changes (Klee and Fitzgerald 1985; Klee and Sahlie 1986; Scarborough et al. 1991). IPSyn derived its grammatical categories and developmental scheme from Assigning Structural Stage (ASS, Miller 1981), a procedure generally hailed for its comprehensiveness. Furthermore, in contrast to measures such as MLU and DSS, IPSyn does not score every structure of every utterance within a corpus. Instead, it scans for a maximum of two structural types within each of its 60 syntactic forms. This approach results in an analysis that is adequately comprehensive yet not so time consuming as methods that evaluate all the structural forms produced by a child. IPSyn has been used in research on typical language development and disordered language development, including

autism (e.g., Tager-Flusberg and Calkins 1990; Tager-Flusberg et al. 1990). It is especially valuable to researchers using designs that require subject matching by language age (Scarborough et al. 1991).

Psychometric Data

No psychometric data are provided. IPSyn is not norm referenced.

Clinical Uses

As a clinical resource, IPSyn can be used to quantify and document linguistic changes that occur over time in successive language samples. A small database ($n = 15$) exists of typically developing children who were sampled at 24, 30, 36, 42, and 48 months (Scarborough 1990). Although these numbers fall short of what is needed for standardization, the available data show how IPSyn could be used for peer comparisons, if and when a larger standardization sample is gathered. Two computerized versions of IPSyn have been developed. In the version contained in Computerized Profiling (Long et al. 2006), the program performs an automatic analysis that must then be reviewed and edited by the user. Another version, designed to work with CHAT transcripts in the CHILDES system, is completely automatic (Sagae et al. 2005).

See Also

- Grammar
- Language Tests
- Syntax

References and Reading

- Brown, R. (1973). *A first language*. Cambridge, MA: Harvard University Press.
- Klee, T., & Fitzgerald, M. (1985). The relation between grammatical development and mean length of utterance in morphemes. *Journal of Child Language*, 12, 251–269.

- Klee, T., & Sahlie, E. (1986). Review of Hixson's DSS computer program. *Child Language Teaching and Therapy*, 2, 231–235.
- Lee, L. (1974). *Developmental sentence analysis*. Evanston: Northwestern University Press.
- Long, S. H., Fey, M. E., & Channell, R. W. (2006). *Computerized profiling (PC Version 970)*. Milwaukee: Marquette University. Website. <http://www.computerizedprofiling.org>.
- Miller, J. F. (1981). *Assessing Language Production in Children*. Baltimore: University Park Press.
- Oetting, J. B., Newkirk, B. L., Hartfield, L. R., Wynn, C. G., Pruitt, S. L., & Garrity, A. W. (2010). Index of productive syntax for children who speak African American English. *Language, Speech & Hearing Services in Schools*, 41, 328–339. <https://doi.org/10.1044/0161-1461>.
- Sagae, K., Lavie, A., & MacWhinney, B. (2005). Automatic measurement of syntactic development in child language. In *Proceedings of the 43rd annual meeting of the Association for Computational Linguistics (ACL)* (pp. 197–204). Ann Arbor. Retrieved 17 Apr 2011 from <http://childes.psy.cmu.edu/grasp/ac105-IPSyn.pdf>.
- Scarborough, H. S. (1990). Index of productive syntax. *Applied PsychoLinguistics*, 11, 1–22.
- Scarborough, H. S., Rescorla, L., Tager-Flusberg, H., Fowler, A. E., & Sudhalter, V. (1991). The relation of utterance length to grammatical complexity in normal and language-disordered groups. *Applied PsychoLinguistics*, 12, 23–45.
- Tager-Flusberg, H., & Calkins, S. (1990). Does imitation facilitate the acquisition of grammar? Evidence from a study of autistic, Down's syndrome and normal children. *Journal of Child Language*, 17, 591–606.
- Tager-Flusberg, H., Calkins, S., Nolin, T., Baumberger, T., Anderson, J., & Chadwick-Dias, A. (1990). A longitudinal study of language acquisition in autistic and Down syndrome children. *Journal of Autism and Developmental Disorders*, 20, 1–21.

India and Autism

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Historical Background

In India, the history of autism dates back to 1960s and is marked by initial sporadic efforts at clinical

descriptions and research followed by a more sustained progress in clinical services, research, and policies. The first case report of *autism* appeared in an Indian journal in 1962 (Bassa 1962). There were a few publications in the 1960s and 1970s referring to infantile autism, with a steady flow after the 1990s. Before the 1980s, awareness of the disorder, both among professionals and the general public was low, that increased gradually (Daley 2004). In the next two decades, i.e., from late 1980s to early 2000s, there were major developments in the field, in terms of services, legislation, policy, and capacity building. Nongovernment organizations and parent led organizations primarily for advocacy for children and adults with autism and their families were established (e.g., Tamanna, New Delhi; Action for Autism, New Delhi). Soon, these organizations forayed into spreading awareness, providing special educational services, resource building through training of teachers, and later quality research (Barua and Daley; Daley 2004). Alongside, two major developments occurred, the enactment of the National Trust for the Welfare of Persons with Autism, Cerebral Palsy, Mental Retardation and Multiple Disabilities Act, 1999 (NTA, 1999). This Act was formulated to provide for the constitution of a trust at the national level for the welfare of persons with autism along with other disorders and disabilities. The NTA was the first time *autism* was recognized as a distinct condition by legislators and policy makers. This opened the doors for government-driven and government-funded initiatives for the welfare of children and adults with autism in India. In early 2000s, the Rehabilitation Council of India (RCI), an autonomous body under the Union Ministry started a 2-year Diploma in Special Education (for autism spectrum disorders) course in four centers. The DSE (ASD) course designed in 2003 was the first of its kind to develop human resource in the field of ASD in India (RCI 2014).

The Central Board of Secondary Education (CBSE), an autonomous body under the Union Ministry of Human Resource Development, in 2009, introduced suitable modifications and concessions in the examination system to meet the special needs of students with disabilities such as

extra time for completion of examination paper, facility of scribe, and exemption from second and third language courses with choice of study subjects (CBSE, year not mentioned). The Right to Education (RTE) Act, 2009 and *Sarva Shiksha Abhiyan* (literal translation – Campaign for Education for All), the Government of India's flagship program for achievement of universalization of education, have a mandate for inclusive education for all children with disabilities including autism and intellectual disability (SSA, GOI). Parallel to this, the Rights of Persons with Disabilities Act (2016) that repeals the Persons with Disabilities (Equal Opportunities, Protection of Rights, and Full Participation) Act, (PDA) (1995) has included autism as one of the disabilities. The new Act provides a mandate for inclusive education, teachers' training, vocational training, and rehabilitation. Inclusive Education for the Disabled at Secondary Stage (IEDSS) was launched in April 2009, where assistance for the inclusive education of children with disabilities including autism, as well as funds for identification and assessment, assistive devices, allowance for transport, escorts, readers, uniforms, books, and stationery have been provided.

Another important development has been the recognition of child and adolescent psychiatry as an academic discipline. The Medical Council of India has made it mandatory for all the departments and institutes of psychiatry imparting post graduate degree (MD) in Psychiatry to have child and adolescent psychiatry services. There have been sustained efforts for providing specialized training in Child and Adolescent Psychiatry (CAP) to psychiatry postgraduates. A postdoctoral fellowship in Child and Adolescent Psychiatry was started in Vellore, Tamil Nadu, and National Institute of Mental Health and Neurosciences (NIMHANS), Bengaluru, followed by starting of a 3-year DM course in CAP in NIMHANS and Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh. The Medical council of India recognizes DM course in Child and Adolescent Psychiatry and has framed the curricular requirements and examination pattern in India. Also, a fellowship program in Developmental Pediatrics and a DM course in Pediatric Neurology have been started at PGIMER, Chandigarh.

Legal Issues, Mandates for Service

The National Trust (NT) established under the NTA, 1999 is an autonomous organization that aims to enable and empower persons with disability (including autism) to live independently and within or as close to the community to which they belong through its various agencies and schemes. The latter include scholarships, training, employment assistance, and health insurance among others. The Trust promotes measures for the care and protection of persons with disability by appointment of guardians and trustees, facilitates realization of equal opportunities, protection of rights, and full participation of these individuals and thus shares some aims with that of PDA, 1995. It also extends support to registered non-governmental organizations to provide need-based services ([National Trust](#), year not mentioned). However, the main aim of the NT remains to answer the worry of parents, "*What will happen to my child when I am no more?*"

The provisions of the Rights of Persons with Disabilities Act (2016) aim to protect the rights of these individuals as envisaged in the United Nations Convention on the Rights of Persons with Disabilities (2006), lay down specific measures to promote and facilitate inclusive education, vocational training and employment, social security, health care, insurance schemes, and rehabilitation. Chapter 3 of the Act specifically deals with education of these individuals, such as mandate for establishing adequate number of teacher training institutions, training and employing teachers who are trained in teaching children with disabilities, training of professionals and staff to support inclusive education and making necessary changes in the curriculum and examination system (Rights of Persons with Disabilities Act, 2016).

Sharing the spirit of the previous two Acts, the RTE Act (2009) addresses the important right, i.e., right to full time elementary education of satisfactory and equitable quality in a formal school which satisfies certain essential norms and standards. As mentioned earlier, inclusive education for children with autism is one of the mandates. With the passage of the RTE Act, changes have

been incorporated into the SSA approach, strategies, and norms. These children are recognized as those with special needs – CWSN. Schools are required to employ trained resource teachers for inclusive education of these children. The RCI Act and the RCI provide for the training curriculum and program of these teachers.

Overview of Current Treatments and Centers

Professionals involved in care are psychiatrists, psychologists, pediatricians, speech therapists, and occupational therapists, who work either individually or in liaison with each other. Treatment and care is provided by both medical institutions and nongovernmental social organizations. In the government sector, most of the cases are seen in the departments of psychiatry and pediatrics of general hospitals and apex teaching institutes like Postgraduate Institute of Medical Education and Research (PGIMER), All India Institute of Medical Sciences (AIIMS), and National Institute of Mental Health and Neurosciences (NIMHANS), etc. Only a few of these departments (of psychiatry) run special child and adolescent clinics, while still fewer have dedicated child and adolescent psychiatric units. However, the number of departments establishing such services is steadily increasing. Still, lack of trained manpower remains a serious handicap (Malhotra and Vikas 2005). Also, there is no specialized center for autism run by the government.

Few private centers which provide specialist care are out of reach of common people (Malhotra and Vikas 2005). There are several treatment and educational centers run by NGOs or parent associations and societies. Some of these have made notable contributions such as promoting greater awareness among professionals, conducting teacher and parent training through courses, workshops, and live demonstrations. Action for Autism (AFA), Academy for Severe Handicaps and Autism (ASHA) are some of the important organizations. AFA practices “open-door teaching method” in which the techniques used around the world are tested and modified to the needs of

Indian children. However, overall, there is a huge skew with most centers located in metropolitan cities (for example, Mumbai alone has 80 registered centers, while 2–4 centers each in other states) and almost all centers are in the state capitals (complete list available at: Autism Information and Resource Centre, an initiative of the National Trust, <http://www.autismresourcecenter.in/Default.aspx>).

Majority of the treatment protocols involve parents and are aimed at training the parents. Principles are largely drawn from TEACCH approach and Applied Behavioral Analysis (Malhotra et al. 2002, 2010). The strategies include behavioral techniques aimed at enhancing eye to eye contact, reduction of maladaptive behavior, structuring time, activities and physical environment (Malhotra et al. 2002), sensory integration, speech therapy, and picture exchange communication systems (Malhotra et al. 2010). Parent counseling and emotional support to parents have also been integral to treatment (Malhotra et al. 2002). There have been few other therapeutic interventions, e.g., ComDEALL (Karanth et al. 2010), Makaton language program, used at select centers. Few centres for complementary and alternative forms of medicine such as homeopathy and Unani also offer treatment for autistic children. Also, many families, as high as 90% opt for or combine the use of complementary and alternative medicines (Krishnamurthy 2008).

Overview of Research Directions

As mentioned earlier, autism research can be dated back to 1960s and 1970s, though it progressed sluggishly before the 2000s. Malhotra and Vikas (2002), Malhotra (2006) and Vijay Sagar KJ (2011) have well-reviewed the research in autism in India in the preceding years. Since late 1980s, several descriptions and observational studies (such as Srinath et al. 1989, Kar et al. 1993, Jaydeokar et al. 1997, Malhotra et al. 2003) appeared from tertiary psychiatric centers across the country in reputed national and international journals. Research in epidemiology of ASD is sparse (Malhotra and Kate 2015) and a

cohesive picture of the prevalence and incidence of autism spectrum disorder is missing in India (Naik 2015). Relation between social maturity and problem behaviors in children with comorbid intellectual disability have been explored (Ganaie et al. 2015). Freeth et al. 2013 conducted a cross-cultural study on autistic traits in India, UK, and Malaysia.

Apart from epidemiological surveys, research areas include descriptions and observational studies, interventional studies, genetic and neurobiological research, health systems research, some culturally oriented research on pathways to care, professionals and parent experiences/orientation, and psychometric validation of autism scales, development of Indian scales (such as Indian Scale for assessment of autism, NIMH, India), reviewed extensively in two publications (Naik 2015; Malhotra and Kate 2015).

For example, there are studies dealing with intervention programs (e.g., parent training programs, PECS, ComDEALL) (Malhotra et al. 2002, 2010; Karanth et al. 2010). Neurobiological studies have included assessment of CSF biochemistry (Narayan et al. 1993) and urinary metabolites (Damodaran and Arumugam 2011), serum levels of transcription factor nuclear factor-kappa B (Naik et al. 2011), chemical analysis of nail and hair (Priya and Geetha 2011) and BERA (Shivashankar and Satishchandra 1989). Cerebral perfusion and metabolism has been studied using PET (Anil Kumar 2012) and SPECT (Gupta and B.V. Ratnam BV. 2009). Both the studies used age-matched controls, while the former study (Anil Kumar 2012) also evaluated its association to behavior and cognitive functioning. Also, there have been efforts focusing on genetics of autism, such as studying *Engrailed-2* gene (Manjunatha et al. 1989), serotonin transporter promoter variants (Guhathakurta et al. 2006), and folate metabolic pathway in Indian patients (Mohammad et al. 2009) and relation to fragile X (Sen et al. 2010; Balasubramanian et al. 2009; Suvrathan and Chattarji 2011).

Socio-cultural issues including parental experiences and perspectives, met and unmet needs of families, parental stress, coping, and parenting styles (Desai et al. 2012; Divan et al. 2012;

Tripathi 2015; Minhas et al. 2015) have been explored. Daley et al. (2014) have attempted to explore routines of adults with autism using a mixed methods approach.

Besides these studies, work has been done in the areas of psychometric validation of autism scales and lately, development of indigenous scales. Recently, Indian Scale for Assessment of Autism (ISAA) has been developed as a measure of symptom severity, and there have been several studies reporting on its reliability and validity (Chakraborty et al. 2015; Mukherjee et al. 2015; Patra and Arun 2011). The ISAA has been recommended for purposes of disability certification as per the guidelines of the Government of India (GOI Notification dated 25th April, 2016). The International Clinical Epidemiology Network (INCLIN) Trust group has developed the INCLIN Diagnostic Tool for Autism Spectrum Disorder (INDT-ASD), and it has been found to have good diagnostic accuracy (Juneja et al. 2014). The tool has been recommended for use in conjunction with the ISAA (GOI Notification dated 25th April, 2016), though larger field trials are needed for ascertaining the psychometric properties and diagnostic stability of the tool (Wong and Singhal 2014).

There have been a few interventional studies. Malhotra et al. (2002) offered a parent intervention package including behavioral, supportive, and educational techniques, delivered in 3–6 sessions of 45–60 min each and found that on the whole parents found this brief contact helpful. Malhotra et al. (2010) studied effects of picture exchange communication system on communication and behavioral anomalies in autism and found improvement in the target behaviors. Karanth et al. (2010) studied the efficacy of an indigenous early intervention program for children with autism spectrum disorders that specifically targets developmental issues in the areas of motor, communication, cognitive, social, and emotional skills in a small sample of 30 children. A small cross-sectional study that used an autism treatment evaluation checklist pointed towards the need for training in order to improve the communication and social interaction in children with autism (Ghosh et al. 2015). Divan et al. (2015) developed

the parent-mediated intervention for Autism Spectrum Disorder in South Asia (PASS) intervention, which is a cultural adaptation of the Preschool Autism Communication Therapy (PACT) program. Harshini and Preeti (2017) have proposed an interventional model deliverable at primary care in nonspecialist setting that focuses on parental training at the center focusing on joint attention, imitation, adaptive and social engagement skills and encouraging parents to engage the child in home-based training. Overall, in resource-limited settings such as those in India, it is intuitive to consider parents as the instrument for therapeutic intervention. However, robust studies are needed to establish the efficacy of indigenously developed or culturally adapted intervention programs before these can be recommended on a larger scale.

In 2010–2011, the Indian Council of medical Research (ICMR) announced plans to conduct extensive and in-depth research in the area of ASDs. Since then, several projects addressing early diagnosis and prevalence studies, evolution of diagnostic thresholds criteria for Indian population, standardization of multidisciplinary assessment, functional imaging studies, correlational studies with neuro-psychiatric morbidities, standardization of therapeutics, genetic studies (e.g., REELIN gene, MeCP2, β -amyloid precursor protein), biochemical markers, intervention studies and services for care, development of training modules for parents & children, development of modules of care at schools and home, special education and remediation, and speech therapy and sensory integration studies have either been launched or are in the pipeline. ICMR has also initiated collaborative projects with Autism Speaks (<http://www.icmr.nic.in/>). The development of INCLIN tools and PASS intervention has been the result of such international collaborations. The NIMH, Secunderabad, have started work on a project studying effectiveness of sensory integration therapy on sensory processing and adaptive behavior (NIMH; <http://www.nimhIndia.org/R&D%20ongoing%20projects%2012-13.pdf>). NIMHANS Bangalore is working towards developing a hospital-based Autism Registry.

Policy and Training

As mentioned earlier, the RCI started Diploma in Special Education (ASD) in four centers; the government run premier institute, National Institute for Mentally Handicapped (NIMH), Secunderabad, India, being one among these. Since then, the course curriculum has been revised every 5 years, the latest revision being in 2014 (RCI, 2014). This 2-year course is now conducted in over 20 institutes recognized by the RCI. The trained teachers can work in regular schools as resource teachers, in special educational settings, and in clinics and hospital-based departments. The RCI along with certain organizations such as Action for Autism conducts periodical workshops and seminars for awareness and training of teachers. Under the SSA program, there are detailed modules and curriculum for training of teachers in educational and behavioral methods for autism, intellectual disability, and other disabilities (Sarva Shiksha Abhiyan website). Also, the postdoctoral fellowships and DM courses shall increase the specialized manpower required in this field in the near future.

With the mandates of the existing and soon to be enacted Acts, deliberately designed policies for capacity building and research, and sustained efforts across various fields such as medical research, education and rehabilitation, and legal, the stage is set for the scenario of autism services and research to change dramatically for the good in India.

References and Reading

- Action for Autism. <http://www.autism-india.org/>
- Anil Kumar, B. N. (2012). *PET scan in autistic spectrum disorder* (M.D. thesis (2009–2012)). Department of Psychiatry, Postgraduate Institute of Medical Education and Research, Chandigarh.
- Autism Information and Resource Centre, An initiative of National Trust for the Welfare of Persons with Autism, Cerebral Palsy, Mental Retardation & Multiple Disabilities. Ministry of Social Justice and Empowerment, Government of India. <http://www.autismresourcecenter.in/Default.aspx>
- Balasubramanian, B. V., Bhatt, C., & Goyel, N. (2009). Genetic studies in children with intellectual disability

- and autistic spectrum of disorders. *Indian Journal of Human Genetics*, 15, 103–107.
- Barua, M., Daley, T. C. Autism. Published for Rehabilitation Council of India, Government of India, New Delhi. Available at: <http://www.rehabcouncil.nic.in/writereaddata/autism.pdf>. Last accessed on 11 July 2015.
- Bassa, D. M. (1962). A case of early infantile autism. *Indian Journal of Psychiatry*, 4(2), 73–76.
- Chakraborty, S., Thomas, P., Bhatia, T., Nimgaonkar, V. L., & Deshpande, S. N. (2015). Assessment of severity of autism using the Indian scale for assessment of autism. *Indian Journal of Psychological Medicine*, 37, 169–174.
- Central Board of Secondary Education, Ministry of Human Resource Development, Government of India (2009). Available at: <http://cbseacademic.in/>. Last accessed on 11 July 2015.
- Daley, T. C. (2004). From symptom recognition to diagnosis: Children with autism in India. *Social Science and Medicine*, 58(7), 1323–1335.
- Daley, T. C., Weisner, T., & Singhal, N. (2014). Adults with autism in India: A mixed-method approach to make meaning of daily routines. *Social Science & Medicine*, 116, 142–149.
- Damodaran, L. P., & Arumugam, G. (2011). Urinary oxidative stress markers in children with autism. *Redox Report*, 16(5), 216–222.
- Desai, M. U., Divan, G., Wertz, F. J., & Patel, V. (2012). The discovery of autism: Indian parents' experiences of caring for their child with an autism spectrum disorder. *Transcultural Psychiatry*, 49(3–4), 613–637. <https://doi.org/10.1177/1363461512447139>.
- Divan, G., Vajartkar, V., Desai, M. U., Strik-Lievers, L., & Patel, V. (2012). Challenges, coping strategies, and unmet needs of families with a child with autism spectrum disorder in Goa, India. *Autism Research*, 5(3), 190–200.
- Divan, G., Hamdani, S. U., Vajartkar, V., Minhas, A., Taylor, S., Aldred, C., et al. (2015). Adapting an evidence-based intervention for autism spectrum disorder for scaling up in resource-constrained settings: The development of the PASS intervention in South Asia. *Global Health Action*, 8, 27278. <https://doi.org/10.3402/gha.v8.27278>.
- Freeth, M., Sheppard, E., Ramachandran, R., & Milne, E. (2013). A cross-cultural comparison of autistic traits in the UK, India and Malaysia. *Journal of Autism and Developmental Disorders*, 43(11), 2569–2583. <https://doi.org/10.1007/s10803-013-1808-9>.
- Ganaie, S. A., Beigh, A. M., Mir, S. M., Shah, S. A., Hussain, A., Dar, A. H., et al. (2015). Social maturity and problem behaviour in children with autism spectrum disorders and intellectual disabilities. *Neuropsychiatry*, 5(1), 16–24.
- Ghosh, A., Mahajan, P. B., Mishra, B. R., Mahapatra, S. C., & Nanda, P. (2015). Exploring Health Situation of Indian Children with Autism Spectrum Disorder Using Autism Treatment Evaluation Checklist (ATEC) in an Urban Area of Odisha: A Case Study. *Journal of Clinical and Diagnostic Research*, 9(12), VC05–VC08.
- Government of India (1999). National Trust for the Welfare of Persons with Autism, Cerebral Palsy, Mental Retardation and Multiple Disabilities Act. Ministry of Social Justice and Empowerment, Government of India, New Delhi, India. Available at: <http://socialjustice.nic.in/pdf/ntact1999.pdf>. Last accessed on 11 July 15.
- Government of India (2014) Rights of Persons with Disabilities Bill. Government of India, New Delhi. <http://www.prsindia.org/uploads/media/Person%20with%20Disabilities/The%20Right%20of%20Persons%20with%20Disabilities%20Bill.pdf>. Last accessed on 11 July 15.
- Guhathakurta, S., Ghosh, S., Sinha, S., Chatterjee, A., Ahmed, S., Chowdhury, S. R., et al. (2006). Serotonin transporter promoter variants: Analysis in Indian autistic and control population. *Brain Research*, 1092, 28–35.
- Gupta, S. K., & B.V. Ratnam BV. (2009). Cerebral perfusion abnormalities in children with autism and mental retardation: A segmental quantitative SPECT study. *Indian Journal of Pediatrics*, 46(2), 161–164.
- Harshini, M., & Preeti, K. (2017). Autism Behavioural Interventional Research in low-resource settings: Overcoming prevailing challenges; an Asian perspective. *Asian Journal of Psychiatry*. <https://doi.org/10.1016/j.ajp.2016.12.004>.
- Indian Council of Medical Research. <http://www.icmr.nic.in/>
- Jaydeokar, S., Bal, G., & Shah, N. (1997). Childhood disintegrative disorder. *Indian Journal of Psychiatry*, 39, 85.
- Juneja, M., Mishra, D., Russell, P. S. S., Gulati, S., Deshmukh, V., Tudu, P., et al. (2014). INCLEN diagnostic tool for autism spectrum disorder (INDT-ASD): Development and validation. *Indian Pediatrics*, 51(5), 359–365.
- Kar, N., Khanna, R., & Kar, G. C. (1993). Autistic features in children with mental retardation. *Indian Journal of Psychiatry*, 39(4), 304–308.
- Karanth, P., Shaista, S., & Srikanth, N. (2010). Efficacy of communication DEALL – An indigenous early intervention program for children with autism spectrum disorders. *Indian Journal of Pediatrics*, 77(9), 957–962.
- Krishnamurthy, V. (2008). A clinical experience of autism in India. *Journal of Developmental and Behavioral Pediatrics*, 29(4), 331–333.
- Malhotra, S. (2006). Autism: An experiment of nature. *Journal of Indian Association for Child and Adolescent Mental Health*, 2(1), 9–17.
- Malhotra, S. & Vikas, A. (2002). Pervasive developmental disorders: Indian scene. *Journal of Indian Association for Child and Adolescent Mental Health*, 1(3), 5.
- Malhotra, S., & Kate, N. (2015). Research endeavors in child psychiatry in India 1 and 2. In S. Malhotra & S. Chakrabarti (Eds.), *Developments in psychiatry in India* (pp. 215–254). Springer. https://doi.org/10.1007/978-81-322-1674-2_12.

- Malhotra, S., & Vikas, A. (2005). Pervasive Developmental Disorders: Indian Scene. *Journal of Indian Association for Child and Adolescent Mental Health*, 1(3), Article 5.
- Malhotra, S., Chakrabarti, S., & Nehra, A. (2002). Psychological interventions with parents of autistic children. *Indian Journal of Psychiatry*, 44(2), 108–117.
- Malhotra, S., Chakrabarti, S., Gupta, N., Kumar, P., & Gill, S. (2003). Pervasive developmental disorders and its subtypes: Sociodemographic and clinical profile. *German Journal of Psychiatry*.
- Malhotra, S., Rajender, G., Bhatia, M. S., & Singh, T. B. (2010). Effects of picture exchange communication system on communication and behavioural anomalies in autism. *Indian Journal of Psychological Medicine*, 32(2), 141–143.
- Manjunatha, K. R., Narayanan, H. S., Rao, B. S. S. R., Srinath, S., & Girimaji, S. (1989). Cryptogenetic investigations in autistic children: a preliminary study on the detection of fragile X chromosome. *NIMHANS Journal*, 7(2), 163–167.
- Minhas, A., Vajaratkar, V., Divan, G., Hamdani, S. U., Leadbitter, K., Taylor, C., Aldred, C., Tariq, A., Tariq, M., Cardoza, P., Green, J., Patel, V., & Rahman, A. (2015). Parents' perspectives on care of children with autistic spectrum disorder in South Asia - Views from Pakistan and India. *International Review of Psychiatry*, 27(3), 247–256. <https://doi.org/10.3109/09540261.2015.1049128>.
- Mohammad, N. S., Jain, J. M., Chintakindi, K. P., Singh, R. P., Naik, U., & Akella, R. R. (2009). Aberrations in folate metabolic pathway and altered susceptibility to autism. *Psychiatric Genetics*, 19(4), 171–176.
- Mukherjee, S. B., Malhotra, M. K., Aneja, S., Chakraborty, S., & Deshpande, S. (2015). Diagnostic accuracy of Indian Scale for Assessment of Autism (ISAA) in children aged 2–9 years. *Indian Pediatrics*, 52(3), 212–216.
- Naik, U. S. (2015). Autism spectrum disorder: 70 years on and the plot thickens. In S. Malhotra & S. Chakrabarti (Eds.), *Developments in psychiatry in India* (pp. 275–311). New Delhi: Springer. https://doi.org/10.1007/978-81-322-1674-2_15.
- Naik, U. S., Gangadharan, C., Abbagani, K., Nagalla, B., Dasari, N., & Manna, S. K. (2011). A study of nuclear transcription factor-kappa B in childhood autism. *PloS One*, 6(5), e19488.
- Narayan, M., Srinath, S., Anderson, G. M., & Meundi, D. B. (1993). Cerebrospinal fluid levels of homovanillic acid and 5-hydroxyindoleacetic acid in autism. *Biological Psychiatry*, 33(8-9), 630–635.
- National Trust. The National Trust, Ministry of Social Justice and Empowerment, Government of India. Available at: http://www.thenationaltrust.co.in/nt/index.php?option=com_content&task=view&id=94&Itemid=143. Last accessed on 11 July 2015.
- Patra, S., & Arun, P. (2011). Use of Indian scale for assessment of autism in child guidance clinic: an experience. *Indian J Psychol Med.*, 33, 217–219.
- Priya, L., & Geetha, A. (2011). Level of trace elements (copper, zinc, magnesium and selenium) and toxic elements (lead and mercury) in the hair and nail of children with autism. *Biological Trace Element Research*, 142(2), 148–158.
- Rehabilitation Council of India (2014). D.Ed. Special Education (Autism Spectrum Disorder). Syllabus norms, regulations & course content.” Rehabilitation Council of India. (Statutory Body under Ministry of Social Justice & Empowerment), Government of India, New Delhi. Available at: <http://www.rehabcouncil.nic.in/writereaddata/dedasd.pdf>. Last accessed on: 11 July 2015.
- Right to Education (2009/10). Ministry of Human Resource Development, Government of India. <http://mhrd.gov.in/rte>
- Sarva Shiksha Abhiyan. Ministry of Human Resource Development, Government of India. <http://ssa.nic.in/>. Last accessed on 11 July 2015.
- Sen, B., Singh, A. S., Sinha, S., Chatterjee, A., Ahmed, S., Ghosh, S., & Usha, R. (2010). Family-based studies indicate association of Engrailed 2 gene with autism in an Indian population. *Genes, Brain and Behavior*, 9(2), 248–255.
- Shivashankar, N., & Satishchandra, P. (1989). Auditory brainstem responses in autistic children. *Nimhans Journal*, 7(2), 159–162.
- Srinath, S., Chowdhury, J., Bhide, A. V., Narayanan, H. S., & Shivaprakash. (1989). Descriptive study of infantile autism. *NIMHANS Journal*, 7(1), 77–81.
- Suvrathan, A., & Chattarji, S. (2011). Fragile X syndrome and the amygdale. *Current Opinion in Neurobiology*, 21(3), 509–515.
- Tamana. <http://www.tamana.org/AutismCenter.aspx>
- Tripathi, N. (2015). Parenting style and parents' level of stress having Children with Autistic Spectrum Disorder (CWASD): A study based on Northern India. *Journal of Neuropsychiatry*, 5(1), 42–49.
- Vijay Sagar, K. J. (2011). Research on autism spectrum disorders in India. *Andhra Pradesh Journal of Psychological Medicine*, 12(1), 69–72.
- Wong, C. M., & Singhal, S. (2014). INDT-ASD : An autism diagnostic tool for Indian children developmental pediatrician's perspective. *Indian Pediatrics*, 51(5), 355–356.

Indicator

► Milestone

Indiscriminate Friendliness

► Attachment Disorder

Indiscriminate Sociability

- [Attachment Disorder](#)

Indiscriminate Social Behavior

- [Attachment Disorder](#)

Indiscriminately Social/Disinhibited Attachment Disorder

- [Attachment Disorder](#)

Individual Appropriates

- [Developmentally Appropriate Practice \(DAP\)](#)

Individual Education Plan

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Definition

Individual Education Program

An Individualized Education Program (IEP) is provided to students with disabilities under the Individuals with Disabilities Education Improvement Act (IDEIA). Current law (IDEIA) dictates that all students who are eligible for educational supports and specialized services have an IEP. The IEP is a legal document outlining the necessary services and supports for children with disabilities (i.e., who have an educational eligibility) ages 3 years through 22 years. IEPs are created in collaboration with parents, teachers, and

therapists working with the student. The purpose of the IEP is to outline educational goals, supports, services, and placement for individual students with disabilities and identify who is responsible for carrying out the student's program. An IEP serves as the framework for individualized instructional programs for students with disabilities. The IEP addresses all of a student's educational needs. It is a written program of educational services and supports developed for each student eligible for services in accordance with the criteria outlined by each state's board of education. The IEP includes procedural safeguards for parents of students with disabilities. The procedural safeguards outline the rights of students and parents. Parents are given a copy of their procedural safeguards at each IEP meeting. The procedural safeguards are determined by each state board of education. These include (but are not limited to) free and appropriate public education for all children age 3 and older, prior written notices for all meetings and decisions, a description of instructional and placement options, and descriptions of all evaluation procedures.

An IEP is required for all students older than 3 years who need specialized services. In most states, children younger than 3 who have an Individual Family Service Plan (IFSP) will transition to an IEP at age three. In some states, this transition occurs at age 5 or once the child enters kindergarten. The transition from an IFSP to an IEP always involves a reevaluation (i.e., parent and team input, informal assessments and observations, and standardized assessments). The eligibility determinations are made by the state-appointed evaluation teams based on the extent to which the disability impacts the students' educational performance.

Historical Background

IEPs are mandated by federal law, specifically the Individuals with Disabilities Act, which was recently amended in 2004. The major purpose of this law is to ensure all children with disabilities to have access to free and appropriate education

(FAPE). The IEP outlines the services and supports needed to ensure the child receives FAPE.

Current Knowledge

IEP Process

For students 3 years and older, the initial IEP process begins with an evaluation of the child's present level of development. An eligibility determination is made based on the assessment information gathered. This includes information gathered through parent interviews, informal classroom observations or assessments, and formal or standardized assessments. The state identified evaluation team determines eligibility for services based on the extent to which child's disability negatively affects educational performance. For example, the evaluation team might determine a child's autism is negatively affecting his ability to communicate, interact with others, access the general education curriculum, access materials in the classroom, and meet grade level expectations. The team might determine this child meets the eligibility requirements and needs specialized services. However, not all children with a medical diagnosis of autism will be eligible for educational services. For example, the evaluation team might determine a child with higher functioning autism (i.e., has above average IQ) can access the general education curriculum, materials, and have positive interactions with peers and adults with the typical classroom-wide structure and supports. Once the child is determined to be eligible for specialized services and an IEP is developed, the child's eligibility is reevaluated every 3 years. The student's IEP is revised each year at an annual review.

IEP Components

There are several components to an IEP (e.g., the educational team, eligibility category, placement, accommodations, strengths, goals, and services). These will be described in more detail.

IEP Team

The student's educational team is made up of a variety of individuals who collaborate to develop

an educational program that will meet the student's needs. The IEP team for students with autism should include the parents, general education teacher, special education teacher, and school district representative (e.g., the case manager, guidance counselor, administrator, or professional at the district level). Depending on the needs of the student, the IEP team also might include an autism specialist, speech/language therapist, occupational therapist, school psychologist, behavior specialist, and the student when appropriate. The special education teacher is a key member of the IEP team for students with autism (Hall 2007). Thus, special education teachers serving students with autism must have specific experience and coursework related to evidence-based practices for students with autism. Occasionally, school districts will hire autism specialists (i.e., school personnel with specific experience and training in evidence-based practices with students with autism) to support students with autism and help teachers in assessment, instructional planning and implementation, and progress monitoring of students with autism (Hall 2007). In sum, the purpose of the IEP team is to determine the extent of the student's needs, design an instructional program, implement the program, evaluate the program, and ensure the student receives the appropriate supports and services. Thus, the role of the IEP does not begin and end at the IEP meeting – it is an ongoing effort. Effective communication among team members is essential to ensure the IEP is followed and the child is making appropriate progress toward goals and objectives.

Educational Needs

The next major section of the IEP is the students' educational needs. This section indicates the student's eligibility category and specific information related to how the disability impacts the child's educational performance. Eligibility categories might include autism, other health impaired, emotional delays, cognitive delays, specific language impairment, speech impairments, visual or hearing impaired, or learning disability. This section includes a statement with specific information related to how the student's disability

impacts his educational performance or academic achievement.

Current Assessment Information

This section includes a statement about the student's present level of development, functioning, and educational performance, including information about what skills the student has mastered and what skills are developing. This section also should include information related to the child's current academic and behavioral repertoire, learning style, interests, and the impact of the student's disability on academic, social, adaptive, communication, or motor skills. This can include information related to assessment results, discipline reports, grades, observational notes, and attendance information. The student's academic and behavioral strengths also should be clearly delineated in this section. Parents *and* teachers should provide information about the student's behavioral and academic strengths. This assessment information should be directly linked to instructional planning. The team will develop a list of areas of instructional priorities based on this assessment information. Successful instructional programs are directly linked to the student's strengths, assessment results, interests, learning style, and parent priorities and concerns (Boutot and Myles 2011).

Educational Needs

The next section on the IEP delineates the student's needs, instructional priorities, and goals. The student's needs are developed based on the assessment results, levels or area of impact of the disability, or academic performance. Annual goals and short-term objectives should be written for each area in which the student has needs. The areas of need for students with autism should include communication, social skills, and affected academic areas (e.g., reading, math). Many students with autism will need goals in fine motor, gross motor, or adaptive skills (e.g., independent functioning). The student's needs should be directly linked to the student's goals. Goals are the skills that the student will attain within the year or other specified amount of time. Goals should be functional, generative, measureable,

and instructional. Functional goals are directly related to skills the student will need for independent performance within daily routines and activities. Generative goals are related to skills the child will need across settings, people, materials, and activities. Measurable goals are clearly operationalized so that the student's progress can be clearly and efficiently monitored over time. Instructional refers to goals that will occasion opportunities for teaching throughout the student's day. Goals also should be written in a positive manner with clear, professional language (e.g., instead of "Elisa will stop hitting peers," the goal should read "Elisa will verbally ask a teacher for help").

Examples of annual goals for children with autism are:

1. For a preschooler: Sammy will appropriately respond (e.g., verbally or with gestures) to social initiations from a peer during classroom routines and activities. We will know he can do this when he appropriately responds to 5 peer initiations per day for 3 consecutive days.
2. For an elementary school student: Juan will follow classroom rules and directives (e.g., sit in chair, raise hand to ask questions, walk in the halls, etc.) given visual and verbal prompts. We will know he can do this when he follows 10 classrooms rules and directives for 2 days in a row.
3. For a high school student: Trevonte will ask questions of others regarding topics initiated by self or others to sustain conversation. We will know he can do this when he participates in five conversations per day for 3 consecutive days.

Accommodations or Modifications

This section delineates accommodations or modifications necessary to support the student's learning. Accommodations are changes made to the instruction or assessment to support the student and provide access to the general education curriculum without altering the content. Accommodations for students with autism might include visuals or graphic organizers, specialized seating arrangement, visual rather than written responses,

adapted keyboards, or having material read aloud. Accommodations do not change grade level or academic expectations. Modifications refer to changes made to the assessment or general education curriculum to meet the needs of the student. Accommodations are required by law to ensure students with disabilities have an equal opportunity to learn and access the general education curriculum. Modifications are made when the expectations are not appropriate for the student. Examples of modifications for students with autism might include the following: a student works on addition, while peers work on division; a student stacks or sorts materials, while peers work on a science experiment; or a student is given a test on continents, while peers are tested on geography or countries across the world. Modifications should target the highest level possible to meet the student's needs (i.e., closest to the general education content as possible) and only used when absolutely necessary.

Related Services

The next section of the IEP describes the services necessary to support the student and supplement the services provided in the classroom. Related services are required to ensure the student is successful in the classroom. Related services for students with autism might include speech/language therapy, occupational therapy, assistive technology, transportation, and counseling. This section will describe these services in detail. The related services section includes the frequency of services (e.g., twice per week for 30 min), the location of these services (e.g., classroom, therapy room), the type of services (e.g., direct service to the individual child, group services with the child and other children with disabilities, or consultation to the teacher or parent), and the person providing these services. Indirect services (i.e., consultation and coaching) are often provided by autism specialist to students with autism in general or specialized classrooms. This might include consultation to the general education teacher about designing a predictable, visually rich environment to support the student with autism or the special education teacher about implementing an evidence-based strategy for teaching communication or social

skills. These services should be provided directly in the classroom when appropriate to support the student with autism in accessing the classroom curriculum and interacting with peers and materials.

Assistive Technology

The next section on the IEP is related to assistive technology for students with disabilities. This includes technologies used by the student to function within the classroom and community. For students with autism, this might include augmentative communication devices, computer software to enhance academic performance, or the use of personal digital assistants (PDAs) to promote independent transitioning and following schedules.

Setting

The IEP also will indicate the setting(s) in which the IEP will be implemented. This is referred to as the educational placement. Placement is not based on district availability or preferences. The IEP team will determine the student's placement based on the student's strengths and needs. Special education law requires that all students with disabilities be educated in the least restrictive environment and with their typical peers to the maximum extent possible. The law requires the general education classroom to be considered first, and if the student is not included in the general education, the IEP should document and specify reasons why. A continuum of placements must be considered from least restrictive (general education classrooms) to more restrictive (e.g., special classroom or school). The IEP team should consider general education classroom with embedded special education services, general education classroom with pull-out to a resource or special education classroom for part of the day, special education classroom within a general education school, or specialized school. In rare cases, service will be provided in an institution or hospital. Extensive research supports the inclusion of student with disabilities in the general education classrooms. For example, inclusive classrooms provide a context for implementing evidence-based practices, occasional social interactions

and communication among students with autism and their typical peers, which represent core skill deficits for children with autism, and research has documented more positive attitudes toward disabilities by peers when students with disabilities are included in the preschool environment (Dawson and Osterling 1997; Strain et al. 2002). If the placement is not a general education classroom, the IEP must specify the amount of time the child will participate in the general education classroom, document that opportunities for the student to interact with typically developing peers will be provided, and include a statement specifying that the least restrictive environment was considered.

Positive Behavior Supports

There also is a section on the IEP that requires the team to indicate if positive behavioral supports have been considered for the student with autism. This is an important service for many students with autism, particularly students who display stereotypic behaviors (e.g., repetitive hand flapping, body rocking), or challenging behaviors (e.g., which are often due to delays in functional communication and social skills) that might interfere with learning. This section will describe the supports (e.g., preventative strategies, replacement skills, and responses to challenging behaviors) and implementation plan.

Evaluation

The IEP also includes a section specifying how student progress will be measured and evaluated. Appropriately written goals and objectives will embed an evaluation process. However, this section ensures the student's progress is intentionally and systematically monitored over year. Progress on each goal must be measured and documented.

Postsecondary Transition

The IEP for students with autism who are 14 years old must include a section for the student's post-school plan or transition to adult care or services. The IEP team considers the student's need for transition services and documents the discussion. When appropriate, the IEP will include a statement of the needs for services and a transition

planning form. This form requires the IEP document the discussion about the student's transition to adult life after school. The form will include a postsecondary vision (e.g., the student's goals for adult life); disability-related needs; plan for supporting the student's self-determination, academic, and adaptive skills to support postsecondary vision; and the roles of school personnel, family, student, and community providers in supporting the student.

504 Plans

Students with disabilities who require accommodations must have a written plan. If the accommodations are considered reasonable, the student can have a 504 plan rather than a full IEP. The 504 plan also is developed by the student's educational team, but requires only minimal accommodations to access and succeed within the general curriculum. For students with autism, this might include a digital timer, longer testing time, or regular breaks during the day.

Future Directions

Summary and Future Directions

An Individualized Education Program (IEP) is the legal documentation of an educational plan developed with a team of professionals based on the strengths and needs of one child. IDEIA mandates that all children with special needs have a right to an IEP and free access to appropriate public education. Effective IEPs ensure that children with autism receive equitable education (the same as children without disabilities) in the least restrictive environment possible. Future research should examine components of effective IEPs and strategies for developing collaborative, effective IEPs.

See Also

- [Free Appropriate Public Education](#)
- [Individualized Plan for Employment \(IPE\)](#)
- [Individualized Transition Plan \(ITP\)](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- Bateman, B. D., & Herr, C. (2006). *Writing measurable IEP goals and objectives*. Verona: Attainment Publications.
- Boutot, E. A., & Myles, B. S. (2011). *Autism spectrum disorders*. Upper Saddle River: Pearson.
- Dawson, G., & Osterling, J. (1997). Early intervention in autism: Effectiveness, common elements of current approaches. In M. J. Guralnick (Ed.), *The effectiveness of early intervention: Second generation research* (pp. 307–326). Baltimore: Brookes.
- Hall, L. J. (2007). *Autism spectrum disorders: From theory to practice*. Upper Saddle River: Pearson.
- Ruble, L. A., McGrew, J., Dalrymple, N., & Jung, L. A. (2010). Examining the quality of IEPs for young children with autism. *Journal of Autism and Developmental Disorders*, 20, 1459–1470.
- Strain, P., McGee, G., & Kohler, F. (2002). Inclusion of children with autism in early intervention environments. In M. J. Guralnick (Ed.), *Early childhood inclusion: Focus on change* (pp. 337–363). Baltimore: Paul H. Brookes.

Individualized Education Program (IEP) Team

► Interdisciplinary Team

Individualized Family Service Plan

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Definition

An Individualized Family Service Plan (IFSP) is a legal documentation of the early intervention process for children with disabilities younger than 3 and their families. The IFSP guides the early intervention process in accordance with Part C of the Individuals with Disabilities Act (IDEA). Part C of IDEA is a federal grant program that supports states in operating a comprehensive system of early intervention for children younger than

3 years old with or at risk for disabilities and their families. As with the IEP (Individual Education Program), the IFSP is completed by a team of professionals (including the child's family and caregivers); the IFSP documents services necessary to support the family and build the family's capacity to help their child grow and develop. An IFSP is required for all children younger than 3 years who are receiving Part C early intervention services. In most states, children younger than 3 who have an IFSP will transition to an IEP at age three. In some states, this transition occurs at age 5 or once the child enters kindergarten, in which case the children have an IFSP until they are 5 years old. The transition from an IFSP to an IEP always involves a reevaluation (i.e., parent and team input, informal assessments and observations, and standardized assessments).

IEP Versus IFSP

IFSPs and IEPs are legal documents outlining the services needed for children with disabilities from birth to age 3 and from age 3 through 21, respectively. The IFSP and IEP are similar in that they are developed by a team for an individual child or student and include the goals, interventions, and supports necessary to support their learning. However, IFSPs are uniquely designed to revolve around the individual family's concerns, priorities, and resources. IFSPs include child and family outcomes relating to supporting the child's development, whereas IEPs are focused only on the student and his/her academic performance. IFSPs identify the family's natural setting and document how and when services will be provided in the family's natural settings (i.e., home, parks, child care). Also, IFSPs have an additional section (as compared to IEPs) where the non-Part C services required by the family are listed along with the funding sources for those services. IEPs focus on the special education and related services in schools. The final difference between IEPs and IFSPs lies within their purpose. In sum, the purpose of the IFSP is to identify and document the supports needed to strengthen the family's capacity to promote their child's development, whereas the IEP outlines the supports and services to be provided by professionals within schools to

promote the student's academic performance (McWilliam 2010; McWilliam et al. 1998).

Historical Background

The Individual Family Service Plan (IFSP) is part of a system of federal and state special education laws. The federal law, the Individuals with Disabilities Education Act (IDEA), specifies the IFSP and defines the basic components that must be in an IFSP. Each state further defines the specific children and families eligible for Part C services and the IFSP components.

Current Knowledge

IFSP Process

For children younger than 3, the initial IFSP process begins with an evaluation of the child's present level of development. An eligibility determination is made based on the assessment information gathered. Eligibility determinations are state-determined. Thus, although all states and eligible territories currently provide Part C services, the children and families who are eligible for these services differs across states. A few states provide Part C services to children younger than 3 who are at risk for delays and children who have delays. Eligibility determination is made based on information gathered through parent interviews (e.g., Routines Based Interview, McWilliam et al. 2009; Routines-based assessment, Woods and Lindeman 2008), informal observations or assessments in natural settings, and formal or standardized assessments. Once the child is determined to be eligible for specialized services and an IFSP is developed, the child's eligibility is reevaluated every year. The IFSP team reviews and evaluates child and family's progress on IFSP outcomes at least every 6 months.

IFSP Components

There are several components to an IFSP (e.g., the team, current assessment information, family concerns, outcomes, etc.). These will be described in more detail.

IFSP Team

The child's educational team is made up of a variety of individuals who collaborate to develop a plan to meet the child and family's needs. The IFSP team for children with autism might include the parents, autism specialist, early interventionist, speech/language therapist, and service coordinator. Depending on the needs of the child and family, the IFSP team also might include a behavioral specialist, social worker, or psychologist. If the child spends more than 15 h per week in child care, the primary care provider from the child care setting should be included on the team (McWilliam 2010). The purpose of the IFSP team is to build a collaborative relationship with the family and determine the supports necessary to build the family's capacity to promote their child's development. For children who spend more than 15 h per week in child care, this includes supporting the primary care providers in the child care setting. As with the IEP team, role of the IFSP team does not begin and end at the IFSP meeting – it is an ongoing effort. Effective communication among team members is essential to ensure the IFSP is followed, families (or child care providers) are receiving the supports they need, and the child and family are making appropriate progress toward outcomes. One member of the team is identified as the primary service coordinator, who organizes and is responsible for ensuring implementation of the IFSP. Effective IFSP teams are guided by a transdisciplinary model of service delivery (i.e., one professional provides the weekly supports with back-up from other team members (McWilliam 2010)) and use an interagency approach (involves professionals from a several agencies).

Present Levels of Development

The next section of the IFSP outlines the child's present level of development. Each family will receive a multidisciplinary assessment of their child's unique strengths and needs. The child's present level of development should be assessed across five areas: motor, cognitive, communication, social emotional, and adaptive. This information should be gathered from valid and reliable assessments and authentic observations

in the natural environments. This section should also include parent or caregiver reports about their child's preferences, strengths, and characteristics.

Family's Resources, Priorities, and Concerns

The IFSP team also will gather information about the family's resources, priorities, and concerns (e.g., Family Interest Survey, Cripe and Bricker 1993). This information can be gathered through informal conversations, interview, or assessments. A statement about the family's concerns, priorities, and resources related to enhancing their child's development is required on all IFSPs.

Child Outcomes

The next section on the IFSP delineates the child outcomes. These are developed by the IFSP team (including the family) and directly related to the assessment information. Many young children with autism will need outcomes related to social or communication skills. Outcomes should be functional, generative, measureable, instructional, and participation-based. Functional goals are directly related to skills the child will need for independent performance within the family's daily routines and activities. Generative goals are related to skills the child will use across the family's routines and materials at home, the various community settings the family and child participate in, and people the child interacts with. Measurable goals are clearly operationalized to directly link to an intervention plan for the family and so that the child's progress can be efficiently monitored over time. Instructional goals refer to behaviors and skills the family will have opportunities to practice with the child across their daily routines with the materials they have at home. Finally, goals for young children should be participation-based. This means they explicitly refer to the context in which the skill is needed to ensure everyone on the team understands how the goal helps the child participate in the family's daily activities and routines

(McWilliam 2010). The criteria, procedures, and timelines for measuring progress also are included in this section. Collaboration in developing child outcomes is essential to build the family's capacity and increase the child's participation in the family's routines and activities. Example child goals are:

1. Kenan will participate in meals, playtime (after bath, after breakfast), and routines (diapering, bath time, getting dressed, waking up) by looking at mom or dad and babbling. We will know he can do this when he looks at mom or dad's eyes and babbles three times during five activities per day for 1 week.
2. Luke will participate in breakfast, lunch, mid-afternoon snack, and dinner by feeding himself independently. We will know he can do this when he uses his hands to pick up 15 small pieces of food to put in his mouth or uses his hands to pick up his spoon to put at least 10 bites of soft foods in his mouth at each meal for 1 week.

Goals should be written so they can be embedded into daily routines and natural settings, emphasize functional skills, and increase participation in activities and settings.

Family Outcomes

The IFSP must include at least one family outcome. As with the child outcomes, the family outcomes also are based on the family's priorities, resources, and concerns. These outcomes are explicitly focused on setting goals to build the family's capacity to support their child's growth and development. The family selects their outcomes based on things they would like to start working on, including the any resources or needs identified through the assessment. Example family outcomes might be (adapted from McWilliam 2010):

1. Janice will keep Maize engaged during at least 30 min of church each week for a month so Janice can start going to church again.

2. All five members of the McDonalds family will spend 1 h each night engaged in a fun activity with the TV off.
3. Anne will rate Alyse's bedtime routine as successful and easy with 4 months.

These family outcomes are based on specific concerns reported by the family, directly related to the families current priorities, and developed in consideration of the family's available resources and include measurable criterion to ensure the team will know when the outcome has been achieved.

Natural Environments

The next section of the IFSP identifies the settings where the family spends most of their time and the activities they enjoy. The natural setting might be the home, child care, relative's house, or community center. The family's valued activities and routines are listed to identify needs and opportunities for learning. All child outcomes should be developed with the natural setting, valued activities, and daily routines in mind; the outcomes should support the child's independent participation in family routines and activities. Also, all service and supports should be provided in the natural settings. In fact, IDEA Part C regulations state: "To the maximum extent appropriate to the needs of the child, early intervention services must be provided in natural environments, including the home and community settings in which children without disabilities participate" 34CFR 303.12(b), and "natural environments means settings that are natural or normal for the child's age peers who have no disabilities" 34CFR 303.18.

Services

The next section of the IFSP delineates the specific services and supports needed to support the child and family in achieving the stated outcomes. The primary service provider will provide or coordinate the provision of material, emotional, or informational supports to build the family's capacity to promote their child's development.

Services and supports are provided through family coaching in the natural setting (often referred to as home visiting) or consultation with the child care center within the child's daily routines. Material supports might include necessary equipment (e.g., a communication device, supported seating) or basic necessities (e.g., calling an agency to help the family get diapers or housing subsidies). Emotional support might include sensitivity to the family's culture and needs, positive feedback, and responsiveness (e.g., listen to the family, respond to calls, or emails quickly). Informational support might include information about the child's disability, community resources, and strategies for providing learning opportunities throughout the day. In general, services and supports should be provided directly to the family rather than to the child (Jung 2003). In this manner, the family is supported and given the resources to provide the "intervention" all day long across daily routines and activities with relevant materials. Effective service providers recognize that the family can support their child's development by providing numerous natural learning opportunities throughout the day between scheduled home visits (McWilliam 2010). Also, supports and services should reflect the family's culture and experiences. This section also will specify the projected dates of when the services will begin, how often they will occur, and how long they will last.

Evaluation

The IFSP also includes a section specifying how the child and family's progress will be measured and evaluated. This section should include the criteria, procedures, and timelines used to measure progress toward outcomes. Appropriately written outcomes will include a measurable criterion (e.g., 5 times across 5 days, 10 times per day for 1 week). However, this section ensures the child and family's progress is intentionally and systematically monitored. Progress on each outcome must be measured and documented, and reviewed at least every 6 months. The team will assess progress and determine if the supports are effective or if changes need to be made to the

IFSP. As mentioned above, the IFSP is meant to delineate an ongoing process of support for the family and child to help the family recognize their own strengths, resources, and capacity to promote their child's development.

Transition Plan

The IFSP includes a section to plan for the transition to special education services (Part B of IDEA which refers to special education services for children 3 years and older) and an IEP, or other appropriate agencies or resources for when the child turns 3. The service coordinator must initiate this conversation and plan with the family at least 6 months prior to the child's third birthday.

Non-part C Services

The final section of the IFSP delineates the activities or services provided by agencies outside of Part C. This is meant to help the service coordinator and family integrate all service and supports. Occasionally, families are charged for these non-Part C services, which should be noted on the IFSP.

Future Directions

An Individualized Education Program (IFSP) is the legal documentation of a service plan developed with a team of professionals based on the strengths and needs of one child and their family. IDEA mandates that all children younger than three receiving Part C services have an IFSP. Effective IFSPs ensure that children with autism and their families receive early and intentional intervention in the natural setting to support the family's capacity to promote their child's development to prevent or alleviate further delays. Despite the plethora of research on the benefits of early, intentional intervention for children with or at risk for disabilities, early intervention (specifically effective family coaching) is a largely understudied topic. Future research should examine effective family coaching, home visiting, and strategies for developing effective IFSP.

See Also

- [Free Appropriate Public Education](#)
- [Individual Education Plan](#)
- [Individualized Plan for Employment \(IPE\)](#)
- [Individualized Transition Plan \(ITP\)](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- Brown, W., Thurman, S. K., & Pearl, L. F. (1993). *Family centered early intervention with infants and toddlers: Innovative cross-disciplinary approaches*. Baltimore: Paul H. Brookes Publishing.
- Cripe, J., & Bricker, D. (1993). *Family Interest Survey*. University of Oregon, unpublished assessment.
- Jung, L. A. (2003). More is better: Maximizing natural learning opportunities. *Young Exceptional Children*, 6, 21–27.
- Jung, L. A. (2010). Can embedding prompts into the IFSP form improve the quality of IFSPs? *Journal of Early Intervention*, 32, 200–213.
- Jung, L. A., & McWilliam, R. A. (2005). Reliability and validity of scores on the IFSP Rating Scale. *Journal of Early Intervention*, 27, 125–136.
- McWilliam, R. A. (2005). Assessing the resource needs of families in the context of early intervention. In M. J. Guralnick (Ed.), *A developmental systems approach to early intervention* (pp. 215–234). Baltimore: Paul H. Brookes Publishing.
- McWilliam, R. A. (2010). *Routines-based early intervention*. Baltimore: Paul H. Brookes Publishing.
- McWilliam, R. A., Casey, A. M., & Sims, J. L. (2009). The routines-based interview: A method for assessing needs and developing IFSPs. *Infants and Young Children*, 22, 224–233.
- McWilliam, R. A., Ferguson, A., Harbin, G., Porter, D. M., & Vaderviere, P. (1998). The family-centeredness of individualized family services plans. *Topics in Early Childhood Special Education*, 18, 69–82.
- Woods, J., & Lindeman, D. P. (2008). Gathering and giving information with families. *Infants and Young Children*, 21, 272–284.

Individualized Family Services Plan (IFSP) Team

- [Interdisciplinary Team](#)

Individualized Plan for Employment (IPE)

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Definition

An Individualized Plan for Employment (IPE) (also known as an Individualized Employment Plan [IEP]) is a contractual agreement between an individual with a disability and a state office of vocational and rehabilitative services. IPEs help inform the individual regarding what services they will receive and help them make informed choices regarding their vocational training. This information includes the cost, duration, and accessibility of potential services. This document is written collaboratively between the client and vocational rehabilitative counselor. The IPE must contain the following information: (1) the date that services begin and the date by which the vocational objective is to be achieved; (2) the specific services that are to be offered to the client or consumer; (3) objective criteria, evaluation procedures, and schedules for determining progress toward goals; and (4) a description of the Client Assistance Program (CAP) (Vision Aware 2010).

For each service described in the IPE, specific information must be listed. Each service must have a date by which the service will be initiated and a date by which the specific vocational goal will be reached. The IPE must describe the services offered to the client in detail. The vocational goals are to be written in a manner that is behaviorally oriented so that the vocational goal is observable and measurable. How the attainment of the goal will be assessed must be written into the IPE. The IPE should be reviewed at least annually (Joint Committee on Administrative Rules n.d.).

The list of services available to a client who is eligible for vocational and rehabilitative services from a state office includes the following:

- Counseling and guidance.
- Physical and mental restoration, including corrective surgery and ocular prosthetic devices.
- Vocational and other training services.
- Services to the family.
- Interpretive services for those who are deaf.
- Reading services.
- Personal adjustment services.
- Provision of telecommunications and other technical aids.
- Placement in suitable employment.
- Post-employment services if the client's job is in jeopardy.
- Assistance in obtaining occupational licenses, tools, equipment, and initial stocks for small businesses.
- Job coaching services.
- Assistance in obtaining other goods and services that could aid the client in obtaining employment.
- In addition, the vocational objective must contain a formal job title that is listed in the *Occupational Outlook Handbook*, with the code number, if available (Vision Aware 2010).

Current Knowledge

The range of unemployment figures varies from 50% to 75% for people with disabilities in general and over 90% for individuals with autism spectrum disorders.

Future Directions

A coordination of entitlements must be developed for young adults who are transitioning between secondary school and postsecondary education. There is potential conflict between the transition plans outlined in IDEA, the new provisions of the Higher Education Opportunity Act of 2008, and the Rehabilitation Act Amendments of 1998 in terms of which law or agency will pay for additional training if that training is conducted in an institution of higher education such as through a Comprehensive Transition and Postsecondary Program (CTP) at a college or university.

See Also

- [Comprehensive Transition Program](#)
- [HEA 2008](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Vocational Rehabilitation Act of 1973](#)
- [Vocational Training](#)

References and Reading

- Joint Committee on Administrative Rules. (n.d.). Administrative code. Title 89: Social services. Chapter IV: Department of Human Services. Subchapter b: Vocational rehabilitation. Part 572 Individualized Plan for Employment (IPE). Retrieved 29 June 2011, from <http://www.ilga.gov/commission/jcar/admincode/089/08900572sections.html>
- U.S. Congress. (1973). The Rehabilitation Act of 1973 (P.L. 93–112). Retrieved 25 July 2011, from <http://www.dotcr.ost.dot.gov/documents/ycr/REHABACT.HTM>
- Vision Aware. (2010). *What is an individualized plan for employment (IPE)?* Retrieved 29 June 2011, from <http://www.visionaware.org/individualized-plan-for-employment>
- Wehman, P. (2001). *Life beyond the classroom: Transition strategies for young people with disabilities* (3rd ed.). Baltimore: Paul H. Brookes.

Individualized Transition Plan (ITP)

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Definition

Individualized Transition Plan, or ITP, is a plan based on informal and formal assessments that is used to identify the desired and expected

outcomes by students and their families once they leave school as well as the supports needed to achieve these outcomes. This plan is developed collaboratively with the student, caregivers, vocational educators, vocational rehabilitation counselors, and current IEP team members. A thorough plan will include the following: post-secondary education opportunities, employment opportunities, living opportunities, financial and income needs, friendship and socialization needs, transportation needs, health and medical needs, and legal/advocacy needs.

Historical Background

Transitional planning, for youth with disabilities, was first mandated by federal legislation in 1990 with the passing of PL 101-476, IDEA, 1990. The reauthorization of IDEA in 1997 added new components to the formal process of transitional services. In 1997, the Individuals with Disabilities Education Act was amended to strengthen accountability and academic expectations for children with disabilities across the United States. In particular, transition services were more clearly outlined and defined as a coordinated set of activities for a student, with a disability, that (a) is designed within an outcome oriented process that promotes movement from school to post-school activities, including postsecondary education, vocational training, integrated employment (including supported employment), continuing and adult education, adult services, independent living, or community participation; (b) is based on the student's needs, taking into account the student's preferences and interests; (c) includes instruction, related services, community experiences, the development of employment and other post-school objectives, and, when appropriate, acquisition of daily living skills and functional vocational evaluation (Section 602).

IDEA '97 states:

- (vii)(I) beginning at age 14, and updated annually, a statement of the transition service needs of the

child...that focuses on the child's courses of study (such as participation in advanced-placement courses of a vocational education program)' (ii) beginning at age 16 (or younger, if determined appropriate by the EP Team), a statement of needed transition services for the child, including when appropriate, a statement of the interagency responsibilities or any needed linkages; (III) beginning at least one year before the child reaches the age of majority under State law, a statement that the child has been informed of his or her rights under this title that will transfer to the child...on reaching the age of majority under 615(m) Section 614(d).

This legislature advised that transition services be addressed in the IEP at age 14; therefore, at age 14, the IEP becomes an Individualized Transition Plan or ITP.

Current Knowledge

In preparation for the development of this ITP, transitional assessment must already be completed.

This was a long range plan for what students will do in their remaining years in school as well as what they will do when they exit school. This plan identifies the supports they will receive in school as well as other nonschool supports. In this statement, interagency supports can be identified as well as activities to address transitional needs within home and community environments.

IDEA 2004 changed the age of transitional planning from age 14 to age 16. This is an interesting change considering that in 1990, the U.-S. House of Representatives Committee who reported on the authorization of 1990 stated:

age 16 may be too late for many students, particularly those at risk for dropping out of school and those with the most severe disabilities. Even for those students who stay in school until age 18, many will need more than two years of transitional services. Students with disabilities are dropping out of school before age 16, feeling that the educational system has little to offer them. Initiating services at a younger age will be critical.

(U.S. House of Representatives Report No. 101-554, 10, 1990) States however, may decide to continue mandating transitional services at age 14.

This model is fairly straightforward, including three major components (Storms et al. 2000). First, every student and his or her family should be advised to (1) think about post-high-school goals and (2) develop a plan including supports and services needed to accomplish these goals. (3) The linkages to post-high-school services, supports, and programs need to be identified and made before the student exits high school. The provisions on transition (IDEA 2004) indicate that transitional services should be based on students' *strengths*, needs, interests, and preferences. The addition of "strengths" indicates that transition assessment summary should not just identify areas of need (deficits) but should also provide a description of student's knowledge and skills relative to functioning in post-school environments as well as the planning that has been accomplished.

See Also

- [Individualized Plan for Employment \(IPE\)](#)
- [Transition Planning](#)

References and Reading

- Council for Exceptional Children (CEC). CEC provides a summary of the law, their recommendations, and a link to the text of the law. <http://www.cec.sped.org/am/template.cfm?section=Home>
- Storms, J., O'Leary, E., & Williams, J. (2000). Transition requirements: A guide for states, districts, schools, universities and families. Retrieved 7 Aug 2012 from http://www.cde.state.co.us/cdesped/download/pdf/TK_TransBasics.pdf
- Wehman, P. (2006). *Life beyond the classroom* (4th ed.). Baltimore: Brookes Publishing.
- Wrightslaw. (2004). *IDEA 2004*. Wrightslaw provides information on changes in the law, as well as some brief explanatory comments. <http://www.wrightslaw.com/law/idea/>

Individuals with Disabilities Education Act (IDEA)

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Definition

The Individuals with Disabilities Education Act (IDEA) is an amended version of a landmark federal law passed in 1975 called the Education for All Handicapped Children Act or Public Law 94–142. The IDEA, which has been in place since 1990 with key amendments and revisions occurring as part of reauthorization proceedings in 1997 and 2004, ensures that children and youth with disabilities are afforded basic rights such as a free appropriate public education (FAPE) in the least restrictive environment (LRE), regardless of the nature or severity of the disability. There are four parts to the IDEA that pertain to the following: general provisions under the law (Part A), eligible children ages 3–21 (Part B) and ages 0–2 (Part C), and technical assistance available to state and local education agencies (Part D). In addition to FAPE in the LRE, the IDEA requirements are in place to ensure each state and locality makes provisions regarding identification, due process, individualized education programs (IEPs) or individualized family services plans (IFSPs), nondiscriminatory evaluations, confidentiality, personnel development, and other basic rights established for eligible children and youth with disabilities. The IDEA is known as funding legislation. Part B of the IDEA provides funds to state educational agencies (SEAs) and local educational agencies (LEAs) to help them ensure that children with disabilities, including children aged 3 through 5, have access to a free appropriate public education to meet each child's unique needs and prepare him or her for further education, employment, and independent living. Part C of the IDEA provides funds to each state-led agency

designated by the governor to implement state-wide systems of coordinated, comprehensive, multidisciplinary interagency programs and make early intervention services available to infants and toddlers with disabilities and their families. While all states participate in IDEA mandates and requirements, the law applies only to those agencies receiving federal funds; for example, an independent or private school not accepting federal dollars may be exempt from IDEA requirements and regulations; however, this does not exempt them from civil rights legislation in place for individuals with disabilities such as the Americans with Disabilities Act (ADA) or Section 504 of the Rehabilitation Act.

Historical Background

As one effort to address years of widespread discrimination against individuals with disabilities with respect to access to quality education and educational services, the United States Congress passed Public Law 94–142 in 1975, the Education for All Handicapped Children Act, mandating school services for children with disabilities. Over time, the law has been amended and revised into its current iteration, the Individuals with Disabilities Education Act or IDEA, renamed in 1990, to reflect its mandates focused on the educational needs of children and youth with disabilities ages birth through the 22nd birthday. Since the renaming of PL 94–142, the IDEA has added protections and regulations with each reauthorization such as the development and review at least annually of an individualized education program (IEP) for eligible children ages 3–21 or an individualized family services plan (IFSP) for infants and toddlers. Families have been specifically named and included in the IDEA as full participants in the decision-making process with legal protections written into the law to ensure parent and student rights are not violated. As part of its most recent modifications in 2004, the IDEA now includes regulations for special services including transition services and protections addressing disciplinary issues for eligible children under the Act.

Current Knowledge

The Individuals with Disabilities Education Act (IDEA) is the primary federal program that authorizes state and local aid for special education and related services for eligible children with disabilities. According to IDEA, the term “children with disabilities” means those children evaluated in accordance with Regs. Secs. 300.530-300.534 as having mental retardation, hearing impairments including deafness, speech or language impairments, visual impairments including blindness, serious emotional disturbance, orthopedic impairments, autism, traumatic brain injury, other health impairments, specific learning disabilities, deaf blindness, or multiple disabilities and who because of those impairments need special education and related services. On December 3, 2004, President Bush signed the Individuals with Disabilities Education Improvement Act (P.L. 108–446), a major reauthorization and revision of IDEA. The IDEA 2004 and its federal statutes and regulations currently serve millions of eligible children and youth with disabilities under its provisions. With a commitment to ensuring the provision of a free appropriate public education for those found eligible for a special education and related services, past and current issues for states and citizens concern funding. The most significant funding stream to state education agencies (SEAs), who in turn disseminate funds to local education agencies (LEAs), is that authorized by Part B, with a majority of funds allocated toward Part B initiatives. Currently, Congress has set a maximum target for the federal contribution to special education spending equal to 40% of the estimated excess cost of educating children with disabilities. Thus, if the program were “fully funded,” the states would receive their maximum grants, calculated at 40% of the national average per pupil expenditure times the number of children with disabilities served in the school year 2004–2005, adjusted for population changes. Under the act, the count of children with disabilities cannot exceed 12% of the state’s total school population. Current knowledge regarding the funding formula and resource allocations to each SEA is that there is a shortfall in IDEA funding, with costs not reimbursed or absorbed by the federal

government assumed by the states and local school districts. The American Recovery and Reinvestment Act of 2009 (ARRA) appropriated new funding for programs under Parts B and C of the Individuals with Disabilities Education Act (IDEA). The IDEA 2004, which is currently up for reauthorization as of this writing, is monitored and enforced at the federal level by the Office of Special Education Programs (OSEP).

Future Directions

The Individuals with Disabilities Education (IDEA) Act of 2004 is under review for reauthorization.

See Also

- ▶ [Free Appropriate Public Education](#)
- ▶ [Least Restrictive Environment \(LRE\)](#)
- ▶ [State Educational Agency](#)
- ▶ [Vocational Rehabilitation Act of 1973](#)

References and Reading

- Connecticut State Department of Education. (2005). Guidelines for identification and education of children and youth with autism spectrum disorders. Retrieved December 30, 2010, from http://www.sde.ct.gov/sde/lib/sde/PDF/DEPS/Special/Guidelines_Autism.pdf
- Connecticut State Department of Education. (2006). Steps to protect a child’s right to special education: Procedural safeguards. Retrieved January 15, 2011, from http://www.sde.ct.gov/sde/lib/sde/pdf/DEPS/Special/Prosaf_fullversion.pdf
- Connecticut State Department of Education. (2007). A parent’s guide to special education. Retrieved December 15, 2010, from http://www.sde.ct.gov/sde/lib/sde/PDF/DEPS/Special/Parents_Guide_SE.pdf
- Federal Education Project. (2010). Individuals with disabilities act funding distribution. Retrieved January 30, 2011, from <http://febp.newamerica.net/background-analysis/individuals-disabilities-education-act-funding-distribution>
- <http://idea.ed.gov/explore/view/p/%2Croot%2Cdynamic%2CTopicalArea%2C1%2C>
- IDEA Reauthorized: 2004 Amendments to the Individuals with Disabilities Education Act and implementing regulations. (2006). LexisNexis: Matthew Bender & Company.

- Smith, D., & McGinnley, K. (2004). Side by side comparison of Senate Bill 1248 (as passed on May 13, 2004) and House Bill 1340 (as passed April 30, 2003) and Parts A and B of IDEA, 1997. Retrieved from <http://www.wrightslaw.com/law/idea/sidebyside.06.04.pdf>
- U.S. Department of Education, Office of Special Education Programs 10.04.06 (2006). Building the Legacy: IDEA 2004.
- Volkmar, F. R., Paul, R., Klin, A., & Cohen, D. (2005). *The handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know*. Hoboken: Wiley.
- Wright, P., & Wright, P. (2007). *Wrightslaw: Special education law* (2nd ed.). Hartfield: Harbor House Law Press.

Other Areas

- Americans with Disabilities Act: ADA. <http://www.ada.gov/>
- Section 504 of the Rehabilitation Act of 1973. <http://www2.ed.gov/policy/rights/reg/ocr/edlite-34cfr104.html>
- Section 504/IDEA 2004. www.504idea.org

Infant Toddler Checklist

- [Infant/Toddler Checklist](#)

Infant/Toddler Checklist

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Synonyms

[Communication and symbolic behavior scales – developmental profile](#); [CSBS-DP](#); [Infant toddler checklist](#); [ITC](#)

Description

The Infant Toddler Checklist (ITC) is a one page developmental screener that asks about early

social communication development. It has been normed on over 2000 infants and toddlers and can be used with children between 6 and 24 months of age.

Historical Background

Wetherby and colleagues (Wetherby et al. 2008; Wetherby et al. 2004) developed an early screening tool that can be used from 6 to 24 months of age – the Infant Toddler Checklist (ITC). The ITC is a broader developmental screen that successfully identifies children with developmental, in particular language and communication, delays as well as children with an autism spectrum disorder (ASD). The ITC is one component of the suite of early social communication assessments, known as the Communication and Symbolic Behavior Scales-Developmental Profile (CSBS-DP: Wetherby and Prizant 2002). The ITC can be downloaded for free at: <http://firstwords.fsu.edu/toddlerChecklist.html> and is currently available in English, Spanish, Slovenian, Chinese, and German. The ITC consists of 24 questions about early social communication behavior (e.g., “Does your child let you know when he/she needs help or wants an object out of reach?”; “When you call your child’s name, does he/she respond by looking or turning toward you?”). Unusually for early ASD screening instruments the ITC has norms developed from a reference sample of over 2000 infants and toddlers (Wetherby and Prizant 2002).

Psychometric Data

In an initial study Wetherby et al. (2004) screened 3021 children from a population sample between 6 and 24 months of age. Low scorers (<10th centile) and a random subsample scoring within the typical range were seen during the second year of life for face-to-face behavioral assessments and again around their third birthday. Eighteen children received a diagnosis of ASD and 18 a diagnosis of developmental delay (DD). Sensitivity of the ITC for identifying ASD was 94.4% (17/18)

and for identifying DD was 83.3% (15/18). Thus, from this initial study the instrument parameters of the ITC for identifying toddlers with ASD and DD appeared highly promising.

In their most recent work, Wetherby and colleagues have shown that it is possible to prospectively identify toward the end of the first year of life children who will go on to have a diagnosis of an ASD (Wetherby et al. 2008). From a larger sample of 5385 were screened on multiple occasions (every 3 months) some from as young as 6 months. The authors describe how children who fell below various 10% centile cut-points on the social composite, symbolic composite, and speech composite were invited for face-to-face assessments. In all, nearly 1000 children were assessed ($N = 978$). The positive predictive value (PPV) of the ITC parental checklist for identifying children with delayed development rose from 42.9% on those screened at 6–8 months of age to 79.0% in those screened at 21–24 months of age. In terms of identifying children with an ASD, 56 of the 60 children with an ASD were “flagged” as screen positive on the ITC at first administration, including some from assessments between 9 and 14 months of age. The instrument properties of the ITC, in common with many other ASD screens will require further follow-up of these sample to determine whether there have been other children who go on to develop an ASD who were picked up by the screen.

Clinical Uses

However, this series of studies on the ITC is one of the most developed screening studies undertaken so far. It shows good promise in identifying children with ASD and other developmental/communication difficulties. It also has three notable features. First, it was developed as a surveillance tool. Surveillance involves a parent-professional partnership that takes a broader look at developmental and behavioral skills and progress over time. It combines the observations of parents with the developmental knowledge of the professional and the deployment of specific tests.

Second, it was developed as part of a suite of assessments from the ITC parental checklist to the direct assessment the CSBS-DP. Finally, it was intended to capture not only ASD but also children with other developmental and communication delays. Within general child health settings this holds promise as a broader screener that clinicians can use to identify children who warrant assessment.

See Also

- [Checklist for Autism in Toddlers \(CHAT\)](#)
- [Modified Checklist for Autism in Toddlers \(M-CHAT\)](#)

References and Reading

- Wetherby, A. M., & Prizant, B. (2002). *Communication and symbolic behavior scales developmental profile* (1st Normed ed.). Baltimore: Brookes.
- Wetherby, A. M., Woods, J., Allen, L., Cleary, J., Dickinson, H., & Lord, C. (2004). Early indicators of autism spectrum disorders in the second year of life. *Journal of Autism and Developmental Disorders*, 34, 473–493.
- Wetherby, A. M., Brosnan-Maddox, S., Peace, V., & Newton, L. (2008). Validation of the Infant-Toddler Checklist as a broadband screener for autism spectrum disorders from 9 to 24 months of age. *Autism*, 12, 487–511.

Infantile Autism

- [Autistic Disorder](#)

Infantile Myoclonic Epilepsy

- [Infantile Spasms/West Syndrome](#)

Infantile Spasms Syndrome

- [Infantile Spasms/West Syndrome](#)

Infantile Spasms/West Syndrome

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Synonyms

Blitz-Nick-Salaam Krämpfe; Infantile myoclonic epilepsy; Infantile spasms syndrome; Massive infantile spasms; Myoclonic spasms of infancy; West syndrome

Infantile spasms or West syndrome consists of infantile spasms, hypsarrhythmia, and mental retardation. The spasms are usually resistant to conventional antiepileptic drugs. The typical syndrome has its onset between 3 and 7 months of age and seldom after age of 1 year.

Now, however, it is known that there is a great variability of the features. Any feature of this classical triad may be missing.

Categorization

According to the up-to-date International League against Epilepsy (ILAE 2001) Classification, West syndrome is an epileptic syndrome consisting of clinical spasms usually in clusters with epileptiform EEG, with onset usually less than 2 years of age. West syndrome is an epileptic encephalopathy where cognitive, motor and/or sensory functions are altered by the epilepsy.

Two main groups of the syndrome are recognized: symptomatic and cryptogenic. In *symptomatic* (80%) spasms, there is a history of pre-, peri-, or postnatal damage or disease, and the predisposing etiology can be identified. In *cryptogenic* (15–20%) spasms, there is normal development prior to the onset of the spasms, and no known cause is revealed by clinical and neuroradiological examinations. This group has a probably symptomatic etiology, but underlying causes remain hidden. The rare idiopathic (5%)

refers to a pure functional cerebral dysfunction. Genetic factors play an important role.

The terms idiopathic and cryptogenic have been used as synonyms of each other in some studies. Classification depends heavily on the extent of the investigations performed.

Epidemiology

The incidence of West syndrome has been estimated to vary from 1.6 to 4.3 per 10,000 live births.

Natural History, Prognostic Factors, Outcomes

Infantile spasms are a time-limited disorder, with spasms usually ceasing after a few (3–4) years, even when treatment is not successful. Spontaneous remission rate is 25% within 1 year of onset. However, most patients continue to have significant developmental delay.

The mortality in the follow-up of 25–35 years is 33–35%.

Prognostic Factors

The primary goals of the treatment of infantile spasms are cessation of the spasms and improvement of the mental developmental outcome.

Factors associated with a favorable outcome (Riikonen 2009) include:

1. No other seizures before onset of infantile spasms
2. Absence of atypical spasms, partial seizures, and asymmetric EEG abnormalities
3. Age of onset between 4 and 9 months

The above-mentioned factors are also associated with cryptogenic etiology.

4. Early and sustained response to treatment
5. Short duration of hypsarrhythmia

In the recent prospective UK study (Lux et al. 2005), the developmental outcome of children treated with hormonal (n = 55) or vigabatrin (VGB) (n = 52) therapy was compared at age 14 months. In infants with no identified

underlying etiology ($n = 46$), the mean Vineland Adaptive Behavioral Scale scores were higher in those allocated to hormone treatment than those allocated to VGB treatment. This difference was even greater at the age of 4 years (median difference 14 scores). Hormone treatment therefore seems to be the better first treatment (Darke et al. 2010).

Outcomes

Short Term

If the etiology is cryptogenic the spasms will cease in 80%. If the etiology seems to be symptomatic the spasms will cease in 50%. However, at 3–4 months after starting treatment only 40–44% are seizure-free.

Long Term

We know the long-term outcome after corticotrophin (ACTH) therapy but not after VGB therapy. In Finland, 214 children were followed up for 25–35 years or until death (Riikonen 2001). This study assessed the death rate, autopsy findings, intellectual outcome using standardized psychometric tests, education, occupations, psychiatric disorders, and occurrence of epilepsy and follow-up EEG. The patients had been participants in a prospective study conducted in 1960s, and follow-up was obtained in 100% of the cases. The patients had been on the following treatments: large doses of ACTH (120 IU = 0.12 mg/day) ($N = 54$) or lower doses (20–40 IU = 0.02–0.04 mg/day) ($N = 97$). Large doses given for a relatively short period of 6 weeks were not more effective than the smaller doses. The long-term intellectual outcome was better in children treated with lower doses than with larger doses.

One third died, of which a third died before the age of 3 years. Normal or mildly abnormal intelligence was present in a quarter. They went to normal school or school for educationally impaired children, a quarter to training school, and the rest were uneducable. Twenty-five percent had employment ranging from professional to manual labor. Ten were married. Five had children, altogether ten healthy children. A quarter was

institutionalized. A third was seizure-free, a fifth had daily seizures, and the rest had less frequent seizures. EEG was normal or slightly abnormal in 22% of the cases at the last examination. Focal abnormalities were seen in 37%. Focal abnormalities were equally frequent in patients with normal and patients with less favorable outcome. In several studies, investigators have failed to find any correlation between various EEG features and long-term outcome.

Kumagai et al. (2001) evaluated the long-term prognosis of 120 patients in Japan at the mean age of 25 years.

This cohort suggested that 60% had a reasonable quality of life (such as walking, communication, eating, dressing, and toileting).

Autism and Infantile Spasms

Epidemiology

Psychiatric disorders following infantile spasm have received relatively little attention in the literature. In Finland, a systematic search for autism was made for 192 children with infantile spasms and 24 patients had infantile autism (Riikonen and Amnell 1981). In a population-based study in a cohort with seizures in the first year of life (95 children), infantile spasms predicted a high risk for autism spectrum disorders (Saemundsen et al. 2008).

Etiology and Pathogenesis

Epilepsy and/or epileptic EEG abnormalities are frequently associated with autistic disorders in children, but this does not necessarily imply that they are the cause. Nevertheless, there are different etiologies where a direct relationship between early epilepsies and autism may be suspected. The direct role of epilepsy can only be evaluated in longitudinal studies. It seems probable that the later psychiatric manifestation is connected with the same brain lesion which had earlier caused the infantile spasms. In the majority of cases, specific underlying causes cannot be identified. However, a number of factors are being investigated including infectious, metabolic, genetic, and environmental causes (Table 1). Large numbers of tests in the absence of a specific clinical indication are

Infantile Spasms/West Syndrome, Table 1 Etiological investigations of infantile spasms

Comprehensive history:	Heritable disorders, pre- and perinatal events
Clinical examination:	Signs of tuberous sclerosis
Funduscopy:	Phacomias, intrauterine infections, metabolic diseases
MRI:	Malformations, tubers, atrophy, myelination
CSF:	Cells, proteins, glucose, glycine, lactic acid, aminoacids, folate metabolites, neurotransmitters
Urine:	Organic acids
Serum:	Amino acids
Chromosomal abnormalities:	If structural brain abnormalities, dysmorphic features
Pyridoxin (phosphate) 100 mg i.v. to screen pyridoxine-dependent seizures	

wasteful and unlikely to generate useful data for clinical care. The frequency of autism in tuberous sclerosis is estimated as high as 8–14% among the subgroup of autistic individuals with a seizure disorder. Autistic behavior can often be seen (in 58%) in tuberous sclerosis and infantile spasms (Hunt and Dennis 1987).

The presence of autism may arise if the tuberous sclerosis mutation occurs at critical stages of neural development of brain regions critical in the development of autism. Early epilepsy and functional deficits associated with the anatomical lesions in the temporal lobes may be associated with autism. There is emerging evidence that early onset electrophysiological disturbances within temporal lobes (and perhaps other locations) have a deleterious effect on the development and establishment of key social cognitive representations concerned with processing social information. Temporal abnormalities were found in 70% of the patients with autism (Riikonen and Amnell 1981). This corresponds with the positron emission tomography (PET) studies where 70% of the patients with infantile spasms and autism have bitemporal hypometabolism (Fig. 3). Location of tubers in the temporal lobes, however, does not seem not be a sufficient risk factor for development of autism. Very recently, it has been shown that seizure

activity in the left temporal lobe is a risk factor for autism spectrum disorders in tuberous sclerosis (Numis et al. 2011).

Differential Diagnosis

Severe sensory deficits may make false interpretation of the behavior of the child. Majority of children with autistic behavior are mentally retarded. Retarded children have a tendency to develop autistic behavior because of their cognitive and perceptual deficits and because of environmental deprivation during long-term stays in hospitals and institutions. It must be emphasized, however, that autism and mental retardation are far from synonymous.

Treatment

Behavioral treatments remain the cornerstone of management. Early diagnosis of autism will allow for earlier treatment and better outcome.

Clinical Expression and Pathophysiology

Clinical Expression

Spasms

The spasms may be flexor, extensor, or, most commonly, mixed spasms. One child can have various types of the spasms. Spasms occur in clusters. The presence of subtle spasms, asymmetric or asynchronous spasms, and focal signs suggests a symptomatic etiology and are associated with unfavorable outcome in the long term. A number of investigators have documented the coexistence of partial seizures occurring in patients with infantile spasms. They are common in drowsiness and especially on arousal or soon thereafter. A vast number of spasms are missed by parents when compared with those seen in video monitoring.

The diagnosis of the spasms is easy when they are typical. However, the spasms are often misinterpreted and considered to be colic, startle responses, or normal infant behavior. Repetitive, stereotyped characterization of any movements in infancy should arouse the suspicion of infantile spasms and lead to an immediate EEG examination.

Electroencephalogram (EEG)

Hypsarrhythmia consists of a pattern of irregular, diffuse, asymmetric, high-voltage slow waves interspersed with sharp waves and spikes, distributed randomly throughout scalp recordings (total chaotic disorganization of cortical electrogenesis) (Fig. 1). The term “modified hypsarrhythmia” (occurring in about 40%) is used if there is some preservation of background rhythm, synchronous bursts of generalized spike-wave activity, significant asymmetry, or burst suppression of tracing. A constant focus of abnormal discharges may also precede, accompany, or follow hypsarrhythmia. Multifocal spikes may also evolve into hypsarrhythmia if there is a long delay before treatment of spasms. A transition from typical hypsarrhythmia to a pattern of multifocal spike activity during treatment with vigabatrin (VGB) is observed in a number of patients. Persistence of multifocality means that there are always spasms which may be subtle.

The most common ictal pattern associated with symmetric spasms is a generalized slow wave, usually followed by attenuation of background

activity (Fig. 2). The initial diagnosis of infantile spasms may require video-EEG studies in infants with symptomatic etiology who may show only subtle spasms. Constantly normal tracing, including sleep recording, rules out the diagnosis of infantile spasms. EEG should always include waking and sleeping states.

Autistic-like Behavior of an Infant Sometimes the infant ceases to take interest in his surroundings, and blindness is suspected. This functional amaurosis is seen in association with missing or grossly abnormal patterns of visually evoked potentials and probably corresponds to perfusion defects involving the parieto-occipital areas. In addition, behavioral regression (autistic-like behavior) of an infant less than 1 year of age should lead to an EEG examination, in which hypsarrhythmia may reveal the cause of regression.

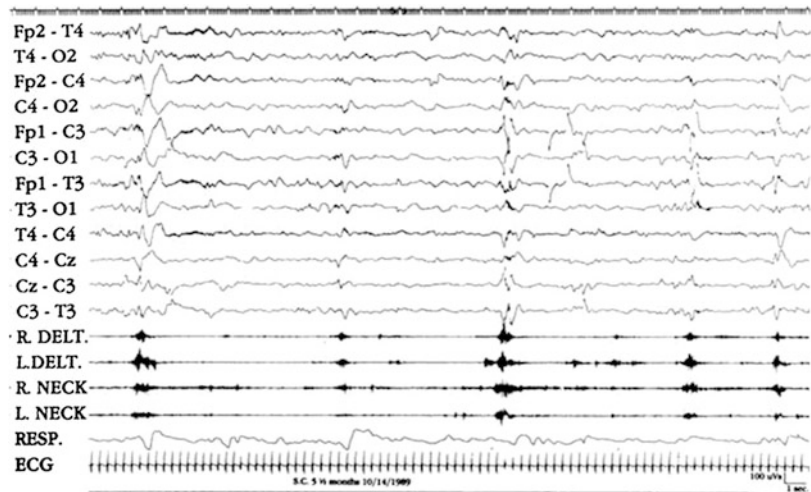
Pathophysiology of Infantile Spasms
Problems with modulation of neurotransmitters at a specific period of brain maturation are believed



Infantile Spasms/West Syndrome, Fig. 1 Typical hypsarrhythmia

Infantile Spasms/West Syndrome,

Fig. 2 Hypsarrhythmia disappears between the spasms



to be fundamental to the development of infantile spasms. The latter usually appears when at the age which corresponds to the critical period of maximal brain development and age and is supported as follows:

Infantile spasms and hypsarrhythmia disappears spontaneously with age of high corticotrophin-releasing factor (CRF) abundance. Glucocorticoids have an accelerating effect on some physiological events.

The molecular mechanisms which lead to the long-term consequences of infantile spasms are poorly understood. It has been suggested that early stress (injury or insult) during a critical pre- and perinatal period activates proconvulsant stress mechanisms, increasing CRF synthesis and activity, with resultant long-term effects (Brunson et al. 2001). Insults or stress in early life may affect the synthesis of insulin-like growth factor-1, which might play a role in disturbance of synaptogenesis and reduction of certain cognitive functions (Riikonen 2010). The existence of a latent period also raises the possibility of preventive intervention.

Evaluation and Differential Diagnosis

Evaluation

The infantile spasms clinical evaluation begins with the history and physical examination of the

patient. Evaluating the EEG is critical for the diagnosis of infantile spasms and for patient management. It is recommended that an EEG evaluation be conducted as soon as possible following the identification of spasms.

Careful search for etiological factors should be done in every case to formulate a more accurate prognosis and establishing the need for genetic counseling. Within symptomatic group, the etiologies for infantile spasms have traditionally been divided into pre-, peri-, and postnatal causes. Most studies identify prenatal etiologies as the most common, accounting almost 50% of symptomatic cases.

More than 200 potential etiological factors for infantile spasms have been reported. However, they are not necessarily causally related. History and physical examination alone may identify the majority of symptomatic spasms. MRI is the most important diagnostic tool. Seventy-percent will have established etiological diagnosis without need to conduct extensive testing (Table 1). MRI may need to be repeated after 18 months to 2 years after myelination of the brain is more complete as early scans may miss gray matter abnormalities. Common abnormalities revealed by MRI are focal cortical dysplasias, thinning of the corpus callosum and delayed myelination. Inborn errors of metabolism are a rare but significant cause of infantile spasms. Often there have been preceding seizure types. Rarely, West

Infantile Spasms/West Syndrome, Table 2 Genetic causes of infantile spasms

Tuberous sclerosis	Aicardi syndrome
	Miller Dieker syndrome
	Progressive encephalopathy-hypsarrhythmia-optic atrophy (PEHO) syndrome
	Down syndrome
X-linked:	ARX aristaless-related homeobox gene
	CDKL5/STK9 kinase gene
	MECP2
	STXBP1

syndrome occurs with single-gene metabolic disorders. While each of the genetic mutations is rare, collectively they comprise an increasing proportion of infantile spasms etiology (Table 2). Tuberous sclerosis is the most important genetically defined disease.

Differential Diagnosis

Syndromes closely related to West’s syndrome include infantile epileptic encephalopathy and early myoclonic encephalopathy. Both are very rare syndromes. They are characterized by onset before 3 months of age and a severe course, with lack of psychomotor development. Response to ACTH is poor. Both syndromes have a burst-suppression pattern in EEG. West’s syndrome may be replaced in a quarter of patients by another severe epilepsy syndrome, Lennox-Gastaut syndrome, which is an important differential diagnosis for patients more than 1 year of age. There is a transition between the four syndromes.

Treatment

The delayed psychomotor development that characterizes epileptic encephalopathy justifies vigorous treatment.

Many studies have shown that the cognitive outcome is better when the lead time to treatment is short (Riikonen 2009).

Infantile Spasms/West Syndrome, Table 3 Alternative therapies in the treatment of infantile spasms: pooled data from different studies

Therapy	Number of patients	Response rate (%)
ACTH	476	59
Prednisone	26	31
High-dose prednisone	30	70
Vigabatrin	1,026	37
High-dose valproate	41	54
Nitrazepam	27	52
Pyridoxine	118	13
Zonisamide	119	27
Lamotrigine	30	29
Topiramate	35	29
Sultiame	17	35
Ketogenic diet	104	37

Short-Term Effects

Infantile spasms are usually resistant to conventional antiepileptic drugs. Most studies are dealing only with short-term effects of the drugs. The outcome measures should be the following: cessation of the spasms, resolution of hypsarrhythmia, relapse rate, absence of epilepsy and epileptiform EEG, cognition and behavior. More than 150 trials have carried out in the treatment of infantile spasms (Table 3). Steroids and vigabatrin are now the first-line drugs for infantile spasms. In a large data-based critical review of American Academy of Neurology and Child Neurology Society, nine ACTH prospective studies were analyzed (Mackay et al. 2004). It was concluded that there is insufficient data to recommend the optimal dosage and duration of ACTH. “ACTH is probably effective and vigabatrin possibly effective for short-term treatment of infantile spasms and possibly effective for children with tuberous sclerosis.”

ACTH or Oral Steroids?

In a small prospective, randomized study, the efficacy of high-dose ACTH (150 IU/m² for 2 weeks) was better than that of prednisone (2 mg/kg for 2 weeks) (Baram et al. 1996) but a

small prospective double-blind study found otherwise.

In a new prospective study from the United Kingdom (UK) (Lux et al. 2004), 107 patients were randomized to tetracosactide (synthetic ACTH) 0.5 mg/day (40 IU) on alternate days and prednisolone 40 mg/day. The response rates were 76% and 70%, respectively. The difference was not significant on day 14 of treatment. Tuberous sclerosis (TS) was excluded in this study because VGB was considered to be the drug of choice in TS (Hancock et al. 2008).

Although ACTH will downregulate CRF via melanotropic receptors and decrease excitability in the limbic system (Brunson et al. 2001), oral steroids themselves, which do not have down-regulating effects of CRF, have clear clinical influences. ACTH and oral steroids might accelerate physiological events during critical stages of brain development through action as a trophic hormone. The reasons for the efficacies of ACTH and steroids require further elucidation.

Dose of ACTH?

The doses vary in different countries: Japan 0.1 mg/day, Finland 0.25 mg/day, and USA 0.8 mg/day (80 IU).

It is not clear why infants in the USA would need considerably larger doses than in Japan.

Corticotrophin, so-called natural ACTH, acts for 12–18 h. Its synthetic derivate, tetracosactide, lasts for 24–48 h. The international units of ACTH and tetracosactide are believed to be equivalent: corticotrophin 80–100 IU is equivalent to tetracosactide 1 mg. Tetracosactide is used on alternate days because of its prolonged action. The relatively serious side effects of tetracosactide are probably due to its prolonged action. Natural ACTH (“Achter”) is not available in Europe, and synthetic ACTH (“Synachten”), not in USA. The first drug is much more expensive than the latter.

In a small blind prospective study by Hrachovy et al. 1994, high doses (150 IU/m²) given for 3 weeks were not more effective than lower doses of ACTH (20–39 IU/day) given for 2–6 weeks or hydrocortisone given for shorter periods. In a Finnish studies, there were no difference in response rate or relapse rate comparing low dose

(20–40 IU/day) and high dose (100 IU/day). The long-term cognitive outcome was better with low dose regimen.

Recently, in a large US review (Pellock et al. 2010) it was concluded that ACTH is the preferred therapy, but the optimal dose is still unknown.

Steroids or Vigabatrin?

Vigabatrin has gained popularity because of its ease of use and its efficacy. In Europe, opinions are divided with regard to vigabatrin as initial treatment. In USA, vigabatrin was approved by FDA in 2009, and both drugs are now the first-line therapies (Pellock et al. 2010). Compared to vigabatrin, more patients will respond with ACTH (Vigevano and Cilio 1997; Lux et al. 2004), with tuberous sclerosis excluded in the latter study. Depending on the study, after 2 weeks of therapy, patients treated with vigabatrin were seizure-free in 26% (Granström et al. 1999), 23% (Elterman et al. 2001), and 54% (Lux et al. 2004) of cases. However, at 3 months, the number of seizure-free patients increased to 60–65% in the two former studies. These data suggest that the response with vigabatrin comes later than with ACTH. After 14 months of treatment, there was no difference in seizure frequency between patients treated with vigabatrin and those treated with oral hormones (Lux et al. 2005). The dose of vigabatrin has ranged from 40 to 120, usually 40–60 mg/kg/day, given in two daily doses.

After pooling the data of 8 separate prospective and retrospective studies, vigabatrin was effective in 44% of 478 patients. In the pooled data of 9 separate prospective and retrospective studies, ACTH was effective in 59% of 476 patients. In the recent UK study (which excluded TS patients), the cessation of the spasms was more likely with hormonal treatment. VGB was effective for spasms in 54% and hormonal treatment 70% on day 14.

Tuberous Sclerosis

VGB is used in tuberous sclerosis (Hancock et al. 2008). The response rate to VGB and ACTH are similar, more than 70%. However, relapse rates are high after both drug trials. Because the use of

ACTH is always given for a specified period and VGB for an indefinite period, VGB is a better choice.

Relapses

The hormonal treatment may be stopped after only 1 month and vigabatrin after 3 months (Willimore et al. 2009; Pellock et al. 2010).

The relapse rate after ACTH treatment is estimated to be 15–24%. There is not enough information of relapse rate after VGB therapy.

The numbers of patients who were seizure-free at 3–4 months after ACTH or VGB treatment are very similar (42–44%). In the recent UK study, the number of patients who were spasm-free at 14 months was very similar.

A new course of ACTH is effective in two thirds of the cases. In refractory cases valproate, nitrazepam, topiramate, or vigabatrin are recommended. Ketogenic diet or surgery should also be considered.

Side Effects

Both drugs have severe side effects. The side effects of ACTH are infections, arterial hypertension, and adrenal hypofunction after therapy. For infants with a history of frequent respiratory infections, prophylactic trimetoprim-sulfaxazole therapy might be recommended.

Hypertension should be monitored and treated. In hypertensive patients, the heart should be studied by ultrasound to recognize hypertrophic cardiomyopathy. Hydrocortisone substitution should be given after ACTH therapy and continued until the adrenal axis has normalized.

In Finland, the therapy is given at minimal effective dose and for the minimal effective time (Table 4). With such treatment, no serious side effects were found. The side effects of ACTH might be severe, but they are all treatable and reversible.

A serious concern in regard of concentric visual field defects (VFDs) has arisen in relation to the use of VGB. The visual field defects it causes seem to be permanent. One recent systematic review (Maguire et al. 2010) showed that VFDs occur in 40% in adults and in 34% in children. The earliest sustained onset of the

VGB-induced retinal defect is at 3 months (Willimore et al. 2009). VFD defects are sufficient to prevent a person from driving. There are also concerns with children who might have a poor underlying function as a part of their overall neurological disabilities.

In 2009, the U.S. Food and Drug Administration approved vigabatrin “if the patients have periodic ophtalmological evaluations at initiation of therapy, at every 3 months while taking VGB as well as 3–6 months after cessation of treatment”. However, accurate assessment of visual field changes in infants is challenging and no consensus exists on a protocol for visual evaluation for infants. Transient changes in basal ganglia, thalamus, brainstem, and dentate nucleus have been recently shown.

The other regimens used in the treatment are seen in Tables 5 and 6.

Infantile Spasms/West Syndrome, Table 4 Regimen 1 for use of ACTH or vigabatrin for infantile spasms in Finland (Finnish Guidelines for Childhood epilepsy 2007; Riikonen 2009)

After a short trial with pyridoxine, ACTH or vigabatrin are the first-line drugs
Start with tetracosactide 0.03 mg/kg or 0.5 mg on alternate days for 2 weeks and then tapering the dose during the next 2 weeks, or alternatively doubling the dose to 0.06 mg/kg for 2 weeks and then tapering
Start with VGB 40 mg/kg/day and continue up to 10 days. In responsive cases, use up to 3 months
In nonresponsive cases, switch to ACTH after 10 days
Use VGB as the first drug for tuberous sclerosis
Use ACTH as the first drug for cryptogenic cases

Infantile Spasms/West Syndrome, Table 5 Regimen 3 U.S. consensus report (14 centers) (Pellock et al. 2010)

Short-term treatment with a first-line treatment with maximum effective dose for 2 weeks
EEG evaluation of effectiveness (“all-or-none” response, presence or absence of infantile spasms, hypsarrhythmia, or epileptiform electroencephalography discharges)
Prompt treatment modification
No consensus of the first-line drug
No consensus on initial treatment dosage

Ketogenic Diet

The ketogenic diet may be an option in drug-resistant epilepsy as an adjunct to pharmacological therapy or as alternative when first-line treatments (ACTH and VGB) fail (Hong et al. 2010).

Surgery

Surgery may be the treatment of choice in a small number of patients, refractory to medical treatment (but no evidence of metabolic or degenerative disorders), and in which localized lesions are present (Fig. 3). The authors recommend that surgical intervention should be considered early in the medically refractory cases prior to the onset of epileptic encephalopathy. When a single lesion is present on the MRI and there is a good correlation with EEG localization, surgical treatment is quite favorable in terms of both seizure control and cognitive development. Positron emission tomography (PET) complements MRI by defining

the full extent of lesion and evaluating the integrity of the full lesion. PET abnormalities may also guide placement of intracranial electrodes. Development of newer more specific PET probes for epilepsy has led to improved and more accurate localization of seizure foci. Recent advances in pediatric epilepsy surgery include resection of multiple tubers in children with tuberous sclerosis. Thus, this development should ultimately improve the outcome of epilepsy surgery in West syndrome.

Conclusion

In West syndrome, many advances have been achieved:

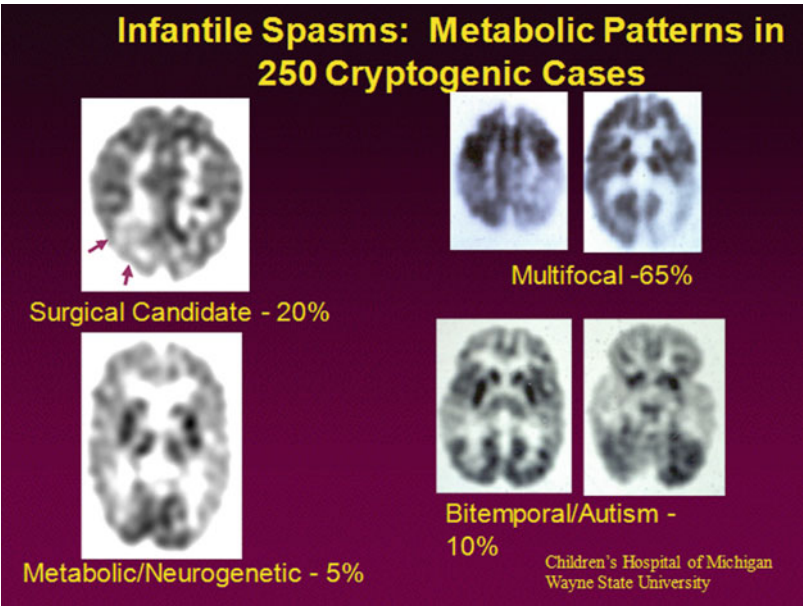
- Identification is more precise, using video and polygraphic recordings.
- More accurate etiological diagnoses can be made by clinical, neuroimaging, genetic, and pathologic methods.
- Hormonal therapy is the most effective therapy in the short term and might bring better cognitive outcome in patients with cryptogenic spasms.

Infantile Spasms/West Syndrome, Table 6 Regimen 4 Japan (Fukuyama 2010)

Vitamin B 20–40 mg/kg/day for 2 (?)weeks
SyntheticACTH 0.0125 mg/kg/day for 2 weeks and tapering

Infantile Spasms/West Syndrome,

Fig. 3 Infantile spasms: metabolic patterns in 250 cryptogenic cases. Note the bitemporal hypometabolism in a case with autism (Children’s Hospital of Michigan, Wayne State University, with courtesy of Professor Harry Chugani, presented in Infantile Spasms Progress in Epileptic Spasms and West syndrome Congress in Rome 1997)



- Cognitive development is not possible if hypsarrhythmia persist. Short lead time to treatment is essential. Effective treatment for infantile spasms should be “all-or-none” response.
- Side effects of the main drugs, ACTH and VGB, are better known.
- In some patients, surgical approach seems of benefit in achieving seizure control.

On the other hand,

- There is lack of consensus on the drug of first choice
- Frequency of visual field defects in children treated with VGB should be studied in addition to the long-term outcome in general

Advances in our understanding of brain maturation, etiologies mechanisms, and genetics underlying West syndrome may facilitate more effective pharmacological interventions.

References and Reading

- A working group appointed by the Finnish Medical Society Duodecim and the Finnish Association of Pediatric Neurology. (2007). *Childhood epilepsy and febrile seizures. Current care guideline*. Helsinki (In Finnish).
- Baram, T., Michell, W., Tournay, A., Snead, O., Hanson, R., & Horton, E. (1996). High-dose corticotrophin (ACTH) versus prednisone for infantile spasms: A prospective, randomized, blinded study. *Pediatrics*, 97, 375–379.
- Brunson, K., Khan, N., Eghbal-Ahmadi, M., & Baram, T. (2001). Corticotrophin (ACTH) acts directly on amygdale neurons to down-regulate corticotrophin-releasing hormone gene expression. *Annals of Neurology*, 49, 304–312.
- Chugani, H., Da Silva, E., & Chugani, D. (1996). Infantile spasms III. Prognostic implications of bitemporal hypometabolism on positron emission tomography. *Annals of Neurology*, 39, 643–649.
- Darke, K., Edwards, S., Hancock, E., Johnson, A., Kennedy, C., Lux, A., et al. (2010). Developmental and epilepsy outcomes at ages 4 years in the UKISS trial comparing hormonal treatments to vigabatrin for infantile spasms: A multi-centre randomized trial. *Archives of Disease in Childhood*, 95, 382–386.
- Elterman, R., Shields, W., Mansfield, K., & Nagawa, J. (2001). Randomized trial of vigabatrin inpatients with infantile spasms. *Neurology*, 57, 1416–1421.
- Fukuyama, Y. (2010). The Japanese scheme of ACTH therapy in West syndrome. Commentary. *Epilepsia*, 51, 2216–2217.
- Granström, M.-L., Gaily, E., & Liukkonen, E. (1999). Treatment of infantile spasms: Results of population-based study with vigabatrin as the first drug for spasms. *Epilepsia*, 40, 950–957.
- Hancock, E., Osborne, J., & Edwards, S. (2008). Treatment of infantile spasms. *Cochrane Database of Systematic Reviews* (4), CD001770.
- Hong, A., Turner, Z., Hamdy, R., & Kossoff, E. (2010). Infantile spasms treated with the ketogenic diet: Prospective single-center experience in 104 consecutive patients. *Epilepsia*, 51, 1403–1407.
- Hrachovy, R., Frost, J., & Kellaway, P. (1983). Zion T double-blind study of ACTH versus prednisone for infantile spasms. *Journal of Pediatrics*, 103, 641–645.
- Hrachovy, R., Frost, J., & Glaze, D. (1994). High-dose, long-duration versus low-dose, short duration corticotrophin therapy for infantile spasms. *Journal of Pediatrics*, 124, 803–806.
- Hunt, A., & Dennis, J. (1987). Psychiatric disorders among children with tuberous sclerosis. *Developmental Medicine and Child Neurology*, 29, 190–198.
- ILEA Commission Report. (2001). A proposed diagnostic scheme for people with epileptic seizures and with epilepsy: Report of the ILEA task force on classification and terminology. *Epilepsia*, 42, 796–803.
- Kumagai, T., Ito, M., Yamazaki, Y., Sekijima, K., Sakakibara, K., Matsumoto, Y., et al. (2001). Long-term prognosis of patients with West syndrome in Japan: Social aspects. *Brain & Development*, 23, 695–697.
- Lux, A., Edwards, S., Hancock, E., Johnson, A., Kennedy, C., Newton, R., et al. (2004). The United Kingdom infantile spasm study comparing vigabatrin with prednisolone or tetracosactide at 14 days: A multicentre, randomized controlled trial. *Lancet*, 364, 1773–1778.
- Lux, A., Edwards, S., Hancock, E., Johnson, A., Kennedy, C., Newton, R., et al. (2005). The United Kingdom Infantile Spasms Study (UKISS) comparing hormone treatment with vigabatrin on developmental and epilepsy outcomes to age 14 months: A multicentre randomized trial. *Lancet Neurology*, 4, 712–717.
- Mackay, M., Weiss, S., Webber, T., Ashwal, S., Stephens, D., Ballaban-Gill, K., et al. (2004). Practice parameter: Medical treatment of infantile spasms: Report of the American Academy of Neurology and Child Neurology Society. *Neurology*, 62, 1668–1691.
- Maguire, M., Hemming, K., Wild, J., Hutton, J., & Marson, A. (2010). Prevalence of visual field loss following exposure to vigabatrin: A systematic review. *Epilepsia*, 51, 2423–2431.
- Numis, A., Major, P., Montenegro, M., Muzykewicz, D., Pulsfer, M., & Thiele, E. (2011). Identification of risk factors for autism spectrum disorders in tuberous sclerosis complex. *Neurology*, 76, 981–981.

- Pellock, J., Hrachovy, R., Shinnar, S., Baram, T., Bettis, D., Dlugos, D., et al. (2010). Infantile spasms: A U.-S. consensus report. *Epilepsia*, *51*, 2175–2189.
- Riikonen, R. (2001). Long-term outcome of patients with West syndrome. *Brain & Development*, *23*, 683–687.
- Riikonen, R. (2009). West syndrome. In M. Nikanorova, P. Genton, & A. Sabers (Eds.), *Long-term evolution of epileptic encephalopathies* (pp. 13–28). Esher, Surrey: J. Libbey.
- Riikonen, R. (2010). Favourable prognostic factors with infantile spasms. *European Journal of Paediatric Neurology*, *14*, 13–18.
- Riikonen, R., & Amnell, G. (1981). Psychiatric disorders in children with earlier infantile spasms. *Developmental Medicine and Child Neurology*, *23*, 747–760.
- Riikonen, R., Jääskeläinen, J., & Turpeinen, U. (2010). Insulin-like growth factor-1 is associated with cognitive outcome in infantile spasms. *Epilepsia*, *51*, 1283–1289.
- Saemundsen, E., Ludvigsson, P., & Rafnsson, V. (2008). Risk of autism spectrum disorders after infantile spasms: A population-based study nested in a cohort with seizures in the first year of life. *Epilepsia*, *49*, 1865–1870.
- Vigevano, P., & Cilio, R. (1997). Vigabatrin versus ACTH as first-line treatment for infantile spasms: A randomized, prospective study. *Epilepsia*, *38*, 1270–1274.
- Willimore, L., Abelson, M., Ben-Menachem, E., Pellock, J., & Shields, W. (2009). Vigabatrin: 2008 update. *Epilepsia*, *50*, 163–173.

Infantile Subacute Necrotizing Encephalopathy

► Leigh Disease

Infants with Autism

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Definition

Autism spectrum disorder (ASD) is a complex neurodevelopmental disorder with initial onset of symptoms early in life. When early infantile

autism was first described by Leo Kanner (1943), the symptoms were assumed to be present from birth. Though the exact etiology of ASD is still unknown, and some children may show a regression or plateau in development, it is still believed that most children with ASD are born with the disorder. However, much remains to be learned about the characteristics of autism in infancy.

ASD is a highly heritable complex genetic disorder which may actually have multiple etiologies and likely involves susceptibility genes as well as epigenetic factors which are not yet well understood (Abrahams and Geschwind 2008). In the absence of any biomarker, ASD is defined by its behavioral profile. Though historically most children with ASD have been diagnosed and treated in the preschool and school-age years, research evidence indicates that the symptoms of ASD emerge in the first and second year of life. Infants with ASD may display emerging signs of the social communication deficits inherent in autism including limited eye contact, reciprocal social smiling, response to name, babbling, communicative gestures, and functional and symbolic play. Stereotyped motor movements and preoccupation with parts of objects may present in the first or second year of life. In the first year, late onset of social smiling and poor responsivity to speech may trigger initial parent concerns. Research indicates that early diagnosis is relatively stable, and early initiation of treatment improves prognosis.

Much of what is currently known about the early development of children with ASD has been learned from prospective studies of infant siblings of children with ASD. These siblings are at increased genetic risk of developing ASD, as well as other developmental delays. As these babies are followed prospectively by experts, their behavior can be analyzed for the earliest signs of an atypical developmental trajectory.

Historical Background

From the first reports by Leo Kanner (1943), autism was presumed to be a congenital disorder with origins in infancy, and research to date has generally supported this notion. The social

communication behaviors which are disrupted in ASD have their origins in the first year of life for typically developing infants, suggesting that the pathogenic processes which give rise to ASD are likely present from a very early age. For example, within the first weeks of life, typically developing infants prefer social stimuli such as faces and speech sounds. By 4 to 6 months of age, infants engage in contingent dyadic interactions with caregivers, and toward the end of the first year and beginning of the second year, infants show emerging capacity for triadic social interaction in the form of joint attention.

However, until recently, ASD was not commonly diagnosed before the preschool years. Therefore, historically, the majority of information about the onset and early progression of autism symptoms came from retrospective parent reports. This early research indicated that up to half of parents of children with ASD recall concerns during the first year of life (De Giacomo and Fombonne 1998; Volkmar et al. 1985), and most parents (80–90%) recall concerns prior to the second birthday (Baghdadli et al. 2003; Chawarska et al. 2007).

The growing use of home video equipment by families to document child development and special occasions at home allowed researchers to retrospectively collect and analyze video recordings of children who later were diagnosed with ASD and controls to assess for the presence of autistic traits in infancy. Indeed, analysis of home videos of children later diagnosed with ASD indicated that eye contact, responsiveness to name, socially directed positive affect, and reciprocal social smiling were less frequent in these children than controls at 12 months of age (Adrien et al. 1993; Baranek 1999; Osterling et al. 2002; Werner et al. 2005).

Though these studies were critical in confirming the early emergence of autism symptoms, given the methodological limitations of retrospective methods and the limited degree to which etiologic factors could be examined, researchers began searching for ways to gather prospective information regarding the characteristics of infants with ASD in order to better inform diagnosis and treatment efforts. However, even

with the prevalence of ASD estimated at 1 in 68 children by the Centers for Disease Control and Prevention (Christensen et al. 2016), without identification of a population of infants at increased risk of developing ASD, the cost of obtaining detailed prospective data on a sufficient number of infants who will later develop ASD has been prohibitive. Given that the genetic risk associated with ASD is considered to be high, siblings of children with ASD are at significantly greater risk of developing the condition than children in the general population, with recurrence rates of ASD in siblings estimated as high as 20% (Bolton et al. 1994; Ozonoff et al. 2011). Therefore, if a large group of infant siblings could be followed prospectively, a proportion of them would develop ASD, thus providing a feasible way to learn about the early developmental characteristics which differentiate infants who do and do not develop ASD (Zwaigenbaum et al. 2007). Based on this logic, multiple large-scale prospective studies of infant siblings of children with ASD were initiated with the objective of identifying earlier neurological and behavioral markers and illuminating the etiology of ASD by following children from the first months of life.

Autism Assessment in Infants

Research indicates that diagnosis of ASD can be reliably made by experts at age two (Chawarska et al. 2008) and is relatively stable in early childhood (Chawarska et al. 2009). At present, assessment using the Autism Diagnostic Observation Schedule, Second Edition (ADOS-2; Lord et al. 2012) and the Autism Diagnostic Interview-Revised (ADI-R; Lord et al. 1994) is considered the “gold standard” for diagnosis of ASD within clinical research. The second edition of the ADOS includes a toddler module (ADOS-T) for children 12–30 months of age with minimal speech (Luyster et al. 2009). However, the ADI-R was not developed for use with infants and toddlers; therefore, professionals using the current version must use caution when interpreting scores for children under 3 years of age. Several other measures have been developed for use in screening for

the presence of autism symptoms in toddlers, such as the Modified Checklist for Autism in Toddlers (M-CHAT; Robins et al. 2001) and the Screening Test for Autism in Two-Year-Olds (STAT; Stone et al. 2000). In addition, the Autism Observation Scale for Infants (AOSI; Bryson et al. 2008) was developed for infants under 18 months of age and is used in many studies of very young children at risk for ASD. Researchers have also utilized other measures such as the Alberta Infant Motor Scale (AIMS) to detect early deviations in development in 3- and 6-month-old high-risk infants (Bhat et al. 2012).

Recent studies suggest that diagnosis is occurring closer to 18 months to 2 years of age due to the increasingly prevalent use of prospective screenings and measures designed for infants (Elsabbagh and Johnson 2010; Guinchat et al. 2012; Shen et al. 2013). Early diagnosis of autism is crucial to ensure access to early intervention services and, subsequently, positive outcomes for at-risk infants and children. As such, the American Academy of Pediatrics (AAP) recommends that all 18- and 24-month-olds be screened for early signs of autism (Zwaigenbaum et al. 2013, 2009).

Additionally, biological and genetic tests are being developed to screen for and potentially diagnose autism in young and at-risk infants. For example, using magnetic resonance imaging (MRI) technology in addition to behavioral assessment, Shen et al. (2013) prospectively studied at-risk infants and found that all infants who were later diagnosed with autism had higher volumes of extra-axial fluid in their frontal lobes at 6 to 9 months than infants not at risk. Furthermore, the larger the volume of fluid at 6 months, the more severe the symptoms of autism were at 24 months of age (Shen et al. 2013). While further research is needed before these tools will be ready for clinical application, considerable progress is anticipated.

Current Knowledge

Existing research on autism symptoms in 2-year-olds indicates that social deficits may include

limited eye contact and gestures, preference for being alone, infrequent shared affect or joint attention, and lack of interest in social games and turn-taking. In addition to delays in the development of expressive language, children may also display limited vocal and motor imitation, lack of pretend play, abnormal quality of vocalizations, and poor responsivity to speech. Some children may display stereotyped motor mannerisms or preoccupation with parts of objects in the second year of life; however, expression of repetitive behaviors can be relatively mild in some young children before 3 years of age and can become more pronounced in the preschool years. This pattern has led some experts to caution that ASD cannot be ruled out in very young children who appear to present with autistic impairments in social communication but do not meet full diagnostic criteria because repetitive behaviors are not prominent (Lord 1995; Lord et al. 2006). Regression is reported in 20–30% of cases, though there is variability in the definition and manifestation of regression, with some children showing atypical development prior to regression, some children showing developmental slowing or plateau, and others showing clear loss of skills previously acquired.

In the second year of life, prior to the second birthday, deficits in eye contact, pointing, and general social engagement are also indicative of later autism. One study identified early markers of autism in infants at 12, 18, and 24 months of age (Barbaro and Dissanayake 2012). At all three time points, poor eye contact and lack of pointing were correlated with higher rates of autism as the children aged. Between 12 and 18 months of age, deficits may be observed in gaze shifting, shared affect, joint attention, gesture use, rate of communication, language comprehension, and symbolic play (Woods and Wetherby 2003). Additionally, irregular or delayed motor behavior, emotional dysregulation, and reduced levels of activity may also predict later autism diagnoses in young infants. Guinchat et al. (2012) polled parents of children diagnosed with autism and determined that motor problems and passivity were identified by parents in children around 14.6 months of age, who were later diagnosed with autism. Later signs

(e.g., problems with sleep, emotional regulation, and hyperactivity) were noticed by parents when their children were closer to 15 or 16 months old. Even later, parents identified problems with communication and social interactions in their children aged 22.3 months, on average (Guinchat et al. 2012). It has also been documented that infant siblings later diagnosed with ASD showed higher levels of repetitive behavior between 12 and 24 months than high-risk siblings who did not later meet ASD criteria (Wolff et al. 2014).

Prospective studies of infant siblings of children with ASD are now providing documentation of impairments emerging even by the first birthday, including impairments in eye contact, response to name, referential communication, social smiling, and engagement, as well as abnormalities in visual attention, object exploration, imitation and sensory behaviors, and emotional expression (Cassel et al. 2007; Gamliel et al. 2007; Landa and Garrett-Mayer 2006; Ozonoff et al. 2008; Paul et al. 2010; Yirmiya et al. 2006; Zwaigenbaum et al. 2005). It has been found that infants with high familial risk of autism may gesture, request, and initiate joint attention at lower rates than infant not at risk (Rozga et al. 2011). A review of the pertinent literature conducted by Jones et al. (2014) summarized that reduced social smiling, directed vocalizations, and social responsiveness are all predictors of later diagnoses of autism in at-risk infants (Jones et al. 2014).

In contrast to this clear evidence of behavioral symptoms which at 12 months seem to differentiate children who will develop ASD, there is limited evidence of observable behavioral differences at 6 months of age which predict later ASD diagnosis. Research has noted that infant siblings of children with ASD at 6 months of age, even those who will go on to develop ASD themselves, appear to smile and vocalize at others and show clear social interest and engagement (Jones et al. 2014; Rogers 2009; Rozga et al. 2011; Young et al. 2009). In a prospective longitudinal study, researchers found that frequencies of gazing at faces, social smiling, and directed vocalizations were similar between at-risk and matched low-risk infants at 6 months of age, though by 12 months, differences were significant between

groups and predictive of later diagnosis of ASD (Ozonoff et al. 2010). Importantly, the neurodevelopmental processes which will eventually lead to autistic symptomatology may already be in progress at this age; however, existing methods for detecting behavioral symptoms of autism are not sensitive enough to detect disruption at this level. Experimental analyses of basic perceptual and cognitive processes involved in development of social interactions and communication are necessary in order to identify biological or neuropsychological markers indicating risk of autism prior to the emergence of clinical symptoms.

At the conclusion of the first decade of this research enterprise, it is clear that early social communication symptoms of ASD often, though not always, emerge between 6 and 18 months of age and show diverse patterns of symptom onset (Chawarska et al. 2014; Szatmari et al. 2016). Researchers have shown that a diagnosis of ASD made at 18 months of age for an at-risk infant is highly stable through 3 years of age, suggesting that early diagnosis is not only possible but important for early intervention (Ozonoff et al. 2015). However, over half of the infant siblings in this study who eventually received ASD diagnosis at 36 months, had not met diagnostic criteria at 18 months, suggesting that for some children the symptoms continue to emerge over the first 2 years of life. Though at present it is still very difficult to predict the individual trajectories of young children and no single atypical behavior at 12 months of age predicts later ASD diagnosis in all children, these prospective studies are providing evidence that children who will go on to develop ASD can be differentiated from children who will not develop ASD based on aspects of their behavioral presentation at 12 months. For instance, patterns of differences in motor behaviors, imitation, disengaging attention, eye contact, social smiling, social interest, joint attention, response to name, gesture, requesting, and vocalization appear in children who go on to receive confirmatory diagnoses of ASD (Mitchell et al. 2006; Ozonoff et al. 2010; Rozga et al. 2011; Szatmari et al. 2016). These findings indicate both the great strides made in recent years and the need for more specific and accurate

measurements of various behavioral observations in infants, especially those 6 months old and younger.

Future Directions

ASD is currently understood to involve disruptions in early emerging mechanisms of socialization. While the exact etiology is currently unknown, the symptoms which result in a diagnosis of ASD are understood to be the outcome of complex interactions between congenital impairments and the effects of these impairments on subsequent environmental learning opportunities (Dawson 2008). It is assumed that the pathogenetic process which gives rise to objectively observable symptoms of autism in the second year of life is already underway before the full syndrome is expressed and behavioral symptoms are detected. In order to improve early detection, understanding of etiology, and ultimately treatment or prevention efforts, identification of biological markers which indicate risk is critically needed (Klin et al. 2008; Tager-Flusberg 2010). Researchers are conducting detailed experimental analyses of a variety of biological and neuropsychological processes in search of behavioral and neurobiological endophenotypes. It is hoped that prospective collection of a variety of possible measures including biological samples, neuroimaging data, audio and video recordings, as well as data from eye tracking and event-related potential (ERP) methods will soon lead to the identification of measures which reliably predict later ASD classification. For instance, Jones and Klin (2015) used an eye tracking task to show that infants later diagnosed with ASD displayed decreasing fixation to the speaker's eye region between 2 and 6 months of age. Evidence of abnormal brain development has also been observed during the first year of life (e.g., Courchesne et al. 2003), prior to overt behavioral abnormalities. Not only will these results provide greater information about symptom onset and aid in earlier identification of children at risk; they are expected to provide important clues as to the

mechanisms of socialization and patterns of disruption in development which give rise to ASD.

Identification of children who are likely to develop ASD, but before the behavioral symptoms can be detected, provides particularly promising opportunity for intervention to be applied. The majority of evidence-based interventions for autism were developed for preschool and school-age children and have not been validated with infants and toddlers with ASD (Boulware et al. 2006; Koegel et al. 2008; Wetherby and Woods 2006); however, the earlier children are identified as at-risk for ASD, the stronger the ethical imperative to develop interventions applicable to these infants and toddlers. Initiation of intervention in infancy allows for targeting the earliest emerging social communication abnormalities. Intervention at this age, before clinical symptoms have fully emerged, also has the potential to alter these infants' developmental trajectories and reduce the severity of symptom presentation (Dawson 2008).

Research is now emerging supporting the potential positive effects of developmentally sensitive interventions for infants (Rogers et al. 2014). Green et al. (2017) found, in a randomized controlled trial of 7- to 10-month-old high-risk siblings, that a video-based feedback program designed to increase communication between infants and their caregivers resulted in increased infant attentiveness to parents and reduced autism risk behaviors. While additional large and well-controlled trials will be needed to establish treatment efficacy and best practices for infants, pilot studies have been promising and warrant further investigation (Bradshaw et al. 2015).

In conclusion, the past decade has seen tremendous progress in understanding the earliest signs of ASD and illuminating the complexity of identifying infants at risk. The large network of prospective studies of infant siblings of children with ASD has yielded invaluable data and insights. It is anticipated that research efforts over the next several years will continue to refine early markers of risk for ASD and provide more clear guidance regarding effective treatment and even prevention approaches.

See Also

- [ADI-R](#)
- [ADOS](#)
- [Autism](#)
- [Autistic Disorder](#)
- [Early Diagnosis](#)
- [PDD-NOS \(Pervasive Developmental Disorder Not Otherwise Specified\)](#)
- [Retrospective Infant Video Analysis](#)

References and Reading

- Abrahams, B., & Geschwind, D. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, 9, 341–355.
- Adrien, J. L., Lenoir, P., Martineau, J., Perrot, A., Hameury, L., Larmande, C., & Sauvage, D. (1993). Blind ratings of early symptoms of autism based on family home movies. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 617–626.
- Baghdadli, A., Picot, M. C., Pascal, C., Pry, R., & Aussilloux, C. (2003). Relationship between age of recognition of first disturbances and severity in young children with autism. *European Child and Adolescent Psychiatry*, 12(3), 122–127.
- Baranek, G. T. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders*, 29(3), 213–224.
- Barbaro, J., & Dissanayake, C. (2012). Developmental profiles of infants and toddlers with autism spectrum disorders identified prospectively in a community-based setting. *Journal of Autism and Developmental Disorders*, 42(9), 1939–1948.
- Bhat, A. N., Galloway, J. C., & Landa, R. J. (2012). Relation between early motor delay and later communication delay in infants at risk for autism. *Infant Behavior and Development*, 35(4), 838–846.
- Bolton, P., Macdonald, H., Pickles, A., Rios, P. A., Goode, S., Crowson, M., et al. (1994). A case control family history study of autism. *Journal of Child Psychology and Psychiatry*, 35(5), 877–900.
- Boulware, G. L., Schwartz, I. S., Sandall, S. R., & McBride, B. J. (2006). Project DATA for toddlers: An inclusive approach to very young children with autism spectrum disorder. *Topics in Early Childhood Special Education*, 26(2), 94–105.
- Bradshaw, J., Steiner, A. M., Gengoux, G., & Koegel, L. K. (2015). Feasibility and effectiveness of very early intervention for infants at-risk for autism spectrum disorder: A systematic review. *Journal of Autism and Developmental Disorders*, 45, 778–794.
- Bryson, S. E., Zwaigenbaum, L., McDermott, C., Rombough, V., & Brian, J. (2008). The autism observation scale for infants: Scale development and reliability data. *Journal of Autism and Developmental Disorders*, 38(4), 731–738.
- Cassel, T. D., Messinger, D. S., Ibanez, L. V., Haltigan, J. D., Acosta, S. I., & Buchman, A. C. (2007). Early social and emotional communication in the infant siblings of children with autism spectrum disorders: An examination of the broad phenotype. *Journal of Autism and Developmental Disorders*, 37, 122–132.
- Chawarska, K., Klin, A., Paul, R., Macari, S., & Volkmar, F. (2009). A prospective study of toddlers with ASD: Short-term diagnostic and cognitive outcomes. *Journal of Child Psychology and Psychiatry*, 50(10), 1235–1245.
- Chawarska, K., Klin, A., & Volkmar, F. R. (Eds.). (2008). *Autism spectrum disorders in infants and toddlers: Diagnosis, assessment, and treatment*. New York: Guilford Press.
- Chawarska, K., Paul, R., Klin, A., Hannigen, S., Dichtel, L. E., & Volkmar, F. (2007). Parental recognition of developmental problems in toddlers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(1), 62–72.
- Chawarska, K., Shic, F., Macari, S., Campbell, D. J., Brian, J., Landa, R., Hutman, T., et al. (2014). 18-month predictors of later outcomes in younger siblings of children with autism spectrum disorder: A baby siblings research consortium study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53, 1317–1327.
- Christensen, D. L., Bilder, D. A., Zahorodny, W., Pettygrove, S., Durkin, M. S., Fitzgerald, R. T., et al. (2016). Prevalence and characteristics of autism spectrum disorder among 4-year-old children in the autism and developmental disabilities monitoring network. *Journal of Developmental & Behavioral Pediatrics*, 37(1), 1–8.
- Courchesne, E., Carper, R., & Akshoomoff, N. (2003). Evidence of brain overgrowth in the first year of life in autism. *JAMA*, 290(3), 337–344.
- Dawson, G. (2008). Early behavioral intervention, brain plasticity, and the prevention of autism spectrum disorder. *Development and Psychopathology*, 20(3), 775–803.
- De Giacomo, A., & Fombonne, E. (1998). Parental recognition of developmental abnormalities in autism. *European Child and Adolescent Psychiatry*, 7(3), 131–136.
- Elsabbagh, M., & Johnson, M. H. (2010). Getting answers from babies about autism. *Trends in Cognitive Sciences*, 14(2), 81–87.
- Gamliel, I., Yirmiya, N., & Sigman, M. (2007). The development of young siblings of children with autism from 4 to 54 months. *Journal of Autism and Developmental Disorders*, 37(1), 171–183.
- Green, J., Pickles, A., Pasco, G., Bedford, R., Wan, M. W., Elsabbagh, M., et al. (2017). Randomized trial of a parent-mediated intervention for infants at high risk for autism: Longitudinal outcomes to age 3 years. *The*

- Journal of Child Psychology and Psychiatry*, 58(12), 1330–1340.
- Guinchat, V., Chamak, B., Bonniau, B., Bodeau, N., Perisse, D., Cohen, D., & Danion, A. (2012). Very early signs of autism reported by parents include many concerns not specific to autism criteria. *Research in Autism Spectrum Disorders*, 6(2), 589–601.
- Jones, E. J. H., Gliga, T., Bedford, R., Charman, T., & Johns, M. H. (2014). Developmental pathways to autism: A review of prospective studies of infants at risk. *Neuroscience and Biobehavioral Reviews*, 39, 1–33.
- Jones, E. J. H., & Klin, A. (2015). Attention to eyes is present but in decline in 2-6-month-old infants later diagnosed with autism. *Nature*, 504, 427–431.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Klin, A., Chawarska, K., & Volkmar, F. (2008). Opportunities for research: Concepts and future directions. In K. Chawarska, A. Klin, & F. R. Volkmar (Eds.), *Autism spectrum disorders in infants and toddlers: Diagnosis, assessment, and treatment* (pp. 327–336). New York: Guilford Press.
- Koegel, L. K., Koegel, R. L., Fredeen, R. M., & Gengoux, G. W. (2008). Naturalistic behavioral approaches to treatment. In K. Chawarska, A. Klin, & F. R. Volkmar (Eds.), *Autism spectrum disorders in infants and toddlers: Diagnosis, assessment, and treatment* (pp. 207–242). New York: Guilford Press.
- Landa, R., & Garrett-Mayer, E. (2006). Development in infants with autism spectrum disorders: a prospective study. *Journal of Child Psychology and Psychiatry*, 47(6), 629–638.
- Lord, C. (1995). Follow-up of two-year-olds referred for possible autism. *Journal of Child Psychology and Psychiatry*, 36(8), 1365–1382.
- Lord, C., Risi, S., DiLavore, P. S., Shulman, C., Thurman, A., & Pickles, A. (2006). Autism from 2 to 9 years of age. *Archives of General Psychiatry*, 63(6), 694–701.
- Lord, C., Rutter, M., DiLavore, P. C., Risi, S., Gotham, K., & Bishop, S. (2012). *Autism diagnostic observation schedule: ADOS-2*. Los Angeles: Western Psychological Services.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(5), 659–685.
- Luyster, R., Gotham, K., Guthrie, W., Coffing, M., Petrak, R., Pierce, K., et al. (2009). The autism diagnostic observation schedule-toddler module: A new module of a standardized diagnostic measure for autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 1305–1320.
- Mitchell, S., Brian, J., Zwaigenbaum, L., Roberts, W., Szatmari, P., Smith, I., & Bryson, S. (2006). Early language and communication development of infants later diagnosed with autism spectrum disorder. *Journal of Developmental & Behavioral Pediatrics*, 27(2), S69–S78.
- Osterling, J. A., Dawson, G., & Munson, J. A. (2002). Early recognition of 1-year-old infants with autism spectrum disorder versus mental retardation. *Development and Psychopathology*, 14(2), 239–251.
- Ozonoff, S., Iosif, A., Baguio, F., Cook, I. C., Hill, M. M., Hutman, T., et al. (2010). A prospective study of the emergence of early behavioral signs of autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(3), 256–266.
- Ozonoff, S., Young, G. S., Landa, R. J., Brian, J., Bryson, S., Charman, T., et al. (2015). Diagnostic stability in young children at risk for autism spectrum disorder: A baby siblings research consortium study. *Journal of Child Psychology and Psychiatry*, 56(9), 988–998.
- Ozonoff, S., Macari, S., Young, G. S., Goldring, S., Thompson, M., & Rogers, S. J. (2008). Atypical object exploration at 12 months of age is associated with autism in a prospective sample. *Autism*, 12(5), 457–472.
- Ozonoff, S., Young, G. S., Carter, A., Messinger, D., Yirmiya, N., Zwaigenbaum, L., et al. (2011). Recurrence risk for autism spectrum disorders: A Baby Siblings Research Consortium study. *Pediatrics*, 128(3), e488–e495.
- Paul, R., Fuerst, Y., Ramsay, G., Chawarska, K., & Klin, A. (2010). Out of the mouths of babes: Vocal production in infant siblings of children with ASD. *The Journal of Child Psychology and Psychiatry*, 52(5), 588–598.
- Robins, D. L., Fein, D., Barton, M. L., & Green, J. A. (2001). The modified checklist for autism in toddlers: An initial study investigating the early detection of autism and pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 31(2), 131–144.
- Rogers, S. (2009). What are infant siblings teaching us about autism in infancy? *Autism Research*, 2, 125–137.
- Rogers, S. J., Vismara, L., Wagner, A. L., McCormick, C., Young, G., & Ozonoff, S. (2014). Autism treatment in the first year of life: A pilot study of infant start, a parent-implemented intervention for symptomatic infants. *Journal of Autism and Developmental Disorders*, 44, 2981–2995.
- Rozga, A., Hutman, T., Young, G. S., Rogers, S. J., Ozonoff, S., Dapretto, M., & Sigman, M. (2011). Behavioral profiles of affected and unaffected siblings of children with autism: Contribution of measures of mother-infant interaction and nonverbal communication. *Journal of Autism and Developmental Disorders*, 41, 287–301.
- Shen, M. D., Nordahl, C. W., Young, G. S., Wootton-Gorges, S. L., Lee, A., Liston, S. E., et al. (2013). Early brain enlargement and elevated extra-axial fluid in infants who develop autism spectrum disorder. *Brain*, 136, 2825–2835.
- Stone, W. L., Coonrod, E. E., & Ousley, O. Y. (2000). Brief report: Screening tool for autism in two-year-olds (STAT): Development and preliminary data. *Journal of Autism and Developmental Disorders*, 30, 607–612.
- Szatmari, P., Chawarska, K., Dawson, G., Georgiades, S., Landa, R., Lord, C., Messinger, D. S., et al. (2016).

Prospective longitudinal studies of infant siblings of children with autism: Lessons learned and future directions. *Journal of the American Academy of Child and Adolescent Psychiatry*, 55, 179–187.

- Tager-Flusberg, H. (2010). The origins of social impairments in autism spectrum disorder: Studies of infants at risk. *Neural Networks*, 23, 1072–1076.
- Volkmar, F. R., Stier, D. M., & Cohen, D. J. (1985). Age of recognition of pervasive developmental disorder. *American Journal of Psychiatry*, 142(12), 1450–1452.
- Werner, E., Dawson, G., Munson, J., & Osterling, J. (2005). Variation in early developmental course in autism and its relation with behavioral outcome at 3–4 years of age. *Journal of Autism and Developmental Disorders*, 35(3), 337–350.
- Wetherby, A. M., & Woods, J. J. (2006). Early social interaction project for children with autism spectrum disorders beginning in the second year of life: A preliminary study. *Topics in Early Childhood Special Education*, 26(2), 67–82.
- Wolff, J. J., Botteron, K. N., Dager, S. R., Elison, J. T., Estes, A. M., Gu, H., Hazlett, H. C., et al. (2014). Longitudinal patterns of repetitive behavior in toddlers with autism. *Journal of Child Psychology and Psychiatry*, 55, 945–953.
- Woods, J. J., & Wetherby, A. M. (2003). Early identification of and intervention for infants and toddlers who are at risk for autism spectrum disorder. *Language, Speech, and Hearing Services in Schools*, 34(3), 180–193.
- Yirmiya, N., Gamliel, I., Pilowsky, T., Feldman, R., Baron-Cohen, S., & Sigman, M. (2006). The development of siblings of children with autism at 4 and 14 months: Social engagement, communication, and cognition. *Journal of Child Psychology and Psychiatry*, 47(5), 511–523.
- Young, G. S., Merin, N., Rogers, S. J., & Ozonoff, S. (2009). Gaze behavior and affect at 6 months: Predicting clinical outcomes and language development in typically developing infants and infants at risk for autism. *Developmental Science*, 12(5), 798–814.
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, 23(2–3), 143–152.
- Zwaigenbaum, L., Thurm, A., Stone, W., Baranek, G., Bryson, S., Iverson, J., et al. (2007). Studying the emergence of autism spectrum disorders in high-risk infants: Methodological and practical issues. *Journal of Autism and Developmental Disorders*, 37(3), 466–480.
- Zwaigenbaum, L., Bryson, S., Lord, C., Rogers, S., Carter, A., Carver, L., et al. (2009). Clinical assessment and management of toddlers with suspected autism spectrum disorder: Insights from studies of high-risk infants. *Pediatrics*, 123(5), 1383–1391.
- Zwaigenbaum, L., Bryson, S., & Garon, N. (2013). Early identification of autism spectrum disorders. *Behavioural Brain Research*, 251, 133–146.

Infant-Toddler Social and Emotional Assessment (ITSEA)

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Description

The Infant-Toddler Social and Emotional Assessment (ITSEA) is an empirically validated clinical tool that was developed to assess social-emotional and behavior problems as well as delays or deficits in the acquisition of competencies that may arise between the ages of 12 and 36 months. The ITSEA includes parent and childcare provider forms that can be completed independently as a questionnaire or administered verbatim as an interview. The ITSEA was designed to provide a comprehensive profile of social-emotional and behavior problems and competencies and includes the following domains and subscales: (1) Externalizing, which is comprised of the Activity/Impulsivity, Aggression/Defiance, and Peer Aggression subscales; (2) Internalizing, which is comprised of Depression/Withdrawal, General Anxiety, Separation Distress, and Inhibition to Novelty; (3) Dysregulation, which is comprised of Negative Emotionality, Sleep Problems, Eating Problems, and Sensory Sensitivity; and (4) Competence, which is comprised of Attention, Mastery, Motivation, Imitation/Play, Empathy, and Prosocial Peer Relations. There are also three Item Clusters, Maladaptive, Social Relatedness, and Atypical, which include clinically significant behaviors that occur rarely in the general population as well as an additional ten infrequently occurring items of clinical significance that did not fit neatly in any of the domains, subscales, or item clusters (e.g., runs away in public places). The ITSEA also includes questions about parental concern.

The ITSEA items are rated on a three point scale (0 = “Rarely/Not True of My Child”; 1 = “Sometimes/Somewhat True of My Child”; 2 = “Often/Very True of My Child”). The Parent Form

is written at a fourth- to sixth-grade level and takes about 25 min to complete. An important feature of the ITSEA for use with young children with or at risk for autism spectrum disorders (ASD) is that all of the scales can be scored for nonverbal children. For some items, a “No Opportunity” response is offered, for example, if parents have not had the opportunity to observe their child engaging in particular behaviors with peers.

The ITSEA can be scored by hand or with a computer scoring program. Cut scores at the 10th percentile, reflecting the *Of Concern* range, are offered for domains, subscales, and item clusters. In addition, T scores, with a mean of 50 and a standard deviation of 10, are provided for domain scores. In addition to reviewing the specific pattern of scores that are in the *Of Concern* range, tables are provided that show how unusual it is for children to obtain multiple *Of Concern* scores within and across domains. For example, in the normative sample, 21.3% of girls had at least one Internalizing subscale in the *Of Concern* range, but only 5.3% of girls had more than one Internalizing subscale in the *Of Concern* range (Carter and Briggs-Gowan 2006).

The ITSEA is published and distributed by Pearson Assessment in English and Spanish. It has been translated and used in research in many other languages, including Dutch, French, Turkish, Hebrew, Portuguese, Mandarin, Cantonese, German, and Arabic.

Data on ITSEA profiles for children with autistic disorder is presented in the ITSEA manual (Carter and Briggs-Gowan 2005). A comparison of children in the national standardization sample to children with autism referred from early intervention clinics whose diagnoses were confirmed by clinical impression and with both the Autism Diagnostic Observational Scales (ADOS; Lord et al. 2000) and the Autism Diagnostic Interview Revised (Rutter et al. 2003) revealed the following:

Consistent with expectations, there were striking differences between children with autism and age-matched controls in the normative sample in the Competence domain and subscale scores with very large effect sizes. Striking differences (with

large effect sizes) were also observed in the Maladaptive, Social Relatedness, and Atypical Index Clusters. Differences were also observed in the Dysregulation domain, with children having autism rated as having more problems in the Eating, Negative Emotionality, and Sensory Sensitivity subscales. Statistically and clinically significant differences were also seen in the Internalizing domain Depression/Withdrawal subscale and the Externalizing domain Activity/Impulsivity subscale, with children with autism rated as having more problems in these areas. Children with autism were rated as having fewer problems on the Peer Aggression subscale, possibly reflecting their lack of engagement with or interest in peers.

The BITSEA (Briggs-Gowan and Carter 2006; Briggs-Gowan et al. 2004) is a briefer screening tool that can be used in pediatric, early intervention and childcare settings. It is comprised of 42 ITSEA items, with 11 items contributing the Competence Scale and 31 items contributing to the Problem Scale. The BITSEA is designed as a broad social-emotional and behavioral health screener, but appears to be sensitive and specific as an ASD screener (Briggs-Gowan and Carter 2006). Although not a focus of this entry, the BITSEA has demonstrated strong psychometric properties and parent acceptability (Briggs-Gowan et al. 2004).

Historical Background

Development of the ITSEA started in the early 1990s, in recognition that there were no questionnaires to assess problem behaviors in the first years of life. The selection of items and development of the ITSEA scales and domains began with a review of the relevant developmental psychology and psychopathology literatures, with a focus on early emerging, clinically significant behaviors that can be reliably assessed in 1- and 2-year-old children. In addition to clinical case studies and research articles, several diagnostic manuals were reviewed, including the Diagnostic and Statistical Manual – Fourth Edition, Text Revision (DSM-IV-TR; American Psychiatric Association

[APA] 2000), and the Diagnostic classification of mental health and developmental disorders of infancy and early childhood (DC: 0–3; Zero-to-Three 1994) to ensure comprehensive coverage of symptoms relevant to early emerging psychiatric conditions. Based on this review, three Problem domains were included: Externalizing, Internalizing, and Dysregulation. For comprehensive coverage, symptoms of rare disorders (e.g., Tourette Disorder, Post-Traumatic Stress Disorder) were also included.

Including the Externalizing and Internalizing domains was consistent with (Achenbach's 1966) extensive work in defining these broad domains and a then emerging consensus that these problems could be identified in early childhood. The inclusion of scales to address problems with the regulation of mood, state (sleep and eating), and sensory sensitivities was deemed particularly relevant for infants and toddlers. The Competence domain was included in an effort to comprehensively characterize early social-emotional adjustment and because the presence of social-emotional competence was viewed as minimizing the emergence and persistence of problem behaviors. In addition, by asking caregivers to rate both problems and competencies, response biases may be minimized.

As part of the development of the ITSEA, each of the items included in the ITSEA were reviewed by a panel of experts who rated developmental appropriateness as well as content fit on subscales and domains. Focus groups were conducted with parents and childcare providers to evaluate comprehensiveness and ease of item comprehension. In addition, items were evaluated in an epidemiological sample and in a national standardization sample for psychometric properties. Acceptability for the ITSEA was very strong, with over 90% of parents indicating that they would recommend the ITSEA to a friend with a young child.

The ITSEA was first piloted in a pediatric setting and then included to assess infant-toddler mental health in a large epidemiological sample. Most recently, it has been nationally standardized for 1- and 2-year olds. An upward extension to age 6 years is planned.

Psychometric Data

The version of the ITSEA Parent Form that is currently published by Pearson Assessment was standardized and normed based on a nationally representative sample that was stratified to match the 2002 US Census. Unfortunately, at present, although the ITSEA Childcare Provider Form item responses and scales can be compared to the Parent Form, there are no Childcare Provider Form norms. The ITSEA Parent Form domains and subscales have adequate-to-good internal consistency, good-to-excellent test-retest reliability, and strong inter-rater reliabilities in the national normative sample, which is consistent with earlier work in an epidemiological study (Carter and Briggs-Gowan 2005). Specifically, in the normative sample, the internal consistency or reliability of the ITSEA Parent Form domains ranged from .85 to .89 for girls and from .85 to .89 for boys. Test-retest coefficients ranged from .76 for Competence to .91 for Dysregulation. Inter-rater reliabilities across parents ranged from .72 for Internalizing to .79 for Competence. The ITSEA also has demonstrated strong validity based on confirmatory factor analytic work (Carter et al. 2003), and strong validity in relation to other measures such as the CBCL 1.5–5 (Achenbach and Rescorla 2000), the Ages and Stages Social-Emotional Questionnaires (ASQ: SE; Squires et al. 2002), the Adaptive Behavior Assessment System: Second Edition (ABAS-II; Harrison and Oakland 2003), and scores on the Bayley Scales of Infant and Toddler Development – Third Edition (Bayley III; Bayley 2006).

Clinical Uses

The ITSEA is an appropriate tool for evaluating a 1- or 2-year-old when the goal of an assessment is to gather a comprehensive profile of a young child's social-emotional and behavior problems and competencies. Thus, the ITSEA may be most appropriately used in the early stages of a clinical evaluation to get an idea of the parents' perceptions of the child's current social-emotional functioning. Although not a diagnostic tool, items

in the ITSEA can inform further inquiry about multiple aspects of social-emotional and behavioral functioning as well as symptoms relevant to ASD and other diagnostic statuses. Thus, while ITSEA items do not address symptom onset, offset or duration, which are required for assigning diagnoses, responses to ITSEA items do provide clinicians with initial information about whether or not in the past month a symptom was present, and if present, the frequency or extent to which the behavior was present. The ITSEA also provides information about the absence of social-emotional competencies, which, for very young children, may be critical for raising concerns about a diagnosis of ASD. Problems are assessed with the Externalizing domain, including symptoms of disruptive behaviors, the Internalizing domain, including symptoms of anxiety, depression, and temperamental shyness or inhibition, and the Dysregulation domain, addressing emotional reactivity and regulation, sensory processing difficulties, and sleep and eating problems. It is not unusual for parents to report clinically concerning behaviors on the ITSEA that are reflected in clinically elevated scores on multiple domains and subscales, yet not be concerned about their young child's behavior or social-emotional development. This may be due to parents' limited knowledge of typical development in this age range. It can be extremely useful for the clinician to have knowledge both about the behaviors that the parent is observing and their level of concern about these behaviors prior to beginning a clinical feedback session.

If using the ITSEA as part of determining eligibility or need for services, it is recommended that the ITSEA be coupled with direct observation of the child and discussion with the parents to better contextualize the caregivers' responses on the ITSEA. Research on young children with ASD has used the ITSEA to document relations between sensory sensitivities and anxiety symptoms (Ben-Sasson et al. 2008; Green et al. 2012). The ITSEA may be useful in documenting the presence of unusual behaviors such as social withdrawal and limited or atypical eye contact,

repetitive behaviors and sensory sensitivities. In addition, it is particularly effective in documenting severity of delays or deficits in competencies such as imitation and play, prosocial peer relations, empathy, attention, and compliance. Moreover, as many children with autism also evidence problems in eating and sleeping, elevated anxiety, and attentional problems that are not central to their ASD symptomatology but that may be crucial for appropriate intervention planning, gathering information about both autism-specific symptoms as well as broader information about social-emotional adaptation is warranted. The ITSEA provides a relatively quick and efficient method for obtaining a great deal of information about caregiver appraisals regarding a comprehensive set of social-emotional problems and competencies that can inform both autism spectrum disorder diagnosis and the need to assess further to identify co-occurring emotional and behavioral concerns.

See Also

- ▶ ADHD
- ▶ Affective Disorders (Includes Mood and Anxiety Disorders)
- ▶ Anxiety Disorders
- ▶ Child Behavior Checklist in Autism
- ▶ Comorbidity
- ▶ Sensory Processing Assessment

References and Reading

- Achenbach, T. M. (1966). The classification of children's psychiatric symptoms: A factor-analytic study. *Psychological Monographs: General and Applied*, 80(7), 1–37. <https://doi.org/10.1037/h0093906>.
- Achenbach, T. M., & Rescorla, L. A. (2000). *Manual for the ASEBA preschool forms and profiles*. Burlington: University of Vermont, Department of Psychiatry.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
- Bayley, N. (2006). *Bayley scales of infant and toddler development*. San Antonio: The Psychological Corporation.

- Ben-Sasson, A., Cermak, S. A., Orsmond, G. I., Tager-Flusberg, H. H., Kadlec, M. B., & Carter, A. S. (2008). Sensory clusters of toddlers with autism spectrum disorders: Differences in affective symptoms. *Journal of Child Psychology and Psychiatry*, 49(8), 817–825. <https://doi.org/10.1111/j.1469-7610.2008.01899.x>.
- Briggs-Gowan, M. J., & Carter, A. S. (1998). Preliminary acceptability and psychometrics of the infant-toddler social and emotional assessment (ITSEA): A new adult-report questionnaire. *Infant Mental Health Journal*, 19(4), 422–445. [https://doi.org/10.1002/\(SICI\)1097-0355\(199824\)19:4<422::AID-IMHJ5>3.0.CO;2-U](https://doi.org/10.1002/(SICI)1097-0355(199824)19:4<422::AID-IMHJ5>3.0.CO;2-U).
- Briggs-Gowan, M. J., & Carter, A. S. (2006). *The Brief Infant-Toddler Social & Emotional Assessment (BITSEA)*. San Antonio: Psychological Corporation, Harcourt Assessment.
- Briggs-Gowan, M. J., & Carter, A. S. (2007). Applying the infant-toddler social and emotional assessment (ITSEA) and Brief-ITSEA in early intervention. *Infant Mental Health Journal*, 28(6), 564–583. <https://doi.org/10.1002/imhj.20154>.
- Briggs-Gowan, M. J., & Carter, A. S. (2008). Social-emotional screening status in early childhood predicts elementary school outcomes. *Pediatrics*, 121(5), 957–962. <https://doi.org/10.1542/peds.2007-1948>.
- Briggs-Gowan, M. J., Carter, A. S., Irwin, J. R., Wachtel, K., & Cicchetti, D. V. (2004). The brief infant-toddler social and emotional assessment: Screening for social-emotional problems and delays in competence. *Journal of Pediatric Psychology*, 29(2), 143–155. <https://doi.org/10.1093/jpepsy/jsh017>.
- Briggs-Gowan, M. J., Carter, A. S., Bosson-Heenan, J., Guyer, A. E., & Horwitz, S. M. (2006). Are infant-toddler social-emotional and behavioral problems transient? *Journal of the American Academy of Child & Adolescent Psychiatry*, 45(7), 849–858. <https://doi.org/10.1097/01.chi.0000220849.48650.59>.
- Carter, A. S., & Briggs-Gowan, M. J. (2005). *The Infant-Toddler Social & Emotional Assessment (ITSEA) and Brief Infant-Toddler Social & Emotional Assessment (BITSEA)*. San Antonio: Psychological Corporation, Harcourt Assessment.
- Carter, A. S., & Briggs-Gowan, M. J. (2006). *The Infant-Toddler Social & Emotional Assessment (ITSEA)*. San Antonio: Psychological Corporation, Harcourt Assessment.
- Carter, A. S., Little, C., Briggs-Gowan, M. J., & Kogan, N. (1999). The Infant-Toddler Social and Emotional Assessment (ITSEA): Comparing parent ratings to laboratory observations of task mastery, emotion regulation, coping behaviors and attachment status. *Infant Mental Health Journal*, 20(4), 375–392. [https://doi.org/10.1002/\(SICI\)1097-0355\(199924\)20:4<375::AID-IMHJ2>3.0.CO;2-P](https://doi.org/10.1002/(SICI)1097-0355(199924)20:4<375::AID-IMHJ2>3.0.CO;2-P).
- Carter, A. S., Briggs-Gowan, M. J., Jones, S. M., & Little, T. D. (2003). The Infant-Toddler Social and Emotional Assessment (ITSEA): Factor structure, reliability, and validity. *Journal of Abnormal Child Psychology: An Official Publication of the International Society for Research in Child and Adolescent Psychopathology*, 31(5), 495–514. <https://doi.org/10.1023/A:1025449031360>.
- Green, S. A., Ben-Sasson, A., Soto, T. W., & Carter, A. S. (2012). Anxiety and sensory over-responsivity in toddlers with autism spectrum disorders: Bidirectional effects across time. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-011-1361-3>.
- Karabekiroglu, K., Rodopman-Arman, A., Ay, P., Ozkesen, M., Akbas, S., Tasdemir, G., et al. (2009). The reliability and validity of the Turkish version of the Brief Infant-Toddler Social Emotional Assessment (BITSEA). *Infant Behavior & Development*, 32(3), 291–297. <https://doi.org/10.1016/j.infbeh.2009.03.003>.
- Karabekiroglu, K., Briggs-Gowan, M. J., Carter, A. S., Rodopman-Arman, A., & Akbas, S. (2010). The clinical validity and reliability of the Brief Infant-Toddler Social and Emotional Assessment (BITSEA). *Infant Behavior & Development*, 33(4), 503–509. <https://doi.org/10.1016/j.infbeh.2010.07.001>.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. T. (2000). *The Autism Diagnostic Observation Schedule (ADOS)*. Los Angeles: Western Psychological Services.
- Rutter, M., LeCouteur, A., & Lord, C. (2003). *The Autism Diagnostic Interview, Revised (ADI-R)*. Los Angeles: Western Psychological Services.
- Squires, J., Bricker, D., & Twombly, E. (2002). *The ASQ: SE user's guide: For the ages & stages questionnaires: Social-emotional*. Baltimore: Paul H Brookes Publishing.
- Van Hulle, C. A., Lemery-Chalfant, K. K., & Goldsmith, H. H. (2007). Genetic and environmental influences on socio-emotional behavior in toddlers: An initial twin study of the infant-toddler social and emotional assessment. *Journal of Child Psychology and Psychiatry*, 48(10), 1014–1024. <https://doi.org/10.1111/j.1469-7610.2007.01787.x>.
- Visser, J. C., Smeekeens, S., Rommelse, N., Verkes, R. J., Van der Gaag, R. J., & Buitelaar, J. K. (2010). Assessment of psychopathology in 2- to 5-year-olds: Applying the infant-toddler social emotional assessment. *Infant Mental Health Journal*, 31(6), 611–629. <https://doi.org/10.1002/imhj.20273>.
- Weitzman, C., Edmonds, D., Davagnino, J., & Briggs-Gowan, M. (2011). The association between parent worry and young children's social-emotional functioning. *Journal of Developmental and Behavioral Pediatrics*, 32(9), 660–667. <https://doi.org/10.1097/DBP.0b013e31822bc76b>.
- Zero-to-Three. (1994). *Diagnostic classification of mental health and developmental disorders of infancy and early childhood*. Arlington: Zero-to-Three/National Center for Clinical Infant Programs.

Inferior Colliculi

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Definition

The inferior colliculus is part of the central auditory system; specifically, it is located in the tectal region of the midbrain, posterior to the superior colliculus, and anterior to the trochlear nerve. The inferior colliculus is the “site of brain stem convergence and multisensory integration” (Winer and Schreiner 2005, p. 3) containing neurons that are sensitive to interaural timing and intensity difference, which are important in sound localization. Other functions that have been shown to be related to this structure include sound integration, multisensory integration including startle reflex and vestibuloocular reflex, and amplitude modulation of frequencies necessary for detection of pitch.

As part of the central auditory pathway, the inferior colliculus receives projections from the superior olivary nuclei and the cochlear nuclei via the lateral lemniscus. The inferior colliculus sends projections to the medial geniculate nucleus, which in turn projects to the primary auditory cortex. The inferior colliculus consists of three regions – central nucleus, lateral nucleus, and dorsal cortex. The central nucleus is exclusively involved in auditory processing and is necessary for normal hearing. The central nucleus has numerous layers, arranged in a tonotopic map that is orthogonal to the layers. The lateral nucleus is multisensory, receiving both auditory and nonauditory input. The dorsal cortex receives projections from the cerebral cortex including auditory and somatosensory information. For an in-depth perspective of the inferior colliculus, please see Wilner and Schreiner *The Inferior Colliculus* (2005).

In animal models of autism, the gene *autism susceptibility candidate 2* (Auts2) is expressed in

the inferior and superior colliculi at embryonic day 19 of the mouse brain (Bedogni et al. 2010). Immunoreactive staining in the murine brain found high density of serotonin (5-HT) fibers in the inferior colliculus (Williams et al. 2005); selective serotonin reuptake inhibitor therapeutics are often prescribed in autism. In general, preclinical and animal models have implicated the brainstem in ASD (e.g., Hashimoto et al. 1995; Rodier 2002), but the specificity to the inferior colliculus is unclear.

Functional activity of the inferior colliculus can be assessed via wave V of the brainstem evoked auditory potential (BAEP), which is thought to result from activity of the upper pons and inferior colliculus. Rosenhall et al. (2003) found increased BAEP wave III-V interpeak intervals in children with autism; Chen et al. (2003) found children with autism had longer latencies in wave III and V, as well as increased wave I–II and I–V interpeak latencies. However, a number of other reports (as reviewed in Minshew et al. 2006) have not found consistent abnormalities in BAEP in individuals with autism or their family members. Deficits in tone perception in ASD have been found even in those children with intact click BAEPs; the authors suggested that early abnormal auditory pruning in the lateral lemniscus and inferior colliculus may lead to disrupted connections between the brainstem and cortex and impact auditory processing of linguistic cues (Russo et al. 2008).

See Also

► [Brainstem Auditory Evoked Response \(BAER\)](#)

References and Reading

- Bedogni, F., Hodge, R., Nelson, B., Frederick, E., Shiba, N., Daza, R., et al. (2010). Autism susceptibility candidate 2 (Auts2) encodes a nuclear protein expressed in developing brain regions implicated in autism neuropathology. *Gene Expressions Patterns*, 10(1), 9–15.
- Chen, W., Chen, Y., Chen, D., Chen, J., Yin, X., & Lin, Q. (2003). Brainstem auditory evoked potentials in children with autistic disorder. *Chinese Mental Health Journal*, 17, 24–26.

- Hashimoto, T., Tayama, M., Murakawa, K., Yoshimoto, T., Miyazaki, M., Harada, M., et al. (1995). Development of the brainstem and cerebellum in autistic patients. *Journal of Autism and Developmental Disorders*, 25, 1–18.
- Hsu, M., Yeung-Courchesne, R., Courchesne, E., & Press, G. (1991). Absence of magnetic resonance imaging evidence of pontine abnormality in infantile autism. *Archives of Neurology*, 48(11), 1160–1163.
- Kandel, E., Schwartz, J. H., & Jessell, T. (2000). Hearing. Part V. Perception. In *Principles of neuroscience* (4th ed.). New York: McGraw Hill. Chapter 30.
- Minshew, N., Sweeney, J., Bauman, M., & Webb, S. J. (2006). Neurological aspects of autism. In F. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook autism and pervasive development disorder* (Diagnosis, development, neurobiology and behavior, Indianapolis, Vol. 1). Indianapolis: Wiley. Chapter 18.
- Rodier, P. (2002). Converging evidence for brain stem injury in autism. *Development and Psychopathology*, 14, 537–557.
- Rosenhall, U., Nordin, V., Brantberg, K., & Gillberg, C. (2003). Autism and auditory brain stem responses. *Ear and Hearing*, 24, 206–214.
- Russo, N., Skoe, E., Trommer, T., Zecker, S., Bradlow, A., & Kraus, N. (2008). Deficient brainstem encoding of pitch in children with autism spectrum disorders. *Clinical Neurophysiology*, 119(8), 1720–1731.
- Williams, S., Bryan-luka, L., & Pow, D. (2005). Quantitative analysis of immunolabeling for serotonin and for glutamate transporters after administration of imipramine and citalopram. *Brain Research*, 1042(2), 224–232.
- Winer, J. A., & Schreiner, C. E. (2005). The central auditory system: A functional analysis. In J. Winer & C. Schreiner (Eds.), *The inferior colliculus*. New York: Springer. Chapter 1.

Inferior Parietal Area

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Synonyms

Inferior parietal gyrus

Definition

Autism spectrum disorder (ASD) is characterized by altered connectivity in cortical regions,

such as the inferior parietal lobe, involved with communication and emotion regulation (Hanaie et al. 2016). The inferior parietal lobe integrates sensory and motor information to generate spatiotemporal representations (Hanaie et al. 2016). Among ASD patients, several studies have shown reduced gray matter and cortex in the inferior parietal lobe (Brieber et al. 2007; Wallace et al. 2010). Furthermore, these studies suggest that cortical thinning of the parietal and temporal cortices results from abnormal cortical growth in ASD patients (Wallace et al. 2010). Further research is required to elucidate the atypical neural growth and developmental mechanisms observed in ASD patients.

See Also

- Fusiform Gyrus (FG)
- Gray Matter

References and Reading

- Brieber, S., Neufang, S., Bruning, N., Kamp-Becker, I., Remschmidt, H., Herpertz-Dahlmann, B., et al. (2007). Structural brain abnormalities in adolescents with autism spectrum disorder and patients with attention deficit/hyperactivity disorder. *Journal of Child Psychology and Psychiatry*, 48(12), 1251–1258. <https://doi.org/10.1111/j.1469-7610.2007.01799.x>.
- Hanaie, R., Mohri, I., Kagitani-Shimono, K., Tachibana, M., Matsuzaki, J., Hirata, I., et al. (2016). White matter volume in the brainstem and inferior parietal lobule is related to motor performance in children with autism spectrum disorder: A voxel-based morphometry study. *Autism Research*, 9(9), 981–992. <https://doi.org/10.1002/aur.1605>.
- Wallace, G. L., Dankner, N., Kenworthy, L., Giedd, J. N., & Martin, A. (2010). Age-related temporal and parietal cortical thinning in autism spectrum disorders. *Brain*, 133(12), 3745–3754. <https://doi.org/10.1093/brain/awq279>.

Inferior Parietal Gyrus

- Inferior Parietal Area

Inferior Temporal Area

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Synonyms

[Inferior temporal gyrus](#)

Definition

The inferior temporal gyrus is positioned within the temporal lobe below the middle temporal sulcus. It runs the course of the temporal lobe from the anterior temporal pole back to the inferior occipital gyrus. It extends down to the ventral or bottom surface of the brain where it stops at the inferior sulcus. Functionally, this area of the cerebral cortex is associated with higher-level visual processing. That is, it helps us to determine what we are seeing and contains regions sensitive to complex object features as well as particularly important categories of objects, such as faces and letters.

See Also

► [Temporal Lobes](#)

References and Reading

Allison, T., McCarthy, G., Nobre, A., Puce, A., & Belger, A. (1994). Human extrastriate visual cortex and the perception of faces, words, numbers, and colors. *Cerebral Cortex*, 4, 544–554.

Inferior Temporal Gyrus

► [Inferior Temporal Area](#)

Inflection

► [Register Variation](#)

Informal Assessment

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Definition

Informal assessment of individuals with autism spectrum disorders (ASD) involves the collection of information about the individual, interpreting that information and applying it in a systematic manner, the purpose of which is to better understand the individual's abilities. Although the overall goals of informal and formal assessment are similar, they differ in the use of the information collected, the manner in which the information is collected, and the type of information collected. The specific purpose of informal assessment is usually to use the information collected in order to set goals, identify intervention strategies, and measure intervention outcomes. In formal assessments, information is collected through the use of standardized, norm-referenced tests, whereas in informal assessment, information is based on careful observation of behaviors by the examiner. Types of information accumulated during the process of informal assessment include profiles of behavior, of capabilities, of learning style, and of motivational strategies, which then serve to assist in the selection of techniques to facilitate development, to set learning goals, and, in general, to support the individual's progress.

Historical Background

Historically, assessment procedures focused on the formal collection of information in areas of

concern for individuals with special needs or for those involved in the care of these individuals. Specifically, data were collected in order to substantiate strengths and weaknesses of the individual. The information was traditionally based on results from standardized testing of intelligence, aptitude, and academic abilities, as well as a medical history and current physical status as part of the assessment battery. The assumption was that the comparison between individuals would be more informative if each person was assessed in a similar manner. Over time, it became clear that, although this type of assessment provided some important information about individuals with ASD, the results obtained needed to be interpreted with caution because the results obtained in formal assessments did not necessarily reflect the abilities of the individual with ASD. In addition, these data did not assess the interactional or motivational aspects of the individual but rather focused on whether or not the individual could perform particular skills. Despite the difficulties with systematic assessment of information, informal assessment continued to be perceived as less professional than formal evaluations until the latter part of the twentieth century, when informal assessment procedures were incorporated into the assessment necessary for the development of the individualized education (IEP) objectives mandated by law (PL-94-142).

Current Knowledge

As a result of recent changes in assessment, the reliance on formal tests has given way to a more person-centered approach (Lord et al. 2005). Professionals acknowledge that assessment needs to recognize changes in behavior and not focus exclusively on successful task performance. Informal assessment procedures target not only what individuals know but also how they interact with the environment and how they learn and understand and function in the world around them. Informal assessment techniques do not require comparative norm-referenced groups against which to measure individuals' performance but rather individuals' performance is gauged by

comparison to their own performance levels within the specific demands of the situation or programs. Assessment differs from diagnosis in that while diagnostic evaluation addresses the grouping of common features under the same label, assessment refers to evaluating the unique and distinctive characteristics of the individual (Klin et al. 2005). A diagnosis may assist in establishing eligibility for intervention services, whereas an assessment identifies the unique and specific profile of strengths and weaknesses of a particular individual. Although many people may share the ASD diagnostic label, the manifestation of ASD in each individual is different, necessitating personal assessment. The purpose of such an assessment is to facilitate the development of individualized educational and behavior management programs tailored to the specifications of the individual (Hogan and Marcus 2008). Thus, in contrast to the process of diagnosis, which examines common characteristics in individuals with ASD, assessment looks at the unique aspects of individuals as the basis for the development of appropriate intervention plans.

Both formal and informal diagnostic assessments yield information which aids in the confirmation of diagnosis by detailing the specific syndrome expression in a particular individual, but informal assessment also augments diagnostic ascertainment with information regarding the identification of intervention needs and/or by providing a method to assess the effects of treatment. To highlight the similarities and differences in the information provided by informal and formal assessments, school psychologists were presented with information gleaned from formal or informal assessments and asked diagnostic and prescriptive questions about the children based on the information provided. Some qualitative differences in the efficacy of the information provided by each of the two types of assessment emerged. The behavioral descriptions from informal assessments provided the type of information necessary for identifying autistic symptomatology more correctly than information from formal assessments (Levy et al. 2009). While the information from both formal and informal assessments was found to be helpful in making Individualized

Educational Program (IEP) decisions, the suggested goals which emerged from each type of assessment were in different domains. Data from informal assessments led to goals in the areas of receptive language and behavior, whereas data from formal assessments led to academic goals, such as reading. In conclusion, this research reinforced the need for the information acquired from each type of assessment. Those who had received reports based on formal assessments requested more informal information and those who had received informal assessment information desired more formal data (Spears et al. 2001). Thus, the complementary information which is obtained from formal and informal assessments is essential to a comprehensive understanding of individuals with ASD.

The goals of informal assessment include gaining the most valuable information about individuals for whom traditional testing situations are inappropriate. Significant impairments in language and communication have an impact on the ability of these individuals to comprehend the language demands of standardized testing procedures. In addition, the social deficits in reciprocity and social cognition which are central to ASD present particular challenges when testing individuals with ASD. Finally, the behavioral and cognitive issues revolving around motivation, sensory interests, and repetitive, restricted areas of interest necessitate adaptations which often invalidate the standardized procedures of administration (Volkmar et al. 2005). Thus, because standardized assessments require the understanding of verbal instructions, are based on social reinforcement, and entail compliance, informal assessments are recommended for individuals with ASD (Lerman et al. 2004). Informal assessment is based on direct behavioral observations. This is accomplished through clinical observations of the individual's functioning in natural settings or in a formal assessment context, and data may be collected in a variety of settings, including classroom, workplace, home, clinic, and others. The physical environment, task presentation, level of interest, and past learning experience can all influence the assessment results and must be considered in understanding the results

from the assessment. Informal assessment requires no formal time constraints or standardized procedures, and therefore, the examiner can become more familiar with the individual and establish rapport.

Designing an assessment to elicit certain behaviors can also provide salient information. Therefore, it is important to take the individual behavioral expression of the triad of impairments upon which the diagnosis of ASD is based (American Psychiatric Association [APA] 2000) into consideration during informal assessments. Noting communication strategies, social interactional patterns, and repetitive, restrictive behaviors is an integral part of any assessment for individuals with ASD. For example, careful attention should be paid to the manner in which the individual asks for help. Likewise, it is important to note with whom the individual chooses to spend time and what behaviors affect the interactions. These parameters change according to the age and level of functioning of the individual being assessed. For example, when assessing a young child with ASD, it may be appropriate to allow the child to choose the object, task, or activity, thus beginning with a high interest task in order to maximize motivation. It is within the nature of this type of assessment that the examiner is free to vary the number and order of tasks. Examiners should be sensitive to the child's activity level, attention span, and tolerance for highly structured activity when presenting the different types of tasks. It has been found helpful, especially with developmentally young children, to balance activities between those which are sedentary and require focused attention and those which are more physical in nature in order to maintain interest and avoid frustration. Most evaluations of this type continue approximately 30–40 min, but they can vary in duration. Assessments such as these should be conducted in a relatively quiet, brightly lit room, but, if appropriate, can incorporate any space and activity such as walking in unfamiliar areas, which provides an opportunity to see how the child reacts to new places, people, and even experiences. Throughout the session, the quality and the quantity of the child's spontaneous initiations in language and behavior should be noted.

The behaviors observed during informal assessments should be recorded as they occur (Herzinger and Campbell 2007). While conducting an informal assessment, the examiner should find a way to document behavior as it appears in order to capture the quality of what is happening as well as the behavior itself. In order to facilitate data collection, it is crucial to establish a method which allows the examiner to take notes about each task, as well as to get an overall view of the items presented. In order to make basic decisions regarding educational placement, services required, and initial programming suggestions, it is necessary to interpret the information. The interpretation should include summary points as well as precise and detailed examples of behavior observed, which then serve as the basis for specific intervention recommendations. Informal assessment of individuals with autism spectrum disorders is a challenging task and involves expertise from various disciplines. Multidisciplinary teams work together to decide what information should be collected in order to understand the individual, and once the information has been collected, they are instrumental in interpreting the significance and implications of this information for the individual. In summary, clinical observation of individuals with ASD is a valuable skill. Despite its complexity, it should lead to a more comprehensive understanding of the person and, as a result, a more individually tailored treatment/intervention plan.

Informal assessment is dependent on making the assessment appropriate for the child's developmental level, bearing in mind that individuals with ASD often have uneven profiles of strengths and weaknesses. It is important to remember that no matter how much clinical and diagnostic information has been gained from an initial evaluation, it may or may not be representative of the individual's functioning in other settings or with other people. In order to put initial evaluation information into perspective, it is helpful to solicit certain information from other sources such as parents and professionals. A parent interview can often provide supplementary information which assists in the interpretation of the information gleaned from informal assessment.

If possible, parents should be interviewed while their child is not present. Parents and other professionals can add specific information about the child's cognitive and language abilities, as well as behavioral issues which may help the examiner make decisions regarding the results of the initial evaluation.

Since successful informal assessment is based on the clinical observational skills of the examiner rather than the specific tasks presented, the tasks themselves become less important. The value of the tasks in informal assessment lies in the fact that they serve as a vehicle for the examiner's observations, with more emphasis placed on other elements which can supply important information regarding the individual being assessed. For example, using appealing and developmentally appropriate materials is central to informal assessment. But carefully choosing the materials used in an assessment, it becomes possible to assess the individual's potential for creativity and interaction, to elicit cognitive and behavior styles, and to assess the conditions under which the individual is more likely to communicate and be socially involved. Thus, the observational skills of the examiner are far more important than the specific tasks which provide the context for the observed behavior. The examiner must be able to remain flexible and react individually according to the individual's interactive style and needs (National Research Council [NRC] 2001). Considerable clinical skill and experience are therefore needed in the use of informal assessment procedures. Above and beyond the results from standardized testing, informal assessment is an attempt to capture abilities and disabilities and behavior as they affect the individual's daily life (Klin et al. 2005).

Few guidelines exist for conducting informal skill assessments or for interpreting the results with individuals with ASD. Some suggestions include evaluating the child's performance on tasks from preexisting curriculum (Callahan et al. 2011), while others propose assessment of reinforcement techniques (Lerman et al. 2004). Herzinger and Campbell (2007) recommend using informal functional assessment techniques which focus on the child's everyday adaptive

functioning. Assessing behavior is an ongoing process designed to guide treatment planning. The results, describing characteristics and identifying the unique functional profile of each individual, can be used in planning intervention services. Informal assessment involves careful observation and integration of available information, while checking to see if additional information is necessary in order to develop a treatment plan. After delineating the various components of a possible intervention, treatment priorities need to be set by the parents and/or the professionals concerned. A treatment plan must be implemented after careful analysis of the information collected through informal assessment procedures outlined above. A mechanism for tracking individual progress and a system for ongoing assessment permit timely adjustments to the intervention program. Thus, informal assessment documents functioning before, during, and after intervention and is crucial for evaluating changes and progress over time. Employing assessment information in order to measure post intervention status is one way to evaluate treatment decisions and to specify the aspects of intervention which succeeded with a particular individual. In sum, ongoing informal assessment should be a part of every intervention program in order to monitor the child's progress toward established goals and aid in decision making in future interventions (Magiati and Howlin 2001). The assessment of individuals with autism spectrum disorders is a challenging task and involves expertise from various disciplines. The triad of impairments present in ASD makes formal assessment of individuals with ASD problematic and the incorporation of informal assessment techniques critical.

Future Directions

In order to be effective, the scope of informal assessment must be as broad as the number of individuals being assessed. The tension between deciding on the elements to be observed and evaluated in informal assessments and the flexibility necessary for adapting the assessment to each individual with ASD seems

insurmountable. The challenge inherent in informal assessment of individuals with ASD lies in abstracting the elements in present behavior from which future behavior can be inferred (NRC 2001). Assessment is an attempt to examine and understand human nature and as such is imperfect. Goals for improving informal assessment include training qualified personnel who are familiar with the characteristics, response patterns, and behaviors which are typical of individuals with ASD. In addition to understanding ASD, training should include a basic understanding of informal assessment, and best practices in ASD, including monitoring outcomes. Many types of informal assessment procedures exist, and the professional must select the measures most appropriate for the individual with ASD being assessed. Informal assessment involves daily and ongoing evaluation of skills and behavior in order to monitor treatment outcomes (Hogan and Marcus 2008). It is particularly important to incorporate interactive modifications in the environment. It is also important to work collaboratively in order to create optimal intervention programs for individuals with ASD.

See Also

- [Clinical Assessment](#)
- [Task Analysis](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Rev.). Washington, DC: Author.
- Callahan, E. H., Gillis, J. M., Romanczyk, R. G., & Mattson, R. (2011). The Behavior Assessment for Social Interactions in young children: An example of convergent and incremental validity. *Research in Autism Spectrum Disorders*, 5(2), 768–774.
- Herzinger, C., & Campbell, J. M. (2007). Comparing functional assessment methodologies: A quantitative synthesis. *Journal of Autism and Developmental Disorders*, 36, 1430–1445.
- Hogan, K., & Marcus, L. M. (2008). From assessment to intervention. In S. Goldstein, J. A. Naglieri, &

- S. Ozonoff (Eds.), *Assessment of autism spectrum disorders* (pp. 318–338). New York: Guilford Press.
- Klin, A., Saulnier, C., Tsatsamis, K., & Volkmar, F. R. (2005). Clinical evaluation in autism spectrum disorders: Psychological assessment within a transdisciplinary framework. In F. R. Volkmar, R. Paul, A. Klin, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 772–798). New York: Wiley.
- Lerman, D. C., Vorndran, C., Addison, L., & Kuhn, S. A. C. (2004). A rapid assessment of skills in young children with autism. *Journal of Applied Behavior Analysis*, 37(1), 11–26.
- Levy, S. E., Mandell, D. S., & Schultz, R. T. (2009). Autism. *The Lancet*, 374(9701), 1627–1638.
- Lord, C., Wagner, A., Rogers, S., Szatmari, P., Aman, M., Charman, T., et al. (2005). Challenges in evaluating psychosocial interventions for autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 35(6), 695–708.
- Magiati, I., & Howlin, P. (2001). Monitoring the progress of preschool children with autism enrolled in early intervention programmes. *Autism*, 5(4), 399–406.
- National Research Council. (2001). *Educating children with autism*. Committee on educational interventions for children with autism. (Division of behavioral and social sciences and education). Washington, DC: National Academy Press. www.nap.edu
- Spears, R., Tollefson, N., & Simpson, R. (2001). Usefulness of different types of assessment data in diagnosing and planning for a student with high functioning autism. *Behavioral Disorders*, 26(3), 227–242.
- Volkmar, F. R., Lord, C., Bailey, A., Schultz, R. T., & Klin, A. (2005). Autism and pervasive developmental disorders. *Journal of Child Psychology and Psychiatry*, 45, 1(1), 1–1), 36.

Information Processing Speed

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Synonyms

Inspection time; Reaction time

Definition

Information processing speed represents an underlying efficiency of perception, reasoning, and problem solving. This efficiency can dampen or enhance an individual's innate intellectual capacities. Processing speed tasks are often included in tests of intelligence and comprise their own index (e.g., Processing Speed Index in the Wechsler scales), although impaired motor skills can impact cognitive processing in these tasks. However, independent inspection time (IT) tasks are generally considered the purest assessments of information processing speed. IT is the amount of time a person needs to be exposed to a stimulus in order to make a simple perceptual judgment (e.g., which line is longer?).

Recent research using the IT task, which is free of motor demands, reveals superior processing speed among lower functioning children with autism spectrum disorder (ASD) and intact (but not superior) processing speed among higher functioning children with ASD. Across development in typically developing individuals, IT performance is consistently correlated with general intelligence. However, IT does not appear to have the same relationship with intelligence in ASD that is seen in individuals with typical development.

See Also

- Processing Speed Index
- Processing Speed Quotient
- Psychological Assessment

References and Reading

- Mayes, S. D., & Calhoun, S. L. (2008). WISC-IV and WIAT-II profiles in children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 38, 428–439.
- Scheuffgen, K., Happé, F., Anderson, M., & Frith, U. (2000). High "intelligence," low "IQ"? Speed of processing and measured IQ in children with autism. *Development and Psychopathology*, 12, 83–90.
- Wallace, G. L., Anderson, M., & Happé, F. (2009). Brief report: Information processing speed is intact in autism but not correlated with measured intelligence. *Journal of Autism and Developmental Disorders*, 39, 809–814.

Informed Consent

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Synonyms

[Consent](#)

Definition

Permission obtained from a patient/participant to have a specific test or procedure performed or to participate in a clinical study. Informed consent is a legal procedure to ensure that a patient/participant knows all of the risks or costs involved in treatment. The elements of informed consent include the nature of the treatment, possible alternative treatments, possible benefits of treatment, and any risks, discomforts, or limitations of confidentiality. This document must be written in language understood by the patient and must be signed and dated by the participant and at least one witness. Patients/participants also must be made aware of their privacy, safety, and freedom to withdraw, as well as any compensation for their participation. In order for informed consent to be considered legal, the patient/participant must be competent and their consent should be given voluntarily.

See Also

- [Consent](#)
- [Ethics](#)

References and Reading

Faden, R., Beauchamp, T., & King, N. (1986). *A history and theory of informed consent*. New York: Oxford University Press.

Inherited Metabolic Diseases

- [Inborn Errors of Metabolism](#)

Initial Communication Processes Scale

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Definition

The Initial Communication Processes Scale is an observational assessment instrument that assesses preverbal and verbal communication of young children (0–36 months). Domains addressed by the Initial Communication Processes Scale include auditory, visual, manual fine motor, oral vocal motor, object play manipulation, object play symbolic, problem solving, affective development, communication skills, and comprehension skills. It can be used to help identify the presence of autism-related communication deficits in children under 3 years old. Although developed in the early 1980s, applications of the Initial Communication Processes Scale have appeared rarely in the literature.

See Also

- [Clinical Linguistic and Auditory Milestone Scale](#)
- [Communication Assessment](#)
- [Communicative Development Inventories](#)
- [Early Language Milestone Scale](#)
- [MacArthur-Bates Communicative Development Inventories, Second Edition](#)

References and Reading

O'Connor, L., & Schery, T. K. (1986). A comparison of microcomputer-aided and traditional language therapy for developing communication skills in nonoral toddlers. *Journal of Speech and Hearing Disorders*, 51, 356–361.

- Paul, R. (2008). Communication development and assessment. In K. Chawarska, A. Klin, & F. R. Volkmar (Eds.), *Autism spectrum disorders in infants and toddlers: Diagnosis, assessment, and treatment* (pp. 76–103). New York: The Guilford Press.
- Schery, T., & Wilcoxon, A. (1982). *The initial communication processes scale*. Monterey: CTB/McGraw-Hill.

experience less significant impairments in IBR. The primary reason for this is that IBR is an instrumental form of social attention coordination, as opposed to IJA, which is used exclusively for social purposes.

See Also

- [RJA/IJA \(Initiating/Responding to Joint Attention\)](#)

Initiating Behavior Regulation

- [Initiating Behavioral Requests \(IBR\)](#)

Initiating Behavioral Requests (IBR)

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Synonyms

[Initiating behavior regulation](#)

Definition

Initiating behavioral requests (IBR) refers to a child's use of eye contact, reaching, giving, or pointing to coordinate with another person to request help in obtaining an object or event. IBR is contrasted with responding to behavioral requests (RBR), which is a child's skill in responding to gestural or verbal directions to obtain an object or event. Along with joint attention behaviors and social interaction behaviors, behavioral requests (initiating and responding) are included in the Early Social Communication Scales (ESCS). The ESCS measures nonverbal communication skills of children between 8 and 30 months of age and is used in research and practice. Many young children with autism display deficits in initiating joint attention (IJA) but

References and Reading

- Mundy, P. (2003). Annotation: The neural basis of social impairments in autism: The role of the dorsal medial-frontal cortex and anterior cingulate system. *Journal of Child Psychology and Psychiatry*, 44, 793–809.
- Mundy, P., & Gomes, A. (1998). Individual differences in joint attention skill development in the second year. *Infant Behavior and Development*, 21(3), 469–482.
- Mundy, P., Sigman, M., Ungerer, J., & Sherman, T. (1986). Defining the social deficits of autism: The contribution of non-verbal communication measures. *Journal of Child Psychology and Psychiatry*, 27, 657–669.
- Mundy, P., Delgado, C., Hogan, A., & Doehring, P. (2003). *A manual for the abridged early social communication scales (ESCS)*. Coral Gables: University of Miami.

Initiating/Responding to Joint Attention Skills (IJA/RJA)

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Synonyms

[IJA/RJA skills](#)

Initiating/Responding to Joint Attention Skills (IJA/RJA), Table 1 Behavior regulation and joint attention gestures^a

General category	Specific skill	Definition	Example
Behavior regulation	Pointing	Child points to an object, person, or event that he or she wants or needs	Child points at a toy on the shelf to indicate that he or she wants to play with it
	Giving	Child gives an object to request help	Child gives a sealed bag of candy to his or her mom to communicate that he or she wants it open
Initiating joint attention	Pointing	Child points at an object or event to share his or her interest	Child points at a dog to share his or her interest about the dog
	Alternating eye contact	Child alternates eye gaze between an object or event and a person to share his or her interest	Child looks back and forth between a dog and his or her mom to share interest about the dog
	Showing	Child holds up an object to share his or her interest	Child holds up a drawing to share with his or her teacher
Responding to joint attention	Pointing	An adult points to something to share his or her interest. The child looks in the direction of the point	An adult points to a neat poster on the wall, and the child looks at the poster

^aThese skills were selected based upon Early Social Communication Scales Manual (Mundy et al. 2003)

Definition

As explained in the section titled “joint attention,” children with autism exhibit a core deficit in joint attention. Joint attention initiation refers to communication used to share interest regarding an object, person, or event with someone else (Mundy et al. 1986). Most commonly, joint attention is initiated by young children through the nonverbal gestures of pointing, showing, giving, and coordinated looking. Responding to joint attention requires that a social partner visibly acknowledges the joint attention initiation of their communication partner. Table 1 provides definitions of specific types of initiating and responding joint attention gestures as well as a few examples of these gestures.

Mundy, P., Delgado, C., Block, J., Venezia, M., Hogan, A., & Seibert, J. (2003). *A manual for the abridged early social communication scales (ESCS)*. University of Miami, Unpublished.

Initiation of Communication

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Synonyms

[Initiation of joint attention](#); [Intentional communication](#); [Social initiation](#)

See Also

► [Joint Attention](#)

References and Reading

Mundy, P., Sigman, M., Ungerer, J., & Sherman, T. (1986). Defining the social deficits of autism: The contribution of non-verbal communication measures. *Journal of Child Psychiatry*, 27, 657–669.

Definition

The initiation of communication refers to the nonprompted and purposeful (i.e., intentional) sending of a message from one conversational partner to another within the context of a communicative exchange. The form of the message may be nonverbal (consisting of some combination of eye gaze, gestures, and/or vocalizations)

or verbal (consisting of single words or word combinations). In general, the individual who initiates the communication act would be considered the speaker and the individual to whom the communication act is directed would be considered the listener. The initiation of communication can serve the pragmatic function of requesting when the speaker is attempting to elicit assistance in obtaining some instrumental goal. Alternatively, the initiation of communication can serve the pragmatic function of joint attention when the speaker is attempting to direct the attention of the listener to an object, action, or event of shared interest. In contrast to a prompted response, initiations require individuals to independently formulate and produce their own communicative behavior without the assistance or contextual support of a communication partner.

See Also

► [Joint Attention](#)

References and Reading

- Bakeman, R., & Adamson, L. B. (1984). Coordinating attention to people and objects in mother-infant and peer-infant interaction. *Child Development*, 55, 1278–1289.
- Koegel, R., & Koegel, L. (2006). *Pivotal response treatment for autism: Communication, social and academic development*. Baltimore: Brookes.
- Landry, S. H., Smith, K. E., Miller-Loncar, C. L., & Swank, P. R. (1997). Predicting cognitive-language and social growth curves from early maternal behaviors in children at varying degrees of biological risk. *Developmental Psychology*, 33, 1040–1053.
- Stone, W. L., & Caro-Martinez, L. M. (1990). Naturalistic observations of spontaneous communication in autistic children. *Journal of Autism and Developmental Disorders*, 20, 437–453.

Injuries in Children with ASD

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Synonyms

[Trauma](#); [Wounds](#)

Definition

Injury may be defined as physical damage to the body caused by sudden or brief exposure to energy at levels that exceed what the body can tolerate (Peden et al. 2008). The energy that most commonly causes injury is mechanical, for example, impact with a moving or stationary object as in a road traffic crash. Other types of energy that cause injury include thermal (e.g., from a flame or hot water), chemical (e.g., from a poison), and electrical energy. Most injuries to children are unintentional, that is, not deliberately caused, for instance, falls, burns, and drowning. Intentional injuries resulting from acts of violence to oneself or by another person, such as assault or suicide, also occur in children, especially during adolescence (Pinheiro 2006). Injuries are a major cause of emergency treatment, hospitalization, death, and disability in children worldwide (Global Burden of Disease Pediatrics Collaboration 2016). Annually, tens of millions of children are injured seriously enough to require medical attention, and more than half a million children die of their injuries.

Compared to adults, children are at greater risk for some types of injuries because their physical and cognitive abilities are still developing and they may fail to perceive or respond to danger (Schwebel and Gaines 2007). The risk of injury is strongly influenced by characteristics of the child, including age, sex, disability, and psychological and behavioral characteristics such as activity level and cognitive function (Schwebel

Initiation of Joint Attention

► [Initiation of Communication](#)

and Gaines 2007). The environment in which the child lives also influences injury risk. This includes both the physical environment, whether natural or man-made, i.e., home, school, roads, and surrounding community, and the social environment, e.g., maternal education, caregiver supervision practices, family relationships, household income, neighborhood cohesion, peer and cultural norms, economic and health policies, and laws.

Several risk factors for injury occur at higher rates in children with autism spectrum disorder (ASD) compared to the general population, including male sex and psychological and behavioral problems such as aggressive behavior, anxiety, attention deficits, cognitive delays, hyperactivity, and sensory deficits (Lyall et al. 2017). While these characteristics could place children with ASD at higher risk for injury, research studies examining injury risk have reported conflicting findings ranging from significantly increased risk to little or no increased risk associated with ASD. The differing results likely reflect methodological variation between studies, including the populations sampled, the definitions used for injury, the data collected, the periods of risk, and the specific sociodemographic, clinical, and behavioral characteristics included as covariates in analytic models. Whether children with ASD are more likely than are other children to suffer injury has not been definitively established.

A wide range of effective strategies exists to prevent, control, and ameliorate the consequences of child injuries (Peden et al. 2008; Pinheiro 2006; Schwebel and Gaines 2007). Legislation and enforcement, such as setting and enforcing laws mandating passenger restraint use, have been shown to prevent child injuries. Product and environmental modification is another key prevention strategy, for example, isolation fencing for swimming pools and child-resistant packaging of medications and poisons. Safety devices, including car safety seats, smoke detectors, bicycle helmets, and window guards, are effective when used correctly in reducing injury occurrence or severity. High-quality supportive home visits have short- and long-term

preventive effects on injury and violence to children, including reductions in verified incidents of child abuse, use of physical punishment, family violence, and physician visits for unintentional injuries and poisonings. Reducing access to lethal means, such as guns, poisons, and certain medications, can decrease suicide risk among older children and adolescents. Timely and effective emergency care (e.g., caregiver training in cardiopulmonary resuscitation and prompt response times for emergency medical services), trauma care, and rehabilitation services can lessen adverse outcomes of injuries, such as disability or death.

See Also

- [Self-Injurious Behavior \(SIB\)](#)
- [Suicidality in Children and Adolescents with Autism](#)
- [Suicide Rates in Adults with Autism](#)
- [Violence and ASD](#)

References and Reading

- Global Burden of Disease Pediatrics Collaboration. (2016). Global and national burden of diseases and injuries among children and adolescents between 1990 and 2013: Findings from the Global Burden of Disease 2013 Study. *JAMA Pediatrics*, 170(3), 267–287.
- Lyall, K., Croen, L., Daniels, J., Fallin, M. D., Ladd-Acosta, C., Lee, B. K., Park, B. Y., Snyder, N. W., Schendel, D., Volk, H., Windham, G. C., & Newschaffer, C. (2017). The changing epidemiology of autism spectrum disorders. *Annual Review of Public Health*, 38, 81–102.
- Peden, M., Oyegbite, K., Ozanne-Smith, J., Hyder, A. A., Branche, C., Rahman, A. K. M. F., Rivara, F., & Bartolomeos, K. (Eds.). (2008). *World report on child injury prevention*. Geneva: World Health Organization. www.who.int/violence_injury_prevention/child_injury/world_report/en/. Accessed 20 Aug 2019.
- Pinheiro, P. S. (2006). *World report on violence against children*. Geneva: United Nations Secretary-General's Study on Violence Against Children. <https://www.unicef.org/violencestudy/reports.html>. Accessed 20 Aug 2019.
- Schwebel, D. C., & Gaines, J. (2007). Pediatric unintentional injury: Behavioral risk factors and implications for prevention. *Journal of Developmental and Behavioral Pediatrics*, 28(3), 245–254.

Innate Reflexes

► Primitive Reflexes

Inner Ear

► Cochlea

Inositol: Definition

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Synonyms

Cyclohexitol; Myo-inositol; Vitamin B8

Definition

Inositol is an abundant sugar responsible for important cellular transduction pathways involving hormones, neurotransmitters, and growth factors (Parthasarathy et al.). Among the nine inositol isomers, the myo-inositol has the greatest biological activity and serves to synthesize cell membrane inositol phospholipids (Parthasarathy et al.). In the brain, myo-inositol links several extracellular signaling pathways activating phospholipase C and increasing cytosolic calcium from the endoplasmic reticulum (Parthasarathy et al.). The resulting calcium surge activates many enzymes and receptors controlling essential physiological and neurological functions (Parthasarathy et al.).

As a result, the inositol pathway remains an attractive pharmacological target towards the treatment and management of many psychiatric disorders. Several clinical studies have demonstrated a correlation between decreased myo-inositol levels and the severity of several

psychiatric conditions, including depression, obsessive-compulsive disorder (OCD), and bipolar disorder (Einat and Belmaker 2001; Levine 1997; Parthasarathy et al.). Furthermore, studies supplementing myo-inositol for depression, panic disorders, and OCD patients showed improved symptom severity and management (Levine 1997). Additional pharmacological advancements using inositol synthase inhibitors, valproate and lithium, showed decreased mood fluctuations among bipolar patients (Levine 1997; Parthasarathy et al.). However, similar outcomes among autistic spectrum disorder (ASD) patients remain elusive.

A double-blind trial of myo-inositol showed no significant improvement in nine children with ASD (Levine et al. 1997). Previous scientific studies reported serotonin binding to 5-HT₂, and 5-HT_{1c} receptors activates inositol signaling, which the authors hypothesized should mimic the clinical improvements seen with SSRIs and ASD patients (Levine 1997; Parthasarathy et al.). Despite the similarities, no additional studies have investigated the therapeutic potential of myo-inositol among ASD patients. However, a proton magnetic resonance spectroscopy (H¹ MRS) analysis found myo-inositol and choline were significantly increased in the anterior cingulate and left striatum of autistic children (Vasconcelos et al. 2008). As such, myo-inositol may provide additional diagnostic and radiologic markers for ASD and other psychiatric conditions. In addition, a genome-wide linkage analysis revealed the Inositol-3-phosphate synthase 1 (ISYNA1) gene may act as a potential autism susceptibility locus (Parthasarathy et al.). Current literature shows Inositol-3-phosphate synthase 1 modulates inositol signaling, which may alter neurological symptoms among ASD patients (Parthasarathy et al.). Therefore, further investigation is warranted to elucidate the full therapeutic and diagnostic potential of inositol among ASD patients.

See Also

► Autonomic Nervous System

References and Reading

- Einat, H., & Belmaker, R. H. (2001). The effects of inositol treatment in animal models of psychiatric disorders. *Journal of Affective Disorders*, 62(1–2), 113–121. [https://doi.org/10.1016/s0165-0327\(00\)00355-4](https://doi.org/10.1016/s0165-0327(00)00355-4).
- Levine, J. (1997). Controlled trials of inositol in psychiatry. *Biological Psychiatry*, 42(1), 289S. [https://doi.org/10.1016/s0006-3223\(97\)88102-4](https://doi.org/10.1016/s0006-3223(97)88102-4).
- Levine, J., Aviram, A., Holan, A., Ring, A., Barak, Y., & Belmaker, R. H. (1997). Inositol treatment of autism. *Journal of Neural Transmission*, 104(2–3), 307–310. <https://doi.org/10.1007/bf01273191>.
- Parthasarathy, L. K., Ratnam, L., Seelan, S., Tobias, C., Casanova, M. F., & Parthasarathy, R. N. Mammalian inositol 3-phosphate synthase: Its role in the biosynthesis of brain inositol and its clinical use as a psychoactive agent. In *Subcellular biochemistry* (pp. 293–314). Springer.
- Vasconcelos, M. M., Brito, A. R., Domingues, R. C., da Cruz, L. C. H., Gasparetto, E. L., Werner, J., & Gonçalves, J. P. S. (2008). Proton magnetic resonance spectroscopy in school-aged autistic children. *Journal of Neuroimaging*, 18(3), 288–295. <https://doi.org/10.1111/j.1552-6569.2007.00200.x>.

Inspection Time

- [Information Processing Speed](#)

“Instruction, Modelling, Rehearsal, and Feedback,” BST

- [Behavioral Skills Training](#)

Instructional Assistant

- [Para-educator](#)
- [Paraprofessional](#)

Instructional Objectives

- [Objective](#)

Instrumental Conditioning

- [Operant Conditioning](#)

Instrumental Learning

- [Operant Conditioning](#)

Integrated Play Groups (IPG) Model

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Definition

The integrated play groups (IPG) model is an intervention designed to facilitate the development of meaningful peer relationships and appropriate play and social-communication skills in children with autism spectrum disorders (ASD) through adult-supported peer interactions. Play groups consist of “novice players” (those with ASD) and “expert players” (typically developing peers). A trained adult facilitator acts as a “play guide” to support and shape interactions between the novice and expert players.

Historical Background

The IPG model was developed by Dr. Pamela J. Wolfberg in an effort to create a model of intervention “that would bring together children with differing abilities, cultural identities, and social backgrounds for the purpose of play” (Wolfberg 1999, p. 6). Wolfberg received a small grant from a community educational foundation to fund an initial pilot study of the IPG program, and this research later became the subject of her master’s research (Wolfberg 1988). Then, along with Dr. Adriana Schuler, Wolfberg (1992)

codirected a research and demonstration project to further develop and field-test the intervention model. Since then, several small-scale efficacy studies have been conducted with positive outcomes (e.g., Lantz et al. 2004; Yang et al. 2003; Zercher et al. 2001), and in 2008 Autism Speaks awarded Wolfberg a \$444,420 grant to conduct a large-scale study of the IPG model. The traditional IPG model has recently been extended to incorporate sensory integration therapy for children who have been identified as having both social-communication and sensory integration deficits (Fuge and Berry 2004), and has paired with the Friend 2 Friend model to provide collaborative services. A final extension of the traditional IPG model is the Integrated Drama Groups (IDG) model. This intervention was developed to support the social communication of adolescents with ASD using a developmentally appropriate model, which includes opportunities for turn taking and role playing within the context of sociodramatic play (www.autismsocialconnection.org). In 2009, Julius, Schuler, and Wolfberg received the Alexander von Humboldt Foundation Transcoop Research Award with matching funds from the Flora Foundation and the Mendelson Family Foundation to evaluate the efficacy of the IDG model (Wolfberg 2010).

Rationale or Underlying Theory

The IPG model draws heavily from the social constructivist work of Lev Vygotsky, who proposed that it is through play that children acquire “symbolic capacities, interpersonal skills, and social knowledge” (Schuler and Wolfberg 2000, p. 181). Current research indicates that play does indeed serve a fundamental role in social competence, language and literacy development, creative expression, and emotional development in children (for a review, see Wolfberg (1999)). Vygotsky further suggests that a child’s learning and acquisition of skills can be facilitated through scaffolding by an adult or more competent peer within the child’s zone of proximal development, which is the difference between what the child is able to do independently and

what they are able to achieve with assistance (Wolfberg 2003). This provides the theoretical basis for the role of the adult play guide within the IPG model.

Wolfberg (2003) also emphasizes that play is fundamental to a child’s sense of belonging within their peer group, as it is the most valued social activity in childhood. Play contributes to a sense of collective identity and enables children to create a world that is separate and unique from the adult world (Wolfberg and Schuler 1999). Therefore, while play intervention could potentially be provided by an adult therapist alone, typical peers provide the novice player with access to “play culture” in a way that an adult is unable to (Wolfberg and Schuler 1999).

Goals and Objectives

Two of the hallmark characteristics of children with ASD described by *The Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition* (DSM-IV) are a lack of varied, spontaneous make-believe play and a failure to develop appropriate peer relationships (American Psychiatric Association 2000). The primary objective of the IPG model is to facilitate children with ASD in developing mutually enjoyable, appropriate peer relationships, while also increasing their play repertoire to include symbolic play (Wolfberg 1999). Symbolic or pretend play can range from the substitution of one object for another (e.g., pretending a banana is a telephone) to engagement in complex sociodramatic play. Because children with ASD are typically inclined to fixate on certain objects or activities for extended periods of time and limit their involvement with nearby peers, scaffolding and play guidance serve as important supports to guide children through more varied and complex play routines than they would seek out on their own (Wolfberg 2004). Additionally, play materials that have a high potential for sociodramatic and imaginative play are made readily available in the play environment to promote engagement in symbolic play activities (Wolfberg 2003).

Treatment Participants

The IPG model was designed specifically for children with ASD and related disorders ranging from age 3 to 11 years. Wolfberg (2003) suggests that, as play never truly ends, adaptations to the IPG model might be made to support children who are either younger or older than the designated age range. Unfortunately, no efficacy studies have been conducted for ages outside of the recommended age range. It is also recommended that children already have an educational and therapy plan in place before considering participation in an IPG intervention (Wolfberg 2003). This is important because the IPG model was developed as a supplement to more traditional language, social-emotional, and behavioral intervention models.

Participants may be highly verbal to functionally nonverbal and may use augmentative and alternative communication strategies. The IPG model is able to support a wide range of communicative abilities because of its fluid nature. Play groups are designed with the specific child's needs and capabilities in mind. Therefore, a play group designed for a child with strong expressive and receptive language abilities will look very different from one that is designed for a functionally nonverbal child. In addition, play guides and expert players are trained to look at a wide range of behaviors, beyond the obvious gestures of verbal language, as possible attempts at play initiation (Wolfberg 2003). Additionally, visual supports are an integral part of the intervention model, making this model ideal for children already using PECS or for children who simply benefit from visual supports, as many individuals with ASD do.

The IPG model is also designed to support children at all points on the autism spectrum, from those who have intensive forms of autism to those diagnosed with Asperger syndrome. Once again, this is due to the fluid nature of the IPG model, as play activities are tailored to fit the individual child's interests and capabilities. However, it should be noted that the IPG model is not recommended for children whose behavior might be considered self-injurious or pose a threat to

others despite the presence of an adult facilitator (Wolfberg 2003).

Although most children with ASD would likely benefit from the addition of an IPG intervention to their current educational and therapy plan, an ideal candidate would exhibit some impairments in the skills that are necessary for successful play interactions. Some of these deficits might include atypical verbal and nonverbal attempts at social initiation, lack of eye contact, general failure to seek out peer relationships, lack of social or emotional reciprocity, inability to maintain conversation, lack of variation in play, lack of symbolic play, and difficulties taking the perspectives of others (Wolfberg 2003).

Treatment Procedures

The basic setup of the IPG model involves a play group that consists of children with autism (*novice players*), typical peers (*expert players*), and an adult facilitator (*play guide*). The groups are generally made up of 3–5 children, with a higher ratio of expert to novice players. The expert players in the group are recruited from environments in which the novice player would typically come into contact with peers such as school, home, or the surrounding community. It is recommended that the play group meet twice a week for 30–60 min sessions over the course of 6–12 months in an environment where the group members would naturally play, such as a classroom, the home, or community recreation areas (Wolfberg 2003). Play areas are carefully designed to provide a sense of organization and predictability and are stocked with a variety of play materials designed to accommodate the diverse interests and abilities of group members.

Each play session incorporates an opening and closing ritual designed to clearly mark the beginning and end of the session and to foster a sense of group identity. Opening rituals generally consist of a greeting, followed by a review of the play group rules and a brief discussion of the previous play session. These discussions allow children time to consider what was fun or challenging during the previous session to make future

sessions more successful (Wolfberg 2003). The closing ritual includes clean-up of play materials followed by a review of the play session, planning for the next meeting, and a goodbye ceremony that includes a special group song or handshake.

Following the opening ceremony, specific strategies that the children will use during play are reviewed. These strategies are a series of verbal and nonverbal social-communication cues designed to assist both novice and expert players in interacting with one another. For example, a social-communication cue for initiating play might be to stand close, look at your playmates, point to the game, and ask, “what are you doing?” or say, “let’s play” (Wolfberg 2003, p. 190). After this review, the players come up with a simple plan for getting play started. The group might come up with specific themes for play or choose what materials they would like to use. This technique is used only to get play started. As play is naturally fluid, children may change or abandon initial play ideas at any time (Wolfberg 2003).

During the session, the adult play guide facilitates interactions between players by applying four key practices: monitoring play initiations, scaffolding play, providing social-communication guidance, and providing play guidance (Wolfberg 2003). Monitoring play initiations refers to the play guide’s responsibility to recognize, interpret, and respond to a novice player’s attempts to initiate play with peers. If the player’s attempts at initiation are unconventional, the play guide will interpret them for other players and assist the child in learning new, more appropriate means of initiation. Throughout the session, the play guide provides varying amounts of support to players by scaffolding play interactions. This requires that the play guide anticipate times at which players might need support to keep a play interaction going, but also to be intuitive of when little to no support is necessary. Ways in which the play guide may scaffold interactions include providing explicit direction or modeling play behaviors, offering suggestions or commenting on play, or simply standing nearby the group.

In addition to scaffolding play, the play guide facilitates play interactions by providing social-communication and play guidance. In providing

social-communication guidance, the play guide supports interactions by cueing players to use verbal and nonverbal strategies that facilitate players’ ability to initiate, respond to, join, or maintain play activities (Wolfberg 2003). Social-communication cues provide players with suggestions of what they might do or say during play interactions. For example, in order to initiate play, a child might be cued to tap their peer on the shoulder and say “let’s play” (Wolfberg 2003). Ultimately, the goal is for children to incorporate these strategies into their interactions without assistance from the play guide. Play guidance refers to a hierarchy of techniques designed to involve the novice player in interactions at a level that is just outside of their current skill set (Wolfberg 2003). Types of play guidance range from orienting, in which the novice player is prompted to observe other players, to role playing, in which novice and expert players engage in elaborate pretend play scenarios. In using this series of supports, the goal is that the children will gradually learn how to play together with decreasing amounts of adult support.

Efficacy Information

Several studies evaluating the efficacy of the IPG model describe positive outcomes for children with ASD, including increased social interaction and reciprocal play with peers, increased in the complexity of play (e.g., functional and symbolic play), and decreased in stereotyped and isolated play (Lantz et al. 2004; Richard and Goupil 2005; Wolfberg 1988; Wolfberg and Schuler 1993; Yang et al. 2003; Zercher et al. 2001). Additionally, Wolfberg and Schuler (Wolfberg and Schuler 1993) noted significant gains in language for two out of three participants with ASD, including an increase in the variety of linguistic forms and communicative functions of utterances. Similarly, Zercher et al. (2001) found that all three participants with ASD demonstrated an increase in the number of verbal utterances over the course of treatment. Studies also indicate that skills observed during intervention were maintained when adult support was withdrawn (Lantz et al. 2004; Richard

and Goupil 2005; Wolfberg and Schuler 1993; Yang et al. 2003; Zercher et al. 2001).

Wolfberg and Schuler (1992) conducted a large-scale study investigating teachers' and speech-language pathologists' (SLP) qualitative perceptions of the impact of participation in an IPG on novice players ($N = 38$). Teachers and SLPs reported increases in social reciprocity between novice and expert players and indicated that the IPG provided more opportunities for relationship development than participation in an inclusive setting alone provided. They also indicated that children interacted more naturally in the IPG setting than in other inclusive settings and described the play groups as a "safe haven" for children to play without added outside pressure.

Preliminary evidence exists to support the generalization of skills to settings outside the play group. For example, in a single-subject study Lantz et al. (2004) reported that increases in socially oriented play behavior (e.g., turn taking, sharing, offering or accepting assistance) and common focus play (play characterized by joint attention and social reciprocity) observed during play group were generalized to the classroom. Wolfberg and Schuler (1993) provide qualitative evidence for generalization gained through semi-structured parent and teacher interviews. Results of these interviews indicate that participants with ASD generalized skills gained during intervention to the classroom and home setting.

A major limitation of the aforementioned studies is the small number of participants. Studies providing quantitative measures have ranged from between one and four novice participants. The limited number of participants in these studies limits the generalizability of results. Despite this, evidence to support the utility of the IPG model is promising.

Outcome Measurement

The IPG manual (Wolfberg 2003) includes a series of assessments that are intended to be used throughout the course of intervention:

- *Play Questionnaire* – Completed by individuals who observe the child's play behaviors on a

regular basis (e.g., parents, therapists, teachers) prior to and at the end of intervention. This questionnaire provides information regarding the play experience, preferences, and style of the novice player, along with information regarding their play patterns within a developmental framework. A comparison of pre- and postintervention questionnaires can provide information regarding the child's progress.

- *Play Preference Inventory* – Completed by the play guide prior to the beginning of intervention and periodically over the course of intervention. This inventory is used to record the play preferences of the novice (what they spontaneously choose to do during free play) and how these preferences might connect with the interests of novices players. For example, the play guide might connect a novice player's interest in hair to expert players interests by coming up with an activity where the group pretends they are hairdressers (Wolfberg 2003). A list of ways the novice player's preference can be connected to those of the expert players may be generated during a brainstorming session involving all group members. This inventory assists the play guide in planning activities that will be engaging and enjoyable for all group members and also provides information regarding the novice player's diversity of play, which is later recorded in the *Profile of Individual Play Development*.
- *Integrated Play Groups Observation* – Completed on a weekly basis by the play guide during the first month of intervention and approximately every other week thereafter. This assessment tool provides the play guide with a structured way to complete naturalistic observations of the novice player over the course of intervention.
- *Profile of Individual Play Development* – Completed by the play guide once a month during intervention. This assessment combines information gathered in the aforementioned assessments to provide a complete profile of the novice player for progress-tracking purposes.
- *Record of Monthly Progress in IPG* – Completed by the play guide on a monthly basis during intervention. This assessment tool is

used to chart the novice player's progress in regard to a specific set of individualized goals determined prior to the start of intervention.

Qualifications of Treatment Providers

Training is required to implement an IPG and become a play guide. Individuals who complete this training are typically those who have some experience caring for or working with children with ASD (Wolfberg 2003). To apply IPG principles in an inclusive setting, you must attend a 2-day introductory and intermediate-level training course. The first day of training provides a basic overview of the theory and methods applied during intervention, while the second day concentrates on designing an IPG model for specific children and developing a plan for implementation (Wolfberg 2010). To be qualified to implement the IPG model as a program or service and be qualified as a "master play guide," completion of an apprenticeship program is required. Supervision during the apprenticeship program may be direct or long distance (involving telephone and email conferencing along with exchange of videotapes) depending on the location of the individual (Wolfberg 2010). Certification from the Autism Institute on Peer Relations and Play is provided upon successful completion of this portion of the training.

See Also

- Parallel Play
- Peer-Mediated Intervention
- Peers, Exposure to
- Play
- Social Communication
- Social Skill Interventions
- Symbolic Play

References and Reading

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: APA Press. Text Rev.

- Fuge, G., & Berry, R. (2004). *Pathways to play! Combining sensory integration and integrated play groups: Theme-based activities for children with autism spectrum and other sensory processing disorders*. Shawnee Mission: Autism Asperger Publishing.
- Lantz, J. F., Nelson, J. M., & Loftin, R. L. (2004). Guiding children with autism in play: Applying the integrated playgroup model in school settings. *Teaching Exceptional Children*, 37(2), 8–15.
- Richard, V., & Goupil, G. (2005). Application des groupes de jeux integres aupres d'eleves ayant un trouble envahissant du developement (Implementation of Integrated Play Groups with autism/PDD students). *Revue Quebecoise de Psychologie*, 26(3), 79–103.
- Schuler, A. L., & Wolfberg, P. J. (1999). Fostering peer interaction, imaginative play and spontaneous language in children with autism. *Child Language Teaching and Therapy Journal*, 15(1), 41–52.
- Schuler, A. L., & Wolfberg, P. J. (2000). Promoting peer play and socialization: The art of scaffolding. In A. M. Wetherby & B. M. Prizant (Eds.), *Autism spectrum disorders: A transactional developmental perspective* (pp. 251–277). Baltimore: Paul H. Brookes Publishing.
- Wolfberg, P. J. (1988). *Integrated Play Groups for children with autism and related disorders*. Unpublished master's thesis, San Francisco State University.
- Wolfberg, P. J. (1999). *Play and imagination in children with autism*. New York: Teachers College Press.
- Wolfberg, P. J. (2003). *Peer play and the autism spectrum: The art of guiding children's socialization and imagination*. Shawnee Mission: Autism Asperger Publishing.
- Wolfberg, P. J. (2004). Guiding children on the autism spectrum in peer play: Translating theory and research into effective and meaningful practice. *The Journal of Developmental and Learning Disorders*, 8, 7–23.
- Wolfberg, P. J. (2010). *Autism institute on peer socialization and play integrated play groups training, research and development*. Retrieved on October 10, 2010, from <http://www.autisminstitute.com>
- Wolfberg, P. J., & Schuler, A. L. (1992). *Integrated play groups: Final report* (Contract #HO86D90016). Washington, DC: United States Department of Education, Office of Special Education Division.
- Wolfberg, P. J., & Schuler, A. L. (1993). Integrated play groups: A model for promoting the social and cognitive dimensions of play in children with autism. *Journal of Autism and Developmental Disorders*, 23(3), 467–489.
- Wolfberg, P. J., & Schuler, A. L. (1999). Fostering peer interaction, imaginative play and spontaneous language in children with autism. *Child Language Teaching & Therapy*, 15, 41–52.
- Yang, T., Wolfberg, P. J., Wu, S., & Hwu, P. (2003). Supporting children on the autism spectrum in peer play at home and school: Piloting the integrated play groups model in Taiwan. *Autism*, 7(4), 437–453.
- Zercher, C., Hunt, P., Schuler, A., & Webster, J. (2001). Increasing joint attention, play and language through peer supported play. *Autism*, 5(4), 374–398.

Integration

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Synonyms

[Inclusion](#); [Mainstreaming](#)

Definition

Integration in special education refers to the practice of including children with disabilities with their nondisabled peers in the educational setting. Prior to the enactment of IDEA (Individuals with Disabilities Education Improvement Act) in 1975 (then known as The Education for All handicapped Children Act, P. L.94–142), many children with disabilities were segregated from their peers in the educational setting and taught in separate classrooms, separate programs, separate schools, or left at home. IDEA not only mandates a free and appropriate education (FAPE) for all children with disabilities but also includes a mandate, that to the maximum extent appropriate, students with disabilities should be educated with their non-disabled peers (20 U.S.C.1415[5][B]) in what is referred to as the least restrictive environment (LRE). Philosophically, the integration of students with disabilities reflects the effort of educators to create a sense of belonging and acceptance for all students. Educationally, the integration of students with disabilities into the regular education environment reflects the community setting the student will be expected to function in when they leave school. Many professionals continue to disagree regarding the educational benefits of an integrated setting for every student with a disability. The student with special needs right to be integrated in the educational environment with students who do not have special needs (least restrictive environment) must be balanced with the student's right

to an educational environment that is appropriate in meeting his instructional needs.

References and Reading

- Boutot, E. A., & Myles, B. S. (2011). *Autism spectrum disorders, foundations, characteristics, and effective strategies*. Upper Saddle River: Pearson Education, Inc.
- Individuals with Disabilities Education Improvement Act of 2004, Pub.L.No.108–446, 118, Stat. 2647 (2004).
- Taylor, R. L., Smiley, L. R., & Richards, S. B. (2009). *Exceptional students, preparing teachers for the 21st century*. New York: McGraw-Hill Higher Education.

Intellectual Developmental Disorder

► [Intellectual Disability](#)

Intellectual Disability

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Synonyms

[Cognitive delay](#); [Developmental delay](#); [Developmental disability \(Ontario\)](#); [Mental retardation \(former term\)](#); [Intellectual developmental disorder](#); [Intellectual impairment](#); [Learning disability \(United Kingdom\)](#)

Short Description or Definition

The definition of intellectual disability (ID) is based on three criteria: (1) significant limitations in intellectual functioning; (2) significant limitations in adaptive behavior as expressed by conceptual, social, and practical adaptive skills; and (3) age of onset before age 18 years (Schalock et al. 2010a). It is estimated that, among people

with autism spectrum disorder (ASD), the occurrence of ID ranges from 25% to 42% (Maenner 2020). Estimates vary due to a number of methodological factors, including the design of the studies, the definitions of ASD and ID, and the ascertainment methods used (LaMalfa et al. 2004; Lecavalier et al. 2011; Matson and Shoemaker 2009; Maenner 2020). ASD is the second most common co-occurring mental disorder in individuals with ID after attention-deficit/hyperactive disorder (AD/HD) (Centers for Disease Control 2010). The diagnosis of ID may be complicated by social communication and behavior deficits inherent to ASD, which may interfere with understanding of and cooperating with assessment procedures.

The authoritative definition of ID is established by the American Association on Intellectual and Developmental Disabilities (AAIDD) Terminology and Classification Committee (Schalock et al. 2010b). A comprehensive psychological assessment is needed to determine whether or not an individual has ID and to tailor a support plan to his or her needs. At a minimum, the assessment must include measures of intellectual functioning and adaptive behavior as well as a history of developmental concerns. Since a diagnosis of ID requires that limitations in intellectual and adaptive functioning have an early onset, specifically before age 18, it is differentiated from other disorders characterized by intellectual and adaptive functioning problems, such as dementia or traumatic brain injury.

Intellectual functioning refers to general mental capacity, which includes the ability to reason, plan, think, and communicate. Intellectual functioning is assessed with a standardized intelligence test, or IQ test. Significant limitations in intellectual functioning are generally considered at or around two standard deviations below the mean of an IQ test. Adaptive behavior is comprised of three skill types:

- Conceptual skills: language and literacy; money, time, and number concepts; and self-direction
- Social skills: interpersonal skills, social responsibility, self-esteem, gullibility, naïveté (i.e., wariness), social problem-solving, and

the ability to follow rules/obey laws and to avoid being victimized

- Practical skills: activities of daily living (personal care), occupational skills, health care, travel/transportation, schedules/routines, and use of the telephone

The following five assumptions are essential in applying the definition of ID and in designing appropriate supports for individuals affected by intellectual disability:

Assumption 1: Limitations in present functioning must be considered within the context of community environments typical of the individual's age peers and culture.

Assumption 2: Valid assessment considers cultural and linguistic diversity, as well as differences in communication, sensory, motor, and behavioral factors.

Assumption 3: Within an individual, limitations often coexist with strengths.

Assumption 4: An important purpose of describing limitations is to develop a profile of needed supports.

Assumption 5: With appropriate personalized supports over a sustained period, the life functioning of the person with ID generally will improve. It is imperative that appropriate supports are identified and provided in order to enhance the individual's functioning, specifically within the areas of development, education, interests, and personal well-being (Schalock et al. 2010a).

Categorization

Adaptive behavior refers to appropriate maturation, learning, and social behaviors required for an individual to function independently and appropriately within one's cultural and age group. For a diagnosis of ID, significant deficits in adaptive behavior are required on a standardized measure normed on the general population, including people with and without disabilities. On these standardized measures, significant limitations in adaptive behavior is approximately two standard deviations below the mean of either (1) one of the

following three types of adaptive behavior: conceptual, social, or practical or (2) an overall score on a standardized measure of conceptual, social, and practical skills. Standardized measures of adaptive functioning are typically informed by parent or caregiver report or, on occasion, self-report. Examples of widely used measures of adaptive behavior include the Vineland Adaptive Behavior Scales (Sparrow et al. 2016), Adaptive Behavior Assessment System (Oakland and Harrison 2015), and the Behavior Assessment System for Children (Kamphaus and Reynolds 2015).

In addition to this criteria, significant deficits in intelligence must be established (at least two standard deviations below the mean) through individually administered, standardized measure of general intelligence. Examples of standardized IQ tests include the Stanford-Binet Intelligence Scales (Roid 2005), Wechsler Intelligence Scale for Children (Wechsler 1991), and Differential Abilities Scales (Elliot 2007). Clinical judgment and expertise are required in the interpretation of information regarding the assessment of adaptive behavior and intellectual functioning and in the formulation of valid diagnoses.

In addition to AAIDD, other widely used classification systems include the *International Classification of Diseases-11* (World Health Organization 2018) and the *Diagnostic and Statistical Manual, 5th Edition* (DSM-5; American Psychiatric Association [APA] 2013). These classification systems each define ID using IQ, adaptive behavior, and age of onset criteria. In addition, the *DSM-5* provides four severity subcategories based on adaptive functioning: mild, moderate, severe, and profound. Both ID and ASD are included under the “neurodevelopmental disorders” category in the *DSM-5* along with communication disorders, AD/HD, specific learning disorder, and motor disorder. The overarching theme of neurodevelopmental disorders is that they originate in early childhood.

There is evidence that the clinical presentation of individuals with ASD varies according to the level of adaptive functioning or

IQ. Level of functioning is negatively correlated with ASD core symptom domains (Buitelaar et al. 1999; Matson and Shoemaker 2009; Munson et al. 2008). In fact, it has been suggested that IQ may be the best predictor of functioning in ASD (Witwer and Lecavalier 2008).

Epidemiology

A meta-analysis of prevalence studies published internationally between 1980 and 2009 found a combined prevalence of ID in the general population to be 10.37 out of every 1000 births (Maulik et al. 2011). The estimated prevalence of ASD in the general population is 62 out of every 10,000 births (Elsabbagh et al. 2012). The determination of an ASD diagnosis may be complicated by genetic conditions that are associated with ID, social deficits, and restricted interests (e.g., Angelman syndrome, Prader-Willi syndrome, fragile X syndrome, Cornelia de Lange syndrome). Individuals with these syndromes may meet criteria for ID and ASD; approximately 12–15% of people with ASD have one of these genetic conditions (Muhle et al. 2004). Genetic testing is needed to diagnose the genetic condition; distinguishing between symptoms of these syndromes and ASD presents additional challenges for clinicians, and in some cases, a child will meet diagnostic criteria for ASD, ID, and a genetic syndrome.

Approximately 75–90% of individuals with ID function with mild impairments, and far fewer (~5%) are impacted to a severe or profound degree. Certain genetic syndromes are associated with severe or profound ID (e.g., Rett syndrome), while other genetic conditions, such as Down syndrome, may be associated with a wide range of intellectual functioning, although usually in the ID range (Jacobson et al. 2008). Males are more likely than females to be diagnosed with both mild (average male/female ratio 1.6:1) and severe (average male/female ratio 1.2:1) forms ID. However, sex ratios vary widely across studies (APA 2013).

Natural History, Prognostic Factors, and Outcomes

Etiology. The etiology of ID is composed of four categories of risk factors (biomedical, social, behavior, and educational) that interact across time, specifically across the life span of the individual and across generations from parent to child. This multifactorial, epigenetic construct replaces prior approaches that divided etiology of ID into two broad types: ID of biological origin and ID due to psychosocial disadvantage. Indeed, biomedical risk factors may be present in persons with ID of cultural-familial origin, and social, behavioral, and educational risk factors may be present in persons with ID that can be attributed to biomedical etiology. It is now estimated that up to 40% of ID are the result of a genetic aberration (Kaufman et al. 2010), though the percentage increases proportionally with severity of ID (McLaren and Bryson 1987). The recommended first-line tests of genetic causes are a chromosomal microarray and fragile X testing (Moeschler et al. 2014).

The increased prevalence of ID in males is largely associated with genetic factors; approximately 40 genes are known to cause ID, and approximately 80% of these reside on the X chromosome. Since males have only one X chromosome, they are less able to compensate for aberrations on the X chromosome associated with these genes than females (Kaufman et al. 2010). This same relationship accounts for the higher prevalence for ASD in males than females (Skuse 2000). According to Harris (1995) and Moser (2004), who studied the prevalence of specific genetic causes of ID in a population of 1,000 births, the 3 most common genetic conditions associated with ID are Down syndrome (1.7), fragile X syndrome (0.5 males, 0.2 females), and Duchenne muscular dystrophy (0.5–1.0). Each of these conditions is characterized by a wide range of physiological and psychological symptoms that manifest themselves within the individual to a varying degree. As such, it is important to note that, although these syndromes are among the leading causes of ID, not all people with these conditions have ID.

ID etiology is based on the interaction of four categories of risk factors across the life span and generations: biomedical, social, behavioral, and educational. The timing of ID etiology is divided into three critical periods: prenatal, perinatal, and postnatal. Table 1 presents these risk factors by category and time period.

Prognostic Factors

Health and Health Care. There is a high risk of health problems among people with co-occurring ASD. Examples include epilepsy, cerebral palsy, hearing and vision impairments, speech-language disorders, and behavioral health problems (Nickel 2000). The prevalence of physical health problems and poor physical health is higher in people with ID compared to people without disabilities (Havercamp and Scott 2015; Young-Southward et al. 2017). Individuals with ID are also susceptible to the primary risk factors of chronic diseases including obesity, decreased physical activity, and smoking. Overall physical health decreases as people with ID age from child to adulthood, and females have been shown to have poorer health than males (Young-Southward et al. 2017). As in the general population, the risk of disease among those with ID increases with age. A study that used a random sample of adults living in the community found that, compared to adults without disabilities, adults with ID were significantly more likely to report being in fair or poor health, more likely to lead sedentary lifestyles, and seven times more likely to report inadequate emotional support. Similar rates of tobacco use and overweight/obesity were reported. Adults with ID had a similar or greater risk of having four of five chronic health conditions compared with peers without disabilities (Havercamp et al. 2004). In addition to these aforementioned health issues, certain etiological conditions are associated with specific health concerns; for example, Alzheimer's disease is especially common in people with Down syndrome (Wisniewski et al. 1985).

People with ASD and ID are at increased risk of exhibiting mental and behavioral health

Intellectual Disability, Table 1 Risk factors for ID

Timing	Biomedical	Social	Behavioral	Educational
Prenatal	Chromosomal disorders Single-gene disorders Syndromes Metabolic disorders Cerebral dysgenesis Maternal illnesses Parental age	Poverty Maternal malnutrition Domestic violence Lack of access to prenatal care	Parental drug use Parental alcohol use Parental smoking Parental immaturity	Parental cognitive disability without supports Lack of preparation for parenthood
Perinatal	Prematurity Birth injury Neonatal disorders	Lack of access to prenatal care	Parental rejection of caretaking Parental abandonment of child	Lack of medical referral for intervention services at discharge
Postnatal	Traumatic brain injury Malnutrition Meningoencephalitis Seizure disorders Degenerative disorders	Impaired child-caregiver interaction Lack of adequate stimulation Family poverty Chronic illness in the family Institutionalization	Child abuse and neglect Domestic violence Inadequate safety measures Social deprivation Difficult child behaviors	Impaired parenting Delayed diagnosis Inadequate early intervention services Inadequate special education services Inadequate family support

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problems, which render many at an increased risk of being prescribed more than one medication to address physiological, psychological, and behavioral concerns (Bowring et al. 2017; Sheehan et al. 2015). Individuals with ID are vulnerable to the same adverse effects of medication use as the general population, but these effects may go unrecognized or ignored because the person’s functional limitation may mask or distort the signs, the person’s may be unable to alert caregivers to the symptoms, and because drug-induced movement disorders may be difficult to discern from preexisting stereotypical movement disorders or symptoms of psychiatric disorders or medical problems (Wilson et al. 2004). It is crucial that caregivers are aware of the possible side effects and are equipped with guidelines for monitoring these effects.

Compared to people without disabilities, people with ID face significant disparities in health care and supports. Several factors contribute to these disparities including limited communication

skills, the extra time and skill required to provide quality health care to patients with ID, and the fact that providing care to patients with disabilities is scarcely addressed in medical and other professional school curricula (Iezzoni and O’Day 2006; Kirschner and Curry 2009). A survey commissioned by Special Olympics found that only 25% of medical schools include content regarding persons with neurodevelopmental disorders in their curricula (Corbin et al. 2005). The US Surgeon General released two reports drawing attention to the importance of training health professionals to provide care for people with ID (U.S. Public Health Service 2002), noting evidence that disability confers worse health status and that resources are inadequate to maintain health, prevent secondary health conditions, and optimize wellness (U.S. Department of Health and Human Services 2005).

Mental Health. Individuals with ID are two to four times more likely to experience comorbid mental illness than the general population, with

an estimated 30–40% of people with ID suffering from a psychiatric disorder (Cooper et al. 2007; Einfeld et al. 2011). As with physical health concerns, mental illness can be hard to recognize and diagnose, especially in individuals with limited verbal ability, as the identification of many psychiatric symptoms requires the patient to introspect and self-report internal states. Symptoms of mental health problems are often mistaken for symptoms of ID; this error is referred to as “diagnostic overshadowing.” Furthermore, discomfort from chronic disorders (e.g., constipation), fluctuating pain (e.g., toothaches), or side effects of medication may present to caregivers and clinicians as disruptive or irritable behaviors (Bradley et al. 2007). Recognizing the difficulty in correctly diagnosing mental health problems in people with ID, the National Association for the Dually Diagnosed (NADD) created the *Diagnostic Manual – Intellectual Disability (DMID-2)*; Fletcher et al. 2016), which was designed to accompany *DSM-5*. The *DMID-2* was developed to facilitate an accurate psychiatric diagnosis in persons who have ID and to provide a thorough discussion of the issues involved in reaching an accurate diagnosis. Mental health problems in people with ID are associated with poorer outcomes in major life areas including employment, independent living, quality of life, and health.

Behavioral Health. The presence of ASD and ID have been shown to independently increase the likelihood of engaging in behavioral and emotional problems in children (Totsika et al. 2011), and rates of problem behavior are highest in those with both ASD and ID (McCarthy et al. 2010). Examples of common problem behaviors include aggression, self-injury, disruption, inappropriate sexual behavior, and elopement. The presence of ID can increase risk for developing problem behavior due to the person’s impaired ability to communicate wants and needs or to cope with stressors. Problem behaviors can increase a person’s risk of negative outcomes as they can reduce the person’s access to the community, in particular educational, social, health care, and familial supports.

Outcomes

People with ID are living longer, with life expectancy approaching that of the general population. Because supports for adults with ID are primarily provided by their families of origin, the aging of people with ID becomes a problem for the family and the community. As the parents of adults with ID age, their ability to support their child with the complex care needs associated with having ID and ASD is reduced, particularly if the adult has comorbid physical, mental, or behavioral health needs. This forces families to consider residential placements for their child more frequently than parents were required to in previous generations.

Residential and other support services should be provided in the *least restrictive environment*, that is, a strong preference is given to inclusive settings with peers who do not have disabilities because of the educational and social benefits associated with learning from and interacting with these peers. As part of the US Individuals with Disabilities Education Act (IDEA ca. 1975), the least restrictive environment was identified as one of the principles governing the education of students with disabilities and other special needs. By law, schools are required to provide a free public education in the least restrictive environment that is appropriate to the individual student’s needs. The IDEA is in concert with the movement toward inclusion for people with ID that began with deinstitutionalization in the 1970s and continues today. The least restrictive environment principle guides the provision of residential, vocational, and other community services for adults as well as children with ID and other disabilities. Positive outcomes in terms of health, mental health, and quality of life are associated with self-determination and the availability of social and other supports that meet the individual needs of the person.

Clinical Expression and Pathophysiology

ID is not a disease, a syndrome, or a designation that encompasses a group of medical disorders. It is instead a construct embedded within the

broader construct of disability and descriptions of human functioning. Because of the varied nature and complex interrelated causal processes contributing to ID, it is not possible to succinctly summarize the clinical expression or pathophysiology of the condition. (Please see preceding section “[Natural History, Prognostic Factors, and Outcomes](#),” for more information.)

Evaluation and Differential Diagnosis

Assessment of ID involves systematically collecting information for decision-making and communication related to three assessment functions: diagnosis, classification, and planning individual supports. Within each of these functions, professionals conduct assessments for a variety of specific purposes. For example, a diagnosis might determine an individual’s eligibility for services or legal protection, or it might establish whether a person could be included in a research sample. In order to achieve specific assessment purposes, three criteria must be met: (1) the assessment tools and process should match the purpose for assessment, (2) the assessment findings should be as valid as possible, and (3) the results should be both useful and purposefully applied. Clinical judgment is essential to enhance the quality, validity, and precision of the clinician’s decision and recommendations. Clinical judgment is defined as a special type of judgment rooted in a high level of clinical expertise and experience, and that emerges directly from extensive training, experience with the person, and extensive data (Schalock et al. 2010b). A higher level of clinical judgment is frequently required when the complexity of the person’s functioning or co-occurring disorders precludes standardized assessment.

A diagnosis of ID is made when three criteria are met: (1) significant limitation in intellectual functioning, (2) significant limitation in adaptive behavior, and (3) the onset of limitations occurs before age 18. The terminology of ID has changed frequently (see ► [Mental Retardation](#) entry), yet for the past 50 years, the definition has remained fairly consistent. The American Association on

Intellectual and Developmental Disabilities (2010) defines a significant limitation in intellectual functioning as two standard deviations below the population mean IQ score, equating to a cutoff of 70 or below (plus or minus standard error of measurement). Limitations in adaptive behavior are to be assessed using a standardized measure that has been normed on the general population. Significant limitation is defined as two standard deviations below the mean in the realms of one or more areas of adaptive functioning, conceptual (language, reading, writing, understanding of time and number concepts), social (relating to others, self-esteem, social problem-solving, naïveté, and rule following), or practical (self-care, vocational skills, health care, safety behaviors, use of transportation, technology, and money), or can be determined based on an overall score from all three of these domains. The use of an age criterion distinguishes ID from cognitive impairments that occur at later points in life, such as those associated with traumatic brain injury, mental illness, or dementia. Once a diagnosis of ID is made or suspected, it may be helpful to explore the etiology, as certain genetic conditions have important implications for the health of the individual and for the risk of transmitting an inherited disorder.

Individuals with ID require accommodations to participate in assessments, including psychiatric assessment. Hurley et al. (2003) recommend the following adjustments to a clinical interview: (1) the use of simple vocabulary when interviewing the individual, (2) short, easy to understand sentences, (3) one question at a time, (4) waiting for the individual’s answer before the next question is asked, and (5) the assurance that the individual has complete understanding of the question that is being asked. The examiner must be explicit when clarifying questions due to a more concrete style of reasoning that is commonly displayed by individuals with ID and ASD. Closed-ended (requiring only yes/no answers) or leading questions should also be avoided as they are associated with biased responding. Family members and direct support professionals should also be interviewed for additional information on the individual’s symptoms, especially chronology of

symptom onset, in what environments the problem is observed, and why evaluation is being requested.

Treatment

There is no known cure for ID; however, intervention should be considered at three levels: primary prevention, with the goal of preventing the condition before its biological onset through appropriate prenatal and early childhood care; secondary prevention, which involves early identification and intervention when early signs of ID are identified but before a formal diagnosis is made; and tertiary prevention, the prevention of symptom exacerbation once a diagnosis is made. In the case of ID or developmental delay, tertiary prevention includes early intervention, special education, and finding effective accommodations to help the individual maximize functioning and community participation. This medical model of prevention is more traditionally applied to diseases and physical conditions, yet it offers a helpful context for the various interventions and supports available to people with ID.

Primary Prevention. The goal of primary prevention is to maximize the health of the mother and baby, preventing diseases and conditions, including ID, before their biological onset. Strategies include promoting maternal fitness and healthy nutrition, which can include taking prenatal vitamins with folic acid to promote healthy neural tube development. During pregnancy, women are encouraged to avoid drinking alcohol, using illegal substances, and taking many prescription medications due to the known teratogenic effects on the fetus. Avoidance of drug and alcohol use is especially important, as prenatal exposure to alcohol has been identified as the leading known preventable cause of ID (CDC 2015). After the child is born, primary prevention initiatives include childhood vaccinations against diseases that pose high risks for the baby's life and healthy development. Childhood diseases such as *Haemophilus influenzae* type b (Hib), measles, mumps, and rubella can cause brain inflammation, brain damage, and death in young children. The

safety of childhood vaccinations has been questioned, and an association was claimed between autistic symptoms and vaccines in a study that was later shown to be a deliberate fraud (Wakefield et al. 1998). The Centers for Disease Control and Prevention established a research agenda to investigate safety concerns, which provided overwhelming evidence of the safety of vaccines for infants and young children. This research definitively established that vaccines are not associated with ASD (CDC 2009; see Interagency Autism Coordinating Committee, National Vaccine Advisory Committee). Unfortunately, the now-discredited Wakefield study panicked many parents and led to a sharp drop in the number of children receiving the vaccine that prevents measles, mumps, and rubella. Measles cases have increased dramatically in the ensuing years. Primary prevention efforts to protect children continue throughout childhood and include environmental health initiatives, such as removing lead from paint and gasoline, and public safety initiatives, such as mandating car seats and bicycle helmets to prevent traumatic head injury.

Secondary Prevention. Secondary prevention consists of the identification and interdiction of diseases that are present in the body but that have not progressed to the point of causing signs, symptoms, and dysfunction. Once an individual has been diagnosed with a disease, the emphasis turns to prevent the emergence of symptoms associated with the condition. For example, phenylketonuria (PKU) is a rare condition in which a baby is born without the ability to properly break down the amino acid phenylalanine. Untreated, PKU is associated with delayed mental and social development, hyperactivity, seizures, and severe intellectual disability. These impairments can be completely avoided when a special diet that is extremely low in phenylalanine is followed. Most states require a PKU screening for all newborns so that treatment can begin immediately.

Tertiary Prevention. Tertiary prevention consists of the preventing disease progression, rehabilitation, and reducing the sequelae of the condition. Services are provided to reduce the number, extent, and severity of direct or indirect side effects associated with the identified condition. For example,

early intervention will enhance the development of young children already exhibiting developmental delays (of known or unknown etiology) both by altering their developmental trajectories and by preventing secondary complications from occurring. For children at risk of ID because of a variety of biological and/or environmental conditions, it is expected that these delays can be prevented entirely or their magnitude minimized through early intervention services (Guralnick 2005). Common services that are funded by federal and state entities include specialized preschool services, special education programs throughout elementary, middle, and high school, and, for young adults, programs to assist in the transition from a high school to a vocational setting. For children with ID who have specific behavioral health concerns, parent training and behavior support services are commonly recommended and used. Individual and group psychotherapy can be helpful in treating behavioral health conditions in children and adults with ID (NICE 2017; Schalock et al. 2010a). Peer support groups are organized for adults with ID to provide and receive social and emotional support, as well as skill building. Adults with ID benefit from job training and case management services to improve employment and community living outcomes. Numerous advocacy groups have also been organized across the United States and other countries to promote equal rights and opportunities for all individuals with ID (United Nations 2006).

For adults with ID and ASD, individual service plans are centered on the individual's interests and goals and focused on providing supports and services to promote overall well-being. Plans are intended to adopt a dynamic, life span approach, with the emphasis on continuous revision of goals to meet the individual as he or she progresses through life. Treatment plans strive to address multiple aspects of life, including self-care skills, social skills, education, and physical, mental, and behavioral health. Self-determination skills are commonly addressed in treatment plans including self-awareness, self-advocacy, self-efficacy, decision-making, independent performance, self-evaluation, and adjustment that contribute to an individual's ability to establish and achieve his or her own goals.

See Also

► Mental Retardation

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association.
- Bowring, D. L., Totsika, V., Hastings, R. P., Toogood, S., & Griffith, G. M. (2017). Challenging behaviours in adults with an intellectual disability: A total population study and exploration of risk indices. *British Journal of Clinical Psychology*, 56(1), 16–32.
- Bradley, E., Summers, J., Brereton, A., Einfeld, S., Haverkamp, S., Holt, G., et al. (2007). Intellectual disabilities and behavioral, emotional, and psychiatric disturbances. In I. Brown & M. Percy (Eds.), *A comprehensive guide to intellectual and developmental disabilities*. Baltimore: Paul H. Brookes.
- Brown, I., & Percy, M. (Eds.). (2007). *A comprehensive guide to intellectual and developmental disabilities*. Baltimore: Paul H. Brookes.
- Buitelaar, J. K., Van der Gaag, R., Klin, A., & Volkmar, F. R. (1999). Exploring the boundaries of pervasive developmental disorder not otherwise specified: Analyses of data from the DSM-IV autistic disorder field trial. *Journal of Autism and Developmental Disorders*, 29, 33–43.
- Centers for Disease Control. (2005). Intellectual disability: How common is intellectual disability? Retrieved 12 June, 2012 from <http://www.cdc.gov/ncbddd/dd/mr3.htm>
- Centers for Disease Control. (2009). Centers for disease control and prevention: Immunization safety and autism thimerosal and autism research agenda. Downloaded 5 May, 2011 from http://www.cdc.gov/ncbddd/autism/documents/vaccine_studies.pdf
- Centers for Disease Control. (2010). Autism and developmental disabilities monitoring (ADDM) network. Retrieved 12 June, 2012 from <http://www.cdc.gov/ncbddd/autism/addm.html>
- Centers for Disease Control and Prevention. (2015). Facts: Developmental disabilities, NCBDDD. Retrieved from <https://www.cdc.gov/ncbddd/developmentaldisabilities/facts.html>
- Cooper, S.-A., Smiley, E., Morrison, J., Williamson, A., & Allan, L. (2007). Mental ill-health in adults with intellectual disabilities: Prevalence and associated factors. *The British Journal of Psychiatry*, 190(1), 27–35.
- Corbin, S., Holder, M., & Engstrom, K. (2005). *Changing attitudes, changing the World: The health and health care of people with intellectual disabilities*. Washington, DC: Special Olympics International. Downloaded 5 May, 2011 from http://resources.specialolympics.org/research-toolkit/Training_of_Health_Professionals.aspx

- Einfeld, S. L., Ellis, L. A., & Emerson, E. (2011). Comorbidity of intellectual disability and mental disorder in children and adolescents: A systematic review. *Journal of Intellectual and Developmental Disability*, 36(2), 137–143.
- Elliot, C. (2007). *Differential Abilities Scale – 2nd edition (DAS-II) manual* (2nd ed.). San Antonio: Harcourt Assessment, Inc..
- Fletcher, R., Barnhill, J., & Cooper, S. A. (Eds.). (2016). *Diagnostic manual-intellectual disability: A textbook of diagnosis of mental disorders in persons with intellectual disability*. Kingston: NADD Press.
- Guralnick, M. J. (2005). Early intervention for children with intellectual disabilities: Current knowledge and future prospects. *Journal of Applied Research in Intellectual Disabilities*, 18, 313–324.
- Harris, J. C. (1995). *Developmental neuropsychiatry, Volume II: Assessment, diagnosis, and treatment of developmental disorders*. Oxford: Oxford University Press.
- Havercamp, S. M., & Scott, H. M. (2015). National health surveillance of adults with disabilities, adults with intellectual and developmental disabilities, and adults with no disabilities. *Disability and Health Journal*, 8(2), 165–172.
- Havercamp, S. M., Scandlin, D., & Roth, M. (2004). Health disparities among adults with developmental disabilities, adults with other disabilities, and adults not reporting disability in North Carolina. *Public Health Reports*, 119, 418–426.
- Horwitz, S. M., Kerker, B. D., Owens, P. L., & Zigler, E. (2000). *The health status and needs of individuals with mental retardation*. New Haven: Yale University.
- Hurley, A. D., Folstein, M., & Lam, N. (2003). Patients with and without intellectual disability seeking outpatient psychiatric services: Diagnoses and prescribing pattern. *Journal of Intellectual Disability Research*, 47, 39–50.
- Iezzoni, L. I., & O'Day, B. L. (2006). *More than ramps: A guide to improving health care quality and access for people with disabilities*. New York: Oxford University Press.
- Jacobson, J. W., Mulick, J. A., & Rojahn, J. (2008). *Handbook of intellectual disabilities and developmental disabilities: Issues in clinical child psychology*. New York: Springer.
- Kamphaus, R. W., & Reynolds, C. R. (2015). *Behavior assessment system for children (BASC-3)* (3rd ed.). San Antonio: Pearson.
- Kaufman, L., Ayub, M., & Vincent, J. B. (2010). The genetic basis of non-syndromic intellectual disability: A review. *Journal of Neurodevelopmental Disorders*, 2(4), 182–209.
- Kirschner, K. L., & Curry, R. H. (2009). Educating health care professionals to care for patients with disabilities. *JAMA*, 302(12), 1334–1335.
- LaMalfa, G., Lassi, G., Bertelli, M., Salvini, R., & Placidi, G. F. (2004). Autism in intellectual disability: A study of prevalence on a sample of the Italian population. *Journal of Intellectual Disability Research*, 48, 262–267.
- Lecavalier, L., Snow, A. V., & Norris, M. (2011). Autism spectrum disorders and intellectual disability. In J. L. Matson & P. Sturmey (Eds.), *International handbook of autism and pervasive developmental disorders* (pp. 37–51). New York: Springer.
- Maenner, M. J. (2020). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 Sites, United States, 2016. *MMWR. Surveillance Summaries*, 69. <https://doi.org/10.15585/mmwr.ss6904a1>
- Matson, J. L., & Shoemaker, M. (2009). Intellectual disability and its relationships to autism spectrum disorders. *Research in Developmental Disabilities*, 30, 1107–1114.
- McCarthy, J., Hemmings, C., Kravariti, E., Dworzynski, K., Holt, G., Bouras, N., & Tsakanikos, E. (2010). Challenging behavior and co-morbid psychopathology in adults with intellectual disability and autism spectrum disorders. *Research in Developmental Disabilities*, 31(2), 362–366.
- McDermott, S., Moral, R., Platt, T., et al. (2005). Prevalence of epilepsy in adults with mental retardation and related disabilities in primary care. *American Journal on Mental Retardation*, 110(1), 48–56.
- Moeschler, J. B., Shevell, M., & Genetics, C. O. (2014). Comprehensive evaluation of the child with intellectual disability or global developmental delays. *Pediatrics*, 134(3), 903–918.
- Moser, H. W. (2004). Genetic causes of mental retardation. *Annals of the New York Academy of Sciences*, 1038, 44–48.
- Muhle, R., Trentacoste, S. V., & Rapin, I. (2004). The genetics of autism. *Official Journal of the American Academy of Pediatrics*, 113(5), 472–486.
- Munson, J., Dawson, G., Sterling, L., Beauchaine, T., Zhou, A., Koehler, E., . . . Abbott, R. (2008). Evidence for latent classes of IQ in young children with autism spectrum disorder. *American Journal on Mental Retardation*, 113(6), 439–452.
- NICE. (2017). *Learning disabilities: Identifying and managing mental health problems | Guidance and guidelines | NICE*. London: NICE.
- Nickel, R. E. (2000). Developmental delay and mental retardation. In R. E. Nickel & L. W. Desch (Eds.), *The physician's guide to caring for children with disabilities and chronic conditions* (pp. 99–139). Baltimore: Paul H. Brookes.
- Oakland, T., & Harrison, P. L. (2015). *Adaptive behavior assessment system-II: Clinical use and interpretation* (3rd ed.). Torrance: Western Psychological Services.
- Percy, M. (2007). Factors that cause or contribute to intellectual and developmental disabilities. In I. Brown & M. Percy (Eds.), *A comprehensive guide to intellectual and developmental disabilities*. Baltimore: Paul H. Brookes.
- Roid, G. (2005). *Stanford-Binet intelligence scales (SB5)* (5th ed.). Torrance: Western Psychological Services.

- Schalock, R. L., Verdugo, M. A., Bonham, G. S., Fantova, F., & Van Loon, J. (2010a). Enhancing personal outcomes: Organizational strategies, guidelines, and examples. *Journal of Policy and Practice in Intellectual Disabilities*, 5(4), 276–285.
- Schalock, R. L., Borthwick-Duffy, S. A., Bradley, V. J., Buntinx, W. H. E., Coulter, D. L., Craig, E. M., Gomez, S. C., Lachapelle, Y., Luckasson, R., Reeve, A., Shogren, K. A., Snell, M. E., Spreat, S., Tassé, M. J., Thompson, J. R., Verdugo-Alonso, M. A., Wehmeyer, M. L., & Yeager, M. H. (2010b). *Intellectual disability: Definition, classification, and systems of supports* (11th ed.). Washington, DC: American Association on Intellectual and Developmental Disabilities.
- Schützwohl, M., Koch, A., Koslowski, N., Puschner, B., Voß, E., Salize, H. J., ... Vogel, A. (2016). Mental illness, problem behaviour, needs and service use in adults with intellectual disability. *Social Psychiatry and Psychiatric Epidemiology*, 51(5), 767–776.
- Sheehan, R., Hassiotis, A., Walters, K., Osborn, D., Strydom, A., & Horsfall, L. (2015). Mental illness, challenging behaviour, and psychotropic drug prescribing in people with intellectual disability: UK population based cohort study. *British Medical Journal*, 351, 4326.
- Skuse, D. H. (2000). Imprinting, the X-chromosome, and the male brain: Explaining sex differences in the liability to autism. *Pediatric Research*, 47(1), 9.
- Sparrow, S. S., Cicchetti, D. V., & Saulnier, C. A. (2016). *Vineland adaptive behavior scales (Vineland-3)* (3rd ed.). San Antonio: Pearson.
- Special Olympics. (2010). Special Olympics finds poor medical school training contributes to health care disparities for people with intellectual disabilities. Retrieved 10 December, 2011 from <http://northamerica.specialolympics.org/research/documents/Health-Research-Release.doc>
- Switzky, H. N., & Greenspan, S. (Eds.). (2006). *What is mental retardation? Ideas for an evolving disability in the 21st century* (revised and updated edition). Washington, DC: Library of Congress: American Association on Mental Retardation.
- The United Nations. (2006). Convention on the rights of persons with disabilities. *Treaty Series*, 2515, 3.
- Tomasulo, D. J., & Razza, N. J. (2007). Post-traumatic stress disorders. In R. Fletcher, E. Loschen, C. Stravraki, & M. First (Eds.), *Diagnostic manual-intellectual disability: A clinical guide for diagnosis of mental disorders in persons with intellectual disability* (pp. 215–224). Kingston: NADD Press.
- Totsika, V., Hastings, R. P., Emerson, E., Lancaster, G. A., & Berridge, D. (2011). A population-based investigation of behavioural and emotional problems and maternal mental health: Associations with autism spectrum disorder and intellectual disability. *Journal of Child Psychology and Psychiatry*, 52(1), 91–99.
- U.S. Department of Health and Human Services. (2005). *The surgeon general's call to action to improve the health and wellness of persons with disabilities*. Washington, DC: U.S. Department of Health and Human Services, Office of the Surgeon General.
- U.S. Public Health Service. (2002). *Closing the gap: A national blueprint for improving the health of individuals with mental retardation. Report of the Surgeon general's conference on health disparities and mental retardation*. Washington, DC: U.S. Public Health Service.
- Van Naarden, B. K., Autry, A., & Boyle, C. (2005). A population-based study of the recurrence of developmental disabilities – Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1991–4. *Pediatric and Perinatal Epidemiology*, 19(1), 69–79.
- Wakefield, A. J., Murch, S. H., Anthony, A., Linnell, J., Casson, D. M., Malik, M., et al. (1998). Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *The Lancet*, 351(9103), 637–641.
- Wechsler, D. (1991). *The Wechsler intelligence scale for children* (3rd ed.). San Antonio: The Psychological Corporation.
- Wilson, J. G., Lott, R. S., & Tsai, L. (2004). Side effects: Recognition and management. In S. Reiss & M. G. Aman (Eds.), *Psychotropic medications and developmental disabilities: The international consensus handbook* (pp. 95–114). Columbus: The Ohio State University Nisonger Center Press.
- Witwer, A. N., & Lecavalier, L. (2008). Validity of autism spectrum disorder subtypes. *Journal of Autism and Developmental Disorders*, 38(9), 1611–1624.
- World Health Organization. (2018). *International statistical classification of diseases and related health problems, 11th revision*. Geneva: World Health Organization.
- Young-Southward, G., Rydzewska, E., Philo, C., & Cooper, S.-A. (2017). Physical and mental health of young people with and without intellectual disabilities: Cross-sectional analysis of a whole country population. *Journal of Intellectual Disability Research*, 61(10), 984–993.

Intellectual Disability (ID)

► DiBAS-R Validation

Intellectual Impairment

► Intellectual Disability

Intellectualization of Affect

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Synonyms

[Intellectualization of emotion](#)

Definition

This term has been used to characterize aspects of the ways in which more able individuals on the autism spectrum, particularly those with Asperger's disorder, cope with their difficulties in empathy and perception of the feelings of others. Asperger (1944) emphasized this as one aspect of the clinical presentation of the latter condition, and subsequent investigators (e.g., Klin et al. 2005) have also underscored this unique approach to understanding other people's feelings.

The concept represents an extension of the more general notion of intellectualization as a defense mechanism, that is, for the more typical population the approach to thinking about troubling situations or anxiety-laden issues may help provide some distance from the experience of unpleasant affects. Although prefigured, as a more general concept, by Sigmund Freud, it was his daughter Anna who elaborated the more specific concept and noted its developmental correlates.

Individuals with Asperger's engage in various approaches to coping with the problem of understanding other people's feelings and intentions. In their often somewhat professorial style, they may try to "decode" the feelings of others by approaching the problem in a highly intellectual, as opposed to more empathic, way. When carried to an extreme, this approach can take the form of

mathematical modeling of people's affective states.

See Also

- [Asperger's Disorder](#)
- [Empathy](#)
- [Theory of Mind](#)

References and Reading

- Asperger, H. (1944). Die "autistischen Psychopathen" im Kindersalter. *Archiv für psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Klin, A., McPartland, J., & Volkmar, F. R. (2005). Asperger syndrome. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 88–125). Hoboken: Wiley.

Intellectualization of Emotion

- [Intellectualization of Affect](#)

Intelligence Norms

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Definition

Intelligence is generally assessed using "norm-referenced tests," which evaluate test-takers not on their absolute performance but on their relative ranking compared to the scores obtained within a predetermined population, most often a nationally representative sample of same-aged peers. Individual scores represent how much better or worse any single person scores relative to the average. Norms can be generated for tests that are standardized or are administered in a consistent

manner to all individuals who take the test. Most often, an individual's performance in relation to the norms are provided in the form of a standard score, which has a mean of 100 (average) and a standard deviation of 15. Beginning in the early twentieth century, it was noted that raw scores on intelligence tests have consistently risen over time, so that the average score at any given time is significantly higher than the average had been several years before. This phenomenon is known as the Flynn Effect, a term coined in the book "The Bell Curve" by authors Richard Herrnstein and Charles Murray. It was named after James R. Flynn, who first documented the effect. Every few years, intelligence tests must be updated and re-normed so that the scores obtained truly reflect an individual's performance with respect to the rest of the population at that time.

Recent studies have suggested the intelligence tests may not accurately capture and may underestimate the abilities of individuals with Autism Spectrum Disorder (ASD). Children with ASD were found to score significantly higher on a test measuring "fluid intelligence", which involves novel problem-solving skills such as task management, understanding rules, and mental abstraction, than they did on the Wechsler Intelligence Scale for Children (WISC IV), which is thought to be heavily reliant on language skills and previously learned information (► [Crystallized Intelligence](#)). Typically developing children tended to score equally well on both tests.

See Also

► [Norm-Referenced Testing](#)

References and Reading

- Bowler, D. (2007). *Autism spectrum disorders: Psychological theory and research*. Hoboken: Wiley.
- Flanagan, D. P., & Kaufman, A. S. (2009). *Essentials of WISC-IV assessment* (2nd ed.). Hoboken: Wiley.
- Groth-Marnat, G. (2009). *Handbook of psychological assessment* (5th ed.). Hoboken: Wiley.

Intelligence Quotient

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Synonyms

[IQ](#)

Definition

Intelligence quotient, abbreviated IQ, was originally the ratio of mental age to chronological age. The term "mental age," popularized by early tests of intelligence, referred to the age of the children in the standardization sample whose performance the testee matched. Most tests of intelligence no longer use this ratio, and IQ instead refers to a person's ability relative to available norms, which are usually age-based. By convention, IQ scores are designed to have a mean of 100 and a standard deviation of 15. Thus, about 95% of people fall within two standard deviations of the mean (i.e., 70–130). Norm-referenced scores have decreased reliability at extreme values; for this reason, most tests do not provide scores more than four standard deviations away from the mean (40–160). Giftedness is sometimes defined as scores more than two standard deviations above the mean. Intellectual disability is defined based on both IQ and adaptive behavior; however, the current Diagnostic and Statistical Manual (APA, 2013) no longer classifies intellectual disability into levels based on IQ. Within the ASD literature, the terms "low functioning" and "high functioning" are sometimes used to denote IQs below 70 and above 70, respectively, though there is no empirical rationale for this practice. Although IQ and "intelligence" are often used interchangeably,

IQ represents intelligence as defined by performance on a specific test.

See Also

- [Giftedness](#)
- [Intellectual Disability](#)
- [Psychological Assessment](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual* (5th ed.). Washington, DC: APA Press.
- Anastasi, A., & Urbina, S. (1997). *Psychological testing* (7th ed.). Upper Saddle River: Prentice Hall.
- Neisser, U., Boodoo, G., Bouchard, T., Boykin, A., Brody, N., Ceci, S., et al. (1996). Intelligence: Knowns and unknowns. *American Psychologist*, 51, 77–101.
- Sattler, J. (2001). *Assessment of children: Cognitive applications* (4th ed.). San Diego: Jerome M. Sattler.
- Schalock, R., Borthwick-Duffy, S., Bradley, V., Buntinx, W., Coulter, D., Craig, E., et al. (2010). *Intellectual disability: Definition, classification, and systems of supports*. Washington, DC: American Association on Intellectual and Developmental Disabilities.
- Sternberg, R., & Detterman, D. (Eds.). (1986). *What is intelligence? Contemporary viewpoints on its nature and definition*. Norwood: Ablex.

Intelligence Tests (see Specific Tests)

- [Psychological Assessment](#)

Intentional

- [Protoimperative](#)

Intentional Communication

- [Initiation of Communication](#)

Interactive Trauma Scale

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Synonyms

ITS

Description

The Interactive Trauma Scale (ITS) is an interactive touchscreen self-report measure of childhood trauma symptoms, administered via touchscreen tablet or smartphone, for children and youth with autism spectrum disorder (ASD). The measure is designed for use with children ages 6–14. Items are read aloud by the examiner as needed. Eight trauma exposure items ask about traumatic and adverse experiences. These are followed by 22 symptom items congruent with DSM-5 criteria for posttraumatic stress disorder (American Psychiatric Association 2013) and 7 neutral items (e.g., “I like dinosaurs”) interspersed to maintain the child’s engagement and comfort. Children respond via a 5-point thermometer-like scale rated from “Never” to “Always.”

History

This screening measure originated from a clinical need to obtain firsthand child and adolescent input in mental health assessments following suspected abuse, neglect, peer victimization (see ► [“Peer Victimization”](#)), and other potentially traumatic events. Children with ASD are frequently exposed to potentially traumatic and other adverse experiences, though posttraumatic stress disorder (PTSD) in this population is only beginning to be studied. Current evidence indicates that children

with ASD are more likely than other children to be abused or maltreated or to be victims of bullying than nondisabled children, their own siblings, and peers with other disabilities (Maïano et al. 2016; McDonnell et al. 2018; Zeedyk et al. 2014). They are also more likely than their typically developing peers to experience adverse childhood experiences such as household mental illness and substance abuse, income insufficiency, parental divorce, and neighborhood violence (Berg et al. 2016; Kerns et al. 2017). Following traumatic events, identifiable symptoms of anxiety, depression, PTSD, regression in adaptive behavior, and suicidality are evidenced in youth with ASD (Bleil Walters et al. 2013; Mayes et al. 2013; Mehtar and Mukaddes 2011; Valenti et al. 2012).

Complicating the assessment of trauma symptoms in children and youth with ASD are associated communication deficits, emotion regulation difficulties, and multiple co-occurring psychiatric problems (Matson and Cervantes 2014). A PTSD diagnosis in youth with ASD may also be missed or overlooked due to “diagnostic overshadowing” in which assessors inaccurately attribute challenging behaviors or anxiety to the ASD itself (Kerns et al. 2015; see ► “Diagnostic Overshadowing”). Likely because of their core deficits in communication, emotional understanding, and expression, children with ASD show differences from typically developing children when asked to report psychological symptoms on behavior rating scales (Mazefsky et al. 2011; Mahan and Matson 2011). On self-report measures, they tend to self-enhance relative to typically developing peers, fail to complete measures, or interpret items in an overly literal manner, though there is evidence that higher functioning youth with ASD show no less self-awareness than typically developing youth (Schriber et al. 2014). Clinicians have come to rely on the reports of parents or other observers for this purpose (Mahan and Matson 2011). However, there is much evidence that even parents of typically developing, emotionally expressive children often rate them much differently than children rate themselves on a variety of measures (Renk and Phares 2004). Child and adolescent self-reports have been shown to add significantly to the diagnostic picture in ways that are

unique and not accounted for by observer reports (Adams et al. 2014).

Treatment providers and educators have increasingly turned to electronic touchscreen platforms including tablets, smartphones, and computers to present material to children with ASD (see ► “Computer-Assisted Instruction to Teach Academic Skills”). Some of these programs have been shown to effectively engage children with ASD for education and treatment in increasingly effective ways (Neumann 2018).

Psychometric Data

During the initial creation of the ITS prototype, graphics and response features were programmed and then posted for review on an online beta testing site ([Testing.org](https://www.testing.org)). The prototype was then reviewed by a group of eight clinicians familiar with both autism and trauma in children. The original versions were improved based on feedback by the reviewers by adjusting the questions and the graphic design to enhance understanding and to provide unambiguous representation of trauma-related stimuli. These changes were incorporated into a revised version of the prototype for psychometric analyses.

Trauma exposure items for the prototype were selected based on commonly reported experiences in youth that are similarly tapped in other scales such as the UCLA Posttraumatic Stress Disorder Reaction Index (UCLA-PTSD-RI; Steinberg et al. 2013). Children are asked whether or not they have had physical neglect experiences, were “bullied or hurt by other kids,” witnessed “bad or scary things like someone getting hurt,” “hit or hurt by an adult,” touched by someone on “your private parts or get you to touch their private parts,” “teased or called mean names,” “in a natural disaster like a hurricane or earthquake,” and lost someone “because they died or went away.” Questions about exposure to bullying and teasing are included because of the high frequency of such experiences in children with ASD.

Symptom items for the prototype were written to assess each of the five DSM-5 PTSD criterion domains (American Psychiatric Association

2013): intrusion symptoms (e.g., “I have bad dreams”), negative mood (e.g., “I am no good”), dissociative symptoms (e.g., “I feel like things aren’t real”), avoidance (e.g., “I try to stay away from people, places or things that remind me. . .”), and arousal symptoms (e.g., “I do things that are not safe”). The measure is scored by summing Likert scale ratings of 3 (moderate level rating) and above in each criterion area. Respondents are judged to screen positive for PTSD symptoms when at least one exposure item is endorsed and one or more symptom item is endorsed in each of the intrusion and avoidance domains, with two or more in the arousal and negative mood domains, consistent with DSM-5 criteria.

User Satisfaction

In a recent validation study (Hoover and Romero 2019), participant ratings were highly positive: 81% rated it as easy or very easy to use; 74% rated it as comfortable and understandable or “very much” so, and 76% rated it as effectively facilitating identification of feelings. The overall experience was rated as “good” or “really good” by 88% of participating children.

Validity and Reliability

Children’s endorsements of traumatic exposures on the ITS were moderately correlated with their responses to similar items on the UCLA Post-traumatic Stress Disorder Reaction Index-Self Report (UCLA-SR; $r = 0.554$; $p < 0.05$) and UCLA parent report form (UCLA-PR; $r = 0.638$; $p < 0.01$). The item “teased or called mean names” was the most frequently endorsed (75%) on the ITS, followed by “bullied or hurt by other kids” (70%). Death/loss of a loved one (65%) and witnessing “bad or scary things like someone getting hurt” (50%) were also frequently endorsed.

Overall, the ITS and UCLA-SR trauma symptom severity ratings were strongly correlated ($r = 0.782$; $p < 0.01$) with lower correlations between the ITS and UCLA-PR ($r = 0.223$) and between the UCLA-SR and UCLA-PR ($r = 0.314$). Table 1 presents Pearson’s r correlations between ratings on items of similar content across the three measures. Of the 19 individual

Interactive Trauma Scale, Table 1 Correlations between similar ITS, UCLA-SR, and UCLA-PR symptom items ($n = 20$)

	ITS/ UCLA- SR	ITS/ UCLA- SR	UCLA- SR/ UCLA- PR
Total correlation	0.782**	0.223	0.314
Symptom items			
Bad dreams	0.155	0.479*	−0.009
Flashbacks	0.341	0.203	0.188
Try not to think	−0.049	−0.202	0.300
Reminders upsetting	0.105	0.452*	0.589**
Avoid people, places	0.348	0.434	0.326
Amnesia for event	0.549*	−0.063	0.413
Self-blame/guilt	0.697**	0.535*	0.469*
Self “bad/no good”	0.620**	0.050	0.216
Sad/not happy	0.233	0.025	−0.230
Scared/afraid	0.514*	−0.028	−0.022
Diminished interests	0.089	0.631**	0.250
Diminished closeness	0.622**	0.364	0.486*
Angry	0.682**	0.332	0.236
Risky/unsafe	0.407	0.272	−0.042
Hypervigilance	0.265	0.422	0.004
Startles easily	0.663**	0.296	0.203
Attention/concentration	0.748***	0.547*	0.445*
Sleep disturbance	0.785***	0.364	0.137
De-realization	0.374	0.362	−0.079

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

item comparisons between the UCLA-SR and ITS, 13 were moderately to strongly correlated. These include the following items: scared/afraid ($r = 0.514$; $p < 0.05$), amnesia ($r = 0.549$; $p < 0.05$), feelings of being “no good” ($r = 0.620$; $p < 0.01$), diminished closeness with others ($r = 0.622$; $p < 0.01$), feeling angry ($r = 0.682$; $p < 0.01$), self-blame/guilt ($r = 0.697$; $p < 0.01$), difficulties with attention

and concentration ($r = 0.748$; $p < 0.001$), and sleep disturbance ($r = 0.785$; $p < 0.001$).

Five (25%) of the participants screened positive for PTSD diagnosis on the ITS as they endorsed the requisite symptoms in each DSM-5 criterion area. These participants reported an average of 6.2 trauma exposure types (SD 2.925) and all of them endorsed experiences of bullying and teasing victimization. Those who did not screen positive for PTSD on the ITS reported experiencing fewer types of traumatic events ($M 3.80$; $SD 1.707$; $t = 2.06$, $p < 0.05$). PTSD-positive screening youth had higher overall frequency scores on the ITS ($M 62.40$; $SD 11.876$) than those who did not screen positive on the ITS ($M 41.53$; $SD 14.366$; $t = 2.78$, $p < 0.05$). Three of the five participants also screened positive for PTSD on the UCLA-SR and four on the UCLA-PR. Age-adjusted PVT scores were essentially uncorrelated with overall symptom frequency ratings on the ITS ($r = 0.187$). Internal consistency reliability was assessed using Cronbach's coefficient alpha ($\alpha = 0.855$), suggesting adequate internal consistency of the ITS symptom items.

Clinical Uses

The ITS employs a tablet or smartphone-based touchscreen delivery method meant to engage children with autism in order to obtain their self-reports of potentially traumatic events and symptoms. Touchscreen devices are popular and familiar to children with ASD and may facilitate screening and assessment in community and treatment settings, for initial identification of trauma, and referral for more comprehensive trauma evaluation.

The measure shows initial evidence for usefulness and reliability in clinical mental health settings with children and youth ages 6–14 who have ASD and suspected exposure to trauma. Given the paucity of studies and lack of trauma measures designed for use with children who have developmental disabilities, the ITS provides a potentially readily accessible screening format that has been

previously unavailable. Besides being a useful clinical tool, including firsthand reports from youth about their traumatic experiences rounds out a complete assessment to help answer as-yet unanswered questions about ASD and PTSD. For example, current research does not clarify whether children with ASD show similar symptoms to their typically developing peers (Hoover 2015). Some argue that children with ASD may be either more or less sensitive to traumatic experiences. Such sensitivity may depend on the child's overall level of adaptive functioning, emotion regulation, and attentiveness to potentially traumatizing information about their surroundings (Kerns et al. 2015).

The ITS asks specifically about peer victimization and its effects, an aspect that is missing in most extant childhood trauma measures. A history of physical and verbal bullying was reported by most of the validity sample (Hoover and Romero 2019) and is commonly reported by children with ASD. As serious negative mental health outcomes have been shown to arise from bullying, especially among youth with ASD (Zablotsky et al. 2013; Mayes et al. 2013), these youth tend to report differently about their victimization experiences than their caregivers, such assessment data is especially needed. Effective treatment of challenging behaviors, anxiety, and regression in adaptive functions will necessarily incorporate an understanding of children's trauma exposures and symptoms.

References and Reading

- Adams, R. E., Fredstrom, B. K., Duncan, A. W., Holleb, L. J., & Bishop, S. L. (2014). Using self- and parent-reports to test the association between peer victimization and internalizing symptoms in verbally fluent adolescents with ASD. *Journal of Autism and Developmental Disorders*, 44(4), 861–872. <https://doi.org/10.1007/s10803-013-1938-0>.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Publishing.
- Berg, K. L., Shiu, C., Acharya, K., Stolbach, B. C., & Msall, M. E. (2016). Disparities in adversity among

- children with autism spectrum disorder: A population-based study. *Developmental Medicine & Child Neurology*, 58(11), 1124–1131. <https://doi.org/10.1111/dmcn.13161>.
- Bleil Walters, J., Hughes, T. L., Sutton, L. R., Marshall, S. N., Crothers, L. M., Lehman, C., et al. (2013). Maltreatment and depression in adolescent sexual offenders with an autism spectrum disorder. *Journal of Child Sexual Abuse*, 22(1), 72–89. <https://doi.org/10.1080/10538712.2013.735357>.
- Hoover, D. W. (2015). The effects of psychological trauma on children with autism spectrum disorder: A research review. *Review Journal of Autism and Developmental Disorders*, 2(3), 287–299.
- Hoover, D. W., & Romero, E. M. G. (2019). The interactive trauma scale: A web-based measure of psychological trauma in children with autism. *Journal of Autism and Developmental Disorders*, 49(4), 1686–1692.
- Kerns, C. M., Kendall, P. C., Zickgraf, H., Franklin, M. E., Miller, J., & Herrington, J. (2015). Not to be overshadowed or overlooked: Functional impairments associated with comorbid anxiety disorders in youth with ASD. *Behavior Therapy*, 46(1), 29–39. <https://doi.org/10.1016/j.beth.2014.03.005>.
- Kerns, C. M., Newschaffer, C. J., Berkowitz, S., & Lee, B. K. (2017). Brief report: Examining the association of autism and adverse childhood experiences in the national survey of children's health: The important role of income and co-occurring mental health conditions. *Journal of Autism and Developmental Disorders*, 47(7), 2275–2281. <https://doi.org/10.1007/s10803-017-3111-7>.
- Mahan, S., & Matson, J. L. (2011). Children and adolescents with autism spectrum disorders compared to typically developing controls on the behavioral assessment system for children, (BASC-2). *Research in Autism Spectrum Disorders*, 5(1), 119–125.
- Maïano, C., Normand, C. L., Salvas, M., Moullec, G., & Aimé, A. (2016). Prevalence of school bullying among youth with autism spectrum disorders: A systematic review and meta-analysis. *Autism Research*, 9(6), 601–615.
- Matson, J. L., & Cervantes, P. E. (2014). Commonly studied comorbid psychopathologies among persons with autism spectrum disorder. *Research in Developmental Disabilities*, 35(5), 952–962. <https://doi.org/10.1016/j.ridd.2014.02.012>.
- Mayes, S. D., Gorman, A. A., Hillwig-Garcia, J., & Syed, E. (2013). Suicide ideation and attempts in children with autism. *Research in Autism Spectrum Disorders*, 7(1), 109–119. <https://doi.org/10.1016/j.rasd.2012.07.009>.
- Mazefsky, C. A., Kao, J., & Oswald, D. P. (2011). Preliminary evidence suggesting caution in the use of psychiatric self-report measures with adolescents with high-functioning autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5(1), 164–174. <https://doi.org/10.1016/j.rasd.2010.03.006>.
- McDonnell, C. G., Boan, A. D., Bradley, C., Seay, K. D., Charles, J. M., & Carpenter, L. A. (2018). Child maltreatment in autism spectrum disorder and intellectual disability: Results from a population-based sample. *Journal of Child Psychology and Psychiatry*, 60(5), 576–584.
- Mehtar, M., & Mukaddes, N. M. (2011). Posttraumatic stress disorder in individuals with diagnosis of autistic spectrum disorders. *Research in Autism Spectrum Disorders*, 5(1), 539–546.
- Neumann, M. M. (2018). Using tablets and apps to enhance emergent literacy skills in young children. *Early Childhood Research Quarterly*, 42, 239–246. <https://doi.org/10.1016/j.ecres.2017.10.006>.
- Renk, K., & Phares, V. (2004). Cross-informant ratings of social competence in children and adolescents. *Clinical Psychology Review*, 24(2), 239–254. <https://doi.org/10.1016/j.cpr.2004.01.004>.
- Schriber, R. A., Robins, R. W., & Solomon, M. (2014). Personality and self-insight in individuals with autism spectrum disorder. *Journal of Personality and Social Psychology*, 106(1), 112–130. <https://doi.org/10.1037/a0034950>.
- Steinberg, A. M., Brymer, M. J., Kim, S., Briggs, E. C., Ippen, C. G., Ostrowski, S. A., et al. (2013). Psychometric properties of the UCLA PTSD reaction index: Part I. *Journal of Traumatic Stress*, 26(1), 1–9. <https://doi.org/10.1002/jts.21780>.
- Valenti, M., Ciprietti, T., Di Egidio, C., Gabrielli, M., Masedu, F., Tomassini, A. R., & Sorge, G. (2012). Adaptive response of children and adolescents with autism to the 2009 earthquake in L'Aquila, Italy. *Journal of Autism and Developmental Disorders*, 42(6), 954–960.
- Zablotsky, B., Bradshaw, C. P., Anderson, C., & Law, P. A. (2013). The association between bullying and the psychological functioning of children with autism spectrum disorders. *Journal of Developmental and Behavioral Pediatrics*, 34, 1), 1–1), 8. <https://doi.org/10.1097/DBP.0b013e31827a7c3a>.
- Zeedyk, S. M., Rodriguez, G., Tipton, L. A., Baker, B. L., & Blacher, J. (2014). Bullying of youth with autism spectrum disorder, intellectual disability, or typical development: Victim and parent perspectives. *Research in Autism Spectrum Disorders*, 8(9), 1173–1183. <https://doi.org/10.1016/j.rasd.2014.06.001>.

Interdisciplinary Assessment and Intervention

► Role Release

Interdisciplinary Team

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Synonyms

Collaborative teaming model; Individualized education program (IEP) team; Individualized family services plan (IFSP) team; Multi-disciplinary team; Planning and placement team (PPT); Transdisciplinary team

Definition

An interdisciplinary team is a group comprised of specialists from multiple and varying backgrounds or fields and/or individuals with expertise or extensive knowledge about the individual, topic area, object, or other unit of analysis, for purposes of combining skills, resources, and information that will guide decisions and practices concerning the focus of study. Teams can be formed in health care, educational, counseling, and other settings concerning the needs of individuals with autism spectrum disorders. In cases of supports and services in educational settings for those with ASD from birth through 21 years of age, members of the planning and placement team (PPT) or IEP team, individualized family services plan (IFSP), and individualized education program (IEP) teams conduct, interpret, and present findings from evaluations conducted independently to the entire group for purposes of cooperation and collaboration in developing programs and planning instruction jointly for children with disabilities. From comprehensive assessments conducting in the medical setting to those included as part of a comprehensive evaluation process for special education eligibility determinations, an interdisciplinary team is necessary for providing the expertise in understanding outcomes, interpreting results, and educating other

team members on how to translate findings into practice and interventions. Since autism spectrum disorders affect multiple developmental domains, utilizing an interdisciplinary team constitutes best practice for a diagnosis of ASD and is an essential component of the assessment process as well as developing and implementing intervention plans. An interdisciplinary team promotes communication among team members and leads to a more integrated, cohesive translation of findings. A quality interdisciplinary process involves individual contributions to inform the construction of the overall picture of the individual with ASD; individual interpretations enable formulation of conclusions and recommendations based upon the combined efforts of the team.

See Also

- ▶ Eligibility (for Services Under IDEA/ADA, etc.)
- ▶ Free Appropriate Public Education
- ▶ Individualized Family Service Plan
- ▶ Least Restrictive Environment (LRE)
- ▶ Multidisciplinary Evaluation

References and Reading

- Connecticut State Department of Education. (2006). Steps to protect a child's right to special education: Procedural safeguards. Retrieved January 15, 2011, from http://www.sde.ct.gov/sde/lib/sde/pdf/DEPS/Special/Prosaf_fullversion.pdf.
- Connecticut State Department of Education. (2007). A parent's guide to special education. Retrieved December 15, 2010, from http://www.sde.ct.gov/sde/lib/sde/PDF/DEPS/Special/Parents_Guide_SE.pdf.
- Federal Education Project. (2010). Individuals with disabilities act funding distribution. Retrieved January 30, 2011, from <http://febp.newamerica.net/background-analysis/individuals-disabilities-education-act-funding-distribution>.
- <http://idea.ed.gov/explore/view/p/%2Croot%2Cdynamic%2CTopicalArea%2C1%2C>
- http://www.sde.ct.gov/sde/lib/sde/PDF/DEPS/Special/Guidelines_Autism.pdf
- IDEA Partnership. (2010). Autism spectrum disorders collection. Retrieved February 15, 2011, from http://www.ideapartnership.org/index.php?option=com_content&view=article&id=1493.

- National Association of Special Education Teachers. (2009). Assessment in special education series. Retrieved February 1, 2011, from <http://www.naset.org/2960.0.html>.
- Smith, D., & McGinnley, K. (2004). Side by Side comparison of Senate Bill 1248 (as passed on May 13, 2004) and House Bill 1340 (as passed April 30, 2003) and Parts A and B of IDEA, 1997. Retrieved from <http://www.wrightslaw.com/law/idea/sidebyside.06.04.pdf>.
- U.S. Department of Education, Office of Special Education Programs 10.04.06. (2006). Building the Legacy: IDEA 2004.
- Volkmar, F. R., Paul, R., Klin, A., & Cohen, D. *The handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know*. Hoboken: Wiley.
- Wright, P., & Wright, P. (2007). *Wrightslaw: Special education law* (2nd ed.). Hartfield: Harbor House Law Press.

Interests, Circumscribed/All Absorbing

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Definition

Interests characterized as “circumscribed” are included in the diagnosis of autism within the general area of restricted and repetitive behaviors

(RRBs). For example, in sign B3, DSM-5 describes “highly restricted, fixated interests that are abnormal in intensity or focus” and provides as examples “strong attachment to or preoccupation with unusual objects, excessively circumscribed or perseverative interest.” Unusually focused, intense, and persistent interests in autism may feature the amassing of large quantities of information and resistance to any diversion from their pursuit (Boyd et al. 2007). Importantly, what are called circumscribed interests in autism are associated with positive emotions and the potential for great satisfaction, thus contrasting with obsessions and compulsions, which are dreaded and troubling (Klin et al. 2007).

Pinpointing the concept of circumscribed interests and distinguishing it from related signs included in the general RRB area is however not easy. Indeed, the delineation between circumscribed interests and other RRB DSM-5 diagnostic signs is somewhat uncertain and arbitrary, with apparent redundancies and overlap across development. For instance, repetitive use of objects (sign B1) may be a form of attachment to or preoccupation with those objects (sign B3), as may be unusual interest in sensory aspects of the environment (including objects; sign B4). Highly restricted, fixated interests (sign B3) and stereotyped motor movements (sign B1) may both be described externally as insistence on sameness and inflexible adherence to routines (sign B2). Further, the word “interest” implies some kind of seeking behavior and positive emotions. Repetitive motor movements or lining up toys (sign B1) and visual fascination with lights or movement (sign B4) may be associated with positive emotions as well. In that sense, the distinction between, for example, persistently seeking out ceiling fans and looking laterally and happily to them, and the same behavior applied to finger movements, is arbitrary and both could be encompassed under circumscribed interests (sign B3).

Other divisions or subgroupings of RRBs have also been proposed. In the Autism Diagnostic Interview (ADI), circumscribed interests are distinguished from “unusual preoccupations.” The latter focuses on the odd or peculiar quality of the interest (e.g., ceiling fans), whereas the former

are also found in the nonautistic population (e.g., hockey statistics) but with different qualitative properties. The autistic quality of an autistic interest is related to its circumscribed nature, degree of focus, overtly or relatively nonsocial quality, and atypical progression over time, as well as to its being at odds with developmental level or context (Lord et al. 1994).

Another subgrouping of the RRB area attempts to distinguish “low-order” behaviors characterized by repetition of movement, associated with lower developmental levels and “simple” behaviors (e.g., gestural stereotypies), from “high-order” behaviors, associated with higher developmental levels and more complex behaviors (e.g., circumscribed interests; Turner 1999). However, both high-order (DSM-5 signs B2, B3) and low-order (signs B1, B4) behaviors share the qualities of interest and focus. This is why some studies collapse verbally/abstractly (circumscribed interests) and nonverbally/concretely (unusually strong attachment to certain objects or parts of objects) defined focused interests, independently of the verbal capabilities of the individual and his developmental level.

Historical Background

In Kanner’s first descriptions, the notion of circumscribed interest is present in the form of intense focus, expressed as repetition and insistence on sameness: “The repetitiousness assumes the form of obsessive preoccupations” (Kanner 1943). It is also positively described as an “excellent, purposeful, and intelligent relation to objects that do not threaten to interfere with their aloneness.” Robinson and Vitale (1954) independently described three individuals under the title “Children with circumscribed interest patterns” and emphasized precocity in knowledge as well as deleterious effect of interests on typical relationships with peers. Kanner’s first use of the term “circumscribed interests” was in his commentary on Robinson and Vitale’s work.

In 1980, the DSM-III description of autism included one mandatory RRB criterion: “Bizarre

responses to various aspects of the environment, for example, resistance to change, peculiar interest in or attachments to animate or inanimate objects.” The expanded 1987 DSM-III-R multiple-item RRB domain included both “persistent preoccupation with parts of objects” and “markedly restricted range of interests and a preoccupation with one narrow interest.” In 1994, this carried over into the DSM-IV attempt to distinguish implicitly verbal interests from concretely defined, implicitly nonverbal interests. Finally, the 2013 DSM-5 added a “sensory” component to the RRB area, encompassing unusual sensory interests.

Current Knowledge

Empirical Findings

A growing body of descriptive knowledge documents the range of autistic interests. In a retrospective compilation of Asperger’s clinical case records, of 44 children diagnosed with “autistic psychopathy” for whom information was available, 82% had “special, original and narrow interests” (Hippler and Klicpera 2003, p. 297; see also Klin et al. 2007, as well as Gilchrist et al. 2001, for similar findings). For 33 of these children, domains of interest were categorized as animals and nature (30%), technical and/or scientific interests (27%), obsessive reading/collecting facts (24%), public transport systems (18%), religion (12%), drawing (12%), music (9%), and space/astronauts (6%). Specific unusual interests included eye muscles, rubbish bins, earthworms, and “inventing own methods for calculation.”

An investigation among the parents of 61 autistic individuals indicated that the most frequent materials or areas of information involved in circumscribed interests were Japanese animation and cartoon characters, electric and mechanical apparatus, dinosaurs, space/physics, natural disasters, historical events, encyclopedias/fact books, videogames, technical manuals, religion or politics, reptiles, rodents, printed material in general, television in general, and facts (South et al. 2005). In a survey of 92 parents, “obsessional interests” in autistics were reported in broad categories

including physics (84%), taxonomy (73%), and TV/audio (64%; Baron-Cohen and Wheelwright 1999). Another survey conducted among the caregivers of a large population of fluently speaking autistic individuals revealed that circumscribed interests frequently involve memorization of facts expressed verbally, including an early interest in letters and numbers (Klin et al. 2007). Eight categories of activities were observed, including two types of fact collection, verbal memory (e.g., naming makes/models of cars) and visual memory with associated activities (e.g., making LEGO dragons). The remaining six categories were sensory behaviors (e.g., lining up objects), math (e.g., naming prime numbers), ordering information (e.g., classifying insects), dates and time (e.g., memorizing birthdays), hoarding (e.g., collecting Frisbees), and letters/numbers (e.g., decoding).

Circumscribed interests may change with development, from early interest for parts of objects (Gilchrist et al. 2001; Richler et al. 2010) and letters and numbers (Ostrolenk et al. 2017) to more complex interests in older individuals (Klin et al. 2007). When longitudinal data are available, this gradual increment is evident even at the individual level (South et al. 2005). The birth of circumscribed interests is understudied but in some cases can be traced back to a unique encounter with a specific material (e.g., Mottron et al. 2006). Circumscribed interests are also associated with higher apparent IQ (Bishop et al. 2006), as well as with typical or precocious speech development, although this may trivially depend on the overlap between the definition of circumscribed interests and speech abilities (Lam et al. 2008). Specificity toward intellectual disability is high (Bodfish et al. 2000), but not toward exceptional typical individuals, at least when it comes to importance of personal investment and association with higher intelligence (DeLoache et al. 2007).

Although never studied in a systematic way, circumscribed interests seem highly associated with savant abilities, given that any reported savant ability involves a circumscribed interest in the relevant domain (Young 1995). Interest can be ascertained through overt interacting behaviors with a specific material. These

behaviors result in the cognitive manipulation of units composing this material, which favors a “frequency effect,” that is, the faster recognition and stronger memorization of the material involved in the savant ability (Mottron et al. 1996). In this sense, focused intense (ergo “circumscribed”) interests may represent the autistic equivalent of the ensemble of behaviors and cognitive modifications associated with *expertise* in nonautistics.

Adaptive properties of circumscribed interests are considered in contrasting ways among researchers. Circumscribed interests may represent the ways in which autistics learn well (Dawson et al. 2008) and are associated with positive emotions across the lifespan (Mercier et al. 2000; Smerbeck 2017). They constitute a heuristic tool to understand the world (Klin et al. 2007), foster skill development and communication (Winter-Messiers 2007), and become the foundation for higher education and employment in adulthood (Patten-Koenig and Hough-Williams 2017). Conversely, they may be regarded as behaviors which require the largest accommodation in parents of autistics (Gabriels et al. 2005), and most interfere with other activities: a large portion of an autistic’s free time is overtly dedicated to their intense interests. The extent to which such interests are judged as “interfering” in preschool and early school years predicts Vineland adaptive behavior scores later in adolescence (Klin et al. 2007).

The overlap or autonomy of circumscribed interests with or toward other RRBs in autism can also be studied through factorization of ADI RRB items, although the relative lack of precision of this tool for the repetitive area limits the informative value of these studies. Whereas factorization studies among RRBs consistently oppose a “repetitive sensory and motor behaviors” factor to an “insistence on sameness” factor, circumscribed interests stand as a distinct factor (Lam et al. 2008) and do not correlate with participant characteristics, supporting its conceptual autonomy.

Neurobiological counterparts of circumscribed interests are found in studies of genetics and brain functioning. Circumscribed interests are partially shared by parents in the form of intense

preoccupations (Hippler and Klicpera 2003; Smith et al. 2009). Involvement of the amygdala when an autistic interacts with the subject of a circumscribed interest has been demonstrated through fMRI (Grelotti et al. 2005). In the case of hyperlexia, a label which groups focused interest in written materials with exceptional abilities in decoding text, autistic expertise is associated with a functional cortical rededication of brain areas involved in reading (Turkeltaub et al. 2004). An involvement of synesthesia has also been suspected in the choice of domain of interest as well as on the nature of mental manipulation operations performed on this material (Motttron et al. 2013).

Theoretical Accounts

There is agreement on the fact that RRBs have a certain kind of etiological and phenotypical autonomy from sociocommunicative behaviors in autism, preventing a straightforward explanation of one by the other (Happé et al. 2006; Richler et al. 2010). Their relative magnitude is not correlated according to current measures, while their distribution in the general population and their developmental profile differ. Available theoretical accounts of circumscribed interests include those which do or do not posit a causal deficit. One account originated with the overt resemblance between perseveration in frontally injured patients and the constancy autistics demonstrates in the pursuit of their circumscribed interest. This led to the proposal of an executive dysfunction account of circumscribed interests (Turner 1999). However, autistic individuals do not consistently display impairments in classic executive tasks, which questions how “executive” the purported deficit is.

Other accounts of circumscribed interests invoke the atypical strength or hyperfunctioning of a cognitive substructure. Two models hypothesize that atypical perception – understood as resulting from diminished top-down processes (weak central coherence; Happé and Vital 2009) or as enhanced perceptual functioning (Motttron et al. 2009) – is involved in the nature and processing of materials which are the object of autistic interests.

According to the EPF model, intense focused interests are the behavioral and cognitive counterpart of enhanced pattern detection and manipulation along autistic development. This is manifested, among other behaviors, by a spontaneous orientation toward genuine regularities loading a domain of interest. Materials involved in circumscribed interests, as well as in savant syndrome, often consist of ubiquitous human codes (e.g., written language in various formats, music, numeration, and three-dimensional graphic representations) rich in regularities across multiple scales, from simple units to overarching structures. Thus, there may be veridical mapping between isomorphic structures through pattern detection mechanisms, which are especially active in autism. In another account, autistics are characterized by *hypersystemizing*. Systemizing is described as detecting and applying strict, inflexible rules or laws, such as those of “if p, then q” form (Baron-Cohen et al. 2009). Narrow or circumscribed interests thus result from the rigidity of hypersystemizing. Whatever the model, there is in the last decade a belated trend toward envisioning circumscribed interests more accurately in autism research, for the benefit they bring to the well-being of the person. This includes preliminary steps toward studying how autistic interests may be harnessed in early intervention, in education, in employment, and in finding a social niche.

Conclusion

The importance of what are called circumscribed interests in autism research, and their appreciation as a major organizer of quality of life for autistics, has fluctuated over the years. However, the area as a whole remains understudied relative to its importance to both autistic and nonautistic individuals and relative to its potential to cast a light on the fundamental nature of autistic cognition. More accurate, less pathologized, and less biased views of autistic interests also offer new perspectives for the integration of autistic people in a modern and open society.

Future Directions

- What role does availability of information play in the development, nature, narrowness, and outcomes of autistic interests?
- To what extent and under what circumstances are autistic interests truly restricted or circumscribed?
- Under which circumstances do autistic interests lead to very good or exceptional outcomes?
- Is there a critical period for intense focused interests in autism, or can they develop across the autistic lifespan, given the opportunity?
- Does the presence or absence of speech delay modify the nature and course of circumscribed interests in autism?
- What is the association between circumscribed interests and the acquisition of literacy in autistic individuals?

See Also

- [Enhanced Perceptual Functioning](#)
- [Savant Syndrome](#)
- [Systemizing](#)

References and Reading

- Baron-Cohen, S., & Wheelwright, S. (1999). 'Obsessions' in children with autism or Asperger syndrome. Content analysis in terms of core domains of cognition. *The British Journal of Psychiatry*, 175(5), 484–490.
- Baron-Cohen, S., Ashwin, E., Ashwin, C., Tavassoli, T., & Chakrabarti, B. (2009). Talent in autism: Hyper-systemizing, hyper-attention to detail and sensory hypersensitivity. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 364(1522), 1377–1383.
- Bishop, S. L., Richler, J., & Lord, C. (2006). Association between restricted and repetitive behaviors and nonverbal IQ in children with autism spectrum disorders. *Child Neuropsychology*, 12(4–5), 247–267.
- Bodfish, J. W., Symons, F. J., Parker, D. E., & Lewis, M. H. (2000). Varieties of repetitive behavior in autism: Comparisons to mental retardation. *Journal of Autism and Developmental Disorders*, 30(3), 237–243.
- Boyd, B. A., Conroy, M. A., Mancil, G. R., Nakao, T., & Alter, P. J. (2007). Effects of circumscribed interests on the social behaviors of children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(8), 1550–1561.
- Dawson, M., Mottron, L., & Gernsbacher, M. A. (2008). Learning in autism. *Learning and memory: A comprehensive reference*, 2, 759–772.
- DeLoache, J. S., Simcock, G., & Macari, S. (2007). Planes, trains, automobiles – and tea sets: Extremely intense interests in very young children. *Developmental Psychology*, 43(6), 1579.
- Gabriels, R. L., Cuccaro, M. L., Hill, D. E., Ivers, B. J., & Goldson, E. (2005). Repetitive behaviors in autism: Relationships with associated clinical features. *Research in Developmental Disabilities*, 26(2), 169–181.
- Gilchrist, A., Green, J., Cox, A., Burton, D., Rutter, M., & Le Couteur, A. (2001). Development and current functioning in adolescents with Asperger syndrome: A comparative study. *The Journal of Child Psychology and Psychiatry and Allied Disciplines*, 42(2), 227–240.
- Grelotti, D. J., Klin, A. J., Gauthier, I., Skudlarski, P., Cohen, D. J., Gore, J. C., . . . Schultz, R. T. (2005). fMRI activation of the fusiform gyrus and amygdala to cartoon characters but not to faces in a boy with autism. *Neuropsychologia*, 43(3), 373–385.
- Happé, F., & Vital, P. (2009). What aspects of autism predispose to talent? *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 364(1522), 1369–1375.
- Happé, F., Ronald, A., & Plomin, R. (2006). Time to give up on a single explanation for autism. *Nature Neuroscience*, 9(10), 1218–1220.
- Hippler, K., & Klicpera, C. (2003). A retrospective analysis of the clinical case records of 'autistic psychopaths' diagnosed by Hans Asperger and his team at the University Children's Hospital, Vienna. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 358(1430), 291–301.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2(3), 217–250.
- Klin, A., Danovitch, J. H., Merz, A. B., & Volkmar, F. R. (2007). Circumscribed interests in higher functioning individuals with autism spectrum disorders: An exploratory study. *Research and Practice for Persons with Severe Disabilities*, 32(2), 89–100.
- Lam, K. S., Bodfish, J. W., & Piven, J. (2008). Evidence for three subtypes of repetitive behavior in autism that differ in familiarity and association with other symptoms. *Journal of Child Psychology and Psychiatry*, 49(11), 1193–1200.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism Diagnostic Interview-Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(5), 659–685.
- Mercier, C., Mottron, L., & Belleville, S. (2000). A psychosocial study on restricted interests in high

- functioning persons with pervasive developmental disorders. *Autism*, 4(4), 406–425.
- Mottron, L., Belleville, S., & Stip, E. (1996). Proper name hypermnesia in an autistic subject. *Brain and Language*, 53(3), 326–350.
- Mottron, L., Lemmens, K., Gagnon, L., & Seron, X. (2006). Non-algorithmic access to calendar information in a calendar calculator with autism. *Journal of Autism and Developmental Disorders*, 36(2), 239–247.
- Mottron, L., Dawson, M., & Soulières, I. (2009). Enhanced perception in savant syndrome: Patterns, structure and creativity. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 364(1522), 1385–1391.
- Mottron, L., Bouvet, L., Bonnel, A., Samson, F., Burack, J. A., Dawson, M., & Heaton, P. (2013). Veridical mapping in the development of exceptional autistic abilities. *Neuroscience & Biobehavioral Reviews*, 37(2), 209–228.
- Ostrolenk, A., d’Arc, B. F., Jelenic, P., Samson, F., & Mottron, L. (2017). Hyperlexia: Systematic review, neurocognitive modelling, and outcome. *Neuroscience and Biobehavioral Reviews*, 79, 134.
- Patten Koenig, K., & Hough Williams, L. (2017). Characterization and utilization of preferred interests: A survey of adults on the autism spectrum. *Occupational Therapy in Mental Health*, 33(2), 129–140. <https://doi.org/10.1080/0164212X.2016.1248877>.
- Richler, J., Huerta, M., Bishop, S. L., & Lord, C. (2010). Developmental trajectories of restricted and repetitive behaviors and interests in children with autism spectrum disorders. *Development and Psychopathology*, 22(1), 55–69.
- Robinson, J. F., & Vitale, L. J. (1954). Children with circumscribed interest patterns. *American Journal of Orthopsychiatry*, 24(4), 755–766.
- Smerbeck, A. (2017). The Survey of Favorite Interests and Activities: Assessing and understanding restricted interests in children with autism spectrum disorder. *Autism*. <https://doi.org/10.1177/1362361317742140>.
- Smith, C. J., Lang, C. M., Kryzak, L., Reichenberg, A., Hollander, E., & Silverman, J. M. (2009). Familial associations of intense preoccupations, an empirical factor of the restricted, repetitive behaviors and interests domain of autism. *J Child Psychol Psychiatry*, 50(8), 982–990. <https://doi.org/10.1111/j.1469-7610.2009.02060.x>.
- South, M., Ozonoff, S., & McMahon, W. M. (2005). Repetitive behavior profiles in Asperger syndrome and high-functioning autism. *Journal of Autism and Developmental Disorders*, 35(2), 145–158.
- Turkeltaub, P. E., Flowers, D. L., Verbalis, A., Miranda, M., Gareau, L., & Eden, G. F. (2004). The neural basis of hyperlexic reading: An fMRI case study. *Neuron*, 41(1), 11–25.
- Turner, M. (1999). Annotation: Repetitive behaviour in autism: A review of psychological research. *The Journal of Child Psychology and Psychiatry and Allied Disciplines*, 40(6), 839–849.
- Winter-Messiers, M. A. (2007). From tarantulas to toilet brushes: Understanding the special interest areas of children and youth with Asperger syndrome. *Remedial and Special Education*, 28(3), 140–152.
- Young, R. R. L. (1995). *Savant syndrome: processes underlying extraordinary abilities/Robyn Young*. Doctoral dissertation.

Interhemispheric White Matter Tract

► Corpus Callosum

Internalization of Emotion Co-regulatory Support in Children with ASD

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Adaptive emotion regulation involves the ability to understand, monitor, and modify internal states in order to achieve one’s goals (Thompson 1994). Although infants demonstrate certain behaviors thought to assist with the regulation of arousal (e.g., gaze aversion, thumb-finger sucking), it is generally agreed that very young children are primarily “co-regulated” by their caregivers (Cole et al. 1994; Kopp 1982). Over the first few years of life, more direct co-regulation (e.g., responsive comforting, instrumental support, emotion contagion) gradually gives way to parental efforts aimed at helping children become more autonomous in their regulatory abilities (Cole et al. 1994; Grolnick and Farkas 2002; Kopp 1982; Morris et al. 2007). Although more general patterns of parent or family behavior continue to contribute to the development of children’s emotion regulation throughout childhood, parents increasingly engage in more didactic socialization strategies, such as intentional modeling and what

is commonly referred to as either emotion “coaching” or “scaffolding” (Baker et al. 2011; Gottman et al. 1996; Morris et al. 2007).

Parental environments characterized by responsive involvement, sensitive structuring, and support for child autonomy are conceptualized as facilitating a shift from external co-regulation to internal regulatory control (Grolnick and Farkas 2002), with parenting contributing critically to underlying biobehavioral processes (Beauchaine et al. 2007; Cole et al. 1994). Internalization of co-regulatory supports is suggested when a child begins to demonstrate autonomous use of strategies or competencies that were originally promoted in the context of external (e.g., parent) support (Baker et al. 2019; Cole et al. 1994; Grolnick and Farkas 2002; Kopp 1982). Theoretical accounts of internalization processes related to the regulation of emotion suggest that great strides in self-regulation occur during the preschool years (Cole et al. 1994; Kopp 1982). Although clear empirical evidence for age markers related to the development of emotion regulation in children is lacking, it is generally assumed and expected that most children demonstrate a reasonable level of self-regulation by middle childhood (Cole et al. 1994).

Children with autism spectrum disorder (ASD), as a group, experience considerable difficulty in regulating their emotions, as demonstrated by higher levels of behavior indicative of emotion dysregulation, use of fewer strategies presumed to be helpful for regulation, and reduced effectiveness of such strategies as compared to peers with neurotypical development (Jahromi et al. 2012; Samson et al. 2015). The longitudinal course of the development of emotion regulation in children with ASD requires empirical study. However, it is clear that many children with ASD continue to exhibit significant emotion dysregulation well into the elementary school years (e.g., Fenning et al. 2018; Jahromi et al. 2012) and at least one cross-sectional study did not find lower dysregulation as a function of higher child age in a sample of 4- to 11-year-olds with ASD (Fenning et al. 2018). This apparent delay in the development of autonomous

regulatory competence in children with ASD may be accompanied by (or potentially a product of) a delay in the internalization of caregiver co-regulatory support. Indeed, Ting and Weiss (2017) did not find any association between parent-child discussion and regulation strategies generated independently by school-aged children with ASD. Fenning et al. (2018) reported that parental co-regulatory scaffolding was related *in the moment* to the quality of their children’s emotion regulation during a frustrating task, but that scaffolding was not significantly associated with individual differences in children’s regulation during an *independent* task, suggesting effective co-regulation but the absence of internalization of the support.

The cross-sectional data from Fenning et al. (2018) were re-analyzed to specifically address age differences in the apparent internalization of co-regulatory support. Baker et al. (2019) hypothesized that increased internalization would be observed at higher child ages, as indexed by stronger consistency in dysregulation scores across parent-child and independent child tasks and a stronger inverse association between parental scaffolding during the dyadic task and child dysregulation during the independent task. Indeed, higher consistency across tasks was observed for the older as compared to the younger children in the sample of children ages 4–11 years. In addition, the association between parental scaffolding and children’s independent dysregulation became increasingly negative at older child ages; however, even at the higher ages, the association was only significant at a trend level, suggesting that internalization may not be observed until late childhood or even early adolescence in children with ASD.

While the apparent internalization of co-regulatory supports in children with ASD may depend upon child chronological (but perhaps not mental) age (Baker et al. 2019), the mechanisms accounting for this potential group delay or individual differences in the development of these processes remain unknown. Neurodevelopmental differences associated with the core features of ASD (e.g., reduced social awareness, cognitive and behavioral inflexibilities) are

thought to contribute significantly to the regulation difficulties experienced by these children (Fenning et al. 2018; Mazefsky and White 2014) and may similarly impact relevant internalization processes. Although environmental factors are unlikely to account for the status-group difference, certain parenting behaviors may contribute to individual differences in internalization processes within this population. For example, Hirschler-Guttenberg et al. (2015) reported that maternal authoritarian parenting was associated with limited seeking of parental co-regulatory support in preschoolers with ASD.

There is clear evidence that children with ASD, as a group, experience difficulties in their ability to regulate emotions. What is desperately needed is information relating to the developmental course of emotion regulation in these children, including the identification of not only what might promote beneficial trajectories but also when and how these influences contribute. The apparent lack of concordance between parent co-regulatory support and children's independent regulatory competence during the early school years is concerning, and, should these early findings be replicated, understanding the degree to which alternative parenting behaviors or scaffolding supports are beneficial as children with ASD age may be key to intervention efforts aimed at promoting regulatory competence in this population.

See Also

- [Predictors of Emotion Regulation in Children with Autism Spectrum Disorder \(ASD\)](#)

References and Reading

- Baker, J. K., Fenning, R. M., & Cmic, K. A. (2011). Emotion socialization by mothers and fathers: Coherence among behaviors and associations with parent attitudes and children's social functioning. *Social Development, 20*, 412–430. <https://doi.org/10.1111/j.1467-9507.2010.00585.x>.
- Baker, J. K., Fenning, R. M., & Moffitt, J. (2019). Brief report: A cross-sectional examination of the internalization of emotion co-regulatory support in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*. Online ahead of print. <https://doi.org/10.1007/s10803-019-04091-0>.
- Beauchaine, T. P., Gatzke-Kopp, L., & Mead, H. K. (2007). Polyvagal theory and developmental psychopathology: Emotion dysregulation and conduct problems from preschool to adolescence. *Biological Psychology, 74*, 174–184. <https://doi.org/10.1016/j.biopsycho.2005.08.008>.
- Cole, P. M., Michel, M. K., & Teti, L. O. (1994). The development of emotion regulation and dysregulation: A clinical perspective. *Monographs of the Society for Research in Child Development, 59*(2/3), 73–100.
- Fenning, R. M., Baker, J. K., & Moffitt, J. (2018). Intrinsic and extrinsic predictors of emotion regulation in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 48*, 3858–3870. <https://doi.org/10.1007/s10803-018-3647-1>.
- Gottman, J. M., Katz, L. F., & Hooven, C. (1996). Parental meta-emotion philosophy and the emotional life of families: Theoretical models and preliminary data. *Journal of Family Psychology, 10*, 243–268. <https://doi.org/10.1037/0893-3200.10.3.243>.
- Grolnick, W. S., & Farkas, M. (2002). Parenting and the development of children's self-regulation. In M. H. Bornstein (Ed.), *Handbook of parenting: Practical issues in parenting* (pp. 89–110). Mahwah: Lawrence Erlbaum Associates Publishers.
- Hirschler-Guttenberg, Y., Feldman, R., Ostfeld-Etzion, S., Laor, N., & Golan, O. (2015). Self- and co-regulation of anger and fear in preschoolers with autism spectrum disorder: The role of maternal parenting style and temperament. *Journal of Autism and Developmental Disorders, 45*, 3004–3014. <https://doi.org/10.1007/s10803-015-2464-z>.
- Jahromi, L. B., Meek, S. E., & Ober-Reynolds, S. (2012). Emotion regulation in the context of frustration in children with high functioning autism and their typical peers. *Journal of Child Psychology and Psychiatry, 53*(12), 1250–1258. <https://doi.org/10.1111/j.1469-7610.2012.02560.x>.
- Kopp, C. B. (1982). Antecedents of self-regulation: A developmental perspective. *Developmental Psychology, 18*(2), 199–214.
- Mazefsky, C., & White, S. (2014). Emotion regulation: Concepts & practice in autism spectrum disorder. *Child and Adolescent Psychiatric Clinics of North America, 23*, 15–24. <https://doi.org/10.1016/j.chc.2013.07.002>.
- Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Social Development, 16*(2), 361–388. <https://doi.org/10.1111/j.1467-9507.2007.00389.x>.
- Samson, A. C., Hardan, A. Y., Podell, R. W., Phillips, J. M., & Gross, J. J. (2015). Emotion regulation in children and adolescents with autism spectrum disorder. *Autism Research, 8*(1), 9–18. <https://doi.org/10.1002/aur.1387>.

- Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development*, 59, 25–52. <https://doi.org/10.1111/j.15405834.1994.tb01276.x>.
- Ting, V., & Weiss, J. A. (2017). Emotion regulation and parent coregulation in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(3), 680–689. <https://doi.org/10.1007/s10803-016-3009-9>.

- The downloading of unwanted files such as viruses or illegally downloaded music
- An increased chance of becoming a target of a potential abuser, stalker or pedophile
- Behaving inappropriately on social network sites
- Spending too much time video gaming with others
- The obsessive collecting of data and images

Internet Safety

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Definition

The Internet has provided people around the world with opportunities to find information and to meet people at the click of a button. Studies have even shown that use of the Internet can increase academic performance and certain other skills (Packard 2007). Despite the benefits however, the Internet can bring about risks, especially for those on the autism spectrum. Some of these risks include the following:

- An increased chance of being bullied
- Inadvertent sharing of personal information such as name, address, phone number, and social security numbers making someone vulnerable to identity theft
- The chance of viewing of inappropriate information or pictures, either violent or pornographic
- On-line sexual activity which can become addictive
- The possibility of unintended contact with militant groups
- The ability to purchase items, both legal and illegal
- The possibility of on-line gambling and possible addiction to virtual casinos due to compulsive behaviors

Historical Background

Studies have consistently shown that people with special needs are at a greater risk of being bullied than their peers (Baumeister et al. 2008). These autistic individuals are set apart as different, and they have characteristics that isolate them from others and make them targets. In one study of children with autism, rates of bullying were over 44% (Montes and Halterman 2007). Children and adolescents with Asperger's syndrome showed increased rates of being a victim of bullying (Carter 2009) with self-reported rates of 75% in one study (Little 2001). Higher rates of verbal and physical attacks are believed to arise from difficulties with social interaction and the failure to read social cues which is a trait of those on the spectrum (American Psychiatric Association 2000). This makes them more likely to be bullied, as they are perceived as odd and different. The frequency of aggressive behaviors in spectrum disorders also increases the chance that they might perpetrate bullying behaviors.

Current Knowledge

Cyber bullying is the result of technology, such as e-mail, instant messaging, cell phone texts, or social network sites being used as a tool for bullying (Kowalski and Limber 2007). There has been little research on cyber bullying and spectrum disorders, but one study that examined the prevalence of cyber bullying via the Internet found that 7% of children aged 12–19 reported being cyber bullied via the Internet and 4% said they had been cyber bullied through text

messaging (Didden et al. 2009). Under 50% of victims of cyber bullying report not knowing the identity of their abuser, which clearly differentiates cyber bullying from traditional bullying as people tend to say and do things anonymously that they would not do in a one-on-one interaction (Kowalski et al. 2008). This can also occur any time of day or night which leaves victims feeling extremely vulnerable. For those on the spectrum, the fear of having technology taken away from them by their parents makes them reluctant to report this bullying to an authority figure. For children with special needs, the Internet might be an easier way of socializing with peers because of their social skills deficits and lack of empathy, yet will likely lead to problems in the virtual world and increased likelihood that they will be involved in cyber bullying. Children who have reported on-line harassment have been found to have less developed social skills (Shea and Wiener 2003). In a study conducted in 2011 by Kowalski and Fedina, over 21% of those surveyed indicated they had been cyber bullied within the past 2 months, while just under 6% said they had perpetrated cyber bullying at least once within the same time period. Parents in this study seemed rather uninformed about their children's experiences with cyber bullying. Seventy-three percent said their child had never been cyber bullied and another 12% reported that they did not know. Twelve percent indicated that they did not know if their child had perpetrated cyber bullying. Eighty-five percent claimed that their child had never cyber bullied anyone, yet only 9% of the parents said that they had ever discussed cyber bullying with their child despite the fact that 51% of the parents acknowledged that they knew something about the topic. The most common means of cyber bullying reported was through instant messaging (66.7%) with social networking sites coming in second (60%). A large percentage of those who were cyber bullied indicated that the perpetrator was a friend (50%) and 37.5% indicating the perpetrator was another student at school. Twenty-five percent said they did not know who the perpetrator was. The results of this bullying often lead to anxiety, depression, and low self-esteem.

While being bullied on the Internet is certainly an issue of concern, it is becoming more common to read about sexual predators finding their victims on the net. Teens especially will use their computers late at night, unmonitored by a parent, and are more likely to come across sex abusers looking for victims (Dennis Debbaudt, *Safety Issues for Adolescents with Asperger Syndrome* 2003). While often, parents are pleased that their child has "a friend," the concern for the type of friend is absent and the very literalness of those on the spectrum puts this population in danger.

The FTC stated that identity theft is the number one complaint they get and has continually increased by 20% each year. On the Internet, this can occur by using unsecured websites and divulging information such as social security numbers. It is of utmost importance that individuals with autism understand the importance of using secure websites when engaging in on-line shopping or whenever using a credit card on the Internet. This needs to be explained as making sure that the URL of the website starts with `https://` not `http://`. That extra "s" is what signifies the website as "secure." It is also imperative that autistic individuals understand the types of information that should not be shared on the Internet. It is not uncommon to discover that many people with autism have unknowingly purchased subscriptions to magazines, web sites, and other web services due to their impulsivity and lack of discipline in reading the terms and conditions that often come with agreements.

Due to individuals on the autism spectrum's impulsivity, they are unlikely to think ahead of possible consequences or their behaviors. They also have a tendency to become obsessed with things rather quickly. This is often what occurs when an autistic person, often by accident through a misdirected search using a word such as "toy," a misspelled URL, a misleading web site or e-mail, or a link or photo sent by a peer or through spam, finds pornography on the Internet (Wolak et al. 2007).

While many believe spending time playing video games can be a waste of time, it has been found that these games help children with autism learn to cope with noise, regulate their balance,

and improve sensory processing (www.newswise.com). Vision Audio, Inc., has created EASe (Electronic Auditory Stimulation effect) video games and audio CD's that are therapeutic tools for children on the autism spectrum disorder and those diagnosed with auditory hypersensitivity, central auditory processing disorder, or sensory integration disorder. Therapists have clinically tested the nonviolent EASe video games since 2007 with positive results. The sensory events in the virtual world of these games eventually create a repertoire of experiences that help the autistic child learn to cope with similar events in the real world.

Future Directions

Interventions

Being concrete and establishing rules for social networking sites is particularly helpful for someone on the spectrum. Talking with the individual and explaining clearly what each rule means is extremely important. Rules should be posted near the computer for a constant reminder of appropriate behaviors such as the following:

1. Talk only with friends.
2. Keep address, phone number, social security number, and banking information secret.
3. Talk to a parent or guardian when someone you do not know tries to talk to you.
4. Think before posting comments. Ask yourself if anyone is going to care before you start typing. Will the person at the other end understand if you are joking?
5. Be careful about posting photos. Make sure all photos are appropriate for viewing and also that other people who may be in the photo are okay with their pictures being posted as well.
6. Do not do anything online that you would not do in real life.

There are also Internet safety symbols created by Widget Software that can be downloaded at <http://www.childnet.com/kia/sen/widgetsmart.aspx>. Each symbol is hand-drawn, clear, and concise and illustrates a single concept without

adding unnecessary information. (<http://www.childnet.com>).

It is crucial that the individual with autism understands the consequences of inappropriate behaviors. For a younger person or teen, it might be losing computer privileges with the understanding that correcting these behaviors will reverse this consequence in time. For someone who is an adult, it might be explaining that their behaviors could bring about danger to the family and this may result in their being asked to find another place to live or having their employment terminated if they are an employed adult.

Sometimes being this concrete is what is necessary for the individual with autism to understand that conforming to social norms is necessary.

As a parent or caregiver, there are other precautions that can be taken when the child with an autism spectrum disorder claims to have met a "friend" on-line such as something known as "time checking." This is when the correspondent holds up a newspaper with the date and time (not unlike a hostage displaying a newspaper), takes a picture of him or herself and emails it back immediately. This instant validation process makes it very difficult for an on-line predator to operate anonymously (<http://www.autismkey.com/internet-safety-and-your-child-with-autism/>). Installing software such as NetDog Filter (<http://www.netdogsoft.com>), will block all porn websites in the background when your child is on the Internet and protect them from stumbling into pornography by accident. For young children, there is a Disney game called Surf Swell Island. It teaches basic safety rules and it is free to download at <http://disney.go.com/surfswell/index.html>. The questions are read out loud by the narrator, but the child has to be able to read and choose the answer. The child goes through four areas (Privacy Falls, Virus Cave, Temple of Tact, and Challenge of Doom).

Other options are social networking sites specifically for individuals with autism (<http://www.autismspeaks.org>) such as:

WeAreAutism.org – Share, talk, and communicate in a user-led social network for individuals,

family members, and those members of the community. Share the wealth of your experience and plan for the future with those like you. Find others with similar interests and goals. WrongPlanet.net – Wrong Planet is a web community designed for individuals (and parents/professionals of those) with autism, Asperger's syndrome, ADHD, PDDs, and other neurological differences. They provide a discussion forum, where members communicate with each other; an article section, with exclusive articles and how-to guides; a blogging feature; and a chat room for real-time communication with other Aspies. AutismSpeaks.Ning.org – Autism Speaks Social Networking Site. This online community was created as a support forum for those affected by autism. WeAreAutism.org – Share, talk, and communicate in a user-led social network for individuals, family members and those members of the community. Share the wealth of your experience and plan for the future with those like you. Find others with similar interests and goals. WrongPlanet.net – Wrong Planet is a web community designed for individuals (and parents/professionals of those) with Autism, Asperger's Syndrome, ADHD, PDDs, and other neurological differences. They provide a discussion forum, where members communicate with each other; an article section, with exclusive articles and how-to guides; a blogging feature; and a chat room for real-time communication with other Aspies. AutismSpeaks.Ning.org – Autism Speaks Social Networking Site. This online community was created as a support forum for those affected by autism. Installing anti-virus software is critical for Internet security and safety. Some highly rated software used in removing viruses, malware and spyware include: McAfee, F-Secure, Norton, Kaspersky, and TrendMicro.

See Also

- ▶ [Asperger Syndrome Epidemiology](#)
- ▶ [Autism](#)
- ▶ [Bullying](#)
- ▶ [Figurative Language](#)
- ▶ [Friendships](#)
- ▶ [Interpersonal Skills](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Bacchini, D., Affuso, G., & Trotta, T. (2008). Temperament, ADHD, and peer relations among schoolchildren: The mediating role of school bullying. *Aggressive Behavior, 34*, 447–459.
- Baumeister, A. L., Storch, E. A., & Gefken, G. R. (2008). Peer victimization in children with learning disabilities. *Child and Adolescent Social Work Journal, 25*, 11–25.
- Carter, S. (2009). Bullying of students with Asperger's syndrome. *Issues in Comprehensive Pediatric Nursing, 32*, 145–154.
- Didden, R., Scholte, R., Korzilius, H., DeMoor, J., Vermeulen, A., O'Reilly, M., et al. (2009). Cyberbullying among students with intellectual and developmental disability in special education settings. *Developmental Neurorehabilitation, 12*, 146–151.
- <http://www.autismkey.com/internet-safety-and-your-child-with-autism>
- <http://www.childnet.com/kia/sen/widgetsmart.aspx>
- <http://www.easecd.com>
- http://www.pcworld.com/article/169120/facebook_etiquette_10_rules_for_better_socializing.html
- Humphrey, N., & Lewis, S. (2008). What does “inclusion” mean for pupils on the autistic spectrum in mainstream schools secondary schools. *Journal of Research in Special Education Needs, 8*, 132–140.
- Humphrey, N., & Symes, W. (2010). Perceptions of social support and experience of bullying among pupils with autistic spectrum disorders in mainstream secondary schools. *European Journal of Special Needs Education, 25*, 77–91.
- Kowalski, R. M., & Fedina, C. (2011). Cyber bullying in ADHA and Asperger syndrome populations. *Research in Autism Spectrum Disorders, 5*, 1201–1208.
- Kowalski, R. M., & Limber, S. (2007). Electronic bullying among middle school students. *Journal of Adolescent Health, 41*, S22–S30.
- Kowalski, R., Limber, S., & Agatston, P. (2008). *Cyber bullying: Bullying in the digital age*. Malden: Blackwell Publishing.
- Little, L. (2001). Research in autism spectrum disorders. Peer victimization of children with Asperger spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*, 995–996.
- Montes, G., & Halterman, J. S. (2007). Bullying among children with autism and the influence of comorbidity with ADHD: A population-based study. *Ambulatory Pediatrics, 7*, 253–257.
- Packard, E. (2007). It's fun, but does it make you smarter? *Monitor on Psychology, 38*(10), 44–46.
- Shea, B., & Wiener, J. R. (2003). Social exile: The cycle of peer victimization for children with ADHD. *Canadian Journal of School Psychology, 18*, 55–91.
- Taylor, L. A., Saylor, C., Twyman, K., & Marcias, M. (2010). Adding insult to injury: Bullying

- experiences of youth with attention deficit hyperactivity disorder. *Children's Health Care*, 39, 59–72.
- Van Cleave, J., & Davis, M. (2006). Bullying and peer victimization among children with special health care needs. *Pediatrics*, 118, 1212–1219.
- Van Roekel, E., Scholte, R. H. J., & Didden, R. (2010). Bullying among adolescents with autism spectrum disorders: Prevalence and perception. *Journal of Autism and Developmental Disorders*, 40, 63–73.
- Wiener, J., & Mak, M. (2009). Peer victimization in children with attention-deficit/hyperactivity disorder. *Psychology in the School*, 46, 116–131.
- Wolak, J., Mitchell, K., & Finkelhor, D. (2006). *Online victimization of youth: Five years later* (pp. 1–96). Alexandria: National Center for Missing & Exploited Children.
- Wolak, J., Mitchell, K., & Finkelhor, D. (2007). Does online harassment constitute bullying? An exploration of online harassment by know peers and online-only contacts. *Journal of Adolescent Health*, 41, s51–s58.

Interoception

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Definition

The sense of the internal, physiological condition of the body and organs (Craig 2002). Interoceptive signals arise from bodily tissues and organs such as the heart, lungs, GI tract, bladder, and skin. Sensing these interoceptive signals leads to the experience of body states such as pain, hunger, satiety, thirst, elimination, and sexual arousal. Furthermore, awareness of these internal body signals leads to the clear detection of emotions including anxiety, frustration, and calmness. An intact interoceptive system is positively correlated with emotional regulation (e.g., Füstös et al. 2012), intuitive eating (e.g., Herbert and Pollatos 2012), typical pain response (e.g., Weiss et al. 2014), self-awareness (e.g. Modinos et al. 2009; Tsakiris et al. 2007), intuitive decision-making (e.g., Bechara and Damasio 2005; Dunn et al. 2010; Gu and Fitzgerald 2014), and emotional perspective taking (e.g., Grynberg and Pollatos 2015; Singer et al. 2004). Conversely,

interoceptive differences are correlated with challenges such as higher levels of depression and anxiety (Paulus and Stein 2010; Avery et al. 2014).

Interoceptive differences have been found in individuals with autism spectrum disorder (Fiene and Brownlow 2015; Garfinkel et al. 2016). Furthermore, the insula, the area of the brain that processes interoceptive information (Critchley et al. 2004), is commonly affected in individuals with ASD providing a brain-based explanation for the interoceptive differences reported by many individuals with ASD (Di Martino et al. 2009, 2014; Uddin et al. 2013; Dickstein et al. 2013; Nomi and Uddin 2015). Given that interoception influences many areas that are often affected in individuals with ASD such as emotional regulation (Laurent and Rubin 2004; Mazefsky et al. 2013, 2014; Samson et al. 2014, 2015), interoception is emerging as an important consideration and area of study within the field of autism.

See Also

- Anxiety
- Emotional Intelligence
- Emotional Regulation
- Feeding Problems
- Perception
- Sensory Processing
- Toilet Training

References and Reading

- Avery, J. A., Drevets, W. C., Moseman, S. E., Bodurka, J., Barcalow, J. C., & Simmons, W. K. (2014). Major depressive disorder is associated with abnormal interoceptive activity and functional connectivity in the insula. *Biological psychiatry*, 76(3), 258–266.
- Bechara, A., & Damasio, A. R. (2005). The somatic marker hypothesis: A neural theory of economic decision. *Games and Economic Behavior*, 52(2), 336–372.
- Craig, A. D. (2002). How do you feel? Interoception: the sense of the physiological condition of the body. *Nature Reviews Neuroscience*, 3(8), 655.

- Critchley, H. D., Wiens, S., Rotshtein, P., Öhman, A., & Dolan, R. J. (2004). Neural systems supporting interoceptive awareness. *Nature Neuroscience*, 7(2), 189.
- Di Martino, A., Ross K., Uddin L., Sklar A., Costellanos, F. X. (2009). Functional brain correlates of social and nonsocial processes in autism spectrum disorders: An activation likelihood estimation meta-analysis. *Biological Psychiatry*, 65, 63–74.
- Di Martino, A., Yan, C. G., Li, Q., Denio, E., Castellanos, F. X., Alaerts, K.,... & Milham, M. P. (2014). The autism brain imaging data exchange: towards a large-scale evaluation of the intrinsic brain architecture in autism. *Molecular Psychiatry*, 19(6), 659–667.
- Dickstein, D. P., Pescosolido, M. F., Reidy, B. L., Galvan, T., Kim, K. L., Seymour, K. E., ... & Barrett, R. P. (2013). Developmental meta-analysis of the functional neural correlates of autism spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(3), 279–289.
- Fiene, L., & Brownlow, C. (2015). Investigating interoception and body awareness in adults with and without autism spectrum disorder. *Autism Research*, 8(6), 709–716.
- Füstös, J., Gramann, K., Herbert, B. M., & Pollatos, O. (2012). On the embodiment of emotion regulation: interoceptive awareness facilitates reappraisal. *Social Cognitive and Affective Neuroscience*, 8(8), 911–917.
- Garfinkel, S. N., Tiley, C., O’Keeffe, S., Harrison, N. A., Seth, A. K., & Critchley, H. D. (2016). Discrepancies between dimensions of interoception in autism: Implications for emotion and anxiety. *Biological Psychology*, 114, 117–126.
- Grynberg, D., & Pollatos, O. (2015). Perceiving one’s body shapes empathy. *Physiology and Behavior*, 140, 54–60.
- Gu, X., & Fitzgerald, T. H. (2014). Interoceptive inference: homeostasis and decision-making. *Trends in Cognitive Sciences*, 18(6), 269–270.
- Herbert, B. M., & Pollatos, O. (2012). The body in the mind: on the relationship between interoception and embodiment. *Topics in Cognitive Science*, 4(4), 692–704.
- Laurent, A. C., & Rubin, E. (2004). Challenges in emotional regulation in Asperger syndrome and high-functioning autism. *Topics in Language Disorders*, 24(4), 286–297.
- Mahler, K. J. (2016). *Interoception: The eighth sensory system: Practical solutions for improving self-regulation, self-awareness and social understanding of individuals with autism spectrum and related disorders*. Shawnee Mission: AAPC Publishing.
- Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(7), 679–688.
- Modinos, G., Ormel, J., & Aleman, A. (2009). Activation of anterior insula during self-reflection. *PloS One*, 4(2), e4618.
- Nomi, J. S., & Uddin, L. Q. (2015). Developmental changes in large-scale network connectivity in autism. *NeuroImage: Clinical*, 7, 732–741.
- Paulus, M. P., & Stein, M. B. (2010). Interoception in anxiety and depression. *Brain Structure and Function*, 214(5–6), 451–463.
- Samson, A. C., Phillips, J. M., Parker, K. J., Shah, S., Gross, J. J., & Hardan, A. Y. (2014). Emotion dysregulation and the core features of autism spectrum disorder. *Journal of Autism and developmental Disorders*, 44(7), 1766–1772.
- Samson, A. C., Hardan, A. Y., Podell, R. W., Phillips, J. M., & Gross, J. J. (2015). Emotion regulation in children and adolescents with autism spectrum disorder. *Autism Research*, 8(1), 9–18.
- Shah, P. (2016). Interoception: the eighth sensory system: practical solutions for improving self-regulation, self-awareness and social understanding of individuals with autism spectrum and related disorders.
- Singer, T., Seymour, B., O’Doherty, J., Kaube, H., Dolan, R. J., & Frith, C. D. (2004). Empathy for pain involves the affective but not sensory components of pain. *Science*, 303(5661), 1157–1162.
- Tsakiris, M., Hesse, M. D., Boy, C., Haggard, P., & Fink, G. R. (2007). Neural signatures of body ownership: A sensory network for bodily self-consciousness. *Cerebral Cortex*, 17, 2235–2244.
- Uddin, L. Q., Supekar, K., & Menon, V. (2013). Reconceptualizing functional brain connectivity in autism from a developmental perspective. *Frontiers in Human Neuroscience*, 7.
- Weiss, S., Sack, M., Henningsen, P., & Pollatos, O. (2014). On the interaction of self-regulation, interoception and pain perception. *Psychopathology*, 47(6), 377–382.

Interpersonal Skills

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Definition

Interpersonal skill is a broad term used to denote all types of human social and emotional interactions with each other. It is encompassing individual’s capabilities to interact efficiently with peers and adults as well as their capabilities to form affective ties or social relationships in the form of attachment with a main caregiver, friendship with peers, and romantic relationships. Social interaction (SI) is defined as a reciprocal process in which children effectively initiate and respond to social stimuli presented

by their peers or adults in diverse social settings and situations. Whereas, social interactions can be sporadic and include various individuals, social relationships inherently denote a mutual process of continuous interactions with a specific exclusive individual over a long period that enables the evolvement of affective bonding, closeness, and intimacy. Interpersonal interactions and skills are the basic unit of which social cognition (the understanding of self and others and the interaction between self and others) derives.

See Also

- Attachment
- Social Cognition

References and Reading

- Cassidy, J., & Shaver, P. (2008). *Handbook of attachment: Theory, research and clinical applications*. New York: Guilford Press.
- Lewis, M. (1999). Social cognition and the self. In P. Rochart (Ed.), *Early social cognition: Understanding others in the first months of life* (pp. 81–100). Mahwah: Lawrence Erlbaum Associates.
- Rubin, K. H., Bukowski, W. M., & Laursen, B. (2009). *Handbook of peer interactions, relationships, and groups*. New York: Guilford Press.
- Schneider, B. H., Atkinson, C., & Tardif, C. (2001). Child-parent attachment and children's peer relations: A quantitative review. *Developmental Psychology, 1*, 86–100.

Interpersonal Supports

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Synonyms

Peer supports; Transactional supports

Definition

Interpersonal Supports are activities or strategies provided by peers, teachers, parents, and community members which increase students' overall interpersonal skills for increased social interaction with one or more individuals. The tasks or skills to be targeted include joint attention, perspective taking, social and pretend play, social engagement, and social problem-solving (Luiselli et al. 2008). Interpersonal supports reflect the understanding that fostering social communication is an integral part of an individuals' day and must occur across a variety of social partners. It is an extension of individual or small group support provided by professionals for generalization. A key component of interpersonal supports is a shifting of emphasis away from the student to an emphasis on training and educating members of the students' interactive day to effectively facilitate appropriate social interaction (Marans et al. in Volkmar et al. 2005).

Students with ASD have a natural interest in their age level peers; therefore, these peers become an important interpersonal support to the student with ASD. Peers who have received training to adjust their communication style and interaction with the student with ASD are effective at facilitating and increasing appropriate interactions. Research supports the use of peer-mediated interventions, including peer mentors in naturalistic settings. Examples which have been published include Peer Integrated Play Groups by Pamela Wolfberg (2003); Peer Buddy Skills Training by K. English et al. (1997); and Peer implemented Pivotal Response Training by K. Pierce and Schreibman (1995, 1997) as cited in Volkmar et al. (2005). Additional resources for implementation of peer mentor programs may be found on the National Mentoring Resource Center website (<https://www.nationalmentoringresourcecenter.org/index.php/what-works-in-mentoring/resources-for-mentoring-programs.html?id=237>).

Although teachers and parents may have extended knowledge and understanding of social communicative competency levels in students with ASD, they will benefit from additional training and support regarding their own

communicative style and its impact on fostering increased social communication in the student with ASD. Teachers and parents, through their daily interaction with students with ASD, will provide valuable interpersonal support for the development of social communicative competency (Marans et al. in Volkmar 2005). An example program that has been designed to provide in depth training to parents and teachers is the Program for the Education and Enrichment of Relational Skills (PEERS) for Adolescents, for Adults, and for Preschoolers by Elizabeth Laugeson (2010). This program utilizes didactic instruction to guide parents toward understanding techniques that support conversational management skills.

Students with ASD will want to participate in community-based activities which reflect their personal interests and preferences. In the outset of participation within these activities, it will be necessary for parents to provide primary support that allows their child to successfully engage in the community. However, it will be necessary to consider means of providing training to those members of the community that will likely engage in interpersonal interaction with the individual with ASD. This training will need to include support strategies to foster and increase student social communicative competency within these community settings (Marans et al. in Volkmar 2005), such as core conversational management skills (eye contact, asking questions, commenting, and body proximity). Additionally, provisions under IDEA (2004) require that all educators within the Pre-K to 12 school setting receive this type of training. While there is not a current policy requiring training at the college/university level, as a growing number of individuals with ASD attend college, it will be imperative to develop training for those who provide interpersonal support to students on campus that includes best methods to encourage use of expected skills of conversational management and social interaction.

Interpersonal supports are beneficial to increasing the social communicative competency of students with ASD by utilizing all of the members of their interpersonal circle to facilitate and

foster positive social interaction that is engaging and reinforcing, and within multiple environments. As a result, individuals with ASD who receive this support, will have a broader peer network that will lead to an increased social capital, thus having a larger impact on successful outcomes.

See Also

- [Interpersonal Skills](#)
- [Relationship Enhancement Methods](#)
- [Social Interventions](#)

References and Reading

- English, K., Goldstein, H., Shafer, K., & Kaczmarek, L. (1997). Promoting interactions among preschoolers with and without disabilities: Effects of a buddy skills-training program. *Exceptional Children*, 63(2), 229–243. <https://doi.org/10.1177/001440299706300206>.
- Individuals with Disabilities Education Act, 20 U.S.C. § 1400 (2004).
- Laugeson, E. & Frankel, F. (2010). *Social skills for teenagers with developmental and autism spectrum disorders: The PEERS treatment manual*. New York, NY: Routledge.
- Luiselli, J., Russo, D., Christian, W., & Wilczynski, S. (Eds.). (2008). *Effective practices for children with autism: Educational and behavioral support interventions that work*. New York: Oxford Press.
- Marans, W., Rubin, E., & Laurent, A. (2005). Addressing social communication skills in individuals with high-functioning autism and Asperger syndrome: Critical priorities in educational programming. In F. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, pp. 989–995). Hoboken: Wiley.
- National Mentoring Resource Center website. (n.d.). Retrieved from <https://www.nationalmentoringresourcecenter.org/index.php/what-works-in-mentoring/resources-for-mentoring-programs.html?id=237>.
- Wolfberg, P. (2003). *Peer play and the autism spectrum: The art of guiding children's socialization and imagination*. Shawnee Mission, Kansas: AAPC.

Interval

- [Latency, Second Edition](#)

Intervention Activity

- [Role Release](#)

Intervention Integrity

- [Treatment Fidelity](#)

Intervention Targets and Strategies that Are Related to Increased Quality of Life Outcomes

- [Developmentally Appropriate Practice \(DAP\)](#)

Interventions for Increasing Acceptance of New Foods

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Definition

Interventions aimed at increasing acceptance of new foods into the diet involve using a range of techniques based on exposure to foods, conditioning, and family training or environmental changes. Acceptance is a broad term that includes any behavior from willingness to try a new food (e.g., tasting, putting food in the mouth) to increasing the number of whole foods eaten by the individual. It is often a goal of these interventions to increase intake of foods from different food groups in order to ensure a variety in the diet. New foods can be identified as foods that are not usually eaten as part of the habitual diet. These food items, however, do not have to be novel because familiar foods, or foods that were previously accepted, can also often be refused.

Historical Background

Autism as first described by Leo Kanner (1943) noted that feeding problems occur in many cases. These problems included food refusal, limited food repertoire, challenging behaviors around eating, and a variety of unusual eating behaviors that were individual to each case. Since then, feeding problems have been estimated to be prevalent in up to 89% of children with autism (Ledford and Gast 2006), encompassing a vast range of atypical eating behaviors. Although feeding problems occur more frequently in children with autism than in typically developing peers, issues with eating are not necessary or sufficient for an autism diagnosis. It has therefore been proposed that factors associated with autism, such as insistence on sameness, social communication deficits at mealtimes, or sensory sensitivities, may either cause or affect the likelihood of food refusal and food selectivity.

The literature regarding treatment of feeding problems has previously addressed food refusal and challenging behaviors more comprehensively than other eating behaviors such as limited food repertoire (limited dietary variety). This has led to a focus on food refusal only in clinical populations, where the problem has led to high levels of medical or psychological distress to the child or their family that it is deemed to warrant professional intervention (Dovey et al. 2009). This often includes conditions resulting in failure to thrive, growth faltering, or impact on cognitive development, usually as a consequence of nutritional or energy deficiencies. Where necessary, treatment may be medical in nature (e.g., percutaneous endoscopic gastrostomy (PEG) fed) to increase consumption of energy; however, the focus of many interventions to treat food refusal with the aim to increase intake of new foods has had a behavioral focus.

Historically, the goal of increasing acceptance of foods has been addressed using applied behavioral analysis (ABA) techniques such as that of operant conditioning (Riordan et al. 1980), including differential reinforcement of alternative behaviors and escape extinction (including non-removal of the spoon). Forced feeding and

overcorrection procedures have also been used previously to increase food intake and to correct unwanted eating behaviors. However, these punishment-based techniques are less frequently reported in the current literature and are only used clinically when other procedures have not been successful. Further techniques based on exposure, such as stimulus fading and demand fading, have also been developed as forms of systematic desensitization toward new foods. Yet despite these techniques being available, it was suggested in the late 1990s that interventions using positive reinforcement are useful to increase acceptance of foods, while non-removal of the spoon may be useful to correct food refusal (Kerwin 1999). However, due to the heterogeneity of the autism population, this conclusion has generally not been accepted, and the implementation of interventions has become more individualized to the needs of each individual child. Interventions are frequently tailored to specific causes of feeding problems, severity of feeding problems, and preferred outcomes in increasing acceptance of new foods.

In the last 20 years, the problems of food refusal and limited dietary variety have been collated with high-frequency intake of single food items under the broad term selective eating (Bandini et al. 2010). Selective eating acknowledges not only the wide range of eating behaviors that are considered problematic but also feeding problems that occur in most children with autism and not only in clinical populations. Therefore, most children with autism could benefit from healthy eating interventions that introduce new foods. This assumption is a direct response to problems of nutritional deficiencies and overweight being highlighted in children with autism. Consequently, interventions that aim to increase acceptance of a variety of new foods have become a priority. However, although interventions generally succeed in the acquisition of appropriate feeding behaviors, in many cases the maintenance of these feeding behaviors has been much more difficult to achieve.

Rationale or Underlying Theory

Interventions for accepting new foods assume that many foods are already refused in the child's diet due to selective eating. Accepting new foods attempts to address food selectivity by increasing the number of different foods that are eaten, as well as the amount of each new food eaten. It is thought that a variety of attributes in a child with autism could lead to selective eating, including unfamiliarity (food neophobia), sensory sensitivity (or tactile defensiveness), fear or previous food experiences (e.g., choking), sensitivity to food stimuli (color, shape, smell, taste, temperature, etc.), biological food intolerance, repetitive behaviors, gastrointestinal problems (e.g., constipation, reflux), and food allergy (Sharp and Postorino 2017). While this list is not exhaustive, it aims to highlight the heterogeneity of the population, which is reflected in the range of interventions available to attempt to treat food selectivity, based on individual needs.

In addition to child-specific traits, parental anxiety and unintentional reinforcement of non-preferred behaviors have also been suggested to maintain selective eating behaviors in the child. This may include preparing special meals for the child that differ from that of the rest of the family or removing the child from the eating situation if they display challenging behaviors (e.g., tantrums, throwing food, holding food in the mouth, etc.). Regardless of the underlying cause of selective eating, all interventions to increase acceptance of new foods consider the problem as a behavior that can be altered.

Goals and Objectives

Treating food selectivity aims to address the effects of selective eating more often than its causes. The effects that are targeted include increasing variety in the limited diet, reducing the amount of foods that are refused, and increasing the volume of non-preferred foods that are consumed. Studies often have secondary goals to

reduce any challenging behaviors during feeding occasions. Likewise, changing parental feeding behaviors may also be an appropriate goal in order to provide structure to mealtimes, although this is usually measured in relation to the child's eating behavior, rather than the parent's feeding behaviors.

The causes of food selectivity can also be addressed, but this is not usually explicitly reported in the literature. For example, sensory sensitivities may be decreased through multiple exposures to target foods, and therefore desensitization to the food stimulus occurs. These goals are not only relevant to psychological practice when a child presents with a feeding problem, but also for parents that have difficulty encouraging their child to eat certain foods and in educational settings where children's consumption of foods may also be limited.

Treatment Participants

All children, whether typically developing or diagnosed with autism, can benefit from an intervention that promotes eating a wider variety of foods and therefore, a healthier and more balanced diet. However in autism, the type of intervention delivered will depend on the individual case and their hypothesized cause of selectivity, what specific behaviors will be a target for change, and the severity of their selectivity. Although very little research has been conducted specifically to compare severities of selective eating, it is thought that severity will affect many aspects of treatment and its outcomes. Due to a more restricted diet, severely selective eaters might first need to increase their intake of a select few target foods before trying new foods and increasing variety, whereas it could be more appropriate for mildly selective eaters to increase acceptance for a variety of new foods, since these children already eat a wider variety of foods than severely selective eaters.

In addition to severity, age is a particular concern for when to intervene. Current research

would suggest an early intervention for a better prognosis (e.g., 2–8 years old), as food selectivity in autism is a stable trait that continues into adolescence (Bandini et al. 2017). Therefore, early intervention that promotes acceptance of a range of new foods is preferred as this behavior will be more likely to continue into adolescence if it is established first in childhood. Although it is suggested that eating behaviors are more difficult to change as the child gets older, a few case studies have reported positive changes in young adults after intervention. Additionally, when targeting the eating behavior of children, parental training is often a large part of delivering an intervention because parents will be the people feeding the child after the intervention finishes. Therefore, the parent must be willing and cooperative in order to learn the techniques that are successful at encouraging their child to accept new foods.

Lastly, despite most intervention research being conducted in children with autism, children targeted by feeding interventions to increase acceptance of new foods need not have a diagnosis of autism. Similar positive outcomes have been observed in children with other neurodevelopmental diagnoses including learning disability or developmental delay. If treatments are tailored to the individual's needs, then factors such as diagnosis and additional needs (e.g., communication ability) may be a barrier to an intervention having success, but will not necessarily make the individual unlikely to benefit from intervention.

Treatment Procedures

For increasing acceptance of new foods, three main categories of intervention have been identified based on exposure to food, conditioning techniques, and methods including family training or environmental changes (Chawner et al. 2019). Each of these groups of intervention has an array of techniques that can be used to increase acceptance of new foods (Table 1). The unique qualities of each category of intervention are determined by its explanation for the eating problem behavior.

Lack of experience or physical sensitivity to food can be addressed by exposure techniques; however, a more complex relationship with the food may need to be addressed with reinforcers and punishments (e.g., removal of a reward) as motivation to eat the food. Lastly, eating problems may stem from, or be maintained by, the environment. Therefore, changing aspects of the environment or training parents to change how feeding occurs can be useful. Most often, successful interventions will utilize a combination of these techniques.

Most research using these techniques implement them in controlled clinical settings. Less studies have implemented interventions in the home and even fewer have attempted school settings. However, as the setting changes, so does the role of the treatment provider. In the clinic, the treatment provider as the therapist has a role to assess the problem, plan an intervention, and change this plan if it does not work, whereas in the home setting, the professional will often train parents to carry out procedures in the hope that parental practices can continue to have positive effects after the intervention finishes. Ideally, all interventions designed to increase acceptance of foods in children should have parental involvement (Ledford et al. 2018). In this case, the treatment provider has a duty to ensure that parents are implementing the procedures correctly and safely, while not inadvertently causing any unwanted behaviors or outcomes.

Additionally, most interventions have been carried out only with individual children on a one-to-one basis. There is very little evidence for implementing interventions in groups due to the individuality of needs and the specific problem that is being addressed, as this will be different for different children. However, some research has reported delivering exposure interventions and environmental changes in schools or cafeterias to groups of children. These interventions make appropriate use of nonclinical settings, so severity of food selectivity is likely to be milder and exposure to new foods may produce preferred changes in behavior. It is also unlikely that exposure techniques alone will have adverse effects on children's eating behavior, unlike the potential adverse effects associated with using more

intensive interventions incorrectly (e.g., escape extinction). Therefore, these types of intervention are more appropriate for nonclinicians to implement safely to groups of children.

Furthermore, duration and frequency of the interventions are also very varied. Frequency can differ between one or two times per week or be as intense as three times per day. Duration has also been reported from 2 sessions to over 200 sessions, with some interventions lasting anywhere from a few days to over 6 months. Each session length is also variable. Although there appears to be no standard for time, this massive range of durations and frequency of session can be explained by the variety of different interventions used, severity and causes of selective eating, behaviors that are targeted to be changed, and clinician time. It is often not reported whether the intervention stops because the target outcome has been achieved. Therefore, there is no guarantee that a shorter, more intense intervention will have preferred effects, nor a longer and slightly less intense intervention. Consequently, interventions are tailored to the individual rather than following a specific standard course of intervention to increase acceptance of new foods.

Efficacy Information

Overall, all interventions and techniques mentioned in Table 1 have been shown to have some positive effects on increasing acceptance of new foods in children with autism (although some techniques changing aspects of the environment alone, e.g., special plates, have not been supported). However, because most interventions have been reported as effective in case studies, there is no one intervention that can be determined as better or worse than the other interventions. For this reason, it has been suggested that there is a hierarchy of interventions and techniques to use in increasingly challenging cases. If food selectivity is mild, environmental changes, systematic desensitization techniques, and family psychoeducation may be a valid form of intervention, while if the food selectivity is more resistant to change, conditioning techniques may be used to increase

Interventions for Increasing Acceptance of New Foods, Table 1 A description of intervention techniques used to increase the acceptance of new foods

Method/intervention	Description
Based on conditioning	
Differential reinforcement of alternative behavior (DRA)	Positive reinforcement of “target” or “good” behaviors (e.g., reinforced with food, toys, stickers, and verbal praise) on a variable schedule
Non-contingent reinforcement (NCR)	Reinforcement that is not dependent on completing a “target” behavior (e.g., swallowing a non-preferred food item)
Lag schedules	A schedule of reinforcement in which a single response, or a sequence of responses, is reinforced only if it varies from previous responses or sequences of responses
Escape extinction (EE)	This technique describes various procedures that prevent escape from the feeding situation (including non-removal of the spoon: NRS and physical guidance)
Non-removal of the spoon (NRS)	A type of EE that holds a spoon close to the mouth until the food is accepted
Physical guidance	Guiding the mouth open or applying small pressure to the jaw to assist with accepting food into the mouth
Based on exposure	
Systematic desensitization	A method designed to reduce avoidance behavior toward an adverse stimulus by gradually increasing exposure to it
Stimulus/texture, portion and demand fading	Three methods of systematic desensitization
	Stimulus/texture fading: Gradually changing the texture of a food (e.g., runny mashed potato can be made increasingly thicker)
	Portion fading: Gradually increasing the portion size of a new food (e.g., from the size of a pea to a recommended serving)
	Demand fading: Gradually increasing behaviors that are required by the participant (e.g., increasing the demand from one bite to three bites)
Simultaneous presentation	A type of flavor-flavor conditioning that pairs a non-preferred food with a preferred food or liked condiment
Using new foods similar to those previously accepted	Using similar foods to those already accepted (e.g., matching by food group, brand, color, texture, etc.)
Modeling	Watching others eat the non-preferred food (e.g., parents, siblings, friends)
High-probability sequences	This requires asking the participant to complete a high-probability task (e.g., put spoonful of preferred food in the mouth) before asking them to perform a low-probability task (e.g., put a spoonful of non-preferred food in the mouth)
Choice of foods	Allowing the participant a choice between different non-preferred foods
Access to preferred food	Allowing or denying access to preferred foods before the non-preferred food is presented
Family and environmental methods	
Psychoeducation	Psychoeducation involves providing education and information to family members about selective eating
Parental training	Most of the techniques described are implemented by clinicians or researchers. Parental training is designed so that parents can implement some strategies themselves
Mealtime plans	Mealtime plans are implemented by the family and focus on areas such as communication, food offered, and the social and physical environment during mealtimes
Positive behavioral support (PBS)	A multicomponent intervention that aims to support an individual. This includes a functional assessment of possible relationships between environment and behavior, which can inform support for eating using appropriate methods (methods in this table) for the individual
Environmental interventions	Environmental interventions change an aspect of the physical setting. This can include changing the layout and placement of healthy foods in lunchrooms, using special plates that show how much of the plate should be filled with portions from each food type and using games to encourage fruit and vegetable intake

motivation to accept new foods by using reinforcers to shape behavior. Further up the hierarchy, extinction procedures may be used if foods are still not accepted, followed by physical guidance as a last resort in cases where food selectivity poses a threat to health or well-being.

As specific intervention techniques cannot be directly compared adequately, efficacy for the characteristics of interventions is compared instead. One component is whether a single intervention is delivered or whether multiple intervention techniques are delivered together. Systematic reviews have highlighted that using multiple techniques usually results in more preferred outcomes. This may be due to the heterogeneity of selective eating, and using multiple techniques could address several areas of the eating problem that allows for better outcomes. However, because of the case study nature of the literature, it is not the individual techniques that are supported for their efficacy, but rather the intervention package that is tailored to the individual that has been shown to be effective to increase acceptance of new foods.

Generally, research has found that selective eating interventions appear to be successful at increasing the volume of target foods consumed, but not necessarily to increase variety in the diet (Marshall et al. 2015). It is suggested that familiar foods may be more easily processed from an oral motor or sensory perspective. Therefore, when target foods are used, there is often a lack of generalization to other foods. This is beneficial when reducing refusal of foods, but less so when the aim is to increase dietary variety. The aims of treatment frequently take a stepped approach which increases the time needed for behavior change to occur. First, the aim is to overcome any food refusal, then to reduce any challenging behaviors, and finally to establish a nutritionally adequate diet, which would include a variety of foods. It is possible this last step is not achieved or reported in most case studies because it is an ongoing process. Due to the length of time needed in some cases to increase dietary variety, the duration of intervention may not be long enough to achieve this goal because of clinician time and

priorities. Therefore, if parents are not taught the therapist's intervention techniques, then any changes to eating could stop when the intervention does. Any further benefit gained from the parent's implementation of techniques after intervention would also not be recorded.

Similarly, persistence of effects is not well understood from the literature. This is because many reports do not include follow-up time points after intervention. However, of those that do, there is often some loss of performance, yet outcomes are generally reported as better than before the intervention. Most studies also do not include generalization measures for new foods, a new location, or a different implementer of intervention, although such information would be very useful in order to evaluate the efficacy of individual interventions. Lastly, while we are aware of factors that predict food selectivity, there is no direct research into which individual differences affect gaining benefit from interventions or for whom interventions work. There are rare reports of other comorbid feeding-related medical problems in each case, and family characteristics or demographics are often not reported to an accurate degree. It has been suggested that higher food selectivity, more feeding-related problems, challenging behaviors, maintenance behaviors from parents, low SES, and lower availability of foods may all increase the difficulty of an intervention being successful. However, these claims are mostly anecdotal and cannot be supported by evidence.

Outcome Measurement

There are many outcome measurements that can be used to determine the success of an intervention for increasing acceptance of foods. These are percentage bite acceptance, number of foods eaten, number of bites eaten (a bite can be defined as a small portion of food that will easily fit in the mouth, e.g., 1.5 cm³), behavioral demands (e.g., accomplishing predetermined behaviors such as putting the food to the lips or inside the mouth),

weighted food intake (grams), and self-report (parental report of what the child has eaten; this could be a food frequency questionnaire of all foods eaten in the last year or a 3-day food diary). Other outcome measures may include the reduction in challenging behaviors, such as throwing food. This can be measured by coding the frequency of such behaviors during each session.

The main difference between outcome measurements is whether it is a macro measurement (e.g., number of whole foods) or a micro measurement (e.g., percentage of bites accepted). This will be based on what is achievable for the child in the available timeframe, what is an acceptable goal to achieve, and what the intervention is designed to target. Choosing an outcome measure, like choosing an intervention, also depends on the severity of each case. In more severe selective eating cases, an attempt to try the new food may be an appropriate goal. It could be deemed a successful intervention if the child puts the food in their mouth even if they do not swallow it. In these cases, it may take a long duration to increase intake and therefore smaller goals must be set initially. However, number of whole new foods eaten may be an achievable goal if the child is mildly selective and already eats foods from different food groups. Although it is acceptable to choose an outcome measure based on what is achievable, much research does not report why the outcome measure they have used was chosen initially.

Qualifications of Treatment Providers

The qualifications needed to provide intervention depends on many factors including the severity of food selectivity, the type of intervention being delivered, and the initial problem that the intervention was designed to address. This is because if a child is very selective and the intervention is not conducted well, it may result in further selectivity or feeding problems for the child. Therefore, conditioning techniques should be implemented by trained professionals or researchers that have

experience delivering behavioral, or applied behavioral analysis (ABA), techniques. Although some studies have trained parents to be able to implement these procedures, these parents are trained by professionals with an ABA qualification. Alternatively, systematic desensitization and environmental change techniques could be implemented by parents or teachers to increase exposure to new foods with low risk of adverse effects. Nonetheless, they should still be trained in the specific intervention by an appropriate person and supported throughout its delivery.

See Also

- [Behavioral Assessment Scale of Oral Functions in Feeding](#)
- [Feeding Problems](#)
- [Using Shaping to Increase Foods Consumed by Children with Autism](#)

References and Reading

- Bandini, L. G., Anderson, S. E., Curtin, C., Cermak, S., Evans, E. W., Scampini, R., ... Must, A. (2010). Food selectivity in children with autism spectrum disorders and typically developing children. *The Journal of Pediatrics*, 157(2), 259–264.
- Bandini, L. G., Curtin, C., Phillips, S., Anderson, S. E., Maslin, M., & Must, A. (2017). Changes in food selectivity in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(2), 439–446.
- Chawner, L. R., Blundell-Birtill, P., & Hetherington, M. M. (2019). Interventions for increasing acceptance of new foods among children and adults with developmental disorders: A systematic review. *Journal of Autism and Developmental Disorders*, 49(9), 3504–3524.
- Dovey, T. M., Farrow, C. V., Martin, C. I., Isherwood, E., & Halford, J. C. (2009). When does food refusal require professional intervention? *Current Nutrition & Food Science*, 5(3), 160–171.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2(3), 217–250.
- Kerwin, M. E. (1999). Empirically supported treatments in pediatric psychology: Severe feeding problems. *Journal of Pediatric Psychology*, 24(3), 193–214.
- Ledford, J. R., & Gast, D. L. (2006). Feeding problems in children with autism spectrum disorders: A review.

Focus on Autism and Other Developmental Disabilities, 21(3), 153–166.

- Ledford, J. R., Whiteside, E., & Severini, K. E. (2018). A systematic review of interventions for feeding-related behaviors for individuals with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 52, 69–80.
- Marshall, J., Ware, R., Ziviani, J., Hill, R. J., & Dodrill, P. (2015). Efficacy of interventions to improve feeding difficulties in children with autism spectrum disorders: a systematic review and meta-analysis. *Child: Care, Health and Development*, 41(2), 278–302.
- Riordan, M. M., Iwata, B. A., Wohl, M. K., & Finney, J. W. (1980). Behavioral treatment of food refusal and selectivity in developmentally disabled children. *Applied Research in Mental Retardation*, 1(1–2), 95–112.
- Sharp, W. G., & Postorino, V. (2017). Food selectivity in autism spectrum disorder. In *Clinical handbook of atypical and complex eating disorders*. New York: Oxford University Press.

Interventions to Support Transition to Adulthood for Individuals with Autism Spectrum Disorder

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Definition

The transition from adolescence to adulthood (i.e., ages 18–25) is a critical developmental period often marked with multiple stressors as individuals become more independent and explore new roles (e.g., living independently, employment, and postsecondary education). For individuals with autism spectrum disorder (ASD), this period is particularly difficult, with most exiting high school and leaving behind the integrated child services and supports they once knew. There is an urgent need to study outcomes and effective

transition to adulthood interventions to help support individuals with ASD and their families during this time.

Historical Background

Current prevalence estimates from the Centers for Disease Control and Prevention (CDC) indicate a 178% increase in school-age children diagnosed with ASD from 1 in 150 8-year-olds in 2002 to 1 in 54 8-year-olds in 2016 (Maenner et al. 2020). Of note, the first cohort of children in these CDC studies is now 26 years old, suggesting that we can anticipate a parallel increase in adolescence and young adults with ASD transitioning into adulthood. Research has indicated that quality of life (QOL) outcomes for adults with ASD are poor and characterized by low rates of employment, a plateau or decline in independent self-care skills, a lack of access to services, and high rates of anxiety and depression (e.g., Anderson and Butt 2017; Dudley et al. 2019; Hillier et al. 2018; Roux et al. 2013). For instance, in a nationally representative sample of 21–25-year-old young adults with ASD ($N = 620$), only 53% had ever received paid work since leaving high school, and only 33% had a current job (Roux et al. 2013). In addition, 50% of adolescents and young adults with ASD experience a disruption in vocation (e.g., job loss) or postsecondary education (e.g., expulsion) within the first 3 years post-high school (Taylor and DaWalt 2017).

At least 42% of individuals with ASD have average or above-average cognitive ability and the potential for competitive employment (Maenner et al. 2020). One might assume that those with ASD and higher IQs would have more positive outcomes; however, data do not support this assumption. Data from the Adult Consumer Survey, a national representative sample of adults with ASD using developmental disability services, showed that only 23% of adults with ASD without a co-occurring intellectual disability had a paid job in the community (Roux et al. 2017). Taylor and Seltzer (2010) found that

young adults with high functioning ASD (i.e., those with average to above-average IQs) were three times more likely to have no daytime activities after high school than those with ASD and comorbid intellectual disability (ID). They also found that only 18% of those with high-functioning ASD received employment services after leaving school compared to 86% of those with comorbid ID. For those that are employed, adults with high-functioning ASD are frequently underemployed, switch jobs often, have difficulty adjusting to new job settings, and make less money than their neurotypical peers (Hendricks 2010). High unemployment rates contribute to the \$1.4 million estimated lifetime cost of supporting an adult with ASD without co-occurring intellectual disability (Buescher et al. 2014). These results suggest that individuals with ASD without comorbid ID may be “falling through the cracks” during the transition to adulthood and are in need of additional services.

Not only are employment outcomes poor for this population, but similar challenges exist related to postsecondary education during the transition to adulthood. While a growing number of individuals with ASD are seeking higher education opportunities (Gobbo and Shmulsky 2014), only a fraction completes a degree (Shattuck et al. 2012). Newman et al. (2011) reported that 39% of students with ASD who started postsecondary completed their program. This contrasts with a graduation rate of nearly 60% for neurotypical students who start postsecondary education. Notably, when young adults with ASD do attend college, the vast majority of them attend 2-year or community colleges for at least part of their college experience. Roux et al. (2015) reported that in a nationally representative sample of those who attended postsecondary education, 88% of young adults with ASD attended 2-year colleges or vocational/technical schools. Because most of those who pursue post-secondary education will go through the community college system, community colleges could be an ideal location to provide transition interventions for young adults with ASD.

To attempt to address the growing needs of adolescents and young adults with ASD, there has been a substantial increase in requests for transition to adulthood services; applications for Vocational Rehabilitation (VR) services to support transition-aged individuals with ASD increased by 571% between 2003 and 2012 (Alverson and Yamamoto 2017). However, simply increasing access to services does not seem to be enough to improve outcomes (Alverson and Yamamoto 2017; Burgess and Cimeria 2014). In a review of over 35,000 transition-aged adults receiving VR services, only one-third of adults with ASD achieved successful employment (Burgess and Cimeria 2014). Thus, traditional VR services and supports appear to be inadequate in fully supporting the needs of individuals with ASD transitioning to adulthood. Thus, there is an urgent need for the development of effective, evidence-based transition to adulthood interventions that target ASD-specific challenges, promote better QOL, and improve employment and post-secondary outcomes.

ASD-Specific Challenges

While the empirical intervention literature is sparse, there is a growing consensus among researchers, adults with ASD, and caregivers that targeted interventions addressing ASD-specific challenges during the transition to adulthood are desperately needed to help adolescents/young adults develop to their full potential. Across studies using stakeholder (e.g., caregivers, adults with ASD, university professionals) focus groups, interviews, and surveys, several distinct challenges for college-bound and college-enrolled students with ASD have emerged including: executive function skills (i.e., time management, organization) (Wallace et al. 2016), adult-appropriate social skills (i.e., adult relationships, employment setting social behavior) (Hendricks 2010; Hillier et al. 2018), and emotional regulation (i.e., anxiety, coping) (Anderson and Butt 2017; Hillier et al. 2018). While there is little to no information regarding the effective targeting of these domains for young adults with ASD, the

childhood and younger adolescent intervention literature identifies evidence-based strategies to target these skills for those with average to above-average IQs. Overall, the following have been found to be evidence-based strategies to target these domains: *executive function* – visual supports (e.g., visual schedules), explicit rules, and routine strategies; *social skills* – scripts, social stories, modeling, self-monitoring, and role play activities; and *emotion regulation* – coping strategies (e.g., relaxation paired with problem solving) and other cognitive-behavioral therapy strategies (see Klinger and Dudley 2019, for a review).

Transition Interventions

Despite poor employment and postsecondary outcomes experienced by many adults with ASD, there are no published large-scale randomized clinical trials examining the effectiveness of transition intervention programs evaluating either short-term (e.g., changes in executive function) or long-term (e.g., employment) outcomes for young adults with ASD. However, there have been four published pilot intervention studies focused on supporting the transition to adulthood for adolescents and young adults with ASD: (1) *Transitioning Together*; (2) *Acquiring Career, Coping, Executive Control, Social Skills* (ACCESS); (3) *Connections*; and (4) *Stepped Transition in Education Program for Students with ASD* (STEPS).

To provide an initial evaluation of a multi-family group program for high school students with ASD and their parents (i.e., *Transitioning Together*), DaWalt and colleagues (2018) conducted a preliminary waitlist control randomized clinical trial (RCT) to examine the impact on the transition from high school to college (DaWalt et al. 2018). They found the intervention produced increased parent well-being and improved adolescent social interactions. Similarly, a pilot RCT found that the ACCESS program, which was designed for young adults with ASD to improve skills necessary for adulthood, significantly improved adaptive skills, self-determination, and increased coping self-efficacy (Oswald et al. 2018). Hillier et al. (2018) conducted one

noncontrolled (open trial) study examining the effectiveness of an intervention for college students with ASD (i.e., *Connections* program) (Hillier et al. 2018). They reported that participation in a college support group for young adults with ASD was associated with decreased anxiety and loneliness. Lastly, White and colleagues conducted a randomized control study examining the efficacy of the *STEPS* program with 59 16–25-year-old adolescents and emerging adults (White et al. 2019). Significant gains were found in parent-reported transition readiness skills for the treatment group but not the control group. Further, students enrolled in postsecondary education enrolled in the treatment group reported increased adaptation to college compared to the control group. Across these four interventions, findings provide preliminary evidence suggesting that transition intervention programs may be effective at increasing social communication and emotion regulation abilities in individuals with ASD. However, additional intervention development and clinical trials are needed to create an evidence base for transition to adulthood services that specifically target the challenges experienced by adolescents and young adults with ASD.

Current Knowledge

The TEACCH School Transition to Employment and Postsecondary Education Program (T-STEP Program) was created to address the current paucity of interventions targeting executive function, professional social skills, and emotion regulation for individuals with ASD transitioning to adulthood. The T-STEP was originally developed for diploma-track high school students and has moved to implementation in community college settings for students with ASD with average to above average IQs. The decision to transition to a community college setting was based on Roux et al.'s (2015) findings that the majority of students with ASD choosing postsecondary education start this education in community college settings. Further, our local North Carolina Community College System requested assistance supporting the needs of college students with

ASD as their largest community college reported an increase from 6 students with ASD to 126 students with ASD requesting disability services between 2003 and 2014. Thus, community colleges represent an ideal opportunity to provide targeted transition to adulthood services for individuals with ASD.

The T-STEP is a comprehensive, manualized, 12-week transition program that is currently being delivered in community college settings across North Carolina. The program is offered as a Workforce Innovation and Opportunity Act Pre-Employment Transition Service (Pre-ETS) program through a collaboration with the North Carolina Community College System and the North Carolina Division of Vocational Rehabilitation Services. The program is offered as a noncredit earning continuing education course open to 16–21-year-old high school or college students who have completed or are in the process of completing a general education high school diploma. The curriculum includes 36 h of group-based instruction delivered within a traditional academic school semester (i.e., 24 90-min classes across 12 weeks). Students also participate in a weekly campus-based internship to practice the skills learned in the intervention group (2 h/week for 10 weeks). In addition, students participate in 12 h of individual counseling services that are consistent with counseling services currently available on college campuses (i.e., higher education counseling, career counseling, and self-advocacy counseling about how to access disability accommodations at college and at work).

T-STEP Class. A student workbook was created through an iterative revision process with the input of a community advisory board consisting of parents of adults with ASD, self-advocates including a current college student with ASD, educators (high school special education teacher, community college instructor), disability support specialists (community college accessibility services, state disability service agencies), and employment services specialists. Additionally, focus groups were conducted with students completing the program.

Each T-STEP class session includes a review of previously learned concepts, introduction and

practice with new concepts, and a wrap-up in which home practice is assigned. The intervention contains six modules, each targeting pivotal employment and postsecondary education skills that address the specific challenges faced by young adults with ASD. Two modules target executive function skills (i.e., planning/organization; time management/flexibility), two modules target social communication skills (i.e., asking for help, engaging in professional social behavior), and two modules target emotion regulation skills (i.e., accepting corrective feedback, effectively coping with distress). Each module includes skill instruction in a group format, video modeling, and role-play opportunities in large group, small group, and individualized instruction activities. Each module is an integration of evidence-based practices as defined by the National Professional Development Center (Wong et al. 2015) including visual supports, video modeling, schedules, modeling, social narratives, self-management, cognitive behavioral intervention, and reinforcement. Students are taught routine strategies that they can implement across multiple settings. For example, an asking for help routine strategy highlights the importance of identifying “who” to ask for help, “when” to ask for help, “where to seek help,” and “what” to say. To support learning and generalization, these strategies are practiced through video modeling, small group activities, and through campus-based internships.

Work-Based Learning Experiences/Internship.

In addition to the group-based instruction, each student participates in a work-based learning experience or internship that provides opportunities for “real-world” practice beyond the classroom. Internship sites occur on community college campuses and include a variety of settings (e.g., library, academic departments, cafeteria, and science labs) that are matched to each student’s individual interests and goals. Students complete an internship agreement with each internship supervisor and bring an internship notebook that identifies T-STEP skills that should be practiced during the internship (e.g., the internship supervisor supports a student to learn an appropriate social greeting expected at the library; the internship supervisor prompts the student to

use a coping routine when corrected by the supervisor). T-STEP instructors consult with internship supervisors and students weekly to monitor progress and support the implementation of T-STEP skills. Students are expected to participate in at least 20 h of internship activities across the T-STEP program (i.e., 2 h/week).

Individual Counseling. College students enrolled in the T-STEP program also receive 12 1-h weekly counseling sessions that parallel services typically available on a college campus (i.e., job exploration/career counseling, postsecondary education counseling, and self-advocacy counseling). Students receive a workbook that provides instruction in each of these skills. Specifically, students complete career interest assessments and collaborate with career counselors in completing an individualized plan for each student that is appropriate given their abilities. Students also receive counseling to explore college course offerings and postsecondary opportunities related to chosen career pathways and are given resources for success in postsecondary education settings. Finally, students receive self-advocacy counseling focused on accessing disability services and supports at college and at work.

Initial Efficacy. Across pilot research in both high school and community college settings, initial research supports the efficacy of the T-STEP. In an open trial pilot study, 57 students (aged 17–21 years; average age 19.3 years) completed the T-STEP across three community colleges. Students were assessed at baseline and following completion of the intervention. Positive impacts were reported by students, caregivers, and via observational assessments. Specifically, caregivers reported that their students showed significant increases in employment readiness skills (Becker Work Adjustment Profile) including increases in work habits, interpersonal effectiveness, and work performance skills. Caregivers also reported significant increases in everyday self-care skills (Waisman Activities of Daily Living). Students reported significant increases in their ability and opportunity to make choices in their lives (AIR Self Determination Scale). They also reported decreases in symptoms of depression (Center for Epidemiological Studies –

Depression). Further, we created an observational measure (TEACCH Transition Readiness and Employability Evaluation, TREE) to assess the specific executive function (i.e., planning/organization, time management), social communication skills (i.e., asking for help, engaging in professional social behavior), and emotion regulation skills (i.e., accepting corrective feedback, effectively coping with distress) targeted by the T-STEP. Students completed two tasks typical of an office setting (e.g., data entry, filing) with a series of structured presses to assess T-STEP skills (e.g., missing materials were a press for the student to ask for help). On the TREE, significant improvements were seen in executive function, social communication, and emotion regulation skills.

In focus groups, students reported that the program helped prepare them to learn how to function in the adult world so that they could be successful as a college student and in the workplace. They responded that the program was “definitely” worth the time and that they would recommend it to others. Thus, across qualitative and quantitative assessments, initial results are promising for the T-STEP as an intervention addressing ASD-specific challenges during the transition to adulthood.

Future Directions

There is an emerging intervention literature describing transition interventions designed to promote critical skills needed in adulthood (e.g., executive function, social skills, and emotion regulation). Initial evidence is promising, indicating that we can improve the transition to adulthood for adolescents and young adults with ASD through targeted interventions. However, larger randomized control trials are necessary to evaluate the efficacy of these newly developed interventions and to identify individual characteristics (e.g., cognitive ability, language skills, and comorbid anxiety) associated with both positive and negative intervention outcomes. With regard to our research on the efficacy of the T-STEP, two randomized clinical trials are ongoing.

Across transition interventions, future research should not only include measures of short-term outcomes, but also measure long-term changes (e.g., employment, completion of post-secondary degrees, QOL). It is also critical that we move to evaluating the effectiveness of these programs in large-scale comparative efficacy studies (i.e., compare interventions to other evidence-based programs, rather than services-as-usual) and aim to conduct full community-based participatory research in which key community members (e.g., young adults with ASD, parents of young adults with ASD, service providers) are actively involved in research design and implementation. Lastly, dissemination research is needed to implement transition interventions in locations that are accessible to individuals from diverse backgrounds (e.g., in community colleges, community clinics) and that can be implemented by community service providers. The long-term goal is to increase the likelihood for community use and the ability to more effectively support individuals with ASD as they transition to adulthood.

See Also

- ▶ [Adulthood, Transition to](#)
- ▶ [College Transitional Programs](#)
- ▶ [Quality of Life for Transition-Age Youth with ASD](#)

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References and Reading

Alverson, C. Y., & Yamamoto, S. H. (2017). Employment outcomes of vocational rehabilitation clients with autism spectrum disorders. *Career Development and*

- Transition for Exceptional Individuals*, 40(3), 144–155. <https://doi.org/10.1177/2165143416629366>.
- Anderson, C., & Butt, C. (2017). Young adults on the autism spectrum at college: Successes and stumbling blocks. *Journal of Autism and Developmental Disorders*, 47, 3029–3039. <https://doi.org/10.1007/s10803-017-3218-x>.
- Buescher, A. V. S., Cidav, Z., Knapp, M., & Mandell, D. S. (2014). Costs of autism spectrum disorder in the United Kingdom and the United States. *JAMA Pediatrics*, 168, 721–728.
- Burgess, S., & Cimera, R. E. (2014). Employment outcomes of transition-aged adults with autism spectrum disorders: A state of the states report. *American Journal on Intellectual and Developmental Disabilities*, 119(1), 64–83. <https://doi.org/10.1352/1944-7558-119.1.64>.
- DaWalt, L. S., Greenberg, J. S., & Mailick, M. R. (2018). Transitioning together: A multi-family group psychoeducation program for adolescents with ASD and their parents. *Journal of Autism and Developmental Disorders*, 48(1), 251–263. <https://doi.org/10.1007/s10803-017-3307-x>.
- Dudley, K. M., Klinger, M. R., Meyer, A., Powell, P., & Klinger, L. G. (2019). Understanding service usage and needs for adults with ASD: The importance of living situation. *Journal of Autism and Developmental Disorders*, 49, 556–568.
- Gobbo, K., & Shmulsky, S. (2014). Faculty experience with college students with autism spectrum disorders: A qualitative study of challenges and solutions. *Focus on Autism and Other Developmental Disabilities*, 29(1), 13–22. <https://doi.org/10.1177/1088357613504989>.
- Hendricks, D. (2010). Employment and adults with autism spectrum disorders: Challenges and strategies for success. *Journal of Vocational Rehabilitation*, 32, 125–134. <https://doi.org/10.3233/JVR-2010-0502>.
- Hillier, A. J., Goldstein, J., Murphy, D., Trietsch, R., Keeves, J., Mendes, E., & Queenan, A. (2018). Supporting university students with autism spectrum disorder. *Autism*, 22(1), 20–28. <https://doi.org/10.1177/1362361317699584>.
- Klinger, L. G., & Dudley, K. M. (2019). Autism spectrum disorder. In M. J. Prinstein, E. A. Youngstrom, E. J. Mash, & R. A. Barkley (Eds.), *Treatment of childhood disorders* (pp. 376–415). New York: Guilford Press.
- Maenner, M. J., Shaw, K. A., Baio, J., et al. (2020). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2016. *MMWR Surveillance Summaries*, 69(SS-4), 1–12.
- Newman, L., Wagner, M., Knokey, A. M., Marder, C., Nagle, K., Shaver, D., & Wei, X. (2011). *The post-high school outcomes of young adults with disabilities up to 8 years after high school: A report from the National Longitudinal Transition Study-2 (NLTS2)*. National Center for Special Education Research (NCSE 2011-3005). Menlo Park: SRI International.
- Oswald, T. M., Winder-Patel, B., Ruder, S., Xing, G., Stahmer, A., & Solomon, M. (2018). A pilot

randomized controlled trial of the ACCESS program: A group intervention to improve social, adaptive functioning, stress coping, and self-determination outcomes in young adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(5), 1742–1760. <https://doi.org/10.1007/s10803-017-3421-9>.

- Roux, A. M., Shattuck, P. T., Cooper, B. P., Anderson, K. A., Wagner, M., & Narendorf, S. C. (2013). Post-secondary employment experiences among young adults with an autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(9), 931–939. <https://doi.org/10.1016/j.jaac.2013.05.019>.
- Roux, A. M., Shattuck, P. T., Rast, J. E., Rava, J. A., Edwards, A. D., Wei, X., . . . Yu, J. W. (2015). Characteristics of two-year college students on the autism spectrum and their support services experiences. *Autism Research and Treatment*, 1–10. <https://doi.org/10.1155/2015/391693>.
- Roux, A. M., Rast, J. E., Anderson, K. A., & Shattuck, P. T. (2017). *National autism indicators report: Developmental disability services and outcomes in adulthood*. Philadelphia: Life Course Outcomes Program, A. J. Drexel Autism Institute, Drexel University.
- Shattuck, P. T., Narendorf, S. C., Cooper, B., Sterzing, P. R., Wagner, M., & Taylor, J. L. (2012). Post-secondary education and employment among youth with an autism spectrum disorder. *Pediatrics*, 129(6), 1042–1049. <https://doi.org/10.1542/peds.2011-2864>.
- Taylor, J. L., & DaWalt, L. S. (2017). Brief report: Post-secondary work and educational disruptions for youth on the autism spectrum. *Journal of Autism and Developmental Disorders*, 47(12), 4025–4031. <https://doi.org/10.1007/s10803-017-3305-z>.
- Taylor, J. L., & Seltzer, M. M. (2010). Changes in the autism behavioral phenotype during the transition to adulthood. *Journal of Autism and Developmental Disorders*, 40(12), 1431–1446. <https://doi.org/10.1007/s10803-010-1005-z>.
- Wallace, G. L., Kenworthy, L., Pugliese, C. E., Popal, H. S., White, E. I., Brodsky, E., & Martin, A. (2016). Real-world executive functions in adults with autism spectrum disorder: Profiles of impairment and associations with adaptive functioning and co-morbid anxiety and depression. *Journal of Autism and Developmental Disorders*, 46, 1071–1083. <https://doi.org/10.1007/s10803-015-2655-7>.
- White, S. W., Smith, I. C., Miyazaki, Y., Conner, C. M., Elias, R., & Capriola-Hall, N. (2019). Improving transition to adulthood for students with autism: A randomized controlled trial of STEPS. *Journal of Clinical Child & Adolescent Psychology*. <https://doi.org/10.1080/15374416.2019.1669157>.
- Wong, C., Odom, S. L., Hume, K. A., Cox, A. W., Fettig, A., Kucharczyk, S., & Schultz, T. R. (2015). Evidence-based practices for children, youth, and young adults with autism spectrum disorder: A comprehensive review. *Journal of Autism and Developmental Disorders*, 45(7), 1951–1966.

Interventions: Child Centered Approaches

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Synonyms

Child-focused approaches

Definition

Child-centered approaches refer to autism interventions that incorporate the child's individual needs, interests, preferences, and choices into the planning and implementation of treatment. Additionally, these approaches are tailored to the child's developmental level and individual strengths.

Historically, treatment plans for children with autism were exclusively adult-directed, with the clinician deciding on the treatment goals, intervention strategies, materials used, and specific activities. Emphasis was placed on using repetitive, drill-like instructional activities as the primary means of teaching the child new skills. This mass trial approach resulted in systematic behavior gains, but the rate of skill acquisition was relatively slow, and children were not highly motivated to learn.

The original call for child-centered approaches was borne out of the establishment of social validity as a central concept in treatment planning, or the consideration of the long-term social value and acceptability of the treatment approach by the child and other key stakeholders invested in the child's outcome (Wolf 1978). By incorporating child-centered components into the intervention approach, the child's motivation to be an active participant in the intervention process is significantly increased, leading to improved task engagement and an increase in the rate of skill acquisition (e.g., Koegel et al. 1987). Examples of

empirically supported child-centered approaches include Pivotal Response Treatment (Koegel and Koegel 2006), Incidental Teaching (McGee et al. 1999), and the SCERTS model (Prizant et al. 2006).

See Also

► [Incidental Teaching](#)

References and Reading

- Koegel, R. L., & Koegel, L. K. (2006). *Pivotal response treatments for autism*. Baltimore: Paul H. Brookes.
- Koegel, R. L., O'Dell, M. C., & Koegel, L. K. (1987). A natural language teaching paradigm for nonverbal autistic children. *Journal of Autism and Developmental Disorders*, 17, 187–200.
- McGee, G. G., Morrier, M. J., & Daly, T. (1999). An incidental teaching approach to early intervention for toddlers with autism. *Journal of the Association for Persons with Severe Handicaps*, 24(3), 133–146.
- Prizant, B. M., Wetherby, A. M., Rubin, E., Laurent, A. C., & Rydell, P. J. (2006). *The SCERTS model: A comprehensive educational approach for children with autism spectrum disorders*. Baltimore: Paul H. Brookes.
- Wolf, M. M. (1978). Social validity: The case for subjective measurement or how applied behavior analysis is finding its heart. *Journal of Applied Behavior Analysis*, 11, 203–214.

Interview-Informed Synthesized Contingency Analysis (IISCA)

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Definition

The interview-informed synthesized contingency analysis (IISCA; Hanley et al. 2014) is a type of functional analysis (FA) that alternates a single

test and a single control condition, both comprehensively informed by an open-ended interview with relevant caregivers. In general, FAs rely on direct observation of problem behavior (PB) under various conditions in which environmental events are systematically manipulated to detect their influence on PB. FAs are commonly applied in research prior to treating PBs primarily associated with autism and intellectual disabilities (e.g., self-injurious behavior [SIB], aggression, property destruction, disruptive behavior; Beavers et al. 2013; Hanley et al. 2003). FAs have been shown to positively moderate the effects of behavioral interventions (Campbell 2003). In an IISCA, all of the co-occurring environmental events reported by caregivers to evoke and maintain PB are synthesized into a single reinforcement contingency. Whether this interview-informed, synthesized contingency influences PB is determined by rapidly alternating between test and control sessions in which the synthesized contingency is present or absent, respectively. The IISCA is a highly efficient, safe, and efficacious approach to understanding the context in which PB occurs and its likely reinforcement prior to its treatment (Hanley et al. 2014; Jessel et al. 2016; Santiago et al. 2016; Slaton et al. 2017).

One of the features of the IISCA that distinguishes it from other FAs of PB is the use of an individualized, single test condition that relies on a synthesized reinforcement contingency rather than multiple test conditions that attempt to isolate the influence of common reinforcement contingencies. For instance, rather than assessing whether PB is sensitive to attention, escape, or tangible reinforcement by evaluating each of these common reinforcement contingencies in distinct test conditions (e.g., Iwata et al. 1982/1994), an IISCA would determine if PB is sensitive to all three at the same time, if these events were reported by caregivers to co-occur following PB. IISCAs also attempt to closely emulate the specific manner in which reinforcers are established and delivered in natural contexts. An example of a synthesized contingency analysis that clearly reveals the effort to emulate the contextual features of the reinforcement contingency

is evident in a school-based analysis described by Ghaemmaghami et al. (2015). Based on the teacher's report of how she attempted to manage a 6-year-old boy's attempts to aggress or destroy property while maintaining the safety of her classroom, these authors arranged for the boy's PB to result in escape *from* group instruction *to* the opportunity to play with preferred toys while receiving undivided attention from the group's teacher. In other words, rather than seek to understand the main (isolated) effects of several generic reinforcement contingencies, the ecology under which PB is prevalent is more closely emulated by those conducting IISCAs, thereby allowing for possible and more powerful interactions to be revealed (see Slaton et al. 2017) and then leveraged to develop contextually relevant social skills that, due to their functional similarity, replace PB. In addition, rather than arrange putative reinforcement for only dangerous behavior (Iwata et al. 1982/1994), IISCAs arrange reinforcement for the target dangerous behavior (e.g., SIB) as well as all other forms of PB (i.e., precursor responses, e.g., yelling and foot stomping) that are reported to co-occur with the more dangerous forms of PB (Jessel et al. 2016). By opening the class of behavior topographies that may receive reinforcement in the FA, analyses are safer to conduct, and successful outcomes are more quickly achieved (Warner et al. 2019). Last, only one variable differs between the rapidly alternating test and control conditions of the IISCA: the presence or absence of the synthesized reinforcement contingency. Each distinguishing feature of the IISCA described above has precedent in the FA literature; however, the IISCA uniquely combines all of them in the initial attempt to analyze PB.

Historical Background

Lovaas et al. (1965) published the first FA of PB, in which they showed that rates of SIB exhibited by a child with schizophrenia increased when statements of concern followed instances of SIB, an initial example of control of PB via social-

positive reinforcement. Carr et al. (1976) provided evidence of the influence of social-negative reinforcement when they demonstrated that aggression and SIB could be evoked by the presentation of demands and that the termination of demands could serve as reinforcement. Sailor et al. (1968) demonstrated a functional relation between PB and a *synthesized* reinforcement contingency involving the simultaneous removal of difficult academic tasks and presentation of easier tasks with a child with mental retardation. After carefully describing general categories of reinforcement that have been shown to influence PB such as attention, escape, and direct sensory consequences, Carr (1977) provided a cogent argument that identifying environmental factors influencing SIB and other PB associated with developmental disabilities on a case-by-case basis may be a more fruitful pursuit than only attempting to understand the underlying biology or psychopathology of these same PBs. Following these recommendations, Iwata et al. (1982/1994) introduced a comprehensive model for analyzing SIB, one that is largely responsible for the apparent shift toward reinforcement-based interventions for PB based on its function and away from treatments relying on aversive procedures (Pelios et al. 1999).

The procedures outlined by Iwata et al. (1982/1994) are often referred to as the standard FA primarily because they occasioned hundreds of subsequent demonstrations of PB sensitivity to isolated environmental contingencies (Beavers et al. 2013; Hanley et al. 2003). These replications of single-subject analyses yielded useful baselines for developing an efficacious treatment technology (Kahng et al. 2002) and provided important information regarding *what was possible* with standard analyses (i.e., many different topographies of PB were shown to be influenced by attention, escape, tangibles, or direct sensory consequences across many individuals with intellectual disabilities). Relying on a consecutive-controlled case-series design in order to minimize any potential selection or publication bias favoring particular outcomes, Hagopian et al. (2013) more recently showed *what was probable* with a

standard FA. These authors reported that standard FAs were unable to detect a behavioral function in 53% of applications (94 of 176). Hagopian et al. showed that procedural modifications improved the ability of the analysis to detect behavioral function from 47% to 87%. In a review of published FAs, Schlichenmeyer et al. (2013) also described many of the unique, case-specific, incremental changes to the standard analysis model in the context of initial analysis failure. Noting the prevalence of particular changes to the standard FA that often yielded detection of behavioral function, Hanley (2010, 2012) suggested that these same changes be incorporated into initial analyses; the most important change being the incorporation of qualitatively unique reinforcement contingencies designed from interviews and observation into the initial FA. Hanley et al. (2014) then provided an empirical demonstration of the utility of these recommendations when they described the socially validated resolution of the PB of three children diagnosed with autism whose treatments were informed by analyses personalized from interviews. This type of analysis was then termed an IISCA (Jessel et al. 2016).

Rationale or Underlying Theory

A fundamental assumption in applying FA methodology is that PB is a learned or operant set of responses, ones that are maintained by their consequences. Certain events that are more likely to be experienced following PB than to be experienced in the absence of PB can serve to increase the future probability (i.e., reinforce) of PB. Certain events that enhance the value of those consequences (referred to as establishing operations [EOs]) and that signal their availability (referred to as discriminative stimuli [SDs]) join in the controlling relation referred to as a contingency. In other words, no matter how extraordinary PB may appear when exhibited by children with autism and related disabilities (e.g., attempts to bite, scratch, and punch others, or banging

one's head against hard objects), those responses are assumed to be sensitive to their consequences. The variability in the occurrence of those behaviors is thus related to the events that momentarily establish or abolish the value of those consequences and to the environmental cues that do or do not signal the opportunity for PB to generate those consequences.

For example, if a child with autism has a history of biting her father whenever she asks him to brush her hair, and that biting has reliably afforded her some respite from the hair brushing task, it may be the case that escape from the demand to complete that task is one of the reinforcers for her biting. Were that to be the case, one might consider the father's imposition of the demand to be an EO (i.e., an event that momentarily enhances the value of escape as a reinforcer) and the presence of the father to be an SD (i.e., a stimulus that signals an opportunity for the child to produce escape by biting). It would not be atypical for a father to assume that biting is a by-product of his daughter's autism, but the operant assumption and FA could reveal the simpler, more amenable controlling relation between the demands he imposes, her biting, and the contingent removal of those demands. By interviewing caregivers who have witnessed a great number of these PBs and the conditions under which they have occurred, an analyst can discover child-specific consequences that *may* be serving as reinforcement for the PB, as well as child-specific environmental events that seem to evoke the PB. By emulating these antecedent and consequent events in an analysis, the analyst may be able to *demonstrate* the influence of these events on PB.

Although there are hundreds of demonstrations of the influence of generic variables on PB (e.g., attention, escape, tangibles; Beavers et al. 2013; Hanley et al. 2003), caregiver descriptions of the contexts in which PB occurs are often complex, suggesting the influence of qualitatively rich contingencies unique to each individual (Bowman et al. 1997; see also Slaton et al. 2017). The IISCA assumes and emulates this complexity, and the interview is therefore a necessary starting

point in the assessment process. The qualitatively rich descriptions provided by caregivers inform the analysis so that each client experiences individualized conditions that closely emulate the typical context in which PB occurs.

Another assumption implicit in an IISCA is that controlling contingencies of PB are more likely to be composed of multiple contingencies operating simultaneously. There are multiple examples in the literature demonstrating the necessity of combining multiple contingencies to evoke (e.g., Dolezal and Kurtz 2010), reinforce (e.g., Call and Lomas-Mevers 2014), or maintain (e.g., Mann and Mueller 2009) PB in FAs (see Slaton & Hanley 2018 for a review of the nature and scope of synthesis in the analysis and treatment of PB). These analyses illustrated that a synthesis of variables may sometimes have a more powerful effect on behavior than its component parts. The holistic notion that whole contingencies have properties not present in their component parts and that whole contingencies are not always reducible to the study of the parts was evident in a participant from Hanley et al. (2014). The analysis revealed that PB exclusively occurred under conditions in which caregiver attention and toys were synthesized into a single reinforcement contingency. When isolated, attention and toys had no influence over PB (i.e., evidence of an interaction without main effects of each contingency). Slaton et al. (2017) provided additional demonstrations of this effect in which whole (synthesized) contingencies controlled behavior, but the same contingencies, when isolated, did not (see also Ghaemmaghami et al. 2015). There are also cases in which the suspected contingency for PB is inextricably synthesized, meaning that the consequent event being tested cannot possibly be broken down into component, isolated parts without fundamentally changing the nature of the contingency (e.g., Bowman et al. 1997; Eluri et al. 2016). Partly due to these analyses, suspected contingencies are usually synthesized in IISCAs.

Although the synthesis of variables in the IISCA precludes the ability to understand the

extent to which each individual contingency influences each PB topography in a single analysis, those who implement the IISCA favor the safety, speed, and consumer acceptability of the analysis over the need to understand the individual contributions of each contingency. Safety in IISCAs is partly achieved due to the synthesized contingency because when PB occurs in the analysis, it is essentially terminated through the provision of all possible reinforcers (Call & Lomas-Mevers 2014). The IISCA also engenders safety by combining all reported topographies of PB as well as their reported precursors into one large class of responses that will receive programmed reinforcement. For instance, if caregivers report that the most dangerous behaviors of SIB and aggression are to be analyzed and treated, but also indicate that their child reliably stomps her feet and whines before engaging in the more dangerous responses, all four of those responses will be eligible for reinforcement in the IISCA. By reinforcing the first emission of any member of the response class with all possible reinforcers, escalation of PB in the analysis is thwarted. This tactic not only minimizes escalation of PB and thereby promotes safety during the analysis, but it also does not appear to confuse interpretations regarding the variables controlling dangerous PB. A study by Warner et al. (2019) demonstrated that less dangerous responses reported to precede or co-occur with more dangerous forms were highly likely to be maintained by the same synthesized reinforcement contingency when all topographies were subjected to analysis. Inferences from open contingency classes and synthesized contingencies are therefore considered reasonable trade-offs for the lack of demonstration of control over each topography of PB or of the relevance of each component reinforcement contingency.

Goals and Objectives

The specific goals of the IISCA are to (a) demonstrate strong control of PB with a contingency that is composed of materials and

interactions described as related to PB in the initial caregiver interview, (b) obtain a stable baseline of PB from which to evaluate the efficacy of a function-based treatment, and (c) identify a motivating set of conditions in which to prompt and differentially reinforce important social skills with the same reinforcers that were shown to maintain PB. By controlling behavior with an ecologically relevant contingency, treatment is likely to have a good fit in the home, classroom, and community contexts in which PB was occurring. By obtaining a baseline, changes in the rate of PB can be quantified and connected to systematic changes in the treatment. By identifying a properly motivating set of teaching conditions, functional communication, delay and denial toleration, and cooperation with adult instruction can be expressly taught to individuals for whom typical social experiences have not yet yielded these skills (Hanley et al. 2014).

Assessment Participants

As of December 2019, there are roughly 189 published cases in which the IISCA procedures were applied, and 178 of them successfully demonstrated control over PB by a reinforcement contingency (see Coffey et al. 2020, for a recent review summarizing IISCA applications and outcomes). The cases included in these studies vary on (a) participant characteristics (e.g., age, language ability, diagnosis), (b) topographies of PB, and (c) settings in which the IISCA was applied.

At present, IISCA participants range in age from 1 to 35 years, with more male participants than female participants. The IISCA has proven efficacious with participants who show no verbal communication, engage in highly sophisticated speech, or display language profiles in between (e.g., can sign but cannot talk or can speak in one- or two-word utterances). The overwhelming majority of individuals for whom the IISCA was successfully applied had an autism diagnosis, but many of the individuals with autism had other comorbid diagnoses, and yet some other

individuals were differently diagnosed (e.g., attention deficit hyperactivity disorder, Fragile X syndrome, generalized anxiety disorder, intellectual disability). Additionally, the IISCA was conducted for some children without any formal diagnosis, but for whom PB was of major concern. The participant profile for which there are no data is typically developing adults.

Although the IISCA has predominantly been used to assess some combination of aggression, SIB, and disruptive behavior, other more idiosyncratic topographies have been successfully evaluated (e.g., elopement, flopping, inappropriate sexualized touching).

The IISCA has been applied primarily in specialized day programs and outpatient clinics. To a lesser extent, it has been applied in homes, residential programs, hospitals, and public schools (Coffey et al. 2020). Factors such as setting, type of PB, language ability, and diagnosis have yet to impede the ability of the IISCA to show PB's sensitivity to environmental contingencies.

Assessment Procedures

The IISCA begins with an open-ended, semi-structured interview (Hanley 2012) with those who regularly deal with the client's PB (i.e., relevant caregivers). Questions are asked regarding (a) the types of PBs and associated less dangerous behaviors, (b) the events that usually evoke these behaviors, and (c) the events that reliably follow the behaviors that lead to its abatement. The interviewer therefore attempts to identify potential (a) target responses to be reinforced in the test condition of the analysis, (b) situations to be presented in the test condition, and (c) reinforcers to be provided contingent on PB in the test condition and continuously in the control condition. Once the interview is complete, the analyst designs the IISCA to emulate caregivers' descriptions of events.

In the analysis, the analyst alternates between conditions in a multielement design, in which functional control over the suspected

reinforcement contingency may be shown with the participant serving as their own experimental control. Caregivers are present for the analysis, either as observers or implementers of the analysis. Feedback from the caregiver as to whether the context and interactions in the IISCA emulated typical circumstances is recruited. One 3-, 5-, or 10-min experience of a given condition constitutes a session. A common pattern of session alternation is control, test, control, test, test. The analysis often begins with the control condition. In the control condition, the client has non-contingent, continuous access to all of the suspected reinforcers for PB (e.g., toys, a tablet, adult availability and compliance, and the absence of demands). The analysis begins with the control condition to verify that PB will not occur under the conditions designed to eliminate all relevant establishing operations.

The test session begins in the same way that the preceding control session ended; the client has continuous access to all suspected reinforcers for PB. The analyst then presents the EO, which usually involves the simultaneous removal of any suspected positive reinforcers (e.g., toys, attention, ability to engage in stereotypy) and presentation of any putative aversive but routinely experienced events. Upon the first instance of any PB or associated less dangerous behavior, the analyst relents, removing any aversive stimuli (e.g., terminating demands) and delivering the suspected positive reinforcers, which essentially returns the client to the same conditions experienced in the control condition but for only 30 s to 1 min. The analyst then presents the EO again in the test session, and this process continues for a brief period, usually 5 min. Data on all PB types are measured and graphically depicted (data are typically plotted as a rate, i.e., PB per minute in each session). The analyst continues to rapidly alternate sessions until a clear difference in level, trend, and variability across test and control data paths is evident. A successful IISCA typically shows zero or near-zero levels of PB across control sessions and consistently elevated, but stable, rates of PB across test conditions (Jessel et al. 2016). If this outcome is not achieved within 45 min, then the IISCA is redesigned after

discussing the just witnessed behavior and additional interviewing.

Efficacy Information

Of the 189 published IISCAs in the literature, 178 were differentiated. Only a few authors have reported on how many iterations of the IISCA were necessary to produce a differentiated outcome, making it difficult to assess the extent to which IISCAs yield differentiated (useful) results on the first attempt. For example, Jessel et al. (2016) reported that 73% of cases (22 of 30) were initially successful. Within a consecutive-controlled case-series design, Slaton et al. (2017) reported that 100% of IISCAs (9 of 9) were initially successful (these authors also showed that standard analyses Iwata et al. (1982/1994) were successful across only 44% [4 of 9] of cases when applied to the same participants). More recently, Jessel et al. (2020) reported that 85% of cases (22 of 26) were initially successful.

Given that the IISCA process identifies unique contingencies through open-ended caregiver interviews, myriad types of synthesized contingencies have been demonstrated to be functionally related to PB. Many different combinations of social-positive, social-negative, and automatic reinforcement have been evaluated in a synthesized manner (e.g., escape from academic instruction to toys and unrestricted access to stereotypy), and the qualitative features of those contingencies vary across participants (i.e., that which clients escape from and to is often unique).

Considering that the ultimate goal of any FA is to design interventions that eliminate PB in relevant contexts with enduring meaningful effects, the treatment literature in which the IISCA was employed deserves mention. The IISCA has produced effective and socially meaningful outcomes in 100% of cases in which treatment was extended to relevant contexts and social validation of the outcomes was sought (i.e., implemented by relevant caregivers, in relevant settings, using practical schedules of reinforcement, e.g., Hanley et al. 2014; Jessel et al. 2018; Taylor et al. 2018). In Slaton et al. (2017), cases in which both types of

analyses – standard FAs and IISCAs – produced conclusive results were subsequently evaluated via a comparison of initial treatment effects. The authors found that treatments based off the IISCA were always successful in eliminating PB and developing a more appropriate replacement response for the learner, whereas treatments based off the standard FA were similarly successful in only half the applications.

Outcome Measurement

Five questions are considered when evaluating outcomes of the IISCA: (1) Does it produce a highly differentiated outcome (i.e., is strong control over PB shown in the IISCA)? (2) Is that outcome produced safely and quickly? (3) Do treatments designed from the IISCA yield efficacious outcomes (e.g., elimination of PB and acquisition of relevant skills)? (4) Are these efficacious outcomes successfully transferred to relevant people, contexts, and time frames? (5) Do treatments designed off the IISCA produce socially meaningful outcomes (i.e., is the amount of behavior change satisfying to caregivers)?

The 94% success rate of the IISCA in the literature suggests that it is highly likely to produce differentiated outcomes, providing robust evidence of the influence of personalized, synthesized contingencies on PB. Success in an analysis can be measured in myriad ways, but differentiation across test and control conditions is the basic requirement. Although it is possible that the documented success rate at present reflects a publication bias for demonstrations of functional control over behavior, the 100% success rates of the IISCAs reported in Jessel et al. (2018) and Slaton et al. (2017) – in which the consecutive-controlled case-series design was employed to explicitly address publication bias – speak to its efficacy.

Differentiated IISCA outcomes are achieved rapidly. According to Coffey et al. (2020), the median duration of published IISCAs is 25 min (range, 15 to 100 min), suggesting that the IISCA is a fast approach to analyzing PB. Recently, Jessel et al. (2019) demonstrated that IISCAs completed in as short as a single, 5-min session

could produce differentiation and could inform socially validated treatment outcomes.

The safety of IISCA procedures has yet to be quantified and measured, but the safety of an IISCA can be gleaned from the facts that (a) clients typically spend fewer than 30 min in the analysis, (b) PB is usually zero or near-zero in the control condition, and (c) the rate of PB in the test condition usually occurs around 2 per min, which, considering the length of the average reinforcement interval (e.g., 30 s), conveys that only single instances of PB often occur when an evocative situation is represented in test sessions. The unique commitment to the provision of *all* suspected reinforcers immediately following a single occurrence of *any* suspected member of the response class prevents escalation of PB once evoked. As opposed to reactive procedures like session termination criteria, these particular methods are the means by which participants, analysts, and caregivers are safeguarded in an IISCA.

Treatments explicitly designed to target the life skills of functional communication, toleration, and compliance with adult instruction have been highly efficacious in all cases when informed by an IISCA (e.g., Ghaemmaghami et al. 2016; Hanley et al. 2014; Jessel et al. 2018; Santiago et al. 2016; Slaton et al. 2017; Taylor et al. 2018). In addition, a few of those demonstrations extended their treatment to be implemented by relevant caregivers under relevant conditions (Hanley et al. 2014; Jessel et al. 2018; Santiago et al. 2016). All cases in which social validation was sought following the treatment development and extension process, it was achieved. The IISCA and subsequent skill-based treatment have been described as highly satisfactory processes with results that have improved the lives of the families for whom they were implemented.

Qualifications of Treatment Providers

The demonstrations of successful outcomes when using the IISCA in the literature have primarily involved implementation by a board

certified behavior analyst (BCBA[®]; www.bacb.com); however, it is encouraging to note that many of the IISCAs conducted in Jessel et al. (2016) and Slaton et al. (2017) were done so by relevant caregivers or teachers under the supervision of a BCBA. FAs require a sound understanding of the principles of learning derived from the field of behavior analysis. They also require the skills to naturally and systematically interact with a child or client in an attempt to emulate their ecology, repeatedly evoke and deescalate PB, and therefore understand PB in a scientific sense. It is recommended that an individual with little or no training in applied behavior analysis not undertake this procedure or only do so with some specific training or supervision of a BCBA.

References and Reading

- Beavers, G. A., Iwata, B. A., & Lerman, D. C. (2013). Thirty years of research on the functional analysis of problem behavior. *Journal of Applied Behavior Analysis*, 46, 1–21. <https://doi.org/10.1002/jaba.30>.
- Bowman, L. G., Fisher, W. W., Thompson, R. H., & Piazza, C. C. (1997). On the relation of mands and the function of destructive behavior. *Journal of Applied Behavior Analysis*, 30, 251–265. <https://doi.org/10.1901/jaba.1997.30-251>.
- Call, N. A., & Lomas Mevers, J. E. (2014). The relative influence of motivating operations for positive and negative reinforcement on problem behavior during demands. *Behavioral Interventions*, 29, 4–20.
- Campbell, J. M. (2003). Efficacy of behavioral interventions for reducing problem behavior in persons with autism: A quantitative synthesis of single-subject research. *Research in Developmental Disabilities*, 24, 120–138. [https://doi.org/10.1016/S0891-4222\(03\)00014-3](https://doi.org/10.1016/S0891-4222(03)00014-3).
- Carr, E. G. (1977). The motivation of self-injurious behavior: A review of some hypotheses. *Psychological Bulletin*, 84, 800.
- Carr, E. G., Newsom, C. D., & Binkoff, J. A. (1976). Stimulus control of self-destructive behavior in a psychotic child. *Journal of Abnormal Child Psychology*, 4, 139–153.
- Coffey, A. L., Shawler, L. A., Jessel, J., Nye, M. L., Bain, T. A., & Dorsey, M. F. (2020). Interview-informed synthesized contingency analysis (IISCA): Novel interpretations and future directions. *Behavior Analysis in Practice*, 13, 217–225.
- Dolezal, D. N., & Kurtz, P. F. (2010). Evaluation of combined-antecedent variables on functional analysis results and treatment of problem behavior in a school setting. *Journal of Applied Behavior Analysis*, 43, 309–314. <https://doi.org/10.1901/jaba.2010.43-309>.
- Eluri, Z., Andrade, I., Trevino, N., & Mahmoud, E. (2016). Assessment and treatment of problem behavior maintained by mand compliance. *Journal of Applied Behavior Analysis*, 49, 383–387. <https://doi.org/10.1002/jaba.296>.
- Fisher, W. W., Greer, B. D., Romani, P. W., Zangrillo, A. N., & Owen, T. M. (2016). Comparisons of synthesized- and individual-reinforcement contingencies during functional analysis. *Journal of Applied Behavior Analysis*, 49, 596–616. <https://doi.org/10.1002/jaba.315>.
- Ghaemmaghami, M., Hanley, G. P., Jin, S., & Vanselow, N. R. (2015). Affirming control by multiple reinforcers via progressive treatment analysis. *Behavioral Interventions*, 31, 70–86.
- Ghaemmaghami, M., Hanley, G. P., & Jessel, J. (2016). Contingencies promote delay tolerance. *Journal of Applied Behavior Analysis*, 49, 548–575. <https://doi.org/10.1002/jaba.333>.
- Hagopian, L. P., Rooker, G. W., Jessel, J., & DeLeon, I. G. (2013). Initial functional analysis outcomes and modifications in pursuit of differentiation: A summary of 176 inpatient cases. *Journal of Applied Behavior Analysis*, 46, 88–100.
- Hanley, G. P. (2010). Prevention and treatment of severe problem behavior. In E. Mayvill & J. Mulick (Eds.), *Behavioral foundations of effective autism treatment* (pp. 233–256). New York: Sloan.
- Hanley, G. P. (2012). Functional assessment of problem behavior: Dispelling myths, overcoming implementation obstacles, and developing new lore. *Behavior Analysis in Practice*, 5, 54–72.
- Hanley, G. P., Iwata, B. A., & McCord, B. E. (2003). Functional analysis of problem behavior: A review. *Journal of Applied Behavior Analysis*, 36, 147–185.
- Hanley, G. P., Jin, C. S., Vanselow, N. R., & Hanratty, L. A. (2014). Producing meaningful improvements in problem behavior of children with autism via synthesized analyses and treatments. *Journal of Applied Behavior Analysis*, 47, 16–36. <https://doi.org/10.1002/jaba.106>.
- Iwata, B. A., Dorsey, M. F., Slifer, K. J., Bauman, K. E., & Richman, G. S. (1994). Toward a functional analysis of self-injury. *Journal of Applied Behavior Analysis*, 27, 197–209. <https://doi.org/10.1901/jaba.1994.27-197>.
- Jessel, J., Hanley, G. P., & Ghaemmaghami, M. (2016). Interview-informed synthesized contingency analyses: Thirty replications and reanalysis. *Journal of Applied Behavior Analysis*, 49, 576–595. <https://doi.org/10.1002/jaba.316>.
- Jessel, J., Ingvarsson, E. T., Metras, R., Kirk, H., & Whipple, R. (2018). Achieving socially significant reductions in problem behavior following the interview-informed synthesized contingency analysis: A summary of 25 outpatient applications. *Journal of Applied Behavior Analysis*, 51, 130–157.

- Jessel, J., Hanley, G. P., Ghaemmaghami, M., & Metras, R. (2019). An evaluation of the single-session interview-informed synthesized contingency analysis. *Behavioral Interventions*, 34(1), 62–78.
- Jessel, J., Metras, R., Hanley, G. P., Jessel, C. & Ingvarsson, E. T. (2020). Evaluating the boundaries of analytic efficiency and control: A consecutive controlled case series of 26 functional analyses. *Journal of Applied Behavior Analysis*, 53, 25–43.
- Kahng, S., Iwata, B. A., & Lewin, A. B. (2002). Behavioral treatment of self-injury, 1964–2000. *American Journal on Mental Retardation*, 107, 212–221. [https://doi.org/10.1352/08958017\(2002\)107<0212:BTOSIT.2.0.CO;2](https://doi.org/10.1352/08958017(2002)107<0212:BTOSIT.2.0.CO;2).
- Lovaas, O. I., Freitag, G., Gold, V. J., & Kassorla, I. C. (1965). Experimental studies in childhood schizophrenia: Analysis of self-destructive behavior. *Journal of Experimental Child Psychology*, 2, 67–84.
- Mann, A. J., & Mueller, M. M. (2009). False positive functional analysis results as a contributor of treatment failure during functional communication training. *Education & Treatment of Children*, 32, 121–149. <https://doi.org/10.1353/etc.0.0044>.
- Pelios, L., Morren, J., Tesch, D., & Axelrod, S. (1999). The impact of functional analysis methodology on treatment choice for self-injurious and aggressive behavior. *Journal of Applied Behavior Analysis*, 32, 185–195.
- Sailor, W., Guess, D., Rutherford, G., & Baer, D. M. (1968). Control of tantrum behavior by operant techniques during experimental verbal training. *Journal of Applied Behavior Analysis*, 1, 237–243. <https://doi.org/10.1901/jaba.1968.1-237>.
- Santiago, J. L., Hanley, G. P., Moore, K., & Jin, C. S. (2016). The generality of interview-informed functional analyses: Systematic replications in school and home. *Journal of Autism and Developmental Disorders*, 46, 797–811.
- Schlichenmeyer, K. J., Roscoe, E. M., Rooker, G. W., Wheeler, E. E., & Dube, W. V. (2013). Idiosyncratic variables that affect functional analysis outcomes: A review (2001–2010). *Journal of Applied Behavior Analysis*, 46, 339–348. <https://doi.org/10.1002/jaba.12>.
- Slaton, J. D., & Hanley, G. P. (2018). Nature and scope of synthesis in functional analysis and treatment of problem behavior. *Journal of Applied Behavior Analysis*, 51(4), 943–973.
- Slaton, J. D., Hanley, G. P., & Raftery, K. J. (2017). Interview-informed functional analyses: A comparison of synthesized and isolated components. *Journal of Applied Behavior Analysis*, 50, 252–277. <https://doi.org/10.1002/jaba.384>.
- Taylor, S. A., Phillips, K. J., & Gertzog, M. G. (2018). Use of synthesized analysis and informed treatment to promote school reintegration. *Behavioral Interventions*, 33(4), 364–379.
- Warner, C. A., Hanley, G. P., Landa, R. K., Ruppel, K. W., Rajaraman, A., Ghaemmaghami, M., ... & Gover, H. C. (2019). Toward accurate inferences of response class membership. *Journal of Applied Behavior Analysis*, 53, 331–354.

Intestinal Permeability Studies

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Definition

Intestinal permeability (IP) refers to the degree of “leakiness” of the intestinal mucosal barrier. The intestinal epithelium regulates the movement of fluids and electrolytes into and out of the gut. It also serves as a barrier to foreign antigens and toxins.

Historical Background

Autism spectrum disorder (ASD) is a complex neurodevelopmental disorder characterized by deficits in social reciprocity, disordered language, and restricted interests and/or repetitive behaviors. Gastrointestinal dysfunction has been reported in 9–51% of children with ASD, with a lifetime prevalence as high as 70%. Children with ASD have been noted to have diarrhea, constipation, gastroesophageal reflux (GER), and abdominal pain. Dietary restriction is common, and up to 33% of families try a gluten- and casein-free (GFCF) diet to address GI symptoms with hopes of improving the behavioral symptoms of ASD. Advocates of the GFCF diet postulated that gluten and casein proteins are incompletely broken down in the intestine, absorbed by a leaky gut, and become “neuroactive peptides” that contribute to the symptoms of autism. Urine peptide analyses in people with ASD collected in the 1990s initially identified compounds that were felt to be opioid peptides found in gluten and casein. The hypothesis arose that these compounds were absorbed across a “leaky gut.” Subsequent studies using newer techniques of mass spectroscopy could not confirm that there were increased amounts of these peptides in the urine of people with ASD. Intestinal permeability has been found to be

increased in many medical conditions including gastrointestinal disorders such as celiac disease, autoimmune disorders such as diabetes, and atopic diseases such as eczema.

Current Knowledge

The intestinal barrier is important for protection from infection and also to maintain tolerance to most of the antigens that the gut is exposed to. Tight junctions are important in regulating movement of water, nutrients, and immune cells between intestinal cells (Lu et al. 2000). The majority of proteins that could result in immune activation cross the intestinal barrier by going through the cells. During this process, the proteins are degraded into smaller, less immunogenic peptides. The rest cross as intact proteins between the cells. When the tight junctions are disrupted, a large number of immunogenic proteins may cross the mucosal barrier, and it is hypothesized that an immune response may develop (Clemente et al. 2003). Increased intestinal permeability has been implicated in the development of type 1 diabetes in individuals who are genetically predisposed. Another study found increased IP in individuals with food allergy and food hypersensitivity (Ventura et al. 2006).

Intestinal permeability (IP) is typically measured by drinking two sugars and measuring their excretion in the urine. One is a large disaccharide such as lactulose that is absorbed from the gut via a paracellular (between the cells) pathway. The other is a smaller monosaccharide such as mannitol which is absorbed from the gut mainly via a transcellular (through the cell) pathway. If the junctions between intestinal epithelial cells are intact, less lactulose will be absorbed and excreted in the urine. If the intestine is more permeable or “leaky,” more lactulose will be absorbed via the paracellular pathway, leading to a higher lactulose to mannitol ratio in the urine.

Five studies have evaluated intestinal permeability in individuals with ASD (D’Eufemia et al. 1996; de Magistris et al. 2010; Horvath and Perman 2002; Kemperman et al. 2008; Robertson et al. 2008). D’Eufemia found abnormal intestinal

permeability in 9 of 21 children with high-functioning autism without gastrointestinal symptoms, compared to control subjects (D’Eufemia et al. 1996). Horvath et al. (2000) also found increased permeability in 19 of 25 children. Kemperman (2007) did not find elevated permeability in 21 children with pervasive developmental disorder, and Robertson et al. (2008) also did not find a difference in IP in 14 children with autism and GI symptoms compared to 7 sibling and 8 typical controls. None of these studies used research-reliable diagnoses of ASD. Horvath and Kemperman did not use control groups. Only D’Eufemia addressed asthma and eczema symptoms as a potential confounding variable. de Magistris et al. (2010) found abnormal IP in 37% of 90 subjects with autism, 21% of 146 first-degree relatives, and 5% of controls. The difference was significant with a p value <0.0001 . Twenty-five percent of the children with ASD were on a gluten-casein-free diet. Those children had a lower IP than those on a regular diet, $p = 0.034$. They did rule out celiac disease but did not get a history of allergy/atopy. They noted no difference in GI symptoms in the group with abnormal IP compared to the group with normal IP. In that study, increased permeability seems to function as an endophenotype (a phenotype/characteristic that is found in unaffected family members at a higher rate than the general population and felt to possibly increase the risk of developing the disease). While the most recent study (de Magistris et al. 2010) of intestinal permeability in children with ASD did have a well-characterized sample with adequate controls, the authors point out that there has been no evidence that increased permeability is associated with neuropsychiatric manifestations or even gastrointestinal disorders. A recent review of gastrointestinal issues in children with ASD agreed with this point as well (Buie et al. 2010).

Future Directions

Future studies should evaluate the impact of increased permeability on health and neuropsychiatric outcomes. It is plausible that embryologic insults may impact development of intestine and

brain, and it is also plausible that genetic disorders alter function in both gastrointestinal and central nervous systems. Studies investigating the immunologic and microbiologic environment of the intestine are ongoing.

See Also

- [Gastrointestinal Disorders and Autism](#)
- [Gluten-Free Diet](#)
- [Leaky Gut Syndrome](#)

References and Reading

- Buie, T., Campbell, D. B., Fuchs, G. J., 3rd, Furuta, G. T., Levy, J., Vandewater, J., et al. (2010). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125(Suppl. 1), S1–S18.
- Clemente, M. G., De Virgiliis, S., Kang, J. S., Macatagney, R., Musu, M. P., Di Pierro, M. R., et al. (2003). Early effects of gliadin on enterocyte intracellular signalling involved in intestinal barrier function. *Gut*, 52(2), 218–223.
- D'Eufemia, P., Celli, M., Finocchiaro, R., Pacifico, L., Viozzi, L., Zaccagnini, M., et al. (1996). Abnormal intestinal permeability in children with autism. *Acta Paediatrica*, 85(9), 1076–1079.
- de Magistris, L., Familiari, V., Pascotto, A., Sapone, A., Frolli, A., Iardino, P., et al. (2010). Alterations of the intestinal barrier in patients with autism spectrum disorders and in their first-degree relatives. *Journal of Pediatrics Gastroenterology and Nutrition*, 51(4), 418–424.
- Horvath, K., & Perman, J. A. (2002). Autistic disorder and gastrointestinal disease. *Current Opinion in Pediatrics*, 14(5), 583–587.
- Horvath, K., Zielke, R. H., Collins, R. M., Rabsztyan, A., Medeiros, L. A., & Perman, J. (2000). Secretin improves intestinal permeability in autistic children. *Journal of Pediatric Gastroenterology and Nutrition*, 31(Suppl. 2), A112A112(Abstract).
- Kemperman, R. (2007). *Nutrition and biomarkers in psychiatry: Research on micronutrient deficiencies in schizophrenia, the role of the intestine in the hyper-serotonemia of autism, and a method for non-hypothesis driven discovery of biomarkers in urine*. Dissertation, University of Groningen. Retrieved from <http://irs.ub.rug.nl/ppn/305278908>. Accessed 6 June 2012.
- Kemperman, R. F., Muskiet, F. D., Boutier, A. I., Kema, I. P., & Muskiet, F. A. (2008). Brief report: Normal intestinal permeability at elevated platelet serotonin levels in a subgroup of children with pervasive developmental disorders in Curacao (The Netherlands antilles). *Journal of Autism and Developmental Disorders*, 38(2), 401–406.
- Lu, R., Wang, W., Uzzau, S., Vigorito, R., Zielke, H. R., & Fasano, A. (2000). Affinity purification and partial characterization of the zonulin/zonula occludens toxin (Zot) receptor from human brain. *Journal of Neurochemistry*, 74(1), 320–326.
- Robertson, M. A., Sigalet, D. L., Holst, J. J., Meddings, J. B., Wood, J., & Sharkey, K. A. (2008). Intestinal permeability and glucagon-like peptide-2 in children with autism: A controlled pilot study. *Journal of Autism and Developmental Disorders*, 38(6), 1066–1071.
- Ventura, M. T., Polimeno, L., Amoroso, A. C., Gatti, F., Annoscia, E., Marinaro, M., et al. (2006). Intestinal permeability in patients with adverse reactions to food. *Digestive and Liver Disease*, 38(10), 732–736.

Intimate Relationships and Dating

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Definition

Intimate relationships, including marriage and long-term dating relationships, are close relationships that include physical and/or emotional intimacy. Typically, intimate relationships have a sexual component. There has been limited research on intimate relationships in autism spectrum disorder (ASD), but it appears that people with ASD may have more difficulty achieving intimate relationships than typically developing people. Certainly, some autistic people marry, but it appears that many people with ASD do not achieve the intimate relationship they desire.

Historical Background

Historically, sexuality was regarded as a taboo topic within the content of developmental disabilities and autism. For years, our knowledge about

sexuality and sexual expression in this population was limited by our reliance on caregiver reports describing institutionalized sample populations. By relying on these samples, the field ran the risk of reporting inaccurate rates of sexual behavior, attraction, and identity (Hellemans et al. 2007; Stokes and Kaur 2005; Haracopos and Pedersen 1992). For instance, the prevalence of asexuality, or the lack of sexual interest or desire for a sexual relationship, is reported by some early studies to be significantly higher in ASD than in the general population. In many studies, particularly older studies, the data on sexual activity among people with ASD were collected via parent report, which may not be accurate.

Current Knowledge

Sexuality, by definition, is a social activity. There appear to be minimal differences in physical maturation between typically developing individuals and those with ASD.

Differences in social reciprocity, social communication, and ability to develop interpersonal relationships – inherent in ASD – surely affect sexuality (Nichols and Blakeley-Smith 2009), and research suggests that higher levels of ASD traits are associated with greater loneliness and fewer and lower quality relationships (Jobe and White 2007). Deficits in social cognition may have particular impact. Perspective taking, or theory of mind (ToM), is crucial in understanding the thoughts, feelings, and behaviors of other people and helps individuals successfully navigate social situations (Doherty 2008). Individuals with autism often display underdeveloped ToM, and have trouble taking other's perspectives (Baron-Cohen et al. 1985; Frith and Happé 1994). It is understandable then that despite their desire to have social relationships, individuals with autism report experiencing higher rates of loneliness than their peers and having fewer friendships (Jobe and White 2007; Bauminger and Kasari 2000). Because theory of mind is essential for understanding social situations, individuals with ASD can

misattribute other's intentions and endorse negative social biases about themselves or others (Blackshaw et al. 2001; Barnhill 2001; Van Roekel et al. 2010). To further understand social deficits in autism, it is imperative that comorbid psychopathologies, like social anxiety (one of the most common comorbid disorders within the ASD population), be taken into consideration (White et al. 2009; Simonoff et al. 2008).

The presence of autism can interfere with the social and cognitive processes under development during puberty, often resulting in a mismatch between a person's physical readiness for sexual intercourse and the individual's socio-sexual understanding required to appropriately engage in these behaviors. Given social and cognitive deficits, it may be hard for children to actively engage with peers, thus reducing their reliance on peers for learning "proper" skills for engaging in social and romantic relationships (Stokes et al. 2007). Despite these difficulties, however, many individuals with ASD express interest in developing and participating in these types of relationships, as well participate in solitary sexual behaviors, like masturbation (Hellemans et al. 2007, 2010; Haracopos and Pedersen 1992; Van Bourgondien et al. 1997; Ousley and Mesibov 1991; Gilmour et al. 2012).

There may be some difference in the type of relationship that a person with ASD pursues. In recent years, studies assessing the identity and behaviors of those actually diagnosed with ASD have found mixed results regarding sexual orientation identification, with some reporting higher sexual minority rates within the community, while others suggest heteronormative sexual interest (Byers et al. 2013; Gilmour et al. 2012; Fernandes et al. 2016). Despite these advances in understanding socio-sexual development, there is still much to learn about the formation of intimate relationships and dating behaviors of individuals with autism.

Future Directions

More and better research to understand perspectives of people with ASD, their sexual behavior,

identification, and attraction is essential to developing interventions to support healthy relationship development. Investigation into the connection, if any, between transgender identity and autism is needed. Anecdotal and emerging empirical evidence suggests a higher rate of trans identity among people with ASD. We do know that trans youth are at risk of bullying, social rejection, and psychopathology, and it is important for ASD providers to be aware and sensitive to the needs of this subset of the ASD population.

Sex education and instruction in intimate relationship development is another domain where more research is needed. Little research has been done to investigate effective sex education programming, yet a number of recent publications offer tips for dating “on the spectrum.” It will be useful to evaluate curricula for dating skills, sex education programs, and implications for people with ASD. For instance, one investigation (Roth and Gillis 2015) looked at use of online dating among people with ASD and found safety concerns.

As methods of social cognitive skills instruction advance, the application of these skills to developing intimate relationships will be an important area of investigation.

See Also

- ▶ [Counterfeit Deviance](#)
- ▶ [Sex Education](#)
- ▶ [Sexuality in Autism](#)

References and Reading

- Barnhill, G. P. (2001). Social attributions and depression in adolescents with Asperger syndrome. *Focus on Autism and Other Developmental Disabilities*, 16(1), 46–53.
- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a “theory of mind”? *Cognition*, 21(1), 37–46.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development*, 71(2), 447–456.
- Blackshaw, A. J., Kinderman, P., Hare, D. J., & Hatton, C. (2001). Theory of mind, causal attribution and paranoia in Asperger syndrome. *Autism*, 5(2), 147–163.
- Byers, E. S., Nichols, S., Voyer, S. D., & Reilly, G. (2013). Sexual well-being of a community sample of high-functioning adults on the autism spectrum who have been in a romantic relationship. *Autism*, 17(4), 418–433.
- Dewinter, J., Vermeiren, R., Vanwesenbeeck, I., & Nieuwenhuizen, C. (2013). Autism and normative sexual development: A narrative review. *Journal of Clinical Nursing*, 22(23–24), 3467–3483.
- Dewinter, J., Vermeiren, R., Vanwesenbeeck, I., Lobbetael, J., & Van Nieuwenhuizen, C. (2015). Sexuality in adolescent boys with autism spectrum disorder: Self-reported behaviours and attitudes. *Journal of Autism and Developmental Disorders*, 45(3), 731–741.
- Doherty, M. (2008). *Theory of mind: How children understand others' thoughts and feelings*. Psychology Press.
- Fernandes, L. C., Gillberg, C. I., Cederlund, M., Hagberg, B., Gillberg, C., & Billstedt, E. (2016). Aspects of sexuality in adolescents and adults diagnosed with autism spectrum disorders in childhood. *Journal of Autism and Developmental Disorders*, 46(9), 3155–3165.
- Frith, U., & Happé, F. (1994). Autism: Beyond “theory of mind”. *Cognition*, 50(1), 115–132.
- Gilmour, L., Schalomon, P. M., & Smith, V. (2012). Sexuality in a community based sample of adults with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 6(1), 313–318.
- Haracopos, D., & Pedersen, L. (1992). Sexuality and autism: A nationwide survey in Denmark. Unpublished manuscript.
- Hellemans, H., Colson, K., Verbraeken, C., Vermeiren, R., & Deboutte, D. (2007). Sexual behavior in high-functioning male adolescents and young adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37(2), 260–269.
- Hellemans, H., Roeyers, H., Leplae, W., Dewaele, T., & Deboutte, D. (2010). Sexual behavior in male adolescents and young adults with autism spectrum disorder and borderline/mild mental retardation. *Sexuality and Disability*, 28(2), 93–104.
- Jobe, L. E., & White, S. W. (2007). Loneliness, social relationships, and a broader autism phenotype in college students. *Personality and Individual Differences*, 42(8), 1479–1489.
- Murphy, N. A., & Elias, E. R. (2006). Sexuality of children and adolescents with developmental disabilities. *Pediatrics*, 118(1), 398–403.
- Nichols, S., & Blakeley-Smith, A. (2009). “Im not sure we’re ready for this. . .”: Working with families toward facilitating healthy sexuality for individuals with autism spectrum disorders. *Social Work in Mental Health*, 8(1), 72–91.
- Ousley, O. Y., & Mesibov, G. B. (1991). Sexual attitudes and knowledge of high-functioning adolescents and

adults with autism. *Journal of Autism and Developmental Disorders*, 21(4), 471–481.

- Roth, M. E., & Gillis, J. M. (2015). “Convenience with the click of a mouse”: A survey of adults with autism spectrum disorder on online dating. *Sexuality and Disability*, 33(1), 133–150.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(8), 921–929.
- Stokes, M. A., & Kaur, A. (2005). High-functioning autism and sexuality: A parental perspective. *Autism*, 9(3), 266–289.
- Stokes, M., Newton, N., & Kaur, A. (2007). Stalking, and social and romantic functioning among adolescents and adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37(10), 1969–1986.
- Van Bourgondien, M. E., Reichle, N. C., & Palmer, A. (1997). Sexual behavior in adults with autism. *Journal of Autism and Developmental Disorders*, 27(2), 113–125.
- Van Roekel, E., Scholte, R. H., & Didden, R. (2010). Bullying among adolescents with autism spectrum disorders: Prevalence and perception. *Journal of Autism and Developmental Disorders*, 40(1), 63–73.
- White, S. W., & Roberson-Nay, R. (2009). Anxiety, social deficits, and loneliness in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(7), 1006–1013.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229.

Intonation

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Synonyms

[Prosody](#); [Tone of voice](#)

Definition

Intonation refers to the melodic patterns of the voice which are not used to differentiate words. It can be used to indicate the function of a sentence (e.g., statement, question, sarcastic comment), the speaker’s affect, or to emphasize important topics in a conversation. Individuals with autism spectrum disorder often have atypical intonation patterns, either using little intonation, resulting in a flat or robotic sound, or using exaggerated intonation, which can sound unusual as well. These characteristic intonation patterns can be diagnostic markers for autism spectrum disorder.

See Also

- [Monotone](#)
- [Prosody](#)

References and Reading

- Diehl, J. J., & Berkovits, L. (2010). Is prosody a diagnostic and cognitive bellwether of autism spectrum disorders? In A. Harrison (Ed.), *Speech disorders: Causes, treatments, and social effects* (pp. 159–176). New York: Nova Science.
- Grossman, R. B., Edelson, L. R., & Tager-Flusberg, H. (2013). Emotional facial and vocal expressions during story retelling by children and adolescents with high-functioning autism. *JSLHR*, 56, 1035–1044.
- McCann, J., & Peppé, S. (2003). Prosody in autism spectrum disorders: A critical review. *International Journal of Language and Communication Disorders*, 38(4), 325–350.
- Paul, R., Augustyn, A., Klin, A., & Volkmar, F. R. (2005). Perception and production of prosody by speakers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 35(2), 205–220.
- Shriberg, L. D., Paul, R., McSweeney, J. L., Klin, A., Cohen, D. J., & Volkmar, F. R. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning autism and Asperger syndrome. *Journal of Speech, Language, and Hearing Research*, 44(5), 1097–1115.

Intraventricular Hemorrhage

► [Periventricular Hemorrhage](#)

Intraverbals

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Definition

Intraverbals are a listener's verbal responses to another speaker (Skinner 1957). These responses include answers to questions (e.g., a speaker asks, "Where are you going?" The listener replies with an intraverbal, "Home.") and responses to comments (e.g., a speaker says, "I like that one." The listener replies with an intraverbal, "Me too." or "I don't like it."). Intraverbal behaviors are an important component of reciprocal (back-and-forth) social communication (Mudford et al. 2009). Social communication is most effective when, individuals respond appropriately to each other's questions and comments.

See Also

► [Mands](#)

References and Reading

- Mudford, O., Ford, E., & Arnold-Saritepe, A. (2009). Efficacy of interventions to promote language. In A. Fitzler & P. Sturmey (Eds.), *Language and autism: Applied behavior analysis, evidence, and practice* (pp. 79–106). Austin: PRO-ED.
- Skinner, B. F. (1957). *Verbal behavior*. New York: Appleton-Century-Crofts.

Introduction to the Delis Kaplan Executive Function System

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Synonyms

[D-KEFS](#)

Description

The Delis-Kaplan Executive Function System (D-KEFS) consists of nine individual tests designed to assess aspects of executive function skills in children and adults ranging from age 8 to 89. Executive function (EF) skills are the mental processes that enable individuals to regulate their thoughts and behaviors. Specific EF skills include planning, focusing attention, remembering instructions, and switching between multiple ideas or tasks successfully. The Center on the Developing Child at Harvard University (2011) likens executive functioning to the brain's "air traffic control system," positing that, like the control center at a busy airport coordinating arrivals and departures, the brain relies on executive function skills to filter distractions, prioritize tasks, set and achieve goals, and control impulses. These discreet skills serve as the foundational cognitive resources for aspects of daily functioning including academic, cognitive, and social functioning.

The D-KEFS measures two general types of executive functioning skills including fundamental cognitive skills (e.g., attention, perception, and language) along with higher-level cognitive processes (e.g., concept formation, inhibition, planning, and cognitive flexibility). The tests that comprise the D-KEFS facilitate a cognitive process approach allowing for the examination of fundamental component skills along with their contributions to higher-order cognitive processes.

Introduction to the Delis Kaplan Executive Function System, Table 1 D-KEFS test description and associated EF skills

D-KEFS tests		
Test	Description	Executive functions tapped
Word context test	Requires the examinee to use verbal cues to deduce to meaning of made-up words	Deductive reasoning Integrating multiple pieces of information Generation (i.e., hypothesis testing) Cognitive flexibility (switching)
Sorting test	Requires the examinee to repeatedly sort and describe a set of cards based on attributes of shape, color, writing, or word meaning. The second part of this test requires the examinee to describe examiner created sorts	Verbal conceptual reasoning Nonverbal conceptual reasoning Generation Cognitive flexibility Inhibition
Twenty questions test	Requires examinee to look at an array of pictures and ask up to 20 yes/no questions to guess which picture the examiner has chosen	Establish/maintain cognitive set (working memory) Monitoring Category recognition Ability to use feedback (correct) Inhibition Planning (i.e., formulate abstract yes/no questions that eliminate the maximum number of objects)
Tower test	Requires examinee to rearrange a set of disks placed on rods to match a predetermined arrangement. The examinee must keep in mind several rules while completing the task	Spatial planning Monitoring Inhibition Cognitive flexibility Establish/maintain cognitive set
Color-word interference test	Requires examinee to: <i>Condition 1:</i> Name colors <i>Condition 2:</i> Read color words <i>Condition 3:</i> Look at color names that are printed in different color inks (e.g., the word “red” in blue ink) and name the color of the ink instead of reading the word <i>Condition 4:</i> Switch between reading the ink color and reading the color word	Monitoring Inhibition Cognitive flexibility Establish/maintain cognitive set
Verbal fluency test	Requires examinee to generate words by first letter, by category, and then by alternating between two categories	Verbal knowledge Systematic retrieval of lexical items Monitoring Cognitive flexibility Establish/maintain cognitive set
Design fluency test	Requires examinee to create unique designs among series of dots using specified number of straight lines. Dots are either filled or empty, and the task builds from single fills (only empty or only filled) into a switching task of between filled dots and empty dots	Cognitive flexibility Monitoring Inhibition Establish/maintain cognitive set
Trail making test	Requires examinee to create a trail with their pencil by connecting numbers, letters, and then alternating numbers and letters. A measure of connecting dots (motor speed) is also included	Cognitive flexibility Inhibition Establish/maintain cognitive set
Proverb test	Requires examinee to interpret proverbs	Verbal abstract thinking Semantic integration Generalization

Each of the nine individual tests taps numerous executive functioning skills (see Table 1 for test descriptions and associated EF skills assessed),

and most involve the administration of multiple conditions. Thus, each test provides a wide spectrum of cognitive variables including measures of

component skills, problem-solving strategies, temporal aspects of response patterns, ratio measures, and various error types. Values obtained across these measures compare an individual's performance to the normative sample for a particular indicator and can assist clinicians in two important ways. First, the D-KEFS allow for the documentation of an individual's unique profile of spared and impaired executive functions. Second, they can support the identification of the neurocognitive mechanisms that underlie an individual's deficient performance by noting patterns of performance across tasks. Performance on these tasks can be impacted by skill deficits at any stage of cognitive processing from lower-level basic skills to higher-level cognitive functions (Delis et al. 2001a).

The D-KEFS tests are considered useful indicators of executive functioning skills for a variety of reasons. They are notable for their sensitivity to detect even mild vulnerabilities in EF skills offering "lower floors" and "higher ceilings" as compared to previous versions of similar tasks (Delis et al. 2001a). The developers have paid particular attention to the measurement of skills that would indicate frontal lobe involvement (e.g., attention and inhibition). Design features also include the incorporation of "switching conditions" across the various subtests that allow multiple opportunities to assess cognitive flexibility or the ability to shift between sets (e.g., rules, categories, or response sets). Additionally, the presence of "capture stimuli" is embedded across multiple tasks to pull for concrete stimulus-bound responding allowing for the detection of basic impulse control deficits. Finally, the testing procedures gradually increase the processing demands placed on an individual, allowing examiners to detect patterns of responding and thresholds of stimulation that appear to be best tolerated by an individual (Delis et al. 2001a). For example, an individual might have greater difficulty on more basic tasks but demonstrate improved performance as the level of challenge and stimulation increases. Such individuals require a high degree of cognitive stimulation to recruit their most effective EF skills. In contrast, for another individual, performance may decline as task complexity increases indicating deficits in more fundamental cognitive skills.

The ability to detect such patterns of performance provides insight into the cognitive processes of individuals and is instrumental for both diagnosis and the development of targeted interventions to improve functioning.

Historical Background

The nine tests composing the D-KEFS represent a combination of tests developed by authors Edith Kaplan and Dean Delis along with their adaptations of tests developed from long-standing clinical instruments and procedures implemented in past experimental studies (Delis et al. 2001a; Homack et al. 2005). Two of the nine tests were developed by the D-KEFS authors. The Word Context Test was designed by Edith Kaplan in the 1940s. Working in collaboration with the developmental psychologist Heinz Werner, Kaplan developed the test to study young children's acquisition of word meanings (Werner and Kaplan 1952). For the present D-KEFS battery, the authors adapted the original version to offer a suitable assessment tool for use with both children and adults. The precursor to the D-KEFS Sorting Test was developed by Dean Delis in the late 1980s (Delis et al. 1995) in the form of the California Sorting Test. This test was later revised to increase the tool's sensitivity and complexity by incorporating more sorting concepts across multiple sets of cards.

The seven remaining D-KEFS tests are modifications of tests developed by authors other than Dean Delis or Edith Kaplan. For example, the Color-Word Interference Test is a modification of the classic Stroop test developed in the 1930s (Stroop 1935). The basic paradigm of the Stroop test compares an individual's performance on a basic task (e.g., reading names of colors) to their performance on an analogous task in which a habitual response needs to be suppressed in support of an unusual one (i.e., naming the ink color that incongruously named color words are printed in). Many different Stroop test versions have been developed over time (e.g., Comalli et al. 1962; Golden 1978), with variations in the color and number of the test items, the number of subtests, and the administration procedure. Delis and

Kaplan increased the complexity of the original task by developing a series of conditions (see Table 1) that allow for both the measurement of basic naming skills as well as complex cognitive flexibility and inhibitory skills creating increasingly difficult conditions in which individuals must alternate between reporting the word written on the page and the color of the ink.

In a similar vein, the Twenty Questions Test is a modification of a popular guessing game often played by children or adults in which an individual must pose a series of yes/no questions in order to identify an unknown entity. The task was first used as a test procedure for youth in the 1960s (see Baron 2004). It has been modified by Delis and Kaplan to provide systematic means of isolating specific skill deficits and identifying the strategies individuals employed to engage in such higher-level problem-solving tasks. For the D-KEFS version, the authors designed a page with pictures of 30 stimuli that requires the examinee to ask questions that eliminate as many stimuli as possible so that a target stimulus can be correctly identified.

The Tower Test is also a modification of earlier tasks used in experimental studies. This D-KEFS test is similar to earlier Tower Tests (e.g., Tower of Hanoi; Welsh 1991, Tower of London; Anderson et al. 1996). Among the modifications, the D-KEFS version adds process measures such as first move completion time, total number of moves, item completion time, final achievement, and number of rule violations.

A description of each test's origin and history is beyond the scope of the present chapter but can be found in the D-KEFS Examiner's Manual (Delis et al. 2001a) as well in a detailed review of the measure published by Ida Sue Barron (2004).

Psychometric Data

The D-KEFS was standardized on a nationally representative sample and stratified sample of 1750 children, adolescents, and adults, ages 8 to 89 years (Delis et al. 2001b). Stratification was

based on the 2000 US Census figures and considered age, sex, race/ethnicity, years of education, and geographic region (Delis et al. 2001b). D-KEFS normative data for each measure were analyzed for age trends, including error measures and scores presented in cumulative frequency distributions. According to the authors, the age effects were evaluated using linear and nonlinear regression techniques. In general, children from the younger age groups (8–10 years) and adults from the oldest age groups (70–89 years) tended to obtain lower raw scores on the D-KEFS executive function measures as compared to examinees from the adolescent, young adult, and middle-aged groups. D-KEFS variables that measure more fundamental verbal skills (e.g., verbal fluency measures) tended to be associated with steep rises in raw scores across the younger age groups, but there was a slower decline in the older age groups (Delis et al. 2001a). Performance on measure that is highly dependent on speed of processing tended to peak in the adolescent and young-adult groups, whereas performance on tests of verbal conceptual reasoning tended to peak in the adult age group. These trends strengthen the construct validity of the D-KEFS, as performance across age ranges is consistent with literature on developmental cognitive processes and the trajectories of skill development across domains over the life course (Delis et al. 2001b). In general, the standard deviations of the D-KEFS measures tended to be larger in the youngest and oldest age groups than in the adolescent, young-adult, and middle-aged groups. For children, these findings are consistent with variable rates of development of cognitive skills, including EF. For elderly adults these finding may reflect variability in the normal aging process and/or subclinical neurological processes (Delis et al. 2001b).

Reliability

Reliability indicators including internal consistency, stability coefficients, and alternate form reliability, which established standard error of measurement and confidence intervals, are

presented by the test developers (see Delis et al. 2001a). Test-retest reliability was estimated with an average time between administrations of 25 days. The majority of the test-retest coefficients scores fell in the moderate range and varied substantially across tests. Twenty Questions (0.24 to 0.43) and Design Fluency (0.32 to 0.58) were among the lowest estimates, whereas Word Context (0.70) and Proverb Test (0.76) were among the highest. Measures of internal consistency also varied considerably across the individual tests and within individual tests based on age groups and testing condition. Some of the lowest internal consistency values were observed on the Twenty Questions Test (0.10 to 0.55 across age groups), while the highest values observed on the Color-Word Interference Test (0.62 to 0.86), Verbal Fluency Test (0.32 to 0.90), and Trail Making Test (0.57 to 0.81). In a critique of the measure, Schmidt (2003) noted that only 17% of the reliability values published in the D-KEFS manual were above a 0.80 value. However, the authors maintain that such values are to be expected based on the task complexity and multifaceted nature of EF skills (Delis et al. 2004).

Validity

The D-KEFS manual provides few validity studies, focusing primarily on clinical evaluations performed on samples of patients with Alzheimer's or Huntington's disease. The test manual suggests that validity for many of the tests had been previously established as they were modified from preexisting measures with proven validity.

Subsequent studies have demonstrated that D-KEFS tests are sensitive to the assessment of executive function deficits in numerous clinical populations including patients with focal frontal lobe lesions (Baldo et al. 2004); multiple sclerosis (Beatty et al. 1995); normal aging (Wecker et al. 2000); autism spectrum disorders (Kleinhans et al. 2003); schizophrenia (Beatty et al. 1994); childhood stroke (Nichols et al. 2003); attention deficit disorder (Donnelly et al. 2001); and fetal alcohol syndrome (Schonfeld et al. 2001).

Clinical Uses

D-KEFS tests allow examiners to systematically generate and evaluate relevant clinical hypotheses related to the executive functioning of a given examinee by comparing and contrasting performance on multiple testing conditions and using contrast measure scores and error analyses. A review of the administration, scoring and interpretation, test construction, standardization, and technical adequacy indicates that the D-KEFS has been useful as both a clinical instrument as well as a research tool for increasing knowledge of the frontal lobe functions (Homack et al. 2005).

The D-KEFS is notable for its utility in assessing and diagnosing strengths and vulnerabilities for both youth and adults in the domains of planning, impulsivity/inhibition, abstract thinking, and problem-solving. It has been particularly useful among clinical populations including those with traumatic brain injury, attention deficit disorders, and autism spectrum disorder as well as dementia and Alzheimer's among older adults (Delis et al. 2004; Homack et al. 2005). In addition to assisting with diagnostic clarity, the tool is useful in supporting clinicians in planning rehabilitation programs and interventions tailored to an individual's profile of executive function strengths and weaknesses.

References and Reading

- Anderson, P., Anderson, V., & Lajoie, G. (1996). The tower of London test: Validation and standardization for pediatric populations. *The Clinical Neuropsychologist*, 10, 54–65.
- Baldo, J. V., Wilkins, D. P., Shimamura, A. P., Kramer, J., Kaplan, E., & Delis, D. C. (2004). Is it bigger than a breadbox? Performance of frontal patients on a new test of concept formation. *Archives of Clinical Neuropsychology*, 19, 407.
- Baron, I. S. (2004). Delis-Kaplan executive function system. *Child Neuropsychology*, 10(2), 147–152. <https://doi.org/10.1080/09297040490911140>.
- Beatty, W. W., Jovic, Z., Monson, N., & Katzung, V. M. (1994). Problem solving by schizophrenic and schizoaffective patients on the Wisconsin and California card sorting tests. *Neuropsychology*, 8, 49–54.

- Beatty, W. W., Hames, K. A., Blanco, C. R., & Paul, R. H. (1995). Verbal abstraction deficit in multiple sclerosis. *Neuropsychology*, 9, 198–205.
- Center on the Developing Child at Harvard University. (2011). Building the brain's "air traffic control" System: How early experiences shape the development of executive function: Working paper No. 11. Retrieved from www.developingchild.harvard.edu
- Comalli, P. E., Jr., Wapner, S., & Werner, H. (1962). Interference effects of Stroop color-word test in childhood, adulthood, and aging. *Journal of Genetic Psychology*, 100, 47–53.
- Delis, D. C., Kaplan, E., & Kramer, J. (1995). *The California card sorting test*. San Antonio: The Psychological Corporation.
- Delis, D. C., Kaplan, E., & Kramer, J. H. (2001a). *The Delis Kaplan executive function system: Examiner's manual*. San Antonio: The Psychological Corporation/A Harcourt Assessment Company.
- Delis, D. C., Kaplan, E., & Kramer, J. H. (2001b). *The Delis Kaplan executive function system: Technical manual*. San Antonio: The Psychological Corporation/A Harcourt Assessment Company.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Holdnack, J. (2004). Reliability and validity of the Delis-Kaplan executive function system: An update. *Journal of the International Neuropsychological Society*, 10, 301–303. <https://doi.org/10.1017/S1355617704102191>.
- Donnelly, J. F., Carte, E., Kramer, J. H., Zupan, B., & Hinshaw, S. (2001). Executive functioning in girls with subtypes of ADHD. *Journal of the International Neuropsychological Society*, 7, 201.
- Golden, C. J. (1978). *Stroop color and word test: Manual for clinical and experimental uses*. Chicago: Stoetling.
- Homack, S., Lee, D., & Riccio, C. A. (2005). Test review: Delis-Kaplan executive function system. *Journal of Clinical and Experimental Neuropsychology*, 27(5), 599–609. <https://doi.org/10.1080/13803390490918444>.
- Kleinmans, N. M., Akshoomoff, N. A., & Courchesne, E. (2003). Executive functioning in autism and Asperger's syndrome: Results from the D-KEFS. *Journal of the International Neuropsychological Society*, 9, 273.
- Nichols, S., Waller, S., Trauner, D., & Delis, D. C. (2003). Performance of children with early focal brain damage on a new test of concept formation. *Journal of the International Neuropsychological Society*, 9, 139.
- Russo, N., Flanagan, T., Iarocci, G., Berringer, D., Zelazo, P. D., & Burack, J. A. (2007). Deconstructing executive deficits among person with autism: Implications for cognitive neuroscience. *Brain and Cognition*, 65, 77–86. <https://doi.org/10.1016/j.bandc.2006.04.007>.
- Schmidt, M. (2003). Hit or miss? Insight into executive functions. *Journal of the International Neuropsychological Society*, 9, 962–964.
- Schonfeld, A. M., Mattson, S. N., Lang, A. R., Delis, D. C., & Riley, E. P. (2001). Verbal and nonverbal fluency in children with heavy prenatal alcohol exposure. *Journal of Studies on Alcohol*, 62, 239–246.
- Shunk, A. W., Davis, A. S., & Dean, R. S. (2006). TEST REVIEW: Dean C. Delis, Edith Kaplan & Joel H. Kramer, Delis Kaplan Executive Function System (D-KEFS), The Psychological Corporation, San Antonio, TX, 2001. \$415.00 (complete kit). *Applied Neuropsychology*, 13(4), 275–227. https://doi.org/10.1207/s15324826an1304_9.
- Stroop, J. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, 18, 643–662.
- Wecker, N. S., Kramer, J. H., Wisniewski, A., Delis, D. C., & Kaplan, E. (2000). Age effects on executive ability. *Neuropsychology*, 14, 409–414.
- Welsh, M. C. (1991). Rule-guided behavior and self-monitoring on the tower of Hanoi disk-transfer task. *Cognitive Development*, 6, 59–76.
- Werner, H., & Kaplan, E. (1952). *The acquisition of word meanings: A developmental study* (Monographs of the Society for Research in child development, 15 (serial no. 51)). Evanston: Child Development Publications.

Intuitive Thought

► Symbolic Thought

Intuniv

► Guanfacine

Inventory of Interpersonal Situations (IIS)

► Glasgow Sensory Questionnaire (GSQ)

Inversion Effect

► Upright/Inverted Figures

Iodothyronine Deiodinase (D2T3)

► Maternal Hypothyroidism and Autism

IPSyn

- [Index of Productive Syntax \(IPSyn\)](#)
-

IQ

- [Intelligence Quotient](#)
-

IQ test

- [Psychological Assessment](#)
-

IRR: Inter-rater Reliability

- [PEAK Relational Training System](#)
-

Irreversible Dyskinesia

- [Tardive Dyskinesia](#)
-

Irritability in Autism

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Irritability in children with autism spectrum disorder (ASD) has been a long-standing target of clinical research including pharmacological and behavioral interventions. Historically, the term irritability was used in ASD literature as an umbrella category for severe disruptive behaviors including temper tantrums, aggression, and non-compliance. The rates of co-occurrence of ASD

with disruptive behavior disorders ranges from 16% to 37% for oppositional defiant and 2–9.6% for conduct disorder. However, up to 65% of children with ASD can have physically aggressive behavior towards their peers or caregivers. (Kanne and Mazurek 2011). Using a more recent conceptualization of irritability, as a cluster of mood symptoms, 45% of children with ASD were reported to have high levels of angry mood and temper tantrums (Mayes et al. 2017). Irritability and associated behavioral problems are distressing in their own right for children and families, and they also contribute to the gap between adaptive functioning and mental age beyond the impairment conferred by ASD (Williams et al. 2006).

Regarding causal mechanisms, irritability may be related to the core symptoms of ASD as well as to the psychosocial and biological mechanisms common across psychiatric disorders. In children with ASD, anger outbursts might be uniquely associated with the inability to communicate effectively, the interruption of repetitive behaviors, or visual, auditory, or tactile sensory sensitivities (Kanne and Mazurek 2011; Reese et al. 2005). Age and cognitive functioning can impact the type and frequency of disruptive behavior problems. For example, temper tantrums decrease with age, and self-injurious behaviors are more common in children with intellectual disability. However, the rates of aggressive behavior and noncompliance were similar in children with ASD with or without cognitive impairment (Mayes et al. 2012).

There can be differences in the expression of anger and triggers of disruptive behavior in children with ASD compared to children only with conduct disorder. For example, anger outbursts in ASD have been described as “immature” with labels such as “meltdowns” being used to reflect the uncontrollable nature of these behaviors. Also, physical aggression in ASD usually lacks hostile intent but is often impulsive in nature and can result in injuring people or destroying property (McDougale et al. 2008). However, irritability and aggression in ASD are also likely to share some common mechanisms with other forms of developmental psychopathology. For example,

the role of positive and negative reinforcement of aggressive behavior in the context of aversive parent-child interaction has been well studied in developmental psychopathology and provided a foundation for parenting interventions for aggression in children with and without ASD.

It is also important to recognize that temper tantrums and rough and tumble play can be developmentally appropriate and various facets of the anger experience and expression may follow different developmental trajectories. Thus, temper tantrums that include crying, stomping, pushing, hitting, and kicking are common in 1- to 4-year-old children and range in frequency from 5 to 9 times per week with the average duration of 5–10 min (Potegal et al. 2003). The intensity and number of tantrums tend to decrease with age although typically developing children continue to outwardly display anger and frustration, behaviors that parents often label as tantrums. This decrease in the frequency of temper tantrums as children age is paralleled by the development of emotion regulation skills and the acquisition of socially appropriate ways to express anger (Blanchard-Fields and Coats 2008). In children with ASD, if temper tantrums and disruptive behavior are present in childhood, they are likely to persist and may escalate in up to one third of adolescents (Shattuck et al. 2007; Simonoff et al. 2013). Aggression and irritability continue to cause significant distress to many adults with ASD and their families and present considerable challenges for clinical care (Siegel et al. 2014).

Following the seminal studies of risperidone for serious behavioral problems in ASD, the Irritability Subscale of the Aberrant Behavior Checklist (Aman and Singh 2017; Aman et al. 1985) has emerged as a gold standard in clinical trials and has been used as a measure of treatment effects in over 120 studies. The 15-item Irritability Subscale includes questions about aggression, self-injury, tantrums, agitation, and unstable mood. The total Irritability Scale score ranges from 0 to 45, with higher scores indicating greater severity. Other measures that have been used to characterize irritability and disruptive behavior in children with

ASD include the disruptive behavior subscales of the Child and Adolescent Symptoms Inventory (Gadow et al. 2008) and more recently, the Affective Reactive Index (Stringaris et al. 2012). Although most studies of irritability in ASD relied on caregiver-rated measures, there is emerging evidence that verbally able children with ASD can provide reliable self-reports of anger and irritability (Patel et al. 2017).

Interventions commonly used for children with ASD include psychotropic medications, psychosocial treatments, and special education programming (Volkmar et al. 2014). The goals of treatment include decreasing the core symptoms of ASD, enhancing specific communication and socialization skills, and reducing problematic behaviors. Atypical antipsychotics, risperidone and aripiprazole, have been reported to reduce irritability in children with ASD (Fung et al. 2016). However, medication side effects, most notably weight gain, metabolic abnormalities (McDougale et al. 2008), and high relapse rate when medication is discontinued (RUPP Autism Network 2005) underscore the value of non-pharmacological treatments for irritability in ASD.

Regarding behavioral interventions for irritability in ASD, applied behavioral analysis (ABA) has been a mainstay for reducing challenging behaviors and developing adaptive behavior skills in younger children with ASD and in children with ASD complicated by cognitive disabilities (Vismara and Rogers 2010). ABA identifies common sources of environmental reinforcement or functions of challenging behaviors in individuals with ASD such as attention from others, escape from task demands, access to preferred items or activities, and automatic or “self-stimulatory” reinforcement (Hanley et al. 2003). Knowledge of the function of challenging behavior is used to design behavior intervention plans that are based on modifying the reason why the behavior occurs in the child’s everyday life. In many forms of behavioral treatments for ASD parents are taught behavioral strategies of reducing disruptive behavior by modifying its antecedents and

consequences (Smith et al. 2000) and increasing compliance (Ducharme et al. 2007). A recent meta-analysis reported eight randomized studies of PMT for children with autism with an average effect size of 0.59 (Postorino et al. 2017). One approach to PMT was developed to address common and unique manifestations of irritability in ASD by enhancing standard PMT techniques with functional analysis, visual schedules for routine events, and teaching communication and daily living skills (Johnson et al. 2007). A randomized trial of 124 children, ages 4–13, with ASD tested this program in a comparison of risperidone alone to risperidone plus PMT and revealed greater reduction of disruptive behavior and lower drug dose in the combined condition (Aman et al. 2009). A recent study tested a preschool version of this program in 180 children with ASD, ages 3–7, relative to a parent education control condition and demonstrated significant reductions in parent-rated irritability and noncompliance in the active treatment condition (Bearss et al. 2015).

References and Reading

- Aman, M. G., & Singh, N. N. (2017). *Aberrant behavior checklist – Second edition (ABC-2)*. Wood Dale: Stoelting.
- Aman, M. G., Singh, N. N., Stewart, A. W., & Field, C. J. (1985). The aberrant behavior checklist: A behavior rating scale for the assessment of treatment effects. *American Journal of Mental Deficiency*, 89(5), 485–491.
- Aman, M. G., McDougle, C. J., Scahill, L., Handen, B., Arnold, L. E., Johnson, C., . . . Wagner, A. (2009). Medication and parent training in children with pervasive developmental disorders and serious behavior problems: Results from a randomized clinical trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(12), 1143–1154.
- Bearss, K., Johnson, C., Smith, T., Lecavalier, L., Swiezy, N., Aman, M., . . . Scahill, L. (2015). Effect of parent training vs parent education on behavioral problems in children with autism spectrum disorder: A randomized clinical trial. *JAMA*, 313(15), 1524–1533. <https://doi.org/10.1001/jama.2015.31502275445>.
- Blanchard-Fields, F., & Coats, A. H. (2008). The experience of anger and sadness in everyday problems impacts age differences in emotion regulation. *Developmental Psychology*, 44(6), 1547–1556.
- Ducharme, J. M., Sanjuan, E., & Drain, T. (2007). Errorless compliance training: Success-focused behavioral treatment of children with asperger syndrome. *Behavior Modification*, 31(3), 329–344.
- Fung, L. K., Mahajan, R., Nozzolillo, A., Bernal, P., Krasner, A., Jo, B., . . . Hardan, A. Y. (2016). Pharmacologic treatment of severe irritability and problem behaviors in autism: A systematic review and meta-analysis. *Pediatrics*, 137, S124–S135. <https://doi.org/10.1542/peds.2015-2851K>.
- Gadow, K. D., DeVincent, C. J., & Drabick, D. A. G. (2008). Oppositional defiant disorder as a clinical phenotype in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 38(7), 1302–1310.
- Hanley, G. P., Iwata, B. A., & McCord, B. E. (2003). Functional analysis of problem behavior: A review. *Journal of Applied Behavior Analysis*, 36(2), 147–185.
- Johnson, C. R., Handen, B. L., Butter, E., Wagner, A., Mulick, J., Sukhodolsky, D. G., . . . Vitello, B. (2007). Development of a parent management training program for children with pervasive developmental disorders. *Behavioral Interventions*, 22, 201–221.
- Kanne, S. M., & Mazurek, M. O. (2011). Aggression in children and adolescents with ASD: Prevalence and risk factors. *Journal of Autism and Developmental Disorders*, 41(7), 926–937.
- Mayes, S. D., Calhoun, S. L., Aggarwal, R., Baker, C., Mathapati, S., Anderson, R., & Petersen, C. (2012). Explosive, oppositional, and aggressive behavior in children with autism compared to other clinical disorders and typical children. *Research in Autism Spectrum Disorders*, 6(1), 1–10.
- Mayes, S. D., Kokotovich, C., Mathiowetz, C., Baweja, R., Calhoun, S. L., & Waxmonsky, J. (2017). Disruptive mood dysregulation disorder symptoms by age in autism, ADHD, and general population samples. *Journal of Mental Health Research in Intellectual Disabilities*, 10(4), 345–359. <https://doi.org/10.1080/19315864.2017.1338804>.
- McDougle, C. J., Stigler, K. A., Erickson, C. A., & Posey, D. J. (2008). Atypical antipsychotics in children and adolescents with autistic and other pervasive developmental disorders. *The Journal of Clinical Psychiatry*, 69(Suppl 4), 15–20.
- Patel, S., Day, T. N., Jones, N., & Mazefsky, C. A. (2017). Association between anger rumination and autism symptom severity, depression symptoms, aggression, and general dysregulation in adolescents with autism spectrum disorder. *Autism*, 21(2), 181–189. <https://doi.org/10.1177/1362361316633566>.
- Postorino, V., Sharp, W. G., McCracken, C. E., Bearss, K., Burrell, T. L., Evans, A. N., & Scahill, L. (2017). A systematic review and meta-analysis of parent training for disruptive behavior in children with autism

- Spectrum disorder. [Review]. *Clinical Child and Family Psychology Review*, 20(4), 391–402. <https://doi.org/10.1007/s10567-017-0237-2>.
- Potegal, M., Kosorok, M. R., & Davidson, R. J. (2003). Temper tantrums in young children: 2. Tantrum duration and temporal organization. *Journal of Developmental and Behavioral Pediatrics*, 24(3), 148–154.
- Reese, R. M., Richman, D. M., Belmont, J. M., & Morse, P. (2005). Functional characteristics of disruptive behavior in developmentally disabled children with and without autism. *Journal of Autism and Developmental Disorders*, 35(4), 419–428.
- RUPP Autism Network. (2005). Risperidone treatment of autistic disorder: Longer-term benefits and blinded discontinuation after 6 months. *The American Journal of Psychiatry*, 162(7), 1361–1369.
- Shattuck, P. T., Seltzer, M. M., Greenberg, J. S., Orsmond, G. I., Bolt, D., Kring, S., ... Lord, C. (2007). Change in autism symptoms and maladaptive behaviors in adolescents and adults with an autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37(9), 1735–1747.
- Siegel, M., Milligan, B., Chemelski, B., Payne, D., Ellsworth, B., Harmon, J., ... Smith, K. A. (2014). Specialized inpatient psychiatry for serious behavioral disturbance in autism and intellectual disability. *Journal of Autism and Developmental Disorders*, 44(12), 3026–3032. doi: <https://doi.org/10.1007/s10803-014-2157-z>.
- Simonoff, E., Jones, C. R. G., Baird, G., Pickles, A., Happé, F., & Charman, T. (2013). The persistence and stability of psychiatric problems in adolescents with autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 54(2), 186–194.
- Smith, T., Groen, A. D., & Wynn, J. W. (2000). Randomized trial of intensive early intervention for children with pervasive developmental disorder. *American Journal of Mental Retardation*, 105(4), 269–285.
- Stringaris, A., Goodman, R., Ferdinando, S., Razdan, V., Muhrer, E., Leibenluft, E., & Brotman, M. A. (2012). The affective reactivity index: A concise irritability scale for clinical and research settings. *Journal of Child Psychology and Psychiatry*, 53, 1109. <https://doi.org/10.1111/j.1469-7610.2012.02561.x>.
- Vismara, L. A., & Rogers, S. J. (2010). Behavioral treatments in autism spectrum disorder: What do we know? *Annual Review of Clinical Psychology*, 6, 447–468.
- Volkmar, F., Siegel, M., Woodbury-Smith, M., King, B., McCracken, J., & State, M. (2014). Practice parameter for the assessment and treatment of children and adolescents with autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 53(2), 237–257. <https://doi.org/10.1016/j.jaac.2013.10.013>.
- Williams, S. K., Scahill, L., Vitiello, B., Aman, M. G., Arnold, L. E., McDougle, C. J., ... Sparrow, S. (2006). Risperidone and adaptive behavior in children with autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45(4), 439.

Israel and Autism

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Historical Background

For several decades, from the early days of Israel's independence in 1948 up until the late 1980s, the care of children as well as adults with autism spectrum disorder (ASD) was the sole responsibility of the Israeli Ministry of Health. During this period, there were neither care centers nor residencies specifically designated for individuals with ASD. Instead, they were mostly referred to general psychiatric hospitals, which lacked specialized treatment modalities to meet these individuals' needs, particularly the distinct needs of children, adolescents, and young adults with ASD.

The early steps towards establishing specialized responses to the needs of individuals with ASD in Israel originated in the late 1960s. At that time, in the absence of a comprehensive national policy in the field, local medical institutions' initiatives led to the establishment of a few preschool day-care units to address the needs of children with ASD. In 1974, parents of individuals with ASD established the Israel Society for Autistic Children (ALUT). In 1977, this group of parents was instrumental in urging the municipality of Tel Aviv to found the first school for children with ASD, called Yachdav (Togetherness).

The late 1980s and early 1990s were marked by important developments in professional awareness and policy principles for services targeting Israeli individuals with ASD and their families. It

was then that the Israel Ministry of Health recognized the need for care centers to serve the adult population with ASD in Israel. As a result, in the early 1990s, a modest multiple-agency initiative was taken to create community-based ASD care centers nationwide that would address the unique needs of individuals with ASD throughout the lifespan, from infancy to adulthood. This initiative comprised a comprehensive and holistic approach for delivery of medical, educational, and rehabilitative social services to individuals with ASD. This approach was facilitated by the collaboration of the Ministries of Education, Health, and Welfare and by local authorities and third sector nongovernmental organizations (NGOs).

Likewise, in 1990, professionals and public representatives established the Association for Children at Risk (ACR). This NGO's aim was, primarily, to advance an integrative model for early diagnosis and intensive community-based treatment and education in preschools and kindergartens serving children with ASD. This model was implemented in Tel Aviv and then in a step-wise fashion in the Tel Aviv metropolitan area. The cumulative experience gained by these integrative kindergartens served as the foundation for the later governmentally approved 14-hour-per-week therapeutic basket of services allocated to these children.

Following such clinical, educational, and public health developments in the field of community-based service delivery to individuals with ASD, in 1993 an official ministerial committee stated that "Autism is not a psychotic disorder, but rather a developmental disability that needs to be treated at multiple levels by multiple government offices." The committee also clearly defined these interministerial responsibilities and interrelationships. In turn, each ministry then established a designated unit to develop its specific responses and direct its work in this rapidly developing field. Furthermore, in 1998, the Ministry of Health issued professional criteria to define preschool children's eligibility for a community-based 14-hour therapeutic service basket for children with ASD. The Ministry of Health assigned responsibility for this basket's delivery,

supervision, and monitoring to its Department of Mental Health. Other general medical and mental health services are rendered by the non-governmental national healthcare system that operates under the National Insurance Law (see further below) through medical insurance organizations (the Israeli equivalent of HMOs in the USA).

In 2001, a new parents' organization, the Israeli Asperger Association (Effe), was established in order to advocate for the unique needs of high-functioning individuals with ASD.

In conclusion, since the 1980s, the services available in Israel for individuals with ASD across the lifespan have started to grow and improve. Yet, specifically regarding preschool children with ASD, who were at the focus of professional and public concern, these changes never matched the growing demand for services. In 1995, health policy planners predetermined a fixed budgetary allocation for the total annual number of therapeutic baskets to be delivered in Israel, allotting an annual quota increase of up to 40 preschoolers. However, the following 15 years witnessed a general upsurge in the worldwide prevalence of the ASD diagnosis, as exemplified in the USA by an increase from ≈ 3 in 10,000 in the 1980s (Burd et al. 1987) to ≈ 1 in 68 in recent epidemiological studies (Christensen et al. 2016). In view of the growing prevalence of ASD in Israel, public professional representatives (ACR) approached governmental and judicial authorities (2006), who responded by abolishing the aforementioned restrictive quota and thus ensuring universal eligibility for the therapeutic basket to all preschool children with ASD in Israel. Consequently, services for these children have widely expanded and improved nationwide.

To this day, consistent efforts are being made at the national level in Israel to raise awareness and improve early diagnosis and treatment that would promote intensive early intervention, thereby fostering improved prognoses for individuals with ASD. As seen in the section below on current services, treatments, and centers, the overall improvement noted in ASD services throughout the country has resulted from successful collaborations between various government ministries,

local authorities, parents' organizations, and professional NGOs. Yet, there is much more to be done in the areas of eligibility for services and service development for adults and elderly people with ASD.

Legal Issues

Legislation on the rights of individuals with ASD and their families in Israel culminated in the following fundamental laws, among others:

1. 16th Amendment (1984) to the Compulsory Education Law (1949) – granting the fundamental right to education also to children aged 3–5 years.
2. Special Education Law (1988) – defining among others: (a) the pedagogical and paraprofessional basket for special education and (b) the school placement committee at the local authority level (e.g., members, procedures).
3. National Health Insurance Law (1994) – mandating national healthcare for all citizens, defining the responsibility of medical insurance organizations and of the Ministry of Health vis-à-vis the citizen.
4. National Insurance Law [integrated version] (1995) – establishing the role of the National Insurance Institute (NII), Israel's social security agency that defines, among others, eligibility for partial or full disability compensation.
5. Law on Equal Rights of Persons with Disabilities (1998).
6. Law on Day-Care Rehabilitation Centers (2000) – mandating the establishment of specialized integrative service settings for infants and toddlers with special needs.
7. 7th Amendment to the Special Education Law (2002) – known as the Integration Law – mandating the right to community integration for children with special needs.
8. 43rd Amendment to the National Health Insurance Law (2008) – pertaining to the equal rights of children and adolescents with ASD to receive 3 weekly hours of specific therapies.

In recent years, parent organizations have been advocating for the introduction of a designated Autism Law that will incorporate all the rights of individuals with ASD.

Mandates for Service and Overview of Current Treatments and Centers

Eligibility for health, education, and welfare support and special services for individuals with ASD in Israel is determined based on a multidisciplinary diagnosis. The diagnostic procedure requires: (a) a diagnosis given by a specialized medical doctor (child development specialist, child neurologist, or child psychiatrist) based on *DSM-5* criteria (APA 2013) and (b) a psychological assessment. The psychological assessment procedure includes standardized ASD measures (e.g., ADOS-2, ADI-R, CARS-2), as well as measures of cognitive abilities (e.g., Mullen and Wechsler scales, according to age) and of adaptive functioning levels (e.g., Vineland adaptive behavior scales).

Multiple government ministries share responsibility for community-based service delivery to individuals with ASD, including diagnostic, therapeutic, and psychiatric services delivered via the Ministry of Health; special education services delivered via the Ministry of Education; rehabilitation services delivered via the Ministry of Welfare; and additional benefits delivered primarily via the NII. To be noted, up to this day families continue to face the burden of paying out of pocket for diagnostic, therapeutic, educational, leisure, occupational, or residential services. In particular, they are required to contribute a considerable amount of money to supplement governmental budgets, in order to support integrated community-based services. The services are offered equally to citizens of all religions and ethnic sectors.

Below is a short overview of the services and benefits that pertain to the relevant government ministries.

Ministry of Health

Health services for children with ASD may be delivered directly by Ministry of Health-approved service providers or may be offered by one of the four official national medical insurance service providers (as part of their developmental pediatric services). Children are eligible for diagnostic, therapeutic, and psychiatric services as follows:

- **Diagnostic services.** Early child development centers (operated directly by the Ministry or by medical insurance organizations) are responsible for the diagnostic procedure that establishes children's eligibility for treatment, special education services, and compensation via NII (see below). As reported (Davidovitch et al. 2013), in 2005, the majority of children with ASD in Israel (85%) were diagnosed before the age of 3 years. To this day, there is a great public shortage of available medical and psychological personnel to supply the mandatory ASD diagnostic assessment procedure to children waiting for this service. As a result, the private market is called upon to support the growing need for diagnostic services. It is this fact, in and of itself, that often causes an increase in individual as well as national health expenditures, although the details of this situation on the ground are beyond the scope of the current discussion.
- **Therapeutic services.** In line with the Israeli national healthcare law of 1994, every preschooler diagnosed with ASD is eligible to obtain the multidisciplinary community-based therapeutic basket of 14 hours per week from the Ministry of Health, through approved service providers (as anchored in the Ministry's internal guidelines). This basket allows for the integration of various kinds of treatments for each child, such as speech and language therapy, occupational therapy, physiotherapy, behavior analysis, psychotherapy (including parental guidance and family therapy), and so on. Approved NGOs deliver this integrative service model to preschoolers with ASD in two different community-based special education settings, the first operated by the Ministry

of Welfare and the second by the Ministry of Education, both in conjunction with the Ministry of Health: (1) rehabilitative day-care centers for infants and toddlers with ASD aged 1–3 years and (2) preschools and kindergartens for young children with ASD aged 3–7 years.

Families who elect to place their preschoolers in mainstream educational settings are entitled to receive the aforementioned basket from community-based NGOs' multidisciplinary clinics. Those preschool children who do not yet have access to these services (due to current shortages in available professional resources) are eligible for 3 weekly hours of ASD-related health care via the medical insurance organizations, as described below.

School-aged children with ASD are entitled to 3 weekly hours of basic therapies through medical insurance organizations. These insurance organizations contract public providers either to render these services in public/private medical clinics or to integrate these services within the education system via NGOs (currently provided by the ACR).

The treatments available to individuals with ASD and their families have evolved in Israel over the years, as ASD prevalence has increased (Davidovitch et al. 2013). The past decade in Israel has seen the development of integrative approaches to treatment. These approaches bring together principles of diverse theories and practices to address the wide range of difficulties encountered by persons with ASD. Treatments range from the traditionally widespread psychoanalytically oriented therapy to neuropsychological developmentally based interventions and empirically supported approaches such as applied behavior analysis (ABA); Treatment and Education of Autistic and Communication related handicapped Children (TEACCH); Developmental, Individual Differences, Relationship-Based model (DIR[®]); the Early Start Denver Model (ESDM[®]); cognitive behavioral therapy; and social skills interventions.

- **Psychiatric services.** Mental health clinics provide child psychiatry services, mostly to

children and adolescents with ASD who have comorbidities (e.g., attention deficit-hyperactivity disorder, anxiety disorders, obsessive compulsive disorder, depression) that require pediatric psychiatric consultation. Children older than 6 years with an ASD diagnosis are usually referred to their medical insurance organization's child psychiatry clinic for consultation. Alternatively, some of these patients are referred to one of three tertiary centers specializing in ASD that are available in central Israel: ACR's Bait Echad, ALUT's Autism Center at Assaf Harofe Medical Center, or the Keshet Center for individuals with ASD at Sheba Medical Center. Since 2015, adult individuals with ASD are eligible for psychiatric services under the National Health Insurance Law, which includes psychiatric and mental health services.

As for the availability of hospitalization for individuals younger than 18 with ASD who experience severe behavioral disruptions, only two specialized hospital units are designated for individuals with ASD in the country. Therefore, most individuals with ASD requiring acute inpatient treatment still receive treatment in general child and adolescent psychiatric settings.

Ministry of Education

The Compulsory Education Law (1949) required all children in Israel from age 3 to 17 years to attend school, free of charge, in an educational setting accredited by the Ministry of Education. However, only in 1988 did Israel endorse the law that pertains to special education in a way that assigned therapeutic services to these special students (aged 3–21), based on their specific developmental needs (3.4 weekly hours per student). The amendment from 2002 further stipulated that children who are eligible to receive special education services should have the option of attending school with typically developing peers and should be entitled to designated funding to supply personnel to help children integrate into mainstream education classes (e.g., in-class aides, additional remedial teachers).

Thus, the Israeli Ministry of Education mandates the formation of a special education school

placement committee in each local authority. Based on integrative multidimensional information about each child diagnosed with ASD, the committee prescribes an optimal placement for that child into an integrative education setting or a self-contained special education setting (e.g., an ASD kindergarten). Accordingly, the committee members represent: the local authority (including the committee chairperson and school psychologist), the Ministry of Education (superintendent), developmental pediatricians, social workers, and parents' organizations.

Children between the ages of 3 and 6 years are entitled to placement in a special education kindergarten. Once placed in such a setting, each child's teachers, developmental specialists, and mental health professionals form an integrated problem-oriented pedagogical-therapeutic individualized plan (taking into consideration the needs of the individual family as well). This plan and its implementation are periodically evaluated by the multidisciplinary team and are monitored by the respective Ministries of Education and Health. The collaborative work of these two ministries is anchored in interministerial guidelines from 2015.

Ministry of Welfare

The Ministry of Welfare is responsible for providing rehabilitative treatments as well as leisure activities for individuals with disabilities over the lifespan. To note, all services for individuals with ASD uphold a family perspective and involve family members in their therapeutic programs as well as outreach programs. In addition, the Ministry of Welfare offers partial funding for family support centers nationwide, primarily operated by NGOs (ALUT), which serve thousands of families by providing support, psychoeducational groups, hotlines (in routine and emergency situations), as well as consumer empowerment activities (ALUT).

Specific services are provided by age group. For example, for infants and toddlers (ages 1–3), the Ministry of Welfare, in conjunction with the Ministry of Health, provides early education, a therapeutic basket of medical services, and social rehabilitation, delivered through approved NGOs

and local authorities, who serve as integrative service providers. The Israeli Ministries of Welfare and Health mandate the formation of regional evaluation committees for the placement of infants and toddlers diagnosed with ASD (ages 1–3) to day-care centers throughout the country.

Once a young child is placed in a day-care center, teachers, nutritional specialists, developmental specialists, and mental health professionals form an integrated problem-oriented pedagogical-therapeutic individualized plan (taking into consideration the needs of the individual family as well). This plan and its implementation are periodically evaluated by the multidisciplinary team and are monitored jointly by teams from the Ministries of Welfare and Health.

For school-age children with ASD, various supportive services are anchored in Ministry of Welfare regulations, such as after-school help at home, after-school therapeutic-rehabilitative activities to promote social and communication skills and to advance social integration, and day camps during school vacations.

For adults with ASD, the Ministry of Welfare offers a wide range of community-based opportunities such as training, supervision, and follow-up regarding employment as well as offering occupational facilities; leisure time activities; and protected or independent-living residences. Overall, services for adults with ASD in Israel are provided through the Ministry of Welfare as well as the NII and various NGOs that were created (often by parents) to improve participation and inclusion. Attempts have been made to provide appropriate solutions for different stages of adult development. In typically developing individuals, following secondary education in Israel, adolescents' transition to adulthood usually involves 2–3 years of mandatory military service or civil service. However, most young adults diagnosed with ASD are exempt from military service. To enable individuals with ASD to volunteer for civil or military service, designated support programs have been developed by NGOs, working in collaboration with the Ministry of Welfare and the Ministry of Defense. These programs for civil service (Bat-Ami, Shlomit, Gvanim) and military

service (Hiburim) offer social training and facilitate individually tailored assignment of young adults with ASD to specific military jobs and units. Another program (Roim Rachok – Looking Ahead) capitalizes on the unique strengths of young people with ASD such as attention to detail and systemizing skills, in order to train them socially and professionally for highly needed specialized roles within the military system that rely on such skills. In all these programs, both the individuals with ASD who end up serving in the military as well as their commanders receive supervision and support from professionals. Preliminary results of such programs show participants' improved subjective well-being and quality of life 6 months into the service (Gal et al. 2015).

Additional services to foster independence in young adults with ASD are provided by NGOs and private sector companies (e.g., Avnei Derech, Beit Ekstein, EDNM, Shekel, Shlav, Tigbur), with the support of the Ministry of Welfare. Such programs focus on helping such individuals to move out of their parents' homes either into designated housing for individuals with ASD or into community housing. NGOs also supply vocational training with the support of the Ministry of Welfare and the NII. Specialized vocational training for individuals with ASD is offered by several organizations (e.g., AQA, Beit Issie Shapiro, By the People), capitalizing on the unique skills of individuals with ASD when training them for software quality assurance, computer programming, or information management. Several academic institutions offer pre-academic courses, focusing mostly on independent, social, and executive functioning for this population. One Israeli academic institution, Ariel University, offers its full academic program for high-functioning adults with ASD. During their studies, the students live in dorms with neurotypical students, who support their social inclusion.

Job markets have been open to persons with disabilities since 1998, when legislation mandated occupational and equal opportunities for these individuals. The 2010 amendment to this law mandated a quota of 5% employment of people with disabilities in large public companies, representing the percentage of such individuals

in the general Israeli population. In line with the cultural change that ensued from this law and in order to enhance integration in the labor market, several organizations (e.g., Beit Ekstein, Effie, Shekel) now offer training, opportunities for accessing and mediating jobs, and on-the-job support and guidance to adults with ASD. In addition, based on their functional abilities, adults with ASD may elect to work within specialized community-based employment centers operated by different organizations (e.g., ALUT, Beit Ekstein) throughout the country.

Leisure time and social activities are offered for adults with ASD through NGOs (e.g., Effie, Etgarim, Keshet, Reim, Shekel), some of which also meet age-related needs for intimacy (e.g., dating and sexual education).

Additional National Insurance Benefits

In addition to the aforementioned services, individuals with ASD are entitled to disability benefits under the Israeli National Insurance Law of 1995. Children diagnosed with ASD receive full (100%) NII disability compensation payments until they are 18 years of age. In addition, children from 6 years of age may also be eligible for further NII benefits to allow for appropriate supports if they continue to depend on adult assistance. Adults with ASD over the age of 18 are entitled to further personally tailored support from NII benefits. Other legislature benefiting individuals with ASD and their families relates to tax benefits, extended sick leave for parents to children with ASD, reduced public transportation fees, and more.

Overview of Research Directions

Compared to the vast development of clinical and social services in Israel since the late 1980s that emerged under the aegis of governmental authorities and delivered through medical insurance organizations in collaboration with NGOs, ASD research in Israel has somewhat lagged behind, if measured by output. However, the last decade has seen a steep rise in Israeli ASD research. According to the PubMed free database of

indexed citations and abstracts, research published by active Israeli researchers has increased markedly, from seven papers in 2006 to 40 papers in 2016. Israeli ASD research covers various academic disciplines including biology, neuroscience, clinical medicine, psychology, education, occupational therapy, and more. Researchers from all eight major universities and several colleges as well as university-affiliated hospitals and NGOs are engaged in ASD research, with the most active universities including the Hebrew University of Jerusalem, Bar-Ilan University, University of Haifa, Tel-Aviv University, Ben-Gurion University, and Ariel University. Empirical ASD research is supported by local funding from the Israel Science Foundation, the NII, and the Ministry of Science, as well as by various international foundations and NGOs. Israeli ASD researchers are highly involved in international research collaborations. Key basic research areas of interest to Israeli investigators include, among others, simplex and multiplex ASD genetics, neuroimaging studies, neuroendocrine studies examining the role of specific hormones in ASD (e.g., oxytocin, cortisol, vasopressin), factors associated with high risk for ASD, social cognition, emotion understanding and emotional regulation, sensory characteristics of ASD, the broader autism phenotype as reflected through parental and sibling studies, and parenting characteristics and their effects on children with ASD. Some central applied research foci include school-based social skills training, technology-based interventions for individuals with ASD, and intervention outcome studies in preschoolers and young children. In addition, the first registered clinical trial of cannabis in the ASD population is currently underway at the Shaare Zedek Medical Center in Jerusalem.

Overview of Training

Professionals who work with individuals with ASD – such as occupational therapists, psychologists, special education teachers, psychiatrists, speech and language therapists, and others – receive academic training within their respective

discipline. In addition, specialized training programs in ASD are operated by: (a) each of the relevant ministerial authorities (i.e., Ministries of Health, Education, and Welfare); (b) national professional associations; and (c) the institutions serving the ASD population, which offer teaching, on-the-job training, and supervision in the various disciplines.

Graduate level academic training in ASD studies is offered through the special education master's programs of two universities (Bar-Ilan, Haifa). More focused case-management training is offered by several colleges (David Yellin, Kibbutzim, Levinsky, Oranim). ABA training and certification is available through universities (Tel-Aviv University, Hebrew University) and colleges (Beit Berl, David Yellin, Kibbutzim, Oranim, Wingate). Professional training on standardized ASD diagnostic instruments is offered through the Hebrew University and the ACR (at Bait Echad Centers).

As of yet, the residency program for medical professionals working with individuals with ASD does not include a designated ASD-oriented curriculum. An exception to the rule is a pilot training program established jointly by Geha Mental Health Center and the ACR. In their outpatient clinic residency, all the Center's child psychiatry residents participate in a pilot community outreach program, delivering the mental health basket to young children attending ASD preschools and kindergartens served by the ACR. These residents receive a structured syllabus of teaching and training and obtain supervision and guidance from senior child psychiatrists specializing in the area of ASD.

Social Policy and Current Controversies

Taking into account the rapidly increasing prevalence of ASD and the accumulating evidence attesting to early intervention's optimal effects for ASD trajectories, it is of paramount importance that the Israeli government has assumed responsibility for lifespan public health planning and service delivery for individuals with ASD. However, this governmental commitment remains

geographically limited and significantly declines after the age of 7 years. That is, school-age children, as well as preschoolers living in peripheral areas who lack community-based services, only have access to a limited number of therapeutic hours per week within their medical insurance organization's basket, regardless of their impairment level. Furthermore, adults with ASD are deprived of such therapeutic support and, under the National Health Insurance Law, are only entitled to a general mental health basket resembling any other chronic psychiatric patient.

Against this background, several controversies have arisen in the Israeli public health discourse, focusing on the following questions:

1. To what extent should ASD be considered unique among other developmental disabilities? Should an ASD diagnosis continue to entitle individuals to the current, more privileged service basket, which is richer than that allotted to intellectual impairment and other various kinds of developmental disorders?
2. In view of the increasing prevalence of ASD in Israel, and its accompanying budgetary demands, should diagnostic services be provided by state-monitored specialized centers rather than by private practitioners?
3. Given the limited window of opportunity for early intervention in ASD, should such intervention be governed only by evidence-based practice, which is still rather limited in Israel, or should policy makers call for responsible integrative maintenance of common clinical practice while supporting continual proliferation of ASD research studies? This question pertains not only to the field of autism but also to the general field of psychotherapy for other psychiatric disorders, especially considering the gap between available intervention research data and the clinical scope of application. An evidence-based policy would require the government to undertake a clinically sensitive and scientifically responsible, concerted educational efforts, to educate, train, and monitor the national therapeutic professional community.
4. Considering that ASD is a heterogeneous condition, where diagnosed individuals

demonstrate a range of functioning levels, should the country's limited budget be spent primarily on support for those individuals with more severe disabilities, or rather on rehabilitation of those individuals who have greater potential for adaptive functioning?

5. In view of the diagnostic changes introduced in the *DSM-5*, specifically the exclusion of the pervasive developmental disorder not otherwise specified (PDD-NOS) and the introduction of the new social communication disorder (SCD) as separate from the ASD diagnosis, what is Israel's commitment to individuals bearing the new SCD diagnosis, who may consequently be deprived of their eligibility for the therapeutic ASD basket and thereby their access to community-based ASD services? In the absence of research concerning the new SCD diagnosis and its required treatment modalities, including their costs, should policy changes be implemented to ensure that necessary services and support are delivered to these diagnosed individuals?
6. How should decisions be made regarding the optimal level of inclusion for individuals with ASD in mainstream educational and occupational settings along the lifecycle? What roles should be played by state legislation, professional and community representatives, family members, and the individual herself/himself?

A few words of conclusion are in order. Israel has seen three decades of significant surges in legislature and social policy aiming to promote the rights of persons with ASD throughout the lifecycle, and their families. These rights pertain to available and accessible integrated and specialized services (education, health, and welfare) as well as to work opportunities. Governmental ministries, local authorities, parental organizations, professional NGOs, public and private service centers, and academic institutions have collaborated to bring about these changes in the areas of: service provision, professional education and training, as well as research support. Nonetheless, much remains desired in terms of national planning with regard to the latter two fields.

Considering that ASD is one of the most complex developmental disorders, involving multiple human functional dimensions that require the attention of numerous and diverse systems and fields of care, countries often endorse a range of social policies, spanning from holistic-integrative policies to loosely coordinated field-specific ones. Israel has undertaken a challenging path leaning more toward the holistic-integrative care delivery approach for persons diagnosed with ASD. Integrative complex systems are more vulnerable and difficult to both create and manage. Hence, it remains to be seen as to how the ASD-related care delivery system in Israel will effectively succeed in overcoming its necessarily inherent conceptual, professional, legal, and bureaucratic challenges. Optimal, flexible systemic solutions for these challenges are hard to come by, but once achieved they would serve the best interests of individuals and subgroups with ASD, within their diverse families and communities.

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Burd, L., Fisher, W., & Kerbeshian, J. (1987). A prevalence study of pervasive developmental disorders in North Dakota. *Journal of American Academy of Child Adolescent Psychiatry*, 26, 700–703. <https://doi.org/10.1097/00004583-198709000-00014>.
- Christensen, D. L., Baio, J., Braun, K. V. N., Bilder, D., Charles, J., Constantino, J. N., . . . Yeargin-Allsopp, M. (2016). Prevalence and characteristics of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2012. *Morbidity and Mortality Weekly Report Surveillance Summaries*, 65, 1–23. <https://doi.org/10.15585/mmwr.ss6503a1>.
- Davidovitch, M., Hemo, B., Manning-Courtney, P., & Fombonne, E. (2013). Prevalence and incidence of autism spectrum disorder in an Israeli population. *Journal of Autism and Developmental Disorders*, 43, 785–793. <https://doi.org/10.1007/s10803-012-1611-z>.
- Gal, E., Selanikyo, E., Erez, A. B.-H., & Katz, N. (2015). Integration in the vocational world: How does it affect quality of life and subjective well-being of young adults with ASD. *International Journal of Environmental Research and Public Health*, 12, 10820–10832. <https://doi.org/10.3390/ijerph120910820>.

Italy and Autism

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Historical Background

In Italy, interest in early-onset mental disorders dates back to the influential work of Sante De Sanctis (1862–1935). In 1905, he described a condition defined as “dementia praecox pre-puberis” (childhood-onset dementia), which was characterized by early emerging symptoms that we would now recognize as autistic features (Feinstein 2011). In 1898, he founded a scientific organization for the care of children with mental disabilities (*Associazione Romana per la cura medico-pedagogica dei fanciulli anormali*) together with Maria Montessori (1870–1952), a pioneer in child psychology and pedagogy. Maria Montessori’s work focused on the role of education in the promotion of mental health in children with and without mental disability. She developed an educational method (Montessori method), which would eventually spread all over in the world, and came to international prominence as an advocate for education for mentally disabled children.

However, Montessori’s “pedagogical science” approach to childhood mental health lost its relevance after World War II, as the psychiatric culture in Italy became progressively influenced by the psychodynamic perspectives coming from France and the USA (Migoni 1999). Early conceptualization of autism in Italy reflects the prevailing influence of the psychoanalytic orientation, with most professionals considering autism as a psychotic reaction caused by poor parenting. Psychoanalytic psychotherapy for both mother and child was the treatment of choice for children, and care for adults with mental disabilities was limited to long-term institutionalization in psychiatric hospitals. Despite relevant

exceptions (e.g., Zappella 1997; Micheli 1999), the conceptualization of autism as caused by “refrigerator mothers” persisted in Italy both among professionals and in the mainstream culture throughout the 1980s, 1990s, and early 2000s (e.g., Bertolini and Neri 1992; Reda 2003).

In 1978, a law was passed that aimed to end long-term institutionalization of “mentally ill” adults in psychiatric hospitals and established the development of a network of community-based mental health facilities designed to provide short-term targeted treatment. However, adults with autism did not benefit from this legislation as they are classified as “intellectually disabled” as opposed to “mentally ill” under the Italian legislation. Community-based services for adults with ASD, as of today, are still under development.

Starting from the 1990s, parent associations such as ANGSA (*Associazione Nazionale Genitori Soggetti Autistici*) and Autismo Italia were the driving force underlying dissemination of evidence-based knowledge and good practices in service organization through (a) scientific international conferences, (b) training on educational and behavioral strategies (mainly inspired by the TEACCH model), and (c) promoting the translation of scientific literature into Italian. This process was facilitated by the establishment of partnerships with professionals who supported the paradigm shift from psychoanalytical-oriented to evidence-based practice in autism (Caffo et al. 1999; Società Italiana di Neuropsichiatria dell’Infanzia e dell’Adolescenza 2005).

Today, after the publication of the guidelines on autism treatment by the National Institutes of Health (Istituto Superiore di Sanità 2011), the notion of autism as a neurodevelopmental disorder requiring educational intervention is accepted in most clinical, research, and academic settings. However, psychoanalytical approaches to therapy are still implemented in private practice, as indicated by a recent national research report (Censis 2012). Moreover, the implementation of evidence-based practice in school- and community-based settings is still largely affected by unequal availability of resources in different areas of the country.

Legal Issues and Mandate for Services

In Italy, promotion and protection of the rights of persons with Autism Spectrum Disorder (ASD) at the national level are regulated by a legislative framework focused on disability. Article 3 of the Italian Constitution guarantees equal social dignity to all citizens. This principle is reaffirmed in several national laws. The most relevant ordinary laws to persons with ASD are the following:

- L. 104/92, promoting the inclusion and social and functional habilitation of persons with disabilities through intervention, services, and benefits aimed at overcoming exclusion. These include (a) free and compulsory education to all children in the mainstream education system, without any exception or distinction based on the nature or severity of disability, and (b) the legal protection of their civil, political, and patrimonial rights.
- L. 328/00, promoting the implementation of harmonized, essential standards of social services aimed at achieving the inclusion and independence of persons with disabilities across the country. Such standards include the allocation of financial support to independent living as well as the implementation of daily or residential services for persons in need of intense support.
- L. 18/2009, ratifying the United Nations Convention on the Rights of Persons with Disabilities, which defines the principle of equality of persons with disabilities in any aspect of life, without any exception or distinction based on disability, including the need for more intense support.

Nevertheless, the L. 3/2011, which reformed the constitution, established the decentralization of social and health services provision. This law allots to the state government the task of defining the essential levels of social benefits, while rules and policies are regulated by the 20 Italian regions, and the city councils are in charge of the implementation of social services. Some, but not all, Italian regions have established guidelines recommending (a) standardized protocols for

intervention, (b) evidence-based treatments, and (c) “hubs and spokes” organization models of public health services.

Overview of Current Treatments and Centers

Notwithstanding the above legislative framework, policies and services for persons with ASD in Italy show relevant weaknesses. The third research report by the Censis Institute (2012) shows that the access to diagnosis of ASD has improved in the last decade, but delays are still common. The majority of the sample waited 1–3 years for the diagnosis, while 13.5% of the sample, mainly families of adolescents and adults, declared to have waited for more than 3 years. Social services generally fail to meet the support needs for persons with ASD. This lack of adequate support and services indeed has a negative impact on employment of at least one parent (65.9%), mainly on mothers’ employment (62.6% of the sample, compared to 25.5% of fathers) and on parents of those who require more intense support (68.9%, compared to 61.3% of parents of those requiring less intense support).

While almost all young children with ASD receive some treatment, up to 30% of adolescents and adults do not have access to any form of intervention. Approximately half of persons with ASD accessing treatment receive behavioral/educational intervention such as ABA-based programs, TEACCH, and related approaches. Intensity of behavioral/educational intervention is as low as 5 h per week, mostly paid by families. A number of other therapies are provided by the public health system including speech therapy (33% of the sample), “psychomotor therapy” (31%), and psychoanalytic psychotherapy (14%). Over 30% of young children with ASD received only these therapies, with no access to behavioral/educational intervention. A relevant proportion of children (32.2%) receive complementary and alternative medicine therapies, which are in part covered by the public health system despite the lack of evidence to support their efficacy.

Research Directions

In the 2000s, autism has become a growing field of study in Italy, with an increased number of scholars (many of them trained abroad) providing research contributions of international relevance. In particular, the work on the mirror neuron system by the Parma group established one of the most productive research directions in the field in the past decade (Cattaneo et al. 2007; Gallese 2006; Rizzolatti and Sinigaglia 2008). In 2008, the network of research centers “Italian Autism Network” was created with the support of the GlaxoSmithKline Foundation to establish a biologic bank at the University of Verona. In 2015 the Italian Association for Autism Research (Associazione Italiana Ricerca Autismo) was established with the goal to facilitate development and dissemination of scientific research in the field of ASD. Other relevant research directions include genetic and metabolic factors (Lintas et al. 2009; Manzi et al. 2008; Persico and Bourgeron 2006), developmental patterns (Maestro et al. 2005), neurobiology (Keller and Persico 2003), medical and psychiatric comorbidities (Canitano 2007; Mazzone et al. 2012), animal models (Scattoni et al. 2012), treatment effectiveness (Vicari et al. 2012), and psychopharmacology (Masi et al. 2009; Zuddas et al. 2011).

Social Policy and Training

Differences exist in the provision of health and social services that are determined by uneven availability of resources in the Italian territory. Access to school however is guaranteed throughout the country as the school system is regulated by a national legislation, and it is not affected by local availability of resources/budgets. Up to 93.4% of children and adolescents with ASD between age 3 and 19 access mainstream schools. After compulsory school years, only 7% of adults over 21 are enrolled in higher education, and 3% only in vocational training, while 50% are included in day care centers, and 22% stay at home and are not involved in any activity. Finally, 30% do not receive any treatment whatsoever, and 10% only have some form of employment (including sheltered or part-time jobs).

Segregating settings that do not implement targeted educational/habilitation programs represent the only available residential option for 86% of adults with ASD with high support needs. Community-based residential services (small residential communities, group homes) represent a small minority of the available options (4%) and are suitable to persons with mild or light disabilities only (DECLOC Report 2008).

In the past decade, training options for professionals working in autism have progressively increased. These include mainly university masters/postgraduate courses focused on various aspects of ASD, including genetics/biology and clinical management/intervention, and educational approaches.

See Also

- [Advocacy](#)
- [Community-Integrated Residential Services for Adults with Autism](#)
- [Culture and Autism](#)
- [Education](#)
- [Psychodynamic Theories](#)

References and Reading

- Bertolini, M., & Neri, F. (1992). In G. Vizziello Fava & D. N. Stern (Eds.), *Dalle cure materne all'interpretazione* (pp. 115–142). Milano: Raffaello Cortina Editore.
- Caffo, E., et al. (1999). International association of child and adolescent psychiatry and allied professions declaration of Venice on autism and pervasive developmental disorders. *European Child and Adolescent Psychiatry*, 8, 157–158.
- Canitano, R. (2007). Epilepsy in autism spectrum disorders. *European Child and Adolescent Psychiatry*, 16(1), 61–66.
- Cattaneo, L., Fabbri-Destro, M., Boria, S., Pieraccini, C., Monti, A., Cossu, G., et al. (2007). Impairment of actions chains in autism and its possible role in intention understanding. *Proceedings of the National Academy of Sciences of the United States of America*, 104(45), 17825–17830.
- Curatolo, P., Porfirio, M. C., Manzi, B., & Seri, S. (2004). Autism in tuberous sclerosis. *European Journal of Paediatric Neurology*, 8(6), 327–332.
- DECLOC – Deinstitutionalization and community living outcomes and costs- Report. (2008). <http://www.kent>.

- ac.uk/tizard/research/DECL_network/documents/DECLOC_Volume_2_Report_for_Web.pdf
- Feinstein, A. (2011). *History of autism: Conversations with the pioneers*. Malden: Wiley.
- Gallese, V. (2006). Intentional attunement: A neurophysiological perspective on social cognition and its disruption in autism. *Brain Research*, 1079(1), 15–24.
- Istat, Istituto Nazionale di Statistica.
- Istituto Censis. (2012). The hidden dimension of disability. http://www.censis.it/censis/xeditor/visual_edit/35?r=http%253A%252F%252Fwww.censis.it%252F%252F%252Fattachment%252Fprotected_download%252F4129%253Fview_id%253D35
- Istituto Superiore della Sanità. (2011). Linee Guida sul “Trattamento dei disturbi dello spettro autistico nei bambini e negli adolescenti”.
- Keller, F., & Persico, A. M. (2003). The neurobiological context of autism. *Molecular Neurobiology*, 28(1), 1–22.
- Lintas, C., Sacco, R., Garbett, K., Mirnics, K., Militeri, R., Bravaccio, C., et al. (2009). Involvement of the PRKCB1 gene in autistic disorder: Significant genetic association and reduced neocortical gene expression. *Molecular Psychiatry*, 14(7), 705–718.
- Maestro, S., Muratori, F., Cavallaro, M. C., Pecini, C., Cesari, A., Paziente, A., et al. (2005). How young children treat objects and people: An empirical study of the first year of life in autism. *Child Psychiatry and Human Development*, 35(4), 383–396.
- Manzi, B., Loizzo, A. L., Giana, G., & Curatolo, P. (2008). Autism and metabolic diseases. *Journal of Child Neurology*, 23(3), 307–314.
- Masi, G., Cosenza, A., Millepiedi, S., Muratori, F., Pari, C., & Salvadori, F. (2009). Aripiprazole monotherapy in children and young adolescents with pervasive developmental disorders: A retrospective study. *CNS Drugs*, 23(6), 511–521.
- Mazzone, L., Ruta, L., & Reale, L. (2012). Psychiatric comorbidities in asperger syndrome and high functioning autism: Diagnostic challenges. *Annals of General Psychiatry*, 11(1), 16.
- Micheli, E. (1999). *Autismo, verso una migliore qualità della vita*. Reggio Calabria: Laruffa.
- Migoni, P. (1999). Introduzione alla psichiatria dell’età evolutiva. In P. Pancheri, & G. B. Cassano (Eds.), *Trattato Italiano di Psichiatria*, Seconda Edizione (Vol. 3, cap. 69, pp. 2680–2691). Milano: Masson.
- Persico, A. M., & Bourgeron, T. (2006). Searching for ways out of the autism maze: Genetic, epigenetic and environmental clues. *Trends in Neurosciences*, 29(7), 349–358.
- Reda, M. A. (2003). *Sistemi cognitivi complessi e psicoterapia*. Roma: Nuova Italia Scientifica.
- Rizzolatti, G., & Sinigaglia, C. (2008). *Mirrors in the brain: How our minds share actions and emotions*. Oxford/New York: Oxford University Press.
- Scattoni, M. L., Martire, A., Cartocci, G., Ferrante, A., & Ricceri, L. (2012). Reduced social interaction, behavioural flexibility and BDNF signalling in the BTBR T + tf/J strain, a mouse model of autism. *Behavioural Brain Research*, 251, 35–40.
- SINPIA-Società Italiana di Neuropsichiatria dell’Infanzia e dell’Adolescenza. (2005). *Linee guida per l’autismo*. Trento: Erickson.
- Vicari, S., Valeri, G., & Fava, L. (2012). *Autismo. Dalla diagnosi al trattamento*. Il Mulino editore.
- Zappella, M. (1997). *Autismo infantile: Studi sull’affettività e le emozioni*. Roma: La Nuova Italia Scientifica.
- Zuddas, A., Zanni, R., & Usala, T. (2011). Second generation antipsychotics (SGAs) for non-psychotic disorders in children and adolescents: A review of the randomized controlled studies. *European Neuropsychopharmacology*, 21(8), 600–620.

ITC

► Infant/Toddler Checklist

Item Analysis

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Definition

Item analysis is a process in which responses to test items are examined in order to test the quality of those items. Statistical methods are routinely used to identify any test items that do not belong on the test. Items are removed when they do not show the same kind of patterns of association with other items that is observed among retained test items. In addition, an item analysis will reveal if an item is too easy, too difficult, and/or will show a difference between different types of test-takers. Item analysis can be a useful method in confirming the value of test items for discriminating test-takers with ASD from test-takers without ASD when both groups are administered the same test (Baron-Cohen et al. 2001).

See Also

► Validity

References and Reading

- Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from Asperger syndrome/high-functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders*, 31(1), 5–17.
- Harris, D. J., & Subkoviak, M. J. (1986). Item analysis: A short-cut statistic for mastery tests. *Educational and Psychological Measurement*, 46, 495–507.
- Hohensinn, C., & Kubinger, K. D. (2011). Applying item response theory methods to examine the impact of different response formats. *Educational and Psychological Measurement*, 71, 732–746.
- Jones, A. T. (2011). Comparing methods for item analysis: The impact of different item-selection statistics on test difficulty. *Applied Psychological Measurement*, 35, 566–571.

Item Response Theory

► Latent Variable Modeling

Itinerant Teacher

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Synonyms

Consulting teacher; Early childhood tutor; Inclusion specialist; Peripatetic teacher; Traveling teacher; Visiting teacher

Definition

Itinerant teachers travel to provide services to students with disabilities. Instead of functioning

as traditional classroom teachers, itinerants visit children on their caseloads in a variety of settings including homes, early childhood centers, schools, community-based programs, and hospitals. They provide direct services and supports to children with autism as specified by the goals and objectives in their Individual Education Plans (IEP). Academic skills, communication skills, cognitive development, social development, adaptive skills, motor skills, and behavioral support are some of the categories addressed. They also may provide consultation services to the student's primary teacher or caregiver. Itinerant services denote the location where the services are provided as opposed to the nature of the services themselves (Odom et al. 1999).

References and Reading

- Dinnebeil, L. A., McInerney, W. F., & Hale, L. (2004). Mini-theme commentary: Itinerant ECSE teachers in action. *Journal of Educational and Psychological Consultation*, 15(2), 167–175. https://doi.org/10.1207/s1532768xjepc1502_4.
- Freeman, R., & Vakil, S. (2004). The role of family childcare providers in early intervention. *Early Childhood Education Journal*, 32(2), 121–125.
- Jenkins, J. R., & Mayhall, W. F. (1973). Describing resource teacher programs. *Exceptional Children*, 40(1), 35–38.
- Odom, S. L., Horn, E. M., Marquart, J., Hanson, M. J., Wolfberg, P., Beckman, P. J., et al. (1999). On the forms of inclusion: Organizational context and individualized service delivery models. *Journal of Early Intervention*, 22, 185–199.
- Sadler, F. (2003). The itinerant special education teacher in the early childhood classroom. *Teaching Exceptional Children*, 35(3), 8.

ITS

► Interactive Trauma Scale

Japan and Autism

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Historical Background

In the field of child psychiatry in Japan, autism has long been one of the most important topics. The first Japanese case of early infantile autism was reported in 1959. Until the end of the 1970s, early infantile autism (Kanner 1943) and “autistische Psychopathie” (autistic psychopathy) (Asperger 1944) were the two major conceptualizations of autism that were employed. Although numerous clinical articles on autistic conditions written in Japanese were published based on these concepts, Japanese child psychiatrists did not have operational diagnostic criteria for mental disorders in place, including such criteria for autistic conditions, until the introduction of the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III) (American Psychiatric Association [APA] 1980). The subsequent DSM-IV’s (APA 1994) diagnostic criteria have come to be commonly used in research and clinical practice in mental disorders, including pervasive developmental disorders (PDDs) (consisting of five subtypes: autistic disorder, Rett disorder, childhood disintegrative disorder, Asperger’s disorder, and PDD not otherwise specified), although diagnoses

in the International Classification of Diseases, Tenth Revision (ICD-10) (World Health Organization 1993) are used for the national health statistics in Japan. A single category of autism spectrum disorder (ASD) in DSM-5 (APA 2013), which unifies the four DSM-IV PDD subtypes other than Rett disorder, will replace the category of PDDs in research and clinical activities. However, professionals in a non-English-speaking country such as Japan would do better to consider any condition diagnosed as one of the four DSM-IV PDD subtypes to be ASD until a reliable and valid domestic language version of the ASD diagnostic criteria is made available.

Historically, Japanese researchers have made especially pioneering contributions in two areas of research on PDDs. One area is PDD epidemiology. Until the end of the 1980s, the prevalence of infantile autism was reported to be 4–5 per 10,000. Then three Japanese epidemiological studies (Matsuishi et al. 1987; Sugiyama and Abe 1989; Tanoue et al. 1988) independently reported a much higher prevalence of infantile autism, ranging from 13.0 to 15.5 per 10,000. These rates were a harbinger of the high prevalence (62.6–116.1 per 10,000) of PDDs that would later be reported in the UK and the USA (Baird et al. 2006; Bertrand et al. 2001; Chakrabarti and Fombonne 2001). The other area is the phenomenon of regression in autism. Regressive autism was demonstrated to show poorer developmental outcomes in childhood (Kurita 1985) and in adolescence and adulthood

(Kobayashi and Murata 1998) compared with non-regressive autism. On the other hand, the total number of original articles on pathophysiology and etiology of PDDs by Japanese authors was relatively small until the end of the twentieth century. However, it has substantially increased since the beginning of the twenty-first century (e.g., Higashida et al. 2012; Kamio and Toichi 2007; Kato et al. 2008; Nakamura et al. 2010; Sadakata et al. 2012; Suzuki et al. 2013) thanks to the growing interest among researchers coupled with an increasing amount of research funds available from both government ministries and private foundations.

For a long period of time, there were no laws in Japan specifically covering persons with disabilities other than intellectual disabilities (IDs) or physical disabilities. To address this situation, several legislative reforms and amendments to the existing laws have been made over the last 10 years. The “Basic Act for Persons with Disabilities” (Act No. 84 of 1970, amended in 2011) paved the way for persons with disabilities to receive various services equally regardless of the types of disabilities (i.e., physical, intellectual, or mental disabilities [including developmental disabilities]). The “Act for Employment Promotion of Persons with Disabilities” obliged private companies and government offices to have 2 % and 2.3 % of their employees (as amended in 2013), respectively, be persons with disabilities and established systems for supported employment of such persons. The “Act for Supporting Persons with Developmental Disorders” (No. 167 of 2004) was enacted to improve the early detection, remedial treatment, education, employment, and welfare of persons with developmental disorders (DDs) other than IDs not covered by the previous laws (e.g., PDDs, attention-deficit/hyperactivity disorder [AD/HD], and learning disorders) and obliged the 47 prefectures and ordinance-designated cities to establish centers that would support persons with these disorders. The “Act for Supporting the Independence of Persons with Disabilities” (No. 123 of 2005, amended as the “Act of Comprehensive Support for Persons with Disabilities” in 2012) was enacted to

improve the welfare of persons with disabilities so as to enable them to live in the community.

In 2007, special needs education (SNE) to replace the special education system in place prior to that time was implemented in all schools (from kindergarten to high school). SNE aims at providing the relevant education for children with disabilities in order to improve their abilities in dealing with difficulties in everyday lives and their active efforts toward independence and social participation. SNE serves not only children with IDs and children with physical disabilities but also children with DDs other than IDs. In public schools (from elementary to high school), children are educated basically at local government expense regardless of whether they have disabilities or not.

Since PDDs are a part of the DDs and persons with PDDs are covered by the services, systems, and laws for persons with DDs or disabilities, the current knowledge on PDDs/DDs in Japan is described in the following text.

Current Knowledge

Early detection and remedial treatment. The importance of early detection and early intervention in the case of infants with PDDs is well recognized in Japan. All infants have developmental checkups several times in infancy at health centers and pediatric clinics at local government expense, especially at the ages of 1.5 and 3 years. Those checkups are important for the early detection of PDDs. Each local government (city level) has a remedial treatment center for infants with disabilities. A remedial treatment center usually has a clinic which provides child psychiatric/pediatric neurologic examinations and developmental/behavioral evaluations of infants with DDs along with parental guidance. Infants with PDDs detected at infant health checkups are typically enrolled in a remedial treatment group of a remedial treatment center. A remedial treatment group provides infants with PDD activities designed to facilitate their play and interaction with other infants with disabilities in a nursery setting under the guidance of professionals (e.g., nursery

teachers, clinical developmental psychologists, and speech therapists). Parents of infants with PDDs over the age of 3 are encouraged to enroll their children in nursery schools or kindergartens along with participation in remedial treatment groups.

Education. Each local government (city level) has a system in which a panel of experts (e.g., educators and psychologists) advises parents of preschool children with PDDs in selecting a relevant class (an ordinary class with or without the partial provision of SNE or a formal SNE class) of an elementary school or a SNE elementary school for their children. In elementary and junior high school, children with PDDs are educated in ordinary classes with or without the partial provision of SNE, or in SNE classes in ordinary schools, or in SNE schools per se, depending on their levels of intellectual functioning and behavior problems as well as the opinion of the parents. Children with PDDs who graduated from SNE classes in junior high or SNE junior high go to SNE high school. In SNE, teaching methods which have been successfully used abroad, such as the “Applied Behavior Analysis” and “Treatment and Education of Autistic and Related Communication Handicapped Children,” are not systematically incorporated. Instead, besides teaching basic academic and gymnastic skills, the SNE used in classes from elementary to high schools is basically carried out according to the principle of learning through experience, the traditional method of teaching in Japanese special education. Children acquire the skills needed in their everyday lives through the preparation for and accomplishing of organized tasks. For example, if the task is to have a festival, children are taught the steps needed for its effective accomplishment. Then children accomplish each step by learning the necessary skills in cooperation with other children under the guidance of teachers so as to be able to actually carry out the festival. SNE coordinators (teachers having expertise in SNE) help the other teachers deal with children with DDs in various school settings and also advise the parents of the children with DDs. Some high-functioning children with PDDs who graduated from ordinary junior high schools go to SNE high schools, while other such

children, especially those with good intelligence and mild autistic symptoms, are able to attend ordinary high schools. Some of the children with PDDs who graduated from ordinary high schools enter universities. Currently, many universities have a system and staff in place which support students with PDDs.

Employment and work. After graduation from SNE high schools, there are four possible courses or paths for persons with DDs, including PDDs depending on their ability to work. The most able ones work in companies, many of which are branch factories of large corporations that provide employment for persons with disabilities (a monthly income of 100,000 yen [1,000 dollars at a rate of 100 yen to a dollar]). The next most able ones work in “type A” sheltered workshops in preparation for supported employment (a monthly income of 71,513 yen [715.13 dollars]). Persons less able than those working in the type A facilities work in type B sheltered workshops or similar facilities (a monthly income of 7,605–13,742 yen [76.05–137.42 dollars]), and the least able attend facilities designed for the daily lives of persons with disabilities. Persons with high-functioning PDDs who were first diagnosed as having PDDs in late adolescence or adulthood and having had no SNE usually start training for the supported employment program after graduation from high school or university.

Living facilities. There are several types of living facilities in which persons with IDs may live as needed (residential facilities for children with severe IDs, residential facilities for adults with severe IDs, and group homes). Persons with PDDs may enter these facilities if eligible. However, the number of residential facilities for persons with severe IDs will not be increased from the current level, due to the policy of the Japanese government to encourage persons with IDs to live in group homes and not residential facilities. In addition, there are not enough group homes to accept persons in need. Moreover, there are only seven residential facilities for persons with PDDs and severe behavior problems nationwide (five medical facilities capable of providing medical care for patients with behavior problems and two welfare-type facilities).

Pharmacological treatment. Antipsychotics, mainly atypical ones, are commonly used to treat severe behavior problems in persons with PDDs. However, the effects of antipsychotics in such cases have not been demonstrated in randomized controlled trials (RCTs) except for pimozide in Japan. Atypical antipsychotics (e.g., aripiprazole and risperidone) are frequently used to treat severe behavior problems in persons with PDDs based on the positive results obtained in RCTs conducted abroad. The effects of the medications used in the treatment of other comorbid mental disorders (e.g., mood disorders, anxiety disorders, and tic disorders) in persons with PDDs have not been demonstrated in RCTs. Recent progress in the pharmacological treatment of PDDs is the introduction of osmotic-release methylphenidate and atomoxetine to treat comorbid AD/HD symptoms based on research findings that demonstrated such comorbidity.

Financial support. In Japan, there are many types of financial support for persons with disabilities covered by laws for which persons with PDDs are eligible. A person (usually a parent) who is under duty to support a child younger than the age of 20 years with ID of moderate to severe grade (or other disabilities of equivalent severity) is eligible to receive a special child-rearing allowance of 33,570 or 50,400 yen (335.7 or 504 dollars) a month depending on the severity of the disability, provided the annual income of the person or any other person in the same household is not exceeding the norm which rises with increasing number of dependents of the person. An adult from the age of 20 years on with a disability is eligible to receive a pension of 65,541 or 81,925 yen (655.41 or 819.25 dollars) a month depending on the severity of the disability. By the "Act for Supporting the Independence of Persons with Disabilities," persons with disabilities pay none or 10 % of the cost of certain welfare services and medical treatments specified in the act. In the case of 10 % payment, persons with disabilities do not need to pay exceeding the upper limits of the monthly payments (2,500–20,000 yen [25–200 dollars]) which are set according to the household income tax levels.

In addition, 70–90 % of medical treatment expenses other than those specified in this act, such as dental care and the treatment of disease, are paid by one of the public health insurance programs which cover all Japanese citizens. Many local governments have systems in which medical treatment is freely provided to all children younger than 16 years of age.

Organizations of parents and professionals. The Autism Society Japan (ASJ) was established in 1967. The ASJ has conducted various activities such as providing counseling services for persons with PDDs and their family members, holding lectures and workshops, collecting information, and supporting research on PDDs. The Japan League on Developmental Disabilities (JLDD) was established in 1974. The JLDD has conducted various activities, such as supporting persons with developmental disabilities and professionals not only in Japan but also in developing countries, collecting data and information worldwide, and holding national and international academic meetings on developmental disabilities. The Japan DDs Network (JDDNET) was established in 2006. The JDDNET, which is comprised of representatives of associations of parents of children with DDs as well as representatives of associations of concerned professionals, also has conducted diverse activities of supporting persons with DDs and their family members, advancing the understanding of DDs in the society, promoting the welfare of persons with DDs, and carrying out lobbying activities.

Research centers and academic societies. There are three national centers and one national institute which have sections for conducting research on DDs, including PDDs: the National Center of Neurology and Psychiatry, the National Center for Child Health and Development, the National Rehabilitation Center for Persons with Disabilities, and the National Institute of Special Needs Education. There are five major academic societies concerned with research on DDs, including PDDs: the Japanese Academy of Autistic Spectrum, the Japanese Association for the Study of Developmental Disabilities, the Japanese Society for Child and Adolescent Psychiatry, the Japanese Association for Medical and

Psychological Study of Infants, and the Japanese Association of Special Education.

Training professionals. All of the 80 medical schools and the great majority of university departments of education, psychology, and welfare train students in fields related to the DDs, including the PDDs, and also perform research. However, the number of professionals (e.g., physicians, teachers, psychologists, and social workers) having expertise in DDs is quite insufficient to deal with the DD population.

Future Directions

Although the early detection, treatment, education, and welfare provided for persons with DDs including PDDs have shown evident progress over the last 10 years in Japan, many issues remain. First, there is a need to improve the systems for employing persons with DDs. Second, it is essential to provide financial assistance for persons with DDs to enable them to live in the community, because the majority of the adults with DDs get only a pension of 65,541 yen (655.41 dollars) a month, which is by no means adequate to actually sustain them. Third, there is a need to provide an adequate number of group homes for persons with DDs and residential facilities for persons with PDDs and severe behavior problems. Fourth, it is critically important to increase the number of professionals having expertise in DDs. Finally, there is a need to investigate the effects of not only pharmacological treatment of behavior problems and comorbid mental disorders but also methods in remedial treatment and SNE in persons with DDs including PDDs to make them evidence-based practices.

See Also

- ▶ Italy and Autism
- ▶ Kuwait and Autism
- ▶ Russia and Autism
- ▶ Turkey and Autism

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association.
- Asperger, H. (1944). Die 'Autistischen Psychopathen' im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Baird, G., Simonoff, E., Pickles, A., Chandler, S., Loucas, T., Meldrum, D., & Charman, T. (2006). Prevalence of disorders of the autism spectrum in a population cohort of children in South Thames: The Special Needs and Autism Project (SNAP). *Lancet*, 368, 210–215. doi:10.1016/S0140-6736(06)69041-7.
- Bertrand, J., Mars, A., Boyle, C., Bove, F., Yeargin-Allsopp, M., & Decoufle, P. (2001). Prevalence of autism in a United States population: The Brick Township, New Jersey, investigation. *Pediatrics*, 108, 1155–1161. doi:10.1542/peds.108.5.1155
- Chakrabarti, S., & Fombonne, E. (2001). Pervasive developmental disorders in preschool children. *JAMA*, 285, 3093–3099. doi:10.1001/jama.285.24.3093.
- Higashida, H., Yokoyama, S., Huang, J. J., Liu, L., Ma, W. J., Akther, S., . . . Munesue, T. (2012). Social memory, amnesia, and autism: Brain oxytocin secretion is regulated by NAD⁺ metabolites and single nucleotide polymorphisms of CD38. *Neurochemistry International*, 61, 828–838. doi:10.1016/j.neuint.2012.01.030.
- Kamio, Y., & Toichi, M. (2007). Memory illusion in high-functioning autism and Asperger's disorder. *Journal of Autism and Developmental Disorders*, 37, 867–876. doi:10.1007/s10803-006-0214-y.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kato, C., Tochigi, M., Ohashi, J., Koishi, S., Kawakubo, Y., Yamamoto, K., . . . Sasaki, T. (2008). Association study of the 15q11-q13 maternal expression domain in Japanese autistic patients. *American Journal of Medical Genetics, Part B, Neuropsychiatric Genetics*, 147B, 1008–1012. doi:10.1097/YPG.0b013e3282fb0064.
- Kobayashi, R., & Murata, T. (1998). Setback phenomenon in autism and long-term prognosis. *Acta Psychiatrica Scandinavica*, 98, 296–303. doi:10.1111/j.1600-0447.1998.tb10087.x.
- Kurita, H. (1985). Infantile autism with speech loss before the age of thirty months. *Journal of the American Academy of Child Psychiatry*, 24, 191–196. doi:10.1016/S0002-7138(09)60447-7.
- Matsuishi, T., Shiotsuki, Y., Yoshimura, K., Shoji, H., Imuta, F., & Yamashita, F. (1987). High prevalence of infantile autism in Kurume City, Japan. *Journal of Child Neurology*, 2, 268–271. doi:10.1177/088307388700200406.

- Nakamura, K., Sekine, Y., Ouchi, Y., Tsujii, M., Yoshikawa, E., Futatsubashi, M., . . . Mori, N. (2010). Brain serotonin and dopamine transporter bindings in adults with high-functioning autism. *Archives of General Psychiatry*, 67, 59–68. doi:10.1001/archgenpsychiatry.2009.137.
- Sadakata, T., Shinoda, Y., Oka, M., Sekine, Y., Sato, Y., Saruta, C., . . . Furuichi, T. (2012). Reduced axonal localization of a Caps2 splice variant impairs axonal release of BDNF and causes autistic-like behavior in mice. *Proceedings of the National Academy of Sciences of the United States of America*, 109, 21104–21109. doi:10.1073/pnas.1210055109.
- Sugiyama, T., & Abe, T. (1989). The prevalence of autism in Nagoya, Japan: A total population study. *Journal of Autism and Developmental Disorders*, 19, 87–96. doi:10.1007/BF02212720.
- Suzuki, K., Sugihara, G., Ouchi, Y., Nakamura, K., Futatsubashi, M., Takebayashi, K., . . . Mori, N. (2013). Microglial activation in young adults with autism spectrum disorder. *JAMA Psychiatry*, 70, 49–58. doi:10.1001/jamapsychiatry.2013.272.
- Tanoue, Y., Oda, S., Asano, F., & Kawashima, K. (1988). Epidemiology of infantile autism in southern Ibaraki, Japan: Differences in prevalence in birth cohorts. *Journal of Autism and Developmental Disorders*, 18, 155–166. doi:10.1007/BF02211943.
- World Health Organization. (1993). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research*. Geneva: World Health Organization.

JASPER

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Definition

JASPER: Joint Attention, Symbolic Play, Engagement, and Regulation (JASPER) is a targeted, comprehensive social communication intervention that is included in the broader category of naturalistic developmental behavioral interventions (NDBI; Schreibman et al. 2015).

Goals and Objectives

First developed as a one-on-one intervention, interventionists assess the child to identify developmentally appropriate social communication and play level targets and then select motivating toys that match and advance the child's current skill level. Sessions are densely packed with targeted, systematic instruction designed to engage the child in the development of play routines. These routines provide opportunities to advance children's play skills (both flexibility and complexity of play) and also create the context for communication. The goals of JASPER are to increase: (a) the child's time jointly engaged in a shared activity with a social partner, (b) the diversity, flexibility, and complexity of children's play skills, and (c) spontaneous initiations of both nonverbal and spoken (and/or augmented) language for the purpose of joint attention (socially sharing with others by pointing, showing, or giving objects and/or commenting on the object) and requesting.

Historical Background

The JASPER approach is grounded in children's early development and informed by experimental research examining the similarities and differences in the emergence of core early childhood domains including social engagement with others, social communication skills, and play skills. Research in the 1980s and 1990s demonstrated significant delays in the development of initiations of joint attention skills specific to young children with ASD when compared to children with other developmental disorders and typically developing children (e.g., Mundy et al. 1986; Sigman et al. 1995). Further studies demonstrated differences in the development of play skills (e.g., Ungerer and Sigman 1981) and joint engagement (e.g., Adamson et al. 2009). Yet, these core domains including nonverbal communication and, specifically, initiations of joint attention skills were found to predict language skills for children with autism (e.g., Mundy et al. 1994; Toth et al. 2006) solidifying their critical importance to early development.

and highlighting the need for interventions to support the development of these core skills.

Rationale or Underlying Theory

JASPER follows a developmental progression of skills informed by typical development and research that indicates differences in communicative development unique to children with ASD (e.g., Paparella et al. 2011). The intervention is designed to be individualized. Target skills (goals) based on the developmental hierarchy of each domain are identified for each child to, in essence, meet the child where they are at present, and support their growth based on developmentally appropriate targets. The intervention works within play with objects, a natural learning context for young children. Play routines provide a sensitive and responsive bidirectional context where the actions of the interventionist are informed moment by moment based on the child's signals of engagement, regulation, and communication. The interventionist works to support the child's entry into and maintenance of a joint engaged state. Unlike states where the child is focused entirely on objects to the exclusion of others (object engaged), entirely on a person (person engaged) or not engaged with people or objects (unengaged), time spent jointly engaged has been linked to gains in spoken language (Adamson et al. 2004). Joint engagement allows for a rich learning context where the child may notice the adult's actions on the materials, as well as nonverbal and verbal communication mapped to the materials in front of them. By establishing periods of joint engagement in play, the interventionist sets the foundation to create further opportunities to work on both play and social communication skills.

Treatment Participants

JASPER trials have included toddlers (e.g., Kasari et al. 2010) and preschool age children (e.g., Kasari et al. 2014b). Further, JASPER has been examined with children who have little or no spoken language (referred to as minimally verbal:

Tager-Flusberg and Kasari 2013) who are school-age (e.g., Kasari et al. 2014a). Additional trials are in progress focusing on infants (12–21 months) at risk for autism, children with Down syndrome, and children with tuberous sclerosis complex.

JASPER has been extended to a caregiver-mediated format where parents are coached to use the intervention strategies with their children in both play and home routines (e.g., Kasari et al. 2014b, 2015). Further, JASPER has also been mediated by teachers and paraprofessionals in preschool classroom settings (e.g., Lawton and Kasari 2012; Chang et al. 2016) and early intervention center-based classrooms (Shire et al. 2017).

Treatment Procedures

In JASPER research trials, sessions are 30–60 min in length, typically delivered one on one with a trained clinician and the child, and can occur in clinics, schools, homes, and other community locations. Treatment targets are set during initial assessments. Community trials have included the Short Play and Communication Evaluation (SPACE: Shire et al. 2016a), a brief play-based tool designed to provide community providers with a structured protocol to identify children's mastered skills and set intervention targets for both social communication and play. Based on this information, the interventionist will select play materials that allow for the development of play routines that include the child's mastered play skills and their target play level. Interventionists then work to engage the child in play-based interactions following a manualized protocol. Fidelity is assessed based on video review and includes seven subscales (see Table 1). Both the quality and quantity of the application of the strategies is evaluated. The interventionist is required to individualize the session to first match the child's developmental level and then to provide structure/prompting as required to support the child's engagement and regulation during the session. Progress is noted each session through logs recorded by the interventionist and summative assessment is conducted approximately every 3 months (or 24 sessions).

JASPER, Table 1 JASPER strategy subscales

Subscale	Description of items
Basic strategies to support engagement and regulation	They include foundational interaction strategies such as modeling affect, providing space for the child to communicate, noticing and following in on the child's choices and appropriate actions, and contingent responding. The adult provides appropriate and timely supports to help the child stay regulated and engaged in the play.
Environment	The adult selects developmentally appropriate toy options, provides an appropriate number of toy choices in the environment, sits directly in front of the child, and removes any distractions from the environment.
Balances imitation and modeling	The adult immediately imitates the child's appropriate play actions where the child can see and notice the action. The adult models developmentally appropriate play acts when more structure and support is needed.
Establishing routines	The adult and child create a play routine that has clear steps, where both parties have active roles as play partners. The steps in the routine are at the child's play level and the routine is motivating for the child.
Expanding routines	The adult provides environmental support to help the child add new steps to the play. If the child expands, the adult follows in. If the child does not expand, the adult provides more structure and support to expand the routine.
Programming for joint attention and requesting	The adult notices and responds to the child's initiations of joint attention and requesting skills. The adult also models appropriate skills throughout the interaction and works to provide explicit opportunities for the child to initiate these skills.
Language strategies	The adult imitates and expands the child's appropriate language, and models language at the child's target level.

Coaching caregivers. JASPER intervention has also been tested when delivered by caregivers (e.g., Kasari et al. 2010, 2014b, 2015), and classroom staff including head classroom teachers and teaching assistants/paraprofessionals (e.g., Chang et al. 2016; Shire et al. 2017). To support the adoption of the JASPER intervention strategies by both caregivers and educational staff, trained interventionists provide coaching and feedback while the caregiver/educator practices with the child. Sessions are typically 30 min in length and the content is delivered in progressive segments based on the seven strategy subscales outlined in Table 1. The session typically begins with a visual content handout to help relay the focus of the session (e.g., engagement states, how to balance imitation and modeling). Discussion is kept to only a couple of minutes in order to focus on engaging the child and providing the maximum time for supported practice. Coaches provide supports ranging from modeling strategies for the caregiver/educator (most support), paired interactive practice where both the coach and the caregiver/educator deliver the strategies in

tandem with the child, verbal feedback and tips, and environmental supports (least support). The balance of strategies will depend on the learning style of the adult, the confidence and quality of their implementation, and how many sessions have occurred by that point. Coaches typically begin the first session with the most support (modeling) with the aim to fade the level of support and quickly transfer first the foundational strategies and then the more complex strategies to the caregiver/educator.

Efficacy and Effectiveness Information

Efficacy. The JASPER intervention has been tested in nine randomized controlled trials (RCTs) involving nearly 500 children with ASD over the course of 20 years. JASPER has been tested first as Joint Attention and Symbolic Play modules provided in addition to an intensive ABA center-based program (Kasari et al. 2006, 2008). These modules were then combined together and refined to what was then tested as JASPER.

JASPER trials have compared receipt of JASPER with community treatment as usual controls including agency-based early intervention services, and preschool-/school-based services, and more recently using adaptive intervention designs. Altogether, these trials demonstrate gains in children's time jointly engaged in play activities with others (noticing both the partner and the shared activity), initiations of joint attention (e.g., Kasari et al. 2010, 2014b), language (e.g., Kasari et al. 2008), and play diversity (e.g., Kasari et al. 2010) and play level (e.g., Kasari et al. 2014b).

Caregiver-Mediated. In addition, similar gains have been demonstrated when JASPER is mediated by caregivers (Kasari et al. 2010, 2014b, 2015). During initial trials, caregivers were invited into a clinic setting and received direct coaching to deliver the intervention in comparison to caregivers receiving information but no direct coaching (Kasari et al. 2010, 2015), while in Kasari et al. (2014b), interventionists worked directly with low resource families in their homes contrasted with assignment to a group information intervention. In all three studies, caregivers who received direct JASPER coaching demonstrated gains in their delivery of the intervention strategies, and children showed gains in the primary outcome time jointly engaged. Gains in secondary outcomes include increases in play level (Kasari et al. 2014b, 2015), toddlers demonstrated gains in responding to joint attention (Kasari et al. 2010), and preschoolers demonstrated gains in initiations of joint attention (Kasari et al. 2014b). Caregivers who receive JASPER coaching have also demonstrated greater gains in their responsiveness to their child's communicative bids over information only (e.g., Shire et al. 2016b), and greater strategy adoption by caregivers was found to be associated with children's time jointly engaged (Shire et al. 2015).

Teacher-Mediated. Further, JASPER has been adapted for small group settings and deployed through teachers and paraprofessionals in preschool and early intervention classroom settings (Chang et al. 2016; Lawton and Kasari 2012; Shire et al. 2017). In both cases, children who received JASPER versus preschool or early

intervention as usual demonstrated greater gains in time jointly engaged in play rotations with the classroom staff, gains in initiations of joint attention, gains in language to request and to share, and preschoolers demonstrated gains in time spent in functional play routines (Chang et al. 2016). These gains were demonstrated in a proximal measure of change (during the interactions with the classroom staff) as well as distal measures conducted by research staff (Chang et al. 2016) or center staff unfamiliar to the students and trained to deliver the measure (Shire et al. 2017).

Focus on children who have minimal spoken language. JASPER intervention trials have also specifically focused on supporting the communicative development of children with autism who have few or no spoken words. This group of children is characterized as being *minimally verbal* where children enter the intervention with fewer than 20 functional spontaneous words (Tager-Flusberg and Kasari 2013) as measured through natural language samples. Two trials have been published focusing on this population including one pilot trial with children aged 3–5 years where participants received 30 h a week of treatment as usual ABA therapy alone or the ABA therapy plus JASPER for 30 min twice a week for 12 weeks (Goods et al. 2013) leading to gains in initiations of gestures and less time unengaged for children receiving JASPER. The second trial included children aged 5–8 years in a Sequential Multiple Assignment Randomized Trial (SMART) design where children received a combination of JASPER with a requesting language prompting paradigm (EMT: Enhanced Milieu Teaching; Kaiser et al. 2000). All children received the combined JASPER plus EMT intervention where children were initially randomized to also receive access to a speech-generating device (SGD) in treatment sessions or not. After 12 weeks, the child's individual response (gains in spontaneous communication) were assessed. For those children responding slowly, the intervention was augmented by increasing the number of sessions to three per week or providing access to the SGD (see Kasari et al. 2014a for study design details). Children who received access to the SGD demonstrated quicker gains in spontaneous

communicative utterances in the first 12 weeks (Kasari et al. 2014a), and also demonstrated greater gains in initiations of joint attention (Almirall et al. 2016). Together, these two trials provide evidence for positive gains in spontaneous communication for preverbal preschoolers with autism and minimally verbal school age children with autism. Additional trials building on these findings are currently in the field.

Independent Replications. JASPER has also been examined in independent replications (Kaale et al. 2012, 2014). Focused on supporting preschool teachers, this trial and the 1-year follow-up data show gains in initiations of joint attention and play skills over preschool as usual control.

Outcome Measurement

Key outcomes reported in JASPER trials include three core domains: (a) engagement, (b) social communication skill, and (c) play skills.

Engagement. JASPER prioritizes extending the amount of time that children spend in a joint engaged state. Joint engagement refers to a period of time where the child notices both the interaction partner and the shared activity (Adamson et al. 2009). Children may actively reference the adult with eye contact, initiate discrete joint attention skills, and direct the play in a coordinated joint engaged state or they may indicate their awareness through imitation of the partner's play models and/or language, and actively take turns in the play in a supported joint engaged state (Adamson et al. 2009). Gains in time jointly engaged have been demonstrated proximally, for example, in session with clinician or in classroom rotation with teacher, and in interactions between the caregiver and their child in caregiver-mediated trials (e.g., Kasari et al. 2010). Further, changes in engagement have been found to generalize to interactions with partners who were not taught the intervention strategies. For example, Goods et al. (2013) delivered JASPER by clinicians to a small sample of minimally verbal 3–5-year-olds and found the children spending less time unengaged in classroom observations, while Kaale and colleagues (2010) who focused on

teaching preschool teachers found gains in time jointly engaged in the caregiver–child interactions over control.

Social communication. Social communication encompasses initiations of joint attention, responding to joint attention, initiations of behavior regulations (requesting), and spontaneous communicative utterances (spoken and augmented words for the purpose of requesting or joint attention). Key to JASPER outcomes is the focus on children's spontaneous initiations and the exclusion of adult-prompted response. Social communication is often examined using the Early Social Communication Scales (Mundy et al. 2003) which is a semistructured toy-based measure where children are presented with a standard set of objects including toys, a book, hat, car, etc. designed to capture nonverbal and verbal initiations of joint attention and requesting. In teacher-mediated trials, teachers are provided with support to administer the Short Play and Communication Evaluation (SPACE: Shire et al. 2016) to set developmentally appropriate social communication and play target skills, and to assess progress (Shire et al. 2017). For very young children (e.g., infants and toddlers), gains are expected in developmentally young skills such as responding to joint attention (e.g., Kasari et al. 2010) and initiations of joint attention for older children (e.g., Kasari et al. 2014).

Play skills. JASPER places a unique focus on targeting the development of play diversity and increase in developmental play level. Based on typical development, the intervention follows a hierarchy of play skills (adapted from Ungerer and Sigman 1981; Lifter et al. 1993). Play is assessed both within the interaction of clinicians/teachers/caregivers with the children as well as on semistructured assessments of play delivered by independent evaluators (e.g., Structured Play Assessment: adapted from Ungerer and Sigman 1981). Children receiving JASPER have demonstrated both gains in play diversity measured as the number of different unique types of play acts at a given level (e.g., Kasari et al. 2015, 2006) as well as advancement in the complexity/level of the play (e.g., gains in symbolic play Kasari et al. 2014, 2006).

Follow up. Across the trials, follow-up assessments range from 1 to 6 months post treatment exit. Overall, continued gains at follow up have been demonstrated for engagement and joint attention. Kaale et al. (2014) also published a 1 year follow-up study on children enrolled in their 2012 RCT which demonstrated gains in joint engagement and initiations of joint attention for children in JASPER over the control group. However, no significant differences in language skills were found.

Longer follow-up studies have specifically addressed language outcomes across treatment groups. Children enrolled in Kasari et al. (2006) were assessed 1 year post treatment where children who received targeted joint attention or symbolic play intervention demonstrated gains in language over children in the ABA-only control (Kasari et al. 2008). Further, 5 years post treatment, assignment to joint attention or symbolic play interventions predicted greater expressive language skills at age 8 years (Kasari et al. 2012).

Qualification of Treatment Providers

Providers/clinicians must receive training to deliver JASPER. JASPER training begins with supporting a clinician to work directly with children, and then additional training is provided to coach others (e.g., caregiver mediated JASPER, training other clinicians). Clinicians applying for training should have a background working with children with ASD.

References and Reading

- Adamson, L. B., Bakeman, R., & Deckner, D. F. (2004). The development of symbol-infused joint engagement. *Child development*, 75, 1171–1187.
- Adamson, L. B., Bakeman, R., Deckner, D. F., & Ronski, M. (2009). Joint engagement and the emergence of language in children with autism and down syndrome. *Journal of Autism and Developmental Disorders*, 39(1), 84.
- Almirall, D., DiStefano, C., Chang, Y. C., Shire, S., Kaiser, A., Lu, X., ..., & Kasari, C. (2016). Longitudinal effects of adaptive interventions with a speech-generating device in minimally verbal children with ASD. *Journal of Clinical Child & Adolescent Psychology*, 45(4), 442–456.
- Chang, Y. C., Shire, S. Y., Shih, W., Gelfand, C., & Kasari, C. (2016). Preschool deployment of evidence-based social communication intervention: JASPER in the classroom. *Journal of Autism and Developmental Disorders*, 46(6), 2211–2223.
- Goods, K. S., Ishijima, E., Chang, Y. C., & Kasari, C. (2013). Preschool based JASPER intervention in minimally verbal children with autism: Pilot RCT. *Journal of Autism and Developmental Disorders*, 43(5), 1050–1056.
- Gulsrud, A. C., Hellemann, G., Shire, S., & Kasari, C. (2016). Isolating active ingredients in a parent-mediated social communication intervention for toddlers with autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 57(5), 606–613.
- Kaale, A., Smith, L., & Sponheim, E. (2012). A randomized controlled trial of preschool-based joint attention intervention for children with autism. *Journal of Child Psychology and Psychiatry*, 53(1), 97–105.
- Kaale, A., Fagerland, M. W., Martinsen, E. W., & Smith, L. (2014). Preschool-based social communication treatment for children with autism: 12-month follow-up of a randomized trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(2), 188–198.
- Kaiser, A. P., Hancock, T. B., & Nietfeld, J. P. (2000). The effects of parent-implemented enhanced milieu teaching on the social communication of children who have autism. *Early Education and Development*, 11(4), 423–446.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in young children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry*, 47, 611–620.
- Kasari, C., Paparella, T., Freeman, S., & Jahromi, L. B. (2008). Language outcome in autism: Randomized comparison of joint attention and play interventions. *Journal of Consulting and Clinical Psychology*, 76(1), 125.
- Kasari, C., Gulsrud, A. C., Wong, C., Kwon, S., & Locke, J. (2010). Randomized controlled caregiver mediated joint engagement intervention for toddlers with autism. *Journal of Autism and Developmental Disorders*, 40(9), 1045–1056.
- Kasari, C., Gulsrud, A., Freeman, S., Paparella, T., & Hellemann, G. (2012). Longitudinal follow-up of children with autism receiving targeted interventions on joint attention and play. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(5), 487–495.
- Kasari, C., Kaiser, A., Goods, K., Nietfeld, J., Mathy, P., Landa, R., ..., & Almirall, D. (2014a). Communication interventions for minimally verbal children with autism: A sequential multiple assignment randomized trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(6), 635–646.
- Kasari, C., Lawton, K., Shih, W., Barker, T. V., Landa, R., Lord, C., ..., & Senturk, D. (2014b). Caregiver-

- mediated intervention for low-resourced preschoolers with autism: An RCT. *Pediatrics*, 134(1), e72–e79.
- Kasari, C., Gulsrud, A., Paparella, T., Hellemann, G., & Berry, K. (2015). Randomized comparative efficacy study of parent-mediated interventions for toddlers with autism. *Journal of Consulting and Clinical Psychology*, 83(3), 554.
- Lifter, K., Sulzer-Azaroff, B., Anderson, S., Cowdery, G. (1993). Teaching Play Activities to Preschool Children with Disabilities: The Importance of Developmental Considerations. *Journal of Early Intervention*, 17, 139–59.
- Lawton, K., & Kasari, C. (2012). Teacher-implemented joint attention intervention: Pilot randomized controlled study for preschoolers with autism. *Journal of Consulting and Clinical Psychology*, 80(4), 687–693.
- Mundy, P., Sigman, M., Ungerer, J., & Sherman, T. (1986). Defining the social deficits of autism: The contribution of non-verbal communication measures. *Journal of Child Psychology and Psychiatry*, 27(5), 657–669.
- Mundy, P., Sigman, M., & Kasari, C. (1994). Joint attention, developmental level, and symptom presentation in autism. *Development and Psychopathology*, 6(3), 389–401.
- Mundy, P., Delgado, C., Block, J., Venezia, M., Hogan, A., & Seibert, J. (2003). *Early social communication scales (ESCS)*. Coral Gables: University of Miami.
- Paparella, T., Goods, K. S., Freeman, S., & Kasari, C. (2011). The emergence of nonverbal joint attention and requesting skills in young children with autism. *Journal of Communication Disorders*, 44(6), 569–583.
- Schreibman, L., Dawson, G., Stahmer, A. C., Landa, R., Rogers, S. J., McGee, G. G., ..., & McNeerney, E. (2015). Naturalistic developmental behavioral interventions: Empirically validated treatments for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(8), 2411–2428.
- Shire, S. Y., Goods, K., Shih, W., Distefano, C., Kaiser, A., Wright, C., ..., & Kasari, C. (2015). Parents' adoption of social communication intervention strategies: Families including children with autism spectrum disorder who are minimally verbal. *Journal of autism and developmental disorders*, 45(6), 1712–1724.
- Shire, S. Y., Shih, W., Chang, Y. C., & Kasari, C. (2016a). Short play and communication evaluation: Teachers' assessment of core social communication and play skills with young children with autism. *Autism*. <https://doi.org/10.1177/1362361316674092>.
- Shire, S. Y., Gulsrud, A., & Kasari, C. (2016b). Increasing responsive parent-child interactions and joint engagement: Comparing the influence of parent-mediated intervention and parent psychoeducation. *Journal of Autism and Developmental Disorders*, 46(5), 1737–1747.
- Shire, S. Y., Chang, Y. C., Shih, W., Bracaglia, S., Kodjoe, M., & Kasari, C. (2017). Hybrid implementation model of community-partnered early intervention for toddlers with autism: A randomized trial. *Journal of Child Psychology and Psychiatry*, 58(5), 612–622.
- Sigman, M., Kasari, C., Moore, C., & Dunham, P. (1995). Joint attention across contexts in normal and autistic children. In Moore, C., Dunham, P.J., & Dunham, P. (eds), *Joint attention: Its origins and role in development*, Psychology press, (pp. 189–203).
- Tager-Flusberg, H., & Kasari, C. (2013). Minimally verbal school-aged children with autism spectrum disorder: The neglected end of the spectrum. *Autism Research*, 6(6), 468–478.
- Toth, K., Munson, J., Meltzoff, A. N., & Dawson, G. (2006). Early predictors of communication development in young children with autism spectrum disorder: Joint attention, imitation, and toy play. *Journal of Autism and Developmental Disorders*, 36(8), 993–1005.
- Ungerer, J. A., & Sigman, M. (1981). Symbolic play and language comprehension in autistic children. *Journal of the American Academy of Child Psychiatry*, 20(2), 318–337.

Job Carving

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Synonyms

[Job development](#); [Tailoring job skills](#)

Definition

Job carving is a supported employment strategy, designed to provide additional employment opportunities for individuals with autism and other disabilities. Job carving involves creating, modifying, or customizing a community-based job such that it can be successfully performed by an individual with disabilities, while simultaneously meeting the needs of an employer. Job carving typically involves conducting a task analysis of a job by breaking it down into a series of smaller steps. This allows an employer or vocational specialist to identify which parts of a job might be completed by an individual with disabilities. Skills carved out from several jobs can be combined into a new job that is tailored to fit the skills, preferences, and level of supports required

for an individual, and meets the needs of the employer. Ideally, job carving capitalizes on the supports available in the natural environment to maximize independence, while minimizing external supports (e.g., a job coach).

See Also

- [Supported Employment](#)
- [Vocational Training](#)

References and Reading

- Gerhardt, P. (2007). Notes from the field: Effective transition planning for learners with autism spectrum disorders approaching adulthood. *Journal for Vocational Special Needs Education*, 29, 35–37.
- Griffin, C. (1996). Job carving as a job development strategy. In D. DiLeo & D. Langton (Eds.), *Facing the future: Best practices in supported employment*. TRN: St. Augustine.
- Nietupski, J. A., & Hamre-Nietupski, S. (2000). A systematic process for carving supported employment positions for people with severe disabilities. *Journal of Developmental and Physical Disabilities*, 12, 103–119.

Job Coach

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Synonyms

[Employment specialist](#); [Vocational trainer](#)

Definition

Supported employment involves providing systems of supports for individuals with disabilities, so they can participate in competitive employment opportunities in integrated workplace settings. One of the most common methods of providing supports to individuals with autism is

job coaching. Job coaching refers to the systematic training of an individual with disabilities in the workplace, by an experienced trainer. The job coach teaches an individual with a disability how to complete an assigned job to the same standards that all workers are expected to achieve. In addition, the job coach teaches other skills that are required to maintain employment in a community setting, such as interacting appropriately with co-workers, accepting feedback from a supervisor, remaining on-task such that an appropriate amount of work is completed each day, etc. Ultimately, the goal of the job coach is to fade their supports over time, and have the individual maintain productivity and appropriate workplace behavior. Job coaching has allowed individuals with a variety of disabilities, including autism, to successfully maintain employment in a wide variety of workplace settings.

See Also

- [Employment Specialist](#)
- [Supported Employment](#)
- [Vocational Training](#)

References and Reading

- Bond, G. R., Drake, R. E., Mueser, K. T., & Becker, D. R. (1997). An update on supported employment for people with severe mental illness. *Psychiatric Services*, 48, 335–346.
- Keel, J. H., Mesibov, G. V., & Woods, A. V. (1997). TEACCH-supported employment program. *Journal of Autism and Developmental Disorders*, 27, 3–9.

Job Counselor

- [Vocational Evaluator](#)

Job Development

- [Job Carving](#)

Job Readiness

► Preemployment Skills for People with ASD

Joint Attention

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Definition

The developmental process of communication is rapid during the first few months of life. Children initially learn to communicate through gestures before they begin talking (Bates 1976; Bruner 1983). Children first communicate requests or interest in an object through eye contact. By 9–14 months of age, children are able to communicate using a variety of gestures, such as pointing, showing, and giving. These early gestures have different functions. Some serve to request that a person do something (protoimperative or requesting gestures). Others are merely intended to share an event or object with another person (protodeclarative or joint attention gestures).

While both protoimperative and protodeclarative gestures develop within the same time frame, children with autism spectrum disorder (ASD) have specific impairments in the development of protodeclarative or joint attention gestures. Many studies have shown that joint attention gestures are specifically impaired in children with ASD (e.g., Curcio 1978; Loveland and Landry 1986; Mundy et al. 1986). Moreover, research has shown that it is easier for young children with ASD to *respond* to the gestures of others rather than to spontaneously *initiate* joint attention gestures (Mundy et al. 1986). Further, close preliminary examination of the sequence in which joint attention skills emerge has indicated differences in children with ASD compared to typically developing peers matched for expressive language age. For children with ASD, pointing and showing to share develop at a later expressive

language age (ELA beyond 20 months), while these skills emerge for typically developing children prior to 20 months ELA. Further, gaze following is last to develop at over 46 months ELA in children with ASD versus at under 20 months in typically developing children. In contrast, the sequence of emergence of requesting skills in children with ASD is similar to typically developing children, with only a delay in pointing to request (Paparella et al. 2011).

Perhaps even more important, the joint attention impairments that young children with autism exhibit impact later development. Mundy et al. (1990) showed that children who had more developed joint attention gesturing demonstrated better language skills concurrently and over time. Other studies note similar effects of joint attention gestures on expressive language (Bono et al. 2004; Dawson et al. 2004; Mundy et al. 1987). Joint attention may also impact other areas of development, such as theory of mind, emotion regulation, and symbolic play (Adamson and Russell 1999; Morales et al. 2005; Mundy and Hogan 1994; Whalen et al. 2006). Thus, joint attention is a core challenge that serves a pivotal role in the development of social communication competence for children with autism.

Joint attention is a core deficit that should be a target of early intervention programs (Mundy and Crowson 1997; Mundy et al. 2009; National Autism Center 2009). After all, it is commonly believed that the long-term social communication outcomes of children with autism may be best if children participate in early intervention that targets the core deficits of autism (Kasari et al. 2005). While it is unlikely that children with autism will “recover” through any early intervention, targeting core deficits may lead to more optimal pruning in certain areas of the brain (Mundy et al. 1997; Mundy 2016). Thus, if intervention is implemented early while the brain is most plastic, it could trigger profound improvements in key domains that depend upon the core deficit (Bruinsma et al. 2004; Mundy et al. 2009).

Several published interventions report success at improving joint attention. Treatment-induced improvements in joint attention are reported for responding to joint attention (e.g., Whalen and Schreibman 2003) and initiating joint attention

(Kasari et al. 2008) and have been shown to increase over time (Gulsrud et al. 2014). Positive changes are also reported for skills that seem to depend upon the successful emergence of joint attention. These skills include joint engagement (e.g., Kasari et al. 2010), vocalizations (e.g., Jones et al. 2006), eye contact (e.g., Kim et al. 2008), turn-taking (e.g., Schertz and Odom 2007), focusing on faces (e.g., Schertz et al. 2007), play (e.g., Whalen et al. 2006), positive affect (e.g., Whalen et al.), emotion regulation (e.g., Gulsrud et al. 2010), empathetic response (Whalen et al. 2006), and friendship quality (Freeman et al. 2015). Targeted naturalistic developmental behavioral interventions (NDBIs) such as Joint Attention, Symbolic Play, Engagement, and Regulation (JASPER) have demonstrated improvements in IJA when delivered not only by clinic based interventionists but when mediated by parents (e.g., Kasari et al. 2014) or teachers (Chang et al. 2016). When delivered by paraprofessionals in the classroom, a targeted intervention was needed to improve IJA over standard practices alone, such as verbal behavior (Shire et al. 2016). IJA has also been shown to increase when a speech-generating device is combined with intervention, as those who have a SGD for the full JASPER intervention show significant IJA gains beyond those who have JASPER without the SGD (Almirall et al. 2016). Not all interventions targeting joint attention are equal as some have shown to have little effect on joint attention outcomes and responding to joint attention bids (Carter et al. 2011; Rogers et al. 2012).

See Also

- [IJA/RJA Skills](#)
- [RJA/IJA \(Initiating/Responding to Joint Attention\)](#)

References and Reading

Adamson, L. B., & Russell, C. L. (1999). Emotion regulation and the emergence of joint attention. In P. Rochat (Ed.), *Early social cognition: Understanding others in the first months of life* (pp. 281–297). Mahwah: Lawrence Erlbaum Associates.

- Almirall, D., DiStefano, C., Chang, Y., Shire, S., Kaiser, A., Nahum-Shani, I., et al. (2016). Longitudinal effects of adaptive interventions with a speech-generating device in minimally verbal children with ASD. *Journal of Clinical Child and Adolescent Psychology*, 45(4), 442–456.
- Bates, E. (1976). *Language and context*. New York: Academic.
- Bono, M. A., Daley, T., & Sigman, M. (2004). Relations among joint attention, amount of intervention, and language gain in autism. *Journal of Autism and Developmental Disorders*, 34, 495–505.
- Bruinsma, Y., Koegel, R. L., & Koegel, L. K. (2004). Joint attention and children with autism: A review of the literature. *Mental Retardation and Developmental Disabilities*, 10, 169–175.
- Bruner, J. (1983). *Child's talk: Learning to use language*. New York: W. W. Norton.
- Chang, Y., Shire, S., Shih, W., Gelfand, C., & Kasari, C. (2016). Preschool deployment of evidence-based social communication intervention: JASPER in the classroom. *Journal of Autism and Developmental Disorders*, 46(6), 2211–2223.
- Curcio, F. (1978). Sensorimotor functioning and communication in mute autistic children. *Journal of Autism and Childhood Schizophrenia*, 8, 281–292.
- Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., et al. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychopathology*, 40, 271–283.
- Freeman, S., Gulsrud, A., & Kasari, C. (2015). Brief report: Linking early joint attention and play abilities to later reports of friendships for children with ASD. *Journal of Autism and Developmental Disorders*, 45(7), 2259–2266.
- Gulsrud, A. C., Jahromi, L. B., & Kasari, C. (2010). The co-regulation of emotions between mothers and their children with ASD. *Journal of Autism and Developmental Disorders*, 40, 227–237.
- Gulsrud, A., Hellemann, G., Freeman, S., & Kasari, C. (2014). Two to ten years: Developmental trajectories of joint attention in children with ASD who received targeted social communication interventions. *Autism Research*, 7(2), 207–215.
- Jones, E. A., Carr, E. G., & Feeley, K. M. (2006). Multiple effects of joint attention intervention for children with autism. *Behavior Modification*, 30, 782–834.
- Kasari, C., Freeman, S., Paparella, T., Wong, C., Kwon, S., & Gulsrud, A. (2005). Early intervention on core deficits in ASD. *Clinical Neuropsychiatry*, 2, 380–388.
- Kasari, C., Paparella, T., Freeman, S., & Jahromi, L. B. (2008). Language outcome in ASD: Randomized comparison of joint attention and play interventions. *Journal of Consulting and Clinical Psychology*, 76, 125–137.
- Kasari, C., Gulsrud, A., Wong, C., Kwon, S., & Locke, J. (2010). Caregiver mediated joint engagement intervention for toddlers with autism. *Journal of Autism and Developmental Disorders*, 40, 1045–1056.

- Kasari, C., Lawton, K., Shih, W., Barker, T., Landa, R., Lord, C., et al. (2014). Caregiver-mediated intervention for low-resources preschoolers with autism: An RCT. *Pediatrics*, 134(1), e72–e79.
- Loveland, K. A., & Landry, S. H. (1986). Joint attention and language in autism and developmental language delay. *Journal of Autism and Developmental Disorders*, 16, 335–349.
- Morales, M., Mundy, P., Crowson, M. M., Neal, A. R., & Delgado, C. E. F. (2005). Individual differences in infant attention skills, joint attention, and emotion regulation behavior. *International Journal of Behavioral Development*, 29, 259–263.
- Mundy, P., & Crowson, M. (1997). Joint attention and early social communication: Implications for research on intervention with autism. *Journal of Autism and Developmental Disorders*, 27, 653–678.
- Mundy, P., & Hogan, A. (1994). Intersubjectivity, joint attention, and autistic developmental pathology. In D. Cicchetti & S. L. Toth (Eds.), *Disorders and dysfunctions of the self* (Rochester symposium on developmental psychopathology, Vol. 5, pp. 1–30). Rochester: University of Rochester Press.
- Mundy, P., Sigman, M., Ungerer, J., & Sherman, T. (1986). Defining the social deficits of ASD: The contribution of non-verbal communication measures. *Journal of Child Psychiatry*, 27, 657–669.
- Mundy, P., Sigman, M., Unger, J., & Sherman, T. (1987). Nonverbal communication and play correlates of language development in autistic children. *Journal of Autism and Developmental Disorders*, 17, 349–364.
- Mundy, P., Sigman, M., & Kasari, C. (1990). A longitudinal study of joint attention and language development in young autistic children. *Journal of Autism and Developmental Disabilities*, 20, 115–128.
- Mundy, P., Sullivan, L., & Mastergeorge, A. M. (2009). A parallel and distributed-processing model of joint attention, social cognition, and autism. *Autism Research*, 2, 2–21.
- National Autism Center. (2009). *National standards report*. Randolph: Author.
- Paparella, T., Goods, K., Freeman, S., & Kasari, C. (2011). The emergence of nonverbal joint attention and requesting skills in young children with autism. *Journal of Communication Disorders*, 44, 569–583.
- Rogers, S., Estes, A., Lord, C., Vismara, L., Winter, J., Fitzpatrick, A., et al. (2012). Effects of a brief Early Start Denver Model (ESDM)-based parent intervention on toddlers at risk for autism spectrum disorders: A randomized controlled trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(10), 1052–1065.
- Schertz, H. H., & Odom, S. L. (2007). Promoting joint attention in toddlers with autism: A parent-mediated developmental model. *Journal of Autism and Developmental Disabilities*, 37, 1562–1575.
- Shire, S., Chang, Y., Shih, W., Bracaglia, S., Kodjoe, M., & Kasari, C. (2016). Hybrid implementation model of community-partnered early intervention for toddlers with autism: A randomized trial. *Journal of Child Psychology and Psychiatry*.
- Whalen, C., & Schreibman, L. (2003). Joint attention training for children with ASD using behavior modification procedures. *Journal of Child Psychology and Psychiatry*, 44, 456–468.
- Whalen, C., Schreibman, L., & Ingersoll, B. (2006). The collateral effects of joint attention training on social initiations, positive affect, imitation, and spontaneous speech for young children with autism. *Journal of Autism and Developmental Disorders*, 36, 655–664.

Joubert Syndrome

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Synonyms

[Joubert-Boltshauser syndrome](#)

Short Description or Definition

Joubert syndrome (JS) is a rare neurodevelopmental disorder defined principally by abnormalities of the cerebellum and brain stem (Kroes et al. 2011; Maria et al. 1999; Parisi 2009). In addition to classic JS, a number of syndromes which all exhibit the “molar tooth sign” (MTS) on brain imaging are subsumed under the term Joubert Syndrome and Related Disorders (JSRD) and include COACH syndrome (with colobomas, hepatic fibrosis), Senior-Loken syndrome (with kidney disease), Dekaban-Arima syndrome, Varadi-Papp syndrome, and Malta syndrome.

Categorization

JSRD can be classified into six phenotypic subgroups: pure JS, JS with ocular defect, JS with renal defect, JS with oculorenal defects, JS with hepatic defect, and JS with orofaciodigital defects. The JSRD have been considered autosomal recessive based on recurrence risks around 25% and a 1:1 male to female ratio.

Epidemiology

The prevalence of JSRD is estimated at 1:100,000 in the United States (Parisi and Glass 2007). However, given the wide range of clinical features, this is likely an underestimate.

Natural History, Prognostic Factors, and Outcomes

Many of the clinical features of JS are apparent in infancy (Joubert et al. 1969; Bolthausen and Isler 1977). The prognosis for individuals with JS is variable depending on the extent of the abnormalities of the brain. Global developmental delay is frequent, and often there is a degree of moderate to severe mental retardation; however, some children have a mild form of JS with minimal motor impairment and good mental development. In infancy, prognosis is related to the severity of breathing problems, and afterward, prognosis depends mostly on the extent of renal and hepatic complications.

Clinical Expression and Pathophysiology

The most common clinical features of JSRD include hypotonia, significant intellectual impairment, impaired language development, ataxia, atypical breathing patterns, and oculomotor abnormalities. Irregular breathing patterns may include episodes of “panting” with short bursts of tachypnea followed by a short period of apnea. Individuals with JS frequently display abnormal eye movements with nystagmus,

accompanied by sideways head thrusting and eye blinking. Additional features may include dysmorphic facial features including a broad forehead, arched eyebrows, eyelid ptosis, wide-spaced eyes, and an open mouth due to facial hypotonia and ataxia. Affected children may also have skeletal abnormalities such as cone-shaped epiphyses and polydactyly, pituitary dysfunction, and other central nervous system anomalies.

Though cognitive impairment in JSRD is variable, most children display severe intellectual disabilities with global developmental delays (Farmer et al. 2006; Fennell et al. 1999). In one study, the average age of independent sitting was 19 months and the average age of walking was 4 years for those who developed these skills (Maria et al. 1999). Approximately one in five children also show maladaptive behaviors such as inattention, overactivity, social withdrawal, and atypical behaviors (e.g., self-injury, repetitive behaviors) (Farmer et al. 2006).

Reports of autism and autistic symptoms in JS emerged from clinical cases and small group studies in the early 1990s (Holroyd et al. 1991; Ozonoff et al. 1999). Parents reported their children failed to develop language and displayed autistic-like behaviors including lack of social interest, poor sensory integration, tactile defensiveness, repetitive or stereotyped motor mannerisms, and impaired executive functioning (e.g., perseveration, difficulties in shifting attention). In a study of 11 children with JS, Ozonoff et al. (1999) found that 3 met full DSM-IV criteria for autistic disorder and one qualified for a diagnosis of Pervasive Developmental Disorder – Not Otherwise Specified (4/11 = 36%). The children who did and did not meet autism diagnostic cutoffs showed similar levels of impairment in communication skills and repetitive behaviors, but differed in the number of impairments in social behaviors and total number of DSM-IV criteria met.

Subsequent studies, however, have provided evidence that JS is not a primary cause of classical autism. Takahashi et al. (2005) evaluated 31 JS families and found none of the children met the clinical cutoff for autism. They also compared genetic characteristics of the JS children with

those of children with autism and with Down syndrome (DS). The sib recurrence risk for these disorders was 32% in the JS families, 4% in the autism families, and 0% in DS families. Moreover, JS family histories revealed significantly less autism, alcoholism, cognitive, and language disorders in family members than was found in families ascertained through a child with autism. They concluded that JS and autism are genetically distinct disorders and JS is not a significant cause of the autism behavioral phenotype.

Analysis of oromotor functioning and communication in JS yielded further insight into how children with JS differ from those with ASD (Braddock et al. 2006). On standardized measures, those with JS showed mildly impaired receptive language and severely impaired expressive language, which is the opposite of what is often found in ASD. Also the children with JS demonstrated significant oromotor apraxia affecting the tongue, lip, and velum movements, which is not generally a feature of ASD. An interesting pattern emerged in the study of gesture use. In spite of their severe impairment in motor functioning, the children with JS produced gestures when communicating. Furthermore, the individuals whose speech was more impaired used a higher rate of gestures than those with more intelligible speech, which differs from what is seen in classic autism.

These detailed studies confirmed that JS and related disorders are not a significant cause of the autism phenotype. Nevertheless, comparing ASD and JS provides an excellent example of how complex neurodevelopmental disorders can overlap in their broad symptomatology and even their neuroanatomical findings. Early on, consideration of a possible causal link between JS and ASD was bolstered by the neuroanatomical work of Courchesne et al. (1988) indicating hypoplasia of the posterior cerebellar vermis in ASD and the recognition of frequent anatomic defects in cerebellar structure in ASD (Miles and Hillman 2000). Clarification of JS as a ciliopathy, however, showed that the similarities were superficial and not causal.

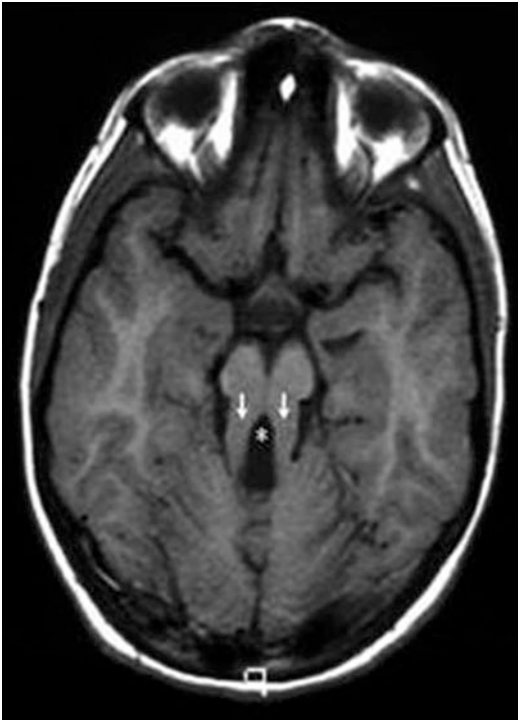
Previous research on the pleiotropic effects of major disruptors of brain development, such as in the ciliopathies, has shown that

neurodevelopmental disorders with disparate causes will often exhibit some symptom overlap. And, as suggested by Alvarez Retuerto et al. (2008), future studies may show that individuals heterozygous for one of the ciliary mutations may have an increased ASD susceptibility. Dissecting these interrelationships depends on careful phenotypic analyses and, as in the case of JS, is ultimately solved by identifying the underlying genetic etiologies of each. Above all, clinicians must be prudent when counseling families on either prognosis or recurrence unless the diagnosis is clear, and preferably the underlying genetic defect is proven.

Evaluation and Differential Diagnosis

The “molar tooth” sign on brain MRI is pathognomonic (Maria et al. 1997). Named because it resembles a cross section through a molar tooth, this brain abnormality is caused by cerebellar vermis hypoplasia, deepened interpeduncular fossa and thick, elongated cerebellar peduncles (see Fig. 1). Because of the multisystemic effects of JS, evaluation must include ophthalmologic, renal, hepatic, dysmorphology, and neurologic assessments (Parisi and Glass 2007).

Recent discovery of genes that cause JSRD has clarified the clinical heterogeneity noted in the syndromes defined by the molar tooth sign (see Parisi 2009 and Brancati et al. 2010, for recent reviews). Mutations in ten genes which cause impairments in the primary ciliary apparatus (*INPP5E* at 9q34.3, *TMEM216* at 11p12-q13.3, *AHI1* at 6q23.3, *NPHP1* at 2q13, *CEP290* at 12q21.32, *TMEM67/MKS3* at 8q21.1, *RPGRIP1L* at 16q12.2, *ARL13B* at 3q11.1, *CC2D2A* at 4p15.32, *OFDI* at Xp22.2) account for about half of causative mutations in JSRD (OMIM # 613037, 613,277, 608,894, 607,100, 610,142, 609,884, 610,937, 608,922, 612,013, 300,170). Each of these genes encodes for proteins of the primary cilium, making JS and related disorders members of the group of “ciliopathies.” Nine are autosomal recessive, one is X-linked recessive, and testing is clinically available. Primary cilia play key roles in the development and function in the brain, kidney, liver, and retinal cells



Joubert Syndrome, Fig. 1 Molar tooth sign. (Courtesy of Dr. Bernard Maria and the Joubert Foundation)

regulating tissue differentiation, establishment of body axis, growth, and intercellular signaling processes (see Lee and Gleeson 2011 for review of the role of cilia in the nervous system). Several of these ciliary proteins have been implicated in the modulation of signaling pathways, such as sonic hedgehog (Shh), Wingless (Wnt), and phosphatidylinositol (Bielas et al. 2009; Valente et al. 2010; Lancaster et al. 2009). Most cells in the brain including neurons, glial cells/astrocytes, and ependymal cells have primary cilia. Involvement of multiple genes that influence ciliary movement helps explain the clinical variability and heterogeneity described in JSRD. It has been shown that the same ciliary gene may cause many different phenotypes (allelic heterogeneity) and several different genes may be associated with the same clinical features (locus heterogeneity). Moreover, there is emerging evidence for oligogenic inheritance, where full penetrance of a phenotype requires mutations in more than one of the cilia genes.

Treatment

Presently, there is no cure for JS and treatment is symptomatic. Children with JS with abnormal breathing should be monitored; therapy includes stimulatory medications or mechanical support. Management of ophthalmologic problems may include surgery, corrective lenses, or therapy. Interventions for hypotonia and cognitive impairment may include early intervention programs for occupational, physical, and educational support. Annual screening is recommended for some of the complications associated with JS, such as liver, retinal, or kidney involvement that may become progressive over time. Individuals with JS also typically require behavioral and rehabilitative therapies and special education services to progress toward individualized goals in learning, communication, motor skills, adaptive behaviors, and socioemotional functioning. Finally, families may benefit from activities that support coping, including parent-to-parent mentoring (e.g., through the Joubert Syndrome Foundation), training in behavioral management strategies, assistance with resource identification, and effective care coordination.

See Also

- ▶ [Cerebellum](#)
- ▶ [Genetics](#)
- ▶ [Magnetic Resonance Imaging](#)

References and Reading

- Alvarez Retuerto, A. I., Cantor, R. M., Gleeson, J. G., Ustaszewska, A., Schackwitz, W. S., Pennacchio, L. A., et al. (2008). Association of common variants in the Joubert syndrome gene (AH11) with autism. *Human Molecular Genetics*, 17, 3887–3896.
- Bielas, S. L., Silhavy, J. L., Brancati, F., Kisseleva, M. V., Al Gazali, L., Sztriha, L., et al. (2009). Mutations in INPP5E, encoding inositol polyphosphate-5-phosphatase E, link phosphatidylinositol signaling to the ciliopathies. *Nature Genetics*, 41, 1032–1036.
- Bolthausen, E., & Isler, W. (1977). Joubert syndrome: episodic hyperpnea, abnormal eye movements, retardation and ataxia, associated with dysplasia of the cerebellar vermis. *Neuropädiatrie*, 8, 57–66.

- Braddock, B. A., Farmer, J. E., Deidrick, K. M., Iverson, J. M., & Maria, B. L. (2006). Oromotor and communication findings in Joubert syndrome: Further evidence of multisystem apraxia. *Journal of Child Neurology*, 21, 160–163.
- Brancati, F., Dallapiccola, B., & Valente, E. M. (2010). Joubert syndrome and related disorders. *Orphanet Journal of Rare Disorders*, 5(20), 20.
- Courchesne, E., Yeung-Courchesne, R., Press, G. A., Hesselink, J. R., & Jernigan, T. L. (1988). Hypoplasia of cerebellar vermal lobules VI and VII in autism. *New England Journal of Medicine*, 318, 1349–1354.
- Farmer, J. E., Deidrick, K. M., Gitten, J. C., Fennell, E. B., & Maria, B. L. (2006). Parenting stress and its relationship to the behavior of children with Joubert syndrome. *Journal of Child Neurology*, 21, 163–167.
- Fennell, E. B., Gitten, J. C., Dede, D. E., & Maria, B. L. (1999). Cognition, behavior, and development in Joubert syndrome. *Journal of Child Neurology*, 14, 592–596.
- GeneReviews: Joubert Syndrome. www.ncbi.nlm.nih.gov/books/NBK1325/
- Holroyd, S., Reiss, A. L., & Bryan, R. N. (1991). Autistic features in Joubert syndrome: A genetic disorder with agenesis of the cerebellar vermis [see comments]. *Biological Psychiatry*, 29, 287–294.
- Joubert Syndrome & Related Disorders Foundation. Websites. www.joubertfoundation.com
- Joubert, M., Eisenring, J. J., & Robb, J. P. (1969). Andermann, F. Familial agenesis of the cerebellar vermis. *Neurology*, 19, 813–825.
- Kroes, H. Y., Hochstenbach, R., Nievelstein, R. A., Den Hollander, A. I., Lugtenberg, D. T., Van Nieuwenhuizen, O., et al. (2011). Chromosomal abnormalities resembling Joubert syndrome: Two cases illustrating the diagnostic pitfalls. *Clinical Dysmorphology*, 20, 136–142.
- Lancaster, M. A., Louie, C. M., Silhavy, J. L., Sintasath, L., Decambre, M., Nigam, S. K., et al. (2009). Impaired Wnt-beta-catenin signaling disrupts adult renal homeostasis and leads to cystic kidney ciliopathy. *Nature Medicine*, 15, 1046–1054.
- Lee, J. E., & Gleeson, J. G. (2011). Cilia in the nervous system: Linking cilia function and neurodevelopmental disorders. *Current Opinion in Neurology*, 24, 98–105.
- Maria, B. L., Hoang, K. B., Tusa, R. J., Mancuso, A. A., Hamed, L. M., Quisling, R. G., et al. (1997). “Joubert syndrome” revisited: Key ocular motor signs with magnetic resonance imaging correlation. *Journal of Child Neurology*, 12, 423–430.
- Maria, B. L., Boltshauser, E., Palmer, S. C., & Tran, T. X. (1999). Clinical features and revised diagnostic criteria in Joubert syndrome. *Journal of Child Neurology*, 14, 583–590.
- Miles, J. H., & Hillman, R. E. (2000). Value of a clinical morphology examination in autism. *American Journal of Medical Genetics*, 91, 245–253.
- Online Mendelian Inheritance in Man, OMIM®. Johns Hopkins University, Baltimore. MIM Number: [613037, 613277, 608894, 607100, 610142, 609884, 610937, 608922, 612013, 300170]. World Wide Web. <http://omim.org/>
- Ozonoff, S., Williams, B. J., Gale, S., & Miller, J. N. (1999). Autism and autistic behavior in Joubert syndrome. *Journal of Child Neurology*, 14, 636–641.
- Parisi, M. A. (2009). Clinical and molecular features of Joubert syndrome and related disorders. *American Journal of Medical Genetics Part C. Seminars in Medical Genetics*, 151C, 326–340.
- Parisi, M. A., & Glass, I. A. (2007). Joubert syndrome. In *GeneReviews at GeneTests-GeneClinics: Medical genetics information resource [database online]*. Copyright. Seattle: University of Washington. Available at <http://www.genetests.org>.
- Takahashi, T. N., Farmer, J. E., Deidrick, K. K., Hsu, B. S., Miles, J. H., & Maria, B. L. (2005). Joubert syndrome is not a cause of classical autism. *American Journal of Medical Genetics Part A*, 132, 347–351.
- Valente, E. M., Logan, C. V., Mougou-Zerelli, S., Lee, J. H., Silhavy, J. L., Brancati, F., et al. (2010). Mutations in TMEM216 perturb ciliogenesis and cause Joubert, Meckel and related syndromes. *Nature Genetics*, 42, 619–625.

Joubert-Boltshauser Syndrome

► Joubert Syndrome

Judgments

► Autism in the Courtroom

Justice

► Autism in the Courtroom

Juvenile Subacute Necrotizing Encephalopathy

► Leigh Disease

K

KABC-II

► [Kaufman Assessment Battery for Children, Second Edition](#)

Kanner, Leo

Adam Feinstein
Autism Cymru and Looking Up, London, UK

Major Appointments (Institution, Location, Dates)

Appointed Assistant Physician at the State Hospital in Yankton County, South Dakota, in 1924

Selected by Adolf Meyer and Edward Park in 1930 to develop the first child psychiatry service in a pediatric hospital at Johns Hopkins Hospital, Baltimore

Appointed Associate Professor of Psychiatry at Johns Hopkins in 1933

Retired in 1959 and replaced as Chief of Child Psychiatry by Leon Eisenberg

Landmark Clinical, Scientific, and Professional Contributions

Dr. Leo Kanner's 1935 book, *Child Psychiatry*, won him international acclaim, but it was his 1943

paper, *Autistic disturbances of affective contact*, with its fine detailed clinical descriptions on what he called "early infantile autism" – which is still referenced by autism professionals around the world to this day, despite a number of deficiencies (in particular, Kanner's startling insistence that the 11 children in his 1943 study had "normal cognitive potentiality"). Kanner considered five features to be diagnostic: a profound lack of affective contact with other people; an anxiously obsessive desire for the preservation of sameness in the child's routines and environment; a fascination with objects mutism or a kind of language that does not seem intended for inter-personal communication; good cognitive potential shown in feats of memory or skills on performance tests; and onset of the condition from birth or before 30 months. For Kanner, the children had never been engaged with the social world. Kanner's 1943 paper certainly represented a seminal landmark in the identification and understanding of autistic behaviour. Nevertheless, Hans Asperger – whom Kanner never cited – had been working in the field from the early-1930s and published a paper on what he called "autistic psychopathy" in 1938. Apart from Kanner's puzzling reference to his patients' normal cognitive potentiality, he also did not seriously consider an organic etiology until Stella Chess's congenital rubella paper in 1971 – despite the fact that two of the 11 children in his 1943 study had epilepsy.

Short Biography

Leo Kanner (pronounced “Konner”) was born Chatskel Leib Kanner in the small Austrian village of Klekotow (called Klelotiw today) on June 13, 1894. As a young boy, he moved to Berlin in 1906 to live with his uncle. He initially wanted to be a writer – even while a medical student at the school of medicine at the Friedrich-Wilhelm University in Berlin – but no one would publish his poems. At the outbreak of the First World War, Kanner was recruited into the medical service of the Imperial and Royal Army of Austria and Hungary. In 1920, he became an assistant physician at the Charité Hospital in Berlin, working in the field of cardiology. He left Germany for the United States in January 1924 with his wife and young daughter. His first US post was that of assistant physician at the State Hospital in Yankton, South Dakota, publishing papers on general paralysis and syphilis. His first book, *Folklore of the Teeth*, was published in 1924. He joined the Henry Phipps Psychiatric Clinic at Johns Hopkins University in Baltimore in October 1928, working under the Swiss psychiatrist Adolf Meyer. Kanner won international renown with the publication of his book, *Child Psychiatry*, in 1935. His landmark paper, *Autistic Disturbances of Affective Contact*, was published in 1943. Although he retired as Chief of Child Psychiatry at Johns Hopkins in 1959 to be replaced by Leon Eisenberg, Kanner continued to publish important papers on autism until 1973. He and his wife, June Lewin, opened their home to many of the European refugees from the Nazis whom he had personally helped to reach American soil safely. Kanner died in Sykesville, Maryland, on April 3, 1981.

References and Reading

- Eisenberg, L. (1956). The autistic child in adolescence. *American Journal of Psychiatry*, 112(8), 607–612.
- Eisenberg, L. (1957). The fathers of autistic children. *American Journal of Orthopsychiatry*, 27, 715–724.
- Feinstein, A. (2010). *A history of autism: Conversations with the pioneers*. Oxford, UK: Wiley-Blackwell.
- Kanner, L. (1935). *Child psychiatry*. Springfield: C.C. Thomas Publishing.

- Kanner, L. (1941). *In defence of mothers*. Springfield: C.C. Thomas Publishing.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kanner, L. (1949). Problems of nosology and psychodynamics in early childhood autism. *American Journal of Orthopsychiatry*, 19(3), 416–426.
- Kanner, L. (1954). General concept of schizophrenia at different ages. *Proceedings of the Association for Research into Nervous and Mental Diseases*, 33, 451–453.
- Kanner, L. (1965). Infantile autism and the schizophrenias. *Behavioural Science*, 10(4), 412–420.
- Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of Autism and Childhood Schizophrenia*, 1(2), 119–145.
- Kanner, L. (1972). How far can autistic children go in matters of social adaptation? *Journal of Autism and Childhood Schizophrenia*, 2(1), 9–33.
- Kanner, L. (1973). *Childhood psychosis: Initial studies and new insights*. Washington, DC: Winston.
- Kanner, L., & Eisenberg, L. (1956). Early infantile autism, 1943–55. *American Journal of Orthopsychiatry*, 26(3), 556–566.

Kanner's Autism

- [Autistic Disorder](#)

Kapvay

- [Clonidine](#)

Karyotype

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Definition

A karyotype is an organized picture of an individual's entire set of chromosomes: 22 pairs of autosomes (nonsex chromosomes) and 1 pair of sex chromosomes (XX or XY). Karyotypes are used

in cytogenetics to identify abnormalities in the number or gross structure of an individual's chromosomes (Jorde et al. 2010). The picture that makes up the karyotype is taken during metaphase because the chromosomes are condensed and more easily visualized during this stage of mitosis. The chromosomes are stained with Giemsa stain, which produces a characteristic set of bands (G bands), and aids in the identification of alterations in chromosome structure, such as large deletions, duplications, or inversions (Jorde et al. 2010).

Recent guidelines recommend obtaining a karyotype as well as testing for fragile X syndrome in the evaluation of all individuals with autism spectrum disorders (ASD) (Lintas and Persico 2009; Schaefer and Mendelsohn 2008). The rationale for this recommendation is that there is evidence that approximately 2–5% of individuals with ASD have gross abnormalities in chromosome structure, which may be identified by karyotype (Reddy 2005; Schaefer and Mendelsohn 2008; Shen et al. 2010). Although identifying a chromosomal abnormality in a child with ASD in most cases does not alter the treatment plan, this knowledge is important for genetic counseling of families with regard to recurrence risk and may identify medical risks associated with certain chromosome abnormalities (Schaefer and Mendelsohn 2008).

At the same time, the information obtained from a karyotype is limited, because submicroscopic deletions or duplications, which accumulating evidence indicates are often associated with ASDs, are not detectable by karyotyping (Shen et al. 2010). Moreover, recent studies have found that a clinical microarray (CMA), or array comparative genomic hybridization, which can detect such submicroscopic variants, would increase the diagnostic yield in ASD to 10%, which does not include the 5% detection rate for karyotyping alone (Schaefer and Mendelsohn 2008). These findings have led to the consideration of CMA as part of the initial diagnostic evaluation of ASD (Shen et al. 2010). Although the findings from a karyotype or CMA do not dictate a specific therapeutic approach at this time, current research in the genetics of ASD aims to utilize knowledge

of the genes that are disrupted by alterations in the structure of chromosomes as an avenue to identify common biological pathways and novel therapeutic interventions.

See Also

► Chromosomal Abnormalities

References and Reading

- Jorde, L. B., Carey, J. C., & Bamshad, M. J. (2010). *Jorde: Medical genetics* (4th ed.). Philadelphia: Mosby.
- Lintas, C., & Persico, A. M. (2009). Autistic phenotypes and genetic testing: State-of-the-art for the clinical geneticist. *Journal of Medical Genetics*, 46(1), 1–8.
- Reddy, K. S. (2005). Cytogenetic abnormalities and fragile-X syndrome in autism spectrum disorder. *BMC Medical Genetics*, 6, 3.
- Schaefer, G. B., & Mendelsohn, N. J. (2008). Clinical genetics evaluation in identifying the etiology of autism spectrum disorders. *Genetics in Medicine*, 10(4), 301–305.
- Shen, Y., Dies, K. A., Holm, I. A., Bridgemohan, C., Sobeih, M. M., Caronna, E. B., et al. (2010). Clinical genetic testing for patients with autism spectrum disorders. *Pediatrics*, 125(4), e727–e735.

Kaufman Assessment Battery for Children, Second Edition

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Synonyms

KABC-II

Description

The KABC-II (Kaufman and Kaufman 2004a) is a standardized cognitive assessment suitable for children aged 3–18. It provides age-based standardized scores, age equivalents, and percentile

ranks. The time for administration varies from an estimated 25–70 min, depending on the age of the child, subscales administered, and individual differences of the participants. Overall, this intelligence assessment includes 18 core and supplemental subtests, which are grouped into four or five distinct scales, depending on the child's age and cognitive model selected. The test is based upon two theoretical models, including Luria's (1970) neuropsychological framework and the Cattell-Horn-Carroll hierarchically organized model (CHC; Carroll 1997).

The user can select the theoretical framework most suitable for the particular child being evaluated; the CHC framework is suggested for typically developing children from a mainstream language and cultural background, whereas the Luria framework tends to be more suited for participants with atypical cognitive functioning, such as Autism Spectrum Disorder (ASD) and other language or hearing impairment, as it excludes verbal ability. The same subtests are administered and the user can interpret results based upon either model. The test itself uses colored pictures and manipulatives to facilitate interest and promote ease of use with young or vulnerable children.

The CHC model is the newer approach, added to this revised edition of the assessment. It consists of Short-Term Memory (Gsm), Visual Processing (Gv), Learning Ability (Gl), Fluid Reasoning (Gf), and Crystallized Ability (Gc) subscales. The overall global score is termed the Fluid-Crystallized Index (FCI) and is developed from these Broad Ability measures.

The Luria model uses four of the five identical subscales to contribute to a global score (Mental Processing Index, MPI), but they are instead referred to as Sequential Processing (Gsm in CHC), Simultaneous Processing (Gv in CHC), Learning (Gl in CHC), and Planning (Gf in CHC). The Gc scale of the CHC model is termed Knowledge Scale using the Luria interpretation but does not contribute to the global score. The key difference between the two global scores is that the Luria approach (MPI) excludes acquired knowledge, whereas the CHC (FCI) includes measures of acquired knowledge and distinguishes between fluid and crystallized

intelligence. Only one of these global measures is calculated for each individual participant.

Each subscale consists of several further subtests. The Simultaneous Scale (Gv) includes Triangles, Face Recognition, Pattern Reasoning (ages 5–6), Block Counting, Story Completion (ages 5–6), Conceptual Thinking, Rover, and Gestalt Closure. The Sequential Scale (Gsm) tests Word Order, Number Recall, and Hand Movements. The Planning (Gf) scale focuses on Pattern Reasoning and Story Completion, only for children aged 7–18. The Learning Scale (Gl) includes Atlantis, Atlantis Delayed, Rebus, and Rebus Delayed. Finally, the Knowledge/Gc scale includes Riddles, Expressive Vocabulary, and Verbal Knowledge but is computed only for the CHC interpretation.

Subtest scores have a mean of 10 and a standard deviation of 3, and both global scores (FCI and MPI) have a mean Scale Index of 100 with a standard deviation of 15, keeping in line with the majority of cognitive instruments. Descriptive categories are also provided (Above Average, Average, Below Average, etc.). Scoring is either done by hand or with a separately purchased computerized system (ASSIST) which provides a score summary, scale profile, achievement comparison, and diagnostic information.

In addition to the FCI and MPI, the KABC-II includes a Nonverbal Scale that can be administered and responded to motorically. The KABC-II includes both Core and Expanded batteries, the latter of which do not contribute to standardized subscores but may be used clinically to follow up hypotheses and provide a comparison of delayed recall of learning to initial learning measures. The authors note that in order to provide a complete picture of a child's abilities across a range of domains, this test should be supplemented with other well-established measures.

Historical Background

The K-ABC was initially developed in 1983 by Alan S. Kaufman and Nadeen L. Kaufman, and was one of the first standardized measures of intelligence tests founded on real theories of cognitive processing. It was based on the Luria (1970)

model of sequential and simultaneous mental processing (see also Das et al. 1979) combined with left brain-right brain research on cerebral specialization (Sperry 1968). According to Luria, the brain is characterized by three functional systems, or “Blocks,” which promote arousal and attention (Block 1), analysis, coding, and storage of information (Block 2), and executive functioning and planning (Block 3). The integration of these functional systems is thought to support overall behavior; thus the K-ABC was created to assess the mental systems which arise from the integration of these blocks.

The initial edition evaluated children aged 2.5–12.5 years, and was touted as a culturally fair instrument, arguably unlike Binet and Weschler scales. This was due to the narrowing of the gap between K-ABC scores of White children and minority groups such as African American and Native American children; traditional cognitive assessments tended to show a prominent difference between such subgroups, but the K-ABC reduced this gap by roughly half a standard deviation. The novelty and importance of this test in particular also stemmed from its goal in evaluating underlying mental processes rather than the products of task performance. Although many supporters of the test praised its theoretical basis and clinical validity, other clinicians questioned whether the test was as internally or externally valid as claimed. In fact, a special issue of the *Journal of Special Education* in 1984 was devoted to these contrasting viewpoints.

Taking these issues into consideration, the authors decided to revise the test in order to strengthen theoretical foundations and enhance clinical use and validity in a wider range of participants, including preschoolers (Lichtenberger and Kaufman 2007). The new edition (KABC-II) has expanded to test an older age range of wider ability levels and cultural backgrounds and was released in 2004. It is theoretically based not only upon Luria’s model, but also the Cattell-Horn-Carroll hierarchically organized model (CHC; Carroll 1997). The CHC is a data-driven theory that merges two separate, but related, models which both focus upon the spectrum of broad human cognitive abilities. Raymond Cattell’s (1941) original two-pronged Gf-Gc theory,

which was subsequently revised by Horn (1965), was combined with John Carroll’s (1993) factor analysis on cognitive abilities. This resulted in a combined theory founded on decades of research on the concept of “g” (a general factor of intelligence) and broad cognitive abilities.

The KABC-II thus remains an intelligence test heavily grounded in empirical data, which sets it apart from other similar instruments. Other benefits from this revised edition include cultural fairness, as the materials contain scarce cultural content in order to equate individuals from diverse backgrounds. The test creators suggest that the KABC-II provides a veridical picture of a child’s abilities, as language difficulties and cultural differences do not contribute to the overall assessment.

Psychometric Data

The KABC-II Test Manual provides a detailed analysis of test development, factor structure, and test interpretation (Kaufman and Kaufman 2004a). The KABC-II has been normed on 3,025 participants ranging from age 3–18, representing a range of ethnicity, parental education, geographical location, and special education or gifted placement (see manual). The sample was stratified to reflect US population samples based upon 2001 Census data, thus providing an accurate distribution of children across the USA. The test manual provides extensive details on the validation study, in addition to reliability and consistency measures. The KABC-II was co-normed with the Kaufman Test of Educational Achievement, Comprehensive Form, Second Edition (KTEA-II; Kaufman and Kaufman 2004b).

Clinical validity has been established with several atypical groups, including children with ASD, ADHD, Learning Disabilities, and Gifted children. The KABC-II has good reliability for core and supplementary subtests, and recent research also reports good construct validity (Reynolds et al. 2007).

The median internal consistency reliability coefficient for ages 3–6 is 0.85, with a 0.87 coefficient for ages 7–18. Test-retest reliability coefficients of the global scales range from mid-0.80s to

mid-0.90s for the FCI and MPI, with retest stability increasing with age. Correlation analysis reveals that scale indexes maintain an appropriate amount of independence and thus tap into different processes, but also stem from a unitary underlying construct.

Clinical Uses

The KABC-II has been used with hearing impaired, learning disabled, and crosscultural populations. Although the test is administered in English, credit is given for answers presented in other languages (e.g., Spanish).

With respect to ASD, this test is ideal for non-verbal participants or those with limited language processing ability, due to option of delivering nonverbal test instructions in the form of modeling/pantomime and accepting motoric rather than verbal responses. This newer edition is thus arguably better suited (Bain and Gray 2008) for detecting a profile of processing strengths and weaknesses, which is essential for capturing the individual differences on the autistic spectrum. The information gleaned from the KABC-II assessment can help facilitate clinical, educational, and intervention planning.

This test may be administered by a variety of qualified users including Registered Psychologists (USA) and Chartered Psychologists recognized by the Health Professions Council (UK). Professionals with certification of in-house training by a Chartered Clinical Psychologist within certain government services are also permitted to use this test.

References and Reading

- Bain, S. K., & Gray, R. (2008). Test reviews: Kaufman, A. S., & Kaufman, N. L. (2004). Kaufman assessment battery for children: Second edition. Circle Pines, MN: AGS. *Journal of Psychoeducational Assessment*, 26(10), 92–101.
- Carroll, J. B. (1993). *Human cognitive abilities: A survey of factor-analytic studies*. Cambridge, MA: Cambridge University Press.
- Carroll, J. B. (1997). The three-stratum theory of cognitive abilities. In D. P. Flanagan, J. L. Genshaft, & P. L. Harrison (Eds.), *Contemporary intellectual assessment: Theories, tests and issues* (pp. 122–130). New York: Guilford.

- Cattell, R. B. (1941). Some theoretical issues in adult intelligence testing. *Psychological Bulletin*, 38, 592.
- Das, J. P., Kirby, J. R., & Jarman, R. F. (1979). *Simultaneous and successive cognitive processes*. New York: Academic.
- Horn, J. L. (1965). *Fluid and crystallized intelligence: A factor analytic study of the structure among primary mental abilities*. Ph.D. Thesis, University of Illinois.
- Kaufman, A. S., & Kaufman, N. L. (1983). *Kaufman assessment battery for children*. Circle Pines: American Guidance Service.
- Kaufman, A. S., & Kaufman, N. L. (2004a). *Kaufman assessment battery for children: Second edition (KABC-II)*. Circle Pines: American Guidance Service.
- Kaufman, A. S., & Kaufman, N. L. (2004b). *Kaufman test of educational achievement* (2nd ed.). Circle Pines: American Guidance Service.
- Lichtenberger, E. O., & Kaufman, A. S. (2007). The assessment of preschool children with the Kaufman Assessment Battery for Children – Second Edition (KABC-II). In B. A. Bracken & R. J. Nagle (Eds.), *Psychological assessment for preschool children*. New York: Routledge.
- Luria, A. R. (1970). The functional organization of the brain. *Scientific American*, 222, 66–78.
- Reynolds, M. R., Keith, T. Z., Fine, J. G., Fisher, M. E., & Low, J. A. (2007). Confirmatory factor structure of the Kaufman Assessment Battery for Children-Second Edition: Consistency with Cattell-Horn-Carroll Theory. *School Psychology Quarterly*, 22(4), 511–539.
- Sperry, R. W. (1968). Mental unity following surgical disconnection of the cerebral hemispheres. In *The Harvey lectures* (pp. 293–323). New York: Academic Press.

Kaufman Brief Intelligence Test

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Synonyms

KBIT-2

Description

The Kaufman Brief Intelligence Test, Second Edition (KBIT-2), is a reliable, valid, and norms-

based brief assessment of intelligence for individuals aged 4–90 years. The KBIT-2 provides a valid and reliable way of assessing both verbal and nonverbal intelligence in approximately 20 min. The KBIT is based on the Cattell-Horn-Carroll (CHC) hierarchical model of general intelligence, in which a number of Broad Abilities are broken down into Narrow Abilities. In addition to an overall measure of cognitive functioning, the KBIT assesses two Broad cognitive abilities: Crystallized (verbal) knowledge and Fluid (nonverbal) knowledge. A strength of the KBIT is that it can be quickly and easily administered to individuals across a very wide chronological age range. However, because of its brevity, it does not provide as thorough an assessment of intelligence as other more in-depth assessments, such as the authors' own Kaufman Assessment Battery for Children (KABC), which takes 25–55 min to administer. Given the limited number of scales on the KBIT-2 it is not possible to profile an individual's profile or pattern of relative strengths and weaknesses.

The KBIT-2 consists of three subtests, which are organized into two scales; all three subtests draw from the longer-form Kaufman Assessment Battery for Children, second edition (KABC).

The Crystallized (Verbal) Scale includes: (1) a Verbal Knowledge subtest, which is designed to assess an individual's general knowledge and receptive language skills, and (2) Riddles, which is designed to assess both receptive and expressive language.

The Fluid (Nonverbal) Scale consists of a Matrices subtest, which is designed to assess an individual's nonverbal, abstract reasoning abilities.

All of the materials, or stimuli, that are presented to the examinee are in color.

The Verbal Knowledge subtest, containing 60 items, is a receptive measure of the child's store of English-language vocabulary and of general information.

The subtest is composed of a series of multiple choice questions that require respondents to point to or say the letter of the correct response.

There are no teaching items; instructions are provided in English only.

The Riddles subtest is composed of 48 items, in which a series of verbal riddles are presented and the examinee is asked to either point to the picture or say the word that provides the answer to the riddle. This test measures an individual's verbal comprehension, verbal reasoning, and word retrieval.

The Matrices subtest is composed of 46 items including several types of visual stimuli (e.g., people and objects as well as designs and symbols) and requires individuals to understand relationships between the stimuli. All of these items are multiple choice, requiring individuals to point to or say the letter corresponding to the correct response. Sample and teaching items are provided, as are Spanish language instructions.

All items are administered from a testing easel, and correct responses and examples of typical responses that need to be queried are printed on the easel facing the examiner.

A major limitation of the KBIT-2 Crystallized (verbal) scale is that it must be administered in English. However, if an examinee provides a correct response in another language they are given credit, and correct Spanish language responses are included on the record form for the Riddles subtest.

Historical Background

The KBIT-2 is a revision of the original version of the Kaufman Brief Intelligence Test. The original KBIT was published in 1990 and also included both verbal and nonverbal intelligence measures, although the verbal subtests were revised in the second edition; the first edition included only one subtest, Vocabulary, as opposed to the KBIT-2's Verbal Knowledge and Riddles subtests.

Psychometric Data

The KBIT-2 generates three standard scores, with means of 100 and standard deviations of 15: an overall measure of intelligence (the KBIT IQ composite), as well as a Crystallized and a Fluid standard score. The examiner records scores for

each tasks and converts the subtests to standard scores using the manual, which also provides tables for comparing individuals' performance on the different scales to determine whether the difference is significant. The KBIT-2 provides age-based norms that were based on a national standardization sample selected to match the US census data. Standard scores can range from 40 to 160 for most ages, with the exception of the very low and high ends of the age range.

Standardization occurred between May 2002 and May 2003 across 34 states and the District of Columbia, and the sample included 2120 children and adults. Random sampling methods were used to select individuals from a larger pool of potential examinees, matching the US Census data.

The KBIT-2 has demonstrated strong reliability and validity, comparable to the Wechsler Intelligence Scales for Children – fourth edition (WISC-IV). Split-half reliability coefficients are high, in the range of .80 to mid-.90s. Mean split-half reliability scores across all age groups were .91 for the Verbal Scale and .93 for the Composite IQ. The only statistically significant sex difference observed was for children of ages 7–12 years, favoring females for 2.4 points. Test-retest reliability coefficients ranged from .88 to .93. Across ages, peak performances for Verbal Knowledge and Riddles were in the 26–35-year-old range, with decreases observed at higher ages. The measure also demonstrates concurrent validity with other assessments of cognitive functioning, including the Wechsler Abbreviated Scale of Intelligence (WASI), where correlations range from .81 to .90 for the composite scores.

While the manual includes data from seven groups of participants with special needs, including individuals with intellectual disability, no data from individuals with Autism Spectrum Disorders is presented.

The KBIT test was normed as a brief test, a strength relative to the short forms of other intelligence assessments that were extrapolated from longer, more comprehensive tests. The KBIT-2 was also co-normed with the Kaufman Test of Educational Achievement – Brief Form for ages 26–90,

which can provide a brief assessment of educational achievement concurrent with the KBIT.

Clinical Uses

Given the brevity of the assessment, the KBIT is a flexible and user-friendly test. Its primary use is to obtain a quick estimate of the intellectual ability of children and adults. However, given its brief nature, it can also be used as a screener to identify children who may require a more comprehensive evaluation. It is relatively simple to administer and does not require a manual. The ease of assessment lends the KBIT to brief administrations, including reevaluations, across a variety of contexts (e.g., clinic, school).

However, also because it is brief, intelligence scores should be held tentatively and should *not* be the basis of major clinical decisions about diagnosis or placement; such decisions require more comprehensive assessments of an individual's cognitive testing. Moreover, although KBIT scores correlate with overall intelligence scores, because of the brevity of the test, it does not assess the full spectrum of cognitive functioning (e.g., there are no rapid responses required on the KBIT).

Because of its brevity and ease of administration, the KBIT and KBIT-2 have been used often as a brief measure of cognitive functioning in research studies of individuals with ASD frequently as a way of differentiating “high functioning” individuals with autism (defined as a verbal or composite IQ score on the KBIT-2 greater than 70). However, extensive research on the use of this measure among individuals on the spectrum has not been conducted. A potential drawback of the KBIT as a measure is its reliance on expressive language on the Riddles subtest.

See Also

- ▶ [Maladaptive Behavior](#)
- ▶ [Vineland Adaptive Behavior Scales \(VABS\)](#)
- ▶ [Wechsler Preschool and Primary Scale of Intelligence](#)

References and Reading

- Bain, S. K., & Jaspers, K. E. (2010). Test review: Review of Kaufman brief intelligence test, second edition. *Journal of Psychoeducational Assessment*, 28, 167–174.
- Gantman, A., Kapp, S. K., Orenski, K., & Laugeson, E. A. (2012). Social skills training for young adults with high-functioning Autism Spectrum Disorders: A randomized controlled pilot study. *Journal of Autism and Developmental Disorders*, 42(6), 1094–1103.
- Kaufman, A. S., & Kaufman, N. L. (1990). *Kaufman brief intelligence test manual*. Circle Pines: American Guidance Service.
- Kaufman, J. C., & Kaufman, A. S. (2001). Time for changing of the guard: A farewell to short forms of intelligence. *Journal of Psychoeducational Assessment*, 19, 245–267.
- Laugeson, E. A., Frankel, F., Mogil, C., & Dillon, A. R. (2009). Parent-assisted social skills training to improve friendship in teens with Autism Spectrum Disorders. *The Journal of Autism and Developmental Disorders*, 39(4), 596–606.

Kazakhstan and Autism

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Historical Background

Autism is a new diagnostic concept for Kazakhstan. There is no accurate data on the number of autistic children in Kazakhstan. In accordance with data of the State Republican Psychological-Medical-Pedagogical Committee as of 2017, the approximate number of autistic children in Kazakhstan is around 3000. In 2003 the number of identified autistic children was 77.

Kazakhstan accepted International Classification of Disease (ICD) in January 2010, so medical services for autistic children in Kazakhstan are based on ICD classification. The main principles of medical services in Kazakhstan are stated in the Code “Health and the health system” of the Republic of Kazakhstan. By the Code, autism is

under category of psychiatric disorder, so child psychiatrists are the only diagnosticians. Parents of autistic children have many fears including lack of access to any educational system, including specialized schools. In case of severe behavioral problems, the child would be put into a child psychiatric dispensary.

At present there is no program of autism screening and diagnosis by state. Extremely limited organizations use evidence-based screening and diagnosis methods for treatment purposes.

By the Code, autistic children in Kazakhstan are eligible for state social allowance till 18 years old. They are considered as disabled children and usually put into specialized state schools, for example, specialized schools for children with speech/hearing/mental/psychological/problems, etc. At present there is no public school inclusion of autistic children into educational system of Kazakhstan.

Until 2017 there was no official state recognition of autism. Limited number of NGOs and foundations started autism support activities and promoted Autism Roadmap, which is finally accepted by the Ministry of Health of the Republic of Kazakhstan. This is the first official sign of autism recognition in Kazakhstan.

Limited numbers of NGOs started autism support activities in 2014 organizing public awareness activities, press conferences, and informational workshops for parents, providing sport activities for autistic children, etc. Some of them promote inclusive education. The total number of NGOs and foundations supporting autistic children is about ten. Only one specialized state department within the National Child Rehabilitation Center serves autistic children in Kazakhstan.

Among NGOs and foundations, four “Asyl Miras” autism centers established by Bulat Utemuratov Foundation in 2015 are the only organizations which use ASD screening and diagnosis methods such as M-Chat-R and ADOS-2; provide learning and language programs using ABA therapy and JASPER program of University of California in LA, USA; and collect and analyze intervention data.

There are no state programs to support autistic children, except one small pilot project of the Ministry of Education, which allows integration of very limited number of ASD children into state education system in Astana city. The project is piloted in five state schools in Astana city.

There is a hope that if accepted in 2017, Kazakhstan Autism Roadmap in 2017 will accelerate autism support activities and promote destigmatization in Kazakhstan.

Legal Issues, Mandates for Service

In accordance with article #119 of the Code “Health and the health system” of the Republic of Kazakhstan, diagnosing of psychiatric disorders (diseases) is made by a psychiatrist in the following classification: childhood autism, atypical autism, Rett’s syndrome, childhood disintegrative disorder, overactive disorder associated with mental retardation and stereotyped movements, and Asperger’s syndrome. The term autism spectrum disorder (ASD) is used only by NGOs and foundations serving autistic children. As noted previously only child psychiatrists make a diagnosis, and children with autism have eligibility for state social allowance until 18 years of age.

There is a lack of field specialists, who would provide interventions. Most of ASD children support activities or programs run by limited number of NGOs established by parents of ASD children and by few foundations.

Overview of Current Treatments and Centers

The Psychoneurological Department of the National Child Rehabilitation Center (the only one-state institution) provides services to autistic children in the capital Astana. In 2016 the Department served 122 autistic children. The Department uses M-Chat and ADOS and provides services: psychologist, speech therapist, nutrition, sensory integration, and music therapy. The major service provider in Kazakhstan is Bulat Utemuratov Foundation. The Foundation

established four “Asyl Miras” autism centers in Kazakhstan to support autistic children and their parents. Autism centers implement “Autism. World is One for All” program to support 0–15-year-old, autistic children in four major cities in Kazakhstan (Astana, Almaty, Kyzylorda, Ust-Kamenogorsk). As of September 2017, four autism centers served 2981 ASD children.

“Asyl Miras” autism centers use M-Chat and ADOS-2 for screening and diagnosis and ABLLS to conduct functional analysis. There are seven intervention programs provided for ASD children:

1. Early Intervention Program for children aged 0–3 years old
2. Language Program
3. Learning Program
4. JASPER Program
5. Social Skills Development Program
6. Leaving Skills Development Program
7. Intense course for parents

The public foundation “Ashyk Alem” promotes inclusive education in Almaty and Astana cities. “Ashyk Alem” uses ABLLS for making functional analysis. It trained limited number of tutors. Other foundations providing some supports/services include the Corporate Foundation and Orda (for sports activities) and DOM (information supports for parents).

Overview of Research Directions

Research at present is extremely limited and mostly is conducted by faculty members of Nazarbayev University (<https://nu.edu.kz>). Ongoing projects include development of a national registry, projects on providing information to parents, and some aspects of genetics.

Overview of Training

Training opportunities are limited and most training is provided by external trainers. These provide some training for clinical staff. For example, Bulat Utemuratov Foundation provided more than

30 trainings for its 120 clinical therapists during 2016–2017, inviting trainers from the USA and Russia. Trained therapists of Asyl Miras autism centers provided more than ten trainings in 2016–2017 for specialists of regional departments of the State Republican Psychological-Medical-Pedagogical Committee on the basics of ASD, using M-Chat and ADOS-2. There are two private commercial training companies in Almaty, which provide fee-based basic ABA trainings for therapist. These companies invite Russian-speaking trainers from Russia for basic ABA trainings. No one in Kazakhstan currently has BCBA certification.

No trainings are available for teachers and healthcare professionals.

Social Policy and Current Controversies

There is limited public awareness of autism. Some of the activities of NGOs and foundations are attempting to address this. Social infrastructure is not adapted for ASD children needs and expectations. As noted about inclusive education opportunities are limited.

Code of the Republic of Kazakhstan about health of people and healthcare system. www.zakon.kz/148589-kodeks-o-zdorovie.html

See Also

► [Russia and Autism](#)

References and Reading

www.asylmiras.org
www.autism.kz
www.bolashakcharity.kz
www.astanaautism.kz
www.detdom.kz
www.balbulak.kz

KBIT-2

► [Kaufman Brief Intelligence Test](#)

Keppra

► [Levetiracetam: Definition](#)

KIAA1650

► [SHANK 3](#)

Kinesthetic Sense

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Definition

This is the sensory experience of movement of a body segment in relation to other body segments and is differentiated from proprioception which refers to the position of a body segment in relation to other body segments. The dynamic sensory experience of reaching away from the body toward a cup or reaching toward the body to button a shirt provides examples of kinesthesia. Although frequently linked with proprioception, kinesthesia is the considered mediated by the dynamic receptors within the muscle spindle, and the proprioception or position sense is mediated through receptors in the ligament and joints. Functionally, when an individual has difficulties with processing kinesthetic sensory information, the position sense is often disrupted. Many functional daily living tasks are dependent upon the accuracy in the speed, force, and direction of movement. Handwriting is one functional task that is particularly mediated by the sense of moving the hand to form letters.

References and Reading

- Gardner, E. P., Martin, J. H., & Jessel, T. M. (2000). The bodily senses. In E. R. Kandel, J. H. Schwartz, & T. M. Jessel (Eds.), *Principles of neural science* (4th ed., pp. 430–450). New York: McGraw-Hill.
- Kushki, A., Chau, T., & Anagnostou, E. (2011). Handwriting difficulties in children with autism spectrum disorders: A scoping review. *Journal of Autism and Developmental Disorders*, 41, 1706–1716.

Klinefelter Syndrome

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Synonyms

KS

Short Description

Klinefelter syndrome (KS) was first described by Dr. Harry Klinefelter, an endocrinologist, in 1942. He identified a group of adults with infertility, hypogonadism, gynecomastia, and increased gonadotrophin levels (Klinefelter et al. 1942). In 1959, these symptoms were linked to the extra X chromosome in these men (Jacobs and Strong 1959). The majority, about 80% of the KS population, carry an additional X chromosome (47, XXY), while the other cases have higher grade aneuploidies (e.g., 48, XXXY, 48, XXYY), mosaic forms (a combination of normal and abnormal cell lines 46, XY/47, XXY), or structurally abnormal X chromosomes (Lanfranco et al. 2004). KS has a highly variable phenotype. Besides physical features, such as described by Klinefelter, KS can manifest itself with a variety of behavioral characteristics, including developmental language problems and language-based learning disabilities, executive and attentional dysfunction, and motor developmental delays (Geschwind et al. 2000). Social function is known to be a vulnerable area of development in

KS (e.g., Mandoki and Hoffman 1991), while intellectual abilities are generally within the average range (Boada et al. 2009).

Categorization

Klinefelter syndrome is a de novo occurring genetic disorder.

Epidemiology

Klinefelter syndrome is the most frequent sex chromosomal disorder in the male, with a prevalence of 1: 600–700 in newborn boys (Bojesen et al. 2003). The extra X chromosome(s) in Klinefelter syndrome result(s) predominantly from a nondisjunction during meiosis; about halve of the cases are paternally derived, hence the other halve maternally. Very seldom Klinefelter (mosaic type) results from an error at an early mitotic division in the very early developmental stage of the embryo. Many boys and men with KS remain undiagnosed. Abramsky and Chapple (1997) calculated that about 10% of expected cases were identified prenatally (during prenatal screening) and 26% were diagnosed in childhood or adult life because of hypogonadism, gynecomastia, or infertility, leaving 64% undiagnosed. A large Danish national registry study confirms that KS is widely underdiagnosed, with less than 10% of the expected diagnoses made before puberty (Bojesen et al. 2003) and only about 24% identified in adulthood.

Natural History, Prognostic Factors, Outcomes

The impact of sex chromosome trisomies on brain function is typically mild compared to the impact of duplication of an autosome, which is typically lethal or associated to major cognitive impairments. When additional X chromosomes are present, one is predominantly inactivated. As the number of X chromosomes increases, the phenotypic severity increases as well.

A large part of the Klinefelter population goes undetected, probably because of relatively mild problems or because clinicians do not associate physical, behavioral, or emotional problems during development or in adulthood to this specific condition. The presence of an extra X chromosome often goes undetected in childhood, unless chromosome studies are undertaken to establish the etiology of educational or behavioral difficulties. Especially the mosaic type, with a milder phenotype, can go undetected.

The diagnosis is commonly made in adolescence or adulthood; gynecomastia, hypogonadism, or infertility may lead to the detection of the extra X chromosome. Klinefelter syndrome accounts for 3% of male infertility (Abramsky and Chapple 1997). Behavioral and emotional problems that can be associated with Klinefelter are not very specific.

Based on birth cohort studies with prospective design and long-term follow-up, it is concluded that there is an increased risk for medical, psychological, social, and educational difficulties in KS. Cognitive deficits are generally mild, and mixed results are reported with respect to the risk for psychopathology. Moderate to severe problems can be found in reading, spelling, and writing. Males with KS tend to leave school earlier and achieve a lower educational level than unaffected males.

Clinical Expression and Pathophysiology

Physical Features and Medical Risk

The phenotype of Klinefelter syndrome is highly variable. In KS, there are no facial characteristics. Because most boys with KS appear similar to boys with normal karyotypes, the disorder typically is identified in adolescence or adulthood. The most overt phenotypic features of Klinefelter are caused by testosterone deficiency and directly or indirectly by unsuppressed follicle-stimulating and luteinizing hormones. Affected men typically present with a tall stature, infertility, small testes, decreased facial hair, gynecomastia (in about 50%), decreased pubic hair, and a small penis. Because of their long legs, men with Klinefelter

syndrome are often taller than predicted based on parental height. Persistent androgen deficiency in adulthood may result in loss of libido, decreased muscle bulk and tone, decreased bone mineral density, a propensity for thromboembolism, and an increased risk for diabetic and cardiovascular complications (Wattendorf and Muenke 2005). However, the clinical picture of XXY males may range from severe signs of androgen deficiency, or even a lack of spontaneous puberty, to normally virilized males who only consult a doctor because of their infertility. In childhood, ascertainment is difficult, because the effects of hypogonadism (small external genitalia and firm testes) may be subtle or not present at all.

Psychopathology

In addition to the physical findings, children, adolescents, and adults with KS may present with a variety of developmental and behavioral features that make up the behavioral phenotype of KS. Based on birth cohort studies it is concluded that KS is associated with an increased risk for verbal cognition and language impairments, reading disabilities as well as behavioral and cognitive difficulties such as attention problems, executive function problems, shyness, social withdrawal, and social difficulties. The cognitive phenotype of KS does not reflect a general decline in intellectual abilities, but, rather, deficits in specific domains of cognition, namely, language and frontal-executive functions.

Systematic review of neurocognitive outcome in patients ascertained at birth by newborn screening showed overall mean cognitive abilities on standardized tests of intelligence to be in the average range; however, mean verbal cognitive scores were approximately 10 points lower than performance scores and significantly decreased in relation to typical developing children. Within the Klinefelter population, intelligence scores have a normal distribution, but the mean score is shifted slightly to the left of the population mean, at low average and borderline range (e.g., Tartaglia et al. 2010).

Specific aspects of cognitive and behavioral outcome are studied in samples of KS that were recruited in clinics or support groups,

ascertainment bias is a weakness of those studies and the results do not necessarily represent the entire spectrum of outcomes seen in KS. However, these studies draw attention to the developmental risk of boys and men with KS.

Language developmental problems are typically first observed as delay in early expressive language and speech milestones. Difficulty in articulation is found, as well as problems in word-finding and phonemic processing. Academic difficulties, related to language abilities, are found in 70–80% of KS boys. Moderate to severe problems can be found in reading, spelling, and writing. Fifty to seventy-five percent of the children meet the criteria of dyslexia (e.g., Bender et al. 1993); 92% have difficulty in learning to read (Geschwind et al. 1998). Language-related academic problems may result in problems in arithmetic and other areas of academic function, probably due to difficulty in problem-solving skills and integration of knowledge.

Especially difficulty with auditory processing and verbal memory problems are supposed to underlie the academic problems (Graham et al. 1988). Difficulty with decoding of words and sentences seem to be the problem in understanding written or spoken language. Studies report a large part of the KS population to have difficulty in verbal learning, word-finding, and verbal fluency. They also show average or above-average scores on tasks for visual-spatial information processing and visual memory. Most boys with Klinefelter syndrome show slow information processing, like most children with dyslexia, in both visual and verbal modalities.

Not much systematic research is done in the area of frontal executive functioning in longitudinal studies of KS. In addition to anecdotal evidence of hyperactivity and distractibility, several studies suggest impairments in concentration, short-term memory, and problem-solving abilities (e.g., Geschwind et al. 2000). Difficulties in attention regulation in boys with KS are reported, as well as difficulties in inhibition, although not always replicated. Inhibition problems and problems in mental flexibility appeared to be related to schizotypal traits in XXY in a study by Van Rijn et al. (2009).

Developmental motor delay is often anecdotally reported. Sports do not seem to be very popular in boys and men with KS. In the Denver birth cohort study, 8 of the 14 boys with KS had delayed independent walking. In a group of more than 30 adolescents with XXY, neurological evaluation showed mirror movements in 40% of the population and a high incidence of positional tremor. Mirror movements suggest a disruption in interhemispheric sensory-motor integration at the level of the supplementary motor area of the anterior corpus collosum, through which right and left motor regions communicate. Fine motor integration, bilateral coordination, visual motor control, and response speed were decreased in 93 predominantly prenatally diagnosed boys with KS (Ross et al. 2009).

Generally, boys and men with KS are described as introverted, shy, with high levels of anxiety and social withdrawal (e.g., Mandoki and Hoffman 1991). This may affect self-esteem and daily functioning. Several studies indicate a high risk for psychosocial problems, psychosis, and bipolar disorder in KS (e.g., Bender et al. 1999). KS is associated with the risk for autistic features and with high levels of schizotypal traits and schizophrenia symptoms (Bruining et al. 2009; van Rijn et al. 2008). Social difficulties and vulnerability for autistic traits are found in adults with KS. Men with KS show increased levels of distress during social interactions and difficulty in expressing emotions to others and in initiating contact with others. The level of autistic traits on the Autism Spectrum Quotient is higher in this group compared to the general population. Social cognition appears to be at risk in KS. Men with KS show difficulty in perception of facial emotions. They experience increased levels of emotional arousal, yet they are less able to identify and verbalize their own emotions. In addition, KS men show difficulty in discriminating emotions in verbal content of speech and in tone of voice. In a cohort of 51 boys with KS of which 50% were identified by prenatal screening, language disorder was found to be most prevalent (65%), followed by attention-deficit disorders (ADHD) and autism spectrum disorders (27%). Behavioral

impairment was most evident in cases classified with psychotic disorder (12%) (Bruining et al. 2009).

Pathophysiology

Language is distributed in and around the perisylvian cortex of the left hemisphere in most people. Developmental language disorders, such as dyslexia, have been associated with functional and anatomical patterns of anomalous dominance. Several findings suggest left hemisphere dysfunction in KS. The high incidence of fine motor mirroring in KS supports the notion that interhemispheric sensory-motor integration is anomalous. The problems in rapid auditory processing in KS are supposed to reflect suboptimal left-hemisphere function. Right ear dominance was less present in KS in dichotic listening to verbal material (Netley and Rovet 1984). These findings suggest left hemisphere dysfunction in KS.

Sex steroids have organizational effects on the central nervous system during development. Especially during fetal development, the influence of sex steroids on cognition is large. One therefore may hypothesize that differences in cognitive abilities in KS may result, in part, from differences in gonadal steroid levels during critical periods in fetal development. In KS, however, androgen deficiency is usually not present until late in adolescence when incomplete secondary sexual development may be seen in half of the KS men (Mandoki and Hoffman 1991). Serum testosterone levels at age 16–18 are within the normal range for 50% of the KS boys. There is no evidence so far for low or high intrauterine testosterone in cord blood, nor is there any attenuation of the normal postnatal rise in serum testosterone in KS infants (Ratcliffe et al. 1994). So far, no systematic research has been done to assess the influence of testosterone on cognitive development in KS and it is not likely that testosterone level can explain the total range of cognitive and behavioral differences in KS (Geschwind et al. 2000).

Cognitive and behavioral difficulties in KS might be related to a gene dosage effect based on the presence of an extra X chromosome. Expression of extra copies of genes residing on the

X chromosome leads to so-called gene dosage effects. There is evidence that a gene on the X chromosome contributes to cerebral laterality and language laterality in the normal population. Imprinting effects have been suggested to be important in social cognition. Parent-of-origin effects on social behavior seem to be associated to specific effects on autistic and schizotypal traits in one study so far (Bruining et al. 2010). Further research is necessary.

Structural and Functional Imaging Findings

Studies so far report discrepant findings from structural and functional MRI studies in KS, due to differences in study design, cohort characteristics, and neuroimaging analyses methods.

MRI results of a prospective follow-up study in a small group of KS men showed significantly smaller whole brain volume and increased volume of the ventricles (Warwick et al. 1999). Reduced total brain volume has been replicated in several studies, though other investigators have found no differences. In the Denver cohort study, the overall brain volumes of 10 KS men did not differ from controls; however, amygdala volume appeared to be smaller. Other studies also reported evidence for specific regional changes, finding small amygdala, hippocampi, STG (superior temporal gyrus), and caudate nuclei. Several studies suggest that frontal and temporal lobe volumes may be smaller in KS than in typical males. Some studies suggest that the lobar differences might be more specific to the left side, specifically in the temporal lobe, which would correspond with the specific phenotype that includes language and executive dysfunction. Functional imaging studies so far are very scarce and show a relative increase in right-sided cerebral activity with a resulting leftward asymmetry, particularly in temporo-parietal regions. KS men appear to display decreased activation in the amygdala and fusiform gyrus when judging faces (van Rijn et al. 2008, 2011).

In summary, the presence of an additional X chromosome in males does appear to have effects on both brain structure and function, in at least a subset of individuals. Decreased lateralization is found compared with controls.

Evaluation and Differential Diagnosis

Klinefelter syndrome has a highly variable phenotype and presents with mostly nonspecific behavioral problems in childhood. Developmental language problems and language-based learning disabilities (especially dyslexia) may be present as well as attention problems, executive function problems and motor developmental delays. Developmental problems, including social problems in KS, may meet the diagnostic criteria for language disorder, attention deficit disorder, autistic spectrum disorders, or other disorders like psychotic disorders. In adolescence and adulthood, physical features like hypogonadism and infertility may lead to the diagnosis of KS. KS should be specifically differentiated from dyslexia, developmental language disorders, autism spectrum disorders, and psychotic disorders.

Treatment

A suspected diagnosis can be based on the combination of typical clinical findings. The most important physical features are very low testicular volume and firm consistency of the testes, although these may not be present in all cases. Testicular volume seems to be the most sensitive indicator. In a cohort of 309 suspected cases based on the above characteristics, diagnosis could be confirmed in 28% by chromosome analysis (Lanfranco et al. 2004). In childhood, physical features hardly ever lead to the diagnosis. Physical differences in males with Klinefelter often are evident after the onset of puberty. Late or incomplete puberty should prompt further examination. The most important behavioral features appear to be in the domain of language developmental delay, specific language-based learning disabilities, attention and executive function deficits, and social adaptation problems. Clinical suspicion warrants diagnostic investigation to ensure appropriate surveillance for testosterone deficit. Chromosome analysis is necessary for the diagnosis of Klinefelter.

As soon as Klinefelter is detected, a comprehensive neurodevelopmental evaluation is recommended. Children with KS should have a

neuropsychological evaluation in early grade school to identify any symptoms of language problems, learning disabilities, attention deficits, or executive function problems. Concerns about social skills should lead to evaluation by a psychologist with sufficient experience to consider the influences of language deficits and neurocognitive problems on social development and who can perform a diagnostic assessment for autism spectrum disorders, if needed. In childhood and adolescence, a multidisciplinary developmental examination may help determine appropriate treatments such as physical therapy, speech therapy, and stimulation programs.

The prepubertal testosterone deficits results in impaired bone mineral density and skeletal muscle development. Androgen therapy should be started when there is laboratory evidence of a testosterone deficit, at about the age of 12–14. When testosterone serum concentrations in individuals with KS are low, lifelong substitution therapy is indicated and should be started in puberty to avoid symptoms and sequelae of androgen deficiency. Testosterone replacement results in increased masculinity, strength, libido, bone mineral density, and body hair. It has a positive effect on mood and behavior, improves self-esteem and reduces fatigue and irritability.

It is critical that boys with KS are provided with appropriate educational interventions that target their learning challenges in school. Psychoeducation can improve understanding of developmental and behavioral vulnerabilities. In individual cases, psychotherapy or social training can be indicated.

See Also

► Chromosomal Abnormalities

References and Reading

- Abramsky, L., & Chapple, J. (1997). 47, XXY (Klinefelter syndrome) and 47, XYY: Estimated rates of and indication for postnatal diagnosis with implications for prenatal counselling. *Prenatal Diagnosis*, 17, 363–368.
- Bender, B. G., Linden, M. G., & Robinson, E. (1993). Neuropsychological impairment in 42 adolescents

- with sex chromosomal abnormalities. *American Journal of Medical Genetics*, 48, 169–173.
- Bender, B. G., Harmon, R. J., Linden, M. G., Bucher-Bartelson, B., & Robinson, A. (1999). Psychosocial competence of unselected young adults with sex chromosome abnormalities. *American Journal of Medical Genetics-Neuropsychiatric Genetics*, 88, 200–206.
- Boada, R., Janusz, J., Hutaff-Lee, C., & Tartaglia, N. (2009). The cognitive phenotype in Klinefelter syndrome: A review of the literature including genetic and hormonal factors. *Developmental Disabilities Research Reviews*, 15, 284–294.
- Bojesen, A., Juul, S., & Gravholt, C. H. (2003). Prenatal and postnatal prevalence of Klinefelter syndrome: A national registry study. *The Journal of Clinical Endocrinology and Metabolism*, 88, 622–626.
- Bruining, H., Swaab, H., Kas, M., & Van Engeland, H. (2009). Psychiatric characteristics in a self-selected sample of boys with Klinefelter syndrome. *Pediatrics*, 123, 865–870.
- Bruining, H., De Sonnevill, L., Swaab, H., De Jonge, M., Kas, M., Van Engeland, H., et al. (2010). Dissecting the clinical heterogeneity of autism spectrum disorders through defined genotypes. *PLoS One*, 5, e10887.
- Geschwind, D. H., Gregg, J., Boone, K., Karris, J., Pawlikowska-Haddad, A., Rao, E., et al. (1998). Klinefelter's syndrome as a model of anomalous cerebral laterality: Testing gene dosage in the X chromosome pseudoautosomal region using a DNA microarray. *Developmental Genetics*, 23, 215–229.
- Geschwind, D. H., Boone, K. B., Miller, B. L., & Swerdloff, R. S. (2000). Neurobehavioral phenotype of Klinefelter syndrome. *Mental Retardation and Developmental Disabilities Research Reviews*, 6, 107–116.
- Graham, J., Bashir, A., Stark, R., Silbert, A., & Waltzer, S. (1988). Oral and written language abilities of XXY boys: Implications for anticipatory guidance. *Pediatrics*, 81, 795–806.
- Jacobs, P. A., & Strong, J. A. (1959). A case of human intersexuality having a possible XXY sex determining mechanism. *Nature*, 183(4657), 302–303.
- Klinefelter, H., Reifstein, E., & Albright, F. (1942). Syndrome characterized by gynecomastia spermatogenesis without A-leydigism, increased excretion of follicle stimulation hormone. *Journal of Clinical Endocrine Metabolism*, 2, 615–627.
- Landfranco, F., Kamschke, A., Zitzman, M., & Nieschlag, E. (2004). Klinefelter's syndrome. *Lancet*, 364, 273–283.
- Mandoki, S., & Hoffman, R. (1991). A review of Klinefelter's syndrome in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 30, 167–172.
- Netley, C., & Rovet, J. (1984). Hemispheric lateralization in 47, XXY Klinefelter syndrome boys. *Brain and Cognition*, 3, 10–18.
- Ratcliffe, S., Read, G., Pan, H., Fear, C., Lindenbaum, R., & Crossley, J. (1994). Prenatal testosterone levels in XXY and XYY males. *Hormone Research*, 42, 106–109.
- Ross, J. L., Zeger, M. P., Kushner, H., Zinn, A. R., & Roeltgen, D. P. (2009). An extra X of Y chromosome: Contrasting the cognitive and motor phenotypes in childhood in boys with 47, XYY syndrome or 47, XXY Klinefelter syndrome. *Developmental Disabilities Research Reviews*, 15, 309–317.
- Tartaglia, N., Cordeiro, L., Howell, S., Wilson, R., & Janusz, J. (2010). The spectrum of behavioral phenotype in boys and adolescents 47, XXY (Klinefelter syndrome). *Pediatric Endocrinology Reviews*, 8, 151–159.
- Van Rijn, S., Aleman, A., Swaab, H., Vink, M., Sommer, I., & Kahn, R. (2008a). Effects of an extra X chromosome on language lateralization: An fMRI study with Klinefelter men (47, XXY). *Schizophrenia Research*, 101, 17–25.
- Van Rijn, S., Swaab, H., Aleman, A., & Kahn, R. (2008b). Social behaviour and autism traits in a sex chromosomal disorder: Klinefelter (47, XXY) syndrome. *Journal of Autism and Developmental Disorders*, 38, 1634–1641.
- Van Rijn, S., Aleman, A., De Sonnevill, L., & Swaab, H. (2009). Cognitive mechanisms underlying disorganization of thought in a genetic syndrome (47, XXY). *Schizophrenia Research*, 112, 91–98.
- Van Rijn, S., Swaab, H., Baas, D., De Haan, E., Kahn, R., & Aleman, A. (2011). Neural systems for social cognition in Klinefelter syndrome (47, XXY): Evidence from fMRI. *Social Cognitive and Affective Neuroscience*. <https://doi.org/10.1093/scan/nsrO41>.
- Warwick, M. M., Doody, G. A., Lawrie, S. M., Kestelman, J. N., Best, J. J., & Johnstone, E. C. (1999). Volumetric magnetic resonance imaging study of the brain in subjects with sex chromosome aneuploidies. *Journal of Neurology, Neurosurgery & Psychiatry*, 66, 628–632.
- Wattendorf, D. J., & Muenke, M. (2005). Klinefelter syndrome. *American Family Physician*, 72, 2259–2262.

Websites

- American Association for Klinefelter Syndrome Information and Support – www.aaksis.org
 Klinefelter Syndrome Support Group – www.klinefelter-syndrome.org

Klonopin

- [Clonazepam: Definition](#)

Klozapol

- [Clozapine](#)

Kolvin, Israel (Issy)

Adam Feinstein

Autism Cymru and Looking Up, London, UK

Major Appointments (Institution, Location, Dates)

Professor of child psychiatry, Newcastle University 1977–1990; John Bowlby Foundation professor of child and adolescent mental health, Royal Free Hospital Medical School, London, 1990–1994; and emeritus professor of child psychiatry, 1994–2002.

Major Honors and Awards

Chair of the Association for Child Psychology and Psychiatry, 1994–1996.

Landmark Clinical, Scientific, and Professional Contributions

While in Oxford, working with Kit Ounsted, Kolvin carried out a particularly important study comparing children with autism and those with psychotic disorders such as schizophrenia. Prior to this work, there had been considerable confusion about the status of autism as a psychotic or a developmental disorder, and Kolvin's (1971) paper – still quoted today – clarified the issue considerably and, with other studies, firmly established autism as a developmental condition.

After his appointment as consultant in charge of the Nuffield Psychology and Psychiatry Unit in Newcastle upon Tyne in the mid-1960s, he carried out the so-called Thousand Family Study to study the effects of social deprivation and disadvantage from one generation to the next. He was able to show significant movement over time toward improved social conditions in a number of individuals. Another of his achievements in Newcastle

was an evaluation of the effectiveness of different forms of psychotherapy delivered in schools. This was groundbreaking work published in an influential book, *Help Starts Here* (1981). Many believed psychotherapy was not capable of evaluation, but this study demonstrated not only that it was possible but that meaningful results with significant implications for practice could be obtained.

Kolvin had been appointed to a personal chair in Newcastle in 1977 to become one of the first chair holders in child psychiatry. In 1990, he made a bold late career move when he was appointed to the John Bowlby chair of child and family mental health jointly held at the Royal Free Hospital and the Tavistock Clinic in London. A multicenter (London, Athens, and Helsinki) study of the effectiveness of psychotherapy in depressed children and a comparative study – published only a fortnight before Kolvin's death in 2002 – of two types of psychotherapy applied to sexually abused girls are particularly notable.

Short Biography

Born in Johannesburg, South Africa, in 1929, Israel (Issy) Kolvin worked on the wards and in the admitting room of Baragwanath Hospital in Soweto as a young doctor. The malnutrition on the children's wards and the severe psychiatric conditions presenting in the emergency room both made a profound impression on him. With few training opportunities open to him in South Africa, Kolvin obtained a place on a psychiatric training scheme in Edinburgh, Scotland.

He died in London on March 12, 2002, at the age of 72.

References and Reading

- Feinstein, A. (2010). *A history of autism: Conversations with the pioneers*. Oxford, England: Wiley-Blackwell.
- Kolvin, I. (1971). Studies in childhood psychoses. I. *Diagnostic criteria and classification*. *The British Journal of Psychiatry*, 118, 381–384.
- Kolvin, I. (1972). Infantile autism or infantile psychoses. *British Medical Journal*, 3(829), 753–755.

Kolvin, I., Ounsted, C., & Roth, M. (1971). Studies in the childhood psychoses. *V. Cerebral dysfunction and childhood psychoses. The British Journal of Psychiatry*, 118(545), 407–414.

Kolvin, I., Berney, T. P., & Yoeli, J. (1990a). Schizophrenia in childhood. In *Handbook of child and adult psychopathology: A longitudinal perspective* (pp. 99–113). Elmsford: Pergamon Press.

Kolvin, I., Miller, F. J. W., & Scott, M. I. (1990b). *Continuities of deprivation? The Newcastle 1,000 family study*. Farnum: Gower Publishing.

KS

► [Klinefelter Syndrome](#)

Kuwait and Autism

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Kuwait Center for Autism, Kuwait, Kuwait

Abbreviations

AAPEP	Adolescent and adult psychoeducational profile
ABA	Applied behavior analysis
ABLLS-R	Assessment of basic language and learning skills – revised
ADOS	Autism diagnostic observation schedule
AIT	Auditory integration training
ASDAN	Award scheme development and accreditation network
DISCO	Diagnostic interview for social and communication disorders
GARS	Gilliam autism rating scale
IEP	Individualized educational plan
MENA	Middle East and North Africa
PDD	Pervasive developmental disorder
PEP-3	Psychoeducational profile – 3rd edition
TTAP	TEACCH transition assessment profile

Historical Background

As a mother of a child with autism, Dr. Samira Al-Saad was unable to enroll her child in the education system of Kuwait when she was diagnosed with autism in 1985. With lack of awareness and knowledge about autism in her country at that time, she decided to pursue her studies in autism in the USA and shift from her specialty in geology which cares for rocks and oil to autism. She was able to earn a master’s degree and PhD degree in the field of autism. The first class started at her home in 1988 and tried unsuccessfully to convince the educational authority to start a school for such handicapped. She moved to Saudi Arabia during the invasion of Kuwait where she started (1992) her second classes as an experimental model. After 2 years, she moved back to Kuwait, and, with the cooperation of the Public Endowment Foundation, she was able to establish the first integrated center for educating children with autism in the MENA region.

The program started with basic educational facilities and focused on extensive training done by professionals from all around the world. The program extended its services throughout the year to include other basic areas such as teacher and parent training and vocational training and diploma in cooperation with well-known universities. As a result, the number of children increased from 6 in 1994 to more than 140 in 2013 and more numbers in other services. The diagnostics unit started at 1997 with well-known international professionals who helped to cover the area of diagnosis which cooperates later with developmental units in the government hospitals.

Legal Issues and Mandates for Services

Services in Kuwait for special needs started long ago since 1955 for blindness, hearing impairment, mental retardation, paralysis, and Down syndrome, but not for autism. When Law No. 13/96 was issued and the Higher Council of Handicapped was established in Kuwait by

which each child has the right to be educated and trained in government schools, and if the program is not available, then all their educational fees will be covered by the council. This encouraged private schools to open few classrooms within their schools for special needs including autism. Nevertheless, some private schools provide day care services rather than educational services and training for children with autism. But by time and more support for the budget of these schools from the council, their programs are getting better, especially when Law No. 8/2010 for the rights of people with disabilities was unanimously accepted by the Kuwaiti parliament.

In addition to educational fees of students, the council covers social help for the families who have a child with autism, if needed.

Overview of Current Treatment Options and Centers

Training using structured teaching, ABA, and sensory integration theories varies from school to school. In the Kuwait Center for Autism, the REACH philosophy was created where we use our experience and the international theories or part of them that work with our children and culture:

- R:** Relationship-focused and its enhancement.
- E:** Environment of the child and its structure are conducive for their development.
- A:** Activities designed to each child level and age through curriculum and continuous assessment.
- C:** Communication focus on the individual level.
- H:** Health enhancement of the child through a special program that is focused on integrated health.

As for awareness which started from 1995 until now with new ideas every year, as creating the Gulf Autism Union in 2002 which includes Qatar, Bahrain, the United Arab Emirates, and Oman and Kuwait as leading this union. The well-known outcome of this union came with success in 2008 when they raised the request to the United

Nations to celebrate World Autism Awareness Day internationally on the 2nd of April to which the United Nations agreed. From then on the whole world started celebrating and presenting autism campaigns. In Kuwait a lot of newspaper advertisements, meetings, and reports were done, and messages on buses which roamed around Kuwait roads carried autism information. A lot of lectures and parties were done yearly in cooperation with the Ministry of Education and Ministry of Health in April.

Research Activities

Research was one of the main goals since the Kuwait Center for Autism was founded. The research is carried in close collaboration with other parties to cover any shortage in medical or scientific professionals whether in or outside Kuwait. Some of the researches published in well-known scientific periodicals or journals in the world:

The Identification of Training and Educational Needs for Autistic Children from Parents' Perspective in Kuwait and Saudi Arabia (Samira Al-Saad, Ph.D., *Educational Journal of Academic Publication Council of Kuwait University, Vol. 12, No. 45, Autumn 1997 (Arabic)*), **Implementation of an Educational Program for Children with Autism: The Case of Kuwait** (Samira Al-Saad, Ph.D., *International Journal of Mental Health, Vol. 29, No. 2, Summer 2000*), **Biological Correlates of Childhood Autism: "Trace Elements"** (Abdullahi Fido, MD, MRCPsych, DPM & Hussein Dashti, MD & Samira Al-Saad, Ph.D., *Trace Elements and Electrolytes, Vol. 19, No. 4, Page 205–8, 2002*), **Toxic Trace Elements in the Hair of Children with Autism** (Abdullahi Fido, MD, MRCPsych, DPM & Samira Al-Saad, Ph.D., *Autism (Journal of Sage Publications & NAS), Vol. 9(3), Page 287–296, July 2005*), **Dental Care of Children in Kuwait Center for Autism: A Preliminary Study** (Samira Al-Saad, Ph.D., & H. Al-Omar, F. Ebrahim, B. Joseph, Faculties of Dentistry, Kuwait University, 2005), **Body Mass Index of Children with Autism in Kuwait** (poster)

(Abdullahi Fido, MD, MRCPsych, DPM & R. Varghese, MD & Samira Al-Saad, Ph.D., 2006), **Establishing Programs for People with Autism in Kuwait and the Gulf Region (poster)** (Samira Al-Saad, Ph.D., *Autism Safari – Cape Town Conference – 2006*), **Olanzapine in the Treatment of Behavioral Problems Associated with Autism. An Open-Label Trial in Kuwait** (Abdullahi Fido, MD, MRCPsych, DPM & Samira Al-Saad, Ph.D., (*Med. Principles Practice*.)17:415–418, 2008), and **Psychological Effects of Parenting Children with Autism** (Abdullahi Fido, MD, MRCPsych, DPM & Samira Al-Saad, Ph.D., (*International Open Journal of Psychiatry*), 2013)

Training

Major training programs are conducted by the Kuwait Center for Autism (more than 190 in 18 years). Different subjects were presented as:

- Understanding autism; play therapy; ABA; structured teaching; diagnostic tests: PEP-3, TTAP, DISCO, ADOS, and GARS; behavior aspects and its effects; art therapy; music therapy; Makaton (alternative communication system); portage; how to design IEP to the parents' expectation; AIT; sound beam and how it works with autism; International Conference 1 (Feb. 14–16, 2000); International Conference 2 (Dec. 9–11, 2003); helping parents cope the stress of having a child with autism; first aid workshops; developing social communication skills; Asperger; environmental aspects and autism; adulthood and autism; REACH philosophy; seizures and autism; how to communicate with your child; vocational trainings; creativity in educational activities; and ASDAN diploma for students.

More than 75 books were published in Arabic language for specialists, parents, siblings, etc.:

- Teaching Children with Autism to Mind-Read
- Supporting the Families of Children with Autism

- Autism: Medical and Educational Aspects
- A Comprehensive and Easy-to-Read Guide to the Most Current Research and Medical Therapies for Autism and PDD
- Understanding and Teaching Children with Autism
- Children's stories such as Captain Tommy, My Brother is Different, Autistic Planet, etc.
- Assessments such as GARS, PEP-R, AAPEP, and ABLLS-R

All of the above to maintain *our vision*:

- Leaders and pioneers in improving the future for autism in the Middle East

and *our mission* is to:

- Care and love
- Teach and train
- Development and research
- Family and community

See Also

- [Egypt and Autism](#)

References and Reading

- Al-Mohaisen, A. (2003). A daylighting and electric lighting study for the Kuwait Autism center project, A technical report submitted to Kuwait Engineering Consultants (KEG), Kuwait: Office of the Consultation and Career Development (OCCD), College of Engineering and Petroleum-Kuwait University.
- Al-Mohaisen, A., & Khattab, O. (2005). Sustainable design: Daylighting study of Kuwait Autism Center. In: *Proceedings of the 7th CTBUH international congress: Renewing the Urban Landscape*, New York.
- Al-Saad, S. (1997). The identification of training and educational needs for autistic children from parents perspective in Kuwait & Saudi Arabia. *Educational Journal of Academic Publication Council of Kuwait University*, 12(45), Autumn (Arabic).
- Al-Saad, S. (2000). Implementation of an educational program for children with autism: The case of Kuwait. *International Journal of Mental Health*, 29(2), Summer.

- Association American Psychiatric. (2002). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.
- Fido, A., & Al-Saad, S. (2005). Toxic trace element in the hair from Autistic children. *Autism (Journal of Sage Publications & NAS)*, 9(3), 287–296.
- Fido, A., Dashti, H., & Al-Saad, S. (2002). Biological correlates of childhood autism: “Trace elements”. *Trace Elements and Electrolytes*, 19(4), 205–208.
- State of Kuwait – Law number 8 in 2010 for (The rights of people with disabilities in Al-Kuwait) Al-Yawm, The Official Gazette, Issue No. 964, Dated 28 Feb 2010.
- The policy of inclusion: Law No. 13/96 – In Kuwait, the Kuwaiti government formed a policy of inclusion (law 13/96), adopted in (1996), which asserts that people with disabilities have a fundamental right to live and grow within their local communities, making Kuwait probably the only Gulf country to adopt such a policy as a law.

L

Label

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Synonyms

Naming; Tact

Definition

A label is a *name or vocabulary item* used to refer to a particular object, person, or concept. Approximately 70% of children's first words are labels. Typical toddlers tend to transition from sounds and babbles to labels at approximately 12 months of age. Through the second year of life, they acquire a range of labels, as well as words with other functions, such as action words (verbs), descriptors (adjectives), and greetings.

For young children with autism whose language is delayed or is developing atypically, labeling may be a relatively strength. Children may say the names of objects without directing their labels to others in or to communicate, or moving towards more meaningful connected speech. A child with ASD may have a large vocabulary of labels but

may not use them for social purposes (e.g., requesting or conversing).

See Also

- Communicative Functions
- Tact(s)

References and Reading

- American Speech-Language Hearing Association (ASHA). (n.d.). *Typical speech and language development*. Retrieved 31 May 2012, from <http://www.asha.org/public/speech/development/>
- Paul, R., Chawarska, K., & Volkmar, F. (2008). Differentiating ASD from DLD in toddlers. In *Perspectives on language learning and education* (Vol. 15, pp. 101–111). Rockville: American Speech-Language-Hearing Association. <https://doi.org/10.1044/le15.3.101>.

LAD

- Language Acquisition Device

L-Adrenalin

- Epinephrine

Lag Schedules

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Definition

Lag schedule is a type of differential reinforcement schedule used to promote the variability of behavioral responses or sequence of responses. On a lag schedule, reinforcement is delivered for a response that is different from a specified number of preceding responses. The schedule is denoted as Lag x in which the x refers to the number of previous responses the current response needs to differ from in order to be reinforced. For example, on a Lag 3 schedule, reinforcement will be provided on the fourth response, only if it is different from the three preceding responses. If the current response is not different, it is then included as one of the preceding responses, for the next response. Therefore, the lag “window” is constantly changing. For example, on a Lag 3 schedule, if a person is asked, “Name an animal that has a tail,” and answers “cat,” “dog,” “tiger,” “horse,” the last answer “horse” would result in reinforcement because it is different from the previous three responses. However, if the person says, “cat,” “dog,” “tiger,” “dog,” the last term would not result in any reinforcement because it is the same as one of the three preceding responses. The person could then say “cat” or “horse” because the lag window is now “dog,” “tiger,” “dog,” and either answer (“cat” or “horse”) would be different from these responses.

Historical Background

Lag schedules have been used as one of the ways to study and improve response variability of behavior. Response variability (or behavioral variability) refers to responses or sequences of responses that are different from each other. High variability of behavior may appear to be unpredictable or even random. Low variability

of behavior may be repetitive or look very similar. Although researchers began to explore in the 1960s whether response variability could be influenced through environmental changes (Pryor et al. 1969; Goetz and Baer 1973), lag schedules were not proposed in the literature until over a decade later, by Page and Neuringer (1985). At the time, variability of responding was recognized as a “side effect,” produced as a secondary characteristic of reinforcement schedules (e.g., intermittent schedules of reinforcement in which extinction produced increased response variability), but there was not a consensus as to whether behavioral variability could be *reinforced* as an operant behavior. Page and Neuringer set out to study exactly this by conducting a series of experiments to establish whether lag schedules could be used to increase response variability in pigeons. They demonstrated that pigeons could not only meet requirements for a Lag 1 and Lag 5 schedule but were also successful on a more sophisticated Lag 50 schedule. Their results supported the idea that variability could be directly reinforced. Page and Neuringer also reported that variability could be brought under stimulus control. They showed that the pigeons emitted higher variable responding when a blue light was on and a repeated sequence of pecking when a red light was on (and then successfully switched light assignments where variable responding was cued by the red light and repeated responding by the blue light). This demonstration of stimulus control of variable responding provided additional support that response variability could be an operant behavior. This seminal article was a catalyst for research aimed at increasing response variability using lag schedules of reinforcement.

Applied researchers of behavior analysis began to investigate the use of lag schedules with human populations, namely, those with autism spectrum disorders (ASD), in the 2000s. For example, Lee et al. (2002) demonstrated that a Lag 1 schedule paired with differential reinforcement of alternative behaviors (DRA) increased appropriate varied verbal responding to the social question “What do you like to do?” when compared to DRA alone. This was effective with two of the three male

participants and was one of the earliest demonstrations of lag schedules utilized with people.

Current Knowledge

Inflexible adherence to routines and engaging in repetitive patterns of behaviors, interests, or activities are some of the hallmark characteristics of ASD. Research indicates that children with ASD respond in ways that do not maximize reinforcement when compared to similarly aged children who are not on the autism spectrum. Instead, children with ASD tended to repeat or stay within a subset of choices rather than sample all of the offered choices leading to reinforcement. With the increase in developing interventions and strategies for individuals with ASD and fundamental need to increase response variability for this population, the application of lag schedules has led to an explosion of research in more applied contexts and settings within recent years.

Behavioral therapists should focus on improving response variability because response variability can be functional and adaptive. For example, response variability can aid in problem-solving. When the typical response is not sufficient to solve a problem, the person may be required to try other potential solutions rather than engaging in the same behavior repeatedly. Creativity is another example of when variability might be beneficial. Creativity may be conceptualized as novel responses that deviate from the status quo. Finally, a fundamental component in behavior-shaping procedures (i.e., new behaviors or skills are taught by reinforcing approximations of behaviors until the final target behavior is reached) requires a person to provide variations of the target response. If the person does not offer different approximations, reinforcement criterion will not be met, and shaping will not be successful. Behavior that is seemingly unpredictable or variable has traditionally been thought to be an uncontrollable or an internal characteristic to human nature. However, recent behavioral research indicates that behavioral variability could be an operant characteristic of behavior. In other words, variability of behavior can be

increased, maintained, or decreased through the environmental contingencies of reinforcement and extinction (Page and Neuringer 1985). This could be significant in developing effective interventions for individuals on the autism spectrum.

Lag schedules are one way to improve behavioral variability and only recently begun to be studied in applied contexts. Most of the studies focused on individuals with ASD and lag schedules are centered around testing the feasibility of lag schedules with various skills. Researchers have explored increasing response variability with people in toy play (Baruni et al. 2014; Lang et al. 2014), verbal responding to social questions such as “What do you like to do?” or “How are you?” (Lee et al. 2002; Lee and Sturmey 2006; Susa and Schlinger Jr 2012), building verbal tacting or intraverbal repertoires (Contreras and Betz 2016; Heldt and Schlinger 2012), increasing vocalizations for children with little echoic responding (Esch et al. 2009; Koehler-Platten et al. 2013), using sign language to mand (Silbaugh and Falcomata 2019), functional communication training (FCT; Adami et al. 2017; Falcomata et al. 2018; Muething et al. 2018), and improving feeding for children with limited food repertoires (Silbaugh et al. 2017).

Many of the current studies to date have demonstrated the use of fairly low lag schedules (i.e., Lag 1 to Lag 3) with people with ASD. For most of the participants, these simple lag schedules have been sufficient for increasing variability in responding. However, some participants required additional prompting in order to see increased variability. There is no consensus among researchers as to who would benefit from lag schedules and who would require the additional supports.

There may be some situations in which the general socially acceptable response would be to respond in a variety of ways but other times in which repeated behavior would be warranted. For instance, if two people meet for the first time, it would be socially acceptable to introduce themselves or ask the other person's name the first or even second time upon meeting. However, after multiple meetings, it might be frowned upon to continue to ask the other person what their name

is. Another context in which varied behavior is more accepted is joke telling. A person generally finds a joke funny the first time the joke teller says the punchline, but may not find it funny if the joke teller repeats the joke a second or third time. It could even be that the social acceptance for the joke teller tends to wane. In contrast, there are situations in which repeated behavior might be more beneficial than varied behavior. There is no shortage of successful people who value their morning rituals or routines to set the day, for example. Habits are behaviors that are so repeated or perfected they are nearly automatic, such that a person does not need to dedicate the mental capacity to engage in the behavior and can instead focus on other important decisions. A professional athlete's success may hinge on their ability to repeatedly perform at a high level, under considerable pressure. Therefore, the advantages of variability and repeated behaviors may be contextually dependent. Scientists in the basic behavioral sciences have demonstrated that variability may be brought under stimulus control in which a stimulus is paired with the lag schedule to increase variability. When the stimulus (colored cue card) is present and the lag schedule in play, the subject offers varied responding, whereas when a different stimulus is presented and a non-lag schedule used, the subject returns to less varied responding. While this type of responding has been shown to occur also with individuals with ASD, the cues have been obvious and contrived. A more naturally occurring clinical application for stimulus control with lag schedules has not been reported.

Future Directions

Since the majority of applied research on lag schedules have been conducted in the last decade, current results are promising but continue to be limited in our understanding to the clinical use of lag schedules with individuals with ASD. Much of the current research with human populations have been translational in nature, as researchers try to use what is currently known from basic behavioral research and understand how lag schedules might be applied to people. Therefore,

researchers may continue to focus on how to develop lag schedules specifically to improve people's lives.

Parameters of Lag Schedules in Applied Settings

Although there seems to be some indication of potential clinical utility to using lag schedules with individuals on the autism spectrum, questions about the parameters of this schedule of reinforcement remain. For instance, in some studies with human participants, the lag contingency was removed and variability of behaviors continued. In other cases, lack of variability returned when lag schedules were removed. Future research might explore the durability of varied behavior when lag schedules are used. While we hope that a person could learn to vary their behaviors in contexts in which varied behavior benefits him or her, does a lag schedule of reinforcement need to always be in effect to do so? At what point could a more natural, less stringent intermittent schedule be utilized and still maintain variability?

Research in applied settings have mainly utilized smaller lag schedules (e.g., Lag 5 or less). It would be beneficial to know whether there is any advantage to using higher lag schedules (e.g., Lag 10). For instance, although Lag 50 has been used with pigeons, it may not be clinically advantageous or even feasible to use such a large lag schedule with human populations. Preliminary results by Falcomata et al. (2018) indicate that there could be "ceiling effects" in which there is not a substantial variability improvement with higher lag schedules. This is an area that requires future research.

Social Validity of Lag Schedules

With applied research, procedures that are feasible or efficient also require social acceptability in their use. In the development of lag schedule applications, researchers should define guidelines for determining the level of variability that is socially desirable and appropriate when using lag schedules. Of the various methods for improving variability (e.g., extinction, percentile schedules, reinforcing novel behaviors), researchers should outline the benefits and drawbacks to

using lag schedules over other methods. In addition, most, if not all lag schedule research to date, has been conducted by researchers as implementers. A logical step for future research might be to understand how caregivers, clinicians, or teachers could effectively utilize lag schedules in various settings such as in a home or school/classroom setting.

Prerequisite Repertoires for Using Lag Schedules

When using lag schedules, responses are reinforced if they are different from a set number of preceding responses. Because this “window” is always changing as responses are offered, smaller lag schedules (e.g., Lag 1 or Lag 2) have resulted in the potential drawback of “higher-order stereotypy” in which the same few responses are given to meet the lag criterion. For example, on a lag 1 schedule, two responses could be alternated repeatedly and still result in reinforcement. While variability would be achieved, this level of variability may not be sufficient. Therefore, alternative lag schedules (e.g., lag-lag schedules, progressive vs. fixed lag schedules) that avoid higher-order stereotypy might be developed. Alternatively, larger lag schedules (e.g., Lag 5 or more) require a larger repertoire of responses to develop this window of responses. However, we do not have information on how large these repertoires need to be for lag schedules to be successful. Finally, there are questions about how lag schedules affect novel behaviors. Current research indicates that lag schedules have promoted novel responding, but these results have been inconsistent among participants. Therefore, researchers should not only explore strategies to consistently increase novel responding but also begin to understand the behavioral repertoire and participant characteristics that make novel responding more likely.

Package Interventions Using Lag Schedules

When therapists develop an intervention or clinical therapy plan for clients, they often include multiple interventions or strategies as part of a multicomponent package to affect targeted behaviors. Schedules of reinforcement might be just one

of the considerations in the intervention plan. While most of the current research has revolved around the basic feasibility of using lag schedules with human populations, as research in lag schedules continues to move forward, it will be important to understand the role lag schedules might play when combined with other intervention strategies. Are lag schedules effective when part of an intervention package? Could lag schedules enhance other interventions or vice versa? Or do they inhibit results? For example, in previous research, some participants required additional prompting to the lag schedules utilized before seeing improved variability.

See Also

- [Differential Reinforcement](#)
- [Reinforcement](#)
- [Schedule of Reinforcement](#)

References and Reading

- Adami, S., Falcomata, T. S., Muething, C. S., & Hoffman, K. (2017). An evaluation of lag schedules of reinforcement during functional communication training: Effects on varied mand responding and challenging behavior. *Behavior Analysis in Practice*, 10, 209–213.
- Baruni, R. R., Rapp, J. T., Lipe, S. L., & Novotny, M. A. (2014). Using lag schedules to increase toy play variability for children with intellectual disabilities. *Behavioral Interventions*, 29, 21–35.
- Contreras, B. P., & Betz, A. M. (2016). Using lag schedules to strengthen the intraverbal repertoires of children with autism. *Journal of Applied Behavior Analysis*, 49, 3–16.
- Esch, J. W., Esch, B. E., & Love, J. R. (2009). Increasing vocal variability in children with autism using a lag schedule of reinforcement. *The Analysis of Verbal Behavior*, 25, 73–78.
- Falcomata, T. S., Muething, C. S., Silbaugh, B. C., Adami, S., Hoffman, K., Shpall, C., & Ringdahl, J. E. (2018). Lag schedules and functional communication training: Persistence of mands and relapse of problem behavior. *Behavior Modification*, 42, 314–334.
- Goetz, E. M., & Baer, D. M. (1973). Social control of form diversity and the emergence of new forms in children's blockbuilding. *Journal of Applied Behavior Analysis*, 6, 209–217.
- Heldt, J., & Schlinger, H. D., Jr. (2012). Increased variability in tacting under a Lag 3 schedule of reinforcement. *The Analysis of Verbal Behavior*, 28, 131–136.

- Koehler-Platten, K., Grow, L. L., Schulze, K. A., & Bertone, T. (2013). Using a lag reinforcement schedule to increase phonemic variability in children with autism spectrum disorders. *The Analysis of Verbal Behavior*, 29, 71–83.
- Lang, R., Machalicek, W., Rispoli, M., O'Reilly, M., Sigafoos, J., Lancioni, G., Peters-Scheffer, N., & Didden, R. (2014). Play skills taught via behavioral intervention generalize, maintain, and persist in the absence of socially mediated reinforcement in children with autism. *Research in Autism Spectrum Disorders*, 8, 860.
- Lee, R., & Sturmey, P. (2006). The effects of lag schedules and preferred materials on variable responding in students with autism. *Journal of Autism and Developmental Disorders*, 36(3), 421–428.
- Lee, R., McComas, J. J., & Jawor, J. (2002). The effects of differential and lag reinforcement schedules on varied verbal responding by individuals with autism. *Journal of Applied Behavior Analysis*, 35, 391–402.
- Muething, C. S., Falcomata, T. S., Ferguson, R., Swinnea, S., & Shpall, C. (2018). An evaluation of delay to reinforcement and mand variability during functional communication training. *Journal of Applied Behavior Analysis*, 51, 263–275.
- Page, S., & Neuringer, A. (1985). Variability is an operant. *Journal of Experimental Psychology: Animal Behavior Processes*, 11(3), 429–452.
- Pryor, K., Haag, R., & O'Reilly, J. (1969). The creative porpoise: Training for novel behavior. *Journal of the Experimental Analysis of Behavior*, 12(4), 653–661.
- Silbaugh, B. C., & Falcomata, T. S. (2019). Effects of a lag schedule with progressive time delay on sign mand variability in a boy with autism. *Behavior Analysis in Practice*, 12, 124–132.
- Silbaugh, B. C., Wingate, H. V., & Falcomata, T. S. (2017). Effects of lag schedules and response blocking on variant food consumption by a girl with autism. *Behavioral Interventions*, 32, 21–34.
- Susa, C., & Schlinger, H. D. (2012). Using a lag schedule to increase variability of verbal responding in an individual with autism. *The Analysis of Verbal Behavior*, 28, 125–130.

Lamictal (lamotrigine)

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Synonyms

Lamotrigine

Mechanisms of Action

Lamotrigine is an anticonvulsant. It works by inhibiting voltage-activated sodium channels, antagonizes N-type calcium channels, and also has antilutamatergic action. The anti-glutamatergic effect is thought to be through inhibition of sodium, calcium, and potassium channels leading to decreased release of the excitatory amino acid glutamate. While there are studies to show potential favorable effects on cognition and mood stabilization, there is currently insufficient evidence to support the use of lamotrigine in Autism Spectrum Disorder.

Specific Compounds and Properties

Lamotrigine is an antiepileptic drug of the phenyltriazine class.

References and Reading

Book

- Schatzberg, A. F., & Nemeroff, C. B. (Eds.). (2017). *The American Psychiatric Association Publishing textbook of psychopharmacology*. American Psychiatric Publishing.
- McVoy, M., & Findling, R. L. (Eds.). (2017). *Clinical manual of child and adolescent psychopharmacology*. American Psychiatric Publishing.

Articles in Journal

- Belsito, K. M., Law, P. A., Kirk, K. S., Landa, R. J., & Zimmerman, A. W. (2001). Lamotrigine therapy for autistic disorder: a randomized, double-blind, placebo-controlled trial. *Journal of Autism and Developmental Disorders*, 31(2), 175–181.
- Kumar, B., Prakash, A., Sewal, R. K., Medhi, B., & Modi, M. (2012). Drug therapy in autism: a present and future perspective. *Pharmacological Reports*, 64(6), 1291–1304.
- Bromley, R. L., Mawer, G. E., Briggs, M., Cheyne, C., Clayton-Smith, J., García-Fiñana, M., ... & Liverpool and Manchester Neurodevelopment Group. (2013). The prevalence of neurodevelopmental disorders in children prenatally exposed to antiepileptic drugs. *Journal of Neurology, Neurosurgery, and Psychiatry*. <https://doi.org/10.1136/jnnp-2012-304270>.

Lamotrigine

► [Lamictal \(lamotrigine\)](#)

Language

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Definition

Language may be defined as a representational rule-based system of arbitrary symbols used to convey information among members of a shared language community. Language symbols may be spoken, written, or signed and may be combined and recombined in ways that enable speakers to convey an infinite number of thoughts, feelings, and ideas. Frith and Happe (1994) distinguish language and communication; we can communicate with others without recourse to a rule-based language system through our gestures, facial expressions, eye gaze, or other actions. Similarly, an individual may possess the words and grammar of his language community but still struggle to communicate. Within ASD, communication disorders are universal, while language impairments are extremely variable (Tager-Flusberg et al. 2005).

Language is a system of five component parts. Although these may be studied separately, in reality, they are highly interactive. *Phonology* refers to the sounds of speech and more specifically to the rules that govern combining speech sounds to form meaningfully distinct words. Phonemes are the minimal units that can differentiate meaning; for example, changing the phoneme /f/ in “phone” to a /t/ creates a new word with a distinct meaning, “tone.” *Semantics* refers to word meaning and is related to vocabulary. However, semantics is more than a simple store of known words. Semantics also considers how words are connected within the lexicon; for example, understanding metaphorical

expressions such as “that lawyer is a shark” requires not only knowledge of lawyers and sharks but also an understanding of the subtle features that are shared between these two seemingly distinct entities. *Morphology* refers to the prefixes and suffixes added to words that modify meaning. For instance, adding “un” to “happy” creates a distinct word which means the exact opposite of happy. Morphemes may also signal grammatical tense; “-ed” added to the end of many verbs will indicate an activity that happened in the past. *Syntax*, or grammar, is a set of hierarchical rules that govern word combinations and sentences. For example, the sentence “the boy was kissed by the girl” cannot be understood by word order alone (i.e., “boy kissed girl”) but requires appreciation of “passive” voice so that the girl may be correctly identified as the agent of the action kiss. As noted earlier, mastery of phonology, semantics, morphology, and syntax does not guarantee communication. For instance, sentences such as “the fish is on the table” have multiple meanings, and the intended meaning may only be clear when nonlinguistic contextual factors are taken into account. *Pragmatics* refers to this use of language in context. Pragmatic abilities require interlocutors to integrate linguistic information with a variety of other factors, including general knowledge, nonverbal cues, and shared knowledge between speakers. Because pragmatics frequently relies on understanding the internal states of communication partners, this aspect of language presents particular challenges to individuals with ASD.

References and Reading

- Frith, U., & Happe, F. (1994). Language and communication in autistic disorders. *Philosophical Transactions of the Royal Society, London B*, 346, 97–104.
- Norbury, C. F. (2011). Developmental language disorders: An introduction (ch. 16). In P. Howlin, T. Charman, & M. Ghazziuddin (Eds.), *Sage handbook of developmental disorders*. London: SAGE.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, R. Paul, A. Klin, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 335–365). Hoboken: Wiley.

Language Acquisition

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Definition

Language acquisition is a process that includes the perception and understanding of language, as well as the meaningful use of it. It is generally conceptualized as referring to the initial mastery of language occurring in infancy rather than the second language learning that can occur later in childhood or adolescence.

Language acquisition progresses through a number of stages, starting very young in infancy. Particularly in the early stages of language acquisition, a distinction can be made between receptive and expressive language. Although these domains of development progress at slightly different rates early in life, the excessive divergence of these areas can be a marker of atypical development. The first task of infants is to master the sound structure used in their native language; they are born with the ability to differentiate all phonemes used in human language. Toward the first birthday, however, through “perceptual narrowing,” the infant loses the ability to perceive phonemes that are not used in their native language. During this same time period, the infant is learning to parse the sounds that they hear into predictable “chunks,” a strategy which will eventually support their learning of words.

The next phase of language acquisition is the mastery of words, both receptively and expressively. Children begin to understand the meanings of words sometime around 10–12 months, and they begin to produce them shortly thereafter. First words are similar to the world around, and they generally refer to the most important aspects of the infant’s world: caregivers, favorite foods, games, etc. The child progresses to multiple word phrases and then to complex sentences; this transition requires the mastery of syntax and grammar. Along with these vocabulary and structural

advancements comes a greater proficiency with the pragmatics – or social usage – of language.

For most individuals, language refers to verbal utterances. However, those individuals who learn a gestural language follow largely the same pattern of development. Furthermore, starting at least in early childhood, and certainly by adulthood, all language – whether first or second, verbal or gestural – is processed predominantly in the left cerebral hemisphere.

Although the timeline of language acquisition has long been known, the mechanisms by which it proceeds remain up for debate. A variety of arguments have been made to explain the rapid developments over the first few years of life. The behaviorist account was one of the earliest accounts for the emergence of language. This account was rooted in the observations made about general principles of learning and suggested that language was taught to infants much the same way that they learned any other skill: through positive and negative reinforcement. This proposal has largely fallen out of favor, although the idea that language learning is the product of general learning mechanisms is still a primary argument in these ongoing discussions. In contrast, the nativist hypothesis proposes that the capacity for language is a product of an inborn cognitive “module.” By this account, language learning is the product of a specialized cognitive structure. A hybrid account is more widely accepted today and emphasizes the importance of both inborn predisposition and environmental input in the attainment of language.

See Also

- ▶ [Communicative Acquisition in ASD](#)
- ▶ [Language](#)
- ▶ [Normal Language Development](#)
- ▶ [Speech](#)

References and Reading

- Hoff, E. (2008). *Language development* (4th ed.). Belmont: Wadsworth Publishing.

- Johnson, S. (Ed.). (2010). *Neoconstructivism: The new science of cognitive development*. New York: Oxford University Press.
- Kuhl, P. (2004). Early language acquisition: Cracking the speech code. *Nature Reviews Neuroscience*, 5, 831–843.
- Saffran, J. (2003). Statistical language learning: Mechanisms and constraints. *Current Directions in Psychological Science*, 12(4), 110–114.
- Tomasello, M. (2008). *Origins of human communication*. Cambridge, MA: MIT Press.

Language Acquisition Device

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Synonyms

LAD; Language organ

Definition

The Language Acquisition Device, or LAD, was proposed by psycholinguist Noam Chomsky as the device or organ within the brain which houses human's innate ability to acquire and produce language. Stemming from the nativist theory which asserts that certain skills innately exist in humans, the LAD is thus in direct opposition with the behaviorist theory which suggests skills are attained through learning and reinforcement. According to Chomsky, the LAD is held responsible for allowing children to derive syntactic structure and rules from their native language through multiple stages of hypothesis testing. Through this process, the LAD transforms generalizations of speech and language into basic grammar. This hypothetical structure helps explain the immense surge in language ability in toddlerhood, but it is unavailable after an unspecified critical period of development.

The LAD is a theoretical construct, and therefore its relationship to language acquisition in Autism Spectrum Disorder has not been specifically tested.

See Also

► [Language Acquisition](#)

References and Reading

- Chomsky, N. (1965). *Aspects of the theory of syntax*. Cambridge, MA: MIT Press.
- Cook, V., & Newson, M. (1988). *Chomsky's universal grammar: An introduction*. Oxford: Blackwell.
- Lightfoot, C., Cole, M., & Cole, S. R. (2009). *The development of children* (6th ed.). New York: Worth.

Language Barriers Impact on Access to Services

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Definition

A "language barrier" constitutes difficulty or inability for two or more people to communicate because they speak different languages. It has been shown that language barriers among parents of children diagnosed with autism spectrum disorder (ASD) have a negative impact on the child's access to and optimization of healthcare and educational services (St. Amant et al. 2017).

Data collected in a large, urban area of California suggest that children of parents whose primary language was English were significantly more likely to have both social skills and communication goals included in their individualized education plan (IEP) compared to children of parents whose primary language was not English. Additionally, children of primary English speakers received significantly more hours of direct services from their state disability program. These data were

significant despite controlling for demographic covariates such as ethnicity, race, age, and gender.

It is doubtful that these disparities in access to ASD services solely represent differences in the variable profile of ASD across primary language. Instead, they most likely reflect the heightened challenge faced by parents whose primary language is not English to advocate for specific and appropriate healthcare and educational services for their child despite this being encouraged by IDEA parts B and C.

Many studies support these and related findings, suggesting that acculturation factors such as primary language, ethnicity, and race play a significant role in access to services for children with ASD (Liptak et al. 2008). A systematic review article recently published by Singh and Bunyak (2019) identified three interdependent themes that contribute to accessing appropriate ASD diagnoses and services including familial, cultural, and structural barriers, of which language barriers were found to compound the effects of cultural beliefs on access to services by negatively impacting the process of obtaining an ASD diagnosis. Further, Zuckerman, Mattox et al. (2014) were able to identify a recurring association between racial, ethnic, and language disabilities and the ability to access ASD diagnosis and services.

It is critically important to analyze acculturation factors impacting access to services for children with ASD because it is well-documented that intensive therapeutic and behavioral services result in better outcomes for these children (Reed et al. 2007). Publicly funded, state disability programs play an integral role in improving outcomes for children with ASD, with a large majority of families confirming that the support their child received made a positive difference in their family's life according to the National Core Indicators Child Family Survey in 2013.

Furthermore, IEPs are inherently designed to be specific and appropriate for a child with special needs, and while ASD represents a continuum of social and behavioral impairments, it is reasonable to conclude that an IEP designed for the

needs of a child with ASD would include goals focused on social and communication skills, because one of the core diagnostic criteria of ASD is persistent deficits in social communication and social interaction. The conclusion that language barriers significantly impact the likelihood that these ASD-specific goals are included in IEPs therefore likely represents true inequity in access to services (St. Amant et al. 2017).

According to the National Institute of Medicine, one of the six aims for improving healthcare is equity. It is therefore a matter of public health to acknowledge and understand the origin of differences in service access in order to adequately address this fundamental problem.

See Also

- [Bias in Assessment Instruments for Autism](#)
- [Education](#)
- [Service Utilization in Autism](#)
- [Social Class and Autism](#)

References

- Liptak, G. S., Benzoni, L. B., Mruzek, D. W., Nolan, K. W., Thingvoll, M. A., Wade, C. M., & Fryer, G. E. (2008). Disparities in diagnosis and access to health services for children with autism: Data from the National Survey of Children's Health. *Journal of Developmental Behavioral Pediatrics*, 29(3), 152–160. <https://doi.org/10.1097/DBP.0b013e318165c7a0>.
- Reed, P., Osborne, L. A., & Corness, M. (2007). Brief report: Relative effectiveness of different home-based behavioral approaches to early teaching intervention. *Journal of Autism and Developmental Disorders*, 37(9), 1815–1821. <https://doi.org/10.1007/s10803-006-0306-8>.
- Singh, J. S., & Bunyak, G. (2019). Autism disparities: A systematic review and meta-ethnography of qualitative research. *Qualitative Health Research*, 29(6), 796–808. <https://doi.org/10.1177/1049732318808245>.
- St. Amant, H. G., Schrager, S. M., Peña-Ricardo, C., Williams, M. E., & Vanderbilt, D. L. (2017). Language barriers impact access to services for children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-017-3330-y>.

Zuckerman, K. E., Mattox, K. M., Sinche, B. K., Blaschke, G. S., & Bethell, C. (2014). Racial, ethnic, and language disparities in early childhood developmental/behavioral evaluations: A narrative review. *Clinical Pediatrics (Phila)*, 53(7), 619–631. <https://doi.org/10.1177/0009922813501378>.

Language Comprehension

► Verbal Comprehension

Language Comprehension Disorder/Impairment/Disability

► Receptive Language Disorders

Language Cortex

► Cortical Language Areas

Language Development Survey

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Synonyms

LDS

Description

The *Language Development Survey* (LDS) is a widely used screening instrument, developed by

Leslie Rescorla, that obtains parent report of a child's expressive vocabulary and beginning word combinations. The LDS includes 310 words organized within 14 semantic categories (e.g., toys, body parts, food, animals, people). High-frequency words (e.g., "more") as well as less common words (e.g., "hamburger") are provided on the list. Lexical chunks (e.g., "all gone," "night night," "Sesame Street") are also listed on the LDS. When completing the LDS, parents are asked to indicate words on the list that their child spontaneously produces but also are allowed to add additional words spoken by the child. Parents may indicate words that the child produces in other languages by circling the English word on the list and writing the first letter of the language in which the child produces the word (e.g., circle "water" and write S if the child says "agua" in Spanish). Words on the list also may be circled if the child mispronounces the words or says it in "baby talk" (e.g., "baba" for bottle). In addition to identifying words that the child can produce, parents are asked if the child combines words and, if so, are asked to provide five examples of the child's best and longest sentences, from which mean length of utterance (MLU) can be calculated. Only expressive language abilities are assessed on the LDS.

The LDS is designed to be a screening instrument for language delays. It collects information regarding possible risk factors for language delays (e.g., history of ear infections, whether the parent is concerned about the child's language development, family history of delayed language development). Furthermore, norms for ages 18–35 months can be used to indicate whether a child's vocabulary and word combinations are delayed. The normative information provided for ages 18–35 also can be used for children with language delays who are older than the normative sample. The use of the LDS as a screening tool is efficient because it can typically be completed in approximately 10 min. Instructions are written on each scoring sheet and do not require additional explanation; however, it could be argued that a more detailed description of spontaneous word production could be provided. In addition, the LDS is written at a fifth-grade reading level (Achenbach 2011), making it a user-friendly tool for adults with differing abilities.

Gender-specific norms are available for ages 18–23, 24–29, and 30–35 months. Scores at or below the 15th percentile indicate delayed vocabulary development (Achenbach 2011). The commonly accepted cut point for identifying children at risk for language delay has been less than 50 words or no word combinations at 24 months of age (Rescorla 1989); thus, the survey is often administered at this age. This cut point identifies approximately 10% of toddlers in the general population.

In order to score the LDS, the total number of words circled and written in by the parents is counted. Total number of words from each semantic category can also be counted to provide more specific information about the child's lexical knowledge. In addition, if the child is combining words, clinicians can estimate MLU from the example sentences provided by the parent. Normative information for average phrase length is provided only for ages 24–29 and 30–35 months. MLU scores falling below the 20th percentile suggest delayed phrase development (Achenbach 2011).

The LDS is included in the Child Behavior Checklist for ages 1½ to 5 (CBCL/1½ to 5/LDS) in both English and a Latino Spanish language version. Translations in other languages are also available. Studies reporting findings for Latino children have been published (e.g., Patterson 1998; Seltzer 1995).

Historical Background

The LDS was created to provide a quick, reliable, and valid screening of vocabulary development in young children. The development of the LDS was based on previous studies utilizing language diaries kept by mothers and other studies assessing parent report of vocabulary (e.g., Benedict 1979). Revisions with lengths ranging from 240 to 353 words have been evaluated (Rescorla 1989) to obtain the final version with 310 words organized under 14 semantic domains. Over 1500 parents of toddlers ranging from 18 to 35 months (primarily between 24 and 30 months) varying in ethnicity and SES have completed the LDS in private pediatric clinics, inner-city hospitals,

day-care centers, family homes, and by mail (e.g., Klee et al. 1998, 2000; Rescorla 1989, 2005).

Psychometric Data

Reliability and validity data for the LDS have been reported in numerous studies. Rescorla (1989) reported 1-week test-retest reliability and Cronbach's alpha internal consistency of .99. LDS test-retest reliabilities of .97 for vocabulary score and .87 for mean length of phrases were found when mothers completed the LDS twice an average of 23 days apart (Rescorla and Alley 2001). Correlations between LDS vocabulary scores and numbers of objects and pictures named on various tests have ranged from .72 to .87 (Klee et al. 1998; Rescorla 1989; Rescorla and Alley 2001; Rescorla et al. 1993). Also, LDS vocabulary scores have been found to highly correlate with other standard measures including the Bayley Mental Development Index ($r = .79$; Bayley 1969), the Reynell Receptive Language Scale ($r = .56$) and the Reynell Expressive Language Scale ($r = .78$; Reynell and Gruber 1985), and the Vineland Adaptive Behavior Scales (composite scores $r = .71$; Sparrow et al. 1984) (Rescorla and Alley 2001).

High levels of sensitivity and specificity also have been reported in several studies (Klee et al. 1998; Rescorla 1989; Rescorla and Alley 2001; Rescorla et al. 1993), indicating validation of LDS cut points for language delay. Despite this, it has been suggested that the LDS yields a rather high false-positive rate (Klee et al. 1998). When compared to cutoff z-scores from the Reynell Expressive Language Scale, the cut point for language delay on the LDS was found to have a sensitivity score of 95% and specificity of 67% (Rescorla and Alley 2001). Therefore, a revised criterion to indicate delayed language has been suggested in an effort to reduce false positives. Klee et al. (2000) suggest using a cut point of fewer than 50 words *or* no word combinations *and* either six or more ear infections within the first 2 years of life *or* parental concerns about the child's language development for 24-month olds.

Rescorla and colleagues (2005) have compared the content validity of the LDS to the MacArthur-Bates Communicative Development Inventory: Words and Sentences Scales (MCDI-WS; Fenson et al. 1993) by having parents of over 290 typically developing toddlers complete both test protocols. At 680 words, the MCDI-WS is a longer instrument and more time consuming to complete than the LDS. However, the MCDI-WS has been widely used in studies of early language development in children with autism as well as children who are typically developing, late talkers, and those with a variety of neurodevelopmental disorders associated with language delay (e.g., Smith et al. 2007). A correlation of .95 ($p < .001$) was found between the LDS and the MCDI-WS vocabulary scores, although absolute vocabulary size was reported to be larger on the MCDI-WS. Mean phrase length as measured by the two instruments showed high agreement ($r = .90, p < .001$). Researchers wanting to make a more accurate estimate of absolute vocabulary size might choose to use the MCDI-WS, especially when assessing vocabulary skills in children within the 90th percentile. Also, researchers evaluating vocabulary size in children older than 24 months may wish to use the MCDI-WS due to frequently observed ceiling scores in children 30–32 months of age (e.g., Rescorla and Achenbach 2002). Furthermore, unlike the LDS, the MCDI-WS contains items asking about the child's vocabulary comprehension and morphological and syntactic development. Despite this, syntax scores from the MCDI-WS were shown to be highly correlated with MLU scores from both the MCDI-WS and the LDS (.81 and .78, respectively; Rescorla).

Clinical Uses

The Language Development Survey can be used as a screening tool in medical, clinical, and educational settings to provide a fast and inexpensive way to gather information about a young child's expressive language skills in order to identify children at risk for language delay.

See Also

- Language
- Language Disorder
- Screening Measures

References and Reading

- Achenbach, T. (2011). The language development survey (LDS). In *Achenbach system of empirically based assessment*. Retrieved October 6, 2011, from <http://www.aseba.org/research/language.html>
- Bayley, N. (1969). *Bayley scales of infant development*. New York: The Psychological Corporation.
- Benedict, H. (1979). Early lexical development: Comprehension and production. *Journal of Child Language*, 6, 183–201.
- Fenson, L., Dale, P. S., Reznick, J. S., Bates, E., Thal, D., Hartung, J., et al. (1993). *Guide and technical manual for the MacArthur communicative development inventories*. San Diego: Singular Press.
- Klee, T., Carson, D. K., Gavin, W. J., Hall, L., Kent, A., & Reece, S. (1998). Concurrent and predictive validity of an early language screening program. *Journal of Speech, Language, and Hearing Research*, 41, 627–641.
- Klee, T., Pearce, K., & Carson, D. K. (2000). Improving the positive predictive value of screening for developmental language disorder. *Journal of Speech, Language, and Hearing Research*, 43, 821–833.
- Patterson, J. L. (1998). Expressive vocabulary development and word combinations of Spanish-English bilingual toddlers. *American Journal of Speech-Language Pathology*, 7, 46–56.
- Rescorla, L. (1989). The language development survey: A screening tool for delayed language in toddlers. *The Journal of Speech and Hearing Disorders*, 54, 587–599.
- Rescorla, L. (2005). Age 13 language and reading outcomes in late-talking toddlers. *Journal of Speech, Language, and Hearing Research*, 48, 459–472.
- Rescorla, L., & Achenbach, T. M. (2002). Use of the language development survey (LDS) in a national probability sample of children 18 to 35 months old. *Journal of Speech, Language, and Hearing Research*, 45, 733–743.
- Rescorla, L., & Alley, A. (2001). Validation of the language development survey (LDS): A parent report tool for identifying language delay in toddlers. *Journal of Speech, Language, and Hearing Research*, 44, 434–445.
- Rescorla, L., & Schwartz, E. (1990). Outcomes of toddlers with specific expressive language delay. *Applied Psycholinguistics*, 11, 393–407.
- Rescorla, L., Hadicke-Wiley, M., & Escarce, E. (1993). Epidemiological investigation of expressive language delay at age two. *First Language*, 13, 5–22.
- Rescorla, L., Ratner, N., Jusczyk, P., & Jusczyk, A. (2005). Concurrent validity of the language development survey: Associations with the MacArthur-Bates

- communicative development inventories: Words and sentences. *American Journal of Speech-Language Pathology*, 14, 156–163.
- Reynell, J., & Gruber, C. (1985). *Reynell developmental language scales*. Los Angeles: Western Publishing.
- Seltzer, S. C. (1995). *Adaptacion, normalizacion, y estudios de validez del "sondeo del desarrollo de lenguaje" (SDL) para la deteccion de retraso de lenguaje expresivo en niños Mexicanos de 15 a 31 meses de edad*. Mexico: Universidad de las Americas.
- Smith, V., Mirenda, P., & Zaidman-Zait, A. (2007). Predictors of expressive vocabulary growth in children with autism. *Journal of Speech, Language, and Hearing Research*, 50, 149–160.
- Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1984). *Vineland adaptive behavior scales: Survey from manual* (Interview ed.). Circle Pines: American Guidance Service.

Language Disorder

► Childhood Aphasia

Language Impairment/ Disorder

► Speech/Communication Disabilities

Language Interventions

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Definition

Language intervention is broadly defined as "instructional interactions designed to enhance language" (Weiss 1993, p. 231). Language is described as a complex and dynamic system of conventional symbols that is used in various modes for thought and communication (American Speech-Language-Hearing Association [ASHA]

1982). Language develops through a socially and culturally accepted set of rules that govern sounds, words, sentences, meanings, and their use. There must be a common understanding of these rules in order to understand or formulate language. The effective use of language for communication requires a broad understanding of human interaction including nonverbal cues (gestures, facial expressions), motivation (a desire to communicate), and sociocultural roles (ASHA 1982). If there is a breakdown in any component of language, then effective communication is compromised.

Historical Background

Early language intervention evolved from psycholinguistic and behavioral research in the 1960s. Before that time, it was believed that intellectual ability was stable and therefore subject to little change. However, this belief was challenged by a group of researchers using principles derived from behavior analysis. These studies demonstrated good response to intervention addressing improvements in speech production in children with significant intellectual disabilities. During this time, information surfaced describing the development of language as opposed to speech production. It was in the 1970s that the first formal language programs appeared and the first conference on language intervention was held. It was also during this period that parents and caregivers were first included in language intervention programs. It was not until the 1980s that broader concepts of language and language intervention were described. These concepts included interdisciplinary models, the importance of the social aspects of language, expanding intervention to include infants and small children, the development of augmentative communication systems, and attention to the environmental context in which language is used (Bricker 1993). During the last two decades, language intervention has continued to evolve, placing increasing importance in the social and functional use of language in context. Additionally, we have observed a

greater emphasis on addressing early prelinguistic stages of language development such as the ability to attend to speech, the ability to understand and use gestures, development of joint attention, and symbolic behaviors such as play.

Rationale or Underlying Theory

Language skills are the foundation for literacy and learning skills. Communicative competence is related to the development of social behavior, reduced problem behavior, and generally more favorable outcomes. Competence in communication skills has been found to primarily determine the extent to which individuals with ASD can develop relationships and successfully participate in daily activities (Prizant and Wetherby 2005). Research has found that IQ and language competence by age of 5 are large predictors of outcomes in children with ASD (National Research Council [NRC] 2001).

Goals and Objectives

Establishing an effective communication system requires competence in language abilities. Children with autism spectrum disorders (ASD) present with deficits in broad range of language domains, including syntax (rules that govern the way words are combined to form phrases and sentences), semantics (meaning), pragmatics (social use of language), and comprehension. Additionally, individuals with ASD have specific impairments in the social aspect of language. Therefore, language intervention, in individuals with ASD, must include strategies to address the social aspects of language.

Prelinguistic Stage

Children with ASD have difficulty with the reciprocal nature of communication. At the early prelinguistic or preverbal stage of communication, language intervention in children with ASD should focus on establishing reciprocity with others. This may include activities that encourage

the back-and-forth flow of interactions (rolling a ball back and forth, peek-a-boo). These activities mirror the to-and-fro interaction of communication. Another core deficit widely described in children with ASD is a deficit in joint attention. Joint attention is broadly described as the ability to coordinate visual attention with a social partner (Munday and Burnette 2005). More recently, early language intervention for children with ASD includes addressing deficits in joint attention and recognizes this skill as a foundational skill in language development. Another core deficit in children with ASD is in the area of other symbolic behaviors. These other symbolic behaviors include difficulties in imaginative or pretend play (Rogers et al. 2005) and the impaired ability to imitate others. Language intervention programs for children with ASD should aim to improve these areas of symbolic behavior as a prerequisite to language learning.

Early Language Intervention

Early intervention in ASD focuses on attending to the speech of others and establishing a functional communication system. Individuals with autism often have core deficits in the reciprocal nature of communication. In order to establish communication, a symbolic system must be taught. This may be in the form of words, pictures, signs, as well as nonverbal communication systems such as gestures and facial expressions. Within the context of teaching a symbolic system, the reciprocal exchange, the idea that there is a giver and receiver of communication, must be taught. Additionally, children with ASD have reduced initiation of communicative acts; therefore, initiation of communication needs to be taught (NRC 2001). In addition to using language, the understanding of language including nonverbal communication should be addressed.

Expanding Language

The goal of language intervention for children with ASD, with an established verbal communication system, is to expand the means (how the child communicates) and function (why the child communicates). Children with ASD primarily use communication to request or regulate the actions

of others. Intervention at this stage focuses on expanding the variety of communicative functions while building longer, grammatically correct utterances. At this stage, intervention addresses the use and comprehension of linguistic concepts such as pronouns, adjectives, prepositions, plurals, etc. In addition to that are understanding and responding to questions, expanding vocabulary including multiple meaning words, and following single and multistep directions. Language intervention should address the use of language in social contexts such as play (Paul and Sutherland 2005).

Advanced Language

Language intervention for more advanced language learners centers on the social use of language. These skills include initiation and ending of conversations, conversational turn taking, and topic maintenance. Advanced language users with ASD have difficulties in understanding abstract language and idioms, making inferences, and recognizing conversational breakdowns and making conversational repairs. These difficulties are frequently masked by relatively intact structural language skills and vocabulary (Marans et al. 2005). Language intervention at this level frequently includes participation in social skills groups. Social skills groups create an environment that provides a more naturalistic context in which to teach the social aspect of language. These groups also allow for rehearsal of learned skills in a supportive setting with peers.

Treatment Participants

Children and adults of all ages may be candidates for language intervention. Language intervention may be initiated (a) to restore lost language abilities secondary to a traumatic head injury or stroke, (b) to maximize residual skills secondary to a degenerative disease, or (c) when a language delay or disorder is diagnosed in young children. This diagnosis may be secondary to a developmental delay or in isolation as in specific language impairment (SLI).

Treatment Procedures

There are a wide variety of language intervention approaches. These fall along a continuum in two major approaches, behavioral and developmental-pragmatic approaches.

Behavioral Approaches

There is a large body of research supporting the use of discrete trial behavioral approaches in initiating speech production and developing attention to and understanding of language. This approach is very structured and relies on adult/teacher direction. It was one of the earliest empirically based interventions for children with ASD. There have been criticisms that discrete trial approaches lead to passive response communication. More recently, behavioral approaches have evolved into what is now commonly referred to as “contemporary applied behavior analysis.” These behavioral approaches apply behavioral methods in a more naturalistic setting and address issues of initiation and generalization. There are studies that show that these more contemporary approaches are effective in improving language outcomes.

Developmentally Based

This approach assumes that children with ASD develop language in the same way and follow the same sequence as typically developing children do. In developmentally based approaches, intervention often focuses on the development of strong interpersonal relationships. The supporters of developmental approaches hypothesize that addressing key ASD-specific deficits, such as difficulty in developing interpersonal relationships, may yield broader changes in a range of behaviors. This approach often “follows the child’s lead,” allowing the child to choose the activities. The therapist creates the intervention around these naturally motivating activities, scaffolding communication and expectations as the child develops. One of the difficulties of the intervention is that it requires a great deal of creativity and decision making on the part of the interventionist. There is no prescribed protocol of intervention, so

caregivers may find the intervention difficult to carry over outside the therapeutic environment. There is some emerging research supporting developmentally based approaches primarily in the area of generalization and social aspects of language.

Efficacy Information

There is mounting evidence supporting the effectiveness of intensive early intervention with a substantial proportion of young children with ASD (NRC 2001). Intervention provided before age of 3 years has an even greater impact than intervention that is initiated after the age of 5.

The NRC (2001) indicates that the body of research supports improvements in children with ASD in response to language intervention. However, outcomes in this population are variable, with some children demonstrating substantial gains in a short period of time and others making slow gains over a long period of time.

Although there is strong support for language intervention, there is no sufficient efficacy data on specific language approaches over another. Comparative efficacy of communication approaches is often weak due to significant variability within the practice of a particular approach, and there is a lack of consideration of the overlap between different approaches, or categories of approaches (Prizant and Wetherby 2005).

Outcome Measurement

Outside of research, language intervention outcomes are frequently measured by periodic reevaluations, such as standardized tests, language or play samples, and progress against measurable objectives in the treatment plan. Additionally, caregiver and teacher reports of the functional use of language provide important information about the generalization and functional use of language across environments.

Qualifications of Treatment Providers

Speech-language pathologists are specifically trained to assess, develop intervention plans, and treat individuals with language deficits. In order to be licensed, they hold a master's degree in speech-language pathology and have successfully completed a nine-month clinical fellowship, the year following their graduation. Speech-language pathologists (SLPs) typically take the lead in the assessment, development, and implementation of language intervention programs. However, there are a variety of professionals that additionally address the language needs of children with ASD. Because intervention needs to take place across a variety of environments for learning and generalization of skills, SLPs work closely with caregivers, educators, psychologists, and other related professionals in enhancing language development across environments.

See Also

- Behavioral Approaches
- Developmental-Pragmatic Approaches/Strategies
- Joint Attention
- Language
- Play
- Pragmatic Language
- Social Skill Interventions
- Speech-Language Pathologist (SLP)

References and Reading

- American Speech-Language-Hearing Association. (1982). *Language* [Relevant Paper]. Retrieved from www.asha.org/policy
- Bricker, D. (1993). Then, now, and the path between: A brief history of language intervention. In D. B. Gray & A. P. Kaiser (Eds.), *Enhancing children's communication: Research foundations for intervention* (pp. 11–31). Baltimore: Brookes.
- Marans, W., Rubin, E., & Laurent, A. (2005). Addressing social communication skills in individuals with high-functioning autism and Asperger syndrome: Critical priorities in educational programming. In F. Volkmar, R. Pual, A. Klin, & D. Cohen (Eds.), *Handbook of*

- autism and pervasive developmental disorders* (pp. 977–1002). Hoboken: Wiley.
- Munday, P., & Burnette, C. (2005). Joint attention and neurodevelopmental model in autism. In F. Volkmar, R. Paul, A. Kiln, & D. Chen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 650–681). Hoboken: Wiley.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Paul, R., & Sutherland, D. (2005). Enhancing early language in children with autism spectrum disorders. In F. Volkmar, R. Paul, A. Kiln, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 946–976). Hoboken: Wiley.
- Prizant, B. M., & Wetherby, A. M. (2005). Critical issues in enhancing communication abilities for persons with autism spectrum disorders. In F. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 925–945). Hoboken: Wiley.
- Rogers, S., Cook, I., & Meryl, A. (2005). Imitation and play in autism. In F. Volkmar, R. Paul, A. Kiln, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 382–405). Hoboken: Wiley.
- Weiss, A. L. (1993). Planning language intervention for young children. In D. Bernstein & E. Tiegerman (Eds.), *Language and communication disorders in children* (3rd ed., p. 231). New York: Merrill.

Language Organ

- [Language Acquisition Device](#)

Language Outcome Measures

- [Measuring Language Change Through Natural Language Samples](#)

Language Processing

- [Receptive Language](#)

Language Style

- [Register Variation](#)

Language Tests

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Synonyms

[Norm-referenced assessment](#); [Norm-referenced testing](#); [Standardized tests](#)

Description

Language tests are designed to assess an individual's receptive and expressive oral and written language skills in one or more of the following language domains: syntax (grammar/word order), morphology (words and word endings that add grammatical information to a sentence such as the past tense “ed”), semantics (vocabulary including words with multiple meanings as in “I’m feeling *blue* today”), and pragmatics (i.e., the social use of language). The purposes of language testing include (1) screening to determine if further and more comprehensive language assessment is needed; (2) diagnosis of a language disorder, impairment, or delay; (3) description of baseline functioning in one or more language domains; (4) development of individualized goals for language intervention; and (5) measurement of changes in language functioning in response to a language intervention. “[See Also](#)” below for a list of various language tests.

Psychometric Data

The design, development, and selection of a particular language test should account for the same considerations as other norm-referenced tests. In a norm-referenced language test, an individual's performance is compared with the performance of a representative group of people, referred to as a norm group or standardization sample. As such, the individual to be tested should be

represented within the norm group. Considerations for representativeness include similarities in age, grade level, gender, geographic region, ethnicity, language use (i.e., similar dialect or first language), and social economic status (usually determined by the individual's or the parent's educational and occupational levels). The size of the norm group should be large enough to ensure representativeness; most experts recommend that the norm group be comprised of at least 100 individuals in each age group (Sattler 2008).

Additional considerations for development and selection of a language test include reliability and validity. Reliability is the repeatability of a measure. Test-retest reliability assesses the repeatability of an individual's performance; that is, whether the individual performs similarly on the same test readministered within a short interval from the first administration (usually within a 1–2-week period). An equally important consideration is interjudge reliability. This type of reliability assesses whether two examiners would score the performance of the tested individual similarly so that an equivalent performance score is obtained.

The validity of a test refers to whether a test measures what it is proposed to measure. Validity must be considered for both test construction and test administration. Content validity refers to whether the items in the test represent the particular language domain being tested. For example, a test of syntactic skills should be comprised of a variety of sentence structure and forms, and the correctness of the response should be based on the use of the correct syntactic form rather than other aspects of the response. Face validity, a subjective judgment, refers to whether examiners and individuals receiving the test perceive the test as a reasonable measure. Construct validity is the degree to which a test measures a specific language construct. For example, higher scores on a language test should reflect a higher degree of skill level; similarly, lower scores should reflect a disorder, impairment, or delay in the language domain being tested. Concurrent validity reflects how well the test results agree with other tests that measure the same language domain.

In addition to the validity of the test construction, validity of the test administration must be considered by the examiner. An individual's test performance can be compromised by a variety of factors including fatigue, distractibility, anxiety, physical handicaps, hearing loss, educational opportunities (such that the child/adult has not been exposed to the same language stimulation as those of his/her peers), and familiarity with the test material. One limitation of most language tests published in the United States is that the tests do not include children and adults in the norm group who are learning English as a second language or who are being raised in a culturally different community than the mainstream community. This limitation precludes the validity of using language test scores to identify and diagnose language impairments in culturally and linguistically diverse children and adults. In any case, language tests should not be the only tool used to determine an individual's language performance (see next section for further discussion of this topic).

Clinical Uses

The most common purpose of language testing is to establish a diagnosis; language testing alone is usually not sufficient to identify intervention goals or to document intervention outcomes. Therefore, language testing is only one aspect of a comprehensive language assessment and evaluation. Language tests should be supplemented with descriptive developmental assessments, criterion-referenced procedures, language sampling, naturalistic observations, and caregiver/teacher report measures so that a complete profile of an individual's language performance across settings and communication partners is obtained.

See Also

- ▶ [Children's Communication Checklist \(CCC-2\)](#)
- ▶ [Comprehensive Assessment of Spoken Language](#)
- ▶ [Norm-Referenced Testing](#)
- ▶ [Peabody Picture Vocabulary Test](#)

- ▶ [Pragmatic Communication](#)
- ▶ [Pragmatic Language Skills Inventory](#)
- ▶ [Pragmatic Rating Scale](#)
- ▶ [Pragmatics](#)
- ▶ [Rossetti Infant-Toddler Language Scale](#)
- ▶ [Social Language Development Test](#)
- ▶ [Standardized Tests](#)
- ▶ [Syntax](#)
- ▶ [Test of Written Language](#)

References and Reading

- Paul, R. (2007). *Language disorders from infancy through adolescence: Assessment & intervention* (3rd ed.). St. Louis: Mosby.
- Sattler, J. (2008). *Assessment of children: Cognitive foundations* (5th ed.). La Mesa: Sattler. ISBN 978-0-97026171-4-6-0.

Language Understanding

- ▶ [Receptive Language](#)
- ▶ [Verbal Comprehension](#)

Language Use

- ▶ [Pragmatics](#)

Language Variation

- ▶ [Register Variation](#)

Language/Autistic Regression

- ▶ [Acquired Autism](#)

Language-Based Learning Disability

- ▶ [Learning Disability](#)

Latency

- ▶ [Response Latency](#)

Latency of Response

- ▶ [Response Latency](#)

Latency, Second Edition

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Synonyms

[Interval](#); [Latent time](#); [Response time](#); [Time interval](#)

Definition

Latency is a type of continuous measurement used to measure behavior. Latency refers to the time from the onset of a stimulus, to the start of a response (i.e., behavior). Latency can be measured in seconds, minutes, or hours. Most commonly latency is represented in seconds or minutes. Latency is utilized as a measure for both adaptive and challenging behaviors.

Latency measures can provide important information about the impact an antecedent has on a behavior. Short latencies suggest a strong relationship between an antecedent and the behavior that follows it. Long latencies suggest a weaker relationship. Latency has been utilized in the assessment of behavior for individuals with Autism Spectrum Disorders (see “► [Functional Analysis](#)”) and may provide a more sensitive measure than other types of measurement (i.e., frequency or duration).

See Also

- [Antecedent-Behavior-Consequence \(A-B-C\) Analysis](#)
- [Functional Analysis](#)

References and Reading

- Call, N. A., Pabico, R. S., & Lomas, J. E. (2009). Use of latency to problem behavior to evaluate demands for inclusion in functional analyses. *Journal of Applied Behavior Analysis*, 42, 723–728.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Thomason-Sassi, J. L., Iwata, B. A., Neidert, P. L., & Roscoe, E. M. (2011). Response latency as an index of response strength during functional analyses of problem behavior. *Journal of Applied Behavior Analysis*, 44(1), 51–67.

Latent Class Analysis

- [Latent Variable Modeling](#)

Latent Profile Analysis

- [Latent Variable Modeling](#)

Latent Time

- [Latency, Second Edition](#)

Latent Variable Modeling

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Synonyms

[Confirmatory factor analysis](#); [Exploratory factor analysis](#); [Factor analysis](#); [Factor mixture modeling](#); [Finite mixture models](#); [Growth mixture modeling](#); [Item response theory](#); [Latent class analysis](#); [Latent profile analysis](#); [Mixture modeling](#); [Structural equation mixture modeling](#); [Structural equation modeling](#)

Definition

Latent variable modeling refers to a varied group of statistical procedures that use one or more unobserved (latent) variables to explain and explore relationships between a larger set of observed variables. There are many different types of analyses that fall under this general heading, but they can be generally grouped into four categories: models that assume one or more latent dimensions (exploratory and confirmatory factor analysis), one or more latent categories (latent class analysis, latent profile analysis), both latent categories and dimensions (factor mixture modeling), and structural models that enable us to examine relationships between latent variables.

Although not strictly latent models, taxometric procedures and cluster analyses also examine the question of whether observed data can be explained by one or more categories.

Together these procedures are useful for understanding: (1) how autism symptoms and related behaviors aggregate into dimensions; (2) the relationships between dimensions; (3) whether autism is qualitatively distinct from typical social and repetitive behavior; (4) the relative contributions of genetic, shared environment, and unique environmental factors to autism symptoms in behavioral genetics models; and (5) whether distinct subcategories of autism can be identified. The latter issue may be particularly important for identifying autism subgroups that more closely link to specific biological abnormalities or molecular etiologies.

See Also

► Factor Analysis

References and Reading

- Bollen, K. A. (1989). *Structural equations with latent variables*. New York: Wiley.
- Frazier, T. W., Youngstrom, E. A., Sinclair, L., Kubu, C. S., Law, P., & Rezaei, A. (2010). Autism spectrum disorders as a qualitatively distinct category from typical behavior in a large, clinically ascertained sample. *Assessment, 17*(3), 308–320.
- Georgiades, S., Szatmari, P., Boyle, M., Hanna, S., Duku, E., Zwaigenbaum, L., Bryson, S., Fombonne, E., Volden, J., Mirenda, P., Smith, I., Roberts, W., Vaillancourt, T., Waddell, C., Bennett, T., Thompson, A., & The Pathways in ASD Study Team. (2013). Investigating phenotypic heterogeneity in children with autism spectrum disorder: A factor mixture modeling approach. *Journal of Child Psychology and Psychiatry, 54*(2), 206–215.
- Ingram, D. G., Takahashi, T. N., & Miles, J. H. (2008). Defining autism subgroups: A taxometric solution. *Journal of Autism and Developmental Disorders, 38*(5), 950–960.
- James, R. J. E., Dubey, I., Smith, D., Ropar, D., & Tunney, R. J. (2016). The latent structure of autistic traits: A taxometric, latent class and latent profile analysis of the adult autism spectrum quotient. *Journal of Autism and Developmental Disorders, 46*(12), 3712–3728.
- Landa, R. J., Gross, A. L., Stuart, E. A., & Bauman, M. (2012). Latent class analysis of early developmental trajectory in baby siblings of children with autism. *Journal of Child Psychology and Psychiatry, 53*, 986–996.
- Markon, K. E., & Krueger, R. F. (2006). Information-theoretic latent distribution modeling: Distinguishing discrete and continuous latent variable models. *Psychological Methods, 11*(3), 228–243.
- McCutcheon, A. L. (2002). Basic concepts and procedures in single- and multiple-group latent class analysis. In J. A. Hagenaars & A. L. McCutcheon (Eds.), *Applied latent class analysis* (pp. 56–85). Cambridge: Cambridge University Press.
- Muthén, B., Asparouhov, T., & Rebollo, I. (2006). Advances in behavioral genetics modeling using Mplus: Applications of factor mixture modeling to twin data. *Twin Research and Human Genetics, 9*(3), 313–324.
- Ronald, A., Happe, F., Bolton, P., Butcher, L. M., Price, T. S., & Wheelwright, S. (2006). Genetic heterogeneity between the three components of the autism spectrum: A twin study. *Journal of the American Academy of Child and Adolescent Psychiatry, 45*(6), 691–699.
- Ruscio, J., Haslam, N., & Ruscio, A. M. (2006). *An introduction to the taxometric method: A practical guide*. Mahwah: Lawrence Erlbaum.
- Szatmari, P., Volkmar, F., & Walter, S. (1995). Evaluation of diagnostic criteria for autism using latent class models. *Journal of the American Academy of Child and Adolescent Psychiatry, 34*(2), 216–222.

Late-Onset Dyskinesia

► Tardive Dyskinesia

Law

► Autism in the Courtroom

Law Enforcement Agencies and Autism

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Definition

Law enforcement agencies include local police, state police, federal bureaus of investigation, state

and federal courts, and all other law enforcement agencies responsible for the enforcement of laws pertaining to the safety, welfare, and security of a country's citizens.

Historical Background

In the United States, autism is on the rise with now approximately 1 in 88 children identified as being on the autism spectrum (Centers for Disease Control and Prevention [CDC] 2012).

With this significant increase in the number of individuals being diagnosed with autism, with individuals with autism spectrum engaging in almost all activities of daily life, and with entitlements and eligibilities through the Individuals with Disabilities Education Improvement Act (IDEIA) as well as civil rights laws including the Americans with Disabilities Act (ADA) and Section 504 of the Vocational Rehabilitation Act (1975), individuals with Autism, as with other developmentally disabled individuals, have a sevenfold increased probability of coming in contact with law enforcement agents than age-matched, nondisabled individuals (Curry et al. 1993).

Affirming this inclusion of people with autism spectrum as being part of the fabric of American society with all of its diversity and ethnicity, culture, gender, religion, sexuality, and disability is their recent inclusion in television, in books, in movies, and in Internet activities.

For example, books such as Jody Picoult's (2010) *House Rules* focus on a young teenage boy with autism who is obsessed with police activities and crime scene replications. This teenager with autism eventually becomes "a person of interest" in a murder in his small town merely because of his obsessive-compulsive interest in the case.

Another recent book by Mark Haddon (2003), *A Curious Incident of the Dog in the Night-Time*, also depicts a boy with autism and his engagement with law enforcement as he tries to unravel the death of his neighbor's dog; he too is a "person of interest" due to his peculiar behavior.

More recently, there are increased depictions of autism on television including "The Big Bang Theory," "Bones," and "Parenthood" (Seginwall 2010).

Movies are also depicting autism. The first such movie was the four Oscar Award winning "Rain Man" (1988) and, more recently, the seven Oscar Award winning movie "Temple Grandin" (2010).

The significant increase in the diagnosis of autism in the United States is reflective of increased awareness by the public, expansion of the diagnostic symptomatology, environmental issues, and more sensitive data (Centers for Disease Control and Prevention [CDC] 2011).

With autism being depicted in media, both print and video, as well as the significant increase in numbers of individuals being diagnosed with the syndrome, it would be expected that our federal, state, and local law enforcement agencies would have a better understanding of the disability and that their reason for existence, i.e., "to protect and serve," would in turn benefit those with autism spectrum disorder (ASD). Such an expectation is not usually the case.

Although law enforcement agencies are becoming more proactive in training their forces to be sensitive to the symptoms and responses by those on the spectrum when confronted by a law enforcement agent, individuals with autism spectrum are continuing to be misunderstood, both by law enforcement agencies as well as the courts (Debbaudt and Rothman 2001). To this end, individuals with autism are being seriously hurt and sometimes even die while in custody.

In Los Angeles, CA, a young man with autism was shot and killed by a police officer merely because he "did not respond to his directives"; he appeared to be "a shadowy figure" as he lay under a balcony behind an apartment building (Watkins 2011).

His mother stated, after the shooting, that he was merely "experimenting with living outdoors" as he was "fascinated by reading about homelessness."

In 2006, a teenager with autism in Miami is now "brain dead" after officers "hog-tied him following an outburst." He apparently suffered from a "lack of oxygen to his brain," anoxia (Local10.com 2006).

Situations like this are increasing in frequency across the nation due to poor or no training for law enforcement agents in how to identify individuals

on the autism spectrum and then how to engage them in order to protect not only the person with autism but also the law enforcement officer[s].

One individual who has pioneered the training of law enforcement officers in dealing with individuals on the autism spectrum is Dennis Debbaudt. He published a handout for law enforcement agencies entitled "Avoiding Unfortunate Situations" (2002). This handout is used in training law enforcement officers to be able to identify when they are dealing with an individual on the autism spectrum and then how to effectively interact with that individual in order to de-escalate the situation and to avoid harm to the individual and/or to the law enforcement agent.

Current Knowledge

Reasons for Law Enforcement Engagement

Before discussing how it is that law enforcement agents should respond to matters involving individuals with autism, it is important to describe what those matters may entail.

First, if a child with autism is self-injurious, parents can be accused of child neglect and abuse when neighbors, acquaintances, family members, school personnel, or medical personnel notice that a child has bruises, abrasions, and/or lacerations on his/her body. In the majority of such cases, law enforcement is contacted by school, medical, and/or social service personnel.

Sometimes, under such conditions, the child is taken into protective custody, leaving the parent to defend his/her reputation all the while mourning for the loss of their child to the situation – one which is unfamiliar to the parents and the child, leaving them anxious and frightened. More often than not such conditions are determined to be self-imposed in nature, and the child returned to his/her family; but the event itself is traumatizing to the parents as well as the child (Rzucidlo 2006).

Another reason for involvement of law enforcement agencies can be when a person with autism is creating "a public nuisance." Such behavior can be as innocent as entering a neighbor's home without permission, looking in the windows of a neighbor's home, or, more

seriously, making "terroristic threats," i.e., yelling epitaphs out of fear or anger, and/or public nudity to name a few.

Another reason for involvement might be failing to heed the directions of law enforcement agents when, for example, it comes to motor vehicle infractions. Failing to stay within the speed limit, come to a full stop at a stoplight, and even driving too slowly can be reasons for law enforcement to attempt to issue summonses.

Another area can be domestic violence, where an individual with autism may be assaulting his mother or father or siblings while having a behavioral meltdown or tantrum. This may include property damage as well.

Currently, an ever-increasing area where law enforcement comes in contact with autism is in illegal Internet activity including hacking into Web sites and databases and, even more devastating, viewing child pornography.

And finally, a disturbing trend that is developing across the United States is when personnel in service agencies responsible for individuals with autism call law enforcement agents when an individual with autism becomes "out of control." For many such NGOs (nongovernment organizations), this is a policy for "managing" behavior, one which is ill-conceived.

Rather than contacting law enforcement to manage such situations, NGOs should have internal policies and training for staff to be able to be the first responders to behavioral outbursts rather than relying on lesser-trained or untrained law enforcement agents to manage these highly volatile and potentially dangerous situations.

With the aforementioned examples in mind, it is apparent that a person with autism can either be the victim or the perpetrator requiring nuanced responding on the part of the law enforcement agencies responsible for the case.

Today, due to heightened potential for civil suits against law enforcement agents, we are finding that law enforcement agencies are concerned not only for the health and safety of all individuals involved in a case but also ensuring that their investigative and retention practices do not expose them to civil or criminal suits. They sometimes find themselves defendants for overreacting

and causing harm, unlawful seizure/retention, and wrongful death (Johnson 2007).

To this end, law enforcement agencies are putting forth a greater effort to properly train law enforcement agents in “tactical communication.” George Thompson, founder and president of the Verbal Judo Institute, Auburn, New York, metaphorically states it quite succinctly:

The American Eagle as shown on the dollar bill holds a spear in one talon and an olive branch in the other. This is the image of the peace warrior – what every police officer should also reflect, however most police training focuses only on the spear. Tactical communication training focuses on the olive branch. (Thompson and Jenkins 2004)

From what is known about individuals with autism, being able to “tactically communicate” with them always results in the best possible outcome not only for the individual with autism but also for the law enforcement agent.

Responses of Individuals with Autism

Research has demonstrated that the majority of individuals with autism have overly active amygdala, the centers of the brain that trigger a fight-or-flight condition (Baron-Cohen et al. 2000). The prefrontal cortex is the area of the brain for executive functioning; it helps to determine whether a condition/situation requires “fight or flight” or should be directed to the hippocampus, the area for calming and happy thoughts (Umeda et al. 2010).

For individuals with autism, the prefrontal cortex and hippocampus are often not as robust as in typical individuals, and so, the amygdala then is the ultimate arbiter of environmental engagements. As such, most individuals with autism are in a state of alertness/vigilance, and any stimulus that has a history of an attribution of trauma or fear on the part of the individual can trigger a significant behavioral response, one that appears to be “overreacting.” Novel experiences also have a tendency to trigger the amygdala and result in fear, anxiety, and panic on the part of the individual with autism (Holmes 2009).

With this in mind, when law enforcement agents engage an individual with autism, more

often than not, if improperly handled, such engagement can trigger a fight-or-flight response on the part of the individual usually resulting in someone getting hurt or worse, mortally wounded.

Future Directions

Information that Law Enforcement Agents Should Have

Individuals in law enforcement should understand that autism is a developmental disorder that severely impacts the capacity of an individual to understand social values and mores and also to comprehend language, especially language that is replete with sarcasm, idioms, and/or suggestion (The Autism Society of America 2011).

To these ends, training for law enforcement agents must focus on tactical communication, i.e., approaches that utilize proper language, e.g., language that is concrete and to the point, and directives that tell an individual *what to do* as opposed to merely *what not to do*.

Individuals with autism become anxious when they are told to stop doing something without being told what they can and should do under the circumstances. For example, telling an individual not to view child pornography without explaining the profound impact on children and the severe legal consequences for the behavior must be replaced with something more prosocial. He must be told the consequences of such behavior and given one or two clear choices to replace that immoral/illegal behavior (Moran et al. 2011).

It is important to note that more alternative choices rather than fewer is not necessarily indicated as it may create anxiety on the part of people with autism as choice making is a conflicting, difficult exercise for them.

Being aware of “body language” is also important as individuals with autism have difficulty reading nonverbal social cues such as facial emotions and body posturing.

In an article “Contact with Individuals with Autism” by Dennis Debbaudt and Darla Rothman published in the FBI Law Enforcement Bulletin (April, 2001), these authors review the many

nuances of autistic individuals' responses in general and specifically when law enforcement agencies are involved with them in order that agents would not feel that the person is dismissing them, mocking them, or resisting their directives.

Additionally, they address how an agent should interact with a person with autism in order to defuse and/or not incite an individual when responding to a crisis or investigating a potential criminal activity. Specifically, they discuss behavior that will help agents recognize persons with autism and then they offer suggested responses on the part of law enforcement agents in order to secure the safety of the person with autism as well as the safety of the law enforcement agent and others.

Recognizing a Person with Autism

A person with autism:

- Has difficulties with communication, maybe nonverbal, maybe echolalic, i.e., repeating exactly what is said to them (may appear as mocking), may utilize a communication system such as the Picture Exchange Communication System (Bondy and Frost 2001), may use a computer-generated augmentative communication system, and/or may use sign language.
- Has significant social challenges and may appear to ignore/dismiss the law enforcement agent.
- May not heed a request to "stop" by running away in a fight-or-flight response.
- Oftentimes has heightened levels of anxiety and may rock back and forth covering ears and moaning or screaming.
- May not recognize the law enforcement agent's badge or uniform or even understand the authority associated with such information.
- Usually only understands concrete information and will respond in a concrete manner. For example, (agent) "What are you doing?"; (person with autism) "Standing here."
- Does not understand sarcasm, jokes, or teasing.
- Generally presents poor eye contact or maladaptive eye movements appearing to be indifferent or lying to the person making requests.
- Often has difficulties in determining personal space, more often than not by moving away, i.e., standing at a great distance from the law enforcement agent or, in some cases, rapidly approaching the agent in an effort to hug or make personal contact with the agent.
- May appear argumentative or belligerent by not responding to demands of the law enforcement agent or constantly inquiring as to why demands are being made.
- May be extremely fearful of dogs (K-9 responders) and sights (flashing lights) and sounds (sirens) associated with law enforcement.
- May be on medication that makes the individual appear to be drunk or on drugs.
- May try to get the law enforcement agent to leave by screaming incessantly or attempting to bite or spit at the law enforcement agent.
- May bang his head against hard surfaces or generally engage in self-injurious or non-directed assaultiveness.
- May try and calm himself/herself by engaging in significant self-stimulatory behavior including hand flapping, head weaving, body rocking, and clapping hands.
- May have epileptic seizure activity including petit and grand mal seizures.
- May incessantly inquire of the law enforcement agent personal information in a perseverative fashion including "What is your name?" "How old are you?" "When were you born?," all of which may appear to be distracting the agent from his line of duty when in actuality he is merely collecting information to better know the agent.
- Will be correct/exact in responding, often agreeing to culpability at a crime scene or during interrogation in an effort to try and get back to some order in their life, as change and disruption in routine is very distressing.
- During interrogation, due to central auditory processing dysfunctions, may affirm portions of what they hear, as they are not able to process all the questioning and information presented to them. This frequently leaves the interrogating agents with testimony that holds individuals with autism accountable for behavior that they did not engage in.

How to Respond to an Individual with Autism

- Speak slowly and concretely; do not use slang or metaphors.
- Allow extra time for the individual to process information, sometimes a 3–5 s delay.
- Position oneself so as not to give the impression of blocking the individual, which may only trigger a fight-or-flight condition.
- Be a calming influence under volatile conditions as individuals with autism can sense fear, anger, and other emotions in others more readily than being able to see/read such emotions on others' faces and body language.
- Do not engage in interrogation activities until the situation is calm and the individual with autism appears safe and more relaxed.
- Ask caregiver/parents about a situation rather than expecting the person with autism to be able to answer questions with a high degree of fidelity.
- Assess the situation for any personal injury to the individual with autism frequently as they may be unaware of personal safety and danger as well as, due to lower sensory feedback mechanisms, lack awareness of physical injury to themselves.
- Due to sensory dysfunction, be aware of excessive lights and noises as this can result in a fight-or-flight response on the part of a person with autism.
- Move slowly with confidence as this will calm the individual with autism and reduce the potential for viewing the agent as a threat.
- Avoid touching the individual as this may be alerting and evoke a fight-or-flight response.
- Be prepared to use restraint procedures only as a last resort and always be aware of the potential for asphyxiation or bodily harm as a person with autism, under such conditions, frequently is in a heightened state of arousal and will be fighting the controlling measures. This can escalate to the point of agents compressing a person's chest and resulting in unintentional death.
- Finally, if a person with autism is required to be brought into custody, be certain to assess the capacities of the individual through consultation with a mental health/human services

professional who understands autism. Be prepared to segregate individuals with autism from the general population of those incarcerated as those with autism will be vulnerable to abuse and assault and do not have the capacity to defend themselves.

In conclusion, law enforcement, when autism is a consideration, requires ongoing in-service training of agents. Such training will help agents to better understand the motives behind the person with autism's behavior and, in turn, defuse what might otherwise be a situation that could result in someone getting hurt or even fatally harmed.

See Also

- [Dispute Resolution Procedures](#)
- [Federal Rules of Evidence](#)
- [Formal Complaint](#)

References and Reading

- Autism Society of America. (2011). *About autism*. Bethesda: ASA Press.
- Baron-Cohen, S., Ring, H., Bullmore, E., Wheelwright, S., Ashwin, C., & Williams, S. (2000). The amygdala theory of autism. *Neuroscience and Biobehavioral Reviews*, 24, 355–364.
- Bondy, A., & Frost, L. (2001). The picture exchange communication system. *Behavior Modification*, 25(5), 725–744.
- Centers for Disease Control and Prevention. (2011). *National centers on birth defects and developmental disabilities [NCBDDD]*. Atlanta: Author.
- Centers for Disease Control and Prevention. (2012). Prevalence of Autism Spectrum Disorders. *MMWR*, 61, 3.
- Curry, K., Posluszny, M., & Krasha, S. (1993). *Training criminal justice personnel to recognize offenders with disabilities*. Washington, DC: Office of Special Education and Rehabilitative Services News in Print.
- Debbaudt, D. (2002). *Avoiding unfortunate situations*. Detroit: Autism Society of North Carolina.
- Debbaudt, D., & Rothman, D. (2001). *Contact with individuals with autism*. Washington, DC: The FBI Law Enforcement Bulletin [04/2001].
- Haddon, M. (2003). *The curious incident of the dog in the night time*. Toronto: Doubleday.
- Holmes, D. L. (2009). *The result of traumatizing events on a child with autism*. Princeton: Eden Press.
- Howell, C. (1988). *Rain man*. Los Angeles: United Artists.

- Johnson, K. (2007). *Police brutality cases on rise since 09/11*. Washington, DC: USA Today.
- Local10.com. (2006). *Family: Miami police hogtied autistic son*. Miami: Law Enforcement News.
- Monger, C., & Johnson, M. (2010). *Temple Grandin: The movie*. Los Angeles: HBO.
- Moran, J., Young, L., Saxe, R., Lee, S., O'Young, D., Mavros, P., et al. (2011). *Impaired theory of mind for moral judgment in high-functioning autism*. Cambridge: MIT, Department of Brain and Cognitive Sciences.
- Picoult, J. (2010). *House rules*. New York: Atria Books.
- Rzucidlo, S. (2006). *Autism 101 for mandated reporters of child abuse*. Minneapolis: Behavioral Institute for Children and Adolescents.
- Sepinwall, A. (2010). *How TV shows try to depict Asperger's syndrome*. Newark: The Star Ledger 02-28-2010.
- Thompson, G., & Jenkins, J. (2004). *Verbal judo; the gentle art of persuasion*. New York: Harper Collins.
- Umeda, S., Mimura, M., & Kato, M. (2010). Acquired personality traits of autism following damage to the medial pre-frontal cortex. *Society of Neuroscience*, 5(1), 19–29.
- Watkins, T. (2011). *Family of autistic man sues ex-cop over his death*. Washington, DC: The Washington Post [01/22/2011].

Suggested Hyperlinks

- Autism and Law Enforcement. Retrieved from www.neurodiversity.com/law_enforcement
- Autism and Law Enforcement: A plea for understanding. Retrieved from www.PsychologyToday.com
- L.E.A.N on Us: Law Enforcement Autism awareness Network. Retrieved from www.leanonus.org
- Police and Autism: Avoiding unfortunate situations. Retrieved from www.policelandautism.cjb.net

Law Enforcement Knowledge of Autism

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Definition

This entry is focused on providing information regarding law enforcement officers' (LEO) knowledge of autism spectrum disorder (ASD). Little research exists on the number of interactions

LEOs have with individuals with ASD; however it is likely, given the current prevalence rates, that such interactions take place regularly. There are a growing number of training programs aimed at providing LEOs with the ability to recognize the presence of behaviors commonly associated with ASD. Through trainings that increase awareness and knowledge of ASD, it is hoped that LEOs are better prepared to respond in professional situations involving individuals on the spectrum.

Historical Background

Exposure of problematic interactions between law enforcement officers (LEOs) and individuals with ASD supports the need for ASD-specific training for LEOs. There have been a number of highly publicized encounters between individuals with ASD and LEOs that have resulted in injury to caregivers or the death of an individual with ASD. Copenhaver and Tewksbury (2019) completed an analysis of media reports involving LEOs and ASD to determine the types of interactions LEOs most commonly have with individuals with autism. Analyses yielded several themes including incidents involving autistic persons being questioned or treated as suspects, incidents where individuals with ASD were the victim of crime, wandering/missing persons with ASD, and positive stories of interactions between LEOs and individuals with ASD. As a result of previous incidents where LEOs were ill-prepared to respond to calls involving individuals with ASD, several US states including Florida, New Jersey, and Pennsylvania now require LEOs to receive training in ASD. Given ASD affects an estimated 1 in 59 individuals 8 years and older in the USA (CDC), it is likely that LEOs interact with individuals with ASD on a fairly regular basis. Lack of knowledge of ASD may result in LEOs misinterpreting ASD-specific behavior as non-compliant, threatening, disorderly, or suspicious.

Interactions among LEOs and individuals with ASD may result from a variety of circumstances. Encounters may include individuals with ASD presenting as a victim or witness to crime; the individual may be suspected of committing a

crime or may have wandered away/gone missing. Previous research documents that persons with developmental delays, including those with ASD, are at increased risk for abuse and victimization (Petersilia 2001). In other instances, interactions between LEOs and individuals with ASD may be as a result of co-occurring psychiatric or medical concerns (Tint et al. 2017). LEOs may also be called upon in cases involving elopement, sensory overstimulation, and behavioral difficulties such as aggression, yelling, and self-injury. Using a nationally representative sample of adolescents and young adults with ASD, Rava et al. (2017) found that by age 21 approximately 20% had been stopped by the police for questioning, and almost 5% had been arrested. Rava et al. (2017) also found that individuals with ASD who display externalizing behaviors are more likely to be involved in the criminal justice system. Previous research offers conflicting findings as to whether children and young adults with ASD are at higher risk for involvement with the criminal justice system than the general population; however, it appears likely based on existing research that people with ASD are somewhat over-represented in the criminal justice system (King and Murphy 2014). As LEOs are the first contact between individuals and the penal system, instruction for LEOs specific to ASD is warranted. Such training provides LEOs with awareness of the behavioral symptoms and social impairments that may make an individual with ASD more vulnerable to be considered a suspect. For example, if an individual with ASD does not respond to police when spoken to, avoids eye contact upon questioning, does not remain still or tries to flee the scene, these behaviors may be misinterpreted as a sign of guilt as opposed to behavioral characteristics of ASD. Although in the UK it has been suggested that officers ask the question “Do you have any difficulties that I may not be aware of?” during initial contact with a person they suspect may have autism, individuals with ASD may not self-disclose their diagnosis to LEOs due to fear of discrimination (Crane et al. 2016) or due to communication/language delays and deficits associated with their ASD diagnosis. Murrie et al. (2002) noted that failure to correctly identify

individuals who are on the autism spectrum, or overlooking the behavioral features characteristic of an ASD diagnosis, may lead to inappropriate forensic assessment of criminal responsibility, legal decisions, or clinical interventions.

Current Knowledge

Research regarding LEO knowledge of ASD and disability awareness is sparse. A study by Modell and Mak (2008) found that 80% of officers were unable to accurately identify characteristics of ASD, had difficulty distinguishing between disabilities, and perceived themselves as competent in ASD when they may not have been. In a study of UK officers’ awareness and understanding of autism, Chown (2009) found that 62% of officers had received no formal training in ASD, and approximately half were able to recognize key features of autism. Although officers in the Chown study rated their competency in ASD lower than officers in the Modell and Mak study, findings suggest officers’ self-assessments may exaggerate competence. Research findings support that the majority of LEOs receive very little or no training in disabilities and ASD (Chown 2009; Eadens et al. 2016). As such, it is not surprising that they demonstrate limited knowledge of the characteristics of ASD and how to appropriately respond to incidents involving individuals with ASD.

Several studies have been conducted examining LEO training requirements and inclusion of training specific to ASD. Laan et al. (2013) conducted interviews with LEO training coordinators in seven states and compared LEO training materials related to recommendations for training on ASD and guidelines for training on other mental health disorders. Results indicated that most officers received between 400 and 770 hours of total training at the basic recruit level, of which only 3 to 12 hours were focused on mental health disorders. Training provided on mental health disorders that includes ASD is problematic as autism is not a mental health disorder. Although individuals with autism may present with co-occurring mental health diagnoses, providing training that

presents ASD as a mental health disorder to officers may result in confusion and impair LEOs' ability to accurately distinguish autism from other mental health disorders (Hepworth 2017). Gardner et al. (2019) found that although over half of LEOs reported having responded to a call involving an individual with ASD within the last year, over 70% of officers reported they had received no formal training in ASD. A quarter of these calls resulted in involuntary psychiatric hospitalization of the person with ASD. Although some states, such as Florida, New Jersey, and Pennsylvania have now mandated ASD training for LEOs, little is known regarding training content, format, or the impact of these trainings on practice. In addition, extension of this training requirement to other states and inclusion of other first responder departments (e.g., firefighters, EMS, etc.) remains unknown.

Research findings indicate that LEOs who have completed ASD-specific training report they feel better prepared to respond to incidents involving individuals with ASD (Gardner et al. 2019). A study of LEOs' perception of the most positive aspects of ASD-specific training included increased general knowledge of ASD, minimizing distress of individuals with ASD when responding to a call, practical application and usefulness for their LEO role, and modifying interview techniques. The top three aspects of training that LEOs deemed not satisfactory included training that lacked focus on ASD in the criminal justice context, overly simplistic content, and lack of practical application and relevance for LEOs (Crane et al. 2016). Chown (2009) police survey respondents suggested training should be streamlined into existing training programs, utilize a variety of delivery mechanisms as people learn best in different ways, include a variety of disabilities, incorporate advice on tactics to use, provide opportunities to interact with persons with ASD, and provide specific questions officers can ask to someone to determine if they have a hidden disability.

In considering the ideal format for increasing LEO awareness of ASD, in-person training is considered superior to online training modules.

Teagardin et al. (2012) provided ASD-related training to police officers via video. Officers who participated in the training outperformed officers who did not receive training on a measure of ASD-related knowledge; however, neither group demonstrated mastery of training material. The authors concluded that video training is likely insufficient to train officers on how to assess and respond to individuals with ASD. Instead of video or online training, in vivo training provided by individuals who have expertise in ASD and law enforcement is ideal as it increases the validity of the ASD training. It is recommended that in vivo training includes opportunities for role-playing and hands-on activities to supplement training. As a vulnerable population within our society, a growing awareness of ASD by LEOs is warranted; however, LEOs' experiences and knowledge specific to ASD remains an under-researched area.

Future Directions

Additional research is needed to determine the ideal format for training to increase LEOs' knowledge of ASD and prepare them for contact they may have with individuals with ASD in the course of their duties as an officer. In addition, further research is needed to determine the most pertinent and appropriate content to provide LEOs about ASD. Previous research suggests trainings should include information regarding the core features of autism, as well as practical knowledge and specific tactics for interacting with autistic persons. More information is needed regarding the types of calls LEOs receive in the line of duty that include individuals with ASD (e.g., missing person, victims of abuse, domestic assault, etc.) to assure that training programs provide information that is most applicable for LEOs.

As the reported prevalence rate of ASD increases, LEOs are increasingly more likely to interact with individuals with ASD within their professional role on a regular basis. Results of previous research supports there is a need for LEOs to receive formalized training in ASD

(Chown 2009; Eadens et al. 2016; Gardner et al. 2019; Modell and Mak 2008; Teagardin et al. 2012). Outcomes of such trainings result in LEOs reporting they feel better equipped to respond to calls involving individuals with ASD (Gardner et al. 2019). However, research is needed to determine the real-world impact of ASD-specific training on outcomes of calls (e.g., use of force, arrest, involuntary psychiatric hospitalization), especially given previous findings that LEOs tend to perceive themselves as competent in ASD when they may not be (Modell and Mak 2008; Chown 2009).

See Also

- First Responders and Autism
- Violence and ASD

References and Reading

- Chown, N. (2009). Do you have any difficulties that I may not be aware of? A study of autism awareness and understanding in the UK police service. *International Journal of Police Science and Management*, 12, 256–273. <https://doi.org/10.1350/ijps.2010.12.2.174>.
- Copenhaver, A., & Tewksbury, R. (2019). Interactions between autistic individuals and law enforcement: A mixed-methods exploratory study. *American Journal of Criminal Justice*, 44(2), 309–333.
- Crane, L., Maras, K. L., Hawken, T., Mulcahy, S., & Memon, A. (2016). Experiences of autism spectrum disorder and policing in England and Wales: Surveying police and the autism community. *Journal of Autism and Developmental Disorders*, 46(6), 2028–2041.
- Eadens, D. M., Cranston-Gingras, A., Dupoux, E., & Eadens, D. W. (2016). Police officer perspectives on intellectual disability. *Policing: An International Journal of Police Strategies & Management*, 39, 222–235.
- Gardner, L., Campbell, J. M., & Westdal, J. (2019). Brief report: Descriptive analysis of law enforcement officers' experiences with and knowledge of autism. *Journal of Autism and Developmental Disorders*, 49(3), 1278–1283.
- Hepworth, D. (2017). A critical review of current police training and policy for autism spectrum disorder. *Journal of Intellectual Disabilities and Offending Behaviour*, 8(4), 212–222.
- King, C., & Murphy, G. H. (2014). A systematic review of people with autism spectrum disorder and the criminal justice system. *Journal of Autism and Developmental Disorders*, 44, 2717–2733. <https://doi.org/10.1007/s10803-014-2046-5>.
- Laan, J. M., Ingram, R. V., & Glidden, M. D. (2013). Law enforcement training on mental disorder and autism spectrum disorders in the Southeastern United States. *Journal of Global Intelligence & Policy*, 6, 10.
- Modell, S. J., & Mak, S. (2008). A preliminary assessment of police officers' knowledge and perceptions of persons with disabilities. *Intellectual and Developmental Disabilities*, 46(3), 183–189.
- Murrie, D. C., Warren, J. I., Kristiansson, M., & Dietz, P. E. (2002). Asperger's syndrome in forensic settings. *International Journal of Forensic Mental Health*, 1(1), 59–70.
- Petersilia, J. R. (2001). Crime victims with developmental disabilities: A review essay. *Criminal Justice and Behavior*, 28(6), 655–694.
- Rava, J., Shattuck, P., Rast, J., & Roux, A. (2017). The prevalence and correlates of involvement in the criminal justice system among youth on the autism spectrum. *Journal of Autism and Developmental Disorders*, 47, 340–346. <https://doi.org/10.1007/s10803-016-2958-3>.
- Teagardin, J., Dixon, D. R., Smith, M. N., & Granpeesheh, D. (2012). Randomized trial of law enforcement training on autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6, 1113–1118. <https://doi.org/10.1016/j.rasd.2012.02.002>.
- Tint, A., Palucka, A. M., Bradley, E., Weiss, J. A., & Lunskey, Y. (2017). Correlates of police involvement among adolescents and adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47, 2639–2647. <https://doi.org/10.1007/s10803-017-3182>.

Lawson, Wendy

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Name and Degrees

Dr. Wendy Lawson (Bss, Bsw [Hons], GDip [PsychStud], GDip [Psych]) is an Australian writer, poet, adult educator, and immensely popular public speaker with Asperger's syndrome. In 2009, she was awarded her Ph.D. in Psychology at Deakin University in Victoria, Australia. Her work explores the influence of neurologically

typical development on that of development in individuals with autism and its impact upon learning styles and related parental distress. Dr. Lawson has been married, separated, and divorced. She brought up four children and has experienced the death of one of her teenage sons. Wendy's younger son, Tim, also has Asperger's syndrome. She has said: "I knew Tim was like me in so many ways. He has a big heart and a wide appealing grin. Seeing him experiencing similar difficulties to my own, though, is painful and I so wish I could make life easier for him."

At school, Wendy was considered to be intellectually disabled and "almost incapable of doing as she is told." In her teens, she was misdiagnosed as "schizophrenic." This label stuck for more than 25 years, until she was diagnosed in 1994 as being on the autism spectrum. She coined the term "diffability" to refer to her view that high-functioning autism is as akin to a difference as to a disability.

In 2008, Dr. Lawson was awarded fourth place as "Victorian Australian of the Year."

References and Reading

Books

- Lawson, W. (2000). *Life behind glass: A personal account of autism spectrum disorder*. London: Jessica Kingsley.
- Lawson, W. (2001). *Understanding and working with the spectrum of autism: An insider's view*. London: Jessica Kingsley.
- Lawson, W. (2003). *Build your own life: A self-help guide for individuals with Asperger syndrome*. London: Jessica Kingsley.
- Lawson, W. (2004). *Sex, sexuality and the autism spectrum*. London: Jessica Kingsley.
- Lawson, W. (2006). *ASpoetry: Illustrated poems from an Aspie Life*. London: Jessica Kingsley.
- Lawson, W. (2008). *Concepts of normality: The autistic and typical spectrum*. London: Jessica Kingsley.
- Lawson, W. (2010). *The passionate mind: How people with autism learn*. London: Jessica Kingsley.

Lead Exposure and Autism

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Synonyms

[Lead poisoning](#); [Plumbism](#)

Definition

Lead exposure results when an individual is exposed to this heavy metal. Lead is toxic to a range of organ systems and is particularly problematic in children where it can interfere with development and lead to a range of problems in behavior and in learning. Physical symptoms such as abdominal pain, irritability and confusion, and anemia can be noted, and with high levels of lead, seizures and even death can result. Lead can be taken into the body through contaminated food, water, and the air. Lead paint has been the most frequent source of exposure in the past (fortunately this is now decreasing because of public health awareness and new standards for paint and other materials) but can still arise in older homes. Prevention is important. Children, e.g., those with autism, who frequently mouthed objects can be at higher risk.

At lower levels of lead exposure, children have few problems although even then subtle problems may exist. The Center for Disease Control and Prevention considers any lead level of 10 µg/dl or above to be abnormal. A level of lead above this level should lead parents/caretakers to search for sources of exposure. At levels of 40 µg/dl or above, anemia may be noted, with high level (60 µg/dl) resulting in developmental and behavioral change and even brain symptoms (coma or seizure).

Primary care providers should routinely screen for lead exposure in younger children and even in older children who may be at risk because of possible exposure, e.g., through repeated

LDS

► [Language Development Survey](#)

mouthings of objects. Clearly removal of sources of exposure is important. A range of medications are available to lower levels of lead through chelation (chemicals that bind with lead so that it is excreted from the body). These agents do have some risks and side effects, and treatment should be carefully managed by a physician.

See Also

- Chelation
- Pica

References and Reading

- Accardo, P., Whitman, B., et al. (1988). Autism and plumbism. A possible association. *Clinical Pediatrics*, 27(1), 41–44.
- Cohen, D. J., Paul, R., Anderson, G. M., & Harcherik, D. F. (1982). Blood lead in autistic children. *Lancet*, 2(8289), 94–95.
- Volkmar, F., & Wiesner, L. (2009). *A practical guide to autism*. Hoboken: Wiley.

Lead Poisoning

- Lead Exposure and Autism

Leaky Gut Syndrome

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Definition

Autism spectrum disorders (ASD) comprise a group of diagnoses that are collectively characterized by qualitative impairments in social interaction and communication along with stereotyped and repetitive behaviors and interests. The underlying cause, or etiology, of ASD is poorly

understood but may be multifactorial with both genetic and environmental factors playing a role. The “leaky gut syndrome,” as it applies to ASD, is based on the theory that the lining, or mucosa, of the intestinal wall is damaged, allowing the contents of the intestine, such as undigested food, toxins, bacteria, and waste, to “leak through” an abnormally permeable bowel wall. There are many theories involving the leaky gut, or increased intestinal permeability specific to ASD. One of these is the leaky gut-opioid excess theory, which posits that incompletely digested proteins derived from the diet are absorbed through a leaky gut, thereby entering the central nervous system and interfering with normal brain function which may cause or exacerbate the behavioral symptomatology of ASD (White 2003).

Historical Background

Many parents of children with ASD report concerns with gastrointestinal symptoms. Associated behavioral symptoms, including behavioral disruption at mealtimes, unusual vocalizations, sleep disruption, and irritability, could be secondary to gastrointestinal distress (Buie et al. 2010). There have also been observations of gastrointestinal symptoms coinciding with the onset of parental concerns regarding development. The hypothesis of a “gut-brain linkage” in ASD is based in part on these reports (White 2003). The suggestion of potential links between celiac disease (a digestive condition triggered by consumption of the protein gluten found in wheat, barley, and rye) and malabsorption in ASD arose in early research conducted in the 1960s and 1970s (Horvath and Perman 2002). This was followed by relatively little investigation into the possibility of gastrointestinal abnormalities in autism until the 1990s when research positing a difference in the immune function and permeability of the gut was published (Erickson et al. 2005). Since that time, exploration of gastrointestinal differences and their association with ASDs has continued, along with debate regarding whether gastrointestinal symptoms are truly more prevalent than in

the general population and whether there is an autism-specific gastrointestinal abnormality (Buie et al. 2010).

The leaky gut-opioid excess theory involves peptides derived from the partial digestion of gluten (a protein found in wheat, barley, and rye) and casein (a protein found in milk), specifically gliadomorphins and casomorphins respectively, being absorbed through a leaky gut and entering the central nervous system (de Magistris et al. 2010; Millward et al. 2009). The theory hypothesizes that these peptides, referred to as exorphins due to their dietary origin, mimic the opioid hormone β -endorphin and interfere with normal brain function. This theory was based in part on a concept posited by Panksepp in 1979 that the behavioral symptomatology of autism may be secondary to increased activity of the opiate system. The concept was based on work performed in young animals. These animals showed symptoms similar to those seen in children with ASD when they were given low doses of opiate drugs (Christison and Ivany 2006; Panksepp 1979). Subsequently, Dohan developed a hypothesis regarding an excess of dietary peptides from gluten and casein interacting with a genetic disposition in the etiology of schizophrenia. This hypothesis was based in part on anecdotal reports of increased incidence of celiac disease in schizophrenia and the decreased prevalence of schizophrenia in populations that consumed little to no grain products (Dohan 1988). Meanwhile, people with ASD were reported to show an increase in 24-h low molecular weight peptide excretion in their urine (Cass et al. 2008). Increased opioid levels were also reported in the cerebrospinal fluid of individuals with ASD (Millward et al. 2009; White 2003). Clinical trials of opiate antagonists in the treatment of ASD followed. Researchers proposed dietary elimination of these proteins as a potential treatment for ASD. Anecdotal reports of this dietary intervention noted improvements in symptoms of ASD (Christison and Ivany 2006). To date, the gluten-free, casein-free diet is a popular but yet unproven intervention in ASD (Buie et al. 2010).

In order for these peptides to be abnormally absorbed, there would have to be evidence of

increased intestinal permeability or a “leaky gut.” In the healthy gut, the intestinal mucosa serves as a barrier against the largest site of exposure to the external environment in the human body. The integrity of this barrier is important to protect the body against bacteria, toxins, or antigenic molecules (a substance that can trigger the production of antibodies by the immune system). A layer of epithelial cells serve as this physical barrier. Underlying this barrier is the highly immunoreactive subepithelium, which serves as a line of defense to invading organisms that breach the epithelium. Breaching the epithelial barrier can lead to pathologic stimulation of the subepithelium by exposing it to the numerous antigenic molecules, including food antigens, located in the lumen of the intestine. Intestinal permeability has been associated with several medical conditions, including celiac disease, inflammatory bowel disease, diabetes mellitus type 1, and atopic disorders such as allergies, eczema, and asthma (Liu et al. 2005). Intestinal permeability is often tested through the differential sugar absorption test. This test involves the ingestion of two indigestible sugars of different molecular sizes and absorption routes, which are secreted in the urine after uptake from the gastrointestinal tract, typically lactulose and mannitol. In the healthy gut, absorption of the larger sugar lactulose is much less than for the smaller sugar mannitol. In pathologic conditions, the permeability for the larger sugars is increased while that for the smaller sugars remains the same or decreases (D’Eufemia et al. 1996; de Magistris et al. 2010; Liu et al. 2005). Therefore, the ratio of the large (lactulose) to small (mannitol) sugar is increased. This has been the method for assessing intestinal permeability in children with autism spectrum disorders. D’Eufemia et al. (1996) performed early work on this topic. They evaluated 21 children with autism who were without gastrointestinal symptoms and 40 healthy, age-matched controls. Nine of the 21 children with autism had an altered lactulose to mannitol ratio compared to none of the controls. The researchers noted that mannitol passes through the gut wall via a trans-cellular pathway. Lactulose, however, passes through the gut wall via intercellular routes as do

other heavier compounds, including peptides. They hypothesized that the increased permeability may be secondary to damage to the tight intercellular junctions of the gut mucosa. These tight intercellular junctions connect the epithelial cells to their neighbors and limit movement of molecules between the cells (Liu et al. 2005).

Although the possible mechanisms are unknown, multiple hypotheses have been suggested regarding the potential cause of increased intestinal permeability in individuals with ASD (White 2003). Gut inflammation may lead to increased permeability as well as diminished digestive enzyme activity including that of peptidases (enzymes that break down proteins), which may account for the incompletely digested peptides. Conversely, the loss of integrity of the gut mucosa could lead to pathologic stimulation of the immune system given the increased exposure of immunogenic compounds (Liu et al. 2005; White 2003). Inflammatory responses in the gut of children with ASD have been reported (Erickson et al. 2005; Horvath and Perman 2002). Intestinal pathology described by Wakefield et al. (2005) included ileal lymphoid nodular hyperplasia and nonspecific colitis in children with autism spectrum disorders. The possibility of an autism-specific gastrointestinal abnormality, referred to as “autistic enterocolitis,” has often been cited as a potential link in the chain of the “leaky gut” hypothesis (Buie et al. 2010; Erickson et al. 2005; Wakefield et al. 2005; White 2003). An environmental trigger, namely, the MMR (measles, mumps, rubella) vaccine, was suggested but never proven as a potential cause of the pathology in this hypothesis (Christison and Ivany 2006; Erickson et al. 2005; White 2003). Inflammatory responses are further thought to regulate the sulfated glycosaminoglycans (GAGs), which are widely distributed in the vascular and connective tissues of the healthy intestine. Decreased sulfated GAGs have been found in patients with inflammatory bowel disease, a finding which has been hypothesized to allow protein and fluid leakage across the wall of the intestine. Sulfation defects have been noted in children with ASD and could be one potential cause of increased intestinal permeability

(Erickson et al. 2005; White 2003). Further, ingestion of gluten itself has been linked with intestinal permeability as the protein component gliadin has been demonstrated to disrupt intercellular junctions (Liu et al. 2005). A genetic factor has also been implicated in impaired gut integrity in individuals by the finding of increased intestinal permeability in first-degree relatives of patients with inflammatory bowel disease (de Magistris et al. 2010; Liu et al. 2005). Further, a mutation affecting the expression of the gene encoding the MET receptor tyrosine kinase has been associated with both ASD and gastrointestinal conditions and functions in both brain development and gastrointestinal repair by modulating intestinal epithelial cell proliferation (Campbell et al. 2009).

Current Knowledge

Increased intestinal permeability has been linked to several hypotheses regarding the etiology or symptomatology of ASD, including immune dysfunction and increased opiate activity (White 2003). Intestinal permeability was initially reported by D'Eufemia et al. in 1996 on a small sample of children with autism spectrum disorders without gastrointestinal symptoms. This finding was not observed in healthy, typically developing controls. Overall, the literature is mixed in regard to the presence of a “leaky gut.” Two additional studies utilizing neurotypical controls were performed. The first study enrolled 14 children with ASD who had current or previous gastrointestinal symptoms by parental report, 7 unaffected siblings of these children, and 8 unrelated children with typical development. There were no differences detected on the differential sugar absorption test using lactulose and mannitol, as described above (Robertson et al. 2008). However, a recent study of 90 children with ASD and 146 of their first-degree relatives found that 37% and 21%, respectively, had increased intestinal permeability based on the differential sugar absorption test using lactulose and mannitol, compared to 5% of 146 healthy adults and none of the 64 healthy child controls. Remarkably, increased intestinal permeability was not associated with intestinal

inflammation when measured by fecal calprotectin in this study. Calprotectin is a protein in the intracellular fluid of neutrophils, which has been used as a noninvasive marker of inflammation in inflammatory bowel disease (de Magistris et al. 2010). These findings suggest that altered intestinal permeability may be a real finding in autism but may not be linked to intestinal inflammation.

Concern regarding an interactional relationship between factors affecting the functioning of the gastrointestinal system and that of the central nervous system underlying the etiology of ASD is based in part on reports of increased gastrointestinal dysfunction in children with autism spectrum disorders. To date, there is no conclusive evidence that the prevalence of gastrointestinal disturbance is elevated in individuals with autism above the general population. The reports of prevalence range from 9%, similar to the typical population, to 84%, most likely secondary to variations in methodology (Buie et al. 2010). Two recent studies again show mixed results. One recent population-based study examined the medical records of children with ASD and age/gender-matched controls using data from the Rochester Epidemiology Project, which captures detailed medical charts from more than 95% of the residents of Olmsted County, Minnesota. Their findings indicate that the overall cumulative incidence of gastrointestinal symptoms in children with ASD was not increased above controls, although there was a statistically significant increase in the cumulative incidence of constipation and feeding selectivity in children with ASD above controls. It is unclear whether the increase in constipation and food selectivity was due to gastrointestinal or neurobehavioral differences. The retrospective nature of data collection, with data being collected from medical records completed in the past, and the homogeneity of the racial and ethnic composition of Olmsted County (i.e., 98% of the population is reported to be white) limit the findings (Ibrahim et al. 2009). Another recent study is the only report to date to examine gastrointestinal problems in children with autism spectrum disorders from families with multiple affected members. Via a structured medical history interview,

data regarding gastrointestinal problems was collected on 589 children with ASD, verified by gold standard measures, and 163 sibling controls from 313 families. The authors found increased parental report of gastrointestinal problems in children diagnosed with an autism spectrum disorder (42%) versus their unaffected siblings (12%), with chronic diarrhea and constipation the most frequently reported. Inclusion of a novel severity ranking scale revealed increased odds of gastrointestinal problems with increased severity of ASD symptoms, a finding that will require replication. The retrospective nature of the data collection, largely relying on recall, limited the findings. Similarly, the inclusion of unaffected siblings may have introduced an additional bias as the children with ASD may have had increased medical visits and scrutiny leading to an increased identification of symptoms over their typically developing siblings (Wang et al. 2011).

The specificity of the endoscopic and histologic findings in the intestine, namely, ileal lymphoid nodular hyperplasia (LNH), to autism has been controversial, particularly as LNH has been noted in typically developing children with constipation and food allergy (Buie et al. 2010; Erickson et al. 2005). Other methodological concerns, including inappropriate control groups and lack of validated definitions, make the findings difficult to interpret (Buie et al. 2010). The connection between autism and the MMR vaccination has since been disproven (Hornig et al. 2008). The original article describing the endoscopic and histologic findings and the temporal relationship of the onset of symptoms of autism to injection of the MMR vaccine has now been retracted due to false claims regarding how the study was conducted (Harris 2010; Wakefield et al. 2010). Although inflammation of the gastrointestinal tract has been noted in children with autism spectrum disorders (Erickson et al. 2005; Horvath and Perman 2002), an autism-specific abnormality has not been established (Buie et al. 2010).

There is also conflicting evidence regarding the opiate excess theory. Studies examining the presence of excess opioid-like compounds in the cerebrospinal fluid and urine of individuals with ASD have revealed no consistent patterns (Cass et al.

2008; Ibrahim et al. 2009; White 2003). The clinical trials examining the efficacy of opiate antagonists in the treatment of ASD reported, at most, small benefit, principally in hyperactivity and self-injurious behaviors (Christison and Ivany 2006; Horvath and Perman 2002). Further, dietary elimination studies of gluten and casein showed mixed results. To date, there are only a few published randomized control trials (which are the most reliable form of scientific evidence in intervention studies) of gluten and casein elimination diets. The results are mixed with some general improvements noted on the diet in two of the three studies. However, methodological issues render the results difficult to interpret (Millward et al. 2009; Whitley et al. 2010). One small study was presented at the International Meeting for Autism Research in 2010 and reported on wheat and milk challenges in children with ASD placed on a gluten and casein elimination diet, finding no significant difference in outcome measures. While this was a randomized, blinded trial, the sample size was small (Hyman et al. 2010). Consequently, there is no current clear evidence on whether the gluten-free, casein-free diet is effective in ameliorating symptoms of ASD.

Research regarding a genetic predisposition involved in increased intestinal permeability in individuals with ASD is still preliminary. A recent study extended the findings of increased intestinal permeability to first-degree relatives of individuals with ASD, implying a genetic factor may be involved (de Magistris et al. 2010). The work regarding the gene encoding the MET receptor tyrosine kinase may imply that genetic factors contribute separately to gastrointestinal and neurobehavioral symptoms in individuals with ASD (Campbell et al. 2009). However, additional studies are needed before any firm conclusions can be made.

In conclusion, increased intestinal permeability has been documented in certain medical conditions such as inflammatory bowel disease and celiac disease. It has been hypothesized to play a role in psychiatric disorders such as schizophrenia. Further, intestinal permeability has been reported to be increased in children with autism spectrum disorders leading to etiologic theories,

including the leaky gut-opioid excess theory. To date, there is preliminary supportive evidence for altered intestinal permeability in children with autism spectrum disorders. Conclusions regarding whether this potential difference plays a role in the gut-brain axis and is therefore either causative of autism spectrum disorders or increases symptoms of ASD cannot be made. At this time, there is very little data to support the opioid excess theory or a gastrointestinal pathology specific to ASD.

Future Directions

Large prospective studies with appropriate control groups that take into account factors such as dietary intake and comorbid psychiatric conditions (e.g., anxiety and depression), which may play a role in functional gastrointestinal problems, are needed to further characterize gastrointestinal differences in children with ASD. Research targeting potential interventions in autism spectrum disorders need to encompass large, properly controlled trials with well-defined outcome measures. It is also important that researchers studying gastrointestinal abnormalities and intestinal permeability adequately characterize the sample with appropriate autism diagnostic measures. Given the heterogeneity of ASD, describing the subjects in terms of both gastrointestinal symptoms/disorders using standardized definitions and the developmental/behavioral phenotype may shed light on the difference between children with and without altered intestinal permeability.

See Also

- [Gastrointestinal Disorders and Autism](#)
- [Measles and Autism](#)

References and Reading

- Buie, T., Campbell, D. B., Fuchs, G. J., Furuta, G. T., Levy, J., Van de Water, J., et al. (2010). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125, S1–S18.

- Campbell, D. B., Buie, T. M., Winter, H., Bauman, M., Sutcliffe, J. S., Perrin, J. M., et al. (2009). Distinct genetic risk based on association of MET in families with co-occurring autism gastrointestinal conditions. *Pediatrics*, 123, 1018–1024.
- Cass, H., Gringas, P., March, J., McKendrick, I., O'Hare, A. E., Owen, L., et al. (2008). Absence of urinary opioid peptides in children with autism. *Archives of Disease in Childhood*, 93, 745–750.
- Christison, G. W., & Ivany, K. (2006). Elimination diets in autism spectrum disorders: Any wheat amidst the chaff? *Journal of Developmental and Behavioral Pediatrics*, 27, S162–S171.
- D'Eufemia, P., Celli, M., Finocchiaro, R., Pacifico, L., Viozzi, M., Zaccagnini, M., et al. (1996). Abnormal intestinal permeability in children with autism. *Acta Paediatrica*, 85, 1076–1079.
- de Magistris, L., Familiari, V., Pascotto, A., Sapone, A., Froli, A., Iardino, P., et al. (2010). Alterations of the intestinal barrier in patients with autism spectrum disorders and their first degree relatives. *Journal of Pediatric Gastroenterology and Nutrition*, 51, 418–424. <https://doi.org/10.1097/MPG.0b013e3181dcc4a5>.
- Dohan, F. C. (1988). Genetic hypothesis of idiopathic schizophrenia: Its exorphin connection. *Schizophrenia Bulletin*, 14, 489–494.
- Erickson, C. A., Stigler, K. A., Corkins, M. R., Posey, D. J., Fitzgerald, J. F., & McDougle, C. J. (2005). Gastrointestinal factors in autistic disorder: A critical review. *Journal of Autism and Developmental Disorders*, 35, 713–727.
- Harris, G. (2010). Journal retracts 1998 paper linking autism to vaccines. *The New York Times*. Retrieved from July 2011. <http://www.nytimes.com>
- Hornig, M., Bries, T., Buie, T., Bauman, M. L., Lauwers, G., Siemietzki, U., et al. (2008). Lack of association between measles virus vaccine and autism with enteropathy: A case-control study. *PLoS One*, 3(9), e3140. <https://doi.org/10.1371/journal.pone.0003140>.
- Horvath, K., & Perman, J. A. (2002). Autism and gastrointestinal symptoms. *Current Gastroenterology Reports*, 4, 251–258.
- Hyman, S., Stewart, P. A., Smith, T., Foley, J., Cain, U., Peck, R., et al. (2010). *The gluten free and casein free (GFCF) diet: A double blind, placebo controlled challenge study*. Platform presentation presented at International Meeting for Autism Research. Retrieved from <http://imfar.confex.com/imfar/2010/webprogram/Paper6183.html>
- Ibrahim, S. H., Voigt, R. G., Katusic, A. K., Weaver, A. L., & Barbaresi, W. J. (2009). Incidence of gastrointestinal symptoms in children with autism in Olmsted County, Minnesota, 1976–1997: A population-based study. *Pediatrics*, 124, 680–686.
- Liu, Z., Li, N., & Neu, J. (2005). Tight junctions, leaky intestines, and pediatric diseases. *Acta Paediatrica*, 94, 386–393.
- Millward, C., Ferriter, M., Calver, S. J., & Connell-Jones, G. G. (2009). Gluten- and casein-free diets for autistic spectrum disorder. *The Cochrane Database of Systematic Reviews*, 2. <https://doi.org/10.1002/14651858.CD003498.pub3>.
- Panksepp, J. (1979). A neurochemical theory of autism. *Trends in Neurosciences*, 2, 174–177.
- Robertson, M. A., Sigalet, D. L., Holst, J. J., Meddings, J. B., Wood, J., & Sharkey, K. A. (2008). Intestinal permeability and glucagon-like peptide-2 in children with autism: A controlled pilot study. *Journal of Autism and Developmental Disorders*, 38, 1066–1071.
- Wakefield, A. J., Ashwood, P., Limb, K., & Anthony, A. (2005). The significance of ileocolonic lymphoid nodular hyperplasia in children with autism spectrum disorder. *European Journal of Gastroenterology and Hepatology*, 17, 827–836.
- Wakefield, A. J., Murch, S. H., Anthony, A., Linnell, J., Casson, D. M., & Malik, M. (2010). Retraction—ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *The Lancet*, 351, 375–445.
- Wang, L. W., Tancredi, D. J., & Thomas, D. W. (2011). The prevalence of gastrointestinal problems in children across the United States with autism spectrum disorders from families with multiple affected members. *Journal of Developmental and Behavioral Pediatrics*, 32, 1–10.
- White, J. F. (2003). Intestinal pathophysiology in autism. *Experimental Biology and Medicine*, 228, 639–649.
- Whitely, P., Haracopos, D., Knivsberg, A. M., Reichelt, K. L., Parlar, S., Jacobsen, J., et al. (2010). The ScanBrit randomised, controlled, single-blind study of a gluten- and casein-free dietary intervention for children with autism spectrum disorders. *Nutritional Neuroscience*, 13, 87–100.

Learned Helplessness

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Definition

A state in which animals and humans act as if events are outside of their control, and so do not try to escape from unpleasant conditions that they could otherwise easily avoid. This state is created by being repeatedly exposed to an aversive stimulus from which escape is impossible, and this state is then generalized to other stimuli. It is believed to contribute to depression and other related conditions.

Historical Background

The phenomenon of learned helplessness was described following a series of experiments led by Seligman and his colleagues beginning in 1967, first involving dogs and rats (Seligman 1968), and then involving humans (Miller and Seligman 1973). In humans, learned helplessness was manifested as a perception that events were outside of the individual's control. This characterization of depressions was central to interventions which subsequently focused on changing cognitions surrounding the individual's perception of themselves, their actions, and the future. A database search of the key terms "autism" and "learned helplessness" revealed very few references to both terms, and so we have integrated past and present discussions related to autism and developmental disabilities under "[Current Knowledge](#)."

Current Knowledge

Learned helplessness was first discussed more extensively in the peer-reviewed literature on ASD when speaking to the challenge of motivating children exposed to repeated failure. Behavior analysts had postulated that the increased opportunity to access reinforcement that results from correct completion of a task should motivate the child to devote increased effort to completing other tasks. Koegel and Egel (1979) provided perhaps the earliest and most specific test of this hypothesis. They examined the level of interest and persistence evident in three young children with autism when presented with a series of fine motor tasks. Using a multiple baseline design, performance was compared under conditions of no prompts (pretreatment condition) and verbal and gestural prompts (treatment condition). During the pretreatment condition, children had low levels of task completion and interest, and decreasing attempts over time. In contrast, during posttreatment conditions, when prompting levels were comparable to pretreatment conditions, two of the three children demonstrated sustained improvements in interest and in the number of

attempts relative to pretreatment levels. Subsequent trials and conditions for the third child revealed that these improvements were only evident when he was successful at least 50% of the time; otherwise, attempts dropped dramatically in frequency. The authors framed these findings as demonstrating learned helplessness: Low rates of success masked the potential relationship between effort and outcome or, in behavioral terms, between response and reinforcement. Creating conditions for success made the response-reinforcement relationship more salient, and thus made children more likely to persist when initially unsuccessful. The potential disconnection between response and reinforcement was striking for the third child, for whom high levels of reinforcement were at times almost unrelated to effort. As the oldest of the three participants, he was the most likely to have experienced learned helplessness.

The Koegels returned to the role of learned helplessness at later points (Koegel and Mentis 1985). They suggested that teaching self-management skills to persons with ASD and other disabilities may help them to better understand the relationship between their own behavior and outcomes, and inoculating them against learned helplessness (Koegel et al. 1995). They also postulated that learned helplessness was central to understanding the potential effectiveness of pivotal response training or PRT (Koegel et al. 2001): "pivotal response interventions that emphasize relations between social communicative responses and their positive consequences appears to increase motivation to respond, thereby improving responsivity and increasing favorable environmental and social stimulation and interaction." (Koegel et al. 2001, p. 21). Learned helplessness may be a natural outcome of other difficulties faced by children with autism (e.g., the difficulty processing complex stimuli) within the first 18 months of life. Instead of seeking to initiate interactions with the environment, fueled by a response-reinforcer relationship growing in strength with each success, children with autism withdraw, effectively decreasing the number and type of learning opportunities available to them. The potential for disruption of the

response-reinforcer relationship may be particularly evident with respect to the deficits in social communicative interactions that are a hallmark of autism. The social and communicative exchanges that naturally emerge in exchanges between typical toddlers and adults (i.e., in the form of requesting, joint attention, social play, and other socio-affective interactions, etc.) simply do not occur in many children with autism or, if they do, only emerge later in development and at much lower rates. By the time autism is identified and intervention begins, the lack of persistence in the face of failure has already had a cascading effect on the number and type of learning opportunities available to the child with autism, especially with respect to social communicative interactions. Yet, just as learned helplessness can disempower the young child with autism, PRT can function (among other things) to reestablish the response-reinforcer relationship. The emphasis in PRT on self-initiation and self-management, on adaptations to help children respond to multiple cues and on the increase in motivation more generally, has an impact on the specific skills being taught, and on the child's perception of themselves as an effective agent in their interactions with their environment.

Learned helplessness has also been discussed in the context of depression and ASD. Barnhill and Myles (2001) found the same relationship between attributional style and depression among adolescents with Asperger syndrome, as is evident in other populations, namely, that depression is associated with a style in which the person blames themselves for negative events and outcomes, across contexts and time. These authors also suggested that positive behavior supports, which emphasize proactive interventions to prevent errors, may help to counter this negative attributional style.

Future Directions

At present, it is unclear how the relationship between learned helplessness and autism will be considered by future researchers and clinicians. The recognition of PRT as an evidence-based

practice, and of depression as an area of concern for adolescents and adults with ASD, suggests that an interest in learned helplessness may be rekindled among researchers and clinicians seeking to help all persons affected by ASD. Researchers and clinicians may also consider revisiting early research suggesting that learned helplessness is related to intellectual disability (Weisz 1979), and may be influenced by adult expectations and feedback (Raber and Weisz 1981; Weisz 1981).

See Also

- [Depressive Disorder](#)
- [Motivation](#)
- [Pivotal Response Training](#)
- [Reinforcement](#)
- [Reinforcer](#)

References and Reading

- Barnhill, G. P., & Myles, B. S. (2001). Attributional style and depression in adolescents with Asperger syndrome. *Journal of Positive Behavior Interventions*, 3, 175–182.
- Koegel, R. L., & Egel, A. L. (1979). Motivating autistic children. *Journal of Abnormal Psychology*, 88, 418–426.
- Koegel, R. L., & Mentis, M. (1985). Motivation in childhood autism: Can they or won't they? *Journal of Child Psychology and Psychiatry*, 26, 185–191.
- Koegel, R. L., Koegel, L. K., & Parks, D. R. (1995). "Teach the individual" model of generalization. In R. L. Koegel & L. K. Koegel (Eds.), *Teaching children with autism: Strategies for initiating positive interactions and improving learning opportunities*. Baltimore: Paul H. Brookes.
- Koegel, R. L., Koegel, L. K., & McNerney, E. K. (2001). Pivotal areas in intervention for autism. *Journal of Clinical Child Psychology*, 30, 19–32.
- Miller, W. R., & Seligman, M. E. (1973). Depression and the perception of reinforcement. *Journal of Abnormal Psychology*, 82, 62–73.
- Raber, S. M., & Weisz, J. R. (1981). Teacher feedback to mentally retarded and nonretarded children. *American Journal of Mental Deficiency*, 86, 148–156.
- Seligman, M. E. (1968). Chronic fear produced by unpredictable electric shock. *Journal of Comparative and Physiological Psychology*, 66, 402–411.
- Weisz, J. R. (1979). Perceived control and learned helplessness among retarded and nonretarded children:

A developmental analysis. *Developmental Psychology*, 15, 311–319.

Weisz, J. R. (1981). Learned helplessness in Black and White children identified by their schools as retarded and nonretarded: Performance deterioration in response to failure. *Developmental Psychology*, 17, 499–508.

Learning Disabilities

► Learning Disorders

Learning Disability

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Synonyms

Language-based learning disability; Learning disorder; Specific learning disability; Specific learning disorder

Short Description or Definition

Learning disability is a term used to describe a heterogeneous group of disorders characterized by significant challenges in acquiring and using listening, speaking, reading, writing, reasoning, and/or mathematical abilities. A learning disability is considered intrinsic to the individual and is related to underlying neurological differences. Although it can co-occur with other disabilities and can be affected by environmental influences, it is not the direct result of these conditions or influences. The National Joint Committee on Learning Disability (NJCLD) has also used the term to represent the discrepancy that might exist between an individual's capacity to learn and their actual achievement level (National Research Center on Learning Disability 2010). Children with learning

disabilities have unique educational needs because they approach learning in different ways. Learning disabilities often have a language basis, and some children may be identified as having a language-based learning disability (Sun and Wallach 2014).

The term learning disability is not used in the DSM-5 (American Psychiatric Association [APA] 2013). Instead, specific learning disorders is the term used to characterize challenges in the acquisition and use of one or more areas of academic performance. These challenges must persist for at least 6 months whether or not intervention has been provided. Considering the most recent research, the DSM-5 expands the definition somewhat to account for persistent difficulties in reading, writing, arithmetic, and/or mathematical reasoning during formal schooling. Children with specific learning disorders are likely to approach reading in a slow and effortful manner with frequent inaccuracies (APA 2013; National Center for Learning Disabilities 2014). Their ability to understand what they read might also be compromised. Writing lacks clarity and organization, while solving mathematical problems and remembering number facts are difficult. Academic skills fall well below the expected level for a child's chronological age and cannot be explained by other developmental, neurological, vision, hearing, or motor disorders (APA 2013; NCLD 2014).

Categorization

There are several descriptions of the varying categories and classifications of learning disabilities. Learning disabilities are often grouped by an area of skill or cognitive weakness and are typically the result of challenges in the ability to process information in spite of intelligence in the average or above average range. Information processing is critical to learning and requires an ability to take in and decode information (input), integrate that information into what a person already knows or is experiencing, storing that information so it can be easily retrieved to facilitate future understanding, and encoding a response (output). Potential

Learning Disability, Table 1 Information processing challenges for individuals with learning disabilities

Levels of information processing	Learning issues for individuals with learning disabilities
Input level	Difficulty with visual perception causing recognition problems
	Sequencing challenges affecting temporal processing
	Challenges in auditory perception impacting ability to determine the relevance of auditory stimuli
	Problems processing tactile information
Integration level	Difficulty telling a story in sequence
	Problems remembering rote information like days of the week
	Challenges in generalizing new learning
	Problems with comprehension
Storage level	Problems with short- and long-term memory or working memory challenging new learning
	Difficulty with visual memory affecting spelling
Output level	Challenges in the formulation of words, gestures, writing, or drawing
	Difficulty with spoken language including answering questions on demand, retrieving vocabulary, organizing thoughts prior to speaking or writing
	Problems with motor output affects written language, physical activity (e.g., riding a bicycle), and daily living or self-help skills (e.g., tying shoes)

challenges in each of these four areas that affect learning are summarized in Table 1.

The DSM-IV-TR and the DSM-5 (APA 2000, 2013) specifically identify three disorder areas that would be considered under the category of learning disabilities or learning disorders (see Table 2 for a description). A diagnosis in any one or more of these categories would qualify a student for support indicating they cannot access, interpret, and formulate learning in an expected manner. The DSM-5 (APA 2013) also describes three severity levels (mild, moderate, severe) for children with learning disorders, highlighting likely difficulties and the level of intervention support required.

Although not part of the learning disorders described in the DSM-5, nonverbal learning disability (NLD) is another category that has often been used to describe students with a specific profile of learning strengths and deficits. Rourke suggests NLD represents a cluster of neuropsychological, academic, and social emotional characteristics reflecting deficiencies in nonverbal processing and reasoning (Rourke and Tsatsanis 1996; Rourke et al. 2002). Generally, children with NLD exhibit assets in repetitive motor skills, learning through repetition and the auditory modality, verbal memory, receptive language,

phonemic awareness, word decoding, and spelling. Deficits, however, are notable in visual discrimination, visual-spatial organization, problem solving, pragmatics, mechanical math, reading comprehension, and social perception. Their challenges in social judgment, interaction, and emotion recognition compromise their abilities to maintain relationships with peers (Volden 2004).

Epidemiology

The National Institutes of Health and Centers for Disease Control and Prevention (2011) report about 15% of Americans, or one in seven, have some type of learning disability. The actual prevalence across academic areas ranges from 5 to 15% for school-age children (American Psychiatric Association 2013) with all cultures and language groups affected. More recent data for the 2013–2014 school year from the National Center for Education Statistics indicates more than 13% of school children (ages 3–21) are receiving special education services. Of all children currently receiving services, 35% have learning disabilities, and a majority of those children have reading as their primary academic challenge (Moats and Lyon 1993; U.S. Department of Education 2001;

Learning Disability, Table 2 Summary of learning disorders as described in the DSM-IV-TR and the DSM-5

Learning disorder	Description	Other terms used
Reading disorder	Difficulty with letter and word recognition	Dyslexia or developmental dyslexia – typically used to represent difficulties in word recognition and not the broader areas described for reading disorder which includes reading comprehension
	Poor phonemic awareness or phonological awareness (e.g., the ability to break up words into sounds, matching letters to sounds)	
	Difficulty understanding words and ideas or reading comprehension	
	Slower reading speed and poorer fluency	
	Weak vocabulary skills	
Mathematics disorders	Difficulties learning math concepts (such as quantity, place value, and time)	Dyscalculia – difficulty understanding math problems and time concepts, and using money
	Problems remembering math facts	
	Challenges organizing numbers and math problems	
Disorder of written expression	Difficulties with handwriting, spelling, organization of ideas, and composition	Dysgraphia – difficulty with handwriting; problems forming letters or writing within a defined space
Learning disorder not otherwise specified	May include problems in reading, math, and written expression that interfere with academic achievement even though test performance is not below age, IQ, or education level	

2015). Learning disabilities often have a familial risk. There appears to be equal gender representation with as many girls as boys having a learning disability, although the actual number of boys versus girls being served is different with more boys identified for and receiving services than girls (Bandian 1999; Coutinho and Oswald 2005).

Natural History, Prognostic Factors, and Outcomes

The learning disabilities field evolved from the influence of professionals and parents who were seeking an educational philosophy that would be responsive to the learning style and unique strengths and challenges of individual children (Smith 1998). The desired focus was that teachers would emphasize what a child could do and not just what they could not do. Through a variety of advocacy efforts (at both a federal level and a parent and professional organization level), a new disability category emerged in special education. Federal legislation giving all children the right to an equal education stimulated growth in

the field, and the enactment of P.L. 94–142 in 1975 assured special services for all children with disabilities. This was the first time children with learning disabilities were among those identified as disabled (Smith 1998). A reenactment of P.L. 94–142 in 1990 as the Individuals with Disabilities Education Act (IDEA; PL 101–476) advanced special services for students with disabilities, and the workforce saw growth in teachers prepared in learning disabilities as well as universities developing training programs in learning disabilities. A reauthorization of IDEA in 2004, now called the Individuals with Disabilities Education Improvement Act (IDEIA), offered an alternative approach to identifying children with learning disabilities. In the past, a learning disability was determined by an assessed discrepancy between achievement and intellectual ability in reading or reading comprehension, oral or written expression, mathematical calculation or reasoning, or listening comprehension. A change was made because of a lack of evidence that the IQ-achievement discrepancy formulas are consistently and reliably applied. Further, this approach has been problematic for students in poverty or

those with culturally and linguistically different backgrounds. The alternative approach to identifying whether or not a child has a learning disability is a response to intervention approach. This approach incorporates an evaluation of how a child responds to scientifically based intervention before making a determination if, in fact, the child has a learning disability.

Research has examined potential predictors of learning disability, including motivation (e.g., goal orientation, self-efficacy, self-concept, goal commitment, motivational force), metacognition (i.e., one's ability to monitor and control one's own learning), and psychopathology (e.g., depression and anxiety) (Sideridis et al. 2006). Results indicate that motivation is highly accurate in identifying students with or at risk for a learning disability with metacognition and psychopathology as strong predictors. Understanding these processes may facilitate our ability to more accurately screen and treat students with LD.

Although the causes of learning disabilities are unclear, it is noteworthy that learning disabilities tend to run in families. Pregnancy problems and difficulties at birth, anomalies in brain development, exposure to toxins, and accidents following birth are some examples of potential causal factors. A diagnosis of a learning disability has a significant impact on the family as it requires an understanding of the unique needs of the child and an ability to manage the learning, social, and behavioral challenges that often accompany the disorder. Learning disabilities impact an individual's ability to manage across their lifespan, and effective coping methods are critical.

Recent gains have been made in brain research examining the neuroplasticity of the brain and the role of brain-behavior findings in our understanding of disorders such as learning disabilities. The brain's ability to change and create new connections encourages applied research that examines the impact of evidence-based treatments on learning and increasing neuronal connections. In the right learning contexts, the brain might have an ability to reorganize old connections and create new connections that increases the ease of learning to read, write, or complete math calculations.

Clinical Expression and Pathophysiology

It is important to pay attention to normal developmental milestones for toddlers and preschoolers as early identification of developmental differences may be an early sign of a learning disability. Individuals with learning disabilities do not constitute a homogeneous group as the observed deficits occur in one or more domains of academic achievement including reading, math, and written expression (Fletcher et al. 2007). To understand the clinical expression and pathophysiology of a learning disability, it is important to recognize the potential influences on outcomes, which have variable expression. For example, there are neurobiological factors such as genetic profiles and brain structure and function differences that influence expression of the disorder. Environmental factors such as socioeconomic status, the amount and level of schooling, as well as the types of interventions received will affect outcomes. In addition, core cognitive processes (e.g., phonemic awareness) and behavioral/psychosocial factors (e.g., motivation, anxiety, attention) will have an impact on the characterization of the disorder for individual students. Thus, the traits that are described for learning disability or learning disorder exist on a continuum.

In the preschool years, some potential markers of a learning disability might be delayed speech and language, poor vocabulary growth, difficulty with phonological awareness (e.g., rhyming words, learning numbers, and letters), trouble following directions, difficulty with peers, and slow fine motor development. During early elementary school, some warning signs might be slower ability to connect sounds and letters, confusion with basic concepts, consistent reading and spelling errors, reversing letters or numbers, difficulty remembering math facts and telling time, and poor coordination. In the later grades, letter sequence reversals, difficulty with morphology (e.g., prefixes, suffixes, root words) and handwriting, avoidance of reading aloud and writing, poor recall, and difficulty making friends are likely behaviors.

Early intervention can reduce the number of children who qualify as learning disabled

(Sternberg and Grigorenko 1999, 2001). Although the provision of classroom accommodations supports the learning environment for students with learning disabilities, Sternberg suggests that schools often fail to recognize children's range of strengths and challenges, and instructional approaches may not be well suited to children's different learning styles. An integrated approach to identifying and supporting the needs of individuals with learning disability is recommended (Bradley et al. 2002; Fletcher et al. 2007). This approach considers the value of a response to instruction model for identifying children at risk, evaluating instructional quality and monitoring progress over time. It also identifies what to do when an inadequate response to instruction occurs. An integrated approach defines a path for more formal assessment in the learning domains as well as a comprehensive evaluation that probes potential language, social, and home factors that could produce underachievement (Bradley et al. 2002).

Evaluation and Differential Diagnosis

The process of diagnosing a learning disability is challenging as it requires not only accurate testing but also comprehensive data collection through a variety of sources. It involves testing, history taking, and observation by a trained specialist. Several professionals may be involved in the evaluation process, including a psychologist or neuropsychologist, an occupational therapist, a speech-language pathologist, and a special educator to name a few. During the diagnostic process, a number of professionals may be involved in coordinating efforts to obtain an accurate diagnosis which includes input from teachers, parents, and the student.

Currently, two approaches are used to identify learning disabilities: the IQ-achievement discrepancy approach and the response to intervention approach (RTI). In the IQ-discrepancy approach, intelligence and academic achievement testing, classroom performance, and social interaction are assessed. In addition, some students may receive speech-language and attention testing.

Assessment results determine if a student's academic performance is commensurate with cognitive ability. If cognitive ability or potential (as measured on an intelligence test) is well above a student's academic performance, then a diagnosis of a learning disability is made. Recently, however, this approach to diagnosis has been criticized because there is little evidence that the measured discrepancy is an indication of LD nor does it predict treatment effectiveness (Aaron 1995; Barnes et al. 2007; Harrison and Flanagan 2005). In the response to intervention (RTI) approach, the diagnostic process is more treatment oriented. All students are screened early on, and those demonstrating some difficulty receive research-based instruction prior to any identification of learning disability. Performance is monitored to determine if providing increasingly intense intervention facilitates the student's progress. Those who respond well to this intensive research-based instruction will not require further intervention or diagnosis, while those who do respond to regular classroom instruction (Tier 1 support) or intensive instruction (Tier 2 support) are referred to special education and are usually diagnosed with a learning disability. A benefit to this diagnostic approach is there is no waiting for the student to fail prior to receiving assistance. The 2004 reauthorization of the Individuals with Disabilities Education Act permitted states and school districts to use RTI as a method of identifying students with learning disabilities. Notably, this process does not consider individual neuropsychological factors that are often used to design instruction, it takes longer to implement the traditional approaches to assessment, it requires implementation of a strong intervention program, and it is driven by general education versus special education (Fletcher et al. 2007).

In the traditional approach to evaluation and diagnosis, several normed assessment tools are used to evaluate a student's academic performance and achievement. The primary academic domains that are assessed include reading, math and written expression, and achievement tests such as the *Woodcock-Johnson 4th edition* (WJ IV), the *Wechsler Individual Achievement Test 3rd edition*, the *Wide Range Achievement*

Test 3rd edition, and the *Stanford Achievement Test 10th edition*. Specialized reading assessment might also include *Gray's Diagnostic Reading Tests 2nd edition*, the *Stanford Diagnostic Reading Assessment 4th edition*, the *Comprehensive Test of Phonological Processing*, *Tests of Oral Reading and Comprehension Skills*, the *Test of Reading Comprehension 4th edition*, the *Test of Word Reading Efficiency 2nd edition*, and the *Test of Silent Contextual Reading Fluency 2nd edition*. Comprehensive assessment is certainly a requirement for intervention as well as helping to determine the contexts in which difficulties are experienced, including the coexistence of language and behavioral challenges.

It is important to recognize that other disorders are often confused with learning disorders and some may co-occur. Typically, those individuals with an intelligence level below 70 on a standardized intelligence test would be characterized as having an intellectual disability (formerly categorized as mental retardation) and would not also be diagnosed with a learning disability, although learning will be comprised for this population because of the identified cognitive impairment.

Attention deficit hyperactivity disorder (ADHD) and receptive and expressive language disorders often co-occur with learning disabilities, although these disorders are not included in the standard definition of learning disabilities. A child with an attention problem often struggles with

learning because of their inability to sufficiently focus on relevant stimuli and organize their responses. A child with a language disorder has difficulties at the core of learning disabilities, affecting listening comprehension and oral and written expression as well as pragmatic language function. All three disorders share executive function challenges and impact an individual's ability to communicate. These shared clinical manifestations complicate differential diagnosis.

Children with learning disabilities are also likely to have language and social challenges. For example, a nonverbal learning disability has a significant impact on an individual's ability to "read" facial and gestural cues during social interaction (Manoach et al. 1995; Rourke 1989). This can lead to odd and poor social behavior. Research is ongoing to determine the overlap or separation of NLD and ASD (Rourke 2000).

Treatment

National Institutes of Health (NIH) research suggests that 67% of students considered at risk for reading failure achieve average or above average reading performance following intervention in the early grades. As previously described, a new approach or conceptual model for intervention (i.e., response to intervention) is being applied to students with a learning disability so that a school

Learning Disability, Table 3 Examples and descriptions for interventions for children with learning disabilities

Examples of interventions	Description
Direct instruction (National Institute for Direct Instruction 2007)	Highly structured, individualized, and intensive instruction usually one-on-one occurring on a daily basis; lessons are preplanned to ensure small learning successes and identifying, and correcting mistakes immediately; progress monitoring is ongoing
Environmental arrangement or classroom adjustments	Modified assignments and testing procedures, preferential seating, quiet space to study and take tests, increased time for assignment or testing completion
Special equipment or learning materials	Access to word processors with spell checkers, voice output devices that use text to speech and speech to print programs, calculators, books on tape, etc.
Classroom supports	Notetakers, readers, proofers, scribes, instructional assistants, or paraeducators to support on task behavior and vigilant responsiveness
Special education	Specified hours or placement in a resource room, enrollment in an LD program, IEP, and related therapies
Speech-language pathologist	Addresses gaps in learning such as reading comprehension, pragmatics, written language

team can determine if a child responds to scientific, research-based instruction as early as possible. A primary focus of RTI is to identify the instructional challenges resulting from the learning disability and then implement high-quality instruction by well-trained educators to differentiate those children with a true disability and those with learning differences.

Treatment for individuals with LD involves a partnership with all team members including a case manager who implements the assessment process and coordinates the student's program; a special educator with credentials to support the specific educational needs of the student; a neurologist to address concerns about brain function and the implications on learning; a speech-language pathologist who provides support for speech, language, and social communication difficulties; and a psychologist who assesses psychological and intellectual development and guides treatment for behavior difficulties and mental and emotional health.

Several interventions are described in the literature, and several examples are summarized in Table 3.

See Also

- [Learning Disorders](#)
- [Nonverbal Learning Disabilities \(NLD\)](#)

References and Reading

- Aaron, P. G. (1995). Differential diagnosis of reading disabilities. *School Psychology Review*, 24(3), 345–360.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.). Washington, DC: APA Press.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual* (5th ed., Text Rev.). Washington, DC: APA Press.
- Bandian, N. A. (1999). Reading disability defined as a discrepancy between listening and reading comprehension: A longitudinal study of stability, gender differences, and prevalence. *Journal of Learning Disabilities*, 32(2), 138–148.
- Barnes, M. A., Fletcher, J., & Fuchs, L. (2007). *Learning disabilities: From identification to intervention*. New York: The Guilford Press.
- Bradley, R., Danielson, L., & Hallahan, D. P. (2002). *Identification of learning disabilities: Research to practice*. Mahwah: Erlbaum.
- Centers for Disease Control and Prevention. (2011). National Health Interview Survey 2007–2009 data. Available at <http://www.cdc.gov/nchs/nhis.htm>.
- Coutinho, M. J., & Oswald, D. P. (2005). State variation in gender disproportionally in special education: Finding and recommendations. *Remedial and Special Education*, 26(1), 7–15.
- Fletcher, J. M., Lyon, G. R., Fuchs, L. S., & Barnes, M. A. (2007). Classification, definition and identification of learning disabilities. In *Learning disabilities: From identification to intervention* (pp. 25–63). New York: The Guilford Press.
- Harrison, P. L., & Flanagan, D. P. (2005). *Contemporary intellectual assessment: Theories, tests, and issues*. New York: The Guilford Press.
- Manoach, D., Sandson, T., & Weintraub, S. (1995). The developmental social-emotional processing disorders is associated with right hemisphere abnormalities. *Neuropsychiatry, Neurophysiology, Behavioral Neurology*, 8, 99–105.
- Moats, L. C., & Lyon, G. R. (1993). Learning disabilities in the United States: Advocacy, sciences, and the future of the field. *Journal of Learning Disabilities*, 26, 282–294.
- National Center for Learning Disabilities. (2014). *The state of learning disabilities* (3rd ed.). New York: Author.
- National Institute for Direct Instruction. (2007). PO Box 11248, Eugene, OR 97440–3448. Retrieved 2007.
- National Research Center on Learning Disabilities. (2010). Retrieved 2010, www.nrcld.org.
- Rourke, B. P. (1989). *Nonverbal learning disabilities: The syndrome and the model*. New York: The Guilford Press.
- Rourke, B. P. (2000). Nonverbal learning disabilities and Asperger syndrome. In A. Klin, F. R. Volkmar, & S. S. Sparrow (Eds.), *Asperger syndrome* (pp. 231–253). New York: The Guilford Press.
- Rourke, B. P., & Tsatsanis, K. D. (1996). Syndrome of nonverbal learning disabilities: Psycholinguistic assets and deficits. *Topics in Language Disorders*, 16, 30–44.
- Rourke, B. P., Ahmad, S., Collins, D., Hayman-Abello, B., Sayman-Abello, S., & Warriner, E. (2002). Child clinical/pediatric neuropsychology: Some recent advances. *Annual Reviews of Psychology*, 53, 309–339.
- Sideridis, G. D., Morgan, P. L., Botsas, G., Padelidu, S., & Fuchs, D. (2006). Predicting LD on the basis of motivation, metacognition, and psychopathology: An ROC analysis. *Journal of Learning Disabilities*, 39(3), 215–229.
- Smith, C. R. (1998). *Learning disabilities: The interaction of learner, task, and setting* (4th ed.). Boston: Allyn & Bacon.
- Sternberg, R. J., & Grigorenko, E. L. (1999). *Our labeled children: What every parent and teacher needs to know about learning disabilities*. Reading: Perseus Publishing Group.
- Sternberg, R. J., & Grigorenko, E. L. (2001). Learning disabilities, schooling, and society. *Phi Delta Kappa*, 83, 335–338.

- Sun, L., & Wallach, G. P. (2014). Language disorders are learning disabilities. Challenges on the divergent and diverse paths to language learning disability. *Topics in Language Disorders*, 34(1), 25–38.
- U.S. Department of Education. (2001). *Twenty-third annual report to congress on the implementation of the individuals with disabilities education act*. Washington: Author.
- U.S. Department of Education, Office of Special Education Programs, Individuals with Disabilities Education Act (IDEA) database. (2015). From <http://www2.ed.gov/programs/osepidea/618-data/state-level-data-files/index.html#bcc>; and National Center for Education Statistics, Common Core of Data (CCD), “State Nonfiscal Survey of Public Elementary/Secondary Education,” 2013–14. See *Digest of Education Statistics 2015*, Table 204.30 and Table 204.50. Retrieved September 25 2015.
- Volden, J. (2004). Nonverbal learning disability: A tutorial for speech-language pathologists. *American Journal of Speech-Language Pathology*, 13, 128–141.

Short Description or Definition

According to the DSM-IV-TR, a learning disorder can be diagnosed “...when the individual’s achievement on individually administered, standardized tests in reading, mathematics, or written expression is substantially below that expected for age, schooling, and level of intelligence. The learning problems significantly interfere with academic achievement or activities of daily living that require reading, mathematical, or writing skills.” The legal definition of a learning disorder has remained unchanged since 1977; according to the U.S. Department of Education, a specific learning disability is “a disorder in one or more of the basic psychological processes involved in using language, spoken or written, which...may manifest itself in the imperfect ability to listen, think, speak, read well, or do mathematical calculations.”

Learning Disability (UK)

► Mental Retardation

Learning Disability (United Kingdom)

► Intellectual Disability

Learning Disorder

► Learning Disability

Learning Disorders

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Synonyms

[Learning disabilities](#)

Categorization

In general, educators use the term learning disability while the medical community uses the diagnosis learning disorder. The U.S. Department of Education (1977) defines a learning disability as a severe, unexpected discrepancy between achievement and intellectual capacity in one or more of the following areas: (1) oral expression, (2) listening comprehension, (3) written expression, (4) basic reading skills, (5) reading comprehension, (6) math calculation, or (7) mathematical reasoning. The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR; American Psychiatric Association [APA] 2000) includes the following learning disorders: reading disorder, mathematics disorder, disorder of written expression, and learning disorder not otherwise specified. Diagnosed difficulties with oral expression and listening comprehension would be captured by communication disorder diagnoses: expressive language disorder, mixed receptive-expressive language disorder, phonological disorder, stuttering, or communication disorder not otherwise specified.

Epidemiology

Estimates of the prevalence of learning disorders range from 2% to 10% depending on definitions and how they are applied (APA 2000). According to Gregorenko (2007), prevalence rates for learning disabilities range from 5% to 12% of the total school-age population. When comparing the number of children who are receiving special services with the total school-age population, the prevalence rate is approximately 5–6% of the total school-age population. These estimates indicate that LDs are observed more frequently in boys than in girls and more frequently in underrepresented minority groups than in Asian Americans or whites. This method of estimating prevalence rates is somewhat flawed, however, as it relies upon each state's and school district's approach to identification and service provision for children with learning disorders and the proportion of children served in different states varies widely. According to Gregorenko (2007), estimates derived from research studies suggest that up to 10–12% of school-age children show specific deficits in selected academic domains. Provision of high-quality classroom instruction and supplemental small-group instruction may reduce this number to approximately 6% of children. It is assumed that these children would meet strict criteria for diagnosis of a learning disability.

Natural History, Prognostic Factors, and Outcomes

Historically, children have not been identified as having a specific learning disability until second grade or later. This is, in part, due to the fact that these children have intact cognitive functioning and have not fallen significantly behind academically in terms of age- or grade- based expectations until that time (e.g., have demonstrated an ability-achievement discrepancy). In 2004 with the reauthorization of the Individuals with Disabilities Act (IDEA), states and school districts were provided with the option of diagnosing learning disabilities using the traditional ability-achievement discrepancy model or an alternative

strategy known as response-to-intervention (RTI). RTI is an educational strategy in which all children receive continuous monitoring of academic progress. Children who exhibit difficulties are provided with evidence-based classroom remediation strategies. If these strategies prove to be ineffectual, the child is provided with more intensive, small-group instruction. If the academic difficulties are not remediated at this level of intervention, the child is referred for special education services and considered to have a specific learning disability. Because this strategy does not rely on waiting for a significant ability-achievement discrepancy to emerge before beginning to implement remediation strategies, intervention can begin at an earlier age in order to provide timely evidence-based remediation strategies and in an effort to prevent the development of other learning problems and comorbid disorders.

According to Mash and Wolfe (2005), about three quarters of children identified as having a reading disorder in elementary school still have major reading problems in high school and young adulthood. Children and adolescents with learning disorders are more likely than their peers to evidence both internalizing disorders, such as anxiety and mood disorders, and externalizing disorders, such as attentional or compliance difficulties. The American Psychiatric Association (APA 2000) states that the school dropout rate for adolescents with learning disorders is nearly 40% or approximately 1.5 times the national average. In addition, adults with learning disorders may have difficulty with employment and social adjustment. The APA estimates that 10–25% of individuals with learning disorders also have attention deficit hyperactivity disorder, oppositional defiant disorder, conduct disorder, major depression, or dysthymic disorder.

Many adults with learning disorders are able to find ways to accommodate their disabilities, build upon their strengths, and excel in their fields of endeavor. E.E. Werner, as part of a longitudinal study of all children born in 1955 on the island of Kauai, Hawaii, found that most children with learning disabilities she followed adapted successfully to adult life. Those who showed the

greatest resiliency had “(1) a basic temperament that elicited positive responses from others; (2) a well-developed sense of efficacy, preparedness, and self-esteem that guided their lives; (3) competent caregivers and supportive adults; and (4) opportunities for a second chance when they made mistakes or got into trouble with the law. Although some of the characteristics are present at birth (e.g., temperament), many of the other supportive factors can be increased through the efforts of family members, schools, and communities” (Mash and Wolfe 2005, p. 336).

Clinical Expression and Pathophysiology

Learning disabilities are thought to have a neurobiological basis, specifically atypicalities in brain maturation and functioning. According to Gregorenko (2007), the working assumption in the field is that genetic factors affect the development, maturation, and functional structure of the brain, which in turn influences cognitive processes associated with learning disorders. In addition, external risk factors, such as poverty and lack of educational opportunities, affect patterns of brain functioning and development. These external factors may worsen the prognosis for a biological predisposition for learning disabilities or serve as a trigger for their development.

Evaluation and Differential Diagnosis

Current methods of diagnosing learning disorders involve individual psychological assessment and/or a response-to-intervention model. In the psychological assessment model, an individual’s cognitive and academic strengths and weaknesses are assessed through a comprehensive evaluation. If a severe discrepancy between the individual’s cognitive and academic functioning is found that cannot be explained by normal variation in academic attainment, lack of opportunity, poor teaching, or cultural factors, then a learning disorder is suspected. Additionally, it is necessary to rule out impaired vision or hearing. It is possible to diagnose learning disorders along with mental

retardation, pervasive developmental disorder, or communication disorders when “academic impairment is significantly below expected levels given the individual’s intellectual functioning and schooling” (APA 2000). The educationally based response-to-intervention model involves early, continuous monitoring of all children’s academic achievement. If children fall behind their peers, they are provided with high-quality research-based, small-group instruction and their progress is continually monitored. If, over time, this level of intervention is not sufficient to bring the child’s skills up to grade level, then the child is diagnosed as having a learning disability and provided with special education services.

Treatment

Interventions for learning disorders are primarily educational, rather than medical, in nature. A direct behavioral instruction approach is often used; tasks are broken down into manageable steps with ample examples, practice, and feedback. For example, Mash and Wolfe (2005) state that children with reading disorders must be taught phonemic awareness and phonemic decoding skills, fluency in word recognition, construction of meaning, vocabulary, spelling, and writing. When direct and explicit instruction in these components is provided, data show dramatic reductions in reading failure. According to Shaywitz (2003), the specific program chosen for remediation is less important than the fact that a scientifically proven program is provided by highly qualified teacher with sufficient intensity and for a sufficient length of time. Mash and Wolfe (2005) also indicate that cognitive behavioral interventions have also been found to be useful for children with learning disorders, particularly for tasks that involve monitoring one’s own thought processes. “Essentially, children are taught to ask themselves several questions as they progress, to make themselves more aware of the material. . . Why am I reading this? What’s the main idea the authors are trying to get across? Where can I find the answer to this question? How does this follow from what I learned a minute

ago?” (Mash and Wolfe 2005, p. 342). Finally, computer-assisted learning programs have been found to be helpful in providing opportunities for practice, feedback, and drills.

See Also

► [Educational Testing](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, fourth edition, text revision (DSM-IV-TR)*. Washington, DC: American Psychiatric Association.
- Gregorenko, E. (2007). Learning disabilities. In A. Martin & F. R. Volkmar (Eds.), *Lewis's child and adolescent psychiatry: A comprehensive textbook* (4th ed.). Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins.
- Mash, E. J., & Wolfe, D. A. (2005). *Abnormal child psychology* (3rd ed.). Belmont: Thomson Wadsworth.
- Shaywitz, S. (2003). *Overcoming dyslexia*. New York: Alfred A. Knopf.
- U.S. Office of Education. (1977). Assistance to states for education for handicapped children: Procedures for evaluating specific learning disabilities. *Federal Register*, 42, G1082–G1085.

Learning Experiences, an Alternative Program for Preschoolers and Their Parents (LEAP)

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Definition

Learning experiences, an alternative program (LEAP), is a model of early intervention that serves children with autism spectrum disorders (ASD) and typically developing children in an inclusive preschool setting. The program was developed to support the social and communicative skills of children with ASD

through a peer-mediated approach (Strain and Bovey 2008). Typical children are taught to use a series of strategies to engage classmates with ASD in social interaction. Training involves explicit teaching, role play, adult cueing and scaffolding, and positive reinforcement. Opportunities for social engagement between typical peers and children with ASD occur within the context of a preschool curriculum designed to facilitate the learning and development of both typical children and those with ASD.

Historical Background

The LEAP program was developed in 1981 by Dr. Phillip Strain. It began as a federally funded demonstration program in Pittsburgh, Pennsylvania, and was one of the first inclusive educational programs, serving children with ASD and their typical peers (Kohler et al. 1996). In 1984, LEAP began to set up replication sites, which provided general training to school districts across the United States and several foreign countries. In 1988, Douglas County, Colorado, became the primary demonstration site for the LEAP program, and as of 2008 over 80 LEAP preschool programs had been established (Strain and Bovey 2008).

Rationale or Underlying Theory

The LEAP program is based on current research suggesting that children with deficits in social communication benefit from peer-mediated intervention in an inclusive setting. To develop the social-communication skills that allow for the development of appropriate peer relationships, children with ASD must be provided with adequate opportunities to interact with typically developing peers (Strain et al. 1998). Inclusive classroom settings can be advantageous because opportunities for social interaction occur on a daily basis. However, Strain et al. (2008) point out that simply placing a child with ASD in an inclusive setting with typical peers does not in and of itself facilitate social interaction. Typically developing peers require instruction regarding

how to interact with their classmates with ASD in order to maximize appropriate development of social communication. Strain and Bovey (2008) cite several studies indicating that typically developing children as young as 36 months can be trained to use strategies that support the social communication of children with ASD, resulting in higher rates of communicative interactions, and that many children with ASD who participate in peer-mediated intervention eventually develop social-communication skills that are considered developmentally typical.

Goals and Objectives

The primary goal of the LEAP program is to support the development of appropriate social-communication skills through peer-mediated intervention. One of the core deficits of children with ASD described by *The Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition* (DSM-IV; American Psychiatric Association 2000) is a qualitative impairment in social interaction, which is frequently characterized by a failure to develop appropriate relationships with peers and a general lack of social reciprocity. The LEAP program incorporates explicit instruction in social-communication strategies into its general curriculum, and typical peers are trained to facilitate the social communication of children with ASD.

Treatment Participants

There are currently no set guidelines to determine eligibility to participate in the LEAP program. Because there is a primary focus on developing social-communication skills and fostering successful peer relationships, children who exhibit deficits in these areas would be candidates. Strain and Bovey (2008) indicate that all children participating in efficacy studies have met the diagnostic criteria for “early childhood autism” on the DSM-III and DSM-III-R. However, it is likely that children with other diagnoses characterized by deficits in social communication would benefit from

this intervention. Specific diagnoses might include childhood disintegrative disorder (CDD), Asperger’s disorder, and pervasive developmental disorder not otherwise specified (PDD-NOS). Participants should be approximately 3–5 years of age, as the intervention is provided within a preschool setting.

Children participating in the LEAP program may have a range of verbal language ability. The program supports the development of verbal language through a variety of naturalistic approaches including incidental teaching and time delay (Strain and Bovey 2008). Additionally, the *picture exchange communication system* (PECS), along with other low-tech and high-tech augmentative and alternative communication systems, has been successfully incorporated into LEAP classrooms, providing communication modalities for children who have little to no verbal language (Strain and Bovey).

Treatment Procedures

LEAP classrooms typically operate 5 days a week for 3 h a day. Each classroom includes 3–4 children with ASD, 8–10 typically developing peers, and at least three adults, who may include an early education teacher, early childhood special education teacher, speech-language pathologist, occupational therapist, and classroom assistant (Strain and Bovey 2008). The program features full-time inclusion, so children with ASD participate in all the same classroom activities alongside their typically developing peers. Every classroom utilizes a general preschool curriculum, along with a social skills curriculum. Although there is variation among sites, most LEAP classrooms utilize the *Creative Curriculum for Preschool* (Dodge et al. 2002) and/or the *Storybook Journey: Pathways to Literacy Through Story and Play* (McCord 1995) to guide the general curriculum (Strain and Bovey 2008).

The LEAP social skills curriculum consists of five social skills: getting a friend’s attention, sharing, requesting sharing, play organizing (“Let’s _____” statements), and giving compliments (Strain and Bovey 2008). These skills were

specifically chosen based on evidence that they facilitate longer interactions between children and support the development of friendships (Kohler et al. 1992). Skills are taught one at a time until children demonstrate understanding of the steps involved and are able to utilize the skill independently during play. Each skill is taught in six basic steps (Strain and Bovey 2008):

- *Step 1 Describe:* The skill is introduced and described, and any steps that are involved in the skill are explained. Visual images depicting the skill are utilized to facilitate understanding. For example, when describing “getting a friend’s attention,” the interventionist might say, “Today we are going to learn how to get our friends to play with us, because it is important that friends learn how to play together. One way to get your friend to play with you is by getting their attention.” The interventionist then details the steps of the skill: “To get your friend’s attention you can look at your friend, tap them on the shoulder, and say their name. If you don’t get their attention right away keep trying.”
- *Step 2 Demonstrate the Skill:* Two adults demonstrate the skill being taught. For example, the interventionist might say, “Let’s practice getting a friend’s attention. I’m going to get Kate’s attention. Tell me if I do it right.” The interventionist then role plays the scenario with the other adult and reiterates what happened.
- *Step 3 Demonstrate the Wrong Way:* Two adults demonstrate the skill again, but do it the wrong way. Following the demonstration, the interventionist asks the children to point out what was wrong with the interaction. For example, the interventionist might tap the other adult on the shoulder, but forget to say her name.
- *Step 4 Child Practice with Adult:* The interventionist reviews the skill and then calls on a child to demonstrate it for the class. The interventionist might say, “Now I want you to show the class how to get your friend’s attention. Let’s pretend I’m your friend and you want to get my attention. Remember to look at me, say my name, and tap me on the shoulder if I’m not looking.”
- *Step 5 Practice with Target Child:* The interventionist reviews the skill again and then instructs the typical peer to practice the skill with the child with ASD.
- *Step 6 Awards and Prizes to Students:* The interventionist awards prizes to reinforce children’s use of the skills being taught. Specific praise is also provided that explains why the child is receiving a prize, such as “Good work, you got your friend’s attention” or “Nice job looking at your friend.”

The LEAP program also incorporates several naturalistic teaching strategies in order to facilitate learning and development of skills throughout the day in a variety of situations. These strategies include incidental teaching, mand-model procedures, time delay, and pivotal-response training (PRT), along with general prompting and modeling by adults (Strain and Bovey 2008). Incidental teaching involves arranging the environment in a way that promotes communicative behavior. For example, desired objects might be placed out of reach so that the child must request them. In the mand-model procedure, the interventionist first prompts the child to communicate by asking a question (the mand) (e.g., “what do you want?”), followed by a model of the target behavior (e.g., “say, I want the bubbles”). When using time delay, the interventionist waits approximately 5–10 s before assisting a child who is having difficulty obtaining or using a desired item with the goal that the child will initiate a request for help. PRT is a technique based on the principles of applied behavioral analysis (ABA) that involves rewarding and shaping attempts at a target behavior. Rewards are in the form of natural reinforcers so that if a child asks for a hat, she receives the hat as a reward as opposed to an unrelated reward, such as a piece of candy.

Family involvement is an integral aspect of the LEAP program. Services available to families participating in the program include training focusing on strategies for behavior management and teaching new skills to children, monthly parent support groups, referral to community

agencies offering relevant services such as respite care, and transition planning and follow-up services (Strain and Bovey 2008). Families are taught to implement certain intervention strategies used in the classroom at home to facilitate the generalization of skills. Families also participate in some aspect of the preschool program ranging from assisting in the creation of instructional materials and preparing activity centers to providing direct intervention (Strain et al. 1996).

Efficacy Information

To date, only one study has assessed the efficacy of the LEAP program as a whole. Strain and Hoyson (2000) conducted a longitudinal study evaluating intervention outcomes for six children enrolled in the LEAP program. Participants were assessed prior to beginning the program and upon completion of the program (2–3 years after beginning the program). Follow-up measures were taken approximately 4 years after completion of the program when the children were 10 years of age. At baseline, all participants fell within the moderate to severe range of autism on the *Childhood Autism Rating Scale* (CARS; Schopler et al. 1988), with a mean score of 35. Upon completion of the program, participants no longer met criteria for autism on the CARS. Participants also exhibited significant increases in the appropriateness of their behavior during interactions with caregivers. At baseline, the mean time engaged in appropriate behavior with caregivers was 51%. Upon completion of the program, participants demonstrated appropriate behavior with caregivers 98% of the time, and parent ratings of behavior rose from “very unacceptable” to “very acceptable.” Additionally, participants’ mean percent of intervals engaged in positive social interaction rose from 3% at baseline to 23% upon program completion. This is comparable to the performance of typical peers whose mean percent of intervals engaged in positive social interaction was 28% and 25% at baseline and program completion, respectively. All program outcomes were maintained at follow-up, 4 years following completion of the program.

The participants described by Strain and Hoyson (2000) were the first participants to complete the research protocol for the LEAP program. An additional 45 children have since been enrolled in the research protocol with the intent of replicating results of the current study in the future (Strain and Hoyson 2000). Although this single study provides limited evidence for the efficacy of the LEAP program as a whole, the effectiveness of the peer-mediated intervention strategies, such as those utilized in the LEAP program, has been widely studied. Current evidence suggests that peer-mediated interventions are effective in increasing both the frequency and duration of social interactions between children with ASD and their typically developing peers (e.g., Laushey and Heflin 2000; Odom and Strain 1986; Ragland et al. 1978; Strain et al. 1979; Roeyers 1996). Based on the positive outcomes demonstrated by Strain and Hoyson (2000) and the well-documented effects of peer-mediated intervention strategies in general, the LEAP program is considered to be efficacious intervention for promoting the development of social-communication skills and appropriate peer relationships in children with ASD.

Outcome Measurement

The LEAP program utilizes a tracking system that measures children’s progress toward individualized objectives in terms of level of independence (Strain and Bovey 2008). Children’s level of independence for each objective is rated daily on a scale from 0 to 4, with a score of 0 indicating that the child refuses to perform the skill and a score of 4 indicating that the child performs the skill independently without adult assistance. Interventionists set criteria for each level that the child must meet before moving to the next level of independence. For example, the child might have to exhibit performance at a level 2 (adult provides partial physical assistance) for 6 out of 7 days before moving to a level 3 (adult points/gestures/models or verbally directs the child). As children move up the scale, the interventionist adjusts their behavior to match the ability of the child.

Qualifications of Treatment Providers

Although Strain and Bovey (2008) note that LEAP staff configurations vary, service providers may include early education and special education professionals, speech and language pathologists, occupational therapists, and trained classroom assistants. Additionally, each classroom typically has at least one teacher with a master's level education (Strain et al. 1996). All staff are provided training through the LEAP program.

See Also

- ▶ Empathy
- ▶ Expressive Language
- ▶ Incidental Teaching
- ▶ Initiation of Communication
- ▶ Integrated Play Groups (IPG) Model
- ▶ Language Interventions
- ▶ Modeling
- ▶ Parent-Professional Partnership
- ▶ Peer-Mediated Intervention
- ▶ Peers, Exposure To
- ▶ Positive Reinforcement
- ▶ Preschool
- ▶ Prosocial Behavior
- ▶ Reciprocal Communication/Interaction
- ▶ Social Cognition
- ▶ Social Communication
- ▶ Social Interventions
- ▶ Theory of Mind
- ▶ Verbal Communication

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Rev.). Washington, DC: APA Press.
- Dodge, D. T., Colker, L., & Heroman, C. (2002). *The creative curriculum for preschool* (4th ed.). New York: Teaching Strategies.
- Kohler, F. W., Strain, P. S., & Shearer, D. D. (1992). The overtures of preschool social skill intervention agents: Differential rates, forms, and functions. *Behavior Modification, 16*, 525–542.
- Kohler, F. W., Strain, P. S., & Shearer, D. D. (1996). Examining levels of social inclusion within an integrated preschool for children with autism. In L. K. Koegel, R. L. Koegel, & G. Dunlap (Eds.), *Positive behavioral support: Including people with difficult behavior in the community* (pp. 305–332). Baltimore: Paul H. Brookes Publishing.
- Laushey, K. M., & Heflin, L. J. (2000). Enhancing social skills of kindergarten children with autism through the training of multiple peers as tutors. *Journal of Autism and Related Disorders, 30*, 183–193.
- McCord, S. (1995). *The storybook journey: Pathways to literacy through story and play*. Englewood Cliffs: Prentice-Hall.
- Odom, S. L., & Strain, P. S. (1986). A comparison of peer-initiation and teacher antecedent interventions for promoting reciprocal social interactions of autistic preschoolers. *Journal of Applied Behavior Analysis, 19*, 59–71.
- Ragland, E. U., Kerr, M. M., & Strain, P. S. (1978). Behavior of withdrawn autistic children: Effects of peer social initiations. *Behavior Modification, 2*, 565–578.
- Roeyers, H. (1996). The influence of nonhandicapped peers on the social interactions of children with a pervasive developmental disorder. *Journal of Autism and Developmental Disorders, 26*, 303–320.
- Schopler, E., Reichler, R. J., & Renner, B. R. (1988). *Childhood autism rating scale*. Los Angeles: Western Psychological Resources.
- Strain, P. S., & Bovey, E. H. (2008). LEAP: Learning experiences, an alternative program for preschoolers and parents. In J. S. Handleman & S. L. Harris (Eds.), *Preschool education programs for children with autism* (pp. 249–281). Austin: Pro-Ed.
- Strain, P. S., & Hoyson, M. (2000). The need for longitudinal social skill intervention: LEAP follow-up outcomes for children with autism. *Topics in Early Childhood Special Education, 20*(2), 116–122.
- Strain, P. S., Kerr, M. M., & Ragland, E. U. (1979). Effects of peer-mediated social initiations and prompting/reinforcement procedures on the social behaviour of autistic children. *Journal of Autism and Developmental Disorders, 9*, 41–54.
- Strain, P. S., Kohler, F. W., & Goldstein, H. (1996). Learning experiences... an alternative program: Peer-mediated interventions for young children with autism. In E. D. Hibbs & P. S. Jensen (Eds.), *Psychosocial treatments for child and adolescent disorders: Empirically based strategies for clinical practice* (pp. 573–587). Washington, DC: American Psychological Association.
- Strain, P. S., Wolery, M., & Izeman, S. (1998). Considerations for administrators in the decision of service options for young children with autism and their families. *Young Exceptional Children, 1*(2), 8–16.
- Strain, P. S., Schwartz, I. S., & Bovey, E. H. (2008). Social competence interventions for young children with autism. In W. H. Brown, S. L. Odom, L. Samuel, & S. R. McConnell (Eds.), *Social competence of young children: Risk, disability, and intervention* (pp. 253–272). Baltimore: Paul H Brookes Publishing.

Learning Language Through Overhearing in Children with ASD

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Language development in the early years of life is of utmost importance for children with autism spectrum disorder (ASD), whose early linguistic skills are strong predictors of a variety of social, adaptive, and academic outcomes. Documenting which mechanisms are and are not contributors to mastery of fundamental language skills is therefore of central clinical (and theoretical) importance (e.g., Arunachalam and Luyster 2016).

Typically developing children have an arsenal of strategies for learning new words in infancy and beyond. Although the prototypical word-learning context for many cultures as well as most research studies is that of “child-directed speech” – settings in which a caregiver explicitly directs a novel word to the child, usually in a 1:1 interaction – children can acquire new words from a broader range of experiences. For instance, toddlers are proficient at learning new words even when they are eavesdropping on a third-party conversation; this skill has been documented in laboratory-based tests (e.g., Akhtar et al. 2001), and cross-cultural research has confirmed the ability of children to learn across a wide range of linguistic contexts (e.g., Ochs and Schieffelin 1984).

Exploring the question of whether children with ASD are able to learn new words via overhearing has the potential to enrich our understanding of what aspects of language development in ASD are “intact” (e.g., Arunachalam and Luyster 2016). Given associations between language input and language mastery for children with ASD, this inquiry also has the potential to support caregivers, clinicians, and therapists in enriching the early language environments of children with ASD.

The first study exploring the ability of children with ASD to learn language by overhearing was a “proof of concept” study (Luyster and Arunachalam 2018) asking whether, in a laboratory setting, children with ASD were able to learn new nouns through overhearing others use them in conversation. The final sample included 13 children with ASD (ages 4 and 5 years old), for whom diagnosis was confirmed using the Autism Diagnostic Observation Schedule – 2nd edition (ADOS-2). Based on scores from the MacArthur Bates Communicative Development Inventory forms II and III (MBCDI) and Language Use Inventory (LUI), children’s communication skills were roughly at the 28-month developmental level.

Using a within-subject design and a paradigm based on previous research (Akhtar et al. 2001), each child was taught new object labels in both directly Addressed (i.e., the experimenter spoke directly to the child) and Overheard (i.e., the experimenter spoke only to a confederate; the child was only an onlooker) training conditions. Each experimental condition (i.e., Addressed, Overheard) used a different novel word (*modi*, *toma*) to introduce one of three novel objects. When introducing the target object, the phrases included a novel label for the object (e.g., “I’m going to show you the toma. Let’s see the toma... I like this toma!”). When introducing nontarget items, the phrases were similar but did not include an explicit label (“I’m going to show you what’s in here. Let’s see what’s in here. I like this one!”). After each training round was completed, the child was asked to identify the target object and place it in a small, decorated bucket.

Overall, children succeeded in the task; they chose the correct novel object 62% of the time in the Addressed condition and 69% of the time in the Overheard condition. Chance performance is 33% given that three novel objects were presented. Binomial tests on the data from each condition were used to determine whether performance was above chance. In the Addressed condition, a binomial test indicated that the observed success rate of 67% was significantly higher than the chance level of 33%, one-tailed

$p < 0.04$. In the Overheard condition, too, the success rate (69%) was significantly higher than chance, one-tailed $p < 0.01$.

These results are consistent with prior research with typically developing children (e.g., Akhtar et al. 2001) and provide new information about the ability of children with ASD to learn through overhearing. In this study, children with ASD succeeded in both Addressed and Overheard contexts, indicating that at least some children on the spectrum can learn nouns from overheard speech. This may inform strategies that clinicians can use to promote language development for some children with ASD, especially those who are overwhelmed by social interactions. It may also be useful for learning personal pronouns, which – for typically developing children – seem to be facilitated by access to overheard speech (e.g., Oshima-Takane 1988).

See Also

- Communicative Acquisition in ASD
- Language
- Language Acquisition
- Theories of Language Development

References and Reading

- Akhtar, N., Jipson, J., & Callanan, M. A. (2001). Learning words through overhearing. *Child Development*, 72(2), 416–430. <https://doi.org/10.1111/1467-8624.00287>.
- Arunachalam, S., & Luyster, R. J. (2016). The integrity of lexical acquisition mechanisms in autism spectrum disorders: A research review. *Autism Research*, 9(8), 810–828. <https://doi.org/10.1002/aur.1590>.
- Luyster, R. J., & Arunachalam, S. (2018). Brief report: Learning language through overhearing in children with ASD. *Journal of Autism and Developmental Disorders*, 1–9.
- Ochs, E., & Schieffelin, B. B. (1984). Language acquisition and socializations: Three developmental stories and their implications. In R. A. Shweder & R. A. LeVine (Eds.), *Culture theory: Essays on mind, self and emotion* (pp. 276–320). Cambridge, UK: Cambridge University Press.
- Oshima-Takane, Y. (1988). Children learn from speech not addressed to them: The case of personal pronouns. *Journal of Child Language*, 15(01), 95–108. <https://doi.org/10.1017/S0305000900012071>.

Learning Styles

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Definition

Learning styles refer to how someone learns best, with the implication that the primary mode of instruction (visual, auditory, kinesthetic, etc.) should be matched to the individual's preference and ability. This idea has gained great currency in general education, but a recent review of empirical evidence (Pashler et al. 2009) concludes that the theory lacks empirical support and should not be the basis of general educational strategy. In the field of special education, however, despite lack of rigorous empirical studies, educational strategy is influenced by the very significant language impairments of children with ASD, which makes them better able to learn and perform tasks with prominent visual or visuospatial components, both to get and hold their attention, and to facilitate comprehension and retention. Many individuals with ASD appear to learn best when provided with visual cues and instruction. This appears true for individuals with ASD with and without intellectual disabilities. This preference for visual learning is the basis for many interventions, including the picture exchange communication system (PECS), visual schedules, and many of the TEACCH strategies. One other dichotomy may be drawn between explicit and implicit learning: The former refers to conscious, intentional learning of facts, rules, and events, which can be learned in a single trial, while the latter refers to automatic, rote, learning of habits and procedures that are mainly learned by repetition. For individuals with ASD and significant intellectual disability, the latter may be a more efficient style of learning. This would involve emphasizing learning goals, such as activities of daily living and

simple communication such as pointing to a desired object, that can be learned through repetition and may need more teaching than usual to be generalized.

References and Reading

- Francis, K. (2005). Autism interventions: A critical update. *Developmental Medicine and Child Neurology*, 47(7), 493–499.
- Pashler, H., McDaniel, M., Rohrer, D., & Bjork, R. (2009). Learning styles: Concepts and evidence. *Psychological Science in the Public Interest*, 9, 105–119.
- Quill, K. (1997). Instructional considerations for young children with autism: The rationale for visually cued instruction. *Journal of Autism and Developmental Disabilities*, 27(6), 697–714.
- Rao, S., & Gagne, B. (2006). Learning through seeing and doing: Visual supports for children with autism. *Teaching Exceptional Children*, 38(6), 26–33.

Least Restrictive Environment (LRE)

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Synonyms

Appropriate educational placement; Inclusion

Definition

Least Restricted Environment (LRE) is a policy mandated by federal law, which requires that students be educated to the greatest extent appropriate with peers who are not disabled, in the schools they would attend if they were not disabled. The requirement extends to both academic and extra-curricular programming. LRE is imposed on all states receiving federal education funding and applies to students aged 3–21.

There is no one definition of LRE. Rather, LRE placement must be individualized for each student

and is not predetermined based on a student's disability or other factors. Instead, the appropriate educational placement (the LRE) must be determined by the IEP (individualized education plan) team.

The IDEIA (Individuals with Disabilities Educational Improvement Act) and its predecessor, the IDEA (Individuals with Disabilities Education Act), express a strong preference for educating students with disabilities in regular classrooms (also known as inclusion), but they recognize that inclusion may not be appropriate for all students. School districts must offer a continuum of services to meet the needs of students with disabilities. When making placement decisions, IEP teams must first consider a regular education classroom placement and must move down the continuum until finding the appropriate place and manner for educational services to be delivered. Accommodations (referred to as supplementary aids and services by the IDEIA) that would enable a student to be in a more inclusive setting must be considered. Examples of accommodations that may be helpful for students with autism spectrum disorders include curriculum modifications, effective behavioral supports, teacher training, and assistive technology.

The principles of LRE stem from the seminal case of *Brown v. Board of Education*, whose opinion established the importance of education for all children. Since *Brown*, federal courts considering cases brought under the IDEA have repeatedly recognized the academic, social, and behavioral benefits of educating children with disabilities with typical peers.

See Also

- [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- Brown v. Board of Education*, 347 U.S. 483 (1954).
- Douvanis, G., & Hulsey, D. (2002). *The least restrictive environment mandate: How has it been defined by the courts?* Retrieved from <http://www.ericdigests.org/2003-3/courts.htm>

- Individuals with Disabilities Education Act of 1990, Pub. L. No. 101-476, 104 Stat. 1142.
- Individuals with Disabilities Education Improvement Act of 2004, 20 U.S.C. §§ 1400 et seq., Pub. L. 108-446, 118 Stat. 2803.
- Pacer Center. (2009). Action information sheets: Least Restrictive Environment (LRE): A simplified guide to key legal requirements. PHP-c7. Retrieved from <http://www.pacer.org/parent/php/PHP-c7.pdf>
- Wrightslaw. Questions and answers on Least Restrictive Environment (LRE) requirements of the IDEA. Retrieved May 2, 2011, from <http://www.wrightslaw.com/info/lre.osers.memo.idea.htm>

Lebanon and Autism

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Country Background

Lebanon is a Middle Eastern country of 10,452km², a population of 4 million Lebanese citizens, more than one million registered Syrian refugees, and 500,000 registered Palestinian refugees. Since its independence in 1943, Lebanon has been a land of war-related turmoil, internal conflicts, and social and economic struggles. All the economy is based on the efforts of the private sector.

Historical Background

The worldwide prevalence of children and people with autism spectrum disorder has been on the rise in the past few decades (El Sabbagh et al. 2012). Research studies conducted in Arab countries indicated a lower prevalence rate than the worldwide estimated prevalence of 62/10000. For example, the United Arab Emirates estimated the prevalence of 29/10,000 (Eapen et al. 2007), Bahrain found a prevalence of 4.3/10,000 (Al-Ansari and Ahmed 2013), the Sultanate of Oman found 1.4/10,000 (Al-Farsi et al. 2011). It was argued

that the lack of standardized and culturally sound tools and procedures, as well as the lack of qualified professionals in diagnosing Autism Spectrum Disorder (ASD), explains this finding (Obeid et al. 2015). Although there are no epidemiological studies on ASD in Lebanon, there is an increase in referrals for ASD to clinicians from as early as the age of 18 months.

The history of the disability field in Lebanon goes back to the Ottoman Empire when the first law in 1876 dealing with “*mentally ill*” people was proclaimed and implemented (Raad 2000). Persons with disabilities were subject to negative terminologies and placed in asylums (Megarbane 1996). In 1973, the first disability categorizations were stated in the law 11/73 which defined persons with “handicap” and differentiated between the *mentally* and the *physically* handicapped people (Raad 2000). In 2000, the Lebanese parliament approved the most recent disability law 220/2000 which for the first time introduced the notion of rights as an underlying principle to disabilities. This law divided people with disabilities in four categories: intellectual disability, visual disability, hearing disability, and physical disability. Autism Spectrum Disorder (ASD) is therefore not considered as an independent category in the Lebanese legislation. Persons with autism are counted under Intellectual Disability (ID) in the national statistics, which constitutes a major bias. In 2004, the Central Administration of Statistics (CAS)’s report indicated that 2% of the Lebanese residents have a disability and 18.4% of them have an intellectual disability. These figures are taken from the Ministry of Social Affairs registry (MOSA) that is not exhaustive of all individuals with disabilities.

The first educational program dedicated for children with autism was founded in 1989 by a nongovernmental organization (NGO). It started with 12 children aged between 8 and 12 years old. The professional team was composed of four special educators, a child psychologist, and a social worker. The program’s aim was to provide each child with a stimulating learning environment, and to address his/her problem behavior. Every 2 to 3 children were grouped with one special education teacher. Parents were provided with

social and psychological support and guidance. No specific school of thoughts was chosen. Later, the program tried to implement the principles and techniques of the Treatment and Education of Autistic and Related Communication-Handicapped Children (TEACCH) (Mesibov et al. 2005). A French approach the “*Thérapie d’échange et de développement* (TED)” (Lelord et al. 1991; Barthelemy et al. 1995; Bataille et al. 2010) is introduced in the early 2000. TED is very similar to TEACCH in the one-to-one setting.

In 1996, the first privately owned early intervention center was opened for children below the age of 4, presenting with autism. With a full capacity of 9 children, the center operated for 4 years and closed short of financial resources. The team was composed of special education teachers, a speech therapist, a psychomotor therapist, a child psychologist. A parents’ support group was established; it became later the first parents’ advocacy group for children with ASD.

As of the year 2000, more programs for children with autism became available, with more specialized interventions and skilled professionals. Lebanon counts now around 13 NGOs offering specialized programs covering early childhood, childhood, and early adulthood. These NGOs operate in a holistic approach more empirically based than designed within an evidence-based therapeutic framework. Children enrolled in their programs have moderate to severe ASD, associated with Intellectual Disabilities.

More recently, a major effort in the early diagnosis of autism is made with the increased number of specialized physicians and allied health professionals established in Lebanon. A handful number of centers are specialized in early assessment and early intervention. More specifically, two centers in a tertiary care facility in Beirut opened in 2011 and in 2014. The first one provides pediatric neurology and individual rehabilitation assessments and follow-up services in the areas of speech therapy, physiotherapy, applied behavioral analysis, psychomotor therapy, and occupational therapy. The second center provides child psychiatry, clinical psychology, and special education assessments and services. These two

centers have a strong team-based approach. They base their work on international standards of care and embed their assessments and interventions on evidence-based tools and techniques.

The advances in service provision have contributed to greater awareness among people in society, leading to changes in perception, mentalities, and quality of care and services (Obeid et al. 2015). Activists’ groups, mainly parents of children with ASD, established NGOs and lobby for their children’s rights through advocacy work at the policy level and the creation of educational and vocational services. In addition, the movement of inclusive education initiated in the 1990s becomes stronger lately. This helps children with mild ASD to be maintained in regular schools.

Legal Issues, Mandates for Services

Lebanese citizens with disabilities are under the jurisdiction of the MOSA, specifically under the broad umbrella of welfare services. As stated above, the only law (220/2000) pertaining to secure their rights in all domains, education, health, work, etc., was issued in 2000. The text is comprehensive, comparable with other jurisdictions in western countries. However, it has not been fully enforced. A person needs to register at the MOSA and receive a disability card. Card holders can then benefit from some tax deductions, access to NGOs’ educational and rehabilitation programs, if the NGO has signed an agreement with MOSA. The NGOs offer services for low fees but no quality control is performed. Various persons with disabilities choose not to get the disability card which they consider to be discriminative.

Lebanon hosts since 1948 Palestinian refugees who receive support from the United Nation Relief and Works Agency for Palestine Refugees in the Near East (UNRWA). UNRWA mandate is to support medical, educational, and rehabilitation services for 450,000 individuals in the 12 Palestinian camps. UNRWA has not developed a specific program to address the needs of individuals with ASD.

As per the United Nations High Commissioner for Refugees (UNHCR), up until December 2016, more than million Syrian refugees fled to Lebanon and 45% of them are children. The UNHCR, international and national NGOs ensure the basic needs only. Nothing is available for individuals with ASD.

The absence of legal framework specific to ASD, the limited access to quality care through governmental and United Nations bodies, pushed the civil society to fill the vacuum. NGOs and charity initiatives assist parents with low incomes in providing their children with ASD with basic services.

Diagnostic, educational, and rehabilitation services are also provided by the private sector, most professionals working in Beirut and its surrounding. The social security and the insurance companies cover the medical treatment only. Psychological and rehabilitation services are not covered. This creates a huge burden on individuals and their families, delays diagnoses, and jeopardizes the access to, and the, continuity of care.

Overview of Current Treatments and Centers

Two centers located in a private tertiary care facility offer diagnostic assessment based on the DSM 5, and specialized follow up. Clinical assessment was supported with a variety of tools such as the Autism Diagnostic Observation Schedule (ADOS-2), the Arabic version of the Childhood Autism Rating Scale (CARS-2) (Akoury-Dirani et al. 2013a, b), and neurological and genetic testing. Other professionals in the private sector assess autism using clinical observation, and parents' informal interviews.

To date, children with disabilities, including those with ASD, may benefit from different academic modalities depending on the severity of their conditions, the availability of services in geographical areas and parents' choice, financial means, awareness, and perception of disability. Another impactful criterion is whether the child has been subject to early intervention services

which would enable him/her to enroll smoother into a school setting.

One option is the private disability centers or institutions. Currently around 13 NGOs offer a day program for children, adolescents, and young adults with moderate to severe autism, with or without dual diagnoses. Most of these grassroots organizations are in the capital city Beirut and its suburbs with some located in Mount Lebanon (the closer Governorate to the capital) and few scattered in further Governorates such as the North, East, and the South. They are either founded by parents of children with ASD or by religious and other civil society entities. Beneficiaries are grouped by severity levels. The educational program covers the contents of the first elementary school cycle. Individual sessions of speech and occupational therapy as well as special education may be offered, in addition to social support for parents. When the NGO is sub-contracted by MOSA, the tuition fees are minimum.

The second option is the inclusion of children in regular private schools. The child is either in the regular classroom accompanied by a shadow teacher or enrolled in a self-contained class in parallel with the regular ones. The child may be also partially attending the regular class and benefiting from individualized pull out sessions in the resource room. In addition to the modified academic curriculum or the Individual Educational Program, the schools provide rehabilitation services such as speech therapy, psychomotor therapy, counseling, and special education.

Inclusive education remains however an expensive option. Tuition fees for children with ASD are double or triple the regular tuition fees for the same grade level; it varies depending on the amount and intensity of academic accommodations and rehabilitation of services needed by the child.

Lately, two privately led inclusive education projects are implemented in 240 public schools as a collaboration between NGOs and the Ministry of Education and High Education (MEHE). The third inclusive education project is a public initiative led by MEHE and implemented in 20 public schools across Lebanon. These pilot

projects target students with learning disorders. If the model succeeds, it is said that it would be expanded to other categories such as students with ASD.

Over the past decade, Lebanon has been witnessing a higher ratio of young children (1–3 years old) being referred to specialized professionals (psychiatrists, psychologists, pediatric neurologists) for diagnosis of ASD. An effort is made on addressing the very early stages of child development. Two early intervention programs (zero to three) exist in Beirut offering diagnoses, assessment, and treatments.

These children are then referred to nurseries where they can be provided with adequate support. Some nurseries have special education interventions and/or more frequently other rehabilitation services such as psychomotor and speech therapists.

Finally, the Applied Behavior Analysis (ABA) has been shyly introduced in Lebanon about 20 years ago. It received initially a lot of resistance from the community of professionals and parents. More recently, less than 10 professionals have been certified in ABA and they operate in private practice. They may either deliver individual session in their office or do home-based interventions.

Overview of Research Directions

Eleven articles were published between 2001 and 2017 in indexed peer-reviewed journals. Scholars worked on prevalence studies, validation of scales, suicidality and autism, early intervention, and genetics.

One prevalence study done in 2016 by Chaaya et al. (2016), on toddlers between 16 and 48 months attending nurseries in Beirut and Mont Lebanon, estimated the prevalence of ASD to be 153/10,000. This prevalence is higher than those published in Arab studies but close to the 147/10,000 reported by the CDC in 2014 (CDC 2014). However, the representativeness of the sample and the usage of the M-CHAT, a screening tool to estimate prevalence, may limit the generalizability of the results. Another study tried to describe the most prevalent associated factors

with ASD (Hamade et al. 2013). The small sample size could not derive conclusive findings.

Three papers were published on the translation to Arabic and the validation of scales supporting the assessment of ASD. One was on the translation and the validation of the Revised Autistic Behavior Summarized Evaluation Scale (BSE-R) (Hreich et al. 2016). The two others were on the translation to Arabic and the validation of the Childhood Autism Rating Scale 2nd edition (Akoury-Dirani et al. 2013a, b).

Two nonconclusive genetic studies were conducted (Kourtian et al. 2017; Soueid et al. 2016).

Two studies described interventions. One targeted the description of speech and language therapy for children with ASD (Kouba-Hreich et al. 2001). The second was a case study on three children aged between 5 and 6 years, who were trained through ABA to take leisure activities independently (Daou 2014).

One study targeted knowledge and social perception of ASD (Obeid et al. 2015). It compared between college students in the USA and college students in Beirut-Lebanon. At baseline, students from Lebanon reported less overall knowledge and higher stigma towards ASD – they were more knowledgeable than students in the USA about disparities in access to care.

Finally, one literature review on suicidality in ASD was conducted and published in a peer-reviewed journal (Richa et al. 2014).

In addition, a voluminous grey literature produced by graduate students from different majors like psychology, special education, speech and language pathology, occupational therapy merits to be explored if we want to build a research strategy or program based on some previous data. In conclusion, the topic is of interest among junior professionals and some scholars. Short of specific research programs, the research initiatives are scattered, established on individual efforts, and supported with limited funding.

Overview of Training

Universities include the theme of autism in their teaching in degrees that are related to the field:

medicine, psychology, education, etc. One academic center includes the TED approach in the training of speech therapists.

In the early 2000, a European NGO funded a large project pertaining to empowering local NGOs offering educational and rehabilitation programs specific to children with autism. This 3-year project deployed a series of on-site training sessions. Special educators, speech therapists, and psychomotor therapists benefited from theoretical information, and hands-on skills. This training contributed to spread the same “professional culture” when it comes to working with persons with autism and their families. Occasionally, an NGO or a University would invite an expert to give a talk or a workshop on autism.

Social Policy and Current Controversies

Addressing the needs of the persons with autism remains an important challenge in Lebanon, as it is for all other disabilities. The national classification of disabilities includes ASD under ID. This shows a major misconception about this disorder. Within the same line, the diagnosis of very young children and of children with mild autism when possessing verbal abilities is rarely posed (Akoury-Dirani et al. 2013b). Diagnostic confusion between mild ASD, Attention Deficit and Hyperactivity Disorder (ADHD), and Disruptive Behavior is clinically observed.

The most important drawback is the absence of national policies guaranteeing rights, guiding the provision of services, controlling for quality, and encouraging research. All accomplishments are based on private initiatives and out of pocket money or private fundraising efforts. The limited financial resources collected for this cause added to the impoverishment of the population, draining the support towards covering the immediate and basic needs of persons with ASD.

Another source of concern is the concentration of the few experts and professionals in Beirut and its surrounding. Therefore, even when individuals live far from the capital and have the financial resources, they cannot access proper care.

The absence of formal social skills training for children and adolescents with ASD contributes to maintaining them in a constricted environment. Some adults, and aging people with moderate to severe autism, are maintained in institutions where they work in sheltered vocational activities while most of them are homebound.

Within this challenging context, parents of children with autism started their rights-based advocacy movement since the 1990s. Two associations exist. Their advocacy movement changed the knowledge and perception about autism. Marathons, fundraising events, awareness campaigns, and media appearances are done by such organizations and other supporting members from the society. This movement also led to the increase of early diagnoses and professional interventions as well as the financial support to create educational settings for children with ASD and fund research projects.

References and Reading

- Akoury-Dirani, L., Alameddine, M., & Salamoun, M. (2013a). Validation of the Lebanese childhood autism rating scale-second edition-standard version. *Research in Autism Spectrum Disorders*, 7, 1097–1103.
- Akoury-Dirani, L., Alameddine, M., & Salamoun, M. (2013b). Validation of the Lebanese childhood autism rating scale - second edition- high functioning version. *Research in Autism Spectrum Disorders*, 7, 1332–1338.
- Al-Ansari, A. M., & Ahmed, M. M. (2013). Epidemiology of autistic disorder in Bahrain: Prevalence and obstetric and familial characteristics. *Eastern Mediterranean Health Journal*, 19(9), 769–774.
- Al-Farsi, Y. M., Al-Sharbaty, M. M., Al-Farsi, O. A., Al-Shafae, M. S., Brooks, D. R., & Waly, M. I. (2011). Brief report: Prevalence of autistic spectrum disorders in the sultanate of Oman. *Journal of Autism and Developmental Disorders*, 41, 821–825. <https://doi.org/10.1007/s10803-010-1094-8>
- Barthélémy, C., Hameury, L., & Lelord, G. (1995). *L'autisme de l'enfant: La thérapie d'échange et de développement*. Paris: Expansion Scientifique Française.
- Bataille, M., Dansart, P., Blanc, R., Mahe, C., Malvy, J., & Barthélémy, C. (2010). Autisme: La Thérapie d'échange et de développement. In C. Tardif (Ed.), *Autisme et pratiques d'interventions* (pp. 59–84). Marseille: SOLAL.
- Centers for Disease Control and Prevention. (2014). Prevalence of autism spectrum disorders among children

- aged 8 years—Autism and developmental disabilities monitoring network, 11 sites, United States, 2010. *Morbidity and Mortality Weekly Report, Surveillance Summaries*, 63(SS02), 1–21.
- Chaaya, M., Saab, D., Maalouf, F. T., & Boustany, R. (2016). Prevalence of autism Spectrum disorder in nurseries in Lebanon: A cross sectional study. *Journal of Autism and Developmental Disorders*, 46(2), 514–522. <https://doi.org/10.1007/s10803-015-2590-7>
- Daou, N. (2014). Conducting Behavioral research with children attending Nonbehavioral intervention programs for autism: The case of Lebanon. *Behavior Analysis in Practice*, 7(2), 78–90. <https://doi.org/10.1007/s40617-014-0017-0>
- Eapen, V., Mabrouk, A. A., Zoubeydi, T., & Yunis, F. (2007). Prevalence of pervasive developmental disorders in preschool children in the UAE. *Journal of Tropical Pediatrics*, 53(3), 202–205. <https://doi.org/10.1093/tropej/fml091>
- El Sabbagh, M., Divan, G., Koh, Y. J., Kim, Y. S., Kauchali, S., Marcín, C., Montiel-Nava, C., Patel, V., Paula, C. S., Wang, C., Yasamy, M. T., & Fombonne, E. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research*, 5, 160–179.
- Hamadé, A., Salameh, P., Medlej-Hashim, M., Hajj-Moussa, E., Saadallah-Zeidan, N., & Rizk, F. (2013). Autism in children and correlates in Lebanon: A pilot case-control study. *Journal of Research in Health Sciences*, 13(2), 119–124.
- Hreich, E., Messarra, C., Roux, S., Barthélémy, C., & Richa, S. (2016). Validation in Arabic of the revised autistic behavior summarized evaluation scale (BSE-R). *Encephale*. <https://doi.org/10.1016/j.encep.2016.04.013>
- Kouba-Hreich, E., Henri, G., & Megarbane, A. (2001). Speech pathology intervention for autistic children in Lebanon: Status, needs and perspectives. *Journal Medical Libanais*, 49(3), 130–131.
- Kourtian, S., Soueid, J., Makhoul, N., Guisso, D. R., Chahrour, M., & Boustany, R. N. (2017). Candidate genes for inherited autism susceptibility in the Lebanese population. *Scientific Reports*, 7, 45336. <https://doi.org/10.1038/srep45336>
- Lelord, G., Barthélémy, C., Martineau, J., Bruneau, N., Garreau, G., & Hameury, L. (1991). Free acquisition, free imitation, physiological curiosity and exchange and development therapies in autistic children. *Brain Dysfunction*, 4, 335–347.
- Mégarbané, A. (1996). *Mieux Comprendre la Trisomie 21*. Librairie Samir: Beirut.
- Mesibov, G., Shea, V., & Schopler, E. (2005). *The TEACCH approach to autism Spectrum disorders, Issues in clinical child psychology series*. New York: Springer.
- Obeid, R., Daou, N., DeNigris, D., Shane-Simpson, C., Brooks, P. J., & Gillespie-Lynch, K. (2015). A cross-cultural comparison of knowledge and stigma associated with autism spectrum disorder among college students in Lebanon and the United States. *Journal of Autism and Developmental Disorders*, 45(11), 3520–3536. <https://doi.org/10.1007/s10803-015-2499-1>
- Raad, H. (2000). Comparative study on the way in which special educational needs is defined in the UK and Lebanon. Evaluation of strengths and weaknesses of each country's approach and presentation of modifications. Unpublished Masters' essay, University of Leeds, UK.
- Richa, S., Fahed, M., Khoury, E., & Mishara, B. (2014). Suicide in autism spectrum disorders. *Archives of Suicide Research*, 18(4, 327), –39. <https://doi.org/10.1080/13811118.2013.824834>
- Soueid, J., Kourtian, S., Makhoul, N. J., Makoukji, J., Haddad, S., Ghanem, S. S., Kobeissy, F., & Boustany, R. (2016). RYR2, PTDSS1 and AREG genes are implicated in a Lebanese population-based study of copy number variation in autism. *Scientific Reports*, 6, 19088. <https://doi.org/10.1038/srep19088>

Legal Competency

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Definition

Legal Competency

Legal Proceedings

The 1960 Supreme Court decision in *Dusky v. United States* articulated the following standard of legal competence: “[T]he test must be whether he has sufficient present ability to consult with his lawyer with a reasonable degree of rational understanding and whether he has a rational as well as factual understanding of the proceedings” This legal standard has been cited in numerous cases since *Dusky* and thus appears as a durable definition. That said, the exact standard does vary by jurisdiction. All jurisdictions agree that the definition may not be a rigid one. Legally, it defies a precise “one size fits all” definition.

Medical Decisions

Healthcare professionals assess the patient's capacity to give informed consent or make

informed decisions based on the patient's ability to understand the treatment, the alternatives, and the risks associated with all options. The mere presence of a cognitive disorder is not, in itself, sufficient grounds to declare the patient incompetent.

No one definition encompasses legal competency. Concluding whether one is legally competent depends on the application of a balancing test of various factors. Four main factors to be considered are the ability to communicate a choice, to understand the relevant information, to appreciate the nature of the situation and its likely consequences, and to manipulate information rationally:

- § Inability to communicate means that the individual is unable to relay his or her treatment wishes either because of inadequate communication skills, or other circumstances such as being in a coma or a vegetative state.
- § The ability to understand the relevant information does not require that the individual understand the situation entirely but only that the person understands each component of the situation when viewed individually.
- § A patient must understand and accept his or her own ailment and the consequences of different treatments and the option to not treat. Any delusional ideas he or she exhibits regarding other issues do not render the patient incompetent to make decisions pertaining to his or her own treatment.
- § The individual's ability to manipulate information rationally does not require that the he or she reach a particular outcome or conclusion, only that the individual logically "compare [s] the risks and benefits of treatment options."

Caveat

Declaring a person incompetent in any setting raises questions of personal liberty. Rights granted by the constitution are taken very seriously in legal settings, and courts are reluctant to deprive liberties by way of court-appointed guardianship or ordered treatment. When a patient is unable to care for himself or herself and lacks the requisite mental capacity to make informed decisions, a declaration of incompetence may be appropriate so long as it is properly substantiated by medical

documentation of the patient's cognitive and/or physical deficits.

See Also

- ▶ [Consent](#)
- ▶ [Dispute Resolution Procedures](#)
- ▶ [Eligibility \(for Services Under IDEA/ADA, etc.\)](#)
- ▶ [Informed Consent](#)

References and Reading

- Appelbaum, P. S., Berg, J. W., & Grisso, T. (1996). Constructing competence: Formulating standards of legal competence to make medical decisions. *Rutgers Law Review*, 48, 345–396.
- Dusky v. United States, 362 U.S. at 402 (1960).
- Garner, B. A. (Ed.). (2009). *Black's law dictionary* (9th ed.). St. Paul: West Publishing.
- Hull, A. (1989). The mentally ill's right to refuse drug treatment: A Panacea or a bitter pill to swallow. *Washburn Law Journal*, 29, 62–105.
- Weyrauch, S. (2000). Decision making for incompetent patients: Who decides and by what standards? *Tulsa Law Journal*, 35, 765–789.

Legal System

- ▶ [Autism in the Courtroom](#)

Legal System Involvement

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An individual with an autism spectrum disorder (ASD) may come into contact with the criminal justice system (CJS) as either a victim or perpetrator of a proven or alleged offense as discussed

in detail elsewhere (Dein and Woodbury-Smith 2010, and references therein; Woodbury-Smith 2013). In brief, there is a body of research data, principally case study in nature, which describes individuals with ASD who have engaged in unlawful behavior and among whom a direct association between this behavior and their ASD is claimed. While the prevalence of unlawful behavior in this population is not well understood, and hence a true association in epidemiological terms has not been established, this remains an important topic. Indeed, whether or not there is an association, such individuals are likely to be vulnerable in the CJS, and for the health professional assessing such individuals, a high level of expertise is demanded in order to unravel the relevant risk factors and thereby make treatment recommendations to reduce future risk. Of course, contact with the CJS may not only follow committing illegal acts but also as a result of falling foul to the illegal acts of others, and it is well established that individuals with developmental disabilities, including ASD, are particularly vulnerable in this regard.

With these points in mind, this current chapter will present a clinically focused consideration of the interface between the CJS and ASD. After a discussion of the factors that are most likely to mediate unlawful behavior among those with ASD, based in part on the author's own clinical experience, the assessment process will be described and then management is considered. For a detailed discussion of the background literature, the reader is referred to the accompanying chapter in this volume, ► [Violent/Criminal Behavior in Autism](#) (Woodbury-Smith 2013).

Throughout, the term "unlawful behavior" will be used for any behavior that is inconsistent with the statute of the jurisdiction in which it occurred. Additionally, and in line with DSM-5 (American Psychiatric Association 2013), the term ASD will be used to describe individuals with developmental impairments in the domains of socio-communicative function and a tendency for ritualistic and repetitive patterns of behavior. As such, the previously used diagnostic label "Asperger syndrome" will be subsumed in this category.

Factors Leading to Contact with the Criminal Justice System

As a victim: People with ASD may come into contact with the CJS as victims or perpetrators. While much of the focus on the literature is on individuals with ASD as perpetrators of unlawful behavior (discussed subsequently), there is a real risk that someone with this diagnosis may be a victim. Their vulnerability in this context can be understood as arising from the interaction between a multitude of factors. First, individuals with ASD may struggle with the rules of social interaction and, therefore, may not appreciate fully when others break these rules by, for example, invading their personal space or encouraging them to engage in behavior that may be illegal. This is compounded by the fact that the social vulnerabilities of the person with ASD may often be apparent to others, even if the diagnosis is not. As such, this may be a red flag for individuals with a criminal predilection to take advantage of them. Further, people with ASD may be lonely as a result of their oftentimes social isolation and hence may have a lower threshold for accepting another individual as a friend, even when it is apparent that this other person is treating them badly or taking advantage of them. Finally, people with ASD are probably more likely than others to take people at face value as a result of their mentalizing impairments.

The tendency for rigid and ritualistic patterns of behavior may also lead to situations where the person with ASD becomes a victim. For example, individuals with ASD often have a strong sense of "right" and "wrong" and may voice these to another person who they deem to be in violation of these strong principles. While in general terms this may simply result in argumentative confrontation with others, on occasion the outcome has been more severe. One example from the recent media is of a young male with ASD who confronted a cyclist for illegally riding on the sidewalk, which resulted in the cyclist assaulting him and inflicting a major head injury resulting in his death.

Indeed, it has only been in relatively recent years that people with ASD have been explicitly

taught social skills, and more general life skills, and therefore it is unusual for a child or young adult with ASD to be “street smart.” Negotiating the social milieu in everyday terms involves much more than can be taught didactically in a one-to-one or group setting, and approximating reality invariably requires real-life exposure to social situations. For example, children and young adults need to negotiate the subtleties of everyday communication and the age-specific street vernacular as well as the rules of acceptable social behavior, arguably difficult to achieve in a classroom setting. Consideration of these different factors clearly requires a more ecologically valid treatment approach to social skills.

As a perpetrator: People with ASD may engage in unlawful behavior for a variety of reasons, and, as in the general population, the interaction between these various factors is often complex and the ultimate reason may be not entirely clear. A variety of offenses have been described in the literature (Woodbury-Smith 2013; Mouridsen 2012), although due to publication bias, these probably represent the more severe end of the spectrum. In more general terms, relatively minor transgressions will be more common, such as inappropriately touching someone’s hair in the context of a sensory preoccupation, stealing in the pursuit of a circumscribed interest, or being hostile in the context of bullying (Wing 1997).

Undoubtedly, the social factors that may mediate victimization may also play a role in the perpetration of unlawful behavior by individuals with ASD. The person with ASD may find themselves socially isolated yet have a wish for relationships with others, leading to feelings of loneliness. They may, therefore, seek out relationships, but do so in an inappropriate manner (Murrie et al. 2002; Stokes et al. 2007). This may involve, for example, following a person because of an inability to initiate verbal contact with them. In the extreme, this may become stalking, even when harmful intent is absent. Similarly, a person with ASD may attempt intimacy, either verbally or nonverbally. However, they may have difficulty interpreting the response given by the other person, who, if disinterested, may show irritability or offense on their face or make a sarcastic comment.

These cues, whether they be facial expression, posture, or verbal in nature, would usually act to inhibit ongoing behavior, but in the absence of an ability to “read” such cues, the behavior may continue unabated and ultimately may transgress acceptable boundaries. Of course, the lack of social experience that may coexist with these social vulnerabilities may compound any social difficulties, and the individual may act toward others in a way they have learnt from books or have observed in the visual media.

There are, of course, other factors that may mediate the perpetration of criminal behavior, such as those related to circumscribed patterns of interest or otherwise rigid behavior (Woodbury-Smith et al. 2010) and those related to risk factors in the population at large (Farrington 1995). Three specific problematic behaviors and their mediating factors demand special attention: harassment and stalking, threats to kill, and behavior related to circumscribed patterns of interest.

ASD, social impairment, and stalking: Cases have been described in both the scientific literature and the media of individuals purported to have ASD who have engaged, or are alleged to have engaged, in harassment and stalking behaviors (Barry-Walsh and Mullen 2004; Stokes and Newton 2004). Harassment and stalking describe the persistent approaches of someone toward another, despite clear signals by the victim that these approaches are unwanted. These approaches may involve physically following the person or repeatedly contacting the person by, for example, phone, text, or email or through unwanted gifts. Such behaviors may occasionally be seen among adolescents and adults with ASD, but generally there is no intended threat of harm. The behavior is often driven by the desire for a romantic relationship with the person concerned, yet barriers exist that interfere with this. For example, there may be no easy opportunity for the individual to be physically in close proximity to the other person, particularly if the person with ASD is socially isolated. Moreover, they may not know how to appropriately initiate or sustain conversation that makes it clear that they have romantic aspirations toward this person. Indeed, in one study, the only significant predictor of romantic functioning

among those with ASD was their social functioning (Stokes et al. 2007).

These vulnerabilities are compounded by difficulty interpreting whether the person concerned, through verbal or nonverbal means, is romantically “interested.” Again, in the study of Stokes and colleagues (2007), individuals with ASD were observed to persist for significantly longer periods of time than typical adolescents when they received negative or no response from the person concerned. A tendency for repetitiveness may result in persistent efforts irrespective of the response given to them. Some of these factors have previously been identified among a study of stalkers more generally (Mullen et al. 1999). In particular, intimacy seeking and romantically “incompetent” (their terminology) represented two of several factors mediating stalking behavior in one sample of 145 stalkers referred for forensic evaluation.

ASD, social impairment, and threats to kill:

There is also a risk that individuals with ASD may make threats to kill or otherwise do harm to others, in some cases acting out on such thoughts (Murrie et al. 2002). It is not unusual for a child who is being victimized by, for example, peers to harbor thoughts of wanting to harm these others, and these thoughts are occasionally verbalized, or written down, and hence come to the attention of family, teachers, or health professionals (Wing 1997). It is important to realize that a relatively innocuous snub made by, for example, a passerby in the street may result in a catastrophic reaction among individuals with ASD. Our current social climate is characterized by several high-profile cases of “angry” adolescents and young adults who have harmed their peers, and, therefore, as a result, any threats are understandably taken very seriously, even though among those with ASD the intent to act out on such thoughts may generally not be present. Bullying will be a significant risk factor for such threats, but more general factors will likely include misinterpretation of others’ vocalizations or gestures, a sensitivity to the comments made by others, and difficulty with emotional regulation, such that the distress in these situations is acted on without thinking (or necessarily knowing) the potential consequences.

ASDs and circumscribed interests: The possibility that the nature of a person’s circumscribed patterns of interest may mediate unlawful behavior has been written about widely (Woodbury-Smith et al. 2010; Barry-Walsh and Mullen 2004; Murrie et al. 2002). A number of case studies have been described in the literature alleging a link between the nature of the interest and the unlawful behaviors observed. These include both interests that are not inherently unlawful, but the pursuit of which has resulted in illegal behavior and interests that are violent or involve a violent theme.

Assessment of an Individual with ASD in the Criminal Justice System Setting

In what follows, it will be assumed that the person in question, whether an adolescent or young adult, is a diagnosed individual with ASD who has been referred after alleged unlawful behavior. In situations where a person is suspected of having an ASD, an additional layer of assessment is required, i.e., diagnosis. While diagnosis is discussed elsewhere in this volume, one crucial point to consider is that whatever the age of the suspect, it is vital that an informant supplies relevant developmental information. It is neither appropriate nor diagnostically valid to take a cross-sectional history and base the diagnosis on this. ASD is, by definition, of developmental onset. Moreover, it is also important to realize that socio-communicative difficulties in young adulthood are common, particularly among those in the CJS, and their origin is often multifactorial.

Beyond diagnosis, it may also be useful to undertake a formal neuropsychological assessment (Clare and Woodbury-Smith 2009). This may include an assessment of both general intellectual ability and level of adaptive skills, as well as more specific areas of cognition, such as social cognition, language, and executive function. There are now a number of commercially available established tests for the assessment of cognitive domains such as these, and these tests have demonstrated good reliability and validity properties across specific age ranges and in particular

populations of individuals. A full description of these tests is out the scope of this current chapter, and the reader is referred to appropriate specialized texts for a full description.

The rationale for completing tests such as these is severalfold. First, they allow for the quantification of the exact degree of impairment. Although it is possible during the clinical assessment to identify the areas of vulnerability, and illustrate these with specific examples, it is sometimes difficult to objectively quantify the extent of the impairment, and the degree to which this is different from same age peers. It is also important to characterize the person's areas of relative strength and vulnerability, as this will be relevant when devising treatment strategies. In general terms, areas of strength are "used" as far as possible to develop strategies to address the vulnerabilities. Finally, it is important to gain an understanding of the person's level of functioning, which is usually gleaned from a measure of global intelligence in combination with a measure of adaptive functioning. Discrepancies between intellectual capacity and level of everyday skills are usually the result of other cognitive impairments (such as social cognition).

In addition to the formal assessments, and where appropriate diagnosis, time will be spent interviewing the individual. As with any clinical interview, this will allow the interviewee to give their own account of events as well as the interviewer to explore possible antecedents and contextual factors. In particular, living and employment (or educational) circumstances can be explored, as well as relationships and personal attitudes and beliefs. This also gives the opportunity to ascertain whether there is any evidence of mental health comorbidity, either in the form of additional neurodevelopmental disorders (such as ADHD or specific learning difficulties) or mental illness (such as depression, anxiety, or psychosis). If necessary, a specialist mental health assessment will need to be sought, and additional formal measures can be used such as the *Beck Depression Inventory* (Beck et al. 1996) and/or the *Beck Anxiety Inventory* (Beck et al. 1988).

Information obtained from a clinical interview may be augmented by specific measures related to

sexual knowledge and social networks. Social networks, in particular, may play a key role in both the etiology of problematic behavior and also in rehabilitation and recovery, and so collecting detailed information on this is particularly important. The person's knowledge of relationships can be ascertained using questions from the ADOS-G modules 3 and 4 (Lord et al. 2000), and their understanding of consent and abuse and their more general sexual knowledge (also useful when interviewing alleged victims) can be obtained using the methods of Murphy and O'Callaghan (2004). Finally, information regarding the person's social support network can be facilitated using a scale such as the "Significant Others Scale" (Power et al. 1988).

It is important to realize that the vulnerabilities of the individual with an ASD may not be immediately apparent to criminal justice system workers, particularly if they function intellectually in the typical range (Allen et al. 2008; Browning and Caulfield 2011). Unlike those with global cognitive delays, who may have language limitations or whose facial appearance (or stature) may act as immediate red flags, recognition of a higher functioning individual may be more problematic, oftentimes compounded by the tendency for such individuals to mask or play down their vulnerabilities, wishing to appear "normal." Beyond their recognition, people with ASD may also be vulnerable during the police interview and during the subsequent trial. These issues are discussed in the subsequent sections.

People with ASD and vulnerability during the police interview: It has long been recognized that people with developmental disabilities, including ASD, are vulnerable when being interviewed in the criminal justice system setting (Allen et al. 2008; Browning and Caulfield 2011). People with ASD have difficulties understanding irony, sarcasm, and, more generally, the "unrevealed" meaning of questions and statements and may therefore misinterpret what is being said to them or asked of them. This is compounded by the fact that, their disability may not be immediately apparent to the untrained eye, which may consequently assume an intact understanding. Any peculiarities of behavior on the part of the ASD

suspect may be misinterpreted as purposeful evasion or obstruction to the process of the interview. There are now measures in place to try to raise awareness of ASD among criminal justice workers such that these individuals are recognized as early in the process as possible.

Related concerns include those of acquiescence and suggestibility during the police interview or in the courtroom during the criminal justice process (Maras and Bowler 2012a, b; North et al. 2008). Acquiescence refers to a tendency to agree to what is being asked or said because the person thinks that this is expected of them. Acquiescence is characterized by a wish to please others by saying what they think the other person wants to hear, whether or not it is a true account of reality. This may occur if the interviewee wishes the interview to be over as quickly as possible or may be motivated by a wish simply to be liked by the interviewer. Suggestibility on the other hand is generally employed to describe the uncritical acceptance of information being offered or susceptibility to misleading information and may have a more complex origin. For example, the questioning style of the interviewer (who may use leading or closed questions) may influence the responses given. The types of language used, particularly language that involves the use of metaphor, irony, and sarcasm, may also influence the accuracy of the responses offered. Moreover, poor problem-solving capacity when thinking on one's feet has been hypothesized to result in a greater tendency to rely on immediate linguistic and social cues rather than the person's actual recall of events.

The assessment of acquiescence and suggestibility will determine the extent to which the individual may be vulnerable during the interview. There are established methods to measure these vulnerabilities, perhaps the widest used being the Gudjonsson Suggestibility Scale (Gudjonsson 1984). Interestingly, this scale has been used previously among adults with ASD and failed to find any tendency for acquiescence or suggestibility compared to a matched group (Maras and Bowler 2012b), although conflicting results have also been reported (North et al. 2008). Similarly, children with ASD have also been shown to be no

more acquiescent or suggestible than their peers (McCrory et al. 2007). However, in this study, gist memory was demonstrated to be impaired among those with ASD, and they did rely more on direct questions, showing vulnerability with free recall.

The trial itself may be a stressful experience for an individual with ASD whether they are a witness or a defendant. Specifically, however, fitness to plead and stand trial can be formally assessed using a fairly well-established series of questions:

- Do you know what the police say you have done?
- Do you know the difference between saying "guilty" and "not guilty"?
- What is your lawyer for?
- Can you tell your lawyer your side of things?
- Do you know what it means to say you can object to people on your jury?
- What happens in court?

Management Issues: Overview

For individuals with ASD who are involved in the criminal justice system, there may be an opportunity for diversion into clinical services that offer rehabilitation. This will, of course, depend on the nature of the offense and the availability of services but ultimately rests with the judicial authority. In the real world, many transgressions will be relatively minor, affording the treating team the opportunity to intervene before anything serious happens. In these situations, there are a number of interventions that may be effective at reducing the risk of subsequent behavior of greater severity.

Reducing risk through management of socio-communicative vulnerabilities: Social skills training may be beneficial for rehabilitation. This affords the opportunity for individuals who are otherwise isolated to meet other people, as well as provide a forum to explicitly teach positive social skills and increase awareness of social *faux pas* or otherwise socially inappropriate behavior. The empirical support for social skills training is far from complete, and the efficacy of social groups vis-à-vis explicit social skills training needs further elaboration (for a critical review

of social skills training for children, see, for example, White et al. 2007 and for adults Bishop-Fitzpatrick et al. 2013). One-to-one therapy may be more beneficial for certain individuals, particularly those who are resistant to the group setting or those who have engaged in more serious behavior. There is some indication that one-to-one therapy using cognitive behavior therapy (CBT) may be beneficial at reducing preoccupations with violent and sexual themes (Barry-Walsh and Mullen 2004) as well as reducing the overall severity of the socio-communicative impairments in ASD (Wood et al. 2009).

Reducing risk through facilitating empathy skills: The ability to “read” simple (happy, sad, and so forth) and complex (sarcasm, irony, and flirtation) emotions in others fundamentally mediates our interactions and will arguably play a role in some of the socially inappropriate and unlawful behaviors seen in ASD. Indeed, the ability to empathize in the population at large is widely believed to protect individuals from victimizing others (Miller and Eisenberg 1988), and an inability to recognize emotions in others has been shown to be associated with offending among individuals with psychopathy (Blair 2001) and individuals with ASD (Woodbury-Smith et al. 2005). It seems reasonable to expect, therefore, that by engendering better empathy skills among people with ASD, their risk of unlawful or otherwise inappropriate behavior will be reduced. There are several empathy training programs that are commercially available, including the *Mind Reading* software package for children and adults (Golan and Baron-Cohen 2006) and the *Transporters* package for children (Baron-Cohen et al. 2009), which are useful for improving the ability to recognize emotions in others. Other approaches attempt to develop perspective taking through role play or the use of videotaped scenarios depicting different interpersonal situations.

Reducing risk through management of comorbidities: The frequency of mental health comorbidity and additional developmental diagnoses is particularly high in this population, and the management of these must also be undertaken using appropriate medical, psychological, and social interventions.

Management: Specific Issues

Aggression: The management of aggression requires a clear understanding of the nature, and interaction between, the different predisposing, precipitating, and perpetuating factors in the medical, psychological, and social domains for the episodes of violence observed. This information may then be used to guide treatment. For example, social factors, such as life events, can be managed through the identification of these events and the implementation of support and active therapy where necessary. This may be true of factors such as loss and bereavement, which would require supportive psychotherapy, and for any unanticipated change in the person’s routines and environment, which may require restoration of a mutually agreed routine as far as is possible. Similarly, psychological factors such as poor judgment and problem-solving skills may benefit from executive building strategies (Cicerone et al. 2011); a lack of empathy and poor emotion recognition and “mentalizing” impairments may benefit from cognitive behavior strategies or more explicit techniques to develop emotional awareness of self and others (Golan and Baron-Cohen 2006); and a low tolerance threshold may benefit from anger management (Day et al. 2008). Although medical factors themselves may not play a huge role in mediating aggressive behavior, the medical management of aggression may include psychopharmacological interventions. The effectiveness of different treatment modalities for violent behavior has been reviewed in detail elsewhere (McGuire 2008).

Stalking and unlawful sexual behavior: Since so little is known about the factors that are associated with sexual offending (in broad terms, i.e., including stalking and harassment) in this population, specific treatments are yet to be developed. Stalking and harassment require some specific management strategies as discussed elsewhere (e.g., see Mullen et al. 2001). In particular, there is an immediate requirement to ensure physical distance between the two parties and, once established, to then stop the behavior and prevent its reemergence. If the behavior is driven by a belief that the person reciprocates their desire for

a relationship, then this will require a therapeutic approach with a particular focus on developing skills to interpret the verbal and nonverbal communication of others. If the behavior is principally driven by the desire for a relationship, then an approach that includes sexual and relationship education is warranted. This may be delivered in a group setting as a didactic education exercise and may be facilitated by the use of social stories (Tarnai and Wolfe 2008). There is also a sexual education program specifically designed for adults with ASD (Hénault 2005), which is a 12-session program covering the key areas of sexual knowledge.

Beyond sexual education, for those who have been convicted of sexual offenses, community and inpatient sex offender treatment programs do exist, and in addition to education, these focus on the development of skills through role play and the reduction of risk through aversion therapy. The group-based nature of these programs reduces the risk of denial and minimization, and their cognitive behavioral basis allows cognitive distortions and victim empathy to be developed. These same strategies may be reasonably applied to those with ASD, but including such people in sex offender treatment groups either in the community or in a custodial setting may be inappropriate. In particular, their level of understanding may impact their ability to keep apace with their peers, and so a modified program may be required.

People with ASDs who make threats: The management of people who make threats will depend on the individual circumstances and the assessment of ongoing risk (and risk of escalation). The immediate aim is to reduce the risk of actual harm, and therefore physical distance must be established and maintained between the two parties involved if at all possible. Once the risk of actual harm is reduced, focus can turn to managing the context (e.g., bullying if present) as well as reducing ongoing risk through engagement with the victimizer. This will usually involve teaching executive function (i.e., problem-solving and good judgment) skills to deal with difficult situations and risk reduction through cognitive interviewing that focuses on the potential consequences for the person if they were to do harm to

another. Methods to develop problem-solving and good judgment skills originate principally in the rehabilitation of individuals with acquired brain injury and are discussed in detail elsewhere (see, e.g., Cicerone et al. 2011, and references therein).

Circumscribed interests and unlawful behavior: Reducing the risk of unlawful behavior in the context of a person's circumscribed interest requires consideration of the purpose of the interest for the person concerned. Notably, interests often maintain self-esteem, and to simply introduce strategies to extinguish a person's interest may make matters worse. Instead, measures must be taken to "contain" the interest within acceptable boundaries and "mold" the interest into something more appropriate. For example, a preoccupation with firefighters that results in repetitively calling 911 can be replaced with books or DVDs containing scenes of firefighters in action. Similarly, for those with an interest in a potentially violent theme (such as weapons or infamous historical figures such as Adolf Hitler), the focus will be on "containing" the interest within acceptable boundaries (such as discouraging talking with others about the interest) as well as attempts to replace the interest with opportunities for an "ordinary life."

Conclusions

This chapter has considered some of the factors that may lead someone with an ASD to come into contact with the criminal justice system and then presented a discussion of the assessment and management of such individuals. There are clearly a number of different factors that may be involved, singularly or in combination, and the only way to fully realize these is through detailed assessment. One of the issues that remain, however, is the paucity of available specialist treatment programs for this population, either in the community, in a mental health inpatient setting, or in the criminal justice system. Paralleling this is the limited availability of research evidence outside of case studies or small case series. Ultimately, service development will require dialogue between social and mental health services and the criminal justice system.

See Also

► Violent/Criminal Behavior in Autism

References and Reading

- Allen, D., Evans, C., Hider, A., Hawkins, S., Peckett, H., & Morgan, H. (2008). Offending behaviour in adults with Asperger syndrome. *Journal of Autism and Developmental Disorders*, 38, 748–758.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: APA.
- Baron-Cohen, S., Golan, O., & Ashwin, E. (2009). Can emotion recognition be taught to children with autism spectrum conditions? *Philosophical Transactions of the Royal Society, B: Biological Sciences*, 364, 3567–3574.
- Barry-Walsh, J. B., & Mullen, P. E. (2004). Forensic aspects of Asperger's syndrome. *Journal of Forensic Psychiatry and Psychology*, 15, 96–107.
- Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting & Clinical Psychology*, 56, 893–897.
- Beck, A. T., Steer, R. A., Ball, R., & Ranieri, W. (1996). Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *Journal of Personality Assessment*, 67, 588–597.
- Bishop-Fitzpatrick, L., Minshew, N. J., & Eack, S. M. (2013). A systematic review of psychosocial interventions for adults with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43, 687–694.
- Blair, R. J. R. (2001). Neurocognitive models of aggression, the antisocial personality disorders, and psychopathy. *Journal of Neurology, Neurosurgery & Psychiatry*, 71, 727–731.
- Browning, A., & Caulfield, L. (2011). The prevalence and treatment of people with Asperger's syndrome in the criminal justice system. *Criminology and Criminal Justice*, 11, 165–180.
- Cicerone, K. D., Langenbahn, D. M., Braden, C., Malec, J. F., Kalmar, K., Fraas, M., Felicetti, T., Laatsch, L., Harley, J. P., Bergquist, T., & others. (2011). Evidence-based cognitive rehabilitation: Updated review of the literature from 2003 through 2008. *Archives of Physical Medicine and Rehabilitation*, 92, 519–530.
- Clare, I. C., & Woodbury-Smith, M. (2009). Autism spectrum conditions. In S. Young, M. Kopelman, & G. Gudjonsson (Eds.), *Forensic neuropsychology in practice*. Oxford: Oxford University Press.
- Day, A., Howells, K., Mohr, P., Schall, E., & Gerace, A. (2008). The development of CBT programmes for anger: The role of interventions to promote perspective-taking skills. *Behavioural and Cognitive Psychotherapy*, 36, 299–312.
- Dein, K., & Woodbury-Smith, M. (2010). Asperger syndrome and criminal behaviour. *Advances in Psychiatric Treatment*, 16, 37–43.
- Farrington, D. P. (1995). The Twelfth Jack Tizard Memorial Lecture. The development of offending and antisocial behaviour from childhood: Key findings from the Cambridge Study in Delinquent Development. *Journal of Child Psychology & Psychiatry*, 36, 929–964.
- Golan, O., & Baron-Cohen, S. (2006). Systemizing empathy: Teaching adults with Asperger syndrome or high-functioning autism to recognize complex emotions using interactive multimedia. *Development and Psychopathology*, 18, 591–617.
- Gudjonsson, G. H. (1984). A new scale of interrogative suggestibility. *Personality and Individual Differences*, 5, 303–314.
- Hénault, I. (2005). *Asperger's syndrome and sexuality: From adolescence through adulthood*. London: Jessica Kingsley.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Leventhal, B. L., DiLavore, P. C., Pickles, A., & Rutter, M. (2000). The autism diagnostic observation schedule-generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism & Developmental Disorders*, 30, 205–223.
- Maras, K. L., & Bowler, D. M. (2012a). Eyewitness testimony in autism spectrum disorder: A review. *Journal of Autism and Developmental Disorders*, 44(11), 2682–97.
- Maras, K. L., & Bowler, D. M. (2012b). Brief report: Suggestibility, compliance and psychological traits in high-functioning adults with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 6, 1168–1175.
- McCrorry, E., Henry, L. A., & Happé, F. (2007). Eye-witness memory and suggestibility in children with Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 48, 482–489.
- McGuire, J. (2008). A review of effective interventions for reducing aggression and violence. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, 363, 2577–2597.
- Miller, P. A., & Eisenberg, N. (1988). The relation of empathy to aggressive and externalizing/antisocial behavior. *Psychological Bulletin*, 103, 324.
- Mouridsen, S. E. (2012). Current status of research on autism spectrum disorders and offending. *Research in Autism Spectrum Disorders*, 6, 79–86.
- Mullen, P. E., Pathé, M., Purcell, R., & Stuart, G. W. (1999). Study of stalkers. *American Journal of Psychiatry*, 156, 1244–1249.
- Mullen, P. E., Pathé, M., & Purcell, R. (2001). The management of stalkers. *Advances in Psychiatric Treatment*, 7, 335–342.
- Murphy, G. H., & O'Callaghan, A. (2004). Capacity of adults with intellectual disabilities to consent to sexual relationships. *Psychological Medicine*, 34, 1347–1357.
- Murrie, D. C., Warren, J. I., Kristiansson, M., & Dietz, P. E. (2002). Asperger's syndrome in forensic settings. *International Journal of Forensic Mental Health*, 1, 59–70.
- North, A. S., Russell, A. J., & Gudjonsson, G. H. (2008). High functioning autism spectrum disorders: An

- investigation of psychological vulnerabilities during interrogative interview. *The Journal of Forensic Psychiatry & Psychology*, 19, 323–334.
- Power, M., Champion, L., & Aris, S. (1988). The development of a measure of social support: The Significant Others (SOS) Scale. *British Journal of Clinical Psychology*, 27, 349–358.
- Stokes, M., & Newton, N. (2004). Autistic spectrum disorders and stalking. *Autism*, 8, 337–339.
- Stokes, M., Newton, N., & Kaur, A. (2007). Stalking, and social and romantic functioning among adolescents and adults with autism spectrum disorder. *Journal of Autism & Developmental Disorders*, 37, 1969–1986.
- Tarnai, B., & Wolfe, P. S. (2008). Social stories for sexuality education for persons with autism/pervasive developmental disorder. *Sexuality and Disability*, 26, 29–36.
- White, S. W., Keonig, K., & Scahill, L. (2007). Social skills development in children with autism spectrum disorders: A review of the intervention research. *Journal of Autism and Developmental Disorders*, 37, 1858–1868.
- Wing, L. (1997). Asperger's syndrome: Management requires diagnosis. *Journal of Forensic Psychiatry*, 8, 253–257.
- Wood, J. J., Drahota, A., Sze, K., Van Dyke, M., Decker, K., Fujii, C., Bahng, C., Renno, P., Hwang, W.-C., & Spiker, M. (2009). Brief report: Effects of cognitive behavioral therapy on parent-reported autism symptoms in school-age children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 39, 1608–1612.
- Woodbury-Smith, M. (2013). Violent/criminal behavior in autism. In F. Volkmar (Ed.), *Encyclopedia of autism spectrum disorders*. New York: Springer.
- Woodbury-Smith, M., Clare, I. C., Holland, A., Kearns, A., Staufenberg, E., & Watson, P. (2005). A case-control study of offenders with high functioning autistic spectrum disorders. *Journal of Forensic Psychiatry and Psychology*, 16, 747–763.
- Woodbury-Smith, M., Clare, I. C., Holland, A., Kearns, A., Staufenberg, E., & Watson, P. (2010). Circumscribed interests among offenders with autistic spectrum disorders: A case-control study. *Journal of Forensic Psychiatry and Psychology*, 21, 366–377.

Leigh Disease

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Synonyms

Infantile subacute necrotizing encephalopathy;
Juvenile subacute necrotizing encephalopathy;

Leigh necrotizing encephalopathy; Leigh syndrome; Leigh's disease; SNE; Subacute necrotizing encephalomyelopathy; Subacute necrotizing encephalopathy

Definition

Leigh disease is a rare neurometabolic disorder characterized by a progressive loss of motor and cognitive abilities. It is estimated to affect 1 in 40,000 newborns, with a higher prevalence in Quebec, Canada (approximately 1 in 2000 newborns) and on the Faroe Islands (approximately 1 in 1700 newborns). The onset of symptoms typically occurs during infancy; although in rare cases, onset occurs during adolescence or adulthood.

The first noticeable signs for Leigh disease are gastrointestinal symptoms (e.g., nausea and recurrent vomiting and diarrhea) and failure to thrive. Additionally, affected individuals may develop severe movement difficulties and experience motor regressions. Symptoms may include: difficulty swallowing (i.e., dysphagia), difficulty coordinating movement (i.e., ataxia), decreased muscle tone (i.e., hypotonia), involuntary muscle contractions (i.e., dystonia), numbness (i.e., peripheral neuropathy), and difficulty articulating speech (i.e., dysarthria).

If the onset of Leigh disease occurs during early childhood, symptoms develop rapidly and lead to the deterioration of the central nervous system. As symptoms become more advanced, lactic acid levels may increase in the blood, brain, urine, or the cerebrospinal fluid. This could cause respiratory, heart, kidney, and further motor impairments. Individuals may also present breathing problems, such as abnormal breathing patterns (i.e., Cheyne-Stokes), temporary cessation of breathing (i.e., apnea), or labored breathing (i.e., dyspnea). This condition may also cause thickening of heart muscle cell (i.e., hypertrophic cardiomyopathy) and degeneration of the optic nerve and extra ocular muscles (i.e., optic atrophy and ophthalmoparesis).

Leigh disease can be caused by over 75 mutations in the nuclear DNA (nDNA)

and – in 20% of cases – in the mitochondrial DNA (mtDNA). These mutations predominantly occur in genes that influence energy production. Specifically, mutations disrupt the protein complexes involved in oxidative phosphorylation and the pyruvate dehydrogenase complex – these protein complexes drive the production of cellular energy (ATP). The reduction in cellular energy disrupts the functioning of the cell; as a result, this deteriorates tissue in the central nervous system and vital organs that require high levels ATP for normal functioning. In rare cases, individuals with autism may also have a mitochondrial disease. This small subset of individuals present with mutations in nDNA and mtDNA that disrupt oxidative phosphorylation. However, there is limited research examining the possible association between autism spectrum disorder and mitochondrial disease.

Individuals with Leigh disease may inherit the condition through different patterns, such as autosomal recessive, mitochondrial inheritance, and X-linked recessive patterns. Autosomal recessive is the most common inheritance pattern. Affected individuals inherit a copy of the mutated gene from each parent. For the X-linked recessive pattern, the mutated gene is located on the X-chromosome. Males with Leigh disease only need one copy of the mutated gene, while females need two altered copies of the gene to have the disease. These inheritance patterns apply to individuals who have mutations in the nDNA. Mutations of genes in mtDNA are mostly inherited through mitochondrial inheritance (i.e., maternal inheritance).

Leigh disease can be diagnosed through laboratory testing, clinical evaluation, neuroimaging, and molecular genetic testing. Prognosis for this condition is poor. Due to the heterogeneity of symptoms, a comprehensive plan is created to address each specific symptom.

See Also

► [Mitochondrial Deficits/Disorders](#)

References and Reading

- Dhillon, S., A Hellings, J., & G Butler, M. (2011). Genetics and mitochondrial abnormalities in autism spectrum disorders: a review. *Current Genomics*, 12, 322–332.
- Finsterer, J. (2008). Leigh and Leigh-like syndrome in children and adults. *Pediatric Neurology*, 39, 223–235.
- Lake, N. J., Compton, A. G., Rahman, S., & Thorburn, D. R. (2016). Leigh syndrome: one disorder, more than 75 monogenic causes. *Annals of Neurology*, 79, 190–203.
- Tiranti, V., Hoertnagel, K., Carrozzo, R., Galimberti, C., Munaro, M., Granatiero, & Bayona-Bafaluy, M. P. (1998). Mutations of SURF-1 in Leigh disease associated with cytochrome c oxidase deficiency. *The American Journal of Human Genetics*, 63, 1609–1621.

Leigh Necrotizing Encephalopathy

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Leigh Syndrome

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Leigh's Disease

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Leisure Participation Patterns for School-Age Youth with Autism

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Definition

Participation in leisure is an integral part of the human experience. Leisure activities are varied,

and participation in leisure is associated with quality of life (QoL) and physical and mental well-being (Potvin et al. 2013). Billstedt et al. (2011) suggested participation in leisure activities as a possible proxy for QoL in individuals with autism spectrum disorders (ASD). Leisure is defined as time spent on activities that are not for survival, work, or sleep (Zeigler 1965). Recreation is defined as engaging in leisure activities for the purpose of pleasure, education, or satisfaction (Zeigler 1965). Across participation literature, the line between leisure and recreation activities is blurry, and differentiating activities between the two categories is not consistent.

Current Knowledge

Participation in leisure provides psychological, spiritual, social, and cultural meaning and value to the human experience (Iwasaki et al. 2018). Iwasaki et al. (2018) describe the role of leisure as providing a joyful, connected, discovered, composed, and empowered life. The authors explain that leisure reduces suffering and provides a joy by the positive emotions elicited. Leisure provides connection via routes of social connectedness and connectedness with nature, religion, and culture. Leisure provides the opportunity to uncover and promote an individual's special talents and characteristics. Leisure allows individuals the opportunities to control their pace and be flexible, promoting control and a sense of autonomy. Finally, engagement in leisure provides healing and stress coping for individuals. These benefits of leisure promote QoL and allow individuals to flourish.

Children with autism spectrum disorders (ASD) report lower QoL (Lin 2014), decreased participation in recreation and leisure (Hilton et al. 2008), and decreased flourishing (Hilton et al. 2019), particularly as they age into adolescence (Ratcliff et al. 2018). Individuals with ASD demonstrate different participation patterns than typically developing individuals and lack regular participation in recreational activities, contributing to poorer QoL (Billstedt et al.

2011). Participation in leisure activities promotes physical and mental health for individuals.

Children, adolescents, and adults with ASD demonstrate poorer mental health with high incidence of comorbidities, such as depression and anxiety (Schall and McDonough 2010), and decreased physical health (Hilton et al. 2014). Children with ASD are at risk for decreased participation in recreational activities (Kaljača et al. 2019). Lack of participation in leisure for individuals with ASD has negative impacts, including poorer mental health, decreased physical fitness, decreased QoL, and inability to flourish. This entry will present the patterns of participation in leisure across participation categories for children with ASD and recommendations for improving participation for children with ASD.

Participation preferences of children with ASD. Several studies have addressed the leisure participation preferences of children with ASD. In one study, the top ten preferences were playing on the computer or video games, watching TV or a rented movie, visiting others, playing with toys, swimming, doing crafts, drawing or coloring, going to a party, playing with pets, playing board or card games, and going to the movies (Eversole et al. 2016). Potvin et al. (2013) had similar findings that children with ASD enjoyed playing computer or video games, watching TV or rented movie, reading, going to a party, visiting others, doing homework, playing with things, shopping, doing crafts, talking on the phone, hanging out, and playing board or card games. The activities enjoyed less by children with ASD from typical peers include physical activities and formal activities (Potvin et al. 2013). An activity that was reported to be enjoyed more was swimming (Eversole et al. 2016).

Participation patterns of children with ASD. Across leisure participation studies, subtypes of activities are inconsistent. They often categorize activities into types of participation based on the types of assessments performed (Hilton et al. 2008; Little et al. 2014; Potvin et al. 2013; Solish et al. 2010). The general trend toward agreement of categories seems to group them into *physical*, *social*, *self-improvement*, and *home-based solitary* activities.

Participation in physical activity. Studies consistently concur that individuals with ASD participate in less physical activity than same-age peers. Several studies have compared the total amount of time spent in moderate and vigorous physical activities. In a study of Irish children, 70.1% of the children with ASD participated in less than 5 days of moderate to vigorous physical activity in a 2-week period compared to 23.9% of the control group (Healy et al. 2017). Stanish et al. (2017) found that adolescents with ASD spent less time in moderate to vigorous physical activity, fewer of them met the daily Physical Activity Guidelines for Americans (USDHHS, 2008), and they participated in fewer physical activities compared to same-age peers. Memari et al. (2015) had similar findings that only 10% of the children with ASD in their study were physically active. McCoy et al. (2016) found that adolescents with ASD were less likely to participate in physical activity compared to same-age peers using a nationally representative dataset.

Limited participation in physical activities has ramifications on physical health, including obesity, high BMI, diabetes, and cardiac health. In a study examining the relationship between physical activity, BMI, and arterial stiffness, children with ASD demonstrated decreased physical activity, higher BMI, and increased arterial stiffness, putting them at greater risk for cardiovascular disease (Heffernan et al. 2018). In another study, children with ASD were 40% more likely to be obese than unaffected children (Lawson and Foster 2016). A study of a nationally representative dataset found that adolescents with ASD were 27% more likely to be overweight and 72% more likely to be obese than typically developing adolescents (McCoy et al. 2016). In the same data set, those adolescents classified as having mild ASD were 56% less likely to engage in regular physical activity, and those classified as having severe ASD were 70% less likely to participate in regular physical activity than the unaffected adolescents. Decreased participation in physical activity results in increased health risks for these children with ASD.

In studies examining participation in formal, organized physical activities, children with ASD

participate in fewer of them compared to their same-age peers. Activities defined as formal physical activities include sports, sports lessons, playing on a team, bicycling, individual physical activity, martial arts, and track and field. Potvin et al. (2013) compared 7–13-year-olds with high-functioning autism and a peer control group and found that the ASD group participated in fewer types of physical activities. Healy et al. (2017) found that children with ASD participated in fewer sports and extracurricular activities than typically developing peers, particularly physical activities with coaches. Taheri et al. (2016), similarly, found that children with ASD and intellectual disability (ID) participated on fewer sports teams.

Participation in organized, formal physical activities is a significant predictor of sports participation and physical fitness activities in young adulthood (Perkins et al. 2004). Additionally, the greater diversity of participation in sport activities predicts better health, higher education levels, and higher activity levels in adulthood (Mäkelä et al. 2016). The decreased participation and lack of diversity that children and adolescents with ASD experience have long-term implications for health and wellness as adults.

Participation in social activities. Participation in social activities consists of those that involve participation with friends; community activities, such as clubs, organizations, volunteering, and worship and religious activities; and organized activities, such as lessons, team sports, and classes. Kaljača et al. (2019) found that children with ASD participate in significantly fewer social activities than typically developing peers. Social activities with particularly limited participation by children with ASD were playing games, birthday parties, playing at a friend's home, having a sleepover, talking on the phone and computer, going to the movies, and going out for meals.

Solish et al. (2010) also found that children with ASD participated in fewer social activities compared to typically developing peers. In a study comparing the social participation of typically developing children, children with ID, and children with ASD and ID, aged 3–19, Taheri et al.

(2016) found that the ID and ASD with ID groups participated in fewer social activities including unstructured play, social outings, special occasions, and community activities. Compared to the ID-only group, the ASD with ID group participated even less in social activities, such as special occasions with friends. In another study, Memari et al. (2015) found that children with ASD spent more time in solitary play than social play.

In a study examining the social participation of young adults with ASD, the characteristics of those with decreased social participation were more likely to be male, from the highest income category, have a higher rate of conversational impairment, live at home with parents, never see friends, never get calls from friends, and never get invited to activities with others (Orsmond et al. 2013). These are consistent with finding from another study (Orsmond et al. 2004), finding that 46.4% of 235 adolescents with ASD reported no peer relationships. Similarly, Taheri et al. (2016) found that children with ASD had significantly fewer school friends and other friends than their typically developing peers and the friendships they did have were of poorer quality.

Decreased social participation is associated with poorer mental health outcomes, such as depression, loneliness, and anxiety. Batsika and Sharpley (2015) found that feelings of depression were associated with lack of self-confidence in social interactions. In a study examining the mediators between autism phenotype and depressive symptomology, social problem-solving was identified as a mediator, along with poor social interactions, perceived social awkwardness, and social anxiety (Jackson and Dritschel 2016). In a similar light, Ratcliffe et al. (2015) found that social skills explained over 40% of the variance in mental health difficulties based on teacher and parent scores for children with ASD. These social difficulties result in limited participation in leisure activities for individuals with ASD from childhood through adulthood. Bohnert et al. (2016) found that decreased participation in organized activity was associated with greater social impairment, depressive symptoms, and loneliness.

Myers et al. (2015) found that higher cognitive function and access to case management

improved social participation, whereas behavioral and communication difficulties were associated with social isolation. Taylor et al. (2016) looked at the relationship from changes in high school to out of high school in structured and unstructured social participation and found that structured activities declined with the transition for most participants and unstructured activities increased for those that were involved in more structured activities during high school. Additionally, those individuals with more internalizing behaviors in high school demonstrated more social isolation outside of high school. Increasing opportunities for participation in social activities improved QoL and decreased loneliness for individuals with ASD. There is a great need for social participation and social activities for individuals with ASD. Jantz (2011) found that ASD support groups provided opportunities for social interaction, information, advice, and structure and resulted in decreased loneliness.

Home-based solitary leisure activities. Consistently across studies, participation in home-based solitary leisure is the area that children with ASD participate in most similarly to their unaffected peers. In addition to swimming, it is an area in which they participate in and enjoy the most. In studies comparing sedentary behaviors between children with ASD and typically developing peers, the ASD groups spend significantly more time in computer use, television, and screen time (Healy et al. 2017; Must et al. 2014; Ratcliff et al. 2018). The typically developing children spend more time doing crafts and playing board games than did the children with ASD. Another study found that children with ASD engaged in significantly more time than typically developing children watching TV and playing video games to a level that became problematic, such as wanting to play video games more than interacting with family and friends and spending a lot of time thinking about playing video games (Mazurek and Wenstrup 2013). Additionally, these children spent less time in socially interactive video games or social media than the typical children, demonstrating that they were less involved and were uninterested in the social outlets associated with social media and video games.

High level of television watching negatively impacts physical fitness levels in childhood and is predictive of poor fitness in adulthood, with adult obesity 1.25 times more likely in adulthood and poor fitness 1.4 times more likely with each mean hour of television viewing during the week as a child (Landhuis et al. 2012).

Barriers to Leisure Participation

Studies report numerous barriers to participation in physical activity for children with ASD. Children with ASD participate in fewer formal, organized physical activities because of social and motoric difficulties (Memari et al. 2015; Stanish et al. 2015). Among the barriers cited were the children's preference to engage in technology-based activity, their difficulty with motor skills, their lack of social support, and their physical or community environments that were not conducive to their participation (Obrunsnikova and Cavalier 2011). Barriers identified by a qualitative study that interviewed youth with ASD, parents of youth with ASD, and service providers were limited understanding of social situations, maladaptive behaviors, and lack of social skills training programs for older children (Ghanouni et al. 2019). In a study examining the parental perceptions of barriers to physical activity, sensory sensitivities, gross motor deficits, behavioral outbursts, and difficulties in social situations, which impacted participation in team sports and group activities, were identified (Nichols et al. 2019). Must et al. (2015) also examined parent perceptions of barriers and reported that many children with ASD required too much supervision compared to typical peers, that supervising adults lack skills needed to include the children with ASD, and that their children have few friends, so are not invited to participate in many activities.

Future Directions

Potential solutions to increase leisure participation by children with ASD come from several directions. Parents are vital in keeping children

and adolescents with ASD physically active. Findings indicate that creating a physical activity routine and making it a habit improve regular participation in physical activity (Nichols et al. 2019). Supports, training, and respite programs for parents are effective interventions to enable their ability to promote successful behaviors of children with ASD. Social skills training has also shown effectiveness for improving social participation for individuals with ASD (Garcia-Villamizar and Dattilo 2010).

Health and public policies to adjust community environments to make them more inclusive and supportive are needed to increase participation in recreation and leisure by children with ASD. Research findings have shown that when physical activity programs are available, individuals with ASD are more physically active (Nichols et al. 2019). Funding is also needed to support programs that address participation in recreation and leisure for individuals with ASD. Community programs that teach social skills and encourage active engagement in areas of interest for children with ASD are needed to encourage participation in recreation and leisure. Programs that educate the public on how to best deal with those on the spectrum can be helpful in building supportive community environments. This knowledge is also important for public servants, such as police officers, who need to know that atypical responses should not always be interpreted as threatening or dangerous.

References and Reading

- Batsika, V., & Sharpley, C. F. (2015). Differences in the prevalence, severity, and symptom profiles of depression in boys and adolescents with an autism spectrum disorder versus normally developing controls. *International Journal of Disability, Development and Education*, 62(2), 158–167. <https://doi.org/10.1080/1034912X.2014.998179>.
- Billstedt, E., Gillberg, I. C., & Gillberg, C. (2011). Aspects of quality of life in adults diagnosed with autism in childhood. *Autism*, 15(1), 7–20. <https://doi.org/10.1177/1362361309346066>.
- Bohnert, A., Lieb, R., & Arola, N. (2016). More than leisure: Organized activity participation and socio-emotional adjustment among adolescents with autism spectrum disorder. *Journal of Autism and*

- Developmental Disorders*. <https://doi.org/10.1007/s10803-016-2783-8>.
- Eversole, M., Collins, D. M., Karmarkar, A., Colton, L., Quinn, J. P., Karsbaek, R., ... Hilton, C. L. (2016). Leisure activity enjoyment of children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46, 10–20. <https://doi.org/10.1007/s10803-015-2529-z>.
- Garcia-Villamisar, D. A., & Dattilo, J. (2010). Effects of a leisure programme on quality of life and stress of individuals with ASD. *Mental Health and Intellectual Disability*, 54(7), 611–619. <https://doi.org/10.1111/j.1365-2788.2010.01289.x>.
- Ghanouni, P., Jarus, T., Zwicker, J. G., Lucyshyn, J., Chauhan, S., & Moir, C. (2019). Perceived barriers and existing challenges in participation of children with autism spectrum disorders: “He did not understand and no one else seemed to understand him”. *Journal of Autism and Developmental Disorders*, 49, 3136–3145. <https://doi.org/10.1007/s10803-019-04036-7>.
- Healy, S., Haegele, J. A., Grenier, M., & Garcia, J. M. (2017). Physical activity, screen-time behavior, and obesity among 13-year olds in Ireland with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(49), 57. <https://doi.org/10.1007/s10803-016-2920-4>.
- Heffernan, K. S., Columna, L., Russo, N., Meyers, B. A., Ashby, C. E., Norris, M. L., & Barreira, T. V. (2018). Brief report: Physical activity, body mass index and arterial stiffness in children with autism spectrum disorder: Preliminary findings. *Journal of Autism and Developmental Disorders*, 48, 625–631. <https://doi.org/10.1007/s10803-017-3358-z>.
- Hilton, C. L., Crouch, M. C., & Israel, H. (2008). Out-of-school participation patterns in children with high-functioning autism spectrum disorders. *American Journal of Occupational Therapy*, 62, 554–563.
- Hilton, C. L., Cumpata, K., Klohr, C., Gaetke, S., Artner, A., et al. (2014). Effects of exergaming on executive function and motor skills in children with autism spectrum disorder: A pilot study. *American Journal of Occupational Therapy*, 68, 57–65.
- Hilton, C., Ratcliff, K., Collins, D., Flanagan, J., & Hong, I. (2019). Flourishing in children with autism spectrum disorders. *Autism Research*, 12(6), 952–966. (IF 4.532).
- Iwasaki, Y., Messina, E. S., & Hopper, T. (2018). The role of leisure in meaning-making and engagement with life. *The Journal of Positive Psychology*, 13(1), 29–35. <https://doi.org/10.1080/17439760.2017.1374443>.
- Jackson, S. L., & Dritschel, B. (2016). Modeling the impact of social problem-solving deficits on depressive vulnerability in the broader autism phenotype. *Research in Autism Spectrum Disorders*, 21, 128–138. <https://doi.org/10.1016/j.rasd.2015.10.002>.
- Jantz, K. M. (2011). Support groups for adults with Asperger syndrome. *Focus on Autism and Other Developmental Disabilities*, 26(2), 119–128. <https://doi.org/10.1177/1088357611406903>.
- Kaljača, S., Dučić, B., & Cvijetić, M. (2019). Participation of children and youth with neurodevelopmental disorders in after-school activities. *Disability and Rehabilitation*, 41(17), 2036–2048. <https://doi.org/10.1080/09638288.2018.1457092>.
- Landhuis, C. E., Poulton, R., Welch, D., & Hancox, R. J. (2012). Programming obesity and poor fitness: The long-term impact of childhood television. *Obesity*, 16(6), 1457–1459. <https://doi.org/10.1038/oby.2008.205>.
- Lawson, L. M., & Foster, L. (2016). Sensory patterns, obesity, and physical activity participation of children with autism spectrum disorder. *American Journal of Occupational Therapy*, 70(5), 7005180070p1–7005180070p8. <https://doi.org/10.5014/ajot.2016.021535>.
- Lin, L. (2014). Quality of life of Taiwanese adults with autism spectrum disorder. *PLoS One*, 9(10), e109567. <https://doi.org/10.1371/journal.pone.0109566>.
- Little, L. M., Sideris, J., Ausderau, K., Baranek, G. T. (2014). Activity participation among children with autism spectrum disorder. *American Journal of Occupational Therapy*, 68(2), 177–185. <https://doi.org/10.5014/ajot.2014.009894>.
- Mäkelä, S., Aaltonen, S., Korhonen, T., & Kaprio, J. (2016). Diversity of leisure-time sport activities in adolescence as a predictor of leisure-time physical activity in adulthood. *Scandinavian Journal of Medicine & Science in Sports*, 27(12), 1902–1912. <https://doi.org/10.1111/sms.12837>.
- Mazurek, M. O., & Wenstrup, C. (2013). Television, video game and social media use among children with ASD and typically developing siblings. *Journal of Autism and Developmental Disorders*, 43, 1258–1271. <https://doi.org/10.1007/s10803-012-1659-9>.
- McCoy, S. M., Jakicic, J. M., & Gibbs, B. B. (2016). Comparison of obesity, physical activity, and sedentary behaviors between adolescents with autism spectrum disorders and without. *Journal of Autism and Developmental Disorders*, 46, 2317–2326.
- Memari, A. H., Panahi, N., Ranjbar, E., Moshayedi, P., Shafiei, M., Kordi, R., & Zaiee, V. (2015). Children with autism spectrum disorder and patterns of participation in daily physical and play activities. *Neurology Research International*. <https://doi.org/10.1155/2015/531906>.
- Must, A., Phillips, S. M., Curtin, C., Anderson, S. E., Maslin, M., Lividini, K., & Bandini, L. G. (2014). Comparison of sedentary behaviors between children with autism spectrum disorder and typically developing children. *Autism*, 18(4), 376–384. <https://doi.org/10.1177/1362361313479039>.
- Must, A., Phillips, S. M., Curtin, C., & Bandini, L. G. (2015). Barriers to physical activity in children with autism spectrum disorders: Relationship to physical activity and screen time. *Journal of Physical Activity*

- & Health, 12(4), 529–534. <https://doi.org/10.1123/jpah.2013-0271>.
- Myers, E., Davis, B. E., Stobbe, G., & Bjornson, K. (2015). Community and social participation among individuals with autism spectrum disorder transitioning to adulthood. *Journal of Autism and Developmental Disorders*, 45, 2373. <https://doi.org/10.1007/s10803-015-2403-z>.
- Nichols, C., Block, M. E., Bishop, J. C., & McIntire, B. (2019). Physical activity in young adults with autism spectrum disorder: Parental perceptions of barriers and facilitators. *Autism*, 23(6), 1398–1407. <https://doi.org/10.1177/1362361318810221>.
- Obrunsnikova, I., & Cavalier, A. R. (2011). Perceived barriers and facilitators of participation in after-school physical activity by children with autism spectrum disorders. *Journal of Developmental and Physical Disabilities*, 23, 195–211. <https://doi.org/10.1007/s10882-010-9215-z>.
- Orsmond, G. I., Krauss, M. W., & Seltzer, M. M. (2004). Peer relationships and social and recreational activities among adolescents and adults with autism. *Journal of Autism and Developmental Disorders*, 34(3).
- Orsmond, G. I., Shattuck, P. T., Cooper, B. P., Sterzing, P. R., & Anderson, K. A. (2013). Social participation among young adults with an autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43(11), 2710–2719.
- Perkins, D. F., Jacobs, J. E., Barber, B. L., & Eccles, J. S. (2004). Childhood and adolescent sports participation as predictors of participation in sports and physical fitness activities during young adulthood. *Youth Society*, 34, 495–520. <https://doi.org/10.1177/0044118X03261619>.
- Potvin, M. C., Snider, L., Prelock, P., Kehayia, E., & Wood-Dauphinee, S. (2013). Recreation participation of children with high functioning autism. *Journal of Autism and Developmental Disorders*, 43, 445–457. <https://doi.org/10.1007/s10803-012-1589-6>.
- Ratcliff, K., Hong, I., & Hilton, C. L. (2018). Leisure participation patterns for school age youth with autism spectrum disorders: Findings from the 2016 national survey of children's health. *Journal of Autism and Developmental Disorders*, 48, 3783–3793. <https://doi.org/10.1007/s10803-018-3643-5>. (5-year IF 4.099).
- Ratcliffe, B., Wong, M., Dossetor, D., & Hayes, S. (2015). The association between social skills and mental health in school-aged children with autism spectrum disorder, with and without intellectual disability. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-015-2411-z>.
- Schall, C. M., & McDonough, J. T. (2010). Autism spectrum disorders in adolescence and early adulthood: Characteristics and issues. *Journal of Vocational Rehabilitation*, 32, 81–88. <https://doi.org/10.3233/JVR-2010-0503>.
- Shattuck, P. T., Orsmond, G. I., Wagner, M., & Cooper, B. P. (2011). Participation in social activities among adolescents with an autism spectrum disorder. *PLoS ONE*, 6(11). <https://doi.org/10.1371/journal.pone.0027176>.
- Solish, A., Perry, A., & Minnes, P. (2010). Participation of children with and without disabilities in social, recreational, and leisure activities. *Journal of Applied Research in Intellectual Disabilities*, 23, 226–236. <https://doi.org/10.1111/j.1468-3148.2009.00525.x>.
- Stanish, H. I., Curtin, C., Must, A., Phillips, S., Maslin, M., & Bandini, L. G. (2015). Enjoyment, barriers, and beliefs about physical activity in adolescents with and without autism spectrum disorder. *Adapted Physical Activity Quarterly*, 32(4), 302–17. <https://doi.org/10.1123/APAQ.2015-0038>.
- Stanish, H. I., Curtin, C., Must, A., Phillips, S., Maslin, M., & Bandini, L. G. (2017). Physical activity levels, frequency, and type among adolescents with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(3), 785–794. <https://doi.org/10.1007/s10803-016-3001-4>.
- Taheri, A., Perry, A., & Minnes, P. (2016). Examining the social participation of children and adolescents with intellectual disabilities and autism spectrum disorder in relation to peers. *Journal of Intellectual Disability Research*, 60(5), 435–443. <https://doi.org/10.1111/jir.12289>.
- Taylor, J. L., Adams, R. E., & Bishop, S. L. (2016). Social participation and its relation to internalizing symptoms among youth with autism spectrum disorder as they transition from high school. *Autism Research*, 10(4), 663–672. <https://doi.org/10.1002/aur.1709>.
- US Department of Health and Human Services (HHS), Office of Disease Prevention and Health Promotion. (2008). *Physical activity guidelines for Americans*. Washington, DC: HHS.
- Zeigler, E. F. (1965). A philosophical analysis of recreation and leisure. *Quest*, 5(1), 8–17. (00336297).

Leiter International Performance Scale-Revised (Leiter-R)

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Synonyms

Leiter-R

Description

The Leiter International Performance Scale-Revised (Leiter-R; Roid and Miller 1997) is a nonverbal measure of intellectual functioning normed for individuals between the ages of 2 years 0 months and 20 years 11 months. The tasks on the Leiter-R, which generally require a matching or pointing response, are meant to be self-explanatory and require minimal pantomimed instructions. Leiter-R materials consist of stimulus easels, cards, and foam shapes. The test takes about 90 min to administer.

The Leiter-R includes both visualization and reasoning (VR) and attention and memory (AM) batteries, comprised of 10 subtests each, though not all subtests are administered to all age groups. The VR and AM batteries may be administered separately or together, depending on the clinical need.

The VR battery is used to obtain IQ estimates and assesses traditional constructs of intelligence including nonverbal reasoning, visualization, and problem solving. The composite scores of the VR battery include a brief IQ screener (ages 2–20 years), full-scale IQ (2–20 years), fluid reasoning (2–20 years), fundamental visualization (2–5 years), and spatial visualization (11–20 years). The VR battery also offers an optional criterion-referenced metric called the Growth Score. The Growth Score is not age standardized or norm referenced and may be used to track change over several years (Roid et al. 2009). VR subtests and their primary tasks are listed in Table 1.

The AM battery measures nonverbal attention and memory function and is often used to assess the effects of attention deficit/hyperactivity disorder (ADHD) or learning disorders. The composite scores derived from the AM battery are memory screener (ages 2–20 years), associative memory (6–20 years), memory span (6–20 years), attention (6–20 years), memory process (6–20 years), and recognition memory (4–10 years). AM subtests and their primary tasks are listed in Table 2.

The Leiter-R also includes four optional social-emotional scales, (1) examiner, (2) parent, (3) self, and (4) teacher. Items are scored on a scale of 0 (rarely/never) to 3 (usually/always). These

Leiter International Performance Scale-Revised (Leiter-R), Table 1 VR battery subtest descriptions

Subtest	Summary of nature of task
Figure ground	Location of a stimulus, presented on a card, embedded within a more complex picture
Design analogies	Completion of abstract analogies presented in matrix form; the more difficult items require mental rotation of objects
Form completion	Mental assembly of a fragmented picture to form a whole
Matching	Matching a visual stimulus with an identical design
Sequential order	Selecting the correct picture or figure to complete a logical progression of stimuli; selection of related stimuli which progress in a corresponding order
Repeated patterns	Identifying the correct stimulus to complete a pictorial or figural pattern
Picture context	Identifying an object that has been removed from a complex display by using contextual clues
Classification	Categorizing stimuli based on salient characteristics such as color or shape
Paper folding	Mentally “folding” an object displayed in two dimensions and selecting the finished product from a lineup
Figure rotation	Identifying a two- or three-dimensional object that has been rotated

scales, which tap behaviors like inattention, hyperactivity, sociability, and mood regulation, are meant to serve as adjunct to the cognitive assessment and to aid in the interpretation of the testing results.

Historical Background

While a graduate student in California, Russell Leiter developed a scale of intelligence that was meant to be free of cultural bias, and this scale was later refined with Hawaiian children. Dr. Leiter was awarded a National Research Council grant to support the standardization of the Leiter Scale, which was published for the first time in 1934. The Leiter has gone through several revisions by Dr. Leiter and others. Although the content,

Leiter International Performance Scale-Revised (Leiter-R), Table 2 AM battery subtest descriptions

Subtest	Summary of nature of the task
Associated pairs	Observing pairs of pictured objects for 5–10 s, then remembering meaningful and non-meaningful associations
Immediate recognition	Discriminating between present and absent objects following the brief presentation and removal of a stimulus array
Forward memory	Recalling the sequence of objects pointed to by the examiner
Attention sustained	Completing clerical tasks such as finding and crossing out all squares within an array of geometric shapes; the tasks are more complex for more advanced individuals
Reverse memory	Recalling the sequence of objects pointed to by the examiner in reverse order
Visual coding	Identifying, from a key, the appropriate stimulus to complete a pair
Spatial memory	Recalling the location of a matrix array of stimulus cards after a brief presentation
Delayed pairs	Recalling the pairs presented in the first subtest, associated pairs, after approximately 30 min
Delayed recognition	Recognizing the objects present in the immediate recognition subtest after approximately 30 min
Attention divided	Attending to a task that requires identification of a target while also learning a card-sorting task

procedures, or norms of the test had not changed in any substantial way since the 1948 version, materials were copyrighted in 1979. These versions of the Leiter (1979) and the Leiter-R (Roid and Miller 1997) are the most recent. Although it was valuable for use in children with communication disabilities, the Leiter (1979) had several shortcomings. The psychometric characteristics of the test were weak, owing to inadequate standardization and norms for children aged 5–17 years last updated in the 1940s (Shah and Holmes 1985). The Leiter also utilized a ratio IQ, which is no longer considered an adequate representation of intellectual functioning. The constructs measured by the Leiter vary widely and are represented by few items. In contrast, the

Leiter-R was developed using several prominent theories of intelligence, and has updated norms and a more sophisticated psychometric profile. One study has shown the full-scale IQ scores on the Leiter and Leiter-R to be highly correlated in children with autism spectrum disorders (ASD) (Tsatsanis et al. 2003). However, because the content of the tests is different, the tests may not be considered as clinically interchangeable.

Psychometric Data

The Leiter-R VR battery standardization sample consisted of 1,719 typically developing individuals aged 2 years 0 months to 20 years 11 months. The sample was stratified by race, ethnicity, and socioeconomic status according to the 1993 US Census. A subset of 763 children were also administered the AM battery. Eleven special criterion groups were examined with the Leiter-R, including intellectual disability ($n = 123$), ADHD ($n = 112$), and severe speech/language impairment ($n = 98$), but not ASD. These criterion groups were not included in the standardization sample.

In the standardization sample, test reliability was established with internal consistency, test-retest, and inter-rater reliability. Average internal consistency for the subtests ranged from 0.75 to 0.90 for the VR battery and from 0.67 to 0.87 for the AM battery. Composite reliabilities ranged from 0.88 to 0.93 for the VR battery and from 0.75 to 0.93 for the AM battery. Of note was the higher reliability of the full-scale IQ (range 0.91–0.93 for the three age groups) relative to the brief IQ screener (range 0.88–0.90). The standard error of measurement was 4.24 for the full-scale IQ on the VR battery and ranged from 4.74 to 6.71 for AM composites. AM subscales were generally less stable across retesting than the VR subscales.

Validity data, including comparison to the original Leiter and other common IQ tests, supported the proposed use and interpretations of the Leiter-R for typically developing populations. The Leiter brief and full-scale IQ scores correlated highly ($r > 0.85$) with scores from the Weschler



Intelligence Scale for Children-Third Edition. Classification accuracy for cognitive delay ($n = 120$) was good, sensitivity (correctly identified as cognitive delay) was 84.2, and the specificity (correctly identified as typical) was 97.3.

The Leiter-R has been examined independently in two samples with ASD. Tsatsanis et al. (2003) found that the Leiter-R had good concurrent validity with the original Leiter in a sample of children with autism aged 4–16 years. Kuschner et al. (2007) examined the cognitive profiles of 16 children with ASDs aged 3.5–5.5 years. A distinct pattern emerged of relative strengths on figure ground and form completion with relative weaknesses on repeated patterns and sequential order. Differences on figure ground, form completion, and repeated patterns were specific to the ASD group, as they were not found in the typically developing or developmental disability comparison groups.

Although the psychometric properties of the Leiter-R have not been well studied in children with ASDs, there is agreement that the test is appropriate for the population (Tsatsanis et al. 2003).

Clinical Uses

The Leiter-R was designed for a wide variety of user groups; although most commonly used by psychologists, it is appropriate for use by speech and language pathologists, educational diagnosticians, and special educators, among others. The AM battery may be especially relevant to professionals concerned with ADHD symptoms or learning disorders. As with other tests of intelligence, interpretation requires advanced training.

Some considerations are warranted when the test is used for people with ASDs (Tsatsanis et al. 2003). Given the limited eye contact and joint attention skills of populations with autism, brief verbal cues (e.g., “touch” or “point”) and hand-over-hand guidance during teaching trials may be required. The Leiter-R allows for three teaching trials, which may be inadequate for some children with ASDs.

The Leiter-R has several features that may be advantageous for individuals with ASDs. The

nonverbal nature of the test minimizes the need for producing or understanding speech. Procedures for each of the subtests are very similar, minimizing the need for set shifts. Finally, nearly all of the tasks are untimed.

Due to these reasons, the Leiter-R has become especially popular in the assessment of children with developmental disabilities and ASDs in both clinical and research settings (Hooper et al. 2000; Tsatsanis et al. 2003). Given its popularity, it is important to note that the Leiter-R should not be seen as a direct measure of general intellectual ability, since it primarily measures nonverbal skills (Klin et al. 2005). Rather, it is to be used as an *estimation* of general intelligence (Campbell et al. 2008). Despite few psychometric data in this population, the Leiter-R is now widely considered to be an acceptable instrument for the cognitive assessment of children with ASDs to whom traditional tests of intelligence may not be applicable.

See Also

- [Intelligence Quotient](#)
- [Nonverbal Intelligence](#)
- [Psychological Assessment](#)

References and Reading

- Campbell, J., Brown, R., Cavanagh, S., Vess, S., & Segall, M. (2008). Evidence-based assessment of cognitive functioning in pediatric psychology. *Journal of Pediatric Psychology*, 33(9), 999–1014.
- Hooper, S. R., Hatton, D. D., Baranek, G. T., Roberts, J. P., & Bailey, D. B., Jr. (2000). Nonverbal assessment of IQ, attention, and memory abilities in children with Fragile-X syndrome using the Leiter-R. *Journal of Psychoeducational Assessment*, 18, 255–267.
- Klin, A., Saulnier, C., Tsatsanis, K., & Volkmar, F. (2005). Clinical evaluation in autism spectrum disorders: Psychological assessment within a transdisciplinary framework. In F. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders*. Hoboken: Wiley.
- Kuschner, E., Bennetto, L., & Yost, K. (2007). Patterns of nonverbal cognitive functioning in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 795–807.
- Leiter, R. (1979). *Instruction manual for the Leiter international performance scale*. Wood Dale: Stoelting.

- Roid, G., & Miller, L. (1997). Leiter international performance scale-revised: Examiner's manual. In G. H. Roid & L. J. Miller (Eds.), *Leiter international performance scale-revised*. Wood Dale: Stoelting.
- Roid, G., Pomplun, M., & Martin, J. (2009). Nonverbal intellectual and cognitive assessment with the Leiter international performance scale-revised (Leiter-R). In J. A. Naglieri & S. Goldstein (Eds.), *Practitioner's guide to assessing intelligence and achievement*. Hoboken: Wiley.
- Sattler, J. M. (2001). *Assessment of children: Cognitive applications* (4th ed.). La Mesa: Jerome M. Sattler.
- Shah, A., & Holmes, N. (1985). Brief report: The use of the Leiter international performance scale with autistic children. *Journal of Autism and Developmental Disorders*, 15, 195–203.
- Tsatsanis, K., Dartnall, N., Cicchetti, D., Sparrow, S., Klin, A., & Volkmar, F. (2003). Concurrent validity and classification accuracy of the Leiter and Leiter-R in low-functioning children with autism. *Journal of Autism and Developmental Disorders*, 33(1), 23–30.

Leiter-R

- [Leiter International Performance Scale-Revised \(Leiter-R\)](#)

L-Epinephrine

- [Epinephrine](#)

L-Epinephrine

- [Epinephrine](#)

Leponex

- [Clozapine](#)

Letter Fluency

- [Verbal Fluency](#)

Letter of Intent

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Definition

A letter of intent (LOI) is a document with personalized information about how to provide care and support for a specific individual with disabilities (hereinafter the “beneficiary” of the LOI) (Garner 2019). A primary caregiver or caregiver group can author an LOI to document the essential knowledge that others should have when assisting a beneficiary in the future. Well-designed LOIs provide meaningful insight into all preferences, needs, and plans concerning a beneficiary’s prospective care. Information summaries within an LOI typically focus on three key subject areas: daily living, healthcare and personal wellness, and finance and property management.

Principles of a Letter of Intent

An LOI can be a useful special needs planning tool for individuals with autism spectrum disorder (ASD) and their close caregivers, often a parent, guardian, family member, or friend. In LOI matters, an individual with disabilities who receives care is also commonly referred to as a “beneficiary” of an LOI. Importantly, LOIs are not legally binding documents and should not be confused with formal contracts agreements or wills. A caregiver’s desires and plans for an individual with disabilities can be judicially enforced through official instruments from contract, trust, estate, and tax law. The design of an LOI should complement these binding instruments.

An LOI serves as an informational addendum to binding legal instruments (Andersen and Gary 2018). LOIs often are used in the special needs planning process as unofficial guidance for the following items: power of attorney (healthcare

and finance/property), guardianship appointments, conservatorship appointments, special needs trusts, ABLE accounts, deeds, and wills (Lauderdale and Huston 2012). Generally, the purpose of an LOI is to collect knowledge about a beneficiary from longtime caregivers who are well-acquainted with and well-informed about the individuals' personal, medical, and financial needs.

The contents of an LOI should cover the beneficiary's unique interests, dislikes, needs, strengths, and challenges. A caregiver's advice and directions in an LOI can help others deliver the same quality care and assistance in the future. The goals of an LOI are twofold: (1) to communicate loved ones' intentions about the types of care that a beneficiary should receive in the future and (2) to record specific details about how others should approach certain care tasks (Tencza and Forsythe 2019). From a design perspective, the details of an LOI should be organized into neat categories with clear headings so that the content can be easily understood and reviewed by other people. An LOI is particularly useful to share a caregiver's wishes and acumen when the caregiver is no longer able to provide assistance (Walker and Hutchinson 2019; see also Krakovich et al. 2016).

Letter of Intent Content

LOIs can be designed as collections of helpful suggestions and recommended best practices to care for an individual with disabilities. LOIs should contain practical insights regarding the individual's skills, challenges, and day-to-day activities. They can also include recommendations on how to approach any known or anticipated concerns. If a legal dispute arises regarding ambiguous wording or conflicting phrases within any legal contract, a court may analyze an LOI as evidence of what the parties intended when entering into the agreement. In these instances, an LOI may help a judge to decide whether the law prescribes a remedy for the lawsuit according to principles of fairness and equity.

An LOI should cover vital details about what works well for the individual in all of the major areas of his or her life. These topics include, but are not limited to, the beneficiary's close friends and family, education, employment, socialization, religion, and medical care (Table 1). Each topic within the LOI should discuss the beneficiary's past history, current status, and future plans or goals (Burke et al. 2018). Moreover, each LOI topic should list the full names of all associated individuals and their contact information with current addresses, phone numbers, and email addresses. Other useful material may include answers to the following questions in the LOI's Family Members and Friends of the Beneficiary Section: (1) Which ones are emotionally closest to the person? and (2) Which ones are geographically closest to the person? (Lindahl et al. 2019).

In the General Information Section of an LOI, authors should organize facts about the LOI beneficiary's marital status, spouse, citizenship status, languages spoken, date of birth, and place of birth. The Personal Preferences Section of an LOI can be especially useful for future caregivers and should encompass the following details: social activities that the beneficiary enjoys, hobbies, favorite foods, beloved locations, and preferred music types/artists (Leung et al. 2016). If the beneficiary has religious or spiritual preferences, it can be helpful to explain whether he or she adheres to a particular religion or attends a local place of worship.

An LOI should also encapsulate the beneficiary's financial, tax, and legal plans. The Special Needs Plan Section should list all associated names and contact information (e.g., preferred attorney, law firm, financial planner, and tax advisor). Ideally, an LOI will contain a summary of the short- and long-term goals for gathering and maintaining funds to support the beneficiary. It should address questions like the following: (1) Does the beneficiary have a legal guardian, conservator, power of attorney, trustee, or estate executor? (2) If he or she should require such assistance in the future, do you know of any

Letter of Intent, Table 1 Key information to include in a letter of intent for future caregivers

Item number	Information category
1	General information about the letter of intent beneficiary
2	General information about the letter of intent author(s)
3	Family members and friends of the letter of intent beneficiary
4	Past and present caregivers
5	Summary of special needs plan (short- and long-term goals for gathering and maintaining funds to support the letter of intent beneficiary)
6	Attorney, financial advisor, and tax advisor for estate and special needs planning
7	Guardianship or conservatorship appointments
8	Trusts and trustees
9	Power of attorney
10	ABLE savings account (also known as a 529A account) and all associated online account password and log-in details
11	Estate executor
12	Advocates
13	Letter of intent beneficiary's clothing size numbers
14	Letter of intent beneficiary's education history
15	Letter of intent beneficiary's employment and/or volunteer work history
16	Past and current residences and living situation preferences
17	Personal preferences, likes, and dislikes
18	Religious and/or spiritual preferences
19	Medical care history
20	Individual functional abilities (strengths and weaknesses)
21	Behavior management therapies and regimens
22	Assistive technology (mobility devices and commonly used items)
23	Orthotics and prosthetics
24	Insurance (primary and supplemental) and/or government benefits (including Medicaid, Medicare, Social Security Disability Insurance (SSDI), and/or Social Security Retirement Insurance (SSRI))
25	End-of-life arrangements and wishes for final remains

individuals who might excel in these in roles? and (3) Does the beneficiary have an ABLE account or any other financial holdings? All in all, the LOI should serve as a roadmap that outlines the contents of much longer official documents (see, e.g., Hershey et al. 2015, discussing how to use a letter of intent to guide a special needs planning trustee).

In the Medical Care History Section of the LOI, authors should include any other information that could help future caregivers to provide a beneficiary with the best possible care. Many LOIs contain a directory of a beneficiary's physicians and other healthcare professionals. They also usually record a summary of the beneficiary's personal health history (conditions, treatments, surgeries, accidents, and hospitalizations). An LOI should state any insurance deductibles and out-of-pocket expenses for healthcare services and goods. An LOI can recommend specific therapeutic programs and educators who have proven especially successful in working with the beneficiary in the past. It is common for an LOI to confirm which particular assistive technology products and resources are utilized by the beneficiary (U.S. National Institute on Disability Independent Living, and Rehabilitation Research 2020). Additionally, LOI authors should consider reporting their knowledge about "individual functional abilities," which can be sorted into three primary categories of strengths and weaknesses: conceptual, social, and practical (Leung et al. 2016; see also Mahdi et al. 2018) (Table 2).

If an LOI document is particularly lengthy, it can be helpful to add subject headings to organize the information in the same manner as book chapter titles. As a best practice, an LOI's author should place the LOI with all other relevant legal and personal documents concerning the beneficiary. These include all medical records, legal instruments, and financial records (Arbaje 2020) (Table 3). LOI creators can also consider organizing an LOI and its attachments using page numbers or bates numbers with a table of contents.

Letter of Intent, Table 2 Caregiver insights on individual functional ability to document in a letter of intent

Individual’s conceptual strengths and weaknesses	Individual’s social strengths and weaknesses	Individual’s practical strengths and weaknesses
• Reasoning/problem-solving Processing speed • Short- and long-term memory • Expressive language • Receptive language (ability to understand communications) • Literacy • Education • Overall knowledge • Learning	• Communication skills • Social judgment • Ability to understand and follow social queues and nuances • Ability to understand and follow rules • Ability to make and keep friendships	• Activities of daily living for personal care • Home care and financial management tasks • Responsibilities • Organizing tasks • Instances where third-party assistance is usually needed • Instances where assistive technology or other goods are helpful

Updating and Distributing a Letter of Intent

To confirm when a document was last updated, most LOI authors sign and date their LOIs. Special needs planning experts strongly recommend that caregivers review the summaries and lists within an LOI annually – the document must be regularly revised and updated to reflect changes in the beneficiary’s life. Russell and Grant (2010) offer the following guidance for parents who are writing an LOI for their children:

Your major concern [in writing an LOI] is to make sure that your child will have a happy and meaningful life. Write clearly enough so that anyone who reads the letter in the future will understand exactly what you meant. . . Each year you should take out the letter and review it to make sure it remains current. Occasionally there will be a significant change in your child’s life, such as a new residential placement or a bad reaction to medication, and the letter should be revised immediately if any such change occurs.

An LOI can be typed or handwritten. It is a best practice consider providing copies of an LOI document to the following individuals: appointed guardian or conservator, power of attorney, trustee, estate executor, and legal counsel for the beneficiary. Some caregivers choose to give LOI copies to close friends, family members, or others who may need to know its details (Heller and Kramer 2009). Each year, all stakeholders should receive a copy of the most recent version of the LOI and its attached documents.

Letter of Intent, Table 3 Official documents to attach with a letter of intent

1. Insurance forms related to healthcare services and goods
2. Healthcare power of attorney (also called medical power of attorney)
3. Power of attorney (finance and property)
4. Do-not-resuscitate (DNR) order
5. Trust documentation
6. Court order for guardianship or conservatorship
7. ABLE savings account (529A) forms and online password and account sign-in information
8. Beneficiary’s last will and testament
9. Documentation of healthcare (primary and supplemental) and personal insurance plans
10. Documentation of government benefit plans
11. Professional retainer agreements (e.g., attorney, financial advisor, and tax advisor for estate and special needs planning)
12. Healthcare records (sorted by date and provider and organized with numbered pages or bates-labeled pages and a table of contents)
13. Court filings concerning the letter of intent beneficiary (e.g., petition for estate administration)

Storage Practices for a Letter of Intent

An LOI must be both readily accessible to those who need its information and protected against prying eyes. LOIs and their attached documents contain a wealth of data that could prove harmful if stolen by third parties. The LOI serves as a record of intimate personal details, addresses, financial information, and sensitive health information. Consequently, LOI writers and recipients

must take care to protect the privacy and security of both electronic and printed LOIs.

Like so many other digital documents, any LOI stored online or on a computer is vulnerable to online information piracy crimes, including identity theft, financial fraud, forgery, stalking, bullying, and cyber-hacking (e.g., National Cybersecurity Alliance 2020; see also U.S. Department of Homeland Security Cybersecurity and Infrastructure Security Agency 2020). There are numerous for-profit software companies and insurance plans who offer protected online databases for storing sensitive documents. Users of these services are encouraged to find an online platform that allows them to share access to LOI documents with others in a secure format. Additionally, an LOI author must be sure to record the access information (website, user name, log-in password) and share it with a backup contact (e.g., Arbaje 2020).

Another option for storing and distributing an LOI is to save it on a portable hard drive and to physically distribute the device to others. These drives should have password protection and data encryption to guard against hacking. Recipients of hard drive-based LOIs must understand that they should not download a drive's contents to their personal computers for data security reasons. Because of cybersecurity and privacy concerns, LOI users should avoid sending an LOI document via a regular email attachment or with a link to a general document storage site.

See Also

- [Achieving a Better Life Experience Savings Account \(ABLE Savings Account, ABLE Account, 529A Savings Plan, and 529A Account\)](#)
- [Conservatorship \(Full Conservatorship and Limited Conservatorship\)](#)
- [Guardianship](#)
- [Power of Attorney \(Financial and Property Management\)](#)
- [Power of Attorney for Healthcare \(Durable Healthcare Power of Attorney and Medical Power of Attorney\)](#)

- [Special Needs Planning \(SNP\)](#)
- [Special Needs Trust \(SNT\)](#)

References and Reading

- Andersen, R., & Gary, S. (2018). *Understanding trusts and estates* (6th ed.). Durham: Carolina Academic Press.
- Arbaje, A. (2020). *Medical records: Getting organized*. The Johns Hopkins University. Retrieved from <https://www.hopkinsmedicine.org/health/wellness-and-prevention/medical-records-getting-organized>
- Burke, M., Arnold, C., & Owen, A. (2018). Identifying the correlates and barriers of future planning among parents of individuals with intellectual and developmental disabilities. *Intellectual and Developmental Disabilities*, 56(2), 90–100. <https://doi.org/10.1352/1934-9556-56.2.90>
- Garner, B. (Ed.). (2019). Letter of intent. *Black's law dictionary* (11th ed.). St. Paul: Thomson/West.
- Heller, T., & Kramer, J. (2009). Involvement of adult siblings of persons with developmental disabilities in future planning. *Intellectual and Developmental Disabilities*, 47(3), 208–219. <https://doi.org/10.1352/1934-9556-47.3.208>
- Hershey, L., Abbey, B., & Stanaland, T. (2015). When to stretch and when not to stretch an inherited IRA: The special case of the special needs trust. *Journal of Financial Service Professionals*. Retrieved from https://www.emich.edu/cob/documents/2015_when_to_stretch_and_when_not_to_stretch_an_inherited_ira.pdf
- Krakovich, T. M., McGrew, J. H., Yu, Y., & Ruble, L. A. (2016). Stress in parents of children with autism spectrum disorder: An exploration of demands and resources. *Journal of Autism and Developmental Disorders*, 46(6), 2042–2053. <https://doi.org/10.1007/s10803-016-2728-2>
- Lauderdale, M., & Huston, S. (2012). Financial therapy and planning for families with special needs children. *Journal of Financial Therapy*, 3(1), 62–81. <https://doi.org/10.4148/jft.v3i1.1494>
- Leung, R., Vogan, V., Powell, T., Anagnostou, E., & Taylor, M. (2016). The role of executive functions in social impairment in autism spectrum disorder. *Child Neuropsychology*, 22(3), 336–344. <https://doi.org/10.1080/09297049.2015.1005066>
- Lindahl, J., et al. (2019). Domains of planning for future long-term care of adults with intellectual and developmental disabilities: Parent and sibling perspectives. *Journal of Applied Research in Intellectual Disabilities*, 32(5), 1103–1115. <https://doi.org/10.1111/jar.12600>
- Mahdi, S., et al. (2018). An international clinical study of ability and disability in autism spectrum disorder using the WHO-ICF framework. *Journal of Autism and Developmental Disorders*, 48(6), 2148–2163. <https://doi.org/10.1007/s10803-018-3482-4>
- National Cybersecurity Alliance (2020). Theft, fraud & cybercrime. Stay Safe Online. <https://staysafeonline.org/stay-safe-online/identity-theft-fraud-cybercrime/identity-theft-fraud/>

- Russell, L. M., & Grant, A. (2010). *Planning for the future: Give your child with a disability the gift of a safe and happy life* (7th ed.). Palatine, Illinois: Planning For The Future.
- Tencza, M., & Forsythe, L. (2019). Transition-of-care planning: Preparing for the future care of the individual with intellectual and developmental disabilities. *Journal of Intellectual Disabilities*. <https://doi.org/10.1177/1744629519883453>.
- U.S. Department of Homeland Security Cybersecurity & Infrastructure Security Agency. (2020). Cybersecurity: Stop think connect. Retrieved from <https://www.cisa.gov/stophinkconnect>.
- U.S. National Institute on Disability Independent Living, and Rehabilitation Research. (2020). AbleData: Tools & technologies to enhance life: Search AbleData. <https://abledata.acl.gov/>
- Walker, R., & Hutchinson, C. (2019). Care-giving dynamics and futures planning among ageing parents of adult offspring with intellectual disability. *Ageing & Society*, 39(7), 1512–1527. <https://doi.org/10.1017/S0144686X18000144>.

Lettick, Amy L. (1921–2017)

Linda Rumanoff Simonson
Benhaven, Inc., North Haven, CT, USA

Name and Degrees

New Haven Teachers College (NHTC), B.A., elementary education teaching certification

Yale University, Doctor of Humane Letters honorary degree

University of New Haven, Doctor of Humane Letters honorary degree

Major Appointments (Institution, Location, and Dates)

- Executive Director, Benhaven, Inc., 1967–1987
- Appointment to Connecticut Governor's Committee on Autistic Children, 1973

Major Honors and Awards

- 1973 – Asked by Dr. Bernard Rimland of National Society for Autistic Children

(NSAC) to tour with him to speak in nine different cities

- 1975 – Received Doctor of Humane Letters honorary degree from Yale University
- 1989 – Received Doctor of Humane Letters honorary degree from the University of New Haven
- Journals and other writings in Special Collection at Yale University's Sterling Memorial Library

Landmark Clinical, Scientific, and Professional Contributions

Amy Ladin Lettick was not a scientist, a researcher, or a clinician. Amy Lettick was a mother, a teacher, and a pioneer in the field of autism.

Amy Lettick was trained as a teacher in college and went on to teach for several years. Her training, experience, and drive served her well after giving birth to a son, Ben, later diagnosed with autism, at a time when there were few educational options available. When Ben turned 7, he aged out of The Yale Child Study Center's nursery school where he had been for 2 years. Amy took matters into her own hands, did her own research, and read two books by Dr. Newell Kephart of Purdue University (Kephart 1960; Strauss and Kephart 1955) which set her on a new path. After reading those books and successfully putting into practice Kephart's teaching procedures and techniques with Ben, Amy's mission became to find the financial backing and supports needed to start her own school where she could share her skills and knowledge with a new generation of teachers, students, and families. She accomplished this in 1967 when the first Benhaven School opened in New Haven, Connecticut, serving four children. Within 3 years, that number grew, necessitating the first of several moves. Over the next 50 years, what started with Amy's desperation and determination grew into a well-recognized and respected agency providing educational, vocational, and consulting services to hundreds of individuals and families.

Part of Amy's legacy was a commitment to lifelong learning. She championed adult

education even though public and most private schools stop providing services at age 21. To this end, volunteers from local high schools are trained to conduct lessons in communication, functional math, art, and even foreign languages to those residents who could not learn in community adult education settings. Many of these volunteers go on to become employees at Benhaven or to pursue an education and eventual career in autism or a related field. Those who can benefit from and tolerate community-based educational opportunities are supported in those settings.

Amy Lettick's vision of a school evolved in response to the needs of the individuals enrolled. Several years after it first opened its doors, Benhaven offered a pre-vocational program and, a few years after that, an employment program that included some workshop-based activities but actively sought community-based employment opportunities. Under Amy Lettick, Benhaven also purchased several acres of rural property on which greenhouses were erected, chicken coops stocked with egg layers, and vegetable gardens planted to provide more work and training options for the individuals it served.

Amy Lettick recognized the need not only for educational and vocational training for individuals with autism but experienced firsthand the lack of appropriate residential facilities, especially for those with disruptive and explosive behaviors. As a parent, Amy was well aware of the attention and energy required to care for a child with autism and the toll it could take on the family. Amy also strongly believed that residential care should not be custodial but be a place for active learning in a setting rich with natural opportunities to learn new skills. To this end, Amy Lettick opened the first of several residential homes in 1972, which served individuals from not only Benhaven's home state of Connecticut but also from Massachusetts, Rhode Island, New York, New Jersey, Texas, Ohio, Maryland, North and South Dakota, and as far away as Mexico during the residential program's first 12 years.

Amy Lettick was eager to collaborate with clinicians and researchers from Yale Child Study Center. That collaboration and cooperation began with Dr. Donald Cohen, Director of the Yale Child

Study Center from 1983 until his untimely death in 2001. It continued with Dr. Fred Volkmar, who replaced Dr. Cohen as Director of Yale Child Study Center, and that connection continues today. Many Benhaven students and residents have participated in a variety of studies over the years, contributing to the scientific and clinical knowledge that this research has provided. Fellows from there spend a year of their training as consulting psychiatrists for Benhaven, getting invaluable experience in reviewing case studies with Benhaven staff, and prescribing and monitoring a range of psychiatric medications and their effects on mood and behavior. Even Yale undergraduates have opportunities to learn from and interact with Benhaven students, residents and staff as they spend time at one of two schools or at one of the residences as part of a class requirement. Internship programs with other local universities and colleges have also been developed which continue to extend Benhaven's contribution to shaping future teachers and clinicians, and giving the community a better understanding of those with autism.

Amy Lettick started with a vision and a mission and turned it into a multifaceted learning, working and living environment that has yielded far reaching benefits for the individuals Benhaven serves, their families, the staff who are trained to support them, clinicians and researchers, college and university students, as well as the communities at large.

Short Biography

Amy Lettick's work with her son, Ben, and the subsequent establishment of Benhaven, had a significant and lasting impact on the field of autism, especially, but not limited, to the areas of education and residential treatment. As Amy's vision became a reality, her mission grew to include connections with other parents through individual relationships and through the National Society of Autistic Children, with Yale University, and in particular, The Yale Child Study Center, and with other institutions and individuals in the community. One of her main messages in those early

days was that these children, who at that time could not be served in traditional classrooms, would learn if taught within a structure of consistency and in ways that maximized their strengths. Although Amy Lettick proved herself to be a force of nature in promoting the education of those with autism, in her early days, she did not see herself in a teaching career.

Amy Lettick was born on August 27, 1921, in New Haven, Connecticut, the youngest of three children. Her parents emigrated from Russia, her mother at 2 years old and her father at age 17. Her father eventually owned a haberdashery store, and her mother worked in a factory until the birth of her children. Money was tight but her parents managed to send all three of the children to college. When Amy graduated from high school as an outstanding student and graduation speaker, she had earned scholarships, but not enough to cover transportation and living expenses at schools like Vassar or Wellesley. She knew that she would have to go to school locally, and so accepted a scholarship to New Haven State Teachers College. In her memoir, *Ways and Means* (Lettick 1998), she states that she never deliberately chose to become a teacher, but that was the only career open to her as a graduate of NHSTC. She said that she had never regretted it, especially in view of her needs years later.

Ben was the third of Amy's four children, and his tantrums, crying, destructiveness, and unpredictability required constant vigilance. She recognized that her family suffered from the need for constant surveillance of Ben, dealing with his odd and often bizarre behaviors and the stress of taking Ben on an outing. This awareness contributed to her desire, years later, to provide residential care in home-like settings as an alternative to the then current institutional facilities available.

Amy and Ben had the benefit of living near the Yale Child Study Center, and Ben was seen by Dr. Sally Provence and Dr. Ellis Perlschwig, a young psychiatrist. Amy brought Ben to the Center twice a week for an hour with the psychiatrist, while she met with a psychiatric social worker. During these years, Ben was enrolled at the Yale Child Study Center's nursery school; but when he was six,

Amy was told that he would be ineligible to attend when he turned seven and that they did not know of an appropriate place for him. Amy's story is familiar to any families who had children with autism in the 1960s, and sadly, many still struggle to find appropriate and adequate resources, especially after age 21.

In 1961, Amy struck up a correspondence with Rosalind Oppenheim, the mother of a child with autism who had written an article in a magazine that Amy had read. In it, she told of being rescued by Dr. Newell Kephart of Purdue University. Amy read two books by Kephart (Kephart 1960; Strauss and Kephart 1955) that Rosalind recommended and almost immediately took Ben by the hand, walked upstairs to a table in his room, and began his first class time. From that significant day in 1961 when Ben was 7 until 1967 when he was 12, she worked with Ben daily (Lettick 1998).

Amy kept detailed records of all those classes with Ben (Silverman 2011) and kept up a correspondence with Rosalind. They shared progress, despair, books and activities, and ultimately hope, as each of their boys made slow but observable improvements.

Amy discussed Ben's progress with Dr. Provence, and they agreed that taking Ben to see Dr. Kephart for an evaluation might give Amy some new methods of teaching Ben. Soon after, she and Ben travelled to Indiana and met with Dr. Kephart and his staff, and Amy said that she felt she was in the right place and getting the help she needed. Ben became a client of Kephart's Achievement Center for Children from 1962 until 1969, and Amy incorporated much of what she learned there into her teaching and training of teachers when she opened Benhaven School in 1967.

Since 1962, Amy had worked tirelessly pressing the New Haven school system to create a class for Ben and others like him. She finally succeeded in getting a class funded in 1966, but it never became a reality because a qualified person to run it was never found. When Ben turned 11 and there was still no place for him, Amy became convinced that unless she started a school, Ben would not get an appropriate education.

After investigating available services and finding nothing appropriate in Connecticut, Amy looked elsewhere. She visited the League School in Brooklyn directed by Dr. Carl Fenichel and the Nassau Center in Long Island directed by Dr. Joan Shodell. Both directors urged her to start a school and promised their help. By the end of that year, Amy started searching for board members, financing, and a location.

Amy shared her thoughts about her school (which her family had already named “Benhaven”), with Drs. Kephart, Fenichel, and Shodell, and their responses to her ideas helped shape her thinking. And although she was warned that parent-run schools had a very poor prognosis, she remained stalwart in her mission and opened Benhaven School on September 7, 1967, with Ben and two other boys as the first students.

Over the next 5 years, Benhaven outgrew that first school, as enrollment increased to 24 and then 34 students. During that period, Amy also began the search for funding and grants to open a residential home. This proved an arduous challenge with many frustrations and disappointments along the way. But in December 1972, just before Christmas, three Benhaven students, including Ben, moved into the first home. By 1974, the school was housed in a large four-story building; the residential home and swimming pool were situated on a 34 acre farm with a farmhouse and barns; Benhaven offered outpatient services, a professional training service, and parent training; and Yale Child Study Center was conducting research with Benhaven students.

Amy wanted to share her expertise and experience and inadvertently became an author. She made the first national presentation about Benhaven at the National Society for Autistic Children Conference in the summer of 1971. She was asked by the NSAC board to repeat the presentation five times so that more attendees could hear it. Amy decided then to turn her talk into a monograph to sell. Later she added lectures and teaching techniques and called the book *Benhaven Then and Now* (Lettick 1975). She turned down a publishing contract and chose to publish it through her own enterprise which she called The Benhaven Press; she sold several thousand copies.

She subsequently authored and sold more publications and tapes.

With all that Amy accomplished, one of her happiest and proudest moments was in December 1974, when she learned that she would receive an honorary degree from Yale University. On May 19, 1975, she received the honorary degree of Doctor of Humane Letters, an official recognition of her efforts and achievements. That was followed in 1989 by a Doctor of Humane Letters honorary degree from the University of New Haven.

Amy’s “can do” attitude and tireless efforts kept Benhaven growing even through many challenges including dismal financial crises. But by the time she retired in 1987, Benhaven had opened five more homes. Amy’s successor as Executive Director, Larry Wood, had worked at Benhaven for 17 years, starting out as a teaching aide, and so Amy was very comfortable turning over the mantle to him. Larry had proven himself as a visionary leader with the skills and determination to keep Benhaven growing and improving its services. Now, in 2019, Benhaven has 2 schools serving 67 students, 9 homes including 7 adult homes serving 36 residents with an eighth adult home in the planning stage, a Career and Transition Services program, 5 shared living homes, Individual Family Supports serving more than 60 families and 47 children and their families receiving support from Benhaven’s Children’s Behavioral Services program, and a consulting group. Those at the helm today demonstrate Dr. Lettick’s drive to find the best ways to serve more individuals and families while continuing to raise expectations for the level of professional development of its staff and the services provided.

In October 2018, Benhaven celebrated 50 years since its founding by Dr. Lettick with a gala affair. Almost a month to that day on November 22, Dr. Amy Lettick died peacefully in her sleep in Tempe, Arizona. She was 96 years old and lived a full and accomplished life.

See Also

- [Benhaven Residential Services](#)
- [Education](#)

- [Family Burden](#)
- [Living Arrangements in Adulthood](#)
- [Parent-Professional Partnership](#)

References and Reading

- Kephart, N. C. (1960). *The slow learner in the classroom*. Columbus: Merrill Publishing.
- Lettick, A. (1975). *Benhaven then and now*. New Haven: The Benhaven Press.
- Lettick, A. L. (1998). *Ways and means*. Tempe: The Benhaven Press.
- Silverman, C. (2011). *Understanding autism: Parents, doctors, and the history of a disorder*. Princeton: Princeton University Press.
- Strauss, A. A., & Kephart, N. C. (1955). *Psychopathology and education of the brain injured child*. New York: Grune and Stratton.

Yale University Sterling Library Special Collections

- Amy Lettick's diaries, 1953–1957.*
- Lettick, Amy. *Records of a 6 year educational program designed for a severely autistic boy.*
- Lettick, Amy. *Amy Lettick's journal, 1961–1967*, Unabridged, 3 Volumes (“Ben’s journal”).
- Lettick, Amy. Videotape series: *Amy Lettick on Teaching*.

Levate

- [Elavil \(Amitriptyline\)](#)

Levetiracetam: Definition

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Synonyms

[Keppra](#)

Definition

Levetiracetam is an anticonvulsant; however, its mechanism of action remains unknown. While it has been suggested for use in many psychiatric disorders, including autism, evidence is conflicting and minimal. Side-effect profiles include headache, somnolence, dizziness, and asthenia. It is important to note that the mechanism of action is not similar to other anticonvulsants.

See Also

- [Anticonvulsants](#)

References and Reading

- Cereghino, J. J., Biton, V., Abou-Khalil, B., Dreifuss, F., Gauer, L. J., & Leppik, I. (2000). Levetiracetam for partial seizures results of a double-blind, randomized clinical trial. *Neurology*, 55(2), 236–242.
- Lynch, B. A., Lambeng, N., Nocka, K., Kensel-Hammes, P., Bajjalieh, S. M., Matagne, A., & Fuks, B. (2004). The synaptic vesicle protein SV2A is the binding site for the antiepileptic drug levetiracetam. *Proceedings of the National Academy of Sciences of the United States of America*, 101(26), 9861–9866.
- Patsalos, P. N. (2000). Pharmacokinetic profile of levetiracetam: Toward ideal characteristics. *Pharmacology and Therapeutics*, 85(2), 77–85.
- Rugino, T. A., & Samscock, T. C. (2002). Levetiracetam in autistic children: An open-label study. *Journal of Developmental and Behavioral Pediatrics*, 23(4), 225–230.
- Wasserman, S., Iyengar, R., Chaplin, W. F., Watner, D., Waldoks, S. E., Anagnostou, E., ... & Hollander, E. (2006). Levetiracetam versus placebo in childhood and adolescent autism: A double-blind placebo-controlled study. *International Clinical Psychopharmacology*, 21(6), 363–367.

Levo-Methylaminoethanolcatechol

- [Epinephrine](#)

Levoreninum

- [Epinephrine](#)

Lexam

► [Escitalopram](#)

Lexamil

► [Escitalopram](#)

Lexapro

► [Escitalopram](#)

Lexicon

► [Vocabulary](#)

Liability

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Definition

Liability

Liability is the type of accountability assumed by an individual or legal entity upon engaging in activity, which imposes on the individual or legal entity a specified standard of action and duty of care. The legal responsibility that is liability is enforceable by civil remedy and or criminal punishment (Garner 2004). The type of liability that attaches to an individual or entity depends on the nature of the action, as well as the nature of the relationship between the individual or to which entity the duty of care is owed to and the individual or entity being held liable.

Tort Liability

Tort liability is “the kind of legal responsibility that adheres to a person or legal entity as a result of an injury done to someone else or to some other entity” (Oller and Oller 2010). This type of liability applies in the event of an *injury*. Courts require a causal link between the injury suffered and an individual’s deviation from an accepted standard of care in order for the individual or entity to be held liable. However, there are circumstances under which a finding of fault is not required in order to hold an individual or entity liable for injury (see “[Strict Liability](#)”). The relationship between the parties plays a large role in the types of legal claims asserted in the event of an injury (see “[Malpractice Liability](#)”).

Malpractice Liability

Malpractice liability attaches to an individual or entity engaged in the practice of rendering services. For example, health care professionals are liable to their patients, and lawyers and legal professionals are liable to their clients. Malpractice liability is one of the most common legal claims made by client-plaintiffs against professionals. (Sales et al. 2005). In a malpractice suit, the client-plaintiff “alleges that he or she suffered a physical, mental, or financial injury because an [individual professional] did not exercise the level of ordinary and reasonable care practiced by the average member of that discipline in the same or similar community” (Sales et al. 2005).

Strict Liability

Strict liability is “liability that does not depend on actual negligence or intent to harm, but that is based on the breach of an absolute DUTY to make something safe” (Garner 2004). Therefore, strict liability will apply in appropriate situations to an individual or entity without requiring a showing that the liable party was at fault for the injury. “[S]trict liability most often applies either to ultrahazardous activities or in products-liability cases,” where individuals claim injuries (physical, mental, financial and otherwise) resulting from defective or otherwise dangerous products (Garner 2004).

See Also

- ▶ [Dispute Resolution Procedures](#)
- ▶ [Eligibility \(for Services Under IDEA/ADA, etc.\)](#)

References and Reading

Sources

- Garner, B. (Ed.). (2004). *Black's law dictionary* (8th ed.). St Paul: Thomson/West.
- Oller, J. W., & Oller, S. D. (2010). *Autism: The diagnosis, treatment, & etiology of the undeniable epidemic*. Sudbury: Jones & Bartlett.
- Sales, B. D., Miller, M. O., & Hall, S. R. (2005). *Laws affecting clinical practice*. Washington, DC: American Psychological Association.

Licensed Behavior Analyst

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A “Professional License” is based on the legal permission of a governmental agency to engage in a regulated activity. Regulated activities for professional licensure are generally a specified set of behaviors that define or belong to a profession, often articulated within the “scope of practice” of that profession found in the legislation developed to create such a license standard. The overarching purpose of licensure is to protect the public by requiring minimum standards to engage in a profession and by informing the public who has met those standards. Mandatory governmental regulations are designed to protect consumers

from inadequately trained and unethical practitioners and give consumers legal recourse should malpractice occur. For professions that require specialized education and training, licensure establishes legally enforceable standards that restrict practice to qualified individuals who have met specific qualifications in education and supervised work experience and have passed a formal examination process.

The requirements for licensure vary across states. Licensing laws generally include provisions to restrict the use of the professional title (e.g., “Applied Behavior Analyst”) to licensees or restrict non-licensed individuals from performing activities that are included within the licensing law’s scope of practice. Some state laws regulate both the title and the practice (e.g., Kentucky, Texas). A licensed behavior analyst is a person who has met the minimum requirements specified in the state’s licensure law and has been issued a license by the governing body, allowing them to engage in behavior analytic activities specified in the licensure law. The purpose of licensure is to protect consumers by restricting practice to individuals who have met specific qualifications as determined by the issuing entity. Licensing laws typically include civil or criminal penalties for the unlicensed use of regulated titles or engaging in the practice of the profession without a license. Licensure laws may also include specified ethical and professional standards and give governing boards the authority to suspend, place on probation, or revoke licenses if licensees have been found in violation of those standards.

Although licensure is common for many professions (e.g., medicine, nursing, K–12 education), licensure of behavior analysts is a fairly recent phenomenon, with Massachusetts, in 2008, being the first state to file a bill to license applied behavior analysts. Pennsylvania enacted the first law in 2008, followed by Nevada and Oklahoma in 2009. To date, 28 states have created statutory provisions for licensure of applied behavior analysts. At present almost all licenses for behavior analysts are issued by the states in the United States.

While the criteria for licensure of behavior analysts vary substantially, virtually all states

have articulated specific minimum requirements in education and supervised work experience as well as achieving a passing score on a formal examination. A number of states require that applicants meet the criteria for attaining certification as a Board Certified Behavior Analyst (BCBA[®]) as required by the Behavior Analysis Certification Board[®], while other states do not require this certification or have adopted more stringent standards. Some state statutes also regulate paraprofessional providers of behavior analysis services. Finally, the behavior analyst licensing statutes typically include exemptions for certain individuals (e.g., family members, academic faculty, and students) or for members of certain professions who might engage in behavior analytic activities within the scope of practice within their discipline (e.g., speech and language pathologists).

Because of the variability with respect to eligibility for licensure, states also differ in terms of reciprocity of licensure and ability to practice. Although some states do not currently license behavior analysts, they may have statutory regulations which prohibit individuals from utilizing the title “behavior analyst” without BCBA[®] certification (Connecticut). In addition, some third party payers may require licensure or certification for full reimbursement of services even if the state does not require practitioners to possess the credential.

In addition to identifying titles and activities that will be regulated, licensure laws also identify which entity will be responsible for overseeing the provisions of the law. The governing entity may be a board or a committee that operates independently or under the oversight of an existing board. Licensure laws also specify who will be a member of the board or committee. The governmental entity and members administering licensure of behavior analysts also vary across states. A few states have independent behavior analyst licensing boards (e.g., Louisiana, Kentucky), while other states have boards that regulate several different professions (e.g., Texas, Massachusetts), and others rely on a specially constituted committee that operates under the licensing board of another profession such as psychology (e.g., Missouri, Ohio).

A final component of licensure laws worth noting is exemptions; identifying certain individuals that are not required to be licensed. These exemptions vary from state to state but are generally centered on a few considerations. Since the licensure of behavior analysts is relatively new to the occupational regulation landscape, it is possible that other disciplines already include behavior analysis in their scope of practice. If this is the case, generally those professionals are exempted from being licensed as behavior analysts if they are already licensed as another type of professional. In addition, individuals who may practice behavior analysis with animals or organizations, academics who teach behavior analysis courses, and students of behavior analysis are also typically exempt from licensure laws.

Despite the variations in title and practice regulation, requirements for licensure of behavior analysts, governing entities, and exemptions across states, each of these efforts has been implemented to protect consumers of behavior analytic services. Restricting the practice of behavior analysis to qualified individuals, creating legally enforceable standards, ensuring behavior analytic practitioners meet minimum standards of competence and ethical behavior, and giving consumers the right to legal recourse are a few of the benefits of the licensure of behavior analysts.

Life Satisfaction in Autism

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Definition

Life satisfaction refers to the subjective appraisal of one's satisfaction with their overall life circumstances. This concept may be conceptualized broadly, as individuals may vary in the relative weight of various life factors (e.g., family, friends, school, self) on their life satisfaction. Life satisfaction may also be domain-specific, such that

individuals may be asked to provide a rating of their satisfaction within specific areas. Further, life satisfaction may be distinguished from objective indicators of functioning, such as quality of life. The study of life satisfaction arose from the field of positive psychology, which emphasizes the scientific study of human flourishing; this emphasis on positive indicators of functioning is particularly important within the field of disability (see Schalock 2004 for a detailed discussion). Research with youth has demonstrated that focusing assessment on disordered symptomatology alone neglects important aspects of functioning, and optimal mental health assessment includes indicators of positive aspects of functioning (Greenspoon and Saklofske 2001; Suldo and Shaffer 2008).

Several life satisfaction measures have been validated for use with children and adolescents, including the Students' Life Satisfaction Scale, which is a brief measure that assesses youth's overall life satisfaction on a Likert scale (Huebner 1991). To obtain domain-specific ratings, the Brief Multidimensional Students' Life Satisfaction Scale may be used (BMSLSS; Huebner 1994). This measure assesses satisfaction in various domains (e.g., friends, family, neighborhood, school, self) on a Likert scale. Both measures require that individuals have the ability to comprehend short sentences and select a response on the Likert scale.

Research has recently begun to seek the perspective of children and adolescents with autism spectrum disorder (ASD) regarding life satisfaction. McDougall et al. (2012) surveyed 400 children and adolescents with chronic conditions, 35 of whom were diagnosed with ASD. Participants completed global and domain-specific measures of life satisfaction, and the results indicated that youth with chronic conditions reported high levels of life satisfaction. On the BMSLSS, participants' mean score corresponded with the response options indicating that youth were "mostly satisfied" or "pleased" with their lives. Further, youth responses were highly correlated with parental estimates of their children's life

satisfaction; this level of agreement was consistent with that obtained in studies assessing caregiver-youth agreement within typically developing populations (Dew and Huebner 1994; Gilman and Huebner 1997). In a study primarily assessing quality of life, Egilson et al. (2017) administered the psychological well-being scale of the KIDSCREEN-27 with 96 youth with ASD. This subscale asks participants to rate their satisfaction with and enjoyment of their lives. When compared to peers without ASD, those with ASD reported lower levels of satisfaction. Consistent with previous research, Franke et al. (2019) administered the BMSLSS with 46 adolescents with ASD and their caregivers, and the results indicated that youth reported high levels of life satisfaction across domains. However, they reported significantly lower levels of family, friend, self, and overall life satisfaction than did a typically developing comparison group. Within this study, youth's self-reported levels of life satisfaction correlated with self-reports of self-efficacy, self-awareness, persistence, school support, family coherence, peer support, emotion regulation, empathy, optimism, and gratitude. While there is limited research directly assessing life satisfaction in individuals with ASD, the implications of the current body of work suggest that youth with ASD view their life as satisfying. However, this level of life satisfaction may be slightly below that of youth without ASD.

See Also

- [Friendship Satisfaction in Children with ASD](#)
- [Personal Perspectives on Autism](#)
- [Quality of Life for Transition-Age Youth with ASD](#)

References and Reading

- Dew, T., & Huebner, E. S. (1994). Adolescents' perceived quality of life: An exploratory investigation. *Journal of School Psychology, 32*, 185–199.

- Egilson, S. T., Olafsdóttir, L. B., Leósdóttir, T., & Saemundsen, E. (2017). Quality of life of high-functioning children and youth with autism spectrum disorder and typically developing peers: Self- and proxy-reports. *Autism, 21*, 133–141. <https://doi.org/10.1177/1362361316630881>.
- Franke, K. B., Hills, K., Huebner, E. S., & Flory, K. (2019). Life satisfaction in adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 49*, 1205–1218. <https://doi.org/10.1007/s10803-018-3822-4>.
- Gilman, R., & Huebner, E. S. (1997). Children's reports of their life satisfaction: Convergence across raters, time and response formats. *School Psychology International, 18*, 229–243.
- Greenspoon, P. J., & Saklofske, D. H. (2001). Toward an integration of subjective well-being and psychopathology. *Social Indicators Research, 54*, 81–108. <https://doi.org/10.1023/A:1007219227883>.
- Huebner, E. S. (1991). Initial development of the Students' Life Satisfaction Scale. *School Psychology International, 12*, 231–243.
- Huebner, E. S. (1994). Preliminary development and validation of a multidimensional life satisfaction scale for children. *Psychological Assessment, 6*, 149–158. <https://doi.org/10.1037/1040-3590.6.2.149>
- McDougall, J., Wright, V., Nichols, M., & Miller, L. (2012). Assessing the psychometric properties of both a global and a domain-specific perceived quality of life measure when used with youth who have chronic conditions. *Social Indicators Research, 114*, 1243–1257. <https://doi.org/10.1007/s11205-012-0200-z>.
- Schalock, R. L. (2004). The emerging disability paradigm and its implications for policy and practice. *Journal of Disability Policy Studies, 14*, 204–215. <https://doi.org/10.1177/10442073040140040201>.
- Seligson, J. L., Huebner, E. S., & Valois, R. F. (2003). Preliminary validation of the Brief Multidimensional Students' Life Satisfaction Scale (BMSLSS). *Social Indicators Research, 61*, 121–145.
- Suldo, S. M., & Shaffer, E. J. (2008). Looking beyond psychopathology: The dual-factor model of mental health. *School Psychology Review, 37*, 52–78.

Life Skills

- Preemployment Skills for People with ASD

Life Skills Classroom

- Self-Contained Classroom

Limbic System

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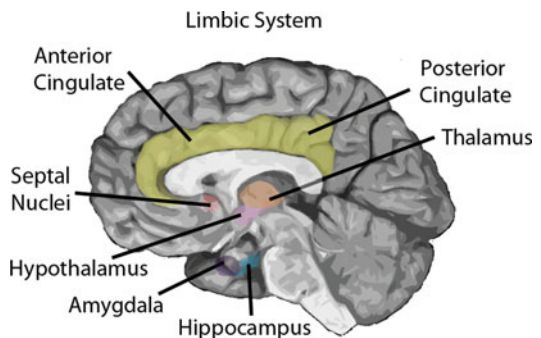
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Definition

The term “limbic system” generally refers to a group of brain structures located on the inner border of the cerebrum. They are thought to play a role in processing emotions (Fig. 1).

Historical Background

The list of brain structures considered to be part of the “limbic system” has evolved significantly since its initial conception (Papez 1937; Maclean 1949). Most often the list includes the amygdala, hippocampus, cingulate cortex, septal nuclei, thalamus, and hypothalamus as core structures. The term “limbic” is considered somewhat obsolete by most neuroscientists since there are no accepted anatomical or functional criteria for which structures should be included as part of the system (LeDoux 2000). However, the term is still used to describe the general group of structures that contribute to emotional function in the brain.



Limbic System, Fig. 1 Brain structures commonly associated with the limbic system

General Function of Limbic System Structures

Amygdala

The amygdala is an almond-shaped brain region made up of a group of nuclei located in the anterior portion of the temporal lobe (Freese and Amaral 2009). A widely held view is that the amygdala is essential for learning the emotional significance of a stimulus in the environment, detecting potential danger, and adjusting levels of vigilance. The amygdala plays a modulatory role in social behavior including recognizing emotion in faces, judging the trustworthiness of a person, and generating a sense of personal space (Adolphs 2010).

Hippocampus

The hippocampus is located in the medial temporal lobe posterior to the amygdala. The primary functions of the hippocampus are to consolidate short-term memories into long-term memories and perform spatial navigation (see “The Hippocampus Book”, ed. Andersen, Morris, Amaral, Bliss, and O’Keefe). The hippocampus was originally the centerpiece for the limbic system concept (Maclean 1949); however, studies of patients with hippocampal lesions, sustained by seizures, Alzheimer’s disease, or surgical removal, demonstrate a distinct deficit in the ability to form and retain new memories while still able to regulate emotional states.

Cingulate Cortex

The cingulate cortex is a gyrus that wraps around the top of the corpus callosum on the medial surface of the brain and is typically divided into two regions, the anterior cingulate and the posterior cingulate. The anterior cingulate plays a role in several cognitive functions such as decision making, empathy, reward anticipation, motivation, and error detection as well as autonomic functions such as blood pressure and heart rate. The posterior cingulate is part of a “resting state” network that is active when a person is introspective or “daydreaming” in which thoughts are directed to the past or future as well as the interaction between

emotions and memory (Maddock 1999) and the detection of familiar voices and faces.

Thalamus

The thalamus is a bulb-shaped mass consisting of a group of subnuclei. The thalamic nuclei are located on the medial surface of the brain under the corpus callosum and above the brainstem and hypothalamus. The primary function of the thalamus is to relay sensory information between cortical and subcortical areas, in that it receives sensory signals (i.e., visual, auditory, somatosensory) and sends them to the appropriate brain regions for processing (Jones 2007). The thalamus also plays an important role in regulating sleep and arousal.

Hypothalamus

The hypothalamus is located below the thalamus and contains a group of subnuclei that regulate the secretion of hormones and metabolic processes, such as body temperature, blood pressure, appetite, thirst, fatigue, sexual and maternal behavior, and circadian rhythm.

Septal Nuclei

The septal nuclei, located below the most anterior portion of the corpus callosum, plays a primary role in experiencing pleasure, analyzing reward, and reinforcing behavior based on the value of the reward.

Current Knowledge

Pathology and Dysfunction of the Limbic System in Autism

Impairments in emotional processing are one of the central features of autism. This suggests the possibility that the structures within the limbic system would be pathological in people with the disorder (Amaral et al. 2008; Courchesne 2004). The amygdala, perhaps more than any other structure in the brain, has been found to be both structurally and functionally abnormal in autism. There is some evidence for hippocampal pathology in autism, but the main function of the hippocampus

in memory does not appear to be affected in autism. There is little evidence that shows abnormalities in the structure of the cingulate gyrus in autism. However, many imaging studies have shown the function of the cingulate to be affected.

There is little evidence for pathology in the thalamus, hypothalamus, and septal nuclei in autism. These regions, however, serve an important role in controlling the physiology and function of many other brain regions. For this reason, they may indirectly lead to the pathology of autism. Since there is clear evidence showing the pathology within the amygdala, hippocampus, and anterior cingulate cortex in autism, these regions of the brain will be discussed further below.

Amygdala

An emerging hypothesis suggests that the amygdala plays a role in directing visual attention to people or things in the environment that pose a potential threat or are critical to survival. In humans, the amygdala particularly directs visual attention to the eye region of the face to allow for detection of important social and emotional cues. However, several studies have now shown that people with autism spend less time looking at the eye region of the face. A study done by Dalton and his colleagues (2005) shows that the amount of time people with autism spend looking at the eye region of the face is strongly and positively correlated with amygdala activation. This means that when people with autism do look at the eye region of the face, the amygdala is activated, but they do this less often and therefore the amygdala is activated less often. However, this is a chicken and egg question. Do people with autism spend less time looking at the eye region of the face because the amygdala does not direct attention there, indicating that they do not go seeking emotional and social cues the same way as typical developing individuals do? This possible explanation suggests that normal function of the amygdala is to direct attention, based on motivation, to attend to the eye region of the face. Thus, individuals with autism are simply less motivated to look at people in the eyes (Pierce et al. 2004). Or, a second possibility is that they purposely do not

look at the eye region of the face because they perceive social interaction and eye contact as threatening and which therefore increases their level of anxiety. Given the amygdala's role in fear and anxiety, one can predict heightened amygdala activation in people with autism when they engage in eye contact, as found in the Dalton et al. (2005) study. This might be caused by increased emotional, or even fearful, response when autistic individuals look at another person's eyes.

The amygdala is also structurally pathological in autism. However, the nature of the pathology changes with age. For example, studies of younger children show that the amygdala is enlarged in those with autism compared to typical children (see Schumann et al. 2009 for review). However, studies of older adolescents, adults, or a wide age range of subjects have found either no difference or even smaller amygdala volumes in individuals with autism. In addition, the amygdala continues to grow in size throughout adolescence in typically developing male children, but this growth pattern does not seem to take place in male children with autism (Schumann et al. 2004). Recent evidence from studies of children early in development suggests considerable heterogeneity in amygdala growth among those with autism. Nordahl et al. (2012) found that within subgroups, some, but not all, children with autism have a rapidly growing amygdala. It is unknown if children in different subgroups also have a different form or cause of autism.

Therefore, on average, the amygdala appears to be initially larger than normal in children with autism. It, however, does not go through the same preadolescent increase in volume that takes place in typically developing children. There is some evidence that a decreased number of neurons may lead to the reduction in amygdala volume later in adulthood (Schumann and Amaral 2006). It is unclear if the reduction in neuron number is present early in development, and some other factor contributes to the early increase in volume, or if there is a degenerative process taking place in the amygdala in people with autism.

Hippocampus

There is some evidence that the anatomical development of the hippocampus in individuals with autism is different from that of a typical development. In general, studies have found either no difference or a moderate enlargement (Schumann et al. 2004) in hippocampal volume between autism and typically developing control subjects. Shape analyses and proton magnetic resonance spectroscopy ($^1\text{H-MRS}$) that are used to characterize concentrations of brain metabolites have been used as an indication of pathology (see Dager et al. 2008 for review).

Margaret Bauman and Tom Kemper carried out the first groundbreaking studies during the mid-1980s to early 1990s to systematically describe pathology in a sample of postmortem brains from people with autism (Bauman and Kemper 1994). Over this series of studies, they collected and described six cases of autism. Five of these cases had mental retardation and four had epilepsy. They did a side-by-side comparison of each autism case with an age- and sex-matched control. They then observed that neurons in the amygdala and hippocampus of most autism cases appeared unusually small and more densely packed (i.e., increased numbers of small neurons per unit volume) than in age-matched controls. These findings suggest abnormalities at an early stage in brain development, a time at which the size of neurons is not fully mature. These changes could result in a curtailment of normal brain maturation.

Recent studies also propose widespread abnormalities in the GABAergic system of the brain in individuals with autism, including the hippocampus (Blatt et al. 2001; Guptill et al. 2007). GABA is the chief inhibitory neurotransmitter in the brain that regulates the communication between neurons. It is unclear if or how an abnormally functioning GABAergic system in the hippocampus might result in behavioral impairments in autism. Another possibility is that an abnormally functioning hippocampus might actually result in improved spatial or declarative memory in people with autism (see Schumann et al. 2004 for review).

Hippocampal pathology in autism may not directly result in obvious behavioral impairments; but it may be linked with the occurrence of seizure disorder. Approximately 30% of individuals with autism also have some form of epilepsy with a strong correlation between intellectual impairment and seizure prevalence (see Tuchman et al. 2010 for review). Abnormalities in the GABAergic system in the hippocampus could reduce the threshold for development of seizures in children with autism.

Cingulate Cortex

Structural MRI studies have not indicated either the anterior or the posterior cingulate as areas of obvious pathology in autism. In a longitudinal MRI study, Schumann et al. (2010) reported that the cingulate cortex, along with the frontal and temporal cortices, grew at an abnormal rate in young children with autism. By 2.5 years of age, these regions were enlarged relative to typical children at the same age. In contrast, there is significant evidence from functional imaging studies that suggest abnormal activation and connectivity in these regions (see Minshew and Keller 2010 for review).

In the seminal postmortem studies carried out by Kemper and Bauman (1993), anterior cingulate cortex was the only region of neocortex that shows abnormalities. The cortex is typically organized into layers of neurons; the layers in the autism cases were not as organized as this typical control cases. There are also abnormalities in the GABAergic system in the anterior cingulate that may affect normal neuron communication (Oblak et al. 2009).

A recent study found abnormalities in the white matter below the anterior cingulate, which is made up of fiber tracts (i.e., axons) for neurons to communicate over various distances in the brain. Specifically, they found an excessive number of thin axons, which link neighboring areas together, and a decreased number of large axons that communicate over long distances (Zikopoulos and Barbas 2010). This study suggests that the connections in the brain did not develop normally in autism, which may affect communication between brain regions and

subsequently lead to behavioral abnormalities. However, this study only examined four brains from people with autism and therefore more studies are needed.

There is little evidence that suggests the posterior cingulate to be pathological in individuals with autism. Oblak et al. (2011) have reported that there are changes in the distribution of neurons. This suggests that there is a defect during prenatal brain development in which neurons do not migrate to the correct location in the cortex. This study also found abnormalities in the GABAergic system as well.

There is some evidence that the anterior cingulate is not functioning properly in autism. One study found that the anterior cingulate is activated more in adults with autism relative to typical adults while performing a task that required sustained attention for a monetary reward (Schmitz et al. 2008). The more socially impaired the person is, the more activated the anterior cingulate becomes during this task. The anterior cingulate is thought to be involved in cognitive aspects of error detection and risk assessment during reward tasks. This increased activation in autistic adults can either be caused by their need to compensate for other impairments or that the monetary reward is a greater incentive.

Individuals with high functioning autism also do not activate the cingulate cortex as much as typical individuals do while looking at familiar faces; this suggests that they have a lower emotional response to people they know personally (Pierce and Redcay 2008). People with autism also do not show normal activation in the “default network” during rest; this lack of activation indicates a lack of introspective and self-reflective thinking (Kennedy and Courchesne 2008).

See Also

- Amygdala
- Amygdaloid Complex
- Hippocampus
- Thalamus

References and Reading

- Adolphs, R. (2010). What does the amygdala contribute to social cognition? *Annals of the New York Academy of Sciences*, 1191, 42–61.
- Amaral, D. G., Schumann, C. M., & Nordahl, C. W. (2008). Neuroanatomy of autism. *Trends in Neurosciences*, 31(3), 137–145.
- Andersen, P., Morris, R., Amaral, D. G., Bliss, T., & O’Keefe, J. (2006). *The hippocampus book*. Oxford Neuroscience Series.
- Bauman, M. L., & Kemper, T. L. (1994). *The neurobiology of autism*. Baltimore: Johns Hopkins University Press.
- Blatt, G. J., Fitzgerald, C. M., Guptill, J. T., Booker, A. B., Kemper, T. L., & Bauman, M. L. (2001). Density and distribution of hippocampal neurotransmitter receptors in autism: An autoradiographic study. *Journal of Autism and Developmental Disorders*, 31(6), 537–543.
- Courchesne, E. (2004). Brain development in autism: Early overgrowth followed by premature arrest of growth. *Mental Retardation and Developmental Disabilities Research Reviews*, 10(2), 106–111.
- Dager, S. R., Corrigan, N. M., Richards, T. L., & Posse, S. (2008). Research applications of magnetic resonance spectroscopy to investigate psychiatric disorders. *Topics in Magnetic Resonance Imaging*, 19(2), 81–96.
- Dalton, K. M., Nacewicz, B. M., Johnstone, T., Schaefer, H. S., Gernsbacher, M. A., Goldsmith, H. H., Alexander, A. L., & Davidson, R. J. (2005). Gaze fixation and the neural circuitry of face processing in autism. *Nature Neuroscience*, 8(4), 519–526.
- Freese, J. L., & Amaral, D. G. (2006). Synaptic organization of projections from the amygdala to visual cortical areas TE and V1 in the macaque monkey. *The Journal of Comparative Neurology*, 496(5), 655–667.
- Freese, J., & Amaral, D. G. (2009). Neuroanatomy of the primate amygdala. In P. J. Whalen & E. A. Phelps (Eds.), *The human amygdala*. The Guilford Press.
- Guptill, J. T., Booker, A. B., Gibbs, T. T., Kemper, T. L., Bauman, M. L., & Blatt, G. J. (2007). [3H]-flunitrazepam-labeled benzodiazepine binding sites in the hippocampal formation in autism: A multiple concentration autoradiographic study. *Journal of Autism and Developmental Disorders*, 37(5), 911–920.
- Jones, E. G. (2007). *The thalamus*. New York: Cambridge University Press.
- Kemper, T. L., & Bauman, M. L. (1993). The contribution of neuropathologic studies to the understanding of autism. *Neurologic Clinics*, 11(1), 175–187.
- Kennedy, D. P., & Courchesne, E. (2008). Functional abnormalities of the default network during self- and other-reflection in autism. *Social Cognitive and Affective Neuroscience*, 3(2), 177–190.
- LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience*, 23, 155–184.
- Maclean, P. D. (1949). Psychosomatic disease and the visceral brain – Recent developments bearing on the Papez theory of emotion. *Psychosomatic Medicine*, 11(6), 338–353.

- Maddock, R. J. (1999). The retrosplenial cortex and emotion: New insights from functional neuroimaging of the human brain. *Trends in Neurosciences*, 22(7), 310–316.
- Minshew, N. J., & Keller, T. A. (2010). The nature of brain dysfunction in autism: Functional brain imaging studies. *Current Opinion in Neurology*, 23(2), 124–130.
- Nordahl, C. W., Scholz, R., Yang, X., Buonocore, M. H., Simon, T., Rogers, S., et al. (2012). Increased rate of amygdala growth in children aged 2 to 4 years with autism spectrum disorders: A longitudinal study. *Archives of General Psychiatry*, 69(1), 53–61.
- Oblak, A., Gibbs, T. T., & Blatt, G. J. (2009). Decreased GABAA receptors and benzodiazepine binding sites in the anterior cingulate cortex in autism. *Autism Research*, 2(4), 205–219.
- Oblak, A. L., Gibbs, T. T., & Blatt, G. J. (2011). Reduced GABAA receptors and benzodiazepine binding sites in the posterior cingulate cortex and fusiform gyrus in autism. *Brain Research*, 1380, 218–228.
- Papez, J. W. (1937). A proposed mechanism of emotion. *Archives of Neurology and Psychiatry*, 38(4), 725–743.
- Pierce, K., & Redcay, E. (2008). Fusiform function in children with an autism spectrum disorder is a matter of “who”. *Biological Psychiatry*, 64(7), 552–560.
- Pierce, K., Haist, F., Sedaghat, F., & Courchesne, E. (2004). The brain response to personally familiar faces in autism: Findings of fusiform activity and beyond. *Brain*, 127(Pt 12), 2703–2716.
- Schmitz, N., Rubia, K., van Amelsvoort, T., Daly, E., Smith, A., & Murphy, D. G. (2008). Neural correlates of reward in autism. *The British Journal of Psychiatry*, 192(1), 19–24.
- Schumann, C. M., & Amaral, D. G. (2006). Stereological analysis of amygdala neuron number in autism. *Journal of Neuroscience*, 26(29), 7674–7679.
- Schumann, C. M., & Nordahl, C. W. (2011). Bridging the gap between MRI and postmortem research in autism. *Brain Research*, 1380, 175–186.
- Schumann, C. M., Hamstra, J., Goodlin-Jones, B. L., Lotspeich, L. J., Kwon, H., Buonocore, M. H., Lammers, C. R., Reiss, A. L., & Amaral, D. G. (2004). The amygdala is enlarged in children but not adolescents with autism; the hippocampus is enlarged at all ages. *Journal of Neuroscience*, 24, 6392–6401.
- Schumann, C. M., Barnes, C. C., Lord, C., & Courchesne, E. (2009). Amygdala enlargement in toddlers with autism related to severity of social and communication impairments. *Biological Psychiatry*, 66(10), 942–949.
- Schumann, C. M., Bloss, C., Barnes, C. C., Wideman, G. M., Carper, R., Akshoomoff, N., Pierce, K., Hagler, D., Schork, N., Lord, C., & Courchesne, E. (2010). Longitudinal MRI study of cortical development through early childhood in autism. *Journal of Neuroscience*, 30(12), 4419–4427.
- Tuchman, R., Cuccaro, M., & Alessandri, M. (2010). Autism and epilepsy: Historical perspective. *Brain & Development*, 32(9), 709–718.
- Zikopoulos, B., & Barbas, H. (2010). Changes in prefrontal axons may disrupt the network in autism. *Journal of Neuroscience*, 30(44), 14595–14609.

Limited Interests

► Restricted Interest

Linear Regression

► Regression Analysis

Linguistic Idiosyncrasies and Neologisms

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Definition

Autistic language often contains unique features and characteristics including linguistic idiosyncrasies and neologisms. Linguistic idiosyncrasies can be defined as the atypical use of a standard word or phrase to express a specific meaning. While the word used is part of the speaker's native language, it is not typically associated with the word or phrase of reference. Neologisms can be described as words that have been created by a speaker and are not considered to be part of the lexicon of a given language. Although, words have been created or coined using old words to create new words. For example, the word “webinar” was originated from “seminar” but one that occurs on the web or the internet. Many children with autism use neologisms or made-up words to refer to specific objects, people, or situations within their environment. For example, a child with autism might call a black and blue mark or bruise, a “blusier.”

As newly coined lexical units, neologisms do create problems for understanding. In fact, in the technical world of computers, translators are often needed to make sense of these new literary units

(Moghadam and Sedighi 2012). Interestingly, there are several ways to create neologisms (see Delabastita 2004, as cited in Moghadam and Sedighi 2012, p.1) including:

- *Borrowing*: integral borrowings (e.g., hobby [English →Dutch, French, etc.]), loans showing graphological or phonological adaptation (e.g., hobby [German]), and structural loans (also known as calques, e.g., sky-scraper).
- *Shifting*: existing words undergo semantic shifts (e.g., bug [concealed microphone]) or grammatical shifts (e.g., foreground, sideline [noun →verb]).
- *Combining*: new formations through derivation (e.g., Thatcherite) and/or compounding (e.g., bubble-headed), or else new collocations (e.g., lateral thinking).
- *Coining*: words are created out of the blue (e.g., Joyce's quark).
- *Imitating*: words are formed by onomatopoeic imitation of noises or sounds (e.g., zoom).
- *Blending*: words are formed from the parts of two others (e.g., channel + tunnel →chunnel).
- *Shortening*: clippings (e.g., science-fiction →sci-fi) acronyms (e.g., AIDS).

Neologisms and idiosyncratic use of language is often seen in psychiatry in individuals with schizophrenia or thought disorders. In these cases, the language is typically difficult to understand or make sense of the context. For children with autism, it appears they often have a unique way of expressing their wants, needs, and observations, particularly as they frequently cannot find the "typical words" to express themselves. Some examples of idiosyncratic language use might be helpful in understanding a child's approach to create meaning with the words to which he or she has access. For example, families have reported the following:

- Instead of asking for a cracker, a child with ASD might say "I want a snack, NOT from the refrigerator and NOT wet. Crunchy from the big cupboard."
- A 6 year old with ASD said "Mommy, can we go again on big snowy mountain rollercoaster

with scary monkey?" instead of asking the name of the roller coaster and then saying "Can we go on Expedition Everest again?"

- A child with ASD might describe things instead of using the correct name. For example, "Fritos are crunch crackers in orange bag that fold to make a letter C."
- A child with ASD responded in the following way when his brother hit him on the arm: "Mommy, Ryan make my skeleton hurt" while holding his arm and crying rather than say "Mommy, Ryan hurt my arm."
- An 8 year old with ASD said "Fix Florida" when he pointed to a scab on his knee in the shape of the state of Florida.
- A 7 year old with ASD asked his mom: "See Dr. Mike" when he had a headache and did not feel well.

Often for children with ASD, with familiarity and context knowledge, family members and care providers can determine the meaning being created by the idiosyncratic use of language or the creation of neologisms. Research in this area of autism is limited but provides an opportunity to examine what approaches (e.g., coining, blending, imitating, etc.) children are typically using to create new words and what profiles of linguistic and cognitive functioning are typical for children with significant idiosyncratic language use.

See Also

- [Idiosyncratic Language](#)
- [Metaphoric Language](#)

References and Reading

- Bogdashina, O. (2005). *Communication issues in autism and Asperger syndrome: Do we speak the same language?* Philadelphia: Jessica Kingsley Publishers.
- Delabastita, D. (2004). Literary style in translation: Archaisms and neologisms. *Übersetzung*, 1, 883–888.
- Moghadam, M. Y., & Sedighi, A. (2012). The study of the translation of neologisms in technical texts: A case of computer texts. *International Journal of Scientific & Engineering Research*, 3(2), 1–6.

- Ohira, K. (1979). The context theory of autism. *Folia Psychiatrica et Neurologica Japonica*, 33(1), 35–41.
- Volden, J., & Lord, C. (1991). Neologisms and idiosyncratic language in autistic speakers. *Journal of Autism and Developmental Disorders*, 21(2), 109–130.

Linguistics of Verbal English (LOVE)

- [Manual Sign](#)
- [Sign Language](#)

Linotril

- [Clonazepam: Definition](#)

Listening Comprehension

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Synonyms

[Comprehension](#); [Receptive language](#); [Spoken language comprehension](#)

Definition

Listening comprehension encompasses the multiple processes involved in understanding and making sense of spoken language. These include recognizing speech sounds, understanding the meaning of individual words, and/or understanding the syntax of sentences in which they are presented. Listening comprehension can also involve the prosody with which utterances are spoken (which can, e.g., change intended meaning from a statement to a question), and making

relevant inferences based on context, real-world knowledge, and speaker-specific attributes (e.g., to what information the speaker has access and about what he/she is likely to be talking). For longer stretches of language or discourse, listening comprehension also involves significant memory demands to keep track of causal relationships expressed within the discourse. It is often viewed as an active process with three main components: attending to the perceptual input (speech), constructing meaning from stretches of speech, and relating what was heard to existing knowledge.

In typical development and developmental disorders other than autism spectrum disorder (ASD), comprehension precedes or is stronger than production, especially early in development. In individuals with ASD, delays in and difficulty with comprehension are common, and the gap between comprehension and production is reduced, though children with ASD still comprehend more words than they produce.

The understanding of written language, or reading comprehension, is assessed separately.

See Also

- [Expressive Language](#)
- [Receptive Language](#)
- [Verbal Comprehension](#)

References and Reading

- Bransford, J. D., & McCarrell, N. S. (1977). A sketch of a cognitive approach to comprehension: Some thoughts about understanding what it means to comprehend. In P. N. Johnson-Laird & P. C. Wason (Eds.), *Thinking: Readings in cognitive science* (pp. 377–399). Cambridge: Cambridge University Press.
- Clark, H. H., & Clark, E. V. (1977). *Psychology and language*. New York: Harcourt Brace Jovanovich.
- Garrod, S. (1986). Language comprehension in context: A psychological perspective. *Applied Linguistics*, 7, 226–238.
- Luyster, R., Kadlec, M., Carter, A., & Tager-Flusberg, H. (2008). Language assessment and development in toddlers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(8), 1426–1438.

Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders*. Hoboken: Wiley.

Literacy

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Synonyms

[Reading](#); [Writing](#)

Definition

Literacy refers to the ability to read and write and to use these skills functionally in social, academic, and vocational contexts. Individuals with proficient literacy skills are able to comprehend text and effectively express ideas through written language. While the decoding skills of individuals with ASD are often within normal limits, they frequently exhibit poor reading comprehension, particularly with narratives and other complex texts. In addition, they may have difficulty with written expression. The ability to read and write effectively can be an important asset for individuals with ASD. Strategies such as providing scripts for activities of daily living (e.g., asking for help at the grocery store) and encouraging the use of written communication (e.g., e-mails) can help enhance their communicative functioning.

See Also

- ▶ [Decoding Skills](#)
- ▶ [Emergent Literacy](#)
- ▶ [Reading](#)
- ▶ [Reading Skills](#)
- ▶ [Writing Disorders](#)
- ▶ [Written Language](#)

References and Reading

- Hedge, M. N., & Maul, C. (2006). *Language disorders in children: An evidence-based approach to assessment and treatment*. Boston: Pearson Education.
- McCauley, R. J., & Fey, M. E. (2006). *Treatment of language disorders in children*. Baltimore: Paul H. Brookes Publishing.
- O'Connor, I. M., & Klein, P. D. (2004). Exploration of strategies for facilitating the reading comprehension of high-functioning students with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 34, 115–127.
- Paul, R. (2007). *Language disorders from infancy through adolescence: Assessment and intervention* (3rd ed.). St. Louis: Mosby.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 335–364). Hoboken: Wiley.

Lithium

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Synonyms

[Lithium carbonate](#); [Lithium citrate](#)

Indications

Mania, depression, aggression, mood disturbances.

Mechanisms of Action

Lithium is an alkali metal and is among the oldest employed psychoactive therapeutic agents. Despite this, its precise mechanism of action remains unclear. These have been as varied as reported modulatory effects on neurotrophic factors, biogenic amines/enzymes, adrenergic function, postsynaptic receptor and supersensitivity,

and second messenger systems including adenylate cyclase, inositol triphosphate, and protein kinase signaling cascades (Alessi et al. 1994). There is, however, a consensus that much of lithium's action is via replacement of sodium ion functionality within the body, particularly in its indirect inhibition of neuronal processes. Inhibition of both adenylate cyclase and protein kinase C has been implicated in anti-manic effects associated with lithium. In terms of its action on biogenic amines, lithium is associated with a stimulatory effect on dopaminergic, noradrenergic, and serotonergic transmission. Recent studies have suggested actions on neurotrophic and neuroprotective mechanisms by changing the ratio of neurotrophic (Bcl-2 and BDNF) to apoptotic (BAX and p-53) factors and inhibition of GSK-3B and of *N*-methyl-d-aspartate receptor-mediated Ca^{2+} influx.

Lithium is also associated with an increase in the white blood cell and platelet count, an effect mediated by its stimulation of granulocyte-macrophage colony-stimulating factor as well as increasing *N*-acetyl-aspartate, a marker of neuronal integrity and gray matter volume. Taken together, it has been suggested that lithium may have protective actions which affect corticolimbic circuitry thought to be involved in affective, behavioral, and emotional regulation. As such, it has been proposed that its action on this circuitry may be involved in its utility in autism.

Specific Compounds and Properties

Lithium is available in a number of salt forms, of which the two most routinely prescribed are lithium carbonate and lithium citrate. Lithium carbonate is generally available in 400 mg tablet formulations, each of which contains 10.8 mmol lithium. Lithium citrate is available in both liquid and tablet formulations (564 mg tablets). Each of the 564 mg tablets contains 6 mmol lithium. The liquid formulation of lithium citrate is available in two strengths (5.4 mmol/5 mL (equivalent to approx. 200 mg of lithium carbonate) and 10.8 mmol/5 mL (equivalent to approximately 500 mg of lithium carbonate)) for twice daily

administration. While the pharmacokinetics of the two most commonly prescribed formulations of lithium are similar, there are important differences in their bioequivalence. It is extremely important that the patient receives the same formulation each time upon a new prescription as confusion in this area could result in suboptimal or even toxic dosing.

The absorption of lithium from the GI tract after oral administration is usually good with maximal blood serum levels usually being achieved in 1–4 h. In terms of its metabolism, this is relatively minor with the primary substance being left unchanged such that 95% of the oral lithium taken is excreted by the renal system. This usually results in typical half-lives of approximately 24 h. Impairment of the kidneys or renal disease will usually impair lithium excretion with the result that the half-life is increased, requiring careful monitoring and reduction of the dosage prescribed. Since lithium acts to replace sodium ion functionality, any change to serum sodium levels can have a major impact on lithium concentrations and their clinical effect. The absorption and excretion of lithium is closely regulated by changes in sodium. For example, excessive sweating, dehydration, or diarrhea will lead to sodium depletion and, as a consequence, plasma lithium levels will be elevated. Equally, dietary changes in salt consumption should be monitored as these can also influence effective lithium serum concentrations.

Clinical Use (Including Side Effects)

Lithium is most commonly utilized as a mood stabilizing agent, particularly in the treatment of manic depression. In the context of autism, it is often used as an antiaggressive agent and for its mood stabilization properties. Other mood stabilizers are also in clinical use in autism such as carbamazepine, which is also employed in the treatment of comorbid seizures. Lithium has key prophylactic actions against both manic and depressive symptoms but is more successful in the management of acute mania compared to acute depression. While initial studies in the

1960s and 1970s demonstrated high clinical response rates in clinical trials, the clinical practice of lithium use has been inhibited by the increasing recognition that its effectiveness as a mood stabilizer in clinical practice is mixed coupled to an increased awareness of its side effect profile. In terms of its use in autism, it is occasionally utilized against aggression and irritability with mixed success.

The utility of lithium as an effective medication in autism is debatable. While some case reports have demonstrated efficacy against aggression measures; full-scale clinical trials have shown mixed results with lithium use. In spite of this, one study in prepubertal children with severe aggression in conduct disorder showed positive effects (Campbell et al. 1995) and its use in autism is typically as an anti-aggressive agent. Furthermore, it has been suggested that the combination of lithium with other pharmacotherapy may offer improvement versus lithium alone. For example, positive results have been reported for the combination of lithium and fluvoxamine against mood disturbances and aggression. One disadvantage of using lithium is that the therapeutic effect is gradual, often taking 1–3 weeks to achieve efficacy and is dependent on patient compliance (since drug administration is orally). Coupled to this, other agents (such as antipsychotics) can be administered in other ways (e.g., intramuscularly) to noncompliant patients and are often associated with more rapid effects on aggression inducing rapid sedation.

In spite of these mixed studies of lithium's efficacy against aggression, it remains a strong candidate where mood disturbances are indicated, but the narrow range of dosing available for titration with lithium in children, adolescents, and adults with autism is of concern. Toxic side effects, notably nephrotoxicity and cardiotoxicity in chronic lithium users, are serious adverse risk events mitigating against the widespread clinical utility of lithium in autism. As a result of such serious adverse events, medication with lithium should be carefully controlled with frequent blood tests necessary for the evaluation of its safety. In other clinical populations, lithium may be of use in treating comorbid aggression (Lettinga et al.

2011), but its treatment in autistic populations may be better performed by atypical antipsychotic agents such as risperidone.

Prior to commencement with lithium therapy, laboratory screening should be performed to assess renal and thyroid function in addition to a complete blood count and ECG. The excretion of lithium is via the kidneys, thus impairment of renal function could alter plasma drug concentrations and dosing required. In terms of thyroid function, lithium is associated with a decrease in free tri-iodothyronine and thyroxine which are normally compensated for in euthyroid patients by elevating thyroid-releasing hormone levels. Patients with impaired thyroid function lose this compensatory mechanism. While lithium does not often induce cardiac complications, it does increase cardiac conductance and is associated with elevated white blood cell and platelet counts. Furthermore, lithium is often associated with cardiac repolarization (ST segment and T-wave) and in one fifth of patients with T-wave flattening. All of these changes should be carefully monitored at half-yearly intervals while lithium is prescribed. In the case of family-planning issues, lithium is often associated with birth defects in pregnant women, and thus its use during pregnancy should be avoided with contraception advised to women of child-bearing age who are taking lithium.

Therapeutic blood concentrations are reported to lie between 0.5 and 1.2 mmol/L with associated side effects being more marked at higher concentrations. Ideally, lithium levels should be measured every 12 h since the last administered dose concurrent with clinical observation.

Therapeutic blood serum levels of lithium are often between 0.5 and 1.2 mEq/L (0.5–1.2 mmol/L) with associated side effects being more marked at higher concentrations. Administration is generally well tolerated, taking 1–2 weeks to achieve a clinical response. Initial doses are usually between 300 and 600 mg orally with elevation by the same amounts in 4–5-day intervals to therapeutic doses of 900–1500 mg daily divided across two doses. In children under 12 years of age, doses are typically 10–30 mg/kg per day resulting in a daily 900 mg dose in a 30 kg child divided across two to three doses. A 450 mg

sustained-release capsule is also available. Dosing resulting in serum levels over 1.5 mEq/L is often toxic. It is advisable to take medication orally after food in order to minimize gastrointestinal (GI) side effects. Ideally, lithium levels should be measured every 12 h since the last administered dose concurrent with clinical observation.

Typically, side effects are moderate but more serious issues can also arise. Children, adolescents, and adults generally tolerate lithium intake well with side effects typically including a dry mouth, metallic taste, nausea, vomiting, diarrhea, headache, tremor, and weight gain. In terms of more serious side effects, ataxia, dysarthria, polyuria, polydipsia, tremor, weakness, and mild, short-term cognitive impairment and confusion can occur. Polydipsia in particular is a side-effect feature seen in twice daily lithium dosing. Lithium-induced tremors tend to be irregular in rhythm and amplitude, often affecting the fingers. These movements of the finger flexion and extension often have a jerky character. The adrenergic beta-blockers such as propranolol, metoprolol, and nadolol can be useful therapeutics in the management of lithium-induced tremors. Some skin conditions such as acne or psoriasis can be exacerbated by lithium usage. Lithium has a low therapeutic index and so regular blood serum monitoring is warranted. Some side effects (notably polyuria) tend to become more frequent when blood serum lithium levels are above 1 mmol/L. In patients taking lithium chronically, monitoring for potential hypothyroidism should be considered which can be remediated by thyroxine replacement therapy. Hypothyroidism as a side effect of lithium is quite common with middle-aged women particular suffering from this side effect (up to 20% of users in this age group). Thyroid function usually returns to normal after discontinuation of lithium use. Lithium treatment can also increase the risk of hyperparathyroidism and as such, chronic lithium use should be accompanied by blood serum calcium level monitoring.

Long-term lithium use can also be associated with nephrotoxicity and cardiotoxicity. Approximately one-fifth of patients demonstrate a decrease in the glomerular filtration rate which is often benign but can result in a very small subset

of patients in interstitial nephritis. Similarly, lithium can also induce nephrogenic diabetes insipidus (hence the occurrence of polyuria and increased thirst) but this is usually reversible except in extreme long-term (>15 years) users.

Above serum lithium levels of 1.5 mmol/L, mild gastrointestinal, cardiac, and CNS toxic effects of lithium are apparent, with disorientation, seizures, coma, and death occurring at serum concentrations above 2 mmol/L of lithium. If suspected, lithium use should be discontinued and blood samples sent for analysis of serum lithium, creatinine, and electrolyte concentrations in order to guide the rate and nature of fluid replacement. Treatment of lithium toxicity should employ osmotic diuresis or forced alkaline diuresis with toxic serum lithium concentrations above 3 mmol/L requiring peritoneal or hemodialysis. An ECG should also be performed if lithium toxicity is apparent as there is a risk of cardiac arrhythmia. Dependent on the individual, there can be quite a wide variation in the degree and severity of toxic side effects coupled to different serum lithium levels and as such it should be managed on a case-by-case basis.

References and Reading

- Alessi, N., Naylor, M. W., Ghaziuddin, M., & Zubieta, J. K. (1994). Update on lithium carbonate therapy in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 33, 291.
- Belmaker, R. H., Avissar, S., & Schreiber, G. (1991). Effect of lithium on human neurotransmitter receptor systems and G proteins. In N. J. Birch (Ed.), *Lithium and the cell: Pharmacology and biochemistry* (pp. 113–119). London: Academic Press.
- Campbell, M., Adams, P. B., Small, A. M., Kafantaris, V., Silva, R. R., Shell, J., Perry, R., & Overall, J. E. (1995). Lithium in hospitalized aggressive children with conduct disorder: A double-blind and placebo controlled study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 34, 445.
- Gitlin, M. J., & Altshuler, L. L. (1997). Unanswered questions, unknown future for one of our oldest medications. *Archives of General Psychiatry*, 54, 21–23.
- Jefferson, J. W., Greist, J. H., & Ackerman, D. L. (1987). *Lithium encyclopedia for clinical practice*. Washington, DC: American Psychiatric Association.
- Lettinga, J. R., Drent, C., Hoekstra, P. J., Buitelaar, J. K., & Glennon, J. C. (2011). Clinical pharmacology of conduct disorder: A critical review. *Child and Adolescent*

Psychopharmacology News, 16(6), 1–10. <https://doi.org/10.1521/capn.2011.16.6.1>.

- Manji, H. K., Potter, W. Z., & Lennox, R. H. (1995). Signal transduction pathways. Molecular targets for lithium's actions. *Archives of General Psychiatry*, 52, 531–543.
- Mitchell, J. E., & MacKenzie, T. B. (1982). Cardiac effects of lithium therapy in man: A review. *The Journal of Clinical Psychiatry*, 43, 47.
- Shou, M. (1997). Forty years of lithium treatment. *Archives of General Psychiatry*, 54, 9–15.
- Sugarman, J. R. (1984). Management of lithium intoxication. *Family Practice*, 18, 347.

Lithium Carbonate

► [Lithium](#)

Lithium Citrate

► [Lithium](#)

Litigation, Parent-Initiated

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Parent-initiated lawsuits alleging that school districts have failed to provide appropriate services for their children with ASD are governed by the Individuals with Disabilities Education Act (IDEA). IDEA establishes a parent right to sue, specifies the applicable procedural steps, and allows for parents to bring suit on their own without the assistance of an attorney.

The Right of Parents to Initiate Litigation

Section 1415(i)(2)(A) of IDEA provides that “any party aggrieved by the findings and decision” of a

school district “shall have the right to bring a civil action.” An aggrieved party may file a civil lawsuit in either state or federal court.

In its 2007 decision in *Winkelman v. Parma City School District*, the United States Supreme Court recognized that parents who object to a school district’s educational plan under IDEA are “parties aggrieved.” Thus, the parents may file suit against school districts.

In *Winkelman v. Parma City School District*, the United States Supreme Court also implicitly recognized three grounds for parental lawsuits under IDEA. Parents may challenge the substantive sufficiency of the Free Appropriate Public Education (FAPE) that the school district provides, may challenge the adequacy of the procedure accorded the parents, and the parents may sue for private school expenditures necessary to provide a child with an appropriate education.

The Procedural Steps

The regulations that accompany IDEA provide that each state must establish a State Educational Agency (SEA) to address complaints filed by parents and other aggrieved parties. In addition, the SEA must promulgate written procedures for resolving complaints.

Unless state law specifies otherwise, aggrieved parties must file complaints with the SEA “within two years of the date the parent or agency knew or should have known about the alleged action that forms the basis of . . . complaint.” The complaint “must remain confidential.”

The school district must respond to the parent’s complaint within 10 days of receiving it. The response must include “[a]n explanation of why the agency proposed or refused to take the action raised in the . . . complaint,” describe the procedures it used to reach its decision, and articulate other options that it considered and rejected.

Within 15 days of receiving notice of the complaint, the district must convene a meeting with the parent and members of the Individualized Education Program (IEP) Team that took the action about which the parent has complained. If

the district fails to resolve the complaint to the parent's satisfaction within 30 days, the SEA may hold a due process hearing to assess the complaint's validity. If the parent fails to participate in the hearing, the SEA hearing officer may dismiss the complaint.

Unless state law specifies otherwise, parents have 90 days following the hearing officer's decision in the due process hearing to file a civil lawsuit.

The IDEA regulations also provide that states allow the parents and school district to resolve disputes through a mediation process. Participation in mediation is voluntary and the utilization of mediation alters the dispute resolution timeline by allowing time for the parties to reach a resolution through mediation.

Finally, the regulations that accompany IDEA provide that states must develop and make available model complaint forms. The state may not, however, require that parents or other aggrieved parties use the model forms.

Right to Proceed Without an Attorney

In *Winkelman v. Parma City School District*, the United States Supreme Court held that parents can prosecute an IDEA civil suit pro se, without utilizing the services of an attorney.

See Also

- ▶ [Free Appropriate Public Education](#)
- ▶ [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- Individuals with Disabilities Education Act (IDEA), 20 U.S.C. § 1400 et seq. (2012).
- Individuals with Disabilities Education Act (IDEA) regulations, 34 CFR § 300.519 et seq. (2012).
- Winkelman v. Parma City School District*, 550 U.S. 516. (2007).
- Zirkel, P. A. (2012). Autism litigation: Disproportionality. *Journal of Special Education Leadership*, 24(2), 92–103.

Little Brain

- ▶ [Cerebellum](#)

Little's Disease

- ▶ [Cerebral Palsy](#)

Living Arrangements in Adulthood

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Definition

Adult residential models offer individuals with autism a range of services that support different levels of independence, input, and community integration. This article reviews independent living, supported living, group home, and ICF-MR models of residence and the variations and issues surrounding these programs.

Historical Background

The history of adult residential programs follows the progression from overcrowded large-scale institutions that function on a custodial care model to a continuum of services designed to support independence and foster community integration for all individuals on the autism spectrum. The deinstitutionalization movement emerged as the world became more aware and accepting of individuals with disabilities, causing a social shift toward inclusion and better treatment. The growth in social consciousness was matched by the

government passing legislation that increased patients' rights, subsidized the creation of new programs, and reduced the financial burden on individuals, allowing them greater choice in treatment.

At the turn of the century, the social and political approach to individuals with disabilities was segregation. There were two options for individuals with disabilities: live with their family with no services or move into a government institution (Gerhardt 2009). Since their creation in the late nineteenth century, institutions were overcrowded. By 1940, a third of American institutions had between 2,000 and 5,000 residents each (the maximum holding 9,177) (Braddock and Parish 2001). The patients had few rights and no public voice. As the overcrowding problem increased, the courts gave more control to the institutions, resulting in increased sterilization, lobotomy, shock treatment, and euthanasia. Institutions functioned on a custodial care model, providing the most basic necessities for life but offering little treatment and no freedom.

Beginning in the 1950s, the parents of disabled children began to organize and advocate for the development of services and programs that could keep their children out of institutions. Private schools and day programs were developed focusing on the needs of children diagnosed with intellectual disabilities, many of which would not accept children with an autism diagnosis (Gerhardt 2009). Autism at this time was still struggling for recognition. It was frequently misdiagnosed as childhood schizophrenia, intellectual disabilities, or various psychiatric disorders. Programs designed for children with ID did not meet many of the social and communication needs of children with autism (Van Bourgondien and Elgar 1990).

With the election of John F. Kennedy, mental health and the country's treatment of disabilities was brought to the forefront of national consciousness. The President's Panel on Mental Retardation produced 95 recommendations to reduce the need for institutions through community services and greater integration into society. It also required all 50 states to develop plans to create preventative services and improved

residential options. JFK transitioned the national attitude away from segregation toward giving citizens with mental illness a place in American society (Braddock and Parish 2001). The 1963 Mental Retardation Facilities and Community Health Centers Construction Act sparked the creation of both public and private nonprofit community health centers.

President Kennedy commenced the migration out of institutions into smaller community-based homes. In the 1969 President's Committee on Mental Retardation, Dr. Bengt Nirje described this as the normalization movement. The driving principle of the movement was to give the mentally disabled a life that is as close to the norm as possible (Mesibov 1976). This was implemented by creating community-based programs and developing new models of residential services that focused on the humanization of the residents.

The early 1970s brought a series of landmark developments in public policy and law that enhanced the lives of individuals with intellectual disabilities and drove society further away from institutions. *Wyatt vs. Stickney* gave the right to treatment to all individuals experiencing mental illness (Mesibov 1976). Institutions could no longer simply maintain a custodial care mentality; they were required to provide treatment to all of their residents. During this time, Medicaid was founded as part of the Social Securities Act, which gave federal monies to facilities that met ICF/MR standards. This meant that states would be refunded 50–78% of the costs of institutions if they could meet a series of requirements, including providing a certain amount of space per resident, causing a steady decline in the number of residents per institution. In 1967, there were 194,000 people in institutions; by 1998, it had been reduced to 52,801 (Braddock and Parish 2001). The guidelines required institutions to become more homelike, but they still maintained a medical model with residents required to utilize in-house doctors and treatment plans (Sullivan 2007).

The further erosion of institutions came in 1981 with the Omnibus Budget Reconciliation Act. Wolf Wolfensberger had shifted the focus of the normalization movement from the individual

to the organizations that shaped their lives (Mesibov 1976). He made it clear that a typical life could never be achieved in an institution and home-oriented alternatives needed to be created. This act empowered Medicaid to reimburse home and community-based services, including case management and new models of residential programs (Laudicina and Burwell 1988). This opened the door to group homes, independent living, and supervised living programs, as well as day programs and vocational support. Programs were now able to offer a range of services, addressing the needs of all individuals on the autism spectrum.

Current Knowledge

In 1982, a survey of 93 adults with autism found that 16% lived in residential programs; that number rose to 53% by 1991 (Howlin 2000). This trend reveals a rising demand for residential services that shows no signs of slowing down. The developmental disabilities community has responded to this need by creating a continuum of residential models designed to address the varied needs of all individuals on the autism spectrum. These programs range from optional services designed to provide unobtrusive support allowing individuals to live a typical life to residences with 24-h staff and a team of professionals implementing individualized treatment plans to address severe behaviors and medical problems. Each model recognizes autistic individuals' desire to be a part of the community, have control of their environment, and maintain the full extent of independence that they are able to achieve. From the programs that require the highest independence level to the lowest, this chapter will review independent living, supported living, group homes, and ICF-MR facilities.

Independent Living

Even high-functioning adults can have trouble living independently. In a literature review by Howlin and Goode, they found that out of 66 adults, only 11 lived without any supportive services (Volkmar 2005), and the British

National Autistic Society noted that only 3% live without any professional support. Independent living programs are designed to provide their clients with the support to live independently in their own residence. They believe that all people can benefit from support no matter what their level of functioning. The average client is independent enough to lead a typical life but invests in services to improve their lifestyle and provide support when necessary. To be in the program, they must be able to determine their own needs and goals. All of the services provided are directed by the consumer and designed to fit individual needs.

Many programs begin by helping their clients find a residence that fits their economic restrictions and select appropriate roommates if desired. Once in their own home, they are offered classes in self-care, creating a nutritional diet, home care, budgeting, and how to navigate social and work environments. They offer different levels of involvement in their clients' lives, from visiting offices and meeting with family to private instruction. A primary goal of these programs is to build a community network of those in similar situations. They have community events to encourage social interaction and involvement.

Income, independence, and social ability are major obstacles preventing individuals from enrolling in this type of program (Leblanc et al. 2008). Acquiring and holding a high-paying job in order to afford your own residence and support services is especially difficult for adults with ASD. They must have basic household and self-care skills to maintain a residence and a healthy lifestyle. The services encourage them to take certain classes, but the client needs the self-awareness to choose the curricula that is most beneficial to them. The client also requires a certain level of social interaction in order to obtain a house or an apartment, enroll in services, and be part of a neighborhood.

The British National Autistic Society utilizes the independent living program model in several areas throughout the United Kingdom. They provide individualized levels of service, ranging from 1 h a week to daily meetings. At the high-functioning end of their service, they help

individuals develop nutritious diets and budgets their income. A major goal is to take part in community events and social activities, such as horseback riding, bowling, or attending the cinema.

The independent living model is emulated in colleges around the country. Students with autism attending school meet with advisers twice a week to discuss roommate relations, academic strategies, time management, home and self-care, and the social aspects of college life. These programs focus on the unique challenges autistic students face in the academic and social aspects of university life.

The Achieving in Higher Education with Autism/Developmental Disabilities (AHEADD) program was developed by Carnegie Mellon and now offers its services to schools across the USA. The program is designed to facilitate independent living along with academic support for college students. Its services help their clients develop advocacy skills for their own academic success by educating them on how to manage classes, professors, and workload. They are taught how to live independently, navigate campus, and utilize all of the services available to them as a student with a disability. Along with academic support, they are paired with a peer who meets with them socially once a week and mentors them on the social aspects of college life. They also provide a series of social events, such as picnics, parties, athletic groups, and bowling, to create a community within the program.

Supported Living

Many adults with ASD prefer to live at home or in their own residence but require increased involvement and supervision from professionals. Supported living is designed to allow individuals who are not a danger to themselves and are able to make responsible health and life choices but do not have the ability to navigate life without significant support. These programs allow individuals the freedom to choose where they want to live and have input into their daily schedules. They target autistic adults who face communication, social, or sensory/motor challenges and are not ready for the vast amount of choices required in

an independent living program but would be too limited in a group home setting.

Clients of supported living programs are visited each day by direct-care workers, who provide them with individualized support. This can range from providing daily schedules and vocational support to aid and instruction in self- and home care. Programs include 24-h crisis care and overnight support when needed. The benefit of this level of residential program is that it provides individuals with the support to avoid inpatient care. They are now able to determine where they live and work and have the freedom to engage in the community whenever they would like.

An excellent example of supported living is the California-based Jay Nolan Community Services. Jay Nolan was originally established in 1975 as a series of group homes serving the autistic community in Los Angeles. In 1992, they found the group home model to be too limiting and decided to shift their focus toward greater community integration. They dissolved their group homes and built a supported living program to replace them. They believe that individuals with developmental disabilities can maintain a full life within the community if they are provided with the right support and assistance.

Their organization provides two main services that are matched together: personalized day support/supported employment and supported living. The personalized day program views involvement with an organization, whether through employment or volunteering, as a critical part of integrating with the community and crucial to reaching independence. Clients are individually matched with an organization that they are involved with each day. If their disability makes a full day of work too difficult, the rest of their day is filled with meaningful community-oriented activities. The supported living program provides assistance and support to fulfill their residential needs. Each client builds a "circle of support" that includes representatives from Jay Nolan, family, friends, health-care professionals, and the client themselves. The circle of support determines which services the individual will receive and the direction of their curriculum and their goals for the coming year.

Group Homes

Group homes exist for individuals that require 24-h direct-care supervision because of physical disabilities or behavior problems that cannot be adequately handled by the family. Each group home is owned and run by an organization that makes all of the staffing selections and residential decisions. The staff provide constant care, supervision, and guidance with everyday decisions. Most have a nurse on staff, immediate response crisis care, and staff trained to handle emergency situations and behavior problems.

Since the deinstitutionalization movement, group homes have been striving to separate themselves from the asylum image that they evoke in many people. The new model for group homes is to be as much like a family home as possible. They reach for a homelike aesthetic with lighting, house design, furniture, and personalized bedrooms. The residents participate in maintaining the household to the best of their ability through chores and cooking. The number of residents per house has fallen from an average of 20 to between 2 and 10 individuals, and many are residences built for families rather than for institutions (Sullivan 2007).

Group homes have moved on from the “custodial care” model of the past to focus more on the resident’s participation in the home, community integration, and self-determination. The organization that runs the group home still decides daily activities, when to visit the community, and the curriculum goals for each individual, but they have built in a system for input from the resident and their families. The curriculum is designed to develop self-care skills and social activities within the household. There are scheduled community outings, and residents are matched with daily work programs.

The major criticism of group homes is that residents and their family have little input into decisions (Gerhardt 2009). The amount of say each resident has in curriculum and programming depends on the group home they are in and the level of their disability. Residents do not have input into large decisions, such as staffing, housemate selection, or house and grounds expansion and construction. The

company that owns the home must balance its resident’s needs with their own financial goals and the needs of the greater disabled community.

Group homes also suffer from exceptionally high staff turnover. There is an average 50% yearly resignation rate with an average of 10–11% of positions left open per home (Gerhardt and Lainer 2010). Working in a group home is both physically and emotionally taxing, requiring the staff to build positive relationships with the residents, handle severe behavior problems, and understand the underlying disability with little financial recompense or training. Direct-care staff are not required to have a degree, special training, or any experience in the field. Staff with degrees quickly move on to higher-paying jobs. Due to the turnover, most homes do not have their target number of staff, which increases the client-to-staff ratio and the workload of each staff. In most cases, new staff are needed to immediately fill the turnover gap resulting in only basic training, leaving a gap in their understanding of the disability (Van Bourgondien and Schopler 1990). Despite their responsibilities, direct-care workers are given low pay, inadequate benefits, and little professional status leading to no direct career path.

Aside from increasing staff stress and workload, the turnover rate can take a toll on the residents themselves. The social and communication deficits experienced by individuals with autism combined with intense desire for sameness and routine can make it difficult to integrate new people into their lives. Each new staff member will teach curriculum in divergent ways or utilize different communication techniques, especially if they have only been briefly trained, which can cause loss of a resident’s cognitive and social gains. A stable workforce creates continuity in the clients’ care and has been shown to increase the resident’s social interactions (Van Bourgondien and Schopler 1990). To combat loss of employees, organizations employ different strategies ranging from creating opportunities for advancement within the organization to having their staff live in the group home itself in a room separate from the residents.

The family teaching model of group homes is designed to maximize staff stability for its residents. A married or committed couple lives rent-free while providing direct-care services to two to four young adults with autism (Gerhardt 2009). They are encouraged to make the residence as much their home as possible and build a family-like rapport with the residents. The Princeton Child Development Institute's Family Focus homes have found success in this model, designing their curricula to include family-style activities and using the family unit as a stepping stone to interact with the community.

ICF-MR Facilities

ICF-MR facilities are designed to address the needs of individuals who have both autism and intellectual disabilities. The majority of residents also have major health problems or comorbid diagnoses, ranging from seizures, mental illness, visual or hearing loss, ambulatory disabilities, or behavior problems (Gerhardt 2009). These facilities follow the same design as a group home with 4–10 individuals per residence and curricula focused on self-care, communication, and social development but are better equipped for severe behavior and health problems. They feature an on-site nurse, advanced staff training in behavior and health problems, and 24-h emergency critical care. They typically maintain a larger number of staff, reducing the client-to-staff ratio and turnover.

The focus on resident's health care and mental illness needs places ICF-MR homes in the same category as hospitals, institutions, and treatment centers (Wall 1990). This status gives them access to federal Medicaid monies, which reduces the cost to individuals and their family and gives them a wider field of treatment options. They are subject to the same restrictions and regulations as medical facilities, requiring them to meet many of the same documentation, bureaucratic, and policy standards as hospitals and large institutions. It is a major challenge to meet these standards while maintaining a homelike, comfortable, family atmosphere.

The residents of ICF-MR facilities require a variety of medical professionals to meet their

needs. The facility maintains a close relationship with the physicians and mental health care professionals in the area but gives their residents the freedom to decide who they go to for care. A key to successful treatment is that the facility builds a consensus within a resident's medical team and constructs a coordinated and consistent plan. The facility then meets with the resident or their guardian and builds their needs and goals into the plan.

If an ICF-MR facility cannot meet the medical or behavioral needs of a resident, they must enter an intensive inpatient behavior care unit. These are short-term residences, not exceeding 6 months, which provide their clients intensive therapy to deal with extreme aggressive behaviors targeted at both themselves and others (Gerhardt 2009). The individuals entering these programs have experienced an increase in behaviors that would result in needing to find a more restrictive living situation or in a medical state where they need to detox off medication that is managing severe behaviors. The professionals utilize behavioral interventions, cognitive therapies, skill trainings, and a process of medication assessment and trials to teach the patient to regulate their own behaviors and maintain their own mental health.

One of the most successful intensive inpatient behavior care units is the Kennedy Krieger Institute's Neurobehavioral Unit. They have provided care to patients from across America and from many countries around the world. Their therapy is derived from applied behavior analysis and focus on assessments of behavior, the effects of reinforcement, and parent-child interaction. A key component to their program is training the care givers and people who work with the individual in unified techniques and strategies to handle behaviors and maintain their client's stability. This process begins months before the client is released, ensuring that they enter a supportive and therapeutic environment that will maintain the gains they made within the institute.

A concern of both group homes and ICF-MR facilities is finding a place for their residents in the workforce. Employment is the foundation of persons placed in the community and provides important opportunities for social interaction and building communication. Kessissoglou and

Farrell showed supported employees gained independence, self-esteem, and became more responsible through employment (Mawhood and Howlin 1999). Many programs provide their clients with a separate day program in a sheltered workshop or supported employment in the community. Farmstead programs merge residential and vocational programs by providing work opportunities on campus.

Farmsteads are group homes or ICF-MR facilities that are built on land that is designed to maintain agriculture and raise farm animals. The residents maintain the grounds, raise animals, grow the crops, and sell what they make alongside direct-care workers who are trained in farm work. This type of work is immediately meaningful, has a visible effect on the surrounding environment, and encompasses a large spectrum of abilities. Tasks that range from harvesting crops and building retention walls to separating seeds and feeding animals provide work that meets the physical and mental needs of each resident. This model gives work opportunities to individuals with severe disabilities or extreme behavior problems who may not be able to work safely in the community.

Division TEACCH's Carolina Living and Learning Center (CLLC) is an ICF-MR facility that resides on 79 acres of land, has 15 residents in two houses, and provides supported employment to 2 individuals from group homes in the area. The center is an exemplary example of the effects of the TEACCH method. Individualized structure has been built into every aspect of resident's life from work systems to social interaction. Residents transitioning into the CLLC show marked increases in communication, social skills, independence, and a reduction of behavior problems (Van Bourgondien et al. 2003).

The vocational program revolves around both residents and the direct-care workers maintaining several large vegetable gardens. By taking part in every step in the life cycle of vegetables, from planting to cultivating, and finally eating what they have grown, residents take pride in their part of the process. They also engage in a variety of nonagricultural work such as making soap and potpourri to cooking pesto

and salsa. Periodically during the year, the residents sell what they have made at local events. Each day after work or on weekends, they go on community outings, either to attend local events, go shopping, or for exercise. Integrated into both the vocational program and their evenings is a curriculum of communication, self-care, and household skills that are designed specifically for each individual.

The continuum of residential models focuses on the needs of the current autistic community. Adults with autism have a wide variety of options that support their independence, foster their growth, and build a place for them in the world. As the population ages, a new complication looms on the horizon: retirement. The level of medical care and the type of activities and outings in the current models are not designed to meet the needs of the elderly, and the typical retirement community does not have staff trained to aid autistic individuals. Residential Services Inc. is the first organization to address this problem head on. They have built a facility that is designed to meet the needs of elderly individuals at all levels of the autism spectrum.

Residential Services Inc. is a North Carolina-based organization who developed their autism program with support from the TEACCH program. They provide the developmentally disabled community with all levels of services from residential programs to independent living support. They maintain 16 residences that are divided into group homes, ICF-MR facilities, and homes that are rented to autistic individuals as part of a supported living arrangement. Their Community Vocational and Learning Services program provides supported employment to residents in need of a job and their Life Options program is an enhanced day program for individuals requiring ICF-MR level of care.

The Spring Glen Retirement Community combines their experience with all levels of the autism spectrum into care for the elderly. The facility is made up of 15 units that are designed to match each model on the residential continuum. Each unit is designed to meet the varied needs of individuals and maximize their independence. Some units are built for highly

independent individuals and include a kitchenette and in some cases are part of a larger two-bedroom suite. Other units are designed to meet the standards of an ICF-MR facility and offer increased supervision, medical care, and support. They share communal activity rooms, spaces for therapy, dining rooms, gardens, walking trails, and a pond. The communal areas are often used for large group activities to foster friendships and build an air of community.

As the number of individuals with autism seeking residential care rises, residential services continue to grow and adapt services to the needs of the autistic community. Adults with autism can choose a model that will maximize their independence, build a place in the community, and find the best path to personal growth.

Future Directions

The United States Census Bureau estimates that there will be over 1,495,264 adults with autism in America by 2016. The majority of these individuals will require some form of residential services. In 1998, the wait list for residential placement in the USA had reached 87,000 and has only risen since (Gerhardt and Lainer 2010). The current system is not prepared to meet the needs of the autistic population today, much less the growing numbers of individuals aging out of student services. In the coming years, residential services will need to not only expand the number of individuals it serves but also the types of services it provides.

For the first time in United States history, autism is high on the national agenda. The rise in the population of individuals with autism has been recognized by the news media which has caused a wave of awareness in society. President Obama has not only made autism a priority but designated his first appointee with autism to the National Council on Disability. This prompted talk of new legislation that will create and fund new residential programs across the country.

With the growing population, the options available to adults with autism must expand to meet new and different needs. If we continue

only down the route of community integration and standardizing residential models, it may lead us to an ineffectual system (Schopler and Hennike 1990). There is already a trend in residential services toward innovation and experimentation, as seen in the family teaching model, farmstead programs, clustered apartments, and many others. The flexibility of the models will include both services offering community integration and others that would not. Some modern ICF-MR facilities follow the institution model to meet the needs of individuals with severe disabilities. The Murdoch Developmental Center houses 550 individuals with autism of all ages, who suffer from extreme behavioral challenges. In the coming years, we will see an expansion of residential programs to house the rising population and meet their changing needs.

See Also

- ▶ [Benhaven Residential Services](#)
- ▶ [Community Services](#)
- ▶ [Community-Integrated Residential Services for Adults with Autism](#)
- ▶ [Daily Living Skills](#)
- ▶ [Independent Living](#)
- ▶ [Normalization](#)
- ▶ [Self-Care](#)
- ▶ [TEACCH Transition Assessment Profile \(TTAP\)](#)

References and Reading

- Braddock, D., & Parish, S. (2001). An institutional history of disability. In G. Albrecht, K. Seelman, & M. Bury (Eds.), *Handbook of disability studies* (pp. 11–69). Thousand Oaks: Sage.
- Gerhardt, P. (2009, January). *The current state of services for adults with autism*. Paper presented at the Advancing Futures for Adults with Autism: Think Tank, New York.
- Gerhardt, P., & Lainer, I. (2010). Addressing the needs of adolescents and adults with autism: A crisis on the horizon. *Journal of Contemporary Psychotherapy*, 41, 37–45.
- Howlin, P. (2000). Outcome in adult life for more able individuals with autism or Asperger syndrome. *Autism*, 4, 63–83.

- Laudicina, S., & Burwell, B. (1988). A profile of medicaid home and community-based care waivers, 1985: Findings of a national survey. *Journal of Health Politics, Policy and Law*, 13(3), 525–546.
- LeBlanc, L., Riley, A., & Goldsmith, T. (2008). *Autism spectrum disorders: A lifespan perspective*. Burlington: Elsevier.
- Mawhood, L., & Howlin, P. (1999). The outcome of a supported employment scheme for high-functioning adults with autism or Asperger syndrome. *Autism*, 3, 229–254.
- Mesibov, G. (1976). Implications of the normalization principle for psychotic children. *Journal of Autism and Developmental Disorders*, 6(4), 360–365.
- Schopler, E., & Hennike, J. M. (1990). Past and present trends in residential treatment. *Journal of Autism and Developmental Disorders*, 20(3), 291–298.
- Sullivan, R. (2007, May). *Autism Society of America Position Paper on The National Crisis in Adult Services for Individuals With Autism*. Adopted by the Autism Society of America, Bethesda.
- Van Bourgondien, M. E., & Elgar, S. (1990). The relationship between existing residential services and the needs of autistic adults. *Journal of Autism and Developmental Disorders*, 20(3), 299–308.
- Van Bourgondien, M. E., & Schopler, E. (1990). Critical issues in the residential care of people with autism. *Journal of Autism and Developmental Disorders*, 20(3), 391–399.
- Van Bourgondien, M. E., Reichle, N., & Schopler, E. (2003). Effects of a model treatment approach on adults with autism. *Journal of Autism and Developmental Disorders*, 33(2), 131–140.
- Volkmar, F. R. (2005). Living arrangements. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, 3rd ed., pp. 297–298). Hoboken: Wiley.
- Wall, A. J. (1990). Group homes in North Carolina for children and adults with autism. *Journal of Autism and Developmental Disorders*, 20(3), 353–366.

Resource List

- Achieving in Higher Education with Autism/Developmental Disabilities (AHEADDD). Retrieved from <http://www.aheaddd.org>
- Carolina Living and Learning Center. Retrieved from <http://teacch.com/programs-and-services/Carolina-living-learning-center>
- Jay Nolan Community Services. Retrieved from <http://www.jaynolan.org/>
- Kennedy Krieger Institutes' Neurobehavioral Unit. Retrieved from http://www.kennedykrieger.org/kki_cp.jsp?pid=1573
- Murdoch Developmental Center. Retrieved from <http://www.murdochcenter.org/>
- Residential Services Incorporated. Retrieved from <http://www.rsi-nc.org/index.html>
- The National Autistic Society Supported Living Services. Retrieved from [http://www.autism.org.uk/en-gb/our-](http://www.autism.org.uk/en-gb/our-services/residential-community-and-social-support/supported-living.aspx)

[services/residential-community-and-social-support/supported-living.aspx](http://www.autism.org.uk/en-gb/our-services/residential-community-and-social-support/supported-living.aspx)

The Princeton Child Development Institute Family Focus Group Homes. Retrieved from <http://www.pcdi.org/programs/familyFocus.asp>

Local Educational Agency

► Local Educational Authority

Local Educational Authority

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Synonyms

Local educational agency; Nexus

Definition

The Local Education Authority (agency) is the public entity that is responsible financially and for planning the educational program for an individual receiving special education services. The LEA is also responsible for monitoring and reporting data to state and federal agencies. Frequently, the LEA is the district in which the individual receiving services lives, but there can be other instances in which a public entity could be determined to be the LEA.

References and Reading

- Education in Texan. www.ritter.tea.state.tx.us/specialeducation. LEA Determination.
- IDEA Partnerships. www.fape.org
- Nation Dissemination Center for Children with Disabilities. www.nichcy.org
- Office of Special Education Programs. www.ed.gov/osep/local_education_agency
- Turnbull, A., Wehmeyer, M. L., & Turnbull, R. (2009). *Exceptional lives: Special education in today's schools*. Upper Saddle River: Prentice-Hall.

Vaughn, S., Bos, C., & Schumm, J. S. (2010). *Teaching exceptional, diverse, and at-risk students* (pp. 2–7). New York: Prentice-Hall.

Wisconsin Department of Public Instruction. www.dpi.state.wi.us/specialeducation. Duties of the LEA.

Locus Ceruleus

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Definition

The locus ceruleus is a small brain structure located in a part of the brainstem called the Pons. This structure is the site in which an important excitatory neurotransmitter, norepinephrine, is manufactured and sent to other parts of the brain. Norepinephrine plays a role in many brain functions including homeostasis, stress, fear, mood, and sleep. The connections of the locus ceruleus are vast but include part of the amygdala, cingulate cortex, hypothalamus, medial frontal cortex, cerebellum, and spinal cord. The locus ceruleus is not directly implicated in autism and there is no evidence of pathology in the structure (Martchek et al. 2006), though there have been speculations of such a link (Mehler and Purpura 2009).

See Also

► [Norepinephrine](#)

References and Reading

- Martchek, M., Thevarkunnel, S., Bauman, M., Blatt, G., & Kemper, T. (2006). Lack of evidence of neuropathology in the locus coeruleus in autism. *Acta Neuropathologica*, 111(5), 497–499.
- Mehler, M. F., & Purpura, D. P. (2009). Autism, fever, epigenetics and the locus coeruleus. *Brain Research Reviews*, 59(2), 388–392.

Lofexidine

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Definition

Lofexidine is a medication of the alpha-2 agonist class with a molecular weight of 259.13 g/mol and a chemical formula of C₁₁H₁₂Cl₂N₂O. It is a structural analog of the alpha-2 agonist clonidine (see ► [“Clonidine”](#)). It was originally approved in Germany to treat hypertension but was withdrawn due to a lack of clinical efficacy (Gish et al. 2010). Lofexidine can be used as a short-acting antihypertensive, but it is primarily used to assist with opioid detoxification. It is approved in the UK for this purpose under the trade name BritLofex. While extended-release formulations of the alpha-2 agonists clonidine and guanfacine (see ► [“Guanfacine”](#)) are approved by the US Food and Drug Administration (FDA) to treat attention deficit-hyperactivity disorder (ADHD), lofexidine has not been studied for this purpose (see ► [“Attention Deficit/Hyperactivity Disorder”](#)). The FDA has not currently approved lofexidine for clinical use in the United States.

Like other alpha-2 agonists, lofexidine works by binding to alpha-2 type adrenoceptors in the central nervous system (see ► [“Noradrenergic System”](#)). This action, specifically in the locus coeruleus, helps to counteract the autonomic symptoms of opioid withdrawal. These alpha-2 receptors respond to high levels of catecholamines (i.e., epinephrine and norepinephrine) in the blood and exert negative feedback on descending sympathetic nerves that project to the adrenal medulla and stimulate catecholamine production. By activating the alpha-2 receptors, lofexidine simulates higher blood catecholamine levels, thereby lessening sympathetic input to the

adrenal medulla and reducing catecholamine production. Catecholamine levels in the blood subsequently drop, reducing both heart rate and blood pressure. Furthermore, increased activation of alpha-2 adrenoceptors in the prefrontal cortex is thought to mediate the effects of alpha-2 agonist medications on ADHD symptoms.

Lofexidine shows a high affinity for the alpha-2A adrenoceptor subtype, which is thought to be responsible for its reduced antihypertensive action relative to clonidine. Hypotension may still occur in some subjects, particularly those who are elderly or taking additional antihypertensive medications. In addition, transient increases in blood pressure have been noted following abrupt discontinuation of lofexidine.

Adverse effects of lofexidine are similar to other alpha-2 agonists and include sedation, insomnia, dry mucous membranes, dizziness, weakness, and hypotension. When compared with clonidine, lofexidine is reported to have a lower incidence of adverse effects, particularly hypotension and sedation.

One small trial has assessed the effect of lofexidine in children with autism, focusing on its reduction of concomitant ADHD symptoms. Using a double-blind placebo-controlled cross-over design, Niederhofer et al. (2002) administered lofexidine to a group of children with autistic disorder who also presented with significant symptoms of distractibility, hyperactivity, and impulsivity. The study reported reductions in both parent and teacher symptom ratings, primarily in the domain of hyperactivity. However, clinician-rated symptoms did not differ between lofexidine and placebo. No sedation was reported in the trial, but several children experienced hypotension, requiring reduced dosing of lofexidine. Although no further studies have replicated lofexidine's effects on ADHD symptoms in autism, a larger placebo-controlled trial of extended-release guanfacine also reported significant reductions in hyperactivity scores (Scahill et al. 2015). While further research is needed to explore the clinical utility of alpha-2 agonists in autism, it is unclear whether lofexidine offers any benefits over similar agents already approved by the FDA.

See Also

- Attention Deficit/Hyperactivity Disorder
- Clonidine
- Guanfacine
- Noradrenergic System

References and Reading

- Arnsten, A. F., Scahill, L., & Findling, R. L. (2007). Alpha-2 adrenergic receptor agonists for the treatment of attention-deficit/hyperactivity disorder: emerging concepts from new data. *Journal of Child and Adolescent Psychopharmacology*, 17(4), 393–406.
- Gish, E. C., Miller, J. L., Honey, B. L., & Johnson, P. N. (2010). Lofexidine, an α_2 -receptor agonist for opioid detoxification. *Annals of Pharmacotherapy*, 44(2), 343–351.
- National Center for Biotechnology Information. PubChem Compound Database; CID=30668, <https://pubchem.ncbi.nlm.nih.gov/compound/30668>. Accessed 29 Dec 2016.
- Niederhofer, H., Staffen, W., & Mair, A. (2002). Lofexidine in hyperactive and impulsive children with autistic disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 41(12), 1396–1397.
- Scahill, L., McCracken, J. T., King, B. H., Rockhill, C., Shah, B., Politte, L., et al. (2015). Extended-release guanfacine for hyperactivity in children with autism spectrum disorder. *The American Journal of Psychiatry*, 172(12), 1197–1206.

Longitudinal

- Longitudinal Research in Autism

Longitudinal Research in Autism

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Synonyms

Cohort studies; Longitudinal; Natural history;
Outcome; Prospective

Definition

Longitudinal studies in autism are important for several reasons: First, they help to inform families and clinicians about the range of outcomes that may be expected in relation to a diagnosis such as autism. Additional reasons include opportunities to monitor change in symptom profile and comorbidities such as medical problems, as well as the ability to follow the acquisition of activities of daily living such as friendship, vocational skills and self-help. If predictor or mediator variables are identified that are potentially modifiable these may inform the development of new intervention strategies that could be tested in randomized control trials.

Longitudinal studies comprise a set of study designs that have the following features in common. The designs are (1) observational, i.e., they involve studying individuals that have been naturally selected to a particular group or exposure as compared to random assignment by researchers to a particular group as in experimental designs; (2) the individuals are sampled by exposure (or diagnosis), not by outcome as in case-control studies; and (3) they are generally prospective, i.e., the individuals of interest are followed chronologically and reassessed at one or more later time points. Such studies are very useful for a number of reasons but commonly used to examine predictors and outcomes. Predictors are early factors or characteristics of the individual or his/her environment that are associated with variation in later-occurring outcomes. Outcomes may be consequences of a diagnosis or of early predictors of interest. Longitudinal studies can also be used to investigate developmental trajectories or the rate of change in a domain over time. Mediators and moderators of outcome can also be studied as a special class of predictor variables. Mediators are variables that might account for change over time, and moderators are variables that modify the association between predictor and outcome.

Prospective cohort studies assemble a group of similar individuals (a “cohort”) at one time point. These individuals are then followed up over one or more time points to determine whether and how variation in certain baseline factors relates to

variation in outcomes of interest. If the cohort is followed over three or more time points (including baseline), trajectories or pathways of particular symptoms, abilities, or characteristics may be plotted to describe the rate and shape of change over time. Such designs also minimize measurement error due to recall bias. Drawbacks of these studies include greater expense and length of time to complete data collection.

See Also

- [Asperger Syndrome Follow-Up Studies](#)
- [Course of Development](#)
- [Factors Affecting Outcomes](#)
- [Outcome Studies](#)

References and Reading

- Robinson, K., Schmidt, T., & Teti, D. M. (2005). Issues in the use of longitudinal and cross-sectional designs. In D. M. Teti (Ed.), *Handbook of research methods in developmental science* (pp. 3–20). Oxford, UK: Blackwell Publishing.
- Sackett, D. L., Straus, S., & Richardson, W. S. (2000). *Evidence-based medicine: How to practice and teach EBM*. Edinburgh: Churchill Livingstone.
- Singer, J. D., & Willett, J. B. (2003). *Applied longitudinal data analysis; Modelling change and event occurrence*. New York: Oxford University Press.

Loose Associations

- [Flight of Ideas](#)

Lorazepam

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Synonyms

[Ativan](#); [Benzodiazepine](#)

Definition

A benzodiazepine medication used to relieve anxiety. Lorazepam is also used to treat irritable bowel syndrome, epilepsy, insomnia, nausea/vomiting from cancer treatment, and to control agitation caused by alcohol withdrawal. Additionally, it is used for surgery to interfere with memory formation and to sedate those who are being mechanically ventilated. Lorazepam works by slowing activity in the brain to allow for relaxation. It acts as a sedative, hypnotic, anticonvulsant, and has muscle relaxant properties. Lorazepam primarily works by modifying the amount and action of gamma-aminobutyric acid (GABA), specifically enhancing the effects of the GABA neurotransmitter at the GABA receptors by increasing the frequency of opening of the chloride ion channels of the GABA receptors.

Lorazepam was developed by D.J. Richards, the President of Research of Wyeth Pharmaceuticals, in 1977. This benzodiazepine comes as a tablet or concentrate to take by mouth and is usually taken two or three times a day with or without food. It can also be given as an injection into a muscle or vein, with the onset of effects occurring between one and thirty minutes and lasting for up to a day. If lorazepam is taken intravenously, the individual should be closely monitored. Lorazepam also comes in the forms of a skin patch, an oral solution, and a sublingual tablet. Lorazepam is high-potency and fast-acting, with a biological half-life of only 10–20 hours. The brain has extra benzodiazepine drug receptor capacity, so larger doses of lorazepam leads to stronger and longer-lasting effects. Since it is fast-acting, lorazepam works best to treat fast onset panic anxiety.

Common side effects of lorazepam include sedation, weakness, sleepiness, unsteadiness, dizziness, confusion, low blood pressure, decreased effort to breathe, increased risk of suicide for those who are depressed, and inhibition of the formation of new memories. Physical and/or psychological dependence may occur. Larger doses of lorazepam may be required to acquire the same effect if being used long-term. Benzodiazepine withdrawal syndrome may occur if lorazepam is stopped suddenly after long-term use. Withdrawal symptoms occur in one-third of individuals who have been using benzodiazepines for

longer than 4 weeks. Relatedly, it may be necessary to increase doses to sustain effectiveness if lorazepam treatment continues for longer than 4–6 months. Lorazepam is used to treat elderly individuals with either anxiety or other related conditions. Long-term cognitive effects, including reduced recall and psychomotor slowing, in addition to the absence of therapeutic benefit, emphasizes the cognitive toxicity of long-term use of lorazepam in this population.

GABA receptors have been found to be reduced in the parietal cortex, frontal cortex, and cerebellum, which are three brain areas that have been associated with the development of autism. This finding suggests widespread GABA dysfunction in the brains of individuals with autism and the relation to the use of lorazepam to enhance the effects of the GABA neurotransmitters. The safety of lorazepam in children with autism has not been proven. There are no currently approved medications to treat the core symptoms of autism. Benzodiazepines are being used to treat the epilepsy and anxiety symptoms in children with autism. Lorazepam has been found to moderately improve catatonic symptoms, or the inability to move normally, in multiple cases of individuals with autism. It is considered a safe and effective medical treatment for catatonia in ASD. Lorazepam has also been studied as a potential medication for aggression in autism. It was not found to have a significant effect on aggression and may have an inhibiting effect on the outcome of decreased aggression due to exercise.

See Also

- [Antiepileptic Drugs \(AEDs\)](#)
- [Anxiolytics](#)
- [Catatonia](#)
- [Sedative Hypnotic Drugs](#)

References and Reading

- Allison, D. B., Basile, V. C., & MacDonald, R. B. (1991). Brief report: Comparative effects of antecedent exercise and lorazepam on the aggressive behavior of an autistic man. *Journal of Autism and Developmental Disorders*, 21(1), 89–94.

- Consoli, A., Gheorghiev, C., Jutard, C., Bodeau, N., Kloeckner, A., Pitron, V., . . . & Bonnot, O. (2010). Lorazepam, fluoxetine and packing therapy in an adolescent with pervasive developmental disorder and catatonia. *Journal of Physiology, Paris*, 104(6), 309–314.
- Dhossche, D. M. (2014). Decalogue of catatonia in autism spectrum disorders. *Frontiers in Psychiatry*, 5, 157.
- Fatemi, S. H., Reutiman, T. J., Folsom, T. D., & Thuras, P. D. (2009). GABA A receptor down-regulation in brains of subjects with autism. *Journal of Autism and Developmental Disorders*, 39(2), 223.
- Pomara, N., Lee, S. H., Bruno, D., Silber, T., Greenblatt, D. J., Petkova, E., & Sidtis, J. J. (2015). Adverse performance effects of acute lorazepam administration in elderly long-term users: Pharmacokinetic and clinical predictors. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 56, 129–135.

Lord, Cathy

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Name and Degrees

Dr. Catherine Lord

Ph.D. in Psychology and Social Relations, Harvard University

Major Appointments (Institution, Location, Dates)

Dr. Lord is currently Director of the Center for Autism and the Developing Brain (CADB) at Weill Cornell Medical College and an attending psychologist at New York Presbyterian Hospital. She also currently holds the following appointments:

Professor of Psychology in Psychiatry, Psychiatry, Weill Cornell Medical College 2012-present.

Professor of Psychology in Pediatrics, Pediatrics, Weill Cornell Medical College 2012-present

Previous positions include:

Visiting professor, NYU Child Study Center and NYU School of Medicine Previously, 2008–2009

Uri Bronfenbrenner Distinguished Professor of Psychology, Psychiatry and Pediatrics, University of Michigan, Director of the University of Michigan Autism and Communication Disorders Center, 2001–2010

Professor of Psychiatry, University of Chicago, 1993–2001

Clinical Professor of Psychiatry, University of North Carolina, Chapel Hill, 1990–1993

Assistant Professor, Associate Professor and then Professor of Pediatrics, University of Alberta School of Medicine, 1982–1990

Assistant Professor at the Institute of Child Development, University of Minnesota, 1977–1981

Major Honors and Awards

Dr. Lord has received over 35 awards and honors from local, national and international bodies, too many to identify individually here, but a few noteworthy honors and awards include:

National Science Foundation Graduate Fellow, 1971–1975

National Institute of Mental Health (NIMH) Research Scientist Award, 1995–2000

Fellow, Society of Clinical Psychology, American Psychological Association, 1998

Chair, Committee on the Effectiveness of Early Intervention in Autism, National Academy of Science/National Research Council 1998–2001

Fellow, Society for Child and Adolescent Clinical Psychology, 2002

Irving B. Harris Early Childhood Award, 2004

Fellow, Association for Psychological Science, 2010

American Psychological Association (APA) Award for Distinguished Contribution to Applied Research, 2010

Award for Distinguished Scientific Contributions to Clinical Psychology, Society of Clinical Psychology, 2011

American Psychological Association (APA) Society of Clinical Child and Adolescent Psychology Lifetime Achievement Award, 2013

Member, National Academy of Medicine, 2014

Landmark Clinical, Scientific, and Professional Contributions

Catherine Lord has contributed significantly to the field of autism research in many domains, including her seminal longitudinal studies on early diagnosis of the disorder (Anderson et al. 2014; Lord et al. 2006), her contributions to understanding the nosology and basic characterization of core deficits of ASD, and her involvement in developing and testing treatments (Lord and Jones 2013; Lord et al. 2005). While these contributions have formed the basis for much of our understanding of the behavioral features of ASD, arguably, her most significant contribution lies in her work on the development and validation of standardized diagnostic instruments for individuals with ASD of different ages and abilities. Through her training and collaborations with Sir Michael Rutter, she created the Autism Diagnostic Interview (Le Couteur et al. 1989), and then the Autism Diagnostic Observation Schedule (Lord et al. 1989), two of the most widely used and well-established instruments for diagnosis and phenotypic characterization in clinical and research settings across the world. Dr. Lord collaborated in the development of the Social Communication Questionnaire (Berument et al. 1999), which is a widely used screening tool. More recently, Dr. Lord has been involved in several other measure development efforts, largely inspired by calls from the ASD research community that different types of measures are needed for different purposes. These include the Autism Screening Interview (ASI), a brief telephone interview of ASD symptoms (Bishop et al. 2016), the Brief Observation of Social Communication Change (BOSCC; Grzadzinski et al. 2016), a measure designed to capture change in social-

communication symptoms in response to treatment, and the Observation of Spontaneous Expressive Language (OSEL), a measure of spontaneous, functional language in children with ASD (Kim et al. 2014).

In addition to her numerous scientific contributions, Dr. Lord has had a major impact on clinical practice and policy in the field of ASD by contributing to various local, national and international efforts. Locally, one of the many examples is her contributions to the development of both research and treatment for TEACCH, a state-wide program in North Carolina that has served as a model for educating children with autism throughout the world. Nationally, Dr. Lord was chosen to lead or serve on many committees charged with developing and publishing guidelines for the field. One highly significant example was her role as chair of the committee on educational interventions for children with autism sponsored by the National Academy of Sciences, which resulted in the landmark publication (Lord and McGee 2001). From 2007 to 2014, she served on the DSM-5 committee that provided updated diagnostic guidelines for ASD and other neurodevelopmental disorders. She has continued to lead the field by providing up to date summaries of research and recommendations about how to implement current best practices for diagnosis and treatment of ASD within the community (Lord and Bishop 2010; Lord and Bishop 2015).

In the last 10 years, Dr. Lord has been part of larger efforts to extend ASD research beyond behavioral characterization of ASD and promote interdisciplinary cooperation between clinical and neurobiological researchers. She has helped lead several highly influential efforts designed to elucidate biological markers and genetic aspects of the disorder. For instance, she was part of a core group of scientists involved in developing the Simons Simplex Collection (SSC), a publicly available repository of cell lines and phenotypic data from probands with ASD and their family members (Fischbach and Lord 2010). Her leadership in behavioral phenotyping for this project has helped facilitate numerous genetic discoveries (e.g., Sanders et al. 2015) and has also allowed for multisite evaluation of diagnostic practices for

the disorder (Lord et al. 2012). Through these efforts, Dr. Lord has repeatedly emphasized the importance of considering the developmental aspects of the disorder, using findings to illustrate that developmental trajectories of behavioral variables may be most important in advancing understanding of the biology of ASD (Lord et al. 2015).

Short Biography

Dr. Lord is a clinical psychologist who has worked as a clinician-researcher in Canada and the USA and has collaborated and contributed to autism research worldwide. In her various positions, she has split her time between evaluating children, adolescents, and adults with ASD and engaging in major research efforts to lead the field in diagnosis and characterization of ASD across the lifespan. Dr. Lord has also advised numerous graduate students, post-doctoral fellows, and early-career researchers, many of whom now hold clinical and/or research positions at institutions in the USA and abroad. She began her career in autism as an undergraduate, where she was a student at UCLA at the time when Dr. Ivar Lovaas was working on his groundbreaking treatment paradigm for children with developmental disabilities, applied behavioral analysis. Having worked directly with children with autism under his supervision, it was at this point that she developed a passion for helping children with autism learn to talk. After graduate school in psychology at Harvard University, she interned at TEACCH, a one-of-a-kind program in North Carolina that includes services throughout the lifespan for individuals with autism, where she later returned to launch one of the longest lasting longitudinal studies of autism that continues to this day. Other positions Dr. Lord has held include her faculty appointment at the University of Alberta, where she was part of a strong multidisciplinary team of clinicians, and her appointment in child psychiatry at the University of Chicago within a collaborative and clinically skilled group of clinician researchers. During the last 15 years, Dr. Lord has founded two major clinical-research centers, the University of Michigan Autism and

Communication Disorders Center (UMACC), and the Center for Autism and the Developing Brain (CADB) at Weill Cornell Medical College. These centers have served as models for the integration of clinical practice and research, yielding data from thousands of clinically referred and research ascertained individuals with ASD and non-ASD diagnoses. In turn, these data have provided critical information about psychometric properties of numerous assessment instruments, which has informed the modification of existing tools and development of new tools. Dr. Lord has also participated in groundbreaking clinical trials for early treatment and characterization of the disorder within the context of behavioral development and ongoing phenotype-genotype efforts.

References and Reading

- Anderson, D. K., Liang, J. W., & Lord, C. (2014). Predicting young adult outcome among more and less cognitively able individuals with autism spectrum disorders. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 55(5), 485–494. <https://doi.org/10.1111/jcpp.12178>.
- Berument, S. K., Rutter, M., Lord, C., Pickles, A., & Bailey, A. (1999). Autism screening questionnaire: Diagnostic validity. *British Journal of Psychiatry*, 175, 444–451.
- Bishop, S. L., Huerta, M., Gotham, K., Alexandra Havdahl, K., Pickles, A., Duncan, A., et al. (2016). The autism symptom interview, school-age: A brief telephone interview to identify autism spectrum disorders in 5-to-12-year-old children. *Autism Research*. <https://doi.org/10.1002/aur.1645>.
- Fischbach, G. D., & Lord, C. (2010). The Simons simplex collection: A resource for identification of autism genetic risk factors. *Neuron*, 68(2), 192–195. <https://doi.org/10.1016/j.neuron.2010.5006>. S0896-6273(10)00830-5 [pii].
- Grzadzinski, R., Carr, T., Colombi, C., McGuire, K., Dufek, S., Pickles, A., & Lord, C. (2016). Measuring changes in social communication behaviors: Preliminary development of the brief observation of social communication shange (BOSCC). *Journal of Autism and Developmental Disorders*, 46(7), 2464–2479. <https://doi.org/10.1007/s10803-016-2782-9>.
- Kim, S. H., Junker, D., & Lord, C. (2014). Observation of Spontaneous Expressive Language (OSEL): A new measure for spontaneous and expressive language of children with autism spectrum disorders and other communication disorders. *Journal of Autism and Developmental Disorders*, 44(12), 3230–3244. <https://doi.org/10.1007/s10803-014-2180-0>.

- Le Couteur, A., Rutter, M., Lord, C., Rios, P., Robertson, S., Holdgrafer, M., & McLennan, J. (1989). Autism diagnostic interview: A standardized investigator-based instrument. *Journal of Autism and Developmental Disorders*, 19(3), 363–387.
- Lord, C., & Bishop, S. L. (2010). Social policy report: Autism spectrum disorders. Diagnosis, prevalence, and services for children and families. *Society for Research in Child Development: Sharing Child and Youth Development Knowledge*, 24(2), 1–27.
- Lord, C., & Bishop, S. L. (2015). Recent advances in autism research as reflected in DSM-5 criteria for autism spectrum disorder. *Annual Review of Clinical Psychology*, 11, 53–70. <https://doi.org/10.1146/annurev-clinpsy-032814-112745>.
- Lord, C., & Jones, R. M. (2013). New strategies and findings for behavioral interventions in autism spectrum disorders. *Annals of the New York Academy of Sciences*, 1304, 70–76. <https://doi.org/10.1111/nyas.12311>.
- Lord, C., & McGee, G. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Lord, C., Rutter, M., Goode, S., Heemsbergen, J., Jordan, H., Mawhood, L., & Schopler, E. (1989). Autism diagnostic observation schedule: A standardized observation of communicative and social behavior. *Journal of Autism and Developmental Disorders*, 19(2), 185–212.
- Lord, C., Wagner, A., Rogers, S., Szatmari, P., Aman, M., Charman, T., et al. (2005). Challenges in evaluating psychosocial interventions for autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 35(6), 695–708.
- Lord, C., Risi, S., DiLavore, P. S., Shulman, C., Thurm, A., & Pickles, A. (2006). Autism from 2 to 9 years of age. *Archives of General Psychiatry*, 63(6), 694–701. <https://doi.org/10.1001/archpsyc.63.6.694>.
- Lord, C., Petkova, E., Hus, V., Gan, W., Lu, F., Martin, D. M., et al. (2012). A multisite study of the clinical diagnosis of different autism spectrum disorders. *Archives of General Psychiatry*, 69(3), 306–313. <https://doi.org/10.1001/archgenpsychiatry.2011.148>.
- Lord, C., Bishop, S., & Anderson, D. (2015). Developmental trajectories as autism phenotypes. *American Journal of Medical Genetics. Part C, Seminars in Medical Genetics*, 169(2), 198–208. <https://doi.org/10.1002/ajmg.c.31440>.
- Sanders, S. J., He, X., Willsey, A. J., Ercan-Sencicek, A. G., Samocha, K. E., Cicek, A. E., et al. (2015). Insights into autism spectrum disorder genomic architecture and biology from 71 risk loci. *Neuron*, 87(6), 1215–1233. <https://doi.org/10.1016/j.neuron.2015.09.016>.

Loss of Voice

► Aphonia

Lovaas Approach

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Definition

The Lovaas approach was pioneered by O. Ivar Lovaas in the 1970s and 1980s. Lovaas himself used the term “*the UCLA Model*” to describe this treatment. Defining features of the Lovaas approach include:

Behavioral Emphasis. Autism is analyzed in terms of separate behaviors that include excesses (behaviors displayed too often or intensely) and deficits (behaviors that are not displayed often or well enough). Excesses and deficits exhibited by children with autism are addressed using principles of behavior analysis and learning psychology (see “[Treatment Procedures](#)” below).

Family Involvement. Lovaas strongly emphasized family participation because research suggested that parental involvement enabled children to make lasting improvements and carry over these improvements from professional settings, such as special education programs, clinics and hospitals, to the home environment community. To optimize treatment effects, parents are trained to become co-teachers for their child; they learn how to manage the child’s challenging behaviors and how to make the child use the skills he or she has learned from treatment in everyday life.

One-to-One Instruction. During the first 6 to 12 months of intervention, the bulk of the teaching is carried out in a one-to-one setting because it allows for highly individualized teaching and because children with autism may initially learn more readily in such a setting than in groups. The individual instruction focuses on teaching the child key communication, play, and social skills to prepare children to enter group settings.

Integration. When the treatment team determines that the child with autism is ready to enter a group setting, the team chooses a setting where the peers are as typical as possible, as children

with autism often display more appropriate behaviors when integrated with typical peers than when placed in group with other children with autism. A member of the treatment team accompanies the child to the integrated setting in order to help the child be successful (see “[Treatment Procedures](#)”).

Comprehensiveness. The curriculum addresses all three core deficits in autism (i.e., deficits in communication, social skills and social interests, and excess stereotyped and ritualistic behaviors). It also addresses other behavioral excesses and deficits that may be associated with autism such as aggression and self-injurious behavior, attention deficits, learning delays, feeding problems, and difficulties with motor development and self-help skills.

Intensity. Together with treatment method, treatment intensity is perhaps the most important element of the Lovaas Approach. An effective intervention is believed to require highly intensive teaching to allow the child with autism (who also may have intellectual disability) to catch up, as much as possible, with typical peers. Research on the Lovaas approach indicates that 30–40 h per week of one-to-one intervention may be required to produce optimal gains.

Duration. In most cases, practitioners of the Lovaas approach recommend that intervention last for a minimum of two years and may continue for several additional years with a reduced number of hours of one-to-one instruction along with support for participation in school.

Historical Background

The Lovaas approach can be traced back to the late 1950s and the early 1960s, when researchers began using operant principles, derived from the animal laboratory, to address human problems. Ferster and DeMyer (1961) conducted the first study demonstrating the use of operant condition to teach individuals with autism to learn new skills. Later, Wolf, Risley, and Mees (1964) reported the first behavioral intervention to reduce problem behaviors in a child with autism.

From 1965 until 1970, Lovaas and colleagues published a number of seminal studies, reporting the first demonstration of an effective way to teach nonverbal children to speak, a study on establishing social (secondary) reinforcers, a procedure for teaching children to imitate, and several studies on interventions to reduce life-threatening self-injury and problem behaviors.

In 1971, they discovered that many children with autism have a detail-focused learning style, which they called *stimulus overselectivity*, that could hinder the children from acquiring important skills. Also in the 1970s, Lovaas and colleagues presented research showing that stereotyped behavior could be reinforced by the sensory and/or perceptual stimuli produced by the behavior itself (e.g., vestibular and visual stimulation as reinforcement for body rocking). Hence, they used the term “*self-stimulatory*” to describe such behavior.

In 1973, Lovaas and colleagues published their first long-term follow-up study. Twenty children with autism participated. After 12–14 months of behavioral treatment, stereotyped behaviors including echolalia decreased and appropriate speech, appropriate play, and social nonverbal behaviors increased. Some of the children were discharged to a state hospital while others remained with their parents, who had received parent training. Follow-up assessments 1–4 years after the end of treatment showed that children whose parents were trained to carry out the behavioral treatment continued to improve, while children who were institutionalized regressed. After a brief reinstatement of the behavioral treatment, however, the institutionalized children recouped some of their original gains.

From the 1973 follow-up study, Lovaas made six important observations that occasioned what has been labeled the 1987 Early Intervention Project, the landmark study for which Lovaas is best known. First, the youngest children in the 1973 study appeared to make the greatest gains; hence, the 1987 study focused on younger children. Second, treatment effects in the 1973 study were situation specific, and as a result, treatment was moved away from a hospital or clinic setting and into the children’s home and everyday

environment. Third, because response generalization such as becoming more social after learning language rarely occurred, treatment in the 1987 study was designed to target most or all of the children's excess and deficit behaviors. Fourth, some parents became skilled teachers of their children and were the best allies in helping accelerate and maintain treatment gains. Fifth, they offered treatment for most of the child's waking hours, for 2 or more years, and focused on teaching the children to develop friendships with typical peers. According to Lovaas, this therapeutic arrangement was designed to resemble the type of learning environment available to typical children, who learn from their environment from the time they wake up in the morning and until they go to sleep, 365 days per year.

Lovaas's 1987 report indicated that children with autism treated with the Lovaas approach achieved vastly better outcomes than similar children who received little or no such treatment. Lovaas described 9 of the 19 intensively treated children as "normal functioning." In so doing, he challenged the prevailing belief that, although children with autism might be able to learn isolated skills, they would always be delayed and socially isolated. The study sparked passionate debate. While some considered it as a breakthrough, others criticized it and argued that it was an exaggeration to describe the children with the most favorable outcomes as "normal functioning."

In the 1990s, Lovaas built on the 1987 study by co-authoring a long-term follow-up of children in the 1987 study as well as several replication studies.

Rationale or Underlying Theory

In the Lovaas approach, a working hypothesis is that children with autism have a learning deficit that is biologically based and is responsible for the behavioral deficits and excesses exhibited by these children but that can be overcome with specialized instruction. The Lovaas approach directly targets these excesses (e.g., stereotyped behaviors, aggression, and self-injurious

behaviors) and deficits (e.g., communication, play, and social skills) rather than attempting to address some hypothesized underlying conditions or processes or attempting to address "autism" *per se*. Lovaas argued that there is no "magic bullet" in autism treatment and that all behavioral excesses and deficits must be addressed separately.

In the Lovaas approach, the treatment environment should differ as little as possible from the typical educational environment in order to help the child with autism transfer skills to the typical environment and function more successfully in that setting. Lovaas emphasized that typically developing children learn during all waking hours, every day. To approximate this learning environment, and to allow the child with autism to catch up as much as possible with his/her peers, Lovaas argued that an appropriate treatment environment should be in effect most of the day, 7 days per week.

Goals and Objectives

The curriculum is individualized based on each child's needs and stages of development that typically developing children go through but tends to follow a sequence described in teaching manuals and is summarized below:

Beginning Curriculum. Beginning targets include learning readiness skills in the areas of attention, communication, social initiations, and play. Examples include sitting in a chair, responding to simple instructions such as "come here" and "wave bye-bye," pointing, requesting favorite items, joint attention, matching identical objects, imitating gross motor actions or imitating actions with objects, imitating sounds and words, identifying and naming objects, playing independently with toys, and basic turn taking and interactive skills such as rolling a ball to and from an adult.

Intermediate Curriculum. Intermediate targets include further language training such as identification and naming of abstract concepts, parallel play, turn taking, imitating sentences, pre-academic skills such as identifying letters and

numbers, drawing and tracing, and self-help skills such as dressing, toileting, drinking from an open cup, and increasing the range of foods and drink taken.

Advanced Curriculum. Advanced targets include conversation and asking questions, advanced pretend play and cooperative play, social-emotional skills such as theory of mind, advanced academic skills, observational learning, and learning in the classroom environment.

Treatment Participants

The Lovaas approach was developed primarily for children with autism who have mild to moderate intellectual disability and are under the age of 42 months at treatment onset. However, it has been evaluated with other groups, including children with autism and more severe intellectual disability, children with autism who entered treatment up to the age of 7 years, and non-autistic children with intellectual disability.

Several studies have examined relations between intake variables and outcome measures. A meta-analysis published in 2010 by Eldevik and colleagues indicated that the most robust finding to date is a strong positive correlation between treatment intensity and outcome.

Treatment Procedures

Intervention methods build on detailed knowledge of principles and procedures from behavior analysis. *Discrete trial teaching* is a highly structured teaching procedure used to maximize learning. It is used to establish a number of important skills, including cognitive skills, communication, play, social, and self-help skills. The teaching strategy involves (a) breaking skills into small steps, (b) teaching each step of the skill individually until mastered, (c) providing repetitions of teaching steps, (d) prompting the correct skill and fading (removing) the prompts whenever possible, and (e) using reinforcement and stimulus control procedures.

Natural environment teaching is used to teach behaviors in the situations where they routinely occur. During mealtime, for example, the child is reinforced for sitting nicely or instructed how to use a knife and fork appropriately. Whenever possible, the reinforcer is the consequence that naturally follows the target behavior such as going outside to perform a desired activity after practicing tying shoes.

Task analyses and chaining are used to break down complex behaviors (e.g., toy play and language) into smaller units that can be reliably measured and more easily taught. For example, to teach language skills the behavioral components of each skill are identified, and teaching begins with the most basic components such as vocal imitation of sounds. These individual behaviors are then systematically taught and chained together to make one complex behavior.

School integration begins once a range of skills have been acquired that enable the child to access materials, follow the curriculum, and interact with peers in the preschool or school environment. The aim is for a child to learn increasingly from peers, the class teacher, and the school curriculum. Initially, a therapist shadows the child at school, prompting and reinforcing the child for engagement in social interactions and academic activities while implementing behavior support strategies to minimize inappropriate or interfering behaviors. Once the child can follow segments of activities, whole activities, or parts of the full school day, the shadow will then help the child to learn new information and new skills directly from the school environment and not specifically from the one-to-one teaching. For a child who does not learn sufficiently from the classroom environment, the therapist will continue to shadow on a long-term basis and concentrate on integrating the child for activities in which he/she can be successful. Time is also allocated to ongoing one-to-one teaching, both at home and school, during which individual learning goals, including independent living skills, can be addressed more efficiently than in the classroom. To promote each child's independence, shadowing is gradually reduced on a task-specific basis or for periods of the school day.

Efficacy Information

In Lovaas's 1987 study, 19 children received 40 h per week of one-to-one treatment based on the Lovaas approach for a minimum of 2 years. A comparison group of 19 children received 10 h or less per week of the same treatment. A second comparison group of 21 children came from the same agency that diagnosed the majority of the other participants and received community treatment as usual. The mean age at intake was 33 months. When reevaluated at a mean age of 7 years, children in the experimental group had gained an average of 20 IQ points to a mean of 83, and they had made major advances in educational placement. Nine of 19 participants achieved IQ scores of 85 or above and regular educational placement without assistance. In contrast, the two comparison groups showed little change in IQ. The experimental group maintained its gains at a second follow-up conducted when the children averaged 13 years of age.

Similar results have been reported by other investigators, though in some studies the effect sizes were smaller than in the 1987 study. In a meta-analysis, Eldevik, Hastings, Hughes, Jahr, Eikeseth, and Cross (2009) reported effect sizes of 1.10 for IQ change and 0.66 for change in adaptive behavior. By convention, these effect sizes are considered large. However, some investigators have reported somewhat smaller effect sizes.

All studies have reported considerable variability in outcome. A meta-analysis by Eldevik et al. (2010a) sought to determine what proportion of children made clinically meaningful improvements with two metrics: the reliable change index (RCI), which is a measure of how many participants make changes that are statistically significant (unlikely to be due to statistical chance), and the number needed to treat (NNT) in order to have one additional success more than would occur without treatment. The RCI indicated that an IQ gain of 27 points between intake and follow-up was required for statistically reliable change and that 30% of children across studies achieved reliable change in IQ. To achieve reliable change in adaptive behavior, a gain of 21 points was required, and 21% of the participants achieved

this. NNT for IQ was 5, and NNT for adaptive behavior was 7, indicating an effective intervention.

While researchers generally agree that the results of studies have generally been positive, there remains controversy about the scientific quality of the studies, and many have called for additional research with improved research designs.

Outcome Measurement

The most common outcome measures used are IQ, language, and adaptive functioning. The Bayley Scales of Infant Development and the Wechsler Intelligence Scales (WIPPSI or WISC) are the most common IQ tests.

The most widely used instrument to assess language is the Reynell Developmental Language Scales, but due to their severe delays in communication, many participants fail to achieve basal on the Reynell at intake.

To assess adaptive behavior, the Vineland Adaptive Behavior Scales is used.

Some studies have also assessed other domains, such as maladaptive behavior (using the maladaptive scale from Vineland Adaptive Behavior Scales, the Personality Inventory for Children, and the Child Behavior Checklist), school performance (using the Woodcock-Johnson III Tests of Achievement), and changes in diagnosis (using the Autism Diagnostic Interview-Revised (ADI-R)).

Qualifications of Treatment Providers

There are currently no established programs to certify professionals to become competent Lovaas-approach therapists or Lovaas-approach supervisors. Practitioners of the Lovaas approach indicate that, to become a competent therapist, individuals should obtain a bachelor's degree in psychology, education, or social work, with coursework in applied behavior analysis, developmental disorders, and child development. In addition, they require therapists to work at least

three months hands-on with a child in an apprenticeship with a trained therapist. At the end of this training, the therapist must be able to apply the behavioral principles correctly during one-to-one teaching.

To become a supervisor, Lovaas and colleagues have required the practitioner to be an experienced Lovaas-approach therapist and hold a master's degree that includes graduate courses in applied behavior analysis, developmental disorders, and child development. In addition, the supervisors must learn how to conduct staff training, develop and individualize the child's curriculum, and work with families. These skills can be learned through working in an apprenticeship with a competent Lovaas-approach supervisor and by studying available teaching manuals, evidenced-based parent training programs, and typical development. Finally, the supervisor should be supervised by a Ph.D. level clinical psychologist specializing in the Lovaas approach.

See Also

- ▶ [Early Intensive Behavioral Intervention \(EIBI\)](#)
- ▶ [Early Intervention](#)
- ▶ [Lovaas, O. Ivar](#)
- ▶ [Psychological Assessment](#)
- ▶ [UCLA Young Autism Project](#)

References and Reading

- Cohen, H., Amerine-Dickens, M., & Smith, T. (2006). Early intensive behavioral treatment: Replication of the UCLA model in a community setting. *Developmental and Behavioral Paediatrics*, 27, 145–155.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). New Jersey: Prentice Hall.
- Davis, B. J., Smith, T., & Donahoe, P. (2002). Evaluating supervisors in the UCLA treatment model for children with autism: Validation of an assessment procedure. *Behavior Therapy*, 33, 601–614.
- Eikeseth, S. (2009). Outcome of comprehensive psycho-educational interventions for young children with autism. *Research in Developmental Disabilities*, 30, 158–178.
- Eikeseth, S. (2010). Examination of qualifications required of an EIBI professional. *European Journal of Behavior Analysis*, 11, 239–246.
- Eikeseth, S., Smith, T., Jahr, E., & Eldevik, S. (2002). Intensive behavioral treatment at school for 4- to 7-year-old children with autism. A 1-year comparison controlled study. *Behavior Modification*, 26, 49–68.
- Eikeseth, S., Smith, T., Jahr, E., & Eldevik, S. (2007). Outcome for children with autism who began intensive behavioral treatment between ages 4 and 7: A comparison controlled study. *Behavior Modification*, 31, 264–278.
- Eldevik, S., Hastings, R. P., Hughes, J. K., Jahr, E., Eikeseth, S., & Cross, S. (2009). Meta-analysis of early intensive behavioral intervention for children with autism. *Journal of Clinical Child and Adolescent Psychology*, 38, 439–450.
- Eldevik, S., Hastings, R. P., Hughes, J. K., Jahr, E., Eikeseth, S., & Cross, S. (2010a). Analyzing outcome for children with autism receiving early intensive behavioral intervention: Secondary analysis of data from 457 children. *The American Journal on Intellectual and Developmental Disabilities*, 34, 16–34.
- Eldevik, S., Jahr, E., Eikeseth, S., Hastings, R. P., & Hughes, C. J. (2010b). Cognitive and adaptive behavior outcomes of behavioral intervention for young children with intellectual disability. *Behavior Modification*, 34, 16–34.
- Ferster, C. B., & DeMyer, M. K. (1961). The development of performances in autistic children in an automatically controlled environment. *Journal of Chronic Diseases*, 13, 312–345.
- Hayward, D. W., Eikeseth, S., Gale, C., & Morgan, S. (2009). Assessing progress during treatment for young children with autism receiving intensive behavioural interventions. *Autism*, 13, 613–633.
- Leaf, R., & McEachin, J. (1999). *A work in progress: Behavior management strategies and a curriculum for intensive behavioral treatment of autism*. New York: DRL Books, L. L. C.
- Lovaas, O. I. (1977). *The autistic child: Language development through behavior modification*. New York: Irvington.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- Lovaas, O. I. (2003). *Teaching individuals with developmental delays: Basic intervention techniques*. Austin: Pro-Ed.
- Lovaas, O. I., & Smith, T. (1989). A comprehensive behavioral theory of autistic children: paradigm for research and treatment. *Journal of Behavior Therapy and Experimental Psychiatry*, 20, 17–29.
- Lovaas, O. I., Koegel, R. L., Simmons, J. Q., & Long, J. (1973). Some generalization and follow-up measures on autistic children in behavior therapy. *Journal of Applied Behavior Analysis*, 6, 131–166.
- Lovaas, O. I., Ackerman, A., Alexander, D., Firestone, P., Perkins, M., & Young, D. B. (1981). *Teaching developmentally disabled children: The ME Book*. Austin: Pro-Ed.
- Maurice, C. (Ed.), Green, G., & Luce, S. (Co-Eds.). (1996). Behavioral intervention for young children with

- autism: A manual for parents and professionals. Austin: Pro-Ed.
- Maurice, C., Green, G., & Foxx, R. M. (Eds.). (2001). *Making a difference: Behavioral intervention for autism*. Austin: Pro-Ed.
- McEachin, J. J., Smith, T., & Lovaas, O. I. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. *American Journal on Mental Retardation*, 97, 359–372.
- Sallows, G. O., & Graupner, T. D. (2005). Intensive behavioral treatment for children with autism: Four-year outcome and predictors. *American Journal on Mental Retardation*, 110, 417–438.
- Smith, T., Eikeseth, S., Klevstrand, M., & Lovaas, O. I. (1997). Intensive behavioral treatment for preschoolers with severe mental retardation and pervasive developmental disorder. *American Journal on Mental Retardation*, 102, 238–249.
- Smith, T., Groen, A. D., & Wynn, J. W. (2000). Randomized trial of intensive early intervention for children with pervasive developmental disorder. *American Journal on Mental Retardation*, 105, 269–285.
- Wolf, M. M., Risley, T., & Mees, H. (1964). Application of operant conditioning procedures to the behavior problems of an autistic child. *Behaviour Research and Therapy*, 1, 305–312.

Lovaas, O. Ivar

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Name and Degrees

Dr. O. Ivar Lovaas received his B.A. in Social Studies from Luther College, Decorah, Iowa, in 1951; an M.S. in psychology from the University of Washington, Seattle, Washington, in 1954; and a Ph.D. in psychology from the University of Washington in 1958.

Tristram Smith: deceased.

Major Appointments

From 1958 until 1961, Lovaas worked at the University of Washington's Child Development Institute as an acting assistant professor. In 1961, he accepted a position as an assistant professor in the University of California, Los Angeles (UCLA), Psychology Department, where he spent the remainder of his career. He became an associate professor in 1965, tenured professor in 1967, and professor emeritus in 2004.

In 1962 Dr. Lovaas founded the UCLA Clinic for the Behavioral Treatment of Children, and between 1972 and 1980, Dr. Lovaas acted as a Research Specialist at Camarillo State Hospital, Camarillo, California.

In 1995, Dr. Lovaas founded the Lovaas Institute for Early Intervention (the LIFE Institute).

Major Honors and Awards

Dr. Lovaas received many honors for his work, including an Honorary Doctorate from Luther College, a Guggenheim Fellowship, the Edgar Doll Award from Division 33 of the American Psychological Association (1994), the California Senate Award (1994), the Research Award from the American Association on Mental Retardation (1994), Distinguished Guest Faculty Award from Ohio State University (1994), a Golden Key Honorary Award (2000), the Distinguished Research Contribution Award from Division 53 of the American Psychological Association (2001), and the California Association for Behavior Analysis award for outstanding contributions to behavior analysis (posthumous, 2011).

In addition, he was an honorary member of a number of organizations including Autism Society of America, the Norwegian Association for Behavior Analysis (NAFO), the Uruguayan Society for Analysis and Behavior Therapy (SUATEC), and the American Board of Medical Psychotherapies.

Landmark Clinical, Scientific, and Professional Contributions

From 1965 and on, Lovaas and colleagues published a number of seminal studies transforming interventions and services for autism. An important initial contribution was the development of an apparatus and recording procedure allowing repeated and reliable measures of deficit language and social behaviors and excess stereotyped, aggressive, and self-injurious behaviors (Lovaas et al. 1965a, b). This recording procedure allowed moment-to-moment assessment of the dependent variables, providing the investigators immediate feedback regarding the effectiveness of various treatment variables. This innovation, together with the use of single subject research designs, facilitated the groundbreaking finding subsequently presented by Dr. Lovaas and colleagues.

From 1965 until 1970, Lovaas and colleagues reported, among other findings, the first demonstration of an effective way to teach nonverbal children to speak (Lovaas, 1966; Lovaas et al. 1966a), a study on establishing social (secondary) reinforcers (Lovaas et al. 1966b), a procedure for teaching children to imitate (Lovaas et al. 1967), and several studies on interventions to reduce life-threatening self-injury and aggression (c.f., Lovaas et al. 1965b) and a pioneering investigation of antecedents and consequences that maintained a problem behavior (Lovaas and Simmons 1969), a forerunner of what is now called experimental functional analysis.

Also in the 1960s, Lovaas and colleagues conducted studies indicating that punishment procedures involving aversives such as mild doses of electric shock could be effective in reducing extreme, sometimes life-threatening aggression or self-injury. By the late 1980s, however, Lovaas felt such procedures had become unnecessary because non-aversive interventions had become so sophisticated and successful that aversives were no longer necessary. Accordingly, he stopped using them at that time.

In 1971, Lovaas and colleagues showed that many children with autism have a learning style

that could hinder them from learning important skills and generalizing these skills outside the intervention setting. This style involved attending to only one element of a complex stimulus (e.g., visual but not auditory components of a stimulus). They called this style *stimulus overselectivity* (Lovaas et al. 1971) and published several additional studies on this topic during the 1970s.

In 1973, Lovaas and colleagues published their first long-term follow-up study (Lovaas et al. 1973). Twenty children with autism participated. After 12–14 months of behavioral treatment, stereotyped behaviors including echolalia decreased, and appropriate speech, appropriate play, and social nonverbal behaviors increased. At the end of treatment, some of the children were discharged to a state hospital, while some children remained with their parents, who had received parent training. Follow-up assessments 1–4 years after the end of treatment showed that children whose parents were trained to carry out the behavioral treatment continued to improve, while children who were institutionalized regressed.

Based on data from this study, Lovaas hypothesized that treatment effects could be optimized if intervention was started *early* in the child's life and if intervention was *comprehensive*, that is, addressing all behavior excesses and deficits exhibited by a particular child. In addition, Lovaas argued that intervention had to be *intensive*; that is, it should provide a learning environment for the child throughout the whole day, be carried out in the child's *natural environment* (such as at home and in school, rather than in an institution), and include persons who are part of the child's natural environment (such as parents, teachers, peers). Finally, Lovaas argued that the children should enter *typical classes* to access typical peers to model appropriate behaviors, rather than attend special classes.

In 1970, Lovaas initiated the UCLA Young Autism Project in order to provide treatment based on these principles. In 1987, he published the first set of results in the landmark study for which he is now best known: the report entitled *Behavioral Treatment and Normal Educational and Intellectual Functioning in Young Autistic*

Children, which appeared in the *Journal of Consulting and Clinical Psychology*. This report indicated that children with autism treated with the Lovaas Approach achieved vastly better outcomes than similar children who received little or no such treatment. Lovaas described 9 of the 19 intensively treated children as “normal functioning” and possibly even recovered. In so doing, he challenged the prevailing belief that, although children with autism might be able to learn isolated skills, they would always be delayed and socially isolated. The study sparked passionate debate. While some considered it as a breakthrough, others criticized it and argued that it was an exaggeration to describe the children with the most favorable outcomes as normal functioning.

Also in 1987, Lovaas and colleagues presented a review of stereotyped behavior (Lovaas et al. 1987), which they called *self-stimulatory* because, they argued, it was maintained by the sensory and/or perceptual stimuli produced by the behavior itself (e.g., vestibular and visual stimulation as reinforcement for body rocking).

In the 1990s, Lovaas built on the 1987 study by co-authoring a long-term follow-up of children in the 1987 study (McEachin et al. 1993), as well as several replication studies. He also obtained two federal grants to support replications by other investigators. He continued to publish important work until he was in his late seventies, notably a revision of his intervention manual (Lovaas 2003).

Short Biography

Lovaas was born on May 8, 1927, in Lier, Norway. His father was a journalist. During the Second World War, Norway was occupied by the Nazis for 5 years (1940–1945). At that time, Lovaas was in his teens, and this experience came to affect Lovaas in many ways. Most importantly, it made him reflect on the “nature versus nurture” issue. He believed that the Germans could not be inherently evil, which in turn, convinced him that the environment could exert highly powerful effects on behaviors.

In 1947, shortly after the Second World War, Lovaas graduated from Gymnasium (high school). In 1950, after serving his military duty in the Norwegian air force as a medic, Dr. Lovaas immigrated to the USA, attending Luther College in Decorah, Iowa, on a violin scholarship. Subsequently, in 1951, he went on to the University of Washington, Seattle, where he stayed for the next 10 years, receiving his M.S. and Ph.D. in psychology and working as an acting assistant professor.

Lovaas’ interest in children with autism came about accidentally. His initial research had been on how a person’s language may influence non-verbal behaviors, a process later described as instructional control or rule-governed behavior. At UCLA, Lovaas sought to extend this research by studying how to teach language to children who had communication delays and testing the effects of improved language on other behaviors such as social interaction. After visiting a clinic for children with autism, he became convinced that he had found the ideal group for this research. Subsequently, at the laboratories at UCLA and Camarillo State Hospital, Dr. Lovaas and colleagues conducted the groundbreaking and pioneering research, which have had tremendous influence on applied behavior analysis (ABA) and services for individuals with autism.

Alongside his research and clinical innovations, Lovaas devoted much of his energy to advocacy on behalf of autism and popularization of ABA. In the 1960s, he helped found the parent organization now called the Autism Society of America. He also became a strong proponent of moving children (and adults) with autism from large institutions into small group homes. Subsequently, as many children in his research successfully entered general education classes in public schools, he also became a proponent of inclusion in these classes.

Lovaas’ popularizations included one of the first films on ABA, produced in 1969, to show interventions for teaching language to children with autism. Lovaas also published two of the first ABA intervention manuals in 1977 and 1981. These manuals laid out how and what to teach, thereby making ABA accessible to many families and providers. Always quotable and not

shy about extolling the benefits of ABA, Lovaas was profiled in many media outlets such as *Life*, *Rolling Stone*, the *New York Times*, and the *CBS Evening News*. He was also a popular instructor, introducing generations of students to autism and ABA.

He is survived by his wife, Nina Lovaas, four children from his first marriage, six grandchildren, and three great-grandchildren.

Lovaas died at the age of 83 on August 2, in Lancaster, California, 2010.

See Also

- [Behavior Modification](#)
- [Early Intensive Behavioral Intervention \(EIBI\)](#)
- [Early Intervention](#)
- [Home-Based Programs](#)
- [Lovaas Approach](#)
- [Structured Behavioral Interventions](#)

References and Reading

- Lovaas, O. I. (1966). A program for the establishment of speech in psychotic children. In J. Wing (Ed.), *Childhood autism*. London: Pergamon Press.
- Lovaas, O. I. (1977). *The autistic child: Language development through behavior modification*. New York: Irvington.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- Lovaas, O. I. (1993). The development of a treatment-research project for developmentally disabled and autistic children. *Journal of Applied Behavior Analysis*, 26, 617–630.
- Lovaas, O. I. (2003). *Teaching individuals with developmental delays: Basic intervention techniques*. Austin: Pro-Ed.
- Lovaas, O. I., & Simmons, J. Q. (1969). Manipulation of self-destruction in three retarded children. *Journal of Applied Behavior Analysis*, 2, 143–157.
- Lovaas, O. I., Freitag, G., Gold, V. J., & Kassorla, I. C. (1965a). Recording apparatus and procedure of observation of behaviors of children in free play settings. *Journal of Experimental Child Psychology*, 2, 108–120.
- Lovaas, O. I., Freitag, G., Gold, V. J., & Kassorla, I. C. (1965b). Experimental studies in childhood schizophrenia: Analysis of self-destructive behavior. *Journal of Experimental Child Psychology*, 2, 67–84.
- Lovaas, O. I., Berberich, J. P., Perloff, B. F., & Schaeffer, B. (1966a). Acquisition of imitative speech by schizophrenic children. *Science*, 151, 705–707.
- Lovaas, O. I., Freitag, G., Kinder, M. I., Rubenstein, B. D., Schaeffer, B., & Simmons, J. W. (1966b). Establishment of social reinforcers in two schizophrenic children on the basis of food. *Journal of Experimental Child Psychology*, 4, 109–125.
- Lovaas, O. I., Freitas, L., Nelson, K., & Whalen, C. (1967). The establishment of imitation and its use for the development of complex behavior in schizophrenic children. *Behaviour Research and Therapy*, 5, 171–181.
- Lovaas, O. I., Schreibman, L., Koegel, R., & Rehm, R. (1971). Selective responding by autistic children to multiple sensory input. *Journal of Abnormal Psychology*, 77, 211–222.
- Lovaas, O. I., Koegel, R. L., Simmons, J. Q., & Long, J. (1973). Some generalization and follow-up measures on autistic children in behavior therapy. *Journal of Applied Behavior Analysis*, 6, 131–166.
- Lovaas, O. I., Ackerman, A., Alexander, D., Firestone, P., Perkins, M., & Young, D. B. (1981). *Teaching developmentally disabled children: The ME book*. Austin: Pro-Ed.
- Lovaas, O. I., Newsom, C., & Hickman, C. (1987). Self-stimulatory behavior and perceptual reinforcement. *Journal of Applied Behavior Analysis*, 20, 45–68.
- McEachin, J. J., Smith, T., & Lovaas, O. I. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. *American Journal on Mental Retardation*, 97, 359–372.
- Smith, T., & Eikeseth, S. (2011). O. Ivar Lovaas: Pioneer of applied behavior analysis and intervention for children with autism. *Journal of Autism and Developmental Disorders*, 41, 375–378.

Lovan

- [Prozac™ \(Fluoxetine\)](#)

Low Registration

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Definition

This term refers to the individual's limited ability to notice or respond to important or salient

sensory information. Although Ayres discussed “registration of sensory information,” the term “low registration” was not included in her writings about sensory integration. The term “low registration” is used in the sensory processing literature and is clearly defined by Dunn (2007) as a pattern of sensory processing where the individual has a high threshold to sensory experiences and does not notice or detect changes in sensory situations at the same rate of others. The individual may appear passive in response to changes in sensory intensities due to limited detection of those changes. Individuals who have low registration of sensory experiences benefit from occupational therapy services designed to enhance sensory processing and improved ability to detect and respond to changes in sensory experiences.

References and Reading

- Dunn, W. (1997). The impact of sensory processing abilities on the daily lives of young children and their families: A conceptual model. *Infants and Young Children*, 9(4), 23–35.
- Dunn, W. (2001). The sensations of everyday life: Theoretical, conceptual and pragmatic considerations. *American Journal of Occupational Therapy*, 55, 608–620.
- Dunn, W. (2007). Supporting children to participate successfully in everyday life by using sensory processing knowledge. *Infants and Young Children*, 20, 84–101.
- Lane, S. J., Miller, L. J., & Hanft, B. E. (2000). Toward a consensus in terminology in sensory integration theory and practice: Part 2: Sensory integration patterns of function and dysfunction. *Sensory Integration Special Interest Section Quarterly*, 23, 1–3.
- Miller, L. J., Anzalone, M. E., Lane, S. J., Cermak, S. A., & Osten, E. T. (2007). Concept evolution in sensory integration: A proposed nosology for diagnosis. *American Journal of Occupational Therapy*, 61, 135–140.

Low-Tech Augmentative and Alternative Communication (AAC)

- [Low-Technology Device](#)

Low-Tech System

- [Low-Technology Device](#)

Low-Technology Device

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Synonyms

[Low-tech augmentative and alternative communication \(AAC\)](#); [Low-tech system](#)

Definition

Although there are differing opinions regarding the precise definition of augmentative and alternative communication (AAC) systems, technically, a low-technology (low-tech) AAC device is one that requires a power source such as a battery to operate and is very easy to program. A no-tech system is any communication device that does not require a power source. An example of a no-tech system is a picture communication board. Examples of low-tech systems are the BIGmack[®], LITTLEmack[®], Step-by-Step Communicator[®], and the SpeakEasy[®] which are sold by AbleNet. See Beukelman and Mirenda (2012) for a full description of low-technology devices and their implementation.

See Also

- [Alternative Communication](#)
- [Assistive Devices](#)
- [Communication Board](#)
- [Pictorial Cues/Visual Supports \(CR\)](#)
- [Total Communication \(TC\) Approach](#)

References and Reading

Beukelman, D. R., & Mirenda, P. (2012). *Augmentative and alternative communication: Supporting children and adults with complex communication needs*. Baltimore: Brooks Publishing.

Loxapine

► [Loxitane](#)

Loxitane

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Synonyms

[Loxapine](#)

Definition

Loxapine is a traditional antipsychotic medication developed for the treatment of schizophrenia. As with the other members of traditional antipsychotic class, it has some risk of neurological adverse effects. Loxapine has not been studied in children or adults with autism spectrum disorder.

See Also

► [Schizophrenia](#)

References and Reading

Martin, A., Scahill, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.

L-Tryptophan

► [Tryptophan](#)

Luria-Nebraska Neuropsychological Battery

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Description

The Luria-Nebraska is a neuropsychological evaluation battery that was developed to uncover the presence of cognitive impairments and to locate focal brain abnormalities that may account for these impairments in individuals 15 years of age and older. The battery consists of 11 clinical scales assessing major areas of neuropsychological functioning (motor functions, rhythm, tactile functions, visual functions, receptive speech, expressive speech, writing, reading, arithmetic, memory, and intellectual processes), two sensorimotor scales (left hemisphere, right hemisphere), and three summary scales (pathognomonic, profile elevation, and impairment). A parallel form of this battery is available and includes an additional clinical scale that evaluates delayed recollection of previously administered memory items (intermediate-term memory scale). Since publication, other scales have been developed and made available to users, including 8 localization scales and 28 factor scales, some of which include few items and must be interpreted with caution (Lezak et al. 2004). Administration time usually lasts 2–3 h. Charles Golden developed a version of this battery for use in children aged 8–12 years (Golden 1981). It uses the same test stimuli with some additional materials (see Lark 2004 and Golden 2011, for reviews of the children's revision of the Luria-Nebraska).

Historical Background

The Luria-Nebraska was published between 1980 and 1985 and was initially known as the Luria-Dakota Neuropsychological Battery during its development. The battery was based on the evaluation techniques practiced by Alexander Luria, which were compiled and organized into test items by Anne-Lise Christensen. The development of the test was based on the principle that behavior consists of sensory, cognitive, and behavioral processes, and disruptions in any of these domains may lead to impairments in behavior. As a result, tasks assessing each area of processing were selected from Christensen's collection by Golden and colleagues, and items that best discriminated between healthy and neurologically impaired individuals were included in the battery.

Psychometric Data

Reports of internal consistency coefficients for the measure range between .40 and .94, with most in the .80s, while test-retest reliability and interrater reliability for all of the scales have been reported to fall between .80 and .95 (Golden et al. 1985). Several factor analytic studies support the factor structure proposed by the authors (Moses 1984a, b, c). Validation of the measure has depended on studies suggesting that this measure can accurately discriminate healthy adults from adults who have suffered a brain injury (Golden et al. 1985; Kane et al. 1985; Moses et al. 1983). However, many of these studies included patients with moderate to severe impairments. Some have argued that the Luria-Nebraska includes tasks that most adults can complete successfully unless they are severely impaired or relatively low functioning, and as a result this battery may be less useful in identifying mild deficits among high-functioning individuals (Adams 1980, 1984; Delis and Kaplan 1982; Lezak et al. 2004). Furthermore, the Luria-Nebraska does not appear to accurately localize the laterality of lesions, particularly among patients with aphasia (Delis and Kaplan 1982; Goldstein et al. 1987; Ryan et al. 1988; Sears et al. 1984).

Clinical Uses

Golden et al. (1982) cautioned against the use of the Luria-Nebraska among professionals who did not receive training in neuropsychology, neurology, and Luria's testing approach. The authors of the battery also reminded users that the results should depend upon scores obtained on the scales of this battery, patterns of responses, behavioral observations, and hypothesis testing. Many neuropsychologists have also cautioned against using this measure beyond the diagnosis of a moderate to severe neurological impairment (Adams 1984; Crosson and Warren 1982; Delis and Kaplan 1982; Spiers 1984). The test was developed at a time when neuropsychologists were being asked to identify the presence, side, and location of lesions; however, this is much less relevant in the current age of advanced imaging techniques.

References and Reading

- Adams, K. M. (1980). In search of Luria's battery: A false start. *Journal of Consulting and Clinical Psychology*, 48, 522–524.
- Adams, K. M. (1984). Luria left in the lurch: Unfulfilled promises are not valid tests. *Journal of Clinical Neuropsychology*, 6, 455–465.
- Crosson, B., & Warren, R. L. (1982). Use of the Luria-Nebraska neuropsychological battery in aphasia: A conceptual critique. *Journal of Consulting and Clinical Psychology*, 50, 22–31.
- Delis, D. C., & Kaplan, E. (1982). The assessment of aphasia with the Luria-Nebraska neuropsychological battery: A case critique. *Journal of Consulting and Clinical Psychology*, 50, 32–39.
- Golden, C. J. (1981). The Luria-Nebraska children's battery: Theory and formulation. In G. W. Hynd & J. E. Obrzut (Eds.), *Neuropsychological assessment and the school-age child: Issues and procedures* (pp. 277–302). New York: Grune & Stratton.
- Golden, C. J. (2011). The Luria-Nebraska neuropsychological children's battery. In A. S. Davis & A. S. Davis (Eds.), *Handbook of pediatric neuropsychology* (pp. 367–378). New York: Springer.
- Golden, C. J., Ariel, R. N., McKay, S. E., Wilkening, G. N., Wolf, B. A., & MacInnes, W. D. (1982). The Luria-Nebraska neuropsychological battery: Theoretical orientation and comment. *Journal of Consulting and Clinical Psychology*, 50, 291–300.
- Golden, C. J., Purisch, A. D., & Hammeke, T. A. (1985). *Luria-Nebraska neuropsychological battery: Forms I and II manual*. Los Angeles: Western Psychological Services.

- Goldstein, G., Shelly, C., McCue, M., & Kane, R. L. (1987). Classification with the Luria-Nebraska neuropsychological battery: An application of cluster and ipsative profile analysis. *Archives of Clinical Neuropsychology*, 2, 215–235.
- Kane, R. L., Parsons, O. A., & Goldstein, G. (1985). Statistical relationships and discriminative accuracy of the Halstead-Reitan, Luria-Nebraska, and Wechsler IQ scores in the identification of brain damage. *Journal of Clinical and Experimental Neuropsychology*, 7, 211–223.
- Leark, R. A. (2004). The Luria-Nebraska neuropsychological battery – children’s revision. In G. Goldstein & S. Beers (Eds.), *Comprehensive handbook of psychological assessment* (Vol. 1, pp. 147–156). New York: Wiley.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
- Moses, J. A. (1984a). An orthogonal factor solution of the Luria-Nebraska neuropsychological battery items: 1. Motor, rhythm, tactile, and visual scales. *International Journal of Clinical Neurology*, 5, 181–185.
- Moses, J. A. (1984b). An orthogonal factor solution of the Luria-Nebraska neuropsychological battery items: 3. Arithmetic, memory, and intelligence scales. *International Journal of Clinical Neurology*, 6, 24–38.
- Moses, J. A. (1984c). An orthogonal factor solution of the Luria-Nebraska neuropsychological battery items: 1. Motor, rhythm, tactile, and visual scales. *International Journal of Clinical Neurology*, 6, 103–106.
- Moses, J. A., Cardellino, J. P., & Thompson, L. L. (1983). Discrimination of brain damage from chronic psychosis by the Luria-Nebraska neuropsychological battery: A closer look. *Journal of Consulting and Clinical Psychology*, 51, 441–449.
- Ryan, J. J., Farage, C. M., Mittenberg, W., & Kasprisin, A. (1988). Validity of the Luria-Nebraska language scales in aphasia. *International Journal of Neuroscience*, 43, 75–80.
- Sears, J. D., Hirt, M. L., & Hall, R. W. (1984). A cross-validation of the Luria-Nebraska neuropsychological battery. *Journal of Consulting and Clinical Psychology*, 52, 309–310.
- Spiers, P. A. (1984). What more can I say? In reply to Hutchinson, one last comment from Spiers. *Journal of Consulting and Clinical Psychology*, 52, 546–552.

Luvox

► Fluvoxamine

Luvox CR

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Luxembourg and Autism

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Historical Background

Luxembourg is a small trilingual (Luxembourgish, French, and German) European country with an estimated population in July 2017 of 594,130 inhabitants. It is however, the country in Europe with the highest population growth rate and the highest net migration rate (Central Intelligence Agency 2017).

There are no official figures as to the prevalence and incidence of autism in Luxembourg. However, according to the most recent statistics provided by Autism-Europe, autism spectrum disorder affects around 1 in 100 people (Autism-Europe 2017). Therefore, it is estimated that approximately 5,900 people live in Luxembourg with a disorder in the autism spectrum. With Luxembourg’s birth rate at 11.5/1000 (Central Intelligence Agency 2017), it can also be estimated that 68 new cases of autism appear each year in the country.

Services dedicated to people with autism started to appear in Luxembourg in the 1980s and have since then increased substantially. The first organization dedicated to autism in Luxembourg was established in 1981 by the initiative of Gilbert Huyberegts, the father of a child with autism and later also the president of Autism-Europe (from 1989 to 2000). This not-for-profit organization, the *Société Luxembourgeoise pour l’Aide aux Personnes Autistiques* [Luxembourgish Society for the Help of Autistic People], which in 1996 changed its name to *Autisme Luxembourg asbl*, was composed of parents, professionals, and supporters. This society had as its aims to create, develop, and manage preventive and curative measures, as well as to put in place educational and accommodation centers, and to search for a continuous integration of people with autism.

To fulfill one of its aims – to develop educational measures for people with autism – the *Société Luxembourgeoise pour l'Aide aux Personnes Autistiques* created in 1982 educational classes for children with autism within a mainstream elementary school. This initiative was audacious at a time when disabled children attended exclusively special education schools. In 1988, the government created an educational structure, the *Institut pour Enfants Autistiques et Psychotiques* [Institute for Autistic and Psychotic Children] and the classes became part of the *Service de l'Éducation Différenciée* [Special Education Service] of the Ministry for National Education. However, most of the classes remained as they were since the beginning – in mainstream schools. The aim of the *Institut* was to provide children and adolescents with autism with an adequate education, social, and professional integration. For this, the special education classrooms insured an education adapted to their needs and implemented speech and psychomotor therapy.

To foster the personal development of people with autism, in 1989 the *Société Luxembourgeoise pour l'Aide aux Personnes Autistiques* also created the *Centre d'Intégration et de Récréation pour Personnes atteintes d'Autisme* (CIRPA) [Integration and Recreation Center for People with Autism] which offered specialized assistance and support to the needs and capacities of people with autism across their life through the organization of daily activities. In addition to the CIRPA, and to respond to a need of professional development of adults with autism, in 1991 the *Société Luxembourgeoise pour l'Aide aux Personnes Autistiques* created the cooperative society *Peter Pan*, which promoted the training and social integration of adults with autism in a protected work environment. The cooperative society *Peter Pan* has since then been dissolved.

Led by the initiative of Gilbert Huyberegts and a group of parents of children with autism, in 1996 the *Fondation Autisme Luxembourg* [Autism Luxembourg Foundation] was created with the support of the *Société Luxembourgeoise pour l'Aide aux Personnes Autistiques*. The *Fondation Autisme Luxembourg* had as aims to develop the capacities of autistic people and

to provide them with a dignified life. To achieve this, they set as aims to (a) create, develop, and manage preventive and curative measures such as educational and accommodation centers for children, adolescents, and adults with autism; (b) facilitate the integration of people with autism in all educational, socio-cultural, and professional domains; (c) create and maintain a consultation service as well as a center for training and information for the parents of people with autism; and (d) facilitate research on autism.

In 2000, a not-for-profit parental association, the *Association des Parents de Personnes Atteintes d'Autisme de Luxembourg* [Association of Parents of Persons with Autism from Luxembourg], was created to protect the rights of people with autism and their families and to help and support them. Additionally, the association aimed to improve the care services of people with autism throughout their life by facilitating and accentuating the dialog between families and policy makers and to sensitize the general public to better integrate people with autism in the educational, socio-cultural, sportive, and professional domains.

As one of the most recent measures put in place to improve the services offered to people with autism and their families, in 2009 the *Unité Autisme* [Autism Unit] was added to the services proposed by the *Service National de Pédiopsychiatrie du Centre Hospitalier du Luxembourg* [National Service of Child Psychiatry from the *Centre Hospitalier du Luxembourg*]. The service exists since 1995 and includes a consultation unit, a hospitalization unit, and a day care center. The aim of the creation of the *Unité Autisme* was to diagnose autism, support the families, and provide continued care for young children with autism.

Legal Issues, Mandates for Service

In Luxembourg, there are no laws or mandates specific to autism. However, there have been several laws and legal measures that have had an impact on the rights and quality of life of people with autism.

Regarding education, two laws had a major impact for people with autism. In 1973, a law was approved for the right of disabled children, who could not attend mainstream schools, to receive education at a special education school. Until that law was voted, disabled children were often excluded from receiving a formal education. In 1994, the law of 1973 was modified to encourage as much as possible the inclusion of disabled children in mainstream education. Under these laws, children with autism have the right to a formal education and can receive their education either at a special education school, such as the *Institut pour Enfants Autistiques et Psychotiques*, or at a mainstream school, or share their weekly school time between special and mainstream education. At the mainstream education, special arrangements such as having the support of a special education teacher in the classroom for a certain amount of hours per week is also an option. Upon request of the teacher or the parents, an inclusion commission recommends the type of education best indicated for the child. However, parents have the final decision regarding the type of education and care that their child will receive.

Regarding health care, the law of 1998 and its amendment in 2005 addressing the creation of a dependence care insurance has had a major impact on the services and type of care people with autism can receive from the government. In order to receive special care from the dependence insurance, four main conditions need to be met: (a) to be dependent (i.e., to need help for personal hygiene, nutrition, and mobility), (b) to need the assistance for at least 6 months, (c) to required assistance for at least 3.5 h per week, and (d) that the need for assistance is a consequence of illness. The *Cellule d'Évaluation et d'Orientation de l'Assurance Dépendance* [Evaluation and Orientation Unit from the Dependence Care Insurance] assesses requests, advises regarding the existence of dependence, and determines the care and support provided to the person or the caregiver at different levels. The intensity of care and support is adapted according to the level of dependence of a person on someone else. Children younger than 8 years old are compared to a

developmental chart corresponding to autonomy capacities acquired at different ages. Children older than 8 years old, adolescents, and adults are compared in terms of autonomy to an adult. The support can be reflected, among other things, on the attribution, substitution, reduction, or increase of the support given; on the proposal of reeducation or re-adaptation measures; on the type and amount of professional care provided; on the monetary support given to finance support not provided by a professional service; and on the support given to the family or caregivers.

Concerning the work conditions of adults with autism, two laws have had an impact on the employability and fair compensation of work. In 2003, a law was approved regarding the right of disabled people to work and to receive a salary equivalent to the national standards. Under this law, people with autism, presenting difficulties which impair their employability, should receive at least the national minimum wage or the standard salary for the type of work performed that it be in sheltered workshops or in the mainstream job market. Based on an evaluation by the Medical Commission and the advice of the Orientation Commission, the government pays a part of the person's salary equivalent to the assessed percentage of the person's handicap and the remaining is paid by the employer. In 2011 the law of 2003 was amended to stipulate that the government pays the totality of the salary of a disabled person working in sheltered workshops. Additionally, under these laws, the Orientation Commission can decide whether a person with autism can receive free of charge support in orientation, training, re-education, professional integration and reintegration, initiation to the job market, and support for apprenticeships.

Finally, regarding special juridical statuses of disabled people, the law of 1982 on the rights of incapacitated adults provides legal protective measures for people who due to a reduction of their mental capabilities are unable to comply with legal acts. The law foresees three protective regimes (from less to more protective measures): (a) safeguard justice clauses, (b) advice and support by a legal guardianship, and (c) representation by a legal guardianship.

Overview of Current Treatments and Centers

In Luxembourg, services and centers dedicated to people with autism and their families are nowadays varied and spread through the individual's entire lifespan. They range from diagnostic services, treatment and support measures, education, professional integration and training, leisure activities, assistance, and housing. Due to the small size of the country, services are nationally governed and common to the whole country. Services are often centralized around the country's capital but some services have branches in different regions to facilitate access to people.

Diagnosis

There are two institutions that diagnose autism in Luxembourg. The *Unité Autisme* at the *Centre Hospitalier du Luxembourg* diagnoses children until the age of 6 years and prioritizes the early diagnosis of children before the age of 18 months using a neurodevelopmental and environmental approach. To achieve this, the unit is active in bringing awareness about autism to professionals. They provide training to pediatricians as well as to professionals working in services dedicated to early screenings of children's health and development. This collaboration enables an early screening of autism and families are redirected towards the *Unité Autisme* for further assessments and orientation. In 2016, the *Unité Autisme* had 37 new cases for consultation (Unité Autisme 2017). The *Fondation Autisme Luxembourg* performs autism diagnosis to children, adolescents, and adults using a multidisciplinary approach and with a variety of psychological, educational, functional, and medical assessments. The *Fondation* is also often asked to verify previously obtained diagnosis. In 2016, the *Fondation* performed 37 new diagnostic assessments, from which 17 were diagnosed in the autism spectrum, 11 did not fulfill the criteria for an autism diagnosis, and the remaining 9 were ongoing assessments (Fondation Autisme Luxembourg 2017).

Treatment and Support

Both the *Unité Autisme* and the *Fondation Autisme Luxembourg* additionally provide support and guidance to the families by offering services themselves and by re-orienting families to other services. The *Unité Autisme* offers group psychotherapy for children and therapeutic interviews with the families. In 2016 the *Unité Autisme* followed 141 children and their families (Unité Autisme 2017). The *Fondation Autisme Luxembourg* offers support to children, adolescents, and adults with autism regardless of their abilities or difficult behaviors, as well as to their families and professionals working with them. This psychosocial and psychoeducational support comprises home help and advice to parents on strategies to implement at home, learning resources, and individual assistance on hygiene, mobility, and nutrition. They also work with siblings and peers, and frequently offer information sessions about autism to professional working with people with autism. Additionally, the *Fondation* helps families in the setting up of medical care, obtaining assistance from the care insurance and other allowances that are foreseen in Luxembourg's health and social systems, as well as in obtaining appropriate care through other existing services. In 2016, the *Fondation* received 469 requests for support throughout its service in "diagnosis, support, and training" comprising new and follow-up cases as well as requests from professionals (Fondation Autisme Luxembourg 2017). Since 2010, an increase of about 20% in requests for diagnosis is observed each year and more than 40% of the requests concern people with autism without intellectual disability.

Education

In the education side, the *Institut pour Enfants Autistiques et Psychotiques* currently insures the education of 71 children and adolescents with autism aged from 4–18 years old at different educational levels, who due to their specific learning needs cannot attend mainstream education classrooms. The main aim of the *Institut* is to be as inclusive as possible and is thus composed of 11 different classrooms from which the majority are special education classrooms integrated within

mainstream education schools. There are five classes in elementary schools and two classes in secondary schools. Additionally, there are four professional propaedeutic classes at the *Institut* that provide training preparing students older than 12 years for a profession. Students with autism who do not attend special education classes can also receive support for their special needs within the mainstream educational system. At the elementary school level, this support can be obtained by having special education teachers working in the classroom with the child. This teacher works in close collaboration with the other teachers and the parents. At the secondary school level, an inclusion commission advises adaptations for the student's needs such as technological or person support in class and develops an individualized education program in consortium with the teachers and parents when the student is not able to follow the regular academic program rhythm. With a restructuring of the Special Education System, the *Institut* will change its name to *Centre pour le développement des enfants et jeunes présentant un trouble du spectre de l'autisme* [Center for the development of children and youth with an autism spectrum disorder] and will become one of eight Specialized Psychopedagogy Competence Centers in Luxembourg. This center will provide a specialized and multidisciplinary educational support with trained professionals to children and youth with autism.

Professional Integration and Training

There are different services in Luxembourg to support the professional development of people with autism. The *Fondation Autisme Luxembourg* offers a small educational farm and five sheltered workshops to those who use their day care centers: the kitchen, the laundry, the garden, the do-it-yourself workshops, and sports. These activities ensure learning retention and development of competences, promote verbal and non-verbal communication, provide a stimulating daily social environment, and a commendable daily work activity. These activities are dedicated to people with autism aged 16 years or older who due to their difficulties and challenging behaviors cannot work in other existing

sheltered workshops. Additionally, the *Fondation* is active in the integration of people with autism in the regular job market by providing support in the process of hiring, providing training to the company, as well as coaching those looking for a job.

Autisme Luxembourg asbl offers professional training for people with autism older than 16 years old, who have finished their compulsory school or who have difficulties finding a job due to their needs. An individualized training program is established based on the needs of the persons and prepares them for a professional activity. The training lasts 2–3 years and aims at the acquisition and improvement of personal skills, autonomy, as well as teaching appropriate attitudes and behavior during work. The training prepares people with autism for an active work life either in a sheltered environment or in the regular job market. *Autisme Luxembourg asbl* also offers different sheltered workplaces for people with autism who can work in a variety of domains such as the development of books and mobile applications, digitalization of printed documents, printing shop, gardening, art and ceramics, cooking, etc. People with autism who integrate the regular job market can also receive assistance to find and keep a job from *Autisme Luxembourg asbl* through their ambulatory intervention center. This service aims at providing social support on an as-needed basis to individuals with autism who live independently or semi-independently.

Leisure Activities

Regarding leisure activities, both the *Fondation Autisme Luxembourg* and *Autisme Luxembourg asbl* offer adapted activities to people with autism of different ages. The *Fondation Autisme Luxembourg* offers on a daily basis during the entire year extracurricular activities for children. These include indoor activities such as crafts, cooking, individual desk-based learning, sensory space, games, and relational games or outdoors activities such as walks, swimming, visits to parks, and attendance to shows. The *Fondation* also organizes regular holiday camps for people with autism of all ages (children, adolescents, and adults) throughout the entire year.

In their day care center and leisure center, *Autisme Luxembourg asbl* offers adults with autism who are not able to exercise any professional activity different occupational activities such as games and puzzles, sportive activities such as walks and swimming, socio-pedagogical and therapeutic activities such as snoezelen and musical sessions, as well as regular outings.

Assistance

Social, physical, and mental health assistances are provided by the *Fondation Autisme Luxembourg* throughout their different services such as the support service (see “[Treatment and Support](#)” section).

Autisme Luxembourg asbl provides a home help service to keep people with autism in contact with others and to help them build a social network of support with the aim of maximizing their autonomy and well-being. They also provide a care and health service that helps centralize their information and manage the contact between the person with autism, their families, and different health-care providers (dentist, psychiatrist, physical therapist, psychotherapist, etc.). Finally, a consultation service is also offered which is composed of a multidisciplinary team that develops interventions in the form of consultations, interviews, individual therapy sessions, group therapy sessions, information sessions, and work with the families.

Housing

Currently, the *Fondation Autisme Luxembourg* has two residential centers for people with autism. One of the centers has three homes that accommodate 20 people with autism aged from 20 to 65 years living in small groups. The other residence accommodates 24 people with autism presenting with different levels of independence, living in three groups of 6–7 people each. In this center, one of the homes is generally dedicated to individuals aged 20–60 years, another home is specifically dedicated to elderly people who have reduced mobility, and the other is specifically dedicated to younger people with autism

such as adolescents. Both centers have permanently at least two respite care rooms for temporary admissions to meet the needs of families when required and for emergency cases when needed. This service is available for people with autism of all ages and levels of abilities regardless of their degree of difficult behaviors.

Autisme Luxembourg asbl offers three types of housing support for adults with autism. They have three centers for supported housing in which residents live autonomously and the association insures through their home help service that the person stays in contact with others in their community and neighborhood, helping their integration, autonomy, and well-being according to their abilities. *Autisme Luxembourg asbl* also has a residence for five people who either work in their sheltered workshops or who attend the day care center. The stay at this residence is intended as a transition period where residents are prepared to a more autonomous life where they can manage their budget and take care of themselves and the place where they live. Finally, *Autisme Luxembourg asbl* also has a residence for substantially dependent people with autism. That residence houses ten people who need a close socio-pedagogical support in their daily life activities. The residence also includes one respite care unit for emergency cases.

Other Non-autism Institutions

In addition to the different services that are dedicated exclusively to people with autism, there are also other institutions and services in Luxembourg dedicated to support people with educational difficulties or disabilities, who can provide help to those with autism. In the support of young children, the *Service de Rééducation Précoce – Hëllef fir de Puppelchen* [Early re-Education Service – Help for the Babies] provides services of observation, assessment, screening, guidance, treatment, and reeducation to children up to the age of 4 years. Their multidisciplinary team offers physical, occupational, and speech therapy as well as playgroups to improve children’s autonomy and psychosocial abilities. The *Service d’Intervention Précoce Orthopédagogique*

[Service of Early Intervention and Orthopedagogy] provides services to children up to the age of 6 years on psychological assessment and testing of abilities, difficulties, and delays, as well as socio-pedagogical and therapeutic support, and psychological interventions for the children and their parents. Additionally, parents can seek treatment, therapy, and support, as well as diagnostic advice on autism from private sector professionals such as pediatricians, psychiatrists, and psychologists.

Associations such as the *Tricentenaire asbl*, the *Association de Parents d'Enfants Mentalement Handicapés* [Association of Parents of Mentally Handicapped Children], the *Ligue HMC*, or companies such as *elisabeth* that act in the social domain provide therapeutic support, day care centers, leisure activities, housing, professional training, and sheltered work structures to disabled people. Finally, *Info-Handicap Luxembourg* informs, orients, and provides support to disabled people, their families, and professionals. The service also provides legal assistance to support people in the access to services, to protect their rights, and to assist them in case of discrimination.

Overview of Research Directions

A research group conducts research on autism at the *Institute for Health and Behavior* at the *University of Luxembourg*. The research is centered on understanding different aspects related to the emotional difficulties of children with autism (e.g., Costa et al. 2017c) and the link between these to the development of internalizing and externalizing problems as well as to difficulties within the family (e.g., Costa et al. 2017a). The group is currently working on the development of technology-based interventions to teach emotional ability to children with autism (e.g., Costa et al. 2017b). Additionally, other works such as Bachelor's and Master's theses about autism have been conducted in collaboration with the University of Luxembourg as well as with other institutions in Luxembourg.

Overview of Training

The *Fondation Autisme Luxembourg* provides training to professionals working with people with autism as well as to parents and interested persons. The *Fondation* also offers the "Earlybird" training under the license of the National Autistic Society from the United Kingdom, to parents of children with autism under the age of 6 years. Training sessions are organized on a regular basis and are provided either at the *Fondation* or in the framework of the trainings offered by the *Institut de Formation de l'Éducation Nationale* [Training Institute from the National Education]. The *Fondation* also acts as a training platform for people learning to become social workers and the trainings provided by the *Fondation* are governmentally recognized. Additionally, the *Fondation* organizes regular parent-professional meetings where parents are informed of state-of-the-art practices and advances related to autism and where parents can ask questions and exchange information with professionals. In these meetings, people with autism without intellectual disability often take part as trainers or facilitators of the training sessions. In addition, the *Unité de Formation et d'Éducation Permanente* [Training and Permanent Education Unit] also offers training to professionals working with people with disabilities, specifically in the social and support domains.

Social Policy and Current Controversies

Currently, the main challenges about autism in Luxembourg are related to parents' and professionals' concerns regarding a more comprehensive and clearly structured care of people with autism. The services have substantially increased since the 1980s and different structures now exist to support people with autism and their families. However, some aspects still need to be considered in order to further develop the offered support and services.

One of the challenges, from which some of the others stem, is a need for an overview

regarding the situation of autism in the country. Autism prevalence and incidence in Luxembourg is unknown and a comprehensive system reporting the education and care that diagnosed people receive is still to be developed. Related to that issue, is a difficulty in the concertation of efforts among the different institutions dedicated to autism. Institutions cooperate among themselves to provide the better care for the person and the family but the absence of a national plan for autism, as is the case for many European countries and is the case for other societal challenges in Luxembourg such as suicide prevention and rare diseases, makes it difficult for the institutions to develop a long-term service adapted to the real needs of the country and coordinate their efforts.

Additionally, a common problem faced by the different institutions is a lack of resources to deal with the increasing number of people requesting a diagnosis and the diagnosed who need care and adapted structures. Even though several structures exist, they are not sufficient for all those requiring support. There is a long waiting list for diagnosis and an early diagnosis, even though given priority, cannot always be guaranteed. This hinders the implementation of early interventions which are a determinant factor to the outcomes of the child and the family. Furthermore, there are no intensive intervention programs dedicated to work on children's abilities that can foster their development and increase the chances of better life outcomes and well-being. There are also a limited number of structures and places that provide adapted support, care, and accommodation to people with autism which cannot answer to all the requests. For instance, a living structure able to accommodate children and adolescents with autism with severe behavior problems, which can cause significant distress and loss of economic power to the family due to the need of a parent remaining constantly at home, is still inexistent.

In Luxembourg, different services, structures, and measures have been developed in the past decades that had an impact in improving the lives and well-being of people with autism and their families. Nowadays, the country strives to

continue to provide quality services that are accessible to all people with autism and their families. To achieve this and answer to the needs of Luxembourg's growing population more services as well as the scope of the existing ones need to be expanded.

References and Reading

- Association des Parents de Personnes Atteintes d'Autisme de Luxembourg (APPAAL). www.appaal.lu.
- Autisme Luxembourg Asbl. www.autisme.lu.
- Autism-Europe. (2017) <http://www.autismeurope.org/>.
- Central Intelligence Agency. (2017). *The world factbook – Luxembourg*. Washington, DC: Central Intelligence Agency. <https://www.cia.gov/library/publications/the-world-factbook/geos/lu.html>.
- Centre Hospitalier de Luxembourg – Centre de jour pédopsychiatrique et Unité Autisme. <https://kannerkliniek.chl.lu/fr/service/pedopsychiatrie-unite-autisme>.
- Costa, A. P., Steffgen, G., & Ferring, D. (2017a). Contributors to well-being and stress in parents of children with autism spectrum disorder. *Research in Autism Spectrum Disorder*, 37, 61–72.
- Costa, A.P., Steffgen, G., Lera, F., Nazarihorram, A., & Ziafati, P. (2017b, March). Socially assistive robots for teaching emotional abilities to children with ASD. In Paper presented at the 3rd workshop child-robot interaction at human-robots-interaction. Conference location: Vienna, Austria.
- Costa, A. P., Steffgen, G., & Samson, A. C. (2017c). Expressive incoherence and alexithymia in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(6), 1659–1672.
- Fondation Autisme Luxembourg. (2017). *Rapport d'Activités 2016*. Retrieved from <http://www.fal.lu/files/73635.pdf>.
- Fondation Autisme Luxembourg (FAL). www.fal.lu
- Institut pour Enfants Autistiques et Psychotiques (IEAP). www.ediff.lu.
- Law from 14 March 1973 on the creation of instituts and services of special education. <http://legilux.public.lu/eli/etat/leg/loi/1973/03/14/n1/jo>.
- Law from 11 August 1982 on the rights of incapacitated adults. <http://legilux.public.lu/eli/etat/leg/loi/1982/08/11/n3/jo>.
- Law from 28 June 1994 amending and completing the modified law of 14 March 1973 on the creation of instituts and services of special education; favoring the participation of disabled children in the mainstream education and their educational integration. <http://legilux.public.lu/eli/etat/leg/loi/1994/06/28/n1/jo>.
- Law from 18 December 1998 on the definition of the rules to determine dependence. <http://legilux.public.lu/eli/etat/leg/rgd/1998/12/18/n9/jo>.

- Law from 12 September 2003 on the work of disabled people. <http://legilux.public.lu/eli/etat/leg/loi/2003/09/12/n1/jo>
- Law from 23 December 2005 modifying different dispositions in the social insurance code regarding the dependence care insurance. <http://legilux.public.lu/eli/etat/leg/loi/2005/12/23/n2/jo>
- Law from 16 December 2011 on the amendment to the modified law of 12 September 2003 regarding the work of disabled people. <http://legilux.public.lu/eli/etat/leg/loi/2011/12/16/n11/jo>
- Ministère de l'Education nationale, de l'Enfance et de la Jeunesse – Service de l'Education différenciée. www.men.public.lu
- Ministère de l'Education nationale, de l'Enfance et de la Jeunesse – Élèves à besoins éducatifs spécifiques ou particuliers. <http://www.men.public.lu/fr/themes-transversaux/eleves-besoins-specifiques/index.html>
- Ministère de la Famille, de l'Intégration et à la Grande Région. www.mfi.public.lu
- Ministère de la Sécurité Sociale – Cellule d'Evaluation et d'Orientation; Assurance dépendance. www.mss.public.lu/dependance
- Service d'Intervention Précoce Orthopédagogique. <http://www.sipo.lu>
- Service de Rééducation Précoce – Hëllef fir de Puppelchen. <http://www.srp.lu>
- Unité Autisme. (2017). *Rapport Succinct d'Activités de l'Unité Autisme 2016*. Luxembourg: Internal, unpublished report from Service National de Pédiopsychiatrie du Centre Hospitalier du Luxembourg.
- Unité de Formation et d'Education Permanente. <http://www.ufep.lu/>
- Université du Luxembourg – Institute for Health and Behavior. https://wwwde.uni.lu/recherche/flshase/inside/research_institutes/health_and_behaviour

Lyodrin

- [Epinephrine](#)

Lysosomal Lipid Storage Disorder

- [Gangliosidoses](#)

MABC

► [Movement Assessment Battery for Children: Second Edition \(MABC-2\)](#)

MABC-2

► [Movement Assessment Battery for Children: Second Edition \(MABC-2\)](#)

MacArthur-Bates Communicative Development Inventories, Second Edition

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Synonyms

[CDIs](#)

Description

The CDIs (Fenson et al. [2007](#)) are a set of parent-informant measures designed to evaluate the

communicative skills of young typically developing children from their “early signs of comprehension, to their first nonverbal gestural signals, to the expansion of early vocabulary and the beginnings of grammar” (Fenson et al., p. 7). To accomplish this, two separate forms were developed. These are the *CDI: Words and Gestures* and the *CDI: Words and Sentences*.

The *CDI: Words and Gestures* is designed for typically developing children ages 8–18 months as a measure of emerging receptive and expressive vocabulary and the use of communicative or symbolic gestures. It has two major parts. Part I, Early Words, is divided into four sections. Section A, First Signs of Understanding, includes three yes-no items (e.g., “[Does your child] respond when name is called?”) intended to determine whether the child has started to respond to language. Section B, Phrases, is a 28-item checklist designed to tap the understanding of everyday language in the context of interactional routines (e.g., “Don’t touch”). Section C, Starting to Talk, consists of two items to assess the frequency (i.e., never, sometimes, often) with which a child imitates words and labels objects. Section D, the largest section, is called the Vocabulary Checklist. It is a 396-item checklist organized into 19 semantic categories. Ten of these reflect different noun classes, but a variety of other categories are also represented (e.g., verbs, adjectives, pronouns, quantifiers). For each item, the respondent indicates whether the child “understands” and/or “understands and says.”

Part II, Actions and Gestures, is divided into five sections. Section A, First Communicative Gestures, includes 12 items to assess the frequency (i.e., never, sometimes, often) of nonverbal communicative intentions (e.g., reaching, pointing, nodding). Section B, Games and Routines, is comprised of six yes-no items (e.g., “playing peekaboo”). Section C, Actions with Objects, uses 17 yes-no items (e.g., “eat with a spoon or fork”). Section D, Pretending to be a Parent, includes 13 yes-no items where respondents indicate the kinds of actions a child engages in during pretend play with a stuffed animal or doll (e.g., “Kiss or hug it”). Section E, Imitating Other Adult Actions, includes 15 yes-no items (e.g., “wash dishes”) intended to assess the child’s attempts to simulate the actions of adults using real or toy implements. A table summarizing the content of the *CDI: Words and Gestures* form is presented in Table 1.

Unlike the *CDI: Words and Gestures* form which is designed to evaluate receptive and expressive language, the *CDI: Words and*

Sentences form is intended to assess expressive language only. It is designed for typically developing children ages 16–30 months as a measure of developing expressive vocabulary and a number of aspects of early grammar development. Part I, Words Children Use, is divided in two sections. Section A, Vocabulary Checklist, is a 690-item checklist organized into 22 semantic categories. The categories are similar to those used in the CDI: Words and Gestures form except some categories are further divided and two categories are added (i.e., helping verbs and connecting words). For each item, respondents simply indicate those items the child “says.” Section B, How Children Use Words, includes five items to assess the frequency (i.e., never, sometimes, often) with which a child talks about things and people that are not in the here and now (e.g., talking about an absent toy, pet, or person). Part II, Sentences and Grammar, is divided into five sections. Section A, Word Endings/Part 1, uses four items to assess the frequency (i.e., not yet, sometimes, often) with which the child produces early emerging morphemes that

MacArthur-Bates Communicative Development Inventories, Second Edition, Table 1 Communicative development inventory: words and gestures

	Description	Example
<i>Part I: early words</i>		
A. First signs of understanding	General items about early comprehension of familiar words and phrases (3 items)	Child responds to his/her name
B. Phrases	Comprehension of everyday phrases and routines (28 items)	Responding to “don’t touch”?
C. Starting to talk	Imitation and labeling (2 items)	i.e., imitating words and naming objects
D. Vocabulary checklist	A 396-item checklist organized into 19 semantic categories with separate columns for comprehension and production	Vroom, moo (sound effects and animal sounds), bear (animals names), ball (toys)
<i>Part II: actions and gestures</i>		
A. First communicative gestures	Nonverbal communicative and intentional behaviors (12 items)	Reaching, pointing, nodding
B. Games and routines	Engagement in games/interactional routines (6 items)	Playing peekaboo
C. Actions with objects	Acting upon objects in conventional ways (17 items)	Eating with a spoon or fork?
D. Pretending to be a parent	Pretending to care for stuff animal or doll in a parental manner (13 items)	Kissing or hugging a doll
E. Imitating other adults actions	Attempts to simulate adult activities with real or toy implements (15 items)	Washing dishes

appear at the end of words (i.e., plural -s, possessive -s, present progressive -ing, regular past tense -ed). Section B, Word Forms, is a checklist of 25 items intended to tap production of irregular nouns (e.g., children) and verbs (e.g., ate). Section C, Word Endings/Part 2, is a 31-item checklist of overregularized nouns (e.g., child) and verbs (e.g., ated) which occur naturally in children's speech and indicate increasing linguistic sophistication. A single question (combining) then asks whether the child has begun to combine words (not yet, sometimes, often). Section D, Examples, is intended to provide a basis for estimating mean length of utterance (MLU). Here, the respondent is asked to write three examples of the longest sentences that the child has said recently. Section E, Complexity, is a 37-item checklist designed to assess syntactic complexity. Each of the 37 items is comprised of a pair of sentences that contrast in complexity (e.g., "two shoe" versus "two shoes," "turn on the light" versus "turn on the light so I can see"). Using a forced-choice

format, respondents are asked to judge which sentence in each pair sounds most like the way their child talks. A table summarizing the content of the *CDI: Words and Sentences* form is presented in Table 2.

Detailed administration and scoring procedures (with examples) are provided in the technical manual, and comprehensive descriptive data are presented for all CDI sections. Separate percentile rank data for boys and girls using 1-month intervals are provided for all sections except two sections of the CDI: Words and Gestures form (i.e., First Signs of Understanding, Starting to Talk) and two sections of the CDI: Words and Sentences (i.e., How Children Use Words, Word Endings/Part 1). When percentiles are not provided, the percentage of affirmative answers is calculated for comparison with the percentages given in the manual. When percentiles are provided, they can be unstable. Specifically, restriction in the range of scores sometimes occurred for ages at which the skills in question were just

MacArthur-Bates Communicative Development Inventories, Second Edition, Table 2 Communicative development inventory: words and sentences

	Description	Example
<i>Part I: words children use</i>		
A. Vocabulary checklist	A 680-item checklist organized into 22 semantic categories with column for production only	Moo (sound effects), tiger (animals), coat (clothing), when (question words)
B. How children use words	Use of language to refer to the past, future, and absent people and objects (5 items)	Talking about an absent toy, pet, or person
<i>Part II: sentences and grammar</i>		
A. Word endings/part 1	Use of plural -s, possessive -s, present progressive (-ing), and regular past tense (-ed) (4 items)	Shoes, Daddy's, running, opened
B. Word forms	A list of irregular plural nouns (5 items) and irregular past tense verbs (20 items)	Children, men, fell, heard
C. Word endings/part 2	A list of overregularized nouns (14 items) and overregularized verbs (31 items)	Foots, ated
Combining	A question about whether the child has begun to combine words (1 item)	i.e., Has the child begun to combine words?
D. Examples	A request to write examples of the three longest sentences that the parent has heard the child say in the recent past (basis for the mean length of utterance)	n/a
E. Complexity	Sentence pairs that contrast in length and grammatical complexity (37 items pairs); asks the parent to indicate which one sounds most like the way the child currently talks	Go bye bye versus wanna go bye bye

emerging. When this happens, small differences in raw scores can have dramatically different effects on percentile ranks. As Fenson et al. (2007) explain:

Because relatively few items can have such a large effect on the child's percentile scores in these instances, users should be very cautious in drawing strong conclusions based on these reported values. (pp. 33–34)

The original and expanded versions of a Basic Information Form are included in the CDI. The forms are designed to gather demographic information useful in interpretation of test scores. The expanded version also contains several questions regarding child health history. Each CDI form takes between 20 and 40 min to complete. It should be noted that CDI short forms are available (Fenson et al. 2000). Although these may be useful to researchers and professionals who seek a quick assessment of early language, the short forms likely lack the precision of the full CDIs and must be administered and interpreted with caution (Fenson et al. 2007). Members of the CDI advisory board have developed Mexican Spanish versions (including short forms) of the CDIs (Jackson-Maldonado et al. 2003). The measures have also been developed in a number of languages other than English by other researchers. A comprehensive list of these versions is available on the CDI web site (<http://www.sci.sdsu.edu/cdi/adaptations.htm>).

Historical Background

As Fenson et al. (2007) explain, the first systematic attempts to use questionnaires to tap parents' knowledge of their children's language skills were conducted by Elizabeth Bates (a codeveloper of the CDIs) and colleagues in the 1970s and 1980s (e.g., Bates et al. 1975). These inventories were later refined to tap parents' knowledge of their children's vocabulary and grammar and, along with the work of another prominent researcher in the area (Leslie Rescorla), laid the foundation for the development of the CDIs (Fenson et al. 2007). The CDI is an example of a measure that relies exclusively on parent report. It has endured the

test of rigorous scientific scrutiny, and there is now tremendous evidence in support of its reliability and validity. As a parent-informant measure, the CDI is especially important as parent-reports can provide a more representative sample of language than may be obtained in clinical settings where spontaneous speech or test results may be influenced by characteristics of the child (e.g., shyness) or testing situation (e.g., familiarity with the examiner) (Fenson et al.). For all of these reasons, the CDI has become a well-respected measure, which continues to lend support to the notion that parents are valuable, reliable, and accurate sources of information. It has been particularly welcomed from a family-centered perspective (e.g., Westby 1998) where parents and other caregivers are recognized as valuable partners in the assessment process.

Psychometric Data

The *CDI: Words and Gestures* is normed on a sample of 1,089, and the *CDI: Words and Sentences* is normed on a sample of 1,461 with a relatively even distribution of girls and boys across the age ranges sampled. Although these 2007 expanded norms more closely approximate national demographic statistics than the original 2,000 norms, the developers note that the sample still underrepresents children and families with low educational and/or socioeconomic backgrounds. In addition, Caucasians are slightly overrepresented, whereas Hispanics are underestimated (a likely result of exclusion criteria requiring children to be from a home where English was the primary language). Normative data for birth order and exposure to a second language are comparable to the US census statistics.

Two types of reliability of the CDIs are well documented. Internal consistency, or the degree to which items on a test measure the same content domain, indicates a very high degree of consistency. The test-retest reliability over an approximate 1–2-month interval is also high indicating excellent stability over time.

The validity of the CDIs is similarly well established. First, the CDIs demonstrate good

content validity, which is the degree to which the test covers the intended content domain. Indeed, the content of the CDIs was developed in line with the language development literature, is supported by over 30 years of child language research, and has been credited as adequately sampling early vocabulary and grammar development (e.g., Westby 1998). Concurrent validity, based on the relationship between CDI scores and various child performance measures, is particularly impressive. Moderate to high correlations have been reported across numerous studies on typically developing children as well as children with language impairment and developmental delays. For example, sections of the CDI are significantly correlated with standardized tests like the *Bayley Expressive Language Scales*, the *Peabody Picture Vocabulary Test*, and the *Expressive One-Word Vocabulary Test* as well as language sample measures like the *Index of Productive Syntax* (Scarborough 1990) and Number of Different Words (NDW).

Clinical Uses

The CDIs were normed on a sample of young typically developing children, and they are not intended to screen for, or identify children with, autism spectrum disorder (ASD). On the other hand, the evaluation of language is important in the assessment of young children with ASD who often demonstrate qualitative impairments in language as well as language delay. In this light, the CDIs have gained utility as a clinical tool in ASD when supplemented by direct measures of child performance, and evidence of the accuracy of the CDIs for screening (Filipek et al. 1999) and diagnosing ASD (e.g., Luyster et al. 2007) is accumulating.

The inventories are well suited as one component of a broader screening procedure (Klee et al. 1998) for language delay, which typically includes the criterion that a child of 24 months uses fewer than 50 words. Experience suggests that this criterion is commonly applied to young children at risk for ASD through use of the CDIs. In a related vein, research shows that deficits in both receptive and expressive language are more

predictive of language impairment than expressive language deficits alone (e.g., Thal et al. 1991). Because the inventories tap both receptive and expressive vocabulary, they can facilitate this analysis with the understanding that no single measure should ever be used as a basis for identification or assessment (Fenson et al. 2007). The use of the CDIs as an evaluation and assessment tool is reinforced by the finding that it has been found to discriminate children with language impairment from those who are developing typical language (Skarakis-Doyle et al. 2009).

Charman et al. (2003) systematically examined the language profiles of preschoolers diagnosed with ASD using the CDIs. Despite considerable variability, children with ASD demonstrated the expected receptive and expressive language deficits described above. Compared to the typical pattern, however, the word production of children with ASD was relatively in advance of word comprehension. With regard to gestures, later developing gestures (pretending to be a parent) were relatively in advance of early developing gestures (e.g., reaching, pointing), which involve joint reference and direct social interaction. Studies such as these are important because they demonstrate how the CDI can be useful in clinical work with young children with ASD (Charman et al. 2003).

Fenson et al. (2007) report that the CDIs are useful for documenting the language abilities of older children representing special populations, including ASD, and they have become increasingly popular for that purpose. Of course, successful implementation of the inventories to assess the language development in children with ASD requires that the scores obtained never exceed the developmental level tapped by the CDI (i.e., 30 months). The developers of the CDI are clear that when scores of older children from special populations exceed this level, serious misjudgments of communicative skills can occur due to ceiling effects.

The CDIs have also been used in the development of intervention strategies. Fenson et al. (2007) suggested that the measure be used to identify overall receptive vocabulary deficits but not to identify specific words that are or are not understood for remediation. They further note that

the case may be different for productive vocabulary where specific words are unambiguously present or absent. “Clinicians may wish to use the CDIs to identify classes of words that appear to require remediation and supplement these findings with more targeted behavioral assessments of individual words” (p. 44).

In summary, the CDIs have proven to be clinically useful for assessment of language deficits, which represents a major characteristic of ASD. When combined with direct measures of child performance, it can be used to screen and evaluate the presence of a language deficit. It can also be used to document the language abilities of children with ASD and guide intervention efforts. As noted above, the CDIs may be particularly attractive because, unlike formal child performance tests, they are not influenced by child situational or motivational factors. Instead, they rely on parent knowledge that has accumulated over time during which the parent has had the opportunity to observe and form impressions about the child’s skills.

Of course, each clinical application raises questions to consider when determining the appropriateness of the measure and interpretation of scores. In particular, use of the CDI norms with children whose developmental age exceeds the upper limit of the inventories is not recommended. It is also prudent to recall that percentile data for some sections of the CDI are unstable, and so interpretation of all percentile data requires care.

See Also

- [Age Appropriate](#)
- [Age Equivalents](#)
- [Communication and Symbolic Behavior Scale](#)
- [Communication Assessment](#)
- [Developmental Delay](#)
- [Early Language Milestone Scale](#)
- [Expressive Language](#)
- [Infant/Toddler Checklist](#)
- [Language Tests](#)
- [Mullen Scales of Early Learning](#)
- [Peabody Picture Vocabulary Test](#)
- [Prelinguistic Communication Assessment](#)
- [Preverbal Communication](#)
- [Receptive Vocabulary](#)
- [Rossetti Infant-Toddler Language Scale](#)
- [Screening Measures](#)
- [Validity](#)
- [Vocabulary](#)

References and Reading

- Bates, E., Camaioni, L., & Volterra, V. (1975). The acquisition of performatives prior to speech. *Merrill-Palmer Quarterly*, 21(3), 205–226.
- Charman, T., Drew, A., Baird, C., & Baird, G. (2003). Measuring early language development in preschool children with autism spectrum disorder using the MacArthur communicative development inventory (infant form). *Journal of Child Language*, 30, 213–236.
- Dale, P. S. (1991). The validity of a parent report measure of vocabulary and syntax at 24 months. *Journal of Speech, Language, and Hearing Research*, 34, 565–571.
- Fenson, L., Pethick, S., Renda, C., Cox, J. L., Dale, P. S., & Reznick, J. S. (2000). Short-form versions of the MacArthur communicative development inventories. *Applied Psycholinguistics*, 21(1), 95–116.
- Fenson, L., Marchman, V. A., Thal, D. J., Dale, P. S., Reznick, J. S., & Bates, E. (2007). *MacArthur-Bates communicative development inventories* (2nd ed.). Baltimore: Paul H. Brookes.
- Filipek, P. A., Accardo, P. J., Baranek, G. T., Cook, E. H., Dawson, G., Gordon, B., et al. (1999). The screening and diagnosis of autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 29(6), 439–484.
- Jackson-Maldonado, D., Thal, D. J., Fenson, L., Marchman, V. A., Newton, T., & Conboy, B. (2003). *MacArthur Inventarios del Desarrollo de Habilidades comunicativas users guide and technical manual*. Baltimore: Paul H. Brookes.
- Klee, T., Carson, D., Gavin, W., Hall, L., Kent, A., & Reece, S. (1998). Concurrent and predictive validity of an early language screening program. *Journal of Speech, Language, and Hearing Research*, 41, 627–641.
- Luyster, R., Qui, S., Lopez, K., & Lord, C. (2007). Predicting outcomes of children referred for autism using the MacArthur-Bates communicative developmental inventory. *Journal of Speech, Language, and Hearing Research*, 50, 667–681.
- Scarborough, H. (1990). Index of productive syntax. *Applied Psycholinguistics*, 11(1), 1–22.
- Skarakis-Doyle, E., Campbell, W., & Dempsey, L. (2009). Identification of children with language impairment: Investigating the classification of the MacArthur-Bates communicative development inventories, level III. *American Journal of Speech-Language Pathology*, 18, 277–288.

- Thal, D., Tobias, S., & Morrison, D. (1991). Language and gesture in late talkers: A 1 year follow-up. *Journal of Speech & Hearing Research*, 34(3), 604–612.
- Westby, C. (1998). Review of the MacArthur communicative development inventories. In J. C. Impara & B. S. Plake (Eds.), *The thirteenth mental measurements yearbook* (pp. 631–632). Lincoln: The Buros Institute of Mental Measurements.

Macedonia and Autism

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Historical Background

The Republic of Macedonia has gone through a long and painful process of transition during last 25 years. The cumulative effect of the transitional recession and the socially stressful process of transition have most seriously affected marginalized groups, such as persons with autism spectrum disorders being among the most vulnerable (Trajkovski 2008).

Autism in Macedonia has a short history. It is not like the history of autism in the USA or other western developed countries. For many decades, especially during the communist period of Yugoslavia, children with autism were diagnosed as mentally retarded, schizophrenic, or some other diagnosis. Children and adults with autism missed regular treatment because of inadequate institutions, lack of finances, parental ignorance, and prejudices in the society. Many of the children with autism have been placed into big institutions such as the Special Institute in Demir Kapija. Because of the poor economic situation, low standard and inappropriate services of social assistance; families were no longer able to care for the medical, physical, and psychological needs of their children and found institutions to be some alternative. Macedonia also has a history

of reporting fewer cases of ASD than can reasonably be assumed to exist nationally, indicating the probable existence of cultural biases or other errors in diagnosis (Trajkovski et al. 2005). So, there are no reliable data about the statistics on how many children with ASD are diagnosed. A National Register of persons with ASD is not established yet.

The first case study about child with autism was described more than three decades ago (Kopachev and Nastov 1983). Then, after 13 years without any activities from the governmental or nongovernmental sector, the first parent association was established in 1996. The Society started its activities and was very productive for 2 years. Children were included for treatment in special schools and institutions for rehabilitation in Skopje. Due to a lack of interest, the society stopped its activities in 1998 (Ajdinski 2005). The author of this entry started with the first doctoral thesis about autism entitled: “Immunogenetic analyses in persons with autism in Republic of Macedonia” in November 1999. Following the research and treatment of autism in the world, the Institute for Special Education and Rehabilitation at the Faculty of Philosophy and the Institute for Immunobiology and Human Genetics at the Faculty of Medicine took the initiative and the Macedonian Scientific Society for Autism (MSSA) was established as an NGO in May 2000. MSSA directed its work towards: primary prevention of people with autism, research on the mechanisms referring to autism, research on the effects of rehabilitation of people with autism, treatment of people with autism, as well as coordination with both governmental and nongovernmental organizations and cooperation with international organizations dealing with autism. In a very short time, the NGO established the number and presented a survey of families with autistic people and started to prepare the National register, introduced two web sites on the Internet (Autism-Macedonia and a web site of the association), became a member organization of Autism Europe, and published a brochure about people with autism. Its members started to participate in scientific projects, attending at conferences in the country and abroad, and had

presentations on TV and radio broadcasting. Their professional and scientific works were published in scientific journals.

There is a lack of knowledge on ASD among professionals who are involved in work with children with ASD, a lack of standardized protocols for early detection, diagnosis, and assessment tools. The precarious use of international classifications and diagnostic tools are resulting in low and late detection of autism. Officially in Macedonia, the ICD-10 diagnostic manual is still used in the health care system (ICD, 1992).

In spite of our lobbying activities during last 17 years, there is still no national strategy about autism spectrum disorders in the country.

Legal Issues, Mandates for Service

Article 39 from the Constitution of the Macedonia guarantees that every citizen has the right to health care. Citizens have the right and the duty to protect and promote their own health and the health of others. Article 44 grants the right to education for every citizen. Education is accessible to everyone under equal conditions. Primary education is compulsory and free of charge. According to the Law for elementary education, special educational plans and curriculum are required to nurture and educate pupils with special educational needs.

The Macedonian constitution does guarantee access to health care to its citizens, but local health care providers are not adequately equipped to serve individuals with autism spectrum disorders. Among other issues, there is limited neonatal screening for genetic disorders, the nation lacks a centralized database on disabilities, and the quality of health care in urban centers does not extend to rural areas. Macedonian schools do not have appropriate staff to work with children with autism according the research conducted by the state attorney office in schools in several towns in Macedonia. It showed 93 children with ASD are going to 109 primary schools. The data analyzed by the state attorney office show that only a small number of the schools have a completely professional team which would work with autistic children. Of all 109 schools covered in the survey, only 39% have a professional team comprised of a

teacher, a psychologist, a pedagogue, and a special education teacher. On the other hand, 28% of the schools have no staff whatsoever to work with children with ASD, nor have the parents hired at their own expense a special education teacher who would work with those children (www.independent.mk).

Social institutions on the local level are not capable of ensuring that people with autism can remain in their area of residence and be provided with adequate educational and social services. The lack of statistical data on people with a disability hampers an evaluation of whether all persons with any given disability are socially jeopardized and if they are able to access their rights in the field of social care. The Law on social protection established measures and services in the field of social protection and care through social prevention, de-institutional care, and protection and the right for social support (Trajkovski 2008).

The Republic of Macedonia ratified the UN Convention on the Rights of Persons with Disabilities and its Optional Protocol on 29 December 2011 and it is a step forward in improving the quality of life of people with ASD in Macedonia. The diagnosis of autism or other developmental disabilities does not entitle the individuals or their families to benefits – they have to apply for the disability status.

Overview of Current Treatments and Centers

In the Republic of Macedonia, there is no systematic approach to early intervention services and programs. There are no service providers for the early intervention of children with ASD. Home-based interventions and other social services available in Western Europe and the United States are relatively limited (Trajkovski 2010). Parents access various kinds of treatments for their children, the most popular are training of social skills, psychomotor re-education, biomedical treatment, and treatment with medicaments. Sometimes they use unproven treatments which can be dangerous for the health. The use of ABA and TEACCH is at an incidental level. Macedonian pediatricians,

psychologists, and rehabilitators lack the skills to conduct such early kinds of treatments. There is no effective law in Republic of Macedonia for early intervention practices (Trajkovski and Jurtoski 2016).

According to the Constitution of the Republic of Macedonia, the concept of freedom and rights is embedded in a citizen-liberal democratic concept, which begins with the principle of citizenship. An essential citizenship freedom and right is personal freedom and the right to acquire education, from the elementary to the university level, which ensures not only personal development and the general development of the individual, but also to the ability to contribute to raising the general cultural level of the society in which they live. According to the Law for elementary education, special educational plans and the curriculum are required to nurture and educate pupils with special educational needs. The regional boards of psychology, medicine, and education give recommendations about the conditions which would best serve the child, but the parents are not legally bound to follow them, and they can enroll the child into a school of their choice. Also for education in schools, there is no legislation to guide and support parents into choosing the type of school to send their children to. Great numbers of parents send their children to mainstream schools with a 1:1 specialist assistant who is financed by the parents. Having a child with autism spectrum disorders in Macedonia can have a huge financial impact for the family. It appears that policy makers are not interested in this area and have no understanding of the problems families face who take care of children with autism. Authorities have to start with certificated programs for training the professionals. Almost half of parents are not satisfied with early intervention programs in our country. One third of parents spend between 250 and 350 euros on early treatment which is a significant amount of their own budget (Trajkovski and Jurtoski 2016). Some of children with ASD stay at their homes out of the system of special or general education. There are no statistics on this issue.

In last 2 years, the Ministry of Labor and Social Policy opened two daily care centers for children with ASD in two towns: Skopje and Shtip. In the

last 5 years, three more parental associations were created in Skopje, Shtip, and Bitola, but they have quite low impact in the field due to lack of money and lack of knowledge.

The condition of adult people with ASD is very bad. Nobody takes care of them. They do not have access to complementary treatment approaches. Many individuals with ASD who do not receive enough support from their families, especially adults, are institutionalized into neuropsychiatric institutions.

Overview of Research Directions

The first publication about autism from Macedonia in a scientific impact factor journal appeared in 2004 (Trajkovski et al. 2004). Two scientific and clinical handbooks such as *Autism* (Trajkovski 2004) and *Autism and Pervasive Developmental Disorders* (Trajkovski 2011) and some articles are published in Macedonian language and because of that not readily available for international audience (<http://www.mssa.org.mk>). Also, there are a significant number of publications in international peer-reviewed journals, such as those dedicated to the plasma concentration of food-specific antibodies in children with autism (Trajkovski et al. 2008), family analysis of immunoglobulin classes and subclasses in children with autism (Spiroski et al. 2009), and immunogenetics and autism (Trajkovski and Spiroski 2015). Some other research groups have published more socially oriented research, such as sexual development, sexual behavior and gender identity of persons with autism (Mladenovska and Trajkovski 2010), employment of people with autism (Stankova and Trajkovski 2010), stress, coping and support among parents of children with autism (Nolcheva and Trajkovski 2015).

Overview of Training

Only physicians (psychiatrists and pediatricians) are allowed to diagnose autism officially. A wider range of specialists provide educational and rehabilitation services. On the initiative of the writer

of this article, the Institute of Special Education and Rehabilitation at the Faculty of Philosophy introduced the subject of Autism into the curriculum of postgraduate studies in 2004 and the subject of Pervasive Developmental Disorders into the curriculum of doctoral studies in 2012 (<http://www.fzf.ukim.edu.mk>). Twelve graduation theses, six master theses, and two doctoral dissertations were defended with autism as a main topic in last 15 years. Our University “Ss. Cyril and Methodius” trains specialists in special education and rehabilitation and speech pathologists (<http://www.ukim.edu.mk>).

The Macedonian Scientific Society for Autism provides free lectures about autism and specific approaches, such as PECS, TEACCH, and ReAttach method. The organization also participated in an EU-funded project regarding parent and professional education in conjunction with the Apollonia Foundation from Gevgelija. However, the majority of events are on an ad hoc basis and are inaccessible to most families (ESIPP project, 2015). Though parent education programs exist in parts of Europe, in others they are extremely limited or nonexistent. In 2015 the Macedonian Scientific Society for Autism started as a partner in a 3-year project (September 2015–August 2018), funded by the European Commission, in which family members, professionals, and academics from five European countries are working together in a strategic partnership to address this inequity (Belgium, Croatia, Cyprus, Macedonia, and UK). This partnership is seeking to develop a core curriculum and ecologically valid parent education materials and methods, provide parent education to families living with autism in three south-eastern European countries where previously it has been unavailable, evaluate impact using quantitative and qualitative methods, share the curriculum and materials with stakeholders, and make recommendations to decision makers (Preece et al. 2016).

Social Policy and Current Controversies

Our society is not sensitized to those persons with autism spectrum disorders. They are looked down

upon, bullied, and regarded as less valuable than children with typical development. Parents hide their children with autism and do not want to expose them in public. They are ashamed of their own children and are not very eager for them to appear in electronic media. There is a huge social stigma connected to having a person with an ASD diagnosis in the family. The authorities are not interested in helping these families. The governmental bodies do not allocate enough financial resources to this sector. In last 6 years, there have been significant changes in public awareness of autism and in social policy since MSSA started to honor international autism awareness day.

Areas of controversy focus on the role of no established treatments (especially ABA and TEACCH), in best approaches to teaching and service delivery, and issues of diagnostic practice. There is a tremendous dearth of work on adults with ASD. The authorities need to begin to see this disorder as treatable and to invest the same energy, money, and efforts into treating autism that we have put into treating cystic fibrosis, leukemia, childhood cancers, and other chronic medical disorders that affect young children.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [ICD 10 Research Diagnostic Guidelines](#)
- [TEACCH Transition Assessment Profile \(TTAP\)](#)

References and Reading

- Ajdinski, L. (2005). Condition, organization and directions in solving the problem of people with autism in the Republic of Macedonia. *Journal of Special Education and Rehabilitation*, 6(1–2), 43–52.
- Kopachev, D., & Nastov, P. (1983). Autistic child – Our case study. *Macedonian Medical Review*. Skopje, 37(1–2): 30–32.
- Mladenovska, B., & Trajkovski, V. (2010). Opinions and attitudes of parents and students for sexual development, sexual behavior and gender identity of persons

- with autism in the Republic of Macedonia. *Journal of Special Education and Rehabilitation*, 11(1–2), 7–24.
- Nolcheva, M., & Trajkovski, V. (2015). Exploratory study: Stress, coping and support among parents of children with autism spectrum disorders. *Journal of Special Education and Rehabilitation*, 16(3–4), 84–100.
- Preece, D., Trajkovski, V., Symeou, L., Stosic, J., Mavrou, K., Theodorou, E., Troshanska, J., Blazevic, S., Ruzic, A., Baranger, A., Fortuna, R., Capper, A., Charalambous-Darden, N., Winstanley, A., Hardcastle, J., Bramble, P., Kokkinos, A., Fernandez, C., & Frey Skrinjar, J. (2016). Developing ecologically valid parent education for families living with autism in a part of Europe. XI Autism Europe Congress “Happy, healthy and empowered”, Edinburgh, Sept 16–18.
- Spiroski, M., Trajkovski, V., Trajkov, D., Petlichkovski, A., Efinska-Mladenovska, O., Hristomanova, S., Djulejic, E., Paneva, M., & Bozhikov, J. (2009). Family analysis of immunoglobulin classes and subclasses in children with autistic disorder. *Bosnian Journal of Basic Medical Sciences*, 9(4), 283–289.
- Stankova, T., & Trajkovski, V. (2010). Attitudes and opinions of employers, employees and parents about the employment of people with autism in the Republic of Macedonia. *Journal of Special Education and Rehabilitation*, 11(3–4), 16–29.
- Trajkovski, V. (2004). *Autism (handbook in Macedonian)*. Skopje: Faculty of Philosophy.
- Trajkovski, V. (2008). *Study about persons with disabilities in Macedonia*. Skopje: United Nations Development Programme/United Nations Population Fund.
- Trajkovski, V. (2010, May 13). *Health care system for people with intellectual disabilities in Macedonia*. Bristol: International Congress of Best Practice in Intellectual Disability Medicine.
- Trajkovski, V. (2011). *Autism and pervasive developmental disorders (handbook in Macedonian)*. Skopje: Faculty of Philosophy.
- Trajkovski, V., & Jurtoski, F. (2016). Early intervention in children with autism spectrum disorders in Republic of Macedonia. In *Early intervention in Special Education and Rehabilitation-thematic collection of international importance* (Vol. c2016, pp. 139–151), Subotica, Oct 14–16.
- Trajkovski, V., & Spiroski, M. (2015). DNA typing of HLA-A, -C, -B, AND -DRB1 in the children with autism in the Republic of Macedonia. *Bratislavské Lekárske Listy*, 116(1), 14–19. https://doi.org/10.4149/BLL_2015_003.
- Trajkovski, V., Ajdinski, L., & Spiroski, M. (2004). Plasma concentration of immunoglobulin classes and subclasses in children with autism in the Republic of Macedonia: Retrospective study. *Croatian Medical Journal*, 45(6), 746–749.
- Trajkovski, V., Vasilevska, K., Ajdinski, L., & Spiroski, M. (2005). Epidemiological characteristics of autism in Republic of Macedonia. *Journal of Special Education and Rehabilitation*, 6(3–4), 25–39.
- Trajkovski, V., Petlichkovski, A., Efinska-Mladenovska, O., Trajkov, D., Arsov, T., Strezova, A., Ajdinski, L., & Spiroski, M. (2008). Higher plasma cocentration of food-specific antibodies in persons with autistic disorder in comparison to their siblings. *Focus on Autism and Other Developmental Disabilities*, 23(3), 176–185.

Macrocephaly Phenotype and Psychiatric Comorbidity in Autism Spectrum Disorder

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Definition

Macrocephaly is defined as a measurement of the head circumference which is 2 standard deviations (SDs) larger than the mean for a given age and sex (Bray et al. 1969). It is also known as occipitofrontal circumference (OFC), and it denotes the size of the cranium. It is different from megalencephaly, which is defined as increase in the size of the brain parenchyma exceeding twice the standard deviation (Fletcher 1900). Megalencephaly and macrocephaly may coexist in the same individual (Gooskens et al. 1988).

Macrocephaly can be idiopathic, familial, and benign. However, it can be the first indication of autism. In contrast megalencephaly is more often syndromic (Tan et al. 2018).

Evidence shows that the head size correlates with the whole brain volume and weight (Aylward et al. 2002), but it is not caused by hydrocephalus (Filipek et al. 1992; Piven et al. 1995).

The head circumference is a useful index of brain size in children, and it is measured along the most prominent diameter of the occiput and the mid forehead.

Historical Background

The presence of macrocephaly, in some children with autism spectrum disorder (ASD), is a phenotypic characteristic described by Leo Kanner in 1943 (Kanner 1943).

Current Knowledge

Macrocephaly affects children with ASD in a range from 15% to 35% compared to 3–5% in the general population without autism (Courchesne et al. 2001; Aylward et al. 2002; Dementieva et al. 2005; Sacco et al. 2007; Dissanayake et al. 2006; Sacco et al. 2007, 2010), and it is the most common physical sign seen in ASD (Courchesne et al. 2003).

The fact that macrocephaly affects only a subset of children is probably the reason why it is not included as a diagnostic criterion for autism in the DSM-5 or ICD classifications.

Interestingly macrocephaly is absent at birth among some individuals with autism (Dissanayake et al. 2006; Sacco et al. 2010; Gray et al. 2012), which is the result of a higher growth rate of the brain during the first year of life (Froehlich et al. 2013; Constantino et al. 2010; Courchesne et al. 2003), predating the diagnosis of autism (Dawson et al. 2007).

Some studies show that macrocephaly is transitory because after 2 years the growth of the head circumference stops and some children with autism will no longer meet the criteria parameters for macrocephaly (Aylward et al. 2002; Courchesne et al. 2001). However recent evidence shows that there is a subgroup of boys with autism who have brain enlargement persisting until 5 years of age (Libero et al. 2016).

Several studies have proven macrocephaly is familial and heritable. First-degree relatives of children with macrocephaly have larger head sizes compared to first-degree relatives of individuals without autism (Sacco et al. 2007, 2010; Constantino et al. 2010).

Interestingly, macrocephaly in children with autism is apparently not present in all regions of the world such as Sweden, Israel, and Denmark

(Cederlund et al. 2014; Davidovitch et al. 2011; Aagaard et al. 2018), and studies performed with representative samples from the general population have shown the absence of this feature in individuals with autism (Barnard-Brak et al. 2011; Sacco et al. 2015).

Moreover, differences of fetal head growth between the Netherlands and Australia in a recent study show that the Dutch group had a lower initial prenatal head circumference associated with later autistic traits but not the Australian group, suggesting a role for environmental factors (Blanken et al. 2018).

Some recognized triggers affecting the fetal brain growth are the maternal thyroid dysfunction and vitamin D deficiency, which could also lead to autism-like symptoms (Whitehouse et al. 2013; Korevaar et al. 2016; Vinkhuyzen et al. 2018).

In the same lines, a recent study showed that valproic acid-exposed children during the first trimester of pregnancy compared to the unexposed children had a large cephalic index. The large cephalic index was present in valproic acid-exposed children both with and without ASD (Stadelmaier et al. 2017).

The association between macrocephaly and autism severity has been studied with mixed results. Lainhart et al. (1997) demonstrated a small negative correlation between macrocephaly and core symptoms measured through Autism Diagnostic Interview-Revised (ADI-R) but found no association between macrocephaly and head circumference percentile with nonverbal IQ, verbal status, seizure disorder, neurological soft signs, or other minor physical anomalies in individuals with autism.

Fidler et al. (2000) found that macrocephaly was associated with higher scores on the stereotyped behavior domain of the ADI-R. Later on, Lainhart et al. (2006) show an association between social deficits measured through ADI-R and delayed language and macrocephaly in a large collaborative study. Davis et al. (2013) reported an association between higher head circumference and social deficits measured through ADI-R in individuals classified as simplex but not in individuals classified as multiplex. In this study a head overgrowth was identified in 90% of children with

autism from both simplex and multiplex families with a nonverbal IQ < 70 and a nonverbal IQ > 100, respectively, suggesting the importance to include the type of family as a variable with an interaction effect (Davis et al. 2013).

In contrast, a recent study by Webb et al. (2007) found no association between head circumference and signs of language or social regression in children with autism despite using the same definition from the Autism Diagnostic Interview-Revised.

There are less studies between the association of macrocephaly in children with ASD and psychiatric comorbidity. Miles et al. (2005) reported that families of probands with autism and macrocephaly had a significantly lower likelihood of having a family history of ADHD compared with the normocephalic group. In the same lines, Aagaard et al. (2018) through a register-based cohort study of all Danish singletons found that macrocephaly was associated with a decreased risk of ADHD (HR 0.90, 95% CI 0.82, 0.99), but neither microcephaly nor macrocephaly was associated with ASD (HR 1.06; 95% CI 0.94, 1.19, and 1.03; 95% CI 0.90, 1.19).

In a clinical setting, Albores-Gallo et al. (2017) demonstrated that 20% of Mexican children with autism had macrocephaly. The frequency of psychiatric comorbidity was very similar in the groups with macrocephaly (78.4%) and without macrocephaly (79%). The most associated comorbidity in the group with autism and macrocephaly was attention deficit hyperactivity disorder (ADHD) with 46%, followed by specific phobia with 16% and separation anxiety with 6.4%.

Future Directions

Macrocephaly should be more studied because evidence shows that stratifying by head circumference increases the power to detect differences in genetic studies as suggested by Conciatori et al. (2004).

More research is necessary with larger sample size and longitudinal studies. Since cephalic growth patterns associated with autism vary in different countries, it is important to investigate

some prenatal environmental factors such as diet, use of vitamins and regional teratogens, maternal exposure to stress, viral or bacterial infection, and thalidomide and valproic acid use as triggers for cerebral overgrowth (Grabrucker 2013). Since macrocephaly precedes the emergence of symptoms of autism, it would be interesting to focus on risk factors postnatally like fever, viral or bacterial infections, and nutritional deficiencies, which could trigger cephalic overgrowth.

The inclusion of the cephalic type as a specifier in the diagnostic classifications of DSM or ICD would be important for detecting associations in prospective and retrospective studies of autism.

References and Reading

- Aagaard, K., Bach, C. C., Henriksen, T. B., Larsen, R. T., & Matthiesen, N. B. (2018). Head circumference at birth and childhood developmental disorders in a nationwide cohort in Denmark. *Paediatric and Perinatal Epidemiology*, 32(5), 458–466.
- Albores-Gallo, L., Fritsche-Garcia, L., Miranda-Aguirre, A. P., & Avila-Acosta, M. (2017). Brief report: Macrocephaly phenotype and psychiatric comorbidity in a clinical sample of Mexican children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 47(9), 2911–2917.
- Aylward, E. H., Minshew, N. J., Field, K., Sparks, B. F., & Singh, N. (2002). Effects of age on brain volume and head circumference in autism. *Neurology*, 59(2), 175–183.
- Barnard-Brak, L., Sulak, T., & Hatz, J. K. I. (2011). Macrocephaly in children with autism spectrum disorders. *Pediatric Neurology*, 44(2), 97–100.
- Blanken, L. M. E., Dass, A., Alvares, G., van der Ende, J., Schoemaker, N. K., El Marroun, H., et al. (2018). A prospective study of fetal head growth, autistic traits and autism spectrum disorder: Fetal head growth and autistic traits. *Autism Research*, 11(4), 602–612.
- Bray, P. F., Shields, W. D., Wolcott, G. J., & Madsen, J. A. (1969). Occipitofrontal head circumference—an accurate measure of intracranial volume. *The Journal of Pediatrics*, 75, 303–305.
- Cederlund, M., Miniscalco, C., & Gillberg, C. (2014). Preschoolchildren with autism spectrum disorders are rarely macrocephalic: A population study. *Research in Developmental Disabilities*, 35(5), 992–998.
- Conciatori, M., Stodgell, C. J., Hyman, S. L., O'Bara, M., Militeri, R., Bravaccio, C., et al. (2004). Association between the HOXA1 A218G polymorphism and increased head circumference in patients with autism. *Biological Psychiatry*, 55(4), 413–419.

- Constantino, J. N., Majmudar, P., Bottini, A., Arvin, M., Virkud, Y., Simons, P., & Spitznagel, E. L. (2010). Infant head growth in male siblings of children with and without autism spectrum disorders. *Journal of Neurodevelopmental Disorders*, 2(1), 39–46.
- Courchesne, E., Karns, C. M., Davis, H. R., Ziccardi, R., Carper, R. A., Tigue, Z. D., et al. (2001). Unusual brain growth patterns in early life in patients with autistic disorder an MRI study. *Neurology*, 57(2), 245–254.
- Courchesne, E., Carper, R., & Akshoomoff, N. (2003). Evidence of brain overgrowth in the first year of life in autism. *JAMA*, 290(3), 337–344. <http://jama.jamanetwork.com/article.aspx?articleid=196951&resultclick=1>. Accessed 26 May 2016.
- Davidovitch, M., Golan, D., Vardi, O., Lev, D., & Lerman-Sagie, T. (2011). Israeli children with autism spectrum disorder are not macrocephalic. *Journal of Child Neurology*, 26(5), 580–585.
- Davis, J. M., Keeney, J. G., Sikela, J. M., & Hepburn, S. (2013). Mode of genetic inheritance modifies the association of head circumference and autism-related symptoms: A cross-sectional study. *PLoS One*, 8(9), e74940.
- Dawson, G., Munson, J., Webb, S. J., Nalty, T., Abbott, R., & Toth, K. (2007). Rate of head growth decelerates and symptoms worsen in the second year of life in autism. *Biological Psychiatry*, 61(4), 458–464.
- Dementieva, Y. A., Vance, D. D., Donnelly, S. L., Elston, L. A., Wolpert, C. M., Ravan, S. A., et al. (2005). Accelerated head growth in early development of individuals with autism. *Pediatric Neurology*, 32(2), 102–108.
- Dissanayake, C., Bui, Q. M., Huggins, R., & Loesch, D. Z. (2006). Growth in stature and head circumference in high-functioning autism and Asperger disorder during the first 3 years of life. *Development and Psychopathology*, 18(2), 381–393.
- Fidler, D. J., Bailey, J. N., & Smalley, S. L. (2000). Macrocephaly in autism and other pervasive developmental disorders. *Developmental Medicine & Child Neurology*, 42(11), 737–740.
- Filipek, P. A., Richelme, C., Kennedy, D. N., Rademaker, J., Pitcher, D. A., Zidel, S., & Caviness, V. S. (1992). Morphometric analysis of the brain in developmental disorders and autism. *Annals of Neurology*, 32, 475.
- Fletcher, H. M. (1900). A case of Megalencephaly. *London:Pathological Society of London*, 51, 230232.
- Froehlich, W., Cleveland, S., Torres, A., Phillips, J., Cohen, B., Torigoe, T., et al. (2013). Head circumferences in twins with and without autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(9), 2026–2037.
- Gooskens RH, Willemse J, Bijlsma JB, Hanlo PW (1988) Megalencephaly: definition and classification. *Brain Dev*, 10(1), 1–7.
- Grubucker, A. M. (2013). Environmental factors in autism. *Front Psychiatry*, 18(3), 118.
- Gray, K. M., Taffe, J., Sweeney, D. J., Forster, S., & Tonge, B. J. (2012). Could head circumference be used to screen for autism in young males with developmental delay?: Head circumference: Children with autism. *Journal of Paediatrics and Child Health*, 48(4), 329–334.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Korevaar, T. I. M., Muetzel, R., Medici, M., Chaker, L., Jaddoe, V. W. V., de Rijke, Y. B., et al. (2016). Association of maternal thyroid function during early pregnancy with offspring IQ and brain morphology in childhood: A population-based prospective cohort study. *The Lancet. Diabetes & Endocrinology*, 4(1), 35–43.
- Lainhart, J. E., Piven, J., Wzorek, M., Landa, R., Santangelo, S. L., Coon, H., & Folstein, S. E. (1997). Macrocephaly in children and adults with autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 36(2), 282–290.
- Lainhart, J. E., Bigler, E. D., Bocian, M., Coon, H., Dinh, E., Dawson, G., et al. (2006). Head circumference and height in autism: A study by the collaborative program of excellence in autism. *American Journal of Medical Genetics. Part A*, 140(21), 2257–2274.
- Libero, L. E., Nordahl, C. W., Li, D. D., Ferrer, E., Rogers, S. J., & Amaral, D. G. (2016). Persistence of megalencephaly in a subgroup of young boys with autism spectrum disorder: Megalencephaly in boys with autism. *Autism Research*, 9(11), 1169–1182.
- Miles, J. H., Takahashi, T. N., Bagby, S., Sahota, P. K., Vaslow, D. F., Wang, C. H., et al. (2005). Essential versus complex autism: Definition of fundamental prognostic subtypes. *American Journal of Medical Genetics*, 135A, 171–180.
- Piven, J., Arndt, S., Bailey, J., Havercamp, S., Andreasen, N. C., & Palmer, P. (1995). An MRI study of brain size in autism. *The American Journal of Psychiatry*, 152(8), 1145–1149.
- Sacco, R., Militeri, R., Frolli, A., Bravaccio, C., Gritti, A., Elia, M., et al. (2007). Clinical, morphological, and biochemical correlates of head circumference in autism. *Biological Psychiatry*, 62(9), 1038–1047.
- Sacco, R., Curatolo, P., Manzi, B., Militeri, R., Bravaccio, C., Frolli, A., et al. (2010). Principal pathogenetic components and biological endophenotypes in autism spectrum disorders. *Autism Research: Official Journal of the International Society for Autism Research*, 3(5), 237–252.
- Sacco, R., Gabriele, S., & Persico, A. M. (2015). Head circumference and brain size in autism spectrum disorder: A systematic review and meta-analysis. *Psychiatry Research: Neuroimaging*, 234(2), 239–251.
- Stadelmaier, R., Nasri, H., Deutsch, C. K., Bauman, M., Hunt, A., Stodgell, C. J., et al. (2017). Exposure to sodium valproate during pregnancy: Facial features and signs of autism: Dysmorphic features associated with valproate. *Birth Defects Research*, 109(14), 1134–1143.
- Tan, A. P., Mankad, K., Gonçalves, F. G., Talenti, G., & Alexia, E. (2018). Macrocephaly: Solving the

diagnostic dilemma. *Topics in Magnetic Resonance Imaging*, 27(4), 197–217.

- Vinkhuyzen, A. a. E., Eyles, D. W., Burne, T. H. J., Blanken, L. M. E., Kruithof, C. J., Verhulst, F., et al. (2018). Gestational vitamin D deficiency and autism-related traits: The generation R study. *Molecular Psychiatry*, 23(2), 240–246.
- Webb, S. J., Nalty, T., Munson, J., Brock, C., Abbott, R., & Dawson, G. (2007). Rate of head circumference growth as a function of autism diagnosis and history of autistic regression. *Journal of Child Neurology*, 22(10), 1182–1190.
- Whitehouse, A. J. O., Holt, B. J., Serralha, M., Holt, P. G., Hart, P. H., & Kusel, M. M. H. (2013). Maternal vitamin D levels and the autism phenotype among offspring. *Journal of Autism and Developmental Disorders*, 43(7), 1495–1504.

Macrographia

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Definition

Macrographia is abnormally large handwriting. It might be seen in patients with cerebellar disease (Haymaker 1956) and basal ganglia dysfunctions such as Huntington disease (Phillips et al. 1994). It has also been observed in a sample of patients with aphasia (Fradis and Leischner 1985). Macrographia has been documented in both children and adults with autism spectrum disorder (Beversdorf et al. 2001; Johnson et al. 2013). In the Johnson et al. study, macrographia was associated with poor manual dexterity. Overall, these findings might reflect abnormalities in the prefrontal/basal ganglia/cerebellar motor network in the ASD population. However, more empirical research is needed to investigate the nature of handwriting abnormalities in autism.

See Also

► [Micrographia](#)

References and Reading

- Beversdorf, D. Q., Anderson, J. M., Manning, S. E., Anderson, S. L., Nordgren, R. E., Felopulos, G. J., et al. (2001). Brief report: Macrographia in high-functioning adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 31(1), 97–101.
- Fradis, A., & Leischner, A. (1985). Die Zahlenagraphie. *European Archives of Psychiatry and Neurological Sciences*, 235, 82–91.
- Haymaker, W. (1956). *Bing's local diagnosis in neurological diseases* (pp. 232–234). St. Louis: C.V. Mosby.
- Johnson, B. P., Phillips, J. G., Papadopoulos, N., Fielding, J., Tonge, B., & Rinehart, N. J. (2013). Understanding macrographia in children with autism spectrum disorders. *Research in Developmental Disabilities*, 34(9), 2917–2926.
- Phillips, J. G., Bradshaw, J. L., Chiu, E., & Bradshaw, J. A. (1994). Characteristics of handwriting of patients with Huntington's disease. *Movement Disorders*, 9, 521–530.

Magnetic Resonance Imaging

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Definition

Magnetic resonance imaging (MRI) is a noninvasive technique by which detailed images of internal anatomy are constructed by measuring the response of atomic nuclei to strong magnetic fields and innocuous radio waves. The hydrogen proton (1H) is the atomic nuclei of choice due to its abundance in water molecules which makes up most of the human body's mass. During the procedure, protons emit unique signals which depend on their distinct chemical environment. These signals are then used to construct a series of two-dimensional images, which taken together, can provide valuable information of soft tissue (i.e., organ, muscle, etc.) anatomy in three dimensions. MRI uses no ionizing radiation and is well known for being a painless and harmless procedure. This

is evidenced by its widespread use in the clinical and research arenas in populations spanning the entire age range from infants to the elderly. In clinical practice, MRI is best known as a diagnostic tool used to differentiate diseased from normal tissue. However, due to its tremendous versatility and ability to provide accurate, detailed information, MRI has widespread applications in contemporary clinical and research practice.

Historical Background

Nuclear magnetic resonance (NMR) describes the physical phenomenon on which MRI technology is based. The phenomenon of NMR was actually discovered in the 1940s and set the stage for the development of MRI for medical diagnostic use in the 1970s. NMR signals are created by certain atomic nuclei when excited by radio frequency energy in the presence of a strong magnetic field. In 1946, Edward Purcell and Felix Bloch independently developed methods for determining precise nuclear magnetic measurements. This was a monumental achievement, and both were recognized with awards for the Nobel Prize in physics (1952). Spurred by the development of computed tomography in the early 1970s and coupled with advances in the development of new image reconstruction algorithms, chemist Paul Lauterbur and physicist Sir Peter Mansfield created different methods to translate information regarding magnetic spin into cross-sectional images. This seminal work began a rapid progression of continuously improving imaging techniques, and both were jointly awarded the Nobel Prize in physiology or medicine (2003). Many further refinements at various stages have resulted in the acquisition of higher resolution depictions through the improved extraction of signal from tissue over background noise and improvements in the generation of tissue contrast.

Current Knowledge

In this section, a brief review is provided on the physics of MRI as there are now numerous texts

covering this topic in detail (please see References). MRI exploits the magnetic properties of the atomic constituents of biological matter to construct a visual representation of tissue. Specifically in medical MRI, the signal from the nuclei of hydrogen atoms (^1H) is used for image generation since hydrogen constitutes two-thirds of all atoms in the human body. The proton of the hydrogen atom is positively charged and possesses spin, an intrinsic property of all elementary particles. In essence, the proton rotates about its axis and is analogous to a spinning top. The proton, which is found in the nucleus of the hydrogen atom, has considerable mass relative to the electron which orbits around the nucleus. As a rotating mass, the proton possesses angular momentum that strives to retain the spatial orientation of its rotational axis. Since the proton is both charged and in constant motion, it additionally has a magnetic moment and can be imagined to behave as a small magnet. What is basically observed in MRI is the motion of the proton's magnetic axis, which is capable of generating a signal in the MRI scanner's receiver coil.

When an external force acts on the hydrogen proton and tries to alter the orientation of its rotation axis, the proton begins to wobble or precess around its axis of rotation. At the same time, friction caused by the interaction between the external force and the proton withdraws energy from the proton and slows down its rotation. As a result, the proton's axis of rotation becomes progressively more inclined and if imagined as a spinning top, finally falls over. More specifically, when hydrogen nuclei are exposed to an external magnetic field, the magnetic moments (or spins) align with the direction of the field and simultaneously undergo precession. The precession of the nuclei occurs at a characteristic speed that is proportional to the strength of the applied magnetic field and is called the Larmor frequency. The Larmor frequency is the rate at which spins wobble when placed in a magnetic field. The spin system eventually relaxes and settles into a stable state, but the longitudinal magnetization (M_z) builds up in the z-direction because the magnetic vectors representing the individual magnetic moments accumulate. The spins tend to

align parallel and antiparallel to the magnetic field, with parallel alignment being slightly more preferred because it is energetically more favorable. Hence, a slightly larger fraction of spins aligns parallel to the main magnetic field. This small difference produces the measurable net magnetization M_z and is represented by the net magnetization vector. This energy difference between the two spin orientations depends on the strength of the external magnetic field with M_z increasing as field strength increases.

Furthermore, energy can be introduced into this spin system by applying an electromagnetic wave of the same frequency as the Larmor frequency, a condition known as the resonance state. This wave is generated in a radio transmitter and applied to the object to be imaged using an antenna coil. The result is that the longitudinal magnetization becomes more and more tipped away from the z -axis toward the transverse xy -plane, which is perpendicular to the direction of the main magnetic field. All of the longitudinal magnetization is rotated into the transverse plane by a radio frequency pulse that is both strong enough and applied long enough to tip the magnetization by exactly 90° . This results in M_{xy} magnetization, which as its name suggests, lies in the xy -plane. Transverse magnetization rotates about the z -axis, which has the effect of an electrical generator and induces an alternating voltage of the same frequency as the Larmor frequency in the MRI scanner's receiver coil. This is called of the magnetic resonance signal which is collected and processed with sensitive receivers and computers to generate the magnetic resonance image.

The magnetic resonance signal rapidly fades due to two independent processes that reduce transverse magnetization and thus return the spin system to the stable state present prior to excitation. These two processes are spin-lattice interaction which causes T_1 relaxation and spin-spin interaction which causes T_2 relaxation. T_1 is defined as the time required for 63% of the original longitudinal magnetization to be recovered. T_2 is defined as the time required for transverse magnetization to decrease to 37% of the original value. T_1 typically ranges from 200 to 2,000 ms and T_2 commonly ranges from 30 to 500 ms. T_2

denotes the process of energy transfer between spins, while T_1 refers to the effects of additional field inhomogeneities contributing to spins losing coherence. A key strategy for how differences in T_1 and T_2 are exploited to generate tissue contrast involves strategic variation of timing and orientation of repetitive radio frequency pulse delivery.

MRI's ability to localize signals in the three-dimensional space of the brain is accomplished by using magnetic gradients which are magnetic fields which change gradually along an axis. Encoding of a three-dimensional volume begins by first dividing the tissue mass into slices. Then two distinct magnetic gradients orthogonal to each other are applied, effectively dividing each slice into rows and columns of pixels. By doing this, each pixel possesses unique precessional frequency and direction. A special mathematical operation, called Fourier transform, converts pixel data back into three-dimensional voxels, which are then assembled to form an image volume reconstruction of the original three-dimensional tissue mass. Optimal spatial resolution currently approximates 1 mm^3 or less.

Proton densities and differential T_1 and T_2 relaxation effects are properties intrinsic to brain tissues, and subsequently, their measurement forms the basis for the provision of differential tissue image contrast in MRI. Different tissue parameters can be differentially weighted, thus, yielding a variety of image types, each providing a unique range of diagnostic information. Different image modalities are usually identified by numeric values of key pulse sequence parameters set during acquisition. Use of shorter repetition time between pulse sequences during image acquisition interrogates tissues at a moment when their intrinsic T_1 differences can be exploited to yield different signal intensity production and hence tissue contrast generation. The resulting image, whose contrast is based on tissue differences in T_1 , is termed T_1 -weighted. A repetition time less than 500 ms is considered short; a repetition time greater than 1,500 ms is considered long. Use of repetitive 90° pulses to detect T_1 -based tissue contrast is called a saturation recovery sequence. Alternatively, inversion recovery involves a sequence that begins with a

180° pulse followed by a 90° pulse. The 180° pulse reverses longitudinal magnetization, and all protons responsible for the net magnetic moment are inverted 180° in the applied longitudinal magnetic field. The signal that is received depends on the time between the 180- and 90° interrogation pulses, which constitutes the repetition time for this particular pulse sequence. On the other hand, a primary strategy for generating T2-weighted images involves use of a pulse sequence termed spin echo. In spin echo, an initial 90° pulse generates transverse magnetization vector component. When 90° pulse is terminated, dephasing follows under the influence of T2. When 180° pulse is administered, the direction of dephasing protons is reversed. More slowly, precessing protons are now ahead of faster ones. Eventually, protons with faster precession frequencies catch up, culminating in proton precession rephrasing and thus detectable signal generation. This process is repeated, and protons again dephase, are again refocused by another 180° pulse, and so on. Therefore, it is possible to obtain more than one signal by repeating the spin-echo sequence.

In clinical practice, substances with short T1 producing high signals on T1-weighted imaging include fat, methemoglobin in subacute hemorrhage, and paramagnetic contrast agents. Substances with long T1 producing low signals on T1-weighted imaging include cerebral spinal fluid and tissues in which any process that increases local water has occurred (i.e., inflammation). T1 images are best for visualizing normal anatomy. Tissues with longer T2 generate higher signals on T2-weighted images. Because of the greater fluid content of a tissue, the longer the tissue's T2, the brighter that tissue's appearance on a T2-weighted image. Thus, T2-weighted images highlight fluid-containing regions, such as sulci and ventricular system. Also in a healthy brain, T2 constitutes the entirety of high intensity signal. Substances with short T2 produce low signals on T2-weighted images. Examples include iron-containing substances. Also, most brain lesions involve associated change in water content, such as cellular injury (infarction) and extracellular inflammatory lesions (mass lesions) which produce vasogenic

edema. Because a final common pathway of many brain lesions involves increases in water content of brain, T2-weighted images, which highlight tissue with higher water content, demonstrate brain pathology as higher signal intensities. T2-weighted images are useful for evaluating sulcal widening in cortical atrophic syndromes and for evaluating hydrocephalus.

Future Directions

In the decades to come, advances in MRI technology include a widespread transition to higher magnetic field imaging, enhanced MRI coil sophistication incorporating even more channels, ultrashort echo-time imaging, tighter integration of multiple imaging modalities, and advances in molecular MRI agents. Currently, the typically used clinical imaging scanners operate at 1.5 T. While systems operating at even higher field strengths have become more prevalent, they are found mainly at major medical centers. Current clinical interest focuses on transitioning to 3 T scanners; however, it is already evident that much higher field strengths will eventually be used to examine patients (up to 7 T, which so far have only been used in research studies). Available data suggests that magnetic field strengths above 2 T involve no increased risk for patients, but the strongest argument for increasing field strength is the expectation that this will significantly boost signal-to-noise ratio. Improved signal-to-noise ratio leads to increased spatial resolution and/or reduced imaging time. An improved spatial resolution permits better anatomical evaluation. Shorter scan time leads to better tolerability and more cost-effective operation (more patients can be examined per unit time). Finally, imaging at 3 T or higher field strengths has the potential to improve more sophisticated applications of MRI such as functional imaging and carbon-13 or hydrogen-1 spectroscopy. For example, it is known that the chemical shift increases in proportion to magnetic field strength and the larger chemical shift is advantageous in spectroscopy because the spectral lines spread farther apart. Thus, this improves

spectral resolution and discrimination of the peaks of fat and water, which in turn enables better calibration of the radio frequency pulse for fat suppression, which can significantly diminish the peaks of interest. Also, increasingly higher numbers of radio frequency channels will become standard, having a significant impact in neurological examinations. Commercial products for simultaneous acquisitions of both positron emission tomography and MRI will become commonplace in many centers where their use will be justified by improved throughput, improved patient compliance, and improved image and diagnostic accuracy. Of key importance in combined modalities, however, is the validation of image quality acquired by one modality in the presence of the other. For example, the verification that positron emission tomography image quality is not impaired by placing the detector system into the magnetic field and that MRI quality is not degraded by the presence of the positron emission tomography detectors. In addition to multimodal imaging, the future will also bring a wider application of current methods into other body areas (such as the use of diffusion tensor imaging in non-neurological examinations). Finally, the development of targeted contrast media is a rapidly expanding area in MRI research and is already being frequently used in animal systems. These contrast agents show areas of abnormality in terms of passive accumulation of agent within the tissue, for example, a normal brain will exclude contrast from the parenchyma, but damaged tissue allows passive diffusion of contrast agent through the compromised blood-brain barrier, leading to image enhancement. The future will also bring very highly sensitivity agents that either are targeted to specific disease processes or provide specific information on regional metabolism.

See Also

- ▶ [Diffusion Tensor Magnetic Resonance Imaging](#)
- ▶ [Functional MRI](#)
- ▶ [Magnetic Resonance Spectroscopy](#)

References and Reading

- Blamire, A. D. (2008). The technology of MRI – the next 10 years? *British Journal of Radiology*, 81, 601–617.
- Goldstein, M. A., Price, B. H., Dougherty, D. D., Rauch, S. L., & Resenbaum, J. F. (2004). *Essentials of neuroimaging for clinical practice*. Arlington: American Psychiatric.
- Gore, J. (2003). Out of the shadows – MRI and the Nobel prize. *The New England Journal of Medicine*, 349, 2290–2292.
- Renshaw, P. F., & Parow, A. M. (2002). *Psychiatric disease in magnetic resonance imaging of the brain and spine*. Philadelphia: Lippincott, Williams & Wilkins.
- Schlid, H. H. (1999). *MRI made easy* (5th ed.). Berlin: Schering AG/Berlex Laboratories.
- Weishaupt, D., Kochli, V. D., & Marincek, B. (2006). *How does MRI work? An introduction to the physics and function of magnetic resonance imaging* (2nd ed.). Heidelberg: Springer.
- Westbrook, C., Roth, C. K., & Talbot, J. (2005). *MRI in practice* (3rd ed.). Oxford, UK: Blackwell.

Magnetic Resonance Spectroscopy

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Definition

Magnetic resonance spectroscopy (MRS) is a technique similar to magnetic resonance imaging (MRI) that can extract chemical information about biological tissues. MRS is also known as nuclear magnetic resonance (NMR) spectroscopy, though this term tends to be avoided in in vivo work. MRS can be accomplished in most clinical MRI scanners and is similarly safe. However, rather than providing an image of the tissue as MRI would, MRS provides a “spectrum,” or chemical signature, of the area under examination. From this, metabolic and biochemical disturbances can be identified in a region-specific fashion. MRS has applications across the body; however, one of the most prominent applications of MRS is to examine biochemical disturbances in the brain.

Historical Background

For a brief history of NMR, please see ► [“Magnetic Resonance Imaging”](#).

Early work in the 1970s showed that MRS could be used to obtain chemical information from living animal tissue, such as blood cells or muscle tissue (de Graaf 2008). However, it was not until the early 1980s that the brain was examined under ^1H MRS in living mammals and humans (Ross and Bluml 2001). These studies paved the way for neurospectroscopic applications, ranging from the evaluation of traumatic brain injuries to predictive markers of Alzheimer’s disease risk to studies of neuropsychiatric disorders such as schizophrenia and autism.

Current Knowledge

Utility and Applicability of MRS

As MRS can typically be conducted with the same equipment as standard MRI, MRS can be considered a very accessible and well-tolerated radiological imaging modality (for comparisons with other modalities, see Siegel and Albers 2006). As with MRI, MRS uses nonionizing radiofrequency pulses for excitation of nuclei and magnetic field gradients for spatial localization. MRS does require, however, specifically built pulse sequences for acquiring data and specialized algorithms and frameworks for analysis. These modifications and additions are widely available on most clinical MR machines.

However, it is also important to note that MRS encompasses a wide range of both clinical and research endeavors. Many types of specialized sequences exist (e.g., see section [“MRS of GABA in Autism,”](#) below) as do specialized protocols (some of which look at other nuclei besides ^1H that are MR-detectable, e.g., ^{31}P , ^{13}C). The bulk of this article will discuss applications that are most relevant to studies of autism, primarily in vivo ^1H MRS of the human brain.

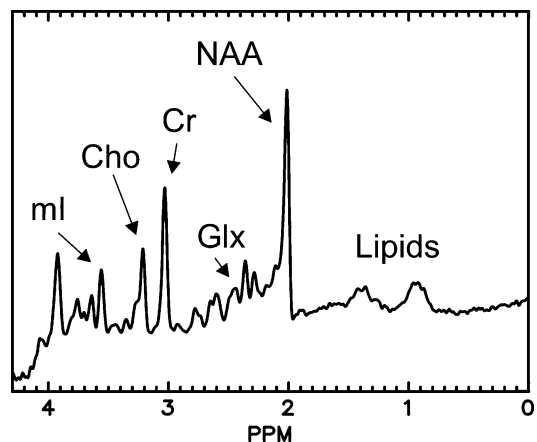
Differences Between MRI and MRS

In standard MRI, pulse sequences are chosen to emphasize specifics regarding the spatial

distribution of protons, resulting in an image. Thus, the strongest contributors to an MRI profile are water and fats. By contrast, MRS seeks to optimize the provision of spectral information, specifically isolating metabolites and other biochemicals in an organism. Because different nuclei on a molecule may exist in distinct chemical environments, and because each of these chemical environments can result in different levels of “shielding” from the external magnetic field of the MRI machine, this results in certain molecules having “chemical signatures” that are different from other molecules (see section [“ \$^1\text{H}\$ MR Spectra in the Human Brain”](#)). However, the concentrations of these chemicals are often very low, and thus the signal from metabolites, e.g., in the brain, is approximately 10,000 times smaller than that of water. For this reason, additional steps are typically taken both at the time of spectral acquisition and data processing for eliminating dominating signals from water or fats so that other metabolites will be visible (de Graaf 2008; Shic et al. 2010).

^1H MR Spectra in the Human Brain

The most common form of MRS in use today for studying in vivo brain biochemistry is ^1H MRS. This form of MRS uses the same type of head coils as MRI and can typically be conducted as part of a standard clinical imaging examination. A typical MR spectrum is shown below.



MR spectra are typically displayed with frequency (chemical shift) on the x-axis, expressed in parts-per-million (PPM). PPM is the frequency

difference between a reference chemical (typically tetramethylsilane) and a particular frequency, all divided by the frequency of the MRI machine and multiplied by a million. Since the frequency of the MRI machine scales linearly with its field strength, PPM provides a field-independent axis of chemical shifts for metabolites. The y-axis of MR spectra is the signal intensity, a value that scales with the concentration of a metabolite.

A detailed discussion of the clinical utility and biochemical processes underlying MRS-detectable metabolites is beyond the scope of this article (for more details, please see Ross and Bluml (2001) and Lin et al. (2005)), as are the optimizations necessary for obtaining high-quality spectra (for practical advice and theoretical considerations, see Blüml 2011). Briefly, however, primary metabolites seen in *in vivo* 1H MRS of the brain include: (1) lipids, typically seen in voxels containing subcutaneous fats from the skull or in necrotic tissue; (2) lactate, which appears as a doublet at 1.33 PPM, a by-product of anaerobic glycolysis which is naturally occurring at low concentration levels in the cerebrospinal fluid but which can also be seen in hypoxia and stroke; (3) N-acetyl aspartate (NAA), a marker for healthy neurons, axons, and dendrites, which is decreased in events such as hypoxic or traumatic brain injury; (4) the glutamine and glutamate complex (Glx), which includes both glutamine and glutamate due to overlapping chemical profiles, is involved in inhibitory and excitatory neurotransmission in the human brain and is disturbed in some neuropsychiatric conditions such as schizophrenia; (5) creatine + phosphocreatine (Cr), energy markers of brain metabolism, which can be disturbed in brain trauma and hyperosmolar conditions such as hyponatremia; (6) choline (Cho), a myelin and membrane marker, elevated in certain tumors and in stroke; and (7) myoinositol, a brain osmolyte and a marker for astrocytes found to be elevated in Alzheimer's disease and diminished in hepatic encephalopathy.

Typically, the levels of metabolites are expressed as either a ratio (e.g., the peak height of NAA divided by the peak height of Cr, i.e., NAA/Cr) or as an absolute concentration level

(typically enclosed in brackets, e.g., [NAA]). Creatine is often used as a reference signal for studies reporting ratios, given its relative homeostatic stability. However, it is important to note that creatine concentrations can be affected by some pathologies (see Ross and Bluml 2001 for details). Several methods exist for quantifying absolute concentration levels of metabolites, but the analysis is more complex and often involves assumptions regarding the spectral shape of metabolites, the creation of model solutions for use as a basis set, and the use of brain water as a reference (Christiansen et al. 1993; Kreis et al. 1993; Pouillet et al. 2008).

Single-Voxel and Multivoxel Spectroscopy

As in MRI, there exist many different protocols for acquiring MRS from the brain. Typical 1H clinical examinations can be broken down into single-voxel techniques and multivoxel techniques. In single-voxel spectroscopy, a small area in the brain is chosen *a priori*, based on scout images or prior MRI in the same session as the MRS session, and 1H MR spectra acquired from those locations. In multivoxel spectroscopy (also called spectroscopic imaging or chemical shift imaging), spectra are obtained from a wide area under additional phase encoding, thus providing chemical profiles from multiple locations within a larger spatial volume. The advantages of multivoxel techniques include increased ability to detect atypical neurochemical profiles over a larger space and greater time efficiency of detection over the volume compared to comparable serial single-voxel MRS. The disadvantages of multivoxel techniques are increased complexities in data analysis, increased difficulties in controlling for magnetic field inhomogeneities (due to the larger size of the whole volume), and “voxel bleeding” (i.e., contamination of chemical profiles within one voxel with spatially adjacent voxels) (see de Graaf 2008 for details).

Short- and Long-Echo Spectroscopy

The appearance of 1H MR spectra is highly dependent on parameters of the pulse sequence employed. The width of peaks in MRS is partially determined by T2* time, a property of a molecule that includes both T2, the transverse relaxation

time of a metabolite, as well as magnetic field inhomogeneity effects. Less mobile molecules, such as lipids, tend to have lower T2 times (and hence lower T2* times), effectively translating into peaks that are broader and shorter. The echo time of an MRS pulse sequence (the TE time) will refocus (i.e., help to eliminate) inhomogeneity effects, but not T2 effects, with the resultant effect that longer TEs result in decreased visibility of short T2 time metabolites. This has advantages as well as disadvantages. The disadvantages include the decreased visibility of important metabolites with shorter TEs which become harder to identify at longer TEs and the generally decreased signal-to-noise (SNR) ratio associated with waiting for metabolite signals to decay. The advantages include less complex baselines, i.e., less contamination by “nuisance” molecules which alter the shape underlying the peaks of metabolites of interest. Some contributors to complex baselines include macromolecules, which have very short TEs (Behar et al. 1994). Typical short-echo times are TE = 30–35 ms, whereas long-echo times range from 135 to 144 ms (lactate, which is a j-coupled resonance, inverts in this range of echo times, making it somewhat easier to detect). Other researchers recommend a midrange TE, e.g., 40–50 ms, as a compromise for relatively flat macromolecular contributions and good SNR (Hetherington et al. 2005).

1H MRS Studies of Autism

Currently, MRS studies have painted a broad picture regarding neurochemical differences between ASD groups and controls, and some researchers have argued, based on low levels of specific neural markers, that autism may be characterized by disruption of neuronal integrity in a region-specific or global pattern (Dager et al. 2008a). The earliest examinations with MRS are of children diagnosed with autism at 3–4 years of age (Friedman et al. 2003).

Region-specific abnormalities in autism reported by 1H MRS studies include decreased NAA in the hippocampus-amygdala (Gabis et al. 2008; Mori et al. 2001; Otsuka et al. 1999), transverse temporal gyrus (Hisaoka et al. 2001), and medial temporal lobe (Endo et al. 2007). Several

groups have also noted functional relationships between NAA concentrations and measures of social functioning (Endo et al. 2007; Hardan et al. 2008; Kleinhans et al. 2009; Oner et al. 2009). It is important to note, however, that several groups have found negative results in similar areas or with different subject populations (Kleinhans et al. 2009; Oner et al. 2009; Perich-Alsina et al. 2002; Zeegers et al. 2007). Nonetheless, abnormal concentrations of metabolites in the temporal lobe, or relationships between temporal lobe and social functioning, are among the most reported results in 1H MRS as applied to the study of ASD. 1H MRS work by some researchers has also identified abnormalities in levels of brain metabolites other than NAA, such as Cho, mI, Cr, and Glx, in a region-specific fashion (e.g., DeVito et al. 2007; Gabis et al. 2008; Hashimoto et al. 1997; Kahne et al. 2002; Levitt et al. 2003; Mori et al. 2001; Murphy et al. 2002; Sokol et al. 2002; Vasconcelos et al. 2008), though the results are somewhat varied, possibly due to differences in subject populations, acquisition parameters, and experimental protocols.

Some groups have also reported what appears to be globally depressed concentrations of all metabolites in autism, primarily as associated with brain gray matter (DeVito et al. 2007; Friedman et al. 2003, 2006; Kleinhans et al. 2007). Friedman et al. (2006), in a particularly relevant study, include as participants the youngest reported group of children with ASD: 45 children 3–4 years of age. Several of these studies, including Friedman et al., also report longer T2 times in autism. T2, also known as spin-spin relaxation time, reflects compartment differences (i.e., where the chemicals are being measured, e.g., intracellularly or extracellularly) and can be interpreted as an indicator of the mobility of the associated metabolite (Dager et al. 2008a). As noted by Dager et al. (2008b), the increased T2 times observed in children with ASD are not compatible with brain overgrowth models of autism which suggest that incomplete neuronal pruning leads to dense neuronal cell packing, as in this case T2 times would be decreased, expressing decreased cellular mobility.

MRS of GABA in Autism

Though standard ¹H MRS protocols can paint a rich picture of neurochemical abnormalities in individuals with ASD, some particularly noteworthy metabolites are difficult to detect using these standard approaches. One such metabolite is GABA, the primary inhibitory neurotransmitter of the central nervous system. GABA exists in low concentrations in the brain, relative to the other metabolites outlined in the section above. Furthermore, the spectrum of GABA is overlapped by other metabolites, obscuring it from view. Recently, Harada et al. (2010) employed a specialized GABA-editing sequence in order to measure GABA levels in the frontal lobe and lenticular nuclei of adults with autism and matched controls. The authors of that study found that adults with autism showed decreased levels of GABA and GABA levels relative to glutamate in the frontal lobe but not the lenticular nuclei, suggesting a frontal lobe-specific disturbance in excitatory-inhibitory neurotransmission.

Mitochondrial Dysfunction in Autism

One application of MRS has been the study of brain mitochondria dysfunction in ASD. Using optimized proton echo-planar spectroscopic imaging, Corrigan et al. (2011) showed no presence of lactate in a longitudinal sample of children with ASD. Since the presence of elevated lactate levels has been suggested to be associated with mitochondrial dysfunction, the finding of no lactate in the study provided evidence against the belief of widespread mitochondrial dysfunction in ASD and, consequently, the practice of hyperbaric oxygen for the treatment of ASD. This study was followed by a healthy exchange of perspectives by researchers on the Corrigan et al. study and proponents of hyperbaric oxygen treatment (Dager et al. 2011; Rossignol and Frye 2011).

Future Directions

Despite some inconsistencies in the literature to date, modern research using ¹H MRS is beginning to reveal rich interactions between brain neurochemistry and cognitive, behavioral, and social

performance in autism. Technological advances, such as more stable spectrometers, increasing field strength, and more reliable and powerful gradients, all contribute to the increased reliability of MRS investigations. Future work will likely replicate and extend prior findings with increasingly specific predictions about the relationships between region-specific neurochemical levels, behavior, and outcome.

The continued application of more advanced MRS techniques, such as GABA and glutamate editing, will deepen our understanding of biochemical abnormalities in autism. As in the example of investigations of mitochondrial dysfunction in ASD, these techniques will augment the tool sets available to researchers for providing evidence against or for new and rising hypotheses about the underlying biological mechanisms of autism. However, given the currently high levels of sophistication necessary to enact some of these protocols and process the results, concerted efforts will be required to delineate the limits of these protocols and to make the techniques more easily available and accessible for translational applications.

A particularly promising application area for MRS research in autism is the use of MRS in tracking the progression and response of individuals with ASD to pharmacological treatment plans. Such strategies have already proven to be useful in other neuropsychiatric disorders (Mason and Krystal 2006). Another area which has been little explored to date is the use of multinuclear MRS for examining nuclei other than proton (¹H), e.g., carbon-13 and phosphorous-31. Multinuclear MRS, especially infusion studies of ¹³C glucose or acetate (e.g., see Bluml et al. 2002), has the potential to provide accurate descriptions of atypical brain metabolism and energetics in autism, potentially identifying points of biochemical vulnerability in a pathway- and individual-specific manner. When combined with genetic studies which may highlight the corresponding bases for these disorders, and the potential of MRS for monitoring and tracking treatment and disease progression, the role of MRS in the study of autism should only increase in the coming years.

See Also

► Magnetic Resonance Imaging

References and Reading

- Behar, K. L., Rothman, D. L., Spencer, D. D., & Petroff, O. A. C. (1994). Analysis of macromolecule resonances in ¹H NMR spectra of human brain. *Magnetic Resonance in Medicine*, 32(3), 294–302. <https://doi.org/10.1002/mrm.1910320304>.
- Blüml, S. (2011). Magnetic resonance spectroscopy: Physical principles. In S. H. Faro, F. B. Mohamed, M. Law, & J. T. Ulmer (Eds.), *Functional neuroradiology* (pp. 141–153). Boston: Springer. Retrieved February 1, 2012, from <http://www.springerlink.com/content/1812025616j28319/>.
- Bluml, S., Moreno-Torres, A., Shic, F., Nguy, C. H., & Ross, B. D. (2002). Tricarboxylic acid cycle of glia in the in vivo human brain. *NMR in Biomedicine*, 15(1), 1–5.
- Christiansen, P., Henriksen, O., Stubgaard, M., Gideon, P., & Larsson, H. B. W. (1993). In vivo quantification of brain metabolites by ¹H-MRS using water as an internal standard. *Magnetic Resonance Imaging*, 11(1), 107–118.
- Corrigan, N. M., Shaw, D. W. W., Richards, T. L., Estes, A. M., Friedman, S. D., Petropoulos, H., et al. (2011). Proton magnetic resonance spectroscopy and MRI reveal no evidence for brain mitochondrial dysfunction in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 42(1), 105–115. <https://doi.org/10.1007/s10803-011-1216-y>.
- Dager, S. R., Friedman, S. D., Petropoulos, H., & Shaw, D. W. W. (2008a). *Imaging evidence for pathological brain development in autism spectrum disorders* (p. 361). Autism: Current Theories and Evidence.
- Dager, S. R., Oskin, N. M., Richards, T. L., & Posse, S. (2008b). Research applications of magnetic resonance spectroscopy (MRS) to investigate psychiatric disorders. *Topics in Magnetic Resonance Imaging: TMRI*, 19(2), 81.
- Dager, S. R., Corrigan, N. M., Estes, A., & Shaw, D. W. W. (2011). Further commentary on mitochondrial dysfunction in autism spectrum disorder: Assessment and treatment considerations. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-011-1352-4>.
- de Graaf, R. A. (2008). *In vivo NMR spectroscopy: Principles and techniques* (2nd ed.). Chichester/Hoboken: Wiley-Interscience.
- DeVito, T. J., Drost, D. J., Neufeld, R. W. J., Rajakumar, N., Pavlosky, W., Williamson, P., et al. (2007). Evidence for cortical dysfunction in autism: a proton magnetic resonance spectroscopic imaging study. *Biological Psychiatry*, 61(4), 465–473.
- Endo, T., Shioiri, T., Kitamura, H., Kimura, T., Endo, S., Masuzawa, N., et al. (2007). Altered chemical metabolites in the amygdala-hippocampus region contribute to autistic symptoms of autism spectrum disorders. *Biological Psychiatry*, 62(9), 1030–1037.
- Friedman, S. D., Shaw, D. W., Artru, A. A., Richards, T. L., Gardner, J., Dawson, G., et al. (2003). Regional brain chemical alterations in young children with autism spectrum disorder. *Neurology*, 60(1), 100–107. Retrieved October 2, 2009.
- Friedman, S. D., Shaw, D. W. W., Artru, A. A., Dawson, G., Petropoulos, H., & Dager, S. R. (2006). Gray and white matter brain chemistry in young children with autism. *Archives of General Psychiatry*, 63(7), 786.
- Gabis, L., Huang, W., Azizian, A., DeVincent, C., Tudorica, A., Kesner-Baruch, Y., et al. (2008). ¹H-magnetic resonance spectroscopy markers of cognitive and language ability in clinical subtypes of autism spectrum disorders. *Journal of Child Neurology*, 23(7), 766.
- Harada, M., Taki, M. M., Nose, A., Kubo, H., Mori, K., Nishitani, H., et al. (2010). Non-invasive evaluation of the GABAergic/glutamatergic system in autistic patients observed by MEGA-Editing proton MR spectroscopy using a clinical 3 Tesla instrument. *Journal of Autism and Developmental Disorders*, 41, 447–454. <https://doi.org/10.1007/s10803-010-1065-0>.
- Hardan, A. Y., Minshew, N. J., Melhem, N. M., Srihari, S., Jo, B., Bansal, R., et al. (2008). An MRI and proton spectroscopy study of the thalamus in children with autism. *Psychiatry Research: Neuroimaging*, 163(2), 97–105.
- Hashimoto, T., Tayama, M., Miyazaki, M., Yoneda, Y., Yoshimoto, T., Harada, M., et al. (1997). Differences in brain metabolites between patients with autism and mental retardation as detected by in vivo localized proton magnetic resonance spectroscopy. *Journal of Child Neurology*, 12(2), 91.
- Hetherington, H., Petroff, O., Jackson, G. D., Kuzniecky, R. I., Briellmann, R. S., & Wellard, R. M. (2005). Magnetic resonance spectroscopy. In R. I. Kuzniecky & G. D. Jackson (Eds.), *Magnetic resonance in epilepsy: Neuroimaging techniques* (pp. 333–384). Burlington: Elsevier Academic.
- Hisaoka, S., Harada, M., Nishitani, H., & Mori, K. (2001). Regional magnetic resonance spectroscopy of the brain in autistic individuals. *Neuroradiology*, 43(6), 496–498.
- Kahne, D., Tudorica, A., Borella, A., Shapiro, L., Johnstone, F., Huang, W., et al. (2002). Behavioral and magnetic resonance spectroscopic studies in the rat hyperserotonemic model of autism. *Physiology & Behavior*, 75(3), 403–410.
- Kleinhans, N. M., Schweinsburg, B. C., Cohen, D. N., Müller, R. A., & Courchesne, E. (2007). N-acetyl aspartate in autism spectrum disorders: Regional effects and relationship to fMRI activation. *Brain Research*, 1162, 85–97.
- Kleinhans, N. M., Richards, T., Weaver, K. E., Liang, O., Dawson, G., & Aylward, E. (2009). Brief report:

- Biochemical correlates of clinical impairment in high functioning autism and Asperger's disorder. *Journal of Autism and Developmental Disorders*, 39(7), 1079–1086.
- Kreis, R., Ernst, T., & Ross, B. D. (1993). Development of the human brain: in vivo quantification of metabolite and water content with proton magnetic resonance spectroscopy. *Magnetic Resonance in Medicine*, 30, 424–424.
- Levitt, J. G., O'Neill, J., Blanton, R. E., Smalley, S., Fadale, D., McCracken, J. T., et al. (2003). Proton magnetic resonance spectroscopic imaging of the brain in childhood autism. *Biological Psychiatry*, 54(12), 1355–1366.
- Lin, A., Ross, B. D., Harris, K., & Wong, W. (2005). Efficacy of proton magnetic resonance spectroscopy in neurological diagnosis and neurotherapeutic decision making. *NeuroRx*, 2(2), 197–214.
- Mason, G. F., & Krystal, J. H. (2006). MR spectroscopy: Its potential role for drug development for the treatment of psychiatric diseases. *NMR in Biomedicine*, 19(6), 690–701.
- Mori, K., Hashimoto, T., Harada, M., Yoneda, Y., Shimakawa, S., Fujii, E., et al. (2001). Proton magnetic resonance spectroscopy of the autistic brain. *No to Hattatsu*, 33(4), 329–335.
- Murphy, D. G. M., Critchley, H. D., Schmitz, N., McAlonan, G., Van Amelsvoort, T., Robertson, D., et al. (2002). Asperger syndrome: A proton magnetic resonance spectroscopy study of brain. *Archives of General Psychiatry*, 59, 885–891.
- Oner, O., Ozgüven, H. D., Oktem, F., Ya murlu, B., Baskak, B., Olmez, S., et al. (2009). Proton magnetic resonance spectroscopy in Asperger's syndrome: Correlations with neuropsychological test scores. *Türk psikiyatri dergisi (Turkish Journal of Psychiatry)*, 20(1), 22.
- Otsuka, H., Harada, M., Mori, K., Hisaoka, S., & Nishitani, H. (1999). Brain metabolites in the hippocampus-amygdala region and cerebellum in autism: An 1H-MR spectroscopy study. *Neuroradiology*, 41(7), 517–519.
- Perich-Alsina, J., Aduna, P. M., Valls, A., & Muñoz-Yunta, J. A. (2002). Thalamic spectroscopy using magnetic resonance in autism. *Revista de Neurologia*, 34, S68.
- Poulet, J.-B., Sima, D. M., & Van Huffel, S. (2008). MRS signal quantitation: A review of time- and frequency-domain methods. *Journal of Magnetic Resonance*, 195(2), 134–144. <https://doi.org/10.1016/j.jmr.2008.09.005>.
- Ross, B., & Bluml, S. (2001). Magnetic resonance spectroscopy of the human brain. *The Anatomical Record*, 265(2).
- Rossignol, D. A., & Frye, R. E. (2011). Substantial problems with measuring brain mitochondrial dysfunction in autism spectrum disorder using magnetic resonance spectroscopy. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-011-1276-z>.
- Shic, F., Lin, A., Brown, J. B., Bluml, S., & Ross, B. D. (2010, May). *An open-source platform for routine clinical 1H magnetic resonance spectroscopy processing*. Presented at the 2010 Meeting of the International Society for Magnetic Resonance in Medicine (ISMRM), E-Poster, Stockholm, Sweden.
- Siegel, G. J., & Albers, R. W. (2006). *Basic neurochemistry: Molecular, cellular, and medical aspects*. Amsterdam/Boston: Academic.
- Sokol, D. K., Dunn, D. W., Edwards-Brown, M., & Feinberg, J. (2002). Hydrogen proton magnetic resonance spectroscopy in autism: preliminary evidence of elevated choline/creatine ratio. *Journal of Child Neurology*, 17(4), 245.
- Vasconcelos, M. M., Brito, A. R., Domingues, R. C., da Cruz, L. C. H., Gasparetto, E. L., Werner, J., et al. (2008). Proton magnetic resonance spectroscopy in school-aged autistic children. *Journal of Neuroimaging*, 18(3), 288–295.
- Zeegers, M., Van Der Grond, J., van Daalen, E., Buitelaar, J., & van Engeland, H. (2007). Proton magnetic resonance spectroscopy in developmentally delayed young boys with or without autism. *Journal of Neural Transmission*, 114(2), 289–295.

Magnetoencephalography

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Definition

Magnetoencephalography (MEG) is a noninvasive neuroimaging technique that detects extracranial magnetic fields produced by electrical activity in the brain, which occurs spontaneously or in response to external stimuli. The extracranial magnetic fields are sampled by an array of sensors, generally arranged in a helmet, within which the head is contained (see Fig. 1). These extracranial magnetic fields, which comprise MEG signals, reflect the synchronous postsynaptic activity of large populations of pyramidal neurons. Electrical activity associated with synaptic transmission produces magnetic fields orthogonal to the direction of the electric currents. The direction of the magnetic fields is governed by the “right-hand rule”: When the right hand is made into a fist with



Magnetoencephalography, Fig. 1 A child sits in a whole-head biomagnetometer (MEG machine). Typically, somatosensory, visual, or auditory stimuli may be presented, and button-box responses collected synchronously with multichannel (in this case 275) recording of the magnetoencephalogram

the thumb pointing upward, the remaining fingers point in the direction of the flow of magnetic fields.

Recording magnetic fields associated with brain activity relies on specialized technology because the magnetic fields in the brain are extremely weak (on the order of 10fT – 1pT , approximately 10 million times smaller than the earth's magnetic field). As such, the coils that detect the brain's magnetic fields and the superconducting quantum interference devices (SQUIDS) to which the coils are coupled are maintained at extremely low temperatures. This is accomplished by surrounding the sensors with liquid helium and encapsulating them within a dewar. In addition, containing the MEG system within a magnetically shielded room reduces competing environmental magnetism.

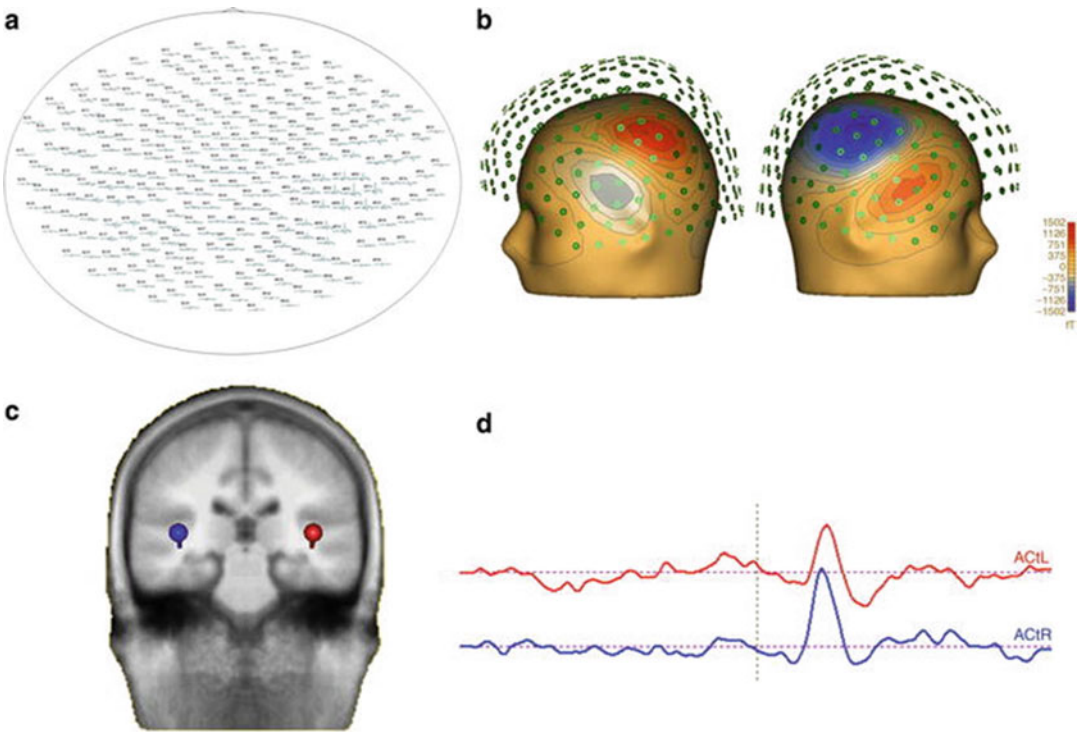
MEG recording takes place while patients or participants are either seated or lying supine, with the head positioned inside of the helmet that contains the MEG sensors. The size of the helmet is optimal for a typical adult head in the vast majority of MEG systems; however, MEG systems designed for infants and small children are currently being developed. Unlike MRI, MEG recording is silent. Patients or participants may be scanned during rest or during a variety of paradigms, from passive sensory processing to

active task performance, and the signals recorded can be characterized as spontaneous, evoked, or induced. Spontaneous activity is that which is not related to the presentation of a stimulus and thus constitutes background activity. Evoked activity is strictly time-locked to a stimulus, whereas induced activity arises in response to a stimulus but not in a strict time-locked manner.

In order to localize the brain activity recorded at the MEG sensors, the source/s of the activity must be modeled. In so doing, researchers and clinicians are often faced with an inverse problem, that is, the data (in this case, MEG signals recorded outside of the head at the sensors) are used to estimate the model parameters (in this case, the source/s of the MEG signals within the brain). These inverse situations are conceptualized as “problems” because there is no unique solution, and it can be challenging to identify the best possible solution.

MEG source modeling is typically accomplished by combining the MEG signals with MRI images. During MEG data collection, several coils are affixed to the individual's head (usually the nasion and left and right periauricular areas) to establish a three-dimensional coordinate system so that his or her MEG data may be coregistered with his or her structural MRI. The most widely utilized method for MEG source localization is the single equivalent current dipole (ECD) method. This method involves modeling the pattern of MEG activity at relevant sensors with the assumption that the source of the activity is a focal location in the brain (see Fig. 2). An alternative approach to the ECD method is beamforming, which uses rastered spatial filtering to identify the neuromagnetic time course of any given point in the brain. Repeating this process for many points in the brain allows researchers and clinicians to construct images in which the MEG activity of interest is overlaid on the structural MRI.

MEG is related to EEG as the two techniques capitalize on the same neurophysiological processes. However, there are several key distinguishing features of MEG. First, whereas EEG signals are susceptible to distortion due to the skull and scalp, MEG signals are impervious



Magnetoencephalography, Fig. 2 (a) Spatial and temporal information in MEG represented by the spatial array of sensors each recording the time-varying magnetic field; (b) at any instant, the magnetic field topography can be described; (c) source modeling is used to transition for “sensor space” to “brain, or source, space,” in this case,

two equivalent current dipole sources in bilateral superior temporal gyri accounting for auditory evoked neuromagnetic fields; (d) time courses of neuronal electrical activity from the two brain sources illustrated in (c) during auditory stimulation

M

to these structures, thus yielding better spatial resolution in MEG than EEG. Second, whereas EEG detects both tangential and radial currents associated with neural activity, MEG detects only tangential currents. This means that MEG is primarily sensitive to activity in sulci. Third, whereas EEG relies on a reference electrode/s, which can be problematic if the reference is active, MEG is reference-free.

Historical Background

To date, magnetoencephalography has been utilized predominantly for clinical purposes. The most common applications are identifying abnormal patterns of neural activity, locating seizure focus in epilepsy, and preoperative assessment of eloquent cortex in patients with intractable

epilepsy or brain tumors. In the latter, MEG findings have been demonstrated to be superior to EEG findings and consistent with (and perhaps eventually supplanting) invasive methods such as electrocorticography (ECoG).

Current Knowledge

Although MEG is widely considered the “gold standard” for noninvasive characterization of neuropathology in patients with epilepsy and brain tumors and the technique has demonstrated usefulness in the study of disorders such as Parkinson’s disease and schizophrenia, MEG has not been widely applied to the study of autism spectrum disorders (ASD). The earliest MEG studies of ASD were published in the late 1990s, and the relatively small ASD MEG literature can

be divided into the following broadly defined categories: (a) epileptiform abnormalities, (b) mirror neuron system integrity, (c) face processing, (d) the somatosensory system, and (e) auditory/language processing.

Epileptiform Abnormalities

One of the earliest MEG studies of ASD (Lewine et al. 1999) investigated the potential presence of abnormal patterns of neuromagnetic activity during sleep in children with regressive ASD, motivated by the substantial clinical overlap between autism and epilepsy. Results indicated a high prevalence of multifocal epileptiform activity in these children, even in the absence of a diagnosable seizure disorder. Similar epileptiform abnormalities have been subsequently reported in children with nonregressive forms of ASD (Muñoz-Yunta et al. 2008). Importantly, this line of research has demonstrated that epileptiform activity in ASD is detectable with MEG even in cases where it goes undetected by simultaneously recorded EEG.

Mirror Neuron System Integrity

Another small subset of the ASD MEG literature consists of studies examining the integrity of the mirror neuron system. Discovered serendipitously by Rizzolatti and colleagues (Gallese et al. 1996), who observed that neurons in a frontal area in monkeys fired not only when the monkey performed an action itself but also when it observed the experimenter performing a similar action, mirror neurons are purportedly involved in imitation and social cognition. Impairments in the mirror neuron system have been implicated in ASD, mostly with the use of EEG and fMRI. Although an early MEG study investigating electrophysiological signatures of biological motion perception in individuals with ASD (Avikainen et al. 1999) failed to identify abnormalities in ~20 Hz oscillatory activity in primary motor cortex, more recent work has identified spatio- and spectro-temporal abnormalities. Specifically, one finding indicated that although the peak activation latencies between adults with ASD and controls did not differ early in the processing stream (occipital cortex, followed by superior temporal

sulcus, followed by inferior parietal lobule), the ASD group showed a significantly delayed response downstream in the inferior frontal lobe (Broca's area), coupled with reductions in the amount of activity in the inferior frontal lobe as well as primary motor cortex (Nishitani et al. 2004). Results from another study revealed that the typical increase in beta (15–25 Hz) activity that occurs after an individual observes an action (the “postmovement beta rebound”) was reduced in adults with ASD in multiple regions in the mirror neuron system, including sensorimotor cortex, premotor cortex, and the superior temporal gyrus, as well as in the medial prefrontal cortex (Honaga et al. 2010).

Face Processing

Despite an extensive literature reporting face-processing abnormalities in ASD using fMRI, only two studies to date have examined face processing in ASD using MEG. One compared the processing of faces to that of mugs and geometrical patterns in adults with ASD (Bailey et al. 2005). Results indicated that the neuromagnetic response to faces at short latencies (30–60 ms) is abnormal in ASD, and equivalent current dipole estimation revealed abnormal face-processing loci in ASD. The second study examined face processing in children with ASD, specifically with respect to processing of faces with direct versus averted gaze (Kylliäinen et al. 2006). Findings demonstrated that at early latencies (up to 100 ms), neuromagnetic activity was similar in the ASD and control groups, save a lack of repetition priming in ASD. However, at longer latencies, group differences were more pronounced, with greater responses in ASD to direct versus averted gaze at 240 ms poststimulus (a pattern inconsistent with that of the control group) and weaker responses at 300 ms poststimulus in ASD compared to controls.

Somatosensory System

Only two studies to date have examined the somatosensory system in ASD. Examining young adults with ASD, one study (Coskun et al. 2009a) investigated the variability of evoked responses to tactile stimulation and failed to find

abnormalities in the ASD group. The other study examined the organization of somatosensory cortex via somatic maps and found abnormal cortical organization in young adults with ASD. Specifically, the cortical representations of the thumb and lip were further apart in the ASD group compared to the control group (Coskun et al. 2009b).

Auditory/Language Processing

The majority of the ASD MEG literature to date capitalizes on the ability of MEG to detect lateralized superior temporal gyrus (STG) activity in order to characterize auditory functioning in ASD, with the goal of elucidating biomarkers associated with language impairment in ASD. A number of studies have investigated auditory evoked responses to simple tones, with the hypothesis that impairments in the processing of simple auditory stimuli early in life would likely have cascading detrimental effects on language development. The evoked response most often examined is the M100, a ~100-ms response elicited in passive listening paradigms that is modulated by stimulus parameters such as frequency. Several studies have shown abnormalities in the M100 response in children with ASD. Among these abnormalities are prolonged M100 latencies (Gage et al. 2003; Gandal et al. 2010; Roberts et al. 2010), abnormal hemispheric asymmetry of the M100 (Schmidt et al. 2009), and abnormal modulation of the M100 by tones of different frequencies (Gage et al. 2003).

Another feature of auditory processing in ASD that MEG studies have revealed to be abnormal is the mismatch field (MMF). The MMF occurs approximately 200 ms after an auditory stimulus and is a measure of acoustic change detection obtained by subtracting the evoked response to a frequently presented standard stimulus from the evoked response to an infrequently presented deviant stimulus. The presence of an MMF indicates passive, preattentive discrimination of the standard and deviant stimuli. MEG studies have demonstrated latency delays in the MMF in children (Oram Cardy et al. 2005; Roberts et al. 2011) and adults (Kasai et al. 2005) with ASD as well as reduced amplitude, or even absence, of the MMF in children with ASD (Tecchio et al. 2003).

The M100 and MMF abnormalities may provide insight into the language impairments characteristic of ASD in that atypical early acoustic processing may have cascading downstream effects on the processing of language, which is inherently more complex and involves processing acoustic units presented in rapid temporal succession. Results from an MEG study of auditory processing of pairs of tones support this hypothesis; in children and adolescents with ASD, the majority of those with language impairment failed to show evoked responses to the second of two tones when the two tones were presented in rapid succession (Oram Cardy et al. 2005).

Future Directions

A number of novel approaches to the study of neural abnormalities in ASD are emerging in the MEG research community. One such approach is the study of resting-state neuromagnetic activity, which has the potential to shed light on abnormal patterns of neural activity in ASD in the absence of specific sensory processing. In addition, analyses of functional connectivity, or interactions between brain regions, both in the resting state and in sensory and cognitive processing are being implemented with increasing frequency. Functional connectivity analyses have the potential to provide insight into neural abnormalities in ASD at the network level. A clear benefit of MEG in this area of research is the ability to examine network interactions at multiple frequencies and with fine-grained temporal resolution.

Other exciting new applications of MEG in ASD research involve multimodal integration of MEG and complementary neuroimaging techniques, including diffusion tensor imaging (DTI) and magnetic resonance spectroscopy (MRS). Multimodal MEG and DTI studies afford the opportunity to investigate associations between electrophysiological measures (e.g., delayed auditory evoked 100-ms responses) and the structural integrity of white matter tracts. Multimodal MEG and MRS studies allow for investigation into associations between electrophysiological measures and neurotransmitter levels. Two particularly

promising targets in ASD research are GABA and glutamate. With the growing number of applications of MEG to ASD research emerges the potential to develop multidimensional classifiers that will help to further elucidate the neuropathology of ASD.

See Also

- [Electroencephalogram \(EEG\)](#)
- [fMRI](#)

References and Reading

- Avikainen, S., Kulomäki, T., & Hari, R. (1999). Normal movement reading in Asperger subjects. *Neuroreport*, 10, 3467–3470.
- Bailey, A. J., Braeutigam, S., Jousmäki, V., & Swithenby, S. J. (2005). Abnormal activation of face processing systems at early and intermediate latency in individuals with autism spectrum disorder: A magnetoencephalographic study. *European Journal of Neuroscience*, 21, 2575–2585.
- Braeutigam, S., Swithenby, S. J., & Bailey, A. J. (2008). Contextual integration the unusual way: A magnetoencephalographic study of responses to semantic violation in individuals with autism spectrum disorders. *European Journal of Neuroscience*, 27, 1026–1036.
- Coskun, M. A., Varghese, L., Reddoch, S., Castillo, E., Pearson, D., Loveland, K., et al. (2009a). How somatic cortical maps differ in autistic and typical brains. *Neuroreport*, 20, 175–179.
- Coskun, M. A., Varghese, L., Reddoch, S., Castillo, E., Pearson, D., Loveland, K., et al. (2009b). Increased response variability in autistic brains? *Neuroreport*, 20, 1543–1548.
- Gage, N. M., Siegel, B., Callen, M., & Roberts, T. P. L. (2003a). Cortical sound processing in children with autism disorder: An MEG investigation. *Neuroreport*, 14, 2047–2051.
- Gage, N. M., Siegel, B., & Roberts, T. P. L. (2003b). Cortical auditory system maturational abnormalities in children with autism disorder: An MEG investigation. *Developmental Brain Research*, 144, 201–209.
- Gallese, V., Fadiga, L., Fogassi, L., & Rizzolatti, G. (1996). Action recognition in the premotor cortex. *Brain*, 119, 593–609.
- Gandal, M. J., Edgar, J. C., Ehrlichman, R. S., Mehta, M., Roberts, T. P. L., & Siegel, S. J. (2010). Validating γ oscillations and delayed auditory responses as translational biomarkers of Autism. *Biological Psychiatry*, 68, 1100–1106.
- Honaga, E., Ishii, R., Kurimoto, R., Canuet, L., Ikezawa, K., Takahashi, H., et al. (2010). Post-movement beta rebound abnormality as indicator of mirror neuron system dysfunction in autistic spectrum disorder: An MEG study. *Neuroscience Letters*, 478, 141–145.
- Kasai, K., Hashimoto, O., Kawakubo, Y., Yumoto, M., Kamio, S., Itoh, K., et al. (2005). Delayed automatic detection of change in speech sounds in adults with autism: A magnetoencephalographic study. *Clinical Neurophysiology*, 116, 1655–1664.
- Kylliäinen, A., Braeutigam, S., Hietanen, J. K., Swithenby, S. J., & Bailey, A. J. (2006). Face- and gaze-sensitive neural responses in children with autism: A magnetoencephalographic study. *European Journal of Neuroscience*, 24, 2679–2690.
- Lewine, J. D., Andrews, R., Chez, M., Patil, A., Devinsky, O., Smith, M., et al. (1999). Magnetoencephalographic patterns of epileptiform activity in children with regressive autism spectrum disorders. *Pediatrics*, 104, 405–418.
- Muñoz-Yunta, J. A., Ortiz, T., Palau-Baduell, M., Martín-Muñoz, L., Salvadó-Salvadó, B., Valls-Santasusana, A., et al. (2008). Magnetoencephalographic pattern of epileptiform activity in children with early-onset autism spectrum disorders. *Clinical Neurophysiology*, 119, 626–634.
- Nishitani, N., Avikainen, S., & Hari, R. (2004). Abnormal imitation-related cortical activation sequences in Asperger's syndrome. *Annals of Neurology*, 55, 558–562.
- Oram Cardy, J. E., Flagg, E. J., Roberts, W., Brian, J., & Roberts, T. P. L. (2005a). Magnetoencephalography identifies rapid temporal processing deficit in autism and language impairment. *Neuroreport*, 16, 329–332.
- Oram Cardy, J. E., Flagg, E. J., Roberts, W., & Roberts, T. P. L. (2005b). Delayed mismatch field for speech and non-speech sounds in children with autism. *Neuroreport*, 16, 521–525.
- Roberts, T. P. L., Khan, S. Y., Rey, M., Monroe, J. F., Cannon, K., Blaskey, L., et al. (2010). MEG detection of delayed auditory evoked responses in autism spectrum disorders: Towards an imaging biomarker for autism. *Autism Research*, 3, 8–18.
- Roberts, T. P. L., Cannon, K. M., Tavabi, K., Blaskey, L., Khan, S. Y., Monroe, J. F., et al. (2011). Auditory magnetic mismatch field latency: A biomarker for language impairment in autism. *Biological Psychiatry*, 70(3), 263–269.
- Rojas, D. C., Maharajh, K., Teale, P. D., & Rogers, S. J. (2008). Reduced neural synchronization of gamma-band MEG oscillations in first-degree relatives of children with autism. *BMC Psychiatry*, 8, 66.
- Schmidt, G. L., Rey, M. M., Oram Cardy, J. E., & Roberts, T. P. L. (2009). Absence of M100 source asymmetry in autism associated with language functioning. *Neuroreport*, 20, 1037–1041.
- Tecchio, F., Benassi, F., Zappasodi, F., Gialloreti, L. E., Palermo, M., Seri, S., et al. (2003). Auditory sensory processing in autism: A magnetoencephalographic study. *Biological Psychiatry*, 54, 647–654.

Wilson, T. W., Rojas, D. C., Reite, M. L., Teale, P. D., & Rogers, S. J. (2007). Children and adolescents with autism exhibit reduced MEG steady-state gamma responses. *Biological Psychiatry*, 62, 192–197.

Mainstreaming

► Integration

Maintenance of Treatment Effects

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Synonyms

[Durability of treatment effects](#); [Sustained treatment benefits](#); [Treatment gain retention](#)

Definition

The continued improvement in functioning and/or use of skills gained through an intervention over time. Maintenance of behavior change may be strengthened through procedures such as the occasional use of the intervention methods originally used to promote behavior change, provision of reinforcement for displaying the newly learned behavior, and overlearning (teaching until the learner is fluent at displaying the new behavior). Maintenance of treatment effects is often addressed alongside generalization of skills to maximize the retention of treatment gains.

See Also

► [Generalization and Maintenance](#)

References and Reading

- Arnold-Saritepe, A. M., Phillips, K. J., Mudford, O. C., De Rozario, K. A., & Taylor, S. A. (2009). Generalization and maintenance. In J. L. Matson (Ed.), *Applied behavioral analysis for children with autism spectrum disorders* (pp. 207–224). New York: Springer.
- McEachin, J. J., Smith, T., & Lovaas, O. I. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. *American Journal of Mental Retardation*, 97(4), 359–372.
- Schreibman, L. (2000). Intensive behavioral/psychoeducational treatments for autism: research needs and future directions. *Journal of Autism and Developmental Disorders*, 30(5), 373–378.
- Smith, R. G., Vollmer, T. R., & St. Peter Pipkin, C. (2007). Functional approaches to assessment and treatment of problem behavior in persons with autism and related disabilities. In P. Sturmey & A. Fitzner (Eds.), *Autism spectrum disorders: Applied behavioral analysis: Evidence, and practice* (pp. 187–234). Austin: Pro-Ed.
- Stokes, T. F., & Baer, D. M. (1977). An implicit technology of generalization. *Journal of Applied Behavioral Analysis*, 20(2), 349–367.

Major Depression

► [Mood Disorders](#)

Major Depressive Disorder

► [Depressive Disorder](#)

Maladaptive Behavior

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Synonyms

[Problem behaviors](#)

Definition

Maladaptive behavior is defined as behavior that interferes with an individual's activities of daily living or ability to adjust to and participate in particular settings.

Maladaptive behaviors lie along a spectrum from more minor, less impairing behaviors (i.e., nail biting, difficulty separating) to more severely impairing behaviors (i.e., self-injurious or oversexualized behaviors) that seriously interfere with individuals' ability to maintain relationships with others, learn, and/or engage in adaptive, age-appropriate activities and settings. Because of their impairing nature, maladaptive behaviors are often the target of interventions. Problem behaviors are often a concern for children with developmental disabilities; maladaptive behaviors commonly associated with autism spectrum disorders include self-injurious behaviors (e.g., headbanging), stereotypies, aggression, and temper tantrums.

Although maladaptive behavior is related to adaptive behavior, or the personal and social skills necessary for day-to-day functioning, these two domains are distinct both conceptually and functionally. Thus, maladaptive behavior is not simply defined as the *absence* of adaptive behavior; reciprocally, adaptive behavior is not the absence of maladaptive behavior.

Maladaptive behavior is a term that is used most often in the discussion of adaptive behavior. In other contexts, maladaptive behaviors are usually considered under a broader umbrella of social-emotional and behavior problems that are measured with checklists.

children with autism and children with a history of language impairment. *Research in Developmental Disabilities*, 28, 145–167.

Horner, R. H., Carr, E. G., Strain, P. S., Todd, A. W., & Reed, H. K. (2002). Problem behavior interventions for young children with autism: A research synthesis. *Journal of Autism and Developmental Disorders*, 32, 423–446.

Matson, J. L., & Nebel-Schwalm, M. (2007). Assessing challenging behaviors in children with autism spectrum disorders: A review. *Research in Developmental Disabilities*, 28(6), 567–579.

Male Turner

► [Noonan and Ras/MAPK Pathway Syndromes](#)

Mand Fluency Training

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Definition

Mand fluency training, or mand training, is an intervention approach designed to teach individuals with limited communication skills to express their needs and wants. A mand commonly takes the form of a request and is defined as a verbal act that is reinforced by a specified consequence (Skinner 1957).

Historical Background

Mand training is based on B. F. Skinner's (1957) operant conditioning framework for analyzing the functions of verbal behavior. This behavioral approach to language training has been used extensively with individuals with developmental delays and autism, in particular, as part of early behavioral intervention programs. For example, intervention programs developed by Lovaas (2003) and Sundberg and colleagues (Sundberg

See Also

- [Adaptive Behavior Scales](#)
- [Maladaptive Behavior](#)
- [Vineland Adaptive Behavior Scales \(VABS\)](#)

References and Reading

Dominick, K. D., Davis, N. O., Lainhart, J., Tager-Fusberg, H., & Folstein, S. (2007). Atypical behaviors in

and Partington 1998) incorporate mand training as a key component for teaching early communication skills.

Rationale or Underlying Theory

Within an operant conditioning framework, mands are controlled by a motivative variable, often referred to as an establishing operation (Sundberg and Michael 2001). For example, hunger and the desire for a cookie may lead an individual to say “I want a cookie.” This behavior is then reinforced by the offering of a small piece of a cookie. A mand training approach can be contrasted with a traditional language perspective that emphasizes teaching the individual the meaning of different words (e.g., learning to say “cookie” when shown a cookie or “ball” when shown a ball; Sundberg and Michael 2001). It has been suggested, however, that some children with autism do not spontaneously request items even if they have demonstrated understanding of the appropriate word for an item and, thus, need to be explicitly taught this skill (Sundberg and Michael 2001). Because of the unique reinforcement provided through the establishing operation, mand training can facilitate children’s willingness to participate in language training, in general (Sundberg and Michael 2001).

Goals and Objectives

The ultimate goal of mand training is to increase an individual’s spontaneous requesting behavior so that the individual can make his or her needs and wants known to others. Short-term objectives may include imitating mands and requesting basic items, such as food within sight. Longer-term objectives may include requesting items or activities that are not present in the immediate environment.

Treatment Participants

Participants in mand training are individuals, typically young children, with developmental

disabilities who have limited expressive language skills and who do not spontaneously make verbal requests to express their needs and wants.

Treatment Procedures

Mand training can be used as part of a larger early intervention program focused on developing language, cognitive, self-help, and play skills. For example, the *I Want* program (Lovaas 2003), which is one component of a large package of intervention techniques for individuals with developmental delays, is designed to teach participants to verbalize their choices when presented with desired and undesired items and to develop spontaneous verbal requesting behavior. The developer of the program recommends starting with teaching the participant to request his or her favorite foods, objects, or activities to reinforce the behavior and possibly reduce problem behavior resulting from frustration with not being able to communicate desires (Lovaas). If necessary, the individual would first need to be taught how to imitate several “I want (item)” statements (e.g., *I want ball*, *I want cookie*, *I want juice*). The participant is then presented with the stimulus “What do you want?” and prompted to say “I want juice,” for example. The participant’s response is then reinforced by having access to a small amount of the item (e.g., a sip of juice, 5 s of playtime with the ball). As the participant reaches specified accuracy benchmarks, the prompts are faded and new items are introduced. Eventually the participant is prompted to request items that are not in sight and then progresses to being reinforced systemically for spontaneous requests that are not prompted. For individuals who are unable to echo sounds or imitate actions, mand training can be done using signs or pictures.

Important issues to include when selecting initial words for mand training include:

- (a) Choosing words that will be reinforcing to the participant and that the instructor can easily control, have access to, and that can be delivered multiple times

- (b) Selecting words the participant can imitate or for which the individual has demonstrated understanding
- (c) Choosing words that are relatively easy for the participant to say
- (d) When using sign language, selecting signs that look like the objects they represent and that the individual can already imitate
- (e) Choosing words for items that are relevant to the participant's daily life
- (f) Selecting words for a variety of motivators (e.g., not all foods)
- (g) Avoiding words that sound alike or rhyme
- (h) Avoiding words that may represent something negative for the individual (Sundberg and Partington 1998)

Efficacy Information

Mand training is often implemented as part of a broader behavioral intervention program, and its efficacy has not been examined extensively in isolation. However, smaller studies have provided some evidence for the efficacy of mand training in children with autism (e.g., Drash et al. 1999). There is also some evidence that it may be more efficient than discrete trial instruction for facilitating the acquisition of requesting behavior (Jennett et al. 2008). Adding opportunities for rapid imitation of modeled motor behaviors during mand training has been shown to increase the efficacy of mand training, with evidence of increased spontaneous manding 3 months later (Ross and Greer 2003). In general, however, there is limited information from published studies on the long-term generalization of skills taught during mand training.

Outcome Measurement

Efficacy research on mand training has utilized various metrics for progress monitoring and outcomes measurement, such as specified trial accuracy criteria (e.g., 9 out of 10 correct responses), percentage of correct responses, percentage of incorrect responses, or the number of spontaneous

mands in a given period. Clinical intervention programs that include mand training typically use specific accuracy criteria for determining when an individual is ready to progress to the next stage (e.g., from imitating mand to being expected to produce them when prompted).

Qualifications of Treatment Providers

Mand training providers are typically professionals or supervised paraprofessionals with formal training in behavioral theory and applied behavioral analysis techniques. In some verbal behavior programs, parents and caregivers are also taught basic mand training techniques to facilitate generalization of the individual's skills beyond the therapy context.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Communication Interventions](#)
- [Early Intensive Behavioral Intervention \(EIBI\)](#)
- [Educational Interventions](#)
- [Functional Communication Training](#)
- [Language Interventions](#)
- [Mands](#)
- [Operant Conditioning](#)
- [Skinnerian Categories of Verbal Behavior](#)
- [Verbal Behavior Interventions](#)

References and Reading

- Drash, P. W., High, R. L., & Tudor, R. M. (1999). Using mand training to establish an echoic repertoire in young children with autism. *The Analysis of Verbal Behavior*, 16, 29–44.
- Jennett, H. K., Harris, S. L., & Delmolino, L. (2008). Discrete trial instruction vs. mand training for teaching children with autism to make requests. *The Analysis of Verbal Behavior*, 24, 69–85.
- Lovaas, O. I. (2003). *Teaching individuals with developmental delays: Basic intervention techniques*. Austin: Pro-Ed.
- Ross, D. E., & Greer, R. D. (2003). Generalized imitation and the mand: Inducing first instances of speech in young children with autism. *Research in Developmental Disabilities*, 24, 58–74.

- Skinner, B. F. (1957). *Verbal behavior*. New York: Appleton-Century-Crofts.
- Sundberg, M. L., & Michael, J. (2001). The benefits of Skinner's analysis of verbal behavior for children with autism. *Behavior Modification*, 25, 698–724.
- Sundberg, M. L., & Partington, J. W. (1998). *Teaching language to children with autism or other developmental disabilities*. Danville: Behavior Analysts.

References and Reading

- Skinner, B. F. (1957). *Verbal behavior*. New York: Appleton-Century-Crofts.
- Sundberg, M. L., & Michael, J. (2001). The benefits of Skinner's analysis of verbal behavior for children with autism. *Behavior Modification*, 25, 698–724.

Mands

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Mania

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Synonyms

[Command](#); [Demand](#); [Request](#)

Synonyms

[Elated, euphoric and grandiose](#); [Elevated, expansive, or irritable mood](#); [Overly talkative](#); [Racing thoughts](#), [distractibility](#), and [psychomotor agitation](#)

Definition

Mands are verbal acts that are reinforced by a specified consequence (Skinner 1957). This term was introduced by B. F. Skinner and refers to a specific type of verbal action within an operant conditioning framework designed to analyze the functions of verbal behavior. Mands are considered to be the first type of verbal behavior that individuals typically acquire (Sundberg and Michael 2001). As part of a verbal behavior training intervention, mands commonly take the form of requests for items, information, or removal of aversive stimuli (Sundberg and Michael 2001). For example, if an individual who wants a cookie says “Cookie, please” and is then given a cookie, the individual’s statement (i.e., “Cookie, please”) would be considered a mand.

Short Description or Definition

The American Psychiatric Association (APA) Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) defines a manic episode as “a distinct period during which there is an abnormally and persistently elevated, expansive, or irritable mood” (American Psychiatric Association [APA] 2000, p. 357). Symptoms of mania include a high activity level which may be directed toward a specific goal or may consist of psychomotor agitation, inflated self-esteem, a decreased need for sleep, racing thoughts, talkativeness, distractibility, and engaging in pleasure-seeking activities without regard for consequences. Individuals experiencing mania are often noted to have a change in personality, in which they become reckless, boisterous, and engage in high-risk activities. If the individual’s mood is elevated, three of the prior symptoms are required; four if the mood is irritable. To qualify as a manic episode in DSM-IV-TR, the episode must last at least one week, or any duration if symptoms are severe enough to require hospitalization, and must result

See Also

- ▶ [Applied Behavior Analysis \(ABA\)](#)
- ▶ [Mand Fluency Training](#)
- ▶ [Operant Conditioning](#)
- ▶ [Skinnerian Categories of Verbal Behavior](#)
- ▶ [Verbal Behavior Interventions](#)

in impairment in the ability to function in daily life. Psychotic features, including delusions (false beliefs) or hallucinations, may be present. The manic episode cannot be caused by the effects of drugs of abuse, medications, exposure to a toxic substance, or a general medical condition (APA 2000).

Categorization

A manic episode is one of the essential components of bipolar I disorder, which historically has been referred to as manic-depressive disorder. Although bipolar I disorder usually also includes episodes of depression, it is the occurrence of the manic episode that is the defining feature. A hypomanic episode is a less severe form of mania, which is not severe enough to cause substantial impairment in functioning in daily life, does not necessitate hospitalization, and there are no psychotic symptoms. Bipolar II disorder is characterized as a mood disorder in which hypomania, as opposed to mania, is present (APA 2000).

Epidemiology

The lifetime prevalence of bipolar I disorder in the community is estimated at 3.9% (Perlis et al. 2004). There is less diagnostic clarity when the mood symptoms have their initial symptoms presenting in childhood, as many other disorders have overlapping symptoms. Twenty is the mean age of onset for a first manic episode, but retrospective studies suggest that about 60% of adults with bipolar disorder demonstrated some mood symptoms in childhood or adolescence. There has been a trend over time for the age of the first onset of symptoms to occur earlier in life. For adolescents who have suffered from recurrent major depression, about 10–15% will experience a manic episode and thus develop bipolar I disorder (Boris et al. 2007). The lifetime prevalence of bipolar I disorder in the community is estimated at 3.9% (Perlis et al. 2004). There is less diagnostic clarity when the mood symptoms have

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Men and women suffer from bipolar I disorder in about equal numbers. However, women more commonly have a major depressive episode first and suffer from more depressive episodes than manic episodes, whereas men most often experience their first episode as mania and have more equal numbers of manic and depressive episodes. Manic episodes frequently occur after a stressful life event. In about 2/3 of cases, a manic episode immediately follows or precedes an episode of major depression. Most individuals have periods of more normal mood states between episodes of either mania or depression. However, about one-quarter of individuals with bipolar I disorder more persistently display a labile (fluctuating) or irritable mood (APA 2000). In individuals over the age of 13 who are diagnosed with autism, approximately 3% suffer from manic episodes (Howlin 2005).

Natural History, Prognostic Factors, and Outcomes

Mania, associated with bipolar disorder is a recurrent illness. In general, the earlier the age of onset of the disorder (either depressive or manic symptoms), the more recurrent and chronic the course of illness. Recovery from the first manic episode is very high (over 70%), but a similar number (up to 80%) will experience recurrence of the disorder within 2–5 years.

Early-onset bipolar disorder also has a high rate of persistent lower level mood symptoms. Child and adolescent onset of the disorder is often characterized by more rapid fluctuations in

mood than when the disorder is not evidenced until adulthood.

Poor prognostic factors for manic episodes are early age of onset, long duration of symptoms, exposure to trauma and negative life events, low socioeconomic status, mixed or rapid cycling episodes, psychotic symptoms, and co-occurring psychiatric disorders. Suicidal ideation and attempts occur most often when an individual is depressed or using substances. For individuals with bipolar I disorder, the completed suicide rate is estimated at 10–15% (Geller et al. 2004).

Clinical Expression and Pathophysiology

Elevated mood is the cardinal symptoms of mania. Individuals who experience mania often feel so elated and omnipotent that they do not recognize that they are ill in any way. It is those around them that notice the change in behavior. The manic individual often demonstrates poor judgment, may change personal appearance to a flamboyant style that is out of character, and may exhibit high-risk behaviors, such as sexual promiscuity, substance use, or spending large sums of money. For youth presenting with mania, increased energy, distractibility, and pressured speech are the most common symptoms, while hypersexuality has been found to be the least frequent (Boris et al. 2007).

Very severe mania often includes psychotic symptoms. More than half of manic individuals experience delusions, usually of grandeur (the false belief that one is famous, has unusual powers, or is special in some other way). Religious delusions are also common. Hallucinations, usually auditory, occur in about one-fourth of individuals. Some individuals may experience symptoms of mania and major depression that alternate rapidly, with both experienced almost every day. This is referred to as a “mixed episode.” Agitation, insomnia, rapid changes in appetite, psychotic features, and suicidal thoughts are frequent symptoms for individuals with mixed episode mood disorders (APA 2000).

Although there are no specific laboratory tests that are diagnostic of a manic episode, some

abnormal test results are frequently found in individuals with mania. These include abnormal polysomnograms (electroencephalograms or EEGs that occur during sleep) and increased cortisol secretion. Studies of neurotransmitter metabolites, neuroendocrine function, and pharmacological challenges suggest that there may be abnormalities of many of the central nervous system neurotransmitters: norepinephrine, serotonin, acetylcholine, dopamine, and gamma-aminobutyric acid (GABA).

Bipolar disorder has been demonstrated to run in families, according to multiple twin and adoption studies. Having a first-degree relative who has been diagnosed with bipolar disorder confers an increased risk (eight- to tenfold over community samples) of also being diagnosed with the disorder. Symptoms typically occur at an earlier age in children with a close family history of bipolar disorder. The heritability for bipolar disorder has been estimated at over 80% (Boris et al. 2007).

Evaluation and Differential Diagnosis

The evaluation for mania includes a patient interview, collateral information from significant others, a medical workup, gathering family history of mood disorder, and the use of assessment scales (see Table 1). Structured and semi-structured interviews (SADS or for children the K-SADS) or a general checklist, such as the Child Behavior Checklist (CBCL), may be helpful diagnostically and for research purposes. For youth, the Young Mania Rating Scale (Young et al. 1978) is the most widely used. Also for youth, parental report is more effective in identifying mania than youth or teacher reports. The Parent Mood Disorder Questionnaire (MDQ) is a widely used rating scale. Mood timelines or diaries may help track mood symptoms, and may also identify events that trigger these symptoms and document response to treatment (Nandagopal and DelBello 2011).

Many other disorders have overlapping symptoms with early-onset bipolar disorder. Attention-deficit/hyperactivity disorder, disruptive behavior disorders and conduct problems, substance abuse disorders, schizophrenia, and pervasive developmental disorder with irritability are most common.

Mania, Table 1 Components of a diagnostic evaluation for mania

Chief complaint	From patient, family, significant others, referral source
Context	In what context does mania occur?
Multiple informants and collateral history	History of symptoms from family, significant others, referral source, primary care physician, etc. (with patient consent or from legal guardian if patient is a minor)
Timeline of mood symptoms	Frequency, intensity, number, and duration (FIND) of mood episodes
Developmental, social, and family history	Developmental milestones, educational history, level of functioning, social relationships, medical history, and family history of psychiatric disorders
Patient interview/mental status examination	Developmentally appropriate interview. Inquire about substance use, high-risk behaviors, suicidality and homicidality, legal problems, trauma history, sleep, and energy level. Assess for psychotic symptoms
Medical evaluation	Routine physical examination; further medical evaluation as indicated: illicit substances (toxicology screen); toxic (lead or toxin levels); autoimmune diseases (screen for lupus, and others); neurological (EEG for epilepsy; brain imaging for multiple sclerosis, etc.); metabolic/infections (thyroid levels, liver function tests, complete blood count, etc.); other diseases (e.g., Cushing's disease by cortisol level)
Rating scales	SADS, K-SADS, Young Mania Rating Scale, Child Behavior Checklist (CBCL), Parent Mood Disorder Questionnaire (MDQ)

Mania, Table 2 Treatment of mania

Treatment: pharmacotherapy	
Lithium	Most evidence base for effectiveness in adults and adolescents. Used as monotherapy or in combination with an antipsychotic or anticonvulsant medication
Anticonvulsant medications: valproate, carbamazepine, lamotrigine, oxcarbazepine, topiramate	The most evidence for effectiveness in adults and adolescents are with valproate, carbamazepine, and lamotrigine. Used as monotherapy or in combination with lithium or an antipsychotic
Atypical antipsychotic medications: olanzapine, risperidone, quetiapine, aripiprazole, clozapine, ziprasidone	May be used as monotherapy but are often used to target symptoms of psychosis or for more rapid treatment response in conjunction with lithium or an anticonvulsant medication
Treatment: psychosocial	
Environmental	Safe and less stimulating environment, sleep hygiene, and routine. For severe mania, this may include psychiatric hospitalization
Psychoeducation and support	Active listening; restoration of hope; case management (collaborate with school, job, community resources, family regarding needed services); education about causes, symptoms, treatment, and prognosis
Therapeutic interventions	Day treatment or partial hospitalization program; individual therapy: cognitive-behavioral therapy, dialectic behavioral therapy, interpersonal therapy, supportive therapy; family therapy; skill-building groups

Medical disorders that may present with manic symptoms include hyperthyroidism, head trauma, brain tumors, or Cushing's syndrome. Medications, such as corticosteroids, antidepressants, and

stimulants, may be accompanied by mood fluctuations or precipitate manic symptoms.

In individuals diagnosed with autism spectrum disorders, the most common other psychiatric

diagnoses are attention-deficit/hyperactivity disorder, depression, and anxiety disorders, with mania occurring with no more frequency than the rest of the population (Howlin 2005; Matson and Nebel-Schwalm 2007).

Treatment

Current treatments for mania aim to control the agitation, impulsivity, aggression, and psychotic symptoms and to help patients regain their pre-illness level of functioning. After the first episode of mania, many individuals demonstrate ongoing milder levels of dysfunction, which may or may not resolve fully. Mania, and thus, bipolar I disorder, is a recurrent disorder, and effective treatment must include acute, continuation, and maintenance phases. Effective treatment includes the use of appropriate medications in the mood stabilizing, antiepileptic, and antipsychotic classes. These medications may be used in monotherapy or combined for enhanced effectiveness. Psychosocial interventions, including education about the disease, case management and coordination of services, family therapy, group therapy, and individual supportive and evidence-based treatments are also essential to optimize prognosis. Table 2 highlights the components of effective treatment.

See Also

► [Comorbidity](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Revision). Arlington: Author.
- American Psychiatric Association. (2002). Practice guideline for the treatment of patients with bipolar disorder (revision). *American Journal of Psychiatry*, 159, 1–50.
- Boris, B., Axelson, D., & Pavuluri, M. (2007). Bipolar disorder. In A. Martin & F. R. Volkmar (Eds.), *Lewis's child and adolescent psychiatry: A comprehensive textbook* (pp. 513–528). Philadelphia: Lippincott Williams & Wilkins.

- Geller, B., Tillman, R., Craney, J. L., & Bolhofner, K. (2004). Four-year prospective outcome and natural history of mania in children with a prepubertal and early adolescent bipolar disorder phenotype. *Archives of General Psychiatry*, 61, 459–467.
- Howlin, P. (2005). Outcomes of autism spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders, volume 1: Diagnosis, development, neurobiology and behavior* (3rd ed., pp. 201–222). Hoboken: Wiley.
- Leibenluft, E., Charney, D. D., Towbin, K. E., Bhangoo, R. K., & Pine, D. S. (2003). Defining clinical phenotypes of juvenile mania. *American Journal of Psychiatry*, 160, 430–437.
- Matson, J. L., & Nebel-Schwalm, M. S. (2007). Comorbid psychopathology with autism spectrum disorder in children: An overview. *Research in Developmental Disabilities*, 28(4), 341–352.
- Nandagopal, J. J., & DelBello, M. P. (2011). Assessment and treatment of childhood and adolescent bipolar disorder. In A. Martin, L. Seahill, & C. Kratochvil (Eds.), *Pediatric psychopharmacology: Principles and practice* (pp. 466–479). New York: Oxford University Press.
- Perlis, R. H., Miyahara, S., Marangell, L. B., et al. (2004). Long-term implications of early onset in bipolar disorder: Data from the first 1000 participants in the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD). *Biological Psychiatry*, 55, 875–881.
- Young, R. C., Biggs, J. T., Ziegler, V. E., & Meyer, D. A. (1978). A rating scale for mania: Reliability, validity and sensitivity. *British Journal of Psychiatry*, 133, 429–435.

Manual Sign

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Synonyms

[American Sign Language \(ASL\)](#); [Conceptually accurate signed english \(CASE\)](#); [Linguistics of verbal english \(LOVE\)](#); [Manually coded english \(MCE\)](#); [Seeing essential english \(SEE I\)](#); [Sign language](#); [Signed english](#); [Signing exact english \(SEE II\)](#)

Definition

Manual sign is a system of communicating visually and spatially through signs created with the hands. The term “manual sign” does not specify a particular sign system such as American Sign Language (ASL) or manual codes of English such as Seeing Essential English (SEE I), Signing Exact English (SEE II), Linguistics of Verbal English (LOVE), and Conceptually Accurate Signed English (CASE). ASL is the natural language of the deaf community in the United States and much of Canada (Neidel et al. 2000). ASL is a distinct language from spoken English (see entry on ► [“American Sign Language \(ASL\)”](#)) while manual codes of English are based on spoken English and are an attempt to represent English on the hands. Manual codes of English were created in the 1960s primarily to aid in increasing the literacy skills of deaf and hard-of-hearing children (Schow and Nerbonne 2007). Manual codes of English can be classified as “sign systems” and not “sign languages” because they borrow the semantics and syntax from spoken English. These sign systems follow English word order and use grammatical markers for morphological endings of words. For example, the word “smiling” is signed with two signs SMILE + ing. Additionally, unlike ASL, manual codes of English use signs for function words such as “the” and “an.” Typically, when signing is employed in the language intervention for children with autism, the type of signing systems that are used is manual codes of English rather than ASL. The main reason for this may be the hope that using manual sign will lead to verbal communication, an outcome which has been supported by research (Schlosser and Wendt 2008).

See Also

- [American Sign Language \(ASL\)](#)
- [Sign Language](#)

References and Reading

Neidel, C., Kegl, J., MacLaughlin, C., Bahan, B., & Lee, R. G. (2000). *The syntax of American sign language*. Cambridge, MA: The MIT Press.

Schlosser, R. W., & Wendt, O. (2008). Effects of augmentative and alternative communication intervention on speech production in children with autism: A systematic review. *American Journal of Speech-Language Pathology*, 17, 212–230.

Schow, R. L., & Nerbonne, M. A. (2007). *Introduction to audiologic rehabilitation* (5th ed.). Boston: Pearson.

Manually Coded English (MCE)

- [Manual Sign](#)
- [Sign Language](#)

Marker

- [Milestone](#)

Marketed as Geodon

- [Geodon \(Ziprasidone\)](#)

MAS

- [Motivation Assessment Scale](#)

Massed Practice

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Definition

The repeated presentation of a task over a short period of time, with no intertrial interval between presentations. The term was first introduced by Hermann Ebbinghaus, and a detailed study was published in the 1885 book *Memory: A Contribution to Experimental Psychology*. Since then, researchers have found that distributed

practice, or spaced practice, in which short periods of learning are interspersed with periods of rest or alternative activities, produces better performance than massed practice in a variety of tasks (Hunter 1929).

Studies investigating the influence of task variation on learning suggest that distributed practice produces superior levels of on-task responding and higher levels of child affect in children with autism (Dunlap 1984). Researchers suggest that interspersing alternative activities with varying difficulty levels serves to increase the child with autism's motivation to respond to the tasks (Koegel and Egel 1979).

Massed practice can also refer to a self-directed behavior change technique in which a person performs an undesired behavior repeatedly, which sometimes decreases the future frequency of the behavior (Cooper et al. 2007).

See Also

- [Distributed Practice](#)
- [Learning Styles](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson Education.
- Dunlap, G. (1984). The influence of task variation and maintenance tasks on the learning and affect of Autistic children. *Journal of Experimental Child Psychology*, 37, 41–64.
- Ebbinghaus, H. (1885). *Memory: A contribution to experimental psychology*. Teachers College: Columbia University.
- Hunter, W. S. (1929). Learning: II. Experimental studies of learning. In C. Murchison (Ed.), *The foundations of experimental psychology* (pp. 564–627). Worcester: Clark University Press.
- Koegel, R. L., & Egel, A. (1979). Motivating Autistic children. *Journal of Abnormal Psychology*, 88(4), 418–426.

Massively Parallel Sequencing

- [Next-Generation Sequencing](#)

Mast Cells and Autism

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Synonyms

[Mastocyte](#)

Definition

Despite the increase prevalence of autism spectrum disorders (ASD), the pathogenesis and therapeutic options for ASD remain poorly understood (Theoharides et al. 2012). In recent years, several reports have suggested the importance of brain inflammation in the pathogenesis of several psychiatric conditions (Theoharides et al. 2016). Among ASD patients, many develop worsening symptoms after vaccination, infections, toxins, or other environmental exposures suggesting an underlying immune dysregulation (Theoharides et al. 2016). Specifically, increased inflammatory cytokines, autoantibodies, and allergic responses reported in ASD patients suggest mast cells may have a prominent role in the onset and progression of ASD (Theoharides et al. 2016). Patients with increased mast cells have an increased prevalence of ASD and other cognitive deficits, suggesting an underlying mast cell hyperactivity among ASD patients (Theoharides 2013). Mast cells are bone marrow-derived cells that modulate the innate immune response and contribute several chronic inflammatory and renal diseases (Balakumar et al. 2009; Holdsworth and Summers 2008; Wasse et al. 2012). Within the central nervous system, mast cells are

Massive Infantile Spasms

- [Infantile Spasms/West Syndrome](#)

concentrated in the diencephalon, which modulates several emotional and behavior responses (Theoharides et al. 2016). In recent years, mast cell interactions with microglial have been implicated in the pathogenesis of ASD and other neuroinflammatory and psychiatric disorders (Theoharides et al. 2016). Several studies suggest neuroinflammation interferes with mast cell-microglial interactions from pruning neural circuits during neurodevelopment producing abnormal microglial growth and activation in ASD patients (Theoharides et al. 2016). Specifically, environmental and psychological stresses during prenatal development may stimulate the release of potent neuropeptides, neurotensin, and corticotropin-releasing factor and stimulate mast cells to increase vascular permeability and disrupt the blood-brain barrier in ASD patients (Theoharides et al. 2016). With the lack of effective treatments for ASD, pharmacological treatments targeting mast cell-microglial interactions may provide a new treatment options for ASD patients and their caregivers (Tsiloni et al. 2015).

See Also

- Allergies
- Leaky Gut Syndrome
- Intestinal Permeability Studies

References and Reading

- Balakumar, P., Reddy, J., & Singh, M. (2009). Do resident renal mast cells play a role in the pathogenesis of diabetic nephropathy? *Molecular and Cellular Biochemistry*, 330 (1–2), 187–192. <https://doi.org/10.1007/s11010-009-0132-3>.
- Holdsworth, S. R., & Summers, S. A. (2008). Role of mast cells in progressive renal diseases. *Journals of the American Society of Nephrology*, 19(12), 2254–2261. <https://doi.org/10.1681/asn.2008010015>.
- Theoharides, T. C. (2013). Is a subtype of autism an allergy of the brain? *Clinical Therapeutics*, 35(5), 584–591. <https://doi.org/10.1016/j.clinthera.2013.04.009>.
- Theoharides, T. C., Angelidou, A., Alysandratos, K.-D., Zhang, B., Asadi, S., Francis, K., et al. (2012). Mast cell activation and autism. *Biochimica et Biophysica Acta (BBA) – Molecular Basis of Disease*, 1822(1), 34–41. <https://doi.org/10.1016/j.bbadis.2010.12.017>.

- Theoharides, T. C., Stewart, J. M., Panagiotidou, S., & Melamed, I. (2016). Mast cells, brain inflammation and autism. *European Journal of Pharmacology*, 778, 96–102. <https://doi.org/10.1016/j.ejphar.2015.03.086>.
- Tsilioni, I., Taliou, A., Francis, K., & Theoharides, T. C. (2015). Children with autism spectrum disorders, who improved with a luteolin-containing dietary formulation, show reduced serum levels of TNF and IL-6. *Translational Psychiatry*, 5(9), e647–e647. <https://doi.org/10.1038/tp.2015.142>.
- Wasse, H., Naqvi, N., & Husain, A. (2012). Impact of mast cell chymase on renal disease progression. *Current Hypertension Reviews*, 8(1), 15–23. <https://doi.org/10.2174/157340212800505007>.

Mastocyte

- Mast Cells and Autism

Maternal and Child Health Bureau

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Major Areas or Mission Statement

The Maternal Child Health Bureau (MCHB), a United States Federal commitment to addressing maternal and child health, was first established in 1912. In 1935, the MCHB was transferred to Title V of the Social Security Act and then converted to a federal block grant administered by the Health Resources and Services Administration (HRSA) in 1981. The mission of the MCHB is to provide leadership, in partnership with key stakeholders, to improve the physical and mental health, safety, and well-being of the nation's women, infants, children, adolescents, and their families, including fathers and children with special health-care needs.

Landmark Contributions

HRSA administers a wide range of programs to pregnant women, mothers, infants, children, and their families. MCHB programs serve more than 34 million women, infants, and children each year in the United States. The MCHB supports programs to reduce infant mortality by providing comprehensive prenatal and postnatal care for women, promotes an advocacy community-based group for Healthy Start programs, and is a proponent of preventative care (including rehabilitative services and immunizations) for children, research and training, genetic services, and newborn screening and treatments.

The HRSA awards grants to improve health care and other services for children and adolescents with autism spectrum disorders and other developmental disabilities. Funds are utilized to enhance awareness and reduce barriers to screening and diagnosis. Funds also support research of evidence-based interventions, evidence-based guidelines for interventions, training professionals to use valid screening tools for diagnosis and intervention, and improving technical assistance to facilities which provide services for children with autism spectrum disorders.

Major Activities

HRSA's implementation of the Combating Autism Act of 2006 (\$48 million) addresses the most urgent issues affecting people with autism and their families. The goal of this act is to enable all infants, children, and adolescents who are at risk for developing or who have autism spectrum disorders and other developmental disabilities to reach their full potential by:

- Establishing a system of services, including early screening of children for autism spectrum disorders
- Conducting early, multidisciplinary evaluations to diagnose or rule out autism spectrum disorders
- Providing evidence-based early interventions when a diagnosis is confirmed

References and Reading

Maternal and Child Health. (2010). Retrieved 17 July 2011, from <http://mchb.hrsa.gov/>

Maternal Hypothyroidism and Autism

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Synonyms

Iodothyronine deiodinase (D2T3); Thyrotropin, Thyroid-stimulating hormone (TSH); Thyroxine (T)

Structure

The thyroid gland is part of the endocrine system. It regulates the rate of metabolism and impacts growth by its regulation of other tissues through secretion of hormones triiodothyronine (T3) and thyroxine (T4). The substrates these hormones are synthesized from are iodine and tyrosine. The rate that these hormones are produced is regulated by thyroid-stimulating hormone (TSH) from the anterior pituitary gland. TSH is itself regulated by thyrotropin-releasing hormone (TRH) produced by the hypothalamus. The fetus begins to make T3 at 18–20 weeks' gestation and T4 by 30 weeks. This may be partially protective from maternal hypothyroidism.

Function

Thyroid hormones are critical to normal brain development.

The thyroid regulates metabolism and growth and impacts both calcium metabolism and catecholamine effects.

Typical symptoms of hypothyroidism (low hormone production) postnatally are decreased energy, constipation, weight gain, fatigue, cold intolerance, hair loss, and slowed heart rate. Typical symptoms of hyperthyroidism (increased hormone production) postnatally are mood swings, overactivity, decreased appetite, and increased heart rate.

Pathophysiology

Among the prenatal conditions that have been associated with autism spectrum disorders is prenatal exposure to maternal hypothyroidism. Although not clearly demonstrated as a cause of autism, this hypothesis has growing support from neuroembryonic research, and some have suggested links between environmental toxins, maternal hypothyroidism, and the autism “epidemic.”

Twenty-five years ago, Gillberg et al. (1992) noted that congenital hypothyroidism was associated with autism spectrum disorders, along with intellectual disability and other physical concerns. In 1999, Haddow et al. confirmed that children exposed in utero to maternal hypothyroidism manifesting as high gestational thyrotropin (thyroid-stimulating hormone, or TSH) levels, arising in response to relative low levels of thyroid hormone, resulted in significant neuropsychologic impacts at school age. Developmental abnormalities were present even in children of women whose hypothyroidism was subclinical or partially treated (Bernal 2007).

Thyroid hormone receptors are present in the fetal cortex by 8–9 weeks of gestation and increase tenfold by 18 weeks (Bernal 2007), and brain T₃ and iodothyronine deiodinase (D₂) activities increase during the second trimester (Kester et al. 2004). D₂ is felt to play an important role in brain compensation for variations in ambient thyroid hormone levels. It is well established that thyroid hormone plays a role in brain development during the fetal period, that different parts of the brain are differentially sensitive to thyroid hormone at any one time during development, and that sensitivity to

thyroid hormone is controlled, in part, by local control of hormone production. These observations imply that the consequences of thyroid hormone insufficiency during fetal development differ from those of thyroid hormone insufficiency during postnatal development (Zoeller 2005). However, it has also been shown that small fluctuations in maternal thyroid hormone levels can have measurable neurocognitive impacts on the developing fetus. One impact of hypothyroidism shown in animal models is reduced expression of reelin, which regulates neuronal migration (Alvarez-Dolado et al. 1999). Abnormality of reelin expression has been associated with ASDs (Fatemi et al. 2005), and although not universally accepted, abnormalities of reelin genes have been associated with ASDs (Serajee et al. 2006; He et al. 2011).

It has been noted that thyroid hormone deficiency before the onset of hearing has negative effects on peripheral and central auditory systems (Knipper et al. 2000). In animal models, maternal hypothyroidism has been shown to result in abnormal cochlear development, in abnormal neurotransmission in the developing auditory brainstem and hippocampus (Friauf et al. 2008) and in cerebrocortical and hippocampal synaptic activity (Taylor et al. 2008). Based on these and other animal studies, it has been proposed that the rat exposed to mild, transient neonatal hypothyroidism may serve as a model for autism (Sadamatsu et al. 2006).

High TSH levels have also been noted to be associated with increased risk of breech presentation in term infants, based on samples of Dutch women followed for TSH elevation or evaluated for breech presentation (Kooistra et al. 2010). Inasmuch as breech presentation is one risk factor for ASDs (Bilder et al. 2009), this supports the relationship. The authors note that infants who present in the breech position often have low tone or reasons for less active body movements such as kicking and that suboptimal maternal thyroid function might have a direct motor effect on the fetus. Alternatively, they quote literature (Van der Meulen et al. 2008) that suggests that the sensory responses of fetuses in breech position are different from those in cephalic presentation.

Sweeten et al. (2003) replicated the findings of Comi et al. (1999), noting increased frequency of autoimmune disorders, with hypothyroidism being the most common, among parents of children with autism spectrum disorders. They acknowledge that this could be an effect of endocrine abnormality or, alternatively, the result of another immunologic abnormality having a direct effect on brain development. A meta-analysis by Wu et al. (2015) and supported by Brown et al. (2015) reasserted this association based on pooled data, with an increased risk overall and greater risk specifically with maternal hypothyroidism.

An extensive review of the hypothyroidism/ASD association by Román (2007) draws an association between maternal hypothyroidism and neuropathologic features found in individuals with ASDs, including abnormalities in the cortex and hippocampus, “consistent with abnormal neuronal migration and alterations in the number, survival, and orientation of neurons up to the time of olivary cell migration. . . before the end of the third month” of gestation. He notes that, in humans, the fetal thyroid forms about mid-gestation and that maternal dietary iodine deficiency and associated hypothyroidism prior to the third trimester of pregnancy can result in endemic cretinism. Multiple possible causes for both transient and long-term maternal hypothyroidism are proposed, including dietary iodine deficiency, exposure to food-based antithyroid substances (particularly soy products and other foods with high levels of isoflavonoids), and other environmental antithyroid substances (herbicides, PCBs, mercury, and coal derivatives). In follow-up to earlier studies, Román et al. (2013) evaluated 4039 mother-child pairs to determine if there was a correlation between maternal hypothyroxinemia before 18 weeks of gestation and autism risk. A “probable autistic child” was defined based on a score >98th percentile on the Pervasive Developmental Problems subscale of the Child Behavior Checklist in conjunction with a score in the top 5% on an 18-item short-form version of the Social Responsiveness Scale, administered at about age 6 years. Eighty children were defined as being a probable autistic child; the adjusted odds ratio of being a probable autistic

child increased 3.9 times when the mother had severe hypothyroxinemia in early gestation, but there was no relation between mild hypothyroxinemia in the mother and autism in the child. Specific ASD diagnosis was not performed, so a significant limitation in this study is the specificity of parent report of problems. Andersen et al. (2014) reported a retrospective population based cohort study of over 30,000 children born to mothers with thyroid disease that suggested an association between ASD (based on medical record review) and hypothyroidism diagnosed in the mother after birth, suggesting that antibody and TSH exposure prenatally could be a factor.

Colborn (2004) has also been outspoken in drawing an association between endocrine-disrupting synthetic chemicals and neurodevelopmental disorders such as ADHD and autism spectrum disorders. Some environmental chemicals, e.g., perchlorate, which has sometimes contaminated water supplies, directly interfere with iodine uptake into the thyroid gland. Other chemicals interfere with thyroid hormone signaling; polychlorinated biphenyls (PCBs) have this effect. Bisphenol A (BPA), involved in the manufacture of plastics, also blocks thyroid action on glial cells. Blazewicz et al. (2016) found lower urinary iodine levels in boys with ASDs, compared to controls, although thyroid hormone levels and thyroid volumes were comparable, with a correlation between clinical ASD severity and lower urinary iodine levels. Hamza et al. (2013) reported similar findings in both children with ASD and their mothers, also noting lower reported intake of iodized salt. Whether these differences were cause or effect of ASD was unclear.

As Zoeller (2005) notes, “a causal relation between (environmental) contaminants, thyroid hormone signaling, and cognitive development will be difficult to obtain given the fact that even small and transient thyroid hormone insufficiency may be detrimental.” If screening of pregnant women for elevated TSH levels and thyroid supplementation when indicated become routine, epidemiologic studies of autism in the children of these women will be informative. Meanwhile, there is not yet clear evidence to associate prenatal

exposure to maternal hypothyroidism with autism spectrum disorders, nor are there clear links from environmental toxins to maternal hypothyroidism to autism spectrum disorders, but the hypotheses presented are provocative and speak to the need for well-controlled study.

See Also

► Medical Evaluation in Autism

References and Reading

- Alvarez-Dolado, M., Ruiz, M., Del Rio, J. A., Alcantara, S., Burgaya, F., Sheldon, M., et al. (1999). Thyroid hormone regulates reelin and dab-1 expression during brain development. *Journal of Neuroscience*, 19, 6979–6993.
- Andersen SL, Laurbert P, Wu CS, Olsen J. (2014). Attention deficit hyperactivity disorder and autism spectrum disorder in children born to mothers with thyroid dysfunction: a Danish nationwide cohort study. *British Journal of Obstetrics and Gynecology (BJOG)* 121 (11), 1365–74.
- Bernal, J. (2007). Thyroid hormone receptors in brain development and function. *Nature Clinical Practice. Endocrinology & Metabolism*, 3, 249–259.
- Bilder, D., Pinborough-immerman, J., Miller, J., & McMahon, W. (2009). Prenatal, perinatal, and neonatal factors associated with autism spectrum disorders. *Pediatrics*, 123, 1293–1300.
- Blazewicz, A., Makarewicz, A., Korona-Glowniak, I., Dolliver, W., & Kocjan, R. (2016). Iodine in autism spectrum disorders. *Journal of Trace Elements in Medicine and Biology*, 34, 32–37.
- Brown, A.S., Surcel, H.M., Hinkka-Yli-Salomaki, S. et al. (2015). Maternal thyroid autoantibody and elevated risk of autism in a national birth cohort. Progress in Neuro-Psychopharmacology and Biological Psychiatry, 57, 86–92.
- Colborn, T. (2004). Neurodevelopment and endocrine disruption. *Environmental Health Perspectives*, 112, 944–949.
- Comi, A. M., Zimmerman, A. W., Frye, V. H., Law, P. A., & Peeden, J. N. (1999). Familial clustering of autoimmune disorders and evaluation of medical risk factors in autism. *Journal of Child Neurology*, 14, 388–394.
- Fatemi, S. H., Snow, A. V., Stry, J. M., Araghi-Niknam, M., Reutiman, T. J., Lee, S., et al. (2005). Reelin signaling is impaired in autism. *Biological Psychiatry*, 57, 777–787.
- Friauf, E., Wenz, M., Oberhofer, M., Nothwang, H. G., Balakrishnan, V., Knipper, M., et al. (2008). Hypothyroidism impairs chloride homeostasis and onset of inhibitory neurotransmission in developing auditory brainstem and hippocampal neurons. *European Journal of Neuroscience*, 28, 2371–2380.
- Gillberg, I. C., Gillberg, C., & Kopp, S. (1992). Hypothyroidism and autism spectrum disorders. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 33, 531–542.
- Haddow, J. E., Palomaki, G. E., Allan, W. C., Williams, J. R., Knight, G. J., Gagnon, J., et al. (1999). Maternal thyroid deficiency during pregnancy and subsequent neuropsychological development of the child. *The New England Journal of Medicine*, 341, 549–555.
- Hamza, R. T., Hewedi, D. H., & Sallam, M. T. (2013). Iodine deficiency in Egyptian autistic children and their mothers: Relation to disease severity. *Archives of Medical Research*, 44, 555–561.
- He, Y., Xun, G., Xia, K., Hu, Z., Lv, L., Deng, Z., & Zhao, J. (2011). No significant association between RELN polymorphism and autism in case-control and family-based association in Chinese Han population. *Psychiatry Research*, 187(3), 462–464.
- Kester, M. H., Martinez de Mena, R., Obregon, M. J., Marinkovic, D., Howatson, A., Visser, T. J., et al. (2004). Iodothyronine levels in the human developing brain: Major regulatory roles of iodothyronine deiodinases in different areas. *Journal of Clinical Endocrinology and Metabolism*, 89, 3117–3128.
- Knipper, M., Zinn, C., Maier, H., Praetorius, M., Rohbock, K., Kopschall, I., et al. (2000). Thyroid hormone deficiency before the onset of hearing causes irreversible damage to peripheral and central auditory systems. *Journal of Neurophysiology*, 83, 3101–3112.
- Kooistra, L., Kuppens, S. M. I., Hasaart, H. M., Vader, H. L., Wijnen, H. A., Oei, S. G., et al. (2010). High thyrotrophin levels at end term increase the risk of breech presentation. *Clinical Endocrinology*, 73, 661–665.
- Lammert, D. B., & Howell, B. W. (2016). RELN mutations in autism spectrum disorder. *Frontiers in Clinical Neuroscience*, 10, 1–9.
- Román, G. C. (2007). Autism: Transient in utero hypothyroxinemia related to maternal flavonoid ingestion during pregnancy and to other environmental antithyroid agents. *Journal of the Neurological Sciences*, 262, 15–26.
- Román, G. C., Ghassabian, A., Bongers-Schokking, J. J., Jaddoe, V. W. V., Hofman, A., et al. (2013). Association of gestational maternal hypothyroxinemia and increased autism risk. *Annals of Neurology*, 74, 733–742.
- Sadamatsu, M., Kanai, H., Xu, X., Liu, Y., & Kato, N. (2006). Review of animal models for autism: Implication of thyroid hormone. *Congenital Anomalies*, 46, 1–9.
- Serajee, F. J., Zhong, H., & Mahbulul Huq, A. H. M. (2006). Association of reelin gene polymorphisms with autism. *Genomics*, 87, 75–83.
- Sweeten, T. L., Bowyer, S. L., Posey, D. J., Halberstadt, G. M., & McDougall, C. J. (2003). Increased prevalence of familial autoimmunity in probands with pervasive developmental disorders. *Pediatrics*, 112, e420–e424.

- Taylor, M. A., Swant, J., Wagner, J. J., Fisher, J. W., & Ferguson, D. C. (2008). Lower thyroid compensatory reserve of rat pups after maternal hypothyroidism: Correlation of thyroid, hepatic, and cerebrocortical biomarkers with hippocampal neurophysiology. *Endocrinology*, 149, 3521–3530.
- Van der Meulen, J. A., Davies, G. A. L., & Kisilevsky, B. S. (2008). Fetal sensory-elicited body movements differ in breech compared to cephalic position. *Developmental Psychobiology*, 50, 530–534.
- Wu, S., Ding, Y., Wu, F., Li, R., Xie, G., Hou, J., & Mao, P. (2015). Family history of autoimmune diseases is associated with risk of autism in children: A systematic review and meta-analysis. *Neuroscience and Biobehavioral Reviews*, 55, 322–332.
- Zoeller, R. T. (2005). Thyroid hormone and brain development: Environmental influences. *Current Opinion in Endocrinology and Diabetes*, 12, 31–35.

Maternal Obesity and ASD Risk

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Definition

Obesity is a nutritional and metabolic life-course condition with excessive accumulation of adipose tissue that affects health adversely. Body mass index (BMI) calculated as weight in kilograms divided by height in meters squared was used for the definition of obesity. By this measure, obesity is defined as a BMI ≥ 30 kg/m² for adults. Furthermore, BMI values were classified in the following categories: underweight < 18.5 ; normal 18.5–24.9; overweight 25–29.9; obese class I 30–34.9, obese class II 35–39.9; obese class III ≥ 40 according to the National Institute of Health guidelines on obesity and overweight (NIH 1998). While there is no existing definition of maternal obesity during pregnancy, a maternal prepregnancy BMI is used for the classification.

Historical Background

In 2011, Dodds et al. (2011) published their study quantifying the relationship between prenatal, obstetric, and neonatal factors and autism risk. They first reported that prepregnancy weight of 90 kg or more was an independent risk factor for autism. Similarly, Lyall et al. (2011) reported their novel finding that maternal late adolescent BMI ≥ 30 may have a doubling of the risk of ASD in offspring. Since then, a growing number of studies assessing maternal obesity and risk of ASD in offspring have been published (Surén et al. 2014; Reynolds et al. 2014; Gardner et al. 2015; Getz et al. 2016; Connolly et al. 2016; Casas et al. 2017; Andersen et al. 2018) and a weak positive association has been evidenced by meta-analyses that quantitatively summarized the association (Li et al. 2016; Lei et al. 2019).

Previous studies have proposed several plausible mechanisms for explanation of the association observed. Dodds et al. (2011) speculated the role of leptin as leptin levels were high in obese mothers and children with autism were also found to have increased plasma leptin levels, while Lyall et al. (2011) proposed hormonally mediated pathway. But no further finding support these hypothesizes. Later, immunological and genetic factors were raised. Placenta inflammation can induce a systemic fetal inflammatory response and the role of cytokines in ASD has been found (van der Burg et al. 2015), this suggested that obesity activated maternal dysimmunity may increase the risk of ASD (Buehler 2011). Several abnormalities of the gene found in obese mothers were also linked to ASD, such as Apo D (Edlow et al. 2014), gene variants (Sebat et al. 2007), and changes in DNA methylation (Ladd-Acosta et al. 2014). Therefore, obese mothers are likely to pass on de novo mutations that confer risk for ASD (Pinto et al. 2010).

Current Knowledge

Current evidence suggests that maternal obesity slightly increases the risk of ASD in offspring. However, confounding effects should not be

ignored. For example, diabetes mellitus, including both gestational and preexisting diabetes, are often accompanied by obesity. Diabetes have also been evidenced to be a risk factor for ASD (Xu et al. 2014). Meanwhile, genetic susceptibility and other unknown risk factors also exist. Therefore, adjustment of potential confounding factors is important for implication of maternal obesity as an independent risk factor.

The association between maternal obesity and ASD risk in offspring may be simultaneously mediated by multiple factors and the explicit mechanism needs further elucidation. Often, maternal obesity is a cause of several other consequences that may play as mediators in the association. Besides, several maternal lifestyle factors, such as fat intake, that associated with ASD risk may also affect obesity (Lyall et al. 2013). Studies need to be methodologically rigorous and comprehensive so as to minimize the influences of other risk factors.

Future Directions

As immunological, genetic, and metabolic factors are involved in the pathogenesis of ASD, hypothesis-guided investigations are encouraged. Of proposed mechanisms, inflammatory factors are the most frequently mentioned and may be predominant. Also, potential mediators are required to be further identified for disentangling maternal obesity as a risk factor for ASD.

See Also

► Obstetrical Complications/Risk Factors

References and Reading

- Andersen, C. H., Thomsen, P. H., Nohr, E. A., & Lemcke, S. (2018). Maternal body mass index before pregnancy as a risk factor for ADHD and autism in children. *European Child & Adolescent Psychiatry*, 27(2), 139–148. <https://doi.org/10.1007/s00787-017-1027-6>.
- Buehler, M. (2011). A proposed mechanism for autism: An aberrant neuroimmune response manifested as a psychiatric disorder. *Medical Hypotheses*, 76(6), 863–870.
- Casas, M., Forns, J., Martinez, D., Guxens, M., Fernandez-Somoano, A., Ibarluzea, J., et al. (2017). Maternal pre-pregnancy obesity and neuropsychological development in pre-school children: A prospective cohort study. *Pediatric Research*, 82(4), 596–606. <https://doi.org/10.1038/pr.2017.118>.
- Connolly, N., Anixt, J., Manning, P., Ping, I. L. D., Marsolo, K. A., & Bowers, K. (2016). Maternal metabolic risk factors for autism spectrum disorder—An analysis of electronic medical records and linked birth data. *Autism Research*, 9(8), 829–837. <https://doi.org/10.1002/aur.1586>.
- Dodds, L., Fell, D. B., Shea, S., Armson, B. A., Allen, A. C., & Bryson, S. (2011). The role of prenatal, obstetric and neonatal factors in the development of autism. *Journal of Autism and Developmental Disorders*, 41(7), 891–902.
- Edlow, A. G., Vora, N. L., Hui, L., Wick, H. C., Cowan, J. M., & Bianchi, D. W. (2014). Maternal obesity affects fetal neurodevelopmental and metabolic gene expression: A pilot study. *PLoS One*, 9(2), e88661. <https://doi.org/10.1371/journal.pone.0088661>.
- Gardner, R. M., Lee, B. K., Magnusson, C., Rai, D., Frisell, T., Karlsson, H., et al. (2015). Maternal body mass index during early pregnancy, gestational weight gain, and risk of autism spectrum disorders: Results from a Swedish total population and discordant sibling study. *International Journal of Epidemiology*, 44(3), 870–883.
- Getz, K. D., Anderka, M. T., Werler, M. M., & Jick, S. S. (2016). Maternal pre-pregnancy body mass index and autism spectrum disorder among offspring: A population-based case-control study. *Paediatric and Perinatal Epidemiology*, 30(5), 479–487.
- Ladd-Acosta, C., Hansen, K. D., Briem, E., Fallin, M. D., Kaufmann, W. E., & Feinberg, A. P. (2014). Common DNA methylation alterations in multiple brain regions in autism. *Molecular Psychiatry*, 19(8), 862–871.
- Lei, X. Y., Li, Y. J., Ou, J. J., & Li, Y. M. (2019). Association between parental body mass index and autism spectrum disorder: A systematic review and meta-analysis. *European Child & Adolescent Psychiatry*, 28(7), 933–947. <https://doi.org/10.1007/s00787-018-1259-0>.
- Li, Y.-M., Ou, J.-J., Liu, L., Zhang, D., Zhao, J.-P., & Tang, S.-Y. (2016). Association between maternal obesity and autism spectrum disorder in offspring: A meta-analysis. *Journal of Autism and Developmental Disorders*, 46(1), 95–102.
- Lyall, K., Pauls, D. L., Santangelo, S., Spiegelman, D., & Ascherio, A. (2011). Maternal early life factors associated with hormone levels and the risk of having a child with an autism spectrum disorder in the nurses health study II. *Journal of Autism and Developmental Disorders*, 41(5), 618–627.
- Lyall, K., Munger, K. L., O'reilly, É. J., Santangelo, S. L., & Ascherio, A. (2013). Maternal dietary fat intake in

- association with autism spectrum disorders. *American Journal of Epidemiology*, 178(2), 209–220.
- National Institute of Health. (1998). Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: The evidence report. *Obesity Research*, 6(2), 51S–209S.
- Pinto, D., Pagnamenta, A. T., Klei, L., Anney, R., Merico, D., Regan, R., et al. (2010). Functional impact of global rare copy number variation in autism spectrum disorders. *Nature*, 466(7304), 368–372.
- Reynolds, L. C., Inder, T. E., Neil, J. J., Pineda, R. G., & Rogers, C. E. (2014). Maternal obesity and increased risk for autism and developmental delay among very preterm infants. *Journal of Perinatology*, 34(9), 688–692.
- Sebat, J., Lakshmi, B., Malhotra, D., Troge, J., Lese-Martin, C., Walsh, T., et al. (2007). Strong association of de novo copy number mutations with autism. *Science*, 316(5823), 445–449.
- Surén, P., Gunnes, N., Roth, C., Bresnahan, M., Hornig, M., Hirtz, D., et al. (2014). Parental obesity and risk of autism spectrum disorder. *Pediatrics*, 133, 2013–3664.
- van der Burg, J. W., Sen, S., Chomitz, V. R., Seidell, J. C., Leviton, A., & Dammann, O. (2015). The role of systemic inflammation linking maternal BMI to neurodevelopment in children. *Pediatric Research*, 79 (1–1), 3–12.
- Xu, G., Jing, J., Bowers, K., Liu, B., & Bao, W. (2014). Maternal diabetes and the risk of autism spectrum disorders in the offspring: A systematic review and meta-analysis. *Journal of Autism and Developmental Disorders*, 44(4), 766–775.

Maternal Opioid Prescription in Autism Spectrum Disorder

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Definition

Since 2000, both prevalence of identified ASD and the proportion of pregnant women receiving an opioid prescription during pregnancy have increased dramatically. Current evidence suggests a possible association between maternal opioid prescription and ASD identification; yet, few studies of outcomes for children exposed to opiates in utero have had the statistical power or

longitudinal duration to assess autism as an outcome. As opioid use continues to be a research priority in the USA, better data will be available to understand the relationship between prescription and/or illicit opioid use and risk of autism.

Historical Background

The prevalence of ASD identified in population-based samples in the USA has increased consistently in the previous two decades (Autism and Developmental Disabilities Monitoring Network Principal Investigators 2007; Baio et al. 2018; Nevison et al. 2018). While some of this increase is attributable to changing diagnostic patterns (Coo et al. 2007; King and Bearman 2009) or increasing accumulation of ASD-like traits across generations (Connolly et al. 2019; Rubenstein and Chawla 2018; Virkud et al. 2009), there is still a component of ASD risk attributable to negative environmental exposures perinatally (Lyll et al. 2017). Here, the “environment” refers to factors that are not genetic (although genetic factors may also be affected by the environment) and include a person’s physical environment, social and cultural environment, and lifestyle choices (Rothman et al. 2008). Identifying which exposures are linked to ASD adds to the understanding of biological mechanisms that cause ASD and enable development of targeted therapeutics to prevent some impairing aspects, associated features, and co-occurring conditions of ASD.

One such perinatal exposure that has increased in prevalence over the past two decades in the USA is the prescription and use of opioid medication. In a study using Medicaid claims from 2000 to 2007, researchers found that 22% of pregnant women received an opioid prescription at any point during pregnancy (Desai et al. 2014). In a study of women with commercial health plans between 2005 and 2011, researchers found that 14% of women received an opioid prescription (Bateman et al. 2014).

Opioids are a class of drug that binds to opioid receptors in the brain and body causing a reduced perception of pain. While often prescribed to manage short-term pain, opioids can be highly

addictive and are often overprescribed (Lind et al. 2017). Opioid use during pregnancy (whether prescribed or illicit) has been seen to be associated with preterm birth, specific birth defects, poor fetal growth, neonatal abstinence syndrome, and poor child development at 6 months (Broussard et al. 2011; Lind et al. 2017; Norgaard et al. 2015; Nygaard et al. 2015; Sundelin Wahlsten and Sarman 2013; Yazdy et al. 2015). Much research and public health attention has been paid to the “opioid epidemic” (Gostin et al. 2017); yet, there had been limited opportunity to study longitudinal childhood outcomes for children exposed to opioids in utero (Reddy et al. 2017). Preliminary studies have assessed ASD as an outcome identified later in childhood in specific samples, but in building large, longitudinal cohorts, evidence can be further clarified, especially among illicit opioid users.

Potential Mechanisms

Exposure to opioids in utero may alter fetal development through a few hypothesized mechanisms. One such mechanism is based on opioids being small lipophilic molecules that can cross the placental and blood-brain barrier. Therefore, a fetus is at risk if the mother is exposed (Hudak et al. 2012). When exposed to an opioid, the fetal brain structure and function, specifically synaptic plasticity and neural connectivity, may be altered (Ross et al. 2015). Alteration in synaptic plasticity has been shown to be associated with development of autistic traits (Bourgeron 2015; Gilbert and Man 2017). Exogenous opioid exposure during pregnancy could alter functioning of opioid receptors which are linked to neurodevelopment (Byrnes and Vassoler 2018; Gonzalez-Nunez et al. 2013; Sahley and Panksepp 1987).

Exposure to opioids may also lead to increased risk for ASD indirectly through increased risk of other pregnancy complications. Women who use opioids during pregnancy are at increased risk of having children born preterm, low birth weight, and being small for gestational age, even after adjusting for confounding factors like socioeconomic status and other health conditions (Brogly et al. 2017; Norgaard et al. 2015; Whiteman et al. 2014). Poor pregnancy outcomes, like preterm birth and size for gestational age, are also

independently associated with ASD risk (Lyll et al. 2017). It is possible that any association between opioid exposure and ASD is mediated through one of these sub-optimal pregnancy outcomes.

Additionally, an association may be seen because of confounding. One type of confounding that may result in seeing an association between opioid use and child ASD is confounding by indication. A mother may be prescribed an opioid for a condition that itself is a risk factor for ASD and the association is through the condition rather than the medication (Signorello et al. 2002). For example, a pregnant woman may experience an injury that causes both pain and inflammation, and an opioid may be prescribed; however, it is the inflammation that is the risk factor for ASD rather than the opioid prescription (Meldrum et al. 2013). Outside of confounding by indication, many conditions linked to ASD are also linked to opioid use, including anxiety and depression (Rai et al. 2013; Whiteman et al. 2014), and it could be the common condition/exposure that leads to the association rather than the opioid exposure.

Current Knowledge

Across the literature, there have been few studies to assess developmental outcomes that can only be assessed after the postnatal period (Reddy et al. 2017). While longitudinal birth cohorts of opioid exposed children are being built, case-control studies can offer insight into the association between prescription opioid use during pregnancy and child ASD. Two studies were identified that assessed ASD as an outcome or maternal opioid prescription during pregnancy.

Prescription Opioid During the Pregnancy Period in the Study to Explore Early Development

In a 2019 study, Rubenstein et al. (2019) used data from the Study to Explore Early Development, a multi-site case-control study of development in preschool age children (Schendel et al. 2012). The sample was comprised of children born between 2007 and 2012 from six US states (California, Colorado, Georgia, Maryland, North

Carolina, and Pennsylvania) who were enrolled after being identified from ASD or other developmental disability service providers or from a random sampling of birth certificates. All children were screened for ASD, and those that screened positive or had past indication of ASD received a full gold-standard evaluation. Mothers answered a series of questionnaires about their environmental exposures during pregnancy, as well as providing their medical records and the child's birth record. Prescription medication, the date prescribed, and the duration of the prescription were listed on the medical records, and two independent reviewers identified the medication containing opioids.

The researchers examined prescription opioid use from 3 months pre-pregnancy to a day before childbirth, ensuring that medication was not related to childbirth or post-birth recovery. Researchers compared the odds of having an opioid prescription (ever in pregnancy, pre-pregnancy, trimester 1, trimester 2, and trimester 3 as determined by childbirth date and gestational age at childbirth) in the ASD group ($N = 1369$) to the odds of having an opioid prescription in the population comparison group ($N = 1577$). The researchers additionally evaluated whether odds of opioid prescriptions differed between a group comprised on children with developmental delay (DD) with autism symptoms ($N = 476$) and the ASD group (children with DD with ASD symptoms and children with ASD, since DD with ASD features were more phenotypically similar to the ASD group than the DD group (Wiggins et al. 2015) and to account for a sub-diagnostic autism presentation) compared to the population comparison group. For all analyses, the researchers ran an unadjusted logistic regression and an adjusted model to account for confounding due to maternal education, race/ethnicity, smoking during pregnancy, psychiatric condition during pregnancy, and year of childbirth.

In the sample 7.7% of mothers had an opioid medication prescribed during the pregnancy period. During the preconception period, mothers that were prescribed an opioid had 2.43 times the odds of having a child with ASD compared to mothers without a prescription opioid in the adjusted model (95% confidence interval, 0.99,

6.02), and there was a similar odds ratio for the DD with ASD features + ASD group (adjusted odds ratio: 2.64, 95% CI: 1.10, 6.31). Odds were also elevated for prescriptions in the third trimester (ASD adjusted odds ratio: 1.26, 95% CI: 0.94, 1.68; DD with ASD features + ASD adjusted odds ratio: 1.30, 95% CI: 0.99, 1.71), but to a lesser extent than during the preconception period. There were no associations between DD without ASD features and opioid prescriptions.

Association Between Prescription Opioid Use and Child ASD in an Israeli Health System

In 2018 Janecka and colleagues published a case-control study from the Israeli health system cohort of children born between 1997 and 2007 and followed until 2015. All children with an ASD diagnosis were sampled along with a 19.5% random sample of the original cohort (total $N = 96,270$, N with ASD = 1405). All medication types were identified through the mother's health record from 280 days before the childbirth and categorized based on drug type. The researchers ran a series of Cox proportional hazard models that varied in which covariates were included, but their confounding set consisted of maternal age, paternal age, maternal affective, anxiety, psychotic, neurologic disorders, and a number of maternal diagnoses. The researchers assessed 55 medication groups, two of which (κ and ϵ receptor agonists) were opioids.

In the sample, 1.94% of children were exposed to opioid μ receptor agonists, 1.91% to κ receptor agonists, and 1.91% to ϵ receptor agonists. In all regression models, the odds ratios were less than 1.0 when comparing odds of ASD in mothers with prescriptions to mothers without prescription; although, only the fully adjusted model assessing κ and ϵ receptor agonists met statistical significance at a 0.05 level (aOR: 0.67, 95% CI: 0.45, 0.99).

Limitations

The two described studies assess whether maternal opioid medication is associated with child ASD. Both studies had sampling methodology based on identifying ASD leading to relatively low opioid exposure. Studies derived from

cohorts of opioid users may increase power and provide further mechanistic detail into child outcomes. Additionally, both studies only assessed prescription opioids. There was no way to validate whether the medication was consumed or whether there was illicit opioid use. Further, both studies were conditioned on live birth, meaning that if opioid exposure was associated with fetal loss or still birth, we would not see that effect in these data. The Janecka study was conducted in Israel, which is not facing the same opioid epidemic as in the USA, so prescribing practice may not be directly comparable.

Future Directions

The opioid epidemic is of key concern to American policy makers and scientific agency. Of high priority are longitudinal cohort studies assessing outcomes for children exposed to opioids in utero. For example, the National Institutes of Health put out a request for applications for the “HEAL Initiative” to create a longitudinal study consortium to assess outcomes of children exposed in utero (National Institutes of Health 2018). ASD or autistic traits can be one such outcome measurement.

Additionally, large national health system data from Nordic countries should be leveraged to examine the association between prescription opioid use and child ASD, with special focus on parsing the effect of indication. Those data would be ideal because of the number of pregnant women that can be assessed and the ability to link the medical data to other data sources to better identify and control for confounding. Further, these data could be used to examine pregnancy outcomes like preterm birth and low birth weight as mediators between opioid prescription and child ASD.

While the relationship between opioid prescription in pregnancy and child ASD is not fully understood, it is important that clinicians avoid prescribing these drugs when it is not necessary and when prescribed for postoperative pain management, clinicians should be judicious in their prescribing and provide close follow-up, limited quantities, and rapid tapering (Reddy et al. 2017).

See Also

► Epidemiology

References and Reading

- Autism and Developmental Disabilities Monitoring Network Principal Investigators. (2007). Prevalence of autism spectrum disorders—autism and developmental disabilities monitoring network, six sites, United States, 2000. *MMWR Surveillance Summaries*, 56, 1), 1–11. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/17287714>.
- Baio, J., Wiggins, L. D., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., . . . Dowling, N. F. (2018). Prevalence of autism Spectrum disorder among children aged 8 years — Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *Morbidity and Mortality Weekly Report. Surveillance Summaries*, 67(6), 1–23. <https://doi.org/10.15585/mmwr.ss6706a1>.
- Bateman, B. T., Hernandez-Diaz, S., Rathmell, J. P., Seeger, J. D., Doherty, M., Fischer, M. A., & Huybrechts, K. F. (2014). Patterns of opioid utilization in pregnancy in a large cohort of commercial insurance beneficiaries in the United States. *Anesthesiology*, 120(5), 1216–1224. <https://doi.org/10.1097/ALN.0000000000000172>.
- Bourgeron, T. (2015). From the genetic architecture to synaptic plasticity in autism spectrum disorder. *Nature Reviews Neuroscience*, 16(9), 551–563. <https://doi.org/10.1038/nrn3992>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/26289574>.
- Brogly, S. B., Turner, S., Lajkosz, K., Davies, G., Newman, A., Johnson, A., & Dow, K. (2017). Infants born to opioid-dependent women in Ontario, 2002–2014. *Journal of Obstetrics and Gynaecology Canada*, 39(3), 157–165. <https://doi.org/10.1016/j.jogc.2016.11.009>. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/28343557>.
- Broussard, C. S., Rasmussen, S. A., Reefhuis, J., Friedman, J. M., Jann, M. W., Riehle-Colarusso, T., . . . National Birth Defects Prevention Study (2011). Maternal treatment with opioid analgesics and risk for birth defects. *American Journal of Obstetrics and Gynecology*, 204(4), 314 e311–311. <https://doi.org/10.1016/j.ajog.2010.12.039>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/21345403>.
- Byrnes, E. M., & Vassoler, F. M. (2018). Modeling prenatal opioid exposure in animals: Current findings and future directions. *Frontiers in Neuroendocrinology*, 51, 1–13. <https://doi.org/10.1016/j.yfme.2017.09.001>. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/28965857>.
- Connolly, S., Anney, R., Gallagher, L., & Heron, E. A. (2019). Evidence of assortative mating in autism Spectrum disorder. *Biological Psychiatry*. <https://doi.org/10.1016/j.biopsych.2019.04.014>. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/31200929>.

- Coo, H., Ouellette-Kuntz, H., Lloyd, J. E. V., Kasmara, L., Holden, J. J. A., & Lewis, M. E. S. (2007). Trends in autism prevalence: Diagnostic substitution revisited. *Journal of Autism and Developmental Disorders*, 38(6), 1036–1046. <https://doi.org/10.1007/s10803-007-0478-x>. Retrieved from <http://search.ebscohost.com/login.aspx?direct=true&db=psyh&AN=2008-07706-005&site=ehost-live&scope=siteoullette@post.queensu.ca>; <http://link.springer.com/article/10.1007/s10803-007-0478-x>.
- Desai, R. J., Hernandez-Diaz, S., Bateman, B. T., & Huybrechts, K. F. (2014). Increase in prescription opioid use during pregnancy among Medicaid-enrolled women. *Obstetrics and Gynecology*, 123(5), 997–1002. <https://doi.org/10.1097/AOG.0000000000000208>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/24785852>.
- Gilbert, J., & Man, H. Y. (2017). Fundamental elements in autism: From neurogenesis and neurite growth to synaptic plasticity. *Frontiers in Cellular Neuroscience*, 11, 359. <https://doi.org/10.3389/fncel.2017.00359>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/29209173>.
- Gonzalez-Nunez, V., Jimenez Gonzalez, A., Barreto-Valer, K., & Rodriguez, R. E. (2013). In vivo regulation of the mu opioid receptor: Role of the endogenous opioid agents. *Molecular Medicine*, 19, 7–17. <https://doi.org/10.2119/molmed.2012.00318>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/23348513>.
- Gostin, L. O., Hodge, J. G., Jr., & Noe, S. A. (2017). Reframing the opioid epidemic as a National Emergency. *JAMA*, 318(16), 1539–1540. <https://doi.org/10.1001/jama.2017.13358>. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/28832871>.
- Hudak, M. L., Tan, R. C., & Committee on Drugs, Committee on Fetus and Newborn, American Academy of Pediatrics. (2012). Neonatal drug withdrawal. *Pediatrics*, 129(2), e540–e560. <https://doi.org/10.1542/peds.2011-3212>. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/22291123>.
- King, M., & Bearman, P. (2009). Diagnostic change and the increased prevalence of autism. *International Journal of Epidemiology*, 38(5), 1224–1234. <https://doi.org/10.1093/ije/dyp261>. Retrieved from <http://ije.oxfordjournals.org/content/38/5/1224.abstract>; <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2800781/pdf/dyp261.pdf>.
- Lind, J. N., Interrante, J. D., Ailes, E. C., Gilboa, S. M., Khan, S., Frey, M. T., . . . Broussard, C. S. (2017). Maternal use of opioids during pregnancy and congenital malformations: A systematic review. *Pediatrics*, 139(6). Retrieved from <http://pediatrics.aappublications.org/content/139/6/e20164131.abstract>
- Lyall, K., Croen, L., Daniels, J., Fallin, M. D., Ladd-Acosta, C., Lee, B. K., . . . Newschaffer, C. (2017). The changing epidemiology of autism Spectrum disorders. *Annual Review of Public Health*, 38, 81–102. <https://doi.org/10.1146/annurev-publhealth-031816-044318>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/28068486>
- Meldrum, S. J., Strunk, T., Currie, A., Prescott, S. L., Simmer, K., & Whitehouse, A. J. (2013). Autism spectrum disorder in children born preterm-role of exposure to perinatal inflammation. *Frontiers in Neuroscience*, 7, 123. <https://doi.org/10.3389/fnins.2013.00123>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/23885233>.
- National Institutes of Health. (2018). RFA-HD-19-025: HEAL initiative: Antenatal opioid exposure longitudinal study consortium (PL1 clinical trial not allowed). Retrieved from <https://grants.nih.gov/grants/guide/rfa-files/rfa-hd-19-025.html>
- Nevison, C., Blaxill, M., & Zahorodny, W. (2018). California autism prevalence trends from 1931 to 2014 and comparison to national ASD Data from IDEA and ADDM. *Journal of Autism and Developmental Disorders*, 48(12), 4103–4117. <https://doi.org/10.1007/s10803-018-3670-2>. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/29974300>.
- Norgaard, M., Nielsson, M. S., & Heide-Jorgensen, U. (2015). Birth and neonatal outcomes following opioid use in pregnancy: A Danish population-based study. *Substance Abuse*, 9(Suppl 2), 5–11. <https://doi.org/10.4137/SART.S23547>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/26512202>.
- Nygaard, E., Moe, V., Slinning, K., & Walhovd, K. B. (2015). Longitudinal cognitive development of children born to mothers with opioid and polysubstance use. *Pediatric Research*, 78(3), 330–335. <https://doi.org/10.1038/pr.2015.95>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/25978800>.
- Rai, D., Lee, B. K., Dalman, C., Golding, J., Lewis, G., & Magnusson, C. (2013). Parental depression, maternal antidepressant use during pregnancy, and risk of autism spectrum disorders: Population based case-control study. *BMJ*, 346, f2059. <https://doi.org/10.1136/bmj.f2059>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/23604083>; <http://www.bmj.com/content/bmj/346/bmj.f2059.full.pdf>.
- Reddy, U. M., Davis, J. M., Ren, Z., Greene, M. F., & Neonatal Abstinence Syndrome and Childhood Outcomes Workshop Invited Speakers Opioid Use in Pregnancy. (2017). Opioid use in pregnancy, neonatal abstinence syndrome, and childhood outcomes: Executive summary of a joint workshop by the Eunice Kennedy Shriver National Institute of Child Health and Human Development, American College of Obstetricians and Gynecologists, American Academy of Pediatrics, Society for Maternal-Fetal Medicine, Centers for Disease Control and Prevention, and the March of Dimes Foundation. *Obstetrics and Gynecology*, 130(1), 10–28. <https://doi.org/10.1097/AOG.0000000000002054>. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/28594753>.
- Ross, E. J., Graham, D. L., Money, K. M., & Stanwood, G. D. (2015). Developmental consequences of fetal exposure to drugs: What we know and what we still must learn. *Neuropsychopharmacology*, 40(1), 61–87. <https://doi.org/10.1038/npp.2014.147>.
- Rothman, K. J., Greenland, S., & Lash, T. L. (2008). *Modern Epidemiology* (3rd ed.). Philadelphia, PA: Lippincott, Williams & Wilkins.
- Rubenstein, E., & Chawla, D. (2018). Broader autism phenotype in parents of children with autism:

- A systematic review of percentage estimates. *Journal of Child and Family Studies*, 27(6), 1705–1720. <https://doi.org/10.1007/s10826-018-1026-3>. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/29731598>.
- Rubenstein, E., Young, J. C., Croen, L. A., DiGuseppi, C., Dowling, N. F., Lee, L. C., . . . Daniels, J. (2019). Brief report: Maternal opioid prescription from preconception through pregnancy and the odds of autism Spectrum disorder and autism features in children. *Journal of Autism and Developmental Disorders*, 49(1), 376–382. <https://doi.org/10.1007/s10803-018-3721-8>. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/30132098>
- Sahley, T. L., & Panksepp, J. (1987). Brain opioids and autism: An updated analysis of possible linkages. *Journal of Autism and Developmental Disorders*, 17(2), 201–216. <https://doi.org/10.1007/BF01495056>.
- Schendel, D. E., DiGuseppi, C., Croen, L. A., Fallin, M. D., Reed, P. L., Schieve, L. A., . . . Yeargin-Allsopp, M. (2012). The Study to Explore Early Development (SEED): A multisite epidemiologic study of autism by the centers for autism and developmental disabilities research and epidemiology (CADDRE) network. *Journal of Autism and Developmental Disorders*, 42(10), 2121–2140. <https://doi.org/10.1007/s10803-012-1461-8>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/22350336>
- Signorello, L. B., McLaughlin, J. K., Lipworth, L., Friis, S., Sørensen, H. T., & Blot, W. J. (2002). Confounding by indication in epidemiologic studies of commonly used analgesics. *American Journal of Therapeutics*, 9(3), 199–205.
- Sundelin Wahlsten, V., & Sarman, I. (2013). Neurobehavioural development of preschool-age children born to addicted mothers given opiate maintenance treatment with buprenorphine during pregnancy. *Acta Paediatrica*, 102(5), 544–549. <https://doi.org/10.1111/apa.12210>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/23432078>.
- Virkud, Y. V., Todd, R. D., Abbacchi, A. M., Zhang, Y., & Constantino, J. N. (2009). Familial aggregation of quantitative autistic traits in multiplex versus simplex autism. *American Journal of Medical Genetics. Part B: Neuropsychiatric Genetics*, 150B(3), 328–334. <https://doi.org/10.1002/ajmg.b.30810>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/18618672>.
- Whiteman, V. E., Salemi, J. L., Mogos, M. F., Cain, M. A., Aliyu, M. H., & Salihu, H. M. (2014). Maternal opioid drug use during pregnancy and its impact on perinatal morbidity, mortality, and the costs of medical care in the United States. *Journal of Pregnancy*, 2014, 906723. <https://doi.org/10.1155/2014/906723>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/25254116>.
- Wiggins, L. D., Reynolds, A., Rice, C. E., Moody, E. J., Bernal, P., Blaskey, L., . . . Levy, S. E. (2015). Using standardized diagnostic instruments to classify children with autism in the study to explore early development. *Journal of Autism and Developmental Disorders*, 45(5), 1271–1280. <https://doi.org/10.1007/s10803-014-2287-3>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/25348175>.
- Yazdy, M. M., Desai, R. J., & Brogly, S. B. (2015). Prescription opioids in pregnancy and birth outcomes: A review of the literature. *J Pediatr Genet*, 4(2), 56–70. <https://doi.org/10.1055/s-0035-1556740>. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/26998394>.

Mathematics Disability

► Dyscalculia

May Institute, Second Edition

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Definition

May Institute is a nonprofit organization. The mission of May Institute is to be the global leader in providing innovative applied behavior analytic services to individuals with autism spectrum disorder and neurobehavioral disorders across the life span. May Institute is staffed by over 2000 employees including board certified behavior analysts, special educators, and direct care professionals. Many of our staff have advanced degrees in their field of study.

Historical Background

In 1955, Dr. Jacques May and his wife, Marie Anne May, opened a small school for children with autism in Chatham, Massachusetts. They had twin boys diagnosed with autism spectrum disorder (ASD) and dedicated the school to helping their boys, and others with ASD, develop skills needed to live as independently as possible.

Today, May Institute serves individuals with ASD, brain injury, and other disabilities across the life span. We have more than 140 locations across the country including five schools, consultation

services, home-based services, vocational programs, and community-based residences for children and adults.

May Institute is invested in advancing the field of behavior analysis through high-quality research and services for people with ASD. May Institute also has played a key role in shaping policy to positively affect the lives of people with disabilities. In addition, we are passionate about training the next generation of professionals. In 2005 May Institute established the National Autism Center (NAC), which is charged with dissemination of research and training in evidence-informed practices to support individuals with ASD and other disabilities. May Institute and NAC are active centers of research and training and maintain affiliations with over 50 universities, hospitals, and human service agencies across the world. Our staff frequently publish in peer-reviewed journals and present research findings at national and international conferences. Staff have federal and private grants and contracts to support cutting edge research. In the area of training, May Institute provides training opportunities via internships and practicum placements to undergraduate and graduate students. There is a formal internship for doctoral-level psychology students that is approved by the Association of Psychology Postdoctoral and Internship Centers (APPIC).

Rationale or Underlying Theory

All clinical services and research are based on the science of behavior analysis. Applied behavior analysis (ABA) is the application of those principles to socially significant problems. There is a significant body of research documenting that interventions based on applied behavior analysis are the most effective approach for addressing the myriad difficulties that may be experienced by people with ASD and neurobehavioral disorders.

Goals and Objectives

It is the mission of May Institute to be the global leader in the provision of innovative applied

behavior analytic services to individuals with ASD and other neurobehavioral disorders across the life span. Thus, all of the services provided at May Institute use the science of behavior analysis to enhance the lives of the people served. People with ASD have deficits in communication and social interaction. For example, some individuals may struggle to communicate basic wants and needs, whereas others communicate well but have difficulty understanding subtle nonverbal communication. Other individuals might exhibit distressing repetitive behaviors and rituals or engage in stereotypic behavior that interferes with their ability to interact with others. Professionals at May Institute use evidence-based assessments and input from stakeholders to develop goals for each person that is served. Individualized, behavior analytic programs that use systematic methods of teaching are used to help people acquire meaningful and valued skills. Teaching at May Institute is positive and proactive and uses state-of-the-art technology.

Treatment Participants

May Institute provides services to children and adults with an ASD and other neurobehavioral disorders. ASD is a spectrum disorder which means that it can present in many different ways. All individuals with autism experience deficits in communication and social interaction and have struggles with restrictive or repetitive patterns of behavior, interests, or activities. However, the way in which these deficits presents can vary greatly. May Institute supports people with ASD and neurobehavioral disorders across the life span and at all levels of need. Academic, social behavioral, and vocational services are provided to individuals across the spectrum.

Treatment Procedures

The treatment procedures used by professionals at May Institute for individuals with an ASD are applied behavior analytic. The goal of all

interventions is to increase the quality of life and independence of the individuals we serve.

In our consultation services to support young children with a diagnosis of ASD, we work in both center-based programs and family's homes. We provide behavior analytic interventions designed to increase skills in social communication and interaction, as well as teaching children skills they will need to succeed in school. All interventions are individualized for a specific child and address that child's specific need. For example, some children have no means of functional communication, and intervention thus focuses on helping those children learn to communicate their wants and needs. For some children, this involves teaching spoken words; for others, we teach augmentative methods of communication, such as using picture icons on a tablet. Many children with ASD can communicate wants and needs but struggle with other aspects of communication or interaction. For example, a child may have difficulty engaging in reciprocal interaction or sharing interest in something with another person (joint attention). In such cases, intervention is directed toward enhancing those skills.

Our school consultation programs are designed to help teachers and other educational professionals develop and enhance skills to support students with ASD. Activities include workshops and training, classroom-based consultation, and direct work with students and teachers. In addition, May Institute operates "model classrooms" through which we help districts train educators to implement state-of-the-art behavior analytic interventions to support students.

May Institute operates five private schools, four support students with ASD and one supports students with brain injury. Across schools, our students each have an Individualized Education Plan (IEP) specifying goals in education (e.g., reading), acquisition of communication and interaction (e.g., increasing peer interactions), and addressing any behavioral challenges (e.g., replacing self-injury with more desirable behavior). All programming is based on principles of behavior analysis, including the way academic instruction is provided, how skills are taught,

how challenging behavior is addressed, and how vocational skills are taught.

For academic instruction, teachers design individualized education plans based on comprehensive assessments of students needs and provide academic instruction using evidence-based methods of instruction in both group and individualized instruction. In addition, May staff utilize behavior analytic assessments and intervention strategies to help each student learn to communicate wants and needs, to initiate and carry on conversations, and to interact with peers and adults in a developmentally appropriate manner. A goal of interventions in this area is to teach in such a way as facilitate independence and generalization to the community. Behavioral challenges are addressed in a comprehensive manner that always begins with a functional behavior assessment (FBA) to identify what evokes the behavior (e.g., requests) and what seems to maintain or reinforce the behavior, such as avoiding instruction, gaining attention from others, or getting access to some preferred activity or item. Results of the FBA are used to develop an intervention that consists of strategies to (a) reduce the likelihood the behavior will occur in the first place, (b) teach new skills, (c) increase the occurrence of desired behavior, and (d) decrease the likelihood of challenging behavior. Vocational and prevocational skills are also taught across the schools utilizing the principles of behavior analysis. Two of our five schools offer community-based residential services. Residential staff members utilize behavior analytic interventions to teach skills ranging from self-care (e.g., tooth brushing) to daily living skills (e.g., meal preparation) with the goal of increase the students' independence and increase their quality of life. Clinicians and educators place an emphasis on involving family members into the educational and treatment process and systematically plan for generalization of acquired skills across various environments.

Adult services provided at May Institute include day programs, shared-living, and community-based residential programs. The primary goal of adult services is to provide individualized supports that are evidence-based. We

serve adults living with ASD, intellectual disabilities, and traumatic brain injury at all stages of their adult lives. We provide a wide range of services that include day programs, shared-living, community-based residential programs, and independent living programs. The services provided to adults focus on teaching skills that increase independence in all areas of their lives. Most of the adults we serve have an Individualized Support Plan (ISP) that is developed by a team of people who are familiar with the individuals' desires and support needs. The ISP specifies the person's vision for their life and the goals they want to work on to achieve their vision. It also identifies the kinds of support the individual will need to meet their goals and live in the least restrictive environment possible. Trained support staff help the adults in our care participate meaningfully in their communities, develop social skills needed to form and maintain relationships, and learn skills that increase their ability to participate fully in daily routines throughout their lives.

Efficacy Information

Interventions based on the science of behavior analysis have repeatedly been demonstrated to be highly effective in addressing the challenges experienced by people with ASD (e.g., Heyvaert et al. 2014; National Autism Center 2015; Odom et al. 2010; Smith and Iadarola 2015; Wong et al. 2015). For example, both NAC and Wong et al. reviewed peer-reviewed intervention studies for individuals with ASD to identify practices with a strong evidence base. Wong et al. restricted the sample to ages birth to 22, whereas NAC examined studies across the life span. Both identified broad categories of practice (e.g., prompting, reinforcement), and there was significant overlap between the two reviews. All evidence-informed practices were, in the words of Wong et al., "...fundamental applied behavior analysis techniques..." (p. 1957). At May Institute, all interventions used fall within the evidence-informed categories identified in the above-referenced reviews.

Outcome Measurement

At May Institute, our staff collect data on all identified goals and target behaviors of the individuals we serve. For example, if a treatment goal is increasing functional communication, staff might record the frequency of targeted communicative responses (e.g., signing, "apple"). If a goal is increasing reading fluency, staff might measure words read correctly per minute to monitor acquisition of reading. When a goal is addressing challenging behavior, staff record the frequency of challenging behavior and also the occurrence of more desired behavior. In addition to recording outcome measures, staff also collect data on treatment integrity, to monitor the fidelity with which programs and interventions are implemented as designed. If outcome data suggest an intervention is not producing desired goals and treatment integrity is good, then the intervention would be adjusted to produce desired outcomes. Alternatively, if treatment integrity was low, additional training would be provided to help staff implement the intervention as designed.

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Qualifications of Treatment Providers

All treatment providers at May Institute are appropriately qualified and credentialed for their given profession and area of expertise. For example, staff include licensed psychologists and licensed behavior analysts as well as certified teachers, licensed or certified speech and language therapists, occupational therapists, and social workers.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Autism Spectrum Disorder](#)
- [Behavior Analysis](#)

References and Reading

Heyvaert, M., Saenen, L., Campbell, J. M., Maes, B., & Onghena, P. (2014). Efficacy of behavioral

interventions for reducing problem behavior in persons with autism: An updated quantitative synthesis of single-subject research. *Research in Developmental Disabilities*, 35(10), 2463–2476. <https://doi.org/10.1016/j.ridd.2014.06.017>.

National Autism Center. (2015). National standards project findings and conclusions, Phase 2. Retrieved from Randolph: Author.

Odom, S. L., Boyd, B. A., Hall, L. J., & Hume, K. (2010). Evaluation of comprehensive treatment models for individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40, 425–436. <https://doi.org/10.1007/s10803-009-0825-1>.

Smith, T., & Iadarola, S. (2015). Evidence base update for autism spectrum disorder. *Journal of Clinical Child and Adolescent Psychology*, 44(6), 897–922. <https://doi.org/10.1080/15374416.2015.1077448>.

Wong, C., Odom, S. L., Hume, K. A., Cox, A. W., Fetting, A., Kucharczyk, S., et al. (2015). Evidence-based practices for children, youth, and young adults with autism spectrum disorder: A comprehensive review. *Journal of Autism and Developmental Disorders*, 45, 1951–1966. <https://doi.org/10.1007/s10803-014-2351-z>.

M-CHAT

► [Modified Checklist for Autism in Toddlers \(M-CHAT\)](#)

MCT

► [Visual-Motor Integration, Developmental \(VMI\) Test](#)

Mean Length of Utterance (MLU)

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Synonyms

[Typical length](#)

Definition

Mean length of utterance (MLU) is the average number of morphemes per utterance. It is an index of expressive language development used beyond the stage of single words, when a child uses two or more words together in an utterance. It is calculated in 100 spontaneous utterances by counting the number of morphemes in each utterance divided by the total number of utterances. MLU is used as a benchmark to assess individual differences and developmental changes in grammatical development in children in the early stages of language acquisition. Children with autism frequently demonstrate differences in MLU compared to non-autistic developmentally delayed children and typically developing children by producing shorter utterances with fewer grammatical morphemes. Recent research has acknowledged the use and utility of using MLU as an index for language development in children with autism (Condouris et al. 2003; Tager-Flusberg et al. 2009).

The use of MLU as a standard for measuring children’s language development was initially described in 1973 when spontaneous language transcription and morpheme analysis methods were codified and subsequent stages of expressive language development were identified (Brown 1973). Table 1 contains the six stages identified

Mean Length of Utterance (MLU), Table 1 Brown’s stages of language development by mean length of utterance

Stage	MLU	Age	Linguistic characteristics
I.	1.0–2.0	12–26 months	Single-word and two-word utterances that express early semantic relationships, such as action-object and action-location
II.	2.0–2.5	27–30 months	Emergence of grammatical inflections, such as present progressive tense - <i>ing</i>

(continued)

Mean Length of Utterance (MLU), Table 1
(continued)

Stage	MLU	Age	Linguistic characteristics
III.	2.5–3.0	31–34 months	Development of sentence types including declarative, interrogative, negative, imperatives
IV.	3.0–3.75	35–40 months	Emergence of sentence coordination using early conjunctions, such as <i>and</i> , <i>because</i>
V.	3.75–4.5	41–46 months	Continued development of complex sentences including coordination, complementation, and relativization
VI.	4.5 +	47+ months	Continued refinement

by Brown (1973), the corresponding MLU, and the linguistic characteristics of each stage.

See Also

- [Communication Disorder/Communication Impairment](#)
- [Expressive Language](#)
- [Expressive Language Disorder](#)

References and Reading

Brown, R. (1973). *A first language: The early stages*. Cambridge, MA: Harvard University Press.

Condouris, K., Meyer, E., & Tager-Flusberg, H. (2003). The relationship between standardized measures of language and measures of spontaneous speech in children with autism. *American Journal of Speech and Language Pathology*, 12, 349–358.

Eigsti, I.-M., Bennetto, L., & Dadlani, M. B. (2007). Beyond pragmatics: Morphosyntactic development in autism. *Journal of Autism and Developmental Disorders*, 37, 1007–1023.

Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 335–364). New York: Wiley.

Tager-Flusberg, H., Rogers, S., Cooper, J., Landa, R., Lord, C., Paul, R., Rice, M., Stoel-Gammon, C., Wetherby, A., & Yoder, P. (2009). Defining spoken language benchmarks and selecting measures expressive language development for young children with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, 52, 643–652.

Measles and Autism

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Birth of a Hypothesis

In 1998, Andrew Wakefield, a British surgeon, published a case series in the *Lancet* of eight children with autistic spectrum disorder; all had received the combination measles-mumps-rubella vaccine and all had lymphonodular hyperplasia of the small intestine. This study gave birth to the notion that MMR vaccine caused autism. To avoid autism, Wakefield suggested that the three vaccines in MMR vaccine should be given separately. The hypothesis that MMR caused autism was subsequently rejected by epidemiological studies (see section titled “MMR and Autism”).

Wakefield’s hypothesis that MMR vaccine caused autism was rooted in his studies examining the relationship between wild-type measles virus infections and inflammatory bowel disease. These studies set the stage for his notion that measles virus – either wild-type virus or attenuated virus – could cause intestinal disease. Using a monoclonal antibody, Wakefield and coworkers detected measles virus nucleoprotein in biopsy specimens from patients with Crohn’s disease. However, this monoclonal antibody was subsequently found to detect a host, not measles virus, protein and bound to intestinal tissues obtained from patients with or

without inflammatory bowel disease (IBD). Studies detecting measles virus genome by polymerase chain reaction also failed to detect measles virus in patients with IBD.

Wakefield's claim that autism could be avoided by separating the MMR vaccine into its three component parts was also born of studies of wild-type measles virus. Wakefield and coworkers performed a study of children born in England during a 1-week period in 1970 by evaluating the relationship between the timing of childhood infections and IBD. Wakefield observed an increased risk for IBD in children who were infected with measles and mumps virus within the same year. However, this study was limited in that (1) few patients were studied and (2) histories of infection were obtained 4–10 years after the event raising the question of recall bias.

Wakefield later stated that his claims that wild-type measles virus caused IBD were incorrect.

Measles-Mumps-Rubella (MMR) Vaccination

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Definition

In 1998, Dr. Andrew Wakefield and coworkers at the Royal Free Hospital in London published a paper in *The Lancet* titled "Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children." The paper detailed the stories of eight children: all had recently received the combination measles-mumps-rubella (MMR) vaccine and then developed signs and symptoms of autism spectrum disorder. By endoscopy, these researchers also found evidence for lymphoid hyperplasia in Peyer's patches of the small intestine. Wakefield and his coworkers reasoned that the measles

vaccine component of MMR was traveling to the intestine, inducing intestinal disease, and allowing for entrance of encephalopathic proteins that traveled to the brain and caused autism.

Media coverage from this paper caused many parents to withhold MMR vaccine for their children. As a consequence, hundreds of children developed measles in England, the United Kingdom, and Ireland, and four children died of complications from the disease. In the United States, parents of 125,000 children withheld the MMR vaccine accounting, in part, for a measles epidemic in 2008 that was larger than any in more than a decade.

The academic and public health communities in several countries responded by examining retrospectively children who either had or had not received MMR vaccine to determine whether autism was associated with vaccination. The results were clear, consistent, and reproducible. MMR vaccine was not associated with autism. Further, the physiological basis of Wakefield's contention was refuted when other researchers were unable to identify persistent measles vaccine virus genome in autistic children more frequently than in non-autistic children. Nor were the encephalopathic proteins posited by Wakefield ever identified.

References and Reading

- Afzal, M., Ozoemena, L., O'Hare, A., Kidger, K. A., Bentley, M. L., Minor, P. D., Davis, R., Kramarz, P., Bohlke, K., Benson, P., Thompson, R. S., Mullooly, J., et al. (2006). Absence of detectable measles virus genome sequence in blood of autistic children who have had their MMR vaccination during the routine childhood immunization schedule of UK. *Journal of Medical Virology*, 78, 623–630.
- D'Souza, Y., Fombonne, E., & Ward, B. (2006). No evidence of persisting measles virus in peripheral blood mononuclear cells from children with autism spectrum disorder. *Pediatrics*, 118, 1664–1675.
- Dales, L., Hammer, S., & Smith, N. (2001). Time trends in autism and in MMR immunization coverage in California. *Journal of the American Medical Association*, 285, 1183–1185.
- Davis, R., Kramarz, P., Kari, B., et al. (2002). Measles-mumps-rubella and other measles-containing vaccines do not increase the risk for inflammatory bowel disease: A case-control study from the Vaccine Safety DataLink

- Project. *Archives of Pediatrics and Adolescent Medicine*, 155, 354–359.
- DeStefano, F., & Chen, R. (1999). Negative association between MMR and autism. *Lancet*, 353, 1986–1987.
- DeStefano, F., Bhasin, T., Thompson, T., Yeargin-Allsopp, M., Boyle, C., et al. (2004). Age at first measles-mumps-rubella vaccination in children with autism and school-matched control subjects: A population-based study in metropolitan Atlanta. *Pediatrics*, 113, 259–266.
- Farrington, C., Miller, E., & Taylor, B. (2001). MMR and autism: Further evidence against a causal association. *Vaccine*, 19, 3632–3635.
- Fombonne, E. (1999). Are measles infections or measles immunizations linked to autism? *Journal of Autism and Developmental Disorders*, 29, 349–350.
- Fombonne, E., & Chakrabarti, S. (2001). No evidence for a new variant of measles-mumps-rubella-induced autism. *Pediatrics*, 108. Retrieved from <http://www.pediatrics.org/cgi/content/full/108/4/e58>.
- Fombonne, E., & Cook, E., Jr. (2003). MMR and autistic enterocolitis: Consistent epidemiological failure to find an association. *Molecular Psychiatry*, 8, 133–134.
- Halsey, N., & Hyman, S. (2001). Measles-mumps-rubella vaccine and autistic spectrum disorder: Report from the new challenges in childhood immunization conference convened in Oak Brook, Illinois, June 12, 2000. *Pediatrics*, 107. Retrieved from <http://www.pediatrics.org/cgi/content/full/107/5/e84>.
- Honda, H., Shimizu, Y., & Rutter, M. (2005). No effect of MMR withdrawal on the incidence of autism: A total population study. *The Journal of Child Psychology and Psychiatry*, 46, 572–579.
- Kaye, J., Melero-Montes, M., & Jick, H. (2001). Mumps, measles, and rubella vaccine and the incidence of autism recorded by general practitioners: A time trend analysis. *British Medical Journal*, 322, 460–463.
- Madsen, K., Hviid, A., Vestergaard, M., Schendel, D., Wohlfahrt, J., Thorsen, P., et al. (2002). A population-based study of measles, mumps, and rubella vaccination and autism. *New England Journal of Medicine*, 347, 1477–1482.
- Mäkela, A., Nuorti, J., & Peltola, H. (2002). Neurologic disorders after measles-mumps-rubella vaccination. *Pediatrics*, 110, 957–963.
- Offit, P., & Coffin, S. (2003). Communicating science to the public: MMR vaccine and autism. *Vaccine*, 22, 1–6.
- Peltola, H., Patja, A., Leinikki, P., Valle, M., Davidkin, I., Paunio, M., Taylor, B., Miller, E., Farrington, C., Petropoulos, M. C., Favet-Mayaud, I., Li, J., et al. (1998). No evidence for measles, mumps, and rubella vaccine-associated inflammatory bowel disease or autism in a 14-year prospective study. *Lancet*, 351, 1327–1328.
- Smith, M., Bell, L., Ellenberg, S., & Rubin, D. (2008). Media coverage of the MMR-autism controversy and its relationship to MMR immunization rates in the United States. *Pediatrics*, 121, e836–e843.
- Taylor, B., Miller, E., Farrington, C., et al. (1999). Autism and measles, mumps, and rubella vaccine: No epidemiological evidence for a causal association. *Lancet*, 353, 2026–2029.
- Taylor, B., Miller, E., Lingam, R., Andrews, N., Simmons, A., Stowe, J., Wakefield, A., Murch, S., Anthony, A., Linnell, J., Casson, D. A., Malik, M., et al. (2002). Measles, mumps, and rubella vaccination and bowel problems or developmental regression in children with autism: Population study. *British Medical Journal*, 324, 393–396.
- Wakefield, A., Murch, S., Anthony, A., et al. (1998). Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *Lancet*, 351, 637–641. (retracted).
- Wilson, K., Mills, E., Ross, C., McGowan, J., & Jadad, A. (2003). Association of autistic spectrum disorder and the measles, mumps, and rubella vaccine. *Archives of Pediatrics & Adolescent Medicine*, 157, 628–634.

Measure

► Criterion

Measurement Error

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Synonyms

Bias; Error of measurement; Standard error of measurement

Definition

Measurement error is a statistical term that refers to the difference between the obtained value or score and the hypothetical “true” value. Psychological tests rarely, if ever, provide a perfect measure of a construct. The degree of uncertainty or

variability surrounding the obtained value is referred to as measurement error. Measurement error can be divided into two parts: random error and systematic error. Random errors are statistical fluctuations (in either direction) in the measured data due to precision limitations of the measurement device. Random errors usually result from the experimenter's inability to take the same measurement in exactly the same way. Random error is variation due to a wide variety of uncontrollable and unpredictable factors and cannot be eliminated within a study or a test.

Systematic error, also called "bias," may occur in the sampling stage of an experiment as when the sample obtained differs systematically from the target sample. Systematic error can also occur at the pre-measurement, measurement, or calculation stage of an experiment as when an instrument is not used as the manual prescribes or when observer-expectancy or subject-expectancy effects are allowed. Systematic error can include discrepancies in measurement tools, observational bias, or shared environment effects. Systematic errors apply universally across the sample and are referred to as "bias" in social science research. Though difficult to prevent, systematic error can be eliminated once identified. Bias in sampling, measurement, or calculation can be corrected statistically.

Measurement error is negatively correlated with reliability. According to classical test theory, reliability is the proportion of true score to obtained score. The higher the reliability coefficient of a test, the more confidence can be had in making interpretations about any individual test score.

IQ testing, often part of the diagnostic procedure for ASD and other developmental disabilities, provides a good illustration of measurement error. Measurement error, the discrepancy between the obtained score and the true score on a test, results from both random and systematic error. If the same person was to take the same IQ test three separate times, they may well receive three different scores. Their scores would cluster around their "true" IQ score. This is the result of random error such as sometimes guessing correctly and sometimes incorrectly. A certain

amount of random error is unavoidable in measuring any construct, including IQ. Test developers have taken care to limit systematic errors that may result from inconsistent administration of the test by carefully describing administration procedures. IQ test developers have also been diligent in minimizing systematic errors that may bias the test according to gender, cultural, racial, and ethnic differences.

See Also

► [IQ Test](#)

References and Reading

- Buonaccorsi, J. P. (2010). *Measurement error: Models, methods, and applications*. Boca Raton: CRC Press.
Helen, G. (2008). *Understanding research methods and statistics in psychology*. Los Angeles: Sage.

Measuring Language Change Through Natural Language Samples

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Synonyms

[Language outcome measures](#)

Definition

Assessing changes in expressive language ability by analyzing recordings of speech from typical, everyday situations.

Expressive language is integral to social communication and plays a central role in development. Its emergence is one of the strongest predictors of positive long-term outcomes in ASD (Howlin et al. 2004), which is why it has become

the target of many interventions (Kasari et al. 2010). However, widely used methods for characterizing language ability in ASD, like parent questionnaires and standardized tests, are often not fit to detect changes in expressive language resulting from treatments and interventions.

In contrast, natural language samples, which are audio and/or video recordings of spontaneous speech, can be a useful tool for measuring language change in ASD (Barokova and Tager-Flusberg 2018). They can be collected in different contexts (e.g., free play, reading a book, specific activity) by different people (e.g., parent, peer, teacher, researcher) in a variety of settings (e.g., home, school, lab) ensuring the samples are representative of the speaker's everyday language use. A single sample can be coded at the level of the sounds, words, sentences, or appropriate use in context, thus providing a comprehensive picture of the speaker's ability. This is especially useful considering language is one of the most heterogeneous characteristics in ASD (Tager-Flusberg et al. 2009). Measures derived from natural language samples (e.g., frequency of utterances, number of different words used) can be sensitive to subtle changes in language ability that could go undetected if parent questionnaires or standardized tests are used instead. Furthermore, other measures derived from the samples, like number of different topics discussed and amount of echolalia, can serve as proxy for restricted interests and repetitive behaviors, thus capturing change in core autism symptoms beyond social communication.

There are no practice effects associated with the elicitation of natural language samples, so they can be collected pre- and post-intervention, and a similar elicitation procedure can be used with individuals from a wide range of age and language ability, who often comprise the participant pool of ASD intervention and treatment studies.

There are ongoing efforts in the field of ASD and other neurodevelopmental disorders to design standard elicitation protocols for natural language samples (e.g., Berry-Kravis et al. 2013) and determine which measures derived from them have the most sound psychometric properties to detect change in language ability and core symptoms. This is a

necessary step before the wide implementation of these measures as measures of change in treatment and intervention studies.

See Also

- [Communication Interventions](#)
- [Outcome Studies](#)

References and Reading

- Barokova, M., & Tager-Flusberg, H. (2018). Commentary: Measuring language change through natural language samples. *Journal of Autism and Developmental Disorders*, 1–20. <https://doi.org/10.1007/s10803-018-3628-4>.
- Berry-Kravis, E., Doll, E., Sterling, A., Kover, S., Schroeder, S., Mathur, S., & Abbeduto, L. (2013). Development of an expressive language sampling procedure in fragile X syndrome: A pilot study. *Journal of Developmental and Behavioral Pediatrics*, 34, 245–251.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45(2), 212–229.
- Kasari, C., Gulsrud, A., Wong, C., Kwon, S., & Locke, J. (2010). Randomized controlled caregiver mediated joint engagement intervention for toddlers with autism. *Journal of Autism and Developmental Disorders*, 40, 1045–1056.
- Tager-Flusberg, H., Rogers, S., Cooper, J., Landa, R., Lord, C., Paul, R., ... Yoder, P. (2009). Defining spoken language benchmarks and selecting measures of expressive language development for young children with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, 52(3), 643–652.

MECP2 Gene

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Synonyms

[Methyl CpG-binding protein 2](#)

Definition

MECP2 is the primary gene implicated in Rett Syndrome, a neurodevelopmental disorder that primarily affects females. *MECP2* is a gene located on the X chromosome that encodes the protein Methyl CpG-binding protein 2 (MECP2). Inactivation of a single copy of *MECP2* leads to impaired language, social skills, and motor coordination, as well as scoliosis, dysregulated breathing, and seizures in females. Deletion of both copies of *MECP2* in mice mimics the symptoms of Rett Syndrome. Duplication in *MECP2* gene has been implicated in severe male intellectual disability–associated premature death. *MECP2* is expressed throughout the body with strongest expression in neurons. *MECP2* functions as a transcriptional repressor, thus turning off expression of other genes.

See Also

► [Rett Syndrome](#)

References and Reading

- Amir, R. E., Van den Veyver, I. B., Wan, M., Tran, C. Q., Francke, U., & Zoghbi, H. Y. (1999). Rett syndrome is caused by mutations in X-linked *MECP2*, encoding methyl-CpG-binding protein 2. *Nature Genetics*, 23(2), 185–188.
- Guy, J., Hendrich, B., Holmes, M., Martin, J. E., & Bird, A. (2001). A mouse *Mecp2*-null mutation causes neurological symptoms that mimic Rett syndrome. *Nature Genetics*, 27(3), 322–326. <https://doi.org/10.1038/85899>.
- Lombardi, L. M., Baker, S. A., & Zoghbi, H. Y. (2015). *MECP2* disorders: from the clinical to mice and back. *The Journal of Clinical Investigation*, 125(8), 2914–2923.

Medial Frontal Negativity (MFN)

► [Feedback-Related Negativity](#)

Medial Temporal Lobe

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Synonyms

[MTL](#)

Definition

The medial temporal lobe (MTL) is a region within the cerebral cortex comprised of a system of anatomically related structures, including the hippocampal region and the adjacent entorhinal, perirhinal, and parahippocampal cortices. This system is involved in the creation of declarative memory (see Squire, 1991 and 2004 for a review) and also in the regulation of emotional reactions. The MTL has been discussed as one of the candidate neural substrates underlying the social deficits in autism. Evidence from monkey studies demonstrates that neonatal lesions to the MTL result in persistent socioemotional abnormalities later in life. Additionally, declarative memory seems to be affected in high-functioning individuals with ASD who were found to have deficits in episodic memory, with relatively intact semantic memory. Postmortem investigation of the brain in eight individuals with autism also revealed microscopic cytoarchitectonic abnormalities (increased cell density and small cell size) in MTL structures.

See Also

- [Declarative Memory](#)
 ► [Episodic Memory](#)
 ► [Semantic Memory](#)

References and Reading

- Bachevalier, J. (1994). Medial temporal lobe structures and autism: A review of clinical and experimental findings. *Neuropsychologia*, 32(6), 627–648.
- Bachevalier, J. (1996). Brief report: Medial temporal lobe and autism: A putative animal model in primates. *Journal of Autism and Developmental Disorders*, 26(2), 217–220.
- Bauman, M. L., & Kemper, T. L. (2005). Neuroanatomic observations of the brain in autism: A review and future directions. *International Journal of Developmental Neuroscience*, 23(2–3), 183–187.
- Malkova, L., Mishkin, M., Suomi, S. J., & Bachevalier, J. (2010). Long-term effects of neonatal medial temporal ablations on socioemotional behavior in monkeys (*Macaca mulatta*). *Behavioral Neuroscience*, 124(6), 742–760.
- Salmond, C., Ashburner, J., Connelly, A., Friston, K. J., Gadian, D. G., & Vargha-Khadem, F. (2005). The role of the medial temporal lobe in autistic spectrum disorders. *European Journal of Neuroscience*, 22(3), 764–772.
- Squire, L. R., & Zola-Morgan, S. (1991). The medial temporal lobe memory system. *Science*, 253(5026), 1380–1386.
- Squire, L. R., Stark, C. E. L., & Clark, R. E. (2004). The medial temporal lobe. *Annual Review of Neuroscience*, 27, 279–306.

Medicaid and Autism Spectrum Disorder

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Definition

Medicaid is a healthcare program in the United States (US) that covers over 65 million Americans (Centers for Medicare and Medicaid Services 2019). In addition to providing coverage for healthcare services for low-income individuals, pregnant women, and children, Medicaid is a critical support for individuals with disabilities including physical disabilities, intellectual and developmental disabilities (IDD), and autism spectrum disorder (ASD)

(Centers for Medicare and Medicaid Services 2019; Mark et al. 2003; Shattuck et al. 2009; Shea et al. 2018; Vladeck 2003; Wang et al. 2013). Since Medicaid is among the only insurance programs available in the USA for transitioning youth with disabilities, more than ten million adults and children with disabilities are enrolled in Medicaid on the basis of their disability including those with ASD (Medicaid and CHIP Payment and Access Commission (MACPAC) 2017).

Historical Background

Medicaid emerged in US healthcare policy in 1965 as Title XIX of the Social Security Act during the Lyndon B. Johnson administration and has, over the past six decades, been a key payer for services for individuals with disabilities including ASD (Centers for Medicare and Medicaid Services 2015). Early Periodic Screening, Diagnosis, and Treatment (EPSDT) was established in 1967, for example, and provides a comprehensive suite of medically necessary services for all Medicaid-enrolled children through age 20 (English 1993; Wirth and Gabor 2016). EPSDT, depending on the state, pays for ASD screening, diagnosis, and evidence-based treatments like applied behavior analysis (ABA) (Autism Speaks 2017; Wirth and Gabor 2016).

Additionally, Medicaid 1915(c) home- and community-based services (HCBS) waivers, created in 1981, allow individuals with disabilities to receive services in their homes and communities as opposed to institutional settings like nursing homes, hospitals, or state centers (Braddock et al. 2011; Centers for Medicare and Medicaid Services 2015; Ng et al. 2012; Rizzolo et al. 2013). Through HCBS waivers, individuals can receive an array of services and supports such as habilitation, employment supports, respite care, transportation, behavior supports, and community support (Braddock et al. 2011). HCBS waivers vary in availability across the lifespan or specifically for children with ASD and are typically capped in enrollment or have limits on amounts

of services that can be provided. However, HCBS waivers are especially critical for adults with ASD after they are no longer able to access entitlement-based services and supports through the education system and EPSDT (Koffler Miller et al. 2017; Rizzolo et al. 2013; Shattuck et al. 2012).

Current Knowledge

There has been increasing attention to the use of Medicaid-funded services by individuals with ASD of all ages as this population grows, as it is among the only insurers available across the lifespan.

Several studies (Semansky et al. 2011; Shattuck et al. 2009; Shea et al. 2018; Wang and Leslie 2010) have explored the prevalence of individuals with ASD enrolled in the Medicaid system, by state and nationally, and have observed increasing use of Medicaid among individuals on the spectrum over time (Semansky et al. 2011). Exploring the use of Medicaid among the ASD population has yielded a tremendous amount of knowledge about the changing clinical, policy, and services landscape including average age of diagnosis as it differs by race/ethnicity (Mandell et al. 2002), expenditures and societal costs (Cidav et al. 2013; Mandell et al. 2006), program experiences and provider training needs (Koffler Miller et al. 2017), and unwanted outcomes, such as hospitalization (Mandell et al. 2012; Turcotte et al. 2018).

Analyzing Medicaid data in studies allows researchers to observe patterns in service use, expenditures, inpatient versus outpatient service utilization, and medication usage and see how that changes over time as individuals age. A study using national Medicaid data from all states, including Washington, D.C., found that as individuals with ASD are aging into adulthood, they are increasingly reliant on publicly funded services through Medicaid (both psychiatric and medical) and the expenditures for those services are also increasing (Shea et al. 2018). This knowledge helps researchers and policymakers alike to understand the changing needs of this growing population.

Future Directions

Because it has become a primary payer for behavioral health, HCBS, and other services that are necessary for many individuals with ASD (Centers for Medicare and Medicaid Services (CMS) 2017; Koffler Miller et al. 2017) and does not rely on employment for eligibility and coverage (Croen et al. 2014; Koffler Miller et al. 2017), Medicaid is an integral support system for individuals with ASD across the lifespan in the USA. As such, it is critical that researchers focus their efforts on understanding the interplay between ASD and Medicaid through service utilization and expenditures and the changes in service access across the lifespan.

References and Reading

- Autism Speaks. (2017). *ABA through medicaid EPSDT tool kit*. Retrieved from <https://www.autismspeaks.org/medicaid-epsdt>.
- Braddock, D., Hemp, R., Rizzolo, M., Haffer, L., Tanis, E., & Wu, J. (2011). *The state of the states in developmental disabilities: 2011*. Washington, DC: American Association on Intellectual and Developmental Disabilities.
- Centers for Medicare & Medicaid Services. (2015). *Medicare & medicaid milestones: 1937–2015*. Retrieved from <https://www.cms.gov/About-CMS/Agency-Information/History/Downloads/Medicare-and-Medicaid-Milestones-1937-2015.pdf>.
- Centers for Medicare & Medicaid Services. (2019). *Medicaid*. Retrieved from <https://www.medicaid.gov/medicaid/index.html>.
- Centers for Medicare & Medicaid Services (CMS). (2017). *Behavioral Health Services*. Retrieved from <https://www.medicaid.gov/medicaid/benefits/bhs/index.html>.
- Cidav, Z., Lawer, L., Marcus, S. C., & Mandell, D. S. (2013). Age-related variation in health service use and associated expenditures among children with autism. *Journal of Autism and Developmental Disorders*, 43(4), 924–931. <https://doi.org/10.1007/s10803-012-1637-2>.
- Croen, L. A., Zerbo, O., Qian, Y., & Massalo, M. L. (2014). Psychiatric and medical conditions among adults with ASD. In *Kaiser Permanente Northern California. 2014 International Meeting for Autism Research*.
- English, A. (1993). Early and periodic screening, diagnosis, and treatment program (EPSDT): A model for improving adolescents' access to health care. *The Journal of Adolescent Health*, 14(7), 524–526.
- Koffler Miller, K. H., Mathew, M., Nonnemacher, S. L., & Shea, L. L. (2017). Program experiences of adults with

- autism, their families, and providers: Findings from a focus group study. *Autism*, 22, 345–356.
- Mandell, D. S., Listerud, J., Levy, S. E., & Pinto-Martin, J. A. (2002). Race differences in the age at diagnosis among medicaid-eligible children with autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(12), 1447–1453. <https://doi.org/10.1097/00004583-200212000-00016>.
- Mandell, D. S., Cao, J., Ittenbach, R., & Pinto-Martin, J. (2006). Medicaid expenditures for children with autistic spectrum disorders: 1994 to 1999. *Journal of Autism and Developmental Disorders*, 36(4), 475–485. <https://doi.org/10.1007/s10803-006-0088-z>.
- Mandell, D. S., Xie, M., Morales, K. H., Lawer, L., McCarthy, M., & Marcus, S. C. (2012). The interplay of outpatient services and psychiatric hospitalization among Medicaid-enrolled children with autism spectrum disorders. *Archives of Pediatrics & Adolescent Medicine*, 166(1), 68–73. <https://doi.org/10.1001/archpediatrics.2011.714>.
- Mark, T. L., Buck, J. A., Dilonardo, J. D., Coffey, R. M., & Chalk, M. (2003). Medicaid expenditures on behavioral health care. *Psychiatric Services*, 54(2), 188–194.
- Medicaid and CHIP Payment and Access Commission (MACPAC). (2017). *People with disabilities*. Retrieved from <https://www.macpac.gov/subtopic/people-with-disabilities/>.
- Ng, T., Harrington, C., Musumeci, M., & Reaves, E. L. (2012). *Medicaid home and community-based services programs: 2012 data update*. Retrieved from <https://www.kff.org/medicaid/report/medicaid-home-and-community-based-services-programs-2012-data-update/>.
- Rizzolo, M. C., Friedman, C., Lulinski-Norris, A., & Brad-dock, D. (2013). Home and Community Based Services (HCBS) waivers: A nationwide study of the states. *Intellectual and Developmental Disabilities*, 51(1), 1–21. <https://doi.org/10.1352/1934-9556-51.01.001>.
- Semansky, R. M., Xie, M., & Mandell, D. S. (2011). Medicaid's increasing role in treating youths with autism spectrum disorders. *Psychiatric Services*, 62(6), 588. <https://doi.org/10.1176/appi.ps.62.6.588>.
- Shattuck, P. T., Grosse, S., Parish, S., & Bier, D. (2009). Utilization of a medicaid-funded intervention for children with autism. *Psychiatric Services*, 60(4), 549–552. <https://doi.org/10.1176/ps.2009.60.4.549>.
- Shattuck, P. T., Roux, A. M., Hudson, L. E., Taylor, J. L., Maenner, M. J., & Trani, J. F. (2012). Services for adults with an autism spectrum disorder. *Canadian Journal of Psychiatry*, 57(5), 284–291.
- Shea, L. L., Xie, M., Turcotte, P., Marcus, S., Field, R., Newschaffer, C., & Mandell, D. (2018). Brief report: Service use and associated expenditures among adolescents with autism spectrum disorder transitioning to adulthood. *Journal of Autism and Developmental Disorders*, 48(9), 3223–3227. <https://doi.org/10.1007/s10803-018-3563-4>.
- Turcotte, P., Shea, L. L., & Mandell, D. (2018). School discipline, hospitalization, and police contact overlap among individuals with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(3), 883–891. <https://doi.org/10.1007/s10803-017-3359-y>.
- Vladeck, B. C. (2003). Where the action really is: Medicaid and the disabled. *Health Affairs (Millwood)*, 22(1), 90–100. <https://doi.org/10.1377/hlthaff.22.1.90>.
- Wang, L., & Leslie, D. L. (2010). Health care expenditures for children with autism spectrum disorders in Medicaid. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(11), 1165–1171. <https://doi.org/10.1016/j.jaac.2010.08.003>.
- Wang, L., Mandell, D. S., Lawer, L., Cidav, Z., & Leslie, D. L. (2013). Healthcare service use and costs for autism spectrum disorder: A comparison between medicaid and private insurance. *Journal of Autism and Developmental Disorders*, 43(5), 1057–1064. <https://doi.org/10.1007/s10803-012-1649-y>.
- Wirth, B., & Gabor, V. (2016). *Treatment for children with autism spectrum disorders and the EPSDT benefit*.

Medical Conditions Associated with Autism

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Synonyms

Genetic disorders; Nutritional issues; Pica; Seizures; Sleep disorders

Background

Children with autism spectrum disorders (ASD) including autism, Asperger's syndrome, and pervasive developmental disorder, not otherwise specified, have similar medical problems as children without disabilities. These children can have colds or ear infections as well as other typical illnesses of childhood and adolescence. Little research has addressed the medical problems of adults with autism. The primary care provider should monitor the health of individuals with ASD in the “medical home.” The medical home

describes a source of primary care that is accessible, continuous, culturally sensitive, and providing coordinated care. A recent report indicated that children with autism spectrum disorders were less likely than children with other special needs to receive their care in a medical home (Brachlow et al. 2007). This leads to more financial needs for the parents and more problems with access to care (Kogan et al. 2008). In addition to routine and disorder-specific anticipatory guidance, individuals with ASD need to be monitored for medical conditions for which they may be at higher risk including seizures, genetic abnormalities, sleep disorders, disordered eating/nutritional issues, pica, and GI issues.

Seizures

Seizures develop in approximately 10–25% of children with autism (Myers et al. 2007). There is no one type of seizure that is more common than other seizure types. Children with ASD can have absence seizures (staring spells), tonic-clonic seizures (grand mal seizures), myoclonic jerks, atonic seizures (drop attacks), or partial seizures (movements without loss of consciousness). The first peak time for onset of seizures in children with ASD is between 1 and 5 years of age. The second peak occurs in adolescence between 12 and 18 years of age (Myers et al. 2007). The anticonvulsant medications used for other individuals with seizures are used to treat seizures in individuals with ASD.

Landau–Kleffner syndrome is a seizure disorder that may be considered in the differential diagnosis of ASD. This rare type of seizure has a characteristic EEG pattern and can cause a regression in language or aphasia (Kliegman et al. 2007). Landau–Kleffner syndrome is more common in boys, with regression in language and seizure onset in previously socially typical children at around age 5. This regression occurs later than that typically seen in ASD. Some of the behaviors can be similar to those seen in children with ASD including agitation, poor attention span, and irritability. In 70% of the children with Landau–Kleffner syndrome, there is another

seizure type. Obsession and insistence on routine are not typically seen. Anticonvulsant medication may improve symptoms in Landau–Kleffner syndrome. Anticonvulsant medication may be useful for agitation or other behaviors in children with ASD in addition to seizure control, but it does not improve language.

In general, seizures are not a sensitive predictor of outcome in children with ASD. However, the prevalence of seizures is higher among individuals with moderate to severe intellectual disability and those with motor deficits. In children with severe intellectual disability and ASD, seizures may occur in up to 42%. In children without intellectual disability, etiologic or genetic diagnosis, or family history of epilepsy, the rate is only 6–8%. Individuals with autism plus epilepsy have on the average lower IQs and poorer adaptive, behavioral, and social outcomes than those without epilepsy. There are also higher rates of abnormalities on electroencephalography (EEG) without clinical seizures among people with ASD. The rate may be as high as 10–72%. The medical literature has not demonstrated benefit to treating these electroencephalographic abnormalities. There is currently not enough evidence to suggest routine screening EEGs without history or clinical evidence of seizures. Since the rate of seizures is higher in children with ASD, EEG should be considered whenever there are symptoms consistent with seizures (Myers et al. 2007).

Genetic Abnormalities

There is a 60–90% concordance rate of ASD in identical twins, which means that most of the time identical twins both have ASD. In fraternal twins, there is only a 30% concordance rate (Kliegman et al. 2007). Recent studies support that a strong genetic predisposition to autism is genetic, although there is a significant environmental impact as well.

No consistent genetic abnormalities are found in individuals with ASD, although the overall prevalence of genetic abnormalities on testing may be as high as 20%. The rate of fragile X is reported to be 0.045%, and the rate of abnormality

on karyotype (or evaluation of the structure of stained chromosomes by microscopy) is 5% or less (Miller et al. 2010). A new genetic test called chromosomal microarray (CGH) compares the units of DNA in an individual to the composite normal to identify duplications (gain of material) or deletions (loss of material). Abnormalities on CGH microarray are seen in approximately 17–20% of the children with autism spectrum disorders who complete this testing (Shen et al. 2010). Not all of the abnormalities found on CGH microarray in individuals with ASD are associated with or cause autistic symptoms. In the most thorough and recent study, 9% of CGH abnormalities were considered abnormal or likely to be causative of the autism symptoms. Children with dysmorphic (or unusual) features and those with lower cognitive skills are more likely to have abnormalities on genetic testing. Newer genetic testing includes whole exome sequencing to look for regions with increased recessive genes. Nineteen genes have been identified as candidate genes for causing ASD through this new testing. The whole exome sequencing leads to a syndromic diagnosis for 12–15% of children with ASD (Tamimi et al. 2015).

Specific syndromes associated with ASD include X-linked intellectual disability, tuberous sclerosis, and Angelman syndrome, among other disorders. Fragile X is the most common inherited cause of intellectual disability. These children have intellectual disability, macrocephaly, large pinnae, hypotonia, and joint laxity (Johnson et al. 2007). Roughly 20% of the children with fragile X also have ASD symptoms (Hatton et al. 2006). However, only 0.045% of children with ASD have fragile X (Shen et al. 2010). Children with tuberous sclerosis have hypopigmented skin areas, have brain and skin tumors (CNS hamartomas and fibroangiomata), are at a high risk for seizures, and have ADHD or autistic-like behaviors. Angelman syndrome is characterized by global developmental delays especially with language, a wide-based ataxic gait, and progressive spasticity with a particularly jovial personality. This is caused by various genetic abnormalities of chromosome 15 q11–13. Females with symptoms of ASD should be

considered for Rett syndrome caused by the MeCP2 gene. Midline hand-wringing or patting behaviors will typically distinguish these children from those with ASD alone.

Sleep Disorders

Sleep problems are very common in children with autism spectrum disorders and can occur throughout childhood and adolescence. The sleep disorders are variable and can include difficulty settling to sleep, early rising, night waking, sleepwalking, or night terrors. The lack of sleep can affect daytime functioning including behaviors, cognitive skills, and memory. Frequent sleep problems greatly affect family stress and functioning.

Sometimes, there is a medical cause for the sleep problems such as gastroesophageal reflux, obstructive sleep apnea, or nutritional deficiency (e.g., decreased iron stores associated with restless legs syndrome). Children with GERD or gastroesophageal reflux disease can have some vomiting or discomfort during the night from stomach acid refluxing into the esophagus. Obstructive sleep apnea is a respiratory problem during sleep where the airway does not remain patent. This causes the person to awaken. With the interrupted sleep, children can be inattentive or agitated during the day (Myers et al. 2007).

The medical literature does not contain data at this time regarding the frequency of medical causes for sleep difficulties in people with ASD. History and physical exam are used to determine the need to perform a medical workup for causes of sleep problems. In most children, behavioral measures are used to treat the sleep problems. Behavioral measures can include setting up a bedtime routine. Medications sometimes used include melatonin, alpha-adrenergic agonists (such as clonidine and guanfacine), and antihistamines (such as diphenhydramine). Further research needs to be done on the causes of sleep difficulties in people with ASD as well as effective treatments.

GI Issues

The prevalence of GI issues in children with autism spectrum disorders varies from 9% to 72%. The most common problems are chronic

constipation or chronic diarrhea. One reason for the difficulty in assessing this problem is the communication difficulty children with ASD often have. Many children are nonverbal, and many of those who can communicate have difficulty with describing subjective feelings or isolating a source of pain. Some children will not have identifiable symptoms relative to the GI tract but instead will have behavioral changes or changes in sleep patterns as evidence of GI issues. Careful evaluation including full physical exam and medical history are indicated anytime there are major changes in the behavior of an individual with ASD.

The common GI problems that are seen in other people can also be seen in people with ASD. An individual with constipation might have hard infrequent stools or might stool daily with distress. They might also have behavioral problems that are only clarified when physical exam palpation stool or an X-ray identifies significant constipation. Treatment might include a bowel regimen. Chronic diarrhea is also reported in people with ASD. The same etiologic workup entertained for other patients should be performed. Abdominal discomfort may be due to common problems such as lactose intolerance or less frequent medical disorders such as celiac disease. History and physical exam are important sources of information to the clinician in determining initial workup and appropriate referral to a gastroenterologist.

The evidence for autistic enterocolitis (leaky gut) is limited at this time. The evaluation of children with ASD and GI symptoms found no difference in enzyme activity, intestinal permeability, or inflammation compared to typically developing children (Kushak et al. 2016). Prospective studies need to be done on the causes and treatments of the GI symptoms reported in individuals with ASD.

Nutritional Deficiencies

Food aversions are commonly reported in children and youth with ASD. In addition to self-restriction, dietary restriction for therapeutic reasons might occur.

The gluten-free and casein-free diet is a popular dietary treatment for the symptoms of autism.

This involves removing all foods that contain gluten from wheat, barley, and rye and casein, found in milk products. This diet may impact protein quality of food consumed (Arnold et al. 2003). It also requires provision of an alternate source of calcium and vitamin D than dairy products.

Children with ASD who have a limited food repertoire are at risk for consumption of an inadequate diet (Bandini et al. 2010). Many children with ASD are supplemented with vitamins, but this can lead to excess in some vitamins. (Stewart et al. 2015) The impact of a limited diet on long-term health requires further study.

Pica

Pica is the ingestion and mouthing of nonfood objects. Chewing or mouthing toys and things in the environment is typical in children less than 12 months of age. Pica can persist because of sensory reasons, delay to a younger developmental level, and anxiety and may be due to nutritional deficiencies such as iron deficiency. Pica can also lead to increased lead levels from ingestion of lead dust in the environment or items manufactured with lead. It is advisable to continue monitoring lead levels in children who continue to have pica beyond the toddler stage when monitoring typically occurs (Myers et al. 2007).

Pathophysiology

There are a number of medical conditions associated with autism spectrum disorders (ASD). Except for some genetic and neurologic abnormalities, medical conditions associated with ASD are not responsible for the symptoms of autism. Discomfort and medical illness can exacerbate behavioral symptoms. Medical and dental causes of new-onset behaviors should be considered.

Hypotheses have been generated associating GI pathology such as leaky gut or increased permeability with symptoms of ASD. More recent studies have not confirmed this association; however, the potential for disorders affecting the brain and gut requires further study. There is no specific

pathology identified to cause the constipation, diarrhea, or abdominal pain reported in children with ASD. Lymphonodular hyperplasia (lymph nodes in the intestine) may be a normal variant. Investigators are pursuing the possibility of immunologic differences in the intestine in children with ASD.

The etiology of the sleep difficulties of children and youth with ASD may be related to abnormal melatonin metabolism, atypical sleep architecture, restless legs syndrome, or behavioral sleep disorders. Etiologic and therapeutic studies are ongoing to address sleep in individuals with ASD.

See Also

- [Gastrointestinal Disorders and Autism](#)
- [Genetics](#)
- [Intestinal Permeability Studies](#)
- [Leaky Gut Syndrome](#)
- [Physical and Neurological Examination](#)
- [Pica](#)
- [Seizure](#)
- [Seizure Disorder](#)

References and Reading

- Arnold, G. L., Hyman, S. L., Mooney, R. A., & Kirby, R. S. (2003). Plasma amino acids profiles in children with autism: Potential risk of nutritional deficiencies. *Journal of Autism and Developmental Disorders*, 33, 449–454.
- Bandini, L. G., Anderson, S. E., Curtin, C., Cermak, S., Evans, E. W., Scampini, R., et al. (2010). Food selectivity in children with autism spectrum disorders and typically developing children. *Journal of Pediatrics*, 157, 259–264.
- Brachlow, A. E., Ness, K. K., McPheeters, M. L., & Gurney, J. G. (2007). Comparison of indicators for a primary care medical home between children with autism or asthma and other special health care needs. *Archives of Pediatrics and Adolescent Medicine*, 161, 399–405.
- Buie, T., Campbell, D. B., Fuchs, G. J., 3rd, et al. (2010). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125(Suppl 1), S1–S18.
- Hatton, D. D., Sideris, J., Skinner, M., Mankowski, J., Bailey, D. B., Jr., Roberts, J., et al. (2006). Autistic behavior in children with fragile X syndrome: Prevalence, stability, and the impact of FMRP. *American Journal of Medical Genetics. Part A*, 140A, 1804–1813.
- Johnson, C. P., Myers, S. M., & American Academy of Pediatrics Council on Children with Disabilities. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120, 1183–1215.
- Kalsner, L., Twachtman-Bassett, J., Tokarski, K., Stanley, C., Dumont-Mathieu, T., Cotney, J., Chamberlain, S. (2008). Genetic testing including targeted gene panel in a diverse clinical population of children with autism spectrum disorder: Findings and implications. *Mol Genet Genomic Med*. 6(2):171–185. <https://doi.org/10.1002/mgg3.354>. Epub 2017 Dec 21.
- Kliegman, R. M., Behrman, R. E., Jenson, H. B., & Stanton, B. M. D. (2007). *Nelson textbook of pediatrics* (18th ed.). Philadelphia: Saunders.
- Kodak, T., & Piazza, C. C. (2008). Assessment and behavioral treatment of feeding and sleeping disorders in children with autism spectrum disorders. *Child and Adolescent Psychiatric Clinics of North America*, 17, 887–905. x–xi.
- Kogan, M. D., Strickland, B. B., Blumberg, S. J., Singh, G. K., Perrin, J. M., & van Dyck, P. C. (2008). A national profile of the health care experiences and family impact of autism spectrum disorder among children in the United States, 2005–2006. *Pediatrics*, 122, e1149–e1158.
- Kushak, R. I., Buie, T. M., Murray, K. F., Newburg, D. S., Chen, C., Nestoridi, E., & Winter, H. S. (2016). Evaluation of intestinal function in children with **autism** and gastrointestinal symptoms. *Journal of Pediatric Gastroenterology and Nutrition*, 62(5), 687–691.
- Levy, S. E., & Hyman, S. L. (2008). Complementary and alternative medicine treatments for children with autism spectrum disorders. *Child and Adolescent Psychiatric Clinics of North America*, 17, 803–820. ix.
- Miller, D. T., Adam, M. P., Aradhya, S., Bisecker, L. G., Brothman, A. R., Carter, N. P., et al. (2010). Consensus statement: Chromosomal microarray is a first-tier clinical diagnostic test for individuals with developmental disabilities or congenital anomalies. *American Journal of Human Genetics*, 86, 749–764.
- Myers, S. M., Johnson, C. P., & American Academy of Pediatrics Council on Children with Disabilities. (2007). Management of children with autism spectrum disorders. *Pediatrics*, 120, 1162–1182.
- Shen, Y., Dies, K. A., Holm, I. A., Bridgemohan, C., Sobeih, M. M., Caronna, E. B., et al. (2010). Clinical genetic testing for patients with autism spectrum disorders. *Pediatrics*, 125, e727–e735.
- Stewart, P. A., Hyman, S. L., Schmidt, B. L., Macklin, E. A., Reynolds, A., Johnson, C. R., James, S. J., & Manning-Courtney, P. (2015). Dietary supplementation in children with autism spectrum disorders: Common, insufficient, and excessive. *Journal of the Academy of Nutrition and Dietetics*, 115(8), 1237–1248.

Tammimies, K., Marshall, C. R., Walker, S., Kaur, G., Thiruvahindrapuram, B., Lionel, A. C., Yuen, R. K., Uddin M., Roberts W., Weksberg R., Woodbury-Smith M., Zwaigenbaum L., Anagnostou E., Wang Z., Wei J., Howe J. L., Gazzellone M. J., Lau L., Sung W. W., Whitten K., Vardy C., Crosbie V., Tsang B., D'Abate L., Tong W. W., Luscombe S., Doyle T., Carter M. T., Szatmari P., Stuckless S., Merico D., Stavropoulos D. J., Scherer S. W., Fernandez B. A. (2015). *JAMA*. 1;314 (9):895–903. <https://doi.org/10.1001/jama.2015.10078>.

Medical Education on Autism

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Historical Background

Research shows that medical practitioners, including family physicians and pediatricians, are often the first healthcare professionals whom families of individuals with a suspected diagnosis of ASD are referred to in order to receive a diagnosis and/or to receive treatment (Self et al. 2015). Nonetheless, families of individuals with ASDs have raised concerns about the diagnostic and treatment processes of ASDs. Families of individuals with ASDs often feel unsupported because of medical practitioners' lack of knowledge and competency around the diagnosis and treatment of ASDs, misdiagnosis with other disabilities (e.g., attention deficit hyperactivity disorder), as well as multiple referrals (Brookman-Frazee et al. 2012; Glazzard and Overall 2012). Furthermore, research investigating the experiences of medical practitioners providing care for individuals with ASDs have revealed that many providers lack expertise about the diagnosis of, or the identification of, ASDs (Brookman-Frazee et al. 2012; Dosreis et al. 2006). Additionally, medical professionals often report that they do not receive ASD-specific education and training during medical school (Finke et al. 2010), and many medical

practitioners feel unskilled when providing care for individuals with ASD (Crais et al. 2014).

Current Knowledge

Despite medical professionals reporting that they often feel unprepared to address ASDs in primary care, relatively little is known about the actual content within medical education regarding ASD and developmental disabilities. Medical practitioners often attribute their lack of preparedness regarding the diagnosis and treatment of ASDs to the lack of education and training they receive during their undergraduate medical education (Carbone et al. 2010; Daniels et al. 2014; Rhoades et al. 2007). It is important to note that formal medical education differs widely across countries, provinces or states, and even medical schools. Therefore, there is a lack of consistency within medical education when it comes to formal training about ASDs. While some medical professionals may feel prepared to work with individuals with ASD and their families, other providers may report that they have had no experience or education about ASD.

By way of example, many pediatricians have reported a lack of ASD-specific education and training during medical school (Finke et al. 2010). Additionally, some of the physicians (i.e., family physicians, pediatricians, developmental pediatricians, and psychiatrists) in Ontario, Canada, have indicated that they may have received only one lecture regarding ASD during their undergraduate medical education and that they often had to educate themselves through attending conferences, workshops, and trainings (Ghaderi and Watson in review). Nonetheless, medical students who have received hands-on and in vivo experience working with individuals with ASDs during their medical education have reported higher levels of knowledge, comfort, as well as more positive attitudes toward working with this population compared to students who have not received such trainings (Havercamp et al. 2016). There is a lack of existing research and poor training (e.g., limited and not very efficient). Consequently, medical providers are often ill-prepared to address the needs of their patients with ASDs.

Future Directions

Given medical practitioners' concerns regarding the lack of education and training on ASD during medical education, it is imperative for future research to investigate the current medical education and the curriculum content on ASDs specifically. There is a demonstrated need for increased exposure to consistent, formalized education on ASD within the medical field.

Some future research and practice recommendations include:

1. The promotion of further ASD-specific education and training in medical schools that includes formal education on key issues regarding the prevalence, identification, diagnosis, and treatment of ASDs. It is possible that students currently do not have an interest in these topics and greater exposure to individuals with ASDs, and other developmental disabilities, may increase their awareness and interest in working in this field.
2. The integration of clinical and hands-on working experiences with the ASD population is important. Employing teaching strategies that include a combination of didactic teaching, small-group discussions, and case studies would improve medical students' knowledge regarding the specific needs of individuals with ASDs as they transition into medical practice.
3. There is a need for further formal education and training opportunities that include experiential learning. Additionally, medical learners may also benefit from lectures or discussions delivered by individuals with ASDs and lived experiences, as well as educational sessions paired with standardized patient encounters in order to improve their knowledge regarding the diagnosis and treatment of ASDs.
4. Given the large number of topics learners must cover in their medical education, students should also feel prepared and be encouraged to add to their own education and acquire information from informed and reliable sources, when necessary. Learners should be encouraged to engage in individual learning opportunities about ASDs and other developmental disabilities.

See Also

Autism Physician Handbook (Canadian Edition): https://autismcanada.org/wp-content/uploads/2018/01/PhyHandbook_EN_2018.pdf

Canadian Paediatric Society: Condition-specific screening tools and rating scales <https://www.cps.ca/en/tools-outils/condition-specific-screening-tools-and-rating-scales#autism>

Clinical Practice Guidelines - recently updated primary care guidelines for adults with intellectual and developmental disabilities: Sullivan, W.F., Diepstra, H., Heng, J., Ally, S., Bradley, E., Casson, I., ... Witherbee, S. (2018). Primary care of adults with intellectual and developmental disabilities: 2018 Canadian consensus guidelines. *Canadian Family Physician*, 64(4), 254–279. <http://www.cfp.ca/content/cfp/64/4/254.full.pdf>

Healthcare professionals in the USA have been mandated to utilize early developmental screening tools in order to identify developmental issues in children as young as 9 months of age (Self et al. 2015).

Theoretical Models and Autism Encyclopedia Entry

References and Reading

- Brookman-Frazee, L., Baker- Ericzén, M., Stadnick, N., & Taylor, R. (2012). Parent perspective on community mental health services for children with autism spectrum disorders. *Journal of Child and Family Studies*, 21(4), 533–544.
- Carbone, P. S., Behl, D. D., Azor, V., & Murphy, N. A. (2010). The medical home for children with autism Spectrum disorders: Parent and pediatrician perspectives. *Journal of Autism and Developmental Disorders*, 40(3), 317–324.
- Crais, E.R., Mccomish, C.S., Humphreys, B.P., Watson, L.R., Baranek, G.T., Reznick, J.S., ..., Earls, M. (2014). Pediatric healthcare professionals' views on autism spectrum disorder screening at 12–18 months. *Journal of Autism and Developmental Disorders*, 44(9), 2311–2328.
- Daniels, A. M., Halladay, A. K., Shih, A., Elder, L. M., & Dawson, G. (2014). Approaches to enhancing the early detection of autism spectrum disorders: A systematic review of the literature. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(2), 141–152.

- Dosreis, S., Weiner, C. L., Johnson, L., & Newschaffer, C. J. (2006). Autism spectrum disorder screening and management practices among general pediatric providers. *Journal of Developmental and Behavioral Pediatrics*, 27, S88–S94.
- Finke, E. H., Drager, K. D. R., & Ash, S. (2010). Paediatricians' perspectives on identification and diagnosis of Autism Spectrum Disorders. *Journal of Early Childhood Research*, 8(3), 254–268.
- Ghaderi, G., & Watson, S.L. (2017). "In medical school, you get far more training on medical stuff than developmental stuff": Perspectives on ASD from Ontario physicians. *Journal of Autism and Developmental Disabilities* (in review).
- Glazzard, J., & Overall, K. (2012). Living with autistic spectrum disorder: Parental experiences of raising a child with autistic spectrum disorder (ASD). *Support for Learning*, 27(1), 37–45.
- Havercamp, S. M., Ratliff-Schaub, K., Macho, P. N., Johnson, C. N., Bush, K. L., & Souders, H. T. (2016). Preparing tomorrow's doctors to care for patients with autism spectrum disorder. *Intellectual and Developmental Disabilities*, 54(3), 202–216 230, 232.
- Rhoades, R. A., Scarpe, A., & Salley, B. (2007). The importance of physician knowledge of autism spectrum disorder: Results of a parent survey. *BMC Pediatrics*, 7(37), 1–10.
- Self, T. L., Parham, D. F., & Rajagopalan, J. (2015). Autism spectrum disorder early screening practices: A survey of physicians. *Communication Disorders Quarterly*, 36(4), 195–207.

Medical Evaluation in Autism

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Definition

All children diagnosed with an autism spectrum disorder should have an evaluation by a medical professional to consider the potential causes of developmental disability and evaluate the individual for common co-occurring medical conditions. The medical evaluation includes a thorough medical history including family history, physical examination, and neurological examination. Consideration of genetic causes requires laboratory testing most commonly using chromosomal

microarray if no specific syndrome requires workup. Other genetic screening tests might include testing for X-linked intellectual disability, Rett syndrome, or other diagnoses suggested by the history and physical examination.

Historical Background

In 1941, Dr. Leo Kanner first described autism as a discrete disorder. Around the same time, Dr. Hans Asperger identified a similar disorder in Germany. While a similarity to childhood schizophrenia led many to consider autism a psychiatric disorder, Dr. Bruno Bettelheim in the 1950s attributed it to cold parenting. In the 1960s, Dr. Bernard Rimland proposed that there was a biologic basis for autism. This led the way to thinking about medical evaluation for etiology of ASD. The capacity to karyotype and see genetic abnormalities in the chromosomes associated with disease states was developed in the 1960s. This advance permitted the identification of the chromosomal abnormalities responsible for genetic syndromes. Chromosome analysis was not routinely done on children with developmental differences until 1966. The biggest breakthrough in etiologic evaluation of developmental disability occurred when phenylketonuria or PKU was identified as a metabolic problem that could cause intellectual disability and autism. This disorder resulted in ASD in 20% of untreated cases. Now, with newborn screening, it is a preventable cause of ASD. It is routine for babies in the USA to be screened for PKU and other metabolic disorders. Prenatal infections need to be considered in the etiology of ASD. Prior to the introduction of the rubella vaccine, congenital rubella was a cause of autism that has now been all but eliminated.

Advances in neuroimaging allow for investigation of central nervous system anomalies when indicated by history and physical examination.

Current Knowledge

The current recommendation for evaluation of children diagnosed with autism spectrum

disorders is to start with a thorough history and physical exam. The clinician should obtain a three-generation genetic family history and full history including past medical history, developmental history, and behavioral history. The history often guides the rest of the evaluation. All children with language delays should have a hearing evaluation. In children who are school age and verbal, school-based hearing screening may suffice (Myers et al. 2007).

An important part of the medical evaluation of an individual with autism spectrum disorders is measuring growth parameters: height, weight, and head circumference. A calculated BMI should be included at each health visit since obesity is a serious co-occurring health problem in people with ASD. Head circumference is often larger than average in early childhood in children with ASD. Medical problems that occur with greater frequency in people with ASD than the general population should be screened for in the medical evaluation and then routinely in health maintenance visits. While physical symptoms may be related to an etiologic cause of ASD, they may be related to core symptoms such as anxiety or repetitive behaviors. Anxiety appears to be related to both intestinal and sleep issues (Ferguson et al. 2017; Mazurek and Petroski 2015). All of these disorders may reflect differences in autonomic tone. Individuals with ASD should be monitored for the following associated medical problems:

- Sleep disorders like insomnia and night waking
- Gastrointestinal problems like constipation and selective eating
- Seizures
- Attention deficit hyperactivity disorder
- Anxiety disorders
- Wandering or elopement (the major cause of death in children with ASD is accidents)

The next level of testing depends on the combined information from the history and physical exam. Routine EEG and neuroimaging are not recommended by the American Academy of Pediatrics at this time. MRI is indicated with recent or atypical regression, an abnormal neurologic

exam, or a suggestive history for neurologic disease. EEG is indicated with recent or atypical regression or a history suggestive of seizures.

The current newborn screening in the USA will pick up many of the inborn errors of metabolism such as PKU, galactosemia, and congenital hypothyroidism. If the newborn screening was not completed or is not available, then metabolic testing with quantitative amino acids should be considered in a child whose history and physical exam suggest possible inborn errors. Maternal screening for rubella identifies pregnancies at risk for congenital rubella syndrome.

Comparative genomic hybridization microarray is considered the first-line genetic evaluation in the workup of ASD without a suspected specific etiology by the American College of Genetics. Studies report that from 10% to 17% of children with an ASD will have an abnormality on this testing (Shen et al. 2010). However, most of these abnormalities are considered benign. Children with intellectual disability, female gender, or dysmorphic feature may be more likely to have a genetic etiology identified from these evaluations (Roesser 2011). While comparative genomic hybridization (CGH) microarray can pick up a large number of duplications or deletions, there are some translocations that can only be seen on karyotype. Karyotype examination is often obtained when there is a family history of greater than two miscarriages or a specific diagnosis such as Down syndrome is suspected.

A newer technology, whole exome sequencing, evaluates the entire genetic code but focuses on areas with more functionally important genes (called the exome). The whole exome sequencing leads to confirmation of a syndromic diagnosis for 12–15% of children with ASD (Tammimies et al. 2015). Overall 30% of children with ASD are found to have genetic abnormalities on WES.

History and examination may guide the clinician to order testing for specific disorders associated with ASD. Genetic testing for Rett syndrome should be considered in girls diagnosed with ASD. They may demonstrate microcephaly or deceleration of head growth in early childhood. Other clinical markers of Rett syndrome include stereotypical hand movements and psychomotor

regression. Patients with webbing of toes, mildly dysmorphic features, or failure to thrive could have Smith-Lemli-Opitz syndrome. In this case, 7-dehydrocholesterol level should be sent. A lipid panel may also pick up the extremely low cholesterol level. Children with ASD and seizures should be considered for a possible diagnosis of Angelman syndrome. It is associated with a chromosome 15 abnormality in the same region as Prader-Willi syndrome. Boys with intellectual disability/learning disability should be considered for the DNA testing for fragile X syndrome especially if there is a family history on the maternal side of learning challenges. About 20–30% of children with fragile X will also have ASD. However the yield of fragile X testing in children with ASD is quite low at 0.05% (Roesser 2011; Chen 2011; Weinstein et al. 2017).

Children with macrocephaly may be at increased risk of PTEN (phosphatase and tensin homolog) gene mutations. PTEN mutations alter tumor suppression and are associated with hamartomatous syndromes such as Bannayan-Riley-Ruvalcaba syndrome and Cowden syndrome. Family members are at risk for tumors in the thyroid, breast, and uterus. Any child with macrocephaly and family history of any of these cancers should have genetic evaluation for PTEN mutations.

Referral to a geneticist may be indicated for assessment of genetic etiologies of ASD for genetic workup, evaluation for suspected metabolic disease, diagnosis of dysmorphic syndromes, and genetic counseling. Children with regression, movement disorders, microcephaly, neurocutaneous lesions, midline facial defects, abnormalities in neurologic examination, or a history of seizures may require referral to a neurologist. MRI may be necessary. A screening MRI is not necessary for macrocephaly alone or single café au lait spot. EEG may be needed if there is suspicion of seizures. Seizures are seen in 25% of children with autism.

Lead testing is not part of the routine etiologic workup for autism but could be considered for children with pica or for those at environmental risk if not previously obtained in the course of well-child care. Children with pica are at

continued risk of lead poisoning at a much older ages. Children with limited diets may benefit from nutritional evaluation to ensure adequate growth and development. Low iron stores might be associated with sleep problems related to restless legs syndrome. Iron stores may be low before anemia occurs.

Future Directions

Over time, genetic diagnostics have become more accurate. Initially testing could only identify large abnormalities using karyotype analysis. Now, smaller abnormalities can be detected. The technology is emerging, so the clinical significance of some of the findings is not clear. The use of large datasets will permit investigation of the relationship of specific genetic abnormalities, neurophysiologic profiles on EEG, and MRI findings with both etiology and treatment response (Payakachat et al. 2016). As the cost of genetic testing decreases and the implications of the results are linked with actionable steps in care, more individuals with ASD will be tested. New uses for MRI, functional imaging, and brain spectroscopy will be evaluated, and as the findings are linked to clinical decision-making, outcomes will change the recommendations for use.

See Also

- [Lead Exposure and Autism](#)
- [Medical Conditions Associated with Autism](#)
- [Rubella](#)
- [Seizure](#)

References and Reading

- Ferguson, B. J., Marler, S., Altstein, L. L., Lee, E. B., Akers, J., Sohl, K., McLaughlin, A., Hartnett, K., Kille, B., Mazurek, M., Macklin, E. A., McDonnell, E., Barstow, M., Bauman, M. L., Margolis, K. G., Veenstra-VanderWeele, J., & Beversdorf, D. Q. (2017). Psychophysiological associations with gastrointestinal symptomatology in autism spectrum disorder. *Autism Research*, 10(2), 276–288. <https://doi.org/10.1002/aur.1646>. Epub 2016 Jun 20. PMID:27321113.

- Johnson, C. P., Myers, S. M., & American Academy of Pediatrics, Council on Children with Disabilities. (2007). Identification and evaluation of children with autism spectrum disorder. *Pediatrics*, 120(5), 1183–1215.
- Lo, S. (2011). Laboratory testing in infants and children. In M. Kleigman (Ed.), *Nelson textbook of pediatrics* (19th ed.). Philadelphia: Saunders. Chapter 707.
- Mazurek, M. O., & Petroski, G. F. (2015). Sleep problems in children with autism spectrum disorder: Examining the contributions of sensory over-responsivity and anxiety. *Sleep Medicine*, 16(2), 270–279. <https://doi.org/10.1016/j.sleep.2014.11.006>. Epub 2014 Nov 28.
- Miller, D. T., Adam, M. P., Aradhya, S., Bisecker, L. G., Brothman, A. R., Carter, N. P., et al. (2010). Consensus statement: Chromosomal microarray is a first-tier clinical diagnostic test for individuals with developmental disabilities or congenital anomalies. *American Journal of Human Genetics*, 86, 749–764.
- Myers, S. M., Johnson, C. P., & American Academy of Pediatrics, Council on Children with Disabilities. (2007). Management of children with autism spectrum disorders. *Pediatrics*, 120(5), 1162–1182.
- Payakachat, N., Tilford, J. M., & Ungar, W. J. (2016). National Database for Autism Research (NDAR): Big data opportunities for health services research and health technology assessment. *Pharmaco Economics*, 34(2), 127–138. <https://doi.org/10.1007/s40273-015-0331-6>.
- Roesser, J. (2011). Diagnostic yield of genetic testing in children diagnosed with autism spectrum disorders at a regional referral center. *Clinical Pediatrics*, 50(9), 834–843.
- Schaefer, G. B., & Mendelsohn, N. J. (2008). Clinical genetics evaluation in identifying the etiology of autism spectrum disorders. *Genetics in Medicine*, 10, 301.
- Shen, Y., Dies, K. A., Holm, I. A., Bridgemohan, C., Sobeih, M. M., Caronna, E. B., et al. (2010). Clinical genetic testing for patients with autism spectrum disorders. *Pediatrics*, 125, e727.
- Tammimies, K., Marshall, C. R., Walker, S., Kaur, G., Thiruvahindrapuram, B., Lionel, A. C., Yuen, R. K., Uddin, M., Roberts, W., Weksberg, R., Woodbury-Smith, M., Zwaigenbaum, L., Anagnostou, E., Wang, Z., Wei, J., Howe, J. L., Gazzellone, M. J., Lau, L., Sung, W. W., Whitten, K., Vardy, C., Crosbie, V., Tsang, B., D'Abate, L., Tong, W. W., Luscombe, S., Doyle, T., Carter, M. T., Szatmari, P., Stuckless, S., Merico, D., Stavropoulos, D. J., Scherer, S. W., & Fernandez, B. A. (2015). Molecular diagnostic yield of chromosomal microarray analysis and whole-exome sequencing in children with autism spectrum disorder. *JAMA*, 314(9), 895–903. <https://doi.org/10.1001/jama.2015.10078>.
- Weinstein, V., Tanpaiboon, P., Chapman, K. A., Ah Mew, N., & Hofherr, S. (2017). Do the data really support ordering fragile X testing as a first-tier test without clinical features? *Genetics in Medicine*. <https://doi.org/10.1038/gim.2017.64>.

Medical Home and ASD

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Definition

An effective approach for delivering comprehensive pediatric primary care medical services to all children, including those with ASD.

Historical Background

Children with autism spectrum disorder (ASD) and their families receive many services delivered in their homes, schools, early care and education programs, and community agencies, but the primary health-care site plays a unique and significant role in the care for children with, and at risk for, ASD. Although all children may not receive home visiting services, or may not attend early care and education programs, more than 90 percent of young children, across all racial, ethnic, and socioeconomic categories, have a visit with a health provider each year (Department of Health and Human Services 2014). The American Academy of Pediatrics (AAP) guidelines for pediatric primary care, which are accepted by state Medicaid programs and commercial insurance plans, call for 13 well-child visits before school entry (American Academy of Pediatrics 2015), at which child health providers are to perform the following: immunizations, sensory and metabolic screenings, developmental screening, complete physical exams, and anticipatory guidance to support parents in raising their children. These visits provide child health providers with frequent opportunities to identify children who may have, or may be at risk for, ASD, connect them to diagnostic and intervention services, and monitor their health and development over time.

An effective approach for delivering pediatric primary care medical services to all children, including those with ASD, is the medical home

model. A pediatric medical home provides access outside of traditional office hours, continuity of care with a team of providers who work in partnership with families, linkage to medical and non-medical services outside of the practice, care that is coordinated across all services that children use, and services that meet the needs of all patients in a context that is respectful of their cultural and religious beliefs (Holt et al. 2010). Within the medical home model, several professionals participate in the care of children, including pediatricians, nurse practitioners, physician assistants, family physicians, and a variety of office staff members that include nurses, medical assistants, care coordinators, and administrative staff. A hallmark of the medical home model is that all staff members work together as a team with families to support the needs of the children served in the practice.

The AAP developed the medical home model to guide child health providers in the care of children with complex medical needs (American Academy of Pediatrics 1992), and the concept has since been expanded to recognize that all children can benefit from having a medical home (American Academy of Pediatrics 2004). The AAP specifically called out the role of medical home in the care for children with ASD in 2013 (American Academy of Pediatrics Autism Expert Panel 2013) with publication of a resource guide for child health providers. The guide includes information on early identification, screening tools, coding to ensure reimbursement, and informational handouts for families.

The medical home has also become the pervasive model of care for adults and is a popular component of health-care reform initiatives in several states (Van Vleet and Paradise 2014) that are working to transform their health-care delivery systems to contain costs, improve population outcomes, and enhance the patient experience. There are four national medical home accreditation programs, any one of which, when successfully completed, allows primary care practitioners to participate in enhanced payment programs offered through insurance plans (American Academy of Family Physicians 2015).

The widescale adoption of the medical home model is based on research showing that care from a medical home is of higher quality and costs less than care delivered in nonmedical home sites (Nielsen et al. 2016). For children with special needs, the medical home model has been shown to improve timeliness of care, health outcomes, family centeredness, and family functioning (Homer et al. 2008).

Current Knowledge

Parents of children with ASD are less likely to report that their child has a medical home than parents of children in general or of children with other types of special needs (Brachlow et al. 2007), despite national trends toward medical adoption, evidence that the model meets the needs of children with special needs, and incentives for child health providers to transform their practices to be medical homes. As a result, families with children with ASD have a harder time gaining access to specialty services (Cheak-Zamora and Farmer 2015) and are more likely than families of children with other neurological disorders to make their own connections to neurology services (Ming et al. 2011). Families of children with ASD who report having a medical home also report fewer unmet needs (Farmer et al. 2014) than children whose parents report that they do not have a medical home.

Lower levels of parent satisfaction with primary care as well as advocacy and education received are also reported by families of children with ASD compared to other children with special needs (Hyman and Johnson 2012; Carbone et al. 2010a). Pediatricians report that they are not adequately trained, lack the necessary time, and are challenged in meeting the needs of children with ASD and their families (Carbone et al. 2010b; Golnik et al. 2009).

In addition to providing accessible, coordinated, and family-centered care, the medical home entails specific responsibilities that ensure early detection of ASD among all their patients, connection of children for whom there are

concerns to diagnostic and intervention services, and ongoing monitoring of health and development.

Early identification of children with ASD is facilitated by developmental surveillance and screening for all children (American Academy of Pediatrics 2006). Developmental surveillance includes the following four major elements that support early detection of children with, or at risk for, developmental delays: soliciting parental concerns, maintaining a longitudinal record of development, observing the child, and obtaining input from individuals other than parents who care for the child (e.g., early care and education providers). Through developmental surveillance, child health providers can probe for clinical cues related to attention, social skills, and communication and facilitate early identification of children who may have ASD (Carbone et al. 2010b). Developmental screening relies on the use of a formal tool. The AAP recommends that child health providers screen for developmental delay at the 9-, 18-, and 30-month well-child visits (American Academy of Pediatrics 2006). The AAP also recommends that child health providers use a formal tool to screen specifically for ASD at the 18- and 24-month visits. The most commonly used screening tool for ASD in primary care is the Modified Checklist for Autism in Toddlers, Revised with Follow-Up (M-CHAT-R/F) (Robins et al. 2009). The tool, scoring instructions, and follow-up questions when parents report concerns on specific items can be obtained at no cost and in more than 30 languages, including several Spanish dialects. The M-CHAT-R/F includes 20 yes/no questions that parents can answer in less than 5 min; scoring by practice staff can be completed in 2 min. In many states, Medicaid and commercial insurers pay for screening on the same day as a well-child visit.

The M-CHAT- R/F is validated for use with children up to 3 years of age. Sometimes parents and child health providers have concerns about older children's social and communication skills and want to screen for ASD. The Childhood Autism Spectrum Test (CAST), previously called the Childhood Asperger Syndrome Test (Williams

et al. 2008), has been validated for use in pediatric primary care for children ages 4–11. It contains 37 items to be completed by parents and is available free of charge and in more than 15 languages.

When surveillance or screening shows concerns that suggest ASD, child health providers rarely perform next level diagnostic assessment but connect children and families to such services. Options for assessment are largely determined by resources available in the local area. Developmental pediatricians, behavioral health professionals, and neurologists may complete ASD and general developmental evaluations to verify a diagnosis. All states have early intervention (EI) programs under Part C of the Individuals with Disabilities Education Act. EI programs are required by the federal law to complete evaluations of children ages 0–3 with suspected developmental delay, including ASD, within 45 days of referral to the program. For children ages 3–5, Part B of the Individuals with Disabilities Education Act similarly requires timely evaluation upon referral. Part B and Part C programs also provide intervention services.

Although the therapeutic interventions for ASD will take place out of the medical home, there are several medical conditions that are associated with ASD and can be managed by pediatric primary care providers. These include epilepsy, gastrointestinal problems, and insomnia (Carbone et al. 2010b). Other conditions, such as motor impairments, sensory processing, and mental health disorders, which are also associated with ASD, may be identified in the medical home and require that the primary care provider connect the child to specialty services (Carbone et al. 2010b).

To meet the medical and nonmedical needs of children with ASD, the medical home actively connects families to services. Other providers in the community expect the pediatric primary care medical home to stay involved by taking on responsibility for acute health care, continued well-child visits, and physical and developmental monitoring. Because there is no cure for ASD, child health providers, who often provide care to children from birth to 21 years, can expect to be involved with a changing team of service

providers in the community as the child with ASD ages (Swiezy et al. 2008). These relationships span from early intervention and childcare providers for younger children to school personnel and employment services for older children. The medical home provider is often the single constant provider for families and children across the first 21 years of life and maintains a relationship with the family and comprehensive history that other service providers do not have. The role of the medical home in coordinating care over time is essential for ensuring best outcomes for children and optimal benefit from other services.

Family support is a critical component of care at the center of the medical home model that is often cited as lacking for children with ASD (Brachlow et al. 2007). In the same way that the medical home connects children and their families to subspecialty and intervention services, it also provides opportunities for families to connect with other families of children with ASD and with community family support opportunities. Many organizations exist for families of children with ASD, and the medical home maintains a list of those that are active in their specific community and contact information for them to give to parents. ASD parent support organizations, such as Autism Speaks, often have state and community level chapters, which offer a variety of services for families, including support groups, play groups, financial advising services, and recreational programs for children with ASD as well as general educational information. There is also a plethora of websites for families that provide information about ASD. Table 1 provides a list of some of the most popular sites. The medical home can have resources, such as this list, for families. There are

also several Facebook and other social media sites that promote a community among children with ASD and their families.

In addition to providing family support resources to families of children with ASD, the medical home also tailors its services to meet the special needs of these children. By the very nature of their condition, children with ASD may require special accommodations in the medical home, including longer visit times, shorter wait times, and accommodations for the performance of the physical exam (Volkmar et al. 2014). Many of the procedures that are expected when children visit the doctor are challenging for children with ASD, including undressing, answering questions, allowing physical contact, and cooperating for immunizations and lab testing. Linking children with ASD to dental and subspecialty services also requires special consideration, and the medical home can maintain a list of referral sources in the community that are known to care for children with special needs.

Beyond medical subspecialty and community services, pediatric medical home providers are responsible for connecting children with ASD when they turn 21 to adult health-care providers (Carbone et al. 2010b). This can present challenges for families and children as well as for the provider (Cheak-Zamora et al. 2013). A family is often reluctant to leave the health-care provider who has cared for their child since birth, and providers struggle with identifying adult medical providers who can care for the young adult with special needs and saying goodbye to longtime patients and their families. The AAP, American Academy of Family Physicians, and American College of Physicians have jointly endorsed a set of guidelines and activities for transitioning patients from pediatric to adult care (American Academy of Pediatrics 2015). The six core elements of transition, which the guidelines recommend should begin at age 12, include developing a practice transition policy for all children, monitoring and tracking transition activities for each patient, assessing transition readiness as part of well-child visits, collaborating with parents to develop a transition plan, preparing and implementing transfer of care with all necessary

Medical Home and ASD, Table 1 Websites for parents of children with ASD

Asperger Syndrome and High Functioning Autism Association www.ahany.org
Autism Highway www.autismhwy.com
Autism Research Institute www.autism.com
Autism Society www.autism-society.org
Autism Speaks www.autismspeaks.org
AutismWeb www.autismweb.com
Disability Scoop www.disabilityscoop.com

information, and following up with families to ensure that transition was successful. The AAP provides several resources to help the pediatric medical home implement each of these elements.

Cultural competency is another important component of the medical home that pertains to children with ASD. Children who live in families who are of lower socioeconomic status, or are members of racial and ethnic minority groups, experience later identification of their ASD (Ennis-Cole et al. 2013). This could result from their parents having different expectations for child development and responding differently to screening questionnaires or developmental surveillance questions that are largely validated in middle-class populations. Utilization of intervention services for nonwhite and low-income children with ASD is also lower than for other children with ASD, suggesting that families from diverse backgrounds may be more reliant on family- and faith-based organizations than professionals to meet their children's needs (Ennis-Cole et al. 2013).

All national organizations that certify practices as medical homes (American Academy of Family Physicians 2015), thereby enabling them to obtain enhanced payment from insurers, require that practitioners participate in ongoing quality improvement activities. Care related to ASD is an excellent area for practice-based quality improvement efforts. Practices can track and improve their implementation of universal ASD screening in well-child care visits. They can also set up practice systems to ensure that children for whom screening shows concerns receive assessments and are linked to intervention services if needed. The AAP's transition tool kit (American Academy of Pediatrics 2015) includes a self-assessment tool that practitioners can use to obtain a baseline measure and then periodically reuse to assess progress in implementing a transition program.

Future Directions

With increasingly high rates of diagnosis of ASD in young children, the medical home model is

undergoing several refinements to ensure better care for this population. Autism-specific medical home models that include parents within the practice to provide peer support and guide service enhancements have shown improved patient outcomes and parental satisfaction and fewer unmet needs (Golnik et al. 2012). Another innovative model of implementing medical home for children with ASD is the health home, which embeds primary care medical services within a behavioral health setting specifically tailored to meet the needs of children with ASD or intellectual disabilities (Fueyo et al. 2015).

At the same time, as primary care medical homes are transforming their services to address ASD, other community providers are also striving to meet the needs of this growing population of children. The Hospital for Special Care in New Britain, Connecticut, was the first ASD specialty service to earn recognition from the National Committee for Quality Assurance. Coordination of care with the pediatric medical home was one component of the recognition process.

There are several challenges to improving the ability of pediatric primary care sites to care for children with ASD within the medical home model. These include the following: lack of training about ASD, reimbursement for time spent in treating and managing children with ASD, and lack of care coordination (Golnik et al. 2009). Training tailored to improve screening of all children is effective in supporting early detection of children with ASD at the population level (Honigfeld et al. 2012), and pediatricians are also eager to receive more training on the ongoing care for children with ASD (Golnik et al. 2009). Ideally such training would begin in medical school and be reinforced with clinical experience in pediatric residency programs, available for the duration of professional careers through continuing medical education and required for pediatric maintenance of certification. Multidisciplinary training opportunities that include health, education, behavioral health, and other service providers would enhance the common understanding of optimal services for children with ASD among the professionals who serve them.

Payment models beyond traditional fee for service, which are increasingly popular in public and private insurance plans, can also facilitate improved medical home care for children with ASD. Bundled payments that provide reimbursement for all of the care for chronic conditions would allow child health providers to spend adequate time addressing the specific challenges in examining children with ASD as well as family support needs. Insurance plans that pay a per member/per month or enhanced capitation payment can support the provision of the extensive care coordination and team participation that medical home providers need to optimally support children with ASD and their families.

In sum, the medical home model is ideal for the care of children with ASD. It can ensure early identification, linkage to diagnostic and intervention services, and ongoing health maintenance to support children's participation in therapeutic services. Enhanced training, payment for time and care coordination, and opportunities to support families can all contribute to increasing the contribution of the pediatric primary care medical home to the health and well-being of children with ASD.

See Also

► Dental Care

References and Reading

- American Academy of Pediatrics Task Force on the Definition of Medical Home. (1992). The medical home. *Pediatrics*, 90, 774.
- American Academy of Pediatrics. (2004) Policy Statement. The medical home. *Pediatrics*, 113, 1545–1547.
- American Academy of Pediatrics Council on Children With Disabilities, Section on Developmental Behavioral Pediatrics, Bright Futures Steering Committee and Medical Home Initiatives for Children With Special Needs Project Advisory Committee. (2006). Identifying infants and young children with developmental disorders in the medical home: An algorithm for developmental surveillance and screening. *Pediatrics*, 118(1), 405–420.
- American Academy of Pediatrics Autism Expert Panel. (2013). *Autism: Caring for children with autism spectrum disorders: A resource toolkit for clinicians* (2nd ed.). Elk Grove: American Academy of Pediatrics.
- American Academy of Pediatrics. (2015). Bright futures, recommendations for Preventive Pediatric Health Care. Retrieved 30 Jan 2017 from https://www.aap.org/en-us/Documents/periodicity_schedule.pdf.
- American Academy of Family Physicians. (2015). PCMH incentive, recognition, and accreditation programs. Retrieved 30 Jan 2017 from <http://www.aafp.org/practice-management/transformation/pcmh/recognition.html>.
- American Academy of Pediatrics Council on Children With Disabilities. (2015). Clinical report: Transitions. Retrieved 2 Feb 2017 from <https://www.aap.org/en-us/about-the-aap/Committees-Councils-Sections/Council-on-Children-with-Disabilities/Pages/Policy-Resources.aspx>.
- Brachlow, A. E., Ness, K. K., McPheeters, M. L., & Gurney, J. G. (2007). Comparison of indicators for a primary care medical home between children with autism or asthma and other special health care needs: National survey of children's health. *Archives of Pediatrics and Adolescent Medicine*, 161(4), 399–405.
- Carbone, P. S., Behl, D. D., Azor, V., & Murphy, N. A. (2010a). The medical home for children with autism spectrum disorders: Parent and pediatrician perspectives. *Journal of Autism and Developmental Disorders*, 40(3), 317–324.
- Carbone, P. S., Farley, M., & Davis, T. (2010b). Primary care for children with autism. *American Family Physician*, 81(4), 453–460.
- Cheak-Zamora, N. C., & Farmer, J. E. (2015). The impact of the medical home on access to care for children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 45(3), 636–644.
- Cheak-Zamora, N. C., Yang, S., Farmer, J. E., & Clark, K. (2013). Disparities in transition planning for youth with autism spectrum disorder. *Pediatrics*, 131(3), 447–454.
- Department of Health and Human Services, Centers for Disease Control, National Center for Health Statistics: National Health Interview Survey. (2014). Retrieved 30 Jan 2017 from https://ftp.cdc.gov/pub/Health_Statistics/NCHS/NHIS/SHS/2014_SHS_Table_C-8.pdf.
- Ennis-Cole, D., Durodoye, B. A., & Harris, H. L. (2013). The impact of culture on autism diagnosis and treatment: considerations for counselors and other professionals. *The Family Journal: Counseling and Therapy for Couples and Families*, 21(3), 279–287.
- Farmer, J. E., Clark, M. J., Mayfield, W. A., Cheak-Zamora, N., Marvin, A. R., Law, J. K., & Law, P. A. (2014). The relationship between the medical home and unmet needs for children with autism spectrum disorders. *Maternal and Child Health Journal*, 18(3), 672–680.
- Fueyo, M., Caldwell, T., Mattern, S. B., Zahid, J., & Foley, T. (2015). The health home: A service delivery model for autism and intellectual disability. *Psychiatric Services*, 66, 1135–1137.
- Golnik, A., Ireland, M., & Borowsky, I. W. (2009). Medical homes for children with autism: a physician survey. *Pediatrics*, 123(3), 966–971.
- Golnik, A., Scal, P., Wey, A., & Gaillard, P. (2012). Autism-specific primary care medical home

- intervention. *Journal of Autism and Developmental Disorders.*, 42(6), 1087–1093.
- Holt, J., Esquivel, M., & Pariseau, C. (2010). *Medical home competencies for LEND trainees*. Association of University Centers on Disabilities. Retrieved 20 May 2012 from <http://www.aucd.org/template/page.cfm?id=633>.
- Homer, C., Klatka, K., Romm, D., Kuhlthau, K., Bloom, S., Newacheck, P., et al. (2008). A review of the evidence for the medical home for children with special health care needs. *Pediatrics*, 122, e922–e937.
- Honigfeld, L., Chandhok, L., & Spiegelman, K. (2012). Engaging pediatricians in developmental screening: the effectiveness of academic detailing. *Journal of Autism and Developmental Disorders*, 42(6), 1175–1182.
- Hyman, S., & Johnson, J. (2012). Autism and pediatric practice: toward a medical home. *Journal of Autism and Developmental Disorders*, 42(6), 1156–1164.
- Ming, S., Hashim, A., Fleishman, S., West, T. Kang, N., Chen, X., & Zimmerman-Bier, B. (2011). Access to specialty care in autism spectrum disorders—a pilot study of referral source. Retrieved 1 Feb 2017 from <http://bmchealthservres.biomedcentral.com/articles/10.1186/1472-6963-11-99>.
- Nielsen, M., Buelt, L., Patel, K., & Nichols, L.. (2016). The patient-centered medical home's impact on cost and quality, review of evidence, 2014–2015.
- Robins, D., Fein, D., & Barton, M. (2009). Modified checklist for autism in children, revised with follow up. Retrieved 31 Jan 2017 from https://www.m-chat.org/_references/mchatdotorg.pdf.
- Swiezy, N., Stuart, S., & Korzekwa, P. (2008). Bridging for success in autism: Training and collaboration across medical, educational and community systems. *Child Adolesc Psychiatric Clinics of North America*, 17, 907–922.
- Van Vleet, A., & Paradise, J. (2014). *The state innovation models (SIM) program: An overview*. Henry K. Kaiser Family Foundation. Retrieved 30 Jan 2017 from <http://kff.org/medicaid/fact-sheet/the-state-innovation-models-sim-program-an-overview/>.
- Volkmar, F. R., Rowberry, J., DeVinck-Baroody, O., Gupta, A. R., Leung, J., Meyers, J., Vaswani, N., & Weisner, L. A. (2014). Medical care in autism and related conditions. In F. Volkmar, S. Rogers, R. Paul, & K. Pelphery (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 532–555). Hoboken: Wiley.
- Williams, J. G., Allison, C., Scott, F., Stott, C., Bolton, P., Baron-Cohen, S., & Brayne, C. (2008). The childhood Asperger syndrome test (CAST): Test-retest reliability. *Autism*, 10(4), 415–427.

Medulla Oblongata

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Synonyms

Medulla

Definition

The *medulla oblongata* is the lowest of the three structures of the brain stem (the pons and the mid-brain are the other two), and as such, it participates in the brain stem's important role in serving as the modulator and integrator of any information exchange between the brain and the proprioceptive and exteroceptive systems of the human body. In the light of the fact that ASD is becoming recognized increasingly as a problem of the nervous system integrity involving many functions of the brain, one might predict that the brain stem, in particular medulla oblongata, would have a significant share in the neuropathology of ASD. In fact, earlier postmortem and MRI structural imaging studies on the brain stem found all three structures significantly smaller in individuals with autism (Gaffney et al. 1988; Hashimoto et al. 1993). These studies, however, suffered from the presence of confounding variables and were contradicted by concurrent and later studies, even though more recent volumetric studies of both white and gray matter continue to implicate the brain stem (Goldberg et al. 1999; Rodier 2002).

Neurochemical abnormalities in individuals with ASD have served as another link between medulla oblongata and autism. Serotonin is a neurotransmitter that is synthesized from tryptophan by neurons in the raphe nuclei of the brain stem reticular formation, which extends along all three structures of the brain stem. Autistic individuals have been found to have elevated whole-blood serotonin 5-HT levels and decreased tryptophan

Medulla

► Medulla Oblongata

retention, both indicating a decrease in brain serotonin binding (Chugani et al. 1997; Carter et al. 2011; Kaluzna-Czaplinska et al. 2010).

See Also

- [Midbrain](#)
- [Serotonin](#)

References and Reading

- Carter, M. D., Shah, C. R., Muller, C. L., Crawley, J. N., Carneiro, A. M., & Veenstra-VanderWeele, J. (2011). The impact of serotonergic stimulation on reelin and glutamate decarboxylase gene expression in adult female rats. *Bratislavské Lekárske Listy*, 11(2), 58–62.
- Chugani, D. C., Muzik, O., Rothermel, R., Behen, M., Chakraborty, P., Mangner, T., et al. (1997). Altered serotonin synthesis in the dentatohalamocortical pathway of autistic boys. *Annals of Neurology*, 42, 666–669.
- Gaffney, G. R., Kuperman, S., Tsai, L., & Minchin, S. (1988). Morphological evidence for brainstem involvement in infantile autism. *Biological Psychiatry*, 24, 578–586.
- Goldberg, J. M. B., Szatmari, P., & Nahmias, C. (1999). Imaging of autism: Lessons from the past. *Canadian Journal of Psychiatry*, 44, 793–801.
- Hashimoto, T., Tayama, M., Miyazaki, M. K., Shimakawa, S., Yonada, Y., et al. (1993). Brainstem involvement in high functioning autistic children. *Acta Neurologica Scandinavica*, 88, 123–128.
- Kaluzna-Czaplinska, J., Michalska, M., & Rynkowski, J. (2010). Determination of tryptophan in urine of autistic and healthy children by gas chromatography/mass spectrometry. *Medical Science Monitor*, 16(10), CR488–CR492.
- Rodier, P. M. (2002). Converging evidence for brain stem injury in autism. *Development and Psychopathology*, 14(3), 537–557.

Mellaril

- [Thioridazine](#)

Mellaril-S

- [Thioridazine](#)

Memantine

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Definition

Memantine: Memantine is a selective blocker of a specific glutamate receptor in the brain. It is approved for the treatment of dementia. It is also used in combination with the cholinesterase inhibitors such as donepezil. Memantine is presumed to have beneficial effects on Alzheimer's dementia because of its blockade of this specific glutamate receptor. It has not been well studied in children or adults with autism.

References and Reading

- Martin, A., Scahil, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford.

Memory

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Synonyms

[Recollection](#); [Remembrance](#)

Definition

The multifaceted process of encoding, storage, and retrieving knowledge of things that we have experienced, imagined, and learned. Memory is often measured using tasks associated with recall, retrieval, or recognition. There are a wide range of memory abilities in individuals with autism spectrum disorder (ASD), with some showing significant impairments across multiple types of memory, and others showing “savant” skills. Most commonly, individuals with ASD have a profile of strengths and weaknesses that vary across specific subtypes of memory.

See Also

- [Declarative Memory](#)
- [Episodic Memory](#)
- [Explicit Memory](#)
- [Free Recall](#)
- [Memory Assessment](#)
- [Memory Development](#)
- [Recognition Memory](#)
- [Retrieval of Information](#)
- [Rote Memory](#)
- [Semantic Memory](#)
- [Short-Term Memory](#)

References and Reading

- Atkinson, R., & Shiffrin, R. (1968). Human memory: A proposed system and its control processes. In K. Spence & J. Spence (Eds.), *The psychology of learning and motivation: Advances in research and theory* (Vol. 2). New York: Academic.
- Boucher, J., & Bowler, D. (2008). *Memory in autism*. Cambridge: Cambridge University Press.
- Mottron, L., Belleville, S., Stip, E., & Morasse, K. (1998). Atypical memory performance in an autistic savant. *Memory*, 6, 593–607.
- Tulving, E. (1985). How many memory systems are there? *American Psychologist*, 40, 385–398.
- Williams, D. L., Goldstein, G., & Minshew, N. J. (2006). The profile of memory function in children with autism. *Neuropsychology*, 20, 21–29.

Memory Assessment

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Definition

The assessment of memory in ASD is carried out by researchers attempting to delineate the pattern of strengths and weaknesses in memory and learning in ASD generally and to understand the psychological, neurobiological, and etiological correlates of the atypical memory profiles delineated. Memory assessment may also be carried out by practitioners to delineate and understand atypical patterns of learning and memory in individuals with ASD with the aim of developing effective intervention strategies. Very little assessment of the neurobiological and etiological correlates of atypical memory and learning in ASD has been carried out, and what follows relates exclusively to the psychological assessment of memory.

Current Knowledge

Memory is involved in the acquisition, storage, retrieval, and everyday use of associations, habits, skills, perceptual images, words, facts, and personal experiences. It is therefore a critical component of all kinds of learning. People with ASD across the spectrum show certain atypical patterns of memory and learning, including some outstanding strengths as well as certain limitations. In lower functioning individuals with ASD, moderate or severe memory limitations contribute to clinically significant learning disabilities. At the same time, memory strengths in both lower and higher functioning individuals may be utilized in compensatory learning. It is therefore practically as well as theoretically important to characterize the pattern of memory strengths and weaknesses in people with ASD and to understand how and why uneven memory profiles occur, both generally and individually. This in turn requires that

appropriate investigative procedures are developed and used by researchers and that appropriate assessment tools are available to practitioners. A representative selection of experimental research procedures and clinical assessment tools is outlined below.

Experimental Assessment Procedures

The Assessment of Implicit, or Non-declarative, Learning

Many associations, habits, skills, and perceptual images are acquired “implicitly,” that is to say, without conscious awareness or control of what is being learned, although there may be an intention to learn. Moreover, what has been learned implicitly is not generally available to report – hence the term “non-declarative.” For example, babies work hard at learning to walk, oblivious of the increasingly well-entrenched changes in brain activity and muscle coordination that are occurring every time they struggle to their feet and stagger a step or two. Similarly, they unconsciously learn the sound system and grammatical rules of their native language without awareness of the rules and regularities they are acquiring. Because implicit learning takes many forms, it can be experimentally assessed in many ways. A few examples, all of which have been used in investigative studies of people with ASD, are given below:

- **Conditioning.** When used with vulnerable people, a typical conditioning test might consist of shining a dim light into an individual’s eye just before delivering a small puff of air onto the eyeball (as in a standard test used by opticians) which produces a reflexive blink, and measuring how many repetitions of this procedure are needed before the dim light elicits a reflexive blink in the absence of a puff of air.
- **Category Formation.** Basic-level categories relating to common objects, actions, or qualities are largely acquired implicitly from the first weeks of life onward. There are various theoretical models of how this is achieved. One well-authenticated model involves the (unconscious) acquisition of a prototype (e.g.,

the most typical form of a “dog” or a “tree”). A commonly used category formation task involves showing the examinee pictures of an imaginary creature (e.g., a “moodle”) defined by a particular set of probabilistic features (e.g., a longish and somewhat crooked nose, a shortish tail) and contrasting moodles with “non-moodles” (which in some ways resemble moodles, but which lack their prototypical features) by sorting into two piles. Examinees are then asked to try to sort a different set of moodles from the non-moodles with correction from the tester. A critical measure of category formation is whether or not representations of a prototypical moodle are correctly sorted.

- **Sequence Learning** (as, e.g., in acquiring motor skills, habitual routines, or other forms of “procedural” learning). A serial response task is commonly used to assess implicit sequence learning. In this task, a stimulus (e.g., an asterisk) appears in one of several locations on a computer screen in a repeating sequence, and participants respond to each appearance by pressing a corresponding key. Learning is demonstrated by a decrease in response time on trials when the locations follow the sequence and by an increase in response time on trials when locations do not follow the sequence.

The Assessment of Explicit, or Declarative, Memory and Learning

Words and their meanings, factual knowledge, and the content of personal experiences are at least potentially available to conscious awareness during learning and potentially available to subsequent report, hence the term “declarative.” The content of declarative memory may broadly be described as “things that we know that we know,” even if we need some prompting to recover the memory.

Whereas experimental tests of implicit learning in people with ASD are currently mainly directed toward answering the question “*Can* people with ASD do this?”, the peaks and troughs of explicit memory in people with ASD are already quite well established (Boucher et al. [in press](#)). In brief, the most able individuals with ASD have

good vocabularies (even though words may not always be used in entirely typical ways); they have excellent ability to acquire factual knowledge, especially relating to topics of special interest; but they have some difficulties in recalling personal experiences, especially the more subjective elements, such as how they themselves were feeling at the time. Less able individuals with ASD may have across the board difficulties with declarative memory. Because profiles of declarative memory abilities across the spectrum are relatively well established, experimental assessments of explicit learning are mainly directed toward answering questions concerning *why* the patterns of memory peaks and troughs occur and differ across the spectrum.

Declarative memory tests may be designed to investigate critical differences in the *kinds of things* examinees can or cannot easily remember. Critical differences that have been investigated in people with ASD include:

- The complexity of the material to be remembered (e.g., comparing the ability to recall lists of unrelated words, as opposed to related words, sentences, or stories; and pictures of single objects, as opposed to scenes)
- The social connotations of the material (e.g., comparing the ability to recognize pictures of faces as opposed to buildings)
- The extent to which the examinees themselves played a role in the action or event to be remembered (e.g., comparing memory for actions they themselves have carried out as opposed to those the tester carried out)
- The modality of the material to be remembered (e.g., comparing memory for spoken as opposed to written words)

Other tests are designed to investigate differences in the *learning and recalling conditions* that may contribute to memory peaks as opposed to memory troughs. For example:

- “Learning over trials” as opposed to “single trial learning.”
- “Intentional” as opposed to “incidental” learning, that is, tests in which the examinee is

instructed to try to remember the pictures they will be shown or the gist of the story they will hear as compared with tests in which they are simply shown some pictures or read a story and then unexpectedly tested for memory of what they have seen or heard.

- Computer-based as opposed to person-led learning.
- Recognition tests (which of these pictures did you see just now?) as opposed to free recall tests (what objects did you see in the pictures you saw just now?) and “free recall” as compared to “cued recall” tests (one of the pictures was of a kind of fruit/began with the sound “b”).
- Immediate as opposed to delayed memory tests.

Yet another group of experimental memory tests focuses on the *processes* that may underlie memory peaks as opposed to memory troughs in ASD. For example:

- Tests of encoding biases, such as tendencies to encode (register into memory) details of a scene rather than the scene as a whole; or the sound rather than the meaning of heard words
- Tests of retrieval processes (i.e., the processes by which what has been unconsciously learned or consciously memorized becomes accessible for use) and especially difficulties that people with ASD may have in effortful and strategic forms of memory search where few or no cues or prompts are available as opposed to associatively triggered memories or recall in response to informative cues or leading questions

The Assessment of Working Memory

“Working memory” involves the ability to maintain information in short-term memory while simultaneously operating on it in some way. Maintaining information in short-term memory is not by itself a major problem for people with ASD, as is evident from their good rote memory ability. However, the simultaneous maintenance and manipulation of information involve executive control and may be problematic. Experimental assessment of working memory in people with ASD is at the stage of delineating the kinds of

working memory tasks on which people with ASD reliably succeed as compared with those they find significantly difficult.

Some experimental assessments of working memory that have been used with people with ASD are described below:

- **Backward Digit Span.** In this task, the examinee must register and maintain in short-term memory a serially presented list of, for example, seven digits, and subsequently report the sequence in reverse order.
- **“N-back Tasks.”** In this kind of task, the examinee might be asked to name individual letters successively presented on a screen and to press a response key if the letter on the screen is the same as the last-but-one letter presented or the same as the last-but-two letter presented.
- **Self-ordered Pointing Tasks.** In this type of task, the examinee might be shown a set of line drawings of, for example, nine everyday objects located on a 3×3 grid. The set is reproduced nine times with no object appearing in the same location twice. The examinee is shown each reproduction in turn and instructed to point to a different item each time, requiring them to hold in mind a cumulative list of items already responded to.
- **Prospective Memory Tasks.** “Prospective memory” involves “remembering to remember” to do something, for example, remembering to take swimming things to school on a Wednesday, to post a birthday card to grandma tomorrow, and to take the saucepan off the hob at a certain time. Experimental tests of prospective memory generally involve giving an instruction such as “remember to press the red button whenever you see an animal on the screen/once a minute” and then giving the examinee some other task to be getting on with, such as sorting pictures on the screen by their color. The measure of prospective memory ability is the number of times the original instruction is acted on, either in response to the “event cue” (an animal occurring on the screen) or in response to the “time” cue (an internal estimate of 1 min).

Clinical Assessments

Most clinical assessments of memory have been developed for use with adults with medical conditions known to be associated with memory impairments or anomalies, such as head injury, stroke, Alzheimer’s disease, or posttraumatic stress disorder. Some of the best-known of these clinical assessments are designed to test a broad range of memory abilities. Others have been designed to assess in greater detail some specific form of memory. Versions of some assessment procedures developed for adults have subsequently been developed for use with children. Other procedures have been developed specifically for use with children and are not suitable for use with adults. A representative selection of clinical assessment procedures, all of which have been used for the assessment of memory in people with ASD, is listed below:

- *The Wechsler Memory Scale, 3rd edition* (Wechsler 1997). This widely used test consists of subtests assessing immediate auditory and visual-spatial memory and subtests assessing story recall, verbal paired-associate learning, face recognition, and recall of the content of pictures of family scenes, this latter group of subtests being assessed in both immediate and delayed recall conditions.
- *The Rivermead Behavioural Memory Test, 3rd edition* (Wilson et al. 2008). This test is designed to assess memory using materials and situations resembling everyday situations. So, for example, prospective memory is assessed, as is memory for a route, and learning to associate a name with a face. There is a children’s version of this test: *Rivermead Behavioural Memory Test for Children* (Wilson et al. 1991).
- *The Doors and People Test of Visual and Verbal Recognition and Recall* (Baddeley et al. 1994). This test, as its name indicates, is designed to assess person-related as compared to object-related memory, visual as opposed to verbal memory, and recognition as opposed to recall. It can be used with children as well as adults.

- *The California Verbal Learning Test-II* (Delis et al. 2000a). This test assesses memory for word lists in a variety of conditions, including comparisons of single trial versus multiple trial learning, immediate versus delayed recall, free recall versus recognition versus cued recall, and “old list” versus “new list” learning. There is a children’s version of this test: *The California Verbal Learning Test for Children-II* (Delis et al. 2000b).
- *The Autobiographical Memory Interview* (Kopelman et al. 1990). This test uses a structured interview format to probe the ability to recall facts and specific incidents from the individual’s past life, from childhood to recent time.
- *The Benton Face Recognition Test* (Benton et al. 1994). In this test, each test item consists of a black and white photograph of a face set above a choice of six other black and white photographs of faces in a variety of orientations, and examinees must indicate whether one or more of these are of the person in the target picture.
- *The Rey-Osterrieth Complex Figure Copying and Memory Test* (Osterrieth 1944). As the name of this test suggests, examinees are first asked to copy a complex abstract figure. This is then removed, and the examinee is immediately asked to draw the figure again, this time from memory. Following a delay, they are again asked to draw the figure from memory.

Tests developed specifically for use with children include:

- *The Wide Range Assessment of Memory and Learning* (Sheslow and Adams 1990). The content of this test broadly corresponds to the content of the adult-targeted Wechsler Memory Scale (see above).
- *The Children’s Memory Scale* (Cohen 1997). The content of this test also corresponds broadly with that of the Wechsler Memory Scale. More specifically, it is described as assessing verbal and visual memory; short delay and long delay memory; recall, recognition, and working memory; and the role of attentional factors in memory.

- *The Children’s Nonword Repetition Test* (Gathercole and Baddeley 1996). This test of immediate short-term memory assesses the ability to repeat accurately a series of made-up words of increasing length.

The above list represents only a small selection of clinical assessment procedures, and many more are available and have potential value for better understanding the nature and causes of uneven memory abilities in ASD. A comprehensive list of assessment procedures can be found in Lezak et al. (2004).

See Also

- [Declarative Memory](#)
- [Episodic Memory](#)
- [Explicit Memory](#)
- [Free Recall](#)
- [Memory](#)
- [Memory Development](#)
- [Recognition](#)
- [Rote Memory](#)
- [Semantic Memory](#)
- [Short-Term Memory](#)

References and Reading

- Baddeley, A. D., Emslie, H., & Nimmo-Smith, I. (1994). *The doors and people test of visual and verbal recognition and recall*. Bury St. Edmunds: Thames Valley Test Company/Pearson Assessments.
- Benton, A. L., Sivan, A., Hamsher, K., Varney, N., & Spreen, O. (1994). *Contributions to neuropsychological assessment: A clinical manual*. New York: Oxford University Press.
- Boucher, J., & Bowler, D. M. (2008). *Memory in autism*. Cambridge, MA: Cambridge University Press.
- Boucher, J., Mayes, A., & Bigham, S. (in press). Memory in autistic spectrum disorder. *Psychological Bulletin*.
- Cohen, M. (1997). *The children’s memory scale*. San Antonio: The Psychological Corporation/Pearson Assessments.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000a). *The California verbal learning test-II*. San Antonio: Psychological Corporation/Pearson Assessments.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000b). *The California verbal learning test for*

- children -II*. San Antonio: Psychological Corporation/Pearson Assessments.
- Gathercole, S. E., & Baddeley, A. D. (1996). *The children's test of nonword repetition*. San Diego: The Psychological Corporation/Pearson Assessments.
- Kopelman, M., Wilson, B., & Baddeley, A. D. (1990). *The autobiographical memory interview*. Bury St. Edmunds: Thames Valley Test Company/Pearson Assessments.
- Lezak, M. D., Howieson, D., & Loring, D. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
- Osterrieth, P. A. (1944). The Rey-Osterrieth complex figure copying and memory test. *Archives de Psychologie*, 30, 286–356.
- Sheslow, D., & Adams, W. (1990). *The wide range assessment of memory and learning*. Wilmington: Jastak Associates.
- Wechsler, D. (1997). *The Wechsler memory scale* (3rd ed.). San Antonio: The Psychological Corporation/Pearson Assessments.
- Wilson, B. A., Ivani-Chalian, R., & Aldrich, F. (1991). *Rivermead behavioural memory test for children*. Bury St. Edmunds: Thames Valley Test Company/Pearson Assessments.
- Wilson, B., Greenfield, E., Clare, L., Baddeley, A. D., Cockburn, J., Watson, P., et al. (2008). *The rivermead behavioural memory test* (3rd ed.). Bury St. Edmunds: Thames Valley Test Company/Pearson Assessments.

Memory Development

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Definition

The concept of *Memory Development* may appear to require few introductions, but closer inspection reveals certain complexities that are important to note. For one, the notion of development must not be misunderstood as referring exclusively to the changes in behavior and cognition that accompany infancy and early childhood. Although this early stage of development is of key interest, important changes also take place later in life, and since adults come to be the way they are through their developmental history, they must not be overlooked as a rich source of information about developmental processes. Just as the

archeologist is able to infer what events took place in a distant past from the study of ancient treasures, so the developmental psychologist can make certain inferences about the principles of development by taking a close look at adult behavior. What also renders the concept of memory development complicated is the fact that “memory” is far from the transparent phenomenon that our common usage of the term may have us believe. Whether we forget the name of a person we have just been introduced to, the capital of a country we once visited, the location of our car in a car park, or a certain sequence of key strokes on the piano, we are always quick to blame our *memory* for our failures. Yet, decades of research have shown that such instances of forgetting are not the result of the failures of a single memory system or process but of the imperfections of a number of such systems and processes that each contributes in specific ways to our ability to learn from the past. It is beyond the scope of this entry to provide a summary of the various approaches that have been taken to carving the nature of memory at its joints (for overviews relevant to the study of autism, see Gardiner 2008 and Mottron et al. 2008), but two distinctions require a brief comment. The first is the division between *procedural* and *declarative* memory. Procedural memory describes memory for sequences of motor behaviors that can be learned and expressed without the necessary involvement of conscious awareness. Declarative memory, by contrast, describes memory for information that is learned and retrieved under the influence of conscious awareness. This entry will only concern issues relating to declarative memory development since the vast majority of research in autism has addressed this domain. The second distinction is based on the observation by Tulving (1985) that not all of our declarative memories are associated with the same kind of conscious experience. The memory of the name of our childhood primary school, for instance, is qualitatively different from that of our first day there. The former is associated with a *noetic* sense of awareness (knowing) that is relatively void of a feeling of personal belonging. It characterizes retrieval from our *semantic memory system* that

constitutes a store of impersonal facts and knowledge about the world. *Autonoetic* awareness (remembering), by contrast, accompanies retrieval from our *episodic memory system*, which encompasses all those memories that revolve around our “self” in the past. These distinctions are critical when considering how memory develops since the lack of language during the earliest stages of development makes it impossible for infants to “declare” what they remember and how they remember it. As a result, the study of memory development in very young children is complicated, and when language development follows an atypical trajectory, such as in the case of autism, matters become even more intricate. It is thus perhaps not surprising that the study of memory development in autism has adopted the archeological route of focusing on adults as a source of information. The patterning of memory strengths and weaknesses in older groups has provided important clues about what role disturbances in memory development may play in the origin and/or maintenance of the clinically defining manifestations of autism, and together with advances in the methods for studying how memory matures during the first few years of life, these clues are beginning to open promising new research avenues for the near future.

Historical Background

Memory research in autism has a long history that begins with the seminal work of Beate Hermelin and Neil O'Connor in the 1960s (Hermelin and O'Connor 1970). At that time, cognitive approaches and associated computer analogies of the mind/brain relation dominated the field of psychology, and autism was finally beginning to be accepted as a neurodevelopmental disorder rather than the consequence of pathological parenting. In the context of these developments, Hermelin and O'Connor set about to determine how the autistic mind *processed* and *stored* information, and they soon made a discovery that remains valid up to the present day. In a series of experiments, they demonstrated that children with autism did not benefit from “meaning” when

trying to remember sets of stimuli. For instance, whereas typically developed children would remember grammatically correct sentences far better than random sequences of words, children with autism would remember both sets of stimuli equally well. Similarly, children with autism did not demonstrate a mnemonic advantage for meaningfully related as compared to unrelated words or pictures, while children without autism reliably did. Together these findings led Hermelin and O'Connor (1970) to conclude that autism was characterized by an impairment in processing information meaningfully and numerous subsequent studies have lent support to this suggestion. Another early discovery was the observation that tests of free recall pose disproportionate difficulties for individuals with autism. Free recall relies almost exclusively on the operations of the episodic memory system and requires the retrieval of previously learned information with only minimal support (e.g., “Tell me all the words you can remember”). Boucher and Warrington (1976) showed that children with autism were significantly impaired in recalling namable pictures, written words, and spoken words despite performing similarly to comparison children when retrieval cues were provided at test (e.g., “Can you remember something you sit on” for the item “stool”). Importantly, this pattern highlighted a parallel with patients who suffered damage to an area of the brain known as the hippocampus, leading Boucher and colleagues to conclude that this medial temporal lobe (MTL) structure was likely to play a critical role in the neurodevelopmental origins of autism (see Mayes and Boucher 2008 for a recent review of the relevant evidence).

As ahead of their time as these early pioneers were, their insights and discoveries were soon to fall by the wayside in favor of other conceptual developments in the field. The discovery that children with autism lacked a “theory of mind” (Baron-Cohen et al. 1985) began to dominate the search for the developmental origins of the disorder during the 1980s and the formulation of the weak central coherence (WCC) theory around the same time soon appeared to provide an elegant explanation for the findings by Hermelin and

O'Connor (1970) in terms of a detail-focused rather than a holistic and meaning-focused processing style in autism (e.g., Shah and Frith 1993). In addition, it was thought that the kind of memory difficulties experienced by individuals with autism could not possibly contribute causally to the development of the disorder since the memory processes relevant to these difficulties (i.e., primarily episodic processes) were widely thought not to mature until the age of 3–4 years in the course of typical development. In other words, there was a widespread conviction that memory difficulties emerged after the clinically defining symptoms of the disorder. For the next 20 years, therefore, the dominant topic of discussion was the nature of the relationship between the detail-focused processing style of individuals with autism and their difficulty in understanding the minds of others. Research on memory merely continued at the fringes of scientific awareness. Today, however, developments in both the autism memory literature and the mainstream memory development literature are beginning to draw attention to the possibility that atypical learning and memory processes may play a much more important role in the development of autism than originally thought.

Current Knowledge

In order to understand why interest in the learning and memory difficulties of individuals with autism is growing, it is necessary to provide an overview of the developments in two distinct literatures – that concerning the patterning of memory strengths and weaknesses in autism and that concerning how memory typically develops during the first years of life.

Memory Strengths and Weaknesses in Autism

As already alluded to, our current knowledge about memory function in autism stems primarily from studies of children, adolescents, and adults who have sufficient language and general intellectual abilities to cope with the demands of declarative memory tasks. Work with these

relatively high-functioning individuals generally confirms the original conclusions by Hermelin and O'Connor (1970) and Boucher and colleagues (e.g., Boucher and Warrington 1976) that individuals with autism experience difficulties in drawing on meaning to facilitate memory and that their performance on free recall measures is disproportionately attenuated compared to supported test procedures such as cued recall and recognition. Close scrutiny of these phenomena in recent years, however, has led to a more refined understanding of why a difficulty in drawing on meaning should coincide with disproportionate difficulties in free recall, and three sets of observations have proven particularly informative in this context. First, it is now widely accepted that autism is characterized by impairments in episodic memory that go beyond a disproportionate difficulty with free recall procedures. For instance, when individuals with autism are asked to describe the quality of their memories on tests of recognition memory (e.g., “Have you seen X before?”), they are less likely than comparison participants to recollect specific details about the study episode (e.g., Bowler et al. 2007). Individuals with autism also struggle to recall contextual details such as when, where, and how they learned something when they are directly tested for such details (e.g., Bowler et al. 2004; Poirier et al. 2011), and they often adopt a third-person rather than a first-person perspective when recalling autobiographical events (Lind and Bowler 2010). The second influential discovery is that, contrary to the original conclusions by Hermelin and O'Connor (1970), individuals with autism appear to draw on “meaning” to facilitate memory under several circumstances. For instance, conceptual relations between words (e.g., crop-grain) have been shown to promote memory in autism as much as in comparison groups when recognition procedures (“Did you see the word apple earlier?”) rather than free recall procedures (“What words did you see earlier?”) are used (e.g., Bowler et al. 2008) or when attention is drawn to such relations during study (Gaigg et al. 2008; Mottron et al. 2001). In addition, individuals with autism are as likely as comparison participants to experience false memories of words that

are meaningfully related to a set of studied words (e.g., night, pillow, tired, bed, dream. . . for a false memory of *sleep*) (Bowler et al. 2000; Hillier et al. 2007), which suggests not only that the concept of “meaning” is too broad to capture what individuals with autism do not process effectively in the service of memory but also that memory difficulties in this disorder are unlikely to be a reflection of a local processing style as proposed by the WCC theory. The third development that has contributed to our understanding of the patterning of memory strengths and weaknesses in autism is the complex information processing framework developed by Minshew and colleagues (see Williams et al. 2008), which notes that memory difficulties in this disorder become more pronounced as tasks become informationally more complex. For instance, Minshew and Goldstein (2001) found that individuals with autism demonstrate a progressively more pronounced memory difficulty for materials that increase in semantic and syntactic complexity from sequences of letters to sequences of words and sentences. Although these observations are consistent with Hermelin and O'Connor’s original observations, Minshew and Goldstein (2001) also found that when asked to learn the path through a maze, individuals with autism compared to those without performed progressively worse as the number of choice points in the maze increased, and this finding is less amenable to an explanation in terms of “meaning.” Taken together, therefore, these findings favor the view that it is “complexity” rather than “meaning” that lies at the root of the memory difficulties in autism, which is also consistent with neuropsychological evidence implicating the frontal lobes in autism (see Williams et al. 2008 for a review). But what is the metric for complexity? How can it be measured and defined, and how does the concept of complexity account for the episodic memory difficulties outlined above? The answer to these questions lies in the mainstream memory literature where a distinction between *item-specific* and *relational* processes (e.g., Hunt and Einstein 1981) will lead back to a developmental consideration of the role memory abnormalities may play in the ontogenesis of autism.

The Mainstream Literature

Around the time of the first systematic investigations of memory in autism, Hunt and Einstein (1981) proposed that the efficacy of memory is principally dependent on the processing of two types of information – item-specific information and relational information. *Item-specific* information is information specific to the individual elements of a to-be-remembered set of materials such as the fact that a banana is yellow, that it is sweet, that it is soft textured, and that it belongs to the category of fruit. *Relational* information, by contrast, is information that defines the relations between elements such as the fact that a banana is similar to a prune. Critically, relational information presupposes item-specific information. One can only relate banana and prune if relevant item-specific knowledge about the banana and prune is available. In other words, the processing of relational information is computationally more complex because it requires (1) the retrieval of item-specific information about elements of experience and (2) the formulation (not necessarily explicitly) of a relation between elements on the basis of such item-specific knowledge.

Bowler and Gaigg (2008) have argued that a definition of Minshew’s “complexity” in terms of item-specific (simple) and relational (complex) information provides a powerful tool for explaining the patterning of memory strengths and weaknesses in autism (see also Bowler et al. 2011 for an up-to-date review). For one, the distinction between relational and item-specific processing is widely thought to parallel that between episodic and semantic memory. More importantly, the distinction also helps to resolve the complex pattern in which “meaning” modulates memory in autism. The observations from memory illusion paradigms are particularly well suited to illustrate this point. In such paradigms, participants study lists of words of meaningful associates of one nonpresented item (e.g., night, pillow, tired, bed, dream. . . for *sleep*). Individuals with autism tend to recall fewer words from to-be-remembered lists in such tasks, but they nevertheless also fall prey to erroneously recalling the nonstudied target words (Bowler et al. 2000). This apparent paradox is explained by the fact that veridical recall of list

items depends on the effective processing of the relations between the items on the list, which, as noted earlier, also involves the bringing to mind of item-specific information about each individual word. False recall of the critical item, on the other hand, is largely the result of it being brought to mind by each of the studied words on the list. The critical word, in other words, constitutes part of the item-specific knowledge one has about the list items. Although this interpretation may seem contrived, it receives support from direct tests of the hypothesis that relational but not item-specific processing is compromised in autism (e.g., Gaigg et al. 2008) and from the neuroscientific literature (see Eichenbaum 2004; Mayes and Boucher 2008; Williams et al. 2008 for relevant reviews).

As noted earlier, until the 1980s, it was widely assumed that children were unable to form long-lasting declarative memories until the age of around 3–4 years. This view was mainly based on the fact that adults can rarely remember events from the first years of life, which seemed to suggest that no such event memories (i.e., episodic memories) are formed during early childhood. Just as archeologists can make mistakes in inferring the origin of certain artifacts, however, so developmental theorists can draw erroneous conclusions about developmental processes on the basis of adult behavior, and the domain of memory development provides a case in point. As it turns out, infants as young as 9 months can remember a surprising amount when tested with appropriate procedures (see Bauer 2006), and the most relevant to the study of autism is the observation that typical infants between 9 and 18 months rapidly acquire abilities that are indicative of relational memory processes. For instance, between 9 and 18 months, infants become increasingly proficient at deferred imitation tasks. In such tasks, an experimenter typically demonstrates a random sequence of actions such as banging a metal ring on a stick, then spinning the ring, and finally balancing it on a block. Following a delay of hours or even weeks, the infant who observed this sequence is presented with the same objects and encouraged to use them. Strikingly, by age 9 months, infants not only imitate the actions but they imitate them significantly above chance in the order in which they were

demonstrated. Since the ordering of events in memory is widely regarded as a product of episodic memory and relational processes (in this case temporal relations are processed), this provides strong support for the idea that relational memory processes begin to develop toward the latter stages of the first year of life (Bauer 2006). Additional evidence for this suggestion stems from intricately designed eye-tracking studies that assess novelty preferences in infants through visual paired comparison tasks. In such tasks, infants are first presented with a series of pictures, and after a short delay, they view the same pictures again alongside new and unfamiliar ones. In standard versions of this task, infants as young as 6 months demonstrate novelty preferences by visually exploring the novel picture for longer which indicates that they recognize the familiar one. Importantly, 6–9-month-olds do not demonstrate novelty preferences when tested in a different room to that in which the pictures were first seen. Such context dependence is thought to reflect a relative immaturity in relational processing since memory representations of the pictures are inflexibly bound to the memory representations of the context in which they were presented. The great advantage of mature relational processes is that they endow us with the ability to flexibly combine and recombine elements of past experiences, and this flexibility begins to emerge by about 12 months when infants begin to show novelty preference irrespective of context changes (see Jones and Herbert 2006; Jones et al. 2011 for further details). Importantly, the first reliable behavioral markers of autism are not evident until around 18–24 months, considerably later than the period during which relational processing capacities emerge in typical development.

Future Directions

The above overview is incomplete and oversimplified but nevertheless demonstrates that it is conceivable that memory atypicalities in autism emerge before the clinically defining features of the condition. In itself, this does not necessarily indicate that memory abnormalities are causally responsible for the clinical features of autism, but

it is a hypothesis that is in desperate need of empirical evaluation. The growing mainstream literature on relational memory development provides ample methodologies that can readily be adapted for this purpose, but in addition, there are other research directions that a relational processing hypothesis of autism invites. These directions are informed by a growing literature, which suggests that relational memory processes are not merely important for what we may narrowly consider to constitute “memory.” For instance, it is now well established that episodic memory and associated relational processes are not only important for dwelling on the past but also for imagining, predicting, and planning for the future (Schacter et al. 2008). Relational processes are also well established to be critical for a form of reasoning known as *transitive inference*, which allows us to make inferences such as “Paul is taller than Bob” given that “Paul is taller than Mark” and “Mark is taller than Bob,” and to infer relations among objects or subjects that share a common relation with a third object/subject (e.g., Susan knows Mary because Susan is Bill’s wife and Mary is Bill’s aunt) (Eichenbaum 2004). Relational processes are also critical for our ability to navigate three-dimensional environments (Eichenbaum 2004), and recent evidence also suggests that relational processes are important for certain forms of category learning (Zeithamova et al. 2008), analogical reasoning (Hummel and Holyoak 2003), symbolic and conceptual thinking (Kumaran et al. 2009), and, most importantly, understanding false belief (Bowler et al. 2005 and see Bowler et al. 2011). In short, there is absolutely no doubt that a disturbance in the typical development of relational processing capacities could have widespread repercussions on other areas of cognition, some of which have already been implicated in autism, others await closer scrutiny.

See Also

- [Episodic Memory](#)
- [Explicit Memory](#)
- [Memory](#)
- [Semantic Memory](#)

References and Reading

- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a ‘Theory of Mind?’. *Cognition*, 21, 37–46.
- Bauer, P. J. (2006). Constructing a past in infancy: A neurodevelopmental account. *Trends in Cognitive Sciences*, 10, 175–181.
- Boucher, J., & Warrington, E. K. (1976). Memory deficits in early infantile autism: Some similarities to the amnesic syndrome. *British Journal of Psychology*, 67, 73–87.
- Bowler, D. M., & Gaigg, S. B. (2008). Memory in ASD: Emerging themes and future prospects. In J. Boucher & D. M. Bowler (Eds.), *Memory in autism: Theory and evidence* (pp. 330–349). Cambridge: Cambridge University Press.
- Bowler, D. M., Gardiner, J. M., Grice, S., & Saavalainen, S. (2000). Memory illusions: False recall and recognition in high functioning adults with autism. *Journal of Autism and Developmental Disorders*, 30, 305–316.
- Bowler, D. M., Gardiner, J. M., & Berthollier, N. (2004). Source memory in adolescents and adults with Asperger’s syndrome. *Journal of Autism and Developmental Disorders*, 34, 533–542.
- Bowler, D. B., Briskman, J., Gurvidi, N., & Fornells-Ambrojo, M. (2005). Understanding the mind or predicting signal-dependent action? Performance of children with and without autism on analogues of the false-belief task. *Journal of Cognition and Development*, 6, 259–283.
- Bowler, D. M., Gardiner, J. M., & Gaigg, S. B. (2007). Factors affecting conscious awareness in the recollective experience of adults with Asperger’s syndrome. *Consciousness and Cognition*, 16, 124–143.
- Bowler, D. M., Gaigg, S. B., & Gardiner, J. M. (2008). Effects of related and unrelated context on recall and recognition by adults with high-functioning autism spectrum disorder. *Neuropsychologia*, 46, 993–999.
- Bowler, D. M., Gaigg, S. B., & Lind, S. (2011). Memory in autism: Binding, self and brain. In I. Roth & P. Rezaie (Eds.), *Researching the autism spectrum: Contemporary perspectives* (pp. 316–339). Cambridge: Cambridge University Press.
- Eichenbaum, H. (2004). Hippocampus: Cognitive processes and neural representations that underlie declarative memory. *Neuron*, 44, 109–120.
- Gaigg, S. B., Gardiner, J. M., & Bowler, D. M. (2008). Free recall in autism spectrum disorder: The role of relational and item-specific encoding. *Neuropsychologia*, 46, 986–992.
- Gardiner, J. M. (2008). Concepts and theories of memory. In J. Boucher & D. M. Bowler (Eds.), *Memory in autism: Theory and evidence* (pp. 3–20). Cambridge, UK: Cambridge University Press.
- Hermelin, B., & O’Connor, N. (1970). *Psychological experiments with autistic children*. Oxford: Pergamon Press.
- Hillier, A., Campbell, H., Kiellor, J., Phillips, N., & Beversdorf, D. Q. (2007). Decreased false memory for visually presented shapes and symbols among

- adults on the autism spectrum. *Journal of Clinical and Experimental Neuropsychology*, 29, 610–616.
- Hummel, J. E., & Holyoak, K. J. (2003). A symbolic-connectionist theory of relational inference and generalization. *Psychological Review*, 110, 220–264.
- Hunt, R. R., & Einstein, G. O. (1981). Relational and item-specific information in memory. *Journal of Verbal Learning and Verbal Behaviour*, 20, 497–514.
- Jones, E. J. H., & Herbert, J. S. (2006). Exploring memory in infancy: Deferred imitation and the development of declarative memory. *Infant and Child Development*, 15, 195–205.
- Jones, E. J. H., Pascalis, O., Eacott, M. J., & Herbert, J. S. (2011). Visual recognition memory across contexts. *Developmental Science*, 14, 136–147.
- Kumaran, D., Summerfield, J. J., Hassabis, D., & Maguire, E. A. (2009). Tracking the emergence of conceptual knowledge during human decision making. *Neuron*, 63, 889–901.
- Lind, S. E., & Bowler, D. M. (2010). Episodic memory and episodic future thinking in adults with autism. *Journal of Abnormal Psychology*, 119, 896–905.
- Mayes, A., & Boucher, J. (2008). Acquired memory disorders in adults: Implications for autism. In J. Boucher & D. M. Bowler (Eds.), *Memory in autism: Theory and evidence* (pp. 43–62). Cambridge, UK: Cambridge University Press.
- Minshew, N. J., & Goldstein, G. (2001). The pattern of intact and impaired memory functions in autism. *Journal of Child Psychology and Psychiatry*, 42, 1095–1101.
- Mottron, L., Morasse, K., & Belleville, S. (2001). A study of memory functioning in individuals with autism. *Journal of Child Psychology and Psychiatry*, 42, 253–260.
- Mottron, L., Dawson, M., & Soulières, I. (2008). A different memory: Are distinctions drawn from the study of non-autistic memory appropriate to describe memory in autism? In J. Boucher & D. M. Bowler (Eds.), *Memory in autism: Theory and evidence* (pp. 330–349). Cambridge: Cambridge University Press.
- Poirier, M., Martin, J. S., Gaigg, S. B., & Bowler, D. M. (2011). Short-term memory in autism spectrum disorder. *Journal of Abnormal Psychology*, 120, 247–252.
- Schacter, D. L., Addis, D. R., & Buckner, R. L. (2008). Episodic simulation of future events: Concepts, data, and applications. *Annals of the New York Academy of Sciences*, 1124, 39–60.
- Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry*, 34, 1351–1364.
- Tulving, E. (1985). How many memory systems are there? *American Psychologist*, 40, 385–398.
- Williams, D. L., Minshew, N. J., & Goldstein, G. (2008). Memory within a complex information processing model of autism. In J. Boucher & D. M. Bowler (Eds.), *Memory in autism: Theory and evidence* (pp. 330–349). Cambridge: Cambridge University Press.
- Zeithamova, D., Maddox, W. T., & Schnyer, D. M. (2008). Dissociable prototype learning systems: Evidence from brain imaging and behaviour. *The Journal of Neuroscience*, 28, 13194–13201.

Memory for Future Actions

► [Prospective Memory in Autism Spectrum Disorder](#)

Memory for Intentions

► [Prospective Memory in Autism Spectrum Disorder](#)

Menarche

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Synonyms

First “period”

Definition

Menarche is the medical term for the onset of menstruation, which is the periodic shedding of the uterine lining in females of reproductive age. Menarche is a hallmark of puberty and is often colloquially referred to as the first “period.” It typically follows thelarche (breast development) by approximately 2 years. In general, females must have at least 17% body fat in order for menarche to occur. The average age of menarche in the United States is between 12.1 and 12.6 years, with some variations across ethnic groups. African American females have a lower average age of menarche. The average age of menarche has been declining in developed countries over the last 150 years, likely as a result of improved adolescent nutrition. Low body weight and poor nutrition can delay menarche. Anovulatory cycles and irregular menses often occur in the first few months following menarche (Joffe 2006).

Girls on the autism spectrum may reach menarche later than the general population, but the

reasons for this delay are unclear. Proposed mechanisms include the role of elevated body mass index as well as increased exposure to androgen hormones during the prenatal period, though this hypothesis is controversial (Whitehouse et al. 2010). Children with autism often experience worsening symptoms during puberty (Knickmeyer et al. 2006). Autistic girls may be at increased risk for mood and behavioral disturbances around menstrual periods. More research is needed regarding the use of oral contraceptive pills (OCPs) for treatment of mood disturbances related to menses in this population (Burke et al. 2010). Medications that are commonly used to treat aggression and mood disturbances in children with autism can cause menstrual irregularities. For example, atypical antipsychotic medications can be associated with weight gain and elevated prolactin levels which in turn can impact the menstrual cycle.

The association between maternal early life factors and autism spectrum disorders is a subject of ongoing research. Maternal factors such as early menarche (age of menarche 10 years or less) and late adolescent obesity (body mass index greater than 30) are possibly linked to autism spectrum disorders (Lyall et al. 2010).

See Also

► **Antipsychotics: Drugs**

References and Reading

- Burke, L. M., Kalpakjian, C. Z., Smith, Y. R., & Quint, E. H. (2010). Gynecologic issues of adolescents with Down syndrome, autism, and cerebral palsy. *Journal of Pediatric and Adolescent Gynecology*, 23, 11–15.
- Joffe, A. (2006). Introduction to adolescent medicine, Chapter 89. In J. McMillan (Ed.), *Oski's pediatrics: Principles and practice* (4th ed.). Philadelphia: Lippincott Williams & Wilkins.
- Knickmeyer, R. C., Wheelwright, S., Hoekstra, R., & Baron-Cohen, S. (2006). Age of menarche in females with autism spectrum conditions. *Developmental Medicine and Child Neurology*, 48, 1007–1008.
- Lyall, K., Pauls, D. L., Santangelo, S., Spiegelman, D., & Ascherio, A. (2010). Maternal early life factors associated with hormone levels and the risk of having a child with an autism spectrum disorder in the nurses health study. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-010-1079-7>. Published online.
- Whitehouse, J. O., Murray, T. M., Hickey, M., & Sloboda, D. M. (2010). Brief report: Autistic-like traits in childhood predict later age at menarche in girls. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-010-1129-1>. Published online.

Meningitis

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Synonyms

Meningococcal disease: a specific form of meningitis that is caused by the bacteria *Neisseria meningitidis*

Definition

The brain and the spinal cord are surrounded by three membranes that are collectively termed meninges. *Meningitis* is inflammation of the meninges that is typically caused by infectious agents such as viruses, bacteria, fungi, and parasites. There are also noninfectious causes of meningitis, including certain drugs, malignancies, and autoimmune diseases. Classic clinical signs of meningitis are fever, headache, neck stiffness, light sensitivity (photophobia), and alterations in consciousness. These symptoms may be absent or difficult to appreciate in infants and very young children. Meningitis can also be associated with nonspecific systemic symptoms such as irritability or loss of appetite. Seizures and focal neurologic deficits can occur as well. The gold standard for diagnosis of meningitis is the lumbar puncture procedure, during which a sample of the cerebrospinal fluid (the fluid encircling the brain and spinal cord) is removed for analysis.

Viral meningitis is relatively more common than other forms and usually resolves without specific medication treatment. Bacterial

meningitis is less common than viral meningitis, but is associated with higher morbidity and mortality and requires antibiotic therapy. The most common bacterial causes of meningitis are *Escherichia coli* and group B streptococcus in neonates, *Streptococcus pneumoniae* and *Haemophilus influenzae* type b in older children, and *Neisseria meningitidis* in adolescents. The incidence of bacterial meningitis caused by *Streptococcus pneumoniae* and *Haemophilus influenzae* has decreased since the introduction of a vaccination program against these bacteria. It is advised that children receive a series of vaccinations against *Streptococcus pneumoniae* and *Haemophilus influenzae* between the ages of 2 months and 15 months. Routine vaccination against *Neisseria meningitidis* is recommended at the 11–12-year-old well-child visit. Older children who have not yet received this vaccine should be vaccinated at the earliest opportunity. Unvaccinated or incompletely vaccinated children are at higher risk of acquiring bacterial meningitis as are children who are immunocompromised. Intellectual disability, seizure disorders, hearing loss, developmental delays, and behavioral disturbances can all be sequelae of bacterial meningitis.

See Also

► [Measles-Mumps-Rubella \(MMR\) Vaccination](#)

References and Reading

- Centers for Disease Control. (2010). *Meningitis* [Data file]. Retrieved February 26, 2011, from <http://www.cdc.gov/meningitis/index.html>
- Harrison, L. H. (2010). Epidemiologic profile of meningococcal disease in the United States. *Clinical Infectious Diseases*, 50, S37–S44.
- Kim, S. K. (2010). Acute bacterial meningitis in infants and children. *Lancet Infectious Diseases*, 10, 32–42.
- Lynch, J. P., & Zhanel, G. G. (2010). *Streptococcus pneumoniae*: Epidemiology and risk factors, evolution of antimicrobial resistance, and impact of vaccines. *Current Opinion in Pulmonary Medicine*, 16, 217–225.
- Prober, C. G. (2007). Central nervous system infections, Chapter 602. In R. Kliegman (Ed.), *Nelson textbook of pediatrics* (18th ed.). Philadelphia: Saunders Elsevier.

Meningococcal Disease: A Specific Form of Meningitis that Is Caused by the Bacteria *Neisseria meningitidis*

► [Meningitis](#)

Mental Age

► [Age Equivalents](#)

Mental Flexibility

► [Cognitive Flexibility](#)

Mental Health and ASD

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Introduction

Autism spectrum disorder (ASD) is a heterogeneous disorder recognized by deficits in social communication and interaction, in addition to restricted and repetitive behaviors and interests (American Psychiatric Association 2013). ASD is also heterogeneous in terms of which symptom

emerges when and when the complete symptom pattern required for a diagnoses become evident. This leads to substantial difficulties in clinical detection and treatment planning. Research show that there is great variability in terms of etiology, behaviors, core symptoms, cognitive skills, adaptive skills, language and communication, the onset of diagnosis and core symptom patterns (Zwaigenbaum et al. 2015). For some, ASD symptoms required for a diagnosis may first become evident when social demands exceeds the capabilities of the individual (Ozonoff et al. 2015). Additionally, symptoms of ASD may manifest differently depending on other factors, such as the child's cognitive abilities and verbal and nonverbal levels of functioning (Chawarska et al. 2014).

The term "mental health" might be understood widely as the emotional, psychological, and social well-being of humans, as well as having few or no mental health problems. Furthermore, our mental health affects how we regulate ourselves, and how we cope with negative experiences, mending social relationships and make choices in challenging settings (WHO 2018). However, the term is frequently used intertwined with psychiatric conditions, which if present, naturally affects the emotional, psychological, and social well-being of that person. Furthermore, mental health in a wider perspective is also associated with the term Quality of Life (QoL), where aspects of both influence the well-being in all human beings.

Most research conducted on mental health related to autism is focused on issues of well-being, stress, and QoL in parents of individuals with ASD (Factor et al. 2018; Halstead et al. 2018; Leadbitter et al. 2018; Lecavalier et al. 2018; Nordahl-Hansen et al. 2018; Salomone et al. 2018; Schiltz et al. 2018), and it is well established that parents of children with ASD are at a higher risk of experiencing mental health problems often associated with increased levels of stress, anxiety, and depression (Lecavalier et al. 2018; Nordahl-Hansen et al. 2018; Øien and Eisemann 2016). Currently, it is far less investigations of mental health in individuals with ASD and has for many years been an under-researched topic (Burgess and Gutstein 2007).

In terms of quality of life, research have revealed that individuals with ASD reports lower levels of QoL than those without an ASD diagnosis (Ayres et al. 2017). Aside from studies on the QoL of individuals with ASD, very few studies to date have directly assessed the mental health of individuals with ASD. Increased focus on research related to improving mental health, well-being, and QoL in those with ASD have the potential of revealing factors of importance in terms of preventive interventions for developing psychiatric conditions. As of now, we know far less of how persons with ASD experiences factors in their own life, and how these influences their trajectories to better or worse mental health and QoL.

Current Knowledge

Currently, there is an understanding that having ASD in itself implies significant challenges for those affected and their families, and with a particular strain when ASD is accompanied by other comorbid or coexisting psychiatric conditions such as anxiety, depression, or with having acute and increasing levels of stress (Benson and Karlof 2009; Lai and Oei 2014; Nordahl-Hansen et al. 2018). It is, however, important to highlight that the term mental health comprises far more than the presence or absence of a psychiatric condition (WHO 2018). Mental health is also about the emotional and social well-being, happiness, as well as having a sense of safe and meaningful life.

Also, it is important to distinguish the terms comorbid and coexisting as representing two different terms. *Comorbidity* refers usually to a state when two diagnoses are present and the conditions are related to each other, while *coexisting* refers to when two diagnoses are present without necessarily being related, i.e., being diagnosed with ASD and diabetes are coexisting conditions, while ASD and intellectual disability (ID) are comorbid conditions (Kaplan et al. 2016).

In terms of prevalence of psychiatric conditions found in individuals with ASD, Simonoff et al. (2008) reported that 70% of children with an ASD diagnosis had one comorbid diagnosis,

while 41% had two or more (Simonoff et al. 2008). This indicates that psychiatric comorbidities are very common in ASD and often presents in higher proportions that would be expected compared to what is known from prevalence in the general populations. Studies have revealed that the most common psychiatric diagnoses are social anxiety disorder, attention deficit hyperactivity disorder (ADHD), and oppositional defiant disorder (ODD) (Mattila et al. 2010; Simonoff et al. 2008). The increased prevalence of comorbid psychiatric conditions or problems in ASD might indicate that a substantial portion of individuals with ASD have poorer mental health and quality of life due to the consequences of the psychiatric condition/problem. However, we do not know how the interplay between having comorbid conditions and having a feeling of quality of life, well-being, and happiness are. It is a knowledge gap in research on understanding what constitutes factors of importance in a preventive perspective as for the development of poor mental health.

It is important to acknowledge that the heterogeneity in symptom patterns in ASD can manifest in different ways, and symptoms can appear similar to phenomena found in other psychiatric conditions (Kaplan et al. 2016). However, it might be that psychotic-like symptoms in individuals with ASD are better explained as a part of their underlying ASD diagnosis and related to specific cognitive challenges and as such will have different implications for our understanding, treatment, prognosis, and access to services (van Schalkwyk et al. 2015). This highlights the need to be cautious in adding diagnosis in individuals with ASD, as they might be associated with different factors than found in non-ASD individuals and as such require different strategies for treatment. However, it is equally important to provide evidence-based treatment for persons with ASD in the case of having other coexisting clinical problems.

A plausible model of what contributes to good/poor mental health in ASD is bidirectionality, where symptoms related to the core of ASD directly affects socio-emotional well-being negatively, increasing the likelihood for developing

psychological problems or conditions such as anxiety and depression, which further spiraling the socio-emotional impairment downwards.

As ASD is recognized by its core deficits in social communication and interaction (American Psychiatric Association 2014), affecting the opportunities they have to establish and maintain social relationships with others, often leading to loneliness and even social anxiety (Bauminger and Kasari 2000; White and Roberson-Nay 2009) (Cacioppo et al. 2006; Leary 1990). Little research has been devoted to understanding the positive mental health development in individuals with ASD, and very few measures have been developed with the purpose of providing how they perceive their own emotional, psychological, and social well-being.

Future Directions

As very few to this date have conducted research on mental health-related phenomena such as the emotional, psychological, and social well-being of individuals with ASD (mental health), future studies should aim at investigating measures that can shed light on mental health in those with an ASD diagnosis. There is, an increasing interest in conduction research on how individuals with ASD perceive their quality of life, and specific measures for this purpose are starting to emerge (Ayres et al. 2017; Burgess and Gutstein 2007; McConachie et al. 2017). There are great difficulties in ascertaining information directly from different subpopulations within the Autism spectrum in regards to ability of an interest in reflecting on quality of life (QoL), emotional, psychological, and social well-being. Many persons within the spectrum have severe intellectual disability and might have little functional language. It is still of paramount importance to develop relevant, valid, and reliable observational measures to use as proxy measures used by parents or other who know the child well. Expected outcomes would be to understand the interplay between everyday conditions that influences the trajectories of mental health issues (positive or negative). In addition, exploring if children with different symptom

patterns constitute different risk markers for a positive or negative development. There has been made some advances in terms of what parents highlights as the most important measures of outcomes to target in interventions (McConachie et al. 2018). Their highest ranked outcomes impacted directly on everyday life and functioning (anxiety, distress, hypersensitivity, sleep problems, happiness, relationships with brothers and sisters, and parent stress) (McConachie et al. 2018). Furthermore, it is important to investigate in further depth how a diagnosis of ASD might not only make them more at risk for developing psychiatric conditions affecting mental health but also how core features of ASD mediate mental health outcomes. More importantly, investigating what constitutes good mental health in individuals with ASD is of great importance to develop new measures.

Longitudinal studies following children and adolescents could potentially provide information on which factors that predict later psychiatric conditions in individuals with ASD and other neurodevelopmental disorders. Such studies could also identify sex-specific information, as well as preventive factors for negative mental health development.

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5[®])*. Washington, DC: American Psychiatric Publishing.
- American Psychiatric Association. (2014). *Diagnostic and statistical manual of mental disorders*. APA DSM. Arlington: American Psychiatric Publishing.
- Ayres, M., Parr, J. R., Rodgers, J., Mason, D., Avery, L., & Flynn, D. (2017). A systematic review of quality of life of adults on the autism spectrum. *Molecular Autism*, 22(7), 774–783. <https://doi.org/10.1177/1362361317714988>.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development*, 71(2), 447–456.
- Benson, P. R., & Karlof, K. L. (2009). Anger, stress proliferation, and depressed mood among parents of children with ASD: A longitudinal replication. *Journal of Autism and Developmental Disorders*, 39(2), 350–362.
- Burgess, A. F., & Gutstein, S. E. (2007). Quality of life for people with autism: Raising the standard for evaluating successful outcomes. *Child and Adolescent Mental Health*, 12(2), 80–86. <https://doi.org/10.1111/j.1475-3588.2006.00432.x>.
- Cacioppo, J. T., Hughes, M. E., Waite, L. J., Hawkley, L. C., & Thisted, R. A. (2006). Loneliness as a specific risk factor for depressive symptoms: Cross-sectional and longitudinal analyses. *Psychology and Aging*, 21(1), 140.
- Chawarska, K., Shic, F., Macari, S., Campbell, D. J., Brian, J., Landa, R., et al. (2014). 18-month predictors of later outcomes in younger siblings of children with autism spectrum disorder: A baby siblings research consortium study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 53(12), 1317–1327.e1. <https://doi.org/10.1016/j.jaac.2014.09.015>.
- Factor, R. S., Swain, D. M., & Scarpa, A. (2018). Child autism spectrum disorder traits and parenting stress: The utility of using a physiological measure of parental stress. *Journal of Autism and Developmental Disorders*, 48(4), 1081–1091.
- Halstead, E., Ekas, N., Hastings, R. P., & Griffith, G. M. (2018). Associations between resilience and the well-being of mothers of children with autism spectrum disorder and other developmental disabilities. *Journal of Autism and Developmental Disorders*, 48(4), 1108–1121.
- Kaplan, B. J., Dewey, D. M., Crawford, S. G., & Wilson, B. N. (2016). The term comorbidity is of questionable value in reference to developmental disorders. *Journal of Learning Disabilities*, 34(6), 555–565. <https://doi.org/10.1177/002221940103400608>.
- Lai, W. W., & Oei, T. P. S. (2014). Coping in parents and caregivers of children with autism spectrum disorders (ASD): A review. *Review Journal of Autism and Developmental Disorders*, 1(3), 207–224.
- Leadbitter, K., Aldred, C., McConachie, H., Le Couteur, A., Kapadia, D., Charman, T., et al. (2018). The autism family experience questionnaire (AFEQ): An ecologically-valid, parent-nominated measure of family experience, quality of life and prioritised outcomes for early intervention. *Journal of Autism and Developmental Disorders*, 48(4), 1052–1062.
- Leary, M. R. (1990). Responses to social exclusion: Social anxiety, jealousy, loneliness, depression, and low self-esteem. *Journal of Social and Clinical Psychology*, 9(2), 221–229.
- Lecavalier, L., Pan, X., Smith, T., Handen, B. L., Arnold, L. E., Silverman, L., et al. (2018). Parent stress in a randomized clinical trial of atomoxetine and parent training for children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(4), 980–987.
- Mattila, M.-L., Hurtig, T., Haapsamo, H., Jussila, K., Kuusikko-Gauffin, S., Kielinen, M., et al. (2010). Comorbid psychiatric disorders associated with Asperger syndrome/high-functioning autism: A community-and clinic-based study. *Journal of*

- Autism and Developmental Disorders*, 40(9), 1080–1093.
- McConachie, H., Mason, D., Parr, J. R., Garland, D., Wilson, C., & Rodgers, J. (2017). Enhancing the validity of a quality of life measure for autistic people. *Journal of Autism and Developmental Disorders*, 48(5), 1596–1611. <https://doi.org/10.1007/s10803-017-3402-z>.
- McConachie, H., Livingstone, N., Morris, C., Beresford, B., Le Couteur, A., Gringras, P., et al. (2018). Parents suggest which indicators of progress and outcomes should be measured in young children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(4), 1041–1051.
- Nordahl-Hansen, A., Hart, L., & Øien, R. A. (2018). The scientific study of parents and caregivers of children with ASD: A flourishing field but still work to be done. *Journal of Autism and Developmental Disorders*, 48(4), 976–979. <https://doi.org/10.1007/s10803-018-3526-9>.
- Øien, R., & Eisemann, M. (2016). Parental response to diagnosis. In *Encyclopedia of autism spectrum disorders* (Vol. 41, pp. 1–4). New York: Springer. https://doi.org/10.1007/978-1-4614-6435-8_102113-1.
- Ozonoff, S., Young, G. S., Landa, R. J., Brian, J., Bryson, S., Charman, T., et al. (2015). Diagnostic stability in young children at risk for autism spectrum disorder: A baby siblings research consortium study. *Journal of Child Psychology and Psychiatry*, 56(9), 988–998. <https://doi.org/10.1111/jcpp.12421>.
- Salomone, E., Leadbitter, K., Aldred, C., Barrett, B., Byford, S., Charman, T., et al. (2018). The association between child and family characteristics and the mental health and wellbeing of caregivers of children with autism in mid-childhood. *Journal of Autism and Developmental Disorders*, 48(4), 1189–1198.
- Schiltz, H. K., McVey, A. J., Magnus, B., Dolan, B. K., Willar, K. S., Pleiss, S., et al. (2018). Examining the links between challenging behaviors in youth with ASD and parental stress, mental health, and involvement: Applying an adaptation of the family stress model to families of youth with ASD. *Journal of Autism and Developmental Disorders*, 48(4), 1169–1180.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(8), 921–929. <https://doi.org/10.1097/CHL.0b013e318179964f>.
- van Schalkwyk, G. I., Peluso, F., Qayyum, Z., McPartland, J. C., & Volkmar, F. R. (2015). Varieties of misdiagnosis in ASD: An illustrative case series. *Journal of Autism and Developmental Disorders*, 45(4), 911–918.
- White, S. W., & Roberson-Nay, R. (2009). Anxiety, social deficits, and loneliness in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(7), 1006–1013.
- WHO. (2018). Mental health: Strengthening our response. Retrieved November 27, 2018, from <https://www.who.int/news-room/fact-sheets/detail/mental-health-strengthening-our-response>
- Zwaigenbaum, L., Bauman, M. L., Stone, W. L., Yirmiya, N., Estes, A., Hansen, R. L., et al. (2015). Early identification of autism spectrum disorder: Recommendations for practice and research. *Pediatrics*, 136(Supplement), S10–S40. <https://doi.org/10.1542/peds.2014-3667C>.

Mental Health Interventions

► Child Psychotherapy

Mental Health Practitioner

► Clinical Social Worker

Mental Representation

► Client Emotional Processing Scale for Autism Spectrum

Mental Retardation

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Synonyms

Cognitive delay; Developmental disability (Ontario); Intellectual disability; Learning disability (UK)

Definition

The condition associated with subaverage intellectual functioning has undergone many changes in name and definition since it was first recognized (circa 1493; Scheerenberger 1983). In fact, the term “mental retardation” is no longer current; “intellectual disability” is the currently accepted term. Although a complete history of terminology and classification of intellectual disability is outside the scope of this entry, the highlights of the past quarter century follow. The term *mental retardation* replaced the term *mental deficiency* in 1987, and the condition underwent several minor definition changes during the following 25 years. Perhaps the most significant change to the definition occurred in 1992 when the concept of support was introduced as a fundamental aspect of the classification system. The current diagnostic criteria for intellectual disability are based on three criteria: (1) *significant limitations in intellectual functioning*; (2) *significant limitations in adaptive behavior expressed in conceptual, social, and practical adaptive skills*; and (3) *age of onset before the age of 18* (Schalock et al. 2010).

Throughout history, people with intellectual disabilities have been devalued and stigmatized. As such, the terms associated with this condition quickly acquire the stigma and are used pejoratively in common vernacular. Earlier terms such as feeble-minded, idiot, moron, imbecile, and mentally deficient each acquired social stigma and became offensive and hurtful to people with intellectual disability and their families. In recent years, the term mental retardation has acquired a derogatory connotation. For this reason, self-advocates, parents, and administrators sought an acceptable substitute. Rosa’s Law, signed into effect on October 5, 2010 by President Obama, changed the official federal terminology from mental retardation to intellectual disability. This law was preceded by the American Association on Intellectual and Developmental Disabilities who made the name change in 2007 and the state of Maryland who made the change in 2009. The change from mental retardation to intellectual disability was a change in name only; there was no

change in the diagnostic criteria, which allowed individuals receiving services under the term mental retardation to continue receiving the same services without interruption.

See Also

- [Developmental Disabilities](#)
- [Intellectual Disability](#)

References and Reading

- Luckasson, R., & American Association on Mental Retardation. (2002). *Mental retardation: Definition, classification and systems of supports*. Washington, DC: American Association on Mental Retardation (AAMR).
- Schalock, R. L., Luckasson, R. A., & Shogren, K. A. (2007). The renaming of “Mental retardation” understanding the change to the term “Intellectual disability”. *Intellectual and Developmental Disabilities*, 45(2), 116–124.
- Schalock, R. L., Borthwick-Duffy, S. A., Bradley, V. J., Buntinx, W. H. E., Coulter, D. L., Craig, E. M., Gomez, S. C., & American Association on Intellectual and Developmental Disabilities. (2010). *Intellectual disability: Definition, classification, and Systems of Supports* (11th ed.). Washington, DC: American Association on Intellectual and Developmental Disabilities.
- Scheerenberger, R. C. (1983). *A history of mental retardation* (1st ed.). Baltimore: Brookes Publishing.
- Taylor, R. L., Richards, S. B., & Brady, M. P. (2005). *Mental retardation: Historical perspectives, current practices and future directions*. New York: Pearson.

Mercyism

- [Rumination](#)
- [Rumination Disorder](#)

Mesencephalon

- [Midbrain](#)

Mesibov, Gary

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Name and Degrees

- Gary B. Mesibov
- Stanford University, AB Psychology, 1967
- University of Michigan, MA Developmental Psychology, 1968
- Brandeis University, PhD Psychology, 1974

Major Appointments (Institution, Location, Dates)

- 2000 – Present Fellow, Frank Porter Graham Child Development Center, University of North Carolina at Chapel Hill
- 1987 – Present Professor of Psychology Emeritus, Department of Psychiatry, University of North Carolina at Chapel Hill
- 1987 – Present Clinical Professor Emeritus, Department of Psychology, University of North Carolina at Chapel Hill
- 1992–2010 Director, Division TEACCH (Treatment and Education of Autistic and Related Communication-Handicapped Children), University of North Carolina at Chapel Hill
- 1988–1992 Codirector, Division TEACCH
- 1981–1987 Associate Professor of Psychology, Department of Psychiatry, University of North Carolina at Chapel Hill
- 1983–1987 Associate Director, Division TEACCH
- 1979–1982 Coordinator of Adolescent and Adult Services, Division TEACCH
- 1975–1981 Assistant Professor of Psychology, Department of Psychiatry, University of North Carolina at Chapel Hill

- 1975–1979 Psychologist, Division for Disorders of Development and Learning, University of North Carolina at Chapel Hill
- 1974–1975 Postdoctoral Fellow, Clinical Child Psychology, Division for Disorders of Development and Learning, University of North Carolina at Chapel Hill

Major Honors and Awards

- 1982 President, Society of Pediatric Psychology (Section 5 of the American Psychological Association Division of Clinical Psychology)
- 1982 Certificate of Appreciation for Contributions to Developmentally Handicapped Citizens of North Carolina (Awarded by Orange County, NC Association for Retarded Citizens)
- 1984 Fellow Status, American Psychological Association Divisions 37, 12
- 1989 Distinguished Professional Contribution Award, Society of Pediatric Psychology, American Psychological Association
- 1990 First recipient of Mesibov Award, given annually by Residential Services, Inc. for excellence in community-based residential service system for developmentally handicapped children and adults in Orange County, NC
- 1994 Opleidingscentrum (Belgium) International Award for contributions translating theory into effective practice in autism
- 1994 Mary G. Clarke Award from the North Carolina Psychological Association given annually to a psychologist in North Carolina for outstanding contributions spanning several years. Contributions must reflect qualities of dedication, competence, high ethical standards, advocacy for the field of psychology, and sensitivity
- 1995 MAPP (International organization to help More Able Autistic People) Award for invaluable service to the organization and to high-functioning people with autism
- 1997 Distinguished Professional Contributions Award for Public Service, American Psychological Association
- 2000 “Doctor Honoris Causa” (Honorary Degree) from the University of Mons-Hainaut

(Belgium) for contributions to improving the quality of life for people with autism throughout the world

- 2000 Emily and Frank Puzio Award from the Eden Institute's Princeton Lecture Series for leadership in improving the quality of life of individuals with autism
- 2006 UNC Chancellor's Award for Excellence for meritorious accomplishments clearly above and beyond what is expected in human relations
- 2006 NC State Employees Award for Excellence representing outstanding performance. This is the highest award an NC state employee can receive
- 2010 Danish National Autism Society Professional of the Year for doing the most to advance treatment and education of people with autism spectrum disorders
- 2010 American Association on Intellectual Developmental Disabilities Award for service to people with disabilities.
- 2010 Autism Society of America Founders Award for career substantive contributions to the field of autism spectrum disorders
- 2011 Honorary Degree from the University of Northampton (UK) for contributions to the education of children and adults on the autism spectrum

Landmark Clinical, Scientific, and Professional Contributions

Gary B. Mesibov has dedicated his career as a psychologist to autism. His contributions include a major assessment instrument; a book series; a comprehensive program of educational, vocational, residential, and family support services; and a unique training program for other professionals. His reputation among students, families, and professional colleagues is one of wisdom, generosity, compassion, and amazing productivity. He brings intellectual rigor to his professional activities and combines it with gentleness and respect for all clients and their families. His influence began in North Carolina and has spread throughout the world.

Short Biography

Gary B. Mesibov defended his doctoral dissertation in psychology at Brandeis University in the spring of 1974. Shortly thereafter, he arrived at the University of North Carolina at Chapel Hill as a post-doctoral fellow in the Division for Disorders of Development and Learning (DDDL). Gary's time at the DDDL marked the beginning of his professional identification with the field of developmental disabilities. Recognized as a gifted and prolific psychologist, after only 18 months, Gary was asked to accept a staff position at the DDDL with an appointment as an Assistant Professor of Psychology in the Department of Psychiatry. Gary's quiet passion for understanding and serving people with developmental disabilities soon led to professional involvement with newly developing service models in North Carolina, including group homes, sheltered workshops, limited guardianship, and social skills training, as well as service on a human rights committee at the regional residential facility for people with severe/profound intellectual disabilities. Gary conducted research, introduced students to the rewards and fascinations of developmental disabilities, and worked closely with clients and families. At the same time, he was teaching in the Department of Psychology, supervising interns and postdoctoral fellows in the Department of Psychiatry, and devoting considerable time and effort to the development of the Society of Pediatric Psychology (Section 5 of the American Psychological Association's Division 12).

Chapel Hill had long been a center of excellence in the field of developmental disabilities. In addition to the DDDL's broad-based training program in developmental disabilities, a small, psychoanalytically oriented program on autism had functioned for some years within the Department of Psychiatry. In 1966, this program was expanded and refocused by Eric Schopler and Robert Reichler on the assumptions that autism was a neurobiological disorder and that parents and professionals could work as cotherapists to help children with autism develop. In 1972, that program was renamed TEACCH (Treatment and Education of Autistic and Related Communication-Handicapped Children) and

funded by the North Carolina General Assembly as the nation's first statewide autism program.

Eric recruited Gary in 1979 to assume the new position of Coordinator of Adolescent and Adult Services at TEACCH. Four years later, Gary became Associate Director; 5 years later, codirector; and in 1992, with Eric Schopler's semiretirement, Director of Division TEACCH. Gary served in this position for 18 years until his retirement in 2010. This was a formative period for the organization as it grew to nine regional centers across the state of North Carolina, serving thousands of individuals on the autism spectrum. It was also a period of both broadening and deepening TEACCH's philosophical underpinnings and, as a result, its approach to intervention for individuals with autism. Eric had first understood the importance of using structure to help children with autism focus and learn; Gary elaborated on the principles of structured teaching and applied them to classroom, vocational, and residential settings. He also emphasized the importance of improving the quality of life for individuals on the autism spectrum, not merely addressing "deficits" or "behavior problems." In 2004, Gary coauthored *The TEACCH Approach to Autism Spectrum Disorders* as an introduction to the TEACCH philosophy, structured teaching, and its application across a wide range of contexts.

From 1983 until 1998, Gary and Eric coedited the "Current Issues in Autism" series for Plenum Press. These books were based on the yearly TEACCH conference held in Chapel Hill each May. The conference has drawn noted autism experts such as Margaret Bauman, Eric Courchesne, Geraldine Dawson, Christopher Gillberg, Temple Grandin, Cathy Lord, Sally Ozonoff, Michael Rutter, Oliver Saks, Wendy Stone, and Lorna Wing. Conference speakers uniformly, and quite independently, speak with affection and admiration for Gary's contributions. Throughout the years, he has demonstrated a remarkable capacity to maintain warm, personal relationships with professionals around the world.

Like most respected academics, Gary began to be asked to give talks or provide training outside his hometown. In the late 1970s, his talks were in rural North Carolina, with occasional well-timed presentations at the Atlantic coast in the summer.

By the 1980s, he was accepting invitations to Kentucky, Ohio, Georgia, Florida, and Oregon. During this time, he was also developing a unique, week-long, multimodal training program for teachers and other professionals working with students with autism. Gary and a training team would arrive at a location on Sunday, set up a model classroom with local students with autism on Monday morning, and take turns giving didactic lectures and operating the classroom. The trainees would observe trainers working with the students and then take over themselves, designing and presenting teaching activities that put into practice the principles they had been hearing in the lectures. After the local students went home for the day, there would be small-group and large-group debriefings of what trainees had tried, what had worked, and what had not worked. This model classroom has been presented every summer since 1985, training more than 2000 teachers of students with autism. Gary and his teams have also presented the classroom and other types of training in Australia, Belgium, China, Denmark, England, France, Germany, Hong Kong, India, Italy, Japan, Kuwait, Mexico, New Zealand, Northern Ireland, Norway, Pakistan, Qatar, Russia, Saudi Arabia, Singapore, Sweden, and Venezuela.

Gary was also involved in developing the Adolescent and Adult Psychoeducational Profile (AAPEP), an assessment instrument for adolescents and adults with autism. In 2007, he and his colleagues issued a major revision of the instrument, renamed the TEACCH Transition Assessment Profile (TTAP), in order to assess more accurately the skills needed for successful employment in community settings and to promote independent functioning in the home, workplace, and community. Gary's vision and leadership have been the impetus behind TEACCH's Supported Employment Program, which continues to be one of the most successful such programs in the nation for adults with autism.

Gary served for 10 years as editor of the *Journal of Autism and Developmental Disorders* and on the editorial boards of the *Journal of Pediatric Psychology*, *Journal of Clinical Child Psychology*, and *Journal of Cognitive Rehabilitation*, in addition to being a guest reviewer for almost a dozen other journals. Over his career, Gary has

earned a reputation as someone who can translate basic research into everyday practice. In recent years, he has helped the autism community think carefully about “evidence-based practice” and how this concept can be applied to specific autism interventions and to broad-based program models.

Gary’s many professional awards demonstrate his commitment to advancing the science and practice of psychology while maintaining a clear focus on improving quality of life for individuals with autism across the lifespan. For example, he received the 1997 American Psychological Association’s Distinguished Professional Contributions Award for Public Service. He has received honorary doctoral degrees from the University of Mons-Hainaut (Belgium, 2000) for “contributions to improving the quality of life for people with autism throughout the world” and from the University of Northampton (United Kingdom, 2011) “for his contribution to the education of children and adults on the autism spectrum.” In 2006, Gary received the University of North Carolina at Chapel Hill Chancellor’s Award for “meritorious accomplishments” on behalf of the university. In 2010, he was named “Professional of the Year” by the Danish Autism Society for “doing the most to advance treatment and education of people with autism spectrum disorders.” Also in 2010, Gary received the Autism Society of America Founders Award for “career substantive contributions to the field of autism spectrum disorders.” These are just the highlights from a career marked by recognition from parents and professionals alike.

Since stepping down as director of Division TEACCH, Gary has found a bit more time for his grandchildren, but he has not lost his passion for improving the quality of life for individuals with autism, particularly for adolescents and adults. He continues to inspire the TEACCH staff and the autism community at large to embrace “the culture of autism” as a metaphor for understanding the unique qualities of the children, adolescents, and adults they serve.

See Also

- [TEACCH Transition Assessment Profile \(TTAP\)](#)

References and Reading

- Keel, J. H., Mesibov, G. B., & Woods, A. V. (1997). TEACCH-supported employment program. *Journal of Autism and Developmental Disorders*, 27, 3–9.
- LaGreca, A. M., & Mesibov, G. B. (1979). Social skills intervention with learning disabled children: Selecting skills and implementing training. *Journal of Clinical Child Psychology*, 8, 234–241.
- Mesibov, G. B. (1996). Division TEACCH: A collaborative model program for service delivery, training and research for people with autism and related communication handicaps. In M. C. Roberts (Ed.), *Model programs in child and family mental health* (pp. 215–230). Hillsdale: Erlbaum.
- Mesibov, G. B., & Handlan, S. (1997). Adolescents and adults with autism. In D. J. Cohen & E. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (2nd ed., pp. 309–322). Silver Spring: Winston.
- Mesibov, G. B., & Shea, V. (2010). The TEACCH program in the era of evidence-based practice. *Journal of Autism and Developmental Disorders*, 40, 570–579.
- Mesibov, G. B., & Shea, V. (2011). Evidence-based practices and autism. *Autism*, 15(1), 1114–1133.
- Mesibov, G. B., Schopler, E., Schaffer, B., & Landrus, R. (1988). *Individual assessment and treatment for autistic and developmentally disabled children: Vol. 4. Adolescent and Adult Psychoeducational Profile (AAPEP)*. Austin: Pro-Ed.
- Mesibov, G. B., Adams, L. W., & Klinger, L. G. (1998). *Autism: Understanding the disorder*. New York: Plenum.
- Mesibov, G. B., Shea, V., & Adams, L. W. (2001). *Understanding Asperger syndrome and high functioning autism*. New York: Plenum.
- Mesibov, G. B., Shea, V., & Schopler, E. (2004). *The TEACCH approach to autism spectrum disorders*. New York: Plenum.
- Mesibov, G. B., Thomas, J. B., Chapman, S. M., & Schopler, E. (2007). *TEACCH transition assessment profile, second edition (TTAP)*. Austin: Pro-Ed.
- Schopler, E., & Mesibov, G. B. (Eds.). (1983). *Autism in adolescents and adults*. New York: Plenum.
- Schopler, E., & Mesibov, G. B. (Eds.). (1984). *The effects of autism on the family*. New York: Plenum.
- Schopler, E., & Mesibov, G. B. (Eds.). (1985). *Communication problems in autism*. New York: Plenum.
- Schopler, E., & Mesibov, G. B. (Eds.). (1986). *Social behavior in autism*. New York: Plenum.
- Schopler, E., & Mesibov, G. B. (Eds.). (1987). *Neurobiological issues in autism*. New York: Plenum.
- Schopler, E., & Mesibov, G. B. (Eds.). (1988). *Diagnosis and assessment in autism*. New York: Plenum.
- Schopler, E., & Mesibov, G. B. (Eds.). (1992). *High-functioning individuals with autism*. New York: Plenum.
- Schopler, E., & Mesibov, G. B. (Eds.). (1994). *Behavioral issues in autism*. New York: Plenum.
- Schopler, E., & Mesibov, G. B. (Eds.). (1995). *Learning and cognition in autism*. New York: Plenum.
- Schopler, E., & Mesibov, G. B. (2000). Cross-cultural priorities in developing autism services. *International Journal of Mental Health*, 29, 3–21.

- Schopler, E., Mesibov, G. B., & Hearsey, K. (1995). Structured teaching in the TEACCH system. In E. Schopler & G. B. Mesibov (Eds.), *Learning and cognition in autism* (pp. 243–268). New York: Plenum.
- Schopler, E., Mesibov, G. B., & Kuncze, L. (Eds.). (1998). *Asperger syndrome or high-functioning autism?* New York: Plenum.

Mesoridazine

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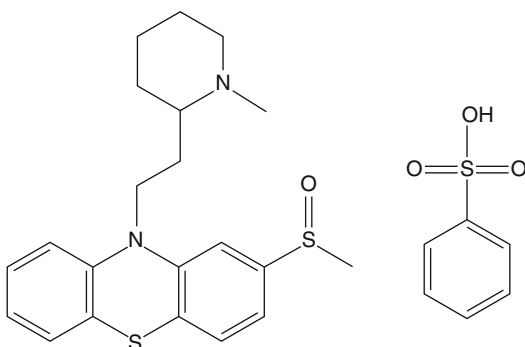
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Synonyms

Mesoridazine besylate; Serentil

Definition



Mesoridazine is a prescription drug in the group of piperidine phenothiazines in the family of first-generation antipsychotics whose active ingredient

mesoridazine besylate has the chemical formula $C_{27}H_{32}N_2O_4S_3$. This drug was initially FDA-approved for medical use in the year 1970 and was produced by the company Novartis, but the production of this drug has been discontinued. This compound has relatively low potency compared to the other first-generation antipsychotics, and its mechanism of action is thought to involve anticholinergic binding. This drug is FDA-approved for the treatment of schizophrenia and can be used to treat aggressive symptoms. Observed side effects include drowsiness/sedation, Parkinsonism, akathisia, orthostatic hypotension, tachycardia, electrocardiogram (ECG) abnormalities, anticholinergic effects, sexual dysfunction, galactorrhea, and weight gain.

See Also

► [Antipsychotics: Drugs](#)

References and Reading

- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001). *Principles and practice of psychopharmacotherapy* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
- Lidanyl. (n.d.). Retrieved from the ChemSpider Wiki: <http://www.chemspider.com/Chemical-Structure.33296.html>.
- Stahl, S. M. (2000). Antipsychotic agents. In *Essential psychopharmacology: Neuroscientific basis and clinical applications* (pp. 401–458). Cambridge, MA: Cambridge University Press.
- U.S. Food and Drug Administration. (2011). *Serentil*. Retrieved from: <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm?fuseaction=Search.Overview&DrugName=SERENTIL&CFID=58052189&CFTOKEN=7a0a0c50a125be98-23E871A7-C2D6-F473-9C9052E3B6C515AD>.
- Wilkaitis, J., Mulvihill, T., & Nasrallah, H. A. (2006). Classic antipsychotic medications. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 211–228). Washington, DC: American Psychiatric Publishing.

Mesoridazine Besylate

► [Mesoridazine](#)

Messages

► Vocabulary

Metabolic Testing

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Description

Definition

Metabolic testing is the laboratory evaluation looking for inborn errors of metabolism. Inborn errors of metabolism are genetic or inherited conditions that cause symptoms because of a lack of a necessary enzyme or biochemical process that results in the inability to convert substrates into the appropriate biologic product in the body. Some inborn errors of metabolism result in toxic accumulations of substances that cannot be metabolized, and others have symptoms that result from a deficit in production of necessary biochemical compounds.

Specific biochemical tests are used to examine the metabolism of compounds such as amino acids, carbohydrates, fatty aldehydes, organic acids, peroxisomes, lysosomes, and others.

Background

Inborn errors of metabolism are seen in the general population at a rate of 0.1–0.4%. It has been suggested that they may be identified in 3–5% of children on the autism spectrum. However, there are many types of inborn metabolic disorders, and each type is extremely rare. Categories of inborn errors of metabolism include metabolism of carbohydrates or sugars, problems with processing protein or amino acids, mitochondrial disorders, storage disorders, disorders of steroid metabolism, and many others.

Metabolic testing may be ordered to look for rare causes of developmental disability in the presence of a history of regression, a family history of early childhood deaths or regression, specific symptoms consistent with a metabolic disorder, or findings on physical or neurologic exam suggesting a metabolic disorder might be present. Metabolic testing is performed through newborn screening in every state in the United States. Different panels are included in newborn testing in each state. Some allow families to refuse. Newborn screening takes a few drops of blood dried on a filter paper that are then evaluated using very specific methodology. Most states include PKU, galactosemia, biotinidase deficiency, congenital adrenal hyperplasia, maple syrup urine disease, tyrosinemia, and other non-metabolic disorders like hypothyroidism, sickle cell syndrome, and cystic fibrosis.

The symptoms of inborn errors of metabolism might occur at birth, sometime in the first 3 years of life or later depending on the disorder and whether their might be some residual metabolic function. Some disorders like phenylketonuria occur when the placenta no longer compensates for the metabolic error. In other situations, decompensation occurs during a serious illness or major stressor. Others are progressive such as infantile ceroid lipofuscinosis, biotinidase deficiency, and Sanfilippo syndrome.

Abnormalities on newborn screening typically require additional testing to confirm that a problem exists. Specific tests are used for each of the identified metabolic disorders. A specialist such as a geneticist or child neurologist often must consult to help guide the evaluation.

The first inborn error of metabolism to benefit from widespread newborn screening was phenylketonuria or PKU. It is due to an absence of the enzyme that metabolizes the amino acid phenylalanine into tyrosine. Without this enzyme, a toxic metabolite builds up and results in neurologic impairment. This is the most common of the inborn errors of metabolism and is seen in 1 in 10,000 live births. The treatment for this is diet by limiting intake of Phe. Untreated children with PKU developed intellectual disability and often autism. The prevalence of autism in PKU has dropped from 20% to around 5% due to early

detection and treatment with diet. Even if PKU is treated with diet, there is a slightly higher chance of lifelong neuropsychiatric problems including ASD (Bilder et al. 2017). If PKU is identified early, the child will have fewer problems. Newborn screening in all US states and Canada detects PKU; however, children born in areas that do not have newborn screening might still be evaluated for this disorder (Baieli et al. 2003).

As in phenylketonuria, an increasing number of metabolic disorders have interventions that improve outcome. While the following are not associated with ASD, they are causes of developmental disability that have treatments that can change outcome if identified on newborn screening. Children with galactosemia, a disorder of sugar metabolism, if untreated will develop cataracts, kidney failure, liver failure, and brain damage. Galactosemia can be treated with dietary elimination of galactose. Children with biotinidase deficiency can have variable presentation but, if left untreated, can have developmental delays, vision or hearing loss, seizures, ataxia, hair loss, and/or candidiasis (a fungal infection). This is treated in some cases with oral biotin and avoiding raw eggs, which decrease absorption of biotin. Tyrosinemia is another disorder of protein metabolism that causes intellectual disability and kidney and liver damage. It is treated with low-protein diet and liver transplant if needed. The most severe form of the organic aciduria, maple syrup urine disease, causes vomiting, dehydration, hypotonia, seizure, brain injury, pancreatitis, coma, and ultimately death. Symptoms are prevented with dietary modification. Other inborn errors may be seen in children with nonspecific symptoms, and many are without specific treatments to date. The disorders included in newborn screening programs are ones that are not as rare and have interventions that prevent symptoms.

Mitochondrial disorders occur when there are mutations in the maternally derived mitochondrial genes. Mitochondria are small organelles that are involved in producing energy in each cell of the body. These organelles contain their own genetic code that is separate from the genetic code in the nucleus. The mitochondria are contained in the egg, but not in the sperm, so all mitochondria

and their DNA are inherited from the mother. However, the mother might have fewer and less intense symptoms, or possibly more symptoms, depending on the number of functional mitochondria that are inherited. These disorders are rare in the general population and no screening tests are accurate. It has been suggested that there is an increased rate of mitochondrial abnormalities among children with ASD. Metabolic findings on laboratory testing could be causative or may be secondary to other causes of the symptoms of ASD. Mitochondrial dysfunctions result in increased oxidative stress and changes in energy metabolism. Symptoms suggestive of mitochondrial disorders are excessive fatigue, severe hypotonia, cyclic vomiting, visual problems, hearing problems, poor growth, and/or seizures. Specialized testing including muscle biopsy may be necessary to identify mitochondrial disorders. Specialists in metabolic genetics or child neurology are consulted.

Treatment Goals

Metabolic testing includes an array of specific biochemical analyses ordered as part of a medical workup of an individual with a history and physical examination suggesting evaluation of a specific category of metabolic disorders.

The goal of the metabolic testing is to identify a specific inborn error of metabolism. Some of these inborn errors of metabolism have identified dietary or vitamin treatments that can help limit or improve the symptoms. Many inborn errors of metabolism do not have a known effective treatment. New therapies are under study for many disorders that range from dietary modification, pharmacologic replacement of enzyme, and gene therapy. Diagnosis of an inborn error of metabolism helps families understand the etiology of the developmental disability or other symptoms present in the child and allows for counseling regarding heritability. Specific treatment interventions may be recommended.

Metabolic testing is not routinely done to evaluate the nutritional status of children with limited diets.

Clinical Uses

The level of metabolic testing indicated varies from child to child and situation to situation.

All children should have some form of newborn screening to identify the most common and most treatable types of inborn errors of metabolism.

Children diagnosed with ASD should have a thorough history and physical exam done by a medical professional with experience in autism spectrum disorders. Additional testing for metabolic disorders is not required in every child with an autism spectrum disorder (Myers 2007; Campistol et al. 2016). Children with a history of unusual regression later than age 3 or regression with illness, weakness unexplained by other causes, organomegaly or other evidence of storage disorders, significant hearing impairment, eye findings consistent with a metabolic disorder, or other findings suggestive of a specific etiology should be considered for metabolic evaluation. Children with autism and history of severe seizures especially those resistant to treatment may also be considered for underlying metabolic etiologies. Unusual gastrointestinal disturbances, especially cyclic vomiting or frequent vomiting with mild illness, may indicate metabolic disorder. Children with prominent hypotonia, ataxia, movement disorders, or motor regression may also be considered for metabolic testing based on history and physical examination.

For many of these children, referral to a geneticist or metabolic expert is the most efficient way to focus the metabolic evaluation. For most patients, a tiered level of testing is done. The least invasive tests indicated by the symptoms that are most likely to give information are ordered first and then work up based on the history to more invasive testing like skin or muscle biopsy.

See Also

- Inborn Errors of Metabolism
- Medical Evaluation in Autism

References and Reading

- Arnold, G. L., Hyman, S. L., Mooney, R. A., & Kirby, R. S. (2003). Plasma amino acids profiles in children with autism: Potential risk of nutritional deficiencies. *Journal of Autism and Developmental Disorders*, 33(4), 449–454.
- Baieli, S., Pavone, L., Meli, C., Fiumara, A., & Coleman, M. (2003). Autism and phenylketonuria. *Journal of Autism and Developmental Disorders*, 33(2), 201–204.
- Bilder, D. A., Kober, J. A., Cohen-Pfeffer, J. L., Johnson, E. M., Jurecki, E. R., & Grant, M. L. (2017). Neuropsychiatric comorbidities in adults with phenylketonuria: A retrospective cohort study. *Molecular Genetics and Metabolism*, 121(1), 1–8. <https://doi.org/10.1016/j.ymgme.2017.03.002>. Epub 2017 Mar 6.
- Campistol, J., Díez-Juan, M., Callejón, L., Fernández-De Miguel, A., Casado, M., García Cazorla, A., Lozano, R., & Artuch, R. (2016). Inborn error metabolic screening in individuals with nonsyndromic autism spectrum disorders. *Developmental Medicine and Child Neurology*, 58(8), 842–847. <https://doi.org/10.1111/dmcn.13114>. Epub 2016 Mar 31.
- Johnson, C. P., Myers, S. M., & American Academy of Pediatrics Council on Children with Disabilities. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120, 1183–1215.
- Manzi, B., Loizzo, A. L., Giana, G., & Curatolo, P. (2008). Autism and metabolic diseases. *Journal of Child Neurology*, 23(3), 307–314.
- Myers, S. M., Johnson, C. P., & American Academy of Pediatrics Council on Children With Disabilities. (2007) *Pediatrics*, 120(5):1162–1182. Epub 2007 Oct 29. Review.
- Rossignol, D. A., & Frye, R. E. (2011). Mitochondrial dysfunction in autism spectrum disorders: A systematic review and meta-analysis. *Molecular Psychiatry*, 17(3), 290–314.
- Siddiqui, M. F., Elwell, C., & Johnson, M. H. (2016). Mitochondrial dysfunction in autism spectrum disorders. *Autism Open Access*, 6(5), 1000190.

Metachromatic Leukodystrophy

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Synonyms

MLD; Scholz's disease; Sulfatide lipidosis; Sulfatidosis

Short Description or Definition

Metachromatic leukodystrophy (MLD) is a rare genetic disorder that worsens over time. Symptoms are primarily neurological and related to white matter degeneration, although other organs can also be affected. Psychiatric symptoms often occur. In the early stages, MLD may be confused with autism.

Categorization

Metachromatic leukodystrophy (MLD) is a degenerative white matter disease, which is also classified as a lysosomal sphingolipid storage disease. Given the sudden neurological regression associated with MLD, early stages of this disease can sometimes be confused with autism spectrum disorders, including childhood disintegrative disorder and Rett disorder. As MLD progresses, the pronounced neurological symptoms distinguish it from autism spectrum disorders.

Epidemiology

MLD is classified as rare, with an estimated prevalence ranging from 1:40,000 to 1:100,000 among individuals of European descent. However, higher prevalence has been recorded in several small ethnic groups such as Navajo Indians in the United States and Habbanite Jews and Arabs in Israel.

MLD is caused by a deficiency of arylsulfatase A, a lysosomal enzyme that helps degrade cerebroside sulfate. As a result, sulfatide builds up intracellularly. This occurs primarily in Schwann cells and oligodendrocytes, causing demyelination and decreased white matter.

Over 100 mutations have been identified as a cause of MLD, making it genetically quite heterogeneous. Among Caucasian patients affected by MLD, 2 alleles are common, A and I. Together, these account for roughly 50% of cases. Furthermore, there is a well-documented genotype-phenotype correlation in MLD. Homozygosity for null alleles such as I, which do not yield any enzyme activity whatsoever, always produces the

most severe form of the disease, with late infantile onset and rapid progression. In contrast, homozygosity for alleles such as A that allow low levels of enzyme activity is typically associated with adult onset and gradual progression. Heterozygosity for both types of alleles typically produces an intermediate form of MLD, though there is significant overlap between juvenile- and adult-onset forms.

There are three onset patterns of MLD, described in more detail in the next two sections. Between 40% and 50% of cases are classified as “late infantile,” 30–40% as “juvenile,” and around 20% as “adult.”

Natural History, Prognostic Factors, Outcomes

There are three main forms of MLD, differentiated according to age at onset. Further differentiation can be made according to the pattern of progression and symptoms. Late infantile MLD arises in the first 2 or 3 years of life. Progression is typically rapid; patients succumb to the disease within 5–6 years. In this form, neurological symptoms are usually the first sign, though early stages of MLD can cause behavioral changes such as social regression and withdrawal. In adult-onset MLD, it is more common for psychological and behavioral symptoms to appear first, with neurological impairment appearing slightly later on. Early on, this form of MLD can be confused with schizophrenia, though the emergence of neurological symptoms clearly distinguishes the two. In contrast to late infantile MLD, progression in the adult form is much more gradual. This type of MLD typically emerges early in adulthood, though some cases have described onset as late as 50–60 years of age. Juvenile-onset MLD begins between the ages of 3 and 16, with widely variable progression. Symptoms can develop as rapidly as the late infantile form, yet some patients can survive into early adulthood.

Clinical Expression and Pathophysiology

The ultimate phenomenology of all forms of MLD is thought to result from demyelination of

neurons, triggered by an accumulation of sulfatide in oligodendrocytes, Schwann cells, and neurons. This accumulation is caused by a deficiency of the enzyme arylsulfatase-A, which is responsible for degrading sulfatide. Although the accumulation of sulfatide appears to trigger demyelination, the mechanism is not yet clear.

As mentioned above, MLD has several patterns of onset. Initial symptoms vary both across and within the subtypes of MLD. Here we focus on the early infantile- and juvenile-onset forms, which, when accompanied by a pronounced regression, may initially be difficult to distinguish from regressive ASDs, such as childhood disintegrative disorder and Rett disorder, but also may have subtle enough initial onset patterns to be mistaken for a previously “unrecognized” ASD. Dramatic regressions of the type seen in both childhood disintegrative disorder and Rett disorder are most commonly associated with the late infantile form of MLD. Generally this form of MLD is accompanied by neurological signs, including gait disturbances, and abnormal movement patterns. It can also be accompanied by a more general loss of developmental milestones, including speech, cognition, and self-help skills. Abnormal movement patterns may be interpreted as the hallmark repetitive behaviors of other ASDs. When coupled with developmental losses, these changes may be interpreted as an ASD. However, a careful neurological exam may reveal other changes, e.g., depressed deep tendon reflexes, positive Babinski sign, and muscular hypotonia, not consistent with ASD (although Rett disorder is also accompanied by neurological changes). Early infantile MLD progresses rapidly, however, to severe neurological signs, including spastic quadriplegia and severe dementia, and ultimately death. The juvenile onset of MLD can manifest more subtly in changes in school performance, psychiatric symptoms, etc. Sometimes the first neurological signs can be as subtle as clumsiness and poor coordination. Once again, when subtle, it is easy to imagine that this constellation of symptoms could be interpreted as a previously unidentified ASD. These symptoms may be static for an extended period, sometimes even years. However, a neurological exam may reveal the changes described above. Once the disease begins

to declare itself in clear neurological signs and symptoms, the progression generally becomes more rapid and similar to early infantile MLD.

Evaluation and Differential Diagnosis

The majority of signs and symptoms of MLD are neurological. Thus, a detailed neurological exam is an essential first step in evaluating suspected MLD, or for that matter, any deterioration of adaptive skills. Subtle neurological signs which point to the early stages of MLD may be picked up on exam, even if there are no obvious neurological deficits. The disease can sometimes first be manifested in behavioral changes. Therefore, it is important that any workup for a developmental derailment, as commonly seen in the ASDs, includes MLD on the differential. However, with the progress of MLD, dramatic neurological disturbances are inevitable and will be obvious both clinically and on exam. Neuroimaging, in particular MRI and MRS, may be a powerful tool in establishing a diagnosis. Areas of T2 hyperintensity (see fMRI section), initially in the periventricular areas but spreading from there, may be the first sign. Other findings are common. For a review of this topic, please see Kim et al. (1997) or Gieselmann and Krägeloh-Mann (2010). Laboratory testing is important. Both blood and urine will show evidence of MLD. The level of arylsulfatase enzyme activity can be tested in the blood leukocytes. Elevated urinary excretion of sulfatide over a 24-h period is also a sensitive marker. There are limitations to genetic testing for MLD due to the large number of mutations that can underlie the disease. Prenatal diagnosis of MLD can be done using cells from chorionic villi. The differential diagnosis for MLD includes a number of psychiatric disorders during the early stages, including attention disorders and psychotic disorders. Later, degenerative neurological disorders, including other leukodystrophies such as Krabbe disease, dominate the differential. Most relevant to this discussion, the combination of developmental derailment, including social changes and the loss of adaptive milestones, coupled with subtle neurological signs can suggest an ASD. Any evaluation of new ASD should

include MLD on the differential, with heightened suspicion in cases of regressive ASD.

Treatment

Therapeutic options for MLD are limited. In some cases, hematopoietic stem cell transplantation can be beneficial, but only under a limited set of conditions (see Gieselmann and Krägeloh-Mann 2010). A number of experimental therapies are being explored currently, including enzyme replacement therapy and gene therapy. However, at this point, there is no cure for MLD.

See Also

- ▶ [Childhood Disintegrative Disorder](#)
- ▶ [Magnetic Resonance Imaging](#)
- ▶ [Regression](#)

References and Reading

- Gieselmann, V., & Krägeloh-Mann, I. (2010). Metachromatic leukodystrophy – an update. *Neuropediatrics*, 41, 1–6.
- Kim, T. S., et al. (1997). MR of childhood metachromatic leukodystrophy. *American Journal of Neuroradiology*, 18, 733–738.

Metacognition in Autism

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Definition

“Metacognition” can be thought of as “thinking about thinking.” More specifically, metacognition is characterized as one’s awareness of and ability to regulate one’s own mental states (Flavell 1979).

John Flavell (1979), who originally termed the definition, established a taxonomy of metacognition, distinguishing between “metacognitive knowledge,” an individual’s beliefs and knowledge about cognitions (including declarative knowledge, procedural knowledge, and knowledge about strategy use), and “metacognitive skills,” an individual’s ability to assess and control their own cognitive processes. Since Flavell’s seminal work, researchers have reinterpreted and adjusted Flavell’s original definition. Currently, most researchers agree that metacognitive skills involve reflective processes that monitor and increase the efficiency of underlying cognitive processes in a number of ways. Take for example the various adaptive metacognitive processes involved in completing a study task. Metacognitive skills are required to form a representation of one’s existing learning and comprehension, evaluate the demands of the task and subsequently choose the appropriate strategy for task completion, monitor one’s progress towards the task goal (perhaps adjusting strategy use), and reflect on one’s decisions/performance during and after task completion (Flavell 1979; Lai 2011).

Unsurprisingly then, metacognitive processes are often considered essential for successful learning and a wide range of literature has explored the relation between metacognitive accuracy and learning success. In particular, accurate metacognition seems to be associated with good study skills. Additionally, training metacognitive skills appears to increase academic achievement (e.g., Flavell 1979; Perry et al. 2018; Van der Stel and Veenman 2014).

Moreover, metacognitive abilities are not just important for schooling/academic achievement. Knowing your own thoughts is crucial to accessing therapeutic treatments. A central pillar of common therapies, such as Cognitive-Behavioral Therapy (CBT) and Mindfulness-Based Therapy, is the ability to identify and reflect on one’s own cognitions. Indeed, recent research has suggested metacognitive accuracy may moderate the efficacy of CBT in children (Jones et al. 2019). Additionally, poor awareness of one’s own emotions appears to influence the outcome of psychodynamic therapy in adults (Ogrodniczuk

et al. 2011). As such, impairments in metacognitive skills likely make it difficult to implement psychological therapies successfully. However, this topic remains understudied, and more research is required to understand the relationship between metacognitive abilities and therapeutic efficacy.

Historical Background

The root of the word autism comes from the Greek word “*autos*,” which directly translates as “self.” Historically, autism has been linked to theories surrounding the self, with early theories conceptualizing autism as an extreme form of egocentrism (Bleuler 1950; Kanner 1943). Indeed, Leo Kanner originally “borrowed” the term autism from the work of Bleuler, who had coined it to describe an extreme self-focus and social withdrawal in his patients (with schizophrenia). In contrast, other theories have attributed various autistic behaviors to having an “absent self” (Frith 2003). While it is now well accepted that autistic individuals do not lack a sense of self, a growing body of research suggests that certain aspects of self-representation may pose challenging to autistic individuals.

Current Knowledge

While metacognitive research in the wider literature has been ongoing for 40 years, researchers have only recently begun to explore metacognitive skills in autism. To date, there are only 25 published studies that have investigated metacognition in autism, with somewhat equivocal results concerning autistic metacognitive abilities. While the majority of these studies suggest metacognitive impairments can be seen in autism (e.g., Grainger et al. 2014; Williams et al. 2018), other studies find metacognitive accuracy to be entirely typical (e.g., Grainger et al. 2016), or report mixed results (see Wojcik et al. 2013; Cooper et al. 2016). Such heterogeneous results make it difficult to make strong conclusions surrounding global metacognitive abilities in autism. Mixed

findings within the literature may be associated with methodological issues within the existing literature; firstly, not all existing studies of metacognitive accuracy in autism have matched their autistic group and comparison group for both age and cognitive ability. While matching for baseline variables is essential in all between-group studies of cognition in autism, this is particularly true for studies of metacognition, given the wider literature that suggests both age and cognitive ability strongly correlate with metacognitive accuracy.

Another possible explanation for mixed findings within autism research is that studies exploring metacognition in autism have employed a wide range of metacognitive tasks, as well as self-report measures, across different underlying domains (e.g., meta-memory, meta-perception). It is likely that self-report measures of metacognitive skills do not measure the same underlying processes as explicitly as those assessed using behavioral measures (Craig et al. *in press*). There is often little consistency between self-reported abilities and metacognitive performance on a task, in both nonautistic and autistic adults (see Grainger et al. 2016). Additionally, recent findings suggest that metacognitive skills in autism appear unimpaired, when assessed using implicit measures of metacognition (e.g., Carpenter et al. 2019; Nicholson et al. 2019). This may suggest a dichotomy between implicit and explicit metacognitive skills in autism, though it is important to note that the underlying processes driving performance on implicit tasks in autistic and nonautistic individuals may differ (see Nicholson et al. 2019 for a discussion of this in more detail).

There does appear to be some consistencies across research findings when patterns of metacognitive performance are broken down by specific task type. Generally, studies find that autistic participants are not as accurate as nonautistic participants when judging explicit confidence in their own performance (see Carpenter et al. 2019 for a review). In contrast, on tasks that ask participants to make judgments-of-learning (JOLs), concerning how well they think they have learnt information, we consistently find no evidence of metamemory impairments in autism (see Grainger et al. 2016; Wojcik et al. 2014). Another “classic”

measure of meta-memory (one's abilities to represent one's own memory states) asks participants to make feeling-of-knowing (FOK) judgments. During a typical feeling of knowing task, participants are tested to see if they can recall some recently learned information (e.g., a list of words). After recall ability is assessed, participants are then asked to assess the likelihood that they would know/recognize information (e.g., words) they currently cannot remember (e.g., assessing the likelihood of correctly recognizing words from a recently learned word list, which you currently cannot recall off the top of your head). Both published studies of FOK accuracy in autism report poorer accuracy on an FOK task compared to comparison participants (Grainger et al. 2014; Wojcik et al. 2013), though Wojcik et al. only found impairments when the FOK judgments assessed participants' episodic memory for unrelated word pairs and not when FOK judgments assessed semantic general knowledge of word-picture pairs. Differences in accuracy in autism across confidence judgments, FOK tasks, and JOL tasks are interesting from a theoretical point of view. Within the typically developing literature, researchers have questioned the idea that individuals (autistic or nonautistic) have stable metacognitive abilities that are employed across different tasks, arguing against a general metacognitive ability. These arguments are largely driven by findings that an individual's metacognitive accuracy does not always appear consistent across tasks, nor demonstrate strong test-retest reliability. Importantly, only domain-general accounts of metacognition predict consistent metacognitive accuracy across tasks/domains in autism.

Finally, to date few studies have considered the role co-occurring conditions may have on metacognitive skills in autism. Research suggests that mental health conditions, such as anxiety or depression, are particularly common in autism (see Simonoff et al. 2008). For example, while estimates suggest that ~10% of individuals in the general population demonstrate high levels of alexithymia (a subclinical condition characterized by difficulties identifying and describing emotions), studies suggest that 50% of autistic individuals may meet the criteria for alexithymia (Hill

et al. 2004). It is important to acknowledge high levels of co-occurring conditions in autism, given findings that metacognition is impacted by mental health conditions. Those with high levels of depression, anxiety, and alexithymia have shown significantly weaker reflection and monitoring skills than those with low levels (e.g., Bressi et al. 2017; Fonagy et al. 1996; Ladegaard et al. 2016). High levels of anxiety in particular have been found to be significantly related to poor accuracy when judging how well material has been learned (confidence judgments; Fonagy et al. 1996). To date, it is unclear to what extent weaker performance on metacognitive tasks is related to the presence of additional co-occurring conditions.

Currently, there is not enough evidence to determine which, if any, of these possibilities best explain the pattern of results found in studies of metacognition in autism. As such, it will be important that future research investigates the nature of the underlying processes that autistic individuals use to inform their metacognitive judgments, and whether these processes depend on the types of metacognitive tasks employed.

Future Directions and Implications

It is important to consider the implications of research exploring metacognitive abilities in autism. Crucially, metacognitive skills are trainable; studies suggest that the academic performance of students significantly improves following training in metacognitive skills. In fact, training in metacognition has been shown to improve academic and work place performance across all levels of cognitive ability (e.g., Perry et al. 2018). Thus, the study of metacognition in autism could have important implications for educational practice. Considering the high incidence of co-occurring conditions in autism and the fact that therapies aimed at alleviating mental health difficulties often rely on good metacognitive skills, metacognitive research could also have important implications for autistic mental health and wellbeing. Moreover, recent research has found metacognitive skills in autism to be strongly associated with social skills (Leung

et al. 2016). While the causal direction of this relationship is not yet clear, it does suggest that training in metacognitive skills has the potential to influence social-interaction proficiency in autism. Ultimately, future research in autism should aim to determine a clearer profile of metacognitive strengths and weaknesses. Identifying particular areas of metacognitive strengths and difficulties may inform autistic educational and training practices with an end focus of positively impacting academic and social skills in autism, as well as potentially increasing the tools autistic individuals have to improve their health and well-being.

See Also

- ▶ [Self-Other Distinction and Social Cognition in ASD.](#)
- ▶ [Self-Understanding of Academic Competencies in ASD.](#)

References and Reading

- Bleuler, E. (1950). *Dementia Praecox or the Group of Schizophrenias*. International University Press, New York.
- Bressi, C., Fronza, S., Minacapelli, E., Nocito, E. P., Dipasquale, E., Magri, L., ... Barone, L. (2017). Short-term psychodynamic psychotherapy with Mentalization-based techniques in major depressive disorder patients: Relationship among alexithymia, reflective functioning, and outcome variables – A pilot study. *Psychology and Psychotherapy: Theory, Research and Practice*, 90(3), 299–313. <https://doi.org/10.1111/papt.12110>.
- Carpenter, K. L., Williams, D. M., & Nicholson, T. (2019). Putting your money where your mouth is: Examining metacognition in ASD using post-decision wagering. *Journal of Autism and Developmental Disorders*, 49(10), 4268–4279.
- Cooper, R. A., Plaisted-Grant, K. C., Baron-Cohen, S., & Simons, J. S. (2016). Reality monitoring and metamemory in adults with autism spectrum conditions. *Journal of Autism and Developmental Disorders*, 46(6), 2186–2198.
- Craig, K., Hale, D., Grainger, C., Stewart, M. (in press) Evaluating Metacognitive Self-reports: Systematic reviews of the value of self-report in metacognitive research. *Metacognition and Learning*. <https://doi.org/10.1007/s11409-020-09222-y>.
- Flavell, J. H. (Stanford University). (1979). Metacognition and cognitive monitoring: A new area of cognitive – Developmental inquiry. *American Psychologist*, 34(10), 906–911. <https://doi.org/10.1037/0003-066x.34.10.906>.
- Fonagy, P., Leigh, T., Steele, M., Steele, H., Kennedy, R., Mattoon, G., ... Gerber, A. (1996). The relation of attachment status, psychiatric classification, and response to psychotherapy. *Journal of Counseling and Clinical Psychology*, 64(1), 22–31. <https://doi.org/10.1037/0022-006X.64.1.22>.
- Frith, U. (2003). *Autism: Explaining the enigma*. Malden, MA: Blackwell Pub.
- Grainger, C., Williams, D. M., & Lind, S. E. (2014). Metacognition, metamemory, and mindreading in high-functioning adults with autism spectrum disorder. *Journal of Abnormal Psychology*, 123(3), 650.
- Grainger, C., Williams, D. M., & Lind, S. E. (2016). Judgment of learning accuracy in high-functioning adolescents and adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(11), 3570–3582.
- Hill, E. L., Berthoz, S., & Frith, U. (2004). Brief report: Cognitive processing of own emotions in individuals with autistic spectrum disorder and in their relatives. *Journal of Autism and Developmental Disorders*, 34(2), 229–235.
- Jones, J., Souchay, C., Moulin, C., Reynolds, S., & Adlam, A. L. (2019). Children's CBT skills, metacognition, empathy, and theory of mind. *Journal of Children's Services*, 14(1), 16–26.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Ladegaard, N., Videbech, P., Lysaker, P. H., & Larsen, E. R. (2016). The course of social cognitive and metacognitive ability in depression: Deficit are only partially normalized after full remission of first episode major depression. *British Journal of Clinical Psychology*, 55(3), 269–286. <https://doi.org/10.1111/bjc.12097>.
- Lai, E. R. (2011). Metacognition: A literature review research report. *Research Reports*. <https://doi.org/10.2307/3069464>.
- Leung, R. C., Vogan, V. M., Powell, T. L., Anagnostou, E., & Taylor, M. J. (2016). The role of executive functions in social impairment in Autism Spectrum Disorder. *Child Neuropsychology*, 22(3), 336–344. <https://doi.org/10.1080/09297049.2015.1005066>.
- Nicholson, T., Williams, D. M., Grainger, C., Lind, S. E., & Carruthers, P. (2019). Relationships between implicit and explicit uncertainty monitoring and mindreading: Evidence from autism spectrum disorder. *Consciousness and Cognition*, 70, 11–24.
- Ogrodniczuk, J. S., Piper, W. E., & Joyce, A. S. (2011). Effect of alexithymia on the process and outcome of psychotherapy: A programmatic review. *Psychiatry Research*, 190(1), 43–48.
- Perry, J., Lundie, D., & Golder, G. (2018). Metacognition in schools: What does the literature suggest about the effectiveness of teaching metacognition in schools? *Educational Review*, 1911, 1–18. <https://doi.org/10.1080/00131911.2018.1441127>.
- Simonoff, E., et al. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47, 921–929.

- Van der Stel, M., & Veenman, M. V. J. (2014). Metacognitive skills and intellectual ability of young adolescents: A longitudinal study from a developmental perspective. *European Journal of Psychology of Education*, 29(1), 117–137. <https://doi.org/10.1007/s10212-013-0190-5>.
- Williams, D. M., Bergstr, Z., & Grainger, C. (2018). Metacognitive monitoring and the hypercorrection effect in autism and the general population: Relation to autism (-like) traits and mindreading. *Autism*, 22(3), 259–270. <https://doi.org/10.1177/1362361316680178>.
- Wojcik, D. Z., Moulin, C. J. A., & Souchay, C. (2013). Metamemory in children with autism: Exploring “Feeling-of-knowing” in episodic and semantic memory. *Neuropsychology*, 27(1), 19–27. <https://doi.org/10.1037/a0030526>.
- Wojcik, D. Z., Waterman, A. H., Lestić, C., Moulin, C. J., & Souchay, C. (2014). Metacognitive judgments-of-learning in adolescents with autism spectrum disorder. *Autism*, 18(4), 393–408.

association between metallothionein abnormalities and autism. Despite this lack of support, some scientists and clinicians have attempted to treat autism by increasing metallothionein function. However, no peer-reviewed clinical trials have provided any evidence to support this treatment (Owens et al. 2008).

See Also

- ▶ [Chelation](#)
- ▶ [Hair Analysis](#)
- ▶ [Lead Exposure and Autism](#)
- ▶ [Measles-Mumps-Rubella \(MMR\) Vaccination](#)
- ▶ [Thimerosal](#)
- ▶ [Toxicology](#)
- ▶ [Vaccinations and Autism](#)

Metallothionein

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Definition

Metallothioneins are proteins that bind metals, controlling the levels of these metals in the body and preventing toxic effects (Owens et al. 2008). Some hair and urine analysis studies have shown that levels of heavy metals are increased in children with autism (Owens et al. 2008). Animal studies have demonstrated that mice with dysfunctional metallothioneins are at increased risk for heavy metal (specifically, mercury) poisoning but do not show baseline behavioral sequelae. Additionally, there are natural differences in metallothionein genetics, which may lead to the creation of faulty metallothionein proteins, predisposing an individual to heavy metal sensitivity. The hypothesis that children with autism have faulty metallothionein proteins and are therefore more sensitive to metals from the environment and potentially vaccines has been proposed as an explanation for the cause of autism. However, there are no peer-reviewed studies showing an

References and Reading

- Owens, S. E., Summar, M. L., & Aschner, M. (2008). Metallothionein and autism. In P. Zatta (Ed.), *Metallothioneins in biochemistry and pathology* (pp. 93–116). Singapore: World Scientific.

Metaphoric Language

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Synonyms

[Idiosyncratic language](#)

Definition

Individuals with autism spectrum disorders (ASD) may produce speech that appears to be irrelevant to the current communicative context. For example, Kanner (1946) described a child with ASD who would yell, “Don’t throw the dog off the balcony!” whenever he was about to throw something. His parents reported that several years earlier, they had been staying in a hotel with a balcony and warned

the child not to throw his stuffed toy dog over the railing. Kanner emphasized that this “metaphorical language” was not irrelevant or meaningless. Rather, individuals with ASD attach unique meanings to these “figures of speech” based on specific past experiences.

See Also

- ▶ [Figurative Language](#)
- ▶ [Idiosyncratic Language](#)

References and Reading

- Kanner, L. (1946). Irrelevant and metaphorical language in early infantile autism. *American Journal of Psychiatry*, 103, 242–246.
- Paul, R., & Norbury, C. (2012). *Language disorders from infancy through adolescence: Listening, speaking, reading, writing, and communicating* (4th ed.). St. Louis: Elsevier.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 335–364). Hoboken: Wiley.

Methyl CpG-Binding Protein 2

- ▶ [MECP2 Gene](#)

Methylphenidate

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Synonyms

[Ritalin](#)

Definition

Methylphenidate: Methylphenidate is also a stimulant medication. Although it also enhances release and blocks reuptake of dopamine, it is less potent than the amphetamines. There are many preparations with methylphenidate including immediate-release compound in which the tablet is rapidly absorbed and the duration of action is approximately 4 h. When using the immediate-release compounds, it is likely that the dose will have to be repeated twice or even three times per day. There are also long-acting formulations marketed under several different trade names. These long-acting formulations do not have to be repeated throughout the day. However, various products may only come in particular strengths. Therefore, becoming familiar with the different brands of medications is important for using these long-acting formulations of methylphenidate in the clinic. Methylphenidate also comes in a transdermal skin patch. The skin patch can be worn for 8–9 h per day and replaced with a new patch each day. The skin patch comes in several strengths and may require some trial and error in that the oral dose of methylphenidate may not immediately translate to the dose of the skin patch. One drawback of the current skin patch preparations is their high cost.

The immediate-release formulation of methylphenidate was studied in a large-scale, multisite trial in children with autism spectrum disorders (Research Units on Pediatric Psychopharmacology 2005). The study included three doses of methylphenidate (low to high) and placebo in a crossover trial (each subject received the study treatments in random order under double-blind conditions). All active dose of methylphenidate was superior to placebo in this short-term study. In contrast to the benefits observed in typically developing children with attention deficit/hyperactivity disorder, however, in children with autism spectrum disorders who were hyperactive, impulsive, and distractible, the beneficial effects were modest. The study in children with autism spectrum disorders showed that doses that are well tolerated in typically developing children with attention deficit/hyperactivity disorder were less well tolerated in children with autism spectrum disorders. Intolerable adverse

effects included irritability, insomnia, loss of appetite, and increased stereotypic behavior. These results suggest that when treating children with autism spectrum disorders accompanied by hyperactivity and impulsiveness, the low-to-medium doses are likely to produce modest benefit. However, attempts to achieve a greater benefit by pushing the dose upward are likely to encounter dose-limiting adverse effects.

See Also

► Stimulant Medications

References and Reading

- Martin, A., Scahil, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.
- Research Units on Pediatric Psychopharmacology (RUPP) Autism Network. (2005). Randomized, controlled, crossover trial of methylphenidate in pervasive developmental disorders with hyperactivity. *Archives of General Psychiatry*, 62(11), 1266–1274. [Journal Article].

Mexico and Autism

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Historical Background

Public awareness of autism spectrum disorder (ASD) in Mexico can be traced back to the late 1970s when families affected by ASD began searching for places where their children could receive treatment. Given that fewer options for diagnosis and treatment existed in other states, most families headed to Mexico City. In the early 1970s, children with ASD were often

misdiagnosed and frequently placed in treatment in special education schools for children with other types of disabilities, thereby preventing them from receiving appropriate intervention.

In the following section, we cover the historical background of ASD in Mexico based on the firsthand experience of the second author, Dr. Carlos Marcín. Dr. Marcín has been active in attending to the needs of children and families affected by ASD for over 40 years, both as an academic and as a clinician.

First Phase: 1970–1990

Autism was first conceptualized as a psychiatric disorder by the National Ministry of Health and treated in Mexico City at the “Dr. Juan N. Navarro” Children’s Psychiatric Hospital, the only public institution treating children and adolescents with autism in the country at that time. Treatment offered was medical, from a psychodynamic perspective, which was the main theory in Mexico for explaining autism. Given the growing need for treatment, families organized to form *Centro Educativo Domus*, a nonprofit organization that began offering applied behavior analysis (ABA), which had been successful in the United States in teaching appropriate behaviors to individuals with autism (Lovaas et al. 1974; Lovaas 1987). Another effort worth mentioning was the opening of the *Center for Autism* at Universidad Intercontinental in Mexico City. This center was created with the support of the Japanese government. Dr. Tetsuo Ishi began providing communication-acceptance treatment. In 1983, at the same university, Dr. Marcín led the creation of a Master’s program in autism. Unfortunately, this program closed after graduating only one class.

In 1990, the Mexican Clinic for Autism and Developmental Disorders (Clínica Mexicana de Autismo y Alteraciones del Desarrollo, A.C., *CLIMA*) was founded by Dr. Marcín and a group of families that needed treatment for their children with autism. *CLIMA*, a nonprofit, private organization, began offering evidenced-based treatment for children, providing family counseling, and training professionals. *CLIMA*’s treatment model integrated elements of applied behavior analysis

(ABA), Treatment and Education of Autistic and Related Communication Handicapped Children (TEACCH), Picture Exchange Communication System (PECS), sensory integration therapy, and social skills training. A goal of the intervention was to promote children's educational and social inclusion as early as possible.

In 1990, *CLIMA* organized the first international meeting on autism in Mexico, *Autismo en el Mundo*, *Autismo en México*, with the participation of Dr. Bernard Rimland, from the San Diego Autism Research Institute. This 3-day meeting involved families and professionals and covered diagnostic, etiological, and treatment issues for people with autism. During his interventions, Dr. Rimland also confronted the psychodynamic idea, prominent in Mexico, of the "refrigerator mother" as the cause of autism, offering instead a possible neurodevelopmental explanation.

Second Phase: 2000–Present

During this phase, *CLIMA* has continued working to ensure the provision of evidence-based treatment, offering continuing education opportunities for professionals and families, and participating in research. In 2005, *CLIMA* organized a second conference on *Autismo en el Mundo*, *Autismo en México*. This time, the conference was also supported by Universidad Iberoamericana. This 2-day meeting had the participation of renowned speakers in the field of autism, such as Simon Baron-Cohen, Clara Lajonchere, and Matthew State, who expounded upon the most current research findings concerning genetic factors and theory of mind. In 2007, *CLIMA*, with the support of Autism Speaks and the Carlos Slim Health Foundation, organized a 3-day international meeting, *Autism Speaks to the World (El Autismo habla al Mundo)*. The meeting was free of charge for 2000 participants (families and professionals) and broadcasted online free of charge to about 30,000 more participants through the Mexican telecommunications company, Telmex. The meeting covered topics about early detection, neurological findings, evidence-based practices, genetic findings, learning and communication disorders in autism, and prevalence. Keynote speakers were from different countries and included Geraldine

Dawson, Eric Courchesne, Pat Levitt, Eric Fombonne, Sam Odom, Clara Lajonchere, Helen Tager-Flusberg, Ami Klin, and Andy Shih (United States); Tony Charman (Great Britain); Carlos Marcín (Mexico); and Cecilia Montiel (Venezuela).

An event of special importance in the history of autism in Mexico was the *Pan-American Autism Awareness Training Initiative (PAAATI)*. From 2009 to 2012, yearly meetings and workshops were held. The first two meetings focused on identifying the needs of Mexican and Latin American children with autism and their families in the home setting. Organizations supporting this initiative were *CLIMA*, Autism Speaks, American Pediatrics Association, Carlos Slim Health Foundation, and Miami University. The third meeting, in 2011, focused on family's burden and in training 70 professionals on ABA interventions. The training was given by Deborah Fein from the University of Connecticut and Lynn Cohen Brennan from Boston University. In 2012, the fourth meeting was focused on collecting information from families that led to the development of a *Manual for Intervention at Home* to be delivered to children with autism by their parents. This meeting was led by Wendy Stone, Inger Brooke, and Carlos Marcín. The motivation was principally that the *Manual* could be helpful to Spanish-speaking families in Latin America and the United States. Because of the PAAATI, in 2010, a national early detection campaign was carried out with the support of the Carlos Slim Health Foundation. Using *The Modified Checklist for Autism in Toddlers (M-CHAT)*, adapted to the Mexican culture, children at risk of autism were identified. A workshop for the families of these children was offered so that ABA intervention at home could be provided. The intervention was compiled on a manual, *The Programa de Intervención Temprana*, and distributed among families.

Over the years, nonprofit organizations have opened in many cities across Mexico. Many of them follow *CLIMA's* service model, but others do not clearly state the type of intervention they provide (http://www.educacionespecial.sep.gob.mx/pdf/doctos/6Boletin/Directorio_teleton.pdf).

Legal Issues, Mandates for Service

From 1970 to the mid-1980s, children with disabilities only received services from the Mexican health system, under the auspice of the General Health Law (Ley General de Salud). Services were provided in rehabilitation centers, psychiatric hospitals, and special education schools (in coordination with the educational system). However, the process of inclusion of students with special needs into the general education system began in the late 2000s. Specifically, in the case of autism, inclusion practices came later, perhaps because of the lack of awareness and epidemiological data about autism in Mexican children. A changing moment for the mandate of services to people with autism came on March 30, 2007, when Mexico, along with other countries, signed the Convention on the Rights of Persons with Disabilities (CRPD) at the United Nations (<https://www.un.org/development/desa/disabilities/convention-on-the-rights-of-persons-with-disabilities.html>). The agreement stated a commitment to avoid discriminating against people with disabilities, viewing them as persons with rights and creating the structure to increase inclusive practices at all levels of society. Mexico committed to report to the UN its progress toward this goal with the UN providing feedback about the progress obtained. Mexico's first report in April 2011 focused on the actions taken to provide rehabilitation to people with physical disabilities. In addition, in the 2010 census, data regarding the number of people with disabilities in Mexico began to be collected, indicating that the Mexican population with disabilities was similar to other countries, ranging between 6% and 9%. In this report, ASD was not included.

In 2011, the *General Law for the Inclusion of Persons with Disabilities* was approved by the Mexican Congress. The law stated that the state had to protect and ensure the human rights and fundamental freedoms of people with disabilities with the goal of their full inclusion in society. The law enunciated five disabilities but ignored ASD as one of them. This would provoke a reaction among families and advocates of ASD, who

argued and fought to have ASD included. This reaction resulted in the General Law for the Attention and Protection of People with Autism Spectrum Condition (see Social Policy and Current Controversies below).

In terms of the education of children with autism, it has been reported that, since 1993 when Mexico enacted a federal law to improve the education of people with disabilities, the Secretariat of Public Education (Secretaría de Educación Pública, SEP) has provided special education services to children with autism (Tuman et al. 2010; Harris and Barton 2016). Before 2015, services were provided by two programs, the Centers of Multiple Attention (Centros de Atención Múltiple, CAM) and the Units of Support Services for Regular Education (Unidades de Servicios de Apoyo a la Educación Regular, USAER). From 2015 to the present, services to educate people with disabilities have been provided in inclusive classrooms through the Unit for Special Education and Inclusive Education (Unidad de Educación Especial y Educación Inclusiva, UDEEI). However, most children with autism attending regular schools have a one-on-one aide, typically paid for by the family.

Overview of Current Treatments and Centers

Provision of services in Mexico is not uniform, and little intervention is provided through the National Ministry of Health or public institutions. According to the National Ministry of Health, about 10,000 children receive health services in its institutions, from which an estimate of 2400 diagnosed with ASD receive some level of monthly intervention (www.gob.mx/salud/prensa/139-deteccion-tardia-de-autismo-impide-acion-oportuna-a-estos-infantes). In 2016, the local government in Mexico City opened the *Clinic for Autism* (Clínica de Autismo, www.cdmx.gob.mx/vive-cdmx/post/clinica-de-autismo-de-la-cdmx) that offers diagnostic evaluation and intervention to families of low socioeconomic background.

Additionally, in 2016, the federal government opened the *Regional Center of Ixtapaluca* (Centro Regional de Ixtapaluca) to provide services to children with developmental disabilities and ASD in the State of Mexico. This Regional Center is 1 of 13 centers that provide services to children with neurodevelopmental disorders in the country, but only this one specializes in ASD (www.gob.mx/salud/prensa/nuevo-centro-regional-de-desarrollo-infantil-y-autismo-en-ixtapaluca). Other major public institutions offering services to children with autism are the *Children's Psychiatric Hospital* (www.sap.salud.gob.mx/principales/%C2%BFen-d%C3%B3nde-me-atiendo/hospital-psiqui%C3%A1trico-infantil-dr-juan-n-navarro.aspx), the nonprofit organization *Autismex* (www.autismex.iap.org.mx), and the *Integral Center for Mental Health* (Centro Integral de Salud Mental, Cisame, www.gob.mx/salud/documentos/centro-integral-de-salud-mental-df), which have serviced families of low socioeconomic background. These institutions are in Mexico City.

Despite the services these public institutions offer, most of the intervention for autism has been provided by private clinics or centers that have different approaches and philosophies (e.g., applied behavior analysis to psychoanalysis). Since specific requirements or criteria that these centers must meet are lacking, a wide array of treatment possibilities exists, but the quality of treatment is difficult to ascertain given that information about their intervention models is not readily available to the public. Most service providers have an undergraduate degree in psychology; some have received specific training in ASD at the few available options (see Overview of Training below), but many have been trained as apprentices of senior therapists.

There are a few private institutions that provide evidence-based services to people with autism. Many of them were created by groups of parents with children with autism and the professionals treating them. As mentioned before, *CLIMA*, founded in 1990 by Dr. Marcín and a few parents of children with ASD, is perhaps the most well-known institution to provide integral services to

families affected by ASD in Mexico. It has 20 affiliated clinics across Mexico. *CLIMA* offers comprehensive diagnostic evaluations and a psychoeducational program of 25 h/week. The service model includes the expertise of psychologists, speech/language pathologists, and occupational therapists. Some therapists of *CLIMA* received the introductory training of the Early Start Denver Model (ESDM, Rogers and Dawson 2009) in 2012, with two of them already completed the certification and two more currently in the process of obtaining it. This is relevant considering that in the whole country, there are only five therapists certified in the ESDM according to the most recent list (April 2018) of the University of California Davis, MIND Institute (http://www.ucdmc.ucdavis.edu/mindinstitute/research/esdm/pdf/esdm_certifiedtherapists.pdf). The clinic also offers a program that supervises the inclusion of children in regular schools with the support of a one-on-one aide. The clinic has also participated in research projects (e.g., Hedley et al. 2010; Elsabbagh et al. 2012; Fombonne et al. 2016) and in training of professionals through courses in association with institutions of higher education.

Another well-recognized center is the Domus Autism Institute (*Instituto de Autismo Domus*) that started offering services to families impacted by ASD in the 1980s. *Domus* offers diagnostic evaluations, therapeutic interventions, and educational inclusion as well as services for adults to become as independent as possible (www.institutodomus.org). The third largest private institution, *Centro Autismo Teletón*, opened in 2012 as a foundation supported by business groups and the media in Mexico. *Teletón* has compiled a directory including other small private centers that offer services to children with disabilities, including children with ASD, but their model of services is not publicly described or easily available (www.educacion.especial.sep.gob.mx/pdf/doctos/6Boletin/Directorio_teleton.pdf). Finally, *Iluminemos de Azul* is another nonprofit organization dedicated to creating awareness about autism in society so that people with autism and their families have a better quality of life.

Overview of Research Directions

Research addressing issues related to ASD is limited in Mexico. However, this is not much different from other research areas. In 2012, at the beginning of the current administration, it was promised that 1% of GDP would be invested into research programs and development by the end of June 2018. However, Mexico currently spends only 0.53% of its GDP in supporting research development, compared to the average of 2.38% of the OECD countries and far from the highest investing countries, Israel (4.25%) and Korea (4.23%) (<https://data.oecd.org/rd/gross-domestic-spending-on-r-d.htm>).

Despite limited support for research, several projects have been carried out, and few research programs are currently under way, varying in approach and sub-area of expertise. One of these projects evaluated the validity of using the Autism Detection in Early Childhood (ADEC) in Mexico. Its findings suggested this instrument could be used with confidence to screen for autism (Hedley et al. 2010). Another relevant study, funded by Autism Speaks, determined that the estimated prevalence of ASD in Mexico is 1 in 115 or 0.87% [95% CI (0.62, 1.1%)] (Fombonne et al. 2016). A preceding study, conducted by the same group, validated the use of John Constantino's Social Responsiveness Scale (SRS) with the Mexican population (Fombonne et al. 2012). Albores-Gallo et al. (2012) examined the validity of the M-CHAT (Robins et al. 1999) for use with Mexican population. In addition, Dr. Albores-Gallo, at the Children's Psychiatric Hospital, has a line of research devoted to studying ASD in children and adolescents and has reported the conclusion of six different projects (<https://sites.google.com/site/hpicomisioninvestigacion/desarrollo>). Furthermore, other projects about ASD have been completed within the same institution as a requirement to obtain a medical degree with a specialty in child and adolescent psychiatry (<https://sites.google.com/site/hpicomisioninvestigacion/psiquiatria-infantil-y-de-la-adolescencia>).

More recently, other studies have been published as a sign of an emergence of such

research programs in Mexico. For example, a study found that Mexican pediatricians could improve their knowledge about early signs of ASD in their practice so that proper referrals for comprehensive assessment by specialists could be completed and early intervention started (Zúñiga et al. 2017). This information has been shared with the Ministry of Health so that steps toward training pediatricians might be taken.

Within the educational setting and considering the international trends of inclusion, Sánchez Lizardi and Sohn McCormick (2016) surveyed general education teachers, K-12, to understand what their perspectives and needs were when educating students with ASD in their classrooms. This study found that teachers' primary concern was their lack or limited knowledge about ASD.

Perhaps the most ambitious of research programs is the *Consortium of Genomic Characterization of ASD in Mexican Population*. This consortium was formed in 2016 by Dr. Humberto Nicolini and includes nine academic, private clinics, public hospitals, and research institutions with the purpose of identifying the genes involved in ASD within the Mexican population. Presently, interviews and samples have been collected from a small number of families affected with ASD. Because academic institutions are part of the consortium, doctoral dissertations and Masters' theses are also in progress.

Overview of Training

Presently, there are no unified efforts for the training of teachers or health-related professionals about ASD in Mexico. Some of the centers mentioned above offer a variety of learning activities that range from lectures and short-term courses to 10-month certified courses. In particular, CLIMA offers a 10-month certified course in conjunction with the National Polytechnic Institute (Instituto Politécnico Nacional, IPN). Dr. Marcín has offered this type of certified courses in different states of Mexico and has trained approximately 3250 professionals in addition to offering workshops to about 8000 families in the last 28 years.

Treatment centers have invited international trainers in diagnostic instruments and interventions (e.g., Autismo Puebla www.autismopuebla.org), with professionals across Mexico earning these continuing education credits. Other opportunities for training have been national and international autism conferences, sponsored by different organizations (e.g., Carlos Slim Health Foundation, Health Ministry of Chiapas, and the National System for Integral Family Development of Yucatán). However, it is difficult to ascertain the implications of these learning opportunities on professional practice given that they are not officially regulated.

Social Policy and Current Controversies

Lay (2016) has closely followed the creation and controversies around the law pertinent to people with ASD. In 2014, an initiative to create a General Law for the Attention and Protection of People with Autism Spectrum Disorder was presented to the Mexican House of Representatives. The controversy was related to the term *disorder*, and it arose among families affected by ASD and advocates, who, from a human rights perspective, argued that this should be changed to the term *condition* because it was more socially inclusive and beyond a mere clinical presentation. Thus, on March 5, 2015, the Mexican House of Representatives approved the General Law for the Attention and Protection of People with Autism Spectrum Condition (http://www.diputados.gob.mx/LeyesBiblio/pdf/LGAPPCEA_270516.pdf). The Senate approved it 21 days later, being signed by the president and officially published on April 30, 2015. However, in June 1, 2015, the National Commission of Human Rights asked for an appeal of the law, arguing that some articles violated the International Convention on the Rights of Persons with Disabilities and that they were unconstitutional. After several months, in February of 2016, the Supreme Court declared unconstitutional only one of the articles and validated the remaining parts of the law.

References and Reading

- Albores-Gallo, L., Roldán-Ceballos, O., Villarreal-Valdes, G., Betanzos-Cruz, B.X., Santos-Sánchez, C., Martínez-Jaime, M.M., . . . Hilton, C.L. (2012). M-CHAT Mexican version validity and reliability and some cultural considerations. *International Scholarly Research Notices Neurology*, 2012, article ID 408694, 7 pages. <https://doi.org/10.5402/2012/408694>
- Clínica Mexicana de Autismo y Alteraciones del Desarrollo, A.C., (CLIMA) (n.d.). <http://www.clima.org.mx/>.
- Directory of centers for the attention of ASD (n.d.) (http://www.educacionespecial.sep.gob.mx/pdf/doctos/6Boletin/Directorio_telefon.pdf).
- Elsabbagh, M., Divan, G., Koh, Y.J., Kim, Y.S., Kauchali, S., Marcín, C., . . . Fombonne, E. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research*, 5, 160–179. <https://doi.org/10.1002/aur.239>.
- Fombonne, E., Marcin, C., Bruno, R., Manero-Tinoco, C., & Diaz-Marquez, C. (2012). Screening for autism in Mexico. *Autism Research*, 5, 180–189. <https://doi.org/10.1002/aur.1235>.
- Fombonne, E., Marcin, C., Manero, A. C., Bruno, R., Diaz, C., Villalobos, M., Ramsay, K., & Nealy, B. (2016). Prevalence of autism spectrum disorders in Guanajuato, Mexico: The Leon survey. *Journal of Autism and Developmental Disabilities*, online version. <https://doi.org/10.1007/s10803-016-2696-6>.
- Harris, B., & Barton, E. E. (2016). Autism services in Mexico: A qualitative survey of education professionals. *International Journal of School and Educational Psychology*, 5(1), 1–13. <https://doi.org/10.1080/21683603.2016.1155514>.
- Hedley, D., Young, R., Juarez-Gallegos, M. A., & Marcin-Salazar, C. (2010). Cross-cultural evaluation of the autism detection I early childhood (ADEC) in Mexico. *Autism*, 14(2), 93–112. <https://doi.org/10.1177/1362361309347676>.
- Instituto de Autismo Domus [Domus Autism Institute] (n.d.) <http://www.institutodomus.org/>.
- Lay, T. (2016). *Políticas públicas, intervención y tecnología. Enfoques multidisciplinarios en la atención a la Condición del Espectro Autista*. México: Universidad de Guadalajara.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- Lovaas, O. I., Schreibman, L., & Koegel, R. L. (1974). A behavior modification approach to the treatment of autistic children. *Journal of Autism and Childhood Schizophrenia*, 4, 111–129.
- México (2015). *Ley General para la Atención y Protección a Personas con la Condición del Espectro Autista* [General Law for the Attention and Protection of People with Autism Spectrum Condition]. Retrieved from

http://www.diputados.gob.mx/LeyesBiblio/pdf/LGAPPCEA_270516.pdf.

Organisation for Economic Co-operation and Development (OCDE) (n.d.). Gross domestic spending on R&D. Retrieved on February 26, 2018 from <https://data.oecd.org/rd/gross-domestic-spending-on-r-d.htm>.

Robins, D. L., Fein, D., & Barton, M. (1999). The Modified Checklist for Autism in Toddlers (M-CHAT). *Self-published*.

Rogers, S. J., & Dawson, G. (2009). *Early start Denver model for young children with autism: Promoting language, learning, and engagement*. New York: Guilford Press.

Sánchez Lizardi, P., & Sohn McCormick, A. (2016). *Educational inclusion of students with autism in Mexico: Trends and needs*. Presentation at the 38th Annual Conference of the International School Psychology Association (Amsterdam, The Netherlands).

Tuman, J. P., Roth-Johnson, D., Baker, D. L., & Vecchio, J. (2010). Autism and special education policy in Mexico. *Global Health Governance*, 2(1), 1 Available at SSRN: <https://ssrn.com/abstract=1578963>.

Zúñiga, S.A.E., Sánchez Lizardi, P., Romero, D.L.L., Pego, M.A. (2017). *Mexican pediatricians' knowledge of autism spectrum disorder: Implications for early identification and intervention*. Poster presented at the International Meeting for Autism Research. (San Francisco).

M-FUN

► Miller Function and Participation Scales

MI (Metacognition Index)

► BRIEF (Behavior Rating Inventory of Executive Functions)

Microcephalic

► Microcephaly

Microcephalus

► Microcephaly

Microcephaly

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Synonyms

Microcephalic; Microcephalus; Nanocephaly

Definition

The term “microcephaly” is used to indicate a head circumference (HC) with two standard deviations (SD) below that expected for age, sex, and race. Some authors consider microcephaly when the HC is 3 SD below. A small skull suggests, in most cases, a small brain. Given that 55% of the brain is cortex, a small brain means less cerebral cortex, and it is associated with intellectual disability in approximately 30–50% of the cases. We distinguish two forms of microcephaly: primary and secondary. When it appears before the first 32 weeks of gestation, it is considered as primary microcephaly and when it appears after birth, it is considered as secondary microcephaly. Most neurons are generated during the first 21 weeks of gestation, and neuronal connections are generated after birth. Therefore, in cases of primary microcephaly, there is often a lack of neurons, whereas in the secondary cases, the initial neuronal production is usually normal, but few neural connections are produced, or there is subsequent neuronal death. The causes of both primary and secondary microcephaly may be genetic, secondary to prenatal infections or metabolic diseases, among others.

References and Reading

- Abuelo, D. (2007). Microcephaly syndromes. *Seminars in Pediatric Neurology*, 14, 118–127.
- Ashwal, S., Michelson, D., Plawner, L., & Dobyns, W. (2009). Practice parameter: Evaluation of the child with microcephaly (an evidence-based of Neurology

and the Practice Committee of the Child Neurology Society review): Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. *Neurology*, 73, 887–897.

Dolk, H. (1991). The predictive value of microcephaly during the first year of life for mental retardation at seven years. *Developmental Medicine & Child Neurology*, 33, 974–983.

Mochida, G. H., & Walsh, C. A. (2001). Molecular genetics of human microcephaly. *Current Opinion in Neurology*, 14(2), 151–156.

Woods, C. G. (2004). Human microcephaly. *Current Opinion in Neurobiology*, 14, 112–117.

Micrographia

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Definition

Micrographia is abnormally small handwriting or handwriting that becomes progressively smaller and harder to read. Micrographia is a common feature of Parkinson's disease, possibly reflecting difficulties with scaling and controlling the amplitude of movement (Rao et al. 2003). Micrographia, however, can develop as a sign of focal neurological lesions that disrupt the cortico-subcortical loop involving the putamen, thalamus, premotor cortex, and sensorimotor cortex (Denes et al. 2005; Kim et al. 1998). Difficulties in handwriting have been documented in children with autism spectrum disorder including issues with legibility (Kushki et al. 2011), letter formation (Fuentes et al. 2009), and macrographia (Beversdorf et al. 2001; Johnson et al. 2013), but not micrographia.

See Also

- ▶ Apraxia
- ▶ Gross Motor Skills
- ▶ Macrographia

References and Reading

- Beversdorf, D. Q., Anderson, J. M., Manning, S. E., Anderson, S. L., Nordgren, R. E., Felopulos, G. J., et al. (2001). Brief report: Macrographia in high-functioning adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 31(1), 97–101.
- Denes, G., Signorini, M., & Volpato, C. (2005). Post graphemic impairments of writing: The case of micrographia. *Neurocase*, 11(3), 176–181.
- Fuentes, C. T., Mostofsky, S. H., & Bastian, A. J. (2009). Children with autism show specific handwriting impairments. *Neurology*, 73(19), 1532–1537.
- Johnson, B. P., Phillips, J. G., Papadopoulos, N., Fielding, J., Tonge, B., & Rinehart, N. J. (2013). Understanding macrographia in children with autism spectrum disorders. *Research in developmental disabilities*, 34(9), 2917–2926.
- Kim, J. S., Im, J. H., Kwon, S. U., Kang, J. H., & Lee, M. C. (1998). Micrographia after thalamomesencephalic infarction: Evidence of striatal dopaminergic hypofunction. *Neurology*, 51(2), 625–627.
- Kushki, A., Chau, T., & Anagnostou, E. (2011). *Handwriting difficulties in children with autism spectrum disorders: A scoping review*. *Journal of Autism & Developmental Disorders*, 41(12), 1706–1716. <http://dx.doi.org/10.1007/s10803-011-1206-0>.
- Rao, G., Fisch, L., Srinivasan, S., D'Amico, F., Okada, T., Eaton, C., et al. (2003). Does this patient have Parkinson disease? *Journal of the American Medical Association*, 289(3), 347–353.

Midazolam

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Synonyms

[Dormicum](#); [Versed](#)

Definition

With increasing numbers of Autism Spectrum Disorders (ASD) children, clinicians have investigated alternative pharmacological and behavioral interventions to minimize perioperative problems among ASD children

(Taghizadeh et al. 2015). Currently, clonidine, dexmedetomidine, midazolam, and ketamine remain the first-line premedication therapies for ASD patients undergoing surgery (Taghizadeh et al. 2015). Midazolam is a benzodiazepine medication used for *anesthesia, procedural sedation, trouble sleeping, and severe agitation*. Several case reports showed midazolam is an effective premedication for mild ASD cases and exhibits comparable efficacy to other administered benzodiazepines (Capp et al. 2010; Jo et al. 2017; Pisalchaiyong et al. 2005; Seo et al. 2014; Shah et al. 2009; Van Der Walt and Moran 2001). However, ASD patients administered midazolam experienced several side effects including: “emergence phenomenon, disorientation, sensory and perceptual illusions and vivid dreams in recovery, nausea and vomiting, nystagmus, hypersalivation, and occasionally laryngospasm” (Taghizadeh et al. 2015). Furthermore, one study showed 30% of ASD patients premedicated with midazolam remained uncooperative before surgery (Van Der Walt and Moran 2001). Thus, current guidelines suggest clinicians incorporate parents and simple behavioral strategies, such as providing comfort items, along with premedication to reduce anxiety and discomfort among ASD children before surgery. Further research is required to determine which combination of medication and behavioral techniques produce optimal outcomes among ASD children undergoing surgical operations

See Also

- Neurontin (gabapentine)
- Zyprexa

References and Reading

Capp, P. L., de Faria, M. E. J., Siqueira, S. R. D. T., Cillo, M. T. P., Prado, E. G. B., & de Siqueira, J. T. T. (2010). Special care dentistry: Midazolam conscious sedation for patients with neurological diseases. *European Journal of Pediatric Dentistry*, 11(4), 162–164.

- Jo, C.-W., Park, C.-H., Lee, J.-H., & Kim, J.-H. (2017). Managing the behavior of a patient with autism by sedation via submucosal route during dental treatment. *Journal of Dental Anesthesia and Pain Medicine*, 17(2), 157. <https://doi.org/10.17245/jdamp.2017.17.2.157>.
- Pisalchaiyong, T., Trairatvorakul, C., Jirakijja, J., & Yuktarnonda, W. (2005). Comparison of the effectiveness of oral diazepam and midazolam for the sedation of autistic patients during dental treatment. *Pediatric Dentistry*, 27(3), 198–206.
- Seo, K. H., Jung, H. S., Kang, E. G., Kim, C. J., Rhee, H. Y., & Jeon, Y. S. (2014). Sedation using 5% lidocaine patches, midazolam and propofol in a combative, obese adolescent with severe autistic disorder undergoing brain magnetic resonance imaging: a case report. *Korean Journal of Anesthesiology*, 67(6), 421. <https://doi.org/10.4097/kjae.2014.67.6.421>.
- Shah, S., Shah, S., Apuya, J., Gopalakrishnan, S., & Martin, T. (2009). Combination of oral ketamine and midazolam as a premedication for a severely autistic and combative patient. *Journal of Anesthesia*, 23(1), 126–128. <https://doi.org/10.1007/s00540-008-0685-4>.
- Taghizadeh, N., Davidson, A., Williams, K., & Story, D. (2015). Autism spectrum disorder (ASD) and its perioperative management. *Pediatric Anesthesia*, 25(11), 1076–1084. <https://doi.org/10.1111/pan.12732>.
- Van Der Walt, J. H., & Moran, C. (2001). An audit of perioperative management of autistic children. *Pediatric Anesthesia*, 11(4), 401–408. <https://doi.org/10.1046/j.1460-9592.2001.00688.x>.

Midbrain

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Synonyms

Mesencephalon

Definition

The midbrain is a component of the brain stem that plays a role in a number of central nervous system functions. It contains the substantia nigra, which has an important role in motor function. It also plays a role in vision, hearing, and arousal. Gaffney et al. (1998) found that the entire brain

stem to be significantly smaller in a group with autism. Other groups have replicated the finding and extended it to the midbrain in particular. Hashimoto et al. (1992) found that the midbrain and pons (another component of the brain stem) were significantly smaller in a group with autism in comparison to a control group. Furthermore, several studies have found functional abnormalities in the brain stems of subjects with autism (Maziade et al. 2000; Thivierge et al. 1990).

See Also

► [Midbrain Raphe](#)

References and Reading

- Gaffney, G., Kuperman, S., Tsai, L., & Minchin, S. (1998). Morphological evidence for brainstem involvement in infantile autism. *Biological Psychiatry*, 24, 578–586.
- Hashimoto, T., Tayama, M., Mori, K., et al. (1986). Short latency somatosensory evoked potentials in children with autism. *Brain & Development*, 8, 428–432.
- Hashimoto, T., Tayama, M., Miyazaki, M., Sakurama, N., Yoshimoto, T., Murakawa, K., et al. (1992). Reduced brainstem size in children with autism. *Brain & Development*, 14(2), 94–97.
- Maziade, M., Chantal, M., Cayer, M., Roy, M., Szatmari, P., Côté, R., et al. (2000). Prolongation of brainstem auditory-evoked responses in autistic probands and their unaffected relatives. *Archives of General Psychiatry*, 57, 1077–1083.
- Thivierge, J., Bédard, C., Côté, R., & Maziade, M. (1990). Brainstem auditory evoked response and subcortical abnormalities in autism. *The American Journal of Psychiatry*, 147, 1609–1613.

Midbrain Raphe

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Synonyms

[Midbrain raphe nuclei](#); [Nuclei of raphe](#); [Raphe nuclei](#)

Definition

“Midbrain raphe” refers to two nuclei found in the reticular formation of the midbrain: the superior central nucleus and dorsal raphe nucleus. Other raphe nuclei are also found in the medulla and pontine reticular formations. The common element between all raphe nuclei is that they form a seam, or ridge, along the medial aspect of the brain stem, making up the most medial aspect of the reticular formation, a phylogenetically ancient part of the brain. The primary purpose of all raphe nuclei appears to be to release the neurotransmitter serotonin to other areas of the brain, including areas responsible for social information processing. They are also thought to play a fundamental role in regulating circadian rhythm (For a discussion of the role of serotonin in autism, please see the *serotonin* section of the encyclopedia. For a more general discussion of the midbrain, please see the *midbrain* section of the encyclopedia.)

See Also

► [Midbrain](#)
► [Serotonin](#)

References and Reading

- Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (2000). *Principles of neural science* (4th ed., p. 324). New York: McGraw-Hill.

Midbrain Raphe Nuclei

► [Midbrain Raphe](#)

Middle Ear Infection

► [Otitis Media](#)

Milestone

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Synonyms

Developmental milestones; Indicator; Marker

Definition

Skills that are indicative of development. Examples are taking a first step, smiling for the first time, and waving “bye-bye.” Children reach milestones in how they play, speak, learn, behave, and move (Center for Disease Control [CDC] 2010). There is a standard pattern and age range for how and when most children achieve these milestones. Children with autism are typically delayed in their acquisition of communicative and social developmental milestones and also in their development of skills such as using eating utensils, drawing, dancing, rhythm, and music (Jones and Prior 1985).

See Also

► Developmental Milestones

References and Reading

- Center for Disease Control (CDC). 2010. *Developmental milestones*. <http://www.cdc.gov/ncbddd/actearly/index.html>
- Jones, V., & Prior, M. (1985). Motor imitation abilities and neurological signs in autistic children. *Journal of Autism and Developmental Disorders*, 15(1), 37–46.

Milestones

► Objective

Milieu Teaching

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Definition

Milieu teaching (Hart and Risley 1975) includes a group of procedures, derived from the behaviorist tradition, that were developed to teach language skills to children by embedding learning opportunities within the child’s everyday (i.e., natural) environment and by taking advantage of a child’s interest in and motivation to gain access to materials. According to Goldstein (2002), incidental teaching represents the key component of milieu teaching. Incidental teaching episodes begin with spontaneous child-initiated communication acts as the child attempts to gain access to preferred materials, objects, or events within the natural environment. These child communication acts serve the pragmatic function of requesting (or manding). In addition to incidental teaching, two other well-known milieu language teaching procedures (described below) are mand model and time delay.

Because they are embedded in the natural environment, milieu language teaching procedures are often taught to parents who can then incorporate milieu language procedures into the child’s everyday routines and activities.

There are now many variants of the milieu teaching approach including prelinguistic milieu teaching, which focuses on teaching preverbal children to use nonverbal communication skills such as gestures, eye gaze, and vocalization; and enhanced milieu teaching, a more comprehensive approach to naturalistic language intervention, that includes environmental arrangement, responsive interaction techniques, and milieu teaching procedures.

According to Gilbert (2008), the following components are characteristic of milieu teaching:

1. Training in everyday environments
2. Arranging the environment to promote spontaneous requesting by the child

3. Offering preferred toys and activities to reinforce child communication
4. Building predictable turn-taking routines
5. Use of time delay (i.e., expectant waiting) to encourage communication
6. Waiting for the child to initiate by gesturing or indicating interest in an object or activity
7. Providing models of more advanced communication following a child initiation
8. Rewarding communication with access to the desired object/activity rather than unrelated reinforcers

Historical Background

Classic behavioral approaches to early language intervention made use of imitation procedures and tangible rewards for imitation. These methods have several drawbacks including communication that is passive or prompt dependent or which does not generalize to contexts outside of those in which the communicative behavior was taught. In addition, children who have not yet acquired the understanding that communicative bids can be used to affect the behavior of other people (i.e., who do not understand the instrumental function of communication as a means to a desired end) may have difficulty acquiring basic communication skills.

In response to these drawbacks of discrete trial interventions, behavioral interventionists began to develop methods that were based upon the strengths of behavioral programming (e.g., predictable structure, use of task analysis, attention to antecedents and consequences of behavior). These methods addressed the need to establish basic communicative behavior and to generalize communicative acts beyond the training setting. These developments also included the use of the natural environment as the context for learning, planning of contingencies carefully engineered to elicit spontaneous communicative acts that could be shaped toward more conventional and functional language, and the inclusion of parents as intervention agents.

Rationale or Underlying Theory

The rationale that led to the emergence of milieu teaching approaches is that it is important for children to learn to communicate within the everyday context within which their communication behaviors will be used. In addition, by arranging the environment to provide objects, actions, and events in which children have an interest, the child will be motivated to direct a request for access to the desired object or activity to the communicative partner who can then model more advanced and conventional forms of communication.

Goals and Objectives

The broad goal of milieu teaching is to increase the child's use of specific requesting behaviors. Related goals include teaching related grammatical forms and vocabulary. According to Hancock and Kaiser (2007), milieu teaching emphasizes reciprocity, turn taking, following the child's lead, meaningful and contingent feedback for child communication, and expansion of child utterances to model more advanced forms.

Treatment Participants

Responsive education/prelinguistic milieu teaching is designed for children at developmental levels between 12 and 18 months who have not yet become clear and frequent communicators. If children use more than 10 words or signs, or understand more than 75 words, then they should be enrolled in an intervention that places more of an emphasis on expressive vocabulary. Children for whom RE/PMT is appropriate should produce less than two acts of intentional communication per minute during play interactions with a caregiver.

Children enrolled in enhanced milieu teaching should be verbally imitative, have at least 10 expressive vocabulary words, and should have a mean utterance length of 1.0–3.5 words.

Treatment Procedures

Incidental teaching, time delay, and mand modeling have been identified as major components of the milieu language teaching procedure.

In the mand-model procedure, the teacher, clinician, or parent arranges the environment to include objects/actions/events that the child is motivated to request access to. When the child approaches the materials, the adult provides a mand, asking: "What do you want?" If this question elicits the child's production of a word or word approximation, the adult expands by providing a semantic or grammatically more advanced version of the child's production. If the child does not respond to the mand, the clinician provides a model: Say, I want truck. If the child does not respond, the teacher provides one more model and provides the child with access to the desired material.

The time delay procedure is a prompt fading strategy used to encourage children to use vocabulary that they do know, but may not frequently produce spontaneously. Time delay is similar to "fill in the blank." Again, desired materials are available to the child. The teacher holds an item (e.g., a puzzle piece of a cow) and says, "I want the _____."

Incidental teaching is a technique in which the adult uses child initiations during ongoing activities to provide language input. Incidental teaching requires that the environment be arranged to include materials and activities that are highly motivating to the child such that the child will initiate to gain access to the materials. Incidental teaching is child-directed in that the adult follows the child's lead and allows the child's current focus of interest to guide the teaching episodes.

Efficacy Information

According to the American Speech-Language-Hearing Association, there is a large body of empirical support for more contemporary behavioral approaches using naturalistic teaching methods that demonstrate efficacy for teaching not only speech and language but also

communication. The following specific intervention strategies have been found to promote initiation and generalization: arrange the environment to provide opportunities for communicating with preferred materials, encourage child initiations and follow the child's attentional focus and interest, intersperse preferred and nonpreferred activities, use embedded instruction in the natural environment, offer choices and encourage choice making, use natural reinforcers that follow what the child is trying to communicate, and use of time delay.

There are only a few studies, all using single-subject design, that have compared traditional discrete trial with naturalistic behavioral approaches. These studies have reported that naturalistic approaches are more effective at leading to generalization of language gains to natural contexts.

Outcome Measurement

Outcome measures used in studies of milieu teaching will typically consist of measures of the rate or frequency of child nonverbal or verbal communication acts, measures of utterance length (mean length of utterance in words), measures of use of grammatical morphemes (mean length of utterance in morphemes), and vocabulary use (lexical diversity, number of different words). Other measures may include parent use of targeted strategies or number of milieu teaching episodes.

Qualifications of Treatment Providers

Individuals who teach parents must have skills in the delivery of milieu teaching procedures but must also have skills in interacting with parents in ways that support parent learning. Professionals must have skills in providing coaching and feedback to parents and must be able to model desired parent behaviors within the context of a parent-child interaction without disrupting the interaction or overwhelming the parent.

See Also

- [MacArthur-Bates Communicative Development Inventories, Second Edition](#)

References and Reading

- American Speech-Language-Hearing Association. (2006). *Guidelines for speech-language pathologists in diagnosis, assessment, and treatment of autism spectrum disorders across the life span [Guidelines]*. Available from www.asha.org/policy.
- Gilbert, K. (2008). Milieu communication training for late talkers. *Perspectives on Language Learning and Education*, 15, 112–118.
- Hart, B. M., & Risley, T. R. (1975). Incidental teaching of language in the preschool. *Journal of Applied Behavior Analysis*, 8, 411–420.
- Hart, B., & Risley, T. R. (1980). In vivo language intervention: Unanticipated general effects. *Journal of Applied Behavior Analysis*, 13, 407–432.
- Hart, B., & Rogers-Warren, A. (1978). A milieu approach to teaching language. In R. Schiefelbusch (Ed.), *Language intervention strategies* (pp. 193–235). Baltimore: University Park Press.
- Hemmeter, M. L., & Kaiser, A. P. (1994). Enhanced milieu teaching: Effects of parent-implemented language intervention. *Journal of Early Intervention*, 18, 269–289.
- Kaiser, A. P. (1993). Parent-implemented language intervention: An environmental perspective. In A. P. Kaiser & D. B. Gray (Eds.), *Enhancing children's communication: Research foundations for intervention* (pp. 63–84). Baltimore: Paul H. Brookes.
- Kaiser, A., & Trent, A. (2007). Communication intervention for young children with disabilities: Naturalistic approaches to promoting development. In S. Odom, R. Horner, M. Snell, & J. Blacher (Eds.), *Handbook of developmental disabilities* (pp. 224–246). New York: Guilford Press.
- Kaiser, A. P., Yoder, P. J., & Keetz, A. (1992). Evaluating milieu teaching. In S. F. Warren & J. Reichle (Eds.), *Causes and effects in communication and language intervention* (pp. 9–47). Baltimore: Paul H. Brookes.
- Kaiser, A. P., Hancock, T. B., & Nietfeld, J. P. (2000). The effects of parent-implemented enhanced milieu teaching on the social communication of children who have autism. *Early Education & Development*, 11, 423–446.
- Warren, S. F., & Gazdag, G. (1990). Facilitating early language development with milieu intervention procedures. *Journal of Early Intervention*, 14, 62–86.
- Warren, S. F., & Kaiser, A. P. (1986). Incidental language teaching: A critical review. *Journal of Speech and Hearing Disorders*, 51, 291–299.
- Yoder, P. J., Warren, S. F., Kim, K., & Gazdag, G. E. (1994). Facilitating prelinguistic communication skills in young children with developmental delay II: Systematic replication and extension. *Journal of Speech and Hearing Research*, 37, 841–851.

Military Families with Children with Autism

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Definition

Military families with children with autism are a subgroup of families with children with autism who have at least one member serving in the Armed Forces. The service members of military families may enlist with any one of several branches of the Armed Forces including the Air Force, Army, Coast Guard (during national emergencies or wartime situations), Marine Corps, and Navy (Blaisure et al. 2012). The service members of military families may be active duty, reserve, or National Guard members. Active duty members are full-time service men and women, which also requires their families to be full-time military families. Reserve and National Guard members are part-time forces, trained and ready if they are called upon to serve under command of the President or state governor, respectively (Blaisure et al. 2012). Since Reservists and National Guard members are part-time, their families are also part-time military families, frequently living in civilian communities with limited access to military supports if their service member is called for duty.

Since military families with children with autism are members of both the autism community and the military community, they have shared characteristics with both groups. For example, as members of the autism community, military families with children with autism share the experience of having a child with autism, service availability issues, and additional stress in the family. Children with autism may have a different constellation of symptoms from two categories – social interaction and communication and restricted and/or repetitive behavior (APA 2013). These characteristics may include difficulty with social interaction, insistence on sameness,

restricted interests, and limited and/or delayed communication (Barbaro and Dissanayake 2009), requiring intervention from several different professionals. Parents of children with autism have long reported difficulty finding the services their child with autism needs to improve a variety of impairments (Kohler 1999). As a result of challenging behavior from the child with autism, difficulty finding services, and other stressors, parents of children with autism have reported higher levels of stress than parents of neurotypical (e.g., typically developing) children or children with other developmental disabilities (Hayes and Watson 2013).

As members of the military community, military families with children with autism also share a common set of characteristics with military families, distinguishing them from civilian families. Segal (1986) described these characteristics as family separations (e.g., deployment), frequent relocations to military installations around the United States or overseas, and dangerous work environments. Some civilian families may experience these events on occasion; however, only military families experience all three characteristics on a routine basis. For example, a parent who works for an international company may travel overseas for meetings, resulting in temporary separation, but would likely not be in a dangerous environment while doing so. In contrast, military families may be separated from their service member multiple times per year for training or combat while their service member is at risk of injury.

This entry is divided into three sections. First, a brief history of the military and views of military families is provided for context of the culture surrounding military families with children with autism. Second, current knowledge related to military families with children with autism is summarized with comparisons to civilian families with children with autism and other military families. Finally, future directions for military families with children with autism are presented.

Historical Background

Prior to the start of an all-volunteer force in the early 1970s, most soldiers were not married and

had not yet started families. In fact, an old adage in military communities was “if the military wanted you to have a wife, they would have issued you one,” implying the military’s sole focus on the service member. However, with the start of an all-volunteer force in 1973, the demographics of the Armed Forces began to change. Service members were now more likely to have a spouse and children than they were before 1973. In 2015, over half (50.5%) of all service members were married and nearly half (41%) had at least one child, the majority of children between the ages of 0 and 5 years (Department of Defense [DoD] 2015). Military family members, also called military dependents, outnumber the service members by over 600,000 (DoD 2015). Military dependents were becoming a part of the military community the DoD could not overlook.

With the increase in service members with families, the DoD began to recognize the importance of military families for military operations, satisfaction with the military way of life, and, ultimately, retention of the service member in the Armed Forces. In 1985, the Military Family Act was passed to establish a government commitment to helping military families for the first time. The military started to view mission readiness (i.e., the ability of the service member to perform duties), as related to family readiness (i.e., the ability of the family to successfully navigate the demands of military life) (National Military Families Association [NMFA] 2006). The family members of service members needed to be taken care of if the service member was to focus on the mission and have support from the family to remain in the military. In response to shifting demographics and recognition of family readiness, the military created informal and formal support systems for families (Albano 1993). For example, each service has a family assistance center to help military families with different military lifestyle-related concerns such as relocation preparation and insurance.

Although military families are generally viewed as resilient to stressors and adaptable in new or difficult circumstances (Blaisure et al. 2012), the recent wars, military operations, and deployments have taken a toll on military families. Since 2011, there have been several

missions in the Middle East (e.g., Operation Enduring Freedom) requiring over two million service members from active duty, reserve, and National Guard components to serve overseas. In a recent survey, Blue Star Families (2016) found 72% of military spouses reported the current climate of military operations is too stressful. Military families have endured a long period of mobilization, testing their resilience, especially those families with additional stressors not related to the military lifestyle.

Current Knowledge

Despite over 1.7 million children considered military dependents (DoD 2015), research on children in military families, particularly those with autism, is limited. Currently, there are private organization reports and surveys (e.g., OAR 2010; Blue Star Families 2013), government reports (e.g., Ohio State University Project Team 2011), and a few peer-reviewed articles (e.g., Davis and Finke 2015; Freuler and Baranek 2016) to aid professionals and military families in supporting military children with ASD. Most of the research for military families with children with autism has been related to deployments and relocations, two of the main characteristics of military families.

Deployments

A deployment refers to a time when the service member is relocated for a period of time (e.g., a few weeks or multiple months) for a military purpose such as wartime needs or training exercises. When a service member deploys, the family must to adapt to their absence. Members take on new roles and responsibilities once performed by the service member, frequently resulting in changes to the family routine and an increase in family stress as the family learns to adjust (Drummet et al. 2003). This may be particularly true for military families with children with autism, given the amount of care often required for a child with autism. Civilian families with children with autism have reported the numerous responsibilities required to care for a child with autism (Myers et al. 2009), even without the added challenge of losing a parent for a few weeks to a few months. Interviews with military spouses with

children with autism confirmed they experience an increase in emotional stress during deployments (Davis and Finke 2015). Many spouses also reported feeling very isolated in their community as many are away from friends and family and without the help of their deployed spouse (Davis and Finke 2015; Freuler and Baranek 2016).

Children may have difficulty adjusting to the absence of their service member parent. A systematic review by Alfano et al. (2016) found young children express greater externalizing behavior (e.g., temper tantrums, defiance) when they have a deployed parent. Studies with older children reported mixed evidence of deployment effects, but some outcomes such as mental health diagnoses, stress, and mood or behavior disorders were more common when a parent was deployed (Alfano et al. 2016). Military spouses have reported similar emotional and behavioral differences in their children with autism when their service member is deployed (Davis and Finke 2015; Freuler and Baranek 2016). Some spouses have commented on their child's emotional withdrawal during deployments and the difficulty the child has in understanding why their parent is not at home (Davis and Finke 2015). Other spouses have noticed an increase in frequency and severity of autism symptoms during deployments (Davis and Finke 2015).

Relocation

When the military needs a service member at another installation, they issue a "permanent change of station" (PCS) and require the family to relocate to a new area. Finding medical and therapeutic care after a relocation is challenging for military families with a child with autism. Each time the family relocates, they must find an assortment of interventions to address the wide range of symptoms associated with autism. For example, parents of children with autism have reported working with an average of approximately seven different professionals over six months (Kohler 1999). Military families with children with autism have reported delays in obtaining therapeutic services, limited therapeutic or medical providers in their area, lack of service continuity, and lack of therapeutic service quality (Davis and Finke 2015; Davis et al. 2016; Freuler

and Baranek 2016). Specifically, the majority of 189 military spouses in a survey reported only some access to interventions their child with ASD required, delays in access to interventions, limited access to assistive technologies when they were needed, difficulties finding physicians near their homes, and inadequate continuity of interventions across locations (Davis et al. 2016). Civilian families with children with autism have also reported limited availability and accessibility of therapeutic services for their child with autism and dissatisfaction with the quality of some of those services as well (Kohler 1999). However, in most circumstances, military families are required to relocate more often than civilian families, compounding service availability issues.

Along with finding medical and therapeutic services, military families must find and enroll their child in a new school. Military children switch schools an average of six to nine times between kindergarten and 12th grade, causing disruption in educational programming (Ohio State University Project Team 2011). Changing schools requires transferring student files, completing paperwork, and learning about the receiving school's requirements. Interviews with school liaisons for the Marine Corps indicated school transitions as the most common and most severe problem for military families related to education (Aronson and Perkins 2013).

Given the number of school professionals children with autism may work with on a weekly basis, military families with children with autism may have even greater challenges related to school transitions after relocations. Military families with children with autism have commented on the lack of therapeutic service continuity between schools as it relates to the implementation of a child's IEP (Davis and Finke 2015; Davis et al. 2016; Freuler and Baranek 2016). Many also felt dissatisfied with the quality of the services provided by the school, some believing their options for legal recourse were limited due to their transitory lifestyle (Davis and Finke 2015). Similarly, civilian families with children with ASD have reported worries about transitioning between schools (Parsons et al. 2009), without the added stress of multiple required relocations during the child's school career.

Frequent relocation may increase a child's risk of emotional and/or behavioral distress. Data from a national health interview survey indicated increased family mobility as a risk to children's emotional and behavioral health and education, even after controlling for other variables (Simpson and Fowler 1994). Evidence with military children and relocation effects are mixed, with some studies supporting positive outcomes and others finding negative outcomes for the military children (Milburn and Lightfoot 2013). The negative impacts of relocation on military children with autism may be more pronounced given common symptoms of autism such as anxiety comorbidity, resistance to change, difficulty with social interaction, and challenging behavior (Barbaro and Dissanayake 2009). Military spouses reported their children with autism demonstrated an increase in challenging behaviors, such as more aggression and temper tantrums, after relocation (Davis and Finke 2015; Freuler and Baranek 2016). Military spouses also stated their child with autism was more anxious after relocation, particularly until the family reestablishes a routine (Davis and Finke 2015). Some military spouses commented on the difficulty their child with autism had establishing new friend support in the new school and community (Davis and Finke 2015).

Military families with children with autism may be even more at risk for stress related problems. Current research has indicated military spouses encounter greater amounts of stress following a relocation than when the family is settled (Davis and Finke 2015). Similarly, civilian parents of children with autism have reported greater amounts of stress than parents of neurotypical children or children with other developmental disorders (Hayes and Watson 2013). Therefore, military families with children with autism are likely starting at a higher baseline of stress than other military families and are likely a higher risk for stress and problems related to stress.

Military Supports for Families with Children with Special Needs

Recently, the DoD has sought to reduce some of the challenges military families with children, including those with special needs, encounter as a

result of being in the military. For example, as a way to alleviate some of the challenges related to school transitioning, the DoD encouraged states to sign the MICCC (2011). The MICCC (2011) is an agreement signed by all 50 states to enhance educational opportunity for military children by reducing barriers to school enrollment, placement and eligibility for classes, and graduation. Another school-based program is the school liaison programs operated by each service to help military families solve issues related to the military and schools (DODEA 2014). The DoD has updated their websites such as Military One Source to be more user-friendly and have added sections containing information specific to military families with children with special needs. They have also established a program called the Exceptional Family Members Program (EFMP), independently operated by each military service to help ensure families are relocated to an installation near resources required to meet the family member's special needs (OAR 2010). EFMP offices may also provide other supports for the families; however, these additional supports are not required and vary in scope and quantity by branch and installation (OAR 2010). The DoD created the Extended Care Health Option (ECHO) program to help provide additional financial support for the medical needs of families with members with special needs; however, families must enroll after each move (OAR 2010). While these supports by the DoD are helpful, they do not address all of the needs for autism and military issues. Further, military families commented the problems did not meet their expectations and are inconsistent (Blue Star Families 2013). Additional research needs to be completed to determine which supports are required and how they should be implemented.

Future Directions

Military families with children with autism report several challenges they encounter as a result of having a child with autism and being a part of the military lifestyle. The challenges reported are similar to those faced by other military families and other families with children with autism; however,

it is the dual membership in both communities susceptible to difficulties that exacerbates the challenges military families with children with autism face. Given the lack of peer-reviewed research in this area, policymakers, professionals, military personnel, and researchers have little information to guide them when developing policies and programs to support these families. Additional research should be completed to help fully address the challenges military families with children with autism experience. Further, it is possible supports developed for military families with children with autism may be useful for civilian families as there are parallels between the two populations. Examples of future research directions include the following:

1. Experiences of male spouses with children with autism
2. Experiences of Reservist and National Guard families with children with autism
3. Concomitant family issues (e.g., a parent with post-traumatic stress disorder) and a child with autism
4. Programs and supports helpful to military families with children with autism
5. Medical and therapeutic professionals' knowledge of military families and their culture

References and Reading

- Albano, S. (1993). Military recognition of family concerns: Revolutionary war to 1993. *Armed Forces & Society*, 20(2), 283–302.
- Alfano, C. A., Lau, S., Balderas, J., Bunnell, B. E., & Beidel, D. C. (2016). The impact of military deployment on children: Placing developmental risk in context. *Clinical Psychology Review*, 43, 17–29.
- American Psychiatric Association (APA). (2013). *Diagnostic and statistical manual of mental disorder* (5th ed.). Washington, DC: American Psychiatric Association.
- Aronson, K. R., & Perkins, D. F. (2013). Challenges faced by military families: Perceptions of United States Marine Corps school Liaisons. *Journal of Child and Family Studies*, 22, 516–525.
- Barbaro, J., & Dissanayake, C. (2009). Autism spectrum disorders in infancy and toddlerhood: A review of the evidence on early signs, early identification tools, and early diagnosis. *Journal of Developmental and Behavioral Pediatrics*, 30, 447–459.

- Blaisure, K. R., Saathoff-Wells, T., Pereira, A., MacDermid-Wadsworth, S., & Dombro, A. L. (2012). *Serving military families in the 21st century*. New York: Taylor & Francis Group, LLC.
- Blue Star Families. (2013). Military families lifestyle survey results. Retrieved from <http://bluestarfam.org/Policy/Surveys>.
- Blue Star Families. (2016). Military family lifestyle survey: Comprehensive report. Retrieved from <http://bluestarfam.org/Policy/Surveys>.
- Davis, J. M., & Finke, E. H. (2015). The experience of military families with children with autism spectrum disorders during relocation and separation. *Journal of Autism and Developmental Disorders*, 45(7), 2019–2034.
- Davis, J. M., Finke, E. H., & Hickerson, B. (2016). Service delivery experiences and intervention needs of military families with children with ASD. *Journal of Autism and Developmental Disorders*, 46(5), 1748–1761.
- Department of Defense (DoD), Office of the Deputy Assistant Secretary of Defense. (2015). Profile of the military community: 2015 Demographics. Retrieved from <http://download.militaryonesource.mil/12038/MOS/Reports/2015-Demographics-Report.pdf>.
- Department of Defense Education Activity (DoDEA). (2014). School liaison officers. Retrieved from www.dodea.edu.
- Drummet, A. R., Coleman, M., & Cable, S. (2003). Military families under stress: Implications for family life education. *Family Relations*, 52(3), 279–287.
- Freuler, A., & Baranek, G. (2016). Military spouses caring for a child with autism: Exploring risk and protective factors. *Journal of Family Medicine*, 3(1), 1–7.
- Hayes, S. A., & Watson, S. L. (2013). The impact of parenting stress: A meta-analysis of studies comparing the experience of parenting stress in parents of children with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43, 629–642.
- Kohler, F. W. (1999). Examining the services received by young children with autism and their families: A survey of parent responses. *Focus on Autism and Other Developmental Disabilities*, 14(3), 150–158.
- Milburn, N. G., & Lightfoot, M. (2013). Adolescents in wartime US military families: A developmental perspective on challenges and resources. *Clinical Child and Family Psychological Review*, 16, 266–277.
- Military Interstate Children's Compact Commission (MICCC). (2011). Interstate compact on educational opportunity for military children act. Retrieved from <http://mic3.net/>.
- Myers, B. J., Mackintosh, V. H., & Goin-Kochel, R. P. (2009). "My greatest joy and my greatest heart ache:" Parents' own words on how having a child in the autism spectrum has affected their lives and their families' lives. *Research in Autism Spectrum Disorders*, 3, 670.
- National Military Family Association (NMFA). (2006, December 20). Statement of the National Military Family Association before the Department of Defense Task Force on mental health. Retrieved from http://support.militaryfamily.org/site/DocServer/NMFA_DoD_Mental_health_Task_Force_12-20-06.pdf?docID=7701.
- Ohio State University Project Team. (2011, January 6). Education services for military dependent children with autism: Phase 2: Final project report submitted to the Department of Defense, Deputy Assistant Secretary of Defense, Military Community and Family Policy. Retrieved from http://download.militaryonesource.mil/12038/MOS/EFMP/DOD_ASD_Final_Report_PhaseII.pdf.
- Organization for Autism Research & Southwest Autism Research and Resource Center (OAR). (2010). Life journey through autism: A guide for military families. Retrieved from <http://www.mccslejeune.com/efmp/LifeJourneyThroughAutism.pdf>.
- Parsons, S., Lewis, A., & Ellins, J. (2009). The views and experiences of parents of children with autism spectrum disorder about educational provision: Comparisons with parents of children with other disabilities from an online survey. *European Journal of Special Needs Education*, 24(1), 37–58.
- Segal, M. (1986). The military and the family as greedy institutions. *Armed Forces and Society*, 13(1), 9–38.
- Simpson, G., & Fowler, M. (1994). Geographic mobility and children's emotional/behavioral adjustment and school functioning. *Pediatrics*, 93(2), 303–309.

Milk Protein

► Casein

Miller Assessment for Preschoolers (MAP)

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Description

The Miller Assessment for Preschoolers (MAP) is designed to evaluate the developmental status of children aged 2 years 9 months to 5 years 8 months

across a broad range of areas including behavioral, motor, and cognitive functioning. The MAP test items are categorized into five performance indices. The foundations and coordination indices assess sensory motor abilities involving basic gross and fine motor tasks, awareness of sensations, and oral motor skills. The verbal and nonverbal indices assess cognitive skills required for language development, problem solving, memory, and perception. And the complex task performance index measures sensorimotor abilities in conjunction with cognitive abilities that require the interpretation of visual-spatial information. For children with severe developmental problems, the MAP can be used to provide a developmental overview and to clarify strengths and weaknesses. The MAP may be administered and scored by a wide range of professionals including psychologists, occupational therapists, physical therapists, speech pathologists, nurses, or special education teachers. Scores are obtained through direct testing, and subjective aspects of performance are included in the behavior during testing checklist as well as in supplemental observations that do not contribute to a child's total score. Total scores and performance index scores are expressed in percentile ranks. Item percentiles can be used to interpret the performance of at-risk or borderline cases.

Historical Background

The Miller Assessment for Preschooler was the culmination of 10 years of research involving 4,000 children and 800 test items. Each item was field tested with small samples of preschoolers, and 530 items were chosen for the MAP tryout edition. It is from this sample that the 27 items that constitute the MAP were selected. The MAP was first published in 1982 and then revised in 1988. The theoretical information that contributed to the development of the MAP comes from a broad range of literature in child development, education, psychology, language development, physical and occupational therapy, and medicine.

Psychometric Data

The MAP was standardized on a sample of 1,200 preschoolers, representing all nine continental geographic regions of the United States. Approximately, equal numbers of children in each region were tested, and the sample was stratified by age, sex, race, size of residential community, and socioeconomic factors.

Reliability

Reliability of the MAP was determined using interrater agreement, test-retest reliability, and internal consistency of the total test.

Interrater reliability was calculated on 40 children. Correlation coefficients were calculated on a sample of 40 children who participated in the interrater reliability study. Scores ranged from .84 to .99 for the total test and the performance indices and are suggestive of a high level of interrater agreement.

Test-retest reliability was calculated on a group of 81 children randomly selected from the standardization sample. The two tests were administered between 1 and 4 weeks after initial testing. Retest stability is reported in percentages and ranged from 72% to 94% for the total score and the five performance indices.

Internal reliability was calculated from the item raw scores for the total sample using split half method and the average item to test correction. Both methods produced internal consistency values around .80.

Validity

Three methods were used to support the validity of the MAP: content, criterion-related, and construct validity.

Content validity of the items was established by experts from a number of specialty areas who were asked to rate the content validity within their fields of expertise. A content specifications by item table supports the content validity of the test.

Criterion-related validity was established using concurrent and predictive validity. MAP scores were correlated with one of the four standardized instruments including the Wechsler Preschool and

Primary Scale of Intelligence (WPPSI), the Illinois Test of Psycholinguistic Abilities (ITPA), the Southern California Sensory Integration Tests (SCSIT), and the Denver Developmental Screening Test (DDST). Correlations of the complex task index and the WPPSI verbal and performance IQ approached statistical significance. Correlations were significant for the ITPA and the MAP total score, the coordination index, and the nonverbal index. One significant correlation was found between the MAP foundation index and the SCSIT score. In comparison to the DDST, 72% of the sample was similarly grouped by both instruments; however, the MAP identified 24% more children in the at-risk categories.

Evidence of predictive validity was obtained from a study of children tested 4 years after initial testing with the MAP. Results suggest that performance obtained on the MAP in the preschool years is predictive of intelligence, motor skill acquisition, and school achievement 4 years later.

Clinical Uses

The MAP may be administered and scored by a wide range of professionals including psychologists, occupational therapists, physical therapists, speech pathologists, nurses, or special education teachers. The purpose of the MAP is to identify developmentally delayed preschool children who need further evaluation and to provide a comprehensive structure clinical framework for identifying a child's strengths and weaknesses. The intent is to identify difficulties in children in the preschool age group (that typically go undiagnosed) that cause them to fall behind their peers in one or more areas of development. The MAP can also be used with children who have more severe developmental problems to provide a developmental overview or strengths and weaknesses when comprehensive standardized evaluations cannot be administered.

The MAP has been translated into Hebrew and Japanese. Studies reflect characterization of clinical populations as well as use as an outcome measure in treatment effectiveness research.

References and Reading

- Banus, B. J. (1983). The Miller assessment for preschoolers (MAP): An introduction and review. *American Journal of Occupational Therapy*, 35, 333–340.
- Daniels, L. E., & Bressler, S. (1990). The Miller assessment for preschoolers: Clinical use with children with developmental delays. *American Journal of Occupational Therapy*, 44, 48–53.
- DeGangi, G. A. (1983). A critique of the standardization of the Miller assessment for preschoolers. *American Journal of Occupational Therapy*, 37, 407–411.
- Fulks, M. A., & Harris, S. R. (2005). Predictive accuracy of the Miller assessment for preschoolers in children with prenatal drug exposure. *Physical & Occupational Therapy in Pediatrics*, 25, 17–37.
- Iwanaga, R., Kawasaki, C., & Tsuchida, R. (2000). Brief report: Comparison of sensory-motor and cognitive function between autism and asperger syndrome in preschool children. *Journal of Autism and Developmental Disorders*, 30, 169.
- Iwanaga, R., Ozawa, H., Kawasaki, C., & Tsuchida, R. (2006). Characteristics of the sensory-motor, verbal and cognitive abilities of preschool boys with attention deficit/hyperactivity disorder combined type. *Psychiatry and Clinical Neurosciences*, 60, 37–45.
- Iwanaga, R., Honda, S., Nakane, H., Tanaka, K., Toeda, H., & Tanaka, G. (2014). Pilot study: Efficacy of sensory integration therapy for Japanese children with high-functioning autism spectrum disorder. *Occupational Therapy International*, 2, 4–11.
- Leosdottir, T., Egilson, S. T., & Georgsdottir, I. (2005). Performance of extremely low birth weight children at 5 years of age on the Miller assessment for preschoolers. *Physical & Occupational Therapy in Pediatrics*, 25, 59–72.
- Miller, L. J. (1987). Response to "A critique of the standardization of the Miller assessment for preschoolers." *American Journal of Occupational Therapy*, 41, 537–538.
- Miller, L. J. (1988a). *The Miller assessment for preschoolers*. San Antonio: Pearson.
- Miller, L. J. (1988b). Longitudinal validity of the Miller assessment for preschoolers. *Perceptual and Motor Skills*, 66, 811–814.
- Parush, S., Yochman, A., Jessel, A. S., Shapiro, M., & Mazor-Karsenty, T. (2002). Construct validity of the Miller assessment for preschoolers and the pediatric examination of educational readiness for children. *Physical & Occupational Therapy in Pediatrics*, 22, 7–27.
- Parush, S., Winkur, M., Golstand, S., & Miller, L. J. (2002a). Long term predictive validity of the Miller assessment for preschoolers. *Perceptual and Motor Skills*, 94, 9221–9226.
- Parush, S., Winkur, M., Golstand, S. M., & Miller, L. J. (2002b). Prediction of school performance using the Miller assessment for preschoolers: A validity study. *American Journal of Occupational Therapy*, 56, 547–555.

- Schneider, E., Parush, S., Katz, N., & Miller, L. J. (1995). Performance of Israeli versus US preschool children on the Miller assessment for preschoolers. *American Journal of Occupational Therapy*, 49, 19–23.
- Yochman, A., Ornoy, A., & Parush, S. (2006). Co-occurrence for developmental delays among preschool children with attention-deficit-hyperactivity disorder. *Developmental Medicine and Child Neurology*, 48, 483–488.

Miller Function and Participation Scales

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Synonyms

M-FUN

Description

The Miller Function and Participation Scales (M-FUN) is a developmental assessment for children ages 2.6–7.11, designed to assist therapists in determining how a child's motor capacities affect participation in home and school environments. Based on the World Health Organization's International Classification of Function, Disability and Health (ICF; WHO 2001), the M-FUN addresses the mandate of occupational therapists to evaluate the whole child and determine how a child's motor problems might be affecting his or her functional abilities. The scales consist of a performance component, which includes workbook activities to tap visual motor abilities and play-based activities that tap gross and fine motor abilities. The performance component is comprised of three subscales: gross motor, fine motor, and visual motor. In addition, there is a participation component, which consists of three observation checklists that rate a child's participation at home, school, and during the testing session. Among the abilities

examined in the M-FUN observation checklists are mobility, postural control, sensory discrimination and modulation, social skills and communication, object manipulation, and participation in daily routines.

Several indicators of a child's performance can be derived from the M-FUN including standard scores, percentile ranks, and age equivalents for each subscale. Criterion-referenced scores are derived for home observation, classroom observation, and test observation checklists. The scales can be used to determine if a child has a developmental delay, or for children older than 7.11 years who have known motor delays, the test can be used to measure progress or to plan treatment. Unique to the M-FUN is the Neurological Foundations Profile, which facilitates the identification of underlying impairments affecting the child's ability to perform tasks and serves as a guide for treatment planning. Additionally, the M-FUN provides information about growth in the child's visual motor, fine motor, and gross motor abilities. Following intervention, the child can be retested using the M-FUN, and progress scored can be derived that reflects the acquisition of new skills even if he or she is not progressing at the same rate as age-ability peers.

Historical Background

The M-Fun was developed out of the need for a functional motor evaluation of skills and abilities that underlie participation in school. Based on the model from the ICF (WHO 2001) and OT Practice Framework (AOTA 2002), the M-FUN employs a competency model rather than a deficit model to assess children.

Psychometric Data

Normative data was collected from over 400 children from four geographic regions of the United States (West, South, North Central, and Northeast). The standardization sample closely matches the demographic characteristics of children ages 2.6–7.11 by sex, race/ethnicity, and

parent education level reported the 2002 United States census. The standardization sample also included children in each age group (5–7% of total) who were diagnosed with a motor delay or impairment.

The reliability of the M-FUN was examined using test-retest reliability, internal consistency, and interrater reliability. Retest of 27 children from the standardization sample was conducted with 0–21 days of initial testing. Moderately high reliability was found across time for all ages with scores ranging from .77 to .82. Internal consistency reliability supports the homogeneity of the items in the scale with the average reliability coefficients ranging from .85 for the visual motor subscale to .90 for the fine motor subscale and .92 for the gross motor subscale. Reliability coefficients for the clinical group on each subscale were similar to those reported for the normative sample. In addition, reliability coefficients for all three observation checklists were excellent, ranging from .87 to .98. Interrater reliability was computed for 29 children with the average decision agreement 93–96% for each subscale.

Validity data was collected for test content, internal structure, relationship to other variables, and diagnostic accuracy.

Content validity was based on ICF, the OT Practice Framework, a review of the literature, and focus group experts. The internal structure was analyzed by examining patterns of scaled score intercorrelations. Coefficients were between .47 and .55 suggesting that each subscale measures a different motor construct/ability. Concurrently validity was examined in order to determine the M-FUN's relatedness with other instruments designed to measure similar constructs. One study compared scores on the Miller Assessment for Preschoolers (MAP) to scores on the M-FUN in a sample of 15 children. Correlations ranged from .47 to .83 suggesting that the two tests supply complementary information about a child's abilities. A study of discriminant validity revealed that the M-FUN subscales discriminate between children with clinically diagnosed delays and those who are typically developing at a meaningful and statistically significant level. Classification

analysis to assess the clinical utility of the M-FUN produced moderate to excellent sensitivity and specificity. For cutoff scores of 1 standard deviation below the mean, the sensitivity was between .69 and .89, and the specificity was between .80 and 1.00.

Clinical Uses

The M-FUN may be administered by a variety of qualified professional including occupational therapists, physical therapists, special education or adaptive physical education specialists, and early childhood interventionists who are interested in determining how a child's motor abilities affect their participation in home and school. Examiners should have experience in test administration, scoring, and interpretation, as well as knowledge of sensory processing and motor development in preschool and school-aged children.

The M-FUN can be used for a variety of purposes:

- To determine a child's eligibility for intervention services to address gross motor, fine motor, and visual motor delays
- To document delays in motor development (gross motor, fine motor, visual motor)
- To determine motor readiness/preparedness for school – strengths and needs in the area of motor abilities that may impact on the child's ability to participate fully in school and home routines
- To make recommendations for optimizing the child's functioning at school and home (least restrictive environment)
- To assist in the development of treatment planning through use of the neurological foundations profile
- To identify underlying neuromotor deficits affecting functional abilities – to identify fine motor, visual motor, and gross motor skill areas that might affect a child's school success

To measure progress over time following a course of intervention

References and Reading

- American Occupational Therapy Association. (2002). Occupational therapy practice framework: Domain and process. *American Journal of Occupational Therapy*, 56, 609–639.
- Miller, L. J. (2006). *Miller function and participation scale*. San Antonio: Pearson.
- The World Health Report. (2001). *Mental health: New understanding, new hope*. Geneva.

Mind Reading

► Pareidolic Faces

Mindblindness

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Definition

“Mindblindness” is a term coined by autism researcher Simon Baron-Cohen (1990). It also is the title of the essay containing work from his doctoral dissertation (Baron-Cohen 1997). In that essay, Baron-Cohen theorized that humans have evolved to be able to “mindread” or to effortlessly, automatically, and unconsciously assess the behavior of others. This is critical because mindreading forms the basis of the ability to successfully engage in social interactions requiring the minute-by-minute interpretation and prediction of the behavior of one’s interaction partners. Baron-Cohen goes on to propose that persons with autism have a selective impairment in mindreading.

The concept of mindblindness is rooted in the fields of both evolutionary biology and cognitive science. It is Baron-Cohen’s contention that natural selection favored species and individuals from those species with greater social intelligence

which included mindreading abilities. Mindreading abilities enabled primates to cohesively bond together in organized social hierarchies to achieve common goals. Those who were more adept at playing social chess through excellent mindreading skills (forward planning, plotting, counterplotting, strategic problem solving to achieve one’s goals) ended up at the top of such social hierarchies. Given the importance of mindreading abilities to survival, in this view, such abilities are believed to be a discrete component of cognition or a “module.”

The human mindreading system is thought to have four separate components. The first – the intentionality detector (ID) – is a perceptual device that interprets approach and avoidance in terms of goals and desires. For example, it enables one to interpret the motion of a mouse toward cheese as reflecting the mouse’s desire to eat. ID is the first mechanism human infants use. It can be cued by all forms of sensory input (i.e., seeing or hearing something). The second mechanism is the eye direction detector (EDD), which works through the visual system and computes whether eye-like stimuli are directed toward or away from something, including whether the eyes are directed at the organism itself or toward another organism. The third component – the shared attention mechanism (SAM) – builds triadic representations which specify relations among the agent, the self, and another object or agent. For example, the SAM would compute that Johnny and I are both looking at the cheese (or mom). Finally, the theory of mind mechanism (TOMM) is a system for inferring the full range of mental states from behavior or for employing a “theory of mind.” Theory of mind is a shorthand term for the capacity to attribute mental states to oneself and others and to interpret behavior in terms of mental states.

References and Reading

- Baron-Cohen, S. (1990). Autism: A specific cognitive disorder of “mind-blindness”. *International Review of Psychiatry*, 2, 79–88.
- Baron-Cohen, S. (1997). *Mindblindness: An essay on autism and theory of mind*. Cambridge: MIT Press.

Mindfulness Therapy for Individuals with Autism Spectrum Disorder

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Synonyms

Mindfulness-based therapy/intervention

Definition

Mindfulness can be defined as attending to the present moment emotions and thoughts without judgment (Kabat-Zinn et al. 1985). Mindfulness therapies teach clients skills, which have their origins in Buddhist traditions, related to enhanced present-moment awareness and acceptance of thoughts, emotions, and experiences using mindfulness meditation (Baer 2003). Mindfulness has been taught via standardized therapies entirely focused on mindfulness, such as Mindfulness-Based Stress Reduction (MBSR; Kabat-Zinn et al. 1985), as well as integrated into other established therapies including cognitive-behavior therapy (CBT), such as Mindfulness-Based Cognitive Therapy (MBCT; Segal et al. 2002) and Dialectical Behavior Therapy (DBT; Linehan 1993).

Mindfulness therapies have been used with children, adolescents, and adults with ASD. Several programs targeting emotion regulation impairment employed mindfulness-based techniques along with cognitive behavioral (CBT) and other behavioral strategies. A study examining MBSR without adaptation for adults with ASD found increased quality of life, positive

outlook, and mindfulness (Beck et al. 2019). Studies have also utilized adaptations of mindfulness therapies for individuals with ASD, such as shortening the duration of mindfulness meditations and sessions, simplifying instructions, and adapting visual supports. Adapted MBCT has been shown to reduce symptoms of depression, anxiety, and rumination among adults with ASD (Kiep et al. 2015; Spek et al. 2013) and was observed to be equally effective as CBT for depression and anxiety (Sizoo and Kuiper 2017). An individual mindfulness therapy was found to be well-accepted and effective for young adults with ASD who had difficulties with emotion regulation (Conner and White 2018). Lastly, an adaptation of a DBT group targeting emotion regulation and social communication difficulties in adults with ASD found decreased use of suppression and increased reappraisal (Hartmann et al. 2019).

Therapies using mindfulness have also been shown to be effective with children and adolescents with ASD (see Cachia et al. 2016, for review). An emotion regulation-focused adaptation of the Secret Agent Society (Secret Agent Society: Operation Regulation (SAS:OR)) for children with ASD includes mindfulness practices and CBT. A randomized controlled trial comparing SAS:OR to a waitlist control group found improved emotion regulation, decreased problem behaviors (Weiss et al. 2018). Several small studies by Singh et al. (2011a, b) taught a mindfulness practice that was associated with decreased levels of aggression in adolescents with ASD. The Intensive Outpatient Program for Emotion Regulation Treatment is an intensive outpatient program for children with ASD and co-occurring Intellectual Disability which includes mindfulness, CBT, and other behavioral techniques (Shaffer et al. 2019). The intensive outpatient program was found to reduce psychiatric and behavioral symptoms (Shaffer et al. 2019). A pilot open trial of the Emotion Awareness and Skills Enhancement (EASE) program which used mindfulness-based techniques to target impaired emotion regulation among adolescents with ASD observed improvements in emotion regulation, depression, anxiety, and irritability (Conner et al. 2019).

Several studies have involved both parents and children with ASD in interventions. Hwang et al.

(2015) had mothers of children with ASD complete an 8-week mindfulness training prior to teaching their child mindfulness practices. Findings indicated improvements in problem behaviors, parenting stress, and mindful parenting (Hwang et al. 2015). MyMind, a concurrent parent and child mindfulness intervention, has been shown to improve social and communicative skills, decrease internalizing and externalizing problems, improve emotion regulation, and increase adaptive functioning skills in youth and increase mindful parenting and quality of life in parents (de Bruin et al. 2015; Ridderinkhof et al. 2018; Salem-Guirgis et al. 2019). The research on mindfulness therapy in ASD, while preliminary, suggests that mindfulness-based therapies may be useful for a range of treatment targets.

See Also

- Anxiety Disorders
- Cognitive Behavioral Therapy (CBT)
- Comorbidity
- Depressive Disorder
- Emotion Awareness and Skills Enhancement (EASE) Program
- Emotion Regulation

References and Reading

- Baer, R. A. (2003). Mindfulness training as a clinical intervention: A conceptual and empirical review. *Clinical Psychology: Science and Practice*, 10(1998), 125–143. <https://doi.org/10.1093/clipsy/bpg015>.
- Beck, K.B., Greco, C.M., Terhorst, L., et al. (2019). Mindfulness-based stress reduction for adults with autism spectrum disorder: Feasibility and estimated effects. *Poster presented at the International Society for Autism Research*, Montreal.
- Cachia, R. L., Anderson, A., & Moore, D. W. (2016). Mindfulness in individuals with autism spectrum disorder: A systematic review and narrative analysis. *Review Journal of Autism and Developmental Disorders*, 3(2), 165–178. <https://doi.org/10.1007/s40489-016-0074-0>.
- Conner, C. M., & White, S. W. (2018). Brief report: Feasibility and preliminary efficacy of individual mindfulness therapy for adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(1), 390–400. <https://doi.org/10.1007/s10803-017-3312-0>.
- Conner, C. M., White, S. W., Beck, K. B., Golt, J., Smith, I. C., & Mazefsky, C. A. (2019). Improving emotion regulation ability in autism: The emotion awareness and skills enhancement (EASE) program. *Autism*, 23(5), 1273–1287. <https://doi.org/10.1177/1362361318810709>.
- de Bruin, E. I., Blom, R., Smit, F. M., van Steensel, F. J., & Bögels, S. M. (2015). MYmind: Mindfulness training for youngsters with autism spectrum disorders and their parents. *Autism*, 19(8), 906–914. <https://doi.org/10.1177/1362361314553279>.
- Hartmann, K., Urbano, M. R., Raffaele, C. T., Kreiser, N. L., Williams, T. V., Qualls, L. R., & Elkins, D. E. (2019). Outcomes of an emotion regulation intervention group in young adults with autism spectrum disorder. *Bulletin of the Menninger Clinic*, 83(3), 259–277. <https://doi.org/10.1521/bumc.2019.83.3.259>.
- Hwang, Y. S., Kearney, P., Klieve, H., Lang, W., & Roberts, J. (2015). Cultivating mind: Mindfulness interventions for children with autism spectrum disorder and problem behaviours, and their mothers. *Journal of Child and Family Studies*, 24(10), 3093–3106. <https://doi.org/10.1007/s10826-015-0114-x>.
- Kabat-Zinn, J., Lipworth, L., & Burney, R. (1985). The clinical use of mindfulness meditation for the self-regulation of chronic pain. *Journal of Behavioral Medicine*, 8(2), 163–190.
- Kiep, M., Spek, A., & Hoeven, L. (2015). Mindfulness-based therapy in adults with an autism spectrum disorder: Do treatment effects last? *Mindfulness*, 6(3), 637–644. <https://doi.org/10.1007/s12671-014-0299-x>.
- Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York: Guilford Press.
- Ridderinkhof, A., de Bruin, E. I., van den Driesschen, S., & Bögels, S. M. (2018). Attention in Children with Autism spectrum disorder and the effects of a mindfulness-based program. *Journal of Attention Disorders*, 24, 108705471879742. <https://doi.org/10.1177/1087054718797428>.
- Salem-Guirgis, S., Albaum, C., Tablon, P., Riosa, P. B., Nicholas, D. B., Drmic, I. E., & Weiss, J. A. (2019). MYmind: A concurrent group-based mindfulness intervention for youth with autism and their parents. *Mindfulness*, 10(9), 1730–1743. <https://doi.org/10.1007/s12671-019-01107-9>.
- Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2002). *Mindfulness-based cognitive therapy for depression: A new approach to relapse prevention*. New York: Guilford.
- Shaffer, R. C., Wink, L. K., Ruberg, J., Pittenger, A., Adams, R., Sorter, M., ... Erickson, C. A. (2019). Emotion regulation intensive outpatient programming: Development, feasibility, and acceptability. *Journal of Autism and Developmental Disorders*, 49(2), 495–508. <https://doi.org/10.1007/s10803-018-3727-2>.
- Singh, N. N., Lancioni, G. E., Manikam, R., Winton, A. S. W., Singh, A. N. A., Singh, J., & Singh, A. D. A. (2011a). A mindfulness-based strategy for self-management of aggressive behavior in adolescents with autism. *Research in Autism Spectrum*

- Disorders*, 5(3), 1153–1158. <https://doi.org/10.1016/j.rasd.2010.12.012>.
- Singh, N. N., Lancioni, G. E., Singh, A. D. A., Winton, A. S. W., Singh, A. N. A., & Singh, J. (2011b). Adolescents with Asperger syndrome can use a mindfulness-based strategy to control their aggressive behavior. *Research in Autism Spectrum Disorders*, 5(3), 1103–1109. <https://doi.org/10.1016/j.rasd.2010.12.006>.
- Sizoo, B. B., & Kuiper, E. (2017). Cognitive behavioural therapy and mindfulness based stress reduction may be equally effective in reducing anxiety and depression in adults with autism spectrum disorders. *Research in Developmental Disabilities*, 64, 47–55. <https://doi.org/10.1016/j.ridd.2017.03.004>.
- Spek, A. A., van Ham, N. C., & Nyklíček, I. (2013). Mindfulness-based therapy in adults with an autism spectrum disorder: A randomized controlled trial. *Research in Developmental Disabilities*, 34(1), 246–253. <https://doi.org/10.1016/j.ridd.2012.08.009>.
- Weiss, J. A., Thomson, K., Burnham Riosa, P., Albaum, C., Chan, V., Maughan, A., ... Black, K. (2018). A randomized waitlist-controlled trial of cognitive behavior therapy to improve emotion regulation in children with autism. *Journal of Child Psychology and Psychiatry*, 59(11), 1180–1191. <https://doi.org/10.1111/jcpp.12915>.

Mindfulness-Based Therapy/Intervention

► Mindfulness Therapy for Individuals with Autism Spectrum Disorder

Mineral Treatments

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Definition

Dietary minerals are chemical elements needed for mammals to maintain basic physiologic functioning. Minerals that are needed in larger

amounts to maintain health (macrominerals) include calcium, phosphorous, magnesium, sodium, potassium, phosphorous, chloride, and sulfur. Lesser amounts of iron, manganese, copper, iodine, zinc, cobalt, fluoride and selenium, and molybdenum are necessary for cellular functioning (trace minerals). Minerals are absorbed from dietary sources. The dietary reference index (DRI) publishes the required intake of minerals for age and gender. If a person with autism spectrum disorder ingests a very restricted diet because of food aversions or is given a restricted diet as a proposed intervention for symptoms of autism, they might be at increased risk for diminished intake of minerals. For example, removal of dairy products from the diet eliminates the major source of calcium in milk products. Calcium is necessary both for bone growth and for many essential biochemical pathways including nerve conduction. Mineral supplementation in individuals with autism might be indicated if there is deficient nutritional intake (Stewart et al. 2015). Many people with/without autism are deficient in the mineral iron. Iron deficiency will ultimately lead to low blood counts or anemia. Prior to that point, lowered iron stores may lead to fatigue and sleep disruption/restless leg syndrome, among other neurobehavioral symptoms. Iron supplementation would be recommended after laboratory documentation of deficiency (Reynolds et al. 2012).

In addition to physiologic replacement of necessary nutrients, a number of controversial therapies provide minerals at amounts greater than recommended by the DRI to target symptoms of autism. Minerals included in novel therapy regimens include magnesium (given with vitamin B6) and zinc. Well-designed clinical trials do not support routine supplementation of minerals at greater than the requirements determined by the DRI.

Blood and hair measurement of minerals has been used without scientific support to date to determine if individuals with autism have altered metabolic requirements for minerals or mineral levels purported to be associated with neurologic symptoms. Laboratory measurement of minerals in blood or hair is not currently a conventional

component of the etiologic workup of individuals with autism. Testing for specific minerals may be necessary when there are suspected toxic exposures such as environmental lead exposure. Elevated lead levels are associated with impaired learning and behavioral challenges. If elevated, chelation may be used medically to remove lead from the blood and body stores. Chelation is a medical procedure advocated by some practitioners for individuals with autism without support of the scientific literature with the goal of increased excretion of heavy metals (a type of mineral) like mercury. There is no scientific evidence at this time that elevated levels of mercury or any other metal are associated with autism. Chelation increases excretion of other minerals from the body as well as the metal of concern and may impact the availability of minerals necessary for routine physiologic functions. Practitioners who prescribe chelation will prescribe supplements containing minerals and trace minerals that may be excreted along with the lead or other metal of concern. Chelation is not a conventional treatment for symptoms of autism and has associated risks although uncommon (James et al. 2015).

See Also

► [Chelation](#)

References and Reading

- American Academy of Pediatrics. (2009). In E. Ronald Klerman (Ed.), *Pediatric nutrition handbook* (pp. 387–443). Elk Grove Village: Author.
- Geraghty, M. E., Bates-Wall, J., Ratliff-Schaub, K., & Lane, A. E. (2010). Nutrition interventions and therapies in autism. *ICAN: Infant, Child and Adolescent Nutrition*, 2(2), 120–133.
- <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0006990/>. Accessed 24 May 2012, Pubmed Health, US National Library of Medicine.
- <https://www.ncbi.nlm.nih.gov/books/NBK56068/table/summarytables.t3/?report=objectonly>. Accessed 25 Aug 2017.
- James, S., Stevenson, S. W., Silove, N., & Williams, K. (2015). Chelation for autism spectrum disorder (ASD). *The Cochrane Database of Systematic Reviews*, 11(5), CD010766. PMID: 26114777.
- Johnson, C. P., Myers, S. M., & American Academy of Pediatrics Council on Children with Disabilities. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120(5), 1183–1215.
- Reynolds, A., Krebs, N. F., Stewart, P. A., Austin, H., Johnson, S. L., Withrow, N., Molloy, C., James, S. J., Johnson, C., Clemons, T., Schmidt, B., & Hyman, S. L. (2012). Iron status in children with autism spectrum disorder. *Pediatrics*, 130(Suppl 2), S154–S159. PMID: 23118246.
- Stewart, P. A., Hyman, S. L., Schmidt, B. L., Macklin, E. A., Reynolds, A., Johnson, C. R., James, S. J., & Manning-Courtney, P. (2015). Dietary supplementation in children with autism spectrum disorders: Common, insufficient, and excessive. *Journal of the Academy of Nutrition and Dietetics*, 115(8), 1237–1248.

Minimal Brain Damage

► [Attention Deficit/Hyperactivity Disorder](#)

Minimal Brain Dysfunction

► [Attention Deficit/Hyperactivity Disorder](#)

Minimal Speech Approach

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Definition

The minimal speech approach was developed by Potter and Whittaker (2001) to enable communication in children with autism. This approach requires the caregiver or interventionist to use simple speech, consisting of one to three words, usually nouns, which are paired with nonverbal communication. The nonverbal communication can consist of a picture, an object, or a gesture, which help to facilitate comprehension. For example, using the minimal speech approach, the therapist might say “lunch” and point to a picture

communication symbol of a lunch box rather than saying “All right, choice time is over now. We need to put everything away and get ready for lunch. Let’s go.”

In order to create a communication-enabling environment, key characteristics of this approach include reducing the use of speech in all situations, appropriate mapping of single words, giving information in nonverbal ways, minimizing running commentary, delaying the use of speech when teaching new tasks, and avoiding a focus on relative and temporal terms (Potter and Whittaker 2001).

Children with autism often experience confusion when excess speech occurs during communicative interactions. This can lead to anxiety and distress. The simplification of language aims to avert social disengagement and challenging behaviors by facilitating understanding. Potter and Whittaker (2001) found that when a minimal speech approach is used consistently and appropriately, children are more socially responsive and demonstrate more spontaneous communication.

References and Reading

- Howlin, P., Marwood, L., & Rutter, M. (2000). Autism and developmental receptive language disorder: A follow-up comparison early adult life II: Social, behavioral and psychiatric outcomes. *Journal of Child Psychology and Psychiatry*, 41(5), 561–578.
- Klin, A. (1991). Young autistic children’s preferences in regard to speech: A possible characterization of the symptom of social withdrawal. *Journal of Autism and Developmental Disorders*, 21(1), 29–42.
- Peterson, S. L., Bonda, A. S., Vincent, Y., & Finnegan, C. S. (1995). Effects of altering communicative input for student with autism and no speech. *Augmentative and Alternative Communication*, 11, 93–100.
- Potter, C., & Whittaker, C. (2001). *Enabling communication in children with Autism*. Philadelphia: Kingsley.
- Prelock, P. A. (2006). Interventions to support communication. In *Autism spectrum disorders: Issues in assessment and intervention* (pp. 409–412). Austin: Pro-Ed.
- Rapin, I., & Dunn, M. (1997). Language disorders in children with autism. *Seminars in Paediatric Neurology*, 4(2), 86–92.
- Schuler, A. L., Prizant, B. M., & Wetherby, A. M. (1997). Enhancing language and communication development: Prelinguistic approaches. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders*. New York: Wiley.

Mirror Mechanisms

► Frontal Lobe Findings in Autism

Mirror Neuron System

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Structure

Mirror neurons are a unique subset of neurons located in motor areas of the brain, which are thought to play a central role in social communication and imitation. They are active not only during the execution of movements but also during the passive observation of movements made by others. It has been hypothesized that the activity of mirror neurons during observation of movement might represent an internal “motor simulation” of the observed movement. Such a simulation may then be used as a basis for imitating the observed movement or as a gateway for accessing the intentions, goals, and emotions associated with the movement in the mind of the observer. According to the hypothesis, the observer then attributes these intentions and emotions to the observed person in order to interpret their behavior. Individuals with autism have difficulties understanding the intentions, states, and emotions of people around them. It has been suggested that these social difficulties may be caused by the development of abnormal mirror neurons. Despite the large amount of attention that this hypothesis has received in the popular and scientific literatures, the evidence supporting it has been weak and inconsistent. Below is a general overview of mirror neuron research in monkeys and in humans followed by a description of the research related specifically to autism.

Function

Monkey Mirror Neurons

In 1996, Gallese et al. (1996) discovered that about 10% of neurons in ventral premotor area F5 of the macaque monkey respond not only when the monkey executes a particular movement – as expected in this cortical motor area – but also when the monkey passively observes the experimenter performing that very same movement. These visuomotor neurons were named “mirror neurons” because their activity in the brain of the passively observing monkey seems to mirror that of motor neurons active in the person actually executing the movement. Following this initial finding, in 1998, Fogassi et al. (1998) reported that mirror neurons also exist in the inferior parietal lobule (IPL) of the macaque and established the idea of a “mirror system” composed of these two cortical areas.

In these and further studies (Caggiano et al. 2009; Ferrari et al. 2003, 2005; Fogassi et al. 2005; Kohler et al. 2002; Umiltà et al. 2001), mirror neurons were identified based on two critical response characteristics: First, mirror neurons respond in a selective manner to a particular preferred movement such that some neurons respond, for example, only to grasping with the whole hand while others respond only to gripping with the fingertips. In this manner, mirror neuron responses distinguish between different movements in an analogous manner to primary visual cortex neurons that distinguish between different visual stimulus orientations. Second, mirror neurons respond similarly to their preferred movement regardless of whether it is being passively observed or actively executed; thus, the same mirror neuron that responds when the monkey grips with the fingertips also responds when the monkey passively observes someone else gripping with the fingertips. These unique characteristics distinguish mirror neurons from the many other visual, motor, and visuomotor neurons that comprise about 90% of the neurons in areas F5 and IPL of the monkey and are involved in a multitude of visuomotor processes needed for the coordination of movement (Castiello 2005).

The discovery of mirror neurons generated much excitement regarding their possible role in mechanisms of movement understanding and imitation. When we observe someone performing a movement, such as waving their hand to indicate “hello,” how do we instantaneously understand that their intention is to greet us? According to the “simulation theory,” we covertly and unconsciously simulate ourselves performing the movement, then access our own associated intentions and goals for that particular movement and assign them to the person we are observing (Rizzolatti and Craighero 2004). This chain of neural computations supposedly enables us to both understand and ascribe intentionality to others. Mirror neurons have, therefore, been proposed as the physiological mechanism that enables a critical step in this process: precise visual to motor mapping (Rizzolatti and Sinigaglia 2010). Specifically, according to the theory, whenever you observe someone performing an action, particular movement-selective mirror neurons embedded in your motor system are activated, enabling you to simulate yourself performing that movement and to access your own associated intentions and goals (probably through activity of other brain areas, including the limbic system). Taken a step further, mirror neurons can be thought of as a sensorimotor gateway for forming an internal representation of the observed person’s intentions and emotions based on their body language, facial expressions, actions, and so on (Rizzolatti and Craighero 2004; Rizzolatti and Sinigaglia 2010).

It has also been suggested that the same act of motor simulation facilitates imitation and thereby underlies our ability to learn new movements by imitating others (Buccino et al. 2004b; Iacoboni et al. 1999). In this case, covert simulation, which involves activating the precise motor neurons that encode the proper kinematics and dynamics necessary for the execution of the observed movement, may be followed by overt imitation, which involves activating the same neurons along with additional motor neurons that actually command the appropriate muscle contractions.

It is important to note two rather surprising points regarding mirror neuron research in non-human primates. First, all of the described mirror

neurons responded only to object-oriented movements such as grasping or ripping an object. Thus, while the macaque monkey does display several social and communicative movements (e.g., friendliness by lip smacking or threatening by exposing teeth), there have been no reports of mirror neurons responding to such movements as one might have expected if this system was to mediate the representation of other's intentionality. This lack of evidence argues against the suggestion that mirror neurons enable the monkey to understand the intentions and emotions of other monkeys. Second, although macaque monkeys clearly possess mirror neurons, this species does not naturally learn by imitation, except for, perhaps, a very short period at infancy (Ferrari et al. 2006). The absence of imitative behavior in the macaque challenges the hypothesis that mirror neurons enable imitation.

Human Mirror System

The excitement regarding the possible role of mirror neurons in social cognition drove many investigators to search for the human homologue of the monkey mirror system. Early neuroimaging studies utilizing positron emission topography (PET), functional magnetic resonance imaging (fMRI), electroencephalograph (EEG), and magnetoencephalograph (MEG) have applied three types of protocols to elicit mirror neuron responses in humans: passive movement observation, active movement execution, and imitation (simultaneous observation and execution). This section will focus on the fMRI studies because this technique offers the best spatial resolution and offers the strongest evidence regarding the cortical location of responsive neural populations. The discussion below, however, is just as applicable to studies using the other neuroimaging techniques that have generally used the same experimental protocols and the same underlying logic.

In the first protocol, subjects passively viewed images or video clips of movements, such as a smiling face or a hand grasping an object, and their neural responses were compared against a rest condition, following the logic that mirror neurons respond during movement observation and not during rest (Buccino et al. 2001, 2004a;

Iacoboni et al. 2005). In the second protocol, responses during movement execution were recorded to first isolate cortical motor areas and then assess responses to movement observation within these predefined areas (mirror neurons are expected to respond both during observation and execution of a movement). In the third protocol, an imitation condition was added and the responses during imitation were compared with responses during observation and during execution with the logic that mirror neurons should be more active during simultaneous observation and execution than during execution or observation alone (Buccino et al. 2004b; Carr et al. 2003; Grezes et al. 2003; Iacoboni et al. 1999; Leslie et al. 2004; Tanaka and Inui 2002).

These fMRI studies consistently reported that two cortical areas respond during movement observation, execution, and imitation: the anterior intraparietal sulcus (aIPS) and the ventral premotor (vPM). Most of the studies proposed that these areas compose the human "mirror system," implicitly suggesting that they contain mirror neurons. Further neuroimaging studies have attempted to characterize the responses of the human "mirror system" during observation, execution, and imitation of different movements. For example, comparing mirror system responses to hand movements that have a goal versus movements that do not (Iacoboni et al. 2005) or in which movements are made by a human arm versus by a robot arm (Gazzola et al. 2007). The majority of these studies interpreted stronger mirror system responses to particular conditions as evidence of mirror neuron preference for particular types of movements rather than others.

There are two concerns with these early experiments and the interpretation of their results. The first is that these experiments were unable to measure exclusive mirror neuron activity. For example, the typical results of passive movement observation and imitation experiments reveal strong responses in many cortical areas, including areas that are not believed to contain mirror neurons (e.g., primary visual cortex). This is clear evidence that many other neurons (e.g., visual neurons) in diverse cortical areas respond strongly during these tasks. Limiting the analysis to

cortical areas that respond during movement execution (by masking out areas that do not) does focus the analysis on more meaningful brain areas but does not solve the problem. As mentioned above, 90% of neurons in “mirror system” areas are not mirror neurons but are rather visual neurons that respond only during movement observation, motor neurons that respond only during movement execution, or visuomotor neurons that respond during both but do not respond selectively to movement identity (e.g., neurons selective for object rather than grasp type (Murata et al. 2000)). Since neuroimaging methods record the average response of very large neural populations, how can one know if the reported responses were generated by the activity of mirror neurons or by the activity of any of these other intermingled neural populations? For example, if a study reports that mirror system areas exhibit stronger fMRI responses during observation of movements with goals than movements without goals (e.g., Iacoboni et al. 2005), does it necessarily mean that mirror neurons responded more strongly? Or could it be that visual neurons responded more strongly because video clips of movements with goals contain richer visual stimuli than video clips of movements without goals?

The second, and perhaps more important concern, is that these studies did not assess movement selectivity. Movement selectivity is a defining physiological signature of mirror neurons in the monkey and is of central importance for the hypothesis that mirror neurons play a role in mapping perception to action. If mirror neurons indeed enable understanding and/or imitation of movements, subpopulations of mirror neurons must distinguish between different movements such that particular mirror neurons must respond selectively to particular movements (Dinstein 2008; Dinstein et al. 2008b). Without clear response selectivity across observation and execution, the mirror neuron theory falls apart. Imagine, for example, a situation where you observe someone executing a “thumbs up” movement. In order to properly understand that movement by simulation, it is critical that you activate the correct motor neurons encoding that particular movement. If you accidentally activate another group of

motor neurons that encode the “thumbs down” movement, you will arrive at the wrong conclusion regarding the intention of the person you are observing. Assessing movement selectivity in mirror system areas is, therefore, a crucial step for confirming or refuting the mirror neuron theory (Dinstein 2008; Dinstein et al. 2008b).

A common method for assessing neural selectivity using fMRI takes advantage of the fact that sensory neurons adapt/habituate when their preferred stimulus is presented repeatedly (Grill-Spector 2006; Grill-Spector and Malach 2001; Krekelberg et al. 2006). Cortical areas containing mirror neurons may, therefore, exhibit reduced fMRI responses when the same movement is presented repeatedly (a single neural population responds repeatedly and adapts) in contrast to when different movements are presented (different neural populations respond and there is no adaptation). Mirror neurons may be expected to adapt during trials where the same movement is repeatedly observed, repeatedly executed, observed and then executed, or executed and then observed (cross-modal adaptation). Showing such response selectivity in mirror system areas would be strong evidence for the existence of mirror neurons and would also offer a tool for further characterizing their selectivity for particular types of movements. For example, does adaptation occur across different movements that share a common goal (waving “hello” with either left or right hand) but not across different movements that do not (waving “hello” with left hand and waving “come here” with right hand). Such results would offer important evidence regarding the movement characteristics that are encoded by human mirror neurons.

Several recent studies have used fMRI adaptation protocols to assess response selectivity for different hand movements. These studies have reported visual (Dinstein et al. 2007; Hamilton and Grafton 2006; Shmuelof and Zohary 2005), motor (Dinstein et al. 2007; Hamilton and Grafton 2009), and cross-modal (Chong et al. 2008; Kilner et al. 2009; Lingnau et al. 2009) adaptation in mirror system areas during observation and execution of different hand movements. While some of the studies used object-oriented hand

movements (e.g., pulling, gripping, and grasping objects) (Hamilton and Grafton 2006; Kilner et al. 2009; Shmuelof and Zohary 2005), others used symbolic or gestural hand movements (e.g., thumbs up, rock, paper, scissors) (Chong et al. 2008; Dinstein et al. 2007; Hamilton and Grafton 2009; Lingnau et al. 2009). A consistent finding across all studies was that mirror system areas exhibited visual adaptation when the same movement was observed repeatedly and motor adaptation when the same movement was executed repeatedly. Such adaptation could hypothetically take place in two independent neural populations, one visual and the other motor, or in a single neural population of mirror neurons. While these results do not prove the existence of mirror neurons in humans, they do suggest that neurons with visual and motor movement selectivity are intermingled within mirror system areas in humans. This is somewhat encouraging because a fundamental feature of cortical organization is that neighboring neurons are strongly connected and perform common computations. The existence of neighboring intermingled neurons with movement selectivity, therefore, does suggest the existence of some form of mirror mechanism in humans. More persuasive is a recent study using object-oriented hand movements, which reported visual and motor adaptation as well as both forms of cross-modal adaptation in mirror system areas (Kilner et al. 2009). Cross-modal adaptation could only take place in a mirror neuron population that adapts when observing and then executing or when executing and then observing the same hand movement. Taken together, this evidence suggests that mirror neurons do exist in humans and that, similarly to mirror neurons found in the macaque monkey, they respond selectively to movements involving objects. It is not clear whether there are mirror neurons in humans that respond selectively to gestural and/or communicative movements.

Another method for assessing neural selectivity using fMRI involves the comparison of voxel-by-voxel response patterns using different classification techniques. This analysis is particularly useful in situations where subpopulations of neurons with different selectivity are distributed

unevenly within multiple neighboring voxels. In such cases, each subpopulation is expected to generate a unique fMRI response pattern that appears in trials containing its preferred stimulus and is distinct from the pattern generated by neighboring subpopulations in trials containing their preferred stimuli. This technique was successfully used, for example, to study the selectivity of human visual cortex neurons to visual orientations (Kamitani and Tong 2005), visual motion (Kamitani and Tong 2006), and visual categories (Haxby et al. 2001).

Two recent fMRI studies have used this technique to assess selectivity for different hand movements in mirror system areas during both observation and execution. The main difference between the studies was that in the first, subjects observed and executed symbolic hand movements (rock, paper, or scissors), while in the second, movements were oriented toward objects (grasping or pulling objects). The first study reported that classification of the fMRI response patterns in aIPS enabled accurate identification of the movement observed or performed on each trial. This shows that each movement was associated with a unique spatial response pattern generated by a unique and selective neural subpopulation (Dinstein et al. 2008a). If aIPS were to contain only mirror neurons, an identical fMRI response pattern would be expected during observation and execution of the same movement. This, however, was not the case as accurate movement identification by response pattern was possible only within the visual or motor modality but not across modalities. The second study, using object-oriented movements, did report accurate movement classification in aIPS both when assessing responses within each modality as well as across the two modalities (Oosterhof et al. 2010). These results seem to suggest a dissociation between responses to symbolic hand movements and object-oriented hand movements. Again, clear evidence supporting the existence of mirror neurons in humans seems to exist when considering object-oriented movements rather than gestural movements.

To conclude, the adaptation and classification studies seem to present converging evidence

suggesting that, like the monkey, humans have mirror neurons that respond selectively to different movements involving object manipulation. The case for mirror neurons selective for gestural and communicative movements seems less clear and is somewhat at odds with the mirror system hypothesis suggesting that mirror neurons enable social communication. Proper social communication is mostly based on correct interpretation of body language, gestures, and expressions rather than interpretation of object-oriented movements such as grasping or pulling.

Pathophysiology

Mirror System Dysfunction in Autism?

Impaired social interaction is one of the three core behavioral symptoms of autism (DSM-IV-TR 2000). Impaired imitation may also be a characteristic of autism, although there is controversy regarding its consistency (Hamilton et al. 2007). According to the “dysfunctional mirror system theory of autism,” these behavioral problems are caused by dysfunctional mirror neurons that impair the individual’s ability to simulate observed movements/actions (Williams et al. 2001). The idea is that inaccurate simulation or perhaps even a complete lack of simulation disrupts the ability to appropriately interpret the goals, intentions, and emotions associated with observed hand gestures, facial expressions, and body language of others. This theory has received an extraordinary amount of attention in both the popular press (Blakeslee 2006; Ramachandran and Oberman 2006) and the scientific press (Buccino and Amore 2008; Iacoboni and Dapretto 2006; Iacoboni and Mazziotta 2007; Keysers and Gazzola 2006; Le Bel et al. 2009; Oberman and Ramachandran 2007; Perkins et al. 2010; Rizzolatti and Fabbri-Destro 2008, 2009; Rizzolatti et al. 2009; Williams 2008; Williams et al. 2001). The empirical evidence supporting it, however, has been remarkably sparse and inconsistent.

Evidence in support of the dysfunctional mirror system theory of autism has come from several EEG/MEG (Honaga et al. 2010; Martineau

et al. 2008; Nishitani et al. 2004; Oberman et al. 2005) and fMRI (Dapretto et al. 2006; Hadjikhani et al. 2007; Schulte-Ruther et al. 2011) studies that have reported significantly weaker responses in mirror system areas of individuals with autism, as compared to controls, during passive observation or active imitation of hand movements or facial expressions. Weaker mirror system responses in autism have been interpreted as evidence for weak mirror neuron responses that are unable to complete the proper simulation processes supposedly needed to understand the meaning of the observed movements or imitate them. There have been, however, a similar number of EEG/MEG (Avikainen et al. 1999; Fan et al. 2010; Oberman et al. 2008; Raymaekers et al. 2009) and fMRI (Dinstein et al. 2010; Marsh and Hamilton 2011; Williams et al. 2006) studies that have reported equivalent mirror system responses in autism and control groups and one fMRI study that has even reported significantly stronger mirror system responses in autism as compared to controls during the observation of hand movements (Martineau et al. 2010). As a side note, it is interesting that all of the studies above, regardless of the reported response strength in autism, used gestural hand movements, finger tapping, or facial expressions rather than object-oriented movements. The mixed results were, therefore, not a consequence of using different movements.

Numerous methodological issues could have generated the heterogeneous results described above. For example, it is difficult to control the behavior of subjects during EEG and fMRI experiments. When subjects are asked to imitate a movement, delays in the timing or length of movement execution may greatly impact the estimated brain response. If subjects with autism imitate the movement later or more slowly than controls, their estimated brain responses will seem weaker because of a temporal mismatch between the expected and actual brain response. In such a situation, reduced mirror system responses would have little to do with mirror system abnormalities in autism and a lot to do with a different choice of behavioral response in

autism. Regardless of the true reason underlying the reduced responses, the results discussed above clearly show that individuals with autism can exhibit normal mirror system responses under some experimental conditions (Avikainen et al. 1999; Dinstein et al. 2010; Fan et al. 2010; Marsh and Hamilton 2011; Martineau et al. 2010; Oberman et al. 2008; Raymaekers et al. 2009; Williams et al. 2006). This argues against the claim of a generally dysfunctional mirror system in autism.

As discussed previously, perhaps the biggest concern with the interpretation of the studies above is their lack of ability to isolate mirror neuron responses and their lack of ability to assess movement selectivity. With this in mind, the reports above actually say very little about mirror neuron integrity in autism. Weak, normal, or excessive fMRI responses in mirror system areas could be generated by different combinations of visual neuron responses during observation and motor neuron responses during imitation. There is, therefore, no reason to assume that any of the reported differences in fMRI responses between autism and control groups were due to differences in mirror neuron responses. Furthermore, these studies do not offer any evidence for determining whether neural subpopulations in mirror system areas exhibit normal or abnormal movement selectivity in autism. The “dysfunctional mirror system theory of autism” would predict weak or even a complete lack of movement-selective responses in autism, which would be expected to yield an impaired simulation process. Is this indeed the case?

A recent fMRI adaptation experiment attempted to address this particular question and reported that mirror system responses in individuals with autism exhibited equivalent movement selectivity to that found in controls (Dinstein et al. 2010). In this study, individuals with autism exhibited not only strong mirror system responses during the observation of symbolic hand movements (e.g., rock, paper, scissors, thumbs up), but they also exhibited adaptation in trials where the same movement was repeatedly observed or repeatedly executed. These adaptation results were interpreted as evidence for typical

movement selectivity in mirror system areas of individuals with autism. Since selectivity is one of the two defining characteristics of mirror neuron responses, these data offer strong evidence against a general mirror system dysfunction in autism. All of the high-functioning individuals with autism who participated in this study exhibited ADOS scores that were well above the criteria for autism rather than the milder diagnoses of PDD-NOS and Asperger’s syndrome. These results, therefore, cannot be attributed to less severe behavioral symptoms in the participating subjects. Note that this study, like all other mirror system studies in autism, used gestural hand movements rather than object-oriented movements. Since cross-modal adaptation is a key signature of mirror neurons, which seems to be apparent only in responses to object-oriented movements, it would be important to also determine the integrity of such responses in autism.

Finally and more generally, it is important to consider how useful it is to describe autism as a disorder of one particular neural population such as mirror neurons. Most of the symptoms associated with autism do not seem related to mirror neurons at all. These include the core diagnostic symptoms of repetitive behaviors and language impairments (DSM-IV-TR 2000) as well as the commonly described secondary symptoms, which include sensory hypo/hypersensitivities (Jones et al. 2003; Kanner 1943; Minshew and Hobson 2008; O’Neill and Jones 1997), sleep problems (Cortesi et al. 2010; Richdale and Schreck 2009), and gastrointestinal problems (Buie et al. 2010a, b). Furthermore, much of the research into autism suggests that multiple neural abnormalities exist in many brain areas. fMRI studies have reported abnormally weak neural responses in autism not only in mirror system areas but also in superior temporal sulcus (Pelphrey and Carter 2008), face processing areas (Dawson et al. 2005), “mentalizing” areas (Marsh and Hamilton 2011), and cingulate cortex (Chiu et al. 2008), among others. Reports of abnormal functional (Minshew and Keller 2010) and anatomical (Courchesne et al. 2007) connectivity have suggested that neural communication and synchronization may be altered (mostly decreased) across multiple brain areas in autism. Developmental

studies have reported early overgrowth of gray and white matter in frontal cortex, temporal cortex, and cerebellum, which is followed by arrested growth during adolescence (Courchesne et al. 2004). Genetic studies have reported numerous mutations, deletions, and single-nucleotide polymorphisms that may increase the risk of developing autism. Most of the associated genes are involved in general cellular development including dendritic growth and synaptic maturation (Geschwind and Levitt 2007). Such genetic abnormalities would be expected to create widespread excitation-inhibition imbalances (Rubenstein and Merzenich 2003) as well as abnormalities in the neural architecture and connectivity of multiple brain areas, thereby, impacting multiple behaviors (Bourgeron 2009). Finally, individuals with autism have abnormally high chances of developing epilepsy throughout life. It is estimated that 20–30% of individuals with autism have epileptic seizures at some point in life and that up to 60–70% may exhibit abnormal epileptiform-like EEG recordings (Tuchman and Rapin 2002). When considering this larger breadth of evidence, it seems that describing autism as a disorder of mirror neurons may not be particularly useful in capturing the multitude of behavioral and physiological elements associated with the disorder.

To conclude, despite its surprising popularity (Iacoboni and Dapretto 2006; Iacoboni and Mazziotta 2007; Rizzolatti and Fabbri-Destro 2008, 2009; Williams et al. 2001), at present, the evidence supporting the “dysfunctional mirror system hypothesis of autism” seems rather sparse, inconsistent, and controversial (Baird et al. 2011; Dinstein et al. 2008b; Hamilton 2009; Hamilton et al. 2007). Further assessment of response selectivity in autism using adaptation and classification techniques is warranted in order to reveal useful data that will support or refute the hypothesis. The integrity of mirror system selectivity in autism should be tested with gestural hand movements and facial expressions, which seem particularly relevant to the social symptoms of autism, as well as with object-oriented movements, which seem to be the most effective movements for generating mirror neuron activity in both monkeys and humans.

See Also

- Cerebral Cortex
- Mirror Neurons
- Superior Temporal Sulcus Region

References and Reading

- Avikainen, S., Kulomaki, T., & Hari, R. (1999). Normal movement reading in Asperger subjects. *Neuroreport*, 10, 3467–3470.
- Baird, A. D., Scheffer, I. E., & Wilson, S. J. (2011). Mirror neuron system involvement in empathy: A critical look at the evidence. *Social Neuroscience*, 6, 327–335. <http://www.ncbi.nlm.nih.gov/pubmed/21229470>
- Blakeslee, S. (2006). Cells that read minds. *New York Times*. <http://www.nytimes.com/2006/01/10/science/10mirr.html?pagewanted=all>
- Bourgeron, T. (2009). A synaptic trek to autism. *Current Opinion in Neurobiology*, 19, 231–234.
- Buccino, G., & Amore, M. (2008). Mirror neurons and the understanding of behavioural symptoms in psychiatric disorders. *Current Opinion in Psychiatry*, 21, 281–285.
- Buccino, G., Binkofski, F., Fink, G. R., Fadiga, L., Fogassi, L., Gallese, V., et al. (2001). Action observation activates premotor and parietal areas in a somatotopic manner: An fMRI study. *European Journal of Neuroscience*, 13, 400–404.
- Buccino, G., Lui, F., Canessa, N., Patteri, I., Lagravinese, G., Benuzzi, F., et al. (2004a). Neural circuits involved in the recognition of actions performed by non-conspecifics: An FMRI study. *Journal of Cognitive Neuroscience*, 16, 114–126.
- Buccino, G., Vogt, S., Ritzl, A., Fink, G. R., Zilles, K., Freund, H. J., et al. (2004b). Neural circuits underlying imitation learning of hand actions: An event-related fMRI study. *Neuron*, 42, 323–334.
- Buie, T., Campbell, D. B., Fuchs, G. J., 3rd, Furuta, G. T., Levy, J., Vandewater, J., et al. (2010a). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125(Suppl. 1), S1–S18.
- Buie, T., Fuchs, G. J., 3rd, Furuta, G. T., Kooros, K., Levy, J., Lewis, J. D., et al. (2010b). Recommendations for evaluation and treatment of common gastrointestinal problems in children with ASDs. *Pediatrics*, 125 (Suppl. 1), S19–S29.
- Caggiano, V., Fogassi, L., Rizzolatti, G., Thier, P., & Casile, A. (2009). Mirror neurons differentially encode the peripersonal and extrapersonal space of monkeys. *Science*, 324, 403–406.
- Carr, L., Iacoboni, M., Dubeau, M. C., Mazziotta, J. C., & Lenzi, G. L. (2003). Neural mechanisms of empathy in humans: A relay from neural systems for imitation to limbic areas. *Proceedings of the National Academy of*

- Sciences of the United States of America*, 100, 5497–5502.
- Castiello, U. (2005). The neuroscience of grasping. *Nature Reviews Neuroscience*, 6, 726–736.
- Chiu, P. H., Kayali, M. A., Kishida, K. T., Tomlin, D., Klinger, L. G., Klinger, M. R., et al. (2008). Self responses along cingulate cortex reveal quantitative neural phenotype for high-functioning autism. *Neuron*, 57, 463–473.
- Chong, T. T., Cunnington, R., Williams, M. A., Kanwisher, N., & Mattingley, J. B. (2008). fMRI adaptation reveals mirror neurons in human inferior parietal cortex. *Current Biology*, 18, 1576–1580.
- Cortesi, F., Giannotti, F., Ivanenko, A., & Johnson, K. (2010). Sleep in children with autistic spectrum disorder. *Sleep Medicine*, 11, 659–664.
- Courchesne, E., Redcay, E., & Kennedy, D. P. (2004). The autistic brain: Birth through adulthood. *Current Opinion in Neurology*, 17, 489–496.
- Courchesne, E., Pierce, K., Schumann, C. M., Redcay, E., Buckwalter, J. A., Kennedy, D. P., et al. (2007). Mapping early brain development in autism. *Neuron*, 56, 399–413.
- Dapretto, M., Davies, M. S., Pfeifer, J. H., Scott, A. A., Sigman, M., Bookheimer, S. Y., et al. (2006). Understanding emotions in others: Mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neuroscience*, 9, 28–30.
- Dawson, G., Webb, S. J., & McPartland, J. (2005). Understanding the nature of face processing impairment in autism: Insights from behavioral and electrophysiological studies. *Developmental Neuropsychology*, 27, 403–424.
- Dinstein, I. (2008). Human cortex: Reflections of mirror neurons. *Current Biology*, 18, R956–R959.
- Dinstein, I., Hasson, U., Rubin, N., & Heeger, D. J. (2007). Brain areas selective for both observed and executed movements. *Journal of Neurophysiology*, 98, 1415–1427.
- Dinstein, I., Gardner, J. L., Jazayeri, M., & Heeger, D. J. (2008a). Executed and observed movements have different distributed representations in human aIPS. *Journal of Neuroscience*, 28, 11231–11239.
- Dinstein, I., Thomas, C., Behrmann, M., & Heeger, D. J. (2008b). A mirror up to nature. *Current Biology*, 18, R13–R18.
- Dinstein, I., Thomas, C., Humphreys, K., Minshew, N., Behrmann, M., & Heeger, D. J. (2010). Normal movement selectivity in autism. *Neuron*, 66, 461–469.
- DSM-IV-TR. (2000). *Diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Press.
- Fan, Y. T., Decety, J., Yang, C. Y., Liu, J. L., & Cheng, Y. (2010). Unbroken mirror neurons in autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 51, 981–988.
- Ferrari, P. F., Gallese, V., Rizzolatti, G., & Fogassi, L. (2003). Mirror neurons responding to the observation of ingestive and communicative mouth actions in the monkey ventral premotor cortex. *European Journal of Neuroscience*, 17, 1703–1714.
- Ferrari, P. F., Rozzi, S., & Fogassi, L. (2005). Mirror neurons responding to observation of actions made with tools in monkey ventral premotor cortex. *Journal of Cognitive Neuroscience*, 17, 212–226.
- Ferrari, P. F., Visalberghi, E., Paukner, A., Fogassi, L., Ruggiero, A., & Suomi, S. J. (2006). Neonatal imitation in rhesus macaques. *PLoS Biology*, 4, e302.
- Fogassi, L., Gallese, V., Fadiga, L., & Rizzolatti, G. (1998). Neurons responding to the sight of goal-directed hand/arm actions in the parietal area PF (7b) of the macaque monkey. *Society for Neuroscience Abstracts*, 24, 257.
- Fogassi, L., Ferrari, P. F., Gesierich, B., Rozzi, S., Chersi, F., & Rizzolatti, G. (2005). Parietal lobe: From action organization to intention understanding. *Science*, 308, 662–667.
- Gallese, V., Fadiga, L., Fogassi, L., & Rizzolatti, G. (1996). Action recognition in the premotor cortex. *Brain*, 119 (Pt 2), 593–609.
- Gazzola, V., Rizzolatti, G., Wicker, B., & Keysers, C. (2007). The anthropomorphic brain: The mirror neuron system responds to human and robotic actions. *NeuroImage*, 35, 1674–1684.
- Geschwind, D. H., & Levitt, P. (2007). Autism spectrum disorders: Developmental disconnection syndromes. *Current Opinion in Neurobiology*, 17, 103–111.
- Grezes, J., Armony, J. L., Rowe, J., & Passingham, R. E. (2003). Activations related to “mirror” and “canonical” neurones in the human brain: An fMRI study. *NeuroImage*, 18, 928–937.
- Grill-Spector, K. (2006). Selectivity of adaptation in single units: Implications for fMRI experiments. *Neuron*, 49, 170–171.
- Grill-Spector, K., & Malach, R. (2001). fMR-adaptation: A tool for studying the functional properties of human cortical neurons. *Acta Psychologica*, 107, 293–321.
- Hadjikhani, N., Joseph, R. M., Snyder, J., & Tager-Flusberg, H. (2007). Abnormal activation of the social brain during face perception in autism. *Human Brain Mapping*, 28, 441–449.
- Hamilton, A. F. (2009). Goals, intentions and mental states: Challenges for theories of autism. *Journal of Child Psychology and Psychiatry*, 50, 881–892.
- Hamilton, A. F., & Grafton, S. T. (2006). Goal representation in human anterior intraparietal sulcus. *Journal of Neuroscience*, 26, 1133–1137.
- Hamilton, A. F., & Grafton, S. T. (2009). Repetition suppression for performed hand gestures revealed by fMRI. *Human Brain Mapping*, 30, 2898–2906.
- Hamilton, A. F., Brindley, R. M., & Frith, U. (2007). Imitation and action understanding in autistic spectrum disorders: How valid is the hypothesis of a deficit in the mirror neuron system? *Neuropsychologia*, 45, 1859–1868.
- Haxby, J. V., Gobbini, M. I., Furey, M. L., Ishai, A., Schouten, J. L., & Pietrini, P. (2001). Distributed and overlapping representations of faces and objects in ventral temporal cortex. *Science*, 293, 2425–2430.

- Honaga, E., Ishii, R., Kurimoto, R., Canuet, L., Ikezawa, K., Takahashi, H., et al. (2010). Post-movement beta rebound abnormality as indicator of mirror neuron system dysfunction in autistic spectrum disorder: An MEG study. *Neuroscience Letters*, 478, 141–145.
- Iacoboni, M., & Dapretto, M. (2006). The mirror neuron system and the consequences of its dysfunction. *Nature Reviews Neuroscience*, 7, 942–951.
- Iacoboni, M., & Mazziotta, J. C. (2007). Mirror neuron system: Basic findings and clinical applications. *Annals of Neurology*, 62, 213–218.
- Iacoboni, M., Woods, R. P., Brass, M., Bekkering, H., Mazziotta, J. C., & Rizzolatti, G. (1999). Cortical mechanisms of human imitation. *Science*, 286, 2526–2528.
- Iacoboni, M., Molnar-Szakacs, I., Gallese, V., Buccino, G., Mazziotta, J. C., & Rizzolatti, G. (2005). Grasping the intentions of others with one's own mirror neuron system. *PLoS Biology*, 3, e79.
- Jones, R. S. P., Quigney, C., & Huws, J. C. (2003). First-hand accounts of sensory perceptual experiences in autism: A qualitative analysis. *Journal of Intellectual and Developmental Disability*, 28, 112–121.
- Kamitani, Y., & Tong, F. (2005). Decoding the visual and subjective contents of the human brain. *Nature Neuroscience*, 8, 679–685.
- Kamitani, Y., & Tong, F. (2006). Decoding seen and attended motion directions from activity in the human visual cortex. *Current Biology*, 16, 1096–1102.
- Kanner, L. (1943). Autistic disturbance of affective contact. *The Nervous Child*, 2, 217–240.
- Keysers, C., & Gazzola, V. (2006). Towards a unifying neural theory of social cognition. *Progress in Brain Research*, 156, 379–401.
- Kilner, J. M., Neal, A., Weiskopf, N., Friston, K. J., & Frith, C. D. (2009). Evidence of mirror neurons in human inferior frontal gyrus. *Journal of Neuroscience*, 29, 10153–10159.
- Kohler, E., Keysers, C., Umiltà, M. A., Fogassi, L., Gallese, V., & Rizzolatti, G. (2002). Hearing sounds, understanding actions: Action representation in mirror neurons. *Science*, 297, 846–848.
- Krekelberg, B., Boynton, G. M., & van Wezel, R. J. (2006). Adaptation: From single cells to bold signals. *Trends in Neurosciences*, 29, 250–256.
- Le Bel, R. M., Pineda, J. A., & Sharma, A. (2009). Motor-auditory-visual integration: The role of the human mirror neuron system in communication and communication disorders. *Journal of Communication Disorders*, 42, 299–304.
- Leslie, K. R., Johnson-Frey, S. H., & Grafton, S. T. (2004). Functional imaging of face and hand imitation: Towards a motor theory of empathy. *NeuroImage*, 21, 601–607.
- Lingnau, A., Gesierich, B., & Caramazza, A. (2009). Asymmetric fMRI adaptation reveals no evidence for mirror neurons in humans. *Proceedings of the National Academy of Sciences of the United States of America*, 106, 9925–9930.
- Marsh, L., & Hamilton, A. (2011). Dissociation of mirroring and mentalising systems in autism. *NeuroImage*, 56(3), 1511–1519.
- Martineau, J., Cochin, S., Magne, R., & Barthelemy, C. (2008). Impaired cortical activation in autistic children: Is the mirror neuron system involved? *International Journal of Psychophysiology*, 68, 35–40.
- Martineau, J., Andersson, F., Barthelemy, C., Cottier, J. P., & Destrieux, C. (2010). Atypical activation of the mirror neuron system during perception of hand motion in autism. *Brain Research*, 1320, 168–175.
- Minshew, N. J., & Hobson, J. A. (2008). Sensory sensitivities and performance on sensory perceptual tasks in high-functioning individuals with autism. *Journal of Autism and Developmental Disorders*, 38, 1485–1498.
- Minshew, N. J., & Keller, T. A. (2010). The nature of brain dysfunction in autism: Functional brain imaging studies. *Current Opinion in Neurology*, 23, 124–130.
- Murata, A., Gallese, V., Luppino, G., Kaseda, M., & Sakata, H. (2000). Selectivity for the shape, size, and orientation of objects for grasping in neurons of monkey parietal area AIP. *Journal of Neurophysiology*, 83, 2580–2601.
- Nishitani, N., Avikainen, S., & Hari, R. (2004). Abnormal imitation-related cortical activation sequences in Asperger's syndrome. *Annals of Neurology*, 55, 558–562.
- O'Neill, M., & Jones, R. S. (1997). Sensory-perceptual abnormalities in autism: A case for more research? *Journal of Autism and Developmental Disorders*, 27, 283–293.
- Oberman, L. M., & Ramachandran, V. S. (2007). The simulating social mind: The role of the mirror neuron system and simulation in the social and communicative deficits of autism spectrum disorders. *Psychological Bulletin*, 133, 310–327.
- Oberman, L. M., Hubbard, E. M., McCleery, J. P., Altschuler, E. L., Ramachandran, V. S., & Pineda, J. A. (2005). EEG evidence for mirror neuron dysfunction in autism spectrum disorders. *Brain Research. Cognitive Brain Research*, 24, 190–198.
- Oberman, L. M., Ramachandran, V. S., & Pineda, J. A. (2008). Modulation of mu suppression in children with autism spectrum disorders in response to familiar or unfamiliar stimuli: The mirror neuron hypothesis. *Neuropsychologia*, 46, 1558–1565.
- Oosterhof, N. N., Wiggett, A. J., Diedrichsen, J., Tipper, S. P., & Downing, P. E. (2010). Surface-based information mapping reveals crossmodal vision-action representations in human parietal and occipitotemporal cortex. *Journal of Neurophysiology*, 104, 1077–1089.
- Pelphrey, K. A., & Carter, E. J. (2008). Brain mechanisms for social perception: Lessons from autism and typical development. *Annals of the New York Academy of Sciences*, 1145, 283–299.
- Perkins, T., Stokes, M., McGillivray, J., & Bittar, R. (2010). Mirror neuron dysfunction in autism spectrum disorders. *Journal of Clinical Neuroscience*, 17, 1239–1243.

- Ramachandran, V. S., & Oberman, L. M. (2006). Broken mirrors: A theory of autism. *Scientific American*, 295, 62–69.
- Raymaekers, R., Wiersema, J. R., & Roeyers, H. (2009). EEG study of the mirror neuron system in children with high functioning autism. *Brain Research*, 1304, 113–121.
- Richdale, A. L., & Schreck, K. A. (2009). Sleep problems in autism spectrum disorders: Prevalence, nature, & possible biopsychosocial aetiologies. *Sleep Medicine Reviews*, 13, 403–411.
- Rizzolatti, G., & Craighero, L. (2004). The mirror-neuron system. *Annual Review of Neuroscience*, 27, 169–192.
- Rizzolatti, G., & Fabbri-Destro, M. (2008). The mirror system and its role in social cognition. *Current Opinion in Neurobiology*, 18, 179–184.
- Rizzolatti, G., & Fabbri-Destro, M. (2009). Mirror neurons: From discovery to autism. *Experimental Brain Research*, 200, 223–237.
- Rizzolatti, G., & Sinigaglia, C. (2010). The functional role of the parieto-frontal mirror circuit: Interpretations and misinterpretations. *Nature Reviews Neuroscience*, 11, 264–274.
- Rizzolatti, G., Fabbri-Destro, M., & Cattaneo, L. (2009). Mirror neurons and their clinical relevance. *Nature Clinical Practice Neurology*, 5, 24–34.
- Rubenstein, J. L., & Merzenich, M. M. (2003). Model of autism: Increased ratio of excitation/inhibition in key neural systems. *Genes, Brain, and Behavior*, 2, 255–267.
- Schulte-Ruther, M., Greimel, E., Markowitsch, H. J., Kamp-Becker, I., Remschmidt, H., Fink, G. R., et al. (2011). Dysfunctions in brain networks supporting empathy: An fMRI study in adults with autism spectrum disorders. *Social Neuroscience*, 6, 1–21.
- Shmuelof, L., & Zohary, E. (2005). Dissociation between ventral and dorsal fMRI activation during object and action recognition. *Neuron*, 47, 457–470.
- Tanaka, S., & Inui, T. (2002). Cortical involvement for action imitation of hand/arm postures versus finger configurations: An fMRI study. *Neuroreport*, 13, 1599–1602.
- Tuchman, R., & Rapin, I. (2002). Epilepsy in autism. *Lancet Neurology*, 1, 352–358.
- Umiltà, M. A., Kohler, E., Gallese, V., Fogassi, L., Fadiga, L., Keysers, C., et al. (2001). I know what you are doing. A neurophysiological study. *Neuron*, 31, 155–165.
- Williams, J. H. (2008). Self-other relations in social development and autism: Multiple roles for mirror neurons and other brain bases. *Autism Research*, 1, 73–90.
- Williams, J. H., Whiten, A., Suddendorf, T., & Perrett, D. I. (2001). Imitation, mirror neurons and autism. *Neuroscience and Biobehavioral Reviews*, 25, 287–295.
- Williams, J. H., Waite, G. D., Gilchrist, A., Perrett, D. I., Murray, A. D., & Whiten, A. (2006). Neural mechanisms of imitation and “mirror neuron” functioning in autistic spectrum disorder. *Neuropsychologia*, 44, 610–621.

Mirror Neurons

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Synonyms

[Mirror neuron system](#)

Definition

Mirror neurons were first reported in macaque monkeys in 1992 and subsequently identified in humans. First localized to the ventral premotor area, the Mirror Neuron System (MNS) has also been linked to the inferior parietal lobule, the dorsal premotor, and primary motor cortex, among other regions. Mirror neurons modulate their activity in at least two instances: when an individual executes a motor action and when observing the same (or similar) action performed by another individual. In this way, their activity is modulated by both action execution and observation of an action, which distinguishes mirror neurons from other neurons that discharge with either execution or observation, but not both. Observed actions are directly mapped onto the MNS of the observer, suggestive of the intimately intertwined natures of action execution and observation. Further, it suggests that the interpretation of the actions of others may require the use of our own motor system.

The MNS has been proposed as a potential etiology for Autism Spectrum Disorder (ASD), though it is a subject of fervent debate. The MNS is thought to encode the goal of an action, not just the motor component, meaning that the MNS contains a mechanism by which one understands other people, and for imitating them. As such, the MNS is thought to be a key component of social cognition. Because poor social cognition is a hallmark of ASD, researchers have theorized that a dysfunctional MNS may be a neurobiological basis for the disorder. This hypothesis is commonly called the Broken Mirror Theory (BMT). One researcher distinguishes three

different variants of the BMT, each of which makes a different claim about the mechanism by which a disrupted MNS contributes to ASD. The imitation hypothesis suggests that an altered MNS leads to poor self-other mapping that is thought to be at the heart of abnormalities in social cognition in ASD (e.g., theory of mind). The simulation hypothesis is predicated on the notion that the MNS provides the basis for simulating other individuals. A disrupted MNS, it is theorized, leads to an inability to simulate not only actions but also mental and emotional states. The action chain variant rests on the claim that some neurons within the MNS are only activated when an action is embedded in a sequence or chain. When a neuron of this type is activated at the beginning of an action sequence, the individual is able to predict how the entire chain of events will unfold. Disruptions in this specific part of the MNS lead to an inability to understand how a given action is embedded in a particular context, thus leading to difficulties in social cognition.

Though the data is far from conclusive, evidence for an abnormal MNS in individuals with ASD can be found across multiple methodologies, including electroencephalography, magnetoencephalography, transcranial magnetic stimulation, eye tracking, structural MRI, and functional MRI.

See Also

- [Empathy](#)
- [Theory of Mind](#)

References and Reading

- Dapretto, M., Davies, M. S., Pfeifer, J. H., Scott, A. A., Sigman, M., Bookheimer, S. Y., & Iacoboni, M. (2006). Understanding emotions in others: Mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neuroscience*, 9, 28–30.
- Hamilton, A. F. D. C. (2013). Reflecting on the mirror neuron system in autism: A systematic review of current theories. *Developmental Cognitive Neuroscience*, 3, 91.
- Kana, R. K., Wadsworth, H. M., & Travers, B. G. (2011). A systems level analysis of the mirror neuron hypothesis and imitation impairments in autism spectrum disorders. *Neuroscience and Biobehavioral Reviews*, 35, 894.
- Kilner, J. M., & Lemon, R. N. (2013). What we know currently about mirror neurons. *Current Biology*, 23, R1057–R1062.
- Rizzolatti, G., & Fabbri-Destro, M. (2010). Mirror neurons: From discovery to autism. *Experimental Brain Research*, 200, 223.
- Williams, J. H. G., Waiter, G. D., Gilchrist, A., Perrett, D. I., Murray, A. D., & Whiten, A. (2006). Neural mechanisms of imitation and “mirror neuron” functioning in autistic spectrum disorder. *Neuropsychologia*, 44, 610–621.

Mirtazapine

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Synonyms

[Remeron](#)

Definition

An antidepressant medication. Mirtazapine exerts its antidepressant effect by inhibiting alpha-2 adrenergic presynaptic receptors, resulting in increased levels of serotonin and norepinephrine. This is an alternate mechanism to that used by SSRI medications. Additionally, mirtazapine blocks H-1 histamine receptors, which may contribute to its sedating effects. Mirtazapine is also associated with increased appetite, higher risk of weight gain, dry mouth, and constipation.

See Also

- [Antidepressants](#)

References and Reading

- Stahl, S. (2009). *The prescriber's guide: Stahl's essential psychopharmacology* (pp. 347–351). Cambridge: Cambridge University Press.

Mismatch Negativity

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Definition

The mismatch negativity (MMN) is an event-related potential (ERP) evoked in response to a perceived change in sensory stimuli. It is commonly elicited in an oddball paradigm in which a standard stimulus is paired with a deviant stimulus, the standard being presented the majority of instances. It generally occurs 150–250 ms after the presentation of the deviant stimulus, though in children its latency is delayed to between 200 and 300 ms. The MMN is typically evaluated by calculating a difference wave, computed by subtracting the ERP in response to the standard stimulus from the ERP to the deviant stimulus.

Historical Background

Electrophysiology provides an avenue to examine mental functioning that may not be manifested behaviorally. It allows for the investigation of aspects of cognition, perception, and other biological bases for behavior. This is especially useful in assessing infant and clinical populations that may lack the requisite capacities for expression.

Electroencephalography (EEG) consists of recording the brain's electrical activity. In humans, this is accomplished by placing electrodes at various locations on the scalp. Electrical surface recordings are limited in terms of spatial resolution, in that it is difficult to precisely identify the source of electrical fields in the brain. However, electrical recordings can provide excellent temporal resolution, revealing the brain's response effectively in real time.

Event-related potentials (ERPs) are electrical activity time-locked to a particular event. ERPs are commonly utilized to assess the brain's processing of external stimuli. In order to demonstrate that the recorded response is related to the target event and not to extraneous influences, a series of trials are averaged together to create a composite waveform. Waveforms can be averaged across trials within a single subject, as well as across individuals and groups.

The mismatch negativity (MMN) was first described by Näätänen and colleagues in a 1978 paper examining negative potentials via EEG in response to deviations in tone frequency. The MMN refers to an automatic neurological response to a change in auditory stimuli, represented by a peak difference generally observed 150–250 ms poststimulus. The degree of variance between the stimuli required to elicit the neural MMN response is approximately the difference required for behavioral discrimination.

ERPs such as the MMN are assessed in terms of amplitude – power or voltage, and latency – timing. MMN amplitude has been associated with degree of deviant contrast and accuracy of discrimination, with increases in amplitude associated with greater contrast and greater discrimination, and decreases in amplitude associated with reduced contrast and reduced discrimination. Earlier latency has also been associated with greater stimulus contrast.

The MMN has been demonstrated in response to wide variety of stimuli. These include computer-generated sounds such as sinusoidal tones and musical chords, as well as human speech sounds such as vowels and consonant phonemes. Among these stimuli, the MMN has been shown to be sensitive to changes in frequency, pitch, intensity, duration, and presentation order. More recently, researchers have begun to explore MMN equivalents in response to visual and olfactory stimuli.

The MMN has been consistently elicited independent of directed attention. Further, it has been demonstrated in the context of multiple-competing auditory streams. However, competing auditory stimuli can diminish MMN amplitude. Competing visual stimuli generally do not impact

the MMN. Conversely, variations in background noise which elicit the MMN response can impact attention, reorienting a person to an anomalous environmental stimulus.

This highlights an important developmental and operational function of the MMN. The MMN represents the brain's perception of an anomalous event. It is often followed by a positive peak occurring between 250 and 300 ms. This positive potential, known as the P3a, is an electrophysiological marker of novelty and represents the automatic reorienting of attention. Thus, the MMN is a functional precursor, evidencing the automatic perception of a deviant stimulus, leading to the actual reorienting of attention as evidenced by the P3a (for review, see Friedman et al. 2001).

The source of the MMN appears to be in the auditory cortex, as evidenced by scalp distribution data, studies of magnetic encephalography, intracranial recordings, and cases of brain lesions. Additional data reveal the frontal lobe as a contributing generator. It is suggested that the frontal lobe involvement may be related to attentional switching associated with the MMN (Alho 1995). Other studies have evidenced that the MMN response elicited by speech sounds is primarily sourced in the left temporal lobe, whereas MMN in response to nonspeech sounds is primarily sourced in the right temporal lobe (Kujala 2007).

Various studies have demonstrated the effects of training on the MMN. Individuals, who at the beginning of a session could not behaviorally discriminate between divergent stimuli and failed to exhibit a corresponding MMN, later learned through exposure to behaviorally discriminate between the stimuli and exhibited a parallel MMN response. This further demonstrates the value of the MMN as a real-time index of learning and discrimination.

Current Knowledge

As an electrophysiological index of auditory perception that closely matches behaviorally observed capabilities, the MMN provides a

valuable tool in the biological assessment of language and associated capacities. It has accordingly been utilized in the scientific examination of autism spectrum disorders (ASD). Given that deficits in language and social orienting are prominent features of autism spectrum disorders, a contributing factor in these impairments may be an inability to perceive and process contrasting auditory stimuli, especially speech sounds. Thus, the MMN provides a potential method for exploring auditory processing in ASD.

A limited number of studies have examined the MMN in ASD. Results from these studies have not been entirely consistent, though a variety of studies have demonstrated MMN and other ERP differences in ASD subjects as compared with controls. These have included differences in amplitude, latency, and hemispheric distribution. Divergent findings may be due to differences in paradigm design, participant characteristics, and diagnostic inclusion criteria.

A series of studies have shown enhanced MMN in ASD (Ferri et al. 2003; Jansson-Verkasalo et al. 2003; Kujala et al. 2007, 2010; Lepisto et al. 2005, 2006, 2007, 2008). The most common finding among these studies is an increase in amplitude, particularly over the right hemisphere. Some studies have also evidenced shorter latencies (Ferri et al. 2003; Jansson-Verkasalo et al. 2003; Kujala et al. 2007; Lepisto et al. 2005, 2007). This is consistent with behavioral findings indicating heightened auditory discrimination abilities in ASD (Gomot et al. 2002). Enhanced perceptual ability may in fact adversely impact language learning, reflecting an inability to ignore irrelevant stimuli.

Other studies have failed to show MMN or shown diminished MMN amplitude in ASD (Dunn et al. 2008; Kuhl et al. 2005; Lepisto et al. 2005, 2006). These findings are generally interpreted to evidence reduced discrimination, with obvious implications for language deficits.

In a noteworthy study, Kuhl et al. (2005) examined MMN and behavioral speech preferences in a sample of school-aged children with and without ASD. The paradigm involved an auditory preference task, assessing a child's preference for speech or nonspeech stimuli, and an ERP task,

assessing MMN responses to changes in consonant-vowel speech sounds. On a group level, children with ASD exhibited a preference for nonspeech sounds and failed to exhibit MMN. Typically developing controls, chronologically and mentally age-matched, exhibited a preference for speech sounds and exhibited a typical MMN response. Interestingly, when the ASD group was subdivided based on speech preference, the children with ASD who preferred speech sounds exhibited MMN responses similar to typical controls. According to the authors, this finding provides evidence for a relationship between phonetic learning and social interaction, suggesting that social interest may have a direct impact on language learning. This further substantiates the MMN as a neural correlate of broader language capacities with potential social implications.

Another study replicated previous findings demonstrating diminished MMN amplitude in ASD subjects in response to unattended stimuli. However, when subjects were instructed to attend to the stimuli, the MMN for ASD subjects was similar to controls (Dunn et al. 2008). This indicates that auditory processing that occurs automatically in typical individuals may require attention to be operative in ASD. This presents a significant disadvantage noting that direct attentional capacities are inherently limited, and thus processing capability may suffer in complex environments. This study also provided an explanation for discrepant findings in previous literature, noting that a prominent study that failed to show MMN differences in ASD had procedurally instructed participants to attend to the stimuli.

MMN may change over time in ASD. Two studies were conducted utilizing the same experimental paradigm with adults and children respectively, diagnosed with Asperger's. MMN responses were recorded to changes in duration and pitch. Both samples demonstrated enhanced MMN to pitch changes. However, duration changes showed a diminished MMN in children and an enhanced MMN in adults (Lepisto et al. 2006, 2007). These findings imply that MMN may have a complex developmental course in ASD.

Many studies revealed differences in hemispheric distribution in ASD as compared with control samples (Gomot et al. 2002; Jansson-Verkasalo et al. 2003; Kujala et al. 2007, 2010; Lepisto et al. 2006, 2007). As a trend, these included enhanced responses over the right hemisphere and diminished responses over the left hemisphere. Whereas control groups often exhibited no hemisphere dominance for the MMN, ASD groups exhibited right hemisphere dominance (Kujala et al. 2007; Lepisto et al. 2006, 2007). These abnormal patterns of MMN lateralization may reflect impaired interhemispheric processing.

Future Directions

Some of the difficulties in interpreting the multiplicity of findings with regard to the MMN in ASD are likely related to varying diagnostic criteria across studies. This is an issue that affects ASD research across the board, given the etiological and clinical heterogeneity of ASD. Utilizing instruments that involve standardized behavioral observation and standardized interviewing, in conjunction with expert clinical judgment, can better constrain research populations ensuring more consistent ASD samples.

Though increased amplitude and shorter latency are associated with greater discrimination, when an increase in amplitude is coupled with extended latency or, conversely, when a decrease in amplitude is coupled with shorter latency, the specific implication with regard to discrimination is unclear. Though differences in amplitude and latency in ASD manifest "abnormal processing," future research may be able to determine more precisely how variations in electrophysiological components relate to global processing.

The MMN can be recorded in young infants reliably between 3 and 5 months. Some studies have even elicited MMN correlates in fetuses. As more specific clinical electrophysiological profiles emerge, MMN may be a promising tool for revealing clinical disorders early in development, leaving open the prospect of increasingly early intervention.

Training and learning have been shown to improve MMN responsiveness. This may indicate an opportunity for intervention in clinical disorders. In ASD specifically, directed attention has been shown to positively impact the MMN (Dunn et al. 2008). Future research could focus on the evaluation of intervention effects in clinical populations using the MMN as a marker. This may provide information that can be translated into evidence-based intervention methodologies, as well as general information about the nature of auditory processing deficits in these populations.

Researchers have also begun exploring methods for assessing MMN at the single-subject level. Currently, the MMN provides a reliable tool for examining group differences in clinical populations. As this methodology progresses, it may be able to reliably identify an individual's auditory discrimination capacity. Such an electrophysiological marker could have useful implications for diagnosis and intervention in ASD.

See Also

- ▶ Auditory Discrimination
- ▶ Auditory Potentials
- ▶ Auditory Processing
- ▶ Neurophysiology

References and Reading

- Alho, K. (1995). Cerebral generators of mismatch negativity (MMN) and its magnetic counterpart (MMNm) elicited by sound changes. *Ear and Hearing*, 16(1), 38–51.
- Bomba, M. D., & Pang, E. W. (2004). Cortical auditory evoked potentials in autism: A review. *International Journal of Psychophysiology*, 53(3), 161–169.
- Dunn, M. A., Gomes, H., & Gravel, J. (2008). Mismatch negativity in children with autism and typical development. *Journal of Autism and Developmental Disorders*, 38(1), 52–71.
- Ferri, R., Elia, M., Agarwal, N., Lanuzza, B., Musumeci, S. A., & Pennisi, G. (2003). The mismatch negativity and the P3a components of the auditory event-related potentials in autistic low-functioning subjects. *Clinical Neurophysiology*, 114, 1671–1680.
- Friedman, D., Cycowicz, Y. M., & Gaeta, H. (2001). The novelty P3: An event-related brain potential (ERP) sign of the brain's evaluation of novelty. *Neuroscience and Biobehavioral Reviews*, 25(4), 355–373.
- Gomot, M., Giard, M. H., Adrien, J. L., Barthelemy, C., & Bruneau, N. (2002). Hypersensitivity to acoustic change in children with autism: Electrophysiological evidence of left frontal cortex dysfunctioning. *Psychophysiology*, 39, 577–584.
- Jansson-Verkasalo, E., Ceponiene, R., Kielinen, M., Suominen, K., Jantti, V., Linna, S. L., et al. (2003). Deficient auditory processing in children with Asperger syndrome, as indexed by event-related potentials. *Neuroscience Letters*, 338, 197–200.
- Kuhl, P., Coffey-Corina, S., Padden, D., & Dawson, G. (2005). Links between social and linguistic processing of speech in preschool children with autism: Behavioral and electrophysiological measures. *Developmental Science*, 8, F1–F12.
- Kujala, T. (2007). The role of early auditory discrimination deficits in language disorders. *Journal of Psychophysiology*, 21(3–4), 239–250.
- Kujala, T., Tervaniemi, M., & Schröger, E. (2007). The mismatch negativity in cognitive and clinical neuroscience: Theoretical and methodological considerations. *Biological Psychology*, 74(1), 1–19.
- Kujala, T., Kuuluvainen, S., Saalasti, S., Lepistö, T., Jansson-Verkasalo, E., & Wendt, L. V. (2010). Speech-feature discrimination in children with Asperger syndrome as determined with the multi-feature mismatch negativity paradigm. *Clinical Neurophysiology*, 121(9), 1410–1419.
- Lepistö, T., Kujala, T., Vanhala, R., Alku, P., Huotilainen, M., & Näätänen, R. (2005). The discrimination of and orienting to speech and non-speech sounds in children with autism. *Brain Research*, 1066, 147–157.
- Lepistö, T., Silokallio, S., Nieminen-von Wendt, T., Alku, P., Näätänen, R., & Kujala, T. (2006). Auditory perception and attention as reflected by the brain event-related potentials in children with Asperger syndrome. *Clinical Neurophysiology*, 117, 2161–2217.
- Lepistö, T., Nieminen-von Wendt, T., von Wendt, L., Näätänen, R., & Kujala, T. (2007). Auditory cortical change detection in adults with Asperger syndrome. *Neuroscience Letters*, 414(2), 136–140.
- Lepistö, T., Kajander, M., Vanhala, R., Alku, P., Huotilainen, M., Näätänen, R., et al. (2008). The perception of invariant speech features in children with autism. *Biological Psychology*, 77(1), 25–31.
- Näätänen, R., Gaillard, A. W. K., & Mantysalo, S. (1978). Early selective attention effects on the evoked potential: A critical review and reinterpretation. *Biological Psychology*, 8, 81–136.
- Näätänen, R., Paavilainen, P., Rinne, T., & Alho, K. (2007). The mismatch negativity (MMN) in basic research of central auditory processing: A review. *Clinical Neurophysiology*, 118(12), 2544–2590.

Misophonia

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Synonyms

[Hatred of sound](#); [Sound sensitivity](#)

Definition

Misophonia is characterized by an individual's sensitivity to/negative reaction to a sound or sounds (Cavanna and Seri 2015). It can be observed in isolation or in combination with other disorders, like autism spectrum disorder. Unfortunately, research on the condition is rather limited and there is not yet agreement on how this phenomenon/condition is best conceptualized, i.e., as a condition in its own right or as a feature of other disorders. The condition is not yet recognized in official diagnostic systems. The term literally means "hatred of sound" and was first proposed in 2000 and noted the combination negative emotions/reactions to specific sound(s) as the hallmark feature (Cavanna and Seri 2015).

Given the lack of consensus on the nature of the condition and its definition, the literature on this condition/phenomenon is rather limited. In some cases, the condition's severity or presentation presents a major problem for the individual, and various treatment strategies have been proposed, e.g., cognitive behavior therapy or exposure therapy (McGuire et al. 2015; Kamody and Del Conte 2017; Bruxner 2016).

The sounds that trigger the negative reactions can be either loud or soft. In one study, the most commonly reported sounds relate to eating and/or the mouth, e.g., sounds of eating, chewing, whispering (Bruxner 2016).

Understanding of the trigger sound(s) remains limited. Individuals with the condition may be quite aware that it is disruptive. Often attempts to avoid the sound triggers can lead to unanticipated avoidance behaviors that further restrict interaction with others.

Given the lack of consensus on the nature of the condition, there are no data, at present, on epidemiology, gender differences, and so forth. While, in some respects, clearly related to anxiety disorders, the fundamental nature of Misophonia remains to be established. As research on this condition/phenomenon continues, it is likely that there will be greater clarity on issues including (a) best approaches to definition, (b) clinical features and associations with other disorders, (c) epidemiology and differences in rates by gender, race, and so forth, and (d) frequent co-morbidities of the conditions.

See Also

► [Functional Behavior-Based Cognitive-Behavioral Therapy for Obsessive-Compulsive Behavior in Children with ASD](#)

References and Reading

- Brout, J. J., Edelstein, M., Erfanian, M., Mannino, M., Miller, L. J., Rouw, R., Kumar, S., & Rosenthal, M. Z. (2018). Investigating misophonia: A review of the empirical literature, clinical implications, and a research agenda. *Frontiers in Neuroscience*, 12, 36.
- Bruxner, G. (2016). 'Mastication rage': A review of misophonia – An under-recognised symptom of psychiatric relevance? *Australasian Psychiatry*, 24, 195.
- Cavanna, A. E., & Seri, S. (2015). Misophonia: Current perspectives. *Neuropsychiatric Disease & Treatment*, 11, 2117–2123.
- Kamody, R. C., & Del Conte, G. S. (2017). Using dialectical behavior therapy to treat misophonia in adolescence. *The Primary Care Companion for CNS Disorders*, 19(5).
- McGuire, J. F., Wu, M. S., & Storch, E. A. (2015). Cognitive-behavioral therapy for 2 youths with misophonia. *The Journal of Clinical Psychiatry*, 76, 573.

Mitochondrial Deficits/Disorders

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Synonyms

Organelle

Definition

Mitochondria are the central hub for energy metabolism in the cell providing the main source of ATP and TCA intermediates for several anabolic and catabolic processes (Siddiqui et al. 2016). Mitochondrial dysfunction can occur due to genetic or biochemical abnormalities affecting ATP synthesis. Specifically, several reports showed abnormalities in autism spectrum disorder (ASD) patients involved in phospholipid production (Siddiqui et al. 2016). Further analysis of N-acetyl-aspartate, a marker for mitochondrial dysfunction, showed elevations in the white and gray matter cortex of ASD patients (Siddiqui et al. 2016). Other studies examining the electron transport chain of mitochondria isolated from ASD patients showed decreases in metabolic activity of several enzymes and transport complexes throughout the brain (Siddiqui et al. 2016). This dysfunction of enzymes and transport activity is believed to contribute to the increased reactive oxygen species and oxidative stress observed in ASD neurological studies. Several mutations associated with the electron transport chain, including complexes III, IV, and VII, showed decreased activity and expression in ASD patients. The results suggest global metabolic abnormalities within the mitochondria, and perhaps the cytoplasm may alter neuronal activity and connectivity within the brains of ASD patients.

See Also

- ▶ Autonomic Nervous System
- ▶ Citrate and Autism

References and Reading

Siddiqui, M. F., Elwell, C., & Johnson, M. H. (2016). Mitochondrial dysfunction in autism spectrum disorders. *Autism-Open Access*, 6(4). <https://doi.org/10.4172/2165-7890.1000190>.

Mixture Modeling

- ▶ Latent Variable Modeling

MLD

- ▶ Metachromatic Leukodystrophy

Moban

- ▶ Molindone

Mobile Work Crew Model

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Synonyms

Community work crew; Community-based work team; Enclave; Supported employment group; Work team

Definition

A mobile work crew model follows the practice of placing together several individuals to work in the community as a team or “crew.” Typically, the individuals have a disability and are accompanied by a nondisabled support person or job coach. This type of work group falls under the category of supported employment. The mobile work crews may work at various locations but often specialize in a specific area such as janitorial services, lawn care, maintenance jobs, etc. These jobs are in the community but may not be integrated with employees outside the work crew and may not require competitive rates of production. The mobile work crews are often associated with an agency providing vocational or day services to people with autism. In some cases, individuals with autism may be able to learn needed vocational skills while on a mobile work crew to lead to a higher level of employment or competitive employment.

See Also

- [Employment](#)
- [Employment in Adult Life](#)
- [Vocational Training](#)

References and Reading

- Keel, J., & Mesibov, G. B. (1997). TEACCH-supported employment program. *Journal of Autism and Developmental Disorders*, 27(1), 3–9.
- Lou, B., Kim, F., Joanne, S., Michele, Z., & Tracy, K. (1999). Work–wage relationships and individuals with disabilities. *Journal of Vocational Rehabilitation*, 13(1), 5.
- Manhood, L. M., & Howlin, P. (1999). The outcome of a supported employment scheme for high functioning adults with autism or Asperger syndrome. *Autism: International Journal of Research and Practice*, 3, 229–253.
- Rogan, P., & Murphy, S. (1991). Support employment and vocational rehabilitation: Merger or misadventure? *Journal of Rehabilitation*, 57, 2.

Möbius Syndrome

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Synonyms

[Moebius syndrome](#)

Definition

This very rare congenital disorder (perhaps 1 in 100,000 births) is characterized by facial paralysis and a lack of ability to move the eyes from side to side. This is a result of failure of normal development of the sixth and seventh cranial nerves. Sometimes, other facial nerves are also affected – if the eighth nerve is affected, hear loss is present as well.

Abnormalities in other areas can include problems with the chest wall and extremities. Given the lack of facial expression, most individuals with the condition are assumed not to have normal intelligence, but this is not correct. The condition is named after the neurologist who described it in the last 1800s. Treatment is supportive in nature. The etiology of the syndrome remains unclear with some suggestion of genetic links as well as links to teratogenic drugs such as thalidomide.

A few studies, mostly case reports and at least one case series, have suggested a potential increased risk for autism. The significance of this association remains unclear.

References and Reading

- Johansson, M., Wentz, E., Fernell, E., Stromland, K., Miller, M. T., & Gillberg, C. (2001). Autistic spectrum disorders in Mobius sequence: A comprehensive study of 25 individuals [Research Support, Non-U.S. Gov't]. *Developmental Medicine & Child Neurology*, 43(5), 338–345.

- Miller, G. (2007). Neurological disorders. The mystery of the missing smile [News]. *Science*, 316(5826), 826–827.
- Stromland, K., Sjogreen, L., Miller, M., Gillberg, C., Wentz, E., Johansson, M., et al. (2002). Mobius sequence – A Swedish multidiscipline study [Research Support, Non-U.S. Gov't]. *European Journal of Paediatric Neurology*, 6(1), 35–45.

Modeling

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Synonyms

[Example, demonstration; Representation](#)

Definition

Modeling refers to an intervention procedure that provides an example or explicitly demonstrates a verbal or social behavior targeted for intervention. The demonstration serves as a standard for imitation or comparison by the learner by providing an example of the structure or content of a targeted language or social behavior. The modeled demonstration can be presented either verbally or visually through pictures or videos. The modeling procedure may take one of six forms: model-recast, model-prompt-imitation, aided modeling, interactive modeling, in vivo modeling, and video modeling.

- Model-recast is a method that provides a model of the structural or semantic properties of language for the child by recasting a child's utterance during discourse interaction. The recast/model supplies missing grammatical aspects or semantic relationships while maintaining the child's central meaning. For example, if the child omits the auxiliary verb and present progressive tense marker when

saying “doggie eat,” the adult recast or model would maintain the meaning but provide the missing grammatical markers, as in “The doggie is eating.”

- Model-prompt-imitation is an elicitation procedure that presents a target language or social behavior and elicits the targeted behavior from the child in a model-prompt-imitation format. For example, if the target language behavior is the appropriate response to question forms, the clinician or adult may model the expected response and prompt the child to imitate as in “What is your name?” followed by the model “My name is Jack,” and then a prompt to imitate: Tell me: “My name is Jack.”
- Aided modeling occurs during instruction in the use of an augmentative and alternative communication (AAC) device wherein the adult uses the AAC device as well as speech to demonstrate the expected communication output.
- Interactive modeling facilitates language development in a socially interactive format. The adult follows the child's lead and provides a verbal gloss of the actions and focused attention to objects by the child. For example, if a child looks at or picks up an object, the adult verbalizes or glosses the child's actions or attention focus by modeling the appropriate language, for example, “Ball,” “This is a ball.” If the child performs an action on the object, the adult would provide a language model or gloss of the action, such as “throw ball,” “Jack is throwing the ball.”
- In vivo modeling involves the real-life situational observation of typical persons performing a task.
- Video modeling involves a child viewing a video demonstration of a target behavior that serves as a model for imitation and comparison.

Modeling has been found to be an effective intervention technique for children and adolescents with autism, especially the use of model-prompt-imitation, aided modeling, in vivo modeling, and video modeling. Less is known

regarding the use of the model-recast method in teaching verbal skills to children with autism spectrum disorders.

See Also

- [Augmentative and Assistive Technology](#)
- [Imitation](#)
- [Picture Exchange Communication System](#)
- [Prompt Hierarchy](#)
- [Video Instruction](#)

References and Reading

- Charlop-Christy, M. H., Le, L., & Freeman, K. A. (2000). A comparison of video modeling with in vivo modeling for teaching children with autism. *Journal of Autism and Developmental Disorders*, 30, 537–552.
- Drager, K. D. R. (2009). Aided modeling interventions for children with autism spectrum disorders who require AAC. *Perspectives on Augmentative and Alternative Communications, American Speech, Language, and Hearing Association*, 18, 114–120.
- Kouri, T. A. (2005). Lexical training through modeling and elicitation procedures with late talkers who have specific language impairment and developmental delays. *Journal of Speech and Hearing Research*, 48, 151–171.
- Matson, J., Matson, M. L., & Rivet, T. T. (2007). Social-skills treatments for children with autism spectrum disorders: An overview. *Behavior Modification*, 31, 682–707.
- Williams, G., Donley, C. R., & Keller, J. W. (2000). Teaching children with autism to ask questions about hidden objects. *Journal of Applied Behavior Analysis*, 33, 627–630.

Modification

- [Reasonable Accommodation](#)

Modifications

- [Modified Testing](#)
- [Special Needs](#)

Modified Checklist for Autism in Toddlers (M-CHAT)

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Synonyms

CHAT; Checklist for autism in toddlers; M-CHAT

Abbreviations

AAP	American Academy of Pediatrics
ASD	Autism spectrum disorder
CESDD	Checklist for Early Signs of Developmental Disorders
ESAT	Early Screening of Autistic Traits questionnaire
FUI	Follow-up interview
NPV	Negative predictive value
PEDS	Parents' Evaluation of Developmental Status
PPV	Positive predictive value
Se	Sensitivity
Sp	Specificity

Description

The Modified Checklist for Autism in Toddlers (M-CHAT; Robins et al. 1999a) is a screening measure developed to identify young children with an elevated risk for autism spectrum disorder (ASD) through parent report. This instrument is one of the most commonly used screening instruments for ASD in toddlers worldwide.

The checklist was developed and validated for children between 16 and 30 months old. Parents are asked to answer 23 yes/no questions about the usual behavior of their child. Responses of parents indicating typical development are balanced. Children screen positive for ASD if they fail three or more items in total or if they fail two or more out of six critical items, derived from discriminant function analysis. These six critical

items ask about the child's interest in other children, response to name being called, following a point of the parent, own pointing and showing to indicate interest, and imitation (Robins et al. 2001b).

In addition to the parent report, a follow-up interview (M-CHAT FUI; Robins et al. 1999b) was designed to ascertain responses of parents for screen positive children. Parents are asked to clarify the items failed by their child in a structured format. To ensure presence or absence of specific behaviors, parents are asked to give some examples of these behaviors. In addition, detailed information about the frequency of the behaviors is elicited. Including this follow-up interview in the screening lowers the amount of false positive screens, without compromising the sensitivity of the instrument (see also the section on “[Psychometric Data](#)”). This interview can be conducted in person or over the phone (Robins and Dumont-Mathieu 2006; Robins et al. 2001b).

Historical Background

The M-CHAT is an adaptation and extension of the Checklist for Autism in Toddlers (CHAT; Baron-Cohen et al. 1992; Baron-Cohen et al. 1996). The CHAT was originally designed to screen for autism, not ASD, in 18-month-olds. The child's general physician or health visitor completes the checklist, for example during the 18-month routine check-up. The CHAT consists of two parts: nine parent questions (part A) and five observation items that have to be administered by trained professionals (part B). For more details about this measure, see entry ► “[CHAT](#).”

The M-CHAT modified the CHAT into an instrument completely reliant on parent report, because this may be more accurate than a brief observation by a professional (Robins and Dumont-Mathieu 2006). The first nine items of the CHAT (part A) were included as such in the M-CHAT. In addition, 21 new items were formulated to compensate for the loss of the observation part (part B) of the CHAT, but also to broaden the signs of ASD included in the checklist. The authors of the M-CHAT wanted to identify a

greater range of children on the autism spectrum, not solely children with autism. Based on preliminary analyses of the first 600 children, eight items were dropped because they lacked discriminant power or because parents misunderstood their content. In addition, one item about social referencing was added, leading to the final 23 items in the checklist (Robins et al. 2001b).

Since the development of the M-CHAT, the instrument has been translated into more than 30 languages, including Arabic, French, Dutch, German, Spanish, Portuguese, Chinese, and Japanese. The checklist itself, the follow-up interview, and the different translations are available for free download for clinical, research, and educational purposes (see www.mchatscreen.com). For use with Chinese and Japanese children, some adaptations to the M-CHAT were made. For the Japanese version, the M-CHAT was translated and some illustrations were added to the items about declarative pointing, showing, gaze following, and social referencing, in order to encourage caregivers to notice negative symptoms (Inada et al. 2011). For Chinese children, Wong et al. (2004) combined the M-CHAT and CHAT into a new screening measure: the CHAT-23. This instrument incorporates a Chinese translation of the 23 items of the M-CHAT (part A) and the five observation items of the CHAT (part B). The authors suggest using part A as a Level 1 screening instrument for use in the general population, followed by administration of part B as a Level 2 screening instrument for screen positive children on part A. For more details on the CHAT-23, see the entry ► “[CHAT](#).”

Psychometric Data

Discriminant Validity

Several studies have evaluated the psychometric properties of the M-CHAT as a screening instrument for ASD in toddlers. The original validation study included 1,122 children from a nonselected population and 171 high-risk children screened through early intervention service providers. All children were between 16 and 30 months old. There were 39 children identified with ASD in

this sample: three from the unselected sample and 36 from the high-risk group. None of the children in this study who were evaluated after screening positive had an entirely typical development. Robins et al. (2001b) estimated the psychometric properties of the M-CHAT through discriminant function analysis classification based on known diagnoses of ASD before follow-up of the entire sample. The sensitivity (Se) was estimated at .87, the specificity (Sp) at .99, the positive predictive value (PPV) at .80, and the negative predictive value (NPV) at .99. They used this approach because calculation of Se and Sp depends on follow-up of both positive and negative screen children to ascertain diagnosis in all children and follow-up was still pending. Dumont-Mathieu and Fein (2005) reported estimates on the Se and Sp of the M-CHAT based on the first 940 children of the original sample who were rescreened at age 4. This rescreen resulted in six possible missed cases. Se was estimated at .85 and Sp at .93.

A study by Kleinman et al. (2008) showed that the PPV of the M-CHAT depends on the inclusion of the follow-up interview and on the sample of children to which it is applied. These authors screened a completely new sample of children between 16 and 30 months old: 3,309 low-risk children from an unselected sample and 484 high-risk children from early intervention service providers. For the total sample, the PPV after a positive screen on the M-CHAT was only .36. However, if this positive screen was also confirmed by the follow-up interview, the PPV increased to .74. There were also some differences found in PPVs between the low- and high-risk groups. Without follow-up interview, the PPVs were .11 and .60 for, respectively, the low- and high-risk group. When positive screens were confirmed with the follow-up interview, the PPVs were .65 and .76 for, respectively, the low- and high-risk group.

Pandey et al. (2008) added 2,983 new children between 16 and 30 months old to the Kleinman et al. (2008) sample in order to compare the PPVs of younger (16–23 months old) versus older children (24–30 months old). In this study, all positive screen children were based on parent report and follow-up interview. In the high-risk sample, the

PPV was .79 for the younger children and .74 for the older children. In the low-risk sample, the PPV was .28 for the younger and .61 for the older children.

Although the M-CHAT has been translated in many different languages, the validation of these translations is still pending. Some findings on the Arabic, French, Portuguese, Spanish, Sinhala, and Japanese version have been published (Canal-Bedia et al. 2011; Eldin et al. 2008; Inada et al. 2011; Losapio and Pondé 2008; Perera et al. 2009; Rogé et al. 2009), but these findings were mostly based on small high-risk samples. Two recent published studies form an exception. Inada et al. (2011) reported on the discriminant validity of the Japanese version of the M-CHAT in 1,187 low-risk children of 18 months old, of whom 20 were diagnosed with ASD at age 3. These authors suggest a different cutoff of two or more failed items in total, at which the Se was estimated at .75, the Sp at .89, the PPV at .11, and the NPV at .99. Canal-Bedia et al. (2011) reported on the validity of the Spanish translation of the M-CHAT in two large samples, but the study lacked data on possible missed cases. Therefore, the Se and Sp cannot be estimated (reported Se was 1). The PPVs they reported were .35 for a first sample of 2,480 children, including 63 high-risk children recruited from early intervention service providers, and .19 for a completely unselected sample of 2,055 children.

Item Analysis

In the initial validation study, all but two items discriminated well between children with and children without ASD. Only the item about enjoyment in being swung or bounced on the knee of the parent and the item asking parents if their child can walk cannot distinguish children with and without ASD (Robins et al. 2001b).

Ventola et al. (2007) compared the scores of 195 children who screened positive on the M-CHAT between 16 and 30 months of age and who were subsequently diagnosed with either an ASD, a global developmental delay, or a developmental language disorder. Even when overall language level was controlled for, children with ASD could still be differentiated from children with other developmental disorders, based on their

scores on four items: response to name, declarative pointing, imperative pointing, and following a point.

Reliability

Two studies reported on the internal consistency of the M-CHAT. Robins et al. (2001b) calculated a Cronbach's alpha of .85 for the entire checklist and .83 for the six critical items. These results were replicated by Kleinman et al. (2008). They reported alpha's of .85 and .84, respectively, for the entire checklist and the six critical items.

Inada et al. (2011) looked at the interrater reliability of the Japanese translation of the M-CHAT by comparing the scoring of mothers and fathers of a limited subsample of 24 children. The scoring results of these parent couples were highly correlated: Pearson's $r = .93$. In addition, they looked at the test-retest reliability by asking 22 mothers to fill out the M-CHAT again after on average 8 days. Again, scores on the M-CHAT were highly correlated: Pearson's $r = .99$.

Clinical Uses

The M-CHAT has been available for both research and clinical use since the late 1990s. With the recent recommendations of the American Academy of Pediatrics (AAP) to conduct population-wide screening for ASD at 18 months (AAP 2006), the use of the M-CHAT as a Level I screening instrument for ASD has been recommended by some. However, validation of the M-CHAT is still ongoing and longitudinal follow-up will have to shed light on possible missed cases to enhance the accuracy of the estimates of sensitivity and specificity for the M-CHAT (see also Mawle and Griffiths 2006; Robins and Dumont-Mathieu 2006). Finally, validation of the different translations is still pending.

Based on the current findings, when using the M-CHAT in clinical practice as a Level 1 screening measure, one can expect many false positive screens, especially when used in unselected samples and when the user does not incorporate the follow-up interview in the screening. Therefore, Kleinman et al. (2008) suggested a higher cutoff

for failing when screening in the general population or to combine the use of the M-CHAT as a Level 1 screening instrument with a Level 2 screening instrument or more in-depth parent interview and behavior observation for borderline cases, before a full-scale autism evaluation is instituted.

Different studies used the M-CHAT as a Level 2 screening instrument. For example, Dereu et al. (2012) used the Checklist for Early Signs of Developmental Disorders (CESDD; Dereu et al. 2010) as a Level 1 screening instrument in an unselected sample of 7,092 infants and toddlers attending day-care centers, combined with the M-CHAT as a Level 2 screening instrument for positive screen children on the CESDD or for children at-risk for ASD because of a delayed language development. They compared the M-CHAT with the early screening of autistic traits questionnaire (ESAT) (Dietz et al. 2006; Swinkels et al. 2006) for a referred sample of 197 children between 16 and 30 months old based on the CESDD results and concluded that the discriminant power of these screening instruments for ASD were comparable, although some differences were found in the Se and the Sp of the different instruments. Glascoe et al. (2007) looked at combining a broadband developmental-behavioral screening test as a Level 1 screening instrument, the Parents' Evaluation of Developmental Status (PEDS), with the M-CHAT as a Level 2 screening instrument. These authors advised to base referrals on three or more discrete types of concerns on PEDS, to reduce over-referrals by 70%, while maintaining high levels of sensitivity (81%). However, Pinto-Martin et al. (2008) showed in their study that the PEDS missed the majority of children who screened positive for ASD on the M-CHAT. They concluded that this supports the use of an ASD-specific tool for all children in conjunction with regular standardized developmental screening.

See Also

- [CHAT](#)
- [Screening Measures](#)

References and Reading

- American Academy of Pediatrics, Council on Children With Disabilities, Section on Developmental Behavioral Pediatrics, Bright Futures Steering Committee, & Medical Home Initiatives for Children With Special Needs Project Advisory Committee. (2006). Identifying infants and young children with developmental disorders in the medical home: An algorithm for developmental surveillance and screening. *Pediatrics*, *118*, 405–420.
- Baron-Cohen, S., Allen, J., & Gillberg, C. (1992). Can autism be detected at 18 months? The needle, the haystack, and the CHAT. *The British Journal of Psychiatry*, *161*, 839–843.
- Baron-Cohen, S., Cox, A., Baird, G., Swettenham, J., Nightingale, N., Morgan, K., et al. (1996). Psychological markers in the detection of autism in infancy in a large population. *The British Journal of Psychiatry*, *168*, 158–163.
- Canal-Bedia, R., Garcia-Primo, P., Martin-Cilleros, V., Santos-Borbujo, J., Guisuraga-Fernandez, Z., Herraiz-Garcia, L., et al. (2011). Modified Checklist for Autism in Toddlers: Cross-cultural adaptation and validation in Spain. *Journal of Autism and Developmental Disorders*, *41*, 1342–1351.
- Charman, T., Baron-Cohen, S., Baird, G., Cox, A., Wheelwright, S., Swettenham, J., et al. (2001). Commentary: The Modified Checklist for Autism in Toddlers. *Journal of Autism and Developmental Disorders*, *31*, 145–148.
- Dereu, M., Warreyn, P., Raymaekers, R., Meirsschaut, M., Pattyn, G., Schietecatte, I., et al. (2010). Screening for autism spectrum disorders in Flemish day-care centres with the Checklist for Early Signs of Developmental Disorders. *Journal of Autism and Developmental Disorders*, *40*, 1247–1258.
- Dereu, M., Raymaekers, R., Warreyn, P., Schietecatte, I., Meirsschaut, M., & Roeyers, H. (2012). Can child care workers contribute to the early detection of autism spectrum disorders? A comparison between screening instruments with child care workers versus parents as informants. *Journal of Autism and Developmental Disorders*, *42*, 781–796.
- Dietz, C., Swinkels, S., van Daalen, E., van Engeland, H., & Buitelaar, J. K. (2006). Screening for autistic spectrum disorder in children aged 14 to 15 months. II: Population screening with the Early Screening of Autistic Traits questionnaire (ESAT). Design and general findings. *Journal of Autism and Developmental Disorders*, *36*, 713–722.
- Dumont-Mathieu, T., & Fein, D. (2005). Screening for autism in young children: The Modified Checklist for Autism in Toddlers (M-CHAT) and other measures. *Mental Retardation and Developmental Disabilities Research Reviews*, *11*, 253–262.
- Eldin, A. S., Habib, D., Noufal, A., Farrag, S., Bazaid, K., Al-Sharbati, M., et al. (2008). Use of M-CHAT for a multinational screening of young children with autism in the Arab countries. *International Review of Psychiatry*, *20*, 281–289.
- Glascoc, F. P., Marcias, M. M., Wegner, L. M., & Robertshaw, N. S. (2007). Can a broadband developmental-behavioral screening test identify children likely to have autism spectrum disorder? *Clinical Pediatrics*, *46*, 801–805.
- Inada, N., Koyama, T., Inokuchi, E., Kuroda, M., & Kamio, Y. (2011). Reliability and validity of the Japanese version of the Modified Checklist for Autism in Toddlers (M-CHAT). *Research in Autism Spectrum Disorders*, *5*, 330–336.
- Kleinman, J. M., Robins, D. L., Ventola, P. E., Pandey, J., Boorstein, H. C., Esser, E. L., et al. (2008). The Modified Checklist for Autism in Toddlers: A follow-up study investigating the early detection of autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *38*, 827–839.
- Losapio, M. F., & Pondé, M. P. (2008). Tradução para o Português da escala M-CHAT para rastreamento precoce de autismo [Translation into Portuguese of the M-CHAT Scale for early screening of autism]. *Revista de Psiquiatria do Rio Grande do Sul*, *30*, 221–229.
- Mawle, E., & Griffiths, P. (2006). Screening for autism in pre-school children in primary care: Systematic review of English language tools. *International Journal of Nursing Studies*, *43*, 623–636.
- Pandey, J., Verbalis, A., Robins, D. L., Boorstein, H., Klin, A., Babitz, T., et al. (2008). Screening for autism in older and younger toddlers with the Modified Checklist for Autism in Toddlers. *Autism*, *12*, 513–535.
- Perera, H., Wijewardena, K., & Aluthwelage, R. (2009). Screening of 18–24-month-old children for autism in a semi-urban community in Sri Lanka. *Journal of Tropical Pediatrics*, *55*, 402–405.
- Pinto-Martin, J. A., Young, L. M., Mandell, D. S., Poghosyan, L., Giarelli, E., & Levy, S. E. (2008). Screening strategies for autism spectrum disorders in pediatric primary care. *Journal of Developmental & Behavioral Pediatrics*, *29*, 345–350.
- Robins, D. L., & Dumont-Mathieu, T. M. (2006). Early screening for autism spectrum disorders: Update on the Modified Checklist for Autism in Toddlers and other measures. *Developmental and Behavioral Pediatrics*, *27*, 111–119.
- Robins, D. L., Fein, D., & Barton, M. (1999a). *Modified Checklist for Autism in Toddlers*. Self-published.
- Robins, D. L., Fein, D., & Barton, M. (1999b). *Follow-up interview for the Modified Checklist for Autism in Toddlers (M-CHAT FUI)*. Self-published.
- Robins, D. L., Fein, D., Barton, M. L., & Green, J. A. (2001a). Reply to Charman et al.'s commentary on the Modified Checklist for Autism in Toddlers. *Journal of Autism and Developmental Disorders*, *31*, 149–151.
- Robins, D. L., Fein, D., Barton, M. L., & Green, J. A. (2001b). The Modified Checklist for Autism in Toddlers: An initial study investigating the early detection of autism and pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, *31*, 131–144.

- Rogé, B., Chabrol, H., & Unsal, I. (2009). Le dépistage précoce de l'autisme: Quelle faisabilité? [Early screening autism: How far?]. *Enfance*, 61, 27–40.
- Swinkels, S. H. N., Dietz, C., van Daalen, E., Kerkhof, I. H. G. M., van Engeland, H., & Buitelaar, J. K. (2006). Screening for autistic spectrum in children aged 14 to 15 months. I: The development of the Early Screening of Autistic Traits questionnaire (ESAT). *Journal of Autism and Developmental Disorders*, 36, 723–732.
- Ventola, P., Kleinman, J., Pandey, J., Wilson, L., Esser, E., Boorstein, H., et al. (2007). Differentiating between autism spectrum disorders and other developmental disabilities in children who failed a screening instrument for ASD. *Journal of Autism and Developmental Disorders*, 37, 425–436.
- Wong, V., Hui, L. S., Lee, W., Leung, L. J., Ho, P. P., Lau, W. C., et al. (2004). A modified screening tool for autism (Checklist for Autism in Toddlers [CHAT-23]) for Chinese children. *Pediatrics*, 114, 166–176.

Modified Checklist for Autism in Toddlers, Revised: Spanish Cultural Validation

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Definition

The Modified Checklist for Autism in Toddlers, Revised – Spanish Cultural Validation is the Spanish version of the M-CHAT-R/F (Robins et al. 2014), a parental report screening questionnaire developed to identify children between 14 and 30 months at risk of autism spectrum disorder (ASD). It is one of the best-known and most

widely used ASD screening tools in the world, because it is concise, easy to administer, and available free of charge on the Internet (<https://mchatscreen.com/mchat-rf/translations/>). It is normally used as a screening tool in the general population (screening level 1) but can also be used to identify signs of ASD in high-risk groups (such as a level 2 screener). The tool consists of a questionnaire with 20 yes/no questions, each of which includes examples to make it easier for parents to understand the questions, and a follow-up interview (FUI) designed to verify the responses of parents who indicate suspected ASD. The questionnaire scoring system establishes three levels of risk according to the number of failed questions. If the questionnaire has two or fewer failed items, it is considered that there is no suspicion of autism; if the questionnaire has eight or more failed items, it is considered that there is a high risk of ASD, and the case should be referred directly for a diagnostic evaluation; finally, if the questionnaire has between three and seven failed items, it is considered that there is a medium risk, and the follow-up interview (FUI) should be administered to clarify with the parents, in a structured way, the failed items of their child. If, after the FUI has been administered, the questionnaire has two or more failed items, the child should be referred for diagnostic evaluation. The FUI can be administered in person or by telephone, and its use is intended to improve the psychometric properties of the tool and limit the costs of the screening program by trying to ensure fewer false positives.

Historical Background

In Spain, interest grew in fostering early detection of autism since the end of the last century, when retrospective studies on early signs became more numerous. Already in the twenty-first century, the first published articles on early detection experiences (Baird et al. 2000; Baron-Cohen et al. 2000; Robins et al. 2001), along with articles on the crucial role of parents in identifying warning signs, studies on factors influencing delayed diagnosis (Coonrod and Stone 2004; Mandell et al. 2002; Mandell et al. 2005), and recommendations

published by several professional organizations and consensus panels recommending systematic screening and early diagnosis (American Academy of Pediatrics Committee on Children with and Disabilities 2001; Filipek et al. 2000; Johnson et al. 2007), prompted interest in adapting a specific tool for early detection of autism in Spain. It is in this context that in 2005 the health authorities of the autonomous region of Castilla y León (Spain) decided to implement a population-based, pilot ASD screening program (for a description of this program, see García Primo et al. (2014)).

The ASD screening program in Spain is administered by primary care pediatricians within the Well Baby Check-up Program, which in Spain is universal and free and includes regular standardized tests that collect data on each child's developmental milestones. From 2005 to 2014, the ASD screening program used the first 23-item M-CHAT questionnaire (Robins et al. 2001) validated and culturally adapted to Spanish by Canal-Bedia et al. (2011). During that 9-year period, more than 9,524 children between 18 and 36 months participated in screening (García Primo et al. 2014).

The Spanish version of M-CHAT has psychometric properties similar to those of the original version, with high sensitivity and specificity, but, like the original version, has high rates of false positives, that is, cases in which the result of the questionnaire recommends referral for a follow-up interview due to suspicion of autism that later turn out not to have an ASD.

In order to streamline the M-CHAT process, The M-CHAT-R/F (Robins et al. 2014) was developed with a focus on reducing false-positive cases without affecting the efficacy of the questionnaire. False positives can be a large hindrance to the health system, especially in the case of diagnosing ASD, as each case needs a lot of time and resources to evaluate. The way the questionnaire is scored was also made easier, thus facilitating its implementation on a populational level.

From 2014 the M-CHAT-R/F Spanish version (Magán-Maganto et al. 2018) has replaced the 23-item M-CHAT and has been implemented in the same region of Spain. Including the Spanish version of the M-CHAT-R/F, a total of 16,149

children in the 14–36-month age range have been screened within the primary health-care system. The inclusion of M-CHAT and M-CHAT-R/F in the Well Baby Check-up Program has facilitated the work of pediatricians in identifying risk signs of ASD, provided a way for families to express concerns about their children's development by responding to the questionnaire, and allowed for coordination between diagnostic services and early intervention services.

Current Knowledge

For the cultural adaptation process, the M-CHAT-R/F was translated and back-translated by two bilingual professionals and compared with the original version. In translating the questionnaire and the FUI, the necessary cultural adaptations were made specially to adjust the examples of each item so that they were clear and understandable in the Spanish cultural context. Several professionals with expertise in child development assessment were also asked to judge the quality of the wording of the questions and examples in Spanish to describe the actions. Finally, 30 randomly selected families filled out the questionnaire to check that there were no significant problems in understanding the questions and examples.

In the validation process, 6,625 children aged between 14 and 36 months were screened. Their parents filled in the questionnaire at the pediatrician's office with their child when they attended for the routine checkup at 18 and 24 months. Also, in some cases, physicians administered the screening protocol before or after these regular visits due to family or professional concerns. The total number of positive cases was 54 (18 failed more than 8 items, indicating high risk; 32 failed 2 or more items after FUI (medium risk); and 4 were referred due to pediatrician concerns). Of those 54 positive cases, 39 (72%) completed the entire assessment protocol. In addition, nine evaluations were administered to children referred for evaluation as possible false negatives. A total of 19 children were diagnosed with ASD (15 true positives and 4 false negatives). The other 24 children who tested positive on the

questionnaire and did not receive a diagnosis of ASD were diagnosed with other disorders or developmental delay and were classified as false positive for ASD. None of the positive screening cases that were evaluated had typical development, suggesting that if the M-CHAT-R/F questionnaire gives a positive result, it is highly likely that the cases require some type of intervention, even if they do not have ASD.

Results of the M-CHAT-R/F Spanish validation were similar to the English version, with sensitivity (Sen) of 0.79, specificity (Spe) of 0.99, positive predictive value (PPV) of 0.39, and negative predictive value (NPV) of 0.99. However, the prevalence was slightly lower in the Spanish sample. Some additional analyses were performed, taking into account the excluded cases (e.g., not answering the phone for the FUI, rejection to attend further assessments, etc.) in order to avoid misrepresentation of the data sample distribution. Estimations of the proportion of cases were made in function of the excluded cases in each stage of the screening procedure. These are the results from the general sample: Sen of 0.86, Spe of 0.99, PPV of 0.41, and NPV of 0.99.

To analyze possible effects of age on the performance of the tool, the psychometrical properties of the M-CHAT-R/F Spanish version were estimated for two different age ranges (14–22 and 23–36 months). For the age range of 14–22 months, the internal consistency was Cronbach's $\alpha = 0.59$ (increasing to a Cronbach's $\alpha = 0.62$ when analyzing the items after FUI); a Sen value of 0.82; a Spe value of 0.99; and a PPV of 0.47. The values for the age range of 23–36 months were the following: Cronbach's $\alpha = 0.63$ (increasing to a Cronbach's $\alpha = 0.72$ after FUI); Sen of 0.75; Spe of 0.99; and PPV of 0.3. Discriminant analyses outcomes were similar in both age strata indicating that the M-CHAT-R/F questionnaire differentiates between ASD, other neurodevelopmental disorders, and language delays and screens negative groups; however the different clusters did have some overlapping with each other.

The results of these analyses showed higher accuracy for the younger age range, supporting the fact that M-CHAT-R/F is a suitable tool for early detection. Although other studies performed

with the original M-CHAT (Sunita and Bilszta 2013; Toh et al. 2018) have demonstrated better outcomes in older ages (24–36 months), there is no certainty as to why this is the case, but possible explanations for this are enhancements of the new version of the tool or in its implementation.

The Spanish validation supported the use of the M-CHAT-R/F as an ASD screening tool on a population level. One of the most important results of this validation was that the number of children that needed a follow-up interview was cut down by 15.1% compared to the old Spanish M-CHAT (Canal-Bedia et al. 2011). This fact provides evidence of the improvement that the new scoring system has given to the health system and affected families.

Professionals collaborating with the Spanish M-CHAT-R/F screening program commented that the combination of a standardized tool and training – focused on red flags of social and communication developmental difficulties at early ages – was key for ASD early detection. The results of the M-CHAT-R/F Spanish cultural validation support the utility of the screening program at a populational level within an NHS and its relevant use in the elaboration of prevention strategies and policies.

Future Directions

Both the original version and the Spanish version of M-CHAT-R/F have succeeded in reducing the number of false positives without this implying that the questionnaire loses psychometric properties (Magán-Maganto et al. 2018), which is an advance in relation to the objective of reducing detection costs. It is a question of reducing not only economic costs but also personal costs. With the use of M-CHAT-R/F, there may be fewer families unnecessarily concerned about unconfirmed suspicion than with previous versions of the same questionnaire. This is a crucial aspect for the development and improvement of universal screening tools that are used even with children with whom neither their parents nor professionals have raised previous concerns about their communicative and social development. From the point of view of personal costs, if a family is

concerned about their child's development, answering the M-CHAT-R/F questionnaire may be a way to resolve their uncertainty. If the questionnaire indicates risk of ASD, the child will be referred for evaluation to confirm or rule out the diagnosis of ASD. In either case, the family will resolve their uncertainty, regardless of whether the case is true or false positive. However, if the family does not have any prior concerns and the questionnaire indicates ASD risk, turning out to be a false-positive case, the use of the screening tool has created an unnecessary concern that did not exist before and that is detrimental. Therefore, the reduction in the number of false positives demonstrated by the validation study of the M-CHAT-R/F Spanish version is a valuable result, because it represents a step in the right direction to support the decision for using this questionnaire in universal screening programs.

Ideally, future versions of M-CHAT-R/F would evolve so that no family would have unnecessary concerns as a result of responding to the questionnaire. Future research should take this need into account, together with the need to reduce the economic costs of an excessive number of unnecessary diagnostic evaluations. That said, progress in reducing false positives should not be based simply on raising the cutoff point (i.e., setting a higher number of failed items to determine the risk of ASD), because that would imply a higher risk of increasing the number of false negatives (Ibañez et al. 2014). The strategy should be aimed at improving the wording of the items to ensure that parents correctly understand the questionnaire questions and that their answers actually represent the child's frequent behavior.

False-negative cases also represent a significant personal cost to families and the child who is excluded from early intervention. The identification of false negatives is also an economic and logistical challenge for any screening program and is key in determining the psychometric properties of a screening tool. In fact, few studies with M-CHAT-R/F have faced the challenge of systematically identifying false negatives and none in the long term. However, in order to know the value of this screening tool, it is necessary to carry out follow-up studies to assess how the cohort of

screening participants has evolved years after responding to M-CHAT-R/F. It is expected that in a few years, as was done with CHAT (Baird et al. 2000) or M-CHAT (Kleinman et al. 2008; Stenberg et al. 2014), some follow-up study on the results of M-CHAT-R/F screening will be published.

Likewise, it is hoped that in the future there will be the possibility to improve the systematization of the follow-up interview process, which should also respond to the purpose of making it easier for parents to better understand the items their child fails and to structure the discussion between the pediatrician and the parents, so that it is possible to share the same vision about the child's behavior and about the risk, sometimes subtle, that the child may be diagnosed with ASD.

In this sense, the M-CHAT-R/F Spanish version could evolve to become the key and initial element of a protocol aimed at facilitating a shared analysis between parents and pediatricians of the child's early communicative and social behavior, at different times of development (Magán-Maganto et al. 2017). The protocol should serve to better organize information and structure parent-pediatrician discussions about concerns related to the child's development. In addition, this approach could be framed in a developmental surveillance perspective in order to systematize information according to different trajectories and key moments of development (18, 24, 36 months, incorporation into kindergarten, preschool, access to primary school, etc.). For example, studies have already suggested that children with an ASD diagnosis and a higher intellectual functioning are detected at later ages when community strains start to be more complex (Christensen et al. 2016; Kleinman et al. 2008).

See Also

- [Checklist for Autism in Toddlers \(CHAT\)](#)
- [Early Diagnosis](#)
- [Modified Checklist for Autism in Toddlers \(M-CHAT\)](#)
- [Predictive Value of Screening Measures](#)
- [Screening Instruments for ASD](#)

- ▶ **Screening Measures**
- ▶ **Utility of Repeated Autism Screening at 18 and 24 months**

References and Reading

- American Academy of Pediatrics Committee on Children with, & Disabilities. (2001). The Pediatrician's role in the diagnosis and Management of Autistic Spectrum Disorder in children. *Pediatrics*, 107(5), 1221–1226. <https://doi.org/10.1542/peds.107.5.1221>.
- Baird, G., Charman, T., Baron-Cohen, S., Cox, A., Swettenham, J., Wheelwright, S., & Drew, A. (2000). A screening instrument for autism at 18 months of age: A 6-year follow-up study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 39(6), 694–702. <https://doi.org/10.1097/00004583-200006000-00007>.
- Baron-Cohen, S., Wheelwright, S., Cox, A., Baird, G., Charman, T., Swettenham, J., ... Doehring, P. (2000). Early identification of autism by the Checklist for Autism in Toddlers (CHAT). *Journal of the Royal Society of Medicine*, 93(10), 521.
- Canal-Bedia, R., Garcia-Primo, P., Victoria Martin-Cilleros, M., Santos-Borbujo, J., Guisuraga-Fernandez, Z., Herraiz-Garcia, L., ... Posada-de la Paz, M. (2011). Modified checklist for autism in toddlers: Cross-cultural adaptation and validation in Spain. *Journal of Autism and Developmental Disorders*, 41(10), 1342–1351. <https://doi.org/10.1007/s10803-010-1163-z>.
- Christensen, D. L., Bilder, D. A., Zahorodny, W., Pettygrove, S., Durkin, M. S., Fitzgerald, R. T., ... Yeargin-Allsopp, M. (2016). Prevalence and characteristics of autism Spectrum disorder among 4-year-old children in the autism and developmental disabilities monitoring network. *Journal of Developmental and Behavioral Pediatrics: JDBP*, 37(1), 1–8. <https://doi.org/10.1097/DBP.0000000000000235>.
- Coonrod, E., & Stone, W. (2004). Early concerns of parents of children with autistic and nonautistic disorders. *Infants & Young Children*, 17(3), 258–268.
- Filipek, P. A., Accardo, P. J., Ashwal, S., Baranek, G. T., Cook, E. H., Dawson, G., ... Kallen, R. J. (2000). Practice parameter: Screening and diagnosis of autism report of the quality standards Subcommittee of the American Academy of neurology and the child neurology society. *Neurology*, 55(4), 468–479.
- Garcia Primo, P., Santos Borbujo, J., Martin Cilleros, M. V., Martinez Velarte, M., Lleras Munoz, S., Posada de la Paz, M., & Canal Bedia, R. (2014). Pervasive developmental disorders screening program in the health areas of Salamanca and Zamora in Spain. *Anales De Pediatria*, 80(5), 285–292. <https://doi.org/10.1016/j.anpedi.2013.06.030>.
- Ibañez, L. V., Stone, W. L., & Coonrod, E. E. (2014). Screening for autism in young children. In F. R. Volkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders: Assessment, interventions, and policy* (Vol. 2, 4th ed., pp. 585–608). Hoboken: Wiley.
- Johnson, C. P., Myers, S. M., & American Academy of Pediatrics Council on Children With Disabilities. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120(5), 1183–1215. <https://doi.org/10.1542/peds.2007-2361>.
- Kleinman, J. M., Robins, D. L., Ventola, P. E., Pandey, J., Boorstein, H. C., Esser, E. L., ... Fein, D. (2008). The modified checklist for autism in toddlers: A follow-up study investigating the early detection of autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(5), 827–839. <https://doi.org/10.1007/s10803-007-0450-9>.
- Magán-Maganto, M., Bejarano-Martín, Á., Fernández-Alvarez, C., Narzisi, A., García-Primo, P., Kawa, R., ... Canal-Bedia, R. (2017). Early detection and intervention of ASD: A European overview. *Brain Sciences*, 7(12). <https://doi.org/10.3390/brainsci7120159>.
- Magán-Maganto, M., Canal-Bedia, R., Hernández-Fabián, A., Bejarano-Martín, Á., Fernández-Álvarez, C. J., Martínez-Velarte, M., ... Posada de la Paz, M. (2018). Spanish cultural validation of the modified checklist for autism in toddlers, revised. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-018-3777-5>.
- Mandell, D. S., Listerud, J., Levy, S. E., & Pinto-Martin, J. A. (2002). Race differences in the age at diagnosis among Medicaid-eligible children with autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 41(12), 1447–1453. <https://doi.org/10.1097/00004583-200212000-00016>.
- Mandell, D. S., Novak, M. M., & Zubritsky, C. D. (2005). Factors associated with age of diagnosis among children with autism Spectrum disorders. *Pediatrics*, 116(6), 1480–1486. <https://doi.org/10.1542/peds.2005-0185>.
- Robins, D. L., Fein, D., Barton, M. L., & Green, J. A. (2001). The modified checklist for autism in toddlers: An initial study investigating the early detection of autism and pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 31(2), 131–144.
- Robins, D. L., Casagrande, K., Barton, M., Chen, C.-M. A., Dumont-Mathieu, T., & Fein, D. (2014). Validation of the modified checklist for autism in toddlers, revised with follow-up (M-CHAT-R/F). *Pediatrics*, 133(1), 37–45. <https://doi.org/10.1542/peds.2013-1813>.
- Stenberg, N., Bresnahan, M., Gunnes, N., Hirtz, D., Hornig, M., Lie, K. K., ... Stoltenberg, C. (2014). Identifying children with autism spectrum disorder at 18 months in a general population sample. *Paediatric and Perinatal Epidemiology*, 28(3), 255–262. <https://doi.org/10.1111/ppe.12114>.
- Sunita, & Bilszta, J. L. (2013). Early identification of autism: A comparison of the checklist for autism in

toddlers and the modified checklist for autism in toddlers. *Journal of Paediatrics and Child Health*, 49(6), 438–444. <https://doi.org/10.1111/j.1440-1754.2012.02558.x>.

Toh, T.-H., Tan, V. W.-Y., Lau, P. S.-T., & Kiyu, A. (2018). Accuracy of modified checklist for autism in toddlers (M-CHAT) in detecting autism and other developmental disorders in community clinics. *Journal of Autism and Developmental Disorders*, 48(1), 28–35. <https://doi.org/10.1007/s10803-017-3287-x>.

Modified Testing

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Synonyms

[Accommodations](#); [Accommodations in testing](#);
[Modifications](#)

Definition

Modified testing refers to a number of accommodations and modifications that can be made to an instructional assessment to meet a student's individual needs. Common accommodations and modifications include:

- Use of computer or scribe for written response
- Use of augmentative communication device for oral response
- Extended testing time
- Individual or small group administration
- Use of a reader
- Administration in quiet room or space

When testing modifications are determined to be necessary by the Planning and Placement Team, the modifications and accommodations should be identified specifically on the Individual Education Plan (IEP).

See Also

- ▶ [Assistive Devices](#)
- ▶ [Test Bias](#)

References and Reading

- IDEA Partnerships. Retrieved 11 July 2011 from www.fape.org
- Luke, S. D., & Schwartz, A. (2007). Assessment and accommodations. *Evidence for Education*, 2, 1.
- Nation Dissemination Center for Children with Disabilities. Retrieved 11 July 2011 from www.nichcy.org
- Turnbull, A., Wehmeyer, M. L., & Turnbull, R. (2009). *Exceptional lives: Special education in today's schools* (pp. 41–49). Upper Saddle River: Prentice-Hall.
- U.S. Department of Education, Office of Special Education Programs. Retrieved 11 July 2011 from www.ed.gov/osep
- Vaughn, S., Bos, C., & Schumm, J. S. (2010). *Teaching exceptional, diverse, and at-risk students* (p. 455). Upper Saddle River: Prentice-Hall.

Modifier Genes and Autism Susceptibility

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Synonyms

[Epistasis](#); [Modifier loci](#)

Structure

Autism spectrum disorders (ASD) are considered to be among the most heritable of neuropsychiatric disorders, yet the genetic architecture of ASD is complex. Unlike monogenic disorders, which may be characterized by simple Mendelian patterns of autosomal dominant or recessive inheritance, the majority of the cases of idiopathic

autism do not follow such a predictable pattern. For this reason, elucidating the genetic mechanisms of ASD presents a considerable challenge. Our current understanding of the genetic architecture of ASD is that it likely involves the complex interplay of both variants that are rare and common in the general population (discussed in more detail below), together with environmental and epigenetic factors, which are not well understood (Gupta and State 2007; Hoffman and State 2010).

In ASD, modifier genes likely contribute to the complex genetic architecture and to susceptibility. A modifier gene is a gene that influences, or *modifies*, the expression of a second gene. More specifically, a modifier gene does not simply mask the expression of another gene but rather alters the way it is expressed without necessarily preventing its expression. For example, in a monogenic disorder, a modifier gene can interact with the known causative gene so as to influence the severity of the disorder. Therefore, modifier genes contribute to variability in the expression of a particular phenotype such that their interaction with the causative gene can exacerbate or diminish the severity of a clinical presentation (Jorde et al. 2010).

For example, in autism, there are many cases of pedigrees in which two siblings carry a genetic variant that is a known risk factor, yet only one child is affected, or one child has a more severe presentation than the sibling. Such findings, which are inconsistent with predictions based on Mendelian patterns of inheritance, are pervasive in the ASD genetics literature. The term *epistasis* describes the phenomenon of the interaction of modifier and disease-causing genes. Epistasis is likely to be a key factor in disorders characterized by complex genetics, which include not only neuropsychiatric disorders, such as autism, but hypertension, diabetes, and asthma as well. The presence of multiple susceptibility loci has the effect of diminishing the power to identify a causative gene (Cordell 2002).

Function

Our current conceptualization of the genetic architecture of ASD is particularly germane for

understanding the potential role of modifier genes in ASD. For example, it is important to observe that the earliest studies of the genetics of ASD and other psychiatric disorders were based primarily on the common disease-common variant hypothesis, which posits that common alleles, which occur at a frequency of $>5\%$ in the general population, account for the majority of the genetic risk of these disorders (El-Fishawy and State 2010). However, after decades of research, common variation alone was found to account for only a small percentage of the predicted heritability of ASD.

By contrast, the rare variant-common disease hypothesis has been gaining in momentum. Evidence for this hypothesis stems in part from findings of an increased risk of *de novo* variation in ASD in simplex families, as well as the lack of consistent findings from three large, unbiased genome-wide association studies (Anney et al. 2010; Wang et al. 2009; Weiss et al. 2009). In addition, rare variant approaches to candidate gene identification have been clearly pivotal in identifying genes of major effect and highlighting relevant molecular pathways in ASD (Hoffman and State 2010).

Regardless of this conceptual shift, it is likely that both common and rare variant alleles contribute to susceptibility in ASD. Because common variants are associated with relatively modest effect sizes, it is likely that common variants may function as modifier genes in ASD. Rare alleles, such as those that are found to be causative in Mendelian syndromes, have large effect sizes. Therefore, with respect to epistasis, one could imagine that a rare allele could be causative, and its expression could be influenced by common alleles at different loci. However, there is emerging evidence that even some rare variants may carry modest effect sizes and, therefore, may also perform a similar role as a modifier to another rare, causative gene in ASD susceptibility (State 2010; State and Levitt 2011).

Moreover, pleiotropy has emerged as a central theme in the genetic architecture of ASD such that the same genetic mutations can manifest in vastly different clinical phenotypes in different individuals. One explanation for this may be

locus heterogeneity, or variation at multiple genetic loci, which together influence the expression of a known risk factor, and have the potential to result in a range of clinical presentations (State and Levitt 2011). Moreover, it is possible that a combination of rare variants with modest effect sizes and/or common variants at different loci serves as modifying genes in ASD. Therefore, modifier genes likely play a key role in autism and contribute to the varying relationship between genetic mutation and clinical manifestation.

Pathophysiology

One of the challenges of identifying modifier genes and gene interactions that contribute to ASD susceptibility is the need for appreciable power in genome-wide studies intended to identify alleles associated with small effect sizes. An alternate approach taken in some studies is to identify *endophenotypes*, or heritable phenotypes with a presumed genetic etiology. These studies utilize quantitative trait loci (QTLs), which are definable traits that are associated with a particular disorder. The rationale for using QTLs in genetic studies is that it can be used to identify the genetic risk for a particular trait, as opposed to relying on all of the diagnostic criteria for a disorder. Clinical diagnostic categories may be associated with multiple alleles, which complicates the assessment of heritable genetic risk (Abrahams and Geschwind 2008).

Further, the presence of subclinical symptoms in family members of an affected individual argues for the relevance of QTL-based approaches (Abrahams and Geschwind 2008) and suggests that there may be common variants with modest effect sizes that influence phenotype in family members as well. For example, studies have shown that siblings and parents of boys with ASD have quantifiable differences in social responses compared to the general population, suggesting that they share some but not all of the risk alleles that contribute to ASD (Constantino et al. 2006; Virkud et al. 2009).

Multiple QTLs may be defined for ASD, given the range of behaviors associated with this group of disorders. One QTL which was utilized in a study to identify common variants associated with ASD was “age at first word,” in order to identify impairments in language development (Alarcon et al. 2008). The rationale for the use of this QTL in part is that many unaffected siblings of children diagnosed with ASD experience speech and language delay. This study identified a QTL on 7q34-7q36, which falls in *contactin-associated protein-like 2* (Alarcon et al. 2008), a gene that has been strongly implicated in ASD by multiple rare variant studies as well. Therefore, QTLs represent a potentially informative approach to identify susceptibility genes with smaller effect sizes in highly heterogeneous disorders with complex genetic architecture. Moreover, our current conceptualization of the genetic architecture of ASD is that it involves the complex interplay of rare variation, both at the level of the sequence of DNA and the structure of chromosomes, which may function in a causative or modifying manner, depending on the effect size of a particular variant, and common alleles, which have a modifying role, together with environmental factors. At this time, larger genome-wide association studies are needed to obtain the power to identify modifier genes with relatively modest effect sizes.

See Also

- [Common Disease-Rare Variant Hypothesis](#)
- [Pleiotropy](#)

References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, 9(5), 341–355.
- Alarcon, M., Abrahams, B. S., Stone, J. L., Duvall, J. A., Perederiy, J. V., Bomar, J. M., et al. (2008). Linkage, association, and gene-expression analyses identify

- CNTNAP2 as an autism-susceptibility gene. *American Journal of Human Genetics*, 82(1), 150–159.
- Anney, R., Klei, L., Pinto, D., Regan, R., Conroy, J., Magalhaes, T. R., et al. (2010). A genome-wide scan for common alleles affecting risk for autism. *Human Molecular Genetics*, 19(20), 4072–4082.
- Constantino, J. N., Lajonchere, C., Lutz, M., Gray, T., Abbacchi, A., McKenna, K., et al. (2006). Autistic social impairment in the siblings of children with pervasive developmental disorders. *The American Journal of Psychiatry*, 163(2), 294–296.
- Cordell, H. J. (2002). Epistasis: What it means, what it doesn't mean, and statistical methods to detect it in humans. *Human Molecular Genetics*, 11(20), 2463–2468.
- El-Fishawy, P., & State, M. W. (2010). The genetics of autism: Key issues, recent findings, and clinical implications. *The Psychiatric Clinics of North America*, 33(1), 83–105.
- Gupta, A. R., & State, M. W. (2007). Recent advances in the genetics of autism. *Biological Psychiatry*, 61(4), 429–437.
- Hoffman, E. J., & State, M. W. (2010). Progress in cytogenetics: Implications for child psychopathology. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(8), 736–751, quiz 856–737.
- Jorde, L. B., Carey, J. C., & Bamshad, M. J. (2010). *Jorde: Medical genetics* (4th ed.). Philadelphia: Mosby.
- State, M. W. (2010). The genetics of child psychiatric disorders: Focus on autism and Tourette syndrome. *Neuron*, 68(2), 254–269.
- State, M. W., & Levitt, P. (2011). The conundrums of understanding genetic risks for autism spectrum disorders. *Nature Neuroscience*, 14(12), 1499–1506.
- Virkud, Y. V., Todd, R. D., Abbacchi, A. M., Zhang, Y., & Constantino, J. N. (2009). Familial aggregation of quantitative autistic traits in multiplex versus simplex autism. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 150B(3), 328–334.
- Wang, K., Zhang, H., Ma, D., Bucan, M., Glessner, J. T., Abrahams, B. S., et al. (2009). Common genetic variants on 5p14.1 associate with autism spectrum disorders. *Nature*, 459(7246), 528–533.
- Weiss, L. A., Arking, D. E., Daly, M. J., & Chakravarti, A. (2009). A genome-wide linkage and association scan reveals novel loci for autism. *Nature*, 461(7265), 802–808.

Modifier Loci

► Modifier Genes and Autism Susceptibility

Moebius Syndrome

► Möbius Syndrome

Moldova and Autism

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Historical Background

Autism in Moldova has a very short history. The story of autism spectrum disorders in Moldova started in 2004, when the first child was diagnosed as childhood autism. However, this is synonymous to autism spectrum disorders (ASD) not existing in Moldova. For many years, especially during the soviet period, children with autism were diagnosed as mentally retarded or some other diagnosis. Most of them had missed the optimal treatment window (early intervention) because of inadequate institutions, insufficient finances, and parental ignorance. Many of the children with autism have been placed into institutions. Because of the poor economic situations and inadequateness of services of social assistance, families were no longer able to care for the medical, physical, and psychological needs of their children and found institutions to be the only viable alternative. This attitudes and approach had lasted for decades in Moldova.

The lack of knowledge on ASD in professionals, who are involved in work with children with autism spectrum disorders (ASD), lack of standardized protocols for diagnosis and

assessment tools, and precarious use of international classifications and diagnostic tools are resulting in low and late detection of autism. Officially in Moldova ICD-10 (1992) diagnostic manual is used, but these days, children with autism spectrum disorders in formal statistics data are getting the diagnosis “mental and behavioral disorders of nonpsychotic character.” So there are no reliable data about the statistics on how many children with ASD are diagnosed in Moldova.

The last years, due the immense influence of nongovernmental organization, supported by Western countries, and the activism of parents associations of children with autism in Moldova, have increased awareness regarding autism in professionals and parents. The organization, “SOS Autism” – one of the most powerful parents associations in Moldova founded in 2008 – is a leader in advocacy of children with ASDs. This organization is lobbying for the development of professional intervention services, inclusive education for children with autism, counseling, informational support, and social assistance for parents raising this children. Today in Moldova, all the burdens related with ASD fall on the shoulders of the parents. In capital city of Moldova, there are several private organizations that offer very expensive ABA – therapy for children with autism. Activities of these companies are not controlled and regulated by the state.

The government published its first autism strategy in 2009. In different regions in Moldova, there have been opened community centers in mental health. But the efficiency of these centers in order to help people with Autism is very low because of lack of professionals working in these centers.

Legal Issues, Mandates for Service

Law no. 60 of 30.03.2012 on the social inclusion of people with disabilities, Chapter IV seeks to provide Education, training, and development of persons with disabilities to participate in all areas of life without discrimination, at the same level with other members of society.

But in reality inclusion of a child with autism in the kindergarten or school depends on parents' education, insistency, and patience. Often parents with higher education are promoters of right approaches toward the children with autism and have a powerful influence in implementations on laws.

Overview of Current Treatments and Centers

The clinic for early intervention in Chisinau is regarded as one of the best clinics working with intervention on kids with ASDs.

SOS Autism – is another clinic in Moldova

Hippo – behavioral intervention center – private center offering ABA sessions

There are somewhere around ten clinics in total in Moldova.

Overview of Research Directions

There is currently no research going on in Moldova.

Overview of Training

Training is in Moldova left to the parents after they have received help in the clinics.

Very few of these kids have personal assistants in school and are left to themselves often.

Social Policy and Current Controversies

Socially, the society is looking down on those with an autism spectrum disorder. They are looked down on, bullied, and regarded as less valuable than children with typical development. Parents often complain that friends push back when they find out that families have a son or a daughter with ASDs. So it is huge social stigma connected to having an ASD diagnosis.

Molindone

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Synonyms

[Moban](#)

Definition

Molindone is a traditional antipsychotic medication developed for the treatment of schizophrenia. It is more potent than chlorpromazine, but less potent than antipsychotic medications such as haloperidol (► [Antipsychotics: Drugs](#), ► [Haloperidol](#)). Molindone has somewhat unique feature in that it is not associated with weight gain, and, indeed, patients treated with molindone may even lose weight. Molindone has been studied in children but has not been studied in children or adults with autism.

See Also

► [Haloperidol](#)

References and Reading

Martin, A., Scahil, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.

Monotone

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Synonyms

[Flat prosody](#); [Monotonic voice](#)

Definition

Monotone speech refers to a flat prosodic pattern in which the voice varies little in pitch or rhythm. This style of speaking can come across as dry and lacking in affective qualities. Many individuals with autism spectrum disorder use this style of speech, which can make them stand out as “different” in conversational situations and can be an obstacle to forming social relationships.

See Also

► [Intonation](#)
► [Prosody](#)
► [Singsong](#)

References and Reading

Diehl, J. J., & Berkovits, L. (2010). Is prosody a diagnostic and cognitive bellwether of autism spectrum disorders? In A. Harrison (Ed.), *Speech disorders: Causes, treatments, and social effects* (pp. 159–176). New York: Nova Science.

McCann, J., & Peppé, S. (2003). Prosody in Autism spectrum disorders: A critical review. *International Journal of Language & Communication Disorders*, 38(4), 325–350.

Paul, R., Augustyn, A., Klin, A., & Volkmar, F. R. (2005). Perception and production of prosody by speakers with

Autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 35(2), 205–220.

Shriberg, L. D., Paul, R., McSweeney, J. L., Klin, A., Cohen, D. J., & Volkmar, F. R. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning Autism and Asperger syndrome. *Journal of Speech, Language, and Hearing Research*, 44(5), 1097–1115.

Monotonic Voice

► Monotone

Monotropism: An Interest-Based Account of Autism

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Synonyms

[Attention tunnelling](#); [Ecological model](#); [Resource distribution](#)

Definition

The idea of monotropism makes most sense in terms of a basic model which sees minds as made up of active *interests* shaped by their histories of paying attention in a world (and in an imagined world).

This interest model of mind is ecological, embodied, and exploratory. Instead of applying emotionally charged values to categorize humans, it offers a more objective way of thinking about autistic and other human variations: it does not pathologize them. This is not just semantics, current diagnostic practice stamps “Rejected!” on the core nature of a large part of the human race, with profound repercussions, as history relates if we attend to it.

The model was developed initially as an approach to relevance or salience, so it is about

what matters to a person; it is about what you pay attention to and why. Interests in action retain – are formally changed by – information acquired as they proceed. Homeostasis and reliability of action are pervasive values which are in tension with each other as bodies move through a world viewed through the lens of prior encounters. Thus interests are adaptively informed by their engagement with attractions, emotions, and possibilities.

“Interests” range from fleeting moments to lasting passions; from narrow fixations and self-seeking plots to duties and universal concerns; and *communities of interest* can extend from family and friends, teams, gangs, brands, and nations with long histories to casual folk at bus stops; and from mutual aid to the self-seeking vested interests of corporations and their actors, and the bureaucratic interests of the state.

Interests can be aroused, expressed in the world or the imagination, and informed. Their basic resource is emotional – an energetic force; its quantity as well as its direction and “flavor” tend to incessant change in any individual or set of individuals, against relatively stable patterns of bound energy within a vast network of n-dimensions and gradients, itself a value system.

The will to proceed and take some degree of risk depends on assuming reliable grounds for your actions: all that stuff you do not need to worry about, because it is already settled – sometimes referred to as your “priors” – informs all active interests. Active interests constantly probe at a person’s (or any living being’s) world-engaged edge. They involve having expectations: rough and ready and often disappointed. They can be aroused, informed, expressed/enacted, connected or disconnected, expanded or shrunk, and boosted or undermined. Emotional valences and gradients within an n-dimensional matrix change: possibilities get assessed; certainty is sought; boundaries are created; other goods are sought. Flows and waves and turbulence occur.

The monotropism idea about autism proposes that a leading interest will tend to get a larger share of the scarce resource, which is diverted at boundaries creating channelled flows. The corollary of this larger share can be rich, interest based, inter-connections in some areas, with real gaps in other

areas which may take others by surprise. Flows may also be held back by a significantly higher proportion of resource being assigned to “guard” the boundary relative to a non-monotropic pattern. When a boundary is at its least permeable, patterns which are hard to dislodge will emerge. Cases both of extreme avoidance and extreme attraction are likely and can be problematic as well as rewarding, even for the individual experiencing them.

These patterns of resource distribution can mean virtually undistracted flow versus virtually uncrossable boundaries. The flow states can be immensely satisfying and sometimes highly productive; the barriers can be like cliffs, “frightful, sheer” (Hopkins 1880). It is particularly important to avoid catastrophic events in this “landscape” as far as possible, as they are atypically hard to recover from.

Catastrophes involving sudden loss of capacity (see Thom 1976) may be precipitated by input of any sort: sensory, social, and imaginary; resources may be so scattered and unavailable, and the emotional temper so unsettled that much recovery time is necessary. A high intensity outburst is hard to get over and an inability to send out soothing vibes may get worse all round, if there is no let-up in the pressure. Monotropic people are less likely to have the resources available to repair the ruptured community of interest, therefore it is only fair to ensure they have a chance to get over it. Neurotypicals have to lead on this as they do have the resources, and must avoid projecting assumptions especially negative ones, or may risk causing lasting harm. This can be the downside of so-called “Theory of Mind”: high levels of very unreliable and often pejorative projections of meaning by “typical” people onto others (borderline “gaslighting”) are especially problematic if the others have no means to talk back. In every way possible, the more verbally capable people should gently, with minimum pressure, try to find out more from the inarticulate person and not assume they know best.

The embedding of the all or nothing monotropic disposition, into a very busy environment steeped in uncertainty and egotistical calculation, causes the problems that attract a diagnosis. Like

everybody else, autistic people prefer to operate in a reliable and generally predictable world (see, e.g., Friston 2016). Autistic people’s ability to recover from the impact of unanticipated events which seem harmless to others can be so strongly diminished that a return to capable processing is long delayed. Some of this can be worsened by social pressures from neurotypical people for autistic people to conform. Pushing someone is always the worst, least mutual way to relate to them; doing that while emitting the hostile emotional vibes of the self-righteous can cause the worst possible experience for the socially wary.

See Also

- [Double Empathy](#)
- [Ecological Model of Autism](#)

References and Reading

- Asma, S. T., & Gabriel, R. (2019). *The emotional mind: The affective roots of culture and cognition*. Cambridge: Harvard University Press.
- Friston, K. (2016). Commentary the Bayesian savant. *Biological Psychiatry*, 80, 87–89. www.sobp.org/journal
- Hopkins, G. M. (1880). Poem: No worst, there is none, pitched past pitch of grief. In G. M. Hopkins (Ed.), *Poems and Prose*. London: Penguin Classics, 1985.
- Lawson, W. (2001). *Understanding and working with the spectrum of Autism: An insider's view*. London: JKP.
- Lawson, W. (2010). *The passionate mind*. London: JKP.
- Mike, L., & Dinah, M. (1998/2020). Mind as a dynamical system from Durham conference papers. Reprinted in D. Milton (Ed.), *The Neurodiversity Reader*. London: Pavilion, in press.
- Deborah, L., & Richards, W. (2009). *Managing meltdowns: Using the S.C.A.R.E.D. calming technique with children and adults with Autism*. Durham/Brighton: JKP & Kindle.
- Milton, D. et al. (2020). *The double empathy problem*. Encyclopedia.
- Miserandino, C. (2003). Cited in Memmott, A (2018), Autism and spoon theory. <http://annsautism.blogspot.co.uk/2018/02/autism-and-spoon-theory.html>. Accessed 28 Feb 2018.
- Murray, D. (2020). Dimensions of difference. In D. Milton (Ed.), *The neurodiversity reader*. Brighton: Pavilion.
- Murray, D. 1st edition of this encyclopedia.
- Murray, D., Lesser, M., & Lawson, W. (2005). Attention, monotropism and the diagnostic criteria for autism. *Autism*, 9, 139–156.

- Murray, F. (2019). Me and monotropism: A unified theory of autism. *The Psychologist*, 32, 44–49.
- Thom, R. (1976). *Structural stability and morphogenesis an outline of a general theory of models*. London: Benjamin.
- Wood, R. (2019). *Inclusive education for autistic children helping children and young people to learn and flourish in the classroom*. Foreword by Dr. Wenn B. Lawson, illustrated by Sonny Hallett. London: JKP.
- Woods, R. (2020). Commentary: Demand avoidance phenomena, a manifold issue? Intolerance of uncertainty and anxiety as explanatory frameworks for extreme demand avoidance in children and adolescents – a commentary on Stuart et al. (2019) *Child and Adolescent Mental Health* Volume, 2020. <https://doi.org/10.1111/camh.12368>.

Monozygotic (MZ) Twins

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Synonyms

[Identical twins](#)

Definition

Twins are two individuals who are the result of the same pregnancy. Monozygotic twins each carry the identical genetic information or deoxyribonucleic acids (DNA).

Monozygotic twins form as follows. When a sperm from the father fuses with an egg from the mother, they form a single cell called a zygote, the earliest stage of an embryo. Further cell divisions occur during embryonic development. At an early point in this development, the embryo can be split into two separate embryos, the cells of each having originated from the initial zygote. Each of these embryos goes on to develop into a separate and complete individual. Since each originated from the same initial cell, each individual is identical in his or her genetic composition. Barring very rare genetic occurrences, since monozygotic

twins share the same genetic material, their physical appearance will be identical, and they will be of the same sex.

In contrast, in the case of dizygotic twins, two separate eggs are released at one time. Each of these is then fertilized by a separate sperm and forms a distinct zygote and embryo. Since each sperm and egg carry distinct genetic material, each of the two embryos will, thus, carry distinct genetic material. On average, dizygotic twins' genetic material is only about 50% identical, as compared to 100% for monozygotic twins. Since each dizygotic twin carries distinct genetic material, his or her physical appearance will be distinct. Dizygotic twins may be of the same sex. However, they might also be of different sexes.

The occurrence of monozygotic twins is rare and more rare, in general, than the occurrence of dizygotic twins. Monozygotic twinning occurs at a rate of approximately 4 in every 1,000 live births worldwide. Monozygotic twins comprise approximately one third of all twins. Two thirds are dizygotic twins. Studies indicate that the rate of monozygotic twinning in the absence of fertility treatments is fairly uniform across different populations around the world. This is in contrast to the uneven ethnic worldwide distribution of dizygotic twinning in the absence of fertility treatments. The rate of both monozygotic and dizygotic twins has increased worldwide since the 1970s. The majority of the increase has resulted from the increase in dizygotic twins born as a result of fertility treatments.

Twin pregnancies of either type increase pregnancy risk and especially the risk of preterm delivery and low birth weight. Approximately 51% of twins are born preterm compared to 9.4% in singletons.

See Also

- [DNA](#)
- [Genetics](#)
- [Twin Studies in Autism](#)

References and Reading

- Alexander, G. R., Kogan, M., Martin, J., & Papiernik, E. (1998). What are the fetal growth patterns of singletons, twins, and triplets in the United States? *Clinical Obstetrics and Gynecology*, 41(1), 115.
- Bortolus, R., Parazzini, F., Chatenoud, L., Benzi, G., Bianchi, M. M., & Marini, A. (1999). The epidemiology of multiple births. *Human Reproduction Update*, 5(2), 179.
- Gilbert, S. F. (1994). *Developmental biology*. Sunderland: Sinauer Associates.
- Hall, J. G. (2003). Twinning. *The Lancet*, 362(9385), 735–743.
- Machin, G. A. (1996). Some causes of genotypic and phenotypic discordance in monozygotic twin pairs. *American Journal of Medical Genetics*, 61(3), 216–228.
- Reeve, E. C. R., & Black, I. (2001). *Encyclopedia of genetics*. London: Routledge.
- Smits, J., & Monden, C. (2011). Twinning across the developing world. *PLoS One*, 6(9), e25239.
- Strachan, T., & Read, A. P. (2004). *Human molecular genetics*. New York: Garland Press.
- Vitthala, S., Gelbaya, T. A., Brison, D. R., Fitzgerald, C. T., & Nardo, L. G. (2009). The risk of monozygotic twins after assisted reproductive technology: A systematic review and meta-analysis. *Human Reproduction Update*, 15(1), 45.

Mood Disorder

► Mood Disorders

Mood Disorders

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Synonyms

Clinical depression; Major depression; Unipolar depression, Mood disorder

Short Description or Definition

Autism spectrum disorders (ASDs) are a heterogeneous group of neurodevelopmental disorders characterized by impairments in social interaction and communication skills, as well as stereotypic behaviors and restricted activities and interests. Also included within ASDs are conditions which were previously classified as Asperger syndrome (AS) and Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS), but now all contained within the diagnosis ASD (DSM-5 2013). Recent studies in Europe and the United States indicate a prevalence of ASD ranging from 1.4 to 212 per 10,000 individuals (CDC 2012; Elsabbagh et al. 2012). Males, compared to females, are circa four times more frequently affected (Rinehart et al. 2002; Williams et al. 2013). The severity specifiers (level 1: requiring support; level 2: requiring substantial support; level 3: requiring very substantial support) may be used to describe succinctly the current symptomatology, with the recognition that severity may vary by context and fluctuate over time (DSM-5 2013).

Recent studies show that more than 70% of all children diagnosed with ASD have at least one and 41% have at least two comorbid psychiatric disorders (Simonoff et al. 2008). In adults with ASD, comorbid psychiatric disorders are common as well. The most common coexisting psychiatric conditions in children and in adults with ASD are intellectual disability, attention-deficit/hyperactivity disorder (ADHD), anxiety disorders, and mood disorders (Geurts et al. 2010). This section will focus on the epidemiology, natural history, prognostic factors, outcomes, clinical expression, pathophysiology, evaluation, differential diagnosis, and treatment of mood disorders in patients with ASD.

Categorization

The term “mood disorder” describes a group of various psychiatric disorders characterized by mood disturbance. According to the DSM-5, this group can be divided into “depressive

disorders” and “bipolar and related disorders.” Depressive disorders include the major depressive disorder, disruptive mood dysregulation disorder, persistent depressive disorder (dysthymia), premenstrual dysphoric disorder, substance/medication-induced depressive disorder, depressive disorder due to another medical condition, other specified depressive disorder, and unspecified depressive disorder. Major depressive disorder (also called major depression, unipolar depression, or clinical depression) is the most common depressive disorder. The disruptive mood dysregulation disorder (DMDD) resulted from the DSM-5 Work Group raised concerns that many youths with severe, nonepisodic irritable mood are inappropriately diagnosed with bipolar disorder. DMDD refers to the presentation of children with persistent irritability and frequent episodes of extreme behavioral dyscontrol which must be present for at least a year. Dysthymia is a condition characterized by a chronic low mood. The unspecified depressive disorder contains any depressive disorder that does not meet criteria for a specific mood disorder. Bipolar disorder is a complex disorder in which the core feature is pathological disturbance in mood ranging from extreme elation or mania to severe depression usually accompanied by disturbances in thinking and behavior, which may include psychotic symptoms. It is an episodic, chronic illness, usually with recovery between episodes. Bipolar disorders include the bipolar I disorder, bipolar II disorder, cyclothymic disorder, substance/medication-induced bipolar and related disorder, bipolar and related disorder due to another medical condition, other specified bipolar and related disorder, and unspecified bipolar and related disorder. The diagnosis bipolar I disorder requires that a person has suffered one or more episodes of mania in their life with or without episodes of depression at other times. Patients with bipolar II disorder experience a milder form of a manic episode, known as a hypomanic episode as well as major depressive episodes. The diagnosis of cyclothymic disorder is given to adults who experience at least 2 years (children: 1 year) of both hypomanic and depressive periods without

ever fulfilling the criteria for an episode of mania, hypomania, or major depression (DSM-5 2013).

Epidemiology

Depressive symptoms are the most common psychiatric concern in patients with ASD and are more likely to occur in adolescence and adulthood. Patients with ASD show a higher risk for developing depressive symptoms compared to the general population (Sterling et al. 2007). Prevalence estimates for any type of co-occurring depressive disorder range from 0.9% to 29% in children and adolescents, which is higher than the prevalence of depression in the general population (Leyfer et al. 2006; Simonoff et al. 2008). Likewise, 77% of outpatient adults with ASD have ever had a diagnosis of depression, compared to 46% of outpatient, non-ASD adults (Joshi et al. 2013). Reported prevalence rates of mood disorders in ASD vary due to differences in study populations, duration of follow-up, and variation in methods (Stewart et al. 2006; Geurts et al. 2010). In addition, autistic symptomatology tends to mask cardinal features of depression and presentation of depressive symptoms might be atypical in patients with ASD, causing a bias in the prevalence rates (Simonoff et al. 2008; Lainhart and Folstein 1994). In study of adults (age 16–60 years) with ASD, the lifetime prevalence rate of depression was 53% and in a younger study population this prevalence ranged from 4% to 38% (Hovander et al. 2009). Leyfer et al. (2006) showed that around 10–15% of children and adolescents (mean age 9) with ASD, ever had depressive symptoms, 10% met criteria for a major depressive disorder, and about 2% met criteria for a depression not otherwise specified. In patients with the former Asperger Syndrome (AS), the prevalence of comorbid depression is reported most frequently with a prevalence of around 30% (Matson and Nebel-Schwalm 2007). Little is known about the prevalence of dysthymia in persons with ASD. Dysthymia is less common compared to depressive disorders (Simonoff et al. 2008). Skokauskas and Frodila (2015) found the

average prevalence of bipolar disorder in persons with ASD to be 7%, with variations between 2% and 31%. It often emerges during adolescence (Vannucchi et al. 2014). Clinical studies on the comorbidity of ASD and bipolar disorder in children are scarce and in particular in the form of case reports. In the following sections, we will focus on the depressive disorder and its symptoms.

Natural History, Prognostic Factors, and Outcomes

Mood disorders are episodic disorders with either a marked change in the context of type of symptoms and variability in intensity of symptoms over time (Leyfer et al. 2006; Matson and Nebel-Schwalm 2007). Due to difficulties in diagnosing a coexisting depression in these patients (see also the sections “Clinical Expression and Pathophysiology” and “Evaluation and Differential Diagnosis”), symptoms are often severe and/or prolonged at time of diagnosis (Skokauskas and Gallagher 2010). Clinical evidence suggests that the outcome of autism might be affected by the presence of coexisting psychiatric disorders in ASD (Ghaziuddin et al. 2002). Especially, depression can have a negative impact on long-term outcome. Effects on a patient and a patient’s family can be wide ranging. Patients with ASD can show a regression of behavioral skills, social withdrawal, and aggressive and oppositional behavior. These changes can interfere with a person’s community participation and his or her social relationships. Furthermore, depression in ASD is associated with poor quality-of-life, inpatient hospitalization, and medical illness (Greenlee et al. 2016; Righi et al. 2017). Depression may also put an autistic patient at risk for catatonia and suicide, especially when symptoms are severe. There are few reports on suicidal behavior among patients with ASD. Causes of underreporting may be the low rate of suicidal behavior among children and preadolescents and the underdiagnosing of ASD in the adult psychiatric setting. Comorbidity is often reported with mood disorders and psychotic disorders, so it is difficult to determine if

suicidality is specifically associated with ASD (Richa et al. 2014).

Clinical Expression and Pathophysiology

Symptoms of ASD and depression show a considerable overlap, which makes it harder to diagnose depressive disorders. Signs of ASD like abnormal speech patterns and social withdrawal might be confused with symptoms of depression such as psychomotor retardation or fatigue. Depressive patients with ASD show a difference in symptom presentation compared to patients without ASD, since expression of depressive symptoms is colored by ASD behavior. Many of these patients are not able to express their feelings, because of difficulties in expressing and communicating emotion due to poor integration between different modalities of nonverbal expressions and insufficient language skills to verbalize changes in feelings and mood (Perry et al. 2001; Stewart et al. 2006; Skokauskas and Gallagher 2010). As in depressed persons without ASD, in depressed patients with ASD one of the main features is a depressed mood. However, in patients with ASD, its presence is more often reported by a patient’s close relative than by self-report (Stewart et al. 2006). This is also the case with behavioral changes, appetite, and sleep disturbance. The majority of patients with ASD and comorbid depression show a decrease in self-care and increased social withdrawal. In youth with ASD, preoccupation with themes of death has also been noted during depressive periods (Ghaziuddin et al. 1995; Perry et al. 2001). When severity of depressive symptoms increases, self-esteem becomes more negative in individuals with ASD. Due to the limited communication skills experienced by many individuals with intellectual disability, symptoms of depression are likely to manifest behaviorally as a regression in adaptive skills and an increase in aggressive and self-injurious behaviors (Magnuson and Constantino 2011; Stewart et al. 2006; Turygin et al. 2013).

With the onset of depression in patients with ASD, both decrease and intensification of autistic symptoms have been reported. Specific

depressive symptoms for individuals with ASD, might be the loss of interest in subject of previous preoccupation and a decline in rigid behavior (Ghaziuddin et al. 2002; Stewart et al. 2006). However, these changes in behavior might be often interpreted as an improvement by their close relatives; in fact, these changes could be a sign of the presence of a depression. Presented symptoms are not always mood-related. The new onset of a depression, for example, is usually associated with a new onset or exacerbation of maladaptive behavior consisting of an increase in aggressive behavior, agitation, irritability, auto-mutilation, compulsive behavior, hypoactivity, or a decline in daily skills (Geurts et al. 2010; Ghaziuddin et al. 2002; Stewart et al. 2006). Especially, self-injury and aggression are often reported. Thus, in all individuals diagnosed with ASD, it is important to investigate whether or not behavior is related to the natural history of ASD, or has changed due to a comorbid disorder. Every ASD patient, especially those with an accompanying intellectual impairment, should be screened for depression if there is a recent history of aggressive outbursts, in the setting of irritability, appetite, and sleep disturbance. In those patients, the most frequent signs of depression are a change in level of functioning; a regression in skills such as incontinence, severe appetite, sleep, and weight disturbance; and the presence of aggression. More unusual features are catatonia and psychotic symptoms (Ghaziuddin et al. 2002).

The exact pathophysiology of depression in patients with ASD remains unclear. Depression is known to be caused by genetic and environmental factors. Children diagnosed with ASD suffering from a depression are more likely to have a family history of mood disorders (Gillberg and Billstedt 2000). Autism and depression seem to cluster in some families, suggesting that in a subgroup of patients, common genetic factors seem to be responsible. However, there is also evidence for an independent genetic origin of these factors (Hallett et al. 2009). The excess of depression in these families does not seem to be related to the stress caused by raising a child with ASD.

Life events play an important role in the onset of depressive symptoms. Psychological processes

such as emotion dysregulation and intolerance of uncertainty have an impact on mood in autistic patients (Hill et al. 2004; Cai et al. 2018). Stressful situations can be overwhelming for patients with ASD, and even to mild life events, they can respond intense. Children with ASD who develop a major depression experienced more negative life events such as family illness, parental divorce, and bereavement (Ghaziuddin et al. 1995). Data on this correlation in adults have not been described yet. Autistic patients might respond with depressive symptoms to negative events because of a genetic predisposition to depression (Ghaziuddin et al. 2002).

Social relationships in patients with ASD seem to be related to the development of depression as well. Interpersonal conflicts with family members have been strongly associated with depressive symptoms in children with autism spectrum disorders. In addition, autistic children with more friendships of poorer quality were found to have the highest levels of depression (Lopata et al. 2010).

Factors affecting the occurrence of depression might be age, level of intelligence, social functioning, and the presence of other psychiatric or other medical disorders. Most cases of depression in patients with ASD have been described in adolescents and adults (Ghaziuddin et al. 2002; Matson and Nebel-Schwalm 2007; Sterling et al. 2007). This could be due to problems of assessment of psychiatric conditions in children with ASD. However, clinical experience suggests that the rate of depression in patients with ASD rises with age (Ghaziuddin et al. 1998; Greenlee et al. 2016). An explanation for this increase with age could be the more pronounced developmental gap between these patients and their same-aged peers and the lack in adaptive skills of executive planning abilities to take necessary steps toward independent living (Sterling et al. 2007). Some researchers have hypothesized that individuals who are higher functioning (e.g., higher verbal capacities, theory of mind, introspective capacities, social strength) may be more likely to experience depression, due to greater awareness of differences and challenges with social interactions (Lerner et al. 2017; Mazurek 2014). Also, higher

functioning autistic adults may be more likely to endorse cognitive symptoms of depression, such as guilty feelings, sense of failure, and pessimism, compared to normal developing individuals (Gotham et al. 2015). It is not clear, however, whether a lack of insight in one's own level of impairment in lower functioning children confers a protection from depressive states. Though, in severe autistic patients with a lower IQ, fewer depressive symptoms were reported (Magnuson and Constantino 2011). Better adaptive skills are associated with increased rates of depression as well.

Evaluation and Differential Diagnosis

As previously described, a coexisting mood disorder in children and adults with ASD can be difficult to diagnose due to impairments in cognitive functions and an overlap in symptoms between different psychiatric conditions. For patients, these impairments make it difficult to describe experiences and mental state. Symptoms are often reported by a patient's close relative instead of by the patient himself/herself. Therefore, it is important to involve the patient's parents and teachers in the diagnostic process. Kanne et al. (2009) found that when using the Achenbach System of Empirically Based Assessment, a higher percentage of parents reported their children as having depressive symptoms than their teachers did. Using the Youth Self-Report form, Hurtig et al. (2009) found strong agreement among adolescent patients with ASD, their parents, and their teachers, indicating awareness of such difficulties.

A mood diagnosis is based on clinical expression of depressive symptoms reported by a patient, its close relatives, and clinician's observation and interpretation. Due to a lack of a "gold standard" diagnostic tool, many different diagnostic instruments have been used for assessment of a depressive disorder in patients with ASD. Traditional measures of diagnosis have, in general, not been tested for validity and reliability in ASD populations. Diagnostic criteria used to identify psychiatric disorders in developmentally disabled

patients may identify only the "tip of the iceberg" (Leyfer et al. 2006). In *children and adolescents*, the Diagnostic Interview for Children and Adolescents, DICA IV, the Child Behavior Checklist CBCL, the Leiter-R-Questionnaires, the Kiddie Schedule for Affective Disorders and Schizophrenia K-SADS-PL, and the Children Depression Inventory CDI (based on the Hamilton Rating Scale for Depression) have been often used. One study used a modification of the Kiddie Schedule for Affective Disorders and Schizophrenia in children and adolescents with autism for the assessment of comorbid psychiatric disorders. They developed additional screening questions and new coding options and found and reported a sensitivity of 100% and a specificity of 93.7% for major depression in their sample (Leyfer et al. 2006). Reiss and Valenti-Hein (1990) developed the Reiss scales for the screening of comorbid psychiatric disorders in children and adolescents with intellectual disability (ID). Results from their study population suggest that these instruments are particularly well suited for screening and for helping in the analysis of the relationships between certain behavior problems and psychopathology in patients with ID (Reiss and Valenti-Hein 1990). In general, observations of behavior and activities are important in the assessment of depression because of their limited verbal abilities and their tendency to express thoughts and feelings through play.

In *adults*, the Beck Depression Inventory (BDI) and the Hamilton Rating Scale for Depression (HRSD) are two of the most widely used scales to assess the severity of depressive symptoms (Stewart et al. 2006). The Beck Depression Inventory, a multiple choice self-report questionnaire containing 21 items relating to symptoms of depression, was found to be an adequate screening instrument for depression in adolescent and adult males with AS (Cederlund et al. 2010). However, even if questions of the BDI and the HRSD have been adapted, some questions may remain difficult for patients with ASD and their relatives. For example, these diagnostic instruments ask patients to rate their mood and if they have feelings of guilt or worthlessness. Therefore, some patients with ASD may not express these feelings

during a depressive episode because these feelings are not in their repertoire (Leyfer et al. 2006; Stewart et al. 2006).

During the lifespan, different problems related to ASD can appear resulting in differences in a person's behavior. To determine whether a person's behavior is due to an underlying autism spectrum disorder or due to a comorbid psychiatric condition, a clinician can assess a patient's developmental history, focusing on the consistency of symptoms over time and pervasivity in different situations. Thus, it will be easier to determine whether a comorbid disorder was already present and if symptoms of comorbidity or ASD are changing (Geurts et al. 2010). As mentioned before, if a change in symptoms of an autistic patient occurs, it is important to screen him or her for comorbid disorders.

The differential diagnosis of mood disorders in patients with ASD is comprehensive because of an atypical presentation of symptoms and includes depressive disorders, bipolar disorders, anxiety disorders, and disruptive disorders. Determined by the presence of episodic manic or hypomanic symptoms, a patient can be diagnosed with a comorbid bipolar I disorder and bipolar II disorder, respectively. When a change of or increase in symptoms is reported in an autistic patient, one should be aware that these symptoms can still exist due to an autism spectrum disorder. An increase in obsessive compulsive behavior can point towards the onset of a depression but be a sign of an obsessive-compulsive disorder as well. Anxiety is often present in patients with ASD, but coexisting anxiety and depressive disorders should be excluded. In school-aged children, a depressive disorder is characterized by agitation and externalizing behavior problems. Therefore, differential diagnostic disruptive disorders have to be considered, such as ADHD and ODD. Psychotic disorders are a part of the differential diagnosis of depression in patients with ASD as well.

Treatment

There is a lack of studies that directly assess the effects of residential programs, treatment

programs, and education programs on depressive symptoms in patients with ASD. However, there are interventions that can reduce mood and related behavioral problems in patients with ASD. Some high-functioning depressive patients with good verbal skills may benefit from psychodynamic psychotherapy. In these cases, a highly structured and directive approach is required. Structured psychotherapy combined with appropriate behavioral and educational interventions are recommended by some studies. In patients with good cognitive capacities and older patients, cognitive behavioral therapy might help them to cope with anger and other depressive symptoms. This therapy is seldom successful without integration and repeating of learned skills in ongoing treatment. Studies in adults with ASD have shown mindfulness-based therapy and social and vocational skills training to be effective for symptoms of depression, but neither intervention has been studied as a treatment for depressive symptoms in children or adolescents with ASD (Spek et al. 2013; Hillier et al. 2011).

The main treatment for depression in patients diagnosed with ASD is pharmacological including selective serotonin reuptake inhibitors (SSRIs), mood stabilizers, antipsychotics, and hypnotics. SSRIs seem to be the most effective drugs in depressive adult patients with ASD (Stewart et al. 2006). These agents are being increasingly used in ASD to control depression and aggression (Ghaziuddin et al. 2002). Serotonin is linked to the mediation of psychological processes such as mood, social interaction, sleep, obsessive-compulsive behaviors, and aggression. Increased rates of platelet serotonin transport and levels of whole blood and platelet serotonin (5-hydroxytryptamine, 5-HT) have been reported in people diagnosed with ASD, and therefore, SSRIs are associated with an improvement of ASD symptoms (Williams et al. 2013). Moreover, treatment in autistic patients with depressive symptoms is in some study populations including children, adolescents, and adults associated with a decrease in low mood, sleep disturbances, and aggressive and self-injurious behavior and an increased capacity for self-care as well (Hollander et al. 2003; Perry et al. 2001; Stewart

et al. 2006). In nondepressed ASD children, low doses of SSRIs (venlafaxine) improved repetitive behaviors, hyperactivity, and communication and language functions (Hollander et al. 2000). However, a Cochrane review found no evidence for the effectiveness of SSRIs as a treatment for children with ASD (Williams et al. 2013). This review focused on the treatment of ASD using SSRIs and did not specifically focus on a coexisting depression.

The Hamilton Rating Scale for Depression (HRSD) is developed to evaluate or report depressive symptoms in nonautistic patients. Since depressive symptoms in patients with ASD show a considerable overlap with these ASD symptoms, but other important signs like increased aggressive behavior are missed, this HRSD is not very sensitive and easy to use in an autistic population. A study by Buchsbaum et al. (2001) reported no significant improvement of depressive symptoms seen in autistic adults treated with SSRIs in depression. However, using the HRSD, some small positive effects of fluoxetine and fluvoxamine on aggressive behavior and anxiety, respectively, in this study population of adults with ASD have been described.

At this time, SSRIs cannot be recommended as a treatment for children with ASD in general. SSRIs are poorly tolerated, and also lack evidence in reducing restricted repetitive behaviors (Goel et al. 2018). Decisions about the use of these agents in depressed patients with ASD should be made on a case by case basis (Williams et al. 2013). Reported side effects of different SSRIs are headaches, sedation, aggressiveness, agitation, and lip dyskinesia by using citalopram. Fluoxetine can cause weight gain and increase agitation, and venlafaxine can cause aggression, nausea, and behavioral activation. Mood stabilizers such as valproate and lithium might improve symptoms related to affective instability and aggression in ASD patients; however, more research is needed on its efficacy (Canitano 2015). The atypical antipsychotic agent risperidone and aripiprazole can improve disruptive behavior in patients with ASD. Tremor, weight gain, and increased appetite are reported adverse effects (Fung et al. 2016;

Hollander et al. 2003). A Cochrane review found that there is limited and inconsistent evidence of the effectiveness of tricyclic antidepressants (TCAs), making clear recommendations impossible at this time (Hurwitz et al. 2012).

Recently, it is hypothesized that the impairment of gut microbiota plays a role in the development of ASD and mood disorders, but the mechanism through which it does is not fully clear. The application of therapeutic modulators of gut microbiota to autism and mood disorders has been experienced only in experimental settings to date, with few but promising results. Well-designed research studies with more participants are needed to provide more evidence that supports the effectiveness of these treatments (Mangiola et al. 2016; Li et al. 2017).

See Also

- [Affective Disorders \(Includes Mood and Anxiety Disorders\)](#)
- [Antidepressant Medications](#)
- [Attention Deficit/Hyperactivity Disorder](#)
- [Broader Autism Phenotype](#)
- [Comorbidity](#)
- [Depressive Disorder](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- Buchsbaum, M., Hollander, E., Haznedar, M., Tong, C., Spiegel Cohen, J., & Wei, T. (2001). Effects of fluoxetine on regional cerebral metabolism in autistic spectrum disorders: A pilot study. *International Journal of Neuropsychopharmacology*, 4, 119–125.
- Cai, R. Y., Richdale, A. L., Dissanayake, C., & Uljarević, M. (2018). Brief report: Inter-relationship between emotion regulation, intolerance of uncertainty, anxiety, and depression in youth with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48, 316–325.
- Canitano, R. (2015). Mood stabilizers in children and adolescents with autism Spectrum disorders. *Clinical Neuropharmacology*, 38(5), 177–182.
- Cederlund, M., Hagber, B., & Gillberg, C. (2010). Asperger syndrome in adolescent and young adult males. Interview, self- and parent assessment of social,

- emotional and cognitive problems. *Research in Developmental Disabilities*, 31, 287–298.
- Centers for Disease Control and Prevention. (2012). Prevalence of autism spectrum disorders – Autism and developmental disabilities monitoring network, 14 sites, United States. *Morbidity and Mortality Weekly Report*, 61(3), 1–19.
- Elsabbagh, M., Divan, G., Koh, Y. J., Kim, Y. S., Kauchali, S., Marcin, C., et al. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research*, 5(3), 160–179.
- Fung, L. K., Mahajan, R., Nozzolillo, A., et al. (2016). Pharmacologic treatment of severe irritability and problem behaviors in autism: A systematic review and meta-analysis. *Pediatrics*, 137, S124–S135.
- Geurts, H. M., Deprey, L., & Ozonoff, S. (2010). De diagnostiek van comorbiditeit bij patiënten met een autismespectrumstoornis. *Tijdschrift voor Psychiatrie*, 52(11), 753–761.
- Ghaziuddin, M., Alessi, N., & Greden, J. (1995). Life events and depression in children with pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 25, 495–502.
- Ghaziuddin, M., Weidmer-Mikhail, E., & Ghaziuddin, N. (1998). Comorbidity of Asperger syndrome: A preliminary report. *Journal of Intellectual Disability Research*, 4, 279–283.
- Ghaziuddin, M., Ghaziuddin, N., & Greden, J. (2002). Depression in persons with autism: Implications for research and clinical care. *Journal of Autism and Developmental Disorders*, 32, 299–306.
- Gillberg, C., & Billstedt, E. (2000). Autism and Asperger syndrome: Coexistence with other clinical disorders. *Acta Psychiatrica Scandinavica*, 102, 321–330.
- Goel, R., Hong, J. S., Findling, R. L., & Ji, N. Y. (2018). An update on pharmacotherapy of autism spectrum disorder in children and adolescents. *International Review of Psychiatry*, 30(1), 78–95.
- Gotham, K., Unruh, K., & Lord, C. (2015). Depression and its measurement in verbal adolescents and adults with autism spectrum disorder. *Autism*, 19, 491–504.
- Greenlee, J. L., Mosley, A. S., Shui, A. M., Veenstra-Vander Weel, J., & Gotham, K. O. (2016). Medical and behavioral correlates of depression history in children and adolescents with autism spectrum disorder. *Pediatrics*, 137(Supplement), S105–S114.
- Hallett, V., Ronald, A., & Happe, F. (2009). Investigating the association between autistic-like and internalizing traits in a community based twin sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 2009(48), 618–627.
- Hill, E., Berthoz, S., & Frith, U. (2004). Brief report: Cognitive processing of own emotions in individuals with autistic spectrum disorder and in their relatives. *Journal of Autism and Developmental Disorders*, 34, 229–235.
- Hillier, A. J., Fish, T., Siegel, J. H., & Beversdorf, D. Q. (2011). Social and vocational skills training reduces self-reported anxiety and depression among young adults on the autism spectrum. *Journal of Developmental and Physical Disabilities*, 23, 267–276.
- Hollander, E., Kaplan, A., Cartwright, C., & Reichman, D. (2000). Venlafaxine in children, adolescents, and young adults with autism spectrum disorders: An open retrospective clinical report. *Journal of Child Neurology*, 15, 132–135.
- Hollander, E., Phillips, A. T., & Yeh, C. (2003). Targeted treatments for symptom domains in child and adolescent autism. *Lancet*, 362, 32–34.
- Hovander, B., Delorme, R., Chaste, P., Nyden, A., Wentz, E., Stahlberg, S., et al. (2009). Psychiatric and psychosocial problems in adults with normal-intelligence autism spectrum disorders. *BioMed Central Psychiatry*, 9, 35.
- Hurtig, T., Kuusikko, S., Mattila, M. L., Haapsamo, H., Ebeling, H., Jussila, K., et al. (2009). Multi-informant reports of psychiatric symptoms among high-functioning adolescents with Asperger syndrome or autism. *Autism*, 13, 583–598.
- Hurwitz, R., Blackmore, R., Hazell, P., Williams, K., & Woolfenden, S. (2012). Tricyclic antidepressants for autism spectrum disorders (ASD) in children and adolescents (Review). *The Cochrane Collaboration*. Cochrane Database Syst Rev, 2012(3), Art. No.: CD008372.
- Joshi, G., Faraone, S. V., Wozniak, J., Petty, C., Fried, R., Galdo, M., et al. (2013). Psychiatric comorbidity and functioning in a clinically referred population of adults with autism spectrum disorders: A comparative study. *Journal of Autism and Developmental Disorders*, 44, 2117–2126.
- Kanne, S. M., Abbacchi, A. M., & Constantino, J. N. (2009). Multi-informant ratings of psychiatric symptom severity in children with autism spectrum disorders: The importance of environmental context. *Journal of Autism and Developmental Disorders*, 39, 856–864.
- Lainhart, J. E., & Folstein, S. E. (1994). Affective disorders in people with autism: A review of published cases. *Journal of Autism and Developmental Disorders*, 24, 587–601.
- Lerner, M. D., Mazefsky, C. A., Weber, R. J., Transue, E., Siegel, M., & Gadow, K. D. (2017). Verbal ability and psychiatric symptoms in clinically referred inpatient and outpatient youth with ASD. *Journal of Autism and Developmental Disorders*, 48(11), 3689–3701.
- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., et al. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism Developmental Disorders*, 36, 849–861.
- Li, Q., Han, Y., Dy, A. B. C., & Hagerman, R. J. (2017). The gut microbiota and autism Spectrum disorders. *Frontiers in Cellular Neuroscience*, 11, 120.
- Lopata, C., Toomey, J. A., Fox, J. D., Volker, M. A., Chow, S. Y., Thomeer, M. L., Lee, G. K., Rodgers, J. D., McDonald, C. A., & Smerbeck, A. M. (2010). Anxiety and depression in children with

- HFASDs: Symptom levels and source differences. *Journal of Abnormal Child Psychology*, 38(6), 765–776.
- Magnuson, K. M., & Constantino, J. N. (2011). Characterization of depression in children with autism spectrum disorders. *Journal of Developmental & Behavioral Pediatrics*, 32, 332–340.
- Mangiola, F., Ianiro, G., Franceschi, F., Fagioli, S., Gasbarrini, G., & Gasbarrini, A. (2016). Gut microbiota in autism and mood disorders. *World Journal of Gastroenterology*, 22(1), 361–368.
- Matson, J. L., & Nebel-Schwalm, M. S. (2007). Comorbid psychopathology with autism spectrum disorder in children: An overview. *Research in Developmental Disabilities*, 28, 341–352.
- Mazurek, M. O. (2014). Loneliness, friendship, and well-being in adults with autism spectrum disorders. *Autism*, 18, 223–232.
- Perry, D. W., Marston, G. M., Hinder, S. A. J., Munden, A. C., & Roy, A. (2001). The phenomenology of depressive illness in people with a learning disability and autism. *Autism*, 5, 265–275.
- Reiss, S., & Valenti-Hein, D. (1990). Development of a psychopathology rating scale for children with mental retardation. *Journal of Consulting and Clinical Psychology*, 62(1), 28–33.
- Richa, S., Fahed, M., Khoury, E., & Mishara, B. (2014). Suicide in autism spectrum disorders. *Archives of Suicide Research*, 18(4), 327–339.
- Righi, G., Benevides, J., Mazefsky, C., Siegel, M., Sheinkopf, S. J., Morrow, E. M., et al. (2017). Predictors of inpatient psychiatric hospitalization for children and adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48, 1–11.
- Rinehart, N. J., Bradshaw, J. L., Brereton, A. V., & Tonge, B. J. (2002). A clinical and neurobehavioural review of high-functioning autism and Asperger's disorder. *Australian and New Zealand Journal of Psychiatry*, 36, 762–770.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47, 921–929.
- Skokauskas, N., & Frodila, T. (2015). Overlap between autism spectrum disorder and bipolar affective disorder. *Psychopathology*, 48, 209–216.
- Skokauskas, N., & Gallagher, L. (2010). Psychosis, affective disorders and anxiety in autistic spectrum disorders: Prevalence and nosological considerations. *Psychopathology*, 43(1), 8–16. Epub 2009 Nov 6. Review.
- Spek, A. A., van Ham, N. C., & Nyklicek, I. (2013). Mindfulness-based therapy in adults with an autism spectrum disorder: A randomized controlled trial. *Research in Developmental Disabilities*, 34, 246–253.
- Sterling, L., Dawson, G., Estes, A., & Greenson, J. (2007). Characteristics associated with presence of depressive symptoms in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 38, 1011–1018.
- Stewart, M. E., Barnard, L., Pearson, J., Hasan, R., & O'Brien, G. (2006). Presentation of depression in autism and Asperger syndrome: A review. *Autism*, 10, 103–116.
- Turygin, N. C., Matson, J. L., MacMillan, K., & Konst, M. (2013). The relationship between challenging behavior and symptoms of depression in intellectually disabled adults with and without autism spectrum disorders. *Journal of Developmental and Physical Disabilities*, 25, 475–484.
- Vannucchi, G., Masi, G., Toni, C., Dell'Osso, L., et al. (2014). Bipolar disorder in adults with Asperger's syndrome: A systematic review. *Journal of Affective Disorders*, 168, 151–160.
- Williams, K., Brignell, A., Randall, M., Silove, N., & Hazell, P. (2013). Selective serotonin reuptake inhibitors (SSRIs) for autism spectrum disorders (ASD) (Review). *The Cochrane Collaboration*. Cochrane Database Syst Rev, 2013(8), Art. No.: CD004677.

Mood Stabilizers

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Definition

Mood stabilizer is a term used to describe a group of medications that are used in the treatment of bipolar disorder. Currently, mood stabilizers including lithium, valproic acid, and its derivatives, carbamazepine and several antipsychotic medications, are now approved for the treatment of bipolar disorder.

See Also

► [Lithium](#)

References and Reading

- Kowatch, R. A., Strawn, J. R., & Danielyan, A. (2011). Mood stabilizers: Lithium, anticonvulsants and others. In A. Martin, L. Scahill, & C. Kratochvil (Eds.), *Pediatric psychopharmacology: Principles and practice* (pp. 297–311). New York: Oxford University Press.

Moral Cognition

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This chapter is intended to show what is to be understood by the term of moral cognition, as it has evolved throughout history, philosophy, and psychology, how it is connected to autism spectrum disorders, and how novel approaches in moral research can improve knowledge and understanding on relationships between moral cognition and pathogenetic models of ASD.

A glance at the current research literature underlines two topical issues: First, moral research in the field of ASD is still a niche product, and second, while many of the studies suggest a deficit in moral reasoning, only few try to analyze the causes behind these deficits in a way that also considers the content of the presented moral tasks.

Definition

A universally valid and uniform definition of what is meant by the term morality is virtually impossible because of so many different approaches and theories arising from disciplines like philosophy, psychology, and sociology. However, it is undisputed that in most definitions,

morality is described as a regulatory, norm-referenced, and value-oriented system which accrues from cultural and religious experience in the context of a social community and serves as a code of conduct within this very community (Gert 2005; Gert and Gert 2017). From the perspective of an individual, moral is a special type of communication that enables one to express respect or disrespect for a person or an action. It is therefore a binary code of good and evil, respectively, good and bad. The repertoire of moral values arises from conditioning and acquisition of conditions of respect and disrespect (Luhmann 2008).

Concerning the concept of moral cognition, there is also no unequivocal definition. But, most researchers in the field would agree with Greene (2015, p. 40) who states: "... the field of moral cognition does not study a distinctive set of cognitive processes. [...] Instead, it studies a set of psychological phenomena bound together by a common function." And many proponents of this functional characterization would argue that the core function of morality is to promote and sustain cooperation (see, e.g., Greene 2015). This view also entails that there is no specialized "moral organ" (Hauser 2006), but to the contrary "that the 'moral brain' is, more or less, the whole brain, applying its computational powers to problems that we, on non-neuroscientific grounds, identify as 'moral'." (Greene 2015, p. 1013).

Based on this functional characterization of the concept of moral cognition and emphasizing the role of cooperation, it is clear that moral cognition and social cognition and their developmental courses are heavily entwined: The beginning of human life is in stark contrast to the fully expanded potential a human being can develop over lifetime. We are physiologically premature, deficient, and obligatory interdependent (Gehlen 2014; Portmann 1941). Without social bonding, intense care, and dissemination of information, we would be unable to bring into being this potential. Thus, human beings are innately intended to fit into a social and cultural context. Therefore cooperation and pro-social behavior has been an evolutionary advantage and can be seen as behavioral expression of social cognition.

Social cognition can be regarded as a dynamic constructive and conceiving process of

perception, categorization, recall, and evaluation of social stimuli, and it is a prerequisite for moral cognition (Schaafsma et al. 2015). The combination of implicit, automatic, and perceptual categories with explicit categories of attention and experience of agency and concentration has influence on the way we perceive social situations with moral content.

From a psychological point of view, moral or moral behavior can be seen as a function of mind and environmental structures. The objective is that (moral) decisions have to be made in an uncertain world under the constraints of limited knowledge, resources, and time. What is at issue is the match or mismatch of social cognition with the structure of a social environment (Gigerenzer 2010).

The difficulties in communication and social reciprocal interactions, typically associated with a diagnosis of ASD, already indicate that especially the preconditions for normative moral assessments, namely, adequate perception and categorization of social stimuli, do not appear to be available. The result could be altered moral judgments and moral behavior in individuals with ASD.

Historical Background

Since human beings have developed writing, they have registered codes of conduct. Listing, how to behave in a particular situation under certain circumstances, was not only a way to develop a complex progression of rules in terms of social coexistence but also an effective instrument to influence the perceptions, emotions, and intuitions (Haidt and Kesebir 2010) of the society for which those codices were written.

These virtue-based and descriptive considerations of moral were always anchored in a cultural and religious context and reached from classical antiquity to modern times. In the late eighteenth century, moral philosophy tried to separate the code of conduct from cultural and religious influences and the philosophical approaches consider moral either from a deontological or a consequential perspective. The deontological perspective – with its most important representative

Kant (1788/ 2003) – focuses on duties and prohibits actions that don't comply with certain rights and obligations, while the consequential approach focuses on the best possible outcome and judges actions only by their consequences (Bentham 1823/ 2008). Although there were first accounts by Freud (1923/ 2001) and Durkheim (1925/ 1973) for a psychological and sociological perspective on moral, the first empirical attempts were conducted by Piaget (1932/ 1965) and Kohlberg and Kramer (1969) from a developmental perspective: In Piaget's studies on "genetic epistemology," up to 6 year old typically developing children judged the culpability of a person by rather use of the outcome than use of the motive. Kohlberg's method was to interview children, adolescents, and adults about hypothetical moral dilemmas. They investigated moral development by analyzing the answers of the participants explaining their decisions. Based on the resultant narrations, Kohlberg formulated a theory of moral development in the sense of universally ordered stages, including pre-conventional, conventional, and post-conventional moral. However, these rationalistic psychological theories of moral development were heavily criticized and do not have many proponents anymore, whereas some of their methods, especially the presentation of moral dilemmas, were maintained and are still applied in many studies.

The complexity of moral cognition can be illustrated by means of being confronted with moral dilemmas: Participants confronted with a moral dilemma have to deal with many different aspects of social cognition. The common band of all dilemmas is the conflict, respectively, the unsolvability of the given problem associated with antagonistic moral rules, making it impossible not to fail in one of the two options of action.

Current Knowledge

In a commentary on the annual research review of Happé and Frith (2014), Charles A. Nelson III (2014) describes moral as a highly complex behavior. He suggests that high-level social abilities are dependent on more elementary core

social constructs. Furthermore, he argues that many aspects of social development are experience-expectant or experience-dependent.

In his book *A natural history of human morality*, Michael Tomasello (2016) describes the development of human values and norms from an evolutionary perspective. He suggests that development of human morality took place in a two-stage process, whose beginning is marked by the ability to be compassionate and empathetic: The joint intentionality of human beings is based on the principle to project one's self into the other's perspective. Both partners have to know the normative standards of each role. The ability to cooperate and to take joint action allows for a first step of what is called a second-personal morality. The second step in the history of moral development took place 150,000 years ago, following from the enlargement of the social unions and tribes. Within these cultural associations, a collective intentionality developed from the second-personal morality that yielded cultural conventions, norms, and institutions (Searle 1995). This group-related objective morality describes what is seen as good or morally adequate within these cultural associations.

However, a closer consideration of moral reasoning reveals a large number of underlying highly complex processes: The moral content of a social situation has to be perceived and categorized. Actual information content needs to be adjusted on the basis of internalized social norms, own experience, and memory. Various possible courses of action must be balanced against the perceived situation and compared with imagined and anticipated consequences of a possible decision (Beißert and Hasselhorn 2016; Derryberry et al. 2005). Since Beißert and Hasselhorn (2016) have provided evidence that moral development measured with everyday life moral transgression scenarios is not correlated with general intelligence, it must be assumed that processes of moral reasoning follow a different principle.

The spectrum of social context expands with advancing age. Besides other members of the family, this can be kindergarten, school, narrow circle of friends, peer group, and media. The acquisition

of these values and moral rules from the extended social environment develops through imitation and identification (Oerter 1974). Kohlberg and Kramer (1969) also assume that even adolescents and adults process and internalize those values and rules predominantly without undertaking an explicit analysis of meaning and source of these behavior rules. Thus, if the majority of moral impartation takes place by means of expressed emotions, the recipient of moral values must be able to "read" human behaviors (Schaafsma et al. 2015), especially those expressing emotions like respect or contempt.

Being a crucial component of human emotional experience and social interaction, empathy is defined as the ability to share affective states and allows us to predict and understand the feelings, motivations, and actions of others (Bernhardt and Singer 2012). In the range of moral research, there is sufficient evidence that empathy rather than the ability to mindreading is a necessary aspect for adequate moral reasoning (Batson et al. 1983; Haidt and Kesebir 2010; Myrsky et al. 2010).

While Kohlberg represented a rationalistic approach, defined as conviction that explicit and deliberate reasoning is the most important and most reliable way to gain moral knowledge, in the 1980s and especially in the 1990s, many researches began to doubt on rationalism, so that implicit cognitive processes became more and more the focus of scholarly interest in reasoning, judgment, and decision-making in general and in moral cognition in particular. The following current psychological theories of moral cognition have all in common that they emphasize the role of implicit, automatic processes. They differ however with respect (i) to which implicit processes are involved (affective vs. non-affective processes) and (ii) to which degree implicit processes impact moral cognition (exclusive vs. complementary).

At present, the following three psychological theories of moral cognition are discussed: (1) the theory of moral grammar (Hauser 2006; Mikhail 2007); (2) the "social-intuitionist" theory by Haidt (2001); and (3) dual-process accounts, especially the variant put forward by Greene et al. (2001).

The theory of moral grammars assumes an innate and universal set of abstract principles on the one hand and culturally switchable parameters on the other for building specific instances of moral systems (Hauser 2006; Mikhail 2007). Hauser writes (2006, p. 298): “Every newborn child could build a finite but large number of moral systems. When a child builds a particular moral system, it is because the local culture has set the parameters in a particular way. If you are born in Pakistan, your parameters are set in such a way that killing women who cheat on their husbands is not only permissible but obligatory, and the responsibility of family members.” Once the parameters have been set, the grammar automatically and unconsciously generates judgments of right and wrong for an infinite variety of acts and inactions. Therefore, moral judgments don’t depend neither on conscious reasoning, nor on emotions. Instead, moral judgments trigger emotions, which are “downstream, pieces of psychology triggered by an unconscious moral judgment” (Hauser, pp. 30–31). Consequently, emotions are a subsequent process stage resulting from a preceding unconscious and automatic moral grammar module.

The social intuitionist model (SIM) by Jonathan Haidt (2001) emphasizes the role of emotion with recourse to David Hume (1739/2011), who claimed that “reason is the slave of passion.” Consequently, moral cognition is based on an implicit, automatic process (e.g., passion, emotion, or intuition). Furthermore, the degree of emotionality depends on the type of moral dilemmas presented to the participants (Greene et al. 2001; Shenhav and Greene 2014). Conscious reasoning about moral concerns is a subsequent process preceded by (moral) intuitions, being “the sudden appearance in consciousness of a moral judgment, including an affective valence (good-bad, like-dislike), without any conscious awareness of having gone through steps of searching, weighing evidence, or inferring a conclusion” (Haidt 2001, p. 818). Consequently “moral intuitions (including moral emotions) come first and directly cause moral judgments” (Haidt 2001, p. 814).

The dual-process theory for moral decisions of Greene et al. (2001) also operates with the

distinction between cognitive and affective subcategories. Dual-process theories differentiate between fast automatic inferences describable as heuristics and slower, conscious decisions demanding complex computations caused by normative principles such as “thou shall not kill” (Kahneman 2011). Greene et al. (2001) states that the influence of affect on moral judgments depends on given contextual factors that frame the moral dilemma. Depending on the personal emotional involvement, the judgment on the one hand can follow utilitarian approaches if the contextual factors suggest low emotional involvement, and on the other hand the judgment is based on categorical principles if the context indicates high emotional involvement. According to this, moral actions in which the individual is involved very closely and personally might be driven by an emotional and automatic component and as opposed to this actions that are remote and impersonal elicit a rational decision. Greene assumes that conscious and rational decisions are utilitarian and on the contrary a high level of emotionality and participation evokes a more categorical behavior, thus turning Kant upside down and putting him on his feet; in other words, rationality is the basis of utilitarian decisions, and emotional involvement leads to categorical moral decisions and actions.

FMRI studies have shown that moral reasoning correlates with the representation of emotions and intuitions (Reniers et al. 2012) and that the degree of emotionality depends on the type of moral dilemma presented to the participants (Greene et al. 2001; Shenhav and Greene 2014). Nevertheless a meta-analysis of imaging studies, concerning different aspects of moral thought (Bzdok et al. 2012), comes to the conclusion that due to neurotopographical aspects, the neural network of moral decisions is domain-global and can be divided in cognitive as well as affective subsystems.

From a psychological perspective, the task to make a moral judgment can only then be successful if the one who judges is able to empathize with the person on whom the moral judgment has to be imposed (Krahn and Fenton 2009). From an evolutionary point of view, the

ability to moral behavior and moral reasoning can be derived from the interdependence hypothesis (Tomasello 2016): All social species within a social group are mutually dependent regarding their survival. The need of collaborative effort urged early humans to cooperate and thus to consider and anticipate the needs of their counterpart. In order to develop a theory of mind that is able to draw conclusions about emotions, thoughts, and intention of the cooperating partner, especially in complex and dangerous situations, it was necessary to decode and categorize facial expression, gesture, and body posture and to derive an inner state of the counterpart. Consequently, if ToM is necessary for moral reasoning and moral judgment, then individuals with ASD should have difficulties to pass moral judgments in the same way as typically developed individuals.

Kohlberg's approach assumes well-developed language and expressive abilities. Since individuals with ASD have deficits in communication skills (WHO 1993), they can make less use of words describing mental states. Since Takeda et al. (2007) could show significant correlations between moral reasoning and verbal ability in ASD, it is obvious that the linguistic quality of their replies leads to lower scores compared to typically developed peers (Shulman et al. 2012).

Turiel (1983) emphasized the ability to distinguish between moral norms on the one hand and mere social conventional norms on the other. Therefore, he developed tasks, where the display takes the form of pictorial representations, and in addition to the description of those pictorial-presented transgressions, child participants were asked for an evaluation of the seriousness of the transgression. The first use of this task including individuals with ASD showed that children with ASD are able to differentiate between moral and conventional transgressions. Furthermore, there was no correlation between theory of mind abilities (level of ability to solve false belief tasks) and the tendency to differentiate between moral and conventional transgressions (Blair 1996).

Typically developed individuals base their judgments of the moral acceptability of behavior

on their emotional response to that behavior. Brewer et al. (2015) suggest that individuals with ASD do not seem to use emotional information and may rely more on rules to judge moral acceptability. Others tend to explain differences between neurotypically developed individuals and individuals with ASD by analyzing the justifications for a moral decision: While participants with typical development explained their decisions with significantly more abstract rules, participants with ASD tended to nonspecific condemnations of the described behavior to justify their judgment (Shulman et al. 2012).

In their theoretical analysis *Autism, empathy and questions of moral agency*, Krahn and Fenton (2009) propose a distinction between cognitive and affective empathy. Dziobek et al. (2008) showed that individuals with high-functioning autism have access to affective empathy but reveal deficits in cognitive empathy. Based on this evidence, it is to be assumed that there is a capability in ASD to show empathy which is a prerequisite for moral cognition and moral behavior. Suggesting that HFA are capable of moral agency, they encourage to find methods to unfold the term of morality. Based on this evidence, it is to be assumed that there is a capability in ASD to show empathy which is a prerequisite for moral cognition and moral behavior.

A further study focused on the difference between quantitative results and reported personal experience. The results in terms of quantitative analysis suggest that adolescents with ASD demonstrate similar empathetic concern like typically developed peers but have significantly higher personal distress and deviant moral reasoning. In the qualitative survey, adolescents with ASD see themselves as individuals with empathetic concern but without the ability to use these feelings in sociomoral situations (Senland and Higgins-D'Alessandro 2013).

The question of the role of implicit processes in moral reasoning remains open. It is still unclear to what extent empathy and the subsequent moral judgment depend on the social relationships of the protagonists depicted in the moral tasks or dilemmas, respectively what access do

participants with ASD have to such social schemas of relationship. In a study by Patil et al. (2016), hypothetical moral choices were investigated in adults with high-functioning autism, and here again, the role of empathy was the focus of the investigation. The results suggest that adults with ASD are able to differentiate between personal and impersonal dilemmas, they make similar behavioral choices compared to controls, and they exhibit equivalent moral judgments despite deficits in social cognition and emotional processing. Thus again, the claim of Krahn and Fenton (2009) for an unfolding of moral cognition and social cognition in terms of research in ASDs should be addressed.

Although compared to other areas of social cognition the number of studies that survey moral reasoning are few and far between, nevertheless there is evidence that the impairment in understanding others' mind hinders the development of an intent-based moral judgment in children with ASD (Margoni and Surian 2016). People with ASD do not seem to use emotional information and may rely more on rules to judge moral acceptability (Brewer et al. 2015). In the study of Fadda et al. (2016), children with ASD who showed difficulties in perspective-taking, nevertheless were able to take account for the consequences of actions but not for psychological information, in particular the subjective state of an agent in a task, where they should judge the consequences of moral and non-moral actions. Baez et al. (2012) describe a pattern of social cognition deficits characterized by the decreased ability to implicitly encode and integrate contextual information in order to gain access to the social meaning. Comparing moral judgments and the evaluation of those by adults with ASD and neurotypical controls, regarding intentional action of protagonists in moral situations, only subtle differences had been found (Buon et al. 2013). In a qualitative analysis of self-rating the own empathetic concern in sociomoral situations, individuals with ASD envision themselves as empathetic but unable to apply those feelings in moral reasoning conditions (Senland and Higgins-D'Alessandro 2013).

Future Directions

The approaches of Haidt (2001) and Greene et al. (2001) seem to be well suited for appropriate experimental designs to investigate moral cognition of individuals with ASD, especially since the emphasis of their accounts is on emotions. In contrast the application of the moral grammar model by Hauser (2006) appears to be a less researched issue regarding the performance of ASD.

A new approach to unfold morality and emotion is the psychological constructionist account of Cameron et al. (2015). It proceeds from a general correspondence of morality and emotion, emerging from basic cross-domain psychological processes. Perception, memory, emotions, and moral judgments are not separate and isolated entities but emerge from a combination of basic psychological elements that can be combined flexibly and yield different mental states (Barrett and Satpute 2013). The relevant ingredients of this large-scale, domain-general network can always be newly combined, creating a coherent system of functional properties matching with every perceived situation. The interaction of a core affect and conceptualized knowledge in a specific situation leads to discrete emotions and an ensuing judgment of what was perceived. In this context core affect means a physiological state and a non-reflective feeling (Russell 2003), perceptible as valence (positive versus negative) and arousal (high versus low). Important for the process of situative conceptualizing is the transformation of core affect by means of conceptual knowledge comprising general semantic knowledge, autobiographical memories, and situation-specific knowledge (Lindquist 2013). Thus, moral reasoning evoked by moral dilemmata induces a neurophysiological state of valence and arousal, and reconciled with conceptual knowledge, it leads to a moral judgment. A closer look upon the constructionist model of core affect and conceptual knowledge shows that there is an evident congruence with the concept of schemas. The combination of affective states with conceptual knowledge can be understood as a constructionist pattern containing causal representations that

epitomize integrative situational models. Following the definition of Dienstbier et al. (1980), a schema is a “complex of values, attitudes, cognitions, and affective responses that are elicited by and interact with new relevant information. That interaction determines the resultant attributions, decisions, and behaviors” (Dienstbier et al. 1980, p. 194). Schemas represent knowledge of all kinds of content, from ideologies to culture and from behavior in social situations to the meaning of a certain word. Schemas do not only include structural elements but also have a processual component and for this unfold several executive functions (Spada and Mandl 1988, p. 126). Development of social cognition as a prerequisite of moral abilities finds its social world of experience in numberless commonly experienced situations that comprise moral values, attitudes, and the affective disposition of the perceiver, having impact on the development of moral schemas. The development of mindreading is a seminal process including qualitative changes in representational ability (Christensen and Michael 2016), and the construction of integrated situation models requires access to a theory of mind (ToM). Since a schema serves as an implicit (intuitive) automatism to categorize social perceptions, Kintsch and van Dijk (1978) consider schemas as a construction of a framework that integrates information and highlights key relationships in order to provide a model for certain social situations (e.g., moral dilemmas). If so, a person who lacks adequate schemas fails to pick out, structure, and organize the relevant causal factors of a situation and will be unable to understand the context and to predict how the situation will develop.

In order to make such multilayered details of a moral problem present, an investigation of holistic inferential processes, having recourse on social schemas, can show what kind of social schemas are deficient in ASD. Using a schema-theoretical perspective in the field of moral cognition, patients with ASD seem to show a different decision and response behavior, particularly when the dilemmas are based on social schemas involving close social relationships (Schaller et al. 2019).

In order to determine the influence of implicit and explicit processes, moral grammar, and social schemas on moral decision-making in its overall complexity, future studies should develop refined methodological procedures to unfold the factors influencing the process of moral cognition.

References and Reading

- Baez, S., Rattazzi, A., Gonzalez-Gadea, M. L., Torralva, T., Vigliecca, N. S., Decety, J., ... Ibanez, A. (2012). Integrating intention and context: assessing social cognition in adults with Asperger syndrome. *Frontiers in Human Neuroscience*, 6(11), 302. <https://doi.org/10.3389/fnhum.2012.00302>.
- Barrett, L. F., & Satpute, A. B. (2013). Large-scale brain networks in affective and social neuroscience: Towards an integrative functional architecture of the brain. *Current Opinion in Neurobiology*, 23(3), 361–372. <https://doi.org/10.1016/j.conb.2012.12.012>.
- Batson, C. D., O'Quin, K., Fultz, J., Vanderplas, M., & Isen, A. M. (1983). Influence of self-reported distress and empathy on egoistic versus altruistic motivation to help. *Journal of Personality and Social Psychology*, 45(3), 706–718. <https://doi.org/10.1037/0022-3514.45.3.706>.
- Beißert, H. M., & Hasselhorn, M. (2016). Individual differences in moral development: Does intelligence really affect children's moral reasoning and moral emotions? *Frontiers in Psychology*, 7, 1–10. <https://doi.org/10.3389/fpsyg.2016.01961>.
- Bentham, J. (2008). An introduction to the principles of morals and legislation (chapters I–V). In M. Warnock (Ed.), *Utilitarianism and on liberty* (pp. 17–51). Oxford: Blackwell Publishing Ltd. <https://doi.org/10.1002/9780470776018.ch1>.
- Bernhardt, B. C., & Singer, T. (2012). The neural basis of empathy. *Annual Review of Neuroscience*, 35, 1–23. <https://doi.org/10.1146/annurev-neuro-062111-150536>.
- Blair, R. J. (1996). Brief report: Morality in the autistic child. *Journal of Autism & Developmental Disorders*, 26(5), 571–579.
- Brewer, R., Marsh, A. A., Catmur, C., Cardinale, E. M., Stoycos, S., Cook, R., & Bird, G. (2015). The impact of autism spectrum disorder and alexithymia on judgments of moral acceptability. *Journal of Abnormal Psychology*, 124(3), 589–595. <https://doi.org/10.1037/abn0000076>.
- Buon, M., Dupoux, E., Jacob, P., Chaste, P., Leboyer, M., & Zalla, T. (2013). The role of causal and intentional judgments in moral reasoning in individuals with high functioning autism. *Journal of Autism and Developmental Disorders*, 43(2), 458–470. <https://doi.org/10.1007/s10803-012-1588-7>.
- Bzdok, D., Schilbach, L., Vogeley, K., Schneider, K., Laird, A. R., Langner, R., & Eickhoff, S. B. (2012).

- Parsing the neural correlates of moral cognition: ALE meta-analysis on morality, theory of mind, and empathy. *Brain Structure & Function*, 217(4), 783–796. <https://doi.org/10.1007/s00429-012-0380-y>.
- Cameron, C. D., Lindquist, K. A., & Gray, K. (2015). A constructionist review of morality and emotions. *Personality and Social Psychology Review*, 19(4), 371–394. <https://doi.org/10.1177/1088868314566683>.
- Christensen, W., & Michael, J. (2016). From two systems to a multi-systems architecture for mindreading. *New Ideas in Psychology*, 40, 48–64. <https://doi.org/10.1016/j.newideapsych.2015.01.003>.
- Derryberry, W. P., Wilson, T., Snyder, H., Norman, T., & Barger, B. (2005). Moral judgment developmental differences between gifted youth and college students. *Journal of Secondary Gifted Education*, 17(1), 6–19. <https://doi.org/10.4219/jsge-2005-392>.
- Dienstbier, R. A., Kahle, L. R., Willis, K. A., & Tunnell, G. B. (1980). The impact of moral theories on cheating – Studies of emotion attribution and schema activation. *Motivation and Emotion*, 4(3), 193–216. <https://doi.org/10.1007/BF00995419>.
- Durkheim, E. (1973). *Moral education*. New York: The free press.
- Dziobek, I., Rogers, K., Fleck, S., Bahnemann, M., Heekeren, H. R., Wolf, O. T., & Convit, A. (2008). Dissociation of cognitive and emotional empathy in adults with Asperger syndrome using the Multifaceted Empathy Test (MET). *Journal of Autism and Developmental Disorders*, 38(3), 464–473. <https://doi.org/10.1007/s10803-007-0486-x>.
- Fadda, R., Parisi, M., Ferretti, L., Saba, G., Foscoliano, M., Salvago, A., & Doneddu, G. (2016). Exploring the role of theory of mind in moral judgment: The case of children with autism spectrum disorder. *Frontiers in Psychology*, 7(April), 523. <https://doi.org/10.3389/fpsyg.2016.00523>.
- Freud, S. (2001). Gesammelte Werke, Band 13: Jenseits des Lustprinzips. In A. Freud (Ed.), *Massenpsychologie und Ich-Analyse. Das Ich und das Es*. Frankfurt am Main: Fischer Verlag.
- Gehlen, A. (2014). *Der Mensch: Seine Natur und seine Stellung in der Welt (16.)*. Wiebelsheim: AULA-Verlag.
- Gert, B. (2005). *Morality: Its Nature and Justification*, Revised Edition. New York: Oxford University Press.
- Gert, B., & Gert, J. (2017). The definition of morality. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy* (Fall 2017 Edition). Retrieved from <https://plato.stanford.edu/archives/fall2017/entries/morality-definition>.
- Gigerenzer, G. (2010). Moral satisficing: Rethinking moral behavior as bounded rationality. *Topics in Cognitive Science*, 2(3), 528–554. <https://doi.org/10.1111/j.1756-8765.2010.01094.x>.
- Greene, J. D. (2014). The cognitive neuroscience of moral judgment and decision making. In M. S. Gazzaniga & G. R. Mangun (Eds.), *The cognitive neurosciences* (5th ed., pp. 1013–1023). Cambridge, MA: MIT Press.
- Greene, J. D. (2015). The rise of moral cognition. *Cognition*, 135, 39–42. <https://doi.org/10.1016/j.cognition.2014.11.018>.
- Greene, J. D., Sommerville, R. B., Nystrom, L. E., Darley, J. M., & Cohen, J. D. (2001). An fMRI investigation of emotional engagement in moral judgment. *Science (New York, N.Y.)*, 293(5537), 2105–2108. <https://doi.org/10.1126/science.1062872>.
- Haidt, J. (2001). The emotional dog and its rational tail: A social intuitionist approach to moral judgment. *Psychological Review*, 108(4), 814–834. <https://doi.org/10.1037//0033-295X.108.4.814>.
- Haidt, J., & Ksebir, S. (2010). Morality. In *Handbook of social psychology* (pp. 797–832). Hoboken: Wiley. <https://doi.org/10.1002/9780470561119.socpsy002022>.
- Happé, F., & Frith, U. (2014). Annual research review: Towards a developmental neuroscience of atypical social cognition. *Journal of Child Psychology and Psychiatry*, 55(6), 553–577. <https://doi.org/10.1111/jcpp.12162>.
- Hauser, M. (2006). *Moral minds: How nature designed our universal sense of right and wrong*. New York: Harper Collins.
- Hume, D. (2000). *A treatise of human nature*. Second ed. Oxford: Oxford University Press. (Originally published 1739).
- Hume, D. (2011). *A treatise of human nature*. Oxford: University Press. ISBN-10: 0199596336 ISBN-13: 978-0199596331.
- Kahneman, D. (2011). *Thinking, fast and slow*. New York: Farrar, Straus and Giroux.
- Kant, I. (2003). Kritik der praktischen Vernunft. In H. D. Brandt, & H. F. Klemme (Eds.) (Philosophi). Hamburg: Meiner.
- Kant, I. (2015). *Critique of Practical Reason*. Cambridge: Cambridge University Press. ISBN-10: 1107467055 ISBN-13: 978-1107467057.
- Kintsch, W., & van Dijk, T. a. (1978). Toward a model of text comprehension and production. *Psychological Review*, 85(5), 363–394. <https://doi.org/10.1037/0033-295X.85.5.363>.
- Kohlberg, L., & Kramer, R. (1969). Continuities and discontinuities in childhood and adult moral development. *Human Development*, 12(2), 93–120. <https://doi.org/10.1159/000270857>.
- Krahn, T., & Fenton, A. (2009). Autism, empathy and questions of moral agency. *Journal for the Theory of Social Behaviour*, 39(2), 145–166. <https://doi.org/10.1111/j.1468-5914.2009.00402.x>.
- Lindquist, K. A. (2013). Emotions emerge from more basic psychological ingredients: A modern psychological constructionist model. *Emotion Review*, 5(4), 356–368. <https://doi.org/10.1177/1754073913489750>.
- Luhmann, N. (2008). *Die Moral der Gesellschaft*. In D. Horster (Ed.) (First Edit). Frankfurt am Main: Suhrkamp.
- Margoni, F., & Surian, L. (2016). Mental state understanding and moral judgment in children with

- autistic spectrum disorder. *Frontiers in Psychology*, 7 (SEP), 1–5. <https://doi.org/10.3389/fpsyg.2016.01478>.
- Mikhail, J. (2007). Universal moral grammar: Theory, evidence and the future. *Trends in Cognitive Sciences*, 11(4), 143–152. <https://doi.org/10.1016/j.tics.2006.12.007>.
- Myyry, L., Juujärvi, S., & Pessa, K. (2010). Empathy, perspective taking and personal values as predictors of moral schemas. *Journal of Moral Education*, 39(2), 213–233. <https://doi.org/10.1080/03057241003754955>.
- Nelson, C. A. (2014). Commentary: Becoming social—a commentary on Happé & Frith (2014). *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 55(6), 578–581. <https://doi.org/10.1111/jcpp.12254>.
- Oerter, R. (1974). *Moderne Entwicklungspsychologie* (9. Auflage ed.). Donauwörth: Auer.
- Patil, I., Melsbach, J., Hennig-Fast, K., & Silani, G. (2016). Divergent roles of autistic and alexithymic traits in utilitarian moral judgments in adults with autism. *Scientific Reports*, 6, 23637. <https://doi.org/10.1038/srep23637>.
- Piaget, J. (1965). *The moral judgement of the child* (trans: Gabain, M.). New York: Free Press (original work published 1932).
- Portmann, A. (1941). Die biologische Bedeutung des ersten Lebensjahres beim Menschen. *Schweizerische Medizinische Wochenschrift*, 71(1941), 921–924.
- Reniers, R. L. E. P., Corcoran, R., Völlm, B. A., Mashru, A., Howard, R., & Liddle, P. F. (2012). Moral decision-making, ToM, empathy and the default mode network. *Biological Psychology*, 90(3), 202–210. <https://doi.org/10.1016/j.biopsycho.2012.03.009>.
- Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychological Review*, 110(1), 145–172. <https://doi.org/10.1037/0033-295X.110.1.145>.
- Schaafsma, S. M., Pfaff, D. W., Spunt, R. P., & Adolphs, R. (2015). Deconstructing and reconstructing theory of mind. *Trends in Cognitive Sciences*, 19(2), 65–72. <https://doi.org/10.1016/j.tics.2014.11.007>.
- Schaller, U. M., Biscaldi, M., Fangmeier, T., Tebartz van Elst, L., & Rauh, R. (2019). Intuitive moral reasoning in high-functioning autism spectrum disorder: A matter of social schemas? *Journal of Autism and Developmental Disorders*, 49(5), 1807–1824. <https://doi.org/10.1007/s10803-018-03869-y>.
- Searle, J. R. (1995). *The construction of social reality*. New York: Free Press.
- Senland, A. K., & Higgins-D'Alessandro, A. (2013). Moral reasoning and empathy in adolescents with autism spectrum disorder: Implications for moral education. *Journal of Moral Education*, 42(2), 209–223. <https://doi.org/10.1080/03057240.2012.752721>.
- Shenhav, A., & Greene, J. D. (2014). Integrative moral judgment: Dissociating the roles of the amygdala and ventromedial prefrontal cortex. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 34(13), 4741–4749. <https://doi.org/10.1523/JNEUROSCI.3390-13.2014>.
- Shulman, C., Guberman, A., Shilling, N., & Bauminger, N. (2012). Moral and social reasoning in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(7), 1364–1376. <https://doi.org/10.1007/s10803-011-1369-8>.
- Spada, H., & Mandl, H. (1988). *Wissenspsychologie*. In H. Mandl, & H. Spada (Eds.), München: Beltz Psychologie Verlags Union.
- Takeda, T., Kasai, K., & Kato, N. (2007). Moral judgment in high-functioning pervasive developmental disorders. *Psychiatry and Clinical Neurosciences*, 61(4), 407–414. <https://doi.org/10.1111/j.1440-1819.2007.01678.x>.
- Tomasello, M. (2016). *A natural history of human morality*. Cambridge, MA: Harvard University Press.
- Turiel, E. (1983). *The development of social knowledge: Morality and convention*. Cambridge: Cambridge University Press.
- World Health Organization (WHO). (1993). *The ICD-10 classification of mental and behavioral disorders. Diagnostic criteria for research*. Geneva: World Health Organization.

More Than Words

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Definition

More Than Words is a parent-based social-pragmatic intervention program for children with autism spectrum disorders (ASD). It includes education and social support for parents and early language intervention for children with ASD under the age of 5. The program is led by a Hanen-certified speech-language pathologist (SLP) and aims to help parents learn more about communication and, in turn, support their children in increasing their language and communication skills.

Historical Background

The Hanen Centre is a Canadian charitable organization that was founded in 1975 by Ayala Hanen

Manolson to increase knowledge and training of adults to help children become more effective communicators. *More Than Words* was adapted from another Hanen program, *It Takes Two to Talk*, to help meet the needs of parents and their children on the autism spectrum. The parent guidebook was written by Fern Sussman, a speech-language pathologist, and was published in 1999 by the Hanen Centre in Toronto, Ontario (The Hanen Centre 2007).

Rationale or Underlying Theory

The goal of this program is to “empower parents to become the primary facilitator of their child’s communication and language development thereby maximizing the child’s opportunities to develop communication skills in everyday situations” (The Hanen Centre 2007). *More Than Words* reflects a family-centered model of intervention. This program believes that since the parent or caregiver is the most important and constant element in the child’s life, they have an invaluable opportunity to provide consistent interventions in a natural setting to assist their children in experiencing meaningful interactions.

A social-pragmatic theory of language acquisition is the theoretical foundation for *More Than Words*, asserting that the development of communication occurs in the context of the interaction between the child and the important adults in his or her life. Strategies taught in the program are intended to help parents become more responsive to their children’s communication attempts in order to facilitate their language skills. Parents also learn ways to manipulate the environment to increase joint attention and the motivation to communicate.

Goals and Objectives

The *More Than Words* program outlines three main objectives for parents in helping to support their children’s communication skills: parent education, early language intervention, and social support for parents (The Hanen Centre 2007). As part of the parent education component, parents develop a

better understanding of their child’s strengths and challenges by learning about their child’s learning style and sensory preferences. They also learn about basic concepts relating to language and communication and what stage of communication their child is in. This helps them to set goals and recognize communication attempts.

The second objective of *More Than Words* is for parents to carry out early communication and language intervention. The Hanen-certified speech-language pathologist (SLP) works collaboratively with parents to develop goals and implement responsive strategies that can be applied to interactions with their child across a variety of contexts. This is a flexible process and is intended to become a consistent, yet natural part of the day-to-day exchanges between the parent and child. As part of this intervention, parents video-record interactions and strategy implementation with their child. The SLP then reviews the video with the parents and provides feedback to help increase their awareness of their interactions along with the communicative behaviors demonstrated by their child. The SLP facilitates the parents’ metacognitive thinking in the video review to encourage their consistent use of the new strategies.

The third objective is to provide social support for parents, who are at greater risk for fatigue, frustration, depression, and anxiety than parents without a child with developmental disabilities. Support within this context is valuable because it comes from a professional who understands the unique strengths and challenges of their child, as well as from other parents in the group who can empathize and share experiences.

Treatment Participants

More Than Words was designed for parents and their children under the age of 5 with ASD. It can be used with children who are verbal or nonverbal.

Treatment Procedures

The program is led by a Hanen-certified SLP and is offered to groups of up to eight families at one

time. There are three major components of this program. First, there is a preprogram assessment and baseline video recording of an interaction between the parent and child. Next, parents participate in seven sessions of group training, totaling a minimum of 17.5 h. These sessions are led by the SLP and include discussion, PowerPoint presentations, and video clips that aim to help parents learn about early communication and language, how to manipulate the environment to help their child communicate, identify their child's stage of communication, and how to set realistic communication goals. The parents' interactions with their children are video recorded, and three of these recordings are reviewed with the SLP. As part of the video feedback sessions, the parent observes and then describes the strategies they implemented with their child. The SLP probes parent understanding of what was observed and how what a parent did either facilitated or failed to facilitate communication change. The SLP provides coaching to the parents, based on the strategies taught during the parent education sessions. The program is supported by a guidebook, DVD, and PowerPoint slides with video clips.

Efficacy Information

This program seeks to target the communication challenges that are present in children with autism spectrum disorders. The guidebook provides illustrations and step-by-step examples, giving parents a readable and easy to understand resource with opportunities to practice what they learn in the context of everyday activities. Intervention strategies used in this approach, including modeling, reinforcement, and imitation, are commonly used best practices in early education and speech-language therapy and are directly related to the individual child's communication goals. The video-recorded interactions between the child and parent are periodically reviewed by the certified SLP, ensuring that the strategies are implemented in an effective and consistent manner.

Two studies have examined the effectiveness of *More Than Words* for families and children with ASD. The first examined the facilitation of parental understanding of ASD and the social

communication of their children following implementation of the *More Than Words* program (McConachie et al. 2005). Results indicated that parents increased their use of facilitative strategies and children with ASD increased their vocabulary size. A second study found similar results for three families of children 2.8–3.2 years of age with parents increasing their use of responsive strategies and children increasing their vocabulary (Girolametto et al. 2007).

Outcome Measurement

A number of studies provide empirical evidence demonstrating positive outcomes of a social-interactionist intervention for children with ASD (Chapman et al. 1986; Duchan 1989; Mirenda and Dollennan 1986). There is also a recent body of literature that supports this type of intervention being successfully carried out by parents (Aldred et al. 2004; Mahoney and Perales 2003). Two published studies specifically examine the efficacy of *More Than Words*. The first was a controlled trial measuring the effectiveness of parent training on the use of facilitative interaction strategies to enhance the communication skills and behaviors of their children (McConachie et al. 2005). This quasi-experimental study included 26 children and parents in an experimental group and 25 children and parents in a control group. Parents in the experimental group participated in the *More Than Words* training program. The results showed a measureable effect on both the parents' and their children's communication skills. The parents of children diagnosed with ASD showed an increase in responsiveness. The children whose parents attended *More Than Words* had significantly greater vocabulary size than those in the control group, as measured by parental report on the *MacArthur-Bates Communicative Development Inventory (CDI)* (McConachie et al. 2005). The CDI is a frequently used measure of word and gesture comprehension and word and sentence production that is used to assess outcomes following the *More Than Words* program.

Another study examined the social interaction of three preschool children with ASD following

their mothers' participation in *More Than Words* (Girolametto et al. 2007). This was a multiple case study design that aimed to extend the previous study by using microanalytic coding procedures on the recordings of parent–child interactions. The results found that mothers increased their use of spontaneous interaction strategies and their children demonstrated increased vocabulary development and social interaction (Girolametto et al. 2007).

Qualifications of Treatment Providers

The *More Than Words* program is led by a certified speech-language pathologist who has completed a specialized training by the Hanen Centre. First, the SLP needs to be trained in the *It Takes Two to Talk* program. Then, they can complete the 3-day, intensive, small-group training on *More Than Words*, led by a Hanen instructor.

See Also

- [Hanen Approach](#)
- [Language Acquisition](#)

References and Reading

- Aldred, C., Green, J., & Adams, C. (2004). A new social communication intervention for children with autism: Pilot randomized controlled treatment study suggesting effectiveness. *Journal of Child Psychology and Psychiatry*, 40, 1–11.
- Chapman, K., Leonard, L., & Mervis, C. (1986). The effects of feedback on young children's inappropriate word use. *Journal of Child Language*, 13, 101–117.
- Duchan, J. (1989). Evaluating adults' talk to children: Assessing adult attunement. *Seminars in Speech and Language*, 10, 7–27.
- Girolametto, L., Weitzman, E., & Greenburg, J. (2003). Training day care staff to facilitate children's language. *American Journal of Speech-Language Pathology*, 12, 299–311.
- Girolametto, L., Sussman, F., & Weitzman, E. (2007). Using case study methods to investigate the effects of interactive intervention for children with autism spectrum disorders. *Journal of Communication Disorders*, 40(6), 470–492.
- Mahoney, G., & Perales, F. (2003). Using relationship-focused intervention to enhance the social-emotional functioning of young children with autism spectrum disorders. *Topics in Early Childhood Special Education*, 23(2), 77–89.
- McConachie, H., Val Randle, V., Hammal, D., & Le Couteur, A. (2005). A controlled trial of a training course for parent of children with suspected autism spectrum disorders. *Journal of Pediatrics*, 147, 335–340.
- Mirenda, P., & Dollennan, A. (1986). Effects of adult interaction style on conversational behavior in students with severe communication problems. *Language, Speech, and Hearing Services in Schools*, 17, 126–141.
- Pearce, P., Girolametto, L., & Weitzman, E. (1996). The effects of focused stimulation intervention on mothers of late-talking toddlers. *Infant-Toddler Intervention*, 6, 213–227.
- Pepper, J., & Weitzman, E. (2004). *It takes two to talk: A practical guide for parents of children with language delays* (2nd ed.). Toronto: The Hanen Centre.
- Siller, M., & Sigman, M. (2002). The behaviors of parents of children with autism predict the subsequent development of their children's communication. *Journal of Autism and Developmental Disorders*, 32(2), 77–89.
- Sussman, F. (1999). *More than words: Helping parents promote communication and social skills in children with autism spectrum disorder*. Toronto: The Hanen Centre.
- The Hanen Centre. (2007). *More than words research summary*. Retrieved from <http://www.hanen/web/Portals/0/HostedFiles/MTWResearchSummary2007.pdf>
- Weitzman, E., & Greenburg, J. (2002). *Learning language and loving it: A guide to promoting children's social, language, and literacy development in early childhood settings* (2nd ed.). Toronto: The Hanen Centre.
- Woods, J., Kashinath, S., & Goldstein, H. (2004). Effects of embedding caregiver implemented teaching strategies in daily routines on children's communication outcomes. *Journal of Early Intervention*, 26, 175–193.

Moro Reflex

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Synonyms

[Startle reflex](#)

Definition

The Moro reflex, also known as the startle reflex, was initially described by the Austrian pediatrician Ernst Moro (1874–1951). It represents one of the infantile reflexes, and it may be observed since birth in all newborns up to 5 months of age. The reflex is triggered when (1) a baby's head falls backward or quickly changes position and/or (2) the baby is startled by an unexpected loud noise. The reflex causes the baby initially to extend the neck and spread out widely the arms and legs (abduction) and then pull the arms back in a clasping motion (adduction). These movements are sometimes associated with cry. The Moro reflex, as well as other infantile reflexes (e.g., the grasping reflex), is considered a residual behavior from when nonhuman primates clung to their mothers swinging through the trees (Thies and Travers 2001).

The absence, persistence, and/or exacerbation of the Moro reflex in childhood and adulthood is generally associated with profound disorders of the motor system (e.g., cerebral palsy). The assessment of the Moro reflex is carried out by pediatricians to evaluate the integration of the central nervous system in early infancy.

As early primitive reflexes, such as the Moro reflex, are gradually extinguished at around 5 months of age, research in this area in the ASD population is very limited. A retrospective study by Teitelbaum et al. (2004) suggested that infants later diagnosed with ASD show abnormal movement patterns that can be interpreted as primitive reflexes “gone astray”; that is, some reflexes are not extinguished at the appropriate age in development, whereas others fail to appear when they should. More empirical research is needed to specifically investigate the development of the Moro reflex in the ASD population.

References and Reading

Teitelbaum, O., Benton, T., Shah, P. K., Prince, A., Kelly, J. L., & Teitelbaum, P. (2004). Eshkol-Wachman movement notation in diagnosis: The early detection of Asperger's syndrome. *Proceedings of the National*

Academy of Sciences of the United States of America, 101(11), 909–11914.

Thies, M., & Travers, J. F. (2001). *Human growth and development through the lifespan*. Thorofare: Slack.

Morocco and Autism

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Historical Background

As the only country in North Africa that was able to conserve its political stability, Morocco has garnered a stellar reputation amongst foreign investors. The growing interest in the Moroccan kingdom by business leaders has undoubtedly contributed to its economic growth over the past decade, which further embellished the country's reputation. In fact, the country ranked 68th out of 190 nations on the *Doing Business 2017* survey. However, behind the country's growing success lie many striking weaknesses in some of its most vital sectors. Moroccan education, for instance, has been controversial for a long time and remains a focus of political debate. After Morocco's independence in 1956, the country's educational system has been suffering from numerous challenges.

Healthcare is a sector that is unarguably underdeveloped. The quality of care provided and the serious lack of staff and resources are a challenge. There are even more challenges for psychiatric care. While Morocco is ahead of its neighboring countries when it comes to mental health policy, the rules are rarely enforced. The lack of trained providers is another challenge.

The diagnosis and management autism has not been a topic of interest until recently. Autism is considered a “disability” in Morocco and policies and legislation focus more globally on disability than on autism per se.

The main laws that have been issued in Morocco to protect disabled people (including

individuals with autism spectrum disorder) have been the following:

1. Law 92-07 concerning social protection of disabled people – adopted in 1993.
2. Law 03-10 relative to accessibility in addition to a certain number of decrees about the application of laws issued in 2003.
3. Several decrees following the previous laws that urge the applications of laws.

However, despite the activeness of the government in issuing these laws to protect the rights of disabled people, there has not been much improvement made in the field. This reflects the lack of resources to implement the issued laws, the limited emphasis on inclusion of people with disabilities, and issues of lack of acceptance – all factors that contribute to a certain extent to perpetuating disability.

In 2004, a National Disability Survey revealed some quite shocking numbers about the case of disabilities in Morocco.

- One million and a half Moroccan (5.12%) live with a disability, and that on average, a household of four people counts at least one disabled person.
- One out of five disabled people in Morocco never go to a specialized institution.
- Only 1% of disabled people in Morocco benefit from health insurance.
- More than 70% of disabled people don't have any level of instruction.
- The rate of schooling amongst disabled people in Morocco is of only 32.4% as opposed to a 92.6% among the nondisabled.
- 88.6% of disabled people of age above 15 have no professional activity.
- The poverty rate among the disabled is multiple times higher than that amongst the nondisabled.

Since the release of this report in 2004, the government has been active in terms of social protection legislations. Attempts have been made to provide obligatory medical insurance; medical assistance policy and labor legislation has been

enacted. The Moroccan government has acted to protect the rights of people with disabilities and these efforts occur in five main fields: legal capacity, education, institutionalization, women, and disability.

In 2008, the government elaborated the project of law 62-09 which is meant to consolidate the rights of disabled people which have been achieved through a national conference in 2008, followed by four regional meetings. Unfortunately implement of this has been difficult. The adoption of 2011 constitution and through the high orders of King Mohamed 6 has encouraged adoption of this law.

Legal Issues and Mandates for Service

In a recent legislative effort by the Moroccan parliament intended for the protection and advancement of persons with disabilities, an initiative known as the Draft Framework Law or the Draft Law 97.13 was adopted, marking the first legislation addressing the rights of the disabled to be considered by the Moroccan parliament since it ratified the International Treaty on Disability Rights in 2009. Although hopeful for the future of disabled Moroccan citizens, the draft was met with much criticism, particularly from the Human Rights Watch.

Disabled citizens of Morocco have often been treated as objects of charity as opposed to equal citizens, which has inevitably led to an environment of discrimination and stigmatization. Many see this draft law as an opportunity to initiate a large-scale change in the perception of disabilities in Morocco, but as it stands, the Draft Framework Law maintains outmoded perceptions of disabilities and in actuality, places even more restraints on the rights of disabled persons.

Morocco's recent signing of the International Human Rights Treaty, which established the right of disabled persons outlined in the Draft Framework Law as below the international standard, preceded the consideration of the Draft Framework Law. In a letter to the Moroccan parliament, the Human Rights Watch noted the many flaws present in the current Draft Framework law,

among which includes the absence of a clear establishment of the right to education for the disabled due to the segregation of students with special needs with students in regular classrooms, which further amplifies the societal disassociation of disabled persons with the general population. This leads many parents to withdraw special needs students from school. In their letter, the Human Rights Watch demanded proper accommodations in educational settings for special needs students in Morocco. In addition, the general Moroccan population does not adhere to the Draft Framework Law due to a lack of awareness, and certain initiatives should be put in place in order for the law to be as effective as possible.

Current Treatments and Treatment Centers

According to Soumia Amrani, the vice president of Moroccan Collection for Promoting Handicap Rights, resources and treatment centers for disabled persons are readily available in Morocco, but are grossly overpriced and unaffordable for most Moroccan families, especially given the increasing unemployment rate. However, the professional disciplines necessary for the treatment and diagnosis autism are relatively new in Morocco. Starting in the 1980s, and especially after 2000, Moroccan parent-activists have been reaching out to foreign experts in order to change the status quo. They have taken their lead from the Anglo-American Neurocognitive behavioral model of the treatment and diagnosis of autism, rather than the French psychoanalytic one. Moroccan activists have been focusing on raising autism awareness, lobbying government agencies, and establishing the proper infrastructure required for the identification and education of autistic children.

Recently, as part of the National Initiative for Human Development (INDH), King Mohammad VI inaugurated the “Ashourouk” Care Center for Children with Autism, which can enroll up to 100 children, in order to ensure that children with autism and special needs are provided with adequate educational and social integration. The

king noted that among the themes outlined by this initiative was assisting those with special needs in preserving their dignity and preventing them from falling into extreme poverty or isolation. Although much progress has been made in recent years, Morocco lags behind European countries with respect to providing resources a care for autistic children.

Overview of Research Directions

Unfortunately, the majority of ASD research has been conducted in the United States, and this commitment to provide accommodations and understand people with this disorder has not been equally reflected in other countries, such as Morocco. Because of this, parents and families of individuals with autism have been pushed to establish start-up organizations with the sole purpose of providing resources and care for individuals affected with autism. These start-up organizations are working with larger, international organizations in an attempt to advance the understanding of those affected with autism in Morocco. One start-up, in particular the Association Amal pour Enfants aux Benzoints Specifiques Mentaux (A.A.E.B.S.M), was founded by Moroccan activists for disability rights – many of whom had a relative affected by the disorder – and is currently centered in Casablanca. The primary goal of this organization is to provide support for individuals and families affected by ASD by improving public perception and educating the general public about ASD.

Along with local organizations, larger international organizations, such as Autism Speaks, are dedicated to providing care and support for those affected by ASD and have similar objectives to locally run organizations. Autism Speaks has recently began an initiative in Morocco dedicated to providing effective resources to Moroccan individuals and families affected by ASD. However, the incomplete analysis of the resources available in the country has been an obstacle and as a result, Autism Speaks has partnered with students from Worcester Polytechnic Institute in a project designed to collect and analyze data regarding

treatment and education options currently available to autistic children in Rabat, Morocco. The analysis and conclusions of this study will be made available to parents of autistic children in Morocco and will hopefully assist in future projects aimed to advance autism treatment in Morocco.

Overview of Training

Historically, Moroccan psychiatry has been heavily influenced by French psychoanalysis. In the 1980s and 1990s, the very first child psychiatrists in Morocco received specialized training in France, since at the time, there were no training programs for child psychiatry in Morocco. This psychoanalytical approach to psychiatry proved to be ineffective, as many of the children who were treated by these psychiatrists were later admitted into adult psychiatric hospitals. The year 2008 marked a significant advancement in the field of child psychiatry in Morocco, with the opening of two new child psychiatry departments in Casablanca and Salé, pioneering the first child psychiatry training programs in Morocco, which distanced itself from the usual psychoanalytic approach, focusing instead on a biological approach. In fact, over the past couple of decades, Moroccan psychiatry has generally been moving away from psychoanalysis and gearing towards a more biologically based psychiatry.

Major psychiatric hospitals in Morocco have been operating in a style which uses international scientific consensus of the DSM or ICD diagnoses while promoting psychotherapy as well as behavioral and cognitive approaches. In general, the younger generation of psychiatrists in Morocco takes an “eclectic” approach to psychiatry, as many were trained in psychoanalytic theory and as a result see some benefit in thinking psychodynamically; however, they were exposed to neurobiology and pharmacology and believe that cognitive and behavioral therapy play a role in the treatment of mental illness and developmental disorders. They also tend to view the treatment methods of the previous generation of French and Moroccan psychiatrists as damaging and counterproductive.

See Also

- [Autism Speaks](#)
- [Egypt and Autism](#)
- [Sub-Saharan Africa and Autism](#)

References and Reading

- Admin. One man's death points to shortcomings in Morocco's mental health infrastructure. *REPORTING MOROCCO*, 14 Feb 2017. morocco.roundearthmedia.org/one-mans-death-points-shortcomings-moroccos-mental-health-infrastructure
- Alami, A. Fewer than 30 percent of Moroccans have health Insurance. *The New York Times*, 27 Mar 2013. www.nytimes.com/2013/03/28/world/middleeast/fewer-than-30-percent-of-moroccans-have-health-insurance.html
- Bibeau, G. (1997). Cultural psychiatry in a creolizing world. *Transcultural Psychiatry*, 34(1), 9–41.
- Chamak, B. (2008). Autism and social movements: French parents' associations and international autistic individuals' organizations. *Sociology of Health & Illness*, 30(1), 76–96.
- Crapanzano, V. (1973). *The Hamadsha: A study in Moroccan ethnopsychiatry*. Berkeley: University of California Press.
- Educational facilities and classes launched for children with autism. *Morocco World New*, 5 May 2017. www.morocoworldnews.com/2017/05/215832/educational-facilities-classes-launched-children-autism/
- Elmallem, S. Morocco: A few key facts about your next business destination. *La Chambre De Commerce Du Montréal Métropolitain*, CCMM, 28 Sept 2017. www.ccm.ca/en/news-morocco-a-few-key-facts-about-your-next-business-destination/
- Hart, B. G. (2016). *Making an autism world in Morocco: Parent activism, therapeutic practice, and the proliferation of a diagnosis*. Columbia University Academic Commons. <https://doi.org/10.7916/D8W66KJN>.
- How is autism treated? *Autism Speaks*, 25 July 2012. [www.autismspeaks.org/what-autism/treatment](http://autismspeaks.org/what-autism/treatment)
- Letter to Moroccan Parliament on draft disability law. *Human Rights Watch*, 26 Oct 2015. www.hrw.org/news/2015/10/26/letter-moroccan-parliament-draft-disability-law
- Moroccan Educational System: Problems and solutions. *Morocco World News*, 9 Mar 2016. www.morocoworldnews.com/2016/03/181000/moroccan-educational-system-problems-and-solutions/
- Jawad Garmah. (2016). Moroccan Educational System: Problems and solutions. *Morocco World News*. www.morocoworldnews.com/2016/03/181000/moroccan-educational-system-problems-and-solutions/
- Morocco: Inauguration of Center for Children With Autism in Bouskoura – Consolidated Roy approach for comprehensive human development. *All Africa*, Maghreb Arabe Presse, 31 Jan 2013. allafrica.com/stories/201302011199.html

Mortality

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Definition

The term “autism” is used in this entry to describe all autism spectrum disorders, which include the various types of pervasive developmental disorders according to DSM-IV (American Psychiatric Association [APA] 2000) and ICD-10 (World Health Organization [WHO] 1992).

The life expectancy of people with autism is of interest to parents, health professionals, and service providers concerned with these peoples' lifetime needs. Mortality information that includes the causes of death is important as it can help parents and professionals focus on reducing associated risks and ultimately the rate of mortality among people with autism.

Historical Background

Occasional deaths have been reported in all general follow-up studies of individuals with autism (Ballaban-Gil et al. 1996; Billstedt et al. 2005; Fombonne et al. 1989; Howlin et al. 2004; Kobayashi et al. 1992; Larsen and Mouridsen 1997). Causes of death include traffic accidents (Fombonne et al. 1989; Larsen and Mouridsen 1997); unrecognized volvulus in a woman in a long-term psychiatric institution (Larsen & Mouridsen); status epilepticus (Howlin et al. 2004); and cases of drowning, pneumonia, and complications arising from long-term psychotropic medication (Ballaban-Gil et al. 1996).

However, systematic studies dealing with mortality and causes of death in autism are rare. There are difficulties in obtaining an adequate sample size to calculate specific risks, in studying samples over a suitable time span, and in establishing the cause(s) of death.

Current Knowledge

Standardized mortality ratio (SMR) is frequently used to measure excessive mortality. The SMR is the quotient of the observed to the expected numbers of deaths, and an SMR of 1 indicates that the observed number of deaths is no different from what would be predicted for the general population: An SMR value greater than 1 indicates that the observed mortality exceeds expectations in the group under study.

Only three systematic follow-up studies specifically dealing with mortality and causes of death in individuals with autism have been published so far. Shavelle et al. (2001) and Pickett et al. (2006) reported mortality and causes of death in 13,111 people with autism who were receiving services from the California Department of Developmental Services between 1983 and 2002. The overall SMR was 2.4, indicating a mortality rate more than twice as high as for the general population. The increase in mortality was larger in females than in males. Mortality was associated with mental retardation with an SMR of 3.1 for participants with moderate, severe, or profound retardation, whereas the SMR was 1.4 for individuals with no or only mild mental retardation. Epilepsy was strongly associated with death risk. SMR was 36.9 in participants with epilepsy and moderate, severe, or profound mental retardation against 22.6 in those with no or mild mental retardation. Suffocation (SMR = 51.4 and 5.7, respectively) and drowning (SMR = 13.7 and 3.9, respectively) were also noted to be frequent specific causes of death and, like epilepsy, associated with the level of cognitive impairment. Similar results were obtained in a Danish study (Isager et al. 1999; Mouridsen et al. 2008) in which an overall SMR of 1.9 was reported in a nationwide cohort of 341 individuals with autism (average age 43 years) observed between 1960 and 2007. Again, the SMR was particularly high in females. Epilepsy (SMR = 35.0) and infectious diseases were among the most common causes of death. Epilepsy was notably marked in deceased females; five of the eight deceased females had epilepsy. Different kinds of infectious diseases (meningitis,

pneumonia, appendicitis) were associated with death in seven individuals. In a Swedish community-based study of 120 individuals with autism, mean age 33.2 years, Gillberg et al. (2010) found an overall SMR of 5.6. They also noted that the mortality rate was particularly high in females. Associated medical diseases (including epilepsy with cognitive impairment) and accidents accounted for most of the observed causes of deaths.

Prevention efforts to decrease mortality in people with autism need to address the conditions that are the immediate causes of death (i.e., infectious diseases, epilepsy, and accidents). However, the behavioral characteristics that define autism often make care difficult and can lead to exacerbations in the state of somatic illness or diagnostic delay, eventually leading to death-causing illness (Larsen and Mouridsen 1997; Mouridsen et al. 2008). Assessing pain and discomfort in a cognitively impaired, nonverbal patient is difficult, and of paramount importance is the involvement of the parent(s) or other care providers, if available. The best pain assessment by proxy is that provided by caregivers or family members who know the patient well. Only they can identify changes from a patient's base line behavior that may signify pain and discomfort. Patients may be uncooperative or combative if they do not understand the need for help. It is important to realize that many people with autism react badly to any change in their environment, and therefore, a visit to a general practice or a hospital can be very alarming for them, in particular, if an unplanned acute hospital admission is necessary (Scarpinato et al. 2010; Souders et al. 2002). In some patients, conventional peri- and postoperative management is impossible and alternative strategies are needed (Dell et al. 2008; Van Der Walt and Moran 2001). Likewise, venipunctures, intravenous insertions, and initiation or change of medical treatment can be difficult to carry through. It is also noteworthy that Williams et al. (2000) found that 62% of parents reported difficulty giving medication to their children with autism.

An association between autism and epilepsy has been consistently reported, and the literature

presents a wide range of co-occurrence estimates from 5% to 46% (Spence and Schneider 2009). These authors also report that more severe intellectual disability is associated with greater rates of epilepsy. Considering the high prevalence of epilepsy in autism, it is therefore important to suspect the comorbidity of epilepsy in every individual presenting with autism. It follows that understanding the needs of this group is particularly important within any major epilepsy service. Since epilepsy is strongly associated with death risk, careful monitoring of anticonvulsant treatment is essential in those individuals with a concomitant occurrence of autism and epilepsy. However, the clinical identification of seizures may be difficult in some cases. The diagnosis of partial complex seizures, in particular, can be complicated by the presence of atypical body movements and behavioral patterns often seen in association with autism (Bauman 2010). Obtaining a high-quality EEG can also be difficult, and effective communication usually has to be through a caregiver.

Since accidents are often avoidable, special attention should be paid to the safety of community surroundings. In the Mouridsen et al. (2008) study, two participants, who both lived in specialized institutions for autistic people, managed to swallow dangerous objects and choke on them during an unsupervised period. According to the standards of the caregivers, the two patients should never be left unsupervised.

As the rates of autism diagnoses increase (Rutter 2005), it becomes more and more imperative for health care providers in any setting – family, institutions, and primary or acute care – to understand the unique challenges of people with autism. The findings published so far underscore the importance of maintaining sufficient competence levels in the fields of internal medicine and neurology within the autism care system to recognize and properly manage somatic as well as neurological diseases. As there is little information about the best practice for providing care to people with autism in institutional or hospital settings, it is important to develop nursing standards that address care for people with autism.

Future Directions

To conclude, mortality is increased in people with autism, and individuals with diminished cognitive functioning or epilepsy are at particularly high risk of a reduction in life expectancy. However, increased understanding of the most common causes of death can help parents and other caregivers focus on reducing risk and, ultimately, the rate of mortality among people with autism.

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.) Washington, DC: APA Press.
- Ballaban-Gil, K., Rapin, I., Tuchamn, R., & Shinnar, S. (1996). Longitudinal examination of the behavioural, language, and social changes in a population of adolescents and young adults with autistic disorder. *Pediatric Neurology*, 15, 217–223.
- Bauman, M. (2010). Medical comorbidities in autism: Challenges to diagnosis and treatment. *Neurotherapeutics*, 7, 320–327.
- Billstedt, E., Gillberg, I. C., & Gillberg, C. (2005). Autism after adolescence: Population-based 13- to 22-year follow-up study of 120 individuals with autism diagnosed in childhood. *Journal of Autism and Developmental Disorders*, 35, 351–360.
- Dell, D. D., Feleccia, M., Hicks, L., Longstreth-Papsun, E., Politsky, S., & Trommer, C. (2008). Care of patients with autism spectrum disorder undergoing surgery for cancer. *Oncology Nursing Forum*, 35, 177–182.
- Fombonne, E., Talan, I., Bouchard, F., & Lucas, G. (1989). A follow-up study of childhood psychosis. *Acta Paedopsychiatrica*, 52, 12–35.
- Gillberg, C., Billstedt, E., Sundh, V., & Gillberg, I. C. (2010). Mortality in autism: A prospective longitudinal community-based study. *Journal of Autism and Developmental Disorders*, 40, 352–357.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45, 212–219.
- Isager, T., Mouridsen, S. E., & Rich, B. (1999). Mortality and causes of death in pervasive developmental disorders. *Autism*, 3, 7–16.
- Kobayashi, R., Mutara, T., & Yoshinaga, K. (1992). A follow-up study of 201 children with autism in Kyushu and Yamaguchi areas, Japan. *Journal of Autism and Developmental Disorders*, 22, 395–411.
- Larsen, F. W., & Mouridsen, S. E. (1997). The outcome in children with childhood autism and Asperger syndrome originally diagnosed as psychotic: A 30-year follow-up study of subjects hospitalized as children. *European Child and Adolescent Psychiatry*, 6, 181–190.
- Mouridsen, S. E., Brønnum-Hansen, H., Rich, B., & Isager, T. (2008). Mortality and causes of death in autism spectrum disorders: An update. *Autism*, 12, 403–414.
- Pickett, J. A., Paculdo, D. R., Shavelle, R. M., & Strauss, D. J. (2006). 1998–2002 update on “Causes of death in Autism”. *Journal of Autism and Developmental Disorders*, 36, 287–288.
- Rutter, M. (2005). Incidence of autism spectrum disorders: Changes over time and their meaning. *Acta Paediatrica*, 94, 2–15.
- Scarpinato, N., Bradley, J., Kurbjun, K., Bateman, X., Holtzer, B., & Ely, B. (2010). Caring for the child with an autism spectrum disorder in the acute care setting. *Journal for Specialists in Pediatric Nursing*, 15, 244–255.
- Shavelle, R. M., Strauss, D. J., & Pickett, J. (2001). Causes of death in autism. *Journal of Autism and Developmental Disorders*, 31, 569–576.
- Souders, M. C., DePaul, D., Freeman, K. G., & Levy, S. E. (2002). Caring for children and adolescents with autism who require challenging procedures. *Pediatric Nursing*, 28, 455–562.
- Spence, S. J., & Schneider, M. T. (2009). The role of epilepsy and epileptiform EEGs in autism spectrum disorders. *Pediatric Research*, 65, 599–606.
- Van Der Walt, J. H., & Moran, C. (2001). An audit of perioperative management of autistic children. *Pediatric Anaesthesia*, 11, 401–408.
- Williams, G. P., Dalrymple, N., & Neal, J. (2000). Eating habits of children with autism. *Pediatric Nursing*, 26, 259–264.

Motivating Operation

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Definition

Motivating operations (MO) is a general term to describe antecedent events which momentarily alter the effects of a reinforcing or punishing consequence, and therefore alter the future frequency of behavior related to that consequence. Motivating operations alter the effectiveness of reinforcers and punishers in two ways, which designates different classifications of MOs.

Establishing operations (EO) increase the effectiveness of a consequence (see ► [“Establishing Operations”](#)), and abolishing operations (AO) decrease the reinforcing or punishing effectiveness (see ► [“Abolishing Operations”](#)) (Laraway et al. 2003). The term MO is important because it refers to “not only how much someone wants something, but how hard they will work to get it” (Langthorne and McGill 2009, p. 22).

Historical Background

Establishing operation was a term originally coined by Keller and Schoenfeld (1950) to describe a series of motivational events and their effect on behavior. Since, the designation, conceptualization, and distinctions of MOs have evolved considerably.

Moving from the term Establishing Operation to Motivating Operation: Researchers in the field of behavior found that the term *EO* was limited as it only included events resulting in the relative increase in the effectiveness of a consequence; discarding those variables which decreased the value of a consequence. While the inherent limitations to this term were recognized (Michael 1982, 1993, 2000), it was deemed cumbersome to have different terms to describe each operation. More recently, there has been a push to utilize the general term, *MO*, and distinguish between EOs and AOs based on their relative effects on consequences and behavior (Laraway et al. 2003). For the remainder of this entry, *EO* will describe only variables that increase the reinforcer/punisher effectiveness, *AO* will denote only events resulting in the decreased reinforcer/punisher effectiveness, and *MO* will denote the general class of variables that alter the effectiveness of such consequences.

The designations are important due to the essential role that all MOs, including both EOs and AOs, have on the understanding of human behavior. The current conceptualization of MOs has been found to be important in the role of reinforcing or punishing consequences for behavior. Importantly, interventions to address challenging behavior and skill deficits in individuals

with autism spectrum disorders (ASD) have benefited from an analysis of motivating operations as an independent variable.

Role in operant conditioning: Historically, operant behavior has been conceptualized as a three-term contingency (Antecedent-Behavior-Consequence), in which a stimulus readily evokes a behavior because of a past history of reinforcement in the presence of that stimulus. The conceptualization of MOs added a variable that provides an explanation for momentary differences of such operants (Sundberg 1993). The importance of the MO cannot be understated. Motivation is paramount to understanding behavior. Although all operant behavior occurs due to its history with reinforcing or punishing consequences, the probability of such behavior being emitted is dependent on the relative effectiveness of such consequences. Specifically, if a reinforcer is more effective due to an EO, behavior will be more likely to be emitted at that time. If, in contrast, there is an AO in effect, the same behavior would be less likely to occur. In its simplest form, motivation can be described by its biological basis in states of deprivation or satiation. More complexly, motivation can come under the control of arbitrary relationships due to conditioning paradigms. Regardless of etiology, the analysis of the MO provides the conceptual foundation for why a reinforcer is effective for a person, at any moment in time (McGill 1999).

Conditioned motivating operations: A series of papers by Michael (e.g., 1982, 1993, 2000) distinguished between MOs that are unconditioned and those that are conditioned. Unconditioned MOs, or UMOs, are those events that change the effectiveness of a reinforcer or punisher with no learning history (e.g., food deprivation, sleep deprivation), while conditioned MOs, or CMOs, are those that are learned. Three distinct CMOs have been classified based on the process by which this learning occurs: the reflexive conditioned motivating operation (CMO-R), the transitive conditioned motivating operation (CMO-T), and the surrogate conditioned motivating operation (CMO-S). Each of these alter the value of some other stimulus, such that its presentation is momentarily establishing or abolishing.

The CMO-S refers to a previously neutral stimulus that has been correlated with another MO, and therefore acquires the same effects on behaviors and consequences as the original MO. The CMO-T refers to motivational events that establish another stimulus as a reinforcer. The CMO-R refers to motivational events which signal an improving or worsening set of conditions, therefore altering its own removal as a type of reinforcement or punishment (Langthorne and McGill 2009). Although complex, a conceptual understanding of these CMOs has enhanced intervention around challenging behavior and teaching language skills to individuals with ASD.

Distinguishing between different antecedent events: Motivating operations have often been confused with other antecedent variables, such as discriminative stimuli (S^D s, see ► [“Stimulus Control”](#)). The important distinction is that while S^D s signal the availability of reinforcement or punishment for a response, MOs increase or decrease the relative value of a reinforcer or punisher as a consequence for that response. This value altering effect (Laraway et al. 2003) results in a relative increase or decrease in the probability of a behavior occurring at that moment, rather than the consistency of responding under similar conditions in the future. In an applied example, a water fountain signals the availability of reinforcement for stopping and drinking (i.e., S^D) but whether you stop and drink depends on whether you are thirsty in the moment. Thirst, a noted state of deprivation, is an unconditioned EO for water as a reinforcer.

Current Knowledge

The concept of the MO is influential for any intervention that relies on reinforcement or punishment for its effect. Without an understanding of the MO, one cannot truly understand the impact of an intervention, as well as the variables in play if one is not working. For example, if a reinforcement program is put into place to increase academic behavior, it is paramount that the child is motivated for the reinforcer (i.e., there is an EO in place). Motivation for a reinforcer in this situation

may be one component of an effective intervention. Conversely, if in the previous example the child is not motivated for the reinforcer, academic work behavior may not increase, and the intervention may be deemed ineffective. This would be an error in the analysis, as in this situation, reinforcer saliency may be impeding progress. This same analysis applies to any situation in which the development of adaptive behaviors, or the omission of challenging behavior is contingent on delivery of a reinforcer. Most prominently, the MO has been highlighted in the areas of verbal behavior, decreasing challenging behavior, and skill acquisition instruction.

Verbal behavior: Motivating operations have been instrumental to providing a full account of language development and the acquisition of skills related to language (see ► [“Analysis of Verbal Behavior \(AVB\)”](#)). A thorough analysis of language development is pertinent for learners with ASD who may have deficits in basic communication skills. To illustrate, the concept of the MO is paramount for teaching mands, otherwise known as requests. Skinner (1957) described the “mand” as reliant on a salient EO as an antecedent condition. Meaning, requesting only occurs in the presence of motivation for something and is specifically reinforced by access to that which was requested. For example, requesting for food items will only occur when someone is hungry (a noted EO, state of deprivation). The specific reinforcer for this request would be food. “Mand training” is a particularly popular and important skill acquisition program for early learners with ASD. Requesting for items is dependent on motivation for that item or activity. Meaning, if a child is not motivated for the cookie, they are not going to independently ask for the cookie, regardless of whether they have the skill in their repertoire or not. This analysis is important in understanding how to teach these types of language skills. Sweeney-Kerwin et al. (2007) implemented a procedure to teach requesting to children with ASD. Importantly, their participants were dependent on seeing the things they were requesting which is inconsistent with the way in which requesting typically evolves. The importance of motivation was a critical component of the intervention as it

was necessary that the individuals were able to request for things, only when there was motivation in place. By manipulating aspects of the EO, the intervention was successful in teaching individuals with ASD to request for the things they wanted, without needing to see them in the environment. This type of requesting is paramount to the development of a requesting repertoire that is independent and generalizes across a variety of variables.

Similarly, the CMO-T has been used to support teaching the skill of asking for information (Hall and Sundberg 1987; Sundberg et al. 2002). In these interventions, individuals were taught to seek out information. Importantly, information had to become momentarily valuable for the questions to occur. An analysis of the CMO-T was pertinent in establishing motivation for information. This was done by repeatedly presenting individuals with preferred items in containers. Then, the instructor removed the items from the containers. Since the individuals had previously experienced the container and the item together, the presentation of the container without the item resulted in motivation for information about the location of the item, evoking the questions of “Where is the item?” This study effectively increased “question asking” as a skill for individuals with ASD who previously did not inquire for information. Without this careful analysis and application of CMOs, it is unlikely that the skill of asking for questions would have developed and persisted for learners with deficits in this complex area of language development.

Challenging behavior: The concept of the MO has implications in the area of challenging behavior as well. Many individuals with ASD engage in challenging behavior that impacts their access to a variety of environments and experiences (See ► “Challenging Behavior”). Literature on functional assessment (See ► “Functional Behavior Assessment; ► Functional Analysis”) has suggested that MOs play a primary role in the understanding of challenging behavior and its functional relationship to environmental events. A series of studies on functional analyses have shown that the presence or absence of MOs have directly influenced the relative success with

understanding the environmental variables related to challenging behavior during behavioral assessments (Call et al. 2004; Iwata et al. 1994). Similarly, MOs have been integral to interventions intended to decrease challenging behaviors in individuals with ASD (Smith and Iwata 1997). Motivation as an independent variable has been shown to impact the efficacy of behavior reduction procedures for stereotypy (Lang et al. 2009), destructive behavior (McComas et al. 2003), aggression (McGuinnis et al. 2010), self-injury (McComas et al. 2003), and noncompliance (Call et al. 2004). One example of this has been conceptualized in the literature on the CMO-R. Carbone and colleagues (2010) discussed the role of the CMO-R as it relates to problem behavior during discrete trial instruction (DTI; see ► “Lovaas Approach”). Specifically, this line of work has provided a conceptual analysis for the conditions during DTI which may establish instruction as aversive, and some practical modifications for reducing said aversiveness, resulting in a decrease in challenging behavior. Another important example relates to a common consequence for challenging behavior time-out. Removal from an environment, contingent on a challenging behavior, is intended to punish or decrease the future frequency of the challenging behavior. Unless there is motivation (i.e., an EO) for the time-in environment, it is unlikely that the time-out procedure, will suppress or decrease challenging behavior. This analysis of motivation is pertinent in understanding the mechanisms responsible for successful interventions.

Skill acquisition: The concept of the MO has also been employed to enhance skill acquisition instruction for individuals with ASD. Several instructional strategies have been identified as AOs to decrease escape-maintained behaviors during work and increase appropriate participation (Carbone 2013). For example, fast-paced instruction (Roxburgh and Carbone 2012), modified task difficulty (McComas, Thompson, implementing choice options (McComas et al. 2000), and the high probability (high-p) request sequence (Mace et al. 1988) have all been demonstrated to increase compliance during instruction by abolishing the aversive value of demands.

In addition, a series of studies have shown that contriving MOs during instruction can enhance repertoires such as engagement (Neely et al. 2013), play skills (Lang et al. 2010), discrimination (Lotfizadeh et al. 2012), eye contact (Carbone et al. 2013), social initiations (Taylor et al. 2005), and joint attention (Dube et al. 2004; Isaksen and Holth 2009). For example, Lang and colleagues (2010) found that when an AO for stereotypy was created prior to play sessions, children with ASD were more likely to engage in functional play with their peers during subsequent play sessions. Taylor et al. (2005) manipulated an establishing operation to increase social interactions for children with ASD. Here, the authors contrived motivations for a reinforcer that a peer was in control of and taught the participants to request for the items from their peers. The results of the study demonstrated that only when there was motivation for the item would the children request from their peers. Additionally, the authors reported that outside of the study, the children who participated were observed to request across other peers. One child requested in an untrained situation and in a novel environment as well. These data suggest the importance of motivation when planning for instruction with learners with ASD.

Future Directions

An understanding of MOs has been demonstrated to increase the efficacy of interventions, understand more fully behavioral presentation, and enhance the overall acquisition of necessary skills for individuals with ASD. Continued research and application of interventions based in the conceptualization of MOs are necessary to expand the analysis of behavior as it relates to these areas.

See Also

- ▶ Abolishing Operations
- ▶ Analysis of Verbal Behavior (AVB)
- ▶ Establishing Operations
- ▶ Functional Behavior Assessment
- ▶ Reinforcement

References and Reading

- Call, N. A., Wacker, D. P., Ringdahl, J. E., Cooper-Brown, L. J., & Boelter, E. W. (2004). An assessment of antecedent events influencing noncompliance in an outpatient clinic. *Journal of Applied Behavior Analysis*, 37, 145–157.
- Carbone, V. J. (2010). The role of the conditioned motivating operation (CMO-R) during discrete trial instruction of children with autism. *Journal of Early Intensive Behavioral Intervention*, 4, 658–680.
- Carbone, V. (2013). The establishing operation and teaching verbal behavior. *The Analysis of Verbal Behavior*, 29, 45–49.
- Carbone, V., O'Brien, L., Sweeney-Kerwin, E. J., & Albert, K. M. (2013). Teaching eye contact to children with autism: A conceptual analysis and single case study. *Education and Treatment of Children*, 36, 139–159.
- Dube, W. V., MacDonald, R. P. F., Mansfield, R. C., Holcomb, W. L., & Ahearn, W. H. (2004). Toward a behavioral analysis of joint attentions. *The Behavior Analyst*, 27, 197–207.
- Hall, G., & Sundberg, M. L. (1987). Teaching mands by manipulating conditioned establishing operations. *The Analysis of Verbal Behavior*, 5, 41–53.
- Isaksen, J., & Holth, P. (2009). An operant approach to teaching joint attention skills to children with autism. *Behavior Interventions*, 24, 215–236.
- Iwata, B. A., Dorsey, M. F., Slifer, K. J., Bauman, K. E., & Richman, G. S. (1994). Toward a functional analysis of self-injury. *Journal of Applied Behavior Analysis*, 27, 197–209.
- Keller, F. S., & Schoenfeld, W. N. (1950). *Principles of psychology: A systematic text in the science of behavior*. E. Norwalk: Appleton-Century Crofts.
- Lang, R., O'Reilly, M., Sigafoos, J., Lancioni, G. E., Machalicek, W., Rispoli, M., & White, P. (2009). Enhancing the effectiveness of a play intervention by abolishing the reinforcing value of stereotypy: A pilot study. *Journal of Applied Behavior Analysis*, 42, 889–894.
- Lang, R., O'Reilly, M., Sigafoos, J., Machalicek, W., Rispoli, M., Lancioni, G. E., & Fragale, C. (2010). The effects of an abolishing operation intervention component on play skills, challenging behavior, and stereotypy. *Behavior Modification*, 34(4), 267–289. <https://doi.org/10.1177/0145445510370713>
- Langthorne, P., & McGill, P. (2009). A tutorial on the concept of the motivating operation and its importance to application. *Behavior Analysis in Practice*, 2(2), 22–31.
- Laraway, S., Snycerski, S., Michael, J., & Poling, A. (2003). Motivating operations and some terms to describe them: Some further refinements. *Journal of Applied Behavior Analysis*, 36, 407–414.
- Lotfizadeh, A. D., Edwards, T. L., & Redner, R. (2012). Motivating operations affect stimulus control: A largely overlooked phenomenon in discrimination learning. *The Behavior Analyst*, 35(1), 89–100.
- Mace, F. C., Hock, M. L., Lalli, J. S., West, B. J., Belfiore, P., Pinter, E., et al. (1988). Behavioral momentum in the

- treatment of noncompliance. *Journal of Applied Behavior Analysis*, 21, 123–141.
- McComas, J., Hoch, H., Paone, D., & El-Roy, D. (2000). Escape behavior during academic tasks: A preliminary analysis of idiosyncratic establishing operations. *Journal of Applied Behavior Analysis*, 33, 479–493.
- McComas, J., Thompson, A., & Johnson, L. (2003). The effects of pre-session attention on problem behavior maintained by different reinforcer. *Journal of Applied Behavior Analysis*, 36, 297–307.
- McGill, P. (1999). Establishing operations: Implications for the assessment, treatment, and prevention of problem behavior. *Journal of Applied Behavior Analysis*, 32, 393–418.
- McGuinnis, M. A., Houchins-Juarez, N., McDaniel, J. L., & Kennedy, C. G. (2010). Abolishing and establishing operation analysis of social attention as positive reinforcement for problem behavior. *Journal of Applied Behavior Analysis*, 43, 119–123.
- Michael, J. (1982). Distinguishing between discriminative and motivational functions of stimuli. *Journal of the Experimental Analysis of Behavior*, 37, 149–155.
- Michael, J. (1993). Establishing operations. *The Behavior Analyst*, 16, 191–206.
- Michael, J. (2000). Implications and refinements of the establishing operation concept. *Journal of Applied Behavior Analysis*, 33, 401–410.
- Michael, J. (2007). Motivating operations. In J. O. Cooper, T. E. Heron, & W. L. Heward (Eds.), *Applied behavior analysis* (2nd ed., pp. 374–391). Upper Saddle River: Merrill Prentice Hall.
- Neely, L., Rispoli, M., Camargo, S., Davis, H., & Boles, M. (2013). The effect of instructional use of an iPad [R] on challenging behavior and academic engagement for two students with autism. *Research in Autism Spectrum Disorders*, 7, 509–516.
- Roxburgh, C., & Carbone, V. J. (2012). The effects of varying teacher presentation rates on responding during discrete trial training for two children with autism. *Behavior Modification*, 37, 1–26.
- Skinner, B. F. (1957). *Verbal behavior*. Englewood Cliffs: Prentice Hall.
- Smith, R. G., & Iwata, B. A. (1997). Antecedent influences on behavior disorders. *Journal of Applied Behavior Analysis*, 30, 343–375.
- Sundberg, M. L. (1993). The application of establishing operations. *The Behavior Analyst*, 16, 211–214.
- Sundberg, M. L., Loeb, M., Hale, L., & Eigenheer, P. (2002). Contriving establishing operations to teach mands for information. *The Analysis of Verbal Behavior*, 18, 14–28.
- Sweeney-Kerwin, E. J., Carbone, V. J., O'Brien, L., Zecchin, G., & Janecky, M. N. (2007). Transferring control of the mand to the motivating operation in children with autism. *The Analysis of Verbal Behavior*, 23, 89–102.
- Taylor, B. A., Hoch, H., Potter, B., Rodriguez, A., Spinnato, D., & Kalaigian, M. (2005). Manipulating establishing operations to promote initiations toward peers in children with autism. *Research in Developmental Disabilities*, 26, 385–392.

Motivation

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Definition

An important body of research relates to interventions that improve motivation in children with autism. This literature has been particularly relevant, as individuals with autism often appear unmotivated to engage in social and learning interactions. It has been hypothesized that a lack of motivation to engage in social and learning interactions may be caused by external variables (see below). Therefore, environmental manipulations should be effective in changing the response-reinforcer relationships that led to the development of these motivational deficits. Along those lines, a number of studies have focused on identifying specific variables that effectively improve motivation in children with autism. To start, child-driven approaches, such as providing the child with the choice of stimulus materials, activities, and conversational topics, have been shown to improve responding and attention (Koegel et al. 1987). This has also been referred to in the literature as “following the child’s lead,” and its effectiveness has been well documented in regard to improving a range of behaviors such as communication, academics (Koegel et al. 2010), and socialization (Koegel et al. 1987). Such approaches have been compared and contrasted with previous empirically

based approaches that focus on an adult-driven curriculum without any input or consideration of the child with autism's changing reinforcer hierarchy and have been shown to be more effective.

Another variable that improves motivation to respond is the interspersal of new (acquisition) tasks that the child has not yet mastered with previously mastered (maintenance) tasks (Dunlap 1984; Koegel and Koegel 1986). Rather than repeatedly presenting new target tasks that are likely to be difficult, interspersing maintenance tasks with acquisition tasks results in increased correct responding in addition to improved overall responsiveness. Similarly, when tasks are varied frequently, rather than being taught in a drill-like context, motivation improves (Dunlap and Koegel 1980). Another body of research relates to the manipulation of consequences for child responses. As opposed to using a strict shaping paradigm that only rewards responses that are as good or better than the previous response, child motivation can be improved by rewarding child's attempts to respond correctly that are free of disruptive or repetitive behaviors (Koegel and Egel 1979; Koegel et al. 1988). Related to this, providing direct and natural rewards, which are inherently linked to the child's response, results in improved motivation in terms of more rapid acquisition of the target behaviors (Koegel and Williams 1980; Williams et al. 1981). That is, rather than providing an arbitrary unrelated reward (e.g., food item or token) contingent upon a correct response, the child is provided with rewards that are directly related to their behaviors. For example, a food may be given to a child learning first words contingent upon labeling the food item so that it is directly related to the child's response. The aforementioned procedures can be used in combination with one another and have been documented to improve motivation as measured by decreased levels of disruptive behavior; improved performance in academic, language, and social domains; and improved child affect (Koegel et al. 1987; Koegel and Koegel 2006). Another variable that has been shown to affect child motivation for some (but not all) children is clinician affect. For example, research suggests that some children respond more

favorably when the clinician shows specific types of affect while providing reinforcers, such as either strong positive affect or low affect depending on the child. As a whole, these studies suggest that motivation appears to be measurable and treatable. At this point, the most effective programs use a combination of motivational procedures, applied simultaneously, to produce large changes in the behavior of children with autism. This body of literature has led researchers to conceptualize the construct of motivation as "pivotal" such that positive changes in a number of untreated symptoms/behaviors occur when a child with autism is motivated to respond (Koegel and Koegel 2006). Thus, the literature on pivotal response treatment (PRT) uses motivational procedures as a core underlying principle of the intervention, and motivational components have effectively been incorporated into interventions for improving articulation (Koegel et al. 1998), first words (Koegel et al. 1987), academic engagement (Koegel et al. 2010), and social conversation (Koegel et al. 1987) in children with autism. Additionally, studies have shown that collateral improvements in disruptive behavior occur when such motivational variables are employed without needing to target the disruptive behavior directly. While the general concept of motivation can be conceptualized by feelings of an individual, such as the desire or incentive to engage in activities, motivation also can be defined by observable and measureable behaviors. Specifically, motivation has been measured by task engagement, responsiveness, decreases in response latency, initiations, and affect, as well as an absence of avoidance behavior (Koegel et al. 2001). During the intervention sessions when motivational procedures are incorporated into the teaching, the children show an increase in the number of responses and decrease in response latency, initiate new related behaviors, and demonstrate positive affect. Task engagement and responsiveness can be monitored by the number or rate of responses the child emits. Latency can be measured by assessing the amount of time that passes before the child begins to engage in a learning task or activity. Initiations are important in that if the children are motivated to engage in

the tasks, they are likely to spontaneously engage in the task, sustain participation, and initiate future activities. This also relates to generalization, as motivated children may spontaneously demonstrate the targeted responses across settings, behaviors, and people. Changes in affect can also be seen in motivated children. Affect is often rated by Likert scales in general categories of positive, neutral, and negative affect and has been defined as interest (e.g., child is alert and involved in the activity), happiness (e.g., the child smiles, laughs, and seems to be enjoying self), and enthusiasm (Baker et al. 1998; Koegel and Egel 1979; Koegel et al. 2009). In addition to child affect, motivation can be measured by lowered levels of disruptive behaviors (Dunlap and Koegel 1980; Koegel et al. 1992). Multiple measures showing improved latency, affect, happiness, enthusiasm, and levels of disruptive behavior have been used to measure motivation with high degrees of reliability (Dunlap and Koegel 1980).

Historical Background

The interest in the construct of motivation, as it relates to autism spectrum disorder (ASD), came about as a result of the apparent lethargy and disinterest individuals with autism may demonstrate in regard to social conversation, learning, and other activities (Lovaas 1977). This led researchers to hypothesize theories for this lack of motivation. A possible theoretical explanation is related to the concept of “learned helplessness.” Specifically, it has been hypothesized that during exposure to an uncontrollable outcome, a lack of contingency between the behavior and the outcome is learned (Seligman and Altenor 1980). Simply put, if an individual receives noncontingent rewards or punishers, the individual will not initiate behaviors and will appear lethargic and inactive. Early animal studies that postulated the learned helplessness theory were carefully controlled and showed the effect of uncontrollable events causing a disruption in behavior. Specifically, the once responsive animals became unresponsive and lethargic. Subsequently, studies replicated this phenomenon using human

subjects in controlled settings. They showed that if individuals’ responses were not reinforced, they too simply stopped responding (Gatchel et al. 1975). Learned helplessness has also been hypothesized to occur in other human conditions, such as stress and depression (Miller and Seligman 1975; Price et al. 1978). Some studies have also shown that physiological changes, such as norepinephrine depletion, weight loss, and ulcers occur at a higher rate when consequences are noncontingent and outcomes are uncontrollable (Abramson et al. 1978; Seligman and Weiss 1980). Data also suggest that this theory may be highly relevant for autism as well (Koegel and Egel 1979; Koegel and Koegel 2006).

The theory of motivation is an important concept in regard to intervention for autism, as it supports the notion that some, or all, of the disability may relate to environmental variables. That is, the state of an apparent lack of motivation is hypothesized to occur when the individual’s responses are uncontrollable, or “learned helplessness.” In regard to children with autism, reduced social responding that begins very early in life due to central nervous system dysfunction may result in the children experiencing high degrees of noncontingent consequences. This may create a situation wherein the child learns that responding and reinforcement are independent. This weakening of the response-reinforcer relationship may cause a decrease in social responding and lower levels of motivation in children with autism.

Current Knowledge

The lack of responsiveness and long response latencies demonstrated by children with autism as well as an apparent lack of motivation and curiosity have been discussed since the 1960s when principles of applied behavior analysis were beginning to show promise with children with autism. Within the broad framework of environmentally manipulated behaviors, researchers discussed the role of reinforcers in this apparent lack of motivation. They proposed that the potency or availability of reinforcers may not be

adequate when a child with autism engages in difficult tasks (Koegel and Egel 1979), thereby creating a lack of motivation. Koegel and Egel (1979) were the first authors to present the possibility of learned helplessness and the importance of motivation to address the problem. Following that publication, a number of studies addressed individual areas of intervention that appeared to directly improve motivation. In 1987, Koegel, O'Dell, and Koegel published work using an intervention package, containing a large number of motivational variables (discussed above), was especially effective.

Future Directions

Although the literature describes specific procedures for improving motivation, there continues to be a need for future research relating to the measurement of motivation, the refinement of existing procedures that improve motivation, and additional intervention procedures for improving motivation.

References and Reading

- Abramson, L. Y., Seligman, M. E., & Teasdale, J. D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology, 87*(1), 49–74.
- Baker, M., Koegel, R. L., & Koegel, L. K. (1998). Increasing the social behavior of young children with autism using their obsessive behaviors. *The Journal of the Association for Persons With Severe Handicaps, 23*, 300–308.
- Dunlap, G. (1984). The influence of task variation and maintenance tasks on the learning and affect of autistic children. *Journal of Experimental Child Psychology, 37*(1), 41–64.
- Dunlap, G., & Koegel, R. L. (1980). Motivating autistic children through stimulus variation. *Journal of Applied Behavior Analysis, 13*(4), 619–627.
- Gatchel, R. J., Paulus, P. B., & Maples, C. W. (1975). Learned helplessness and self-reported affect. *Journal of Abnormal Psychology, 84*(6), 732–734.
- Koegel, R. L., & Egel, A. L. (1979). Motivating autistic children. *Journal of Abnormal Psychology, 88*(4), 418–426.
- Koegel, L. K., & Koegel, R. L. (1986). The effects of interspersed maintenance tasks on academic performance in a severe childhood stroke victim. *Journal of Applied Behavior Analysis, 19*(4), 425–430.
- Koegel, R. L., & Koegel, L. K. (2006). *Pivotal response treatments for autism: Communication, social, & academic development*. Baltimore: Paul H Brookes Publishing.
- Koegel, R. L., & Williams, J. A. (1980). Direct versus indirect response-reinforcer relationships in teaching autistic children. *Journal of Abnormal Child Psychology, 8*(4), 537–547.
- Koegel, R. L., Dyer, K., & Koegel, L. K. (1987). The influence of child-preferred activities on autistic children's social behavior. *Journal of Applied Behavior Analysis, 20*(3), 243–252.
- Koegel, R. L., O'Dell, M., & Dunlap, G. (1988). Producing speech use in nonverbal autistic children by reinforcing attempts. *Journal of Autism and Developmental Disorders, 18*(4), 525–538.
- Koegel, R. L., Koegel, L. K., & Surratt, A. (1992). Language intervention and disruptive behavior in pre-school children with autism. *Journal of Autism and Developmental Disorders, 22*(2), 141–153.
- Koegel, R. L., Camarata, S., Koegel, L. K., Ben-Tall, A., & Smith, A. E. (1998). Increasing speech intelligibility in children with autism. *Journal of Autism and Developmental Disorders, 28*(3), 241–251.
- Koegel, R. L., Koegel, L. K., & McNeerney, E. K. (2001). Pivotal areas in intervention for autism. *Journal of Clinical Child Psychology, 30*(1), 19–32.
- Koegel, R. L., Vernon, T. W., & Koegel, L. K. (2009). Improving social initiations in young children with autism using reinforcers with embedded social interactions. *Journal of Autism and Developmental Disorders, 39*(9), 1240–1251.
- Koegel, L. K., Singh, A. K., & Koegel, R. L. (2010). Improving motivation for academics in children with autism. *Journal of Autism and Developmental Disorders, 40*(9), 1057–1066.
- Lovaas, O. I. (1977). *The autistic child: Language development through behavior modification*. Oxford: Irvington.
- Miller, W. R., & Seligman, M. E. (1975). Depression and learned helplessness in man. *Journal of Abnormal Psychology, 84*(3), 228–238.
- Price, K. P., Tryon, W. W., & Raps, C. S. (1978). Learned helplessness and depression in a clinical population: A test of two behavioral hypotheses. *Journal of Abnormal Psychology, 87*(1), 113–121.
- Seligman, M. E. P., & Altemor, A. (1980). Part II: Learned helplessness. *Behaviour Research and Therapy, 18*(5), 462–473.
- Seligman, M. E., & Weiss, J. M. (1980). Coping behavior: Learned helplessness, physiological change and learned inactivity. *Behaviour Research and Therapy, 18*(5), 459–512.
- Williams, J. A., Koegel, R. L., & Egel, A. L. (1981). Response-reinforcer relationships and improved learning in autistic children. *Journal of Applied Behavior Analysis, 14*, 53–60.

Motivation Assessment Scale

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Synonyms

MAS

Description

The Motivation Assessment Scale (MAS) is a rating scale that assesses functions of problem behavior in individuals with developmental disabilities through informant responses. It includes 16 questions and is comprised of four subscales that each represents a possible function of the behavior: attention, escape, sensory, and tangible. Each question has six response options (0 = never, 1 = almost never, 2 = seldom, 3 = half the time, 4 = usually, 5 = almost always, and 6 = always). Scores are calculated by summing the item ratings within a particular subscale/function and calculating the mean rating for that subscale. High scores for one or more of the subscales suggest that those functions may be maintaining the individual's problem behavior (Durand and Crimmins 1988), although the authors of the instrument do not specify what constitutes a high score. An example item on the MAS is "*Does this behavior occur when you are talking to other persons in the room?*" (Durand & Crimmins, p. 102). The MAS is considered an indirect assessment method because it is removed in time and place from the behaviors being measured (Floyd et al. 2005).

Historical Background

The MAS was originally developed to assess the functions of self-injurious behavior, in particular (Durand and Crimmins 1988), within a framework of applied behavior analysis. It was

designed to be a more efficient alternative to direct functional behavior analysis methods (Durand and Crimmins 1988). Based on previous literature identifying common functions of self-injurious behavior (i.e., social attention, tangible consequences, escape from unpleasant situations, and sensory consequences), Durand and Crimmins (1988) created four subscales, each representing a specific function, that comprise the MAS. The items were generated following interviews with teachers, clinicians, and parents of children with developmental disabilities.

Psychometric Data

In their original study based on 50 children with developmental disabilities who exhibited frequent self-injurious behavior, Durand and Crimmins (1988) reported interrater reliability coefficients ranging from .66 to .92 for the individual items and .80 to .95 for the raw mean subscale scores. Test-retest reliability, over a 30-day period, was calculated and the coefficients ranged from .89 to .98 for the individual items and .92 to .98 for the mean subscale raw scores. The authors also found that teachers' ratings on the MAS were highly predictive of student's behavior in an analogue experimental condition (Durand & Crimmins).

However, the psychometric properties of the MAS have since been questioned by other researchers (e.g., Paclawskyj et al. 2001), as subsequent studies examining the validity and reliability of the measure have produced inconsistent results. It has also been argued that research on the MAS and other indirect functional assessment measures has not carefully examined validity evidence based on scale content and informant response processes, evidence of user satisfaction with the measure, or the treatment utility of the scale (Floyd et al. 2005).

Some factor analytic research has provided support for the intended four-factor structure of the MAS (i.e., sensory, escape, attention, and tangible), although the factor structure was somewhat different for a sample with lower frequency problem behaviors (Singh et al. 1993). Other researchers have not found support for the four-

factor model, specifically the sensory function subscale (Kearney et al. 2006). Joosten and Bundy (2008) also failed to find support for the construct validity of the MAS in the form of a four-factor structure or as a unidimensional measure of motivating factors for stereotyped behavior. In addition, Duker and Sigafoos (1998) results did not fully support the factor structure of the MAS, and their results suggest that the type of problem behavior may be differentially related to reliability and factor structure.

Research examining the internal consistency of the MAS has also produced inconsistent results. Cronbach's alpha coefficients for the subscales ranged from .68 to .86 in a study by Sreat and Connelly (1996) and .68 to .87 in a study by Duker and Sigafoos (1998). However, Shogren and Rojahn (2003) reported values ranging from .80 to .96.

Subsequent research on the interrater reliability of the rating scale has produced lower values than those reported in the original Durand and Crimmins (1988) study. Shogren and Rojahn (2003) found interrater reliability coefficients for the four subscales ranging from .35 to .73, and Sreat and Connelly (1996) reported values ranging from .31 to .57. Another study found that interrater agreement depended upon the type of problem behavior (Duker and Sigafoos 1998). For example, raters were less likely to agree on the functions of destructive behaviors (e.g., aggressive behavior) as compared to maladaptive (e.g., self-injurious behavior) and disruptive (e.g., screaming) behaviors (Duker & Sigafoos).

Research examining other types of reliability evidence for the MAS has also failed to provide clear evidence that the instrument is psychometrically sound. For example, Shogren and Rojahn (2003) reported test-retest reliability coefficients ranging from .71 to .89 for the four subscales, which are lower values than those reported originally by Durand and Crimmins (1988). Sreat and Connelly (1996) also found evidence of poor reliability in the difference scores between subscales, which are used to make clinical decisions regarding the function of the problematic target behavior.

Clinical Uses

The MAS has been used clinically to identify possible factors that are maintaining an individual's problem behavior. The identified function can then be used to develop an intervention program to reduce the problem behavior and to select reinforcers for appropriate behavior. Some have argued that the MAS should be used in conjunction with direct observation methods because of its psychometric limitations (e.g., Duker and Sigafoos 1998; Shogren and Rojahn 2003; Sreat and Connelly 1996).

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavioral Assessment](#)
- [Functional Analysis](#)
- [Functional Behavior Assessment](#)

References and Reading

- Duker, P. C., & Sigafoos, J. (1998). The motivation assessment scale: Reliability and construct validity across three topographies of behavior. *Research in Developmental Disabilities, 19*, 131–141.
- Durand, V. M., & Crimmins, D. B. (1988). Identifying the variables maintaining self-injurious behavior. *Journal of Autism and Developmental Disorders, 18*, 99–117.
- Floyd, R. G., Phaneuf, R. L., & Wilczynski, S. M. (2005). Measurement properties of indirect assessment methods for functional behavioral assessment: A review of research. *School Psychology Review, 34*, 58–73.
- Joosten, A. V., & Bundy, A. C. (2008). The motivation of stereotypic and repetitive behavior: Examination of construct validity of the motivation assessment scale. *Journal of Autism and Developmental Disorders, 38*, 1341–1348.
- Kearney, C. A., Cook, L. C., Chapman, G., & Bensaheb, A. (2006). Exploratory and confirmatory factor analyses of the motivation assessment scale and resident choice assessment scale. *Journal of Developmental and Physical Disabilities, 18*, 1–11.
- Paclawskyj, T. R., Matson, J. L., Rush, K. S., Smalls, Y., & Vollmer, T. R. (2001). Assessment of the convergent validity of the questions about behavioral function with analogue functional analysis and the motivation assessment scale. *Journal of Intellectual Disability Research, 45*, 484–494.

- Shogren, K. A., & Rojahn, J. (2003). Convergent reliability and validity of the questions about behavioral function and the motivation assessment scale: A replication study. *Journal of Developmental and Physical Disabilities, 15*, 367–375.
- Singh, N. N., Donatelli, S. S., Best, A., Williams, D. E., Barrera, F. J., Lenz, M. W., et al. (1993). Factor structure of the motivation assessment scale. *Journal of Intellectual Disability Research, 37*, 65–74.
- Spreat, S., & Connelly, L. (1996). Reliability analysis of the motivation assessment scale. *American Journal on Mental Retardation, 100*, 528–532.

Motor Control

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Synonyms

[Cerebellum](#); [Motor cortex](#)

Definition

Motor control refers to the coordination of information exchanged between the neocortex in the brain and the muscular and somatosensory systems to bring about controlled, or voluntary, movements. Controlled movements are planned, organized, and initiated in the prefrontal, premotor, and primary motor cortices. Instructions for the movement are then sent through the spinal cord to the appropriate muscles to execute the movement. The somatosensory system provides feedback about the movement, and the basal ganglia, brain stem, and cerebellum fine-tune the movement by adjusting force and correcting for movement errors.

See Also

- ▶ [Cerebellum](#)
- ▶ [Motor Cortex](#)

References and Reading

- Kolb, B., & Whishaw, I. Q. (2009). *An introduction to brain and behavior* (3rd ed.). New York: Worth.

Motor Cortex

- ▶ [Motor Control](#)
- ▶ [Precentral Gyrus](#)

Motor Development Assessment

- ▶ [Psychomotor Development Index](#)

Motor Planning

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Definition

Motor planning can be broadly defined as the capacity to plan the necessary steps to achieve purposeful movements. It is often considered under the larger concept of praxis, which comprises the conceptualization, organization, and execution of an action sequence. Although the terms “motor planning” and “praxis” are sometimes used interchangeably, motor planning may be more accurately thought of as one element of praxis, distinct from both the idea behind the movement and its actual implementation. In addition to being dependent in part on neural motor systems, motor planning may be affected by the multimodal integration of sensory processes involved in proprioception, vestibular functioning, and vision, and by capabilities for preemptively formulating a mental sequence of actions.

The clinical assessment of motor planning typically occurs as part of a broader evaluation of motor functioning. Few clinical tests specifically measure motor planning; however, a number of standardized instruments can provide relevant information. These include batteries that assess a range of motor abilities, such as fine and gross motor skills, limb and verbal praxis, and sensorimotor functioning. In addition, the assessment of adaptive skills and motor performance during everyday activities can provide some indication of motor planning ability. A thorough developmental history is also usually conducted for children with motor planning or praxis difficulties, including the documentation of major motor milestones and early motor behaviors.

Disorders related to motor planning and praxis include apraxia and dyspraxia. Apraxia is typically thought of as an acquired neurological condition in which skilled movement is impaired in the presence of intact basic sensorimotor and attentional functioning. The term “dyspraxia” is often used to describe similar symptoms that are present early in development and have a less concrete etiology. Deficits in praxis in autism spectrum disorder (ASD) generally fall under the category of dyspraxia or developmental dyspraxia. Disorders of praxis can affect multiple modalities, including fine motor functioning, gross motor functioning, oculomotor control, orofacial movement, and speech production.

Historical Background

General impairments in motor functioning have been documented in individuals with ASD since both Kanner’s and Asperger’s initial descriptions of the conditions. Since then, basic motor deficits have been consistently found in ASD, with many individuals displaying abnormalities in gait, postural control, coordination, balance, tone, and/or strength. Furthermore, there is evidence that aspects of motor dysfunction are often identifiable early in development, present throughout the lifespan, and pervasive across levels of cognitive functioning.

The study of praxis has been an area of increasing interest within the larger domain of motor

functioning in ASD. One focus of earlier work related to praxis was the assessment of clumsiness, or motor incoordination, as a possible symptom to differentiate subtypes within the autism spectrum. Historically, clumsiness was often considered a particular characteristic of Asperger syndrome, whereas individuals with classic autism were thought to have preserved motor function. Most evidence now suggests that motor clumsiness does not discriminate among subtypes; individuals with both Asperger syndrome and autism show similar degrees of impairment. Although clumsiness and dyspraxia are sometimes used synonymously, it is important to recognize that clumsiness is a clinical description that does not address the specific nature of an underlying motor impairment. Current research has taken a more systematic and componential approach to studying dyspraxia, with the goal of identifying the precise roots of the movement disturbances seen in individuals with ASD.

Current Knowledge

Dyspraxia in ASD

The growing body of literature focused on dyspraxia in ASD has, on the whole, confirmed that both children and adults with ASD display deficits on overall measures of praxis. In fact, difficulties with skilled limb movements have become one of the more reliable motor findings in the field. Impairments have been found in the ability to produce actions in response to command, imitate actions, and carry out actions involving tools. Moreover, these impairments are seen in transitive movements, or those involving objects, as well as intransitive movements, which do not include objects and are often more symbolic. Although basic motor skills are associated with praxis ability, individuals with ASD consistently appear to display deficits in praxis beyond what can be accounted for by general motor dysfunction. This suggests that praxis is a specific area of weakness for individuals with ASD within the larger motor domain. In addition, although correlations between praxis and both age and cognitive ability have been reported, the

available data largely indicate that symptoms of dyspraxia in ASD remain even after controlling for these factors. Of particular interest, praxis impairment has been shown to predict the severity of autistic symptoms, whereas correlations between basic motor skill and ASD symptomatology have not been found.

Motor Planning in ASD

Research has also sought to examine the motor planning component of praxis more directly. One approach has utilized paradigms in which potential impairments at the planning or preparation stage of a goal-directed motor act can be assessed separately from impairments at the execution stage. An example is a task in which participants are required to generate hand movements toward alternating targets. The planning stage is defined as the time between the appearance of the target and the point at which the hand begins to move; the execution stage is defined as the time in which the hand is in motion. Research employing these types of tasks has found that the planning stage of movement is generally atypical in individuals with ASD, while the execution stage is similar to typically developing controls.

Motor planning abilities in individuals with ASD have also been investigated using a reach, grasp, and place paradigm, in which participants are instructed to reach for an object, pick it up, and place it in a specified location or orientation. In one study in which children were required to reach, grasp, and place a rod in a certain position, typically developing children consistently chose initial movements that allowed for a comfortable end state. In contrast, children with ASD were more likely to favor an easier initial grip, even when this forced an uncomfortable final hand posture. In a similar study, children were asked to pick up an object and place it in either a small or large container. Typically developing children took longer during both the reach/grasp and place stages on the trials with the smaller compared to the larger target container. This suggests global planning of actions, such that when the final step of a motor act requires more precision, the execution of earlier steps is also affected. In contrast, for children with ASD, only the place

stage was longer, signifying that although they were affected by the increased precision required at the end, they were less likely to plan for this in their initial actions.

Overall, the performance of individuals with ASD on reach, grasp, and place tasks indicates that they are less likely than typically developing individuals to plan goal-directed actions holistically. Instead, they seem to execute the necessary steps for a motor act independently, such that knowledge of the end goal does not influence the initial movements in an action sequence. However, there is some evidence to suggest that the motor planning behavior of children with ASD may not differ from their peers when tasks have fewer higher order cognitive demands. For example, on a more constrained grip selection paradigm in which children reached for and grasped a handle and turned it in a specified direction, differences in performance were not found between age-matched groups.

Related research has found that some individuals with ASD may have difficulty with coordinating the stages of a reach-to-grasp movement, failing to temporally couple the velocity of the reach stage with the timing of their hand opening during the grasp stage. One study found that lower functioning children with ASD did not seem to synchronize the timing of their reach and grasp based on target location and size in the same way as controls. Instead, they performed the movement in two distinct stages, completing the reaching movement first, and then opening their hand to grip the object. However, high-functioning children with ASD performed similarly to typically developing children, with the timing of the reach and handgrip movements well synchronized.

A final area of research on motor planning in ASD has explored how the advanced presentation of information about a target affects goal-oriented movements. One hypothesis is that goal-directed actions are planned in a predictable sequence, where the appropriate hand is chosen first, followed by the direction of the movement, then by the amount of movement necessary to achieve the target. This model has been tested through the use of a precue technique, in which prior information about hand, direction, and/or movement

extent is given to the participant. Results indicate that young adults with and without ASD display a similar, predictable pattern on this task, suggesting that they can effectively use advanced information to plan movements. Individuals in both groups tend to derive the most benefit from information pertaining to which hand to use, followed by information about direction, and finally by information about the amount of movement. Nevertheless, the reaction times of individuals with ASD were slower and more variable than those of their peers.

Clinical Significance

Motor deficits are not a diagnostic characteristic of ASD, yet the importance of studying how various aspects of motor dysfunction may contribute to the condition has been increasingly recognized. Without a doubt, impairments in planning purposeful movement can have deleterious effects on many aspects of daily functioning, including basic tasks like feeding and dressing. In addition, the impact of difficulties with motor planning on social and communicative functioning is a topic of growing interest. Some theories posit that the motor system plays a foundational role in certain interpersonal skills. The ability to effectively plan and produce movements within an appropriate time frame may be crucial for reciprocal social interaction. In addition, praxis ability has been empirically associated with imitation. Difficulties with imitation have been widely documented in ASD across ages and functioning levels: individuals with ASD have been shown to imitate less than their peers, display poorer approximations of imitated movements, and consistently make specific types of errors during imitation tasks. Furthermore, abnormalities have been found for the imitation of nonmeaningful movements, as well as movements with more social or symbolic significance, suggesting that a fundamental problem with skilled motor functioning may be involved in imitation impairments in ASD.

Future Directions

The extant research has established motor planning as an area worthy of further investigation in ASD.

An important direction for future research will be to build on existing work exploring components of motor acts to identify the specific aspects of goal-directed movement that are affected in ASD, both globally and at different points in development. There is also a need for further research on how functioning in other domains may impact the components of movement. Although basic motor functioning, general intellectual ability, attentional skills, motor learning, and sensory abilities have all been implicated in the organization of movement, the relationship between these various skill domains and motor planning in individuals with ASD is still not well understood.

Recent work has specifically highlighted the need to distinguish between motor planning and higher order planning, the latter of which is thought to rely on executive functioning skills. Motor planning is distinctly dependent on the ability to implement learned movements, whereas executive planning requires abstraction and mental sequencing of goal-oriented decisions. Both motor and executive planning are often necessary to effectively prepare and execute actions, making it difficult to pin down the precise nature of movement planning difficulties in ASD. One viewpoint has been that individuals with ASD perform poorly on motor planning tasks because of executive functioning impairments, and that they would demonstrate intact performance on tasks that do not require executive skills. However, at least one study has shown that individuals with ASD perform poorly across a range of motor planning tasks regardless of the level of executive demand, suggesting that motor planning may be impaired even in situations that do not require higher order abilities.

Another direction for future research is continued exploration of the neurobiological underpinnings of motor planning and related functions in ASD. Although research in ASD has implicated abnormalities in a number of regions important in motor functioning, less work has specifically addressed functional atypicalities during motor tasks. The neurobiology of the motor system in typically developing individuals is well understood. Thus, studying the neural bases of motor dysfunction in ASD may offer a more straightforward avenue for identifying structural and

connectivity abnormalities in this disorder, potentially leading to a better understanding of the neural bases of other affected domains. In addition, functional neuroimaging studies can provide further information on how individuals with ASD approach motor planning tasks, helping to answer questions about the degree to which executive and other skills play a role in motor planning in this condition.

Finally, further research is needed to better understand if and how motor planning difficulties might influence the pathogenesis of core symptoms in ASD, including social and communication impairments. The answers to these questions will benefit from more research on the developmental timeframe of motor planning in ASD, and how this interfaces with other aspects of development.

See Also

- [Apraxia](#)
- [Developmental Apraxia](#)
- [Developmental Dyspraxia](#)
- [Dyspraxia](#)
- [Executive Function \(EF\)](#)
- [Imitation](#)
- [Praxis](#)

References and Reading

- Baranek, G. T., Parham, L. D., & Bodfish, J. W. (2005). Sensory and motor features in autism: Assessment and intervention. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 831–857). Hoboken: Wiley.
- Dewey, D., & Bottos, S. (2006). The effect of motor disorders on imitation in children. In S. J. Rogers & J. H. G. Williams (Eds.), *Imitation and the social mind: Autism and typical development* (pp. 399–430). New York: Guilford Press.
- Dewey, D., Cantell, M., & Crawford, S. G. (2007). Motor and gestural performance in children with autism spectrum disorders, developmental coordination disorder and/or attention deficit hyperactivity disorder. *Journal of the International Neuropsychological Society*, 13, 246–256.
- Dowell, L. R., Mahone, E. M., & Mostofsky, S. H. (2009). Associations of postural knowledge and basic motor skill with dyspraxia in autism: Implication for abnormalities in distributed connectivity and motor learning. *Neuropsychology*, 23(5), 563–570.
- Dziuk, M. A., Gidley Larson, J. C., Apostu, A., Mahone, E. M., Denckla, M. B., & Mostofsky, S. H. (2007). Dyspraxia in autism: Association with motor, social, and communicative deficits. *Developmental Medicine & Child Neurology*, 49, 734–739.
- Fabbri-Destro, M., Cattaneo, L., Boria, S., & Rizzolatti, G. (2009). Planning actions in autism. *Experimental Brain Research*, 192, 521–525.
- Gidley Larson, J. C., & Mostofsky, S. H. (2006). Motor deficits in autism. In R. Tuchman & I. Rapin (Eds.), *Autism: A neurological disorder of early brain development* (pp. 231–247). London: Mac Keith Press.
- Glazebrook, C. M., Elliott, D., & Szatmari, P. (2008). How do individuals with autism plan their movements? *Journal of Autism and Developmental Disorders*, 38, 114–126.
- Hughes, C. (1996). Brief report: Planning problems in autism at the level of motor control. *Journal of Autism and Developmental Disorders*, 26(1), 99–107.
- Leary, M. R., & Hill, D. A. (1996). Moving on: Autism and movement disturbance. *Mental Retardation*, 34, 39–53.
- Mari, M., Marks, D., Marraffa, C., Prior, M., & Castiello, U. (2003). Autism and movement disturbance. In U. Frith & E. Hill (Eds.), *Autism: Mind and brain* (pp. 225–246). New York: Oxford University Press.
- Mostofsky, S. H., Dubey, P., Jerath, V. K., Jansiewicz, E. M., Goldberg, M. C., & Denckla, M. B. (2006). Developmental dyspraxia is not limited to imitation in children with autism spectrum disorders. *Journal of the International Neuropsychological Society*, 12, 314–326.
- Nazarali, N., Glazebrook, C. M., & Elliott, D. (2009). Movement planning and reprogramming in individuals with autism. *Journal of Autism and Developmental Disorders*, 39, 1401–1411.
- Rinehart, N. J., Bradshaw, J. L., Brereton, A. V., & Tonge, B. J. (2001). Movement preparation in high-functioning autism and Asperger disorder: A serial choice reaction time task involving motor reprogramming. *Journal of Autism and Developmental Disorders*, 31(1), 79–88.
- Rinehart, N. J., Bellgrove, M. A., Tonge, B. J., Brereton, A. V., Howells-Rankin, D., & Bradshaw, J. L. (2006). An examination of movement kinematics in young people with high-functioning autism and Asperger's disorder: Further evidence for a motor planning deficit. *Journal of Autism and Developmental Disorders*, 36(6), 757–767.
- Rogers, S. J., & Williams, J. H. G. (2006). Imitation in autism: Findings and controversies. In S. J. Rogers & J. H. G. Williams (Eds.), *Imitation and the social mind: Autism and typical development* (pp. 277–309). New York: Guilford Press.
- Smith, I. M. (2000). Motor functioning in Asperger syndrome. In A. Klin, F. R. Volkmar, & S. S. Sparrow (Eds.), *Asperger syndrome* (pp. 97–124). New York: Guilford Press.
- Smith, I. M. (2004). Motor problems in children with autistic spectrum disorders. In D. Dewey & D. E.

Tupper (Eds.), *Developmental motor disorders: A neuropsychological perspective* (pp. 152–168). New York: Guilford Press.

Smith, I. M., & Bryson, S. E. (1994). Imitation and action in autism: A critical review. *Psychological Bulletin*, 116(2), 259–273.

van Swieten, L. M., Williams, J. H. G., Plumb, M. S., van Bergen, E., Wilson, A. D., Kent, S. W., et al. (2010). A test of motor (not executive) planning in developmental coordination disorder and autism. *Journal of Experimental Psychology: Human Perception and Performance*, 36(2), 493–499.

ICC	Intra-class Correlation Coefficient
MABC	Movement Assessment Battery for Children
MABC-2	Movement Assessment Battery for Children, Second Edition
PDD	Pervasive Developmental Disorders
PDMS-2	Peabody Developmental Motor Scales – Second Edition
SD	Standard Deviation
SDDMF	Specific Developmental Disorder of Motor Function
TOMI	Test of Motor Impairment
TOMI-H	Test of Motor Impairment-Henderson Revision
UK	United Kingdom

Movement Assessment Battery for Children, Second Edition

► [Movement Assessment Battery for Children: Second Edition \(MABC-2\)](#)

Synonyms

[MABC](#); [MABC-2](#); [Movement assessment battery for children, second edition](#)

Movement Assessment Battery for Children: Second Edition (MABC-2)

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Abbreviations

AB	Age bands
ADHD	Attention deficit/hyperactivity disorder
AS	Asperger’s Syndrome (AS)
ASD	Autism Spectrum Disorder
BOTMP	Bruininks-Oseretsky Test of Motor Proficiency
BOT-2	Bruininks-Oseretsky Test of Motor Proficiency, Second Edition
CFA	Confirmatory Factor Analysis
DCD	Developmental Coordination Disorder

Description

The *Movement Assessment Battery for Children, Second Edition* (MABC-2) (Henderson et al. 2007) is a revision of the *Movement Assessment Battery for Children* (MABC) (Henderson and Sugden 1992) and is one of the most widely used assessment tools by occupational therapists, physiotherapists, psychologists, and educational professionals (Barnett and Henderson 1998; Brown and Lalor 2009; Wiart and Darrah 2001). The purpose of the MABC-2 is the identification and description of impairments in children’s motor function. It is composed of two parts: the Performance Test and the Checklist (see Table 1). The Psychometrics Centre at the Cambridge Judge Business School (n.d.) states that the MABC-2 Performance Test or the Checklist “can be used to identify a child with motor difficulties by comparing the child’s score to the normative data. However, neither instrument should be used on its own as a predictive screening instrument” (para 1).

The MABC-2 Performance Test involves children completing a series of fine and gross motor

Movement Assessment Battery for Children: Second Edition (MABC-2), Table 1 Movement Assessment Battery for Children, Second Edition (MABC-2; Henderson et al. 2007) summary of Performance Test and Checklist data

Test section	MABC-2 performance test	MABC-2 checklist
Purpose	The identification and screening of children with delay or impairment of their motor development, provision of appropriate intervention planning, clinical exploration, program evaluation, and as a research tool with children who are believed to be at risk of motor difficulties	The efficient and economical assessment of the movement competence of children and to identify children who are likely to have difficulty with their movement. The MABC-2 checklist takes assessment into the everyday situations in which the child has to function, including the extent to which a child's attitudes and feelings about motor tasks are situation specific or more generalized. Therapists can also obtain parents' or teachers' views on a child's movement in everyday settings
Test age range	3:0–16:11 years across three age bands (AB) <i>AB1</i> 3:0–6:11 years <i>AB2</i> 7:0–10:11 years <i>AB3</i> 11:0–16:11 years	5:0–12:11 years
Who can administer?	Individuals who are certified by a professional organization recognized by Pearson assessment or who have a graduate and/or postgraduate qualification relevant to their profession. This qualification code would include psychologists, speech therapists, physiotherapists, occupational therapists, mental health professionals, health practitioners, and education professionals. No additional specialized training is required; however, familiarity with test items is required. It is envisaged in the future that specialized training modules will be set up to allow a wider range of professionals' access to the MABC-2	Available to psychologists, classroom teachers, special education teachers, physical education specialists, pediatricians, occupational therapists, speech therapists, and physiotherapists. A child's parent or primary caregiver may also contribute to the completion of MABC-2 checklist if requested by a professional
Time to administer	Individual administration usually takes approximately 20–40 min depending on the age of the children; however, 50 min is suggested for setup, testing, and completion of the record form	The MABC-2 checklist can be completed with a group or individual and takes the respondent approximately 10 min to complete
Time to score	10–15 min	10 min
Materials/equipment required	Stopwatch, clipboard, pencil, correct age band record form, test manual, and test kit containing full set of testing materials and manipulatives	Checklist and pencil
Method of administration	Physical demonstration of all items is required to ensure each child assessed fully understands the verbal instructions for each task. Verbal instructions are not scripted, allowing for the assessor to alter their language according to the age of the child and the child's level of comprehension. Important features of the physical demonstration are emphasized verbally to the child. The child is provided a practice phase (that is not scored/rated) and a formal trial (which is scored/rated)	A basic list of specific motor behaviors is outlined for the adult rater to observe and rate the child's performance and competency. A total score is obtained by summing the ratings, and this is then mapped onto a "traffic light" scoring system (outlined below under the "scores provided" heading)

(continued)

Movement Assessment Battery for Children: Second Edition (MABC-2), Table 1 (continued)

Test section	MABC-2 performance test		MABC-2 checklist
Response format	The child's performance on a task can be recorded in 1 of 4 ways		In sections A and B, the rater has four alternative responses for the child's task competence
	1	The child's best result for an individual task *	0 – very well 2 – almost
	2	An "F" is recorded for a failed attempt	1 – just OK 3 – not close
	3	An "R" is recorded for refusal to attempt or complete a task	The summed score of sections A and B represents the total motor score and interpreted with the "traffic light" system outlined below. Section C allows for either a yes or no response to factors that may affect a child's movement and is not meant to be summed. Rather, these factors should be reviewed by the assessor to determine how much the child is prevented from demonstrating their true capability due to the influence of the observed factors
	4	An "I" is recorded if it is inappropriate for a child to attempt a task	
	* if a child completes a task successfully according to the item parameters, the item is scored according to criteria and then converted to a scaled score reported as 1–5, where 5 indicates a poor performance. Some items are scored based on number of correct repetitions, length of time, or accuracy of responses		
Scale construction/ test structure	Eight tasks are administered across three specific components: (a) manual dexterity, (b) aiming and catching, and (c) balance. Adding all eight scores yields a total motor impairment score (maximum 40). Overall scores that are below the 15th percentile are deemed <i>at risk</i> ; those below the 5th percentile indicate a <i>definite movement impairment</i>		The checklist comprises three sections
			<i>Section A:</i> 15 items regarding movement in a static or predictable environment
			<i>Section B:</i> 15 items regarding movement in a dynamic or unpredictable environment
			<i>Section C:</i> 13 items regarding non-motor factors that may affect movement
Standardization sample	The norms of the MABC-2 are derived from a stratified sample of 1172 children (48.3% male, 51.7% female) in the United Kingdom (UK), who were assessed between November 2005 and July 2006. This sample closely approximated the UK 2001 census data. The test sample included 12 UK geographic regions, 16 age levels (e.g., ages 3–16 years), four ethnic groups (e.g., white, 90.2%; black, 2.6%; Asian, 3.9%; and other, 3.2%), and five levels of parental education		656 children of the 1172 sample fell into the age range of the MABC-2 checklist. Of the 656, teachers for 395 children (50.9% male, 49.1% female) completed the MABC-2 checklist. The test sample included 12 UK geographic regions, 8 age levels (e.g., ages 5–12 years), four ethnic groups (e.g., white, 90.4%; black, 2.6%; Asian, 3.5%; and other, 3.5%), and five levels of parental education. This sample closely approximated the UK 2001 census data
Scores provided	Standard scores and percentile ranks are provided. "Total standard scores are calculated and converted into percentiles to determine how a child's motor coordination compares to typically developing children of the same age" (Camden et al. 2013, p. 1). Additional score interpretation according to a "traffic light" system is provided. This system is the same for both the MABC-2 performance test and checklist, allowing for direct comparison if required. Any child whose score falls at or below the 5th percentile is regarded as having a <i>significant movement difficulty</i> (red zone), between the 6th and 15th percentile <i>at risk</i> (amber zone), and above the 16th percentile as unlikely to have a movement difficulty (green zone)		A percentile cut score is provided. The "traffic light" system shows whether a child is in the "green zone" which is within the age-expected normal range, in the "amber zone" indicating a need for monitoring due to minor delay or movement problem, or in the red zone where it is highly likely that child has a serious movement problem

tasks on which they are scored and rated. The Performance Test is designed for use with children aged 3–17 years in one of three age bands (AB) (AB1, 3:0–6:11 years; AB2, 7:0–10:11 years; and AB3, 11:0–16:11 years) and evaluates motor skills under three categories: (1) Manual Dexterity, (2) Aiming and Catching, and (3) Balance.

The MABC-2 Checklist requires an adult who knows the children being assessed well to rate their motor competence on a 30-item scale. Since the MABC-2 is a revision of an existing instrument, it is important for professionals who use the motor skill battery to be familiar with its age range, scoring format, standardization sample, reliability, validity, and clinical utility. Details of the purpose, age range, administration, response format, scale construction, standardization, and scores provided of the MABC-2's Performance Test and Checklist are reported in Table 1.

The MABC (Henderson and Sugden 1992) was normed with children from Canada, the United States, and the United Kingdom and has subsequently been translated into several European languages (including Swedish, Danish, Dutch, Italian, and Finnish) (Livesey et al. 2007) and Chinese (Chow and Henderson 2003). Predominantly used throughout the United Kingdom, Canada, Australia, several European countries (e.g., the Netherlands, Belgium, Italy, Finland, Denmark, Sweden, and Greece), and Asia to assess pediatric motor impairments, it has been shown to correlate positively with other pediatric motor assessments used worldwide (Smits-Engelsman et al. 1998; Missiuna et al. 2006) and is well recognized as one of the most extensively utilized motor impairment assessments in the world (Chow and Henderson 2003; Chow et al. 2006a, 2006b; Croce et al. 2001; Tan et al. 2001; Wiat and Darrah 2001).

The MABC norms have been evaluated in studies completed in other cultural contexts including Sweden (Rosblad and Gard 1998), Japan (Miyahara et al. 1998), the Netherlands (Smits-Engelsman et al. 1998), Hong Kong (Chow et al. 2001; Chow et al. 2006), Taiwan (Chow et al.), Israel (Engel-Yeger et al. 2010),

United States (Van Waelvelde et al. 2008), Greece (Ellinoudis et al. 2008), and Singapore (Wright et al. 1994). Generalizability across European cultures has shown to be satisfactory; however, those studies conducted in Hong Kong, Taiwan, Singapore, and Japan suggested some cultural differences existed and that the MABC norms for those countries may need some adjustment (Livesey et al. 2007).

In addition to the worldwide recognition that the MABC has, the instrument has been utilized in many international studies covering a broad range of diagnostic categories such as developmental coordination disorder (DCD), learning disabilities, and autism spectrum disorder (ASD). Additionally, the MABC has been noted to identify more children with motor impairments more successfully (e.g., greater sensitivity and specificity) than that of the “gold standard” *Bruininks-Oseretsky Test of Motor Proficiency* (BOTMP) (Dewey and Wilson 2001), while Crawford et al. (2001) have found that the MABC appears to be more sensitive and is able to better identify children with additional problems associated with learning or attention.

The authors have revised the MABC to generate the MABC-2, a “reliable, easily administered and valid measure of competence in three broad and carefully selected areas of motor performance” (Henderson et al. 2007, p. 117). The three broad motor skill categories that are assessed are (1) Manual Dexterity, (2) Aiming and Catching, and (3) Balance. Changes undertaken to produce the second edition involved revising existing items and introducing some new items. These changes are outlined in two sections below: *test content changes* and *test structure changes* (see Tables 2, 3, and 4 for details).

The MABC-2 *test content changes* are described under three areas: (1) *materials*, (2) *tasks*, and (3) *instructions*:

1. *Materials*: Brightly colored plastic pieces have been introduced to replace pieces originally made of wood. This change was undertaken to standardize the pieces and eliminate any room for variation between kits as well as

Movement Assessment Battery for Children: Second Edition (MABC-2), Table 2 Changes made to age band 1 (3–6 years) from initial version to second edition

Motor skill task	MABC ^a task age band 1	MABC-2 ^b task age band 1
Manual dexterity 1	Post coins	Post coins
Manual dexterity 2	Threading beads	Threading beads
Manual dexterity 3	Bicycle trail	Drawing trail 1 (shape of visual trail has changed)
Aiming and catching 1	Catching beanbag	Catching beanbag
Aiming and catching 2	Rolling ball between goal posts	Throwing beanbag onto mat (new item)
Balance 1	One-leg balance	One-leg balance
Balance 2	Walking heels raised	Walking heels raised
Balance 3	Jumping over cord	Jumping on mats (new item)

Henderson et al. 2007, p. 118

^aMABC Movement Assessment Battery for Children

^bMABC-2 Movement Assessment Battery for Children, Second Edition

Movement Assessment Battery for Children: Second Edition (MABC-2), Table 3 Changes made to age band 2 (7–10 years) from initial version to second edition

Motor skill task	MABC ^a task age bands 2/3	MABC-2 ^b task age band 2
Manual dexterity 1	Placing pegs/shifting pegs by rows	Placing pegs (new starting position and layout)
Manual dexterity 2	Threading lace/threading nuts on bolt	Threading lace (lacing board is longer)
Manual dexterity 3	Flower visual trail/flower visual trail	Drawing trail 2 (shape of visual trail has changed)
Aiming and catching 1	Two-hand catch/one-hand bounce and catch	Catching with two hands
Aiming and catching 2	Throwing beanbag/throwing beanbag into box	Throwing beanbag onto mat (mat with target now used instead of box)
Balance 1	Stork balance/one-board balance	One-board balance
Balance 2	Heel-to-toe walking/ball balance	Walking heel-to-toe forward
Balance 3	Jumping in squares/hopping in squares	Hopping on mats (mats used for this task)

Henderson et al. 2007, p. 118

^aMABC Movement Assessment Battery for Children

^bMABC-2 Movement Assessment Battery for Children, Second Edition

- taking into account health and safety regulations regarding item pieces when used with children in various settings.
2. **Tasks:** The test has maintained the original structure of the test by retaining eight test items across each age band organized into the three motor skill categories of Manual Dexterity, Aiming and Catching, and Balance. However, individual items have been altered as well as new individual items being introduced. The task changes across the age bands are outlined in Tables 2, 3, and 4.
 3. **Instructions:** In previous research articles, the test instructions of the MABC have been called into question. Although no standardized verbal instructions are included in the second edition, clarification of the administration, scoring of the test, and aspects of tasks to emphasize during demonstration have been provided to minimize the potential for ambiguity. The MABC-2 authors state that this provides flexibility in the mode of presentation and allows the assessor to ensure the examinee understands individual tasks.

Movement Assessment Battery for Children: Second Edition (MABC-2), Table 4 Changes made to age band 3 (11–16 years) from initial version to second edition

Motor skill task	MABC ^a task age band 3	MABC-2 ^b task age band 3
Manual dexterity 1	Turning pegs	Turning pegs
Manual dexterity 2	Cutting out elephant visual trail with scissors	Triangle with nuts and bolts (new item)
Manual dexterity 3	Flower visual trail	Drawing task 3 (shape of visual trail has changed)
Aiming and catching 1	One-hand catch	Catching with one hand
Aiming and catching 2	Throwing ball at wall-mounted target	Throwing ball at wall-mounted target
Balance 1	Two-board balance	Two-board balance
Balance 2	Walking backward	Walking toe-to-heel backward
Balance 3	Jumping and clapping	Zigzag hopping (new item)

Henderson et al. 2007, p. 118
^aMABC Movement Assessment Battery for Children
^bMABC-2 Movement Assessment Battery for Children, Second Edition

The MABC-2 *test structure changes* are described under two areas: (1) *age extension* and (2) *reduction of age bands*:

1. *Age Extension*: The MABC-2 has been extended to encompass the assessment of children aged 3 years 0 months–16 years 11 months. The MABC-2 authors believed that a gap existed for the appropriate motor performance assessment for children aged 3 years. In order to assess children of this age, items were slightly adjusted to ensure the attention of a typical 3-year-old could be maintained as well as being fun, easily understood, and requiring minimal verbal communication. Similarly, as the MABC has been utilized and widely recognized for its use with assessment of children with DCD, two sources of requests for suitable motor performance assessments for children aged between 11 and 16 years have been identified. The first is due to the increase in the recognition of DCD as a clinical diagnosis and the importance of early assessment, identification, and intervention service provision to assist with coordination difficulties encountered so as to maximize children’s developmental and motor skills. Current intervention for DCD occurs predominantly in during the primary school years due to the

effectiveness of early intervention. However, the MABC-2 authors have noted that an increasing proportion of adolescents have not been identified earlier with coordination difficulties or have failed to benefit from intervention.

The second source identified was due to ongoing worldwide research into children who have been born “at risk” of damage to their nervous system (e.g., born prematurely). This group of children exhibit cognitive skills within normal limits and do not meet the diagnostic criteria for cerebral palsy, but do experience severe difficulties with their motor performance which in turn affects their academic progression. Therefore, with the development and revision of the MABC, the MABC-2 can provide improved motor performance measurement instrument for younger and older children. This in turn will enable therapists and researchers to develop new intervention techniques, allow for more accurate and sound intervention, and provide a greater understanding of motor performance problems and their consequences over time.

2. *Reduction of Age Bands*: Previously the MABC had 4 ABs; however, pilot work by the test authors provided evidence that existing items could be revised and/or adapted across

the extended age bands. Thus, AB1 and the previous AB4 had an age range of 4 years, and so AB2 and AB3 were collapsed in to one age band. Additionally, and perhaps more importantly, the test authors were aware of problems associated with children changing between age bands in the MABC during the course of an intervention program. Although correlations between similar items across the age bands were high, the authors recognized that they were not perfect. Hence, the final age bands for the MABC-2 are AB1: 3–7 years, AB2: 7–11 years, and AB3: 11–17 years.

The Qualitative Observations section remains a part of the MABC-2 test and allows the testing clinician to supplement the formal test results gathered from administering the Performance Test to a child. A chapter of the manual provides some assistance regarding the process of observation and how to utilize the Qualitative Observations section of the test in conjunction with the formal scores as well as taking into account both motor and non-motor factors that can affect movement and a child's motor performance.

Historical Background

The MABC-2 is a composite of two complementary assessments: the Performance Test and the Checklist. The performance-based portion of the MABC-2 was developed from the *Test of Motor Impairment* (TOMI) (Stott et al. 1972). Development of the TOMI began in 1966 with a primary focus of identifying impaired or nonstandard motor skill performance. Since the TOMI provided little overall motor ability information about the children who were assessed with it, the TOMI was revised in 1984 (see Table 5). This revision, known as the *Test of Motor Impairment-Henderson Revision* (TOMI-H) (Stott et al. 1984), decreased the number of items that a child was required to complete, added a behavioral checklist, and included room for recording qualitative observations related to children's motor skill performance.

Movement Assessment Battery for Children: Second Edition (MABC-2), Table 5 Evolution of the versions of the *Movement Assessment Battery for Children – Second Edition* (MABC-2)

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1. *Test of Motor Impairment* (TOMI) (Stott et al. 1972)
 2. *Test of Motor Impairment – Henderson Revision* (TOMI-H) (Stott et al. 1984)
 3. *Movement Assessment Battery for Children* (MABC) (Henderson and Sugden 1992)
 4. *Movement Assessment Battery for Children-2* (MABC-2) (Henderson et al. 2007)
-

In 1992, the Henderson and Sugden *Movement Assessment Battery for Children* (MABC) was published and retained the same items as the TOMI-H (see Table 5). However, the MABC was developed as a means of identifying children aged between 4 and 12 years considered being at risk of a motor impairment, and thus, normative data was added, and the scoring criteria and item descriptions were revised. The MABC consisted of 32 tasks that increased in difficulty across four age bands (AB): 4–6 years, 7–8 years, 9–10 years, and 11–12 years. Simultaneously, Keogh (1968), and subsequently Sugden (1972), developed a teacher checklist which served as preliminary to further evaluative assessments of a child's motor performance and to alert “teachers to the existence of children with movement difficulties” (Henderson et al. 2007, p. 113). Further development has led to it becoming the MABC Checklist (Henderson and Sugden 1992; Reynard 1975; Sugden 1972).

Psychometric Data

Types of reliability data often reported for scales, tests, and measures include internal consistency, test-retest/time sampling reliability/temporal stability, inter-rater/inter-scorer reliability, intra-rater/intra-scorer reliability, alternate form reliability, and split-half reliability (American Educational Research Association [AERA], American Psychological Association [APA], & National Council on Measurement in Education [NCME] 1999; Anastasi and Urbina 1997). Types

of validity often reported for tests include face validity, content validity, criterion-related validity, and construct validity (AERA et al. 1999). Two subtypes of criterion-related validity frequently included are concurrent validity and predictive validity, while subtypes of construct validity often reported include factor analysis validity, discriminant validity, convergent validity, divergent validity, diagnostic validity, and rating scale validity (Fawcett 2007). Details of the reliability and validity of the MABC-2 are reported in its manual.

The MABC-2 is a major revision of the well-known and frequently used MABC (Barnett and Henderson 1998; Henderson et al. 2007). The test authors assume that the reliability data and validity information reported for the MABC are generalizable to the MABC-2. “Confidence in the MABC-2 score interpretation can be derived not only from the UK standardisation study but also from the extensive validation data reported in this and earlier manuals” (Henderson et al. 2007, p. 132). The MABC and MABC-2 may assess the same motor skill constructs in a similar format, but since the MABC-2 has added four new items, revised some of the retained items, reduced number of age ABs (the MABC had 4 ABs, while the MABC-2 has 3), and increased age range that it covers (the MABC-2 now covers ages 3–17 years), it is essentially a new, discrete test that needs to have its own specific measurement properties evaluated singly. This appears to be an inaccurate assumption made by the MABC-2 authors.

The MABC-2 test manual reports some preliminary reliability data for its Performance Test based on the results of several studies completed by other investigators that involved experimental versions of the MABC-2 AB1 and AB3 tasks (see Table 6). Visser and Jongmans (2004) investigated the test-retest results for the MABC-2 AB1 with a group of 55 3-year-old children from the Netherlands. Chow et al. (2002), in another study involving a sample of 31 adolescents, evaluated the inter-rater and test-retest reliability of a translated version of the MABC-2 AB3 tasks into Chinese. Smits-Engelsman et al. (2008) reported about the inter-rater reliability of the MABC-2. In

another inquiry involving 64 young adults, Faber and Nijhuis van der Sanden (2004) examined the intra-rater and inter-rater reliability of a total score calculated for the MABC-2 AB3 tasks originally used by Chow et al. (2002). Details of the translation process for the Chinese version of the MABC-2 AB3 tasks used in the Chow et al. (2002) study were not reported in the test manual. Similarly, it was not reported if the version of the MABC-2 AB1 tasks used in the Visser and Jongmans (2004) investigation was translated into another language since it was used in the Netherlands. The studies completed by Visser and Jongmans (2004) and Faber and Nijhuis van der Sanden (2004) are both unpublished manuscripts, thus have not been peer reviewed nor are they readily accessible for review. The Faber and Nijhuis van der Sanden (2004) study involved 64 young adults aged between 18 and 28 which were outside the age limits of the MABC-2 and the geographical location where the study was completed was not reported. With all of these studies, there are issues of cultural context, translation of the MABC-2 Performance Test items, and only evaluating one AB at a time.

Henderson et al. (2007) reported a test-retest study involving 20 3-year-old children. Pearson product moment correlations ranged from 0.86 to 0.91 for the three Manual Dexterity tasks, while the Aiming and Catching and Balance tasks were less reliable with coefficients of 0.48 and 0.68. The test authors suggested that the test-retest reliability problems lie with younger children, aged between 3 and 4 years. In another study completed by the test authors, the test-retest reliability of the whole test involving all three ABs was completed. Sixty children, 20 from each AB, were included. Using the standard scores for the three test sections (Manual Dexterity, Aiming and Catching, and Balance) as well as the total test score, the Pearson product moment correlations were 0.77, 0.84, 0.73, and 0.80, respectively (Henderson et al. 2007). This indicates reasonable test-retest reliability results for the MABC-2. It would have been informative if the test authors had completed similar studies evaluating the intra-rater reliability, inter-rater reliability, and internal consistency of the Manual Dexterity, Aiming and Catching,

Movement Assessment Battery for Children: Second Edition (MABC-2), Table 6 *Movement Assessment Battery for Children – Second Edition (MABC-2; Henderson*

et al. 2007) summary of MABC-2 Performance Test and Checklist validity and reliability data

Test section	MABC-2 performance test	MABC-2 checklist
Reliability data reported	Limited reliability data reported is reported in the manual that is specific to the MABC-2. A study completed by Visser and Jongmans (2004) that reports test-retest results for AB1 for a group of 55 3-year-old children from the Netherlands is included in the test manual. Pearson product moment correlations ranged from 0.49 to 0.70 The test authors reported a second test-retest study involving 20 3-year-old children. Pearson product moment correlations ranged from 0.86 to 0.91 for the three manual dexterity tasks, while the aiming and catching tasks were less reliable with coefficients of 0.48 and 0.68. The test authors suggest that the test-retest reliability problems lie with younger children, aged between 3 and 4 years In another study that involved a translated version of AB3 tasks into Chinese, inter-rater reliability and test-retest reliability were evaluated on a sample of 31 teenagers by Chow et al. (2002). Intraclass correlation (ICC) coefficients for inter-rater reliability ranged from 0.92 to 1.00, while test-retest coefficients ranged from 0.62 to 0.92 In another study involving 64 young adults, Faber and Nijhuis van der Sanden (2004) examined the intra-rater and inter-rater reliability of a total score calculated for the AB3 tasks originally used by Chow et al. (2002). The ICC scores were 0.79 (intra-rater) and 0.79 (inter-rater) In a final study completed on the test authors, the test-retest reliability of the whole test involving all three ABs was completed. Sixty children, 20 from each AB, were included. Using the standard scores for the three test sections (manual dexterity, aiming and catching, and balance) as well as the total test score, the Pearson product moment correlations were 0.77, 0.84, 0.73, and 0.80, respectively	The test manual states that no reliability data has been collected on the MABC-2 checklist; thus, none is reported. The test authors do report reliability data about the MABC checklist, but this is redundant since the MABC-2 checklist has been revised
Validity data reported	Extensive validity data about the MABC is reported in the manual, and limited preliminary validity evidence (content, face, criterion-related) about the MABC-2 is also reported. Content validity of the MABC-2 was established by input of an expert panel. According the test manual, the expert panel was unanimous that the MABC-2 contents/items were representative of the motor domain it was intended to evaluate The test manual reports that face validity has been established by feedback obtained from a wide range of professionals who have used the MABC including psychologists, therapists, educational	Two validity studies are reported in the test manual. The first study involves a group of 20 children with DCD ages 6–11 years (Barnett et al. 2007). The MABC checklist was completed by the children’s parents. Scatterplots of section A and section B of the checklist suggested a weak relationship between the two section scores. The MABC-2 checklist traffic light system was able to indicate that the children continued to have motor skill difficulties in 19 of the 20 children. The test authors claim that this is evidence of criterion-related validity, but this is not clearly demonstrated

(continued)

Movement Assessment Battery for Children: Second Edition (MABC-2), Table 6 (continued)

Test section	MABC-2 performance test	MABC-2 checklist
	professionals, and physicians. The face validity appears to be based on the MABC and not the MABC-2 The test manual reports subtest and total test standard score correlations as evidence of the MABC-2 subtests evaluating related, but different motor abilities. The manual dexterity subtest was correlated with the aiming and catching (0.26), balance (0.36), and total test score (0.76). The aiming and catching subtest was correlated with the balance (0.25) and the total test score (0.65). The balance subtest was correlated with the total test score (0.73) Evidence of criterion-related validity of the MABC-2 was reported in three studies in the test manual. First, using a sample of 31 Cypriot children, Kavazi (2006) examined the relationship MABC-2 manual dexterity items and the children’s performance on the Goodenough and Harris draw-a-man test. Correlations with the posting coins and drawing trail items and the draw-a-man test were 0.66 The preliminary results of a second study completed by the test authors involving a small sample of 20 children who were initially assessed with the MABC and were reassessed using the MABC-2. The MABC-2 was able to indicate that the children continued to have motor skill difficulties. The type of validity being evaluated here is not clearly reported. The third study reports the results of 25 boys diagnosed with Asperger’s syndrome who were assessed by the MABC-2 (Siaperas et al. 2007). The MABC-2 total test scores indicated that the majority of the boys (21/25) have impaired motor skills. The test authors are purporting that this is evidence of the MABC’s discriminative validity	The second study involves 24 boys with Asperger’s syndrome with a mean age of 13.3 years (Siaperas et al. 2007). The MABC checklist was completed by the children’s parents. The MABC-2 checklist traffic light system was able to indicate that in 23 of the 24 boys with Asperger’s syndrome experienced motor skill difficulties. Again, the test authors make the claim that this is evidence of criterion-related validity, but how this is actually demonstrated is not clear
Clinical utility	The MABC-2 appears to have reasonable to good clinical utility. The international recognition of the MABC and its use with identifying children with DCD as well as other motor impairments has seen it become one of the most commonly utilized motor assessments. In addition, the brevity of the test across a wide age range of children furthermore enhances the utility of the MABC-2 in various clinical, educational, and research contexts. In summary, the MABC-2 is an improvement over the MABC and is best used as a screening instrument. Blank et al. (2012) noted that attentional problems in children may impact on their performance on the MABC-2 and that “there seems to be a training effect of the M-ABC if repeated within 4 weeks, although this effect seems to be less in children with severe DCD” (p. 71)	

and Balance test sections as well as the total test score involving all three MABC-2 ABs. At this time, this fundamental information is lacking.

The test manual states that no reliability data has been collected on the MABC-2 Checklist; thus, none is reported. The test authors do report reliability data about the MABC Checklist, but this is redundant since the MABC-2 Checklist has been revised. Test-retest reliability and internal consistency reliability data needs to be reported about the MABC-2 Checklist.

When it comes to the validity of the MABC, the test authors include a large body of evidence in the MABC-2 test manual (Barnett and Henderson 1998; Henderson et al. 2007). However, generalizations from the MABC's validity findings to the MABC-2's validity cannot be readily made for a number of reasons. First, the age range is different between the two test versions with the MABC-2 now including children as young as 3 years and adolescents as old as 16 years. The ABs have fundamentally changed between the two test versions. The MABC ABs 2 and 3 have been collapsed into the MABC-2 AB 2. Finally, a number of test items have been revised, and four new test items have been added to the MABC-2. Hence, the validity properties of the first test cannot be generalized to the newer revised version in the opinion of this author.

Limited preliminary content, face, criterion-related validity, and evidence about the MABC-2 Performance Test are reported in its test manual. Content validity of the MABC-2 Performance Test was established by input of an expert panel. According to the test manual, the expert panel was unanimous that the Performance Test contents/items were representative of the motor domain it was intended to evaluate. The content validity of the MABC-2 Performance Test appears reasonable (see Table 6). The test manual reports that face validity has been established by feedback obtained from a wide range of professionals who have used the MABC including psychologists, therapists, educational professionals, and physicians. However, the face validity appears to be based on the MABC and not the MABC-2 Performance Test. Similarly, face validity of an instrument does not have inherent psychometric,

objective, or numeric data attached to it to ensure that an instrument is evaluating the skills, attributes, or constructs it claims to assess. Face validity is mainly based on an overall subjective impression of a test (AERA et al. 1999).

The test manual reported section and total test standard score correlations as evidence of the three MABC-2 Performance Test sections evaluating related but distinct motor skill abilities (see Table 6). The Manual Dexterity section was correlated with the Aiming and Catching section (0.26), Balance section (0.36), and MABC-2 total test score (0.76). The Aiming and Catching section was correlated with the Balance section (0.25) and the total test score (0.65). The Balance section was correlated with the total test score (0.73). This provides evidence that the three MABC-2 Performance Test sections are measuring related but discrete skills and "the fact that each component correlates well with the Total Test Score is re-assuring" (Henderson et al. 2007, p. 142).

Evidence of criterion-related validity of the MABC-2 Performance Test was reported in the form of three studies in the test manual (see Table 6). First, enlisting a sample of 31 Cypriot children, Kavazi (2006) examined the relationship MABC-2 Manual Dexterity section items and the children's performance on the *Goodenough and Harris Draw-a-Man Test* (Goodenough and Harris 1963). Correlations with the Posting Coins and Drawing Trail items and the Draw-a-Man Test were 0.66. Limitations of this study were a small sample size (31 children) and correlating the children's fine motor skill performance with their performance on an outdated person drawing test. Given the fact that the Kavazi study only involved the Manual Dexterity section items from one MABC-2 AB1, the concurrent validity results are not generalizable to the whole measure. The scope of the Kavazi (2006) study was very narrow.

The preliminary results of a second study completed by the test authors involved a small sample of 20 children who were initially assessed with the MABC and were reassessed using the MABC-2 Performance Test (Henderson et al. 2007). The MABC-2 was able to indicate that the children

continued to have ongoing motor skill difficulties. The type of validity being evaluated here was not evident to the reader. It would have been more prudent to compare the whole MABC-2 to a well-established motor skill tests such as the *Peabody Developmental Motor Scales – 2nd edition* (PDMS-2) or the *Bruininks-Oseretsky Test of Motor Proficiency, 2nd edition* (BOT-2). Spironello et al. (2010) examined the construct validity and concurrent validity between the short form of the BOTMP and the MABC, while Van Waelvelde et al. (2007a) investigated the convergent validity between the MABC and the PDMS-2.

The third study reported the results of 25 boys diagnosed with Asperger's syndrome who were assessed by the MABC-2 Performance Test Siaperas et al. (2007). The MABC-2 Performance Test total scores indicated that the majority of the boys (21/25) had impaired motor skills. The test authors purported that this is evidence of the MABC-2 Performance Test's discriminative validity. The study by Siaperas et al. (2007) did not include a matched control group for comparison purposes, therefore, claims that the MABC-2 exhibits discriminant validity cannot really be supported.

One major weakness with the validity evidence reported in the MABC-2 test manual is the lack of construct validity evidence. There is no evidence that clearly indicated the MABC-2 Performance Test items are actually evaluating the motor skill constructs they claim they are. Evidence of construct validity can include divergent/convergent validity, factor analysis validity, diagnostic validity, rating scale validity, and/or discriminative validity. The completion of a confirmatory factor analysis, a component of classical test theory, would provide valuable evidence if the items from the Manual Dexterity, Aiming and Catching, and Balance sections load on three discrete factors and whether the eight items that make the whole MABC-2 load on one overall motor impairment factor. Hypotheses about the MABC-2 Performance Test factor structure need to be explored further (Anastasi and Urbina 1997). Blank et al. (2012) noted that there "is a lack of research on the discriminant validity of the M-ABC" (p. 71).

In regard to the MABC-2 Checklist, two validity studies are reported in the test manual (see Table 6). The first study involves a group of 20 children with DCD ages 6–11 years (Barnett et al. 2007). The MABC Checklist was completed by the children's parents. Scatterplots of Section A and Section B of the Checklist suggested a weak relationship between the two section scores. The MABC-2 Checklist Traffic Light system was able to indicate that 19 of the 20 children continued to have motor skill difficulties. The test authors claim that this is evidence of criterion-related validity, but how this is demonstrated is not clear.

The second MABC-2 Checklist study involved 24 boys with Asperger's syndrome with a mean age of 13.3 years (Siaperas et al. 2007). The MABC-2 Checklist was completed by the children's parents. The MABC-2 Checklist Traffic Light system was able to indicate that 23 of the 24 boys with Asperger's syndrome experienced motor skill difficulties. Again, the test authors make the claim that this is evidence of criterion-related validity, but how this is actually demonstrated is not clear. Similar to the MABC-2 Performance Test, the Checklist has limited content, concurrent, and construct validity evidence. Further validity evidence is needed since one is left asking the question: do the MABC-2 Checklist items load on the constructs they claim to measure?

A number of studies have been completed by other researchers that have evaluated different aspects of the MABC-2's reliability. These studies have been completed in a variety of cross-cultural settings as well including Greece, Norway, Japan, mainland China, Taiwan, Israel, and Australia. This provides valuable insights about how suitable the MABC-2 is for use in cross-cultural settings. For example, Engel-Yeger et al. (2010) examined the construct validity of the MABC-2 and its appropriateness to use in Israel. Participants in the study were 249 typically developing children between four and 12 years of age. The findings indicated that children's age and gender, as well as mother's education level and socioeconomic status had an influence on the children's motor performance as assessed by the MABC-2.

Engel-Yeger et al. (2010) concluded that the “M-ABC may serve as a suitable tool for examining the motor performance of children in Israel” (p. 87).

In a Greek setting, Ellinoudis et al. (2011) completed an investigation of the reliability and validity of the age band 1 of the MABC-2 involving 183 children ranging in from 36 to 64 months old (mean = 50 months, SD = 9 months). “Cronbach’s alpha coefficient values were 0.51, 0.70 and 0.66 for manual dexterity, aiming and catching, and balance task groups, respectively” (Ellinoudis et al. 2011, p. 1049). Thirty children completed the age band 1 of the MABC-2 items on two occasions 1 week apart. The test-retest reliability correlations for the eight items ranged from 0.66 to 0.96 and for the manual dexterity, aiming and catching, balance task and total scores were 0.82, 0.61, 0.90, and 0.85. “A confirmatory factor analysis was performed to test the goodness-of-fit of the items. The analysis revealed values for the three-domain model (manual dexterity, aiming and catching and balance) that are similar to the structure proposed by Henderson et al. (2007)” (Ellinoudis et al. 2011, p. 1049).

Wuang et al. (2012a) examined the internal consistency, test-retest reliability, and responsiveness to change of the MABC-2 in a group of 144 children from Taiwan diagnosed with DCD. A Cronbach alpha of 0.90 was reported for the internal consistency coefficient for the MABC-2 while the coefficients for the Manual Dexterity, Aiming and Catching, and Balance subscales were 0.81, 0.84, and 0.88, respectively. An intraclass correlation coefficient of 0.97 was reported as the test-retest reliability for the MABC-2 total score for a 20-day interval between test administrations and the ICCs for the items ranging between 0.88 and 0.88. “The minimal detectable change was 0.28 points whereas the minimal important difference (MID) values were from 2.36 to 2.50” (Wuang et al. 2012, p. 160). The authors suggest that the minimal detectable change and minimal important difference scores for the MABC-2 provide the point of reference for practitioner decision-making in clinical management of children presenting with DCD.

Similar to the study by Ellinoudis et al. (2011), Hua et al. (2013) evaluated the internal consistency, test-retest reliability, criterion-related validity, and construct validity of the age band 1 of MABC-2. Children from mainland China were recruited from 10 public nursery schools with resultant sample size of 1823 participants (915 boys and 908 girls) aged 36–72 months old (mean = 61.284 months, SD = 10.212 months). The Cronbach’s alpha coefficient for the eight age band 1 MABC-2 items ranged from 0.43 to 0.52 (Hua et al. 2013). The test-retest reliability ($n = 184$) ranged between 0.83 and 0.99 for the eight age band 1 MABC-2 items with a 14-day interval between test administrations (Hua et al. 2013). The inter-rater reliability ($n = 184$) for the eight age band 1 MABC-2 items ranged between 0.89 and 0.99 (Hua et al. 2013). A subsample of 184 children completed both the MABC-2 and the PDMS-2 and their scores were correlated. The correlations between the PDMS-2 total scale scores and the MABC-2 Manual Dexterity, Aiming and Catching, and Balance subscales and Total scores were 0.38, 0.063, 0.17, and 0.63, respectively (Hua et al. 2013). “The results showed that the total standard scores on the two tests were correlated moderately with each other” (Hua et al. 2013, p. 805). The authors completed a confirmatory factor analysis to examine the three-factor structure of the MABC-2 with the eight items as proposed by Henderson et al. (2007). The CFA results indicated that a six-item version of the MABC-2 met the goodness-of-fit indices of the adjusted model (each above 0.9), indicating a reasonable fit to the model. “The factor loadings of two items (‘Drawing trail’ and ‘Walking heels raised’) for their domains in the model were not significant statistically” (Hua et al. 2013, p. 807).

Kita et al. (2016) investigated the applicability of the MABC-2 age band 2 to Japanese children. A group of 132 typically developing Japanese children completed the age band 2 assessment items. The internal consistency, factorial validity, gender differences, and a comparison to the MABC-2 normative sample was examined. Cronbach’s alpha coefficient for the eight test items was 0.602 and ranged from 0.53 to 0.58.

All indices from the confirmatory factor analysis of the eight MABC-2 items indicated “a good fit between the postulated model and the data” and “these results demonstrated high factorial validity of the AB2 in the Japanese samples” (Kita et al. 2016, p. 710). When the item scores were in the Japanese sample were compared with the MABC-2 normative sample, the results suggested that the sample of Japanese children overall tended to have higher scores on the age band 2 compared with the group of MABC-2 normative sample who were British children (Kita et al. 2016). When the performance of Japanese boys and girls were compared on the three MABC-2 domain scores, girls obtained higher scores on Manual Dexterity and Balance while there were no significant gender differences in Aiming and Catching.

Thirty-eight children and parents was recruited from Victoria, Australia were recruited by Kennedy et al. (2013) to investigate whether the Physical Self-Description Questionnaire completed by children or the MABC-2 Checklist completed by the parents of the children were predictive of the children’s BOT-2 performance results. The findings indicated that two significant predictive relationships were identified based on parents’ report of their children’s motor skills: (i) the total score of the MABC-2 Checklist was found to be a significant predictor of the BOT-2 Manual Coordination motor composite score, accounting for 8.35% of its variance, and (ii) the MABC-2 Checklist total score was a significant predictor of the BOT-2 Strength and Agility motor composite score, accounting for 11.6% of its variance (Kennedy et al. 2013). No predictive relationships were identified between the PSDQ and the BOT-2 performance scores.

Holm et al. (2013) investigated the intra- and inter-rater reliability of the MABC-2 amongst a group of 45 healthy children from Norway (mean age 8.7 years). Thirty children were included in the inter-rater reliability component of the study, 29 children in the intra-rater part, 14 of them took part in both studies. Qualified physiotherapists completed the MABC-2 assessments. The intra-rater reliability test administrations occurred 1–2 weeks apart. The ICC values for the intra-

rater reliability of eight MABC-2 items ranged from 0.24 to 0.77 (Holm et al. 2013). For the three age band 2 domains, the intra-rater reliability ICCs were 0.62 for manual dexterity, 0.49 for aiming and catching, and 0.49 for balance (Holm et al. 2013). For the MABC-2 total test score, the intra-rater reliability ICC was 0.68. In relation to inter-rater reliability, for the MABC-2 age band 2 items, the ICCs ranged from 0.39 to 0.68. For the three age band 2 domains, the inter-rater reliability ICCs were 0.63 for manual dexterity, 0.77 for aiming and catching, and 0.29 for balance (Holm et al. 2013). The inter-rater reliability for the MABC-2 age band 2 total test score was 0.62. The authors concluded that while the MABC-2 exhibited reasonable intra- and inter-rater reliability, the “findings may indicate that the MABC-2 might be more suitable for diagnostic or clinical decision making purposes, than for evaluation of change over time” (Holm et al. 2013, p. 799).

Lane and Brown (2015) investigated the convergent validity between BOT-2 and the MABC-2 age bands 2 and 3. A sample of 50 children from Australia with no history of motor or intellectual problems was recruited. The results of the study indicated that MABC-2 age band 3 was significantly associated with the BOT-2; however, there were no significant correlations between the MABC-2 age band 2 and the BOT-2.

Clinical Uses

The MABC, and now the MABC-2, is one of the most widely used pediatric motor impairment assessments (Getchell et al. 2007; Livesey et al. 2007; Slater et al. 2010; Van Waelvelde et al. 2007b). It has been used to assess a variety of diagnostic groups including children with (1) attention deficit/hyperactivity disorder (ADHD) (Miyahara et al. 2006; Pitcher et al. 2003), (2) autistic spectrum disorders (ASD) (Green et al. 2002; Liu and Breslin 2013; Smith 2004), (3) special language impairments (Hill et al. 1998), (4) developmental coordination disorder (DCD) (Cairney et al. 2009; Chen et al. 2009; Chow et al. 2006; Cox et al. 2015; Niemeijer et al. 2006; Rosblad and Gard 1998;

Ruckser-Scherb et al. 2013; Spittle et al. 2013), (5) cognitive impairments or learning difficulties (Henderson and Sugden 1992; Jongmans et al. 2003), and (6) hearing impairment (De Kegel et al. 2012). Other diagnostic studies where the MABC has been used include congenital hypothyroidism (Kooistra et al. 1998), childhood encephalitis (Rantala et al. 1991), epilepsy (Beckung et al. 1994), neurofibromatosis 1 (North et al. 1994), generalized joint hypermobility (de Boer et al. 2015), birth-related brachial plexus injury (Bellows et al. 2015), hemiplegia (Mercuri et al. 1999), preterm infants at older ages who are at risk for poor motor outcomes (Brown et al. 2015; Keunen et al. 2016; Setänen et al. 2016), and intellectual disability (Wuang et al. 2012). Individuals diagnosed with ASD, Asperger's syndrome (AS), and other related pervasive developmental disorders (PDD) often present with motor skill difficulties (Dziuk et al. 2007; Fournier et al. 2010; Fuentes et al. 2009; Gidley Larson and Mostofsky 2006; Green et al. 2009; Jasmin et al. 2009; Ozonoff et al. 2008; Pan et al. 2009; Provost et al. 2007; Staples and Reid 2010). Hence, the MABC-2 is a useful and relevant measure to use when assessing children with ASD, AS, PDD, and other related diagnoses.

The MABC-2 Performance Test and Checklist items provide invaluable information for practitioners related to the identification of and intervention with children presenting with motor difficulties. It can be used in a range of settings including clinical, community-based, child care, early intervention, early childhood education, primary and secondary school settings, private practice, hospital, and rehabilitation. It can be used as either a quick screening instrument or as one component of a comprehensive diagnostic evaluation. The MABC-2 Performance Test and Checklist items can be used by a variety of health and education professionals (including occupational therapists, psychologists, early childhood education teachers, social workers, counselors, physiotherapists, speech-language pathologists, nurses, pediatricians, physicians, and early intervention specialists among others) to evaluate the motor skills of children and adolescents aged 3–17 years.

It is recommended that other sources of information (e.g., medical records, clinical observations, interviews with parents and teachers) be accessed as well. The clinical purposes the MABC-2 could be used for are establishing the baseline of a client's gross and fine motor skills (often done in the context of an initial assessment), assisting with the formulation of a client's intervention goals, monitoring a client's progress after receiving intervention/remedial services, or providing evidence to justify the need for a client to receive continued services. The MABC-2 Performance Test and Checklist can also be used for research purposes such as evaluating the effectiveness of educational, psychological, therapeutic, or medical services provided to pediatric clients. Two limitations were recently noted about the MABC-2 by Blank et al. (2012): (i) the potential for floor effects in task age band 1 for children 3–6 years of age and (ii) the “discontinuation’ of the scales moving from one age band to another may be a problem in longitudinal comparisons, when children, for example, move from kindergarten to school age and for the comparison of children in first grade (6- to 7-year-olds)” (p. 71) since these age bands are often significant for the diagnosis and treatment monitoring of DCD.

In a recent review of assessment tools that are recommended for use with children presenting with suspected DCD at the 2011 European Academy for Childhood Disability, Blank et al. (2012) stated that the studies published about the MABC-2 “show good to excellent interrater reliability, good to excellent test–retest reliability and fair to good validity (construct validity and concurrent validity with BOTMP)” (p. 71). Blank et al. (2012) also remarked that considering the overall “strengths and limitations of the M-ABC, the level of evidence on quality and suitability of the M-ABC (-2) for the diagnosis of DCD (SDDMF) is rated as moderate to good” (p. 71). In summary, the MABC-2 is highly recommended for use by practitioners. It can play an important role in the assessment of and intervention planning for children and adolescents presenting with ASD, AS, and PDD.

See Also

- ▶ [Bruininks-Oseretsky Test of Motor Proficiency](#)
- ▶ [Fine Motor Development](#)
- ▶ [Gross Motor Skills](#)
- ▶ [Occupational Therapy \(OT\)](#)
- ▶ [Peabody Developmental Motor Scales \(PDMS\)](#)
- ▶ [Procedural Reliability](#)
- ▶ [Sensorimotor Development](#)
- ▶ [Standardized Tests](#)
- ▶ [Validity](#)

References and Reading

- American Educational Research Association, American Psychological Association, & National Council on Measurement in Education. (1999). *Standards for educational and psychological testing*. Washington, DC: American Psychological Association.
- Anastasi, A., & Urbina, S. (1997). *Psychological testing*. Upper Saddle River: Prentice Hall International.
- Barnett, A. L., & Henderson, S. E. (1998). *An annotated bibliography of studies using the TOMI/Movement ABC: 1984–1996*. London, UK: The Psychological Corporation/Harcourt Brace.
- Barnett, A. L., Sugden, D. A., & Henderson, S. E. (2007). *Review of the Movement ABC Checklist – Second Edition*. Paper presented at the 8th motor control and human skill conference, Fremantle, Western Australia.
- Beckung, E., Uvebrandt, P., Hedstrom, A., & Rydenhag, B. (1994). The effects of epilepsy surgery on the sensorimotor function of children. *Developmental Medicine and Child Neurology*, 36, 803–901.
- Bellows, D., Bucevska, M., & Verchere, C. (2015). Coordination and balance in children with birth-related brachial plexus injury: A preliminary study. *Physiotherapy Canada*, 67(2), 105–112.
- Blank, R., Smits-Engelsman, B., Polatajko, H., & Wilson, P. (2012). European Academy for Childhood Disability (EACD): Recommendations on the definition, diagnosis and intervention of developmental coordination disorder (long version). *Developmental Medicine and Child Neurology*, 54, 54–93.
- Brown, T., & Lalor, A. (2009). The Movement Assessment Battery for Children – second edition (MABC-2): A review and critique. *Physical & Occupational Therapy in Pediatrics*, 29(1), 86–103.
- Brown, L., Burns, Y. R., Watter, P., Gibbons, K. S., & Gray, P. H. (2015). Motor performance, postural stability and behaviour of non-disabled extremely preterm or extremely low birth weight children at four to five years of age. *Early Human Development*, 91(5), 309–315.
- Cairney, J., Hay, J., Veldhuizen, S., Missiuna, C., & Fought, B. E. (2009). Comparing probable case identification of developmental coordination disorder using the short form of the Bruininks–Oseretsky Test of Motor Proficiency and the Movement ABC. *Child: Care, Health and Development*, 35, 402–408.
- Camden, C., Rivard, L., Pollock, N., & Missiuna, C. (2013). Facilitating a DCD diagnosis: Movement Assessment Battery for Children (MABC-2). Retrieved on 4 Feb 2017, from http://elearning.canchild.ca/dcd_pt_workshop/assets/identification/mabc-2.pdf
- Chen, Y. W., Tseng, M. H., Hu, F. C., & Cermak, S. A. (2009). Psychosocial adjustment and attention in children with developmental coordination disorder using different motor tests. *Research in Developmental Disabilities*, 30, 1367–1377.
- Chow, S. M. K., & Henderson, S. E. (2003). Brief report – Interrater and test-retest reliability of the Movement Assessment Battery for Chinese preschool children. *American Journal of Occupational Therapy*, 57(5), 574–577.
- Chow, S. M. K., Henderson, S. E., & Barnett, A. L. (2001). The Movement Assessment Battery for Children: A comparison of 4-year-old to 6-year-old children from Hong Kong and the United States. *American Journal of Occupational Therapy*, 55, 55–61.
- Chow, S. M. K., Chan, L. L., Chan, C. P. S., & Lau, C. H. Y. (2002). Reliability of the experimental version of the Movement ABC. *British Journal of Therapy and Rehabilitation*, 9, 404–407.
- Chow, S. M. K., Hsu, Y., Henderson, S. E., Barnett, A. L., & Lo, S. K. (2006a). The Movement ABC: A cross-cultural comparison of preschool children from Hong Kong, Taiwan, and the USA. *Adapted Physical Activity Quarterly*, 23(1), 31–48.
- Chow, S. M. K., Hsu, Y.-W., Henderson, S. E., & Yu, T.-Y. (2006b). The Movement Assessment Battery for Children: A within-culture and cross-cultural comparison of pre-school children from Hong Kong, Taiwan and the USA. *Adapted Physical Activity Quarterly*, 23, 31–49.
- Cox, L. E., Harris, E. C., Auld, M. L., & Johnston, L. M. (2015). Impact of tactile function on upper limb motor function in children with Developmental Coordination Disorder. *Research in Developmental Disabilities*, 45, 373–383.
- Crawford, S. G., Wilson, B. N., & Dewey, D. (2001). Identifying developmental coordination disorder: Consistency between tests. *Physical & Occupational Therapy in Pediatrics*, 20, 29–50.
- Croce, R. V., Horvat, M., & McCarthy, E. (2001). Reliability and concurrent validity of the Movement Assessment Battery for Children. *Perceptual and Motor Skills*, 93, 275–280.
- de Boer, R. M., van Vlimmeren, L. A., Scheper, M. C., Nijhuis-van der Sanden, M. W. G., & Engelbert, R. H. H. (2015). Is motor performance in 5.5-year-old children associated with the presence of generalized joint hypermobility? *Journal of Pediatrics*, 167(3), 694–701.
- De Kegel, A., Maes, L., Baetens, T., Dhooge, I., & Van Waelvelde, H. (2012). The influence of a vestibular dysfunction on the motor development of

- hearing-impaired children. *Laryngoscope*, 122(12), 2837–2843.
- Dewey, D., & Wilson, B. N. (2001). Developmental coordination disorder: What is it? *Physical & Occupational Therapy in Pediatrics*, 20, 5–27.
- Dziuk, M. A., Gidley Larson, J. C., Apostu, A., Mahone, E. M., Denckla, M. B., & Mostofsky, S. H. (2007). Dyspraxia in autism: Association with motor, social, and communicative deficits. *Developmental Medicine and Child Neurology*, 49(10), 734–739.
- Ellinoudis, T., Kourteissis, T., & Kiparissis, M. (2008). Suitability of the Movement Assessment Battery for Children in Greece: Comparison between a Greek sample and the North-American normative sample of 9 and 11 year old children. *International Journal of Health Science*, 1(4), 132–137.
- Ellinoudis, T., Evaggelinou, C., Kourteissis, T., Konstantinidou, Z., Venetsanou, F., & Kambas, A. (2011). Reliability and validity of age band 1 of the Movement Assessment Battery for Children – Second Edition. *Research in Developmental Disabilities*, 32, 1046–1051.
- Engel-Yeger, B., Rosenblum, S., & Josman, N. (2010). Movement Assessment Battery for Children (M-ABC): Establishing construct validity with Israeli children. *Research in Developmental Disabilities*, 31(1), 87–96.
- Faber, I. R., & Nijhuis van der Sanden, M. W. G. (2004). *The Movement Assessment Battery for Children. Standardisation and reliability of age band 5: Young adults*. Unpublished manuscript Netherlands.
- Fawcett, A. L. (2007). *Principles of assessment and outcome measurement for occupational therapists and physiotherapists: Theory, skills and application*. John Hoboken: Wiley.
- Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Caurough, J. H. (2010). Motor coordination in autism spectrum disorders: A synthesis and meta-analysis. *Journal of Autism and Developmental Disorders*, 40(10), 1227–1240.
- Fuentes, C. T., Mostofsky, S. H., & Bastian, A. J. (2009). Children with autism show specific handwriting impairments. *Neurology*, 73(19), 1532–1537.
- Getchell, N., Pabreja, P., Neeld, K., & Carrio, V. (2007). Comparing children with and without dyslexia on the Movement Assessment Battery for Children and the Test of Gross Motor Development. *Perceptual and Motor Skills*, 105(1), 207–214.
- Gidley Larson, J. C., & Mostofsky, S. H. (2006). Motor deficits in autism. In R. Tuchman & R. I. Rapin (Eds.), *Autism: A neurological disorder of early brain development* (pp. 231–247). London: MacKeith Press.
- Goodenough, F. L., & Harris, D. B. (1963). *Children's drawings as measures of intellectual maturity*. New York: Harcourt Brace.
- Green, D., Baird, G., Barnett, A. L., Henderson, L., Huber, J., & Henderson, S. E. (2002). The severity and nature of motor impairment in Asperger's syndrome: A comparison with specific developmental disorder of motor function. *Journal of Child Psychology and Psychiatry*, 43(4), 655–688.
- Green, D., Charman, T., Pickles, A., Chandler, S., Loucas, T., Simonoff, E., & Baird, G. (2009). Impairment in movement skills of children with autistic spectrum disorders. *Developmental Medicine and Child Neurology*, 51(4), 311–316.
- Henderson, S. E., & Sugden, D. A. (1992). *Movement Assessment Battery for Children*. Kent: The Psychological Corporation.
- Henderson, S. E., Sugden, D. A., & Barnett, A. L. (2007). *Movement Assessment Battery for Children-2 second edition (Movement ABC-2)*. London: The Psychological Corporation.
- Hill, E. L., Bishop, B. V. M., & Nimmo-Smith, I. (1998). Representational gestures in developmental coordination disorder and specific language impairment: Error-types and the reliability of ratings. *Human Movement Science*, 17, 655–678.
- Holm, I., Tveter, A. T., Aulie, V. S., & Stuge, B. (2013). High intra- and inter-rater chance variation of the Movement Assessment Battery for Children 2, ageband 2. *Research in Developmental Disabilities*, 34(2), 795–800.
- Hua, J., Gu, G., Meng, W., & Wu, Z. (2013). Age band 1 of the Movement Assessment Battery for Children-Second Edition: Exploring its usefulness in mainland China. *Research in Developmental Disabilities*, 34(2), 801–808.
- Jasmin, E., Couture, M., McKinley, P., Reid, G., Fombonne, E., & Gisel, E. (2009). Sensori-motor and daily living skills of preschool children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(2), 231–241.
- Jongmans, M. J., Smits-Engelsman, B. C. M., & Schoemaker, M. M. (2003). Consequences of comorbidity of developmental coordination disorders and learning disabilities for severity and pattern of perceptual-motor dysfunction. *Journal of Learning Disabilities*, 36(6), 528–537.
- Kavazi, E. (2006). *Motor competence in young Cypriot children. An examination of cross-cultural differences and the value of human figure drawings in motor assessment*. Unpublished Master's thesis, Oxford Brookes University, Oxford.
- Kennedy, J., Brown, T., & Stagnitti, K. (2013). Top-down and bottom-up approaches to motor skill assessment of children: Are child-report and parent-report perceptions predictive of children's performance-based assessment results? *Scandinavian Journal of Occupational Therapy*, 20, 45–53.
- Keogh, J. F. (1968). Incidence and severity of awkwardness among regular school boys and educationally subnormal boys. *Research Quarterly*, 39, 806–808.
- Keunen, K., Işgum, I., van Kooij, B. J. M., Anbeek, P., van Haastert, I. C., Koopman-Esseboom, C., Fieret-van Stam, P. C., Nievelstein, R. A. J., Viergever, M., de Vries, L. S., Groenendaal, F., & Benders, M. J. N. L. (2016). Brain volumes at term-equivalent age in

- preterm infants: Imaging biomarkers for neurodevelopmental outcome through early school age. *Journal of Pediatrics*, 172, 88–95.
- Kita, Y., Suzuki, K., Hirata, S., Sakihara, K., Inagaki, M., & Nakai, A. (2016). Applicability of the Movement Assessment Battery for Children-Second Edition to Japanese children: A study of the Age Band 2. *Brain & Development*, 38(8), 706–713.
- Kooistra, L., Schellekens, J. M. H., Schoemaker, M. M., Vulsma, T., & van der Meere, J. J. (1998). Motor problems in early treated congenital hypothyroidism: A matter of failing cerebellar control? *Human Movement Science*, 17, 609–629.
- Lane, H., & Brown, T. (2015). Convergent validity of two motor skill tests used to assess school-age children. *Scandinavian Journal of Occupational Therapy*, 22(3), 161–172.
- Liu, T., & Breslin, C. M. (2013). The effect of a picture activity schedule on performance of the MABC-2 for children with autism spectrum disorder. *Research Quarterly for Exercise and Sport*, 84(2), 206–212.
- Livesey, D., Coleman, R., & Piek, J. (2007). Performance on the Movement Assessment Battery for Children by Australian 3- to 5-year-old children. *Child: Care, Health and Development*, 33(6), 713–719.
- Mercuri, E., Jongmans, M., Bouza, H., Haataja, L., Rutherford, M., Henderson, S., et al. (1999). Congenital hemiplegia in children at school age: Assessment of hand function in the non-hemiplegic hand and correlation with MRI. *Neuropediatrics*, 30, 8–13.
- Missiuna, C., Rivard, L., & Bartlett, D. (2006). Exploring assessment tools and the target intervention for children with developmental coordination disorder. *Physical & Occupational Therapy in Pediatrics*, 26(1/2), 71–89.
- Miyahara, M., Tsuji, M., Hanai, T., Jongmans, M., Barnett, A. L., Henderson, S. E., et al. (1998). The Movement Assessment Battery for Children: A preliminary investigation of its usefulness in Japan. *Human Movement Science*, 17, 679–697.
- Miyahara, M., Piek, J., & Barrett, N. (2006). Accuracy of drawing in a dual-task and resistance-to-distraction study: Motor or attention deficit. *Human Movement Science*, 25, 100–109.
- Niemeijer, A. S., Schoemaker, M. M., & Smits-Engelsman, B. C. M. (2006). Are teaching principles associated with improved motor performance in children with developmental coordination disorder? A pilot study. *Physical Therapy*, 86, 1221–1230.
- North, K., Joy, P., Yuille, D., Cocks, N., Mobbs, P., McHugh, K., et al. (1994). Specific learning difficulties in children with neurofibromatosis type 1: Significance of MRI abnormalities. *Neurology*, 44, 878–883.
- Ozonoff, S., Young, G. S., Goldring, S., Greiss-Hess, L., Herrera, A. M., Steele, J., Macari, S., Hepburn, S., & Rogers, S. J. (2008). Gross motor development, movement abnormalities, and early identification of autism. *Journal of Autism and Developmental Disorders*, 38(4), 644–656.
- Pan, C., Tsai, C., & Chu, C. (2009). Fundamental movement skills in children diagnosed with autism spectrum disorders and attention deficit hyperactivity disorder. *Journal of Autism and Developmental Disorders*, 39(12), 1694–1705.
- Pitcher, T. M., Piek, J. M., & Hay, D. A. (2003). Fine and gross motor ability in males with ADHD. *Developmental Medicine and Child Neurology*, 45(8), 525–535.
- Provost, B., Heimerl, S., & Lopez, B. R. (2007). Levels of gross and fine motor development in young children with autism spectrum disorder. *Physical & Occupational Therapy in Pediatrics*, 27(3), 21–36.
- Psychometrics Centre, Cambridge Judge Business School, University of Cambridge. (n.d.). Movement Assessment Battery for Children (MABC). Retrieved 4 Feb 2017 from <http://www.psychometrics.cam.ac.uk/services/psychometric-tests/mabc-ii>
- Rantala, H., Uhari, M., Saukkonen, A., & Sorri, M. (1991). Outcome after childhood encephalitis. *Developmental Medicine and Child Neurology*, 33, 858–867.
- Reynard, C. L. (1975). *The nature of motor expectancies and difficulties in kindergarten children*. Unpublished M.Sc thesis, University of California at Los Angeles, Los Angeles, CA.
- Rosblad, B., & Gard, L. (1998). The assessment of children with developmental coordination disorders in Sweden: A preliminary investigation of the suitability of the Movement ABC. *Human Movement Science*, 17, 711–719.
- Ruckser-Scherb, R., Roth, R., Lothaller, H., & Endler, C. (2013). Motor abilities and coping in children with and without developmental coordination disorder. *British Journal of Occupational Therapy*, 76(12), 548–555.
- Setänen, S., Lehtonen, L., Parkkola, R., Matomäki, J., & Haataja, L. (2016). The motor profile of preterm infants at 11 y of age. *Pediatric Research*, 80(3), 389–394.
- Siaperas, P., Holland, T., & Ring, H. (2007). Discriminative validity of the Movement ABC Test and Checklist for use with children with Asperger Syndrome. In S. E. Henderson, D. A. Sugden, & A. L. Barnett (Eds.), *Movement Assessment Battery for Children-2 second edition (Movement ABC-2)*. London, UK: The Psychological Corporation.
- Slater, L. M., Hillier, S. L., & Civetta, L. R. (2010). The clinimetric properties of performance-based gross motor tests used for children with developmental coordination disorder: A systematic review. *Pediatric Physical Therapy*, 22, 170–179.
- Smith, I. M. (2004). Motor problems in children with autistic spectrum disorders. In D. Dewey & D. E. Tupper (Eds.), *Developmental motor disorders: A neuropsychological perspective* (pp. 152–169). New York: The Guildford Press.
- Smits-Engelsman, B. C. M., Henderson, S. E., & Michels, C. G. J. (1998). The assessment of children with developmental coordination disorders in the Netherlands: The relationship between the Movement Assessment Battery for Children and the

- Korperkoordinations Test fur Kinder. *Human Movement Science*, 17, 699–709.
- Smits-Engelsman, B. C. M., Fiers, M. J., Henderson, S. E., & Henderson, L. (2008). Interrater reliability of the Movement Assessment Battery for Children. *Physical Therapy*, 88(2), 286–294.
- Spironello, C., Hay, J., Missiuna, C., Faught, B. E., & Cairney, J. (2010). Concurrent and construct validation of the short form of the Bruininks-Oseretsky Test of Motor Proficiency and the Movement-ABC when administered under field conditions: Implications for screening. *Child: Care, Health and Development*, 36(4), 499–507.
- Spittle, A. J., Spencer-Smith, M. M., Cheong, J. L., Eeles, A. L., Lee, K. J., Anderson, P. J., & Doyle, L. W. (2013). General movements in very pre-term children and neurodevelopment at 2 and 4 years. *Pediatrics*, 132(2), e452–e458.
- Staples, K., & Reid, G. (2010). Fundamental movement skills and autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(2), 209–217.
- Stott, D. H., Moyes, F. A., & Henderson, S. E. (1972). *Test of Motor Impairment*. Guelph, Ontario, Canada: University of Guelph, Department of Psychology.
- Stott, D. H., Moyes, F. A., & Henderson, S. E. (1984). *The Test of Motor Impairment-Henderson Revision (TOMI-H)*. San Antonio, TX: The Psychological Corporation.
- Sugden, D. A. (1972). *Incidence and nature of motor problems in kindergarten school children*. Unpublished M.A. thesis, University of California at Los Angeles, CA.
- Sugden, D. A., & Wann, C. (1987). Kinaesthesia and motor impairment in children with moderate learning difficulties. *British Journal of Educational Psychology*, 57, 225–236.
- Tan, S. W., Parker, H. E., & Larkin, D. (2001). Concurrent validity of motor tests used to identify children with motor impairment. *Adapted Physical Activity Quarterly*, 18, 168–182.
- Van Waelvelde, H., Peersman, W., Lenoir, M., & Smits Engelsman, B. C. M. (2007a). Convergent validity between two motor tests: Movement-ABC and PDMS-2. *Adapted Physical Activity Quarterly*, 24(1), 59–69.
- Van Waelvelde, H., Peersman, W., Lenoir, M., & Smits Engelsman, B. C. M. (2007b). The reliability of the Movement Assessment Battery for Children for preschool children with mild to moderate motor impairment. *Clinical Rehabilitation*, 21(5), 465–470.
- Van Waelvelde, H., Peersman, W., Lenoir, M., Engelsman, B. C. M., & Henderson, S. E. (2008). The Movement Assessment Battery for Children: Similarities and differences between 4- and 5-year-old children from Flanders and the United States. *Pediatric Physical Therapy*, 20(1), 30–38.
- Visser, J., & Jongmans, M. (2004). *Extending the Movement Assessment Battery for Children to be suitable for 3-year-olds in the Netherlands*. Unpublished manuscript Netherlands.
- Wiat, L., & Darrah, J. (2001). Review of four tests of gross motor development. *Developmental Medicine and Child Neurology*, 43, 279–285.
- Wright, H. C., Sugden, D. A., Ng, R., & Tan, J. (1994). Identification of children with movement problems in Singapore: Usefulness of the Movement ABC Checklist. *Adapted Physical Activity Quarterly*, 11, 150–157.
- Wuang, Y.-P., Su, J.-H., & Su, C.-Y. (2012a). Reliability and responsiveness of the Movement Assessment Battery for Children-Second Edition Test in children with developmental coordination disorder. *Developmental Medicine and Child Neurology*, 54(2), 160–165.
- Wuang, Y.-P., Su, C.-Y., & Huang, M.-H. (2012b). Psychometric comparisons of three measures for assessing motor functions in preschoolers with intellectual disabilities. *Journal of Intellectual Disability Research*, 56(6), 567–578.

Movie and TV Depictions of Autism Spectrum Disorder

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Definition

This chapter focuses on the topic of character portrayals with autism spectrum disorders (ASD) in film and TV-series. We address this topic by presenting research from the general psychiatric field before we discuss the research on portrayals of characters with ASD. We will also provide the reader with some examples of films and TV-series that have been given particular attention in the popular media and that will be known to many, also outside of the autism community. We address both advantages and disadvantages that are consequences of popular-media portrayals of ASD.

Historical Background

In the last few years, there has been a surge of new characters in fictional film and in TV-shows that either are labelled as having an autism spectrum disorder by the makers or by the public due to resembling traits and behaviors of the characters with that on the autism spectrum. In general, portrayals of people with psychiatric disorders in film and TV-series are not an uncommon topic of research particularly in disciplines like psychiatry and media-science (Butler and Hyler 2005; Stuart 2006) but may also be found in fields like sociology in, for instance, analyzing trends in society. Conditions like memory loss or disorders like schizophrenia, depression, anxiety, and split personality have been thoroughly addressed (REF). Schneider notes that the link between psychiatry and movies go as far back as to the start of the twentieth century with films like “*The Escapees from Charenton*” in 1901 (released in Britain under the name “*Off to Bedlam*,” and in USA under the name “*Off to Bloomingdale Asylum*”) (Schneider 1987). Whether it is portrayals of psychiatrists or persons with psychiatric disorders, there are a lot of films, from drama to thrillers, from horror to comedy, available. Some of the films may be regarded as classics and films like “*The Three Faces of Eve*” (1957), “*Psycho*” (1960), “*One Flew Over the Cuckoo’s Nest*” (1975), “*Fight Club*” (1999), or “*Shutter Island*” (2010) and have had an impact on public perceptions of psychiatric disorders. Such portrayal on the screen influences the individual patient and their family, healthcare professionals, and policymakers as well as contributing to affect global, societal attitudes (Wahl 1995). However, a problem is that representations of psychiatric disorders in the mass media are often inaccurate and often overrepresent links between, for instance, violence and mental illness (Edney 2004; Stout et al. 2004; Thornton and Wahl 1996; Wahl 2003). Also, fictional portrayals of persons with psychiatric disorders or neurological conditions on film have also contributed to mystification and misconceptions. For instance, portrayals of amnesia on film and TV usually bear little resemblance to reality and in turn distort

public conception of the condition (Baxendale 2004; Hacking 2009). Further, Butler and Hyler (2005) argue that it is a common feat in Hollywood movie portrayals of children and adolescents with mental health issues or psychiatric disorder to be presented in a mythical fashion, which may be harmful in terms of misguiding public understanding of different conditions and disorders.

During the past couple of decades, portraying characters with autism spectrum disorders (ASD) have become very popular in films and in TV-shows. So much that autism is now, by many, regarded as part of the general pop-culture (Belcher and Maich 2014; Hacking 2009). Since TV and film are highly influential sources in shaping public perception of ASD, surpassing sources like clinicians, researchers, or actual encounters with people on the spectrum, it is important to address and investigate in what ways characters in TV and film are portrayed.

Current Knowledge

ASD, once considered a rare disorder, is today regarded as a common neurodevelopmental disorder or condition with estimates of prevalence somewhere around 1% in the population (Baird et al. 2006; Kogan et al. 2009).

Following the breakthrough film *Rain Man* (1988), the increase in characters with ASD in films and TV-series has been substantial (Conn and Bhugra 2012; Hacking 2010). Of course, there are examples of characters that have been portrayed as having autistic traits and behaviors before the 1980s, for example, “Philip” in “*Run Wild, Run Free*” from 1969, and “Chance, the gardener” in “*Being There*” from 1979, but the knowledge of autism at that point in time was limited and the disorder was not even a formal diagnosis in DSM before 1980. Either way it is safe to say that the release of the movie *Rain Man* marks a starting point for when autism, both as a term and as a disorder, reached a larger part of the society’s perception.

As discussed by Damjanovic et al. (2009) and Draaisma (2009), characters with ASD on film

and TV may contributed in influencing public knowledge and shaping attitudes towards the condition. But how are characters with ASD portrayed on the screen? As we noted, earlier films with characters with psychiatric disorders are known to presenting disorders in an unfortunate way in terms of, for instance, violent behaviors. However, autistic characters are typically not portrayed in this way. Instead, some argue that characters with ASD have a tendency to be portrayed more like heroes (Belcher and Maich 2014) and with special talents (Hacking 2009) and although this perhaps looks fine at first glance it may not give an accurate picture of ASD.

Publications in scholarly journals on the topic of ASD on TV can be found in various disciplines such as film history, media, philosophy, sociology, psychology, and psychiatry to name a few. Many of the publications take the form of in-depth analyses on advantages and disadvantages of portrayals of ASD on screen. Examples of key publications here are Douwe Draaisma's article on Stereotypes of autism (2009) and Stuart Murray's article where the public consciousness of autism is analyzed in light of the increased fascination of the disorder (2006). Other publications are case studies where a character in a film or TV-show is being dissected in light of his or her ASD-diagnosis or his or her autistic like traits or behaviors (see for instance Cockain 2016; Tobia and Thoma 2015).

There are few studies that report on quantitative data from larger samples of films portraying characters with ASD. Conn and Bhugra (2012) described a sample of 23 films noting that most films with characters with ASD are in the drama genre. They also argue that although there are concerns as to whether portrayals are realistic or not, there are possibilities for using fictional films for educational purposes within health sciences such as medicine and psychiatry.

Another quantitative approach on autism and film was undertaken in a PhD thesis published in 2014 (Garner 2014) as well as a publication the following year (Garner et al. 2015). Garner and colleagues used the Childhood Autism Rating Scale – Second edition (CARS2: Schopler et al. 2010) to score 15 characters with ASD from

various films. The authors concluded that character portrayals, compared to an average autism-affected population, were more severe in their display of autism symptomology (Garner et al. 2015).

A recent study used the DSM-5 (APA 2013) to investigate whether diagnostic criteria lined up with behaviors and traits displayed by fictional portrayals of characters with ASD (Nordahl-Hansen et al. 2017). In the sample of 26, there were 22 films and 4 TV-series that included characters in highly popular TV-series that has been linked to the autism spectrum (*Community*, 2009–2015, *The Big Bang Theory*, 2007–, *The Bridge*, 2011–, *Alphas*, 2011–2012). The results from this study was much in line with results found in Garner et al. (2015) as a majority in the sample was in very close alignment with DSM-5 diagnostic criteria for an autism spectrum diagnosis.

In general, the researchers conclude that character portrayals are somehow overemphasized when it comes to displaying autistic traits, which in turn may reinforce stereotypes of the disorder. This is interesting in considering a common criticism from the autism community that most portrayals, at least this past decade, are so-called “high-functioning” verbal characters with low levels of support needed in, for instance, everyday living. In this respect, it is interesting to note that a character like Abed Nadir in the TV-show “*Community*” has the lowest score on autistic traits in the Nordahl-Hansen et al. (2017) sample but is one of the most embraced characters in various “Asperger-communities.”

A topic of very high interest to the people making films and TV-shows about fictional characters with ASD that display exceptional skills or savant skills. It is easy to understand that savant skills are fascinating and just as easy to understand that screenwriters “give” these features to their characters in the hopes of making them more interesting. However, this has led to the impression that savant skills are something that most people with ASD have. But, for instance, the DSM-5 criteria for ASD (APA 2013) do not make particular notice of savant skills or savant syndrome although fixated or special interests

may or may not be regarded as savant like skills. It is not clear how many individuals within the ASD population that actually have savant skills much due to varying definitions of what constitutes savant skills. However, research indicates that the prevalence might be somewhere between 10% (Treffert 2014) to approximately 30% (Howlin et al. 2009) within the ASD population. Although these prevalence estimates are higher than in other populations, it is not a common feat in persons with ASD. Even so, there is a tendency to portray characters with ASD on screen as having savant skills (Conn and Bhugra 2012; Garner et al. 2015; Nordahl-Hansen et al. 2017) or as being intellectually stimulating geniuses (Belcher and Maich 2014).

Future Directions

Although there are similarities between many films and TV-shows in the ways characters with ASD are portrayed, there are also large differences both in terms of the portrayals but also of thematic difficulties addressed. Some films show scenes that will relate to families and people on the spectrum. Problems as a consequence of hypersensitivity to environmental stimuli, problems in school and struggles with academic success to severe bullying are some recurring themes. Further, difficulties regarding dating, sex, and relationships are frequent topic but also more overarching existential issues and general difficulties with social communication that leads to feelings of social ostracism. Nevertheless, critique has been raised about the overrepresentation of middle-class white males either in their teens or young adults that are so-called “high functioning.” This is particularly evident in the typical “Hollywood” mainstream portrayals of ASD and the debate has increased in strength following the release of TV-series like “*The Big Bang Theory*” and more recently “*The Good Doctor*” and “*Atypical*,” the latter two both aired for the first time in autumn 2017.

For the purposes of giving the public a nuanced view of the heterogeneity that resides within the autistic spectrum, a variety of portrayals of

persons with ASD on screen would be a step in the right direction. That being said, one character portrayal in a film or TV-show cannot capture the complexity that resides with the entire autism spectrum, nor do justice to the variety of autistic lived experience (Nordahl-Hansen 2017). There are, however, fictional films and TV-series available that portray characters with ASD in a wide variety of ways but many of these depictions do not reach as wide an audience as the so-called mainstream “Hollywood” productions. In addition to more films and TV-series portraying characters with ASD that have higher needs of everyday support, it would be welcoming to see more films with characters that are minimally or nonverbal or even display self-injurious behaviors. Further, although there are exceptions, like *Snow Cake* (2006), *The Bridge* (2011–), *Fly Away* (2011), *Molly* (1999), and *Mozart and the Whale* (2005) to name a few, more female characters with ASD on film and TV would also be welcome.

Additionally, portrayals of autism and aging is almost entirely missing; so although there has been a marked increase in characters with ASD in fictional films and TV-shows, there are still many gaps to fill.

Many of the films and TV-series that are available can be viewed by children but in most cases, parental guidance are encouraged but British and American TV-channels have started including characters with ASD in TV-programs that target the child as the main consumer. It is of high importance that character portrayals of persons with ASD on TV for kids do not lead to stereotype thinking of the disorder. If portrayed with care, respect, and adjusted to the target age groups, characters with ASD on the screen may teach and inform children in ways to act and behave in an inclusive manner. Of course, herein there is a great potential in reducing bullying. Furthermore, feedback from the adult autism community on portrayals of characters with ASD is that although many do not familiarize with characters on screen, many others do. This may be of particular importance for children who are experiencing being different from other peers of similar age and thus having difficulty fitting in. Characters with autistic traits in TV-shows for children could be a

potential help for children with ASD to better understand their condition.

Finally, portrayals of characters with ASD in film and TV have been proposed for use in higher education, for example, in training of healthcare personnel (Conn and Bhugra 2012; Gabbard and Horowitz 2010; Safran 1998; Sartorius et al. 2010; Butler and Hyler 2005). However, to what extent portrayals of individuals with ASD are good or bad, or right or wrong, is not easy to determine since individual portrayals cannot capture the heterogeneity that resides within the autism spectrum. Therefore, although on-screen portrayals of autism can be a good point of departure for further discussion in educational settings (Conn and Bhugra 2012; Gramaglia et al. 2013; Nordahl-Hansen et al. 2017), competent educators with in-depth knowledge and experience with persons with ASD will be key as character portrayals can never capture real life. To make use of insights from actually autistic people would further strengthen the potential of usage of film and TV for educational purposes.

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub.
- Baird, G., Simonoff, E., Pickles, A., Chandler, S., Loucas, T., Meldrum, D., & Charman, T. (2006). Prevalence of disorders of the autism spectrum in a population cohort of children in South Thames: The Special Needs and Autism Project (SNAP). *Lancet*, 21, 210–215.
- Baxendale, S. (2004). Memories aren't made of this: Amnesia at the movies. *British Medical Journal*, 329, 1480–1483.
- Belcher, C., & Maich, K. (2014). Autism spectrum disorder in popular media: Storied reflections of societal views. *Brock Education: A Journal of Educational Research and Practice*, 23, 97–115.
- Butler, J. R., & Hyler, S. E. (2005). Hollywood portrayals of child and adolescent mental health treatment: Implications for clinical practice. *Child and Adolescent Psychiatric Clinics of North America*, 31, 509–522.
- Cockain, A. (2016). Making autism through Ocean Heaven (Haiyang tiantang) and the possibilities of realizing disability differently. *Disability & Society*, 31(4), 535–552.
- Conn, R., & Bhugra, D. (2012). The portrayal of autism in Hollywood films. *International Journal of Culture and Mental Health*, 1, 54–62.
- Damjanović, A., Vuković, O., Jovanović, A. A., & Jašović-Gašić, M. (2009). Psychiatry and movies. *Psychiatria Danubina*, 21, 230–235.
- Draaisma, D. (2009). Stereotypes of autism. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 27, 1475–1480.
- Edney, D. R. (2004). *Mass media and mental illness: A literature review*. Ontario: Canadian Mental Health Association.
- Gabbard, G., & Horowitz, M. (2010). Using media to teach how not to do psychotherapy. *Academic Psychiatry*, 1, 27.
- Garner, A.R. (2014). What's showing: Film industry portrayals of autism spectrum conditions and their influences on preservice teachers in Australia, University of Wollongong. <http://ro.uow.edu.au/theses/4282>
- Garner, A., Jones, S., & Harwood, V. (2015). Authentic representations or stereotyped 'outliers': Using the CARS2 to assess film portrayals of autism Spectrum disorders. *International Journal of Culture and Mental Health*, 8, 414–425.
- Gramaglia, C., Jona, A., Imperatori, F., Torre, E., & Zeppugno, P. (2013). Cinema in the training of psychiatry residents: Focus on helping relationships. *BMC Medical Education*, 13(1), 90.
- Hacking, I. (2009). Autistic autobiography. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 364(1522), 1467–1473.
- Hacking, I. (2010). Autism fiction: A mirror of an internet decade? *University of Toronto Quarterly*, 79(2), 632–655.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2009). Savant skills in autism: Psychometric approaches and parental reports. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 27, 1359–1367.
- Kogan, M. D., Blumberg, S. J., Schieve, L. A., Boyle, C. A., Perrin, J. M., Ghandour, R. M., et al. (2009). Prevalence of parent-reported diagnosis of autism spectrum disorder among children in the US, 2007. *Pediatrics*, 1, 1395–1403.
- Nordahl-Hansen, A. (2017). Atypical: A typical portrayal of autism? *The Lancet Psychiatry*, 4(11), 837–838.
- Nordahl-Hansen, A., Tøndevold, M., & Fletcher-Watson, S. (2017). Mental health on screen: A DSM-5 dissection of portrayals of autism spectrum disorders in film and TV. *Psychiatry Research*. <https://doi.org/10.1016/j.psychres.2017.08.050>.
- Safran, S. P. (1998). Disability portrayal in film: Reflecting the past, directing the future. *Exceptional Children*, 64(2), 227–238.
- Sartorius, N., Gaebel, W., Cleveland, H. R., Stuart, H., Akiyama, T., Arboleda-Flórez, J., et al. (2010). WPA guidance on how to combat stigmatization of psychiatry and psychiatrists. *World Psychiatry*, 9(3), 131–144.
- Schneider, I. (1987). The theory and practice of movie psychiatry. *American Journal of Psychiatry*, 144(8), 996–1002.
- Schopler, E., Van Bourgondien, M. E., Wellman, G. J., & Love, S. R. (2010). *The childhood autism rating scale, (CARS2)*. Los Angeles: WPS.

- Stout, P. A., Villegas, J., & Jennings, N. A. (2004). Images of mental illness in the media: Identifying gaps in the research. *Schizophrenia Bulletin*, 30, 543–561.
- Stuart, H. (2006). Media portrayal of mental illness and its treatments. *CNS Drugs*, 1, 99–106.
- Thornton, J. A., & Wahl, O. F. (1996). Impact of a newspaper article on attitudes toward mental illness. *Journal of Community Psychology*, 24(1), 17–25.
- Tobia, A., & Toma, A. (2015). Rethinking Asperger's: Understanding the DSM-5 diagnosis by introducing Sheldon Cooper. *Journal of Communication Disorders, Deaf Studies & Hearing Aids*, 3, 146.
- Treffert, D. A. (2014). Savant syndrome: Realities, myths and misconceptions. *Journal of Autism and Developmental Disorders*, 44, 564–571.
- Wahl, O. (1995). *Media madness*. New Brunswick: Rutgers University Press.
- Wahl, O. (2003). Depictions of mental illnesses in children's media. *Journal of Mental Health*, 12(3), 249–258.

Behavioral analysis may be an effective way to analyze the function of scripting for some children. By analyzing the antecedents and consequences of scripting for children and adults with ASD, it may be possible to identify functional replacements for this behavior.

See Also

► [Echolalia](#)

References and Reading

- Rydell, P., & Prizant, B. (1995). Assessment and intervention strategies for children who use echolalia. In K. Quill (Ed.), *Teaching children with autism* (p. 110). New York: Delmar.

Movie Talk

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Synonyms

[Delayed echolalia](#); [Echolalia](#); [Parroting](#); [Scripting](#)

Definition

Repeating or reciting lines heard in TV shows, movies, songs, books, etc., is a common language pattern among children with ASD. "Movie talk" refers to repetitive speech taken from such a source, as opposed to a child's using novel, spontaneous language. Also known as scripting, the behavior involves reciting phrases or "chunks" of previously heard language and is synonymous with delayed echolalia. Some researchers believe "movie talk" is a form of self-stimulation used to tune others out or to work through a stressful or anxiety-producing moment. Others believe it is a means to initiate communication with others, or as a way to talk about an area of special interest.

MSEL:AGS

► [Mullen Scales of Early Learning](#)

MTL

► [Medial Temporal Lobe](#)

Mu Rhythm

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Synonyms

μ ; μ rhythm; [Arciform rhythm](#)

Definition

Mu (μ) rhythm is a type of emitted brain wave that can be measured via electroencephalography (EEG). The mu rhythm frequency band is defined by activity falling between 8 and 13 Hz and

recorded by scalp electrodes over the sensorimotor cortex during waking neural activity. The mu rhythm band is posited to reflect the conductance of synchronized activity in large groupings of pyramidal neurons in the brain's motor cortex (Pfurtscheller et al. 1997) but has also been proposed to reflect activity of the mirror neuron system (Pineda 2005). The gradual loss of intensity and desynchronicity of neural activity, referred to as attenuation, is suggestive of an increased load on those specific cells and indicative of significant activation (Pfurtscheller et al. 1997). Attenuation of the mu rhythm has been noted during both the execution and observation of actions falling within one's behavioral repertoire. Physical movement in the body itself does not account for mu wave attenuation during an individual's observation of other's actions. The first neurological evidence of mu rhythm attenuation came from the work of Gastaut and Bert (1954) during observations of activity and response characteristics of subjects during motor tasks. It was noted that desynchronization of mu rhythm activity from centrally located scalp electrodes occurred not only during the active movements of the subjects, but also during subjects' passive observation of the same actions performed by others in the absence of any other overt motor activity. Because of the observed attenuation of the EEG mu rhythm during the execution and observation of actions, it has been proposed to reflect mirror neuron system activity (Pineda 2005).

Given the noted impairments in imitation, empathy, and social understanding in ASD and given that the mirror neuron system has been proposed to subserve many of these social cognitive abilities, the mu rhythm has been examined among ASD populations as an assessment of the mirror neuron system. Oberman and colleagues first found that children with ASD showed reduced attenuation of the EEG mu rhythm in response to the observation of simple actions (Oberman et al. 2005), providing support for the theory that the mirror neuron system is disrupted in ASD. Bernier, Dawson, Webb, and Murias (2007) replicated this finding in adults and observed that attenuation of the EEG mu rhythm is correlated with imitative abilities. This finding provided further support that the EEG mu rhythm may reflect activity of an

execution/observation matching system, such as the mirror neuron system and that this system may be related to imitation abilities. Although the existence of a human mirror neuron system is controversial and the literature contains conflicting findings regarding the presence of a disruption of the EEG mu rhythm in ASD, the EEG mu rhythm continues to be utilized in the investigation of action execution and observation among individuals with ASD.

See Also

- [Electroencephalogram \(EEG\)](#)
- [Mirror Neuron System](#)

References and Reading

- Bernier, R., Dawson, G., Webb, S., & Murias, M. (2007). EEG mu rhythm and imitation impairments in individuals with autism spectrum disorder. *Brain and Cognition*, 64(3), 228–237.
- Gastaut, H. J., & Bert, J. (1954). EEG changes during cinematographic presentation. *Electroencephalography and Clinical Neurophysiology*, 6, 433–444.
- Oberman, L. M., Hubbard, E. M., McCleery, J. P., Altschuler, E. L., Ramachandran, V. S., & Pineda, J. E. (2005). EEG evidence for mirror neuron dysfunction in autism spectrum disorders. *Cognitive Brain Research*, 24(2), 190–198.
- Pfurtscheller, G., Neuper, C., Andrew, C., & Edlinger, A. (1997). Foot and hand area mu rhythms. *International Journal of Psychophysiology*, 26, 121–135.
- Pineda, J. E. (2005). The functional significance of mu rhythms: Translating “seeing” and “hearing” into “doing”. *Brain Research Reviews*, 50, 57–68.

Mullen Scales of Early Learning

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Synonyms

MSEL:AGS; [Mullen scales of early learning, american guidance service edition](#)

Abbreviations

ELC Early learning composite

Description

The Mullen Scales of Early Learning: American Guidance Service Edition is an individually administered, multidomain measure of early development designed for children from birth to 68 months of age. It was developed by Eileen M. Mullen, Ed.D., and published in 1995. The MSEL:AGS consists of five individual scales: four cognitive scales that cover the age range from birth through 68 months (visual reception, fine motor, receptive language, and expressive language) and one gross motor scale that is administered from birth to 33 months. The four cognitive scales can be combined to produce an Early Learning Composite (ELC) that is said to represent “g” or “general intelligence.”

The MSEL:AGS was based on the author’s information processing and neurodevelopmental theories that conceptualize intelligence “as a network of interrelated but functionally distinct cognitive skills” (Manual, p. 1). By assessing these individual areas of development independently, the examiner can identify specific areas of strength and weakness. From the author’s perspective, this developmental profile can be used to aid in making diagnoses and also in developing strategies for individualized intervention plans (Manual, p. 33). Brief descriptions of each scale are included below.

The visual reception scale consists of 33 items. It assesses the child’s processing of visually presented information and includes tasks that require visual discrimination, categorization, and visual memory skills. While the scale is not completely “nonverbal,” it was designed to minimize the amount of language needed to understand instructions and give a response. Items range from visually fixating and tracking moving objects in early infancy to matching shapes, pictures, letters, and words and remembering visual forms for older children.

The fine motor scale consists of 30 items and (similar to visual reception) also requires minimal receptive and expressive language skills. Fine motor items measure visual-motor planning and

control, motor imitation, unilateral and bilateral manipulation of objects, and writing readiness skills. Individual items range from reflexively holding objects and bringing fist to mouth to stacking blocks, cutting with scissors, and copying shapes and letters with a pencil.

The receptive language scale consists of 33 items. This area focuses on the child’s ability to process linguistic input and assesses auditory comprehension and auditory memory skills. At the earliest age levels, the items assess the infant’s orienting to sounds and responding to voices. As the item difficulty increases, the young child is asked to follow verbal directions and respond to questions about spatial, color, size, length, and number concepts. Most items can be answered by pointing to a picture or handing over an object, but some of the more advanced items require brief spoken responses, such as answering general knowledge questions.

The expressive language scale consists of 28 items and involves the production of sounds/words and the use of auditory memory. Items range from vocalizing early vowel and cooing sounds in infancy to defining vocabulary words, repeating sentences, and solving verbal analogies.

The gross motor scale consists of 35 items. The manual states that this scale measures “central motor control and mobility” (p. 2) in a variety of positions that range from being held in the caregiver’s arms to the child being fully upright and independently walking, hopping, and running.

Time required for administration of the full MSEL:AGS varies by the child’s chronological age and ability level. The manual estimates administration takes approximately 15 min for 1-year-olds, 30 min for 4-year-olds, and 60 min for 5-year-olds. Directions include information on positioning infants up to 8–10 months of age and supplemental drawings. Some items can be scored based on parent report and therefore do not have to be directly observed. This is particularly helpful in expressive language items where it is often difficult to elicit vocalizations or spoken responses from young children in an unfamiliar setting. It is also permissible to vary the order of administration of both the scales and individual items within each scale and to readminister some items after a child has “warmed up.” For children

born prematurely (at or before 36 weeks gestation), scores may be calculated based on the child's corrected age up to 2 years.

The components of a complete MSEL:AGS include the Mullen test kit, stimulus booklet, record forms, and two manuals. The test kit contains most of the objects needed for item administration. However, some of these items lack the durability needed for young children (e.g., picture cards), and others present a potential choking hazard (e.g., small beads). The examiner must also supply a number of items not contained in the test kit, such as paper, cereal, large ball, colored tape or white chalk line, 6-in.-high bench, and stairs. Consequently, some preparation time is needed before each administration to insure that all the necessary materials are available.

The stimulus booklet contains black and white line drawings, primarily for use on visual matching, prewriting, receptive language, and expressive vocabulary tasks. On the record form, items are arranged in increasing order of difficulty for each domain. Arrows denote starting points for different ages, but examiners may use their judgment about which starting point to choose (e.g., the examiner may begin at an earlier start point for children with developmental delays). Brief descriptions are included for each item, along with cues for positioning infants and a scoring column. The MSEL:AGS Item Administration Book provides instructions for giving items in all five scales. For each item, this manual includes a list of needed materials, the recommended position for the younger infants (e.g., supported sitting, standing), administration directions, and scoring criteria. The instructions are generally understandable, particularly for professionals with experience in infancy and early childhood. The MSEL:AGS Manual provides information about the test's theoretical background, scale descriptions, general administration, scoring procedures, standardization and psychometric data, and raw score conversion tables. There are also three case studies provided to illustrate the use and interpretation of the MSEL:AGS.

Scoring for each scale varies from 1 point for a correct response to 0 points for an incorrect response on some items – to items where multiple

points can be earned for correctly completing component parts. Basal and ceiling rules are the same for each scale. A basal has been established for a scale when a child earns at least 1 point for each of three consecutive items beginning at a starting point. Testing continues until the child obtains scores of 0 points on three consecutive items. The sums of raw scores for each scale are used to compute derived scores that include T scores (mean = 50; SD = 10), percentile ranks, and age equivalents for each motor and cognitive scale and an Early Learning Composite (mean = 100; SD = 15) based on the four cognitive scales. Descriptive categories are also available (e.g., very high to very low).

Historical Background

The Mullen Scales of Early Learning: AGS Edition (1995) represents a revision of the original Mullen Scales (1992) and combines the Infant MSEL (1989) and the Preschool MSEL (1992) into a single test with norms that span the age range from birth through 68 months. The author, Eileen M. Mullen, Ed.D., developed the MSEL:AGS based on 30 years of clinical experience testing young children with developmental disabilities. She designed it to include multiple developmental domains based on the assumption that specific abilities mature at different rates in infants and young children. Viewed from this perspective, a test yielding only a global score would not provide information about patterns of cognitive strengths and weaknesses that can be used to plan interventions (Manual, p. 9). To arrive at the item sets for the current edition, item analyses were conducted using the Rasch one-parameter IRT model in order to obtain item difficulty estimates and indices of goodness of fit. A few items were dropped from the earlier versions, and the remaining items were sequenced by order of difficulty for the MSEL:AGS.

The structure of the individual scales was built on the author's theory of information processing where gross motor learning is considered to be the foundation for conceptual development in both visual and auditory modalities (Manual, p. 7).

Tasks from each of the four cognitive scales were analyzed in terms of the component processes required to perform them. Individual items in each scale are purported to target intrasensory (auditory or visual) processing or intersensory (auditory-visual) processing. More specifically, the visual reception and fine motor scales are designed to assess visual intrasensory processing, while the receptive language and expressive language scales are designed to assess auditory intrasensory processing. In addition, approximately half of the items on the receptive language scale are reported to assess auditory-visual intersensory processing. However, when one carefully analyzes the individual items, it is difficult to categorize them into purely one type of sensory processing since problem solutions often require multiple skills across multiple modalities. For example, solving the form-board task on the visual reception scale involves understanding the spoken directions (auditory), matching the shapes to their recesses (visual), using fine motor control to place the shapes into their respective holes (visual-motor), and so on.

The MSEL:AGS is the second most widely used measure of early development after the Bayley Scales of Infant and Toddler Development, Third Edition (2006) (Chawarska and Bearss 2008, p. 55). Before the Bayley III (2006) was redesigned to include cognitive, language (receptive and expressive communication), and motor (fine and gross motor) scales to replace the previous more global mental and motor scales, the Mullen Scales was frequently chosen for research studies because investigators wanted to be able to assess individual developmental domains (Klin et al. 2005). In addition, the relatively long age span (birth to 68 months) was very helpful for longitudinal projects (e.g., studies of the development of baby siblings of children with autism spectrum disorders).

Psychometric Data

The Mullen Scales was standardized on a sample of 1,849 children ranging in age from 2 days to 69 months and grouped into 16 age groups

ranging from 2-month age intervals from birth to 14 months to 5-month age intervals from 15 to 68 months. However, there were uneven numbers of children across the 16 age groups, varying from a low of 84 children at 11–12 months to a high of 156 children at 27–32 months. Standardization utilized 71 clinicians from a variety of disciplines and took place at more than 100 sites across the United States. It extended over an 8-year period and was conducted in two phases that covered different time periods and included different geographic regions. Phase 1 spanned June 1981 to February 1986 and included children from the Northeast. Phase 2 covered the period between December 1987 and April 1989 and included samples from the South, West, and North, and South Central areas of the United States. The standardization sample is described as “geographically diverse,” but it is not representative of the entire United States. For example, no children in the 1–14-month age range were recruited from the North/South Central region (Bradley-Johnson 1997). For the total sample, there were 48.7% females and 51.3% males which were very close to the US population estimates in 1990. However, there was some variability among the 16 age groups, e.g., males were overrepresented at 5–6 months (59.8%) and underrepresented at 27–32 months (43.6%). Parental consent and information on demographic variables (age, sex, race/ethnicity, father’s occupation, and urban/rural residence) were obtained for all children in the sample. Settings for testing included kindergarten, day care, nursery, and homes. All children in the sample were from homes where English was the primary language. It should also be noted that children with known physical or mental disabilities were excluded from the standardization sample.

Chapter 6 of the manual provides information about the reliability and validity of the MSEL:AGS. In terms of reliability, information is provided about internal consistency, test-retest reliability, and interscorer reliability. As a measure of internal consistency, the manual presents split-half reliability coefficients for the 16 age groups of the standardization sample (Manual, p. 56). Modified split-half internal consistency

coefficients were calculated for the five scales and the Early Learning Composite. The median values ranged from 0.75 to 0.83 for the scales and 0.91 for the ELC. However, four coefficients that were significantly lower than this range were omitted from the estimation of the median split-half coefficients. The author explained these omissions as being due to a ceiling effect on visual reception for the two oldest age groups and floor and ceiling effects on receptive language for the youngest and oldest age groups. With these same four age groups omitted, the standard error of measurement had medians ranging from 4.1 to 5.0 T score points for the scales and 4.5 standard score points for the ELC.

Test-retest reliability was reported in the manual for two samples based on administration of the original Mullen Scales of Early Learning. The first sample included 50 children from 1 to 24 months of age and the second sample included 47 children ranging from 25 to 56 months. The retest interval ranged from 1 to 2 weeks, with a mean test-retest interval of 11 days. For the younger sample, test-retest reliability was high for gross motor (0.96) and ranged from 0.82 to 0.85 for the cognitive scales. For the older sample (which did not include children from 57 to 68 months), the median stability coefficients ranged from 0.71 to 0.79. Interscorer reliability was reported to be high (0.91 to 0.99), but the number of scorers was not reported, and the sample only went up to 44 months.

The manual provides information about construct, criterion-related, and concurrent validity. Support for construct validity is provided by showing an increase in mean raw scores with increasing chronological age across all five scales. Intercorrelations and squared correlations were calculated for the five Mullen Scales for 5 age groups and the total standardization sample. Scales correlated with each other at low to moderate levels. This was interpreted as indicating both unique and shared variances. Exploratory factor analyses were also conducted for the five scales by 5 age groups. Only one factor was reportedly extracted at each age level and this was interpreted as supporting the Mullen ELC as a measure of "g" or general cognitive ability.

However, establishing construct validity for this measure is problematic because there is a lack of supportive data for the author's theory about intrasensory and intersensory processing leading to the identification of the child's learning style and its subsequent implications for remediation (Bradley-Johnson 1997).

Evidence for concurrent validity is based on three types of studies: (1) studies analyzed by the publisher correlating the MSEL:AGS with other measures such as the Bayley Scales of Infant Development (1969), Preschool Language Assessment (1979), and Peabody Developmental Motor Scales (1983), (2) studies reported in the manuals for the original Infant and Preschool Mullen Scales, and (3) studies reported in the research literature which primarily focused on differences between low-birth-weight and normal-birth-weight children. The MSEL:AGS correlated moderately well with the measures mentioned above – with higher correlations obtained when the specific Mullen scale more closely matched what the other instrument was purported to measure. For example, the receptive language scale on the Mullen correlated more highly with Auditory Comprehension on the Preschool Language Assessment (1979) than with the Bayley Scales (1969) Mental Development Index (that includes both verbal and nonverbal items).

Clinical Uses

The MSEL:AGS was designed for use by early childhood professionals working in a variety of settings, such as early intervention programs, preschool and kindergarten special education, universities, research laboratories, hospitals, rehabilitation centers, and private practice. The author described three major uses: (1) eligibility determination for early intervention and special education, (2) diagnostic assessment of children with special needs, and (3) individualized program planning (Manual, p. 5–6).

Under the Individuals with Disabilities Education Act (IDEA), infants and young children must meet specific eligibility criteria in order to qualify for early intervention or special education

services. The MSEL:AGS includes three of the five areas of functioning that must be assessed (i.e., physical (including gross and fine motor skills), cognition, and communication), but additional assessment with another measure is needed to cover personal-social and adaptive/self-help skills.

Because the MSEL:AGS includes five individual scales, it is useful in diagnostic assessments where clinicians are trying to determine whether children are progressing within the typical range of development or whether they are demonstrating significant delays in one or more areas of development. Determining a profile of strengths and weaknesses is essential in diagnostic assessments of children suspected to have a wide variety of disabilities, such as specific speech-language impairments, motor impairments (e.g., cerebral palsy), autism spectrum disorders, and global developmental delays. Clinicians also use patterns of performance to formulate recommendations for skills to address in intervention. However, as noted in the validity discussion above, the modality-specific approach to individualized program planning proposed in the MSEL:AGS has not been substantiated.

In summary, the MSEL:AGS has been widely used in developmental assessments of infants and young children. Both clinicians and researchers have appreciated the multiple domain structure and relatively broad age range. Although it is regularly used for diagnostic evaluations and eligibility determination for services, children with physical and mental disabilities were excluded from the standardization sample. In addition, much of the data used for standardization was gathered in the 1980s, so the norms are outdated, and there are no current plans to renorm this measure.

References and Reading

- Akshoomoff, N. (2006). Use of the Mullen scales of early learning for the assessment of young children with autism spectrum disorders. *Child Neuropsychology*, 12, 269–277.
- Bayley, N. (1969). *Bayley scales of infant development*. San Antonio: Psychological Corporation.

- Bayley, N. (2006). *Bayley scales of infant and toddler development* (3rd ed.). San Antonio: Harcourt Assessment.
- Bradley-Johnson, S. (1997). Mullen scales of early learning. *Psychology in the Schools*, 34, 379–382.
- Chawarska, K., & Bearss, K. (2008). Assessment of cognitive and adaptive skills. In K. Chawarska, A. Klin, & F. R. Volkmar (Eds.), *Autism spectrum disorders in infants and toddlers: Diagnosis, assessment and treatment* (pp. 50–75). New York: The Guilford Press.
- Klin, A., Saulnier, C., Tsatsani, K., & Volkmar, F. R. (2005). Clinical evaluation in autism spectrum disorders: Psychological assessment with a transdisciplinary framework. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, pp. 772–798). Hoboken: Wiley.
- Mullen, E. M. (1989). *Infant Mullen scales of early learning*. Cranston: T.O.T.A.L. Child.
- Mullen, E. M. (1992). *Mullen scales of early learning*. Cranston: T.O.T.A.L. Child.
- Mullen, E. M. (1995). *Mullen scales of early learning: AGS edition*. Circle Pines: American Guidance Service.

Mullen Scales of Early Learning, American Guidance Service Edition

► Mullen Scales of Early Learning

Multidisciplinary Evaluation

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Definition

Multidisciplinary evaluation refers to the process of gathering both formal and informal data from a variety of sources to determine whether a student is eligible for special education services and to provide information about his or her current levels of functioning. “Multidisciplinary” implies that evaluations will be conducted using a team approach in which a number of professionals (e.g.,

speech/language pathologists, occupational therapists, physical therapists, psychologists) gather assessment data about the student.

Historical Background

Prior to the enactment of Public Law 94-142, many students with disabilities were either not being served in public schools or were not receiving appropriate education services. Lack of adequate or appropriate assessments contributed to inaccurate labeling as well as education of students with disabilities. With the support of family associations, the federal government began to develop practices for children with disabilities in the 1950s and 1960s. The Elementary and Secondary Education Act (PL 89-10) and the State Schools Act (PL 89-313), both enacted in 1965, provided states with funding to improve the quality of education for students with disabilities. In 1975, the Education for All Handicapped Children Act, now referred to as IDEA (Individuals with Disabilities Education Act), guaranteed all students with disabilities the right to a free, appropriate public education. Another provision of IDEA included the need for a multidisciplinary assessment of students suspected of having a disability. Using a multidisciplinary approach to assessment, involving a variety of professionals with expertise in each area of need, contributed to more accurate diagnosis of children and the provision of more appropriate special education services for students with disabilities.

Current Knowledge

Developmentally based assessments including information about a child's cognitive, communication, social emotional, and adaptive skills are integral to the diagnosis and decision-making process for those suspected of having an autism spectrum disorder (ASD). Current research supports the need for evaluation of students suspected of having an ASD to take place within a framework that incorporates the knowledge and expertise of various disciplines. In addition to psychological

assessment and evaluation of the student's communication abilities, assessment data from other sources such as occupational therapy or physical therapy are also valuable. Outside of the school setting, pediatric, neurologic, and genetic assessment data are also important (Klin et al. 2005).

Autism spectrum disorders are characterized by atypical development in multiple areas, including cognitive development, communication abilities, social interactions, and play skills. A comprehensive approach to assessment that addresses all areas of concern is therefore necessary. To assess these multiple areas of functioning, a multidisciplinary team of professionals with expertise in these various areas must be involved in the assessment process (Noland and Gabriels 2004).

Future Directions

While a multidisciplinary approach to assessment is essential for children suspected of having an autism spectrum disorder, data from various professionals allows for multiple and, potentially, conflicting views of the child. In order to avoid this possibility, it is recommended that professionals operate in a transdisciplinary manner by which the team collaborates to present a single, coherent picture of the child's strengths and needs. By working together to present this single picture of the child, professionals are able to offer a more accurate view of the child and more fully address the impact of deficits in one area on other areas of functioning (Klin et al. 2005).

References and Reading

- Brock, S. E., Jimerson, S. R., & Hansen, R. L. (2006). *Identifying, assessing, and treating autism at school*. New York: Springer.
- Burnette, J. (2000). *Assessment of culturally and linguistically diverse students for special education eligibility*. Retrieved from http://www.cec.sped.org/Content/NavigationMenu/NewsIssues/TeachingLearningCenter/Assessment_of_Culturally_and_Linguistically_Diverse_Students_for_Special_Education_Eligibility.htm
- Klin, A., Saulnier, C., Tsatsanis, K., & Volkmar, F. R. (2005). Clinical evaluation in autism spectrum

- disorders: Psychological assessment within a transdisciplinary framework. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 772–798). Hoboken: Wiley.
- National Joint Committee on Learning Disabilities. (2010). *Comprehensive assessment of evaluation of students with learning disabilities*. Retrieved from <http://www.idanatl.org/pdf/NJCLD%20Comp%20Assess%20Paper%206-10.pdf>
- Noland, R. M., & Gabriels, R. L. (2004). Screening and identifying children with autism spectrum disorders in the public school system: The development of a model process. *Journal of Autism and Developmental Disorders*, 34(3), 265–277.
- Prelock, P. A. (2006). *Autism spectrum disorder: Issues in assessment and intervention*. Austin: Pro-Ed.
- Turnbull, A., Turnbull, R., & Wehmeyer, M. L. (2010). *Exceptional lives: Special education in today's schools* (6th ed.). Upper Saddle River: Pearson Education.
- United States Department of Education. (2007). *History: Twenty-five years of progress in educating children with disabilities through IDEA*. Retrieved from <http://www2.ed.gov/policy/speced/leg/idea/history.html>

Multidisciplinary Team

► Interdisciplinary Team

Multidisciplinary Training (TEACCH Component)

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Definition

Training of professionals in approaches to intervention in autism has been an integral part of the TEACCH program since its inception in 1972. Training includes a wide range of topics in diagnosis, assessment, treatment, and education and occurs at TEACCH center throughout North Carolina and in multiple sites throughout North

Carolina, other states, and across the world. The training methodologies encompass lectures with audiovisual supports, activities designed for participant experiential learning, and hands-on experience with student trainers (i.e., children, adolescents, and adults with autism). The trainings are led by TEACCH directors (typically a Ph.D. licensed psychologist) and TEACCH staff therapists who have been working in the program for many years and have been trained in the various training models. Over the years, hundreds of professionals from across the world have been trained to be trainers, working as part of a TEACCH-led team.

Historical Background

When TEACCH was established as a statewide mandated program for serving individuals with autism and their families in 1972, there was virtually nothing known about service delivery, evidence-based practices, nor effective ways of preparing professionals to work with this population. There was little interest in autism as a field and more misunderstanding than accurate understanding of the condition. TEACCH grew out of a NIMH-funded project led by Eric Schopler and Robert Reichler which, on a small scale, documented effective ways of supporting children and their parents. With the legislation creating TEACCH at the University of North Carolina came the task of starting three diagnostic and treatment centers, training staff, and putting a model into practice. Simultaneously, 11 classrooms for students with autism were established, 10 of which were in public schools across North Carolina. Most, if not all, of these school districts had never educated a child with autism nor were prepared to recruit and train teachers. TEACCH took on this responsibility and organized a major training program for the new teachers as well as clinical staff at the three regional centers. The first trainings were lengthy, intensive, and complex and were based in a classroom setting with students with autism, a training team, and new teachers and assistant for 2 weeks. Over the course of the 2 weeks, along with lectures and group

discussions, trainees observed the training staff, learned the principles and practices of TEACCH, and gradually assumed responsibility for running the classroom during the second week. Staff to trainee ratio was high, close to one to one. This first model became a template for later variations of the training approach, based on the collaboration of trainer and trainee, experiential learning, and individualization. A major development in the model occurred about a decade later when a team of three TEACCH trainers led by Dr. Gary Mesibov, Director of TEACCH, adapted the earlier model with a less intense trainer to trainee ratio and shortening the process. The curriculum remained the same, but the training methods modified to effectively train more participants. Over the next three decades, there have been other modifications to the core structure of this training model and development of briefer basic training workshops that can reach larger audiences. The efficiency and accessibility of the trainings enable the program to be disseminated across the entire United States and dozens of countries across the world. Coordination of these training programs was led for much of this period by Dr. Roger Cox and implemented by TEACCH directors and staff across North Carolina.

Paralleling the professional service training models has been the center-based training of students at all levels of training and across many disciplines. The centerpiece of this training has been the Clinical Psychology Internship Program, started in 1974, and the Psychology Postdoctoral Training Program, started in the mid-1980s. The internship program is part of the UNC Department of Psychiatry's broad Psychology Internship Program, a program accredited by the American Psychological Association since 1962. Former TEACCH interns have gone onto prominence in the field of autism as leading researchers, clinicians, and program directors. Students receive intensive and comprehensive training in the diagnostic, assessment, and intervention methods used by TEACCH and regardless of their discipline or time commitment to their training experience receive exposure to the basic philosophy and practices of TEACCH, as well as to the field of autism as a whole.

A third area of training, also center-based, is placement of professionals from countries outside the United States who are interested in learning TEACCH methods and practices that can be applied to their home country. These international interns spend anywhere from a month to a year at a TEACCH center learning about all aspects of the program and most have returned to their countries and made significant contributions to services for individuals with autism, their families, and professionals.

Rationale or Underlying Theory

The rationale underlying TEACCH training is based on a key component of its mission statement:

The University of North Carolina TEACCH Autism Program creates and cultivates the development of exemplary community-based services, training programs, and research to enhance the quality of life for individuals with Autism Spectrum Disorder and their families.

TEACCH, while a North Carolina state agency, established to meet the needs of families in the state through direct services is also a significant part of the University of North Carolina and its School of Medicine. The dissemination of its knowledge and experience through training within North Carolina and around the world has been a primary activity since its inception in 1972.

Goals and Objectives

The goals and objectives of the TEACCH multidisciplinary training model are as follows:

1. To disseminate current knowledge, principles, and practices of the comprehensive TEACCH approach to the understanding and treatment of autism.
2. To provide state of the science training through effective and relevant ways to professionals in the field of autism in North Carolina, other states, and internationally.

3. To provide preservice education and training to undergraduate, graduate, and postdoctoral students across disciplines, including, but not limited to, psychology, psychiatry, speech pathology, education, social work, occupational therapy, pediatrics, and family medicine.
4. To provide experiential learning programs to students and professionals on specific tools and strategies, developed by TEACCH, such as structured teaching and the use of visual supports, working with parents as co-therapists, Child Autism Rating Scale (CARS2), Psychoeducational Profile – Third Edition (PEP-3), and TEACCH Transitional Assessment Profile (TTAP).

Treatment Participants

Historically, there have been three types of multidisciplinary training at TEACCH, as described above: in-service training of professionals, preservice training of students, and international interns. These types of training continue today, and each type tends to attract a different group of participants. The in-service training can be further divided into a five-day, intensive training model, which includes hands-on learning with student trainers, and other, more didactic workshops, typically of shorter duration (2–3 days) and with written scenarios to provide an experiential component. Thus far, two studies have examined these training models to evaluate their relative efficacy (Grindstaff 1998, 2000). In 1999, participants of the 5-day model were primarily special educators (55.4%), followed by speech language pathologists (10.9%), psychologists (8.9%), occupational therapists (5.0%), educational consultants (5.0%), parents (4.0%), teaching assistants (3.0%), school administrators (3.0%), and regular education teachers (1.0%). Participants in the comparison group were similar, with special educators in the majority (51.6%), followed by teaching assistants (16.1%), speech language pathologists (9.7%), regular education teachers (6.5%), parents (6.5%), school administrators (3.2%), psychologists (3.2%), and educational consultants (3.2%). An interesting shift has taken place in the last 12 years, such that, in the summer of 2011, the

group of participants in the five-day training model included fewer classroom teachers and a greater number of consultants who provide support to students with autism in regular education settings. Typically, the five-day model has attracted participants from across the United States, as well as a variety of foreign countries. The workshops have tended to attract a more local audience, specific to the region of North Carolina where the workshop has been held.

Preservice training includes students of all levels of training and from a variety of disciplines. Most often, these have been graduate students in psychology, speech language pathology, occupational therapy, and special education. Medical residents and fellows from pediatrics and psychiatry have also been an important subset. The greatest amount of training time and effort has been expended to develop the Clinical Psychology Internship. The TEACCH Intern spends at least 50% of his or her 12-month internship involved at TEACCH in autism-specific diagnosis, assessment, consultation, and intervention. Competition for this internship can be fierce, as applicants come from across the US and Canada; many of them will go on to take leadership positions in the field as researchers, clinicians, and program directors.

International interns have come from a variety of countries; many of them were first exposed to TEACCH principles during a five-day training held either here in the US or in their home country. Collaborations have been particularly rich with professionals from Japan, and more recently, from Sweden and Norway, leading to a greater percentage of international interns from those areas.

Treatment Procedures

The five-day training model is the most intense of the in-service training programs. Participants first attend lectures about the culture of autism and structured teaching to orient them for the week. Subsequently, they spend a small portion each day in lecture; the bulk of their learning takes place in small teams as they observe the TEACCH staff, develop activities for the student trainers, implement these activities with the student trainers, and

reflect on their collective experiences. Throughout the week, the principles of structured teaching are applied to the curriculum areas most relevant to students with autism: informal assessment, communication, independence, leisure skills, social engagement, and behavior management. Teams work with a different student trainer each day, ensuring that they have broad exposure to the diversity of the autism spectrum. The five student trainers are, of course, unique individuals, who also vary by age, cognitive ability, and severity of autism symptoms. Most training participants report that the opportunity to teach in the model classroom and interact with our student trainers is the most memorable component of this training experience.

Workshops vary considerably because they cover a wide range of topics. The Fundamentals Workshop introduces participants to the culture of autism and structured teaching, similar to the first 2 days of the five-day training model. Other workshops focus on the diagnosis of autism spectrum disorders, approaches to social skills instruction, and adapting curriculum for students in regular education, early intervention, and many others. New workshops are developed as the need arises. Most recently, workshops for parents have expanded to address their need for information about upcoming transitions: preparing for kindergarten, middle school, and adulthood. In all cases, an attempt is made to include a component of experiential learning – typically some type of written exercise or thought experiment that provides an opportunity for participants to apply what they are learning to their classroom, client, or child (in the case of parents). It is much less common to be able to provide the “live” interaction with student trainers that is so powerful during the five-day training.

Preservice training is often scheduled to coincide with the academic calendar. Residents or graduate students may observe for as little as 1 day; many future pediatricians, for example, spend a day observing a TEACCH diagnostic evaluation in order to increase their awareness of autism spectrum diagnoses and to facilitate appropriate referrals. Other trainees spend a month, a semester, or an academic year at their local

TEACCH center. Their experiences are tailored to meet their training goals and departmental expectations. As previously mentioned, the TEACCH psychology intern spends a 12-month year in the most intense preservice training. He or she may choose to spend an additional, fellowship year with TEACCH to develop greater independence as a clinician, participate in training efforts, and pursue research interests.

Training for international interns is also individualized, based on the expertise and interest of the intern. Many of these professionals are quite experienced in their discipline and with people with autism; however, they are seeking additional training specific to TEACCH principles and procedures. Often, they hope to acquire enough skill to implement TEACCH methodology in their home country, as well as the ability to communicate TEACCH principles to their colleagues. An intern may be responsible for establishing a new program or modifying an existing one to better reflect TEACCH principles. To that end, interns typically maintain an ongoing relationship with their TEACCH center to receive consultation and, sometimes, additional training.

Efficacy Information

Informal program evaluation takes place during every form of TEACCH training. Participants at all levels of training (in-service, preservice, and international interns) complete rating scales, answer written questions, and are generally encouraged to provide feedback to TEACCH staff. These responses have been universally positive. It is not uncommon for participants to report that their TEACCH training experience has proven more valuable than any other training program in which they have participated. Sometimes, participants also offer constructive criticism. Suggestions are always taken seriously and have sometimes resulted in a helpful revision to the delivery of the training model.

As mentioned above, there have been two formal evaluations of TEACCH’s in-service training efforts, designed to compare the relative efficacy of the five-day training model versus the two-day

Fundamentals Workshops (Grindstaff 1998, 2000). Outcomes were compared across the participants (1) knowledge of the training content, (2) attributions of controllability with regard to the extreme behavior of students with autism, (3) self-efficacy, (4) negative affect, and (5) use of structured teaching. Measures were completed at the very start of the respective training programs and again 6 weeks post-training. Results indicated that both training models were effective in increasing the participants' knowledge of the training content; however, neither model was associated with significant change in participants' attributions, self-efficacy, or affect. Participants in the five-day training model reported a significant increase in their use of structured teaching strategies back in their home environments; workshop participants did not. Grindstaff (2000) concluded that either training model could effectively convey the principles and philosophy of structured teaching but that only the five-day model (with its hands-on component) achieved the more elusive goal of helping participants apply structured teaching strategies to their classroom, clinic, or other work setting. Results from a small pilot study (Grindstaff 2000) indicated that the Structured Teaching Checklist could be used by a trained observer to evaluate the degree to which a classroom has successfully implemented the principles of structured teaching; this tool could prove useful as a more objective measure of training participants' use of structured teaching, as the initial studies relied solely on self-report.

Outcome Measurement

Since its inception, the Structured Teaching Checklist (STC; Grindstaff 2000) has been adapted for use as a standardized measure of TEACCH treatment fidelity (Hume et al. 2011). The TEACCH Fidelity Form has been demonstrated to have strong psychometric properties (e.g., inter-rater agreement, test-retest reliability, and internal consistency) and reliably discriminates between classrooms that adhere to the TEACCH model versus other treatment approaches (Hume et al. 2011). The TEACCH

Fidelity Form is specific to preschool classrooms, but perhaps, it could be elaborated to include classrooms for students at other stages of development, including a version for self-contained classrooms and one for students who are included in regular education settings. Once these additional versions are added, the TEACCH Fidelity Form will make an ideal measure for the effectiveness of TEACCH training.

Qualifications of Treatment Providers

For preservice training programs, there is typically a Ph.D. level psychologist who serves as the administrator and clinical director. Other trainers are often Masters level TEACCH staff who have served with the TEACCH program for a number of years and have demonstrated mastery of the principles of TEACCH training, as well as an ability to communicate effectively. There is a multistep process for becoming a TEACCH trainer that applies to TEACCH staff, as well as interested professionals from other organizations. First, the potential trainer must attend the five-day training model as a participant. He or she then returns as a "shadow trainer" – which means that he or she is apprenticed to an experienced trainer for the five days, with a focus on working with a specific student trainer, instead of the training participants. Shadow training is often repeated two or three times, until the potential trainer feels comfortable with a variety of student trainers. In the final stage of preparation, the potential trainer returns as a "reverse shadow" – which means that he or she now takes the lead not only with the student trainer but also with the training participants, with support from an experienced TEACCH trainer "behind the scenes." The potential trainer may continue as a "reverse shadow" until he or she is comfortable communicating about TEACCH principles and strategies, while also improvising with a student trainer. There is no specific time requirement for the completion of these stages of training, but the commitment is considerable.

In-service trainees and international interns receive supervision from the staff of their assigned

TEACCH center. This always includes the center director, a Ph.D. level psychologist, as well as the Masters level psychoeducational therapists, who come from a variety of disciplines. If there is a TEACCH staff member from the same discipline as the trainee, the two are often matched in a mentor-mentee relationship.

See Also

- ▶ [Informal Assessment](#)
- ▶ [Structured Classrooms](#)
- ▶ [Structured Teaching](#)
- ▶ [Treatment Fidelity](#)
- ▶ [Visual Supports](#)

References and Reading

- Grindstaff, J. P. (1998). *Attributions and autism: An evaluation of TEACCH's experiential training programs*. Unpublished master's thesis, University of North Carolina at Chapel Hill, Chapel Hill.
- Grindstaff, J. P. (2000). *Further evaluation of TEACCH's experiential training programs: Change in participants' knowledge, attributions, and use of structure*. Doctoral dissertation, University of North Carolina at Chapel Hill, Chapel Hill, NC, 2000. *Dissertation Abstracts International-B*, 62, 5374.
- Hume, K., Boyd, B., McBee, M., Coman, D., & Gutierrez, A. (2011). Assessing implementation of comprehensive treatment models for young children with ASD: Reliability and validity of two measures. *Research in Autism Spectrum Disorders*, 5(4), 1430–1440.
- Mesibov, G. B., Shea, V., & Schopler, E. (2004). Training issues. In G. B. Mesibov, V. Shea, & E. Schopler (Eds.), *The TEACCH approach to autism spectrum disorders* (pp. 189–201). New York: Springer.

be in the category of autism, mental retardation, hearing impairment or deafness, visual impairment or blindness, emotional disturbance, speech and language impairment, orthopedic impairment, or health impairment. When referred to as multihandicapped, the person typically has disabilities that are severe enough to require intensive support to handle the functions of daily living. In education, the term “multiple disability” is used for students who have two or more disabilities that effect their ability to access education through traditional means. Children with multiple disabilities have a combination of affected areas such as speech and language, mobility, learning, intellectual functioning, sensory losses or dysfunctions, and behavior or social problems. Children are impacted in different ways, and the disabilities will vary in level of severity and how they manifest in everyday performance. Children with autism may be identified as having multiple disabilities if diagnosed with autism and intellectual disability. Educational support is often needed in the classroom, and some children will need support in all settings throughout their lives. Programming for these children will be based on the characteristics they display which will vary widely depending on the areas involved. Typically, the educational team includes a variety of professionals such as special education and regular education teachers, physical therapists, occupational therapists, speech and language pathologist, adaptive physical education teachers, nurses, paraprofessionals, psychologist, and possibly medical professionals.

Historical Background

The term “multi” means many or several and is used frequently in numerous contexts. The term handicap has a more involved history, some of which is not based on facts that can be substantiated. There is a story that is associated with the word “handicap” that leads people to believe it was first used when people with disabilities were allowed to beg in the streets with their “cap in hand.” There have been several attempts to dispel this myth but it seems to live on. The word does have a connection to a competitive sport dating

Multihandicapped

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Definition

Multihandicapped refers to a person that has more than one disabling condition. The disability may

back to 1650s but was not used in conjunction with a person with a disability until the early 1900s. As the term “handicap” evolved, it now refers to a person with a disability. The term alone does not give information as to what type of disability a person may have. Once multi was added to handicap, it came to have the meaning we know today of more than one disabling condition which makes functioning in daily activities more difficult when compared to a person without the disabilities.

See Also

► Disability

References and Reading

- Amundson, R. (2011). About the meaning of “handicap”. Retrieved July 26, 2011, from <http://www.uhh.hawaii.edu/~ronald/HandicapDefinition.htm>.
- Klewe, L. (1993). Brief report: An empirical evaluation of spelling boards as a means of communication for the multihandicapped. *Journal of Autism and Developmental Disorders*, 23(3), 559–566.
- Miller, G. (1993). Expanding vocational options. *Review*, 25(1), 27.
- NICHCY. (2004). *Disability fact sheet number 10*. Washington, DC: Author. Retrieved July 2011, from <http://www.accessiblesociety.org/topics/demographics-identity/dkaplanpaper.htm>.
- Reid, D., Phillips, J. F., & Green, C. W. (1991). Teaching persons with profound multiple handicaps: A review of the effects of behavioral research. *Journal of Applied Behavior Analysis*, 24(2), 319–336.

locus) – this can imply multiple disease variants acting simultaneously in each patient, or single mutations in each patient, but in different genes (unsure of this definition). Tuberous sclerosis, for example, can be caused by mutations in either TSC1 or TSC2 (EntrezGene, OMIM). Coronary artery disease, on the other hand, could be due to more than one gene working in concert (e.g., Saade et al. 2011).

Some ASD cases are attributable to specific chromosomal abnormalities, or to deletions/rearrangements known to induce autism (when these rearrangements confer other symptoms and phenotypes in addition to autism, they are known as “syndromic” autism). No one cause has been found that explains more than 1–2% of autism cases (gross chromosomal abnormalities). A recent review summarized that taken together, all gross chromosomal abnormalities, all syndromes associated with autism, disruptions to known autism loci (e.g., SHANK3), and rare variants in candidate genes account for only 12–17% of autism cases, meaning that the majority of autism cases remain unclear with regard to specific genetic etiology (Buxbaum 2009). Another estimate put it at 5–15% (Pinto et al. 2010). Additionally, even syndromic causes that associate strongly with autism, such as fragile X syndrome, are not completely sufficient to induce autism; there exist fragile X patients who do not present with autism or ASD. Though some cases are largely influenced by highly deleterious single-gene mutations, even these are likely modulated by mutations in other genes. Similar ASD phenotypes need not necessarily involve the exact same genes in each case; it is possible that many autism cases can be caused by any one of a number of combinations of different genes in neurological pathways.

Multilocus Genetic Models

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Definition

A multilocus genetic model means that a given phenotypic outcome is influenced by multiple genes (an individual gene is referred to as a

Historical Background

Modern understanding of genetic inheritance is widely accepted to have begun with Gregor Mendel and his experiments with trait inheritance in garden peas. What we call Mendelian inheritance is the simplest form of transmission of genetic information: one gene, two or more alleles, a

relationship between the alleles (dominant, recessive, codominant, incompletely dominant, etc.). Sex-linked traits modify the situation somewhat; disruptive mutations on the X-chromosome that may be recessive in females, being offset by the healthy X-chromosome, can be deleterious in males, who have only one X-chromosome. Multilocus disease models, while always theoretically possible, became more discussed in the late 1970s and early 1980s, particularly in the context of autoimmune diseases (see Rotter and Landaw 1984). A consensus for autism as a primarily genetic disorder did not fully emerge until the late 1980s to mid-1990s (Bailey et al. 1995; Bolton et al. 1994; Smalley et al. 1988; Szatmari et al. 1998), and fairly quickly, it became apparent that single-gene inheritance models did not explain most autism cases (e.g., Jorde et al. 1991). An early multilocus model of family recurrence in autism determined that 3 interacting genes, in a range of 2–10, best accounted for their data. Another multilocus model was put forth by Risch et al. in 1999. They concluded that on the basis of evidence seen in 97 affected sibling pairs (ASPs; both siblings diagnosed with autism) and 51 discordant sibling pairs (DSPs; one sibling had autism and one was unaffected), as well as follow-up in 50 ASPs and 29 DSPs, one or a few mutated gene loci in a given sibling pair did not adequately explain the increase in shared DNA inheritance between the affected siblings relative to the discordant siblings. On the basis of the families they assessed, the authors determined that a model based on 15 or more interacting gene loci explained their data better than a model with 10 or fewer gene loci (Risch et al. 1999).

As autism genetic models were being debated, a broader debate was emerging in the genetic field about the etiology of complex (multigenic), relatively common diseases. The common disease-common variant theory held that for a disease to remain relatively common in the population, the mutations being inherited must not be damaging enough, on their own, to reduce reproductive success of those carrying them, and therefore more severe common disease phenotypes must be due to the interaction of

many common variants, each of a small effect size (contributing a small amount to the disease phenotype, and not disruptive enough to reduce reproductive fitness on its own) but significant enough when inherited in combination to induce a disease state. In contrast, the common disease-rare variant theory held that multiple rare variants, each of larger effect (more disruptive), were sufficient to cause common diseases. These deleterious rare variants would in theory be more prone to reducing reproductive success, but if many of these rare mutations were being generated spontaneously (*de novo*) rather than inherited from parents, in a large enough number of genes, it could explain the baseline prevalence of even severely disruptive alleles.

As evidence accumulated that methodologies driven by the common disease-common variant assumption (exemplified by genome-wide association studies) could account for only a modest proportion of common complex disorders, the common disease-rare variant theory gained traction in research on these disorders. Autism in particular was poorly accounted for by genetic methods seeking to associate common variation with the disorder (e.g., Anney et al. 2010; Curran et al. 2011; Hirschhorn and Gajdos 2011). A few implicated genes and regions were replicated in other studies (e.g., *MET*, *CNTNAP2*, and *5p14.1*) (Alarcon et al. 2008; Arking et al. 2008; Campbell et al. 2010; Jackson et al. 2009; Sousa et al. 2009; Wang et al. 2009; Weiss et al. 2009); however, common variants overall likely account for a small proportion of autism heritability (Pinto et al. 2010). Rare copy number variants (Bucan et al. 2009; Pinto et al. 2010; Sanders et al. 2011) began to gain attention as several studies suggested a role in autism. With the emergence of dense genome-wide marker SNP genotyping as well as exome sequencing, and follow-ups on these studies, evidence accumulated for a multihit etiology (i.e., mutations in multiple genes in the same person) in at least some autism cases, for example, *FOXP1* and *CNTNAP2* (O’Roak et al. 2011), *CNTNAP5* and *DOCK4* (Pagnamenta et al. 2010), and *DMD* and *TRPM3* (Pagnamenta et al. 2011). The emerging methods allow us to concretely demonstrate the multilocus

model in patient data, beyond just discussing its theoretical fit to the existing data.

Current Knowledge

The current state of the field is that large-scale chromosome rearrangements, genetic syndromes, and the few known definitively autism-associated loci (e.g., SHANK3, 15.q11-13) are of large effect size, but account for a modest proportion of total autism cases, and likely in conjunction with other genes in a given case. The majority of cases are likewise caused by mutations in a variety of genes, the list of which researchers are constantly working to validate, corroborate, and expand. The challenge is no longer to demonstrate the validity of the theoretical underpinnings of the multilocus model but to use more and more precise genetic tools to establish exactly what genes, and perhaps networks of genes (e.g., gene families or biological pathways), are involved in autism.

Future Directions

As the prices of whole-exome and whole-genome resequencing continue to drop, these technologies, combined with wider- and wider scale genotyping of SNP markers across the entire genome, it will be possible to analyze patients with autism at a finer and finer scale, potentially uncovering new viable candidate genes and contributing mutations, and strengthening or weakening the cases for existing candidate genes as we examine larger numbers of people in greater detail than ever before. These thorough analyses of individuals and their families represent the emerging present and near future in autism research.

See Also

- [Common Disease-Common Variant Hypothesis](#)
- [Common Disease-Rare Variant Hypothesis](#)

References and Reading

- Alarcon, M., Abrahams, B. S., Stone, J. L., Duvall, J. A., Perederiy, J. V., Bomar, J. M., et al. (2008). Linkage, association, and gene-expression analyses identify CNTNAP2 as an autism-susceptibility gene. *American Journal of Human Genetics*, 82(1), 150–159.
- Anney, R., Klei, L., Pinto, D., Regan, R., Conroy, J., Magalhaes, T. R., et al. (2010). A genome-wide scan for common alleles affecting risk for autism. *Human Molecular Genetics*, 19(20), 4072–4082.
- Arking, D. E., Cutler, D. J., Brune, C. W., Teslovich, T. M., West, K., Ikeda, M., et al. (2008). A common genetic variant in the neurexin superfamily member CNTNAP2 increases familial risk of autism. *American Journal of Human Genetics*, 82(1), 160–164.
- Bailey, A., Le Couteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda, E., et al. (1995). Autism as a strongly genetic disorder: Evidence from a British twin study. *Psychological Medicine*, 25(1), 63–77.
- Bolton, P., Macdonald, H., Pickles, A., Rios, P., Goode, S., Crowson, M., et al. (1994). A case-control family history study of autism. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 35(5), 877–900.
- Bucan, M., Abrahams, B. S., Wang, K., Glessner, J. T., Herman, E. I., Sonnenblick, L. I., et al. (2009). Genome-wide analyses of exonic copy number variants in a family-based study point to novel autism susceptibility genes. *PLoS Genetics*, 5(6), e1000536.
- Buxbaum, J. D. (2009). Multiple rare variants in the etiology of autism spectrum disorders. *Dialogues in Clinical Neuroscience*, 11(1), 35–43.
- Campbell, D. B., Warren, D., Sutcliffe, J. S., Lee, E. B., & Levitt, P. (2010). Association of MET with social and communication phenotypes in individuals with autism spectrum disorder. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 153B(2), 438–446.
- Curran, S., Bolton, P., Rozsnyai, K., Chiacchetti, A., Klauck, S. M., Duketis, E., et al. (2011). No association between a common single nucleotide polymorphism, rs4141463, in the MACROD2 gene and autism spectrum disorder. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 156B(6), 633–639.
- Hirschhorn, J. N., & Gajdos, Z. K. (2011). Genome-wide association studies: Results from the first few years and potential implications for clinical medicine. *Annual Review of Medicine*, 62, 11–24.
- Jackson, P. B., Boccuto, L., Skinner, C., Collins, J. S., Neri, G., Gurrieri, F., et al. (2009). Further evidence that the rs1858830 C variant in the promoter region of the MET gene is associated with autistic disorder. *Autism Research*, 2(4), 232–236.
- Jorde, L. B., Hasstedt, S. J., Ritvo, E. R., Mason-Brothers, A., Freeman, B. J., Pingree, C., et al. (1991). Complex segregation analysis of autism. *American Journal of Human Genetics*, 49(5), 932–938.

- O'Roak, B. J., Derziotis, P., Lee, C., Vives, L., Schwartz, J. J., Girirajan, S., et al. (2011). Exome sequencing in sporadic autism spectrum disorders identifies severe de novo mutations. *Nature Genetics*, 43(6), 585–589.
- Pagnamenta, A. T., Bacchelli, E., de Jonge, M. V., Mirza, G., Scerri, T. S., Minopoli, F., et al. (2010). Characterization of a family with rare deletions in CNTNAP5 and DOCK4 suggests novel risk loci for autism and dyslexia. *Biological Psychiatry*, 68(4), 320–328.
- Pagnamenta, A. T., Holt, R., Yusuf, M., Pinto, D., Wing, K., Betancur, C., et al. (2011). A family with autism and rare copy number variants disrupting the Duchenne/Becker muscular dystrophy gene DMD and TRPM3. *Journal of Neurodevelopmental Disorders*, 3(2), 124–131.
- Pinto, D., Pagnamenta, A. T., Klei, L., Anney, R., Merico, D., Regan, R., et al. (2010). Functional impact of global rare copy number variation in autism spectrum disorders. *Nature*, 466(7304), 368–372.
- Risch, N., Spiker, D., Lotspeich, L., Nouri, N., Hinds, D., Hallmayer, J., et al. (1999). A genomic screen of autism: Evidence for a multilocus etiology. *American Journal of Human Genetics*, 65(2), 493–507.
- Rotter, J. I., & Landaw, E. M. (1984). Measuring the genetic contribution of a single locus to a multilocus disease. *Clinical Genetics*, 26(6), 529–542.
- Saade, S., Cazier, J. B., Ghassibe Sabbagh, M., Youhanna, S., Badro, D. A., Kamatani, Y., et al. (2011). Large scale association analysis identifies three susceptibility loci for coronary artery disease. *PLoS One*, 6(12), e29427.
- Sanders, S. J., Ercan-Sencicek, A. G., Hus, V., Luo, R., Murtha, M. T., Moreno-De-Luca, D., et al. (2011). Multiple recurrent de novo CNVs, including duplications of the 7q11.23 Williams syndrome region, are strongly associated with autism. *Neuron*, 70(5), 863–885.
- Smalley, S. L., Asarnow, R. F., & Spence, M. A. (1988). Autism and genetics: A decade of research. *Archives of General Psychiatry*, 45(10), 953–961.
- Sousa, I., Clark, T. G., Toma, C., Kobayashi, K., Choma, M., Holt, R., et al. (2009). MET and autism susceptibility: Family and case-control studies. *European Journal of Human Genetics*, 17(6), 749–758.
- Szatmari, P., Jones, M. B., Zwaigenbaum, L., & MacLean, J. E. (1998). Genetics of autism: Overview and new directions. *Journal of Autism and Developmental Disorders*, 28(5), 351–368.
- Wang, K., Zhang, H., Ma, D., Bucan, M., Glessner, J. T., Abrahams, B. S., et al. (2009). Common genetic variants on 5p14.1 associate with autism spectrum disorders. *Nature*, 459(7246), 528–533.
- Weiss, L. A., Arking, D. E., Daly, M. J., Chakravarti, A., & Gene Discovery Project of Johns Hopkins & the Autism Consortium. (2009). A genome-wide linkage and association scan reveals novel loci for autism. *Nature*, 461(7265), 802–808.

Multiple Complex (or Multiplex) Developmental Disorder

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Definition

The greatly missed Cohen et al. (1986) thought we should try and specify subgroups within the so-called not otherwise specified areas within the broader category of pervasive developmental disorders. From cluster analyses of a big group of individuals with atypical development, within the area on the fringe of autistic disorders, two categories emerged: Asperger's disorder (that was added to DSM IV 1994) and the group so-called multiplex developmental disorders (later renamed multiple complex developmental disorder to avoid confusion with MDD – “major depressive” disorder).

In this entry, this “heuristic” category is described.

Historical Background

Along with defined categories from the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition, text revision (DSM-IV-TR) includes so-called Not Otherwise Specified (NOS) categories meant for lesser variants or borderline conditions to the clearly defined cases.

The NOS subcategory for pervasive developmental disorder (PDD-NOS) was introduced in DSM-III-R (1986). Cohen et al. (1986) were concerned that having only two categories for autism in DSM-III-R would be too few to provide a more fine-tuned description of subgroups within what is now commonly named the autistic spectrum. These subgroups had been identified by

on-cluster analyses on large groups of children with “atypical” behavior (Prior et al. 1975; Dahl et al. 1986).

Cohen et al. suggested that two new categories should be introduced in DSM-IV. The first one would be Asperger’s disorder (Asperger 1944; Wing 1981), referring to individuals with developmental problems in the areas of social reciprocity, communication, and rigid and restricted patterns of behavior. These individuals differed from the classical Kanner autism in that they developed language at typical stages (yet not the adequate pragmatics), and they had motor clumsiness and intellectual preoccupations more than motor stereotypies. This category was finally included in DSM-IV, but the criteria did not include many of the features Asperger actually described (Miller and Ozonoff 1997).

The second proposal (Cohen et al. 1986, 1991) on multiple complex developmental disorders (MCDD) was not included in DSM-IV. It was a heuristic proposal aimed at promoting research on a category well known in clinical practice (Green and Jones 1998) and clearly emerging as a distinct cluster in the analysis of the children with atypical developmental (Dahl et al. 1986). These children had previously been described under different diagnostic labels, including “borderline children (Bemporad et al. 1982; Pine 1974; Vela et al. 1983), schizoid personality in childhood (Wolff 2003), and schizotypal children (Nagy and Szatmari 1986). Ironically, it had been included in DSM-III (American Psychiatric Association [APA] 1980) with the label “Childhood-Onset Pervasive Developmental Disorder” but, for reasons that are not clear, was not continued into DSM-III-R and thus not considered in DSM-IV.

All these labels recognized children who could be placed “in the borderlands of autism.” They share with more classically autistic children a lack of social sensitivity and empathy, but yet differ importantly from autistic children. Where children with “classic Kanner autism” lack imagination, these atypical children tend to get carried away by a far too-vivid imagination that blurs their reality testing.

Current Knowledge

The defining criteria for MCDD include (1) consistently impaired social behavior and sensitivity, (2) impaired regulation of affective state, and (3) impaired cognitive processing “thinking disorder.”

The discriminative potential of these criteria in categorizing these children reaches sensitivity and specificity levels comparable to those for autistic disorder (Buitelaar and van der Gaag 1998) (Table 1). The face validity of the concept was demonstrated in a series of independent studies (Towbin et al. 1993; Van der Gaag et al. 1995). The predictive validity is high (Van der Gaag 1993). There is longitudinal persistence of symptoms in the area of development of social reciprocity and thinking disorders. The extreme problems in the regulation of affective states are less prominent in adolescents and adults. There is a marked shift toward symptoms from the schizophrenia spectrum in adults, with up to 17% of the cases meeting criteria of schizophrenia after one or several psychotic episodes and over 60%

Multiple Complex (or Multiplex) Developmental Disorder, Table 1 Criteria for multiple complex developmental disorder (Cohen et al. 1994 – van der Gaag et al. 2005)

- | |
|---|
| 1. Impaired regulation of states of mind and anxieties |
| A. Unusual or peculiar fears and phobias or frequent idiosyncratic or bizarre anxiety reactions |
| B. Recurrent panic episodes or flooding with anxiety |
| C. Episodes of behavioral disorganization punctuated by markedly immature, primitive, or violent behavior |
| 2. Impaired social behavior |
| A. Social disinterest, detachment, avoidance, or withdrawal |
| B. Markedly disturbed and/or ambivalent attachments |
| 3. The presence of thought disorder |
| A. Irrationality, magical thinking, sudden intrusion on normal thought processes, bizarre ideas, neologisms, or repetitions of nonsense words |
| B. Perplexity and easy confusability |
| C. Overvalued ideas, including fantasies of omnipotence, paranoid preoccupations, overengagement with fantasy figures, and referential ideation |

Note. A total of five (or more) items from (1), (2), and (3), with at least one item from (1), one item from (2), and one item from (3) (Buitelaar and van der Gaag 1998)

meeting the criteria for schizoid or schizotypal personality disorder (Van Engeland and Van der Gaag 1994). Other differences come from studies on the stress-regulation characteristics in the MCDD group (Jansen et al. 2003; Kemner et al. 1999) that show marked differences in these areas between individuals with autistic disorder and individuals with MCDD who, on these dimensions, are more similar to adults within the schizophrenia spectrum. The third characteristic of MCDD children includes “disordered thinking,” which is commonly thought to be specific to schizophrenia.

Future Directions

In DSM V, the search for specific subgroups will be dropped. But research should be pursued along the dimensions of impaired regulation of affective states and thought disorder in individuals with autism spectrum disorders. This research will be important for a well-suited guidance and treatment program and to explore the boundaries between autism spectrum and schizophrenia spectrum disorders as apparently at least a portion of high functioning individuals with autism spectrum disorders are at risk of having psychotic episodes in their adult life and for developing schizophrenia and/or addictive behavior.

See Also

- [Asperger's Disorder](#)
- [Psychosis](#)
- [Schizophrenia](#)

References and Reading

- Buitelaar, J. K., & van der Gaag, R. J. (1998). Diagnostic rules for children with PDD-NOS and multiple complex developmental disorder. *Journal of Child Psychology and Psychiatry*, 39(6), 911–919.
- Cohen, D. J., Volkmar, F. R., & Paul, R. (1986). Issues in the classification of pervasive developmental disorders: History and current status of nosology. *Journal of the American Academy of Child & Adolescent Psychiatry*, 25(2), 158–161.
- Dahl, E. K., Cohen, D. J., & Provencher, S. (1986). Clinical and multivariate approaches to the nosology of pervasive developmental disorders. *Journal of the American Academy of Child & Adolescent Psychiatry*, 25(2), 170–180.
- de Bruin, E. I., de Nijs, P. F., Verheij, F., Hartman, C. A., & Ferdinand, R. F. (2007). Multiple complex developmental disorder delineated from PDD-NOS. *Journal of Autism and Developmental Disorders*, 37(6), 1181–1191.
- Herba, C. M., de Bruin, E., Althaus, M., Verheij, F., & Ferdinand, R. F. (2008). Face and emotion recognition in MCDD versus PDD-NOS. *Journal of Autism and Developmental Disorders*, 38(4), 706–718.
- Jansen, L. M., Gispen-de Wied, C. C., Van der Gaag, R. J., ten Hove, F., Willemsen-Swinkels, S. W., Harteveld, E., et al. (2000). Unresponsiveness to psychosocial stress in a subgroup of autistic-like children, multiple complex developmental disorder. *Psychoneuroendocrinology*, 25(8), 753–764.
- Jansen, L. M., Gispen-de Wied, C. C., van der Gaag, R. J., & van Engeland, H. (2003). Differentiation between autism and multiple complex developmental disorder in response to psychosocial stress. *Neuropsychopharmacology*, 28(3), 582–590.
- Klin, A., Mayes, L. C., Volkmar, F. R., & Cohen, D. J. (1995). Multiplex developmental disorder. *Journal of Developmental & Behavioral Pediatrics*, 16(Suppl. 3), S7–S11.
- Lahuis, B. E., Durston, S., Nederveen, H., Zeegers, M., Palmen, S. J., & Van Engeland, H. (2008). MRI-based morphometry in children with multiple complex developmental disorder, a phenotypically defined subtype of pervasive developmental disorder not otherwise specified. *Psychological Medicine*, 38(9), 1361–1367.
- Lahuis, B. E., Van Engeland, H., Cahn, W., Caspers, E., Van der Geest, J. N., Van der Gaag, R. J., et al. (2009). Smooth pursuit eye movement (SPEM) in patients with multiple complex developmental disorder (MCDD), a subtype of the pervasive developmental disorder. *World Journal of Biological Psychiatry*, 10(4) Pt. 3, 905–912.
- Lincoln, A. J., Bloom, D., Katz, M., & Boksenbaum, N. (1998). Neuropsychological and neurophysiological indices of auditory processing impairment in children with multiple complex developmental disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 37(1), 100–112.
- Sprong, M., Becker, H. E., Schothorst, P. F., Swaab, H., Ziermans, T. B., Dingemans, P. M., et al. (2008). Pathways to psychosis: A comparison of the pervasive developmental disorder subtype multiple complex developmental disorder and the “at risk mental state”. *Schizophrenia Research*, 99(1–3), 38–47.
- Towbin, K. E., Dykens, E. M., Pearson, G. S., & Cohen, D. J. (1993). Conceptualizing “borderline syndrome of childhood” and “childhood schizophrenia” as a developmental disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 32(4), 775–782.
- Van der Gaag, R. J., Buitelaar, J., Van den Ban, E., Bezemer, M., Nijio, L., & Van Engeland, H. (1995).

A controlled multivariate chart review of multiple complex developmental disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 34(8), 1096–1106.

van der Gaag, R. J., Caplan, R., van Engeland, H., Loman, F., & Buitelaar, J. K. (2005). A controlled study of formal thought disorder in children with autism and multiple complex developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 15(3), 465–476.

Multiple Exemplar Instruction (MEI)

► Multiple Exemplar Training

Multiple Exemplar Training

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Synonyms

Multiple exemplar instruction (MEI); General case teaching, general case instruction

Definition

Multiple exemplar training (MET), which is also known as multiple exemplar instruction or general case teaching, is a term in applied behavior analysis (ABA) for the use of related stimuli samples during instruction to increase a person's ability to respond to novel, untrained stimuli (Greer et al. 2005). For example, teaching a child to say “lime” when shown various examples of limes (i.e., photographs, illustrations, physical objects) increases the likelihood that the child will be able to say “lime” when he/she is later shown an untrained image of a lime that possesses some characteristics related to previously taught exemplars. The ability

for an individual to deduce that two similar stimuli are related or the same, such as labeling untrained images of limes, is crucial to engage in generative behavior (the ability to “generate” novel behaviors based on previously learned stimuli). It's worth noting that learning both physical and arbitrary relations may contribute to generative behaviors. That is, like learning to label a “lime,” one can also learn to label a lime with arbitrary labels, such that when asked, “what shape is this?” or “what color is this fruit?”, one answers “round/green.” Successful generative responses may occur when interpretation of context-specific stimuli occurs, in which many cases involve a combination of both physical and arbitrary conditions such that when one is asked, “give me a round, green, fruit,” one can deduce that the speaker is likely asking for a lime (Holth 2017).

The term “multiple exemplar training” was first coined by Hughes and Rusch (1989) in a study that taught people with intellectual disabilities to solve work-related problems using MET and self-instruction (Holth 2017). Although the term MET did not appear until 1989, there are multiple records in which MET teaching strategies were implemented before the term was conceived. For instance, a study in 1967 noted an increase in correct responding to other, untrained imitations after teaching and reinforcing various models of imitation (Baer et al. 1967). The use of MET to teach children with autism spectrum disorder (ASD) first emerged in a study that taught four high school students with ASD to initiate and sustain social interactions with neurotypical developing peers through a “multiple exemplar strategy” (Breen et al. 1985). People with autism often have difficulty engaging in generative behaviors which tends to result in limited responses for a wide variety of situations (Schnell et al. 2018). Because the use of generative behaviors is imperative for adapting to an environment with varying stimuli, and given that people with ASD often have difficulty naturally learning to respond to varying stimuli, MET is often used as a teaching strategy in ABA to assist people with ASD learn to emit generative behaviors and promote the maintenance of skills (Greer et al. 2005).

MET studies have noted the emergence of two MET teaching variations, serial MET (S-MET), in

which exemplars are introduced one at a time, and concurrent MET (C-MET), in which a group of exemplars are all introduced at the same time (Schnell et al. 2018). Although MET studies have successfully taught a variety of skills and related generative responses, such as expressive labeling, imitation, listener responding, behavioral variability, matching, social behavior, and responding to wh-questions (Holth 2017; Schnell et al. 2018), contrasting findings exist between studies regarding which type of MET strategy is more efficient (Schnell et al. 2018). A study by Schnell, Vladescu, Kodak, and Nottingham compared the mean training time, total training time, exposure to mastery, and training sessions to mastery between S-MET and C-MET when teaching expressive labeling and observed that S-MET had the lowest mean training time and total training time, which differed from previous studies that indicated C-MET is a more efficient strategy (Schnell et al. 2018). Be that as it may, more research should be conducted to replicate the efficacy of MET teaching variations since there are currently two studies that have examined the efficacy of MET regarding expressive labeling tasks. Researchers also stressed the importance of expanding MET efficacy research for teaching other tasks, such as comparing efficacy of S-MET and C-MET when applied to imitation tasks, listener responding tasks, and other types of behaviors. In addition, future studies should focus on evaluating whether MET teaching strategies are more effective when individualized to the students. For example, Schnell et al. (2018) noted that, despite S-MET had the lowest mean training time, efficiency varied between students and across teaching strategies. In other words, all the participants were able to master exemplars the fastest with the use of S-MET, but each participant's performance varied in both time and number of exposures to mastery.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Generalization and Maintenance](#)
- [Stimulus Control](#)

References and Reading

- Baer, D. M., Peterson, R. F., & Sherman, J. A. (1967). The development of imitation by reinforcing behavioral similarity to a model. *Journal of the Experimental Analysis of Behavior*, 10, 405–416. <https://doi.org/10.1901/jeab.1967.10-405>.
- Breen, C., Haring, T., Pitts-Conway, V., & Gaylord-Ross, R. (1985). The training and generalization of social interaction during breaktime at two job sites in the natural environment. *The Association for Persons with Severe Handicaps*, 10, 41–50. <https://doi.org/10.1177/154079698501000105>.
- Greer, R. D., Yaun, L., & Gautreaux, G. (2005). Novel dictation and Intraverbal responses as a function of a multiple exemplar instructional history. *The Analysis of Verbal Behavior*, 21, 99–116. <https://doi.org/10.1007/bf03393012>.
- Holth, P. (2017). Multiple exemplar training: Some strengths and limitations. *Behavior Analyst*, 40, 225–241. <https://doi.org/10.1007/s40614-017-0083-z>.
- Hughes, C., & Rusch, F. R. (1989). Teaching supported employees with severe mental retardation to solve problems. *Journal of Applied Behavior Analysis*, 22, 365–372. <https://doi.org/10.1901/jaba.1989.22-365>.
- Schnell, L. K., Vladescu, J. C., Kodak, T., & Nottingham, C. L. (2018). Comparing procedures on the acquisition and generalization of facts for children with autism spectrum disorder. *Journal of Applied Behavior Analysis*, 51, 769–783. <https://doi.org/10.1002/jaba.480>.

Multiple Exemplar Video Modeling and Unscripted Social Communication

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Definition

Multiple exemplar video modeling involves the creation and presentation of several videos depicting multiple response options and varied stimuli (e.g., setting, materials) of desired

behaviors to promote response and stimulus generalization.

Historical Background

Based on social learning theory (Bandura 1977), video modeling (VM) is a highly effective intervention used to teach new behaviors through imitation of a model presented via video technology. VM is considered an evidence-based practice by the National Autism Center (NAC 2015) and the National Professional Development Center on Autism Spectrum Disorder (NPDC; Wong et al. 2015). Video modeling researchers have reported the following inherent factors in VM that may produce positive feasibility and acceptability: (a) although recording and editing videos can take time, once these materials are made, their repeated use is infinite; (b) the increasing availability and usability of digital video equipment increase their probable use (Ayres and Langone 2005); (c) videos are often presented in socially acceptable technologies such as iPods and iPads that do not limit the individual's opportunities to interact with peers (Shane et al. 2012); and (d) researchers have also reported that VM is cost- and time-effective (Charlop-Christy et al. 2000). Though none of these factors have been empirically evaluated, they may influence the likelihood that VM interventions are delivered in natural settings (e.g., school, home, and community), which may contribute to the long-term effectiveness of VM.

Researchers and practitioners have evaluated and implemented VM to teach scripted play verbalizations to children with autism spectrum disorder (D'Ateno et al. 2003; MacDonald et al. 2005; MacManus et al. 2015) and have involved typically developing peers as part of the intervention (Joint Video Modeling; Dueñas et al. 2019; MacDonald et al. 2009; Maione and Mirenda 2006). Despite positive social communication outcomes for children with ASD reported in the extant VM literature, little is known about the generality of social communication repertoires acquired following brief periods of VM exposure, as few studies measure generalization effects

(Bellini and Akullian 2007; Goldstein et al. 2014; Sutton et al. 2018). This is an important direction for future research as the aim of social communication interventions for children with ASD is the generalization of acquired social communication behaviors across people, settings, and contexts that have social and applied value. That is, the degree to which a skill generalizes can have crucial implications over the utility of the skill for a child with ASD.

For individuals with ASD who struggle to generalize acquired social communication, it is necessary to program generalization in instructional or treatment arrangements. There are numerous recommended strategies for promoting generalization, primarily stemming from reviews of behavior-analytic literature (Haring 1988; Stokes and Baer 1977; Stokes and Osnes 1989). One of these strategies is to program multiple exemplars. Programming various exemplars involves providing various examples for responding until untrained responses emerge.

Current Knowledge

Preliminary research in VM has shown that varying the social communication behaviors modeled in videos during social interactions may evoke several potential responses when presented with similar contexts (see Dueñas et al. 2019; MacManus et al. 2015; Maione and Mirenda 2006; Plavnick and Dueñas 2018). Programming multiple exemplars is an instructional strategy that may promote the acquisition of repertoires as opposed to rote responses (Stokes and Baer 1977). This focus may help children with ASD to demonstrate more natural social communication across varied contexts (Kasari et al. 2016). Novel social communication skills are a desired focus of social interactions between children with ASD and their peers, as predictability and rote responding are less typical among children who are not diagnosed with ASD.

Video modeling is an intervention that can be used for programming several generalization strategies that promote response and stimulus generalization. For example, recording several

videos to model multiple response options for promoting response generalization (i.e., multiple exemplar video modeling). Dueñas et al. (2019) demonstrated that multiple exemplar video modeling was a promising intervention for teaching scripted and *unscripted* social communication during pretend play with typically developing peers in an inclusive early childhood setting. This multicomponent intervention combined video modeling with peer-mediated intervention. Video models were presented to both children prior to play sessions as a way to prompt children to engage in verbalizations during pretend play interactions. The procedure involved an initial invitation from the typically developing peer to invite the child with ASD to play. The children were seated at a small table side by side according to the roles they were imitating in the video. The iPad was presented on the table directly in the middle of both children, and they were exposed to the audio in the video using headphones with a splitter. The video was shown twice, and the researcher ensured their bodies were positioned in front of the screen and their faces were directed to the screen. Three video exemplars were rotated across sessions to ensure equal number of presentations for each video exemplar. The children were instructed to “Do and say what you saw in the video” and were given 3 min to play with the toys that were set up at the table. For a sample of video scripts, see Table 1. Although children with ASD increased scripted and unscripted verbalizations toward peers, participants with ASD in the study all responded differently to the intervention. Dueñas et al. (2019) hypothesized that as children with ASD began to deviate from scripts, typically developing peers struggled to respond consistently.

Researchers can also record various people to model social behaviors (e.g., peers, teachers, and parents), using portable electronic devices that optimize implementation across settings (e.g., home, community, classrooms). For example, in Playnick and Dueñas (2018), researchers evaluated the effects of video-based group instruction on spontaneous social questions and comments of four adolescents with ASD. The researchers created three variations of video clips for five

Multiple Exemplar Video Modeling and Unscripted Social Communication, Table 1 Sample video script

Objects	Scripted play actions	Scripted verbalizations
Mom bunny	Holds bunny and jumps up	“Dinner’s ready”
Sister bunny	Holds bunny and moves downstairs	“I’m coming”
Mom bunny	Moves bunny to table	“Made your favorite”
Sister bunny	Sits bunny on chair	“What is it?”
Mom bunny	Has bunny put two plates on table	“It’s pizza”
Sister bunny	Has bunny knock down table	“No, my pizza”
Mom bunny	Has bunny set table	“I got it”
Sister bunny	Moves bunny to chair	“Thanks, mom”

different social behaviors. The videos varied in terms of models, materials used, and the vocal statements modeled in the videos. All participants demonstrated an increase in spontaneous social questions and comments.

Future Directions

Although preliminary research shows promise in promoting generalized responding, future research is needed to determine whether a relationship exists between multiple exemplar video modeling and novel social communication. That is, precise procedures that lead to novel social communication continue to be unknown. Specifically, whether typically developing peer involvement, multiple exemplar videos, or both led to children with ASD’s deviation from scripts. In addition, when designing multiple exemplar video modeling procedures, several questions remain, for example, What is the ideal number of video exemplars?, What is the amount of video exposure needed and is this skills- and/or student-dependent?, and Are there prerequisite or language skills of children with ASD that make them ideal candidates for this intervention?

See Also

- [Communication Interventions](#)
- [Generalization and Maintenance](#)
- [Video Modeling/Video Self-Modeling](#)

References and Reading

- Ayres, K. M., & Langone, J. (2005). Intervention and instruction with video for students with autism: A review of the literature. *Education and Training in Developmental Disabilities, 40*, 183–196.
- Bandura, A. (1977). *Social learning theory*. Englewood: Prentice Hall.
- Bellini, S., & Akullian, J. (2007). A meta-analysis of video modeling and video self-modeling interventions for children and adolescents with autism spectrum disorders. *Exceptional Children, 73*, 264–287. <https://doi.org/10.1177/001440290707300301>.
- Bellini, S., Peters, J. K., Benner, L., & Hopf, A. (2007). A meta-analysis of school-based social skills interventions for children with autism spectrum disorders. *Remedial and Special Education, 28*, 153–162.
- Charlop-Christy, M. H., Le, L., & Freeman, K. A. (2000). A comparison of video modeling with in vivo modeling for teaching children with autism. *Journal of Autism and Developmental Disorders, 30*, 537–552. <https://doi.org/10.1023/A:1005635326276>
- D'Ateno, P., Mangiapanello, K., & Taylor, B. (2003). Using video modeling to teach complex play sequences to a preschooler with autism. *Journal of Positive Behavior Interventions, 5*, 5–11. <https://doi.org/10.1177/10983007030050010801>.
- Dueñas, A. D., Plavnick, J. B., & Bak, M. Y. S. (2019). Effects of joint video modeling on unscripted play behavior of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 49*, 236–247. <https://doi.org/10.1007/s10803-018-3719-2>.
- Goldstein, H., Lackey, K. C., & Schneider, M. J. B. (2014). A new framework for systematic reviews: Application to social skills interventions for preschoolers with autism. *Exceptional Children, 80*, 262–286. <https://doi.org/10.1177/0014402914522423>.
- Haring, N. G. (1988). *Generalization for students with severe handicaps: Strategies and solutions*. Seattle: University of Washington Press.
- Kasari, C., Dean, M., Kretzmann, M., Shih, W., Orlich, F., Whitney, R., Landa, R. . . . King, B. (2016). Children with autism spectrum disorder and social skills groups at school: A randomized trial comparing intervention approach and peer composition. *The Journal of Child Psychology and Psychiatry, 57*, 171–179. <https://doi.org/10.1111/jcpp.12460>.
- MacDonald, R., Clark, M., Garrigan, E., & Vangala, M. (2005). Using video modeling to teach pretend play to children with autism. *Behavioral Interventions, 20*, 225–238. <https://doi.org/10.1002/bin.197>.
- MacDonald, R., Sacramone, S., Mansfield, R., Wiltz, K., & Ahearn, W. (2009). Using video modeling to teach reciprocal pretend play to children with autism. *Journal of Applied Behavior Analysis, 42*, 43–55. <https://doi.org/10.1901/jaba.2009.42-43>.
- MacManus, C., MacDonald, R. P. F., & Ahearn, W. H. (2015). Teaching and generalizing pretend play in children with autism using video modeling and matrix training. *Behavioral Interventions, 30*, 191–218. <https://doi.org/10.1002/bin.1406>.
- Maione, L., & Mirenda, P. (2006). Effects of video modeling and video feedback on peer-directed social language skills of a child with autism. *Journal of Positive Behavior Interventions, 8*, 106–118. <https://doi.org/10.1177/10983007060080020201>.
- National Autism Center. (2015). *National standards project findings and conclusions*. Randolph: National Autism Center.
- Plavnick, J. B., & Dueñas, A. D. (2018). Brief report: Effects of video-based group instruction on spontaneous social interaction of adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 48*, 2231–2236. <https://doi.org/10.1007/s10803-018-3481-5>.
- Shane, H. C., Laubscher, E. H., Schlosser, R. W., Flynn, S., Sorce, J. F., & Abramson, J. (2012). Applying technology to visually support language and communication in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 42*, 1228–1235. <https://doi.org/10.1007/s10803-011-1304-z>.
- Stokes, T. F., & Baer, D. M. (1977). An implicit technology of generalization. *Journal of Applied Behavioral Analysis, 10*, 349–367. <https://doi.org/10.1901/jaba.1977.10-349>.
- Stokes, T. F., & Osnes, P. G. (1989). An operant pursuit of generalization. *Behavior Therapy, 20*, 337–355.
- Sutton, B., Webster, A. A., & Westerveld, M. F. (2018). A systematic review of school-based interventions targeting social communication behaviors for students with autism. *Autism, 1*–13. <https://doi.org/10.1177/1362361317753564>.
- Wong, C., Odom, S. L., Hume, K. A., Cox, A. W., Fettig, A., Kucharczyk, S., Brock, M. E. . . . Schultz, T. R. (2015). Evidence-based practices for children, youth, and young adults with autism spectrum disorder: A comprehensive review. *Journal of Autism and Developmental Disorders, 45*, 1951–1966. <https://doi.org/10.1007/s10803-014-2351-z>.

Multiple Phenotypes Associated with Disruption of a Single Gene

- [Pleiotropy](#)

Multiple Regression

► Regression Analysis

Multiple Stimulus with Replacement (MSW) Preference Assessment

► Preference and Reinforcer Assessment

Multiple Stimulus Without Replacement (MWSO) Preference Assessment

► Preference and Reinforcer Assessment

Multiplex-Simplex Comparisons

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Definition

“Multiplex” refers to families in which multiple individuals are affected by a specific disease, while “simplex” refers to families in which only a single individual has a specific disease. This distinction is important when designing a study to identify risk factors for ASD, as multiplex families are thought to be more likely to have shared risk factors (i.e., inherited risk), while simplex families are more likely to show de novo genetic risks. Accordingly major collections of ASD samples for genetic analysis are often designed to enrich for specific types of families, such as the Autism Genetic Research Exchange (AGRE) with many multiplex families and the

Simons Simplex Collection (SSC) with exclusively simplex families.

Categorizing families as being multiplex or simplex can be difficult. For example, the presence of only a single affected individual among siblings may not truly reflect the underlying genetic architecture of that family. For example, an unaffected sibling could simply not have inherited a transmitted risk factor that is still present in the parents (and might be transmitted to the next sibling to be born). Alternatively, it is common to observe individuals carrying known genetic risk variants for ASD without showing clinical evidence of autism. The objective of making the distinction is to determine whether shared or unique risk factors are most likely to account for disease. Since ASD is likely to be the combination of many genetic and environmental risk factors, it is probable that many individuals with ASD have a combination of both shared and unique risk factors. The distinction between multiplex and simplex families is therefore best considered as a continuous spectrum that enriches for shared or unique risk factors at each respective extreme.

The distinction between multiplex and simplex families also has bearing on the risk of a further child having ASD. This risk is considerably higher in families with a previous child with ASD, especially if more than one child is affected (Ozonoff et al. 2011).

See Also

- American Board of Genetic Counseling
- Common Disease-Common Variant Hypothesis
- Common Disease-Rare Variant Hypothesis
- DNA
- Genetics

References and Reading

- Ozonoff, S., Young, G. S., Carter, A., Messinger, D., Yirmiya, N., Zwaigenbaum, L., et al. (2011). Recurrence risk for autism spectrum disorders: A Baby Siblings Research Consortium study. *Pediatrics*, 128(3), e488–e495 .peds.2010-2825[pil]. <https://doi.org/10.1542/peds.2010-2825>.

Multi-skilling

► Role Release

MUSAD

► Music-Based Scale for Autism Diagnostics (MUSAD) Assessing Adults with Intellectual Disability

Music Therapy

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Definition

Music therapy uses musical activities such as singing, playing instruments, improvisation, and music listening to promote interaction between the therapist and the client and to meet the client's therapeutic needs. American Music Therapy Association (2005) defined music therapy as "the clinical and evidence-based use of music interventions to accomplish individualized goals within a therapeutic relationship by a credentialed professional who has completed an approved music therapy program."

There are many different approaches in music therapy. In a broad sense, music therapy methods can be divided into either active or receptive music therapy, depending on whether the client is actively involved in live music-making processes with the therapist or the client is involved in a preselected music listening using either a recorded music or a live performance. In active, especially the improvisational music therapy intervention, interactive use of live and spontaneous music-making process between the therapist and the client, the client is encouraged and facilitated.

Therefore, musical-therapeutic context evolves through musical interaction between the therapist and the client here and now and moment by moment. In receptive music therapy intervention, music is selected or precomposed based on the preference and needs of the client to accomplish certain therapeutic outcomes for individuals with autism spectrum disorders (ASD). Research evidences have indicated that both active and receptive music therapy methods are found to be effective at facilitating and improving communication and social interaction skills and to modify certain behaviors of individuals with ASD (Accordino et al. 2007; Geretsegger et al. 2014; Simpson and Keen 2011). In clinical practice, music therapists tend to use the method that serves their client's individual needs, sometimes resulting in eclectic approaches.

Historical Background

There have been various approaches in music therapy, depending on the different philosophies and principles of the founder of each music therapy approach in different clinical settings. Early music therapy pioneers such as Juliette Alvin, Paul Nordoff, and Clive Robbins worked with children with special needs including autism from the 1950s. These early pioneers used live interactive music (improvisation) to engage the children with ASD and to address their difficulties in communication and social interaction (Alvin 1978; Nordoff and Robbins 1971). Now, there are many music therapists working within the improvisational model of music therapy worldwide, especially in the UK and Europe.

Following are four internationally known improvisational models of music therapy:

1. Free improvisation therapy founded and developed by Juliette Alvin
2. Creative music therapy founded and developed by Paul Nordoff and Clive Robbins
3. Analytically oriented music therapy founded and developed by Mary Priestley
4. Interactive music therapy founded and developed by Amelia Oldfield

All four approaches above have been mainly developed in the UK.

Another significant approach in music therapy has been behavioral modification model of music therapy that was chiefly developed in the USA and is still the primary music therapy intervention in the USA. The influence of behavioral modification model of music therapy is also prevalent worldwide especially for children with special needs including autism.

Simpson and Keen (2011) has identified composed songs and improvisation as the predominant music therapy techniques used for children with ASD. Research evidence indicated that behavioral music therapy and improvisational music therapy all work well with individuals with ASD (Geretsegger et al. 2014; Kaplan and Steele 2005; Simpson and Keen 2011).

Rationale or Underlying Theory

Music is often described as a universal or emotive language. Music therapists have attributed this mainly due to its power to reach and affect human emotions. No matter how disabled a person can be, anyone can respond to music, and musical responses are innate ability deeply engrained in human nature (Alvin 1978; Kim et al. 2009; Nordoff and Robbins 1971). Moreover, musical experiences are often intrinsically joyful and interesting. Music often inspires, motivates, and brings people together even when they are total strangers. Kim and her colleagues (2009) claim: "Music acts as an essentially emotional, relational and motivational medium when, in music therapy, it is purposefully created to engender 'interpersonal relatedness' by employing a well-measured systematic intervention." Thaut (1987) found that children with autism engaged significantly more time with the music than age-matched typically developing children. He also analyzed musical responsiveness of children with autism, and that of children without autism through music improvisation they played, and found no significant difference in musical responsiveness between them. The results indicated

intact musical responsiveness and musical aptitude in children with ASD. Clinical implication of these findings suggests significant therapeutic potential in music that can be used in helping individuals with ASD in everyday life.

Music can be an excellent medium for emotional communication and social interaction for individuals who cannot easily assimilate the conventional ways of human communication and interaction. Instead of verbal exchange, nonverbal aspects of music such as playing simple instruments, vocalizing, and listening to music can provide an alternative way of communicating and engaging the individuals with ASD and others without ASD. Music can be utilized to teach new skills (Buday 1995; Kern and Aldridge 2006), to help children with ASD perceive and recognize specific emotions (Katagiri 2009), and to modify children's behaviors (Brownell 2002; Kern et al. 2007) in a certain direction that can be helpful and adaptable in everyday life. As children with ASD are reported to respond better to songs than spoken words, a music therapist might create original songs (Brownell 2002; Katagiri 2009; Kern and Aldridge 2006), or lyrics with familiar melody (Kern et al. 2007) about a specific behavior, and use these songs with children with ASD with specific therapeutic goals in mind including speech and language development.

Goals and Objectives

Goals and objectives of music therapy intervention for individuals with ASD are usually established through music therapy assessment. Music therapists assess the individual's needs, especially the domains of emotional, communicative, behavioral, and social responses and cognitive skills for individuals with ASD. Systematic review of music therapy literature has indicated primary goal areas in music therapy intervention: (1) to promote socialization, (2) to increase communication and language skills, and (3) to modify maladaptive behaviors (Accordino et al. 2007; Geretsegger et al. 2014; Simpson and Keen 2011). Once broad goals are set up, the therapist

identifies specific objectives that are observable and measurable such as “when therapist sings a ‘hello song’ in the beginning of the session, child will look at the direction of therapist 30% of the time.” Objectives of music therapy will change in time as therapy progresses. As autism spectrum disorders are complex neurodevelopmental disorders showing variety and individuality across all ages and abilities, therapeutic objectives for individuals with ASD varies a lot from person to person and stage of each therapy process. Musical responses of the brain is not localized in one area, but known to be widespread. Therefore music has the strong potential to strengthen and optimize brain function that is known to be damaged and/or weak. In the improvisational music therapy, the main goal of the therapy with individuals with ASD is to establish, maintain, and develop interaction between the therapist and the client through active music-making processes and to deal with developmental and therapeutic issues that are presented by the client during each stage of therapy process.

Treatment Participants

Music therapy has been used for preschool and school-age children, adolescents, and adults with wide range of different abilities and symptom severity. The most common participants are preschool and school-age children with ASD in music therapy (Geretsegger et al. 2014).

Treatment Procedures

When referral is made to a music therapist, the music therapist assesses the individual’s needs, plans either individual or group music therapy sessions based on the needs of the individual, and implements and evaluates music therapy sessions where appropriate. Following are the typical music therapy procedures:

1. Referral: Referral to music therapy can be made by the parents, doctors, speech therapists, teachers, social workers, etc.

2. Assessment: The music therapist assesses the individual’s needs in terms of his/her strengths and weaknesses through musical responses using various musical activities.
3. Goal setting: Treatment goals and objectives are developed based on the findings of assessment. Therefore, assessment will lead to setting up appropriate treatment plan for individuals with ASD.
4. Treatment implementation: Music therapy sessions can be one-on-one or in a group, which consist of musical activities that are designed to meet the individual’s needs and therapeutic goals.
5. Evaluation: Music therapy sessions are regularly evaluated by the music therapist and often within the interdisciplinary team.
6. Termination of music therapy: The duration of music therapy varies from person to person, depending on the needs of the individual. Short-term therapy typically consists of 10–12 sessions. Long-term therapy can be up to 2 or 3 years. For young children with ASD, music therapy sessions are typically more than 3 months. Children with ASD typically have once, twice, or thrice weekly music therapy sessions for about 20–50 min, depending on attention span and needs of the child. Music therapy is typically offered in an individual (one-to-one), group, and/or family-based (family members such as parents and other siblings) setting.

Kaplan and Steele (2005) investigated 40 music therapy clients with ages ranging from 2 to 49 years with ASD in Cleveland, USA. They found individual music therapy as the most common session type, followed by partner, small group (3–5 subjects), large group (more than 6 subjects), etc.

Efficacy Information

In the last few decades, there have been accumulating research evidences indicating the efficacy and the effectiveness of music therapy for individuals with ASD, with some promising results in

the domains of social, communicative, and behavioral skills (Bieleninik et al. 2017a; Brownell 2002; Buday 1995; Geretsegger et al. 2014; Katagiri 2009; Kern and Aldridge 2006; Kim et al. 2008, 2009). Recently, there have been some systematic or narrative reviews on effects of music therapy for individuals with ASD. It was found that majority of published articles in music therapy for individuals with ASD were case studies with anecdotal evidences, and there have been limited numbers of well-designed empirical investigations typically with small samples (Accordino et al. 2007; Geretsegger et al. 2014; Simpson and Keen 2011). In the recently updated Cochrane meta-analysis of music therapy for ASD, Geretsegger and associates (2014) reported sufficient evidences for music therapy as effective intervention in improving core symptoms of ASD that is in the areas of social communication, social emotional reciprocity, and parent-child relationship. Effect sizes were moderate-to-large. Included ten studies (nine RCTs, 165 children aged between two to nine) of Cochrane review examined short- to medium-term effects of music therapy compared to either standard care (SC) or similar intervention without music. Seven out of ten studies were short-term trials (1–5 weeks) employing a highly structured therapist-directed approach largely using receptive techniques. Remaining three studies were medium-term trials (3–7 months) utilizing a child-centered and interactive approach using improvisation as means of communication between the therapist and the child.

In contrast to the recent Cochrane findings, Bieleninik and associates (2017a) conducted a large-scale, well-designed, multicenter randomized clinical trial (364 children from nine countries) of improvisational music therapy (IMT) for young children with ASD (4–7 years-old), comparing IMT plus standard care (SC) with SC to determine effects of IMT on the core symptoms of ASD. They found no significant difference between IMT and SC on the reduction of core symptoms of ASD. They employed the ADOS (Autism Diagnostic Observation Schedule) social affect domain as primary outcome and SRS (Social Responsiveness Scale) as secondary

outcome measures. The children in their study showed slight reduction of core symptoms over time in both conditions from the ADOS social affect domain. Although there were no significant differences in the primary outcome measure, post hoc exploratory analyses revealed that the children in IMT showed higher proportion of improvement in social communication at 5 months (95/182 [52%]) than the children in SC (76/182 [42%]). They also found significant effects in several SRS subscales. For example, music therapy was found to be more effective at improving social motivation than SC over 5 months.

There could be different levels of explanation and debates on how to understand the contradictory results between the Cochrane review and the most recent RCT as these two types of studies are considered the most rigorous clinical evidence.

Bieleninik et al. (2017b) conducted meta-analysis of 5771 children with ASD aged 7 months to 16.5 years to determine temporal stability of ASD diagnosis and core symptom severity over time. The meta-analysis of their study indicated core symptoms of ASD were markedly stable over time during childhood of these children. Bieleninik and associates (2017b) stated that clinical outcome studies should focus on “other areas, such as quality of life and adaptive functioning.”

In Kaplan and Steele’s investigation (2005) on 40 music therapy clients with ages ranging from 2 to 49 years with ASD, 100% of parents and caregivers of these clients reported that these individuals with ASD generalized skills and responses obtained in music therapy to other environments.

Outcome Measurement

Abovementioned clinical outcome studies used generalized (intervention outcome measured outside of music therapy setting) and nongeneralized (intervention outcome measured within music therapy) outcome measurements to examine effects of music therapy on people with ASD.

Four studies (Bieleninik et al. 2017; Gattino et al. 2011; Kim et al. 2008; Thompson 2012) used standardized and published scales such as

the ADOS/the SRS; the Childhood Autism Rating Scale (CARS); the Early Social Communication Scale (ESCS)/the Pervasive Developmental Disorder Behavior Inventory (PDDBI); and the Vineland Social-Emotional Early Childhood Scales (SEEC)/the SRS.. The ADOS and the CARS are primarily developed as diagnostic tool adapted to intervention studies whereas the ESCS, the PDDBI, the Vineland-EEC, and the SRS are developed and/or used to examine responsiveness to interventions in individuals with ASD. These four studies measured generalized social communication skills utilizing these standardized scales.

However, nongeneralized outcome measurements are still the most common assessments in music therapy, mostly based on either direct or through video recording observations, and/or substantial clinical documentation. For observational outcome measurements, specific target behaviors are defined prior to conducting outcome evaluation. Some music therapy researchers measured specific target behaviors through direct observation using event recordings (Brownell 2002; Kern et al. 2007). Others used video recordings to evaluate behavioral outcomes for children with ASD (Buday 1995; Kern and Aldridge 2006; Kim et al. 2008, 2009).

While it is important to use observational outcome measurements in music therapy, it is also pertinent to develop standardized outcome measurement in music therapy capturing unique benefit of music therapy for the individuals with ASD that is valid, reliable, and comparable to other standardized measurements.

Qualifications of Treatment Providers

Music therapy services are provided by registered music therapists who have completed an accredited music therapy degree program by the music therapy association in their respective country. In the USA, Australia, many countries in Europe, and South Korea, music therapists typically hold either undergraduate or master's degree in music therapy. In the UK, music therapists typically hold a master's degree in music therapy and have state registration with Health Professions Council. In

the USA, there are large numbers of American Music Therapy Association-approved programs providing equivalency or certificate degrees in music therapy for people that have already obtained a degree in related fields. Individuals who have completed an approved music therapy degree program are qualified to take the national board certification examination and can earn the credential of MT-BC (Music Therapist-Board Certified) after passing the exam. South Korea has benchmarked the American system and has set up the national music therapy certification examination for music therapists.

See Also

American Music Therapy Association: www.musictherapy.org
 Australian Music Therapy Association: www.austmta.org.au
 British Society for Music Therapy/Association of Professional Music Therapists: www.apmt.org
 Canadian Association for Music Therapy: www.musictherapy.ca
 National Association of Korean Music Therapists: www.nakmt.or.kr
 Nordoff-Robbins Center in the UK: www.nordoff-robbins.org.uk
 The European Music Therapy Confederation: www.emtc-eu.com
 World Federation of Music Therapy: <http://wfmt.info/WFMT/Home.html>

References and Reading

- Accordino, R., Comer, R., & Heller, W. B. (2007). Searching for music's potential: A critical examination of research on music therapy with individuals with autism. *Research in Autism Spectrum Disorders*, 1, 101–115.
- Alvin, J. (1978). *Music therapy for the autistic child*. London: Oxford University Press.
- American Music Therapy Association. (2005). What is music therapy?: Definition and quotes about music therapy. Retrieved May 2012 from www.musictherapy.org/about/musictherapy/.
- Bieleninik, L., Geretsegger, G., Mössler, K., Assmus, J., ... Gold, C. (2017a). Effects of improvisational music

- therapy vs enhanced standard care on symptom severity among children with autism spectrum disorder. The TIME-A randomized clinical trial. *Journal of American Medical Association*, 318(6): 525–535.
- Bieleninik, L., Posserud, M. B., Geretsegger, M., Thompson, G., Elefant, C., & Gold, C. (2017b). Tracing the temporal stability of autism spectrum diagnosis and severity as measured by the Autism Diagnostic Observation Schedule: A systematic review and meta-analysis. *PLoS One*, 12(9), e0183160. <https://doi.org/10.1371/journal.pone.0183160>.
- Brownell, M. K. (2002). Musically adapted social stories to modify behavior in students with autism: Four case studies. *Journal of Music Therapy*, 39, 117–144.
- Buday, E. M. (1995). The effects of signed and spoken words taught with music on sing and speech imitation by children with autism. *Journal of Music Therapy*, 32, 189–202.
- Gattino, G. S., Riesgo, R. D. S., Longo, D., Leite, J. C. L., & Faccini, L. S. (2011). Effects of relational music therapy on communication of children with autism: A randomized controlled study. *Nordic Journal of Music Therapy*, 20(2), 142–154.
- Geretsegger, G., Elefant, C., Mössler, K. A., & Gold, C. (2014). Music therapy for people with autism spectrum disorder. *Cochrane Database Systematic Reviews*, (6), CD004381. <https://doi.org/10.1002/14651858.CD004381.pub3>.
- Kaplan, R. S., & Steele, A. L. (2005). An analysis of music therapy program goals and outcomes for clients with diagnoses on the autism spectrum. *Journal of Music Therapy*, 42, 2–19.
- Katagiri, J. (2009). The effect of background music and song texts on the emotional understanding of children with autism. *Journal of Music Therapy*, 46(1), 15–31.
- Kern, P., & Aldridge, D. (2006). Using embedded music therapy interventions to support outdoor play of young children with autism in an inclusive community-based child care program. *Journal of Music Therapy*, 43(4), 270–294.
- Kern, P., Wolery, M., & Aldridge, D. (2007). Use of songs to promote independence in morning greeting routines for young children with autism. *Journal of Autism and Developmental Disorders*, 37, 1264–1271.
- Kim, J., Wigram, T., & Gold, C. (2008). The effects of improvisational music therapy on joint attention behaviors in autistic children: A randomized controlled study. *Journal of Autism and Developmental Disorders*, 38, 1758–1766.
- Kim, J., Wigram, T., & Gold, C. (2009). Emotional, motivational and interpersonal responsiveness of children with autism in improvisational music therapy. *Autism*, 13(4), 389–409.
- Mössler, K., Gold, C., Assmus, J., Schumacher, K., Calvet, C., Reimer, S., Iversen, G., & Schmid, W. (2017). The therapeutic relationship as predictor of change in music therapy with young children with autistic spectrum disorder. *Journal of Autism Developmental Disorder*. <https://doi.org/10.1007/s10803-017-3306-y>.
- Nordoff, P., & Robbins, C. (1971). *Therapy in music for handicapped children*. London: Gollancz.
- Simpson, K., & Keen, D. (2011). Music interventions for children with autism: Narrative review of the literature. *Journal of Autism and Developmental Disorders*, 41, 1507–1514.
- Thaut, M. (1987). Visual versus auditory (musical) stimulus preferences in autistic children: A pilot study. *Journal of Autism and Developmental Disorders*, 17, 425–432.
- Thompson, G. (2012). Family-centered music therapy in the home environment: Promoting interpersonal engagement between children with autism spectrum disorder and their parents. *Music Therapy Perspectives*, 30(2), 109–116.

Music-Based Scale for Autism Diagnostics (MUSAD) Assessing Adults with Intellectual Disability

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Synonyms

MUSAD

Description

The MUSAD is a standardized observational instrument to assess autistic traits in adults with intellectual disabilities (ID) suspected of having an additional autism spectrum disorder (ASD). The nonverbal and age-independent quality of musical interaction is used as a framework to assess people with speech impairments.

The assessment comprises 11 structured musical-interactive situations to prompt diagnostically relevant behaviors. Detailed step-by-step instructions are given to ensure stability of the application across time and between clients and different raters. First of all, the assessor attempts to engage the client in musical activity,

e.g., joint drumming. In this particular situation, several prompts – tempo change, alternating of hands (right-left stroke), invitation to dialogue by playing a short motif, and a joyful crescendo with a final blow – are set to elicit client reactions in diagnostic relevant behaviors. Secondly, observation priorities are given to recapitulate the assessment with note-taking in free text. In the drumming situation, focus is placed on interpersonal attunement in synchronizing to tempo changes, motor coordination in right-left strokes, nonverbal social reciprocity in back-and-forth playing, and social affect in sharing musical dynamics and intensity. Video recording is recommended to support the assessor in changing roles from being in playful contact with the client to being a distant observer, closely assessing and monitoring client behavior. Thirdly, each of 46 items is coded on a 4-point Likert scale according to ascending symptom severity. Each item is assigned to a situation/prompt (e.g., drumming → “reciprocity in musical interaction”) or even overall (e.g., “eye contact”), and each point is described by quantitative or qualitative behavioral markers. The diagnostic algorithm is formed by 26 items representing the dyadic ASD-construct of social impairments and stereotyped behaviors, including sensory issues, according to DSM-5. A total score is formed by summing up the values of the marked algorithm items. A cutoff point of 30 is proposed to indicate the presence vs. absence of ASD. Especially in borderline cases around 30 points, the diagnostic indication may be discussed, considering ID level, comorbidities including sensory and motor impairments not associated with ASD, or score inconsistencies across domains.

Historical Background

Going far back in the history of ASD, Kanner (1943) described pronounced musical interest and skills in 6 of 11 children in his groundbreaking case series. Up to now, music has played an important role in the therapy and education of children with ASD (Geretsegger et al. 2014, Srinivasan and Bhat 2013). Alongside several

music-based approaches in fostering people with ASD, there have been some semi-structured attempts using the music-therapy setting as a framework to observe and assess autism-related behaviors in children for diagnostic purposes (Oldfield 2006; Wigram 1999).

For decades, the main focus in developing scales and instruments for proper ASD diagnosis was on children and youths, resulting in numerous screening tools and a diagnostic gold standard for this age group. The fact of frequently undiagnosed ASD in adulthood was only recognized relatively recently (Brugha et al. 2011), as was the high prevalence of ASD in the ID population (Saemundsen et al. 2010). Within the low-functioning ASD group, a significantly larger number of comorbid conditions were found as compared with people diagnosed only with ID, demanding individually tailored treatment and support (Cervantes and Matson 2015). However, especially in adults with ID, the symptom overlap of ASD and severe cognitive impairment combined with comorbid conditions represents a major diagnostic challenge. Certain symptoms may be misinterpreted or not recognized, as they may be biased by ► [diagnostic overshadowing](#) and a lack of information about early childhood. This led to the development of various ID-specific ASD screeners that capture an adult ASD-phenotype (e.g., DIBAS-R). Even if the diagnostic gold standard represented by the combination of ► [ADOS](#) and ► [ADI-R](#) is principally applicable in adults with ID, its usefulness is limited owing to reduced feasibility, imbalanced algorithms, and age-inappropriate materials (Sappok et al. 2013). This unsatisfactory situation was the starting point for modifying a semi-structured music-therapy approach for ASD-diagnostic purpose and developing the MUSAD (Bergmann et al. 2009). The concept of assessing interactional skills and stereotyped behaviors in structured but naturalistic situations in direct contact with the client was oriented toward the ADOS. In addition to using a musical-interactional setting to prompt ASD-related behaviors, the MUSAD differs with its concept of increasing demands throughout the entire examination to increase feasibility in

cases of high irritability, along with including motor coordination as a potential ASD marker.

Items were created based on DSM-5 ASD criteria, a review of scales and literature, and clinical experience of the test developers in the field of the targeted population and music-based interventions. The framework for developing the MUSAD was a specialized treatment center with both in- and outpatient clinics, for adults with ID and mental health problems.

A guideline-compliant diagnostic work-up has been established on the basis of an expert consensus conference supported by various ASD measures (Bergmann et al. 2016). This allowed the evaluation and validation of the MUSAD assessment based on consecutive samples and an external criterion for diagnostic classification reflecting clinical reality. The future direction will be to develop a brief version of the instrument (MUSAD-Short) aiming to improve economy and user-friendliness and allowing ASD screening in people with ID including children and adolescents.

Psychometric Data

Psychometric properties of the MUSAD have been evaluated in a pilot study assessing feasibility, user-friendliness, and construct validity ($N = 80$; Bergmann et al. 2015a, b) and a validation study focusing mainly on inter-rater reliability and criterion validity ($N = 124$; Bergmann et al. 2019).

Feasibility was 95% ($N = 80$). Compared with the play-based ADOS applied in parallel, the MUSAD showed considerably lower dropout rates (5% vs. 15%) in $n = 40$ cases. Practicability of the test draft was supported by the successful video-based coding of one case by 12 raters from different professional backgrounds who were unfamiliar with the instrument. Plausibility of the items and coding descriptions were judged as good by questionnaire, even if the substantial coding effort was criticized.

A confirmatory factor analysis supported a three-dimensional construct ($F1 =$ social interaction, $F2 =$ stereotyped behaviors including sensory issues, $F3 =$ motor coordination). Testing

the MUSAD construct fit indices indicated a good model fit ($CFI = 0.97$, $RMSEA = 0.06$, 90% CI [0.05–0.07] and $WRMR = 1.02$), even if the chi-squared test of exact model fit suggested significance (χ^2 ($df = 626$) = 822.8, $p < 0.001$).

Concurrent validity was supported by substantial and significant correlation with the convergent ASD screener DiBAS-R ($r(111) = 0.62$, $p < 0.001$) and a negligible nonsignificant correlation with the discriminant aggression scale MOAS ($r(56) = 0.15$, $p = 0.252$). Scale reliability was high ($\alpha = 0.95$).

Inter-rater agreement based on the sum scores across 4 raters in $n = 22$ cases was excellent ($ICC = 0.92$). Test-retest reliability in $n = 9$ ad hoc test repetitions was moderate ($ICC = 0.73$).

Criterion validity was calculated by applying an ROC analysis. With a cutoff score of 30 points, 76.6% of all individuals assessed were classified correctly ($\kappa = 0.52$; sensitivity = 0.79, 95% CI [0.68–0.87] and specificity = 0.74, 95% CI [0.59–0.85]. The positive predictive value was 0.82, 95% CI [0.74–0.88], and the negative predictive value was 0.69, 95% CI [0.59–0.78]. The AUC values were 0.81, 95% CI [0.73–0.88] for the overall score, 0.79, 95% CI [0.71–0.88] for the social interaction score, and 0.78, 95% CI [0.70–0.87] for stereotyped behaviors including sensory and motor issues.

Clinical Uses

The MUSAD is used clinically as one part in a multidimensional team-based diagnostic work-up as proposed by current guidelines (NICE 2012). The targeted population is adults (age >18 years) with ID and impaired speech. The implementation requires approximately 90 min, including video-based coding. Because of this relatively high effort, comparable to the ADOS and ADI-R, it might be useful to apply the MUSAD in a two-stage diagnostic work-up after ASD screening in case of diagnostic uncertainty. The assessment can be performed by a music therapist or other professionals (psychologists, pedagogues) with basic musical skills. For all users a specialized training is required. This includes correct implementation as well as behavioral observation and

coding supporting inter-rater agreement. A special feature of the MUSAD is the inclusion of motor coordination, considered a cardinal feature of ASD throughout the entire spectrum (Fournier et al. 2010). This may support differential diagnostic discrimination, e.g., over against psychosis or trauma-related disorders/hospitalization. As with other ASD scales, the MUSAD should not be used as a stand-alone instrument for diagnostic classification, but it may be a useful resource in team-based diagnostic procedures.

See Also

- ▶ Autism Diagnostic Observation Schedule
- ▶ Barriers to Formal Diagnosis of Autism Spectrum Disorder in Adults
- ▶ Diagnostic Overshadowing
- ▶ DIBAS-R Validation
- ▶ Intellectual Disability
- ▶ Music Therapy

References and Reading

- Bergmann, T., Sappok, T., Schumacher, K., & Diefenbacher, A. (2009). Musiktherapeutischer Ansatz in der Behandlung von Erwachsenen mit Autismus und geistiger Behinderung [Music-therapeutic approach in the treatment of adults with autism and intellectual disability]. In F. Schneider & M. Groezinger (Eds.), *Psychische Erkrankungen in der Lebensspanne: Abstractband zum DGPPN Kongress 2009*. Berlin: Deutsche Gesellschaft für Psychiatrie, Psychotherapie und Nervenheilkunde.
- Bergmann, T., Sappok, T., Diefenbacher, A., Dames, S., Heinrich, M., Ziegler, M., & Dziobek, I. (2015a). Music-based Autism Diagnostics (MUSAD): A newly developed diagnostic measure for adults with intellectual developmental disabilities suspected of autism. *Research in Developmental Disabilities*, 43–44, 123–135. <https://doi.org/10.1016/j.ridd.2015.05.011>.
- Bergmann, T., Sappok, T., Diefenbacher, A., & Dziobek, I. (2015b). Music in diagnostics: Using musical interactional settings for diagnosing autism in adults with intellectual developmental disabilities. *Nordic Journal of Music Therapy*, 25(4), 319–351. <https://doi.org/10.1080/08098131.2015.1039567>.
- Bergmann, T., Diefenbacher, A., Heinrich, M., & Sappok, T. (2016). Perspektivenverschränkung: Multiprofessionelle Autismusdiagnostik bei erwachsenen Menschen mit Intelligenzminderung und Autismusverdacht [Entangled perspectives. A multiprofessional approach in diagnosing autism in adults with intellectual disability]. *Zeitschrift für Psychiatrie, Psychologie und Psychotherapie*, 64, 257–267. <https://doi.org/10.1024/1661-4747/a000287>.
- Bergmann, T., Heinrich, M., Ziegler, M., Dziobek, I., Diefenbacher, A., & Sappok, T. (2019). Developing a diagnostic algorithm for the music-based scale for autism diagnostics (MUSAD) assessing adults with intellectual disability. *Journal of Autism and Developmental Disorders*, 49(9), 3732–3752. <https://doi.org/10.1007/s10803-019-04069-y>.
- Brugha, T. S., McManus, S., Bankart, J., Scott, F. J., Purdon, S., Smith, J., . . . Meltzer, H. (2011). Epidemiology of autism spectrum disorders in adults in the community in England. *Archives of General Psychiatry*, 68(5), 459–465. <https://doi.org/10.1001/archgenpsychiatry.2011.38>
- Cervantes, P. E., & Matson, J. L. (2015). Comorbid symptomology in adults with autism spectrum disorder and intellectual disability. *Journal of Autism and Developmental Disorders*, 45(12), 3961–3970. <https://doi.org/10.1007/s10803-015-2553-z>.
- Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism spectrum disorders: A synthesis and meta-analysis. *Journal of Autism and Developmental Disorders*, 40(10), 1227–1240. <https://doi.org/10.1007/s10803-010-0981-3>.
- Geretsegger, M., Elefant, C., Kim, J., & Gold, C. (2014). Music therapy for people with autism spectrum disorder. *The Cochrane Database of Systematic Reviews*, 6, CD004381. <https://doi.org/10.1002/14651858.CD004381.pub3>.
- Kanner, L. (1943). Autistic disturbance of affective contact. *The Nervous Child*, 2, 217–250.
- National Institute for Health and Clinical Excellence. (2012). Autism in adults: Diagnosis and management. Retrieved from <https://www.nice.org.uk/guidance/cg142/chapter/1-guidance>
- Oldfield, A. (2006). *Interactive music therapy in child and family psychiatry: Clinical practice, research, and teaching*. London/Philadelphia: Jessica Kingsley.
- Saemundsen, E., Juliusson, H., Hjaltestad, S., Gunnarsdottir, T., Halldorsdottir, T., Hreidarsson, S., & Magnusson, P. (2010). Prevalence of autism in an urban population of adults with severe intellectual disabilities: A preliminary study. *Journal of Intellectual Disability Research*, 54(8), 727–735. <https://doi.org/10.1111/j.1365-2788.2010.01300.x>.
- Sappok, T., Diefenbacher, A., Budczies, J., Schade, C., Grubich, C., Bergmann, T., . . . Dziobek, I. (2013). Diagnosing autism in a clinical sample of adults with intellectual disabilities: How useful are the ADOS and the ADI-R? *Research in Developmental Disabilities*, 34(5), 1642–1655. <https://doi.org/10.1016/j.ridd.2013.01.028>.
- Srinivasan, S. M., & Bhat, A. N. (2013). A review of “music and movement” therapies for children with autism: Embodied interventions for multisystem development. *Frontiers in Integrative Neuroscience*, 7, 22. <https://doi.org/10.3389/fnint.2013.00022>.

Wigram, T. (1999). Contact in music: The analysis of musical behaviour in children with communication disorder and pervasive developmental disability for differential diagnosis. In T. Wigram & J. de Backer (Eds.), *Clinical applications of music therapy in developmental disability, paediatrics and neurology* (pp. 69–118). London: Jessica Kingsley.

Sample References

Book

American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.). Washington, DC: APA Press.

Chapter in a Book

Howlin, P. (2005). Outcomes in autism spectrum disorders. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. I, pp. 640–649). Hoboken: Wiley.

Mesibov, G. B., Shea, V., & Schopler, E. (2004). *The teach approach to autism spectrum disorders*. New York: Springer.

Article in a Journal

Bachevalier, J. (1996). Brief report: Medical temporal love and autism: A putative animal model in primates. *Journal of Autism and Developmental Disorders*, 26 (2), 217–220.

Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.

Music-Evoked Emotion

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Definition

“Emotion elicited through musical stimuli, including subjective, cognitive, and physiological responses”.

Historical Background

Considerable research and study has been dedicated to understanding emotional processing in autism spectrum disorder (ASD). Differences in emotion recognition and emotional responsiveness are commonly noted in clinical presentations (Begeer et al. 2008). As a result, considerable attention has also been dedicated to better understand emotional processing in the research literature. Historically, these investigations focused on social stimuli such as viewing emotional faces. Results from multiple meta-analyses indicate that, despite considerable heterogeneity, overall impairments in facial emotion recognition exist and cannot be completely accounted for by intellectual ability of people with ASD (Lozier et al. 2014; Uljarevic and Hamilton 2013). These differences seem to be related, at least in part, to different ways of processing faces such as reduced eye fixation (Jones et al. 2008; Klin et al. 2002; Pelphrey et al. 2002) and appear to be strongest for the emotions of *anger* and *fear* (Lozier et al. 2014). The question then arises if these patterns are present for other emotional material, such as music.

There is a long history of people with ASD showing an interest in music. Pioneering work by Heaton and colleagues highlighted strengths in musical processing of ASD children compared to children without ASD. Their work and that from other labs have shown musical strengths including memory of musical information and pitch perception (e.g., discrimination, detection, and memory) (see Quintin (2019) for a review). Additionally, the experience of music may be largely similar for people with ASD with evidence showing similar levels of interest in, time spent listening to, and reasons for listening to music as the typical listener (Allen et al. 2009; Bhatara et al. 2013).

Music has a powerful influence on emotional states of humans, and there is a wealth of information highlighting how music can activate neural pathways similar to other nonmusical forms of emotional stimuli and people are consistently able to identify specific emotions depicted through music (see Koelsch (2014) for a review; Schaefer 2017). The actual musical elements that tend to

elicit a particular emotion is varied and complex; they include pitch variation, rhythm, tempo, tonality, timbre, articulation, musical mode, harmony, and melodic direction and motion, to name a few (Gabrielsson and Lindström 2011). The scientific interest in music-evoked emotion and ASD has direct applications, most notably with regards to the use of music therapy. Music therapy, or complementing traditional therapy with music, is a promising avenue for improving outcomes (see reviews by Geretsegger et al. 2014; James et al. 2015) including verbal (Chenausky et al. 2016), general communication (Sharda et al. 2018), and social skills of children with ASD such as social-emotional reciprocity, quality of social interactions, and initiation of social exchanges (LaGasse 2017).

Current Knowledge

Identification of Music-Evoked Emotion

There have been several studies that have directly investigated the question of “can people with ASD identify emotions in instrumental music as well as their peers without ASD?” Thus far, the evidence suggests that the answer to that question is likely “yes.” There are studies that have clearly showed intact identification of specific emotions in music including *happy*, *sad* (Heaton et al. 1999; Whipple et al. 2015), *fear* (Quintin et al. 2011; Stephenson et al. 2016), *disgust*, and *anger* (Whipple et al. 2015). Other studies give evidence for intact ability to match emotional social scenes with corresponding musical excerpts (Heaton et al. 2008) and assigning emotional valence (i.e., positive vs. negative) to *happy* and *sad* music (Gebauer et al. 2014). One study found that children and adolescents with ASD did not differ in their emotional labeling skills, but this was the case only after statistically controlling for differences in verbal intellectual ability (Quintin et al. 2011). Another study found possible age-related differences between youth with and without ASD, specifically for *scary* music, in that younger autistic children tended to be slightly more accurate in identifying *scary* music compared to their older autistic peers, the opposite

age-related pattern was found for children and adolescents without autism (Stephenson et al. 2016). Thus, emotion identification of people with ASD appears to be largely intact, although there may be nuanced effects of age and verbal ability that will need to be explored and clarified with future research.

Response to Music-Evoked Emotion

In addition to the behavioral aspect of recognizing emotional characteristics of music, there is also the psychological and physiological responsiveness to music-evoked emotion. Parents of children with ASD rate their children’s emotional responsiveness to music similarly to those of their non-ASD peers (Bhatara et al. 2013) and children themselves do not differ in their self-reported perception of intensity of music-evoked emotion in laboratory-based studies using simple rating scales (Quintin et al. 2011; Stephenson et al. 2016), with similar results appearing for adults (Caria et al. 2011). However, according to studies with autistic adults, they tend to describe emotional reactions to music using different words than non-autistic adults, which may be related to difficulties understanding one’s internal emotion states and struggling to put words to emotional experiences, often referred to as alexithymia, (Allen et al. 2013; Allen et al. 2009), which is often associated with ASD (Bird and Cook 2013; Poquérousse et al. 2018). Given the intact ability of autistic people to recognize and match emotional music to expressed emotions, music has potential to be used as a tool in developing emotional understanding and expression outside of music for people with ASD (Allen and Heaton 2010).

Evidence on physiological responsiveness to music is just emerging. Adults with ASD showed no difference in their galvanic skin response compared to non-autistic adults (Allen et al. 2013), whereas autistic children and adolescents had reduced physiological response compared to their neurotypical peers in another study when listening to the same instrumental music stimuli (Stephenson et al. 2016). Neuroimaging studies have shown that brain networks typically involved in the processing of music-evoked emotion, including reward circuitry, are intact for

people with ASD, at least for *happy* and *sad* music (Caria et al. 2011; Gebauer et al. 2014). Although some results indicate that listening to *happy* music evoked somewhat *reduced* activation in certain areas including insula (Caria et al. 2011), other results show *increased* activation for *happy* vs. *sad* music in insula as well as dorsolateral prefrontal cortex, possibly related to increased arousal and cognitive processing (Gebauer et al. 2014). Notably, current neuroimaging studies have focused exclusively on autistic adults and a fairly narrow range of music-evoked emotions (i.e., *happy* and *sad*). Investigating a wider range of emotions is needed, especially given the propensity towards anxiety and fear in autistic individuals including differences in sensory and cognitive processing of fear (South and Rodgers 2017).

Despite heterogeneity in research findings within ASD samples, which is not uncommon, the growing literature paints a picture of overall intact psychological and physiological responsiveness to music-evoked emotion. However, similar to results from emotion identification, there may exist nuanced differences in emotional experience, particularly when different emotions are considered.

Future Directions

Despite the current knowledge of the processing of music-evoked emotion of people with ASD, there are still many unanswered questions. Additional research is needed using a wider range of emotions, especially in neurophysiological studies. In particular, research is needed regarding response to *anger* and *fear*, which are important for both emotional processing and emotional regulation of people with ASD. Another potential avenue of further research is variations in musical stimuli used in studies. The majority of studies have relied on existing music from classic repertoire and film, with only a few selections crafted specifically for research use (Whipple et al. 2015). Additionally, the use of lyrics in emotional music is also understudied as well as the impact of pairing emotional music to other social-emotional

contexts such as movies. Many studies show relatively high levels of accuracy in emotion identification among participants possibly suggesting that the completion of the tasks is too easy. Finding ways to increase the difficulty of musical tasks may provide more variability from which group differences may arise. Only one study has investigated music-evoked emotions using stimuli which vary in level of emotional intensity (Bhatara et al. 2010). Bhatara et al. found that participants with ASD did not provide ratings with as much variability as those without ASD, but the participants with ASD also had lower verbal intellectual ability than those in the comparison group. Additional work varying level of intensity or study designs using congruent vs. incongruent emotional information (e.g., *happy* music with *angry* lyrics) may be possible ways to achieve different levels of difficulty to increase the variability within emotional identification accuracy.

The vast majority of research investigations up to this point have included people with intact intellectual ability. Similar to other areas of research, music-evoked emotion studies need to include people at all levels of the autism spectrum in order to understand the musical experience among those with minimal verbal ability. Preliminary results indicate that children and adolescents with ASD with verbal and cognitive skills ranging from the lower to higher ranges can accurately recognize music-evoked emotions (Dahary et al. 2018). Additionally, relatively little is known about the developmental trajectory of musical experience and ASD, although some research points to possible age-related differences (Stephenson et al. 2016). Longitudinal studies investigating music-evoked emotion at all points of development, from early childhood into older adulthood, would shed valuable light into this area. With a better understanding of the differences and similarities of processing of and responses to music-evoked emotion, we may have access to additional tools for the diagnosis and treatment of autism. One music-based autism diagnostic assessment tool for adults with developmental disabilities has already been developed (Bergmann et al. 2015), and there is potential for other applications of music therapy including

development of executive functioning skills in young children (Frischen et al. 2019). Furthermore, with improving technology of wearable physiological measurement devices, neural imagining, and behavioral strategies, the prospect of including a wide range of individuals is better than ever.

Many of these promising avenues are already being pursued (Dahary et al. 2018; Sivathanan et al. 2019), although there is room for more research to be done in this area. As with much autism-related research, studies of music-evoked emotion and ASD is lacking in larger, high statistical power studies. Multisite collaborations can help with this as well as researchers considering including musical stimuli and paradigms in larger clinical trials and lab-based research of emotion processing.

In sum, people with autism display generally intact accuracy and processing of music-evoked emotion. The use of music-evoked emotion has great potential for scientific and clinical benefit in autism spectrum disorder and remains a promising field for future endeavors.

References and Reading

- Allen, R., & Heaton, P. (2010). Autism, music, and the therapeutic potential of music in alexithymia. *Music Perception: An Interdisciplinary Journal*, 27(4), 251–261. <https://doi.org/10.1525/mp.2010.27.4.251>.
- Allen, R., Hill, E., & Heaton, P. (2009). ‘Hath charms to soothe . . .’: An exploratory study of how high-functioning adults with ASD experience music. *Autism*, 13(1), 21–41. <https://doi.org/10.1177/1362361307098511>.
- Allen, R., Davis, R., & Hill, E. (2013). The effects of autism and alexithymia on physiological and verbal responsiveness to music. *Journal of Autism and Developmental Disorders*, 43(2), 432–444. <https://doi.org/10.1007/s10803-012-1587-8>.
- Begeer, S., Koot, H. M., Rieffe, C., Meerum Terwogt, M., & Stegge, H. (2008). Emotional competence in children with autism: Diagnostic criteria and empirical evidence. *Developmental Review*, 28(3), 342–369. <https://doi.org/10.1016/j.dr.2007.09.001>.
- Bergmann, T., Sappok, T., Diefenbacher, A., Dames, S., Heinrich, M., Ziegler, M., & Dziobek, I. (2015). Music-based Autism Diagnostics (MUSAD) – A newly developed diagnostic measure for adults with intellectual developmental disabilities suspected of autism. *Research in Developmental Disabilities*, 43–44, 123–135. <https://doi.org/10.1016/j.ridd.2015.05.011>.
- Bhatara, A., Quintin, E. M., Levy, B., Bellugi, U., Fombonne, E., & Levitin, D. J. (2010). Perception of emotion in musical performance in adolescents with autism spectrum disorders. *Autism Research*, 3(5), 214–225. <https://doi.org/10.1002/aur.147>.
- Bhatara, A., Quintin, E. M., Fombonne, E., & Levitin, D. J. (2013). Early sensitivity to sound and musical preferences and enjoyment in adolescents with autism spectrum disorders. *Psychomusicology: Music, Mind, and Brain*, 23(2), 100–108. <https://doi.org/10.1037/a0033754>.
- Bird, G., & Cook, R. (2013). Mixed emotions: The contribution of alexithymia to the emotional symptoms of autism. *Translational Psychiatry*, 3, e285. <https://doi.org/10.1038/tp.2013.61>.
- Caria, A., Venuti, P., & de Falco, S. (2011). Functional and dysfunctional brain circuits underlying emotional processing of music in autism spectrum disorders. *Cerebral Cortex*, 21(12), 2838–2849. <https://doi.org/10.1093/cercor/bhr084>.
- Chenauksy, K., Norton, A., Tager-Flusberg, H., & Schlaug, G. (2016). Auditory-motor mapping training: Comparing the effects of a novel speech treatment to a control treatment for minimally verbal children with autism. *PLoS One*, 11(11), e0164930. <https://doi.org/10.1371/journal.pone.0164930>.
- Dahary, H., Sivathanan, S., & Quintin, E. M. (2018). *Comparing intensity ratings of emotions in music and faces by adolescents with autism spectrum disorder*. Presented at the International Society for Autism Research (INSAR) Annual Meeting, Rotterdam, Netherlands.
- Frischen, U., Schwarzer, G., & Degé, F. (2019). Comparing the effects of rhythm-based music training and pitch-based music training on executive functions in preschoolers. *Frontiers in Integrative Neuroscience*, 13. <https://doi.org/10.3389/fnint.2019.00041>.
- Gabrielsson, A., & Lindström, E. (2011). The role of structure in the musical expression of emotions. In P. N. Juslin & J. Sloboda (Eds.), *Handbook of music and emotion: Theory, research, applications*. Oxford: Oxford University Press.
- Gebauer, L., Skewes, J., Westphal, G., Heaton, P., & Vuust, P. (2014). Intact brain processing of musical emotions in autism spectrum disorder, but more cognitive load and arousal in happy vs. sad music. *Frontiers in Neuroscience*, 8. <https://doi.org/10.3389/fnins.2014.00192>.
- Geretsegger, M., Elefant, C., Mössler, K. A., & Gold, C. (2014). Music therapy for people with autism spectrum disorder. *Cochrane Database of Systematic Reviews*, 6. <https://doi.org/10.1002/14651858.CD004381.pub3>.
- Heaton, P., Hermelin, B., & Pring, L. (1999). Can children with autistic spectrum disorders perceive affect in music? An experimental investigation. *Psychological Medicine*, 29(6), 1405–1410. <https://doi.org/10.1017/s0033291799001221>.

- Heaton, P., Allen, R., Williams, K., Cummins, O., & Happé, F. (2008). Do social and cognitive deficits curtail musical understanding? Evidence from autism and down syndrome. *British Journal of Developmental Psychology*, 26(2), 171–182. <https://doi.org/10.1348/026151007X206776>.
- James, R., Sigafoos, J., Green, V. A., Lancioni, G. E., O'Reilly, M. F., Lang, R., . . . Marschik, P. B. (2015). Music therapy for individuals with autism spectrum disorder: A systematic review. *Review Journal of Autism and Developmental Disorders*, 2(1), 39–54. <https://doi.org/10.1007/s40489-014-0035-4>.
- Jones, W., Carr, K., & Klin, A. (2008). Absence of preferential looking to the eyes of approaching adults predicts level of social disability in 2-year-old toddlers with autism spectrum disorder. *Archives of General Psychiatry*, 65(8), 946–954. <https://doi.org/10.1001/archpsyc.65.8.946>.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809–816. <https://doi.org/10.1001/archpsyc.59.9.809>.
- Koelsch, S. (2014). Brain correlates of music-evoked emotions. *Nature Reviews Neuroscience*, 15(3), 170–180. <https://doi.org/10.1038/nrn3666>.
- LaGasse, A. B. (2017, February 20). Social outcomes in children with autism spectrum disorder: A review of music therapy outcomes. <https://doi.org/10.2147/PROM.S106267>.
- Lozier, L. M., Vanmeter, J. W., & Marsh, A. A. (2014). Impairments in facial affect recognition associated with autism spectrum disorders: A meta-analysis. *Development and Psychopathology*, 26(4pt1), 933–945. <https://doi.org/10.1017/S0954579414000479>.
- Pelphrey, K. A., Sasson, N. J., Reznick, J. S., Paul, G., Goldman, B. D., & Piven, J. (2002). Visual scanning of faces in autism. *Journal of Autism and Developmental Disorders*, 32(4), 249–261. <https://doi.org/10.1023/A:1016374617369>.
- Poquérusse, J., Pastore, L., Dellantonio, S., & Esposito, G. (2018). Alexithymia and autism Spectrum disorder: A complex relationship. *Frontiers in Psychology*, 9. <https://doi.org/10.3389/fpsyg.2018.01196>.
- Quintin, E.-M. (2019). Music-evoked reward and emotion: Relative strengths and response to intervention of people with ASD. *Frontiers in Neural Circuits*, 13. <https://doi.org/10.3389/fncir.2019.00049>.
- Quintin, E.-M., Bhatara, A., Poissant, H., Fombonne, E., & Levitin, D. J. (2011). Emotion perception in music in high-functioning adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 41(9), 1240–1255. <https://doi.org/10.1007/s10803-010-1146-0>.
- Schaefer, H.-E. (2017). Music-evoked emotions—Current studies. *Frontiers in Neuroscience*, 11. <https://doi.org/10.3389/fnins.2017.00600>.
- Sharda, M., Tuerk, C., Chowdhury, R., Jamey, K., Foster, N., Custo-Blanch, M., . . . Hyde, K. (2018). Music improves social communication and auditory–motor connectivity in children with autism. *Translational Psychiatry*, 8. <https://doi.org/10.1038/s41398-018-0287-3>.
- Sivathasan, S., Burack, J. A., & Quintin, E.-M. (2019). *Multimodal emotion recognition in children with autism spectrum disorders: Vocalizations are more informative than faces or music*. Presented at the International Society for Autism Research (INSAR) Annual Meeting, Montreal, Quebec, Canada.
- South, M., & Rodgers, J. (2017). Sensory, emotional and cognitive contributions to anxiety in autism spectrum disorders. *Frontiers in Human Neuroscience*, 11. <https://doi.org/10.3389/fnhum.2017.00020>.
- Stephenson, K. G., Quintin, E. M., & South, M. (2016). Age-related differences in response to music-evoked emotion among children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46(4), 1142–1151. <https://doi.org/10.1007/s10803-015-2624-1>.
- Uljarevic, M., & Hamilton, A. (2013). Recognition of emotions in autism: A formal meta-analysis. *Journal of Autism and Developmental Disorders*, 43(7), 1517–1526. <https://doi.org/10.1007/s10803-012-1695-5>.
- Whipple, C. M., Gfeller, K., Driscoll, V., Oleson, J., & McGregor, K. (2015). Do communication disorders extend to musical messages? An answer from children with hearing loss or autism spectrum disorders. *Journal of Music Therapy*, 52(1), 78–116. <https://doi.org/10.1093/jmt/thu039>.

Mutual Gaze

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Synonyms

Eye contact; Eye-to-eye gaze; Social gaze; Visual attention to eyes; Visual orienting to eyes

Definition

What Is It?

Mutual gaze occurs when two people make eye contact or look into each other's eyes. Mutual

gaze is an important part of social communication and perception of others' emotion states and is the one of the foundational skills necessary in the development of joint attention (George and Conty 2008; Morales et al. 2005; Saito et al. 2010; Senju and Johnson 2009). Mutual gaze has been described as "the most powerful mode of establishing a communicative link between humans" (Farroni et al. 2002).

When Does It Occur?

From birth, infants are innately motivated to gaze into their caregivers' eyes. In fact, the field of vision of newborns is approximately the distance required to make eye contact when held by an adult (Stern et al. 1985). Infants prefer to look at faces over other stimuli, especially faces that engage them in mutual gaze (Farroni et al. 2002). Eye-tracking studies have revealed that when looking at faces, even infants preferentially fixate on the eyes over other facial features (Mauer and Salapatek 1976).

Mutual Gaze and ASD

One of the most prominent features of autism spectrum disorder (ASD) is an atypical pattern of eye contact and reduced levels of mutual gaze with social partners (Klienmann et al. 2010). Although reduced levels of gaze behavior at 6 months was not found to be predictive of autism diagnosis at 24 months (Young et al. 2009), toddlers with autism do show abnormal gaze to the eye region and decreased shared affect (Chawarska et al. 2010). Eye-tracking data for children with ASD have shown active avoidance of direct eye contact through increased gaze shifts away from the eye area (Klienmann et al. 2010). The DSM-IV-TR includes reduced eye contact as one of the symptoms of ASD: "marked impairment in the use of multiple nonverbal behaviors (e.g. eye-to-eye gaze, . . .) to regulate social interaction and communication" (American Psychiatric Association 2000, p. 70).

There are currently four models that are used to explain the decreased levels of mutual gaze or eye contact in children with ASD:

1. *The affective hyperarousal model* which posits that gaze avoidance is an adaptive response for individuals with ASD who find focusing on the face and eyes of others to be strongly aversive
2. *The affective hypoarousal model* which posits that individuals with ASD experience hypoactivation of the amygdala, which interferes in the association of a positive social reward with making eye contact, resulting in reduced levels of eye contact
3. *The communicative intention detector model* that proposes that individuals with ASD engage in less eye contact because they do not understand the social salience of eye contact in understanding others' states
4. *The fast-track modulator model* which states that atypical eye contact in ASD could be caused either by an impairment in the subcortical face and eye contacting detecting route or in the network of cortical and subcortical structures specializing in the processing of social information (Senju et al. 2009)

See Also

► [Eye Gaze](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, DSM-IV-TR* (4th ed.). Washington, DC: Author. Text revision.
- Chawarska, K., Volkmar, F., & Klin, A. (2010). Limited attentional bias for faces in toddlers with autism. *Archives of General Psychiatry*, 67, 178–185.
- Dawson, G., Hill, D., Spencer, A., Galpert, L., & Watson, L. (1990). Affective exchanges between young autistic children and their mothers. *Journal of Abnormal Child Psychology*, 18, 335–345.
- Elsabbagh, M., Volein, A., Csibra, G., Homboc, K., Garwood, H., Tucker, L., et al. (2009). Neural correlates of eye gaze processing in the infant broader autism phenotype. *Biological Psychiatry*, 65, 31–38.
- Farran, D. C., & Kasari, C. (1990). A longitudinal analysis of the development of synchrony in mutual gaze in mother-child dyads. *Journal of Applied Developmental Psychology*, 11, 419–430.
- Farroni, T., Csibra, G., Simion, F., & Johnson, M. H. (2002). Eye contact detection in humans from birth. *Proceedings of the National Academy of Sciences of the United States of America*, 99, 9602–9605.

- George, N., & Conty, L. (2008). Facing the gaze of others. *Clinical Neurophysiology*, 38, 197–207.
- Gibson, J. J., & Pick, A. D. (1963). Perception of another's looking behavior. *The American Journal of Psychology*, 76, 386–394.
- Johnson, M. H., Bziurawiec, S., Ellis, H., & Morton, J. (1992). The effects of movement of internal features on infants' preferences for face-like stimuli. *Cognition*, 40, 1–19.
- Klienmann, D., Dziobek, I., Hatri, A., Steimke, R., & Heekeren, H. R. (2010). Atypical reflexive gaze patterns on emotional faces in autism spectrum disorders. *Journal of Neuroscience*, 30, 12281–12287.
- Kylliäinen, A., & Hietanen, J. K. (2006). Skin conductance responses to another person's gaze in children with autism. *Journal of Autism and Developmental Disorders*, 36, 517–525.
- Mauer, D., & Salapatek, P. (1976). Developmental changes in the scanning of faces by young infants. *Child Development*, 47, 523–527.
- Morales, M., Mundy, P., Crowson, M. M., Neal, A. R., & Delgado, C. E. F. (2005). Individual differences in infant attention skills, joint attention, and emotion regulation behaviour. *International Journal of Behavioral Development*, 29, 259–263.
- Ozonoff, S., Iosif, A., Baguio, F., Cook, I. C., Hill, M. M., Hutman, T., et al. (2010). A prospective study of the emergence of early behavioral signs of autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49, 256–266.
- Saito, D. N., Tanabe, H. C., Izuma, K., Hayashi, M. J., Morito, Y., & Komeda, H. (2010). “Stay tuned”: Inter-individual neural synchronization during mutual gaze and joint attention. *Frontiers in Integrative Neuroscience*, 4, 1–12.
- Senju, A., & Johnson, M. H. (2009). Atypical eye contact in autism: Models, mechanisms, and development. *Neuroscience and Biobehavioral Reviews*, 33, 1204–1214.
- Senju, A., Yaguchi, K., Tojo, Y., & Hasegawa, T. (2003). Eye contact does not facilitate detection in children with autism. *Cognition*, 89, B43–B51.
- Senju, A., Yaguchi, K., Tojo, Y., & Hasegawa, T. (2005). Deviant gaze processing in children with autism: An ERP study. *Neuropsychologia*, 43, 1297–1306.
- Stern, D. N., Hofer, L., Haft, W., & Dore, J. (1985). Affect attunement: The sharing of feeling states between mother and infant by means of inter-modal fluency. In T. M. Field & N. A. Fox (Eds.), *Social perception in infants* (pp. 323–378). Norwood: Ablex.
- Volkmar, F. R., & Mayes, L. C. (1990). Gaze behavior in autism. *Development and Psychopathology*, 2, 61–69.
- Young, G. S., Merin, N., Rogers, S. J., & Ozonoff, S. (2009). Gaze behavior and affect at 6 months: Predicting clinical outcomes and language development in typically developing infants and infants at risk for autism. *Developmental Science*, 12, 798–814.

Mutual Regulation

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Synonyms

Co-regulation; Emotion regulation; Emotional synchrony; Self-regulation

Definition

The construct of emotion regulation refers to “the extrinsic and intrinsic processes responsible for monitoring, evaluating and modifying emotional reactions” (Thompson 1994). Important to this definition is the emphasis on both intrinsic and extrinsic processes, which may include both internal or self-driven processes and external or mother-driven support. This mother-driven support of the child's emerging emotional processes is co-regulation.

The development of emotion regulation is a process that occurs over the first years of life. Beginning in infancy, children learn to regulate emotions, attention, and behaviors through the external scaffold of mothers. The process gradually becomes an internal, self-driven process (Kopp 1989). As children gain a greater ability to regulate these processes, they also show a greater ability to remain flexible while facing the ongoing social demands of their environment.

Mother-child interaction research has contributed to our knowledge of emotion co-regulation by emphasizing the dynamic and interpersonal nature of the construct. These studies have found that typical mothers and children are sensitive to each other's emotional states and that mothers regulate child emotional states by reading emotion signals and modulating child arousal (Field 1994). In addition, work in this area has found that the quality of interpersonal regulation between

mother and child predicts later social-emotional outcomes in the child, including self-control and social competence (Feldman et al. 1999).

Several studies have begun to examine the development of emotion regulation in children with autism. Bieberich and Morgan (2004) employed parent report measures to examine emotion regulation outcomes. In another study, emotion regulation and temperament in children with autism spectrum disorder was measured by behavioral coding of maladaptive and adaptive regulation strategies during a mildly frustrating delay task. Results showed that children with autism employed less adaptive strategies and a greater range of strategies compared to typical controls (Konstantareas and Stewart 2006). These findings are among the first empirical support for a specific disturbance in emotion self-regulation in children with autism.

A recent study explored the development of emotion co-regulation in young children with autism and their mothers within the context of an early social-communication intervention (Gulsrud et al. 2010). Results showed that children displayed significant distress in almost half of their interactions with their mothers, and these episodes of distress lasted on average 20% of the total length of the interaction. Children most frequently engaged in active strategies (tension release, avoidance, and distraction). Similarly, mothers most frequently engaged in active strategies (redirection of attention, prompting/helping, and physical strategies) during child negativity. These results suggest that both mothers and children are actively working together to regulate the child's emotions and that co-regulation is a viable construct for study in this population.

See Also

- [Co-regulation](#)
- [Emotion Regulation](#)
- [Self-regulation](#)
- [Temperament](#)

References and Reading

- Bieberich, A., & Morgan, S. B. (2004). Self-regulation and affective expression during play in children with autism or Down syndrome: A short-term longitudinal study. *Journal of Autism and Developmental Disorders*, 34, 439–448.
- Feldman, R., Greenbaum, C. W., & Yirmiya, N. (1999). Mother-infant affect synchrony as an antecedent to the emergence of self-control. *Developmental Psychology*, 35, 223–231.
- Field, T. (1994). The effects of mother's physical and emotional unavailability on emotion regulation. In N. A. Fox (Ed.), *The development of emotion regulation: Biological and behavioral considerations* (Monographs of the society for research in child development, Vol. 59, pp. 208–227). Chicago: University of Chicago Press.
- Gulsrud, A., Jahromi, L. B., & Kasari, C. (2010). The co-regulation of emotions between mothers and their children with autism. *JADD*, 34, 439–448.
- Konstantareas, M., & Stewart, K. (2006). Affect regulation and temperament in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 36, 143–154.
- Kopp, C. B. (1982). Antecedents of self-regulation: A developmental perspective. *Developmental Psychology*, 18, 199–214.
- Kopp, C. B. (1989). Regulation of distress and negative emotions: A developmental view. *Developmental Psychology*, 25, 343–354.
- Thompson, R. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development*, 59(2–3, Serial No. 240), 25–52

Mutually Acceptable Written Agreement (MAWA)

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Definition

A “mutually acceptable written agreement” is a binding document that is the result of a mediation session. Mediation is a negotiation session, typically used as an informal alternative to the adversary system, facilitated by a neutral third party who tries to help the parties in the dispute to reach a mutually acceptable written agreement.

The agreement can cover a wide range of issues including issues that would not be recognized in a court of law. There is some uncertainty as to whether a mutually acceptable written agreement is legally enforceable. Most states require that these agreements be reduced to writing and be signed by both parties, and many states require that the agreement be filed in court. Furthermore, there are often statutes that provide requirements for an agreement reached in a particular type of mediation setting. In the absence of legislation, one must look to common law contract principles as authority on whether the agreement is enforceable. These agreements may also be subject to agency law for the purpose of determining whether those entering into the agreement have the authority to do so.

See Also

- [Consent](#)
- [Liability](#)

References and Reading

Bush, B., & Richard, A. (2008). *Staying in orbit or breaking free: The relationship of mediation to the courts*

over four decades. Grand Forks: North Dakota Law Review.

D'Alo, G. E. (2003). *Accountability in special education mediation: Many a slip 'twixt vision and practice?* Cambridge, MA: Harvard Negotiation Law Review.

Feinberg, E., Breyer, J., & Moses, P. (2002). *Beyond mediation: Strategies for appropriate early dispute resolution in special education*. Eugene: The Consortium for Appropriate Dispute Resolution in Special Education (CADRE).

Parker, B. R. (1992). *What can be done to enforce mediation agreements*. Chicago: IADC. Defense Counsel Journal.

Thorpe, R. W., & Boyens, J. (1998). *Mediation settlement agreements: Legal, ethical, and practical issues*. New York: The International Institute for Conflict Prevention and Resolution. Alternatives to the High Cost of Litigation.

Myoclonic Spasms of Infancy

- [Infantile Spasms/West Syndrome](#)

Myo-inositol

- [Inositol: Definition](#)

Naltrexone

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Synonyms

Depade; Naltrexone hydrochloride; Revia

Indications

Alcohol dependence

Mechanisms of Action

Naltrexone is an opiate antagonist with minimal agonist activity.

Clinical Use (Including Side Effects)

Naltrexone is not currently approved for the treatment of ASD-related symptoms but enjoys some off-label use.

A series of early open-label studies had suggested significant improvements in self-injurious behaviors (SIB). The side effect profile was relatively benign.

Following early data, there have been a few small controlled studies (max sample size 41, Campbell et al. 1993) treating children between the ages of 2.8 and 19 years with naltrexone. A nice systematic review by Elchaar et al. in 2006 has summarized available data in ASD. Overall, the data does not support the use of naltrexone for the treatment of core symptom domains of ASD, despite original hypothesis regarding abnormalities in the endogenous opioid system in ASD (Panksepp 1979). However, preliminary data suggests that at least a subgroup of children with ASD may show improvements in self-injurious behaviors with naltrexone. Still there is no clarity regarding the dose (doses used vary from 0.5 mg/kg weekly to 2.35 mg/kg single dose) and/or duration of treatment. There is also lack of clarity about whether higher doses are more or less effective, with some studies suggesting that doses of 2 mg/kg/day or higher may have less selectivity for the opiate receptor, therefore competing with the opiate antagonism effect.

The most commonly reported side effect in ASD studies is sedation. The bitter taste of naltrexone may interfere with compliance. Naltrexone carries an FDA black box warning regarding hepatic insufficiency, but no elevation in liver enzymes has been reported so far in patients

with ASD. Gastrointestinal upset and two cases of panic attacks have also been reported.

At this point, it is hard to make a recommendation regarding naltrexone. If it were to be used, a reasonable dose range is between 0.5 and 2 mg/kg/day. Safety needs to be monitored, especially regarding the FDA black box warning. A large randomized controlled trial of naltrexone targeting SIB and exploring predictors of response is likely warranted.

See Also

- [Trexan](#)

References and Reading

- Campbell, M., Anderson, L. T., Small, A. M., Adams, P., Gonzalez, N. M., & Ernst, M. (1993). Naltrexone in autistic children: Behavioral symptoms and attentional learning. *Journal of the American Academy of Child & Adolescent Psychiatry*, 32, 1283–1291.
- Elchaar, G. M., Maisch, N. M., Augusto, L. M., & Wehring, H. J. (2006). Efficacy and safety of naltrexone use in pediatric patients with autistic disorder. *Annals of Pharmacotherapy*, 40, 1086–1095.
- Pansepp, J. (1979). A neurochemical theory of autism. *Trends in Biochemical Sciences*, 2, 174–177.

Naltrexone Hydrochloride

- [Naltrexone](#)

Naming

- [Label](#)

Nanocephaly

- [Microcephaly](#)

Narrative Analysis

- [Narrative Assessment](#)
- [Story Structure Decision Tree](#)

Narrative Assessment

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Synonyms

[Narrative analysis](#)

Definition

A narrative assessment is the analysis of a story told by an individual to assess language and communication. A narrative can be used to measure pragmatic language use because it involves recounting a personal experience or retelling a story to another individual. Narratives can be examined for their structure (e.g., sentence complexity, story components), content (e.g., use of mental state terms), and/or style of presentation (e.g., use of prosody, gestures, facial expressions).

See Also

- [Communication Assessment](#)
- [Pragmatic Communication](#)
- [Pragmatic Language Impairment](#)
- [Pragmatics](#)
- [Story Structure Decision Tree](#)
- [Strong Narrative Assessment Procedure \(SNAP\), Second Edition](#)

References and Reading

- Baixauli, I., Colomer, C., Roselló, B., & Miranda, A. (2016). Narratives of children with high-functioning autism spectrum disorder: A meta-analysis. *Research in Developmental Disabilities, 59*, 234–254.
- Diehl, J. J., Bennetto, L., & Young, E. C. (2006). Story recall and narrative coherence of high-functioning children with autism spectrum disorders. *Journal of Abnormal Child Psychology, 34*, 87–102.
- Losh, M., & Capps, L. (2003). Narrative ability in high-functioning children with autism or Asperger's syndrome. *Journal of Autism and Developmental Disorders, 33*, 239–251.
- Loveland, K., McEvoy, R., Tunali, B., & Kelley, M. L. (1990). Narrative story telling in autism and Down's syndrome. *British Journal of Developmental Psychology, 8*, 9–23.
- Tager-Flusberg, H. (1995). 'Once upon a rabbit': Stories narrated by autistic children. *British Journal of Developmental Psychology, 13*, 45–59.
- Young, E. C., Diehl, J. J., Morris, D., Hyman, S. L., & Bennetto, L. (2005). Pragmatic language disorders in children with autism: The use of two formal tests to distinguish affected children from controls. *Language, Speech, and Hearing Services in Schools, 36*, 62–72.

Narrative Comprehension

► Visual Narrative Comprehension

Narrative Language in ASD

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Definition

Narrative (i.e., storytelling) is a ubiquitous form of communication and primary means of sharing experiences with others. Organizing experiences

through narrative imposes temporal and causal order to events, providing meaning and interpersonal significance to experiences. Narrative is the primary means of sharing one's world of experience with others and plays a major role in forging interpersonal connections. In autism spectrum disorder (ASD), narrative abilities are impaired, particularly in open-ended conversational contexts that pervade daily interactions. Given the centrality of narrative to social interactions, characterizing the narrative profile in ASD is critical to understanding the origins of this group's difficulties with more complex social language use and intervention development.

The development of narrative competence draws on a number of linguistic, cognitive, and social cognitive skills that are impacted in ASD. Observed narrative impairment in ASD has been linked to deficits in structural language and social cognition (e.g., failure to identify the needs of one's listener) and may also be associated with executive function deficits (e.g., difficulties with attention, inhibition, and working memory) and a weak central coherence cognitive style (e.g., a focus on irrelevant details at the expense of the whole). Difficulties across these domains likely can impact both microstructural (e.g., length, complexity of word use, grammatical markers) and macrostructural (e.g., overall form and content) elements of narrative. For instance, impairments in structural language may result in reduced grammatical complexity, limiting one's ability to communicate temporality of events and to establish causal relationships between events. Further, social cognition is important for recognizing the emotional experiences of those involved in the story and adapting narrative style and content based on listener responses.

Although narrative abilities vary within individuals with ASD, particularly as a function of comorbid cognitive and language impairment, studies suggest a pattern of relative strengths in highly structured contexts (e.g., wordless picture books) and weaknesses in more open-ended conversational contexts that are typical of daily interactions. Generally, in more structured narrative tasks, individuals with ASD produce narratives

of similar length, with comparable rates of grammatical markers, emotion-state words, and basic narrative elements (e.g., main characters or setting) as those of typically developing peers. However, individuals with ASD demonstrate relative challenges in many aspects of narrative, with the most consistent difficulties observed in the use of evaluative devices, or statements that infuse psychological perspective within a narrative, such as a causal explanation of a character's emotion. Narratives of individuals with ASD have also been characterized by greater frequency of off-topic remarks, overly formal language, and reduced overall coherence. These difficulties have been observed across the lifespan of individuals with ASD, although research has primarily focused on childhood and early adolescence.

Given that difficulties with narration are most prominent in conversational contexts, it will be critical for future research to focus on the assessment of this form of narrative in order to elucidate difficulties most relevant to daily interactions. Similarly, a targeted intervention approach that focuses on developing conversational narratives has the potential to improve the social and communicative experiences of individuals with ASD.

See Also

- [Narrative Assessment](#)
- [Pragmatics](#)

References and Reading

- Capps, L., Losh, M., & Thurber, C. (2000). The frog ate a bug and made his mouth sad: Narrative competence in children with autism. *Journal of Abnormal Child Psychology*, 28, 193–204.
- Colle, L., Baron-Cohen, S., Wheelwright, S., & van der Lely, H. K. (2008). Narrative discourse in adults with high functioning autism or Asperger syndrome. *Journal of Autism and Developmental Disorders*, 38, 28–40.
- Losh, M., & Capps, L. (2003). Narrative ability in high-functioning children with autism or Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 33(3), 239–251.
- Ochs, E., & Capps, L. (2001). *Living narrative: Creating lives in everyday storytelling*. Cambridge: Harvard University Press.
- Tager-Flusberg, H., & Sullivan, K. (1995). Attributing mental states to story characters: A comparison of narratives produced by autistic and mentally retarded individuals. *Applied Psycholinguistics*, 16(3), 241–256.

Narrative Understanding

- [Visual Narrative Comprehension](#)

National Guideline for the Assessment and Diagnosis of Autism Spectrum Disorders in Australia

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Definition

The Australian National Guideline for the Assessment and Diagnosis of Autism Spectrum Disorders is a clinical Guideline comprising 70 recommendations designed to “create greater consistency in diagnostic practices across the country to ensure individuals on the autism spectrum and their families can receive the optimal clinical care.”

Historical Background

Australia is a high-income country with a population of approximately 25 million people spread over a geographical area around the same size as the USA or continental Europe. The prevalence of ASD in Australia is estimated between 1 and 2.5% (Randall et al. 2016).

The Australian system of government includes a central federal government and six states which,

together with two self-governing territories, have their own constitutions, parliaments, governments, law, as well as systems for health, education, and community services, including those impacting individuals with disability and autism spectrum disorder (ASD). Local government is the third tier of government in Australia, with each city or municipal area providing services for individuals with disability, including those with ASD.

As previous report commissioned by the Cooperative Research Centre for Living with Autism (Autism CRC) and the Commonwealth Department of Social Services, variability between states and territories in diagnostic practices and standards complicates the provision of ASD diagnosis in Australia (Taylor et al. 2016). For example, some states require diagnostic evaluations to be conducted by multi-disciplinary teams, while in other states, diagnostic decisions can be made by a sole clinician. Disparity in resources and in the availability of trained clinicians between urban and rural or remote areas was highlighted as a further challenge. An additional source of fragmentation is the difference in diagnostic standards between the health, education, and disability public services at the state and federal level, resulting in the risk that diagnostic decisions provided by one service (e.g., health system) would not be recognized by another system (e.g., the school system). As highlighted in the report, uneven diagnostic standards involve the risk of unequal service provision.

Current Knowledge

In response to the challenges highlighted in the 2014 report, the National Disability Insurance Agency (an agency of the Australian federal government) sponsored the creation of the first Australian National Guideline for the Assessment and Diagnosis of Autism Spectrum Disorders, which was developed by the Autism CRC with the stated purpose to “create greater consistency in diagnostic practices across the country to ensure individuals on the autism spectrum and their

families can receive the optimal clinical care.” The specific objectives were to develop a guideline that:

1. Describes a rigorous framework for accurately determining whether an individual meets diagnostic criteria for ASD
2. Outlines a comprehensive approach to identify related support needs
3. Contains sufficient flexibility to apply to the assessment of a child, adolescent, or adult of any age, gender, cultural or language background, communication or intellectual capacity, and medical complexity, living anywhere in Australia
4. Describes a feasible process for clinical service providers to administer across the full breadth of community settings in Australia, including public and private healthcare settings
5. Meets the needs and expectations of individuals being assessed and their caregivers

Published in 2018, the guideline content was developed by an expert committee chaired by Andrew Whitehouse (Whitehouse et al. 2018). The expert committee was responsible for conducting a detailed research process that collected and collated new and existing scientific, clinical, and consumer knowledge and generating clinical recommendation that would best apply to the local context.

The guideline is notable for the extensive consultation with a range of stakeholders, including family advocates and self-advocates, and the extensive research to assess the evidence. The development process included a steering committee, which comprised members of the national peak bodies of professions commonly involved in the diagnosis and management of individuals on the autism spectrum, as well as members of service providers and advocacy groups (including adults with a diagnosis of autism). Face-to-face consultation workshops were also held in each state of Australia, as well as a “virtual” workshop held via videoconference that focused on issues for rural consumers. An open feedback process also allowed any individual in Australia to comment on a draft version of the guideline.

The guideline was developed in accordance with six guiding principles:

1. Evidence based: Clinical diagnosis is most effective and safe when it is based on rigorous scientific evidence.
2. Individual and family centered: The primary sources of information required during an assessment of ASD concerns are the individual undergoing assessment and their family members (most notably, caregivers and support people).
3. Holistic framework: An individual is far more than the autistic behaviors, and an exclusive focus on the evaluation of ASD behaviors during the diagnostic process will fail to provide an adequate clinical appraisal of the individual.
4. Strengths focused: The strengths of an individual and their family are as important for service delivery as identifying their challenges.
5. Equity: All individuals, regardless of age, gender, cultural background, socioeconomic status, or geographical location, must be able to access a timely and rigorous assessment.
6. Lifespan perspective: People continue to grow and change throughout their lives as they are faced with new tasks, challenges, and opportunities, and consideration in clinical decision-making of the lifetime of a client is critical to providing optimal clinical care.

There are several key features of the guideline. First, the guideline is notable for the adoption of a person-centered approach, including the indication that the child's profile of strengths and needs, rather than the diagnostic status alone, should be used to make decisions on treatment and services.

Second, the guideline adopts a multistage approach to diagnosis. This includes a comprehensive needs assessment (including an assessment of functioning and medical evaluation), followed by a diagnostic evaluation by a single clinician and the involvement of a larger diagnostic team for more complex cases.

Third, the guideline provides recommendations around how, where, and when each stage of the diagnostic process should occur and also the competencies for professionals involved in the diagnostic process.

Finally, the guideline provides a detailed summary of the latest scientific and clinical knowledge about how the behaviors used to diagnose autism can vary according to certain factors. These factors include age, intellectual and/or communication capacity, sex and/or gender, culturally and linguistically diverse backgrounds, and other complex psychosocial factors.

Each of the 70 clinical recommendations is linked to an individual table that describes the evidence underpinning a given recommendation from various scientific, clinical, or community sources.

In July 2018, the guideline was endorsed by the peak organization in Australia for health evidence, the National Health and Medical Research Council, as representing optimal clinical care.

Future Directions

The Australian government will release a nationwide implementation plan for the guideline in the second half of 2019. The guideline is to be updated every 5 years, with the next update scheduled in 2023.

See Also

► [Australia and Autism](#)

References and Reading

- Randall, M., Sciberras, E., Brignell, A., Ihsen, E., Efron, D., Dissanayake, C., & Williams, K. (2016). Autism spectrum disorder: Presentation and prevalence in a nationally representative Australian sample. *Australian and New Zealand Journal of Psychiatry*, 50(3), 243–253.
- Taylor, L. J., Eapen, V., Maybery, M. T., Midford, S., Paynter, J., Quarmby, L., . . . Whitehouse, A. J. O. (2016). Diagnostic evaluation for autism spectrum disorder: A survey of health professionals in Australia. *BMJ Open*, 6(9). <https://doi.org/10.1136/bmjopen-2016-012517>.
- Whitehouse, A. J. O., Evans, K., Eapen, V., & Wray, J. (2018). *A national guideline for the assessment and diagnosis of autism spectrum disorders in Australia*. Brisbane: Cooperative Research Centre for Living with Autism.

National Research Council

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Major Areas or Mission Statement

The National Research Council (NRC) is part of the National Academy of Sciences in the United States. It is a private, nonprofit society of distinguished scholars engaged in scientific research, mandated by Congress to advise the US federal government on scientific matters. The relevance to autism is that the NRC published a report in 2001 integrating scientific evidence concerning the effects and features of early intervention programs for children with autism. This report was written by an interdisciplinary committee of experts from education, psychology, psychiatry, neurology, speech-language pathology, and augmentative communication convened by the NRC for several years specifically for this project. The charge to the committee was to review the scholarly literature, including research, theory, and policies, to create a framework for evaluating the scientific evidence concerning educational interventions for children under age 8 who have autism. The charge also included specific suggestions to consider the role of early intervention, diagnosis and classification, inclusion, assistive technology, and the rights of children with autism within the United States (Individuals with Disabilities Education Act).

Landmark Contributions

The NRC report is similar to clinical guidelines provided by organizations in other countries (such as by the National Health Service in the United Kingdom) and from particular groups within the United States (e.g., National Autism Standards project; Wilczynski et al. 2009; American

Academy of Neurology; Filipek et al. 2000) but differed from some other reports in that the NRC committee members were well-known scholars from a range of disciplines and theoretical perspectives appointed by a panel of eminent scientists at the Academy, as opposed to members of a specific professional organization (e.g., American Academy of Pediatrics 2007) or a particular bureaucratic entity (e.g., National Health Service or New York Board of Health; New York State Department of Health Early Intervention Program 2005) or a self-nominated group (National Autism Standards; Wilczynski et al. 2009). The committee agreed on standards with which they would evaluate research. Public meetings were held in which researchers and agencies presented information; much of this information is summarized in a series of papers published in the *Journal of Autism and Developmental Disorders* in 2002 (Baranek 2002; Goldstein 2002; Horner et al. 2002; Kasari 2002; Mandlawitz 2002; McConnell 2002; Turnbull et al. 2002; Wolery and Garfinkle 2002). The committee achieved consensus for a number of recommendations which helped to shape educational services for young children with autism in the United States. The NRC report has been used by school systems and state agencies as well as the federal government and families as a guideline for scientifically based approaches to diagnosis, education, treatment, and policies.

Major recommendations include (see National Research Council [NRC] 2001):

1. Children with any autism spectrum disorder (autism, Asperger syndrome, atypical autism, PDD-NOS, childhood disintegrative disorder, Rett syndrome), regardless of level of severity or function, should be eligible for special education services within the category of autism, as appropriate to the child's needs.
2. Early identification and screening for autism, as is done for vision and hearing, should be promoted through research and training of first-line professionals.
3. Children should receive multidisciplinary assessments as soon as possible after identification. Follow-up assessments 1–2 years after the initial evaluation should be considered

- standard because of the many changes that occur during preschool years in autism.
4. Educational approaches to autism must include provision of information to families as well as ongoing consultation and individualized problem-solving and an opportunity to learn techniques for teaching their children new skills and reducing difficult behaviors. Families' concerns and perspectives should help shape educational planning.
 5. Ongoing measurement of treatment objectives and progress must be documented frequently and interventions adjusted accordingly. Objectives should be accomplished within a year, and interventions or objectives should be changed if there is little progress in 3 months. Objectives should be behaviors that are anticipated to affect a child's participation in education, the community, and/or the family and include generalization across environments.
 6. Educational services should begin as soon as a child is suspected to have an autism spectrum disorder. Services should include a minimum of 25 h a week, 12 months a year in which a child is engaged in systematically planned, developmentally appropriate educational activities directed toward identified objectives. What constitutes these hours and how structured the interventions are may vary dramatically depending on the child's age and abilities, the priorities, and supports available to a family within a community but should be specified individually for each child.
 7. Priorities of focus are functional spontaneous communication, social instruction delivered throughout the day in various settings, cognitive development, play, and academics (when appropriate). As much as is appropriate for an individual child, specialized instruction should take place in a setting in which ongoing positive social interactions can occur with typically developing children.
 8. Coordinated, systematic strategies should be developed to fund interventions in local communities and schools so that the cost is not borne by parents, caregivers, or local school systems and so that there is continuity of care.
 9. A federal joint agency task force should be created to address policy issues related to autism spectrum disorders (see US Interagency Autism Coordinating Council; <http://iacc.hhs.gov/>). It was recommended to the Office of Special Education Programs that personnel preparation programs for teaching of children with autism be accelerated.
 10. Standards for adequate description and design for intervention studies were recommended in order to allow more useful interpretation of results, particularly given the range of skills of children with autism. A call was made for stricter requirements to tie measures of efficacy to program development in autism.
 11. Comparisons of different interventions were prioritized as well as the development of research designs that identify the "active ingredients" of interventions and that consider interactions between child and family variables and the effectiveness of different approaches.

Major Activities

The NRC Committee on the Effectiveness of Early Intervention was a committee convened only for this specific project and so is no longer in effect. The National Research Council, along with the Institute of Medicine and National Academy of Engineering, continues to provide reports and makes recommendations as charged by the National Academy of Science and the US federal government on other issues.

References and Reading

American Academy of Pediatrics. (2007). *Autism: Caring for children with autism spectrum disorders: A resource toolkit for clinicians*. ElkGrove Village: Author.

- Baranek, G. T. (2002). Efficacy of sensory and motor interventions for children with autism. *Journal of Autism and Developmental Disorders*, 32(5), 397–422.
- Filipek, P. A., Accardo, P. J., Ashwal, S., Baranek, G. T., Cook, E. H., Jr., Dawson, G., et al. (2000). Practice parameter: Screening and diagnosis of autistic spectrum disorders – Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Child Neurology Society. *Neurology*, 55(4), 468–479.
- Goldstein, H. (2002). Communication intervention for children with autism: A review of treatment efficacy. *Journal of Autism and Developmental Disorders*, 32(5), 373–396.
- Horner, R. H., Carr, E. G., Strain, P. S., Todd, A. W., & Reed, H. K. (2002). Problem behavior interventions for young children with autism: A research synthesis. *Journal of Autism and Developmental Disorders*, 32(5), 423–446.
- Kasari, C. (2002). Assessing change in early intervention programs for children with autism. *Journal of Autism and Developmental Disorders*, 32(5), 447–461.
- Koegel, L. K., LaZebnik, C., Stone, W. L., DiGeronimo, T. F., Smerling, K., & Notbohm, E. (2008). *Autism speaks first 100 days kit*. Retrieved Accessed 8 June 2012 from <http://www.autismspeaks.org/family-services/tool-kits/100-day-kit>
- Mandlawitz, M. R. (2002). The impact of the legal system on educational programming for young children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 32(5), 495–508.
- McConnell, S. R. (2002). Interventions to facilitate social interaction for young children with autism: Review of available research and recommendations for educational intervention and future research. *Journal of Autism and Developmental Disorders*, 32(5), 351–372.
- National Research Council. (2001). *Educating children with autism*. Committee on Educational Interventions for Children with Autism (C. Lord & J. P. McGee, Eds.). Washington, DC: National Academy Press.
- New York State Department of Health Early Intervention Program. (2005). *Report of the guideline recommendations: Autism/pervasive developmental disorders, assessment and intervention for young children (age 0–3 years)*. Retrieved Accessed 8 June 2012 from http://www.health.ny.gov/community/infants_children/early_intervention/disorders/autism/
- Turnbull, H. R., III, Wilcox, B. L., & Stowe, M. J. (2002). A brief overview of special education law with focus on autism. *Journal of Autism and Developmental Disorders*, 32(5), 479–493.
- Wilczynski, S., Green, G., Ricciardi, J., Boyd, B., Hume, A., Ladd, M., et al. (2009). *National Standards Report: The national standards project – addressing the need for evidence-based practice guidelines for autism spectrum disorders*. Randolph: The National Autism Center. Retrieved Accessed 8 June 2012 from <http://www.nationalautismcenter.org/pdf/NAC%20Standards%20Report.pdf>
- Wolery, M., & Garfinkle, A. N. (2002). Measures in intervention research with young children who have autism. *Journal of Autism and Developmental Disorders*, 32(5), 463–478.

Natural Environment

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Definition

Natural Environment Intervention (NEI) or Natural Environment Training (NET) is a language intervention approach for children with autism that was developed by Sundberg and Partington (1999) based upon Skinner's Analysis of Verbal Behavior (1957). NET represents an extension of ABA and discrete trial training approaches. In a manner similar to Pivotal Response Training and Milieu Language Teaching, NET emphasizes the use of intrinsically motivating materials, focuses on the child's immediate interests (following the child's lead), and teaches in the child's everyday environment. This type of approach addresses child motivation and generalization issues by using materials that are reinforcing for the child and teaching within the same contexts that skills will be used. In addition, by making available materials that are reinforcing to the child, NET focuses on allowing the child to initiate teaching episodes to improve manding (requesting) and to increase the initiation of spontaneous communication attempts.

Natural History

- [Course of Development](#)
- [Longitudinal Research in Autism](#)

Natural Language Paradigm

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Definition

The Natural Language Paradigm (Koegel et al. 1987) is a teaching approach targeting verbal language acquisition for children with severe language deficits associated with autism. Compared to more structured behavioral treatments (e.g. discrete trial teaching), this loosely structured intervention aims to create a learning environment for the child that more closely resembles a natural language speaking situation and apply teaching approaches based on operant conditioning in that environment. This combination is intended to increase and generalize the child's use of communicative speech across settings.

Historical Background

NLP has its origins in work on incidental teaching conducted in the 1960s (e.g., Hart and Risley 1968, 1974). In the 1980s, Risley and McGee (McGee et al. 1983, 1985) began studying ways to adapt incidental teaching for children with autism. Combining this work and research by Koegel, Schreibman, and others on the unique learning styles of these children (e.g., a tendency to focus on only one component of a complex stimulus), Koegel and colleagues developed the Natural Language Paradigm. They presented it as an alternative to more structured operant teaching procedures (e.g. Hewett 1965; Lovaas 1966, 1977). Although there was evidence that these structured procedures were effective in teaching vocabulary and other language skills, many argued that they did not demonstrate sufficient generalized improvement

in functional speech. In an effort to address this inadequacy, researchers started to explore other ways to teach communication, such as sign language and symbol language. This shortcoming also served as the impetus for NLP. As the treatment evolved, the developers focused increasingly on enhancing children's motivation and later recast NLP as Pivotal Response Treatment or PRT (Koegel et al. 1989, 1999).

Rationale or Underlying Theory

The rationale for the Natural Language Paradigm is largely derived from past research indicating that children with autism spectrum disorders often lack the motivation necessary to communicate or learn new skills (e.g. Dunlap 1984; Dunlap and Egel 1982). The intervention strategies, such as the use of reinforcers directly related to the communication (e.g., giving a child access to an item that he or she has requested), the reinforcement of all attempts to communicate, the frequent variation and sharing of tasks and stimulus materials, and the use of incidental teaching all are designed to contribute to increasing the child's motivation to interact and communicate with the therapist. The presumption is that, once the child understands, he or she is able to control access to a desired item or situation through initiating and responding to verbal communication, his or her motivation to use language increases.

Additionally, the developers of NLP regard each of these strategies as a key component of the language acquisition process that typically developing children go through. By refocusing the treatment on more naturally occurring speech interactions, rather than structured or didactic behavioral teaching methods, NLP aims to increase the generalization of language skills to settings outside of the clinic (Koegel et al. 1987).

Goals and Objectives

The intervention is designed to increase the child's use of spontaneous and appropriate language, encourage the generalization of these skills

to other settings, and increase social interactions and child-initiation of activities.

Treatment Participants

Ideal treatment participants meet criteria for autism as defined in the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association 1994) and demonstrate severe language deficits and delays associated with the disorder. These children are generally classified as nonverbal, meaning they produce no spontaneous words or utterances, but may produce other types of vocalizations (Koegel et al.). The participants in the few studies specifically examining the NLP have been children between 4 and 10 years of age (Koegel et al.; Delprato 2001).

Treatment Procedures

NLP is designed as an individualized therapy that closely approximates the interactions and components present in a natural speaking situation. To accomplish this, the therapist presents a series of play trials using several stimulus items that have been previously selected by the child. These items are varied between trials and should be familiar to the child from his or her everyday environment. At the beginning of each trial, the therapist presents an item and then models appropriate verbal responses and play actions with the stimulus items to prompt the child to use speech. All of the child's attempts at verbal communication are repeated and expanded upon by the therapist and reinforced with praise and opportunities to play with the stimulus item.

Efficacy Information

Due to the relatively brief existence of the NLP in its original form, efficacy information is somewhat limited. In the original comparative study, each subject participated in both the analog teaching condition and the NLP condition consecutively. Koegel and colleagues reported that subjects, after treatment with the NLP, demonstrated “steady

and durable increases in both immediate and deferred utterances,” while these same subjects showed less, if any, improvement in these areas while in the analog teaching condition. More importantly, these gains were maintained and generalized to situations outside of the clinical setting by subjects treated with the NLP, but not by those in the analog teaching condition. Later studies comparing naturalistic language approaches to traditional behavior treatments reported similar results (Delprato 2001), which served to confirm the findings from the initial study by Koegel et al. However, because the studies on NLP have involved on a small number of subjects and focused on a very narrow range of communication skills, general conclusions cannot be drawn regarding the relative efficacy of NLP compared to other teaching approaches (Goldstein 2002, JADD).

Outcome Measurement

Treatment outcomes for the NLP intervention have most often been measured using spontaneous language samples, or “probes,” throughout the course of treatment. Probes assess the total number of utterances emitted by the child over a specified period of time and are conducted in a variety of settings, including intervention sessions, break periods, and during free play. The child's utterances are coded and categorized as one of three types: imitative, deferred imitation, or spontaneous (Koegel et al.).

Qualifications of Treatment Providers

In Koegel and colleagues' initial study, the clinicians administering the NLP intervention were supervised hearing and speech students with advanced training in behavior modification and language treatment.

See Also

- [Incidental Teaching](#)
- [Pivotal Response Training](#)

References and Reading

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.
- Delprato, D. (2001). Comparisons of discrete-trial and normalized behavioral language intervention for young children with autism. *Journal of Autism and Developmental Disorders*, 31(3), 315–325.
- Dunlap, G. (1984). The influence of task variation and maintenance tasks on the learning and affect of autistic children. *Journal of Experimental Child Psychology*, 37, 41–46.
- Dunlap, G., & Egel, A. L. (1982). Motivational techniques. In R. L. Koegel, A. Rincover, & A. L. Egel (Eds.), *Educating and understanding autistic children*. San Diego: College-Hill Press.
- Goldstein, H. (2002). Communication intervention for children with autism: A review of treatment efficacy. *Journal of Autism and Developmental Disorders*, 32(5), 373–396.
- Hart, B., & Risley, T. (1968). Establishing use of descriptive adjectives in the spontaneous speech of disadvantaged preschool children. *Journal of Applied Behavior Analysis*, 1, 109–120.
- Hart, B., & Risley, T. (1974). Using preschool materials to modify the language of disadvantaged children. *Journal of Applied Behavior Analysis*, 7(2), 243–256.
- Hewett, F. M. (1965). Teaching speech to an autistic child through operant conditioning. *Journal of Orthopsychiatry*, 35, 927–936.
- Koegel, R. L., O'Dell, M. C., & Koegel, L. K. (1987). A natural language teaching paradigm for nonverbal autistic children. *Journal of Autism and Developmental Disorders*, 17(2), 187–200.
- Koegel, R. L., Schriebman, L., Good, A., Cerniglia, L., Murphy, C., & Koegel, L. K. (1989). *How to teach pivotal behaviors to children with autism: A training manual*. Santa Barbara: California.
- Koegel, L. K., Koegel, R. L., Harrower, J. K., & Carter, C. M. (1999). Pivotal response intervention I: Overview of approach. *Journal of the Association for Persons with Severe Handicaps*, 24(3), 174–185.
- Laski, K. E., Charlop, M. H., & Schreibman, L. (1988). Training parents to use the Natural Language Paradigm to increase their autistic children's speech. *Journal of Applied Behaviour Analysis*, 21(4), 391–400.
- Lovaas, O. I. (1966). A program for the establishment of speech in psychotic children. In J. K. Wing (Ed.), *Early childhood autism*. Boston: Houghton Mifflin.
- Lovaas, O. I. (1977). *The autistic child: Language development through behaviour modification*. New York: Irvington.
- McGee, G. G., Krantz, P. J., Mason, D., & McClannahan, L. E. (1983). A modified incidental-teaching procedure for autistic youth: Acquisition and generalization of receptive object labels. *Journal of Applied Behavior Analysis*, 16(3), 329–338.
- McGee, G. G., Krantz, P. J., & McClannahan, L. E. (1985). The facilitative effects of incidental teaching on prepositional use by autistic children. *Journal of Applied Behavior Analysis*, 18(1), 17–31.

Naturalistic Behavioral Intervention

► Pivotal Response Training

Naturalistic Interventions

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Definition

Naturalistic interventions are behavior teaching procedures that occur in the context of naturally occurring activities (Koegel et al. 1987; McGee et al. 1984; Vismara and Rogers 2010). Intervention procedures embed teaching opportunities within the environment where the skills will be needed (Vismara & Rogers).

Historical Background

Individuals with an autism spectrum disorder exhibit deficits in communication, social interaction, and restricted and repetitive behavior (American Psychiatric Association [APA] 2000). Intensive early intervention programs have been shown to produce positive outcomes in a large percentage of the children diagnosed as having autism (Paul 2008; Harper et al. 2008). However, Carr (1983) reviewed behavior modification procedures for language instruction of children with autism and identified a weakness in many operant language programs. In intervention strategies employed in highly structured environments, treatment gains that were observed in the clinic

rarely generalized to other settings and behaviors were not often maintained (Miranda-Linne and Melin 1992). Many operant language programs also frequently resulted in cue dependency, rote responding, and little spontaneity of the target behavior (Miranda-Linne & Melin). Furthermore, treatment programs that did succeed in improving language skills required a great amount of time for even a minimal vocabulary (Koegel et al. 1987).

In response to these difficulties, many researchers investigated new behavioral interventions. In a study by Koegel et al. (1987), the effects of a traditional analog approach were compared to a natural language teaching condition (Koegel and Koegel 2006). The analog approach consisted of many successive trials, with each item chosen and presented by the clinician until the child reached a specific criterion (Koegel et al. 1987). In the natural language paradigm, procedures were modified to follow a more natural language-speaking situation and incorporated child choice, maintenance tasks, rewarding attempts to respond, and natural reinforcers directly related to the task (Koegel et al.). It was shown that children in the natural language condition had a higher rate and accuracy of correct responding, a greater increase in spontaneous utterances, and a greater generalization of language skills outside of the clinic setting (Koegel and Koegel 2006).

In 1986, the Congress issued a federal mandate PL 99-457 to provide early intervention for young children with disabilities in the least restrictive and most typical environments of the children (Kaiser and Trent 2007). Subsequent research has expanded the benefits of naturalistic interventions and has addressed the limitations of analog procedures that occur in highly structured settings (Koegel et al. 1987; Warren and Gazdag 1990). Data has shown that naturalistic interventions result in a higher generalization and maintenance of target behaviors (Barnet et al. 1993; Koegel et al. 1987, 1998; Miranda-Linne and Melin 1992; Vismara and Rogers 2010). These results have influenced current treatment programs to include procedures across multiple settings and within the child's natural environment in order to

effectively increase language, socialization, and behavioral skills for children with autism (Vismara & Rogers).

Rationale or Underlying Theory

Naturalistic interventions embed procedures in the context of naturally occurring activities and have shown to increase generalization, create more spontaneity, and improve the maintenance of the target behavior (Barnet et al. 1993; Ingersoll et al. 2007; Koegel et al. 1987, 1998; Miranda-Linne and Melin 1992; Vismara and Rogers 2010). As opposed to analog procedures that are directed by the clinician, involve drilling of the target behavior until acquisition is reached, and incorporate arbitrary reinforcers, natural interventions manipulate traditional variables (Koegel et al. 1987; Koegel and Koegel 2006). In naturalistic interventions, stimulus items are functional and chosen by the child, communicative attempts are rewarded, and natural reinforcers that directly relate to the task are employed (Koegel et al.; Koegel and Williams 1980; Miranda-Linne and Melin 1992). Procedures are implemented in the environment where the skills will be needed (i.e., the home, school, or community), so gains are rapidly produced, able to be generalized, and maintained after intervention (Brown and Odom 1995; Koegel and Koegel 2006; McGee et al. 1984).

Goals and Objectives

Naturalistic intervention procedures were developed with the following goals:

1. Embed treatment procedures in the context of naturally occurring activities and incorporate child choice, task variation, rewarding attempts, and direct and natural reinforcers to increase acquisition of language skills (Kaiser and Trent 2007; Koegel et al. 1987).
2. Improve the generalization and spontaneous use of target language skills across various settings and with typical peers and individuals

- in the child's daily routine (Hart and Risley 1975; McGee et al. 1984).
3. Increase the maintenance of treatment gains following intervention (Mirenda-Linne and Melin 1992).
 4. Create a nonaversive treatment program and improve the affect of children with autism and their treatment providers (Vismara and Rogers 2010).

Treatment Participants

Children with autism receive naturalistic interventions for deficits in social interaction, communication skills, and appropriate behavior (Williams and Williams 2010). A unique aspect of naturalistic interventions is the high level of participation of parents, paraprofessionals, and typical peers in the child's natural environment (Kaiser and Trent 2007; Kalyva and Avramidis 2005; Koegel and Koegel 2006). The procedures are designed for children with autism to receive treatment through interactions with individuals in their everyday life. Research shows that intensive intervention throughout the day is effective to increase the social, communication, and behavioral skills of children with autism; however, resources can be limited (Paul 2008). In order for children with autism to receive "round-the-clock" intervention services, the individuals that they interact with on a daily basis must be trained to implement treatment procedures (Barnet et al. 1993). In naturalistic intervention programs, parents, educators, and typical peers are trained in order to increase the quantity and availability of intervention across natural settings (Brown and Odom 1995; Koegel et al. 1996).

Parents are active participants in naturalistic interventions (Koegel et al. 1978, 1996). Research has shown that parents of children with autism can effectively implement behavioral, social, and communication procedures (Albert and Kaiser 1992; Koegel and Koegel 2006). Naturalistic treatment programs often incorporate a parent education component to teach caregivers how to employ intervention procedures and embed opportunities into the child's daily living routine

(Koegel and Koegel 2006). Effective parent participation can increase positive outcomes as well as have a collateral effect on family interactions (Koegel et al. 1996).

Paraprofessionals and typical peers are also significant contributors to natural intervention procedures. Increasing the inclusion of children with autism in regular classrooms and creating opportunities to engage in interactions with typical peers have been shown to increase the child's social development (Brown and Odom 1995; Fisher and Meyer 2002). In naturalistic intervention programs, teachers and counselors are trained in the treatment procedures and prompt typically developing children to engage in verbal interaction and social activities with children with autism (Koegel and Koegel 2006).

Parents, paraprofessionals, teachers, and typical peers all collaborate as key participants in naturalistic intervention procedures for children with autism.

Treatment Procedures

Naturalistic interventions use treatment procedures employed in the context of naturally occurring activities (McGee et al. 1984). Procedures are implemented in inclusive environments, and treatment strategies are embedded into everyday activities across multiple settings (Vismara and Rogers 2010). There are numerous teaching programs that use naturalistic interventions, and all incorporate the following procedural components or variations thereof. Treatment protocols take place in the child's natural environment (i.e., home, school, and community), and procedures are based on the foundation of child choice and the use of direct and natural reinforcers (Gillum et al. 2003; Kaiser and Trent 2007; Koegel et al. 1987, 1998; Koegel and Williams 1980). Common naturalistic methods to increase language acquisition are milieu therapy, incidental teaching, and pivotal response treatment (Hart and Risley 1975; Kaiser and Trent 1992; Koegel and Koegel 2006).

Naturalistic treatment programs are designed to fit each child's individual routine, and procedures incorporate child choice of stimulus

materials (Hart and Risley 1975; Kaiser and Trent 1992; Koegel et al. 1998). Child choice refers to following the child's lead and using child-preferred stimulus items, topics, toys, and interests (Koegel et al. 1999; Thiemann and Warren 2004; Yoder et al. 1995). Instead of the traditional approach in which the clinician arbitrarily selects a stimulus item, naturalistic procedures involve the clinician presenting stimulus items that the child selected from a pool of items (Koegel et al. 1987). The treatment provider uses the child's eye gaze, reaching, touching, etc., to determine the preferred items and uses those items in the intervention. Compared to the teacher selecting stimulus items, child choice of toys results in higher levels of engagement with toys, which creates a foundation from which more complex play behaviors can be taught (Reinhartsen et al. 2002). Furthermore, child choice has been shown to decrease problem behavior as well as increase autonomy (Reinhartsen et al.).

In addition to following the child's lead, naturalistic interventions incorporate direct and natural rewards (Koegel and Williams 1980; Williams et al. 1981). This means that the child is immediately reinforced with a reward that is directly related to the language opportunity (Koegel and Koegel 2006). For example, if the child makes an attempt to say "milk," then he or she will be immediately be given the milk so that a connection is made between their verbalization and the desired item (Koegel and Koegel 2006). This varies from the traditional approach which rewarded the child with an arbitrary item, for example, the child would receive an M&M's for any correct response that he or she provided (Koegel et al. 1987). This procedural component strengthens the connection between verbalizations and consequences (Williams et al. 1981).

Several natural interventions have been developed around the basic naturalistic treatment procedures for children with autism. Milieu therapy, incidental teaching, and pivotal response treatment all use naturalistic procedures to improve symptoms of autism (Hart and Risley 1968; Kaiser and Trent 1992; Koegel and Koegel 1996).

Milieu language intervention is a common naturalistic treatment that uses the procedures of

incidental teaching, modeling, mand-modeling, and time delay (Albert and Kaiser 1992; Warren and Gazdag 1990). Hart and Risley (1968a) developed the incidental teaching procedure to demonstrate the effectiveness of child-initiated interactions in treatment. Incidental teaching uses occasions when the child is verbally or non-verbally requesting materials or assistance as beneficial therapy opportunities (Thiemann and Warren 2004). Teachers, parents, or other children wait to prompt the child until he or she shows an interest by requesting or reaching for an object in the natural environment (Hart and Risley 1975). Once the child is interested, the opportunity exists for a model procedure, mand-model procedure, or time delay procedure (Albert and Kaiser 1992). A model procedure involves the adult producing a syllable, word, phrase, or sentence with the intention that the child will imitate the production (Albert and Kaiser 1992). Milieu therapy also uses the mand-model technique in which the adult presents a question or instruction for the child to verbalize within a naturally occurring context (Warren and Gazdag 1990; Yoder et al. 1995). Time delay is another procedure often incorporated into milieu therapy (Albert and Kaiser 1992; Yoder et al. 1995). Time delay involves the adult presenting a stimulus and then waiting anywhere between 5 and 30 s for the child's response (Mancil 2009).

Pivotal response treatment (PRT) is another naturalistic intervention model that targets pivotal areas of a child's development, such as motivation, responsivity to multiple cues, self-management, and social initiations (Koegel et al. 1999). Instead of targeting each individual behavior, PRT focuses on pivotal areas that when changed generally produce large collateral improvements in other areas (Koegel and Koegel 2006). Research has shown that improving the child's motivation level during treatment sessions can accelerate language learning, decrease problem behavior such as aggression and tantrums, and increase the affect of the child and the treatment provider (Koegel et al. 1997, 1987, 1998). PRT uses naturalistic and motivational procedures of child choice, task variation, interspersing maintenance tasks, rewarding attempts, and the use of

direct and natural reinforcers, with the overall goal of providing individuals with autism the social and educational skills to succeed in inclusive settings (Koegel and Koegel 2006). As opposed to the traditional analog approach that reinforces only correct responses, PRT broadens the reinforcement contingency so that the child is rewarded if they make a clear and direct attempt at the target behavior (Koegel et al. 1987). For example, if a child says “ba” instead of “ball,” he or she will be given the ball as a natural reward for their verbal attempt to communicate. PRT treatment procedures are used to teach language, decrease disruptive/self-stimulatory behaviors, and increase social, communication, and academic skills (Koegel and Koegel 2006).

In addition, floortime therapy, reciprocal imitation training, and positive behavioral support incorporate components of naturalistic procedures in their intervention techniques (Gillum et al. 2003; Ingersoll et al. 2007). Common treatment procedures used in these naturalistic intervention programs may include in vivo modeling, turn taking, shared control, priming, conversational recast, and self-management (Brown and Odom 1995; Camarata 1993; Gillum et al. 2003; Koegel and Koegel 1996; McGee et al. 1984).

Efficacy Information

Naturalistic interventions are effective in improving generalization across settings, increasing spontaneous speech, improving maintenance following intervention, and incorporating parents and teachers into the treatment program (Ingersoll et al. 2007; Koegel et al. 1987, 1998; Koegel and Koegel 1996; Koegel and Williams 1980; McGee et al. 1984; Miranda-Linne and Melin 1992). Traditional analog procedures succeed in increasing language, social, play, and behavioral skills of children with autism, but the effectiveness is limited in regard to generalization and maintenance of treatment gains (Vismara and Rogers 2010). Subsequent research has led to the formation of naturalistic interventions, which diminish the limitations of traditional approaches

and expand treatment procedures to the natural environment (Koegel et al. 1987).

A key benefit in implementing naturalistic interventions is the high level of generalization that occurs in the absence of special programming (Ingersoll et al. 2007; McGee et al. 1984; Warren and Gazdag 1990). Although analog procedures can create positive outcomes, the treatment gains frequently do not carry over to natural settings (Brown and Odom 1995; Camarata 1993). In naturalistic interventions, treatment takes place in already existing activities, so the child learns how to utilize skills in the environment in which they are needed (McGee et al. 1984). Procedures can be embedded into everyday activities such as the home, the classroom, and the community, and generalization of language gains does not require additional teaching programs (Hart and Risley 1975; Vismara and Rogers 2010). For example, Hart and Risley (1968) showed that traditional teaching procedures were effective in generating adjective-noun combinations in that restricted setting, but the child’s spontaneous vocabularies increased with the use of environmental contingencies. Furthermore, results from a study by Brown and Odom (1995) show that naturalistic peer interventions can easily be incorporated into a school setting and can lead to gains in socialization that can be applied across multiple settings.

Naturalistic interventions are also effective in improving the maintenance of treatment gains following intervention (Koegel et al. 1987). Miranda-Linne and Melin (1992) found that traditional methods can produce positive outcomes during home generalization probes, but the gains dissipated 1 week after intervention was completed. Incidental methods led to slower treatment gains, but the skills acquired were more permanent and continued to increase after teaching had been discontinued (Miranda-Linne and Melin 1992). A similar study found that generalization from trained to spontaneous language skills was maintained during a 4.5-month follow-up probe when teaching was provided in naturally occurring environments (Koegel and Koegel 1996; McGee et al. 1984).

Parents and families have found naturalistic interventions useful in expanding existing resources and reducing parental stress (Albert and Kaiser 1992; Koegel and Koegel 1996). According to Thiemann and Warren (2004), one of the primary challenges in treatment focused on communication is effectively implementing the intervention procedures into everyday practice. Including individuals in the child's natural environment (i.e., parents and teachers) can increase the intervention opportunities and the child's rate of progress (Barnet et al. 1993). In addition to improving and maintaining the child's treatment gains with the parent education component, families who implement naturalistic treatment procedures report higher amounts of leisure time spent together (Koegel and Koegel 1996). When parents are incorporated into naturalistic interventions, they show increased affect, more interest, lower levels of stress, and a positive engagement of communication with their child with autism (Koegel et al. 1996).

Naturalistic interventions are effective in increasing language acquisition, improving generalization and maintenance, and creating positive interactions for parents and family members with a child with autism.

Outcome Measurement

The National Standards Project has found sufficient evidence to show that naturalistic teaching strategies produce favorable outcomes for individuals on the autism spectrum. Through a systematic review of 32 studies, it was found that naturalistic strategies increase communication, interpersonal skills, learning readiness, and play behavior (National Autism Center 2009). With respect to age, naturalistic interventions demonstrate favorable outcomes for children aged zero to nine that have a diagnosis of autism or pervasive developmental disorder-not otherwise specified (PDD-NOS) (National Autism Center 2009).

A main diagnostic criterion of children with autism is deficits in communication and language skills (Mancil 2009). In 2009, the National Autism Center evaluated the literature and found

empirical support that naturalistic intervention procedures improve nonverbal and verbal methods of sharing emotions and information. Outcome measures have shown that children receiving naturalistic treatments can improve their speech intelligibility and functional use of target sounds during conversation (Camarata 1993; Koegel et al. 1998). Additionally, there is evidence that the intervention techniques are effective at increasing the level of responding and initiating communication in children with autism (Koegel et al. 2003).

Outcome measures also show that naturalistic treatment procedures improve interpersonal skills (National Autism Center 2009). Intervention strategies lead to increased play, social interaction, and assertions with peers (McGee et al. 1984; Stahmer and Ingersoll 2009; Vismara and Rogers 2010). Comprehensive social programs that embed natural procedures increase the level of appropriate conversation and peer interactions (Koegel and Frea 1993; Koegel et al. 2009). The National Autism Center also found sufficient evidence for support of treatment procedures as a method to increase learning success in complex skills.

Studies regarding natural intervention were rated high in experimental rigor, quality of the dependent variable, evidence of treatment fidelity, demonstration of participant ascertainment, and generalization of data collected. Thorough examination of naturalistic strategies by the National Autism Center has found empirical evidence to show that the intervention procedures produce positive gains in many core areas of autism and can be successfully implemented for children of many ages on the autism spectrum.

Qualifications of Treatment Providers

One key benefit of naturalistic interventions is the ease with which treatment providers can be taught to embed procedures into existing activities (Vismara and Rogers 2010). Treatment providers can be specialized therapists, members of the regular staff, or family members that are involved in

the child's daily life (Kaiser and Trent 1992; Kalyva and Avramidis 2005; Koegel and Koegel 2006).

Due to the fact that intervention procedures are typically employed in a setting outside the clinic, it is especially important that treatment providers are well trained in intervention strategies. Interventionists, whether they are students, parents, or consultants, must be proficient in the protocols and routinely be given feedback on their performance. In order to successfully implement intervention procedures, treatment providers typically receive training in the specific intervention that includes lectures, observations, role-play practice, and a written exam on policies and procedures (National Research Council 2001). Furthermore, some intervention programs require audiotaped or videotaped interactions that show the provider implementing the intervention with children (Albert and Kaiser 1992).

Treatment providers must meet fidelity of implementation in the intervention procedures in order for them to effectively employ the protocol. Treatment providers are typically trained in the intervention and then scored in vivo or through videotape by an observer. Fidelity of implementation is obtained when intervention procedures are performed correctly for a specified amount of time during the scoring period (Koegel and Koegel 2006). If intervention procedures do not meet the specified criterion level, then the trainer can identify areas that need improvement and may provide training on those specific components. Once the treatment providers are able to employ each intervention component at the specified level of fidelity, then they will be able to successfully perform the intervention with a child.

Feedback should be given on a routine basis for ongoing tracking of child progress and treatment fidelity. Supervision can take place by direct observation of the treatment provider interacting with the child, teacher-collected data, and videotaped samples of intervention sessions (National Research Council 2001).

In summary, naturalistic interventions reduce problems of generalization and maintenance. Further, they reduce family stress when compared to more structure intervention procedures. As such,

research suggests the importance of providing naturalistic interventions for children with autism.

See Also

- [Generalization and Maintenance](#)
- [Incidental Teaching](#)
- [Natural Language Paradigm](#)
- [Pivotal Response Training](#)

References and Reading

- Albert, C., & Kaiser, A. (1992). Training parents as milieu language teachers. *Journal of Early Intervention*, 16(1), 31–52.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: American Psychiatric Association.
- Barnet, D., Carey, K., & Hall, J. (1993). Naturalistic intervention design for young children: Foundations, rationales, and strategies. *Topics in Early Childhood Special Education*, 13(4), 430–444.
- Brown, W., & Odom, S. (1995). Naturalistic peer interventions for promoting preschool children's social interactions. *Preventing School Failure*, 39(4), 38–43.
- Camarata, S. (1993). The application of naturalistic conversation training to speech production in children with speech disabilities. *Journal of Applied Behavior Analysis*, 26, 172–182.
- Carr, E. G. (1983). Behavioral approaches to language and communication. In E. Schopler & G. Mesibov (Eds.), *Current issues in Autism* (Communication problems in Autism, Vol. III). New York: Plenum.
- Fisher, M., & Meyer, L. H. (2002). Development and social competence after two years for students enrolled in inclusive and self-contained educational programs. *Research & Practice for Persons with Severe Disabilities*, 27(3), 165–174.
- Gillum, H., Camarata, S., Nelson, K., & Camarata, M. (2003). A comparison of naturalistic and analog treatment effects in children with expressive language disorder and poor preintervention imitation skills. *Journal of Positive Behavior Interventions*, 5(3), 171–178.
- Harper, C., Symon, J., & Frea, W. (2008). Recess is time-in: using peers to improve social skills of children with autism. *Journal of Autism and Developmental Disorders*, 38(5), 815–826.
- Hart, B., & Risley, T. (1968a). Developing correspondence between the non-verbal and verbal behavior of preschool children. *Journal of Applied Behavior Analysis*, 1(4), 267–281.
- Hart, B., & Risley, T. (1968b). Establishing use of descriptive adjectives in the spontaneous speech of disadvantaged preschool children. *Journal of Applied Behavior Analysis*, 1, 109–120.

- Hart, B., & Risley, T. (1975). Incidental teaching of language in the preschool. *Journal of Applied Behavior Analysis*, 8, 411–420.
- Ingersoll, B., Lewis, E., & Kroman, E. (2007). Teaching the imitation and spontaneous use of descriptive gestures in young children with Autism using a naturalistic behavioral intervention. *Journal of Autism and Developmental Disorders*, 37, 1446–1456.
- Kaiser, A. P., & Trent, J. A. (2007). Communication intervention for young children with disabilities: Naturalistic approaches to promoting development. In S. Odom, R. Horner, M. Snell, & J. Blacher (Eds.), *Handbook of developmental disabilities*. New York: Guilford Press.
- Kalyva, E., & Avramidis, E. (2005). Improving communication between children with Autism and their peers through the “circle of friends”: A small-scale intervention study. *Journal of Applied Research in Intellectual Disabilities*, 18(3), 253–261.
- Koegel, R. L., & Frea, W. D. (1993). Treatment of social behavior in Autism through the modification of pivotal social skills. *Journal of Applied Behavior Analysis*, 26, 369–377.
- Koegel, R. L., & Koegel, L. K. (2006). *Pivotal response treatments for Autism*. Baltimore: Paul H. Brookes.
- Koegel, R., & Williams, J. (1980). Direct versus indirect response-reinforcer relationships in teaching Autistic children. *Journal of Abnormal Child Psychology*, 8(4), 537–547.
- Koegel, R., Glahn, T., & Nieminen, G. (1978). Generalization of parent-training results. *Journal of Applied Behavior Analysis*, 11, 95–109.
- Koegel, R., O'Dell, M., & Koegel, L. (1987). A natural language paradigm for nonverbal Autistic children. *Journal of Autism and Developmental Disorders*, 17(2), 187–200.
- Koegel, R., O'Dell, M., & Dunlap, G. (1988). Producing speech use in nonverbal Autistic children by reinforcing attempts. *Journal of Autism and Developmental Disorders*, 18(4), 525–538.
- Koegel, R., Bimbela, A., & Schreibman, L. (1996). Collateral effects of parent training on family interactions. *Journal of Autism and Developmental Disabilities*, 26(3), 347–359.
- Koegel, L. K., Koegel, R. L., & Smith, A. (1997). Variables related to differences in standardized test outcomes for children with Autism. *Journal of Autism and Developmental Disorders*, 27, 233–243.
- Koegel, R., Camarata, S., Koegel, L., Ben-Tall, A., & Smith, A. (1998). Increasing speech intelligibility in children with Autism. *Journal of Autism and Developmental Disorders*, 28, 3.
- Koegel, L. K., Koegel, R. L., Harrower, J. K., & Carter, C. M. (1999). Pivotal response intervention I: Overview of approach. *The Journal of the Association for Persons With Severe Handicaps*, 24(3), 174–185.
- Koegel, R. L., Koegel, L. K., & Brookman, L. I. (2003). Pivotal response training for Autistic disorder. In A. E. Kazdin & J. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents*. New York: Guilford Publications.
- Koegel, L. K., Robinson, S., & Koegel, R. L. (2009). Empirically supported intervention practices for Autism spectrum disorders in school and community settings. In W. Sailor, G. Dunlap, G. Sugai, & R. Horner (Eds.), *Handbook of positive behavior support* (Issues and practices, pp. 149–176). New York: Springer.
- McGee, G., Krantz, P., & McClannahan, L. (1984). Conversational skills for Autistic adolescents: Teaching assertiveness in naturalistic game settings. *Journal of Autism and Developmental Disorders*, 14(3), 319–330.
- Miranda-Linne, F., & Melin, L. (1992). Acquisition, generalization, and spontaneous use of color adjectives: A comparison of incidental teaching and traditional discrete-trial procedures for children with Autism. *Research in Developmental Disabilities*, 13, 191–210.
- National Autism Center. (2009). *National standards project – addressing the need for evidence-based practice guidelines for Autism spectrum disorders*. From <http://www.nationalautismcenter.org/about/national.php>
- National Research Council. (2001). *Educating children with Autism*. Committee on educational interventions for children with Autism. In C. Lord & J. P. McGee (Eds.), *Division of behavioral and social sciences and education*. Washington, DC: National Academy Press.
- Paul, R. (2008). Interventions to improve communication in Autism. *Child and Adolescent Psychiatric Clinics of North America*, 17, 835–856.
- Reinhartsen, D., Garfinkle, A., & Wolery, M. (2002). Engagement with toys in two-year-old children with Autism: Teacher selection versus child choice. *Research & Practice for Persons with Severe Disabilities*, 27(3), 175–187.
- Stahmer, A., & Ingersoll, B. (2009). Inclusive programming for toddlers with Autism spectrum disorders: Outcomes from the children's Toddler school. *Journal of Positive Behavior Interventions*, 6(2), 67–82.
- Thieman, K., & Warren, S. F. (2004). Programs supporting young children's language development. In R. E. Tremblay, R. G. Barr, & R. Peters (Eds.), *Encyclopedia on early childhood development (online)* (pp. 1–11). Montreal: Centre of Excellence for Early Childhood Development.
- Vismara, L., & Rogers, S. (2010). Behavioral treatments in Autism spectrum disorder: What do we know? *Annual Review of Clinical Psychology*, 6, 447–468.
- Warren, S., & Gazdag, G. (1990). Facilitating early language development with Milieu intervention procedures. *Journal of Early Intervention*, 14(1), 62–85.
- Williams, B., & Williams, L. (2010). *Effective programs for treating Autism spectrum disorder: Applied behavior analysis models*. New York: Routledge.
- Williams, J., Koegel, R., & Egel, A. (1981). Response-reinforcer relationships and improved learning in Autistic children. *Journal of Applied Behavior Analysis*, 14, 53–60.
- Yoder, P., Kaiser, A., Goldstein, H., Alpert, C., Moussetis, L., Kaczmarek, L., et al. (1995). An exploratory comparison of Milieu teaching and responsive interaction in classroom application. *Journal of Early Intervention*, 19(3), 218–242.

Navane

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Synonyms

Thiothixene

Definition

One of the first-generation neuroleptic (major tranquilizer) medications, thiothixene, is of medium potency. Like other agents in this class, its primary mode of action is through the dopamine neurotransmitter system. Compared to the newer, atypical neuroleptics (several of which have been approved for treatment of autism), these earlier agents were more likely to cause various side effects. These have been widely used for decades in the treatment of psychosis (particularly schizophrenia) but also have been used to treat maladaptive behaviors in autism (stereotyped movements, agitation, and self-injurious behaviors). Side effects of this medication can include muscle stiffness, dry mouth, weight gain, and a cluster of features often referred to as extrapyramidal symptoms (restlessness and movement problems that resemble some aspects of Parkinson's disease). Some of the side effects can be treated by anticholinergic agents like diphenhydramine. The most serious long-term side effect is tardive dyskinesia. Neuroleptic malignant syndrome is a rare but serious (and potentially fatal) side effect associated with fever, movement problems, and changes in mental state.

See Also

- [Diphenhydramine](#)
- [Dopamine](#)
- [Thiothixene](#)

References and Reading

- Scahill, L. (2008). How do I decide whether or not to use medication for my child with autism? Should I try behavior therapy first? *Journal of Autism & Developmental Disorders*, 38(6), 1197–1198.
- Scahill, L., & Martin, A. (2005). Psychopharmacology. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, pp. 1102–1122). Hoboken: Wiley.
- Scheffer, R. E. (2006). Psychopharmacology: Clinical implications of brain neurochemistry. *Pediatric Clinics of North America*, 53(4), 767–775.
- White, S., Scahill, L., Klin, A., Koenig, K., & Volkmar, F. (2007). Educational placements and service use patterns of individuals with autism spectrum disorders. *Journal of Autism & Developmental Disorders*, 37(8), 1403–1412.

Need for Caregiver Support for Families of Children with ASD, The

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Definition

The need for in-home support for caregivers of children with ASD.

Historical Background

Prevalence rates of ASD have been steadily increasing for a number of years, and currently it is estimated that 1:59 children in the United States are impacted by ASD (Center for Disease Control 2019). Individuals with this disability have varying social-communication and behavior challenges. These challenges impact a person from infancy into adulthood and not only impact the quality of life of the individuals

themselves, these impairments also affect their caregivers and other family members (Gupta and Singhal 2004). Caregivers of children with disabilities have been widely studied in research, with a more recent intense focus on understanding how parenting a child with ASD specifically impacts the family (i.e., Ekas and Whitman 2011; Freedman et al. 2012).

Parents and guardians of individuals with autism spectrum disorders have significantly high levels of burden and stress in caring for their child (Goedeke et al. 2019). Research suggests that parents of individuals with autism experience both emotional and financial burdens (Krakovich et al. 2016). The financial burdens may vary across the lifespan, but for toddlers and school-age children, these financial hardships may be attributed to the cost of early intervention services, child care, medical expenses, or additional services not provided by the school district (i.e., private therapy services). Increased stress levels can be attributed to the level of severity of behavior and social-communication challenges of the child (Estes et al. 2009) and the financial costs associated with their care (Krakovich et al. 2016).

Historically, parents of individuals with ASD have reported higher levels of stress when compared with parents of children without disabilities or of children with other disabilities (Paynter et al. 2013). The stress of caring for a child with ASD has been found to trigger a number of adverse psychological reactions. The profound changes in families resulting from having a child with autism often lead to tension and stress. There are often anxious and depressive impacts on parents (Ben Soussia et al. 2017). Mothers of children with ASD have been studied more frequently than fathers, with reports indicating more stress, depression, and anxiety in mothers of individuals with ASD as compared to mothers of individuals with other disabilities (Davis and Carter 2008).

Parents have reported that this heightened tension has a direct impact on the entire family (Carbone et al. 2010). Freedman et al. (2012) identifies speculated reports that have indicated that parents of individuals with autism have a higher divorce rate (nearly 80%) than parents of

typically developing children. It is important to note that this statistic has not been substantiated in empirical research. Although an accurate divorce rate has not been identified, it is clear that marital relationships are impacted by raising a child with ASD (Freedman et al. 2012). While there is limited validated research on divorce rates of parents, it is important to acknowledge that parents and caregivers still require additional supports. Krakovich et al. (2016) identify examples of external supports as respite and formal ASD services and examples of internal supports such as education level and income.

Current Knowledge

A survey conducted by the State Department of Education in 2015 in Connecticut aimed to better understand the perspectives of parents of children with disabilities educated in CT schools. When looking at questions related to parent involvement and supports, 32% of parents did not know whether parent training opportunities existed within their district; more specifically, only 41% of parents with children with autism reported that they knew whether parent training opportunities existed within their district (Department of Special Education 2015). Additionally, only 39% of families of children with autism reported they were involved in a support network for families of children with disabilities, and only 44% of families of children with ASD reported they knew that parent support opportunities even existed (Department of Special Education). These findings indicate that services for parents do exist, but accessing the services is limited, which may be attributed to a variety of factors (i.e., location of supports, awareness of needs, finances, etc.). In another study (Carbone et al. 2010), parents report that although family supports are challenging to find, they were found to have positive results with these services (i.e., mental health services, parent and sibling support groups, and respite care). Additionally, parents reported that they had more positive experiences with informal parent-parent interactions (Carbone et al.). These findings indicate that there is a need to help parents identify quality

resources and supports that are available to them. Families rely on these informal and formal supports in order to help cope with caring for their child (Searing 2014).

Using education and training to empower families of children with ASD is best practice (Dawson-Squibb et al. 2019). The definition of parent training in ASD, however, is ambiguous due to the complexity of ASD and the number of areas that are targeted for intervention. After a review of the literature, Bearrs et al. (2015) suggest that parent training has a broad application in areas such as psychoeducation, care coordination, parent-mediated interventions for maladaptive behaviors, and core symptoms.

Future Directions

There is existing research exploring parent perspective of what their children need in terms of services and programming and findings pertaining to the increased levels of stress of families of children with ASD (Estes et al. 2009). However, there is still limited knowledge on the specific needs of the parents of children with autism. More research needs to be conducted to determine exactly what parents feel they need, what interventions are beneficial for parents, and how parents can gain access to available resources.

Historical data and current knowledge indicate that there is a need for caregiver support for families of children with ASD. It is imperative that other professionals help parents find the right support (Vernhet et al. 2019). For instance, local education agencies should understand the role that they have in supporting families. School districts can assist in identifying the specific needs of parents of students in their schools. Then districts can offer a variety of services and trainings for parents. Parent training can be offered as a related service in a child's individualized education plan (IDEA 2004). Offering training to meet the specific needs of parents can help with consistency of implementation of evidence-based practices used in school settings. Training

should also be provided through a variety of modalities (face to face, online webinars, presented in multiple languages, etc.). By having a variety of options, these trainings may reach a more diverse audience of caregivers (i.e., parents, grandparents, community members, babysitters, etc.). Additionally, if parents understand how to use these evidence-based practices, they can share these strategies with other caregivers. Training has been found to positively increase knowledge and skills of parents. Parent education of ASD and strategies can have a positive impact on stress levels (Tonge et al. 2006) and also improve family relationships.

Additionally, districts may need to market these services to parents in various ways in order to make sure families know what supports are available to them. These districts could collaborate with parent advocacy groups. Some research indicates that parents supporting other parents can help reduce stress levels (Dykens et al. 2014). Parent advocacy groups can help to offer assistance, programs, and support groups for other parents. Additionally, it will be vital for parent advocacy groups to continue to reach out in a variety of different ways to help support parents. These parent advocacy groups can reach out to parents within their network to inform them of trainings and programs being offered. Social media and webinars are also ways for parents to learn more about specific interventions, support services, and trainings.

Parents of children with ASD report that they do not have enough time for parental responsibilities or enough time for their romantic relationships (Hirsch and Paquin 2019). Programs should be more available to help caregivers find more personal time (Ekas and Whitman 2011). Respite care includes temporary caregiving services that can occur in the home setting. This may be an option for some families and can enable a parent to take a needed break from the daily responsibilities of raising a child with autism. However, it is important that people providing these caregiving services understand the challenges associated with autism (Goedeke et al. 2019).

Additionally, challenges associated with the degree of ASD symptoms the child exhibits are related to obstacles to engaging in family activities (i.e., eating out, vacations, etc.) (Hirsch and Paquin 2019). There is a need to help support families with parent training on strategies (e.g., Bearss et al. 2015). Additionally, these resources that are offered should be researched to determine if in fact they help reduce levels of stress within the home (Krakovich et al. 2016). Although predictors of stress have been identified, and recommendations have been suggested to help support parents, there is limited knowledge on which specific interventions have been reported to reduce parent stress levels (Krakovich et al.; Ekas and Whitman 2011) and improve the quality of lives of families with ASD. Further research should be conducted in this area in order to validate specific programs, training, or support that positively impact families of children with ASD (Goedeke et al. 2019).

See Also

- Family-Centered Care, Second Edition
- Medical Home and ASD
- Parent Training

References and Reading

- Bearss, K., Burrell, T. L., Stewart, L., & Scahill, L. (2015). Parent training in autism spectrum disorder: What's in a name? *Clinical Child and Family Psychology Review*, 18(2), 170–182. <https://doi.org/10.1007/s10567-015-0179-5>.
- Ben Soussia, R., Bouallagui, A., Khouadja, S., Marrag, I., & Nasr, M. (2017). Psychoaffective repercussions of autism on parents. *European Psychiatry*, 41, S123. <https://doi.org/10.1016/j.eurpsy.2017.01.1922>.
- Carbone, P. S., Behl, D. D., Azor, V., & Murphy, N. A. (2010). The medical home for children with autism spectrum disorders: Parent and pediatrician perspectives. *Journal of Autism & Developmental Disorders*, 40(3), 317–324. <https://doi.org/10.1007/s10803-009-0874-5>.
- Center for Disease Control and Prevention. (2019). Autism spectrum disorders. Retrieved from <https://www.cdc.gov/ncbddd/autism/data.html>
- Davis, N. O., & Carter, A. S. (2008). Parenting stress in mothers and fathers of toddlers with autism spectrum disorders: Associations with child characteristics. *Journal of Autism and Developmental Disorders*, 38, 1278–1291.
- Dawson-Squibb, J.-J., Davids, E. L., Harrison, A. J., Molony, M. A., & de Vries, P. J. (2019). Parent education and training for autism spectrum disorders: Scoping the evidence. *Autism*. <https://doi.org/10.1177/1362361319841739>
- Department of Special Education. (2015). Connecticut special education parent survey: Summary report. https://portal.ct.gov/-/media/SDE/Special-Education/Parent_Survey_Summary_Report_2015.pdf
- Dykens, E. M., Fisher, M. H., Taylor, J. L., Lambert, W., & Miodrag, N. (2014). Reducing distress in mothers of children with autism and other disabilities: A randomized trial. *Pediatrics*, 134(2), e454–e463. <https://doi.org/10.1542/peds.2013-3164>.
- Ekas, N. V., & Whitman, T. L. (2011). Adaptation to daily stress among mothers of children with an autism spectrum disorder: The role of daily positive affect. *Journal of Autism & Developmental Disorders*, 41(9), 1202–1213. <https://doi.org/10.1007/s10803-010-1142-4>.
- Estes, A., Munson, J., Dawson, G., Koehler, E., Zhou, X. H., & Abbott, R. (2009). Parenting stress and psychological functioning among mothers of preschool children with autism and developmental delay. *Autism*, 13(4), 375–387.
- Freedman, B., Kalb, L., Zablotsky, B., & Stuart, E. (2012). Relationship status among parents of children with autism spectrum disorders: A population-based study. *Journal of Autism & Developmental Disorders*, 42(4), 539–548. <https://doi.org/10.1007/s10803-011-1269-y>.
- Goedeke, S., Shepherd, D., Landon, J., & Taylor, S. (2019). How perceived support relates to child autism symptoms and care-related stress in parents caring for a child with autism. *Research in Autism Spectrum Disorders*, 60, 36–47. <https://doi.org/10.1016/j.rasd.2019.01.005>.
- Gupta, A., & Singhal, N. (2004). Positive perceptions of parents with disabilities. *Asia Pacific Disability Rehabilitation Journal*, 15(1), 22–25.
- Hirsch, K. H., & Paquin, J. D. (2019). The stress of the situation has changed us both: A grounded theory analysis of the romantic relationship of parents raising children with autism. *Journal of Child and Family Studies*, 28(10), 2673–2689. <https://doi.org/10.1007/s10826-019-01448-y>.
- Individuals With Disabilities Education Act, 20 U.S.C. § 1400 (2004).
- Krakovich, T., McGrew, J., Yu, Y., & Ruble, L. (2016). Stress in parents of children with autism spectrum disorder: An exploration of demands and resources. *Journal of Autism & Developmental Disorders*, 46(6), 2042–2053. <https://doi.org/10.1007/s10803-016-2728-2>.

- Paynter, J., Riley, E., Beamish, W., Davies, M., & Milford, T. (2013). The double ABCX model of family adaptation in families of a child with an autism spectrum disorder attending an Australian early intervention service. *Research in Autism Spectrum Disorders*, 7(10), 1183–1195. <https://doi.org/10.1016/j.rasd.2013.07.006>.
- Searing, B. (2014). Support needs of families living with children with autism spectrum disorder in New Zealand (Unpublished Master's Degree), University of Otago, Dunedin, New Zealand Statistics New Zealand. (2006). *New Zealand: An urban/rural update*. New Zealand: Wellington.
- Tonge, B., Brereton, A., Kiomall, M., MacKinnon, A., King, N., & Rinehart, N. (2006). Effects on parental mental health of an education and skills training program for parents of young children with autism: A randomized controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45(5), 561–569.
- Vernhet, C., Dellapienza, F., Blanc, N., Cousson-Gelie, F., Miot, S., Roeyers, H., & Baghdadli, A. (2019). Coping strategies of parents of children with autism spectrum disorder: A systematic review. *European Child & Adolescent Psychiatry*, 28, 747–758. Retrieved from <https://doi.org/10.1007/s00787-018-1183-3>.

response to a target stimulus in the presence of distracters. For example, participants may see two letters on a computer screen and be instructed to name the upper case letter (e.g., from May et al. 1995), e.g., “H” and “p.” In this trial, the participant would respond with “H.” Importantly, on a later trial, the letters might be “P” and “r.” The inhibition applied to “p” as a distracter on the earlier trial carries over into the current trial, when the “P” is now a target. This “negative priming” results in slower response times, i.e., people are slower to respond to targets that were previously distracters.

The effect need not be driven by exactly the same stimulus; it can be caused by targets occupying the same location as a prior distracter, targets sharing attributes or category membership with earlier distracters, or targets that require responses that were inhibited by earlier distracters. The negative priming effect is robust across a wide range of tasks.

Negative Priming Effect

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Definition

Negative priming is an experimental phenomenon hypothesized to reflect inhibitory aspects of executive function. Executive function encompasses complex processes such as planning, organization, reflection, and metacognition and more basic processes such as engaging and disengaging attention and initiating or inhibiting responses. Inhibition is a fundamental component of executive function and is thought to be captured by the negative priming effect on simple cognitive tasks.

The negative priming effect occurs in experiments where participants are required to make a

See Also

- [Attention](#)
- [Executive Function \(EF\)](#)

References and Reading

- Hill, E. (2004). Executive dysfunction in autism. *Trends in Cognitive Sciences*, 8(1), 26–32.
- May, C., Kane, M., & Hasher, L. (1995). Determinants of negative priming. *Psychological Bulletin*, 118(1), 35–54.
- Ozonoff, S., & Strayer, D. (1997). Inhibitory function in nonretarded children with autism. *Journal of Autism and Developmental Disorders*, 27(1), 59–78.

Negative Punishment

- [Omission Training](#)
- [Type II Punishment, Second Edition](#)

Negative Reinforcement

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Definition

Negative reinforcement is a key principle in the behavioral theory of learning. Negative reinforcement is defined as the removal, reduction, or avoidance of a stimulus contingent upon a behavior, which results in an increased frequency of that behavior in the future. For example, a child who screams until an adult gives them a preferred item may negatively reinforce the adult's behavior. The termination of the screaming may serve as negative reinforcement for the adult's behavior of giving the child a preferred item when he/she is screaming. Negative reinforcement often involves the removal of an aversive stimulus, but it is not a requirement that the stimulus be undesirable. Negative reinforcement is similar to positive reinforcement in that both lead to increased responding in the future, but are dissimilar in that negative reinforcement involves the termination of a stimulus, and positive reinforcement requires the presentation of a stimulus. Negative reinforcement is typically confused with punishment; however, punishment results in a decrease in responding, not an increase.

See Also

► [Positive Reinforcement](#)

References and Reading

- Devlin, S., Healy, O., Leader, G., & Reed, P. (2008). The analysis and treatment of problem behavior evoked by auditory stimulation. *Research in Autism Spectrum Disorders*, 2(4), 671–680.
- Iwata, B. A. (1987). Negative reinforcement in applied behavior analysis: An emerging technology. *Journal of Applied Behavior Analysis*, 20, 361–378.
- Iwata, B. A. (2006). On the distinction between positive and negative reinforcement. *Behavior Analyst*, 29, 121–123.
- Michael, J. (1975). Positive and negative reinforcement, a distinction that is no longer necessary; or better ways to talk about bad things. *Behavior*, 3, 33–45.
- Skinner, B. F. (1953). *Science and human behavior*. New York: Macmillan.

Neostriatum

► [Caudate Nucleus](#)

NEPS

► [NEPSY-II](#)

NEPS-U

► [NEPSY-II](#)

NEPSY-II

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Synonyms

[NEPS](#); [NEPS-U](#)

Definition

The title of this test, “NEPSY,” is not a true acronym but, rather, a combination of letters, across countries and languages that reflect the term “neuropsychology.”

Description

The NEPSY-II is a comprehensive neuropsychological battery for children aged 3–12. The test provides measures of sensor-motor, language, visuospatial processing, memory and learning, attention/executive functions, and social cognition. It is one of the few well-normed and standardized neuropsychological batteries for children and adolescents.

Similar to its predecessor, the NEPSY-II continues to be based on the theoretical foundation of Luria (1966) wherein designated brain functions correspond with selected assessment tasks. The neuropsychological domains covered by the NEPSY-II are clearly multidimensional and representative of the broad range of neurocognitive functions espoused by most neuropsychological models. Consistent with the NEPSY, the NEPSY-II includes the domains of Attention and Executive Functioning, Language, Memory and Learning, Sensorimotor, and Visuospatial Processing. Further, the NEPSY-II contains a new domain, Social Perception. This latter addition is innovative with respect to its inclusion in such assessment batteries, and provides an additional avenue for assessment from the neuroscience literature that has not been routinely available to most clinicians. These tasks also will be relevant for professionals conducting neuropsychological assessments for children and adolescents with autism spectrum disorder (ASD). It is important to note that the neuropsychological domains of the NEPSY-II are not empirically derived, or even statistically independent. Although this is clearly noted in the manual, the user is left to determine how specific subtests relate within and across domains and, importantly, how these relationships may change over the 3.0–16.11 age range proposed by the NEPSY-II. Specific subtests and their age range are provided below.

Domain/subtest	Age range
Sensorimotor	
Fingertip tapping	5–16
Imitating hand positions	3–12
Manual motor sequences	3–12
Visuomotor precision	3–12
Language	
Body part naming and identification	3–4
Comprehension of instructions	3–16
Oromotor sequences	3–12
Phonological processing	3–16
Repetition of nonsense words	5–12
Speeded naming	3–16
Word generation	3–16
Visuospatial processing	
Arrows	5–16
Block construction	3–16
Design copying	3–16
Geometric puzzles	3–16
Picture puzzles	7–16
Route finding	5–12
Memory and learning	
List memory	7–12
List memory delayed	7–12
Memory for design	3–16
Memory for designs delayed	5–16
Memory for faces	5–16
Memory for faces delayed	5–16
Memory for names	5–16
Memory for names delayed	5–16
Narrative memory	3–16
Sentence repetition	3–6
Word list interference	7–16
Auditory attention and executive functioning	
Animal sorting	7–16
Auditory attention	5–16
Auditory attention response set	7–16
Clocks	7–16
Design fluency	5–12
Inhibition	5–16
Statue	3–6
Social perception	
Affect recognition	3–16
Theory of mind	3–16

The NEPSY and the NEPSY-II represent significant efforts to extend neuropsychological testing into the preschool years. Outside of selected intellectual batteries and single test approaches, there are few batteries that have attempted to

provide neuropsychological measurement for such a young population. This version of the test should facilitate increased precision with respect to description of neurocognitive functions for a wide variety of preschool children with both neurological and neurodevelopmental disorders and, subsequently, prescription of early intervention strategies.

Administration and Scoring

For the NEPSY-II, there is a separate *Administration Manual* that provides detailed, manualized procedures for administration and scoring of each subtest. The domain, description, materials, starting points, discontinue rule, timing, recording, general guidelines, and specific administration and scoring details are provided for each subtest. These details are particularly important for several subtests, such as Clocks, Design Copying, Visuomotor Precision, and Word Generation, where increased attention to detail in administration and scoring is required. The materials provided in the NEPSY-II kit are easy to use, colorful and attractive, and appear to be relatively durable. Separate test forms are provided for the preschool battery (ages 3–4) and the school-age battery (ages 5–16). There also are additional teaching items, which should facilitate assessment to children with ASD.

One modification asserted for the NEPSY-II was to provide increased flexibility in the order of subtest administration. Although this was present in the 1998 version of the NEPSY, it was not clear how different orders of subtest administration would affect scores in a larger test profile. For the NEPSY-II, the test developers examined the flexibility systematically via four different administration orders during the standardization process. By not showing any order effects, they were successful in being able to document that the subtests in the NEPSY-II could be administered in a variety of orders without affecting results. This should facilitate subtest administration, particularly in difficult-to-test samples of children (e.g., child psychiatric disorders, ADHD-Hyperactive Type), and this benefit will extend into both the clinical and research realms.

Scoring procedures for the NEPSY-II subtests are clearly presented in the *Administration Manual*. While most of these can be calculated manually, the efficiency of their use likely will increase with use of the NEPSY-II computer scoring program. To obtain the various scores on the NEPSY-II, however, the *Clinical and Interpretive Manual* also will be needed as this is where the normative tables are located. If the computer scoring program is utilized, it will be important for the examiner to consult the *Clinical and Interpretive Manual* to determine how to use these scores, especially when employing the various contrast scores and behavioral observations that can be calculated. The deletion of the NEPSY domain scores should facilitate the scoring process, but it will require additional knowledge with respect to how various subtests interrelate for a selected clinical population of interest. Unlike many intelligence tests, an overall score is not calculated.

Interpretation

Just like other neuropsychological assessment approaches, the interpretation of the NEPSY-II requires a certain amount of examiner knowledge with respect to brain functioning and, at a minimum, a knowledge base in the underlying neurocognitive functions of selected conditions. So, while this battery can be administered by many different psychological examiners, its interpretation does require additional knowledge that will facilitate its use and clinical utility across settings and patient populations. This will be especially pertinent to the nonneuropsychologist who may be working with the complexities inherent in a population of children and adolescents with ASD.

Historical Background

The American version of the NEPSY was published in 1998, with approximately 10 years of development going into its evolution. This version of the NEPSY included 36 subtests arranged across five major neuropsychological domains based on Lurian Theory: Attention/Executive Functions, Sensorimotor, Language,

Visuospatial, and Memory/Learning. The test had normative data that extended from ages 3.0 through age 12.11, with selected tasks being devoted to preschoolers (3.0–5.11) and school-age children (ages 6.0–12.11), with an array of different scores being available to assist with interpretation (e.g., standard scores, percentile ranks, age equivalents). This version of the NEPSY, along with its Scandinavian counterparts, provided one of the first well-normed neuropsychological batteries for children.

The NEPSY was developed out of a specific professional need; that is, the lack of adequate, developmentally appropriate neuropsychological assessment tools for children. Dr. Marit Korkman, a Finnish pediatric neuropsychologist, assembled a variety of tasks into a brief neuropsychological assessment for children 5 through 6 years of age – the NEPS. This seminal version of the NEPSY was subsequently revised by expanding the scoring from a pass/fail criterion assessment measure to allowing for standardized age-based scores for children from ages 3.6 to 9.5. This set of tasks was called the NEPS-U in Finland and the NEPSY in English. Similar versions of the NEPSY were published in Sweden and Denmark, with the current version, the NEPSY-II, appearing in 2010.

Psychometric Data

Standardization

The normative sample was extracted from the most recently available census data, with excellent concurrence with the available census figures with respect to race/ethnicity, parent education, and geographic region of the country. The minority representation was noteworthy across nearly all of the stratification cells. The NEPSY-II also was stratified by chronological age and evenly divided by gender. More specifically, the NEPSY-II was standardized on 1200 children across the ages of 3 through 16 years. Of the 29 possible subtests included in the standardization, 17 were administered to children ages 3–4 years; children 5–6 years of age were administered 22 subtests and 2 delay tasks; children ages 6–12 years were given 23 subtests

and 2 delay tasks; and the adolescent children ages 13–16 years received 24 subtests and 3 delay tasks.

Despite these efforts, a number of NEPSY subtests were not included in the current standardization, but were still included in the NEPSY-II. These subtests were not included in an effort to shorten the time required for a child to complete the standardization battery, but also because the development team determined that the targeted tasks required no modifications and likely would show little change in normative values. Consequently, these subtests (i.e., Design Fluency, Oromotor Sequences, Repetition of Nonsense Words, Manual Motor Sequences, Route Finding, and Imitative Hand Positions) were not included in the standardization version and, for the NEPSY-II, retained the normative scores from the 1998 version of the NEPSY.

Reliability

The area of reliability is critical in test construction as not only does it determine the test's capabilities of replicating results, but it also sets the upper limit on validity. For the NEPSY-II, most subtests have adequate-to-high internal consistency estimates, and the standard error of measure for all of the subtests ranged from 0.85 to 2.18 across all of the age ranges. Temporal stability of the NEPSY-II subtests across six age groups (i.e., ages 3–4; 5–6; 7–8; 9–10; 11–12; and 13–16) revealed little change in scores over an average of a 3-week period (range 12–51 days), suggesting that there was little practice effect across a relatively short time frame. This is critical in a test where alternative form reliability is not available, and provides an evidence-based foundation to use many of the subtests in various types of intervention studies. Given the potential application of the NEPSY-II to children with ASD, it is important to note that the reliability estimates of the subtests were relatively stable for both typical and clinical samples.

Although not directly related to reliability, the issue of tests floors and ceilings can have a direct effect on reliability by potentially restricting the range of scores available. This is especially important for both lower and higher functioning

individuals on the autism spectrum. For the NEPSY-II the test developers focused significant resources on improving the test floors. This is absolutely critical for a test that is designed to uncover neurocognitive weaknesses across different presenting problems and disorders. In this regard, nearly all of the NEPSY-II subtests across the age bands have at least a two standard deviations limit; that is, a child or adolescent can continue to show test variability even when the items begin to become too challenging for them. An adequate floor is essential for differentiating the nature and severity of weaknesses. There is a difference across the age range, however, with all or nearly all of the subtests having an adequate floor from ages 9.0 to 16.9, but 5 to 13 of these subtests not having an adequate floor for ages 5–13 years. In general, the number of subtests having at least a two standard deviation floor increases with age. Conversely, the test developers were not as focused on having at least a two standard deviations ceiling on the subtests. Although this is consistent with the notion of assessment via the NEPSY-II, this is unfortunate as it may place a limit on determining the presence of neurocognitive strengths; consequently, the concept of utilizing neurocognitive strengths to facilitate intervention may be limited. This notion is apparent in the NEPSY-II normative data as from ages 3.0 to 5.9, few subtests meet this criterion (0–5 subtests); from ages 6.0 to 9.9, only 3–8 subtests meet this criterion; and from ages 10.0 to 16.9, only 11–17 subtests meet this criterion.

Validity

Content validity procedures produced a battery of tasks that adequately sample the targeted constructs of interest. For construct validity, the NEPSY-II provides an interesting challenge, in that while there appears to be an overarching set of neuropsychological domains (e.g., Language, Visuospatial Processing, and Social Perception), the test is not designed to provide scores for these domains. As such, and in accordance with the guidance in the Clinical and Interpretive Manual, the administration and interpretation of the NEPSY-II should be guided by the subtests. Consistent with this philosophy, the NEPSY-II

continues to focus on the subtests and their interrelationships. For the NEPSY-II, a multitrait-multimethod correlational model demonstrated support for construct validity across both typical and clinical samples. Although previous efforts to examine the factor structure of the 1998 version of the NEPSY (Mosconi et al. 2008; Stinnett et al. 2002) provided mixed support for the NEPSY, it is unfortunate that these additional analyses were not explored. In support of the test developers' contentions, however, it is likely that different factor structures would be present across different clinical groups, much as is seen in the pattern of intercorrelations of subtests between clinical samples and the normative sample, and this question will require ongoing examination.

Further, with an ongoing emphasis on test interpretation at the subtest level, it would have been beneficial for each task to have accompanying subtest specificity estimates (i.e., an index for what proportion of a subtest's variance is unique to the subtest and reliable). This would have facilitated interpretation and utilization of the subtests in the way they were intended, but with empirical evidence as to their ability to measure a specific function. Criterion-related validity was determined primarily by using concurrent validity studies (i.e., the relationship of the NEPSY-II with other tests measuring similar constructs) wherein good correlations were obtained (e.g., NEPSY-II Memory and Learning subtests correlated most highly with selected subtests from the Children's Memory Scale).

Taken together, these findings support a convergent validity line of evidence for the NEPSY-II; however, it is important to note that these patterns of correlation may change depending on the age of the child, the presenting clinical condition, and the method of assessment (e.g., parent rater, clinician ratings). Additionally, few empirical data are available with respect to the utility of the preschool battery for the NEPSY – either from the publisher or independent investigators, and the same seems to be true with respect to the release of the NEPSY-II. The field will need to determine the ultimate clinical utility of the preschool version of this test, particularly for very young children suspected of being on the autism

spectrum. This issue will require additional examination as the NEPSY-II begins to be employed in a variety of clinical settings, particularly with the wide spectrum of cognitive and behavioral manifestations seen in ASD and the movement toward earlier diagnosis.

Clinical Uses

Clinical Application to ASD

Similar to its neurocognitive predecessors, including the NEPSY, the NEPSY-II utilized available data to showcase its utility with both neurological and neurodevelopmental disorders. The utility with neurological disorders continues the long-standing standard of use of such tests and procedures with frank neurological conditions, but the inclusion of neurodevelopmental disorders – conditions that presume neurological involvement (e.g., ASD) – clearly expands the overall application of this test to a wide variety of potential clients. To facilitate this process, the manual proposes eight different referral batteries from which to facilitate subtest selection.

One of those suggested batteries pertains to children with autism, or suspected of being on the autism spectrum. Suggested tasks span all six of the NEPSY-II neurocognitive domains and include Attention/Executive Functioning (Statue, Design Fluency, Auditory Attention and Response Set, Inhibition, and Animal Sorting); Language (Comprehension of Instructions, Speeded Naming, Word Generation, and Phonological Processing); Memory and Learning (Memory for Designs and Delayed Recall, Narrative Memory, Memory for Faces and Delayed Recall, and Word List Interference); Sensorimotor (Imitating Hand Positions, Visuomotor Precision, Manual Motor Sequences, and Fingertip Tapping); Social Perception (Affect Recognition and Theory of Mind); and Visuospatial Processing (Block Construction, Geometric Puzzles, Design Copying, Geometric Puzzles, Arrows, and Picture Puzzles).

Special Group Studies

The special group studies conducted with the NEPSY-II are noteworthy in that the test developers employed 10 different clinical conditions.

Comparison groups were derived from the normative sample and matched on chronological age, gender, race, and parent education levels. These studies have promised to determine the differential sensitivity of the NEPSY-II to the neuropsychological profiles that can be manifested by specific disorders. Findings from these special group studies generally support the clinical utility of the NEPSY-II in the assessment of children referred for different conditions and disorders. More specifically, the special groups typically differed from the typical groups on variables where they would be expected to deviate, as well as in a wide range of other variables. For example, children with intellectual disabilities and ASD performed more poorly than the typical group on nearly all of the NEPSY-II subtests. The separate examination of the newly added Theory of Mind Subtest for the ASD versus Controls, and for the Asperger's Disorder versus Controls, also provided support for the use of this subtest with these types of clinical referrals. Although useful, these group comparisons should be carefully considered, given their limitations (e.g., small sample sizes, inclusion/exclusion criteria, comorbidity, etc.) until more independent, contemporary data on the use of the NEPSY-II emerge over time.

Empirical Findings in ASD

With the original NEPSY, there were a number of studies that were conducted with samples of children and adolescents with ASD to demonstrate its ability to differentiate between various neurocognitive functions as well as between different diagnostic groups (Hooper et al., 2006; Joseph et al. 2005; Kjelgaard & Tager-Flusberg, 2001; Planche & Lemonnier, 2012). Since the emergence of the NEPSY-II in 2010, there have been a number of studies that have utilized this revised set of procedures for children with ASD. Several studies have demonstrated the utility of the NEPSY-II in differentiating the cognitive abilities of children with high functioning ASD. Narzisi et al. (2012) compared the neurocognitive performance of 22 children with high functioning to 44 healthy controls matched by age, race, gender, and maternal education. Results indicated the presence of significant deficits in Attention and Executive Functions, Language, Learning and

Memory, and Sensorimotor Processing, with only Visuospatial Processing being similar to healthy controls. Further, Narzisi et al. found the expected Theory of Mind deficits in the ASD sample, but this was noted only in the verbally-based tasks. Barron-Linnankoski et al. (2015) conducted a similar study for children, ages 6 to 11 years, and reported the high functioning ASD group to show relative strengths in their verbal reasoning and the expected weaknesses in tasks reflecting cognitive flexibility, verbal fluency, and narrative memory.

In addition to these emergent studies showing the utility of the NEPSY-II with children with ASD, another important trend that is surfacing is the use of the NEPSY-II subtests in interventional science as targeted outcome measures. Specifically, Young and Posselt (2012) used the Affect Recognition Subtest as one of the outcome measures of their examination of The Transporters DVD as a learning tool for children with ASD, aged 4–8 years of age. In addition to their positive findings overall, the NEPSY-II Affect Recognition Test performed well in this intervention, showing positive changes from pre- to posttest over the course of a 3-week trial. Using the same intervention, Williams et al. (2012) used the Affect Recognition and Theory of Mind subtests as outcome measures for their trial. Both NEPSY-II tasks performed well in this 4-week intervention, which also included a 1-month post-intervention follow-up.

Conclusion

The NEPSY-II is one of the few well-standardized and normed neuropsychological batteries for children. The NEPSY-II has benefited from a long developmental history, with the most recent revision capitalizing on the most contemporary findings in child neuropsychology. Changes to the NEPSY-II in terms of its content and administration should provide a fruitful avenue for neuropsychological assessment of children with an existing ASD diagnosis and those suspected as being on the spectrum. The NEPSY-II maintains good psychometric properties, and its use with children and adolescents with ASD has begun to

surface. These findings have demonstrated its clinical utility in detailing relative strengths and weaknesses in neurocognitive functions in individuals with ASD as well as in differentiating between different clinical groups. Importantly, subtests of the NEPSY-II have begun to be used in interventions, and the selected tasks have performed well with respect to the obtainability of reliable data and being sensitive to changes secondary to the targeted intervention. It is important to note that while the NEPSY-II will not provide information on the specific diagnostic symptoms of ASD, it will compliment ongoing diagnostic efforts by characterizing the neurocognitive manifestations of ASD in an individual child, and the emergent empirical literature has begun to show this to be the case. This neurocognitive profile should prove useful in educational planning and cognitive remediation efforts, assisting in how to structure other interventions (e.g., specific therapies, social interactions, etc.), and using the NEPSY-II and its subtests as targeted outcome measures for interventional science.

See Also

- [Executive Function \(EF\)](#)
- [Wisconsin Card Sorting Test \(WCST\)](#)

References and Reading

- Barron-Linnankoski, S., Reinval, O., Lahervuori, A., Voutilainen, A., Lahti-Nuuttila, P., & Korkman, M. (2015). Neurocognitive performance of children with higher functioning autism spectrum disorders on the NEPSY-II. *Child Neuropsychology*, 21, 55–77.
- Hooper, S. R., Poon, K. K., Fine, C., & Marcus, L. (2006). Neuropsychological characteristics of school-age children with high-functioning autism: Performance on the NEPSY. *Child Neuropsychology*, 12, 299–305.
- Joseph, R. M., McGrath, L. M., & Tager-Flusberg, H. (2005). Executive dysfunction and its relation to language ability in verbal school-age children with autism. *Developmental Neuropsychology*, 27, 361–378.
- Kemp, S. L., & Korkman, M. (2010). *Essentials of NEPSY-II assessment*. Hoboken: Wiley.
- Kjelgaard, M. M., & Tager-Flusberg, H. (2001). An investigation of language impairment in autism: Implications

- for genetic subgroups. *Language and Cognitive Processes*, 16, 287–308.
- Korkman, M., Kirk, U., & Kemp, S. (1998). *NEPSY: A developmental neuropsychological assessment*. San Antonio: The Psychological Corporation.
- Korkman, M., Kirk, U., & Kemp, S. (2007). *NEPSY-II*. San Antonio: The Psychological Corporation.
- Luria, A. R. (1966). *The working brain*. New York: Penguin Press.
- Mosconi, M., Nelson, L., & Hooper, S. R. (2008). Confirmatory factor analysis of the NEPSY for younger and older school-age children. *Psychological Reports*, 102, 861–866.
- Narzisi, A., Muratori, F., Calderoni, S., Fabbro, F., & Urgesi, C. (2013). Neuropsychological profile in high functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43, 1895–1909.
- Planche, P., & Lemonnier, E. (2012). Children with high-functioning autism and Asperger's syndrome: Can we differentiate their cognitive profiles? *Research in Autism Spectrum Disorders*, 6, 939–948.
- Stinnett, T. A., Oehler-Stinnett, J. O., Fuqua, D. R., & Palmer, L. S. (2002). Examination of the underlying structure of the NEPSY: A developmental neuropsychological assessment. *Journal of Psychoeducational Assessment*, 20, 66–82.
- Williams, B. T., Gray, K. M., & Tonge, B. J. (2012). Teaching emotion recognition skills to young children with autism: a randomized controlled trial of an emotion training programme. *The Journal of Child Psychology and Psychiatry*, 53, 1268–1276.
- Young, R. L., & Posselt, M. (2012). Using The Transporters DVD as a learning Tool for children with autism spectrum disorders (ASD). *Journal of Autism and Developmental Disorders*, 42, 984–991.

Netherlands and Autism, The

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Historical Background – Legal Aspects – Current Practice

Van Krefeld introduced the concept of autism in the Netherlands in the 1950s from Leiden who, interestingly, referred both to the work of Kanner as well as to that of Asperger! And

though the work of the Creak party and Lotter and Rutter was known in this country, most of the research and clinical practice focused on what was then referred to as “developmental psychosis” (Utrecht: Profs Kamp, Van Engeland). At the end of the 1970s, the primacy of psychoanalysis in child and adolescent psychiatry was replaced by a far more scientific and empirical and pragmatic approach. The introduction of DSM III had a great impulse on the scientific approach spread over the country: Rotterdam Epidemiology (Frank Verhulst) Utrecht Developmental Disorders Autism and ADHD and Multiplex Developmental Disorders (Herman van Engeland) and PDD-NOS Groningen (Ruud Minderaa). The greatly inspiring and dynamic Donald Cohen closely linked the two latter centers with Yale Child Study Center, thus encouraging a fruitful transatlantic cooperation.

At the same time, parents of children with autism joined forces and in the late 1970s started the Netherlands Autism Association (NVA). At the start it was only a small group of parents of severely impaired children. They did not rest before the 1984 Autism Bill was passed in parliament acknowledging that autism is a serious developmental disorder and a great burden on families. The bill states that individuals with autism and their families should be entitled access to relevant services and remuneration of necessary care and educational services.

The Bill on Autism assumed that the prevalence of autism would hover around 5/10,000 as the epidemiological finding by Lotter suggested, though the prevalence in the Camberwell Study (1979) adding up to 30/10,000 was precluded. The “passport” to access special education, specialized services, or social benefits for handicapped people was set as a DSM diagnosis of autism or PDD-NOS. The social benefits include a so-called “Personalized Budget” (PGB). This is community funded and may add up to € 80,000 per year. The patient/parents may submit a budget including the salaries for those that will provide the support they/their child requires. To date no systematic epidemiological studies have been undertaken in the Netherlands, and it is assumed that the prevalence will be in line with

those of our neighbors with an estimate of around 1% in the total population (Baird et al. 2010). Yet figures from primary education show that nearly 1.4% of the pupils are coined as “leaners with special needs” having received a diagnosis of “autism spectrum disorder” according to DMS V. Likewise, the prevalence of autism in Eindhoven (cradle of high-tech industry) is much higher (2.3%) than in other Dutch towns like Harlem (0.84%) and the university town Utrecht (0.57%). This finding or skewed repartition is in line with findings of higher prevalence of ASD in, e.g., Silicon Valley and Cambridge.

The National Health Council produced two cardinal reports: the first one in 2009 “Autism: a life-long *different*” (<https://www.gezondheidsraad.nl/taak-werkwijze/werkterrein/optimale-gezondheidszorg/autismespectrumstoornissen-een-leven-lang-anders> with a summary in English.) pointed attention to the increasing amount of adults and also the underdiagnosed group of girls and women along with the observation that as modern society speeds up and requires a great flexibility, individuals with ASD are more at risk in schools, universities, and the workplace. This report was followed in 2012 by the report “*Knowledge Infra-structure for Autism Spectrum Disorders*” (https://www.gezondheidsraad.nl/sites/default/files/201209_1.pdf with a summary in English.) notifying the Minister of Health that expertise in the field of Autism should be present in centers countrywide and knowledge on autism spectrum disorders should become available to experts as well as to the general public. Since 2012, multidisciplinary Guidelines are available for professionals and the general public. These address the diagnostic issues as well as the evidence of stimulation, treatment, and support programs. In 2014, the secretary of State for (Mental)Health launched a working group called: “*Autism: insiders views*.” In this working group, {adult}patients and parents of children and adolescents with autism spectrum disorder addressed sixty bottlenecks within the homecare, educational system, leisure time, respite care, working-place offering comments from hand-on experts, and making suggestions for improvement, changes, and innovation. The working group after termination of its advices to the government has pursued its activities in a

coalition “*Taking the perspective of the person with autism*” including more than a hundred patients with specific expertise on one of the sixty issues that from their point of view merit investment and improvement.

Early Detection and Intervention Programs

Many parents with a child with a developmental disorder, e.g., autism, have endured a painful endeavor, being referred from one professional to the other most often with the meant as reassuring news “we cannot tell precisely yet. . . so let us wait and see how your child develops, before getting a proper diagnosis and adequate help.” Extremely frustrating for parents and detrimental for their child as precious time when adequate stimulation could have made a difference. So, very understandably, parents that have had that frustrating experience call for early detection in order to prevent others going through the same nightmare as they themselves.

Three different approaches have been set up to date. SOSO (translated: Screening and Assessment of Social Development) (Publications between 2006 and 2007.) was conducted by the department of Child and Adolescent Psychiatry from the University Medical Centre in Utrecht. It was ambitious in the sense that it aimed at diagnosing children with autism as early in life as possible. The population was a year birth cohort including 30,000 newborn children all screened at the age of 14 months at the community pediatric consultations attended by 99% of all newborns in our country. Parents were asked to fill in a questionnaire, the ESAT (Early Screening for Autistic Traits). When enough “red flags” pointed at a deviant development, the parents were offered a home visit by a trained psychologist and if through direct observation the suspicion was confirmed a thorough assessment with ADOS, ADI, and neuropsychological and neurological/psychiatric assessment followed. The lessons learned were that many parents were reluctant to accept the invitation (though at a later stage they all sought help for their child). A second project

built on these experiences and was called the DIANE (Publications between 2008 and 2011.) (DIagnosing Autism in the NEtherlands) project (Nijmegen area). The team here took a slightly different approach. Community pediatricians were trained to enhance their awareness for autism in very young children. When they detected such a child in their Baby-Wellbeing clinics (attended by 98% of all the children born in the Netherlands), they signaled this. The ESAT was now performed during a home visit where the psychologist observed the child while interviewing the parents. After the formal assessment in the university clinic, the parents were offered a home-based intervention: an interactive program in which parents were trained in better acknowledging the child's messages and needs and to meet them of to discipline them in a far more effective manner. Yet the program was not successfully implemented as the essence lies in the continuous training of the grassroots workers and the involvement and motivation of parents. The third attempt is on its way with yet a more elaborated approach. The Papageno Foundation (Jaap & Aaltje van Zweden) has joined forces to develop a nationwide early detection and intervention network for parents that have concerns about the development of their child. Twenty-two teams of professionals involved in early intervention in autism have joined forces and coordinate their assessment programs in order to offer the same program through the whole country. Moreover, experts in different fields of autism are prepared to give videoconference consultation anywhere in the country to the parents at professionals request. This is aimed at making sure children and their families get the best possible treatment near to their homes. The parents manage the child's file and provide access to all professionals involved. The file is part of a secured electronic environment where forum conversations can take place addressing any questions relevant for any of the participants, thus making parents less dependent on their professional support team. Finally, the network includes a database where all that involved have access to all the relevant literature (with annotations of evidence and relevance). Finally, every referral leads to the home visit, as soon as possible, by a trained

professional who provides a home-based basic intervention to help parents cope with the basics of helping their child given its peculiarities. At the same time, this intervention provides opportunity to observe the child and thus determine which assessment of medical examination would prove most valuable for this child in its social and family context. The first two pilot regions are currently active; further implementation will follow in 2018.

Current Treatment and Centers

Throughout the whole country alongside all university clinics, both mental health institutions and psychologists and psychiatrists in private practice are well knowledgeable of the diagnostic and treatment/guidance needs of individuals with autism spectrum disorders and their families and close dear ones. There are guidelines for all phases of development. And just like the network for early detection and intervention, there are networks for adults with autism (CASS 18+) and a special network for autism in women and girls (Women Inc. and FANN). Finally, adults with autism spectrum disorder are well organized in an own organization (PAS) that offers advice and combats stigmatization in the workplace.

Education and Autism Spectrum Disorders

A DSM classification of ASD qualifies those children as a learner with special needs. Extra support can be given in the classroom in a regular school both for remedial teaching or addressing challenging behavior or anxieties in the classroom/playground situation. But if necessary, the Dutch education system includes special schools in different categories. Learners with autism can be helped in so-called Cluster 2 schools for learners with hearing, speech, and communication problems; Cluster 3 schools for learners with developmental delays; and finally cluster 4 for learners with emotional and/or conduct problems. The beauty of the system is that it offers specific

support or a secure environment for children and adolescents with autism spectrum disorders regardless their intellectual capacities. The vulnerability of a system is much adapted to the peculiarities that it makes integration in the “real world” where individuals with ASD will to a certain degree have to adapt to less ideal situation, more difficult.

Autism and Comorbidity

Specialized Centers for Autism (e.g., Centrum Autisme Leiden (<https://www.rivierduinen.nl/autisme>) and/or Centrum OntwikkelingsStoornissen (Centre for Developmental Disorders) Dimence GGz Deventer (<https://centrumontwikkelingsstoornissen.dimence.nl/>)) and the University Clinics offer countrywide consultation for (Mental Health) professionals encountering difficulties in coping with and offering adequate treatment for individuals with autism with comorbid conditions ranging from learning disability, cerebral palsy, psychosis, depression, addiction, etc.

Overview of Research Directions

The Netherlands has a solid track record in autism research in a great variety of areas. These include taxonomy, neuroimaging, and electrophysiology as well as neuropsychological studies. Strangely no countrywide epidemiological studies have been conducted in the Netherlands, though some studies suggest that the prevalence of autism spectrum disorders may be quite high.

Social Policies

Healthcare in the Netherlands is undergoing a profound change. More and more healthcare is integrated with (social) welfare. Healthcare is supported by a national insurance scheme in which all citizens are enrolled for reimbursement of basic healthcare. If one cannot afford it, the social welfare provides assistance. Social welfare is provided by the municipalities, which

are responsible for the well-being of their inhabitants. Care for people with a handicap or chronic condition is offered in various manners: they can receive support given according to national guidelines either from healthcare agencies of welfare providers. They can also opt for a “Personalized Health Budget.” The individual, his parents, or legal mentors present a budget plan including the cost of help and support they estimate required for the well-being of the individual concerned and for which budget they are accountable year after year.

The national report “Autism: a lifelong *different*” also looks into the requirements for education, meaningful daytime activities, and preferably integration in paid labor.

Finally, individuals with autism spectrum disorder, can from their 18th birthday on, benefit from an invalidity pension provided by the state (WAJONG: “invalidity bill for youth”) that will support them lifelong, and is interrupted if the individual finds paid work.

Tentative Conclusions

Autism spectrum disorders are quite a common problem in the Netherlands. Despite the absence of any systematic epidemiological data, the figure of 1.4% learners with ASD in primary education reflects a high prevalence of autism spectrum disorders in this country.

Along with a nationwide network for early detection, services for individuals with ASD and their families are well organized. Social welfare and health insurance provides the necessary support.

References

- Oosterling IJ, Wensing M, Swinkels SH, van der Gaag RJ, Visser JC, Woudenberg T, Minderaa R, Steenhuis MP, Buitelaar JK (2010) Advancing early detection of autism spectrum disorder by applying an integrated two-stage screening approach. *J Child Psychol Psychiatry* 51(3):250–8.
- Salomone E, Beranová Š, Bonnet-Brilhault F, Briciet Lauritsen M, Budisteanu M, Buitelaar J, Canal-Bedia

R, Felhosi G, Fletcher-Watson S, Freitag C, Fuentes J, Gallagher L, Garcia Primo P, Gliga F, Gomot M, Green J, Heimann M, Jónsdóttir SL, Kaale A, Kawa R, Kylliäinen A, Lemcke S, Markovska-Simoska S, Marschik PB, McConachie H, Moilanen I, Muratori F, Narzisi A, Noterdaeme M, Oliveira G, Oosterling I, Pijl M, Pop-Jordanova N, Poustka L, Roeyers H, Rogé B, Sinzig J, Vicente A, Warreyn P, Charman T (2016) Use of early intervention for young children with autism spectrum disorder across Europe. *Autism* 20 (2):233–49.

Network Coherence

► Brain Connectivity Theories of Autism

Neural Mechanisms in Autism

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Definition

Minicolumns are elemental anatomical and functional modules of the neocortex that serve as cytoarchitectural templates for the arrangement of cells and their connections. A chain of excitatory neurons, along with their axonal and dendritic bundles, constitutes its core. These excitatory neurons extend radially, i.e., perpendicular to the neocortex, through layers II to VI. Among other anatomical elements present at the periphery of the minicolumn are (a) synaptic elements which integrate translaminar connections and (b) interneurons that provide for a vertical sheath of surround inhibition to the information being processed by the core. The minicolumn exhibits variability between different brain regions, across hemispheres within a given brain, as well as among individuals within and across species. Nevertheless, the basic organization of microcircuitry is maintained throughout the cortex of all mammals (Buxhoeveden and Casanova 2005; Douglas and Martin 2004).

Historical Background

The neocortex covers the massive network of myelinated fibers in the white matter like the crust on a camembert, yet the vast majority of connections in the brain are not provided by corticocortical projections in the white matter. The majority of cortical connections take place between neighboring cells in a dimensional scale spanning only a few microns. These connections are typically contained within stereotyped microcircuits which are distributed iteratively throughout the cortex. Early neuroanatomists, like Cajal and Meynert, were among the first to describe the anatomical skeleton of these circuits when they noted the distribution of pyramidal cells in recurring arrays perpendicular to the pial surface (DeFelipe 2005). von Economo and Koskinas called them “radii” as they paralleled the radial arrangement of white matter bundles within the cortex (von Economo and Koskinas 1925). Other distinguished neuroscientists further defined the anatomy of these modules as consisting of a pyramidal cell column core with synaptic elements and inhibitory interneurons at its periphery (Peters and Sethares 1991, 1996; Szentágothai 1978). It is now known that these circuit arrangements, termed “minicolumns” by the pioneering neurophysiologist Vernon Mountcastle, feature a polarity in which information ordered by evolution and development is represented in the topographical pattern of minicolumnar inputs directed vertically toward inputs from across the cortex (Casanova 2008a, 2010; Casanova and Tillquist 2008; Mountcastle 1998).

Although the presence of these vertical arrangements cannot be questioned, a functional role was never suggested or described by early researchers. It was not until the late 1930s that these anatomical units acquired physiological significance as elementary units of information processing. Based on his experience with the Golgi silver impregnation technique, Rafael Lorente de Nó (1938) proposed the existence of clusters of cells arranged in vertical modules that applied canonical operations on thalamocortical and corticocortical inputs. This idea receives some anatomical support from the fact that the

neocortex unvaryingly uses layers II and III for association (e.g., corticocortical integration), layer IV for reception, and layers V and VI for efferent connectivity. The work of Lorente de Nó antedated our current understanding of computer science wherein a single logic gate (i.e., NOR or the minicolumn in our case) in different configurations can provide for higher-level functions capable of implementing any computer program (Casanova et al. 2003). During the last few decades, many authors have revived and expanded de Nó's idea of a canonical neocortical circuit (Creutzfeldt 1995; Douglas and Martin 2004).

Current Knowledge

The advantage of short, stereotyped microcircuits contained within minicolumns is that patterns of information can be represented by synchronous outputs coordinated among units. The white matter connections linking distant cortical areas are sparse thus providing a “small-world” architecture which minimizes wiring. The end result is a balance between neural clusters and wiring that minimizes connection steps between local circuits across the neocortex. In effect, most neurons maintain short connection lengths within clusters that themselves are linked by longer range projections. The preferred clustering configuration for neurons within the neocortex is the minicolumn (Casanova 2010).

Ontogeny

Pyramidal cell arrays are derived from the primordial neuroepithelium, i.e., progenitor cells that attach themselves both to the ependymal (ventricular) and pial surfaces. Their epithelial origin is betrayed by the presence of tight junctions within their end feet which together constitute a pia externa glia limiting membrane. The dividing neuroepithelium provides for migrating neuroblasts that wedge themselves between the first (primordial) lamina and the subplate. Neurons fated for the infragranular layers are thought,

during asymmetric division, to inherit the pial fiber from the radial glial cell, which then allows the neuroblast to translocate into the lower layers of the cortical plate. In contrast, neural progeny meant for superficial cortical layers attaches and locomotes along the radial glial scaffolding until the neuroblast's pial-directed extension reaches the marginal zone, at which point it disengages from the radial glial scaffolding and finishes its journey via somal translocation (Casanova et al. 2009; Williams and Casanova 2011a). As neuroblasts lose their glial attachment, they begin to mature into pyramidal cells having as a common characteristic their attachment by their apical dendrites to the first lamina. In humans, the cortical plate starts to form at approximately the 7th week of gestation and is almost completed by the 15th week of gestation (Marin-Padilla 2011). Interneurons are later on coupled to the pyramidal cell arrays following an inside out pattern juxtaposed on concomitant lamination, vascularization, and the introduction of protoplasmic astrocytes (Marin-Padilla 2011). Four excitatory-inhibitory systems have been described in the literature: pyramidal Martinotti, pyramidal basket, pyramidal chandelier, and pyramidal double bouquet cells. The end result of this complex radial glia-mediated migration (pyramidal cells) and coupling to horizontal transversing neuronal precursors (interneurons) is a cortical plate that is a unique mammalian innovation (Gressens and Evrard 1993).

Although minicolumns are readily recognized in fetal/embryonic material, the characteristic radial configuration is less readily ascertained with aging. This may be due to the translation of pyramidal cells around the central axis of the minicolumn, growth or atrophy of anatomical elements, and the introduction of glial elements. At least two studies have looked at the continuity between the ontogenetic minicolumn and its putative postnatal counterpart. Krmpotic-Nemanic et al. (1984) using a limited series of human fetal material from the auditory cortex traced the developmental transformation of ontogenetic cell columns into mature minicolumns. Similar results were observed in a larger series ($n = 67$) of human postmortem specimens that modeled the

rate of change in neuropil space (Casanova et al. 2007). The latter study used Nissl-stained sections and computerized image analysis to measure the median-free path through the neuropil in the radial and tangential directions. No significant change was noted in the ratio of both directions in either the prenatal or postnatal sample population. The maintenance of constant ratios of radial to tangential median neuropil space over the course of development suggests that these morphometric relations reflect an intrinsic organization of circuitry present from the earliest stages of cortical development. Results from the study also suggested that the total cell numbers within a minicolumnar lamina and the corresponding minicolumnar dimensions are constrained by packing limits imposed by their associated vertically oriented processes (Casanova et al. 2007). In effect, corticalization may be determined by the total number of radial units (i.e., radial unit hypothesis of Rakic) only as they ultimately define the total number of minicolumns in the neocortex (Rakic 2003).

Recent studies have given credence to Rakic's radial unit hypothesis (Rakic 1995). It is now commonly accepted that changes in the number of germinal cell divisions giving rise to minicolumns provide for corticalization and brain parcellation (Casanova and Tillquist 2008). Thus, a single microinjection of fibroblast growth factor (FGF) 2 into the cerebral ventricles of rat embryos doubles the number of pyramidal cells while opposite results are noted in a knockout mouse of the *Fgf2* gene (Vaccarino et al. 1999). Similarly, preliminary work on the mouse model for velocardiofacial syndrome suggests that deletion of regulatory genes that control the cell cycle for germinal cells provides for changes in both brain volume and the specification of cells within different cortical layers (Tucker et al. 2008).

Morphometry (Lateralization and Minicolumnar Width)

Comparative neuroanatomy has long studied the existence and meaning of asymmetries in various parts of the human and nonhuman primate brains.

In areas of the human brain associated with language, such as the planum temporale, MRI studies reported essentially identical asymmetries in humans and in African great apes but not in the Old or New World monkey (Gannon et al. 1998; Hopkins et al. 1998). However, an examination of minicolumns between the two hemispheres revealed that minicolumns in homologous regions of the cortex can be lateralized. Perhaps Seldon (1981a) was the first researcher to report an example of minicolumnar asymmetry in his studies of the human auditory cortex. Seldon (1981a) found that cell columns and neuropil space were wider in the left hemisphere as compared to the right. Buxhoeveden et al. (2001) examined cell columns in both hemispheres of humans, chimpanzees (*Pan troglodytes*), and monkeys (*Macaca mulatta*). They reported that minicolumns were lateralized in humans but not in the chimpanzee or monkey brain. The asymmetry consisted of wider cell columns in the left hemisphere. The asymmetrical morphology of cell columns within a brain is highly suggestive of organizational differences between hemispheres (Seldon 1981a, b). Still, despite these interhemispheric differences, gross variability in minicolumnar width appears to be constrained within a brain and within particular species.

In a sample population of autopsy specimens from 67 human patients (*vide supra*; Casanova et al. 2007), five brain regions (areas 4, 17, 21, 22, and 40) were selected to assess minicolumnar width. The width of minicolumns averaged between 40 and 50 μm . The mean minicolumnar width for association cortex (area 40) was 46.8 μm and 31.6 μm for primary visual cortex (area 17). One might conclude that the mean size of minicolumns in human brain falls within a specific range that is generally greater than that of other primates but seemingly equal or smaller than that reported for many other small-brained mammals such as the cat, rabbit, or rat (Buxhoeveden and Casanova 2005; for a review see Casanova 2008a). Comparisons of independent studies in human cortex confirm these numbers (Buldyrev et al. 2000; Del Río and DeFelipe 1997a, b; Seldon 1981a; Schlaug et al. 1995). A study of minicolumns in humans was done by

creating a GLI-profile analysis wherein a linear combination of parameters provided a verticality index (a measure of columnarization) (Schlaug et al. 1995). Regions of the cingulate cortex were examined in normal humans using digitized images of Nissl-stained material. The authors reported a mean minicolumnar spacing of 41.7 μm . Of the five areas examined, area 7 (periculate) displayed the smallest column width of 39.9 μm and area 31 was the largest with 46.6 μm .

Molecular Biology

The genetics underlying neocortical development in autism is complex, reflective of the intricacies of stem cell proliferation, differentiation, migration, neurite outgrowth, and synaptic development. Particular to a significant number of cases of autism is an increase in total minicolumnar number within the neocortex, indicative of an increase of the neural stem cell population (Casanova et al. 2002a). Subject to genetic and epigenetic influences are numerous pathways involved in cell cycle regulation, cellular proliferation, and cell survival. Activation or inhibition of key signaling molecules along these pathways has the potential to upregulate proliferation of the neural germinal population, thereby increasing total minicolumnar number. Targeting of even one or a few molecules can affect activation of related pathways due to crosstalk among them. In addition, upregulation of these pathways may alter migration, neuritogenesis, and synaptogenesis as well because these pathways have been exapted for a variety of forms of growth and development within the cell, linking aberration in cellular proliferation and later minicolumnar morphometry to abnormalities in cell-to-cell connectivity (for review, see Williams and Casanova 2011b). Genetic and epigenetic animal models of autism can illustrate how varying genotypes lead to a common neuroanatomic phenotype. For example, in a subtype of autism spectrum disorders known as Rett syndrome, approximately 80% of cases display a germline mutation of the methyl-CpG-binding protein 2 (*MECP2*) gene on the X chromosome. Due to paternal parent of

origin effects, Rett syndrome mainly targets females. Knockout mice heterozygous for *Mecp2* expression are now used to study the condition at the molecular, neuroanatomical, neurophysiological, and behavioral levels. *MECP2* is a regulatory molecule intimately involved in methylation and deacetylation of DNA within the central nervous system, forming complexes which frequently repress promoter activation. Researchers have found that DNA which is not both methylated and deacetylated remains available for transcription, indicating the vital regulatory position this complex occupies. Even though *MECP2* is ubiquitously transcribed, it exhibits temporal heterogeneous expression in frontal cortex, as *MECP2*^{hi} levels increase with postnatal age and *MECP2*^{lo} levels simultaneously decrease with age (Samaco et al. 2004). This increase in *MECP2*^{hi} levels may help to explain the timing of regressive events seen in Rett syndrome and may offer some insight into regression in idiopathic autism as well. While idiopathic autism rarely presents with *MECP2* mutations, the conditions often exhibit abnormal *MECP2* activation, indicating aberrant regulation upstream of this molecular complex (Samaco et al. 2004).

Another intracellular signaling molecule, phosphatase and tensin homolog (PTEN), is encoded on chromosome 10q23 and regulates cellular growth in part by inhibiting PI3K/Akt pathway activation. While autism is a highly heterogeneous group of conditions with a wide variety of associated mutations, approximately 7% of the autistic population harbors *PTEN* germline mutations (McBride et al. 2010). Within this subpopulation, autism is almost always associated with co-occurring macrocephaly, and in heterozygous *Pten* knockout mice, this macrocephaly is especially apparent. Therefore, given the high rates of *PTEN* mutations within the autistic population, these mice have been proposed as a viable animal model for autism (Kwon et al. 2006).

Tuberous sclerosis is a single gene condition in which 25–50% of those affected also exhibit autistic-like symptoms. The mutations which give rise to the condition target the gene products, tuberous sclerosis complex (TSC) 1 or 2, although

mutations in *TSC2* are more frequently associated with co-occurring autism (Jones et al. 1999). A mutation within either *TSC 1* or *2* can significantly alter its functionality, decreasing inhibition of the mTOR pathway, which is a sensitive detector of growth factors, insulin, and oxidative stress and which also maintains a prime position to either upregulate or downregulate growth based on this incoming information. The resultant phenotype of the condition presents with global or localized forms of megalencephaly, tubers, subependymal nodules, and other proliferative and migratory malformations. Of interest, similar malformations have also been noted within idiopathic autism (Bailey et al. 1998; Wegiel et al. 2010), illustrating how genetic heterogeneity can still lead to a common phenotype.

Recently, the Autism Genome Project Consortium published work comparing 996 autistics to 1,287 matched controls and found significant copy number variations across three functional domains: (1) cellular proliferation, (2) cell projection and motility, and (3) GTPase/Ras signaling (Pinto et al. 2010). While GTPase/Ras signaling may be subsumed under the previous two functions, neuroscientists have had a tendency to view proliferation and projection/motility as distinct cellular processes, and their research have subsequently “split” them by their differences rather than “lump” them by their inherent similarities. But the molecular parsimony which exists between cellular proliferation, neurite outgrowth, synaptic development, and cell movement is considerable. *Molecular parsimony*, otherwise known as the “small toolkit” of molecular biology, suggests that evolution consistently exapts the same types of molecules and signaling pathways to perform new and related functions, such that conserved “families” of genes emerge within and across species. Therefore, the division of a cell into two, the podlike projection of a neuritic arm, the budding of a new synapse, or the slinking movement of a cell along its path have each been refined from a baser process so that they now serve different purposes. And the baser process from which these functions have been borrowed is the general process of growth and division: a common thread tying many, if not all, forms of autism

together. Viewing these conditions in a broader molecular light may help us understand how individuals with such seemingly disparate genetic backgrounds, and arising from complex ecologies, can each be at risk for the development of autism.

Minicolumnar Networks: Electrophysiology and Circuitry

The minicolumn is defined by cytoarchitectural arrangements of cell elements aligned with the core of pyramidal cells and the apical dendrite and myelinated axon bundles arising from them. Apical dendrite bundles and myelinated axon bundles vary in number and laminar origin and cell type of their constituents, according to species and area. They are spaced in register with pyramidal cell columns and interneurons; the distribution and structure of these constituents provide complementary parameters for morphometric analysis. The basis for the computational flexibility of microcircuit functions in the minicolumn lies in the regulation of the core pyramidal cells’ electrotonic properties by interneuron class-specific networks and their synaptic distributions in the various membrane compartments of pyramidal cells. Coordinated inhibition on proximal dendritic segments results in the functional segregation of the soma and distal dendrite compartments. Tonic inhibition applied to basal dendrites or proximal to the apical dendrite tuft in layers I and II disrupts excitatory postsynaptic potentials (EPSPs) arising from cells in neighboring minicolumns and limits their input. Moreover, the disposition of interneuron connections modulates activity in the recurrent pyramidal cell circuits whose positive feedback properties render them unstable. Interneuron regulation of the reverberant properties and gain of these circuits allow for the maintenance of information in each minicolumnar circuit in a metastable state. In this way, a signal input could allow for the mutual relaxation of minicolumn subsets into a stable state, providing a basis for pattern completion or matching. Thus, each subclass of interneuron plays critical functional roles in control of trans-laminar circuits.

In the canonical cortical microcircuit proposed by Douglas and Martin (2004), the flow of excitatory information from upstream areas or thalamocortical inputs is directed toward middle layers. As was noted earlier, most circuits in cortex are recurrent and local. In cat visual cortex, thalamocortical synapses were calculated to constitute less than 7% of the total number of inputs to layer IVc spiny stellate cells (Ahmed et al. 1994). While sparse, this input is highly divergent and convergent both on other spiny stellate stars and on target pyramidal neurons in layer II/III (Lübke et al. 2000, 2003). Thalamocortical inputs also provide direct input to GABA neurons, primarily parvalbumin-expressing (PV+) basket cells or vasoactive intestinal peptide (VIP+) bipolar cells (Freund et al. 1985; Hájos et al. 1997; Staiger et al. 1996, 1997). Subpopulations of spiny pyramidal and stellate neurons in layer IV form precise retinotopic patterns in primary visual cortex (VI) of cats and rodents (Burkhalter 1989; Parnavelas et al. 1977). What is notable is that these precise patterns of inputs feed ordered arrangements of fine-scale microcircuits. Double cell intracellular recordings of pyramidal cell pairs in layer II/III (Yoshimura et al. 2005) revealed that pyramidal cell pairs which received common afferents were significantly more likely to be reciprocally connected. This specificity was also established for connections between fast-spiking interneurons and pyramidal neurons, with fast-spiking interneurons preferentially forming reciprocal rather than one-way connections with pyramidal cells, providing a dedicated feedback circuit (Yoshimura and Callaway 2005). In addition, these interneurons only received input from pyramidal neurons which were already interconnected or from strong layer 4 afferents. Fast-spiking interneurons were dedicated to fine-scale pyramidal cell circuits, thereby providing a strong source of feature-tuned feed-forward inhibition to these circuits (Yoshimura and Callaway).

The brain's limited long-range wiring cannot directly sustain coordinated activity across arbitrary cortical locations, but it can convey *patterns* of synchronous activity as oscillatory neuronal fluxes, represented by local field potentials measured by electroencephalography (EEG).

Coordination of oscillations at varying interacting frequencies allows for relatively efficient and unconstrained segregation or integration of cognitive representations in varying forms and across hierarchical levels. Disrupted patterns of coordinated oscillatory output in distributed minicolumnar networks might be associated with cortical disconnection in autism. Likewise, altered oscillatory activity in developing cortical circuits may contribute to impaired development of intra-areal and transcortical connections (Casanova and Trippe 2009).

Minicolumnar Networks: Neurochemistry

Oscillations of pyramidal cells in minicolumns and across assemblies of minicolumns are maintained by networks of different species of inhibitory, GABA-expressing, interneurons. GABA neurons are classified according to characteristic combinations of morphological, physiological, or molecular phenotypes (Ascoli 2008). Cell morphologies are conventionally classified as basket cells (interneurons having a large proportion of terminals on soma or proximal dendrites), chandelier cells (defined by vertical arrays of cartridge terminals aligned on pyramidal cell initial segments), and double bouquet cells (interneurons having predominantly radially aligned axodendritic arbors). Most interneurons express only one of the calcium-binding proteins parvalbumin or calretinin, or the neuropeptide somatostatin but not the others. Radially oriented interneurons containing calretinin commonly coexpress vasoactive intestinal peptide (VIP). Basket cells and chandelier cells containing parvalbumin have a fast-spiking synaptic dynamics with high amplitude. Other basket cells containing the neuropeptide cholecystokinin, and never parvalbumin, have irregular-spiking profiles. Radially oriented cells typically have an accommodating broader synaptic profile which varies according to cell type and connections. While tangentially arrayed basket cells function, in part, to coordinate activity among remote neuronal ensembles, by contrast, radially oriented

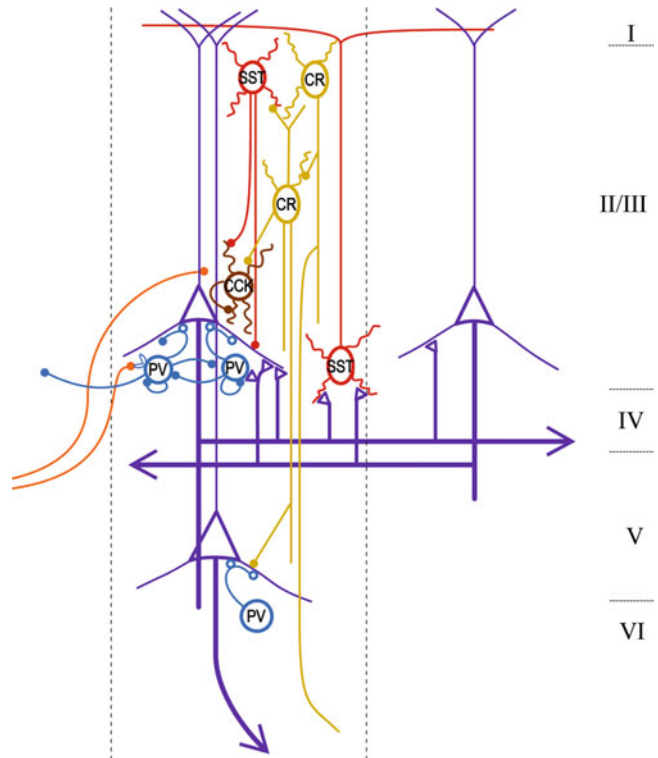
inhibitory interneurons prominently located in the peripheral neuropil space surrounding pyramidal cell columns likely function to segregate columns from interference, both from other minicolumns within an array and from fields of activity or inhibition in neighboring minicolumnar arrays.

Cholinergic inputs to cells of either cholecystokinin (CCK+) or PV + basket cell subclasses are complementary and nonoverlapping (Fig. 1). Cholinergic activation of alpha-7 subunit-containing nicotinic receptor expressed in CCK + basket cells results in depolarization and increased GABAergic output. In rodent hippocampus, CCK + cell depolarization profiles vary according to interneuron size or molecular features (Kawaguchi 1997). By contrast, PV + interneurons express presynaptic muscarinic M₂ receptors (Hajos et al. 1998) which serve to inhibit GABA release from PV + basket cell and GABAergic drive on pyramidal neurons. Given acetylcholine's well-established role in refining neuronal circuits and in increasing attention and perceptual feature selectivity (Hajos et al. 1998;

Kawaguchi 1997), this complementary expression pattern suggests a mechanism whereby increased presynaptic activation of CCK + interneurons and modulation of their gamma-band activity facilitates dynamic restructuring of connections between established networks in response to environmental or state-dependent cholinergic inputs; "clocklike" (Freund 2003) oscillatory drive of PV + neurons is suspended, and long-term plasticity of these interneuron networks will depend on the circuit remodeling driven by CCK + cells. Disruptions of the dynamic GABAergic balance synergistically regulated by cholinergic pathways, endocannabinoids, monoamines, and other neuroactive molecules would therefore affect the integration of local microcircuits into wider networks, a pathogenetic trajectory consistent with our current understanding of the behavioral pathology of autism as a "disconnection syndrome" (Casanova et al. 2006a; Just et al. 2004).

CCK + basket cells are notable for the restricted axodendritic arbor field to within

Neural Mechanisms in Autism, Fig. 1 Role of CCK cells within minicolumnar cell architecture (see text for explanation). *Circles* represent interneurons, *triangles* represent pyramidal cells. Abbreviations: cholecystokinin (CCK), calretinin (CR), parvalbumin (PV), and somatostatin (SST) *Right:* laminae; *dotted, vertical lines:* minicolumnar boundaries



minicolumnar dimensions, together with radial interneurons expressing varying combinations of calretinin (CR), calbindin (CB), VIP, or somatostatin (SOM). As recently shown in monkey prefrontal cortex (Zaitsev et al. 2009), some CR + radial interneurons extending across most cortical layers or into white matter give rise to perisomatic baskets in superficial layers which surround small cell profiles resembling interneurons. Moreover, CBR1 mRNA and protein expression are distributed along a vertical translaminal gradient, with mRNA expression most intense in superficial layer II and CBR1 immunoreactivity increasing to a maximum in deep layer III (Eggan and Lewis 2007; Zaitsev et al. 2009). One implication of this finding is that CBR1/CCK + basket cell terminals may have a vertical distribution extending from their cell body. Radially oriented interneurons may therefore serve to coordinate activity of CCK + basket cells across layers of a minicolumn thereby regulating development of its recurrent circuits during development. CCK + interneuron circuits are more susceptible to disruption than are PV + interneurons and that disruption has greater pathological impact (Freund 2003). This may indicate that they, along with CR + or VIP + radial interneurons, serve as “keystones” in interneuronal networks driving synchronous interneuron activity across supra- and infragranular layers of a minicolumn.

Minicolumnopathy of Autism

Recent postmortem studies have found area-specific alterations in minicolumnarity. All of these studies have been carried out targeting pyramidal cell arrays in Nissl stains as immunocytochemical markers may prove unreliable given the long postmortem intervals before autopsy and the variability in agonal conditions between patients and normative individuals. The first study was provided by Casanova et al. (2002a) using celloidin-embedded Nissl material from nine autistic patients and an equal number of controls. Three brain areas (9, 21, and 22) were studied. In this study, the brains of autistic individuals were found to have smaller minicolumns ($p = 0.034$)

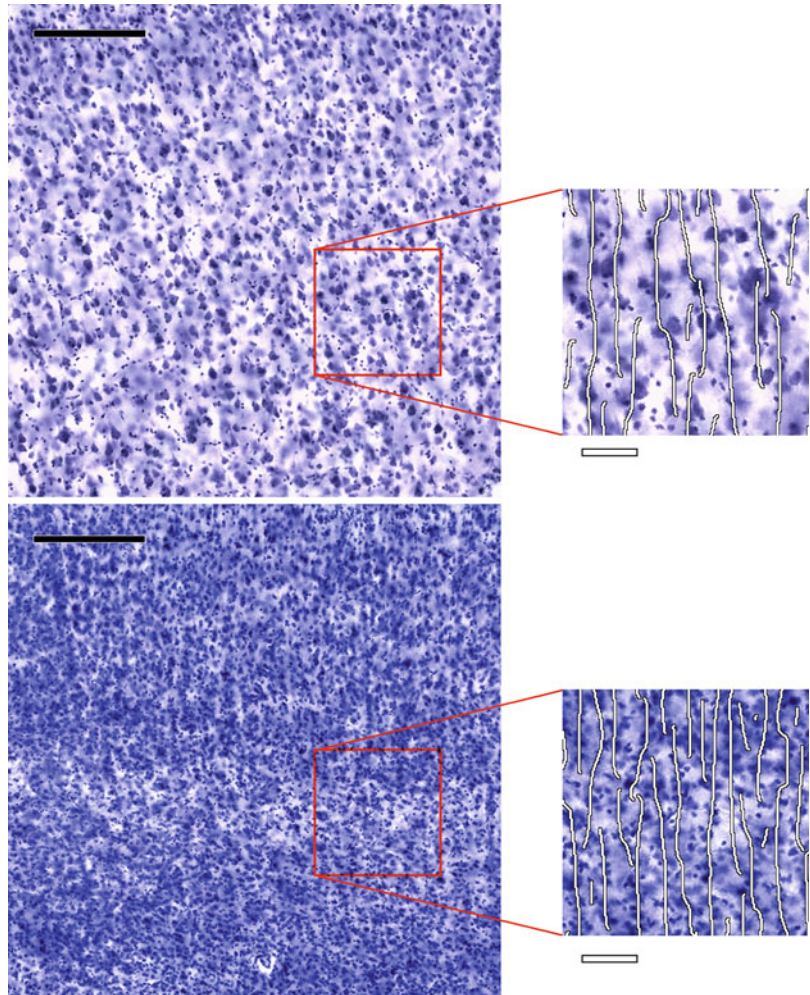
with most of the decrease attributable to a reduction in the peripheral neuropil space. Results were later on validated when the same series was analyzed according to the gray level index (GLI) (Casanova et al. 2002b).

A more recent study used an independent sample to analyze minicolumnarity within representative primary sensory, motor, and prefrontal association cortices (areas 17, S1, 4, and 9) (Casanova et al. 2006a). Differences in tissue preparation and section thickness prevented direct comparisons with earlier studies. However, minicolumnar width was significantly diminished in autistic individuals as compared to controls ($p = 0.023$) (Fig. 2). The finding was also corroborated by using GLI measures of distance between amplitude peaks. Nucleolar area was thresholded from the background and found to be smaller in autistic individuals. A Boolean model found greater density of Nissl-stained elements (neurons). A Delaunay triangulation method suggested a diminution in the distance between minicolumns but an otherwise normal complement of neurons within minicolumns. The authors concluded that the decrease in neuronal and nucleolar size noted in autistic subjects could constrain the length of corticocortical projections thereby biasing their circuitry toward shorter connections (arcuate fibers) at the expense of longer ones (i.e., commissural projections) (Casanova et al.).

A study of minicolumnar abnormalities in postmortem samples of brains of autistic individuals and controls was expanded on previous findings by exploring nine cortical areas that included paralimbic, heteromodal association, unimodal association, and primary or idiopathic areas (Casanova et al. 2006b). Computerized image analysis clustered neurons into minicolumnar fragments. The authors found an interaction of diagnosis and region for neuropil space ($p = 0.041$). Post hoc analysis showed significant differences for the frontopolar region (area 10) and the anterior cingulate gyrus (area 24). The smaller minicolumns in autism translate into more of them per unit of length. The assumption of an increase total number of minicolumns was modeled to true columnar parameters (three dimensions) with a strong correlation (Casanova and Switala 2005).

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Fig. 2 Minicolumns in a section of primary motor cortex (Brodmann area 4), lamina III, in an autistic patient (*bottom*) and an age-matched control case (*top*). *Insets* highlight the cores of minicolumn fragments. Scale bars measure 200 μm on *left* and 50 μm on *right*



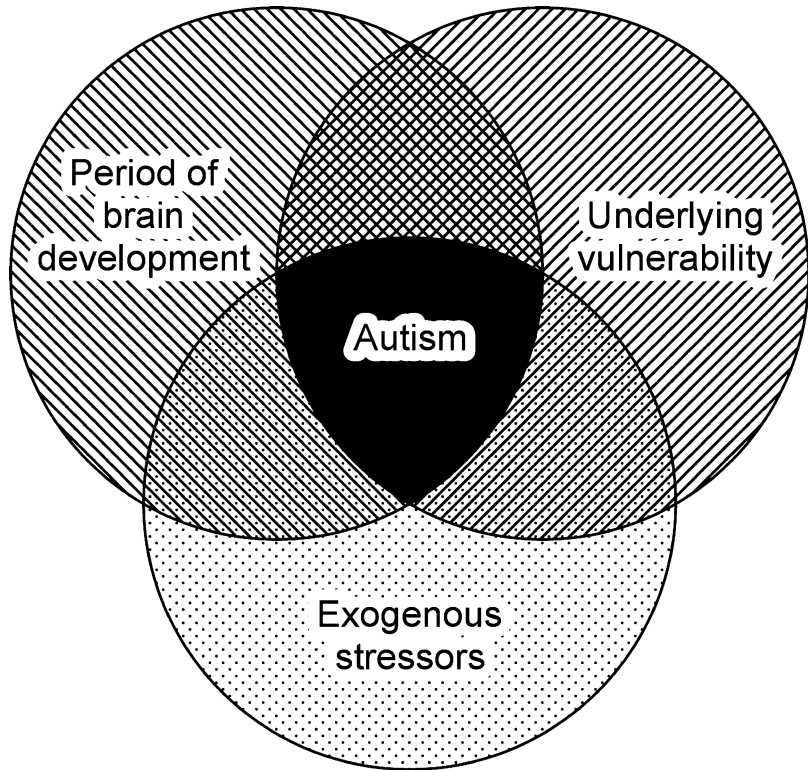
Estimates of cortical interconnectivity suggest that each module (minicolumn) is connected to 10^3 other modules (Casanova 2004). If connectivity is fixed as brain size increases, the number of connections would scale as the number of modular units squared. Most of the addition in white matter takes the form of short corticocortical fibers as longer connections incur a penalty of increased conduction time and metabolic demands. Not surprisingly, MRI studies parcellating the white matter have found a disproportionate increase in the outer white matter compartment (short late myelinating fibers) as opposed to inner compartment containing longer bridging connections (Herbert et al. 2004).

Future Directions

Genome-wide linkage screen have refuted the concept of autism as having a Mendelian inheritance and rather sustain the condition as a multifactorial disorder. The author has proposed a “triple hit hypothesis” where manifestations supervene whenever three factors converge on a particular infant: (a) a critical period of brain development, (b) an underlying genetic vulnerability, and (c) exogenous stressor(s) (Casanova 2008b) (Fig. 3). While it is important to investigate the genetics of autism, it is also important to remain aware that phenotype is a combination of genetic and epigenetic influences. And therefore, environmental agents which target pathways

Neural Mechanisms in

Autism, Fig. 3 Autism is a multifactorial disorder where manifestations supervene when three factors impinge to various degrees upon a particular infant: (1) a critical period of brain development, (2) an underlying vulnerability (genetics), and (3) exogenous stressors (Casanova 2008a)



common to autism may lead to comparable phenotype yet not be reflected within the genotype. A variety of teratogenic agents have already been associated with the conditions, and more epigenetic influences will likely come to light with further research (Williams and Casanova 2011a). The overriding theme of autism is that overactivation of key growth-related pathways during vulnerable time periods of development may lead to the conditions (Levitt and Campbell 2009; Williams and Casanova 2011a).

The value of understanding the putative minicolumnopathy of autism lies in its explanatory powers and translational possibilities. Early results predicted changes in brain volume, alterations in the ratio of gray to white matter, bias in white matter parcellation (inner vs. outer white matter), and abnormalities in gamma-binding frequencies (Casanova et al. 2002c). More recently, the idea that autistic individuals have defects in their minicolumnar peripheral neuropil space has provided for a possible clinical intervention using rTMS to strengthen the inhibitory surround of

these modules (Sokhadze et al. 2009a, b, c). Other therapeutic attempts based on the minicolumnopathy of autism include the shifting of the terminal fields of sensory stimuli as a way of compensating for the bias in short connections observed in this condition.

See Also

- Cerebral Cortex
- Pyramidal System
- Rett Syndrome
- Tuberous Sclerosis Complex

References and Reading

- Ahmed, B., Anderson, J. C., Douglas, R. J., Martin, K. A. C., & Nelson, J. C. (1994). Polyneuronal innervation of spiny stellate neurons in cat visual cortex. *The Journal of Comparative Neurology*, 341, 39–49.
- Amir, R. E., Van den Veyver, I. B., Wan, M., Tran, C. Q., Francke, U., & Zoghbi, H. Y. (1999). Rett syndrome is

- caused by mutations in X-linked *MECP2*, encoding methyl-CpG-binding protein 2. *Nature Genetics*, 23, 185–188.
- Ascoli, G. A. (2008). Neuroinformatics grand challenges. *Neuroinformatics*, 6, 1–3.
- Bailey, A., Luthert, P., Dean, A., Harding, B., Janota, I., Montgomery, M., et al. (1998). A clinicopathological study of autism. *Brain*, 121, 889–905.
- Buldyrev, S. V., Cruz, L., Gomez-Isla, T., Gomez-Tortosa, E., Havlin, S., Le, R., et al. (2000). Description of microcolumnar ensembles in association cortex and their disruption in Alzheimer and Lewy body dementias. *Proceedings of the National Academy of Sciences of the United States of America*, 97, 5039–5043.
- Burkhalter, A. (1989). Intrinsic connections of rat primary visual cortex: Laminar organization of axonal projections. *The Journal of Comparative Neurology*, 279, 171–186.
- Buxhoeveden, D., & Casanova, M. F. (2005). The cell column in comparative anatomy. In M. F. Casanova (Ed.), *Neocortical modularity and the cell minicolumn* (pp. 93–116). New York: Nova Biomedical.
- Buxhoeveden, D. P., Switala, A. E., Litaker, M., Roy, E., & Casanova, M. F. (2001). Lateralization of minicolumns in human planum temporale is absent in nonhuman primate cortex. *Brain, Behavior and Evolution*, 57, 349–358.
- Casanova, M. F. (2004). White matter volume increases and minicolumns in autism. *Annals of Neurology*, 56, 453.
- Casanova, M. F. (2008a). The significance of minicolumnar size variability in autism: A perspective from comparative anatomy. In A. Zimmerman (Ed.), *Autism: Current theories and evidence* (pp. 349–360). Totowa: Humana Press.
- Casanova, M. F. (2008b). The neuropathology of autism. *Brain Pathology*, 14, 101–118.
- Casanova, M. F. (2010). Cortical organization: Anatomical findings based on systems theory. *Translational Neuroscience*, 1, 62–71.
- Casanova, M. F., & Switala, A. E. (2005). Minicolumnar morphometry: Computerized image analysis. In M. F. Casanova (Ed.), *Neocortical modularity and the cell minicolumn* (pp. 161–180). New York: Nova Biomedical.
- Casanova, M. F., & Tillquist, C. R. (2008). Encephalization, emergent properties, and psychiatry: A minicolumnar perspective. *The Neuroscientist*, 14, 101–118.
- Casanova, M. F., & Trippe, J. (2009). Radial cytoarchitecture and patterns of cortical connectivity in autism. *Philosophical Transactions of the Royal Society, B. Biological Sciences*, 364, 1433–1436.
- Casanova, M. F., Buxhoeveden, D. P., Switala, A. E., & Roy, E. (2002a). Minicolumnar pathology in autism. *Neurology*, 58, 428–432.
- Casanova, M. F., Buxhoeveden, D., Switala, A., & Roy, E. (2002b). Neuronal density and architecture (gray level index) in the brains of autistic patients. *Journal of Child Neurology*, 17, 515–521.
- Casanova, M. F., Buxhoeveden, D. P., & Brown, C. (2002c). Clinical and macroscopic correlates of minicolumnar pathology in autism. *Journal of Child Neurology*, 17, 692–695.
- Casanova, M. F., Buxhoeveden, D., & Gomez, J. (2003). Disruption in the inhibitory architecture of the cell minicolumn: Implications for autism. *The Neuroscientist*, 9, 496–507.
- Casanova, M. F., Van Kooten, I. A. J., Switala, A. E., Van Engeland, H., Heinsen, H., Steinbusch, H. W. M., et al. (2006a). Minicolumnar abnormalities in autism. *Acta Neuropathologica*, 112, 287–303.
- Casanova, M. F., Van Kooten, I., Switala, A. E., Van Engeland, H., Heinsen, H., Steinbusch, H. W. M., et al. (2006b). Abnormalities of cortical minicolumnar organization in the prefrontal lobes of autistic patients. *Clinical Neuroscience Research*, 6, 127–133.
- Casanova, M. F., Trippe, J., II, & Switala, A. (2007). A temporal continuity to the vertical organization of the human neocortex. *Cerebral Cortex*, 17, 130–137.
- Casanova, M. F., El-Baz, A. S., Vanbogaert, E., Narahari, P., & Trippe, J. (2009). Minicolumnar width: Comparison between supragranular and infragranular layers. *Journal of Neuroscience Methods*, 184, 19–24.
- Creutzfeldt, O. D. (1995). *Cortex cerebri: Performance, structural and functional organization of the cortex*. New York: Oxford University Press.
- DeFelipe, J. (2005). Reflections on the structure of the cortical minicolumn. In M. F. Casanova (Ed.), *Neocortical modularity and the cell minicolumn* (pp. 57–92). New York: Nova Biomedical.
- Del Río, M. R., & DeFelipe, J. (1997a). Colocalization of parvalbumin and calbindin D-28k in neurons including chandelier cells of the human temporal neocortex. *Journal of Chemical Neuroanatomy*, 12, 165–173.
- Del Río, M. R., & DeFelipe, J. (1997b). Double bouquet cell axons in the human temporal neocortex: relationship to bundles of myelinated axons and colocalization of calretinin and calbindin D-28k immunoreactivities. *Journal of Chemical Neuroanatomy*, 13, 243–251.
- Douglas, R. J., & Martin, K. A. C. (2004). Neuronal circuits of the neocortex. *Annual Review of Neuroscience*, 27, 419–451.
- Eggan, S. M., & Lewis, D. A. (2007). Immunocytochemical distribution of the cannabinoid CB₁ receptor in the primate neocortex: A regional and laminar analysis. *Cerebral Cortex*, 17, 175–191.
- Freund, T. F. (2003). Interneuron diversity series: Rhythm and mood in perisomatic inhibition. *Trends in Neurosciences*, 26, 489–495.
- Freund, T. F., Martin, K. A. C., Somogyi, P., & Whitteridge, D. (1985). Innervation of cat visual areas 17 and 18 by physiologically identified X- and Y- type thalamic afferents, II. Identification of postsynaptic targets by Gaba immunocytochemistry and Golgi impregnation. *The Journal of Comparative Neurology*, 242, 275–291.
- Gannon, P. J., Holloway, R. L., Broadfield, D. C., & Braun, A. R. (1998). Asymmetry of chimpanzee planum

- temporale: Humanlike pattern of Wernicke's brain language area homolog. *Science*, 279, 220–222.
- Gressens, P., & Evrard, P. (1993). The glial fascicle: An ontogenic and phylogenetic unit guiding, supplying and distributing mammalian cortical neurons. *Developmental Brain Research*, 76, 272–277.
- Groszer, M., Erickson, R., Scripture-Adams, D. D., Lesche, R., Trumpp, A., Zack, J. A., et al. (2001). Negative regulation of neural stem/progenitor cell proliferation by the *Pten* tumor suppressor gene in vivo. *Science*, 294, 2186–2189.
- Guy, J., Hendrich, B., Holmes, M., Martin, J. E., & Bird, A. (2001). A mouse *Mecp2*-null mutation causes neurological symptoms that mimic Rett syndrome. *Nature Genetics*, 27, 322–326.
- Hájos, N., Papp, E. C., Ácsády, L., Levey, A. I., & Freund, T. F. (1998). Distinct interneuron types express m2 muscarinic receptor immunoreactivity on their dendrites or axon terminals in the hippocampus. *Neuroscience*, 82, 355–376.
- Herbert, M. R., Ziegler, D. A., Makris, N., Filipek, P. A., Kemper, T. L., Normandin, J. J., et al. (2004). Localization of white matter volume increase in autism and developmental language disorder. *Annals of Neurology*, 55, 530–540.
- Hopkins, W. D., Marino, L., Rilling, J. K., & MacGregor, L. A. (1998). Planum temporale asymmetries in great apes as revealed by magnetic resonance imaging (MRI). *Neuroreport*, 9, 2913–2918.
- Jacquemont, M. L., Sanlaville, D., Redon, R., Raoul, O., Cormier-Daire, V., Lyonnet, S., et al. (2006). Array-based comparative genomic hybridisation identifies high frequency of cryptic chromosomal rearrangements in patients with syndromic autism spectrum disorders. *Journal of Medical Genetics*, 43, 843–849.
- Jones, A. C., Shyamsundar, M. M., Thomas, M. W., Maynard, J., Idziaszyk, S., Tomkins, S., et al. (1999). Comprehensive mutation analysis of *TSC1* and *TSC2*-and phenotypic correlations in 150 families with tuberous sclerosis. *American Journal of Human Genetics*, 64, 1305–1315.
- Just, M. A., Cherkassky, V. L., Keller, T. A., & Minshew, N. J. (2004). Cortical activation and synchronization during sentence comprehension in high-functioning autism: evidence of underconnectivity. *Brain*, 127, 1811–1821.
- Kawaguchi, Y. (1997). Selective cholinergic modulation of cortical GABAergic subtypes. *Journal of Neurophysiology*, 78, 1743–1747.
- Kim, D. H., Sarbassov, D. D., Ali, S. M., King, J. E., Latek, R. R., Erdjument-Bromage, H., et al. (2002). mTOR interacts with raptor to form a nutrient-sensitive complex that signals to the cell growth machinery. *Cell*, 110, 163–175.
- Kolkova, K., Novitskaya, V., Pedersen, N., Berezin, V., & Bock, E. (2000). Neural cell adhesion molecule-stimulated neurite outgrowth depends on activation of protein kinase C and the Ras-mitogen-activated protein kinase pathway. *Journal of Neuroscience*, 20, 2238–2246.
- Korada, S., Zheng, W., Basilico, C., Schwartz, M. L., & Vaccarino, F. M. (2002). Fibroblast growth factor 2 is necessary for the growth of glutamate projection neurons in the anterior neocortex. *Journal of Neuroscience*, 22, 863–875.
- Krmpotic-Nemanic, J., Kostovic, I., & Nemanic, D. (1984). Prenatal and perinatal development of radial cell columns in the human auditory cortex. *Acta Oto-Laryngologica*, 97, 489–495.
- Kwon, C. H., Luikart, B. W., Powell, C. M., Zhou, J., Matheny, S. A., Zhang, W., et al. (2006). *Pten* regulates neuronal arborization and social interaction in mice. *Neuron*, 50, 377–388.
- Levine, A. J. (1997). p53, the cellular gatekeeper for growth and division. *Cell*, 88, 323–331.
- Levitt, P., & Campbell, D. B. (2009). The genetic and neurobiologic compass points toward common signaling dysfunctions in autism spectrum disorders. *Journal of Clinical Investigation*, 119, 747–754.
- Li, J., Yen, C., Liaw, D., Podsypanina, K., Bose, S., Wang, S. I., et al. (1997). *PTEN*, a putative protein tyrosine phosphatase gene mutated in human brain, breast, and prostate cancer. *Science*, 275, 1943–1947.
- Lorente de Nó, R. (1938). The cerebral cortex: Architecture, intracortical connections, and motor projections. In J. F. Fulton (Ed.), *Physiology of the nervous system* (pp. 291–339). London: Oxford University Press.
- Lübke, J., Egger, V., Sakmann, B., & Feldmeyer, D. (2000). Columnar organization of dendrites and axons of single and synaptically coupled excitatory spiny neurons in layer 4 of the rat barrel cortex. *Journal of Neuroscience*, 20, 5300–5311.
- Lübke, J., Roth, A., Feldmeyer, D., & Sakmann, B. (2003). Morphometric analysis of the columnar innervation domain of neurons connecting layer 4 and layer 2/3 of juvenile rat barrel cortex. *Cerebral Cortex*, 13, 1051–1063.
- Marin-Padilla, M. (2011). *The human brain: Prenatal development and structure*. New York: Springer.
- McBride, K. L., Varga, E. A., Pastore, M. T., Prior, T. W., Manickam, K., Atkin, J. F., et al. (2010). Confirmation of study of *PTEN* mutations among individuals with autism or developmental delays/mental retardation and macrocephaly. *Autism Research*, 3, 137–141.
- Mountcastle, V. B. (1998). *Perceptual neuroscience: The cerebral cortex*. Cambridge, MA: Harvard University Press.
- Nan, X., Ng, H. H., Johnson, C. A., Laherty, C. D., Turner, B. M., Eisenman, R. N., et al. (1998). Transcriptional repression by the methyl-CpG-binding protein MeCP2 involves a histone deacetylase complex. *Nature*, 393, 386–389.
- Parnavelas, J. G., Sullivan, K., Lieberman, A. R., & Webster, K. E. (1977). Neurons and their synaptic organization in the visual cortex of the rat: Electron microscopy of Golgi preparations. *Cell and Tissue Research*, 183, 499–517.

- Peters, A., & Sethares, C. (1991). Organization of pyramidal neurons in area 17 of monkey visual cortex. *The Journal of Comparative Neurology*, 306, 1–23.
- Peters, A., & Sethares, C. (1996). Myelinated axons and the pyramidal cell modules in monkey primary visual cortex. *The Journal of Comparative Neurology*, 365, 232–255.
- Pinto, D., Pagnamenta, A. T., Klei, L., Anney, R., Merico, D., Regan, R., et al. (2010). Functional impact of global rare copy number variation in autism spectrum disorders. *Nature*, 466, 368–372.
- Raballo, R., Rhee, J., Lyn-Cook, R., Leckman, J. F., Schwartz, M. L., & Vaccarino, F. M. (2000). Basic fibroblast growth factor (*Fgf2*) is necessary for cell proliferation and neurogenesis in the developing cerebral cortex. *Journal of Neuroscience*, 20, 5012–5023.
- Rakic, P. (1995). Radial glial cells: Scaffolding for brain construction. In H. Kettenmann & B. R. Ransom (Eds.), *Neuroglia* (pp. 746–762). New York: Oxford University Press.
- Rakic, P. (2003). Developmental and evolutionary adaptation of cortical radial glia. *Cerebral Cortex*, 13, 541–549.
- Samaco, R. C., Nagarajan, R. P., Braunschweig, D., & LaSalle, J. M. (2004). Multiple pathways regulate MeCP2 expression in normal brain development and exhibit defects in autism-spectrum disorders. *Human Molecular Genetics*, 13, 629–639.
- Schlaug, G., Schleicher, A., & Zilles, K. (1995). Quantitative analysis of the columnar arrangement of neurons in the human cingulate cortex. *The Journal of Comparative Neurology*, 351, 441–452.
- Sebat, J., Lakshmi, B., Malhotra, D., Troge, J., Lese-Martin, C., Walsh, T., et al. (2007). Strong association of de novo copy number mutations with autism. *Science*, 316, 445–449.
- Seldon, H. L. (1981a). Structure of human auditory cortex, I. Cytoarchitectonics and dendritic distributions. *Brain Research*, 229, 277–294.
- Seldon, H. L. (1981b). Structure of human auditory cortex, II. Axon distributions and morphological correlates of speech perception. *Brain Research*, 229, 295–310.
- Skene, P. J., Illingworth, R. S., Webb, S., Kerr, A. R., James, K. D., Turner, D. J., et al. (2010). Neuronal MeCP2 is expressed at near histone-octamer levels and globally alters the chromatin state. *Molecular Cell*, 37, 457–468.
- Sokhadze, E., Baruth, J., El-Baz, A., Ramaswamy, R., Sears, L., & Casanova, M. (2009a). Transcranial magnetic stimulation study of gamma induction in response to illusory figures in patients with autism spectrum disorders. *Journal of Neurotherapy*, 13, 271–272.
- Sokhadze, E., Baruth, J., Tasman, A., Sears, L., Mathai, G., El-Baz, A., et al. (2009b). Event-related potential study of novelty processing abnormalities in autism. *Applied Psychophysiology and Biofeedback*, 34, 37–51.
- Sokhadze, E., El-Baz, A., Singh, S., Mathai, G., Sears, L., & Casanova, M. F. (2009c). Effects of low frequency transcranial magnetic stimulation (rTMS) on gamma frequency oscillations and event-related potentials during processing of illusory figures in autism. *Journal of Autism and Developmental Disorders*, 39, 619–634.
- Staiger, J. F., Zilles, K., & Freund, T. F. (1996). Distribution of GABAergic elements postsynaptic to ventroposteromedial thalamic projections in layer IV of rat barrel cortex. *European Journal of Neuroscience*, 8, 2273–2285.
- Staiger, J. F., Freund, T. F., & Zilles, K. (1997). Interneurons immunoreactive for vasoactive intestinal polypeptide (VIP) are extensively innervated by parvalbumin-containing boutons in rat primary somatosensory cortex. *European Journal of Neuroscience*, 9, 2259–2268.
- Steelman, L. S., Abrams, S. L., Whelan, J., Bertrand, F. E., Ludwig, D. E., Bäsecke, J., et al. (2008). Contributions of the Raf/MEK/ERK, PI3K/PTEN/Akt/mTOR and Jak/STAT pathways to leukemia. *Leukemia*, 22, 686–707.
- Szentágothai, J. (1978). The Ferrier Lecture, 1977: The neuron network of the cerebral cortex: A functional interpretation. *Proceedings of the Royal Society B: Biological Sciences*, 201, 219–248.
- Trappe, R., Laccone, F., Cobilanschi, J., Meins, M., Huppke, F., Hanefeld, F., et al. (2001). *MECP2* mutations in sporadic cases of Rett syndrome are almost exclusively of paternal origin. *American Journal of Human Genetics*, 68, 1093–1101.
- Tucker, E. S., Segall, S., Gopalakrishna, D., Wu, Y., Vernon, M., Polleux, F., et al. (2008). Molecular specification and patterning of progenitor cells in the lateral and medial ganglionic eminences. *Journal of Neuroscience*, 28, 9504–9518.
- Vaccarino, F. M., Schwartz, M. L., Raballo, R., Nilsen, J., Rhee, J., Zhou, M., et al. (1999). Changes in cerebral cortex size are governed by fibroblast growth factor during embryogenesis. *Nature Neuroscience*, 2, 246–253.
- Von Economo, C. F., & Koskinas, G. N. (1925). *Die cytoarchitektonik der Hirnrinde des erwachsenen menschen*. Wien: Springer.
- Wegiel, J., Kuchna, I., Nowicki, K., Imaki, H., Wegiel, J., Marchi, E., et al. (2010). The neuropathology of autism: Defects of neurogenesis and neuronal migration, and dysplastic changes. *Acta Neuropathologica*, 119, 755–770.
- Williams, E. L., & Casanova, M. F. (2011a). Autism or autisms? Finding the lowest common denominator. *Boletín de la Asociación Médica de Puerto Rico*, 102(4), 17–24.
- Williams, E. L., & Casanova, M. F. (2011b). Above genetics: Lessons from cerebral development in autism. *Translational Neuroscience*, 2(2), 106–120.
- Wiznitzer, M. (2004). Autism and tuberous sclerosis. *Journal of Child Neurology*, 19, 675–679.
- Yoshimura, Y., & Callaway, E. M. (2005). Fine-scale specificity of cortical networks depends on inhibitory cell type and connectivity. *Nature Neuroscience*, 8, 1552–1559.

- Yoshimura, Y., Dantzer, J. L. M., & Callaway, E. M. (2005). Excitatory cortical neurons form fine-scale functional networks. *Nature*, 433, 868–873.
- Zaitsev, A. V., Povysheva, N. V., Gonzalez-Burgos, G., Rotaru, D., Fish, K. N., Krimer, L. S., et al. (2009). Interneuron diversity in layers 2–3 of the monkey prefrontal cortex. *Cerebral Cortex*, 19, 1597–1615.

Neural Mechanisms of Emotional Dysregulation

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Synonyms

[Amygdala-prefrontal network](#); [Cognitive control circuitry](#)

Definition

Circuitry Implicated in the Cognitive Control of Emotion

Emotion regulation refers broadly to the implementation of automatic or intentional strategies to modulate the trajectory of an emotion or emotional state in order to promote adaptive, goal-directed behavior. Further, the use of functional MRI (fMRI) has provided insight into the neural systems of cognitive control that support regulatory strategies and the modulation of emotional reactivity. Examples of cognitive-based strategies that are amenable to functional imaging paradigms of cognitive control include attentional deployment, which involves controlling the gating of emotional stimuli (e.g., selective attention, distraction); response modulation, which involves strategies to modulate expression of negative emotions (e.g., suppression); and cognitive change, which involves altering the interpretation or meaning of a stimulus (e.g., reappraisal).

Several cognitive processes and neural systems are involved in regulating emotions. In particular, prefrontal regulatory regions including the ventromedial and ventrolateral prefrontal cortices (PFC), ventral anterior cingulate cortex, dorsolateral prefrontal, and parietal regions are primarily involved in modulating the emotional reactivity of regions such as the amygdala, insula, ventral striatum, and periaqueductal gray (Dolcos and McCarthy 2006; Etkin et al. 2015). For instance, the ventromedial PFC, a key region implicated in emotion regulation, downregulates amygdala reactivity in response to threat and other negative stimuli (Ochsner et al. 2012; Silvers et al. 2016). Thus, intact emotion regulation (e.g., cognitive reappraisal) can be characterized by the activation of regions involved in emotional reactivity in response to aversive or negative stimuli, such as threat and fear, and this response is then dampened by ventral prefrontal regions involved in the cognitive control of emotion (Ochsner et al. 2012).

Neural Bases of Emotion Dysregulation in ASD

Deficits in emotion regulation are postulated as a mechanism underlying several psychiatric disorders including internalizing disorders (Jazaieri et al. 2015), disruptive behavior disorders, and autism spectrum disorder (ASD) (Mazefsky et al. 2014). On a neural-systems level, models of emotion dysregulation implicate perturbations in functional connectivity, a measure of the synchronization in activity in between two brain regions, between the amygdala and ventral prefrontal cortex (vPFC) as a possible shared underlying mechanism across several psychiatric disorders characterized by impairments in cognitive control (Etkin et al. 2015; Ochsner et al. 2012). For instance, hyperactive circuitry of emotional reactivity, such as the amygdala, paired with reduced vPFC response to emotional stimuli is associated with anxiety disorders (Goldin et al. 2013), disruptive behavior disorders (Coccaro et al. 2011), and ASD particularly in the presence of co-occurring disorders including anxiety (Herrington et al. 2017) and disruptive behaviors (Ibrahim et al. 2019).

Emotion dysregulation in ASD may be characterized by reduced functional connectivity

between the amygdala and vPFC during emotional perception and cognitive control tasks. For instance, decreased connectivity between the amygdala and ventrolateral PFC was shown during cognitive reappraisal in response to disgust-inducing images in children with ASD (Pitskel et al. 2014). Similarly, reduced prefrontal response during cognitive reappraisal was also shown in adults with ASD, suggesting a shared mechanism of emotion dysregulation among children as well as adults with ASD (Richey et al. 2015). Recent work also implicates differential patterns of reduced connectivity between the amygdala and ventrolateral PFC in children with ASD and co-occurring disruptive behavior in comparison to children with ASD without disruptive behavior (Ibrahim et al. 2019). Similar to findings from fMRI studies in non-ASD youths with conduct problems (Lozier et al. 2014), distinct relationships between callous-unemotional traits and severity of disruptive behaviors with amygdala reactivity have been shown in children with ASD. In particular, greater levels of callous-unemotional traits were found to be related to reduced amygdala reactivity, while the opposite pattern of greater levels of disruptive behavior was found to be related to increased amygdala reactivity to emotional faces (e.g., fearful) in children with ASD when controlling for the variance of the other variable (Ibrahim et al. 2019). Thus, it is possible that emotion dysregulation in ASD, particularly in the presence of co-occurring disorders, could be associated with connectivity patterns that are shared across children without ASD neural systems.

Given that face processing impairments are common in ASD, it is also important to note that perturbed responses in cognitive control circuitry, particularly the amygdala and vPFC circuit, have been shown during social perception including emotion processing tasks such as viewing emotionally expressive faces (Davies et al. 2011; Swartz et al. 2013) and social cognitive tasks such as viewing biological motion (Di Martino et al. 2009). Therefore, it is possible that aberrant recruitment of emotional reactivity and control circuitry during social perception could indicate misinterpretation or reduced

saliency of social cues in individuals with ASD. Conversely, amygdala hyper-reactivity as well as amygdala-vPFC hyper-connectivity was also shown in individuals with ASD during face perception tasks (Kleinhans et al. 2011; Monk et al. 2010; Tottenham et al. 2013), which could indicate heightened sensitivity to threat or lack of differentiation of threat versus safe social cues (Top et al. 2016).

In terms of future work, it is not clear which neural components underlying emotion regulation are most affected in ASD in the presence of a co-occurring disorders when different cognitive control processes are engaged (e.g., reappraisal vs. suppression vs. selective attention). Likewise, more work is needed to elucidate whether emotion regulatory impairments in ASD are due to a failure to activate specific cognitive control regions or abnormalities in the synchronization or connectivity between large-scale networks. Few studies have also examined the neural response to multiple sensory stimuli in ASD based on an emotion regulation framework (Green et al. 2015). Given that sensory over-responsivity including severe negative responses to stimuli (e.g., sounds, noisy environments, textures) is prevalent among youths with ASD, future studies are needed to understand possible dysfunction in emotion regulation circuitry during exposure to sensory stimuli. Further, the use of a dimensional approach to model cognitive control along a continuum in ASD, as well as in comparison to other clinical comparison groups, could reveal new insights into the relationship between emotion regulation circuitry and severity of associated symptoms, supporting the identification of possible neuroendophenotypes that could serve as mechanism-based targets for personalized treatments in ASD.

See Also

- [Amygdala](#)
- [Emotional Regulation](#)
- [Prefrontal Cortex](#)
- [RDoC and Autism](#)

References

- Coccaro, E. F., Sripada, C. S., Yanowitch, R. N., & Phan, K. L. (2011). Corticolimbic function in impulsive aggressive behavior. *Biological Psychiatry*, 69(12), 1153–1159.
- Davies, M. S., Dapretto, M., Sigman, M., Sepeta, L., & Bookheimer, S. Y. (2011). Neural bases of gaze and emotion processing in children with autism spectrum disorders. *Brain and Behavior: A Cognitive Neuroscience Perspective*, 1(1), 1–11. <https://doi.org/10.1002/brb3.6>.
- Di Martino, A., Ross, K., Uddin, L. Q., Sklar, A. B., Castellanos, F. X., & Milham, M. P. (2009). Functional brain correlates of social and nonsocial processes in autism spectrum disorders: An activation likelihood estimation meta-analysis. *Biological Psychiatry*, 65(1), 63–74. <https://doi.org/10.1016/j.biopsych.2008.09.022>.
- Dolcos, F., & McCarthy, G. (2006). Brain systems mediating cognitive interference by emotional distraction. *Journal of Neuroscience*, 26(7), 2072–2079.
- Etkin, A., Büchel, C., & Gross, J. J. (2015). The neural bases of emotion regulation. *Nature Reviews Neuroscience*, 16(11), 693.
- Goldin, P. R., Ziv, M., Jazaieri, H., Hahn, K., Heimberg, R., & Gross, J. J. (2013). Impact of cognitive behavioral therapy for social anxiety disorder on the neural dynamics of cognitive reappraisal of negative self-beliefs: Randomized clinical trial. *JAMA Psychiatry*, 70(10), 1048–1056.
- Green, S. A., Hernandez, L., Tottenham, N., Krasileva, K., Bookheimer, S. Y., & Dapretto, M. (2015). Neurobiology of sensory overresponsivity in youth with autism spectrum disorders. *JAMA Psychiatry*, 72(8), 778–786.
- Herrington, J. D., Maddox, B. B., McVey, A. J., Franklin, M. E., Yerys, B. E., Miller, J. S., & Schultz, R. T. (2017). Negative valence in autism spectrum disorder: The relationship between amygdala activity, selective attention, and co-occurring anxiety. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 2(6), 510–517.
- Ibrahim, K., Eilbott, J., Ventola, P., He, G., Pelphrey, K. A., McCarthy, G., & Sukhodolsky, D. G. (2019). Reduced amygdala-prefrontal functional connectivity in children with autism spectrum disorder and co-occurring disruptive behavior. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 4(12), 1031–1041.
- Jazaieri, H., Morrison, A. S., Goldin, P. R., & Gross, J. J. (2015). The role of emotion and emotion regulation in social anxiety disorder. *Current Psychiatry Reports*, 17(1), 531. <https://doi.org/10.1007/s11920-014-0531-3>.
- Kleinmans, N. M., Richards, T., Johnson, L. C., Weaver, K. E., Greenson, J., Dawson, G., & Aylward, E. (2011). fMRI evidence of neural abnormalities in the subcortical face processing system in ASD. *NeuroImage*, 54(1), 697–704.
- Lozier, L. M., Cardinale, E. M., VanMeter, J. W., & Marsh, A. A. (2014). Mediation of the relationship between callous-unemotional traits and proactive aggression by amygdala response to fear among children with conduct problems. *JAMA Psychiatry*, 71(6), 627–636. <https://doi.org/10.1001/jamapsychiatry.2013.4540>.
- Mazefsky, C. A., Borue, X., Day, T. N., & Minshew, N. J. (2014). Emotion regulation patterns in adolescents with high-functioning autism spectrum disorder: Comparison to typically developing adolescents and association with psychiatric symptoms. *Autism Research*, 7(3), 344–354. <https://doi.org/10.1002/aur.1366>.
- Monk, C. S., Weng, S.-J., Wiggins, J. L., Kurapati, N., Louro, H. M., Carrasco, M., . . . Lord, C. (2010). Neural circuitry of emotional face processing in autism spectrum disorders. *Journal of Psychiatry & Neuroscience*, 35(2), 105.
- Ochsner, K. N., Silvers, J. A., & Buhle, J. T. (2012). Functional imaging studies of emotion regulation: A synthetic review and evolving model of the cognitive control of emotion. *Annals of the New York Academy of Sciences*, 1251, E1–E24. <https://doi.org/10.1111/j.1749-6632.2012.06751.x>.
- Pitskel, N. B., Bolling, D. Z., Kaiser, M. D., Pelphrey, K. A., & Crowley, M. J. (2014). Neural systems for cognitive reappraisal in children and adolescents with autism spectrum disorder. *Developmental Cognitive Neuroscience*, 10, 117–128.
- Richey, J. A., Damiano, C. R., Sabatino, A., Rittenberg, A., Petty, C., Bizzell, J., . . . Dichter, G. S. (2015). Neural mechanisms of emotion regulation in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45, 3409. <https://doi.org/10.1007/s10803-015-2359-z>.
- Silvers, J. A., Insel, C., Powers, A., Franz, P., Helion, C., Martin, R. E., . . . Ochsner, K. N. (2016). vPFC–vmPFC–amygdala interactions underlie age-related differences in cognitive regulation of emotion. *Cerebral Cortex*, 27(7), 3502–3514.
- Swartz, J. R., Wiggins, J. L., Carrasco, M., Lord, C., & Monk, C. S. (2013). Amygdala habituation and prefrontal functional connectivity in youth with autism spectrum disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(1), 84–93.
- Top, D., Stephenson, K. G., Doxey, C. R., Crowley, M. J., Kirwan, C. B., & South, M. (2016). Atypical amygdala response to fear conditioning in autism spectrum disorder. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 1(4), 308–315.
- Tottenham, N., Hertzog, M. E., Gillespie-Lynch, K., Gilhooly, T., Millner, A. J., & Casey, B. (2013). Elevated amygdala response to faces and gaze aversion in autism spectrum disorder. *Social Cognitive and Affective Neuroscience*, 9(1), 106–117.

Neural Reorganization

► Plasticity, Neural

Neural Science

► Neuroscience

Neural Signatures of Treatment Response

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Definition

As a field, clinical research is increasingly moving toward the goal of precision medicine (Insel 2014), an initiative that highlights the importance of taking into consideration individual differences from the genetic to behavioral level when formulating effective treatment plans. Recent advancements have increasingly adopted an interdisciplinary approach to overcome two major barriers: (1) to better characterize *how* a treatment can elicit change at the *individual* level, and (2) to predict *who* will most likely respond to the intervention. Successful integration of neuroimaging techniques and behavioral measures have the potential to guide identification of sensitive and objective biomarkers that can reflect and predict response to specific evidence-based treatment models in individuals with autism spectrum disorder (ASD). By integrating knowledge from social and cognitive neuroscience that have shown atypical neural response among individuals with ASD when processing social stimuli compared to typically developing (TD) individuals (Adolphs 2009), as well as established neural signatures of ASD (Kaiser et al. 2010), neuroimaging techniques can facilitate the progression toward precision medicine in two ways.

First, clinical research relying on behavioral measures alone are not sufficient for overcoming the challenge of equifinality, where similar behavioral gains observed across participants following treatment might mask differential underlying mechanisms of change. Integrating neuroimaging techniques such as functional magnetic resonance imaging (fMRI) can help characterize the heterogeneous neural mechanisms of change underlying similar behavioral gains observed across participants. fMRI examines functional changes in brain response to social stimuli that are both common at the group pathology level of ASD, and unique changes at the individual level (Venkataraman et al. 2016; Ventola et al. 2015). Therefore, the utilization of neuroimaging tools can help clinicians to better understand not only *how* a treatment can elicit change, but also how change is occurring at the *individual* level, which can inform future treatment decisions. Second, tracking clinically sensitive biomarkers can enable clinicians to both objectively quantify and qualitatively assess trajectories of change over the course of treatment (Ventola et al. 2015; Voos et al. 2012), and data collected over time can help generate predictive models of responder profiles to treatment (Yang et al. 2016), assisting the understanding of *who* will most likely respond to intervention.

One evidence-based intervention that has seen a recent expansion in identifying neural signatures of treatment response is pivotal response treatment (PRT), a behavioral-based intervention primarily aimed to target social communication skills in children with ASD (Koegel et al. 1987; Koegel and Egel 1979; Koegel and Koegel 2012; Verschuur et al. 2013). Based on principles of applied behavior analysis (ABA), PRT adopts a more naturalistic approach and employs a variety of strategies to increase children's motivation, such as following the child's choice, interspersing maintenance and acquisition tasks, and reinforcing both correct responses and appropriate attempts to increase the frequency of children's exposure to naturally contingent reinforcements (Koegel et al. 1987; Koegel and Egel 1979). Building upon its large empirical evidence base from behavioral studies (Verschuur et al.

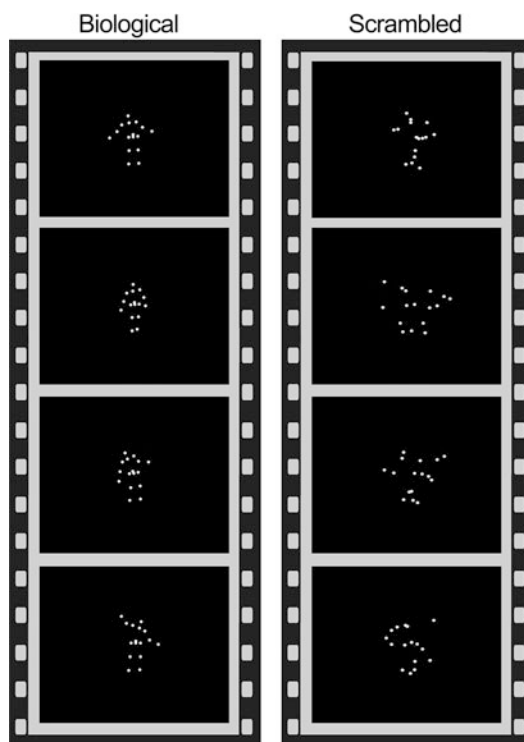
2013), recent advancements have begun to integrate neuroimaging techniques to identify both neural signatures of treatment response (Ventola et al. 2015; Voos et al. 2012), as well as neural biomarkers to help predict responder profiles to PRT (Yang et al. 2016).

This entry will first outline the current state of science behind the identification of brain areas of interest to serve as potential neural signatures of treatment response in individuals with ASD, as well as the evidence supporting the use of functional paradigms sensitive to detect clinically meaningful outcomes in terms of neural response to social stimuli. Next, a critical evaluation of recent advancements in identifying neural signatures of treatment response to PRT for individuals with ASD is offered. Finally, future directions for expanding research to help uncover neural signatures of treatment response can increase the use of other noninvasive and more cost-effective techniques such as electroencephalography (EEG), as well as eye-tracking are discussed, alongside limitations to take into consideration for future research.

Background: Examining the Current State of Science

Overview of Neural Signatures of ASD

Recent advancements in the identification of neural signatures of treatment response for individuals with ASD have relied on the assimilation of knowledge from social and cognitive neuroscience research, to first establish a robust profile of neural correlates that can reliably detect differences in social cognition among individuals with ASD compared to their TD peers. One well-established paradigm known to reliably elicit differential patterns of neural response in brain regions associated with social cognition among individuals with and without ASD is a biological motion paradigm (Klin et al. 2009; Pavlova 2012; Pelphrey et al. 2003), which depicts a series of either biologically coherent (BIO) or scrambled (SCR) motion by using a series of point-light displays (Fig. Fig. 1). For the biologically coherent displays, the point-light displays depict social



Neural Signatures of Treatment Response, Fig. 1 Stimuli showing biological and scrambled motion paradigm

movements such as a person dancing, walking, or playing games that are familiar to children such as pat-a-cake (Pelphrey et al. 2003). The ability to perceive form from biological motion capitalizes on the intrinsic visual sensitivity toward kinematics that is congruent with the spatiotemporal dynamics of movements elicited by biological agents (Johansson 1973; Pavlova 2012; Puce and Perrett 2003).

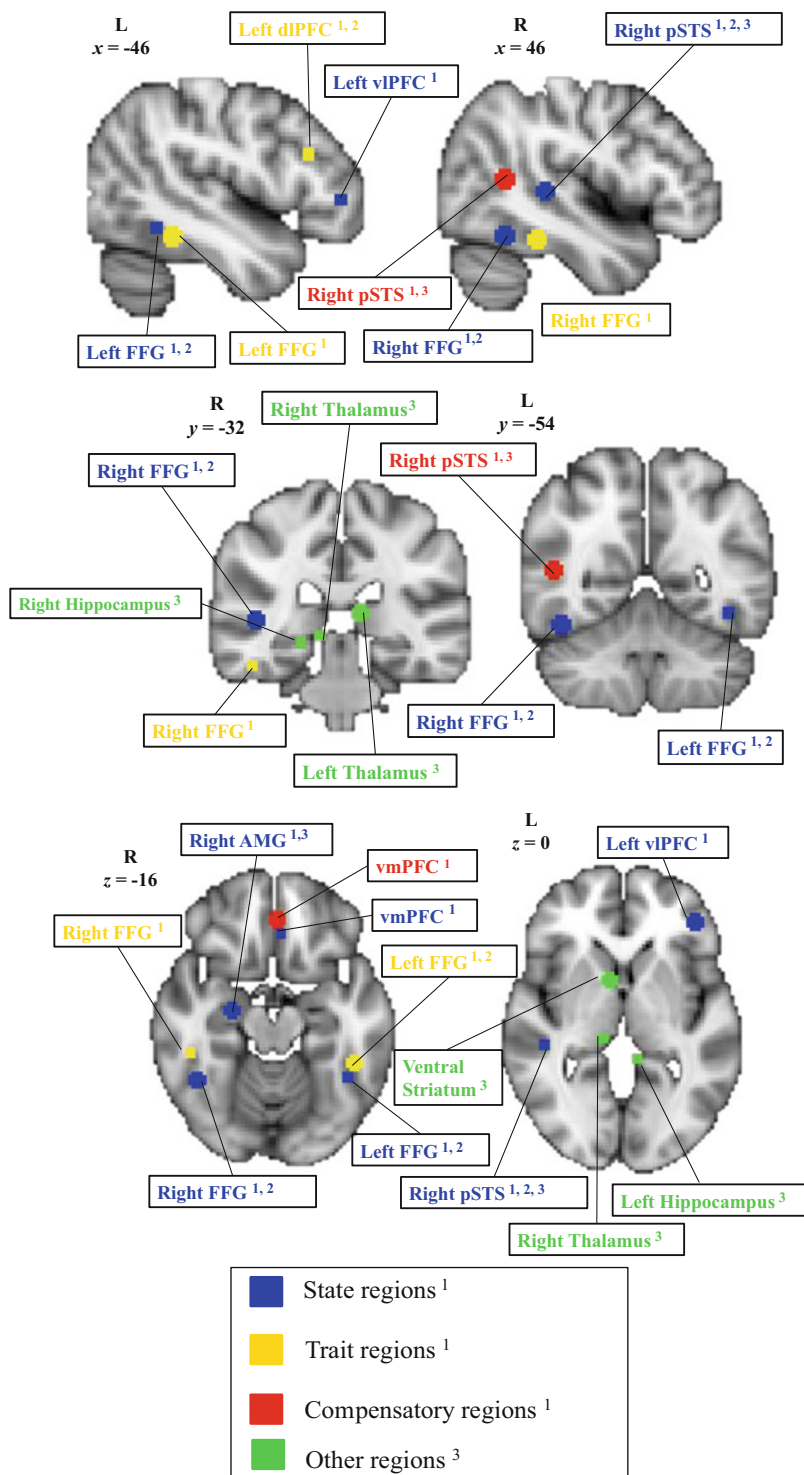
Biological motion paradigms have been widely used in both primate and human neuroimaging research to study social cognition, and have generated convergent findings that highlight the role of superior temporal sulcus (STS) in the recognition of socially salient information (Oram and Perrett 1994, 1996; Puce and Perrett 2003; Vaina et al. 2001). Primate findings have shown that sensory integration occurs when motion-related information from the dorsal pathway and form-related information from the ventral pathway derived from the same biological object

converge at the superior temporal polysensory area (STP; found within the STS) within 100 ms, enabling perception of coherent biological motion (Oram and Perrett 1994, 1996). In humans, similar response profiles are seen in typically developing adults, where biological motion have been shown to activate both the ventral and dorsal visual pathways as well as the point of confluence at the superior temporal gyrus (STG), the homologue region to the STP found in primates (Pelphrey et al. 2003; Vaina et al. 2001), as well as the fusiform gyrus (FG) known for facial recognition. In TDs, preferential looking toward biologically coherent motion emerges during early infancy, and serves to bias one's attention toward socially salient stimuli (Klin et al. 2009), which is an important behavioral precursor to later-emerging social cognition and affect regulation skills (Baron-Cohen 1997; Frith and Frith 1999). This is further supported by neurobiological findings, where STS projects to both the amygdala and orbitofrontal cortex (OFC) which are involved in processing emotionally salient information and affect regulation, and together form part of the social cognition network associated with theory of mind (Adolphs 2009; Baron-Cohen 1997; Brothers 2001). Therefore, biological motion is a well-validated paradigm that is known to elicit robust response profiles in brain areas involved in social information processing, and atypical profiles of neural response to BIO versus SCR motion may be indicative of pathology that impacts social cognitive processing (Kaiser et al. 2010; Ventola et al. 2015; Voos et al. 2012).

To better characterize the distinct neural profile associated with ASD that might underlie differences in social cognition when compared to TD peers, Kaiser et al. (2010) compared and contrasted the neural response to BIO versus SCR motion in children with ASD ($n = 25$) with their unaffected siblings (US; $n = 20$) and TD children ($n = 17$). The authors conducted whole-brain analysis at voxel-wise uncorrected threshold level of $p < 0.05$, to evaluate brain regions that showed increased activation during BIO versus SCR motion (BIO>SCR) in TD>SD, TD>US, US>ASD, and US>TD group comparisons. To ensure that the false positive discovery rate of

active voxels is $<5\%$, only clusters that exceeded a cluster threshold of >20 contiguous voxels at $\alpha < 0.05$ within each group level comparison were selected. The further determine α value for each cluster, the relative occurrence of each cluster during 5000 iterations of Monte Carlo simulation was noted. In order to determine distinct neural profiles of (a) "state," (b) "trait," and (c) "compensatory" patterns of activation in response to biological motion, higher-level conjunction analyses were conducted using an uncorrected voxel-wise threshold level of $p < 0.0025$, and a cluster threshold of $k > 20$ to further control for the rate of false positives (Fig. 2).

The "state," "trait," and "compensatory" regions served as unique neural signatures that characterized the differential response to biological motion across ASD, US, and TD children. "State" regions refer to brain areas that showed reduced activation during BIO>SCR in children with ASD only, thus indicative of atypical neural response to social stimuli unique to the state of ASD pathology. Such regions included ventromedial and left ventrolateral prefrontal cortex (vmPFC; vlPFC), right amygdala (rAMG), bilateral fusiform gyrus (FG), and right posterior superior temporal sulcus (rpSTS). "Trait" regions refer to brain areas that showed reduced activation during BIO>SCR in both children with ASD and US relative to TD children, thus indicative of an endophenotype underlying atypical neural response to social stimuli that may be associated with shared genetic vulnerability between ASD and TD. Such regions included right inferior temporal gyrus (ITG), left dorsolateral prefrontal cortex (dlPFC), and anterior bilateral fusiform gyri (FG). "Compensatory" regions refer to differential neural response to BIO>SCR found in US group only, suggesting possible compensatory effects to overcome their shared vulnerability with the ASD group that affects their ability to process social stimuli. Such regions included anterior rostral vmPFC and caudal rpSTS. Taken together, the "state," "trait," and "compensatory" profiles guide the selection of sensitive biomarkers that can detect atypical brain response to social stimuli both specifically related to ASD pathology, and



those that are related to the broader autism phenotype, providing a more comprehensive overview of the range of phenotypic abnormalities associated with disrupted social information processing related to ASD.

Evaluating Sensitivity of Neural Signatures of ASD as Clinical Biomarkers

Building upon the empirical data that have characterized biomarkers indicative of atypical response to social stimuli among children with ASD (Kaiser et al. 2010), such biomarkers may be clinically informative when monitored over the course treatment to help objectively quantify both the quality and magnitude of change in brain responsiveness to social stimuli. Furthermore, the demarcation of whether changes occur in areas that correspond to “state” or “compensatory” neural profiles can inform clinicians if following treatment, children with ASD may show increased normalization of brain response to social stimuli within the BIO>SCR contrast compared to TD children (by increased activation in “state” and “trait” regions), or if there are compensatory effects analogous to that observed in their unaffected siblings (by increased activation in “compensatory” regions), or both.

To assess the clinical sensitivity of the identified neural signatures as potential biomarkers of treatment response, Voos et al. (2012) conducted the first fMRI study (case study) to evaluate whether behavioral changes in increased social functioning following 16 weeks of PRT is accompanied by simultaneous increased brain activation to biological motion in two high-functioning children with ASD. Primarily targeting social communication skills in children with ASD, PRT embeds learning opportunities naturally within games that are intrinsically motivating to each child, and delivers natural reinforcements contingent upon each child’s appropriate social attempts and responses to increase social initiations, use of functional language, and overall social reciprocity (Koegel et al. 1998; Koegel et al. 1987; Koegel and Egel 1979). Individualized treatment goals were established for each participant at the onset of treatment, which included lower-level social skills such as appropriate body positioning and voice modulation for participant one, and higher-

level social skills such as social reciprocity, perspective taking, and use of functional language to initiate and make requests for participant two. Following PRT, clinical assessments by using the Autism Diagnostic Observation Schedule (ADOS) and Clinical Evaluation of Language Fundamentals (CELF-4) showed significant improvements in social functioning and use of adaptive language for both children. In response to biological motion, significant increases in activation were only observed in “state” and “trait” regions, where both children showed gains in bilateral FG. In addition, participant two showed more widespread collateral gains in other “state” areas including rpSTS and vIPFC, reflective of higher-order social processing skills targeted during PRT. The overlapping, yet distinct neural profiles of change across both participants, is important to note, as it helped to partition differential mechanisms of change underlying similar behavioral gains assessed by clinical measures, and helps to overcome the problem of equifinality by noting how treatment response might differ at the *individual* level.

The use of fMRI also helped to elucidate the mechanism underlying *how* PRT may be inducing social gains in children with ASD, as the lack of change in activation of “compensatory” regions highlights that PRT elicited social gains in both children were driven by an increased normalization of neural response to social stimuli that is more comparable to the developmental trajectory of their TD peers. However, there are several limitations in Voos et al. (2012)’s findings. First, it was a case study with two participants so not generalizable to the wider clinical population. Second, no control groups were included, thus making it challenging to conclude whether the changes in brain activation to BIO>SCR were specific to PRT per se, or reflective of broader developmental changes that naturally occurred over the course of 16 weeks for both participants. Nonetheless, the Voos et al. (2012) study is important to note as it showed that the neural signatures of ASD identified by Kaiser et al. (2010) can potentially serve as clinically sensitive biomarkers that can track changes in social functioning at the brain level, within the timeframe of 16 weeks of PRT for children with ASD.

Current Knowledge: Neural Signatures of Pivotal Response Treatment

Overcoming the Challenge of Equifinality

Based on exploratory results from Voos et al. (2012) that demonstrated the clinical sensitivity of neural signatures of ASD in detecting change in social functioning at the brain level in individuals with ASD, it is important to further examine how individual variances in neural profiles presented at baseline might further influence the trajectories of functional changes observed over the course of PRT. Identifying changes in functional neural pathways might indicate possible heterogeneous mechanisms of biological change underlying behavioral gains in social functioning observed in children with ASD, and help to address not only *how* a treatment might be eliciting positive social outcomes, but also how such outcomes are occurring at the *individual* level.

To capture heterogeneity that might underlie similar behavioral manifestations of reduced social responsiveness such as inattention to faces and poor eye contact in children with ASD, Ventola et al. (2015) examined the brain response profile to BIO>SCR in the rpSTS in ten high-functioning children with ASD compared to five TD children. Relative to the mean activation level in rpSTS at the group level to biological motion at baseline found in TD children, five children with ASD demonstrated relatively hyperactive rpSTS activation (ASD>TD), and five children demonstrated relatively hypoactive rpSTS activation (TD>ASD). The authors hypothesized that the hyper- and hypoactive groups at baseline might experience different challenges that disrupted their response to social stimuli at baseline. Specifically, the hyperactive group might experience reduced sensory gating and poor attentional control, resulting in hyperarousal when exposed to socially salient information at baseline (Markram and Markram 2010), and reflected by increased activation of thalamus, amygdala, and temporal cortical regions. In comparison, the hypoactive group might experience reduced social motivation when attending to social stimuli, reflected by reduced activation in reward processing regions such as the ventral striatum (VS), nucleus accumbens (NAcc), and amygdala (Chevallier et al. 2012).

Following 16 weeks of PRT, both hyper- and hypoactive groups showed significant improvements in social functioning assessed by behavioral measures, though distinct patterns of underlying neural mechanisms of change emerged. For the hyperactive group, the authors found reduced activation in rpSTS and other brain areas related to sensory gating and attention control (thalamus, amygdala, and hippocampus) during biological motion, suggesting improvements in regulating sensory gating and arousal levels in response to social stimuli following PRT. For the hypoactive group, the authors found increased activation and functional connectivity between both the rpSTS and the VS, a major region involved in reward processing, as well as other regions of the mirror neuron system (Fig. 2), suggesting improvements in social reward processing. Therefore, Ventola et al. (2015) successfully showed that not only does PRT elicit normalized neural response profiles to socially meaningful stimuli that accompanies behavioral social gains in children with ASD, but that changes occur differentially at the individual level, suggesting each child with ASD may be benefitting from PRT in individualized ways. Unmasking the heterogeneous neural mechanisms of change underlying similar behavioral gains can help clinicians to better identify how different active ingredients of PRT may be elicit therapeutic potentials for each child with ASD, and help to develop more individualized goals matched with appropriate intervention techniques to more effectively address each individual's social deficits, thus moving toward the goal of precision medicine.

Finally, given that functional changes in response to social stimuli can be seen at the regional level following PRT, it is interesting to conjecture whether co-occurring changes in functional connectivity between brain regions involved in lower- and higher-order social information processing may be altered at the brain circuitry level. This is of particular interest given that PRT simultaneously targets many social skills ranging from lower-level skills, such as increasing frequency of social initiations, to higher-level perspective-taking and self-monitoring. Investigating how such co-occurring behavioral social gains may elicit functional

rewiring of the brain in response to social stimuli can further examine whether PRT may be beneficial in increasing the brain's overall efficiency at processing meaningful social information. In a sample of 19 high-functioning school-aged children with ASD, Venkataraman et al. (2016) evaluated co-occurring regions that showed changes in functional connectivity that accompanied aggregated functional change in response to biological motion in the posterior cingulate cortex (PCC) following 16 weeks of PRT. The authors found notable changes in brain circuitry associated with processing and assignment of reward values to external stimuli implicated by reduced functional connectivity to orbitofrontal cortex (OFC), as well as circuitry associated with facial recognition implicated by increased functional connectivity to occipital-temporal cortex. Taken together, PRT is shown to elicit both normalized trajectories of brain response to social stimuli both at the regional and brain circuitry level, suggesting there may be improved neural efficiency in processing socially salient information in children with ASD.

Predictors of Responder Profile

In addition to addressing *how* treatment may elicit change at the individual level, incorporating neuroimaging tools can also help to characterize neural profiles that might *predict*, at baseline, an individual's response to treatment. Identifying neural predictors of treatment response can guide clinicians to select more appropriate treatments for individuals with ASD, and to maximize the cost and time efficacy for eliciting positive therapeutic gains. Furthermore, identifying neural predictors of treatment response and the corresponding behavioral profiles can increase clinicians' awareness of areas that may be more treatment resistant, and require greater care when formulating treatment plans to develop effective strategies targeting such areas without breaching fidelity of treatment protocol. From a neuroimaging perspective, it is possible to conduct a retrospective regression analysis to assess how brain activation in response to social stimuli at baseline might predict the magnitude of improvement in social functioning following treatment, thus identifying possible neural predictors of treatment response.

Monitoring changes in brain response to biological motion, Yang et al. (2016) conducted regression-based multivariate pattern analyses (MVPA) to decode how unique distributions of voxel activation pattern at baseline within neural networks can be associated with the efficiency of social cognitive processing. The authors found that response to BIO>SCR in four neural clusters at baseline subsequently predicted the magnitude of increase in social competency following 16 weeks of PRT in 20 children with ASD, which included areas such as the STS, superior parietal lobule, FG, vmPFC, VS, and putamen. Functionally, the identified brain areas corresponded to a range of social and cognitive functions including motion and object recognition, emotion regulation and response inhibition, visuospatial attention, and reward processing. Furthermore, the authors utilized a cross-validation design and found that the MVPA model of pretreatment response trained using one cohort of patient data was able to successfully predict changes in social functioning in a second independent cohort of patient data, suggesting that neural predictors identified may be generalizable across children with ASD. However, treatment specificity to PRT of the biomarkers identified could not be assessed, as the authors did not include an alternative treatment group for comparison, which warrants further attention. Despite the limitation, the successful implementation of MVPA by integrating fMRI and behavioral data has shown a promising first step for moving toward the goal of identifying *who* will most likely benefit from PRT among children with ASD, a major obstacle en route to establishing precision medicine in psychiatry.

Implications and Future Directions

In summary, research evaluating neural signatures of treatment response has many valuable clinical implications. Characterizing neural signatures that can both objectively quantify and show qualitative differences in trajectories of change over the course of treatment can help clinicians to better monitor progression toward treatment goals for each patient, as well as more flexibly

adapt the dosage, intensity, and treatment targets tailored to meet individuals' needs in a more time-efficient manner. Furthermore, determining responder neural profiles to treatment at baseline is important, especially in children where brain plasticity and windows of opportunity become increasingly sparse over the course of development, further enhancing the difficulty for interventions to effectively elicit therapeutic changes. Expanding clinical research to better characterize neural signatures that predict response to treatment can help guide clinicians when selecting an appropriate intervention type for the individual in question, and therefore serve as an important clinical tool in future pediatric psychiatry.

To further widen the evidence base for neural signatures of treatment response, as well as to increase the dissemination of affordable ways to identify reliable neural signatures of treatment response to the community, future directions and considerations are outlined. Current advancements in identifying neural signatures of PRT have solely relied on the use of fMRI, which provides good spatial resolution to help identify brain regions of interest. However, fMRI is very expensive and lacks good temporal resolution to resolve the sequence of subprocesses underlying any neural response. A practical implication to consider is the relatively low success rate for young children with ASD to complete fMRI scans, even after extensive training and preparation, as they often show elevated levels of head motion that affect the quality of images collected for analyses. Therefore, future research should continue to look to integrate other more cost-effective neuroimaging tools with enhanced temporal resolution and higher success rates for completion, such as EEG and eye-tracking, to gain insight into not only which areas might serve as neural signatures of treatment response, but how these atypical neural activations might sequentially interact to affect overall neural efficiency in the processing of social stimuli.

One limitation of the current published findings is that participants have been high-functioning children with ASD ($IQ > 70$). Future studies should use larger samples with more heterogeneous symptomatology and a wider range of cognitive levels, to examine the generalizability of

results to lower-functioning children with ASD. Another major limitation is the lack of inclusion of either a waitlist control group, or a group that receives an alternative form of intervention. Inclusion of a waitlist control group of children well-matched on ASD symptomatology, age, IQ, and gender is critical to ensure that neural signatures showing changes in response to social stimuli are truly reflective of the therapeutic effects of the target intervention, rather than associated with general effects of development over the passage of time. Future studies should also include stringent control groups to better address the question of *specificity* of neural signatures of treatment response in children with ASD. Furthermore, inclusion of an alternative form of intervention will address the question of *specificity* of the neural signatures identified, to determine whether they are predictors of treatment response specific to PRT, or whether they may be neural signatures more broadly reflective of improved social cognition following interventions that target social skills in children with ASD. All of these future developments will further advance the field of clinical research toward the goal of developing precision medicine in pediatric psychiatry.

See Also

- Behavior Therapy
- Biological Motion
- Event-Related Functional Magnetic Resonance Imaging (MRI)
- Social Cognition
- Social Interventions

References and Reading

- Adolphs, R. (2009). The social brain: Neural basis of social knowledge. *Annual Review of Psychology*, 60, 693–716. <https://doi.org/10.1146/annurev.psych.60.110707.163514>.
- Baron-Cohen, S. (1997). *Mindblindness: An essay on autism and theory of mind*. Cambridge: MIT Press.
- Brothers, L. (2001). *Friday's footprint: How society shapes the human mind*. Oxford, New York: Oxford University Press.
- Chevallier, C., Kohls, G., Troiani, V., Brodtkin, E. S., & Schultz, R. T. (2012). The social motivation theory of

- autism. *Trends in Cognitive Sciences*, 16(4), 231–239. <https://doi.org/10.1016/j.tics.2012.02.007>.
- Frith, C. D., & Frith, U. (1999). Interacting minds – A biological basis. *Science*, 286(5445), 1692–1695.
- Insel, T. R. (2014). The NIMH research domain criteria (RDoC) project: Precision medicine for psychiatry. *American Journal of Psychiatry*, 171(4), 395–397. <https://doi.org/10.1176/appi.ajp.2014.14020138>.
- Johansson, G. (1973). Visual perception of biological motion and a model for its analysis. *Perception & Psychophysics*, 14(2), 201–211. <https://doi.org/10.3758/BF03212378>.
- Kaiser, M. D., Hudac, C. M., Shultz, S., Lee, S. M., Cheung, C., Berken, A. M., et al. (2010). Neural signatures of autism. *Proceedings of the National Academy of Sciences of the United States of America*, 107(49), 21223–21228. <https://doi.org/10.1073/pnas.1010412107>.
- Klin, A., Lin, D. J., Gorrindo, P., Ramsay, G., & Jones, W. (2009). Two-year-olds with autism orient to non-social contingencies rather than biological motion. *Nature*, 459(7244), 257–261. <https://doi.org/10.1038/nature07868>.
- Koegel, L. K., Camarata, S. M., Valdez-Menchaca, M., & Koegel, R. L. (1998). Setting generalization of question-asking by children with autism. *American Journal of Mental Retardation*, 102(4), 346–357.
- Koegel, R. L., Dyer, K., & Bell, L. K. (1987). The influence of child-preferred activities on autistic children's social behavior. *Journal of Applied Behavior Analysis*, 20(3), 243–252. <https://doi.org/10.1901/jaba.1987.20-243>.
- Koegel, R. L., & Egel, A. L. (1979). Motivating autistic children. *Journal of Abnormal Psychology*, 88(4), 418–426.
- Koegel, R. L., & Koegel, L. K. (2012). Treatment of pivotal areas. In R. L. Koegel & R. L. Koegel (Eds.), *The PRT pocket guide: Pivotal response treatment for autism spectrum disorders* (pp. 13–38). Baltimore: Paul H. Brookes Publishing.
- Markram, K., & Markram, H. (2010). The intense world theory – A unifying theory of the neurobiology of autism. *Frontiers in Human Neuroscience*, 4. <https://doi.org/10.3389/fnhum.2010.00224>.
- Oram, M. W., & Perrett, D. I. (1994). Responses of anterior superior temporal polysensory (STPa) neurons to “biological motion” stimuli. *Journal of Cognitive Neuroscience*, 6(2), 99–116. <https://doi.org/10.1162/jocn.1994.6.2.99>.
- Oram, M. W., & Perrett, D. I. (1996). Integration of form and motion in the anterior superior temporal polysensory area (STPa) of the macaque monkey. *Journal of Neurophysiology*, 76(1), 109–129.
- Pavlova, M. A. (2012). Biological motion processing as a hallmark of social cognition. *Cerebral Cortex*, 22(5), 981–995. <https://doi.org/10.1093/cercor/bhr156>.
- Pelphrey, K. A., Mitchell, T. V., McKeown, M. J., Goldstein, J., Allison, T., & McCarthy, G. (2003). Brain activity evoked by the perception of human walking: Controlling for meaningful coherent motion. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 23(17), 6819–6825.
- Puce, A., & Perrett, D. (2003). Electrophysiology and brain imaging of biological motion. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 358(1431), 435–445. <https://doi.org/10.1098/rstb.2002.1221>.
- Vaina, L. M., Solomon, J., Chowdhury, S., Sinha, P., & Belliveau, J. W. (2001). Functional neuroanatomy of biological motion perception in humans. *Proceedings of the National Academy of Sciences*, 98(20), 11656–11661. <https://doi.org/10.1073/pnas.191374198>.
- Venkataraman, A., Yang, D. Y.-J., Dvornek, N., Staib, L. H., Duncan, J. S., Pelphrey, K. A., & Ventola, P. (2016). Pivotal response treatment prompts a functional rewiring of the brain among individuals with autism spectrum disorder. *Neuroreport*, 27(14), 1081–1085. <https://doi.org/10.1097/WNR.0000000000000662>.
- Ventola, P., Yang, D. Y., Friedman, H. E., et al. (2015). Heterogeneity of neural mechanisms of response to pivotal response treatment. *Brain Imaging and Behavior*, 9(1), 74–88.
- Verschuur, R., Didden, R., Lang, R., Sigafos, J., & Huskens, B. (2013). Pivotal response treatment for children with autism Spectrum disorders: A systematic review. *Review Journal of Autism and Developmental Disorders*, 1(1), 34–61. <https://doi.org/10.1007/s40489-013-0008-z>.
- Voos, A. C., Pelphrey, K. A., Tirrell, J., Bolling, D. Z., Wyk, B. V., Kaiser, M. D., et al. (2012). Neural mechanisms of improvements in social motivation after pivotal response treatment: Two case studies. *Journal of Autism and Developmental Disorders*, 43(1), 1–10. <https://doi.org/10.1007/s10803-012-1683-9>.
- Yang, D., Pelphrey, K. A., Sukhodolsky, D. G., Crowley, M. J., Dayan, E., Dvornek, N. C., et al. (2016). Brain responses to biological motion predict treatment outcome in young children with autism. *Translational Psychiatry*, 6(11), e948. <https://doi.org/10.1038/tp.2016.213>.

Neurexin 1

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Synonyms

NRXN1

Definition

Neurexin 1 (NRXN1) is a candidate gene in autism spectrum disorders (ASD). *NRXN1* is one of three members of the neurexin gene family, which encodes a group of cell adhesion molecules that are found at the synapses of nerve cells. Each neurexin gene can produce two types of neurexin proteins, both a long and a short version, which are referred to as α - and β -neurexins, respectively. Interestingly, the structure of neurexin genes is such that it is possible for one gene to generate many different protein isoforms due to the alternative splicing of RNA transcripts. This means that combinations of neurexin isoforms can result in tremendous diversity at synapses at the molecular level (Sudhof 2008).

There is growing interest in neurexins because they bind to *neuroligins*, another family of cell adhesion molecules that have been strongly implicated in ASD. Specifically, neurexins are present in the presynaptic region, while neuroligins are found postsynaptically, i.e., on the opposite side of the synapse. The interaction of neurexins and neuroligins is likely to play an important role in the formation of synapses. Moreover, neuroligins interact within the postsynaptic cell with members of the SHANK family of proteins, which have also been found in independent studies to be candidate genes in ASD (e.g., *SHANK3*) (Sudhof 2008). Taken together, the identification of *NRXN1* and neuroligins, such as *Neuroligin 4, X-linked (NLGN4X)*, as candidate genes indicates that cell adhesion molecules found at the synapse, and synaptic functioning in general, may represent common pathways in the biology of ASD.

A number of studies have implicated *NRXN1* in ASD. These studies identified rare variants disrupting *NRXN1* in affected individuals at level of the DNA sequence and at the structural level in chromosomes (Feng et al. 2006; Glessner et al. 2009; Marshall et al. 2008; Szatmari et al. 2007). Three of these studies used genome-wide, array-based cytogenetics to identify *copy number variants* (CNVs) associated with ASD. One study found a 300 kilobase deletion, which occurred *de novo*, disrupting *NRXN1* in two affected female siblings (Szatmari et al. 2007), and other identified deletions disrupting *NRXN1* that were

inherited by affected individuals (Glessner et al. 2009). CNVs disrupting *NRXN1* were not found in unaffected individuals (Glessner et al. 2010; Szatmari et al. 2007). Another study identified CNVs at *NRXN1* in individuals with ASD that were *de novo* and inherited (Marshall et al. 2008). However, this study also reported finding structural variation at *NRXN1* in a control population (Marshall et al. 2008).

The strength of the rare variant approach taken by these studies is that finding outliers which occur at a frequency of <5% in the general population favor the discovery of mutations with relatively large effect sizes. However, because these mutations are by definition rare, they are likely to occur in only a very small proportion of individuals with idiopathic ASD (State 2010). In addition, neurexins and neuroligins have been implicated in other psychiatric disorders. CNVs affecting *NRXN1* have been identified in individuals with schizophrenia, such that *NRXN1* is also considered a risk gene for schizophrenia (Kirov et al. 2009). Moreover, as discussed, CNVs disrupting this gene have been found in unaffected individuals as well (Marshall et al. 2008). While these findings are difficult to interpret, they are consistent with *pleiotropy* as an emerging theme in the genetics of ASD. According to this model, variation in risk genes may predispose to a range of clinical presentations due to interactions with other genetic and environmental factors (State 2010). Most importantly, the identification of candidate genes, such as *NRXN1*, has the potential to illuminate common biological mechanisms that underlie these disorders. Therefore, the identification of neurexins and neuroligins as risk genes has led investigators to focus on studying synapse function as a potential common pathway that may contribute to the biology of a range of psychiatric disorders in addition to ASD.

See Also

- [Common Disease-Rare Variant Hypothesis](#)
- [Neuroligins](#)
- [Pleiotropy](#)
- [SHANK 3](#)

References and Reading

- Feng, J., Schroer, R., Yan, J., Song, W., Yang, C., Bockholt, A., et al. (2006). High frequency of neuexin 1beta signal peptide structural variants in patients with autism. *Neuroscience Letters*, 409(1), 10–13.
- Glessner, J. T., Wang, K., Cai, G., Korvatska, O., Kim, C. E., Wood, S., et al. (2009). Autism genome-wide copy number variation reveals ubiquitin and neuronal genes. *Nature*, 459(7246), 569–573.
- Glessner, J. T., Reilly, M. P., Kim, C. E., Takahashi, N., Albano, A., Hou, C., et al. (2010). Strong synaptic transmission impact by copy number variations in schizophrenia. *Proceedings of the National Academy of Sciences of the United States of America*, 107(23), 10584–10589.
- Kirov, G., Rujescu, D., Ingason, A., Collier, D. A., O'Donovan, M. C., & Owen, M. J. (2009). Neurexin 1 (NRXN1) deletions in schizophrenia. *Schizophrenia Bulletin*, 35(5), 851–854.
- Marshall, C. R., Noor, A., Vincent, J. B., Lionel, A. C., Feuk, L., Skaug, J., et al. (2008). Structural variation of chromosomes in autism spectrum disorder. *American Journal of Human Genetics*, 82(2), 477–488.
- State, M. W. (2010). The genetics of child psychiatric disorders: Focus on autism and Tourette syndrome. *Neuron*, 68(2), 254–269.
- Sudhof, T. C. (2008). Neuroligins and neuexins link synaptic function to cognitive disease. *Nature*, 455(7215), 903–911.
- Szatmari, P., Paterson, A. D., Zwaigenbaum, L., Roberts, W., Brian, J., Liu, X. Q., et al. (2007). Mapping autism risk loci using genetic linkage and chromosomal rearrangements. *Nature Genetics*, 39(3), 319–328.

Historical Background

Most of what we know about the neuroanatomy of autism is derived from two sources: neuropathologic analyses of autistic brain tissue and structural MRI studies of populations of living patients with autism. By far, the latter provide us with the most information as MRI provides a noninvasive methodology allowing anatomic analysis of the whole brain in a large sample at multiple time points. Despite these potential advantages, early structural MRI studies of autism have been hampered by small, cross-sectional samples and differing techniques in the face of great clinical heterogeneity and change over the developmental span. For practical reasons, most imaging studies have been performed on higher-functioning adults with autism (a diagnosis of autism cannot usually be made until at least 2 years of age). Therefore, there is scant representation of the developmental periods (gestation and infancy) in which most brain abnormalities seem to emerge in autism (see below). Furthermore, because most studies are restricted to high-functioning individuals with autism, existing differences could theoretically be too subtle to detect. However, despite variable and inconsistent findings, some intriguing patterns have emerged (see Amaral et al. 2008; Lainhart 2006; Schumann and Nordahl 2011; and Stanfield et al. 2008 for excellent reviews).

Neuroanatomy

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Definition

Neuroanatomy is the study of the structure (morphology) and organization of the nervous system. Numerous morphometric studies have implicated perturbations in the processes of neocortical growth and organization in the development of autism.

Current Knowledge

Increased Head/Brain Size (Macrocephaly/Macroencephaly)

Studies examining head circumference (HC) and brain volume (by MRI) in autistic infants and children have demonstrated an abnormal developmental trajectory associated with autism. One of the most consistent neuroanatomic findings in ASD, derived from both structural MRI and post-mortem studies, is an abnormally enlarged brain in a significant subset of young children with autism. Retrospective studies of head circumference (HC) in the fetal (Hobbs et al. 2007) and perinatal (Courchesne and Pierce 2005) periods have generally reported little difference between

infants destined to be diagnosed with autism and control infants. However, by 2–4 years of age, average HC and brain volume (BV) are significantly (10–15%) increased in up to 70% of children with autism. Up to 25% of children in this subset actually meet formal criteria for macrocephaly ($HC \geq 2.0$ S.D. above the mean) and/or megalencephaly ($BV \geq 2.5$ S.D. above the mean) (Aylward et al. 2002; Courchesne et al. 2001; Redcay and Courchesne 2005; Sparks et al. 2002).

These findings are interpreted to be the result of early abnormal growth of the brain in the first 2 years of life before autism is diagnosed. While not yet confirmed directly, recent evidence strongly indicates this is the case. One large-scale, longitudinal study (Hazlett et al. 2011) found no difference in growth rates between autistic children and controls from the age of 2 years until 4–5 years. Rather, abnormal brain enlargement was already established by the age of 2 in a significant subset of their sample and remained unchanged by the age of 4–5. Interestingly, a recent neuropathologic study of seven autistic children (Courchesne et al. 2011) found that brain overgrowth in these individuals involved a 67% increase in the numbers of neurons in the prefrontal cortex relative to age-matched controls. Neurogenesis, the birth and early proliferation of neurons, is largely a prenatal process. At birth, cortical neurons are typically small so that an appreciable excess might not translate into a significant change in head size. However, in the first years of life, the typical dramatic increase in cytoplasmic volumes (both of the cell body and axons/dendrites) occurring in more than the usual complement of frontal neurons could account for abnormally accelerated brain growth in the first years of life. The first 2 years of life are usually a period of rapid brain growth in infants as neurons undergo significant postnatal growth in cell size and elaboration (actually overproduction) of axons, synapses, and dendrites. The degree of overgrowth appears to be correlated with severity of symptoms (Courchesne et al. 2001, 2003).

Following the initial period of rapid growth, growth rates in autism tend to decline significantly causing an apparent “normalization” of BV by adolescence/early adulthood (Redcay and Courchesne

2005). The functional significance of this is unknown as up to one third of individuals with autism may clinically deteriorate during this period while others may make significant gains in functioning. Mean HC remains slightly elevated into adulthood, and rates of macrocephaly, although lower, do remain increased overall. It must be noted that the above pattern does not hold true for all individuals with autism. Many show typical rates of head and brain growth, and a small subset even meet criteria for microencephaly, although this is more common in the setting of syndromic autism.

Gray and White Matter Compartments

Many investigators have attempted to determine which aspects of brain anatomy (i.e., gray or white matter compartments or structures/regions) contribute most to altered brain size in autism. It must be noted that GM and WM are not separate entities biologically as WM contains the myelinated axons of the neurons whose cell bodies reside in GM regions/structures. Many have noted an overall rostral-caudal (front to back) gradient in early brain growth trajectories. At the time of maximal brain growth during infancy and early childhood, Courchesne et al. (2001) found that cerebral gray matter (GM) and white matter (WM) were both increased by 18% and 38%, respectively. The frontal cortical GM and WM showed the most enlargement followed by the temporal lobe GM and WM and the parietal GM. The occipital GM and WM and parietal GM tended not to vary significantly from controls. Within the frontal lobes, the GM areas most affected were the dorsolateral and mesial prefrontal cortex (Carper and Courchesne 2005).

Similarly, the white matter most affected appears to be the radiate compartment immediately underlying these same prefrontal cortical areas (Herbert et al. 2004), whereas deeper WM and sagittal/bridging white matter (e.g., corpus callosum and internal capsule) are not enlarged. This suggests overgrowth of later-myelinating, intrahemispheric (corticocortical) connections (originating from layer III neurons) with relative sparing of the interhemispheric (layer III) and cortical to subcortical (layers V and VI) connections.

After childhood, WM is no longer increased relative to typically developing controls although

GM volumes remain elevated. It is therefore thought that “normalization” of BV may be due to a decrease in WM relative to GM, a complete reversal of the age-dependent cortical thinning that typically occurs during this period. Cerebral GM thinning is thought to be a sign of increasing maturity and is generally attributed to the concurrent processes of synaptic pruning and myelination. Excess (presumably weaker or imprecise) connections are eliminated during this period as both axon collaterals and terminal arbors are pruned through retraction or degeneration. This is necessary to develop precise functional connections and provide a substrate for neural plasticity. This process may be aberrant in autism, although this has not been demonstrated directly.

Cortical Surface Area/Thickness

Other cortical aspects, such as thickness, surface area, and gyration patterns, have been investigated by MRI and may provide important insights into the developmental mechanisms implicated in autism. Cortical thickness is a function of multiple factors, such as neuronal density, dendritic arborization, myelination, or sharpness of the gray-white junction, operating in various combinations. One study of cortical thickness (Hardan et al. 2006) reported a global increase in cortical thickness in 8–12-year-old autistic children, most pronounced in the parietal and temporal cortices. Other studies have, conversely, found thinning in the same cortical areas in adults. Early overgrowth followed by later loss may be a trend in critical development in autism, but until more large-scale, longitudinal studies are conducted, this remains speculative.

Cortical surface area also seems to be expanded early in autism. In their study of 59 young autistic children, Hazlett et al. (2011) found cortical gray matter volume increases appeared to be due to increased surface area rather than cortical thickness. Similarly, Hardan, Jou, Keshavan, Varma, and Minshew (2004) found an increase in cortical gyration in the frontal lobe. As folding is related to surface area, this finding implicates increased surface area. Abnormal morphology of the sylvian fissure, superior temporal sulcus, and inferior frontal gyrus have also been reported. Cortical surface area is

generally thought to be a function of the number of early, symmetric divisions by radial glial cells immediately prior to the onset of neurogenesis and migration. Minicolumns are the vertical cell columns created by sequential waves of migrating neurons traveling along radial glial fibers during early corticogenesis. It may be that reports of increased numbers of narrow minicolumns (Buxhoeveden et al. 2006; Casanova et al. 2006) in the frontal cortex of autistic brains may be related to these differences in surface area.

Corpus Callosum

Volumetric studies of the corpus callosum have consistently found decreased total, anterior, middle, and/or posterior callosal volumes in children and adults with autism (e.g., Hardan et al. 2000). This may serve to impede interhemispheric connectivity, thereby contributing to the clinical features of autism. These findings are relatively robust when controlling for IQ, BV, and gender.

Limbic System

Decreased hippocampal volumes have been reported in some high-functioning children, adolescents, and adult males with autism relative to age-matched controls and corrected for BV (e.g., Saitoh et al. 2001). The amygdala, likely involved in social cognition functions such as empathy or theory of mind, has been found to be relatively enlarged in children and adolescents with autism but that this difference tends to decrease with age. Furthermore, the degree of early enlargement appears to be negatively correlated with later social and communicative functioning (Munson et al. 2006). Interestingly, these findings may correspond to the neuropathologic findings of Kemper and Bauman (1993), who reported smaller, more packed neurons with attenuated dendritic arbors in many structures of the limbic system (particularly the amygdala, hippocampus, entorhinal cortex, and mammillary bodies) in autistic children.

Cerebellum

By far, the most consistent finding in neuropathologic studies of postmortem autism brain samples is a reduction in the density of cerebellar Purkinje neurons of the cerebellar cortex. This has engendered great interest in the cerebellum, and

numerous neuroanatomic studies of cerebellar volume/morphology have been performed. A handful of small-scale studies initially reported decreased volumes of cerebellar hemispheres; however, these were not replicated when total BV and IQ were controlled for. In contrast, a number of well-controlled studies actually found increased cerebellar GM and WM volumes in high-functioning children and young adult individuals with autism. It must be noted, however, that these findings were also highly sensitive to gender, IQ, and BV (Piven et al. 1997; Sparks et al. 2002).

A number of studies (performed by and reviewed in Courchesne et al. 1994) found specific areal reductions in cerebellar vermis lobules VI and VII in children and adults with autism relative to age-matched controls. Again, these studies were not matched for IQ and BV; however, their findings were replicated in 46 male autistic children with and without intellectual disability (Carper and Courchesne 2000). In their retrospective review of all 78 subjects, Courchesne et al. (1994) found that 87% of the patients had reduction and 13% had enlargement of vermal lobules VI–VII, suggesting that calculated means may be obscuring a bimodal or polymodal population in many of these studies.

Future Directions

In conclusion, despite a growing number of structural MRI studies examining morphology and growth of the autistic brain, only a few relatively consistent trends have emerged. Principle among these is an increased rate of brain growth in infancy and early childhood, predominately in the frontal, temporal and parietal lobes, and cerebellum. This appears to be followed by abnormally slow cerebral and cerebellar growth in later childhood and adolescence. Variable structural abnormalities in the corpus callosum, amygdala, and hippocampus have also been reported. These findings are consistent with the clinical evidence indicating the social and language functions reliant upon functional connectivity among these areas are aberrant in autism. It could be hypothesized that disturbances in early brain developmental processes (e.g., neuronal

proliferation, differentiation, migration, process elaboration, and/or synaptogenesis) underlie abnormal growth trajectories and result in altered brain anatomy and functional connectivity.

In order to overcome the limitations of previous morphometric analyses, future quantitative MRI studies will have to employ very large sample sizes of well-characterized individuals with autism and attempt to image them longitudinally over the developmental span, starting at birth if possible. Rigorous control matching for age, gender, and IQ will undoubtedly continue; however, samples will hopefully be expanded to include lower-functioning autistic and control individuals whenever feasible. Fortunately, significant trends toward this goal are already evident in recent literature. Larger, pooled samples and meta-analyses are increasingly employed. Earlier detection and diagnosis of autism will also be possible as evidenced by findings from at-risk infant sibling studies. Finally, recent computer-based measurement techniques, e.g., high definition fiber tracking, will permit the detection of subtle morphologic differences not appreciated by traditional quantitative/structural MRI studies or even histopathologic examination of brain tissue.

N

See Also

- ▶ [Amygdala](#)
- ▶ [Cerebellar Abnormalities in Autism](#)
- ▶ [Corpus Callosum Abnormalities in Autism](#)
- ▶ [Prefrontal Cortex](#)
- ▶ [Purkinje Cells](#)
- ▶ [Temporal Lobes](#)

References and Reading

- Amaral, D. G., Schumann, C. M., & Nordahl, C. W. (2008). Neuroanatomy of autism. *Trends in Neurosciences*, 31, 137–145.
- Aylward, E., Minshew, N., Field, K., Sparks, B., & Singh, N. (2002). Effects of age on brain volume and head circumference in autism. *Neurology*, 59, 175–183.
- Buxhoeveden, D., Semendeferi, K., Buckwalter, J., Schenker, N., Switzer, R., & Courchesne, E. (2006). Reduced minicolumns in the frontal cortex of patients with autism. *Neuropathology and Applied Neurobiology*, 32, 483–491.

- Carper, R., & Courchesne, E. (2000). Inverse correlation between frontal lobe and cerebellum sizes in children with autism. *Brain*, 123, 836–844.
- Carper, R., & Courchesne, E. (2005). Localized enlargement of the frontal cortex in early autism. *Biological Psychiatry*, 57, 126–133.
- Casanova, M., van Kooten, I., Switala, A., van Engeland, H., Heinsen, H., Steinbusch, H., et al. (2006). Minicolumnar abnormalities in autism. *Acta Neuropathologica*, 112, 287–303.
- Courchesne, E., & Pierce, K. (2005). Brain overgrowth in autism during a critical time in development: Implications for frontal pyramidal neuron and interneuron development and connectivity. *International Journal of Developmental Neuroscience*, 23, 153–170.
- Courchesne, E., Saitoh, O., Yeung-Courchesne, R., Press, G., Lincoln, A., Haas, R., et al. (1994). Abnormality of cerebellar vermal lobules VI and VII in patients with infantile autism: Identification of hypoplastic and hyperplastic subgroups with MR imaging. *American Journal of Roentgenology*, 162, 123–130.
- Courchesne, E., Karns, C., Davis, H., Ziccardi, R., Carper, R., Tigue, Z., et al. (2001). Unusual brain growth patterns in early life in patients with autistic disorder: An MRI study. *Neurology*, 57, 245–254.
- Courchesne, E., Carper, R., & Akshoomoff, N. (2003). Evidence of brain overgrowth in the first year of life in autism. *Journal of the American Medical Association*, 290, 337–344.
- Courchesne, E., Mouton, P. R., Calhoun, M. E., Semendeferi, K., Ahrens-Barbeau, C., Hallet, M. J., et al. (2011). Neuron number and size in prefrontal cortex of children with autism. *Journal of the American Medical Association*, 306, 2001–2010.
- Hardan, A., Minshew, N., & Keshavan, M. (2000). Corpus callosum size in autism. *Neurology*, 55, 1033–1036.
- Hardan, A., Jou, R., Keshavan, M., Varma, R., & Minshew, N. (2004). Increased frontal cortical folding in autism: A preliminary MRI study. *Psychiatry Research*, 131, 263–268.
- Hardan, A., Muddasani, S., Vemulapalli, M., Keshavan, M., & Minshew, N. (2006). An MRI study of increased cortical thickness in autism. *The American Journal of Psychiatry*, 163, 1290–1292.
- Hazlett, H. C., Poe, M. D., Gerig, G., Styner, M., Chappell, C., Gimpel Smith, R., et al. (2011). Early brain overgrowth in autism associated with an increase of cortical surface area before age 2 years. *Archives of General Psychiatry*, 68, 467–476.
- Herbert, M., Ziegler, D., Makris, N., Filipek, P., Kemper, T., Normandin, J., et al. (2004). Localization of white matter volume increase in autism and developmental language disorder. *Annals of Neurology*, 55, 530–540.
- Hobbs, K., Kennedy, A., Dubray, M., Bigler, E. D., Petersen, P. B., McMahon, W., et al. (2007). A retrospective fetal ultrasound study of brain size in autism. *Biological Psychiatry*, 62, 1048–1055.
- Kemper, T. L., & Bauman, M. L. (1993). The contribution of neuropathologic studies to the understanding of autism. *Neurologic Clinics*, 11, 175–187.
- Lainhart, J. (2006). Advances in neuroimaging research for the clinician and geneticist. *American Journal of Medical Genetics. Part C, Seminars in Medical Genetics*, 142C, 33–39.
- Munson, J., Dawson, G., Abbott, R., Faja, S., Webb, S., Friedman, S., et al. (2006). Amygdalar volume and behavioral development in autism. *Archives of General Psychiatry*, 63, 686–693.
- Piven, J., Saliba, K., Bailey, J., & Arndt, S. (1997). An MRI study of autism: The cerebellum revisited. *Neurology*, 49, 546–551.
- Redcay, E., & Courchesne, E. (2005). When is the brain enlarged in autism? A meta-analysis of all brain size reports. *Biological Psychiatry*, 58, 1–9.
- Saitoh, O., Karns, C., & Courchesne, E. (2001). Development of the hippocampal formation from 2 to 42 years: MRI evidence of smaller area dentate in autism. *Brain*, 124, 1317–1324.
- Schumann, C., & Nordahl, C. (2011). Bridging the gap between MRI and postmortem research in autism. *Brain Research*, 1380, 175–186.
- Sparks, B., Friedman, S., Shaw, D., Aylward, E., Echelard, A., Artu, A., et al. (2002). Brain structural abnormalities in young children with autism spectrum disorder. *Neurology*, 59, 184–192.
- Stanfield, A. C., McIntosh, A. M., Spencer, M. D., Philip, R., Gaur, S., & Lawrie, S. M. (2008). Towards a neuroanatomy of autism: A systematic review and meta-analysis of structural magnetic resonance imaging studies. *European Psychiatry*, 23, 289–299.

Neurobiology

► Neuroscience

Neurochemistry

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Definition

Neurochemistry is the study of chemicals and their reactions in the nervous system. Potentially a tremendously broad field, neurochemistry could encompass nearly all the anatomy and physiology

of the central and peripheral nervous systems. Practically, “neurochemical” research has tended to focus on endogenous chemicals (especially neurotransmitters) and nongenetic molecular aspects of the nervous system, the pathogenesis of neuropsychiatric disorders, and the identification of disease biomarkers.

Current Knowledge

Neurochemical research in autism has included studies on serotonin, noradrenaline (norepinephrine), dopamine, glutamate, GABA, and cholinergic and stress response systems. Most of the studies have examined levels of neurochemicals in either cerebrospinal fluid, plasma, or urine; only limited postmortem brain data are available and are not definitive.

Research on serotonin has focused on the well-replicated platelet hyperserotonemia of autism and apparent decrease in function and expression of the platelet and brain serotonin type 2 receptor. Global dopaminergic metabolism and functioning appear normal based on the determination of dopamine metabolite levels in plasma and urine and on measurements of the dopamine-modulated hormone prolactin in plasma. Research on stress response systems, including the noradrenergic-sympathetic/adrenomedullary system and the hypothalamic-pituitary-adrenal (HPA) axis, has found basal functioning to be similar in autism and typically developing individuals. However, in autism, the systems appear hyperreactive to stress. Decreased production of melatonin in autism has been consistently reported and focuses attention on circadian processes.

Future Directions

Future research will continue to attempt to understand the underlying causes of the well-replicated platelet hyperserotonemia, the extent and consequences of altered serotonin type-2 receptor expression and function, of possible cholinergic alterations, and the lower melatonin production

seen in autism. In addition, metabolomic studies will attempt to identify patterns of alteration specific to autism. In general, studies will need to take account of the large degree of heterogeneity in the autism category.

See Also

- [Dopamine](#)
- [Norepinephrine](#)
- [Serotonin](#)

References and Reading

- Anderson, G. M. (2009). Conceptualizing autism: The role for emergence. *Journal of the American Academy of Child & Adolescent Psychiatry*, 48, 688–691.
- Anderson, G. M., Horne, W. C., Chatterjee, D., & Cohen, D. J. (1990). The hyperserotonemia of autism. *Annals of the New York Academy of Sciences*, 600, 331–342.
- Cook, E., & Leventhal, B. (1996). The serotonin system in autism. *Current Opinion in Pediatrics*, 8, 348–354.
- Deutsch, S. I., Urbano, M. R., Neumann, S. A., Burket, J. A., & Katz, E. (2010). Cholinergic abnormalities in autism: Is there a rationale for selective nicotinic agonist interventions? *Clinical Neuropharmacology*, 33, 114–120.
- Lam, K. S., Aman, M. G., & Arnold, L. E. (2006). Neurochemical correlates of autistic disorder: A review of the literature. *Research in Developmental Disabilities*, 27(3), 254–289.
- McDougle, C. J., Erickson, C. A., Stigler, K. A., & Posey, D. J. (2005). Neurochemistry in the pathophysiology of autism. *Journal of Clinical Psychiatry*, 66(Suppl. 10), 9–18.
- Perry, E. K., Lee, M. L., Martin-Ruiz, C. M., Court, J. A., Volsen, S. G., Merrit, J., et al. (2001). Cholinergic activity in autism: abnormalities in the cerebral cortex and basal forebrain. *American Journal of Psychiatry*, 158, 1058–1066.
- Pickett, J., & London, E. (2005). The neuropathology of autism: A review. *Journal of Neuropathology & Experimental Neurology*, 64, 925–935.
- Polleux, F., & Lauder, J. M. (2004). Toward a developmental neurobiology of autism. *Mental Retardation & Developmental Disabilities Research Reviews*, 10, 303–317.
- Siegel, G. J., Albers, R. W., Brady, S. T., & Price, D. L. (2006). *Basic neurochemistry* (7th ed.). Boston: Elsevier Academic Press.
- Waterhouse, L., Fein, D., & Modahl, C. (1996). Neurofunctional mechanisms in autism. *Psychological Review*, 103, 457–489.

Neurodiversity

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The concept of “neurodiversity” refers to the diversity of neurocognitive and/or sensory functioning differing from that associated with the “neurotypical” population (also known as the “predominant neurotype”). In defining “neurodiversity” one should first consider the meaning of “neurotypical.” Perszyk (2013, p. 1) defines “neurotypical” as:

Description of a medically and psychologically healthy individual demonstrating a normative pattern of neurodevelopment. Typically used specifically in contrast with individuals experiencing an atypical developmental course, such as autism

The implication in Perszyk’s definition that a nonnormative pattern of neurodevelopment is a “psychologically unhealthy” one is incorrect as many neurodivergent conditions simply represent the wide variety of differences amongst humanity. For instance, autistic advocates argue persuasively that autism is an example of ordinary human difference. However, the reference to atypical development in the Perszyk definition captures a majority view of autism and other conditions included within the compass of neurodiversity.

An individual whose cognitive and/or sensory functioning differs from that of the neurotypical population is said to be “neurodivergent,” whereas the concept of “neurodiversity” expresses the diversity of cognitive and sensory functioning and in so doing refers to the entire population, i.e., the concept encompasses neurodivergent people *and* neurotypical people.

Hughes (2015, pp. 5/6) is one of many scholars to define neurodiversity by listing conditions that she considers fall within the ambit of the term. In doing so, she refers to the expansion of a definition “around ‘high-functioning’ autism . . . to describe any kind of cognitive processing or way of making sense of the world that deviates from ‘typical’ ways of thinking and being.” Hughes

describes hers as a narrow definition of neurodiversity. A broader definition in her terms is one that includes all autistic people irrespective of their so-called functioning level. The UK Developmental Adult Neuro-Diversity Association (DANDA) defines neurodiversity as “difficulties with organisation, memory, concentration, time, direction, perception, sequencing. Poor listening skills—leading to low self-esteem, anxiety, depression but creative, original, determined.” DANDA’s definition of neurodiversity is a broad one in Hughes’ terms. The DANDA definition also refers to some of the strengths associated with neurodivergence. Steve Silberman (2015, p. 17) is another author who refers to strengths when defining “neurodiversity” as: “the notion that conditions like autism, dyslexia and attention-deficit/hyperactivity disorder . . . should be regarded as naturally occurring cognitive variations with distinctive strengths that have contributed to the evolution of technology and culture rather than mere checklists of deficits and dysfunctions.” His definition is also of interest for its reference to naturally occurring variations.

Taken literally, the term “neurodivergence” could also encompass any condition marked by a difference from the typical condition of the human nervous system (including brain, spinal column, and peripheral nerves). For example, many conditions causing physical impairment, such as cerebral palsy, Parkinson’s disease, or multiple sclerosis, could be included. However, these “physical neurological” conditions are generally not included within the concept of neurodivergence. This is perhaps in part because the interests of people with such conditions are generally represented by disabled people’s movements which have historically centered around people with physical impairments and related access or assistance needs. The neurodiversity movement evolved separately from these disabled people’s movements, though arguably with some conceptual roots in them (Graby 2015). However, the absence of these conditions from the scope of “neurodivergence,” as commonly used, does call into question the accuracy of the “neuro” part of this term.

Some people are beginning to identify with the concept of neurodiversity who have been

classified by psychiatry in categories more commonly associated with “mental health” than with “disability” (such as “schizophrenia” and “bipolar disorder”). For example, Suzanne Antonetta, who was diagnosed with “bipolar disorder,” gave her 2005 book *A Mind Apart* – which describes her experience, and that of autistic author, Dawn Prince-Hughes, as well as those of friends with diagnoses such as “dissociative identity disorder” (formerly known as “multiple personality disorder”) – the subtitle “Travels in a Neurodiverse World.” An online community of self-defined “multiples” also exists, who see their separate “personalities” not as a dissociative pathology but as different “people” sharing a brain and body, each of whom has a right to exist. This has links to, and overlaps with, the autistic and broader neurodiverse communities (Baggs et al. 2006), although it should be noted that not all proponents of neurodiversity accept that the term should be as broad as this.

Finally, we should draw attention to the disagreement over whether or not individuals with intellectual disability only (i.e., not autistic, dyslexic, dyspraxic) should be classed as neurodivergent. We believe the majority of scholars regard such individuals as neurotypical but are aware that some scholars argue that these individuals are neurodivergent.

There are no definitive definitions in relation to neurodiversity.

See Also

- [Broader Autism Phenotype](#)
- [Neurotypical](#)

References and Reading

- Antonetta, S. (2007). *A mind apart: Travels in a neurodiverse world*. London: Penguin.
- Baggs, A. M., Schwarz, P., Smith, J., & Tisoncik, L. (2006). *Who can call themselves autistic?* <http://archive.autistics.org/library/whoisautistic.html>. Last accessed 3 Jan 2019.
- DANDA (Developmental Adult Neuro-Diversity Association). (2011). *Neurodiversity*. Available at <http://www.danda.org.uk>. Accessed 7 Feb 2011.

Graby, S. (2015). Neurodiversity: Bridging the gap between the disabled people’s movement and the mental health system survivors’ movement. In H. Spandler, J. Anderson, & B. Sapey (Eds.), *Madness, distress and the politics of disablement* (pp. 231–244). Bristol: Policy Press.

Hughes, J. M. F. (2015). *Changing conversations around autism: A critical, action implicative discourse analysis of US neurodiversity advocacy online*. Doctoral thesis.

Perszyk, D. (2013). Neurotypical. In F. R. Volkmar (Ed.), *Encyclopedia of autism spectrum disorders*. New York: Springer.

Silberman, S. (2015). *NeuroTribes: The legacy of autism and how to think smarter about people who think differently*. London, UK: Allen & Unwin.

Neuroleptic-Induced Dyskinesia

- [Tardive Dyskinesia](#)

Neuroleptics

- [Antipsychotics: Drugs](#)

Neuroligins

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Synonyms

NLGN1; NLGN2; NLGN3; NLGN4X; NLGN4Y

Structure

Neuroligins are a family of proteins which are expressed in the brain at the surface of nerve

cells. There are five members as follows, ranging in size from 823 to 853 amino acids in length:

NLGN1 located at chromosome 3q26.31

NLGN2 located at chromosome 17p13.1

NLGN3 located at chromosome Xq13.1

NLGN4X located at chromosome Xp22.32

NLGN4Y located at chromosome Yq11.221

Neuroligins are postsynaptic membrane proteins in the nervous system. In other words, they exist on the outside membrane of the nerve cell which receives information in the form of chemical neurotransmitters from presynaptic cells. They are classified as type 1 membrane proteins; therefore, they pass from the outside of the cell through the membrane to the inside of the cell exactly once, unlike some proteins which pass through the membrane two or more times. The amino acid structure consists of a large N-terminal extracellular domain followed by a single transmembrane region and a short cytoplasmic sequence. The extracellular domain consists of an acetylcholinesterase (AChE)-like domain which is the site for extracellular binding (Lise and El-Husseini 2006). Mutations in *NLGN3* and *NLGN4X* have been implicated in autism spectrum disorders (ASDs). They are known binding partners of β -neurexins, which in turn complexes with Shank (SH3 and multiple ankyrin repeat domain) molecules, two other neuronal proteins which have also been associated with ASDs. These molecules provided among the first evidence that the neural synapse may be dysfunctional in ASDs.

Function

In addition to serving as cell adhesion molecules across a synapse, neuroligins have an important role in synapse formation. They bind to β -neurexin, a protein which exists on the presynaptic side of the synaptic cleft. This binding helps lead to the connection between two nerve cells and the creation of a synapse (Fabrichny 2007). On the inside of the postsynaptic cell, neuroligins bind to Shank, a structural protein which also

plays an important role in the development and support of the synapse. It has been shown that the different neuroligins express at different points during development. Neuroligins 3 and 4 express significantly during the fetal stage, whereas neuroligin 1 expression increases in the childhood years into adulthood (Sudhof 2008). In addition, neuroligins have been suggested to play a role in the formation of new blood vessels from pre-existing vessels (Bottos et al. 2009).

Pathophysiology

Thomas et al. (1999) reported on three girls with ASD who had deletions in the short arm of chromosome X. Upon further examination, the deleted region was revealed to contain the gene *NLGN4X*. This was the first evidence that neuroligins may be related to ASDs.

Jamain et al. (2003) found two deleterious mutations in neuroligin genes. The first was in a Swedish family with two affected brothers, one with Asperger syndrome and one with autism. Both of the brothers had the same mutation in *NLGN4X* which was inherited from their mother. This mutation was not found in any other family members, including the unaffected brother, and not in 350 unrelated controls. This de novo mutation in the mother resulted in a premature stop codon approximately halfway into the sequence and truncated the protein. It completely deleted the transmembrane portion of the protein. The mutation may not have had a significant impact on the mother because she had a second normal copy of *NLGN4X* on her other X chromosome. A missense mutation in *NLGN3* was identified in a second Swedish family. This mutation was also maternally inherited by two brothers, one with Asperger syndrome and one with autism. This mutation caused an amino acid change resulting in a nonfunctional esterase domain, impairing its ability to bind neurexin. This mutation was not found in any of the 200 control individuals used for the study. The mutations found in this study are very damaging and highly suggestive that *NLGN3* and *4X* have a significant impact on neurodevelopment. Numerous studies since have

been published examining *NLGN* mutations in other cohorts; they continue to be an important source of rare mutations in ASD.

Scientists have created *NLGN3* knockout mice, meaning that these mice do not have a functional copy of the *NLGN3* gene. They have decreased brain size and, behaviorally, show decreased context and cued conditioning, hyperactivity, abnormal social skills, and vocalization (Radyushkin et al. 2009). Replicating symptoms similar to those in humans with a *NLGN3* deficiency lends further evidence to the case that mutations in *NLGNs* contribute to the development of ASD.

See Also

► [SHANK 3](#)

References and Reading

- Bottos, A., Destro, E., Rissone, A., Graziano, S., Cordara, G., Assenzio, B., et al. (2009). Synaptic proteins neurexins and neuroligins are widely expressed in the vascular system and contribute to its functions. *Proceedings of the National Academy of Sciences USA*, 106(49), 20782–20787.
- Bourgeron, T. (2009). Synaptic trek to autism. *Neurobiology*, 19, 231–234.
- Dean, C., & Dresbach, T. (2006). Neuroligins and cell adhesion, synapse formation and cognitive function. *Trends in Neurosciences*, 29(1), 21–29.
- Fabrichny, I. P. (2007). Structural analysis of the synaptic protein neuroligin and its β -neurexin complex: Determinants for folding and cell adhesion. *Neuron*, 56, 979–991.
- Gupta, A. R., & State, M. W. (2007). Recent advances in the genetics of autism. *Biological Psychiatry*, 61, 429–437.
- Jamain, S., Quach, H., Betancur, C., Råstam, M., Colinaux, C., Gillberg, I. C., et al. (2003). Mutations of the X-linked genes encoding neuroligins *NLGN3* and *NLGN4* are associated with autism. *Nature Genetics*, 34, 27–29.
- Lise, M. F., & El-Husseini, A. (2006). The neuroligin and neurexin families: From structure to function at the synapse. *Cell Molecular Life Sciences*, 63, 1833–1849.
- Peca, J., Ting, J., & Feng, G. (2011). SnapShot: Autism and the synapse. *Cell*, 147, 706–707.
- Penzes, P., Cahill, M. E., Jones, K. A., VanLeeuwen, J. E., & Woolfrey, K. M. (2011). Dendritic spine pathology in neuropsychiatric disorders. *Nature Neuroscience*, 14(3), 285–293.
- Radyushkin, K., Hammerschmidt, K., Boretius, S., Varoqueaux, F., El-Kordi, A., Ronnenberg, A., et al. (2009). Neuroligin-3-deficient mice: Model of a monogenic heritable form of autism with an olfactory deficit. *Genes, Brain, and Behavior*, 8(4), 416–425.
- Sudhof, T. C. (2008). Neuroligins and neurexins link synaptic function to cognitive disease. *Nature*, 7215, 903–911.
- Thomas, N. S., Sharp, A. J., Browne, C. E., Skuse, D., Hardie, C., & Dennis, N. R. (1999). XP deletions associated with autism in three females. *Human Genetics*, 104, 43–48.
- Yan, J., & Feng, J. (2008). Analysis of the neuroligin 4Y gene in patients with autism. *Psychiatric Genetics*, 18, 204–207.

Neurologist

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Definition

A physician specializing in the evaluation and treatment of disorders of the nervous system, e.g., disorders of the brain, spinal cord, peripheral nerves, and muscles. In the USA and Canada, these specialists have received an M.D. or D.O. degree followed by 1–3 years of pediatric or internal medicine training and then a 3-year fellowship in neurology. The neurology training can focus entirely on adults or can have an emphasis on children. Following specialty training, board certification is available in neurology or neurology with special competence in child neurology. Thereafter, the individual can practice general adult or general child neurology, or pursue subspecialty training in a wide range of areas such as behavioral neurology, genetics, epilepsy, developmental disabilities, geriatrics, stroke, dementia, etc. In addition, there are neurologists who have also completed a Ph.D. in an area of interest that confers additional expertise. Training systems differ somewhat in other countries.

Neurologists may be consulted for the evaluation and treatment of headaches, epilepsy or seizures, deterioration in function, learning

disabilities, cerebral palsy, delayed or abnormal development, weakness, and a range of signs and symptoms that suggest a brain, spinal cord, peripheral nerve, or muscle origin. Neurologists are often consulted in the assessment of an individual with ASD when the possibility of seizures is raised or in cases of regression to determine specific etiologies other than ASD that may require a specific treatment or have different genetic implications. It is important that the neurologist evaluating an individual with ASD or suspected of having ASD has expertise and experience in ASD. Neurologists also contribute to research in autism (articles listed below provide a few examples of these contributions).

See Also

► [American Academy of Neurology](#)

References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2010). Connecting genes to brain in the autism spectrum disorders. *Archives of Neurology*, 67(4), 395–399. Review.
- Bauman, M., & Kemper, T. L. (1985). Histoanatomic observations of the brain in early infantile autism. *Neurology*, 35(6), 866–874.
- Dowell, L. R., Mahone, E., & Mostofsky, S. H. (2009). Associations of postural knowledge and basic motor skill with dyspraxia in autism: Implication for abnormalities in distributed connectivity and motor learning. *Neuropsychology*, 23(5), 563–570.
- Gant, J. C., Thibault, O., Blalock, E. M., Yang, J., Bachstetter, A., Kotick, J., et al. (2009). Decreased number of interneurons and increased seizures in neuropilin 2 deficient mice: Implications for autism and epilepsy. *Epilepsia*, 50(4), 629–645. Epub 2008 Jul 24.
- Mazefsky, C. A., & Minshew, N. J. (2010). The spectrum of autism – from neuronal connections to behavioral expression etiology, pathophysiology, and diagnosis of autism spectrum disorder. *Virtual Mentor*, 12(11), 867–872.
- Minshew, N. J., Goldstein, G., & Siegel, D. J. (1997). Neuropsychologic functioning in autism: Profile of a complex information processing disorder. *Journal of International Neuropsychological Society*, 3(4), 303–316.
- Spence, S. J., & Schneider, M. T. (2009). The role of epilepsy and epileptiform EEGs in autism spectrum disorders. *Pediatric Research*, 65(6), 599–606. Review. PubMed PMID: 19454962; PubMed Central PMCID: PMC2692092.
- Tuchman, R., & Rapin, I. (2002). Epilepsy in autism. *Lancet Neurology*, 1(6), 352–358.
- Vasa, R. A., Ranta, M., Huisman, T. A., Pinto, P. S., Tillman, R. M., & Mostofsky, S. H. (2011). Normal rates of neuroradiological findings in children with high functioning autism. *The Journal of Autism and Developmental Disorders*.
- Voineagu, I., Wang, X., Johnston, P., Lowe, J. K., Tian, Y., Horvath, S., et al. (2011). Transcriptomic analysis of autistic brain reveals convergent molecular pathology. *Nature*, 474(7351), 380–384. <https://doi.org/10.1038/nature10110>.
- Wassink, T. H., Hazlett, H. C., Epping, E. A., Arndt, S., Dager, S. R., Schellenberg, G. D., et al. (2007). Cerebral cortical gray matter overgrowth and functional variation of the serotonin transporter gene in autism. *Archives of General Psychiatry*, 64(6), 709–717.

Neurontin

- [Gabapentin](#)
- [Neurontin \(Gabapentine\)](#)

Neurontin (Gabapentine)

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Synonyms

[Neurontin](#)

Definition

Among Autism Spectrum Disorder (ASD) patients, insomnia and disruptive-compulsive behaviors remain difficult to manage from limited efficacy or adverse side effects with current

pharmacological therapies (Blackmer and Feinstein 2016; Brown and Malow 2016; Guglielmo et al. 2013; Kotagal 2012). In recent years, antiepileptic drugs, specifically gabapentine, have become increasingly utilized for impulsive and aggressive behavior among neurodevelopmental disorders (Guglielmo et al. 2013). Gabapentin is an amino acid derivative used to treat anxiety disorders, alcohol dependence, insomnia, obsessive-compulsive disorder, and somatoform disorder (Guglielmo et al. 2013). Current research suggests gabapentin's acts through α^2 - δ subunits of voltage-gated calcium channels, which modulate synaptic homeostasis within the nervous system (Guglielmo et al. 2013; Wang et al. 2016). Although effective, large-scale trials of gabapentine are needed to determine its efficacy and safety for ASD and other neurodevelopmental disorders.

See Also

- Midazolam
- Zyprexa

References and Reading

- Blackmer, A. B., & Feinstein, J. A. (2016). Management of sleep disorders in children with neurodevelopmental disorders: A review. *Review of Therapeutics*, 36(1), 84–98.
- Brown, K. M., & Malow, B. A. (2016). Pediatric Insomnia. *Chest*, 149(5), 1332–1339. <https://doi.org/10.1378/chest.15-0605>.
- Guglielmo, R., Ioime, L., Grandinetti, P., & Janiri, L. (2013). Managing disruptive and compulsive behaviors in adult with autistic disorder with gabapentin. *Journal of Clinical Psychopharmacology*, 33(2), 273–274. <https://doi.org/10.1097/jcp.0b013e318285680c>.
- Kotagal, S. (2012). Treatment of Dyssomnias and parasomnias in childhood. *Current Treatment Options in Neurology*, 14(6), 630–649. <https://doi.org/10.1007/s11940-012-0199-0>.
- Wang, T., Jones, R. T., Whippen, J. M., & Davi, G. W. (2016). $\alpha^2\delta$ -3 is required for rapid trans-synaptic homeostatic signaling. *Cell Reports*, 16(11), 2875–2888.

Neuropathology

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Definition

Neuropathology is the scientific study of disorders of the nervous system. Neuropathologic studies of autism spectrum disorders (ASD) are conducted on postmortem brain tissue obtained from individuals who were diagnosed with an ASD during life as well as normal controls. Tissue from each donor individual is stored in special research banks, both fixed (for microscopic anatomic and antibody expression studies) and frozen (for molecular and biochemical studies).

Historical Background

Due to early issues of variation in diagnostic precision, tissue availability, collection protocols, sample demography, postmortem interval, and research goals, early postmortem research into ASD is mainly represented by scattered case studies or small series of individuals with relatively poorly documented autistic features or ill-defined syndromes. Although conditions for ASD researchers have improved greatly due to coordinated tissue collection and banking efforts, neuropathologic studies continue to be hampered by small sample size in the face of great underlying heterogeneity in etiology, comorbidity, and sampling. For these reasons, less than 100 brains have been examined to date and have yielded somewhat inconsistent, albeit intriguing results.

The first detailed postmortem report of a well-documented, nonsyndromic case of ASD was published in 1985 by Bauman and Kemper. After completing 10 additional cases over the next decade (reviewed in Kemper and Bauman

1993), three common findings emerged: (1) increased brain weight in children and decreased brain weight in adults, (2) increased packing densities of smaller-sized neurons (with underdeveloped dendritic arbors) in select forebrain structures such as the anterior cingulate gyrus, and (3) decreased numbers of Purkinje neurons in the cerebellum. These findings have been replicated and expanded upon by more recent research, outlined in the next section.

Current Knowledge

Autism and its clinically similar spectrum disorders (ASDs) are now conceptualized as developmental neurobiological disorders affecting elaboration of the forebrain circuitry that underlies those abilities most unique to human beings. The development of neural circuitry depends upon the coordinated interactions of numerous genetic/molecular cascades and environmental/experiential exposures. At the basic level, the physical substrate of neural circuitry is provided by neurons, their processes (axons and dendrites), and their synapses on neighboring or distant neurons. Wiring the brain requires that neurons proliferate, acquire the correct laminar identities, migrate to the appropriate locations, extend axons which must make guidance decisions with a high degree of spatial and temporal fidelity, and finally, establish synaptic connections with appropriate target neurons. Neuropathologic studies have reported subtle abnormalities in multiple brain regions (cerebral cortex, cortical white matter, limbic structures, brainstem, and cerebellum), indicating more than one of the abovementioned pre- and early postnatal developmental processes may be altered in various combinations to produce the heterogeneous phenotypes observed in ASD.

Most of the information we have concerning the physical or anatomic substrate of altered connectivity in ASD comes from clinical studies using magnetic resonance imaging (MRI). Structural MRI studies have uncovered ample evidence of altered developmental growth trajectories as well as size differences both of the whole brain and specific structures/compartments in

substantial subsets of adults and children with ASD (reviewed in Lainhart et al. 2006). Most striking is the finding of early overgrowth of the brain in ASD. While not significantly different from controls at birth, up to 70% of infants with ASD exhibit abnormally accelerated brain growth in the first year of life followed by a relative deceleration in age-related growth in some. This is particularly marked in the frontal and temporal cortices and the amygdala.

While these findings strongly indicate altered events in brain development, there is no way to directly attribute the gross findings to these events as MRI technology currently lacks the required resolution. Therefore, complementary neuropathologic studies of postmortem brain tissue are necessary to directly examine alterations in brain microarchitecture which (presumably) reflect the neurobiological underpinnings of ASD at the neuronal level. “Complementary” because there are both advantages and drawbacks to tissue-based research relative to structural MRI studies. Histologic studies allow a greater degree of resolution, i.e., the ability to examine neuronal structure, distribution, and gene/protein expression within the same individual. However, they are often more laborious and time consuming, making integrated analysis of the whole brain prohibitive. Despite these difficulties, recent efforts have yielded some fascinating patterns, outlined by region below.

Brain Size

Several neuropathologic studies have replicated the MRI findings of early increased brain growth followed by deceleration or, occasionally, persistence of macrocephaly in individuals with ASD as evidenced by increased brain weight. The white matter below the frontal lobe is enlarged in young children with autism but not in adults as assessed by structural imaging (Herbert et al. 2004). Kemper and Bauman noted that the brain weight of younger autistic individuals was significantly greater than comparable controls. Bailey et al. (1998) likewise noted increased brain size/weight in some young adults. A recent preliminary report of seven autistic children (Courchesne et al. 2011) found that brain overgrowth in these individuals involved a 67% increase in the numbers of

neurons in the prefrontal cortex relative to age-matched controls. Neurogenesis, the birth and early proliferation of neurons, is largely a prenatal process. At birth, cortical neurons are typically small so that an appreciable excess might not translate into a significant change in head size. However, in the first years of life, the typical dramatic increase in cytoplasmic volumes (both of the cell body and axons/dendrites) occurring in more than the usual complement of frontal neurons could account for abnormally accelerated brain growth in the first years of life.

Cortex

Besides recent findings, discussed above, of excess neurons in the prefrontal cortex of autistic children, more than half of all *postmortem investigations have uncovered* features of cortical dysgenesis, i.e., abnormalities of neuronal layering or distribution caused by dysregulation of neurogenesis, neuronal migration, or maturation, in ASD brains. In the seminal postmortem studies by Kemper and Bauman, the only consistent abnormality in the cerebral cortex was small neuronal cell size and increased cell packing in the anterior cingulate cortex (ACC) in five of six subjects with ASD compared to age-matched controls. The ACC, a component of the limbic system involved in monitoring of social interaction, is one of the cortical areas most consistently associated with abnormalities in imaging and neuropathologic studies. Using stereologic techniques, Simms, Kemper, Timbie, Bauman, and Blatt (2009) variably found decreased cell size and decreased cell packing in different subregions of the ACC. Other findings included irregular lamination, increased subcortical white matter neurons, and densities of von Economo neurons that were either significantly more or less than seen in age-matched controls. Avino and Hutsler (2011) similarly found a poorly defined boundary between gray matter and white matter in the dorsolateral frontal and parietal cortices as well as the superior temporal gyrus. Van Kooten et al. (2008) conducted a stereologic study on the fusiform gyrus, involved in face processing, and found significantly lower neuronal densities within layer III and lower total neuron numbers in layers

III, V, and VI, as well as smaller average cell volumes of neurons in layers V and VI.

Bailey et al. (1998) observed more significant and widespread cortical dysgenic lesions in four of six subjects with ASD. These included a thickened cortical ribbon, increased neuronal densities, gray matter heterotopias, excess neurons in the molecular layer and subcortical white matter, and irregular lamination. Increased brain size and weight, present in four of the subjects, tended to be associated with the presence of heterotopias. Similarly, in a recent large-scale study, Wegiel et al. (2010) reported a wide variety of dysgenic lesions and heterotopias in multiple cortical regions in 12 of 13 subjects with ASD. These included excessive ongoing neurogenesis (evidenced by a thickened subependymal layer or subependymal nodular dysplasia), subcortical and periventricular heterotopias, as well as other disruptions of the cytoarchitecture. Differences in neuronal morphology have also been noted in the cortex. Hutsler and Zhang (2010), counting dendritic spines on individual cortical pyramidal neurons, found increased spine densities in the temporal lobes of individuals with ASD and intellectual disability relative to age-matched controls. ASD subjects with either mild or no intellectual disability were similar to controls.

Recent studies have also found subtle but pervasive microstructural abnormalities in cortical architecture, even in the absence of more obvious dysgenic lesions. Although most of these analyses are not rigorously stereologic, the impression is of increased numbers of narrow minicolumns containing increased densities of neurons (Buxhoeveden et al. 2006; Casanova et al. 2002, 2006) in the frontal cortex of ASD brains. This trend appears to be most pronounced in the frontal lobe, particularly the dorsolateral prefrontal cortex, and is not seen in more posterior regions such as the visual cortex. Minicolumns are the vertical cell columns created by sequential waves of migrating neurons travelling along radial glial fibers during early corticogenesis. Increased numbers of such arrays may reflect excess early divisions of radial glial cells immediately prior to the onset of neurogenesis and migration. Furthermore, the distribution of minicolumn

abnormalities correlates with patterns of accelerated growth in the early postnatal period (i.e., frontal > posterior) and, so far, the excess neurons reported in the prefrontal cortex of young males with autism (Courchesne et al. 2011). Again, while probably not detectable at birth, the presence of even a small proportion of excess neurons could conceivably cause rapid postnatal growth in frontal regions.

In a recent study, (Zikopoulos and Barbas 2010) stereologically investigated the fine structure by light and electron microscopy (EM) of myelinated axons in the white matter below the ACC, orbitofrontal cortex (OFC), and lateral prefrontal cortex (LPFC). They found similar overall axonal density between normal and ASD groups below all prefrontal areas. However, the ASD group had significantly fewer large axons (many presumably representing long-range corticocortical connections) in the deep white matter below the ACC associated with a significantly greater density of smaller axons (thought to connect adjacent cortical areas) in the corresponding superficial white matter. There were no discernable differences in neuronal densities or distributions in the overlying gray matter. The white matter below ACC also exhibited a significantly higher proportion of branched axons of medium caliber. This corresponded to higher levels of GAP-43 expression. Axons in the superficial white matter below the OFC exhibited significantly thinner myelin sheaths when controlled for axon diameter despite similar numbers of oligodendrocytes (the glia responsible for myelination).

Limbic System

As in the ACC, Kemper and Bauman (1993) reported smaller, more packed neurons with attenuated dendritic arbors in many other structures of the limbic system (particularly the amygdala, hippocampus, entorhinal cortex, and mammillary bodies) in autistic individuals relative to age-matched controls. In contrast, septal neurons were unusually large in young autistic children but relatively smaller and depleted in numbers in autistic adults. Bailey et al. (1998) found increased cell packing in the hippocampus

of one out of five cases. The findings in the amygdala may correspond to MRI reports of early enlargement of the amygdala; however, (Schumann and Amaral 2006) found a 15% decrease in the number of neurons in the amygdala of adolescents and adults with autism in their stereologic study, thereby indicating the possibility that neuron number is either not responsible for the increase in amygdala size in young autistic children or that significant neuron loss occurs starting in adolescence. Given the trends seen in the septum, the latter may be plausible. Wegiel et al. (2010) additionally found dysplastic laminar irregularities in the hippocampus of four brains, again implicating subtle irregularities in neuronal migration.

Cerebellum

By far, the most consistent finding in neuropathologic studies of postmortem ASD brain samples is a reduction in the density of cerebellar Purkinje neurons of the cerebellar cortex (Bailey et al. 1998; Kemper and Bauman 1993; Palmen et al. 2004, and many others), although there is no discernable clinical correlate of this abnormality. Kemper and Bauman first described a decrease in the number of Purkinje cells in all the autistic brains they analyzed regardless of age. This was particularly marked in the posterior-lateral part of the lateral lobes. The neurons of the deep cerebellar nuclei (the targets of Purkinje cell projections) were abnormally large in younger individuals and abnormally small, and sometimes reduced in number, in older individuals. It must be noted that these studies have not been strictly stereologic and not always replicated. For instance, Fatemi et al. (2002) reported no difference in Purkinje neuron density but did note an approximately 25% reduction in cell size. Whitney et al. (2009) found that only a proportion of their cases of ASD showed reduced Purkinje cell densities as well as decreases in basket and stellate cell densities. Wegiel et al. (2010) reported variable occurrences of cerebellar flocculonodular dysplasia, focal vermal dysplasia, cerebellar hypoplasia, and cerebellar heterotopias, indicating that evidence of altered neuronal migration could also be found in this structure.

Brain Stem

Kemper and Bauman (1993) reported similar alterations in the inferior olive as detected in the septum and deep cerebellar nuclei. Younger ASD brains exhibited unusually large neurons in the inferior olive, whereas in the adult cases, these neurons were small and pale. The overall numbers of inferior olivary neurons were similar to controls in both instances but tended to be concentrated at the nuclear periphery – the part of the complex that projects to the posterior-lateral part of the lateral lobes of the cerebellum. The synaptic relationship between the olivary and Purkinje neurons is established at approximately 28 weeks of gestation and is tight to the point that if Purkinje cells die for some reason, there is retrograde loss of the corresponding olivary neurons. Therefore, they reason, the “loss” of Purkinje neurons in ASD predates the formation of this relationship as olivary neuronal numbers do not appear to be reduced. While the mechanism for this is not understood, the age-related changes in neuronal size in the cerebellar nuclei, inferior olive, and septum are evidence of ongoing alterations in developmental processes, at least before adulthood. Bailey et al. (1998) reported olivary dysplasia or ectopic olivary neurons in five cases of ASD.

Future Directions

In conclusion, relatively consistent neuropathologic observations in autism have included early increased brain size associated with increased numbers of prefrontal neurons, more densely packed smaller neurons in the limbic system, decreased numbers of cerebellar Purkinje neurons, and features of neuronal lamination/migration disturbances – all of which may be traced to prenatal disturbances in brain development. The identification of substantial trends is actually quite remarkable given the staggering heterogeneity in core and comorbid clinical features of ASD and demonstrates the great promise neuropathologic studies hold for identifying the elusive developmental neurobiological underpinnings of altered connectivity in ASD.

For this promise to be fulfilled, future studies will have to be increasingly systematic – employing rigorous stereologic and molecular neuropathologic methods, e.g., large-scale as well as single-cell gene and protein expression analyses. This will require larger samples of ASD brain tissue, derived from a broader age range, in order to provide the necessary statistical power to contend with distinct clinical and genetic subphenotypes as well as dynamic changes observed over the developmental lifespan by structural MRI. Fortunately, significant trends toward this goal are already evident in recent literature, fueled by noteworthy improvements in the availability and quality of ASD postmortem brain tissue through efforts such as the Autism Tissue Project.

See Also

- ▶ [Amygdala](#)
- ▶ [Cerebellar Abnormalities in Autism](#)
- ▶ [Corpus Callosum Abnormalities in Autism](#)
- ▶ [Prefrontal Cortex](#)
- ▶ [Purkinje Cells](#)
- ▶ [Temporal Lobes](#)

References and Reading

- Amaral, D., Schumann, C., & Nordahl, C. (2008). Neuroanatomy of autism. *Trends in Neurosciences*, 31, 137–145.
- Avino, T., & Hutsler, J. (2011). Abnormal cell patterning at the cortical gray-white boundary in autism spectrum disorders. *Brain Research*, 1360, 138–146.
- Bailey, A., Luthert, P., Dean, A., Harding, B., Janota, I., Montgomery, M., Rutter, M., & Lantos, P. (1998). A clinicopathological study of autism. *Brain*, 121, 889–905.
- Bauman, M., & Kemper, T. (1985). Histoanatomic observations of the brain in early infantile autism. *Neurology*, 35, 866–867.
- Buxhoeveden, D. P., Semendeferi, K., Buckwalter, J., Schenker, N., Switzer, R., & Courchesne, E. (2006). Reduced minicolumns in the frontal cortex of patients with autism. *Neuropathology and Applied Neurobiology*, 32(5), 483–491.
- Casanova, M., Buxhoeveden, D., Switala, A., & Roy, E. (2002). Minicolumnar pathology in autism. *Neurology*, 58, 428–432.

- Casanova, M. F., van Kooten, I. A. J., Switala, A. E., van Engeland, H., Heinsen, H., Steinbusch, H. W. M., et al. (2006). Minicolumnar abnormalities in autism. *Acta Neuropathologica*, 112, 287–303.
- Courchesne, E., Mouton, P. R., Clahoun, M. E., Semendeferi, k., Ahrens-Barbeau, C., Hallet, M. J., Barnes, C. C., & Pierce, K. (2011). Neuron number and size in prefrontal cortex of children with autism. *Journal of the American Medical Association*, 9, 2001–2010.
- Fatemi, S. H., Halt, A. R., Stry, J. M., Kanodia, R., Schulz, S. C., & Realmuto, G. R. (2002). Glutamic acid decarboxylase 65 and 67 kDa proteins are reduced in autistic parietal and cerebellar cortices. *Biological Psychiatry*, 52, 805–810.
- Herbert, M. R., Ziegler, D. A., Makris, N., Filipek, P. A., Kemper, T. L., Normandin, J. J., et al. (2004). Localization of white matter volume increase in autism and developmental language disorder. *Annals of Neurology*, 55(4), 530–540.
- Hutsler, J. J., & Zhang, H. (2010). Increased dendritic spine densities on cortical projection neurons in autism spectrum disorders. *Brain Research*, 1309, 83–94.
- Kemper, T., & Bauman, M. (1993). The contribution of neuropathologic studies to the understanding of autism. *Behavioral Neurology*, 11, 175–187.
- Kemper, T., & Bauman, M. (2002). Neuropathology of infantile autism. *Molecular Psychiatry*, 7, S12–S13.
- Lainhart, J. E., Bigler, E. D., Bocian, M., Coon, H., Dinh, E., Dawson, G., et al. (2006). Head circumference and height in autism: A study by the collaborative program of excellence in autism. *American Journal of Medical Genetics Part A*, 140A(21), 2257–2274.
- Palmen, S. J. M. C., van Engeland, H., Hof, P. R., & Schmitz, C. (2004). Neuropathological findings in autism. *Brain*, 127(12), 2572–2583.
- Schmitz, C., & Rezaie, P. (2008). The neuropathology of autism: Where do we stand? *Neuropathology and Applied Neurobiology*, 34, 4–11.
- Schumann, C. M., & Amaral, D. G. (2006). Stereological analysis of amygdala neuron number in autism. *The Journal of Neuroscience*, 26(29), 7674–7679. <https://doi.org/10.1523/JNEUROSCI.1285-06.2006>.
- Schumann, C., & Nordahl, C. (2011). Bridging the gap between MRI and postmortem research in autism. *Brain Research*, 1380, 175–186.
- Simms, M., Kemper, T., Timbie, C., Bauman, M., & Blatt, G. (2009). The anterior cingulate cortex in autism: Heterogeneity of qualitative and quantitative cytoarchitectonic features suggests possible subgroups. *Acta Neuropathologica*, 118, 673–684.
- van Kooten, I. A. J., Palmen, S. J. M. C., von Cappeln, P., Steinbusch, H. W. M., Korr, H., Heinsen, H., et al. (2008). Neurons in the fusiform gyrus are fewer and smaller in autism. *Brain*, 131(4), 987–999.
- Wegiel, J., Kuchna, I., Krzysztof Nowicki, K., Imaki, H., Jarek Wegiel, J., Elaine Marchi, E., et al. (2010). The neuropathology of autism: Defects of neurogenesis and neuronal migration, and dysplastic changes. *Acta Neuropathologica*, 119, 755–770.
- Whitney, E. R., Kemper, T. L., Rosene, D. L., Bauman, M. L., & Blatt, G. J. (2009). Density of cerebellar basket and stellate cells in autism: Evidence for a late developmental loss of Purkinje cells. *Journal of Neuroscience Research*, 87(10), 2245–2254.
- Zikopoulos, B., & Barbas, H. (2010). Changes in prefrontal axons may disrupt the network in autism. *The Journal of Neuroscience*, 30(44), 14595–14609.

Neurophysiology

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Synonyms

Electrophysiology; Psychophysiology

Structure

Neural Structures Implicated Through Neurophysiological Assessment in ASD

The study of neurophysiology in ASD has implicated specific brain structures, such as the superior temporal sulcus in association with processing biological movement, the fusiform gyrus in relation to face processing, the anterior cingulate cortex in relation to joint attention abilities, and the cerebellum in attention and engagement, among others. Neurophysiological studies have also implicated brain regions such as the limbic system, including the amygdala and hippocampus in relation to affective processing and memory, the mirror neuron system in relation to action understanding and imitation, and the ventromedial prefrontal cortex in association with stimulus-reward associations.

Function

Neurophysiological Tools and Measurements

Neurophysiology concerns the study of the functions of the nervous system. Neurophysiological measures are utilized to examine both brain

structures and neural pathways. Methods such as electroencephalography, electromyography, and assessment of skin conductance provide information about various aspects of the nervous system including sensory, motor, and cognitive functioning. Neurophysiological studies provide important information about the connections between brain structures and systems as well as what areas and networks of the brain may be impacted in individuals with disabilities such as autism spectrum disorder.

Electroencephalography (EEG)

One of the primary methods for studying neural pathways in the brain is electroencephalography (EEG), which is the measurement of electrical activity produced by the brain. EEG is recorded from the scalp and is a noninvasive method of measuring postsynaptic neuronal activity. EEG recordings reflect the propagation of electrical activity to the scalp from the synchronous activation of a specific population of neurons.

EEG can provide important information about the functioning of neural networks. EEG oscillatory activity, measured by the frequency and amplitude of synchronized neural activity, reveals information about the state of the brain. Evoke potentials (EPs) and event-related potentials (ERPs) are the result of averaging EEG activity to examine time-locked responses to the presentation of a stimulus (visual, somatosensory, or auditory). EEG coherence refers to the functional connectivity of neural networks measured by the relation of signals over distal neuronal regions.

EEG is a noninvasive technique in which the individual wears an electrode net on the scalp while observing/listening to stimuli or simply resting. EEG has excellent temporal resolution but relatively poorer spatial resolution compared to other imaging modalities. EEG studies of individuals with ASD allow us to better understand the timing of brain functioning, alterations in resting and active brain states, and potential under- and overconnectivity of the brain. EEG studies can be designed such that individuals do not need to make a behavioral, motor, or vocal response, allowing for the use of the method across age groups, cognitive and verbal functioning, and the autism spectrum. In addition, it has

the added benefit of being able to examine neuronal processing over time to better understand the stages of processing of a phenomenon.

Electrooculogram (EOG)

By recording activity from electrodes placed around the eye, eye movements can be carefully characterized and studied. Eye movement studies provide information about the cortical and subcortical regions of the brain that control eye movement activity. For instance, studies that examine saccadic (e.g., fast) eye movements are able to assess frontal brain regions as well as the brain stem and cerebellum, whereas voluntary eye movements and pursuit eye movements (used for visual tracking) can determine if anterior and posterior regions are involved. These basic visual processes can also be studied using eye tracking.

Electromyography (EMG)

EMG is a noninvasive technique used to measure and record the electrical activity of skeletal muscles. This has implications for neurophysiological functioning through the use of experimental paradigms. Fear potential startle response (FPS) is a noninvasive psychophysiological methodology that has been widely utilized in the non-ASD population to facilitate better understanding of amygdala functioning. In FPS, a sudden onset of an acoustic, visual, or tactile stimulus elicits a startle response. Acoustic startle amplitude can be enhanced by exposure to a conditioned stimulus that was paired with an aversive stimulus. This conditioned stimulus or signal produces a state of fear that potentiates the startle response and increases reflexive behavior. The threat cue primes an individual for response, creating a fear state, manifested by an exaggerated startle reflex to any suddenly imposed stimulus (Lang et al. 2000). The startle response is characterized by a fast series of muscle contractions around the head, neck, and shoulders; the first, fastest, and most stable component in this reaction is a sudden closure of the eyelid (Minshew et al. 1999). Intensity of the startle response is measured by electromyographic activity of the orbicularis muscle during the eyeblink, or blink magnitude (Lang et al. 1990).

Skin Conductance

The assessment of the electrical conductance of the skin, which varies with its moisture level, is called skin conductance or the galvanic skin response (GSR). The measurement of GSR provides insight into physiological arousal levels and the sympathetic nervous system as the sweat glands are controlled by the sympathetic nervous system.

Electrocardiography (ECG)

ECG is the measurement of heart rate and blood pressure.

Magnetoencephalography (MEG)

MEG is a method of recording neural activity by recording magnetic fields produced by electrical currents in the brain. MEG records neuronal activity directly and has high temporal (<1 ms) and spatial resolution (1–5 mm), making it a useful tool for neurophysiological research. MEG can be used to examine temporal correlations or coherence within a particular brain region or across regions as well as auditory processing deficits in ASD.

Pathophysiology

Processes Elucidated Through Neurophysiological Assessment in ASD

Visual Processing

Individuals with ASD have often been described as having altered perceptual processing. An ERP study of children with ASD found a faster early visual component (C1) (thought to be generated by the primary visual cortex) to simple sinusoidal luminance patterns compared to typically matched controls (Milne and Scope 2008). This is thought to reflect faster visual perceptual detection in individuals with ASD, suggesting that activity in the primary visual cortex may be enhanced compared to typically developing individuals. However, individuals with ASD may demonstrate perceptual impairment in extrastriate cortex (V2). Studies have found that individuals with ASD show decreased specialized processing

of visual frequencies, decreased extrastriate activity, and reduced perceptual boundary detection (Vandenbroucke et al. 2008; Boeschoten et al. 2007). Bertone et al. (2005) suggest that individuals with ASD have difficulties with integration of visual information.

Eye movement studies have found that children with ASD make more saccades in passive viewing tasks than typically developing children (Kemner et al. 1998). Minshew et al. (1999) found that adults with ASD had significant abnormalities in volitional eye movements, indicating frontal dysfunction rather than cerebellum dysfunction since visually guided saccades (which are governed by the cerebellum) were normal. These findings suggest that the basic attentional and sensorimotor system may be intact in ASD and that difficulties in attention shifting may be due to frontal dysfunction and executive functioning abilities. Studies of pursuit eye movements, which rely on multiple brain systems, provide insight into functional connectivity in ASD. A study of pursuit eye movements found bilateral pursuit deficits in individuals with ASD, suggesting that there may be abnormalities in connectivity in the sensorimotor domain possibly due to abnormal brain maturation (Takarae et al. 2004).

Face Processing

ERPs can be used to better understand both low-level perceptual processing of faces as well as more complex cognitive processing. Young children with ASD show delayed social processing of faces but intact processing of objects. Webb et al. (2010) found that 18–30-month-old children with ASD had ERP responses to familiar and unfamiliar faces that were more similar to younger neurotypical children; the authors suggested that ERPs could be used to document the nature of delays in basic social processing. This response changes in development similar to neurotypicals, but may continue to be delayed. Electrophysiological studies have also found altered patterns of early stage components when adolescents and adults with ASD process faces such as a slowed N170 (an ERP component that is evoked to faces in typically developing individuals) (McPartland

et al. 2004; O'Connor et al. 2007), but not when the eye region was cued before stimulus onset (Webb et al. 2010). Overall, ERP studies of individuals with ASD suggest both abnormal processing speed and cortical organization for processing faces both in early and later stage processing components, but mixed results in adulthood when compensatory strategies may allow more normative brain activation.

Facial Emotion Processing

ERPs allow examination of the neural network underlying emotional processing by measuring brain responses to emotionally salient stimuli. ERP studies of younger children with ASD suggest delayed processing speed and atypical recruitment of cortical areas for processing facial emotion. Dawson, Webb, & Carver, Panagiotides, and McPartland (Dawson et al. 2004) found that 3–4-year-old children with ASD showed a slower early brain response to fear faces and failed to show a larger amplitude, negative slow wave response to fearful faces seen in typically developing children. In addition, children with ASD who exhibited a faster early component processing speed demonstrated better social attention, suggesting a relationship between speed of processing emotional stimuli and social attention impairments in children with ASD. This finding suggests early ASD is associated with abnormal processing of facial expressions of emotion and that these abnormalities begin at early stages of processing. In older children, differential processing of emotions via ERPs has not shown pervasive impairments in ASD. Wong et al. (2008) found normal patterns of ERP and behavioral responses to emotional expressions. However, using source localization, the children with ASD showed slower and weaker responses to emotional expressions in regions responsible for face perception and emotion processing (Wong et al. 2008).

Connectivity

Theories of ASD have conceptualized the disorder as an impairment in connections among neural systems (Belmonte et al. 2004; Rippon et al. 2007; Courchesne and Pierce 2005; Just et al. 2004).

EEG measures of coherence assess communication across neural populations by examining spontaneous activity across predefined frequency bands that represent the speed of neural oscillations; these frequencies include delta (< 4 Hz), theta (4–8 Hz), alpha (8–12 Hz), beta 1 (12–20 Hz), beta 2 (20–30 Hz), and gamma (30–80 Hz). An examination of cortical connectivity in adults with autism found globally reduced EEG coherence in the alpha range in the frontal lobe but increases in the theta range in the temporal region (Murias et al. 2007). This finding suggests that there is overconnectivity in the frontal region but that the frontal lobe has weak functional connections with the rest of the cortex. EEG coherence studies in children with ASD report decreased coherences in delta, theta, and alpha ranges, suggesting underconnectivity (Coben et al. 2008; Isler et al. 2010).

MEG studies of connectivity have found mixed results, with some studies finding decreased connectivity in individuals with ASD (Muñoz-Yunta et al. 2008; Rojas et al. 2008) and another finding no differences between individuals with ASD and a control group in a simple evoked-response task (Coskun et al. 2009). Meanwhile, a MEG study examining coherence during an executive functioning task found decreased synchronization in the prefrontal cortex in the ASD group but increased synchronization in the parietal cortex (Velazquez et al. 2009). These findings provide support for abnormal functional connectivity with evidence supporting both a decreased or increased connectivity theory of ASD.

Imitation

The assessment of power in the mu frequency range has been used as a neurophysiological examination of imitation. The EEG mu rhythm is typically defined as brain activity oscillating in the 8–13-Hz frequency range recorded via centrally located scalp electrodes. When an individual is at rest, the underlying cell assemblies fire synchronously, but when an individual executes and, importantly, observes an action, the cell assemblies activate which attenuates the mu rhythm amplitude. As a result of this consistent

finding, it has been suggested that mu rhythm attenuation reflects activity of the mirror neuron system (Pineda 2005). Given the proposed role of the mirror neuron system in the neurobiology of imitation (Iacoboni 2009), examination of the EEG mu rhythm provides a noninvasive assessment of neurological activity playing a role in imitative ability. An EEG assessment imitation in adults with autism found that individuals with autism showed intact mu rhythm functioning (attenuation of the EEG mu rhythm) when executing movement but atypical activity (reduced attenuation) when observing actions compared to typical adults (Bernier et al. 2007). Further, imitation skills were associated with more attenuation when observing actions for both individuals with autism and typical individuals, suggesting that the neurological circuitry reflected in the mu rhythm play a significant role in imitative ability.

Attention and Disengagement

A study examining attention processing found that when task load was increased, children and adolescents with ASD showed increased early responses to unattended (visual) probes and failed to show normal reduction of processing to attended (auditory) probes. Additional studies have found that children with ASD demonstrate reduced attention ERP components when attending to targets in the periphery (Kemner et al. 1999; Townsend et al. 2001; Verbaten et al. 1991). These studies suggest that individuals with ASD may have subtle impairments in the integrative stages of attentional allocation.

Neurophysiological studies of attentional disengagement have found abnormalities of disengagement in adults with ASD (Kawakubo et al. 2007) and children with ASD (Kikuchi et al. 2010). In this study, typically developing children exhibited a larger saccade-related ERP components when viewing faces compared to children with ASD, whereas disengagement from objects did not differ between individuals with ASD and controls. These results support previous results of face processing deficits in ASD and suggest that disengagement from faces may require a higher level of cortical recruitment than disengagement from objects in individuals with ASD.

Auditory Processing

Neurophysiological methods provide insights into early lexical and semantic processing deficits observed in individuals with ASD. An auditory component (P450) was reduced in individuals with ASD and also was associated with increased EEG gamma power, which the authors suggested was indicative of abnormal inhibitory control (Orekhova et al. 2008). A study of brainstorm responses to speech syllables found that individuals with autism had deficient pitch tracking, suggesting subcortical abnormalities contributing to prosody-encoding deficits (Russo et al. 2008).

ERP studies have found abnormalities in neural processing of semantic information in children with ASD. Dunn and colleagues found that children with ASD failed to elicit an electrophysiological component (N400) indicative of detection of semantic incongruency (e.g., a mismatch) between animal and nonanimal words suggestive of semantic classification impairments (Dunn and Bates 2005; Dunn et al. 1999). Another ERP study aimed to examine if this deficit was limited to the verbal domain and found that high functioning children with ASD showed no electrophysiological index of the detection of semantic incongruence (i.e., no N400 effect) in response to words that mismatched the pictures; participants did show this N400 effect for nonverbal, environmental sounds. These results suggest a semantic abnormality specific to the verbal domain (McCleery et al. 2009). Other ERP studies report decreased component amplitude in response to repetitive speech, but not to repetitive nonspeech, and decreased orienting to novel tones in the sequence of speech sounds, but not in a sequence of tones (Whitehouse and Bishop 2008). These results suggest that inhibition occurs in individuals with ASD to repeated streams of speech.

MEG studies have also proved useful in understanding auditory and language processing difficulties in AS. A magnetoencephalography (MEG) study found an electrophysiological signature in detecting auditory processing deficits in children who have ASD and other language disorders (Roberts et al. 2008). In addition, MEG studies assessing auditory cortex functioning have found that children with ASD have reduced gamma

band power (Rojas et al. 2008) and abnormal left-hemispheric gamma oscillations (Wilson et al. 2007).

Executive Functioning

Neurophysiological studies have also uncovered underlying neural abnormalities to executive functioning deficits often observed in individuals with ASD such as perseverative responding, stereotyped repetitive behaviors, and an inability to accurately monitor ongoing behavior. Henderson et al. (2006) found that highly verbal children with ASD displayed reduced error-related negativity (ERN) amplitudes in response to an executive function task than control children, consistent with difficulties in self-monitoring ongoing behavior in social settings. A study examining response monitoring found that adults with ASD showed reduced error-related negativity and positivity amplitudes and reduced rostral anterior cingulate cortex (ACC) activation compared with controls and that reduced ACC activation was associated with increased social impairments and autism symptoms (Santesso et al. 2011). The authors suggest that the anterior cingulate cortex may be implicated in the social impairments observed in ASD. Another study examining error monitoring also found reduced ERN localized in the ACC (Sokhadze et al. 2010). Finally, an ERP study found a reduced error-related negativity and positivity to an auditory task, indicating an insensitivity to detect the possibility of errors (Vlamings et al. 2008). Together, these results suggest general impairment in self-monitoring and other executive functioning abilities underlying social and behavioral difficulties in ASD.

Reward Processing

Researchers have suggested that individuals with ASD have reduced social motivation such that they do not properly anticipate and appreciate the pleasure of social stimuli. Behavioral studies have also shown that children with ASD have difficulty learning stimulus-reward associations for both social and nonsocial rewards (Dawson et al. 1998). Event-related brain potential (ERP) studies aimed at assessing reward anticipation

(initiated by cue signals) and a potential rewarding outcome employed a modified go/no go paradigm. During this paradigm, a salient, motivating stimulus elicits a larger amplitude in the ERP component, P3. An ERP study of reward incentive found that children with ASD did not exhibit a bigger P3 to reward compared to nonreward stimuli, irrespective of reward type (social vs monetary) (Kohls et al. 2011). These results suggest that individuals with ASD have an atypical motivation-related brain response to reward cues that may be suggestive of a general reward processing deficit rather than social reward dysfunction. No behavioral differences were found in this study, suggesting that neurophysiological methods may be more sensitive than behavioral measures at identifying abnormal reward processing.

Amygdala Functioning

Atypical amygdala function has been implicated in the development and maintenance of emotional and social impairments in ASD. The amygdala and its interconnections with other brain regions play a crucial role in perceiving emotional stimuli, appraising novel situations, detecting danger, and modulating fear response. Amygdala function can be assessed via a fear conditioning paradigm known as fear potentiated startle (FPS). Thus far, findings fail to find differences in potentiated startle response in individuals with ASD compared to control groups (Bernier et al. 2005; Salmond et al. 2003), despite strong implications for amygdala dysfunction in the disorder.

See Also

- ▶ Amygdala
- ▶ Auditory Potentials
- ▶ Brainstem Auditory Evoked Potentials
- ▶ Electroencephalogram (EEG)
- ▶ Event-Related Potential
- ▶ Evoked Potentials
- ▶ Eye Gaze
- ▶ Face Perception
- ▶ Functional Connectivity
- ▶ Limbic System

- ▶ Mirror Neuron System
- ▶ Mu Rhythm
- ▶ Neuroanatomy
- ▶ Neuropathology
- ▶ Visual Evoked Potential (VEP)

References and Reading

- Belmonte, M. K., Cook, E. H., Jr., Anderson, G. M., Rubenstein, J. L., Greenough, W. T., Beckel-Mitchener, A., et al. (2004). Autism as a disorder of neural information processing: Directions for research and targets for therapy. *Molecular Psychiatry*, 9, 646–663.
- Bernier, R., Dawson, G., Panagiotides, H., & Webb, S. (2005). Individuals with autism spectrum disorder show normal responses to a fear potential startle paradigm. *Journal of Autism and Developmental Disorders*, 35, 575–583.
- Bernier, R., Dawson, G., Webb, S., & Murias, M. (2007). EEG mu rhythm and imitation impairments in individuals with autism spectrum disorder. *Brain and Cognition*, 64, 228–237.
- Bertone, A., Mottron, L., Jelenic, P., & Faubert, J. (2005). Enhanced and diminished visuo-spatial information processing in autism depends on stimulus complexity. *Brain: A Journal of Neurology*, 128(10), 2430–2441.
- Boeschoten, M. A., Kenemans, J. L., van Engeland, H. H., & Kemner, C. C. (2007). Abnormal spatial frequency processing in high-functioning children with pervasive developmental disorder (PDD). *Clinical Neurophysiology*, 118(9), 2076–2088.
- Coben, R., Clarke, A. R., Hudspeth, W., & Barry, R. J. (2008). EEG power and coherence in autistic spectrum disorder. *Clinical Neurophysiology*, 119(5), 1002–1009.
- Coskun, M., Varghese, L., Reddoch, S., Castillo, E. M., Pearson, D. A., Loveland, K. A., & Sheth, B. R. (2009). Increased response variability in autistic brains? *Neuroreport: For Rapid Communication of Neuroscience Research*, 20(17), 1543–1548.
- Courchesne, E., & Pierce, K. (2005). Why the frontal cortex in autism might be talking only to itself: Local over-connectivity but long-distance disconnection. *Current Opinion in Neurobiology*, 15(2), 225–230.
- Dawson, G., Meltzoff, A. N., Osterling, J., Rinaldi, J., & Brown, E. (1998). Children with autism fail to orient to naturally occurring social stimuli. *Journal of Autism and Developmental Disorders*, 28(6), 479–485.
- Dawson, G., Webb, S., Carver, L., Panagiotides, H., & McPartland, J. (2004). Young children with autism show atypical brain responses to fearful versus neutral facial expressions of emotion. *Developmental Science*, 7(3), 340–349.
- Dunn, M., & Bates, J. (2005). Developmental change in neutral processing of words by children with autism. *Journal of Autism and Developmental Disorders*, 35(3), 361–376.
- Dunn, M., Vaughan, H. R., Kreuzer, J., & Kurtzberg, D. (1999). Electrophysiologic correlates of semantic classification in autistic and normal children. *Developmental Neuropsychology*, 16(1), 79–99.
- Groen, W. B., Zwiens, M. P., van der Gaag, R. J., & Buitelaar, J. K. (2008). The phenotype and neural correlates of language in autism: An integrative review. *Neuroscience and Biobehavioral Reviews*, 32, 1416–1425.
- Haeson, B., Boets, B., & Wagemans, J. (2011). A review of behavioural and electrophysiological studies on auditory processing and speech perception in autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5, 701–714.
- Henderson, H., Schwartz, C., Mundy, P., Burnette, C., Sutton, S., Zahka, N., et al. (2006). Response monitoring, the error-related negativity, and differences in social behavior in autism. *Brain and Cognition*, 61, 96–109.
- Iacoboni, M. (2009). Neurobiology of imitation. *Current Opinion in Neurobiology*, 19, 661–665.
- Isler, J. R., Martien, K. M., Grieve, P. G., Stark, R. I., & Herbert, M. R. (2010). Reduced functional connectivity in visual evoked potentials in children with autism spectrum disorder. *Clinical Neurophysiology*, 121(12), 2035–2043.
- Just, M., Cherkassky, V. L., Keller, T. A., & Minshew, N. J. (2004). Cortical activation and synchronization during sentence comprehension in high-functioning autism: Evidence of underconnectivity. *Brain: A Journal of Neurology*, 127(8), 1811–1821.
- Kawakubo, Y., Kasai, K., Okazaki, S., Hosokawa-Kakurai, M., Watanabe, K., Kuwabara, H., & Maekawa, H. (2007). Electrophysiological abnormalities of spatial attention in adults with autism during the gap overlap task. *Clinical Neurophysiology*, 118(7), 1464–1471.
- Kemner, C., Verbaten, M. N., Koelega, H. S., Camfferman, G. G., & van Engeland, H. H. (1998). Are abnormal event-related potentials specific to children with ADHD? A comparison with two clinical groups. *Perceptual and Motor Skills*, 87(3), 1083–1090.
- Kemner, C., van der Gaag, R., Verbaten, M., & van Engeland, H. (1999). ERP differences among subtypes of pervasive developmental disorders. *Biological Psychiatry*, 46(6), 781–789.
- Kikuchi, Y., Senju, A., Akechi, H., Tojo, Y., Osanai, H., & Hasegawa, T. (2010). Atypical disengagement from faces and its modulation by the control of eye fixation in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41(5), 629–645.
- Kohls, G., Peltzer, J., Schulte-Ruther, M., Kamp-Becker, I., Remschmidt, H., Herpertz-Dahlmann, B., et al. (2011). Atypical brain responses to reward cues in autism as revealed by event-related potentials. *Journal of Autism and Developmental Disorders*, 41(11), 1523–1533.

- Kuhl, P. K., Coffey-Corina, S., Padden, D., & Dawson, G. (2005). Links between social and linguistic processing of speech in preschool children with autism: Behavioral and electrophysiological measures. *Developmental Science*, 8(1), F9–F20.
- Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1990). Emotion, attention, and the startle reflex. *Psychological Review*, 97, 377–395.
- Lang, P. J., Davis, M., & Öhman, A. (2000). Fear and anxiety: Animal models and human cognitive psychophysiology. *Journal of Affective Disorders*, 61(3), 137–159.
- McCleery, J. P., Akshoomoff, N., Dobkins, K. R., & Carver, L. J. (2009). Atypical face versus object processing and hemispheric asymmetries in 10-month-old infants at risk for autism. *Biological Psychiatry*, 66(10), 950–957.
- McPartland, J., Dawson, G., Webb, S. J., Panagiotides, H., & Carver, L. J. (2004). Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 45(7), 1235–1245.
- Milne, E., & Scope, A. (2008). Are children with autistic spectrum disorders susceptible to contour illusions? *British Journal of Developmental Psychology*, 26(1), 91–102.
- Minshew, N. J., Luna, B., & Sweeney, J. A. (1999). Oculomotor evidence for neocortical systems but not cerebellar dysfunction in autism. *Neurology*, 52(5), 917–922.
- Minshew, N., Webb, S. J., Williams, D. L., & Dawson, G. (2006). Neuropsychology and neurophysiology of autism spectrum disorders. In S. Moldin & J. Rubenstein (Eds.), *Understanding autism: From basic neuroscience to treatment*. CRC Press.
- Muñoz-Yunta, J. A., Ortiz, T. T., Palau-Baduell, M. M., Martín-Muñoz, L. L., Salvadó-Salvadó, B. B., Valls-Santasusana, A. A., et al. (2008). Magnetoencephalographic pattern of epileptiform activity in children with early-onset autism spectrum disorders. *Clinical Neurophysiology*, 119(3), 626–634.
- Murias, M., Webb, S. J., Greenson, J., & Dawson, G. (2007). Resting state cortical connectivity reflected in EEG coherence in individuals with autism. *Biological Psychiatry*, 62, 270–273.
- O'Connor, K., Hamm, J. P., & Kirk, I. J. (2007). Neurophysiological responses to face, facial regions and objects in adults with Asperger's syndrome: An ERP investigation. *International Journal of Psychophysiology*, 63(3), 283–293.
- Orehova, E., Stroganova, T., Prokofyev, A., Nygren, G., Gillberg, C., & Elam, M. (2008). Sensory gating in young children with autism: Relation to age, IQ, and EEG gamma oscillations. *Neuroscience Letters*, 434, 218–223.
- Pineda, J. (2005). The functional significance of mu rhythms: Translating “seeing” and “hearing” into “doing”. *Brain Research Reviews*, 50, 57–68.
- Rippon, G., Brock, J., Brown, C., & Boucher, J. (2007). Disordered connectivity in the autistic brain: Challenges for the “new psychophysiology”. *International Journal of Psychophysiology*, 63(2), 164–172.
- Roberts, T. P., Schmidt, G. L., Egeth, M., et al. (2008). Electrophysiological signatures: Magnetoencephalographic studies of the neural correlates of language impairment in autism spectrum disorders. *International Journal of Psychophysiology*, 68, 149–160.
- Rojas, D. C., Maharajh, K., Teale, P., & Rogers, S. J. (2008). Reduced neural synchronization of gamma-band MEG oscillations in first-degree relatives of children with autism. *BMC Psychiatry*, 8, 66.
- Russo, N. M., Skoe, E. E., Trommer, B. B., Nicol, T. T., Zecker, S. S., Bradlow, A. A., & Kraus, N. N. (2008). Deficient brainstem encoding of pitch in children with autism spectrum disorders. *Clinical Neurophysiology*, 119(8), 1720–1731.
- Salmond, C. H., de Haan, M. M., Friston, K. J., Gadian, D. G., & Vargha-Khadem, F. F. (2003). Investigating individual differences in brain abnormalities in autism. In U. Frith & E. Hill (Eds.), *Autism: Mind and brain* (pp. 247–265). New York: Oxford University Press.
- Santesso, D. L., Drmic, I. E., Jetha, M. K., Bryson, S. E., Goldberg, J. O., Hall, G. B., & Schmidt, L. A. (2011). An event-related source localization study of response monitoring and social impairments in autism spectrum disorder. *Psychophysiology*, 48(2), 241–251.
- Takarae, Y., Minshew, N. J., Luna, B., Krisky, C. M., & Sweeney, J. A. (2004). Pursuit eye movement deficits in autism. *Brain*, 127, 2584–2594.
- Townsend, J., Westerfield, M., Leaver, E., Makeig, S., Jung, T.-P., Pierce, K., & Courchesne, E. (2001). Event-related brain response abnormalities in autism: Evidence for impaired cerebello-frontal spatial attention networks. *Cognitive Brain Research*, 11(1), 127–145.
- Vandenbroucke, M. W., Scholte, H. S., van Engeland, H., Lamme, V. A., & Kemner, C. (2008). A neural substrate for atypical low-level visual processing in autism spectrum disorder. *Brain*, 131, 1013–1024.
- Velazquez, J., Barcelo, F. F., Hung, Y. Y., Leshchenko, Y. Y., Nenadovic, V. V., Belkas, J. J., & Dominguez, L. (2009). Decreased brain coordinated activity in autism spectrum disorders during executive tasks: Reduced long-range synchronization in the frontoparietal networks. *International Journal of Psychophysiology*, 73(3), 341–349.
- Verbaten, M. N., Roelofs, J. W., van Engeland, H., Kenemans, J. K., & Slangen, J. L. (1991). Abnormal visual event-related potentials of autistic children. *Journal of Autism Developmental Disorders*, 21(4), 449–470.
- Vlamings, P. H., Jonkman, L. M., Hoeksma, M. R., van Engeland, H., & Kemner, C. (2008). Reduced error monitoring in children with autism spectrum disorder: An ERP study. *European Journal of Neuroscience*, 28(2), 399–406.
- Webb, S. J., Bernier, R., Burner, K., & Muriás, M. (2009a). Electrophysiology & Evoked Potentials. In D. Amaral, G. Dawson, & D. Geschwind (Eds.), *Autism Spectrum Disorders*. New York: Oxford University Press.

- Webb, S. J., Faja, S., & Dawson, G. (2009b). Face processing in autism. In A. Calder, G. Rhodes, J. Haxby, & M. Johnson (Eds.), *Handbook of face perception*. New York: Oxford University Press.
- Webb, S. J., Jones, E., Merkle, K., Murias, M., Greenson, J., Richards, T., Aylward, E., & Dawson, G. (2010). Response to familiar faces, newly familiar faces, and novel faces as submitted by ERPs is intact in adults with autism spectrum disorders. *International Journal of Psychophysiology*, 77, 106–117.
- Whitehouse, A. O., & Bishop, D. M. (2008). Do children with autism ‘switch off’ to speech sounds? An investigation using event-related potentials. *Developmental Science*, 11(4), 516–524.
- Wilson, T. W., Rojas, D. C., Reite, M. L., Teale, P. D., & Rogers, S. J. (2007). Children and adolescents with autism exhibit reduced MEG steady-state gamma responses. *Biological Psychiatry*, 62(3), 192–197.
- Wong, T. W., Fung, P. W., Chua, S. E., & McAlonan, G. M. (2008). Abnormal spatiotemporal processing of emotional facial expressions in childhood autism: Dipole source analysis of event-related potentials. *European Journal of Neuroscience*, 28(2), 407–416.

psychology. Neuroscientists work at many levels of the nervous system from the molecular to the neural systems level, and they address many kinds of questions ranging across the molecular, cellular, developmental, structural, functional, evolutionary, computational, and medical aspects of the nervous system. The techniques used by neuroscientists have evolved rapidly, from molecular and cellular studies of individual nerve cells to imaging of sensory and motor tasks in the brain, to computational modeling of neural networks. These technological advances have revolutionized the study of neural systems and their development in the living, thinking, human brain.

See Also

► [Cerebral Cortex](#)

Neuroscience

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Synonyms

[Neural science](#); [Neurobiology](#)

Definition

Neuroscience is the scientific study of the nervous system and the ways in which the nervous system gives rise to behavior and cognition. Neuroscience is developed as a subfield or branch of biology. As such, the term neurobiology has often been used interchangeably with neuroscience. Neuroscience as a field has evolved as a highly interdisciplinary field or science of its own that incorporates techniques and concepts from many distinct fields outside of biology proper including chemistry, computer science, engineering, linguistics, genetics, mathematics, medicine (especially psychiatry), philosophy, physics, and

References and Reading

- Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (2000). *Principles of neural science* (4th ed.). New York: McGraw-Hill.

Neurotic Disorders (DSM-II Terminology)

► [Anxiety Disorders](#)

Neurotransmitter

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Definition

A chemical released by neurons that is involved in the transmission of neural impulses or that affects

the transmission in some way. Neurotransmitters are typically contained in synaptic vesicles and upon neuronal stimulation are released at the terminals of neurons into the synapse or gap between neurons. They then diffuse rapidly across the synapse and interact with receptors on the postsynaptic neuron. Neurotransmitters can also bind to autoreceptors found on the releasing neuron and thereby modulate their own release and synthesis. Major classes of neurotransmitters include acetylcholine; the monoamines, including serotonin, dopamine, norepinephrine, and histamine; the amino acid neurotransmitters, including glutamate and GABA; the neuropeptides; and the gaseous transmitters including nitric oxide, carbon monoxide, and hydrogen sulfide.

See Also

► Serotonin

References and Reading

- Anderson, G. M., & Martin, A. (2007). Neurochemistry, pharmacodynamics and biological psychiatry. In A. Martin (Ed.), *Child and adolescent psychiatry: A comprehensive textbook* (4th ed., pp. 234–249). Baltimore: Williams and Wilkins.
- Heckers, S., Konradi, C., & Anderson, G. M. (2011). Synaptic function and biochemical neuroanatomy. In A. Martin, L. Scahill, & C. J. Kratochvil (Eds.), *Pediatric psychopharmacology: Principles and practice* (pp. 23–37). New York: Oxford University Press.
- Mancuso, C., Navarra, P., & Preziosi, P. (2010). Roles of nitric oxide, carbon monoxide, and hydrogen sulfide in the regulation of the hypothalamic-pituitary-adrenal axis. *Journal of Neurochemistry*, 113(3), 563–575.

Neurotypical

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Synonyms

Control; Normal; Typical; Typically developing

Definition

Description of a medically and psychologically healthy individual demonstrating a normative pattern of neurodevelopment. Typically used specifically in contrast with individuals experiencing an atypical developmental course, such as autism, and often used to describe the control group in clinical research. Its use varies from scientific publications (Cashin 2006) to tongue-in-cheek descriptions highlighting the subjective nature and valenced language often employed in research on social behavior (Larsen 2010).

References and Reading

- Cashin, A. (2006). Two terms—one meaning: The conundrum of contemporary nomenclature in autism ([Case Reports, Review]). *Journal of Child and Adolescent Psychiatric Nursing*, 19(3), 137–144. Official Publication of the Association of Child and Adolescent Psychiatric Nurses, Inc.
- Larsen, A. (2010). *Neurotypical*. From <http://www.neurotypical.com/>

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New Zealand and Autism

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Historical Background

The current autism picture in Aotearoa New Zealand is similar to that of other technologically developed countries in the Western hemisphere. That is, the identification of autism, especially high functioning autism, has skyrocketed in the last 15 years because resources and full enrolment in schools from age 5 have allowed identification to increase. (Aotearoa is the indigenous Maori name for NZ.)

The picture has not always been this way, as NZ did not follow the recognition of autism from

Leo Kanner (1943) or Hans Asperger (1991/1944) until the mid-1960s. At that time, child psychiatrist Mildred Creak visited New Zealand on her way to Perth Australia (Feinstein 2011). She had established the first (9-pt) behavioral observation checklist in London Hospital, and she brought current understanding of autism (and some clinical services) to NZ. This led to the setting up by families of a unit within the IHC (then the Society for Intellectually Handicapped Children).

From these beginnings, also in the 1960s, with international stimulation, New Zealander Marion Bruce founded the Autistic Association (Manson and Stace 2016), now known as Autism New Zealand, a nongovernmental organization with over 6,000 members – families and professionals, in many branches headquartered in Auckland (Autism New Zealand 2017). They provide parent training, supported employment, special tuition, some professional support, and information on “Autism Spectrum Disorders including Asperger’s Syndrome.” Currently they work with limited government funding and their waiting lists are long; for example, they are reluctant to recommend Discrete Trial Training because the waiting list is nearly 2 years (Autism New Zealand 2017). Another NGO, Altogether Autism, was established in 2007 by the Ministry of Health, as a spinoff from the Parent-to-Parent Network. It provides somewhere to go for families with unusual or extreme cases to seek support (Altogether Autism 2017).

The IHC has provided support for the full range of intellectual disabilities in children since 1949 (IHC 2017). It now supports thousands of children and adults. Historically, this support has included individuals with autism, but the contract ended in March 2017, with responsibility transferred to a relatively new provider of individualized supports Explore Specialist Advice (2017). IHC has been notable for its rejection by its founders of large institutions, and of all institutions since the 1950s, thanks to the dedication of Harold and Margaret Anyon, founders of IHC, whose son had an intellectual disability (New Zealand History 2017). In 1959, the NZ branch of the British Medical Association publicly opposed the use of large institutions for those

with intellectual disabilities. Consequently, the IHC received government funding to provide community-based services (New Zealand History 2017). Between 1980 and 2006, all major institutions were phased out.

Legal Issues, Mandates for Service

The Ministries of Health and Education have the possibly awkward joint responsibility for services and support for autism-related conditions. On the positive side, they have commissioned the development of the *New Zealand Autism Spectrum Disorder Guideline* (2008, revised in 2016), following the Curry Report (1998). This document (Ministry of Health 2016) provides evidence-based information concerning people “on the autism spectrum” for families and professionals. A process was set up for regular updates over time.

Since 2014, children and adults with ASD have been eligible for a full range of support services (Ministries of Health and Education 2016). Eligibility and subsequent access to state-funded services are determined by various work groups of the Ministry of Health. Currently, services are available (funded) only for children and adolescents with ASD and for adults who also have an intellectual disability. The recommendation that specialist diagnostic assessments be available for all ages has yet to be acted upon – see the NZ ASD Guideline (Ministry of Health 2016).

Overview of Current Treatments and Centers

Services in New Zealand are provided by the Ministries of Health and Education, related government agencies, and an evolving selection of NGOs. Since the 1980s, NZ government services in general have increasingly been “outsourced,” from the post office and telephone to the road works. Effective models have been difficult to establish, with many large agencies maintaining a monopoly with government control and restrictions on trade. Even so, special needs services for education and mental health have recently

developed considerably, even positively, except not being able to keep up with the demand.

The services required can be considered to fall into fall categories of (1) mental health and education; (2) age groups of preschool, school (ages 5–19), and adulthood including further education; and (3) high functioning versus intellectual disabilities in autism. The Ministry of Health website describes the “autism spectrum disorder” as a range of conditions including “autism and Asperger syndrome.” It notes ASD is thought to affect 1 in 100 New Zealanders; there has been no effort yet to make a systematic count. The website lists sources of support for autism as two other Ministries of (Education; Social Development) and at least seven nongovernment agencies: Autism NZ, Altogether Autism, IDEA Services (IHC), Kid’s Health, Cloud 9 Children’s Foundation, and the Werry Centre. Others are listed in the “[See Also](#)” section below.

The Ministry of Education offers diagnostic referrals for children including preschoolers, or young adults seeking higher education. Clusters of schools employ Resource Teachers for Learning and Behaviour (RTLBs) in school Years 1–10 (5–14 years olds) for children with serious behavioral needs (disabilities) and an extra mandate for Maori and Pasifika. They have struggled since 2000 to provide significant improvement in school performance for their caseload. Schools also have some, but limited, access to psychologists, speech and language therapists, physiotherapists, occupational therapists, and social workers (<http://rtlb.tki.org.nz/>). They also have some funding for teacher aides to improve inclusive schooling. There are limited enrolments in special schools and special classrooms.

Schools cooperate with NGOs who attempt outreach and the provision of services to families. These agencies typically get 10% or less of their funding from the government, relying on grants and charitable donations to make most services free to the public. For example, Autism NZ provides family support, individual training, and advocacy. Their mandate includes preschool, school-age, and adult individuals. For example, they may be the only ones offering training and personnel for supported employment. They are

the largest nongovernment agency specifically for autism in NZ with over 6,000 members and 17 branches. They work directly with families in collaboration with schools and other agencies. Explore Services is a relatively new agency, taking over from IDEA in 2014. They provide “behavior support” for over 4,000 people with disabilities and challenging behavior, nearly 50% with autism, having a mandate both broader and narrower than Autism NZ. They employ a large number of health professionals with sector experience, including a most significant number of psychologists.

Altogether Autism is emerging as a growing resource nationally, based in Hamilton. For example, in 2017 it staged a national conference which attracted a large number of outspoken “Autistics and Aspies,” as well as professionals and family members, and it has joined the Asia-Pacific Autism consortium. That is a recognition of Asia as the fastest growing region in the world, in many respects including the recognition and services for autism. The agency was established in 2007 by the Ministry of Health to provide high quality disability information and advice, especially around specific issues that have eluded others. For this they retain a panel of experts and part-time consultants. The Ongoing Resourcing Scheme has funding for those with the most extreme difficulties (1%). There are several other agencies providing advice and support for autism. These include the Special Education Early Intervention Service with extra support and preschool referrals, and the Children’s Autism Foundation, a charitable trust, providing advice for families with children up to the age of 21, plus other agencies noted elsewhere.

Overview of Research Directions

A modest but increasing amount of research on autism has been achieved in NZ. Based at the University of Auckland, the recently established Minds for Minds Research Network has the subtitle “Unlocking Autism Together” (Virues-Ortega et al. 2017). It includes a major thrust in genetic research and smaller studies in learning, neuropsychology, and speech and language. The

physiological studies, led by Russell Snell, are linking DNA to variations in autism and the functional connections between the stomach and the autistic brain (e.g., Arons et al. 2016). These are large studies, sometimes including dozens of authors, not only from NZ but California and other places.

Recent work is expanding with significant funding for the study of sleep problems in children with autism, following behavioral case studies (e.g., McLay et al. 2017). Significant “small-n” studies in learning, behavior, and speech and language are growing in number (e.g., Smith et al. 2014). These studies, which so far do not include adults, can be expected to generalize worldwide to children with autism and are not specific to NZ.

Overview of Training

Ongoing training of providers (teachers, health care professionals, and others)

Social Policy and Current Controversies

There are always too many controversies surrounding autism, from “something in the drinking water” to “genius unable to express itself.” Currently, they are overshadowed by vaccines as a cause – never mind that the NZ government has prohibited thimerosal. All agencies are outspokenly opposed to the view that Measles-Mumps-Rubella vaccines (free to everyone after the age of 12 months) can cause autism. Recently, the incidence of mumps has doubled and there have been outbreaks of measles, because the national rates of vaccination have been so low and a few individuals have brought these diseases from overseas. Such myths have long been debunked as fraudulent (e.g., Taylor et al. 1999) by Altogether Autism (March 2017) and others, but persist, spread by alarmists and even a popular film (Kohn 2016).

Many other treatments have drifted in from overseas and have minority followings. While

the majority of endorsed autism “treatments” are based in scientific research, historically some of the proposed causes of autism developed from less scientific methods. The theory of “refrigerator mothers” has gone. In the 1950s and 1960s, schizophrenia was often “diagnosed,” despite it usually being presented in late adolescence. Subsequent treatments with little or no empirical support include electric shock treatment (an attempt to stop self-injury), the use of D-lysergic acid diethylamide (LSD), elimination diets, sensory integration therapy, and testosterone treatment.

Training

Universities in NZ offer degree programs and coursework that contribute to professional training. The University of Auckland has an accredited Postgraduate Diploma in Applied Psychology that includes approaches to autism, emphasizing operant methods and some modeling/self-modeling. Canterbury and Victoria Universities are strong through Psychology or Education. To become a special educator or resource teacher requires an initial teaching qualification followed by a postgraduate diploma, available from Massey University campuses or distance education. The Ministries of Health and Education offer printed guidelines and substantial workshops for educators, therapists, and family members.

Other training programs specific to children with autism are mainly provided by various NGOs. For example, Altogether Autism provides a Professional Development Series as in-service training related to the autism spectrum. Autism NZ provides several substantial programs aimed at individuals with autism, educators, professionals, and parents, focused on understanding and practical strategies for quality of life and learning for those with autism. The programs educate on how to build independence, manage inappropriate behavior, and facilitate inclusion. Explore, the new government provider of autism services, is developing an array of training programs for families and professionals.

See Also

► Australia and Autism

References and Reading

- Altogether Autism. (2017). *What is autism?* Retrieved from <https://altogetherautism.org.nz/autism-information/3023/autism-spectrum-disorder-asd/>
- American Psychiatric Association. (2013). *Diagnostic and statistical manual, DSM-5* (5th ed.). Arlington: American Psychiatric Association.
- Arons, M., Lee, K., Thynne, C. J., Kim, S. A., Schob, C., Kindler, S., . . . Garner, C. C. (2016). Shank3 is part of a zinc-sensitive signaling system that regulates excitatory synaptic strength. *Journal of Neuroscience*, 36, 9124–9134.
- Asperger, H. (Trans.). (1991) [1944]. “Autistic psychopathy” in childhood. In U. Frith (Ed.), *Autism and Asperger syndrome* (pp. 37–92). Cambridge: Cambridge University Press.
- Autism New Zealand. (2017). *About Autism New Zealand*. Retrieved from https://www.autismnz.org.nz/about_us
- Curry, D. (1998). *Autism services in New Zealand*. Wellington: Autism Services Project Team.
- Explore Specialist Advice. (2017). *Explore Specialist Advice to provide specialist autism services*. Retrieved from <https://www.healthcarenz.co.nz/why-choose-us/>
- Feinstein, A. (2011). A history of autism: Conversations with the pioneers. *Social History of Medicine*, 24, 847–848. <https://doi.org/10.1093/shm/hkr109>.
- IHC. (2017). *About us*. Retrieved from <https://www.ihc.org.nz/about-us>
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–253.
- Kohn, E. (2016, April). Vaxxed: From cover-up to catastrophe’ is designed to trick you (Review). Indiewire.
- Manson, B., & Stace, H. (2016). A life story – Marion Bruce, Founder of Autism NZ, dies aged 90. Retrieved from <http://www.stuff.co.nz/national/health/77869899/a-life-story%2D%2Dmarion-bruce-founder-of-autism-nz-dies-aged-90>
- McLay, L., France, K., Blampied, N., & Hunter, J. (2017). Using functional behavioral assessment to treat sleep problems in two children with autism and vocal stereotypy. *International Journal of Developmental Disabilities*, on line September 2017. <https://doi.org/10.1080/20473869.2017.1376411>.
- Ministries of Health and Education. (2016). *New Zealand autism spectrum disorder guideline*. Wellington: Ministry of Health.
- New Zealand History. (2017). Foundation of IHC: 25 October 1949. Retrieved from <https://nzhistory.govt.nz/foundation-of-ihc>
- Ongoing Resourcing Scheme. <https://www.education.govt.nz/school/student-support/special-education/ors/>
- Rawdon, P. D. (2012). *Experiences of families of people with Autism Spectrum Disorder in the Canterbury/West Coast area*. Unpublished Master’s thesis, University of Canterbury, Christchurch.
- Smith, J., Hand, L., & Dowrick, P. W. (2014). Video feedforward for rapid learning of a picture-based communication system. *Journal of Autism & Developmental Disabilities*, 44, 926–936. <https://doi.org/10.1007/s10803-013-1946-0>.
- Swarbrick, N. (2017). Care and carers – Care of people with disabilities, Te Ara. Encyclopedia of New Zealand. <http://www.TeAra.govt.nz/en/care-and-carers/page-4>
- Taylor, B., Miller, E., Farrington, C. P., Petropoulos, M. C., Favot-Mayaud, I., Li, J., & Waight, J. A. (1999). Autism and measles, mumps, and rubella vaccine: No epidemiological evidence for a causal association. *Lancet*, 353, 2026–2029.
- Virues-Ortega, J., Lehnert, K., Swan, B., Taylor, M. W., Southee, A., Dougan, D., . . . Jacobsen, J. C. (2017). The New Zealand minds for minds autism spectrum disorder self-reported cohort. *Research in Autism Spectrum Disorders*, 36, 1–7. <https://doi.org/10.1016/j.rasd.2016.21.003>.

References, Readings, Resources (This can include relevant web sites)

- Altogether Autism. <https://altogetherautism.org.nz>
- Autism New Zealand. <https://www.autismnz.org.nz>
- Catalogue of Disability Information and Resources. <http://www.makoa.org/index.htm>
- CCS Disability Action. <http://www.ccsdisabilityaction.org.nz/>
- Creating Success. <http://www.creatingsuccess.co.nz/>
- Disabled Persons Assembly New Zealand. <http://www.dpa.org.nz/>
- Enable New Zealand. <https://www.enable.co.nz/>
- Hohepa. <http://www.hohepa.com/>
- IHC. <https://ihc.org.nz/>
- New Zealand Riding for the Disabled Association. <http://www.rda.org.nz/>
- Parent to Parent. <https://parent2parent.org.nz/>
- Resource Teachers for Learning and Behaviour. <http://rtlb.tki.org.nz/>
- Supporting Weka. <https://firstport.co.nz/>
- TKI: ASD in Education. <http://seonline.tki.org.nz/ASD>

Newson's Syndrome

► Pathological Demand Avoidance (PDA)

Next-Generation Sequencing

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Synonyms

[High-throughput sequencing](#); [Massively parallel sequencing](#); [Second-generation sequencing](#); [Whole-genome sequencing](#)

Definition

Next-generation sequencing (NGS) refers to DNA sequencing methods that have superseded the traditional method of Sanger DNA sequencing. In contrast to the Sanger method of chain termination followed by capillary electrophoresis, NGS technology typically uses sequencing by synthesis coupled with advanced methods to detect nucleotide incorporation. Importantly, NGS allows massive parallelization and efficient use of reagents, thereby drastically reducing cost and increasing throughput. These characteristics are essential for sequencing the many genomes and exomes required for sufficiently powered gene discovery studies in ASD and many other disorders. Over the last 10 years, the cost of sequencing has dropped 1,000,000-fold due to the use of NGS methods; however, Sanger sequencing, due to its high accuracy and independent methodology, is still used to confirm sequences called by NGS methods. In essence, NGS consists of three steps: (1) library preparation, (2) DNA amplification and array of clonal DNA clusters, and (3) massively parallel sequencing by alternating cycles of nucleotide addition and detection by various technologies, including fluorescent imaging by a camera or changes in pH measured by a semiconductor chip.

During library prep, DNA is fragmented and ligated to nonspecific adaptor sequences. Next, DNA amplification is achieved via several methods (such as PCR, using adaptor-specific primers), and,

usually, clusters of clonal DNA are arranged on the sequencing substrate in an array. This process allows for sequencing in a massively parallel fashion by pairing the sequencing signal from a given stretch of DNA with a specific, constant location on the sequencing chip. Since there are millions of clusters arrayed across each sequencing chip, the sequence of millions of fragments is simultaneously determined in parallel (Shendure and Ji 2008).

During sequencing by synthesis, primed templates are serially extended by incorporation of nucleotides, which is generally detected by one of the two methods: fluorescence imaging or voltage monitoring. Fluorescence imaging uses fluorescently marked nucleotides, with a different color of fluorescence for each of the four nucleotides (A, G, C, or T). A digital camera records the color of each cluster of clonal DNA and uses this to determine the base present. The fluorescence is then removed and the next cycle of sequencing begins. Thus, the sequence of nucleotides composing a particular fragment of DNA can be recorded by the color pattern of nucleotide incorporation. Data collection can also be achieved by detecting the changes in pH (via changes in electrical potential) produced by the incorporation of a nucleotide into a strand of DNA. The repeated, sequential addition of the four nucleotides allows for the identification of which base comes next in the sequence, according to which addition produces a decrease in pH (Rothberg et al. 2011).

The data produced by all types of NGS is in the form of short (10–1000 base pair) reads, depending on the NGS technology, instrument, and settings used. The final step, in order to interpret the sequence, necessitates the alignment of each read to the human genome, a process that requires considerable computing power. Each technology has both pros and cons regarding cost, accuracy, speed, and versatility and varies in usage across different research and clinical settings.

See Also

- ▶ [Common Disease-Rare Variant Hypothesis](#)
- ▶ [Copy Number Variation](#)
- ▶ [DNA](#)
- ▶ [Genetics](#)

References and Reading

- Drmanac, R., Sparks, A. B., Callow, M. J., Halpern, A. L., Burns, N. L., Kermani, B. G., et al. (2010). Human genome sequencing using unchained base reads on self-assembling DNA nanoarrays. *Science*, 327(5961), 78–81. <https://doi.org/10.1126/science.1181498>.
- Eid, J., Fehr, A., Gray, J., Luong, K., Lyle, J., Otto, G., et al. (2009). Real-time DNA sequencing from single polymerase molecules. *Science*, 323(5910), 133–138. <https://doi.org/10.1126/science.1162986>.
- Hui, P. (2012). Next generation sequencing: Chemistry, technology and applications. In *Chemical Diagnostics* (pp. 1–18). Berlin/Heidelberg: Springer.
- Quail, M. A., Smith, M., Coupland, P., Otto, T. D., Harris, S. R., Connor, T. R., et al. (2012). A tale of three next generation sequencing platforms: Comparison of ion torrent, Pacific biosciences and Illumina MiSeq sequencers. *BMC Genomics*, 13(1), 341. <https://doi.org/10.1186/1471-2164-13-341>.
- Rothberg, J. M., Hinz, W., Rearick, T. M., Schultz, J., Mileski, W., Davey, M., et al. (2011). An integrated semiconductor device enabling non-optical genome sequencing. *Nature*, 475(7356), 348. <https://doi.org/10.1038/nature10242>.
- Shendure, J., & Ji, H. (2008). Next-generation DNA sequencing. *Nature Biotechnology*, 26(10), 1135–1145. <https://doi.org/10.1038/nbt1486>.

Nexus

- [Local Educational Authority](#)

Nicotine and Autism

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Synonyms

[Alpha-4 beta-2 nicotinic agonists](#); [Alpha-7 nicotinic agonists](#)

Definition

Nicotine binds to nicotinic receptors throughout the brain and the body. Acetylcholine is the naturally occurring molecule that also binds to these receptors. Transdermal nicotine is given to smokers to prevent withdrawal symptoms. Some studies have suggested alterations in nicotine receptors and acetylcholine levels in individuals with autism spectrum disorder. Nicotine has been found to reduce symptoms of aggression in individuals with Alzheimer's disease and schizophrenia in early trials. Nicotine also has anti-aggressive effects in mouse models. A single case report describes improvement in aggression in an adolescent with autism spectrum disorder following nicotine administration (Van Schalkwyk et al. 2015).

References and Reading

- Van Schalkwyk, G. I., Lewis, A. S., Qayyum, Z., Koslosky, K., Picciotto, M. R., & Volkmar, F. R. (2015). Reduction of aggressive episodes after repeated transdermal nicotine administration in a hospitalized adolescent with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(9), 3061–3066.

Nigeria and Autism

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Definition

Nigeria, also known as the Federal Republic of Nigeria, is a country located in the West African subregion bordered by the Republic of Benin in

the West, Chad and Cameroon in the East, and the Niger Republic in the North. The Nigerian coast in the South lies on the Gulf of Guinea on the Atlantic Ocean. Nigeria is a country with over two hundred and fifty ethnic groups, but the three major traditional groups are the Hausa-Fulani, the Yoruba, and the Igbo. Geopolitically, the country consists of 6 regions or zones and 36 states and a Federal Capital Territory located in Abuja.

There is growing momentum recognizing Autism Spectrum Disorder (ASD) globally and advocating for the needs and rights of children in low and middle income countries (LMICs) (Munir et al. 2016). In 2010, there were approximately 52 million people living with ASD worldwide (Elsabbagh et al. 2012; Baxter et al. 2015). Whereas in the United States, ASD is recognized to affect 1 in every 68 children (1 in 42 boys and 1 in 189 girls) (Christensen et al. 2016), the data on prevalence of ASD in LMICs is limited (Bakare et al. 2019). Most research into the epidemiology, causes, clinical presentations, diagnosis, and treatment of ASD is based on studies conducted in high-income countries (HICs). This has led to a misperception that ASD is more prevalent in HICs versus LMICs. For example, while only 20% of the world's current population resides in HICs, 86.5% of all reported cases of ASD have been in HICs (Elsabbagh et al. 2012; Population Reference Bureau 2015). Nigeria is the most populous country in sub-Saharan African with an estimated population of over 180 million people, with children under the age of 15 years accounting for about 35 percent of this population. Barring geographic environmental and genetic variation, there is no reason to expect that prevalence of ASD in LMICs will be lower than that consistently reported in HICs. There is a need to strengthen national capacities in Nigeria in ASD and neurodevelopmental disorders. This chapter will outline the current knowledge of ASD in Nigeria and highlight challenges currently facing the communities as they aim to advocate for services for persons with ASD across the lifespan.

Historical Background

Origins

Following Leo Kanner's pioneering work on ASD dating back to 1943 (Kanner 1943), the first documented cases of ASD in Nigeria were in fact noted in 1972 by Longe and Asuni in a scientific paper read at the Third Pan African Psychiatric Conference in Khartoum, Sudan, where they reported four cases of Infantile Autism among Nigerian children (Longe and Asuni 1972). Subsequently in 1978, Victor Lotter screened children with intellectual disabilities in nine major cities of six African countries that included Ghana, Nigeria, Kenya, Zimbabwe, Zambia, and South Africa. Nine out of 1,312 children with intellectual disabilities studied in these countries met the eligibility criteria to be classified as having ASD, a rate which was 1 in 145 (Lotter 1978). Onuora in 1993 also reported another case of ASD in Nigeria in a case report published in Nigerian Journal of Psychiatry (Onuora 1993). Therefore, with these exceptions, in the years prior to the turn of the twenty-first century, cases of ASD were only sparsely reported in Nigeria or any other sub-Saharan African country.

Emergence of Interest in Knowledge and Awareness

Focus on increasing knowledge and awareness about ASD in Nigeria was heralded by the pioneer work of African Network for the Prevention and Protection against Child Abuse and Neglect (ANPPCAN) Nigeria Chapter in 2007. ANPPCAN Nigeria Chapter in collaboration with the World Bank carried out a survey to ascertain the level of knowledge and awareness of healthcare workers and the general public in Enugu State on ASD in Southeastern Nigeria and implemented a project aimed at increasing the level of knowledge and awareness about ASD through public enlightenment, stake holders' meeting, and a radio program. The report of the survey showed that there is a very low level of knowledge about ASD among the general public and a low to average level of knowledge and

awareness about ASD among various categories of healthcare workers, with relatively highest level of knowledge noted among healthcare workers working in Psychiatric Facilities (ANPPCAN 2007; Bakare et al. 2008). The heightened recognition of ASD can also be attributed to knowledge of elevated prevalence figures recorded in the HICs over the previous two decades among number of investigators in sub-Saharan African region (Fombonne 2003; Bakare et al. 2008).

Since 2007, there has been an increase in the number of research papers on knowledge assessment among Nigerian population, highlighting the clinical characteristics of children with ASD. These have included case reports describing associated co-morbidities and socio-cultural context of ASD among Nigerian children, as well as pockets of prevalence studies among clinical and specialized populations (Bakare and Ikegwuonu 2008; Bakare et al. 2009a; b, 2011a, b, 2012, 2014, 2015, 2016; Igwe et al. 2010, 2011; Bakare and Munir 2011a, b, c; Bello-Mojeed et al. 2011, 2013, 2016, 2017; Bello-Mojeed and Bakare 2013; Ezegwui et al. 2014; Lagunju et al. 2014; Esegibe et al. 2015; Izuwah et al. 2016; Chinawa et al. 2016; Oshodi et al. 2017).

From a public health perspective, the reported overrepresentation of nonverbal cases in clinical populations (Bakare and Munir 2011a) and late identification of ASD (Bello-Mojeed et al. 2011) have emphasized the urgent need for shifting research and public health focus from infectious to Non-Communicable Disease (NCD) priorities, with prioritization of early identification of neurodevelopmental disorders and early interventions for them (Bakare and Munir 2011b; c; Bakare et al. 2014). Unfortunately, the studies on Nigerian adults with ASD have so far been lacking.

Legislative Guidance

At the moment, there is no legislation in Nigeria directed at guiding or mandating services for individuals with ASD specifically. However, the Child Rights Act provides for the rights of children to education, health, and other social

needs. Though this law has been poorly implemented across the nation, Nigeria adopted the Child Rights Act in 2003. The law incorporates the principles of the United Nations Convention on the Rights of the Child, providing a framework for preventing and responding to violence abuse, neglect, and exploitation of children. To date, only 23 of the 36 states in Nigeria have adopted the law, with predominantly Northern Nigerian States Legislative Assemblies yet to accept it (UNICEF Nigeria 2017). In the Nigerian States where the law has been adopted, it has also been poorly implemented, as many cases of violation of children's rights go unreported and unpunished because of reluctance in challenging such cases in courts of law (UNICEF Nigeria 2017; Bakare et al. 2009a).

While the Child Rights Act may address the social and policy needs of children with ASD, adults with ASD in Nigeria may have some relief in the nearest future because of the recent passage and accent to Nigeria Disability Law that is meant to protect the rights of persons with serious neurodevelopmental disabilities (Pulse.ng 2019). The rights of adult with ASD ideally would be taken care of by the Nigeria Disability Law if it is subjected repeatedly to judicial tests to reinforce acceptance of the Law by the general populace. Negative cultural and social issues are among the factors hindering the full implementation of Child Rights Act and may affect acceptance of Nigeria Disability Bill as well. A lot of public health education is still needed to effect change in perception and behavior towards people with disabilities in Nigeria.

Current Treatment and Centers

Treatment and management of any disease condition is often influenced by health behavior of the people, which in turn is a function of attributed etiological explanations. Close to 50% of Nigerian populace, especially in rural communities still attributed the etiology of ASD to spiritual causes (Bakare and Munir 2011a). Hence, the tendency for parents and relatives of children with ASD to seek succor from traditional and religious healers as first point of contact

before seeking help from orthodox practitioners (Bakare and Munir 2011a).

Traditional practice and interventions for ASD in Nigeria are available at Psychiatry and Pediatric facilities of National and States Teaching Hospitals, Specialized Psychiatric Hospitals, and General Hospitals spread across the 36 States of Nigeria and Federal Capital territory in Abuja. Pockets of privately owned Hospitals spread across the country and also provide traditional interventions. Treatments offered in these settings focus largely at alleviating associated problem behaviors and treating medical co-morbidities like seizure disorders and attention deficit hyperactivity disorder symptoms, among other disruptive or injurious behaviors. In view of the fact that majority of cases of ASD presenting to such orthodox practices have tendency for late diagnosis, arising from late or delayed identification (Bakare and Munir 2011a), and the fact that period of onset of ASD in Nigeria and most sub-Saharan African settings coincide with period of neurological infections and complications like cerebral malaria, meningitis, kernicterus among others, cases of ASD seen in Nigerian hospitals and clinics are often in the severe end of the autism spectrum with attendant neurological problems presenting as medical co-morbidities, intellectual disability, and lack of expressive language ability (Bakare and Munir 2011c).

Treatment Centers available in Nigeria that the patients and their relatives present to can be categorized as follows:

Traditional Healers: Based across the country, especially in the rural communities, the traditional healers provide a form of psychological support or comfort that compound the problem of early identification and intervention.

Religious Healers: These include mostly Alfas and Pentecostal Churches that often promise spiritual healing and exorcism. Such centers provide support and reassurance, and false hope, resulting in delaying presentation in health settings in most cases.

Private Homes/Schools and Non-Governmental Organizations (NGOs): Most of these private

homes and schools as well as NGOs found across the country often do not provide services specific to ASD. Most house orphaned and vulnerable children with ASD and intellectual disability. Many provide stimulating play environments and some form of remedial education for the children. The practice of these private homes and schools and the NGOs are not properly regulated, and the local standard of care or interventions they offer may not meet ethical and scientific standards to be considered evidence-base. However, they provide a ray of hope in an environment where there is little or no hope. The services are often expensive and mostly unaffordable for average parents.

Orthodox (Traditional) Medical Practices: These consist of settings like teaching hospitals, specialized psychiatric hospitals, general hospitals, and private hospitals that offer services to children with neurodevelopmental disorders. These practices also provide limited psychological support and address associated problem behaviors and medical co-morbidities.

The core of treatment which is special education that focuses on social stimulation, language development (speech therapy), occupational and physical therapies, and community social inclusion programs are largely lacking in most of these centers. This is an area where future social policies and legislations need to be targeted in the future. Most educational settings in Nigeria are normal main-stream schools designed for normally developing children. Most special schools available, which are quite few and exorbitant in costs, are privately owned. Future direction need to enact a legislation to incorporate special education facilities into normal main-stream schools. This would promote social stimulation for Nigerian children with ASD and promote community inclusion and therefore reduce stigma.

Overview of Research Direction

There is no established or nationally adopted research direction on ASD in Nigeria. However,

suggested research direction based on the present state of ASD in Nigeria should include:

1. Defining the magnitude of the problem of ASD, and needs assessment of affected individuals through a well-implemented nationwide epidemiological study, augmented with genetic and environmental studies.
2. Administrative and research capacity development effort in terms of training for healthcare personnel, promoting human resource development in the area of interventions for ASD and other developmental disability.
3. Establishment of Special Education Departments that should be incorporated into normal mainstream schools to promote social stimulation and community inclusion to reduce stigma. This process would be better enhanced by legislation and policy formulation.
4. Full implementation of existing legislations like Child Rights Act and Nigeria Disability Law. These would promote educational and employment opportunities for people with various disabilities, including ASD. Social policy formulation should also address financial aspect of healthcare and special education provision for individuals with developmental disability since this is not presently covered under the Nigeria National Health Insurance Scheme (NHIS) or any existing social policies.
5. Lastly, research direction should focus on massive public health education to positively influence help seeking behavior for children with ASD and other neurodevelopmental delay, which often leads to late diagnosis and interventions (Bakare et al. 2016).

Social Policies and Current Controversies

Despite the adoption of Child rights Acts in year 2003 that promised equal opportunities to every Nigerian child, the full implementation has experienced clog wheel because not all the Nigerian States, in particular Northern and Southern, have adopted it and those States that have adopted it has had its implementation hindered by a number of political, cultural, and social factors. Many children with ASD and other neurodevelopmental

disabilities are therefore still being locked up in the house and have been prevented by the parents and their relatives from accessing any form of intervention. This is largely because of common derogatory comments and associated stigma of having those children that parents experienced. In addition, health insurance is not available for most parents and where available it does not cover cases like ASD and other developmental disabilities. This exposed parents to out-of-pocket huge cost of care for their children with ASD and other developmental disabilities. So in the cases of ASD in Nigeria, a mixture of stigma and financial burden of care have denied many individuals with ASD the opportunity of any form of intervention, either medical or special education services despite the availability of law that promised equal opportunities for every Nigerian child.

Adults with ASD and intellectual disability are also denied access to their rights. Many lack employment opportunities despite being qualified to take available job spaces due to stigma and discrimination. Unlike the children that have the advantage of Child Rights Act, the Nigeria Disability Law is just freshly enacted and has not gone through the rigor of judicial tests to assess its acceptance by the Nigerian public population.

The factors of poverty and ignorance that promote the culture of wandering and begging children in form of Almajiri system of Islamic education mainly in the Northern Nigeria also promote the culture of labeling children with any form of mental illness or intellectual disability in the southern Nigeria witches, leading to further violation of their rights and other privileges that the Child Rights Act promised every Nigerian Child (Stepping Stones Nigeria 2010; AbdulQadir and Chiranchi 2017).

See Also

- [Autism and the Caribbean](#)
- [Brazil and Autism](#)
- [China and Autism](#)
- [Developing Countries and Autism](#)
- [Egypt and Autism](#)

- Ethiopia and Autism
- India and Autism
- Kuwait and Autism
- Macedonia and Autism
- Pakistan and Autism
- Palestine and Autism
- Singapore and Autism Spectrum Disorder
- Social Class and Autism
- South Africa and Autism
- STEM Education and Autism Spectrum Disorder
- Turkey and Autism

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References and Reading

- AbdulQadir, I. A., & Chiranchi, H. M. D. (2017). The Almajiri system of education in Nigeria Today. <http://www.gamji.com/article5000/NEWS5956.htm>. Accessed 18 Oct 2017.
- African Network for the Prevention and Protection against Child Abuse and Neglect (ANPPCAN), Nigeria Chapter. (2007). Communique on a project to increase the level of awareness on autism and develop a mechanism for care and support of children with autism in Enugu State, South-Eastern Nigeria.
- Bakare, M. O., & Ikegwuonu, N. N. (2008). Childhood autism in a 13 year old boy with oculocutaneous albinism: A case report. *Journal of Medical Case Reports*, 2, 56. <https://doi.org/10.1186/1752-1947-2-56>.
- Bakare, M. O., & Munir, K. M. (2011a). Excess of non-verbal cases of autism spectrum disorders presenting to orthodox clinical practice in Africa – A trend possibly resulting from late diagnosis and intervention. *The South African Journal of Psychiatry*, 17(4), 118–120.
- Bakare, M. O., & Munir, K. M. (2011b). Autism spectrum disorders (ASD) in Africa: A perspective. *African Journal of Psychiatry (Johannesbg)*, 14(3), 208–210. <https://doi.org/10.4314/ajpsy.v14i3.3>.
- Bakare, M. O., & Munir, K. M. (2011c). Autism spectrum disorders in Africa. In M. R. Mohammadi (Ed.), *A comprehensive book on autism spectrum disorders*. ISBN: 978-953-307-494-8, InTech. Available from: <http://www.intechopen.com/books/a-comprehensive-book-on-autism-spectrumdisorders/autism-spectrum-disorders-in-africa>. Accessed 18 Oct 2017.
- Bakare, M. O., Ebigbo, P. O., Agomoh, A. O., & Menkiti, N. C. (2008). Knowledge about childhood autism among health workers (KCAHW) questionnaire: Description, reliability and internal consistency. *Clinical Practice and Epidemiology in Mental Health*, 4, 17. <https://doi.org/10.1186/1745-0179-4-17>.
- Bakare, M. O., Ebigbo, P. O., Agomoh, A. O., Eaton, J., Onyeama, G. M., Okonkwo, K. O., Onwukwe, J. U., Igwe, M. N., Orovwigho, A. O., & Aguocha, C. M. (2009a). Knowledge about childhood autism and opinion among healthcare workers on availability of facilities and law caring for the needs and rights of children with childhood autism and other developmental disorders in Nigeria. *BMC Pediatrics*, 9, 12. <https://doi.org/10.1186/1471-2431-9-12>.
- Bakare, M. O., Agomoh, A. O., Ebigbo, P. O., Eaton, J., Okonkwo, K. O., Onwukwe, J. U., & Onyeama, G. M. (2009b). Etiological explanation, treatability and preventability of childhood autism: A survey of Nigerian healthcare workers' opinion. *Annals of General Psychiatry*, 8, 6. <https://doi.org/10.1186/1744-859X-8-6>.
- Bakare, M. O., Munir, K. M., & Kinney, D. K. (2011a). Association of hypomelanotic skin disorders with autism: Links to possible etiologic of vitamin-D levels in autism? *Hypothesis*, 9(1), e2.
- Bakare, M. O., Igwe, M. N., Odinka, P. C., & Iteke, O. (2011b). Neuropsychiatric diagnosis and psychotropic medication prescription patterns in a mental hospital-based child and adolescent psychiatric service in Nigeria. *Journal of Health Care for the Poor and Underserved*, 22(3), 751–755. <https://doi.org/10.1353/hpu.2011.0078>.
- Bakare, M. O., Ebigbo, P. O., & Ubochi, V. N. (2012). Prevalence of autism spectrum disorder among Nigerian children with intellectual disability: A stopgap assessment. *Journal of Health Care for the Poor and Underserved*, 23(2), 513–518. <https://doi.org/10.1353/hpu.2012.0056>.
- Bakare, M. O., Munir, K. M., & Bello-Mojeed, M. A. (2014). Public health and research funding for childhood neurodevelopmental disorders in Sub-Saharan Africa: A time to balance priorities. *Healthcare in Low-resource Settings*, 2(1), 2014.
- Bakare, M. O., Tunde-Ayinmode, M. F., Adewuya, A. O., Bello-Mojeed, M. A., Sale, S., James, B. O., Yunusa, M. A., Obindo, J. T., Igwe, M. N., Odinka, P. C., Okafor, C. J., Oshodi, Y. O., Okonoda, K. M., Munir, K. M., & Orovwigho, A. O. (2015). Recognition of Autism Spectrum Disorder (ASD) symptoms and knowledge about some other aspects of ASD among final year medical students in Nigeria, Sub-Saharan Africa. *BMC Research Notes*, 8, 454. <https://doi.org/10.1186/s13104-015-1433-0>.
- Bakare, M. O., Bello-Mojeed, M. A., Munir, K. M., Ogun, O. C., & Eaton, J. (2016). Neurodevelopmental delay among children under the age of three years at immunization clinics in Lagos State, Nigeria – Preliminary report. *Scientific Reports*, 6, 25175. <https://doi.org/10.1038/srep25175>.
- Bakare, M. O., Taiwo, O. G., Bello-Mojeed, M. A., & Munir, K. M. (2019). Autism spectrum disorders in

- Nigeria: A scoping review of literature and opinion on future research and social policy directions. *Journal of Health Care for the Poor and Underserved*, 30(3), 899–909. <https://doi.org/10.1353/hpu.2019.0063>.
- Baxter, A. J., Brugha, T. S., Erskine, H. E., Scheurer, R. W., Vos, T., & Scott, J. G. (2015). The epidemiology and global burden of autism spectrum disorders. *Psychological Medicine*, 45(3), 601–613.
- Bello-Mojeed, M. A., & Bakare, M. O. (2013). Improving treatment of children with autism spectrum disorder in low- and middle-income countries: The role of non-specialist care providers. *PLoS Medicine*, 10(12), e1001573. <https://doi.org/10.1371/journal.pmed.1001573>.
- Bello-Mojeed, M. A., Ogun, O. C., Omigbodun, O. O., & Adewuya, O. A. (2011). Late identification of autistic disorder in Nigeria: An illustration with 2 case reports. *Nigerian Journal of Psychiatry*, 9(2), 31–35.
- Bello-Mojeed, M. A., Bakare, M. O., & Munir, K. (2013). Identification of Autism Spectrum Disorders (ASD) in Africa; Need for shifting research and public health focus; Comprehensive guide to autism; <http://www.springerreference.com/docs/html/chapterdbid/331260.html>. Accessed 18 Oct 2017.
- Bello-Mojeed, M., Ani, C., Lagunju, I., & Omigbodun, O. (2016). Feasibility of parent-mediated behavioural intervention for behavioural problems in children with Autism Spectrum Disorder in Nigeria: A pilot study. *Child and Adolescent Psychiatry and Mental Health*, 10(1), 28. <https://doi.org/10.1186/s13034-016-0117-4>.
- Bello-Mojeed, M. A., Omigbodun, O. O., Bakare, M. O., & Adewuya, A. O. (2017). Pattern of impairments and late diagnosis of autism spectrum disorder among a sub-Saharan African clinical population of children in Nigeria. *Global Mental Health (Cambridge, England)*, 4, e5. <https://doi.org/10.1017/gmh.2016.30>.
- Centers for Disease Control and Prevention. (2016). Autism spectrum disorder (ASD), Data & statistics. Available from: <http://www.cdc.gov/ncbddd/autism/data.html>
- Chinawa, J. M., Manyike, P. C., Aniwada, E. C., Chinawa, A. T., Obu, H. A., Odetunde, O. I., Nwokocha, A. R., & Ibekwe, R. R. (2016). Prevalence and socioeconomic correlates of autism among children attending primary and secondary schools in south east Nigeria. *African Health Sciences*, 16(4), 936–942. <https://doi.org/10.4314/ahs.v16i4.8>.
- Christensen, D. L., Baio, J., Braun, K. V., Bilder, D., Charles, J., Constantino, J. N., et al. (2016). Prevalence and characteristics of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2012. *Morbidity and Mortality weekly Report, Surveillance Summaries*, 65(3), 1–23.
- Elsabbagh, M., Divan, G., Koh, Y. J., Kim, Y. S., Kauchali, S., Marcin, C., et al. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research*, 5(3), 160–179.
- Eseigbe, E. E., Nuhu, F. T., Sheikh, T. L., Eseigbe, P., Sami, K. A., & Olisah, V. O. (2015). Knowledge of childhood autism and challenges of management among medical doctors in Kaduna State, Northwest Nigeria. *Autism Research and Treatment*, 2015, 892301. <https://doi.org/10.1155/2015/892301>.
- Ezegwui, I. R., Lawrence, L., Aghaji, A. E., Okoye, O. I., Okoye, O., Onwasigwe, E. N., & Ebigo, P. O. (2014). Refractive errors in children with autism in a developing country. *Nigerian Journal of Clinical Practice*, 17(4), 467–470. <https://doi.org/10.4103/1119-3077.134042>.
- Fombonne, E. (2003). The prevalence of autism. *JAMA*, 289, 87–89.
- Fuentes, J., Bakare, M., Munir, K., Aguayo, P., Gaddour, N., Öner, Ö., & Mercadante, M. (2012). Autism spectrum disorders. In J. M. Rey (Ed.), *IACAPAP e-textbook of child and adolescent mental health*. Geneva: International Association for Child and Adolescent Psychiatry and Allied Professions.
- Igwe, M. N., Bakare, M. O., Agomoh, A. O., Onyeama, G. M., & Okonkwo, K. O. (2010). Factors influencing knowledge about childhood autism among final year undergraduate medical, nursing and psychology students of University of Nigeria, Enugu State, Nigeria. *Italian Journal of Pediatrics*, 36, 44. <https://doi.org/10.1186/1824-7288-36-44>.
- Igwe, M. N., Ahanotu, A. C., Bakare, M. O., Achor, J. U., & Igwe, C. (2011). Assessment of knowledge about childhood autism among paediatric and psychiatric nurses in Ebonyi state, Nigeria. *Child and Adolescent Psychiatry and Mental Health*, 5(1), 1. <https://doi.org/10.1186/1753-2000-5-1>.
- Izuwala, D. N., Okoh, B. A., & Alior, E. A. (2016). Clinical pattern of autism in Nigeria. *Autism Research*, 9(3), 376–381. <https://doi.org/10.1002/aur.1531>.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217.
- Lagunju, I. A., Bella-Awusah, T. T., & Omigbodun, O. O. (2014). Autistic disorder in Nigeria: Profile and challenges to management. *Epilepsy & Behavior*, 39, 126–129. <https://doi.org/10.1016/j.yebeh.2014.08.020>.
- Longe, C. I., & Asuni, T. (1972). Four cases of Infantile autism in Nigerian children. Paper read at Third Pan African Psychiatric Conference, Khartoum.
- Lotter, V. (1978). Childhood autism in Africa. *Journal of Child Psychology and Psychiatry*, 19(3), 231–244.
- Munir, K. M., Lavelle, T. A., Helm, D. T., Thompson, D., Prestt, J., & Azeem, M. W. (2016). *Autism: A global framework for action*. Doha: World Innovation Summit for Health. <http://www.wish-qatar.org/wish-2016/forum-reports/forum-reports>.
- Onuora, A. B. (1993). Infantile autism: A case report. *Nigerian Journal of Psychiatry*, 1(3), 169–171.
- Oshodi, Y. O., Olagunju, A. T., Oyelohunnu, M. A., Campbell, E. A., Umeh, C. S., Aina, O. F., Oyibo, W., Lesi, F. E., & Adeyemi, J. D. (2017). Autism spectrum disorder in a community-based sample with

neurodevelopmental problems in Lagos, Nigeria. *Journal of Public Health in Africa*, 7(2), 559. <https://doi.org/10.4081/jphia.2016.559>.

Population Reference Bureau. World population data sheet Washington, DC, 2015. Available from: http://www.prb.org/pdf15/2015-world-population-data-sheet_eng.pdf

Pulse.ng. (2019). Buhari signs disability bill into law, criminalizes discriminations (24/01/2019); <https://www.pulse.ng/news/local/buhari-signs-disability-bill-into-law-criminalises-discrimination/8ls6ezq>

Stepping Stones, Nigeria. (2010). Witchcraft stigmatisation and children's rights in Nigeria – Report prepared for the 54th Session of the UN Committee on the Rights of the Child Geneva, May 2010; <https://www.google.com.ng/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&ved=0ahUKEwjZvbHq3fzWAhXL1RQKHUTxDtYQFggIMAA&url=http%3A%2F%2Fwww.crin.org%2Fen%2Fdocs%2FUNCRC%2520Shadow%2520Report%2520SSN%2520FINAL%2520171209.doc&usg=AOvVaw3li5jsUp2gZkEwtS1l-Tup>. Accessed 18 Oct 2017.

UNICEF, Nigeria. (2017). Child protection – The situation. <https://www.unicef.org/nigeria/protection.html>. Accessed 18 Oct 2017.

Niravam

- [Alprazolam](#)

NLGN1

- [Neuroligins](#)

NLGN2

- [Neuroligins](#)

NLGN3

- [Neuroligins](#)

NLGN4X

- [Neuroligins](#)

NLGN4Y

- [Neuroligins](#)

Noncontingent Reinforcement (NCI)

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Definition

Noncontingent reinforcement is the use of positive reinforcement that is not related to the occurrence of a target behavior. It involves delivering reinforcement on a fixed-time schedule independent of whether the individual exhibits the target behavior during the interval. In other words, the individual's behavior does not influence whether or not reinforcement is provided. For example, noncontingent reinforcement can be used with a child with autism who exhibits disruptive behavior in the classroom for the function of gaining attention from the teacher. The teacher could implement a noncontingent fixed-time schedule of reinforcement where she gives the student attention at fixed times throughout the day. Since he is receiving the attention throughout the day, this results in a decrease of disruptive behavior. Noncontingent reinforcement weakens the contingency between the target response (disruptive behavior) and the reinforcement delivery. If extinction is used at the same time, this means that no connection is made between the target response and the reinforcement and the behavior decreases.

See Also

- [Contingencies of Reinforcement](#)
- [Negative Reinforcement](#)
- [Operant Conditioning](#)
- [Positive Reinforcement](#)

References and Reading

- Matson, J. L. (2009). *Applied behavior analysis for children with Autism spectrum disorders*. New York: Springer.
- Matson, J. L., & Santino, L. V. (2008). A review of behavioral treatments for self-injurious behaviors of persons with Autism spectrum disorders. *Behavior Modification*, 32, 61–76.
- Tucker, M., Sigafoos, J., & Bushell, H. (1998). Use of noncontingent reinforcement in the treatment of challenging behavior: A review and clinical guide. *Behavior Modification*, 22, 529–547.

Nondirective Play Therapy

► Play Therapy

Nonfluency

► Fluency and Fluency Disorders

Nonfluent Aphasia

► Broca's Aphasia

Nonliteral Language

► Figurative Language

Nonparametric Statistics

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Synonyms

Distribution-free statistics

Definition

Nonparametric statistics encompasses the statistical methods that do not make any assumptions about

the underlying distribution of data. It can be contrasted with *parametric statistics*, which makes explicit assumptions about the distribution of observed data and which uses the data to estimate *parameters* of that distribution. For example, it is common to assume that data is drawn from a normal distribution with unknown parameters μ (mean) and σ^2 (variance); a parametric approach would estimate μ and σ by the sample mean and the sample standard deviation, respectively. Nonparametric statistics, on the other hand, would not assume the data is normally distributed *a priori*, and instead would estimate the shape of the distribution itself.

There are two main types of nonparametric methods in statistics: methods that attempt to discover the unknown underlying distribution of the data, and methods that make statistical inferences without regard to any underlying distribution. Methods of the first type include the histogram, and kernel methods such as kernel density estimation. Methods of the second type include hypothesis tests based on the rank ordering of the data rather than the actual data values. Frequently used nonparametric tests include the Wilcoxon signed rank test, the Kruskal-Wallis test, and Spearman's and Kendall's rank-correlation coefficients.

References and Reading

- Tamhane, A. C., & Dunlop, D. D. (2000). *Statistics and data analysis: From elementary to intermediate* (pp. 562–604). Upper Saddle River: Prentice-Hall.
- Wasserman, L. (2004). *All of statistics* (pp. 303–326). New York: Springer.
- Wasserman, L. (2006). *All of nonparametric statistics*. New York: Springer.

Nonstandard Assessment

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Definition

A *nonstandard assessment* is one that does not rely on a standardized test, often because

the student being evaluated does not fit the normative sample for the test. For example, if a student is an English language learner, ideally he or she should be assessed using a standardized test developed and normed on a population speaking the same native language as the student. However, for some languages, such tests may simply not be available. In this situation, an examiner might need to rely primarily on nonstandard assessment procedures such as observations of the student's language use in multiple settings and information from the family about the student's past language development and current language functioning.

The term *nonstandard assessment* also is sometimes used to describe accommodations of standard testing conditions for students with disabilities; these nonstandard assessment accommodations are intended to “level the playing field” for the student without altering the basic nature, reliability, or validity of the test. For instance, if an examiner wanted to administer a written test of science content knowledge to a student with very limited reading skills, then a nonstandard assessment involving reading the test items aloud and having the student give a verbal response would usually be acceptable; however, if the standardized test was intended to measure reading fluency, then the same nonstandard assessment would be inappropriate because the nonstandard assessment would not measure the student's reading fluency. As another example, a relatively lengthy test administration might be broken into several shorter testing segments for a student with an autism spectrum disorder in order to help maintain the student's consistent attention during testing. Although nonstandard assessments can provide useful information, they should be implemented and interpreted with caution.

See Also

- [Norm-Referenced Testing](#)
- [Standardized Tests](#)
- [Validity](#)

References and Reading

- American Educational Research Association, American Psychological Association, & National Council on Measurement and Evaluation. (1999). *Standards for educational and psychological testing*. Washington, DC: American Educational Research Association.
- Salvia, J., Ysseldyke, J., & Bolt, S. (2009). *Assessment in special and inclusive education*. Belmont: Wadsworth.

Nonsymbolic Communication

► [Gestures](#)

Nonverbal Communication

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Definition

There are children with autism who may be considered nonverbal, that is, children who use very few spoken words on a daily basis or for whom spoken language is not their primary means of communication. Some children are young and have not yet developed spoken language skills. These young children would be considered preverbal. Other children or older individuals, who are above the age of 5 years, who have not developed functional spoken language skills can be considered nonverbal. Development of the ability to use nonprompted (i.e., spontaneous) spoken language in a flexible manner (i.e., across many contexts) prior to age 6 is known to predict positive long-term outcomes for individuals with autism; this ability is sometimes termed “useful speech.” Thus, individuals with autism who fail to develop useful speech during the preschool years may require other approaches to allow them to achieve the ability to communicate functionally by other means. Communication systems designed to be used by individuals without

functional spoken language skills are termed “augmentative and alternative communication” (AAC) systems; these systems include the use of gestures, eye gaze, body postures, sign language, photographs, printed words, objects, and symbols as the means of conveying meaning. In fact, research has shown that the use of an AAC system can support the development of spoken language in individuals who are considered nonverbal. Research is now underway to identify those children who may not respond well to behavioral interventions focusing on the development of spoken language and who may need earlier access to AAC systems.

The picture exchange communication system (PECS) is an example of an AAC system that can be used with children with autism who are nonverbal or minimally verbal. In the PECS approach, children use a picture exchange with a communicative partner to access desired materials. The partner responds to the child’s communication act (i.e., giving the picture) by putting the child’s communicative message into words. The use of PECS can be incorporated into a variety of intervention approaches (e.g., prelinguistic milieu teaching) to provide the means by which a child can produce a communication act.

See Also

- [Communication Interventions](#)
- [Communicative Acquisition in ASD](#)

References and Reading

- Bondy, A., & Frost, L. (2002). *A picture’s worth: PECS and other visual communication strategies in autism*. Bethesda: Woodbine House.
- Flippin, M., Reszka, S., & Watson, L. R. (2010). Effectiveness of the picture exchange communication system (PECS) on communication and speech for children with autism spectrum disorders: A meta-analysis. *American Journal of Speech-Language Pathology*, 19, 178–195.
- Mirenda, P. (2003). Toward a functional augmentative and alternative communication for students with autism: Manual signs, graphic symbols, and voice output communication aids. *Language, Speech, and Hearing Services in Schools*, 34, 203–216.
- Mirenda, P., & Iacona, P. (2011). *Autism spectrum disorders and AAC*. Baltimore: Brookes.
- Romski, M., Sevcik, R. A., Cheslock, M., & Barton, A. (2006). The system for augmenting language: AAC and emerging language intervention. In R. J. McCauley & M. E. Fey (Eds.), *Treatment of language disorders in children* (pp. 123–147). Baltimore: Brookes.
- Schlusser, R. W., & Wendt, O. (2008). Effects of augmentative and alternative communication intervention on speech production in children with autism: A systematic review. *American Journal of Speech-Language Pathology*, 17, 212–230.
- Sevcik, R. A., & Romski, M. (2005). Early visual-graphic symbol acquisition by children with developmental disabilities. In L. L. Namy & L. L. Namy (Eds.), *Symbol use and symbolic representation: Developmental and comparative perspectives* (pp. 155–170). Mahwah: Lawrence Erlbaum.
- Yoder, P., & Stone, W. L. (2006). A randomized comparison of the effect of two prelinguistic communication interventions on the acquisition of spoken communication in preschoolers with ASD. *Journal of Speech, Language, and Hearing Research*, 49, 698–711.

Nonverbal Intelligence

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Definition

Nonverbal intelligence describes thinking skills and problem-solving abilities that do not fundamentally require verbal language production and comprehension. This type of intelligence involves manipulating or problem solving about visual information and may vary in the amount of internalized, abstract, or conceptual reasoning and motor skills that are required to complete a task. Nonverbal intelligence is often closely linked with the Performance IQ domain of intellectual ability tests that evaluates nonverbal abilities, a domain which is often viewed in comparison to the Verbal IQ domain.

Historical Background

The earliest intelligence tests developed in the mid- to late-1800s primarily measured sensory and motor abilities rather than conceptual or language abilities. However, the introduction of the 1905 Binet-Simon Scale and factor analytic theories of intelligence brought a wave of new perspectives on intelligence testing, separating out visual processing/reasoning/perception as unique components of intelligence. Historically, nonverbal IQ assessments were developed by the US military in World War I with the goal of assessing recruits who had impaired verbal/language abilities (e.g., illiterate, less proficient with English).

Nonverbal intelligence often plays a key role in understanding cognition and intellectual abilities for individuals with autism spectrum disorder (ASD). An uneven pattern of cognitive strengths and weaknesses in individuals with ASD has been documented as far back as Kanner's early descriptions of the disorder. His observations of attention to detail at the expense of global meaning, and a wealth of subsequent research findings in the field, led to the belief that enhanced nonverbal abilities may be a core feature for all individuals with ASD. It is now understood that these nonverbal abilities vary based on age, language level, overall level of functioning, and diagnosis, but are still crucial to the understanding of cognitive profiles in many individuals with ASD.

Current Knowledge

Assessment of Nonverbal Intelligence

Measurement of nonverbal intelligence serves two key functions: First, tests of nonverbal intelligence offer an assessment of nonverbal abilities, such as disembedding, visual sequencing, and pattern recognition skills. Some tests are designed to solely assess nonverbal abilities (e.g., Leiter International Performance Scale-Revised, Universal Nonverbal Intelligence Test), while others offer index or composite scores for nonverbal skills alongside global scores for verbal skills (e.g., Perceptual Reasoning Index on the Wechsler Scale for Children, fourth edition; Nonverbal Cluster on the Differential Ability Scales). In

addition to assessing nonverbal abilities, nonverbal tests of intelligence can provide a mechanism for quantifying intellectual ability in individuals without spoken language or who have very impaired expressive and receptive language skills.

Intelligence tests vary in the extent to which they meet each of these purposes. For example, the Performance IQ (PIQ) subtests on the Wechsler scales measure non-language visual reasoning skills but provide instructions verbally. In contrast, on the Leiter International Performance Scale-Revised (Leiter-R) and the Test of Nonverbal Intelligence, Fourth Edition (TONI-4), all item prompts are administered with gestures and facial expressions, and individuals respond by pointing to pictures or by moving cards/shapes. These tests of intelligence that are nonverbal offer the advantage of removing verbal language demands from both administration and response. However, it should be noted that internalized language often still assists performance on nonverbal intelligence tests. For instance, when completing a sequencing or pattern recognition task, performance is often improved by orally or internally labeling the components of the sequence/pattern (e.g., car, plane, car, plane). In other words, nonverbal tests may still be measuring aspects of verbal abilities.

Nonverbal Intelligence in ASD

Assessment of intellectual ability can be very complicated for individuals with ASD given the uneven pattern of abilities across domains of functioning. For individuals with ASD, a Full Scale IQ (FSIQ) score can be misrepresentative since it will average across peaks and troughs in functioning. There is the risk that interpretations of these average scores will dampen the presence of particular areas of strength and mask the significance or impact of areas of impairment.

It has been consistently documented that individuals with ASD have an uneven or spiky profile of intellectual and cognitive abilities, with nonverbal abilities often shown to be stronger than verbal abilities. This pattern has most frequently been discussed as a characteristic profile of scores across domains on the Wechsler intelligence tests, with PIQ exceeding Verbal IQ (VIQ). Peaks in PIQ performance are evident on the Block Design and Matrix Reasoning subtests that tap into

visuoperceptual processing. In Block Design, a set of colored blocks is used to create a design presented in a picture (i.e., form a “whole” from its “parts”), and Matrix Reasoning requires the identification of missing parts of pictures. In contrast, a relative weakness on the Wechsler scales is consistently demonstrated on the Comprehension subtest of the VIQ index, a task requiring the integration of social knowledge and conventions (e.g., “What is the thing to do if you find an envelope in the street that is sealed, addressed, and has a new stamp on it?”). It should be noted that this is *not* the typical profile of individuals who meet the diagnostic criteria for Asperger’s disorder. This group has more frequently demonstrated high verbal IQ compared to lower performance IQ.

Valid assessment of intellectual functioning, and consideration of nonverbal intelligence, also plays an important role in ASD research. A measure of intellectual functioning is often needed to characterize the group of individuals with ASD who are participating in the study (e.g., Are the individuals with ASD of average, high, or low functioning abilities?). In addition, a group of individuals with ASD is often matched to another group of individuals (e.g., typically developing controls, individuals with another non-ASD developmental disorder) on a global measure of intellectual ability so that the two groups can be compared on a more specific cognitive ability or construct. The unevenness among cognitive abilities leads to difficult decisions regarding what type of test to use for matching across individuals or groups (e.g., Match based on the highest strength? The lowest weakness? An average across all skills?). This is such a complicated question that an entire issue of the *Journal of Autism and Developmental Disorders* (Vol. 34, No. 1) was devoted to these questions.

Variability Within Nonverbal Intelligence in ASD: A Nonverbal Perceptual Strength

Although nonverbal intelligence is often globally higher than verbal intelligence for individuals with ASD on molar tests, such as PIQ, there is variability among nonverbal abilities. Relative weaknesses are evident in nonverbal conceptual

tasks that require some abstraction of ideas (e.g., recognizing the sequence or pattern of a set of items, identifying the semantic relationship among the items). In contrast, and as described above, strengths in nonverbal intelligence are generally seen in nonverbal tasks that are perceptually demanding (e.g., the Block Design subtest, puzzles). More specifically, individuals with ASD are particularly good at disembedding details from the global elements or gestalt of stimuli and the world around them. This detail-focused processing style has been observed anecdotally by families and clinicians, and has been extensively explored in research studies. In addition to Kanner’s early descriptions, parents of individuals with ASD have often described their children’s uncanny ability to notice single books out of place on bookshelves, or complete puzzles with the pieces turned over. In empirical research, this processing style has been examined with a wide variety of tasks, including the Embedded Figures Test and hierarchical letter tasks. The Embedded Figures Test involves identifying a simple shape (e.g., a triangle) embedded in a complex picture (e.g., a baby carriage), while the hierarchical letter test requires attending to the detailed “local” aspects of a stimulus versus the gestalt or “global” aspects (e.g., a large A letter made up of little S letters). The general theme across these tasks is that individuals with ASD are able to process the detailed or local level of stimuli more accurately and/or efficiently than the global level. Researchers have theorized that these abilities may be a result of an enhanced ability to perceive details and ignore the global gestalt. Alternatively, these strengths may result from the inability (or reduced ability) to integrate whole and maintain the precedence of global features. Regardless of the mechanism, this area of research has consistently highlighted an area of strength for individuals with ASD, and has been conceptualized as a cognitive style rather than a cognitive deficit. Although there is some disagreement regarding how pervasive this detail-focused processing style is across individuals with ASD of different levels of functioning and subtypes, weak central coherence theory has sought to employ this perceptual bias in explaining other core features of ASD.

Future Directions

A clear understanding of nonverbal intelligence has allowed clinicians and researchers to capture areas of strength for individuals with ASD that might not otherwise be evident in verbally based measures of intellectual ability. However, the discrepancy between nonverbal and verbal abilities leads to the dilemma of what is “true” intelligence for individuals with ASD, and whether this is even possible to identify given wide variability within the population. For example, individuals with an Asperger syndrome diagnosis often contrast other individuals on the spectrum and demonstrate better verbal abilities and relatively lower nonverbal intelligence. With the anticipated change in DSM-V diagnostic criteria for ASD and the merging of all diagnoses into one ASD spectrum category, there will be added heterogeneity in cognitive profiles and further difficulty with capturing “true” intelligence for individuals with ASD.

Finally, intelligence is used as a predictor of outcome, and for some individuals with ASD, nonverbal and verbal intelligence scores would predict two very different trajectories for the same individual: (1) Do higher nonverbal IQ scores represent true potential when unburdened of verbal language? (2) Do these higher scores represent splinter skills that do not efficiently or effectively translate to other areas of functioning? Future research on the predictive abilities of nonverbal intelligence will be needed to provide clear answers to these questions.

See Also

- Block Design Subtest
- Clinical Assessment
- Cognitive Skills
- Crystallized Intelligence
- Figure-Ground Discrimination
- Fluid Intelligence
- Intelligence Quotient
- Psychological Assessment
- Savant Skills (in Autism)
- Subtest Scatter

- Verbal Intelligence
- Weak Central Coherence
- Wechsler Preschool and Primary Scale of Intelligence

References and Reading

- Brian, J. A., & Bryson, S. E. (1996). Disembedding performance and recognition memory in autism/PDD. *Journal of Child Psychology and Psychiatry*, 37(7), 865–872.
- Burack, J. A., Iarocci, G., Flanagan, T. D., & Bowler, D. (2004). On mosaics and melting pots: Conceptual considerations of comparison and matching strategies. *Journal of Autism and Developmental Disorders*, 34(1), 65–73.
- Charman, T. (2004). Matching preschool children with autism spectrum disorders and comparison children for language ability: Methodological issues in autism-related research. *Journal of Autism and Developmental Disorders*, 34(1), 59–64.
- Happé, F. (1999). Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Science*, 3(6), 216–222.
- Happé, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36(1), 5–25.
- Jarrold, C., & Brock, J. (2004). To match or not to match? Methodological issues in autism-related research. *Journal of Autism and Developmental Disorders*, 34(1), 81–86.
- Jolliffe, T., & Baron-Cohen, S. (1997). Are people with autism and Asperger syndrome faster than normal on the Embedded Figures Test? *Journal of Child Psychology and Psychiatry*, 38(5), 527–534.
- Jolliffe, T., & Baron-Cohen, S. (1999). A test of central coherence theory: Linguistic processing in high-functioning adults with autism or Asperger syndrome: Is local coherence impaired? *Cognition*, 71(2), 149–185.
- Kuschnir, E. S., Bennetto, L., & Yost, K. (2007). Patterns of nonverbal cognitive functioning in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(5), 795–807.
- Lee, P. S., Foss-Feig, J., Henderson, J. G., Kenworthy, L. E., Gilotty, L., Gaillard, W. D., & Vaidya, C. J. (2007). Atypical neural substrates of Embedded Figures Task performance in children with autism spectrum disorder. *NeuroImage*, 38, 184–193.
- Manjaly, Z. M., Bruning, N., Neufang, S., Stephan, K. E., Brieber, S., Marshall, J. C., Kamp-Becker, I., Remschmidt, H., Herpertz-Dahlmann, B., Konrad, K., & Fink, G. R. (2007). Neurophysiological correlates of relatively enhanced local visual search in autistic adolescents. *NeuroImage*, 35, 283–291.
- Mottron, L. (2004). Matching strategies in cognitive research with individuals with high-functioning autism: Current practices, instrument biases, and recommendations. *Journal of Autism and Developmental Disorders*, 34(1), 19–27.

- Mottron, L., & Belleville, S. (1993). A study of perceptual analysis in a high-level autistic subject with exceptional graphic abilities. *Brain and Cognition*, 23(2), 279–309.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36(1), 27–43.
- Navon, D. (1977). Forest before trees: The precedence of global features in visual perception. *Cognitive Psychology*, 9, 353–383.
- Plaisted, K. C., Swettenham, J., & Rees, L. (1999). Children with autism show local precedence in a divided attention task and global precedence in a selective attention task. *Journal of Child Psychology and Psychiatry*, 40(5), 733–742.
- Ring, H. A., Baron-Cohen, S., Wheelwright, S., Williams, S. C. R., Brammer, M. J., Andrew, C., & Bullmore, E. T. (1999). Cerebral correlates of preserved cognitive skills in autism: A functional MRI study of Embedded Figures Task performance. *Brain*, 122, 1305–1315.
- Shah, A., & Frith, U. (1983). An islet of ability in autistic children: A research note. *Journal of Child Psychology and Psychiatry*, 24, 613–620.
- Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry*, 34(8), 1351–1364.
- Witkin, H., Oltman, P., Raskin, E., & Karp, S. (1971). *A manual for the Embedded Figures Test*. Palo Alto: Consulting Psychologists Press.

Nonverbal Learning Disabilities (NLD)

► Right-Hemisphere Syndrome

Nonverbal Learning Disabilities (NLD), Second Edition

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Synonyms

Developmental right-hemisphere syndrome; Nonverbal learning disorder; Social-emotional learning disability

Short Description of Definition

Nonverbal learning disability (NLD) is a neurobehavioral disorder that impacts a person's intelligence due to dysfunctions of the brain. NLD is a developmental disorder which constrains a child's linguistic performance in the nonverbal domain. While an individual with NLD shows deficits in understanding nonlinguistic signs, he/she can be highly functional in verbal communications. Other neuropsychological disorders related to NLD can impact a child's abilities to perform in academic, social/emotional, and vocational areas (Palombo 2006). A critical challenge for an individual with NLD is social interaction due to the lack of visual processing abilities such as recognizing facial expressions and lack of spatial perception. NLD is not apparent at a very young age, such as preschool age, although the inability to understand nonverbal signs significantly manifests at the age when children begin to understand social interaction through engagement in verbal conversation (Thompson 1996). NLD does not necessarily constrain academic skills, but impairs mainly social interactions with characteristics similar to what was previously identified as Asperger's syndrome, now part of Autism Spectrum Disorder (ASD), and pragmatic language impairment (PLI).

Categorization

Nonverbal learning disorder was first identified by Doris Johnson and Helmer Myklebust in 1967 as a subtype of learning disability. Based on research by Rourke et al. (1989), this class of learning disability is organized into two categories: phonological processing disorder and nonverbal learning disorder (p. 169). Typically, people with learning disabilities fail to perform at age level and exhibit difficulty in rote-verbal learning and in phonological processing. Unlike children with general learning disabilities, children with NLD demonstrate significantly higher verbal IQ than performance IQ. For example, children with NLD show primary impairments in visual processing, tactile perception, complex psychomotor

skills, fine motor skills, and manipulation of materials (Rourke 1989). Accordingly, they need significant support in adapting to changes, in generalizing what they have learned, and in following instructions with multiple steps.

There is no consensus on the cluster of learning disabilities that comprises NLD. Accurate procedures to diagnose children with NLD are not yet available as there have been multiple changes in classification. The American psychiatric association (APA) uses the diagnostic and statistical manual of mental disorder (DSM) to produce an accurate diagnosis within various mental disability categories (APA 2000). NLD was not included in the DSM-IV or in the current revision of the DSM-5 (APA 2013). Moreover, many symptoms of NLD are similar to those of ASD and PLI. The differences among these three disorders have not been clearly defined. It has been proposed that NLD may be a disorder on the “borderlands of autism” (Bishop 1989, p. 107). Children with ASD, who perform well in cognitive and linguistic areas, have features in common with both NLD and PLI. PLI is also considered to be a social communication disorder, meaning that children with PLI have difficulty using language appropriately in social situations. Overall, NLD, ASD, and PLI have overlapping, but not identical, symptoms. For example, an individual diagnosed with NLD will show some of the features of PLI, but the reverse may not be true (Bishop and Norbury 2002). The overlap of symptoms among these three learning disorders involves patterns of behavior in a social context. There is yet no evidence of restricted or repetitive patterns of behavior that clearly distinguishes the disorders.

The following is a set of criteria used to identify NLD (Rourke et al. 2002):

1. Bilateral deficits in tactile perception, usually more marked on left side of the body. Simple tactile perception may reach normal levels as the child ages, but interpreting complex tactile stimulation remains impaired.
2. Bilateral deficits in psychomotor coordination, usually more marked on the left side of

the body. Simple, repetitive motor skills may reach normal levels with age, but complex motor skills remain impaired or worsen relative to age norms.

3. Extremely impaired visual-spatial-organizational abilities. Simple visual discrimination can reach normal levels with age, particularly when stimuli are simple. Compared to age norms, complex visual-spatial-organizational abilities worsen with advancing years.
4. Substantial difficulty in dealing with novel or complex information or situations. A strong tendency to rely on rote, memorized reactions, approaches, and responses (often inappropriate for the situation), and failure to learn or adjust responses according to informational feedback. Also, frequent use of verbal responses in spite of the requirements of the novel situation. These tendencies remain or worsen with age.
5. Notable impairments in nonverbal problem solving, concept formation, and hypothesis testing.
6. Distorted sense of time. Estimating elapsed time over an interval and estimating time of day are both notable impairments.
7. Well-developed rote verbal abilities (e.g., single-word reading and spelling), frequently superior to age norms, in the context of notable poor reading comprehension abilities (particularly so in older children).
8. High verbosity that is rote and repetitive, with content disorders of language and deficits in functional/pragmatic aspects of language.
9. Substantial deficits in mechanical arithmetic and reading comprehension relative to strengths in single-word reading and spelling.
10. Extreme deficits in social perception, judgment, and interaction, often leading to eventual social isolation/withdrawal. Easily overwhelmed in novel situations, with a marked tendency toward extreme anxiety, even panic in such situations. High likelihood of developing internalized forms of psychopathology (e.g., depression) in later childhood and adolescence.

Epidemiology

Nonverbal learning disorder (NLD) is diagnosed less frequently than other language-based learning disorders (e.g., reading and written expression disorder). While approximately 10% of the population has been diagnosed with having a learning disability, only 0.1–1.0% of the population has been found to have NLD. NLD has been diagnosed equally in female and male populations (Thompson 1996). Moreover, based on social behavioral problems, children with NLD also show some features of attention deficit hyperactivity disorder (ADHD) and are easily misdiagnosed as having ADHD (Antshel and Khan 2008). Educators often define NLD as a language-based learning disability based on academic and cognitive assessments. Thus, the data regarding NLD are unclear and, as a result, NLD is often misdiagnosed as other types of learning disorder.

Analysis from Case Study

Chris, a child diagnosed with NLD, was able to read before she started school. On a widely used intelligence test, she achieved a verbal IQ score of 120 and a performance IQ score of 102 indicating that she has high functioning verbal ability that is not matched by her nonverbal ability. When she was in preschool she had difficulties in comprehending, transitioning, socializing with other peers, and making self-decisions. She was unable to choose how she wanted to play during free-choice time because reading was the only activity Chris enjoyed in school. When someone stopped her from reading, she became belligerent and refused to comply. This behavior caused the other students to become wary of being close to Chris during free-choice time. When Chris became a 1st grader, she was exceptional in reading and spelling. She was a fast learner with high auditory attention ability. She also liked to create imaginative stories. However, she was not interested in writing down her stories because handwriting was a challenge. Moreover, Chris struggled with math and tended to avoid the

teacher's attention during math class. Aside from math, her scores on other achievement tests were higher than average.

Chris also showed some difficulties in music and art classes. The music class included lots of rhythmical activities. Chris mostly excused herself from those activities to use the bathroom or read books. Drawing, coloring, and creating were the main activities in the art classes. Chris rushed to finish these activities in order to read books. When her art creation was returned to her with feedback to revise, she got angry and threw away her art. Due to her lack of physical coordination and endurance, gym classes were also not enjoyable for Chris. She refused to go to the playground for activities.

This case study contains multiple prognostic factors for NLD. NLD impacted the child's academic and social activities. Chris had difficulties with handwriting, math reasoning, and fine motor skills. These difficulties are related through deficits in visual processing, motor function, and the child's social/emotional profile. Children with NLD generally show lack of both gross and fine motor skills. In this case study, the gym classes preponderantly required gross motor skills (e.g., big movements) and involvement of larger muscles. The handwriting and art projects required mostly fine motor skills (e.g., dexterity) and use of small muscles. Regardless of size of movements, children with NLD demonstrate inadequate kinesthetic processing due to weakness of spatial perception. Moreover, non-reaction towards non-verbal signs is significant evidence of NLD. The case study also contains a significant cue to Chris's diagnosis with NLD by emphasizing how well she responded to auditory directions.

The natural history of NLD shows that symptoms of NLD may be confused with those of ADHD and what used to be termed Asperger's syndrome (a part of ASD). However, main indicators of NLD include critical deficits in visual-spatial processing, nonverbal recognition, and kinesthetic discrepancy. Challenges in these areas greatly impact how children with NLD improve in academic and social/behavioral performances (Palombo 2006).

Clinical Expression and Pathophysiology

Nonverbal learning disorder is a disability characterized by accurate psycholinguistic skills and impoverished visual-spatial orientation, tactile-perceptual, psychomotor, and non-verbal problem-solving skills. These strengths and weaknesses are observed in the following ways:

Assets	Deficits
Intact repetitive motor skills	Bilateral tactile-perceptual and psychomotor deficits (primarily left side)
Responsive to learning through repetition and the auditory modality	Impaired visual discrimination
Well-developed auditory perceptual skills	Impaired visual-spatial organization (e.g., drawing patterns from memory)
Well-developed rote verbal and verbal memory skills	Difficulty with novel and complex situations
Ability to sustain attention to simple, repetitive verbal information	Deficits in nonverbal problem-solving concept formation, hypothesis testing, and use of feedback
Strong receptive language skills including rote verbal memory and verbal associations	Difficulty with cause-effect and recognizing incongruities
Advanced phonemic awareness, including blending and segmentation	Verbose with poor pragmatics (i.e., miss social cues), including inefficient prosody and over reliance on language for relating socially and decreasing anxiety
Average single-word reading and spelling skills	Deficits in math (i.e., functions, decimals, percents, ratios, estimation, geometry, and any visual-spatial math function)
	Impairments in social perception, judgment, and interaction skills, including tendency to withdraw
	Increased risk for suicide

Adapted from Hooper (2000), Rourke and Tsatsanis (1996), Rourke et al. (2002)

In addition to the challenges outlined in the chart above, deficits in higher-level reasoning and problem-solving skills impact on social-emotional learning of an individual with NLD (Rourke 1989). In other words, children with NLD often lack executive functions related to cognitive abilities. These functions engage with complex goal-directed behavior and adaptation in environmental changes. Because these executive functions require self-monitoring and self-awareness to produce appropriate behavior, children with NLD commonly show difficulties with cause-and-effect reasoning, problem solving in various social contexts, and time perception. These are the critical foundations and skills needed for social interactions (Palombo 2006). Issues related to executive function also impact breakdown in social interaction. Children with NLD are less motivated to be social with their peers because of difficulty processing new information and adapting to unfamiliar interpersonal interactions (Volden 2004). Psychopathologies are more evident in children with NLD more than neurotypical children. For example, there is greater risk for depression, withdrawal, and suicide in NLD (Fuerst and Rourke 1995). Awkwardness with other social peers is a distinctive feature of NLD.

Mothers of children with NLD have high levels of maternal stress and high levels of dysfunctional interaction with their children. Indeed, the more severe the disability that children have, the greater the stress and emotional breakdown that the parents have (Antshel and Joseph 2006). The lack of relationship between a mother and a young child with NLD will create a challenge to maintain the relationship in later years. Thompson (1997) described the developmental progression of Nonverbal learning disorder and included additional signs and symptoms presented in early preschool age (3–5 years old). These additional symptoms included strong ability in verbatim memory skills, extreme verbosity, development of early reading skills (e.g., strong letter/number recognitions, spelling skills), and use of literal translations. However, symptoms of impaired gross motor development remained, including balance and

body coordination (e.g., problems riding a bike and spatial confusion).

As they grow into elementary school age (ages 6–10), individuals with NLD will demonstrate deficits in copying text, continue to make literal translations, and continue misjudging and misinterpreting social information. At ages 11–14, individuals with NLD will continue having difficulties in understanding emotional expectations and this will lead to miscommunication with teachers and other social peers at school. Furthermore, difficulties with visual-spatial organizational skills become obstacles to children with NDL and their productivity on tasks. The same behavior deficits involving literal translation, misinterpretation, and misunderstanding cause children with NLD to have difficulties understanding abstract concepts as well as communicating with other social peers. Because of these continued, under-appreciated behavior deficits, children with NLD start to experience some level of depression. Although in high school years (ages 15–18) they appear, at best, to be immature in socializing, they will show improvements in some social domains. Peer tolerance tends to increase so that children with NLD may establish intimate friendships. Relationships between sexes will gradually develop. Eventually, their NLD symptoms will disrupt the ability to become independent adults. Their continuing challenges with literal understanding, lack of ability to solve abstract concepts, and visual-spatial impairments will increase their low self-esteem, depression, withdrawal, and anxiety.

Neuroscience studies have shown that NLD involves abnormalities in the right cerebral hemisphere of the brain which is important for nonverbal processing skills. Rourke and colleagues have spent three decades designing a neuropsychological model to explain the strengths and challenges of individuals with NLD. Their work, *White Matter Model*, was published in 1987. White matter refers to long myelinated fibers in the brain. Their work shifted the focus on the origin of NLD from a primary dysfunction of the right cerebral hemisphere to consideration of an underdevelopment of white matter in the brain (Rourke 1987, 1989, 1995; Rourke et al. 2002). This novel idea

stemmed from observations that cerebral white matter malfunctions in various conditions manifesting impairments in perceptual and cognitive functions similar to NLD, such as agenesis of the corpus callosum, in which the large bundle of white matter connecting two cerebral hemispheres has failed to develop. In addition, children who experienced severe head injuries or who have received repeated radiation treatment on their brains may have damage to white matter and, therefore, may have higher risk to develop the symptoms of NLD.

Evaluation and Differential Diagnosis

The fact that NLD does not produce outstanding symptoms on its own, and has considerable similarities to other disorders, necessitates careful consideration of the evaluation and diagnostic procedures for NLD. The clinical diagnosis of NLD evaluates neuropsychological strength and weakness by implementing Rourke's classification criteria and the preliminary communication profile (Rourke and Tsatsanis 1996). Differential diagnosis should involve a comprehensive neuropsychological evaluation incorporating examination of academic, social-emotional, and cognitive skills. An interdisciplinary team approach should be used to assess functional impairments and disabilities. These professional disciplines may include occupational therapy, speech-language pathology, medicine, psychology, and education. It is critical that various professionals and experts work together to establish concrete understanding of the individual's challenges over time and across multiple developmental domains. From a history of the child, physicians can share profiles of the child's impairments with other professionals to prevent missing possible red flags (e.g., poor psychomotor development, clumsiness, and challenges with early handwriting tasks). It is clearly important that the child be evaluated later in their development such as in middle school or high school. A discrepancy in performance IQ and verbal IQ at any stage of development is a critical piece of evidence.

Based on evidence obtained by multiple professionals, schools can support individuals with

NLD using relevant services (Thompson 1996). Assessment of communication skills must proceed along with examinations in semantic (meaning of words) and pragmatic (functional language use) skills, as these are the areas of common concern for children with NLD. Challenges in literacy or language are often observed in the relative strengths with phonological processing (speech sound processing) and syntax (grammar). Children with NLD may seem to have a rich vocabulary because of favorable scores on receptive/expressive language measures (e.g., *Peabody Picture Vocabulary Test-4th edition (PPVT-4)*, *Expressive One-Word Vocabulary Test-2nd edition (EVT-2)*). However, these test measurements are limited by their examination of the use of socially accepted words, use of multiple words across various contexts, or use of those words that occur with less frequency. The tests do not measure the extent to which individuals with NLD miss meaningful contents in their conversations because they do not fully understand the words they are using. Thus, it is essential to evaluate the higher-level semantic skills of children with NLD including understanding of multiple meanings and figurative language. Standardized measures available to administer tests of these linguistic skills include the *Test of Problem Solving-3rd edition (TOPS-3)*, *The Word Test-3*, and the *Test of Language Competence-2nd edition (TLC-2)*. Professionals should be encouraged to collect valuable information on the appropriate social use of language (e.g., conversational turn taking, maintaining and building on topics, and recognizing and repairing communication breakdowns). Assessments may also be implemented by using *Children's Communication Checklist-2 (CCC-2)*, *Pragmatic Protocol, Assessment of Language Impaired Children's Conversations, Topic Checklist*, and *Narrative Analysis* (i.e., analyzing a child's narrative for story grammar and other components).

Treatment

Because the symptoms of NLD are varied, the most efficient treatment for children with NLD

involves establishing individualized goals and specialized instruction. Educators can then coordinate the interventions to produce the best long-term outcomes. These interventions should include functional practices children with NLD can use on a daily basis. The ultimate goal of all interventions is to improve active processing of new information by helping individuals with NLD to explain concepts, instructions, and stories in their own words rather than relying on repeating known information verbatim from memory (Rourke 1995). Sets of compensations, accommodations, modifications, and individualized learning strategies (CAMs) should be implemented for the appropriate needs of the individuals with NLD (Thompson 1997). For example, a child with NLD may be provided with extra time to navigate himself to a place when given verbal cues. This intervention will require the child to practice utilizing his understanding of spatial and directional concepts. Task analysis is a procedure based on Applied Behavior Analysis (ABA) that is another intervention strategy for children with NLD. Task analysis is a strategy that divides a targeted behavior into a series of step-by-step instructions (Smith 1999). Rather than teaching the multiple steps of the task all at once, an educator can introduce small sets of tasks so that children with NLD have ample time to adapt and process the new information. Depending on the types of tasks, an instructor starts the instruction sequence beginning with the first step (called Forward Chaining) or the children with NLD can start from the last step of the instruction sequence (called Backward Chaining).

Berg (2000) introduced the use of gestalt imagery as an intervention for children with NLD. Children with NLD often get frustrated when creating a gestalt to support their comprehension levels. Since individuals with NLD show deficits in abstract-concept processing, they are not able to organize the parts of a concept from the whole. Implementation of gestalt instruction will help children with NLD to connect incoming language (both oral and written) to prior knowledge. Therefore, these gestalt interventions facilitate smooth socialization of children with NLD by helping them to interpret what they see, hear, and feel

from the surrounding environments. To utilize the gestalt imagery, one needs to learn how to describe a picture, an image, a simple sentence, and then add interpretation and critical thinking to this imaging process (Bell 1986).

The following is a list of accommodations, modifications, and teaching strategies (CAMS) based on the work of Sue Thompson, M.A., C.E. (Thompson 1996):

1. Expectations for this child should always be applied with flexibility, taking into consideration the fact that she has different needs and abilities than her peer group.
2. Independence should be introduced gradually in controlled, nonthreatening situations. The more completely those around her understand this child and her particular strengths and weaknesses, the better prepared they will be to promote attitudes of personal independence. Never leave this child to her own devices in new activities or situations which lack sufficient structure.
3. School assignments which require merely copying text need to be modified or omitted, owing to the visual-spatial nature of such an exercise. Active verbalization and/or subvocalization are the best memory approaches for this child.
4. Adults need to check often for understanding and present information in plain and clear verbal terms (i.e., “spell out” everything). A “parts-to-whole” verbal teaching approach should be utilized. A child with NLD will need to ask a lot of questions, as this is her primary means of gathering information.
5. All expectations need to be direct and explicit. Do not require this child to “read between the lines” to glean your intentions. Avoid sarcasm, figurative speech, idioms, slang, etc., unless you plan to explain your usage.
6. This student’s schedule needs to be as predictable as possible. He should be prepared in advance for changes in routine, such as assemblies, field trips, vacation days, finals, etc.
7. Placement must be in an environment, which has a well-established routine because this child will not decipher nonverbal cues. She cannot adjust well to constant changes in routine (this child lacks the ability to “wing it” in times of doubt) and has learned to fear all new and/or unknown situations and experiences.
8. This child will benefit from cooperative learning situations (when grouped with “good role models”). Active verbalization is an important element in how this child learns.
9. Tell the child everything and encourage her to give you verbal feedback. The most effective instructional procedures are those that associate verbal labels with concrete situations and experiences.
10. Isolation, deprivation, and punishment are not effective methods to change the behavior of a child who is already trying his best to conform (but misinterpreting all kinds of nonverbal cues). If inappropriate behaviors are causing problems at school, a functional analysis and behavioral intervention plan detailing a course of action needs to be completed.

See Also

- Academic Skills
- Academic Supports
- Asperger Syndrome
- Learning Disability
- Multidisciplinary Evaluation
- Pragmatic Language Impairment
- Semantic Pragmatic Disorder

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders*. In 4th ed. text Rev. Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- Antshel, K. M., & Josphe, G. R. (2006). Maternal stress in nonverbal learning disorder: A comparison with reading disorder. *Journal of Learning Disabilities*, 39(3), 194–205.
- Antshel, K. M., & Khan, F. M. (2008). Is there an increased familial prevalence of psychopathology in children

- with nonverbal learning disorders? *Journal of Learning Disabilities*, 41(3), 208–217.
- Bell, N. (1986). *Visualizing & verbalizing: For language comprehension and thinking*. San Luis Obispo, CA: Gander Educational Publishing.
- Berg, M. (2000). Nonverbal learning disabilities. In Presentation at the first annual pine ridge school conference on learning disabilities. Burlington, VT, April 2000.
- Bishop, D. V. M. (1989). Autism, Asperger's syndrome and semantic-pragmatic disorder: Where are the boundaries? *British Journal of Disorders of Communication*, 24, 104–121.
- Bishop, D. V. M. (2000). Pragmatic language impairment: A correlate of SLI, a distinct subgroup, or part of the autistic continuum? In D. V. M. Bishop & L. B. Leonard (Eds.), *Speech and language impairments in children: Causes, characteristics, intervention and outcome* (pp. 99–113). Hove, UK: Psychology Press.
- Bishop, D. V. M., & Norbury, C. F. (2002). Exploring the borderlands of autistic disorders and specific language impairment: A study using standardized diagnostic instruments. *Journal of Child Psychology and Psychiatry*, 43, 917–929.
- Fletcher, J. M. (1989). Nonverbal learning disabilities and suicide: Classification leads to prevention. *Journal of Learning Disabilities*, 22(3), 176.
- Fuerst, D. R., & Rourke, B. P. (1995). Psychosocial functioning of children with learning disabilities at three age levels. *Child Neuropsychology*, 1, 38–55.
- Hooper, S. (2000). Nonverbal learning disabilities. In Presentation at the first annual pine ridge school conference on learning disabilities. Burlington, VT, April.
- Little, S. S. (1993). Nonverbal learning disabilities and socioemotional functioning: A review of recent literature. *Journal of Learning Disabilities*, 26(10), 653–665.
- Myklebust, H. R. (1975). Nonverbal learning disabilities: Assessment and intervention. In H. R. Myklebust (Ed.), *Progress in learning disabilities* (Vol. 3, pp. 85–121). New York: Grune & Stratton.
- Palombo, J. (2006). *Nonverbal learning disabilities: A clinical perspective*. New York: W.W. Norton.
- Roman, M. (1998). The syndrome of nonverbal learning disabilities: Clinical description and applied aspects. *Current Issues in Education* (on-line), 1(7). Available: <http://cie.edu.asu.edu/volume1/number7/>.
- Rourke, B. P. (1987). Syndrome of nonverbal learning disabilities: The final common pathway of white-matter disease/dysfunction? *The Clinical Neuropsychologist*, 1(3), 209–234.
- Rourke, B. P. (1989). Introduction: The NLD syndrome and the white matter model. In B. P. Rourke (Ed.), *Nonverbal learning disabilities: The syndrome and the model* (pp. 80–100). New York: The Guilford Press.
- Rourke, B. P. (Ed.). (1995). *Syndrome of nonverbal learning disabilities: Neurodevelopmental manifestations*. New York: The Guilford Press.
- Rourke, B. P., & Tsatsanis, K. D. (1996). Syndrome of nonverbal learning disabilities: Psycholinguistic assets and deficits. *Topics in Language Disorders*, 16, 30–44.
- Rourke, B. P., Young, G. C., & Leenaars, A. A. (1989). A childhood learning disability that predisposes those afflicted to adolescent and adult depression and suicide risk. *Journal of Learning Disabilities*, 22(3), 169–175.
- Rourke, B. P., Ahmad, S., Collins, D., Hayman-Abello, B., Sayman-Abello, S., & Warriner, E. (2002). Child clinical/pediatric neuropsychology: Some recent advances. *Annual Reviews of Psychology*, 53, 309–339.
- Smith, G. J. (1999). Teaching a long sequence of behavior using whole task training, forward chaining, and backward chaining. *Perceptual and Motor Skills*, 89(3), 951–965.
- Thompson, S. S. (1996, Fall). Nonverbal learning disorders. *The Gram* (LDA-CA newsletter).
- Thompson, S. (1997). *The source for nonverbal learning disabilities*. Lingui Systems. http://www.nldontheweb.org/Sue_Thompson_pubs.htm
- Volden, J. (2004). Nonverbal learning disability: A tutorial for speech-language pathologists. *American Journal of Speech-Language Pathology*, 13, 128–141.
- Walls, R. T., Zanes, T., & Ellis, W. D. (1981). Forward and backward chaining, and whole task methods: Training assembly tasks in vocational rehabilitation. *Behavior Modification*, 15(1), 61–74. <https://doi.org/10.1177/014544558151005>.

Nonverbal Learning Disorder

► [Nonverbal Learning Disabilities \(NLD\), Second Edition](#)

Nonverbal Oral Apraxia

► [Oral-Motor Apraxia](#)

Noonan and Ras/MAPK Pathway Syndromes

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Synonyms

[Male Turner](#); [Ras/MAPK pathway syndromes](#)

Short Description or Definition

Noonan syndrome (NS) is a multiple malformation genetic disorder characterized by dysmorphic facies including hypertelorism, ptosis, low-set posteriorly rotated ears, short neck, and relative macrocephaly. Associated features include short stature, chest deformity, undescended testes, muscle hypotonia, frequent learning difficulties, and congenital heart disease most commonly valvular pulmonary stenosis. In 2001 (Tartaglia et al. 2001), a germline mutation in the *PTPN11* gene in the Ras/MAPK (mitogen-activated protein kinase) signal transduction pathway was found to be a cause of nearly 50% of the cases of NS. Since then, a number of other genes in the same pathway have been identified but in about 25% the causative gene for NS has yet to be identified.

Before NS was recognized as a distinct syndrome, some females were misdiagnosed as having Turner syndrome, and many males, given the diagnosis of male Turner, are now recognized as having NS. The recognition of this entity as a distinct new syndrome was presented first in 1963 (Noonan and Ehmke 1963) and then published in 1968 (Noonan 1968). Dr. John Opitz (1985) proposed the eponym “Noonan syndrome” because Dr. Jacqueline Noonan was the first to recognize that the syndrome occurred in both males and females, had normal chromosomes, could be inherited, and frequently had valvular pulmonary stenosis.

Categorization

NS is the most common of a number of conditions that involve a gene mutation in the RAS/MAPK pathway. These include cardio-facial-cutaneous syndrome, Costello syndrome, Leopard syndrome, and neurofibromatosis₁ (NF₁).

Epidemiology

The estimated incidence of NS is 1:1000–1:2500 births. It occurs worldwide with a slight

predominance in males. It is transmitted as an autosomal dominant, but sporadic cases are frequent.

Natural History, Prognostic Factors, and Outcomes

Infancy

NS may be suspected in fetal life because of the frequent finding of cystic hygroma identified on routine fetal ultrasound. This abnormality is not unique to NS and is not uniformly present. If either parent has NS, and the causative gene is known, prenatal testing of the fetus can be accomplished. There is an increased incidence in polyhydramnios but most newborns are within normal range for length and weight. In a minority of infants with NS, the newborn period may be complicated by significant distress due to pleural effusions with respiratory compromise. Dysmorphic facies may be subtle but the presence of pulmonary stenosis should suggest the possibility of NS. Most cases of NS are not diagnosed in the newborn.

Failure to thrive with poor feeding and vomiting is severe enough in about one quarter of infants to prompt hospitalization for evaluation, and many require treatment for reflux and undergo tube feedings. In the remainder, there may be slower than average motor development with both sitting up and walking somewhat delayed probably attributed to decrease in muscle tone. Speech is often delayed.

Childhood

Slow growth in early childhood is common with height falling off the growth curve so that by 4–5 years of age, short stature becomes a real concern prompting both a genetic and endocrine evaluation. The majority of children with NS are not recognized until 6–7 years of age. It is important to consider the diagnosis of NS as early as possible so that early intervention can be started. Learning difficulties in school require evaluation of vision and hearing. Both vision problems and hearing loss are relatively common, and early intervention before starting school should be the goal.

Adolescence

The adolescent with NS often feels out of place with peers. Puberty is often delayed so the growth spurt occurring in peers is delayed in NS making the short stature more evident. Although growth hormone deficiency is uncommon in NS, it does occur and an endocrine evaluation is appropriate. Today, there is an increase in the use of growth hormone in NS even without growth hormone deficiency. There is no question that growth hormone treatment will significantly increase growth velocity and also accelerate the usual delay toward puberty. Some feel that growth hormone treatment will increase the estimated adult height but no control study has been carried out to confirm this. For those with very short stature below the third percentile, the addition of 3–4 in. over expected adult height may make daily living activities easier similar to patients with Turner syndrome. The use of growth hormone in all NS patients with more modest short stature is more controversial. Some concerns include the high cost of growth hormone treatment and the message sent that being short is a real handicap and needs treatment. More attention should be given to preparing the NS individual for adulthood. Most patients with NS graduate from high school and many attend college and go on to become teachers, social workers, nurses, and even physicians and lawyers.

Adulthood

The phenotype of NS changes over time (Allanson et al. 1985) and by adulthood many blend into the normal population. The adult with NS has sharper features with a narrow nasal root and a thin bridge. The neck is longer and webbing more prominent and the skin somewhat transparent. It is interesting that more and more adults are being recognized to have NS for the first time after giving birth to an affected child. This is not surprising since there is variation in the overall physical appearance of a NS patient even among affected family members. Both parents should be carefully evaluated when an infant or child is diagnosed with NS. If NS is suspected, this should be confirmed by genetic consultation so parents can be informed of the 50% risk for subsequent offspring.

Outcomes

There is surprising little information on the natural history of NS. Shaw (Shaw et al. 2007) reported a long-term study on 112 NS individuals (age 12–71 years). There were 57 males and 55 females. Mean years of follow-up were 12 years. This study confirmed the need for long-term follow-up of NS patients. The mean age in that study was only 25.3 years and the mean years of follow-up only 12 years. Most patients felt their quality of life was satisfactory or good. A lack of social life and inability to fit in was cited as a problem for some. The only other publication is a report (Noonan 2005a) of 56 patients diagnosed with NS with a range of age 21–59 years. In this report, 12 (23% of 51) reported a diagnosis of depression and were on antidepressant medication. One had a diagnosis of a bipolar disorder and another with oppositional behavior. Two were recovering alcoholics. It was of interest that the majority of the 30 diagnosed in childhood were unmarried (75%), while the 15 diagnosed as adults (85%) were married, and all of those had one to three affected children which had prompted the diagnosis of NS.

Clinical Expression and Pathophysiology

All patients with suspected NS should have a cardiology consultation including an electrocardiogram and echocardiogram. Over 80% of children with NS have some cardiac problem. Valvular pulmonary stenosis is the most common. Fortunately in many, the obstruction is mild and requires only periodic follow-up. Severe pulmonary stenosis may require surgery if the valve is very dysplastic making balloon valvuloplasty unsuccessful. Many with moderate pulmonary stenosis can be treated with cardiac cath. Atrial septal defects are also common and may be isolated or associated with pulmonary stenosis. Nearly every kind of congenital heart disease has been reported. Even with mild lesions, lifelong follow-up is important. Late developments of valvular and vascular disease have been reported. Although hypertrophic cardiomyopathy is present in only 20% of NS patients, there are particular

mutations, such as RAF1, with a very high incidence. For the uncommon infant with NS and symptomatic HCM, close follow-up is essential. The development of heart failure in such an infant should prompt early referral for heart transplant evaluation since the mortality is high. For the remainder, regular follow-up is needed, but in many the cardiac status remains stable and asymptomatic for many years.

Many with NS experience easy bruising which may be attributed to a number of coagulation factor deficiencies such as factor XI, XII, and VIII and less commonly factor IX and II. Thrombocytopenia and platelet dysfunction are common. In most, the symptoms are mild but in cases of excessive bruising, epistaxis, menorrhagia, or an anticipated surgical procedure, a hematology consult should be obtained. Aspirin should be avoided. Some infants with NS have enlargement of the liver and spleen which may be due to a myeloproliferative disorder which resembles the highly fatal juvenile myelomonocytic leukemia (e.g., Kratz et al. 2005). Fortunately in NS with a PTPN11 mutation, this condition has a favorable prognosis with gradual improvement without specific therapy in the majority of such infants.

Lymphatic problems occur in about 20% and include an increased risk of chylous thorax following cardiac surgery but this condition may occur spontaneously and may be difficult to manage. Both pulmonary lymphangiectasia and intestinal lymphangiectasia are rare findings but peripheral edema often seen in infancy which resolves spontaneously may become a problem in adults. Orthopedic problems such as scoliosis and kyphosis require close follow-up and referral as needed.

Neurologic, cognitive, and behavioral aspects of NS are extremely variable and are still poorly understood. Seizures are not common but occur and are usually responsive to common drug therapy. Arnold-Chiari malformation is uncommon but is increasingly being recognized. Although most individuals with NS have normal intelligence as a group, IQ tends to be ten points lower than unaffected family member or that of the general population (Van der Burgt et al. 1999).

There is a difference depending on the gene mutation. SOS1 mutations as a group have normal intelligence and good school performance compared to PTPN11 (Tartaglia et al. 2007) mutations, although specific PTPN11 mutations have no cognitive delay (Cesarini et al. 2009). Muscle weakness results in some clumsiness, and vision may contribute to poor coordination. As a group, they are happy, animated, social, and active children.

Only recently have there been any cognitive, neuropsychological, and behavior studies of NS related to genotype. Pierpont in 2009 (Pierpont et al. 2009) demonstrated that cognitive impairments were common among individuals with a PTPN11 mutation, while all SOS1 individuals had verbal and nonverbal cognitive skills in the normal range. As expected hearing loss, motor dexterity, and parental education levels accounted for significant variability in cognitive outcome. In a more recent paper, Pierpont (Pierpont et al. 2010) studied the language phenotype of children and adolescents with NS. She studied 66 NS patients and found that variation in language skill is closely related to cognitive, perceptual, and motor factors. There was no evidence that specific aspects of language are selectively affected by NS. Venhaeven et al. (2007) reported in 2008 a study of ten NS patients ranging in age from 16 to 59 years. This study confirmed previous studies showing variability in IQ scores but as a whole in the low-average range. In regard to psychoneuroticism, anxiety, and depression, using a study on a self-report instrument (SCL-90-R) showed higher-than-average results of comorbidity in this population. Conversely, testing on quality of life and satisfaction showed average-to-high satisfaction with different aspects of life. Investigators found the participants as "remarkably friendly, cooperative, and very willing to please." They found no behavioral phenotype associated with NS but measurements of social cognition and adaptation appeared to be somewhat impaired suggesting some degree of alexithymia is present. This term refers to impaired ability to identify and communicate one's emotional state. This finding may help explain the report of depression in adult NS patients.

Noonan Syndrome and Autism Spectrum Disorders

It is somewhat surprising that, although NS involves a germline mutation in a pathway playing a major role in brain development, reports of autism in NS are very few. The two cases reported in the literature include a 3-year-old (Ghaziuddin et al. 1994) with a clinical diagnosis of NS but with little details of his appearance provided to confirm a diagnosis of NS although the clinical description of autism is convincing. The second report is of a 13-year-old with a clinical diagnosis of NS (Watanabe et al. 2011) but the physical description given is limited, and no genetic studies were carried out. The description of autism is, however, convincing. A familial case (Tidyman and Rauen 2008) of Leopard syndrome with high-functioning autism spectrum disorder has also been reported, in which a father and three sons with Leopard syndrome all had clinical symptoms consistent with an autism spectrum disorder. Considering the large number of patients with NS from the available published reports, NS would seem to have fewer reports of autism than the general population.

Evaluation and Differential Diagnosis

NS is the most common of a number of conditions due to a gene mutation in the RAS/MAPK pathway (Romano et al. 2010). These include cardio-facial-cutaneous (CFC) syndrome, Costello syndrome (CS), Leopard syndrome (LS), and neurofibromatosis₁ (NF₁). Cardio-facial-cutaneous (CFC) syndrome, Costello syndrome (CS), and Leopard syndrome (LS) are very rare and may be distinguished by genetic testing. About 10% of patients with NF₁ have a Noonan-like phenotype but most of these will demonstrate a neurofibromin deletion. There is considerable similarity in phenotype and associated problems among all these conditions. Gene testing is available. BRAF, MEK1, and MEK2 are the genes in CFC, HRAS in CS, PTPN11 and RAF1 in Leopard syndrome, and neurofibromin in NF1. Other syndromes to be considered include Aarskog syndrome, which is caused by an X-linked recessive

disorder from a mutation in FGD1 gene; fetal alcohol syndrome, which is caused by maternal alcohol consumption; mosaic trisomy 22, which is characterized by similar facial features but with a chromosomal abnormality; and Turner syndrome, which is characterized by loss of one sex chromosome.

Treatment

Treatment for NS has been discussed under the various systems affected. Early recognition is essential as appropriate management can be started early. Early genetic evaluation should be sought. Genetic testing can be diagnostic but about 25% of NS patients still have no specific genetic test available. There is a need for additional studies in neuropsychological and behavioral aspects of NS so that the most helpful interventions can be started early. A recent publication (Romano et al. 2010) for the primary care physician on management guidelines in NS has recently been published. NS and the other RAS-MPK disorders have stimulated basic scientists to seek more knowledge of this pathway not only for its role in fetal development and cancer but the role of these mutated genes as we age. There is hope that some of the adverse associations such as postnatal short stature, learning difficulties, and progressive hypertrophic cardiomyopathy may be improved with appropriate genetic therapy. There is still much to learn about Noonan syndrome.

See Also

- [Aarskog Syndrome](#)
- [Turner Syndrome](#)

References and Reading

- Allanson, J. E., Hall, J. G., Hughes, H. E., Preus, M., & Witt, R. D. (1985). Noonan syndrome: The changing phenotype. *American Journal of Medical Genetics*, 21(3), 507–514.
- Cesarini, L., Alfieri, A., Pantaleoni, F., Vasta, I., Cerutti, M., Petrangeli, V., et al. (2009). Cognitive

- profile of disorders associated with dysregulation of the RAS/MAPK signaling cascade. *American Journal of Medical Genetics Part A*, 149A(2), 165–170.
- Ghaziuddin, M., Bolyard, B., & Alessi, N. (1994). Autistic disorder in Noonan syndrome. *Journal of Intellectual Disability Research*, 38(Pt 1), 67–72.
- Kratz, C. P., Niemeyer, C. M., Castleberry, R. P., Cetin, M., Bergsträsser, E., Emanuel, P. D., et al. (2005). The mutational spectrum of PTPN11 in juvenile myelomonocytic leukemia and Noonan syndrome/myeloproliferative disease. *Blood*, 106(6), 2183–2185.
- Noonan, J. A. (1968). Hypertelorism with Turner phenotype. *American Journal of Diseases of Children*, 116(4), 373–380.
- Noonan, J. A. (2005a). Noonan syndrome. In S. Goldstein & C. R. Reynolds (Eds.), *Handbook of neurodevelopmental and genetic disorders in adults* (pp. 308–319). New York: Guilford Press.
- Noonan, J. A. (2005b). Noonan syndrome and related disorders. *Progress in Pediatric Cardiology*, 20(2), 177–185.
- Noonan, J. A., & Ehmke, D. A. (1963). Associated non-cardiac malformations in children with congenital heart. *Journal of Pediatrics*, 31, 150–153.
- Opitz, J. M. (1985). The Noonan syndrome. *American Journal of Medical Genetics*, 21(3), 515–518.
- Paul, R., Cohen, D. J., & Volkmar, F. R. (1983). Autistic behaviours in a boy with Noonan syndrome. *Journal of Autism and Developmental Disorders*, 13(4), 433–434.
- Pierpont, E. I., Pierpont, M. E., Mendelshon, N. J., Roberts, A. E., Tworog-Dube, E., & Seidenberg, M. S. (2009). Genotype differences in cognitive functioning in Noonan syndrome. *Genes, Brain and Behavior*, 8(3), 275–282.
- Pierpont, E. I., Weismer, S. E., Roberts, A. E., Tworog-Dube, E., Pierpont, M. E., Mendelshon, N. J., et al. (2010). The language phenotype of children and adolescents with Noonan syndrome. *Journal of Speech, Language, and Hearing Research*, 53, 917–932.
- Romano, A. A., Allanson, J. E., Dahlgren, J., Gelb, B. D., Hall, B., Pierpont, M. E., et al. (2010). Noonan syndrome: Clinical features, diagnosis and management guidelines. *Pediatrics*, 126(4), 746–759.
- Shaw, A. C., Kalidas, K., Crosby, A. H., Jeffery, S., & Patton, M. A. (2007). The natural history of Noonan syndrome: A long-term follow-up study. *Archives of Disease in Childhood*, 92(2), 128–132.
- Tartaglia, M., Mehler, E. L., Goldberg, R., Zampino, G., Brunner, H. G., Kremer, H., et al. (2001). Mutations in PTPN11, encoding the protein tyrosine phosphatase SHP-2 cause Noonan syndrome. *Nature Genetics*, 29(4), 465–468.
- Tartaglia, M., Pennacchio, L. A., Zhao, C., Yadav, K. K., Fodale, V., Sarkozy, A., et al. (2007). Gain-of-function SOS1 mutations cause a distinctive form of Noonan syndrome. *Nature Genetics*, 39(1), 75–79.
- Tidyman, W. E., & Rauen, K. A. (2008). Noonan, Costello and cardio-facio-cutaneous syndromes: Dysregulation of the Ras-MAPK pathway. *Expert Reviews in Molecular Medicine*, 10, e37.
- Van der Burgt, I., Toonen, G., Roosenboom, N., Assman-Hulsmans, C., Gabreels, F., Otten, B., et al. (1999). Patterns of cognitive functioning in school-aged children with Noonan syndrome associated with variability in phenotypic expression. *Journal of Pediatrics*, 135(6), 707–713.
- Verhoeven, W. M. A., Wingbermühle, E., Egger, J., Van Der Burgt, I., & Tuinier, S. (2007). Noonan syndrome: Psychological and psychiatric aspects. *American Journal of Medical Genetics*, 146A, 191–196.
- Watanabe, Y., Yano, S., Niihori, T., Aoki, Y., Matsubara, Y., Yoshino, M., et al. (2011). A familial case of LEOPARD syndrome associated with a high-functioning autism spectrum disorder. *Brain and Development*, 33(7), 576–579.

Noradrenaline

► Norepinephrine

Noradrenergic System

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Introduction

Autism spectrum disorders (ASDs) are complex neurodevelopmental disorders with a wide range of behavioral manifestations (Persico and Bourgeron 2006). Research in the neurobiology of ASD will lead to optimal treatments for this heterogeneous group of ASDs, and the impact of ASD can be minimized through intensive intervention, especially during early childhood (Holden and Liu 2005; Johnston and Blue 2006; Lam et al. 2006; Persico and Bourgeron 2006).

Abnormalities in brain structure and function are common in ASD patients, but there is variability among subjects. Several reports have described postnatal macrocephaly in ASD patients (Amaral et al. 2008). Qualitative magnetic resonance imaging (MRI) scans suggest the

occurrence of neuronal migration defects in some cases of ASD, and enlarged occipital and parietal lobes have also been reported. Causes of macrocephaly in ASD patients may include increased neurogenesis, decreased neuronal cell death, and increased production of non-neuronal tissue. Because neurotransmitters and neuromodulators are involved in both the differentiation and the maintenance of neurons, their over- or underproduction or availability may influence the numbers of specific neurons in different regions of the brain (Holden and Liu 2005).

Neurochemical investigations in ASD have examined a wide array of transmitter systems, including serotonin, noradrenaline, dopamine, acetylcholine, oxytocin, glutamate, and gamma-aminobutyric acid (GABA) systems. These studies have been complicated by the fact that ASD is a very heterogeneous disorder, which often presents with comorbid behavioral problems (Johnston and Blue 2006; Lam et al. 2006; Koves and Heinzlmann 2007).

Noradrenaline (NA, also known as norepinephrine), an important classical neurotransmitter derived from dopamine, appears first in the notochord and then in neural crest before the differentiation of neuroblasts (Koves and Heinzlmann 2007). Increasingly complex theories have described the functional role of the noradrenergic system, beginning with vigilance, sensory perception, attention, and memory processes, and culminating in complex models concerning prediction errors, decision making, and unexpected uncertainty.

Neuroanatomy of Noradrenergic System

Initial evidence for the existence of monoamine containing neurons in the central nervous system came from pioneering studies of Dahlstrom and Fuxe (1964). The first description of the cortical distribution of noradrenergic terminals was provided soon after by the same group, followed by a wave of neuroanatomical studies using various methods, and culminating in a definitive autoradiographic study describing the extensive projections from a small pontine nucleus to most regions of the brain.

Neurons that synthesize NA are restricted to the pontine and medullary tegmental regions. Seven noradrenergic cell groups, designated A1-A7, have been described in rodents, and most of these have been observed in primates, including human. Groups A1 and A2 are located in the lower portion of the medulla oblongata. Cells corresponding to group A3 lie just dorsal to the inferior olivary complex and have not been observed in primates. Group A4 consists of a band of subependymal neurons extending along the superior cerebellar peduncle. Group A5 is situated in the caudolateral part of the pontine tegmentum and consists of rather loosely arranged cells. Group A6 is a densely packed accumulation of cells within the locus coeruleus (LC), located in the floor of the fourth ventricle at rostral pontine levels, as described in detail below. The cells of the A7 group are situated in the rostral pontine part of the lateral reticular formation and lie mainly medial to the lateral lemniscus (Dahlstrom and Fuxe 1964; Nieuwenhuys 1985; Koves and Heinzlmann 2007).

There are two major and one minor central noradrenergic pathways. One major pathway arises in the LC as the A4 and A6 regions consisting entirely of noradrenergic neurons. The second noradrenergic pathway arises from neurons diffusely distributed in the brainstem of the subcoeruleus region. A third, periventricular system arises in the dorsal raphe nucleus and projects to the pretectal area, habenula, thalamus, and hypothalamus (Nieuwenhuys 1985).

The LC complex, containing approximately half of the total number of NA-synthesizing neurons, is quantitatively the most important noradrenergic center of the brain. The LC is a small-pigmented nucleus nestled in the rostral dorsolateral pontine tegmentum and, in humans, consists of approximately 40,000 neurons with the most widespread efferent projections of any neurons in the brain. Noteworthy is that NA is crucial for the normal development of Cajal-Retzius cells, which are responsible for the migration of other neurons and the formation of lamination in the cortex (Koves and Heinzlmann 2007). All layers of the cortex are innervated with a regional specificity in the density and distribution of noradrenergic

varicosities. Some areas of the cortex are more highly innervated (Chandler 2016). This innervation has been found to be higher in the frontal cortex, compared to the motor somatosensory and piriform cortices, with the highest found in the anterior cingulate cortex (Agster et al. 2013). Although the prefrontal regions receive the most NA innervation, the insular cortex uniquely is innervated by both the LC and non-LC derived NA containing fibers.

NA functions as a neurotransmitter and/or neuromodulator that can be distributed via the bloodstream, via volume transmission as well as having widespread release sites such as synaptic varicosities. Massive axonal arborizations have huge numbers of release sites (Agnati et al. 2010; Dayan 2012). Neuromodulation can aid in hyperpolarizing or depolarizing neurons, changing their responsivity to input, altering the strengths of synapses, and shaping the plasticity of those synapses. Input from a small number of neuromodulatory cells can abruptly interrupt the activity of neural networks and reorganize the elements into new functional networks (London 2018).

Neurochemical Alterations of NA

NA is a catecholamine that is synthesized from dopamine through the action of the enzyme dopamine beta-hydroxylase. Almost all regions of the brain receive input from noradrenergic neurons, and the cell bodies of the most important system are located in the LC, as described above. The activity of the noradrenergic system is thought to play a critical role in attention, filtering of irrelevant stimuli, stress response, anxiety, and memory. Since many of these functions are impaired in individuals with ASD, researchers have investigated whether noradrenergic functioning is altered. Noradrenergic activity has been assessed in ASD patients by measurement of NA and its central and/or peripheral metabolites in blood, urine, and cerebrospinal fluid (Lam et al. 2006).

Noradrenergic function can be measured in the blood as NA itself, and as its principal central metabolite, 3-methoxy-4-hydroxyphenylglycol.

Unlike some of the other neurotransmitter systems, central and peripheral noradrenergic systems are reported to couple with blood and cerebrospinal fluid concentrations. Some studies examining NA levels in the blood showed higher concentrations in patients with ASD compared to control subjects, whereas the results of other studies failed to show any differences between patients with ASD and normal controls. In addition, the results of studies examining the excretion of NA and its metabolites in patients with ASD have been inconsistent (Lam et al. 2006).

Studies measuring levels of 3-methoxy-4-hydroxyphenylglycol in cerebrospinal fluid showed no significant difference between ASD patients and control subjects. The only consistent abnormal finding with regard to noradrenergic functioning in ASD patients is elevated plasma NA levels, as mentioned above. It is known that plasma NA has an extremely short half-life and largely reflects the state of sympathetic arousal at the time of blood drawing. It is plausible that baseline noradrenergic functioning is normal in subjects with ASD, but that the clinical procedures may lead to hyperarousal and a heightened sympathetic response, resulting in transient high levels of NA in the blood.

Evidence for the relationship of dopamine and NA with ASD was gathered from the studies reported decrease in dopamine- β -hydroxylase activity and increased serum NA levels in children with ASD and in their parents. Study using positron emission tomography (PET) in ASD patients has revealed that increased activity of dopamine transporter (DAT) at the orbitofrontal cortex (Nakamura et al. 2010). A de novo mutation of DAT gene (SLC6A3) in individuals with ASD has been reported (Neale et al. 2012).

NA-Related Genes and the Expression of ASD

Genes Involved in the Synthesis of NA

NA is synthesized from dopamine by dopamine-beta-hydroxylase. Therefore, it is of interest that mothers of children with ASD had a higher frequency of alleles associated with lower

dopamine-hydroxylase activity, suggesting that maternal genes may influence early development of the fetal brain. Tyrosine is synthesized from phenylalanine by phenylalanine hydroxylase, and defects in the gene for phenylalanine-hydroxylase result in phenylketonuria, which is often associated with ASD. Tyrosine hydroxylase is the rate-limiting enzyme in the synthesis of dopamine and NA. The tyrosine hydroxylase gene is located on the short arm of chromosome 11 at 11p15 and has been examined as a candidate gene in a number of different populations, including schizophrenia (Holden and Liu 2005).

Genes Involved in the Catabolism of NA

Monoamine oxidase A (MAO-A) catalyzes the oxidation of three neurotransmitters (dopamine, NA, and serotonin) implicated in the pathogenesis of neurobehavioral disorders. The MAOA gene is located on the X-chromosome at p11.23–0.4. Mutations or variations that affect levels of this enzyme may result in changes in the levels of NA. Both aggression and borderline retardation have been reported in a family with a point mutation in the MAOA gene. In addition, MAOA deficient mice show both enhanced fear conditioning and passive avoidance learning. A frequent occurrence of aggressive behaviors and apparent lack of emotional responses in some individuals with ASD have been reported. Thus, this gene is considered a candidate gene for either an etiological role in ASD or in modifying the phenotype in affected individuals (Holden and Liu 2005). A variable number tandem repeat polymorphism in the promoter region of the MAOA gene has been reported to be associated with several psychiatric disorders. The two most common polymorphisms are the 3- and 4-repeat alleles, which are thought to be associated with decreased and increased transcriptional activity, respectively (Roohi et al. 2009).

Genes Involved in the Uptake of NA

Genes affecting the synthesis and breakdown of NA and NA transporters, as well as receptors, can affect the effective level of dopamine and NA available at the synapse. Therefore, these genes should be considered as candidate genes for ASD.

The NA transporter is expressed in the placenta and is exposed to maternal blood, suggesting a role for this protein in the transplacental transport of the neurotransmitter to the fetus. Altered transport, either increased or decreased, is likely to affect exposure of the fetus to inappropriate neurotransmitter levels, resulting in abnormal brain development. The NA transporter gene is located on chromosome 16 (16q12.2). Using linked polymorphisms, these transporter genes appear to be important in susceptibility to ASD (Holden and Liu 2005). A de novo mutation of DAT gene in individuals with ASD has been reported (Neale et al. 2012). The NA transporter gene (SLC6A2) was found not to be associated with cognitive and behavioral phenotypes of patients with ASD (Park et al. 2014). Direct postmortem study of ASD found no difference in the total cell count, the volume of the LC, and the numerical density.

The New Theory

The theory proposed by Mehler and Purpura (2009) is that ASD is caused by impaired regulation of the LC. The theory stems from decades of anecdotal observations that some children with ASD exhibit improved behaviors and enhanced communication during febrile episodes. The behavioral-state changes associated with fever in ASD are considered to depend upon normalization of a functionally impaired the LC-noradrenergic system. Fever transiently restores the modulatory functions of the LC-noradrenergic system and ameliorates autistic behaviors (Mehler and Purpura 2009). There is also evidence for separate corticotropin-releasing hormone inputs to the LC that specifically modulate noradrenergic neuronal activation by physiological and environmental stressors (Mehler and Purpura 2009). Prenatal stressors, appropriately timed and sufficiently intense, could also be important in dysregulating the LC-noradrenergic system. LC neurons may be selectively vulnerable to stress-induced functional dysregulation. Prenatal stressful events are reported more frequently in mothers of children with ASD than in mothers of normal control children. A significant increase in ASD

prevalence following maternal exposure to hurricanes and tropical storms has been reported. Furthermore, maternal exposure to severe storms at mid-gestation results in the highest prevalence of ASD in affected cohorts. LC activation is considered to occur when there is a change in environmental imperative, such as the appearance of a novel, unexpected event, or a change in stimulus-reinforcement contingencies. In trials, LC neurons are driven by stimuli that require a rapid behavioral adjustment such as a preparatory signal or an unexpected reward. The LC-noradrenergic system is dysregulated by the interplay of environment, genetic, and epigenetic factors, such as chemical substances both within, as well as outside the genome, that regulate the expression of genes. It is believed that stress plays a central role in the dysregulation of the LC-noradrenergic system, especially in the latter stages of prenatal development when the fetal brain is particularly vulnerable.

The new hypothesis has been proposed by London (2018) to reframe the conceptualization of ASD. Much of the pathology observed in ASD is due to inadequate regulation of circuits rather than intrinsic flaws in the circuitry themselves and the activating and deactivating of circuitry is a central function of the noradrenergic system that plays a central role and explains much of the dysregulation which create the symptoms present in ASD, although other neuromodulators play an important role. Flaws in the functioning of the noradrenergic system are likely not due to primary pathology in the LC neurons themselves, but rather might be secondary to afferent input to the LC or a deficit in the circuitry. This hypothesis needs to be understood as dysregulation rather than hyper- or hypo-functioning. Thus, ASD could be conceptualized as a disorder largely due to a failure of homeostatic mechanisms.

Clinical Implications

Benefits from NA agonists and antagonists in ASD patients have been reported, inconsistent, and clouded with adverse events. Tricyclic antidepressants such as imipramine, desipramine, and nortriptyline are NA reuptake blockers, although they

have other actions, such as dopamine presynaptic reuptake blockade. Both desipramine and clomipramine reduced hyperactivity, whereas clomipramine had additional effects on stereotypic, compulsive, and ritualistic behavior (Lam et al. 2006). Alpha 2 agonists such as clonidine and guanfacine, which dampen NA action, may be helpful for managing hyperactivity, impulsivity, and irritability in young people with ASD. These findings suggest a role of the noradrenergic system in ASD or some of the behaviors that are often present in persons with ASD (Holden and Liu 2005). Postsynaptic beta-adrenergic blockers such as propranolol and nadolol may be useful for managing aggression, self-injury, and agitation (Ward et al. 2013), but they appear to have no consistent effect on manifestations of ASD (Lam et al. 2006). The use of molecular biology to validate clinical psychiatric phenotypes in children with ASD may clarify the response to treatment and long-term clinical management (Roohi et al. 2009).

The neuromodulation of the brain of individuals with ASD could be a powerful strategy, as virtually all of the medications used in psychiatry function as neuromodulators operating on the serotonin, dopamine acetylcholine and GABA, as well as NA neuron systems. Through recognition of the neuromodulatory mechanisms for the symptoms, treatments can be devised (London 2018).

References and Reading

- Agnati, L. F., Guidolin, D., Guescini, M., Genedani, S., & Fuxe, K. (2010). Review: Understanding wiring and volume transmission. *Brain Research Reviews*, 64, 137–159.
- Agster, K. L., Mejias-Aponte, C. A., Clark, B. D., & Waterhouse, B. D. (2013). Evidence for a regional specificity in the density and distribution of noradrenergic varicosities in rat cortex. *Journal of Comparative Neurology*, 521, 2195–2207.
- Amaral, D. G., Schumann, C. M., & Nordahl, C. W. (2008). Neuroanatomy of autism. *Trends in Neurosciences*, 31, 137–143.
- Chandler, D. J. (2016). Evidence for a specialized role of the locus coeruleus noradrenergic system in cortical circuitries and behavioral operations. *Brain Research*, 641, 97–206.
- Dahlstrom, A., & Fuxe, K. (1964). Evidence for the existence of monoamine-containing neurons in the central

- nervous system. *Acta Physiologica Scandinavica*, 62 (Suppl 232), 1–55.
- Dayan, P. (2012). Twenty-five lessons from computational neuromodulation. *Neuron*, 76, 240–256.
- Holden, J. J., & Liu, X. (2005). The roles of dopamine and norepinephrine in autism: From behavior and pharmacotherapy to genetics. In L. Bauman & L. Kemper (Eds.), *The neurobiology of autism* (2nd ed., pp. 303–318). Baltimore: The Johns Hopkins University Press.
- Johnston, M., & Blue, M. (2006). Neurobiology of autism. In T. Tuchman & I. Rapin (Eds.), *Autism: A neurobiological disorder of early brain development* (pp. 80–92). London: Mac Keith.
- Koves, K., & Heinzlmann, A. (2007). Neurotransmitters and neuropeptides in autism. In B. S. Mesmère (Ed.), *New autism research development* (pp. 25–85). Nova Science Publishers: New York.
- Lam, S. L., Aman, M. G., & Arnold, L. E. (2006). Neurochemical correlates of autistic disorders: A review of the literature. *Research in Developmental Disabilities*, 27, 254–289.
- London, E. B. (2018). Neuromodulation and a reconceptualization of autism Spectrum disorders: Using the locus Coeruleus functioning as an exemplar. *Frontiers in neurology* www.frontiersin.org. Published 19 Dec 2018. 9, Article 1120.
- Mehler, M. F., & Purpura, D. P. (2009). Autism, fever, epigenetics and the locus coeruleus. *Brain Research Reviews*, 59, 388–392.
- Nakamura, K., Sekine, Y., Ouchi, Y., Tsujii, M., Yoshikawa, E., & Futatsubashi, M. (2010). Brain serotonin and dopamine transporter bindings in adults with high functioning autism. *Archives of General Psychiatry*, 67, 59–68.
- Neale, B. M., Kou, Y., Liu, L., Ma'ayan, A., Samocha, K. E., & Sabo, A. (2012). Patterns and rates of exonic de novo mutations in autism spectrum disorders. *Nature*, 485, 242–245.
- Nieuwenhuys, R. (1985). *Chemoarchitecture of the brain* (pp. 11–52). Berlin: Springer.
- Park, S., Park, J. E., Cho, S. C., Kim, B. N., Shin, M. S., & Kim, J. W. (2014). No association of the norepinephrine transporter gene (SLC6A2) and cognitive and behavioral phenotypes of patients with autism spectrum disorder. *European Archives of Psychiatry and Clinical Neuroscience*, 264, 507–515.
- Persico, A. M., & Bourgeron, T. (2006). Searching for ways out of the autism maze: Genetic, epigenetic and environmental clues. *Trends in Neurosciences*, 29, 349–358.
- Roohi, J., DeVincent, C. J., Hatchwell, A. E., & Gadow, K. D. (2009). Association of a monoamine oxidase-a gene promoter polymorphism with ADHD and anxiety in boys with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 39, 67–74.
- Ward, F., Tharian, P., Roy, M., Deb, S., & Unwin, G. L. (2013). Efficacy of beta blockers in the management of problem behaviours in people with intellectual disabilities: A systematic review. *Research in Developmental Disabilities*, 34, 4293–4303.

Norepinephrine

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Synonyms

Noradrenaline

Definition

Norepinephrine (NE) is a central and peripheral neurotransmitter. The cell bodies of most NE neurons in the brain are located in the brain stem, but project throughout the central nervous system where they are especially important in arousal and attention. NE is also the principal neurotransmitter of the sympathetic nervous system in the periphery (the body), a system critical in arousal and which is closely integrated with the central NE system. Norepinephrine is produced from the essential amino acid tyrosine after ring and side-chain hydroxylation and decarboxylation. Measurement of NE and a range of related compounds in cerebrospinal fluid, plasma, and urine have indicated that global basal function of central and peripheral NE appears unaltered in autism; however, the NE systems may be hyperresponsive to stress.

See Also

► [Neurotransmitter](#)

References and Reading

- Cook, E. H. (1990). Autism: Review of neurochemical investigation. *Synapse*, 6(3), 292–308.
- Lam, K. S., Aman, M. G., & Arnold, L. E. (2006). Neurochemical correlates of autistic disorder: A review of the literature. *Research in Developmental Disabilities*, 27(3), 254–289.
- Minderaa, R. B., Anderson, G. M., Volkmar, F. R., Akkerhuis, B. S., & Cohen, D. J. (1994). Noradrenergic and adrenergic functioning in autism. *Biological Psychiatry*, 36, 237–241.

Normal

► Neurotypical

Normal Curve

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Synonyms

[Bell-shaped curve](#); [Normal distribution](#); [Standard normal distribution](#)

Definition

The normal curve represents the shape of an important class of statistical probabilities (see Fig. 1 below). The normal curve is used to characterize complex constructs containing continuous random variables. Many phenomena observed in nature have been found to follow a normal distribution. Some human attributes such as height, weight, intelligence, and even social skills can be said to be normally distributed. For example, most people's height clusters around the population mean, and an equally small proportion of people are represented at either extreme end of

the distribution. When represented graphically, the resulting shape resembles that of a bell where there is a single peak at the mean, while the tails extend to the right and left into infinity. Thus, the probability of being 5'10" is relatively high, while the probability of being 7'4" is much smaller. Raw data from any number of disciplines can be transformed into *standard scores* using a simple formula. Once scores have been standardized, the mean of the curve = 0 and the standard deviation = 1. This is referred to as the *standard normal distribution*. Performance on most major standardized tests of intelligence produces standardized scores defined as IQ score and reflects a population mean of 100 and a standard deviation of 15 points. The normal curve is the most prominent probability distribution model used in statistics and psychometrics.

All normal distributions comply with the following postulates:

- 68% of all scores fall within + or – 1 SD from the M.
- 95% of all scores fall within + or – 2 SD from the M.
- 99% of all scores fall within + or – 3 SD from the M.

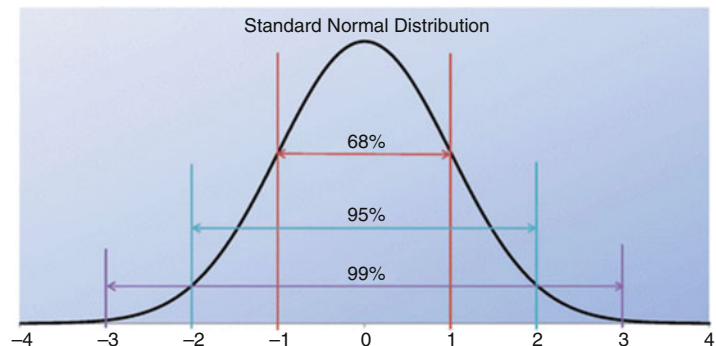
See Also

- [Intelligence Norms](#)
- [Intelligence Quotient](#)
- [Psychological Assessment](#)
- [Standard Scores \(Z and T Scores\)](#)

N

Normal Curve,

Fig. 1 Standard normal distribution depicting the percentage of cases falling within 1, 2, and 3 standard deviations from the mean



References and Reading

- Anastasi, A., & Urbina, S. (1997). *Psychological testing* (7th ed.). Upper Saddle River: Prentice-Hall.
- Aron, A., Aron, E. N., & Coups, E. J. (2008). *Statistics for psychology* (5th ed.). Upper Saddle River: Pearson Prentice Hall.
- Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). Mahwah: Lawrence Erlbaum Associates.
- Gregory, R. J. (2004). *Psychological testing: History, principles, and applications* (4th ed.). Boston: Allyn and Bacon.
- Keppel, G., & Wickens, T. D. (2004). *Design and analysis: A researcher's handbook* (4th ed.). Upper Saddle River: Pearson Prentice Hall.

Normal Distribution

► Normal Curve

Normal Language Development

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Synonyms

[Child language](#); [Language acquisition](#)

Definition

Normal language development involves the acquisition of the rules for producing and understanding the sounds, words, sentences, and conventions for their socially appropriate use in the speech community in which a child is living. Language acquisition is a uniquely human capacity. Although there are many forms of animal communication, these do not show the grammatical generativity or cultural transmission characteristic of human languages (Tomasello

2008). Milestones of communication acquisition, such as first words by 12 months, word combinations by 24 months, and sentences by 36 months, are often used to monitor language growth in children and help parents and professionals determine whether a child is showing signs of falling behind and needing intervention. “Communicative competence,” the ability to use language in an age-appropriate way to accomplish social goals, depends on the acquisition of skills in each of three domains of language specified by Bloom and Lahey (1978): form, content, and use. Form refers to the acquisition of rules for producing sounds to form words (phonology) and words to form sentences (grammar); content refers to vocabulary development and understanding the relationships between words (semantics); use refers to pragmatic aspects of language that govern its social appropriateness. Early communication warning signs for infants or toddlers who may be at risk for developmental disorders such as hearing impairment, autism, language, or developmental delay include failure to respond to sound or voice by 4–6 months; failure to babble or respond to name by 12 months; lack of awareness of and interest in others by 12 months; failure to talk, attempt to communicate, or show interest in pretending by 18 months; producing very few words by 24 months; and failing to combine words by 30 months. In addition, any child who loses previously developed speech-language or social skills may be evidencing risk. Children presenting with these signs should receive evaluations for developmental disabilities with experienced early interventionists. It is very common for children with ASD to show delays in language development; however, many children (10–15% of toddlers) show delays in language acquisition that are NOT associated with ASD. In these children, 75% resolve their language delays by the age of 5 and go on to have adequate language and school achievement.

See Also

- [Expressive Language](#)
- [Receptive Language](#)

References and Reading

- American Speech-Language-Hearing Association. (1997–2010). How does your child hear and talk? In www.ASHA.org. Retrieved October 28, 2010, from <http://www.asha.org/public/speech/development/chart.htm>
- Bloom, L., & Lahey, M. (1978). *Language development and disorders*. New York: Wiley.
- Tomasello, M. (2008). *Origins of human communication*. Cambridge: MIT Press.

Normalization

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Synonyms

[Developmentally appropriate practice](#)

Definition

A principle that promotes providing services for individuals with developmental disorders (e.g., autism spectrum disorders, or ASDs) and learning disorders in order to ensure their access to environments and experiences as similar as possible to those available to typically developing individuals. Nirje (1980) defined normalization as making “the regular circumstances and ways of life or society” (p. 33) available to individuals with learning difficulties.

When first adopted, normalization was instrumental in reducing the frequently inhumane institutionalization of these individuals and providing both community-based and other alternative services. The normalization movement began in the 1960s and 1970s in Scandinavia, with other European countries and the United States following. Initially, normalization served as a philosophical foundation for reorganizing the provision of services for individuals with developmental delays (Rehm and Bradley 2005). Following the normalization-inspired

deinstitutionalization movement, some interventionists and researchers have contended that normalization is not the ideal approach to current interventions for ASDs and have argued instead for the necessity of a more individual-focused and strength-based philosophy (e.g., Mesibov 1990). However, normalization has continued to be an overarching philosophy in approaches to treatment for ASDs and other developmental disabilities.

Normalization reflects the need for individuals with ASDs and other disabilities to have access to equality, quality of life, and human rights (Renzaglia et al. 2003). This concept serves as an ideological framework for inclusion, a principle emphasizing the need for programs to support integration of individuals in schools, neighborhoods, and communities, regardless of disability status. The Americans with Disabilities Act and similar laws in other countries, which prohibit discrimination against individuals based on disability, are also consistent with normalization.

In theory, normalization is designed to be applied along a continuum of disability, with various levels of inclusion possible, depending on the level of impairment. Because of the difficulty of conducting randomized controlled studies of normalization practice, current evidence on the outcomes of inclusion practices in schools is preliminary (Ferraioli and Harris 2011). Inclusion of children with ASDs who have not received early intensive behavioral intervention or who have not made significant treatment gains is not supported, but inclusion for children who have made these gains can more often be feasible and successful (Ferraioli and Harris 2011).

See Also

- [Academic Supports](#)
- [Americans with Disabilities Act](#)
- [Inclusion](#)

References and Reading

- Ferraioli, S. J., & Harris, S. L. (2011). Effective educational inclusion of students on the autism spectrum. *Journal of Contemporary Psychotherapy*, 41, 19–28.

- Mesibov, G. B. (1990). Normalization and its relevance today. *Journal of Autism and Developmental Disabilities*, 20, 379–390.
- Nirje, B. (1980). The normalization principle. In R. J. Flynn & K. E. Nitsch (Eds.), *Normalization, social integration, and community services* (pp. 31–50). Baltimore: University Park Press.
- Rehm, R. S., & Bradley, J. F. (2005). Normalization in families raising a child who is medically fragile/technology dependent and developmentally delayed. *Qualitative Research*, 15, 807–820.
- Renzaglia, A., Karvnone, M., Drasgow, E., & Stoen, C. (2003). Promoting a lifetime of inclusion. *Focus on Autism and Other Developmental Disabilities*, 18, 140–149.

weight-for-age norms provided by the CDC (Kuczmarski et al. 2002) and WHO (WHO Multicentre Growth Reference Study Group 2007) are examples.

See Also

- [Standard Scores \(Z and T Scores\)](#)
- [Standardization](#)
- [T Scores](#)
- [Vineland Adaptive Behavior Scales \(VABS\)](#)
- [Wechsler Preschool and Primary Scale of Intelligence](#)

Normative Data

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Synonyms

[Benchmark data](#); [Norms](#); [Population norms](#)

Definition

Normative data is data from a reference population that establishes a baseline distribution for a score or measurement, and against which the score or measurement can be compared. Normative data is typically obtained from a large, randomly selected representative sample from the wider population. They can be used to easily transform individual scores or measurements directly into standardized z-scores, T scores, or quantiles. Examples of psychological tests that make use of normative data in scoring include the Wechsler Adult Intelligence Scale (WAIS), the Wechsler Intelligence Scale for Children (WISC), and the Vineland Adaptive Behavior Scales. Normative data can also incorporate additional variables such as age and gender, when these variables are expected to have significant effects on the distribution of measurements; head-circumference-for-age, height-for-age, and

References and Reading

- Kuczmarski, R. J., Ogden, C. L., Guo, S. S., Grummer-Strawn, L. M., Flegal, K. M., Mei, Z., . . . Johnson, C. L. (2002). 2000 CDC growth charts for the United States: Methods and development. *Vital Health Statistics*, 11(246), 1–190.
- Mitrushina, M. N., Boone, K. B., Razani, J., & D'Elia, L. F. (2005). *Handbook of normative data for neuropsychological assessment*. Oxford: Oxford University Press.
- WHO Multicentre Growth Reference Study Group. (2007). *WHO child growth standards: Head circumference-for-age, arm circumference-for-age, triceps skinfold-for-age and subscapular skinfold-for-age – Methods and development*. Geneva: World Health Organization.

Norm-Referenced Assessment

- [Language Tests](#)

Norm-Referenced Testing

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Norm-referenced testing measures by comparing a characteristic of an individual with the same

characteristic in comparable group of others, the normative group.

Assessment or evaluation of individuals with ASD commonly involves psychological or psychometric tests. These are systematic (rule-governed or standardized) procedures devised to measure psychological characteristics. Commonly, these attributes, characteristics, states, and functioning are not directly observable but are inferred from behavior – what people say and do. Psychological tests are designed to provoke specific instances of behaviors believed to reflect the attribute of interest, enabling these to be quantified: getting someone to complete shape puzzles and then counting the number correctly completed is a way of quantifying behaviors assumed to reflect underlying spatial abilities; or the count of the number of “Yes” answers to questions about the presence or absence of certain characteristics might be used as the basis of an index of the severity of ASD characteristics.

The number of correctly completed puzzles or other index derived directly from performance constitutes the “raw score” which, on its own, is meaningless. Meaning is achieved when the raw score is compared with some standard or norm, such as the average performance of comparable individuals – a normative group – on the same tasks. The number of correct responses or answers can be compared with the group average: if above the group average, it is then inferred that their *ability* is above (the group) average. Likewise, a scale to identify severity of ASD symptoms may contain a list of clinical signs and a severity rating scale for each. The total of the severity ratings when compared with the average score of a group of individuals diagnosed with ASD provides a measure of severity. Generally, where an individual’s raw score on a test can be interpreted in relation to an index from a group given the same test, the procedure is a “norm-referenced” test. There are a number of different but interchangeable ways of expressing this comparative relationship using more refined units, including percentiles, standard scores, T-scores, and stanines (Kaplan and Saccuzzo 2009).

The nature of the normative data is a key determinant of the interpretability of any test. If the psychological attribute is something common to people, for instance, intelligence, the normative criterion should be derived from a large sample representative of the population with given the same test. This would enable interpretations such as “X’s” intelligence (a test score from a test designed to measure intelligence) is above, similar to, or below the average. A norm-referenced test for a particular feature of ASD such as degree of social interest among individuals with ASD would require its reference group to be a representative sample of individuals reliably diagnosed with ASD given the same procedure.

Appropriate representative sampling is one of the many requirements for a *clinically acceptable test*. Other requirements include instrument validity (evidence that it measures what it is supposed to) and other indicators of test quality (Groth-Marnat 2009; Kaplan and Saccuzzo 2009). A *clinically acceptable test result* will depend on the use of a valid test with a normative group whose relevant main characteristics (age, gender, etc.) match those of the testee, cooperation of the testee, and a competent tester.

See Also

► [Language Tests](#)

References and Reading

- Groth-Marnat, G. (2009). *Handbook of psychological assessment* (5th ed.). Hoboken: Wiley.
- Kaplan, R. M., & Saccuzzo, D. P. (2009). *Psychological testing: Principles applications and issues* (7th ed.). Belmont: Wadsworth, Cengage Learning.

Norms

► [Normative Data](#)

Norpramin

► Desipramine

Nortriptyline

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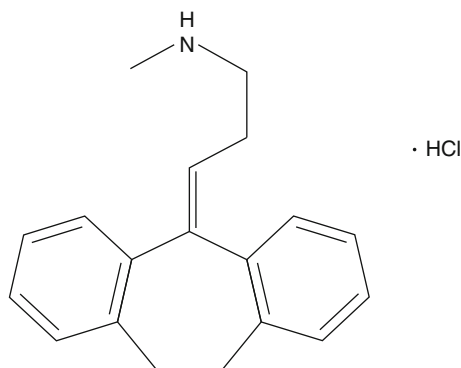
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Synonyms

Aventyl hydrochloride; Nortriptyline hydrochloride

Definition



Nortriptyline is a prescription drug in the group of tricyclic antidepressants initially FDA-approved

for medical use in the year 1964 whose active ingredient nortriptyline hydrochloride has the chemical formula $C_{19}H_{21}N \cdot HCl$. This drug inhibits histamine, 5-hydroxytryptamine, and acetylcholine activity; increases the pressor effect of norepinephrine; and blocks that of phenethylamine. Nortriptyline is metabolized by the liver. This drug is FDA-approved for the treatment of depressive symptoms especially endogenous depression and can be used to treat symptoms of ADHD and Tourette's disorder. Observed side effects include sedation, anticholinergic effects, weight gain, and orthostatic hypotension.

See Also

► Antidepressant Medications

References and Reading

- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001). *Principles and practice of psychopharmacotherapy* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
- Schatzberg, A. F., & Nemeroff, C. B. (Eds.). (2006). *Essentials of clinical psychopharmacology* (2nd ed., pp. 181–197). Washington, DC: American Psychiatric Publishing.
- Stahl, S. M. (2000). *Essential psychopharmacology: Neuroscientific basis and clinical applications*. Cambridge, NY: Cambridge University Press.
- U.S. Food and Drug Administration. (2011). *Aventyl hydrochloride*. Retrieved from <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm?fuseaction=Search.Overview&DrugName=AVENTYL%20HYDROCHLORIDE&CFID=58052189&CFTOKEN=7a0a0c50a125be98-23E871A7-C2D6-F473-9C9052E3B6C515AD>

Nortriptyline Hydrochloride

► Nortriptyline

Norway and Autism

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Historical Background

The history of autism in Norway is not very well documented. However, we know that the first diagnosis using the term autism was applied during the 1950s, as a result of Leo Kanner's seminal work in the early 1940s (Kanner 1943). During the 1950s and 1960s, most individuals diagnosed with autism were institutionalized in centralized institutions, so-called institutions for "the mentally retarded" together with other disabled and impaired individuals. The National Competence Program for Autism released a report in 1997, which dealt with the life span and quality of life for ten of the first diagnosed individuals in Norway (Solbakken 1997). At that time, the individuals were in their late 40s, and the parents which were interviewed were in their elderly days. The general commonalities reported for the cases comprised institutionalizing, few resources, lack of individual facilitation, abuse, and malpractice during the 1950s, 1960s, and parts of the 1970s. Normally, the parents of children who were diagnosed with autism in this period were resourceful and had contacts within the field of autism, whereas others were often diagnosed with oligophrenia. Treatment was often related to sedation by drugs and strapping, and there was a lack of structured behavioral and skill training. The institutionalization was more

custody facility than anything else during this period of time.

After Norway had become an increasingly prospering oil nation in the late 1960s and during the 1970s, major changes happened in both the health sector and also in the educational system. There was a heavy debate in the 1970s and 1980s focusing on the wrongdoings of the larger institutions, and the human right to participate in the society was proclaimed. A law regulating special schools for disabled was incorporated into the general law of education in 1975, and the responsibility for education and training of children with autism was transferred from the state-funded institutions to each county council which resulted in a reduction of the larger institutions and a stronger focus on special schools for children with disabilities. Noteworthy, this implied that children and individuals with autism passed the same system as individuals with other disabilities during the time span from the 1950s to as late as the 1980s. In 1965, the Autism Association of Norway (Autismeforeningen) was established; however, it was not before 1982 that the same society established a professional autism advisory board with the intended goal of securing parents, caregivers, teachers, and special education teachers with professional advice on autism. Both parents and professionals were recommended to partnership through the autism association. Today, the association has approximately 4,500 members across Norway.

Together with a number of new educational laws and governmental regulations during the 1990s and early 2000s, the prevailing special schools were discontinued, and children within the autism spectrum and other disabilities were included into their local kindergarten and school. Though extra resources were made available through assessments, the educational school psychology services enable them to participate in special need education programs provided by specially trained teachers. Over the past two decades, the assessment and diagnostic procedures have been conducted by specialized child disability clinics (habilitation units or child and adolescent

psychiatry), which can be found in each county in Norway. Norway has a public healthcare system, securing everyone the right to the same assessment and diagnostic procedures. Children with autism are mostly living at home with their parents, attending regular kindergartens and schools mainstreamed with typically developed children, with follow-up by the local special education advisors. Intervention methods used in Norway during the 1980s, 1990s, and 2000s have been primarily either a combination of visual communication training or TEACCH (Treatment and Education of Autistic and Related Communication Handicapped Children) or ABA (applied behavior analysis) (Lovaas et al. 1981).

Legal Issues, Mandates for Service

Norway has a wide range of services for children with special need and which are governed by law. All children, including those with disabilities such as an autism spectrum disorder, have the right to an equal education that considers each individual's needs. Those with special needs have the right to an individually tailored school day with special education (The Norwegian Education Law 1998). There are different financial aid schemes for individuals with ASD and their families. The Norwegian Work and Welfare Agency (NAV) provides individuals with different impairments of financial aid if their impairment necessitates extra care and supervision. This financial aid shall also ensure stimulation, education, and training at home. The same agency also offers an extra financial aid to those who have additional expenses related to the diagnosis, e.g., if a special diet creates additional costs to the family (The Norwegian Work and Welfare Agency 2014).

Each municipality is responsible for handling applications for subsidies for care from parents or caregivers of children with disabilities such as autism. Such a subsidy can be given as a form of salary that is often given to parents with an additional and heavy workload as parents of a child/children with disabilities (The Norwegian Department of Health and Care 2011). All caregivers of children with disabilities are also entitled to

20 days with full payment to stay at home with sick children.

Diagnosis and Prevalence

With the ICD-10 (1992), which is the diagnostic system used in Norway, the term autism spectrum disorders and the subgroup diagnoses was established in Norway as a way to standardize the diagnostics and assessment of individuals suspected for ASD. There are national guidelines for assessment and diagnostics of children with suspected ASD. The guidelines, however, are not detailed and are subject to different practice at each clinic. A regional study conducted in the southeastern part of Norway indicated that age at diagnosis is during preschool age at approximately 46.4 months and thus resembling similar countries' diagnostic age (Larsen 2015). Another study using the Norwegian Patient Registry indicates that some of the subgroups within ASD often is not diagnosed until late childhood or early adolescence (Surèn et al. 2012).

Sponheim and Skjeldal (1998) reported prevalence estimates in Norway to be approximately 4–5 per 10,000 for childhood autism in a population of children from 3 to 14 years, a somewhat low prevalence compared to other studies at the time, though they focused on autistic disorder specifically and did not recruit children across the whole spectrum. A recent study indicated a fourfold increase with prevalence of 51 per 10,000 when considering the whole range of the spectrum (Isaksen et al. 2012). Other estimates from other studies with Norwegian samples (Posserud et al. 2010; Surèn et al. 2012) indicate that there has been a rise in prevalence of ASD akin to what is typically found in other international studies (e.g., UK and US).

Overview of Current Treatments and Centers

Norway has a public health system, providing all individuals the same rights to assessment and treatment. Each regional hospital and all the

major university hospitals are state funded and provide assessment and some kind of intervention for children with suspected and diagnosed ASD. There are national guidelines for assessment and diagnostics of children with suspected ASD. The guidelines, however, are not detailed and are subject to different practice at each clinic. Current intervention programs provided by the hospital clinics are mainly ABA and derivatives of ABA (such as Early Intensive Behavioral Interventions (EIBI)). Many use versions of Picture Exchange Communication System (PECS), a form of augmentative and alternative communication for children with ASD and limited language. There exist some regions in Norway where pivotal response therapy (PRT) is the most established intervention method; however, this is only the case in a fraction of Norway's 19 counties. The municipalities are also responsible for intervention and training in the context of kindergartens and schools; the most used intervention focus is parts of the TEACCH method, whereas hospital clinics are to a higher degree recommend ABA and PRT also in kindergartens and schools. There are currently agencies in each healthcare region in Norway dealing with among other autism spectrum disorders. Currently, there still exist a few schools that act as resource centers for children with ASD: Nordvoll and Haug in the Oslo region and Frydenlund in Drammen as the most known. They teach a substantial number of pupils, both in the school, but also through supervising other schools working with children with ASD.

Overview of Research

The most significant research in Norway on ASD during the last 10 years is the MoBa study (mother-child study), a longitudinal pregnancy population cohort, following over 100,000 children as they grow older. Linked to this study is the Autism Birth Cohort study (Stoltenberg et al. 2010) with the purpose of identifying all children with ASD in MoBa. The key aims are to disentangle causes of ASD, both genetic and environmental and their interaction, linked to timing of exposure. The most known publication from this study is mother's

usage of folic acid around conception and the reduce risk of having a child with autistic disorder (Surén et al. 2013b). Other Norwegian studies have investigated a range of topics such as Asperger syndrome related to the theory of mind (Kaland et al. 2002, 2008), comorbidity (Gjevik et al. 2015; Bakken et al. 2010; Helverschou et al. 2011), language (Kalandadze et al. 2016; Nordahl-Hansen et al. 2014; Tetzchner and Martinsen 1981; Vulchanova et al. 2012) and ASD related to symptoms of depression (Andersen et al. 2015). Different researchers have also been involved in publications related to screening (Havdahl et al. 2016; Posserud et al. 2009; Stenberg et al. 2014; Øien et al. 2016) and other issues of measures and assessment of persons with ASD (e.g., Bishop et al. 2016; Øien and Eisemann 2016; Nordahl-Hansen et al. 2016). Some of the notable psych-educational treatment studies have mainly targeted preschool children and investigated effects of early intensive behavior interventions (Eikeseth et al. 2002), low-intensity behavioral treatment (Eldevik et al. 2006), and joint attention and joint engagement intervention (Kaale et al. 2014).

(Most studies and researchers on ASD in Norway is conducted or linked to non-private institutions including different universities, university hospitals, and university colleges.)

Social Policy and Current Controversies and Training

The largest organization is the Norwegian Autism Association, which is a parent and care provider organization with approximately 4,500 members with the aim of increasing the knowledge about ASDs among parents, teachers, other professionals, and the individuals with ASDs themselves. Besides this organization, there is a lack of social interest and focus on ASDs in Norway. The latest prevalence study in Norway showed that 0.9% of all children aged 12 were diagnosed with ASDs, and the number for children between 6 and 12 years old was 0.6% (Surén et al. 2013). There are few organized training programs for parents, teachers, and other professionals, with differences between the 19 counties. However,

the regional competence units and hospital clinics offer various courses for parents, teachers, and others who are interested in learning more. Obviously, there is much room for improvement and especially for developing national guidelines for intervention for children and individuals with ASDs in Norway.

See Also

- ▶ DSM-III
- ▶ DSM-III-R
- ▶ ICD 10 Research Diagnostic Guidelines

References and Reading

- American Psychiatric Association. (1987). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Andersen, P. N., Skogli, E. W., Hovik, K. T., Egeland, J., & Øie, M. (2015). Associations among symptoms of autism, symptoms of depression and executive functions in children with high-functioning Autism: A 2 year follow-up study. *Journal of Autism and Developmental Disorders*, 45(8), 2497–2507.
- Autism Association of Norway (Autismeforeningen). (2014, September). Retrieved from www.autismeforeningen.no
- Bakken, T. L., Helverschou, S. B., Eilertsen, D. E., Heggelund, T., Myrbakk, E., & Martinsen, H. (2010). Psychiatric disorders in adolescents and adults with autism and intellectual disability: A representative study in one county in Norway. *Research in Developmental Disabilities*, 31(6), 1669–1677.
- Bishop, S. L., Havdahl, K. A., Huerta, M., & Lord, C. (2016). Subdimensions of social-communication impairment in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 57(8), 909–916.
- Eikeseth, S., Smith, T., Jahr, E., & Eldevik, S. (2002). Intensive behavioral treatment at school for 4-to 7-year-old children with autism: A 1-year comparison controlled study. *Behavior Modification*, 26(1), 49–68.
- Eldevik, S., Eikeseth, S., Jahr, E., & Smith, T. (2006). Effects of low-intensity behavioral treatment for children with autism and mental retardation. *Journal of Autism and Developmental Disorders*, 36(2), 211–224.
- Gjevik, E., Sandstad, B., Andreassen, O. A., Myhre, A. M., & Sponheim, E. (2015). Exploring the agreement between questionnaire information and DSM-IV diagnoses of comorbid psychopathology in children with autism spectrum disorders. *Autism*, 19(4), 433–442.
- Havdahl, K. A., von Tetzchner, S., Huerta, M., Lord, C., & Bishop, S. L. (2016). Utility of the child behavior checklist as a screener for autism spectrum disorder. *Autism Research*, 9(1), 33–42.
- Helverschou, S. B., Bakken, T. L., & Martinsen, H. (2011). Psychiatric disorders in people with autism spectrum disorders: Phenomenology and recognition. In J. L. Matson & P. Sturmey (Eds.), *International handbook of autism and pervasive developmental disorders* (pp. 53–74). New York: Springer.
- Isaksen, J., Diseth, T. H., Schjølberg, S., & Skjeldal, O. H. (2012). Observed prevalence of autism spectrum disorders in two Norwegian counties. *European Journal of Paediatric Neurology*, 16(6), 592–598.
- Kaale, A., Fagerland, M. W., Martinsen, E. W., & Smith, L. (2014). Preschool-based social communication treatment for children with autism: 12-month follow-up of a randomized trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(2), 188–198.
- Kaland, N., Møller-Nielsen, A., Callesen, K., Mortensen, E. L., Gottlieb, D., & Smith, L. (2002). A new advanced test of theory of mind: Evidence from children and adolescents with Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 43(4), 517–528.
- Kaland, N., Callesen, K., Møller-Nielsen, A., Mortensen, E. L., & Smith, L. (2008). Performance of children and adolescents with Asperger syndrome or high-functioning autism on advanced theory of mind tasks. *Journal of Autism and Developmental Disorders*, 38(6), 1112–1123.
- Kalandadze, T., Norbury, C., Nærland, T., & Næss, K. A. B. (2016). Figurative language comprehension in individuals with autism spectrum disorder: A meta-analytic review. *Autism*, 1362361316668652.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Larsen, K. (2015). The early diagnosis of preschool children with autism spectrum disorder in Norway: A study of diagnostic age and its associated factors. *Scandinavian Journal of Child and Adolescent Psychiatry and Psychology*, 3(2), 136–145.
- Lovaas, O. I., Ackerman, A., Alexander, D., et al. (1981). *Teaching developmentally disabled children: The me book*. Austin: PRO-ED.
- Magnus, P., Irgens, L. M., Haug, K., Nystad, W., Skjærven, R., Stoltenberg, C., & MoBa Study Group. (2006). Cohort profile: The Norwegian mother and child cohort study (MoBa). *International Journal of Epidemiology*, 35(5), 1146–1150.
- Nordahl-Hansen, A., Kaale, A., & Ulvund, S. E. (2014). Language assessment in children with autism spectrum disorder: Concurrent validity between report-based assessments and direct tests. *Research in Autism Spectrum Disorders*, 8(9), 1100–1106.
- Nordahl-Hansen, A., Fletcher-Watson, S., McConachie, H., & Kaale, A. (2016). Relations between specific and global outcome measures in a social-communication intervention for children with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 29, 19–29.
- Øien, R., & Eisemann, M. R. (2016). Brief report: Parent-reported problems related to communication, behavior and interests in children with autistic disorder and their impact on quality of life. *Journal of Autism and Developmental Disorders*, 46(1), 328–331.

- Øien, R. A., Siper, P., Kolevzon, A., & Grodberg, D. (2016). Detecting autism spectrum disorder in children with ADHD and social disability. *Journal of Attention Disorders*. <https://doi.org/10.1177/1087054716642518>.
- Posserud, M. B., Lundervold, A. J., & Gillberg, C. (2009). Validation of the autism spectrum screening questionnaire in a total population sample. *Journal of Autism and Developmental Disorders*, 39(1), 126–134.
- Posserud, M., Lundervold, A. J., Lie, S. A., & Gillberg, C. (2010). The prevalence of autism spectrum disorders: Impact of diagnostic instrument and non-response bias. *Social Psychiatry and Psychiatric Epidemiology*, 45(3), 319–327.
- Solbakken, S. (1997). *Autism and lifespan: Perspective on the lifespan of the ten first diagnosed in Norway*. Bodø: The National Program for Competence Development on Autism.
- Sponheim, E., & Skjeldal, O. (1998). Autism and related disorders: Epidemiological findings in a Norwegian study using ICD-10 diagnostic criteria. *Journal of Autism and Developmental Disorders*, 28(3), 217–227.
- Stenberg, N., Bresnahan, M., Gunnes, N., Hirtz, D., Hornig, M., Lie, K. K., et al. (2014). Identifying children with autism spectrum disorder at 18 months in a general population sample. *Paediatric and Perinatal Epidemiology*, 28(3), 255–262.
- Stoltenberg, C., Schjølberg, S., Bresnahan, M., Hornig, M., Hirtz, D., Dahl, C., et al. (2010). The Autism Birth Cohort: A paradigm for gene-environment-timing research. *Molecular Psychiatry*, 15, 676–680.
- Surén, P., Bakken, I. J., Aase, H., et al. (2012). Autism spectrum disorder, ADHD, epilepsy, and cerebral palsy in Norwegian children. *Pediatrics*, 130, e152–e158.
- Surén, P., Bakken, I. J., Lie, K. K., Schjølberg, S., Aase, H., Reichborn-Kjennerud, T., Magnus, P., Øyen, A.-S., Svendsen, B. K., Aaberg, K. M., Andersen, G. L., Stoltenberg, C. (2013a). *I den norske legeförening*. <http://tidsskriftet.no/2013/10/originalartikkel/fylkesvise-forskjeller-i-registrert-forekomst-av-autisme-adhd-epilepsi-og>
- Surén, P., Roth, C., Bresnahan, M., et al. (2013b). Association between maternal use of folic acid supplements and risk of autism spectrum disorders in children. *JAMA*, 309(6), 570–577. <https://doi.org/10.1001/jama.2012.155925>.
- Tetzchner, S. V., & Martinsen, H. (1981). Autism and receptive dysphasia: Evaluation of comparative studies. *Scandinavian Journal of Psychology*, 22(1), 283–296.
- The National Competence Unit for Autism. (2014, September). Retrieved from www.autismeenheten.no
- The Norwegian Health and Care Department. (2011). Care pay. *Applicable law and practice*. Retrieved from <http://www.regjeringen.no/nb/dep/hod/dok/nouer/2011/nou-2011-17/1/1.html?id=660571>
- The Norwegian Institute of Public Health. (2014, September). *Autism birth coherent study*. Retrieved from <http://www.fhi.no/studier/abc-studien>
- The Norwegian Law of Education. (1998, July). *The education law*. Retrieved from <http://lovdata.no/dokument/NL/lov/1998-07-17-61>
- The Norwegian Work and Welfare Agency. (2014, September). *Financial aid*. Retrieved from <https://www.nav.no/no/Person/Familie/Grunn+og+hjelpestnad/Hjelpest%C3%B8nad.961.cms>
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 5–41). Hoboken: Wiley.
- Volkmar, F. R., Cicchetti, D. V., et al. (1992). Three diagnostic systems for autism: DSM-III, DSM-III-R, and ICD-10. *Journal of Autism & Developmental Disorders*, 22(4), 483–492.
- Vulchanova, M., Talcott, J. B., Vulchanov, V., & Stankova, M. (2012). Language against the odds, or rather not: The weak central coherence hypothesis and language. *Journal of Neurolinguistics*, 25(1), 13–30.
- World Health Organization. (1994). *Diagnostic criteria for research*. Geneva: World Health Organization.

No-Tech Communication Device

► Communication Board

Notice of Recommended Educational Placement (NOREP)

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Synonyms

[IEP signature page](#)

Definition

The Notice of Recommended Educational Placement, or NOREP, is a form completed at the end of the process of developing an individualized educational program (IEP) for a student receiving

special education services. It must be provided to parents whenever a school district or other educational entity proposes to change the student's program or placement. The NOREP form summarizes the educational placement for the student and explains the parents' rights to agree or disagree. By signing the NOREP, parents agree to the placement decisions contained within the IEP as well as the IEP goals and specially designed instruction that will govern the child's special education program. If parents disagree with the provisions contained within the IEP and NOREP, the NOREP form provides options for requesting an informal meeting, mediation, or due process hearing. By requesting mediation or a due process hearing, parents of school age children can ensure that their child will stay in his or her current educational placement until dispute resolution occurs. This benefit is called the stay-put requirement or pendency. Parents may agree with portions of the NOREP and IEP and disagree with other parts. In such instances, the parents may indicate the provisions accepted on the NOREP form and note the areas of disagreement.

See Also

- [Due Process](#)
- [Stay-Put Requirement](#)

References and Reading

Individuals with Disabilities Education Improvement Act of 2004, 20 U.S.C. § 1400 et seq.

Novel Antipsychotics

- [Atypical Antipsychotics](#)

Novo-Peridol

- [Haloperidol](#)

N-Pthaloylglutamide

- [Thalidomide](#)

NRDLS

- [Reynell Developmental Language Scales](#)

NRXN1

- [Neurexin 1](#)

Nuclei of Raphe

- [Midbrain Raphe](#)

Nutritional Interventions

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Definition

Nutrition interventions involve supplementing the diet with vitamins, minerals, or other substances in an effort to improve health and alleviate the core symptoms of autism and associated behavior. A common example is for individuals with autism to consume high doses of vitamin B6 combined with magnesium.

Historical Background

Nutritional supplements are often used in an attempt to improve disorders, especially mental

health conditions, and have been used for autism in particular since the 1960s (Levy and Hyman 2008). Bernard Rimland, a proponent of vitamin B6 therapy in particular, traces the origin back to the 1966 report of abnormal metabolites in the urine of children with autism by A. F. Heeley and G. E. Roberts; these metabolites were said to be normalized by vitamin B6 (Rimland 2008). The next article to address the use of vitamins to treat autism was published by Bonisch in 1968, and research continued for the next four decades.

Other nutritional interventions proposed during this time include high doses of vitamins C, A, and D, as well as minerals such as magnesium, iron, selenium, calcium, and zinc. Enzymatic supplements include probiotics and digestive enzymes. Proteins/amino acids, specifically carnosine, tryptophan, and tetrahydrobiopterin, are supplemented because of proposed metabolic disturbances in children with autism (see ► [Amino Acids](#)). Fatty acids (polyunsaturated, omega 3) are administered because they are known to have a role in typical brain development (Levy and Hyman 2008). In addition to dietary supplements and nutrition changes, the following supplements are sometimes used: secretin (see ► [Secretin](#)), melatonin, DMG/TMG (see ► [DMG](#)), piracetam, folate, carnitine, *Ginkgo biloba*, St. John's wort, and inositol (Levy and Hyman 2008; Rossignol 2009).

Rationale or Underlying Theory

Nutritional supplements are given to children with autism because of observed or suspected nutritional, hormonal, metabolic, or digestive abnormalities.

Goals and Objectives

Nutritional supplements are meant to correct nutritional imbalances that some theorize may be responsible for the core symptoms of autism and/or comorbid behavioral disturbances.

Treatment Procedures

Nutritional interventions are supplemented to the diets of children with autism and are administered in pills, capsules, powders, or liquids. A multivitamin/mineral complex may deliver some or all of these vitamins and minerals at once. A common example is combined B6-magnesium treatment.

Efficacy Information

Currently, no reliable evidence exists to support the efficacy of any nutritional supplement for the core symptoms of autism or other behaviors associated with autism. In a 2011 review, Huffman et al. categorized all of the following as weakly supported and in need of more, better research: vitamins, iron, melatonin, fatty acids, and digestive enzymes. Although improvement in social interaction after protein/amino acid supplementation (i.e., carnosine and tetrahydrobiopterin) has been reported, more research is still needed (Huffman et al. 2011). A 2005 Cochrane review of one of the most popular nutrition interventions, combined B6-magnesium treatment, concluded that this treatment cannot be recommended at this time due to methodological weaknesses in the efficacy studies (Nye and Brice 2005). A large majority of the vitamin B6 efficacy studies performed since 1968 (16 of 19) had too many limitations to be included in the review. The remaining three studies found no benefit from B6 therapy but were too small to support definitive conclusions (Nye and Brice). Accordingly, Nye and Brice concluded that at this time, no recommendation can be made regarding this treatment.

Outcome Measurement

Nutrition interventions are intended to reduce autism symptoms and improve adaptive functioning. Therefore, if clinical trials of nutrition interventions are undertaken, outcome measures should include measures of autism symptoms

such as the ADOS and measures of adaptive behavior such as the Vineland. Also, because many nutrition interventions are meant to target other comorbid behavior problems, well-validated measures of these behaviors (i.e., sleep, aggression, self-injury, etc.) should be included.

Qualifications of Treatment Providers

Supplementing diet with nutritional interventions should be discussed with a board-certified physician, as high doses of vitamins, minerals, or other substances may produce side effects.

See Also

- ▶ [Amino Acids](#)
- ▶ [DMG](#)
- ▶ [Mineral Treatments](#)
- ▶ [Secretin](#)

References and Reading

- Huffman, L. C., Sutcliffe, T. L., Tanner, I. S. D., & Feldman, H. M. (2011). Management of symptoms in children with autism spectrum disorders: A comprehensive review of pharmacologic and complementary-alternative medicine treatments. *Journal of Developmental and Behavioral Pediatrics*, 32, 56–68.
- Levy, S. E., & Hyman, S. L. (2008). Complementary and alternative medicine treatments for children with autism spectrum disorders. *Child and Adolescent Psychiatric Clinics of North America*, 17, 1–15.
- Nye, C., & Brice, A. (2005). Combined vitamin B6-magnesium treatment in autism spectrum disorder. *Cochrane Database of Systematic Reviews*, 4, 1–18.
- Rimland, B. (2008). The use of vitamin B6, magnesium, and DMG in the treatment of children and adults with autism. In W. Shaw (Ed.), *Biological treatments for autism and PDD* (3rd ed.). Lenexa: Great Plains Laboratory.
- Rossignol, D. A. (2009). Novel and emerging treatments for autism spectrum disorders: A systematic review. *Annals of Clinical Psychiatry*, 21, 213–236.

Nutritional Issues

- ▶ [Gastrointestinal Disorders and Autism](#)
- ▶ [Medical Conditions Associated with Autism](#)

Nytol[®] Quick Caps [OTC]

- ▶ [Diphenhydramine](#)

Nytol[®] Quick Gels [OTC]

- ▶ [Diphenhydramine](#)

O

Object Permanence

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Definition

Object permanence is the awareness that objects exist when they are no longer in sight. Between 8 and 10 months of age, infants begin to search for objects that disappear, indicating that they understand that objects still exist after they disappear from view.

Piaget (1952) first described this phenomenon as part of the sensorimotor stage of cognitive development. Over the first several months of life, infants gradually become more and more interested in the environment, particularly in toys and objects. Before about 8 months of age, infants lose interest in objects that are removed from plain sight. During the next several months, however, infants begin to mentally represent objects beyond what is perceived in the moment, and thus begin to search for things that are hidden from view. Object permanence has not been extensively assessed in infants and toddlers with ASD but is thought to be intact in preschoolers with ASD (Carpenter et al. 2002; Dawson and McKissick 1984; Sigman and Ungerer 1981).

See Also

- [Piagetian Stages](#)
- [Sensorimotor Development](#)

References and Reading

- Carpenter, M., Pennington, B., & Rogers, S. (2002). Interrelations among social-cognitive skills in young children with autism. *Journal of Autism & Developmental Disorders*, 32(2), 91–106.
- Dawson, G., & McKissick, F. C. (1984). Self-recognition in autistic children. *Journal of Autism and Developmental Disorders*, 14(4), 383–394.
- Piaget, J. (1952). *The origins of intelligence in children*. New York: International Universities Press.
- Sigman, M., & Ungerer, J. (1981). Sensorimotor skills and language comprehension in autistic children. *Journal of Abnormal Child Psychology*, 9(2), 149–165.

Objective

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Synonyms

[Behavioral objectives](#); [Benchmarks](#); [Instructional objectives](#); [Milestones](#); [Performance objectives](#); [Steps](#)

Definition

Objectives are often called instructional objectives in the individual educational plan for children

receiving special education services. These instructional objectives are statements that describe the specific instruction for an individual student and provide criteria to measure the effectiveness of the instruction. Mager's performance objective model describes three essential criteria for an objective. An objective must contain a clearly stated behavior or skill, the conditions under which that behavior or skill will occur, and how that behavior or skill will be measured (criteria) to determine achievement of the objective (Mager 1984). It is imperative that objectives be written in terms that are both observable and measurable. An example of an objective that aligns with this model might be: "When given a piece of primary paper and a pencil, Susie will print her first and last name with 100% accuracy in spelling."

See Also

- ▶ [Annual Review](#)
- ▶ [Behavioral Objective](#)
- ▶ [Individual Education Plan](#)

References and Reading

- Bigge, J. L., & Stump, C. S. (1999). *Curriculum, assessment and instruction*. Belmont: Wadsworth Publishing.
- Gibb, G. S., & Dyches, T. T. (2007). *Guide to writing quality individualized education programs* (2nd ed.). Boston: Pearson Education.
- Mager, R. F. (1984). *Preparing instructional objectives*. Belmont: David S. Lake.

Observational Assessments

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Definition

Observational assessments involve obtaining evaluative information through direct observation.

Although observational assessments could potentially be used in any domain, they most often are used for behavior, social-emotional functioning, and language. A record of the examiner's observations is kept for later interpretation. Considerations in conducting observational assessments include decisions about which specific behaviors to target for observation; when, where, and for how long observations should be conducted; and the validity and reliability of observational data. If observational assessments are reliable, then multiple examiners observing a student's behavior at the same time should have a high degree of agreement in their records of specific behaviors; if observations are valid, they should target behaviors that are well defined and representative of concerns about a student's social-emotional, behavioral, or linguistic functioning.

Interpretation of observational assessments is another important issue. In some situations, as when a teacher has implemented a behavioral or linguistic intervention, post-intervention observational data may simply be interpreted in relation to pre-intervention data on the same student. In other situations, for example, when a child is evaluated for a possible autism spectrum disorder (ASD), observational data may be interpreted with reference to normative data on students of the same age and/or in relation to checklists of behaviors typical of children with ASD.

See Also

- ▶ [Applied Behavior Analysis \(ABA\)](#)
- ▶ [Child Behavior Checklist in Autism](#)
- ▶ [Communication Assessment](#)
- ▶ [Ecological Validity](#)
- ▶ [Validity](#)

References and Reading

- Salvia, J., Ysseldyke, J., & Bolt, S. (2009). *Assessment in special and inclusive education*. Belmont: Wadsworth.
- Steege, M. W., & Watson, T. S. (2009). *Conducting school-based functional behavioral assessments: A practitioner's guide*. New York: Guilford.

Observational Learning

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Definition

Observational learning occurs when a person develops a new behavior or skill after watching someone else receive reinforcement for engaging in the behavior or performing the skill. The process of observational learning occurs without any direct instruction or reinforcement (Catania 2007; MacDonald and Ahearn 2015). Observational learning is comprised of four discrete skills: attention, generalized imitation, delayed imitation, and consequence discrimination. In real-life situations, observational learning occurs in the following sequence: (1) observing someone else perform a behavior, (2) observing the consequence for performing the behavior (i.e., reinforcement), and (3) performing the behavior (MacDonald and Ahearn 2015). An example of this would be a young child watching a sibling play with a parent. In an observational learning situation, the sibling would pick up a toy, play with it in a certain way, and the parent would provide praise and interaction. The observing child might then pick up a toy and play in the same way. This would be a naturalistic example of observational learning in that it involves observation of a behavior (toy play), observation of a consequence (parent praise and interaction), and performing the behavior (toy play in the same way). Although observational learning is not a concept or intervention specific to individuals with autism spectrum disorders (ASD), individuals with ASD benefit from the analysis and interventions related to observational learning.

Historical Background

Early work in observational learning focused on the role of the model in behavior change (Bandura and Huston 1961; Bandura et al. 1963). Bandura et al. (1963) demonstrated that behavior change can occur through observation of a model in a natural context without direct reinforcement. Bandura and Huston (1961) also explored the role of vicarious reinforcement in learning new skills after observing a model. They noted that vicarious reinforcement occurs when there is no extrinsic reinforcement for a behavior. Instead, they hypothesized that intrinsic reinforcement may be relevant in this type of learning situation. Additional descriptions of observational learning have included social watching and imitation (Miller and Dollard 1941), modeling, copying, imitation, echoing, parroting, and vicarious behavior change (Greer et al. 2006).

Kazdin (1973) evaluated whether children who observed other children receiving social reinforcement such as praise for target behavior would then engage in the same behavior. Results of the study demonstrated that the participants who observed children engaging in a target behavior then engaged in those same responses without direct teaching or reinforcement. This study further strengthened the conceptual understanding of observational learning (Birnbrauer 1976; Williams et al. 2004). More recent work differentiates observational learning from imitation and modeling. Although imitating is part of observational learning, observational learning also involves responding based on the observed consequences (MacDonald and Ahearn 2015; Taylor et al. 2012).

Rationale or Underlying Theory

A variety of studies have demonstrated the importance of learning from others and found that many individuals with ASD do not learn in this way. For example, individuals with ASD do not always imitate the behavior of others, which is a primary component of observational learning (Williams et al. 2004). We know that individuals with ASD

often require direct, intensive instruction and reinforcement to learn new skills; yet learning in this manner could be time consuming and may require a high level of resources. Learning through observation is not only foundational in child Development, but it is advantageous in maximizing learning opportunities for individuals with ASD who are exposed to a variety of environments. Learning through observation is not only foundational in child development, but it is advantageous in maximizing learning opportunities. Individuals with an ASD are exposed to numerous locations in which they learn and practice new skills. Across these environments, there is a wide variability in terms of direct reinforcement or support for facilitation of expected behaviors. Given this, an understanding of observational learning may facilitate greater inclusion, participation, and learning across a number of environments for individuals with ASD (Greer et al. 2006).

Conceptual analysis of observational learning: The four areas of observational learning are (1) engaging in a behavior already in the repertoire because of on observation of someone else receiving reinforcement for that behavior (i.e., watching someone do something you have done before and doing it because you see them receive reinforcement), (2) learning a new behavior as a product of watching someone else being taught a behavior (i.e., learning to do something new because you saw someone else receive reinforcement for doing something), (3) the acquisition of conditioned reinforcers through observation (i.e., learning to like new things because you saw someone else engaging with an item or activity), and (4) developing the skill of observational learning (Greer et al. 2006). One conceptual analysis which accounts for the emergence of observational learning is that of automatic reinforcement (Gladstone and Cooley 1979; Bruzek and Thompson 2007). Gladstone and Cooley (1979) described that simply doing the same thing you see someone else doing may itself function as a reinforcer for imitative and observational responses. This is important in understanding why children imitate behaviors that are not immediately reinforced. This type of reinforcement is discussed as intrinsic to the child

(Bandura and Huston 1961) and imperative for a robust, independent repertoire of imitation and observation not facilitated by adults.

Goals and Objectives

The goal of observational learning interventions is to teach individuals new skills or behavior through observation.

Treatment Participants

Research on observational learning has evaluated its impact with typically developing children (Greer et al. 2006) and children and young adults with ASD (Taylor et al. 2012; MacDonald and Ahearn 2015). Research has also included individuals who have other types of disabilities such as developmental delay and chromosomal abnormalities (Rehfeldt et al. 2003; Werts et al. 1996), and Down syndrome (Griffen et al. 1992). Observational learning is an intervention that benefits all people. For individuals with an ASD, observational learning is an effective intervention if the individual has the previously described prerequisite skills to learn in this manner.

Treatment Procedures

The procedures of an observational learning intervention vary based on the environment, the model, and the skill being taught. As previously described, observational learning interventions all include the steps of (1) observing someone else perform a behavior, (2) observing the consequence for performing the behavior (i.e., reinforcement), and (3) performing the behavior (MacDonald and Ahearn 2015). Importantly, all procedures require that individuals have mastered a series of prerequisite skills (referenced previously) prior to implementation. If individuals do not have those skills in their repertoire, instruction around each of those skills must precede an observational learning intervention.

Developing the skill/process of observational learning: Research in the field of observational learning has evaluated interventions to teach the skills related to observational learning, as well as assess for an observational learning repertoire (Taylor et al. 2012; MacDonald and Ahearn 2015). Before teaching observational learning, a relevant skill deficit is identified. Next, a scenario involving someone modeling that skill would be created. Scenarios may be embedded into naturally occurring situations, such as during play time, or contrived, such as when a teacher recreates a situation that is often challenging for the individual (e.g., sharing a toy). Peers who perform the desired skill consistently may be used as a model. The individual then observes the model performing the skill and receiving reinforcement for that skill. Lastly, it is evaluated if the individual then engages in that skill independently. Taylor et al. (2012) first assessed if three children with ASD could monitor their peers' reading and subsequently acquire sight words. Pre-assessment showed that observational learning did not occur when children with ASD watched their peers read sight words and receive praise and tokens for reading. Once the children with ASD were taught to watch and repeat what their peers said, a session was then conducted to again test for observational learning. The study showed that directly teaching observational skills increased monitoring responses in all participants with ASD, enhancing the observational learning repertoire. This subsequently resulted in increased accuracy of reading previously unknown words, after observing their peers read and receive reinforcement for reading. In another example, MacDonald and Ahearn (2015) used several tasks to test whether an individual with an ASD demonstrated an observational learning repertoire. One task involved hiding a preferred food under a cup, something less preferred under a second cup, and nothing under a third cup. One person revealed what was under each cup, sampled the food, and commented (e.g., "Yummy!" or "Ew, gross!"). In the case of the empty cup, the comment was, "There is nothing here." The other person observing was then given an opportunity to interact with the three cups. If they chose the cup with a treat,

this suggested that they demonstrated observational learning. Another task involved setting out toys, some working toys and some broken toys. One person interacted with the toys to show the other person which toys worked and which ones were broken. If the next person selected only the working toys, this suggested that they demonstrated observational learning.

Interventions involving observational learning: In addition to teaching direct skills related to observational learning, the literature in this area has demonstrated the efficacy of observational learning as an intervention across a variety of factors.

Firstly, the types of models used during observational learning have varied. Models have included in vivo peer models (e.g., a child may watch another child say, "car" and then receive a toy car), video models (a child may watch a video of another child saying "car" and then receive a toy car), in vivo adult models (a child may watch an adult say "Hi" to another person and receive a hug), and even auditory stimuli (a child may hear on an audio recording an individual make a statement and receive praise for that statement; Werts et al. 1996). Secondly, research in this area has enhanced skill repertoires across a variety of domains. These include but are not limited to play skills (Bruzek and Thompson 2007), academic skills (Ramirez and Rehfeldt 2009), expressive language (Goldstein and Moussetis 1989), preference for items (Leaf et al. 2012; Singer-Dudek et al. 2011), and reading skills (Rehfeldt et al. 2003; Taylor et al. 2012). Bruzek and Thompson (2007) had young children watch their peers play. They found that simply watching peers play with previously non-preferred items may establish non-preferred items as preferred for young children. The change in preference resulted in those children playing with novel or less preferred items or activities in subsequent play sessions. This application of observational learning provides a simple procedure to increase play skills and may additionally increase the motivation to engage with new items or activities. In another example, Rehfeldt et al. (2003) employed an observational learning procedure with individuals with ASD and other developmental

disabilities. The individuals first observed peers being taught picture names, site words, and matching. Next, they demonstrated the same skills without direct instruction. The study demonstrated the generality of observational learning procedures to academic skills. The authors concluded that observational learning opportunities may be an economical and efficient method for providing instruction to individuals with developmental disabilities. Greer and Singer-Dudek (2008) utilized an observational learning intervention to increase conditioned reinforcers for young children. In this study the researchers evaluated if an item conditioned as a reinforcer through observation would effectively reinforce correct responses during academic instruction. Academic tasks included matching pictures and numbers, site word identification, and counting. Number of correct response across all participants increased, demonstrating the efficacy of observational learning procedures to condition items as reinforcers for academic tasks. In an extension of this research, Singer-Dudek et al. (2011) were also able to establish books as reinforcers for children in a preschool through observational learning. Lastly, observational learning procedures have been implemented across a variety of environments and activities. Settings for these interventions have included but are not limited to, general education classrooms, homes, individual work areas, and special education classrooms. Importantly, many of the interventions have involved educational settings.

Generality of observational learning: Observational learning has also helped to inform the validity of inclusive opportunities for individuals with ASD. We know from previous literature on best practice approaches to instruction for individuals with ASD that discrete trial instruction (DTI) is often the intervention of choice for establishing new skills (Smith 2001). Unfortunately, this type of intervention often fails to include the natural setting or ensure that individuals with ASD are learning from peers. One barrier to successful inclusion and instruction in the natural environment is the variability that may occur. This is no different for procedures which involve observational learning. Variability in a natural

environment may involve the inconsistency of the model, the immediacy or consistency of adult prompting, or the schedule of reinforcement. To counter these types of variability, many observational learning interventions have occurred outside of the natural environment with peer confederates who are trained to engage in a series of responses at a consistent rate. An important study, conducted by Brody et al. (1978), demonstrated that intermittent modeling was as effective as consistent modeling in an observational situation. This study involved typically developing children who had previously mastered the skill of learning through observation. Specifically, the authors of this study found no significant difference in skills learnt when the model occurred in 50% of opportunities, versus 100% of opportunities. These data have important implications for the utility of observational learning interventions with individuals with ASD. Educational teams should ensure that learners have the necessary repertoires to learn through observation and subsequently measure that learning through observation is happening in controlled environments. Once an observational learning repertoire is established, learning through observation should lead to the development of new skills for individuals with ASD.

Efficacy Information

The benefits of observational learning cannot be understated. At minimum, the benefits of observational learning to individuals with ASD include but are not limited to (1) reductions in adult directed instruction, (2) increased time with nondisabled peers, (3) increased learning opportunities, and (4) learning new skills (Werts et al. 1996).

Outcome Measurement

Experimental studies have demonstrated that individuals with ASD who participated in an observational learning intervention developed new skills. Individuals with ASD who were taught the core skills of observational learning

demonstrated a generalized observational repertoire across environments and skills. Importantly, observational learning interventions enhanced individual skill repertoires without direct instruction or reinforcement. The time to skill acquisition varied across studies and interventions. Also, individual modifications were necessary for some participants to acquire skills. A common theme among previous research was that outcomes were dependent on each individual having prerequisite skills described above (e.g., delayed imitation, consequence discrimination).

The effects of observational learning are measured through continuous or discontinuous data which describe the level of independent skills acquired. Additionally, the degree to which these skills generalize and maintain in the absence of observational opportunities are paramount for judging the outcome of observational learning interventions.

Qualifications of Treatment Providers

Although formal qualifications are not required to implement observational learning, professionals implementing observational learning should have educational and practical experience in Applied Behavior Analysis, Psychology, or Education.

See Also

- [Imitation](#)
- [Inclusion](#)
- [Peers, Exposure to](#)
- [Reinforcement](#)

References and Reading

- Bandura, A., & Huston, A. C. (1961). Identification as a process of incidental learning. *Journal of Abnormal and Social Psychology*, 63, 311–318.
- Bandura, A., Ross, D., & Ross, S. A. (1963). Vicarious reinforcement and imitative learning. *Journal of Abnormal and Social Psychology*, 67, 601–607.
- Birnbrauer, J. S. (1976). Mental retardation. In H. Leitenberg (Ed.), *Handbook of behavior modification and behavior therapy* (pp. 361–404). Englewood Cliffs, NJ: Prentice Hall.
- Birnbrauer, J. S., Hopkins, N. R., & Kauffman, J. M. (1981). The effects of vicarious prompting on attentive behavior of children with behavior disorders. *Child Behavior Therapy*, 3, 27–41.
- Brody, G. H., Lahey, B. B., & Combs, M. L. (1978). Effects of intermittent modeling on observation learning. *Journal of Applied Behavior Analysis*, 11, 87–90.
- Bruzek, J. L., & Thompson, R. H. (2007). Antecedent effects of observing peer play. *Journal of Applied Behavior Analysis*, 40, 327–331.
- Catania, A. C. (2007). *Learning* (4th ed.). Cornwall-on-Hudson: Sloan.
- Detguchi, H. (1984). Observational learning from a radical behavioristic viewpoint. *The Behavior Analyst*, 7, 83–95.
- Gladstone, B. W., & Cooley, J. (1979). Behavioral similarity as a reinforcer for preschool children. *Journal of the Experimental Analysis of Behavior*, 23, 357–368.
- Goldstein, H., & Moussetis, L. (1989). Generalized language learning by children with severe mental retardation: Effects of peers' expressive modeling. *Journal of Applied Behavior Analysis*, 22, 245–259.
- Greer, R. D., & Singer-Dudek, J. (2008). The emergence of conditioned reinforcement from observation. *Journal of the Experimental Analysis of Behavior*, 89, 15–29.
- Greer, R., Dudek-Singer, J., & Gautreaux, G. (2006). Observational learning. *International Journal of Psychology*, 41(6), 486–499.
- Griffen, A. K., Wolery, M., & Schuster, J. W. (1992). Triadic instruction of chained food preparation responses: Acquisition and observational learning. *Journal of Applied Behavior Analysis*, 25, 193–204.
- Kazdin, A. E. (1973). The effect of vicarious reinforcement on attentive behavior in the classroom. *Journal of Applied Behavior Analysis*, 6, 71–78.
- Leaf, J. B., Oppenheim-Leaf, M., Leaf, R., Courtemanche, A. B., Taubman, M., McEachin, J., Sheldon, J., & Sherman, J. A. (2012). Observational effects on the preferences of children with autism. *Journal of Applied Behavior Analysis*, 45, 473–483.
- MacDonald, J., & Ahearn, W. H. (2015). Teaching observational learning to children with autism. *Journal of Applied Behavior Analysis*, 48(4), 800–816.
- Masia, C. L., & Chase, P. (1997). Vicarious learning revisited: A contemporary behavior analytic interpretation. *Journal of Behavior Therapy and Experimental Psychiatry*, 28, 41–51.
- Miller, N., & Dollard, J. (1941). *Social learning and imitation*. New Haven: Yale University Press.
- Nikopoulos, C. K., & Keenan, M. (2003). Promoting social initiation in children with autism using video modeling. *Behavioral Interventions*, 18(2), 87–108.
- Ramirez, J., & Rehfeldt, R. A. (2009). Observational learning and the emergence of symmetry relations in

teaching Spanish vocabulary words to typically developing children. *Journal of Applied Behavior Analysis*, 42, 801–805.

- Rehfeldt, R. A., Latimore, D., & Stromer, R. (2003). Observational learning and the formation of classes of reading skills by individuals with autism and other developmental disabilities. *Research in Developmental Disabilities*, 24, 333–358.
- Singer-Dudek, J., Oblak, M., & Greer, R. D. (2011). Establishing books as conditioned reinforcers for pre-school children as a function of an observational intervention. *Journal of Applied Behavior Analysis*, 44, 421–434.
- Smith, T. (2001). Discrete trial training in the treatment of autism. *Focus on Autism and Other Disabilities*, 16, 86–92.
- Taylor, B. A., DeQuinzio, J. A., & Stine, J. (2012). Increasing observational learning of children with autism: A preliminary analysis. *Journal of Applied Behavior Analysis*, 45, 815–820.
- Werts, M. G., Caldwell, N. K., & Wolery, M. (1996). Peer modeling of response chains: Observational learning by students with disabilities. *Journal of Applied Behavior Analysis*, 29, 53–66.
- Williams, J. H., Whiten, A., & Singh, T. (2004). A systematic review of action imitation in autistic spectrum disorder. *Journal of Autism and Developmental Disorders*, 34, 285–299.

See Also

- ▶ [Obsessive-Compulsive Disorder \(OCD\)](#)
- ▶ [Perseveration](#)
- ▶ [Repetitive Behavior](#)
- ▶ [Self-Stimulatory Behavior](#)
- ▶ [Stereotypic Behavior](#)

References and Reading

- Gillott, A., Furniss, F., & Walter, A. (2001). Anxiety in high-functioning children with Autism. *Autism*, 5, 277–286.
- Hermelin, B., & Frith, U. (1991). Psychological studies of childhood Autism: Can Autistic children make sense of what they see and hear? *Focus on Autism and Other Developmental Disabilities*, 6, 6–13.
- Morgan, S. (1988). Diagnostic assessment of Autism: A review of objective scales. *Journal of Psychoeducational Assessment*, 6, 139–151.
- Prior, M., & Macmillan, M. B. (1973). Maintenance of sameness in children with Kanner's syndrome. *Journal of Autism and Childhood Schizophrenia*, 3(2), 154–167.
- Simons, J. M. (1974). Observations on compulsive behaviors in Autism. *Journal of Autism and Childhood Schizophrenia*, 4(1), 1–10.

Obsessive Desire for Sameness

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Definition

Obsessive desire for sameness refers to an aspect of ritualistic and repetitive behaviors and patterns of interest observed in autism spectrum disorders (ASD). It is one criterion in diagnosing ASD and refers to the insistence that aspects of a person's environment (involving items or even people) remain the same in appearance, sequence, or context. Obsessive desire for sameness often results in a restricted range of activities, rigidity of behaviors, resistance to change, and insistence on performing idiosyncratic rituals and routines.

Obsessive-Compulsive Disorder (OCD)

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Short Description or Definition

Obsessive-Compulsive Disorder (OCD): OCD is an anxiety disorder that is defined by the presence of recurring worries (obsessions) and/or repetitive behavior (compulsions). Obsessions and compulsions often occur together, but not in all cases, and

both are not required for the diagnosis. OCD is relatively common condition affecting 2–4% of adults, but less common in children. It appears to have a genetic component, but environmental factors also likely play a role.

Clinical Expression and Pathophysiology

The individual with OCD may describe intrusion of unwanted thoughts having to do with contamination, fears of harm coming to themselves or others in the family, and the strong urge to check possible sources of harm. In many such cases, the compulsive behavior that follows is related to the obsessional worry. For example, the patient who is fearful of contamination may become involved in excessive washing rituals. The patient who fears that harm may come to the self or to the family members may perform various checking behaviors to prevent that harm from occurring. In other cases, the link between the worry and behavior is less clear. For example, some patients may describe the need to carry out a repetitive ritual such as tapping or counting in order to prevent something bad from happening to themselves or others. In such cases, the ritualized behavior has no connection to the worry.

In some cases, the compulsive behaviors are performed without a clear cognition. For example, some individuals will say that they would feel deeply uncomfortable or unsettled if they do not complete a given ritual. This may be the repetition of a routine activity such as opening and closing a door or setting down an object on the table where it will have to be repeated certain number of times in order to achieve a sense of completion. In such cases, there is a need to achieve a physical sense of completion rather than to prevent harm.

Evaluation and Differential Diagnosis

There is interest in OCD because children and adults with autism often have repetitive behavior. The repetitive behavior in children and adults with autism, however, may not be the same as repetitive behavior in individuals with OCD.

For example, children with autism may insist on reading the same book over and over again, or watching the same video over and over again. This is a preferred behavior – i.e., a behavior that the person wants to do. The difficulty for the child and family emerges when the family tries to move the child away from the preferred activity to accomplish activities of daily living. Thus, there is a common feature of repetitive behavior, but in OCD, the repetitive behavior is unwanted and often motivated by the urge to prevent harm.

Treatment

Another potential difference in a repetitive behavior in OCD and the repetitive behavior in autism relates to the issue of anxiety. In individuals with OCD, the compulsive behavior is in fact being performed in order to reduce anxiety. That anxiety could come from fears of harm or contamination or other sources. As the anxiety about this fear increases, the compulsive behavior is performed and anxiety declines. The reduction in anxiety is powerful reinforcement for repeating the behavior. By contrast, anxiety reduction does not appear to be the driving force for repetitive behavior in children and adults with autism – though this possibility warrants careful assessment.

This question about the similarity or difference in repetitive behavior in autism versus OCD has implications for treatment. The SSRIs are commonly used for the treatment of OCD and are generally regarded as effective for the treatment of OCD. The use of SSRIs for the treatment of repetitive behavior in children with autism spectrum disorders has been disappointing (link to citalopram and fluoxetine).

Two studies have shown that the SSRIs are not effective in reducing repetitive behavior in children and adolescents with autism spectrum disorders. In one study, 149 children with autism spectrum disorders and moderate levels of repetitive behavior were treated with citalopram or placebo for 12 weeks. After 12 weeks of treatment, there was no difference in the overall improvement in those on citalopram versus those on placebo. In addition, in measures of a repetitive

behavior, there was no difference between citalopram and placebo groups. Another study involving 158 children with a new formulation of fluoxetine was also negative with no difference on a measure of repetitive behavior between fluoxetine and placebo.

See Also

► [Anxiety](#)

References and Reading

- Autism Speaks. (2009, February). *Autism speaks announces results reported for the Study of Fluoxetine in Autism (SOFIA)*. Retrieved 16 Aug 2011, from Autism Speaks: <http://www.autismspeaks.org/about-us/press-releases/autism-speaks-announces-results-reported-study-fluoxetine-autism-sofia>
- Baron-Cohen, S., & Wheelwright, S. (1999). "Obsessions" in children with autism or Asperger syndrome: Content analysis in terms of core domains of cognition. *The British Journal of Psychiatry*, 175, 484–490.
- Fischer-Terworth, C., & Probst, P. (2009). Obsessive-compulsive phenomena and symptoms in Asperger's disorder and high-functioning autism: An evaluative literature review. *Life Span and Disability*, 12(1), 5–27.
- King, B. H., Hollander, E., Sikich, L., McCracken, J. T., Scahill, L., Bregman, J. D., et al. (2009). Lack of efficacy of citalopram in children with autism spectrum disorders and high levels of repetitive behavior. Citalopram ineffective in children with autism. *Archives of General Psychiatry*, 66(6), 583–590.
- Lehmkuhl, H. D., Storch, E. A., Bodfish, J. W., & Geffken, G. R. (2008). Brief report: Exposure and response prevention for obsessive compulsive disorder in a 12-year-old with autism. *Journal of Autism and Developmental Disorders*, 38(5), 977–981. <https://doi.org/10.1007/s10803-007-0457-2>.
- Mack, H., Fullana, M. A., Russell, A. J., Mataix-Cols, D., Nakatani, E., & Heyman, I. (2010). Obsessions and compulsions in children with Asperger's syndrome or high-functioning autism: A case-control study [Comparative Study]. *The Australian and New Zealand Journal of Psychiatry*, 44(12), 1082–1088.
- Zandt, F., Prior, M., & Kyrios, M. (2007). Repetitive behaviour in children with high functioning autism and obsessive compulsive disorder. *Journal of Autism and Developmental Disorders*, 37(2), 251–259.
- Zandt, F., Prior, M., & Kyrios, M. (2009). Similarities and differences between children and adolescents with autism spectrum disorder and those with obsessive compulsive disorder: Executive functioning and repetitive behaviour. *Autism*, 13(1), 43–57.

Obstetrical Complications/ Risk Factors

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Definition

A range of factors can increase the risk to the developing fetus during pregnancy and at the time of birth. These range from genetic abnormalities in the fetus, complications for the mother in carrying the child to term, and various environmental factors in utero – for example, exposure to alcohol. Although much interest has centered on possible issues of obstetrical risk in autism and although such factors can clearly impact on development of the child, data for children with autism have been somewhat lacking and/or difficult to interpret.

Historical Background

Kanner (1943) suggested that autism is a congenital condition. Recent work has confirmed a very strong genetic basis for autism while leaving open the possibility for the operation of nongenetic factors to some, although relatively to a much smaller, degree (Rutter 2005; Wing and Potter 2002). The early literature on this topic was reviewed by Tsai (1987) and others (e.g., Bryson et al. 1988; Levy et al. 1988; Lanzi et al. 1991; Levy 1991), with conflicting findings. Various difficulties in comparing studies were noted – these related to differences in approach and sample, comparison groups, and so forth. Issues of birth order are also important since obstetric complications differ in frequency based on birth order (this effect may reflect the issue of “stoppage rules” in autism (see Jones and Szatmari 1988)) and are more frequent among first-, fourth-, and later-born offspring in a sibship (Bakan 1971;

Thomson and Barron 1983). A further complication is posed by the fact that obstetrical problems might reflect the underlying genetic abnormality associated with autism, that is, they are best viewed as a consequence rather than a cause; this notion is supported by work that suggests that associations of obstetrical abnormalities and autism are weaker once adjustment is made for parity (Lord et al. 1991). So, for example, individuals with Down syndrome are more likely to have birth complications (Bolton et al. 1994).

Current Knowledge

The numerous possible alternative explanations for a relationship between autism and obstetrical complications have various implications. As noted by Bolton et al. (1997), each of the various hypotheses leads to various predictions. In their 1997 study, Bolton and colleagues evaluated obstetrical risks in families with a child with autism and families with a child with Down syndrome. Both groups had elevated obstetrical risk compared to unaffected siblings. Various factors have been associated with autism risk, but to date none has been sufficiently large in terms of effect to implicate a single factor in the pathogenesis of autism. In their 2011 meta-analysis, Gardener and colleagues reviewed over 60 perinatal and neonatal factors in data drawn from 40 studies. A number of factors that increased autism were identified (e.g., abnormal presentation, low birth weight, and birth injury, among others), but the authors concluded that there was insufficient evidence to implicate any one specific risk factor. Clearly a complication is the substantial methodological variation over the available studies.

Future Directions

As genes and possible brain mechanisms are identified, it may be possible to do a more thorough and hypothesis-guided examination of pre-, peri-, and neonatal risk factors in autism. At present, the influence of genetic factors appears to be strongly predominant.

References and Reading

- Bakan, P. (1971). Handedness and birth order. *Nature*, 229, 195.
- Bolton, P. F., Macdonald, H., Pickles, A., et al. (1994). A case-control family history study of autism. *Journal of Child Psychology and Psychiatry*, 35, 877–900.
- Bolton, P. F., Murphy, M., et al. (1997). Obstetric complications in autism: Consequences or causes of the condition? *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(2), 272–281.
- Bryson, S. E., Smith, I. M., et al. (1988). Obstetrical suboptimality in autistic children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 27(4), 418–422.
- Gardener, H., Spiegelman, D., & Buka, S. L. (2011). Perinatal and neonatal risk factors for autism: A comprehensive meta-analysis. *Pediatrics*, 128(2), 344–355.
- Jones, M. B., & Szatmari, P. (1988). Stoppage rules and genetic studies of autism [published erratum appears in *J Autism Dev Disorders* 1988 Sep;18(3):477]. *Journal of Autism and Developmental Disorders*, 18(1), 31–40.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Lanzi, G., Zambrino, C. A., et al. (1991). Obstetrical suboptimality as risk factor in autistic children. *Brain Dysfunction*, 4(6), 363–370.
- Levy, S. (1991). Note on obstetrical records and autism: A response to Gillberg and Gillberg. *Journal of Autism and Developmental Disorders*, 21(2), 255.
- Levy, S., Zoltak, B., et al. (1988). A comparison of obstetrical records of autistic and nonautistic referrals for psychoeducational evaluations [see comments]. *Journal of Autism and Developmental Disorders*, 18(4), 573–581.
- Lord, C., Mulloy, C., et al. (1991). Pre- and perinatal factors in high-functioning females and males with autism. *Journal of Autism and Developmental Disorders*, 21(2), 197–209.
- Rutter, M. (2005). Genetic influences and autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 425–452). Hoboken: Wiley.
- Thomson, A. M., & Barron, S. L. (1983). *Perinatal mortality in obstetrical epidemiology*. London: Academic.
- Tsai, L. Y. (1987). Pre-, peri-, and neonatal factors in autism. In E. Schopler & G. B. Mesibov (Eds.), *Neurobiological issues in autism* (pp. 180–189). New York: Plenum.
- Wing, L., & Potter, D. (2002). The epidemiology of autistic spectrum disorders: Is the prevalence rising? *Mental Retardation & Developmental Disabilities Research Reviews*, 8(3), 151–161.

Obstipation

► Constipation

Occipital Lobe

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Synonyms

[Occipital lobes](#); [Visual cortex](#)

Definition

The occipital lobes are positioned at the back region of the cerebral cortex. The name “occipital” derives from the overlying occipital bone, which is named from the Latin occiput, or “back of the head.” Functionally, these regions of the cerebral cortex are the main centers for visual processing. For example, this area contains the primary visual cortex as well as an array of other, nearby visual regions specialized for different visual tasks, such as visuospatial processing, color discrimination, and motion perception.

See Also

► [Cerebral Cortex](#)

References and Reading

Watanabe, M., Cheng, K., Murayama, Y., Ueno, K., Asamizuya, T., Tanaka, K., et al. (2011). Attention but not awareness modulates the BOLD signal in the human V1 during binocular suppression. *Science*, 334(6057), 829–831.

Occipital Lobes

► [Occipital Lobe](#)

Occupational Therapy (OT)

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Definition

Definition of Occupation

The term “occupation” is of special significance in the profession of occupational therapy. Occupations are those activities that surround us and occur at various times in our lives. The founders of the profession used the term “occupation” to denote the proper use of time including work, leisure, and self-care behaviors (Meyer 1922/1977). Occupations are those activities that are grouped together by a culture according to their purpose. These occupations may hold special significance or may seem ordinary. For example, the occupation of eating may seem to be an ordinary everyday occurrence, but during special holidays or events, the foods and accompanying environmental situations create a special significance and meaning. Even the everyday occupation of eating a meal may be disrupted when a child has severely limited acceptance of foods or rejects sitting at a table. In this situation, the seemingly ordinary occupation of eating a meal can become challenging. The term occupation serves as the core of the profession of occupational therapy and forms the perspective focused on health, well-being, and participation in those activities that are part of the culture (Kielhofner and Burke 1977). Occupations have been identified and classified into groups as follows (American Occupational Therapy Association [AOTA] 2008):

- Activities of Daily Living (ADLs): care of oneself including bathing, bowel and bladder management, toilet hygiene, dressing, eating including swallowing, self-feeding, functional mobility, sexual activity, personal hygiene, and grooming.

- **Instrumental Activities of Daily Living (IADLs):** activities seen in the care of home and community including care of others whether parents, siblings or children, care of pets, communication management by using equipment effectively, community mobility using both public and private transportation, financial management, health management and maintenance, home establishment and management, meal preparation and clean-up, safety and emergency maintenance, religious observance, and shopping.
- **Rest and Sleep:** these occupations include rest, sleep, and sleep preparation and participation and address the need to establish appropriate habits and routines for rest and sleep.
- **Education:** this occupation includes both formal educational participation and informal personal educational pursuits and participation.
- **Work:** includes activities related to both employment and volunteer work and includes employment interests and pursuits, employment seeking and acquisition skills, job performance, retirement preparation and adjustment, and volunteer exploration and participation.
- **Play:** includes play exploration and participation in various forms.
- **Leisure:** includes leisure exploration and participation in various forms.
- **Social Participation:** these occupations are “organized patterns of behavior that are characteristic and expected of an individual or a given position within a social system” (Mosey 1996, p. 340) and include participation at various levels such as the community, family, and with peers/friends.

These occupations are addressed within the profession of occupational therapy and serve as the foundation for practice. The complexity of occupations has been further studied in the field of occupational science.

Occupational Therapy

Occupational therapy is a client-centered profession frequently referred to as the “art and science

of helping people do the day-to-day activities that are important and meaningful to their health and well-being through engagement in valued occupations” (Crepeau et al. 2009, p. 217). The important characteristic of this profession is the collaboration with the client in the engagement in occupations. This profession includes three key elements of being client-centered, occupation-centered, and evidence-based.

The concept of being client-centered within the profession of occupational therapy recognizes the need for the client to be a collaborative member in the therapeutic process. This can assume many forms. A child may want to learn to play a video game where the family is concerned about that same child’s ability to interact with peers. The occupational therapy practitioner, using a client-centered approach, would incorporate both the use of video games and peer interaction to meet the client needs. In this situation, the client is not singularly the child but the child positioned within a family and society. Client-centered intervention can be used to meet the needs of individuals, organizations, and populations (AOTA 2008). Although most often client-centered is thought of in reference to a single individual, the profession of occupation therapy identifies the client from a broader perspective and services may be used to support organizations such as a school district where occupational therapy is provided to foster stress management through sensory strategies and sensory breaks for all children. Entire populations may also benefit from occupational therapy services such as addressing the occupational needs of homeless children.

The profession of occupational therapy is clearly linked with occupation-centered practice (Crepeau et al. 2009). The focus of intervention on helping a client become successful with day-to-day activities is the hallmark of occupational therapy. This is illustrated when an occupational therapist is working with a child to master brushing teeth or use of utensils during a meal. Within a school setting, this same child may benefit from occupational therapy services focused on peer interaction on the playground or

in the classroom when this child is expected to complete a written assignment.

The profession, from its very beginnings, has sought to provide evidence of the effectiveness of intervention focused on engagement in occupations. To practice evidence-based occupational therapy requires the practitioner to examine three factors (Crepeau et al. 2009). First, the occupational therapy practitioner must know how to “access, evaluate, and interpret relevant research” (p. 219). This requires knowledge of current publications and the type of research literature available. The second element the occupational therapist must address is the collection of appropriate data to support recommended interventions. The final factor within evidence-based occupational therapy requires the practitioner to disseminate this information to the client, so the client can make an informed decision regarding the recommended intervention.

The focus of occupational therapy intervention is not merely on addressing client deficits, although that is within the practice of the profession. Occupational therapy intervention has identified five approaches as follows (AOTA 2008; Dunn et al. 1998):

- Create/promote (health promotion): a disability is not assumed, and intervention is designed to provide an enriched experience, an example can be seen in health promotion activities for adults or children to develop healthy habits for leisure and play.
- Establish, restore, or remediate: intervention is focused on developing the skills or abilities needed for participation in occupations; examples can be seen when an occupational therapist helps a child develop the skills needed to play with peers or when an occupational therapist helps a child develop the motor memory needed for handwriting.
- Maintain: this intervention is designed to preserve client capacities to engage in occupations and is provided to avoid a decline in functional abilities; an example can be seen when an occupational therapist provides organizers for a child’s backpack at the beginning of each school year.
- Modify, compensate, or adapt: this intervention is designed to change the environment,

situation, or setting to allow the client to participate in the occupation; examples can be seen when an occupational therapist suggests use of voice-activated software for a student who has difficulties completing written assignments or the use of a seating cushion for a child who needs movement within a classroom setting.

- Prevent/disability prevention: this approach is designed to prevent problems, and examples can be seen when an occupational therapist provides soft-cushioned pencil grips to the entire classroom of children before they begin to write or the use of a slant board in classrooms to help children assume an upright posture during written assignments.

These forms of occupational therapy intervention reflect the diversity of services provided. The profession has a wealth of knowledge that extends beyond working only on a specified disabling condition and includes a wide range of clients with occupational needs.

Historical Background

History

Occupational therapy as a profession started in 1917, but the foundations for this profession dedicated to occupational engagement were established far earlier than that date. Prior to the 1800s, individuals who displayed mental health disorders were treated with approaches meant to control their behavior instead of treat the behavior. Those with mental health problems were viewed as dangerous and incurable. The use of purgatives, emetics, beatings, and bloodletting was used to weaken the individual displaying unacceptable behaviors. With the advent of moral treatment in the beginning of the nineteenth century, those who displayed mental health problems were viewed as needing humane intervention instead of punishment. Asylums were established to provide treatment for those with mental health problems and the value of engaging in occupations was established. The treatment for those with mental health disorders often included engagement in various occupations such as gardening, sewing, or working in a shop.

The formal beginning of the profession of occupational therapy occurred in Clifton Springs, New York, USA, on March 17, 1917, with the foundation of the National Society for the Promotion of Occupational Therapy (NSPOT). This society was formed by individuals from varied professional backgrounds, reflecting the complexity of occupational therapy. A psychiatrist, William Dutton, considered the father of the profession, and Eleanor Clark Slagle, a welfare worker, considered the mother of the profession, along with architects, nurses, and teachers established the society. Adolf Meyer, a psychobiologist, was instrumental during the initial formation of the profession and published “The Philosophy of Occupational Therapy” in 1922, describing the need for occupational engagement with an understanding of the role of time and temporality as part of the profession. The use of habit training was a core element to the profession of occupational therapy and addressed the temporality described by Meyer. Another core element of the profession is energy conservation. One of the founding members of the NSPOT was an architect by the name of George Barton. He was a leading proponent of “efficiency techniques” and was instrumental in having the concepts of efficiency techniques included in the profession.

World War I (WWI) brought about an expansion in the profession of occupational therapy. Initially formed to provide intervention for those who had mental health problems, the needs of injured soldiers from WWI pressed the profession to develop quickly. The United States (US) military sought the assistance from NSPOT to recruit and train 1,200 “reconstruction aides” to meet the needs of wounded and injured soldiers including those suffering from “shell shock.” The efforts of NSPOT and the reconstruction aides were praised by the US Surgeon General’s office for their service to WWI veterans. In Canada, a similar growth in the need for occupational therapists was seen during WWI. They were identified as “ward aides” or “occupation aides” in Canada and served returning veterans.

Following WWI, the profession of occupational therapy established training standards and criteria for practitioners of occupational therapy in the USA. In 1921, the NSPOT changed its name to

the American Occupational Therapy Association (AOTA) and in 1931 created the first national registry of qualified occupational therapy practitioners. The profession continued to advance and refine its training but more slowly during the Great Depression. With the beginning of World War II (WWII), the profession of occupational therapy saw a dramatic increased demand for trained professions and the number of educational programs expanded. From 1941 to 1946, the number of trained occupational therapy professionals in the USA doubled to meet the needs of WWII veterans. Canada experienced similar increased demand, and Canadian occupational therapists were recruited to serve in Britain to meet the needs of veterans. The educational training of occupational therapists expanded to include activities of daily living in addition to the use of therapeutic activities.

Global Occupational Therapy

The profession of occupational therapy continued to expand, and in 1952, the World Federation of Occupational Therapy (WFOT) was formed. Representatives from the USA, Canada, England, Scotland, South Africa, Sweden, New Zealand, Australia, Israel, India, and Denmark initiated the organization. WFOT was admitted into official relations with the World Health Organization (WHO) in 1959 and was recognized as a non-governmental organization (NGO) by the United Nations (UN) in 1963. The purpose of this organization is to act as the international organization for the promotion of the profession. Included in WFOT’s initial objectives was the education and training of occupational therapists with the advancement of standards of practice. There are currently 57 member organizations of WFOT.

Current Knowledge

Educational Requirements for Occupational Therapy Practitioners

The education of an occupational therapy practitioner includes coursework in the psychological, psychosocial, medical, biological, social behavioral, and occupational sciences. Within the USA, there are two levels of occupational therapy

practitioners, the occupational therapist and an occupational therapy assistant. The entry-level occupational therapist is educated at the minimum with a master's degree of 5–6 years of college with thorough preparation in occupational therapy theory and practice, research, internships, and successful passing of the national registry examination. The completion of the master's degree is not sufficient to practice occupational therapy. The occupational therapy postbaccalaureate graduate must also pass the rigorous national registry examination to be allowed to practice as an occupational therapist. Once a graduate has passed this exam, the designation of Occupational Therapist, Registered (OTR) can be used. Most states within the USA also require licensure, and the designation of OTR/L is frequently used to designate a practitioner who is registered and licensed to practice occupational therapy.

The training at the assistant level is typically completed within a 2-year period of college work in the areas of psychological, psychosocial, medical, biological, social behavioral, and occupational sciences. The student pursuing an occupational therapy assistant degree must also complete coursework in occupational therapy practice and internships prior to sitting for the national certification examination. A graduate from an occupational therapy assistant program who has successfully completed the national examination is eligible to use the designation of Certified Occupational Therapy Assistant (COTA).

There are several programs within the USA that offer entry-level occupational therapy degrees at the doctoral level using the designation of Occupational Therapy Doctorate (OTD). Student enrolled in OTD program must also complete a course of study that includes psychological, psychosocial, medical, biological, social behavioral, and occupational sciences. A thorough preparation in occupational therapy theory and practice, research, internships, and successful passing of the national registry examination is included in this course of study over 6–7 years of college. The OTD program often allows further study in a specific area of

occupational therapy or occupational science, but the graduate with an entry-level OTD degree is still required to complete internships and successfully pass the national registration examination before being able to practice as an occupational therapist.

Advanced degrees in occupational therapy are also offered at the OTD level where the practitioner has already completed the entry-level requirements and is seeking an advanced degree. The field of occupational science, the science involved with the study of occupations, includes PhD preparation to further the knowledge of occupations.

Occupational therapy practitioners may demonstrate a level of knowledge and competence beyond entry level through additional certifications and degrees. An example is seen with the AOTA specialty and board certification programs (AOTA 2011). The specialty certification program provides both levels of practitioners to demonstrate advanced skills in specific areas of practice. Areas included in the specialty certification are the following: Driving and Community Mobility, Environmental Modification, Feeding, Eating, and Swallowing, and Low Vision. Board certification identifies occupational therapists that have achieved advanced knowledge and competence in large domains of practice and includes Pediatrics, Physical Rehabilitation, Gerontology, and Mental Health.

Conclusion

The profession of occupational therapy continues to change to meet the occupational needs of clients worldwide. In the USA, the profession of occupational therapy will reach its 100-year mark in 2017 and the Centennial Vision of AOTA articulates this concept of meeting the occupational needs of all clients:

We envision that occupational therapy is a powerful, widely recognized, science-driven, and evidence-based profession with a globally connected and diverse workforce meeting society's occupational needs. (AOTA 2006).

References and Reading

- American Occupational Therapy Association. (2006). Centennial vision. Retrieved May 29, 2011, from <http://www.aota.org/News/Centennial.aspx>.
- American Occupational Therapy Association. (2008). Occupational therapy practice framework: Domain and process (2nd ed.). *American Journal of Occupational Therapy*, 62, 625–683.
- American Occupational Therapy Association. (2011). Board and specialty certification programs. Retrieved May 29, 2011, from <http://www.aota.org/Practitioners/ProfDev/Certification.aspx>.
- Crepeau, E. B., Schell, B. A. B., & Cohn, E. S. (2009). Contemporary occupational therapy practice in the United States. In E. B. Crepeau, E. S. Cohn, & B. A. B. Schell (Eds.), *Willard and Spackman's occupational therapy* (11th ed., pp. 216–221). Philadelphia: Lippincott, Williams & Wilkins.
- Dunn, W., McClain, L. H., Brown, C., & Youngstrom, M. J. (1998). The ecology of human performance. In M. E. Neistadt & E. B. Crepeau (Eds.), *Willard and Spackman's occupational therapy* (9th ed., pp. 525–535). Philadelphia: Lippincott Williams & Wilkins.
- Gordon, D. M. (2009). The history of occupational therapy. In E. B. Crepeau, E. S. Cohn, & B. A. B. Schell (Eds.), *Willard and Spackman's occupational therapy* (11th ed., pp. 202–215). Philadelphia: Lippincott, Williams & Wilkins.
- Kielhofner, G., & Burke, J. (1977). Occupational therapy after 60 years: An account of changing identity and knowledge. *American Journal of Occupational Therapy*, 31, 675–689.
- Meyer, A. (1922/1977). The philosophy of occupational therapy. *American Journal of Occupational Therapy*, 31, 639–642.
- Mosey, A. C. (1996). *Applied scientific inquiry in the health professions: An epistemological orientation* (2nd ed.). Bethesda: American Occupational Therapy Association.
- Schwartz, K. B. (2003). The history of occupational therapy. In E. B. Crepeau, E. S. Cohn, & B. A. B. Schell (Eds.), *Willard and Spackman's occupational therapy* (10th ed., pp. 5–13). Philadelphia: Lippincott, Williams & Wilkins.
- World Federation of Occupational Therapists. (2011). WFOT information. Retrieved May 29, 2011, from <http://www.wfot.org/information.asp>.

Oculoauriculovertebral (OAV) Spectrum or Dysplasia

- **Goldenhar Syndrome**

Office of Rehabilitation

- **Department of Vocational Rehabilitation**

Olanzapine

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Definition

Olanzapine is a second-generation antipsychotic (SGA) with a greater 5HT_{2A}/D₂ dopamine receptor antagonist ratio as compared to a first-generation antipsychotic (FGA). As such, SGAs are purported to have a more benign side-effect profile, with a lower risk for causing extrapyramidal side effects (EPS) and tardive dyskinesia associated with long-term exposure.

Historical Background

Olanzapine is an SGA originally introduced for use in adults with psychosis. It is structurally similar to clozapine but without the propensity toward agranulocytosis, has relatively lower rates of anticholinergic side effects, and has lower risk for drug interactions. When compared to FGAs, olanzapine offers a lower risk of EPS. Based on the promising clinical findings in adults, investigators became interested in olanzapine's effects in youth.

Studies have emerged evaluating the efficacy and tolerability of olanzapine in children and

adolescents. One study of adolescents with bipolar mania, randomized to either olanzapine or placebo, demonstrated significantly improved response and remission criteria in the treatment group (Tohen et al. 2007). A schizophrenia study of adolescents randomized to olanzapine demonstrated improvement in all primary outcome measures including the Clinical Global Impression Scale-Severity of Illness, Brief Psychiatric Rating Scale for Children, and Positive and Negative Syndrome Scale (PANSS) compared with placebo (Kryzhanovskaya et al. 2009). In the Treatment of Early Onset Schizophrenia Spectrum Disorders Study (TEOSS), patients were randomized to olanzapine, risperidone, molindone, or placebo with no significant differences in response rates between active treatment groups (Sikich et al. 2008). What was consistent among the three randomized studies was the olanzapine arms consistently demonstrated statistically significant weight gain and changes in metabolic indices. In TEOSS, olanzapine was discontinued when interim data showed a greater increase in weight than with either risperidone or molindone without greater efficacy. An open-label study of five hospitalized preadolescent children, administered with olanzapine for bipolar disorder, psychosis NOS, schizophrenia, and ADHD, demonstrated either adverse effects or lack of clinically significant therapeutic responses leading to its being discontinued in all patients (Krishnamoorthy and King 1998). It is due to these findings that olanzapine is largely administered as a second-line or third-line treatment for schizophrenia in pediatric patients.

There is a dearth of data on prescribing rates of olanzapine in children and adolescents, but Constantine and Tandon describe changing trends in antipsychotic use in Florida (Constantine and Tandon 2008). They refer to the conclusions of both the Consensus Statement of the American Diabetes and the American Psychiatric Associations in February of 2004, including olanzapine and clozapine being associated with the greatest risk of weight gain, diabetes, and dyslipidemia (American Diabetes Association et al. 2004). By December of 2005, olanzapine was the least prescribed SGA in pediatric patients in Florida's

Medicaid program. In another study of privately insured children, risperidone accounted for 74.3% of antipsychotic prescriptions, followed by aripiprazole (13.9%), and ziprasidone and paliperidone both prescribed in less than 1% of cases (Olfson et al. 2010).

While the Food and Drug Administration has approved risperidone and aripiprazole for the treatment of symptoms associated with autism, other medications are often used "off-label" to target problem behaviors. Haloperidol has been demonstrated in placebo-controlled studies to be effective in treating these behaviors in children and adolescents (Anderson et al. 1984; Campbell et al. 1978; Cohen et al. 1980; Naruse et al. 1982). Its use is now limited due to the incidence of side effects including sedation, EPS, and tardive dyskinesias (Campbell et al. 1978, 1997). Therefore, SGAs have largely replaced FGAs to improve disruptive behaviors associated with ASDs (Hollander et al. 2006; McCracken et al. 2002).

In 1997, the National Institute of Mental Health funded five university-affiliated medical centers with expertise in the treatment of autism to constitute the Research Units on Pediatric Psychopharmacology (RUPP) autism network (King and Bostic 2006; McDougale et al. 2008). Because of a number of case reports describing the potential benefit of risperidone in children with autism and other developmental disabilities, the RUPP network selected risperidone for a multicenter trial and found that it improved aggression, hyperactivity, and irritability over 8 weeks (King and Bostic 2006; McCracken et al. 2002). Since the initial RUPP risperidone trial, there have been a number of other randomized, double-blind, placebo-controlled trials evaluating risperidone, but much less research has focused on olanzapine in children with ASD (Nagaraj et al. 2006; Shea et al. 2004).

Current Knowledge

Several case reports have been published regarding olanzapine use in children with ASD. In one case, a 10-year-old boy with bipolar disorder, autism, and mental retardation was switched

from thioridazine and fenfluramine to olanzapine and lithium and demonstrated a decrease in both aggression and repetitive behaviors (Horrigan et al. 1997). An 8-year-old boy with autism and hyperactivity and disruptive behaviors who had failed multiple drug trials was given a trial of olanzapine which reportedly decreased his hyperactivity and resolved his aggression (Malek-Ahmadi and Simonds 1998). London reported a case of olanzapine-induced mania in a child with PDD (London 1998).

In an initial open-label pilot study examining safety and tolerability of olanzapine in children, adolescents, and adults with PDDs (Potenza et al. 1999), eight patients were given treatment over 12 weeks. The mean dose at the end of the study was 7.8 mg/day. Seven patients completed the study, and six were considered either “much improved” or “very much improved” on the Clinical Global Impression-Improvement Scale or CGI-I ($p < 0.001$). The Vineland Adaptive Behavior Scale and Maladaptive Behavior Subscales (VMBS) captured significant improvements from baseline in temper tantrums, impulsivity, anxiety, and social withdrawal ($p < 0.001$). Rocking, property destruction, and inappropriate sexual behavior were also significantly decreased after olanzapine ($p < 0.005$). Significant improvement in overall symptoms with olanzapine was also reflected by Ritvo-Freeman Real Life Rating Scale overall scores ($p < 0.001$). No significant difference was noted in repetitive behaviors over the duration of the study. Significant weight gain was seen in six of the eight patients during the 12-week trial ($p = 0.008$). There were no cases of EPS.

In an open-label 6-week study, 12 children (mean age 7.8 years) with ASD were randomized to either olanzapine or haloperidol (Malone et al. 2001). The mean doses for haloperidol were 1.4 mg/day and 7.9 mg/day of olanzapine. The primary outcome measure was the CGI scale, with a secondary outcome based on the Children’s Psychiatric Rating Scale (CPRS). Five of the six subjects in the olanzapine group were considered responders on the CGI, either “much improved” or “very much improved,” compared to three of the six in the haloperidol group. There was no

statistically significant difference between groups. The CPRS autism factor assesses for social withdrawal, rhythmic motion/stereotypy, abnormal object relations, underproductive speech, and unspontaneous relation to the examiner. For the CPRS autism factor, both groups demonstrated significance in improvement from baseline to endpoint ($p = 0.0008$). Only the olanzapine group demonstrated significance in decreasing the anger/uncooperativeness and hyperactivity CPRS factors. The most common side effects included time-limited sedation and weight gain. All but one child taking haloperidol gained weight, and the olanzapine group demonstrated significantly more weight gain than the haloperidol group ($p < 0.04$). All six olanzapine children gained over 5 lb., compared with two in the comparator group. There was no EPS in the olanzapine group. The authors concluded that olanzapine appeared to be a safe alternative to haloperidol for short-term treatment of behavior symptoms in PDD.

A 3-month open-label study was performed in order to assess the effect of olanzapine on communication in children with PDD (Kemner et al. 2002). Twenty-five children between the ages of 6–16 (mean = 11.2) years with either a diagnosis of autism or PDD NOS were followed and evaluated. The mean maximum dose at the end of the study was 10.7 mg/day. Psychometric measures included the CGI, Aberrant Behavior Checklist, and TARGET (a clinician-generated checklist consisting of five target symptoms chosen by parents). Communication skills were assessed in children with fluent speech ($n = 20$) via behavioral analyses of a playroom session. The results of this study showed significant improvements on TARGET and three subscales of the Aberrant Behavior Checklist for irritability, hyperactivity, and inappropriate speech. Based on CGI-I, only three children could be considered responders. Twelve children were considered significantly less ill after olanzapine based on the CGI-severity score. These results did not replicate previous studies demonstrating much or very much improvement on CGI-I after treatment with olanzapine where the majority of children showed minor improvement. Adverse events included

weight gain (average 4.7 kg), increased appetite, and decreased strength, and three children developed EPS that resolved after lowering the dose of olanzapine.

A case series looking at the long-term effects of olanzapine was performed by Stavrakaki, Antochi, and Emery (2004). Seven subjects (ages 8–52) with PDD were monitored over 17 months for improvements in the CGI scale and Global Assessment of Function (GAF). Five of the seven had diagnoses of autism and two with PDD NOS. The mean dose of olanzapine was 7.1 mg/day. In this series, only two of the cases were children. The two boys were also on additional medication: risperidone, methylphenidate, and clonidine in an 8-year-old and dextroamphetamine in an 11-year-old. There was a significant increase in GAF scores from 37.7 pretreatment to 70.7 at end of study ($p < 0.0001$). By week 52, one subject had “improved,” three subjects were “much improved,” and three other subjects were “very much improved,” making six responders. The most common side effect was sedation, and one patient had a seizure not considered attributable to olanzapine. Interestingly, there was no significant weight gain ($p = 0.97$), but the authors note that subjects were monitored closely and treated with dietary and behavioral interventions which may have accounted for this finding.

The first and only randomized, double-blind, placebo-controlled study of olanzapine in PDD was an 8-week study conducted by Hollander et al. (2006). Six of 11 children (ages 6–14 years) were diagnosed with autism. Dosages of olanzapine were based on weight and ranged from 7.5 to 12.5 mg/day. The primary outcome measure was the clinician-rated CGI-I score at 8 weeks, and secondary outcome measures included the compulsion subscale of the Children’s Yale-Brown Obsessive Compulsive Scale (CY-BOCS) and the Overt Aggression Scale-Modified (OAS-M) irritability and aggression subscales. There was a nonsignificant improvement in the primary outcome of the treatment group averaging 4.0–2.25 ($+/-1.26$) versus the control group 4.0–3.5 ($+/-1.0$). Three of the six in the treatment group were responders, and one out of five placebo subjects was a responder (50%

vs. 20%). There were no significant changes noted on other outcome measures for irritability or aggression. The subjects receiving olanzapine gained more weight than controls, averaging 7.5 lbs. versus 1.5 lbs. in the control group. Four of the six olanzapine-treated subjects (66.6%) had more than 7% weight gain compared to one of five control subjects (20%). Weight gain and sedation were the most common side effects in the treatment group. There were no reported extrapyramidal symptoms associated with olanzapine.

Children have been shown to be more susceptible than adults to the side effects of FGAs and SGAs (Sikich et al. 2004). In a randomized controlled trial of 40 youth with psychotic disorders, haloperidol, risperidone, and olanzapine were compared. EPS was found in 67% of the haloperidol arm versus 56% and 53% of the risperidone and olanzapine arms, respectively. Those on haloperidol reported more severe EPS. Despite the greater risk of side effects, antipsychotics are increasingly being used in children and adolescents (Olfson et al. 2006).

In regard to long-term side effects, a meta-analysis by Correll and Kane looked at the use of SGAs for various psychotic and nonpsychotic diagnoses, including ASD. Ten studies were evaluated to assess the 1-year risk of tardive dyskinesia (TD) in children under 18 years old. There were 783 youth who received risperidone ($n = 737$), quetiapine ($n = 27$), or olanzapine ($n = 19$). Of these, after 3 years of treatment, there were three new cases of TD diagnosed. One-year TD rates are relatively low (annualized rate 0.42%), but the data was limited for SGAs other than risperidone, and many children are treated with higher doses of SGAs for longer durations which may lead to a greater TD risk not demonstrated in this study (Correll and Kane 2007).

Future Directions

With only a limited amount of data available regarding the use of olanzapine in the treatment of ASD, its use remains primarily empirical. On the other hand, there is extensive data concerning children with other psychiatric disorders treated with olanzapine

that warrants concern about side effects, specifically metabolic effects and weight gain. In the Sikich study, subjects in all treatment arms gained significant weight above normal growth, but olanzapine was the highest, followed by risperidone, then haloperidol (Sikich et al. 2004).

It remains crucial to continue reviewing emerging data regarding SGAs and their associated risks, particularly weight gain. At this time, given the state of the evidence, alternative SGAs should be considered prior to olanzapine for children with autism spectrum disorders given the chronicity of the disease and prolonged need for antipsychotic medications.

See Also

- ▶ Aberrant Behavior Checklist
- ▶ Atypical Antipsychotics
- ▶ Ritvo-Freeman Real Life Rating Scale
- ▶ Vineland Adaptive Behavior Scales (VABS)
- ▶ Zyprexa

References and Reading

- American Diabetes Association, American Psychiatric Association, American Association of Clinical Endocrinologists, & North American Association for the Study of Obesity. (2004). Consensus development conference on antipsychotic drugs and obesity and diabetes. *Journal of Clinical Psychiatry*, 65(2), 267–272.
- Anderson, L. T., Campbell, M., Grega, D. M., Perry, R., Small, A. M., & Green, W. H. (1984). Haloperidol in the treatment of infantile autism: Effects on learning and behavioral symptoms. *The American Journal of Psychiatry*, 141(10), 1195–1202.
- Campbell, M., Anderson, L. T., Meier, M., Cohen, I. L., Small, A. M., Samit, C., et al. (1978). A comparison of haloperidol and behavior therapy and their interaction in autistic children. *Journal of the American Academy of Child Psychiatry*, 17(4), 640–655.
- Campbell, M., Armenteros, J. L., Malone, R. P., Adams, P. B., Eisenberg, Z. W., & Overall, J. E. (1997). Neuroleptic-related dyskinesias in autistic children: A prospective, longitudinal study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(6), 835–843. <https://doi.org/10.1097/00004583-199706000-00022>.
- Cohen, I. L., Campbell, M., & Posner, D. (1980). A study of haloperidol in young autistic children: A within-subjects design using objective rating scales. *Psychopharmacology Bulletin*, 16(3), 63–65.
- Constantine, R., & Tandon, R. (2008). Changing trends in pediatric antipsychotic use in Florida's Medicaid program. *Psychiatric Services*, 59(10), 1162–1168. <https://doi.org/10.1176/appi.ps.59.10.1162>.
- Correll, C. U., & Kane, J. M. (2007). One-year incidence rates of tardive dyskinesia in children and adolescents treated with second-generation antipsychotics: A systematic review. *Journal of Child and Adolescent Psychopharmacology*, 17(5), 647–656. <https://doi.org/10.1089/cap.2006.0117>.
- Hollander, E., Wasserman, S., Swanson, E. N., Chaplin, W., Schapiro, M. L., Zagursky, K., et al. (2006). A double-blind placebo-controlled pilot study of olanzapine in childhood/adolescent pervasive developmental disorder. *Journal of Child and Adolescent Psychopharmacology*, 16(5), 541–548. <https://doi.org/10.1089/cap.2006.16.541>.
- Horrigan, J. P., Barnhill, L. J., & Courvoisie, H. E. (1997). Olanzapine in PDD. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(9), 1166–1167. <https://doi.org/10.1097/00004583-199709000-00007>.
- Kemner, C., Willemsen-Swinkels, S. H., de Jonge, M., Tuynman-Qua, H., & van Engeland, H. (2002). Open-label study of olanzapine in children with pervasive developmental disorder. *Journal of Clinical Psychopharmacology*, 22(5), 455–460.
- King, B. H., & Bostic, J. Q. (2006). An update on pharmacologic treatments for autism spectrum disorders. *Child and Adolescent Psychiatric Clinics of North America*, 15(1), 161–175. <https://doi.org/10.1016/j.chc.2005.08.005>.
- Krishnamoorthy, J., & King, B. H. (1998). Open-label olanzapine treatment in five preadolescent children. *Journal of Child and Adolescent Psychopharmacology*, 8(2), 107–113.
- Kryzhanovskaya, L., Schulz, S. C., McDougale, C., Frazier, J., Dittmann, R., Robertson-Plouch, C., et al. (2009). Olanzapine versus placebo in adolescents with schizophrenia: A 6-week, randomized, double-blind, placebo-controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(1), 60–70. <https://doi.org/10.1097/CHI.0b013e3181900404>.
- London, J. A. (1998). Mania associated with olanzapine. *Journal of the American Academy of Child and Adolescent Psychiatry*, 37(2), 135–136. <https://doi.org/10.1097/00004583-199802000-00002>.
- Malek-Ahmadi, P., & Simonds, J. F. (1998). Olanzapine for autistic disorder with hyperactivity. *Journal of the American Academy of Child and Adolescent Psychiatry*, 37(9), 902. <https://doi.org/10.1097/00004583-199809000-00006>.
- Malone, R. P., Cater, J., Sheikh, R. M., Choudhury, M. S., & Delaney, M. A. (2001). Olanzapine versus haloperidol in children with autistic disorder: An open pilot study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40(8), 887–894. <https://doi.org/10.1097/00004583-200108000-00009>.
- McCracken, J. T., McGough, J., Shah, B., Cronin, P., Hong, D., Aman, M. G., et al. (2002). Risperidone in children with autism and serious behavioral problems.

- The New England Journal of Medicine*, 347(5), 314–321. <https://doi.org/10.1056/NEJMoa013171>.
- McDougle, C. J., Stigler, K. A., Erickson, C. A., & Posey, D. J. (2008). Atypical antipsychotics in children and adolescents with autistic and other pervasive developmental disorders. *The Journal of Clinical Psychiatry*, 69(Suppl. 4), 15–20.
- Nagaraj, R., Singhi, P., & Malhi, P. (2006). Risperidone in children with autism: Randomized, placebo-controlled, double-blind study. *Journal of Child Neurology*, 21(6), 450–455.
- Naruse, H., Nagahata, M., Nakane, Y., Shirahashi, K., Takesada, M., & Yamazaki, K. (1982). A multi-center double-blind trial of pimozide (Orap), haloperidol and placebo in children with behavioral disorders, using crossover design. *Acta Paedopsychiatrica*, 48(4), 173–184.
- Olson, M., Blanco, C., Liu, L., Moreno, C., & Laje, G. (2006). National trends in the outpatient treatment of children and adolescents with antipsychotic drugs. *Archives of General Psychiatry*, 63(6), 679–685. <https://doi.org/10.1001/archpsyc.63.6.679>.
- Olson, M., Crystal, S., Huang, C., & Gerhard, T. (2010). Trends in antipsychotic drug use by very young, privately insured children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(1), 13–23.
- Potenza, M. N., Holmes, J. P., Kanes, S. J., & McDougle, C. J. (1999). Olanzapine treatment of children, adolescents, and adults with pervasive developmental disorders: An open-label pilot study. *Journal of Clinical Psychopharmacology*, 19(1), 37–44.
- Shea, S., Turgay, A., Carroll, A., Schulz, M., Orlik, H., Smith, I., et al. (2004). Risperidone in the treatment of disruptive behavioral symptoms in children with autistic and other pervasive developmental disorders. *Pediatrics*, 114(5), e634–e641. <https://doi.org/10.1542/peds.2003-0264-F>.
- Sikich, L., Hamer, R. M., Bashford, R. A., Sheitman, B. B., & Lieberman, J. A. (2004). A pilot study of risperidone, olanzapine, and haloperidol in psychotic youth: A double-blind, randomized, 8-week trial. *Neuropsychopharmacology*, 29(1), 133–145. <https://doi.org/10.1038/sj.npp.1300327>.
- Sikich, L., Frazier, J. A., McClellan, J., Findling, R. L., Vitiello, B., Ritz, L., et al. (2008). Double-blind comparison of first- and second-generation antipsychotics in early-onset schizophrenia and schizo-affective disorder: Findings from the treatment of early-onset schizophrenia spectrum disorders (TEOSS) study. *The American Journal of Psychiatry*, 165(11), 1420–1431. <https://doi.org/10.1176/appi.ajp.2008.08050756>.
- Stavarakaki, C., Antochi, R., & Emery, P. C. (2004). Olanzapine in the treatment of pervasive developmental disorders: A case series analysis. *Journal of Psychiatry & Neuroscience*, 29(1), 57–60.
- Tohen, M., Kryzhanovskaya, L., Carlson, G., Delbello, M., Wozniak, J., Kowatch, R., et al. (2007). Olanzapine versus placebo in the treatment of adolescents with bipolar mania. *The American Journal of Psychiatry*, 164(10), 1547–1556. <https://doi.org/10.1176/appi.ajp.2007.06111932>.

Omission Training

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Synonyms

Negative punishment; Type II punishment

Definition

Omission training is a behavior-analytic term that refers to a form of punishment in which an event is withdrawn contingent on the occurrence of a target behavior (e.g., property destruction, aggression toward other people). In the behavior-analytic literature, omission training typically is considered to be negative or Type II punishment. Everyday examples of omission training include loss of privileges (e.g., having to leave the zoo early contingent on hitting your mom, loss of one's driver's license for getting too many speeding tickets). Time-out and response cost are the two most commonly used forms of omission training. Time-out is the removal of a positive event contingent on the occurrence of a challenging behavior. Response-cost is the removal of a specified amount of a positive reinforcer contingent on the occurrence of a challenging behavior (see ► [“Time-out”](#) and ► [“Response Cost”](#) entries for additional details).

See Also

- [Punishment](#)
- [Time-out](#)
- [Response Cost](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied Behavior Analysis* (2nd ed.). Upper Saddle River: Pearson Education.

- Foxx, R. M. (1982). *Decreasing Behaviors of Severely Retarded and Autistic Persons*. Champaign: Research Press.
- Kazdin, A. E. (2001). *Behavior Modification in Applied Settings* (6th ed.). Belmont: Wadsworth/Thomson Learning.

One-on-One

- [Paraprofessional](#)

One-to-One

- [Para-educator](#)

Onset

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Synonyms

[Age of onset](#); [Age of recognition](#)

Definition

In his original report of the autistic syndrome, Kanner speculated that the condition he described was congenital, i.e., that the children were born with it (Kanner 1943). Parental report of age of onset was one of the major significant variables noted to separate childhood autism from other forms of severe disturbance – notably childhood schizophrenia (Kolvin 1971; Rutter 1972). Early onset was also a feature included in some of the first more operationalized definitions of the disorder (e.g., American Psychiatric Association [APA] 1980; Rutter 1972).

Subsequent work has generally supported the notion that autism is an early-onset disorder present at birth or becoming apparent in the first year of life. Although the term onset is typically used, in fact recognition is a more appropriate term since various factors might complicate recognition of the disorder, e.g., parental sophistication, education, denial, and so forth (Chawarska et al. 2007; Volkmar et al. 1985). A small body of work has begun to focus on direction observation, e.g., of high-risk samples followed over time, home videotapes, and so forth, and these frequently suggest that subtler difficulties may precede the more obvious ones that can give rise to parental concern (Chawarska et al. 2007; Osterling and Dawson 1994; Werner and Dawson 2005; Werner et al. 2000).

Most studies reliant on parent report suggest that parents are typically worried in the first or second year of life about their child's development. For example, Short and Schopler (1988) examined reported age of onset in a large group of cases at Division TEACCH and noted that in 76% of cases, parents were worried by age 2. In the DSM-IV field trial for autism, about 90% of parents had experienced concern by age 2 years (Volkmar et al. 1994). Recognition can be influenced by several factors including parental sophistication (or denial), severity of associated developmental delay, and/or autistic symptoms (Rogers and DiLalla 1990; Short and Schopler 1988).

With respect to onset, it should also be noted that in about 20–25% of cases, parents report a pattern of regression as part of the onset of autism (Rogers and DiLalla 1990). This issue remains somewhat controversial and poorly understood since frequently studies have relied primarily on parental report, with little attention to aspects of either reliability or exploration of the nature of the regression reported.

Sometimes, the issue is more one of a failure of development to progress, and at other times parents note a regression but in the context of a pattern of ongoing delay. The most noteworthy pattern, i.e., of marked developmental regression in the face of clearly normal development previously, is the least common but also probably the most important for research (Siperstein and Volkmar 2004).

See Also

- [Autistic Disorder](#)
- [Childhood Disintegrative Disorder](#)
- [Regression](#)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Chawarska, K., Paul, R., Klin, A., Hannigen, S., Dichtel, L. E., & Volkmar, F. (2007). Parental recognition of developmental problems in toddlers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(1), 62–72.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kolvin, I. (1971). Studies in the childhood psychoses. I. Diagnostic criteria and classification. *British Journal of Psychiatry*, 118(545), 381–384.
- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders*, 24(3), 247–257.
- Rogers, S. J., & DiLalla, D. L. (1990). Age of symptom onset in young children with pervasive developmental disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 29, 863–872.
- Rutter, M. (1972). Childhood schizophrenia reconsidered. *Journal of Autism and Childhood Schizophrenia*, 2(4), 315–337.
- Short, A., & Schopler, E. (1988). Factors relating to age of onset in autism. *Journal of Autism and Developmental Disorders*, 18, 207–216.
- Siperstein, R., & Volkmar, F. (2004). Brief report: Parental reporting of regression in children with pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 34(6), 731–734.
- Volkmar, F. R., Stier, D. M., & Cohen, D. J. (1985). Age of recognition of pervasive developmental disorder. *American Journal of Psychiatry*, 142(12), 1450–1452.
- Volkmar, F. R., Klin, A., Siegel, B., Szatmari, P., Lord, C., Campbell, M., et al. (1994). Field trial for autistic disorder in DSM-IV. *The American Journal of Psychiatry*, 151(9), 1361–1367.
- Werner, E., & Dawson, G. (2005). Validation of the phenomenon of autistic regression using home videotapes. *Archives of General Psychiatry*, 62(8), 889–895.
- Werner, E., Dawson, G., Osterling, J., & Dinno, N. (2000). Brief report: Recognition of autism spectrum disorder before one year of age: A retrospective study based on home videotapes. *Journal of Autism and Developmental Disorders*, 30(2), 157–162.

Operant Behavior

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Definition

Operant behavior is defined as voluntary behavior which is influenced by the consequences that follow it (Cooper et al. 2019).

The term “operant” was coined by B.F. Skinner and used to reflect the idea that an organism “operates” on its environment when engaged in operant behavior. In his early works, Skinner studied the behavior of rats and pigeons under laboratory settings to gain a controlled understanding of the principles of operant behavior (Skinner 1938). Once he established a foundational understanding, he evaluated his findings further, and within human populations. Through this work, he identified operant behavior as well as the process of operant conditioning (Pear and Eldridge 1984).

Operant behavior is best understood in the context of what is called the three-term contingency or the antecedents, behavior, and consequences which comprise an instance of operant behavior. Antecedents refer to the stimuli which are present in the environment directly before an instance of behavior. Behavior is the phenomena of interest, or the response of an organism to said antecedents. Consequences refer to the stimulus change which occurs after behavior. Understanding consequences is critical to examining operant behavior. It is well understood that the consequences which follow a behavior make it more or less likely that the behavior will occur again in the future (Cooper et al. 2019). That is to say, a reinforcing consequence will increase the future frequency of an operant behavior, and a punishing consequence will decrease its future frequency (Cooper et al. 2019).

The decades of research and applied work on operant behavior and operant conditioning have

been critical in the development of evidenced-based interventions to support individuals with autism spectrum disorder (ASD; Wolf et al. 1963). As the science of applied behaviour analysis predominantly focuses on operant behaviors, interventions which have emerged from the field aim to understand the operant conditioning process and change consequences to alter behavior (Sturmey et al. 2020). Interventions which aim to aid in meaningful behavior change for individuals with ASD and related disorders, such as functional communication training, teaching self-help, social and leisure skills, and supports for maladaptive behavior, all focus specifically on operant behavior (Sturmey et al. 2020). Hence, operant behaviors are an essential area of study as they relate to ASD.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavior](#)
- [Operant Conditioning](#)
- [Punishment](#)
- [Reinforcement](#)
- [Respondent Behavior](#)
- [Skinner's Verbal Behavior](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2019). *Applied behavior analysis* (3rd ed.). Hoboken: Pearson Education.
- Pear, J. J., & Eldridge, G. D. (1984). The operant-respondent distinction: Future directions. *Journal of the Experimental Analysis of Behavior*, 42(3), 453–467.
- Skinner, B. F. (1938). *The behavior of organisms: An experimental analysis*. New York: Appleton-Century.
- Sturmey, P., Ward-Horner, J., & Doran, E. (2020). Respondent and operant behavior. In Sturmey, P. (Ed.), *Functional analysis in clinical treatment: Second edition* (pp. 25–56). London, U.K.: Academic Press. <https://doi.org/10.1016/B978-0-12-805469-7.00002-4>
- Wolf, M., Risley, T., & Mees, H. (1963). Application of operant conditioning procedures to the behavior problems of an autistic child. *Behaviour Research and Therapy*, 1(2–4), 305–312.

Operant Conditioning

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Synonyms

[Instrumental conditioning](#); [Instrumental learning](#)

Definition

A process of learning in which a behavior's consequence affects the future occurrence of that behavior. B. F. Skinner (1953) derived the principles of operant conditioning from Thorndike's "law of effect," which suggests that a behavior producing a favorable or *satisfying* outcome is more likely to reoccur, while a behavior producing an unfavorable or *discomforting* outcome is more likely to decrease in frequency (Thorndike 1911).

Skinner's experimental work focused on the effects of different schedules on the rates of operant responses made by rats and pigeons (Skinner 1953). His work revealed that the frequency of a behavior could be increased through reinforcement. Two types of reinforcement include positive reinforcement, the giving of a rewarding stimulus following a behavior, and negative reinforcement, the removal of an aversive stimulus following a behavior.

Similarly, the frequency of a behavior can be decreased through punishment. Positive punishment is the giving of an aversive stimulus following a behavior, and negative punishment is the removal of a rewarding stimulus following a behavior. The frequency of behavior can also be decreased through extinction, the discontinuation of the behavior's reinforcer (Skinner 1953).

Alternatively, a new behavior can be produced by means of shaping, the reinforcement of

increasingly accurate approximations at the desired behavior.

Operant conditioning has played an integral role in the development of effective interventions targeting the modification of behavior, specifically operant language training, in individuals with autism spectrum disorder (ASD) (Hewett 1965; Lovaas et al. 1966).

Additionally, applied behavior analysis (ABA) or the “Lovaas technique,” introduced by Ivar Lovaas, has validated the efficacy of operant conditioning principles in the treatment of individuals with ASD (Lovaas 1987).

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behaviorism](#)
- [Negative Reinforcement](#)
- [Positive Reinforcement](#)
- [Reinforcement](#)

References and Reading

- Hewett, F. M. (1965). Teaching speech to an autistic child through operant conditioning. *The American Journal of Orthopsychiatry*, 35(5), 927–936. <https://doi.org/10.1111/j.1939-0025.1965.tb00472.x>.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55(1), 3–9.
- Lovaas, O. I., Berberich, J. P., Perloff, B. F., & Schaeffer, B. (1966). Acquisition of imitative speech by schizophrenic children. *American Association for the Advancement of Science*, 151(3711), 705–707. <https://doi.org/10.1126/science.151.3711.705>.
- Skinner, B. F. (1953). *Science and human behavior*. New York: Macmillan.
- Thorndike, E. L. (1911). *Animal intelligence*. New York: Macmillan.

Operational Exhaustion

- [Posttraumatic Stress Disorder](#)

Operational Stress Injury

- [Posttraumatic Stress Disorder](#)

Optimal Arousal

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Synonyms

[Readiness to learn](#); [State of alertness](#)

Definition

The state of alertness and attention that best facilitates learning. For children with ASD, arousal is often disturbed and is especially labile. Maintaining optimal arousal, by providing the appropriate balance between stimulation and calming, is one of the challenges facing teachers working with this population.

References and Reading

- Joosten, A., Bundy, A., & Einfeld, S. (2012). Context influences the motivation for stereotypic and repetitive behaviour in children diagnosed with intellectual disability with and without autism. *Journal of Applied Research in Intellectual Disabilities*. <https://doi.org/10.1111/j.1468-3148.2011.00663.x>. Epub before print.
- Logan, K. R., Bakeman, R., & Keefe, E. B. (1997). Effects of instructional variables on engaged behavior of students with disabilities in general education classrooms. *Exceptional Children*, 63, 481–498.
- Van Rie, F., & Heflin, J. (2009). The effect of sensory activities on correct responding for children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 3, 783–796.
- Zentall, S. S., & Zentall, T. R. (1983). Optimal stimulation: A model of disordered activity and performance in normal and deviant children. *Psychological Bulletin*, 94, 446–471.

Optimal Outcome

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A small percentage of individuals with autism spectrum disorders (ASDs) may go on to lose core symptoms of the diagnosis and achieve “optimal outcomes.” Helt et al. (2008) defined an individual with an optimal outcome as having a history of an ASD diagnosis, demonstrating average or above average academic and adaptive functioning, receiving minimal special education supports specific to autism symptoms, and not meeting criteria for a diagnosis of ASD diagnosis as determined by administration of the Autism Diagnostic Observation Schedule (ADOS).

It should be noted that losing an ASD diagnosis is not the only good outcome that can be achieved. Finding strengths in individual children with ASD that give them satisfaction and can be nurtured, maximizing their ability to communicate and have relationships with others, and successfully treating comorbid sources of distress (e.g., anxiety, depression) are other criteria for good outcomes. In fact, comorbid conditions such as anxiety and depression can persist in individuals who lose the ASD diagnosis and can be more distressing than the ASD symptoms themselves.

Lovaas (1987) pioneered the study of “recovery” from ASD when he reported strong cognitive and academic outcomes in a small sample of individuals with high-functioning ASD following early, intensive behavioral intervention. Since Lovaas’ study, a number of researchers have carried out further studies of outcome from ASD, including losing the diagnosis, with stronger experimental designs and more comprehensive measures of outcome (e.g., Fein et al. 2013; Salows and Graupner 2005).

Helt et al. (2008) estimate that between 3% and 25% of children diagnosed with ASD lose the diagnosis and exhibit average cognitive, adaptive, and social abilities. Helt and colleagues argue that misdiagnosis of ASD does not explain this phenomenon. The authors suggest that this outcome from ASD could be limited to certain subsets of ASD and is likely strongly tied to early detection and treatment, characteristics of the child (e.g., cognitive abilities), and maturation.

Recent studies have examined specific characteristics of children and adolescents with optimal outcomes using a variety of measures of psychological and neuropsychological functioning, as well as brain imaging. A comprehensive study by Fein and colleagues (summarized in Fein et al. 2013) indicated that a group of children and teenagers with optimal outcomes had similar academic, executive functioning, language, and social skills in comparison to typically developing study participants. When compared to typically developing peers, the optimal outcome participants did display some subtle differences in their pragmatic language. They also presented with a higher incidence of psychiatric concerns than typically developing controls, particularly symptoms of attention-deficit/hyperactivity disorder and specific phobias (Orinstein et al. 2015).

An important longitudinal study by Anderson et al. (2014) found that about 10% of their sample had “very positive outcome,” with a definition similar to Fein et al.’s definition of “optimal outcome”; they found that remitting of repetitive behaviors between ages 2 and 3 with intervention was the strongest predictor of this outcome.

Eigsti et al. (2016) have also explored ways in which brain networks responsible for language may differ for individuals with optimal outcomes. Findings indicated largely similar patterns of activation for individuals with optimal outcomes compared to those with ASD across several brain regions. However, the optimal outcome group also demonstrated “compensatory” activation in numerous regions, differing from both a typically developing control group and the ASD group. Results suggest that early treatment and

learning experiences may result in normalized language performance for optimal outcome individuals but that differing brain activity may underlie these observable behavioral similarities in language processing.

Further research is needed to understand the nature of differences between individuals with “optimal outcomes” and those with other developmental trajectories. Research suggests that intensive early intervention, specifically applied behavior analysis approaches to therapy, may contribute to better outcomes in ASD (Fein et al. 2013; Orinstein et al. 2015).

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Residual Autism](#)

References and Reading

- Anderson, D. K., Liang, J. W., & Lord, C. (2014). Predicting young adult outcome among more and less cognitively able individuals with autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 55(5), 485–494. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5819743/>.
- Eigsti, I. M., Stevens, M. C., Schultz, R. T., Barton, M., Kelley, E., Naigles, L., et al. (2016). Language comprehension and brain function in individuals with an optimal outcome from autism. *NeuroImage: Clinical*, 10, 182–191.
- Fein, D., Barton, M., Eigsti, I., Kelley, E., Naigles, L., Schultz, R. T., et al. (2013). Optimal outcome in individuals with a history of autism. *Journal of Child Psychology and Psychiatry*, 54(2), 195–205.
- Helt, M., Kelley, E., Kinsbourne, M., Pandey, J., Boorstein, H., Herbert, M., & Fein, D. (2008). Can children with autism recover? If so, how? *Neuropsychology Review*, 18, 339–366.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- Orinstein, A. J., Helt, M., Troyb, E., Tyson, K. E., Barton, M. L., Eigsti, I. M., et al. (2014). Intervention for optimal outcome in children and adolescents with a history of autism. *Journal of Developmental & Behavioral Pediatrics*, 35, 247–256.
- Orinstein, A., Tyson, K. E., Suh, J., Troyb, E., Helt, M., Rosenthal, M., et al. (2015). Psychiatric symptoms in youth with a history of autism and optimal outcome. *Journal of Autism and Developmental Disorders*, 45, 3703–3714.
- Salloos, G. O., & Graupner, T. D. (2005). Intensive behavioral treatment for children with autism: Four-year outcome and predictors. *American Journal on Mental Retardation*, 110, 417–438.

Options Method

- [Son-Rise Program](#)

Oral Language

- [Expressive Language](#)

Oral Sensitivity

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Definition

Oral sensitivity is defined as an atypical response to oral stimulation. Atypical responses include hyporeactive responses, hyperreactive responses, or sensory defensiveness. Hyporeactive responses are characterized by a diminished response to sensory input. Individuals who are hyporeactive to stimulation in the oral and pharyngeal regions may have delayed triggering of the swallowing mechanism, putting them at risk for aspiration. Hyperreactive responses are characterized by an excessive reaction to sensory input (Arvedson and Brodsky 2002). Hyperreactive responses to oral stimulation may develop into a conditioned facial defensiveness. This is because individuals may come to associate discomfort with the feeding process and develop a conditioned avoidance of food and other oral and facial stimulation (Arvedson and Brodsky 2002; Dodrill et al. 2004). Oral sensory defensiveness is characterized by an emotional

response to sensory input. Individuals who are orally defensive may take only a limited variety of tastes and textures orally and frequently refuse food. Treatment of oral sensitivity problems may involve adaptation of the sensory environment, implementation of specific techniques, and/or modifications of sensory qualities of food or feeding utensils (Arvedson and Brodsky 2002).

See Also

► [Feeding Problems](#)

References and Reading

- Arvedson, J. C., & Brodsky, L. (2002). *Pediatric swallowing and feeding: Assessment and management* (2nd ed.). Albany: Thomson Delmar Learning.
- Dodrill, P., McMahon, S., Ward, E., Weir, K., Donovan, T., & Riddle, B. (2004). Long-term oral sensitivity and feeding skills of low-risk pre-term infants. *Early Human Development*, 76, 23–37.
- Morris, S. E., & Klein, M. D. (2000). *Pre-feeding skills: A comprehensive resource for feeding development* (2nd ed.). Tucson: Therapy Skill Builders.

Oral-Facial Imitation

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Synonyms

[Facial imitation](#); [Oral-motor imitation](#); [Orofacial imitation](#)

Definition

Oral-facial imitation refers to the imitation of others' actions that specifically involve the mouth or face. It has been extensively researched in infants, and is thought to be the earliest form of

imitation present in typical development. There is evidence that neonates possess the capacity to imitate some oral-facial movements. This has led to theories positing that this type of early imitation plays a central role in the development of interpersonal skills, possibly via the creation of shared representations of the self and others. Research also suggests a relationship between oral-facial imitation skills and speech development. In older individuals, the focus of oral-facial imitation research is often on the imitation of facial expressions, particularly those conveying emotion. Oral-facial imitation should be distinguished from oral-facial mimicry or contagion. Whereas mimicry and contagion are considered automatic matching responses to others, imitation is thought to be volitional. There is some debate over whether neonatal oral-facial responses are automatic or volitional, but it is clear that older children and adults display behaviors of both types. A number of studies have implicated oral-facial imitation as a specific area of impairment in autism spectrum disorder (ASD). Given that oral-facial imitation abilities may be important for language development and for social-emotional reciprocity and learning, difficulties within this area could partially underlie the social-communicative symptoms associated with ASD.

See Also

- [Apraxia](#)
► [Developmental Dyspraxia](#)
► [Dyspraxia](#)
► [Imitation](#)
► [Oral-Motor Apraxia](#)
► [Oral-Motor Skills](#)

References and Reading

- Anisfield, M. (1991). Neonatal imitation. *Developmental Review*, 11, 60–97.
- Meltzoff, A. N., & Decety, J. (2003). What imitation tells us about social cognition: A rapprochement between developmental psychology and cognitive neuroscience. *Philosophical Transactions of the Royal Society of London B Biological Sciences*, 358, 491–500.

- Rogers, S. J., Hepburn, S. L., Stackhouse, T., & Wehner, E. (2003). Imitation performance in toddlers with autism and those with other developmental disorders. *Journal of Child Psychology and Psychiatry*, 44(5), 763–781.
- Rogers, S. J., Cook, I., & Meryl, A. (2005). Imitation and play in autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 382–405). Hoboken: Wiley.

References and Reading

- Duffy, J. R. (1995). *Motor speech disorders: Substrates, differential diagnosis, and management*. St. Louis: Mosby.
- Freed, D. (2000). *Motor speech disorders: Diagnosis and treatment*. San Diego: Singular.
- Zemlin, W. R. (1998). *Speech and hearing science: Anatomy and physiology* (4th ed.). Boston: Allyn and Bacon.

Oral-Motor Apraxia

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Synonyms

[Nonverbal oral apraxia](#)

Definition

Oral-motor apraxia is the inability to volitionally sequence oral movements of the speech structure for nonspeech tasks in the absence of neuromuscular deficits such as paralysis or muscle weakness (Zemlin 1998). Patients with oral-motor apraxia demonstrate off-target effortful groping for correct movements involving the speech structure (e.g., clicking the tongue, licking the lips, whistling), or inconsistent trial-and-error attempts. Spontaneous and reflexive movements, such as swallowing while eating and smiling at a joke, are not affected (Freed 2000). Oral-motor apraxia often occurs in conjunction with apraxia of speech, although there is not a one-to-one correspondence. Either apraxia may exist in the absence of the other (Duffy 1995).

See Also

- [Apraxia](#)
- [Developmental Apraxia](#)
- [Dyspraxia](#)

Oral-Motor Imitation

- [Oral-Facial Imitation](#)

Oral-Motor Skills

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Definition

Oral-motor skills refer to the movement of the muscles of the face and oral area (e.g., mouth, jaw, lips, tongue, soft palate). These skills are influenced by muscle tone, muscle strength, range of motion, speed, coordination, and dissociation (the ability to move oral structures, such as the tongue and lip, independently of each other) (Kumin n.d.). Clinical experience suggests that the acquisition and maturation of oral-motor movements underlie sound production and feeding skills (e.g., sucking, biting, and chewing) (Arvedson and Brodsky 2002). Children with developmental disabilities may demonstrate oral-motor patterns that are not observed in typical development. Atypical oral-motor patterns include jaw thrusting, tongue thrust, tonic bite reflex, lip retraction, tongue retraction, and nasal regurgitation (Morris 1978). A child's oral-motor skills may be assessed by a speech-language pathologist.

See Also

- Apraxia
- Dysarthria
- Feeding Problems
- Speech

References and Reading

- Arvedson, J. C., & Brodsky, L. (2002). *Pediatric swallowing and feeding: Assessment and management* (2nd ed.). Clifton Park: Thomson Delmar Learning.
- Kumin, L. (n.d.) *Resource guide to oral motor skill difficulties in children with down syndrome*. Retrieved 21 Jan 2011 from <http://www.ndscenter.org/resources/documents/speech/OralMotor.pdf>.
- Morris, S. (1978). Oral motor development: Normal and abnormal. In J. M. Wilson (Ed.), *Oral-motor function and dysfunction in children*. Chapel Hill: Division of Physical Therapy, UNC.
- Morris, S. E., & Klein, M. D. (2000). *Pre-feeding skills: A comprehensive resource for feeding development* (2nd ed.). Tucson: Therapy Skill Builders.

Orap

- Pimozide

Organelle

- Mitochondrial Deficits/Disorders

Organization of the Physical Learning Environment

- Classroom Structure

Organizational Prevention

- Systems Intervention

Organizational Skills in Individuals with Autism Spectrum Disorders

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Definition

Organizational skills are a set of techniques used by an individual to facilitate the efficiency of future-oriented learning, problem-solving, and task completion. Organization requires the integration of several elements to reach a planned goal. Dawson and Guare (2010) define organization as “the ability to design and maintain systems for keeping track of information or materials” (p. 1). The broader domain of executive functioning plays a large role in the development and execution of organizational skills. Executive functioning is the multifaceted construct used to describe higher order, goal-directed thinking involving planning, inhibition, flexibility, organized search, and working memory and is thought to be impaired in individuals with ASD (Gyori 2006). Organization can be considered a sub-component of the larger executive function construct.

Evidence suggests that individuals with autism spectrum disorders (ASD) have deficits in executive functioning abilities. For instance, impairments in flexibility, attention, and planning have been documented (O’Hearn et al. 2008). Those with ASD also have been noted to evidence dysfunction in their organizational skills (Kenworthy et al. 2005). This disorganization may be due to the presence of the aforementioned executive

functioning deficits and related regulatory sequelae, such as the fact that individuals with ASD have a greater tendency to overfocus on irrelevant details to the exclusion of conceptualizing the “big picture” (Kuschner et al. 2009). In contrast, people with ASD have been anecdotally noted to be highly organized regarding their special interests, such that they know every single detail about a topic and have organized the information quite well (Baron-Cohen 2009). To date, however, there is a relative dearth of research specifically focusing on the subcomponent of organization in individuals with ASD, their relationship to ASD symptoms and characteristics, their developmental unfolding, and their importance to developmental functions and outcomes. What literature is available has tended to address higher functioning individuals, with a focus on broader executive functions (e.g., Hill 2004). In this chapter, we provide an overview of the history of research on organizational skills within the context of executive functions in individuals with ASD; compare and contrast the development of organizational skills in typically developing children and children with ASD, along with potential etiological agents; and present emergent interventions that aim to improve organizational skills.

Historical Background

Organizational skills lie within the broader cognitive domain of executive functions. The empirical investigation of executive functions began in the 1980s, and research using samples of individuals with ASD soon followed, suggesting the presence of an array of difficulties as compared to typically developing individuals. In one of the first studies examining executive functioning in individuals with ASD, Rumsey (1985) used the Wisconsin Card Sorting Test (WCST) to compare the cognitive flexibility of adult men with high-functioning autism to the flexibility of a sample of age-matched typical adults. Rumsey found that individuals with autism demonstrated significant perseveration when compared to the control group, a finding that has since been replicated with

children and adolescents (Ozonoff et al. 2005; Prior and Hoffman 1990). Additional studies in the early 1990s revealed that individuals with high-functioning ASD had executive functioning deficits relative to other disorders, such as attention-deficit/hyperactivity disorder and learning difficulties, and across different cultures, suggesting that selected executive dysfunctions likely were core deficits in individuals with ASD (Ozonoff et al. 2005).

The study of executive functioning has evolved to better address the multidimensional nature of the construct, using a wider variety of outcome measures to consider other abilities such as categorization, working memory, inhibition, selective attention, and managing verbal feedback (Hughes 2001; Ozonoff et al. 2005). Indeed, a variety of models of executive function have now evolved (e.g., Stuss 2007; Friedman and Miyake 2017), and these are ripe for continued examination of their relevance for this population. Despite these advances, there remains a paucity of research specifically examining organizational skills in individuals with ASD.

The scientific examination of organization skills also is complicated by the tendency for individuals with ASD to be overly focused on details rather than conceptualizing the broad whole. Does this represent intact organization or, rather, disorganization due to the lack of attention to peripheral but important environmental details? One result of this tendency to overfocus on selected tasks is the difficulty with the generalization or the appreciation that skills learned in one context may be applied to other contexts (Volkmar and Wiesner 2009). Further, this approach to tasks may indirectly contribute to social skills difficulties, as to extract social information from various contexts requires both flexibility and the ability to organize this information in a meaningful way (Hughes 2001). Organizational skills also directly relate to an individual's success with academic and vocational demands, as well as activities of daily living, such as paying bills, grocery shopping, and keeping up with doctor's appointments. As such, the area of organizational skills is a critical one for clinical investigation and day-to-day functioning. For individuals with ASD,

organization and related executive functions have been associated with adaptive behavior (Pugliese et al. 2016), social functioning (Freeman et al. 2017), driving (Cox et al. 2016), and affective regulation (Wallace et al. 2016).

Current Knowledge

The Development of Executive Functions and Organization in Typically Developing Children

In a typically developing child, the development and application of organizational strategies begins in infancy. As children remember information, they store it in an orderly manner through the use of categorization (Berk 2002). Three- to five-month-olds have been found to categorize stimuli by shape, size, number, and other physical characteristics. Berk (2002) cited a number of studies providing evidence that infants not only organize their physical world through the use of categorization but their emotional and social worlds as well. This includes infants sorting people and voices by gender and age and the emerging ability to discriminate between emotional expressions. During early childhood (ages 2 through 6 years), children begin to use planning and sequencing in order to reach a goal with familiar or simple tasks. Children also begin to use strategies in problem-solving and move from less to more efficient strategies through practice, reasoning, and adult assistance, among other factors (Berk 2002). Generally, during these years, children will engage in a variety of executive functions, but they tend to be task or situation specific, with generalization of these skills being limited. By middle childhood (ages 6 through 11 years), children make more use of planning skills and begin to engage in generalizing these skills to other tasks and situations. For instance, they will decide how to order steps so as to accomplish a complex task and then utilize those strategies for another task or situation. In addition, children are able to utilize multiple strategies concurrently, and their long-term knowledge base becomes organized into more sophisticated, hierarchically structured networks (Berk 2002). Finally, adolescence to adulthood marks the development of more focused and

regulated attention, more effective and efficient strategy development and use, and an expansion of metacognition (Berk 2002). Taken together, these lead to more highly developed organizational capabilities and skills.

The Development of Executive Functions and Organization in Individuals with ASD

Although executive functions have been shown to be pervasively affected in many neurodevelopmental disorders across a range of ages and functional levels, less is known about the development of organizational skills in children with ASD or, more broadly, the developmental course of executive functions (O'Hearn et al. 2008). It has been theorized that although selected executive function deficits (i.e., set shifting and flexibility) have been demonstrated in young children with ASD (mean age = 5 years) relative to a control group of typically developing peers, these deficits are not autism-specific; but, instead, they are related to developmental delays. Another perspective with some empirical support is that executive functioning in preschool ASD is similar to that of young children with developmental delays matched on chronological and mental age, and suggests that executive dysfunction may emerge over the course of development and is not yet present (or observable) in early childhood. In that regard, these deficits are secondary in nature (Ozonoff et al. 2005). There is evidence that executive dysfunction and impairments may be most prominent during adolescence (Tsatsanis 2005; Rosenthal et al. 2013), particularly the application of planning and the goal-directed organization of information (Ozonoff et al. 2004). Kushner et al. (2009) discovered that while typically developing individuals demonstrated improvements in organizational and planning skills with age when reproducing the Rey-Osterrieth Complex Figure (ROCF), no similar qualitative differences in performance were displayed between children and adolescents/adults with HFA. In contrast, other findings have demonstrated improvements in executive function from childhood to adolescence in individuals with ASD, perhaps secondary to neuropsychological maturation; nevertheless, executive functioning

in adulthood was found to remain relatively impaired despite these developmental gains (Brady et al. 2017; O'Hearn et al. 2008). Longitudinal research is needed to better illuminate the developmental trajectory of the various dimensions of executive functions in children with ASD, with a specific need to include carefully considered covariates (e.g., nonverbal learning difficulties) (Hagberg et al. 2015).

Although much is to be learned regarding the development of executive functions, evidence related to planning and organization deficits has begun to surface over the past 10–15 years. Geurts et al. (2004) compared children with high-functioning ASD and attention-deficit/hyperactivity disorder (ADHD) on measures of executive functioning. These investigators reported that the errors made by the ASD group were related to difficulties with planning and cognitive flexibility, whereas errors by the ADHD group were better attributed to deficits in inhibition and fluency. Problems with planning efficiency and self-monitoring, which are closely related to organizational skills, have been documented in a number of studies (Tsatsanis 2005). Impaired performance on the Tower of London task was reported for children with ASD relative to typically developing controls, and was attributed to impaired planning skills and reduced strategy formation (Robinson et al. 2009).

Examining a sample of individuals with subthreshold autism traits, Christ et al. (2010) found a pattern of deficits in a number of executive functioning domains including planning, organizing, and setting goals as measured by the Behavior Rating Index of Executive Function (BRIEF). In contrast, Christ et al. (2010) did not find a relationship between autism symptomatology and the tendency to keep one's work space and materials orderly, as measured by the Organization of Materials Scale of the BRIEF. Overall, their work suggested that even individuals with subthreshold autism traits exhibit profiles of executive dysfunction, including deficits in organization/planning, similar to those of individuals with diagnoses of ASD. These findings suggest that executive dysfunction may appear on a continuum of

impairment, although it appears that problems with organizational skills of children with ASD may not be clearly dissociable from other childhood conditions (e.g., Tourette Syndrome, ADHD) (Hovik et al. 2017) – or even different subgroups of ASD (Blijd-Hoogewys et al. 2014). Adults with ASD who completed self-ratings of their broader executive functions did report their planning and organization skills to be mildly impaired, and significantly lower than typical controls (Davids et al. 2016).

Additionally, individuals with ASD have been noted to have difficulty copying and remembering the Rey-Osterrieth Complex Figure (ROCF), a task which requires visuospatial abilities, planning, and organization. Kenworthy et al. (2005) considered difficulty reproducing the ROCF, along with verbal organization deficits, to suggest impairments in organization. Kenworthy et al. (2005) expanded upon this suspicion to document deficits in the organization skills of individuals with high-functioning autism (HFA) and Asperger's disorder (AD) across several verbal and visual tasks. For example, although both high-functioning ASD groups were found to have intact ability to interpret discrete data, relative impairments were noted in their ability to integrate and organize complex information. Another discrepancy that the authors attributed to a deficit in organizational skills was the participants' ability to copy abstract visual information from a model but comparative difficulty with perceiving a visual gestalt when organizing puzzle pieces. Despite mean scores within the average range on several general cognitive measures, these investigators found relative impairments in a number of domains of executive functioning in the high-functioning ASD groups, including tasks specifically measuring organization [e.g., the Planning/Organization Scale of the BRIEF, the Story Memory subtest from the Wide Range Assessment of Memory and Learning (WRAML), and the Object Assembly subtest from the Wechsler Intelligence Scale for Children-III (WISC-III)]. In sum, while many previous studies have emphasized deficits in planning, a construct closely linked with organization, this study provided more direct evidence that

individuals with ASD have executive deficits in flexibility and organizational skills.

Despite these findings, however, the deficits do not appear to be directly linked to the core symptoms present in ASD, although recent theories have made an effort to assert this linkage (Baron-Cohen 2009). Further, from a clinical perspective, many clinicians will agree that their clients with ASD can be highly organized with respect to their restricted interests, perhaps being overorganized with respect to the intensity of their focus on these interests and their various components. For example, clinicians and researchers working with this population no doubt have been favorably impressed with the amount of focus, logic, and organization of information pertaining to a restricted interest. In many instances, these individuals actually have a knowledge base that would rival many experts in the targeted area, and their attention to the details pertaining to this information is exquisite. Anecdotally, this indicates that individuals with ASD are capable of goal-directed organization, although this skill is not always generalized and applied outside of their area of restricted interest and, for many individuals with ASD, herein lies the challenge.

Explaining Organizational Dysfunction

There are a number of possible underlying causes of, or contributors to, the impairments noted in organizational skills and other related aspects of executive functioning. Frith's theory of weak central coherence (Frith 1989) posits that individuals with ASD tend to process information in fragments rather than in terms of the coherent whole. Consequently, individuals with ASD may focus on minor, irrelevant details and fail to process the underlying meaning of the cohesive information (Hughes 2001). Without an understanding of the broader concept, it is challenging to effectively organize and process presented information. Mesibov et al. (2004) proposed that in addition to focusing on minute details, individuals with ASD are less capable of assessing and prioritizing their relative importance. For example, Mesibov et al. (2004) described a young man with ASD

who recalled having difficulty following a conversation that went from red balloons to birthday parties to the children enjoying themselves, because he became focused on "red balloons" and was unable to organize a schema for a birthday party. Although not stated directly, it is likely that this individual, or others like him, could tell you an enormous amount of information about (red) balloons in a detailed and organized fashion.

In addition, differences in executive functioning in individuals with ASD have been attributed to abnormalities in brain maturation (e.g., decreased synaptic pruning early in development, structural differences in regions of the brain such as the cerebellum, and decreased connectivity within the brain due to volumetric differences of white matter in the corpus callosum; Han and Chan 2017; Haznedar 2011; O'Hearn et al. 2008). For example, Just et al. (2007) found evidence of reduced intracortical connectivity resulting in a lower degree of integration of information across certain cortical areas. The investigators concluded the presence of deficits in the integration of information at the neural level which, in turn, contributed to cognitive impairments. While a fuller discussion of these neurobiological features of ASD is beyond the scope of this chapter, it is important to note that the neurological bases of executive dysfunctions in autism have yet to be fully explained (Han and Chan 2017; O'Hearn et al. 2008).

Interventions

Impairments in executive function skills, such as planning, prioritizing, and organizing, may become more apparent when children begin school. In this situation, they are required to keep track of their personal items, follow multi-step directions, engage in multistep assignments, and complete tasks in a set amount of time. Despite a need for structure and order, children with ASD may have messy desks and turn-in incomplete assignments (Moore 2002). In this regard, there does appear to be a significant need for specific interventions to improve selected types of organization skills. There is some notion

that various executive functions can be improved through intervention; however, there is a lack of strong empirical research looking at remediating executive functioning in children with ASD (Ozonoff et al. 2005).

One randomized controlled trial has emerged examining the utility of the Unstuck and On Target intervention versus social skills training to address executive dysfunction in children with high functioning ASD (Kenworthy et al. 2014). Forty-seven third to fifth grade students were assigned to the Unstuck and On Target intervention, and they were matched by age, gender, and race, IQ, ASD symptomatology, medication status, and parents' education to 20 students who were randomized to the social skills training. The interventions were implemented in 28 small group sessions (30–40 min in length) and were conducted in the school setting. Findings revealed significant improvements in organization and planning for the Unstuck and On Target group, with effect sizes being in the small to medium range. Improvements also were noted in problem solving and behavioral flexibility, with these findings translating into improved rule-following behaviors and significantly better capabilities to make transitions. Given the potentially pervasive nature of these problems, interventions may require collaboration between schools and families, organizational aids, and significant day-to-day structure (Volkmar and Wiesner 2009). Although there are some medical treatments that have shown promising early results (e.g., repetitive transcranial magnetic stimulation) (Ameis et al. 2017), educational and behavioral strategies currently remain the best options for improving organizational skills.

To date, from a best practice perspective, a number of strategies have been offered to address organizational problems. Volkmar and Wiesner (2009) recommended a stepwise approach, consistency and predictability, the use of functional routines, providing time to process/prepare, and the use of aids such as visuals, organizers, and computers. Moore (2002) suggested physically organizing the classroom and using color coding, visuals, daily schedules, checklists, teaching children to find the main idea rather than focusing on

irrelevant details, and using an assignment notebook, among other aids. Dawson and Guare (2010) proposed that children lacking intact organizational capacity should be provided with organizational schemes. The children should be cued to use the organizational scheme and receive reinforcement for using it. An example for organizing a backpack provided by Dawson and Guare (2010) is to use specific pockets and compartments, which can be labeled or color coded for lunch, permission slips, money, and homework. This latter example also may capitalize on the cognitive rigidity, or need for "sameness," typically evidenced by many individuals with ASD. More generally, this approach also suggests the need for a thorough evaluation of various executive functions, including organizational capabilities.

Many of the aforementioned suggestions to remedy the difficulties associated with executive dysfunction are related to concepts from structured teaching. Structured teaching, a cornerstone of the TEACCH (Treatment and Education of Autistic and Communication handicapped CHildren) program, is based on the premise that clear structure and organization make the environment more navigable for individuals with ASD by clarifying expectations and enabling communication. This is achieved through the use of physical organization, visual support, and schedules (Hartford and Marcus 2011). Goals of physical organization (e.g., dividers, clearly delineated areas for specific activities) include the addition of contextual clues, clarification of boundaries, and the minimization of distractions and stimulation. Visual supports are utilized to capitalize on the tendency for individuals with ASD to have relatively strong visual learning abilities and include picture prompts, video modeling, ordered pictures, or step-by-step written directions, which can help in attention and sequencing. Schedules can be used to establish functional routines and add predictability to the day, and they can indicate a sequence through words, pictures, or objects to facilitate smooth transitions and temporal understanding. In general, built-in organization through structured teaching methods can help individuals with autism effectively understand and participate in the world around them (Hartford and Marcus 2011).

Direct instruction interventions with empirical support, such as providing organizational skills training, can also be used to improve the skills of children with ASD in the classroom. For example, in one of the few evidence-based studies related to organization training in ASD, elementary-age children with high-functioning autism and Asperger's syndrome were found to increase the percentage of items correctly filed and decrease the number of seconds needed to retrieve a requested item following an intervention teaching the children self-monitoring skills and the use of a file box system to enhance organization (Dorminy et al. 2009).

Future Directions

This encyclopedia entry has addressed an important but targeted area of executive functioning in the ASD population; namely, organizational skills. Despite the range of studies that have been focused on executive functions in this population, few empirical studies have specifically examined the subcomponent of organization. The empirical literature that does exist in the domain of organization, and even executive functions more broadly, typically has studied individuals with high-functioning ASD, although it is noteworthy that more recent studies have begun to include individuals with ASD who have a broader range of overall functioning. It would be worthwhile for future research to not only examine profiles of executive functions that would include organization, but also to examine these profiles in lower functioning populations. While some might argue that these problems may not be high on the priority list of needs for many of these individuals, such research could shed light on how organizational deficits contribute to the presence of aggressive, self-injurious, and repetitive behaviors as well as to the overall adaptation of individuals with ASD who are higher functioning.

For instance, it has been theorized that individuals engage in repetitive behaviors when interpreting social situations as unpredictable as a means to impose order (Hughes 2001); thus, an

understanding of how individuals with ASD differentially order and organize both social and nonsocial information might provide additional strategies for implementing successful interventions. In addition, although there is a breadth of research on neurodevelopmental constructs such as attention, memory, language, and temporal-sequential ordering, there is limited research specifically investigating organization in individuals with ASD, the developmental course of organizational skills throughout the life span in this population, or how this cognitive function contributes to ASD symptom manifestation.

Finally, there remain a myriad of additional questions related to the organizational skills of individuals with ASD. In addition to examining the multidimensionality of these skills across all ages and levels of functioning, questions pertaining to their generalized versus specific nature, associated measurement strategies, their relationships to core and associated symptoms in ASD, and their linkages to evidence-based practices all remain unanswered at present. Specific studies addressing the organizational skills of individuals with ASD will continue to advance our knowledge of how these skills facilitate our overall understanding of ASD.

See Also

- ▶ [Attention](#)
- ▶ [Classroom Structure](#)
- ▶ [Cognitive Flexibility](#)
- ▶ [Executive Function \(EF\)](#)
- ▶ [Frontal Lobe Findings in Autism](#)
- ▶ [Memory](#)
- ▶ [Neuropathology](#)
- ▶ [Neurophysiology](#)
- ▶ [Perseveration](#)
- ▶ [Retrieval of Information](#)
- ▶ [Structured Classrooms](#)
- ▶ [Structured Teaching](#)
- ▶ [Synaptic Pruning](#)
- ▶ [Theory of Mind](#)
- ▶ [Time Management](#)
- ▶ [Visual Schedule](#)
- ▶ [Visual Supports](#)

References and Reading

- Ameis, S. H., Daskalakis, Z. J., Blumberger, D. M., Desarkar, P., Drmic, I., Mabbott, D. J., Lai, M.-C., Croarkin, P. E., & Szatmari, P. (2017). Repetitive transcranial magnetic stimulation for the treatment of executive function deficits in autism spectrum disorder: Clinical trial approach. *Journal of Child and Adolescent Psychopharmacology*, 27, 413–421.
- Baron-Cohen, S. (2009). Autism: the empathizing-systemizing (E-S) theory. *Annals of the New York Academy of Science*, 1156, 68–80.
- Berk, L. E. (2002). *Infants, children, and adolescents* (4th ed.). Boston: Allyn and Bacon.
- Blijd-Hoogewys, E. M. A., Bezemer, M. L., & van Geert, P. L. C. (2014). Executive functioning in children with ASD: An analysis of the BRIEF. *Journal of Autism and Developmental Disorders*, 44, 3089–3100.
- Brady, D. I., Saklofske, D. H., Schwean, V. L., Montgomery, J. M., Thorne, K. J., & McCrimmon, A. W. (2017). Executive functions in young adults with Autism Spectrum Disorder. *Focus on Autism and Other Developmental Disabilities*, 32, 31–43.
- Christ, S. E., Kanne, S. M., & Reiersen, A. M. (2010). Executive function in individuals with subthreshold autism traits. *Neuropsychology*, 24, 590–598.
- Cox, S. M., Cox, D. J., Kofler, M. J., Moncrief, M. A., Johnson, R. J., Lambert, A. E., Cain, S. A., & Reeve, R. E. (2016). Driving simulator performance in novice drivers with autism spectrum disorder: The role of executive functions and basic motor skills. *Journal of Autism and Developmental Disorders*, 46, 1379–1391.
- Davids, R. D., Groen, Y., Berg, I. J., Tucha, O. M., & van Balkom, I. D. C. (2016). Executive Functions in Older Adults With Autism Spectrum Disorder: Objective Performance and Subjective Complaints. *Journal of Autism and Developmental Disorders*, 46, 2859–2873.
- Dawson, P., & Guare, R. (2010). *Executive skills in children and adolescents: A practical guide to assessment and intervention* (2nd ed.). New York: The Guilford Press.
- Dorminy, K. P., Luscre, D., & Gast, D. L. (2009). Teaching organizational skills to children with high functioning autism and Asperger's syndrome. *Education and Training in Developmental Disabilities*, 44(4), 538–550.
- Freeman, L. M., Locke, J., Rotheram-Fuller, E., & Mandell, D. (2017). Brief report: Examining executive and social functioning in elementary-aged children with autism. *Journal of Autism and Developmental Disorders*, 47, 1890–1895.
- Friedman, N. P., & Miyake, A. (2017). Unity and diversity of executive functions: Individual differences as a window on cognitive structure. *Cortex*, 86, 186–204.
- Frith, U. (1989). *Autism: Explaining the enigma*. Oxford: Blackwell Scientific.
- Geurts, H. M., Verté, S., Oosterlaan, J., Roeyers, H., & Sergeant, J. A. (2004). How specific are executive functioning deficits in attention deficit hyperactivity disorder and Autism? *Journal of Child Psychology and Psychiatry*, 45, 836–854.
- Gyori, M. (2006). *Autism and cognitive architecture: Domain specificity and psychological theorising on autism (neurocognitive development and impairments)*. Budapest: Akademiai Kiado.
- Hagberg, B., Billstedt, E., Nydén, A., & Gillberg, C. (2015). Asperger syndrome and nonverbal learning difficulties in adult males: self- and parent-reported autism, attention and executive problems. *European Journal of Child and Adolescent Psychiatry*, 24, 969–977.
- Han, Y. M. Y., & Chan, A. S. (2017). Disordered cortical connectivity underlies the executive function deficits in children with autism spectrum disorders. *Research in Developmental Disabilities*, 61, 19–31.
- Hartford, D., & Marcus, M. (2011). Educational approaches. In E. Hollander, A. Kolevzon, & J. Coyle (Eds.), *Textbook of autism spectrum disorders* (pp. 537–553). Washington, DC: American Psychiatric Publishing.
- Haznedar, M. M. (2011). Structural neuroimaging. In E. Hollander, A. Kolevzon, & J. Coyle (Eds.), *Textbook of autism spectrum disorders* (pp. 385–394). Washington, DC: American Psychiatric Publishing.
- Hill, E. (2004). Executive dysfunction in autism. *Trends in Cognitive Sciences*, 8, 26–32.
- Hovik, K. T., Egeland, J., Isquith, P. K., Gioia, G., Skogli, E. W., Andersen, P. N., & Øie, M. (2017). Distinct patterns of everyday executive function problems distinguish children with Tourette Syndrome from children with ADHD or autism spectrum disorders. *Journal of Attention Disorders*, 21, 811–823.
- Hughes, C. (2001). Executive dysfunction in autism: Its nature and implications for the everyday problems experienced by individuals with autism. In J. Burack, T. Charman, N. Yirmiya, & P. Zelazo (Eds.), *The development of autism, perspectives from theory and research* (pp. 255–275). Mahwah: Lawrence Erlbaum Associates.
- Just, M. A., Cherkassky, V. L., Keller, T. A., Kana, R. K., & Minshew, N. J. (2007). Functional and anatomical cortical underconnectivity in autism: Evidence from an fMRI study of an executive function task and corpus callosum morphometry. *Cerebral Cortex*, 17, 951–961.
- Kenworthy, L., Anthony, L. G., Naiman, D. Q., Cannon, L., Wills, M. C., Werner, M. A., Alexander, K., Strang, J., Bal, E., Sokoloff, J. L., & Wallace, G. L. (2014). Randomized controlled effectiveness trial of executive function intervention for children on the autism spectrum. *Journal of Child Psychology and Psychiatry*, 55, 374–383.
- Kenworthy, L. E., Black, D. O., Wallace, G. L., Ahluvalia, T., Wagner, A. E., & Sirian, L. M. (2005). Disorganization: The forgotten executive dysfunction in high-functioning autism (HFA) spectrum disorders. *Developmental Neuropsychology*, 28, 809–827.
- Kuschner, E. S., Bodner, K. E., & Minshew, N. J. (2009). Local vs. global approaches to reproducing the Rey Osterrieth Complex Figure by children, adolescents,

- p>and adults with high-functioning autism.
- International Society for Autism Research*
- , 2, 348–358.
- Mesibov, G., Shea, V., & Schopler, E. (2004). *The TEACCH approach to autism spectrum disorders*. New York: Springer.
- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. (2000). The unity and diversity of executive functions and their contributions to complex “frontal lobe” tasks: A latent variable analysis. *Cognitive Psychology*, 41, 49–100.
- Moore, S. T. (2002). *Asperger syndrome and the elementary school experience, practical solutions for academic and social difficulties*. Shawnee Mission, KS: Autism Asperger Publishing.
- O’Hearn, K., Asato, M., Ordaz, S., & Luna, B. (2008). Neurodevelopment and executive function in autism. *Development and Psychopathology*, 20, 1103–1132.
- Ozonoff, S., Cook, I., Coon, H., Dawson, G., Joseph, R. M., Klin, A., et al. (2004). Performance on Cambridge neuropsychological test automated battery subtests sensitive to frontal lobe function in people with autistic disorder: Evidence from the collaborative programs of excellence in autism network. *Journal of Autism and Developmental Disorders*, 34, 139–150.
- Ozonoff, S., South, M., & Provençal, S. (2005). Executive functions. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *The handbook of autism and pervasive developmental disorders* (pp. 606–627). Hoboken: Wiley.
- Prior, M. R., & Hoffman, W. (1990). Neuropsychological testing of autistic children through an exploration with frontal lobe tests. *Journal of Autism and Developmental Disorders*, 20, 581–590.
- Pugliese, C. E., Anthony, L. G., Strang, J. F., Dudley, K., Wallace, G. L., Naiman, D. Q., & Kenworthy, L. (2016). Longitudinal examination of adaptive behavior in autism spectrum disorders: influence of executive functions. *Journal of Autism and Developmental Disorders*, 46, 467–477.
- Robinson, S., Goddard, L., Dritschel, B., Wisley, M., & Howlin, P. (2009). Executive functions in children with autism spectrum disorders. *Brain and Cognition*, 71, 362–368.
- Rosenthal, M., Wallace, G. L., Lawson, R., Wills, M. C., Dixon, E., Yerys, B. E., & Kenworthy, L. (2013). Impairments in real world executive function increase from childhood to adolescence in autism spectrum disorders. *Neuropsychology*, 27, 529–536.
- Rumsey, J. M. (1985). Conceptual problem-solving in highly verbal, nonretarded autistic men. *Journal of Autism and Developmental Disorders*, 15, 23–36.
- Stuss, D. T. (2007). New approaches to prefrontal lobe testing. In B. Miller & J. Cummings (Eds.), *The human frontal lobes: functions and disorders* (2nd ed., pp. 292–305). New York: Guilford Press.
- Tsatsanis, K. D. (2005). Neuropsychological characteristics in autism and related conditions. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *The handbook of autism and pervasive developmental disorders* (pp. 365–381). Hoboken: Wiley.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know*. Hoboken: Wiley.
- Wallace, G. L., Kenworthy, L., Pugliese, C. E., Popal, H. S., White, E. I., Brodsky, E., & Martin, A. (2016). Real-world executive functions in adults with autism spectrum disorder: profiles of impairment and associations with adaptive functioning and co-morbid anxiety and depression. *Journal of Autism and Developmental Disorders*, 46, 1071–1083.

Organized Work Systems

► Structured Teaching

Orienting Response

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Definition

Orienting as has been defined as the aligning of attention with a source of sensory input (Posner et al. 1998). Research in autism has focused on how individuals with autism orient to different types of stimuli (e.g., visual, auditory, social, non-social) in different contexts (e.g., computer paradigms, naturalistic settings). Further, the components to orienting, disengagement of attention and shifting attention, have been studied to evaluate possible deficits in both areas. Results have varied, however, depending upon the age of participants, comparison groups, and orienting paradigms. Considerable research has found that children with autism orient less frequently to social information than their peers and that their orienting to nonsocial stimuli is relatively less impaired (Dawson et al. 1998, 2004; Swettenham et al. 1998). Further, social-orienting impairments are more commonly observed in natural settings than when completing computerized tasks (Greene et al. 2011). Social-orienting impairments, often

recognized clinically when children do not respond to hearing their name called, are among the earliest observable symptoms of autism (e.g., Baranek 1999; Nadig et al. 2007). Research has demonstrated that children who orient more readily to social stimuli develop stronger joint attention and language skills over time (Dawson et al. 2004), supporting theories of the potential pivotal role social orienting may play in development of children with autism (Dawson and Lewy 1989; Mundy and Neal 2001).

References and Reading

- Baranek, G. T. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders*, 29(3), 213–224.
- Dawson, G., & Lewy, A. (1989). Arousal, attention, and the socioemotional impairments of individuals with autism. In G. Dawson (Ed.), *Autism: Nature, diagnosis, and treatment* (pp. 49–74). New York: The Guilford Press.
- Dawson, G., Meltzoff, A. N., Osterling, J., Rinaldi, J., & Brown, E. (1998). Children with autism fail to orient to naturally occurring social stimuli. *Journal of Autism and Developmental Disorders*, 28(6), 479–485.
- Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., et al. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology*, 40(2), 271–283. Research Support, U.S. Gov't, P.H.S.
- Greene, D. J., Colich, N., Iacoboni, M., Zaidel, E., Bookheimer, S. Y., & Dapretto, M. (2011). Atypical neural networks for social orienting in autism spectrum disorders. *NeuroImage*, 56(1), 354–362.
- Mundy, P., & Neal, R. (2001). Neural plasticity, joint attention, and a transactional social-orienting model of autism. *International Review of Research in Mental Retardation*, 23, 139–168.
- Nadig, A. S., Ozonoff, S., Young, G. S., Rozga, A., Sigman, M., & Rogers, S. J. (2007). A prospective study of response to name in infants at risk for autism. *Archives of Pediatrics & Adolescent Medicine*, 161(4), 378–383.
- Posner, M. I., Rothbart, M. K., Thomas-Thrapp, L., & Gerardi, G. (1998). The development of orienting to locations and objects. In R. D. Wright (Ed.), *Visual attention* (Vol. 8, pp. 269–288). New York: Oxford University Press.
- Swettenham, J., Baron-Cohen, S., Cox, A., Baird, G., Drew, A., Charman, T., et al. (1998). The frequency and distribution of spontaneous attention shifts between social and nonsocial stimuli in autistic, typically developing, and nonautistic developmentally delayed infants. *Journal of Child Psychology and Psychiatry*, 39(5), 747–753.

Ornitz, Edward

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Name and Degrees

Edward M Ornitz

- 1945–1948 B.S. Stanford University, Palo Alto, California
- 1948–1952 M.D. Stanford University, School of Medicine, Palo Alto, California.

Major Appointments (Institution, Location, Dates)

- 1952–1953 Internship, Los Angeles County General Hospital, Los Angeles, California
- 1955–1958 Psychiatry Residence and Research Fellow, Yale University School of Medicine, New Haven, CT
- 1958–1960 Fellow in Child Psychiatry, Child Study Center, Yale University, New Haven, CT
- December 1960–February 1965 Staff Psychiatrist, Reiss-Davis Clinic for Child Guidance, Los Angeles, California.
- September 1963–July 2006 Clinical Instructor, Assistant, Associate, and Full Professor in Residence, Department of Psychiatry, UCLA School of Medicine.
- July 2006–Present Professor Emeritus, Department of Psychiatry, UCLA School of Medicine.

Major Honors and Awards

Dr. Ornitz has been invited to and delivered presentations at numerous national and international meetings. He has been the recipient of numerous grants.

Landmark Clinical, Scientific, and Professional Contributions

A pioneer in the study of psychophysiological methods in the developmental neuropsychiatric disorders, Dr. Ornitz's research has focused on brain mechanisms used to modulate arousal and stress. In addition to his long-standing interest in autism, he has conducted work in areas such as anxiety disorders, response to pain, posttraumatic stress disorder, and various medical conditions. His laboratory has developed many techniques now widely used in the field.

Short Biography

A pioneer in the study of neurobiological process in autism, Dr. Ornitz and his colleagues were among the first to question the widespread early views of psychogenetic models of autism. This work included earliest studies of a range of neurophysiological processes including responses to vestibular stimulation, sleep, and brain stem-evoked responses. He also worked to apply some of the measures he developed in study of anxiety and PTSD. The author of 129 scientific publications, Dr. Ornitz was an active teacher as well as a research and trained several generations of workers at UCLA.

See Also

- [Anxiety](#)
- [Electrophysiology](#)

References and Reading

- Frankland, P. W., Wang, Y., Rosner, B., Shimizu, T., Balleine, B. W., Dykens, E. M., et al. (2004). Sensorimotor gating abnormalities in young males with fragile X syndrome and Fmr1-knockout mice. *Molecular Psychiatry*, 9(4), 417–425.
- Ornitz, E. M. (1974). The modulation of sensory input and motor output in autistic children. *Journal of Autism and Childhood Schizophrenia*, 4(3), 197–215.
- Ornitz, E. M. (1985). Neuropsychology of infantile autism. *Journal of the American Academy of Child Psychiatry*, 24(3), 251–262.

- Ornitz, E. M., & Ritvo, E. R. (1976). The syndrome of autism: A critical review. *American Journal of Psychiatry*, 133(6), 609–621.
- Ornitz, E. M., Ritvo, E. R., Panman, L. M., Lee, Y. H., Carr, E. M., & Walter, R. D. (1968). The auditory evoked response in normal and autistic children during sleep. *Electroencephalography and Clinical Neurophysiology*, 25(3), 221–230.
- Ornitz, E. M., Ritvo, E. R., Brown, M. B., La Franchi, S., Parmelee, T., & Walter, R. D. (1969). The EEG and rapid eye movements during REM sleep in normal and autistic children. *Electroencephalography and Clinical Neurophysiology*, 26(2), 167–175.
- Ornitz, E. M., Lane, S. J., Sugiyama, T., & de Traversay, J. (1993). Startle modulation studies in autism. *Journal of Autism and Developmental Disorders*, 23(4), 619–637.
- Stark, Z., Bruno, D. L., Mountford, H., Lockhart, P. J., & Amor, D. J. (2010). De novo 325 kb microdeletion in chromosome band 10q25.3 including ATRNL1 in a boy with cognitive impairment, autism and dysmorphic features. *European Journal of Medical Genetics*, 53(5), 337–339.
- Tanguay, P. E., Ornitz, E. M., Forsythe, A. B., & Ritvo, E. R. (1976). Rapid eye movement (REM) activity in normal and autistic children during REM sleep. *Journal of Autism and Childhood Schizophrenia*, 6(3), 275–288.
- Yuhas, J., Cordeiro, L., Ballinger, E., Schneider, A., Hessel, D., Tassone, F., et al. (2011). Brief report: Sensorimotor gating in idiopathic autism and autism associated with fragile X syndrome. *Journal of Autism and Developmental Disorders*, 41(2), 248–253.

Orofacial Imitation

- [Oral-Facial Imitation](#)

Otitis Media

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Synonyms

[Middle ear infection](#)

Definition

Otitis media is a middle ear infection that can cause a temporary hearing loss in the affected ear, or ears. The middle ear is a closed cavity in the temporal bone that is connected to the nasopharynx by the Eustachian tube. In early stages of otitis media (acute otitis media), the middle ear space becomes inflamed and pressure builds causing the tympanic membrane to stretch leading to intense pain. Acute otitis media can be treated with antibiotics, but most cases of otitis media resolve on their own within 2–4 weeks. When otitis media becomes chronic, fluid builds up in the middle ear space, but it is less likely to be painful.

Chronic otitis media is not typically responsive to antibiotic treatment but can be treated with tympanostomy tubes. Tympanostomy tubes can be placed in the tympanic membrane when the condition is either chronic, causing a hearing loss greater than 15 dB, influencing speech/language, or disrupting the learning process in school. Otitis media is reportedly more common in individuals with autism spectrum disorders than in the general population (Konstantareas and Homatidis 1987; Rosenhall et al. 1999; Smith et al. 1988), but more systematic research needs to be done to further investigate this relationship.

See Also

► [Auditory System](#)

References and Reading

- Clarke, W., & Ohlemiller, K. (2008). *Anatomy and physiology of hearing for audiologists*. Clifton Park: Thomson, Delmar Learning.
- Konstantareas, M. M., & Homatidis, S. (1987). Ear infections in autistic and normal children (Brief report). *Journal of Autism and Developmental Disorders*, 17, 585–593.
- Musiek, F. E., & Baran, J. A. (2007). *The auditory system: Anatomy, physiology, and clinical correlates*. Boston: Pearson.
- Rosenhall, U., Nordin, V., Sandstrom, M., Ahlsen, G., & Gillberg, C. (1999). Autism and hearing loss. *Journal of Autism and Developmental Disorders*, 29(5), 349–357.
- Smith, D. E. P., Miller, S. D., Stewart, M., Walter, T. L., & McConnell, J. V. (1988). Conductive hearing loss in autistic, learning-disabled, and normal children. *Journal of Autism and Developmental Disorders*, 18, 53–65.

Outcome

- [Course of Development](#)
- [Longitudinal Research in Autism](#)

Outcome Studies

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Definition

Outcome studies describe the progress of individuals with autism as they move through adolescence and into adulthood. Some such studies have followed up groups of individuals first diagnosed as children over a number of years; others have involved investigations of different individuals at different ages. Follow-up studies of the same individuals from child to adulthood are particularly informative as they allow the investigation of factors in early childhood that may help to predict outcome in later years.

Historical Background

The earliest studies of outcome in autism were conducted in the 1950s by Leon Eisenberg (a colleague of Leo Kanner) in USA and Mildred Creak in England. Eisenberg, writing in the 1950s, documented the wide variety of possible outcomes. Many of the individuals he described

remained very dependent on others for support, but about one-third were found to have made at least a “moderate social adjustment,” despite the lack of any specialist provision or treatment available at that time. A minority had managed to achieve good independence, although even among this group social impairments remained apparent. In a slightly later report from Britain, Mildred Creak described 100 cases of “childhood psychosis,” the term then often used for children with autism. A minority was rated as having made significant improvements, but around half were permanently institutionalized. Some years later, Kanner followed up 96 young adults whom he had first diagnosed as children. Just over 10% were described as achieving relatively highly as adults and “mingling, working, and maintaining themselves in society.” Kanner (1973) noted that in the majority of these cases, “a remarkable change took place” around their mid-teens. “Unlike most other autistic children they became uneasily aware of their peculiarities and began to make a conscious effort to do something about them.” In particular, they tried to improve their interactions with their peers, often using their obsessional preoccupations or special skills “to open a door for contact.” Asperger (1944) also reported positive outcomes for some of the individuals he had initially described, including a professor of astronomy, mathematicians, technologists, chemists, high-ranking civil servants, and an expert in heraldry. Again, it was often individuals’ special skills or interests that facilitated social integration. However, like Eisenberg, both Asperger (1944) and Kanner (1971) highlighted the wide heterogeneity in outcome among individuals with autism, and how trajectories of development could change markedly over the years “ranging all the way from complete deterioration to a combination of occupational adequacy with limited, though superficially smooth social adjustment” (Kanner 1971).

Over the course of the last 60–70 years, many subsequent studies have been conducted exploring the prognosis for individuals with autism as they move into adulthood, and the factors affecting outcome.

Current Knowledge

In recent years, there has been a steady increase in the number of studies that have followed up individuals with autism from childhood into early adulthood and beyond. In order to facilitate comparisons between studies, many such studies have adopted a somewhat simplified categorization system, classifying participant outcomes as “very good” if they were employed, had friends, and high levels of independence; “good” if they were working with support, had some friends, and could travel independently; “fair” if they required support in daily living but had some autonomy; “poor” if they had very limited autonomy; and “very poor” if they were living in highly specialized or very restrictive environments. Magiati et al. (2014) conducted a systematic review of follow-up studies published between 1967 and 2013 (25 studies; n participants = 2043; age range 17–44 years). Although the average IQ of most of the cohorts involved was relatively high (mean = 79.0), almost 50% of participants were rated as having “poor” or “very poor” outcomes. Most remained highly dependent on others for their care and few had developed close friendships or intimate relationships. Average employment rates were 30%, and among those in employment, jobs were mainly low-skilled, part time, and very poorly paid. Indeed, adults with autism tended to be more economically, educationally, and socially disadvantaged than other groups of individuals with developmental or intellectual disabilities (Roux et al. 2013). A subsequent review by Steinhausen et al. (2016) reported similar, rather depressing findings. Thus, of the 12 studies included in that review (dates 1967 to 2014; n = 828 individuals), 48% of participants were rated as having a “poor”/“very poor” outcome, and only in 20% was outcome classified as “Good.” Outcomes for some recent cohorts of individuals with higher levels of ability are somewhat more positive (e.g., Gotham et al. (2015), Helles et al. (2017), Roy et al. (2015), and Taylor et al. (2015). See Howlin and Magiati 2017 for review), but even among these groups, social attainments are much lower than in the general population.

In their review, Steinhausen et al. (2016) also highlight the wide variation across studies. Thus, the mean estimated percentage of individuals reported to have a “good outcome” was 20% but with 95% confidence intervals varying from 14% to 27%; confidence intervals for the proportion with a “poor outcome” ranged from 37% to 59%. Many of these differences are due to the heterogeneity of the cohorts involved, with respect to age, IQ and language, autism severity, and many other factors. Different outcome measures also add to the complexity of comparing across studies, or over time. In particular, there is growing dissatisfaction with traditional “objective” measures of outcome that focus primarily on employment, jobs, independent living, and close relationships. More recently, therefore, there has been a move toward more subjective outcome measures that may be more appropriate for individuals with autism. These include factors such as satisfaction with life, good physical and mental health, adequate living conditions, and supportive and positive social and family relationships. When these variables are taken into account, adult outcomes can appear much more positive than usually reported (Bishop-Fitzpatrick et al. 2016; Hong et al. 2016; Helles et al. 2017).

Although “normative” measures of social outcome generally indicate that, as a group, adults with autism are much more socially, economically, and educationally disadvantaged than their peers, follow-up studies have also been important in highlighting the very positive changes that can occur at an individual level. Thus, typically, there are improvements in verbal and nonverbal communication and in social interactions, while ritualistic, stereotyped, and “maladaptive” behaviors tend to decrease from child- to adulthood (Lord et al. 2015; Woodman et al. 2015; see Howlin and Magiati 2017 for review). However, it is also evident that individual trajectories can be very variable, and it remains very difficult accurately to predict adult outcomes from early child characteristics. Lord et al. (2015) have described a number of different trajectories of development in cognitive, verbal, and social skills, and in repetitive behaviors, and they highlight the importance of further work in this area, both for identifying the specific genetic etiologies

underlying different phenotypes, and for developing more individually tailored interventions.

Future Directions

It is evident that, in the 70 years since autism was first formally described by Kanner, there have been significant improvements in outcome. In cohorts studied before 1980, almost no individuals had received full-time education, very few lived independently or had jobs, and over half of all adults were placed in long-stay hospitals or institutions for people with mental retardation (Howlin 2014). For example, in the very early studies of Rutter and colleagues (Lockyer and Rutter 1969, 1970; Rutter et al. 1967), most children had never attended full-time school and many spent their adult years in institutions. Over the last few decades, access to early diagnosis, more intensive intervention programs, specialist educational support, in both school and college, and supported employment programs have greatly increased. There have also been increases in the numbers of individuals living in their own homes, either independently or with some minimal supervision, and of the numbers of individuals in employment (see Howlin and Moss 2012). Nevertheless, the overall findings from current follow-up research indicate a poor prognosis for most individuals with autism, at least when standard measures of “good” social outcome are employed. Relatively few individuals, even those of high IQ, achieve full independence and social integration in adult life. The financial costs of supporting adults with autism also remain very high, due to their continuing need for medical care, residential or supported living accommodation, and to productivity loss, both by caregivers as well as by individuals themselves (Leigh and Du 2015). Furthermore, despite the huge expansion in intervention research for children with autism over recent years, there is, so far, little evidence that early intervention programs have had any significant impact on improving life-time prospects. In the field of autism, adult intervention research lags far behind child intervention research in both quantity and quality (NICE 2012). A lack

of appropriate services for adults, especially those of average IQ, also means that they remain very disadvantaged compared with the general population and indeed with adults with other forms of disability (Roux et al. 2013). There is also a significant gap in knowledge about what happens to individuals with autism as they approach old age (Wright et al. 2016). Almost all current adult outcome studies have focused on younger individuals in their 20s to 40s. There is little research on individuals in the middle years and no large-scale studies of outcome in those aged over 60. To date, knowledge about the aging process in autism is extremely limited and systematic information about the physical and mental health needs of elderly people with autism is lacking. For example, it is not known whether rates of dementia are likely to be higher or lower in this group than in the population as a whole. It is also evident that many individuals remain highly dependent on their families for support throughout adulthood. How they will cope when parents are no longer able to support them is a further issue with major implications for society as a whole.

See Also

- [Adult Follow-Up Studies](#)
- [Adulthood, Transition To](#)
- [Asperger Syndrome](#)
- [Course of Development](#)
- [Employment](#)
- [Employment in Adult Life](#)
- [Factors Affecting Outcome](#)
- [Living Arrangements in Adulthood](#)
- [Optimal Outcome](#)
- [Psychotic Disorder](#)

References and Reading

- Asperger, H. (1944). "Autistic psychopathy" in childhood (U. Frith, trans., and annotated in autism and Asperger syndrome (1991)). In U. Frith (Ed.), *Autism and asperger syndrome* (1st ed., pp. 37–92). Cambridge: Cambridge University Press.
- Bishop-Fitzpatrick, L., Hong, J., Smith, L. E., Makuch, R. A., Greenberg, J. S., & Mailick, M. R. (2016). Characterizing objective quality of life and normative outcomes in adults with autism spectrum disorder: An exploratory latent class analysis. *Journal of autism and developmental disorders*, 46(8), 2707–2719.
- Creak, M. E. (1963). Childhood psychosis: A review of 100 cases. *The British Journal of Psychiatry*, 109, 84–89.
- Eisenberg, L. (1956). The autistic child in adolescence. *The American Journal of Psychiatry*, 112, 607–612.
- Gotham, K., Marvin, A. R., Taylor, J. L., et al. (2015). Characterizing the daily life, needs, and priorities of adults with autism spectrum disorder from interactive autism network data. *Autism*, 19(7), 794–804.
- Helles, A., Gillberg, I. C., Gillberg, C., & Billstedt, E. (2017). Asperger syndrome in males over two decades: Quality of life in relation to diagnostic stability and psychiatric comorbidity. *Autism*, 21(4), 458–469.
- Hong, J., Bishop-Fitzpatrick, L., Smith, L. E., et al. (2016). Factors associated with subjective quality of life of adults with autism spectrum disorder: Self-report versus maternal reports. *Journal of Autism and Developmental Disorders*, 46(4), 1368–1378.
- Howlin, P. (2014). Outcomes in adults with autism Spectrum disorders. In F. Volkmar, S. J. Rogers, R. Paul, & K. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (4th ed., pp. 97–116). Hoboken: Wiley.
- Howlin, P., & Magiati, I. (2017). Autism spectrum disorder: outcomes in adulthood. *Current Opinion in Psychiatry*, 30(2), 69–76.
- Howlin, P., & Moss, P. (2012). Adults with autism spectrum disorders. *Canadian Journal of Psychiatry*, 57, 275–283.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of autism and childhood schizophrenia*, 1(2), 119–145.
- Kanner, L. (1973). *Childhood psychosis: Initial studies and new insights*. New York: Winston/Wiley.
- Leigh, J. P., & Du, J. (2015). Brief report: Forecasting the economic burden of autism in 2015 and 2025 in the United States. *Journal of Autism and Developmental Disorders*, 45(12), 4135–4139.
- Lockyer, L., & Rutter, M. (1969). A five to fifteen year follow-up study of infantile psychosis: III. Psychological aspects. *The British Journal of Psychiatry*, 115, 865–882.
- Lockyer, L., & Rutter, M. (1970). A five to fifteen year follow-up study of infantile psychosis: IV. Patterns of cognitive ability. *The British Journal of Social and Clinical Psychology*, 9, 152–163.
- Lord, C., Bishop, S., & Anderson, D. (2015). Developmental trajectories as autism phenotypes. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics*, 169(2), 198–208.
- Magiati, I., Tay, X. W., & Howlin, P. (2014). Cognitive, language, social and behavioural outcomes in adults with autism spectrum disorders: A systematic review of longitudinal follow-up studies in adulthood. *Clinical Psychology Review*, 34, 73–86.

- Moss, P., Howlin, P., Savage, S., Bolton, P., & Rutter, M. (2015). Self and informant reports of mental health difficulties among adults with autism: Findings from a long-term follow-up study. *Autism, 19*(7), 832–841.
- National Institute for Health and Clinical Excellence (NICE). (2012). Autism: Recognition, referral, diagnosis and management of adults on the autism spectrum. *Clinical guideline*, 142. <http://guidance.nice.org.uk/CG142>.
- Roux, A. M., Shattuck, P. T., Cooper, B. P., et al. (2013). Postsecondary employment experiences among young adults with an autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 52*(9), 931–939.
- Roy, M., Prox-Vagedes, V., Ohlmeier, M. D., & Dillo, W. (2015). Beyond childhood: Psychiatric comorbidities and social background of adults with Asperger syndrome. *Psychiatria Danubina, 27*(1), 50–59.
- Rutter, M., Greenfield, D., & Lockyer, L. (1967). A five to fifteen year follow-up study of infantile psychosis. II. Social and behavioural outcome. *The British Journal of Psychiatry, 113*, 1183–1199.
- Steinhausen, H. C., Mohr Jensen, C., & Lauritsen, M. B. (2016). A systematic review and meta-analysis of the long-term overall outcome of autism spectrum disorders in adolescence and adulthood. *Acta Psychiatrica Scandinavica, 133*(6), 445–452.
- Taylor, J. L., Henninger, N. A., & Mailick, M. R. (2015). Longitudinal patterns of employment and post-secondary education for adults with autism and average-range IQ. *Autism, 19*(7), 785–793.
- Woodman, A. C., Smith, L. E., Greenberg, J. S., & Mailick, M. R. (2015). Change in autism symptoms and maladaptive behaviors in adolescence and adulthood: The role of positive family processes. *Journal of Autism and Developmental Disorders, 45*(1), 111–126.
- Wright, S. D., Wright, C. A., D'Astous, V., & Wadsworth, A. M. (2016). Autism aging. *Gerontology & Geriatrics Education, 17*, 1–17.

Overcorrection

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Definition

Overcorrection is a behavioral intervention developed by applied behavior analysts and is based on

the belief that the challenging behavior of persons with autism spectrum disorders are maintained by social factors (e.g., attention from other people, escape or avoidance from low-preference academic activities such as math) or nonsocial factors (e.g., sensory reinforcement). Overcorrection is a name given to a package of procedures that consists of two components (restitution and/or positive practice). Restitution consists of requiring the person displaying challenging behavior to correct their actions to a state that is vastly improved from what existed prior the occurrence of the challenging behavior, sometimes repeatedly. For example, if a child throws a toy truck at his or her teacher, they might be required to pick up the truck and then clean up the play area of their classroom. Positive practice consists of having an individual engage an adaptive behavior that is functionally incompatible with their challenging behavior (e.g., playing with the truck appropriately and then putting it away in the correct location), sometimes repeatedly. Depending on the problem behavior targeted, restitution and positive practice may be used either in combination or alone.

Historical Background

Overcorrection is an applied behavior analytic intervention developed by Richard Foxx and Nathan Azrin in the early 1970s. In their early studies, Foxx and Azrin demonstrated that overcorrection could successfully reduce a wide range of challenging behavior including (a) aggression toward other people, (b) biting of fingernails, (c) inappropriate mealtime behavior (e.g., tossing food on the floor), (d) pica (e.g., eating nonedible objects), (e) stereotypic or other interfering repetitive behavior, (f) self-injury, and (g) toileting accidents of individuals with autism and other developmental disabilities (Foxx and Bechtel 1983). For example, in 1974, Azrin and Wesolowski showed that overcorrection could be used to reduce the food stealing of 34 persons with autism or other intellectual disabilities living in an institution. If a person took food from another person, they were required to return the food (restitution) and to buy more food and give

it to the person from whom they had stolen (positive practice). Since the publication of Foxx and Azrin's initial studies, multiple independent researchers have successfully replicated the findings of their studies providing data-based evidence that overcorrection is an evidence-based intervention. **That is, overcorrection meets the standards for independent replication established by panels of experts.**

Rationale or Underlying Theory

In the behavior analytic literature, overcorrection is classified as a punishment-based strategy (Foxx 1982; Foxx and Bechtel 1983). If overcorrection is used contingent on the occurrence of a targeted challenging behavior and the frequency of the behavior decreases in the future, then the behavioral definition of punishment is met (see Azrin and Holz 1966 for a technical behavior analytic definition of punishment). According to Foxx and Bechtel (1983), overcorrection also often works due to negative reinforcement. That is, individuals often engage in behaviors designed to avoid or terminate its use. In their early publications, Foxx and Azrin provided two rationales for the use of overcorrection. First, overcorrection was described as promoting "normalization" by employing consequences which typically developing people apply to themselves when they display challenging behavior which is judged to be socially inappropriate. For example, someone who is angry and throws paper on the floor would pick it up. Second, overcorrection, unlike most other behavioral reductive procedures (e.g., time-out, response cost), provides the clinician with the advantage of being able to provide an educative consequence which is functionally equivalent to an individual's challenging behavior. Thus, overcorrection may be beneficial to an individual by helping them experience the effort required by other people to correct the damage caused by their challenging behavior. Some researchers also have argued that when overcorrection includes a positive practice component, it is less restrictive than other punishment-based strategies (e.g., time-out; contingent exercise)

because it also teaches adaptive functionally equivalent behavior. Although overcorrection has typically been implemented contingent on the occurrence of challenging behavior, the authors of a small number of studies have implemented it based on the absence of a behavior. For example, Foxx (1977) used overcorrection to increase the eye contact of children with autism and intellectual disabilities (e.g., contingent on the lack of eye contact, a child was required to practice looking at the face of another person).

Goals and Objectives

Overcorrection is an intervention designed to reduce the challenging behavior of persons with autism (e.g., aggression, property destruction, self-injury, and pica).

Treatment Participants

Individuals who are dually diagnosed with an autism spectrum disorder and a moderate to profound intellectual disability are most likely to benefit from overcorrection. Prior to the participation in the clinical use of overcorrection, a comprehensive functional behavioral assessment should be conducted and less restrictive; potentially effective interventions should be attempted (e.g., functional communication training, differential reinforcement of other behavior). Given the restrictiveness of the intervention, evaluation of overcorrection using a single-case experimental design that rules out threats to internal validity and the conducting of frequent fidelity checks is recommended.

Treatment Procedures

The published literature contains a variety of procedural variations of overcorrection. Both the topography of the overcorrection responses and duration of the application have differed widely from study to study. Gross motor movements with

physical guidance termed “functional movements training” by Foxx and Azrin (1973) are the most commonly used positive practice procedure reported in the literature. This procedure involves instructing a person to hold a body part in a particular position for predetermined length of time while providing physical guidance to ensure that the person engages in the correct topographical form for the required number of repetitions. Contingent on stereotypic hand clapping, Foxx and Azrin (1973), for example, required a 7-year-old boy with autism to engage in 5 min of functional movement training. The boy was required to engage in a series of hand placements each for approximately 15 s (e.g., hands above head, hands straight out in front of him, hands in pockets). While the majority of studies have used gross motor movements which are topographically similar to the target behavior, a few studies have employed arbitrary movements suggesting that the contingent relationship between the targeted behavior and motor movements is more important than the similarity of the topography. For problem behavior involving physical aggression toward other people or property destruction, positive practice typically involves requiring a person to be quiet and relaxed for a prespecified period of time (e.g., laying calmly on a mat for 15 min before practicing picking up and putting away toys). For inappropriate mouthing or ingesting of objects, positive practice usually involves cleaning a person’s mouth and lips with mouthwash contingent on the targeted behavior. In the published literature, researchers have examined the use of varying durations of overcorrection (e.g., 10 s, 15 min). However, the lack of parametric studies comparing the use of varying durations of overcorrection makes it difficult to identify the most appropriate duration to recommend to clinicians.

Efficacy Information

A variety of strengths and limitations of overcorrection have been identified in the published literature. First, several studies have demonstrated that overcorrection has produced a clinically significant reduction in behavior after other behavioral

interventions had failed (e.g., differential reinforcement of other behavior, verbal reprimands, time-out, simple correction). Second, published research studies using single-case experimental methods have shown that often overcorrection produces a rapid reduction in challenging behavior for many participants. Therefore, overcorrection may be a useful intervention strategy for persons whose challenging behavior routinely results in injury to self or other people (e.g., bruising) or is potentially life threatening (e.g., pica). Researchers have also suggested that the educative aspects of positive practice shift the attention of parent and teacher from focusing on the suppression of problem behavior to the adaptive skills that need to be taught to replace the challenging behavior.

Several potential limitations of overcorrection also have been identified. First, overcorrection requires the person participating in the intervention to engage in the corrective and alternative behaviors immediately following the occurrence of challenging behavior. Although many people immediately respond to verbal instructions to engage in the corrective and alternative behaviors, some people require physical prompts to do so. A person may resist attempts to physically prompt them, and in some cases, they may become aggressive or require a fair amount of physical effort to guide them through the intervention. Alternative interventions should be used with individuals who are too aggressive or resistant to potentially experience injuries. Another potential limitation is the high level of supervision and time required to implement overcorrection. In order to implement overcorrection correctly, the person implementing it needs to be continuously available and in close-enough physical proximity to implement the intervention and to consistently use physical prompting, when necessary.

Since the publication of Foxx and Azrin’s early research, there has been much debate about the use of punishment-based intervention strategies. This debate has centered around two positions. From a right-to-effective treatment perspective, some people have argued that persons with autism and other developmental disabilities have the right to the most effective treatment and for some individuals, this might support the use of a potentially quick-acting punishment-based

intervention. Other people argue that advancements in intervention strategies such as functional communication have eliminated the need for the use of punishment-based intervention strategies. The controversy concerning the pros and cons related to the use of punishment-based interventions warrants careful consideration by parents, teachers, and clinicians prior to the implementation of overcorrection.

The majority of research studies evaluating overcorrection have used single-case research designs and methods and have demonstrated that overcorrection generally produced a clinically significant change in behavior. The overcorrection literature could potentially benefit from the conducting of clinical trials, the manualization of intervention procedures, and the conducting of quantitative meta-analytic reviews.

Outcome Measures

Direct observational measures such as frequency or rate of occurrence of the targeted behavior or percentage of observational intervals in which the target problem behavior occurs are the outcome measures used to evaluate whether overcorrection produces a clinically significant reduction.

Qualifications of Treatment Providers

Minimal qualifications for the implementation of overcorrection include a master's degree or doctoral degree in applied behavior analysis or psychology with specific training in functional behavioral assessment and applied behavior analytic interventions for challenging behavior.

References and Reading

- Azrin, N. H., & Holz, W. C. (1966). Punishment. In W. K. Honig (Ed.), *Operant behavior: Areas of research and application*. New York: Appleton-Century-Crofts.
- Azrin, N. H., & Wesolowski, M. D. (1974). Theft reversal: An overcorrection procedure for eliminating stealing by retarded persons. *Journal of Applied Behavior Analysis*, 7, 577–581.
- Foxx, R. M. (1982). *Decreasing behaviors of severely retarded and autistic persons*. Champaign: Research Press.

- Foxx, R. M., & Azrin, N. H. (1972). Restitution: A method of eliminating aggressive-disruptive behavior of retarded and brain damaged patients. *Behaviour Research and Therapy*, 10, 15–27.
- Foxx, R. M., & Azrin, N. H. (1973). The elimination of autistic self-stimulatory behavior by overcorrection. *Journal of Applied Behavior Analysis*, 6, 1–14.
- Foxx, R. M. (1977). Attention training: The use of overcorrection avoidance to increase the eye contact of autistic and retarded children. *Journal of Applied Behavior Analysis*, 10, 489–499. <https://doi.org/10.1901/jaba.1977.10-489>.
- Foxx, R. M., & Bechtel, E. D. (1983). Overcorrection: A review and analysis. In S. Axelrod & J. Apsche (Eds.), *The effects of punishment on human behavior* (pp. 133–220). New York: Academic Press.
- Kratochwill, T. R., Hitchcock, J., Horner, R. H., Levin, J. R., Odom, S. L., Rindskopf, D. M., & Shadish, W. R. (2010). Single-case designs technical documentation. Retrieved from What Works Clearinghouse website: http://ies.ed.gov/ncee/wwc/pdf/wwc_scd.pdf.
- Van Houten, R., Axelrod, S., Bailey, J. S., Favell, J. E., Foxx, R. M., Iwata, B. A., & Lovaas, O. I. (1988). The right to effective behavioral treatment. *The Behavior Analyst*, 11(2), 111–114.

Overly Talkative

- [Mania](#)

Oxazepam

- [Serax](#)

Oxytocin

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Synonyms

[Endocrine](#); [Hormone](#)

Definition

The social and cognitive deficits in autistic spectrum disorder (ASD) patients remain a therapeutic

challenge for both health care professionals and family (Cataldo et al. 2018). In recent years, several clinical studies showed the neuropeptide oxytocin, which facilitates trust, bonding, and communication, is decreased in ASD patients and other neuropsychiatric disorders (Cataldo et al. 2018; Peñagarikano et al. 2015; Vanya et al. 2017; Zhang et al. 2017). Specifically, several clinical studies demonstrated that administration of oxytocin improved social and cognitive skills in animal models and ASD patients (Cai et al. 2018; Cataldo et al. 2018; Peñagarikano et al. 2015; Vanya et al. 2017). Furthermore, these studies indicate that oxytocin improves social-cognitive function through three G-protein coupled receptors: OXTr, AVPR1a, and AVPr1b (Cataldo et al. 2018). Further clinical trials are needed to assess the efficacy and diagnostic potential of oxytocin in the treatment and prognosis of ASD.

See Also

- Hypothalamic-Pituitary-Adrenal Axis
- Sex Hormones

References and Reading

- Cai, Q., Feng, L., & Yap, K. Z. (2018). Systematic review and meta-analysis of reported adverse events of long-term intranasal oxytocin treatment for autism spectrum disorder. *Psychiatry and Clinical Neurosciences*, 72(3), 140–151. <https://doi.org/10.1111/pcn.12627>.
- Cataldo, I., Azhari, A., & Esposito, G. (2018). A review of oxytocin and Arginine-Vasopressin receptors and their modulation of autism spectrum disorder. *Frontiers in Molecular Neuroscience*, 11. <https://doi.org/10.3389/fnmol.2018.00027>.
- Peñagarikano, O., Lázaro, M. T., Lu, X.-H., Gordon, A., Dong, H., Lam, H. A., et al. (2015). Exogenous and evoked oxytocin restores social behavior in the Cntnap2 mouse model of autism. *Science Translational Medicine*, 7(271), 271ra278–271ra278. <https://doi.org/10.1126/scitranslmed.3010257>.
- Vanya, M., Szucs, S., Vetro, A., & Bartfai, G. (2017). The potential role of oxytocin and perinatal factors in the pathogenesis of autism spectrum disorders – Review of the literature. *Psychiatry Research*, 247, 288–290. <https://doi.org/10.1016/j.psychres.2016.12.007>.
- Zhang, R., Zhang, H.-F., Han, J.-S., & Han, S.-P. (2017). Genes related to oxytocin and arginine-vasopressin pathways: Associations with autism spectrum disorders. *Neuroscience Bulletin*, 33(2), 238–246. <https://doi.org/10.1007/s12264-017-0120-7>.

Pain in Autism Spectrum Disorders

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injury/tissue damage), it is often possible to see the noxious input, although this is not always the case. This potential noxious input is then communicated along peripheral fibers before reaching the spine, at which time it can be modulated either up or down. Subsequently, in the brain, neural responses to this noxious input are then contextualized with associated suffering to generate a pain percept.

Definition: Pain

Pain refers to “an unpleasant sensory and emotional experience associated with actual or potential tissue damage.” It is important therefore to understand that pain is more than a simple “ouch” and refers to the emotional, cognitive, and social aspects associated with potentially noxious physical sensations. Therefore, the current entry will focus on responses to tactile, thermal, and chemical stimuli which have the potential to result in unpleasant sensations. This entry will not consider distressing responses to emotional or other sensory experiences; for a review of these, see the entry ► [“Sensory Processing”](#).

To understand both the experience of pain, the expression of pain, and the relationship of these to ASD, it is also important to consider the process by which pain is perceived and then communicated. When considering painful events that start in the periphery (e.g., pain resulting from an

Pain in ASD: Context

In addition to the social-communicative and repetitive behavioral routines which are characteristic of autism, there is a growing recognition that a significant percentage of those who have an autism diagnosis also show evidence of altered sensory responses (for more comprehensive reviews, see ► [“Sensory Features as a Marker of Autism Spectrum Disorders”](#), ► [“Short Sensory Profile in Autism”](#), ► [“Sensory Impairment in Autism”](#), ► [“Sensory Processing”](#), and others). Within the *Diagnostic and Statistical Manual* 5th edition, a specific example of sensory atypicality was given as an “insensitivity to cold/heat/pain.” Thus, a greater understanding of pain responsiveness in ASD is warranted.

Pain response in ASD is an important issue, as pain may be the first or only sign of illness or injury. Importantly, individuals with ASD are both at greater risk of injury and having other medical complaints that require potentially

painful medical procedures; thus, altered pain responsivity could be highly problematic. Concern about a proper understanding of pain in ASD is compounded by the communicative and intellectual challenges present in a significant proportion of individuals with ASD, which may mean many are less able to accurately report the degree and nature of pain experience. Further, self-injurious behaviors and co-occurring pain-related medical conditions, such as chronic gastrointestinal distress, are common in ASD and could reflect altered pain perception. Successful pain management is crucial: pain hyposensitivity can result in inadvertent injury and reduced treatment, while hypersensitivity to innocuous sensory stimuli can result in pain-like behaviors or anxiety. Without a clear understanding of how pain is experienced and communicated in ASD, there is a greater risk for mismanaged pain in ASD.

The aim of the current entry is to discuss the evidence for an altered pain experience in ASD. This entry is organized into a number of subsections: Case Studies, Self-Reported Pain Experiences and Responses, Observational Evidence, Laboratory Psychophysical Studies, and Neuropsychological and Neuroimaging, followed by a Summary with Future Directions for Research.

Case Studies

The earliest accounts of pain alterations in ASD come from a number of either clinical or anecdotal observations of people with ASD who did not display typical signs of pain or distress in response to experiences which most people find painful. Early anecdotal accounts include a child putting a car cigarette lighter into their mouth without crying out or a child who did not respond “even when struck hard.”

Further clinical accounts have more formally reported altered pain behaviors. The most widely discussed case is of a girl, “Sally,” who would play in the snow without clothes on (Wing 1976). Other accounts include a boy who placed his hand on a stove and was only alerted to the burning sensation by the smell.

These cases are often more complex: in two cases adolescents with ASD were reported to respond atypically to injuries (i.e., not responding when holding the hot handle of a frying pan); however, the same individuals reported having frequent headaches and chronic abdominal pain. See below in this entry for more information about chronic pain in ASD (Bursch et al. 2004).

Estimates of the number of individuals with ASD who might have reduced pain sensitivity have suggested that for those in inpatient care, this may be between 25% and 55% who have an observable reduction in pain (Gillberg et al. 1985). These populations were however generally nonverbal, and the modes of determining reductions in pain responses are unclear.

Self-/Parent Report

Self-report of pain is considered the “gold standard” of pain assessment. As such, most studies in ASD populations have compared self-report and caregiver reports of pain to the general population. These measures rely on memories for pain, which may be unreliable, as well as the ability to make judgements of others’ private pain state. Similarly, self-report requires internal assessment of one’s own pain to then make a comparison. This may present particular challenges given the evidence of difficulties with interoception in ASD (see the entry ► “[Interoception](#)” for more information).

Self-reported studies of pain response in ASD provide contrary evidence with some studies suggesting that individuals (8–54 years old) with ASD reported lower pain thresholds compared to IQ-matched typical controls on the sensory sensitivity questionnaire (Minshew and Hobson 2008). However, scores in the Charleston Pediatric Pain Pictures and a Faces Pain Rating Scale suggest no difference with adolescents with ASD- and IQ-matched controls (Bandstra et al. 2012).

Others have taken the alternative approach by asking parents to report on the pain that their children experience. Approximately 40% of parents have reported that their child shows either reduced or greatly reduced pain sensitivity. There are challenges using this approach as this still

involves the ability to compare one person's private experience to another. Parents must also make assumptions about their child's internal state, which assumes a shared socially communicative representation.

It is also important to consider how those with ASD discuss their pain with others. It has been suggested that children with ASD can use body diagrams to indicate the location of pain; however, they may struggle in using intensity scales for pain (neurotypical children often also find these difficult). Although there are vast differences regarding the language that children may be able to use to discuss pain (some using words like "excruciating" or "twisting"), they most generally prefer to discuss "hurt" rather than "pain." It was also notable that children generally preferred not to spend time discussing their pain. Children with ASD may also prefer to use parents or carers as a "gatekeeper" to discuss their pain. Thus, studies are needed to incorporate differences in pain communication across the lifespan for people with ASD.

Observational Studies

A major approach to understanding the pain responses of people with ASD, and in particular those who are either unable to communicate verbally or have limited verbal communication, has been to observe behaviors in the context of potentially painful medical procedures. These have been conducted with both recognized/standardized schedules and more general measures. These studies still tend to measure only socially communicative displays of "pain," yet they benefit from an impartial observer and discrete episode of standardized pain.

Studies have generally focused on observations of venepuncture procedures; however, the measures of pain taken during these procedures have varied. Studies have shown that children with ASD show greater facial activity (Childhood Facial Action Coding System) during the needle insertion phase of the procedure, as well as extended facial and behavioral reactions following the procedure. Others have found

reduced behavioral reactivity, however elevated heart rate and plasma β -endorphin levels and increased self-injurious behaviors following the procedure.

These data reporting increased reactivity to pain are in contrast to hyporeactive diagnostic criteria and self-, parent, and clinician reports in individuals with ASD. Yet these studies cannot separate specific pain behaviors from a distress reaction due to an unfamiliar environment, with unfamiliar people and experiencing an unfamiliar sensation.

A further challenge of this approach and these studies is that independent of ASD, individuals with intellectual delay might have reduced social communication of emotions and in particular pain (Gilbert-MacLeod et al. 2000). Given that the populations in these studies were unable to report on their experiences during the procedure, it is unclear what the precise cause of their behaviors might have been.

Psychophysics

A wide range of psychophysical approaches have been used to quantify the response of individuals with ASD to potentially painful stimuli. Most studies have been broadly based on the German Neuropathic Pain Network (DFNS). These studies investigate the graded psychophysical response to stimuli, allowing quantification of clinically relevant perception and pain thresholds. Standardized protocols are essential in order to minimize variability and produce reliable and comparable results, as well as clinical feasibility in its application.

Early studies exploring this question generally focused on single modalities for pain assessment. These showed no differences between ASD adults and controls for electrocutaneous pain; however, ASD adults were more sensitive to pressure pain. Further, there have been conflicting findings about thermal pain thresholds with studies showing either no difference or greater sensitivity in ASD. More recently, two studies have utilized comprehensive psychophysical batteries. In each case, no clear evidence was found for altered pain perception in ASD (Vaughan et al. In press).

Neuropsychological Evidence

Examining neural responses to pain in autism provides evidence of altered neural processing of pain and a communication-independent assessment of pain responses. The *neural pain signature* (NPS, somatosensory regions, insula, anterior cingulate) (Wager et al. 2013) reliably responds in a linear fashion to perceived intensity of a pain stimulus. However, in adults with ASD, NPS responses are initially intact but dramatically decrease *prior* to the end of the painful stimulus, even while participants continue to report pain (Failla et al. 2018). In other studies, there is evidence that people with ASD have heightened neural responses during pain anticipation (Gu et al. 2018).

Neural responses to viewing pain in others (vicarious pain) overlap heavily with the NPS and may provide insight into pain processing and differences in communication about pain evident in autism. Vicarious pain is also important for understanding social learning about pain expression norms. Results from neuroimaging studies of vicarious pain in autism are mixed, with evidence of intact (Hadjikhani et al. 2014) and decreased neural responses (Krach et al. 2015).

Chronic Pain

Although research on co-occurring conditions in autism has received substantial interest in recent years (see the entry ► “Comorbidity” for review), comparatively little of this work has specifically explored co-occurrence with pain conditions and experiences. A recent service review has suggested that although autism has a population prevalence of approximately 1%, as much as 13.7% of patients in a pediatric chronic pain service might meet criteria for autism. Therefore, children and adolescents with ASD may be more susceptible to developing chronic pain complaints than their neurotypical peers (Lipsker et al. 2018). Interestingly, this increase in chronic pain presentation in ASD may be particularly prevalent in females with ASD; however, more data is needed to confirm these effects.

Importantly, individuals with ASD also experience a number of co-occurring conditions that

are inherently painful or could negatively impact pain (i.e., mood disorders (Failla et al. 2019) or sleep disorders (Richdale and Schreck 2009). In particular, gastrointestinal distress is commonly observed in children with ASD; estimates have suggested that approximately 25% of children with ASD may experience significant and persistent gastrointestinal distress at any time, and about half of these report abdominal pain as an important feature of this experience.

Potential Evidence from Animal Models

Studies in animal models suggest potential biological alterations in pain responsivity related to specific genetic vulnerabilities in ASD. Mouse models of ASD exhibit altered pain behaviors and/or neurobiology (i.e., *SHANK3*; Han et al. 2016), parvalbumin knockout (Wöhr et al. 2015). In this context, it is unclear if aberrant pain behaviors represent downstream consequences of primary sensory processing differences specific to ASD or nociceptive-specific functional differences. Interestingly, mice lacking pain-related genes (i.e., mu-opioid receptor) also exhibit ASD-like behaviors (Becker et al. 2014). Future work is needed to understand this complex overlap in ASD and pain biology.

Future Considerations

At this stage, it is important to appreciate the heterogeneity in this population. There is considerable need for a larger, more comprehensive study of pain behaviors and consequences in people with autism. It is also important to note that the laboratory approaches used above are based on a rather artificial context in which to experience pain. These studies rely on participants actively attending to a pain stimulus within a sterile medical or laboratory context with no clear motivational, emotional, social, or cognitive state. This is not how people typically experience pain in their environment where pain might be competing for attention with daily tasks/interests and associated with emotional states and concerns over health and recovery. It is therefore important that we

consider issues surrounding pain in daily context and how this might be experienced.

Understanding of the role of other psychiatric or emotional contributions to pain in ASD is needed. Presently, research has established that the magnitude of pain reactivity in ASD during injection and dental procedures is predicted by depression and anxiety (Garcia-Villamizar et al. 2019). This effect mirrors work in neurotypical populations and suggests that people with greater anxiety and depression experience greater pain and also suggests that factors related to pain in ASD may be similar to the general population. The role of anxiety in pain experience in ASD is an important issue, as anxiety has been shown to be highly prevalent in ASD (see the entries ► “Anxiety”, ► “Anxiety Disorders”, and ► “Generalized Anxiety Disorder” for more information about anxiety).

Further data also suggests that reduced thermal pain sensitivity may relate to IQ, suggesting that those with lower IQ scores also report higher pain thresholds (Duerden et al. 2014). It is however important to note here that the methods used to determine pain thresholds in this study are based on a reaction time-sensitive measure, and therefore slower motor or general processing speed may also result in greater reported thresholds (Williams et al. 2019). Intriguingly, these are also suggestions that greater reductions in pain behavior may be associated with more severe ASD symptoms.

One of the most frequently discussed issues related to pain processing in ASD is that of self-injurious behaviors (for more information about self-injurious behaviors, see the entry ► “Self-Injurious Behavior (SIB)”). A prevalent early opinion was that self-injurious behaviors were a sign of reduced pain sensitivity. Opinions have however changed in recent years, with observations that individuals with ASD may engage in self-injurious behaviors following potentially painful episodes suggesting that this might be a coping mechanisms to deal with pain. The role of self-injurious behaviors in pain experience and expression is however still unclear in ASD.

Understanding pain perception, experience, and expression in ASD is of critical importance. The presence of pain-related comorbidities, uncertainty regarding pain responsivity, and

communication difficulties that limit the reliability of standard pain assessments **makes the problem of pain management in ASD difficult and urgent**. Additionally, there are reported case studies showing that traditional analgesic approaches may not be as effective in some patients with ASD and that prescribing practices may differ in ASD. Yet, there are many other non-pharmacological options for pain management in neurotypical populations. Future work is needed to identify best pain assessment and management strategies in ASD.

See Also

- Anxiety
- Anxiety Disorders
- Comorbidity
- Generalized Anxiety Disorder
- Interoception
- Self-Injurious Behavior (SIB)
- Sensory Features as a Marker of Autism Spectrum Disorders
- Sensory Processing
- Short Sensory Profile in Autism

References and Reading

- Bandstra, N. F., Johnson, S. A., Filliter, J. H., & Chambers, C. T. (2012). Self-reported and parent-reported pain for common painful events in high-functioning children and adolescents with autism spectrum disorder. *The Clinical Journal of Pain*, 28(8), 715–721. <https://doi.org/10.1097/AJP.0b013e318243ecf6>.
- Becker, J. A., Clesse, D., Spiegelhalter, C., Schwab, Y., Le Merrer, J., & Kieffer, B. L. (2014). Autistic-like syndrome in mu opioid receptor null mice is relieved by facilitated mGluR4 activity. *Neuropsychopharmacology*, 39, 2049.
- Bursch, B., Ingman, K., Vitti, L., Hyman, P., & Zeltzer, L. K. (2004). Chronic pain in individuals with previously undiagnosed autistic spectrum disorders. *The Journal of Pain*, 5(5), 290–295. <https://doi.org/10.1016/j.jpain.2004.04.004>.
- Duerden, E. G., Card, D., Roberts, S. W., Mak-Fan, K. M., Chakravarty, M. M., Lerch, J. P., & Taylor, M. J. (2014). Self-injurious behaviours are associated with alterations in the somatosensory system in children with autism spectrum disorder. *Brain Structure and Function*, 219(4), 1251–1261.

- Failla, M. D., Moana-Filho, E. J., Essick, G. K., Baranek, G. T., Rogers, B. P., & Cascio, C. J. (2018). Initially intact neural responses to pain in autism are diminished during sustained pain. *Autism*, 22(6), 669–683.
- Failla, M. D., Schwartz, K. L., Chaganti, S., Cutting, L. E., Landman, B. A., & Cascio, C. J. (2019). Characterizing co-occurring conditions by age at diagnosis in autism spectrum disorders. medRxiv:19002527.
- Garcia-Villamizar, D., Moore, D., & Garcia-Martínez, M. (2019). Internalizing symptoms mediate the relation between acute pain and autism in adults. *Journal of Autism and Developmental Disorders*, 49(1), 270–278.
- Gilbert-MacLeod, C. A., Craig, K. D., Rocha, E. M., & Mathias, M. D. (2000). Everyday pain responses in children with and without developmental delays. *Journal of Pediatric Psychology*, 25(5), 301–308. <https://doi.org/10.1093/jpepsy/25.5.301>.
- Gillberg, C., Terenius, L., & Lönnerholm, G. (1985). Endorphin activity in childhood psychosis: Spinal fluid levels in 24 cases. *Archives of General Psychiatry*, 42(8), 780–783. <https://doi.org/10.1001/archpsyc.1985.01790310042005>.
- Gu, X., Zhou, T. J., Anagnostou, E., Soorya, L., Kolevzon, A., Hof, P. R., & Fan, J. (2018). Heightened brain response to pain anticipation in high-functioning adults with autism spectrum disorder. *European Journal of Neuroscience*, 47, 592–601.
- Hadjikhani, N., Zürcher, N., Rogier, O., Hippolyte, L., Lemonnier, E., Ruest, T., . . . Billstedt, E. (2014). Emotional contagion for pain is intact in autism spectrum disorders. *Translational Psychiatry*, 4(1), e343.
- Han, Q., Kim, Y. H., Wang, X., Liu, D., Zhang, Z.-J., Bey, A. L., Lay, M., Chang, W., Berta, T., Zhang, Y., Jiang, Y.-H., & Ji, R.-R. (2016). SHANK3 deficiency impairs heat hyperalgesia and TRPV1 signaling in primary sensory neurons. *Neuron*, 92, 1279–1293.
- Krach, S., Kamp-Becker, I., Einhäuser, W., Sommer, J., Fräse, S., Jansen, A., Rademacher, L., Müller-Pinzler, L., Gazzola, V., & Paulus, F. M. (2015). Evidence from pupillometry and fMRI indicates reduced neural response during vicarious social pain but not physical pain in autism. *Human Brain Mapping*, 36, 4730–4744.
- Lipsker, C. W., Bölte, S., Hirvikoski, T., Lekander, M., Holmström, L., & Wicksell, R. K. (2018). Prevalence of autism traits and attention-deficit hyperactivity disorder symptoms in a clinical sample of children and adolescents with chronic pain. *Journal of Pain Research*, 11, 2827.
- Minshew, N., & Hobson, J. (2008). Sensory sensitivities and performance on sensory perceptual tasks in high-functioning individuals with autism. *Journal of Autism and Developmental Disorders*, 38(8), 1485–1498. <https://doi.org/10.1007/s10803-007-0528-4>.
- Richdale, A. L., & Schreck, K. A. (2009). Sleep problems in autism spectrum disorders: prevalence, nature and possible biopsychosocial aetiologies. *Sleep Medicine Reviews*, 13(6), 403–411. <https://doi.org/10.1016/j.smrv.2009.02.003>.
- Vaughan, S., McGlone, F., Poole, H., & Moore, D. J. (In press). A quantitative sensory testing approach to pain in autism spectrum disorders. *Journal of Autism and Developmental Disorders*.
- Wager, T. D., Atlas, L. Y., Lindquist, M. A., Roy, M., Woo, C.-W., & Kross, E. (2013). An fMRI-based neurologic signature of physical pain. *New England Journal of Medicine*, 368(15), 1388–1397.
- Williams, Z. J., Failla, M. D., Davis, S. L., Heflin, B. H., Okitondo, C. D., Moore, D. J., & Cascio, C. J. (2019). Thermal perceptual thresholds are typical in autism spectrum disorder but strongly related to intra-individual response variability. *Scientific Reports*, 9(1), 1–14.
- Wing, L. (1976). Diagnosis, clinical description and prognosis. In L. Wing (Ed.), *Early childhood autism* (2nd ed.). Oxford: Pergamon Press.
- Wöhr, M., Orduz, D., Gregory, P., Moreno, H., Khan, U., Vörckel, K. J., Wolfer, D. P., Welzl, H., Gall, D., Schiffmann, S. N., & Schwaller, B. (2015). Lack of parvalbumin in mice leads to behavioral deficits relevant to all human autism core symptoms and related neural morphofunctional abnormalities. *Translational Psychiatry*, 5, e525.

Paired-Choice Preference Assessment

► Preference and Reinforcer Assessment

Pakistan and Autism

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Introduction

Pakistan is situated in South Asia, and with a population of about 210 million, it is the sixth most populous country in the world. Almost

35% of this population is under the age of 14 years. As is true for other Asian countries, the child and adolescent psychiatry services in Pakistan, including the services for autism and other developmental disabilities, are significantly lagging in comparison to its Western peers. There are few general psychiatry departments in major cities of Karachi, Lahore, Rawalpindi, Islamabad, Faisalabad, Multan, Peshawar, and Quetta; however, because of limited resources, their outreach services to rural areas are almost nonexistent, and it is in rural areas where nearly two-thirds of the country's population resides. The situation of divisions of child and adolescent psychiatry is bleaker as there are only a handful of child psychiatrists and very few training sites in the country. At present, there is only one inpatient child and adolescent psychiatry unit in Pakistan, which was established in 2012 at King Edward Medical University/Mayo Hospital in Lahore (Imran and Azeem 2014).

Current Status

Autism spectrum disorder is an evolving concept (Nazeer et al. 2019), and the current prevalence of autism spectrum disorder (ASD) in Pakistan is unknown. Because of the lack of integrated services, various health professionals, including child psychiatrists, general psychiatrists, neurologists, psychologists, pediatricians, and speech and language therapists, find themselves to be the first-line providers for this population and to make the diagnosis of autism. Because of the lack of any formal training for the above wide array of health care providers, no standardized screening tools for ASD are used in most practices. Along with these professionals, there are also strong cultural beliefs in getting treatment from traditional healers, who also sees a bulk of children with autism and other developmental disabilities. Current literature has reveals a low level of awareness and knowledge of ASD among healthcare workers and school teachers (Imran et al. 2011; Rahbar et al. 2011; Farooq et al. 2018). Various misbeliefs and misconceptions were found in these surveys regarding causes, diagnosis, and treatment interventions for autism.

In the above landscape, there are few glimmers of light as there are very few schools that are promoting the cause of adequate mental health for children and are providing specialized programs for this population. One of the earliest established schools in this regard is "Amin Maktab School" in Lahore, the second-largest city of Pakistan. Amin Maktab School was established in 1962, and by 1991, this institution was successful in building outreach programs for underserved areas of Lahore.

With the increased use of social media, increased literacy rate, and parental advocacy, different institutions are taking a more proactive role in serving this population. Children's Hospital, Lahore is one such example, which, as a tertiary care pediatric hospital, has developed a well-established program for autism and provides diagnostic and therapeutic services. Dow University in Karachi has also started an autism program in 2013. Sindh Provincial Government has also taken the lead in establishing the Center for Autism Rehabilitation and Training-Sindh (C-ARTS) in 2018. This center provides a wide array of comprehensive services for children with autism spectrum disorder in the largest city of Pakistan.

There are limited subspecialty training programs in the country, and at present, only one private teaching institute, Agha Khan University, Karachi, Sindh, is providing subspecialty training in child and adolescent (CAP) psychiatry in the country. To further the status of subspecialty training in this field, the College of Physicians and Surgeons Pakistan (CPSP) has recently approved a CAP fellowship of their own, which is based on the United States model of 2 years subspecialty training after finishing postgraduation in general psychiatry. Various individuals have played a key part in developing this fellowship from Pakistan, United Kingdom, and the USA including Prof. Mowadat Rana, Prof. Fareed Minhas, Dr. Nazish Imran, Dr. Ayesha Mian, Dr. Ayesha Minhas, Dr. Ayesha Sarwat, Prof. Atif Rehman, Dr. Dorothy Stubbe, and Prof. Muhammad Waqar Azeem. Once started, this fellowship will fill the much-needed gap in the training of child psychiatrists with adequate knowledge of

autism spectrum disorder and developmental disabilities.

In recent years, various parental groups have also played an important part in educating and supporting the families of children with autism spectrum disorder. The most well-known group in this regard is “Pakistan Autism Meet up,” which was established by one of the parents, Ms. Saira Salman, in 2003. This support group currently has hundreds of members across the country and around the world, including parents and health care professionals. This group was started in the city of Karachi and now has branches in Lahore, Rawalpindi/Islamabad, and Quetta, as well as an active online forum. Ms. Irum Rizwan and another volunteer, Mr. Qazi Fazli Azeem, along with several parents and volunteers, have played a considerable part in the ongoing success of this group as well as leading the advocacy efforts around the country. This group meets regularly and is instrumental in the support and services for ASD in Pakistan. Various nongovernmental organizations (NGOs) like Autism Spectrum Disorders Welfare Trust (a project of Mahvish & Jahangir Siddiqui Foundation with Ramaq Center for Awareness and Social Responsibility) are working to improve awareness regarding ASD all over Pakistan.

Future Directions

Unavailability of mental health law is an area in which Pakistan has not made much progress. At present, there are no federal laws related to services and education for individuals with autism and developmental disabilities. Sindh Empowerment of “Persons with Disabilities” Act, 2018, is perhaps the only such law at a provincial level, which specifically mention Autism as well as other neurodevelopmental disorders.

As noted above, there is much need for the establishment of a basic framework and integrated services for this population in need. This is why it is important for the public and private sector to join hands in providing comprehensive diagnostic, treatment, educational, and support services for these children and families in most need across the country. Authors believe that if we come together, the future is bright.

References and Reading

- Farooq, Z., Imran, N., Warraich, S., Baluch, A. S., & Hussain, R. (2018). Awareness and knowledge about autism spectrum disorder among mainstream and special school teachers in Lahore. *Pakistan Pediatric Journal*, 42(4), 247–254.
- Imran, N., & Azeem, M. W. (2014). Child psychiatry in Pakistan. *AACAP News*, 45(1), 5–6.
- Imran, N., Chaudry, M. R., Azeem, M. W., Bhatti, M. R., Choudhary, Z. I., & Cheema, M. A. (2011). A survey of autism knowledge and attitudes among the healthcare professionals in Lahore, Pakistan. *BMC Pediatrics*, 11, 107.
- Nazeer, A., Hashemi, N., Imran, N., & Azeem, M. W. (2019). Autism spectrum disorder: A concept in evolution. *Psychiatric Annals*, 49, 103–108.
- Rahbar, M. H., Ibrahim, K., & Assassi, P. (2011). Knowledge and attitude of general practitioners regarding autism in Karachi, Pakistan. *Journal of Autism and Developmental Disorders*, 41(4), 465–474.

Paleostriatum

► [Globus Pallidus](#)

Palestine and Autism

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Historical Background

Since the end of the British mandate over Palestine in 1948 and the creation of Israel, the region has witnessed continuing territorial disputes, protracted violence, and political instability between the Arab Palestinians and the Israeli Jews. After the Oslo Accord signed in Washington, DC, in 1993, the Palestinian Authority was installed in the West Bank and Gaza Strip in a yet complicated peace process envisaging a two-state solution. Today, Palestine is a nonsovereign state that has been accepted as the 195th Member State by the United Nations Educational, Scientific

and Cultural Organization (UNESCO) in 2011 (UNESCO 2011). Palestine still holds a non-member observer state in the United Nations. The West Bank of Palestine is a landlocked area surrounded by Israel and bordered to the East by the Hashemite Kingdom of Jordan, while the Gaza Strip is 25 miles long and 4–5 miles wide land bounded by Israel and Egypt. The majority of the West Bank is controlled by Israel and a blockade has been in place since long limiting entry to and exit of Palestinians from the Gaza Strip. There are nearly 3 million Palestinians living in the West Bank and about 1.85 million living in the Gaza Strip.

Attention to autism spectrum disorders (ASDs) in Palestine has increased recently. This could be attributed to the increasing exposure to mass media, in part, and to the disseminated research carried out in the region and elsewhere around the world (Ashbee 2016; Dababnah and Bulson 2015; Dababnah and Parish 2013). This might have led to increasing the awareness of both healthcare professionals as well as laypersons on the issue of ASDs (Basha 2014). Tracking down the history of ASDs in this particular region of the world would be a difficult task in the absence of disseminated case reports. In addition, the incidence and prevalence of ASDs in Palestine are still unknown due to the absence of large epidemiological studies carried out for these objectives. Furthermore, Palestinian refugees in neighboring countries were excluded from studies conducted to determine the prevalence of ASDs, notably the one conducted in Lebanon although the number of Palestinian refugees in Lebanon registered by the United Nations Relief and Works Agency for Palestine Refugees in 2010 was approximately 440,000 (Chaaya et al. 2016). Recently, there have been calls to prioritize research needs in mental health in Gaza Strip and ASDs were among these priorities (Abu-El-Noor and Aljeesh 2014).

Probably, light was shed on ASDs when the Palestinian Bureau of Statistics has conducted its first specialized national disability survey in 2011 which included ASDs within the category of “learning difficulties” when the Palestinian Central Bureau of Statistics has conducted its first specialized national disability survey in 2011

which included ASDs within the category of “learning difficulties” (PCBS 2011). Prior to this disability survey, the Child Statistics survey made no mention of ASDs. However, next year (2012), the Child Statistics survey alluded to ASDs which were included within “learning disability/difficulty” (Ashbee 2016; PCBS 2012). Another light shedding event was the organization of the first conference related to ASDs which was held in April 2012 in Bethlehem to coincide with the World Autism Day (Ashbee 2016). The conference was well attended and policies were discussed. In April 2016, the An-Najah Child Institute of An-Najah National University took a next step and held another conference on “Autism Spectrum Disorder, Evidence Based Practices in Palestine.”

In Palestine, there are four main providers of healthcare to the Palestinians. These providers are: (a) the Ministry of Health (the public sector), (b) the private sector, (c) nongovernmental organizations (NGOs), and (d) refugee services operated by the United Nations Relief and Works Agency (UNRWA). NGOs provide many services including psychosocial support and rehabilitation for physical as well as mental disabilities. Utilization of the services provided by the NGOs accounts for 11% of the overall healthcare utilization in the West Bank (World Bank 2006). In spite of the reported dearth of services for children with ASDs in Palestine, some not-for-profit and NGOs provide services to individuals with intellectual disability and ASDs.

An-Najah National University, which is the largest university in Palestine, has established An-Najah Child Institute in 2013. This institute provides clinical, training, and research services related to ASDs. A comprehensive and multidisciplinary assessment approach is applied for the diagnosis of ASDs at this institute using internationally accepted tools which were translated into Arabic, adapted to culture, tested, and formally validated in Palestine. Currently, there is a kindergarten catering to the needs of children with ASDs. Once admitted, children are assessed, monitored, and individualized educational and skills acquiring plans are made for every child. Children with ASDs are grouped in small groups, each group of about five children and children

with high functioning ASDs are later included in classes with the mainstream educational programs along with other children without ASDs. The institute has many research initiatives and plans to use evidence-based practice in catering to the needs of children with ASDs.

The Jerusalem Princess Basma Centre for Disabled Children was established in 1965 initially as a home for children with physical disabilities. Later the center was opened to include both children with disabilities and those without disabilities. Today, the center offers services related to physical therapy, occupational therapy, speech therapy, and special education. Since 2011, the center offers services to children with ASDs (Jerusalem Princess Basma Centre for Disabled Children 2017). The center is unique in the Palestinian context. The staff at the center are well-resourced and trained in approaches for working with children with ASDs. The staff includes teachers and occupational and speech therapists. Unfortunately, Palestinians in the West Bank face difficulties accessing and utilizing the services offered at this center as it is located in East Jerusalem. Palestinians need to obtain a travel permit (a *laissez-passé*) from the Israeli authorities to enter Jerusalem. For Palestinians of the Gaza Strip, it is nearly impossible to utilize the services of the center as they are restricted to travel to Jerusalem by Israel (Dababnah and Bulson 2015).

Other centers catering to the needs of children with ASDs in Palestine include the Star Mountain Center which was started as a hospital and now offers services and cater to the needs of children with low-functioning ASDs and intellectual disabilities (Star Mountain 2017). Parents of children with ASDs established a community-based program known as Friends of Autistic Children Society (Friends of Autistic Children Society 2017). This center was established particularly for children with ASDs. Families of children with ASDs are closely involved. No formal training was provided to the staff; instead, they turned to the internet to seek guidance (Ashbee 2016).

Although the number of projects and programs that cater to the special educational needs of individuals with physical, intellectual, and learning

disabilities ran by not-for-profit organizations and NGOs has grown, many parents questioned the quality of the services provided and claimed the costs were unaffordable to a considerable percentage of those interviewed (Dababnah and Bulson 2015; Dababnah and Parish 2013; Nasir-Tucktuck et al. 2017). Rigorous data on the quality of screening, diagnosis, therapy, and other services available and accessible to individuals with ASDs in Palestine are still needed (Dababnah and Bulson 2015). In best practice, diagnosis of ASDs requires a comprehensive assessment which is carried out with the help of a multidisciplinary team using formally validated tools. Recent studies reported healthcare professionals lacking awareness and knowledge of ASDs (Dababnah and Bulson 2015; Dababnah and Parish 2013; Shawahna et al. 2017). In many cases, parents of children with ASDs reported noticing signs and symptoms of ASDs on their children before pediatricians. Parents also reported that pediatricians did not use any formal screening tools. Parents of children suspected of having ASDs struggle finding healthcare professionals trained to perform the diagnosis. Receiving inaccurate diagnosis and information on ASDs and being recommended to use treatments without a solid evidence for effectiveness have also been reported (Dababnah and Bulson 2015).

Traveling outside the country is a common practice by Palestinians seeking diagnosis and/or treatments of disorders including ASDs (Dababnah and Bulson 2015; Habash and Fteiha 2015). Obtaining a referral from the Palestinian Ministry of Health and a travel permit (a *laissez-passé*) from the Israeli authorities is often required for Palestinians to be allowed to visit Israeli hospitals and healthcare centers. Palestinians also often seek diagnosis and/or treatment in Jordan. Al Jabery and colleagues reported services including physical, occupational, and speech therapies available for individuals with ASDs in Jordan (Jabery et al. 2014). They also reported treatment services using hyperbaric oxygen and special nutritional plans. Extensive barriers to utilizing these services were reported, most notably, the cost. Geographic and political barriers also exist. Parents also reported challenges related to

checkpoints and transportation limiting their access and utilization of services related to ASDs. Beyond cost and geographic and political barriers, individuals with ASDs and their families often face a heavy burden of social and cultural barriers to access and utilize services pertaining to ASDs (Dababnah and Bulson 2015).

Legal Issues, Mandates for Service

The passage, in 1998, of the Right of Education for All law by the Palestinian Authorities led to reforms in teacher training programs and educational facilities to accommodate the different needs of students, including those with disabilities (Palestinian Ministry of Education 2000). Prior to this law, schools did not cater to the needs of school children with physical, intellectual, and/or learning disabilities (Nasir-Tucktuck et al. 2017). Following the passage of this law, progress has been witnessed in regards to changing the attitudes and perception of providing equitable education to school children with disabilities. In the last two decades, many grant-funded teacher training projects have been administered in the Palestinian territories and many new schools accepted students with learning disabilities and mild disabilities. Despite these efforts of inclusion, unfortunately many children with disabilities in Palestine skip education. According the Palestinian Central Bureau of Statistics, more than 1 out of 3 people with disabilities aged 15 and above had never been enrolled in any educational system and out of those who did enroll, nearly 1 out of 5 dropped out as a result of their disabilities (Ashbee 2016; PCBS 2011).

Financial assistance to individuals with ASDs and their families is limited. In Palestine, any person with disability is eligible to apply for support from the Ministry of Social Affairs. Eligibility for financial support is decided following an extensive scrutiny. Support consists of an amount equivalent to about \$215 every 3 months. This amount is hardly enough to cover the cost of diagnosis of ASDs. Financial restrains related to caring for children with ASDs in regard to treatment, vitamins, tests, diapers, costly specialty

items, speech therapy were reported by parents (Dababnah and Bulson 2015).

Overview of Current Treatments and Centers

Despite the dearth of services offered to individuals with ASDs in Palestine, the number of centers offering services to individuals with ASDs has grown recently. Some centers are striving to apply evidence-based approaches. Currently, the national mental health thematic group is developing the national policy for the diagnosis and treatment of ASDs. The policy was recently drafted and the formal approval is underway. Initiatives by parents and community-based programs have started and probably would be growing in the near future. Ambitious plans have been envisaged to create a village for individuals with ASDs (Ashbee 2016). However, these plans are beyond realization in the near future due to lack of funds and governmental support. Probably, this has come after the growing awareness of the importance of exposure to peers and acquiring of social skills.

Overview of Research Directions

Unfortunately, research programs are underdeveloped. Recently, some qualitative studies reported access to ASDs-related services as well as parents' perspectives of raising children with ASDs (Dababnah and Bulson 2015; Dababnah and Parish 2013). Another study investigated the awareness and knowledge of pharmacists on ASDs (Shawahna et al. 2017). A study investigated a possible link between iron deficiency and ASDs (Al-Ali et al. 2015). Another study sequenced whole exome and revealed complex inheritance patterns and identified two gene mutations implicated in ASDs and intellectual disability (Khalaf-Nazzal et al. 2016).

Large clinical trials are completely absent in Palestine as laws prohibited conduction of such trials. Recently, a law was passed which permits the conduction of clinical trials in Palestine. The passage of this law would probably open

new avenues for therapeutic interventions and research. Obviously, there is a dearth of interventional research in Palestine.

Overview of Training

The importance of training teachers to cater to the needs of children with ASDs has recently been recognized (Ashbee 2016). The Palestinian Ministry of Education has promised more commitment to develop in the field of education for children with ASDs. Resource rooms have been established in some schools to support children with additional needs. Some schools like the Friends school in Ramallah are including pupils with ASDs in the mainstream environment. The Jerusalem Princess Basma Centre for Disabled Children offers training to professionals in the West Bank in a trial to share experience and transfer know-how to their peers. More efforts are needed to accelerate the formal approval of the national policy for the diagnosis and treatment of ASDs and setting healthcare agenda in relation to ASDs.

Social Policies and Current Controversies

In a survey conducted in the West Bank by the Palestinian Central Bureau of Statistics, 1.6% of children aged 0–17 years were reported to have either physical or intellectual disability (PCBS 2011). It is important to mention that disabilities in the region are underreported and therefore, these figures should be interpreted with caution. Families with individuals with ASDs were reported to hide their afflicted siblings (Ashbee 2016).

Unfortunately, there is stigma in regards to individuals with disabilities in general. Social stigma and discrimination were reported by parents of children with ASDs (Dababnah and Bulson 2015; Dababnah and Parish 2013). Denial, depressive symptoms, poor coping, social isolation, and increased burden on siblings were also reported by parents of children with ASDs (Dababnah and Bulson 2015; Dababnah and Parish 2013). Parents

were able to cope with these challenges by religious practices, social support, and access to information. Fortunately, there is a growing body of information freely available on the internet that parents can access and learn about ASDs.

In Arabic speaking regions, words to denote disability are reported to be derogatory and pejorative. They are also used to swear. It is not uncommon for families to hide their children with disabilities fearing that the community will reject them.

A report by the Palestinian Central Bureau of Statistics on children and disability found that there is a tendency to blame the mother for her child's perceived disability (PCBS 2012). ASDs were often blamed as a "punishment from god." Recently surveyed pharmacist lacked adequate awareness and knowledge of and believed in myths in regard to ASDs (Shawahna et al. 2017).

Interestingly, today there is a louder parent voices demanding better governmental services for their children with ASDs. Increasing awareness and education might have an impact on reducing stigma and dispelling myths about individuals with ASDs.

See Also

- ▶ [DSM-III](#)
- ▶ [DSM-III-R](#)
- ▶ [ICD 10 Research Diagnostic Guidelines](#)

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References and Reading

- Abu-El-Noor, N. I., & Aljeesh, Y. I. (2014). Identifying and prioritizing the research needs related to mental health in Gaza Strip, Palestine. *Open Journal of Psychiatry*, 5(01), 19.
- Al-Ali, S. F., Russo, S., & Alkaissi, A. (2015). Association between autism spectrum disorder and iron deficiency in children diagnosed autism spectrum disorder in the northern West Bank. *Journal of Health, Medicine and Nursing*, 16, 1–10.

- Ashbee, E. (2016). *Educational inclusion for children with autism in Palestine: what opportunities can be found to develop inclusive educational practice and provision for children with autism in Palestine: with special reference to the developing practice in two educational settings*. Doctoral thesis, University of Birmingham, UK. <http://ethos.bl.uk/OrderDetails.do?uin=uk.bl.ethos.687454>. Accessed 12 January 2017.
- Basha, S. (2014). First national public opinion survey: Palestinians knowledge and understanding of autism, 2014. *Italian Journal of Special Education for Inclusion*, 2(1), 87–96.
- Chaaya, M., Saab, D., Maalouf, F. T., & Boustany, R. M. (2016). Prevalence of autism spectrum disorder in nurseries in Lebanon: A cross sectional study. *Journal of Autism and Developmental Disorders*, 46(2), 514–522. <https://doi.org/10.1007/s10803-015-2590-7>.
- Dababnah, S., & Bulson, K. (2015). “On the sidelines”: Access to autism-related services in the West Bank. *Journal of Autism and Developmental Disorders*, 45(12), 4124–4134. <https://doi.org/10.1007/s10803-015-2538-y>.
- Dababnah, S., & Parish, S. L. (2013). “At a moment, you could collapse”: Raising children with autism in the West Bank. *Children and Youth Services Review*, 35(10), 1670–1678.
- Friends of Autistic Children Society. (2017). <http://www.mhpss.ps/en/organization/friends-of-autistic-children-society/ttEG.NPeDa0=>. Accessed 18 January 2017.
- Habash, M., & Fteiha, M. (2015). *Study of beliefs of parents of children with autism regarding complementary and traditional medicine in 4 Middle-East countries*. Poster session at the International Meeting of Autism Research, Salt Lake City, UT.
- Jabery, M. A. A., Arabiat, D. H., Khamra, H. A. A., Betawi, I. A., & Jabbar, S. K. A. (2014). Parental perceptions of services provided for children with autism in Jordan. *Journal of Child and Family Studies*, 23(3), 475–486.
- Jerusalem Princess Basma Centre for Disabled Children. (2017). <http://www.basma-centre.org/>. Accessed 20 February 2017.
- Khalaf-Nazzal, R., Dweikat, I., & Maqboul, A. (2016). *Whole exome sequencing reveals complex inheritance patterns and identifies two gene mutations implicated in the development of Autism and Intellectual Disability in a consanguineous Palestinian family*. Poster session at the Educational Neuroscience, Abu Dhabi, United Arab Emirates.
- Star Mountain. (2017). <http://www.starmountain.org/>. Accessed 10 February 2017.
- Nasir-Tucktuck, M., Baker, J. N., & Love, M. L. (2017). Educating learners with disabilities in Palestine: The past, present, and future. *Intervention in School and Clinic*, 52(3), 182–187.
- Palestinian Ministry of Education. (2000). *Special report*. Palestine Ministry of Education: Ramallah. <http://www.pna.net/reports/edu>. Accessed 03 January 2017.
- PCBS. (2011). *Disability survey*. Ramallah, Palestine: Palestinian Central Bureau of Statistics and Ministry of Social Affairs. http://www.pcbs.gov.ps/portals/_pcbs/pressrelease/disability_e2011.pdf. Accessed 13 February 2017.
- PCBS. (2012). *Palestine children – issues and statistics*. Ramallah, Palestine: Palestinian Central Bureau of Statistics. http://www.pcbs.gov.ps/Portals/_PCBS/Downloads/book1863.pdf. Accessed 14 February 2017.
- Shawahna, R., Fahed, B., Qadri, D., Sharawi, L., Soroghli, M., & Dweik, M. (2017). Awareness and knowledge of autism spectrum disorders among pharmacists: A cross-sectional study in Palestinian pharmacy practice. *Journal of Autism and Developmental Disorders*, 47(6), 1618–1627.
- UNESCO. (2011). United Nations Educational, Scientific and Cultural Organization. <http://www.un.org/apps/news/story.asp?NewsID=40253#.WLV1xfmGOUk>. Accessed 20 February 2017.
- World Bank. (2006). *The role and performance of Palestinian NGOs in health, education and agriculture*. Washington, DC: World Bank. <http://unispal.un.org/pdfs/NGOreportDec06.pdf>. Accessed 14 January 2017.

Palliative Care

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Definition: Palliative Care for Adults with Autism Spectrum Disorders

The oldest cohort of adults with autism spectrum disorders (ASD) is aging, but little has been done to prepare for their end-of-life needs. A review of the literature does not reveal a substantial body of work on end-of-life care for adults with ASD. The purpose of this entry is to introduce caregivers and general clinicians who care for adults with autism to some of the unique challenges of palliative care in this population. It is also intended to familiarize palliative care specialists with some of the unique features of ASD.

There is tremendous variability in an autism spectrum that encompasses nearly every degree of functioning, but some issues in palliative care will be applicable to broad subpopulations of those with ASD. Obstacles related to patient autonomy will include communication challenges, decisional incapacity, and surrogate decision-making. First, some subgroups with ASD will express preferences and clinical guidance in atypical ways, and these communication mismatches can create challenges trying to manage pain and suffering in ways concordant with patients' wishes. Second, fear of dying, pain, and worry about an uncertain future are discomfiting in palliative care, generally, but may be exacerbated by features of autism that can lend themselves to a patients' isolation, confusion, or incomplete comprehension of what is going on around them. Broader issues will arise including the intertwined questions of decision-making capacity and surrogate decision-making. Some elderly adults with ASD will have full capacity to communicate long-term care plans, sign advance directives, and designate potential surrogates, while others will be able to do so only in a limited or attenuated manner. In these cases, surrogates will play integral roles in decision-making by augmenting and extending patients' self-determination; they will frequently be family members who have provided care for many years and now face new tasks that include shouldering the burden of surrogate decision-making at the end of life.

These challenges, and many further unfamiliar ones, will arise when elderly adults with autism enter palliative care settings. When these challenges do occur, strong psychosocial support and collaboration between caregivers, patients, and families will increase quality of life by ascertaining the preferences and goals of care of adults with ASD, thereby enhancing autonomy.

Historical Background

Palliative Care

Palliative care aims to improve quality of life for patients with serious or terminal illness and focuses on family-centered care designed to relieve suffering and increase comfort. Palliative

care originated in the modern hospice movement pioneered in 1967 by Dame Cicely Saunders (Fins 2006) and is initiated when "cure is no longer the prevailing goal of care" (Fins 2006). Comprehensive strategies for palliative care cover multiple domains of care, including physical, emotional, social, intellectual, and cultural needs. These domains can be addressed by interdisciplinary care teams that provide multidimensional management of symptoms; social, spiritual, cultural, and psychological support as ways of providing comfort and relief; and clear channels of communication between providers and families in order to provide support, acknowledge sensitive legal and regulatory issues, and resolve complex ethical challenges (The National Consensus Project on Quality Palliative Care 2013).

The domains of palliative care can be weighted differently. Some strategies of care "focus more comprehensively on the technical aspects of end-of-life care, such as drug dosing or the management of common symptomology" (Fins 2006). Other models emphasize the humanistic approach: a person-centered, shared-understanding model in which patients and physicians collaborate to understand disease trajectory and articulate goals about quality of life, intentions of care, and resolution of end-of-life issues (Fins 2006; Friedman et al. 2010). Humanistic models of palliative care aim to increase quality of life while keeping patient autonomy in the foreground.

As palliative care involves numerous decisions by patients and families, palliative care organizations seek to enhance access to information and choice. Short-term decisions include treatment preferences such as specifics of comfort care and pain management, location of care (e.g., hospital, hospice, or a patient's home), and which family members are wanted close at hand. Longer-term decisions dictate preferences that reflect the overarching values of the patient and how they want those values to guide or shape critical moments at the end of life. These include passive approaches to care, such as withdrawal of artificial nutrition and signing "Do Not Resuscitate" (DNR) forms, and active strategies including aggressive interventional treatment. Ideally, these decisions develop through ongoing conversations among patients, families, and providers, in which values

are articulated and expectations refined. After these conversations are completed, important procedures codify preferences into legally binding terms and documents, such as advance care plans that designate proxy decision-makers and treatment preferences in the event of decisional incapacity.

Adults with Autism Spectrum Disorders

Autism is a persistent developmental disorder with impairments in two broad categories: social communication and social interaction. Often diagnosed early in life, individuals typically exhibit restricted, repetitive patterns of behavior, interests, or activities (American Psychiatric Association 2013).

First identified in 1943 by pediatric psychiatrist Leo Kanner (1968), public awareness of autism increased during the 1950s and 1960s through now-discredited theories about “refrigerator mothers” as a cause, and diagnostic recognition expanded further with formal codification of autism in the 1980 publication of the DSM-III. Given this timeline, the oldest adults to receive a formal diagnosis of autism are in their late 50s, 60s, and early 70s. In many cases they have lived most of their lives with family or in supervised group homes.

Recent focus has been on early detection and early intervention in autism. Presently, children are diagnosed on average several years earlier than their counterparts 60 years ago, which allows for a greater amount of early intensive behavioral therapy during critical developmental periods (Howlin et al. 2014). However, this focus on early intervention has led to inadvertent neglect of adult and lifespan perspectives of older individuals with ASD: “We know next to nothing about people aging with ASD. There are...no data on the quality of life of the elderly with ASD” (Fombonne 2012). Emphasis on childhood autism has left gaps in care and services for adults with ASD.

Elderly adults with autism will face challenges that are extensions of childhood difficulties, as well as obstacles that are novel to old age. First, the core features of classical childhood autism extend into and take on new forms during adulthood. Children with autism who have modest or

higher IQ (≥ 70) in childhood remain relatively cognitively stable into adulthood (Seltzer et al. 2004). Some adults with autism are able to improve in their social and verbal skills and attain more stable social roles throughout adulthood, but this has been proposed to occur in a “splinter” fashion, with progress in discrete skills rather than improvement across the board (Seltzer and Shattuck 2003). Second, social interaction and communication deficits tend to lead to difficulties later in life in employment, independent living, and relationships and trouble with law enforcement for adolescents and adults with ASD (Volkmar et al. 2014), many of whom present with disadvantaged physical or mental health and overall quality of life as a result (Howlin and Moss 2012). As a result, 60–75% of individuals with autism have “poor” or “very poor” outcomes as adults (Seltzer et al. 2004; Howlin et al. 2014), but these tendencies vary based on the degree of early impairment, family support, and access to services. Trends in outcomes are likely more acute, i.e., a downward shift in function, in older generations of adults with ASD who have missed key therapeutic interventions that are now standard practice.

As diagnostic recognition has improved over time, the number of adults formally diagnosed with autism is expected to grow progressively larger. However, the prevalence of formal diagnoses likely underestimates the true rate of autism. The first comprehensive study to include undiagnosed adults found a relatively static rate of autism over time in the UK: 1.1% of 16- to 44-year-olds, 0.9% of 45- to 74-year-olds, and 0.8% of those 75-year-olds, roughly the same prevalence as the childhood population (Brugha et al. 2011). In many cases undiagnosed individuals with ASD were socially disadvantaged and unrecognized and did not access services.

Palliative Care Plans and Challenges (Current Knowledge)

The first group of adults with a formal diagnosis of autism is nearing the age when they might receive end-of-life palliative care. Features of autism vary in presentation among individuals,

so it is imperative to distinguish between individuals who are capable of articulating their vision for care procedures, those who will need support to do so, and those who might be cognitively incapable of doing so altogether. Providers should respond differently to these groups, tailoring their care, presence, and interactions to enhance self-determination and the quality of life of adults with ASD receiving palliative care.

As palliative care is a collaborative endeavor, providers can draw on various resources. The adult patient – the focus of care – has wishes, needs, and goals of care to be articulated. Family members and caregivers can be consulted as doing so can enhance the patient's communicative capacity and ability to make informed choices. There are also legal and ethical perspectives to take into account. It is in the spirit of palliative care to collaboratively address obstacles through open communication that involves all relevant parties in processes of decision-making and end-of-life choice.

Palliative care providers may face many challenges when caring for elderly adults with ASD, including:

Communication Differences

Communication is a prerequisite for choice and preference setting in palliative contexts. However, some adults with autism are unable to clearly articulate preferences and overarching values, which is an obstacle for palliative care personnel seeking to provide care in accordance with patients' wishes.

There is wide variation in verbal acumen of adults with ASD, reflecting the full breadth of the autism spectrum. By adulthood, many of those with partially or fully nonverbal autism will have identified noncanonical strategies for augmenting dialectical communication. Helpful strategies may include augmentative and alternative communication technologies (AAT) (Beukelman and Mirenda 2013), sign languages, physical or behavioral communication, facilitated communication, and visual communication such as PECS (Picture Exchange Communication System) (Dawson and Burner 2011). These mediums are used at different rates: 40% of people with ASD

use sign language, 26% use a high-tech assistive device (e.g., voice output system), 40% use a low-tech assistive device (e.g., Picture Exchange Communication System), and 95% reportedly use gestures (many individuals with ASD use more than one mode of assistive communication) (Gotham et al. 2015). These strategies may be adapted for a palliative care context; for example, social stories and books are used to communicate about the deaths of other people (The National Autistic Society: Death, Bereavement, and Autism Spectrum Disorders 2015), often older family members, but have not yet been extended to palliative care. Alternative communication techniques will play a crucial role in avoiding communication mismatch between palliative care providers and patients with ASD.

Understanding wishes conveyed directly by a patient is key, but in some cases adults with ASD may rely on caregivers who can assist in conveying a message. These intermediaries will often be able to aid in or facilitate the patient's own communication. Familiarity with alternative communication can help palliative care providers navigate complex clinical dynamics and ascertain the consent, choices, and preferences of elderly adults with autism, regardless of their verbal status.

Sensory Integration and Experience of Pain

Atypical sensory processing is a common feature of autism. It involves perception of tactile, auditory, visual, or painful stimuli as more or less noxious than their true intensity. Hyposensitivity to pain is troublesome in general medical contexts due to underreporting of illness, but hypersensitivity to pain is particularly worrisome as a primary goal of palliative care is the relief of terminal pain. Atypical processing of pain can severely affect quality of life and precipitate anxiety or agitation. In cases of pain hypersensitivity, there may be a need to adjust pharmacotherapy accordingly.

However, in cases of pain hypersensitivity, some adults with ASD will express desired strategies for pain management, while others will have difficulty with effective communication of internal experience. In the latter case, providers can

rely on nonverbal signs of pain and distress. These may include unexplained rises in heart rate and blood pressure or pupillary dilation, all signs of sympathetic overdrive. There are also behavioral observation techniques for assessing pain, such as the Critical Care Pain Observational Tool (Pun and Dunn 2007), which measures behaviors including muscle rigidity, body movements, and facial expressions, and the Adult Nonverbal Pain Scale (Rothman 2006), which can be used to assess pain and anxiety in critically ill, nonverbal adults by observing signs such as “stimming” or signs of overstimulation that can include grimacing, moaning, shaking, and hunched walking (Dell 2008). In short, effective terminal pain management for adults with ASD may necessitate awareness of nonverbal and physiological cues, nondialectical communication of discomfort, and potentially heightened importance placed on pain relief.

By communicating, patients can engage in the clinical decision-making process; however, communication alone is insufficient for robust choice. In some cases of ASD, beyond questions of communication and patient engagement, there will be additional concerns about the durability of the decisional process.

Decisional Capacity and Choice

Decisional capacity is the ability one has to make decisions for oneself. Insofar as decision-making relies on functional abilities that can be pathologically affected by certain disorders, in many cases it will be important to evaluate whether a patient has robust decisional capacity. Modern capacity evaluations focus on functional abilities-based criteria for capacity, typically the abilities to understand information relevant to a decision, appreciate the information (i.e., apply it to one’s own situation), reason with the information, and communicate a stable choice (Appelbaum and Grisso 1988). Other models of capacity assessment focus on whether the patient’s decision has been made in light of personal beliefs and values (Fins 2006).

Several features of decisional capacity are important to note. First, the threshold for capacity can vary depending on the decision at hand. The

“sliding scale” model of capacity articulates a threshold that varies depending on the consequences of a decision (Drane 1984). Thus, patients may be able to make some decisions but lack capacity for other, riskier ones. For example, a patient’s decision to sign a “Do Not Resuscitate” order should be subject to a stringent evaluation and a high capacity bar, but that same patients’ preference for some hospital foods over others needs not be questioned in the same way. A second, related point is that capacity “is not a global determination of cognitive abilities” (Fins 2006), rather, it is assessed for each *specific* decision in question. Thus, incapacity to make one highly complex medical decision should not be equated with incapacity for all decisions.

Some adults with ASD will meet functional capacity criteria to make weighty medical decisions, while others will not. Particularly those with Asperger’s, high IQ, or higher-functioning autism will have capacity to make medical decisions for themselves (Volkmar et al. 2005). Others with lower-functioning autism will lack decisional capacity, due in large part to comorbidity between autism and intellectual disability that occurs 17–70% of the time (Matson and Shoemaker 2009). However, even in cases of adults with autism who have severe intellectual disability such as mental retardation (MR), it will be important to evaluate patients and decisions on a case-by-case basis. “Most adults with mild and no mental retardation, and almost half of adults with moderate MR are able to make and justify treatment choices and fully or partially understand treatment information” (Cea and Fisher 2003). Additionally, 50% of individuals with mild MR, and 18 % with moderate MR, are able to “partially appreciate relevance of treatment choice to [their] situation and weigh treatment risks against benefit” (Cea and Fisher 2003).

Communicating with patients about their choices makes end-of-life care better. In palliative care settings, adults with autism should be presented with a full range of decisions including pain management, location of palliative care, and questions of comfort. As there is vast heterogeneity in ASD, no categorical state of capacity or incapacity will be found in elderly adults with

ASD in making palliative care decisions. Some adults with ASD will meet the threshold to set preferences about comfort care, but not to make a broader decision to withdraw life-sustaining nutrition. One method of ascertaining a choice if capacity is limited is to present possible options sequentially, such as possible locations of care, then allowing the individual to choose based on experience. When adults with autism have limited decisional capacity, communication between providers, the patient, family members, and surrogates will determine which elements of a care plan are best decided by the individual patient and which aspects should be decided by surrogates.

Adults with autism face obstacles to self-determination during palliative care. Many will have their decisions questioned or refused due to provider belief that the patient lacks capacity (Institute of Medicine 2006). A recent study of a Dutch residential facility retrospectively examined the files of 47 individuals with ID who had died during a determined span of time. Twenty-seven of these individuals had an end-of-life decision made about their care, and families were involved in only half of those decisions (Wagemans et al. 2010). There was also “no evidence in the notes that any of the people with IDs was asked for his or her own opinion in taking an end-of-life decision.” Palliative care providers, caretakers, and family members must ensure that the preferences of adults with autism are taken into account. Another challenge is the possibility of decisional conflict, which can arise among the patient, providers, and surrogates, when a patient with known limited capacity articulates a choice, but it is unclear whether they have capacity to make this particular decision. Finally, there may be disagreement among various proxies, each with legitimate claims to surrogacy. If this occurs, providers should seek out the legal surrogate. If none exists, a clinical ethics consult may be requested.

Surrogate Decision-Making

Surrogates and healthcare proxies may assume decisional responsibility for a patient who has been determined to have limited capacity or who cannot make independent judgments (Fins 2006). The goal of shared decision-making among

surrogates and palliative care teams is to reach decisions balancing the patients’ interests, medical needs, and goals of care. Decisions that surrogates may be called on to make include cessation of treatment and other difficult, emotionally demanding decisions. Since surrogate decision-making is not a simple task, guidelines (Fins 2006) aid proxy decision-makers in deciding on behalf of others: first, surrogates should make decisions in line with the “expressed wishes” of the patient, i.e., what the patient said they would want if they were ever in this scenario. If no wishes have been shared on the subject, it is appropriate to use a “substituted judgment” standard, i.e., how the patient “would have decided” if they were able to do so, which a proxy can determine in light of a patient’s preferences and values. Finally, in the “absence of information” on the patient’s desires, a decision can be made that is in the patients’ “best interest” (Fins 2006). Accurate decision-making on behalf of patients is a mechanism that helps patients extend their self-determination and autonomy in medical contexts.

Surrogate decision-makers for adults with autism will face challenges during palliative care. First, many adults with autism never express choices about palliative care, while others might do so but with suspect capacity. In these cases, a substituted judgment standard is ideal. Second, parents who have served as long-term caregivers are often best suited for surrogacy, but may no longer be living. In some of these cases, there will be court-appointed guardians who may be unfamiliar with the patient. Such guardians should consider that, under a “best interests” standard, the relative weighting of values and interests is unique for some adults with ASD. Even in cases of a long-standing surrogate who knows the patient well, deciding for others is a difficult, complex task, and others may have useful input. Family members, caretakers, guardians, group home staff, physicians, therapists, friends, and teachers may have extensive knowledge of the patient’s preferences and can assist in the decision-making process. Finally, when an adult with autism enters palliative care, surrogates may want to discuss potential issues with the care team and draw up plans in advance, in order to facilitate

decision-making during distressing, acute scenarios (King et al. 2004).

There are also legal hurdles in surrogate decision-making. Conflicting laws, practices, and principles can make it difficult to determine who should be making medical decisions (King et al. 2004). Family members and guardians of adults who have autism should always choose durable and valid healthcare proxies and codify these choices in proper documentation that is easily available.

Avoiding Futile Care

Not all medical care is good – there are harms of futile care. Medical futility can harm patients by causing unnecessary suffering with no possibility of direct benefit, injure the emotional state of a family, exhaust hospital resources, and put physicians in the uncomfortable position of having to provide care they know is ineffective (Fins 2006). Ideally, planning for futility questions takes place before futility questions actually arise and involves conversations between families, palliative care teams, and the patient.

Futile care decisions involving adults with autism raise at least two major considerations. One concern is that in the case of adults with autism, palliative care providers might worry about stigma of deciding that care is futile for vulnerable individuals, which can therefore lead to overtreatment and greater suffering. A second concern is that there may be an implicit presumption that adults with ASD and cognitive disabilities cannot participate at all in medical decisions, which would rob them of a voice. Obtaining assent and respecting dissent are both important when a patient has less-than-robust capacity. In particular, if pain sensitivity is a known concern for an adult with autism, assent and dissent may be highly informative as to treatment preferences and futile care. However, relying heavily on proxies to make decisions involving assent, dissent, and medical futility can place a severe burden on surrogates. Balancing these two considerations will require palliative care providers, families, and patients with ASD to draw up in advance ways of determining the point at which care might be futile for an individual patient.

Unique Palliative Care Comorbidities

Many unique challenges can arise during palliative care for adults with autism. First, several conditions frequently comorbid with adult autism, including anxiety (Bradley et al. 2004), depression (Ghaziuddin et al. 2002), ADHD, and epilepsy (Bailey et al. 1998), can create auxiliary complications. Some adults with autism rely on sameness and can be resistant to change, engendering anxiety as hospice is an unfamiliar setting, and as novelties such as an array of unfamiliar symptoms, tests, pains, and confusion are associated with many end-of-life scenarios. Anxiety can be mitigated in many ways, such as encouraging adults with autism to participate in the decision-making process regarding location of palliative care. There are also pharmacologic and non-pharmacologic techniques, including distraction, favorite rituals or practices, medication, art, and relaxation, that can help reduce anxiety and physical or emotional distress. Second, there is a risk that natural, somatic diseases go unreported among adults with nonverbal autism who are receiving palliative care. Communication deficits can make medical care more difficult or lead to diagnostic delay of various treatable diseases (Sea 2008). Adults with ASD may have increased or decreased internal sensitivity in addition to sensitivity to pain. These factors can make it difficult to assess and treat the secondary health status of adults with autism, especially those who are receiving palliative care focused on a primary condition.

Finally, adults with autism – particularly severe or nonverbal ASD – face a heightened risk that their own condition or care will be talked about in their presence by care providers, with the assumption that they do not understand the ongoing discussion. This would negatively affect the quality of life at its end, diminish the overall quality of care received by the patient, and violate principles of confidentiality and respect for persons.

Bereavement

Bereavement can come in many forms. Most elderly adults with autism will have experience with some form of death and may feel anticipatory grief, before death. For an adult with autism,

anticipatory grief may manifest in typical or atypical ways depending on the level of understanding of death and emotional engagement. There might be preconceived notions on the part of the patient of what a good death can or should look like. Palliative care providers should discuss and dissect these expectations, especially if they are inappropriately anticipated. Furthermore, many adults with autism live in group homes. If palliative care takes place in a group home, there is the possibility of grief or bereavement affecting other members of the group home, particularly those who observe the progression of care. Finally, in all cases, but perhaps most acutely those in which surrogates make decisions about futile care, providers should expect surrogate bereavement.

Future Directions

As the oldest adults with autism spectrum diagnoses begin to reach the natural end of life and receive palliative care, these adults, their families and surrogates, and healthcare providers will face novel challenges that require flexibility in care and communication. Future research should focus on understanding these challenges, including barriers to decisional capacity, how patients' capacity can be augmented to increase autonomy, and which issues most frequently arise during surrogate decision-making. What preferences do elderly adults with ASD express about the end of life, how are those preferences conveyed, and, most importantly, are they followed? Furthermore, how are the decisions surrogates face in palliative care substantively, emotionally, and psychologically different than the medical decisions they make for adults with autism earlier in life? Creative research programs are currently lacking and might conceivably be championed by national advocacy programs.

While palliative care has evolved profoundly as a field, further preparation will help care for adults with ASD at the end of their lives. Broad ethical issues can arise in palliative care including respecting preferences ascertained through consent, assent, or dissent so as to avoid under- or overaggressive treatment. Parents of children and adolescents with autism commonly express worry about an uncertain

future, particularly when they are no longer able to act as their child's surrogates. It is critical that parents or guardians, and adults with autism, take the time to have conversations during which values, preferences, and choices about the end of life are ascertained and formally codified to ensure legal authority. This will allow the palliative medical practice to bring its expertise in providing comfort to bear in a way that respects and honors the autonomy, capacity, and choices of an often-vulnerable population (Fins and Pohl 2015).

References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5*. 5th ed. Arlington: American Psychiatric Association. xlv, 947 p. p.
- Appelbaum, P. S., & Grisso, T. (1988). Assessing patients' capacities to consent to treatment. *The New England Journal of Medicine*, 319, 1635–1638.
- Bailey, A., Luthert, P., Dean, A., Harding, B., Janota, I., Montgomery, M., Rutter, M., & Lantos, P. (1998). A clinicopathological study of autism. *Brain*, 121, 889–905.
- Beukelman, D., & Mirenda, P. (2013). *Augmentative and alternative communication: Supporting children and adults with complex communication needs* (4th ed.). Baltimore: Brookes Publishing.
- Bradley, E. A., et al. (2004). Comparing rates of psychiatric and behavior disorders in adolescents and young adults with severe intellectual disability with and without autism. *Journal of Autism and Developmental Disorders*, 34(2), 151–161.
- Brugha, T., et al. (2011). Epidemiology of autism spectrum disorders in adults in the community in England. *Archives of General Psychiatry*, 68, 459–466.
- Cea, C. D., & Fisher, C. B. (2003). Health care decision-making by adults with mental retardation. *Mental Retardation*, 41(2), 78–87.
- Clinical Practice Guidelines for Quality Palliative Care*, Pittsburgh: National Consensus Project for Quality Palliative Care Task Force led by Ferrell, B., Meier, D. The National Consensus Project represents the American Academy of Hospice and Palliative Medicine, Center to Advance Palliative Care, Hospice and Palliative Nurses Association, National Association of Social Workers, National Hospice and Palliative Care Organization, National Palliative Care Research Center.
- Dawson, G. B., & Burner, K. (2011). Behavioral interventions in children and adolescents with autism spectrum disorder: A review of recent findings. *Current Opinion in Pediatrics*, 23, 616–620.
- Dell, D. (2008). Care of patients with autism spectrum disorder undergoing surgery for cancer. *Clinical Challenges*, 35(2), 177–182.

- Drane, J. (1984). Competency to give informed consent: A model for making clinical assessments. *JAMA*, 257(7), 925–927.
- Du Boulay, S. (1984). 268 p., 8 p. of plates p *Cicely Saunders, founder of the modern Hospice Movement*. London: Hodder and Stoughton.
- Fins, J. (2006). *A palliative ethic of care: Clinical wisdom at life's end*. Sudbury: Jones and Bartlett. xxxiv, 281 p. p.
- Fins, J., & Pohl, B. (2015). Neuro-palliative care and disorders of consciousness. Chapter 5.3. In N. Cherny, M. Fallon, S. Kaasa, R. Portenoy, & Currow D. (Eds.), *Oxford textbook of palliative medicine* (5th ed.). Oxford, UK: Oxford University Press.
- Fombonne, E. (2012). Autism in adult life. *The Canadian Journal of Psychiatry*, 57(5), 273–274.
- Friedman, S.L., & Helm, D.T., American Association on Intellectual and Developmental Disabilities. (2010). *End-of-life care for children and adults with intellectual and developmental disabilities*. Washington, DC: American Association on Intellectual and Developmental Disabilities. . xix, 369 p. p.
- Ghaziuddin, M., et al. (2002). Depression in persons with autism: Implications for research and clinical care. *Journal of Autism and Developmental Disorders*, 32(4), 299–306.
- Gotham, K., et al. (2015). Characterizing the daily life, needs, and priorities of adults with autism spectrum disorder from Interactive Autism Network data. *Autism*, 19, 794–804.
- Howlin, P., & Moss, P. (2012). Adults with autism spectrum disorders. *Canadian Journal of Psychiatry*, 57(5), 275–283.
- Howlin, P., Savage, S., Moss, P., Tempier, A., & Rutter, M. (2014). Cognitive and language skills in adults with autism: A 40-year follow-up. *Journal of Child Psychology and Psychiatry*, 55, 49–58.
- Institute of Medicine. (2006). Committee on crossing the quality chasm: Adaptation to mental health and addictive disorders.
- Kanner, L. (1968). Autistic disturbances of affective contact. *Acta Paedopsychiatrica*, 35(4), 100–136.
- King, E.A., et al.. (2004). End of life care for people with developmental disabilities: Philosophy and recommendations. The Last Passages Project.
- Matson, J. L., & Shoemaker, M. (2009). Intellectual disability and its relationship to autism spectrum disorders. *Research in Developmental Disabilities*, 30(6), 1107–1114.
- Pun, B. T., & Dunn, J. (2007). The sedation of critically ill adults part 1: Assessment. *The American Journal of Nursing*, 107(8), 40–48.
- Rothman, A. (2006). Manage the power of pain. *Men in Nursing*, 1(4), 20–28.
- Sea, M. (2008). Mortality and causes of death in autism spectrum disorders. *Autism*, 12(4), 403–414.
- Seltzer, M. M. K. M., & Shattuck, P. T. (2003). The symptoms of autism spectrum disorders in adolescence and adulthood. *Journal of Autism and Developmental Disorders*, 33, 565–581.
- Seltzer, M. M. S. P., Abbeduto, L., & Greenberg, J. S. (2004). Trajectory of development in adolescents and adults with autism. *Mental Retardation and Developmental Disabilities Research Reviews*, 10, 234–247.
- The National Autistic Society: Death, Bereavement, and Autism Spectrum Disorders. [Internet]. Cited Retrieved 13 July 2015.
- Volkmar, F. R., Klin, A., & Schultz, R. T. (2005). Pervasive developmental disorders. In B. J. Sadock & V. A. Sadock (Eds.), *Comprehensive textbook of psychiatry* (Vol. 2, 8th ed., pp. 3164–3175). Philadelphia: Lippincott Williams and Wilkins.
- Volkmar, F. R., et al. (Eds.). (2014). *Adolescents and adults with autism spectrum disorders*. New York: Springer Science + Business Media.
- Wagemans, A., Van Schrojenstein Lantman-de-Valk, H., Tuffrey-Wijne, I., Widdershoven, G., & Curfs, L. (2010). End-of-life decisions: An important theme in the care for people with intellectual disabilities. *Journal of Intellectual Disability Research*, 54, 516–524.

Palpebronasal Fold

► Epicanthic Fold

Para

- Para-educator
- Paraprofessional

Paraeducator

► Paraprofessional

Para-educator

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Synonyms

Aide; Classroom aide; Educational assistant;
Instructional assistant; One-to-one; Para;
Paraprofessional; Teacher's aide; Teaching assistant;
Tutor

Definition

Para-educators are school employees who work directly under the supervision of licensed professionals and who deliver instructional and direct services to students and their families. The licensed professionals, however, maintain responsibility for assessing learner and family needs and for planning, evaluating, and modifying programs. Pickett (1996) was the first to introduce the title para-educator to convey a level of training analogous to paramedic in the medical field and paralegal in the legal field. Historically, para-educators provided assistance with tasks ranging from clerical to individualized living tasks. In the present, they have become a key part of the specialized education team in delivering individualized instruction and services. Para-educators who work with children with autism spectrum disorders (ASD) and other developmental disabilities have responsibilities that may include assisting students with instruction; social, emotional, and behavioral skill development; and personal care. Additionally, they may also engage in behavioral data collection and collaboration with other educators (Rossetti and Goessling 2010). National Research Council (2001) lists para-educators as one of the potential resources for providing special services for children with autism.

See Also

► [Paraprofessional](#)

References and Reading

- Carroll, D. (2001). Considering paraeducator training, roles, and responsibilities. *Teaching Exceptional Children*, 34(2), 60.
- Council for Exceptional Children. (2004). *Parability: The CEC paraeducator standards workbook*. Arlington: Author. ISBN 0-86586-410-1.
- Cullen, D. (2000). *How to be a para pro: A comprehensive training manual for paraprofessionals*. Higganum: Starfish Specialty Press.
- Lord, C., & McGee, J. P. (Eds.). (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- National Research Council. (2001). *Educating Children with Autism*. Washington, DC: The National Academies Press.
- National Resource Center for Paraprofessionals in Education and Related Services. (1996). *A state of the art report on paraeducators in education and related services*. New York: Pickett, A. L.
- Pickett, A. L. (1996). *A State of the Art Report on Paraeducators in Education and Related Services*. New York: City University of New York, National Center for Paraprofessionals in Education and Related Services, Center for Study in Advanced Education.
- Rossetti, Z. S., & Goessling, D. P. (2010). Paraeducators' roles in facilitating friendships between secondary students with and without autism spectrum disorders or developmental disabilities. *Teaching Exceptional Children*, 42(6), 64–70.

Paralinguistic Communication Assessment

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Definition

“Paralinguistic communication” has been defined as “not WHAT you say, but THE WAY you say it.” Paralanguage, sometimes known as nonverbal communication, is communication by means other than words, although (usually) operating alongside language. Paralanguage is often impaired in people with autism spectrum disorder, to a lesser or, more often, greater degree than their language. Here, prosody (also known as intonation) and voice, which operate in conjunction with speech, are considered. More peripheral aspects of paralanguage would include eye-gaze, body language, and gesture: all often impaired in ASD.

“Prosody” is an umbrella term for modulations of the speech stream in terms of its loudness, length, and pitch. These modulations manifest themselves as the relative duration of syllables (determining speech-rhythm and speech-rate), the placing and quantity of stress in words, and changes of pitch (which produce pitch-patterns or contours). These affect the linguistic impact of utterances, conversational interaction, and the

verbal punctuation of speech; they also give indications of the speaker's identity (accent) and attitude to what is said. "Intonation" is another umbrella term for these modulations, which can also be described as "suprasegmental": "segmental" refers to the makeup of words in terms of consonants and vowels, and "suprasegmental" to levels which span individual sounds/letters, that is, generalizations that apply to syllables, whole words, phrases, and discourse.

"Voice" here denotes vocal quality, and the way this can affect the impact of the spoken word; for example, vocal huskiness suggests the imminence of tears and profound sadness in the speaker. Atypical vocal quality has often been noted in people with ASD.

"Assessment" refers to methods of gauging ability and the severity of disability. Assessment protocols range from procedures devised on an ad hoc basis for research projects to formal tests. Formal tests assess the degree, usually described in numerical terms, to which a speaker may be impaired on a particular aspect of speech. Such tests may be standardized, that is, provide data from large numbers of unimpaired speakers with which the performance of individual speakers can be compared.

Historical Background

Although the importance of prosody in speech has long been recognized, it was not until the 1980s that ways of assessing prosodic ability began to develop. Some protocols were devised as parts of investigations into speech impairment in conditions such as dysarthria or aphasia. These procedures were not comprehensive as tests of prosodic ability: They often assessed only a few aspects of prosody, for example, whether stress was placed correctly. Moreover, they focused on prosody in speech-production without assessing how well prosody was understood. They usually required transcription: A disadvantage, in that systems for transcribing prosody were (and are still) unsettled, and few people are familiar with them.

David Crystal designed a series of procedures for measuring disability in different aspects of

speech and language, one of which concerned prosody: This was known as "*Prosody Profile*" or "PROP" (Crystal 1982). The PROP sampled spontaneous speech which was prosodically transcribed and scored according to how correctly the prosody was deemed to reflect the speaker's intentions as to phrasing and conversational interaction. Since then, the PVSP (q.v.) has been devised, on lines similar to PROP in that it involves the sampling and transcription of spontaneous speech.

For the assessment of vocal quality, Laver devised the Vocal Profile Analysis (VPA) in the 1980s. This is a technique for assessing laryngeal, phonatory, and articulatory settings. Similarly, the GRBAS (Grade, Roughness, Breathiness, Asthenia, Strain) scale is used for evaluating hoarse voice quality.

There has been an awareness of prosodic and vocal impairment in autism since the earliest descriptions of the condition; in 1943, Leo Kanner, writing about children with autism mentions "a monotonous singsong manner" (Case 7) and a voice "peculiarly unmodulated, hoarse; utters her words in an abrupt manner" (Case 11). He also notes that in the repetitions (echolalia) of what was said to the children he examined "even the intonation is retained." Hans Asperger, writing in 1944, similarly observed odd prosody in the children with ASD that he examined.

The work of Christiane Baltaxe in the 1980s was pioneering in that it focused particularly on the assessment of expressive prosody in autism. See "References and Readings," McCann and Peppé 2003, for a review of articles addressing the issue and a description of assessment methods used. No publication has been made of the findings of the VPA and the GRBAS with speakers with ASD.

Current Knowledge

There are essentially four tests for assessing prosody and voice: one that assesses both, one devoted to prosody, and two to voice. There are also several other ad hoc procedures that have been used in research papers. Automated assessment is likely to increase: This will involve the collection of large amounts of speech-data which can be

scored in increasingly sophisticated ways by computer programs and will thus focus on prosody in speech-production as opposed to understanding of prosody.

Prosody and Voice

The *Prosody-Voice Screening Profile (PVSP)* assesses five suprasegmental speech factors (phrasing, rate, stress, pitch, and loudness) as well as two aspects of voice quality (laryngeality and resonance) in spontaneous speech for their appropriateness.

A major study used this test to compare the utterances of young people with ASD and typical development, and found inappropriate resonance and stress placement in the speakers with ASD (see “References and Readings”).

Prosody

The *Profiling Elements of Prosody in Speech-Communication (PEPS-C)* test assesses receptive and expressive ability in four functional aspects of prosody as well as auditory discrimination and imitation of prosody.

Several studies (see “References and Readings”) have used this test to research prosody in speakers with ASD, and have found impairment in some aspects, for example, the ability to place stress correctly, the use of prosody for conveying affect, and imitation of prosody.

Voice

Voice quality is assessed by making judgments about vocal sound, using adjectives such as “hoarse” and “husky” to describe it. Little has been done in the way of assessment of vocal quality in autism, and the standard diagnostic reference for ASD does not include vocal characteristics as a marker of ASD. As mentioned above, hoarse vocal quality was noted by Kanner in his first evaluation of autism. Vocal quality is generally taken as a good indicator of a speaker’s emotional and physical state, and hoarseness normally suggests tiredness or ill-health; this could be misleading if the hoarseness is part of the autistic condition. It is, however, true that listeners rapidly learn to discount the information value of habitual voice quality.

The Vocal Profile Analysis assesses laryngeal, phonatory, and articulatory settings, but no

publication has been made of its use with speakers with ASD.

The GRBAS (Grade, Roughness, Breathiness, Asthenia, Strain) scale is used for evaluating hoarse voice quality, but has apparently not been used for this in people with ASD. This scale was developed in Japan in the 1980s, but has no published standardized English protocol.

The Consensus Auditory-Perceptual Evaluation of Voice (CAPE-V) has recently been developed under the auspices of the American Speech-Language-Hearing Association (ASHA) and compares well with the GRBAS for reliability.

Automated Assessment

Programs have been developed to analyze speech automatically in terms of syllable-length, loudness, and pitch-variation. The advantage of automated assessment is that it can process very large samples, for example, of continuous day-long recordings. A disadvantage is that the programs have some way to go before they can accurately assess appropriateness for language use: Whether the variations of length, loudness, and pitch are typical of the language or accent being spoken, or adequately convey stress, phrasing, questions, etc.

Oller and associates (see “References and Readings”) have used such automated procedures with no human intervention; comparisons show that human judgment and automated assessment agreed well. They found that vocalizations of typically developing children and those of children with ASD were distinguished by spectral differences related especially to syllabification and by features of voice quality.

Van Santen and colleagues are also working on automated assessment. They have found that children with ASD show a difference in the balance between the various prosodic cues, such as pitch, amplitude, and duration, and not necessarily a difference in the strength or clarity with which prosodic contrasts are expressed.

Future Directions

Recent research suggests that certain prosodic functions are generally impaired in autism

spectrum conditions, for example, stress placement and the expression of emotion, and the understanding of prosody. It also seems that the degree varies more or less according to the severity of the autism condition. There is, however, scope for further research, for example in people with ASD who speak *languages other than English*.

A generalization in objective terms about the *acoustic characteristics of autistic prosody* has however proved elusive, although many people who work with autism would find it highly recognizable.

The period of time-consuming manual data collection and transcription for research purposes appears to be reaching its end, although this is still important for clinical investigation of individuals. In research, the manual process is being overtaken by *automation*.

The ability to understand prosody is central to language development and it seems that *prosodic processing* in the brains of those with autism may be different from non-autistic processing. Magnetic resonance imaging is being used to investigate this.

Assessment of Prosody in Other Languages

The PEPS-C test has been translated into French, Spanish, Dutch, and Norwegian, as described in Peppé et al. (2010). It is also available in various accents of English: UK General, UK Scottish, Irish, North American General, and Australian General. Requests for translated versions have been received from researchers working in Finnish, Portuguese, Farsi, Chinese, and Egyptian Arabic, and translation into more languages is a possibility.

Acoustic Characteristics of Prosody in ASD

The ability to generalize about the acoustic characteristics of prosody in ASD is still at an early stage. Diehl, Paul and colleagues found that their group of children with ASD had overall longer

utterance durations than children with learning difficulties and typical development, and that children with high-functioning autism show greater pitch variation in their speech (see “References and Readings” for details of this work). With increasing scope for acoustic analysis such generalization may be extended.

Automation

Having written nearly 20 years ago, Shriberg and colleagues regret that “completely objective measurement of all relevant parameters for prosody-voice assessment is currently not a technical option . . . use of inappropriate lexical, emphatic, and sentential stress, inappropriate intonation in pragmatic contexts, breathiness, nasality, denasality, and other parameters cannot be accomplished by current voice or speech recognition programs.” Assessment of prosody by automated acoustic analysis is increasingly available as technology develops, as indicated by the work of Oller and colleagues (see “References and Readings”). This focuses on the acoustic characteristics rather than the functions of voice and prosody: Their findings that atypical vocal quality and syllabification in children with ASD can be determined with automated analysis can be expected to contribute to early diagnosis and screening for ASD.

Prosodic Processing

There is increasing interest in using magnetic resonance imaging to establish whether there are differences in prosodic processing in the brains of people with and without ASD. Some differences have been found: for example, that in people without ASD the processing of speech sounds occurs in a different part of the brain than the processing of non-speech sounds, but that this is not the case in people with ASD; and that patterns of brain activation suggested a decrease of activation for emotional stimuli in people with ASD. For more recent research in this area, see the work of the Yale Center for Research in Autism and of Isabelle Hesling in Bordeaux, France.

See Also

- PEPS-C
- Prosody
- PVSP

References and Reading

- Asperger, H. (1944). Autistic psychopathy in childhood (U. Frith, Trans., & Annotated). In Frith, U. (Ed.), *Autism and Asperger syndrome* (pp. 37–92). Cambridge, UK: Cambridge University Press. (Original work published 1991).
- Crystal, D. (1982). *Profiling linguistic disability*. London: Edward Arnold.
- Dejonckere, P. H., Obbens, C., de Moor, G. M., & Wieneke, G. H. (1993). Perceptual evaluation of dysphonia: Reliability and relevance. *Folia Phoniatrica (Basel)*, 45(2), 76–83.
- Diehl, J. J., & Paul, R. (2009). The assessment and treatment of prosodic disorders and neurological theories of prosody. *International Journal of Speech Language Pathology*, 11(4), 287–292.
- Diehl, J. J., & Paul, R. (in press). Acoustic and perceptual measurements of prosody production on the PEPS-C by children with autism. *Applied Psycholinguistics*.
- Diehl, J. J., Watson, D., Bennetto, L., McDonough, J., & Gunlogson, C. (2009). An acoustic analysis of prosody in high-functioning autism. *Applied PsychoLinguistics*, 30, 385–404.
- Gilbert, J. B. (2005). *Clear speech: Teacher's book: Pronunciation and listening comprehension in American English* (3rd ed.). Cambridge: Cambridge University Press.
- Hesling, I., Dilharreguy, B., Peppé, S., Amirault, M., Bouvard, M., et al. (2010). The integration of prosodic speech in high functioning autism: A preliminary fMRI study. *PLoS One*, 5(7), e11571. <https://doi.org/10.1371/journal.pone.0011571>.
- Jarvinen-Pasley, A., Peppé, S., King-Smith, G., & Heaton, P. (2008). The relationship between form and function level receptive prosodic abilities in autism. *Journal of Autism and Developmental Disorders*, 38(7), 1328–1340.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kempster, G. B., Gerratt, B. R., Abbott, K. V., Barkmeier-Kraemer, J., & Hillman, R. E. (2009). Consensus auditory-perceptual evaluation of voice: Development of a standardized clinical protocol. *American Journal of Speech-Language Pathology*, 18, 124–132. [https://doi.org/10.1044/1058-0360\(2008\)08-0017](https://doi.org/10.1044/1058-0360(2008)08-0017).
- Laver, J. (1980). *The phonetic description of voice quality*. Cambridge: Cambridge University Press.
- McCaleb, P., & Prizant, B. (1985). Encoding of new versus old information by autistic children. *Journal of Speech and Hearing Disorders*, 50, 226–230.
- McCann, J., & Peppé, S. (2003). Prosody in autistic spectrum disorders: A critical review. *International Journal of Language and Communication Disorders*, 38, 325–350.
- Oller, D. K., Niyogi, P., Gray, S., Richards, J. A., Gilkerson, J., Xu, D., et al. (2010). Automated vocal analysis of naturalistic recordings from children with autism, language delay and typical development. *Proceedings of the National Academy of Sciences*, 107(30), 13354–13359. Retrieved from www.pnas.org/cgi/doi/10.1073/pnas.1003882107
- Paul, R., Augustyn, A., Klin, A., & Volkmar, F. (2005). Perception and production of prosody by speakers with autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 35, 205–220.
- Peppé, S., & McCann, J. (2003). Assessing intonation and prosody in children with atypical language development: The PEPS-C test and the revised version. *Clinical Linguistics & Phonetics*, 17(4/5), 345–354.
- Peppé, S., McCann, J., Gibbon, F., O'Hare, A., & Rutherford, M. (2007). Receptive and expressive prosodic ability in children with high-functioning autism. *Journal of Speech Language and Hearing Research*, 50, 1015–1028.
- Peppé, S. J. E., Martínez Castilla, P., Coene, M., Hesling, I., Moen, I., & Gibbon, F. (2010). Assessing prosodic disorder in five European languages. *International Journal of Speech-Language Pathology*, 12, 1, 1–1, 7.
- Peppé, S., Cleland, J., Gibbon, F., O'Hare, A., & Martínez Castilla, P. (2011). Expressive prosody in children with autism spectrum conditions. *Journal of Neurolinguistics*, 24, 41–53.
- Shriberg, L., Kwiatkowski, J., Rasmussen, C., Lof, G. L., & Miller, J. F. (1992). The Prosody-Voice Screening Profile (PVSP): Psychometric data and reference information for children (Tech. Rep. No. 1). Phonology Project. Waisman Center on Mental Retardation and Human Development, University of Wisconsin-Madison.
- Shriberg, L. D., Paul, R., McSweeney, J. L., Klin, A., Cohen, D. J., & Volkmar, F. R. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning autism and Asperger's syndrome. *Journal of Speech, Language, and Hearing Research*, 44, 1097–1115.
- van Santen, J. P., Prud'hommeaux, E. T., & Black, L. M. (2010). Computational prosodic markers for autism. *Autism*, 14(3), 215–236.

Parallel Play

- Play

Paraphasia

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Synonyms

[Speech impairment](#)

Definition

Paraphasia is a speech disorder with neurological origins. Although the hearing and comprehension of speech may not be inhibited, the production of speech is not correct. The individual may be able to speak fluently, but with errors. The errors range from the mispronunciations of single words to the combination of words in inappropriate or meaningless ways. Because the sounds or words are mixed up, it may be difficult to understand the intended meaning.

There are three types of paraphasia:

1. Literal or phonemic paraphasia – incorrect phonemes are substituted. For example, one may say “spot” instead of “pot.” Literal paraphasia could also be switching syllables or creating reverse compound words such as “markbook” instead of “bookmark.”
2. Verbal paraphasia – saying a completely different word than the one intended. It could be a semantic replacement and be related to the intended word, or it could be remote with no clear connection to the intended word. An example of semantic verbal paraphasia would be saying “drive” instead of “car.” Remote verbal paraphasia would be saying “dog” instead of “car.”
3. Neologistic paraphasia – more than half of a word is incorrect. Out of context, it is difficult to guess what the intended word was. An example would be substituting “camalee” for “camera.”

See Also

- ▶ [Apraxia](#)
- ▶ [Speech Impairment](#)

References and Reading

- Dell, G. S., Juliano, C., & Govindjee, A. (1993). Structure and content in language production: A theory of frame constraints in phonological speech errors. *Cognitive Science*, 17, 149–195.
- Fargo, J. D., Schefft, B. K., Dulay, M. F., Privitera, M. D., & Yeh, H. (2005). Confrontation naming in individuals with temporal lobe epilepsy: A quantitative analysis of paraphasic error subtypes. *Neuropsychology*, 19(5), 603–611.
- Geschwind, N. (1972). Language and the brain. *Scientific American*, 226, 76–83.
- Goodglass, H., Wingfield, A., & Hyde, M. R. (1998). The Boston corpus of aphasia naming errors. *Brain and Language*, 64, 1–27.
- Loring, D. W. (1999). *INS dictionary of neuropsychology*. London: Oxford University Press.

Paraphilias

- ▶ [Sexuality and Problem Behaviors](#)

Paraprofessional

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Synonyms

[Aide](#); [Assistant](#); [Associate](#); [Classroom aide](#); [Instructional assistant](#); [One-on-One](#); [Para](#); [Paraeducator](#)

Definition

Paraprofessionals are employees who, following appropriate academic education/instruction and/or on-the-job training, work with children

with disabilities, including those with autism, during the day to support their educational needs. The intention of a paraprofessional is to supplement the work of a teacher/service provider (American Speech-Language-Hearing Association 1999). The paraprofessional is becoming better known as a resource for providing services to the individual with ASD (National Research Council 2001).

See Also

- Paraeducator
- Para-educator

References and Reading

- American Speech-Language-Hearing Association. (1999). *Learning disabilities: Use of paraprofessionals* (Relevant Paper). Available from <http://www.asha.org/policy>
- Broer, S. M., Doyle, M. B., & Giangreco, M. F. (2005). Perspectives of students with intellectual disabilities about their experiences with paraprofessional support. *Exceptional Children*, 71(4), 415–430.
- Giangreco, M. F., & Broer, S. M. (2005). Questionable utilization of paraprofessionals in inclusive schools: Are we addressing symptoms or causes? *Focus on Autism and Other Developmental Disabilities*, 20(1), 10–26.
- Giangreco, M. F., Suter, J. C., & Doyle, M. B. (2010). Paraprofessionals in inclusive schools: A review of recent research. *Journal of Educational and Psychological Consultation*, 20, 41–57.
- Giangreco, M. F., Broer, S. M., & Suter, J. C. (2011). Guidelines for selecting alternatives to overreliance on paraprofessionals: Field-testing in Inclusion-oriented schools. *Remedial and Special Education*, 32, 22–38.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.

Pareidolic Faces

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Synonyms

Asperger's; Autism Spectrum Disorder (ASD);
Face blindness; Mind reading; Prosopagnosia

Definition

Pareidolia is the overinterpretation of stimuli in the external world to impose patterns where none exist. This can apply to any sensory modality but is most commonly applied to visual stimuli. Within this realm, the term is often used to refer to the common human tendency to see faces where no faces are present. Familiar instances would include faces in the clouds, images of saviors in burnt toast, and the Man in the Moon (Liu et al. 2014).

Faces are perhaps the most socially significant visual stimuli encountered in the human environment (Palermo and Rhodes 2007). Autism spectrum disorder (ASD) is characterized by deficits in response to social stimuli (APA 2013). Thus, it has been hypothesized that those with ASD may be less susceptible to this illusion should neurotypical humans. Furthermore, it has been suggested that this feature may be used as a diagnostic tool to identify ASD at a relatively early developmental stage. Both of these hypotheses have received some empirical support.

In respect of the former, there is evidence that children with ASD do not pay particular attention to real faces (Kikuchi et al. 2009), at least not spontaneously (Guillon et al. 2016).

In respect of the latter, it has been found that children with ASD are less sensitive to pareidolic faces – that is, to objects that most people spontaneously report as possessing a face (Ryan et al. 2016).

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Guillon, Q., Rogé, B., Afzali, M. H., Baduel, S., Kruck, J., & Hadjikhani, N. (2016). Intact perception but abnormal orientation towards face-like objects in young children with ASD. *Scientific Reports*, 6, 22119, 1–9.
- Kikuchi, Y., Senju, A., Tojo, Y., Osanai, H., & Hasegawa, T. (2009). Faces do not capture special attention in children with autism spectrum disorder: A change blindness study. *Child Development*, 80(5), 1421–1433.
- Liu, J., Li, J., Feng, L., Li, L., Tian, J., & Lee, K. (2014). Seeing Jesus in toast: Neural and behavioral correlates of face pareidolia. *Cortex*, 53, 60–77.

Palermo, R., & Rhodes, G. (2007). Are you always on my mind? A review of how face perception and attention interact. *Neuropsychologia*, 45, 75–92.

Ryan, C., Stafford, M., & King, R. J. (2016). Brief report: Seeing the man in the moon: Do children with autism perceive Pareidolic faces? A pilot study. *Journal of Autism and Developmental Disorders*, 46(12), 3838–3843.

Parent Attitudes Toward Genetic and Epigenetic Tests for ASD

► [Parent Perspectives Toward Biological Testing for Autism](#)

Parent Expectations Mediate Outcomes for Youth with ASD

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Definition

“Parent expectations” is a term used to describe the beliefs parents hold about the likelihood their child will achieve certain outcomes. ASD literature on parent expectations has primarily focused on expectations about outcomes related to the transition to adulthood (e.g., participate in paid employment, live on their own without supervision).

Historical Background

Parent expectations have been widely studied in psychological and educational literature and have demonstrated relevance to a variety of youth achievement-related outcomes in many studies (e.g., Jeynes 2005; Yamamoto and Holloway 2010). The expectancy-value theory of achievement motivation, researched extensively by Allan

Wigfield and Jacquelynne Eccles (e.g., Wigfield and Eccles 2000), describes the role of expectations in informing outcomes and achievement in a wide range of areas. According to Wigfield and Eccles (2000), what individuals expect to achieve and what they value directly influence their efforts, persistence, decision-making, and, ultimately, performance. In this theory, expectations are also thought to be influenced by other personal and societal factors including demographics, the influences of others, gender stereotypes, and self-concept. As written, the theory articulates the role that one’s own expectations for oneself will have on their own outcomes. However, given the important role that parents play in the lives of youth with disabilities, this theory has been proposed to be extended to relate to parent expectations for their youth with ASD (Kirby 2016).

Current Knowledge

Scholarship over the past decade has suggested that parent expectations are relevant considerations for research related to the transition to adulthood for youth with ASD. Several studies using the National Longitudinal Transition Study-2 (NLTS2; IES n.d.) have provided evidence of the relevance of parent expectations during adolescence as important factors predicting outcomes for young adults with ASD. The NLTS2 involved data collection on over 11,000 US students receiving special education services in the year 2000 and involved 5 waves of data collection across 8 years. Students were ages 13 to 16 years at the start of data collection and 21 to 25 at the completion of the study (IES n.d.). In the NLTS2 survey, parent expectations for various young adult outcomes were measured using a four-point scale (i.e., definitely will not, probably will not, probably will, definitely will). Although the reasons are poorly understood, parents of youth with ASD in the NLTS2 reported among the lowest (i.e., least independent) expectations for young adult outcomes when compared with youth with other educational disability classifications (Newman 2005).

Using data from the NLTS2, Doren et al. (2012) identified parent expectations of future

employment (i.e., the likelihood that their child would eventually have a paid job) as a significant predictor of later employment for youth combined across multiple disability categories. Carter et al. (2012) also found parent expectations to be significant predictors of later employment for youth with severe disabilities (i.e., those eligible for alternative assessments at school). Both Doren et al. (2012) and Carter et al. (2012) looked at broader disability groups that included youth with ASD. With just the sample of youth with ASD in the NLTS2, Chiang et al. (2013) identified that parent expectations of future employment were able to significantly differentiate individuals with ASD who were later employed versus unemployed; however, this variable was no longer significant when included in a multivariate logistic regression model. Similarly, when looking at predictors of postsecondary educational participation, Chiang et al. (2012) found that parent educational expectations were able to significantly differentiate individuals with ASD who later participated in postsecondary education. This remained significant in a multivariate model, suggesting that parents who expected their adolescent child with ASD would participate in postsecondary education were 3.6 times more likely to have a child who did later participate. Across studies, higher parent expectations were associated with more independent outcomes for youth.

Using the NLTS2 sample of youth with ASD, Kirby (2016) applied structural equation modeling techniques to test parent expectations as a statistical mediator of outcomes in young adulthood. The key variables studied were family background (i.e., annual household income, maternal education, race/ethnicity), functional performance (i.e., self-care skills, academic skills, and social skills), and parent expectations about if their child will eventually have a paid job and live independently at the first data collection wave, as well as young adult outcomes (i.e., had a paid job and lived independently) at the final data collection wave. Kirby first ran a model that excluded parent expectations demonstrating how family background and functional performance variables in adolescence significantly predicted to later

young adult outcomes. Kirby then added parent expectations as a mediator into the structural equation model and found that the effects of both family background and functional performance were significantly mediated through parent expectations. In the updated model, neither family background nor functional performance directly predicted young adult outcomes; parent expectations were the only variable with a significant direct prediction of young adult outcomes (Kirby 2016). Kirby's (2016) findings suggest that both family background and youth functional performance significantly and directly predict parent expectations about if their child will get a paid job and live independently, which in turn significantly and directly predict young adult outcomes. This study suggested that although parent expectations have often been studied as predictors within regression models (i.e., independent variables), it may be more appropriate to conceptualize them as mediators (Kirby 2016). Furthermore, this study led to questions about if parent expectations causally influence outcomes or if they are an artifact of other causal influences.

Although Kirby (2016) demonstrated that parent expectations were mediators of outcomes for young adults with ASD, that study did not directly test possible reasons why this may be the case. The two leading hypotheses presented by Kirby (2016) were that (1) parents have a nuanced understanding about their child's future potential and (2) parent expectations directly inform the preparatory actions that parents and youth engage in to prepare for adulthood, thus leading to outcomes that are in line with expectations. Studies have since contributed new information about possible reasons – or mechanisms through which – parent expectations predict outcomes for youth with ASD. A qualitative study of parent expectations for 18 transition-aged youth with ASD suggested that as parents consider their future visions for their children with ASD, it can be difficult for them to disentangle expectations from hopes, fears, and uncertainty (Chen et al. 2018). Another qualitative study, by Kirby et al. (2019a), involved interviews with seven mothers of youth with ASD to examine how they had developed their expectations. Although not the

initial intent, the parents they interviewed also described the ways that their expectations have then influenced the ways they approached planning for the future with their youth. This result suggests that the expectations held by parents inform behaviors and decision-making that can then affect outcomes. For example, parents who believe their child is likely to be employed in young adulthood are more likely to encourage and help their child gain job-like experiences (e.g., chores, volunteer work) in adolescence, which will lead to them being more prepared for paid work. This directly ties back to the expectancy-value theory of achievement motivation (Wigfield and Eccles 2000) and its assertion that expectations influence decisions and behavior. These results also support Yamamoto and Holloway's (2010) hypothesis that parent expectations influence the extent to which parents engage in preparation for outcomes with their children.

Two papers lead by Holmes (Holmes et al. 2016; Holmes et al. 2018) looked at parent expectations as both independent and dependent variables to better understand the role they may play in the transition to adulthood for youth with ASD. These studies provide additional insight into the potential role that parent expectations play as mediators and also contribute to understanding of mechanisms that may explain how expectations influence outcomes. In the first study, Holmes et al. (2016) explored the role of parent expectations as related to parent-child sexuality communication for youth with ASD. They found that for youth with greater ASD symptoms, parents had lower expectations that they would have future romantic relationships and in turn reported having communicated about fewer sexuality-related topic areas with their child. Interestingly, parents of youth with ASD and IQ scores below 70 who did have expectations that their child would have healthy sexual relationships in the future reported having engaged in more parent-child sexuality communication. However, they did not see the same effect with youth who had average or above-average IQ scores (Holmes et al. 2016). In the second study, Holmes et al. (2018) found that greater ASD symptoms and IQ scores below

70 were significant predictors of lower (i.e., less independent) parent expectations for the future in the areas of financial independence, post-secondary education, citizenship, and independent living. When looking at the role of youth gender, parents of daughters with ASD had lower total expectations for the future compared to parents of sons. Since other factors were controlled for, this difference in parent expectations may be related to gender differences in societal expectations or different parental priorities for female versus male youth (Holmes et al. 2018). Holmes et al. (2018) also explored if parent expectations predicted transition preparatory activities, further extending the work to understand mechanisms described previously. They found that parent expectations predicted many of the transition preparatory activities they measured. For example, they found that parent expectations for the future predicted the likelihood that parents reported talking about jobs with their youth, providing chores or other responsibilities at home, and their youth's participation in life skills classes (Holmes et al. 2018). Both studies provide further support for the idea that parent expectations influence outcomes for young adults through the actions of parents (i.e., through a mechanism of decisions and behaviors).

In summary, the current state of research on the role of parent expectations as related to the transition to adulthood in ASD suggests that family- and youth-level factors (e.g., socioeconomic status and functional skills, respectively) inform the expectations parents develop for their child's future. Those expectations then influence decision-making and behaviors engaged in by parents and youth to prepare for adulthood. In turn, those preparatory actions, or lack thereof, lead to the independence-related outcomes of young adults with ASD (e.g., employment, independent living).

Future Directions

The existing body of research suggests that parent expectations for youth with ASD related to outcomes in adulthood have relevance for future

research and practice. Most critically, there is a need to understand the extent to which parent expectations during the transition to adulthood are changeable. If the expectations held by parents of youth with ASD are malleable, then it is possible that interventions to change parent expectations could have a positive influence on outcomes. A pilot study presented at the 2019 Annual Meeting of the International Society for Autism Research suggested that a group-based parent intervention, titled *Maximizing Adolescent Post-Secondary Success (MAPSS)*, resulted in modest increases in parent expectations after the 6-week program that were also maintained after 1 month (Kirby et al. 2019b). Additional work is needed to confirm that parent expectations are changeable through intervention and to determine if those changes are longitudinally associated with any improvement in youth outcomes.

There are also broader aspects of our understanding of parent expectations and their role in the transition to adulthood for youth with ASD that should be further explored. Notably, research on “parent” expectations in ASD typically has primarily measured the expectations of mothers. Expanding our understanding to include the expectations of fathers and the agreement or disagreement between co-parents and subsequent influences on outcomes will be important for the field. Additionally, more qualitative work and mixed methods research approaches with larger and more diverse study samples could uncover new aspects of the ways that parent expectations contribute to outcomes for youth with ASD. Finally, although the NLTS2 has provided an excellent resource for longitudinal research on this topic, the dataset is aging, and additional large, representative longitudinal studies could substantially further this body of research.

See Also

- ▶ [Adulthood, Transition to](#)
- ▶ [Employment in Adult Life](#)
- ▶ [Factors Affecting Outcomes](#)
- ▶ [Living Arrangements in Adulthood](#)

References and Reading

- Carter, E. W., Austin, D., & Trainor, A. A. (2012). Predictors of postschool employment outcomes for young adults with severe disabilities. *Journal of Disability Policy Studies, 23*, 50–63.
- Chen, J., Cohn, E. S., & Orsmond, G. I. (2018). Parents’ future visions for their autistic transition-age youth: Hopes and expectations. *Autism, 23*, 1363–1372.
- Chiang, H.-M., Cheung, Y. K., Hickson, L., Xiang, R., & Tsai, L. Y. (2012). Predictive factors of participation in postsecondary education for high school leavers with autism. *Journal of Autism and Developmental Disorders, 42*, 685–696.
- Chiang, H.-M., Cheung, Y. K., Li, H., & Tsai, L. Y. (2013). Factors associated with participation in employment for high school leavers with autism. *Journal of Autism and Developmental Disorders, 43*, 1832–1842.
- Doren, B., Gau, J. M., & Lindstrom, L. E. (2012). The relationship between parent expectations and post-school outcomes of adolescents with disabilities. *Exceptional Children, 79*(1), 7–23.
- Holmes, L. G., Himle, M. B., & Strassberg, D. S. (2016). Parental romantic expectations and parent–child sexuality communication in autism spectrum disorders. *Autism, 20*, 687–699.
- Holmes, L. G., Kirby, A. V., Strassberg, D. S., & Himle, M. B. (2018). Parent expectations and preparatory activities as adolescents with ASD transition to adulthood. *Journal of Autism and Developmental Disorders, 48*, 2925–2937.
- Institute of Education Sciences (IES) (n.d.). Welcome to NLTS2. Retrieved from www.nlts2.org
- Jeynes, W. H. (2005). A meta-analysis of the relation of parental involvement to urban elementary school student academic achievement. *Urban Education, 40*, 237–269.
- Kirby, A. V. (2016). Parent expectations mediate outcomes for young adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 46*, 1643–1655.
- Kirby, A. V., Bagatell, N., & Baranek, G. T. (2019a). The formation of postsecondary expectations among parents of youth with autism spectrum disorder. *Focus on Autism and Other Developmental Disabilities*. <https://doi.org/10.1177/1088357619881221>.
- Kirby, A. V., Cottle, K. J., Himle, M. B., Diener, M., Wright, C., & Hoffman, J. (2019b). Pilot test of an intervention for parents of youth with ASD focused on life skills and preparing for adulthood. *Poster presented at the Annual Meeting of the International Society for Autism Research*, Montreal.
- Newman, L. (2005). NLTS2 data brief: Family expectations and involvement for youth with disabilities. *A Report from the National Longitudinal Transition Study-2, 4*(2), 1–5. Retrieved from <http://www.ncset.org/publications/viewdesc.asp?id=2473>.

- Wigfield, A., & Eccles, J. S. (2000). Expectancy-value theory of achievement motivation. *Contemporary Educational Psychology*, 25, 68–81.
- Yamamoto, Y., & Holloway, S. D. (2010). Parental expectations and children's academic performance in socio-cultural context. *Educational Psychology Review*, 22, 189–214.

Parent Involvement

► Parent-Professional Partnership

Parent Perspectives Toward Biological Testing for Autism

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Synonyms

Parent attitudes toward genetic and epigenetic tests for ASD

Definition

Autism spectrum disorder (ASD) has many known genetic risk factors (Carter and Scherer 2013). As part of the comprehensive evaluation process for idiopathic ASD, genetic testing is recommended by the American Academy of Pediatrics (AAP) guidelines (Johnson and Myers 2007). Yet the cause of ASD remains unknown. Gene-environment interactions are believed to be involved in the etiology of the disorder (Lai et al. 2014). This has prompted interest in the epigenetic signatures unique to children with ASD. Epigenetic factors regulate gene expression in response to both internal (cellular) and external environments, but do not modify the actual DNA

sequence. There is potential for both genetic and epigenetic measurements to inform our biologic understanding of ASD and perhaps aid in ASD diagnosis.

Recent advances in biomedical research have demonstrated the potential for molecular testing to provide an early, objective, and accurate adjunct to childhood ASD diagnosis (Hicks et al. 2018, in press). Yet, parent perspectives toward genetic and epigenetic testing for ASD are not well-studied. It is imperative for scientists, clinicians, and the public to work collaboratively in assessing novel biotechnology that may impact medical decisions related to child health. The scientific community must take a collaborative approach to technology development, by understanding and incorporating parent needs and perspectives toward biological tests for ASD.

In a study investigating parents' understanding of genetics and epigenetics, concerns and attitudes toward ASD testing, and motives for participation in epigenetic research, 244 parents were administered a 16-question survey. To explore the influence of child developmental status on parental views, the study included parents of children with ASD (n = 131), non-ASD developmental delay (n = 39), and typical development (n = 74).

Parents had positive perceptions of genetic/epigenetic research in ASD. Most parents were strongly altruistic, indicating that "helping other children in the future" (85%) was the main reason for being involved in molecular research for ASD. Despite increasing public awareness about molecular technology (88% of parents were familiar with genetics), few fully understood how genetics differed from epigenetics (19%). Although parents did not doubt the scientific evidence behind genetic or epigenetic technology (0%), some had concerns about implications of genetic/epigenetic testing for ASD on privacy (14%) and insurance status (10%). The lack of a clear understanding of the science behind epigenetic testing did not impact parent interest. Most parents (95%) desired a molecular test to aid in ASD diagnosis when their child was 12 months of age or younger (71%). Surprisingly, overall interest in ASD testing and many of the parental perspectives reported

in the study were not dependent on child developmental status. Parents of typically developing children endorsed value in molecular testing, despite the absence of a child with ASD. The majority of parents (76%) desired a full return of genetic/epigenetic results, regardless of the known medical implications. This finding suggests a strong need for genetic/epigenetic tests to be ordered and interpreted by a healthcare provider, especially given the dichotomy between parents' understanding of genetic/epigenetic science and their desire for full results.

A molecular diagnostic aid for ASD has the potential to enhance the current behavior-based diagnostic process while addressing parent preferences for rapid, objective ASD assessment. It will be crucial that such a test carefully consider ethical, policy, and social challenges before introducing new technology to the healthcare system. It will also be important to consider the potential ramifications associated with the release of genetic/epigenetic information to parents, schools, and health professionals, as well as the policy implications surrounding treatment that might occur as a result of early, biologic ASD assessment. Understanding the motives, concerns, and wishes of parents regarding the development and delivery of genetic/epigenetic technology provides an ethical framework for translating molecular research into socially responsive medical advancement with the potential to benefit families and children with ASD.

See Also

- [Biomarker Research in Autism Spectrum Disorder](#)
- [Ethics](#)
- [Gene Regulatory Networks in Autism](#)
- [Metabolic Testing](#)
- [Parental Response to Diagnosis](#)

References and Reading

Carter, M. T., & Scherer, S. W. (2013). Autism spectrum disorder in the genetics clinic: A review. *Clinical Genetics*, 83(5), 399–407.

Hicks, S. D., Rajan, A. T., Wagner, K. E., Barns, S., Carpenter, R. L., & Middleton, F. A. (2018). Validation of a salivary RNA test for childhood autism spectrum disorder. *Frontiers in Genetics*, 9, 534.

Hicks, S. D., Carpenter, R. L., Wagner, K. E., Pauley, R., Barros, M., Tierney-Aves, C., ... & Middleton, F. A. (in press). Saliva MicroRNA differentiates children with autism from peers with typical and atypical development. *Journal of the American Academy of Child & Adolescent Psychiatry*

Johnson, C. P., & Myers, S. M. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120(5), 1183–1215.

Lai, M. C., Lombardo, M. V., & Baron-Cohen, S. (2014). Autism. *Lancet*, 383(9920), 896–910.

Parent Protections

► [Procedural Safeguards](#)

Parent Responsiveness to Children at Risk of ASD

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Definition

Marisa claps as her toddler, Stela, fits a star into the shape sorter. “Star!” Marisa says. Marisa offers her daughter another shape, but Stela shakes her head and pats the music player.

“Ah!” Stela says, glancing at her mother.

“Music?” Marisa asks. Stela grins at her, giggling. “Okay!” Marisa says. “Here’s the music!” She turns on a collection of children’s songs, singing as Stela dances to the music.

This interaction provides several examples of **parent responsiveness**. Parents demonstrate “responsiveness” when they: (1) recognize their child’s current focus of attention and (2) quickly respond to that interest in a positive and meaningful way, such as by playing with or talking about the child’s interest. Other qualities of “responsiveness” include being affectionate and animated,

offering praise and encouragement, sharing control of the interaction, taking conversational or play turns, and interacting within or slightly above the child's current developmental level. When parents use these behaviors that take into account their child's interests, communicative signals, affective state, and/or developmental level, they promote their child's further development in domains such as communication, social skills, and cognition (Landry et al. 2001). Because parent responsiveness fosters social interaction and communication, this topic is important to consider in the context of children with or at-risk for autism spectrum disorder (ASD), who demonstrate deficits in social communication. Based on the growing research in this area, this chapter will discuss the following topics: (1) the historical context of parent responsiveness, (2) current understandings of parent responsiveness to children at-risk for ASD, (3) ASD interventions targeting parent responsiveness, and (4) future directions.

Historical Background

Recognizing ASD as a biological condition is critical when discussing parent responsiveness. Historically, professionals blamed parents for causing autism, viewing the disorder as relationship-based, rather than biologically based. Decades of research now contradict this belief, indicating that ASD is caused by brain differences, rather than any way that parents interact with their child, but the harm to families has been deep and long-lasting. A brief review of this history is warranted to clarify how views of parent responsiveness in ASD have changed over time.

Parent-child relationships in the context of autism were first described by Dr. Kanner in 1943, who stated that the parents of these children were not "warmhearted." The idea of "cold" parenting took root in popular culture, leading to the harmful concept of "refrigerator mothers" – or the idea that children develop autism because of their mothers' frigid personalities. The "refrigerator mother" movement caused extreme harm to families, leading to parents being separated from their children, stigmatization, and feelings of guilt

(Feinstein 2011). In the 1960s and 1970s, parents and researchers questioned the validity of blaming autism on parent-child relationships and made discoveries that pointed toward biological causes of autism, ushering in the concept of an "autism spectrum disorder" (Feinstein 2011). In 2011, researchers discovered that infant siblings of children with ASD had an approximately 1:5 chance of developing ASD, as compared to 1:68 children in the general population, providing further evidence for the genetic nature of ASD (Ozonoff et al. 2011). Researchers also found that supportive and warm parenting styles in families with ASD are not inherently different than other families (Choi et al. 2019; Ku et al. 2019; Siller and Sigman 2002; Watson 1998). Meanwhile, advances in technology allowed for research into biomarkers of ASD, generating further findings that ASD involves neurological disruptions (e.g., Hazlett et al. 2017).

Although our understanding of what causes ASD is still unfolding, one point is clear: ASD is not caused by "cold" parenting. When considering parent responsiveness to children at-risk for ASD, then, it is critical to make the distinction between: (1) blaming parents for not acting "warm enough," an idea that is outdated and detrimental; versus (2) recognizing that parents are responding to their children in typical ways, but that the child's disorder impedes the process of a fluid social interaction. Interventions targeting "parent responsiveness" are not meant to "remedy" parent behaviors but rather to provide strategies to help parents overcome the social difficulties created by their child's ASD.

Current Knowledge

With this history in mind, the next section will detail what is currently known about parent responsiveness to children at-risk for ASD. The Transactional Model of Development posits that children's characteristics influence how parents act and, in turn, these parent behaviors influence children's development (Sameroff 2009). Using this model in the context of children at-risk for ASD, it is important to consider: (1) how early

characteristics of ASD influence parent responsiveness and conversely (2) how parent responses to children at-risk for ASD impact child development.

Impact of ASD Characteristics on Parent Responsiveness

Children are primarily identified as “at-risk” for ASD for two reasons: (1) the child has been flagged as “at-risk” during a developmental screening and/or (2) the child has an older sibling diagnosed with ASD, which increases the probability developing ASD. Research with each “at-risk” group provides insight into how children at-risk for ASD could influence parent responsiveness by demonstrating symptoms in one or both ASD diagnostic categories: (a) social interaction and communication impairments and/or (b) restricted and repetitive patterns of behaviors and interests.

Social interaction and communication.

Social communication impairments have been the most widely studied, in terms of their influence on parent-child interactions. Among typically developing children, strong communicators elicit more parent responses (Abraham et al. 2013). Communication behaviors among children at-risk for ASD similarly impact parent responses – the main difference, compared to typically developing infants, is how well children communicate. Infants at-risk for ASD tend to develop poorer social communication skills than low-risk infants, producing fewer consonant-vowel combinations (Garrido et al. 2017b), less sophisticated gestures, and fewer gestures overall (Choi et al. 2019; Wan et al. 2019). By 18 months, infants at-risk for ASD use: (1) one-eighth as many “pointing and showing” gestures as low-risk infants (Leezenbaum et al. 2014) and (2) significantly fewer gesture-speech combinations than low-risk infants (Choi et al. 2019; Wan et al. 2019). These types of gestures are considered sophisticated forms of communication, because they are later-developing and bring other people into a shared interaction (Choi et al. 2019; Paul and Norbury 2012). Because vocalizations and gestures both impact how parents respond, each will be described below.

Looking at vocalizations, an infant’s use of consonant-vowel combinations is important for eliciting responses. Parents of 9-month-old infants tend to respond more when their baby combines consonants and vowels (e.g., “bah-bah”), as opposed to only using vowels (e.g., “ah-ah”). This pattern is the same across low-risk and high-risk infants (Talbot et al. 2015); however, infants at-risk for ASD tend to produce fewer consonant-vowel combinations (Garrido et al. 2017b), use fewer directed vocalizations in a social back-and-forth manner (Garrido et al. 2017b; Swanson et al. 2017), and use fewer gesture-speech combinations than typically developing infants (Choi et al. 2019). Thus, infants at-risk for ASD may evoke less feedback from their caregivers because their vocalizations are often: (a) less sophisticated (i.e., vowels-only; not supplemented by gestures) and (b) not directed to others. Another way these vocalizations may impact parent behaviors is through prosodic matching: parents of typically developing infants tend to decrease their use of “motherese” (e.g., higher-pitched speech directed toward infants) between 12 and 18 months, whereas mothers of infants at-risk for ASD tend to increase the pitch of their voice, possibly in response to the slower development of sophisticated vocalizations among the high-risk group (Wan et al. 2019).

In addition to vocalizations, using more sophisticated gestures also encourages parents to respond more frequently. One way of responding to gestures is by “translating” the gesture – or putting the gesture’s meaning into words (e.g., saying “up” when infants raise their hands). When 13-month-olds have no family history of ASD, parents translate “giving and requesting” gestures (early developing) less frequently, instead providing more translations for “pointing and showing” gestures (later developing). However, parents of “high-risk” siblings translate “giving and requesting” gestures more frequently than parents of low-risk infants, while also providing translations for other gestures (Leezenbaum et al. 2014). Interestingly, parents of high-risk siblings who do not go on to develop ASD tend to gesture more frequently themselves, using gestures with a greater variety of meanings

than parents of low-risk infants or infants who later develop ASD (Wan et al. 2019). It may be that having an older child with ASD prompts parents to be more attuned to and/or more concerned about their younger child's communication, making them more likely to model a variety of gestures and translate all forms of their child's gestures, rather than selectively responding to more advanced gestures (Leezenbaum et al. 2014; Wan et al. 2019).

Another factor influencing parent responses is the child's ability to walk while gesturing. When typically developing 13-month-olds move while gesturing, mothers tend to direct the child's actions (e.g., "Throw the ball!"); however, if the child gestures while remaining stationary, mothers vary between offering affirmations, descriptions, directives, or no verbal response. Overall, mothers tend to provide: (a) more verbal responses to children who are walkers and (b) no verbal responses to crawlers (Karasik et al. 2014). Currently, it is unknown how the motor abilities of infants at-risk for ASD impact parent responsiveness; however, research examining motor skills sheds some light on this issue. When compared to low-risk infants, high-risk infants demonstrate poorer fine and gross motor skills from 12 to 36 months of age (Garrido et al. 2017a). High-risk infants who go on to develop ASD also tend to have poorer motor skills at 6 months of age (LeBarton and Landa 2019). Among 2- to 17-year-old-children with ASD, higher gross and fine motor skills are associated with better interpersonal skills (Mody et al. 2017). Collectively, these findings suggest a possible link between the development of motor skills and social communication. Because of these motor and social interaction deficits, it seems possible that infants at-risk for ASD would demonstrate difficulty gesturing while walking, reducing opportunities for parents to respond.

Thus far, we have discussed how the ability to *produce* communication impacts parent behaviors. Similarly, the ability to *understand* communication influences how frequently parents respond. Among typically developing infants and toddlers, parents talk about the child's interest more frequently if the child demonstrates greater

receptive language; unsurprisingly, this pattern holds true for toddlers and preschoolers with ASD (Watson 1998). Young children with or at-risk for ASD vary in their language skills but often demonstrate two patterns: (1) lower expressive and receptive language than expected (Garrido et al. 2017a; Paul and Norbury 2012) and (2) relatively weaker receptive than expressive language, suggesting that the foundations of comprehension supporting expressive language are less robust among young children with ASD (Paul and Norbury 2012). Along the same lines, the overall developmental level of children with ASD (e.g., cognitive and language skills) impacts parent-child interactions. Specifically, parents of children with ASD act more controlling when their children have lower developmental levels than expected for their age, possibly reflecting the extra challenge of parenting a child with both ASD and an intellectual disability (Ku et al. 2019).

Restricted and repetitive patterns of behavior. In addition to social communication, infants at-risk for ASD elicit different parent responses based on symptoms in the category of "restricted and repetitive patterns of behavior." This category has not received much attention in parent-responsiveness literature; however, available research suggests that the following behaviors could change parent-child interactions: unusual sensory patterns, activity levels, object play, and stereotyped behaviors.

One study has examined how patterns of sensory reactivity in 1-year-olds at-risk for ASD influences parent responsiveness (Kinard et al. 2017). In this study, infants were identified as "at-risk" for ASD based on a community screening, rather than family history, and elicited different types of behavior from parents, depending on their communication and sensory profiles. When children communicated less and were more hypo-reactive (i.e., exhibited diminished or delayed response to sensory stimuli), parents tended to respond by talking less and using more physical play actions. Conversely, when children communicated more and demonstrated less hypo-reactivity, parents responded with fewer physical play actions but more talking. Both the children's

patterns of sensory reactivity and communication accounted for unique variance in parent responsiveness. The authors speculate that parents of toddlers with hyporeactivity have learned that talking to their child fails to get his/her attention and so rely on physical play responses to engage the child (Kinard et al. 2017). Infants at high familial risk for ASD often demonstrate hyporeactivity (Van Etten et al. 2017); thus, this sensory pattern has the potential to be influential among families with infants at-risk for ASD.

Kinard et al. (2017) also found that a combination of hyperreactivity (i.e., overreaction or painful response to sensory stimuli) and communication skills influenced how parents responded to their children; however, the findings varied, depending on whether communication and hyperreactivity were measured via parent report or clinician observation, making it unclear how to interpret the results. Hyperreactivity in non-ASD populations could provide insight into how this sensory pattern impacts parent behaviors. When infants (not identified as at-risk for ASD) show tactile hyperreactivity, mothers tend to respond more with talking than physical touch and communicate from a farther distance than if their infant has no sensory difficulties (DeGangi et al. 1997). Parents of infants with sensory processing disorders also seem to have a harder time matching their responses to their infants' behaviors (DeGangi et al. 1997).

In addition to sensory patterns, children with or at-risk ASD may demonstrate additional behaviors that make the parent-child interaction difficult, such as low activity levels (Wan et al. 2012), high activity levels (Meek et al. 2012; Watson 1998), wandering without engaging (Watson 1998), overfocusing on one toy (Watson 1998), less functional object play (e.g., spinning objects, visual examination) (Kaur et al. 2015), or producing large amounts of undirected vocalizations, which may function as an early repetitive or stereotyped interest (Swanson et al. 2017). Parents of children with ASD respond in a warm, supportive manner to their child as frequently as parents of typically developing children. However, parents of children with or at-risk for ASD also tend to manage their child's behavior more frequently

(Meek et al. 2012; Ku et al. 2019; Wan et al. 2012; Watson 1998) and demonstrate more negative behaviors, such as dismissing the child's ideas (Ku et al. 2019). These differences in behavior management have been seen as early as 7 months (Wan et al. 2019) and might reflect an attempt to stimulate, focus, or expand the children's play repertoires (Watson 1998). In the case of younger siblings, it may also be that parents have developed a directive style based on interactions with their older child with ASD, which they now use with their infant, or they may be experiencing stress from parenting an older child with ASD that contributes to the expression of negative behaviors and/or influences how much they direct their infant's behaviors (Ku et al. 2019; Wan et al. 2012). The relationship between parental directive actions and children's restricted and repetitive behaviors needs to be examined in future research.

Impact of Parent Responsiveness on Children at-Risk for ASD

With an understanding of how a child's ASD characteristics could influence parent responses, this section will discuss how, in turn, parent responsiveness could impact children at-risk for or with ASD. The role of parent responsiveness on child outcomes can be viewed in three ways: (1) intensity level and consistency over time, (2) type of response and how well the response "matches" the child's current needs, and (3) parent responses in the context of parent-implemented interventions. Each of these aspects will be discussed below, in terms of how they contribute to child outcomes.

Intensity and consistency of parent response. The intensity and consistency of parent responses is crucial for achieving optimal child outcomes. Parents who are highly responsive spend much of their interactions: (1) observing their child to determine his/her interests or communication attempts and (2) quickly responding to the child's actions in a way that is supportive, affectionate, meaningful, and appropriate to the child's current interests and abilities. Parents who are consistently responsive will maintain these high levels of responsivity from infancy through

childhood. When parents demonstrate a combination of high-intensity and consistent responsiveness, their children are likely to develop stronger cognition, communication, and social skills than if parents provide low or inconsistent levels of responding (Landry et al. 2001). Children can still benefit from periods of high-responsiveness if parents are inconsistent in maintaining these levels (e.g., highly responsive during infancy but minimally responsive during preschool years); however, such inconsistent responsiveness is not as effective as consistent responsiveness in supporting children's long-term cognitive, linguistic, and social gains (Landry et al. 2001). Inconsistent patterns of responding could occur for many reasons, such as (a) beliefs about what children need as infants versus as preschoolers (Landry et al. 2001) and/or (b) characteristics of the child that make it increasingly difficult to respond, as described earlier in this chapter.

Type and developmental “match” of parent response. In addition to intensity and consistency, children achieve different outcomes depending on: (a) the type of parent response and (b) how well this response matches the child's current needs. Parent responses are often divided into two categories: (1) physical actions and (2) verbal feedback. Physical actions are defined as play actions or other physical movements parents use in response to the child's focus of attention (e.g., imitating actions, demonstrating new actions, helping the child, pointing to or showing objects, or providing encouragement or affection in a physical way). Verbal feedback is defined as talking in response to the child's current focus of attention (e.g., verbally imitating, expanding, labeling, translating gestures, describing, affirming, and directing or prompting).

When defining verbal responses, a distinction should be made between “nondirective,” “directive,” and “re-directive/intrusive” verbal behaviors. Nondirective verbal feedback involves commenting on a child's interest without directing his/her actions (e.g., “That's a cow”). In contrast, directive verbal feedback is responsive to the child's interest but requires that the child change his/her behavior (e.g., “Put the cow in the barn”).

Redirections or “intrusive” comments are not considered directive feedback, because they are unresponsive to any aspect of the child's attention (e.g., “We're done with the cow, let's read a book”) (Siller and Sigman 2002). Directive feedback is beneficial for both children with typical development and ASD, as long as parents are responding to the child's focus of attention (Masur et al. 2005; McDuffie and Yoder 2010); thus, both directive and nondirective feedback will be included in this discussion.

Children seem primed to benefit from a variety of verbal and physical responses that are well matched to their current needs. Around 9- to 10-months-old, infants make gains in motor skills and intentional communication that allow them to crawl and explore their environment, manipulate objects in more advanced ways, and initiate social games (Kaur et al. 2015; Paul and Norbury 2012; Wan et al. 2012). For example, infants at low-risk for ASD begin purposefully dropping objects around this age to explore sound effects and initiate social games with their parents (Kaur et al. 2015). Infants often imitate their first words sooner when parents describe these exploring behaviors (Tamis-LeMonda et al. 2001). In contrast, infants at-risk for ASD demonstrate less activity, movement, and object manipulation at this age (Wan et al. 2012) and are less likely to drop objects to initiate social games (Kaur et al. 2015). As detailed earlier, parents of infants at-risk for ASD tend to redirect their children more frequently, possibly due to the lack of functional play and presence of stereotyped or repetitive behaviors that make it challenging to respond. The impact of these directive behaviors is unclear; however, among infants with familial risk for ASD, parent responsiveness has been associated with more frequent parent-directed smiles, whereas directiveness has been associated with slower development of directed smiling between 9 and 18 months of age (Wan et al. 2019). Similarly, research with typically developing infants indicates that *responsive* directive behaviors are associated with positive language outcomes, but *intrusive* directive behaviors are associated with smaller vocabulary growth (Masur et al. 2005).

In addition to motor and object exploration, 9- to 10-month-olds are making advances in their vocalizations, babbling with consonant-vowel combinations to engage in back-and-forth social interactions (Paul and Norbury 2012). When parents respond to vocalizations at this age, infants seem ready to learn vocabulary, saying their first words sooner when parents provide: nondirective verbal feedback (e.g., describing, affirming), directive verbal feedback (e.g., prompting play), and demonstrations of play actions. Directive feedback (e.g., prompting play and demonstrating play actions) also helps children acquire more vocabulary and combine words at earlier ages (Tamis-LeMonda et al. 2001). When compared with low-risk infants, infants at-risk for ASD produce more vowel-only vocalizations, less social babbling, and fewer conversational turns (Garrido et al. 2017b; Swanson et al. 2017), possibly eliciting fewer or different parent responses (Talbot et al. 2015). More research is needed to explore how this dynamic impacts later child outcomes; however, infants at-risk for ASD may be missing verbal and/or physical feedback that could be beneficial for later vocabulary development.

Between 10 and 12 months, infants advance from babbling to word attempts (Paul and Norbury 2012). Responses that support early word imitation become important for later language outcomes. At 10 months, children's language seems most supported when parents respond with physical actions, as opposed to verbal responses. When parents physically respond in a way that matches the child's interests, engages them in an interaction, and is meant to help the child learn, infants tend to have larger expressive vocabularies at 13 months of age (Masur et al. 2005). In contrast, infants have lower expressive vocabularies at 13 months if their parents were very talkative at 10 months (Masur et al. 2005). Shorter utterances and more physical play actions may better match children's current abilities than "streams" of talking, which could be hard for 10-month-olds to process (Masur et al. 2005).

Around their first birthday, infants reach new milestones in communication, motor skills, and play, allowing them to benefit from parent

feedback in more complex ways. In terms of verbal communication, 1-year-olds say their first nonimitated words, which are formed with consonant-vowel shapes and can be used in conjunction with pointing to comment on objects (e.g., saying "ba"/ball while pointing to a ball) (Paul and Norbury 2012). Whereas physical responses seem most influential at 10 months, verbal responses become most powerful at this new stage, scaffolding 1-year-olds' attempts at word production. Types of verbal responses that seem most beneficial include: (a) verbal imitation, which helps children build their vocabulary; (b) verbal imitations, expansions, and play prompts, which help children combine words sooner; and (c) asking questions, which (along with verbal imitations and expansions) help children learn to talk about the past (Masur et al. 2005; Tamis-LeMonda et al. 2001). When compared to parents of low-risk infants, parents of infants at-risk for ASD make smaller increases in how much they respond to their infant's vocalizations over 13–18 months of age (Leezenbaum et al. 2014), possibly because of the nonsocial and unsophisticated nature of their child's vocalizations (Garrido et al. 2017b). More research is needed to examine how reduced verbal feedback at 1 year of age could impact child language outcomes; however, these limited parent responses could have negative implications, given the power of verbal input at this age.

As more sophisticated gestures emerge around the end of the first year, parents are prompted to "translate" the gesture's meaning into words (Leezenbaum et al. 2014). In turn, these translations help children develop more vocabulary, such as (a) more nouns by 17 months when parents translate "commenting" gestures with object-labels and (b) more verbs by 17 months when parents translate "requesting" gestures with action words (Olson and Masur 2015). Similar patterns have been found for infants at-risk for ASD. Both infants at low- and high-risk for ASD produce more words at 18 months when their parents translate "pointing and showing" gestures at 13 months (Leezenbaum et al. 2014). In addition to these verbal responses, parents who share positive affect with their 12-month-old seem to foster

more positive affect from their child in return, along with more child vocalizations, gazing at faces, and combinations of communicative means, regardless of risk status for ASD (Schwichtenberg et al. 2019).

As typically developing toddlers approach their second birthdays, they experience a language burst, producing 50–100 words by 18 months, combining words, and needing less contextual support to understand language (Paul and Norbury 2012). Children seem able to process more complex verbal feedback at these older ages, developing a larger subsequent vocabulary when parents provide verbal descriptions and expansions (e.g., Child: “Shoe”; Parent: “Mama’s shoe”), as opposed to simply imitating the child’s vocalization (Masur et al. 2005). Responding with physical actions also helps children develop larger vocabularies (Masur et al. 2005). When children with ASD attempt to communicate, parents respond with nondirective and directive verbal responses, both of which help children increase their vocabulary (McDuffie and Yoder 2010). Similar to typically developing children, expanding the communication attempt of a child with ASD, as opposed to simply imitating, seems to have a greater impact on vocabulary outcomes 6 months later (McDuffie and Yoder 2010). This verbal feedback appears most beneficial when parents adjust the complexity of their verbal input to the child’s current language. When toddlers with ASD are minimally verbal, nondirective verbal feedback is associated with language gains; however, these same benefits are not seen for verbally fluent toddlers with ASD (Haebig et al. 2013). It may be that simplistic verbal input that supports language in minimally verbal toddlers with ASD is not sufficiently complex to scaffold the language of toddlers with ASD with more advanced language skills (Haebig et al. 2013).

Along with communication, toddlers are expanding their play repertoire and ability to pay attention, so that, by 18 months, they are playing simple pretend games and following one-step commands with minimal contextual support (Paul and Norbury 2012). Parents who respond to their child’s focus during play, as opposed to redirecting the child, continue facilitate their child’s language-

learning during the toddler stage (Masur et al. 2005). Although redirections negatively impact vocabulary development, parents who direct their child’s attention seem to be providing examples of how to initiate an interaction, making it more likely that their child will also initiate joint engagement (Meek et al. 2012). In contrast to the play skills and attention levels of typically developing toddlers, toddlers at-risk for ASD continue to mouth objects at 15 months, may only be beginning to use objects with functional ways, and demonstrate limited social attention (Kaur et al. 2015). As long as parents respond to their child’s focus of attention, both directive and nondirective verbal responses to children with ASD have been associated with greater short- and long-term increases in social communication and language (Haebig et al. 2013; McDuffie and Yoder 2010; Siller and Sigman 2002, 2008). Similar to typically developing populations, when parents model how to point to or show a toy, children with ASD tend to initiate joint attention more frequently (Meek et al. 2012; Siller and Sigman 2002).

Thus, for both low-risk infants and infants with or at-risk for ASD, children achieve positive language outcomes when their parents respond to the child’s focus of attention, adapting these responses over time to match the child’s current developmental needs. It is encouraging that responsive parenting helps children with ASD make long-term gains in social and language skills; however, it is also critical to note the challenges that parents face when trying to establish responsive parent-child interactions. To address these needs, researchers have developed parent-implemented interventions for infants and toddlers with or at-risk for ASD, designed to achieve two main outcomes: (1) enhance parent responsiveness through interventionist coaching and (2) improve child outcomes through parent use of responsive strategies. A brief review of parent responsiveness interventions is provided below.

Parent responsiveness interventions. Parent-implemented interventions for ASD have been identified as evidence-based practices by both the National Professional Development Center on Autism Spectrum Disorders (NPDC; Wong et al. 2015) and the National Standards Project

(NSP 2015). Examples of early ASD interventions that include a “parent responsiveness” component are listed in Table 1, along with examples of targeted child outcomes and parent strategies. These types of programs may be most successful

for children at early stages of language development (Siller and Morgan 2018). Although each intervention program offers unique content to families, they share many common goals and strategies. A complete review of parent

Parent Responsiveness to Children at Risk of ASD, Table 1 Examples of parent-implemented interventions incorporating parent responsiveness strategies

Examples of interventions	Examples of child goals	Examples of parent strategies	Reference
Preschool Autism Communication Trial (PACT) intervention	Shared attention; initiating communication; communicating for a variety of reasons; pragmatics	Match parent language to child’s level; interpret child’s behavior as intentional communication; follow child’s lead; provide verbal imitations and expansions; motivate child to communicate with “teasers” or “sabotage”	Green et al. (2010)
Enhanced Milieu Teaching	Word combinations; requesting	Provide verbal imitations and expansions; follow child’s lead; match parent language to child’s targeted level; balance turn-taking with child; provide milieu prompts (e.g., modeling, time delay); give positive feedback	Kaiser et al. (2000)
Joint Attention, Symbolic Play, Engagement, and Regulation (JASPER) intervention	Joint engagement; initiating and responding to joint attention; functional play; symbolic play	Follow child’s lead; imitate child’s actions; describe child’s actions; provide imitations and expansions; give corrective feedback; arrange environment to facilitate engagement (e.g., face-to-face with eye contact)	Kasari et al. (2010)
Joint Attention Mediated Learning (JAML) intervention	Focusing on faces; turn-taking; responding to joint attention; initiating joint attention	Play face-to-face games with vocalization; imitate child gestures; interpret child’s behavior as meaningful communication; join child’s repetitive play; follow child’s lead; pause for child response; play teasing games; create animation, excitement, and suspense; offer “surprises”	Schertz and Odom (2007)
Responsive Teaching (RT)	Cognition; communication; social-emotional functioning	Match responses to child’s developmental level, interest, and behavioral style (e.g., follow child’s lead); create reciprocal interactions (e.g., play face-to-face games without toys); share control (e.g., wait silently for a more mature response); provide contingent feedback (e.g., respond to unintentional behaviors as if meaningful conversation); demonstrate positive affect (e.g., respond to child in playful ways)	Mahoney and MacDonald (2007)
Adapted Responsive Teaching (ART)	Social communication; sensory-regulatory behaviors	Incorporates similar strategies as RT intervention, but uses strategies to target social communication and sensory-regulatory behaviors	Watson et al. (2017)

responsiveness interventions is out of the scope of this current chapter; however, further details and videos of several interventions can be accessed on the following websites: (a) the NPDC evidence-based practices page (<http://autismpdc.fpg.unc.edu/evidence-based-practices>), (b) the ASD video glossary on the Autism Navigator website (<http://resources.autismnavigator.com/>), and (c) the Autism Internet Modules (AIM) website (<http://www.autisminternetmodules.org/>).

Based on current research, recommendations have been proposed to make parent responsiveness interventions as successful as possible. These recommendations include the following: start intervention as soon as risk for ASD is identified; target a variety of developmental domains; adjust goals and strategies to match the child's changing developmental needs; provide parent coaching for at least 9–12 months, including both video feedback and direct, hands-on instruction; allow parents to learn strategies in small doses and in a structured environment; practice strategies in multiple settings and in daily routines to support generalization; include booster sessions to refresh learning; consider using augmentative and alternative communication (AAC) to support language development in children with speech delays; include both professional- and parent-delivered intervention components; and ensure that the intervention is implemented with fidelity (Landa 2018).

Summary and Future Directions

Parent responsiveness is critical for helping children with or at-risk for ASD develop social, communication, and cognitive skills. From a transactional framework, early symptoms of ASD impact how parents respond to their child; in turn, these responses influence child outcomes. Children with better communication skills elicit more responses from parents; however, children at-risk for ASD commonly exhibit deficits in social, communication, and motor skills that may limit how parents respond. Restricted and repetitive patterns of behavior also make parent-

child interactions more challenging for parents. Despite these challenges, parents help their children develop important skills when they: (a) frequently respond to their child's focus of attention, (b) limit how much they redirect their child's attention, (c) maintain these high levels of responsiveness across infancy and childhood, (d) provide a variety of verbal and physical responses that align with the child's skill level, and (e) adapt these responses over time to meet the child's changing needs. Based on the benefits of parent responsiveness, parent-implemented interventions for children with or at-risk for ASD have been developed to: (a) help parents overcome challenges in parent-child interactions and (b) improve child outcomes through enhanced parent responsiveness. Researchers are continuing to study these interventions, so that programs can be tailored to families' needs.

Although parent-implemented interventions have a strong evidence base, researchers are continuing to examine the effectiveness of these interventions for infants and toddlers at-risk for ASD. Interventions have been successful at enhancing parent responsiveness and parent-child interactions, but have achieved varied outcomes for children. These varied outcomes may stem from initial characteristics of the child and family, differences in study design, and/or the fact that there are a wide variety of intervention programs that incorporate parent responsiveness strategies, making it difficult to determine which intervention components are most successful and for whom (Edmunds et al. 2019; Oono et al. 2013; Shalev et al. 2019; Siller and Morgan 2018). Because of these varying outcomes, the field is moving toward an ideal of "tailored" intervention approaches, where programs can be matched to families based on their initial characteristics. For future research, it will be important to reach a consensus on which "active ingredients" are most effective at supporting child outcomes across a variety of parent-implemented intervention programs. It will also be beneficial to examine initial child and family characteristics in at-risk populations and the extent to which these characteristics predict long-term treatment outcomes.

See Also

- ▶ Communication Disorder/Communication Impairment
- ▶ Communication Interventions
- ▶ Developmental Milestones
- ▶ Early Intervention
- ▶ Expressive Language Disorder
- ▶ First Words Project
- ▶ Gross Motor Skills
- ▶ Hyperresponsiveness
- ▶ Hyporesponsiveness
- ▶ Infants with Autism
- ▶ Initiating/Responding to Joint Attention Skills (IJA/RJA)
- ▶ Initiation of Communication
- ▶ JASPER
- ▶ Joint Attention
- ▶ Kanner, Leo
- ▶ Language Interventions
- ▶ Milieu Teaching
- ▶ Naturalistic Interventions
- ▶ Nonverbal Communication
- ▶ Parent Training
- ▶ Parent-Professional Partnership
- ▶ Preverbal Communication
- ▶ Receptive Language Disorders
- ▶ Reciprocal Communication/Interaction
- ▶ Refrigerator Mother
- ▶ Repetitive Behavior
- ▶ Restricted Interest
- ▶ Sensorimotor Development
- ▶ Sensory Impairment in Autism
- ▶ Sensory Processing
- ▶ Social Behaviors and Social Impairment
- ▶ Social Communication
- ▶ Social Interventions
- ▶ Speech Delay
- ▶ Symbolic Play
- ▶ Verbal Communication
- ▶ Verbal Comprehension
- ▶ Zone of Proximal Development (ZPD)

References and Reading

- Abraham, L. M., Crais, E., & Vernon-Feagans, L. (2013). Early maternal language use during book sharing in families from low-income environments. *American Journal of Speech-Language Pathology*, 22(1), 71–83. [https://doi.org/10.1044/1058-0360\(2012/11-0153\)](https://doi.org/10.1044/1058-0360(2012/11-0153)).
- Choi, B., Shah, P., Rowe, M. L., Nelson, C. A., & Tager-Flusberg, H. (2019). Gesture development, caregiver responsiveness, and language and diagnostic outcomes in infants at high and low risk for autism. *Journal of Autism and Developmental Disorders*, 1–17. epub ahead of print <https://doi.org/10.1007/s10803-019-03980-8>
- DeGangi, G. A., Sickel, R. Z., Piserchia Kaplan, E., & Santman Wiener, A. (1997). Mother-infant interactions in infants with disorders of self-regulation. *Physical & Occupational Therapy in Pediatrics*, 17(1), 17–44. https://doi.org/10.1080/J006v17n01_02.
- Edmunds, S. R., Kover, S. T., & Stone, W. L. (2019). The relation between parent verbal responsiveness and child communication in young children with or at risk for autism spectrum disorder: A systematic review and meta-analysis. *Autism Research*, 12(5), 715–731.
- Feinstein, A. (2011). *A history of autism: Conversations with the pioneers*. New York: Wiley.
- Garrido, D., Petrova, D., Watson, L. R., Garcia-Retamero, R., & Carballo, G. (2017a). Language and motor skills in siblings of children with autism spectrum disorder: A meta-analytic review. *Autism Research*, 10(11), 1737–1750. <https://doi.org/10.1002/aur.1829>.
- Garrido, D., Watson, L. R., Carballo, G., Garcia-Retamero, R., & Crais, E. R. (2017b). Infants at-risk for autism spectrum disorder: Patterns of vocalizations at 14 months. *Autism Research*, 10(8), 1372–1383. <https://doi.org/10.1002/aur.1788>.
- Green, J., Charman, T., McConachie, H., Aldred, C., Slonims, V., Howlin, P., . . . Byford, S. (2010). Parent-mediated communication-focused treatment in children with autism (PACT): A randomised controlled trial. *The Lancet*, 375(9732), 2152–2160.
- Haebig, E., McDuffie, A., & Ellis Weismer, S. (2013). Brief report: Parent verbal responsiveness and language development in toddlers on the autism spectrum. *Journal of Autism and Developmental Disorders*, 43(9), 2218–2227. <https://doi.org/10.1007/s10803-013-1763-5>.
- Hazlett, H. C., et al. (2017). Early brain development in infants at high risk for autism spectrum disorder. *Nature*, 542(7641), 348–351.
- Kaiser, A. P., Hancock, T. B., & Nietfeld, J. P. (2000). The effects of parent-implemented enhanced milieu teaching on the social communication of children who have autism. *Early Education and Development*, 11(4), 423–446.
- Karasik, L. B., Tamis-LeMonda, C. S., & Adolph, K. E. (2014). Crawling and walking infants elicit different verbal responses from mothers. *Developmental Science*, 17(3), 388–395. <https://doi.org/10.1111/desc.12129>.
- Kasari, C., Gulsrud, A. C., Wong, C., Kwon, S., & Locke, J. (2010). Randomized controlled caregiver mediated joint engagement intervention for toddlers with autism. *Journal of Autism and Developmental Disorders*, 40(9), 1045–1056.

- Kaur, M., Srinivasan, S. M., & Bhat, A. N. (2015). Atypical object exploration in infants at-risk for autism during the first year of life. *Frontiers in Psychology*, 6, 798. <https://doi.org/10.3389/fpsyg.2015.00798>.
- Kinard, J. L., Sideris, J., Watson, L. R., Baranek, G. T., Crais, E. R., Wakeford, L., & Turner-Brown, L. (2017). Predictors of parent responsiveness to 1-year-olds at-risk for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(1), 172–186. <https://doi.org/10.1007/s10803-016-2944-9>.
- Ku, B., Stinson, J. D., & MacDonald, M. (2019). Parental behavior comparisons between parents of children with Autism Spectrum Disorder and parents of children without Autism Spectrum Disorder: A meta-analysis. *Journal of Child and Family Studies*, 28(6), 1445–1460.
- Landa, R. J. (2018). Efficacy of early interventions for infants and young children with, and at risk for, autism spectrum disorders. *International Review of Psychiatry*, 30(1), 25–39. <https://doi.org/10.1080/09540261.2018.1432574>.
- Landry, S., Smith, K. E., Swank, P. R., Assel, M. A., & Vellet, S. (2001). Does early responsive parenting have a special importance for children's development or is consistency across early childhood necessary? *Developmental Psychology*, 37(3), 387–403. <https://doi.org/10.1037/0012-1649.37.3.387>.
- LeBarton, E. S., & Landa, R. J. (2019). Infant motor skill predicts later expressive language and autism spectrum disorder diagnosis. *Infant Behavior and Development*, 54, 37–47.
- Leezenbaum, N. B., Campbell, S. B., Butler, D., & Iverson, J. M. (2014). Maternal verbal responses to communication of infants at low and heightened risk of autism. *Autism*, 18(6), 694–703. <https://doi.org/10.1177/1362361313491327>.
- Mahoney, G., & MacDonald, J. D. (2007). *Autism and developmental delays in young children: The responsive teaching curriculum for parents and professionals manual*. Austin: PRO-ED.
- Masur, E. F., Flynn, V., & Eichorst, D. L. (2005). Maternal responsive and directive behaviours and utterances as predictors of children's lexical development. *Journal of Child Language*, 32(1), 63–91.
- McDuffie, A., & Yoder, P. (2010). Types of parent verbal responsiveness that predict language in young children with autism spectrum disorder. *Journal of Speech, Language, and Hearing Research*, 53, 1026–1039.
- Meek, S. E., Robinson, L. T., & Jahromi, L. B. (2012). Parent-child predictors of social competence with peers in children with and without autism. *Research in Autism Spectrum Disorders*, 6(2), 815–823. <https://doi.org/10.1016/j.rasd.2011.11.001>.
- Mody, M., Shui, A. M., Nowinski, L. A., Golas, S. B., Ferrone, C., O'Rourke, J. A., & McDougale, C. J. (2017). Communication deficits and the motor system: Exploring patterns of associations in autism spectrum disorder (ASD). *Journal of Autism and Developmental Disorders*, 47(1), 155–162. <https://doi.org/10.1007/s10803-016-2934-y>.
- NSP. (2015). *Findings and conclusions: National Standards Project, Phase 2. Addressing the need for evidence-based practice guidelines for autism spectrum disorder*. Randolph: National Autism Center.
- Olson, J., & Masur, E. F. (2015). Mothers' labeling responses to infants' gestures predict vocabulary outcomes. *Journal of Child Language*, 42, 1–23. <https://doi.org/10.1017/s0305000914000828>.
- Oono, I. P., Honey, E. J., & McConachie, H. (2013). Parent-mediated early intervention for young children with autism spectrum disorders (ASD). *Cochrane Database of Systematic Reviews*, 4, Cd009774. <https://doi.org/10.1002/14651858.CD009774.pub2>.
- Ozonoff, S., Young, G. S., Carter, A., Messinger, D., Yirmiya, N., Zwaigenbaum, L., ... Stone, W. L. (2011). Recurrence risk for autism spectrum disorders: A Baby Siblings Research Consortium study. *Pediatrics*, 128(3), e488–495. <https://doi.org/10.1542/peds.2010-2825>.
- Paul, R., & Norbury, C. F. (2012). *Language disorders from infancy through adolescence: Listening, speaking, reading, writing, and communicating* (4th ed.). St. Louis: Elsevier Mosby.
- Sameroff, A. J. (2009). *The transactional model of development: How children and contexts shape each other*. Washington, DC: American Psychological Association.
- Schertz, H., & Odom, S. (2007). Promoting joint attention in toddlers with autism: A parent-mediated developmental model. *Journal of Autism and Developmental Disorders*, 37(8), 1562–1575. <https://doi.org/10.1007/s10803-006-0290-z>.
- Schwichtenberg, A., Kellerman, A. M., Young, G. S., Miller, M., & Ozonoff, S. (2019). Mothers of children with autism spectrum disorders: Play behaviors with infant siblings and social responsiveness. *Autism*, 23(4), 821–833.
- Shalev, R. A., Lavine, C., & Di Martino, A. (2019). A systematic review of the role of parent characteristics in parent-mediated interventions for children with autism spectrum disorder. *Journal of Developmental and Physical Disabilities*, 1–21. epub ahead of print <https://doi.org/10.1007/s10882-018-9641-x>
- Siller, M., & Morgan, L. (2018). Systematic review of research evaluating parent-mediated interventions for young children with autism: Years 2013 to 2015. In *Handbook of parent-implemented interventions for very young children with autism* (pp. 1–21). Cham: Springer.
- Siller, M., & Sigman, M. (2002). The behaviors of parents of children with autism predict the subsequent development of their children's communication. *Journal of Autism and Developmental Disorders*, 32(2), 77–89. <https://doi.org/10.1023/A:1014884404276>.
- Siller, M., & Sigman, M. (2008). Modeling longitudinal change in the language abilities of children with autism: Parent behaviors and child characteristics as predictors of change. *Developmental Psychology*, 44(6), 1691–1704. <https://doi.org/10.1037/a0013771>.
- Swanson, M. R., Shen, M. D., Wolff, J. J., Boyd, B., Clements, M., Rehg, J., ... Piven, J. (2017). Naturalistic language recordings reveal “hypervocal”

- infants at high familial risk for autism. *Children Development*. <https://doi.org/10.1111/cdev.12777>.
- Talbott, M. R., Nelson, C. A., & Tager-Flusberg, H. (2015). Maternal vocal feedback to 9-month-old infant siblings of children with ASD. *Autism Research*, 9(4), 460–470. <https://doi.org/10.1002/aur.1521>.
- Tamis-LeMonda, C. S., Bornstein, M. H., & Baumwell, L. (2001). Maternal responsiveness and children's achievement of language milestones. *Child Development*, 72(3), 748–767. <https://doi.org/10.2307/1132453>.
- Van Etten, H. M., Kaur, M., Srinivasan, S. M., Cohen, S. J., Bhat, A., & Dobkins, K. R. (2017). Increased prevalence of unusual sensory behaviors in infants at risk for, and teens with, autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(11), 3431–3445. <https://doi.org/10.1007/s10803-017-3227-9>.
- Wan, M. W., Green, J., Elsabbagh, M., Johnson, M., Charman, T., & Plummer, F. (2012). Parent-infant interaction in infant siblings at risk of autism. *Research in Developmental Disabilities*, 33(3), 924–932. <https://doi.org/10.1016/j.ridd.2011.12.011>.
- Wan, M. W., Green, J., & Scott, J. (2019). A systematic review of parent–infant interaction in infants at risk of autism. *Autism*, 23(4), 811–820.
- Watson, L. R. (1998). Following the child's lead: Mothers' interactions with children with autism. *Journal of Autism and Developmental Disorders*, 28(1), 51–59.
- Watson, L. R., Crais, E. R., Baranek, G. T., Turner-Brown, L., Sideris, J., Wakeford, L., ... Nowell, S. W. (2017). Parent-mediated intervention for one-year-olds screened as at-risk for autism spectrum disorder: A randomized controlled trial. *Journal of Autism and Developmental Disorders*, 47(11), 3520–3540.
- Wong, C., et al. (2015). Evidence-based practices for children, youth, and young adults with autism spectrum disorder: A comprehensive review. *Journal of Autism and Developmental Disorders*, 45(7), 1951–1966.

professional delivering the training, and the targets of the PT. The term “PT” has also been used interchangeably with parent management training, parent effectiveness training, parent-mediated training, and parent-assisted training depending on the target group and foci of treatment goals. PT as a model of service delivery has been described extensively in numerous fields to include the mental health and psychological sciences, education, and health sciences. Broadly defined, PT involves teaching parents and caregivers knowledge and skills to more distally impact a child. The specific content and format of PT may vary, as does the delivery and duration of the program. PT programs may include sessions on child development, behavior management, communication, and interaction styles but more specific skills such as increasing a child's compliance to a medical regimen. PT may be delivered individually, in group setting, and via distant learning mediums (Internet, DVD). For purposes of PT related to autism spectrum disorders (ASD), PT assumes parents are taught to intervene with their child with ASD to decrease challenging, interfering behaviors (e.g., aggression, disruptive behaviors) and/or to teach new behaviors. These new behaviors may be directly related to core and early emerging deficits (e.g., social communication behaviors) but also developmentally appropriate skills (e.g., preacademic skills) and self-help, adaptive skills.

Parent Training

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Definition

Over the past 40 years, parent training (PT) has been defined to have slightly differing meaning depending on the intended population, the

Historical Background

As already suggested, teaching parents in the concepts and skills to in turn impact their children's behavior has a long history for a range of childhood issues. PT is a well-established intervention for children with noncompliance and disruptive behaviors. PT, often referred to as parent management training for these populations, has been studied for children with attention deficit/hyperactivity disorder (Barkley et al. 2001) and conduct problems (Kazdin 2003). PT has more recently been demonstrated to be a promising treatment mode for childhood anxiety disorders (Barrett and Shortt 2003) and for tics and Tourette's

(Scahill et al. 2006). PT training during the preschool years to offset the sequelae of childhood behavioral issues has been an active area of inquiry as well (Eyberg et al. 1995; Webster-Stratton and Taylor 2001). Further, PT as treatment or prevention of child abuse is a well-recognized approach (Lundahl et al. 2006).

PT also has a long tradition for children with developmental delay and intellectual disability (ID) broadly defined. Parent involvement is generally accepted to be particularly critical for improved outcome in this population. Baker and colleagues have been instrumental in establishing parent training as an efficacious approach for children with a range of ID (Baker 1996; Baker et al. 1997). Parent training in this line of work focused on teaching parents basic behavioral principles to improve behaviors as well as teaching procedures to promote skill development. Other lines of research have focused more specifically on intervening at a younger age for children to promote early development (Kaiser and Hancock 2003).

A parallel literature has been devoted to interventions for children ASD where the parent is trained to deliver the intervention in lieu of a trained therapist. A plethora of single-subject publications demonstrate that parents of children with ASD can learn techniques for decreasing challenging behaviors as well as improving functional skills, features of autism, and overall compliance (Ducharme and Drain 2004; Lerman et al. 2006; Moes and Freas 2002; Smith et al. 2000; Symon 2005). Across the United States, comprehensive educational and intervention programs for children with ASD very often have a PT component as part of the package of services. However, only more recent years have PT in ASD have withstood the rigors of randomized controlled trials.

Rationale or Underlying Theory

Parent training programs have varied in the theoretical underpinning. The bulk of parent training programs have been behavioral in nature, either based primarily on operant conditioning or social learning theory. Much has been written about coercion theory and more recently on dynamic

systems principles as explanatory of the development of antisocial childhood behaviors (Granic and Patterson 2006). For parents of children with disruptive behaviors, but not ASD (non-ASD), there is a core assumption that deficit or dysfunctional parenting and parent-child interactions are at least contributory to the child's maladaptive behaviors. This hypothesis is not made for parents of children with ASD. Rather, the assumption is that parenting a child with ASD is particularly challenging given the child's core deficits, and parenting a child requires a higher level of skill and knowledge and higher level of input than that of developing a child without ASD.

PT programs specifically for ID and ASD have been primarily based on operant learning procedures but have also included developmental theory as well. The empirical support for operant learning procedures borne out of the field of applied behavior analysis (ABA) over the past 30 years is unequivocal for those with ID and ASD. These procedures have been in turn taught to parents. The rationale for including parents is based on several assumptions such as the child is most influenced by the parent, the parent spends considerable time with the child, and, of course, the parent is motivated, willing to learn and implement the behavioral procedures, and has the necessary resources to do so.

Goals and Objectives

While some PT programs for children with ASD have targeted disruptive, challenging behaviors (self-injury), other programs have specifically target core deficits to include communication skills, social skills, and adaptive skills. In keeping with a more stringent ABA model, PT for ASD has also focused more on determining the function of behaviors in children with communication deficits. In our own work, an emphasis has been placed on teaching parents to identify the function of their child's behavior. An emphasis has also been on teaching parents a range of antecedent management approaches such as the use of visual strategies and functional communication approaches as well as more traditional consequence approaches

(reinforcement, extinction, and mild punishments). Other PT programs have emphasized skill development related to core deficits and developmentally appropriate skills to include early prerequisite skills such as joint attention, imitation skills, non-verbal and verbal communication skills, and social engagement. More descriptive information of representative programs is offered in section G. PT has often been an adjunct to other interventions but has also been the sole treatment for children with ASD.

Treatment Participants

Within a PT model, both the parent/caregiver and child are considered participants. While the intervention is delivered to the parent, the primary outcomes of interest are those distal effects on the child. However, changes in parent behaviors are of interest as well. Parent variables associated with better versus poorer outcomes have been closely evaluated in the non-ASD literature, but are only now being evaluated more systematically in the PT literature specific to ASD. For PT for children with disruptive and noncompliance behaviors but typically developing, parent's adherence to the treatment has been shown to predict outcome, and specific attention to enhancing adherence has been studied (Nock and Ferriter 2005). Adherence has been defined in various ways but has included such variables as whether the parent regularly attended to sessions, parent completed requested homework, and parent implemented taught strategies.

While child variables associated with more or less response to PT have not specifically been determined in the ASD field, child variables more responsive to psychosocial treatments more broadly defined have been suggested. Higher developmental, cognitive, and language levels are believed to be associated with better outcomes in general and larger gains with psychosocial treatments. Not surprisingly, better outcome has been shown to be associated with fewer symptoms of ASD at outset. Much more research is necessary to determine which parent and which child with ASD will most likely benefit from a PT model of service delivery and then to

what PT program more specifically. Considerably more research efforts are needed before such questions can be answered.

Treatment Procedures

PT programs and procedures have widely differed across several variables. These include the intended target of PT program, the ages of the children with ASD included, duration or "dose" of training parents received, and how the program was delivered (individual, group, distant/online program). The PT may have been highly specific to the child or a manualized approach. Moreover, as already mentioned, the goals of the PT have been broad. For purposes of discussing the current literature, an overview of a few representative programs addressing challenging problems, those addressing core skill deficits and teaching of new skills, and finally those emerging programs via distant, Internet format are provided. It should be noted, however, that many programs overlap, particularly the more comprehensive, manualized PT programs. Table 1 provides more detailed information about these PT programs.

Programs Addressing Challenging Problem Behaviors

As already mentioned, research utilizing primarily single-subject designs has demonstrated parents of children with ASD can be taught ABA procedures and techniques to alter their children's targeted problem behaviors such as aggression, self-injury, noncompliance, and other disruptive behaviors. Much of this single-subject literature reported on teaching parents very specific, individualized ABA procedures to target one or two challenging behaviors. More comprehensive PT programs have taught parents and caregivers a range of ABA-based procedures (e.g., antecedent management, reinforcement contingencies, extinction, planned ignoring) to decrease their children's maladaptive behavior and teach new functional and adaptive skills. Two large-scale randomized control trials (RCTs) have provided evidence toward the efficacy of teaching a range of behavioral management

Parent Training, Table 1 Parent training programs

Reference	Participants N and age in months	Target behaviors	Dose of PT	Experimental design	Description
Smith et al. (2000)	N = 6 35–45 months	Children's receptive actions, nonverbal imitation, verbal imitation, and expressive object labels	Six (6 h) 1-day training workshops over 3 months	Multiple baseline across subjects	Taught parents to design and oversee a Lovaas-based discrete trial home-based program
Tonge et al. (2006)	N = 105 30–60 months	Parent mental health and adjustment	Twenty weeks of ten small group (90 min) sessions	RCT	Treatment groups Both manual-based PEBM: Parent education and behavioral management PEC: behavioral management and counseling
Solomon et al. (2008)	N = 19 60–144 months	Child problem and adaptive behaviors, parent stress, and shared positive affect	Two phases: six sessions each	RCT	Parent-Child Interaction Therapy includes two phases: (1) child-directed interaction and (2) parent-directed interaction
Anan et al. (2008)	N = 72 25–68 months	Child cognitive and adaptive functioning	Twelve weeks (180 h total/3 h each session)	Within single subject	Group Intensive Family Training: individual and group sessions with parent-child dyads Parents applied behavior analytic techniques in vivo with child. Staff modeling, coaching, and feedback provided
Whittingham et al. (2009)	N = 59 24–108 months	Parenting measures (i.e., laxness, overreactivity, verbosity, efficacy, satisfaction)	Nine sessions	RCT	Use of Stepping Stones Triple P in combination with ASD-specific interventions (comic strip conversations and social stories)
Nefdt et al. (2010)	N = 27 < 60 months	Parent elicited speech from child, parent confidence, treatment fidelity Child functional utterances		RCT	Self-directed learning model DVDs of PRT techniques
Kasari et al. (2010)	N = 38 21–36 months	Child joint attention, diversity of functional play	Twenty-four sessions, follow-up 1 year prior	RCT	Parent-mediated Joint engagement intervention, individual therapist-led sessions
Dawson et al. (2010)	N = 48 18–30 months	Child developmental levels and adaptive behaviors	Two years	RCT	Delivered by therapists and parents Combination of ABA, PRT, the Denver Model
Green et al. (2010)	N = 152 24–59 months	ASD severity rating Parent-child interaction, child language, adaptive functioning in school	Twice a week (2-h clinic sessions) and 6 months of booster sessions (total 18 sessions)	RCT	Preschool Autism Communication Trial: individualized therapist sessions to address social communication deficits

strategies to parents through a structured, manual-based approach. Sessions were individual and delivered through didactic teaching, use of videos, regular homework assignments, role-playing, and rehearsal with immediate feedback (Aman et al. 2009; Johnson et al. 2007).

A few other programs utilized a combination of individual and group sessions. Whittingham and colleagues (Whittingham et al. 2009) conducted a large study evaluating a PT model that incorporated ABA principles, social learning theory, coercion theory, and ASD-specific interventions (i.e., Social Stories™, Comic Strip Conversations™). This program extended an already established program (i.e., Stepping Stones Triple P) for parents of children with various developmental disabilities for use with parents of children with ASD. Training techniques (i.e., observation, practice, feedback) were employed throughout individual and group sessions to provide instruction in behavioral techniques (e.g., development of positive relationships, management of maladaptive behaviors, planning for high-risk situations, maintenance of behavioral changes). This method of instruction was shown to decrease problem behaviors and improve parenting skills (e.g., less overreactivity, verbosity).

Anan et al. (2008) reported on a program they called Group Intensive Family Training (GIFT). While the PT sessions were administered individually, these were followed by in vivo instruction of parent-child dyads in a group setting. In this case, staff taught parents to work with the other children also enrolled and to extend their use of behavioral principles by training an additional individual (i.e., spouse) to acquire the same skill set. GIFT not only decreased problem behaviors in young children with ASD but also significantly improved their cognitive and adaptive functioning.

Another group of investigators piloted the Parent-Child Interaction Therapy (PCIT) to children with ASD but who were higher functioning (Solomon et al. 2008). PCIT is a well-established, manualized parent training approach for children with early emerging conduct problems (Eyberg et al. 1995) and may hold promise for some parents of children with ASD. In this small pilot study, results reported improvements of parent

rating of child behaviors and improved “shared positive affect.”

Programs Focusing on Core Symptoms and Skill Acquisition

Parent training approaches to target improvements in the three core deficit areas (i.e., communication, social, atypical behaviors) of ASD diagnosis have received considerable research attention. These often include principles of ABA in combination of other theoretical approaches (e.g., developmental, transactional). Generally assumed in these programs is that in order to move children toward a typical developmental trajectory, parents should be taught specific skills to address core deficiencies in social, communication, and/or adaptive functioning. Representative programs are described below.

Smith et al. (2000) earlier investigated the use of group workshops as an effective mechanism to teach parents to design and oversee a Lovaas-based discrete trial home-based program, a commonly implemented treatment approach for this population. Children acquired new skills (i.e., receptive language, expressive language, nonverbal imitation, and verbal imitation) when parents served as therapists. However, the gains were not as substantial as made when the discrete trial therapy was delivered by a therapist.

Koegel and colleagues have developed and tested PT programs targeting what they describe as pivotal areas (i.e., motivation to engage in social communication, self-initiation, self-management, and response to multiple cues) (Schreibman and Koegel 2005). These key areas have been addressed through pivotal response training (PRT), an approach originally designed with a strong emphasis on parent-practitioner collaboration, and have been shown to be an effective intervention for all professionals (e.g., speech language pathologists, behavioral therapists, special educators) working with this population. PRT differs from more behaviorally based approaches due to its unstructured nature that capitalizes on the child’s motivation while embedding natural learning opportunities into daily activities. PRT parent programs have been shown to significantly improve communication and play skills but also a decrease in problem behaviors (Stahmer and Gist 2001).

Green et al. (2010) reported on a large multisite RCT investigation of a PT program called Preschool Autism Communication Trial (PACT). Therapist-guided individualized sessions taught parents to use communication responses and language appropriate for their children's developmental levels. Video feedback and a written summary of key session aims achieved were used to elicit discussion of parental acquisition of skills. Similar to PRT, PACT targeted core deficits in shared attention, form (e.g., nonverbal language, vocalizations) and function (e.g., request, gain attention) of language, and communicative intent. Although significant improvements in the parent-child interaction dynamic (i.e., child initiations, shared attention) were reported, a significant reduction in ASD symptoms was not found.

Other PT programs have focused on instructing and coaching parents how to teach imitation and joint attention given children with ASD do not learn these through typical parent-child interactions. The Early Start Denver Model (ESDM), a combination of PRT, ABA, and the original Denver Model, was reported on as a PT program to teach parents how to elicit critical behaviors (e.g., joint attention, imitation, attention) necessary for the development of functional, purposeful speech in their children. This manualized program includes a PT component along with a therapist-delivered intervention component. In a large RCT, program was shown to improve the child's skills toward a more typical developmental trajectory (Dawson et al. 2010). Similarly, Kasari and colleagues reported on what was described as a parent-mediated intervention to improve joint engagement in a sample of toddlers (Kasari et al. 2010).

Parent Training Programs That Promote Training for Distance Learners

As the prevalence of ASD escalates, so does the need for feasible, accessible training for caregivers living in areas where services for children with ASD are not readily available. The delivery of PT through distance-based telepractice (e.g., video training, phone consultation) has been shown to be a possible alternative to clinic-based

practice. A recent RCT examined self-directed learning through a structured PRT curriculum delivered sequentially through DVD instruction in the home environment. Functional speech and parental confidence in treatment implementation were noted as positive outcome measures in this case (Nefdt et al. 2010). Other programs have been developed but have yet to report on effectiveness. A couple examples can be viewed at www.autismacademy.com and www.simplestepsautism.com.

Efficacy Information

PT as an efficacious intervention depends on the criteria used to define efficacy. While the ABA single-subject literature offers strong support for the use of PT as an intervention for children with ASD, only a few studies have used randomized controlled or controlled trials. Hence, in a systematic review, McConachie and Diggle (2007) concluded that further research is needed and suggested future studies should include adequate sample sizes, use standardized outcome measures, as well as attention to the components of the training. There is an urgent need for more large-scale RCTs using assembled manuals to more fully evaluated PT as well as other behavioral and psychosocial interventions (Smith et al. 2007). However, the need for effective intervention for children with ASD is now. At the time of writing, several other large-scale PT studies are under way and hopefully will begin to fill the gaps in our knowledge.

Outcome Measurement

Measure of outcome in the field of ASD has been fraught with many challenges. For psychosocial interventions, the choice of outcomes is complicated by the frequent multiple or less than exact goals of the intervention, the heterogeneity of children with ASD, the feasibility of completing direct measures with often uncooperative children with ASD, and the desire to have measures blind to the participants and/or investigators (single- or

double-blind studies). In the single-subject literature, the outcome measures have often been highly specific, operationally defined directly observed behaviors (e.g., frequency of use of joint attention, frequency of aggressive behavior, percent compliance). Direct observation methodology has often been used in some larger RCT PT programs. Parent and child are videotaped for later coding of both child and parent behaviors (e.g., Johnson et al. 2009; Kasari et al. 2010).

Broader outcomes incorporated have been behavior rating scale of behaviors completed by an informant (i.e., parent/caregiver, teacher) when the PT is primarily targeting behavior problems. In one large RCT, the Aberrant Behavior Checklist (Aman et al. 1985) was used, given it had previously been shown to be sensitive to treatment change. Global ratings completed by an independent evaluator who is naïve to intervention arm have been suggested as a measure. The Clinical Global Impression Scale (Guy 1976) was used successfully in the RUPP-PI Autism Study (Aman et al. 2009). Measures to assess the presence of core features over time have also been included in a number of PT studies. These have included the Autism Diagnostic Observation Schedule (Lord et al. 2002) and the Autism Diagnostic Interview-Revised (Lord et al. 1994). A wide range of developmental, cognitive, language, and adaptive behavior measures have also been included as outcome instruments. A few examples include the Mullen Scales of Early Learning (Mullen 1995), the MacArthur CDI (Fenson et al. 1993), and the Vineland Adaptive Behavior Scales (Sparrow et al. 2005).

Measures of parent well-being and satisfaction have often been included as outcomes in PT programs. The Parenting Stress Index (Abidin 1995) is a frequently reported parent outcome. Program-specific parent satisfaction measures are often employed as well.

Qualifications of Treatment Providers

The background and training of those providing the parent training have varied in what has been reported in the literature. For those providing

parent training steeped in ABA, a working understanding of these concepts and how they apply to procedures and strategies for children with ASD is essential. While it is imperative to have this conceptual and technical knowledge base, it is also essential that PT providers have the additional therapeutic skills to establish working alliances with parents/caregivers and are able to motivate parents toward change. Hence, PT providers require the therapeutic skills essential for other more traditional therapies. Others have more explicitly described important skills in those providing parent training such as a collaborative style with the parent, ability to provide immediate feedback, and ability to individualize interventions even if the treatment delivered is within a manualized program (Kaiser and Hancock 2003). All in all, an effective PT provider is also an effective clinician in general.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavior Modification](#)
- [Operant Conditioning](#)

References and Reading

- Abidin, R. R. (1995). *Parenting stress index (PSI)*. Odessa: Psychological Assessment Resources.
- Aman, M. G., Singh, N. N., Stewart, A. W., & Field, C. J. (1985). The Aberrant behavior checklist: A behavior rating scale for the assessment of treatment effects. *American Journal of Mental Deficiency, 89*, 485–491.
- Aman, M. G., McDougle, C. J., Scahill, L., Handen, B., Arnold, E., Johnson, C., Stigler, K. A., Bearss, K., et al. (2009). Medication and parent training in children with pervasive developmental disorders and serious behavioral problems. *Journal of the American Academy of Child & Adolescent Psychiatry, 48*, 1143–1154.
- Anan, R. M., Warner, L. J., McGillivray, J. E., Chong, I. M., & Hones, S. J. (2008). Group intensive family training (GIFT) for preschoolers with autism spectrum disorders. *Behavioral Interventions, 23*, 165–180.
- Baker, B. L. (1996). Parent training. In J. Jacobson & J. Mulick (Eds.), *Manual of diagnosis and professional practice in mental retardation* (pp. 289–299). Baltimore: Paul H. Brookes.
- Baker, B. L., Brightman, A., Blacher, J., Heifetz, L., Hinshaw, S. P., & Murphy, D. M. (1997). *Steps to*

- independence: Teaching everyday skills to children with special needs* (3rd ed.). Baltimore: Paul H. Brookes.
- Barkley, R. A., Edwards, G., Laneri, M., Fletcher, K., & Metevia, L. (2001). The efficacy of problem-solving communication training alone, behavior management training alone, and their combination for parent-adolescent conflict in teenagers with ADHD and ODD. *Journal of Consulting and Clinical Psychology*, 69, 926–941.
- Barrett, P. M., & Shortt, A. L. (2003). Parental involvement in the treatment of anxious children. In A. E. Kazdin & J. R. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents* (pp. 101–119). New York: Guilford Press.
- Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., Donaldson, A., & Varley, J. (2010). Randomized, controlled trial of an intervention for toddlers with Autism: The early start Denver model. *Pediatrics*, 125, 17–23.
- Ducharme, J. M., & Drain, T. (2004). Errorless academic compliance training: Improving generalized cooperation with parental requests in children with Autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43, 163–171.
- Eyberg, S. M., Boggs, S. R., & Algina, J. (1995). Parent-child interaction therapy: A psychosocial model for the treatment of young children with conduct problem behavior and their families. *Psychopharmacology Bulletin*, 31, 83–91.
- Fenson, L., Dale, P., Reznick, J. S., Thal, D., Bates, E., Hartung, J., Pethick, S., & Reilly, J. S. (1993). *MacArthur communicative development inventories: User's guide and technical manual*. Baltimore: Brookes.
- Granic, I., & Patterson, G. (2006). Toward a comprehensive model of antisocial development: A dynamic systems approach. *Psychological Review*, 113, 101–131.
- Green, J., Charman, T., McConachie, H., Aldred, C., Slonims, V., Howlin, P., Le Couteur, A., et al. (2010). Parent-mediated communication-focused treatment in children with Autism (PACT): A randomised controlled trial. *Lancet*, 375, 2152–2160.
- Guy, W. (1976). *ECDEU assessment manual for psychopharmacology* (NIMH Publication No. 76–338). Washington, DC: DHEW, NIMH.
- Johnson, C. R., Handen, B. L., Butter, E., Wagner, A., Mulick, J., Sukhodolsky, D. G., et al. (2007). Development of a parent training program for children with pervasive developmental disorders. *Behavioral Interventions*, 22, 201–221.
- Johnson, C. R., Butter, E. M., Handen, B. L., Sukhodolsky, D. G., Mulick, J., Lecavalier, L., Aman, M. G., et al. (2009). Standardized observation analogue procedure (SOAP) for assessing parent and child behavior in clinical trials. *Journal of Intellectual and Developmental Disability*, 34, 1–9.
- Kaiser, A., & Hancock, T. (2003). Teaching parents new skills to support their young child's development. *Infants & Young Children*, 16, 9–21.
- Kasari, C., Gulsrud, A., Wong, C., Kwon, S., & Locke, J. (2010). Randomized controlled caregiver mediated joint engagement intervention for toddlers with autism. *Journal of Autism and Developmental Disorders*, 40, 1045–1056.
- Kazdin, A. E. (2003). Problem-solving skills training and parent management training for conduct disorder. In A. E. Kazdin (Ed.), *Evidence-based psychotherapies for children and adolescents* (pp. 241–262). New York: Guilford Press.
- Lerman, D. C., Addison, L., & Kodak, T. (2006). A preliminary analysis of self-control with aversive events: The effects of task magnitude and delay on the choices of children with Autism. *Journal of Applied Behavior Analysis*, 39, 227–232.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24, 659–685.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2002). *Autism diagnostic observation scale*. Los Angeles: Western Psychological Services.
- Lundahl, B., Nimer, J., & Parsons, B. (2006). Preventing child abuse: A meta-analysis of parent training programs. *Research on Social Work Practices*, 16, 251–262.
- McConachie, H., & Diggle, T. (2007). Parent implemented early intervention for young children with Autism spectrum disorder: A systematic review. *Journal of Evaluation in Clinical Practice*, 13, 120–129.
- Moes, D. R., & Freas, W. D. (2002). Contextualized behavior support in early intervention for children with Autism and their families. *Journal of Autism and Developmental Disorders*, 32, 519–533.
- Mullen, E. J. (1995). *Mullen scales of early learning*. Bloomington: Pearson Assessments.
- Nefdt, N., Koegel, R., Singer, G., & Gerber, M. (2010). The use of a self-directed learning program to provide introductory training in pivotal response treatment to parents of children with Autism. *Journal of Positive Behavioral Interventions*, 12, 23–32.
- Nock, M., & Ferriter, C. (2005). Parent management of attendance and adherence in child and adolescent therapy: A conceptual and empirical review. *Clinical Child and Family Psychology Review*, 8, 142–166.
- Seahill, L. D., Sukhodolsky, D., Bearss, K., Findley, D., Hamrin, V., Carroll, D., & Rains, A. (2006). Randomized trial of parent management training in children with tic disorders and disruptive behavior. *Journal of Child Neuropsychology*, 21, 650–656.
- Schreibman, L., & Koegel, R. L. (2005). Training for parents of children with Autism: Pivotal responses, generalization, and individualization of interventions. In E. D. Hibbs & P. S. Jensen (Eds.), *Psychosocial treatment for child and adolescent disorders: Empirically based strategies for clinical practice* (2nd ed., pp. 605–631). Washington, DC: American Psychological Association.
- Smith, T., Buch, G. A., & Gamby, T. E. (2000). Parent-directed, intensive early intervention for children with

pervasive developmental disorder. *Research in Developmental Disorders*, 21, 297–309.

- Smith, T., Scahill, L., Dawson, G., Guthrie, D., Lord, C., Odom, S., Rogers, S., & Wagner, A. (2007). Designing Research Studies on Psychosocial Interventions in Autism. *Journal of Autism and Developmental Disorders*, 37, 354–366.
- Solomon, M., Ono, M., Timmer, S., & Goodlin-Jones, B. (2008). The effectiveness of parent child interaction therapy for families of children on the Autism spectrum. *Journal of Autism and Developmental Disorders*, 38, 1767–1776.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales* (2nd ed.). San Antonio: Pearson Education.
- Stahmer, A. C., & Gist, K. (2001). The effects of an accelerated parent education program on technique mastery and child outcome. *Journal of Positive Behavior Interventions*, 3, 75–82.
- Symon, J. (2005). Expanding interventions for children with Autism: Parents as trainers. *Journal of Positive Behavior Interventions*, 7, 159–173.
- Tonge, B. J., Brereton, A., Kiomall, M., MacKinnon, A., King, N., & Rinehart, N. (2006). Effects on parental mental health of an education and skills training program for parents of young children with autism: A randomized controlled trial. *Journal of the American Academy of Child Adolescent Psychiatry*, 45, 561–569.
- Webster-Stratton, C., & Taylor, T. (2001). Nipping early risk factors in the bud: Preventing substance abuse, delinquency, and violence in adolescence through interventions targeted at young children (0–8 years). *Prevention Science*, 2, 165–192.
- Whittingham, K., Sofronoff, K., Sheffield, J., & Sanders, M. R. (2009). Stepping stones triple P: An RCT of a parenting program with parents of a child diagnosed with an autism spectrum disorder. *Journal of Abnormal Child Psychology*, 37, 469–480.

Parental Action and Referral Patterns in Spatial Clusters of Childhood Autism Spectrum Disorders

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Synonyms

Disparities in ASD diagnosis

Definition

Spatial clusters of ASD cases were first identified in California, where children born in certain geographic areas had a substantially higher “risk” of diagnosis (i.e., higher per capita rates of ASD) compared to children born elsewhere (Mazumdar et al. 2010). For young children, Liu et al. (2010) even found that the risk of diagnosis increased when their families moved to within closer proximity to already-diagnosed children. Having ruled out environmental causes, the authors hypothesized that “information diffusion” was the cause – essentially, parents of some children were more likely to seek professional medical help if they were exposed to information about ASD (see Fountain et al. 2011). One possibility was that mothers were meeting and talking about their children’s development while they exercised at the mall and waited for their kids at school (e.g., Liu and Bearman 2015).

An alternative to the parental help-seeking hypothesis is that geographic disparities in ASD diagnosis are simply a consequence of physician referral patterns (e.g., Emerson et al. 2016). Kalkbrenner et al. (2011), for example, found that living in close proximity to neurologists and psychiatrists was a good predictor of diagnosis, and it is reasonable to assume that some primary care physicians are more likely than others to refer borderline cases to diagnosing specialists. In Denmark, Lauritsen et al. (2014) found that the risk of diagnosis increased when children’s families moved to more urban areas, where diagnostic services were more readily available.

Because these hypotheses are difficult to test in settings with decentralized diagnosis (e.g., diagnosing physicians everywhere) and extreme variation in access to medical care, Schelly and his colleagues considered the case of Costa Rica. Children in Costa Rica have universal healthcare, and there is one centralized diagnosing clinic. Schelly et al. (2015) started by identifying spatial clusters of ASD that exhibited similar characteristics as those in California (e.g., high socioeconomic status) and elsewhere (e.g., high urbanicity). Next, the authors interviewed parents of diagnosed children to determine what

information they had been exposed to, and what children with ASD they knew, leading to the diagnosis (Schelly et al. 2018). There was little evidence that parents were influencing each other; rather, higher socioeconomic status parents were able to avoid delays in the public healthcare system by paying out of pocket for private specialist care, increasing the likelihood of being referred directly to the diagnosing clinic. Schelly et al. (2019) outline barriers to an information effect that likely also apply in other settings: early symptoms of ASD are often generic, giving parents no reason to suspect ASD in particular, and parents are almost universally aware of early problems and seek medical care for them long before there are opportunities at the mall or in school for other parents to influence them; furthermore, because children with ASD can appear so different – one will stare at walls and another will throw tantrums – knowing a child with ASD may only help in rare cases when the child in question happens to have similar behaviors as one's own.

For now, physician referral behaviors appear to have a stronger influence than parental help seeking behaviors on spatial clusters of childhood ASD, but both mechanisms likely play a role. One solution, though, would alleviate diagnostic disparities no matter the cause: provide better healthcare for all children, and make it easier for all families to access specialist care.

See Also

- [Asperger Syndrome Epidemiology](#)
- [Epidemiology](#)
- [Time Trends in Diagnosis](#)

References and Reading

- Emerson, N. D., Morrell, H. E. R., & Neece, C. (2016). Predictors of age of diagnosis for children with autism spectrum disorder: The role of a consistent source of medical care, race, and condition severity. *Journal of Autism and Developmental Disorders*, 46(1), 127–138. <https://doi.org/10.1007/s10803-015-2555-x>.
- Fountain, C., King, M. D., & Bearman, P. S. (2011). Age of diagnosis for autism: Individual and community factors

- across 10 birth cohorts. *Journal of Epidemiology & Community Health*, 65(6), 503–510. <https://doi.org/10.1136/jech.2009.104588>.
- Kalkbrenner, A. E., Daniels, J. L., Emch, M., Morrissey, J., Poole, C., & Chen, J. C. (2011). Geographic access to health services and diagnosis with an autism spectrum disorder. *Annals of Epidemiology*, 21(4), 304–310. <https://doi.org/10.1016/j.annepidem.2010.11.010>.
- Lauritsen, M. B., Astrup, A., Pedersen, C. B., Obel, C., Schendel, D. E., Schieve, L., et al. (2014). Urbanicity and autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(2), 394–404. <https://doi.org/10.1007/s10803-013-1875-y>.
- Liu, K., & Bearman, P. S. (2015). Focal points, endogenous processes, and exogenous shocks in the autism epidemic. *Sociological Methods & Research*, 44(2), 272–305. <https://doi.org/10.1177/0049124112460369>.
- Liu, K. Y., King, M., & Bearman, P. S. (2010). Social influence and the autism epidemic. *American Journal of Sociology*, 115(5), 1387–1434. <https://doi.org/10.1086/651448>.
- Mazumdar, S., King, M., Liu, K. Y., Zerubavel, N., & Bearman, P. (2010). The spatial structure of autism in California, 1993–2001. *Health & Place*, 16(3), 539–546. <https://doi.org/10.1016/j.healthplace.2009.12.014>.
- Schelly, D., González, P. J., & Solís, P. J. (2015). The diffusion of autism spectrum disorder in Costa Rica: Evidence of information spread or environmental effects? *Health & Place*, 35, 119–127. <https://doi.org/10.1016/j.healthplace.2015.07.007>.
- Schelly, D., González, P. J., & Solís, P. J. (2018). Parental action and referral patterns in spatial clusters of childhood autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(2), 361–376. <https://doi.org/10.1007/s10803-017-3327-6>.
- Schelly, D., González, P. J., & Solís, P. J. (2019). Barriers to an information effect on diagnostic disparities of autism spectrum disorder in young children. *Health Services Research and Managerial Epidemiology*, 6, 1–6. <https://doi.org/10.1177/2333392819853058>.

Parental Attitudes to Technology Use

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Definition

This entry will focus on the attitudes and concerns of parents and caregivers about their autistic child's use of technology. The role of digital

technologies in the lives of children and young people has become a popular debate in recent years, with concerns about the impact of increased media use on a range of developmental domains, including social interaction (Bell et al. 2015). In this entry, the focus will be on technology used by autistic children and young people for leisurely purposes in home environments.

Historical Background

Technology and related topics are recognized as a common interest within the autistic population, and technological skills and affinities are a common stereotype made about autistic people. Baron-Cohen and Wheelwright (1999) asked 92 parents of children with autism about their child's special interests, and 64.1% of them reported that their child had an interest in television or audio media. A more recent survey of 687 autistic adults showed that after autism, computers and gaming were the most popular special interest topics, and especially popular among autistic men (Grove et al. 2018).

Technology develops at such a fast rate that research about the use of technology is quickly outdated, as new devices proliferate the consumer market. For instance, reports which predate the launch of mobile touch screen devices, such as tablet computers and smartphones (e.g., Shane and Albert (2008)), cannot provide insight into current uses of digital media. And likewise, reports which focus on only one device, such as the Apple iPad® (e.g., Clark et al. (2015)), tell us little about what the impact of having multiple devices within households has on parental attitudes to technology and child development.

The term “technology” nowadays refers to a broad range of devices, including different interfaces (screen, touch screen, handheld, robotic), different software (including different types of content, apps), and different contexts of use. By contexts of use, I mean, for example, that in a given morning, we might use our smartphone to check the weather forecast, purchase a bus ticket, listen to music, check emails, pay for a coffee, and text a friend, all before arriving to work for the day. A child might also use a computer “or tablet”

to watch a cartoon, do homework, play a video game, or do their own research about a specific topic. All of these activities and contexts are pulled together when considering “technology use” more broadly, which greatly limits conclusions drawn from sweeping generalisations about technology use.

Current Knowledge

A range of technologies play an important role in the lives of autistic children and young people and are a common reported leisure activity at home (Laurie et al. 2018). The concerns that families report about their autistic child's technology use will be discussed in three domains: screen time, social interaction, and online safety. Screen time concerns are closely related to the repetitive behaviors domain of autism, where parents are concerned about the amount of time that children spend using technology. Concerns about the impact of technologies on social interaction relate to the social differences often observed with autistic children, with the assumption that technology will increase or exacerbate social difficulties over time.

Screen Time

Then time that children spend using digital devices is a highly reported concern, among parents of autistic and non-autistic children alike. Parental concern about the time that autistic children spend using digital media and devices has been shown in a number of reports (Laurie et al. 2018; Mazurek et al. 2012; Mazurek and Wenstrup 2013). Whether autistic children spend more time using technology than non-autistic children is a hotly debated question and has been suggested in some studies which compare the use of technology by autistic teenagers with their non-autistic siblings (Mazurek and Wenstrup 2013). However, a large-scale study which included data from over 65,000 children did not find evidence that screen time in autistic children (aged between 6 and 17 years of age) differed from screen time in non-autistic children (Montes 2016).

Nonetheless, the amount of time that autistic children spend using digital technology at an

individual level is still of concern to parents and families. A majority of parents report using strategies to limit their autistic child's use of technology, including using timetables, timers, and letting children access technology after they have done a certain task, such as homework (Engelhardt and Mazurek 2014). One possible reason is the displacement hypothesis – the prediction that time spent online or using technology is replacing time spent doing other activities, which are presumably healthier, more beneficial, or more productive than technology use. One example of such a concern is that time spent on screens is replacing the time that children spend engaged in outdoor or active play, thus increasing the risk of obesity through sedentary behavior (Must et al. 2014). However, other studies report that autistic children engage in a mixture of both digital and outdoor play (Shane and Albert 2008), therefore suggesting that time spent using digital devices is not replacing time spent on other activities.

Technology and Social Interaction

The social displacement hypothesis also predicts that the more time people spend on the Internet, the less time they spend engaging in face-to-face interactions. Autism is associated with differences in social interest and motivation, which can lead to a preference for solitary activities, such as those traditionally offered through the use of technology (Mazurek and Wenstrup 2013). However, a number of reports have demonstrated the role of seemingly “non-social” technology use in the lives of autistic children. Technology can provide important cultural knowledge for children to connect with their peers, including shared interests in video games or television programs (Fletcher-Watson and Durkin 2015).

One question is whether autistic people's use of technology displaces social interaction in the real world. The “rich get richer” hypothesis predicts that those who are socially popular in offline contexts will use online-mediated communications to enhance their already-existing social relationships and thus benefit more from online communications. Meanwhile, the “social compensation” hypothesis predicts that those who are more introverted or have fewer social relationships will seek out online interactions and therefore benefit

from social connectedness with others as mediated by technology. Early studies supported the rich get richer hypothesis by reporting that autistic teenagers were less likely to use technology for social purposes, including the use of social media (Mazurek and Wenstrup 2013). However, more recent studies have reported that autistic people are active on social media platforms and reportedly prefer to socialize with others over online platforms (Gillespie-Lynch et al. 2014).

Online Safety

The online safety of autistic children is a concern to many parents, particularly of older children who are active Internet users. Just and Berg (2017) report on a series of workshops with parents of autistic children, to identify major concerns that they had about their autistic child's online safety. The concerns that were raised in the workshop included spending money online (e.g., downloading apps, loot boxes, etc.), awareness and understanding online interactions, and accessing inappropriate online material. Some parents reported that their children's motivation to play online games resulted in high amounts of, and sometimes illicit, online transactions. One parent noted that their child was so computer-literate that they could divert any blocks and restrictions put on the account or device and would memorize passwords to access payment details. Another concern reported by parents was their child's social vulnerability transferring into online contexts. Some parents shared that their child had been bullied online, and others said that their child had difficulty managing online risks in relation to social or identifiable information. For instance, some children had no awareness of social privacy and had difficulty recognizing online interactions that were inappropriate (Just and Berg 2017).

Future Directions

Much research has investigated how technology can provide educational and therapeutic benefit for autistic people (Bölte et al. 2010). Increasingly, research is starting to look at the broader role that technology plays in the lives of autistic

people, including the potential opportunities for social connectedness and well-being via use of technology (Stiller et al. 2019). However, parents have also raised a number of concerns about their autistic children's technology use, and research should be responsive to those concerns. This includes evaluating technologies which autistic people are regularly using and focusing on areas of concern for families, including the impact of technology on children's attention, behavior, learning, and interaction.

One challenge for researchers is keeping up to date with the latest digital innovations. On the one hand, research is quickly outdated by technical development, and so research on newly released or up and coming devices is warranted, such as the study of virtual reality and robotics (Good et al. 2016). However, families and caregivers will only benefit at present from evidence on the devices that they are currently using. Parents seldom reported that their autistic child used apps and technologies specifically designed for autistic people and instead the most common types of devices used in the family home were mainstream commercial products (Laurie et al. 2018). There needs to be a balance between preparing for the digital future and also being responsive to current concerns about technology use. One potential solution is research collaborations with individuals and families, to devise and share strategies for managing their concerns about technology. One example of this is the guidelines offered by the National Autistic Society in the UK, based on research and interviews with parents about their autistic child's technology use. Here, the National Autistic Society offers guidance for parents in choosing specific devices for their child and managing their child's use of technology and encourages parents to think about the benefits of technology use for their child (National Autistic Society 2015).

One potential research solution is to identify the digital contexts or features which lead to certain outcomes for autistic people, rather than studying technology at the level of specific devices or apps. This would allow for more general statements about features and contexts, which could be translated across multiple

devices or software, such as screen-based technologies, or apps with this type of interactive mechanism (Fletcher-Watson 2015). For instance, identifying what is it about online games that are appealing to autistic children or moments of technology use where children specifically choose or don't choose to interact with others (Laurie et al. 2019). By identifying the contexts and motivations for autistic people to use technology, better theories can be proposed to explain autism and digital affinity (Laurie et al. 2018). For instance, current theories about digital affinity relate to technical features of technology suiting the learning style for autistic people, e.g., visually presented, predictability, and replicability of digital content (Rajendran 2013; Stiller et al. 2019). Firsthand perspectives from autistic people could provide further insight toward the development of theory in this area. These perspectives could also encourage parents of younger autistic children to think more positively about their child's use of technology or identify ways in which they could support their child to stay safe online.

One final suggestion for future research is to improve representation of non-verbal autistic people, people with learning or physical disabilities, and younger children. Parents of children and young people within these demographics may have different concerns or views about technology use. However, reports of parental concerns are not without their limitations. Parental perspectives are often used in proxy to gather information about technology use from particular groups, such as young children or those with learning disability (Laurie et al. 2018; Mazurek and Wenstrup 2013). Parents' reports of their child's technology use are influenced by their own attitudes about their child's use of digital devices. For instance, parents who reported more concerns about their child's screen time were more likely to report that their child spent more time using technology (Laurie et al. 2018). And when recruiting for research about technology, it is hard to achieve true representation, since those with more extreme (positive or negative) views are more likely to be vocal or responsive to research calls.

See Also

- [Alternative Communication](#)
- [Computer-Assisted Instruction to Teach Academic Skills](#)
- [Video Games and Violence](#)

References and Reading

- Baron-Cohen, S., & Wheelwright, S. (1999). Obsessions? in children with autism or Asperger syndrome. Content analysis in terms of core domains of cognition. *The British Journal of Psychiatry*, 175, 484–490. <https://doi.org/10.1192/bjp.175.5.484>.
- Bell, V., Bishop, D. V. M., & Przybylski, A. K. (2015). The debate over digital technology and young people. *BMJ (Clinical Research Ed.)*, 351, h3064. <https://doi.org/10.1136/bmj.h3064>.
- Bölte, S., Golan, O., Goodwin, M. S., & Zwaigenbaum, L. (2010). What can innovative technologies do for Autism Spectrum Disorders? *Autism: The International Journal of Research and Practice*, 14(3), 155–159. <https://doi.org/10.1177/1362361310365028>.
- Clark, M. L. E., Austin, D. W., & Craike, M. J. (2015). Professional and parental attitudes toward iPad application use in Autism Spectrum Disorder. *Focus on Autism and Other Developmental Disabilities*, 30(3), 174–181. <https://doi.org/10.1177/1088357614537353>.
- Engelhardt, C. R., & Mazurek, M. O. (2014). Video game access, parental rules, and problem behavior: A study of boys with autism spectrum disorder. *Autism: The International Journal of Research and Practice*, 18(5), 529–537. <https://doi.org/10.1177/1362361313482053>.
- Fletcher-Watson, S. (2015). Evidence-based technology design and commercialisation: Recommendations derived from research in education and autism. *TechTrends*, 59(1), 84–88. <https://doi.org/10.1007/s11528-014-0825-7>.
- Fletcher-Watson, S., & Durkin, K. (2015). Uses of new technologies by young people with developmental disorders. *Neurodevelopmental Disorders: Research Challenges and Solutions*, 243–267. <https://www.crcpress.com/Neurodevelopmental-Disorders-Research-challenges-and-solutions/Herwegen-Riby/p/book/9781848723290>. ISBN 9781848723290
- Gillespie-Lynch, K., Kapp, S. K., Shane-Simpson, C., Smith, D. S., & Hutman, T. (2014). Intersections between the autism spectrum and the internet: Perceived benefits and preferred functions of computer-mediated communication. *Intellectual and Developmental Disabilities*, 52(6), 456–469. <https://doi.org/10.1352/1934-9556-52.6.456>.
- Good, J., Parsons, S., Yuill, N., & Brosnan, M. (2016). Virtual reality and robots for autism: Moving beyond the screen. *Journal of Assistive Technologies*, 10(4), 211–216. <https://doi.org/10.1108/JAT-09-2016-0018>.
- Grove, R., Hoekstra, R. A., Wierda, M., & Begeer, S. (2018). Special interests and subjective wellbeing in autistic adults. *Autism Research : Official Journal of the International Society for Autism Research*, 11(5), 766–775. <https://doi.org/10.1002/aur.1931>.
- Just, M., & Berg, T. (2017). Keeping children safe online: Understanding the concerns of Carers of Children with Autism. In R. Bernhaupt, G. Dalvi, A. Joshi, D. K. Balkrishnan, J. O'Neill, & M. Winckler (Eds.), *Human-computer interaction – INTERACT 2017* (Vol. 10515, pp. 34–53). Cham: Springer International Publishing. https://doi.org/10.1007/978-3-319-67687-6_3.
- Laurie, M. H., Warreyn, P., Uriarte, B. V., Boonen, C., & Fletcher-Watson, S. (2018). An international survey of parental attitudes to technology use by their autistic children at home. *Journal of Autism and Developmental Disorders*, 49(4), 1–14. <https://doi.org/10.1007/s10803-018-3798-0>.
- Laurie, M. H., Manches, A., & Fletcher-Watson, S. (2019). Design implications from cognitive event analysis: A case study of digitally mediated interaction in autistic children. *IDC '19: Proceedings of the 18th ACM International Conference on Interaction Design and Children*, 476–481. <https://doi.org/10.1145/3311927.3325325>. <https://dl.acm.org/citation.cfm?id=3311927.3325325>.
- Mazurek, M. O., & Wenstrup, C. (2013). Television, video game and social media use among children with ASD and typically developing siblings. *Journal of Autism and Developmental Disorders*, 43(6), 1258–1271. <https://doi.org/10.1007/s10803-012-1659-9>.
- Mazurek, M. O., Shattuck, P. T., Wagner, M., & Cooper, B. P. (2012). Prevalence and correlates of screen-based media use among youths with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(8), 1757–1767. <https://doi.org/10.1007/s10803-011-1413-8>.
- Montes, G. (2016). Children with autism spectrum disorder and screen time: Results from a large, nationally representative US study. *Academic Pediatrics*, 16(2), 122–128. <https://doi.org/10.1016/j.acap.2015.08.007>.
- Must, A., Phillips, S. M., Curtin, C., Anderson, S. E., Maslin, M., Lividini, K., & Bandini, L. G. (2014). Comparison of sedentary behaviors between children with autism spectrum disorders and typically developing children. *Autism: The International Journal of Research and Practice*, 18(4), 376–384. <https://doi.org/10.1177/1362361313479039>.
- National Autistic Society. (2015). Parents and technology – National Autistic Society. Retrieved August 6, 2019, from <https://www.autism.org.uk/about/family-life/using-technology.aspx>
- Rajendran, G. (2013). Virtual environments and autism: A developmental psychopathological approach. *Journal of Computer Assisted Learning*, 29(4), 334–347. <https://doi.org/10.1111/jcal.12006>.
- Shane, H. C., & Albert, P. D. (2008). Electronic screen media for persons with autism spectrum disorders: Results of a survey. *Journal of Autism and*

Developmental Disorders, 38(8), 1499–1508. <https://doi.org/10.1007/s10803-007-0527-5>.

Stiller, A., Weber, J., Strube, F., & Mößle, T. (2019). Caregiver reports of screen time use of children with autism Spectrum disorder: A qualitative study. *Behavioral Sciences (Basel, Switzerland)*, 9, 5. <https://doi.org/10.3390/bs9050056>.

Parental Caregivers of Adults with Autism

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Definition

A caregiver is a person who provides physical, emotional, social, and financial care or support for another person who is physically, cognitively, or emotionally challenged; has a disability including autism; or a chronic/serious illness that impacts his/her ability to function independently. Caregiving can be temporary or long-term depending on the condition and needs of the individual requiring care. Autism spectrum disorder (ASD) is a continuum that ranges from very mild to very severe, generally is diagnosed in early childhood, and requires reliance on caregivers to help meet daily needs. Caregiving for individuals with ASD typically is provided by parents and begins early in life and continues throughout adulthood. As individuals with ASD age and move into adulthood, they often continue to rely on their parents for care. Parents often provide care until they are no longer able and then caregiving generally falls to siblings, other family members, or close friends. In some cases, the adult with ASD may require formal caregiving either in group homes or through shared living arrangements.

Current Knowledge

During the past two decades, the prevalence of autism spectrum disorder (ASD) has become

a national concern, with the percentage of individuals diagnosed with this condition increasing by 269% (Van Naarden Braun et al. 2015). ASD is a developmental disability that is characterized by impairments in communication and social interaction, as well as restrictive, repetitive behavior (Autism Speaks 2016). Although most individuals with ASD are diagnosed at a young age, they do not outgrow the condition and continue to exhibit symptomology across the lifespan. While the typical belief is that ASD is primarily a childhood condition, an estimated 6.6 million individuals (5.3 million adults and 1.3 million children and adolescents) worldwide have been diagnosed with ASD (Nightingale 2012). Even as adults, individuals with this condition experience a range of impairments that impact functioning with regard to social, communication, and behavioral atypicalities (DaWalt et al. 2017; Leung et al. 2018).

Research on ASD generally has focused on children and their parents, while adults with ASD and their parental caregivers have not been studied to the same extent (Wright et al. 2013). As individuals with ASD transition from childhood to adulthood, the manifestations of their conditions, while changing, can be expected to continue throughout their lives. Many children with ASD have comorbid conditions that can cause greater challenges for caregiving. For example, many children with ASD also have intellectual deficits and mental health issues that complicate the caregiving process (Brondino et al. 2019; Hudson et al. 2019). These comorbid conditions do not go away, but extend across the lifespan.

Many parents can expect to continue to serve as caregivers, varying to some degree, depending on the extent of their adult child's ability to function independently. Caregiving can be burdensome, requiring parents to forego normal aging milestones and causing them to become advocates for obtaining social support services for their adult child with ASD (Marsack and Perry 2018). Understanding their challenges related to realities of caregiving and overall quality of life (QOL) is important as they continue to support their adult children with ASD.

Realities of Caregiving

Caregiving is ongoing and relentless because ASD continues across the lifespan and can pose intergenerational consequences for family members. Regardless of how the caregiver feels, she/he must provide care for their adult child with ASD. They often assist with activities of daily living that could include bathing, feeding, managing atypical behaviors, and helping with money matters. Parents rarely get time off to rest and reflect because they have few people (paid or unpaid) in their lives on whom they can depend to provide the care their adult child needs. Caregiving can feel thankless and exhausting, with many parents overwhelmed by the constantness of caregiving and lack of respite from their responsibilities (Oti-Boadi et al. 2019).

The term “panini sandwich generation” has been used to refer to parents who are caring for multiple generations at the same time (Abramson 2015). As discussed by Marsack and Hopp (2018), some parental caregivers may be responsible for caring for their aging parents, other children with typical and atypical development, spouses as they age, as well as other family members with chronic conditions (e.g., dementia, schizophrenia, or intellectual/developmental disabilities). Caregivers providing care for adults with ASD and other loved ones with varying levels of need may feel particularly “pressed,” while they navigate their own personal and work-related responsibilities.

Caring for an adult child with ASD can result in parents’ loss of identity and inability to have lifetime experiences similar to their same age peers. Parents providing care to an adult with ASD may become isolated from their normally aging friends because they lack time to socialize or find a responsible caregiver (either paid or unpaid) to stay with their adult child for extended periods of time. They are aware that their adult children with ASD may never marry, have children, or live independently. Caregiving becomes burdensome when parents perceive they are missing out on the normal life stages and have to subsume their goals constantly to meet the needs of their adult children with ASD or other family members requiring their attention.

Caregiver Burden

Caregivers’ perceptions of stress and fatigue resulting from ongoing efforts needed to provide physical and emotional support for individuals with conditions that require special care. Four types of caregiver burden, time dependence, financial, developmental, and emotional burdens, often associated with caring for an adult with ASD have been identified in the literature (Novak and Guest 1989). Time required to provide care for an adult with ASD is considered time dependence burden. Parents often postpone or forego participation in personal activities to meet the demands of their adult with ASD (Marsack and Perry 2018). Parents of adults with ASD can expect to experience financial burden due to the costs associated with informal and formal support, as well as lost wages while caregiving (Rogge and Janssen 2019). Developmental burden occurs when the parents of an adult with ASD recognize that their lives are incongruent with peers who have adult children without disabilities (De Fazio et al. 2015). Caregiving can result in emotional burden for parents of adults with ASD, including psychological distress and impacts on psychological health (Herrema et al. 2017; Keenan et al. 2016; Marsack and Samuel 2017). Caregiver burden, regardless of the type, experienced by parental caregivers can have a negative effect on QOL.

Quality of Life

Quality of life is defined by the World Health Organization (WHO) as “individuals’ perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards, and concerns” (WHO 1996, p. 1). QOL is a multifaceted construct comprised of six components: (a) social, (b) physical, (c) psychological, (d) spiritual, (e) cognitive, and (f) environmental well-being (Kelley-Gillespie 2009; Salvador et al. 2014). Each of these components is associated with caregiver burden that parents of adults diagnosed with ASD experience. Individuals self-assess the quality of the interactions between personal and environmental factors to determine their QOL. If parents of adult children perceived less emotional burden (i.e., stress and depression

in their lives), their QOL was likely to be higher. Yoong and Koritsas (2012) used a sample of older parental caregivers of adults with developmental disabilities to study the association between caregiver burden and QOL, based on the WHO definition of QOL. Time needed to provide care for their adult children had a negative effect on parents' participation in leisure activities that impacted their QOL (Yoong and Koritsas 2012).

Informal Social Support

A negative relationship exists between caregiver burden and QOL for parental caregivers who have an informal social support network that helps provide care for the adult child with ASD (Marsack 2016). Informal social support networks generally are comprised of family and friends who provide emotional and physical support in caring for an adult with ASD. For example, a family member could spend an afternoon with the adult with ASD to allow the caregiver to shop, see a physician, or just rest. While parental caregivers continue to age, many will experience a shrinking informal social support network due to death or other naturally occurring reasons (Carstensen 1991). However, these results demonstrate particular vulnerabilities for small, diminishing, or absent informal social support networks or circles. Given the propensity for older adults to experience shrinking networks, it is particularly important to be aware of this tendency.

Formal Social Support

In addition to informal social support, caregivers and adults with ASD may be able to access social support services through formal networks available from government, nonprofit organizations, or community agencies (Roux et al. 2017). Parental caregivers face a myriad of challenges when attempting to coordinate and navigate services as their adult children with ASD transition from adolescence to adulthood (Anderson et al. 2018). While children receive many formal supports through the public education systems in their states at no cost, many of these services have costs associated with them, adding to the financial burden of providing care for adults with ASD (Anderson and Butt 2018; Müller and Cannon

2016). For approximately 25% of parental caregivers, services that could have helped their adult children with ASD were unavailable in their areas, with another 26% unaware of services that could benefit their adult child (Dudley et al. 2019). However, variations in service availability often are based on location (e.g., state-to-state, within-state, rural vs. suburban; Marsack and Perry 2018), with some parents choosing to move to obtain better services for their adult with ASD. Fighting for services and working through bureaucratic systems that were challenging and complex could prove to be too much for some parental caregivers. The inconsistency of some formal supports could be problematic for parents because of high employee turnover, inadequate training, negative attitudes, as well as subpar wages that exist among direct care workers and professional staff. Because of dissatisfaction with, or unavailability of support programs, some parents of adults with ASD used their own resources to develop specialized activities or personally hire and pay staff.

Rewards

Although being a parent of an adult with ASD has been associated with challenges and stressors that add to caregiver burden, many parents also reflect on the rewards resulting from seeing their child make changes, however, small. In recognizing these changes, many parents reported increased spirituality and faith (Pakenham et al. 2004). They have developed greater patience and compassion for others in the community who are experiencing similar caregiving issues. Some parents become active in the community, advocating for greater awareness and improved services for both their adult children, as well as for other parents who are caregiving for adults with developmental disabilities. Some parents consider the reciprocity in care between parents and their adult children with ASD as a reward that reduces the stress associated with caregiving. Both emotional and tangible benefits can emerge from the relationship between the parent and adult child dyad (Perkins and Haley 2010, 2013; Williams and Robinson 2001). Rewards from parenting an adult with ASD can serve as a buffer to reduce stress associated with caregiver burden.

Planning for the Future

In addition to helping their child with ASD become as successful as possible as they become adults, parents also have to consider future and potential plans that will be needed to accommodate their own aging, as well as their adult child's changing needs. Making future plans differs among parents relative to the severity of ASD, family composition, and financial stability. For example, some parents of adults with ASD may have other children who are willing to assume caregiving, while other families may have little or no familial support on which they can rely. Minimal planning by parents is needed if their adult child is high functioning, with more extensive plans required for adult children with ASD who are more impaired or have multiple conditions.

Although parents understand that making plans for the future is necessary, they may struggle with finding a person who is willing to assume responsibilities associated with caring for an adult with ASD at the same level as the parent (Oti-Boadi et al. 2019). Parents may be reticent regarding future planning because they have been busy with today's problems and have not taken the time to consider the need for a comprehensive plan for their adult child's caregiving needs (Bowey and McGlaughlin 2007; Mansell and Wilson 2010).

Most parents are aware that, as they age, providing care for their adult with ASD will become more stressful (Grossman and Webb 2016). They worry about who will provide care for their adult child when they are unable to meet the demands associated with caregiving. In a study by Marsack-Topolewski and Graves (2019), aging parents of adult children with ASD shared a number of future-oriented concerns, including (a) difficulty identifying caregiving support for the future, (b) barriers to making plans/decisions, (c) fear of the unknown, and (d) feeling the need to make plans and decisions now. Many aging parents feel that a new caregiver will encounter difficulties when attempting to navigate the service system for their adult children with ASD, causing concerns that their child will not receive services needed for daily life.

Future Directions

In the upcoming decades, the number of individuals diagnosed with ASD reaching adulthood is anticipated to increase, along with the number of parental caregivers who can expect to continue providing some level of support to their adult children (Gerhardt and Lainer 2011). While these parents have reported many rewards associated with having an adult child with ASD, these parents also face challenges as they continue to age. To diminish effects of caregiver burden, professionals working with parents can help them join or form informal social support groups with others who have an adult child with a disability (Marsack and Church 2019). Practitioners need to understand the complexity associated with caring for an adult with ASD and the needs that change over time, as well as the complexity of family dynamics that effect caregiving.

Maintaining a balance between providing care for the adult child and caring for themselves can become difficult as parents age (Hines et al. 2014). Caregivers need to be encouraged to start making future plans for their adult child, including finding guardians who are willing to assume responsibility for caregiving. At the same time, future planning should provide help for parents as they experience age-related decline. Professionals, such as social workers, case managers, and health professionals, need to help parents develop contingency plans for when they are unable to provide care for their adult with ASD.

See Also

► Family Burden

References and Reading

- Abramson, T. A. (2015). Older adults: The "Panini Sandwich" generation. *Clinical Gerontologist*, 38(4), 251–267. <https://doi.org/10.1080/07317115.2015.1032466>.
- Anderson, C., & Butt, C. (2018). Young adults on the autism spectrum: The struggle for appropriate services. *Journal of Autism and Developmental Disorders*, 48, 1–14. <https://doi.org/10.1007/s10803-018-3673-z>.

- Anderson, C., Lupfer, A., & Shattuck, P. T. (2018). Barriers to receipt of services for young adults with autism. *Pediatrics*, 141(Suppl 4), S300–S305. <https://doi.org/10.1542/peds.2016-4300G>.
- Autism Speaks. (2016). DSM V diagnostic criteria. Retrieved from <https://www.autismspeaks.org/what-autism/diagnosis/dsm-5-diagnostic-criteria>
- Bowey, L., & McGlaughlin, A. (2007). Older carers of adults with a learning disability confront the future: Issues and preferences in planning. *British Journal of Social Work*, 37(1), 39–54. <https://doi.org/10.1093/bjsw/bcl052>.
- Brondino, N., Fusar-Poli, L., Miceli, E., Di Stefano, M., Damiani, S., Rocchetti, M., & Politi, P. (2019). Prevalence of medical comorbidities in adults with autism spectrum disorder. *Journal of General Internal Medicine*, 34, 1–3. <https://doi.org/10.1007/s11606-019-05071-x>.
- Carstensen, L. L. (1991). Selectivity theory: Social activity in life-span context. In K. W. Schaie & M. P. Lawton (Eds.), *Annual review of gerontology and geriatrics* (Vol. 11, pp. 195–217). New York: Springer.
- DaWalt, L. S., Usher, L. V., Greenberg, J. S., & Mailick, M. R. (2017). Friendships and social participation as markers of quality of life of adolescents and adults with fragile X syndrome and autism. *Autism*, 23, 383. <https://doi.org/10.1177/1362361317709202>.
- De Fazio, P., Ciambro, P., Cerminara, G., Barbuto, E., Bruni, A., Gentile, P., et al. (2015). Depressive symptoms in caregivers of patients with dementia: Demographic variables and burden. *Clinical Interventions in Aging*, 10, 1085–1090. <https://doi.org/10.2147/CIA.S74439>.
- Dudley, K. M., Klinger, M. R., Meyer, A., Powell, P., & Klinger, L. G. (2019). Understanding service usage and needs for adults with ASD: The importance of living situation. *Journal of Autism and Developmental Disorders*, 49(2), 556–568. <https://doi.org/10.1007/s10803-018-3729-0>.
- Gerhardt, P. F., & Lainer, I. (2011). Addressing the needs of adolescents and adults with autism: A crisis on the horizon. *Journal of Contemporary Psychotherapy*, 41(1), 37–45. <https://doi.org/10.1007/s10879-010-9160-2>.
- Grossman, B. R., & Webb, C. E. (2016). Family support late in life: A review of the literature on aging, disability and family caregiving. *Journal of Family Social Work*, 19(4), 348. <https://doi.org/10.1080/10522158.2016.1233924>. 2480395.
- Herrema, R., Garland, D., Osborne, M., Freeston, M., Honey, E., & Rodgers, J. (2017). Mental wellbeing of family members of autistic adults. *Journal of Autism and Developmental Disorders*, 47(11), 3589–3599. <https://doi.org/10.1007/s10803-017-3269-z>.
- Hines, M., Balandin, S., & Togher, L. (2014). The stories of older parents of adult sons and daughters with autism: A balancing act. *Journal of Applied Research in Intellectual Disabilities*, 27(2), 163–173. <https://doi.org/10.1111/jar.12063>.
- Hudson, C. C., Hall, L., & Harkness, K. L. (2019). Prevalence of depressive disorders in individuals with autism spectrum disorder: A meta-analysis. *Journal of Abnormal Child Psychology*, 47(1), 165–175. <https://doi.org/10.1007/s10802-018-0402-1>.
- Keenan, B. M., Newman, L. K., Gray, K. M., & Rinehart, N. J. (2016). Parents of children with ASD experience more psychological distress, parenting stress, and attachment-related anxiety. *Journal of Autism and Developmental Disorders*, 46(9), 2979–2991. <https://doi.org/10.1007/s10803-016-2836-z>.
- Kelley-Gillespie, N. (2009). An integrated conceptual model of quality of life for older adults based on a synthesis of the literature. *Applied Research in Quality of Life*, 4(3), 259–282. <https://doi.org/10.1007/s11482-009-9075-9>.
- Leung, R. C., Pang, E. W., Anagnostou, E., & Taylor, M. J. (2018). Young adults with autism spectrum disorder show early atypical neural activity during emotional face processing. *Frontiers in Human Neuroscience*, 12(57). <https://doi.org/10.3389/fnhum.2018.00057>.
- Mansell, I. A. N., & Wilson, C. (2010). ‘It terrifies me, the thought of the future’: Listening to the current concerns of informal carers of people with a learning disability. *Journal of Intellectual Disabilities*, 14(1), 21–31. <https://doi.org/10.1177/1744629510373045>.
- Marsack, C. N. (2016). *An examination of quality of life of parents of adult children diagnosed with autism spectrum disorder*. Detroit: Wayne State University.
- Marsack, C. N., & Hopp, F. (2018). The moderating effect of health between informal social support and caregiver burden among parents of adult children with ASD. *The Gerontologist*, 59, 1112. <https://doi.org/10.1093/geront/gny082>.
- Marsack, C. N., & Perry, T. E. (2018). Aging in place in every community: Social exclusion experiences of parents of adult children with autism spectrum disorder. *Research on Aging*, 40(6), 535–557. <https://doi.org/10.1177/0164027517717044>.
- Marsack, C. N., & Samuel, P. S. (2017). Mediating effects of social support on quality of life for parents of adults with autism. *Journal of Autism and Developmental Disorders*, 47(8), 2378–2389. <https://doi.org/10.1007/s10803-017-3157-6>.
- Marsack-Topolewski, C. N., & Church, H. L. (2019). Impact of caregiver burden on quality of life for parents of adult children with autism spectrum disorder. *American Journal on Intellectual and Developmental Disabilities*, 124(2), 145–156. <https://doi.org/10.1352/1944-7558.124.2.145>.
- Marsack-Topolewski, C. N., & Graves, J. M. (2019). ‘I worry about his future!’: The delay in future planning for adult children with ASD. *Journal of Family Social Work*, 1. <https://doi.org/10.1080/10522158.2019.1578714>.
- Müller, E., & Cannon, L. (2016). Parent perspectives on outcomes and satisfaction levels of young adults with autism and cognitive impairments. *Focus on Autism*

- and Other Developmental Disabilities, 31(2), 92–103. <https://doi.org/10.1177/1088357614528800>.
- Nightingale, S. (2012). Autism spectrum disorders. *Nature Reviews Drug Discovery*, 11, 745–746. <https://doi.org/10.1038/nrd3892>.
- Novak, M., & Guest, C. (1989). Application of a multi-dimensional caregiver burden inventory. *The Gerontologist*, 29(6), 798–803. <https://doi.org/10.1093/geront/29.6.798>.
- Oti-Boadi, M., Asante, K. O., & Malm, E. K. (2019). The experiences of ageing parents of young adults with autism spectrum disorders (ASD). *Journal of Adult Development*, 1–12. <https://doi.org/10.1007/s10804-018-09325-6>.
- Pakenham, K. I., Sofronoff, K., & Samios, C. (2004). Finding meaning in parenting a child with Asperger syndrome: Correlates of sense making and benefit finding. *Research in Developmental Disabilities*, 25(3), 245–264. <https://doi.org/10.1016/j.ridd.2003.06.003>.
- Perkins, E. A., & Haley, W. E. (2010). Compound caregiving: When lifelong caregivers undertake additional caregiving roles. *Rehabilitation Psychology*, 55, 409–417. <https://doi.org/10.1037/a0021521>.
- Perkins, E. A., & Haley, W. E. (2013). Emotional and tangible reciprocity in middle-and older-aged carers of adults with intellectual disabilities. *Journal of Policy and Practice in Intellectual Disabilities*, 10(4), 334–344. <https://doi.org/10.1111/jppi.12061>.
- Rogge, N., & Janssen, J. (2019). The economic costs of autism spectrum disorder: A literature review. *Journal of Autism and Developmental Disorders*, 49, 1–28. <https://doi.org/10.1007/s10803-019-04014-z>.
- Roux, A. M., Rast, J. E., Anderson, K. A., & Shattuck, P. T. (2017). *National autism indicators report: Developmental disability services and outcomes in adulthood*. Philadelphia: Life Course Outcomes Program, A.J. Drexel Autism Institute, Drexel University. Retrieved from <https://drexel.edu/autismoutcomes/publications-and-reports/publications/National-Autism-Indicators-Report-Developmental-Disability-Services-and-Outcomes-in-Adulthood/>
- Salvador, Á., Crespo, C., Martins, A., Santos, S., & Canavarro, M. (2014). Parents' perceptions about their child's illness in pediatric cancer: Links with caregiving burden and quality of life. *Journal of Child and Family Studies*, 24, 1–12. <https://doi.org/10.1007/s10826-014-9921-8>.
- Van Naarden Braun, K., Christensen, D., Doernberg, N., Schieve, L., Rice, C., Wiggins, L., et al. (2015). Trends in the prevalence of autism spectrum disorder, cerebral palsy, hearing loss, intellectual disability, and vision impairment, metropolitan Atlanta, 1991–2010. *PloS one*, 10(4). <https://doi.org/10.1371/journal.pone.0124120>.
- Williams, V., & Robinson, C. (2001). “He will finish up caring for me”: People with learning disabilities and mutual care. *British Journal of Learning Disabilities*, 29, 56–62. <https://doi.org/10.1046/j.1468-3156.2001.00111.x>.
- World Health Organization. (1996). *WHOQOL-BREF: Introduction, administration, scoring and generic version of the assessment*. Geneva: Author. Retrieved from <http://apps.who.int/iris/handle/10665/63529>.
- Wright, S. D., Brooks, D. E., D'Astous, V., & Grandin, T. (2013). The challenge and promise of autism spectrum disorders in adulthood and aging: A systematic review of the literature (1990–2013). *Autism Insights*, 5, 21–73. <https://doi.org/10.4137/AUI.S11072>.
- Yoong, A., & Koritsas, S. (2012). The impact of caring for adults with intellectual disability on the quality of life of parents. *Journal of Intellectual Disability Research*, 56(6), 609–619. <https://doi.org/10.1111/j.1365-2788.2011.01501.x>.

Parental Response to Diagnosis

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Introduction

Autism spectrum disorders (ASD) are a range of neurodevelopmental disorders classified in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V) (American Psychiatric Association 2013) to encompass autistic disorder, Asperger's syndrome, childhood disintegrative disorder, and Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS) (American Psychiatric Association 2000). The disorders have a life-long expectancy and demand for care and treatment, both from parents and professionals. In most Western cultures, children with ASD attend school during daytime, either regular schools with adaptation or specialized schools. Some individuals also do have structured intervention programs implemented at home.

Even if these individuals spend a great amount of time together with professionals, e.g., teachers or other caregivers, parents are spending most time and are thereby considered as the primary caregiver for the individual. The consequences are that parents often experience and bear a great deal of responsibility and care (Volkmar et al. 2014). It should be noted that an ASD diagnosis does not only affect parents but also to a great extent siblings and other close relatives. Lovaas et al. (1973) stated that collaboration between parents and professionals, such as teachers or behavior analysts, is beneficial to the outcome of structured intervention programs. However, this collaboration can be difficult to establish and preserve, often because parents have different prerequisites, and not all parents have financial or nonfinancial resources, which would allow such collaboration.

Parents often start worrying when the child does not make the same milestones as other children, and referral to a specialized clinic is often based upon the concerns parents have when their child shows delay in development of speech and motor skills. Other concerns are often related to stereotypic and/or repetitive behavior (Volkmar et al. 2014). The process of receiving a diagnosis is often a troublesome one, mostly because parents tend to hope that the child will grow out from the delay but also because of long referral time to specialized clinics (Volkmar et al. 2014). Obviously this period can be demanding for parents, due to the amount of concern they encounter and especially due to the waiting time for a clinical assessment.

Post-Diagnosis Emotions

Each parent of a recently ASD-diagnosed child will experience this period in their own unique way; however there are similarities. Previous studies have shown that parents with a recently diagnosed child have elevated levels of depression and even post-traumatic stress disorder (Dabrowska and Pisula 2010; Casey et al. 2012). Others experience a sorrow process including denial, grief, anger, and acceptance (Lutz et al. 2012), while others have a hard time coping with everyday life which often is

experienced as being in “a vacuum,” not knowing what to do. Emotional stress is just one of the consequences parents experience. Mothers, in particular, have an elevated level of emotional stress and depression (Volkmar et al. 2014). One factor that might explain this is that mothers more often take on the role as primary caregiver (Volkmar et al. 2014) and give up their professional career than fathers. There is also a common feeling of disappointment among the parents, not over the child, but over the fact that they are not going to experience all the normal milestones and experiences of a typically developed child (Lutz et al. 2012; Schwartz 2001). As the child grows up and turns into adolescence and young adulthood, a very common concern among parents of individuals with ASD rises, related to the future quality of life of the child if the parents either could not provide the necessary care anymore or if they would pass away (Schwartz 2001; Kalash 2009). The concern is often related to a concern whether or not the child will receive necessary care and support and how this will affect their child’s quality of life.

It is important to address the emotional stress a parent is experiencing when their child is diagnosed with ASD and the related ruminations concerning their child’s future. On the other side, one may not forget the positive sides of being a parent, no matter if your child receives an ASD diagnosis or of another developmental disorder. Parents of children with ASD can also experience a positive effect onto personality and by advocating for their child (Scorgie and Sobsey 2000). Pakenham et al. (2005) described the ASD diagnosis also as exerting a possible positive effect on patience, tolerance, and empathy. From recent studies it emerged that parents of children with ASD also tend to have a great property in caring, sharing, and enjoying the smaller things in life, such as returning a wink or a kiss (Lutz et al. 2012). Many of the parents prefer to speak about their children’s strengths rather than the consequences of the diagnosis, by advocating heavily their personality and interests. Sometimes parents become even motivated by their child to take a degree on the disorder, and some end up as clinicians and researchers. Having a child with an autism spectrum disorder will in most cases

involve heavy workloads, little sleep, and concern for both the present and the future. However, it is also important that parents, clinicians, social workers, and other professionals address the positive sides and encourage parents to enjoy the bright spots and the achievements the child is accomplishing. Having a child with autism can definitely imply joy for the parents.

Reactions to Choice of Treatment and Services

Parents often experience the decision-making process around treatment and services for their child as stressful. Often they feel overstressed when facing all the different treatment and intervention options, which make them feeling unsure about which one is the optimal for their child (Ewart 2002). The different services are provided depending on the location (countries, states, counties, and cities). In addition, there are different procedures on how to obtain these services. Most children with ASD in the United States and other developed countries attend schools with professionals where they get familiar with the staff. However, services such as respite services are in many cases not used, because of the emotional stress the child is experiencing when spending time with a less familiar person. Other parents report that the information from service providers often are lacking, giving a feeling of hopelessness (Kalash 2009). Ewart (2002) regarded obtaining school services and rights as difficult. An example from Norway is that many parents are not familiar with the wide range of services they as a family are entitled to, leaving many with negative attitudes toward specialized clinics and state-funded organizations.

Everyday Life Aspects

As mentioned there are often emotional stress-provoking challenges parents of children with ASD are encountering in terms of adaptation, family life, and also stigmatization when in public with their child. It is also time consuming to

follow up on what the child might have experienced at school, causing parents to customize their life and that of other family members (Lutz et al. 2012). For many parents, it is a struggle just to go to the store with their child, not necessarily because of the child's behavior, but more often because of the stigma felt from other adults (Schwartz 2001). Each parent having a child with ASD will remember the different looks people are throwing (Hunt-Jackson 2007). This can be offending and hurtful situation for parents, making them feel embarrassed, pitiful, or even as if they were bad parents. Many parents experience that the more subtle the public reactions are, the more judgmental they are concerning their child's behavior (Neely-Barnes et al. 2011).

Spousal Relationship

Previous studies indicated that parents of children with ASD had a high rate of divorces. However, more recent studies have shown that despite an increased number of marital conflicts and stress (Benson and Kersh 2011), the divorce rate is much lower than initially found (20 % divorce rate reported by Hartley et al. 2010). Parents often refer to the difficulties of balancing marital needs and the needs of their child. It seems especially hard to combine a marital need with the adjustments and the considerations they have to take concerning their child (Schwartz 2001). Parents also often have problems with maintaining their social network after such a diagnosis (Volkmar et al. 2014). For many it can be rather sore to be around friends with typically developed children, which can cause a marital stress. However, coping with social situations can also strengthen the spousal relationship, which is reflected by many reports about spouses being regarded as the most important support person (Volkmar et al. 2014).

Summary

It is clear that parents of children with ASD experience numerous challenges both as individuals and as couples. Many find the immediate time

after a diagnosis as both emotionally stressful and a grief process comparable to individuals mourning the loss of a close family member. When parents get familiar with their child's diagnosis, there are often positive effects to be observed, such as personal growth and becoming supercharged in advocating both their child in particular and in some cases autism spectrum disorders, in general. Spousal relationships have also been studied, without finding an increased risk of separation or divorce compared to parents of typically developed children. Many parents also regard that they find their spouse as their closest supporter, even if there is an increased marital stress in these marriages.

From the different studies referred to in this chapter, it can be concluded that information about treatment and services should be systemized in a better way, to relieve parents from some of the stress they experience during the time of diagnosis and beyond. Support groups for parents are also a very powerful tool for their marriage, and an increased focus on marital difficulties and issues is warranted. In general, there are challenges and consequences of having a child with ASD, which can be difficult to handle for many individuals. The demands parents are experiencing both concerning to sustain a healthy spousal relationship and a well-functioning family situation often require everyday life to be planned and rigorous. It is therefore important that clinicians, social workers, and other professionals manage to provide parents of a recently diagnosed child with information on both the challenges and possibilities arising in the wake of this event.

References and Reading

- American Psychiatric Association (APA). (2000). *Diagnostic and statistical manual of mental disorders: Text revision*. Washington: American Psychiatric Association.
- American Psychiatric Association (APA). (2013). *Diagnostic and statistical manual of mental disorders 5*. Washington: American Psychiatric Association.
- Benson, P. R., & Kersh, J. (2011). Marital quality and psychological adjustment among mothers of children with ASD: Cross-sectional and longitudinal relationships. *Journal of Autism and Developmental Disorders*, 41(12), 1675–1685. <https://doi.org/10.1007/s10803-011-1198-9>.
- Casey, L. B., Zankas, S., Meindl, J. N., Parra, G. R., Cogdal, P., & Powell, K. (2012). Parental symptoms of posttraumatic stress following a child's diagnosis of autism spectrum disorder: A pilot study. *Research in Autism Spectrum Disorders*, 6, 1118–1193.
- Dabrowska, A., & Pisula, E. (2010). Parenting stress and coping styles in mothers and fathers of preschool children with autism and down syndrome. *Journal of Intellectual Disability Research*, 54, 266–280.
- Ewart, K. H. (2002). Parents' experience of having a child with autism. Doctoral dissertation. Available at: ProQuest dissertations and theses. (Order No. 3062724).
- Hartley, S. L., Barker, E. T., Seltzer, M. M., Floyd, F., Greenberg, J., Orsmond, G., & Bolt, D. (2010). The relative risk and timing of divorce in families of children with an autism spectrum disorder. *Journal of Family Psychology*, 24(4), 449.
- Hunt-Jackson, J. (2007). Finding fathers' voices: Exploring life experiences of fathers of children with autistic spectrum disorders. Available at: ProQuest Dissertations database.
- Kalash, L. A. (2009). Order no. 3406196, The University of North Dakota. ProQuest dissertations and theses, 113. Available at: <http://search.proquest.com/docview/276068082?accountid/414780>
- Lovaas, O. I., Koegel, R. L., Simmons, J. Q., & Long, J. S. (1973). Some generalization and follow-up measures on autistic children in behavior therapy. *Journal of Applied Behavior Analysis*, 3, 131–165.
- Lutz, H. R., Patterson, B. J., & Klein, J. (2012). Coping with autism: A journey toward adaptation. *Journal of pediatric nursing*, 27(3), 206–213.
- Neely-Barnes, S. L., Hall, H. R., Roberts, R. J., et al. (2011). Parenting a child with an autism spectrum disorder: Public perceptions and parental conceptualizations. *Journal of Family Social Work*, 14(3), 208–225.
- Pakenham, K. I., Samios, C., & Sofronoff, K. (2005). Adjustment in mothers of children with Asperger syndrome An application of the double ABCX model of family adjustment. *Autism*, 9(2), 191–212.
- Schwartz, M. (2001). Understanding the well-being of the primary care-takers of autistic children. Available at: ProQuest Dissertations database.
- Scorgie, K., & Sobsey, D. (2000). Transformational outcomes associated with parenting children who have disabilities. *Mental Retardation*, 38(3), 195–206. [https://doi.org/10.1352/0047_6765\(2000\)038<0195:TOAWPC>2.0.CO;2](https://doi.org/10.1352/0047_6765(2000)038<0195:TOAWPC>2.0.CO;2).
- Volkmar, F. R., Paul, R., Rogers, S. J., & Pelphrey, K. A. (2014). *Handbook of autism and pervasive developmental disorders* (Vol. 2). Hoboken: Wiley.

Parent-Implemented Bedtime Fading and Positive Routines

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Definition

Stimulus control is the principle underlying the most common behavioral interventions targeting sleep habits, such as positive routines and bedtime fading. Stimulus control posits that falling asleep is the final step of a behavior chain, in which initial steps are pre-bed behaviors that lead to a state of calmness.

Positive routines are defined as a series of five to seven calming activities that move from active to passive and are undertaken during wakefulness to facilitate quietude and sleep onset. When establishing positive routines, completion of each step in the routine is met by praise, notifying the individual of the transition to the next step.

Bedtime fading is defined as a sleep intervention in which bedtime is moved to match an individual's natural sleep onset time and increase the likelihood of quick sleep onset. Bedtime is then systematically altered over time to more closely approximate developmental norms and family habits.

Historical Background

Good quality sleep is vital for health and overall well-being. Sleep disorders affect 25–40% of children (Owens 2007), most of whom will continue to face sleep difficulties throughout their lifetime if untreated (Mazurek et al. 2019).

Poor sleep negatively impacts on cognitive function, health, behavior, and well-being (Abel et al. 2018). Although quite prevalent in the general population, sleep disorder rates double among

those with developmental disorders, such as autism spectrum disorder (ASD) (Souders et al. 2009). Increased sleep onset latency and night wakings, decreased sleep efficiency, and variable sleep patterns have been identified more frequently among children with ASD in comparison to typically developing peers (Vriend et al. 2011). Children with ASD are affected by additional side effects of poor sleep, among which increased stereotypy, decreased social skills, and increased aggression (Schreck et al. 2004).

Biology is pivotal to our understanding of the causes of sleep difficulties. Finger (1982) posited that all beings follow the circadian cycle of sleep and wake. Research, however, has shown differences between adult and infant sleep with infants experiencing quicker transitions between rapid eye movement (REM) and non-REM stages of sleep (Jin et al. 2013). Studies using electroencephalography have demonstrated a link between REM sleep and wakings. Thus, due to higher frequencies of REM sleep, infants are more likely to wake. These high frequency REM arousals in infants when combined with maladaptive parenting techniques can facilitate the development of sleep difficulties (Anders et al. 1992).

Behavioral interventions have demonstrated success with sleep disorders, with most studies reporting clinically significant improvements (Vriend et al. 2011). Behavioral interventions mostly rely on the principle of stimulus control, defining falling asleep as an operant behavior under the control of discriminative stimuli (e.g., darkness, cool temperatures, and bedding) and sleep as a reinforcer itself (Bootzin 1977).

A central assumption of stimulus control interventions is that if consistent sleep is to occur, steps in the behavior chain that precedes sleep must come under the control of suitable cues or discriminative stimuli (Bootzin 1977). As is the case with any behavior, discriminative properties are obtained by stimuli in the environment which reliably signal the availability of reinforcement, in this case sleep. Motivating operations also play a central role in sleep onset, as they can increase or decrease the value of sleep as a reinforcer and

consequently increase or decrease the likelihood of an individual engaging in behaviors that have previously led to sleep. Relevant motivating operations for sleep are the quality, duration, and time elapsed since previous sleep (Laraway et al. 2003). Knowledge of stimulus control and motivating operations have formed the basis of the most widely used sleep strategies, positive routines, and bedtime fading, which in previous research have generally been combined in multicomponent packages targeting sleep improvements.

Positive routines. Bedtime routines are an easy to implement, low-cost means to improve sleep that reduces non-compliance and increases constructive sleep behaviors (Kodak and Piazza 2008). Positive routines emulate a successful intervention within adult insomnia, namely, stimulus control therapy (Perlis et al. 2011), which states that poor sleep results from absence of stimulus control and therefore establishing strong, appropriate discriminative stimuli for sleep can result in a reduction in sleep disorders. Positive routines consist of a series of five to seven pleasant calming activities undertaken during wakefulness to facilitate sleep onset (Gruber et al. 2011). Studies have noted that routine occurrence rather than content is important (Kodak and Piazza 2008). How distinct and consistent a cue is depends on how often the behavioral sequence is practiced. Through repeated practice of each step in the sequence, the behavioral chain terminating in sleep onset becomes stronger. Additionally, if discriminative stimuli which cue sleep onset are encountered when the child wakes, independent sleep resumption is more likely. Therefore, by training appropriate cues at bedtime, the length of nighttime awakenings may also decrease. Positive routines are in most occasions implemented by caregivers, the primary change agents for children with ASD, who often make use of visual schedules to facilitate understanding and adherence to the positive routine (Honaker and Meltzer 2016; Sanberg et al. 2018).

Bedtime fading. The central aim of bedtime fading is to manipulate the sleep-wake cycle so as to increase the likelihood of sleep. Bedtime fading involves temporarily moving bedtime to more closely coincide with the child's natural sleep

onset and then fading this earlier if sleep onset latency remains short to more closely approximate developmental norms and parental wishes (Morgenthaler et al. 2006). Bedtime fading facilitates rapid sleep initiation preventing inappropriate behaviors from being practiced in bed, as the more inappropriate behaviors are practiced, the more likely they are to access reinforcement. By remaining awake and out of bed longer, a motivating operation emerges which establishes sleep as a potent reinforcer and increases the likelihood of behaviors that facilitate sleep. A scheduled wake time is also established, and sleep is not permitted outside of established sleep times. This further acts to establish sleep as valuable. Faded bedtime has also been noted to closely resemble sleep restriction therapy (Perlis et al. 2011), which has been found effective in reducing adult insomnia.

A number of advantages to bedtime fading have been noted. The intervention is run at a time which decreases the likelihood of parental fatigue and as such minimizes implementation errors (Piazza and Fisher 1991). Further, the likelihood of non-compliant behavior emerging is reduced, as children are only instructed to go to bed once motivation is high. Two biological factors aiding the procedure have been noted. First, increased sleep pressure makes rapid onset more likely. Secondly, a steady bed and wake time may regulate the child's circadian rhythms and enhance synchronization with the desired schedule (Piazza and Fisher 1991).

The use of bedtime fading was first presented by Piazza and Fisher (1989), who discussed its use for a 6-year-old girl with attention deficit hyperactivity disorder who experienced multiple sleep difficulties. The intervention was effective for increasing sleep duration. The first published study occurred at a later date (Piazza and Fisher 1991). Within this, bedtime fading was used for two typically developing children for whom other sleep interventions had been ineffective. Results found the intervention effective in increasing total sleep duration. This study was based on the work of Adams and Rickert (1989) who combined positive routines with a simplistic version of bedtime fading. Piazza and Fisher's (1991) study however removed positive routines and evaluated bedtime

fading alone. Piazza and Fisher (1991) conducted a follow-up study in which bedtime fading and response cost were used to increase sleep duration for four individuals with intellectual disabilities aged between 3 and 19. Response cost is defined as removing the child from their bed for a predetermined period of time if they do not fall asleep within the designated sleep onset latency or if they awake during night. All four participants demonstrated increased nighttime sleep duration and decreased daytime sleep. Similar results have been found by other researchers.

Current Knowledge

In recent years, a significant number of studies have examined the effectiveness of positive routines and bedtime fading.

Positive Routines: Positive routines were first utilized by Milan et al. (1981) as a means to combat negative side effects experienced with extinction. Instead, a fading and chaining intervention was used to alter child sleep behavior. Participants were three children with severe intellectual disabilities. For all participants, the intervention was successful in reducing nighttime tantrums and increasing sleep duration. A second study conducted by Adams and Rickert (1989) compared positive routines with graduated extinction and a control. The dependent variable again was bedtime tantrums. Participants consisted of 36 typically developing toddlers. Similarly to Milan et al. (1981) a bedtime fading component was added though not termed as such. This was the first use of bedtime fading and went on to form the basis of future research. Both treatment groups demonstrated comparable decreases in bedtime tantrums. However faster effects and greater parental satisfaction were found for positive routines compared to graduated extinction.

A number of studies have utilized positive routines within multicomponent interventions implemented by parents in the home environment, combining positive routines with extinction-based strategies or sleep scheduling (e.g., Christodulu and Durand 2004; Knight and Johnson 2014). Other studies have evaluated positive routines in isolation. For example, Mindell et al. (2009) evaluated the

efficacy of positive routines for 405 typically developing toddlers aged between 7 and 36 months. Positive routines were found to be effective at reducing sleep onset latency and night wakings, while a follow-up study found positive outcomes were maintained. A more recent evaluation of the efficacy of positive routines was undertaken by Mindell et al. (2014) who sought to examine the impact of routine “dose” on intervention efficacy. Dose was defined as the amount of routine exposure the child received. This study was cross-cultural with over 10,000 typically developing participants. Positive routines were found effective with routine exposure negatively correlated with bedtime difficulties.

In sum, positive routines are a parent-friendly intervention that allows for individualization, while reducing parental fatigue and increasing implementation accuracy (Kodak and Piazza 2008). Interventions which occur at the child’s bedtime have shown to be more acceptable (Honaker and Meltzer 2016). Bedtime non-compliance has also been noted to decrease as a result of intervention (Morgenthaler et al. 2006). These benefits led Durand (1998) to consider positive routines an errorless approach.

Bedtime Fading. In a further extension of the behavioral literature focusing on sleep, Piazza et al. (1991) evaluated the efficacy of bedtime fading combined with response cost for three children with Rett syndrome aged 4–8 years. All participants demonstrated increased appropriate sleep, decreased daytime sleep, and decreased challenging behaviors. In a further study, a comparison of bedtime fading with response cost and sleep scheduling was also evaluated (Piazza et al. 1997). Results indicated that sleep duration increased more for those in the bedtime fading condition compared to the response cost and sleep scheduling conditions. These studies suggest that bedtime fading combined with response cost is effective in improving overall sleep patterns.

More recent empirical evaluation of bedtime fading is multicomponential in nature. Christodulu and Durand (2004) examined positive routines and bedtime fading for four young children with developmental disorders aged 2–5 years. Results indicated increased total sleep duration for all participants. Vriend et al. (2011) evaluated the efficacy of a multicomponent intervention for three

individuals with ASD and primary insomnia. The intervention consisted of parent-implemented bedtime fading and response cost. Increased sleep efficiency and decreased sleep onset latencies were noted. Results were maintained at a 3-week follow-up. More recently, Sanberg et al. (2018) evaluated the efficacy of bedtime fading combined with response cost on decreasing sleep disturbance in children with ASD. This intervention was found to be effective at reducing sleep onset latency and bedtime resistance. Research to date supports the efficacy of bedtime fading in combination with other interventions. Gradisar et al. (2016) conducted a randomized control trial comparing the efficacy of graduated extinction, bedtime fading, and sleep education on sleep onset latency in infants. Significant improvements were achieved for both graduated extinction and bedtime fading groups. Cooney et al. (2018) investigated the utility of group parent education to train parents in the implementation of bedtime fading for preschool children. Improvements in sleep onset latency night wakings were noted and maintained at a 2-year follow-up.

Limited research has evaluated bedtime fading in isolation. DeLeon et al. (2004) sought to evaluate the efficacy of bedtime fading to reduce night wakings in a 4-year-old boy with ASD. Results noted an 81% reduction in night wakings and an 82% reduction in self-injurious behavior.

To date only one study has compared the efficacy of positive routines versus bedtime fading. Delemere and Dounavi (2017) compared the efficacy of parent-implemented bedtime fading versus positive routines on sleep onset and night wakings of six children with ASD aged 2.5–6.5 years. While positive results were found for both groups, greater gains were noted for participants of the bedtime fading condition.

Future Directions

It is clear that both positive routines and bedtime fading have demonstrated efficacy in decreasing sleep difficulties in children. The apparent efficacy of stimulus control techniques for sleep is beneficial as it minimizes the need to utilize

extinction-based protocols which often present multiple side effects (Jin et al. 2013). Reid et al. (1999) noted a dropout rate of 20% of parent participants from extinction-based interventions due to a reluctance to ignore crying. Stimulus control interventions in comparison do not result in prolonged periods of crying or increased rates of challenging behaviors (Burke et al. 2004). A key advantage of stimulus control interventions is the facilitation of sleep-related skill acquisition by children, in contrast to extinction that does not permit this (Cortesi et al. 2010). Finally, intervention implementation at the child's bedtime is advantageous, as it minimizes parental fatigue increasing implementation fidelity (Kodak and Piazza 2008). The high social validity, treatment fidelity, and acceptability scores obtained across several studies (e.g., Delemere and Dounavi 2017) also support the positive effects of implementing sleep interventions at this time. This suggests that future research on stimulus control interventions rather than extinction-based approaches is warranted.

Functional relations surrounding sleep require further investigation. Functional assessments are the hallmark of behavioral interventions (Hanley et al. 2003). Sleep problems have been exempt from such analyses, although they represent a severe challenge for children and families. Albeit effective interventions have been documented, the conditions under which each strategy should be implemented to maximize benefit have not been established. Jin et al. (2013) reported the efficacy of individualized function-based sleep interventions for young children. The lack of environmental contingency evaluation of participants' sleep difficulties may have adversely impacted results in research to date. Future research should examine the function of sleep problems and use this to guide intervention design and implementation.

References and Reading

- Abel, E. A., Schwichtenberg, A. J., Brodhead, M. T., & Christ, S. L. (2018). Sleep and challenging behaviours in the context of intensive behavioural intervention for children with autism. *Journal of Autism and Developmental Disorders*, 48(11), 3871–3884.

- Adams, L., & Rickert, V. (1989). Reducing bedtime tantrums: Comparison between positive routines and graduated extinction. *Paediatrics*, 84(1), 756–761.
- Anders, T. F., Halpern, L. F., & Hua, J. (1992). Sleeping through the night: A developmental perspective. *Paediatrics*, 90(1), 554–560.
- Bootzin, R. R. (1977). Effects of self-control procedures for insomnia. In R. B. Stuart (Ed.), *Behavioural self-management: Strategies, techniques and outcomes* (pp. 176–196). New York: Brunner/Mazel.
- Burke, R., Kuhn, B., & Peterson, L. (2004). A “storybook” ending to children’s bedtime problems: The use of a rewarding social story to reduce bedtime resistance and frequent night waking. *Journal of Paediatric Psychology*, 29, 389–396.
- Christodulu, K. V., & Durand, V. M. (2004). Reducing bedtime disturbance and night waking using positive bedtime routines and sleep restriction. *Focus on Autism and Other Developmental Disabilities*, 19(1), 130–139.
- Cooney, M. R., Short, M. A., & Gradisar, M. (2018). An open trial of bedtime fading for sleep disturbances in preschool children: A parent education approach. *Sleep Medicine*, 46, 98–106.
- Cortesi, F., Giannotti, F., Ivanenko, A., & Johnson, K. (2010). Sleep in children with autism spectrum disorder. *Sleep Medicine*, 11, 659–664.
- Delemere, E., & Dounavi, K. (2017). Parent-implemented bedtime fading and positive routines for children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 48(4), 1002–1019.
- DeLeon, I., Fisher, W., & Marhefka, J. (2004). Decreasing self-injurious behaviour associated with awakening in a child with autism and developmental delays. *Behavioral Interventions*, 19(2), 111–119.
- Durand, V. M. (1998). *Sleep better! A guide to improving sleep for children with special needs*. Baltimore: Brookes.
- Finger, F. W. (1982). Circadian rhythms: Implications for psychology. *New Zealand Psychologist*, 11(1), 1–12.
- Gradisar, M., Jackson, K., Spurrier, N. J., Gibson, J., Whitman, J., Williams, A. S., Dolby, R., & Kennaway, D. J. (2016). Behavioural interventions for infant sleep problems: A randomised controlled trial. *Pediatrics*, 137(6), e20151486.
- Gruber, R., Cassoff, J., & Knäuper, B. (2011). Sleep health education in paediatric community settings: Rationale and practical suggestions for incorporating healthy sleep education into paediatric practice. *Paediatric Clinics of North America*, 58(1), 735–754.
- Hanley, G. P., Iwata, B., & McCord, B. (2003). Functional analysis of problem behavior: A review. *Journal of Applied Behavior Analysis*, 36(2), 147–185.
- Honaker, S. M., & Meltzer, L. J. (2016). Sleep in pediatric primary care: A review of the literature. *Sleep Medicine Reviews*, 25, 31–39.
- Jin, C. S., Hanley, G. P., & Beaulieu, L. (2013). An individualised and comprehensive approach to treating sleep problems in young children. *Journal of Applied Behavior Analysis*, 46(1), 161–180.
- Knight, R. M., & Johnson, C. M. (2014). Using a behavioural treatment package for sleep problems in children with autism spectrum disorders. *Child and Family Behavior Therapy*, 36(3), 204–221.
- Kodak, T., & Piazza, C. C. (2008). Assessment and behavioural treatment of feeding and sleeping disorders in children with autism spectrum disorders. *Child and Adolescent Psychiatric Clinics of North America*, 17(4), 887–905.
- Laraway, S., Snyckerski, S., Michael, J., & Poling, A. (2003). Motivating operations and terms to describe them: Some further refinements. *Journal of Applied Behavior Analysis*, 36(3), 407–414.
- Mazurek, M. O., Dovgan, K., Neumeyer, A. M., & Malow, B. A. (2019). Course and predictors of sleep and co-occurring problems in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49(5), 2101–2115.
- Milan, M. A., Mitchell, Z. P., Berger, M. I., & Pierson, D. F. (1981). Positive routines: A rapid alternative to extinction for eliminative of bedtime tantrum behaviour. *Child Behavior Therapy*, 3(1), 13–25.
- Mindell, J. A., Telofski, L. S., Wiegand, B., & Kurtz, E. S. (2009). A nightly bedtime routine: Impact on sleep in young children and maternal mood. *Sleep*, 32(1), 599–606.
- Mindell, J. A., Li, A. M., Sadeh, A., Kwon, R., & Goh, D. Y. (2014). Bedtime routines for young children: A dose dependent association with sleep outcomes. *Sleep*, 38(5), 717–722.
- Morgenthaler, T. I., Owens, J., Alessi, C., Boehlecke, B., Brown, T. M., Coleman, J., Friedman, L., Kapur, V. K., Lee-Chiong, T., Pancer, J., & Swick, T. J. (2006). Practice parameters for behavioural treatment of bedtime problems and night wakings in infants and young children. *Sleep*, 29(10), 1277–1281.
- Owens, J. A. (2007). Classification and epidemiology of childhood sleep disorders. *Sleep Medicine Clinics*, 2(1), 353–361.
- Perlis, M., Aloia, M., & Kuhn, B. (2011). *Behavioural treatments for sleep disorders, A comprehensive primer of behavioural sleep medicine interventions*. Oxford, UK: Academic.
- Piazza, C. C. & Fisher, W. (1989). Utilizing behavioural principles and circadian rhythms in the treatment of paediatric sleep disorders: The faded bedtime protocol with response cost. Paper presented at the 15th annual convention of the Association for Behavior Analysis, Milwaukee.
- Piazza, C. C., & Fisher, W. (1991). A faded bedtime with response cost protocol for treatment of multiple sleep problems in children. *Journal of Applied Behavior Analysis*, 24(1), 129–140.
- Piazza, C. C., Fisher, W., & Moser, H. (1991). Behavioural treatments of sleep dysfunction in children with the rett syndrome. *Brain and Development*, 13(1), 232–237.
- Piazza, C. C., Fisher, W., & Sherer, M. (1997). Treatment of multiple sleep problems in children with developmental disabilities: Faded bedtime with response cost

- versus bedtime scheduling. *Developmental Medicine and Child Neurology*, 39(1), 414–418.
- Reid, M. J., Walter, A. L., & O’Leary, S. G. (1999). Treatment of young children’s bedtime refusal and night-time wakings: A comparison of “standard” and graduated ignoring procedures. *Journal of Abnormal Child Psychology*, 27(1), 5–16.
- Sanberg, S. A., Kuhn, B. R., & Kennedy, A. E. (2018). Outcomes of a behavioural intervention for sleep disturbances in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(12), 4250–4277.
- Schreck, K. A., Mulick, J. A., & Smith, A. F. (2004). Sleep problems as possible predictors of intensified symptoms of autism. *Research in Developmental Disabilities*, 25(1), 57–66.
- Souders, M. C., Martin, T. B., Valladares, O., Bucan, M., Levy, S. E., Mandell, D. S., Weaver, T. E., & Pinto-Martin, J. (2009). Sleep behaviours and sleep quality in children with autism spectrum disorders. *Sleep*, 32(12), 1566–1578.
- Vriend, J. L., Corkum, P. V., Moon, E. C., & Smith, I. M. (2011). Behavioural interventions for sleep problems in children with autism spectrum disorders: Current findings and future directions. *Journal of Paediatric Psychology*, 36(9), 1017–1029.

Parent-Professional Collaboration

► Parent-Professional Partnership

Parent-Professional Partnership

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Synonyms

Family-centered programming; Parent involvement; Parent-professional collaboration

Definition

A Parent-Professional Partnership is a collaborative relationship between the parent of a child with

a disability and a professional who is involved with helping that child. Educators are the professionals most frequently thought of when referencing a Parent-Professional Partnership. However, any professional who provides a direct or indirect service to the child with the disability or to the child’s family, or who is involved in the policies affecting the child, can be a part of this collaborative partnership. In addition to educators, other professionals with whom a parent may form a partnership include policy makers, trainers, and therapists.

The Individuals with Disabilities Education Improvement Act of 2004 (IDEIA) specifically endorses partnerships between parents and educators. The IDEIA encourages parents to be actively involved in their children’s education and empowers them with rights and responsibilities with respect to the assessment process and the development of the individualized education program. In particular, the early intervention system requires a family-centered approach that takes family values and priorities into account, which necessitates the development of a Parent-Professional Partnership.

Parent-Professional Partnerships exist in many venues outside the education setting as well. For example, partnerships are frequently formed between parents and professionals delivering therapies and services outside of the education setting (e.g., through a state’s mental health or behavioral health system or through private practice). Parent-Professional Partnerships may also be created to train teachers, staff, or college students pursuing a career in special education. On a larger scale, federal, state, and local agencies or advisory groups also rely on Parent-Professional Partnerships by having representation from both professionals and parents who work collaboratively to set policies for children with disabilities. For example, every state has a special education advisory committee (SEAC), and many of these committees exist at the local level as well.

Effective Parent-Professional Partnerships are based on open communication, commitment, trust, sensitivity, and mutual respect. Both sides must recognize the importance of what the other can contribute. While professionals bring expertise on interventions, resources, specialized

instruction, and available programming, parents are the experts of their children. While professionals often see the child in only one environment, parents are frequently accustomed to dealing with multiple service systems and have the benefit of knowing what has been successful or unsuccessful in the past. Parents can also facilitate communication among service providers, thus assisting with continuity and convergence of services.

Obstacles to the Parent-Professional Partnership include attitudinal barriers, socioeconomic and cultural barriers, communication barriers, and fear. Yet the benefits of an effective Parent-Professional Partnership outweigh the difficulties in overcoming these hurdles: Research has shown that family involvement is one of the most important factors in ensuring a child's success. Parent training organizations, support groups, and resource centers are available to help prepare parents for this collaborative role, and increasingly professional organizations are devoting time to train their constituents to work as partners with parents.

See Also

- [Advocacy](#)
- [Early Intervention](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Parent Training](#)

References and Reading

- Blue-Banning, et al. (2004). Dimensions of family and professional partnerships: Constructive guidelines for collaboration. *Exceptional Children*, 70(2), 167–184. Retrieved from http://cms-kids.net/providers/early_steps/training/documents/dimensions_of_family.pdf
- Edmondson, R. (n.d.). Parent-professional partnerships in early intervention. In Pierce, P. (Ed.), *Baby power: A guide for families for using assistive technology with their infants and toddlers*. Chapel Hill: The Center for Literacy and Disabilities Studies, University of North Carolina. Retrieved from http://www2.edc.org/NCIP/library/ec/power_2.htm
- Far North Family Support Council. (2004). *Far north parent-professional partnerships: Building parent-professional partnerships, understanding basic*

- systems, best outcomes – Person-centered and self-directed*. Retrieved from <http://www.alaskachd.org/products/partnership/content/pdfs/print.pdf>
- Individuals with Disabilities Education Improvement Act of 2004, 20 U.S.C. §§ 1400 et seq.
- Parents Make the Difference. Retrieved from <http://www.nhparentsmakethedifference.org/research.htm>
- Technical Assistance ALLIANCE for Parent Centers. (2008). *Fostering parent and professional collaboration: Research brief*. Retrieved from <http://www.parentcenternetwork.org/assets/files/Parent%20and%20Professional%20Collaboration%20Research%20Brief%20-%20Final.pdf>

Parents' Use of Internal State Language with Toddlers with ASD

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Definition

Internal state language (ISL): Internal state language is vocabulary used to convey others' or one's own inner perceptions, desires, emotions, and cognitions; also referred to as mental states (Baixauli et al. 2016). ISL includes several categories, including terms referring to perception, physiology, emotion/affect, volition/ability, cognition, and moral judgment/obligation. Examples of categories of ISL, adapted from Bretherton and Beeghly (1982), are below in tabular form (Table 1). ISL is frequently studied in relation to theory of mind and emotion socialization in childhood (Bretherton and Beeghly 1982).

Historical Background

Internal State Language (ISL) is pervasive in human conversation. Parents label and comment on their children's internal states starting in infancy (Meins et al. 2001). As they grow,

Parents' Use of Internal State Language with Toddlers with ASD, Table 1 Categories and exemplars of ISL

ISL category	Words	Examples of ISL verbatim productions from children
Perceptual	See, look, watch, hear, listen, taste, smell, feel, hurt	"I'm going to be a cloud in the sky so you can't see me."
Physiological	Hungry, thirsty, sleepy, tired, sick	"Me not sleepy, mommy."
Emotional/affective	Happy, fun, proud, feel, better, good, like, love, surprised, sad, angry, scared, messy	"Santa will be happy if I pee in the potty."
Volition/ability	Want, need, have to, can	"My baby needs me."
Cognition	Know, think, remember, forget, understand, guess	"Dolly knows where it is."
Moral judgment/obligation	Good, bad, naughty, may, let, must, should, have to	"Matthew won't let me play!"

children are continuously exposed to ISL in conversation with parents, siblings, and peers. This is an important factor for children's social-emotional development (Tompkins et al. 2018).

Children begin to speak about mental states usually in the second postnatal year (Bretherton and Beeghly 1982). By 2 years of age, children refer to a range of feeling states in themselves and in others and discuss the causes of characters' feelings in book reading, pretend games, and conversations with others (Dunn et al. 1987). Talk about cognitions, desires, and feelings increases between ages 2 and 4 years, with cognitive talk continuing to increase from 4 to 6 years of age (Jenkins et al. 2003), while other ISL remains consistent or slightly declines.

Understanding and talking about mental states is critical for many developmental skills, including sharing, understanding emotions, prosocial behavior, and theory of mind (Brownell et al. 2013). Observational studies conducted with young, typically developing children indicate

that increases in internal state labelling are associated with greater cooperation, conciliatory behavior with siblings (Dunn and Munn 1986), and greater concern and attention to adult distress and emotions (Garner 2003).

In addition to children's ISL, researchers have investigated parents' ISL. The two are related, such that parents who use a lot of ISL in conversation have children with relatively advanced ISL (Taumoepeau and Ruffman 2008). Parents' ISL also predicts the child's later theory of mind, even when controlling for language, age, parent education, and verbosity (Ruffman et al. 2002).

Given the strong predictive power of parents' ISL to children's social-emotional development, it is understandable that parental ISL would be of substantial interest in the study of Autism Spectrum Disorder. A toddler's use of internal state language is reflective of their theory of mind (Capps et al. 1999), that is, understanding other people as separate beings with their own thoughts, feelings, and concerns. However, children with ASD demonstrate difficulties with ISL: Children with autism are less likely to identify causes of characters' internal states or to elaborate on internal emotions or cognitions (Capps et al. 1999). Since core deficits of ASD include social-communication skills and theory of mind, ISL provides an interesting vantage point for the study of the development of theory of mind and social-communication skills in this clinical population.

Current Knowledge

Do Parents of Children with ASD Produce Less Internal State Language?

Some work has noted that parental ISL use with children with ASD is qualitatively and quantitatively different than with typically developing (TD) children. For instance, in a case study conducted with a 43-month-old female with ASD, parental language was recorded and analyzed over a 3-day period. Results indicated that the mother and father used ISL terms in 33% and 24% of their utterances, respectively (Kay-Raining Bird et al. 2008). These values are

compared to other studies reporting percentages of ISL ranging from 33% to 60% for typically developing toddlers (Beeghly et al. 1986). In the case study, the most prominent ISL categories included sensory and desire utterances, which occurred more frequently than cognitive ISL categories. Parental elaboration or use of causal language – such as explaining why a story character may have felt a particular way – was minimal. Thus, both parents in this case used ISL less frequently and less explicitly than a parent of a TD child of the same age. This suggests that parental use of ISL may be qualitatively and quantitatively different for children with ASD, involving less ISL speech overall and fewer causal explanations.

However, other evidence suggests that there may be more similarities than differences in parental ISL towards children with ASD. For instance, during book reading with children ages 4–9 years of age, mothers of ASD-diagnosed and TD children produced narratives of similar length, similar numbers of word tokens, and were similar in the number of times they mentioned cognition, affect, and perception/attention (Slaughter et al. 2007). However, more subtle differences emerged when looking at how ISL was elaborated and clarified in these samples. Mothers of the ASD group produced significantly fewer clarifications and elaborations of cognition and affect terms. For instance, they were less likely to explicitly state the thoughts of characters, including explaining the source of information or noting discrepancies in how two different characters were thinking. After controlling for verbal mental age, maternal education, and maternal verbosity, clarification of affect was associated with theory of mind in the ASD group. These results suggest that while the length of utterances and communicative interchanges by parents to their children with ASD may be equivalent to those of TD peers, the content may be different. Parents of children with ASD may be less likely to elaborate and explain characters' inner thought and feelings.

These findings and conclusions were replicated in work by Hutchins et al. (2017), who compared story narratives of mothers of TD children to mothers of children with ASD (ages

4–11 years). Similar to Slaughter et al. (2007), the authors found that mothers did not differ in the overall amount of ISL talk, but that TD mothers made more causal and clarifying statements connecting internal states to external behaviors. In both groups, mothers who used more cognitive and affective clarifications rated their children as having better theory of mind than mothers who used less ISL. These results seem to confirm that children with ASD are exposed to similar amounts of ISL as their TD peers, but they hear fewer explanatory or clarifying statements about internal states. This could be an important area for intervention since children with ASD may particularly benefit from explanatory, clarifying talk about internal states of characters during play and reading (Slaughter et al. 2007).

However, it may be how parents *elicit* children's talk about emotions, more so than parental production of ISL, which influences children's social behavior (Brownell et al. 2013). For example, in a wordless picture book reading task with toddlers, Campbell et al. (2019) investigated parental ISL to three groups: infants at high risk for developing ASD (as indicated by having an older sibling with an ASD diagnosis) but who did not go on to develop ASD, infants at high risk who went on to develop ASD, and infants at low risk for developing ASD. Their results were consistent with other findings in that parents in all three groups used similar frequencies of overall ISL at 22 and 34 months of age. However, parents of children with ASD demonstrated less ISL elicitation from their infants than parents of low risk or high risk, non-ASD children. Thus, perhaps the difference-maker in these parent-child interactions is not the quality or quantity of speech input to the child but how well parents are drawing out ISL from their children. This is a promising area for future study.

For TD children, the quantity and quality of parental ISL influences a child's theory of mind and social cognition (Taumoepeau and Ruffman 2006). It is not yet clear if the same holds true for children with ASD. Mixed results indicate that theory of mind training – which typically incorporates internal state language – may, or may not, be effective for individuals with ASD (e.g.,

Fletcher-Watson et al. 2014). Although it stands to reason that exposure to explanatory ISL speech may improve ASD children's theory of mind, there has been no direct research on this topic. The efficacy of parent training to improve ISL, and its subsequent impact on theory of mind and/or social behaviors for children with ASD, are therefore promising areas for future research.

What Factors Influence Parental ISL Production?

Parental ISL production may vary depending on many characteristics, including the parent's own proclivities and preferences, their child's developmental abilities, and parental beliefs about their child's development (Beeghly et al. 1986). Parental beliefs, in particular, may be linked to how mothers and fathers parent their young children. Mothers with more complex beliefs about toddler and child knowledge (complex epistemologies) reportedly use mental state language more often to encourage child reflection, which results in greater frequency of mental state terms used by children (Hutchins et al. 2009). Similarly, parents of children with ASD reportedly use similar numbers of mentalistic descriptors when asked to describe their child, as compared to parents of TD children. However, the quality of those descriptors is significantly different (e.g., more negative; Kirk and Sharma 2017). Here again, parents of children with ASD seem to use similar amounts of mentalistic descriptors as TD parents, but those verbalizations are qualitatively different.

Parental ISL may also depend on the perceived conversational readiness of the child. Results from Slaughter et al. (2009) revealed that the earlier infants produced imperative gestures, the more frequently their mothers referenced internal, volitional states at 15 months of age. These results indicate that maternal communication about infants' wants and desires is linked to their infant's production of communicative gestures (Slaughter et al. 2009). This linkage of infant gestures to parental ISL is particularly interesting in the realm of ASD, as children with ASD typically produce fewer gestures and a decreased variety of gestures during parent-child interactions (Mastrogiuseppe et al. 2014). Therefore, infants

at greater risk for developing ASD may gesture less, reducing their parents' opportunities to use internal state language.

Another interesting interaction is present when considering the openness to emotions and emotionality of the parent. Previous works have indicated that variance in emotion-related socialization behaviors of parents – specifically the use of ISL – was explained partially by openness to emotional processes and children's personality (Mazzone and Nader-Grosbois 2017). Given the Broader Autism Phenotype account of Autism Spectrum Conditions (e.g., Wheelwright et al. 2010), parents of children with ASD may possess traits suggesting genetic liability to autism, including less emotional openness or a decreased ability to label emotions in others. As such, parents of children with ASD may naturally use less ISL in everyday interactions, including interactions with their children.

Lastly, there are demographic factors that impact parental ISL use with toddlers and children. One such factor is sex of the speaker and of the child. Although almost all research has been conducted with mothers, some work has looked into parental ISL by mothers *and* fathers to investigate the role of the sex of the caregiver. With typically developing toddlers, Jenkins et al. (2003) highlighted that mothers spend a higher proportion of their time talking about internal mental states than fathers. This finding held across all ISL categories. Furthermore, different caregivers may utilize ISL in different ways. LaBounty et al. (2008) found that each caregiver's use of ISL predicted unique constructs concurrently and later in a TD-child's life. While mothers' references to emotion and emotion causal explanatory language predicted children's concurrent emotion understanding, fathers' use of causal explanatory language referring to desires and emotions predicted children's concurrent and later theory of mind. The way in which mothers and fathers engage with ISL may also have gendered transmission to children. For instance, mothers and older siblings mention feeling states more frequently to TD girls than to TD boys. Accordingly, by 24 months of age, TD girls refer to their own feeling states significant more than

TD boys (Dunn et al. 1987). These sex differences have only just begun to be explored in ASD. One study found that girls with ASD verbalize internal states more often than boys with ASD, but that both girls and boys with ASD fall behind TD children in production of affective state words and ISL (Kauschke et al. 2016). The sex differences in parental ISL production and parent-child transmission of ISL is a significant future direction of research given the sex diagnostic discrepancy in ASD.

Summary

Parental internal state language is an important type of communication that contributes to a child's developing emotion socialization and theory of mind. ISL is used in upwards of one third of parental vocalizations to TD toddlers and children and may even be upwards of 50% of total vocalizations in book-reading tasks (e.g., Beeghly et al. 1986). However, it is not yet clear if these frequencies hold among parents with children with ASD. While some case studies suggest that the quantity and quality of parental ISL may be lower in families with a child with ASD, other observational studies suggest that parents of ASD children may, in fact, use the same number of utterances and ISL words. The differences, however, in ASD families are how explicitly parents label ISL and elaborate or clarify their ISL statements. While parents of TD children tend to use causal and explanatory ISL language with their children, parents with ASD children do not engage in the same practice of being explicitly causal when talking about internal states, even though children with ASD might benefit from such talk. These differences in parental ISL use may be due to many factors, including parental factors (e.g., parenting beliefs, epistemology, emotional availability, and gender) and child factors (e.g., communicative readiness, gestural production, developmental ability, personality, and gender). Importantly, parental ISL production is likely an interaction of parent and child factors that evolve and change over the course of development. Given the established connections

between ISL and socio-emotional outcomes such as theory of mind and certain social skills in TD children, parental ISL toward children with ASD is an important avenue for continued and future research.

Future Directions

Parental ISL is an under-studied area in the fields of clinical psychology and social-emotional development. Many avenues are pressing for future research, including paternal transmission of ISL (e.g., focusing on fathers' ISL use and the different types of activities that stimulate mothers and fathers to produce ISL spontaneously), sex differences in ISL within ASD samples (e.g., parental ISL to sons versus daughters, sex differences in the processing and use of ISL), and closer analyses of differences in ISL use by parents of children with ASD. While a few studies have used book-reading or structured play tasks to elicit ISL, future work should aim to increase external validity by investigating ISL in school settings, social settings with peers, and naturalistic family settings. These settings may include a variety of conversational partners, such as teachers, grandparents, and siblings; all of whom are understudied in their contributions to ISL for children with ASD. Future longitudinal work should also aim to better understand the developmental course and causal pathways of parental ISL. For instance, if gestural use by the infant is a signal to parents to commence using mental state language, this is a hypothesis that could be tested longitudinally. Finally, careful investigation of associations between exposure to ISL and social-cognitive outcomes for children with ASD may inform novel ISL-targeted interventions for this clinical group.

See Also

- ▶ [Figurative Language](#)
- ▶ [Language](#)
- ▶ [Language Acquisition](#)
- ▶ [Metaphoric Language](#)
- ▶ [Narrative Language in ASD](#)

References and Readings

- Baixauli, I., Colomer, C., Rosello, B., & Miranda, A. (2016). Narratives of children with high-functioning autism spectrum disorder: A meta-analysis. *Research in Developmental Disabilities*, 59, 234–254. <https://doi.org/10.1016/j.ridd.2016.09.007>.
- Beeghly, M., Bretherton, I., & Mervis, C. B. (1986). Mothers' internal state language to toddlers. *British Journal of Developmental Psychology*, 4, 247–261. <https://doi.org/10.1111/j.2044-835X.1986.tb01016.x>.
- Bretherton, I., & Beeghly, M. (1982). Talking about internal states: The acquisition of an explicit theory of mind. *Developmental Psychology*, 18, 906–921. <https://doi.org/10.1037/0012-1649.18.6.906>.
- Brownell, C. A., Svetlova, M., Anderson, R., Nichols, S. R., & Drummond, J. (2013). Socialization of early prosocial behavior: Parents' talk about emotions is associated with sharing and helping in toddlers. *Infancy*, 18, 91–119. <https://doi.org/10.1111/j.1532-7078.2012.00125.x>.
- Campbell, S. B., Mahoney, A. S., Brownell, C. A., Moore, E. L., & Tavares, A. B. (2019). Parents' use of internal state language with toddlers at high and low genetic risk for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49, 1366–1377. <https://doi.org/10.1007/s10803-018-3839-8>.
- Capps, L., Losh, M., & Thurber, C. (1999). "The frog at the bug and made his mouth sad": Narrative competence in children with autism. *Journal of Abnormal Child Psychology*, 28, 193–204.
- Dunn, J., & Munn, P. (1986). Siblings and the development of prosocial behaviour. *International Journal of Behavioral Development*, 9, 265–284. <https://doi.org/10.1177/016502548600900301>.
- Dunn, J., Bretherton, I., & Munn, P. (1987). Conversations about feeling states between mothers and their young children. *Developmental Psychology*, 23(1), 132–139. <https://doi.org/10.1037/0012-1649.23.1.132>.
- Fletcher-Watson, S., McConnell, F., Manola, E., & McConachie, H. (2014). Interventions based on the theory of mind cognitive model for autism spectrum disorder. *Cochrane Database of Systematic Reviews*, 3, CD008785. <https://doi.org/10.1002/14651858.CD008785.pub2>.
- Garner, P. W. (2003). Child and family correlates of toddlers' emotional and behavioural responses to a mishap. *Infant Mental Health Journal*, 24, 580–596. <https://doi.org/10.1002/imhj.10076>.
- Hutchins, T. L., Bond, L. A., Silliman, E. R., & Bryant, J. B. (2009). Maternal epistemological perspectives and variations in mental state talk. *Journal of Speech, Language, and Hearing Research*, 52, 61–80. [https://doi.org/10.1044/1092-4388\(2008/07-0161\)](https://doi.org/10.1044/1092-4388(2008/07-0161)).
- Hutchins, T. L., Deraway, C., Prelock, P., & O'Neill, A. (2017). Mothers' and children's storytelling: A study of dyads with typically developing children and children with ASD. *Journal of Autism and Developmental Disorders*, 47, 1288–1304. <https://doi.org/10.1007/s10803-016-3022-z>.
- Jenkins, J. M., Turrell, S. L., Kogushi, Y., Lollis, S., & Ross, H. S. (2003). A longitudinal investigation of the dynamics of mental state talk in families. *Child Development*, 74, 905–920. <https://doi.org/10.1111/1467-8624.00575>.
- Kauschke, C., van der Beek, B., & Kamp-Becker, I. (2016). Narratives of girls and boys with autism spectrum disorders: Gender differences in narrative competence and internal state language. *Journal of Autism and Developmental Disorders*, 46, 840–852. <https://doi.org/10.1007/s10803-015-2620-5>.
- Kay-Raining Bird, E., Cleave, P. L., Curia, J., & Dunleavy, M. (2008). Parental talk about internal states to their child with autism. *Focus on Autism and Other Developmental Disabilities*, 23, 166–175. <https://doi.org/10.1177/1088357608319530>.
- Kirk, E., & Sharma, S. (2017). Mind-mindedness in mothers of children with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 43–44, 18–26. <https://doi.org/10.1016/j.rasd.2017.08.005>.
- LaBounty, J., Wellman, H. M., Olson, S., Lagattuta, K., & Liu, D. (2008). Mothers' and fathers' use of internal state talk with their young children. *Social Development*, 17, 757–775. <https://doi.org/10.1111/j.1467-9507.2007.00450.x>.
- Mastrogioseppe, M., Capirci, O., Cuva, S., & Venuti, P. (2014). Gestural communication in children with autism spectrum disorders during mother-child interaction. *Autism*, 19, 469–481. <https://doi.org/10.1177/1362361314528390>.
- Mazzone, S., & Nader-Grosbois, N. (2017). Emotion-related socialization behaviours in parents of children with an autism spectrum disorder. *Psychology*, 8, 1134–1160. <https://doi.org/10.4236/psych.2017.88.074>.
- Meins, E., Fernyhough, C., Fradley, E., & Tuckey, M. (2001). Rethinking maternal sensitivity: Mothers' comments on infants' mental processes predict security of attachment at 12 months. *Journal of Child Psychology and Psychiatry*, 42, 617–648. <https://doi.org/10.1111/1469-7610.00759>.
- Ruffman, R., Slade, L., & Crowe, E. (2002). The relation between children's and mothers' mental state language and theory-of-mind understanding. *Child Development*, 73, 734–751. <https://doi.org/10.1111/1467-8624.00435>.
- Slaughter, V., Peterson, C. C., & Mackintosh, E. (2007). Mind what mother says: Narrative input and theory of mind in typical children and those on the autism spectrum. *Child Development*, 78, 839–858. <https://doi.org/10.1111/j.1467-8624.2007.01036.x>.
- Slaughter, V., Peterson, C. C., & Carpenter, M. (2009). Maternal mental state talk and infants' early gestural communication. *Journal of Child Language*, 36, 1053–1074. <https://doi.org/10.1017/S0305000908009306>.
- Taumoepeau, M., & Ruffman, T. (2006). Mother and infant talk about mental states relates to desire language and emotion understanding. *Child Development*, 77(2), 465–481. <https://doi.org/10.1111/j.1467-8624.2006.00882.x>.
- Taumoepeau, M., & Ruffman, T. (2008). Stepping stones to others' minds: Maternal talk relates to child mental state language and emotion understanding at 15, 24, and 33 months. *Child Development*, 79, 284–302. <https://doi.org/10.1111/j.1467-8624.2007.01126.x>.

Tompkins, V., Benigno, J., Lee, B., & Wright, B. (2018). The relation between parents' mental state talk and children's social understanding: A meta-analysis. *Social Development*, 27, 223–246. <https://doi.org/10.1111/sode.12280>.

Wheelwright, S., Auyeung, B., Allison, C., & Baron-Cohen, S. (2010). Defining the broader, medium and narrow autism phenotype among parents using the Autism Spectrum Quotient (AQ). *Molecular Autism*, 1(10). <https://doi.org/10.1186/2040-2392-1-10>.

Paroxetine

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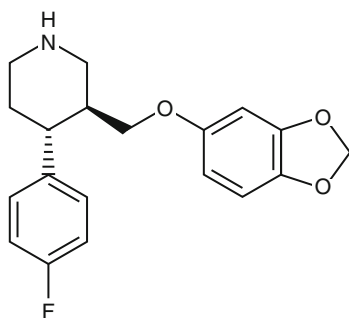
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Synonyms

Paroxetine hydrochloride; Paxil

Definition



Paroxetine is a selective serotonin reuptake inhibitor (SSRI) used to treat depression, social anxiety

disorder, obsessive-compulsive disorder, and post-traumatic stress disorder. Paroxetine is available in immediate- and controlled-release formulations and is the most potent inhibitor of serotonin reuptake compared to currently available SSRIs. Originally developed in the 1970s, its side effects include constipation, sedation, sexual dysfunction, discontinuation syndrome, and weight gain. Given the growing body of research that suggests that abnormalities in serotonin function are linked to autism, the use of SSRIs in patients with autism has grown. Case studies of patients with autism using paroxetine indicate mixed results, with reports of improved and worsened aggressive behavior.

See Also

- Antidepressants
- Selective Serotonin Reuptake Inhibitors (SSRIs)

References and Reading

- Kolevzon, A., Mathewson, K. A., & Hollander, E. (2006). Selective serotonin reuptake inhibitors in autism: A review of efficacy and tolerability. *The Journal of Clinical Psychiatry*, 67(3), 407–414.
- Marks, D. M., Park, M. H., Ham, B. J., et al. (2008). Paroxetine: Safety and tolerability issues. *Expert Opinion on Drug Safety*, 7(6), 783–794.
- Moore, M. L., Eichner, S. F., & Jones, J. R. (2004). Treating functional impairment of autism with selective serotonin-reuptake inhibitors. *The Annals of Pharmacotherapy*, 38(9), 1515–1519.
- Pae, C., & Patkar, A. A. (2007). Paroxetine: Current status in psychiatry. *Expert Review of Neurotherapeutics*, 7(2), 107–120.
- Paroxetine. Retrieved from the ChemSpider Wiki: <http://www.chemspider.com/RecordView.aspx?rid=efcc3d6d-ed52-46ce-a3ae-6f8e8a870a88>.
- U.S. Food and Drug Administration. (2011). *Paroxetine*. Retrieved from: <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm?fuseaction=Search.Overview&DrugName=PAROXETINE%20HYDROCHLORIDE>.

Paroxetine Hydrochloride

- Paroxetine

Parroting

► [Movie Talk](#)

Partial Agenesis

► [Agenesis of Corpus Callosum](#)

Participation of Black and African-American Families in Autism Research

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Definition

The majority of research articles focused on autism spectrum disorder (ASD) do not provide sufficient data on race and ethnicity (Pierce et al. 2014); thus, the level of participation of racial and ethnic minorities in ASD research is largely unknown. While approximately 13% of the US population identifies as Black (US Census Bureau 2018), Black children and their families are underrepresented in ASD research (Burkett et al. 2015; Hilton et al. 2010; West et al. 2016). Yet, racial disparities concerning timely ASD diagnoses and healthcare access are well-documented (e.g., see Thomas et al. 2007); and service delays persist for Black children regardless of family socioeconomic status (Magaña et al. 2012; Mandell et al. 2010). In this entry the term “Black” refers to people of the African diaspora, including those of African American descent, African-Caribbean descent, African-Latinx descent, and those from the African continent.

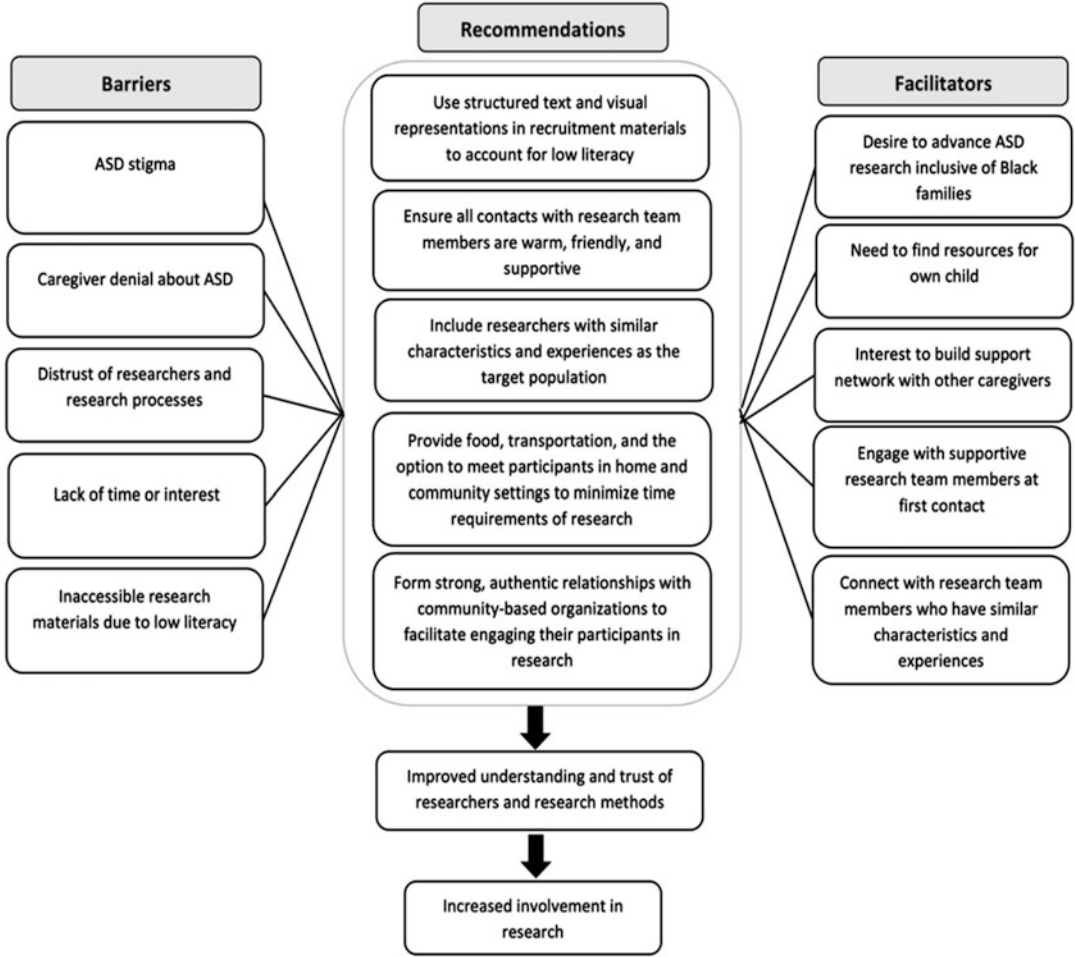
Historical Background

While research on Black families is still emerging, studies have identified potential factors leading to racial disparities in ASD service access and quality. Primary healthcare provider bias toward Black families may negatively impact parent-provider relationships and ASD screening of children, thus delaying children’s access to valuable early intervention (Burkett et al. 2015; Dababnah et al. 2018). Black parents report reluctance to share ASD-related concerns with their doctors (Donohue et al. 2017), in part because parents often encounter providers who disagree with them about the presence of ASD symptoms (Neuhaus et al. 2018; Dababnah et al. 2018; Pearson and Meadan 2018). Miscommunication between healthcare providers and parents, as well as cultural differences in symptom interpretation, can also lead clinicians to diagnose children differently based on race (Barton et al. 2012; Burkett et al. 2015; Mandell et al. 2007).

Current Knowledge

A recent study outlined several barriers and facilitators to the involvement of Black children and their families in ASD research (Shaia et al. 2019). The study directly solicited the perspectives of 22 parents and other primary caregivers of Black children with ASD using qualitative interview methods. The majority of participants were mothers of Black children with ASD; the remaining participants were grandmothers and other female caregivers. Approximately half of the participants had a high school education or less and annual household incomes less than \$50,000. Service providers referred over one-third of participants to the study; the remaining parents heard about the study from social media, schools, and community sources.

Figure 1 summarizes facilitators, barriers, and recommendations to increase Black children’s involvement in ASD research. Table 1 lists suggestions from caregivers of Black children for research recruitment sources (Shaia et al. 2019). The most common suggestion was school



Participation of Black and African-American Families in Autism Research, Fig. 1 Barriers and facilitators to involvement in ASD research by caregivers (Shaia et al. 2019)

Participation of Black and African-American Families in Autism Research, Table 1 Suggested methods to recruit Black families for ASD research

Suggested methods
School outreach
Services outreach
General flyers
Word of mouth
Faith-based organizations
Social media

recruitment; caregivers also wished to hear about studies from other parents and trusted sources. Notably, researchers should consider recruitment resources other than service providers, as Black

caregivers often do not see service providers as trustworthy information sources (Burkett et al. 2015).

Facilitators

Desire to Contribute to Inclusive Research
Shaia et al. (2019) found that caregivers were motivated to advance and contribute to ASD research. A common motivating factor included caregivers’ desire to help others learn more about ASD, particularly research specifically targeting Black families. These findings were consistent with the Burkett et al. (2015) study of Black children with ASD and their families, which

reported families tried to increase ASD awareness and education in their communities.

Seeking Information and Support

Some caregivers were motivated to take part in research in order to receive information and/or resources for their own children. Another related factor was increasing support for themselves, particularly to address isolation and to speak with others who understood their challenges related to raising a child with ASD.

Engaging with Culturally Responsive Research Team Members

Positive interactions with research team members were important facilitators that increased some caregivers' willingness to become involved in research. Shaia et al. (2019) reported study participants stated that it was important the interviewer identified herself as a Black mother of a child with ASD.

Barriers

ASD Stigma and Denial

Shaia et al. (2019) found stigma (from both inside and outside the Black community) was a potential barrier to Black families' involvement in ASD research. Their study also reported stigma was related to denial of the ASD diagnosis, which made parents reluctant to participate in ASD studies. Several other research studies focused on Black families have also identified ASD-related denial and stigma (Burkett et al. 2015; Dababnah et al. 2018; Pearson and Meadan 2018).

Distrust of Research

Suspicion and distrust of the research process was a potential barrier to Black families' engagement with research (Shaia et al. 2019). Concerns about sharing information, particularly with non-Black investigators, were common among caregivers raising Black children with ASD. Other research has similarly found perceived power differentials and cultural differences can be a barrier to Black participants' involvement in studies (Alvarez et al. 2006).

Lack of Time or Interest

Shaia et al. (2019) reported study participants cited lack of time, feeling overwhelmed, and

general disinterest as barriers to research involvement. Dababnah et al. (2018) found poverty and other external stressors can be barriers to following up on ASD service referrals, thus suggesting families who are experiencing poverty and other stressors may require additional supports to engage in research.

Inaccessible Research Materials

Difficulty in understanding research materials due to low literacy is a barrier for some potential research participants. Shaia et al. (2019) found some study participants were uncomfortable sharing their thoughts when the interview was being recorded. Skinner et al. (2008) likewise concluded lower educational levels were barriers to research participation.

Future Directions

Literature inclusive of Black children with ASD and their families is developing. Investigators should consider diverse community-based methods to increase involvement of Black participants, including schools, community services, and faith-based organizations. Partnerships between ASD researchers and community-based organizations can potentially facilitate recruitment of Black and other underrepresented children and their families.

Researchers should be aware of potential barriers to the involvement of Black families in ASD research, including ASD stigma, caregivers' denial of the ASD diagnosis, lack of time to dedicate to studies, and low literacy. Furthermore, Black parents' distrust of the research process or investigators can limit their willingness to engage in research. On the other hand, several factors can facilitate Black families' participation, such as their desire to contribute to studies that advance knowledge of ASD in their communities; to identify peer support; and to locate information for their children. In order to counteract possible parent distrust of the research, investigators should ensure their research team members are representative of the target study sample and engage with potential participants in a warm and empathic manner. In particular, research teams should be

aware of the many ways in which their personal attitudes and characteristics may influence participants' comfort and willingness to engage fully in studies. Figure 1 lists additional recommendations to increase participation of underrepresented families. Structured text and visual representations can aid individuals with low literacy to access research materials. Food, transportation support, childcare, and data collection in nontraditional settings (e.g., homes, libraries) can encourage participation. Researchers should also be careful to minimize the time needed to participate in studies.

Shaia et al. (2019) focused on the perspectives of urban caregivers of Black children with ASD. Like some other studies, most of the participants were mothers (Pearson and Meadan 2018; Burkett et al. 2015). Future research should explore how to increase participation of suburban and rural families, as well as non-maternal caregivers. More studies are needed to understand specific methods to support Black families overcome stigma; approaches researchers should employ to recruit Black families to research; how to improve the quality of services families receive; and how to increase the accessibility of research materials for all participants. With the increase in ASD diagnoses across all races (Baio et al. 2018), it is important that future ASD research is more fully representative of the diversity of the ASD community.

See Also

- [Bias in Assessment Instruments for Autism](#)
- [Parental Response to Diagnosis](#)
- [Service Utilization in Autism](#)
- [Social Class and Autism](#)

References and Reading

- Alvarez, R. A., Vasquez, E., Mayorga, C. C., Feaster, D. J., & Mitrani, V. B. (2006). Increasing minority research participation through community organization outreach. *Western Journal of Nursing Research*, 28(5), 541–560.
- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J. . . . Dowling, N. F. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summaries*, 67(SS-6), 1–23.
- Barton, M. L., Dumont-Mathieu, T., & Fein, D. (2012). Screening young children for autism spectrum disorders in primary practice. *Journal of Autism and Developmental Disorders*, 42(6), 1165–1174.
- Burkett, K. W., Morris, E., Manning-Courtney, P., Anthony, J., & Shambley-Ebron, D. (2015). African American families on autism diagnosis and treatment: The influence of culture. *Journal of Autism & Developmental Disorders*, 45(10), 3244–3254.
- Dababnah, S., Shaia, W., Campion, K., & Nichols, H. (2018). “We had to keep pushing”: Caregivers’ perspectives on autism screening and referral practices of black children in primary care. *Intellectual and Developmental Disabilities*, 56(5), 321–336.
- Donohue, M. R., Childs, A. W., Richards, M., & Robins, D. L. (2017). Race influences parent report of concerns about symptoms of autism spectrum disorder. *Autism*. <https://doi.org/10.1177/1362361317722030>.
- Hilton, C. L., Fitzgerald, R. T., Jackson, K. M., Maxim, R. A., Bosworth, C. C., Shattuck, P. T., . . . Constantino, J. N. (2010). Brief report: Underrepresentation of African Americans in autism genetic research: A rationale for inclusion of subjects representing diverse family structures. *Journal of Autism and Developmental Disorders*, 40(5), 633–639.
- Magaña, S., Parish, S. L., Rose, R. A., Timberlake, M., & Swaine, J. G. (2012). Racial and ethnic disparities in quality of health care among children with autism and other developmental disabilities. *Intellectual and Developmental Disabilities*, 50(4), 287–299.
- Mandell, D. S., Ittenbach, R., Levy, S., & Pinto-Martin, J. (2007). Disparities in diagnoses received prior to a diagnosis of autism spectrum disorder. *Journal of Autism & Developmental Disorders*, 37(9), 1795–1802.
- Mandell, D. S., Morales, K. H., Xie, M., Lawer, L. J., Stahmer, A. C., & Marcus, S. C. (2010). Age of diagnosis among Medicaid enrolled children with autism, 2001–2004. *Psychiatric Services*, 61(8), 822–829.
- Neuhaus, E., Beauchaine, T. P., Bernier, R. A., & Webb, S. J. (2018). Child and family characteristics moderate agreement between caregiver and clinician report of autism symptoms. *Autism Research*, 11(3), 476–487.
- Pearson, J. N., & Meadan, H. (2018). African American parents’ perceptions of diagnosis and services for children with autism. *Education and Training in Autism and Developmental Disabilities*, 53(1), 17–32.
- Pierce, N. P., O’Reilly, M. F., Sorrells, A. M., Fragale, C. L., White, P. J., Aguilar, J. M., & Cole, H. A. (2014). Ethnicity reporting practices for empirical research in three autism-related journals. *Journal of Autism and Developmental Disorders*, 44(7), 1507–1519. <https://doi.org/10.1007/s10803-014-2041-x>.
- Shaia, W. E., Nichols, H. M., Dababnah, S., Campion, K., & Garbarino, N. (2019). Brief report: Participation of

- black and African-American families in autism research. *Journal of Autism and Developmental Disorders*, 25, 1–6. <https://doi.org/10.1007/s10803-019-03926-0>.
- Skinner, C. S., Schildkraut, J. M., Calingaert, B., Hoyo, C., Crankshaw, S. S., Fish, L., Susswein, L., Jasper, C., & Reid, L. (2008). Factors associated with African Americans' enrollment in a national cancer genetics registry. *Public Health Genomics*, 11(4), 224–233.
- Thomas, K. C., Ellis, A. R., McLaurin, C., Daniels, J., & Morrissey, J. P. (2007). Access to care for autism-related services. *Journal of Autism and Developmental Disorders*, 37(10), 1902–1912.
- U. S. Census Bureau. (2018). QuickFacts. Retrieved from <https://www.census.gov/quickfacts/fact/table/US/PST045217>
- West, E. A., Travers, J. C., Kemper, T. D., Liberty, L. M., Cote, D. L., McCollow, M. M., & Lynn Stansberry, L. (2016). Racial and ethnic diversity of participants in research supporting evidence-based practices for learners with autism spectrum disorder. *The Journal of Special Education*, 50(3), 151–163.

Passive Group

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Synonyms

Inactive; Unengaged

Definition

The various different sets of criteria for autistic disorders usually include repetitive routines, language abnormalities or mutism, clumsiness, and odd responses to sensory input, but all emphasize marked impairment of social interaction as a fundamental problem. Kanner (1943) and Kanner and Eisenberg (1956), in their definitions of early infantile autism, emphasized social aloofness and indifference as an essential criterion for diagnosis. Asperger (1944) described children with what he called “autistic psychopathy,” whose pattern of social interaction was active, initiating contact with other people, but inappropriate in form.

Wing and Gould (1979) and Wing (1981) suggested that these authors were describing different aspects of an “autism spectrum.” This spectrum included those who were “aloof and indifferent” to others, those who were “passive” in their social interaction, and those who were “active but odd” in their approach to others. It must be emphasized that these groups merge into each other – there are no sharp dividing lines. It is also possible for someone to change from one type to another in their pattern of social behavior with increasing age or even in different environments.

The typical passive child does not make social approaches but accepts approaches from others. He or she may make eye contact, especially if the person approaching them makes positive effort to obtain eye contact. Many passive children are amenable and willing to do as they are told, so they can be accepted into a group of children playing together. The passive child will often accept the role assigned to him or her, such as that of a baby in a game of mothers and fathers. But, when the game is over, the passive child may continue in the role while the others turn to other pursuits. Passive children are much less likely than others in the autism spectrum to be difficult in behavior, to refuse to cooperate, or to have temper tantrums so do not draw attention to themselves (but see description of PDA below). However, Wing and Shah (2006) found in a study of individuals with autism spectrum disorders and a history of catatonic features, who developed catatonic deterioration, that this pattern occurred significantly more often in those with a history of social passivity. The clinical picture included movement problems (slowness and difficulty in initiating movements unless prompted, odd gait, odd stiff posture, freezing during actions, difficulty crossing lines, and inability to cease actions) and behavior problems (impulsive acts, bizarre behavior, awake at night but sleeping in the day, incontinence, and excited phases). In addition, there was marked reduction in the amount of speech or complete mutism.

In recent years, attention has been given to the prevalence of autism spectrum conditions in females. Attwood (2007) found that girls are more likely than boys to be passive, and, for this reason, their autism often remains undiagnosed.

Kopp and Gillberg (1992) and Kopp et al. (2010) have reported that girls are much more likely than boys to refuse demands passively, thus fitting into the group called pathological demand avoidance (PDA) (Newson et al. 2003).

In the past, it has been assumed that the passive group is markedly less common than the aloof or the active but odd groups. However, the recent rise of interest in this group, especially its prevalence in females, may change this view of passivity in autism. It is to be hoped that, in future, research will be carried out into the neurology underlying this mode of presentation.

See Also

- ▶ Active-But-Odd Group
- ▶ Aloof Group
- ▶ Catatonia
- ▶ Gender Differences
- ▶ Spectrum/Continuum of Autism
- ▶ Subtyping Autism

References and Reading

- Asperger, H. (1944). Die "autistischen psychopathen" im kindesalter. *Archives fur Psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Attwood, T. (2007). *The complete guide to Asperger syndrome*. London: Jessica Kingsley.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kanner, L., & Eisenberg, L. (1956). Early infantile autism 1943–1955. *American Journal of Orthopsychiatry*, 26, 55–65.
- Kopp, S., & Gillberg, C. (1992). Girls with social deficits and learning problems: Autism, Asperger syndrome or a variant of these conditions. *European Child & Adolescent Psychiatry*, 1, 89–99.
- Kopp, S., Kelly, K. B., & Gillberg, C. (2010). Girls with social and/or attention deficits: A descriptive study of 100 clinic attendees. *Journal of Attention Disorders*, 14, 197–181.
- Newson, E., Le Marechal, K., & David, C. (2003). Pathological demand avoidance syndrome: A necessary distinction within the pervasive developmental disorders. *Archives of Diseases of Childhood*, 88, 595–600.
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11, 115–129.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11–29.
- Wing, L., & Shah, A. (2006). Systematic examination of catatonia-like clinical pictures in autism spectrum disorders. In D. Dhossche, L. Wing, M. Ohta, & K. J. Neumarker (Eds.), *Catatonia in autism spectrum disorders*. San Diego: Academic Press.

Pathological Demand Avoidance (PDA)

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Synonyms

Autism spectrum disorder and PDA traits (O’Nions et al. 2018); Extreme demand avoidance (Gillberg 2014); Newson’s syndrome (Ogundeell 2018); Pathological demand avoidance syndrome (Newson et al. 2003); Rational demand avoidance (Milton 2017)

Definition

Elizabeth Newson developed the concept of pathological demand avoidance (PDA) to describe a subset of children referred to her clinic based in Nottingham, UK, in the 1970s (Newson et al. 2003). Newson applied the term PDA to children that were traditionally diagnosed as either having pervasive developmental disorder-not otherwise specified or atypical autism, yet both parents and clinicians thought these terms did not adequately describe the child’s presenting difficulties (Newson et al. 2003). Consequently, PDA was first proposed in 1980 as a distinct pervasive development disorder with the following behavior profile (Gillberg 2014): consistent resistance to every day demands, ease at role-play and pretend play, language delay due to passivity, lability of mood and impulsivity due to need for control, neurological involvement, obsessive behavior, passive early history, and surface sociability that lacks social identity/pride/shame (Newson et al. 2003).

The modern profile provided by a charity has been reduced by removing delayed speech development, neurological involvement, and passive early history (Green et al. 2018). This is due to focus on diagnosing PDA as a specifier for education support, producing autism spectrum disorder + PDA traits diagnosis by some UK clinicians (O’Nions et al. 2018). However, the higher prevalence rates being reported than Newson and colleagues originally observed (Green et al. 2018) are generating concerns about whether these profiles are representing the same underlying condition. PDA is viewed as clinically useful (Christie 2007; O’Nions et al. 2016), due to its behaviors and educational needs being distinct from autistic persons. These will be explored later.

PDA is frequently contrasted against autism profiles, with growing interest arising from parents, clinicians, and educational practitioners (Christie 2007; Langton and Frederickson 2016). A medium-size early investigation compared PDA to classical autism and Asperger’s syndrome. This study suggested that the following behaviors are more common in PDA than established autism subtypes (Newson et al. 2003): (1) strategies used for avoidance being socially manipulative, such as by distracting the adult, acknowledging the demand but excusing themselves, physically incapacitating themselves, and retreating into fantasy and role-play (these are examples of demand avoidance); (2) not identifying themselves as a child; (3) instantly changing mood; (4) taking on second-hand roles such as those of a teacher; and (5) their obsessions being often focused on other persons. Significantly, PDAers (preferred terminology of those identified with PDA) make more sustained eye contact than those with autism (Newson et al. 2003). Contemporarily, research into demand avoidance behaviors indicate that they could be described as strategic instead of “manipulative” and are also identified with many triggers of changes in mood or avoidance ranging from uncertainty to novelty (O’Nions et al. 2018); the latter is also expressed by autistic persons, highlighting the overlap between the PDA and autism profiles. While PDA has a behavior profile, this is not proof of its existence as a separate entity, and so it has many competing ontologies which are discussed next.

As a proposed syndrome, there are various attempts to explain and categorize PDA, which due to its omission from the diagnostic manuals is fiercely debated, in part because of PDA lacking substantial and compelling evidence to settle the debates for its inclusion into the diagnostic manuals (Green et al. 2018; O’Nions et al. 2016; Woods 2017). Previously, PDA was not included in International Classification of Diseases 10th Revision, neither is it recognized in the 11th Revision of the International Classification of Diseases (World Health Organisation 2018). Some view PDA as a pervasive developmental disorder (Newson et al. 2003). The dominant ontology views PDA as part of the autism spectrum (Christie 2007; Langton and Frederickson 2016; O’Nions et al. 2018), as a consequence of commonly referring to umbrella pervasive developmental disorders as autism spectrum disorders. Additionally, arguing against debating it’s ontologically, as it distracts from diagnosing PDA and thus utilizing its strategies (Christie 2007).

PDA’s less widely accepted ontologies range from it being autism, a product of autism interacting with its comorbidities in a person (Green et al. 2018), a female form of autism, variations of attachment disorder or personality disorder (Christie 2007), and an expression of autistic trauma (Milton 2017). Particularly, the behaviors associated with the PDA profile overlap with common comorbidities in autism, specifically attachment disorder (Milton 2017), anxiety disorder, attention deficit and hyperactivity disorder, conduct disorder, and oppositional defiant disorder (Green et al. 2018). PDA behaviors may eventually be shown to have heterogeneous origins and mechanisms involving many comorbid conditions (Gillberg 2014). Lastly, its cultural construction is being questioned, notably as the commodification of autism, by pathologizing autistic self-advocacy (Woods 2017), via the double empathy problem, and how different autism stakeholders frequently have different outlooks compared to each other, autistic persons self-agency is pathologized (Milton 2017).

Much of the ontology debates focus on the cause of high anxiety levels found in PDA. The dominant ontology, as a form of behaviorism (Milton 2017), views the high anxiety level to

be intrinsic to the PDAer (Green et al. 2018). Conversely, its critiques contest that the demand avoidant behavior is caused by anxiety through an interactive, transactional process, wherein differences in the subjective experiences of PDAers render different types of activities aversive compared to the experiences of neurotypicals, promoting anxiety and avoidant behavior (Green et al. 2018; Milton 2017). Until an overwhelming empirical case is presented or it is adopted in the diagnostic manuals, there will be ample debate and confusion over the nature of PDA. Nonetheless, PDA has specific strategies and so potentially remains clinically relevant; for its diagnostic and screening tools, see O’Nions et al. (2014) and (2016).

Phil Christie assisted Elizabeth Newson in the development of PDA as a concept and later refined its educational strategies with input from staff at an autism specialist school (Christie 2007). The strategies for PDA include having a specific keyworker to build a trusting relationship, making demands in an indirect way rather than using direct language, avoiding conflict, being flexible, and adapting and negotiating by providing choices to pupils. Other recommendations were to develop a positive relationship, providing indirect praise, using humor to control the child, and use of role-play, novelty, and variety in lesson material to engage the child capturing their interest (Christie 2007). Newson et al. (2003) argued that these strategies are needed because traditional autism strategies emphasizing adherence to routines and structure reportedly do not work on those with PDA. Notably PDAers are disruptive in specialist autism classes and have high exclusion rates, necessitating these precise strategies, which is an important motivation for clinical recognition or diagnosis of PDA (Christie 2007). Pertinently, the PDA strategies are not to be practiced separate from autism strategies, and each individual is to receive bespoke support.

Langton and Frederickson (2016) explore the educational needs of pupils with PDA, comparing their results to those for autism; their findings suggest that need for extra support for PDA is no more than that of autism and cast doubts about the need for an additional diagnostic label of PDA. Critique of PDA strategies includes that in the time since

Newson’s description of PDA, there has been a move away from rigidly practiced educational support to bespoke packages (Green et al. 2018). Milton (2017) argues that many of PDA strategies are suitable for other children and that many autism strategies do not work with autistic persons. Though for the foreseeable future its ontology is contested, there is an urgent need for more research into it and adoption of PDA strategies.

See Also

- Atypical Autism
- Behaviorist Theory
- Conduct Disorder
- England and Autism
- Personality Disorders
- Reactive Attachment Disorder

References and Reading

- Christie, P. (2007). The distinctive clinical and educational needs of children with pathological demand avoidance syndrome: Guidelines for good practice. *Good Autism Practice*, 8(1), 3–11.
- Gillberg, C. (2014). Commentary: PDA – Public display of affection or pathological demand avoidance? – Reflections on O’Nions et al. (2014). *Journal of Child Psychology and Psychiatry*, 55(7), 769–770.
- Green, J., Absoud, M., Grahame, V., Malik, O., Simonoff, E., Le Couteur, A., & Baird, G. (2018). Pathological demand avoidance: Symptoms but not a syndrome. *Lancet Child & Adolescent Health*, 2(6), 455–464.
- Langton, E., & Frederickson, N. (2016). Mapping the educational experiences of children with pathological demand avoidance. *Journal of Research in Special Educational Needs*, 16(4), 254–263.
- Milton, D. E. M. (2017). *A mismatch of salience: Explorations of the nature of autism from theory to practice*. Hove: Pavilion Publishing and Media Ltd..
- Newson, E., Le Maréchal, K., & David, C. (2003). Pathological demand avoidance syndrome: A necessary distinction within the pervasive developmental disorders. *Archives of Disease in Childhood*, 88(7), 595–600.
- O’Nions, E., Christie, P., Gould, J., Viding, E., & Happe, F. (2014). Development of the ‘Extreme demand avoidance questionnaire’ (EDA-Q): Preliminary observations on a trait measure for pathological demand avoidance. *Journal of Child Psychology and Psychiatry*, 55(7), 758–768.
- O’Nions, E., Gould, J., Christie, P., Gillberg, C., Viding, E., & Happé, F. (2016). Identifying features

of 'pathological demand avoidance' using the diagnostic interview for social and communication disorders (DISCO). *European Child & Adolescent Psychiatry*, 25(4), 407–419.

- O'Nions, E., Viding, E., Floyd, C., Quinlan, E., Pidgeon, C., Gould, J., & Happé, F. (2018). Dimensions of difficulty in children reported to have an autism spectrum diagnosis and features of extreme/'pathological' demand avoidance. *Child and Adolescent Mental Health*, 23(3), 220–227.
- Ogundele, M. (2018). Behavioural and emotional disorders in childhood: A brief overview for paediatricians. *World Journal of Clinical Pediatrics*, 7(1), 9–26.
- Woods, R. (2017). Pathological demand avoidance: My thoughts on looping effects and commodification of autism. *Disability & Society*, 34(5), 753–758.
- World Health Organisation. (2018). *ICD-11: The 11th revision of the international classification of diseases*. Geneva: World Health Organisation. <https://icd.who.int/browse11/l-m/en#/http://id.who.int/icd/entity/120443468>

Pathological Demand Avoidance Syndrome

► Pathological Demand Avoidance (PDA)

Patient- and Family-Centered Care

► Family-Centered Care, Second Edition

Patricia Howlin

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Emeritus Professor Patricia Howlin

BA, University of Sheffield, UK (1967); MSC
Clinical Psychology, Institute of Psychiatry,

London (1968); PhD, Institute of Psychiatry,
London (1979); Fellow, British Psychological
Society (1990).

Major Appointments (Institution, Location, Dates)

Emeritus Professor Patricia Howlin held her first academic appointment as lecturer and then senior lecturer at the Institute of Psychiatry (IOP) at King's College London, UK from 1973 to 1992. She subsequently became a professor in clinical psychology at St. George's Hospital Medical School, University of London, UK. In 2006, she was appointed to the first Chair of Clinical Child Psychology in the UK at the Institute of Psychiatry, Psychology and Neuroscience (IOPPN), King's College London, until her retirement from full-time academia in 2014. She continues to be actively involved in research and teaching and is currently an emeritus professor of clinical child psychology at the IOPPN and a professor of developmental disability at the University of Sydney, Australia. She is also an adjunct professor at Curtin University, Western Australia, and Griffiths University, Queensland, and visiting professor at the Oslo University Hospital, Norway.

Major Honors and Awards

Prof. Howlin has received numerous honors and awards throughout her professional career. She was made a fellow of the British Psychological Society in 1990. More recently, in 2009, the Society for the Study of Behavioural Phenotypes, UK named their annual lecture after her (the Patricia Howlin Prize Lecture), while the Autism Association of Western Australia honored her for her services to autism. In 2013, she won the 2013 International Society for Autism Research (INSAR) Lifetime Achievement Award, and in 2015, she was honored for her contributions to autism research by the German, Austrian, and Swiss Society for Research in Autism Spectrum Conditions.

Landmark Clinical, Scientific, and Professional Contributions

In the late 1970s and early 1980s, Patricia Howlin together with Prof. Sir Michael Rutter and other colleagues from the Maudsley Hospital, London published a landmark systematic study – the first in the UK and one of the very first internationally – to demonstrate the effectiveness of parents as therapists for their children with ASD.

Among her many contributions to conducting high-quality, systematic research on issues that were always highly relevant to the needs and priorities of the autism community over the years, three aspects of her work in particular were most prominent in driving the field forward:

1. Her work in the 1970s, 1980s, and 1990s on systematically monitoring, evaluating, and reporting outcomes following behavioral, communication, social, and other comprehensive and focal interventions for individuals with ASD.
2. Her committed focus on the importance of research evidence and of “translating” empirical findings for families and clinicians to support them in making informed decisions.
3. Her research studies published in the 1990s and 2000s on adult outcomes. These were some of the largest and longest term internationally follow-up studies of individuals with ASD first assessed in childhood and followed up in adulthood, and were instrumental in documenting the poor educational, employment, and social outcomes of many adults with ASD, thus firmly placing the urgent need for research and support for adults with ASD on the ASD “map” and agenda.

Short Biography

Emeritus Prof. Patricia Howlin, Ph.D., is a consultant clinical psychologist and one of the world’s leading experts in autism spectrum and related neurodevelopmental/genetic disorders. Throughout her career, she has held a number of

academic and clinical appointments in the UK and internationally and has been involved in numerous collaborative and impactful research studies on the effectiveness of comprehensive and focal interventions, mental health and quality of life, and adult outcomes in ASD.

She is the author of more than 200 peer-reviewed research publications on ASD, which have been cited thousands of times. She has also written or cowritten many books on ASD and related conditions which have been translated into a number of different languages, of which *Treatment of Autistic Children* in 1987 with Michael Rutter, *Children with Autism and Asperger Syndrome* in 1998, and *Autism and Asperger syndrome – Preparing for adulthood* in 2004 are especially noteworthy for positively influencing and supporting families and professionals and for bringing scientific knowledge about ASD into the wider community.

In addition to her work in ASD, she has in more recent years extended her research focus to include other neurodevelopmental genetic disorders (i.e., Williams, Down, Angelman, and Cornelia de Lange syndromes). The recipient of multiple awards for her contributions to the field of ASD (including an INSAR lifetime achievement award and the Kanner Asperger award from the German Scientific Association for Autism Research), she was the founding editor of *Autism: the International Journal of Research and Practice*, while she currently serves as the President of the Society for the Study of Behavioural Phenotypes.

She continues to lecture internationally and holds a number of visiting appointments and collaborations with leading learning and research institutions in Japan, Australia, Norway, and elsewhere. In addition to her scientific contributions, she has worked closely over many years as an advisor, consultant, or advocate with the National Autistic Society (NAS) in the UK and many other voluntary and community organizations to improve awareness, understanding, support, and funding for individuals with ASD and other genetic neurodevelopmental conditions across all ages and levels of ability.

References and Reading

Selected Peer-Reviewed Publications

- Green, J., Charman, T., McConachie, H., Aldred, C., Slonims, V., Howlin, P., Le Couteur, A., et al. (2010). Parent-mediated communication-focused treatment for preschool children with autism (MRC PACT); a randomised controlled trial. *The Lancet*, 375(9732), 2152–2160.
- Howlin, P. (2000). Outcome in adult life for more able individuals with autism or Asperger syndrome. *Autism: International Journal of Research and Practice*, 4, 63–84.
- Howlin, P. (2004a). Interventions for individuals with autism: Transition to adulthood. *Journal of Cognitive and Behavioral Psychotherapies*, 4, 223–232.
- Howlin, P. (2010). Evaluating psychological treatments for children with autism spectrum disorders. *Advances in Psychiatric Treatment*, 16, 133–140.
- Howlin, P. (2013). Social disadvantage and exclusion: Adults with autism lag far behind in employment prospects. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(9), 897–899.
- Howlin, P., & Udwin, O. (2006). Outcome in adult life for people with Williams syndrome – Results from a survey of 239 families. *Journal of Intellectual Disability Research*, 50, 151–160.
- Howlin, P., Marchant, R., Rutter, M., Berger, M., Hersov, L., & Yule, W. (1973). A home-based approach to the treatment of autistic children. *Journal of Autism and Childhood Schizophrenia*, 3, 308–336.
- Howlin, P. (1982).
- Howlin, P., Mawhood, L. M., & Rutter, M. (2000). Autism and developmental receptive language disorder – a comparative follow-up in early adult life. II: Social, behavioural and psychiatric outcomes. *Journal of Child Psychology and Psychiatry*, 41, 561–578.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcomes for children with autism. *Journal of Child Psychology and Psychiatry*, 45, 212–229.
- Howlin, P., Alcock, J., & Burkin, C. (2005). An 8 year follow-up of a specialist supported employment service for high-ability adults with autism or Asperger syndrome. *Autism*, 9(5), 533–549.
- Howlin, P., Magiati, I., & Charman, T. (2009). A systematic review of early intensive behavioral interventions (EIBI) for children with autism. *American Journal on Intellectual and Developmental Disabilities*, 114, 23–41.
- Howlin, P., Elison, S., & Stinton, C. (2010). Cognitive, linguistic and adaptive functioning in Williams syndrome: Trajectories from early to middle adulthood. *Journal of Applied Research in Intellectual Disabilities*, 23(4), 322–336.
- Howlin, P., Moss, P., Savage, S., Tempier, A., & Rutter, M. (2014). Cognitive and language skills in adults with autism: A 40 year follow-up. *Journal of Child Psychology and Psychiatry*, 55(1), 49–58.
- Magiati, I., Tay, X. W., & Howlin, P. (2014). Cognitive, language, social and behavioural outcomes in adults with autism spectrum disorders: A systematic review

of longitudinal follow-up studies in adulthood. *Clinical Psychology Review*, 34(2014), 73–86.

- Mawhood, L. M., Howlin, P., & Rutter, M. (2000). Autism and developmental receptive language disorder – a comparative follow-up in early adult life. I: Cognitive and language outcomes. *Journal of Child Psychology and Psychiatry*, 41, 547–559.

Selected Books

- Howlin, P. (1997). *Autism: Preparing for adulthood*. London: Routledge. (Winner of the NASAN 1997 Academic Book Award).
- Howlin, P. (1998). *Children with autism and Asperger syndrome*. Chichester: Wiley.
- Howlin, P. (2004b). *Autism and Asperger syndrome – preparing for adulthood*. London: Routledge.
- Howlin, P., & Rutter, M. (1987). *Treatment of autistic children*. Chichester: Wiley.
- Howlin, P., Baron-Cohen, S., Hadwin, J., & Swettenham, J. (1998). *A practical manual for teaching theory of mind to children with autism*. Chichester: Wiley.
- Howlin, P., Charman, T., & Ghazziudin, M. (Eds.). (2011). *The sage handbook of developmental disorders*. London: Sage.

Patterning (Doman-Delacato Method)

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Definition

The Doman-Delacato method, commonly known as patterning, is designed to improve a child's "neurological organization" through a series of specific prescribed sensory and motor experiences conducted on a rigorous daily schedule. These methods were presumed to improve functioning of the central nervous system in children with severe brain injuries (Doman et al. 1960).

Historical Background

The Doman-Delacato method is an approach to address neurological functioning by a series of

motor activities thought to alter the structure and function of specific areas of the brain. The method was developed by Glen Doman, a physical therapist, and Carl Delacato, a doctor of education. Doman and Delacato focused on maximizing the development of typical children. Spurring what Doman coined as the “Gentle Revolution,” he began to publish books aimed at teaching parents how to make their babies mentally and physically superior. Titles of their published books include “How to Teach Your Baby to Read” (Doman 1964b), “How to Teach Your Baby Math” (Doman et al. 1979), and “How Smart Is Your Baby?: Develop and Nurture Your Newborn’s Full Potential” (Doman and Doman 2006). Many of the titles from the Gentle Revolution series were coauthored with Doman’s son and daughter, Douglas and Janet Doman. Doman also published a book focused primarily on children with brain injury. In 1974, he published “What to Do About Your Brain Injured Child: Or Your Brain Damaged, Mentally Retarded, Mentally Deficient, Cerebral-Palsied, Spastic, Flaccid, Rigid, Epileptic, Autistic, Athetoid, Hyperactive Child” (Doman 1974).

In 1955, Doman founded the headquarters for the Institutes for the Achievement of Human Potential in the Philadelphia area. The IAHP (commonly referred to as “the Institutes”) is a nonprofit organization offering inpatient and outpatient treatment for brain-damaged children. The IAHP was created as a means for distributing the Doman-Delacato method, as described in a paper on neurological organization published in the *Journal of the American Medical Association* in 1960 (Doman et al. 1960). The IAHP has gained a global following, with offices in Japan, Italy, Mexico, Guatemala, Singapore, Brazil, Spain, and France.

Rationale or Underlying Theory

Temple Fay first applied the recapitulationist view of ontogenesis to children with nervous system disorders (Fay 1955), and the Doman-Delacato method is an extension of this work (MacKay et al. 1986). According to the recapitulationist theory, ontogeny (the development of an

individual from fertilized egg to its adult form) mimics phylogeny (the evolutionary history of a species). Stated another way, the development of an individual being imitates the evolutionary steps of the species. The recapitulationist school of thought was popular in the 1920s and 1930s. However, the theory has been refuted in modern biology and has not found support in the greater scientific community (Novella 1996).

Doman and Delacato advocate that children are expected to move through a series of locomotor patterns which reflect earlier forms of movements that human evolutionary ancestors performed, such as creeping and crawling. As such, skipping one of these evolutionary steps in one’s own development is believed to result in perceptual and motor difficulty as well as disturbances in language and communication skills (Doman 1974). The method presumes that basic motor sequences are essential to the neurological organization of an individual. According to the authors, the different stages of crawling, creeping, and walking are each associated with a unique neurological function and a gap in the appropriate sequence does not allow for an individual to fully develop (Doman 1964b). For example, a child who failed to crawl before walking is hypothesized to have skipped a critical step unique to human development, and this gap leaves the child subjected to both higher and lower order neurological deficits. Based on this rationale, it follows that the child must be “patterned” by a team of adults trained to position the child’s body in such a way that mimics this critical prerequisite step. As a result, the child’s neurons would be “repatterned” and reorganized in a way that allows them to continue with their typical development and encourage learning readiness in academic skills. According to this theory, intellectual disabilities, learning, and behavior disorders are caused by brain damage. Most importantly, these deficiencies are believed to exist on a single continuum for which the only solution is to regress to earlier forms of primitive movement (Cohen et al. 1970).

On the phylogenetic continuum, human movement may be divided into five main classes:

1. Truncal movement: Comparable to the swimming movement of a fish and believed to impact the medullary level of the brain.

2. Homolateral crawling: Defined as a crawling motion in which the arm and leg on the side to which the head is turned and flexed, while the opposite extremities are extended. This is hypothesized to reflect amphibian motility and believed to affect a pontine level of brain organization.
3. Cross-pattern creeping: Defined as creeping with a flexed arm and extended leg on the side toward which the head is turned. This creeping is believed to be related to reptilian movement and is hypothesized to be related to midbrain functioning.
4. Crude walking: Defined as walking without a cross pattern. This reflects a primitive upright form of locomotion consistent with cortical functioning.
5. Cross-pattern walking: Defined as the only uniquely human gait and associated with advanced cerebral function and hemispheric dominance.

In addition, the importance of establishing cerebral dominance is believed to be unique to humans, and this lateral neurological function is said to account for the human ability to read, write, and talk. As such, the lateralization of movement is central in the theory and practice of this method (Holm 1983). The authors also emphasize the importance of adding ongoing sensory stimulation to the patterning movements, as learning begins with the stimulation of the senses.

Goals and Objectives

Treatment goals of the Doman-Delacato method aim to improve physical, intellectual, and social capability in children with brain injuries by correcting “neurological organization.”

Treatment Participants

Proponents of the method believe that treatment is suitable for all children classified as “brain-injured.” As defined by the IAHP, the term “brain-injured” encompasses nearly 300 potential

childhood disorders. The IAHP does not differentiate across severity, and even the most severely handicapped children may be admitted. Doman and Delacato have repeatedly emphasized that only those parents who are most dedicated to their children’s recovery can expect to see a result from the prescribed demanding regimen.

Treatment Procedures

Predetermined patterns of movement are imposed on the child by manipulation of the child’s extremities. Such manipulations are performed by teams of up to five people, often consisting of therapists, parents, and volunteers. In the sessions, each adult is responsible for manipulating one of the child’s limbs or the head. It is required that it should be performed smoothly and rhythmically and in complete accordance with the movement of the other limbs in order to mimic the natural movement of the body (Doman et al. 1960). The IAHP recommends that these exercises be conducted a minimum of at least 5 min, four times a day every day of the week. While the treatment team is initially composed of therapists, it gradually becomes the responsibility of parents and volunteers to conduct the sessions. Typically, the treatment therapists conduct follow-up visits in 60- to 90-day intervals. It is hypothesized that such patterning sessions will ingrain the movements into the central nervous system.

Parents are also required to provide their children with a program of sensory stimulation. In addition, masking, or rebreathing expired air into a face mask for 30 to 60 s once every waking hour, is promoted to increase cerebral blood flow, increase carbon dioxide intake, and aid in the establishment of hemispheric dominance (Freeman 1967). According to the IAHP, this rebreathing treatment is also recommended in the treatment of seizure disorders, and the IAHP requires that all patients be gradually weaned off of anticonvulsant medications to maximize the effectiveness of their own treatment regimen. Restricting salt, sugar, and fluids and limiting exposure to music are additional recommendations.

Efficacy Information

The Doman-Delacato method has not been scientifically investigated with individuals on the autism spectrum. To date, the concept of neurological organization that serves as the foundation for the method has not been subjected to scientific research, and those programs which offer patterning have not been shown to improved learning or functioning. Many have contested that the theoretical rationale for the treatment is without merit and is inconsistent with accepted views of neurologic development (Novella 1996). Although any intervention that calls for one-to-one daily interaction for hours at a time on a daily basis might have the potential to have some positive effect, evidence for any permanent and lasting change from patterning is lacking (Howlin 1997). As a result, this method has been met with controversy and criticism.

The American Academy of Pediatrics Committee on Children with Disabilities has issued several cautionary statements regarding the Doman-Delacato method. Due to the lack of empirical support for the strategies, warnings were published as early as 1968, with the most recent statement reaffirmed in 2005 (American Academy of Pediatrics 1982; 1999). This was a joint statement approved by several organizations, including but not limited to the American Academy for Cerebral Palsy, American Academy of Neurology, American Academy for Physical Medicine and Rehabilitation, American Academy of Orthopedics, Canadian Association for Retarded Children, and the National Association for Retarded Citizens (Hyatt 2007).

Outcome Measurement

The primary outcome measurement tool for this therapy was developed by the inventors of this method. The Doman-Delacato developmental profile, which is used for both planning treatment and monitoring progress, enables the therapist to ascertain at which level of neurological organization a brain-injured child is functioning. The profile is based on chronological age development

when the following domains are hypothesized to develop mobility, language, manual competence (writing), vision (reading), auditory competence, and tactile competence. Each of these domains is divided into seven chronological stages of functional development which corresponds with ascending brain levels.

The validity of this tool for planning treatment or measuring outcome has yet to be demonstrated (Sparrow and Zigler 1978). Because the administration of this instrument at intake informs the course of treatment, changes with treatment may reflect teaching to the test and do not necessarily correlate with a generalizable improvement in functioning. To date, this measurement has not been standardized against any accepted measures of development, although inter-rater reliability is reported to be valid. The dimensions measured by the assessment make the comparison to any standard measure difficult.

Qualifications of Treatment Providers

Staff at the IAHP are trained as “child brain developmentalists.” These developmentalists specialize in one of the following: intellectual excellence, physical excellence, or physiological excellence. According to IAHP literature, this certification requires rigorous training and yearly recertification. No certification or licensure process exists outside of the Institutes for the Achievement of Human Potential, and the required qualifications of these child brain developmentalists are not publically disclosed. The IAHP continually holds courses in seminars throughout the world to educate parents on how to conduct patterning with their children.

References and Reading

- American Academy of Pediatrics, Committee on Children With Disabilities. (1982). The Doman-Delacato treatment of neurologically handicapped children. *Pediatrics*, 70, 810–812.
- American Academy of Pediatrics, Committee on Children With Disabilities. (1999). The treatment of neurologically impaired children using patterning. *Pediatrics*, 104(5), 1149–1151.

- Cohen, H. J., Birch, H. G., & Taft, L. T. (1970). Some considerations for evaluating the Doman-Delacato "patterning" method. *Pediatrics*, 45(2), 302–314.
- Doman, G. J. (1964a). *The five principles of human development through organization of the brain*. Retrieved 28 June 2012 from http://www.iahp.org/fileadmin/PDFs/Five_Principles.pdf.
- Doman, G. J. (1964b). *How to teach your baby to read*. New York: Random House.
- Doman, G. (1974). *What to do about your brain-injured child: Or your brain-damaged, mentally retarded, mentally deficient, cerebral-palsied, spastic, flaccid, rigid, autistic, athetoid, hyperactive, Down's child*. New York: Doubleday.
- Doman, G. J., & Doman, J. (2006). *How smart is your baby?: Develop and nurture your newborn's full potential*. New York: Square One.
- Doman, R. J., Spitz, E. B., Zucman, E., Delacato, C. H., & Doman, G. (1960). Children with severe brain injuries. *Journal of the American Medical Association*, 174, 257–262.
- Doman, G. J., Doman, J., Aisen, S., & Institutes for the Achievement of Human Potential. (1979). *How to teach your baby math*. New York: Simon and Schuster.
- Fay, T. (1955). The origin of human movement. *American Journal of Psychiatry*, 111(9), 644–652.
- Freeman, R. D. (1967). Controversy over "patterning" as a treatment for brain damage in children. *Journal of the American Medical Association*, 202(5), 83–86.
- Holm, V. A. (1983). A western version of the Doman-Delacato treatment of patterning for developmental disabilities. *The Western Journal of Medicine*, 139(4), 553–556.
- Howlin, P. (1997). Prognosis in autism: Do specialist treatments affect long-term outcome? *European Child & Adolescent Psychiatry*, 6(2), 55–72.
- Hyatt, K. J. (2007). Brain gym: Building stronger brains or wishful thinking? *Remedial and Special Education*, 28(2), 117–124.
- MacKay, D. N., Gollogly, J., & McDonald, G. (1986). The Doman-Delacato treatment methods. *British Journal of Mental Subnormality*, 32, 3–19.
- Novella, S. (1996). Psychomotor patterning. *The Connecticut Sceptic*, 1(4). Retrieved 28 June 2012 from <http://www.srmhp.org/archives/patterning.html>.
- Sparrow, S., & Zigler, E. (1978). Evaluation of a patterning treatment for retarded children. *Pediatrics*, 62(2), 137–150.

Pavlovian Conditioning

► Classical Conditioning

Paxil

► Paroxetine

Pay

► Employment

PDD

► Asperger Syndrome

PDD-NOS (Pervasive Developmental Disorder Not Otherwise Specified)

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Definition

The equivalent, if somewhat ambiguous, terms PDD-NOS, "subthreshold" autism, and "atypical" autism refer to the broad range of conditions characterized by problems in social communication as well as in communication and/or restricted interests and behaviors that are suggestive of more strictly defined autism but fall short of the latter concept in terms of symptom thresholds. This term was used in DSM-II-R and DSM-IV and would be somewhat analogous to the term "broader autism phenotype." DSM-5 did not include this term, and most cases with it (under ► "DSM-IV") would no longer receive a diagnosis (McPartland et al. 2012), but such cases, if they had a "well-established" diagnosis before DSM-5, were "grandfathered" in.

Historical Background

The concept has, in many respects, its origins in Rank's use of the term atypical personality development to describe a range of difficulties in social-

emotional development and regulation (Rank 1949; Caplan 1955). In DSM-III (1980), the word atypical was used to describe “subthreshold” autism as the latter diagnosis was included in the PDD class for the first time as an official diagnosis. This use of the term unintentionally hearkened back to Rank’s earlier use of the same word you describe a rather similar diagnostic concept. In recent years, an awareness of the “broader autism phenotype” and the complex genetics surrounding autism have lent increased interest to work on this concept (Towbin 2005; Towbin et al. 2005) which clearly outnumbered more classical autism (Fombonne 2005).

Current Knowledge

At present, this and related terms refer to a “residual” category included in DSM and ICD for individuals whose problems do not meet the threshold for diagnosis but which are sufficiently severe as to serve as a source of impairment/distress and are relevant focus for treatment. In reality, the term refers to a relatively large group (probably about 1 in 150 children) of children. Clearly, there is even greater heterogeneity within this condition than there is with autism. Understandably, many attempts have been made to define specific subgroups/subtypes, e.g., individuals with greater intentional difficulties versus those with more mood/affective problems (Hellgren et al. 1994; Landgren et al. 1996). Study of the condition may be particularly relevant to the delineation of specific genetic mechanisms (Rutter 2005; Towbin et al. 2005).

In terms of clinical presentation, individuals with this condition frequently exhibit social difficulties, emotional lability, and unusual sensitivities, although, as a group, they probably have cognitive and language abilities than in more “classical” autism. Frequently, clinicians equate the concept with Asperger’s disorder, although some data suggest that in the latter condition social deficits are more severe (Klin et al. 2005; Volkmar et al. 1994; Volkmar and Klin 2000). Treatment approaches are rather similar to those for autism but with an awareness of often reasonably good cognitive potential and the presence of

comorbid conditions like anxiety and mood problems as well as affective lability (Towbin 2005). As noted above the lack of explicit recognition in DSM-5 is problematic.

Future Directions

Over the coming years, the identification of specific genetic subgroups/subtypes of autism will likely lead to a refinement of this concept.

See Also

- ▶ Autistic Disorder
- ▶ Broader Autism Phenotype
- ▶ Childhood-Onset Pervasive Developmental Disorder
- ▶ DAMP Syndrome
- ▶ DSM-5
- ▶ DSM-III
- ▶ DSM-IV

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual* (3rd ed.). Washington, DC: APA Press.
- Fombonne, E. (2005). Epidemiological studies of pervasive developmental disorders. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 42–69). Hoboken: Wiley.
- Hellgren, L., Gillberg, I. C., Bågenholm, A., & Gillberg, C. (1994). Children with deficits in attention, motor control and perception (DAMP) almost grown up: Psychiatric and personality disorders at age 16 years. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 35(7), 1255–1271.
- Klin, A., Pauls, D., Schultz, R., & Volkmar, F. (2005). Three diagnostic approaches to Asperger syndrome: Implications for research. *Journal of Autism and Developmental Disorders*, 35(2), 221–234.
- Landgren, M., Pettersson, R., Kjellman, B., & Gillberg, C. (1996). ADHD, DAMP and other neurodevelopmental/psychiatric disorders in 6-year-old children: Epidemiology and co-morbidity. *Developmental Medicine and Child Neurology*, 38(10), 891–906.
- McPartland, J. C., Reichow, B., & Volkmar, F. R. (2012). Sensitivity and specificity of proposed DSM-5 diagnostic criteria for autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(4), 368–383.

- Rank, B. (1949). Adaptation of the psychoanalytic technique for the treatment of young children with atypical development. *American Journal of Orthopsychiatry*, 19, 130-139. American Psychological Association/Educational Publishing Foundation, USA.
- Rank, B. (1955). Intensive study and treatment of pre-school children who show marked personality deviations, or "atypical development", and their parents. In G. Caplan (Ed.), *Emotional problems of early childhood* (pp. 491-501). Oxford: Basic Books.
- Rutter, M. (2005). Genetic influences and autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 425-452). Hoboken: Wiley.
- Towbin, K. E. (2005). Pervasive developmental disorder not otherwise specified. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 165-200). Hoboken: Wiley.
- Towbin, K. E., Pradella, A., Gorrindo, T., Pine, D. S., & Leibenluft, E. (2005). Autism spectrum traits in children with mood and anxiety disorders. *Journal of Child and Adolescent Psychopharmacology*, 15(3), 452-464.
- Volkmar, F. R., & Klin, A. (2000). Diagnostic issues in Asperger syndrome. In A. Klin & F. R. Volkmar (Eds.), *Asperger syndrome* (pp. 25-71). New York: The Guilford Press.
- Volkmar, F. R., Klin, A., Siegel, B., Szatmari, P., Lord, C., Campbell, M., et al. (1994). Field trial for autistic disorder in DSM-IV. *The American Journal of Psychiatry*, 151(9), 1361-1367.

PDDRS

- [Pervasive Developmental Disorders Rating Scale \(PDDRS\)](#)

PDMS

- [Peabody Developmental Motor Scales \(PDMS\)](#)

PDMS-2

- [Peabody Developmental Motor Scales \(PDMS\)](#)

PDMS-II

- [Peabody Developmental Motor Scales \(PDMS\)](#)

Peabody Developmental Motor Scales (PDMS)

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Synonyms

[PDMS](#); [PDMS-II](#); [PDMS-2](#)

Description

The Peabody Developmental Motor Scales-2 (PDMS-2; Folio and Fewell 2000) is a criterion-referenced and norm-referenced developmental assessment comprised of two scales: the Gross Motor Scale and the Fine Motor Scale. The Gross Motor Scale includes four categories: reflexes, stationary gross motor skills, locomotion, and object manipulation. The reflex subtest is administered to children from birth through 11 months. It contains eight items that measure reactions to environmental events. The stationary subtest includes 30 items that measure equilibrium and sustained control of the body within the center of gravity. The locomotion subtest measures the child's skills in moving from one place to another through 89 items that cover the developmental progression of various modes of movement. The object manipulation subtest includes 24 items that measure the ability to manipulate balls. Test items specifically measure skills such as:

- Reflex integration and postural adjustments
- Balancing on one foot and on tiptoes
- Crawling, walking, running, jumping, and skipping
- Throwing, catching, and kicking a ball

The Fine Motor Scale is comprised of two categories: grasping and visual-motor integration. The grasping subtest includes 26 items that

measure the ability of the child to use hands independently or together. The visual-motor integration subtest contains 72 items that measure visual perceptual skills and eye-hand coordination. Test items specifically measure skills such as:

- Demonstrating various grasp patterns as appropriate for different objects
- Stacking or configuring blocks to replicate a demonstrated design
- Manipulating buttons, using scissors, folding paper, and drawing to copy designs

A child's performance on each test item is scored a 0, 1, or 2. A score of 0 indicates no success on the item, 1 indicates emerging abilities to complete the item, and 2 indicates mastery or success in meeting all criteria for the test item. Test scores can be calculated as percentiles, standard scores, and age equivalents for the Gross Motor Quotient, Fine Motor Quotient, and Total Motor Quotient.

As a performance-based measure, the PDMS-2 can be administered in approximately 20–30 min per scale and 45–60 min for the total test (Folio and Fewell 2000).

Historical Background

The Peabody Developmental Motor Scales (PDMS; Folio and Fewell 1983), originally published in 1983, consisted of a Gross Motor Scale and Fine Motor Scale which were normed for children ages 1–83 months. The scales could be used separately to measure the respective motor domain or used together to obtain an overall motor performance composite score. The test was widely used throughout North America and frequently referenced in the published literature. The PDMS was revised in the late 1990s in order to update test items and establish contemporary norms. Normative data were collected between 1997 and 1998 using a sample of 2,003 North American children representing four major geographic regions from 46 states and one Canadian province. The test continues to be widely used by occupational and physical therapists and can also

be administered by diagnosticians, early intervention specialists, psychologists, and others with training in motor skill assessment.

Psychometric Data

Characteristics of the normative sample for the PDMS-2 were consistent with the 1997 US Census demographic data with respect to geographical area, gender, race, residence location (rural, suburban, urban), ethnicity, and socioeconomic status. Overall, the sample is considered representative of the US population at the time the test was developed. Scoring norms are available for typically developing children from birth through age 5 (1–84 months). No normative data are available for children with disability conditions.

Reliability. Internal consistency was evaluated using Cronbach's coefficient alphas. Values ranged from 0.84 to 0.98 indicating strong associations among test items within the same construct (e.g., fine motor, gross motor). Measures of test-retest reliability were limited to two groups of young children ages 2 through 11 months ($n = 20$) and 12 through 17 months ($n = 30$). Resulting correlation coefficients ranged from 0.73 to 0.96, suggesting acceptable test-retest reliability within the items applicable to these age groups. Inter-rater reliability for test scores yielded coefficient values of 0.96 to 0.98. Inter-rater reliability for individual test items has not been examined.

Validity. Content validity of the PDMS-2 is supported through developmental theory and various item analysis techniques including item response theory and logistic regression. Age-related trends, such as increases in mean test scores occurring with increasing age, have been observed. The various subtests within each composite are supported through confirmatory factor analysis. Concurrent validity was examined between the PDMS-2 and the initial version of the test, the PDMS. The resulting correlations were strong with 0.84 for the fine motor composite and 0.91 for the gross motor composite. Moderate to strong correlations were also noted with

scores from the gross and fine motor scales of the Mullen Scales of Early Learning: AGS Edition.

Clinical Uses

The PDMS-2 is often used in clinical and educational settings to assess gross and fine motor skills along the developmental trajectory (Richardson 2010). The tool can be used to identify children with delays in motor skills and to plan intervention.

References and Reading

- Chien, C., & Bond, T. G. (2009). Measurement properties of fine motor scale of Peabody Developmental Motor Scales-Second Edition: A Rasch analysis. *American Journal of Physical Medicine & Rehabilitation*, 88, 376–386.
- Darrah, J., Magill-Evans, J., Volden, J., Hodge, M., & Kembhavi, G. (2007). Scores of typically developing children on the Peabody Developmental Motor Scales-infancy to preschool. *Physical & Occupational Therapy in Pediatrics*, 27, 5–19.
- Folio, M. R., & Fewell, R. R. (1983). *Peabody developmental motor scales and activity cards*. Allen: Teaching Resources.
- Folio, R., & Fewell, R. (2000). *Peabody developmental motor scales-2* (2nd ed.). Austin: Pro-Ed.
- Maring, J. R., & Elbaum, L. (2007, Summer). Concurrent validity of the early intervention developmental profile and the Peabody developmental motor scale-2. *Pediatric Physical Therapy*, 19(2), 116–120.
- Mulligan, S. (2003). *Occupational therapy evaluation for children: A pocket guide*. Philadelphia: Lippincott, Williams, & Wilkins.
- Provost, B., Heimerl, S., & Lopez, B. R. (2007). Levels of gross and fine motor development in young children with autism spectrum disorder. *Physical & Occupational Therapy in Pediatrics*, 27, 21–36.
- Richardson, P. K. (2010). Use of standardized tests in pediatric practice. In J. Case-Smith & J. C. O'Brien (Eds.), *Occupational therapy for children* (6th ed., pp. 216–243). Maryland Heights: Mosby.
- Stewart, K. B. (2010). Purposes, processes, and methods of evaluation. In J. Case-Smith & J. C. O'Brien (Eds.), *Occupational therapy for children* (6th ed., pp. 193–215). Maryland Heights: Mosby.
- Van Waelvelde, H., Peersman, W., Lenoir, M., & Smits Engelsman, B. C. M. (2007). Convergent validity between two motor tests: Movement-ABC and PDMS-2. *Adapted Physical Activity Quarterly*, 24(1), 59–69.

Peabody Individual Achievement Test, Revised

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Synonyms

PIAT-R; PIAT-R/NU

Description

The PIAT-R/NU assesses achievement across six different content areas: general information, reading recognition, reading comprehension, mathematics, spelling, and written expression. The PIAT-R/NU does not consolidate each content area under a specific domain, and only the subtests that contain content related to the individual's educational concern should be administered. However, in order to calculate composite scores, multiple tests must be administered. The administration of multiple tests allows for a more in-depth understanding of an individual's strengths and weaknesses. The PIAT-R/NU allows for composite scores to be derived from the reading (Total Reading) and writing (Written Language) subtests. A total composite score (Total Test) can also be calculated utilizing five of the six subtests (excludes Written Expression). The PIAT-R/NU is published by Pearson Assessments and the author is Frederick C. Markwardt, Jr.

The General Information subtest contains 100 items that assess general knowledge. The topics included within this subtest are: science, social studies, fine arts, and factual knowledge. These items are administered verbally. The Reading Recognition subtest contains 100 items that measure recognition of printed letters and assess an individual's ability to read words out loud. These items also require a verbal response. The Reading Comprehension subtest consists of 82 items that measure reading comprehension. This task necessitates an individual to be able to

select a picture (by pointing or verbal response) that best illustrates a sentence that she/he has just read. The Mathematics subtest contains 100 questions. In this subtest, an individual is read a math question and then is required to respond via multiple choice either orally, or by pointing. The Spelling subtest consists of 100 items that assess both letter recognition (both by name and by sound) and spelling words correctly. The spelling portion of this subtest requires the examinee to choose the correctly spelled word from a choice of several words, either verbally or by pointing. The Written Expression subtest assesses both prewriting skills (i.e., the ability to copy and write letters, words, and sentences from dictation) and the ability to write a story about a presented picture (Sattler 2001).

Criticisms of the PIAT-R/NU include that the Reading Comprehension subtest may be more of an assessment of memory, as opposed to reading comprehension. The Mathematics subtest does not include any paper and pencil computations and covers a large range of mathematical skills and therefore does not narrow in on any one topic. The format of the spelling test (choosing from a group of variations of the target word) may not adequately assess for spelling ability. Lastly, the Written Expression subtest has low test-retest reliabilities, inter-scorer reliabilities, and internal consistency reliabilities (Sattler 2001).

A score of "1" indicates a correct score and a score of "0" indicates an incorrect score, with the exception of Written Expression. Raw scores are converted to standardized scores, grade equivalents, age-equivalent scores, percentile ranks, normal curve equivalents, and stanine scores. The Written Expression subtest requires a general achievement score to be calculated, and then grade-based stanines and a developmental scaled score are provided (Sattler 2001).

Scoring software is available to decrease the amount of time spent scoring and increase accuracy. The software is available for both Windows and Macintosh operating systems. The scoring software also allows reports to be generated in both English and Spanish.

In addition to being used with typically developing children, the PIAT-R/NU is an adequate

assessment tool for individuals with developmental difficulties such as intellectual disability. Furthermore, the PIAT-R/NU can be a useful tool for children with Autism Spectrum Disorders (ASDs) given the flexibility of the instrument. Individuals with an ASD often have varied needs across the spectrum resulting in atypical testing approaches being a necessary component of a quality assessment (Koegel et al. 1997). The PIAT-R/NU allows for this flexibility with six subtests that can be used interchangeably in response to an individual's needs and/or a spoiled administration of a subtest. Additionally, many of the subtests allow for responses in a multiple choice approach. For example, the mathematics subtest requires the examiner to read a question out loud and then the individual chooses the best answer from a limited selection. Other subtests allow for responses to be counted if they are gestured, written, or spoken. This format is especially useful for testing individuals with adequate receptive and low expressive language abilities, which often describe individuals with an ASD (Sattler 2001).

Historical Background

The Peabody Individual Achievement Test (PIAT) was first published in 1970 by Llyod Dunn and Frederick C. Markwardt, Jr. The original version of the achievement test only contained five subtests. When the PIAT was re-normed in 1989 the subtest Written Expression was added, thus resulting in the Peabody Individual Achievement Test, Revised (PIAT-R). In addition to the creation of a new subtest, most of the items of the original five subtests were replaced with new items to address changes in school curriculum (Allinder and Fuchs 1992). The PIAT-R was then re-normed between 1995 and 1996 as a part of a coordinated norming initiative, thus resulting in the Peabody Individual Achievement Test, Revised/Norms Update (PIAT-R/NU). The re-norming process allowed for an extension of the age range from 22-years-0-months to 22-years-11-months. The PIAT-R/NU is the most up-to-date version of the test.

Psychometric Data

The PIAT-R/NU was standardized between 1995 and 1996 as part of a re-norming project that included the KeyMath-Revised, Kaufman Test of Educational Achievement-Brief Edition, Kaufman Test of Educational Achievement-Comprehensive Edition, and Woodcock Reading Mastery Tests-Revised. The sample that the PIAT-R/NU was standardized with included 3184 children between the grades of kindergarten and the 12th grade and 245 young adults between the ages of 18 and 22. Between 204 and 295 children were administered each subtest per grade level. Special needs populations were included in the standardization process consistent with the nationwide representation. The sample is representative of 40 of the 52 states of the United States of America (USA). The sample was representative of the 1994 US census data with respect to gender, race/ethnicity, parental education, educational placement and geographic region. Most children did not receive the complete PIAT-R during the norming process (Sattler 2001).

For five of the six subtests, internal consistency reliabilities were quite good ranging from .87 to .98. For the Written Expression subtest, internal consistency reliability ranged from .60 to .91, relatively weaker in comparison to the other subtests (Sattler 2001).

Test-retest reliability was acceptable with correlations ranging in the high .80s to the mid-.90s for grade norms and with correlations ranging in the low to mid-.90s for age norms. Inter-rater reliability was low for Written Expression with Level II reliability being .58 for Prompt A and .69 for Prompt B.

The validity of the PIAT-R/NU is considered acceptable. The clinical validity studies completed have suggested that the PIAT-R/NU is a sensitive tool used to distinguish strengths and weaknesses across five of the six subtests.

Clinical Uses

Achievement testing is a necessary component of any special needs assessment protocol. Utilizing a measure, like the PIAT-R/NU, that allows for

broad screening and is adaptive and flexible allows for easing the challenges of testing individuals who are restricted verbally. Achievement testing allows for better Individual Education Plans (IEPs) to be developed that are specific to the needs of the student. Without the use of achievement testing IEPs risk being broad and nonspecific to the individual student, thus running the risk of not receiving adequate services and school placement.

The PIAT-R/NU is advantageous in that there are norms for both grade level and age. This allows for nontraditional students to be assessed and compared on multiple levels. For example, if a student with mild intellectual disability was held back a year and was being assessed to determine grade placement for the following year, it would be useful to consult the grade norms as opposed to the age norms to determine advancement.

Not only can the PIAT-R/NU be helpful in the school setting to aid in assessing strengths and weaknesses and creating goals for IEPs, but they also can assist in procuring services such as need-based tutoring, special education placement, and Title I services.

Appropriate intervention following a screen demonstrating academic weaknesses could result in significant gains for many individuals. The PIAT-R/NU is unique in that it allows for even individuals with low expressive language to be evaluated which offers many opportunities for helping these individuals work on building both their strengths and weaknesses.

References and Reading

- Allinder, R. M., & Fuchs, L. S. (1992). Screening academic achievement: Review of the Peabody Individual Achievement Test – Revised. *Learning Disabilities Research and Practice*, 7(1), 45–47.
- Koegel, L. K., Koegel, R. L., & Smith, A. (1997). Variables related to differences in standardized test outcomes for children with autism. *Journal of Autism and Developmental Disorders*, 27(3), 233–243. 0162-3257/97J0600-0233\$12.50/0.
- Markwardt, F. C. (1997). *Peabody individual achievement test – Revised/normative update*. Bloomington: Pearson Assessments.
- Sattler, J. M. (2001). *Assessment of children: Cognitive applications* (4th ed.). San Diego: Jerome M. Sattler.

Peabody Picture Vocabulary Test

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Synonyms

Peabody Picture Vocabulary Test, Fourth Edition (PPVT)

Description

The PPVT is a test of receptive vocabulary – that is, it assesses the lexicon of words that a person can understand when he or she hears them. It is also designed to serve as a screening test for verbal ability. The test has a straightforward structure. Examinees see a page on an easel with four-color pictures. For each item, the examiner says a word, and the examinee responds by selecting one picture out of four that best illustrates that word's meaning. Because the examinee points to the appropriate item, the test requires no reading, writing, or expressive verbal language, and it can be used with nonreaders and those without fluent verbal abilities. The test is untimed and individually administered. In total, the PPVT contains 228 items, divided into 19 “sets” of 12 items each; an examinee completes all items within a set. The *basal* level is set when an examinee correctly responds to 11 or more items in a set; the *ceiling* is established as the set where an examinee makes eight or more errors. Basal and ceiling levels are thought to represent the levels of difficulty below which an examinee is expected to know all items and above which the examinee is predicted to fail most items, respectively.

The PPVT was conformed with the Expressive Vocabulary Test – Second Edition, which serves as a measure of what words a person can speak aloud after seeing a pictorial representation of the item; PPVT and EVT scores were correlated at $r(3540) = 0.82$ in the normative sample. PPVT

scores are provided as standard scores with a mean of 100 and a standard deviation of 15. Grade- and age-equivalent scores can also be computed and can help with interpreting an examinee's relative knowledge. However, such scores are not recommended for research use given discontinuities between months or grades; that is, there are meaningful differences in the rate of development over different age periods, or between grade levels, which makes the steps between levels noncontinuous. Furthermore, consumers of such scores may not always remember that, as an average score, there must by necessity be 50% of examinees that score below the mean for a given age or grade level. Finally, users can also calculate a growth scale value, which tracks vocabulary over time.

Historical Background

The first edition of the PPVT was published by Lloyd M. Dunn in 1959. Subsequent revisions were published by Lloyd and Leota Dunn in 1981 (PPVT-R), in 1997 (PPVT-III), and by Douglas Dunn (son of Lloyd and Leota, Ph.D.) in (2007) The PPVT-4 is the current version. Lloyd Dunn, an expert in the fields of special education and child development, founded the Kennedy Center in 1965 and was central in efforts to train researchers in the field of mental retardation. His early work was instrumental in promoting *educational mainstreaming* and, more generally, addressing the educational needs of children whose abilities differ from the average. He served as the first director of the Institute on Mental Retardation and Intellectual Development, an institute of the Kennedy Center, developed the first doctoral program in special education at the Vanderbilt Peabody College of Education and Human Development, and developed a number of tools designed to assess language and cognitive skills in the service of educational intervention.

Recently, the PPVT-4 was incorporated into the NIH Toolbox – Cognition Battery (Gershon et al. 2014), based on its high test–retest reliability and strong construct validity when compared with relevant gold-standard measures. The NIH

Toolbox provides a battery of very brief measures designed to assess core functions (cognitive, emotional, sensory, and motor processes) using an iPad, with the guidance of an experimenter.

Psychometric Data

Words for the PPVT were originally selected from the dictionary on the basis of their imageability; the target word and the three distractors all had to be amenable to representation via line drawings (originally in black and white, all items are now depicted in color; all can be distinguished by color-blind individuals). Items were selected from the categories of body parts, emotions, foods, clothing, toys and recreation, and so on. In earlier versions of the test, there was a high proportion of items depicting verbs, but the PPVT-4 contains a smaller proportion of these items; they were found to be disproportionately difficult for young children. All items in a given trial are balanced for detail and visual complexity. There are four training items, for which the examiner is permitted to give feedback. An examinee must correctly complete two of these items in order for testing to be valid. The training items permit the examiner to establish whether the examinee is capable of responding in a standard fashion.

The PPVT-4 was standardized on a sample of 3540 individuals between the ages of 2 years, 6 months, and 90 years. Participants were representative of the general US population (according to census data) in terms of gender (male, female), ethnicity (Latino/Latina/Hispanic, African American, White, and "other," a group comprising American Indians, Alaska Natives, Asian Americans, Pacific Islanders, and all other groups not otherwise classified), geographic region, socioeconomic status, and special education placement. Twelve percent of the sample had parents whose educational achievement was grade 11 or lower; 28% had achieved 12th grade or a GED; 31% had 1–3 years of college; and 28% had 4 or more years of college. To establish age norms, the sample consisted of 28 age groups, with approximately 100–200 cases in each group; for ages

2–6 years, intervals were 6 months to account for the rapidly changing vocabulary levels in young children. All other groups were stratified by year. Fifty-seven percent ($n = 2003$) of the sample also contributed to a grade-stratified sample, comprising 26 groups (fall and spring, kindergarten through twelfth grade).

In addition, some participants in the normative sample had developmental concerns: speech or language impairment (5–15 years, $n = 178$ for ages 5–15; $n = 60$ ages 50–96), language delay (3–7 years, $n = 63$), intellectual disability (6–17 years, $n = 70$), reading disability (ages 8–14 years, $n = 71$), ADHD (6–17 years, $n = 91$), hearing impairment (4–12 years, $n = 99$), and several low-incidence disabilities. ASD was not specifically targeted. Proportions of special populations were intended to match population percentages. Data for average scores for these subgroups relative to the larger reference group are provided in the technical manual and range from a relatively small difference of -5.6 points for individuals with speech impairment to a relatively large difference of -29.7 points for hearing-impaired individuals with cochlear implants.

Two alternate forms are available, providing the option to repeat assessments more frequently. The reliability of these forms, which are identical in format and organization, with parallel but non-identical words tested, was assessed at 0.94. This permits the PPVT to serve as an assessment of *response to intervention* and for other methods requiring multiple test administrations. Test-retest correlations range from 0.92 to 0.96.

Construct validity of the PPVT-4 was assessed using the Comprehensive Assessment of Spoken Language (CASL; Carrow-Woolfolk 1999); for ages 3–5 years ($n = 68$), CASL subtest scores correlated with the PPVT as follows: basic concepts, $r = 0.50$; antonyms, $r = 0.41$; and sentence completion, $r = 0.54$. For ages 8–12 years ($n = 62$), CASL subtest scores correlated with the PPVT as follows: synonyms, $r = 0.65$; antonyms, $r = 0.78$; sentence completion, $r = 0.63$; and lexical/semantic composite, $r = 0.79$. Correlations with core language scores from Clinical Evaluation of Language Fundamentals –

Fourth Edition ranged from 0.73 (ages 5–8) to 0.72 (ages 9–12), with slightly lower correlations for the receptive ($r = 0.67$ and 0.75 for the two age groups) and expressive ($r = 0.75$ and 0.86 for the two age groups) subscales. These moderate-to-high correlations indicate that the other oral language assessments measure a constellation of similar but nonidentical skills. The PPVT-4 test is reliable, with all reliability and validity coefficients in the .90s range.

Clinical Uses

Research partially supports the utility of the PPVT as an assessment of general language abilities in the autism spectrum disorders. A study of 44 children with autism, ages 4–14, found that standardized measures of language ability (PPVT, EVT, and Clinical Evaluation of Language Fundamentals) correlated well with spontaneous assessments (mean length of utterance, index of productive syntax, and number of different word roots; Condouris et al. 2003). Furthermore, the assessments of vocabulary and semantic knowledge more generally were significantly correlated with assessments of grammatical knowledge. A meta-analysis of 133 publications in the field of ASD between 1999 and 2002 (Mottron 2004) found that a vocabulary measure (British Picture Vocabulary Scale, or BPVS; nearly identical in structure to the PPVT) had been used as a matching variable for 22% of publications, second only to overall IQ estimations using the Wechsler scales (47%). Thus, research in the field has acted in accord with the assumption that vocabulary skills, estimated with the PPVT, may be a useful proxy for general verbal language skills. Certainly, data from typical development indicates that vocabulary correlates highly with other language abilities and is the single best predictor of academic success for children starting school.

However, there is significant evidence that vocabulary assessed by the PPVT may be a strength for individuals with ASD relative to morphosyntax (Eigsti et al. 2007) or discourse-level comprehension (Asberg 2011). That is,

PPVT scores may overestimate general verbal abilities. Supporting this proposal, Mottron (2004) reported that receptive vocabulary may be a particular area of strength in ASD, even compared to tests considered to tap into ASD-specific expertise, such as block design tasks. Thus, vocabulary skills, estimated with the PPVT, may overestimate the functional abilities of participants with ASD.

The PPVT-4 has several specific limitations. First, it assesses only vocabulary items that are imageable – primarily, concrete nouns and verbs. As noted above, verbs, function words (*and*, *if*), and grammatical markers (*-ing*, plural “*s*”) are absent from the test. Thus, if an individual has a specific deficit in morphosyntactic abilities, the PPVT will fail to identify this difficulty. Second, the PPVT is inadequate for assessing individuals who are not fluent in English. There is also a Spanish version (Test de Vocabulario en Imagenes, TVIP; 1986) whose format is similar to the English version but with distinct items; because word frequencies differ dramatically across languages, it is not possible to simply translate items into another language (see an example of the challenge in a Danish study; Brynskov et al. 2017).

A third important limitation of the PPVT, specific to the case of ASD, is the presence of atypical vocabulary skills, such as the production of jargon words or echolalia (Eigsti et al. 2007). Children with ASD have been found to show some similar word-learning biases to typically developing children, in that they are able to map novel words onto novel objects, suggesting that their word learning is constrained by a “mutual exclusivity” bias that category labels apply to mutually exclusive objects (de Marchena et al. 2011). Furthermore, they are able to sort objects according to typical semantic categories. In contrast, research suggests that individuals with ASD produce less prototypical words, have difficulty in learning words that refer to mental states, and show differential priming effects (as reviewed in Eigsti et al. 2011); they also fail to show the developmentally typical shape bias (Tek et al. 2008). Because the PPVT is organized according to how vocabularies are structured in typically developing individuals, it may fail to

detect differences in ASD. This lack of sensitivity is further possible because the PPVT does not have any overt semantic organization in the scoring procedure; it is not possible to report a particular deficit in acquisition of words in any particular semantic (or structural) language category.

One final topic bears explicit mention. Clinicians have occasionally reported better responses by individuals with ASD in the PPVT when assessed using facilitated communication strategies. However, studies explicitly comparing responses by examinees with the assistance of facilitators who could or could not see the task materials have conclusively demonstrated that any improvement in scores using assistance primarily reflects the facilitator rather than the examinee's knowledge (Beck and Pirovano 1996).

See Also

- Echolalia
- Idiosyncratic Language
- Verbal Comprehension
- Vocabulary

References and Reading

- Asberg, J. (2011). Patterns of language and discourse comprehension skills in school-aged children with autism spectrum disorders. *Scandinavian Journal of Psychology*, 51(6), 534–539.
- Beck, A. R., & Pirovano, C. M. (1996). Facilitated communicators' performance on a task of receptive language. *Journal of Autism and Developmental Disorders*, 26, 497–512.
- Brynskov, C., Eigsti, I. M., Jørgensen, M., Lemcke, S., Bohn, O.-S., & Krøjgaard, P. (2017). Syntax and morphology in Danish-speaking children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(2), 373–383. <https://doi.org/10.1007/s10803-016-2962-7>.
- Carrow-Woolfolk, E. (1999). *Comprehensive assessment of spoken language (CASL)*. Bloomington: Pearson Assessments.
- Condouris, K., Meyer, E., & Tager-Flusberg, H. (2003). The relationship between standardized measures of language and measures of spontaneous speech in children with autism. *American Journal of Speech-Language Pathology*, 12(3), 349–358.
- de Marchena, A., Eigsti, I. M., Worek, A., Ono, K. E., & Snedeker, J. (2011). Mutual exclusivity in autism spectrum disorders: Testing the pragmatic hypothesis. *Cognition*, 119, 96–113.
- Dunn, L. M., & Dunn, D. M. (2007). *Peabody picture vocabulary test* (4th ed.). Circle Pines: American Guidance Service.
- Eigsti, I. M., Bennetto, L., & Dadlani, M. B. (2007). Beyond pragmatics: Morphosyntactic development in autism. *Journal of Autism and Developmental Disorders*, 37(6), 1007–1023.
- Eigsti, I. M., de Marchena, A. B., Schuh, J. M., & Kelley, E. (2011). Language acquisition in autism spectrum disorders: A developmental review. *Research in Autism Spectrum Disorders*, 5, 681–691.
- Gershon, R. C., Cook, K. F., Mungas, D., Manly, J. J., Slotkin, J., Beaumont, J. L., & Weintraub, S. (2014). Language measures of the NIH toolbox cognition battery. *Journal of the International Neuropsychological Society*, 20(6), 642–651. <https://doi.org/10.1017/S1355617714000411>.
- Mottron, L. (2004). Matching strategies in cognitive research with individuals with high-functioning autism: Current practices, instrument biases, and recommendations. *Journal of Autism and Developmental Disorders*, 34, 19–27.
- Tek, S., Jaffery, G., Fein, D., & Naigles, L. R. (2008). Do children with autism spectrum disorders show a shape bias in word learning? *Autism Research*, 1(4), 208–222. <https://doi.org/10.1002/aur.38> [doi].

Peabody Picture Vocabulary Test, Fourth Edition (PPVT)

- [Peabody Picture Vocabulary Test](#)

PEAK Relational Training System

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Synonyms

ABLLS: Assessment of Basic Language and Learning Skills; IRR: Inter-rater Reliability;

PEAK: Promoting the Emergence of Advanced Knowledge; PPVT: Peabody Picture Vocabulary Test; VABS-II: Vineland Adaptive Behavior Scales-II; VB-MAPP: Verbal Behavior Milestones and Placement Program

Description

The Promoting the Emergence of Advanced Knowledge (PEAK) relational training system is a criterion referenced assessment and curriculum designed to provide a measure of an individual's language and verbal abilities. The PEAK relational training system is composed of four separate modules: PEAK-Direct Training (PEAK-DT; Dixon 2014a), PEAK-Generalization (PEAK-G; Dixon 2014b), PEAK-Equivalence (PEAK-E; Dixon 2015), and PEAK-Transformation (PEAK-T; Dixon 2016). Each module contains a 184-item assessment designed to identify language skills present within an individual's repertoire and potential skill deficits the individual is lacking. Scores on the assessment are representative of an individual's current verbal abilities, in which a lower score on the assessment is indicative having a limited verbal repertoire, whereas higher scores are indicative of having a more complex verbal repertoire. Because procedures for conducting the assessments vary slightly for each module, each contains specific instructions for conducting the assessments for that specific module. Also contained within each module is a curriculum containing 184-programs that correspond to each of the items from the assessment. The programs aim to teach any of the skill deficits identified as lacking indicated by results from the assessment. Each program sheet contains a goal statement for the targeted skill, a material list, instructions for teaching the skill, and example stimuli typically utilized during instruction for that particular program. Although the modules advance in complexity with the PEAK-DT module being the least complex and the PEAK-T being the most complex, it is intended for program instruction of the four modules to be conducted simultaneously. Program instruction for all for modules is conducted using discrete trial training

(DTT) format. In this format, instruction consists of clearly defined units that allow for the delivery of a task or question issued to the learner, followed by specific feedback for the response to that particular task/question. Following the delivery of the task/question, the implementor waits for the learner to emit a response and provides a reinforcer or reward for correct responses and corrective feedback through the use of prompts or modeling for incorrect responses. Because each response contacts some type of consequence, the learner's responding will be modified accordingly. As individuals acquire the targeted skills necessary for program mastery, more advanced programs are then introduced, which allow for more complex language and verbal skills to be targeted.

The first two modules, the PEAK-DT module (Dixon 2014a) and the PEAK-G module (Dixon 2014b), share similarities in terms of assessment administration and program implementation. Items on the PEAK-DT and PEAK-G modules can be conducted using either an indirect or direct assessment. For indirect assessment administration, the assessor simply reviews each item on the assessment and indicates whether the individual has been observed to engage in the targeted skill previously or not. If the individual has the skill in their repertoire, then the assessor marks the item as "Yes" on the assessment scoring sheet. If the individual has not been observed as engaging in the targeted skill, then the assessor marks the item as "No" on the assessment scoring sheet. If the assessor is unsure whether the individual has the targeted skill in their repertoire, then the assessor marks the item as "?" on the assessment scoring sheet. All items marked as "?" on the scoring sheet will be directly assessed subsequently. To conduct a direct assessment, the assessor systematically presents each item to an individual and records whether they engage in the targeted skill. The assessor marks "Yes" on the assessment scoring sheet if the individual engages in the targeted skill and "No" if the individual does not respond or engages in an incorrect form of the targeted behavior. Although both indirect and direct assessment methods can be utilized to identify potential skill deficits, the authors recommend

direct assessment over indirect assessment, as this allows for more accurate assessment results. Regarding program implementation, both the PEAK-DT and PEAK-G modules focus on contingency-based learning approaches to teaching language and verbal skills. Many of the first programs in the PEAK-DT module focus on establishing basic prerequisite skills necessary for program instruction to take place (e.g., eye contact, turn-taking, and following instructions) or focus on simpler, basic language and verbal skills (e.g., oral motor imitation, making requests using yes and no, and vocal imitation). Later programs build upon earlier programs and target more complex language and verbal skills (e.g., receptive and expressive identification of stimuli, demonstrating adverb action, and guessing). Program instruction for the PEAK-DT module is conducted using DTT procedures, in which every response is followed by feedback. Similarly, programs within the PEAK-G also increase in complexity as an individual progresses through the programs. There are, however, differences in terms of program implementation. The PEAK-G module differs from the PEAK-DT module in that it incorporates a train-test methodology for program instruction. This methodology involves presenting untrained stimuli that are embedded within blocks of directly trained stimuli, in order to promote stimulus and response generalization.

The last two modules, PEAK-E module (Dixon 2015) and the PEAK-T module (Dixon 2016), move beyond the other modules to promote derived responding similar to that commonly exhibited in deductive and inductive reasoning tasks. Assessment administration of these two modules differs slightly than that of the previously mentioned modules, in that these modules incorporate the use of a pre-assessment. The pre-assessment is brief assessment designed to provide an initial evaluation of an individual's complex language and verbal abilities. Scores on the pre-assessment are then used to determine a starting point of the full assessments for the corresponding modules. The PEAK-E pre-assessment consists of a single 48-item assessment that requires both receptive and expressive responses from the learner. The PEAK-T module

pre-assessment consists of a 96-item receptive assessment and a 96-item expressive assessment, which are conducted separately from one another. The PEAK-E and PEAK-T full assessments are divided into different levels of complexity, with the least complex levels occurring near the beginning of the assessment and the most complex levels occurring toward the end of the assessment. Higher scores on the pre-assessment are indicative of an individual having more complex language and verbal skills and lower scores indicative of lower language and verbal skills. Therefore, higher scores on the pre-assessment mean an assessor will start the full assessment at a higher level, and for lower scores the assessor will begin at a lower level. Regarding program implementation, although some discrepancies exist between the two modules, the general method for program implementation is similar. Program implementation involves teaching a specific set of stimulus relations while testing another set of stimulus relations to evaluate the extent to which individuals infer the untrained stimulus relations. The PEAK-E and PEAK-T modules are designed to promote the emergence of complex verbal and deductive reasoning skills so that they occur naturally without direct training and facilitate this pattern of responding as it applies to a larger societal context.

Historical Background

The first two PEAK books, the PEAK-DT and PEAK-G modules, were published in 2014, followed by the PEAK-E module in 2015 and the PEAK-T module in 2016. All modules are in their first editions; however, analyses have been conducted to evaluate the underlying constructs of the programs within the modules. Principal component analyses were conducted for the PEAK-DT module (Rowsey et al. 2015) and the PEAK-G module (Rowsey et al. 2017). The principal component analyses provided information regarding the internal consistency of the modules and allowed the developers to categorize programs into specific components and arrange programs in order of their complexity.

Psychometric Data

Since the PEAK relational training system first emerged in 2014, data has been generated regarding the psychometric properties of the system. A majority of studies examining psychometric properties of the PEAK system were conducted on the PEAK-DT and PEAK-G modules. Psychometric data for the PEAK-E and PEAK-T exists but is limited, due to these modules being the most recently published of the series.

In terms of reliability, several studies have been conducted that assessed the reliability of the PEAK relational training system. Dixon, Stanley, Belisle, and Rowsey (2016) evaluated the test-retest reliability of the PEAK-DT module. In this study involving 39 participants (ages 6–20), the authors administered the PEAK-DT assessment on two separate occasions, during an initial assessment and during a follow-up assessment 3 weeks to 1 month later. The results suggested the PEAK-DT module had high test-retest reliability (intraclass correlation = 0.987, $p < 0.001$). Test-retest reliability has not been evaluated on the other modules in the PEAK relational training system. Another form of reliability that has been evaluated is inter-rater reliability (IRR). In addition to evaluating test-retest reliability, Dixon et al. (2016) also evaluated inter-rater reliability with a subset of ten participants in their study. To accomplish this, a second assessor scored assessments for the subset of participants, and these scores were then compared to the assessment scores of the primary assessor. Both assessors were blind to the other assessors' scores. IRR was assessed by determining the percentage of items in which the two observers scored an assessment item the same. The IRR analyses revealed a strong relationship between the two assessors scores, with a Cohen's kappa reliability coefficient of 0.981 ($p < 0.001$) and total agreement score of 99%. Several other studies have reported IRR for the PEAK-DT module. For example, McKeel et al. (2015b) evaluated IRR for a small portion of participants in their study ($n = 8$; ages 2–22) and reported a total agreement of 92.3%. Other studies have reported similar findings in terms of total IRR agreement scores,

with scores of 87.11% (Dixon et al. 2014b) and 85% (Dixon et al. 2014c).

Studies have also been conducted assessing the validity of the PEAK relational training system. Convergent validity has been assessed by correlating scores on the PEAK assessments with other similar measurement tools. For instance, Dixon et al. (2014c) correlated the PEAK-DT module with IQ. Measures were obtained for 64 participants, and the results suggested a moderately strong correlation between the two ($r = 0.759$, $p < 0.01$). McKeel et al. (2015b) correlated the PEAK-DT module with receptive and expressive one-word picture vocabulary tests (Martin and Brownell 2011a, b) and found a strong correlation between the measures. A Pearson correlation coefficient of 0.908 ($p < 0.01$) was reported for the correlation between the PEAK-DT and receptive one-word picture vocabulary tests, and a correlation coefficient of 0.901 ($p < 0.01$) was found between the PEAK-DT and the expressive one-word picture vocabulary test. In another study, researchers evaluated the relationship between the PEAK-DT, Peabody Picture Vocabulary Test (PPVT; Dunn and Dunn 2007), and Illinois Early Learning Standards. The findings revealed a strong positive correlation ($r = 0.908$, $p < 0.01$) between PEAK-DT and PPVT scores, as well as with the PEAK-DT and Illinois Early Learning Standards ($r = 0.916$, $p < 0.01$; Dixon et al. 2014b). The extent to which the PEAK-DT module was correlated with the Assessment of Basic Language and Learning Skills (ABLSS; Partington 2008) and Vineland Adaptive Behavior Scales-II (VABS-II; Sparrow et al. 2005) was conducted by Malkin et al. (2016). The authors report a strong positive correlation between the PEAK-DT and the ABLSS ($r = 0.951$, $p < 0.05$) and a moderate correlation between the PEAK-DT and the VABS-II ($r = 0.453$, $p < 0.05$). Finally, correlations between the PEAK-DT and PEAK-G modules and the Verbal Behavior Milestones Assessment and Placement Program (VB-MAPP; Sundberg 2008) have also been conducted (Dixon et al. 2015). The analyses revealed a strong positive correlation between the PEAK-DT module and the VB-MAPP ($r = 0.93$, $p < 0.01$) and a moderate relationship

between the PEAK-G module and the VB-MAPP ($r = 0.57$, $p < 0.01$). Taken together, these data support the convergent reliability of portions of the PEAK relational training system.

In addition to convergent validity, the PEAK-DT and PEAK-G modules have evaluated in terms of their content validity (Dixon et al. 2014a, 2017). To assess content validity, PEAK-DT and PEAK-G assessments were conducted with a normative sample consisting of 206 (ages 1–21) and 183 participants (ages 1–21), respectively. Scores on the assessment were then correlated with the ages of the participants. The results suggested that assessment scores were strongly correlated with age for both the PEAK-DT ($r = 0.659$, $p < 0.01$) and PEAK-G ($r = 0.753$, $p < 0.01$) modules. The same analyses were also conducted in a sample with autism and suggest that age was not significantly correlated with assessment scores on the PEAK-DT ($n = 94$; $r = 0.021$, $p = 0.861$) or PEAK G ($n = 94$; $r = 0.200$, $p = 0.068$). Content validity has not yet been assessed for the PEAK-E or PEAK-T modules.

As previously mentioned, principal component analyses have been conducted on the PEAK-DT module (Rowsey et al. 2015) and the PEAK-G module (Rowsey et al. 2017). These analyses were conducted to assess the content validity for each of the PEAK modules. The results of the component analyses suggested that the modules were internally consistent, providing support for the content validity of the assessments. Additionally, the component analysis allowed the researchers to categorize each item within the assessment into specific factors for each module. For the PEAK-DT module, four factors were identified: (1) Foundational Learning Skills, (2) Perceptual Learning Skills, (3) Verbal Comprehension Skills, and (4) Verbal Reasoning, Memory, and Math Skills. Four components were also identified for the PEAK-G module, which include (1) Foundational Learning and Basic Social Skills; (2) Basic Verbal Comprehension, Memory, and Advanced Social Skills; (3) Advanced Verbal Comprehension, Reading and Writing, and Basic Problem Solving Skills; and (4) Verbal Reasoning, Problem Solving, Logic, and Mathematical Skills.

Taken together, these preliminary data support the PEAK relational training system as both a reliable and valid tool. The PEAK-DT module has generated the most psychometric data compared to the other three modules. The PEAK-DT module has generated test-retest and inter-rater reliability, as well as content and convergent validity. Similarly, the PEAK-G module has generated psychometric data in the form of content validity. It should be noted, however, that much of the existing psychometric data is limited to the PEAK-DT and the PEAK-G modules. The PEAK-E and PEAK-T modules have not yet generated the same amount of psychometric data. All sample sizes in the psychometric studies were relatively small, suggesting further replications are warranted.

Clinical Uses

Research supports the use of the PEAK relational training system in terms of the assessment identifying specific skill deficits an individual may have, as well as the use of the curriculum in teaching the targeted skills. Effectiveness research has emerged supporting the use of all PEAK modules (see Dixon et al. 2017). Most of the effectiveness research on the PEAK Relational Training System was evaluated utilizing single subject research designs. A number of varying skills have been targeted for program instruction, with all studies reporting positive outcomes in producing the targeted skills. A majority of effectiveness research has been conducted on the PEAK-E module; however, several studies exist for the other modules as well. Only one effectiveness study has employed a group research design in the form of a randomized control trial (McKeel et al. 2015a). In this study, the researchers compared pre-test and post-test scores on the PEAK-DT assessment for two groups of individuals with autism. One group received treatment as usual, in the form of their school's typical instruction, while the other group received PEAK-DT program instruction twice a week. The results suggested a statistically significant change in scores from pre-test to post-test for the PEAK

training group, but not for the treatment as usual group. Another study evaluated the effectiveness of the PEAK-DT module when implemented by classroom teachers and direct care staff (Dixon et al. 2018). In this study, an intervention group (19 participants) received PEAK instruction, while a quasi-randomized control group (15 participants) received treatment as usual (i.e., typical classroom instruction). PEAK-DT assessments were conducted prior to and 1 year following the intervention. The results suggested the PEAK instruction group gained more language skills than that of the control group, and the difference was statistically significant (PEAK: $M = 16.0$, $SD = 17.8$; control: $M = 6.1$, $SD = 14.4$, $F(1,33) = 10.66$, $p < 0.05$). This study supports the effectiveness of the PEAK curriculum when implemented by teachers and direct care staff, who typically have minimal training in terms of behavior analytic program instruction. Since the publication of this study, there has not been a study published utilizing a group research design for any of the other PEAK modules. Despite the lack of group design research on PEAK, significant evidence has been reported using single subject methodology which supports the use of the PEAK Relational Training System in application with autism and related disabilities.

Although the PEAK Relational Training System has generated research supporting it in terms of reliability, validity, and effectiveness, there are a few limitations worth discussing. First, only a small percentage of the overall programs have been evaluated in terms of their effectiveness. Each module contains 184 items within the curriculum. As such, research has not been conducted for every item in terms of its effectiveness in teaching the targeted skill. Second, it is recommended that program instruction for all PEAK modules be conducted synchronously; however, the extent to which the modules interact with each other has not yet been evaluated. Furthermore, normative data has not been collected on the PEAK-E or PEAK-T modules. As a result, the applicability of these modules to certain age ranges is not known. Another consideration is that reliability and validity have not yet been assessed on the PEAK-E or PEAK-T modules. Therefore,

information regarding the relative content and consistency of these modules is limited at this time. A final topic worth noting is that although the PEAK system was designed to be implemented by frontline staff and clinicians, most of the literature surrounding PEAK has been conducted by individuals familiar with PEAK programming. A few studies have incorporated the use of trainers not explicitly trained on the PEAK programming methods; however, limited research exists regarding the extent to which familiarity with programming may have on treatment outcomes.

Taken together, the PEAK Relational Training System contains an assessment and curriculum designed to identify specific skill deficits and teach the targeted skills to individuals with autism and related disabilities. Research supports the PEAK modules as a reliable and valid assessment tool, and effectiveness data support the use of the curriculum in teaching targeted skills to individuals. Overall, the data suggest the PEAK Relational Training System has demonstrated applicability to individuals with autism and related disabilities.

See Also

► [Peabody Picture Vocabulary Test](#)

References and Reading

- Dixon, M. R. (2014a). *The PEAK relational training system: Direct training module*. Carbondale: Shawnee Scientific Press.
- Dixon, M. R. (2014b). *The PEAK relational training system: Generalization module*. Carbondale: Shawnee Scientific Press.
- Dixon, M. R. (2015). *The PEAK relational training system: Equivalence module*. Carbondale: Shawnee Scientific Press.
- Dixon, M. R. (2016). *The PEAK relational training system: Transformation module*. Carbondale: Shawnee Scientific Press.
- Dixon, M. R., Belisle, J., Whiting, S. W., & Rowsey, K. E. (2014a). Normative sample of the PEAK Relational Training System: Direct training module and subsequent comparisons to individuals with autism. *Research in Autism Spectrum Disorders*, 8, 1597–1606. <https://doi.org/10.1016/j.rasd.2014.07.020>.

- Dixon, M. R., Carman, J., Tyler, P. A., Whiting, S. W., Enoch, M. R., & Daar, J. H. (2014b). PEAK relational training system for children with autism and developmental disabilities: Correlations with Peabody picture vocabulary test and assessment reliability. *Journal of Developmental and Physical Disabilities, 26*, 603–614. <https://doi.org/10.1007/s10882-014-9384-2>.
- Dixon, M. R., Whiting, S., Rowsey, K. E., & Belisle, J. (2014c). Assessing the relationship between intelligence and the PEAK relational training system. *Research in Autism Spectrum Disorders, 8*, 1208–1213. <https://doi.org/10.1016/j.rasd.2014.05.005>.
- Dixon, M. R., Belisle, J., Stanley, C., Rowsey, K., Daar, J. H., & Szekely, S. (2015). Toward a behavior analysis of complex language for children with autism: Evaluating the relationship between PEAK and the VB-MAPP. *Journal of Developmental and Physical Disabilities, 27*(2), 223–233.
- Dixon, M. R., Stanley, C., Belisle, J., & Rowsey, K. E. (2016). The test-retest and interrater reliability of the promoting the emergence of advanced knowledge-direct training assessment for use with individuals with autism and related disabilities. *Behavior Analysis: Research and Practice, 16*, 34–40. <https://doi.org/10.1037/bar0000027>.
- Dixon, M. R., Belisle, J., McKeel, A., Whiting, S., Speelman, R., Daar, J. H., & Rowsey, K. (2017). An internal and critical review of the PEAK relational training system for children with autism and related intellectual disabilities: 2014–2017. *The Behavior Analyst, 40*(2), 493–521.
- Dixon, M. R., Belisle, J., Stanley, C. R., & Rowsey, K. (2018). Student outcomes after 1 year of front line staff implementation of the PEAK curriculum. *Behavioral Interventions, 33*, 185.
- Dunn, D. M., & Dunn, L. M. (2007). *Peabody picture vocabulary test: Manual*. Minneapolis: Pearson.
- Malkin, A., Dixon, M. R., Speelman, R., & Luke, N. (2016). Evaluating the relationship between the PEAK relational training system – Direct training module, assessment of basic language and learning skills – Revised, and the Vineland adaptive behavior scales – II. *Journal of Developmental and Physical Disabilities, 28*(1), 1–12. <https://doi.org/10.1007/s10882-016-9527-8>.
- Martin, N. A., & Brownell, R. (2011a). *Expressive one-word picture vocabulary test 4*. Novato: Academic Therapy Publications.
- Martin, N., & Brownell, R. (2011b). *Receptive one-word picture vocabulary test*. Novato: ATPAssessment, a division of Academic Therapy Publications.
- McKeel, A. N., Dixon, M. R., Daar, J. H., Rowsey, K. E., & Szekely, S. (2015a). The efficacy of the PEAK relational training system using a randomized controlled trial of children with autism. *Journal of Behavioral Education, 24*, 230–241. <https://doi.org/10.1007/s10864-015-9219-y>.
- McKeel, A. N., Rowsey, K. E., Dixon, M. R., & Daar, J. (2015b). Correlation between PEAK relational training system and one-word picture vocabulary tests. *Research in Autism Spectrum Disorders, 12*, 34–39. <https://doi.org/10.1016/j.rasd.2014.12.007>.
- Partington, J. (2008). *Assessment of basic language and learning skills-revised (The ABLSS-R)*. Pleasant Hill: Behavior Analysts.
- Rowsey, K. E., Belisle, J., & Dixon, M. R. (2015). Principal component analysis of the PEAK relational training system. *Journal of Developmental and Physical Disabilities, 27*, 15–23. <https://doi.org/10.1007/s10882-014-9398-9>.
- Rowsey, K. E., Belisle, J., Stanley, C. R., Daar, J. H., & Dixon, M. R. (2017). Principal component analysis of the peak generalization module. *Journal of Developmental and Physical Disabilities, 29*(3), 489–501.
- Sparrow, S., Cicchetti, D., & Balla, D. (2005). *Vineland-II: Vineland adaptive behavior scales*: (2nd ed.). Circle Pines, MN: American Guidance Services.
- Sundberg, M. L. (2008). *VB-MAPP: Verbal behavior milestones assessment and placement program*. Concord: AVB Press.

PEAK: Promoting the Emergence of Advanced Knowledge

► PEAK Relational Training System

Pedantic Speech

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Definition

Pedantic speech refers to an overly formal speaking style that is inappropriate to the conversational setting. It can be characterized by didactic patterns of prosody and very precise articulation, as well as unnecessarily complex vocabulary. Some individuals diagnosed with Asperger syndrome speak in this manner, which can be an impediment to social interactions as a conversational partner may

interpret this type of speech as condescending. Pedantic speech is particularly marked in children as it is developmentally inappropriate; such children are often described as “little professors” for their very precise style of speaking.

See Also

- [Intonation](#)
- [Monotone](#)
- [Prosody](#)

References and Reading

- Diehl, J. J., & Berkovits, L. (2010). Is prosody a diagnostic and cognitive bellwether of autism spectrum disorders? In A. Harrison (Ed.), *Speech disorders: Causes, treatments, and social effects* (pp. 159–176). New York: Nova Science.
- Ghaziuddin, M., & Gerstein, L. (1996). Pedantic speaking style differentiates Asperger syndrome from high-functioning autism. *Journal of Autism and Developmental Disorders*, 26, 585–595.
- McCann, J., & Peppé, S. (2003). Prosody in autism spectrum disorders: A critical review. *International Journal of Language & Communication Disorders*, 38(4), 325–350.
- Paul, R., Augustyn, A., Klin, A., & Volkmar, F. R. (2005). Perception and production of prosody by speakers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 35(2), 205–220.
- Shriberg, L. D., Paul, R., McSweeney, J. L., Klin, A., Cohen, D. J., & Volkmar, F. R. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning autism and Asperger syndrome. *Journal of Speech, Language, and Hearing Research*, 44(5), 1097–1115.

PEDI

- [Pediatric Evaluation of Disability Inventory \(PEDI\)](#)

PediaCare® Children's Allergy [OTC]

- [Diphenhydramine](#)

PediaCare® Children's NightTime Cough [OTC]

- [Diphenhydramine](#)

Pediatric Autism Communication Therapy (PACT Therapy)

- [Autism Family Experience Questionnaire \(AFEQ\)](#)

Pediatric Evaluation of Disability Inventory (PEDI)

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Synonyms

[PEDI](#)

Description

The Pediatric Evaluation of Disability Inventory (PEDI; Haley et al. 1992) is a clinical assessment instrument designed for use with children aged 6 months to 7.5 years who have disabilities resulting in delays or impairments in functional independence. Specific applications of the PEDI include ascertaining the extent of functional delay in children, monitoring progress in rehabilitation and intervention programs, and measuring outcomes in therapeutic and educational programs. Though the instrument also may be given as part of an initial developmental or diagnostic assessment, caution is warranted because extensive validation studies of using the PEDI for this purpose have not been completed.

Administration of the PEDI is typically completed through a 45–60-min structured interview with the parent or primary caregiver, direct assessment by therapists or educators who are familiar with the child, or a combination of these two approaches. If there are severe time constraints, the PEDI may be completed directly by the parents as a “paper-and-pencil” checklist; however, the authors strongly recommend that a clinician review responses carefully with the parent and interpret the resultant data with caution.

PEDI content is based on a conceptual model that centers upon identifying the child’s functional limitations in the context of developmental expectations, task conditions, and caregiver assistance. Consistent with this model, the PEDI has three sets of scales (i.e., functional skills, caregiver assistance, and modifications), each of which is organized by three “content areas” (i.e., self-care, mobility, and social function; see Table 1). The functional skills scales include items on daily living activities, such as use of utensils, toilet transfers, and interactive play. The caregiver assistance scales assess the amount of help a child requires to complete functional skills, with responses rated on a 6-point rating system that is “weighted” so that the greatest degree of specification is provided at the “higher” end of the scale (i.e., toward the independent end). The modifications scale presents the same items as the caregiver assistance scale but asks caregivers to assess the degree to which environmental modifications and equipment are necessary for use by the child to complete functional or routine daily living

activities. The ratings from the modifications scale cannot be converted into normative data; therefore, they can only be analyzed as frequency counts.

Historical Background

The Pediatric Evaluation of Disability Inventory (PEDI; Haley et al. 1992) was developed at the New England Medical Center and first published in 1992. The development and testing of the test in the prior decade noted favorable comparison to the Battelle. The PEDI was designed to assess the functional independence of children with developmental disabilities. Scaled scores in multiple domains estimate the child’s capabilities. It has been used internationally, and in 2008, a computer-assisted version was developed (i.e., the Pediatric Evaluation of Disability Inventory-Computer Adaptive Test or PEDI-CAT).

Psychometric Data

For each of the functional skills scales and the caregiver assistance scale, two different domain scores can be determined: “normative standard scores” and “scaled scores.” The normative standard scores have a mean of 50 and a standard deviation of 10 and compare the individual child’s functioning to a group of children within the same age range in the normative sample. The normative group is composed of a small sample of

Pediatric Evaluation of Disability Inventory (PEDI), Table 1 Organization of the PEDI

Scales	Number of content items			Rating scale	Scoring system
	Self-care	Mobility	Social function		
Functional skills	63	59	65	Dichotomous	Standard scores ^a and scaled scores ^b
				Unable/capable	
Caregiver assistance	8	7	5	6-point scale	Standard scores ^a and scaled scores ^b
				“Total assistance” to “independent”	
Modifications	8	7	5	4-point scale	Frequency counts
				“None” to “extensive”	

^aMean of 50, standard deviation of 10
^bRange from 0 to 100 and used to compare child’s relative strengths and weaknesses

412 typically developing children residing in the Northeastern United States. This sample was composed of a nearly equal number of males and females (49.3% and 50.7%, respectively). Distribution by race is commensurate with 1980 US census data, with the exception of an overrepresentation of African-American children (18.7% in the normative sample versus 11.7% in the US population).

The “scaled scores” are used to provide an estimate of functional independence across the items that compose each of the scales by comparing the child’s performance relative to the maximum number of points possible (Haley et al. 1992). The scaled score range is 0–100, with “0” reflecting low functioning and “100” reflecting high functioning. These scores can be graphed on the score form for analysis of an individual child’s relative strengths and weaknesses. As the authors point out, these scaled scores do not take into account the age of the child but, rather, provide an estimate of the child’s capability in each content domain. To this end, scores can be plotted on a “score profile” located on the score summary page of the score form for visual analysis of a child’s relative strengths and weaknesses. Confidence intervals are provided for both normative standard scores and scaled scores, and a 95% confidence interval is recommended by the authors.

The PEDI manual contains evidence of high internal consistency (i.e., the degree to which the items in each of the domains measure a similar concept). Internal consistency has since been replicated and found to be adequate in a population of children with cerebral palsy (McCarthy et al. 2002). Additional evidence of instrument reliability is provided when comparing the results of separate PEDI administrations by two different examiners with two respondents who know the child well (e.g., a child’s parent and member of his or her rehabilitation team). Interrater/intrarater reliability was shown to be fairly consistent when two caregivers of a child were interviewed by the same clinician (Berg et al. 2004). However, when multiple clinicians interviewed providers, reliability of results was lower (Berg et al. 2004). Test-retest reliability data are not available.

The PEDI authors provide evidence of satisfactory validity on several dimensions. The results of reviews of the instrument by developmental experts suggest that the PEDI does measure the presence of a pediatric functional disability (i.e., content validity). A strong positive correlation between the age of the children in the normative sample and their PEDI scores suggests that the skills measured by the instrument are closely tied to overall child development (i.e., construct validity). Scores on the PEDI were found to be strongly correlated with scores on the Battelle Developmental Inventory Screening Test (BDIST; Newborg et al. 1984) for both a group of typically developing children and a group of children with disabilities (Feldman et al. 1990). Evidence of strong discriminant validity is provided in the PEDI manual as well, suggesting that the instrument effectively distinguishes between children with and without disabilities. A comparison of PEDI scores of a group of children at hospital intake for traumatic injuries with their follow-up scores at 1 and 6 months postrehabilitation showed that the PEDI detected change in the functional status of children over time, suggesting evaluative validity (i.e., the ability of the instrument to detect change in functional status of the individual).

Since its publication in 1992, the PEDI has been translated and used in several other countries with solid psychometric results (e.g., Ganotti and Cruz 2001; Wassenberg-Severijnen et al. 2003), suggesting that the instrument is a good measure of overall functional skill development. A recent attempt to abbreviate the measure and make a computer version shows promise (Coster et al. 2008). The PEDI has been utilized with a variety of populations (e.g., premature infants, brain injuries, autism, cerebral palsy) but may be best suited for populations with severe motor deficits. For example, while the PEDI has been consistently shown to be effective in identifying motor progress of children with cerebral palsy (e.g., Vos-Vromans et al. 2005), it is less sensitive in detecting subtle deficits of children with primary language impairments (Mayrand et al. 2009).

In 2012, a version of the computer-assisted version of the PEDI specifically for use with children

with ASD, the PEDI-CAT ASD, was developed (Kramer et al. 2012). Subsequent research provides some evidence that this version demonstrates strong test-retest reliability and high construct validity with children with ASD and that this instrument may be preferable over administration of lengthier instruments, such as the Vineland Adaptive Behavior Scales (Kramer et al. 2016).

Clinical Uses

For children with autism, the PEDI is most likely to be administered as part of an intake evaluation (e.g., such as during referral for early intervention services) or when there is a concern about additional physical disabilities or delays that impact the independent functioning of the child. Given the “prompt dependency” that many children with ASD demonstrate (i.e., the necessity of caregivers to direct a child to initiate and/or complete a task that is otherwise within his or her skill repertoire), the PEDI may be a useful tool for monitoring independence and timely use of functional skills. Repeated administrations of the PEDI as part of ongoing treatment for a child with ASD may allow caregivers and clinicians the ability to monitor progress with functional skills over time. Given that the data used to establish the norms were collected over 30 years ago, it is recommended that normative standard scores be interpreted with great caution.

The PEDI is most useful in the assessment of children with motor disabilities such as children who were premature infants or who have cerebral palsy.

See Also

- [Functional Life Skills](#)
- [Vineland Adaptive Behavior Scales \(VABS\)](#)

References and Reading

Berg, M., Jahnsen, R., Frøslie, K., & Hussain, A. (2004). Reliability of the pediatric evaluation of disability inventory (PEDI). *Physical & Occupational Therapy in Pediatrics*, 24, 61–77.

- Coster, W., Halye, S., Ni, P., Duams, H., & Fragala-Pinkham, M. (2008). Assessing self-care and social functioning using a computer adaptive testing version of the pediatric evaluation of disability inventory. *Archives of Physical Medicine and Rehabilitation*, 89(4), 622–629.
- Feldman, A., Haley, S., & Coryell, J. (1990). Concurrent and construct validity of the pediatric evaluation of disability inventory. *Physical Therapy*, 70, 602–610.
- Ganotti, M., & Cruz, C. (2001). Content and construct validity of a Spanish translation of the pediatric evaluation of disability inventory for children living in Puerto Rico. *Physical & Occupational Therapy in Pediatrics*, 20(4), 7–24.
- Haley, S., Coster, W., Ludlow, L., Haltiwanger, J., & Andrellos, P. (1992). *Pediatric evaluation of disability (PEDI)*. Boston: New England Center Hospitals/PEDI Research Group.
- Kramer, J. M., Coster, W. J., Kao, Y.-C., Snow, A., & Orsmond, G. I. (2012). A new approach to the measurement of adaptive behavior: development of the PEDI-CAT for children and youth with autism spectrum disorders. *Physical & Occupational Therapy in Pediatrics*, 32(1), 34–47. <https://doi.org/10.3109/01942638.2011.606260>.
- Kramer, J. M., Liljenquist, K., & Coster, W. J. (2016). Validity, reliability, and usability of the pediatric evaluation of disability inventory-computer adaptive test for autism spectrum disorders. *Developmental Medicine and Child Neurology*, 58, 255–261. <https://doi.org/10.1111/dmcn.12837>.
- Mayrand, L., Mazier, B., Menard, S., & Chiningaryan, G. (2009). Screening for motor deficits using the pediatric evaluation of disability inventory (PEDI) in children with language impairment. *Developmental Neuropsychology*, 12(3), 139–145.
- McCarthy, M., Silberstein, C., Atkins, E., Harryman, S., Sponseller, P., & Hadley-Miller, N. (2002). Comparing reliability and validity of pediatric instruments for measuring health and well-being of children with spastic cerebral palsy. *Developmental Medicine and Child Neurology*, 44(7), 468–476.
- Newborg, J., Stock, J., Wnek, L., & Svinicki, J. (1984). *Battelle developmental inventory*. Allen: DLM Teaching Resources.
- Vos-Vromans, D., Ketelaars, M., & Gorter, J. (2005). Responsiveness of evaluative measures for children with cerebral palsy: The gross motor function measure and the pediatric evaluation of disability inventory. *Disability and Rehabilitation*, 27(20), 1245–1252.
- Wassenberg-Severijnen, J., Custers, J., Hox, J., Vermeer, A., & Helders, P. (2003). Reliability and validity of the Dutch pediatric evaluation of disability inventory (PEDI). *Clinical Rehabilitation*, 17, 457–462.

Pediatric Onset Schizophrenia

- [Childhood Schizophrenia](#)

Pediatric Speech Intelligibility Test

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Synonyms

PSI test

Description

The Pediatric Speech Intelligibility (PSI) test is a closed-set test composed of 20 monosyllabic words and a 10-sentence procedure. The monosyllabic word lists consist of simple nouns like “bear” and “fork.” The sentences consist of two formats. In the first format, the sentences are composed of a noun phrase, verb-ing, and a noun phrase and are preceded by the carrier phrase “show me.” An example of a sentence in the first format is “Show me a rabbit painting an egg.” In the second format, the sentences are composed of a noun phrase, auxiliary verb-ing, and a noun phrase. An example of a sentence in the second format is “A rabbit is painting an egg” (Jerger et al. 1980; Northern and Downs 2002). The child is instructed to point to one of the five pictures corresponding to the sentence or word that is heard (Jerger et al. 1981). The PSI test can be administered in quiet, as well as in the presence of a competing message.

Historical Background

The Pediatric Speech Intelligibility (PSI) test was developed to evaluate both peripheral and central components of auditory disorders in children between the ages of 3 and 6 years. The test takes into consideration children’s receptive language function since the test stimuli were generated by typically developing children between 3 and 7 years of age. The word and sentence stimuli

were elicited by picture stimulus cards which were chosen from lists of words and actions comprising the vocabularies of young children (Jerger et al. 1980).

Psychometric Data

Validity studies using the PSI test indicate that test performance on the Format I and Format II sentences was significantly different in children between the ages of 3 and 6 depending on their chronological age as well as receptive language ability. Performance on the monosyllabic word materials did not change with receptive language abilities. Trends in chronological age were evident for both the word and sentence materials (Jerger et al. 1981). Performance intensity functions were reported in quiet and in noise and performance reached 100% in children with normal hearing on words and sentences in both quiet and noise at a presentation level of 50 dB SPL (Jerger and Jerger 1982). Test-retest measures indicated high test-retest reliability for both normal hearing and hearing-impaired children (Jerger et al. 1983). Furthermore, the PSI test has been shown to have high sensitivity and specificity to central auditory nervous system lesions in children (Jerger 1987).

Clinical Uses

The PSI test has been used to evaluate speech intelligibility in quiet and in noise in a variety of populations, both children and adult as follows: speech intelligibility in children with recurrent otitis media (Jerger et al. 1983), central nervous system lesions (Jerger et al. 1983; Jerger 1987), and hearing impairment (Jerger et al. 1983). In addition, the PSI test has been used to evaluate speech intelligibility in children with cochlear implants and hearing aids (Somers 1991). Clinically, the person administering the PSI should take into consideration the cognitive, hearing, speech, and language abilities of the individual being tested as these factors may affect outcomes. Thus, the PSI is not typically administered to children with autism, and performance on this test should be interpreted with caution in this population.

See Also

► Central Auditory Processing Disorder

References and Reading

- Jerger, S. (1987). Validation of the Pediatric Speech Intelligibility test in children with central nervous system lesions. *Audiology*, 26(5), 298–311.
- Jerger, S., & Jerger, J. (1982). Pediatric Speech Intelligibility test: Performance-intensity characteristics. *Ear and Hearing*, 3, 325–334.
- Jerger, S., Lewis, S., Hawkins, J., & Jerger, J. (1980). Pediatric Speech Intelligibility Test I. Generation of test materials. *International Journal of Pediatric Otorhinolaryngology*, 2(3), 217–230.
- Jerger, S., Jerger, J., & Lewis, S. (1981). Pediatric speech intelligibility test II. Effect of receptive language age and chronological age. *International Journal of Pediatric Otorhinolaryngology*, 3(2), 101–118.
- Jerger, S., Jerger, J., & Abrams, S. (1983a). Speech audiometry in the young child. *Ear and Hearing*, 4(1), 56–66.
- Jerger, S., Jerger, J., Alford, B., & Abrams, S. (1983b). Development of speech intelligibility in children with recurrent otitis media. *Ear and Hearing*, 4(3), 138–145.
- Northern, J., & Downs, M. (2002). *Hearing in children* (5th ed.). Philadelphia: Lippincott, Williams, & Wilkins.
- Somers, M. (1991). Speech perception abilities in children with cochlear implants and hearing aids. *Otology & Neurotology*, 12(Suppl), 174–178.

Peer Mentors for Students with ASD on College Campuses

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Definition

A peer mentor is an individual with an expected level of experience in a particular area who

provides support to a similar-aged individual with less experience in that area (mentee). The critical roles of peer mentors on college and university campuses include support in coursework, degree completion, and navigation of social networks.

In this model, peer mentors typically share common characteristics, attributes, or circumstance with the mentee in such areas as age, ability, interests, or community environment (i.e., school, classes, dormitories, etc.). The desirable traits and skills of peer mentors include patience, flexibility, and effective communication. Peer mentors typically receive training that provides them with skills such as effectively using peer modeling to demonstrate appropriate utilization of targeted skills, facilitating interaction, and providing indirect guidance and positive reinforcement (Battaglia and Radley 2014). The peer mentor/mentee relationship should be one of reciprocity (Gilman 2006); therefore, at its very core, this model provides support on a social level.

Historical Background

Over the last 10–15 years, there has been an increase in the number of adults with autism spectrum disorder (ASD) who are capable of engaging in postsecondary education. The literature indicates, however, that many of these individuals either do not attend an institution of postsecondary education or, when they attend, struggle with successful completion of a terminal degree (Shattuck et al. 2012). Overarching factors that negatively impact success include challenges in the areas of social interaction, executive function, and emotional regulation (Adreon and Durocher 2007; Gotham et al. 2015). Many who do complete a terminal degree experience difficulty securing gainful employment; this appears to be connected to ongoing challenges with social interaction and poorly developed social capital (Dipeolu et al. 2015).

Social capital refers to connectedness and engagement with individuals, organizations, and communities. Higher levels of social capital are linked to better outcomes in education,

employment, health, political participation, safety, and well-being for all individuals (Clark et al. 2015; Hill 2011; Seibert et al. 2001). Building social capital takes time and investment and a strong capacity for socially interacting with a variety of individuals. Students with ASD who attend postsecondary education institutions have difficulty building social capital due to numerous challenges they face in areas such as time management, social interaction, emotional regulation, and navigation of the curriculum.

Many students with ASD, who received support or accommodations in secondary education, no longer receive assistance at their postsecondary education institutions for a variety of reasons, including a lack of willingness to disclose their disability. Furthermore, there is a lack of knowledge and understanding of autism by peers, student affairs staff, and faculty which further impedes potential natural support across multiple settings (Van Hees et al. 2015). Gelbar et al. (2014) reviewed 20 articles that focused on the collegiate experiences and/or support of students with ASD. Commonly reported emotions among these students included anxiety, loneliness, depression, and peer rejection; all factors possibly related to poorly developed peer networks and lower levels of social capital. The authors concluded that there is a scarcity of research on postsecondary education experiences of students with ASD, noting that most articles are case studies and very few are empirical in nature.

It is crucial to identify the most effective supports to address core challenges so that these capable individuals will be able to live up to their potential to complete a postsecondary education degree. One such support, commonly referred to as peer-mediated intervention or peer mentoring, is rising in popularity. Given the current ubiquity and success of peer mentor programs on college and university campuses, it would appear to be a reasonable approach with significant potential. Peer mentors can assist students with ASD by modeling social behavior, assisting in making new friends, and supporting involvement in extracurricular activities, areas critical for the development of a peer network and increased levels of social capital.

Current Knowledge

Evidence exists to support the value of the use of peer mentors in the college setting for all students, particularly for incoming freshmen. These types of interactions provide the opportunity for students to build social networks. Social networks that are developed with faculty, staff, peers, friends, and mentors are strongly linked to student success, e.g., satisfaction, persistence, etc. (Kuh et al. 2012). In addition to acknowledging the effectiveness of peer mentors for all students at the college level, evidence of how to use peer mentors to increase social and academic success for individuals with ASD is needed if this is to be considered a reliable support. There is ample evidence in the K-12 setting of the effectiveness of the use of peer mentors to assist students with ASD in improving social interaction, emotional regulation skills, and academic skills (Hart et al. 2010; Tichenor 2016).

The strategies used in studies of peer-mediated intervention (PMI) in the secondary education population may be useful for students in postsecondary education. Bambara et al. (2016) trained a group of high school students for a period of 18 weeks, 3–4 days per week, to serve as conversational peers to three students with ASD. At the end of the intervention period, the authors found an increase in the following: number of conversational acts, initiation and length of conversations, asking of follow-up questions, making comments, and assertive conversation acts. The authors cited the necessity of systematic intervention in training the peer mentors, concluding that proximity alone would not be sufficient to promote the social interaction. Although there is a paucity of research that addresses the utilization of peer mentors in assisting students with ASD in institutions of postsecondary education, it is reasonable to assume that many of the same factors that have led to success with younger students would also apply to this age group.

Peer mentors have been observed to be helpful in modeling appropriate social behavior and assisting students with ASD in meeting new friends and broadening their friendship circle at the precollege level. However, given the shift in

the structure and ambiguity of social engagement that occurs at the college or university level, it is necessary to investigate the feasibility of implementing a structured, formal peer mentor program at this level. Neville and White (2011) surveyed college students with typical development (TD) regarding their openness to peers who demonstrate behaviors typical of individuals with ASD. Results indicated that students who have a first-degree relative with ASD are more open to college peers with ASD and are more likely to be comfortable socializing and/or living in close proximity to them, suggesting a link between awareness and comfort levels. A follow-up study conducted by Gardiner and Iarocci (2014) revealed similar findings. In addition, they found that student acceptance of individuals with disabilities increased the possibility of intent to volunteer. Therefore, if a college or university systematically increases awareness of autism, there will potentially exist a strong pool of peer mentors to assist with the development of social networks, thus leading to increased social capital and the fostering of success in the ASD college population.

Several studies have explored the use of systematically trained and engaged peer mentors at the college or university level geared toward students with ASD. A 1-year longitudinal study (Madriaga et al. 2008) was completed in the United Kingdom that examined the postsecondary education experiences of 1st-year students with ASD. Eight students, across six different universities, were followed for a period of 1 year. Among the goals of the study were to enable a wider group of students with ASD to achieve postgraduate education, raise the profile of students with ASD, promote issues of awareness and disability among staff, and understand societal barriers in order to facilitate a more successful transition to the postsecondary education setting. Six of the eight students were assigned mentors at some point during the year. The narrative reports of the experience with the peer mentors were largely positive. The mentors were viewed as mediators, counselors, or simply as individuals who were available to help. This further supports the feasibility and positive impact of the use of peer mentors.

Weiss and Rohland (2015) reported success in the use of a form of peer mentor support at the University of Rhode Island (URI) in their Communication Coaching Program. Group communication coaching sessions for students with ASD focused on teaching skills to support social communication and executive function. Peer coaches, paired with these students, reinforced the skills in natural settings across the campus. Over the course of 5 years, 55 students (which represented 42% of the students who self-identified with ASD) chose to participate in the program. Progress reports written by the coaches revealed growth in social communication skills, particularly for conversational management; all participants were successful in completing their degrees at URI. The authors of this program acknowledged a need for additional systematic data to further support the effectiveness of peer coaches on the success of this population of students.

Ames et al. (2015) reported on a relatively long-standing mentorship program at York University in Toronto. Overarching goals of the program were for students to feel a sense of belonging within the university community and to assist the students in building a peer network while building social, organizational, and vocational skills. Students were paired with graduate student mentors from the clinical psychology program who had extensive clinical experience and academic knowledge of ASD. Mentors and mentees met weekly, and there were additional group meetings and events that took place regularly. Evaluation of the success of the program was based on a number of measures, including personal interviews, year-end evaluations, student participation, student satisfaction, etc. More than half of the mentees reported meeting weekly with their mentors and high levels of satisfaction in achieving their goals.

Siew et al. (2017) described a mentoring program at Curtin University in Perth Western Australia in which ten undergraduate students with ASD were individually mentored by an undergraduate or graduate student for up to 2 h weekly for one semester. The mentors and mentees also attended weekly group sessions that addressed a number of different topics. The mentors participated in a training program and

met weekly for group supervision sessions. Questionnaires were completed by the mentees pre- and post-study. Results indicated a perceived increase in social support and a reduction in general communication apprehension. Overall anxiety scores, however, did not demonstrate a pre-post change. Qualitative results indicated three positive features of the program: constant and stable support, comfort of peer-to-peer support, and flexible and individualized support. Mentors reported a high level of participant satisfaction, demonstrated a 29% pre-post increase in acquisition of knowledge regarding autism, and reported an increased awareness of some challenges faced by individuals with autism, including sensory differences, anxiety, and underlying cognitive processes (Hamilton et al. 2016).

In a unique approach, Cook and Weiss (2016, 2017) reported on a 3-year pilot study in which the building of social networks was accomplished through an undergraduate college class. The course, comprised of students with ASD or other social communication challenges and undergraduate students with typical development, provided students with an in-depth study of social communication and cognition and provided a context to support students with personal or professional challenges. Graduate students of speech-language pathology participated in the study as student teachers. The classroom environment was designed to facilitate the incidental development of peer support networks. Daily group activities and regular rotation of students in the class among the groups ensured that there would be ample opportunity for students to interact with all members of the class. Typical activities included role-play scenarios, review and analysis of targeted movie scenes, and the creation of group and/or individual videos for analysis. In addition, readings on peer mentoring were assigned and were incorporated into focused discussions with the intent of facilitating mentoring opportunities within and external to the class for the students.

Student course reviews indicated a very high level of satisfaction with the class. Pre-post surveys demonstrated an increase in some measures of overall level of comfort toward individuals with ASD among the students in the class with

typical development. In pre-post questionnaires, the student teachers demonstrated a statistically significant increase in multiple aspects of understanding and feeling comfortable facilitating peer mentorship activities, while course participants demonstrated a statistically significant increase in a basic level of understanding peer mentorship. Observations of the class during a pre- and post-class social activity indicated increased social interaction and initiation among all students. The advantage of such an indirect approach to peer mentorship is the potentially more naturalistic development of peer networks when compared to a more systematic, direct instructional approach such as the one described by Bambara et al. (2016). In one instance, a peer mentor-mentee relationship developed between two of the course participants. Both individuals reported that the opportunities provided in the class contributed to the development of their relationship. In addition, the indirect approach reduces the necessity for the student with autism to self-disclose.

Future Directions

As the number of students with ASD continues to grow at the college and university levels, it will be necessary to provide appropriate supports and services that enable these students to successfully complete their programs of study. Although student resource support offices typically provide academically based supports, they often do not provide adequate support in the areas of social interaction and communication. In addition to experiencing academic challenges related to executive function, individuals with ASD face greater challenges in social interaction skills and require support in these areas in order to be afforded an equitable opportunity for success in their programs of study.

Given the past and current successful use of peer mentors for all students at the college or university level, it is imperative that these institutions continue to explore and expand upon this support. Fortunately, there currently exist a growing number of programs that provide peer mentors for students with ASD that can be tapped to learn

about the best approaches for a particular college or university. In addition, there is clear evidence that supports ease of implementation of such programs when they are combined with either existing peer mentor programs or a college level course. It is possible to provide training to existing peer mentor volunteers in the area of ASD while working toward developing a more systematic program. This approach is supported by the research of Nevill and White (2011), Gardiner and Iarocci (2014), and Cook and Weiss (2016, 2017a, b) that has demonstrated that increasing knowledge and awareness of ASD and facilitating contact with individuals with ASD increases the willingness to support these individuals.

A review of the literature reveals a paucity of empirical studies that have investigated the effectiveness of peer mentors on postsecondary outcomes for college-aged students with ASD. Most of the literature consists of qualitative analyses and includes narrative accounts of approaches used to implement peer mentor programs. Although these latter articles are promising, additional empirical research is needed to determine whether or not peer mentoring can be adopted as an evidence-based practice for this population. In order to increase the quantity of empirical research, collaborations between practitioners and researchers should be encouraged. Together they can identify the most appropriate empirical research design to study the effects of systematically developed (direct) and/or naturally occurring (indirect) peer mentor-mentee relationships. Critical outcomes to be measured are the impact on degree completion, gainful employment, and positive community engagement.

See Also

- [Peer-Mediated Intervention](#)
- [Peers, Exposure to](#)

References and Reading

- Adreon, D., & Durocher, J. (2007). Evaluating the college transition needs of individuals with high-functioning autism spectrum disorders. *Intervention in School and Clinic, 42*(5), 271–279.
- Ames, M., McMorris, C., Alli, L., & Bebko, J. (2015). Overview and evaluation of a mentorship program of university students with ASD. *Focus on Autism and Other Developmental Disabilities, 31*(1), 27–36.
- Bambara, L., Cole, C., Kunsch, C., Tsai, S., & Ayad, E. (2016). A peer-mediated intervention to improve the conversational skills of high school students with Autism Spectrum Disorder. *Research in Autism Spectrum Disorders, 27*, 29–43.
- Battaglia, A., & Radley, K. (2014). Peer-mediated social skills training for children with Autism Spectrum Disorder. *Beyond Behavior, 23*(2), 4–13.
- Clark, G., Marsden, R., Duncan-Whyatt, J., Thompson, L., & Walker, M. (2015). It's everything else you do...: Alumni views on extracurricular activities and employability. *Active Learning in Higher Education, 16*(2), 133–147.
- Cook, B., & Weiss, D. (2016). Social networks: Supporting college/university students with high-functioning autism spectrum disorder. Presented at ASHA Annual Convention, Philadelphia, PA.
- Cook, B., Weiss, D., & Hodge, V. (2017a). Social networks: Lecture, supporting college/university students with high-functioning autism spectrum disorder. Presented at OCALI Annual Conference, Columbus OH.
- Cook, B., Weiss, D., & Hodge, V. (2017b). A facilitated natural mentoring program. *The ASHA Leader, 22*(7), 40–42. <https://doi.org/10.1044/leader.AE.22072017.40>.
- Dipeolu, A., Storlie, C., & Johnson, C. (2015). College students with high-functioning autism spectrum disorder: Best practices for successful transition to the world of work. *Journal of College Counseling, 18*, 175–190.
- Gardiner, E., & Iarocci, G. (2014). Students with Autism Spectrum Disorder in the university context: Peer acceptance predicts intention to volunteer. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-013-1950-4>. Retrieved from <http://autismlab.psyc.sfu.ca/sites/default/files/users/emily/Gardiner%20%26%20Iarocci,%202013.pdf>.
- Gelbar, N., Smith, I., & Reichow, B. (2014). Systematic review of articles describing experience and supports of individuals with autism enrolled in college and university programs. *Journal of Autism and Developmental Disorders, 44*(10), 2593–2601.
- Gilman, D. (2006). The power of peer mentoring. In *Wisconsin healthy & ready to work: A series of materials supporting youth with special health care needs*. Madison: Waisman Center, University of Wisconsin-Madison. Wisconsin Council on Developmental Disabilities.
- Gotham, K., Lounds, T., Warren, Z., Anderson, C., Law, P., Law, J., & Lipkin, M. (2015). Characterizing the daily life, needs, and priorities of adults with ASD from interactive autism network data. *Autism, 19*(7), 794–804.
- Hamilton, J., Stevens, G., & Girdler, S. (2016). Becoming a mentor: The impact of training and the experience of mentoring university students on the autism spectrum. *PLoS One, 11*(4), 1–13.
- Hart, D., Grigal, M., & Weir, C. (2010). Expanding the paradigm: Postsecondary education options for individuals with autism spectrum disorder and intellectual

- disabilities. *Focus on Autism and Other Developmental Disabilities*, 20(10), 1–17.
- Hill, S. (2011). Making connections: The role of social capital in the enhancement of employability of first generation business studies graduates. *Widening Participation and Lifelong Learning*, 13(2), 33–50.
- Kuh, G., Kinzie, J., Buckley, J., Bridges, B., & Hayek, J. (2012). What matters to student success: A review of the literature. Commissioned report for the National Symposium of Postsecondary Student Success: Spearheading a dialog on student success. National Postsecondary Education Cooperative. Retrieved January 21, 2016, from: http://nces.ed.gov/npec/pdf/kuh_team_report.pdf.
- Madriaga, M., Goodley, D., Hodge, N., & Martin, N. (2008). *Enabling transition into higher education for students with Asperger syndrome*. Project report. Higher Education Academy. [internet]. UK: Sheffield Hallam University; [cited 2015 Aug 10]. Available from: http://shura.shu.ac.uk/1004/1/manuel_madriaga_report.pdf.
- Nevill, R., & White, S. (2011). College students' openness toward autism spectrum disorders: Improving peer acceptance. *Journal of Autism and Developmental Disorders*, 41, 1619–1628. <https://doi.org/10.1007/s10803-011-1189-x>.
- Seibert, S., Kraimer, M., & Liden, R. (2001). A social capital theory of career success. *Academy of Management Journal*, 44(2), 219–237.
- Shattuck, P., Narendorf, S., Cooper, B., Sterzing, P., Wagner, M., & Taylor, J. (2012). Postsecondary education and employment among youth with an autism spectrum disorder. *American Academy of Pediatrics*, 129, 1042–1049. <https://doi.org/10.1542/peds.2011-2864>. Retrieved from <http://www.pediatrics.org/cgi/doi/10.1542/peds.2011-2864>.
- Siew, C., Mazzucchelli, T., Rooney, R., & Girdler, S. (2017). A specialist peer mentoring program for university students on the autism spectrum: A pilot study. *PLoS One*, 12(7), 1–18.
- Tichenor, K. (2016). The effects of peer mentoring on students with autism spectrum disorder. *The University of Arkansas Undergraduate Research Journal*, 20, 53–66.
- Van Hees, V., Moyson, T., & Roeyers, H. (2015). Higher education experiences of students with autism spectrum disorder: Challenges, benefits, and support needs. *Journal of Autism and Developmental Disorders*, 45, 1673–1688. <https://doi.org/10.1007/s10803-014-2324-2>.
- Weiss, A., & Rohland, P. (2015). Implementing a communication coaching program for students with autism spectrum disorders in postsecondary education. *Topics in Language Disorders*, 35(4), 345–361.

Peer Victimization

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Definition

Peer victimization is typically defined as being the target, directly or indirectly, of an aggressive action. In this context, aggression is defined as an action/behavior that is intended to harm the target of that action/behavior. Here, it is important to distinguish between the terms peer victimization and bullying, which are often incorrectly used to mean the same thing. Bullying refers to being a target of aggression, but differs from peer victimization in that bullying requires that the aggressor has power – in terms of status, physical size or strength, or psychological control – over the target of the aggression. Thus, all bullying falls under the definition of peer victimization but not all peer victimization falls under the definition of bullying. Peer victimization targeting those with ASD, in most instances, should be classified as bullying since the symptoms of ASD and associated social difficulties make it unlikely that they have more power than someone targeting them. Similarly, in many cases, the aggressive actions by those with ASD would not be classified as bullying since those with ASD often do not have power over the targets of their actions. Instead, much of the aggression that individuals with ASD exhibit is considered reactive aggression rather than instrumental aggression to be used as a tool for maintaining power over the individual being targeted.

Using terms like bullying, teasing, or being mean in the measurement of peer victimization

Peer Supports

► Interpersonal Supports

can be problematic since these terms, from the perspective of participants, can be subjective, interpreted quite broadly, and are often seen as being synonymous with one another. In measuring peer victimization, it is critical to use items that describe the specific aggressive behaviors (e.g., hit, kick, call me mean names) targeted at the victim. This specificity impacts measurement validity and in turn internal validity of studies of peer victimization. It is also important to distinguish between the various forms of peer victimization, such as physical, verbal, electronic, relational, and physical. This is key to building effective intervention strategies since different forms of peer victimization are associated with different outcomes and predictors (see next section).

Historical Background

Not relevant

Current Knowledge

This section will outline the current knowledge about the prevalence of peer victimization in children and adolescents with ASD, as well as the factors found to be associated with peer victimization in this population.

Prevalence of Peer Victimization

Rates. Numerous studies indicate that individuals with ASD tend to be viewed less positively by their typically developing (TD) peers (Bauminger and Kasari 2000; Chamberlain et al. 2007; Humphrey and Symes 2010, 2011; Jones and Frederickson 2010; Locke et al. 2010; Lyons et al. 2011; Petrina et al. 2014; Wright and Wachs 2019). Thus, it is not surprising that children and adolescents with ASD are commonly targeted for peer victimization. Multiple studies have shown that those with ASD experience higher rates of peer victimization than TD peers (Bear et al. 2015; Chan et al. 2018; Humphrey and Symes 2011; Rose et al. 2015; Rowley et al. 2012; Twyman et al. 2010; Wainscot et al. 2008; Zeedyk

et al. 2014) and peers with other disabilities (Humphrey and Symes 2011; Rowley et al. 2012; Twyman et al. 2010; Zeedyk et al. 2014). Parents of children and adolescents with ASD report peer victimization rates ranging from around 40% to 90% across studies (Cappadocia et al. 2012; Little 2002; Maiano et al. 2016; Pfeffer 2016; Sterzing et al. 2012), with variability across studies explained, to some extent, by the time frame used for measuring peer victimization (Maiano et al. 2016; e.g., lifetime vs. weekly occurrence). Nevertheless, even if the true rates of peer victimization for this group fall at the lower end of this range that would indicate a substantial number of individuals with ASD are encountering negative peer experiences.

Types of victimization. A few studies have examined differences in rates for different types of victimization, such as verbal, relational, and physical (Adams et al. 2014; Bear et al. 2015; Kloosterman et al. 2013; Maiano et al. 2016; Nowell et al. 2014; Wainscot et al. 2008; Zeedyk et al. 2014). Overall, these studies find that verbal victimization occurs the most often, which is similar to what is seen in studies of TD children and adolescents. Findings have been mixed when examining frequencies of relational and physical victimization relative to TD peers (Adams et al. 2014; Bear et al. 2015; Kloosterman et al. 2013; Zeedyk et al. 2014). More recently, studies have also examined cybervictimization in those with ASD, finding similarly high rates as those found in more traditional types of peer victimization. Further, cybervictimization was associated with depression and anxiety (Ashburner et al. 2019; Kowalski and Fedina 2011; Wright and Wachs 2019).

Factors Associated with Peer Victimization

To obtain a more complete picture of peer victimization in children and adolescents with ASD, some studies have begun to test the associations between peer victimization and various correlates. Here studies are reviewed that identify which individuals with ASD are at-risk for being targeted and outcomes associated with victimization.

Which individuals with ASD are targeted. One of the more consistent findings related to risk for

victimization is that those in general education setting are more likely to be targets of peer victimization than those in special educational settings (Bitsika and Sharpley 2014; Blake et al. 2012; Maiano et al. 2016; Zablotsky et al. 2013; Zeedyk et al. 2014). Thus, studies focused on negative peer experiences may want to put a special focus on individuals who are “mainstreamed” in general education.

A few studies have tried to understand how variability in social-communication skills in this group might be associated with peer victimization, with mixed findings (Cappadocia et al. 2012; Chou et al. 2019; Fink et al. 2018; Rowley et al. 2012; Sterzing et al. 2012; Storch et al. 2012). Some of these studies find social-communication skills to be positively associated, others negatively associated, and some studies find null effects. A few explanations have been provided for these discrepant findings: higher abilities may make it more likely that self-reporting adolescents with ASD are aware of these negative interactions, or it may be that certain settings (e.g., mainstreamed classrooms or unsupervised settings) can result in difficulties in social-communication skills becoming more or less of a liability for being a target of peer victimization. In other words, if individuals with better skills are more likely to have access to more inclusive environments, this may raise their risk of being targeted by similar-aged peers.

Outcomes associated with victimization. Externalizing problems, such as poor impulse/emotional control, ADHD, and aggression, are commonly found to both predict and result from peer victimization experiences in TD children and adolescents (Card and Little 2006). Similarly, externalizing problems (which commonly co-occur with ASD) are associated with peer victimization in individuals with ASD (Cappadocia et al. 2012; Chou et al. 2019; Hebron and Humphrey 2014; Rieffe et al. 2012). Recent studies suggest that special attention should be given to situations when classmates provoke an aggressive reaction from their ASD classmate by purposely directing behaviors that they know will make their ASD classmate overreact emotionally (i.e., “meltdown”; Zablotsky et al. 2013).

Among adolescents with ASD, peer victimization has also been found to be associated with internalizing problems such as symptoms of depression and anxiety (Adams et al. 2014; Cappadocia et al. 2012; Rosbrook and Whittingham 2010; Storch et al. 2012; Ung et al. 2016; Wright and Wachs 2019). This association is particularly significant given that lifetime estimates of internalizing disorders for those with ASD are as high as 52% for depression and 65% for anxiety disorders, compared to rates of 5% and 20% reported in TD adolescents (Costello et al. 2005a, b; Lever and Geurts 2016; Leyfer et al. 2006; van Steensel et al. 2011). These higher rates of internalizing problems might result, at least in part, from peer victimization experiences.

Finally, peer victimization has been shown to be associated with school outcomes in individuals with ASD. Adams et al. (2016) found, in two separate data sets, that both parent- and peer-reports of peer victimization were associated with feelings about school (i.e., safety, liking, belonging), behavioral problems at school, and academic performance problems. Similar to internalizing symptoms, associations between peer victimization and school experiences are particularly concerning given the overwhelming problems that adolescents with ASD have with these same school outcomes. Even among those with high cognitive abilities, relative to TD peers, individuals with ASD underperform across various measures of academic performance (Ashburner et al. 2010; Estes et al. 2011; Jones et al. 2009; Keen et al. 2015; Wei et al. 2013) are less likely to enjoy school, less likely to engage cooperatively and independently at school (Jahromi et al. 2013), and more likely to be removed from a public educational setting by way of either suspensions (Starr and Foy 2010) or being home schooled (Parsons and Lewis 2010). Similar to internalizing, these high rates of school problems might be a caused, in part by peer victimization experiences.

Future Directions

The first substantial wave of studies examining peer victimization in children and adolescents

with ASD has occurred over the past 7–10 years, and now the next wave of studies that build upon the finding of these early studies is beginning. The following are three suggested areas that should be considered for the next wave of studies.

Advanced methods. Most of the initial studies on peer victimization in ASD were cross-sectional survey studies that utilized single-item measures to test questions of the frequency of the occurrence of peer victimization. Studies utilizing more advanced methods have just started to appear, and this trend must continue in order to inform the development of interventions that target these negative peer experiences. The focus must move from testing frequencies of these experiences, to testing associations between predictors and outcomes so that specific processes of peer victimization can be identified for this group. While it is known that those with ASD are common targets of peer victimization, little is known about what this process looks like, who is targeted, why they are targeted, or the long-term ramifications of these experiences. Unfortunately, studies that test such associations are all cross-sectional. This means that we do not yet know if a construct under study is a predictor of and/or an outcome of peer victimization. This is particularly problematic because many constructs that have been shown to be important for understanding peer victimization are both predictors *and* outcomes in studies of TD populations. For instance, among TD children, internalizing problems are both predictors and outcomes of peer victimization (Reijntjes et al. 2010).

Additionally, the dearth of longitudinal studies has resulted in a lack of understanding of the developmental trajectories of peer victimization over the course of childhood and adolescence (e.g., when do rates peak? decrease?). Finally, the field must move away from using single item measures (e.g., is your child bullied) and ensure that the measures being utilized provide proper measurement validity. Not only should these measures have multiple items that tap into various forms of peer victimization that this group experiences, but these measures should be tested to ensure that they are psychometrically sound, especially for self-reports. Self-report measures of

peer victimization are very important for understanding such experiences and their outcomes (e.g., Adams et al. 2014). Particularly for those who spend a majority of their school in inclusive settings, there might not be adults present to witness such experiences and thus self-reports are crucial.

Predictors. For many groups at heightened risk for being targeted for peer victimization, it is a unique physical characteristic that makes them a target, such as being obese (Adams and Bukowski 2008; Adams and Cantin 2013). Those with ASD often do not, at first glance, appear physically different than their peers. Instead, it is the behaviors they exhibit that may cause them to seem different and, in turn, put them at risk for being targeted for peer victimization. However, there is tremendous phenotypic heterogeneity in the behaviors exhibited by adolescents with ASD; knowing what puts someone with ASD at increased risk for negative peer experiences requires more than simply knowing that he/she has ASD. For example, it is possible that any number of common ASD symptoms, including decreased social and emotional insight, restricted and repetitive behaviors, or communication difficulties, as well as common behaviors exhibited by this group (e.g., issues of hygiene, tattling, rigidity around rule breaking), might potentially place this group at increased risk for negative peer experiences. Future studies must test the various symptoms of ASD, as well as the various comorbid problems and behaviors, as possible predictors. Until these predictors are known it is difficult to create interventions to address this issue.

Gender. Gender differences are apparent in studies of TD adolescents across most aspects of social development (Rose and Rudolph 2006), as well as in peer victimization (e.g., rates of different types and content of victimization; Hong and Espelage 2012). Males and females often experience different forms of bullying, with boys experiencing more physical bullying (Carbone-Lopez et al. 2010), girls experiencing more relational forms (Murray-Close et al. 2007), and boys and girls experiencing similar levels of verbal bullying but with topics of verbal bullying differing by gender (Duncan 1999; Gruber and Fineran

2008). Because social experiences often vary as a function of gender, successful interventions targeting social experience *must* take gender into account. A few studies have examined gender differences in global peer victimization for those with ASD and found no gender differences. This is not surprising, as many studies in TD individuals also find no gender differences when examining only global indicators (as already stated, differences emerge when examining types of peer victimization) (Fink et al. 2018; Schrooten et al. 2018; Ung et al. 2016). There has been one study that tested gender differences for various types of peer victimization among those with ASD and found a marginal effect (e.g., $p = 0.10$) for relational aggression (Ung et al. 2016). It is important to note that most studies are underpowered to detect gender differences, as ASD samples tend to be small and occur in four to five males for every female (CDC morbidity and mortality report, 2018). Future research with larger samples is needed to understand peer victimization experiences of males vs females.

See Also

- Bullying
- Interpersonal Skills
- Peers, Exposure to
- Social Interventions
- Social Skill Interventions
- Stigmatization
- Victimization

References and Reading

- Adams, R. E., & Bukowski, W. M. (2008). Peer victimization as a predictor of depression and body mass index in obese and non-obese adolescents. *Journal of Child Psychology and Psychiatry*, 49(8), 858–866. <https://doi.org/10.1111/j.1469-7610.2008.01886.x>.
- Adams, R. E., & Cantin, S. (2013). Self-disclosure in friendships as the moderator of the association between peer victimization and depressive symptoms in overweight adolescents. *The Journal of Early Adolescence*, 33(3), 341–362. <https://doi.org/10.1177/0272431612441068>.
- Adams, R. E., Fredstrom, B. K., Duncan, A. W., Holleb, L. J., & Bishop, S. L. (2014). Using self- and parent-reports to test the association between peer victimization and internalizing symptoms in verbally fluent adolescents with ASD. *Journal of Autism and Developmental Disorders*, 44(4), 861–872. <https://doi.org/10.1007/s10803-013-1938-0>.
- Adams, R., Taylor, J., Duncan, A., & Bishop, S. (2016). Peer victimization and educational outcomes in mainstreamed adolescents with autism spectrum disorder (ASD). *Journal of Autism and Developmental Disorders*, 46(11), 3557–3566. <https://doi.org/10.1007/s10803-016-2893-3>.
- Ashburner, J., Ziviani, J., & Rodger, S. (2010). Surviving in the mainstream: Capacity of children with autism spectrum disorders to perform academically and regulate their emotions and behavior at school. *Research in Autism Spectrum Disorders*, 4(1), 18–27. <https://doi.org/10.1016/j.rasd.2009.07.002>.
- Ashburner, J., Saggars, B., Campbell, M. A., Dillon-Wallace, J. A., Hwang, Y., Carrington, S., & Bobir, N. (2019). How are students on the autism spectrum affected by bullying? Perspectives of students and parents. *Journal of Research in Special Educational Needs*, 19(1), 27–44. <https://doi.org/10.1111/1471-3802.12421>.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development*, 71(2), 447–456. <https://doi.org/10.1111/1467-8624.00156>.
- Bear, G., Mantz, L. S., Glutting, J. J., Yang, C., & Boyer, D. E. (2015). Differences in bullying victimization between students with and without disabilities. *School Psychology Review*, 44, 98–116. <https://doi.org/10.17105/spr44-1.98-116>.
- Bitsika, V., & Sharpley, C. F. (2014). Understanding, experiences, and reactions to bullying experiences in boys with an autism spectrum disorder. *Journal of Developmental and Physical Disabilities*, 26(6), 747–761. <https://doi.org/10.1007/s10882-014-9393-1>.
- Blake, J. J., Lund, E. M., Zhou, Q., Kwok, O. M., & Benz, M. R. (2012). National prevalence rates of bully victimization among students with disabilities in the United States. *School Psychology Quarterly*, 27(4), 210–222. <https://doi.org/10.1037/spq0000008>.
- Cappadocia, M. C., Weiss, J. A., & Pepler, D. (2012). Bullying experiences among children and youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(2), 266–277. <https://doi.org/10.1007/s10803-011-1241-x>.
- Carbone-Lopez, K., Esbensen, F., & Brick, B. T. (2010). Correlates and consequences of peer victimization: Gender differences in direct and indirect forms of bullying. *Youth Violence and Juvenile Justice*, 8(4), 332–350. <https://doi.org/10.1177/1541204010362954>.
- Card, N. A., & Little, T. D. (2006). Proactive and reactive aggression in childhood and adolescence: A meta-analysis of differential relations with psychosocial adjustment. *International Journal of Behavioral*

- Development*, 30(5), 466–480. <https://doi.org/10.1177/0165025406071904>.
- Chamberlain, B., Kasari, C., & Rotheram-Fuller, E. (2007). Involvement or isolation? The social networks of children with autism in regular classrooms. *Journal of Autism and Developmental Disorders*, 37(2), 230–242. <https://doi.org/10.1007/s10803-006-0164-4>.
- Chan, K. L., Lo, C. K. M., & Ip, P. (2018). Associating disabilities, school environments, and child victimization. *Child Abuse & Neglect*, 83, 21–30. <https://doi.org/10.1016/j.chiabu.2018.07.001>.
- Chou, W. J., Hsiao, R. C., Ni, H. C., Liang, S. H., Lin, C. F., Chan, H. L., . . . Yen, C. F. (2019). Self-reported and parent-reported school bullying in adolescents with high functioning autism spectrum disorder: The roles of autistic social impairment, attention-deficit/hyperactivity and oppositional defiant disorder symptoms. *International Journal of Environmental Research and Public Health*, 16(7), 1117. <https://doi.org/10.3390/ijerph16071117>.
- Costello, E. J., Egger, H., & Angold, A. (2005a). 10-Year research update review: The epidemiology of child and adolescent psychiatric disorders: I. Methods and public health burden. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44(10), 972–986. <https://doi.org/10.1097/01.chi.0000172552.41596.6f>.
- Costello, E. J., Egger, H. L., & Angold, A. (2005b). The developmental epidemiology of anxiety disorders: Phenomenology, prevalence, and comorbidity. *Child and Adolescent Psychiatric Clinics of North America*, 14(4), 631–648, vii. <https://doi.org/10.1016/j.chc.2005.06.003>.
- Duncan, N. (1999). *Sexual bullying: Gender conflict and pupil culture in secondary schools*. New York: Routledge.
- Estes, A., Rivera, V., Bryan, M., Cali, P., & Dawson, G. (2011). Discrepancies between academic achievement and intellectual ability in higher-functioning school-aged children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41(8), 1044–1052. <https://doi.org/10.1007/s10803-010-1127-3>.
- Fink, E., Olthof, T., Goossens, F., van der Meijden, S., & Begeer, S. (2018). Bullying-related behaviour in adolescents with autism: Links with autism severity and emotional and behavioural problems. *Autism*, 22(6), 684–692. <https://doi.org/10.1177/1362361316686760>.
- Gruber, J. E., & Fineran, S. (2008). Comparing the impact of bullying and sexual harassment victimization on the mental and physical health of adolescents. *Sex Roles*, 59(1–2), 1–13. <https://doi.org/10.1007/s11199-008-9431-5>.
- Halladay, A. K., Bishop, S., Constantino, J. N., Daniels, A. M., Koenig, K., Palmer, K., Messinger, D., Pelphrey, K., Sanders, S. J., Singer, A. T., Taylor, J. L., & Szatmari, P. (2015). Sex and gender differences in autism spectrum disorder: Summarizing evidence gaps and identifying emerging areas of priority. *Molecular Autism*, 6, 36. <https://doi.org/10.1186/s13229-015-0019-y>.
- Hebron, J., & Humphrey, N. (2014). Exposure to bullying among students with autism spectrum conditions: A multi-informant analysis of risk and protective factors. *Autism*, 18(6), 618–630. <https://doi.org/10.1177/1362361313495965>.
- Hong, J. S., & Espelage, D. L. (2012). A review of research on bullying and peer victimization in school: An ecological system analysis. *Aggression and Violent Behavior*, 17(4), 311–322. <https://doi.org/10.1016/j.avb.2012.03.003>.
- Humphrey, N., & Symes, W. (2010). Perceptions of social support and experience of bullying among pupils with autistic spectrum disorders in mainstream secondary schools. *European Journal of Special Needs Education*, 25(1), 77–91. <https://doi.org/10.1080/08856250903450855>.
- Humphrey, N., & Symes, W. (2011). Peer interaction patterns among adolescents with autistic spectrum disorders (ASDs) in mainstream school settings. *Autism*, 15(4), 397–419. <https://doi.org/10.1177/1362361310387804>.
- Jahromi, L. B., Bryce, C. I., & Swanson, J. (2013). The importance of self-regulation for the school and peer engagement of children with high-functioning autism. *Research in Autism Spectrum Disorders*, 7(2), 235–246. <https://doi.org/10.1016/j.rasd.2012.08.012>.
- Jones, A. P., & Frederickson, N. (2010). Multi-informant predictors of social inclusion for students with autism spectrum disorders attending mainstream school. *Journal of Autism and Developmental Disorders*, 40(9), 1094–1103. <https://doi.org/10.1007/s10803-010-0957-3>.
- Jones, C. R., Happe, F., Golden, H., Marsden, A. J., Tregay, J., Simonoff, E., Pickles, A., Baird, G., & Charman, T. (2009). Reading and arithmetic in adolescents with autism spectrum disorders: Peaks and dips in attainment. *Neuropsychology*, 23(6), 718–728. <https://doi.org/10.1037/a0016360>.
- Keen, D., Webster, A., & Ridley, G. (2015). How well are children with autism spectrum disorder doing academically at school? An overview of the literature. *Autism*, 20(3), 276–294. <https://doi.org/10.1177/1362361315580962>.
- Kloosterman, P. H., Kelley, E. A., Craig, W. M., Parker, J. D. A., & Javier, C. (2013). Types and experiences of bullying in adolescents with an autism spectrum disorder. *Research in Autism Spectrum Disorders*, 7(7), 824–832. <https://doi.org/10.1016/j.rasd.2013.02.013>.
- Kowalski, R. M., & Fedina, C. (2011). Cyber bullying in ADHD and Asperger syndrome populations. *Research in Autism Spectrum Disorders*, 5(3), 1201–1208. <https://doi.org/10.1016/j.rasd.2011.01.007>.
- Lever, A. G., & Geurts, H. M. (2016). Psychiatric co-occurring symptoms and disorders in young, middle-aged, and older adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(6), 1916–1930. <https://doi.org/10.1007/s10803-016-2722-8>.

- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., Tager-Flusberg, H., & Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36(7), 849–861. <https://doi.org/10.1007/s10803-006-0123-0>.
- Little, L. (2002). Middle-class mothers' perceptions of peer and sibling victimization among children with Asperger's syndrome and nonverbal learning disorders. *Issues in Comprehensive Pediatric Nursing*, 25(1), 43–57. <https://doi.org/10.1080/014608602753504847>.
- Locke, J., Ishijima, E. H., Kasari, C., & London, N. (2010). Loneliness, friendship quality and the social networks of adolescents with high-functioning autism in an inclusive school setting. *Journal of Research in Special Educational Needs*, 10(2), 74–81. <https://doi.org/10.1111/j.1471-3802.2010.01148.x>.
- Lyons, J., Cappadocia, M. C., & Weiss, J. A. (2011). Brief report: Social characteristics of students with autism spectrum disorders across classroom settings. *Journal on Developmental Disabilities*, 17(1), 77–82.
- Maiano, C., Normand, C. L., Salvas, M. C., Moullec, G., & Aime, A. (2016). Prevalence of school bullying among youth with autism spectrum disorders: A systematic review and meta-analysis. *Autism Research*, 9(6), 601–615. <https://doi.org/10.1002/aur.1568>.
- Murray-Close, D., Ostrov, J. M., & Crick, N. R. (2007). A short-term longitudinal study of growth of relational aggression during middle childhood: Associations with gender, friendship intimacy, and internalizing problems. *Development and Psychopathology*, 19(01), 187–203. <https://doi.org/10.1017/S0954579407070101>.
- Nowell, K. P., Brewton, C. M., & Goin-Kochel, R. P. (2014). A multi-rater study on being teased among children/adolescents with autism spectrum disorder (ASD) and their typically developing siblings: Associations with ASD symptoms. *Focus on Autism and Other Developmental Disabilities*. <https://doi.org/10.1177/1088357614522292>.
- Parsons, S., & Lewis, A. (2010). The home-education of children with special needs or disabilities in the UK: Views of parents from an online survey. *International Journal of Inclusive Education*, 14(1), 67–86. <https://doi.org/10.1080/13603110802504135>.
- Petrina, N., Carter, M., & Stephenson, J. (2014). The nature of friendship in children with autism spectrum disorders: A systematic review. *Research in Autism Spectrum Disorders*, 8(2), 111–126. <https://doi.org/10.1016/j.rasd.2013.10.016>.
- Pfeffer, R. D. (2016). Childhood victimization in a national sample of youth with autism spectrum disorders. *Journal of Policy and Practice in Intellectual Disabilities*, 13(4), 311–319. <https://doi.org/10.1111/jppi.12203>.
- Reijntjes, A., Kamphuis, J. H., Prinzie, P., & Telch, M. J. (2010). Peer victimization and internalizing problems in children: A meta-analysis of longitudinal studies. *Child Abuse and Neglect*, 34(4), 244–252. <https://doi.org/10.1016/j.chiabu.2009.07.009>.
- Rieffe, C., Camodeca, M., Pouw, L. B. C., Lange, A. M. C., & Stockmann, L. (2012). Don't anger me! Bullying, victimization, and emotion dysregulation in young adolescents with ASD. *European Journal of Developmental Psychology*, 9(3), 351–370. <https://doi.org/10.1080/17405629.2012.680302>.
- Rosbrook, A., & Whittingham, K. (2010). Autistic traits in the general population: What mediates the link with depressive and anxious symptomatology? *Research in Autism Spectrum Disorders*, 4(3), 415–424. <https://doi.org/10.1016/j.rasd.2009.10.012>.
- Rose, A. J., & Rudolph, K. D. (2006). A review of sex differences in peer relationship processes: Potential trade-offs for the emotional and behavioral development of girls and boys. *Psychological Bulletin*, 132(1), 98–131. <https://doi.org/10.1037/0033-2909.132.1.98>.
- Rose, C. A., Simpson, C. G., & Moss, A. (2015). The bullying dynamic: Prevalence of involvement among a large-scale sample of middle and high school youth with and without disabilities. *Psychology in the Schools*, 52(5), 515–531. <https://doi.org/10.1002/pits.21840>.
- Rowley, E., Chandler, S., Baird, G., Simonoff, E., Pickles, A., Loucas, T., & Charman, T. (2012). The experience of friendship, victimization and bullying in children with an autism spectrum disorder: Associations with child characteristics and school placement. *Research in Autism Spectrum Disorders*, 6(3), 1126–1134. <https://doi.org/10.1016/j.rasd.2012.03.004>.
- Schrooten, I., Scholte, R. H. J., Cillessen, A. H. N., & Hymel, S. (2018). Participant roles in bullying among Dutch adolescents with autism spectrum disorders. *Journal of Clinical Child and Adolescent Psychology*, 47(6), 874–887. <https://doi.org/10.1080/15374416.2016.1138411>.
- Starr, E. M., & Foy, J. B. (2010). In parents' voices: The education of children with autism spectrum disorders. *Remedial and Special Education*, 33(4), 207–216. <https://doi.org/10.1177/0741932510383161>.
- Sterzing, P. R., Shattuck, P. T., Narendorf, S. C., Wagner, M., & Cooper, B. P. (2012). Bullying involvement and autism spectrum disorders: Prevalence and correlates of bullying involvement among adolescents with an autism spectrum disorder. *Archives of Pediatrics & Adolescent Medicine*, 166(11), 1058–1064. Retrieved from www.archpediatrics.com.
- Storch, E. A., Larson, M. J., Ehrenreich-May, J., Arnold, E. B., Jones, A. M., Renno, P., . . . Wood, J. J. (2012). Peer victimization in youth with autism spectrum disorders and co-occurring anxiety: Relations with psychopathology and loneliness. *Journal of Developmental and Physical Disabilities*, 24(6), 575–590. <https://doi.org/10.1007/s10882-012-9290-4>.
- Twyman, K. A., Saylor, C. F., Saia, D., Macias, M. M., Taylor, L. A., & Spratt, E. (2010). Bullying and ostracism experiences in children with special health care needs. *Journal of Developmental and Behavioral Pediatrics*, 31(1), 1–8. <https://doi.org/10.1097/DBP.0b013e3181c828c8>.

- Ung, D., McBride, N., Collier, A., Selles, R., Small, B., Phares, V., & Storch, E. (2016). The relationship between peer victimization and the psychological characteristics of youth with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 32, 70–79. <https://doi.org/10.1016/j.rasd.2016.09.002>.
- van Steensel, F. J., Bogels, S. M., & Perrin, S. (2011). Anxiety disorders in children and adolescents with autistic spectrum disorders: A meta-analysis. *Clinical Child and Family Psychology Review*, 14(3), 302–317. <https://doi.org/10.1007/s10567-011-0097-0>.
- Wainscot, J. J., Naylor, P., Sutcliffe, P., Tantam, D., & Williams, J. V. (2008). Relationships with peers and use of the school environment of mainstream secondary school pupils with Asperger syndrome (high-functioning autism): A case-control study. *International Journal of Psychology and Psychological Therapy*, 8(1), 25–38.
- Wei, X., Lenz, K. B., & Blackorby, J. (2013). Math growth trajectories of students with disabilities: Disability category, gender, racial, and socioeconomic status differences from ages 7 to 17. *Remedial and Special Education*, 34(3), 154–165. <https://doi.org/10.1177/0741932512448253>.
- Wright, M. F., & Wachs, S. (2019). Does peer rejection moderate the associations among cyberbullying victimization, depression, and anxiety among adolescents with autism spectrum disorder? *Children*, 6(3), 41. <https://doi.org/10.3390/children6030041>.
- Zablotsky, B., Bradshaw, C. P., Anderson, C., & Law, P. A. (2013). The association between bullying and the psychological functioning of children with autism spectrum disorders. *Journal of Developmental and Behavioral Pediatrics*, 34(1), 1–8. <https://doi.org/10.1097/dbp.0b013e31827a7c3a>.
- Zeedyk, S. M., Rodriguez, G., Tipton, L. A., Baker, B. L., & Blacher, J. (2014). Bullying of youth with autism spectrum disorder, intellectual disability, or typical development: Victim and parent perspectives. *Research in Autism Spectrum Disorders*, 8(9), 1173–1183. <https://doi.org/10.1016/j.rasd.2014.06.001>.

Peer-Mediated Intervention

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Definition

Peer-mediated interventions (PMIs) involve training peers without disabilities to be responsive and

interactive social partners to children with autism spectrum disorders (ASD). Both peers and students with ASD are taught to initiate and respond to each other within engineered (i.e., instruction in small groups) and typical school social contexts (e.g., centers, game play, recess, and lunch) across the school day. For adolescents with ASD, programs involving peers without disabilities are generally referred to as peer networks. A group of 5–6 peers is recruited and meets regularly to plan social activities, seek solutions to social problems, and create daily opportunities for students with ASD to have positive social experiences both in and outside of the classroom. The primary focus of PMIs, regardless of age, is to increase social reciprocity, positive interactions, and possibly the development of friendships.

Historical Background

The concept of training peers without disabilities to be responsive social partners to young children with autism began in the 1970s. This landmark research was particularly important given the core deficits in social communication characteristic of autism and failure to develop peer relationships (American Speech-Language-Hearing Association [ASHA] 2006). In the 1980s, these initial positive findings were replicated, and different PMI approaches were explored with preschoolers with much success. Some of these approaches included teaching peers to (1) use responsive language facilitation strategies (e.g., repeat and expand on language used by focus child with autism), (2) use social initiation strategies (e.g., establish joint attention, initiate play routines, comment on one's own play), and (3) learn language scripts that matched dramatic play themes (e.g., hamburger stand, birthday party) (Goldstein et al. 2007). In the 1990s, PMI approaches were extended to include elementary school-age students with ASD and, to a lesser degree, middle school and high school students. The focus over the last decade has been to create comprehensive school-based PMIs that integrate peer training with evidence-based direct instruction to achieve more widespread and robust social outcomes in inclusive settings.

Two instructional approaches more recently incorporated within PMIs are video modeling and the use of written-text or graphic-cueing systems (Thiemann and Goldstein 2001; Thiemann-Bourque and Goldstein 2004). For example, social group interactions can be videotaped then played back for self-evaluation and feedback. Words or short phrases (written-text cues) are used to prompt what to say in a specific social context, along with picture symbols or photographs of children interacting. These strategies capitalize on visual learning styles and strengths of children with autism.

Rationale or Underlying Theory

Friendships build on social interactions. There must be opportunities for interactions and the outcomes of those interactions must be positive for friendships to begin to develop. Social interdependence theory considers how the rewards or benefits of a social interaction outweigh the costs. It is more likely that children will engage in social interactions when their social exchanges are rewarding and fun than if interactions require effort to maintain. This applies to both children with autism and those without developmental disabilities. Adults in the children's environment are the key to planning effective social programming to assure maximum benefits for all involved.

In inclusive settings, exposure to peers alone is not enough to impact the social and communication deficits characteristic of children with ASD. When an adult sets up the social environment to meet individual child needs, then models, prompts, and reinforces social communication and social responsiveness – many benefits can occur. For students with ASD, involvement in PMIs usually leads to increased reciprocal communication with peers, greater friendship ratings among classmates, and a decrease in behaviors that can lead to awkward or uncomfortable interactions. Group contexts provide naturally occurring social opportunities with socially sophisticated peers who can model important communication skills. Consequently, children with ASD will have a greater chance of generalizing communication

skills across social settings and partners. Benefits to peers include learning social and communication skills to engage in positive interactions with children with disabilities and other classmates and enjoyment in helping others. Further, involving available peers as social change agents can decrease time demands on teachers or other staff and lead to spillover effects to untrained peers. Without training, peers may reinforce children's inappropriate social behaviors and ignore desirable behaviors.

Several possible reasons may explain the benefits of using written-text, graphic, or other picture cues to teach social communication within peer groups. First, it is a widely held belief that children with ASD have a superior ability to recall visually presented information (Grandin 1995). Second, written-text cues are basically “scripts” that provide children with constant access to a relevant and appropriate “social outline” to follow and language to use within a social context. Peers are taught to remind focus children to use this cue to initiate, respond, and stay engaged in the interaction. Third, having picture and text cues available throughout an activity provides opportunities for repeated practice of the same target skill(s), and when peers express the same utterances, they provide many models of the skill in a short time period. For children who are verbal yet not intelligible, communication attempts will be better understood given that everyone in the group knows the target communication skill and script. Finally, peers provide feedback on the timing and social appropriateness of the child's behavior in different social contexts. This feedback and reinforcement may lead to greater independent use of targeted communication skills.

Goals and Objectives

The primary goals and objectives of PMIs are:

1. To include peers as intervention agents and train them to use strategies that lead to successful social exchanges with children with ASD
2. To provide regular and frequent opportunities for children with ASD and peers without disabilities

to practice age-appropriate social communication skills in typical school social settings

3. To provide direct instruction that increases children’s repertoire of verbal and nonverbal social communication behaviors to (for example):
 - Engage in joint play routines
 - Respond to peer’s bids for joint play
 - Take turns and share
 - Gain attention/greet others
 - Comment or describe actions and objects
 - Request actions, objects, or information
 - Keep talking to maintain topics
 - Be flexible about topics
 - Use social niceties (e.g., compliments, cheers, and sharing affection)
4. To improve reciprocal child-peer social interactions and enjoyment across varied social activities and environments
5. To reduce the divergent social path and social competency gap that occurs between children with and without ASD as they advance through higher grades
6. To foster friendships and relationships between individuals with and without ASD

Children with better social skills are more likely to make friends, and having friends further supports the acquisition of social skills. If appropriate social skills provide a foundation for relationship and friendship development, what social goals are most important? Table 1 lists possible social communication targets that can lead to improved initiations and reciprocal interactions for young children with ASD. The difficulty in deciding which skills to teach is complicated by two factors. Children need to learn not simply a repertoire of skills but when to use which social skills, when to vary them based on the situation, with what frequency of use, and such. This is further complicated by the need to adjust to the ever-changing expectations of one’s peer group, family members, teachers, and community members as the child grows older. Thus, the interventionist needs to be cognizant of shifting social demands and plan ahead to equip children with social skills that will be needed in future social encounters.

Peer-Mediated Intervention, Table 1 Potential social communication goals and example written-text cues

Preschool	School age
Descriptive comments (e.g., “I have two”; “Mine is blue”)	Descriptive comments (e.g., “You have a match”; “I got one!”)
Provide assistance/offer help (e.g., “You want help?”)	Requests for actions or objects (e.g., “Roll it” or “Can I have the dice?”)
Share/give/accept toys (e.g., “Here you go” + giving toy)	Requests to gain information (e.g., “What do I do?”)
Establish joint attention (e.g., tap on shoulder, move closer)	Secures for attention (before initiations) (e.g., “(name), _____” or tap on shoulder)
Provide affection and praise (e.g., high five, pat on back)	Social niceties (e.g., “Way to go!” or “Maybe next time”)
Play organizers (e.g., “Let’s play _____”; “You be the cook”)	Make suggestions (e.g., “I have an idea, let’s do rock-paper-scissors”)

Treatment Participants

PMI approaches have been successfully implemented for individuals with autism spectrum disorders (ASD), including autism, Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS), and Asperger’s syndrome. Participants with ASD are most often between the ages of 3 and 13 years and attend preschool or elementary school with typically developing peers. However, with modifications, peer network programs can be successfully implemented for middle and high school students as well (Thiemann-Bourque 2010). Participants with ASD demonstrate a wide range of cognitive, social, and communication impairments. Peer implementers are generally the same age as their classmate with ASD and are recruited based on select criteria (see section “[Peer Participation](#)” below):

1. Age-appropriate social skills
2. Has similar interests to student with ASD
3. Well-liked by the majority of classmates (i.e., high-status peers)
4. Consistent school attendance
5. Similar academic groupings or classes with student with ASD

6. Not overtly shy or quiet
7. Willingness to participate

Treatment Procedures

Selecting Instructional Strategies

Successful implementation of PMIs can be accomplished by attending to the intervention components listed in Table 2. These are established guidelines for planning, implementing, and monitoring PMIs, with the understanding that there is an ongoing need to examine specific social behaviors to teach and in what contexts to optimize the social outcomes. The following guiding principles can be used to select instructional strategies (Goldstein and Kaczmarek 1992):

1. Strategies that teach behaviors observed in high-quality interactions between children in general (e.g., engagement, responsiveness, and smiling or other expressions of positive affect)
2. Strategies that have a higher chance of eliciting responsive social behaviors (e.g., mutual attention to an activity, descriptive comments about current activity, and acknowledging a speaker’s communication)
3. Strategies that generate reciprocal interactions as reflected in more balanced turn-taking (e.g., sharing objects in play, taking turns in games, and being verbal in conversations)
4. Strategies that optimize the social environment to enhance social interactions between children with and without ASD (e.g., attention to group size, use of preferred activities and activity choices, and adult guidance)

Peer-Mediated Intervention, Table 2 Components of comprehensive peer-mediated school social programs

Component	Description and examples
Supportive social environment	Visual schedule of group events, choice of two preferred and motivating activities, outline expectations and social roles, games with simple rules, 1–2 peers involved at one time
Regular social opportunities	Structured: 3–4 times/week for minimum of 15–20 min Less structured: 5–10 min across daily school routines
Naturalistic settings	Free play, centers, thematic play (e.g., grocery store, restaurants, hairdresser), phonics or math games, snack, recess, lunch, social activities before or after school
Age-appropriate activities	Board games, art projects, science experiments; incorporate interests and familiar themes so motivating and fun for all
Visuals and picture cues	Peer “buddy” manual of targeted social skills; conversation topic starter cards; real photographs of the children; placemats at snack or lunch with topic starters Velcroed on silly pictures, favorite foods, cartoon characters, etc.
Written-text cues	Write out 2–3-word simple phrases on skill sheets or in topic bubbles that match the game/ social context; represent social skills with pictures or line drawings; peers use same cues as focus children; place in 5 × 7 or 8 × 10 clear photo frames
Peer training	Recruit 4–6 peers; well-liked and same age; teach 3–5 facilitative or responsive social skills; peers cue focus child to talk/interact using written-text and picture cues; peers can be taught to monitor skill use by self-evaluation – mark boxes on monitoring sheets; use cooperative progress charts for all
Adult mediation	Adult introduces and defines social communication skill; models examples using selected activity; role-plays with peers then focus child; prompts through peer first once per minute, then if unsuccessful, assists focus child; reinforces use of skill often at first, then fades, prompts, and reinforces
Videotape self- or peer modeling	Interactions are videotaped, then played back to discuss models of social skills, use of target skills, etc.
Plan for generalization	Train multiple peers; embed practice in natural inclusive settings; all staff aware of target skills; prompt and reinforce across the day; provide choices for activities; class-wide minisocial lessons, text cues, and visuals available across settings; coordinate home and community social activities

Peer Participation

Teachers recommend a group of 4–6 peers to participate in PMIs. To give ample time for social interactions and friendships to develop, the same groups of peers are involved in a PMI for the entire school year. After students begin to show increased interactions and engagement outside of PMI sessions, additional peers can be recruited. Parent consent may or may not be necessary based on classroom and school social curriculums.

Once a group of 4–6 peers have been recruited, an adult meets with them to explain the purpose of the groups, to review what it means to be a “buddy,” to find out preferred activities and popular games, and to talk about the social group schedule. This can be accomplished within one to two 30-min group sessions. Two peers are paired up with the focus student with ASD in small groups, and these pairings rotate across the week so each peer participates once per week. For younger children or children with more severe social needs, one peer can be paired up in a dyad with the focus child to decrease social demands and to increase opportunities for social skill practice.

Scheduling Social Groups

For direct teaching of social communication skills, groups meet 3–4 times per week for 20–30 min. A combination of structured practice and more naturalistic social opportunities is recommended. An adult sets up the social environment for success by including visual schedules, behavioral and reinforcement systems for increasing engagement, and preferred activities and games with simple rules. Opportunities to practice target social communication skills are embedded within less structured social times, when the context matches the skill (e.g., greeting and saying goodbye to peers, asking for items from peer at snack, asking peer for help in small group work). Multiple social opportunities exist within preschool centers, circle time, meal times, and dramatic play. As children advance through the grades and as the emphasis on academics increases, there is a need to become creative in providing social opportunities. Groups could

meet before or after school as part of school enrichment programs (e.g., breakfast club, games galore, drama club), at lunch (e.g., “lunch bunch”), at recess, or during the day when talking is allowed (small group work, art projects, etc.). The main idea is to provide numerous opportunities across the day to practice social skills taught in the small group format. This will greatly impact rate of learning and generalization of skill use.

Training Peers

Methods vary for training peers to be responsive conversational partners and to initiate and maintain interactions with children with ASD. In general, these methods are focused on teaching peers to model and prompt different social and language skills and provide corrective feedback and reinforcement to children with ASD. Peer training may take the form of meeting with peer buddies separately for group orientation, buddy training, and teaching specific facilitative social skills before joining a group with a child with autism. Alternatively, both peers and the child with autism can begin the groups together and be taught the same skills from the start of intervention. With this latter option, it is important for the adult to attend to the amount and level of language used when explaining the group goals, defining skills, etc. The child with autism may need a preferred reinforcer or behavior system to sit at the table with the group during the “teaching” time. Currently, the recommended method for training peers is to teach all children together and modify sessions accordingly to meet the needs of all children.

Two published curriculums for teaching preschoolers buddy skills include “Stay-Play-Talk” (English et al. 2005) and “Play Time/Social Time: Organizing your classroom to build interaction skills” (Odom and McConnell 1997). Examples of adult verbal prompts and social skills from Play Time/Social Time curriculum are listed in Table 3.

Table 4 lists examples of facilitative social communication skills and behavior substeps successfully taught to peers to be responsive conversational partners (Thiemann et al. 2012). Peers

Peer-Mediated Intervention, Table 3 Example prompts used in peer training from “play time/social time”

1. Sharing:
“___ wants to trade pictures with you. Please trade”
“___, give ___ a letter to put with the picture”
“___, ask ___ if she wants the picture”
2. Requesting to share:
“___, ask ___ to give you a letter. Point to the one you want”
“___, ask ___ if your puppet can share the word cards”
3. Play organizing:
“___, tell ___ to put his cat in the barn”
“___, tell ___ to put the word cat by the animal cat”
4. Agreeing:
“___, say, ‘Yes, my rat is thirsty’”
“___, make your cat follow ___’s cat”
“___, say, ‘Yes, I’ll help you say the words’”
5. Assisting and requesting assistance:
“___, help ___ sort the picture cards”
“___, ask ___ to help you match the letters to the pictures”
6. Persistence:
“___, show me ‘keep on trying’ with the word cards”
“___, show me ‘try another way’ with the picture matching”
“___, show ___ matching the cat to the word card, then ask again”

can also be taught to model and role-play target skills, use time delay prompts, and respond quickly and appropriately to their classmate with ASD.

Selecting Social Communication Goals

Young students with autism have a significantly restricted range or repertoire of social communication skills (e.g., language used to request objects or social routines, protest, greet, request information, and comment on personal or peer interests), and most demonstrate limited initiation skills. Evidence documenting characteristic social-communicative profiles of young students with PDD has distinguished them from children with other developmental disabilities and peers without disabilities (Wetherby and Prutting 1984). Typically developing preschool children exhibit 3–5 social initiations per minute during free play with peers, and this may vary up to 12 initiations. In comparison,

Peer-Mediated Intervention, Table 4 Examples of social communication skills to teach peers

<i>Skill #1: keep talking</i>
1. Listen to what a friend is saying
2. Show you are listening by saying, “sure” or nodding your head “yes”
3. Ask a question to find out more
<i>Skill #2: look, wait, and listen</i>
1. Look at friend who is talking
2. Show interest by sitting quietly
3. Hold up hands to show friend you are waiting
<i>Skill #3: answer questions</i>
1. Listen to friend’s question
2. Answer friend’s question
3. Tell friend if you do not understand
<i>Skill #4: start talking</i>
1. Choose a friend to talk to, say their name
2. Talk about what you see a friend doing
3. Tell a friend what you are doing
<i>Skill #5: say something nice</i>
1. Decide what to say something nice about
What your friend is making or doing
If a friend is winning or needs encouragement
What a friend is wearing
2. Give compliments in an honest, friendly way
3. Choose a good time to say something nice

students with autism primarily will initiate to adults and rarely to peers. Given the considerable limitations in social communication skills, and language spontaneity directed to peers, selecting and prioritizing communication goals is a challenging yet important first step.

Table 1 describes specific social communication skills and example text cues/scripts that have been successfully taught to preschool and school-age children within PMIs. One important consideration in selecting appropriate communication objectives is to choose behaviors that are effective in obtaining a positive response from peers or that enhance children’s responsiveness to their peers. For example, peers are more likely to respond to initiations of children with autism when their initiation attempt is combined with an attention-getting device (e.g., child looks at peer or says their name to get attention). Other strong predictors of successful communicative interactions are responding to what a peer just said and talking

about topics related to peer interests. Furthermore, children with autism may be more responsive to peer's verbal and nonverbal *requests* compared to other types of communication acts (English et al. 1997). Additional considerations in selecting appropriate target communication skills include (1) the child's language abilities in different social situations (e.g., performance vs. specific skill deficits), (2) behaviors that may impede positive peer interactions (e.g., physical aggression, being "bossy," talking too close or too quiet, or difficulty losing), (3) current IEP goals, and (4) family and teacher perceptions of social priorities.

Adult-Mediated Instruction

Each 30-min treatment session typically consists of 10 min of direct instruction, 15-min engagement in a social activity, and 5 min of adult feedback and reinforcement. Steps for direct teaching of social communication goals generally include:

1. Adult writes out group tasks on a visual agenda and reviews session goals.
2. Adult defines target social skill and substeps of skill (e.g., "Today we are going to learn how to say nice things to our friends. This means we can cheer for them or give compliments.").
3. Adult reads written-text cues that match target skill (inserted in standing clear 5 × 7 photo frame) and explains symbols or pictures that represent the communication skill (e.g., "Look, these children are cheering for their friend who won the game.").
4. Adult models target skill by reading the text cues, using parts/pieces of the upcoming activity or game.
5. Peers read cues and model/role-play target skill with each other.
6. Peers model/role-play the target skill with the child with ASD.
7. Adult teaches peer to prompt child to read/say the text cues of target skill.
8. Adult explains rules and expectations of social activity and shows children the reinforcement card that will be marked when they use the target skill. Visual cues of target skill are left on the table in child's direct line of view.
9. Adult sits back from the group and monitors child-peer communicative interactions during a 15-min activity. If no interactions occur, adult prompts peer once every 30 s to 1 min to prompt child with ASD to use the text cue card of the target skill. If peer is unsuccessful in gaining an appropriate response from the focus child, the adult intervenes and prompts accordingly.
10. Adult provides specific feedback on rate of skill use, shows performance of skill based on reinforcement card, and gives reinforcer as appropriate.

Efficacy Information

PMIs have long been recommended best practice for preschool children with autism based on intervention studies that span the last 40 years (McConnell 2002). Over the past 10–15 years, a growing number of studies have demonstrated positive outcomes for school-age children with ASD (Chan et al. 2009; National Standards Project 2009). Across these age groups, peer participation in social interventions should be considered recommended best practice for all individuals with autism. More evidence supporting implementation of peer training in natural school environments for older students is needed (Reichow and Volkmar 2010). Studies that are available for older students show that children with ASD and their peers can learn to communicate using functional or contextually appropriate language during age-appropriate games and activities, recess, lunch, and centers, for example. Communication improvements relate to greetings and gaining attention (e.g., saying hello, inviting others to play, saying child's name), commenting (e.g., "I have a match."; "You won."), requesting objects or actions (e.g., "Can I have the dice?"; "Roll the dice"), requesting information (e.g., "Who's winning?"; "Is it my turn?"), maintaining topics, and giving compliments (e.g., "Way to go!"; "Maybe next time") (Goldstein et al. 2007; Thiemann-Bourque 2010; Thiemann and Goldstein 2001; Thiemann-Bourque and Goldstein 2004; Thiemann and

Kamps 2008). Effective strategies used to teach communication skills within PMIs include video modeling, written-text cues, graphics, and other visual cues. Video modeling and other symbolic representations of social skills may be more effective when combined with other teaching techniques, as part of more comprehensive social programs.

Literature on social interventions for this population has emphasized the need for comprehensive programming that integrates or “layers” effective techniques in planning programs powerful enough to affect generalized and durable social outcomes (National Research Council 2001; Schwartz 2000). These model intervention “packages” often combine social-pragmatic and developmental approaches (e.g., targeting functional social-communicative skills in natural contexts) (Prizant and Wetherby 1998) with principles of contemporary behavioral interventions (e.g., multiple treatment components, systematic instruction of functional behaviors, access to preferred activities, and manipulation of ecological variables) (Horner et al. 1990). Schwartz discussed a promising intervention model that focuses on adult mediation, child’s skill repertoire, the social environment, and peer skills, supports, and expectations to guide children’s social relationships in natural settings. Recent PMI studies incorporating similar components of this model have reported the following benefits to students with ASD: (a) increased social initiations, (b) improved functional expressive language, (c) decreased unintelligible utterances, (d) use of new skills across novel and varied settings and social partners, (e) fewer inappropriate social utterances and behaviors, (f) improved friendship ratings, and (g) enhanced overall quality of interactions.

Evidence supporting PMIs is based primarily on multiple-baseline and alternating treatment experimental designs. There are over 50 single-subject experimental design studies, most of which contribute to evidence of robust effects of social skill interventions for young children with autism. Much has been learned from this literature. The focus is now shifting toward larger randomized control studies that compare PMIs with “business as usual” or traditional

interventions. Furthermore, there are currently a number of therapy resources that promote the use of pictures, symbols with text cues, and other illustrations of social interactions (e.g., Baker 2003; Kelly 1996); additional studies are necessary to document the effectiveness of these approaches on social interactions. These studies will be important to increase understanding of what components of comprehensive PMIs are critical to impact more lasting social changes and how to ensure feasibility of implementation for school staff.

Outcome Measurement

Reports of social outcomes have historically focused on measuring changes in the duration of an interaction, proximity to peers, and changes in rates of social initiations. There has been a growing emphasis on the need to measure multiple social outcomes that include social communication competence, social acceptance, and friendship development. Outcome measures have advanced to include verbal social communication (e.g., requests, comments, offers to share), nonverbal communication (e.g., facial expressions, body language), prelinguistic communication (e.g., gestures, eye gaze, joint attention), and conversational skills (e.g., topic maintenance). Measuring skill used within contexts that involve new (untrained) peers, novel activities, and games and that took place in nontreatment settings would indicate generalization of treatment gains. If generalization of skills is not observed, students need continued adult prompts and peer supports embedded within these novel, naturalistic settings.

A true measure of social success is the ability of individuals with and without ASD to engage in positive and successful interactions leading toward development of friendships. Haring and Breen (1992) adeptly provided the groundwork to measure this outcome by asking questions about friendship supports and relationships for junior high students (with autism and DD) following participation in peer networks. Measures included peer reports of interactions with the focus students based on invitations to

extracurricular activities and interactions in the hallway, locker areas, cafeteria, and school field. Children perceived as being more socially competent are more likely to be accepted by peers and to be chosen as friends. These outcomes can be measured by asking peers to complete friendship or acceptance questionnaires at the beginning and end of the school year. For younger children, one way to assess acceptance is to ask them to place a photo of a “buddy” in their class that they would like to take on a train ride at the zoo (with connected paper train cars). For older students, rating scales with smiley faces can be used to assess acceptance with questions such as “Would you like to sit beside _____ at lunch time?” or “Do you play with _____ on the playground?” or “Would you like to be partners with _____ to do an art project?” etc. Knowledge of how student’s social engagement and acceptance are developing outside of the treatment setting is critical to guide intervention efforts to enhance long-term friendships.

Qualifications of Treatment Providers

The ASHA (2006) published guidelines for speech-language pathologists (SLPs) working with individuals with ASD. These guidelines stress the critical role of SLPs in supporting the child with ASD and the communication partner (e.g., peers). Effective PMIs require strong collaborative relationships between SLPs and members of school support teams. In-service training and workshops should be provided to SLPs and other service providers such as school psychologists, counselors, paraprofessionals, and special education teachers. Regular education teachers are not typically involved in direct peer instruction, given that they have a classroom of students to manage; however, they can be instrumental in capitalizing on classroom or playground social opportunities to reinforce target social communication goals in inclusive settings. Training and involvement of multiple treatment providers can lead to improved generalized social outcomes if everyone is aware of and working on the same peer-related social communication goals.

See Also

- Circle of Friends
- Peers, Exposure to
- Service Delivery Model
- Social Behaviors and Social Impairment

References and Reading

- American Speech-Language-Hearing Association. (2006). *Principles for speech-language pathologists in diagnosis, assessment, and treatment of autism spectrum disorders across the life span: Technical report*. Retrieved March 4, 2011 from <http://www.asha.org/members/deskref-journal/deskref/default>
- Baker, J. (2003). *The social skills picture book: Teaching play, emotion, and communication to children with autism*. Arlington: Future Horizons.
- Chan, J. M., Lang, R., Rispoli, M., O’Reilly, M., Sigafos, J., & Cole, H. (2009). Use of peer-mediated interventions in the treatment of autism spectrum disorders: A systematic review. *Research in Autism Spectrum Disorders*, 3, 876–889.
- English, K., Goldstein, H., Shafer, K., & Kaczmarek, L. (1997). Promoting interactions among preschoolers with and without disabilities: Effects of a buddy skills-training program. *Exceptional Children*, 63, 229–243.
- English, K., Shafer, K., Goldstein, H., & Kaczmarek, L. (2005). Teaching buddy skills to preschoolers. In M. L. Wehmeyer & M. Agran (Eds.), *Mental retardation and intellectual disabilities: Teaching students using innovative and research-based strategies* (pp. 177–195). Annapolis Junction: American Association on Mental Retardation.
- Goldstein, H., & Kaczmarek, L. (1992). Promoting communicative interaction among children in integrated intervention settings. In S. Warren & J. Reichle (Eds.), *Causes and effects in communication and language intervention* (pp. 81–111). Baltimore: Paul H. Brookes.
- Goldstein, H., Schneider, N., & Thiemann, K. (2007). Peer-mediated social communication intervention: When clinical expertise informs treatment development and evaluation. *Topics in Language Disorders*, 27, 182–199.
- Grandin, T. (1995). The learning style of people with autism: An autobiography. In K. A. Quill (Ed.), *Teaching children with autism: Strategies to enhance communication and socialization* (pp. 33–52). New York: Delmar.
- Haring, G. T., & Breen, G. C. (1992). A peer-mediated social network intervention to enhance the social integration of persons with moderate and severe disabilities. *Journal of Applied Behavioral Analysis*, 25, 319–333.
- Horner, R. H., et al. (1990). Toward a technology of “non-aversive” behavioral support. *Journal of the Association for Persons with Severe Handicaps*, 15, 125–132.

- Kelly, A. (1996). *Talkabout: A social communication skills package*. Bicester: Winslow.
- McConnell, S. (2002). Interventions to facilitate social interactions for young children with autism: Review of available research and recommendations for educational interventions and future research. *Journal of Autism and Developmental Disorders*, 32, 351–372.
- National Research Council. (2001). *Educating children with autism. Committee on Educational Interventions for Children with Autism. Division of Behavioral and Social Sciences and Education*. Washington, DC: National Academy Press.
- National Standards Project. (2009). *The national standards project addressing the need for evidence based practice guidelines for autism spectrum disorders*. Randolph: National Autism Center. Retrieved March 10, 2011 from www.nationalautismcenter.org
- Odom, S., & McConnell, S. (1997). *Play time/social time: Organizing your classroom to build interaction skills*. Minneapolis: Institute on Community Integration, University of Minnesota.
- Prizant, B., & Wetherby, A. (1998). Understanding the continuum of discrete-trial traditional behavioral to social-pragmatic developmental approaches in communication enhancement for young children with Autism/PDD. *Seminars in Speech and Language*, 19, 1–45.
- Reichow, B., & Volkmar, F. (2010). Social skills interventions for individuals with autism: Evaluation for evidence-based practices within a best evidence synthesis framework. *Journal of Autism and Developmental Disorders*, 40, 149–166.
- Schwartz, I. (2000). Standing on the shoulders of giants: Looking ahead to facilitating membership and relationships for children with disabilities. *Topics in Early Childhood Special Education*, 20, 123–128.
- Thiemann, K., & Goldstein, H. (2001). Social Stories, written text cues, and video feedback: Effects on social communication of elementary school-age children with autism. *Journal of Applied Behavior Analysis*, 34, 425–446.
- Thiemann, K., & Kamps, D. (2008). Promoting social communication competence of children with autism in integrated environments. In R. Simpson & B. Myles (Eds.), *Educating children and youth with autism* (2nd ed., pp. 267–298). Austin: Pro-Ed.
- Thiemann, K., Goldstein, H., & Vuong, N. (2012). *Partnering with elementary school staff to increase social communication of students with autism across-the-school day*. Manuscript submitted for publication.
- Thiemann-Bourque, K. (2010). Navigating the transition to middle school: Peer network programming for students with autism. *The ASHA Leader*, 15, 12–15.
- Thiemann-Bourque, K., & Goldstein, H. (2004). Effects of peer training and written text cueing on social communication of school-age children with autism. *Journal of Speech, Language, & Hearing Research*, 47, 126–144.
- Wetherby, A., & Prutting, C. (1984). Profiles of communicative and cognitive-social abilities in autistic children. *Journal of Speech and Hearing Research*, 27, 364–377.

PEERS

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Definition

The Program for the Education and Enrichment of Relational Skills (PEERS®), developed at the University of California, Los Angeles (UCLA), by Drs. Elizabeth Laugeson and Fred Frankel, is a caregiver-assisted group treatment intervention aimed at improving social skills among youth with autism spectrum disorder (ASD) and other social challenges. This international program, which is used in over 90 countries and has been translated into over a dozen languages, is one of the only evidence-based social skills interventions for youth with ASD. The group-based intervention has been developed and tested for transitional youth from adolescence to early adulthood and has generated four published evidence-based treatment manuals for practitioners and educators.

Social Skills for Teenagers with Developmental and Autism Spectrum Disorders: The PEERS® Treatment Manual (Laugeson and Frankel 2010), which is a 14-week parent-assisted intervention for adolescents with ASD, was the first published manual to highlight this empirically supported program. This manual has also been translated into Korean (Laugeson and Frankel 2014), following a cross-cultural validation trial. *The PEERS® Curriculum for School-Based Professionals: Social Skills Training for Adolescents with Autism Spectrum Disorder* (Laugeson 2014) extends the PEERS® intervention into the school setting using a teacher-facilitated model with a 16-week daily lesson format. *PEERS® for Young Adults: Social Skills Training for Adults with Autism Spectrum Disorder and Other Social Challenges* (Laugeson and Frankel 2017) is the most recent publication. Geared for young adults with ASD, this caregiver-assisted intervention includes 16 weekly group treatment sessions to improve social functioning.

The PEERS[®] curriculum teaches ecologically valid rules and steps of social etiquette in a simple way for teens and young adults with ASD to understand social contexts. While typically developing children and adolescents often learn basic social rules through observation of peer behavior and/or specific instruction from parents, understanding and utilizing social etiquette is often more difficult for adolescents and young adults with ASD. Instead, teaching social skills through the identification of concrete rules and steps of social etiquette, demonstrating these social behaviors through role-playing examples, and providing behavioral rehearsal exercises with performance feedback are the essential ingredients to improving social functioning using the PEERS[®] method. Moreover, repetition of rehearsal through homework and supervised implementation of the skills through parent, caregiver, and/or teacher coaching is also critical to the development of social skills among adolescents and young adults using the PEERS[®] method.

Historical Background

Numerous studies have shown that social skills are an important factor in long-term adjustment for individuals with ASD. Consequently, social skills training is a well-documented intervention strategy for children on the spectrum, having the potential to greatly impact quality of life and increase independence. Studies investigating the effectiveness of social skills training for individuals with ASD indicate that intervention during childhood and adolescence is critical. However, to date there are very few evidence-based treatments focused on improving social skills for adolescents or young adults with autism. Meta-analyses of the literature suggest that social skills training does not produce large, socially important, long-term, or generalized changes in social competence among individuals with ASD (Reichow and Volkmar 2010; Rao et al. 2008; Reichow et al. 2013). These limitations are what lead to the development of PEERS[®].

Originally developed in 2004 at UCLA by Drs. Elizabeth Laugeson and Fred Frankel, PEERS[®] is

one of the only evidence-based social skills interventions for adolescents and adults with ASD. Numerous scientific papers have highlighted the efficacy and effectiveness of the program for adolescents with ASD (Laugeson et al. 2009, 2012, 2014; Van Hecke et al. 2015; Karst et al. 2015; Schohl et al. 2013; Yoo et al. 2014), adults with ASD (Gantman et al. 2012; Laugeson et al. 2015), and even youth with other social challenges like attention-deficit/hyperactivity disorder (ADHD; Gardner et al. 2015). Most of these studies have utilized randomized controlled trial designs, which is widely considered the gold standard for assessing treatment outcome.

Rationale or Underlying Theory

Utilizing the principles of cognitive behavioral therapy (CBT), PEERS[®] applies the teaching methods of didactic instruction/psychoeducation, role-play demonstration, cognitive strategies, behavioral rehearsal, performance feedback, homework assignment and review, and parent involvement within a small group treatment format (e.g., eight to ten group members). The general effectiveness of the PEERS[®] method might be credited to the use of evidence-based methods of treatment delivery, borrowed from the CBT approach, in combination with the inclusion of parents and/or other caregivers in treatment, who ensure the generalizability and durability of treatment gains in the months and years following intervention (Mandelberg et al. 2014).

Goals and Objectives

PEERS[®] focuses on teaching social skills related to making and keeping friends, as well as managing conflict and rejection. Didactic content for developing romantic relationships is also included in the young adult curriculum. Lessons include starting and maintaining conversations, finding a source of friends, electronic forms of communication, using humor appropriately, entering and exiting conversations, good sportsmanship (adolescents only), organizing and having successful get-togethers,

dating etiquette (adults only), managing disagreements, changing a bad reputation (adolescent only), and handling direct and indirect bullying and other forms of rejection (teasing, physical bullying, cyberbullying, and rumors/gossip).

Rules and steps of social behavior are developed from research evidence regarding (1) the common social errors often committed by those with ASD, (2) the core social skills needed to make and keep friends and develop romantic relationships, and (3) the ecologically valid ways in which socially accepted individuals handle peer conflict and rejection.

The PEERS[®] manuals are meant to be used as complete programs in their entirety. Lessons are intended to be delivered in the order they are presented as each skill builds upon the last. Didactic lessons are based upon ecologically valid social skills known to be used by socially successful people. The techniques used, such as role-play demonstrations, behavioral rehearsal exercises, and homework assignments, have been shown to improve social skills outcomes through research.

Treatment Participants

The PEERS[®] intervention is intended for socially motivated adolescents and adults with ASD without significant intellectual disabilities. The treatment was originally developed as a parent-assisted intervention focusing on teens in middle school and high school who were having difficulty making and keeping friends. The program has been field tested extensively with teens and young adults with ASD and to a lesser extent with adolescents diagnosed with intellectual disabilities and fetal alcohol spectrum disorders, as well as adolescents and adults with attention deficit hyperactivity disorder (ADHD). PEERS[®] is also used clinically with teens and young adults with depression, anxiety, and other social challenges.

Treatment Procedures

Research assessing psychosocial functioning among adolescents and adults with ASD has

found that these individuals often have extensive need for help from their families and/or society. Studies report that very few adults on the spectrum live in typical psychosocial conditions. Instead, they most often live with parents or alone, seldom have relationships with partners, and are often without employment of any kind. These outcomes suggest the need for parent, caregiver, and/or teacher involvement in treatment to improve social functioning for transitional youth with ASD.

One method utilized by PEERS[®] for teaching social skills to youth with ASD includes parent assistance. In this case, parents are included in the treatment, attending separate but concurrent weekly group sessions in which they are taught to be social coaches to their teens. Meanwhile, adolescents are given instruction on the rules and steps of social etiquette related to making and keeping friends while given opportunities to practice newly learned skills.

Another method used by PEERS[®] for teaching social skills to youth with ASD involves teacher facilitation. In this case, social skills are taught in the classroom, much in the way one might teach math or science. Lessons are broken down using concrete rules and steps of social etiquette, taught by teachers, demonstrated by teaching staff, and practiced daily by students in the classroom.

In addition to parent-assisted and teacher-facilitated social skills training, PEERS[®] also utilizes a caregiver-assisted model in working with young adults with ASD. In this case, social coaching is provided by caregivers close to the young adults, such as parents, adult siblings, peer mentors, job coaches, life coaches, or other family members.

In the parent-/caregiver-assisted interventions, treatment consists of 14–16 weekly 90-minute group sessions. Adolescents, young adults, and their caregivers attend separate concurrent sessions. Didactic instruction is provided using concrete rules and steps of ecologically valid social behavior. Within the adolescent and young adult groups, social rules and steps are presented using a Socratic method of questioning, intending to promote and enhance participation in the lesson. Role-play demonstrations of targeted behaviors

are also used to model appropriate and inappropriate examples of the rules and steps. In order to enhance social cognition, role-play demonstrations are followed by perspective-taking questions in which participants are asked to take on the perspective of the receiver of the appropriate or inappropriate social behavior. Structured practice follows each lesson through a behavioral rehearsal exercise in which young adults practice the appropriate newly learned skills while receiving performance feedback through social coaching by the treatment team. Socialization homework assignments are given for each of the targeted social skills to support generalization of skills outside of the treatment setting. Homework review takes place in the following week, with sufficient time to troubleshoot any issues that may have arisen and to individualize the treatment to participants as needed.

Within the parent and caregiver groups, homework review comprises the majority of the session in order to include specific instructions on how to provide assistance with social coaching to adolescents and young adults during weekly homework assignments. Didactic instruction follows homework review in the parent and caregiver groups through the distribution and review of social coaching handouts, which provide an outline of the rules and steps of the targeted social skills, along with a comprehensive description of the upcoming homework assignments. Within the didactic lesson, parents and caregivers are also given specific instruction on how to provide social coaching to adolescents and young adults outside of the treatment setting. These strategies are tailored to the specific needs of each individual as necessary. The use of parent/caregiver-assistance in treatment is utilized in order to enhance generalization of social skills through in vivo social coaching in natural social settings (when appropriate) and to increase homework compliance and practicing of newly learned skills. Parents and caregivers are also expected to enhance the durability of treatment gains through continual social coaching even after the PEERS[®] intervention had ended.

Within the teacher-facilitated intervention, the PEERS[®] curriculum consists of daily 30–50 min

lessons, delivered in the school setting 5 days per week over the course of 16 weeks. Key elements of the intervention are also taught using evidence-based methods of instruction provided by teaching staff. Didactic instructions about simple rules and steps of social etiquette related to making and keeping friends and handling peer conflict and peer rejection are provided using ecologically valid social skills based on the norms established by socially accepted teens. Didactic lessons are followed by role-play demonstrations of targeted skills by teaching staff. Students practice newly learned skills in the classroom while receiving performance feedback from teaching staff during behavioral rehearsal exercises. Socialization homework assignments are given to generalize skills outside of the school setting and are reviewed by students and teaching staff at the beginning of each week. Parents also receive psychoeducation about skills needed for teens to develop and maintain friendships via weekly parent handouts, which are generally sent home through classroom communication logs or via email. Through these handouts, parents are provided instruction about how to help their teen make and keep friends and assist their teen in expanding his/her social network through the enrollment in extracurricular activities.

Efficacy Information

While social skills training is an important treatment priority for many young adults with ASD and other social challenges, much of the research literature in this area has focused on interventions with children. Very few social skills intervention studies have been devoted to investigating the efficacy and effectiveness of social skills training for adolescents or young adults. Moreover, few social skills programs focus on the development and maintenance of friendships and romantic relationships. Even among the social skills intervention studies conducted with the ASD population, most have not been formally tested in terms of improving social competence or the development of close friendships, nor have they assessed the benefit of including caregivers in training in order

to promote social functioning. The lack of evidence-based social skills instruction to improve social competence and promote the formation of friendships for young adults with ASD and other social challenges is what inspired the development of PEERS®.

Most of the research examining the efficacy of PEERS® has been conducted at the UCLA Semel Institute for Neuroscience and Human Behavior, under the direction of Dr. Elizabeth Laugeson, the co-developer of the intervention. Multiple independent replication trials have also been conducted at Marquette University, under the direction of Dr. Amy Van Hecke, and several cross-cultural validation trials of PEERS® have also taken place outside of North America, with the first cross-cultural validation study lead by Dr. Heejeong Yoo at Seoul National University in South Korea. Across all of these trials, most of which have included randomized controlled trial designs, the treatment effects have been strikingly similar.

Results from the aforementioned research labs demonstrate that PEERS® is efficacious in improving social skills for adolescents and young adults with ASD without intellectual disability (Laugeson et al. 2009, 2012, 2014, 2015; Van Hecke et al. 2015; Karst et al. 2015; Schohl et al. 2013; Yoo et al. 2014; Gantman et al. 2012). Research findings primarily come from parents, teachers, and youth, using standardized measures of social functioning. Improvements in social functioning following PEERS typically include:

- Improved overall *social skills* in the areas of:
 - Cooperation
 - Assertion
 - Responsibility
- Decreased *problematic social behaviors* in the areas of:
 - Self-control
 - Externalizing behavior
- Improved *social responsiveness* in the areas of:
 - Social communication
 - Social awareness
 - Social motivation
 - Social cognition
 - Decreased autistic symptoms

- Decreased social anxiety
- Increased frequency of peer interactions and get-togethers
- Decreased loneliness
- Improved empathy
- Improved friendship quality
- Improved knowledge of adolescent or young adult social skills

Unlike most social skills interventions cited in the research literature, extensive follow-up research has been conducted with PEERS®, investigating the sustainability of these improvements over time (Mandelberg et al. 2014). A long-term follow-up study conducted at UCLA with families 1–5 years after receiving the PEERS® treatment revealed that improvements in social skills, social responsiveness, frequency of peer interactions, and social skills knowledge were maintained over time and in some cases improved even further. These findings are encouraging when you consider that the social trajectory for many young people with ASD sadly tends to worsen with age and upon entering adulthood.

Also noteworthy is that the abovementioned studies not only comprise the largest number of participants reported in the social skills treatment literature for older adolescents and young adults with ASD, but the improvements are far greater than what is typically reported in the autism research literature. Most social skills treatment studies for young people with ASD tend to show minimal or modest improvements, often with a very small group of people, and with improvements rarely sustained or reported over time. Conversely, research using the PEERS® model has shown much greater improvements in social skills among larger groups of people, with improvements generally maintained over time and improved even further in some cases.

Outcome Measurement

Tracking progress is an essential part of determining whether treatment is working. Below is a list of the tests used in published scientific studies

with PEERS[®]. Standardized assessments of social functioning are included. These measures are widely available and have shown substantial change following the program.

Test of Adolescent/Young Adult Social Skills Knowledge (TASSK/TYASSK; Laugeson and Frankel 2010; Laugeson 2014; Laugeson 2017). The TASSK and TYASSK are 30-item criterion-referenced tests developed for PEERS[®] to assess knowledge about the specific social skills taught during the intervention. Items are derived from key elements from each of the 15 didactic lessons. Adolescents (TASSK) and young adults (TYASSK) are presented with sentence stems and asked to choose the best option from two possible answers. Scores range from 0 to 30, with higher scores reflecting greater knowledge of adult social skills. Previous research using the TASSK/TYASSK to track treatment outcome in PEERS[®] generally yields a 6–8 point raw score improvement from pre- to posttest.

Quality of Socialization Questionnaire (QSQ; Laugeson and Frankel 2010; Laugeson 2014; Laugeson 2017). The QSQ is independently administered to adolescents, young adults, and caregivers to assess the frequency of get-togethers with peers in the previous month and the level of conflict during get-togethers with peers. Previous research using the QSQ to track treatment outcome in PEERS[®] generally reveals two to four additional get-togethers per month from pre- to posttest.

Social Responsiveness Scale – Second Edition (SRS-2; Constantino 2012). The SRS-2 consists of 65 items assessing the presence and severity of social deficits (i.e., social awareness, social cognition, social communication, social motivation, and restricted interests and repetitive behaviors) associated with ASD as they occur in natural social settings. The SRS-2 can be completed by parents, teachers, and other caregivers. Previous research using the SRS-2 to track treatment outcome in PEERS[®] generally yields a full standard deviation improvement (10 T-score points) from pre- to posttest.

Social Skills Improvement System (SSIS; Gresham and Elliott 2008). The SSIS is a standardized, 75-item measure that assesses social skills (communication, cooperation, assertion,

responsibility, empathy, engagement, self-control) and problem behaviors (externalizing and internalizing behaviors, bullying, hyperactivity and inattention, autism spectrum disorder-related behaviors). Although developed for youth between 13 and 18 years, the SSIS has also been used successfully with young adults to track change in social skills following PEERS[®] (Gantman et al. 2012; Laugeson et al. 2015). Using a four-point rating system, parents, teachers, and caregivers rate the frequency and relative importance of various social skills and problem behaviors. Previous research using the SSIS to track treatment outcome in PEERS[®] typically reveals nearly a standard deviation improvement (10 standard score points) from pre- to posttest.

Qualifications of Treatment Providers

Clinicians need not have much experience running social skills groups in order to use the manual effectively, but they should have background knowledge in working with adolescents or young adults with ASD or other social challenges and their caregivers. Since anyone is able to purchase the PEERS[®] manuals commercially, the program is used extensively throughout the world by practitioners and educators with very little training in social skills treatment. For those wishing to obtain more extensive training on the implementation of this intervention, PEERS[®] Certified Training Seminars for qualified medical/mental health professionals and educators are regularly conducted at and outside of UCLA by the program developer, but such training is not required to run a PEERS[®] program.

See Also

- ▶ [Social Behaviors and Social Impairment](#)
- ▶ [Social Cognition](#)
- ▶ [Social Communication](#)
- ▶ [Social Interventions](#)
- ▶ [Social Responsiveness Scale](#)
- ▶ [Social Skills Improvement System](#)
- ▶ [Social Skill Interventions](#)

References and Reading

- Constantino, J. N. (2012). *Social responsiveness scale-second edition*. Los Angeles: Western Psychological Services.
- Gantman, A., Kapp, S. K., Orenski, K., & Laugeson, E. A. (2012). Social skills training for young adults with autism spectrum disorders: A randomized controlled pilot study. *Journal of Autism and Developmental Disorders*, 42(6), 1094–1103.
- Gardner, D. M., Gerdes, A. C., & Weinberger, K. (2015). Examination of a parent-assisted, friendship-building program for adolescents with ADHD. *Journal of Attention Disorders*. <https://doi.org/10.1177/1087054715588188>.
- Gresham, F. M., & Elliott, S. (2008). *The social skills improvement system*. Minneapolis: American Guidance Service.
- Karst, J. S., Van Hecke, A. V., Carson, A. M., Stevens, S., Schohl, K., & Dolan, B. (2015). Parent and family outcomes of PEERS: A social skills intervention for adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(3), 752–765.
- Laugeson, E. A. (2014). *The PEERS curriculum for school based professionals: Social skills training for adolescents with autism spectrum disorder*. New York: Routledge.
- Laugeson, E. A., & Frankel, F. (2010). *Social skills for teenagers with developmental and autism spectrum disorders: The PEERS treatment manual*. New York: Routledge.
- Laugeson, E. A., & Frankel, F. (2014). *Social skills for teenagers with developmental and autism spectrum disorders: The PEERS Korean treatment manual*. New York: Routledge.
- Laugeson, E. A. (2017). *The PEERS treatment manual for young adults with autism spectrum disorder: Evidence-based social skills training*. New York: Routledge.
- Laugeson, E. A., & Park, M. N. (2014). Using a CBT approach to teach social skills to adolescents with autism spectrum disorder and other social challenges: The PEERS method. *Journal of Rational-Emotive & Cognitive-Behavior Therapy*, 32(1), 84–97.
- Laugeson, E. A., Frankel, F., Mogil, C., & Dillon, A. R. (2009). Parent-assisted social skills training to improve friendships in teens with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 596–606.
- Laugeson, E. A., Frankel, F., Gantman, A., Dillon, A. R., & Mogil, C. (2012). Evidence-based social skills training for adolescents with autism spectrum disorders: The UCLA PEERS program. *Journal of Autism and Developmental Disorders*, 42(6), 1025–1036.
- Laugeson, E. A., Ellingsen, R., Sanderson, J., Tucci, L., & Bates, S. (2014). The ABC's of teaching social skills to adolescents with autism spectrum disorder in the classroom: The UCLA PEERS program. *Journal of Autism and Developmental Disorders*, 44, 2244–2256.
- Laugeson, E. A., Gantman, A., Kapp, S. K., & Orenski, K. (2015). A randomized controlled trial to improve social skills in young adults with autism spectrum disorder: The UCLA PEERS Program. *Journal of Autism and Developmental Disorders*, 45(12), 3978–3989.
- Mandelberg, J., Laugeson, E. A., Cunningham, T. D., Ellingsen, R., Bates, S., & Frankel, F. (2014). Long-term treatment outcomes for parent-assisted social skills training for adolescents with autism spectrum disorders: The UCLA PEERS program. *Journal of Mental Health Research in Intellectual Disabilities*, 7(1), 45–73.
- Rao, P. A., Beidel, D. C., & Murray, M. J. (2008). Social skills interventions for children with Asperger's syndrome or high-functioning autism: A review and recommendation. *Journal of Autism and Developmental Disorders*, 38, 353–361.
- Reichow, B., & Volkmar, F. R. (2010). Social skills interventions for individuals with autism: Evaluation for evidence-based practices with a best evidence synthesis framework. *Journal of Autism and Developmental Disorders*, 40, 149–166.
- Reichow, B., Steiner, A. M., & Volkmar, F. (2013). Cochrane review: Social skills groups for people aged 6 to 21 years with autism spectrum disorders (ASD). *Evidence-Based Child Health*, 8, 266–315.
- Schohl, K. A., Van Hecke, A. V., Carson, A. M., Dolan, B., Karst, J., & Stevens, S. (2013). A replication and extension of the PEERS intervention: Examining effects on social skills and social anxiety in adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 354–366.
- Van Hecke, A. V., Stevens, S., Carson, A. M., Karst, J. S., Dolan, B., Schohl, K., et al. (2015). Measuring the plasticity of social approach: A randomized controlled trial of the effects of the PEERS intervention on EEG asymmetry in adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 45, 316–335.
- Yoo, H. J., Bahn, G., Cho, I. H., Kim, D. K., Kim, J. H., Min, J. W., et al. (2014). A randomized controlled trial of the Korean version of the PEERS parent-assisted social skills training program for teens with ASD. *Autism Research*, 7(1), 145–161.

Peers, Exposure to

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Definition

Exposure to typically developing peers is often used as an intervention to improve the socialization

of children with autism spectrum disorders (ASD). Exposure to typical peer models provides examples of age-appropriate social behavior as well as opportunities to engage in those age-appropriate activities. Exposure to peers is also used to try to improve peer acceptance of children with ASD and appreciation of differences. Interventions utilizing exposure to typically developing peers often occur in the school setting, in inclusive classrooms or environments. Peers may be trained or untrained, and adults may or may not facilitate interactions between the peers and child with ASD. However, there is an overall emphasis to reduce adult intrusion and instead arrange the physical space to increase the likelihood of interactive play (Bellini et al. 2007b).

Historical Background

Peer relationships are considered to be a crucial factor for healthy academic and social development (Birch and Ladd 1998). There has been an increasing push to include children with ASD into regular education classrooms (Falvey 1995; Guralnick 1990; Kasari et al. 1999; Villa and Thousand 1995). One goal of inclusion is to increase the exposure of children with ASD to typically developing peers, and ultimately develop social relationships (Gallagher et al. 2000). In autism-specific classrooms, children are often surrounded by children with similar social challenges to themselves and do not have the same opportunities to observe or practice peer-peer positive social interactions. Children with ASD have difficulties in social interaction as a hallmark of the disorder (American Psychiatric Association [APA] 2000). These social deficits can affect children with ASD's theory of mind (the ability to take another person's perspective; Peterson et al. 2007), communication skills (both verbal and nonverbal), interpretation skills of other's behavior, as well as their ability to stay on socially relevant topics (Jackson et al. 2003; Palmen et al. 2008). Children with ASD seem to lack two of the most important qualities for successful peer relationships: the ability to relate

positively and reciprocally to peers and the ability to easily adapt to changes in social situations (Schopler and Mesibov 1986). Complex communication skills require a high level of sensitivity in order to recognize social cues, and children with ASD often lack the social skills required to interpret those cues and connect with others (Chawarska et al. 2003). An inability to effectively communicate can severely impede the development and maintenance of peer relationships, when children cannot accurately understand one another, and having two children with social difficulties trying to interact only increases this challenge. By exposing the child with ASD to typically developing peers, there is an increased opportunity for the peer to adapt to the child with ASD's unique communication style and interact successfully. Children with ASD also do not attend to social cues, such as eye gaze, similar to their typical peers, and appear to process these visual cues differently (Chawarska et al. 2003). Other social challenges of children with ASD include inappropriate behaviors, such as failing to initiate conversations and/or respond to others, difficulties in turn-taking, and perseveration (APA 2000). In addition, children with ASD may respond to failures in communication by getting frustrated and reverting to earlier forms of communication, such as crying and throwing tantrums, instead of working to repair the miscommunication (Koegel et al. 2001). Children with ASD may also have disruptive, aggressive, age-inappropriate, or repetitive behaviors, such as hand-flapping, echolalia, and prosody of speech (Downs and Smith 2004; McEvoy et al. 1993). As a result of these challenges, many children with ASD have difficulty developing and maintaining peer relationships. Exposure to typically developing peers has the goal of improving social engagement and addressing some of these core deficits.

Rationale or Underlying Theory

Children with disabilities are often stigmatized, and the negative effects of stigmatization can be carried into adulthood (Koegel et al. 2001). Similarly, children who demonstrate difficulties

forming peer relationships in their earlier years have a higher incidence of emotional maladjustment in later years (Schopler and Mesibov 1986). Children with ASD do not engage in spontaneous play with other children and often fail to develop meaningful friendships (Koegel et al. 2001). However, positive peer relationships are related to improved academic performance, physical and behavioral outcomes, and increased feelings of companionship, self-worth, and self-esteem and may serve as a protective factor (Brendgen et al. 2005; Farmer et al. 2008; Gest et al. 2001). Thus, promoting the development of peer relationships is crucial for the social and emotional development of children with ASD.

Exposure to typically developing peers offers a unique method of social training for the child with ASD. Peers are of similar age to the child with ASD and thus can become highly salient for the child with ASD to observe and try to emulate (as opposed to exposure to adults). Peers can also provide a desired acceptance and social praise for the child with ASD. Some children with ASD have been shown to be aware of their social status within the classroom and have shown increased ratings of loneliness as a result of that understanding (Bauminger and Kasari 2000). Using peers to teach children with ASD how to appropriately interact socially may provide some inherent social rewards and reinforcement for the child with ASD to not only increase the desired social behavior but also increase their motivation to continue to emulate and seek out social interactions with their peers. Often when trying to shape the behavior of a child with ASD, artificial reinforcers are used (e.g., stickers, tokens, time on the computer) to reward the child for desired behavior. However, when peers respond positively to the child with ASD for exhibiting appropriate social behavior, that natural reward may be a more powerful reinforcer and motivator to increase the desired behavior than artificial tokens. Even using artificial rewards for appropriate behavior in real-life social experiences (e.g., rewarded for play on the playground) is more likely to achieve real and longer-lasting results than rewards for appropriate role-play in artificial clinical settings.

Goals and Objectives

Children with ASD are exposed to typical peers with the goal of increasing interaction and appropriate social skills as well as exposing the child to more age-appropriate behavior. There is a hope that exposure to typical peer models will result in a corresponding increase in the social skills and generalizability of those skills in the natural setting by children with ASD. Since play is important in forming a basis for peer relationships, many interventions focus on teaching and promoting specific play skills (Schopler and Mesibov 1986). Targeting specific behaviors, such as decreasing stereotypic behaviors and tantrums and increasing play and cooperation, may also play an important role in long-term development and contribute to subjective observers' improved global impressions of the child with ASD (Koegel et al. 2001).

Exposure to typically developing peers also provides naturalistic rewards for age-appropriate social behavior as well as increased opportunities for social communication, play, and social engagement. Some of the goals of exposure for peers to children with ASD are to increase their acceptance of children with special needs as well as increasing their understanding, empathy, and adaptive skills.

Treatment Participants

Treatment involving exposure to peers has focused mainly on children with ASD under the age of 15 years and their typically developing, preferably same-aged peers. Only a few studies have examined older students (Harris 1998). Typically developing peers can be selected based on classroom proximity or on specific qualities of the peer (e.g., empathetic, having good social skills, or of average social acceptance themselves; Kasari et al. 2010). Adults, such as parents and teachers, are also used to teach typical peers how to appropriately model social skills to children with ASD. However, the involvement of adults is often for peer training and observation purposes only and is otherwise minimal (Bellini et al. 2007b).

Treatment Procedures

Studies looking at exposure to peers can take many forms. Inclusion models often use classroom proximity alone to expose children with ASD to typically developing peer models. Classroom proximity may also be used with school- or class-wide training in appropriate positive social behavior (e.g., school-wide rules such as “everyone is allowed to play”). Targeted training of peers has also been used to facilitate interaction with children with ASD, where peers are specifically trained in social engagement techniques. Children with ASD can also be trained in specific social skills prior to social exposure to peers to increase the likelihood of appropriate social interaction. Finally, adults can serve as facilitators to increase the opportunities for interaction between the peers and the child with ASD (Luiselli et al. 2008). Procedures used in training of the child with ASD or typically developing peers can range from modeling, role-play, discrete trial training, social stories, and pivotal response training to self-management training or assigning peer buddies (Ferraioli and Harris 2010; Luiselli et al. 2008). Peers play a variety of roles, acting as tutors, models, partners, and trainers (Luiselli et al.).

Most of the interventions promoting exposure to peers have utilized single-subject designs, with a variation of multiple-baseline or probe design being the most common (Bellini et al. 2007a). There is an overall lack of consistency in interventions, with the number of sessions ranging from 8 to 73 sessions and treatment ranging from 2.5 to 28 h and 10–210 days (Bellini et al. 2007a). The interventions have been conducted in schools, in classrooms, and in common areas, such as playgrounds and cafeterias, the child’s home, and the community.

Peer exposure training is also used to teach a variety of social skills and behaviors. Much of the research literature has focused on verbal communication, such as initiation and responses, and social behaviors, such as sharing, helping, and showing affection (Bellini et al. 2007b; Rotheram-Fuller and Kasari 2010). There are two main categories of behaviors targeted by peer-mediated interventions: specific, well-defined

skills or behaviors or more diffuse socialization ability without a focus on particular skills (Rotheram-Fuller and Kasari 2010). Some of the specific behaviors that have been studied are smiling and laughing with peers, entering ongoing peer activities, and giving compliments and praise (Schopler and Mesibov 1986). The more general approach has focused on promoting overall social interaction skills in a less specified manner and may utilize social skills or friendship groups to increase participation in peer activities or cooperative play or measure complex concepts, such as friendship quality (Rotheram-Fuller and Kasari 2010; Schopler and Mesibov 1986). According to Koegel et al. (2001), intervention focus should shift away from studying specific targeted behaviors to a more global approach that focuses on results, and emphasis should be placed on identifying the behaviors that will impact other behaviors, rather than treating each individual behavior independently.

Efficacy Information

There is mixed evidence for the efficacy of exposure to peers as an intervention technique. Exposure to peers has been shown to help children with ASD form successful relationships with typically developing peers and to improve social skills (Carr and Darcy 1990; Garfinkle and Schwartz 2002; Ryndak et al. 1995). Exposure to multiple peers has also been shown to increase the generalizability and maintenance effects of the social skills treatments (Bellini et al. 2007b).

Exposure to peers may also indirectly improve the social skills of the typical peer in addition to the social skills of the child with ASD. Typical peers may become more sympathetic and empathetic, show increased tolerance and social awareness, and learn how to reduce the stress of interacting with the child with ASD (Koegel et al. 2001).

However, other peer-mediated interventions have had limited success due to the difficulty of teaching children with ASD how to spontaneously initiate social interactions (Harris 1998). Much of the training has been focused on

initiating social interactions; however, children with ASD do not easily transform initiations into extended conversations (Rotheram-Fuller and Kasari 2010). Another challenge in evaluating the efficacy of peer-mediated interventions is the lack of consistency of interventions and their outcome measures. Many studies collect outcomes measuring the specific skills that were targeted in the interventions, which make it difficult to determine if improvements have been made in other areas, such as the child's global social functioning.

Traditional programs of simple exposure through proximity have been minimally effective in teaching social skills to children with ASD; however, programs that target specific social skills have been found to be more effective than programs focusing on global social functioning (Bellini et al. 2007b).

Peer interactions with children with ASD occur more frequently in inclusive settings; although, research on inclusion suggests that inclusion and peer exposure are not sufficient for teaching children with ASD the more subtle social skills, such as keeping up with topic changes in conversation (Wagner 1999). Children in inclusive classrooms are simply placed in proximity to the other children in the class, without scaffolding on how to appropriately interact. While typically developing peers naturally tend to interact with others similar to themselves (Kemple 2004), this leaves children with ASD within that classroom at a disadvantage. It is often necessary for adults to facilitate social interaction in order for inclusion to be truly successful (Kemple 2004).

In a recent study comparing the efficacy of training the child with ASD compared to training typical peers to engage with the child with ASD, it was found that training peers can be more successful at improving the social involvement of the child with ASD into the regular classroom (Kasari et al. 2010). Peer interventions have been shown to be particularly effective in generalizing the skills across settings and people (Bellini et al. 2007b; Luiselli et al. 2008) and also have been found to be maintained over time (Mastergeorge et al. 2003). Parents and teachers have also reported increases in language and appropriate play skills and decreases in solitary play

(Wagner 1999). However, it is recommended that interventions should be implemented more frequently and intensely than is typical (Bellini et al. 2007b). More information is needed, however, to identify which interventions work best with which children and who might benefit most from inclusion practices.

Outcome Measurement

The outcome measures used across studies of exposure to peers vary considerably. There is no standard measurement to determine success of the intervention, and studies utilize both specific and global measures. The majority of outcomes could be categorized into two main areas: observations of specific skills to gauge improvement and standardized measures (including teacher, parent, child, or peer reports; Bellini et al. 2007a; White et al. 2007). Given the spectrum nature of ASD, there are a broad set of target behaviors that are examined to determine social success. While an intervention targeting initiations must record initiations as an outcome, it is difficult to compare such an intervention to one that focuses on a different specific skill, such as increasing the number of friendships for the child. The specific skills that have been measured include social initiations, greetings, and responses, while the more global social skills include play skills, problem-solving skills, and exercising self-control (Bellini et al. 2007a; Rao et al. 2008).

Qualifications of Treatment Providers

No specialized training has been identified as necessary for providing treatment in peer exposure interventions. Parents, teachers, care providers, and siblings, as well as peers are typically used to provide training to the child with ASD (or typically developing peers). In the most basic form of exposure (proximity), children with ASD are exposed to typical peers in a naturalistic setting with no adult intervention or prior training. However, across studies, those that used more facilitation from adults or training of peers

prior to exposure appear more effective at improving the social engagement of the child with ASD. Thus, training on social skills curriculums or specific intervention methods (pivotal response training, discrete trial training, etc.) is recommended to increase the efficacy of this approach. Training for treatment providers typically involves ensuring that data is collected correctly; additional training should be efficient (quick, minimal resources), effective (produce the desired outcomes), and acceptable (address needs and promote generalization of skills; Luiselli et al. 2008). Similarly for teachers, no intervention is expected as part of an inclusion classroom; however, providing guidance or activities with the class can increase or facilitate social engagement among all students, including the child with ASD.

See Also

► Inclusion

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR* (4th ed.). Washington, DC: Author. Text rev.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendships in high-functioning children with autism. *Child Development, 71*, 447–456.
- Bellini, S., Peters, J. K., Benner, L., & Hopf, A. (2007a). A meta-analysis of school-based social skills interventions for children with autism spectrum disorders. *Remedial and Special Education, 28*, 153–162.
- Bellini, S., Peters, J. K., & Hopf, A. (2007b). Social skills training. In B. S. Myles, T. C. Swanson, J. Holwerstott, & M. M. Duncan (Eds.), *Autism spectrum disorders: A handbook for parents and professionals*. Westport: Greenwood.
- Birch, S. H., & Ladd, G. W. (1998). Children's interpersonal behaviors and the teacher-child relationship. *Developmental Psychology, 34*, 934–946.
- Brendgen, M., Wanner, B., Morin, A. J. S., & Vitaro, F. (2005). Relations with parents and with peers, temperament, and trajectories of depressed mood during early adolescence. *Journal of Abnormal Child Psychology, 33*, 579–594.
- Carr, E. G., & Darcy, M. (1990). Setting generality of peer modeling in children with autism. *Journal of Autism and Developmental Disorders, 20*, 45–59.
- Chawarska, K., Klin, A., & Volkmar, F. (2003). Automatic attention cueing through eye movement in 2-year-old children with autism. *Child Development, 74*, 1108–1122.
- Downs, A., & Smith, T. (2004). Emotional understanding, cooperation, and social behavior in high-functioning children with autism. *Journal of Autism and Developmental Disorders, 34*, 625–635.
- Falvey, M. A. (1995). *Inclusive and heterogeneous schooling: Assessment, curriculum, and instruction*. Baltimore: Paul H. Brookes.
- Farmer, T. W., Estell, D. B., Hall, C. M., Pearl, R., Van Acker, R., & Rodkin, P. (2008). Interpersonal competence configurations, behavior problems, and social adjustment in preadolescence. *Journal of Emotional and Behavioral Disorders, 16*, 195–212.
- Ferraioli, S. J., & Harris, S. L. (2010). Treatments to increase social awareness and social skills. In B. Reichow, P. Doehring, D. V. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-based practices and treatments for children with autism*. New York: Springer.
- Gallagher, P. A., Floyd, J. H., Stafford, A. M., Taber, T. A., Brozovic, S. A., & Alberto, P. A. (2000). Inclusion of students with moderate or severe disabilities in educational and community settings: Perspectives from parents and siblings. *Education and Training in Mental Retardation and Developmental Disabilities, 35*, 135–147.
- Garfinkle, A. N., & Schwartz, I. S. (2002). Peer imitation: Increasing social interactions in children with autism and other developmental disabilities in inclusive preschool classrooms. *Topics in Early Childhood Special Education, 22*, 26–38.
- Gest, S. D., Graham-Bermann, S. A., & Hartup, W. W. (2001). Peer experience: Common and unique features of number of friendships, social network centrality, and sociometric status. *Social Development, 10*, 23–40.
- Guralnick, M. J. (1990). Major accomplishments and future directions in early childhood mainstreaming. *Topics in Early Childhood Special Education, 10*, 1–17.
- Harris, S. L. (1998). Behavioral and educational approaches to the pervasive developmental disorders. In F. R. Volkmar (Ed.), *Autism and pervasive developmental disorders*. Cambridge, UK: Cambridge University Press.
- Jackson, C. T., Fein, D., Wolf, J., Jones, G., Hauck, M., Waterhouse, L., et al. (2003). Responses and sustained interactions in children with mental retardation and autism. *Journal of Autism and Developmental Disorders, 33*, 115–121.
- Kasari, C., Freeman, S., Bauminger, N., & Alkin, M. (1999). Parental perceptions of inclusion: Effects of autism and Down syndrome. *Journal of Autism and Developmental Disorders, 29*, 297–305.
- Kasari, C., Rotheram-Fuller, E., Locke, J., & Gulsrud, A. (2010). *Social inclusion of children with ASD at school: Effects of a randomized controlled treatment study*. Presentation at the 9th annual international meeting for autism research, Philadelphia.
- Kemple, K. M. (2004). The importance of peer competence and social inclusion. In *Let's be friends: Peer competence and social inclusion in early childhood programs* (pp. 1–14). New York: Teachers College Press.

- Koegel, L. K., Valdez-Menchaca, M., Koegel, R. L., & Harrower, J. K. (2001). Autism. In M. Hersen & V. B. Van Hasselt (Eds.), *Advanced abnormal psychology* (pp. 165–191). New York: Plenum Press.
- Luiselli, J. K., Russo, D. C., Christian, W. P., & Wilczynski, S. M. (Eds.). (2008). *Effective practices for children with autism: Educational and behavioral support interventions that work*. New York: Oxford University Press.
- Mastergeorge, A. M., Rogers, S. J., Corbett, B. A., & Solomon, M. (2003). Non-medical interventions for autism spectrum disorders. In S. Ozonoff, S. J. Rogers, & R. L. Hendren (Eds.), *Autism spectrum disorders: A research review for practitioners* (pp. 133–160). New York: American Psychiatric Publishing.
- McEvoy, R. E., Rogers, S. J., & Pennington, B. F. (1993). Executive function and social communication deficits in young autistic children. *Journal of Child Psychology and Psychiatry*, 34, 563–678.
- Palmen, A., Didden, R., & Arts, M. (2008). Improving question asking in high-functioning adolescents with autism spectrum disorders: Effectiveness of small-group training. *Autism*, 12, 83–98.
- Peterson, C. C., Slaughter, V. P., & Paynter, J. (2007). Social maturity and theory of mind in typically developing children and those on the autism spectrum. *Journal of Child Psychology and Psychiatry*, 48, 1243–1250.
- Rao, P. A., Beidel, D. C., & Murray, M. J. (2008). Social skills interventions for children with Asperger's syndrome or high-functioning autism: A review and recommendations. *Journal of Autism and Developmental Disorders*, 38, 353–361.
- Rotheram-Fuller, E., & Kasari, C. (2010). Peer relationships: Challenges and interventions. In E. Hollander, A. Kolevzon, & J. T. Coyle (Eds.), *Textbook of autism spectrum disorders* (pp. 555–564). Arlington: American Psychiatric Publishing.
- Ryndak, D. L., Downing, J. E., Jacqueline, L. R., & Morrison, A. P. (1995). Parents' perceptions after inclusion of their children with moderate or severe disabilities. *Journal of the Association for Persons with Severe Handicaps*, 20, 147–157.
- Schopler, E., & Mesibov, G. B. (Eds.). (1986). *Social behavior in autism*. New York: Plenum Press.
- Villa, R., & Thousand, J. (1995). *Creating an inclusive school*. Alexandria: Association for Supervision and Curriculum Development.
- Wagner, S. (1999). *Inclusive programming for elementary students with autism*. Arlington: Future Horizons.
- White, S. W., Keonig, K., & Scahill, L. (2007). Social skills development in children with autism spectrum disorders: A review of the intervention research. *Journal of Autism and Developmental Disorders*, 37, 1858–1868.

PEG

- [Pneumoencephalography](#)

Pemoline

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Synonyms

[Cylert](#)

Definition

Pemoline is an older psychostimulant that is now off the market. Pemoline shares features in common with amphetamine and methylphenidate. It was removed from the market due to concern about rare problems with liver failure. Pemoline has not been well studied in children with pervasive developmental disorders.

See Also

- [Stimulant Medications](#)

References and Reading

- Martin, A., Scahil, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.

People-First Language

- [Person-First Language](#)

PEP-3

- [Psychoeducational Profile – Revised \(PEP-3\)](#)

PEPS-C

► Profiling Elements of Prosody in Speech-Communication (PEPS-C)

Perception

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Definition

Perception includes the selection, organization, and interpretation of external information coming from the senses up to a complete representation of a stimulus. The extraction of elementary features within primary cortical receiving areas in each sensory modality is called low-level perception. It is domain general such that the same mechanisms are involved in processing social and nonsocial information. At a higher level of integration, there is pattern construction in which elementary features (e.g., of visual or auditory stimuli) are grouped into basic configurations. Visual motion perception depends on information integrated at various levels of the processing hierarchy: first-order motion is perceived in primary areas, while second-order motion requires a

network of associative regions. High-level perception involves the matching of constructed configurations with memorized templates as the hierarchy of perceptual processing becomes gradually more domain specific. In turn, domain-specific perception includes face and emotion perception, language perception, and, for dynamic information, perception of biological motion.

Perception typically feeds to and receives information from other major functions in the cognitive architecture, including emotion, language, memory, attention, expectations, and conscious reasoning. Thus, perceptual configurations may be associated with innate or memorized patterns of biological significance, pattern construction is influenced by language categories, and pattern recognition involves an active comparison of perceived and stored configurations. Perception is a dynamic process which modifies (bottom-up) and is modified by (top-down) higher-level cognitive processes.

Historical Background

Kanner's initial writings emphasized autistics' attention to and production of perceptual information (e.g., gazing at moving objects, lining up objects by color) as essential elements of autistic repetitive movements and behaviors. He vividly described extraordinary autistic achievements in memory, construction, and attention to detail. Perception-based behaviors, he noted, were associated with extreme emotions, such as being "ecstatic" while watching spinning objects or displaying panic in response to sounds. In his later writing, Kanner (1965, p. 412) underlined that being "so concerned with the external world that they watch (it) with tense alertness to make sure that their surroundings remain static (...) in full photographic and phonic identity" is inconsistent with the withdrawal into one's own world implied by the label autism.

Early models such as *perceptual inconstancy* (Ornitz 1973) were formed around the apparently paradoxical co-occurrence, within the same modality, of overt hypo- and hyperreactions to perceptual information (e.g., to voices and

vacuum cleaners, respectively). Atypical social and nonsocial representations were linked to subcortical abnormalities distorting perceptual input. However, absence of replicated brainstem event-related potential (ERP) abnormalities coupled with lack of empirical confirmation that perception in autism is unstable led to these models being abandoned in the early 1980s.

Meanwhile, early studies by Hermelin, O'Connor, and Frith in the 1960s and 1970s (Hermelin and O'Connor 1970; Frith and Hermelin 1969) established alternatively the absence of gross abnormalities in low-level processing. Performance in pattern reproduction, recognition, and memory was therefore considered to be unremarkable in autism. These studies were focused on the detection and imposition of a structure on visual or verbal materials, corresponding partly to pattern construction and partly to what would be now called top-down influence on perception. They concluded that autistic cognition was skewed toward meaningless or raw information, while structure imposition and recoding were diminished or absent. However, examples brought to support these positions – hyperlexia, echolalia, and 3-D drawing – implied pattern manipulation and not raw material. A decisive synthesis by Frith and Baron-Cohen (1987) concluded that “low level processes are intact in autistic children” (p. 98) and “what appear to be signs of a lower level dysfunction can be explained more powerfully in terms of a higher level cognitive dysfunction” (p. 87).

Those two conclusions, that autistic perception was unremarkable and that apparent atypicalities resulted from higher level impairment, dominated autism research for two ensuing decades, including by influencing the interpretation of autistic perceptual strengths. When administering visuo-spatial tasks, autistics were found to perform better than predicted by their other abilities and better than groups of intellectually disabled individuals matched on apparent IQ. In testing with Wechsler scales of intelligence, Rutter (1966) reported superior autistic performance in the block design subtest, which is based on visuo-spatial abilities. In 1983, this early accidental finding was extended by Shah and Frith in a

deliberate investigation of autistics' performance on the embedded figure task, which is also based on perception and manipulation of visual patterns. Autistics performed at a level superior to that predicted by their IQ (Shah and Frith 1983).

The weak central coherence (WCC) model, elaborated by Frith (and later, with Happé) starting in the late 1980s, accounted for such autistic peaks in performance by positing a causal deficit: autistics were not displaying atypical perceptual *strengths* but a *failure* to form global or high-level representations. WCC-based interpretations were consistent in direction with the period's cognitive research, which in the 1980s and 1990s remained oriented toward multiple high-level processing deficits, such as deficits in theory of mind, executive functioning, and complex tasks in general. In addition, Minshew's group discouraged diagnostic use of perceptual peaks by emphasizing that they were not found in all autistics. Fine-grained research on autistic perception emerged in the 1990s as a minority current.

The tenets of WCC were questioned in 1993 in the first study extensively investigating visual perception in an autistic adult, according to the concepts and instruments of cognitive neuropsychology at the time (Mottron and Belleville 1993). The local–global hierarchical aspect of visual perception was atypical in the form of greater interference from local to global levels and random access to features of perceptual representation but without deficits in pattern construction. Findings inconsistent with WCC-predicted deficits in auditory and visual pattern construction at the group level, together with discoveries of superior discrimination of low-level visual arrangements (Plaisted et al. 1998) and superior pitch processing (Heaton et al. 1998), then led to the enhanced perceptual functioning (EPF) model. This model originally aimed to relocate autistic perceptual peaks within an overall enhanced activity and performance of the perceptual system, including enhanced pattern construction mechanisms. However, EPF did not initially question Frith's or Minshew's preconception that autistics' overall superior perceptual performance was a secondary consequence of deficits in high-level

processes. This step was accomplished in an update of EPF (Mottron et al. 2006) based on the blossoming of studies on perception in autism after 2000.

The updated EPF model proposed that autism is characterized by enhanced perceptual *performance* in low-level (e.g., pitch processing) and mid-level (e.g., pattern detection) perception, enhanced *autonomy* of perception toward non-perceptual systems (emotional and higher-order architecture), and enhanced *role* of perception in higher cognitive processes (e.g., enhanced role of perception in intelligence and in social tasks). In other words, enhanced perception in autism was therefore proposed as such, rather than as evidence for speculated high-level deficits. Multiple other groups (including M. Behrmann, A. Bertone, P. Heaton, C. Manning, E. Milne, P. Mitchell, E. Pellicano, K. Plaisted, J. Remington, A. Swettenham, J. Wagemans) now investigate the relation between low-level perceptual processing and various aspects of autistic cognition.

Distant from the expanding interest in research, the importance of perception has also evolved within formal autism diagnostic criteria. Autism in DSM-III included indirect reference to perception-guided repetitive behaviors: “Bizarre responses to various aspects of the environment, e.g., resistance to change, peculiar interest in or attachments to animate or inanimate objects.” DSM-III-R referred to “Persistent preoccupation with parts of objects (for example, sniffing or smelling objects, repetitive feeling of texture of materials, spinning wheels of toy cars)” and DSM-IV to “Persistent preoccupation with parts of objects.” Perceptual issues are now more explicitly included in the DSM-5, albeit as “hyper- or hyporeactivity to sensory input or unusual interest in sensory aspects of the environment.” Perception-related behaviors are included in ADI-R and ADOS-G diagnostic instruments as atypical positive and negative reactions to sensory information (“unusual sensory interest,” “undue general sensitivity to noise,” and “abnormal, idiosyncratic, negative response to sensory stimuli”) or as repetitive behaviors possibly related to perception, as in “repetitive use of objects, hand and finger mannerisms.”

Current Knowledge

Perception-Related Behaviors

Perception-related behaviors are usually investigated under the overgeneral definitions of “repetitive” or “sensory” behaviors and by means of wide-ranging scales (e.g., Infant Toddler Sensory Profile; Dunn 2002) using a posteriori scoring in natural, nonempirical settings, with imprecise behavior definitions (e.g., scoring a behavior as present or absent). Their reported lack of specificity and poor sensitivity therefore reflect major methodological issues (Rogers and Ozonoff 2005). In a limited number of cases, studies of perception-related behaviors in empirical settings or using strict definitions have provided evidence regarding their specificity to autism and mechanisms involved in these behaviors. This is the case for atypical visual exploratory behaviors and lateral glances, which are linked to periodic motion (Mottron et al. 2007) and nonrandom visual (geometric) or multimodal (audiovisual synchrony) structures (Klin et al. 2009); and for shorter visual fixations to and faster disengagement from face images, which are associated with an absence of a mandatory bias for faces (Chawarska et al. 2010).

Psychophysical Studies of Low-Level Perceptual Processing

The lowest levels of cortical visual processing, located in V1, include perception of first-order luminance-defined information, to which autistics show enhanced sensitivity compared to non-autistics, when detecting orientation of gratings. Six-month-old siblings of autistic spectrum children also demonstrate enhanced sensitivity to luminance-defined stimuli. Whereas autistics appear to discriminate high and low spatial frequencies at the same level behaviorally as non-autistics, EEG and fMRI reveal between-group differences in brain activity, mainly during high spatial frequency processing. Visual stimuli that fall within the mid-frequency range may be processed using the same mechanisms as those devoted to the processing of high spatial frequency information, providing EEG correlates for the autistic “local bias” evident in pattern

analysis. At a higher level of integration, crowding is the deleterious influence of nearby contours on visual discrimination of a target, attributed to lateral inhibitory interaction of neurons encoding visual properties of nearby distracters. Diminished crowding effects, for which there is preliminary evidence in autism, may allow enhanced segregation of a target from close distracters, offering an explanation for superiorities in visual search.

Autistics have demonstrated atypicalities in some low-level but integrative tasks (Bertone et al. 2005), but as of now, findings are insufficiently homogeneous to posit a deficit in integration. Second-order texture-generated perception requires an integration of primary V1 and associative brain regions V2/V3. The threshold for discriminating the orientation of static second-order gratings is elevated in autism. Early local “binding” mechanisms of contour integration have been investigated, in a search for the lowest-level indication of a putative deficit in grouping processes. However, most recent studies using this method have shown typical behavioral performance in autism, although electrophysiological investigations of contour integration reveal processing differences between autistics and nonautistics. Colors are processed in a complex stream (V1, V2, V4, V8), as second-order visual information, and careful studies show diminished discrimination performance at this level in autistics compared to nonautistics. Components of motion perception, which involve areas V1, V2, and V5/MT, have been studied thoroughly, based on initial findings of diminished influence of perceived motion (optical flow) on postural control and higher thresholds for global motion coherence. When motion perception is decomposed according to the neural complexity required to perceive the motion, perception of first-order motion is preserved, but second-order motion is diminished. Motion coherence tasks measure the ratio of dots within a dot set that have to move together in order to produce a perception of movement, and this perception specifically requires the middle temporal (MT) area. Most recent studies report no group differences, but the variability of results for

motion coherence does not permit firm conclusions with respect to its typicality in autism or its influence on overt behaviors. Sporadically reported autistic differences may result from atypical feeding of information from lower levels and may not be motion specific. Overall there are multiple sources of evidence for atypical low-level visual perceptual processing in autism, some being associated with enhanced behavioral performance, but low-level causative roles in superiorities in pattern detection and manipulation remain to be specified.

In the auditory modality, multiple types of tasks involving pitch perception are performed at a superior level by autistics, representing one of the most replicated areas of autistic superior performance. Task types have included discriminating and categorizing pitches, mapping tonal intervals in a visual schema, memorizing tone-picture associations, determining the direction of pitch-interval correspondence, detecting a named pitch within a chord, memorizing isolated tones and sequences of tones, and detecting a deviant pitch in a sequence of complex sounds. Absolute pitch is considerably more frequent in autistic than nonautistic individuals and, frequently but not always, is associated with savant musical ability. Superior pitch processing has been observed in autistics with or without intellectual disability (according to various measures) but is more frequently observed in autistics whose measured intelligence is in the normal range and who have a history of speech delay.

There are currently no behavioral task findings involving nonsocial simple or complex sounds and a matched group that demonstrate a deficit in autistics. The predicted auditory parallel with possibly diminished processing of second-order visual information has not been found. Robustness of superior pitch processing in autism is confirmed by event-related potentials, which also indicate enhanced detection of pitch changes, mostly in the form of shorter mismatch negativity, shorter latency, or enhanced amplitude. Enhanced low-level auditory perception has a firmly established relation with frequent giftedness in music as well as with atypical language processing and behaviorally observed speech delay.

Other modalities – tactile, olfactory, and taste – have not yet been sufficiently studied to allow any conclusive report. Multimodal integration, which was prematurely considered to be dysfunctional, results in behaviorally normal performance by autistics for integration of low-level perceptual stimuli. However, autistics' verbal labeling of audiovisual integrative patterns may demonstrate sporadic, variable atypicalities.

Cognitive Studies on Pattern Detection, Construction, and Manipulation

Autistics display enhanced performance in tasks requiring detection of a pre-identified local target embedded in a larger probe. Visual search tasks typically consist of detecting the presence or absence of an item among a series of distractors sharing one or several features with the target. The superiority of autistics in visual search is well replicated, encompassing shorter visual fixations, superior discrimination of targets, as well as parallel processing of a larger number of distractors than controls (Caron et al. 2006). Embedded Figures Task (EFT) consists of detecting a simple figure hidden within a complex one. Here also autistics demonstrate a well-replicated superior performance, mostly in the form of increased speed. Another type of task, using hierarchical stimuli, also reveals increased detection of local targets and diminished bias for processing global aspects of a pattern in autistics. Hierarchical or Navon stimuli are typically large “global” letters, numbers, or geometric shapes, composed of small “local” elements, such as a big H composed of small Bs. The task consists of detecting, naming, or matching a component situated at either global (e.g., H) or local levels (e.g., B). Autistics can show more accurate local target detection than typical individuals, while global-level stimuli reaction times are more affected, compared to those of typical individuals, by incongruent local stimuli. In the auditory modality, with musical hierarchical stimuli, autistics reliably display a typical global advantage but either a superior detection of local changes or a diminished global to local interference.

Pattern construction and manipulation are also autistic strengths, first quantitatively reported in

Wechsler Block Design. In this task, the participant has to reproduce a red and white geometric design with a set of red, white and bicolor blocks. Autistics usually perform one to three SDs above their level of IQ, and they outperform controls both in accuracy and RT. In autism, a Block Design (BD) peak is associated with the presence of speech delay. The BD peak has two proposed sources, enhanced perception (e.g., autistics with BD peak are also superior in visual memory, visual search and discrimination) and diminished mandatory influence of global aspects of the figure to be reproduced on local parts matching and manipulation. While visual “local bias” as a general autistic perceptual superiority has been challenged by meta-analyses of Navon- and BD-type performance, it still characterizes a large subgroup of autistic people across a variety of task conditions.

Superior performance in mental rotation tasks in autistics also suggests enhanced construction and manipulation of mental images, which in turn may be attributed to an overall superiority in perceptual performance and to a particular strength in *veridical mapping*, the ability to efficiently detect isomorphisms among entities and make correspondence between these isomorphic features. Autistics also display an enhanced perceptual capacity in visual and auditory modalities.

Domain-Specific Perception: Biological Motion, Faces, and Language

Biological motion tasks consist in recognizing typical human or animal activity from point-light displays. Although the verbal labeling of the motion may be less salient or less automatically produced by autistics, a large fraction of studies have found typical perceptual performance at this level. An fMRI study reporting both similarities and differences in task performance suggests in autistics a diminished involvement of the superior temporal sulcus (STS), an associative region considered central for perception of biological motion in nonautistics.

Atypical overt behaviors toward human faces are diagnostically important in autism. Face processing has therefore been the focus of intense research interest in the past 40 years, yet results

are surprisingly tenuous. Following early experiments suggesting a domain-general local bias, and based on perceptual theories of autism, research on face perception in autism has often focused on the processing of holistic versus local properties of face images. Empirical strategies have included comparison of impact of low- versus high-pass filtering on face image processing, inversion effects including the Thatcher illusion, composite face effects, natural versus nonnatural segmentation of face images, and effects of face context on face part recognition. No clear autistic deficit in the processing of holistic aspects of face images has been found; for example, autistics can display typical face inversion effects, even if not under all experimental conditions. In contrast, there are indications of superior use of face parts for further processing of face images, consistent with an amodal and domain-general local orientation. Typical performance may be obtained through an equalization of relative informative values of low and high spatial frequencies (Simmons et al. 2009).

Studies of ERP responses similarly do not overall indicate an autistic deficit in face processing. Differences reported between responses to face and object manipulation are observed in autistics, indicative of diminished specialization or category specificity. In the same direction, a meta-analysis of regions involved in face-processing tasks indicates that despite activity in typical face-processing regions, other regions are active only in autistics. Autistics show, for example, greater activity bilaterally in extrastriate (BA 18, 19) and striate (BA 17) cortex compared to nonautistics when processing face images (Samson et al. 2012).

In sum, it cannot be stated, as was routinely written in the previous decade, that autistics are characterized by a face-processing *deficit* in that their performance in perceptual tasks involving face images has been comparable to that of nonautistics in most of the tasks used. The ability to use specific parts for face recognition cannot accurately be reduced to a simple deficit; instead, autistics are less strategic and more versatile when scanning faces for whatever purpose. Face processing in autistics seems to rely on a large network of occipital and temporal areas

specifically responsive to certain visual categories in nonautistics.

Atypicalities in speech development and behaviors when exposed to vocalizations are key diagnostic features of a large fraction of the autistic spectrum, such that one could expect perceptual processing of speech to be precociously impaired. The most reliable finding on the relation between auditory perception and speech processing is the coexistence of speech delay in infancy and superior pitch processing at an adult age, albeit the latter is not predictive of speech level at an adult age. Behaviorally, discrimination of the physical aspect of speech (pitch contour) may be superior in autistics, whereas nonautistics cannot attend to a physical property of an auditory pattern without being distracted by its linguistic dimension (Jarvinen-Pasley et al. 2008). A perceptual component may be implicated in the reported difficulty in speech recognition in noise or atypicalities in prosody perception. However, it should be interpreted in relation with superior processing of written alphabetical and numerical material, commonly observed in autistic children with a speech delay.

In terms of material-specific cortical specialization, a reduced leftward asymmetry has been reliably observed for speech processing. Discrimination and orientation to spectrally and temporally complex speechlike material results in atypical, generally diminished brain activity for ERP components indicative of attention to sounds. Autistics exhibit diminished activity in nonprimary auditory cortex and increased activity in primary auditory cortex in response to the presentation of temporally, but not of spectrally complex sounds. Greater temporal complexity effects in primary auditory regions sensitive to acoustic features and reduced temporal complexity effects in auditory regions sensitive to more abstract sound features could represent a greater focus toward perceptual aspects of speech sounds in autism.

Relation with Other Elements of the Cognitive Architecture

Perception is a multilevel system that provides information to but also receives information

from multiple other components in the cognitive architecture. Both feedforward and feedback influences involving perception present some atypicalities in autistics. For example, in non-autistics, feedback from pattern formation to low-level perception sometimes produces visual illusions and distortions. In a visual illusion, the judgment on properties of a visuospatial element is altered by its inclusion in a larger visual context. Autistics display less (but still some) susceptibility to some visual illusions when compared to typical individuals. Categorical perception also produces distortions in nonautistics, as feedback influences from learned categories alters the saliency of perceptual properties of a stimulus. Despite similar categorization abilities, autistics' discrimination is less influenced by categorical knowledge.

There are indications that in autism, visual perception plays a superior role in high-level cognitive processes, which are more language-mediated in nonautistics. For example, lateral glances toward faces and prolonged lateral inspections of rotating objects influence the course of directed attention in autistic toddlers. One of the strongest findings in autistic visual perception, robust enough to be valid across tasks, is revealed by a functional imaging meta-analysis of all tasks implicating visually presented material, in which autistics present superior activity across a broad expanse of brain regions involved in visual perception and perceptual expertise. This suggests that perception plays a superior role in complex cognitive operations – encompassing language, problem-solving, reasoning, etc. – with effects on performance that are not necessarily detrimental and are sometimes beneficial (Samson et al. 2012).

Atypicalities in the relation between components of perceptual processing, as well as between perception and other parts of the cognitive architecture, have naturally oriented toward Bayesian and predictive coding re-interpretations of perception autism. While this has produced elegant and concise theoretical constructs, it has not yet enriched our body of empirical results on autistic perception.

Connections and influences between perceptual brain regions and other brain regions have

now been studied through investigations of structural and functional connectivity. Structural studies including the visual cortex reported altered connectivity in the direction of a diminished integrity of white matter fibers in autistics compared to nonautistics. Despite the small number of studies and the variability in the participants' diagnoses and age, these investigations revealed that visual areas are atypically connected mainly to regions in the temporal lobes but also in frontal and parietal lobes as well as thalamus. Task-related functional connectivity studies mainly show results in the same direction, that is, less correlation between the activity of visual areas and other cortical regions in the frontal, parietal, or temporal lobes, during a wide range of tasks.

Conclusion

Atypical autistic perception was one of the key elements of Kanner's seminal description. The early accounts of perception in autism hypothesized either an abnormal and inconsistent distorting of perceptual input or, alternatively, an unremarkable perception, producing a raw, uncoded representation of the world. First accounts of perception-related behaviors concluded that there was a lack of specificity compared to other neurodevelopmental conditions. The role of these behaviors in diagnosis was minimized, as they were merged with other repetitive behaviors in DSM definitions of autism.

Within the last two decades, these views have profoundly changed. There is now a consensus that the autistic visual and auditory systems provide the rest of the brain with qualitatively, and quantitatively, different information than in nonautistics. Superior performances in nonsavant auditory perception have as yet mainly been found through investigations limited to low-level processes. However, wider-ranging studies have revealed that superior performances in visual perception often imply pattern detection and manipulation, indicating that autistic perception does not produce a raw, uncoded representation of the world. In both modalities, peaks in perception can be correlated with speech delay, suggesting a

relation between the two factors. Relations with nonperceptual components of the autistic cognitive architecture are asymmetrical. On the one hand, perception is more autonomous with regard to emotions, expectations, and language-mediated processes. Autistic perception is more veridical and immune to distortions resulting from top-down influences than in typical individuals. On the other hand, superior brain activity in perceptual associative visual areas is evident in a large array of tasks, suggesting that perception plays a superior role in complex, language-loaded, intelligent cognitive operations, without mandatory detrimental effects on performance.

Concerning relations between perception and other areas of atypicality, autistic perception cannot be straightforwardly explained by cognitive models focused on high-level processes: “perceptual alterations are present in ASD, independent of social function” (Behrmann et al. 2006, p. 263). However, non material-specific peculiarities in perception have the potential to explain autistic characteristics in the diagnostic sociocommunicative as well as in the repetitive behaviors and restricted interests domains. This potential remains underexplored, but there is growing recognition of the “great need for further exploration” of fundamental low-level perceptual atypicalities in autism (Belmonte et al. 2004, p. 658).

Future Directions

- The cartography of autistic low-level perceptual processes is far from complete. Still to be sufficiently investigated are the following: in vision, contrast and (to a lesser extent) colors; and in audition, lateral inhibition, inspection time, among others.
- Multiple auditory equivalents of visuospatial peaks have not yet been investigated.
- The aggregation of auditory and visual perceptual peaks in the same individual is yet unknown.
- The biological substrates, at the cellular level, of enhanced discrimination of low-level information (e.g., pitch) are unknown.

- What is the relation between actual or proposed delineations of the autistic phenotype (e.g., having or not a speech delay) and perceptual characteristics?
- What is the relation between “sensory” issues, as mentioned in the DSM 5, and neuroscience accounts of autistic perception?

See Also

- [Enhanced Perceptual Functioning](#)
- [Weak Central Coherence](#)

References and Reading

- Behrmann, M., Thomas, C., & Humphreys, K. (2006). Seeing it differently: Visual processing in autism. *Trends in Cognitive Sciences*, 10(6), 258–264.
- Belmonte, M. K., Cook, E. H., Jr., Anderson, G. M., Rubenstein, J. L., Greenough, W. T., Beckel-Mitchener, A., et al. (2004). Autism as a disorder of neural information processing: Directions for research and targets for therapy. *Molecular Psychiatry*, 9(7), 646–663.
- Bertone, A., Mottron, L., Jelenic, P., & Faubert, J. (2005). Enhanced and diminished visuo-spatial information processing in autism depends on stimulus complexity. *Brain*, 128(Pt 10), 2430–2441.
- Caron, M. J., Mottron, L., Berthiaume, C., & Dawson, M. (2006). Cognitive mechanisms, specificity and neural underpinnings of visuospatial peaks in autism. *Brain*, 129(Pt 7), 1789–1802.
- Chawarska, K., Volkmar, F., & Klin, A. (2010). Limited attentional bias for faces in toddlers with autism spectrum disorders. *Archives of General Psychiatry*, 67(2), 178–185.
- Dunn, W. (2002). *Infant-toddler sensory profile: User's manual*. San Antonio: Therapy skill builders, Psychological Corp.
- Frith, U., & Baron-Cohen, S. (1987). Perception in autistic children. In D. J. Cohen, A. Donnellan, & R. Paul (Eds.), *Handbook of autism and pervasive developmental disorders*. New York: Wiley.
- Frith, U., & Hermelin, B. (1969). The role of visual and motor cues for normal, subnormal and autistic children. *Journal of Child Psychology and Psychiatry*, 10, 153–163.
- Heaton, P., Hermelin, B., & Pring, L. (1998). Autism and pitch processing: A precursor for savant musical ability? *Music Perception*, 15(3), 291–305.
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. Oxford: Pergamon Press.
- Jarvinen-Pasley, A., Wallace, G. L., Ramus, F., Happé, F., & Heaton, P. (2008). Enhanced perceptual processing

- of speech in autism. *Developmental Science*, 11(1), 109–121.
- Kanner, L. (1965). Infantile autism and the schizophrenias. *Behavioral Science*, 10(4), 412–420.
- Klin, A., Lin, D. J., Gorrindo, P., Ramsay, G., & Jones, W. (2009). Two-year-olds with autism orient to non-social contingencies rather than biological motion. *Nature*, 459(7244), 257–261.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36(1), 27–43.
- Mottron, L., & Belleville, S. (1993). A study of perceptual analysis in a high-level autistic subject with exceptional graphic abilities. *Brain and cognition*, 23(2), 279–309.
- Mottron, L., Mineau, S., Martel, G., Bernier, C. S., Berthiaume, C., Dawson, M., et al. (2007). Lateral glances toward moving stimuli among young children with autism: Early regulation of locally oriented perception? *Development and Psychopathology*, 19(1), 23–36.
- Mottron, L., Bouvet, L., Bonnel, A., Samson, F., Burack, J. A., Dawson, M., & Heaton, P. (2013). Veridical mapping in the development of exceptional autistic abilities. *Neuroscience and Biobehavioral Reviews*, 37(2), 209–228.
- Ornitz, E. M. (1973). Childhood autism. A review of the clinical and experimental literature. *California Medicine*, 118(4), 21–47.
- Ostrolenk, A., Forgeot d'Arc, B., Jelenic, P., Samson, F., & Mottron, L. (2017). Hyperlexia: Systematic review, neurocognitive modelling, and outcome. *Neuroscience and Biobehavioral Reviews*, 79, 134–149.
- Palmer, C. J., Lawson, R. P., & Hohwy, J. (2017). Bayesian approaches to autism: Towards volatility, action, and behavior. *Psychological Bulletin*, 143(5), 521–542.
- Plaisted, K., O'Riordan, M., & Baron-Cohen, S. (1998). Enhanced discrimination of novel, highly similar stimuli by adults with autism during a perceptual learning task. *Journal of Child Psychology and Psychiatry*, 39(5), 765–775.
- Remington, A. M., Swettenham, J. G., & Lavie, N. (2012). Lightening the load: Perceptual load impairs visual detection in typical adults but not in autism. *Journal of Abnormal Psychology*, 121(2), 544–551.
- Rogers, S. J., & Ozonoff, S. (2005). Annotation: What do we know about sensory dysfunction in autism? A critical review of the empirical evidence. *Journal of Child Psychology and Psychiatry*, 46(12), 1255–1268.
- Rutter, M. (1966). Behavioural and cognitive characteristics of a series of psychotic children. In L. Wing (Ed.), *Early childhood autism: Clinical, educational and social aspects* (pp. 51–81). Oxford: Pergamon Press.
- Samson, F., Mottron, L., Soulières, I., & Zeffiro, T. A. (2012). Enhanced visual functioning in autism: An ALE meta-analysis. *Human Brain Mapping*, 33(7), 1553–1581.
- Shah, A., & Frith, U. (1983). An islet of ability in autistic children: A research note. *Journal of Child Psychology and Psychiatry*, 24(4), 613–620.
- Simmons, D. R., Robertson, A. E., McKay, L. S., Toal, E., McAleer, P., & Pollick, F. E. (2009). Vision in autism spectrum disorders. *Vision Research*, 49(22), 2705–2739.
- Van de Cruys, S., Evers, K., Van der Hallen, R., Van Eylen, L., Boets, B., de-Wit, L., & Wagemans, J. (2014). Precise minds in uncertain worlds: Predictive coding in autism. *Psychological Review*, 121(4), 649–675.

Perceptual Development

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Definition

The perceptual system recreates the surrounding environment in the brain based on information provided from the senses: vision, hearing, smell, taste, and touch. Therefore, perception provides the *experience* of the environment and is a means to act according to what is occurring in the environment (Berk 2000; Goldstein 2002).

The general perceptual process involves a series of steps that originate from the environment and involve the recognition of a stimulus and subsequent action regarding that stimulus (Goldstein 2002). Perception is not a static process but rather is dynamic and continually changing based on successive actions and perceptions. For instance, an infant's advancing motor development allows the infant to better explore and learn about the world around him/her. The improved perception resulting from advancing motor skills in turn brings about more effective motor activity. Therefore, general motor development and the resulting perceptive experience support each other in providing infants with increasingly advanced motor and perceptual systems (Bertenthal and Clifton 1998).

Likewise, perceptual development is a continually changing process that advances rapidly in the first years of life and involves an interaction

between the environment and innate bodily systems. This entry will review the development of human perception in typically developing infants and discuss what is known about perceptual development in autism.

Historical Background

Knowledge of perceptual development has grown significantly over the centuries. The belief of preformationism abounded during medieval times. This meant that infants and children were considered to be just smaller versions of adults, with the same perceptual capabilities (Berk 2000). Later centuries watched the pendulum sway in the other direction, with respect to child development. The writings of the British philosopher John Locke propelled the belief that human infants were born as a “tabula rasa” or “blank slate.” Experience was deemed to be necessary for perception to occur (Thorne and Henley 2001). For example, Locke believed that a blind man given sight would have to learn to recognize objects visually. Newborn infants were believed to be capable of nothing and required adult teaching to be shaped into a functioning child and adult. Similarly, nineteenth-century psychologists believed that young infants were not able to perceive or make sense of the stimulation received from their environment (Goldstein 2002). Infants were believed to be functionally deaf until they experienced stimulation from the environment.

G. Stanley Hall, an influential psychologist in the early twentieth century, was inspired by Charles Darwin’s work in evolution. He put forth the idea that child development is genetically predetermined and unfolds automatically (Berk 2000). James Mark Baldwin was an American psychologist publishing at approximately the same time and believed that both the child’s innate capabilities and the surrounding environment are crucial to development. Baldwin’s line of thinking can be seen in many modern-day views on perceptual and general development in children.

More accurate ways of measuring infant perception have allowed psychologists to discover that babies are able to perceive input from all

five senses at birth and that these abilities advance rapidly in the first months of life. While perceptual development tends to follow a general trend that is partially dictated by biological capabilities, perceptual development is dependent on appropriate experiences with the environment.

Current Knowledge

Perceptual Development in Typical Children

Overview

In typical development, most perceptual capacities develop rapidly during the first year of life. Those abilities that are not present at birth generally surface during an infant’s first month. Perception serves as an infants’ first knowledge of the world gathered through the senses and provides the basis for cognitive development (Berk 2000). Many researchers posit that an infants’ general development shifts from an early emphasis on perception and understanding sensations in the first year of life to a later focus on cognition (e.g., Mandler 1998). Infant perception of the environment is informative and influential in other aspects of development as well (Hatton et al. 1997). For instance, hearing is clearly very important in the development of language. Touch, vision, and hearing provide a means for humans to interact with one another and are therefore fundamental in emotional and social development.

Additionally, infants are able to combine information from various modalities and sensory systems. This intermodal perception appears to be present from birth as demonstrated by studies examining infants’ ability to combine diverse sensory inputs. For instance, one study demonstrated that newborns preferred to look at a pacifier shape that they had previously sucked versus one they had not, indicating that infants can integrate touch and vision from birth (Meltzoff and Borton 1979). Additionally, by 7 months of age, typically developing infants were able to match emotional facial expressions that they saw on a screen to varying vocal expressions in an adult’s voice (Soken and Pick 1992). This finding suggests that older

infants can integrate relatively advanced input received from vision and hearing senses.

The development of perceptual abilities is a dynamic process that depends on the interaction between the environment and biological capabilities. A baby's desire to explore its environment coupled by neural and central nervous system development, capabilities of the body, and environmental support provides the medium in which typical development occurs (Berk 2000; Goldstein 2002).

Certain aspects of stimulation are particularly important to young infants, without which deviations from typical development can occur. For instance, a number of studies have been conducted on orphans from Romania who were raised in institutions with horrific conditions (Rutter and The English and Romanian Adoptees Study Team 1998). Noticeable differences in outcome were observed in infants who were adopted between 0–6 months, 6–12 months, and 12–24 months of age, with those infants living in the conditions longer demonstrating greater impairment (Rutter and The English and Romanian Adoptees Study Team).

White and Held (1966) describe a study examining the timing of reaching in infants living in institutions who were provided with high, moderate, and low levels of stimulation. They reported that institutionalized infants who received a moderate amount of stimulation in the first months of their lives reached for objects 6 weeks earlier than those given low levels of stimulation. Although babies receiving a high amount of stimulation also reached for objects sooner than those babies given low levels of stimulation, these infants tended to cry and fuss more often than the other two groups. The authors interpreted findings to mean they were perhaps overwhelmed by the amount of stimulation provided in their environment and suggested that appropriate levels of stimulation (not over- or understimulation) are important to development and adjustment in human infants (White and Held 1966). Studies such as this and the studies on Romanian adoptees demonstrate that typical development cannot occur without proper environmental support.

Assessment of Infant Perception

Infant perception is difficult to assess because babies cannot clearly express what they are thinking or perceiving. This can be especially difficult in newborns and young infants when researchers need to wait for infants to enter a “quiet alert” phase, obtain permission from new mothers, and adapt to rapid changes in infant attention (Goldstein 2002). Therefore, researchers have altered their research questions and methodologies to answer the more basic question: Can infants tell the difference between stimulus A and stimulus B (Goldstein 2002).

A number of creative methodologies are used to examine perception in infancy. One procedure uses visual attention and is called “preferential looking.” If infants look longer at a particular stimulus versus another, researchers assume that the infant can tell the difference between the two. If they look at the two stimuli for the same amount of time, then experimenters assume that infants cannot tell the difference (Goldstein 2002).

An additional methodology takes advantage of infants' preference for novel versus familiar stimuli. Using a procedure called “habituation,” a stimulus is shown repeatedly to an infant over a number of trials. When the infant's looking time significantly decreases (because the novel stimulus has become familiar), they are considered “habituated.” Once the infant is habituated, a novel stimulus is introduced, and researchers note if the infant's looking time significantly increases with the novel stimulus (“dishabituation”). If it does, researchers assume that the infant can tell the difference between the old and new stimulus. If not, it is assumed that they cannot tell the difference. A variation on this procedure involves following the infant's sucking patterns on a pacifier. After habituation occurs, a novel stimulus is introduced, and researchers track whether or not the sucking pattern changes with the new stimulus. If it does, researchers again assume that infants can differentiate the two stimuli. If not, it is assumed they cannot. This procedure is often used to assess perceptive abilities in response to other sensations (e.g., hearing and smell).

Vision

Vision is the least well-developed sensation at birth. However, visual acuity significantly improves over the course of the first year of life, beginning at approximately 20/400 acuity at birth and advancing to adult visual acuity (20/20) after 1 year (e.g., Courage and Adams 1990). Although newborns can see objects with high contrast at very close distances, they cannot distinguish objects with low contrast and those at a distance. Poor visual abilities at birth are present because the visual cortex is not fully developed and cones in retina (fovea) have poorly developed receptors (Goldstein 2002).

Visual perception contributes substantially to other aspects of development as well. Infants who have limited visual input (i.e., those infants who are blind or have very poor vision) have been found to be delayed in other areas, including cognition, motor, and language (Hatton et al. 1997). Social development in blind infants is also impacted. Infants who are blind do not make eye contact and show limited insight into other non-verbal cues as well. Compared to sighted infants, blind infants less often initiate social interactions and have difficulty interpreting others' reactions and responding appropriately (Preisler 1991). Emotional expressiveness is also limited in this population (Tröster and Brambring 1992). These factors can impact the parent-child relationship in infants with visual impairment, and increased stimulation combining other senses (e.g., sound and touch) is recommended to address the social needs of blind infants. Research in this area provides further evidence that perceptual abilities have a direct association on other aspects of human development.

Faces and Facial Expression

The lack of contrast in human faces makes it difficult for typical newborns and very young infants (i.e., less than a month of age) to differentiate faces and recognize facial expressions. Studies do show that 2–3-day-old infants can recognize their mother's faces as demonstrated by a tendency for infants to look longer at their mother's face (both in person and on videotape)

versus an unknown woman (e.g., Bushnell et al. 1989). However, follow-up studies reported that it seems to be the mother's hairline that the infants recognize rather than the face itself because when scarves covered the women's hairlines, newborns showed no preference for one versus the other (Pascalis et al. 1995). By 3–4 months, typical infants can see facial expressions because their contrast perception has improved and can discriminate happy faces from angry, surprised, and neutral faces (e.g., LaBarbera et al. 1976). Other studies show these skills emerging at 7–10 months of age (Ludemann 1991).

Infants as young as 2 months scan between the eyes and mouth of a human face (Bronson 1991) and track face-like patterns moving across their visual field farther than they will track other objects (Morton and Johnson 1991). While some researchers interpret this to mean that humans have a built-in capacity for perception of faces, others have argued that infants younger than 2 months cannot do so, and therefore, the skill cannot be innate. It is possible that the high level of face-to-face interaction that occurs between infants and caregivers contributes to the refinement of face perception. Whether it be innate versus environmental, infant sensitivity to the human face paves the road for the earliest social relationships (Berk 2000).

Refinement of Visual Abilities

Movement may be the earliest form of visual attention. Infants prefer stimuli that move across their visual field, and movement helps define boundaries of objects, likely allowing very young infants to distinguish two objects (Kellman 1996). Infants have a preference for biological motion representing the visual phenomenon of moving, animate objects. By 4–6 months, infants prefer to look at biological motion versus random dots on a screen (Fox and McDaniel 1982). Color perception develops in the first few months of life, and by 2–3 months of age, infants experience a wide range of colors (Brown 1990).

Depth perception involves the ability to judge the distance of objects from one another and

ourselves. In order for infants to reach for objects, they must have a sense of depth. Since each retina of the eye captures images in two-dimensional images, a translation is necessary to perceive three-dimensional input. Binocular disparity (when retinal images of an object fall on different points of the two retinas) helps in depth perception and develops in typical infants by 3 months (Birch 1993). Visual cues also help to detect depth: pictorial depth cues such as overlap, relative height, and relative size develop by 7 months (Yonas et al. 1986).

Depth perception has also been assessed using a paradigm called a “visual cliff,” in which a “deep” side of a box is covered with glass. Researchers found that crawling infants do not cross from the “shallow” to the “deep” side, suggesting that they can perceive apparent differences in depth (Gibson and Walk 1960). Those with more experience crawling were less likely to cross the cliff (Bertenthal et al. 1984), suggesting again that experience with the environment is important in developing perceptual abilities. Independent motor movement (such as crawling) is crucial in developing problem-solving skills and promotes a new level of brain organization (Bell and Fox 1996). Specifically, it encourages synchrony across the regions of the cerebral cortex and strengthens neural connections involving vision, motor planning, and understanding of space.

Hearing

By approximately 6 months of age, an infant’s ability to hear is similar to that of an adult. Prior to 6 months, infants can hear high- but not overly low-intensity sounds (Olsho et al. 1988). In terms of sound location, by 8 weeks, infants can locate two objects that are within 27°. By 18 months, they locate objects within 5°, which is close to adult levels of 1° difference detection (Morrongiello 1988).

Preference for Speech

Typical infants prefer complex sounds, such as the human voice, to other pure tones (Bench et al. 1976). Specifically, babies show a preference for human speech that is high pitched, expressive, and rises in tone at the end of words and phrases (Aslin et al. 1998). This type of speech is often

termed “motherese.” Infants also seem to prefer the sound of their mother’s voice to that of a stranger. After a learning trial, 2-day-old infants changed their sucking patterns on a pacifier to hear their mother’s voice over a stranger’s voice (Spence and Decasper 1987). Infants as young as 1 month can identify differences between certain speech sounds (such as “ba” vs. “pa,” Eimas et al. 1971). Infants are also able to classify vowels regardless of whether the speaker is a male or a female, which demonstrates “equivalence classification” (Kuhl 1983). This is important because when babies begin to make their own sounds, their voices will be much higher pitched than adults, yet they still need to know that their sound production is equivalent. Infants are also able to obtain information about emotions through the human voice. They are able to distinguish happy versus sad-sounding voices by 3 months (Walker-Andrews and Grolnick 1983).

Speech perception is also influenced by experience. At birth, infants are able to distinguish all speech sounds within all languages. Over the course of the first year of life, they begin to specialize in their native language and are then only able to distinguish sounds that are in their native language. For instance, young Japanese infants can distinguish the English/r/ versus/l/ but cannot at 1 year old (e.g., Kuhl et al. 1992).

Smell and Taste

Smell and taste are the most highly developed senses at birth. Young infants show a disgusted facial expression to the smell of rotten eggs and shrimp and a happy expression to the smell of bananas and vanilla (Steiner 1979). Nursing infants can distinguish the smell of their mother’s breast from that of another nursing mother. In one study, infants spent more time turning toward their own mother’s breast pads than another woman’s pad (Marlier et al. 1998). Newborn babies can discriminate sweet-, sour-, and bitter-tasting stimuli (Steiner 1979). The perception of salty tastes comes a few months later, perhaps in preparation for infants to eat solid foods (Beauchamp et al. 1994). These findings suggest that infants can perceive differences in smell and taste at very early ages.

Touch

Sensitivity to touch is also well developed at birth. Newborns respond to touch, especially around the mouth, palms, and soles of feet. They also react to changes in temperature as well as pain. Sensitivity to touch makes babies more responsive to their environment. In one study, the soft touch of an experimenter caused infants to smile and become more attentive to the adults' face (Stack and Muir 1992). Once infants are able to grasp and reach for objects themselves, touch becomes the primary means through which they explore the world. For instance, they often mouth new objects, then remove the object, then look carefully at it, and then return it to their mouth. Mouthing declines as infants are able to do more elaborate touching with hands by, for example, turning it over, poking it, and feeling the surface while looking intently (Ruff et al. 1992). The famous developmental psychologist Jean Piaget considered touch to be crucial for early cognitive development.

Perceptual Development in Autism Spectrum Disorder

Overview

As discussed above, typical infants have a seemingly innate preference for human interaction and a perceptual system that is prepared to remember and discriminate human faces and voice from a very early age. However, perception of human faces and voice in young children with autism spectrum disorder (ASD) appears to be different. As such, the majority of studies of perceptual development in ASD have involved visual processing, in particular processing of human faces. Preference and processing of particular sounds has also been well studied in ASD. Touch, smell, and taste are less frequently examined in ASD and will not be reviewed in this entry. However, in general, children with ASD often demonstrate sensory sensitivities in these areas and are frequently sensitive to particular textures, tastes, and smells. At times, children react negatively to certain tastes, textures, and smells, while other children are interested in these sensations and actively seek them out. Therefore, perceptual development of these senses may be different in ASD but has been not extensively studied.

Visual Processing

Many studies of visual perception in ASD have focused on face processing and the derivation of organized wholes from perceptual parts.

Local Versus Global Processing

Local processing involves the tendency to focus on parts of a stimulus instead of an organized whole, while global or holistic processing involves perception of the overall stimulus rather than its parts. Weak central coherence is a related concept and involves a difficulty in integrating components to create an overall picture due to an overfocus on parts rather than whole. Children with ASD tend to exhibit weak central coherence and perform well on tasks that capitalize on a local versus a global focus, such as the block design subtest of the Wechsler Intelligence Scale for Children (e.g., Happe 1999). Additionally, they are successful at identifying hidden figures within larger drawings (e.g., Joliffe and Baron-Cohen 1997). However, a number of studies have reported intact global processing in individuals with ASD (e.g., Ozonoff et al. 1994).

Faces

As mentioned, the perception of faces and memory for faces is impacted in ASD (e.g., Klin et al. 1999). Perception of facial affect also seems to be impaired in ASD (Hobson et al. 1988) as well as the perception of direction of eye gaze (Joliffe and Baron-Cohen 1997).

Different brain responses to familiar versus unfamiliar faces and various emotional expressions are noted in 6–7-month-old infants (Nelson and De Haan 1996). In typical individuals, the right fusiform gyrus is more activated during faces than nonfaces stimuli (e.g., Haxby et al. 1994). However, both behavioral and neuroimaging studies indicate that individuals with ASD demonstrate face-processing impairments (e.g., Klin et al. 1999).

Infants at risk for developing ASD due to the presence of an older sibling with the disorder fail to show different brain responses to faces versus objects compared to infants who are not at risk for ASD (McCleery et al. 2009). Young children with ASD again do not show different brain responses to familiar versus unfamiliar faces (Dawson et al.

2002) or faces versus objects (Webb et al. 2006). Additionally, inconsistent patterns of fusiform gyrus activation have been reported with some researchers reporting that areas of the brain involved in processing objects show increased activation during perception of face stimuli (e.g., Schultz et al. 2000). This difference may continue into later adolescence and adulthood (McPartland et al. 2004). The trait is likely familial as parents of children with ASD also show an atypical brain response to faces (Dawson et al. 2005). These face-processing challenges all occur in spite of a normal visual system and retina in ASD. It is important to note that a number of studies have failed to report face-processing differences in ASD (e.g., Webb et al. 2010).

A tendency for configural/local processing and unusual face perception may be related in ASD. Individuals with ASD tend to process faces in a feature-based fashion rather than a holistic approach (Hobson et al. 1988). For instance, when looking at faces, individuals with ASD attend to different elements of the face, such as the lower mouth, compared to individuals without autism (Joseph and Tanaka 2003). Merin et al. (2007) found that infants at risk for ASD spent more time looking at their mother's mouth versus her eyes compared to a low-risk infant group. Additionally, inverting a face does not impact face processing as much in ASD as it does in typical individuals (e.g., McPartland et al. 2004). This lack of difference between processing upright versus inverted faces provides evidence that individuals with ASD may process the face in parts rather than whole. Behrmann and colleagues (2006) reported that the speed in discriminating novel from familiar faces was related to the ability to identify local versus global elements in stimuli, suggesting that the bias for local processing might negatively impact the ability to process faces.

Visual Attention and Disengagement in Infancy

Prospective studies have followed infants at heightened risk for ASD due to having an older sibling with ASD. These studies have documented difficulty disengaging visual attention and increased attention to nonsocial stimuli in a subgroup of high-risk infants compared to control infants (e.g., Bryson et al. 2007;

Zwaigenbaum et al. 2005). In fact, the smoothness of visual tracking at 12 months (but not 6 months) successfully differentiated infants who eventually developed ASD from those who did not (Zwaigenbaum et al. 2005). Infants who would later develop ASD took longer to disengage from an active stimulus to view another stimulus between the ages of 6 and 12 months. Bryson et al. (2007) indicated that high-risk infants at 6 months demonstrated an unusual visual interest and reactivity to objects and lacked smooth visual tracking. High-risk infants in another study spent more time looking at a toy than their caregivers during free play (Bhat et al. 2010). Overall, these studies suggest that ASD-related difficulties with visual disengagement and/or with nonsocial interest in the environment are present in infancy.

Hearing

Delays and impairments in both verbal and non-verbal (e.g., ► [Gestures](#)) communication are often present in ASD and emerge at approximately 12 months of age. As such, perception of speech has been studied in young children with ASD to better understand how infants and toddlers with ASD process and perceive speech.

As discussed, typical infants prefer "motherese" to other types of sounds and speech tones. The use of motherese is influential in other aspects of development, including babies' attention to speech and general language development. Because of this, the responses of young children and infants at risk for ASD to motherese have been examined. Nadig and colleagues (2007) reported that 6-month-old infants at risk for ASD were more likely to show a preference for adult-directed speech over motherese compared to low-risk infants. Furthermore, those infants who preferred adult-directed speech had lower language scores (Nadig et al.). Preschoolers and older children with ASD continue to demonstrate atypical responses to motherese (e.g., Kuhl et al. 2005). Children with ASD preferred listening to mechanical-sounding auditory signals ("sine-wave analogs" that are acoustically matched to speech) over motherese (Kuhl et al. 2005). This preference predicted lower language ability, more severe ASD symptoms, and abnormal brain response to speech sounds.

Social Motivation Theory

Some autism experts believe that perceptual abnormalities such as abnormal face and speech perception are not primary to ASD but are secondary to a fundamental impairment in social motivation (likely due to genetic risk factors). Called the “social motivation hypothesis,” this theory posits that a primary deficit in social motivation causes infants with ASD to fail to attend to and tag relevant stimuli within a social context (e.g., Dawson et al. 2002).

According to this theory, infants at risk for ASD spend less time attending to and truly engaging with people. They may instead be overly focused on objects (Zwaigenbaum et al. 2005). The resulting decreased social engagement discourages the development of a specialization in the processing of the human face and speech. Because experience drives cortical specialization (Nelson 2001), reduced attention to people, including their faces, gestures, and speech, also results in a failure of specialization and less efficient function of brain regions that mediate social cognition (McPartland et al. 2004).

The abnormal perception of interactive stimuli such as face and speech perception is not attributable to a lack of exposure to people. Parents of infants with ASD interact with their infant just as parents of typically developing infants do. Therefore, despite faces and speech being directed at them, ASD infants may not be attending and learning from that context because it may not be interpreted as relevant to them. Social interaction is crucial for many aspects of development. For instance, Kuhl, Tsao, and Liu (2003) indicate that mere exposure to speech in infancy is not enough to learn a language. For language acquisition to develop normally, it must be experienced within a socially interactive environment. Early intervention administered when the brain is still plastic can help guide brain and behavioral development back toward a normal trajectory (Dawson et al. 2010).

Future Directions

Perceptual development is well understood in typically developing infants. While abnormalities in

perception have been documented in older children with ASD, they only recently have been examined in infants at risk for ASD due to the presence of an older sibling with the diagnosis in the family. Prospective studies examining this population from very early in life have provided insight into the development of the disorder in infancy. Additionally, they provide an understanding of how perceptual abnormalities in ASD develop. The few studies examining perceptual development in ASD in infancy have suggested that high-risk infants indeed show different perceptive abilities compared to control infants. Specifically, high-risk infants have difficulty shifting visual attention and show unusual face processing. Expanding these studies to examine the perception of other sensations in ASD, such as hearing and touch, as well as following high-risk infants into preschool and older childhood will broaden understanding of perceptual development in ASD.

See Also

- [Face Perception](#)
- [Perception](#)
- [Sensorimotor Development](#)
- [Visual Processing](#)

References and Reading

- Aslin, R. N., Jusczyk, P. W., & Pisoni, D. B. (1998). Speech and auditory processing during infancy: Constraints on and precursors to language. In D. Kuhn & R. S. Siegler (Eds.), *Handbook of child psychology* (Cognition, perception, and language) (Vol. 2, 5th ed., pp. 147–198). New York: Wiley.
- Beauchamp, G. K., Cowart, B. J., Mennella, J. A., & Marsh, R. R. (1994). Infant salt taste: Developmental, methodological, and contextual factors. *Developmental Psychobiology*, 27, 353–365.
- Behrmann, M., Avidan, G., Leonard, G. L., Kimchi, R., Luna, B., Humphreys, K., et al. (2006). Configural processing in autism and its relationship to face processing. *Neuropsychologia*, 44(1), 110–129.
- Bell, M. A., & Fox, N. A. (1996). Crawling experience is related to changes in cortical organization during infancy: Evidence from EEG coherence. *Developmental Psychobiology*, 29, 551–561.
- Bench, R. J., Collyer, Y., Mentz, L., & Wilson, I. (1976). Studies in infant behavioural audiometry: I. Neonates. *Audiology*, 15, 85–105.

- Berk, L. (2000). *Child development* (5th ed.). Needham heights: Pearson.
- Bertenthal, B. I., & Clifton, R. K. (1998). Perception and action. In D. Kuhn & R. S. Siegler (Eds.), *Handbook of child psychology* (Cognition, perception, and language) (Vol. 2, pp. 51–102). New York: Wiley.
- Bertenthal, B. I., Campos, J. J., & Barrett, K. (1984). Self-produced locomotion: An organizer of emotional, cognitive, and social development in infancy. In R. Emde & R. Harmon (Eds.), *Continuities and discontinuities in development* (pp. 174–210). New York: Plenum.
- Bhat, A. N., Galloway, J. C., & Landa, R. J. (2010). Social and non-social visual attention patterns and associative learning in infants at risk for autism. *Journal of Child Psychology and Psychiatry*, 51(9), 989–997.
- Birch, E. E. (1993). Stereopsis in infants and its developmental relation to visual acuity. In K. Simons (Ed.), *Early visual development: Normal and abnormal* (pp. 224–236). New York: Oxford University Press.
- Bronson, G. W. (1991). Infant differences in rate of visual encoding. *Child Development*, 62, 44–54.
- Brown, A. M. (1990). Development of visual sensitivity to light and color vision in human infants: A critical review. *Vision Research*, 30, 1159–1188.
- Bryson, S. E., Zwaigenbaum, L., Brian, J., Roberts, W., Szatmari, P., Rombough, V., & McDermott, C. (2007). A prospective case series of high-risk infants who developed autism. *Journal of Autism and Developmental Disorders*, 37, 12–24.
- Bushnell, I. W. R., Sai, F., & Mullin, J. T. (1989). Neonatal recognition of the mother's face. *British Journal of Developmental Psychology*, 7, 3–15.
- Courage, M. L., & Adams, R. J. (1990). Visual acuity assessment from birth to three years using the acuity card procedures: Cross-sectional and longitudinal samples. *Optometry and Vision Science*, 67, 713–718.
- Dawson, G., Carver, L., Meltzoff, A. N., Panagiotides, H., McPartland, J., & Webb, S. J. (2002). Neural correlates of face and object recognition in young children with autism spectrum disorder, developmental delay, and typical development. *Child Development*, 73(3), 700–717.
- Dawson, G., Webb, S. J., Wijsman, E., Schellenberg, G., Estes, A., Munson, J., et al. (2005). Neurocognitive and electrophysiological evidence of altered face processing in parents of children with autism: Implications for a model of abnormal development of social brain circuitry in autism. *Development and Psychopathology*, 17, 679–697.
- Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., et al. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The early start Denver model. *Pediatrics*, 125(1), 17–23.
- Eimas, P. D., Siqueland, E. R., Jusczyk, P., & Vigorito, J. (1971). Speech perception in infants. *Science*, 171, 303–306.
- Fox, N. A., & McDaniel, C. (1982). The perception of biological motion by human infants. *Science*, 218, 486–487.
- Gibson, E. J., & Walk, R. D. (1960). The “visual cliff”. *Scientific American*, 202, 64–71.
- Goldstein, E. B. (2002). *Sensation and perception* (6th ed.). Pacific Grove: Wadsworth Group.
- Happe, F. G. E. (1999). Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Sciences*, 3(6), 216–222.
- Hatton, D. D., Bailey, D. B., Jr., Burchinal, M. R., & Ferrell, K. A. (1997). Developmental growth curves of preschool children with vision impairments. *Child Development*, 68, 788–806.
- Haxby, J. V., Horwitz, B., Ungerleider, L. G., Maisog, J. M., Pietrini, P., & Grady, C. (1994). The functional organization of human extrastriate cortex: A PET-rCBF study of selective attention to faces and locations. *Journal of Neuroscience*, 14, 6336–6353.
- Hobson, R. P., Ouston, J., & Lee, A. (1988). What's in a face? The case of autism. *British Journal of Psychology*, 79, 441–453.
- Jolliffe, T., & Baron-Cohen, S. (1997). Are people with autism and Asperger syndrome faster than normal on the embedded figures test? *Journal of Child Psychology and Psychiatry*, 38, 527–534.
- Joseph, R. M., & Tanaka, J. (2003). Holistic and part-based face recognition in children with autism. *Journal of Child Psychology and Psychiatry*, 44(4), 529–542.
- Kellman, P. J. (1996). The origins of object perception. In W. Epstein & S. Rogers (Eds.), *Handbook of perception and cognition* (pp. 3–48). New York: Academic Press.
- Klin, A., Sparrow, S. S., de Bildt, A., Cicchetti, D. V., Cohen, D. J., & Volkmar, F. R. (1999). A normed study of face recognition in autism and related disorders. *Journal of Autism and Developmental Disorders*, 29(6), 499–508.
- Kuhl, P. K. (1983). Perception of auditory equivalence classes for speech in early infancy. *Infant Behavior & Development*, 6, 263–285.
- Kuhl, P. K., Williams, K. A., Lacerda, F., Stevens, K. N., & Lindblom, B. (1992). Linguistic experience alters phonetic perception in infants by 6 months of age. *Science*, 255, 606–608.
- Kuhl, P., Tsao, F., & Liu, H. (2003). Foreign-language experience in infancy: Effects of short-term exposure and social interaction on phonetic learning. *Proceedings of the National Academy of Sciences of the United States of America*, 100, 9096–9101.
- Kuhl, P. K., Coffey-Corina, S., Padden, D., & Dawson, G. (2005). Links between social and linguistic processing of speech in preschool children with autism: Behavioral and electrophysiological evidence. *Developmental Science*, 8, 1–12.
- LaBarbera, J. D., Izard, C. E., Vietze, P., & Parisi, S. A. (1976). Four- and six-month-old infants' visual responses to joy, anger, and neutral expressions. *Child Development*, 47, 535–538.
- Ludemann, P. M. (1991). Generalized discrimination of positive facial expressions by seven- and ten-month-old infants. *Child Development*, 62, 55–67.

- Mandler, J. M. (1998). Representation. In D. Kuhn & R. S. Siegler (Eds.), *Handbook of child psychology* (Cognition, perception, and language) (Vol. 2, 5th ed., pp. 255–308). New York: Wiley.
- Marlier, L., Schaal, B., & Soussignan, R. (1998). Neonatal responsiveness to the odor of amniotic and lacteal fluids: A test of perinatal chemosensory continuity. *Child Development*, 69, 611–623.
- McCleery, J. P., Akshoomoff, N., Dobkins, K. R., & Carver, L. J. (2009). Atypical face versus object processing and hemispheric asymmetries in 10-month-old Infants at risk for autism. *Biological Psychiatry*, 66, 950–957.
- McPartland, J., Dawson, G., Webb, S., Panagiotides, H., & Carver, L. (2004). Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorders. *Journal of Child Psychiatry and Psychology*, 45, 1235–1245.
- Meltzoff, A. N., & Borton, R. W. (1979). Intermodal matching by human neonates. *Nature*, 282, 403–404.
- Merin, N., Young, G. S., Ozonoff, S., & Rogers, S. J. (2007). Visual fixation patterns during reciprocal social interaction distinguish a subgroup of 6-month-old infants at-risk for autism from comparison infants. *Journal of Autism and Developmental Disorders*, 37, 108–121.
- Morrongiello, B. A. (1988). Infants' localization of sounds along the horizontal axis: Estimates of minimum audible angle. *Developmental Psychology*, 24, 8–13.
- Morton, J., & Johnson, M. H. (1991). CONSPEC and CONLERN: A two-process theory of infant face recognition. *Psychological Review*, 98, 164–181.
- Nadig, A. S., Ozonoff, S., Young, G. S., Rozga, A., Sigman, M., & Rogers, S. J. (2007). A prospective study of response to name in infants at risk for autism. *Archives of Pediatric and Adolescent Medicine*, 161, 378–383.
- Nelson, C. A. (2001). The development and neural bases of face recognition. *Infant and Child Development*, 10, 3–18.
- Nelson, C. A., & De Haan, M. (1996). Neural correlates of infants' visual responsiveness to facial expressions of emotion. *Developmental Psychobiology*, 29, 577–595.
- Olsho, L. W., Koch, E. G., Carter, E. A., Halpin, C. F., & Spetner, N. B. (1988). Pure-tone sensitivity of human infants. *Journal of the Acoustical Society of America*, 84, 1316–1324.
- Ozonoff, S., Strayer, D. L., McMahon, W. M., & Filloux, F. (1994). Executive function abilities in autism and Tourette syndrome: An information processing approach. *Journal of Child Psychology and Psychiatry*, 35(6), 1015–1032.
- Pascalis, O., deSchonen, S., Morton, J., Deruelle, C., & Fabre-Grenet, M. (1995). Mother's face recognition by neonates: A replication and an extension. *Infant Behavior & Development*, 18, 79–85.
- Preisler, G. M. (1991). Early patterns of interaction between blind infants and their sighted mothers. *Child: Care, Health and Development*, 17, 65–90.
- Ruff, H. A., Saltarelli, L. M., Capozzoli, M., & Dubiner, K. (1992). The differentiation of activity in infants' exploration of objects. *Developmental Psychology*, 28, 851–861.
- Rutter, M., & The English and Romanian Adoptees Study Team. (1998). Developmental catch-up, and deficit, following adoption after severe global early privation. *Journal of Child Psychology and Psychiatry*, 39, 465–476.
- Schultz, R. T., Gauthier, I., Klin, A., Fulbright, R. K., Anderson, A. W., Volkmar, F. R., et al. (2000). Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and Asperger syndrome. *Archives of General Psychiatry*, 57, 331–340.
- Soken, H. H., & Pick, A. D. (1992). Intermodal perception of happy and angry expressive behaviors by seven-month-old infants. *Child Development*, 63, 787–795.
- Spence, M. J., & DeCasper, A. J. (1987). Prenatal experience with low-frequency maternal voice sounds influences neonatal perception of maternal voice samples. *Infant Behavior & Development*, 10, 133–142.
- Stack, D. M., & Muir, D. W. (1992). Adult tactile stimulation during face-to-face interactions modulates five-month-olds' affect and attention. *Child Development*, 63, 1509–1525.
- Steiner, J. E. (1979). Human facial expression in response to taste and smell stimulation. In H. W. Reese & L. P. Lipsitt (Eds.), *Advances in child development and behavior* (Vol. 13, pp. 257–295). New York: Academic Press.
- Thorne, B. M., & Henley, T. B. (2001). *Connections in the history and systems of psychology*. Boston: Houghton Mifflin.
- Tröster, H., & Brambring, M. (1992). Early social-emotional development in blind infants. *Child: Care, Health and Development*, 18, 207–227.
- Walker-Andrews, A. S., & Grolnick, W. (1983). Discrimination of vocal expressions by young infants. *Infant Behavior & Development*, 6, 491–498.
- Webb, S. J., Dawson, G., Bernier, R., & Panagiotides, H. (2006). ERP evidence of atypical face processing in young children with autism. *Journal of Autism and Developmental Disorders*, 36, 881–890.
- Webb, S. J., Jones, E. J. H., Merkle, K., Murias, M., Greenson, J., Richards, T., et al. (2010). Response to familiar faces, newly familiar faces, and novel faces as assessed by erps is intact in adults with autism spectrum disorders. *International Journal of Psychophysiology*, 77(2), 106–117.
- White, B., & Held, R. (1966). Plasticity of sensorimotor development in the human infant. In J. F. Ronsenblith & W. Allinsmith (Eds.), *The causes of behavior* (pp. 60–70). Boston: Allyn and Bacon.
- Yonas, A., Grandrud, E. C., Arterberry, M. E., & Hanson, B. L. (1986). Infants' distance perception from linear perspective and texture gradients. *Infant Behavior & Development*, 9, 245–256.
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Neuroscience*, 23, 143–152.

Perceptual Organization Index (POI)

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Synonyms

POI

Definition

The *Perceptual Organization Index* is part of the Performance Scale of the Wechsler cognitive assessment measures, the Wechsler Intelligence Scale for Children, 3rd edition (WISC-III, 1991), and the Wechsler Adult Intelligent Scale, 3rd edition (WAIS-III, 1997). The POI measures visual-spatial organization and visual-motor skills within a time limit. For the WISC-III, the subtests *Picture Completion*, *Picture Concepts*, *Block Design*, and *Object Assembly* load onto the POI. For the WAIS-III, the subtests *Picture Completion*, *Block Design*, and *Matrix Reasoning* load onto the POI.

The WISC-III and WAIS-III are older versions of the Wechsler cognitive assessment measures, and have since been replaced by the WISC-IV (2003) and WAIS-IV (2008). For both the WISC-IV and the WAIS-IV, the Perceptual Organization Index has been renamed the Perceptual Reasoning Index (PRI). The PRI measures fluid reasoning (use of sequential, inductive, and deductive reasoning) and visual processing (the ability to synthesize and process visual stimuli). Unlike the POI, scores on the PRI are less influenced by speed and the ability to work quickly.

Research has shown that performance on the Block Design task, which loads onto the POI for both the WISC-III and the WAIS-III, is often an area of strength for children with ASD.

See Also

► [Psychological Assessment](#)

References and Reading

- Flanagan, D. P., & Kaufman, A. S. (2004). *Essentials of WISC-IV assessment*. New York: Wiley.
- Sattler, J. M. (2002). *Assessment of children: Behavioral and clinical applications* (4th ed.). San Diego: Jerome M. Sattler.
- Wechsler, D. (1991). *Wechsler intelligence scale for children* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (1997). *Wechsler adult intelligence scale* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2003). *Wechsler intelligence scale for children* (4th ed.). San Antonio: Harcourt Assessment.
- Wechsler, D. (2008). *Wechsler adult intelligence scale: Technical and interpretative manual* (4th ed.). San Antonio: The Psychological Corporation.

Perfectionism

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Synonyms

[Compulsiveness](#); [Exactingness](#); [Preciseness](#)

Definition

Perfectionism refers to the setting of unrealistically high standards accompanied by negative self-evaluations when those standards are not met. When these unrealistic goals are not attained, people with perfectionism characteristics engage

in overly negative self-evaluations, anticipation of catastrophic future consequences, and experience difficult emotions. Perfectionism is linked with psychological disorders including anxiety and depression. Perfectionism may occur in individuals with autism spectrum disorder with some frequency. Perfectionistic tendencies may occur due to cognitive inflexibility that is associated with executive dysfunction of the frontal lobe. Difficulty with shifting attention has been found in this population, with an individual repeatedly performing a task or attempting to solve a problem in the same way over and over. Repetitive behaviors of this type may increase when the person with ASD is under stress or is anxious about something, similar to the type of anxiety that occurs in obsessive-compulsive disorder (Spiker et al. 2012).

In the typically developing population, perfectionism has been linked to social anxiety, depression, and obsessive-compulsive disorder. Perfectionism research in the typically developing population has been conducted using multi-dimensional models (Frost et al. 1990; Hewitt and Flett 1991). Frost et al. (1990) proposed that perfectionism is theoretically comprised of four dimensions: (a) excessive concern with mistakes, (b) a self-doubt about one's abilities, (c) overemphasis on parental expectations and evaluations, and (d) an excessive preference for precision, order, and organization. The maladaptive self-evaluative dimensions of perfectionism have been found to be significantly correlated with anxiety disorders in the typically developing population, specifically social anxiety (social phobia), trait anxiety, and worry (Kawamura et al. 2011). When depression was controlled for, the maladaptive self-evaluative component of perfectionism was not associated with OCD, only with social phobia.

Within the ASD population, it has been found that anxiety disorders occur with great frequency. Recently, there has been research investigating the cognitions and attributions of people with ASD who have anxiety and depression (Greenaway and Howlin 2010). Results indicated that children and

youth with ASD exhibited more dysfunctional attitudes and perfectionism than normal controls. The dysfunctional attitudes were significantly associated with obsessive-compulsive symptoms, and cognitive inflexibility along with social impairments was implicated.

See Also

- [Cognitive Behavioral Therapy \(CBT\)](#)
- [Obsessive-Compulsive Disorder \(OCD\)](#)
- [Social Behaviors and Social Impairment](#)

References and Reading

- Frost, R. O., Marten, P., Lahert, C., & Rosenblate, R. (1990). The dimensions of perfectionism. *Cognitive Therapy and Research*, 14(5), 449–468.
- Frost, R. O., Lahart, C. M., & Rosenblate, R. (1991). The development of perfectionism: A study of daughters and their parents. *Cognitive Therapy and Research*, 15(6), 469–489.
- Greenaway, R., & Howlin, P. (2010). Dysfunctional attitudes and perfectionism and their relationship to anxious and depressive symptoms in boys with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40, 1179–1187.
- Hewitt, P. L., & Flett, G. L. (1991). Perfectionism in the self and social contexts: Conceptualization, assessment, and association with psychopathology. *Journal of Personality and Social Psychology*, 60(3), 456–470.
- Kawamura, K. Y., Hunt, S. L., Frost, R. O., & DiBartolo, P. M. (2011). Perfectionism, anxiety, and depression: Are the relationships independent? *Cognitive Therapy and Research*, 25(3), 291–301.
- Spiker, M., Lin, E., Van Dyke, M., & Wood, J. J. (2012). Restricted interests and anxiety in children with autism. *Autism: International Journal of Research and Practice*, 16(3), 306–320.
- Wood, J. J., & Gadow, K. D. (2010). Exploring the nature and function of anxiety in youth with autism spectrum disorders. *Clinical Psychology: Research and Practice*, 17, 281–292.

Performance Objectives

- [Objective](#)

Performance Reviews for Direct Care Staff

- ▶ [Feedback on Provider Work Performance](#)

Peridol

- ▶ [Haloperidol](#)

Period Prevalence

- ▶ [Prevalence](#)

Peripatetic Teacher

- ▶ [Itinerant Teacher](#)

Peripheral Nervous System (PNS)

- ▶ [Autonomic Nervous System](#)

Periventricular Hemorrhage

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Synonyms

[Intraventricular hemorrhage](#)

Definition

Periventricular hemorrhage or intraventricular hemorrhage (PVH-IVH) is a common complication

seen in preterm infants. PVH-IVH occurs in this population because of the metabolically active germinal matrix in preterm infants (before 32 weeks of gestation). This germinal matrix is supplied by a highly vascular and fragile capillary network that is vulnerable to flow and pressure changes. Bleeding in this region may damage the nearby white matter tracts leading to later motor disability and other cognitive deficits.

See Also

- ▶ [Periventricular Leukomalacia](#)

References and Reading

Kliegman, M. (2011). *Nelson textbook of pediatrics* (19th ed.). Philadelphia: Saunders Elsevier. Chapter 93.

Periventricular Leukomalacia

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Synonyms

[PVL](#)

Definition

Periventricular leukomalacia (PVL) is the most common ischemic brain injury in premature infants and occurs in the white matter adjacent to the lateral ventricles, just distal to the regions supplied by the deep branches of the middle cerebral arteries. PVL is diagnosed by the typical findings of echodensities on cerebral ultrasound or signal abnormalities of MRI. PVL is an important finding because of the high association with later development of nonprogressive developmental impairments such as cerebral palsy.

See Also

- ▶ [Intraventricular Hemorrhage](#)
- ▶ [Periventricular Hemorrhage](#)

References and Reading

- Volpe, J. J. (2008). *Neurology of the newborn* (5th ed.). Philadelphia: Saunders Elsevier. Chapter 8.

Perphenazine

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Synonyms

[Trilafon](#)

Definition

Perphenazine is a phenothiazine that continues to be used for the treatment of schizophrenia. Compared to chlorpromazine, perphenazine is a more potent medication which may translate into decreased likelihood of sedation and hypotension. The increased potency may increase the risk of neurological adverse effects.

See Also

- ▶ [Chlorpromazine](#)

References and Reading

- Martin, A., Scahill, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd. Aufl.). New York: Oxford University Press.

Perseveration

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Definition

Perseveration refers to the tendency for behaviors or thoughts to persist independent of social consequences or feedback from the environment. This includes inappropriate maintenance of a current category, repetition of a previous response to a new stimulus, or continued and uninterrupted repetition of a behavior. Perseveration is a deficit in executive functioning and manifests through an inability to shift from one cognitive strategy to another.

See Also

- ▶ [Echolalia](#)
- ▶ [Obsessive Desire for Sameness](#)
- ▶ [Repetitive Behavior](#)
- ▶ [Self-Injurious Behavior](#)
- ▶ [Stereotypic Behavior](#)

References and Reading

- Ozonoff, S. (1998). Assessment and remediation of executive dysfunction in autism and Asperger syndrome. In E. Schopler, G. Mesibov, & L. J. Kuncie (Eds.), *Asperger syndrome or high functioning Autism?* (pp. 263–292). New York: Plenum.
- Sandson, J., & Albert, M. L. (1984). Varieties of perseveration. *Neuropsychologia*, 22(6), 715–732.
- South, M., Ozonoff, S., & McMahon, W. M. (2007). The relationship between executive functioning, central coherence, and repetitive behaviors in the high-functioning autism spectrum. *Autism*, 11, 437–452.

Persistent Toe Walking

- ▶ [Toe Walking](#)

Personal Perspectives on Autism

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Definition

Definitions of self-advocacy vary; it has been described as follows:

An individual's ability to effectively communicate, convey, negotiate or assert his or her own interests, desires, needs and rights. It involves making informed decisions and taking responsibility for those decisions. (Van Reusen et al. 1994 as cited in West et al. 1999)

Also, as a civil rights movement (Williams and Shoultz 1982 as cited in Goodley, D. and Ramcharan, P. 2005).

However, it is currently defined and it is most often referred to by what could be termed as outsiders – those who aim to define and enhance the concept of self-advocacy but who do not consider themselves to be engaged in self-advocacy; definitions are laden with assumptions and values framed within a normalization agenda. Self-advocacy is seen as something that disabled people may engage in to improve their lot and something that may increase their chances of a more normal or self-directed life. Nondisabled individuals do not generally see self-advocacy as a process that they themselves would be engaged in, but they will debate its meanings.

What of Autistic People's Organisations (organisations run and controlled by Autistic people), those who promote self-advocacy from the inside out as it were? It appears that on the whole, the official line is very much in synch with the aforementioned definition. Looking at www.autreat.com, the website of Autism Network International (ANI), they state that “the best advocates for autistic people are autistic people themselves” but also that some autistic people “are too young, or because they do not have adequate communication skills, are not able to advocate

for themselves.” This, to me, highlights the limited definition of self-advocacy that implies or explicitly states that a level of ability as judged by others is inherently needed to engage in self-advocacy activities. This idea is in line with the thinking that claims that self-advocacy is an acquired skill, something that must be taught in a formal way.

I would like to reconsider these definitions of self-advocacy, to include what may be termed more fundamental or more natural forms of expression of needs, wants, and desires. Is the person who finds their environment unpleasant or even intolerable not also engaging in self-advocacy when they stands and moves to leave a room? How about the person who refuses to board a bus to school but cannot explain that the reason behind this is because of bullying or other abusive experiences that they will encounter on the journey or at their destination? I believe that self-advocacy has become narrowed to such an extent that we are moving it into an elite form of expression; we have allowed nondisabled and more specifically here nonautistic people to define what can and what cannot be termed as self-advocacy. A narrowing to rely on thought through intent, high levels of reasoning, and sophisticated communication with high levels of understanding (as judged by outsiders) leaves those within our autistic communities who are not able to reach these standards languishing.

History

The history of self-advocacy as a named concept can be traced back to the 1960s. At that time, a Swedish activist Dr Bengt Nirje helped disabled people to organize a group which was composed of college students and the disabled people themselves who were expected to make their own plans for activities and were encouraged to make their own decisions, even if mistakes were made. Dr Bengt Nirje described this as “akin to any decent revolt” (Nirje, B. 1969 as cited in *Parallels of Time a History of Developmental Disabilities*).

In the 1970s, the first self-advocacy conferences were held, but still the majority voice was

that of nondisabled people. In 1974 though, the then new organization People First held its first conference with nearly 600 self-advocates in attendance, in Oregon, USA.

At this time, those predominantly engaged in formally recognized self-advocacy were those persons labeled as having learning difficulties, people who had been inspired by the civil rights movement and recognized that disabled people were being left behind.

The inclusion of autistic people in the self-advocacy movement at this time was not overt, but there's no doubt that they were there. Autistic self-advocacy organizations as distinct from the wider self-advocacy movement can be traced back to the early 1990s. Autism Network International (ANI), an advocacy organization run by and for autistic people, was formed in 1992 by Jim Sinclair, Kathy Lissner, and Donna Williams. ANI describes itself as “an autistic run self help and advocacy organisation for autistic people” (autreat.com). ANI went on, in 1996, to present the first Autreat, (www.autreat.com), an annual retreat style conference run by and for autistic people.

Autreat and ANI have become the inspiration for other events, such as Autscope (www.autscope.org), a UK-based annual conference also run by and for autistic people.

Events such as Autreat and Autscope and organizations such as ANI have, over the years, inspired many autistic individuals to speak out, either as individuals or by collaborating and joining together to form a larger more powerful group voice.

ARGH, the organization that I belong to and of which I was a cofounder (www.arghighland.co.uk), was formed when a small group of autistic people came together in 2005 to try to change things for ourselves and other autistic people. Principally a lobbying and campaigning group, we see ourselves as an autistic people's organization engaged in collective advocacy, while at the same time calling for more recognition of self-advocacy.

One of the most well-known autistic self-advocacy organizations is the Autistic Self-Advocacy Network (ASAN) (www.autisticadvocacy.org), founded in 2006. ASAN

has chapters across the USA and more recently in Australia. Ari Ne'eman, the president of ASAN, says, “The object of autism advocacy should not be a world without autistic people – it should be a world in which autistic people can enjoy the same rights, opportunities and quality of life as any of our neurotypical peers” (Ne'eman 2010).

Online organization through message boards and lists has been around almost since the Internet began; in 1994, the ANI listserv started, and in 1996, Martijn Dekker set up Independent Living or InLv for short, an online support group for autistic people.

Communication with each other, sharing experiences, and supporting each other, all of these things contribute to the environment that allows self-advocacy to flourish, at all levels.

Current Knowledge

Even though self-advocacy by individuals and the formation of organizations promoting self-advocacy have been established for some time, the idea of autistic people speaking for themselves is still seen as a novelty by many. One prominent autistic activist says, “Self advocacy is fundamentally about true equality, respect and power, and about recognising and changing the current imbalances in all of those things. Real self advocacy will always upset the status quo in some way” (Baggs 2005).

The dominance of nonautistic voices in conversations about us has remained. Nonautistic advocates speak for us, often without engaging with those they claim to represent to gain a mandate before taking their place at the table to speak for us. Self-advocacy is in itself a contradictory concept in that it only exists as a necessity because of societal expectations, discrimination, and prejudice. Nondisabled people do not engage in self-advocacy when they make choices and speak up for their rights; they are just living their lives.

Autistic people, just as other disabled people and people from other minority groups, are disempowered by societal norms that lead us to engage in what has been labeled as self-advocacy.

This label can be used to replace the more empowering and perhaps more threatening (to those around us) descriptions of what we are actually doing. It raises the question: has our fight for equality been manipulated in meaning to suit an ablest agenda? Self-advocacy is promoted as a way to empower people, but sometimes it feels as though the label is more about presenting requests for equality and rights as a more manageable and acceptable concept to nondisabled people. In this way, a power imbalance can be maintained; norms of society allow us to speak within siloed concepts of “specialness,” with special language describing what others take for granted as usual everyday occurrences or actions.

Mark Neary explores the language of the social care system that can make people’s lives, in this case his son, seem less normal in “ten jargon phrases used for my autistic son,” one example he gives:

If I shout and swear, I’m angry about something. If Steven shouts and swears, it is challenging behaviour and new behaviour management plans need to be drawn up. (Neary 2013)

Neary’s ideas neatly sum up the division between action and perception of action from an outsider’s perspective – nondisabled people. Autistic people are exposed to societal norms that many of us find uncomfortable or alien to us from birth. Many of us receive early intervention – this being seen as essential for young autistic people. Although the term interventions is very broad, and they cannot all be judged to be alike, the pressure to conform, to be indistinguishable, can lead us to a lack of sense of self. Many interventions such as ABA are based upon modifying outward behavior by adherence to the therapist’s instructions; in some cases, this will go as far as how and where a child holds their hands and focuses on an insistence that the nonautistic way is the only way to communicate.

It is difficult to see how these approaches are teaching our young people about selfhood, autonomy, and the value of learning when to say “no.” There appears to be little regard to personhood as an autistic individual, and no examination of what happens to self-expression if autistic expression is

stifled. As Simon Baron-Cohen says, “the need to respect how a person thinks and feels, respecting their real nature, rather than simply focussing on whether they can be trained to change their surface behaviour is essential.”

Future Directions

It is surely our duty as caregivers or community members to ask the following question of all those we entrust to guide our autistic loved ones: how are they being safeguarded to ensure that their personhood and future are protected?

Many parents have been allies to autistic organizations from their inception; this is a concern not only for autistic people but for many who recognize the need for their children (of all ages) to be able to speak up and make themselves heard in any way that they can. For some of us as we age, home living choices, such as what we eat, where we sit, and who we communicate with and how, do grow into political action. Whether the self-advocacy label applies to activism and campaigning (for ourselves or others as self-advocacy is not just about us as individuals) or is about our everyday lives, it is as equally important and valid.

Schools, parents, and other caregivers have a pivotal role to play. It is quite natural for very young children to engage in what may be termed as self-advocacy for us: shouting “no,” turning away from food offered on a spoon, for example. This is quite usual and expected behavior, but after a diagnosis of autism and as we grow, particularly for those of us with limited or non-mainstream forms of communication, our actions can start to be labeled challenging behavior. Our attempts to be heard, to make our needs known, to signal danger, or fear, can be misinterpreted and punished because of our struggle to communicate and to be understood by people who are not experienced in our methods of communication. It is often accepted that self-advocacy is all about the person advocating, that they need to be skilled, and that the onus is upon them to make themselves understood. In fact, this is like any

other form of communication, the receiver has to be in tune with what is being communicated, they must understand and be sensitive, and they must anticipate that a person will be making self-advocacy attempts, understanding the individual's communication style, and adapting to active listening is essential to promote that persons well-being.

Misunderstandings when self-advocacy communication is ignored or mistaken for challenging behavior and the consequences: punishments and being ignored (sometimes quite unintentionally) can start at a very early age. It is easy for people to lose the tools needed for self-advocacy; internalized oppression can become a barrier to effective self-advocacy and mental wellness. If this can be recognized and the need to promote healthy choice making and boundaries that are no more restrictive than those placed upon our nonautistic peers, then we can more effectively sow the seeds for self-advocacy at all levels. By promoting early skills across all learning environments and within the home, we can engineer a situation that guards against loss of self-advocacy skills and ensures that self-determination is more easily fostered throughout the life span. Current self-advocacy training often takes place during transition to adulthood rather than being a lifelong learning experience. All children and young people, not just adults and not just disabled people, would benefit from this approach.

Approaches that encourage autistic people to explore their way of being without expectation of nonautistic behavior and the promotion of autistic voices at all levels: from the playground to the workplace, the boardroom, in government, and always in decisions that affect us or our community are overdue. We need to stop promoting self-advocacy as a novelty, an elite concept for the few, and to recognize that it is for everyone.

In modern society, with the closure of long-stay hospitals, the increasing public awareness of autistic people (and disabled people in general) in our communities and as equals, there should be no need to still separate the right to be heard under a label – self-advocacy. However, in reality, we

have to remember that autistic rights and the fight for equality are still in its infancy, and many of us are still not being heard. The concept of self-advocacy is essential, and the label does have its place to act as a reminder to others to listen; it is very much of its time, but while it is still necessary to give special labels to equal rights, we must reflect upon how much progress we are truly making toward equality from the autistic community and the wider disability community points of view.

References and Reading

- Baggs, A. M. (2005). The meaning of self advocacy, the real voice of autism. <http://archive.autistics.org/library/self-advocacy.html>
- Baron-Cohen, S. (2014). Radical behaviourism. What scientific idea is ready for retirement? Edge.org. www.edge.org/response-detail/25473
- Bascom, J., 2012. Loud Hands: Autistic People, Speaking, Washington, DC: Autistic Self Advocacy Network.
- Goodley, D., & Ramcharan, P. (2005). Advocacy, campaigning and people with learning difficulties. In *Learning disability: A life cycle approach to valuing people* (p. 150), Open University Press, McGraw - Hill Education, Maidenhead.
- Ne'eman, A. (2010). The future (and the past) of autism advocacy. *Disability Studies Quarterly, Piece 1*, 30(1).
- Neary, M. (2013). Viewpoint: 10 Jargon phrases use for my autistic son. BBC NEWS. <http://www.bbc.co.uk/news/blogs-ouch-23423541>
- Parallels of Time a History of Developmental Disabilities. mn.gov/mmddc/parallels/index.html. The Minnesota Governor's Council on Developmental Disabilities.
- The Same as You? (2000). Scottish Government. <http://www.scotland.gov.uk/Resource/Doc/159140/0043285.pdf>
- West, L. L., Corbey, S., Boyer-Stephens, A., Jones, B., et al. (1999). Transition and self advocacy. LD online. <http://www.ldonline.org/article/7757/>
www.arghighland.co.uk
www.autisticadvocacy.org
www.autreat.org
www.autscape.org
www.grasp.org

Personality

► Temperament

Personality Disorders

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Synonyms

[Personality structure in autism spectrum disorders](#)

Short Description or Definition

Personality disorders in autism refer to comorbidity (co-occurring psychiatric disorders). In medicine, comorbidity is relatively straightforward because it mostly concerns well-defined disease entities with known causes (e.g., a fractured leg co-occurring with diabetes). In psychiatry, however, disorders are defined as clusters of signs and symptoms (syndromes), with complicated and uncertain causal pathways. As a consequence, psychiatric disorders show a considerable overlap. This makes it difficult to decide whether symptoms have to be ascribed to either disorder A or disorder B (differential diagnostics) or to both A and B (comorbidity). This issue is important for achieving diagnostic clarity and for managing treatment.

This chapter deals with the dilemma posed when signs and symptoms point to a personality disorders (PD) as well as to an autism spectrum disorder (ASD).

Categorization

Autism Spectrum Disorders (ASD)

In the previous version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), pervasive developmental disorders were subtyped by the Autistic Disorder (AD), Asperger's Syndrome (AS), and the Pervasive Developmental Disorder Not Otherwise Specified

(PDD-NOS). In the new DSM version (DSM-5), these subcategories have been replaced by a single category: Autism Spectrum Disorder.

For the purpose of this chapter, it is important to note that whereas DSM-IV required an onset of symptoms before the age 3 years for the autistic disorder, in DSM-5 this restriction is less strict. Symptoms must be present in early childhood but may not become fully manifest until social demands exceed limited capacities, mostly in adolescence. ASD is diagnosed when there are persistent deficits in social communication and social interaction across contexts and restricted, repetitive patterns of behavior, interests, or activities.

Personality Disorders (PD)

In DSM-IV and DSM 5, 10 categorically defined personality disorders are characterized by enduring and pervasive patterns of inner experiences and behavior (manifested in the areas cognition, affectivity, interpersonal functioning and impulse control); the onset of the patterns can be traced back to adolescence or early adulthood (American Psychiatric Association [APA] 2000). The categorical nature of personality disorders in DSM-IV poses clinicians with classification problems when symptoms are subtle or can also be explained (but not necessarily better) by other disorders or comorbid conditions.

Personality Disorder and/or Autism Spectrum Disorder

The relationship between personality disorders and symptom disorders is complex. Different models have been postulated to describe the shared etiological and pathophysiological factors, such as the predisposition/vulnerability model and the complication/scar model. A promising approach is the model which describes the development and co-occurrence of syndrome and personality disorders as psychobiological in scope (Dolan-Sewell et al. 2001). The Cloninger model (see further below) is an example of this conceptualization. The relationship between PDs and ASD is even more complicated because of the developmental nature of both disorders. While the onset of PDs is theoretically in early adulthood, the first manifestation of behavioral

maladaptive patterns occurs in many instances much earlier on in life. In the diagnostic process of distinguishing PD from ASD, untangling the pathways of symptoms along the lifespan, especially in retrospect, can be a daunting task.

Comorbidity is defined by the presence of two or more disorders present at the same time (Angold et al. 1999; Goldsmith 1999). DSM-IV, however, dictates that if a consistent pattern of experiences or behavior can be better ascribed to the expression or consequence of another psychiatric disorder (e.g., ASD), a PD cannot be diagnosed.

A practical answer to the comorbidity question is that if the clinical presentation of ASD is dominated by major symptoms that would be left underreported if only ASD were classified, it is possible to speak of comorbidity without violating the classification rules. So by classifying a comorbid personality disorder, the clinician draws attention to the complexity of the presentation requiring more than the usual ASD treatment.

However, for clinical purposes, it can be worthwhile to assess personality pathology and traits regardless of whether or not a personality disorder is classified. This is much in line with the proposed DSM-5 methodology.

Epidemiology

There is no reliable data on the prevalence of personality disorders in autism. This is partly due to the earlier mentioned classification rules discouraging diagnosing both at the same time. Like in ASD, assessment of PDs with the “golden standard” (semistructured interviews) is time consuming.

Studies that examined personality profiles in adults with ASD with the Temperament and Character Inventory (Cloninger et al. 1993) show that ASD is characterized by high harm avoidance compared with the norm population, pointing to worrying and pessimistic individuals who are tense in unfamiliar situations or with strangers. In addition, patients with ASD had low scores for reward dependence, indicating little sentimentality and social attachment, and little dependence

on approval of others (Anckarsäter et al. 2006; Sizoo et al. 2009; Söderstrom et al. 2002). The clinical significance of the temperament and character patterns found in patients with ASD is that these profiles sketch in a broad outline the nature and problematic quality of interpersonal relationships between them and others. Other studies showed similar patterns using other instruments like the Autism Spectrum Quotient and the Neuroticism Extraversion Openness Personality Inventory Revised (NEO-PI-R) (Austin 2005; Wakabayashi et al. 2006; Strunz et al. 2015).

Murphy et al. found that particular personality traits may also aggregate in the family members of autistic individuals (broad autism phenotype) and furthermore that some of these traits may be a manifestation of the liability to autism (Murphy et al. 2000; Piven et al. 1997).

In summary, little is yet known about the epidemiology of comorbidity patterns with ASD and PD.

Natural History, Prognostic Factors, Outcomes

In general, comorbidity with personality disorders becomes manifest in adolescence. In the current system of DSM 5, ASD often presents with symptoms that also address the criteria for a PD. This may cause confusion, however, when the first symptoms of ASD occur in adulthood. ASD can then be mistaken for a personality disorder, and vice versa, depending on the focus of the clinical assessment. Intelligence is an important prognostic factor in ASD (Seltzer et al. 2004), meaning that children with higher IQs have a better functional outcome in adulthood than those with lower IQs. Although it is possible that this is also the case in ASD comorbid with PD, clinical practice indicates that there are adults with ASD and high IQs whose functioning is severely hampered by comorbid personality traits. The outcome of ASD with PD has, however, not been studied in a systematic way. The clinical evidence suggests that, like in other symptom disorders, the presence of a PD in ASD increases the burden.

Clinical Expression and Pathophysiology

In ASD, impaired information processing in the brain is especially invalidating in complex situations involving social interaction and emotional communication. In addition, the insight in mental processes of others, but also the self, is often limited because of an impaired intuitive understanding of social relationships and contextual conventions. This implies that people with ASD require more time to evaluate (social) situations because cognitive compensation strategies are utilized to understand what people without ASD know without consciously thinking (Frith 2003).

The developing personality is also influenced by environmental factors, like in ASD. In this respect, the presence of ASD contributes to a pertinent environmental factor because impaired communication and difficulty interpreting social contexts lead to negative reactions of others, which in turn lead to avoidance, anxiety, or a lack of flexibility. The adverse experiences with social interaction may accentuate schizoid, avoidant, or dependent traits when looked at from the personality perspective. The impaired information processing and difficulty with correctly evaluating social contexts in ASD make a clear demarcation between reality and fantasy difficult, enhancing schizotypal personality traits (Towbin 2005). Early trauma and insecure attachment are risk factors for developing a borderline personality disorder (Fonagy and Bateman 2008). When a person with autism is exposed to these risk factors, the resulting phenotype can resemble both disorders. The overlap in phenomenology of ASD and borderline personality disorder, both characterized by altered social cognition, has been noted for some time (e.g., Dudas et al. 2017).

On the other hand, extremes in temperament that are distinctive of personality disorders can color the clinical picture of ASD. Social typologies used in ASD like “active but odd” are in part an expression of personality traits.

Evaluation and Differential Diagnosis

In the assessment, a reliable developmental history is mandatory to diagnose or rule out ASD and to

find evidence for causal factors that could contribute toward the development of the personality disorder. An assessment of personality should also be a part of the ASD assessment. Early trauma, insecure attachment (Fonagy and Bateman 2008) in the case of borderline personality disorder and an arrested developmental phase in the case of narcissistic personality disorder (Fernando 1998) are among the descriptive etiological hypotheses.

The assessment can lead to a classification of ASD and/or a PD. However, more informative is a comprehensive descriptive diagnosis in which traits and behavior, neuropsychological and other impairments, and an overview of contributing (causal) factors combine to serve as an explanatory model for the patient, his close relatives, and the clinician.

Treatment

In case of ASD comorbid with a PD, the effect of programs for personality disorders can be seriously reduced if no account is taken of the impairments that accompany ASD. For example, exposure therapy for social anxiety in an avoidant PD will be less effective when the origin of the avoidance is rooted in a fundamental inability to understand the other in social encounters, as in ASD. In fact, it may for some patients even be considered undesirable to change the avoidant behavior in autism as this could, in the worst case, deteriorate functioning by inducing more (disturbing) social stimuli.

In ASD comorbid with borderline personality disorder, clinical in-patient settings can undermine the autonomy of the individual, leading to serious regression with self-harm and aggressive behavior. Here, the clinical dilemma is between insisting that the patient takes her own responsibility (autonomy) while bearing in mind the cognitive and affective impairments resulting from ASD that prevents her from fully taking this self-responsible stance.

There is currently no systematic evidence to guide treatment of ASD comorbid with a PD or with prominent personality traits. Understanding the impairments (and adaptive aspects) of the individual is the best approach to tailor treatment. This

requires a clinician to be familiar with treatment programs for personality disorders and to study the ins and outs of ASD in order to be able to adopt the content or pace of the programs targeting the comorbid PD. Unadjusted PD treatment in patients with ASD probably has a high failure rate, although this had not been systematically studied.

See Also

- [Clinical Assessment](#)
- [Comorbidity](#)
- [Diagnostic Process](#)
- [Personality Inventory for Children](#)
- [Personality, Clinical Assessment](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (DSM IV)* (4th text Rev. ed.). Washington, DC: American Psychiatric Association.
- Anckarsäter, H., Stahlberg, O., Larson, T., Hakansson, C., Jutblad, S. B., Niklasson, L., et al. (2006). The impact of ADHD and autism spectrum disorders on temperament, character, and personality development. *American Journal of Psychiatry*, *163*, 1239–1244.
- Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry*, *40*, 57–87.
- Austin, E. J. (2005). Personality correlates of the broader autism phenotype are assessed by the autism spectrum quotient (AQ). *Personality and Individual Differences*, *38*, 451–460.
- Cloninger, C. R., Svrakic, D. M., & Przybeck, T. R. (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry*, *50*, 975–990.
- Dolan-Sewell, R. T., Krueger, R. F., & Shea, M. T. (2001). Co-occurrence with syndrome disorders. In W. J. Livesley (Ed.), *Handbook of personality disorders*. New York: Guilford.
- Dudas, R. B., Lovejoy, C., Cassidy, S., Allison, C., Smith, P., & Baron-Cohen, S. (2017). The overlap between autistic spectrum conditions and borderline personality disorder. *PLoS One*, *12*(9), e0184447.
- Fernando, J. (1998). The etiology of narcissistic personality disorder. *Psychoanalytic Study of the Child*, *53*, 141–158.
- Fonagy, P., & Bateman, A. (2008). The development of borderline personality disorder – A mentalizing model. *Journal of Personality Disorders*, *22*, 4–21.
- Frith, U. (2003). *Autism: Explaining the enigma* (2nd ed.). Malden: Blackwell.
- Goldsmith, R. J. (1999). Overview of psychiatric comorbidity. Practical and theoretic considerations. *Psychiatric Clinics of North America*, *22*, 331–349, ix.
- Murphy, M., Bolton, P. F., Pickles, A., Fombonne, E., Piven, J., & Rutter, M. (2000). Personality traits of the relatives of autistic probands. *Psychological Medicine*, *30*, 1411–1424.
- Piven, J., Palmer, P., Jacobi, D., Childress, D., & Arndt, S. (1997). Broader autism phenotype: Evidence from a family history study of multiple-incidence autism families. *American Journal of Psychiatry*, *154*, 185–190.
- Seltzer, M. M., Shattuck, P., Abbeduto, L., & Greenberg, J. S. (2004). Trajectory of development in adolescents and adults with autism. *Mental Retardation and Developmental Disabilities Research*, *10*, 234–247.
- Sizoo, B., van den Brink, W., van Gorissen-Eenige, E. M., & van der Gaag, R. J. (2009). Personality characteristics of adults with autism spectrum disorders or attention deficit hyperactivity disorder with and without substance use disorders. *The Journal of Nervous and Mental Disease*, *197*, 450–454.
- Söderstrom, H., Rastam, M., & Gillberg, C. (2002). Temperament and character in adults with Asperger syndrome. *Autism*, *6*, 287–297.
- Strunz, S., Westphal, L., Ritter, K., Heuser, I., Dziobek, I., et al. (2015). Personality pathology of adults with autism spectrum disorder without accompanying intellectual impairment in comparison to adults with personality disorders. *Journal of Autism and Developmental Disorders*, *45*, 4026–4038.
- Towbin, K. E. (2005). Pervasive developmental disorder not otherwise specified. In F. R. Volkmar, P. Rhea, A. Klin, & J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 165–200). Hoboken: Wiley.
- Wakabayashi, A., Baron-Cohen, S., & Wheelwright, S. (2006). Are autistic traits an independent personality dimension? A study of the autism-spectrum quotient (AQ) and the NEO-PI-R. *Personality and Individual Differences*, *41*, 873–883.

Personality Inventory for Children

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Synonyms

PIC

Description

The Personality Inventory for Children, or PIC, is used to assess the emotional, behavioral,

cognitive, and interpersonal adjustment of children from kindergarten to 12th grade (Lachar and Gruber 2007). It is a series of statements that the parent or guardian rates as true or false in relation to their child. The goal of this clinical assessment tool is to learn more about the child and his or her problems in order to recommend clinical treatment (Sattler and Hoge 2006). It is used in a clinical and school setting.

Historical Background

Introduced in 1956 by Robert D. Wilt, the original 600-question booklet has gone through extensive testing and revision. After the first introduction, and extensive reliability testing, the PIC was altered in 1973. Thirteen questions that could be interpreted as offensive were removed (Lachar 1982). The first version of the PIC was officially published in 1977 (Sattler and Hoge 2006). It is currently in its second version, which is referred to as the PIC-2 (Lachar 1982). The normative tables and scores for the PIC-2 were derived from tests done on two samples of 2306 parents of children; Children in these samples were in Kindergarten through 12th grade, resided in 12 states, attended both urban and rural schools, and included 1551 parents of children who had been referred for clinical support (Personality inventory for children 2007). It takes approximately 40 min to an hour for a parent or guardian to complete the PIC-2. It also has a shortened form called the Behavioral Summary (Sattler and Hoge 2006). The Behavioral Summary contains only 96 items and can be completed in 15 min (Sattler and Hoge 2006).

Psychometric Data

The PIC-2 is comprised of 275 true or false questions (Lachar and Gruber 2007). The test-retest reliability rate is 0.82–0.92 with internal consistency rate of 0.81–0.92 (Lachar and Gruber 2007). There are 9 scales, composite scales, and 21 subscales in the PIC. The test can be administered

as a whole, administering only the Behavioral Summary, or administering the subscales that comprise the composite scores. The following scales and subscales, presented with brief descriptions, comprise the PIC-2:

Cognitive Impairment (COG)

Questions on the Cognitive impairment scale assess the child's difficulty in thinking or processing information (Molano 2010). There are three subscales in Cognitive Impairment in the PIC. Each subscale has 13 questions (Herson 2004). The first subscale is called Inadequate Abilities. This is designed to measure how the parent/guardian feels their child understands information in their environment. The second subscale within Cognitive Impairment is Poor Achievement. This is designed to measure how the parent/guardian feels about the child's performance in their school setting (Herson 2004). The third subscale is Developmental Delay. This is designed to measure how the parent/guardian evaluates specific developmental milestones, such as riding a bike or talking (Herson 2004).

Impulsivity and Distractibility (ADH)

Impulsivity and Distractibility has two subscales. The first subscale is Disruptive Behavior. Disruptive Behavior consists of 21 questions about how the child is able to focus on tasks in multiple settings (Herson 2004). The second subscale measures Fearlessness. It contains nine questions that address how the child would respond to dares, challenges, or other possibly dangerous situations (Herson 2004).

Delinquency (DLQ)

Delinquency is the lack of conformity to social norms. Delinquency has the following three subscales: Antisocial Behavior, Dyscontrol, and Non-compliance. Antisocial Behavior has 15 questions regarding how the child behaves in social situations (Herson 2004). Dyscontrol, or regulation of behavior (Grigsby et al. 1992), which has 16 questions, is designed to assess how the parent interprets their child's behavior in social situations (Herson 2004). Noncompliance has 11 questions

that focus on how the child responds to rules and consequences (Herson 2004).

Family Dysfunction (FAM)

Family Dysfunction has the following two subtests: Conflict Among Members and Parent Maladjustment. Conflict Among Members has 15 questions about the relationship of family members and the strategies that they employ to deal with different family situations (Herson 2004). Parent Maladjustment has 10 questions that focus on how parents deal with stress (Herson 2004). For example, there is an item about parent drinking habits on the Parent Maladjustment subscale (Herson 2004).

Reality Distortion (RLT)

Reality Distortion has the following two subscales: Developmental Deviation and Hallucinations and Delusions. Developmental Deviation has 14 questions about how the child perceives everyday dangers (Herson 2004). Hallucinations and Delusions has 15 questions about the child's level of paranoia (Herson 2004).

Somatic Concern (SOM)

Somatic Concern has the following two subtests: Psychosomatic Preoccupation and Muscular Tension and Anxiety. Psychosomatic is a physical ailment caused by mental processes, stress, or unhappiness (Shorter 1992). Psychosomatic Preoccupation is measured by 17 questions about how the child perceives or feels illness (Herson 2004). Muscular Tension and Anxiety has 11 questions about how often the child feels stress-related pain or other physical symptoms of stress (Herson 2004).

Psychological Discomfort (DIS)

Psychological Discomfort is comprised of the following three subtests: Fear and Worry, Depression, and Sleeping Disturbance/Preoccupation with Death. Fear and Worry has 13 questions about how child handles unknown situations (Herson 2004). Depression has 18 questions about how the child expresses emotions (Herson 2004). Sleeping Disturbance/

Preoccupation with Death is measured with 8 questions about how the child sleeps and whether the child evidences any suicidal thoughts or tendencies (Herson 2004).

Social Withdrawal (WDL)

Social Withdrawal has the following two subtests: Social Introversion and Isolation. Social Introversion has 11 questions about the child's shyness and social competence (Herson 2004). Isolation has eight questions regarding the child's social preferences. For example, Isolation addresses whether the child stays in rooms most of the time (Herson 2004).

Social Skill Deficits (SSK)

Social Skill Deficits has the following two subscales: Limited Peer Status and Conflict with Peers. Limited Peer Status has 13 questions about the child's social standing with peers, types of friendships, and number of friends (Herson 2004). Conflict with Peers has 15 questions about how other children react socially to their child (Herson 2004).

Composite Scales

The PIC has the following three composite scales: Externalizing, Internalizing, and Social Adjustment. The Externalizing composite scale focuses on disruptive behavior and inattention. It is measured in the PIC by adding the scores of Impulsivity and Distractibility and Delinquency (Herson 2004). The Internalizing composite scale focuses on negative cognitions, feelings, and emotions. It is computed by adding the scores from Reality Distortion and Somatic Concern (Herson 2004). The Social adjustment composite provides a measure of overall social adaptation. It is computed by adding the Social Withdrawal and Social Skill Deficits (Herson 2004).

Composite scales can be used to look at one aspect of the child's behavior. For example, a researcher could use a selection of subscales to assess one area of a child's abilities (e.g., social adaptation). Researchers often use composite scores when their research does not require all of the information that is collected in the full PIC-2.

Clinical Uses

In the past, clinicians used the PIC to separate children who needed clinical support into the following six groups: Delinquent, Hyperactive, Cerebral Dysfunction, Somatizing, Mental Retardation (now termed intellectual and developmental disability), and Psychotic (Lachar et al. 1982). Cerebral Dysfunction is also known as brain damage. Somatizing is a physical illness due to stress, unhappiness, or other mental processes (Molano 2010). Currently, the PIC-2 is used to obtain a profile of a child's problem behaviors along with indicators for family dysfunction.

The PIC has been used in a wide range of research projects. For example, it has been included in studies of child achievement (Gough 1946), effects of behavioral treatment for children with autism (Sallows et al. 2005), psychosocial effects of siblings of children with Autism and Mental Retardation (Bagenholm and Gilberg 1991), Psychosocial adjustment of refugees (Rohr 1996), and anxiety studies. In Autism research, the PIC-2, along with other diagnostic tests, is administered before behavioral intervention and re-administered after intervention to understand whether behavior therapies have had an impact on social and emotional aspects of a child's behavior. The PIC-2 can also be used to determine eligibility of participants for a research study.

References and Reading

- Bagenholm, A., & Gilberg, C. (1991). Psychosocial effects on siblings of children with autism and mental retardation: A population-based study. *Journal of Intellectual Disability Research*, 35, 291–307. <https://doi.org/10.1111/j.1365-2788.1991.tb00403.x>.
- Gough, H. G. (1946). The relationship of socioeconomic status to personality inventory and achievement test scores. *Journal of Educational Psychology*, 37(9), 527–540. <https://doi.org/10.1037/h0055013>.
- Grigsby, J., Kaye, K., & Robbins, L. (1992). Reliabilities, norms, and factor structure of the behavioral dyscontrol scale. *Perceptual and Motor Skills*, 74, 883–892.
- Herson, M. (2004). *Comprehensive handbook of psychological assessment*. Hoboken: Wiley.
- Lachar, D. (1982). The personality inventory for children. *90th annual meeting of the American Psychological Association*. Lecture conducted from American Psychological Association, Washington, DC.
- Lachar, D., & Gruber, C. P. (2007). Personality inventory for children, 2nd Ed.: PIC-2 (W-375). *WPS*. Retrieved February 14, 2012, from http://portal.wpspublish.com/portal/page?_pageid=53,112601&_dad=portal&_schema=PORTAL
- Lachar, D., & Wirt, R. D. (1981). A data-based analysis of the psychometric performance of the personality inventory for children (PIC): An alternative to the Achenbach review. *Journal of Personality Assessment*, 45(6), 614–616.
- Lachar, D., Butkus, M., & Hryhorczuk, L. (1978). Objective personality assessment of children: An exploratory study of the personality inventory for children (PIC) in a child psychiatric setting. *Journal of Personality Assessment*, 42(5), 529–537.
- Lachar, D., Gdowski, C., & Snyder, D. (1982). Broad-band dimensions of psychopathology: Factor scales for the personality inventory for children. *Journal of Consulting and Clinical Psychology*, 50(5), 634–642.
- Molano, J. (2010). Vitamin D and older adults more than just a bone problem? *American Academy of Neurology*, 72(2), e2–e4.
- Personality Inventory for Children, 2nd Edition. (2007). *Searchable inventory of instruments assessing violent behavior and related constructs in children and adolescents*. Retrieved February 16, 2012, from <http://vinst.umdj.edu/VAID/TestReport.asp?Code=PIC>
- Rohr, M. E. (1996). Identifying adolescent runaways: The predictive utility of the personality inventory for children. *Adolescence*, 31(123), 605–624.
- Sallows, G., Graupner, T., & MacLean, W., Jr. (2005). Intensive behavioral treatment for children with autism: Four-year outcome and predictors. *American Journal of Mental Retardation*, 110(6), 417–438.
- Sattler, J. M., & Hoge, R. D. (2006). Goals of behavioral and clinical assessment. In *Assessment of children: Behavioral, social, and clinical foundations* (5th ed., p. 5). San Diego: J.M. Sattler.
- Shorter, E. (1992). Preface. In *From paralysis to fatigue: A history of psychosomatic illness in the modern era* (p. 4). New York: The Free Press.

Personality Structure in Autism Spectrum Disorders

► Personality Disorders

Personality, Clinical Assessment

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Definition

Clinical assessment is an evaluation of a patient's physical condition and prognosis based on information gathered from behavioral, physical, and laboratory examinations and the patient's medical history (Mosby 2009). Psychologists or physicians typically assess and diagnose autism spectrum disorders. These clinicians generally are trained as clinical or educational psychologists, psychiatrists, behavioral and developmental pediatricians, and neurologists. Specialized speech and language pathologists and occupational therapists also may provide ASD evaluations. Gold standard assessments consist of thorough parent and child interviews about history and current behavior, consider the role of development, and employ standardized measures.

Historical Background

Practice parameters for ASD assessment have been published by the American Academy of Neurology (Filipek et al. 2000), by the American Academy of Child and Adolescent Psychiatry (Volkmar et al. 1999), and by a consensus panel with representation from multiple professions (Filipek et al. 1999). Each describes two levels of screening consisting of routine developmental surveillance that provides services for young children (i.e., pediatricians) and more comprehensive diagnostic assessment by experienced clinicians for children who fail screening. Prior to the issuance of these practice parameters, oral traditions and subjective observations dominated the assessment of ASD.

The publication of two standardized assessment tools – the parent interview known as the Autism Diagnostic Interview-Revised (ADI-R; Lord et al. 1994; Rutter et al. 2003) and the observer rated semi-structured play interview known as the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2000), also resulted in greatly standardized and reliable ASD diagnoses. These measures currently are considered the “gold standard” in ASD diagnosis. They both require specialized training to administer.

Current Knowledge

A thorough clinical assessment of the child with ASD includes multiple steps. The first of these is to review the child's developmental history and the parents' current concerns. This history must include the review of communication, social and behavioral development, and screening for comorbid medical and psychological conditions including anxiety and depression, and attention deficit symptoms. A review of existing records (medical, school, previous testing, and intervention reports) rounds out the perspective provided by the parent interview. In addition to the history, good assessment includes observation of the child. If possible, this observation should be supplemented by teacher reports of child functioning in the less-structured school setting.

As mentioned above, one of the “gold standard” autism measures – the ADI-R – can be used to interview the parent about the development of autism symptoms in their child. ADI-R is a semi-structured interview that takes approximately 3 h to administer. The much briefer Social Communication Questionnaire (Berument et al. 1999) is a parent-report instrument that contains the same questions as are used to make a diagnosis on the ADI-R, but they are presented in yes/no format and parents can complete it on their own.

The other “gold standard” instrument, the ADOS is a semi-structured interactive play-based assessment of symptoms. It consists of four age and language-graded modules containing

social “presses.” Module 1 is used with children who do not consistently use phrase speech, module 2 with those who use phrase speech but are not verbally fluent, module 3 with fluent children, and module 4 with fluent adolescents and adults. Activities used in ADOS modules 1 and 2 are designed to assess joint attention, communication, symbolic play, and atypical behaviors (restricted and repetitive behaviors and sensory interests) and consist of toy, bubble, and pretend play, reading simple books, and participating in common social scripts like a birthday party. Activities used in modules 3 and 4 assess conversation skills, empathy, and insight into social relationships and include reading books, discussing pictures, engaging in pretend play with toys and objects, acting out stories presented in pictures, and discussing one's feelings, social relationships, and school and/or work situation. Participants are rated based on their level of social interaction and social language, taking their chronological age into account.

In addition to the assessment of ASD symptoms, a good clinical evaluation should include measurement of cognitive abilities. This is important because cognitive level often influences the expression of autism symptoms and may be associated with the ability to acquire skills and the individual's potential level of adaptive functioning. Longitudinal studies also suggest that cognitive ability level is the best predictor of long-term outcome for persons with ASD, with those with intellectual disability typically faring worse. Studies also consistently point out that, even in persons with ASD with cognitive abilities in the average range, adaptive functioning tends to lag behind. Recently, it also has been demonstrated that persons with autism and Asperger syndrome perform better on intelligence tests that assess their perceptual reasoning abilities (Raven's Progressive Matrices) versus more language-based tests like the Wechsler Scales. There also are tests that are more appropriate for children with lower mental ages like the Leiter International Performance Scales-Revised (Roid and Miller 1997). For children less than 5 years of age, the Bayley Scales of Infant Development-II (Bayley 1993) and the Mullen Scales of Early Learning (Mullen 1995) can be used.

After IQ, expressive language ability is the most consistent predictor of outcomes. Those who fail to develop spoken language by early childhood (age 5 or 6) tend to remain minimally verbal, although there have been recent advances that challenge this widely held assertion. There are many general language tests that can be used to provide a clearer picture of expressive and receptive language abilities. Two such tests are the Peabody Picture Vocabulary Test (Brownell 2000) and the Clinical Evaluation of Language Fundamentals (Semel et al. 2003).

However, even when children with autism have adequate spoken language, they frequently demonstrate challenges with language pragmatics or social language, which involves:

Using language for different purposes, like greeting, informing, demanding, and requesting.

Changing language according to the needs of a listener and/or the situation like talking differently to a child than to an adult, providing background information to an unfamiliar listener, and following topic and reciprocity norms for conversations (turn taking, appropriate topics, staying on topic).

Individuals with pragmatic language problems may say inappropriate or unrelated things during conversations, tell stories in a disorganized manner, and without meaning to, say things that are blunt or offensive. It is difficult to assess pragmatic language, and not all speech and language pathologists are equipped to do so. There are several measures developed to assess this form of language. They include the Test of Language Competence (TLC; Wiig and Secord 1989), which is administered by a trained professional, and the Children's Communication Checklist-2 (Bishop and Baird 2001), which is a parent, teacher, or other caregiver report questionnaire that assesses aspects of language pragmatics.

Adaptive behavior is the final domain that should be assessed in a comprehensive evaluation. To make a diagnosis of intellectual disability, information about functioning must be considered alongside information about cognitive ability level. Furthermore, knowing about an individual's adaptive functioning is important for setting appropriate treatment and educational goals.

In individuals with ASD, the most widely used measure of adaptive functioning is the Vineland Adaptive Behavior Scales (Sparrow et al. 1984). It assesses functioning in domains of communication, daily living skills, socialization, and motor skills (under age 5). It is a parent or teacher report interview. There are also several questionnaire-based measures of adaptive functioning including the Adaptive Behavior Assessment System (ABAS; Harrison and Oakland 2003).

There are several domains that frequently also are assessed beyond this core diagnostic battery. A neuropsychological assessment may provide greater clarity about the individual's pattern of strengths and challenges in information processing. This can provide valuable information for schools and transition planning. Neuropsychological assessments in autism frequently focus on executive functions, and selective attention, and generally are administered by the examiner. Recently, more investigators and clinicians are using the Behavioral Rating Inventory of Executive Function (BRIEF; Gioia et al. 2000). This parent- or teacher-rated questionnaire measures inhibition, cognitive flexibility, organization, planning, metacognition, emotional control, and initiation. A neuropsychological assessment also may examine whether the individual displays a nonverbal learning disability profile (Rourke 1995). Children with this profile exhibit well-developed rote verbal skills, verbal memory, and auditory learning capabilities, but have problems with psychomotor coordination, math problem solving, and visuospatial organization. Some children with Asperger syndrome are thought to display this set of strengths and challenges.

Academic assessments can provide additional school-relevant information. Children with ASD have many relative academic strengths related to reading decoding and memory for facts (especially those about their circumscribed interests). Others may possess strong math and/or computer skills. Alongside these strengths, however, they frequently display deficits in reading comprehension, expository writing, and mathematics story problems. It takes considerable skill to tear apart this pattern of strengths and challenges. An understanding of such information is

essential for helping affected individuals to achieve their potential.

Finally, as mentioned above, it is important to assess comorbid psychiatric conditions in individuals with ASD. Approximately 30–40% of them display symptoms of anxiety, depression, and behavioral problems. These can be assessed using instruments from the Achenbach System of Empirically Based Assessment (ASEBA; Achenbach 2009) or the Behavioral Assessment System for Children (BASC-2; Kamphaus et al. 1999). The psychosocial and cognitive symptoms found in autism can make it difficult to diagnose psychopathology in individuals with ASD. For example, their restricted and repetitive behaviors may intensify during periods when they are anxious – a symptom of anxiety not found as commonly in the general population.

Although DSM-IV-TR does not permit the diagnosis of attention deficit hyperactivity disorder (ADHD) symptoms in persons with ASD, in reality, attention symptoms are present in about half of them. This precedence rule is likely to change in DSM 5. ADHD symptoms can be assessed using the Conners Scales.

Finally, when assessing autism, it is critical to maintain a developmental perspective as symptoms change with age. For example, what would be considered a normal level of reciprocal conversational ability for a young child would be quite different than that of a teenager. Similarly, normal levels of overactivity also decline with age.

Future Directions

Assessment-related research and information are badly needed in several areas. First, given what is known about the benefits of early intervention, one critical area is improving our ability to diagnose autism very early. Diagnosis should be across multiple settings and leverage the perspectives of multiple types of service providers. At the present time, considerable research dollars are being spent on developing early behavioral and biological markers of ASD.

Second, as mentioned above, it has been demonstrated that persons with autism and Asperger

syndrome perform better on intelligence tests that assess perceptual reasoning abilities (i.e., Raven's Progressive Matrices) versus more language-based abilities that are tapped in more commonly used measures such as the Wechsler Scales. Additional work is needed to better understand this finding and to use results to help design more comprehensive estimates of the strengths in the cognitive abilities of individuals with ASD and intellectual disability who are nonverbal.

Third, there are several areas that warrant the development of new, more comprehensive and ecologically valid assessment instruments. These include pragmatic language, treatment efficacy, cognitive flexibility, and social skills measures. Better measurement of these areas would provide excellent information for treatment planning.

Finally, as in many areas of autism research, we know little about adults with the disorders. Better measures are needed to diagnose autism in adulthood in individuals with higher levels of functioning. The field also currently lacks good and specific measures of functional outcomes for this population.

See Also

► Executive Functions

References and Reading

- Achenbach, T. M. (2009). *The Achenbach system of empirically based assessment (ASEBA): Development, findings, theory, and applications*. Burlington: University of Vermont Research Center for Children, Youth and Families.
- Bayley, N. (1993). *The Bayley scales of infant development* (2nd ed.). San Antonio: Psychological Corporation.
- Berument, S. K., Rutter, M., Lord, C., Pickles, A., & Bailey, A. (1999). Autism screening questionnaire: Diagnostic validity. *The British Journal of Psychiatry*, 175, 444–451.
- Bishop, D. V. M., & Baird, G. (2001). Parent and teacher report of pragmatic aspects of communication: Use of the Children's Communication Checklist in a clinical setting. *Developmental Medicine and Child Neurology*, 43, 809–818.
- Brownell, R. (2000). *Expressive one-word picture vocabulary test*. Novato: Academic Therapy.
- Filipek, P. A., Accardo, P. J., Baranek, G. T., Cook, E. H., Dawson, G., Gordon, B., et al. (1999). The screening and diagnosis of autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 29(6), 439–484.
- Filipek, P. A., Accardo, P. J., Ashwal, S., Baranek, G. T., Cook, E. H., Jr., Dawson, G., et al. (2000). Practice parameter: Screening and diagnosis of autism: Report of the quality standards subcommittee of the American Academy of Neurology and the Child Neurology Society. *Neurology*, 55(4), 468–479.
- Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2000). *Behavior rating inventory of executive function*. Lutz: Psychological Assessment Resources.
- Harrison, P., & Oakland, T. (2003). *Adaptive behavior assessment system* (2nd ed.). San Antonio: Psychological Corporation.
- Kamphaus, R. W., Reynolds, C. R., & Hatcher, N. M. (1999). Treatment planning and evaluation with the BASC: The Behavior Assessment System for Children. In M. E. Maruish (Ed.), *The use of psychological testing for treatment planning and outcomes assessment* (2nd ed., pp. 563–597). Mahwah: Erlbaum.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(5), 659–685.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., DiLavore, P. C., et al. (2000). The autism diagnostic observation schedule-generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30(3), 205–223.
- Mosby. (2009). *Mosby's medical dictionary* (8th ed.). Amsterdam: Mosby, Elsevier.
- Mullen, E. M. (1995). *Mullen scales of early learning*. Circle Pines: American Guidance Service.
- Reynolds, C. R., & Kamphaus, R. W. (2004). *Behavior assessment system for children* (2nd ed.). Circle Pines: American Guidance Service.
- Roid, G., & Miller, L. (1997). *Leiter international test of intelligence – Revised*. Chicago: Stoelting.
- Rourke, B. P. (Ed.). (1995). *Syndrome of nonverbal learning disabilities: Neurodevelopmental manifestations*. New York: Guilford.
- Rutter, M., LeCouteur, A., & Lord, C. (2003). *Autism diagnostic interview – Revised manual*. Los Angeles: Western Psychological Services.
- Semel, E., Wiig, E. H., & Secord, W. A. (2003). *Clinical evaluation of language fundamentals* (4th ed.). San Antonio: Psychological Corporation.
- Sparrow, S., Balla, D., & Cicchetti, D. (1984). *Vineland adaptive behavior scales*. Circle Pines: American Guidance Service.
- Volkmar, F., Cook, E. H., Pomeroy, J., Realmuto, G., & Tanguay, P. (1999). Practice parameters for the assessment and treatment of children, adolescents, and adults with autism and other pervasive developmental disorders. American Academy of Child and Adolescent

Psychiatry Working Group on Quality Issues. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38(12 Suppl), 32S–54S.

Wiig, E., & Secord, W. (1989). *Test of language competence –Expanded edition*. San Antonio: Psychological Corporation.

Person-First Language

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Synonyms

People-first language

Definition

Person-first language is a method of referring to individuals with disabilities, medical conditions, or functional impairments that emphasizes the person over their disability, condition, or impairment. The term “a child with autism” would be consistent with person-first language, whereas the term “an autistic child” would not. Proponents of person-first language assert that individuals with disabilities should not be defined by their disabilities to avoid dehumanization and equating the individual with his or her impairment (Folkins 1992; Research and Training Center on Independent Living 2008; Smart 2001). However, some disability self-advocacy groups do not prefer person-first language when referring to members of their group, as they do not necessarily view their labels as disabilities, characteristics of which to be ashamed, or traits that can be separated from themselves as individuals (Autistic Self-Advocacy Network Australia n.d.; Smart 2001; Streeter 2010).

See Also

- Advocacy
- Disability

References and Reading

- Autistic Self Advocacy Network Australia. (n.d.). *The A word: why use the word autistic?* Author. Retrieved 5 Feb 2011 from http://www.asan-au.org/?page_id=35.
- Folkins, J. (1992). *Resource on person-first language: The language used to describe individuals with disabilities*. Rockville: American Speech-Language-Hearing Association. Retrieved 5 Feb 2011 from http://www.asha.org/publications/journals/submissions/person_first.htm.
- Research and Training Center on Independent Living, University of Kansas. (2008). *Guidelines for reporting and writing about people with disabilities* (7th ed.). Lawrence: Author.
- Smart, J. (2001). *Disability, society, and the individual*. Austin: Pro-Ed.
- Streeter, L. E. (2010, May). The continuing saga of people-first language. *Braille Monitor*, 53. Retrieved 5 Feb 2011 from <http://www.nfb.org/images/nfb/Publications/bm/bm10/bm1005/bm100509.htm>

Perspective Taking

- Social Cognitive Interventions

Pertofrane

- Desipramine

Pertussis and Autism

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Definition

On April 19, 1982, a local NBC affiliate in Washington, DC, aired a 1-h documentary titled *DPT: Vaccine Roulette*. The program featured the stories of several children: all had received the whole-cell pertussis vaccine and all had

subsequently developed epilepsy and mental retardation. Several parents who watched this show – and collectively believed that their children’s developmental delays were caused by pertussis vaccine – formed an advocacy group called “Dissatisfied Parents Together.” By the early 1990s, this group changed its name to the National Vaccine Information Center, one of the most powerful anti-vaccine organizations in the United States.

The head of the National Vaccine Information Center, Barbara Loe Fisher, later wrote a book titled *A Shot in the Dark: Why the P in the DPT Vaccination May be Hazardous to Your Child’s Health*. In that book Fisher wrote, “With the increasing number of vaccinations American babies have been required to use has come increasing numbers of reports of chronic immune and neurological disorders being suffered by older children and young adults including asthma, chronic ear infections, autism, learning disabilities, attention deficit disorder, diabetes, rheumatoid arthritis, multiple sclerosis, chronic fatigue syndrome, lupus, and cancer.” This book gave birth to the notion that vaccines, especially the pertussis vaccine, could cause developmental delays, including autism.

During the next 10 years, several groups of investigators examined children who either had or had not received pertussis vaccine to determine whether there were differences in the incidence of developmental delays or epilepsy in the two groups. All investigators found that the pertussis vaccine did not cause permanent neurological damage. In 2006, a neurologist working at the University of Melbourne in Australia, using genetic tools that had only recently become available, found that many children whose parents had claimed that pertussis vaccine had caused brain damage actually had a neuronal sodium channel transport defect, specifically, an SCN1A mutation. One hundred percent of children with this mutation will develop seizures and developmental delays in the first year of life independent of vaccination. From the standpoint of scientists, the combination of epidemiological and biological studies put an end to the notion that the whole-cell pertussis vaccine, which has not been used in the United States since the mid-1990s, caused permanent harm.

References and Reading

- Ad Hoc Committee for the Child Neurology Consensus Statement on Pertussis Immunization and the Central Nervous System. (1991). Pertussis immunization and the central nervous system. *Annals of Neurology*, 29, 458–460.
- Bedson, S., Armitage, P., Cockburn, W. C., Conybeare, E. T., Cruickshank, R., Downie, A. W., et al. (1956). Vaccination against whooping cough: Report to the Medical Research Council. *British Medical Journal*, 2, 454–462.
- Berkovic, S., Harkin, L., McMahon, J., Pelekanos, J. T., Zuberi, S. M., Wirrell, E. C., et al. (2006). De-novo mutations of the sodium channel gene *SCN1A* in alleged vaccine encephalopathy: A retrospective study. *Lancet Neurology*, 5, 488–492.
- Cherry, J. (1990). Pertussis vaccine encephalopathy: It is time to recognize it as the myth that it is. *Journal of the American Medical Association*, 263, 1679–1680.
- Corsellis, J., Janoti, I., & Marshall, A. (1983). Immunization against whooping cough: A neuropathological review. *Neuropathology and Applied Neurobiology*, 9, 261–270.
- Coulter, H., & Fisher, B. (1991). *A shot in the dark: Why the P in the DPT vaccination may be hazardous to your children’s health*. Garden City Park: Avery Publishing.
- Gale, J., Thapa, P., Wassilak, S., Bobo, J. K., Mendelman, P. M., & Fey, H. M. (1994). Risk of serious acute neurological illness after immunization with diphtheria-tetanus-pertussis vaccine: A population-based case-control study. *Journal of the American Medical Association*, 271, 37–41.
- Golden, G. (1990). Pertussis vaccine and injury to the brain. *The Journal of Pediatrics*, 116, 854–861.
- Griffin, M., Ray, M., Mortimer, E., Fenichel, G. M., & Schaffner, W. (1990). Risk of seizures and encephalopathy after immunization with the diphtheria-tetanus-pertussis vaccine. *Journal of the American Medical Association*, 263, 1641–1645.
- Hellström, B. (1962). Electroencephalographic studies in triple-immunized infants. *British Medical Journal*, 2, 1089–1091.
- Offit, P. (2010). *Deadly choices: How the anti-vaccine movement threatens us all*. New York: Basic Books.
- Pollack, T., & Morris, J. (1983). A 7-year survey of disorders attributed to vaccination in North West Thames Region. *Lancet*, 1, 753–757.
- Shields, W., Nielson, C., Buch, D., Jacobsen, V., Christenson, P., Christenson, B. Z., et al. (1988). Relationship of pertussis immunization to the onset of neurologic disorders: A retrospective epidemiologic study. *The Journal of Pediatrics*, 113, 801–805.
- Walker, A., Jick, H., Perera, D., Knauss, T. A., & Thompson, R. S. (1988). Neurologic events following diphtheria-tetanus-pertussis immunization. *Pediatrics*, 81, 345–349.

Pervasive Developmental Disorder

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Synonyms

[Autism spectrum disorders](#)

Definition

Pervasive developmental disorders are a group of neurodevelopmental conditions and include autistic disorder, Asperger's disorder, childhood disintegrative disorder, and Rett's disorder. These disorders are characterized by some or all of the following: significant social skills deficits, language abnormalities, restricted interests, and repetitive motor mannerisms. Although there is a strong genetic basis for all except Rett's disorder, the precise genetic cause has not been delineated. Typically, the diagnosis of these conditions is made clinically on the basis of characteristic developmental criteria described in the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition Text Revision* (DSM-IV-TR). These diagnoses have a wide range of symptom complexes that are within the scope of the spectrum, and children vary widely in abilities, intelligence, and behavior.

See Also

► [Pervasive Developmental Disorder Not Otherwise Specified](#)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Pervasive Developmental Disorder Not Otherwise Specified

► [Childhood-Onset Pervasive Developmental Disorder](#)

Pervasive Developmental Disorders

► [Childhood-Onset Pervasive Developmental Disorder](#)

Pervasive Developmental Disorders Rating Scale (PDDRS)

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Synonyms

[PDDRS](#)

Description

The PDDRS is a behavior rating scale designed to identify individuals potentially meeting criteria for a pervasive developmental disorder (PDD). It

was developed by Eaves (1990; Eaves and Hooper 1987–1988) and contains 51 items that measure three dimensions: arousal, affect, and cognition. The “arousal” domain contains 22 items dealing with avoidance of others, sensory stimulation, and fascination with objects; “affect” includes 19 items concerning aggression, fear, anxiety, or distorted affect; and “cognition” contains 10 items regarding speech and language, skill development, and savant skills. Raters are requested to answer each item using a five-point Likert scale according to the degree to which the child generally shows the behavior described. The total score is obtained by summing the separate scores for each subscale. Raw scores can be transformed into standard scores and percentile ranks as an indicator of autism symptoms.

Historical Background

The PDDRS was developed in the early 1990s by Ronald Eaves of Auburn University based on his theory that central nervous system processing in all humans, including those with PDD, can be reduced to three internal processes: arousal, affect, and cognition (Eaves 1993). The author attempted to utilize his theory of human behavior to adapt DSM-III-R criteria into a measure for indexing PDD. The integrated theory of human behavior suggests that individuals with PDD, in addition to individuals with other disabilities, experience problems in one or more of these three internal human attributes. As such, the scale attempted to identify PDD through alterations in these attributes. The items were selected based on the developer’s theory, a review of clinic files of individuals with PDD, as well as existing instruments and DSM-III-R criteria for PDD.

Psychometric Data

Published psychometric data regarding the PDDRS from research groups not directly affiliated with the primary author is very limited (Eaves 1993). According to the instrument developer, the PDDRS was initially normed on a

sample of 500 individuals diagnosed with PDD. The authors purported high initial levels of internal consistency (.92 total score) and high test-retest reliability over short intervals of time. Data from the same sample suggested less robust reliability characteristics with varying raters over longer periods of time. Final instrument structure was also reportedly based on a factor analysis within this same sample of 500. Subsequent studies from groups affiliated with the test developer have demonstrated substantial agreement with other measures, including the Autism Behavior Checklist (see Eaves et al. 2000) and the Gilliam Autism Rating Scale (see Eaves et al. 2006). Limited data regarding the ability of the instrument to discriminate autism spectrum disorders from other complex neurodevelopmental disorders is available.

Clinical Uses

The PDDRS is a rating scale administered to caregivers and designed to be a screening instrument for autism. It is not currently in wide use in either clinical or research settings. The author/developer discourages the use of the instrument for eligibility determinations.

See Also

- [Gilliam Autism Rating Scale \(GARS\)](#)
- [Social Communication Questionnaire](#)
- [Social Responsiveness Scale](#)

References and Reading

- Eaves, R. C. (1990). *The factor structure of autistic behavior*. Paper presented at the Annual Alabama Conference on Autism, Birmingham.
- Eaves, R. C. (1993). *The pervasive developmental disorders rating scale*. Opelika: Small World.
- Eaves, R. C. (2003). *The pervasive developmental disorders rating scale*. Opelika: Small World.
- Eaves, R. C., & Awadh, A. M. (1998). The diagnosis and assessment of autistic disorder. In H. B. Vance (Ed.), *Psychological assessment of children* (2nd ed., pp. 385–417). New York: Wiley.

- Eaves, R. C., & Williams, T. O., Jr. (2006). Exploratory and confirmatory factor analyses of the pervasive developmental disorders rating scale for young children with autistic disorder. *Journal of Genetic Psychology*, 167, 65–92.
- Eaves, R. C., Campbell, H. A., & Chambers, D. (2000). Criterion-related and construct validity of the pervasive developmental disorders rating scale and the autism behavior checklist. *Psychology in the Schools*, 37, 311–321.
- Eaves, R. C., Woods-Groves, S., Williams, T. O., Jr., & Fall, A. (2006). Reliability and validity of the pervasive developmental disorders rating scale and the Gilliam autism rating scale. *Education and Training in Developmental Disabilities*, 41(3), 300–309.
- Williams, T. O., Jr., & Eaves, R. C. (2002). The reliability of test scores for the pervasive developmental disorders rating scale. *Psychology in the Schools*, 39, 605–611.
- Williams, T. O., Jr., & Eaves, R. C. (2005). Factor analysis of the pervasive developmental disorders rating scale with teacher ratings of students with autistic disorder. *Psychology in the Schools*, 42, 207–216.

Pervasive Developmental Disorders Screening Test (PDDST)

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Synonyms

[Pervasive Developmental Disorders Screening Test-II \(PDDST-II\)](#)

Description

The PDDST-II is validated for early screening of autistic spectrum disorder (ASD) in children 18 months to 48 months of age based on both positive signs (symptomatic behaviors) and negative signs (delayed milestones) weighed in a manner that numbers of specific signs additively yield a sensitive and specific screen. The PDDST-II is designed around three key principles: First, parents are uniquely positioned to report on both

habitual as well as low-frequency behaviors that may correlate with an ASD, so they are the best starting point for diagnostic identification process; second, parents of a not-yet-diagnosed child will be inexpert in calibrating quality and severity of reported behavior in the same manner as a skilled clinician, so screening must include a clinician-validated final scoring; and third, a screener should aim to minimize false positives both for humane reasons and for diagnostic efficiency.

Historical Background

Development of the PDDST began in 1986 as an effort to create a brief screen for autism that would be helpful in recognizing autism across (1) primary care settings, (2) specialized developmental care settings, and (3) tertiary diagnostic clinics specializing in autism spectrum disorders. It was clear from the outset that the contrast populations in which potential cases would be embedded would be very different in each of these three settings, and therefore, the sensitivity and specificity of screening items would vary depending on the contrast population. The plan was to test a pool of items and examine relative predictive validity in each of the three settings in index and contrast populations. Items with highest predictive validity in each setting would be retained as screener items and assigned to the screener where the item had high predictive validity. Items with high sensitivity but low specificity (but overall lower predictive validity) would be retained as descriptive items.

Data collection began at the Stanford Autism Clinic in 1986, where a pilot screener was completed for all clinically referred cases under age 5. When the author moved to UCSF in 1989, data collection carried on there until approximately 2005. In addition, in the early 1990s, a comparison group of toddlers who had been very low birth weight (<1,000 g) and experienced neonatal intraventricular hemorrhage (VLBW/IVH) were sampled at 2 years of age as a “developmentally at high-risk, but not specifically autism-risk,” contrast group. Ultimately, the stage 1 screening for pediatric clinics was normed on the all clinically autism-referred cases against the VLBW/IVH

cases as it was felt that overall sensitivity of an instrument that detected no-risk children from autism-referred children would lack specificity. The stage 2 screener for developmental clinics was normed within the population of ASD-referred cases with the index sample being confirmed ASD cases and the contrast population being those for whom an ASD had been ruled out. The stage 3 severity screener which aimed to identify more from less severe cases of autism based on these early signs used autism-confirmed cases as the index population and non-autistic disorder ASD as the contrast population.

An initial version of the three-stage PDDST was compiled in 1991. Data analyses reported in the late 1990s and early 2000s reflect work on this initial version (essentially the PDDST-I), and then in 2004, the final data set with revised psychometrics plus the manual, glossary, and supplemental items (those not included in stage 1 or 2 or 3 but highly sensitive) was compiled and published by PsychCorp as the PDDST-II. The manual gives the psychometrics for the final sample of over 1,000 cases, and the glossary is an item by item inventory of the qualities, thresholds, and examples for each item and is intended for the clinician who scores the parent-completed form to use and to validate a parent-reported positive screen – before referring the child on for further assessment.

Psychometric Data

The PDDST-II Kit (consisting of the technical manual, scoring glossary, and three stages of screeners) (in English or Spanish) is available at <http://www.pearsonassessments.com/haiweb/cultures/en-us/productdetail.htm?pid=076-1635-106&mode=summary>.

Depending on the clinical or research application, one or more stages of screening can be used together. Questions on each screener are organized by age at which the sign/item has typically been reported present. Table 1 shows numbers of items, cutoff number, and sensitivity (Se) and specificity (Sp) for each screener stage.

The PDDST-II Stage 1 Primary Care Screener was shown to be very efficient, with few false

positives (<10%), correctly classifying over 90% of cases that had been clinically screened to an autism-specific clinic after extensive telephone screening. The PDDST-II was validated on 977 cases (N of ASD = 519, N of non-ASD = 458), including ASD-confirmed, ASD-referred, and VLBW control cases. Table 2 gives examples of key items for each screening stage. Importantly, unique (high Sp) items were readily identifiable in analyses that contrasted clinical (telephone) screened ASD-positive cases from a sample of at-risk cases (VLBW) Ss. At stage 2, when ASD-assessed positive cases were contrasted with ASD-assessed negative cases, specificity was more difficult to ascertain than sensitivity, as was the case when examining signs within the ASD-positive population for indicators of severity based on early signs. Table 2 shows exemplar items at each screening stage.

The PDDST-II Glossary – When the parent submits a positive PDDST-II screen, the clinician

Pervasive Developmental Disorders Screening Test (PDDST), Table 1 Item # cutoffs, Se and Sp by screening stage in norming sample

Stage	Cutoff	Se	Sp	N
I	5/22	0.92	0.91	577
II	5/18	0.73	0.49	379
III	8/13	0.58	0.60	260

Pervasive Developmental Disorders Screening Test (PDDST), Table 2 Sample items by stage of screener

Stage	Item	
	Se	Sp
<i>Stage I: primary care screener</i>		
Seems bored or uninterested in conversations around him	0.79	1.0
Alert to some sounds but not to others	0.68	1.0
Ignores new toys	0.60	1.0
<i>Stage II: developmental clinic screener</i>		
Cries when left, does not greet upon reunion	0.38	0.87
Not using gestures to communicate	0.47	0.83
Fixates on fingers	0.31	0.87
<i>Stage III: autism versus PDD (severity) screener</i>		
Not interested in peers	0.88	0.26
Uninterested in talking	0.82	0.23

is directed to the PDDST-II glossary which contains specific verbal probes to query the parents’ response to each positive item to validate that the parent has understood the definition, qualities, and threshold at which this item should be considered truly positive. If a positive score remains after clinician validation, a referral for an ASD assessment is supported; see Table 3.

Supplemental Descriptive Items – In addition to items contained on the three screener stages, there are 41 additional items that characterize common parental observations about delays and atypical development in young children with autism. These additional items are not useful in predicting a positive screening score on stage I, II, or III (mainly because they reflect concerns common, but not unique to ASD) but do provide a more full picture of parental concerns and may be useful, especially where time constraints mandate obtaining a concise, highly focused parent-reported developmental history. While low in specificity, the supplemental items also may help identify a differential diagnosis to pursue in further assessment; see Table 4.

Clinical Uses

Insuring Clinical Validity

The key issue in the development of any screening test is to be useful by detecting cases and at-risk

cases readily, but not so readily that there is a high false-positive rate, rendering the screener highly inefficient. The first “cut” in collection of effective screening data is often reliance on the effected individual (or as is the case in autism, the parents). Parents can underreport because of lack of familiarity with child norms or because of fear of the consequences of a positive screen. Likewise, parents may overreport if a positive screen is seen as leading to more clinical attention – and that is deemed a good thing. The second “cut” therefore is validation of the reporter. In screening, it is desirable if the validation can quickly and efficiently be done on the site of screening. The PDDST utilizes a glossary for the administering clinician to check items reported with positive scores by reading a few sentences about the qualities linked to an autism diagnosis (specificity) and needed threshold for the item to reach clinical significance. For each item, the glossary provides verbatim probes the clinician can use to elicit further report that should clarify whether the positive score is a valid one. Likewise, an item the parent reported as negative but observed as potentially positive by the clinician can be further probed using guidance from the glossary.

Pervasive Developmental Disorders Screening Test (PDDST), Table 3 Sample PDDST-II glossary entry

If you talked to your baby in baby talk, was it hard to get him to “talk” back to you?
<i>Qualities:</i> This measures prelinguistic communicative reciprocity, the first component of which is vocal turn-taking. It is like an early “sound check” that tells us the baby can hear, process what he hears, and reproduce it with some accuracy.
<i>Threshold:</i> By the end of the first 6 months, most parents know some sounds that they can sometimes get their baby to repeat back once or more when the baby is “in the mood.”
<i>Probe for:</i>
Babbling of prespeech sounds
Ability of baby to anticipate and wait for a parent response

Pervasive Developmental Disorders Screening Test (PDDST), Table 4 Sample supplemental items (one sample item per age group)

Birth to 6 months
1. Did your baby seem to stare too much at moving objects or moving lights?
6–12 Months
4. Did your baby not show he/she wanted to go “up” with his/her arms – even when he/she was fussing to be picked up?
12–18 Months
5. Did your baby only rarely or never babble?
18–24 Months
16. Did your toddler ever pose or wave his/her fingers or hands when excited?
24–30 Months
24. Would your toddler pull you to a desired object as his/her way of showing you what he wanted?
30–36 Months
38. Did your child seem more of a perfectionist than other children his/her age?

Three Clinical Settings for the Identification of ASDs

The PDDST-II has been developed for use in three different clinical settings where children with ASD are regularly seen because children with ASD will be compared to different samples of other children depending on where they are assessed. *Stage 1 Primary Care Screener* is for use in primary care settings where children with ASD need be distinguished from among all children of the same age, so screen-positive children can be referred to specialized developmental assessment centers. *Stage 2 Developmental Clinic Screener* is for point of entry to developmental services (Child Find, Early Start, special education, or Department of Developmental Services or Public Health) where children with ASD will need to be distinguished from other children at risk for some neurodevelopmental disorder, so screen-positive children can be referred for more extensive (and costly) confirmatory assessment of an ASD. *Stage 3 ASD Severity Screener* is for use in an autism clinic where it may be helpful clinically or for research purposes to predict severity of ASD (e.g., AD vs. PDD, NOS) from early signs.

Referral for Assessment of Other Neurodevelopmental Disorders

If a positive result is *not* obtained on the PDDST-II, the next clinical question is “What instead might be the nature of this child’s difficulties?” Most commonly, children who screen ASD negative on the PDDST-II may have some form of transient developmental delay (especially under 2 years), overall intellectual disability, a receptive-expressive language disorder, an anxiety disorder, a severe attention deficit, or some combination of these. Additionally, very unstructured parenting combined with extreme temperamental variations, coupled with non-specific neurodevelopmental signs, can be very difficult to delineate from an ASD without in-depth assessment. This complexity speaks to the importance of not mistaking a screening test for comprehensive diagnostic assessment.

Children who do not screen positive for an ASD should be referred to another tertiary diagnostic clinic if something other than transient delay is suspected.

See Also

► [Pervasive Developmental Disorder](#)

References and Reading

References Related to the Development of the PDDST-II

- Adrien, J. L., Lenoir, P., Martineau, J., Perrot, A., Hameury, L., Larmande, C., & Sauvage, D. (1993). Blind ratings of early symptoms of autism based upon family home movies. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32(2), 617–626.
- Baird, G., Charman, T., Cox, A., Baron-Cohen, S., Swettenham, J., Wheelwright, S., & Drew, A. (2000). A screening instrument for autism at 18 months of age: A 6-year follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(6), 694–702.
- Baron-Cohen, S., Cox, A., Baird, G., Swettenham, J., Nightingale, N., Morgan, K., & Charman, T. (1996). Psychological markers in the detection of autism in infancy in a large population. *The British Journal of Psychiatry*, 168(2), 158–163.
- Filapek, P. A., Accardo, P. J., Ashwal, S., Barenak, G. T., Cook, E. H., Dawson, G., et al. (2000). Practice parameter: Screening and diagnosis of autism. *Neurology*, 55(2), 468–479.
- Johnson, S., Siddons, F., & Morton, U. (1992). Can autism be predicted on the basis of infant screening tests? *Developmental Medicine and Child Neurology*, 34(4), 316–320.
- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday videotapes. *Journal of Autism and Developmental Disorders*, 24(3), 247–254.
- Stone, W. L., & Hoffman, E. L. (1994). Early recognition of autism: Parental reports versus clinical observation. *Archives of Pediatrics & Adolescent Medicine*, 148(2), 174–179.
- Stone, W., Conrod, E. E., & Ousley, O. Y. (2000). Brief report: Screening tool for autism in 2-year-olds (STAT): development and preliminary data. *Journal of Autism and Developmental Disorders*, 30(6), 607–612.

References to Abstracts Reporting on the Development of the PDDST-II

- Siegel, B. (1991). Developmental trajectory of early signs of autism: Applications for early identification. In *Symposium, "Early diagnosis of Autism."* Autism Society of America.
- Siegel, B. (1993). *Signs of autism in the first two years.* American Academy of Child and Adolescent Psychiatry.
- Siegel, B. (1994). *The Pervasive Developmental Disorders Screening Test (PDDST): Detection of autism and PDD in infants and toddlers.* International Association of Child and Adolescent Psychiatry.
- Siegel, B. (1996a). 'New developments in the early identification and treatment of autism'. Invited workshop. Society for Behavioral and Developmental Pediatrics. (This presentation was subsequently abstracted in *Pediatrics News* and *Family Practice News*, and the PDDST widely disseminated as a result of reprint requests).
- Siegel, B. (1996b). *The pervasive developmental disorders screening test.* San Francisco: Langley Porter Psychiatric Institute, University of California.
- Siegel, B. & Lindblom, R. (1993). *Observational acumen & detection of atypical development in 1-year-olds.* Society for Research in Child Development.
- Siegel, B. & Sheinkopf, S. (1992). *Precursor signs of autism in the first three years of life.* American Academy of Child and Adolescent Psychiatry.

Suggestions and Readings

- Siegel, B. (1996). Atypical ontogeny: Atypical development from a developmental perspective. In N. Cohen, J. H. Beitchman, R. Tannock, & M. Konstantareas (Eds.), *Language, learning and behavior: Emerging perspectives* (pp. 38–58). Cambridge: Cambridge University Press.
- Siegel, B. (2003). *Helping children with autism learn: Treatment approaches for parents and professional* (p. 1996). New York: Oxford University Press.
- Siegel, B. (2004). *Pervasive developmental disorders screening test-II.* San Antonio: Psychological Corporation/Harcourt Assessment.

Pervasive Developmental Disorders Screening Test-II (PDDST-II)

- [Pervasive Developmental Disorders Screening Test \(PDDST\)](#)

PET

- [Positron-Emission Tomography](#)

Pharmacokinetics

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Definition

Pharmacokinetics is the study of the time course of bodily absorption, distribution, metabolism, and elimination of a drug in a living organism. Clinical pharmacokinetics deals with the application of pharmacokinetic principles for safe and effective drug management in human patients.

References and Reading

- DiPiro, J. T., Spruill, W. J., Wade, W. E., Blouin, R. A., & Preuemer, J. M. (2010). *Concepts in clinical pharmacokinetics.* Bethesda: American Society of Health System Pharmacists.
- Pies, R. (2005). *Handbook of essential psychopharmacology.* Washington, DC: American Psychiatric Publishing.

Phenocopies

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Definition

In some cases, particular phenotypes can be attributed directly to a specific known genetic change in an individual or family. When identical or very similar traits result from some other factor, including environmental causes, these traits or the individuals carrying them are called phenocopies.

A condition called “congenital cataracts” serves as a useful example of the difference between a phenotype and a phenocopy. Congenital cataract is a disorder in which children are born with clouded lenses in their eyes. In this condition, mutations in certain genes have been found to result directly in the phenotype-clouded lenses (Churchill and Graw 2011). These forms of congenital cataracts are often inherited. Sometimes, however, the same phenotype can result from an environmental cause that mimics the phenotype caused by the abovementioned specific genetic changes. For example, in utero infection by the Rubella virus can lead to clouded lenses in the absence of the genetic changes that lead to the inherited form of congenital cataracts (Churchill and Graw). The trait of having this form of congenital cataracts caused largely by an environmental factor or the individual with this form of congenital cataracts would be referred to as a “phenocopy.”

Phenocopies must be considered in efforts at gene discovery. For example, in certain types of genetic analyses, the a priori assignment of affected status within a family is an essential prerequisite. Here the presence of a phenocopy may lead to contradictory evidence with regard to the presence or location of a disease-related mutation. With regard to autism, certain factors such as drug exposures, “profound institutional privation,” (Volkmar 2005) or other conditions, such as congenital blindness and severe disorders of receptive

language (Brown et al. 1997; Volkmar 2005), may lead to ASD phenocopies.

See Also

- ▶ DNA
- ▶ Receptive Language Disorders

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (DSM-IV-TR)*. Washington, DC: Author.
- Brown, R., Hobson, R. P., Lee, A., & Stevenson, J. (1997). Are there “Autistic like” features in congenitally blind children? *Journal of Child Psychology and Psychiatry*, 38(6), 693–703.
- Churchill, A., & Graw, J. (2011). Clinical and experimental advances in congenital and paediatric cataracts. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 366(1568), 1234.
- Rutter, M., Andersen Wood, L., et al. (1999). Quasi autistic patterns following severe early global privation. *Journal of Child Psychology and Psychiatry*, 40(4), 537–549.
- Volkmar, F. R. (2005). *Handbook of autism and pervasive developmental disorders: Assessment, interventions, and policy*. Hoboken: Wiley.

Phenomenological Approach

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Definition

The term phenomenology has several different meanings (including an entire body of work within philosophy about ways of knowing the world). In medicine and science, it is more frequently used to refer to an approach in which empirical observation, rather than theory, plays a central role. Within psychiatry and medicine, this

approach has guided recent editions of the Diagnostic and Statistical Manual starting in 1980 with DSM-III. Prior editions of DSM had been strongly based in theory, but this complicated the usefulness of the classification schemes and, paradoxically, inhibited research. With DSM-III and subsequent editions, the emphasis has been less on theory-driven descriptions and much more on observable phenomena.

The emphasis on observable phenomena as opposed to theory has led to a dramatic increase in research. Theories have been developed over time using data gathered from phenomenologically based classification schemes. In reality, boundaries between theory and phenomenology can become blurry. Further, as Andreasen (2006) notes, unfortunately, this approach itself has tended to become somewhat reified with an overemphasis on the descriptions provided in DSM and a lack of attention to the entire context of the patient and context in which problems develop.

See Also

► [DSM-III](#)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th text revision ed.). Washington, DC: APA Press.
- Andreasen, N. C. (2006). DSM and the death of phenomenology in America: An example of unintended consequences. *Schizophrenia Bulletin*, 33(1), 108–112.
- Rutter, M., & Tuma, A. (1988). Diagnosis and classification: Some outstanding issues. In M. Rutter, A. Tuma, et al. (Eds.), *Assessment and diagnosis in child psychopathology* (pp. 437–452). New York: Guilford Press.
- Spitzer, R. L., Endicott, J. E., & Robbins, E. (1978). Research diagnostic criteria. *Archives of General Psychiatry*, 35, 773–782.

Phenomenology

► [Qualitative Versus Quantitative Approaches](#)

Phenothiazine

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Definition

Phenothiazines were the first type of antipsychotic medication introduced to the marketplace. This class of medications includes chlorpromazine (Thorazine), perphenazine (Trilafon), and fluphenazine (Prolixin) to name a few. These medications block dopamine receptors in particular brain areas. The blockade of these dopamine receptors is presumed to be the mechanism of action that reduces the hallucinations and delusions of schizophrenia. The success of these medications in reducing these so-called positive symptoms of schizophrenia had a major impact on mental health care worldwide. For example, the reduction of these disabling symptoms made it possible for individuals with chronic schizophrenia to leave institutions and be treated in outpatient settings.

Despite the major advance of the traditional antipsychotic medications, they are associated with adverse effects. Of particular importance are the neurologic adverse effects such as tremor, dyskinesia (abnormal movements), and dystonia during the acute phase of treatment. In some cases, dystonia can be an urgent adverse effect with neck stiffness and oculogyric symptoms (eyes tend to roll up backward into the eyelid). This is an acute situation and often quite disturbing to the patient. Dystonias, dyskinesia, and oculogyric crisis are treated with anticholinergic medications (► [Anticholinergic](#)). Long-term treatment with the traditional antipsychotics can also be associated with tardive dyskinesia, which is a potentially chronic movement disorder.

See Also

- ▶ [Anticholinergic](#)
- ▶ [Dystonia](#)
- ▶ [Thorazine](#)
- ▶ [Trilafon](#)

References and Reading

Martin, A., Scahil, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.

Philozac

- ▶ [Prozac™ \(Fluoxetine\)](#)

Phobia

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Synonyms

[Simple phobia](#)

Short Description or Definition

The DSM-IV-TR (American Psychiatric Association 2000) defines specific phobia as a “marked and persistent fear that is excessive or unreasonable, cued by the presence of a specific object or situation.” Exposure to the feared

stimulus must immediately provoke an anxiety response, and the individual must experience avoidance or intense distress that significantly interferes with his or her daily functioning.

Categorization

The DSM-IV-TR lists five categories of specific phobia: animal type, natural environment type (e.g., heights, storms, water), blood-injection-injury type, situational type (e.g., airplanes, elevators, enclosed places), and other type.

While the most common phobias in typically developing (TD) children are doctors, dentists, thunderstorms, and the dark (Muris et al. 1998), children with ASD have been found to have more phobias related to situations and to medical procedures (Evans et al. 2005). For example, the most common fears in children with ASD include needles or injections (De Bruin et al. 2007; Leyfer et al. 2006), the dark (De Bruin et al. 2007), and crowds (Leyfer et al. 2006). Fear of loud noises is also far more common in children with ASD than in TD children (Leyfer et al. 2006).

Epidemiology

Rates of specific phobia in the typically developing population are as follows: 5% of children in community samples have specific phobia, while 15–20% of children presenting to anxiety clinics have specific phobia (Gadow et al. 2004; Kessler et al. 2005; Ollendick et al. 2002).

Specific phobia is one of the most prevalent anxiety disorders in children with ASD (De Bruin et al. 2007; Leyfer et al. 2006; Muris et al. 1998; Sukhodolsky et al. 2008), with rates cited from 31% to 63% in school-age children and adolescents with ASD (De Bruin et al. 2007; Gadow et al. 2004; Leyfer et al. 2006; Muris et al. 1998; Sukhodolsky et al. 2008) and closer to 20% in preschool children with ASD (Gadow et al. 2004). Specific phobia also tends to be more severe in children with ASD than in TD children (Gadow et al. 2005). There is much less research focusing on anxiety disorders in adults with ASD, but Hofvander et al. (2009) found that

6% of adults with ASD and with normal intelligence had specific phobia.

Natural History, Prognostic Factors, and Outcomes

There is little research on prognostic factors or outcomes of specific phobia in ASD, but in typically developing populations, it is thought that genes, temperament, and environment contribute to the etiology and prognosis of specific phobia. Children with specific phobia are more likely to have parents with specific phobia, which may be due to both genetic factors and parenting style (Ollendick et al. 2002). For example, parents with phobias may be more likely to set an example of phobic avoidance and/or be overprotective towards their children, which may increase the likelihood of their children developing specific phobia (Ollendick et al. 2002). Temperament also appears to play a role: children who have inhibited temperaments are at elevated risk for developing specific phobia. Those whose inhibited temperaments are stable between about 2 and 7 years develop specific phobia at rates close to 50% (Kagan et al. 1987, 1988). Children with ASD have been found to have more withdrawn and less adaptable temperaments than typically developing children (Bailey et al. 2000; De Pauw et al. 2011), and it has been theorized that this may lead to higher rates of anxiety (Bellini 2006).

Genetic factors seem to be involved in an individual's propensity towards general "fearfulness," but the environment (e.g., a frightening experience) contributes to the specific fear (Ollendick et al. 2002). However, while most adults attribute a specific phobia to a direct experience of fear conditioning, most children attribute specific phobia to observing a fear response in others, to reading or hearing about a phobia, or to no obvious cause (Ollendick et al. 2002).

Clinical Expression and Pathophysiology

Clinical Features. Individuals with specific phobia experience excessive or unreasonable fear of a

particular object or situation. The fear can occur in the presence of the object or in anticipation of encountering it. When exposed to the phobic object or situation, the individual will almost always experience intense anxiety. In children, this may be expressed by crying, tantrums, or freezing. Individuals with specific phobia often avoid the phobic situation. According to the DSM-IV-TR (American Psychiatric Association 2000), adults must recognize that the fear is excessive or unreasonable, but this may not be true for children. This feature therefore may also not be present in individuals with developmental disabilities such as autism or mental retardation. Several typical manifestations of phobia in children are as follows: Children with a phobia of loud noises may cover their eyes, cry, or have a temper tantrum when exposed to sounds like fireworks, sirens, or thunder, and they may also avoid situations with loud noises, for example, refusing to go to school when they know there will be a fire drill. Children with a phobia of animals or insects often experience fear when exposed to the animal. They may check a room for an insect or refuse to go into a room where the insect had previously been seen. Children may avoid outdoor activities such as going to a park or going camping due to their fear of insects. Other common behaviors include tantrumming or running out of the room when receiving an injection (medical fears), insisting on sleeping with a nightlight on and refusing to walk through the house at night (fear of the dark), or walking up multiple flights of stairs to avoid an elevator (elevator phobia).

Relation to Age. TD children generally follow a natural developmental progression of fears, i.e., fears related to situations, their natural environment, and strangers decrease as they get older, while fears of harm and medical fears increase (Evans et al. 2005). Children with ASD do not seem to follow this same developmental progression; unlike for TD children and children with intellectual disability, mental age does not correlate with fears for children with ASD.

Relation to IQ and Severity of ASD. As with other types of anxiety, rates of specific phobia are higher in children with ASD and average IQ than in those with ASD and intellectual disability.

However, even in children with ASD and below average IQ, rates are still much higher than in the general population. Sukhodolsky et al. (2008) found rates of 31% and 37% in children with ASD with and without intellectual disability, respectively. The majority of studies of anxiety in the ASD population have examined individuals with high-functioning ASD or pervasive developmental disorder—not otherwise specified (PDD-NOS). However, those studies that have compared rates of specific phobia in autism and autism spectrum conditions (e.g., PDD-NOS) have found that rates of specific phobia are much higher in PDD-NOS (e.g., Muris et al. 1998). Nonetheless, given that much research has also illustrated the difficulty with accurately differentiating between autistic disorder and PDD-NOS (e.g., Chawarska et al. 2007; Lord et al. 2000), it is unclear how to interpret these findings.

Relation to Other Problems. In children with ASD, specific phobia is associated with conduct problems, impulsivity, hyperactivity, and somatic complaints. In particular, specific phobia is more related to behavioral problems and other types of anxiety in children with ASD than in TD children (Evans et al. 2005). Children with ASD may be more likely to display challenging behaviors such as tantrums rather than communicate their fears verbally due to their deficits in communication skills.

Pathophysiology. There is little research on the pathophysiology of specific phobia in ASD, but there is emerging research linking anxiety to neurological and genetic abnormalities in individuals with ASD. For example, amygdala volume in children with ASD has been found to be positively correlated with anxiety (Amaral et al. 2008; Juranek et al. 2006), and a particular polymorphism (the 10–10 repeat allele) of the dopamine transporter gene (DAT1) has been associated with greater severity of social anxiety in children with ASD (Gadow et al. 2008).

Evaluation and Differential Diagnosis

Evaluation. Diagnostic markers of specific phobia include an intense and irrational fear of a specific

stimulus or situation, an exaggerated reaction to the feared stimulus (e.g., crying, tantruming, running away), and extreme measures to avoid the feared stimulus. Individuals with specific phobia may also display physical symptoms such as shortness of breath, pounding heart, sweating, nausea, or light-headedness. Individuals with blood or injection phobias may feel dizzy or faint.

In clinical practice, specific phobia is commonly assessed through clinical judgment and parent-report measures, such as the Fear Survey Schedule for Children–Revised (Ollendick 1983), on which parents report how fearful their children are of particular situations and objects. This measure has been used to assess fears of low-functioning children with ASD (Matson and Love 1990).

Research studies often diagnose specific phobia and other anxiety disorders in children with high-functioning ASD using a structured diagnostic interview such as the Anxiety Disorders section of the Diagnostic Interview Schedule for Children (DISC; Shaffer et al. 2000), the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS; Ambrosini 2000), and the Anxiety Disorders Interview Schedule (ADIS; Silverman and Albano 1996). The DISC is usually administered with parents only, while the KSADS and ADIS are administered with both parents and children. The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al. 1994) is the adult version of the DISC and has been used with adults with ASD and normal IQ (Hofvander et al. 2009). These standardized interviews assess DSM-IV criteria for anxiety disorders, including the presence of symptoms, the frequency and severity of symptoms, and the extent of functional impairment. Leyfer et al. (2006) created a modified version of the KSADS for use in children and adolescents with ASD, which is called the Autism Comorbidity Interview–Present and Lifetime Version (ACI-PL).

Many research studies also use parent rating scales to examine fears or symptoms of specific phobia in children with ASD. One commonly used parent rating scale is the Child Symptom Inventory (Gadow and Sprafkin 1997), which has slightly different versions for children of

different ages, including the Early Childhood Inventory-4, ages 3–5; Child Symptom Inventory-4, ages 5–12; and the Adolescent Symptom Inventory-4, ages 12–18. However, these checklists measure a number of childhood symptoms and include only one specific phobia item: “Excessive fear to specific objects.”

While some of these measures have not been used with low-functioning children, they may still be appropriate for individuals with cognitive delays. The DISC, the Early Child Inventory and Child Symptoms Inventory, and the Fear Survey for Children (as well as its counterpart, the Fear Survey for Adults) have been used with individuals with mental retardation (Davis et al. 2008). The Fear Survey for Adults has been adapted for adults with mental retardation, and this version demonstrated good internal consistency and correlations with other measures of anxiety (Ramirez and Luckenbill 2007).

Differential Diagnosis. Specific phobia may present similarly to other types of anxiety such as separation anxiety disorder (SAD) or social phobia (SOP) as well as to symptoms associated with ASD such as sensory sensitivities.

Children who have fear of or avoid specific places or situations such as school, extracurricular activities, or playdates may present with similar symptoms as children with SAD or SOP. Children who experience distress around going to school or other places due to separation from their caregivers and/or excessive fear that harm may befall themselves or their caregivers while separated may be more accurately diagnosed with SAD (DSM-IV-TR). However, if children fear or avoid school due to fear of school itself rather than fear of separation, they may meet criteria for school phobia. Likewise, children who avoid school or playdates due to fear of social situations or fear of being judged may be more accurately diagnosed with social phobia.

Specific phobia may also overlap with sensory issues in individuals with ASD. For example, the finding that over 10% of children with ASD have a fear of loud noises (Leyfer et al. 2006) may be due to their sensory sensitivity (Baranek et al. 2006; Ben-Sasson et al. 2007). Reports of fear of objects such as balloons (Muris et al. 1998),

which can make loud, startling noises, may also be related to children’s sensory issues. Individuals with ASD who display fears should be evaluated for sensory sensitivities as well.

Treatment

Behavioral Treatments. Well-established treatments for TD children with fears and phobias usually combine behavioral exposures with a reinforcement system. A core mechanism of the exposure is systematic desensitization (e.g., Wolpe et al. 1973) in which the child is gradually exposed to increasingly anxiety-provoking stimuli. Other specific effective components of these treatments include reinforced practice, participant modeling, contingent reinforcement, prompting, modeling, extinction/blocking, and use of distracting stimuli (Davis and Ollendick 2005; Ollendick and King 1998). No randomized controlled trials or even larger-scale treatment studies have examined the effectiveness of behavioral treatments for specific phobia in children with ASD. However, a number of case studies have used this type of treatment (i.e., gradual exposure with reinforced practice, modeling, and contingent reinforcement) with children with ASD and found it to be effective in reducing their fears (Ellis et al. 2006; Gillis et al. 2009; Love et al. 1990; and Ricciardi et al. 2006). For example, Love et al. (1990) treated a child with a fear of going outside by dividing the front yard into seven distances from the front door and putting a retrievable object (e.g., a sticker or newspaper) in each marked-off section. In this treatment, the parent led the intervention and was coached by the therapist. The parent first modeled walking into the yard, then led the child into the yard, and then prompted the child to go into the yard on his own. The child received praise and a reward (e.g., food or a sticker) contingent upon attempting the exposure. The child was gradually prompted to go further and further into the yard.

Cognitive behavioral therapy (CBT) has been shown to be efficacious in treating anxiety in children with ASD (e.g., Chalfant et al. 2007; Reaven and Hepburn 2003; Reaven et al. 2009;

Sofronoff et al. 2005; Sze and Wood 2007; Wood et al. 2009). The CBT in these studies is similar to CBT used with TD children in that it includes psychoeducation, cognitive restructuring, self-talk, relaxation, and exposure to feared stimuli. However, it has been adapted for children with ASD by simplifying the cognitive aspects and emphasizing concrete aspects such as relaxation and exposure and, in some cases, by addressing poor social and adaptive skills in addition to anxiety (e.g., Wood et al.). While none of these studies examined effects on specific phobia in particular, the CBT used does include similar methods (e.g., graduated exposure, contingent reinforcement) to the behavioral treatments often used to treat specific phobia.

Pharmacological treatments for anxiety have been used off-label with some children and adolescents with ASD, including SSRIs, buspirone, and dextromethorphan. However, these drugs have not yet been tested against a control group or placebo condition in this population (White et al. 2009), and none of these medications have been tested in specific phobia.

See Also

- ▶ [Cognitive Behavioral Therapy \(CBT\)](#)
- ▶ [General Anxiety](#)

References and Reading

- Amaral, D. G., Schumann, C. M., & Nordahl, C. W. (2008). Neuroanatomy of autism. *Trends in Neuroscience*, 31(3), 137–145.
- Ambrosini, P. J. (2000). Historical development and present status of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS). *Journal of the American Academy of Child & Adolescent Psychiatry*, 39(1), 49–58.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.). Washington, DC: APA Press.
- Bailey, D. B., Hatton, D. D., Mesibov, G., Ament, N., & Skinner, M. (2000). Early development, temperament, and functional impairment in Autism and Fragile X syndrome. *Journal of Autism and Developmental Disorders*, 30(1), 49–59.
- Baranek, G. T., David, F. J., Poe, M. D., Stone, W. L., & Watson, L. R. (2006). Sensory experience questionnaire: Discriminating sensory features in young children with autism, developmental delays, and typical development. *Journal of Child Psychology and Psychiatry*, 47(6), 591–601.
- Bellini, S. (2006). The development of social anxiety in adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 21(3), 138–145.
- Ben-Sasson, A., Cermak, S. A., Orsmond, G. I., Carter, A. S., Kadlec, M. B., & Dunn, W. (2007). Extreme sensory modulation behaviors in toddlers with autism. *American Journal of Occupational Therapy*, 61(5), 584–592.
- Chalfant, A. M., Rapee, R., & Carroll, L. (2007). Treating anxiety disorders in children with high functioning autism spectrum disorders: A controlled trial. *Journal of Autism and Developmental Disabilities*, 37, 1842–1857.
- Chawarska, K., Klin, A., Paul, R., & Volkmar, F. (2007). Autism spectrum disorder in the second year: stability and change in syndrome expression. *Journal of Child Psychology and Psychiatry*, 48(2), 128–138.
- Davis, T. E., & Ollendick, T. H. (2005). Empirically supported treatments for specific phobia in children: Do efficacious treatments address the components of a phobic response? *Clinical Psychology: Science and Practice*, 12, 144–160.
- Davis, E., Saeed, S. A., & Antonacci, D. J. (2008). Anxiety disorders in persons with developmental disabilities: Empirically informed diagnosis and treatment. *Psychiatry Quarterly*, 79, 249–263.
- De Bruin, E. I., Ferdinand, R. F., Meester, S., de Nijs, P. F. A., & Verheij, F. (2007). High rates of psychiatric co-morbidity in PDD-NOS. *Journal of Autism and Developmental Disorders*, 37, 877–886.
- De Pauw, S. S. W., Mervielde, I., Van Leeuwen, K. G., & De Clercq, B. J. (2011). How temperament and personality contribute to the maladjustment of children with autism. *Journal of Autism and Developmental Disabilities*, 41, 196–212.
- Ellis, E. M., Ala'i-Rosales, S. S., Glenn, S. S., Rosales-Ruiz, J., & Greenspoon, J. (2006). The effects of graduated exposure, modeling, and contingent social attention on tolerance to skin care products with two children with autism. *Research in Developmental Disabilities*, 27, 585–598.
- Evans, D. W., Canavera, K., Kleinpeter, F. L., Maccubbin, E., & Taga, K. (2005). The fears, phobias, and anxieties of children with autism spectrum disorder and Down syndrome: Comparisons with developmentally and chronologically age matched children. *Child Psychiatry and Human Development*, 36(1), 3–27.
- First, M. B., Spitzer, R. L., Gibbon, M., et al. (1994). *Structured clinical interview for axis I DSM-IV disorders*. New York: Biometrics Research.
- Gadow, K. D., & Sprafkin, J. (1997). *Early childhood symptom inventory-4 norms manual*. Stony Brook: Checkmate Plus.

- Gadow, K. D., Devincent, C. J., Pomeroy, J., & Azizian, A. (2004). Psychiatric symptoms in preschool children with PDD and clinic and comparison samples. *Journal of Autism and Developmental Disorders*, 34, 379–393.
- Gadow, K. D., Devincent, C. J., Pomeroy, J., & Azizian, A. (2005). Comparison of DSM-IV symptoms in elementary school-age children with PDD versus clinic and community samples. *Autism*, 9(4), 392–415.
- Gadow, K. D., Roohi, J., DeVincent, C. J., & Hatchwell, E. (2008). Association of ADHD, tics, and anxiety with dopamine transporter (DAT1) genotype in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 49(12), 1331–1338.
- Gillis, J. M., Natof, T. H., Lockshin, S. B., & Romanczyk, R. G. (2009). Fear of routine physical exams in children with autism spectrum disorders: Prevalence and intervention effectiveness. *Focus on Autism and Other Developmental Disabilities*, 24, 156–168.
- Hofvander, B., Delorme, R., Chaste, P., Nydén, A., Wentz, E., Stahlberg, O., et al. (2009). Psychiatric and psychosocial problems in adults with normal-intelligence autism spectrum disorders. *BMC Psychiatry*, 9(35). <https://doi.org/10.1186/1471-244X-9-35>.
- Juranek, J., Filipek, P. A., Berenji, G. R., Modahl, C., Osann, K., & Spence, M. A. (2006). Association between amygdala volume and anxiety level: Magnetic resonance imaging (MRI) study in autistic children. *Journal of Child Neurology*, 21, 1051–1058.
- Kagan, J., Reznick, J. S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition. *Child Development*, 58, 1459–1473.
- Kagan, J., Reznick, J. S., & Snidman, N. (1988). Biological bases of childhood shyness. *Science*, 240, 167–171.
- Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62, 617–627.
- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., et al. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36, 849–861.
- Lord, C., Risi, S., Lambrecht, L., Cook, E., Leventhal, B. L., DiLavore, P. C., Pickles, A., & Rutter, M. (2000). The Autism Diagnostic Observation Schedule-Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30(3), 205–223.
- Love, S. R., Matson, J. L., & West, D. (1990). Mothers as effective therapists for autistic children's phobias. *Journal of Applied Behavior Analysis*, 23, 379–385.
- Matson, J. L., & Love, S. R. (1990). A comparison of parent-reported fear for autistic and nonhandicapped age-matched children and youth. *Australia and New Zealand Journal of Developmental Disabilities*, 16(4), 349–357.
- Muris, P., Sterneman, P., Merckelbach, H., Holdrinet, I., & Meesters, C. (1998). Comorbid anxiety symptoms in children with pervasive developmental disorders. *Journal of Anxiety Disorders*, 12(4), 387–393.
- Ollendick, T. H. (1983). Reliability and validity of the revised Fear Survey Schedule for Children (FSSC-R). *Behaviour Research and Therapy*, 21, 685–692.
- Ollendick, T. H., & King, N. J. (1998). Empirically supported treatments for children with phobic and anxiety disorders: Current status. *Journal of Clinical Child Psychology*, 27, 156–167.
- Ollendick, T. H., King, N. J., & Muris, P. (2002). Fears and phobias in children: Phenomenology, epidemiology, and aetiology. *Child and Adolescent Mental Health*, 7(3), 98–106.
- Ramirez, S. Z., & Luckenbill, J. F. (2007). Development of the fear survey for adults with mental retardation. *Research in Developmental Disabilities*, 28, 225–237.
- Reaven, J., & Hepburn, S. (2003). Cognitive-behavioral treatment of obsessive-compulsive disorder in a child with Asperger syndrome. *Autism*, 7, 145–164.
- Ricciardi, J. N., Luiselli, J. K., & Camare, M. (2006). Shaping approach responses as intervention for specific phobia in a child with autism. *Journal of Applied Behavior Analysis*, 39, 445–448.
- Shaffer, D., Fisher, P., Lucas, C., Dulcan, M., & Schwab-Stone, M. (2000). NIMH Diagnostic Interview Schedule for Children version IV (NIMH DISC-IV): description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child & Adolescent Psychiatry*, 39, 28–38.
- Silverman, W. K., & Albano, A. M. (1996). *The anxiety disorders interview schedule for children for DSM-IV: (child and parent versions)*. San Antonio: Psychological Corporation.
- Sofronoff, K., Attwood, T., & Hinton, S. (2005). A randomized controlled trial of a CBT intervention for anxiety in children with Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 46(11), 1152–1160.
- Sukhodolsky, D. G., Scahill, L., Gadow, K. D., Arnold, L. E., Aman, M. G., McDougle, C. J., et al. (2008). Parent-rated anxiety symptoms in children with pervasive developmental disorders: Frequency and association with core autism symptoms and cognitive functioning. *Journal of Abnormal Child Psychology*, 36, 117–128.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29, 216–229.
- Wolpe, J., Brady, J. P., Serber, M., Agras, W. S., & Liberman, R. P. (1973). The current status of systematic desensitization. *American Journal of Psychiatry*, 130(9), 961–965.
- Wood, J. J., Drahota, A., Sze, K., Har, K., Chiu, A., & Langer, D. A. (2009). Cognitive behavioral therapy for anxiety in children with autism spectrum disorders: A randomized, controlled trial. *Journal of Child Psychology and Psychiatry*, 50(3), 224–234.

Phonemes

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Synonyms

[Speech sound](#); [Segment](#)

Definition

A phoneme is a speech sound – the smallest contrastive sound unit in a language. When children learn their native languages, part of what they learn is to correctly perceive, produce, and combine the phonemes of their language. Though most children with autism acquire speech normally, some have difficulties with spoken language. These difficulties can range from “residual speech errors,” which are persistent mispronunciations of one or more phonemes (such as “w” for “r”) past the age of 8, to the complete absence of spoken language.

A phoneme is a cognitive construct, not a feature of the acoustic signal. Acoustically, speech is a continuous signal; however, it is perceived as consisting of a series of distinct phonemes. Though repeated examples of the same phoneme differ slightly from each other acoustically, they are still perceived as belonging to the same phonetic category.

Major classes of phonemes are vowels (such as “a” or “i”), consonants (such as “b” or “s”), and glides (such as “w”) or liquids (such as “l”). Vowels are produced with the vocal tract largely open and unobstructed. On the other hand, consonants are produced with a tight constriction or momentary closure somewhere in the vocal tract. Glides and liquids are produced with an intermediate degree of constriction. Phonemes are classified in terms of phonetic features that describe *place* of articulation (where in the vocal tract the phoneme is produced), *manner* of articulation (how it is produced), and *voicing* (whether the larynx is vibrating during production of the phoneme). For example, “v” is classified as a voiced

labiodental fricative. “Voiced” means that the larynx is vibrating while it is produced. This is in contrast with “f,” which is unvoiced because the larynx does not vibrate during its production. Like “f,” “v” is labiodental; it is produced with a constriction between the lower lip and the upper front teeth. Finally, “v” is a fricative, which means that air passes continuously through the labiodental constriction and causes a white-noise-like sound called frication.

Each language employs a different selection, or *inventory*, of all possible phonemes. Some phonemes, like the “th” sound in English “thin,” are rare across languages. Other phonemes, such as “b,” are used in nearly every language. Phonemes also combine with each other in ways that are determined by each language individually. For example, in Bantu words may begin with the “ng” sound, as in the name of the Ngorongoro Crater. In English, on the other hand, “ng” is only permissible at the ends of words (e.g., “sing”); it never appears word-initially.

It is important not to confuse phonemes with letters. While phonemes can be represented by letters (as we have done in the paragraphs above), the two are not the same. In English, the letter “a” can represent many phonemes, such as the “ey” sound in “apex,” the “ae” sound in “atom,” the “ah” sound in “watt,” or the “uh” sound in “banana.” Also, different letters may represent the same phoneme in different words. For example, the sound “ah” is spelled with the letter “a” in “watt” but with the letter “o” in “stop.” In order to standardize the representation of speech sounds across languages, the International Phonetic Association (IPA) has devised an alphabet in which each phoneme is represented by a unique symbol.

See Also

► [Phonetics](#)

References and Reading

Chenausky, K., & Tager-Flusberg, H. (2017). Acquisition of voice onset time in toddlers at high and low risk for autism spectrum disorder. *Autism Research*. <https://doi.org/10.1002/aur.1775>.

- Chenauksy, K., Nelson, C., & Tager-Flusberg, H. (2017). Vocalization rate and consonant production in toddlers at high and low Risk for autism. *Journal of Speech Language and Hearing Research*, 60, 865–876. https://doi.org/10.1044/2016_JSLHR-S-15-0400.
- Constantino, J. N., Yang, D., Gray, T. L., Gross, M. M., Abbacchi, A. M., Smith, S. C., & Kuhl, P. K. (2007). Clarifying the associations between language and social development in autism: A study of non-native phoneme recognition. *Journal of Autism and Developmental Disorders*, 37(7), 1256–1263.
- Kjelgaard, M., & Tager-Flusberg, H. (2001). An investigation of language impairment in autism: Implications for genetic subgroups. *Language & Cognitive Processes*, 16(2/3), 287–308.
- Rapin, I., Dunn, M., Allen, D., Stevens, M., & Fein, D. (2009). Subtypes of language disorders in school-age children with autism. *Developmental Neuropsychology*, 34(1), 66–84.
- Shriberg, L., Paul, R., Black, L., & van Santen, J. (2011). The hypothesis of apraxia of speech in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41, 405–426. <https://doi.org/10.1007/s10803-010-1117-5>.

Phonemic Errors

► Phonological Disorders

Phonemic Fluency

► Verbal Fluency

Phonetically Consistent Form

► Protowords

Phonetics

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Definition

Phonetics is the study of human vocal sounds, and linguistic phonetics is the study of those sounds

that are used in speech. Some authors use the term “phonetics” to refer only to the study of speech sounds, but the study of nonspeech vocal sounds such as infant cries, coughs, and singing can employ many of the same methods as are used in linguistic phonetics even though speech per se is not involved.

There are many aspects to the study of phonetics, but three main ways of investigating it are by using *perceptual* methods, *acoustic* methods, or *instrumentation*. Perceptual methods involve the auditory analysis of speech or vocal sounds, using either live or recorded stimuli. Since much research concerns the intelligibility, comprehensibility, or other type of impression of the vocal sound on another human, perceptual methods are considered the most ecologically valid. However, the human speech perceptual system is noted for its biases, many gained through experience with speech. These biases help humans filter extraneous information from the speech signal and extract just the information that will be useful for making quick decisions about what is being communicated. However, information which is “extraneous” in this sense is often scientifically useful, so *acoustic* methods are often paired with perceptual ones to provide a fuller picture of the speech signal.

Acoustic research in phonetics incorporates knowledge of normal and disordered vocal tract physiology, mathematical acoustics, signal processing, and perceptual psychophysics. Recordings of vocalizations are digitally processed (generally using an algorithm of the Fourier transform) and displayed on a computer screen in the form of a spectrogram, which is a graph showing the changes in frequency and amplitude of the utterance over time. From this, visual-perceptual analyses can be performed, along with quantitative measurements of frequency and amplitude at various points in the utterance.

Other instrumentation is also used in phonetic research. For example, a palatometer shows where the tongue contacts the hard palate during speech production. A nasometer measures airflow through the nasal passages during speech. X-ray videography and electropalatography give views of how the articulators of speech (lips, tongue, palate, velum, pharynx, and others) move during

utterances. More recently, facial movements involved in speech have been studied using reflective dots and motion-sensitive cameras.

In order to standardize the representation of speech sounds across languages, the International Phonetic Association (IPA) has devised an alphabet used to phonetically transcribe normal and disordered speech.

References and Reading

- Constantino, J. N., Yang, D., Gray, T. L., Gross, M. M., Abbacchi, A. M., Smith, S. C., Kuhl, P. K., et al. (2007). Clarifying the associations between language and social development in autism: A study of non-native phoneme recognition. *Journal of Autism and Developmental Disorders*, 37(7), 1256–1263.
- Erwin, R. J., Van Lancker, D., Guthrie, D., Schwafel, J., Tanguay, P., & Buchwald, J. S. (1991). P3 responses to prosodic stimuli in adult autistic subjects. *Electroencephalography and Clinical Neurophysiology: Evoked Potentials*, 80(6), 561–571.
- Riches, N. G., Loucas, T., Baird, G., Charman, T., & Simonoff, E. (2011). Non-word repetition in adolescents with specific language impairment and autism plus language impairments: A qualitative analysis. *Journal of Communication Disorders*, 44(1), 23–36.
- Spek, A., Schatorj, T., Scholte, E., & van Berckelaer-Onnes, I. (2009). Verbal fluency in adults with high functioning autism or Asperger syndrome. *Neuropsychologia*, 47(3), 652–656 (Comparative Study).

Phonological Deficits

► Phonological Disorders

Phonological Development

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Definition

Phonological development refers to the gradual acquisition of an adultlike system of speech

sounds that are used to convey meaning in a language. Phonological development can be considered in terms of both perception and production of speech sounds.

Historical Background

Historically, the development of speech sounds has been characterized in terms of the development of articulation abilities, with recent attention being paid to the notion that a larger set of rules and linguistic representations may also govern how individuals acquire speech sounds.

Current Knowledge

Perception

Research has demonstrated that infants are aware of speech sounds long before they are able to produce them. For example, infants at 1 month of age are able to discriminate speech sound categories such as the difference between phonemes /p/ and /b/ (i.e., categorical perception; Eimas et al. 1971). Over time, infants also develop the ability to track features of speech such as prosody (i.e., changes in pitch and loudness or the “melody” of speech) and stress patterns, as well as realize which distinctions between sounds are not meaningful in their native language (e.g., variability between speakers producing the same sound as in the case of a male versus female speaker, versus the meaningful difference between two distinct speech sounds). In addition, infants as young as 6 months of age have been shown to track the frequency with which speech sounds occur within a speech stream (i.e., phonotactic characteristics) (Saffran et al. 1996), a skill that has been linked to later vocabulary learning. In addition, by 10–12 months, infants demonstrate a reduced ability to recognize nonnative speech sound contrasts as the infant appears to have honed in on the sounds that occur within the language(s) to which he or she has regularly been exposed. It is in this early period that infants typically begin to demonstrate recognition of single words. During the second year of life, infants refine their vocabulary

comprehension abilities to be able to discriminate between words that sound very similar (e.g., “bih” and “dih”) and associate those words to new objects that are encountered in the environment, recognize words when provided with only a portion of the relevant phonetic information, and are able to comprehend words even if they are mispronounced. Perceptual abilities continue to progress as the child begins to associate phonological features with aspects of spoken language (see chapter ► [“Phonology”](#)).

Production

Infants begin to explore and experiment with their vocal tracts from birth. The earliest sounds that are produced are reflexive vocalizations, such as sneezing and breathing. As the infant matures, sounds include cooing and laughter, with increased variety of consonant and vowel-like sounds. Vowel sounds are typically produced the earliest, followed by consonant-like approximations that are produced in the back of the throat (e.g., /g/ and /k/) which are then followed by sounds that are produced in the front of the mouth (e.g., /m/, /n/, /b/, /p/). In addition to adding new sounds to the repertoire, the speech sounds are combined in sequences of increased length and complexity as the infant engages in vocal play. From 6 to 10 months, infants typically produce “reduplicated” syllables (i.e., repeated syllables containing the same consonant and vowel, e.g., “babababa”). From 10 to 14 months, babbling incorporates more variety in terms of sounds/syllables and pitch contours and is referred to as “nonreduplicated.” In addition, at approximately 1 year of age, infants use speech sounds to produce their first spoken words. In typical development, spoken vocabulary continues to grow well into school age and beyond.

As the child acquires more and more words, systematic patterns of sounds often emerge known as phonological processes (see examples of English phonological processes below, Hoff 2005).

- Weak syllable deletion: omission of an unstressed syllable in the target word, e.g., “nana” for “banana.”

- Final consonant deletion: omission of the final consonant in the target word, e.g., “ca” for “cat.”
- Reduplication: production of two identical syllables based on one of the target word syllables, e.g., “baba” for “bottle.”
- Consonant harmony: target word consonant takes on features of another target word consonant, e.g., “guck” for “duck.”
- Consonant cluster reduction: omission of a consonant in a target word cluster, e.g., “top” for “stop.”
- Velar fronting: a sound that is typically produced at the back of the mouth (e.g., /k/ or /g/) is replaced by a sound that is produced further forward, e.g., “ti” for “key.”
- Stopping: a fricative (i.e., a consonant that is produced with friction such as /s/, “sh,” /f/, or /v/) is replaced by a stop consonant in which airflow is completely obstructed (e.g., “ti” for “sea”).

By early school age, most phonological processes have resolved in typical development, as the child’s productions more closely match the adult form. However, some children demonstrate lingering phonological processes that impact intelligibility (see chapter ► [“Phonological Disorders”](#)).

Future Directions

Current research in phonological development is exploring the existence of what would be considered linguistic universals that govern all languages versus the acquisition of language-specific phonological rules.

In addition, a body of research exists that explores phonological development and its impact on the acquisition of reading and writing skills, in hopes of developing targeted instructional techniques for academic success.

See Also

► [Articulation](#)

References and Reading

- American Speech-Language-Hearing Association (ASHA). (1993). Definitions of communication disorders and variations. *American Speech-Language-Hearing Association*, 35(Suppl. 10), 40–41.
- Chomsky, N., & Halle, M. (1968). *The sound pattern of English*. New York: Harper & Row.
- Eimas, P. D., Siqueland, E. R., Jusczyk, P., & Vigorito, J. (1971). Speech perception in infants. *Science*, 171, 303–306.
- Gierut, J. (2008). Treatment efficacy summary: Phonological disorders in children. Retrieved 1 May 2011, from <http://www.asha.org/public/EfficacySummaries.htm>
- Hoff, E. (2005). *Language development*. Belmont: Thomson Wadsworth.
- Ladefoged, P. (1982). *A course in phonetics* (2nd ed.). London: Harcourt Brace Jovanovich.
- Saffran, J., Aslin, R., & Newport, E. (1996). Statistical learning by 8-month-old infants. *Science*, 274, 1926–1928.
- Sander, E. K. (1972). When are speech sounds learned? *Journal of Speech and Hearing Disorders*, 37, 55–63.
- Vihman, M. (1996). *Phonological development. The origins of language in the child*. Oxford: Blackwell.
- Werker, J., & Tees, R. (1984). Cross-language speech perception: Evidence for perceptual reorganization during the first year of life. *Infant Behavior and Development*, 7, 49–63.
- Werker, J., Fennel, C., Corcoran, K., & Stager, C. (2002). Infants' ability to learn phonetically similar words: Effects of age and vocabulary size. *Infancy*, 3, 1–30.

Phonological Disorders

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Synonyms

Phonemic errors; Phonological deficits

Short Description or Definition

A phonological disorder is an inability to articulate speech sounds accurately. The disorder may have a motoric (phonetic) component as well as a linguistic or cognitive (phonemic) basis. Therefore, phonological disorders may affect both the

intelligibility of a child's speech and his or her internalized knowledge of the language's sound system. The errors committed are usually rule governed, i.e., they show a pattern across all words spoken.

Categorization

The Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5, American Psychiatric Association 2013), specifies four criteria that must be present in order for Speech Sound Disorder (which includes Phonological Disorder) to be diagnosed. First, an individual must exhibit persistent unintelligible speech consisting of phoneme addition, omission, distortion, or substitution, which interferes with verbal communication. Second, the deficits must interfere with social participation, academic performance, and/or occupational performance. Third, the onset of symptoms is in childhood. Fourth, the symptoms cannot be accounted for by another medical or neurological condition.

Epidemiology

Phonological disorders are among the most prevalent communication disabilities diagnosed in preschool and school-age children, affecting 10% of this population. Children with phonological disorders are also at risk for reading and writing disabilities. If left unresolved, phonological disorders have long-term consequences that may interfere with an individual's future social, academic, and vocational well-being, largely resulting from persistent, reduced intelligibility of speech.

For individuals with ASD, impairments of phonological skills may be present, but compared to the difficulties shown in other language domains (social communication, stereotyped lexical usage, etc.), these skills are relatively preserved. Approximately one-third to one-half of individuals with ASD present with significant difficulty using speech as a functional and effective means of communication. In these individuals, it

is not uncommon for vocal attempts to be of limited intelligibility due to difficulties producing a variety of consonant sounds and using more complex syllable structures, such as those in multisyllabic words. The nature of these difficulties is not well documented in the literature; however, underlying difficulties may include challenges with oromotor planning and/or delays in phonological development (Lord and Paul 1997; National Research Council 2001). An estimated one-fourth of children with ASD are non-verbal, i.e., they lack spoken language. Most children with ASD who speak begin to do so by age 7 and almost never after age 13 (Pickett et al. 2009).

Among speaking children with ASD, there is typically no specific developmental impairment at the level of segmental phonology (consonant and vowel production). The speech that they produce will generally be understood, though, as with all children, it will undergo a process of developmental change to become more and more adultlike. Although unintelligible speech is not a core feature, atypical cases have been reported in the literature of children with ASD who exhibit severe phonological difficulties (e.g., Wolk and Giesen 2000). Moreover, there is evidence that distortion errors – which represent a phonetic rather than phonemic disorder – may persist into adulthood (Shriberg et al. 2001). Perception of speech sounds appears to be different in individuals with ASD. Functional imaging studies have indicated that both children and adults with autism show deficits in speech-sound processing (Boddaert et al. 2004). This impairment is limited to speech sounds; it does not affect the perception of tones (Whitehouse and Bishop 2008). The relationship between these deficits and the speech production abilities of individuals with ASD is not yet known.

Natural History, Prognostic Factors, and Outcomes

The development of speech begins at birth. As the speech mechanism systems of breathing, voice, and articulation mature, an infant is able to make a

controlled sound. This begins with soft, repetitive “cooing” vocalizations and by 6–7 months has progressed to repetitive syllables such as “ba, ba, ba” or “da, da, da.” This babbling becomes more elaborated and melodic so that it often has the tone and cadence of adult speech but does not contain real words. By the end of their first year, most children are starting to say a few simple words.

During the second year of life, children slowly develop a single-word vocabulary and by the end of that year most are putting words together. During the period of single-word speech, it is believed that children learn the sound structure of words as wholes. After the point where they have learned around 50 words, they begin to show awareness of the individual sounds or phonemes in words. The errors they commit start to show regularity, so that the same mistakes are committed on the same phonemes in different words (e.g., /s/ will be replaced by /t/ in the words sun, see, sick, etc.).

During the remainder of the preschool years and, for some children, extending into the early school years, there is a gradual process of phonological learning that can be described from two perspectives, one of sound mastery and the other of error suppression. In the first perspective, children are seen to slowly master all the vowels, individual consonants, and consonant sequences of their native language. Certain classes of sounds are mastered earlier than others. For example, many early emerging sounds are made in the front of the mouth by blocking the flow of air or directing it into the nasal cavity: /p, b, t, d, m, n/. Later emerging sounds are either made in the back of the mouth (e.g., /k, g/) or by partially blocking airflow to create fricative sounds (e.g., /s, z, th, sh/). In the second perspective on phonological development, children are observed to show regular ways of simplifying their pronunciations compared to those of adults. These patterns, or phonological processes, include changes to the syllable structure of words (e.g., omitting unstressed syllables or eliminating one of the sounds in a consonant sequence), omissions of sounds (especially at the ends of syllables), and substitutions of earlier/simpler sounds for later/more complex ones (e.g., substituting /t/ for /k/). Many phonological processes occur commonly in

children and are considered to be part of their biological predisposition; these are referred to as “natural” processes. Others may be idiosyncratic or unique to an individual child.

Phonological disorders in children are characterized by speech that is difficult to understand, especially by individuals who do not know the child speaker well. As a rule of thumb, children should be understood by strangers about half the time at age 2, about 75% of the time at age 3, and nearly all the time at age 4. When speech is unintelligible, it is usually due to a combination of common and uncommon errors. Certain of the normal or “natural” phonological processes may persist beyond the ages at which they are normally suppressed. Other atypical or idiosyncratic processes may also be present. There may also be a combination of phonemic (substitution, omission, and addition of sounds) and phonetic (distortions of sounds) errors together in the same child. Individuals with ASD may present unintelligible speech for all these reasons and, in addition, may exhibit disturbances of prosody that further compromise their intelligibility.

Clinical Expression and Pathophysiology

Phonological disorder is an impairment in the ability to comprehend or produce the sound system of a language. This sound system is characterized as having two parts: segmental phonology, which pertains to consonants and vowels and their combination into syllables, and nonsegmental phonology (also called suprasegmental phonology or prosody), which includes speech variables such as rate, stress, intonation, and pause. In the field of speech-language pathology, the term phonological disorder is typically used to refer to segmental problems, specifically difficulty in inducing the rules that govern sound combinations. Thus, a phonological disorder is viewed as a subtype of language disorder (Bauman-Waengler 2012).

With such disorders, errors are observed in the production of individual speech sounds and in the formation of syllable structures of words. Sounds

may be omitted, substituted by other sounds, or added to the normal form of a word. When these errors are numerous or result in sounds quite different from the targets, they may produce speech that is partially to fully unintelligible to a listener, especially one who is unfamiliar with the speaker. Phonological disorders are distinguished from phonetic disorders (also described as articulation disorders) that result from slight misalignments of the articulators during speaking and are manifested as distortion errors. Unlike phonetic disorders, phonological disorders are frequently part of a larger language disorder characterized by impairments of one or more other linguistic domains such as vocabulary, comprehension, morphosyntax, or literacy.

Although nonsegmental phonological or prosodic impairments are normally not considered part of a phonological disorder from a clinical perspective, they clearly belong within the linguistic domain of phonology. Moreover, unusual prosodic features are a concomitant behavior in many individuals with autism. It is one of a number of behavioral red flags that distinguish toddlers with autism from those either developing normally or with other types of developmental delay (McCann and Peppe 2003; Wetherby et al. 2004). Among the speech production behaviors where differences are reported to exist are stress, rate, chunking (verbal phrasing), intonation, and expression of affect. Problems have also been noted in the detection and comprehension of prosodic variation in speech. In spite of this, accounts of prosodic disability in autism are inconsistent and have utilized widely varying populations and methodologies.

Evaluation and Differential Diagnosis

Phonological disorders in young children are assessed through standardized and non-standardized procedures. Optimally, two speech samples are obtained: one of single-word speech, typically elicited through a picture- or object-naming task, and the second of connected speech, gathered through a play interaction or short

interview. To elicit the first type of sample, a large number of standardized, norm-referenced tests of speech-sound production are available for children 3 years or older (e.g., *Clinical Assessment of Articulation and Phonology*, Second Edition, Secord and Donohue, 2013; *Goldman-Fristoe Test of Articulation-Third Edition*, Goldman and Fristoe 2015; *Arizona Articulation Proficiency Scale, Third Edition*, Fudala 2000).

Speech samples of both types are transcribed phonetically and then analyzed to determine whether the child displays primarily speech production deficits (phonetic errors or sound distortions) and/or deficits associated with phonological constraints (phonemic errors of omission, substitution, or addition of sounds). Independent analyses such as a phonetic inventory (listing of all sounds produced, regardless of correctness) are used to evaluate possible sensory or motor limitations. The child is also screened for auditory acuity and is given an oral-peripheral examination to determine whether there exist structural or physiological limitations to speech. Relational analyses such as an assessment of phonological processes (patterns of sound simplification, such as replacing posterior /k, g/ sounds with anterior /t, d/ sounds) are used to determine whether errors are developmentally common or idiosyncratic. The identification of error patterns also forms the basis for treatment to improve speech intelligibility.

Treatment

In the treatment of phonological disorders, the primary goal is to improve a child's speech intelligibility to facilitate effective communication. This entails both teaching the accurate production of speech sounds and improving the conceptual organization of speech-sound information so that phonemic contrasts are clearly marked. Effective and efficient treatment relies on a generalization of learning. The goal is to induce a widespread change in a child's sound system so that it is more in line with the phonology of the target language.

Treatment often occurs in the context of an interdisciplinary service delivery team that may include speech-language pathologists, audiologists, nurses and physicians, occupational and physical therapists, parents, psychologists, social workers, special educators, and teachers. The composition of this team is dependent on the child's needs not only in the area of communication but in development generally. The team initiates and coordinates the optimal intervention program for the child and facilitates the program's transfer and utility in daily settings.

After assessing the phonological disorder and identifying and prioritizing the errors that exist, a treatment plan is developed to correct speech-sound production. The goal of treatment is to improve accuracy and use of speech sounds so that intelligibility is improved in both single words and connected speech. Generalization is sought across all settings in which children communicate. No single treatment approach is used. In general, all approaches utilize structured sound production practice at gradually increasing levels of difficulty.

Intervention for individuals with ASD must often be adapted to suit their unique interests, styles of interaction, and sensory preferences and aversions. For example, the commonly used technique of saturating the child with auditory models of a target speech sound might prove disastrous with an individual who is auditorily hypersensitive. Another common technique, contrasting minimal word pairs such as "call" and "tall," may be difficult to implement because it relies on role reversal, the teacher playing the student, and vice versa.

See Also

- [Articulation](#)
- [Articulation Disorders](#)
- [Auditory Acuity](#)
- [Intonation](#)
- [Prosody](#)
- [Speech Delay](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR*. Washington, DC: Author.
- American Speech-Language-Hearing Association (ASHA). (1993). Definitions of communication disorders and variations. *American Speech-Language-Hearing Association*, 35(Suppl. 10), 40–41.
- American Speech-Language-Hearing Association (ASHA). (2006). *Guidelines for speech-language pathologists in diagnosis, assessment, and treatment of autism spectrum disorders across the life span (Guidelines)*. Retrieved April 26, 2011, from www.asha.org/policy
- Bauman-Waengler, J. (2012). *Articulatory and phonological impairments: A clinical focus* (4th ed.). Upper Saddle River: Pearson Education.
- Boddaert, N., Chabane, N., Belin, P., Bourgeois, M., Royer, V., Barthélemy, C., et al. (2004). Perception of complex sounds in autism: Abnormal auditory cortical processing in children. *American Journal of Psychiatry*, 161, 2117–2120.
- Fudala, J. B. (2000). *Arizona articulation proficiency scale* (3rd ed.). Los Angeles: Western Psychological Services.
- Gierut, J. (2008). *Treatment efficacy summary: Phonological disorders in children*. Retrieved April 26, 2011, from <http://www.asha.org/public/EfficacySummaries.htm>
- Goldman, R., & Fristoe, M. (2015). *The Goldman-Fristoe test of articulation* (3rd ed.). San Antonio: Pearson.
- Lord, C., & Paul, R. (1997). Language and communication in autism. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (2nd ed., pp. 195–225). New York: Wiley.
- McCann, J., & Peppe, S. (2003). Prosody in autism spectrum disorders: A critical review. *International Journal of Language and Communication Disorders*, 38, 325–350.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press, Committee on Educational Interventions for Children with Autism, Division of Behavioral and Social Sciences and Education.
- Pickett, E., Pullara, O., O'Grady, J., & Gordon, B. (2009). Speech acquisition in order nonverbal individuals with autism: A review of features, methods, and prognosis. *Cognitive and Behavioral Neurology*, 22, 1–21.
- Secord, W., & Donohue, J. (2013). *Clinical assessment of articulation and phonology*, (2nd ed.). Greenville: Super Duper Publications.
- Shriberg, L., Paul, R., McSweeney, J., Klin, A., Cohen, D., & Volkmar, F. (2001). Speech and prosody characteristics of adolescents and adults with high functioning autism and Asperger syndrome. *Journal of Speech Language and Hearing Research*, 44, 1097–1115.
- Wetherby, A. M., Woods, J., Allen, L., Cleary, J., Dickinson, H., & Lord, C. (2004). Early indicators of autism spectrum disorders in the second year of life. *Journal of Autism and Developmental Disorders*, 34, 473–493.

Whitehouse, A. J. O., & Bishop, D. V. M. (2008). Do children with autism 'switch off' to speech sounds? An investigation using event-related potentials. *Developmental Science*, 11, 516–524.

Wolk, L., & Giesen, J. (2000). A phonological investigation of four siblings with childhood autism. *Journal of Communication Disorders*, 33, 371–389.

Phonology

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Synonyms

Articulation; Speech

Definition

Phonology refers to the system of speech sounds within a particular language. Phonology differs from articulation in that phonology is concerned with the rule-governed ways in which speech sounds are used and interact with each other to encode meaning within a language, while articulation is generally associated with the movement of structures to produce speech sounds.

See Also

- [Articulation](#)
- [Speech](#)

References and Reading

- Bowen, C. (1998). Children's speech sound disorders: Questions and answers Retrieved 25 Apr 2011, from <http://www.speech-language-therapy.com/phonol-and-artic.htm>
- Crystal, D. (1991). *A dictionary of linguistics and phonetics* (3rd ed.). Cambridge, MA: Basil Blackwell.
- Ladefoged, P., & Maddieson, I. (1996). *The sounds of the world's languages*. Oxford: Blackwell.
- Sander, E. K. (1972). When are speech sounds learned? *The Journal of Speech and Hearing Disorders*, 37, 55–63.

Phrase Length

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Definition

Phrase length is a measure of the complexity of expressive language. A phrase is a grammatical construction (e.g., noun phrase, verb phrase) that does not contain both a subject and a predicate; therefore, it does not qualify as a full sentence. It is one type of measure that can be used to estimate an individual's level of spoken language.

See Also

- ▶ [Grammar](#)
- ▶ [Language Acquisition](#)
- ▶ [Narrative Assessment](#)
- ▶ [Syntax](#)

Phrenology

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Synonyms

[Craniognomy](#); [Craniology](#); [Cranioscopy](#)

Definition

Phrenology was an early psychological theory that mental abilities were compartmentalized in

specific areas in the brain and that the strength of these abilities could be determined by the size of the bump in the skull for that particular ability. Phrenology was originally proposed by Francis Gall in 1796 and was one of the first to suggest that a brain function could be localized to a specific area of the brain. Phrenologists would palpate the exterior skull and, based on this procedure, would generate predictions and psychological analyses of an individual's strengths and weaknesses. This procedure depends on the inaccurate assumption that the exterior surface of the bony scalp is a faithful reflection of the configuration of soft internal brain tissue. This theory received little research support and is typically only mentioned as a historical prologue to modern psychology.

References and Reading

- Fancher, R. E. (1979). *Pioneers of psychology*. New York: Norton.
- Fodor, J. A. (1983). *Modularity of mind: An essay on faculty psychology*. Cambridge, MA: MIT Press.

Physical and Neurological Examination

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Definition

The physical examination is performed by a physician, nurse practitioner, or other trained medical care provider to determine the state of health of an individual. It is important for a physical examination to be performed as part of an initial evaluation of a child with an autism spectrum disorder to identify co-occurring medical or etiologic disorders. A standard approach to the assessment of all organ systems is used. The neurological examination determines if abnormalities exist in the central or peripheral nervous system using a standard

approach based on the understanding of motor, sensory, coordination, and cognitive systems.

Historical Background

Kanner's original 1943 case reports of children with autism contain few references to the physical or neurological exam. One child was described as resembling his father physically. Several had an "intelligent physiognomy." There was one child with a supernumerary nipple, but no other congenital anomalies are reported. Physically they were described as essentially normal though 5 of the 11 had an increased head circumference. Gross motor clumsiness was also present in several cases. As the diagnosis was more widely evaluated, initially more focus was placed on a psychoanalytic approach to understanding autism. When interest in exploring the biological basis for autism increased, physical findings were still minimally noted. A review of the literature in 1973 indicated physical findings that were primarily soft signs including poor tone, poor coordination, clumsiness, exaggerated reflexes, and strabismus (Ornitz 1973). Maurer and Damasio in 1983 explored the neurological basis for autism relating core behaviors to known diagnoses and their anatomic correlate to localize areas of dysfunction and better understand the etiology of autism. As autism became understood as a neurobiological disorder, there was interest in the role known congenital medical disorders played in autism. Cases associated with a known syndrome or medical disorder were reported at 10–20% (Barton and Volkmar 1998). However, that number is decreasing as fewer children identified with ASD have a coexisting intellectual disability and intellectual disability is associated with a higher rate of identifiable disorders. A distinction is made between syndromic autism and idiopathic autism with an essentially normal exam (Johnson and Mayes 2007).

Current Knowledge

The complete evaluation of a child suspected of having an autism spectrum disorder is a multipart

process. The physical and neurological exam is informed by a comprehensive medical and developmental history. While performing a detailed physical, dysmorphicologic, and neurological exam is important, the clinical history increases the clinician's index of suspicion. It prompts evaluation of associated findings while performing the exam.

History of delays in reaching multiple developmental milestones can suggest intellectual disability which is a comorbid diagnosis with autism spectrum disorder in some 50% of children. The physical exam can provide findings suggestive of a genetic disorder associated with intellectual disability. History can indicate symptoms of disorders that are increased in children with autism. These include sleep disorders and gastrointestinal problems. Oral findings, body habitus, and growth should be assessed as contributors to these problems.

The history of functional abilities will characterize eating, sleeping, toileting, and other activities of daily living such as dressing. Performance of these tasks can help characterize motor findings associated with autism. Motor clumsiness or dyspraxias (motor planning problems) will present as difficulties learning to chew or drink from a cup or, in the older child, delays in learning to dress or to write. Sensory integration difficulties can present as issues with food textures, temperature, and taste. Tactile sensitivity to clothing tags or tight clothes can complicate getting dressed. The clinician will want to evaluate tone and coordination and motor function to better characterize these presenting concerns.

Family history will indicate others with autism, intellectual disability, genetic disorders, or birth defects. Preconception events such as advanced parental age as well as preterm birth complications may raise the risk for ASD. The exam can identify findings that are familial or complications of prematurity such as chronic lung disease, visual impairment, or growth problems. Prenatal exposures can have effects that are identifiable on exam such as microcephaly and facial findings seen with fetal alcohol syndrome.

Parents who are bringing a child either for further evaluation following diagnosis or with

significant behavioral concerns prior to diagnosis may wish to review resources detailing how to approach the exam and prepare the child. This will engender less stress on the child's part and assist the clinician in having time for observation and examination. Children can be prepared visually with videos, social stories, books, or pictures. Doctor kit toys can allow the child to practice the exam. Office staff appreciate a parent letting them know what to expect from the patient. For example, a child who is distressed with noise and movement may be brought in as soon as possible to wait in a quiet room instead of a busy waiting room. Staff may suggest other appointment times that will facilitate this. Vital signs may need to be done by hand rather than machine. In making the appointment, staff can let parents know how much time will be needed and what information they should make available. If the clinician has time to review school reports or testing results before the visit, additional adjustments can be made knowing the child's level of function.

It is not unusual for children with disabilities to have difficulty with office-based screenings of hearing and vision. The child may not be able to follow the directions due to cognitive, verbal, or attention problems, but it may be assumed that the child did "not want to cooperate." A normal newborn hearing screening test does not rule out a progressive hearing loss. Staff will want to tell the examiner about the interaction as well as the results and see that a reliable result is obtained or a referral made to an audiologist where a variety of evaluations are available. There is an increase in hearing and visual impairment in autism spectrum disorders. Autism spectrum disorders are more frequent in children with optic nerve hypoplasia such as septo-optic dysplasia.

Interaction during the physical examination provides opportunities for additional informal observation of core behaviors diagnostic of an autism spectrum disorder including deficits in social interaction and communication and repetitive behavior. A young autistic child may be indifferent to the examiner. Other young children may appreciate the chance to play with the "doctor's toys," and this can be a measure of engagement, enjoyment, and turn taking. Response to the

examiner's directions allows observation of eye contact and engagement with the examiner and the ability to imitate. For the verbal child, vocal quality, tone and prosody, and the ability to follow verbal directions as well as the patient's comments and questions can be assessed. Equally important are associated behavioral findings that include pica, self-injury, irritability, ADHD-type symptoms, anxiety, or depression. The physical exam offers opportunities to observe activity level and impulsivity related to ADHD. A child who can talk but will not can be assessed for autism but also for selective mutism, social anxiety, or depression.

Physical evaluation for a child with a suspected autism spectrum disorder includes a comprehensive pediatric assessment. Vital signs and growth parameters should be reviewed. Symmetric growth delays can be familial or indications of a genetic syndrome. Overgrowth can also suggest a genetic contribution (e.g., Sotos syndrome). A short child with a preserved head circumference may have a genetic or endocrine disorder.

Measurement of head circumference is important. Macrocephaly is seen in 25–30% of children with autism. Microcephaly is present in 5–15% and is associated with a poorer outcome. If microcephaly is present at birth, this can indicate prenatal or genetic problems. A normal head circumference at birth followed by poor growth may be due to perinatal event. In Rett syndrome, head growth decelerates in the second half of the first year of life.

Head circumference is normal or below average in autism spectrum disorders at birth followed, in some children, by a rapid increase in growth leading to macrocephaly as a toddler. This does not persist into adulthood being present in 1–3% of adults with ASD (Myers 2009). Macrocephaly is associated with PTEN along with associated skin findings of lipoma and penile freckling.

Examination should look for comorbid medical conditions. These include, for example, evaluation of middle ear status where infection or fluid can lead to conductive hearing loss. A small mid-face, high arched palate, or enlarged tonsils with a history of sleep disturbance suggests a risk of obstructive sleep apnea. A child with GI

complaints may have abdominal fullness, discomfort, or a palpable mass in the left lower quadrant consistent with constipation. Obesity can complicate the presentation for a child with hypotonia and/or motor delays. The presentation of some disorders changes over time and repeated exams can be informative. Head growth trends can be confirmed, and findings may become more typical of a particular syndrome.

A careful dysmorphism examination is indicated. In general, dysmorphic features and a lower IQ increase the likelihood of finding a medical biologic diagnosis.

Examination of the head includes (in addition to head circumference) examination of head shape and hair whorl patterns. Facial proportion evaluation can identify prominent forehead, frontal bossing, small midface, or chin. Ear placement, size, and structure should be noted, as well as the presence of tags or pits. Eye exam includes eyebrow, eyelids, shape, position, and patterns and pigmentation of the iris. Examination continues with the shape of the nose and formation of the philtrum. Size of the mouth, shape of lips, and position (upturned or downturned lips) should be noted. Oral exam includes tooth shape and enamel defects, shape of hard palate and uvula, and the tongue.

Examination of the hands and feet is also informative. Hand exam includes finger length and relative proportion, separation or syndactyly, clinodactyly, and palmar creases. Significant second and third toe syndactyly is a finding in Smith–Lemli–Opitz syndrome which is associated with autism spectrum disorder. Other findings include ptosis, cleft palate, and microcephaly and growth retardation (Smith’s 2006).

Detailed skin exam with an ultraviolet Wood’s lamp looks for findings such as café au lait spots or axillary freckling characteristic of neurofibromatosis or shagreen patch and pale areas/ash leaf spots characteristic of tuberous sclerosis (Smith’s 2006). Lipomas and penile freckling are associated with PTEN.

Known genetic syndromes include chromosomal and single gene disorders. Down syndrome is a chromosomal disorder (trisomy 21) that is associated with a characteristic pattern of findings. Up to 7% of individuals with Down

syndrome are identified as having an autism spectrum disorder. Findings on exam can include microcephaly, flattened occiput, small ears, epicanthal folds and speckled iris, short neck, single palmar crease, and hypotonia.

Fragile X is a single gene disorder (*FMR1* gene) found in 1–3% of children with an autism spectrum disorder. Physical findings include macrocephaly, prominent ears, hypotonia, and macroorchidism identifiable after puberty.

The neurological exam typically includes assessment of cranial nerves, muscle strength and tone, reflexes, and gait. In assessing children on the autism spectrum, hand, finger, and gait stereotypies are often observed. Additional observation may be necessary to discriminate tics from stereotypy. Differences can be seen qualitatively in the performance of skills.

Details of the exam change with the age of the child. In the infant, this will include an assessment of the primitive reflexes and whether they are abnormally persistent. Focal findings and asymmetries of tone leading to delays raise concerns for intracranial processes. In an evaluation of children age 2–6 with an autism spectrum disorder and no diagnosable medical condition, over 60% had hypotonia and/or hyporeflexia (Akshoomoff et al. 2007). Hypotonia with regression and FTT may raise suspicion of a mitochondrial disorder. Assessment of functional abilities provides additional information about delays, regression, or disordered patterns of development. Unusual gait patterns have been associated with autism spectrum disorders, though not all researchers have found differences (Ozonoff et al. 2008). Persistence of primitive reflexes, regression, or asymmetry of motor findings may appear with repeated exams.

In older children, additional assessment of neurological soft signs will help characterize motor deficits or delays. These include assessments such as posturing or motor overflow when performing stressed gaits, motor impersistence or tremor, synkinesis, and motor movement. A drawing with the child’s name offers information about motor and cognitive skills. Additional assessment of verbal and nonverbal language enriches this evaluation.

Future Directions

As noted, autism without a known medical or syndromic diagnosis, referred to as idiopathic autism, is less likely to be associated with intellectual disability (Johnson and Mayes 2007). However, an increased number of dysmorphic findings have been identified in individuals with idiopathic autism (Ozgen et al. 2011). Use of a systematic dysmorphology examination may eventually identify phenotypes that can be correlated with newer genetic information. Currently, microarray analysis can identify genetic differences in up to 10% of cases of idiopathic autism (Miles 2011).

There are also consistent reports of motor delays and deficits in gait and praxis reported in the children with nonsyndromic autism that distinguish them from nonautistic peers (Fournier et al. 2010). It is hoped that a more systematic evaluation of these findings and their course over time can also identify diagnostic subgroups that will correlate with outcome.

See Also

- Ataxia
- Congenital Disorders
- Constipation
- Course of Social Avoidance in Fragile X Syndrome, The
- Down Syndrome
- Dystonia
- Eyeblink Reflexes
- Genetics
- Hypotonia
- Medical Conditions Associated with Autism
- Microcephaly
- Periventricular Leukomalacia
- Tics

References and Reading

Akshoomoff, N., Farid, N., Courchesne, E., & Haas, R. (2007). Abnormalities on the neurological examination and EEG in young children with pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 37(5), 887–893.

- Barton, M., & Volkmar, F. (1998). How commonly are known medical conditions associated with autism. *Journal of Autism and Developmental Disorders*, 28(4), 273–276.
- Coury, D. (2010). Medical treatment of autism spectrum disorders. *Current Opinion in Neurology*, 23(2), 131–136.
- Esposito, G., & Venuti, P. (2008). Analysis of Toddlers' Gait after six months of independent walking to identify autism: A preliminary study. *Perceptual and Motor Skills*, 106(1), 59–269.
- Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism spectrum disorders: A synthesis and meta-analysis. *Journal of Autism and Developmental Disorders*, 40, 1227–1240.
- Gardener, H., Spiegelman, D., & Buka, S. (2009). Prenatal risk factors for autism: Comprehensive meta-analysis. *British Journal of Psychiatry*, 195(1), 7–14.
- Johnson, C. P., & Mayes, S. (2007). The committee on children with disabilities identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120(5), 1103–1215.
- Maurer, R., & Damasio, A. (1982). Childhood autism from the point of view of behavioral neurology. *Journal of Autism and Developmental Disorders*, 12(2), 195–205.
- Miles, J. H. (2011). Autism spectrum disorders – A genetics review. *Genetics in Medicine*, 13(4), 278–294.
- Miles, J. H., McCathren, R. B., Stichter, J., & Shinawi, M. (2003). Autism spectrum disorders. In R. A. Pagon, T. D. Bird, C. R. Dolan, et al. (Eds.), *Gene reviews™ (Internet)*. Seattle: University of Washington, Aug 27, 2003, Updated Apr 13, 2010.
- Miles, J. H., Takahashi, T. N., Hong, J., Munden, N., Flounoy, N., Braddock, S. R., et al. (2008). Development and validation of a measure of dysmorphology: Useful for autism subtype classification. *American Journal of Medical Genetics Part A*, 146(9), 1101–1116.
- Minor Anomalies. (2006). In K. L. Jones (Ed.) *Smith's recognizable patterns of human malformation* (6th ed., pp. 817–834). Philadelphia: Elsevier Saunders.
- Montgomery, T. R. (2008). Neurodevelopmental assessment of school-age children. In *Capute and Accardo's neurodevelopmental disabilities in infancy and childhood* (Vol. I, 3rd ed., pp. 405–417). Baltimore: Paul H. Brookes.
- Moss, J., & Howler, P. (2009). Autism spectrum disorders in genetic syndromes: Implications for diagnosis, intervention and understanding the wider autism spectrum. *Journal of Intellectual Disability Research*, 53(10), 852–873.
- Myers, S., & Challman, T. D. (2011). Autism spectrum disorders. In R. G. Voight et al. (Eds.), *Developmental and behavioral pediatrics* (pp. 249–267). Itasca: American Academy of Pediatrics.
- Nazeer, A., & Ghaziuddin, M. (2012). Autism spectrum disorders: Clinical features and diagnosis. *Pediatric Clinics of North America*, 59(1), 19–25.

- Ornitz, E. (1973). Childhood autism: A review of the clinical and experimental literature. *California Medicine*, 118(4), 21–47.
- Ozgen, H., Hellemann, G. S., Stellato, R. K., Lahuis, B., van Daalen, E., Staal, W. G., et al. (2011). Morphological features in children with autism spectrum disorders: A case matched case-control study. *Journal of Autism and Developmental Disorders*, 41(1), 23–31.
- Ozonoff, S., Young, G. S., Goldring, S., Greiss-Hess, L., Herrera, A. M., et al. (2008). Gross motor development, movement abnormalities and early identification of autism. *Journal of Autism and Developmental Disorders*, 38(4), 644–656.
- Punt, M., De Jong, M., De Groot, E., & Haddus-Algra, M. (2010). Minor neurologic dysfunction in children with autism. *Developmental Medicine & Child Neurology*, 52(12), 1127–1132.

Physical Medicine

► Physical Therapy

Physical Therapy

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Synonyms

Physical medicine; Physiotherapy; PT;
Rehabilitation

Definition

Physical therapy involves the treatment and prevention of physical disorders or injuries that cause a person to lose normal mobility, strength, range of motion, or quality of their physical functioning. Physical disorders can be due to medical problems related to congenital conditions, acquired diseases and illnesses, or the result of an individual's inadequate maintenance of proper posture and joint alignment over time.

Physical therapy is performed and overseen by a licensed physical therapist, also known as a

PT. Physical therapists are trained healthcare providers who evaluate, diagnose, and treat people of all ages, from newborns to the oldest individuals. PTs use a patients' history, medical records, and a full physical examination to determine a plan of care that involves education, treatment, and maintenance of any gains achieved. PTs are also involved with the training and education of a patients' caregivers, including parents, spouses, teachers, aides, and other individuals.

Physical therapy can utilize many treatment modalities such as specific exercises, manual treatments, gait and movement retraining, manipulations of tissue and/or joints, education, and other interventions. Treatments can also be performed by physical therapy assistants who act under the direction of the physical therapist.

Because autism is a pervasive developmental disorder, it is common for people with autism to have delays, differences, and disorders of their gross motor skills which can benefit from physical therapy intervention. In very young children, physical therapy can improve basic motor skills like sitting, rolling, standing, and playing. PTs can provide education to parents for techniques they can use to help the child gain strength, coordination, and improved movement skills.

As the child grows older, the PT intervention usually shifts to a school-based program that focuses on more advanced skills such as running, throwing, catching, and climbing. These physical skills can facilitate participation in recess, play, and sports which in turn can improve the child's social engagement with other children. These treatments can be performed one-on-one or in typical school activities. Frequently, physical therapists will pull in non-autistic kids to work in group settings so that the social aspects of the child's physical skills are also developed. The educational training from the PT in the school focuses on tools that help the staff further build the child's social/physical skills.

References and Reading

- <http://www.apta.org>
Rudy, L. J. (2010). *Physical therapy and autism: The basics*. autism.about.com/od/autismtherapy101.

Physiological Measures of Parental Stress

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Synonyms

Heart rate variability (HRV); Respiratory sinus arrhythmia (RSA); Stress reactivity

Definition

Caregivers of children with Autism Spectrum Disorder (ASD) experience greater levels of parenting stress than parents of typically developing (TD) children (Davis and Carter 2008; Estes et al. 2013). Although self-reported stress measures are informative, physiological measures of stress provide a more objective method for studying individual differences and underlying biological influences in stress reactivity. High-frequency heart rate variability (HRV) is the variation in interval between heartbeats and is parasympathetically mediated, whereas heart rate measures the number of times the heart beats per min and includes both sympathetic and parasympathetic influences. Higher child ASD traits have been positively related to greater HRV increase or change in mothers during a stressful interaction (Factor et al. 2017). In the same study, maternal self-report of stress and HRV were not significantly related, which suggests that physiological and self-report measures of stress may tap into different components of stressful experiences (e.g., trait versus state, context specific). Research suggests that the measurements of baseline HRV and HRV reactivity likely capture various regulatory abilities. HRV reactivity provides an estimate of the degree of adaptability in certain contexts, whereas baseline

HRV suggests the capacity to change or engage in regulatory strategies.

Since HRV has been used to indicate emotional responding and flexibility, as well as psychopathological features and processes (Thayer and Lane 2000), there are different possible interpretations for the finding of increased HRV in mothers. If context is not considered, higher baseline HRV may reflect a greater capacity for regulated emotional responses, such that parents whose children have more severe ASD traits might have developed more adaptive coping mechanisms. However, previous research has suggested the importance of considering HRV reactivity in the context of the situation. Hastings et al. (2008) indicated that Respiratory Sinus Arrhythmia (RSA), another index of HRV, changes depending on the situation and demonstrated findings that showed lower RSA in threatening situations might reflect the use of effective coping strategies, whereas increased RSA in nonthreatening situations might reflect more adaptive responding. With this in mind, the increase in HRV during the parent-child stressor actually may reflect an atypical coping response during a stressful situation in parents with children with ASD traits.

Preliminary findings also support the hypothesis that RSA reactivity moderates the relationship between maternal rigidity (Condy, Factor, Swain, Strege, Scarpa, in press), often associated with the Broad Autism Phenotype (BAP), and maternal affect during a challenging interaction between mother and child. BAP rigidity and RSA reactivity predicted maternal affect above and beyond the powerful effect of child social impairment. While these findings are preliminary, the results support further investigation of the role of physiological responding in this population in a larger sample. Results also likely suggest that parents with RSA augmentation and higher inflexibility may be less able to physiologically mobilize resources during a stressful task to engage positively with their children.

Overall, further exploration of treatments that target autonomic flexibility is needed in the field, as it may serve as a mechanism for change in parent and child behavior. This would be a rich area of further exploration as there appear to be

differences in physiological presentations of parents of children with ASD compared to those of TD children.

References and Reading

- Davis, N. O., & Carter, A. S. (2008). Parenting stress in mothers and fathers of toddlers with autism spectrum disorders: Associations with child characteristics. *Journal of Autism and Developmental Disorders*, 38(7), 1278–1291.
- Estes, A., Olson, E., Sullivan, K., Greenson, J., Winter, J., Dawson, G., & Munson, J. (2013). Parenting-related stress and psychological distress in mothers of toddlers with autism spectrum disorders. *Brain and Development*, 35(2), 133–138.
- Factor, R. S., Swain, D. M., & Scarpa, A. (2017). Child autism spectrum disorder traits and parenting stress: The utility of using a physiological measure of parental stress. *Journal of Autism and Developmental Disorders*, 1–11. <https://doi.org/10.1007/s10803-017-3397-5>.
- Hastings, P. D., Nuselovici, J. N., Utendale, W. T., Coutya, J., McShane, K. E., & Sullivan, C. (2008). Applying the polyvagal theory to children's emotion regulation: Social context, socialization, and adjustment. *Biological Psychology*, 79(3), 299–306. <https://doi.org/10.1016/j.biopsycho.2008.07.005>.
- Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders*, 61(3), 201–216. [https://doi.org/10.1016/S0165-0327\(00\)00338-4](https://doi.org/10.1016/S0165-0327(00)00338-4).

Further Reading

- Beauchaine, T. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology*, 13(2), 183–214.
- Condy, E. E., Scarpa, A., & Friedman, B. H. (2017). Respiratory sinus arrhythmia predicts restricted repetitive behavior severity. *Journal of Autism and Developmental Disorders*, 47, 2795–2804. <https://doi.org/10.1007/s10803-017-3193-2>.
- Graziano, P., & Derefinko, K. (2013). Cardiac vagal control and children's adaptive functioning: A meta-analysis. *Biological Psychology*, 94(1), 22–37.
- Patriquin, M. A., Hartwig, E., Friedman, B. H., & Porges, S. W., & Scarpa, A. (2019, advanced online publication). Autonomic response in autism spectrum disorder: Relationship to social and cognitive functioning. *Biological Psychology*, 145, 185–197. <https://doi.org/10.1016/j.biopsycho.2019.05.004>.
- Scarpa, A. (2015). Physiological arousal and its dysregulation in child maladjustment. *Current Directions in Psychological Science*, 24, 345–351. <https://doi.org/10.1177/0963721415588920>.

Physiotherapy

► Physical Therapy

Piagetian Stages

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Definition

Jean Piaget advanced the idea that the growth and development of cognitive skills from infancy through adolescence undergoes a series of defined stages that begin with reflexes and eventually develop into the ability to engage in formal logical thinking about abstract concepts. He described four major stages of development: (1) sensorimotor, (2) preoperational, (3) concrete operations, and (4) formal operations.

Historical Background

Arguably the most influential figure in the field of developmental psychology, Jean Piaget authored seminal work on the development of children's intelligence. He posited that cognitive and intellectual changes are the result of development, an active process that the child brings about through interactions with her environment. This process encompasses a series of successive qualitative changes of cognitive structures or schemata created by the child through experience. Piaget's background as a biologist shaped his thinking about the process as a continuous exchange between the individual and the physical world; in other words, “intelligence finds itself entangled in a network of relations between the organism and the environment” (Piaget 1952, p. 19). Piaget's body of work emerged based on extensive, meticulously detailed documentation of his own three children's behavior from birth through adolescence (Piaget 1951, 1952).

The Sensorimotor Stage (Birth to 2 Years)

Piaget (1952) described four stages of cognitive development, the first of which is sensorimotor development. This is the period from birth to approximately 24 months of age that begins with a few reflexes and ends with the appearance of language and symbolic representation of the world. Knowledge in this stage can only be derived from interactions with the physical world, and development proceeds from this exploration. Infants' sensorimotor intelligence proceeds through six increasingly complex stages, each one building on the achievements of the last. In this stage, infants and toddlers do not yet possess the full power of symbolic representation, or language, so must rely on their direct experiences with objects. By around age 2, the child's capacity to represent action in the mind increases, allowing for conceptual thought, thus lessening the dependence on physical and sensory experience.

Substage I: The Use of Reflexes (0–1 Month)

Infants are born with several reflexes, or general action patterns, such as sucking, grasping, and looking. Each reflex constitutes an "organized totality" that includes perceptions, coordinated movements, and a need; it is more than just a "summation of movements" (Piaget 1952, p. 38). These reflexes differ from simple reflexes (i.e., blinking) in that they are modified with experience. At first there is no differentiation among stimuli; for example, newborn infants will suck on a blanket and on a nipple with equal vigor. After several weeks, the infants adjust their sucking to the specific object in their mouth.

Substage II: Primary Circular Reactions (1–4 Months)

When action schemes incorporate new objects, infants move into the second substage of sensorimotor development. In this period, reflexes expand into larger coordinated behaviors, primarily centered on the infants' own bodies. Piaget described systematic thumb sucking and tongue protrusion, looking, hearing and phonation, and prehension in this substage. Out of the single behaviors of grasping objects and sucking objects

grows the integration of these two actions: grasping an object and bringing it to the mouth to suck. The ability to thumb suck at will is one of the *first acquired adaptations* that develops during this period. Turning to find the source of a voice is another primary circular reaction, reflecting coordination between hearing and vision. The term *primary circular reactions* refer to such behaviors. Reactions are primary because they focus on the infants' own bodies, and they are circular because they form "cycles of movements that repeat an interesting sensation discovered by chance" (Muller 2009, p. 208).

Substage III: Secondary Circular Reactions, or the Procedures Destined to Make Interesting Sights Last (4–8 Months)

One of the notable changes in this period is that the child's behavior becomes increasingly oriented toward the environment, beyond her own body, including people, animals, objects, and events. At this time, infants begin to reproduce interesting actions, such as banging, shaking, and waving, especially when these actions cause an interesting visual effect or sound. "Everything thus goes back to movements of legs or feet, arms or hands, and it is these 'circular' movements of prehension which become differentiated in movements directed at shaking, swinging, displacing, rubbing, etc." (Piaget 1952, p. 178). Infants begin to understand that the interesting events are related to their physical activity and thus reproduce the actions. Another characteristic of this period is the shift toward intentional, goal-directed behavior. During this period, infants still lack the concept of object permanence (see section "Substage IV").

Substage IV: Coordination of Secondary Schemata (8–12 Months)

Infants begin to use means to attain ends that are not attainable directly. For example, they will push aside a pillow to reach a toy, or pull a string to obtain an object attached to one end. The main achievement of this period is *object permanence*, the awareness that objects exist when they are no longer in sight. During this period, infants begin to search for objects that disappear, indicating that

they understand that objects still exist after they disappear from view. The fragility of this mental representation, however, is exemplified in the *A-not-B error*. Once infants have searched for a hidden object at one location (A), when the location is moved (B) they continue to search for it at location A. Not until around the first birthday is the infant able to inhibit search at the previous location and look for the object at the new location.

Substage V: Tertiary Circular Reactions, or the Discovery of New Means Through Active Experimentation (12–18 Months)

Beginning at 12 months of age, infants begin to explore their worlds more actively, “experimenting” with objects, intent on seeing how they behave in new situations. Once making such discoveries, they are repeated, but these *tertiary circular reactions* differ from the previous kinds in that the infant varies the actions slightly each time. In this stage, rather than applying the same schemata to problems, they now find new means through trial and error. In contrast to secondary circular reactions, in which infants attempt to recreate a certain event, tertiary circular reactions aim to “ferret out new phenomena” (Piaget 1952, p. 274) in the process.

Substage VI: Invention of New Means Through Mental Combinations (18–24 Months)

In this final stage, the child begins to invent as well as discover. Rather than relying on trial-and-error experimentation, the child begins to be able to devise solutions to simple problems through representation (thinking), independent of immediate experience. In effect, the experimentation happens in thought. This development of mental representation fosters another hallmark of this period, *deferred imitation*, whereby children form mental representations of events so enduring that they can repeat others’ behaviors hours or even days after it was first observed.

The Preoperational Stage (Ages 2–7)

During the preoperational stage, the child becomes progressively more able to internally represent events. One manifestation of this is

symbolic play. In such play, a child might take a piece of bread and play with it as if it were an airplane. Often, but not always, these symbols resemble their targets visually. Piaget recognized the value of symbolic play as “. . .the creation of symbols at will in order to express everything in the child’s life experience that cannot be formulated and assimilated by means of language alone” (Piaget and Inhelder 1969, p. 61). Spoken language, itself symbolic, begins to develop more fully during this stage, from one-word utterances to two-word phrases followed by short sentences and finally a gradual acquisition of grammatical structures. Children’s drawings during this period progress from scribbles to realistic representations of objects.

Piaget described two specific limitations to children’s cognitive development in this phase, *egocentrism* and *centration*. Preschool children still possess notable *egocentrism*, or the perception of the world strictly from one’s own point of view. Illustrating this concept is Piaget’s three mountains task (Piaget and Inhelder 1969), wherein 4-year-olds sat at a table in front of a model of three mountains while a doll was placed in a chair across them. The children were shown photographs of the model from different angles and were asked which one the doll would see, for which most children chose the picture depicting the scene as it looked to them. The ability to take another’s perspective does not emerge in most children until the age of 6. Egocentric communication and thinking is also prominent at these ages. In *centration*, children focus on a perceptually salient dimension of a problem at the expense of other less salient but equally important dimensions. For example, Piaget presented children with two trains running on parallel tracks in the same direction, but one ahead of the other. When they stopped, Piaget asked which train traveled for the longer time/the longer distance/at the faster speed. For all three questions, most of the 4- and 5-year-olds answered that the train that was farther ahead on the track was the one that traveled longer/farther/faster, regardless of the actual duration, length, or speed of the journey. Not until several years later do children consider multiple dimensions of a problem.

The Concrete Operations Stage (Ages 7–12)

It is not until about age 7 that children begin to reason logically about physical phenomena. Typifying the abilities of this stage of development is the concept of *conservation*. The basic premise of this concept is that changing the appearance of objects does not change their key properties, such as amount or material. Below the age of 7, children typically cannot hold one dimension constant while it changes in another dimension. Problems of this kind are tested in three phases. First, for example, the child is shown two rows of eight coins parallel to each other, and then watches while one row is stretched out. In the third phase, she is asked if the number of items is the same or different. Before the concrete operations stage, the child will typically say that this longer row now contains more coins, although she observed the process and saw that no new coins were added. By centering on the perceptual feature of length and ignoring the transformation, she mistakenly believes that one row contains more coins. In addition to the conservation of number, there exists conservation of liquid quantity, conservation of area and mass, and conservation of volume. Understanding such transformations in all of these situations is mastered in the concrete operations stage. As the name suggests, however, understanding is limited to concrete situations, and does not extend to more abstract or hypothetical situations. Through the development of logic, advances are made during this period in the development of seriation and categorization, forming the basis for number concepts, as well as the development of moral concepts such as laws, rules, lying, intentions, and justice.

The Formal Operations Stage (Age 12 and Beyond)

Formal operational thinking involves the development of abstract reasoning and logic to solve a variety of problems. Unlike the other three stages, Piaget believed that formal operations are not universal; not all adolescents fully attain the capabilities of this stage. While concrete thought is limited to tangible concrete problems of the present, formal operations involve propositions, hypothetical problems, or problems in the future.

During this stage children can also become aware of their own feelings and thoughts; in other words, they become capable of introspection. Formal thought has three types of structures: hypothetical-deductive reasoning, scientific-inductive reasoning, and reflective abstraction.

Hypothetical-deductive reasoning involves reasoning from premises which are hypotheses rather than verified facts. Problems of this sort are exemplified by questions like the following: “The car is smaller than the SUV and the SUV is smaller than the truck. Is the car smaller than the truck?” The child who can work this out in her head as opposed to needing objects or drawings to illustrate the problem is using deductive reasoning. One can also reason about hypotheses based on false premises and still come to a logical conclusion.

Scientific-inductive reasoning is reasoning from specific facts to general conclusions, much as scientists do. At this stage, children are capable of forming hypotheses, experimenting, controlling variables, and drawing conclusions in a systematic fashion. One feature of scientific reasoning is the ability to think about several different variables at the same time; another feature is the ability to reason systematically about all possible outcomes of a situation. The pendulum problem requires such reasoning. Children are presented with a pendulum consisting of a weight at the end of a string, set in motion. They are then provided with strings of varying lengths and different weights, and are asked to determine what controls the pendulum’s rate of movement. Children often consider important the length of the string, the weight at the end of the string, the force in setting the pendulum in motion, and the height from which the weight is dropped. The rate of oscillation, however, is controlled solely by the length of the string. The task is to isolate this factor and be able to exclude the others. Children at the level of formal operations work through this in a systematic fashion, exploring all possibilities and finally determining that a single factor influences the rate of motion.

Reflective abstraction is the key mechanism of the construction of new knowledge. Through internal thought or reflection, one can generate

novel information, particularly logical relationships. Understanding of analogies is a prototypical example of this mechanism as it requires construction of relationships between members that comprise the analogy (Wadsworth 1984, p. 146). What is needed for complete mastery of analogical reasoning is not only which members fit together (this could be solved in an associative manner) but also why they do and the awareness of the relationship between two pairs of items. Reasoning about such a relationship goes beyond what is observable, an ability that does not arise until this stage of development.

Current Knowledge

Piagetian Stages and Autism

(Note: For a review of research on the sensorimotor stage in autism, please see the entry ► [“Sensorimotor Development”](#)).

Preoperational

Of all the skills described in Piaget’s body of work, the one perhaps most heavily studied in children with autism has been symbolic play. Wing and Gould (1979) early large-scale study of children with suspected autism even characterized the syndrome by deficits in imagination in addition to the core features of impaired communication and social interaction. Impairments in pretend play have been noted in children with autism of various ages (Bartak et al. 1975; Demyer et al. 1967; Doherty and Rosenfeld 1984) and also when compared to developmentally matched (Rutherford and Rogers 2003; Sigman and Ungerer 1984; Stone et al. 1990; Wing et al. 1977) or language-matched (Baron-Cohen 1987; Charman et al. 1997; Jarrold et al. 1996; Libby et al. 1998; Riguet et al. 1981) controls using a variety of metrics of pretend play. The few studies that have reported no imaginative play deficits may have done so due to floor effects in the comparison groups (e.g., Lewis and Boucher 1988). Another preoperational skill, visual perspective-taking does not seem to be impaired in children with autism (Hobson 1984; Reed and Peterson 1990; Tan and Harris 1991; but

see Yirmiya et al. 1994) or adolescents with Asperger syndrome (David et al. 2009).

Concrete Operations

Relatively few studies have been conducted on conservation and seriation skills; adolescents and adults with autism and intellectual impairment were found to perform at the same level or better than intellectually impaired controls but worse than mental-age-matched typically developing children (Yirmiya and Shulman 1996). In contrast, categorization abilities of children with autism have garnered much interest in recent years. Results of these studies have been mixed, depending on the type of categorization task employed. Generally, children with autism do not appear to be impaired in categorization tasks that are relatively simple, that is, using relatively simple sorting principles such as color, size, or shape (Sigman and Ungerer 1984; Tager-Flusberg 1985). As tasks become more complex and involve more atypical exemplars, for example, categorizing a groomed French poodle among other dogs, individuals with autism show impairments in terms of accuracy or reaction time (Gastgeb et al. 2006; Klinger and Dawson 1995; Shulman et al. 1995).

Formal Operations

Analogical reasoning has been studied in high-functioning children and adolescents with autism, and was found to be intact both for picture analogies (Morsanyi and Holyoak 2009; Scott and Baron-Cohen 1996) and scene analogies with thematic content (Morsanyi and Holyoak 2009). Furthermore, no group differences were observed in a task of inferential transitive reasoning (Scott and Baron-Cohen 1996). A number of studies, however, have reported deficits in conceptual reasoning in high-functioning adolescents and adults with autism as measured by various standardized tests (e.g., the Verbal Reasoning domain of the Stanford-Binet; Carpentieri and Morgan 1994; Minshew et al. 1997).

See Also

- [Object Permanence](#)
- [Sensorimotor Development](#)

References and Reading

- Baron-Cohen, S. (1987). Autism and symbolic play. *British Journal of Developmental Psychology*, 5(2), 139–148.
- Bartak, L., Rutter, M., & Cox, A. (1975). A comparative study of infantile autism and specific developmental receptive language disorder: 1. The children. *British Journal of Psychiatry*, 126, 127–145.
- Carpentieri, S. C., & Morgan, S. B. (1994). Brief report: A comparison of patterns of cognitive functioning of autistic and nonautistic retarded children on the Stanford-Binet, fourth edition. *Journal of Autism and Developmental Disorders*, 24(2), 215–223.
- Charman, T., Swettenham, J., Baron-Cohen, S., Cox, A., Baird, G., & Drew, A. (1997). Infants with autism: An investigation of empathy, pretend play, joint attention, and imitation. *Developmental Psychology*, 33(5), 781–789.
- David, N., Aumann, C., Bewernick, B. H., Santos, N. S., Lehnhardt, F.-G., & Vogeley, K. (2009). Investigation of mentalizing and visuospatial perspective taking for self and other in Asperger syndrome. *Journal of Autism and Developmental Disorders*, 40(3), 290–299.
- Demyer, M. K., Mann, N. A., Tilton, J. R., & Loew, L. H. (1967). Toy-play behavior and use of body by autistic and normal children as reported by mothers. *Psychological Reports*, 21(3), 973–981.
- Doherty, M. B., & Rosenfeld, A. A. (1984). Play assessment in the differential diagnosis of autism and other causes of severe language disorder. *Journal of Developmental and Behavioral Pediatrics*, 5(1), 26–29.
- Gastgeb, H. Z., Strauss, M. S., & Minshew, N. J. (2006). Do individuals with autism process categories differently? The effect of typicality and development. *Child Development*, 77(6), 1717–1729.
- Hobson, R. P. (1984). Early childhood autism and the question of egocentrism. *Journal of Autism and Developmental Disorder*, 14(1), 85–104.
- Jarrold, C. (2003). A review of research into pretend play in autism. *Autism*, 7(4), 379–390.
- Jarrold, C., Boucher, J., & Smith, P. K. (1996). Generativity deficits in pretend play in autism. *British Journal of Developmental Psychology*, 14(3), 275–300.
- Klinger, L. G., & Dawson, G. (1995). A fresh look at categorization abilities in persons with autism. In E. Schopler & G. Mesibov (Eds.), *Learning and cognition in autism* (pp. 119–136). New York: Plenum Press.
- Lewis, V., & Boucher, J. (1988). Spontaneous, instructed and elicited play in relatively able autistic children. *British Journal of Developmental Psychology*, 6(4), 325–339.
- Libby, S., Powell, S., Messer, D., & Jordan, R. (1998). Spontaneous play in children with autism: A reappraisal. *Journal of Autism and Developmental Disorders*, 28(6), 487–497.
- Minshew, N. J., Goldstein, G., & Siegel, D. J. (1997). Neuropsychologic functioning in autism: Profile of a complex information processing disorder. *Journal of the International Neuropsychological Society*, 3(04), 303–316.
- Morsanyi, K., & Holyoak, K. J. (2009). Analogical reasoning ability in autistic and typically developing children. *Developmental Science*, 13(4), 578–587.
- Muller, U. (2009). Infancy. In U. Müller, J. I. M. Carpendale, & L. Smith (Eds.), *The Cambridge companion to Piaget*. New York: Cambridge University Press, Cambridge Collections Online.
- Piaget, J. (1951). *Play, dreams, and imitation in childhood*. New York: W. W. Norton.
- Piaget, J. (1952). *The origins of intelligence in children*. New York: International Universities Press.
- Piaget, J., & Inhelder, B. (1969). *The psychology of the child*. New York: Basic Books.
- Reed, T., & Peterson, C. (1990). A comparative study of autistic subjects' performance at two levels of visual and cognitive perspective taking. *Journal of Autism and Developmental Disorders*, 20(4), 555–567.
- Riguet, C. B., Taylor, N. D., Benaroya, S., & Klein, L. S. (1981). Symbolic play in autistic, Down's, and normal children of equivalent mental age. *Journal of Autism and Developmental Disorders*, 11(4), 439–448.
- Rutherford, M. D., & Rogers, S. J. (2003). Cognitive underpinnings of pretend play in autism. *Journal of Autism and Developmental Disorders*, 33(3), 289–302.
- Scott, F. J., & Baron-Cohen, S. (1996). Logical, analogical, and psychological reasoning in autism: A test of the Cosmides theory. *Development and Psychopathology*, 8(01), 235–245.
- Shulman, C., Yirmiya, N., & Greenbaum, C. (1995). From categorization to classification: A comparison among individuals with autism, mental retardation, and normal development. *Journal of Abnormal Psychology*, 104(4), 601–609.
- Sigman, M., & Ungerer, J. A. (1984). Cognitive and language skills in autistic, mentally retarded, and normal children. *Developmental Psychology*, 20(2), 293–302.
- Stone, W. L., Lemanek, K. L., Fishel, P. T., Fernandez, M. C., & Altemeier, W. A. (1990). Play and imitation skills in the diagnosis of autism in young children. *Pediatrics*, 86(2), 267–272.
- Tager-Flusberg, H. (1985). Basic level and superordinate level categorization by autistic, mentally retarded, and normal children. *Journal of Experimental Child Psychology*, 40(3), 450–469.
- Tan, J., & Harris, P. J. (1991). Autistic children understand seeing and wanting. *Development and Psychopathology*, 3(02), 163–174.
- Ungerer, J. A., & Sigman, M. (1987). Categorization skills and receptive language development in autistic children. *Journal of Autism and Developmental Disorders*, 17(1), 3–16.
- Wadsworth, B. (1984). *Piaget's theory of cognitive and affective development*. New York: Longman.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9(1), 11–29.

Wing, L., Gould, J., Yeates, S. R., & Brierley, L. M. (1977). Symbolic play in severely mentally retarded and in autistic children. *Journal of Child Psychology and Psychiatry*, 18(2), 167–178.

Yirmiya, N., & Shulman, C. (1996). Seriation, conservation, and theory of mind abilities in individuals with autism, individuals with mental retardation, and normally developing children. *Child Development*, 67(5), 2045–2059.

Yirmiya, N., Sigman, M., & Zacks, D. (1994). Perceptual perspective-taking and seriation abilities in high-functioning children with autism. *Development and Psychopathology*, 6(02), 263–272.

PIAT-R

- [Peabody Individual Achievement Test, Revised](#)

PIAT-R/NU

- [Peabody Individual Achievement Test, Revised](#)

PIC

- [Personality Inventory for Children](#)

Pica

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Synonyms

[Feeding disorder](#); [Scavenging](#)

Short Description or Definition

Pica is an eating disorder narrowly defined as the ingestion of nonfood items or nonnutritive items (e.g., clay, hair, paint chips, paperclips, coins)

which has been present for at least 1 month. The word “pica” comes from the Latin word for the magpie bird (*Pica pica*) known for eating anything. Currently, pica is included as one of three subtypes under Feeding and Eating Disorders of Infancy and Early Childhood in DSM-IV-TR (APA, 2000). A broader definition of pica has been used in the literature to include the eating of food from inappropriate places (e.g., out of the trash can, off floor). While it is developmentally appropriate for children under the age of 24 months to mouth and potentially swallow non-edible objects, it is considered pathological when this behavior persists into older years (Baltrop 1966; Stiegler 2005). Individuals with intellectual disability (ID) and autism spectrum disorders (ASD) often present with comorbid pica that ranges from a mild, occasional behavior to life threatening. In light of the potential serious consequences, screening at minimal and further evaluation and treatment of pica as indicated should be considered in the care of individuals with ASD.

Categorization

Pica items or targets are strikingly diverse and include household products, small toy parts, organic materials, cosmetics, and art supplies. Table 1 lists a variety of pica subtypes that have been identified and specifically named, all ending in “-phagia.” Other pica targets are not formally

Pica, Table 1 Subtypes of pica

Subtype	Target
Acuphagia	Sharp objects
Amylophagia	Laundry starch
Cautopyreiophagia	Burnt matches
Coprophagia	Human or animal feces
Foliophagia	Leaves, grass, acorns, pinecones
Geomelophagia	Raw potatoes
Geophagia	Dirt, clay, sand
Lignophagia	Wood, bark, twigs
Lithophagia	Rocks, gravel, pebbles
Pagophagia	Ice, freezer frost
Plumbophagia	Paint chips
Tobaccophagia	Cigarette butts
Trichophagia	Hair

labeled (e.g., rubber gloves, coins, buttons, insects, pillow stuffing, candles, toiletries) but have been reported in individuals with ID and/or ASD (Stiegler 2005).

Epidemiology

Pica has been found in a wide range of cultural groups across the world (Ali 2001). However, we are unaware of any large-scale studies on the prevalence of pica in the general population. Pica is generally thought to often go unrecognized and underreported (Ali 2001). Prevalence rates reported for pica specifically in individuals with ASD with ID have ranged from 6% to 25%, but samples have included primarily institutionalized samples (Ali 2001; Stiegler 2005). Pica is one of the more commonly found eating disorders in ASD, but future studies are needed to understand the prevalence of pica in ASD, particularly in individuals with ASD but without co-occurring ID.

Natural History, Prognostic Factors, and Outcomes

Pica has been reported throughout the world and, in fact, is an accepted practice in some cultures. Geophagia, the ingestion of dirt, clay, and soil, has specifically been associated with pregnancy in some societies, including regions of Southern United States as well (Ali 2001; Stiegler 2005). Pica has also been observed in the presence of hunger, gastrointestinal distress, and micronutrient deficiencies such as iron and zinc (Young 2010). Conversely, pica may result in micronutrient deficiencies (Ali 2001). There has been limited conjecture that pica is associated with alteration in the dopamine system (Singh et al. 1994).

Gender differences of pica have not been reported in young children, but pica is rare in males *without* ID. Pica becomes more common in those individuals with more severe ID. ASD has been more frequently associated with pica than other developmental differences such as Down syndrome (Kinnell 1985). Little is known about the long-term prognosis and outcome of

pica in ASD. Although long term, improvement management in an institutionalized setting has been reported for individuals with ID (Williams et al. 2009), pica has been noted to be resistant to treatment (Kern et al. 2006).

Clinical Expression and Pathophysiology

Individuals with pica may be very specific about chosen pica targets (i.e., seek out only metal objects) or very indiscriminate (ingesting any nonfood items within their environment). Likewise, the pica behavior may seem to have a compulsive quality whereby individuals spend extraordinary time and effort in searching out pica targets. These individuals may engage in disruptive, aggressive behaviors when pica is thwarted or interrupted (Bogart et al. 1995). This driven nature of pica has led to speculation that pica may be an obsessive-compulsive disorder. In other individuals, the pica behavior may occur in a more random fashion whereby the opportunity presents itself (e.g., cigarette butts are left in an open container). In those with ASD who have high rates of sensory differences, there has been considerable speculation that pica may be a form of seeking sensory input.

Pica is seen in a wide range of children with ASD but is believed to continue into adult hood, at least in individuals with co-occurring severe ID. Individuals in institutional setting are generally believed to present with more pica than other populations. However, as already mentioned in a previous section, our knowledge of the course of pica in ASD specifically is limited by the lack of large-scale, rigorous studies.

Pica presents with a variety of subsequent health risks, including toxicity, obstructions, perforations, and even death (Bell and Stein 1992; McLoughlin 1988; Rapp et al. 2001). Sharp or bulky objects that have been ingested have the potential to cause perforations or blockages of the respiratory or gastrointestinal tracts. Persons who eat paint chips are at risk of lead poisoning. Parasites that live in dirt or clay can cause infections if eaten. Patients who have eaten an irregular amount of a nonfood substance risk malnutrition

and the under- or overconsumption of calories. Dental health is also threatened with repeated chewing hard or abrasive items.

In addition to the health consequences of pica, there are adverse social consequences. This is particularly for individuals who engage in more “unclean” pica such as ingestion of feces, trash, and cigarette butts. Caregivers and peers may be likely to limit their interactions to avoid the unpleasantness associated with the pica behavior.

Evaluation and Differential Diagnosis

Screening and Diagnostic Evaluation

Screening for pica within the context of any evaluation for ASD is well warranted given the high rate of pica in individuals with ASD. This screening may simply be systematic interview questions directed toward parents and other caregivers as part of an evaluation for ASD. Screening questions for pica should also be included in intake assessments for individuals with ASD of all ages. That is, any educational, therapeutic, and other programs for individuals with ASD should screen for pica. If an individual is screened to be positive for pica, a more comprehensive assessment of pica should be pursued. This would first involve interviewing those involved in the care of the individual with ASD regarding observed pica behavior. When possible, an interview with the individual with ASD regarding pica practices should be pursued.

Medical and Nutritional Evaluation

A thorough medical history and evaluation should be conducted when pica is present. Medical testing to rule out the presence of parasites, rule out leading poisoning or other toxins, and to rule out nutritional deficits such as iron and zinc deficiencies should be considered. Additional nutritional testing may be justified when pica behavior is suspected of resulting in poor nutrition. Imaging studies may be warranted to determine the presence of ingested pica item(s). More extensive gastrointestinal work-ups may be necessary when further complications are suspected.

Behavioral Evaluation

A behavioral functional assessment is important in the treatment planning of pica, as it is important to distinguish what environmental factors may be maintaining or at least influencing the pica behavior. Functional assessments typically involve indirect methods such as starting with a focused interview of caretakers regarding situations where pica is observed and then not observed. The latter is just as important in treatment planning as determining when pica is most likely to occur. Functional analogue observations to specifically assess for pica would include safe replications of pica targets (“paint chips” made of cornstarch and water spread and dried on wax paper) along with the manipulation of common antecedent and consequence contingencies. Antecedents manipulated may be the presentation of a recent snack/meal or not, the presence of others (to determine whether there is a social component or not), the presence of toys or not (to determine whether pica is related to the impoverishment of environment), and the presence of appropriate food items (to determine whether pica is a substitution when food is not available). Consequence manipulation could include the attention of an adult’s attention for engaging in pica behavior, the access to a preferred item/activity contingent on pica, or the ability to escape a situation contingent on pica. Previous, albeit limited, work has suggested that pica may be reinforced by automatic reinforcement (Kern et al. 2006; Piazza et al. 2002). That is, the behavior of engaging in pica is reinforcing in and of itself or stated in less behavioral terms as sensory pleasing to the individual. While conducting such functional analyses whereby antecedent and consequence variables are experimentally manipulated is widely recommended prior to designing behavioral treatments, the use of functional analysis in the evaluation of pica has a limited research base to date (Kern et al. 2006; Mace and McKnight 1986; Piazza et al. 2002; Rapp et al. 2001). Nonetheless, a thorough behavioral assessment including functional analyses is accepted to be important in the development of empirically derived behavioral treatments that are least intrusive.

Additionally important for treatment planning is a preference assessment whereby reinforcers and punishers are empirically derived. Methods to determine possible reinforcers include indirect measures such as completion of a reinforcement inventory by a caregiver to direct measures (Hagopian et al. 2004). Direct measures have included observations of an individual approach to a stimulus when presented, duration of stimulus engagement, and the contingent presentation of stimuli to observe subsequent changes in rate of a chosen behavior (Fisher et al. 1994; Hagopian et al. 2004).

Treatment

The treatment of pica in ASD has a very limited empirical. As with other areas of the pica literature, there have been few, if any, large-scale treatment studies and the studies in the literature almost all combine individuals with ASD with those with ID and other developmental disabilities. Moreover, treatment studies have been nearly all either case reports or single subject studies with a small number of participants.

Pharmacological Treatment

Some very limited research has supported the use of medication, such as selective serotonin reuptake inhibitors (SSRIs) to treat pica (Stiegler 2005). Anecdotal report has suggested positive outcomes in children whose pica was treated with stimulant medications (Barrett 2008). At present, our knowledge base does not support the use of medication to target specifically pica.

Nutritional Treatments

Nutritional deficiencies have been linked to pica, especially iron and zinc. As discussed elsewhere in this chapter, there remains debate about whether nutritional deficiencies of these minerals may cause a person to engage in pica (eat iron- or zinc-containing substances), or whether that the ingestion of certain pica items results in subsequent mineral deficiencies. When iron and zinc are demonstrated to be deficit, supplementation is

warranted, and some have suggested pica behaviors may abate, but there is lack of empirical support for this.

Behavioral Treatment

Behavioral approaches have been the most common for individuals with pica and ASD and ID. The specific behavioral intervention should be based on the functional assessment with respect to environmental variables maintaining the pica. Such an assessment is described in a previous section on the evaluation of pica. Behavioral treatment procedures for pica may be categorized as preventive or antecedent strategies, reinforcement consequences, punishment consequences, and specific teaching procedures of a new skill. More likely than not, treatment will require a treatment package to include several behavioral procedures.

Preventive and antecedent strategies have been used to limit access to potential pica items from the environment. This may include increased straightforward approaches such as regularly vacuuming, storing trash cans away from the child, and timely waste disposal. Environmental enrichment as a preventive approach has also been used as well as teaching appropriate discrimination of what to place in the mouth (Johnson et al. 1994). Other prevent approaches have attempted to identify alternative, safe items which may be mouthed, chewed on in lieu of the pica item. Such an approach is appropriate when pica is suspected to be motivated by a need for sensory input, or in more behavioral terms, pica is automatically reinforced. Protective devices have also been used as a preventive approach. For example, arm splints and a facemask to prevent pica have been used. Such restrictive procedures should be used only in severe pica and as a temporary measure as a treatment program is being developed. Further, use of such procedures may be prohibited in some settings depending on local regulations.

In addition to prevention and antecedent strategies, reinforcement procedures have also been implemented to reduce pica behavior. These have included differential reinforcement procedures (differential reinforcement of other

behaviors, lower rates of pica behavior, or an alternative behavior). Noncontingent reinforcement has also been used. Reinforcement procedures presume reinforcers have been identified as part of the evaluation process which may compete from the reinforcing value of engaging in the pica behavior itself.

While a least restrictive approach should be considered, the seriousness of pica may justify consideration of punishment procedures to suppress the pica behavior. The treatment literature on the use of punishment includes a disproportionate number of individuals with severe and profound ID who had potentially life-threatening pica. Punishment procedures have included timeout, contingent presentation of aversive stimuli, overcorrection, positive practice, and visual screening, as well as contingent placement of protective equipment or other physical restraint (Bell and Stein 1992; McAdam et al. 2004). The consideration of the use of a punishment procedure should take into account the response of other approaches, the seriousness of the pica behaviors, and, again, local regulations regarding use of punishment procedures and consent requirements.

In addition to these behavioral procedures, teaching the individual to discriminate between edible and inedible items has been undertaken as a treatment approach to pica but in combination with a punisher (Bogart et al. 1995; Johnson et al. 1994). How the discrimination skill taught depends on the participant's level of development. The teaching of a replacement or alternative behavioral response behavior such as giving a pica to an adult and reinforcement for this has been described (Kern et al. 2006).

See Also

- ▶ [Applied Behavior Analysis \(ABA\)](#)
- ▶ [Autism](#)
- ▶ [Autistic Disorder](#)
- ▶ [Behavior Modification](#)
- ▶ [Eating Disorders](#)
- ▶ [Feeding Problems](#)
- ▶ [Functional Analysis](#)

- ▶ [Functional Behavior Assessment](#)
- ▶ [Medical Conditions Associated with Autism](#)
- ▶ [Obsessive-Compulsive Disorder \(OCD\)](#)
- ▶ [Operant Conditioning](#)
- ▶ [Overcorrection](#)
- ▶ [Reinforcement](#)

References and Reading

- Ali, Z. (2001). Pica in people with intellectual disability: A literature review of aetiology, epidemiology and complications. *Journal of Intellectual and Developmental Disability*, 26, 205–215.
- American Psychological Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. Text revision). Washington, DC: Author.
- Baltrop, D. (1966). The prevalence of pica. *American Journal of Diseases in Children*, 112, 116–123.
- Barrett, R. P. (2008). Pica: Toward understanding a complication condition. *The Brown University Child and Adolescent Behavior Letter*, 24(6), 5–6.
- Bell, K. E., & Stein, D. M. (1992). Behavioral treatments for pica: A review of empirical studies. *International Journal of Eating Disorders*, 11(4), 377–389.
- Bogart, L., Piersel, W., & Gross, E. (1995). The long-term treatment of life-threatening pica: A case study of a woman with profound mental retardation living in an applied setting. *Journal of Developmental and Physical Disabilities*, 7, 39–50.
- Fisher, W., Piazza, C. C., Bowman, L. G., Hagopian, L. P., & Langon, N. A. (1994). Empirically derived consequences: A data-based method for prescribing treatment for destructive behavior. *Research in Developmental Disabilities*, 15, 133–149.
- Hagopian, L. P., Long, E. S., & Rush, K. S. (2004). Preference assessment procedures for individuals with developmental disabilities. *Behavior Modification*, 28, 668–677.
- Johnson, C. R., Hunt, F. M., & Siebert, M. J. (1994). Discrimination training in the treatment of pica and food scavenging. *Behavior Modification*, 18, 214–229.
- Kern, L., Starosta, K., & Adelman, B. E. (2006). Reducing pica by teaching children to exchange inedible items for edibles. *Behavior Modification*, 30(2), 135–158.
- Kinnell, H. G. (1985). Pica as a feature of autism. *British Journal of Psychiatry*, 147, 80–82.
- Mace, F. C., & McKnight, D. (1986). Functional analysis and treatment of severe pica. *Journal of Applied Behavior Analysis*, 19, 411–416.
- Matson, J. L., & Bamburg, J. W. (1999). A descriptive study of pica behavior in persons with mental retardation. *Journal of Developmental and Physical Disabilities*, 11, 353–361.
- McAdam, D. B., Sherman, J. A., Sheldon, J. B., & Napolitano, D. H. (2004). Behavioral interventions to

- reduce the pica of persons with developmental disabilities. *Behavior Modification*, 28, 45–72.
- McLoughlin, J. (1988). Pica as a cause of death in three mentally retarded persons. *British Journal of Psychiatry*, 152, 842–845.
- Piazza, C. C., Fisher, W. W., Hanley, G. P., LeBlanc, L. A., Worsdell, A. S., Lindauer, S. E., et al. (1998). Treatment of pica through multiple analyses of its reinforcing functions. *Journal of Applied Behavior Analysis*, 31, 165–189.
- Piazza, C. C., Roane, H. S., Keeney, K. M., Boney, B. R., & Abt, K. A. (2002). Varying response effort in the treatment of pica maintained by automatic reinforcement. *Journal of Applied Behavior Analysis*, 35, 233–246.
- Rapp, J. T., Dozier, C. L., & Carr, J. E. (2001). Functional assessment and treatment of pica: A single case experiment. *Behavioral Interventions*, 16, 111–125.
- Singh, N. N., Ellis, C. R., Crews, W. D., & Singh, Y. N. (1994). Does diminished dopaminergic neurotransmission increase pica? *Journal of Child and Adolescent Psychopharmacology*, 4, 93–99.
- Stiegler, L. N. (2005). Understanding pica behavior: A review for clinical and education professionals. *Focus on Autism and Other Developmental Disabilities*, 20, 27–38.
- Williams, D. E., Kirkpatrick-Sanchez, S., Enzinna, C., & Dunn, J. (2009). The clinical management and prevention of pica: A retrospective follow-up of 41 individuals with intellectual disabilities and pica. *Journal of Applied Research in Intellectual Disabilities*, 22, 210–215.
- Young, S. L. (2010). Pica in pregnancy: New ideas about an old condition. *Annual Review of Nutrition*, 30, 403–422.

Pictorial Cues/Visual Supports (CR)

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Definition

Pictorial cues or visual supports as used in language and cognitive therapy for individuals with autism are defined as any aid such as picture schedules, picture cards, symbols, communication boards, posters, models, visual scene displays, or videos that present information

visually. As suggested in the previous list, visual supports can be very low- or no-tech such as in picture communication boards or can be used in high-tech communication devices such as visual scene displays. Compensating for difficulties with attention, organization, sequencing, and processing of verbal information can be facilitated with visual supports. The most commonly used visual supports will be described here. They are visual schedules, communication boards, the picture exchange communication system, and visual scene displays. Communication boards are discussed as another entry in this encyclopedia and so will not be discussed in detail here. Suffice it to say that communication boards are used to augment the spoken communication of individuals with ASDs.

Visual schedules or schedule systems (Mesibov et al. 2002) or timetables (Bloomberg 1996) are designed to make an individual's agenda explicit. They have been used with children with autism spectrum disorders (ASDs) to provide them with a sense of security and consistency. Between-activity visual schedules are those which allow the user to see the activities that will be completed during any length of time such as a therapy session or an entire day. Within-activity visual schedules show the steps necessary to complete a task and are designed to help an individual become more independent.

The picture exchange communication system (PECS) was developed by Bondy and Frost to aid in the functional communication of nonverbal children with ASDs. There is a large literature on this communication system, and so a brief description of it will be provided here. PECS is a multiphase system that begins with requesting. The child is first taught to exchange a picture symbol for an object that is motivating and rewarding. This occurs through hand-over-hand assistance from another adult. The second phase of PECS training requires the child to be persistent in his/her requesting. The communication partner and the picture symbol are moved so the child must seek out the picture and seek out the communication partner to request a desired item. In the third phase of PECS, the child is taught to discriminate between picture

symbols. Phases four through six of PECS training involves helping the child combine symbols together to make sentences, respond to questions, and comment with the picture symbols. See Bondy and Frost (1994) for a more detailed description of PECS.

Visual scene displays are often whole pictures that represent an activity, place, or situation. They are useful for providing context for a communicative intention and aid in organization. For example, a scene of a park can be used for a child with autism. The picture can be either a real picture of the park the child frequents every afternoon, or a cartoon or line drawing of a generic park depending on the symbol transparency needs of the child. Given the picture, the child can request the swings, slide, or merry-go-round or the child can comment on the other children playing on the playground equipment. Drager et al. (2009) make the comment that visual scene displays are an interesting way to organize an augmentative and alternative communication system for individuals with ASDs because they allow for information to be presented as a whole and not in fragmented vocabulary items.

Historical Background

One of the earliest studies that involved the use of picture symbols with children with ASDs was conducted by Lancioni (1983). Two children were exposed to line drawings and were taught to interpret the line drawings and follow directions with them. Lancioni found that the children were successfully able to comprehend the symbols and accurately follow directions and that their ability to follow directions was generalized to other line drawings to which they had not previously been exposed. The use of picture symbols and visual supports for individuals with autism has grown to such an extent that at the time of this writing, when the words “picture symbol” and “autism” are fed into a search engine such as Google Scholar[®], nearly 4,500 results appear just since the year 2005.

Rationale or Underlying Theory

Hermelin and O’Conner (1970) were the first to state that some children with autism process visuospatial information better than auditory information. Additionally, it has been found that the memory of children with autism is superior for visual information compared to auditory information (Prior and Chen 1976). Finally, research on the attention of children with autism supports the notion that children with autism have difficulty selecting the most salient features of a stimulus (Frith and Baron-Cohen 1987). Overall, individuals with autism have been shown to have more difficulty with verbal information compared to visual information (Hodgdon 1995). Temple Grandin (1995) is an individual with autism. She states that she thinks in still pictures and in “videos” and that language-based information is difficult for her to process and retain. The use of other forms of augmentative communication for children with autism seems logical based on the strengths that children with autism possess. The use of sign language relieves the burden of having to process verbal information and builds on the strength of processing visuospatial information. The memory difficulties of children with autism can be alleviated by using picture communication symbols which do not require the child to rely on their own memories to create a message. If using picture symbols, children can attend to the symbols for as long as necessary to comprehend the information, alleviating the difficulties related to attention needed for verbal information transfer.

Although the use of picture symbols and visual supports are well supported by research, many laypersons fear that the use of these strategies will not allow oral speech to develop. The apprehension revolves around the idea that the individual with autism will use the visual supports as a crutch and will not need to use speech. In fact, the opposite has been shown to occur. Lloyd and Kangas (1994) and Ronski and Sevcik (1996) agree that visual supports serve to lower the linguistic and cognitive demands of speech production and reduce the stress associated with speech,

thereby facilitating speech development. Mirenda (2003) argues that the theory of automatic reinforcement is at play. When an individual with ASDs requests an item with a picture symbol, the communication partner verbally labels the item. The picture symbol and the verbal label occur so frequently together, and both are reinforced by the item; production of the verbal label and use of the symbol should increase.

Goals and Objectives

Like much of the field of AAC, the goal for the use of picture symbols and visual supports is functional communication. Because speech production may not be possible for all individuals with ASDs, delaying the introduction to other forms of communication can be damaging to the language, cognitive, and emotional development of these individuals and may hinder their social development as well.

Treatment Participants

Any individual who is unable to communicate to the level expected for their age is a candidate for augmentative and alternative communication of any form (Beukelman and Mirenda 2005). Therefore, because communication impairments are a hallmark of autism spectrum disorders (ASDs) (Mirenda 2009), many individuals with ASDs are candidates for a total communication approach.

Treatment Procedures

Some of the picture systems mentioned have very specific treatment procedures (see the PECS description). Others are more specific to AAC, and treatment procedures that are appropriate for the application of AAC are appropriate for the application of picture symbols and visual supports. Using the participation model (Beukelman and Mirenda 2005) as a guide, AAC interventions focus on allowing an individual with speech

impairment to participate in their environment to the same extent as that of their peers. Beukelman and Mirenda (2005) also provide strategies and recommendations for implementing AAC for both nonsymbolic and symbolic beginning communicators. Nonsymbolic beginning communicators are those who use nonsymbolic communication such as gestures, facial expression, cries, or grunts. Symbolic beginning communicators use some form of symbolic communication such as words (spoken or written) or symbols with low- or high-tech communication devices. The authors state “opportunity for communication is at least as important to the success of a communication intervention as the availability of an appropriate system” (p. 272). Additionally, the authors provide techniques related to shaping intentional communication, using scripted routines, providing natural consequences, and using structured instructional techniques such as the adapted strategic instruction model (A-SIM), structured practice, and conversational coaching.

Efficacy Information

A research synthesis was written by Wendt (2009) that discussed the application of picture symbols and visual supports for individuals with autism outside the domain of high-tech speech-generating devices. All of the studies included used a single-subject experimental design, and most of the studies focused on the ability of the individual with ASD to request objects or actions. A few studies focused on using visual supports to transition between activities, and one focused on recognition of orthographic symbols. The results of the review support the use of picture symbols for requesting. Children and adults were able to use picture symbols successfully to request a variety of objects and actions. Additionally, visual schedules were found to be helpful for individuals with ASDs in transitioning from one activity to another independently without the anxiety associated with inconsistent routines and without the need for constant prompting from another person.

Outcome Measurement

Because the goal of AAC use is functional communication, the outcome measurement should be the same. Functional communication will be defined differently based on the cognitive skills of the individual and the type of AAC system that is in place.

Qualifications of Treatment Providers

AAC (augmentative and alternative communication) interventions are most typically introduced by a speech-language pathologist. Unfortunately, many speech-language pathologists do not report having adequate training or education in the field of AAC (King 1998; Marvin et al. 2003; Simpson et al. 1999), and a survey of education programs for speech-language pathologists has uncovered a need for better education in this area (Ratcliff et al. 2008). Although this is the case, speech-language pathologists are the best equipped of all professionals who work with individuals with autism to provide intervention that includes AAC. A listing of speech-language pathologists who are certified by the American Speech-Language-Hearing Association can be found on their website. A few short questions posed to the speech-language pathologist can reveal whether they are comfortable with the area of AAC.

See Also

- [Alternative Communication](#)
- [Assistive Devices](#)
- [Communication Board](#)
- [Low-Technology Device](#)
- [Total Communication \(TC\) Approach](#)

References and Reading

- Beukelman, D. R., & Mirenda, P. (2005). *Augmentative and alternative communication: Supporting children and adults with complex communication needs*. Baltimore: Brooks Publishing.
- Bloomberg, K. (1996). *PrAACtically speaking information and resource booklet*. Melbourne: Yooralla Society of Victoria/Functional Communication Outreach Service.
- Bondy, A. S., & Frost, L. A. (1994). The picture exchange communication system. *Focus on Autism and Other Developmental Disabilities*, 9, 1–19.
- Drager, K. D. R., Light, J. C., & Finke, E. H. (2009). Using AAC technologies to build language and social communication with young children with autism spectrum disorders. In P. Mirenda & T. Iacono (Eds.), *AAC for individuals with autism spectrum disorders*. Baltimore: Paul H. Brookes.
- Frith, U., & Baron-Cohen, S. (1987). Perception in autistic children. In D. J. Cohen & A. M. Donnellan (Eds.), *Handbook of autism and pervasive developmental disorders*. New York: Wiley.
- Grandin, T. (1995). The learning style of people with autism: An autobiography. In K. A. Quill (Ed.), *Teaching children with autism: Strategies to enhance communication and socialization* (pp. 265–286). New York: Delmar.
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. London: Pergamon.
- Hodgdon, L. Q. (1995). Solving social-behavioral problems through the use of visually supported communication. In K. A. Quill (Ed.), *Teaching children with autism: Strategies to enhance communication and socialization* (pp. 265–286). New York: Delmar.
- King, J. (1998). Preliminary survey of speech-language pathologists providing AAC services in health care settings in Nebraska. *Augmentative and Alternative Communication*, 14, 222–227.
- Lancioni, G. (1983). Using pictorial representation as communication means with low-functioning children. *Journal of Autism and Developmental Disorders*, 13, 87–105.
- Lloyd, L. L., & Kangas, K. (1994). Augmentative and alternative communication. In G. H. Shames, E. H. Wiig, & W. A. Secord (Eds.), *Human communication disorders* (4th ed., pp. 606–657). New York: Merrill/Macmillan.
- Marvin, L. A., Montano, J. J., Fusco, L. M., & Gould, E. P. (2003). Speech-language pathologists' perception of their training and experience in using alternative and augmentative communication. *Contemporary Issues in Communication Science and Disorders*, 30, 76–83.
- Mesibov, G., Browder, D., & Kirkland, C. (2002). Using individualized schedules as a component of positive behavioral support for students with developmental disabilities. *Journal of Positive Behavior Interventions*, 4, 73–79.
- Mirenda, P. (2003). Toward functional augmentative and alternative communication from students with autism: Manual signs, graphic symbols and voice output communication aids. *Language, Speech, and Hearing Services in Schools*, 34, 203–216.
- Mirenda, P. (2009). Introduction to AAC for individuals with autism spectrum disorders. In P. Mirenda & T. Iacono (Eds.), *Autism spectrum disorders and AAC*. Baltimore: Paul H. Brookes.

- Prior, M., & Chen, C. (1976). Short-term and serial memory in autistic, retarded, and normal children. *Journal of Autism and Childhood Schizophrenia*, 6, 121–131.
- Ratcliff, A., Koul, R., & Lloyd, L. L. (2008). Preparation in augmentative and alternative communication: An update for speech-language pathology training. *American Journal of Speech-Language Pathology*, 17, 48–59.
- Romski, M. A., & Sevcik, R. A. (1996). *Breaking the speech barrier: Language development through augmented means*. Baltimore: Paul H. Brookes.
- Simpson, K., Beukelman, D., & Bird, A. (1999). Survey of school speech and language service provision to students with severe communication impairments in Nebraska. *Augmentative and Alternative Communication*, 14, 212–221.
- Wendt, O. (2009). Research on the use of manual signs and graphic symbols in autism spectrum disorders. In P. Mirenda & T. Iacono (Eds.), *Autism spectrum disorders and AAC*. Baltimore: Paul H. Brookes.

Picture Arrangement

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Definition

Picture Arrangement is a subtest on both the Wechsler Intelligence Scale for Children-3rd edition (WISC-III; Wechsler 1991) and the Wechsler Adult Intelligence Scale-3rd edition (WAIS-III; Wechsler 1997). This subtest was dropped from the most recent versions of both the child and adult versions of the Wechsler tests (WISC-IV and WAIS-IV). In this subtest, individuals are presented with a series of cards in an incorrect order that must be placed in the correct order to tell a story that makes sense. The stories are similar to short comic strips, and placing them in order relies on the individual's ability to recognize the cause and effect relationship of events depicted in the cards. This task gives information about an individual's reasoning abilities, and performance is related to the ability to understand precursors and consequences of events. The pictures on the cards involve human characters and interactions, so to some degree the test draws on

the ability to understand antecedents and consequences of social interactions. Because of this, there has been a long-standing assumption that performance on Picture Arrangement is associated with social competence, especially given that performance on this subtest by individuals with autism is relatively weak compared to other subtests on the Wechsler tests (Lincoln et al. 1988). However, more recent evidence has suggested that performance does not in fact predict social understanding or competence (Campbell and McCord 1999; Lipsitz et al. 1993), and performance may instead be more related to general intelligence. Low scores may be related to difficulties with visual organization and visual acuity, or in anticipating events and their consequences; inattention to details and anxiety might also interfere with performance.

See Also

- [Wechsler Preschool and Primary Scale of Intelligence](#)

References and Reading

- Campbell, J. M., & McCord, D. M. (1999). Measuring social competence with the Wechsler picture arrangement and comprehension subtests. *Assessment*, 6, 215–223.
- Lincoln, A., Courchesne, E., Kilman, B. A., Elmasian, R., & Allen, M. (1988). A study of intellectual abilities in high-functioning people with autism. *Journal of Autism and Developmental Disorders*, 18, 505–523.
- Lipsitz, J. D., Dworkin, R. H., & Erlenmeyer-Kimling, L. (1993). Wechsler comprehension and picture arrangement subtests and social adjustment. *Psychological Assessment*, 5, 430–437.
- Wechsler, D. (1991). *Wechsler intelligence scale for children* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (1997). *Wechsler adult intelligence scale* (3rd ed.). San Antonio: The Psychological Corporation.

Picture Board

- [Communication Board](#)

Picture Exchange Communication System

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Definition

The Picture Exchange Communication System (PECS) (<http://pecsusa.com/pecs.php>) is an aided picture-based augmentative intervention used to teach functional communication within a social context (Charlop-Christy et al. 2002; Frost and Bondy 2002). It requires an individual to spontaneously select a picture representing a desired item and exchanging that picture with a communication partner to retrieve the desired item. It is one of several interventions used to augment the communication of individuals with no or limited functional communication, particularly those with autism spectrum disorders (ASD).

Historical Background

Lori Frost and Andrew Bondy originally developed PECS at the Delaware Autism Institute for children with autism spectrum disorder (ASD) and other socio-communicative disorders who had limited or no functional communication. PECS was designed to address some of the problems associated with more traditional language training programs that require prerequisite skills such as motor and verbal imitation, visual attention, pointing, and initiation. It was also developed to circumvent the frequent difficulty children with ASD have with prompt dependency and obtaining a listener's attention (Bondy and Frost 1994, 1998; Frost and Bondy 1994). The first manual for implementing PECS was published in 1994. In 2002, Frost and Bondy updated the PECS by broadening their view on

the importance of establishing a foundation for communication training by systematically structuring the learning environment, redefining their intervention as the Pyramid Approach to Education (Bondy 2011), and emphasizing “functional activities and communication, powerful reinforcers and behavior intervention plans” (Frost and Bondy 2002, p. vii).

Rationale or Underlying Theory

PECS is designed to address the failure to initiate communication, a common deficit area for those with autism, which has implications for long-term outcomes related to the development of functional communication (National Research Council 2001). Further, as individuals with ASD often lack the motivation and the social skills (Ogletree et al. 2007) to communicate effectively in social contexts (e.g., failing to orient or attend to others, difficulty displaying affection and emotions in social contexts, and impairment in gesturing and pointing), a system that supports the development of social communication is critical. PECS offers a systematic approach to teach individuals to use functional communication skills to facilitate an exchange with a communicative partner. Bondy and Frost (1998) believe that an effective communication training system should use meaningful reinforcers through the application of request situations rather than labeling situations, emphasize spontaneous communication, avoid prompt dependency, and not require extensive training prior to the initiation of the system.

The underlying theoretical framework for PECS is grounded in the principles of behaviorism (Skinner 1953) and applied behavioral analysis (Baer et al. 1968), suggesting that the environment controls the learning of verbal and nonverbal communication skills and techniques such as modeling, prompting, and shaping can be used to facilitate that learning. PECS also capitalizes on the belief that many individuals with ASD are considered visual learners and a communication system that can effectively and efficiently incorporate visual and graphic symbols is likely to be more successful than those without a visual

emphasis (Mirenda 2001; National Research Council 2001). In addition, PECS uses a direct reinforcement system to motivate its communicators. That is, communicators with limited expressive language skills are taught to initiate nonverbal communicate to achieve their desired outcomes. PECS users learn that they can exchange a picture or icon for their actual desired item or activity. Thus, receiving what they requested rewards communicators.

Goals and Objectives

Bondy and Frost emphasize the importance of establishing educational objectives and activities that are functional and can be taught and utilized at school, home, and in the community. Intervention goals in the PEC system serve two broad communication functions, more directive communication attempts that serve as requests or commands and social communication attempts that include commenting, describing, and naming. Goals are established for each of the six phases of intervention with current levels of performance and the criteria for achieving each objective defined.

Treatment Participants

PECS has been utilized to support the communication of a variety of individuals. Originally, it was designed for young children with ASD (Bondy and Frost 2001; Ganz and Simpson 2004), but it has been successfully used with older children and adolescents who have limited expressive language or lack a functional communication system. Similarly, PECS has been applied to augment the communication of individuals with a range of language and developmental levels beyond ASD.

Although a formal assessment of who might be the most appropriate candidates for PECS is not available, the Pyramid Educational Consultants pose several questions for consideration when identifying possible candidates. These include whether or not a person has a functional

communication system, the extent to which their communication attempts are understood, how effective their communication system is at addressing their wants and needs, and whether their current system facilitates spontaneous communication and the ability to respond to a variety of questions.

Treatment Procedures

PECS has some advantages over other communication interventions in that communicators are not required to exhibit prerequisite skills such as attention and eye contact nor are they required to demonstrate comprehension of the pictures or icons used prior to initiating the intervention. PECS focuses on developing a number of key receptive and expressive language skills (Frost and Bondy 2002). Receptively, PECS targets the ability to respond to directions, follow visual cues, and follow a direction to wait. Expressively, PECS emphasizes requesting help, a break, or desired item or activities and rejecting or affirming offers of items or activities. Instructors determine which skills the communicator needs and then plan to address these within the six phases of PECS.

Prior to implementation of the six intervention phases, a reinforcer assessment is completed with the individual for whom the intervention is being used to determine what items are highly preferred, preferred, and nonpreferred. Individualized reinforcers as well as the corresponding pictures and/or icons that match the communicators' desired items are determined by the communicators' interests, needs, and wants. Input is gathered from parents or caregivers and probing the individual's preferences from 5 to 8 selected items (Frost and Bondy 1994).

Following the reinforcer assessment, *Phase I: Teaching the physically Assisted Exchange* is initiated. This phase requires two trainers: one person who serves as the communication partner and receives the picture and one who provides, at least initially, hand-over-hand assistance. The goal of Phase I is to have the communicator spontaneously pick up a picture of a highly preferred

item, reach toward the trainer, and release the picture into the trainer's open hand (Frost and Bondy 1994).

In *Phase II: Expanding Spontaneity*, the distance between the communicator and the communication board and the communicator and the trainer is increased. Spontaneity is expanded to increase communicative persistence so that the individual goes to his/her communication board, removes a picture, finds the trainer, and releases the picture into the trainer's hand (Bondy and Frost 1998; Frost and Bondy 1994).

Phase III: Simultaneous Discrimination of Pictures teaches the communicator to discriminate between pictures. The communicator requests a desired item by going to his/her communication board, selects the picture representing the desired item from an array, moves to the communication partner, and releases the picture into the partner's hand (Frost and Bondy 1994).

Phase IV: Building Sentence Structure teaches the communicator to request using the carrier phrase, "I want." The individual selects a desired picture and places it on a sentence strip next to a picture depicting "I want ____" to complete the phrase. The individual then gives the entire sentence strip to the communication partner.

In *Phase V: Responding to "What do you want?"* the communicator is taught to respond to this question using delayed prompting. Throughout the communicator's daily activities, he/she is expected to spontaneously request desired items and answer the "What do you want?" question.

The final phase, *Phase VI: Commenting in Response to Questions*, requires the communicator to respond to different questions (e.g., "What do see?" "What do you have?") using carrier phrases (e.g., "I see ____." "I have ____.") to teach communicative functions like labeling or naming. The communicator is taught to respond to the communication partner's questions related to a particular referent such as pulling a car out of a bag and asking, "What do you see?" with the expectation that the communicator will select the correct picture for the item and create a sentence strip that says "I see car."

Beyond Phase VI, communicators are taught a variety of language concepts (e.g., location, verb

concepts, attributes) using the basic PECS principles. A variety of teaching procedures that fall under the umbrella of applied behavior analysis are used to facilitate the success of the communicator during the training phases. These include the use of backward chaining, incidental training, shaping, discrete trials, delayed prompting, and discrimination training (Bondy and Frost 1994).

Efficacy Information

Over the last 20 years, several studies have investigated the effectiveness of PECS as a communication intervention for children with ASD. PECS has been investigated in small groups and single subject designs including both efficacy (those occurring in controlled settings like research labs and clinics) and effectiveness (those occurring in natural settings like home and school) studies (Prelock et al. 2011). Research has focused primarily on children with ASD 18 months to 12 years of age with limited investigation of older children, adolescents, and adults. Some comparisons of PECS to other interventions (Beck et al. 2008; Tincani 2004; Yoder and Stone 2006) exist, although this is an area of research requiring further examination to determine what outcomes might best be facilitated by PECS versus other interventions.

A number of functional communication outcomes have been reported following PECS training ranging from speech facilitation (Bondy and Frost 1994, 1998; Ganz et al. 2008) and increased initiations (Carr and Felce 2007a, b; Howlin et al. 2007) to gains in joint attention, eye contact, and toy play with a reduction of problem behaviors (Charlop-Christy et al. 2002; Yoder and Lieberman 2010). In addition, results have revealed increases in vocabulary (Anderson et al. 2007; Jurgens et al. 2009; Magiati and Howlin 2003; Yoder and Stone 2006) and utterance length (Charlop-Christy et al. 2002; Ganz and Simpson 2004; Jurgens et al. 2009). Maintenance of speech gains and generalized use of trained and untrained functions across settings have also been documented (Charlop-Christy et al. 2002; Kravitz et al. 2002; Schwartz et al. 1998; Yokoyama et al.

2006). Significant increases in spontaneous communication using pictures, speech, or both were found in a randomized control trial with 84 school children, 4–10 years of age following the use of PECs (Gordon et al. 2011).

Ostry et al. (2008) suggested that PECS should be part of a multimodal communication system to facilitate joint attention and question asking, and Tien (2008) reviewed 13 studies with results suggesting PECS effectively improves functional communication in children with ASD. A meta-analysis of PECS research completed between 1994 and 2009 found it to be a promising intervention for facilitating communication in children 1–11 years of age, although speech gains were less consistent (Flippin et al. 2010). Ganz et al. (2012) also completed a meta-analysis with similar findings that PECS was most successful in supporting functional communication outcomes and particularly for preschool children with ASD.

PECS appears to be an appropriate strategy for supporting young children at the early stages of communication (Prelock et al. 2011). More research, however, is needed to identify the profiles of those children most likely to benefit from PECS versus other intervention methods.

Outcome Measurement

No specific tools are used to measure treatment outcomes, although consistent with the principles of applied behavior analysis, PECS incorporates a data-based system to measure progress across all phases of the program. An 80 % or higher correct response rate for a minimum of 3 days is required for a communicator to move across each phase of PECS (Frost and Bondy 1994).

Practitioners and researchers have been interested in the development of spoken language following the implementation of PECS. Frost and Bondy (1994) reported that children learning PECS began speaking. Ganz and Simpson (2004) confirmed that spoken language sometimes follows training in PECS. Interest also exists in the impact of PECS on reducing behavior problems and aberrant responses (Charlop-Christy et al. 2002; Ogletree et al. 2007).

Qualifications of Treatment Providers

Speech-language pathologists, teachers, families, and others can use PECS in a variety of settings as it does not require complex or expensive technology or equipment, materials are readily available, training is accessible, and the intervention is manualized.

Pyramid Educational Consultants offer PECS training globally. Providers seeking implementation certification status (<http://www.pecsusa.com/certification.php>) must provide proof of attendance at a basic PECS workshop and must demonstrate competency in implementing the six phases of PECS, error correction procedures, implementation during functional activities, writing PECS lessons and summaries, and data collection. Those receiving a *PECS Implementer Certificate* can renew their certificate every 3 years. Pyramid Educational Consultants have explicit rules and guidelines for those who complete each level of PECS training. They also provide training for supervisors. This is an advanced training for individuals who are skilled in applying the Pyramid Approach to PECS implementation in the worksite and with those who have completed the basic PECS training.

See Also

- ▶ Alternative Communication
- ▶ Applied Behavior Analysis (ABA)
- ▶ Communicative Functions
- ▶ Initiation of Communication
- ▶ Pictorial Cues/Visual Supports (CR)
- ▶ Picture Arrangement
- ▶ Visual Supports

References and Reading

- Anderson, A., Moore, D., & Bourne, T. (2007). Functional communication and other concomitant behavior change following PECS training: A case study. *Behaviour Change*, 24(3), 173–181.
- Baer, D., Wolf, M., & Risley, T. (1968). Some current dimensions of applied behavior analysis. *Journal of Applied Behavior Analysis*, 1, 91–97.

- Beck, A. R., Stoner, J. B., Bock, S. J., & Parton, T. (2008). Comparison of PECS and the use of a VOCA: A replication. *Education and Training in Developmental Disabilities, 43*, 198–216.
- Bondy, A. S. (2011). *Pyramid approach to education* (2nd ed.). New Castle: Pyramid Educational Consultants, Inc.
- Bondy, A. S., & Frost, L. A. (1994). The picture exchange communication system. *Focus on Autistic Behavior, 9*(3), 1–19.
- Bondy, A. S., & Frost, L. A. (1998). The picture exchange communication system. *Seminars in Speech and Language, 19*(4), 373–388.
- Bondy, A. S., & Frost, L. A. (2001). The picture exchange communication system. *Behavior Modification, 25*(5), 725–744.
- Carr, D., & Felce, J. (2007a). The effects of PECS teaching to Phase III on the communicative interactions between children with autism and their teachers. *Journal of Autism and Developmental Disorders, 37*(4), 724–737.
- Carr, D., & Felce, J. (2007b). Brief report: Increase in production of spoken words in some children with autism after PECS teaching to Phase III. *Journal of Autism and Developmental Disorders, 37*(4), 780–787.
- Charlop-Christy, M. H., Carpenter, M., Le, L., LeBlanc, L. A., & Kellet, K. (2002). Using the Picture Exchange Communication System (PECS) with children with autism: Assessment of PECS acquisition, speech, social-communicative behavior, and problem behavior. *Journal of Applied Behavior Analysis, 35*(3), 213–231.
- Flippin, M., Reszka, S., & Watson, L. D. (2010). Effectiveness of the Picture Exchange Communication System (PECS) on communication and speech for children with ASD: A meta-analysis. *American Journal of Speech-Language Pathology, 19*(2), 178–195.
- Frost, L. A., & Bondy, A. (1994). *The picture exchange communication system training manual*. Cherry Hill: Pyramid Educational Consultants.
- Frost, L. A., & Bondy, A. S. (2002). *The picture exchange communication system training manual* (2nd ed.). Newark: Pyramid Educational Products.
- Ganz, J. B., & Simpson, R. L. (2004). Effects on communicative requesting and speech development of the picture exchange communication system in children with characteristics of autism. *Journal of Autism and Developmental Disorders, 34*(4), 395–409.
- Ganz, J. B., Simpson, R. L., & Corbin-Newsome, J. (2008). The impact of the picture exchange communication system on requesting and speech development in preschoolers with autism spectrum disorders and similar characteristics. *Research in Autism Spectrum Disorders, 2*, 157–169.
- Ganz, J. B., Davis, J. L., Lund, E. M., Goodwyn, F. D., & Simpson, R. L. (2012). Meta-analysis of PECS with individuals with ASD: Investigation of targeted versus non-targeted outcomes, participant characteristics, and implementation phase. *Research in Developmental Disabilities, 33*(2), 406–418.
- Gordon, K., Pasco, G., McElduff, F., Wade, A., & Howlin, P. (2011). A communication-based intervention for nonverbal children with autism: What changes? Who benefits? *Journal of Consulting and Clinical Psychology, 79*(4), 447–457.
- Howlin, P., Gordon, R. K., Pasco, G., Wade, A., & Charman, T. (2007). The effectiveness of Picture Exchange Communication System (PECS) training for teachers of children with autism: A pragmatic, group randomized controlled trial. *Journal of Child Psychology and Psychiatry, 48*(5), 473–481.
- Jurgens, A., Anderson, A., & Moore, D. W. (2009). The effect of teaching PECS to a child with autism on verbal behaviour, play and social functioning. *Behaviour Change, 26*(1), 66–81.
- Kravitz, T. R., Kamps, D. M., Kemmerer, K., & Potucek, J. (2002). Brief report: Increasing communication skills for an elementary-aged student with autism using the Picture Exchange Communication System. *Journal of Autism and Developmental Disorders, 32*(3), 225–230.
- Magiati, I., & Howlin, P. (2003). A pilot evaluation study of the picture exchange communication system for children with autistic spectrum disorders. *Autism, 7*, 297–320.
- Mirenda, P. (2001). Autism, augmentative communication and assistive technology: What do we really know? *Focus on Autism and Other Developmental Disabilities, 16*(3), 141–151.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Ogletree, B., Oren, T., & Fischer, M. (2007). Examining effective intervention practices for communication impairment in autism spectrum disorder. *Exceptionality, 15*(4), 233–247.
- Ostry, C., Wolfe, P. S., & Rusch, F. R. (2008). A review and analysis of the Picture Exchange Communication System (PECS) for individuals with autism spectrum disorders using a paradigm of communication competence. *Research and Practice for Persons with Severe Disabilities, 33*(1–2), 13–24.
- Prelock, P. A., Paul, R., & Allen, E. (2011). Evidence-based treatments in communication for children with autism spectrum disorders. In B. Reichow, P. Doehring, D. V. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-based treatments for children with autism* (pp. 93–169). New York: Springer.
- Schwartz, I. S., Garfinkle, A. N., & Bauer, J. (1998). The Picture Exchange Communication System: Communicative outcomes for young children with disabilities. *Topics in Early Childhood Special Education, 18*(3), 144–159.
- Skinner, B. F. (1953). *Science and human behavior*. New York: Macmillan.
- Tien, K.-C. (2008). Effectiveness of the Picture Exchange Communication System as a functional communication intervention for individuals with autism spectrum disorders: A practice-based research synthesis. *Education and Training in Developmental Disabilities, 43*(1), 61–76.
- Tincani, M. (2004). Comparing the picture exchange communication system and sign language training for children with autism. *Focus on Autism and Other Developmental Disabilities, 19*(3), 152–163.

- Yoder, P. J., & Lieberman, R. G. (2010). Brief report: Randomized test of the efficacy of PECS on highly generalized picture exchanges in children with ASD. *Journal of Autism & Developmental Disorders*, 40(5), 629–631.
- Yoder, P., & Stone, W. (2006). Randomized comparison of two communication interventions for preschoolers with autism spectrum disorders. *Journal of Consulting and Clinical Psychology*, 74(3), 426–435.
- Yokoyama, K., Naoi, N., & Yamamoto, J. (2006). Teaching verbal behavior using the picture exchange communication system (PECS) with children with autism spectrum disorders. *Japanese Journal of Special Education*, 43(6), 485–503.

Picture Schedule

- [Visual Schedule](#)

Picture Thinking

- [Visual Thinking](#)

Pimozide

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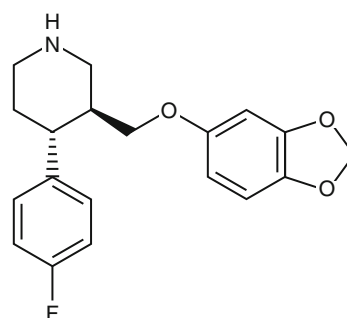
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Synonyms

1-[1-[4,4-bis(p-fluorophenyl)butyl]-4-piperidyl]-2-benzimidazolinone; Orap

Definition



Pimozide is an antipsychotic in the diphenylbutylpiperidine class with the chemical formula $C_{28}H_{29}F_2N_3O$. This drug was initially FDA-approved in 1984 for the treatment of severe tics associated with Tourette syndrome. It is used outside of the United States for schizophrenia and has also been found to be effective in the treatment of monosymptomatic hypochondriacal psychosis, body dysmorphic disorder, metastatic melanoma, trichotillomania, and trigeminal and postherpetic neuralgia. Adverse effects include extrapyramidal reactions, akathisia, dystonia, tardive dyskinesia, neuroleptic malignant syndrome, menstrual irregularities, and galactorrhea. The drug has also been reported to have adverse cardiac effects. Unexpected deaths have been reported in patients using high doses of this drug.

See Also

- [Antipsychotics: Drugs](#)

References and Reading

- Lorenzo, C. R., & Koo, J. (2004). Pimozide in dermatologic practice: A comprehensive review. *American Journal of Clinical Dermatology*, 5(5), 339–349.
- Pimozide. (n.d.). Retrieved from ChemSpider: The free chemical database website: <http://www.chemspider.com/RecordView.aspx?rid=9b50bf87-08b4-4616-be97-81e5b4a466b2>.
- Rathbone, J., & McMonagle, T. (2007) Pimozide for schizophrenia or related psychoses. *Cochrane Database of Systematic Reviews*, 3. Retrieved from Cochrane Library database.
- U.S. Food and Drug Administration. (2010). *ORAP® (Pimozide) Tablets*. Retrieved from: http://www.accessdata.fda.gov/drugsatfda_docs/label/2010/017473s0451bl.pdf.

Pincer Grasp

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Definition

When a child employs a pincer grasp, the tips of the thumb and index finger are used in opposition to pick up and manipulate small objects. The ability to perform this task is a milestone of fine motor development that typically emerges before a child's first birthday. Prior to the emergence of this skill, infants grasp objects in more rudimentary ways. Ordinarily, by 6–7 months, objects are raked into the palm with the fingers. By 8–9 months, the thumb and radial fingers are used to grasp and pick up small objects. Then, by 10–12 months, the fine pincer grasp appears. It can be tested by allowing a child to pick up a small object (e.g., a piece of breakfast cereal) from a flat surface. Once this skill is added to a child's fine motor repertoire, their ability to explore their world through object manipulation is greatly increased.

See Also

- [Fine Motor Development](#)
- [Motor Control](#)

References and Reading

Edwards, S. J., Buckland, D. J., & McCoy-Powlen, J. D. (2002). Development of grasp. In *Developmental and functional hand grasps* (pp. 41–56). Thorafare: Slack Incorporated.

Pivotal Response Training

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Synonyms

[Natural language paradigm](#); [Naturalistic behavioral intervention](#); [Pivotal Response Teaching[®]](#); [Pivotal Response Treatment[®]](#); [PRT[®]](#)

Definition

Pivotal response training[®] (PRT[®]) is an individualized, comprehensive treatment model that uses the child's motivation to present learning opportunities within their natural environment. It was developed by Lynn and Robert Koegel and initially called the natural language paradigm (Koegel et al. 1987). Grounded in applied behavior analysis and developmental approaches, PRT[®] targets pivotal areas such as motivation, responsivity to multiple cues, self-management, and self-initiations. Focusing on these pivotal areas results in collateral gains in untargeted areas and generalized sustained improvement in language, behavior, and social outcomes. Its comprehensive approach encourages consistent and coordinated programming across the child's contexts, with particular focus on parent education. Parents play an integral role in the habilitation process and receive training in PRT[®] using a collaborative, ecocultural approach. Through this partnership-based model, parents identify clinically meaningful goals and intervention strategies that can be easily incorporated into their already existing daily routines. The aim of PRT[®] is to improve the quality of life for children with autism spectrum disorders by creating opportunities to lead meaningful lives in fully inclusive settings.

Pivotal Response Teaching[®]

- [Pivotal Response Training](#)

See Also

- [Natural Language Paradigm](#)
- [Naturalistic Interventions](#)

References and Reading

- Koegel, R. L., & Koegel, L. K. (Eds.). (2006). *Pivotal response treatments for autism: Communication, social, and academic development*. Baltimore: Paul H. Brookes.
- Koegel, R. L., O'Dell, M. C., & Koegel, L. K. (1987). A natural learning paradigm for nonverbal autistic children. *Journal of Autism and Developmental Disorders*, 18, 525–538.
- Koegel, R. L., Koegel, L. K., Vernon, T. W., & Brookman-Frazee, L. I. (2010). Empirically supported pivotal response treatment for children with autism spectrum disorders. In J. R. Weisz & A. E. Kazdin (Eds.), *Evidence-based psychotherapies for children and adolescents* (2nd ed., pp. 327–344). New York: Guilford Press.
- Smith, I. M., Koegel, R. L., Koegel, L. K., Openden, D. A., Fossum, K. L., & Bryson, S. E. (2010). Effectiveness of a novel community-based early intervention model for children with autistic spectrum disorder. *American Journal on Intellectual Disabilities*, 115(6), 50–523.

Pivotal Response Treatment®

- [Pivotal Response Training](#)

PL94-142

- [Education for All Handicapped Children Act of 1975 \(PL94-14 L\)](#)

Placement-Pending Requirement

- [Stay-Put Requirement](#)

Placenta

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Definition

If we could noninvasively analyze the detailed neuroanatomy of all newborns at a cellular, synapse by synapse level, we might be able to identify those children who will go on to have an autistic spectrum disorder (ASD). As that is unlikely to be possible anytime soon, doctors seek surrogates that can make the same diagnoses. Luckily, the placenta, an organ that is often routinely discarded at birth, may be able to identify those babies who will exhibit overt signs of autism months or years later.

The human placenta is not only an integral part of the fetus during all of pregnancy, but it also shares in the vast majority of cases the exact genetic makeup of the fetus. As such, it offers the potential to reveal abnormal morphologic patterns that may be at the basis of ASD and other genetic and developmental abnormalities.

Historical Background

Formation of the Placenta

Humans start off as a symmetrical ball of cells. Even as our first few dozen cells begin to separate into an inner cell mass (which will become the embryo, fetus, and eventually baby) and the trophoblasts (which will become the placenta), genes are regulating the creation of the developmental axes that will form the basis of the entire organism (Kliman 1999). Defects in the genes that regulate these processes lead to a wide range of embryonic, fetal, and neonatal defects from minor cosmetic

abnormalities to disasters that terminate pregnancy within a few days to weeks after fertilization.

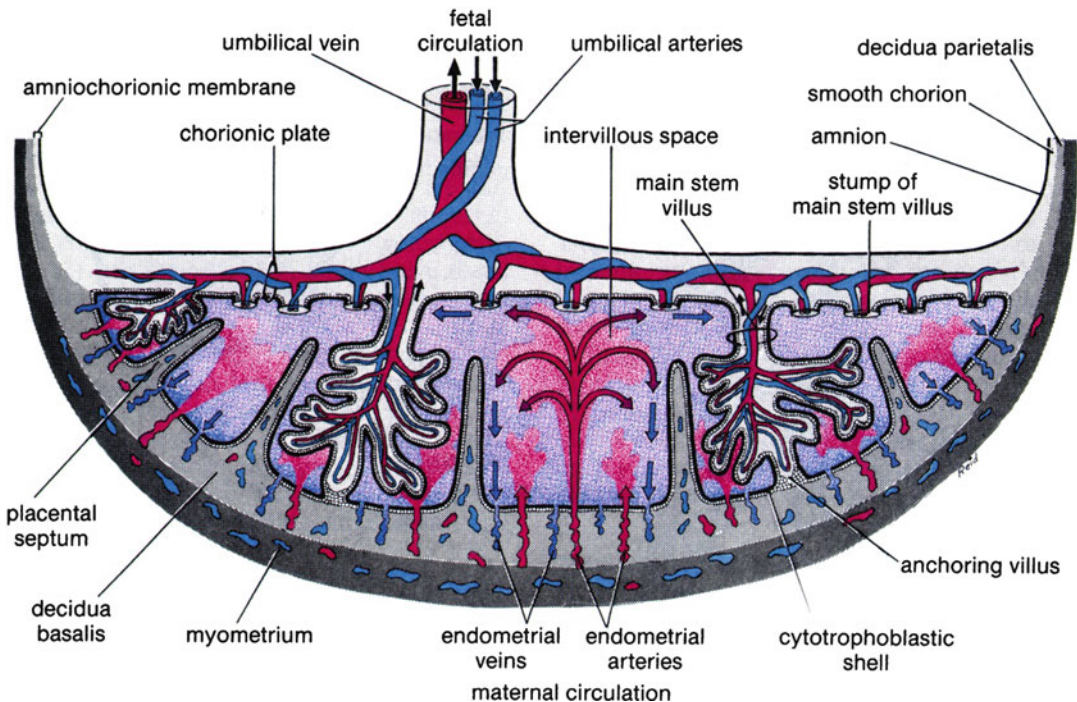
By 21 days after fertilization, the trophoblasts have begun to sort themselves into what will become the treelike structures that make up the placenta: the chorionic trees, branches, and villi (Fig. 1). The terminal villi (from the Latin for shaggy hair) are fingerlike structures that are covered with a double cell layer. This trophoblast bilayer is made up of an inner cytotrophoblast layer made of single nucleated cells and an outer syncytiotrophoblast layer made of giant multinucleated sheets (Fig. 2). Starting with purified cytotrophoblasts, we demonstrated using in vitro time lapse cinematography that cytotrophoblasts fuse to form syncytiotrophoblast (Kliman et al. 1986) (Fig. 3), an observation that has been confirmed in situ (Huppertz et al. 2001). The critical

conclusion from these studies is that only the cytotrophoblasts proliferate, making the growth of the syncytiotrophoblast layer completely dependent on the absorption of fusing cytotrophoblasts (Fig. 4).

Current Knowledge

Trophoblast Inclusions

The relative rates of cytotrophoblast proliferation and incorporation into the outer syncytiotrophoblast layer appear to determine the morphology of the fingerlike chorionic villi (Huppertz et al. 2001; Kliman and Segel 2003; Rejniak et al. 2004). In the normal placenta, new villus branches are formed by outward bending bulges of the trophoblast bilayer (Fig. 5). However, when these critical processes go awry, the

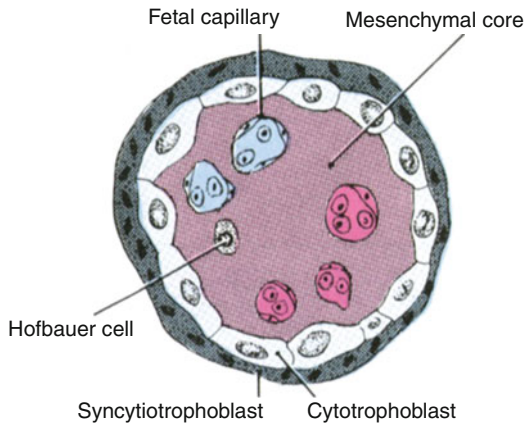


Placenta, Fig. 1 *Diagram of the human placenta.* The placenta, which is part of the fetus, attaches to the maternal decidua via the anchoring villi. The maternal blood is injected into the intervillous space where it circulates around the chorionic villi and then returns to the maternal circulation via the endometrial veins. The fetus pumps its blood into the placenta via the umbilical arteries, which

branch in the chorionic plate and eventually dive down to form the villus trees. The fetal circulation terminates in the fingerlike chorionic villi, which are covered by a layer of trophoblast cells (see Fig. 2). (From Moore KL, *The Developing Human*, 4th edition, WB Saunders, 1988, used with permission)

bilayer can inappropriately bulge inward into the villi, creating invaginations (Fig. 6) and trophoblast inclusions (Fig. 7) that can be readily detected upon histological examination of sectioned placental tissue.

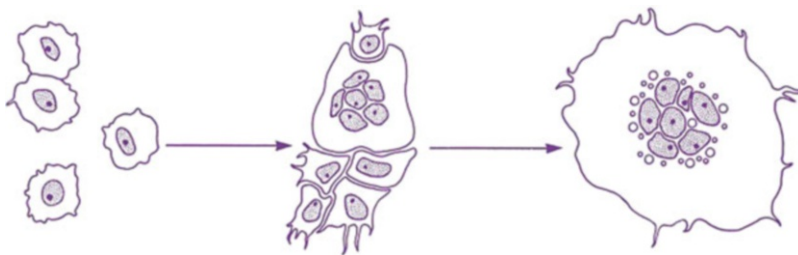
The basis of trophoblast invaginations and inclusions, therefore, appears to be an imbalance between the rate of cytotrophoblast proliferation



Placenta, Fig. 2 Diagrammatic cross section of first-trimester chorionic villus. The villus core of villi contains fetal capillaries embedded in a loose matrix which contains fibroblasts and macrophages (also called Hofbauer cells). In the first trimester, a villus cross section reveals two distinct trophoblast layers, the outer syncytiotrophoblast layer which is in direct contact with maternal blood and the inner cytotrophoblast layer, the stem cell of the placenta and the source of new trophoblasts. (Modified from Moore KL, The Developing Human, 4th edition, WB Saunders, 1988, used with permission)

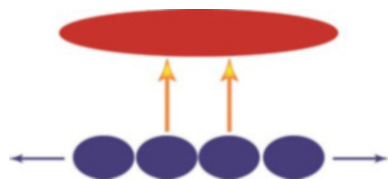
and fusion (Rejniak et al. 2004). This could be the result of either an increased rate of proliferation – due to either endogenous genetic factors within these cells or exogenous factors such as increased exposure to paracrine or endocrine growth factors – or the result of a decreased rate of fusion – due to decreased production of the factors that facilitate cell fusion. Endogenous cell proliferation rates may be affected by genes that regulate the mitotic cycle, such as the cyclins (Gillett and Barnes 1998). Equally potent are hormones that regulate cellular proliferation, such as growth hormone and insulin. Abnormalities of cytotrophoblast fusion have been shown in the placentas of Down syndrome (trisomy 21) children, showing a direct relationship between this particular genetic disorder and placental morphogenesis (Malassine et al. 2010).

Understanding how trophoblast invaginations and inclusions are formed helps to identify them in placental tissues and leads to criteria for diagnosing these dysmorphic features. In almost all cases of invagination, there is an increased number of cytotrophoblasts at the point of infolding compared to the density of cytotrophoblasts away from the invagination (Fig. 8). Likewise, careful examination of a trophoblast inclusion reveals a central region of syncytiotrophoblasts, surrounded by cytotrophoblasts (Fig. 8). Like an epidermal inclusion cyst, which continues to get larger and larger over time, the cytotrophoblasts continue to proliferate and fuse with the adjacent

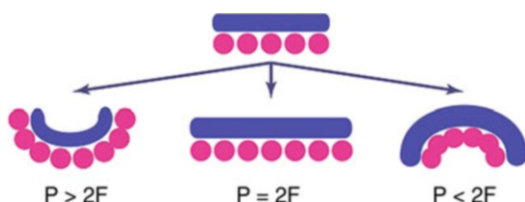


Placenta, Fig. 3 *In vitro* conversion of cytotrophoblasts into syncytiotrophoblasts. Individual cytotrophoblasts (left) migrate like amoeba, eventually making contact with each other. Once in contact, the cytotrophoblasts form aggregates (middle), and in time, the cells fuse to form syncytiotrophoblasts. Eventually, all the cytotrophoblasts have merged and fused to make a large

syncytiotrophoblast (right). (From Kliman HJ, Nestler JE, Sermasi E, Sanger JM, and Strauss JF III. (1986) Purification, characterization and *in vitro* differentiation of cytotrophoblasts from human term placentae. *Endocrinology* 118: 1567–1582, used with permission. Copyright 1986, The Endocrine Society)



Placenta, Fig. 4 Cytotrophoblast proliferation and fusion. Cytotrophoblasts (blue) either proliferate to increase the number of trophoblastic stem cells (blue arrows) or occasionally fuse upward (gold arrows) into the multinucleated syncytiotrophoblast layer (red). The balance of these two processes – proliferation and fusion – determines the overall morphology of the placenta's chorionic villi (see also Fig. 5)



Placenta, Fig. 5 Proliferation fusion model. A model illustrating the different ratios of proliferation (P) and fusion (F). Cytotrophoblasts (pink circles) proliferate and intermittently fuse into the upper syncytiotrophoblast layer (blue bars). Stability (relative flatness) of the bilayer is maintained at, or near, an ideal ratio: $P = 2F$. Normal outward budding (evagination) is observed from the ratio: $P < 2F$, while an abnormal trophoblast inclusion (invagination) results from the ratio: $P > 2F$. (Kliman HJ, Segel L. (2003) The placenta may predict the baby. *J Ther Biol*, 225: 143–145, used with permission)

syncytiotrophoblasts, which in some cases leads to very large trophoblast inclusions (see Fig. 7d). Adherence to the criteria of identifying increased numbers of cytotrophoblasts along the invagination and around the syncytiotrophoblast core of an inclusion helps to distinguish trophoblast invaginations and inclusions from tangential sections of curved chorionic villus surfaces.

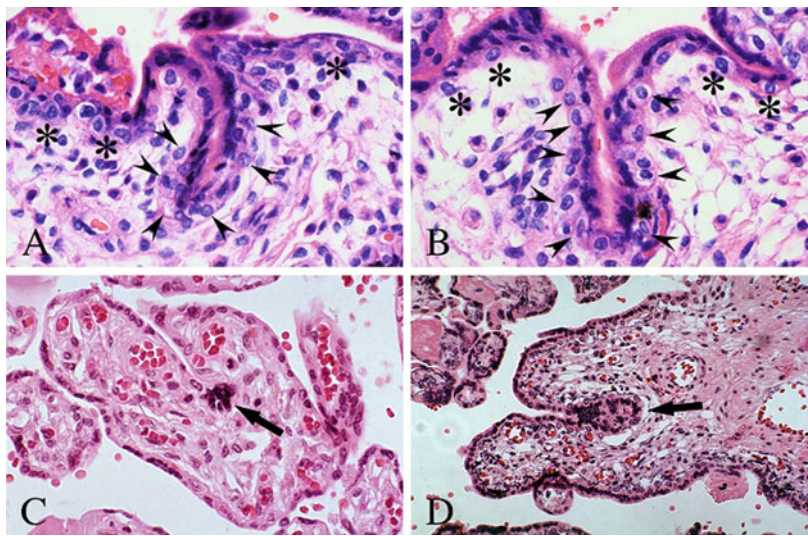
Although trophoblast inclusions were first described as a marker of triploid gestations (a complete extra one set of chromosomes) (Szulman et al. 1981), it is now appreciated that the presence of trophoblast inclusions in placentas is associated with a long list of genetically abnormal gestations, including tetraploidy (a complete extra two sets of chromosomes), trisomies (an extra individual chromosome, such as trisomy

21 or Down syndrome, trisomy 18, and trisomy 13), Turner's syndrome (female with one X chromosome missing), and even genetic diseases without obvious chromosome abnormalities (Honore et al. 1976; Novak et al. 1988; Silvestre et al. 1996; Szulman 1984; van Lijnschoten et al. 1993). Thus, many different genetic defects manifest themselves in the placenta as trophoblast inclusions. Since cytotrophoblast proliferation and absorption is no doubt regulated by many genes, it appears that abnormalities in any part of these multigene processes may result in these villus dysmorphic features.

Frequency of Trophoblast Inclusions

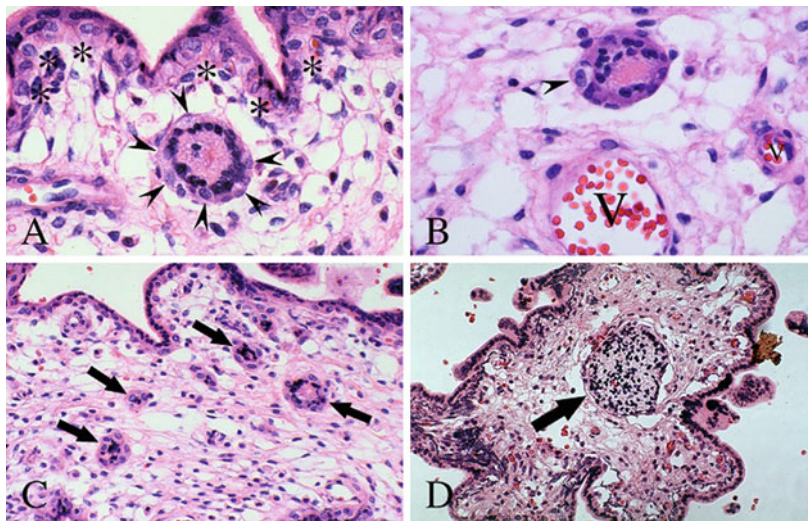
Fewer than 3% of placentas from uncomplicated, normal gestations manifest trophoblast inclusions. But 70% of placentas of fetuses with known chromosomal abnormalities exhibit inclusions (Kliman et al. 2003). Since the presence of a normal karyotype does not exclude the possibility of a genetic defect (e.g., cystic fibrosis, Tay-Sachs, sickle cell disease), it should not be surprising that trophoblast inclusions are also seen in cases with a normal karyotype. The most common example of the later situation is a spontaneous pregnancy loss where the karyotype is normal, but clearly, there is something abnormal about the gestation. The genetic basis of such losses is reinforced by the finding of both a high recurrence risk for these families and the fact that the losses almost always occur at the same gestational age for each particular family, suggesting a specific programming error.

The more severe the genetic abnormality, or the earlier the pregnancy loss, the more inclusions are found (Fig. 9). This is consistent with studies that have concluded that over 90% of first-trimester losses are secondary to genetic causes (Zhang et al. 2009). As pregnancies progress from the first to the third trimester, the likelihood of a genetic basis for a pregnancy loss decreases but does not go to zero even at term. Not surprisingly, the number of trophoblast inclusions seen in cases that reach term are fewer than those that terminate early in pregnancy. This is most likely related to the severity of the genetic abnormality, with the most severe terminating before 13 weeks of gestation.



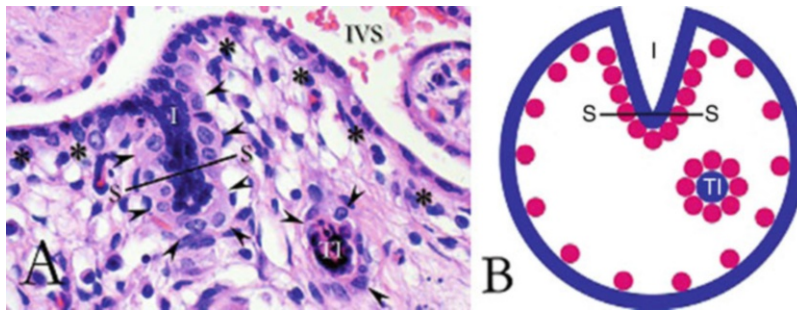
Placenta, Fig. 6 *Trophoblast invaginations.* (a, b) Trophoblast invaginations forming cleft-like structures. Note the many cytotrophoblasts lining the invaginations (arrowheads) and the fewer cytotrophoblasts underlying the normal bilayers (*). (c) Invagination ending in an area of increased syncytiotrophoblasts (arrow). Even though

the bilayer is invaginated, syncytiotrophoblasts still form. (d) Bulb-like prominence of syncytiotrophoblasts at base of an invagination (arrow). (Kliman HJ, Segel L. (2003) The placenta may predict the baby. *J Ther Biol*, 225: 143–145, used with permission)



Placenta, Fig. 7 *Trophoblast inclusions.* (a) Trophoblast inclusion within the villous core. Note how the cytotrophoblasts of the bilayer (arrowheads) and the cytotrophoblasts of the inclusion (*) both are adjacent to the villous core. (b) Trophoblast inclusion with a prominent syncytiotrophoblast layer and a lone cytotrophoblast

(arrowhead). Fetal vessels (V). (c) Chorionic villous with four prominent trophoblast inclusions (arrows). (d) Trophoblast inclusion with very expanded syncytiotrophoblast component (arrow). (Kliman HJ, Segel L. (2003) The placenta may predict the baby. *J Ther Biol*, 225: 143–145, used with permission)



Placenta, Fig. 8 *Formation of trophoblast invaginations and inclusions.* (a) Histologic section of a placental villus which exhibits both a trophoblast invagination (I) and inclusion (TI). Note the increased numbers of cytotrophoblasts (arrow heads) beneath the syncytiotrophoblast layer in the region of the invagination and their paucity away from the invagination (*). When an invagination is sectioned perpendicular to its long axis (S-S), it appears as an inclusion (TI), with dark syncytiotrophoblast nuclei in its center surrounded by

cytotrophoblasts (arrow heads). IVS Intervillous space. (b) Diagram of a villus cross section showing the outer syncytiotrophoblast layer (blue) and inner cytotrophoblast layer (pink cells) with a trophoblast invagination (I) and inclusion (TI) illustrating the relevant morphology and disposition of cytotrophoblasts in the region of the invagination. (From Anderson GM, Jacobs-Stannard A, Chawarska K, Volkmar FR, Kliman HJ. (2007) Placental Trophoblast Inclusions in Autism Spectrum Disorder, *Biological Psychiatry*, 61:487–491, used with permission)

The only documented nongenetic cause for trophoblast inclusions appears to be gestational diabetes. In such cases, it is believed that increased glucose and insulin levels lead to increased cytotrophoblast proliferation, which as described above, would lead to invagination of the trophoblast bilayer. This is consistent with the observation that cultured cytotrophoblasts grow better in high glucose media compared to normal media (Kliman et al. 1986). It is also of interest that diabetics have a much higher fetal anomaly rate (Correa et al. 2008), suggesting that the high glucose insulin environment leads not only to abnormalities in the placenta but also in the fetus. This has been confirmed in rat gestations where it has been shown that high glucose levels alone can lead to both deformed fetuses and pregnancy terminations (Reece et al. 1994).

Trophoblast Inclusions and Autism

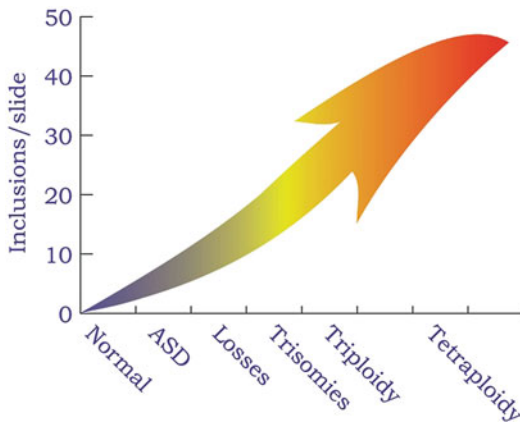
It was in the context of trophoblast inclusions as a general marker of genetic and developmental abnormalities that the association of trophoblast inclusions and autism was first suggested. The linkage was first observed anecdotally in two cases of Asperger's syndrome, which was then followed by a retrospective study. Trophoblast inclusions were found in significantly more

cases of ASD than would be predicted from the normal population (Anderson et al. 2007).

This result fits into the consensus view that ASD is largely genetically based. It also suggests that this seemingly polygenetic, heterogeneous condition may ultimately be caused by subtle abnormalities in common morphogenetic processes, such as bilayer folding.

A Problem of Folding

There are only a few developmental processes in the embryology tool box. These include cell proliferation, cell death, cell migration, cell hypertrophy, differentiation, fusion, and cellular dissociation. With these tools, all the various organs and body parts are made. One of the common problems in development is how to increase surface area. There are two basic solutions to this problem: increased branching or increased folding. In both cases, these are most often achieved by differential growth of layers of cells, which due to increased tension being built up in one layer or another, results in bending. This is exactly how the placenta forms new buds and villi to make the villus tree. It is also how the heart forms, the bronchi branch in the lung, the kidney tubules form, the gut surface area increases, and most dramatically how the brain, especially the human brain, fits into the skull.



Placenta, Fig. 9 *Trophoblast inclusions as a function of genetic abnormality severity.* Normal placentas rarely exhibit trophoblast inclusions. As the degree of genetic abnormality increases, the more trophoblast inclusions can be identified per unit area of placenta examined. The earlier the pregnancy loss, the more frequent the inclusions. Many pregnancy losses reveal normal karyotypes; however, a significant number of trophoblast inclusions can be seen in many of these cases. The trisomies (e.g., trisomy 21, 13, 18) may or may not lead to an early pregnancy loss, depending on which chromosome is affected. In very abnormal gestations, such as triploidy and tetraploidy, as many as 50 or more inclusions per slide can be seen. ASD, a subtle condition that does not lead to pregnancy loss, usually exhibits only 1–3 trophoblast inclusions per placenta slide examined

Since the upper limit of human skull diameter is directly related to the size of the female pelvis, increases in brain surface area could only be achieved by folding. And the human brain is one of the most folded brains in the animal kingdom (Hilgetag and Barbas 2006). Behind this folded structure are the cellular processes that lead to differential growth in the many neural layers that make up the brain. Could the developmental abnormalities that lead to abnormal folding and trophoblast inclusions in the placenta also be at work in the brains of ASD children?

Researchers have demonstrated significantly abnormal brain folding in children on the autism spectrum using surface mapping and magnetic resonance imaging (MRI) (Awate et al. 2008; Kates et al. 2009; Nordahl et al. 2007). These abnormalities may in part explain the observation of increased head size in ASD children (Awate et al. 2008; McCaffery and Deutsch 2005). Basically, with less folding, the brain tissue has no

other option but to expand the skull to make up for the less compact nature of the neural tissue. Translating this gross macroscopic observation to cellular processes and ultimately to abnormal behaviors is much harder. However, neuropathologists have described abnormalities in ASD children at the tissue level of the brain that may be associated with these macroscopic changes (Bauman and Kemper 2003, 2005; Kemper and Bauman 1998; Whitney et al. 2009).

Future Directions

If one of the basic pathologies in ASD is related to problems in tissue folding, then we should see evidence for this in any tissue where folding is a critical part of the anatomy or function of that tissue. Some organs may be impervious to low frequencies of misfolding, such as the liver, which does not have a critical requirement for multilayer cellular organization. Other organs, such as the gut, may not function as well if its folded structure is disrupted. If commonalities can be ascertained in cases of ASD, then candidate genes could be sought that control and regulate these processes. Further, in the future, one might anticipate that genetic or medical interventions might be forthcoming that can ameliorate the abnormalities that may exist in ASD children. In the meantime, knowing that trophoblast inclusions are related to ASD could lead to early identification and intervention before overt symptoms arise.

See Also

► [Developmental Change](#)

References and Reading

- Anderson, G. M., Jacobs-Stannard, A., Chawarska, K., Volkmar, F. R., & Klinman, H. J. (2007). Placental trophoblast inclusions in autism spectrum disorder. *Biological Psychiatry*, 61, 487–491.
- Awate, S. P., Win, L., Yushkevich, P., Schultz, R. T., & Gee, J. C. (2008). 3D cerebral cortical morphometry in autism: Increased folding in children and adolescents in

- frontal, parietal, and temporal lobes. *International Conference on Medical Image Computing and Computer-Assisted Intervention*, 11, 559–567.
- Bauman, M. L., & Kemper, T. L. (2003). The neuropathology of the autism spectrum disorders: What have we learned? *Novartis Foundation Symposium*, 251, 112–122; discussion 122–128, 281–297.
- Bauman, M. L., & Kemper, T. L. (2005). Neuroanatomic observations of the brain in autism: A review and future directions. *International Journal of Developmental Neuroscience*, 23, 183–187.
- Correa, A., Gilboa, S. M., Besser, L. M., Botto, L. D., Moore, C. A., Hobbs, C. A., et al. (2008). Diabetes mellitus and birth defects. *American Journal of Obstetrics & Gynecology*, 199, 237.e1–237.e9.
- Gillett, C. E., & Barnes, D. M. (1998). Demystified . . . cell cycle. *Molecular Pathology*, 51, 310–316.
- Hilgetag, C. C., & Barbas, H. (2006). Role of mechanical factors in the morphology of the primate cerebral cortex. *PLoS Computational Biology*, 2, e22.
- Honore, L., Dill, F. J., & Poland, B. J. (1976). Placental morphology in spontaneous human abortions with normal and abnormal karyotypes. *Teratology*, 14, 151–166.
- Huppertz, B., Tews, D. S., & Kaufmann, P. (2001). Apoptosis and syncytial fusion in human placental trophoblast and skeletal muscle. *International Review of Cytology*, 205, 215–253.
- Kates, W. R., Ikuta, I., & Burnette, C. P. (2009). Gyrfication patterns in monozygotic twin pairs varying in discordance for autism. *Autism Research*, 2, 267–278.
- Kemper, T. L., & Bauman, M. (1998). Neuropathology of infantile autism. *Journal of Neuropathology & Experimental Neurology*, 57, 645–652.
- Kliman, H. J. (1999). Trophoblast to human placenta. In E. Knobil & J. D. Neill (Eds.), *Encyclopedia of reproduction* (Vol. 4, pp. 834–846). San Diego: Academic.
- Kliman, H. J., & Segel, L. (2003). The placenta may predict the baby. *Journal of Theoretical Biology*, 225, 143–145.
- Kliman, H. J., Nestler, J. E., Sermasi, E., Sanger, J. M., & Strauss, J. F., 3rd. (1986). Purification, characterization, and in vitro differentiation of cytotrophoblasts from human term placentae. *Endocrinology*, 118, 1567–1582.
- Kliman, H. J., McSweet, J. C., Franco, A., Ying, X., Zhao, Y., & Stetten, G. (2003). Trophoblast inclusions are rare in elective terminations and normal deliveries, but common in cases with karyotypic abnormalities. *Fertility and Sterility*, 80, 88–88.
- Malassine, A., Frendo, J.-L., & Evain-Brion, D. (2010). Trisomy 21-affected placentas highlight prerequisite factors for human trophoblast fusion and differentiation. *International Journal of Developmental Biology*, 54, 475–482.
- McCaffery, P., & Deutsch, C. K. (2005). Macrocephaly and the control of brain growth in autistic disorders. *Progress in Neurobiology*, 77, 38–56.
- Nordahl, C. W., Dierker, D., Mostafavi, I., Schumann, C. M., Rivera, S. M., Amaral, D. G., et al. (2007). Cortical folding abnormalities in autism revealed by surface-based morphometry. *The Journal of Neuroscience*, 27, 11725–11735.
- Novak, R., Agamanolis, D., Dasu, S., Igel, H., Platt, M., Robinson, H., et al. (1988). Histologic analysis of placental tissue in first trimester abortions. *Pediatric Pathology*, 8, 477–482.
- Reece, E. A., Pinter, E., Homko, C., Wu, Y. K., & Naftolin, F. (1994). The yolk sac theory: Closing the circle on why diabetes-associated malformations occur. *Journal of the Society for Gynecologic Investigation*, 1, 3–13.
- Rejniak, K. A., Kliman, H. J., & Fauci, L. J. (2004). A computational model of the mechanics of growth of the villous trophoblast bilayer. *Bulletin of Mathematical Biology*, 66, 199–232.
- Silvestre, E., Cusi, V., Borrás, M., & Antich, J. (1996). Cytogenetic and morphologic findings in chorionic villi from spontaneous abortions. *Birth Defects Original Article Series*, 30, 353–357.
- Zsulman, A. E. (1984). Syndromes of hydatidiform moles. Partial vs. complete. *The Journal of Reproductive Medicine*, 29, 788–791.
- Zsulman, A. E., Philippe, E., Boue, J. G., & Boue, A. (1981). Human triploidy: Association with partial hydatidiform moles and nonmolar conceptuses. *Human Pathology*, 12, 1016–1021.
- van Lijnschoten, G., Arends, J. W., De La Fuente, A. A., Schouten, H. J., & Geraedts, J. P. (1993). Intra- and inter-observer variation in the interpretation of histological features suggesting chromosomal abnormality in early abortion specimens. *Histopathology*, 22, 25–29.
- Whitney, E. R., Kemper, T. L., Rosene, D. L., Bauman, M. L., & Blatt, G. J. (2009). Density of cerebellar basket and stellate cells in autism: Evidence for a late developmental loss of Purkinje cells. *Journal of Neuroscience Research*, 87, 2245–2254.
- Zhang, Y. X., Zhang, Y. P., Gu, Y., Guan, F. J., Li, S. L., Xie, J. S., et al. (2009). Genetic analysis of first-trimester miscarriages with a combination of cytogenetic karyotyping, microsatellite genotyping and arrayCGH. *Clinical Genetics*, 75, 133–140.

PL-ADOS

► Prelinguistic Autism Diagnostic Observation Schedule

Planning and Placement Team (PPT)

► Interdisciplinary Team

Plasma Adiponectin and Autism Spectrum Disorder

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Synonyms

Adipocytokine

Definition

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by social and communication deficits and repetitive behaviors (Lyall et al. 2017). There are currently no biomarkers for ASD and diagnosis is based mainly on children's altered behaviors, which is not evident until 2–4 years. Yet, early detection and intervention is important for successful ASD management. Therefore, there has been a heightened interest to identify reliable markers that can facilitate early risk assessment and be a potential target for therapeutic interventions (Ashwood et al. 2006).

The precise mechanism underlying ASD's pathophysiology is still elusive; however, emerging evidence suggests that both metabolic (Li et al. 2016; Panjwani et al. 2019) and immune system (Brucato et al. 2017; Raghavan et al. 2019b) may be implicated. The interface between immune and nervous system is complex, and extensive communication between these systems occur during health and disease states (Ashwood et al. 2006). The immune system, once thought to

be only responsible for defending against invading pathogens, has now shown to play an important role in neurodevelopment and regulation of neural plasticity (Heuer et al. 2019). Several studies have suggested immune abnormalities in those with ASD (Ashwood et al. 2011; Ghaffari et al. 2016; Molloy et al. 2006; Raghavan et al. 2018). This is also supported by postmortem studies that have reported an increase in pro-inflammatory cytokines in the brain of subjects with ASD (Vargas et al. 2005).

Adipose tissue, beyond its energy storing capabilities, functions as a highly active endocrine organ that orchestrates inflammatory response through the production of adipocytokines (Hansen-Pupp et al. 2015; Mazaki-Tovi et al. 2009a; Villarreal-Molina and Antuna-Puente 2012). Adipose tissue releases a variety of inflammatory factors including leptin, adiponectin, and resistin that control various physiological systems (Ghaffari et al. 2016). Adiponectin is the most abundantly produced cytokine by adipose tissue, which exerts anti-inflammatory action on a number of cell types (Mazaki-Tovi et al. 2009b; Ouchi and Walsh 2007). It is downregulated in response to pro-inflammatory cytokines and oxidative stress (Michalska-Jakubus et al. 2019). Adiponectin has neuroprotective functions, with evidence suggesting altered adiponectin levels in neurologic disorders with an underlying etiology involving metabolic and inflammatory pathways (Ghaffari et al. 2016; Molloy et al. 2006). Adiponectin receptors are highly expressed in different brain regions, and its presence in the central nervous system has been attributed to neuroprotective and antidepressant properties (Bloemer et al. 2018).

Emerging research has assessed whether adiponectin levels are altered in individuals with ASD. A cross-sectional study conducted in Japanese boys reported that adiponectin levels are reduced in subjects with ASD than age-matched neurotypical controls (Fujita-Shimizu et al. 2010). The authors also reported a negative correlation between adiponectin levels and an abnormality in social interactions but not with restricted or repetitive behaviors (Fujita-Shimizu et al. 2010). Another cross-sectional study conducted in a

tertiary health facility reported no difference in adiponectin levels between ASD subjects and controls, who were matched for age, sex, and maternal age; however, they showed a negative correlation between adiponectin levels and Social Responsiveness Scale, a measure of social impairment in a social setting (Rodrigues et al. 2014). A longitudinal study conducted in pubertal children showed that adiponectin levels did not significantly differ between age- and sex-matched neurotypical and ASD children at baseline and 1 year after (Blardi et al. 2009). All these studies assessed adiponectin in children diagnosed with ASD and it was unclear if alteration in adiponectin levels preceded ASD diagnosis. A prospective birth cohort study published recently answered this question; this US-based study showed that both cord and early childhood adiponectin levels prior to ASD diagnosis were inversely associated with ASD risk (Raghavan et al. 2019a). Specifically, the association between cord adiponectin levels and ASD was robust suggesting that alteration in adiponectin levels may have its origins in fetal life.

In summary, the available literature suggests that adiponectin levels may be altered in subjects with ASD; however, the current literature is limited by small sample sizes, cross-sectional study design, heterogeneity in age of adiponectin measurement, and lack of adjustment of key confounders – all of which reduce the confidence in the strength of association. Adiponectin as an ASD biomarker is not yet ready for prime time, and more prospective birth cohort studies are warranted to understand the role of adiponectin as well as other adipocytokines such as leptin (Raghavan et al. 2018), in the development of ASD and underlying mechanisms.

References and Reading

- Ashwood, P., Wills, S., & Van de Water, J. (2006). The immune response in autism: A new frontier for autism research. *Journal of Leukocyte Biology*, 80(1), 1–15.
- Ashwood, P., Krakowiak, P., Hertz-Picciotto, I., Hansen, R., Pessah, I., & Van de Water, J. (2011). Elevated plasma cytokines in autism spectrum disorders provide evidence of immune dysfunction and are associated with impaired behavioral outcome. *Brain, Behavior, and Immunity*, 25(1), 40–45.
- Blardi, P., de Lalla, A., D'Ambrogio, T., Vonella, G., Ceccatelli, L., Auteri, A., et al. (2009). Long-term plasma levels of leptin and adiponectin in Rett syndrome. *Clinical Endocrinology*, 70(5), 706–709.
- Bloemer, J., Pinky, P. D., Govindarajulu, M., Hong, H., Judd, R., Amin, R. H., et al. (2018). Role of adiponectin in central nervous system disorders. *Neural Plasticity*, 2018, 4593530.
- Brucato, M., Ladd-Acosta, C., Li, M., Caruso, D., Hong, X., Kaczaniuk, J., et al. (2017). Prenatal exposure to fever is associated with autism spectrum disorder in the Boston birth cohort. *Autism Research*, 10(11), 1878–1890.
- Fujita-Shimizu, A., Suzuki, K., Nakamura, K., Miyachi, T., Matsuzaki, H., Kajizuka, M., et al. (2010). Decreased serum levels of adiponectin in subjects with autism. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 34(3), 455–458.
- Ghaffari, M. A., Mousavinejad, E., Riahi, F., Mousavinejad, M., & Afsharmanesh, M. R. (2016). Increased serum levels of tumor necrosis factor- α , Resistin, and Visfatin in the children with autism spectrum disorders: A case-control study. *Neurology Research International*, 2016, 9060751.
- Hansen-Pupp, I., Hellgren, G., Hard, A. L., Smith, L., Hellstrom, A., & Lofqvist, C. (2015). Early surge in circulatory adiponectin is associated with improved growth at near term in very preterm infants. *The Journal of Clinical Endocrinology and Metabolism*, 100(6), 2380–2387.
- Heuer, L. S., Croen, L. A., Jones, K. L., Yoshida, C. K., Hansen, R. L., Yolken, R., et al. (2019). An exploratory examination of neonatal cytokines and chemokines as predictors of autism risk: The early markers for autism study. *Biological Psychiatry*, 86(4), 255–264.
- Li, M., Fallin, M. D., Riley, A., Landa, R., Walker, S. O., Silverstein, M., et al. (2016). The association of maternal obesity and diabetes with autism and other developmental disabilities. *Pediatrics*, 137(2), e20152206.
- Lyall, K., Croen, L., Daniels, J., Fallin, M. D., Ladd-Acosta, C., Lee, B. K., et al. (2017). The changing epidemiology of autism spectrum disorders. *Annual Review of Public Health*, 38, 81–102.
- Mazaki-Tovi, S., Romero, R., Vaisbuch, E., Erez, O., Mittal, P., Chaiworapongsa, T., et al. (2009a). Dysregulation of maternal serum adiponectin in preterm labor. *The Journal of Maternal-Fetal & Neonatal Medicine*, 22(10), 887–904.
- Mazaki-Tovi, S., Romero, R., Vaisbuch, E., Kusanovic, J. P., Erez, O., Gotsch, F., et al. (2009b). Maternal serum adiponectin multimers in preeclampsia. *Journal of Perinatal Medicine*, 37(4), 349–363.
- Michalska-Jakubus, M., Sawicka, K., Potembska, E., Kowal, M., & Krasowska, D. (2019). Clinical associations of serum leptin and leptin/adiponectin ratio in systemic sclerosis. *Postępy Dermatologii i Alergologii*, 36(3), 325–338.

- Molloy, C. A., Morrow, A. L., Meinzen-Derr, J., Schleifer, K., Dienger, K., Manning-Courtney, P., et al. (2006). Elevated cytokine levels in children with autism spectrum disorder. *Journal of Neuroimmunology*, 172(1–2), 198–205.
- Ouchi, N., & Walsh, K. (2007). Adiponectin as an anti-inflammatory factor. *Clinica Chimica Acta*, 380(1–2), 24–30.
- Panjwani, A. A., Ji, Y., Fahey, J. W., Palmer, A., Wang, G., Hong, X., et al. (2019). Maternal obesity/diabetes, plasma branched-chain amino acids, and autism spectrum disorder risk in urban low-income children: Evidence of sex difference. *Autism Research*, 1562–1573.
- Raghavan, R., Zuckerman, B., Hong, X., Wang, G., Ji, Y., Paige, D., et al. (2018). Fetal and infancy growth pattern, cord and early childhood plasma leptin, and development of autism spectrum disorder in the Boston birth cohort. *Autism Research*, 11(10), 1416–1431.
- Raghavan, R., Fallin, M. D., Hong, X., Wang, G., Ji, Y., Stuart, E. A., et al. (2019a). Cord and early childhood plasma adiponectin levels and autism risk: A prospective birth cohort study. *Journal of Autism and Developmental Disorders*, 49(1), 173–184.
- Raghavan, R., Helfrich, B. B., Cerda, S. R., Burd, I., Wang, G., Hong, X., et al. (2019b). Preterm birth subtypes, placental pathology findings, and risk of neurodevelopmental disabilities during childhood. *Placenta*, 83, 17–25.
- Rodrigues, D. H., Rocha, N. P., Sousa, L. F., Barbosa, I. G., Kummer, A., & Teixeira, A. L. (2014). Changes in adipokine levels in autism spectrum disorders. *Neuropsychobiology*, 69(1), 6–10.
- Vargas, D. L., Nascimbene, C., Krishnan, C., Zimmerman, A. W., & Pardo, C. A. (2005). Neuroglial activation and neuroinflammation in the brain of patients with autism. *Annals of Neurology*, 57(1), 67–81.
- Villarreal-Molina, M. T., & Antuna-Puente, B. (2012). Adiponectin: Anti-inflammatory and cardioprotective effects. *Biochimie*, 94(10), 2143–2149.

Plasticity, Neural

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Synonyms

Cortical remapping; Cortical rewiring; Neural reorganization

Structure

Autism spectrum disorders are characterized by profound impairments in language acquisition and social interaction that are often comorbid with repetitive behaviors and sensory and motor abnormalities. Communicative and social skills emerge in early childhood and grow exponentially in complexity. The establishment of appropriate neural circuitry in the cerebral cortex subserving language acquisition, social interaction, and indeed, all aspects of cortical function, including vision and motor control, relies on the proper execution of genetic programs that regulate neural proliferation, migration, differentiation, axon guidance, and recognition of synaptic targets. A characteristic conserved between these disparate systems is that this circuitry is ineffective at birth. Sensory experience within specified sensitive or so-called “critical” periods during development sculpts the fine structure and function of cortical circuitry. However, the timing and influence of these critical periods varies dramatically among the functional domains of the cerebral cortex. This entry examines recent advances in our understanding of how autism-candidate genes regulate neural growth, synaptic plasticity, and the closure of critical periods.

Anatomical Plasticity of Neural Structure

The establishment of precise neural circuitry in the cortex hinges on five major events. First, neurons must be born in correct numbers. Second, these neurons must migrate to appropriate locations to establish the basic lamination of the cortex. Third, these neurons must grow specialized processes – dendrites that integrate information coming into the neuron and axons that provide the information output to other neurons in the cortical network. Fourth, axons must identify appropriate target dendrites and establish synapses. Fifth, these newly formed synapses are stabilized or eliminated by neural activity largely driven by sensory experience. In the cortex this last step of experience-dependent changes in the number and strength of synapses peaks during critical periods. In this entry, we examine recent

work on synaptic and circuit plasticity in mice with genetic mutations in genes associated with autism.

Function

Critical Periods of Experience-Dependent Plasticity in the Developing Human Brain

Critical periods are present in all areas of cortex examined from the evolutionarily earliest primary sensory cortices to the phylogenetically more recent frontal cortices. Perhaps the most well known examples are critical periods in sight and language. Poor vision during childhood results in a permanent reduction in high acuity vision – a condition known as amblyopia. In children born with congenital cataracts, for example, restoration of optical clarity through the lens will restore sight if the surgery is completed before about age 5. The older the age at treatment the worse the final outcome (Holmes et al. 2011). If corrective surgery is delayed past puberty the patient will remain effectively blind to that eye, despite the restoration of normal optical clarity through the eye and otherwise normal signal transduction in the retina. This permanent loss of sight results from the development and ultimate crystallization of cortical circuitry exposed to poor vision. Vision, therefore, profoundly shapes cortical circuitry, effectively establishing our sense of sight by its actions on cortical connectivity, but only during a limited period of adolescence. Cataracts that form in adults do not induce permanent deficits in sight once removed. Similarly, language acquisition is entirely dependent on exposure to language and its actions on cortical wiring. Socially isolated children fail to acquire normal language if initial exposure occurs past puberty (Fromkin et al. 1974). A second language learned after puberty is heavily accented with improper intonation, whereas fluency of speech and comprehension are readily attained if the second language is learned before 7 years of age (Johnson and Newport 1991). The critical period for learning social interactions, which is heavily dependent on frontal cortical circuitry, is not well defined, but in humans it can be argued that it extends throughout life.

Typical Development of Human Frontal and Temporal Cortex

The closure of cortical critical periods is tied to cessation of neural growth, the strengthening of cortical inhibition (Hensch 2005), and the establishment of axonal myelination (McGee et al. 2005). In frontal cortex, the region most severely affected in autism and the region of the brain that regulates executive function, neurons are remarkably immature at birth. Dendrites of frontal cortical pyramidal neurons are very rudimentary, extending to perhaps 3% of their ultimate adult size (Huttenlocher 1990). Dendritic growth proceeds slowly, reaching 35% of adult size by 6 months of age, 50% of final size by 2 years of age, and are adult-like by 7–10 years of age (Huttenlocher 1990). The slow, progressive growth of dendrites and concomitant increase in neuropil are reflected in functional brain imaging studies demonstrating a progressive thickening of frontal gray matter that peaks at about age 12 (Giedd et al. 1999). Notably, the language areas of frontal and temporal cortex, another region that is thought to malfunction in autistic patients, continue to thicken well past puberty (Sowell et al. 2004). The prolonged development of these cortical areas may reflect their phylogenetically recent emergence in the forebrain (Hill and Walsh 2005). Relative to phylogenetically older sensory cortices, frontal cortex has experienced a tremendous expansion from basal shrew-like mammals through primates and ultimately humans. Paralleling this expansion is the emergence of complex social structure and language – both of which require decades of learning to master.

The establishment of synapses on growing dendrites and their subsequent refinement by experience also occurs at a slower pace in frontal cortex than in sensory cortices. Whereas synaptic density peaks at 3 months of age in primary auditory and visual cortices, it peaks at 3.5 years of age in frontal cortex. It is widely held that experience sculpts cortical circuitry largely by stabilizing some synapses while eliminating others. This process of synaptic elimination is complete by 12 years of age in primary sensory cortices, but is more gradual in frontal cortex, continuing

throughout mid-adolescence (Huttenlocher and Dabholkar 1997). Myelination of subcortical white matter is perhaps most prolonged, continuing into the second decade of life (Giedd et al. 1999; Klingberg et al. 1999).

The slow, progressive growth and refinement of this cortical circuitry enables sensory experience to profoundly impact the final structure and function of frontal and temporal cortices. As discussed below, these processes are accelerated in autism, forcing a premature closure of critical periods for language and social learning.

Pathophysiology

Development of Frontal and Temporal Cortex in Autism

There are no postmortem studies of neural anatomy in autistic children ages 2–4, the typical age of diagnosis. Thus, we do not know how the fine structure of neurons and refinements in synapse number and connectivity are impacted by autism. However, a growing number of brain imaging studies indicate that gray matter in frontal and temporal cortices grows precociously in the autistic brain. At the earliest ages measured, 2.5 years of age, the thickness of frontal and temporal gray matter is already 6% and 9% greater, respectively, than typical, and this accelerated growth continues through at least 5 years of age (Schumann et al. 2010). Despite this early overgrowth, there is some evidence that it is short lived – frontal and temporal gray matter increase by only 1% between ages 6 and 8 in autistic children during which time the normally developing brain experiences increases of 17–20% (Carper et al. 2002). Thus, frontal and temporal gray matter thickness increases rapidly in the autistic brain, reaching adult thickness a decade or more ahead of normal pace. The precocious overgrowth of the autistic brain is perhaps most apparent when noting that the average brain weight in 3–5-year-old autistic children is equivalent to that of normal adults (Courchesne et al. 1999). The normally slow growth of cortical gray matter parallels the emergence and slow acquisition of language and social skills. The premature cessation of cortical growth

in autism would almost certainly impair their refinement.

Synaptic density measurements support a view of premature closure of critical periods. The density of dendritic spines – the postsynaptic specializations of dendrites that receive excitatory inputs – is increased in autism (Hutsler and Zhang 2010; van Spronsen and Hoogenraad 2010), suggesting an absence of the normal pruning processes necessary for the refinement of circuit function.

How the signaling pathways that drive autism impact the structure and function of individual neurons and their synapses can only be achieved by studying mouse models of autism. We discuss recent advances in this line of investigation below.

Synaptic Plasticity in Mice Carrying Mutations in Autism-Candidate Genes

The maturation of appropriate cortical connectivity and function depends on long-term changes in synaptic strength. These changes include enhancements in synaptic strength (long-term potentiation; LTP) and reductions in synaptic strength (long-term depression; LTD), as well as the formation of new synapses and the retraction of others. It is conceivable that abnormal synaptic plasticity could contribute to deficits in circuit refinement and behavioral inflexibility seen in autistic patients. To discover whether alterations in synaptic plasticity play a role in the cognitive and behavioral deficits suffered by patients with autism, studies of cortical and hippocampal LTP and LTD have been performed in a number of mouse models with deletions of autism-related genes; these studies are summarized in Table 1. Additionally, recent advances in nonlinear microscopy permit longitudinal imaging studies of synaptic structure in vivo in mouse models of autism.

Altered Synaptic Strengthening

Mice engineered to have the R451C neuroligin-3 mutation found in a small number of patients with autism, have social behavioral deficits and enhanced learning (Tabuchi et al. 2007; Etherton et al. 2011). They have enhanced inhibitory transmission in the cortex, and enhanced excitatory transmission in the hippocampus (Etherton et al.).

Plasticity, Neural, Table 1 Autism related genes and neural plasticity

Gene	Protein function	Syndrome	Synaptic plasticity/ function	Learning deficits	Molecular features
<i>CACNA1C</i>	Calcium channel	Timothy syndrome	Impaired Schaffer collateral late LTP	Impaired hippocampal spatial memory	
<i>CNTNAP2</i>	Cell adhesion molecule	Autism/epilepsy/MR	—	Differential expression in bird song learning nuclei	Clusters potassium channels
<i>CYFIP1</i>	Synaptic translation regulation	Autism	—	—	Binds and regulates FMR1
<i>DISC1</i>		Autism/Schizophrenia	Impaired hippocampal short term plasticity; short dendrites; decreased spine density	Abnormal spatial working memory	Controls Rac1 activation by NMDA receptors
<i>FMR1</i>	Activity-dependent synaptic translation regulation	Fragile X syndrome	Increased mGluR dependent LTD; impaired LTP in the prefrontal and visual cortex. Decreased spine stability; reduced experience-dependent changes in spine stability	Impaired hippocampal spatial memory. Altered ocular dominance plasticity	
<i>NGLUT3</i>	Glucose transporter	Autism		Abnormal spatial and working memory	
				Cognitive inflexibility	
<i>Neurologin3</i>	Synaptic adhesion molecule	Autism/MR	Increased cortical inhibition; Increased hippocampal excitation. Increased hippocampal LTP	Improved spatial memory	
				Social Social behavior deficit	
<i>MECP2</i>	Transcriptional regulator	Rett Syndrome	Impaired cortical and hippocampal LTP. Impaired hippocampal LTD	Impaired Hippocampus-dependent spatial memory, contextual fear memory, and social behavior	Binds methylated cytosine to regulate activity dependent transcription
<i>PTEN</i>	Cell/dendritic growth regulator	Autism/MR; Cowden syndrome; or Bannayan-Riley-Ruvalcaba syndrome	Impaired hippocampal LTD; increased dendritic growth, decreased spine stability; increased spine density	Social behavior deficits	Inhibits activation of mTOR pathway
<i>TSC1</i>	Cell growth	Tuberous sclerosis	decreased spine density; increased AMPAR currents	Impaired hippocampal spatial memory	Inhibits activation of mTOR pathway

(continued)

Plasticity, Neural, Table 1 (continued)

Gene	Protein function	Syndrome	Synaptic plasticity/ function	Learning deficits	Molecular features
<i>TSC2</i>	Cell growth	Tuberous sclerosis	Abnormal induction of hippocampal Late-LTP; decreased spine density	Impaired hippocampal learning behavior	Inhibits activation of mTOR pathway
<i>UBE3a</i>	Proteosomal regulator	Autism; Angelman syndrome	Impaired visual cortical LTD; decreased spine density	Impaired ocular dominance plasticity	E3 ubiquitin ligase
<i>Shank3</i>	Synaptic adhesion molecule	Autism	Impaired hippocampal NMDAR LTP, LTD. Enhanced mGluR dependent LTD	Social behavioral deficits	

In addition, they show enhanced hippocampal LTP, likely because of an upregulation of the NMDA receptor NR2B subunit (Etherton et al.).

Mice with Shank3 C-terminus truncation mimicking the human autism-related mutation have loss of Shank3 at synapses. These mice have social behavioral deficits, loss of NMDA receptors at synapses, and loss of NMDAR dependent LTP (Bangash et al. 2011).

Deletion of CACNA1C, encoding a calcium channel mutated in Timothy Syndrome, an autistic spectrum syndrome, results in deficient Schaffer Collateral Late-LTP in the hippocampus (Moosmang et al. 2005). Deletion of MECP2, encoding a chromatin binding protein mutated in Rett Syndrome, another autism spectrum disorder, also results in deficient hippocampal (and cortical) LTP (Moretti et al. 2006; Weng et al. 2011). Conversely, loss of activity induced phosphorylation of MECP2 results in enhanced binding of MECP2 to target promoters, results in enhanced hippocampal LTP, and enhanced learning in hippocampal-dependent tasks (Li et al. 2011). Deletion of FMR1, the gene implicated in Fragile X syndrome, results in decreased LTP in the visual cortex (Wilson and Cox 2007), and loss of spike-timing dependent LTP in the prefrontal cortex (Meredith et al. 2007), but enhanced hetero-synaptic LTP in the hippocampus (Connor et al. 2011). Conversely, mice with deletions in TSC2, a gene mutated in the autism-related syndrome, tuberous sclerosis, show abnormal induction of late-LTP by stimuli that only induce early-LTP in control mice (Ehninger et al. 2008).

Altered Synaptic Weakening

MECP2 knockout mice (Moretti et al. 2006), as well as mice with conditional deletion of PTEN, a regulator of the mTOR pathway that is mutated in a significant cohort of patients with autism and extreme macrocephaly, show impaired hippocampal LTD (Jurado et al. 2010). PTEN conditional knockout mice show dramatic social behavioral deficits as well as seizures. Mice with deletions of UBE3a, an E3 ubiquitin ligase, implicated in the autism-related Angelman syndrome show impaired LTD in visual cortex (Yashiro et al. 2009). Consistent with a role of LTD in experience-dependent plasticity in the cortex, these mice show impaired ocular dominance plasticity when challenged with monocular deprivation (Yashiro et al.). In contrast to the above models, mice with deletions of FMR1, the gene implicated in Fragile X syndrome as well as Shank3 mutant mice have *increased* mGluR-dependent LTD in the hippocampus (Huber et al. 2002; Hou et al. 2006). FMR1 mice also show abnormal visual cortical plasticity (Dolen et al. 2007) and both FMR1 and Shank3 mice show impairments on a number of spatial and social behaviors (Bangash et al. 2011; Krueger et al. 2011).

Deficits in Synapse Number

Changes in the total number of synapses made onto single neurons can profoundly impact the firing properties of these cells and alter network function. In cultured neurons, loss of TSC1 or TSC2 decreases the density of dendritic spines,

the morphological correlates of synaptic contacts. Decreases in cortical spine density are also observed following loss of UBE3a (Dindot et al. 2008; Sato and Stryker 2010) or DISC1 point mutation (Lee et al. 2011) in vivo. Notably, spine density is not affected by mutations of MET, a receptor upstream of the AKT signaling pathway. Taken together these observations suggest that either the normal establishment of spines is impaired or that their subsequent stabilization is compromised. Recent in vivo longitudinal imaging studies, presented below, support the latter view.

Deficits in Synaptic Stability

In addition to impairments in synaptic strength, a growing body of research implicates deficits in the physical growth and elimination of synapses in autism. Recent advances in nonlinear microscopy have enabled investigations of synaptic stability in wild-type and mutant mice. Using 2-photon excitation, the fine structure of individual neurons, their dendrites, and the spines emerging from these dendrites can be visualized in vivo. Dendritic spines are the postsynaptic element of excitatory synapses, and thus each spine represents a single synapse. Because this approach does not damage the imaged cortex, these same neurons, dendrites, and dendritic spines can be reimaged daily or weekly and the fates of individual spines followed for months. This approach has been used to longitudinally image dendrites and dendritic spines in mice with mutations in Pten and FMR1. In both mutants, the lifetimes of spines are reduced, indicating that cortical connectivity is far more kinetic and thus less stable. Notably, spine stability was less sensitive to changes in experience in FMR1 mutant mice. These observations suggest that the refinement of synaptic connections that is the hallmark of the critical period cortex is driven more by intrinsic activity patterns than by sensory experience.

Conclusion

Changes in neural structure, synaptic strength, and synaptic density occur throughout life but

are most pronounced after the onset of sensory experience. Observations from mice harboring mutations of autism-candidate genes indicate that synapse number and stability are reduced and changes in their strength and stability are less sensitive to the pattern of neural activity. Impairments in use-dependent synaptic plasticity would severely impact the final structure and function of neural circuitry in all areas of the brain, but particularly so in cortex. Recent evidence showing that autism-like behaviors can be induced by altering the balance of excitation and inhibition in cortex in adult mice or by manipulating autism-candidate genes in adult mice supports a view of the disease in which postnatal plasticity, rather than initial patterning, is a prime driver of abnormal social behavior in autistic individuals.

See Also

- [Synaptic Pruning](#)
- [Synaptogenesis](#)

References and Reading

- Bangash, M. A., et al. (2011). Enhanced polyubiquitination of Shank3 and NMDA receptor in a mouse model of autism. *Cell*, 145, 758–772.
- Carper, R. A., Moses, P., Tighe, Z. D., & Courchesne, E. (2002). Cerebral lobes in autism: Early hyperplasia and abnormal age effects. *NeuroImage*, 16, 1038–1051.
- Connor, S. A., Hoeffer, C. A., Klann, E., & Nguyen, P. V. (2011). Fragile X mental retardation protein regulates heterosynaptic plasticity in the hippocampus. *Learning and Memory*, 18, 207–220.
- Courchesne, E., Muller, R. A., & Saitoh, O. (1999). Brain weight in autism: Normal in the majority of cases, megalencephalic in rare cases. *Neurology*, 52, 1057–1059.
- Dindot, S. V., Antalffy, B. A., Bhattacharjee, M. B., & Beaudet, A. L. (2008). The Angelman syndrome ubiquitin ligase localizes to the synapse and nucleus, and maternal deficiency results in abnormal dendritic spine morphology. *Human Molecular Genetics*, 17, 111–118.
- Dolen, G., Osterweil, E., Rao, B. S., Smith, G. B., Auerbach, B. D., Chattarji, S., & Bear, M. F. (2007). Correction of fragile X syndrome in mice. *Neuron*, 56, 955–962.
- Ehninger, D., Han, S., Shilyansky, C., Zhou, Y., Li, W., Kwiatkowski, D. J., Ramesh, V., & Silva, A. J. (2008).

- Reversal of learning deficits in a Tsc2+/- mouse model of tuberous sclerosis. *Nature Medicine*, 14, 843–848.
- Etherton, M., Foldy, C., Sharma, M., Tabuchi, K., Liu, X., Shamloo, M., Malenka, R. C., & Sudhof, T. C. (2011). Autism-linked neuroligin-3 R451C mutation differentially alters hippocampal and cortical synaptic function. *Proceedings of the National Academy of Sciences of the United States of America*, 108(33), 13764–13769.
- Fromkin, V., Krashen, S., Curtin, S., Rigler, D., & Rigler, M. (1974). The development of language in Genie: A case of language acquisition beyond the “critical period”. *Brain and Language*, 1, 81–107.
- Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., Paus, T., Evans, A. C., & Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience*, 2, 861–863.
- Hensch, T. K. (2005). Critical period plasticity in local cortical circuits. *Nature Reviews Neuroscience*, 6, 877–888.
- Hill, R. S., & Walsh, C. A. (2005). Molecular insights into human brain evolution. *Nature*, 437, 64–67.
- Holmes, J. M., Lazar, E. L., Melia, B. M., Astle, W. F., Dagi, L. R., Donahue, S. P., Frazier, M. G., Hertle, R. W., Repka, M. X., Quinn, G. E., & Weise, K. K. (2011). Effect of age on response to amblyopia treatment in children. *Archives of Ophthalmology*, 129(11), 1451–1457.
- Hou, L., Antion, M. D., Hu, D., Spencer, C. M., Paylor, R., & Klann, E. (2006). Dynamic translational and proteasomal regulation of fragile X mental retardation protein controls mGluR-dependent long-term depression. *Neuron*, 51, 441–454.
- Huber, K. M., Gallagher, S. M., Warren, S. T., & Bear, M. F. (2002). Altered synaptic plasticity in a mouse model of fragile X mental retardation. *Proceedings of the National Academy of Sciences of the United States of America*, 99, 7746–7750.
- Hutsler, J. J., & Zhang, H. (2010). Increased dendritic spine densities on cortical projection neurons in autism spectrum disorders. *Brain Research*, 1309, 83–94.
- Huttenlocher, P. R. (1990). Morphometric study of human cerebral cortex development. *Neuropsychologia*, 28, 517–527.
- Huttenlocher, P. R., & Dabholkar, A. S. (1997). Regional differences in synaptogenesis in human cerebral cortex. *The Journal of Comparative Neurology*, 387, 167–178.
- Johnson, J. S., & Newport, E. L. (1991). Critical period effects on universal properties of language: The status of subadjacency in the acquisition of a second language. *Cognition*, 39, 215–258.
- Jurado, S., Benoist, M., Lario, A., Knafo, S., Petrok, C. N., & Esteban, J. A. (2010). PTEN is recruited to the postsynaptic terminal for NMDA receptor-dependent long-term depression. *EMBO Journal*, 29, 2827–2840.
- Klingberg, T., Vaidya, C. J., Gabrieli, J. D., Moseley, M. E., & Hedeus, M. (1999). Myelination and organization of the frontal white matter in children: A diffusion tensor MRI study. *Neuroreport*, 10, 2817–2821.
- Krueger, D. D., Osterweil, E. K., Chen, S. P., Tye, L. D., & Bear, M. F. (2011). Cognitive dysfunction and prefrontal synaptic abnormalities in a mouse model of fragile X syndrome. *Proceedings of the National Academy of Sciences of the United States of America*, 108, 2587–2592.
- Lee, F. H., Fadel, M. P., Preston-Maher, K., Cordes, S. P., Clapcote, S. J., Price, D. J., Roder, J. C., & Wong, A. H. (2011). Disc1 point mutations in mice affect development of the cerebral cortex. *Journal of Neuroscience*, 31, 3197–3206.
- Li, H., Zhong, X., Chau, K. F., Williams, E. C., & Chang, Q. (2011). Loss of activity-induced phosphorylation of MeCP2 enhances synaptogenesis, LTP and spatial memory. *Nature Neuroscience*, 14, 1001–1008.
- McGee, A. W., Yang, Y., Fischer, Q. S., Daw, N. W., & Strittmatter, S. M. (2005). Experience-driven plasticity of visual cortex limited by myelin and Nogo receptor. *Science*, 309, 2222–2226.
- Meredith, R. M., Holmgren, C. D., Weidum, M., Burnashev, N., & Mansvelder, H. D. (2007). Increased threshold for spike-timing-dependent plasticity is caused by unreliable calcium signaling in mice lacking fragile X gene FMR1. *Neuron*, 54, 627–638.
- Moosmang, S., Haider, N., Klugbauer, N., Adelsberger, H., Langwieser, N., Muller, J., Stiess, M., Marais, E., Schulla, V., Lacinova, L., Goebbels, S., Nave, K. A., Storm, D. R., Hofmann, F., & Kleppisch, T. (2005). Role of hippocampal Cav1.2 Ca²⁺ channels in NMDA receptor-independent synaptic plasticity and spatial memory. *Journal of Neuroscience*, 25, 9883–9892.
- Moretti, P., Levenson, J. M., Battaglia, F., Atkinson, R., Teague, R., Antalffy, B., Armstrong, D., Arancio, O., Sweatt, J. D., & Zoghbi, H. Y. (2006). Learning and memory and synaptic plasticity are impaired in a mouse model of Rett syndrome. *Journal of Neuroscience*, 26, 319–327.
- Sato, M., & Stryker, M. P. (2010). Genomic imprinting of experience-dependent cortical plasticity by the ubiquitin ligase gene Ube3a. *Proceedings of the National Academy of Sciences of the United States of America*, 107, 5611–5616.
- Schumann, C. M., Bloss, C. S., Barnes, C. C., Wideman, G. M., Carper, R. A., Akshoomoff, N., Pierce, K., Hagler, D., Schork, N., Lord, C., & Courchesne, E. (2010). Longitudinal magnetic resonance imaging study of cortical development through early childhood in autism. *Journal of Neuroscience*, 30, 4419–4427.
- Sowell, E. R., Thompson, P. M., Leonard, C. M., Welcome, S. E., Kan, E., & Toga, A. W. (2004). Longitudinal mapping of cortical thickness and brain growth in normal children. *Journal of Neuroscience*, 24, 8223–8231.
- Tabuchi, K., Blundell, J., Etherton, M. R., Hammer, R. E., Liu, X., Powell, C. M., & Sudhof, T. C. (2007). A neuroligin-3 mutation implicated in autism increases inhibitory synaptic transmission in mice. *Science*, 318, 71–76.
- van Spronsen, M., & Hoogenraad, C. C. (2010). Synapse pathology in psychiatric and neurologic disease.

- Current Neurology and Neuroscience Reports*, 10, 207–214.
- Weng, S. M., McLeod, F., Bailey, M. E., & Cobb, S. R. (2011). Synaptic plasticity deficits in an experimental model of rett syndrome: Long-term potentiation saturation and its pharmacological reversal. *Neuroscience*, 180, 314–321.
- Wilson, B. M., & Cox, C. L. (2007). Absence of metabotropic glutamate receptor-mediated plasticity in the neocortex of fragile X mice. *Proceedings of the National Academy of Sciences of the United States of America*, 104, 2454–2459.
- Yashiro, K., Riday, T. T., Condon, K. H., Roberts, A. C., Bernardo, D. R., Prakash, R., Weinberg, R. J., Ehlers, M. D., & Philpot, B. D. (2009). Ube3a is required for experience-dependent maturation of the neocortex. *Nature Neuroscience*, 12, 777–783.

Play

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Synonyms

Exploratory play; Functional play; Games with rules; Parallel play; Pretend play; Sensorimotor play; Social

Definition

Play is a difficult phenomenon to define. Many theorists have attempted to develop strict definitions of play, but it is an occurrence which eludes simple definition (Pelligrini 2009). Because play differs so dramatically across individuals, contexts, and cultures, there currently exist lists of characteristics often indicative of play situations, rather than a strict definition itself. For example, play may be distinguished in terms of functional dimensions, in that it often does not have a functional or goal-directed consequence (e.g., play fighting does not hurt others), and it is often more oriented toward means than ends. In addition, play may be distinguished in causal dimensions, in that play will

frequently be interrupted by more pressing, functional concerns, players exhibit relaxed motivation (i.e. play is voluntary), and play is often characteristic of juveniles (i.e. children play more frequently than adults). Because there is not a universally-accepted definition of play, researchers apply different descriptions of play characteristics to identify play in different contexts. Nevertheless, such characteristics converge toward a definition of a complex phenomenon.

Historical Background

Two developmentalists have had extraordinary influence in defining play and its development. Parten (1932) focused on social participation within play, and Piaget (1952) focused on the development of object play as a reflection of developing cognitive processes. Prior to the 1930s, only “scattered individual attempts” (Parten 1932, p. 246) had been made to study children’s social behavior more generally and play more specifically. Mildred Parten, at the University of Minnesota, recognized the need for systematic study of child group behavior. She pioneered the longitudinal observational study of children’s social behavior within the nursery school of the Institute of Child Welfare at the University of Minnesota. Results from this study formed the basis of categorization of types of children’s play.

Parten (1932) observed 42 children across 8 months’ time. Although children’s IQ ranged from 81 to 145, the mean IQ indicated above-average mental ability, and a high percentage of children had fathers with professional careers. Generalization of research results drawn from such studies, as well as considerations of cross-cultural studies of play, is discussed below. After several weeks of careful observation, Parten (1932) developed a scale of social participation within play. The following stages represent increasingly socialized forms of play:

1. Unoccupied behavior: The child does not seem to be playing but instead occupies him/herself with watching anything that happens to be of

momentary interest. When nothing external interests him/her; he/she plays with his/her own body, gets on and off chairs, stands or sits unoccupied, or follows the teacher.

2. Onlooker: The child observes a group of children playing but does not overtly enter into the play activity. The child may, however, talk to the children whom he/she is observing, ask questions, or give suggestions.
3. Solitary independent play: The child plays alone and independently with toys different from those used by nearby children. He/she makes no effort to get close to other children or to reference what others are doing.
4. Parallel activity: The child plays beside, rather than with, other children. The activity that he chooses naturally brings him/her close to other children, but he/she plays independently. Toys used are similar to those used by others around him/her, but he/she plays with them as he/she desires and does not try to influence the activity of the other children.
5. Associative play: The child plays with other children. In this group play, there is an overt recognition by the group members of their common activity, interests, and personal associations. Although all group members engage in similar or identical activity, there is no division of labor and no organization of the activity around any material goal or product.
6. Cooperative or organized supplementary play: The child plays in a group that is organized for the purpose of making some material product, attaining some competitive goal, dramatizing situations of adult or group life, or playing formal games. This represents the most highly organized group activity and is marked by elements of division of labor, group censorship, centralization of control in the hands of one or two members, and the subordination of individual desire to that of the group.

Parten's observational study involved 2–4-year-olds, and she found a clear progression through stages of play as children aged. The younger children either played alone or in parallel groups, while the older children played in the more highly organized groups. Parten's play

categories are still respected and widely used as a meaningful framework within which to examine children's increasing maturity of play.

Piaget provided the first detailed theory of play development from a cognitive perspective. Although many writers prior to Piaget theorized that play was important to children's development, Piaget elaborated upon play's significance in terms of cognitive development and defined three types of play. Like Parten (1932), Piaget (1952) theorized that these categories of play were organized into stages which children move through as they develop. According to Piaget (1952), children move through three primary ordered stages during their first four years of life. His theory, like that of Parten (1932) two decades prior, has remained highly influential. Piaget suggested that children's play moves from motor actions to underlying cognitive representation over time. Piaget (1952) outlined the following three stages:

1. Practice play: Characteristic of children from approximately 2 months to 2 years of age. During this stage, children are not yet motivated or influenced by convention, symbols, pretending, or rules. Instead, they utilize sensorimotor activities to gain pleasure – mastery motivation – based on agency, via moving their bodies or objects and repeating learned actions. Infants in this stage develop the ability to combine different sensorimotor action schemes in their practice play, begin to define objects by their use, and perform ritualistic action patterns in which typical actions with conventional objects are performed. However, the play in this stage is limited in that infants will not apply these actions to new objects or display an awareness of make-believe.
2. Symbolic play: Characteristic of children from approximately 2–4 years of age. During this stage, children make the transition from sensorimotor schemes to mental operations or representations. Piaget (1952) detailed eight ordinal levels of symbolic play, leading from the earliest forms involving the use of familiar actions on novel objects (e.g., putting a box on the foot and saying it is a shoe) to the reproduction of

scenes from reality, corrected to “fix” certain outcomes (e.g., after a recent trip to the doctor, the child might pretend that doctor did not give him/her a shot, but instead gave him/her a lollipop).

3. Games with rules: Characteristic of children from approximately 4–7 years of age. During this stage, children become interested in social games involving rules, structure, and social interaction. Over time, the types of rules, as well as the reasons for following them change. Children at first follow rules centered on the sensorimotor aspects of play which provide structure and repetition. Later, rules become focused on the social aspects of play and are connected to group acceptance. Finally, competitive games and games with codes of rules become more frequently played.

The theories described above can easily be combined to imagine a typical child’s progression through play development both in social interaction and cognition. A 2-year-old child might, for example, engage in parallel play while projecting symbolic schemes onto new objects, while a 4-year-old child might engage in cooperative supplemental play while exploring anticipatory combinations. For all the research on play that has occurred since Parten (1932) and Piaget (1952), their research and theories continue to influence current play research.

Current Knowledge

Play, in its various forms and stages, affects children’s development widely (Bjorklund 1997). Many theorists argue, in fact, that it is primarily through play that children’s cognition develops (Dansky 1980; Piaget 1952). Play can be viewed from an evolutionary perspective as a mechanism by which children master skills that will be important for their survival and development to maturity (Bruner 1972). According to Bjorklund (1997), the play exhibited by children has an adaptive role in both evolution and human development. He argues that the play exhibited by children in humans’ protracted period of immaturity is a

necessary component of development and leads to successful learning by children of the complexity and diversity of contemporary human community. Play is a forum through which children develop skills and knowledge related to motor skills, culturally acceptable social behaviors (Bruner 1972), spatial and classification skills (Connolly and Doyle 1984), language fluency, abstract thought and intelligence (Weininger 1978), and innovative problem-solving (Smith and Dutton 1979).

Many categories of play cited today correspond to the stages of play development outlined by Parten (1932) and Piaget (1952). Categories of play include:

- Sensorimotor or exploratory play: This is also known as practice play and corresponds to the first stage of Piaget’s (1952) theory outlining development of play. In sensorimotor play, children manipulate objects as a means for practice and mastery of action schemas. Sensorimotor play involves repetition of behaviors, routines, and subroutines that have been mastered. Examples include infants’ rhythmic motor behaviors, such as repetitive kicking or shaking of an object (Pelligrini 2009; Rogers et al. 2005).
- Functional play: Functional play is categorized in one of two ways. According to some researchers, functional play is a synonym for the sensorimotor or practice play described above (Pelligrini 2009). For others, it is a separate category of play involving the child combining objects and forming play acts in ways that reflect social conventions. In this category of play, children use objects in the ways that they are typically used. Examples include a child eating and drinking from play dishes and placing a toy cup on a saucer (Rogers et al. 2005).
- Pretend play: Also called symbolic play, fantasy play, or make-believe play, this corresponds to the second stage of Piaget’s (1952) theory outlining the development of play. Pretend play is a primarily assimilative behavior where in the child takes a behavior out of its functional context or recombines everyday behavioral sequences in novel orders

(Pelligrini 2009). In pretend play, absent elements are often represented through objects, gestures, and language. It grows out of the child's developing ability for mental representation and provides a means for practicing and gaining understanding of the events of the social world (Rogers et al. 2005). Examples include a child utilizing a stick in place of a spoon to stir pretend liquid in a cup or pretending that one is a train conductor.

- **Games with rules:** This type of play corresponds to the third stage of Piaget's (1952) theory outlining the development of play. In this type of play, games are primarily accommodative and governed by a priori rules. Although there is some negotiation in the rules of children's games, these rules are generally not flexible (Pelligrini 2009). Examples include baseball and board games.
- **Parallel play:** This type of play corresponds to the fourth stage of Parten's (1932) theory outlining the development of play. In this type of play, the child plays beside, rather than with, other children. Although the child uses similar toys as those around him, he/she plays with them independently and as he/she desires, and he/she makes no effort to influence the play of others. Examples include playing with dump trucks in a sandbox or playing with toy boats in a water table, next to other children playing similar games.
- **Social play:** This type of play corresponds to the fifth and sixth stages of Parten's (1932) theory outlining the development of play (associative play and cooperative play). Here, the child plays with other children. As evidenced by the multiple corresponding stages, there is a continuum of social play. As in cooperative play, the group of playing children may organize itself in order to make some material product, attain some competitive goal, dramatize situations of adult or group life, or play formal games. As in associative play, the children may merely play with one another, with an overt recognition by the members of their common activity, interests, and personal associations but without division of labor or organization.

- **Exploratory play:** It has been argued that exploration is a separate activity than play. Some researchers argue that exploratory behaviors occur prior to play behaviors in the first few months of life. They note that exploration is performed in a deliberate manner, often in novel contexts, in order to gather information, while play is exhibited in familiar contexts with positive affect in order to practice (Pelligrini 2009). Other researchers, however, view exploration as a type of play and hypothesize that children learn cause and effect relationships through such play (Schultz and Bonawitz 2007). Piaget himself argued that children construct knowledge via active exploration (Piaget 1930), and exploratory play is believed to accomplish such knowledge attainment. Examples of exploratory play include mouthing objects and dropping objects to observe what happens.

Cultural Influences on Play

Roopnarine, Johnson, and Hooper (1994) describe cultural-ecological models of behavior and stress the importance of applying such models to research of play. According to a cultural-ecological model, there are three interacting layers of environmental influence on play: (1) physical and social aspects of children's immediate settings; (2) historical influences that affect the way adults and children conceptualize play; and (3) cultural and ideological beliefs regarding the meaning of play. A wide range of variables contribute to the development, significance, and value of play and are highly influenced by ecological context at many levels. Play is an expression of a particular culture and an important context for transmission of that culture (Schwartzman 1978). Although some research has been conducted on the cross-cultural development of play, the majority of taxonomies of play forms, as well as discussions of the significance and value of play, have been based on studies of Western children and have made global assumptions. In order to achieve a more comprehensive, coherent, and integrated knowledge base of play, it is necessary to expand these cross-cultural considerations in future play research (Roopnarine et al. 1994).

Play in Autism

Many studies have repeatedly isolated symbolic play deficits as a core feature of autism. In Ricks and Wing 1975, for example, Ricks and Wing reviewed what was known about autism up to that point and concluded that a central deficit within children diagnosed with autism is the inability to understand symbols. This inability affected, among other areas, the symbolic play skills of children with autism. Expanding on that paper, Wing, Gould, Yeates, and Brierley (1977) published the first major research paper on symbolic play in autism. They confirmed that in children with autism, there is a lack of spontaneous symbolic play, and that for those who did demonstrate some amount of symbolic play, the play acts were repetitive and stereotypic. Children with autism do not perform symbolic play acts as frequently as other children, and when they do perform these play acts, the play lacks variety.

Additional research has replicated these original findings and strengthened the viewpoint that children with autism engage in fewer and poorer quality symbolic play acts (Hammes and Langdell 1981; Riguette et al. 1981). Sigman and colleagues (Mundy et al. 1986; Sigman and Ungerer 1984) replicated these findings but also found that non-symbolic play was affected in children with autism.

Although Sigman and colleagues (Mundy et al. 1986; Sigman and Ungerer 1984) found that children with autism exhibit a paucity of functional and sensorimotor play skills in addition to exhibiting a lack of symbolic play skills, evidence on this has been mixed. While Baron-Cohen (1987) and Sigman and Ruskin (1999) found no deficits in functional play skills in children with autism, the majority of studies have found differences in functional and sensorimotor play skills in children with autism as compared to matched comparison groups. For example, Sigman and Ungerer (1984) reported that in spontaneous play conditions, young children with autism produced fewer functional play acts, fewer different functional play acts, and fewer sequences than intellectually disabled children in their comparison group. In addition, Williams, Reddy, and Costall (2001) found deficits in both functional

and sensorimotor play in children with autism. These children exhibited lower rates of both types of play acts and performed more immature than mature play acts. The research across the symbolic, functional, and sensorimotor play skills in children with autism demonstrates similar patterns of findings: children with autism exhibit fewer play acts, more repetition, less novelty, and less diversity of play schemas.

Interventions have been demonstrated to improve the play skills of children with autism. Direct teaching and naturalistic teaching approaches have demonstrated significant effects on children's play (Kasari et al. 2006, 2008; Kasari et al. 2008; Reddy et al. 2005; Rogers 2005), addressing both functional and symbolic play deficits. Targeting play for interventions serves to increase children's play as a learning tool and also provides older children with appropriate recreational activities.

Future Directions

Future needs related to autism play research fall under two primary areas: brain behavior correlates and effects of play skills on later development. Neuroscience has not yet been incorporated into models or studies of play (Rogers et al. 2005). Play is a cognitive activity as well as a motor and social-emotional activity. Observers can distinguish between adaptive acts and play acts in children's behavior, with the assumption that some differing neural processes are involved, even when the overt act is the same. It will be helpful for researchers to begin to consider the neural systems underlying play and the neuropsychology of play. Play is considered an important therapeutic medium for a number of childhood disorders (Reddy et al. 2005). Most childhood interventions for autism spectrum disorder target play skills, without much knowledge about the relationships between childhood play skills and their effects on later development. Longitudinal research in this area is sorely needed. Expanding the multilevel accounts of play should assist future researchers to understand the change process facilitated by play, both developmentally and therapeutically.

See Also

► Symbolic Play

References and Reading

- Baron-Cohen, S. (1987). Autism and symbolic play. *British Journal of Developmental Psychology*, 5, 139–148.
- Bjorklund, D. F. (1997). The role of immaturity in human development. *Psychological Bulletin*, 122(2), 153–169.
- Bruner, J. S. (1972). Nature and uses of immaturity. *American Psychologist*, 27, 687–708.
- Connolly, J. A., & Doyle, A. (1984). Relation of social fantasy play to social competence in preschoolers. *Developmental Psychology*, 20(5), 797–806.
- Dansky, J. L. (1980). Make-believe: A mediator of the relationship between play and associative fluency. *Child Development*, 51, 576–579.
- Hammes, J. G. W., & Langdell, T. (1981). Precursors of symbol formation and childhood autism. *Journal of Autism and Developmental Disorders*, 11, 331–346.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in young children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry*, 47(6), 611–620.
- Kasari, C., Paparella, T., Freeman, S., & Jahromi, L. B. (2008). Language outcome in autism: Randomized comparison of joint attention and play interventions. *Journal of Consulting and Clinical Psychology*, 76(1), 125–137.
- Mundy, P., Sigman, M., Ungerer, J., & Sherman, T. (1986). Defining the social deficits of autism: The contribution of non-verbal communication measures. *Journal of Child Psychology and Psychiatry*, 27, 657–669.
- Parten, M. B. (1932). Social participation among pre-school children. *Journal of Abnormal and Social Psychology*, 27(3), 243–269.
- Pelligrini, A. D. (2009). *The role of play in human development*. New York: Oxford University Press.
- Piaget, J. (1930). *The child's conception of physical causality*. New York: Harcourt, Brace.
- Piaget, J. (1952). *Play, dreams, and imitation in childhood*. New York: W.W. Norton.
- Reddy, L. A., Files-Hall, T., & Schaefer, C. E. (2005). *Empirically based play interventions for children*. Washington, DC: APA Press.
- Ricks, D. M., & Wing, L. (1975). Language, communication, and the use of symbols in normal and autistic children. *Journal of Autism and Childhood Schizophrenia*, 5, 191–221.
- Riguet, C. B., Taylor, N. D., Benaroya, S., & Klein, L. S. (1981). Symbolic play in autistic, Down's, and normal children of equivalent mental age. *Journal of Autism and Developmental Disorders*, 11, 439–448.
- Rogers, S. J. (2005). Play interventions for young children with autism spectrum disorders, ch. 11. In L. A. Reddy, T. Files-Hall, & C. E. Schaefer (Eds.), *Empirically based play interventions for children* (pp. 215–240). Washington, DC: APA Press.
- Rogers, S. J., Cook, I., & Meryl, A. (2005). Imitation and play in autism. In F. Volkmar, A. Klin, R. Paul, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Assessment, interventions, and policy, Vol. 2, pp. 882–896). New York: Wiley.
- Roopnarine, J. L., Johnson, J. E., & Hooper, F. H. (1994). *Children's play in diverse cultures*. Albany: State University of New York.
- Schultz, L. E., & Bonawitz, E. B. (2007). Serious fun: Preschoolers engage in more exploratory play when evidence is confounded. *Developmental Psychology*, 43(4), 1045–1050.
- Schwartzman, H. (1978). *Transformations: The anthropology of children's play*. New York: Plenum.
- Sigman, M., & Ruskin, E. (1999). Continuity and change in the social competence of children with autism, Down syndrome, and developmental delays. *Monographs of the Society for Research in Child Development*, 64, 1–114.
- Sigman, M., & Ungerer, J. (1984). Cognitive and language skills in autistic, mentally retarded, and normal children. *Developmental Psychology*, 20, 293–302.
- Smith, P. K., & Dutton, S. (1979). Play and training in direct and innovative problem solving. *Child Development*, 53(3), 830–836.
- Weininger, O. (1978). Play and education of the young child. *Education*, 99(2), 127–135.
- Williams, E., Reddy, V., & Costall, A. (2001). Taking a closer look at functional play in children with autism. *Journal of Autism and Developmental Disorders*, 31, 67–77.
- Wing, L., Gould, J., Yeates, S., & Brierley, L. (1977). Symbolic play in severely mentally retarded and in autistic children. *Journal of Child Psychology and Psychiatry*, 18, 167–178.

Play Intervention

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Definition

Play intervention is a term used to describe psychosocial interventions designed to improve play skills in children with ASD. The term has also been used to describe interventions that take place within a play setting, but focus instead on increasing social or language skills. This entry will focus on intervention developed specifically to target play skills in children with ASD.

Play is a broad term that describes a variety of skills, including object-oriented play, pretense, and play with peers. Children with ASD have significant deficits in all of these areas, and a lack of varied, spontaneous make-believe play or social imitative play is a hallmark feature of the disorder (American Psychiatric Association [APA] 2000). Thus, a variety of intervention strategies have been developed to target play skills in children with ASD, including discrete trial training (DTT), naturalistic behavioral interventions, integrated play groups, video modeling, differential reinforcement of appropriate behavior (DRA), self-management, and play scripts (see Stahmer et al. 2003 for review).

The term “play intervention” is most commonly used to describe a group of naturalistic intervention techniques that take place in a play setting, and may or may not involve typically developing peers. As such, this entry is focused on play intervention of this type. Within a play setting, the intervention provider uses a number of techniques to engage the child with the materials and with the provider (e.g., following along with the child’s focus of interest, acting excited and animated about the child’s play). With the child engaged in the interaction, the intervention provider then provides models and prompts (either verbal or physical) to encourage the child to complete more complex or advanced play actions than he is currently using. When the child responds by completing the prompted play actions, the intervention provider typically praises the child verbally and rewards the child by allowing him continued access to the toys.

Historical Background

Limited and perseverative play has been described as a difficulty for children with autism since Kanner’s first description of the syndrome in 1943. More systematic research as early as 1964 indicates that, compared to typically developing or intellectually disabled children, children with autism show more stereotyped play, less diversity of play, and less symbolic play (Wulff 1985). When autism first appeared in the third edition

of the Diagnostic and Statistical Manual of Mental Disorders in 1980, difficulty with imaginative play and social play were included as symptoms of the disorder. The first treatment to specifically target play skills in children with autism and other developmental delays was discrete trial training. However, given concerns about generalization of skills and appropriateness of such structured programs for young children, most play interventions now take place in a more naturalistic setting. Pivotal response training (PRT), a naturalistic behavioral intervention originally designed to teach language to children with autism, was the first intervention of its type to be successfully adapted to teach symbolic play skills to children with autism (Stahmer 1995). Since this time, a number of other naturalistic behavioral interventions have been used to teach play as well.

Rationale or Underlying Theory

The play deficits (particularly regarding symbolic play) seen in children with autism have been theoretically linked to difficulties with symbol formation more generally. Other skills that rely heavily on symbol formation are language and abstract reasoning (Wulff 1985). In addition, play is seen as a fundamental way in which young children connect socially, both with adults and with other children (e.g., Mueller and Brenner 1977). Therefore, interventions that improve symbolic play skills in children with autism may also have collateral effects on language, cognitive skills, and social skills. Early toy play skills are associated with later communication development (Toth et al. 2006). In addition, children receiving play intervention have shown increases in other skills not specifically targeted in these interventions, such as language, joint attention, and interaction skills (Stahmer et al. 2003).

Goals and Objectives

Play intervention is designed to target a variety of object-interaction and social play skills. For children who interact little with objects or engage

primarily in stereotyped play, play intervention targets are likely to be simple meaningful toy interactions, such as combining toys in a meaningful manner (e.g., stacking blocks). This type of simple relational play is seen as a necessary precursor to more complex symbolic or pretend play (van Berckelaer-Onnes 2003), and increases in appropriate play are associated with decreases in less-meaningful stereotyped play (Stahmer and Schreibman 1992). For children with more advanced play skills or who are further into a play intervention treatment program, more complex actions and play schemes such as doll play, multiple-step pretend schemes, and sociodramatic play with a partner are likely to be targeted.

Treatment Participants

Play intervention has primarily been targeted at relatively young children (approximately 2–6 years of age). Some research has indicated that play intervention is likely to be more successful for children with higher IQ's (Wulff 1985) and higher initial object-interaction skills (Ingersoll 2010).

Treatment Procedures

Play intervention is typically carried out in a play-based setting, with a number of toys available to the child and therapist. Structure of the sessions may vary depending on the specific approach used, but most play interventions include at least a portion of the session that provides opportunities for teaching within a semi-structured play setting. More structured approaches, such as pivotal response training, may include table-top drills to teach new skills during a portion of the session. Less structured approaches, such as incidental teaching, are more child-directed and rely heavily on teaching around the child's chosen focus of interest. Sessions are usually conducted two to seven times per week for 30–60 min. Major tenets of play intervention include the use of techniques to engage the child in interaction (e.g., following the child's lead, talking about what the child is

doing, sitting close to the child) and the use of modeling, prompting, and reinforcement strategies (e.g., telling or showing the child what to do, giving verbal praise for correct responses, providing corrective feedback, rewarding the child's correct responses with continued access to toys). Play intervention has been successful in a number of treatment formats, including therapist-, parent-, peer-, and sibling-implemented models (Stahmer et al. 2003).

Efficacy Information

Play intervention has been successful at teaching functional and symbolic play skills for children with autism (Kasari et al. 2006). In many cases, collateral benefits on other skills, such as language and social skills, have also been observed (Kasari et al. 2008). These skill improvements have been found to persist up to 1 year later. Increases in both spontaneous and prompted play skills have been observed in nontreatment settings (e.g., standardized assessments, parent-child play sessions) following play intervention. However, generalization of these skills to settings with typically developing peers has not been evident, suggesting that peer play may need to be targeted explicitly. In addition, the play of children with autism often remains qualitatively different from that of typically developing children even following play intervention (Stahmer et al. 2006).

Outcome Measurement

Researchers have measured play in a variety of ways, including total time spent playing with toys, time spent in appropriate play, diversity of object play, and play complexity or level. All of these elements of play have been found to significantly discriminate children with autism from those with typical development, intellectual disability, hearing impairment, and language impairment (Stone et al. 1990).

Several structured assessments have been used to assess treatment outcome and relationships between play skills and other skills in children

with autism. The Structured Play Assessment (SPA; Ungerer and Sigman 1981) provides children with four different sets of toys and a variety of structured play prompts designed to elicit play skills at a number of different play levels. Both prompted and spontaneous play acts are coded for play level: manipulative, relational, functional, or symbolic. Manipulative play consists of actions such as mouthing, banging, fingering, or throwing toys. Relational play consists of combining objects in nonfunctional ways (e.g., nesting or stacking objects). Functional play consists of using objects in a way they were intended, such as brushing a doll's hair, placing a truck in a garage, or holding a phone to one's ear. Symbolic play consists of object substitution (pretending one object is another), use of a doll as an independent agent, and imaginary play (creation of an object or effect that is not actually present). The SPA has been used by a number of researchers to measure treatment outcome and relationship among play skills and other social-communication skills in children with autism (Kasari et al. 2008; Sigman and Ungerer 1984; Ungerer and Sigman 1981).

The Developmental Play Assessment (DPA; Lifter et al. 1988) consists of a 30-min videotaped free play session with a child and an administrator. The administrator comments on the child's play, but does not provide prompts or instructions for play. Each play act on the videotape is later coded to determine what play category the act fits in to. Each play act is then placed into one of eight play categories (e.g., indiscriminate actions, child as agent, doll as agent, sociodramatic play) and the total number and diversity of acts in each category are tallied to determine whether the child has absent skills, emerging skills, or mastery in each category. Play categories were designated based upon numerous studies of the development of play skills in children with typical development and with disabilities. The DPA has been used as a measure of play skills in children with and without autism in number of research studies (Pierce-Jordan and Lifter 2005; Lifter et al. 1993; Yoder and Stone 2006).

Qualifications of Treatment Providers

Play intervention is provided by a wide range of treatment providers, including teachers, speech pathologists, psychologists, social workers, and physicians. In addition, family members and peers can be trained to serve as treatment providers or treatment aids in play intervention. A number of play intervention programs are accessible through training workshops or published materials.

See Also

- ▶ [Naturalistic Interventions](#)
- ▶ [Pivotal response Training](#)
- ▶ [Play](#)
- ▶ [Self-Management Interventions](#)
- ▶ [Symbolic Play](#)
- ▶ [Video Modeling/Video Self-Modeling](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Rev). Washington, DC: Author.
- Ingersoll, B. (2010). Brief report: Pilot randomized control trial of reciprocal imitation training for teaching elicited and spontaneous imitation to children with autism. *Journal of Autism and Developmental Disorders*, 40, 1154–1160.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in young children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry*, 47, 611–620.
- Kasari, C., Paparella, T., Freeman, S., & Jahromi, L. (2008). Language outcome in autism: Randomized comparison of joint attention and play interventions. *Journal of Consulting and Clinical Psychology*, 76, 125–137.
- Lifter, K., Edwards, G., Avery, D., Anderson, S. R., & Sulzer-Azaroff, B. (1988, November). The Developmental Play Assessment (DPA) instrument (Revised 9/94). Presented originally as: Developmental assessment of children's play: Implications for intervention. Mini-seminar presented at the Annual Convention of

the American Speech-Language-Hearing Association, Boston.

- Lifter, K., Sulzer-Azaroff, B., Anderson, S. R., & Cowdery, G. E. (1993). Teaching play activities to preschoolers with developmental disabilities: The importance of developmental considerations. *Journal of Early Intervention, 17*, 139–159.
- Mueller, E., & Brenner, J. (1977). The origins of social skills and interactions among playgroup toddlers. *Child Development, 48*, 854–861.
- Pierce-Jordan, S., & Lifter, K. (2005). The interaction of social and play behaviors in preschoolers with and without pervasive developmental disorders. *Topics in Early Childhood Special Education, 21*, 34–47.
- Sigman, M., & Ungerer, J. (1984). Cognitive and language skills in autistic, mentally retarded, and normal children. *Developmental Psychology, 20*, 293–302.
- Stahmer, A. (1995). Teaching symbolic play skills to children with autism using pivotal response training. *Journal of Autism and Developmental Disorders, 25*, 123–141.
- Stahmer, A., & Schreibman, S. (1992). Teaching children with autism appropriate play in unsupervised environments using a self-management treatment package. *Journal of Applied Behavior Analysis, 25*, 447–459.
- Stahmer, A., Ingersoll, B., & Carter, C. (2003). Behavioral approaches to promoting play. *Autism, 7*, 401–413.
- Stahmer, A., Schreibman, L., & Powell, N. (2006). Social validation of symbolic play training for children with autism. *Journal of Early and Intensive Behavior Intervention, 3*, 196–210.
- Stone, W., Lemaneck, K., Fishel, P., Fernandez, M., & Altemeier, W. (1990). Play and imitation skills in the diagnosis of autism in young children. *Pediatrics, 86*, 267–272.
- Toth, K., Munson, J., Meltzoff, A., & Dawson, G. (2006). Early predictors of communication development in young children with autism spectrum disorders: Joint attention, imitation, and toy play. *Journal of Autism and Developmental Disorders, 36*, 993–1005.
- Ungerer, J., & Sigman, M. (1981). Symbolic play and language comprehension in autistic children. *Journal of the American Academy of Child Psychiatry, 20*, 318–337.
- van Berckelaer-Onnes, I. A. (2003). Promoting early play. *Autism, 7*, 415–423.
- Wulff, S. B. (1985). The symbolic and object play of children with autism: A review. *Journal of Autism and Developmental Disorders, 15*, 139–148.
- Yoder, P., & Stone, W. (2006). A randomized comparison of the effect of two prelinguistic communication interventions on the acquisition of spoken communication in preschoolers with ASD. *Journal of Speech, Language, and Hearing Research, 49*, 698–711.

PLAY Project

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Definition

The PLAY Project (PLAY) is a parent-implemented, intensive (10–15 h/week) early intervention program for young children (18 m–6 y) with autism spectrum disorders (ASD) that uses a developmental, relationship-based (DRB) framework. The goal of the PLAY Project Organization is to help as many families as possible engage in a joyful and effective way with their child to help their child reach key functional developmental milestones. To achieve this goal, a national network of certified PLAY Consultants provide monthly (2–3 h) or weekly (1 h) visits, coach and partner with families to build trust, promote family confidence and competence, and strengthen family-child relationships through playful engagement. PLAY uses videotape and written feedback to help families learn the manualized principles, methods, techniques, and activities of the model. Rigorous NIMH research on PLAY (Solomon et al. 2014) has shown that families gain the knowledge and skills needed to enrich their children's interactional and developmental abilities. As a result, children in the research study made social, emotional, and developmental progress. PLAY is one of the few evidence-based parent-implemented models to show a reduction in the child's autism symptomatology.

Historical Background

From 2000 to 2005, in response to the lack of autism early intervention services in Michigan, Richard Solomon MD, then section head of developmental and behavioral pediatrics at the

University of Michigan, introduced the PLAY Project's (PLAY) developmental, relationship-based model (see Rationale/Underlying Theory below) to the community. A pre-post pilot study of the first 75 families enrolled in PLAY was published in the journal *Autism* in 2007 and indicated that parents could be coached to interact successfully with their young child with autism in a way that promoted the child's social-emotional functioning (Solomon et al. 2007). Subsequently, from 2009 to 2012, the PLAY Project was funded by the National Institutes of Mental Health's (NIMH) Small Business Innovations Research (SBIR) grant to implement a large ($n = 128$) randomized controlled trial comparing the PLAY Project plus Community Standard Services (primarily preschool) to Community Standard Services alone. The research results, published in 2014 in the *Journal of Developmental and Behavioral Pediatrics*, confirmed and extended the findings of the pilot study (Solomon et al. 2014). PLAY parents were successfully coached by PLAY Consultants to be more sensitive to, responsive to, and effective in interacting with their child with autism than control parents. Compared to control children, PLAY children improved in their interaction, compliance, initiation, and functional emotional development. PLAY children showed less autism symptomatology on the ADOS (Autism Diagnostic Observation Scale) than control children. PLAY parents were *not* more stressed for being asked to put in the time of PLAY (1–2 h/day) and, as a group, become less depressed over the year of intervention. After establishing its clinical and research foundations, PLAY has been broadly disseminated in dozens of states. In 2011, PLAY was implemented statewide by Ohio's Department of Developmental Disabilities in their early intervention (birth to 3) program for children with or at risk for ASD and now provides PLAY to over 70% of Ohio counties at no cost to families.

Rationale or Underlying Theory

The PLAY Project uses a parent-implemented model (PIM) to provide a developmental,

relationship-based (DRB) intervention for young children with autism spectrum disorders (ASD).

The theoretical foundations of DRB intervention date back to the 1950s and derive from the fields of psychology, medicine, and education. Bowlby, Bruner, A. Freud, Piaget, Ainsworth, Mahler, Vygotsky, and others established the theoretical and clinical basis for a developmental, transactional approach to intervention that relies on responsive caregivers and affective interaction. A foundational premise of DRB interventions is that there is an inherent biological drive toward increasing complexity of interaction, communication, and social repertoires in children, including those with autism. Greenspan integrated a Piagetian cognitive developmental framework with the psychoanalytic perspective and created an overarching developmental model called DIR (developmental, individual-difference, relationship-based) (Greenspan 1992; Greenspan and Wieder 2005).

Greenspan and Wieder then applied the DIR model clinically to children with ASD, emphasizing that the needs of children with ASD must be addressed from a holistic rather than piecemeal approach, with a view toward internal psychological affective development. They observed that many children with ASD have the capacity for sense of self and advanced levels of psychological and cognitive growth. Since then, several well-done studies have confirmed the effectiveness of using the DRB framework for parent-implemented interventions (Binns and Oram-Cardy 2019; Sandbank et al. 2019; Smith and Iadarola 2015; Mercer 2015; Wong and Odom 2014; Siller et al. 2013, 2014; Green et al. 2010).

Most empirical studies on DRB interventions use parent-implemented models (PIM) with *methods* that are child directed and *strategies* that utilize natural play rather than structured teaching or operant conditioning. The emphasis in intervention is on the role of the child as an active learner in the context of dyadic relationships. In PIM, parents are coached to sensitively read the child's cues, respond to the child's intention, challenge the child to move forward developmentally, and playfully join with the child through co-regulated interactions. Over the

past 30 years, and especially in the last 10 years, rigorous research has been accumulating about the effectiveness of DRB interventions (aka also as developmental social-pragmatic (DSP)) and PIM by documenting measurable improvements in joint attention, social engagement, initiative, reciprocity, functional development, and symbolic thinking (Hyman et al. 2020; Binns and Oram-Cardy 2019; Smith and Iadarola 2015; Mercer 2015; Wong and Odom 2014; Siller et al. 2013, 2014; Green et al. 2010).

Goals and Objectives

The PLAY Project (PLAY) trains pediatric professionals and child development experts to help parents engage their child with ASD to help the child make functional social developmental gains. Parent-implemented models, such as PLAY, can reach thousands of families many of whom are not receiving necessary services (Mandell et al. 2016). Thus, PLAY has three overarching goals:

1. The training of PLAY Consultants to a high degree of fidelity.
 2. Through PLAY Consultation, effectively coach and support parents to help their child to address the core deficits of ASD.
 3. Develop a model that can be replicated with high fidelity and disseminated on a large scale to address the large unmet need of families in the USA who have a young child with ASD.
1. *The training of PLAY Consultants to a high degree of fidelity.*

Qualified pediatric professionals and child development experts (SLP, OTs, MEd, PhD, and MD) are trained by following a three-step approach:

- (a) Attending a 2-day PLAY workshop to learn the basics of the PLAY Project model
- (b) Completing a 6-week online course to learn how to complete video reviews and write-ups
- (c) Undergoing 12–18 months of supervision to assure clinical fidelity to the model

After taking the online course, *PLAY Consultants-in-Training* begin serving families while under supervision. Supervision involves submitting 15 online videos and write-ups based on their clinical caseload to PLAY Project supervisors. The supervisors then evaluate consultant write-ups according to the PLAY Project Fidelity Manual. As part of the NIMH-SBIR research study (Solomon et al. 2014), fidelity of the study's PLAY Consultants was assessed. Twenty percent of all PC videos and write-ups were reviewed by two supervisors who assessed each consultant using the PLAY Project's Consultant Training Fidelity Manual. Results showed that each of the consultants had high fidelity to the model.

2. *Through PLAY Consultation, effectively coach and support parents to help their child to address the core deficits of ASD.*

The Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5), is widely considered to be the universal authority for psychiatric diagnosis (APA 2013). At the core of the DSM 5's revised criteria for autism spectrum disorders (ASD) are "challenges in social communication and social interaction" including lack of "social-emotional reciprocity," "back and forth conversation," problems with "nonverbal/gestural communication," and difficulties with "imaginative play." Developmental, relationship-based interventions that use parent-implemented models are directed primarily at these core deficits described in the DSM 5, making them especially appropriate for young children with ASD. The NIMH-SBIR research confirmed that parents trained by PLAY Consultants improved in their ability to effectively engage their child with ASD and PLAY children improved in their functional development and social interactions.

In terms of supporting parents, mothers of children with ASD are reported to experience higher levels of depression than either mothers of typically developing children or mothers of children with other disabilities (e.g., Abbeduto et al. 2004; Singer 2006). A longitudinal study of 143 mothers of toddlers with confirmed

diagnoses of autism (Carter et al. 2009) reported that 35% of these mothers displayed clinical levels of depression symptoms when their children were less than 3 years of age, and 42% displayed clinical depression symptoms 2 years later. Parent-implemented models that offer social support to families, build family competence, and help family members engage their often hard-to-engage children make PIM especially appropriate for this population of young children with ASD. One of the unexpected outcomes of the NIMH-SBIR research on PLAY showed that parental depression of those who received the PLAY Project intervention declined significantly over the year of the study.

3. *Develop a public service model that can be replicated with high fidelity and disseminated on a large scale to address the large unmet need of families in the USA who have a young child with ASD.*

With the marked increase in the prevalence of autism (1 in 59) and the need to provide intensive early intervention for this growing number of children, too many children in the USA are not getting the services they need (Mandell et al. 2016). As part of its vision, the PLAY Project Organization (PPO) is dedicated to reaching as many families as possible. Parent-implemented models are much less costly than therapist delivered services and can be disseminated effectively on a large scale.

In 2011, the PLAY Project Organization was invited by the state of Ohio's Department of Developmental Disabilities to train early intervention workers across the state. Over the next 3 years, nearly 150 early intervention (birth to 3) providers were trained to certification. Currently, over 70% of the counties in Ohio offer PLAY. Families whose children have been diagnosed with ASD or who have "red flags" for ASD are offered PLAY services at no cost. A full evaluation of the implementation of PLAY services in Ohio was conducted independently by Marilyn Espe-Sherwindt PhD and her study group at Akron Children's Hospital ([https://www.](https://www.playproject.org/assets/PLAY-Evaluation-Final-Report.pdf)

[playproject.org/assets/PLAY-Evaluation-Final-Report.pdf](https://www.playproject.org/assets/PLAY-Evaluation-Final-Report.pdf)).

Treatment Procedures

Since 2001, the PLAY Project's (PLAY) training and certification program has trained hundreds of masters-/doctoral-level consultants (SLP, OTs, MEd, PhD, and MD), reaching thousands of children nationally with dissemination in dozens of states and several countries. PLAY typically supplements existing services (e.g., special education, language and occupational therapies, and/or applied behavioral analysis/behavioral interventions) but has been implemented as a primary intervention for ASD in early intervention settings.

PLAY services – provided by certified PLAY Consultants – can be delivered in the home or in the clinic setting. Home visits range from a single 2–3-h home visit monthly to hourly weekly visits (e.g., in early intervention (birth to 3) settings). PLAY services last from 1 to 3 years depending on the needs of the child and family. One week before the first visit, parents are encouraged to review PLAY's "Welcome to the PLAY Project" online course, PLAY Parent's Manual, and book recommendations. The online course introduces parents to PLAY principles, methods, activities, and techniques through e-learning methods combined with video examples. The PLAY Parent's Manual describes in detail the six functional developmental levels (FDLs) of Greenspan and Wieder's developmental, individual-difference, and relationship-based (DIR) theoretical framework and provides descriptions of dozens of PLAY techniques and activities based on the FDLs.

During the visits, the primary caregiver is targeted for instruction, but all caregivers are welcomed to attend PLAY coaching sessions. PLAY Consultants (PC) train parents through coaching, modeling, and video feedback. During *coaching*, PCs helped parents identify their child's subtle and hard-to-detect cues, respond contingently to the child's intentions, and effectively engage the child in reciprocal exchanges. Parents

are taught to provide appropriate developmental challenges to promote progress in the child's FDLs. During *modeling*, PCs play for 15–30 min with the child to demonstrate PLAY methods and techniques. During *video feedback*, the PC obtains a 10-min representative sample of parent play, and the parent obtains a 5-min representative sample of the consultant modeling. A written analysis of the video, sent between visits, reviews the parent-child and consultant-child video interactions, summarizes the child's developmental profile, and recommends methods and techniques. The program is revised to address the child's evolving developmental profile. For example, as children became easier to engage, the intervention emphasizes increasing the length of engagement, waiting for more initiation, and expecting more reciprocal social exchanges, thus increasing the complexity of interaction and helping the child move up FDLs. Typically PLAY Consultants are available between visits as needed by e-mail or phone. Families were encouraged to engage their child in 15- to 20-min play sessions and throughout daily for a total of 2 h/day of active engagement.

Parent support is a key element of PLAY services. PLAY Consultants are masters-level trained child development experts and are additionally trained through the certification process to be aware of the needs of families for referrals to social and/or mental health services. PCs partner with families to build trust, promote family confidence and competence, and strengthen family-child relationships through playful engagement.

Efficacy Information

Studies on the PLAY Project (PLAY) join a growing body of research literature on parent-implemented models that use a developmental, relationship-based framework (Binns and Oram-Cardy 2019). This literature has consistently found that parents are able to learn the methods of play-based interaction and, importantly, implement them in the natural environment of the home. Even in a single parent or two-working-

parent home, the parents spend over 40 h of waking time with their child who has ASD. Parents in PLAY's NIMH-SBIR study reported putting in an average of 11 h per week in active play sessions, without evidence of increased stress, and PLAY parents reported a reduction in depression over the 1 year of the intervention (Solomon et al. 2014). Family support was key factor contributing to the benefits of PLAY. When consultants coach well and parents learn well, then children with ASD do well. Treatment benefits for children in the PLAY studies showed clinically significant gains in engagement, purposeful activity, contingent reciprocal exchanges, and initiation. Functional emotional development improved. Gains in development and language for PLAY children were significant pre- to post-intervention but did not differ from the control group children. Autism symptoms as measured by the Autism Diagnostic Observation Scale (ADOS) markedly improved in the PLAY group versus controls. The NIMH-SBIR grant was implemented in five US cities, large (Detroit, MI) and small (Billings, MT), with a broad range of families in terms of SES, race, and family structure suggesting that PLAY was effective in diverse settings. Finally, for the last 9 years, PLAY has been disseminated statewide in early intervention (birth to 3) system with plans to implement statewide in Michigan and Illinois, suggesting that PLAY can be implemented effectively on a large scale.

Outcome Measurement

To measure the most important outcomes of parent-implemented models that use a developmental, relationship-based intervention (DRB) intervention, research questions must focus on the *interactional process*. Do parents interact with their child who has ASD better after learning DRB methods? Do the children subsequently interact better with their parents? Do the children show improvement in their functional development as a result? Only video analysis can validly provide these answers. We found that the Pivotal Behavior Rating Scale (PBRs) (Mahoney and Kim 2007) and

the Maternal Behavior Rating Scale (MBRS) (Mahoney et al. 1986) had valid and reliable psychometric properties. The Functional Emotional Assessment Scale (FEAS) (Greenspan et al. 2001) measured outcomes relevant to the PLAY Project's autism early intervention program. The Mullen Scales of Early Learning is a very good measure of overall development. For language, we used the MacArthur Communicative Developmental Inventory but its two-scale structure did not fit well with our ASD population. For models that make demands on families, stress measures like the Parenting Stress Index and a depression scale are indicated. We found the ADOS (Autism Diagnostic Observation Scale) and ADI-R measures to be very important in assessing autism symptomatology.

Qualifications of Treatment Providers

PLAY Consultants (PCs) must go through a rigorous training to become certified. Most PCs are masters level or better, licensed pediatric professionals (SLP, OT, MSW, psychologist, MD) or child development experts (EI providers, MEd) who are trained by following a three-step approach:

- (a) Attending a 2-day PLAY workshop to learn the basics of the PLAY Project model
- (b) Completing a 6-week online course to learn how to complete video reviews and write-ups
- (c) Undergoing 12–18 months of supervision to assure clinical fidelity to the model

After taking the online course, *PLAY Consultants-in-Training* may begin seeing families while under supervision. Supervision involves submitting 15 online videos and write-ups based on their clinical caseload to PLAY Project supervisors. The supervisors evaluate their write-ups according to the PLAY Project Fidelity Manual. Upon completion of supervision, consultants are then certified and licensed to use the PLAY Project model.

See Also

- [Developmental Intervention Model](#)
- [Parental Response to Diagnosis](#)
- [Social Stories](#)
- [US Social Policy and Autism Spectrum Disorders](#)

References and Reading

- Abbeduto, L., Seltzer, M. M., Shattuck, P., et al. (2004). Psychological well-being and coping in mothers of youths with autism, Down syndrome or Fragile X syndrome. *American Journal on Mental Retardation*, 109(3), 237–254.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5)*. Arlington: American Psychiatric Association.
- Binns, & Oram-Cardy. (2019). Developmental social pragmatic interventions for preschoolers with autism spectrum disorder: A systematic review. *Autism & Developmental Language Impairments*, 4(1), 1–18. <https://doi.org/10.1177/2396941518824497>.
- Carter, A. S., Martinez-Pedraza, F. d. L., & Gray, S. A. O. (2009). Stability and individual change in depression symptoms among mothers raising young children with ASD: Maternal and child correlates. *Journal of Clinical Psychology*, 65(12), 1270–1280.
- Green, J., Charman, T., McConachie, H., Aldred, C., Slonims, V., Howlin, P., et al. (2010). Parent-mediated communication-focused treatment in children with autism (PACT): A randomized controlled trial. *The Lancet*, 375(9732), 2152–2160.
- Greenspan, S. I. (1992). *Infancy and early childhood: The practice of clinical assessment and intervention with emotional and developmental challenges*. Madison: International Universities Press.
- Greenspan, S. I., & Wieder, S. (2005). *Infant and early childhood mental health: A comprehensive developmental approach to assessment and intervention*. Arlington: American Psychiatric Publishing, Inc..
- Greenspan, S., DeGangi, G., & Wieder, S. (2001). *Functional emotional assessment scale*. Bethesda: Interdisciplinary Council on Developmental and Learning Disorders.
- Hyman, S. L., Levy, S. E., Myers, S. M., & AAP COUNCIL ON CHILDREN WITH DISABILITIES, SECTION ON DEVELOPMENTAL AND BEHAVIORAL PEDIATRICS. (2020). Identification, evaluation, and management of children with autism spectrum disorder. *Pediatrics*, 145(1), e20193447.
- Mahoney, G., & Kim, J. M. (2007). Pivotal behavior model of developmental learning. *Infant Young Child*, 20, 311–325.

- Mahoney, G., Powell, A., & Finger, I. (1986). The maternal behavior rating scale. *Topics Early Childhood Special Education*, 6, 44–56.
- Mandell, D., Barry, C. et al. (2016). Effects of autism spectrum disorder insurance mandates on the treated prevalence of autism spectrum disorder. *JAMA Pediatrics*. <https://doi.org/10.1001/jamapediatrics.1049>. Published online 11 July 2016.
- Mercer, J. (2015). Examining DIR/floortime as a treatment for children with autism spectrum disorders: A review of research and theory. *Research on Social Work Practice*, 5, 1–11.
- Sandbank, M., Bottema-Beutel, K., Crowley, S., Cassidy, M., Dunham, K., Feldman, J. I., Crank, J., Albarran, S. A., Raj, S., Mahbub, P., & Woynaroski, T. G. (2019, November 25). Project AIM: Autism intervention meta-analysis for studies of young children. *Psychological Bulletin*. Advance online publication. <https://doi.org/10.1037/bul0000215>
- Siller, M., Hutman, T., & Sigman, M. (2013). A parent-mediated intervention to increase responsive parental behaviors and child communication in children with ASD: A randomized, clinical trial. *Journal of Autism and Developmental Disorders*, 43(3), 540–550.
- Siller, M., Swanson, M., Gerber, A., Hutman, T., & Sigman, M. (2014). A parent-mediated intervention that targets responsive parental behaviors increases attachment behaviors in children with ASD: Results from a randomized, clinical trial. *Journal of Autism and Developmental Disorders*, 44, 1720–1732.
- Singer, G. H. (2006). Meta-analysis of comparative studies of depression in mothers of children with and without developmental disabilities. *American Journal on Mental Retardation*, 111(3), 155–169.
- Smith, T., & Iadarola, S. (2015). Evidence-based update for autism spectrum disorders. *Journal of Clinical Child & Adolescent Psychology*, 44(6), 897–922.
- Solomon, R., Necheles, J., Ferch, C., & Bruckman, D. (2007). Pilot study of a parent training program for young children with autism: The P.L.A.Y. Project home consultation program. *Autism*, 11(3), 205–224.
- Solomon, R., Van Egeren, L., Mahoney, G., Quon-Huber, M., & Zimmerman, P. (2014). PLAY project home consultation intervention program for young children with autism spectrum disorders: A randomized controlled trial. *Journal of Developmental and Behavioral Pediatrics*, 35(8), 475–485.
- Wong, C., Odom, S. L., et al. (2014). *Evidence-based practices for children, youth, and young adults with autism spectrum disorder*. Chapel Hill: The University of North Carolina, Frank Porter Graham Child Development Institute, Autism Evidence-Based Practice Review Group. This report is available at: <http://autismpdc.fpg.unc.edu/sites/autismpdc.fpg.unc.edu/files/2014-EBP-Report.pdf>.

Play Space Design in Autism

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Definition: Play Space Design in Autism

Play is defined as behavior that is “freely chosen, personally directed and intrinsically motivated,” a definition drawn from Playwork Principles (PPSG 2005). It is a human right (UNCRC 1989), so it is incumbent on community facilities, notably schools and local authorities, to provide suitably designed spaces for children to be able to play. Play space design for children (and indeed adults) with autism has to involve consideration of what is “suitable” for the capacities and motivations of this group. This entry covers considerations of outdoor play space for primarily school-aged children. It does not cover play as in gaming and touches only briefly on virtual spaces.

Background

There is a long history of designing for children’s play, from the earliest construction of the first toys, objects specifically designed for children to play with, to the influence of the German movement of the nineteenth century to provide spaces for children to play off the street and the first purpose-built playground, built in Manchester, UK, in 1859 (ESPPLAY 2012). The evolution of design for special needs, and autism in particular, has been driven by changes in thinking about design more generally. Changes in the past decades include ideas about inclusive and universal design, consideration of separate or shared facilities, the importance of physical health and mental well-being, and the sometimes conflicting properties of “natural” play and forest schools vs. the use of technology to design virtual and augmented play spaces.

Current Knowledge

Development and Play

Play is deemed by psychologists to be a crucial part of children's development, with a special role in cognitive and social growth. Very generally, functional play (such as play with objects) helps children learn about physical properties of the world, causal relations, and how things work. Differences in object play have been reported in autism, and tangible toys are sometimes developed and recommended to support shared object play (e.g., Farr et al. 2010). Symbolic play involves using objects, actions, or ideas to stand for other such objects and in particular concerns pretend play. Pretend play is marked as being delayed or different in autism (REF) (see Chaudry and Dissanayake 2015). It can be a solo activity, in which objects are treated as if they were something else, e.g., using a banana as if it were a telephone. This kind of play has a crucial role in the theoretical position that autism involves a lack of theory of mind (Leslie 1987). Pretend play can also be social, as in role-playing games such as "playing house" or superhero games. Social play with peers can be a valuable support for developing social skills such as turn-taking, reciprocity, and cooperation (Whitebread et al. 2017). In turn, cooperating with others can be a crucial stepping stone to developing complex group relations and is claimed as an important human evolutionary step (Moll and Tomasello 2007). Again, social play is often different or delayed in autism (Chaudry and Dissanayake 2015).

Given these developmental benefits of play, the design of play spaces for children with autism is clearly important, with potential for providing opportunities for development in exactly those areas that are known to be different or delayed in autism.

Playground Design and Social Interaction

As noted above, children with autism tend to show less or different pretend play, and social forms of play, than typically developing (TD) children. A valuable empirical base demonstrating greater time in solitary and less in social play among

children with autism (30% of time vs. 9% for solitary in ASC vs TD, 40% vs. 70% for more social play) is provided by Locke et al. (2016). The role of design in supporting social play is evidenced in a study by Yuill et al. (2007), who took advantage of the redesign of a school playground to compare play in two settings. Eight 6-year-old autistic boys in the unit were observed for three 45-min lunchtime playground sessions in the old playground, which they shared with children with language difficulties. The ASC group then moved to a new playground designed specifically for them and were observed for three further sessions. The playground was designed to have an appropriate level of physical challenge (e.g., height of slide and climbing walls), support for pretend play (a painted railway track), plans for supporting guided movement (e.g., positioning of the track and the slide exit), and points enabling solitary observation and nonparticipation. Solitary play reduced from about 37% to 21% of the time, and the more social form of group play increased from 10% to 24%. Social initiations also increased significantly. Surprisingly little further naturalistic research of this type has been reported.

Important Considerations in Play Design

Two concepts in the field of design have particular relevance for autism. First, there is a strong movement for participatory design, where the people who might use facilities are involved, from the very earliest stages, in planning and designing the facilities. In autism in particular, many advocate that research and intervention generally need to be much more participatory than in the past (Frauenberger et al. 2017). For example, Frauenberger et al. asked minimally verbal autistic children what objects they wanted to design, rather than designing to an external brief and then providing a set of limited, pre-defined choices of features such as color or shape.

Second, design can be approached by tailoring designs to the specific needs of a particular group (such as dropped curbs for wheelchair users) or a universal design approach that provides designs to make access possible for an otherwise excluded group, but does not provide obstacles for any

other users: automatic doors are an example (<http://universaldesign.ie/what-is-universal-design/the-10-things-to-know-about-ud/10-things-to-know-about-ud.html>).

A good example of design considerations for integrated intergenerational participation is provided at <http://nda.ie/Publications/Others/Research-Promotion-Scheme/Community-Parks-and-Playgrounds-Intergenerational-Participation-through-Universal-Design1.pdf>.

A further question in design is whether the space is designed to cater for particular behaviors or to change those behaviors in some way, either by discouraging behaviors some might perceive as unhelpful (e.g., banning fidget objects that support stimming) or encouraging behaviors seen as desirable (e.g., more social forms of play, opportunities for physical challenge). Christensen and Romero (2016) provide a useful scoping review on design considerations for outdoor play in autism, though this does not include a review of evaluative research.

Integration or Separate Provision?

In relation to play space design in autism, this raises the question of who the space is for. The space might be specially tailored for the perceived needs and preferences of autistic children, or there might be a specific goal to support integration of these children with TD children, perhaps because each group might benefit from interaction with the other. For example, autistic children might observe and learn about typical social behaviors such as cooperation and pretence, while TD children could benefit from understanding better the needs and differences of children with autism. Discussion of integrated vs. separate spaces and information on the design and legislative frameworks in Australia, the UK, and the USA are provided by Burke (2013).

Pragmatically, play leaders will need to decide what will benefit users in particular situations, and thus spaces might be used separately or together at different times. For example, one group might benefit from play equipment being changed frequently, but others, such as many children with autism, might find a predictable pattern more supportive. Play leaders will

also have different aims, and it is useful to be explicit about what these are. Given that play is seen as especially valuable for facilitating social skills and opportunities and that these aspects are part of the definition of differences in autism, much of the research and intervention in playground design has involved design to support social interaction.

Play Interventions

Designs could be made to support interaction between children with autism, or such children with TD peers, or such children with adults. A review of teaching play skills in autism is provided by Jung and Sainato (2013). In the last case, most work has involved structured play interventions, and these will be mentioned only briefly here.

Adult-Supported Play Interventions

Two well-developed interventions of this sort are:

The Integrated Play Group (IPG) model involves adults and “expert” players supporting the play of “novice” players (autistic children are seen as novices here).

<https://www.iidc.indiana.edu/pages/Play-Time-An-Examination-Of-Play-Intervention-Strategies-for-Children-with-Autism-Spectrum-Disorders>

Floor time

<https://www.iidc.indiana.edu/pages/Play-Time-An-Examination-Of-Play-Intervention-Strategies-for-Children-with-Autism-Spectrum-Disorders>

Risk, Resilience, Independence, and Agency

There is little work on risky play in relation to autism in particular, and as one might expect, caregivers of children with any difference or disability often have protection from risk as a main concern, rather than opportunity to learn about risk. Serman et al. (2016) provided a systematic review of the few studies in this area highlighting considerations for play spaces for children with disabilities, and Goodley and Runswick-Cole (2010) argue for the need to include risk in spaces for children with

disabilities. Clearly, risk assessments are crucial in any play space design: for a simple approach, see Coleman (2019). Spaces for children with autism need to involve consideration of risks likely in the particular group. For example, a shared space for children with speech and language difficulties and those with autism involved a conflict between frequently changing play objects to provide novelty vs. providing predictability, which some autistic children preferred Yuill et al. (2007). There are also considerations appropriate to children at developmental levels below their chronological age, or with sensory needs, e.g., preventing children ingesting bark chippings or providing a range of objects with interesting sensory properties of touch or smell.

There is little theoretical work in this area in relation to autism, but an increasing amount of practical work and design in relation to TD children and mainstream schools. There is extensive documentation of the philosophy, policy, background, and evaluation of this approach in schools on the Outdoor Play and Learning (OPAL) site: <https://outdoorplayandlearning.org.uk/home/for-schools/research/>.

There is also research into the benefits of teaching children to evaluate risk, particularly in relation to outdoor play, against a background of concerns that perceived fear of litigation for injury will reduce children's opportunities to learn about risk management by making decisions during free play. A small systematic review suggests that benefits of such play include physical health but also social benefits (Brussoni et al. 2015).

Physical Fitness

Menear and Neumeier (2015) provide suggestions based on research about how to support physical activity in play activities for children with autism, with particular focus on the motor difficulties that many such children experience.

Natural Play Spaces

The concept of forest schools and the value of natural objects and environments for well-being generally have been cited as a reason to provide such environments for children with autism.

There is little robust research to evaluate this question, but a useful review of the views and experiences of parents of autistic children in Tongji, China, is provided by Li et al. (2019), suggesting that parents often see the sensory, physical, and social benefits of play may fear its risks.

Virtual Play Spaces

There has been a comparatively large amount of work on technology-supported play spaces (such as virtual spaces) in relation to autism, reflecting the idea that some aspects of physical spaces and the physical presence of others can be overwhelming in autism, with virtual others and spaces providing room to step out of the situation and to have more control over stimulation levels. Several of these are showcased in the Digital Bubbles archive (digitalbubbles.org.uk especially seminar 4]). For example, Pares and colleagues describe an augmented-reality environment, Lands of Fog, that subtly supports users to physically interact with each other in shared exploration of a technology-augmented environment representing a land covered in virtual fog, which can be revealed through peepholes as the players explore it, as well as to play games such as catching "fireflies." New elements and creatures can only be found by acting collaboratively with another player. Over multiple lab sessions, children made more initiations (Mora-Guiard et al. 2017) and different features of the game supported social interaction (Crowell et al. 2019).

Future Directions

As technology and the use of augmented objects and spaces develop apace, there will be further possibilities for the design of augmented play spaces, though it is hard to predict which features and tools will move beyond the experimental demonstration. Concerns about physical fitness and mental well-being through "natural environments" may become increasingly pressing, particularly given environmental degradation and changing global conditions. At the same time, there is substantial room for larger, more

prolonged studies of children's behavior in existing play settings, to test existing ideas about how design influences social behavior. For example, Yuill et al. (2014) report a study of an augmented "knight's castle" playset in which they tracked how the design of the object – which provides play objects with apparent agency to make relevant sounds and speeches as they are placed in different locations – prompted increases in the likely success of a child's attention bid, which in turn seemed to lead to longer bouts of social play. The wider availability of video tracking and automated coding in natural play settings makes this sort of research increasingly possible, and this would deepen our understanding of the mechanics of playground design in shaping and supporting different patterns of behavior in children on the autism spectrum.

References and Reading

- Brussoni, M., Gibbons, R., Gray, C., Ishikawa, T., Sandseter, E., Bienenstock, A., . . . & Pickett, W. (2015). What is the relationship between risky outdoor play and health in children? A systematic review. *International Journal of Environmental Research and Public Health*, 12(6), 642–6454.
- Brussoni, M., Ishikawa, T., Brunelle, S., & Herrington, S. (2017). Landscapes for play: Effects of an intervention to promote nature-based risky play in early childhood centres. *Journal of Environmental Psychology*, 54, 139–150.
- Burke, J. (2013). Just for the fun of it: Making playgrounds accessible to all children. *World Leisure Journal*, 55(1), 83–95.
- Chaudry, M., & Dissanayake, C. (2015). Pretend play in children with autism spectrum disorders. *Children's Play, Pretense, and Story: Studies in Culture, Context, and Autism Spectrum Disorder*, 31–50.
- Christensen, K., & Romero, L. P. R. (2016). Creating outdoor play environments to support social interactions of children with autism spectrum disorder; a scoping study. *Landscape Research Record*, 5, 128–140.
- Coleman, N. (2019). *Managing school playtimes safely*. Available at: <http://outdoorplayandlearning.org.uk/blogs-and-news/opal-blogs/>
- Crowell, C., Mora-Guiard, J., & Pares, N. (2019). Structuring collaboration: Multi-user full-body interaction environments for children with Autism Spectrum Disorder. *Research in Autism Spectrum Disorders*, 58, 96–110.
- ESPPLAY. (2012). <https://www.espplay.co.uk/the-history-of-playgrounds/>
- Farr, W., Yuill, N., & Raffle, H. (2010). Social benefits of a tangible user interface for children with autistic spectrum conditions. *Autism*, 14(3), 237–252.
- Frauenberger, C., Makhaeva, J., & Spiel, K. (2017). Blending methods: Developing participatory design sessions for autistic children. In *Proceedings of the 2017 conference on interaction design and children* (pp. 39–49). New York: ACM.
- Goodley, D., & Runswick-Cole, K. (2010). Emancipating play: Dis/abled children, development and deconstruction. *Disability & Society*, 25(4), 499–512.
- Jung, S., & Sainato, D. M. (2013). Teaching play skills to young children with autism. *Journal of Intellectual and Developmental Disability*, 38(1), 74–90.
- Leslie, A. M. (1987). Pretense and representation: The origins of "theory of mind." *Psychological Review*, 94(4), 412.
- Li, D., Larsen, L., Yang, Y., Wang, L., Zhai, Y., & Sullivan, W. C. (2019). Exposure to nature for children with autism spectrum disorder: Benefits, caveats, and barriers. *Health & Place*, 55, 71–79.
- Locke, J., Shih, W., Kretzmann, M., & Kasari, C. (2016). Examining playground engagement between elementary school children with and without autism spectrum disorder. *Autism*, 20(6), 653–662.
- Meneer, K. S., & Neumeier, W. H. (2015). Promoting physical activity for students with autism spectrum disorder: Barriers, benefits, and strategies for success. *Journal of Physical Education, Recreation and Dance*, 86(3), 43–48.
- Moll, H., & Tomasello, M. (2007). Cooperation and human cognition: The Vygotskian intelligence hypothesis. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 362(1480), 639–648.
- Mora-Guiard, J., Crowell, C., Pares, N., & Heaton, P. (2017). Sparking social initiation behaviors in children with autism through full-body interaction. *International Journal of Child-Computer Interaction*, 11, 62–71.
- PPSG. (2005). http://www.skillsactive.com/PDF/sectors/Playwork_Principles.pdf
- Sterman, J., Naughton, G., Froude, E., Villeneuve, M., Beetham, K., Wyver, S., & Bundy, A. (2016). Outdoor play decisions by caregivers of children with disabilities: A systematic review of qualitative studies. *Journal of Developmental and Physical Disabilities*, 28(6), 931–957.
- UNCRC. (1989). <https://www.unicef.org.uk/what-we-do/un-convention-child-rights/>
- Whitebread, D., Neale, D., Jensen, H., Liu, C., Solis, S. L., Hopkins, E., Hirsh-Pasek, K., & Zosh, Z. M. (2017). *The role of play in children's development: A review of the evidence*. https://www.legofoundation.com/media/1065/play-types--development-review_web.pdf
- Yuill, N., Strieth, S., Roake, C., Aspden, R., & Todd, B. (2007). Brief report: Designing a playground for children with autistic spectrum disorders – Effects on playful peer interactions. *Journal of Autism and Developmental Disorders*, 37(6), 1192–1196.

Yuill, N., Hinske, S., Williams, S. E., & Leith, G. (2014). How getting noticed helps getting on: Successful attention capture doubles children's cooperative play. *Frontiers in Psychology*, 5, 418.

Play Therapy

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Synonyms

[Directive play therapy](#); [Nondirective play therapy](#); [Play-based interventions](#)

Definition

Play therapy is a method of treatment that is provided to children by a trained professional. It uses play within a therapeutic setting to address social, emotional, and behavioral difficulties. Traditionally, the underlying premise of play therapy was that children use symbolic play as a way to represent their internal experiences (e.g., beliefs, feelings) and, simultaneously, can develop and practice healthy coping skills through play. Play therapy is significantly different from the play children engage in with their peers or other untrained adults in their lives (e.g., parents or babysitters) because the therapist interprets children's play behaviors in an effort to achieve treatment goals. There are various forms of play therapy interventions as well as growing empirical evidence for treating childhood difficulties, such as trauma and anxiety. In addition, play therapy has been incorporated into other empirically established treatments for children, such as cognitive behavior therapy. Play therapy varies based on the reason the child enters into treatment, the age of the child, and the training background of the therapist. The therapist observes and joins in the play to obtain a better understanding of the child, connects the information gathered

from these interactions to the reasons for which treatment was initially requested, and then applies this understanding to develop and implement treatment goals. Positive changes are achieved by sharing interpretations with the child in an understandable and child-friendly manner within the play, collaborating with the child to develop and practice better coping skills using play, facilitating communication of feelings and thoughts in a safe and emotionally corrective environment, as well as involving and coordinating treatment goals with the child's family.

At this time, there is no established empirical support for the use of play therapy in treating youth with ASD (National Research Council 2001). Historically, traditional talk and play therapies have not been considered a helpful intervention to address the core symptoms of autism spectrum disorder (ASD), a point noted early in the field by Kanner and others. Impairments in symbolic, imaginary play characteristic of ASD (Stahmer et al. 2003) as well as the strong empirical evidence in support of behavioral interventions based on the principles of applied behavior analysis to treat core ASD impairments (Dawson et al. 2010; Koegel et al. 2010; Lovaas 1987) have precluded play therapy as an intervention for ASD. However, one area of focus of behavioral intervention for children with ASD has been to develop play skills, including symbolic play (Stahmer et al. 2003). Additionally, for children with ASD, play has been used to facilitate functional communication and social skills (e.g., Dawson et al. 2010; Koegel et al. 2010).

See Also

- ▶ [Early Intensive Behavioral Intervention \(EIBI\)](#)
- ▶ [Naturalistic Interventions](#)
- ▶ [Pivotal Response Training](#)
- ▶ [Symbolic Play](#)

References and Reading

Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenon, J., et al. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The early start Denver model. *Pediatrics*, 125(1), e17–e23.

- Drewes, A. A. (2009). *Blending play therapy with cognitive behavioral therapy: Evidence-based and other effective treatments and techniques*. Hoboken: Wiley.
- Koegel, R. L., Koegel, L. K., Vernon, T. W., & Brookman-Frazee, L. I. (2010). Empirically supported pivotal response treatment for children with autism spectrum disorders. In J. R. Weisz & A. E. Kazdin (Eds.), *Evidence-based psychotherapies for children and adolescents* (2nd ed., pp. 327–344). New York: Guilford Press.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55(1), 3–9.
- National Research Council. (2001). Educating children with autism. Committee on educational interventions for children with autism. In C. Lord & J. P. McGee (Eds.), *Division of behavioral and social sciences and education*. Washington, DC: National Academy Press.
- Reddy, L. A., Files-Hall, T. M., & Schaefer, C. E. (2005). *Empirically based play interventions for children*. Washington, DC: American Psychological Association.
- Stahmer, A. C., Ingersoll, B., & Carter, C. (2003). Behavioral approaches to promoting play. *Autism: The International Journal of Research and Practice*, 7, 401–413.

Play-Based Interventions

► Play Therapy

Pleiotropy

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Synonyms

Multiple phenotypes associated with disruption of a single gene

Structure

An individual's set of genes is referred to as their *genotype*, and how these genes influence the expression of physical characteristics, or the

presence of a disorder, is considered an individual's *phenotype*. Because most genes encode an RNA molecule or a protein sequence, disruption of a gene by a mutation that prevents it from functioning has the potential to interfere with whatever processes the gene product influences. For this reason, mutations in a single gene or pair of genes may affect organ systems ranging from, for example, the central nervous system, cardiovascular system, and connective tissue. *Pleiotropy* is the genetic phenomenon in which disruption of the same gene results in a range of clinical symptoms. Because multiple organ systems may be affected, often, it is challenging to explain how disruption of one gene leads to such a diversity of phenotypes or clinical presentations (Jorde et al. 2010; Nussbaum et al. 2007).

Although ASDs are considered to be the most genetic of developmental neuropsychiatric disorders, based on studies of monozygotic twins and recurrence risk in siblings, marked heterogeneity at both the genotypic and phenotypic levels has made elucidating the genetic underpinnings of ASD a formidable challenge (O'Roak and State 2008). ASD is characterized by marked *locus* and *allelic heterogeneity*, such that multiple genes at different locations throughout the genome and distinct variants within the same gene, respectively, can influence an individual's clinical presentation. Our current conceptualization of the genetic architecture of ASD is that it is complex, involving the interplay of genetic variation, which may be present in multiple risk genes throughout the genome, inherited or *de novo*, rare or common, and present at the level of the sequence of DNA (single nucleotide variants) or the structure of chromosomes (copy number variants, CNVs); environmental; and epigenetic components – all of which contribute to clinical presentation, which also may be heterogeneous (State and Levitt 2011).

Further complicating our ability to elucidate how genes associated with risk cause clinical symptoms in ASD are emerging findings from recent studies showing that these genes are highly pleiotropic, in that they appear to confer risk for multiple clinical presentations. On the one hand, there is strong evidence for a number of candidate

genes and chromosomal regions, such as *Neurologin 4X (NLGN4X)*, *SH3 and multiple ankyrin repeat domains 3 (SHANK3)*, *contactin-associated protein-like 2 (CNTNAP2)*, 16p11, 22q11, and others (see below), in ASD. However, recent large-scale studies have implicated the same genes and structural variants across a range of psychiatric disorders, including schizophrenia, attention-deficit hyperactivity disorder (ADHD), and Tourette syndrome (TS), and in some cases, neurological disorders, such as seizure disorder, and even obesity (discussed in more detail below). Therefore, pleiotropy has emerged as a central theme in the genetics of ASD, forcing us to reexamine our understanding of how genetic risk informs phenotypic specificity (State 2010; State and Levitt 2011).

Function

There are a growing number of examples of variation in the same gene or chromosome region predisposing to a diversity of phenotypes, including ASD. All of the examples discussed here are genes or CNVs that were initially identified as susceptibility genes/regions through what can be described as a rare variant approach. This approach involves looking for outliers, in particular rare, *de novo* abnormalities in chromosome structure, and is hypothesized to enrich for the identification of genes of major effect. Therefore, there is strong evidence implicating these risk genes and CNVs in ASD, which makes finding these same variants in other disorders difficult to interpret.

One chromosomal region that has been strongly implicated in ASD in multiple studies is 16p11.2. Specifically, structural abnormalities affecting this region have been found in ~1% of families with ASD. These studies showed that recurrent deletions and duplications of this region occur with increased frequency in individuals with ASD. CNVs in this region were found to be either transmitted or *de novo* (Kumar et al. 2008; Marshall et al. 2008; Sanders et al. 2011; Sebat et al. 2007; Weiss et al. 2008). Despite the strong evidence for this region in ASD, CNVs at 16p11 have also been implicated across psychiatric

diagnoses. For example, Weiss and colleagues identified deletions in this region in individuals with schizophrenia, bipolar disorder, ADHD, and dyslexia.

Additional studies provided further support for an association of CNVs, specifically duplications at 16p11 in schizophrenia and childhood-onset schizophrenia (McCarthy et al. 2009; Walsh et al. 2008). Moreover, one of these studies specified that 16p11.2 microduplications were significantly associated not only with the risk of schizophrenia but of bipolar disorder and autism, while the reciprocal microdeletion was associated with autism and developmental disorders (McCarthy et al. 2009). In addition, more recent studies have reported that structural abnormalities in this region are found in intellectual disability that is not associated with autism (Bijlsma et al. 2009) and even in obesity (Bochukova et al. 2010).

Therefore, it would seem that instability of chromosome structure in this region predisposes to a range of psychiatric disorders. However, it is important to observe that CNVs in this region have also been found in unaffected individuals (Glessner et al. 2009). In addition, even some of the first deletions in this region identified in families with multiple affected individuals with ASD by Weiss and colleagues did not segregate with the presence of the disorder, such that affected siblings did not share this CNV. While this observation flies in the face of what would be expected in Mendelian disorders, it is consistent with findings in disorders with complex genetic architecture, such as ASD (O’Roak and State 2008).

Another highly pleiotropic region is chromosome 22q11.2. Deletion of this region causes velocardiofacial syndrome or DiGeorge syndrome, a pleiotropic syndrome that leads to abnormalities in multiple organ systems, including immune deficiencies, cardiac defects, and mild dysmorphic facial features. While CNVs in this region have been associated with ASD (Glessner et al. 2009; Marshall et al. 2008), 22q11 deletions have also been found to increase the risk of a range of psychiatric disorders, most notably early-onset psychotic disorders, schizophrenia, ADHD, and anxiety (Green et al. 2009).

While it seems plausible that pleiotropy might occur due to disruption of a chromosomal region,

which encompasses many genes, it is surprising that single genes and gene families strongly associated with ASD are being implicated in other psychiatric disorders as well. This finding has led to the conclusion that perhaps similar biological mechanisms underlie these seemingly disparate disorders (see discussion below). For example, *neuroligins* and *neurexins*, which are cell adhesion molecules that are found at the synapse and play a role in synapse formation by interacting with each other across the synaptic cleft, and *SHANKs*, which interact intracellularly with neuroligins (Sudhof 2008), and were first identified as susceptibility genes in ASD (Durand et al. 2007; Glessner et al. 2009; Jamain et al. 2003; Laumonnier et al. 2004; Marshall et al. 2008), have also been implicated in schizophrenia (Gauthier et al. 2010, 2011).

Similarly, *CNTNAP2* was identified as a risk gene for ASD because it was disrupted by a single base pair deletion that was found in consanguineous families, such that individuals homozygous for the mutation had a syndrome characterized by focal epilepsy and cortical abnormalities (Strauss et al. 2006). Multiple additional studies, including those adopting both rare and common variant approaches, have provided evidence that disruption of this gene is likely to be associated with an increased risk of ASD. As was the case with the neurexins and neuroligins, subsequent studies have implicated *CNTNAP2* in multiple psychiatric and neurodevelopmental disorders, including intellectual disability, Tourette syndrome, language disorders, ADHD, and schizophrenia (State 2010).

Pathophysiology

How then do we interpret the overlapping clinical presentations associated with the same structural and single gene variants, as described above? Possible explanations that have been proposed in response to these observations include differences in diagnostic assessments, such that social disability could be defined as ASD in one study, and as representative of the negative symptoms of schizophrenia in another (State and Levitt 2011). However, as discussed in State and Levitt (2011),

this scenario is unlikely to account for the breadth of findings described above, particularly given that ASD and schizophrenia, as defined in *DSM-IV-TR*, have distinct diagnostic criteria, ages of onset, and clinical courses. Alternatively, it is possible that variation in the same chromosomal region or at the same locus might produce either gain- or loss-of-function effects, with respect to the gene product that is disrupted, or divergent effects on gene expression, ultimately leading to different clinical presentations. Another possibility is type I error associated with publication bias (State & Levitt).

At the same time, one current model proposes that the high degree of pleiotropy associated with these genes and chromosomal regions predisposes to a diverse range of clinical presentations. More specifically, structural variation at regions such as 16p11 or 22q11, or sequence variation in risk genes, including *NLGN4X* or *CNTNAP2*, may represent a risk for the disruption of common biological processes, such as synapse formation. Variation at other sites throughout the genome, i.e., locus heterogeneity, interacts with the known risk variant(s) to influence the same biological processes. Moreover, environmental and epigenetic factors also affect gene expression and these same processes in ways that are not well understood (State 2010; State and Levitt 2011).

Support for this hypothesis comes from recent whole-exome sequencing studies of families with developmental brain malformations, in which homozygous mutations in the gene *WDR62* were found to lead to multiple distinct cortical abnormalities, including microcephaly, lissencephaly, and hypoplasia of the corpus callosum (Bilguvar et al. 2010). Because psychiatric disorders involve behavioral abnormalities, which are markedly heterogeneous, it is possible that disruption of a single gene is even more likely to predispose to a range of behavioral symptoms. Taken together, according to this model, neurodevelopmental processes are uniquely shaped by the combination of variation in pleiotropic genes/regions, their interaction with other genetic loci, and environmental factors, which has the potential to result in a diverse range of neuropsychiatric disorders (State 2010; State and Levitt 2011).

See Also

- [Chromosomal Abnormalities](#)
- [Neuroligins](#)

References and Reading

- Bijlsma, E. K., Gijsbers, A. C., Schuurs-Hoeijmakers, J. H., van Haeringen, A., Fransen van de Putte, D. E., & Anderlid, B. M. (2009). Extending the phenotype of recurrent rearrangements of 16p11.2: Deletions in mentally retarded patients without autism and in normal individuals. *European Journal of Medical Genetics*, 52(2–3), 77–87.
- Bilguvar, K., Ozturk, A. K., Louvi, A., Kwan, K. Y., Choi, M., Tatli, B., et al. (2010). Whole-exome sequencing identifies recessive WDR62 mutations in severe brain malformations. *Nature*, 467(7312), 207–210.
- Bochukova, E. G., Huang, N., Keogh, J., Henning, E., Purmann, C., Blaszczak, K., et al. (2010). Large, rare chromosomal deletions associated with severe early-onset obesity. *Nature*, 463(7281), 666–670.
- Durand, C. M., Betancur, C., Boeckers, T. M., Bockmann, J., Chaste, P., Fauchereau, F., et al. (2007). Mutations in the gene encoding the synaptic scaffolding protein SHANK3 are associated with autism spectrum disorders. *Nature Genetics*, 39(1), 25–27.
- Gauthier, J., Champagne, N., Lafreniere, R. G., Xiong, L., Spiegelman, D., Brustein, E., et al. (2010). De novo mutations in the gene encoding the synaptic scaffolding protein SHANK3 in patients ascertained for schizophrenia. *Proceedings of the National Academy of Sciences of the United States of America*, 107(17), 7863–7868.
- Gauthier, J., Siddiqui, T. J., Huashan, P., Yokomaku, D., Hamdan, F. F., Champagne, N., et al. (2011). Truncating mutations in NRXN2 and NRXN1 in autism spectrum disorders and schizophrenia. *Human Genetics*, 130(4), 563–573.
- Glessner, J. T., Wang, K., Cai, G., Korvatska, O., Kim, C. E., Wood, S., et al. (2009). Autism genome-wide copy number variation reveals ubiquitin and neuronal genes. *Nature*, 459(7246), 569–573.
- Green, T., Gothelf, D., Glaser, B., Debbane, M., Frisch, A., & Kotler, M. (2009). Psychiatric disorders and intellectual functioning throughout development in velocardiofacial (22q11.2 deletion) syndrome. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(11), 1060–1068.
- Jamain, S., Quach, H., Betancur, C., Rastam, M., Colineaux, C., Gillberg, I. C., et al. (2003). Mutations of the X-linked genes encoding neuroligins NLGN3 and NLGN4 are associated with autism. *Nature Genetics*, 34(1), 27–29.
- Jorde, L. B., Carey, J. C., & Bamshad, M. J. (2010). *Jorde: Medical genetics* (4th ed.). Philadelphia: Mosby.
- Kumar, R. A., KaraMohamed, S., Sudi, J., Conrad, D. F., Brune, C., & Badner, J. A. (2008). Recurrent 16p11.2 microdeletions in autism. *Human Molecular Genetics*, 17(4), 628–638.
- Laumonnier, F., Bonnet-Brilhault, F., Gomot, M., Blanc, R., David, A., Moizard, M. P., et al. (2004). X-linked mental retardation and autism are associated with a mutation in the NLGN4 gene, a member of the neuroigin family. *American Journal of Human Genetics*, 74(3), 552–557.
- Marshall, C. R., Noor, A., Vincent, J. B., Lionel, A. C., Feuk, L., Skaug, J., et al. (2008). Structural variation of chromosomes in autism spectrum disorder. *American Journal of Human Genetics*, 82(2), 477–488.
- McCarthy, S. E., Makarov, V., Kirov, G., Addington, A. M., McClellan, J., & Yoon, S. (2009). Microduplications of 16p11.2 are associated with schizophrenia. *Nature Genetics*, 41(11), 1223–1227.
- Nussbaum, R. L., McInnes, R. R., & Willard, H. F. (2007). *Nussbaum: Thompson & Thompson genetics in medicine* (7th ed.). Philadelphia: Saunders Elsevier.
- O’Roak, B. J., & State, M. W. (2008). Autism genetics: Strategies, challenges, and opportunities. *Autism Research*, 1(1), 4–17.
- Sanders, S. J., Ercan-Sencicek, A. G., Hus, V., Luo, R., Murtha, M. T., & Moreno-De-Luca, D. (2011). Multiple recurrent de novo CNVs, including duplications of the 7q11.23 Williams syndrome region, are strongly associated with autism. *Neuron*, 70(5), 863–885.
- Sebat, J., Lakshmi, B., Malhotra, D., Troge, J., Lese-Martin, C., Walsh, T., et al. (2007). Strong association of de novo copy number mutations with autism. *Science*, 316(5823), 445–449.
- State, M. W. (2010). The genetics of child psychiatric disorders: Focus on autism and Tourette syndrome. *Neuron*, 68(2), 254–269.
- State, M. W., & Levitt, P. (2011). The conundrums of understanding genetic risks for autism spectrum disorders. *Nature Neuroscience*, 14(12), 1499–1506.
- Strauss, K. A., Puffenberger, E. G., Huentelman, M. J., Gottlieb, S., Dobrin, S. E., Parod, J. M., et al. (2006). Recessive symptomatic focal epilepsy and mutant contactin-associated protein-like 2. *The New England Journal of Medicine*, 354(13), 1370–1377.
- Sudhof, T. C. (2008). Neuroigins and neurexins link synaptic function to cognitive disease. *Nature*, 455(7215), 903–911.
- Walsh, T., McClellan, J. M., McCarthy, S. E., Addington, A. M., Pierce, S. B., Cooper, G. M., et al. (2008). Rare structural variants disrupt multiple genes in neurodevelopmental pathways in schizophrenia. *Science*, 320(5875), 539–543.
- Weiss, L. A., Shen, Y., Korn, J. M., Arking, D. E., Arking, D. E., Miller, D. T., & Fossdal, R. (2008). Association between microdeletion and microduplication at 16p11.2 and autism. *The New England Journal of Medicine*, 358(7), 667–675.

Plica Palpebronasalis

- [Epicanthic Fold](#)

PLS-5

- [Preschool Language Scale – 5](#)

PLSI

- [Pragmatic Language Skills Inventory](#)

Plumbism

- [Lead Exposure and Autism](#)

PM

- [Prospective Memory in Autism Spectrum Disorder](#)

Pms-Haloperidol

- [Haloperidol](#)

Pneumoencephalography

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Synonyms

[Encephalography](#); [PEG](#); [Ventriculography](#)

Definition

Pneumoencephalography is a medical procedure allowing for the structure of the brain to show up more clearly on an x-ray. First introduced in 1919, this procedure uses a lumbar puncture to remove cerebrospinal fluid from the brain, and then air, helium, or oxygen is injected into the lumbar subarachnoid space. See Brown (2012) for description of the procedure and example image. The procedure is rarely used due to significant side effects. It has been replaced by CT scan and other imaging techniques (Kirkman 2015).

In 1968, Aarkrog reported 25 of 46 children with infantile autism or borderline autism had an abnormal PEG (Aarkrog 1968; also see Melchior et al. 1965). Hauser et al. (1975) reported on 18 cases with autism and PEG and found enlargement of the left ventricular system (13/18) and widened left temporal horn reflected in flattening of the hippocampal contours (15/18); only one case was reported as “normal.” The authors concluded that deficits of the medial temporal lobe may be a contributing factor to infantile autism.

See Also

- [Computed Tomography](#)
- [Magnetic Resonance Imaging](#)

References and Reading

- Aarkrog, T. (1968). Organic factors in infantile psychoses and borderline psychoses. Retrospective study of 46 cases subject to pneumoencephalography. *Danish Medical Bulletin*, 15, 283–288.
- Brown, T. C. K. (2012). Neuroradiology investigations before scanning. *Paediatric Anaesthesia*, 22(8), 826–827.
- Hauser, S. L., DeLong, R., & Rosman, N. P. (1975). Pneumographic findings in the infantile autism syndrome. *A correlation with temporal lobe disease*. *Brain*, 98, 667–688.
- Kirkman, M. A. (2015). The role of imaging in the development of neurosurgery. *Journal of Clinical Neuroscience*, 22(1), 55–61.
- Melchior, J. C., Dyggve, H. V., & Glystorff, H. (1965). Pneumonencephalographic examination of 207 mentally retarded patients. *Danish Medical Bulletin*, 12, 38–42.

POI

► Perceptual Organization Index (POI)

Point Prevalence

► Prevalence

Point-Light Displays

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Definition

Point-light displays are constructed by recording movements of people, animals, or objects with markers or point lights attached to the major joints and then processing the videos so that only the point-lights are visible in the resultant displays. These stimuli are used to isolate motion perception processes as they contain limited form information.

Individuals with autism are able to identify human actions in point-light displays. Some behavioral studies indicate typical performance by individuals with ASD on a variety of tasks with point-light displays. However, disruptions in their visual sensitivity to point-light displays of human motion have been found in a variety of tasks including preferential attention, direction detection, and coherence detection. Some studies have reported deficits in labeling emotion in point-light displays. Unlike typically developing individuals, those with autism do not show an enhanced ability to detect human versus animal motion in point-light displays.

Functional magnetic resonance imaging studies present more consistent results as individuals with ASD have repeatedly been found to exhibit

atypical neural responses to biological motion in point-light displays. The posterior superior temporal sulcus is one area of dysfunction reported in such studies. Some researchers have proposed that PSTS dysfunction and disrupted perception of biological motion (in point-light displays) serve as a hallmark of autism spectrum disorders.

See Also

► Biological Motion

References and Reading

- Blake, R., & Shiffrar, M. (2007). Perception of human motion. *Annual Review of Psychology*, 58, 47–73.
- Freitag, C. M., Konrad, C., Haberlen, M., Kleser, C., von Gontard, A., Reith, W., et al. (2008). Perception of biological motion in autism spectrum disorders. *Neuropsychologia*, 46, 1480–1494.
- Herrington, J. D., Baron-Cohen, S., Wheelwright, S. J., Singh, K. D., Bullmore, E. T., Brammer, M., et al. (2007). The role of MT+/V5 during biological motion perception in Asperger syndrome: An fMRI study. *Research in Autism Spectrum Disorders*, 1, 14–27.
- Johansson, G. (1973). Visual perception of biological motion and a model for its analysis. *Perception & Psychophysics*, 14, 201–211.
- Kaiser, M. D., & Pelphrey, K. A. (2011). Disrupted action perception in autism: Behavioral evidence, neuroendophenotypes, and diagnostic utility. *Developmental Cognitive Neuroscience*, 2, 25–35. <https://doi.org/10.1016/j.dcn.2011.05.005>.

Police-Citizen Interactions, Theory of Mind, and ASD

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Definition

This entry focuses on how theory of mind (ToM) deficits may (a) lead to ASD individuals becoming involved in problematic interactions with the police and (b) shape their ability to negotiate such interactions effectively.

Historical Background

Discussions of problematic social interactions that are often experienced by ASD individuals frequently refer to a deficit in theory of mind (ToM) – or what in everyday parlance might be labeled perspective taking – as a key underlying factor. Indeed, some have argued that a ToM deficit is a core deficit which can explain many of the behaviors that characterize ASD individuals (e.g., Baron-Cohen 1995), although this perspective is not universally accepted (e.g., Van de Cruys et al. 2014). ToM refers to the ability to understand the mental states of others, that is, the ability to take the perspective of others and interpret their intentions, their desires, and even their emotions. Individuals with well-developed ToM should be able to infer the underlying or real meaning of the nonverbal and verbal behavior of those with whom they are interacting. Thus, they should be able to read the meaning behind another individual's facial expressions and bodily gestures. They should be able to read the real meaning behind the vocal tone and verbal expressions of others, thereby enabling them to infer if someone is being sarcastic or is trying to trick them, if someone is using figurative speech (e.g., a metaphor) and thus what they say should not be taken literally, or if the other person's reaction to something they have said indicates they have made a faux pas. In other words, well-developed ToM allows the individual to behave in what might generally be considered to be a socially appropriate and adaptive manner in interactive contexts. An impaired ToM provides a convenient explanation for a variety of social inadequacies and misdemeanors.

Perhaps not surprisingly, therefore, ToM deficits have been implicated in hypothesizing about why some ASD individuals become (a) involved in criminal activity and (b) why their ensuing interactions with the police and the courts may become problematic. We use the term “hypothesizing” because much of the available “evidence” comes from case examples. For example, Brewer and Young (2015) described numerous cases where ASD adults apparently became involved, almost unwittingly, in some criminal activity

because, in a desire to accommodate, please, or impress some third party, they did not recognize that party's criminal intentions or failed to appreciate the wrongfulness of their behavior and the impact their behavior had on the victim(s). Further, when interacting with the criminal judicial system, irrespective of any wrongdoing, they behaved in a way that was not helpful. In each of the cases presented, ASD individuals either failed to understand or react to the caution that they were not required to say anything when questioned and that what they said may be used in evidence. Consequently, they continued to engage with the police, often leading to a negative outcome.

How might ToM deficits have contributed to problems such as those just outlined? ASD individuals with deficits in ToM may have (i) behaved inappropriately toward another person because they failed to recognize the cues from that person that indicated their behavior was problematic, (ii) raised the ire of police officers because, having failed to accurately interpret their expectations, they reacted with inappropriate antipathy toward them, (iii) responded inappropriately to reasonable requests from police or court officials because they interpreted figurative language literally rather than in the manner it was intended, or (iv) failed to appreciate that behaviors (e.g., gaze aversion, fidgeting and shuffling, inappropriate emotional reactions) they manifested during investigative or courtroom interviews may have been interpreted as signs of untrustworthiness, indicative of attempts to deceive, or signs of non-cooperation.

ToM deficits may contribute in other ways that prejudice the outcomes of ASD individuals' interactions with police. In all the cases described by Brewer and Young (2015), the interactions with police led to the production of evidence that was used against them. In none of the cases did any of the individuals exercise their right to silence. Given poor ToM, an ASD individual may fail to appreciate that the mindset of the police officers contrasts to their own mindset. For example, Kassin and Norwick (2004) argued that when people believe they have nothing to hide, they also believe their innocence will

save them, referred to as the “innocents’ mindset.” This “illusion of transparency” leads people to infer that their “inner thoughts or emotions are readily apparent to others.” A well-developed ToM may, however, enable one to detect that the other person’s mindset differs from theirs, rendering the individual less vulnerable to engaging in interactions that do not help their case.

Our focus here, however, is not to rehash the array of case study evidence that is consistent with the perspective that ToM deficits may contribute to problematic interactions between ASD adults and the police and ultimately to adverse interactions in the courts. Rather, we present empirical evidence – limited as it is – that highlights the importance of ToM deficits and provide some direction for future research that should broaden our understanding of the nexus between ToM deficits and problematic interactions between ASD individuals and criminal justice system professionals.

Current Knowledge

From the outset it is important to emphasize that we are not suggesting some well-established connection between ASD and crime commission. A number of studies have attempted to examine that issue using a variety of approaches, including investigating whether autistic individuals are overrepresented in prisons and forensic psychiatric facilities, among specific categories of offenders or individuals with recorded interactions with the justice system, or in crime statistics within community samples. Making sense of that body of research literature is not easy due to question marks about issues such as the reliability of the ASD diagnostic information, the possible contributions of comorbid psychiatric disorders, the adequacy of study sample sizes, and biases within the justice system with respect to the differential disposition of cases involving ASD versus typically developing individuals. Nevertheless, following a comprehensive review of the various types of “crime prevalence” studies, Brewer and Young (2015) concluded that the most justifiable conclusion based on present knowledge

is that the prevalence of ASD individuals’ involvement in crime is probably not too dissimilar to that of the broader population.

Nevertheless, as we indicated in the previous section, some researchers have suggested – based largely on case study analysis – that deficits in ToM or perspective taking typically associated with ASD could, under adverse circumstances, heighten the risk of involvement in crime and contribute to negative interactions with police officers and (ultimately) within the court system. Here we examine (what we believe to be) the only published studies to date that have investigated an empirical relationship between ToM abilities and the possibility that ASD individuals may find themselves falling foul of the law. Notably, all of these studies have only been published very recently. Two of the studies focus on the relationship between ToM and the ability to detect suspicious behavior or attempts to deceive in others. In other words, they explore the possibility that ToM deficits could – by preventing an ASD individual from recognizing the true intentions of the individuals with whom they are interacting – contribute to their becoming involved almost unwittingly in criminal activity. The third study demonstrates how ToM predicts whether ASD individuals suspected of involvement in some criminal activity are able to allay the suspicions of police officers and extricate themselves from a problematic legal situation.

In the first study, Brewer et al. (2018) examined whether ToM predicted how quickly individuals detected there was something suspicious about the way an interaction was unfolding. The researchers examined whether individuals who are poor at taking the perspective of others were slow to recognize that they were being lured into a problematic interaction that may lead them to become enmeshed in some criminal activity. Using a laboratory research paradigm, typically developing participants listened to a number of audio vignettes and were asked to press a button as soon as they detected that something suspicious was going on (or decided that there was nothing untoward about the interaction). Some vignettes gradually unveiled subtle indicators of suspicious behavior by one of the characters, with

the sequence of events culminating in another character becoming unwittingly involved in a crime. Other vignettes had similar story lines but did not culminate in a crime. ToM was assessed with the Frith-Happé animations task (White et al. 2011) in which participants completed a number of trials on which they had to decide whether the “interactions” between a pair of animated geometric stimuli (i.e., triangles) were (i) random, (ii) purely physical, or (iii) mental (i.e., social in nature) with one triangle responding to the other triangle’s mental state. And, if participants deemed the interaction to be mental or social in nature (i.e., requiring ToM), they were asked to attribute feelings to the interacting stimuli.

The findings (and meta-analysis) of two experiments confirmed that, even after controlling for verbal IQ, participants who performed more poorly on the ToM measure were slower to respond to the subtle cues to suspicious behavior. Specifically, the slower detectors of suspicious behavior were less effective at (a) discriminating mental state (cf. non-mental state) interactions between the animated triangles, (b) identifying the specific nature of those mental state interactions, and (c) categorizing the feelings of the animations during the mental state interactions.

Some important caveats were highlighted by the authors. The study was correlational in nature, the relationships (while statistically significant) were not strong, and the sample comprised only typically developing participants (likely limiting the variance in ToM). And, of course, there is no guarantee that the observed slower detection of suspicious behavior associated with poorer ToM would be duplicated in real-life contexts or translated into involvement in criminal acts. Nevertheless, the findings are promising, especially as the strength of the relationships identified was likely constrained by (a) latency measures that inevitably will be “noisy” when based (as they were) on a very small number of trials and (b) limited variance on the ToM measures within a purely typically developing sample. Moreover, the research provides one example of an experimental paradigm that could be refined and used to explore questions that hitherto have evaded systematic empirical investigation.

In the second study, Williams et al. (2018) examined the relationship between ToM (or mindreading as they labeled it) and the ability to detect deception. Clearly an inability to detect attempts to deceive by an interaction partner could render an individual vulnerable to becoming involved in some criminal activity and incurring the wrath of the law. The deception detection paradigm involved participants viewing video clips of interviews with university students who either lied or told the truth about cheating in an experiment; the ToM measures were the triangles’ animations described above and the Reading the mind in the eyes task (Baron-Cohen et al. 2001a).

In their first experiment with typically developing participants, Williams et al. (2018) found no relationship between lie detection performance and ToM measures, but they did detect a significant negative relationship between lie detection performance and scores on the Autism-Spectrum Quotient (AQ; Baron-Cohen et al. 2001b), a measure of autistic traits. In their second experiment, they compared deception detection performance of an ASD sample with that of neurotypical controls. There were two main findings. First, in the combined sample, AQ performance was again negatively correlated with deception detection performance. Second, the ASD group performed significantly worse than controls on the deception detection task. Unfortunately, however, ToM (or mind-reading) was not measured in the second experiment, and thus the possibility that it may have played some mediating role in the sizable group differences reported cannot be assessed. The absence of a relationship between lie detection performance and ToM measures in their first experiment led Williams et al. to conclude that lie detection ability is not related to ToM. However, in line with the comments we made previously in relation to the Brewer et al. (2018) studies, this conclusion seems premature given the likely restricted variance on the ToM tasks when only neurotypical participants are assessed. Given the meaningful relationships detected between the degree of autistic characteristics and deception detection performance, and the sizable ASD-control group differences in deception detection, further exploration of the possible

nexus between ToM deficits and deception detection, particularly within an ASD cohort, certainly seems warranted if we are to appreciate fully the potential vulnerabilities of ASD individuals.

The third empirical study examined concerned another type of situation. Individuals who become involved in criminal activity – whether that involvement is unwitting or not – are obviously at heightened risk of finding themselves in very difficult interactions with the police. But, of course, some individuals may become innocent suspects in a criminal matter through circumstances beyond their control. For such individuals the nature of their interactions with police may determine whether they remain under suspicion, are charged with committing a crime, or are no longer suspected of involvement in the crime. Young and Brewer (2019) hypothesized that ToM (or perspective taking) deficits might affect whether ASD individuals are able to allay (misguided) suspicions that police officers may have about their involvement in a crime – before the police become absolutely convinced of their guilt. They argued that a person who is (mistakenly) suspected of being involved in some criminal activity needs to present information to the investigating officer that will demonstrate their innocence (e.g., providing an alibi that places themselves elsewhere at the time of the crime or suggesting witnesses who might corroborate their account of events).

Young and Brewer identified an array of factors that might contribute to how effectively a person could allay a police officer's suspicions and extricate themselves from a problematic situation (e.g., deductive reasoning capacity, sociability, effective relational processing that permitted coordinated episodic memory recall). One other potentially important factor is their ability to put themselves in the shoes of the police officer and appreciate their perspective on what had transpired. Then, guided by any insights gained from considering the officer's perspective, they would identify and present to the police any information that would demonstrate their innocence, thereby extricating themselves from a very difficult situation. Following this rationale, Young and Brewer (2019) examined two main questions. First,

did ASD adults perform more poorly than IQ-matched controls in providing critical information that would allay misplaced suspicions about their involvement in some crime? And, second, did perspective taking deficiencies play a role in any group differences observed in the individuals' ability to extricate themselves from a difficult situation?

The key perspective taking (i.e., theory of mind) measure was provided by the social items of the adult-theory of mind test (A-ToM; Brewer et al. 2017) in which participants view six videos of interpersonal interactions between actors and are asked to explain the social inferences (e.g., sarcasm, bluff, faux pas) underpinning the behavior of one actor toward the other. The extrication measure comprised five audio scenarios, with participants asked to assume the role of a person in the scenario who is suspected of committing a crime (refer Young and Brewer 2019). Participants listened to each scenario and were then asked to offer as much relevant information as they could in order to convince police they were not involved in the crime. There were four items of information in each scenario that, collectively, would clearly demonstrate the individual's innocence, with participants awarded one point for each item correctly reported.

Young and Brewer's (2019) findings were as predicted and the effect sizes robust. The ASD participants performed significantly worse than the controls on both the ToM and the extrication measures. And, after partialling out verbal IQ and performance on a standardized test of episodic memory, ToM task performance made a substantial contribution to the prediction of extrication task performance. These results parallel case study observations (e.g., Brewer and Young 2015) that ASD adults, wrongly suspected by police of criminal behavior, may not be particularly effective at allaying police suspicions and removing themselves from the focus of any ongoing investigations.

The evidential base provided by these three studies is clearly limited. Although it is not unusual to read that some characteristics of ASD individuals may render them vulnerable to being ensnared in criminal activity and experiencing

problematic interactions with police officers, empirical investigations of these issues are scarce. This almost certainly reflects, at least in part, an absence of well-developed research paradigms for exploring such issues. We have discussed the few studies of which we are aware that represent initial explorations of these issues. The results suggest that the ToM or perspective taking deficits that are more likely to characterize ASD than typically developing individuals may impede their ability to recognize suspicious behavior, to detect deception, and to make a case to police that they should not be a suspect for a crime in which they weren't involved. Clearly, these findings are preliminary. The results have not yet been replicated. In the first two studies examined, the effects were not strong. And methodologies that involve participants reacting to audio or video recordings are some distance from the real-life contexts the studies are seeking to simulate. That said, the findings to date highlight the likely connections between ToM deficits and the ability of ASD adults to respond adaptively in a complex social environment. Moreover, from an applied perspective, empirical demonstrations of how problematic interactions with the criminal justice system could surface for ASD individuals are crucial for illuminating potential biases in the operation of the justice system and developing an informed framework for the education of police and court officers.

Future Directions

Despite the abundance of comments in the literature about the characteristics of ASD that could, in some situations, render individuals vulnerable to becoming involved in crime and experiencing quite negative interactions with police and the courts, it is clear from the preceding analysis that empirical work in this domain has barely got off the ground. This of course means that the opportunities for future research are boundless. Consequently, in this section, we have chosen to highlight a few issues – not in any particular order – that we think are of high priority.

One important focus of future research should be on enhancing our understanding of ToM in

adult ASD individuals. Although implicating deficits in ToM may provide a parsimonious account of unusual or problematic social behavior, there is only limited empirical evidence regarding the nature and extent of those deficits and how they are reflected in ASD adults' behavior. There is some evidence of significant variability in ToM deficits across adults (Brewer et al. 2017), with some individuals differing little from typically developing controls and others being markedly impaired. But it is important to recognize that the breadth of behaviors sampled by ToM measures, such as the A-ToM used by Brewer et al. (2017), is quite limited, so little is known about whether the deficits are pervasive or, perhaps depending on the individual's social learning experience, manifested only in quite specific behavioral domains (cf. Brewer and Young 2015). Further, although poor ToM may account for social and communication difficulties, its link to stereotypical and ritualistic behaviors, sensory responses, and other core features of the disorder is unknown. Therefore, the inter-relationships between all these features of ASD that may lead to ASD individuals becoming involved in problematic interactions with the police, and shape their ability to negotiate such interactions effectively, remain unexplored.

Another focus should be to replicate and extend the preliminary findings we have described above. Extending this initial research will obviously require the development of novel research paradigms that ideally will more closely parallel the demands of real-life interactions. It would also be desirable for such research to capture other dimensions of police-citizen interactions in order to appreciate the extent to which ToM deficits constrain the ability of ASD individuals to interact effectively. For example, tendencies to acquiesce to other people's requests and to interpret statements in a completely literal manner are often mentioned as characteristics that could seriously disadvantage ASD individuals when interacting with the police or lawyers (e.g., Beardon et al. 2018). Likewise, being insensitive to facial expressions or vocal tone and volume of a police officer with whom they are interacting may contribute to a rapid deterioration in their

relationship. Yet, empirical work that helps to clarify the individual perspective taking characteristics and the potentially enabling situational variables required for such problems to emerge has not yet been conducted.

Similarly, there are numerous behaviors often seen in ASD individuals (e.g., gaze aversion, fidgeting, inappropriate affect, monologues) that if manifested in an interaction with a police officer (or in the courtroom) may lead to the individual being judged quite harshly (for a detailed discussion, see Brewer and Young 2018). However, empirical work that captures whether ASD individuals are sensitive to these judgments of their behavior, and then able to adjust their behavior accordingly – or are even motivated to do so – is non-existent.

Finally, we draw attention to an area previously highlighted by Brewer and Young (2018). It is well documented that the Internet offers opportunities for individuals to be deceived and exploited. Whether ASD individuals are particularly vulnerable when interacting in this environment is unknown. But survey data do indicate that many ASD individuals keenly maintain interests that are often pursued using the Internet, potentially exposing individuals with limited social networks and ToM deficits to interactions in chat rooms or other networking frameworks with individuals who may have nefarious intentions. And those with perspective taking deficits may be slow to see where such interactions are heading and unskilled at extricating themselves from any problematic situations before they end up violating the law. Systematic empirical research in this domain using imaginative new research methodologies certainly appears to be a potentially worthwhile direction.

The research directions we have outlined are obviously just a select few. The relevance of many or all of them has doubtless been raised in policy documents, canvassed in the informal observations of clinicians, and so on. However, we believe that persuading police, lawyers, and other justice system professionals of some of the barriers to ASD individuals receiving equitable treatment in the justice system will be enhanced significantly if, rather than relying on clinical

anecdotes, there exists a body of systematic and demonstrably reliable scientific evidence that clearly pinpoints the nexus between prevalent ASD characteristics and the likelihood of problematic justice system interactions. In sum, this seriously neglected research area offers enormous challenges to interested and skilled researchers, challenges that, if resolved, can help ASD individuals and increase the chances that they receive equitable treatment when interacting with justice system professionals.

See Also

- [First Responders and Autism](#)
- [Legal System](#)

References and Reading

- Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory of mind*. Boston: MIT Press/Bradford.
- Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001a). The “Reading the mind in the eyes” test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry*, 42, 241–251.
- Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001b). The autism spectrum quotient (AQ): Evidence from Asperger syndrome/high-functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders*, 31, 5–17.
- Beardon, L., Chown, N., & Cossburn, K. (2018). First responders and autism. In F. R. Volkmar (Ed.), *Encyclopedia of autism spectrum disorders* (pp. 1–9). Springer: New York.
- Brewer, N., & Young, R. L. (2015). *Crime and autism spectrum disorder: Myths and mechanisms*. London, UK: Jessica Kingsley. ISBN: 9781849054041.
- Brewer, N., & Young, R. L. (2018). Interactions of individuals with autism spectrum disorder with the criminal justice system: Influences on involvement and outcomes. In J. L. Johnson, G. S. Goodman, & P. C. Mundy (Eds.), *The Wiley handbook of autobiographical memory, autism spectrum disorder, and the law* (pp. 231–244). Newark: Wiley. ISBN: 978-1-119-15843-1.
- Brewer, N., Young, R. L., & Barnett, E. (2017). Measuring theory of mind in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47, 1927–1941.

- Brewer, N., Bay Wei Ying, A., Young, R. L., & Nah, Y.-H. (2018). Theory of mind and the detection of suspicious behavior. *Journal of Applied Research in Memory and Cognition*, 7, 123–131.
- Kassin, S. M., & Norwick, R. J. (2004). Why people waive their Miranda rights: The power of innocence. *Law and Human Behavior*, 28, 211–221.
- Van de Cruys, S., Evers, K., Van der Hallen, R., Van Eylen, L., et al. (2014). Precise minds in uncertain worlds: Predictive coding in autism. *Psychological Review*, 121, 649–675.
- White, S. J., Coniston, D., Rogers, R., & Frith, U. (2011). Developing the Frith-Happé animations: A quick and objective test of theory of mind for adults with autism. *Autism Research*, 4, 149–154.
- Williams, D. M., Nicholson, T., Grainger, C., Lind, S. E., & Carruthers, P. (2018). Can you spot a liar? Deception, mindreading, and the case of autism spectrum disorder. *Autism Research*, 11, 1129–1137.
- Young, R. L., & Brewer, N. (2019, online first). Perspective taking deficits, autism spectrum disorder, and allaying police officers' suspicions about criminal involvement. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03968-4>.

Politics of Autism

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Synonyms

Autism policy; Public understanding of autism

Definition

Autism viewed as an issue about which an individual can have an opinion and for which the government can program. The politics of autism began with the term's first use by Eugen Bleuler in 1910, developed as knowledge about autism grew, and continue to change.

Scientific research informs autism policy, but so do less trustworthy sources such as personal accounts, lived experience, and media coverage. Theories compete for finite funding and interest, creating conflict between groups that have the same goal: to improve the lives of people affected

by autism. Each new controversy regarding autism increases its visibility. Unfortunately, controversies also breed conflict, which fragments understanding and policy.

Multiple key aspects of autism have been in dispute all along: prevalence, theories of causation, interventions, and outcomes.

As prevalence numbers have increased, so has the perceived importance of autism. What began as a rare condition has since been referred to as an epidemic, as the theory that autism cases were on the rise peaked in the 1990s. Leo Kanner's 1943 definition of autism was updated with the publication of the DSM in 1952, followed by multiple revisions progressing to the most recent DSM-5. Diagnostic changes have played a part in fluctuating prevalence figures and have also complicated professional and public understanding. Without agreement on what autism is, we can't say for certain how common it is.

Early causal theories such as the refrigerator mother theory in the 1960s contributed to a stigma associated with autism that persisted for decades. The parental experience of autism influenced its politics, as parents began by being blamed, then became central to intervention and advocacy. All along, parents faced public perceptions of their children's condition and the stress of living with it. As each generation of autistic children matured, they had more opportunities to advocate for themselves. The development of the Internet accelerated the spread of information, misinformation, and support (Pitney 2015).

Causal theories and related treatments that were later debunked attracted funding and public attention. In the case of the late 1990s vaccine theory of autism, the process of debunking the theory also drained funding for a prolonged time period (Offit 2008). More recently evidence of genetic causes drew research funding but drew it away from those who were already living with autism.

As certain interventions emerged as more effective than others, funding and interest followed. Here was another central controversy. Does autism intervention aim to cure autism or to help those with autism accommodate themselves to a mostly non-autistic world? And what fields would provide interventions? Medicine? Early

Intervention? Education? Social Services? Did we set people with autism apart to treat them, or did we include them in mainstream settings? In the 1990s, applied behavior analysis drove demand for costly early intervention programs, shifting the focus away from the lifetime approach advanced by state-funded programs such as Division TEACCH (Mesibov et al. 2004).

Finally, desired outcomes are still in question. One person's perceived success could resemble another's perceived failure. For some, the ability to live independently is enough. But what about those for whom such an outcome is out of reach, no matter how much intervention they receive? Conversely, have those who completed college, found meaningful work, and raised families been cured? The focus on a cure peaked as applied behavior analysis offered a 50 % cure rate at the height of its popularity (Lovaas 1987). At the same time, though, adults with autism emphasized the positive aspects of their differences and questioned the need for a cure. More recent thinking has turned to the most likely positive outcomes and how to use resources to achieve these (Siegel 2018).

Just as a focus on prevention, treatment, or palliative care varies in popularity during a person's lifespan, autism has meant different things to different people over the generations since the term was first used.

See Also

- Culture and Autism
- History of Autism as a Diagnostic Concept
- Public Perception of Autism Treatments: Science Versus Pseudoscience in the Age of Mass Media

References and Reading

- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- Mesibov, G. B., Shea, V., & Schopler, E. (2004). *The Teacch approach to autism spectrum disorders*. New York: Springer.

Offit, P. A. (2008). *Autism's false prophets*. New York: Columbia University Press.

Pitney, J. J. (2015). *The politics of autism: Navigating the contested spectrum*. Lanham: Rowman and Littlefield.

Siegel, B. (2018). *The politics of autism*. New York: Oxford University Press.

Politics of Autism Transition Services

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Definition

A political analysis of why the transition and adult service systems are such problematic parts of autism policy, how transition has been addressed by stakeholders with differential success, and what the implications are for politics and policy moving forward.

Historical Background

Autism Transition as an Escalating Policy Crisis

Not long ago, autism was a condition unknown to the American public. Those who had it were typically kept at home or thrown into large institutions with other special needs groups society had given up on. Now, autism is known to be the fastest-growing serious developmental disability in the United States (Ardhanareeswaran and Volkmar 2015) – affecting an estimated 1 in 54 Americans (Centers for Disease Control 2020). With increasingly high rates of diagnosis and the boundaries of the autism world continuing to push out, the condition has officially emerged as a major issue in American public policy.

Although much of the attention of researchers has been on autistic children, many of the biggest policy challenges revolve around the transition services system – meant to support young adults with autism in their transition to adulthood.

For any young adult, transitioning into adulthood – by attending college or finding a job – comes with challenges and a period of adjustment. But for people with the social, emotional, and communication challenges that often coincide with autism, this period of transition becomes significantly more difficult to traverse, especially given the continued growth in white-collar and service sector jobs that require exactly the interpersonal skills autistic people often struggle with (Pitney 2015). With over half a million Americans on the spectrum legally entering adulthood and aging out of school-based services over the next decade (Harwood and Novotny 2017), this problem will only gain in significance.

More than half of autistic young adults remain unemployed or unenrolled in higher education for their first 2 years out of high school, which is the highest rate among disability groups (Autism Speaks 2020). This is despite the fact that employment is the primary transition goal of students with special needs exiting high school (Roux et al. 2015). Mass unemployment is taking a toll on autistic adults, who are losing out on the benefits associated with job activities, including greater independence, the reduction of autism symptoms, and increased daily living skills (Autism Speaks 2020). According to Autism Speaks, the national cost of supporting people with autism reached \$268 billion in 2015 and is expected to rise to \$461 billion by 2025 without more effective support across the lifespan (Autism Speaks 2020).

The diversity of the autism experience makes transition particularly challenging to tackle from a policy perspective. What works for one person in terms of transition likely won't work for many others, which makes individualization of programming and services important. This often requires significant investment in staff and training, which comes at a high cost.

A common feature of the transition experience for autistic individuals and their families is confronting the challenge of the services cliff. "Falling off the cliff" refers to the shift from receiving school district entitlement services to traversing a dizzying bureaucracy of non-entitlement programs once an autistic individual

turns age 22. Its presence largely exists because the United States lacks a well-organized infrastructure to implement necessary services once people with autism leave the education system. "After the cliff, finding services is a maze with high walls and a hidden entrance. You don't know where to start" (Pitney 2020).

Multi-year waitlists for transition and adult programs are not uncommon in many states after age 21, largely due to limits in state and federal funding. Families are often lost trying to navigate a complex system of state agencies and other service providers that help with job placement, vocational training, and other employment-related programming. A significant burden is placed on parents, as fear mounts regarding who will take care of their child once they die. Alleviating the harsh impact of the services cliff is crucial to facilitating greater independence among the autistic population in a way that elevates quality of life and lessens the financial strain on state and federal budgets (Loomis 2014).

Political Science: A Newly Studied Window into Understanding Transition

The way in which the transition issue is addressed by government provides a window into the politics of social and education policy for historically stigmatized groups, as well as the way policymaking plays out in a decentralized federal system. Moreover, politics provides a lens with which to better understand the tensions that challenge the transition services system, helping to answer the core question of why it is such a problematic part of autism policy.

Only a handful of political scientists have closely studied autism as a policy issue. This entry builds on Professor John Pitney's scholarship articulated in his book *The Politics of Autism: Navigating the Contested Spectrum*, which serves as a primary framework for understanding how politics operates in this space.

Federalism – or the distribution of political power across federal, state, and local government units – shapes the environment in which autism transition politics play out. It provides multiple venues at which to obtain services but also adds to the complexity of navigating sometimes-dizzying

bureaucracies to find them (Pitney 2015). Because autism transition services are intertwined within this decentralized federalist political structure, there's not one central place where policy and services are created and implemented. Instead, "various providers offer various services, with various levels of support from the government, which largely depends on where one lives" (Pitney 2015, p. 5). This makes coordination among entities within the autism transition system an ongoing challenge.

Utilizing federalism as a lens helps illustrate how the transition system is constructed and, moreover, portrays the tensions, money clashes, implementation battles, and mobilization efforts that continue to shape and challenge its structure, across states and localities nationwide.

A Brief Political History of Autism as a Policy Issue

The historical background of how autism became an increasingly visible political issue throughout the twentieth century helps portray the larger forces – including misunderstanding, prejudice, "legislative oversight," (defined here as the unintentional failure to accommodate a group when creating policy) and policy fragmentation – that have shaped the current transition services system.

Nowadays, autism is understood as a national issue affecting millions of Americans, impacted by the actions of the government, school system, and numerous welfare policies. The boundaries of the autism world continue to push outward as more people receive an autism label and see themselves as part of the issue (Pitney 2015). But this was not always the case. For an issue to enter the public agenda, it must first be seen as a problem, which often relies on a shift in awareness and understanding (Pitney 2015). In the case of autism, this did not happen until the late twentieth century. "Its emergence hinged on its identification as a distinct disorder, on public knowledge of its existence, and on changing beliefs of what causes it and what to do about it" (Pitney 2015, p. 16).

Misunderstanding and misinformation characterized what many early researchers and clinicians

said about autism, often describing it as an affliction of the young caused by "refrigerator parents," who lacked warmth in raising their children. Casting blame on parents suggested there was little the government could do to help, which not only kept autism off the political agenda but also "immobilized the child's strongest ally" (Rimland 1970, p. 20). Additionally, the historic inability to recognize autism as a lifelong condition had reverberating effects on why today's service system is built more for autistic children than adults.

As scientific misunderstandings slowly began to clear, and diagnostic criteria became more refined, various political forces were at work paving the way for autism's emergence on the agenda. The Civil Rights Movement of the 1950s and 1960s helped make way for the Disability Rights Movement that would follow, which started gaining traction after the election of John F. Kennedy – whose sister lived in an institution for people with intellectual disabilities (Pitney 2015). The years that followed were characterized by the passage of key pieces of civil rights legislation that banned discrimination or exclusion based on disability, including the 1973 Rehabilitation Act, 1975 Education for All Handicapped Children Act, and 1990 Americans with Disabilities Act.

These pieces of legislation helped frame autism as a rights-based issue, making lawsuits and implementation battles common, particularly between parents and school districts in relation to special education. It is crucial to note that many of these foundational laws, still shaping the government's response to autism and other conditions today, were ambiguous and typically not written with autistic people in mind – because they were created before autism had a more prominent place on the political agenda (Pitney 2015). These weren't deliberate decisions, but they created a need to infer the standards conveyed by the laws without specific reference to autism. The challenge is that services required for people with autism are typically far more complex and sophisticated than these laws recognized (Pitney 2020). This historic "legislative oversight" helps explain why such a massive burden is placed on parents when seeking services for autistic children,

whether related to transition or other facets of life (Pitney 2020).

The tide began to change in the 1990s, “when legislative, scientific, and social trends converged to make autism an increasingly prominent political issue” (Pitney 2015, p. 26). Congress reauthorized the Education for All Handicapped Children Act, and this time autism was listed as a distinct category for special education. This new law, renamed the Individuals with Disabilities Education Act (IDEA), would have major consequences on educating people with autism, including within the transition space.

Now that the autistic population was clearly included under IDEA and formally entitled to a free, appropriate public education and related services from ages 3–21, schools had to begin identifying people with autism who qualified for services. As a result, the diagnostic criteria became much broader, and the number of people receiving services for autism spiked rapidly. From 1995 to 2018, the number of autistic children aged 3–21 receiving IDEA services increased from 28,000 to 710,000, with ASD now accounting for about 10% of the total population served under the law (National Center for Education Statistics 2009, 2019). This drastic growth in identification and observed prevalence drove public awareness of autism and enabled more families to push for services for their children (Pitney 2015). But it also increased financial pressure on school districts and, later, transition service providers, who now had to provide more programs for people with ASD.

For most of American history, states and localities dominated education policy. Starting in the 1950s, however, the federal government began to assume a bigger role. The Education for All Handicapped Children Act and IDEA – which increased federal funding for special education and required greater inclusion of people with disabilities in K-12 schooling – were a product of this growing federal involvement (Pitney 2015). But when Congress passed these landmark bills, national leaders failed to anticipate how much educating a child with special needs would cost. Nor did they foresee how many young Americans would qualify for such support. Under the 1975

Education for All Handicapped Children Act, the federal government was authorized to cover “40% of each state’s ‘excess cost’ of educating children with disabilities” (Pitney 2015, p. 65). Yet the federal government has consistently fallen far short of this federal financing standard. As a result, states and school districts have struggled to meet federal standards. This imbalance between federal requirements and federal support has contributed to the challenges many autistic young adults face when trying to obtain sufficient transition support.

Current Knowledge

Federalism and the Politics of Autism Transition

The historical background of autism as a policy issue begins to illustrate the configuration of political forces continuing to challenge and shape the transition system today. Ultimately, political and policy fragmentation, “legislative oversight,” and a culture of low expectations for autistic adults combine to create an unstable and unnecessarily complex transition services infrastructure riddled with implementation battles, clashes of money and values, and confusion. As a result, people with autism face a service system – often consisting of poorly matched parts originally designed for other populations – that is extremely complex to navigate, places undue burden on students and parents, and varies vastly throughout the country.

Tensions and clashes are common in autism transition politics because there are many stakeholders motivated by different priorities and limited by different resources and pressures. Autistic people are the ultimate stakeholder, trying to obtain the services and opportunities necessary to support their unique abilities and help them live a fulfilling life. Autistic voices have often been absent from public conversations on this topic, largely due to prejudice and stigma.

Parents are another key stakeholder in autism transition. An immense burden is placed on them to enforce their children’s rights and find the services and support needed. Parents often know

the unique needs and abilities of their children best and try to demand more than simply the “free and appropriate education” IDEA guarantees. Some parents are more equipped to take on this advocacy role given factors such as wealth and education.

As the primary provider of services through age 21, school districts are another key stakeholder in the system – organizing the creation and implementation of the individualized education and transition plans (IEP and ITP). The major challenge is that autism can be an expensive label at a time when states and local districts are almost universally under budget pressure (Pitney 2015). Thus, when attempting to provide the transition services necessary to best prepare students for adulthood, school districts are often at odds between their federal obligation and surviving financially.

Particularly once school district IDEA entitlements expire, out-of-school service providers offer employment support including vocational training, job coaching, and day habilitation programs. There is no specific government agency designed to exclusively meet the needs of young adults with autism transitioning into employment (Gerhardt et al. 2014). As a result, adolescents transitioning into the vocational world have to choose from a variety of private and public programs designed for people with many different developmental disabilities.

Ultimately, the state and federal governments are the macro-level stakeholders within the larger system. State legislatures appropriate budgets and funding to state agencies and school districts, which are used for programs that support autistic young adults. Meanwhile, federal funding has a significant impact on state reimbursements for special education, services such as Medicaid, and federal autism research. In addition, state and federal governments set up the policy structures under which the rest of the system operates, as illustrated by IDEA’s influence on special education nationwide.

When these stakeholders, with varying priorities, pressures, and resource limitations, come into contact with one another in the transition services system, tensions, implementation battles, and

clashes of money and values play out. A primary example of this occurs between families and schools, particularly during meetings involving the creation, and yearly review, of IEPs and ITPs.

“IEP meetings often represent a clash of conflicting outlooks” between parents who want the best outcome for their child and school districts that are often dutiful but naturally see the child as one of many (Pitney 2015, p. 70). These meetings are often contentious and function as local battlegrounds for the implementation of IDEA on a case-by-case basis. Nearly all parents are at an IEP disadvantage compared to school districts (Pitney 2015). Whereas schools have the advantage of being repeat players in the system, generally having handled many cases in the bureaucracy and developed professional expertise on the subject, parents only have the experience of their own child (Pitney 2015) – creating an intimidating environment with a severe power imbalance.

It’s important to remember that local implementation battles between parents and schools are impacted by the money clashes between stakeholders higher up in the system. Within education services, the “theater of conflict is all about inter-governmental relations. States and localities are primarily responsible for education, but federal statutes set the rules of engagement” (Pitney 2015, p. 14). Complying with federal IDEA law comes at a large expense to states and school districts already under financial pressure – particularly when the federal government doesn’t fulfill its original promise to shoulder 40% of states’ “excess costs” to educate students with disabilities.

Variation Across the System

The US federalist system creates many partially autonomous venues for policy action, which encourages significant diversity in policy structures and outcomes (Baumgartner and Jones 1993). According to Professor John Pitney, there is always going to be variation in a federalist system, “but perhaps with autism, the variation is more extreme” (Pitney 2020). When it comes to transition services, “what you get from the government depends on where you live” (Pitney

2015, p. 64). Some schools have greater resources and are more likely to provide high levels of service to people with autism. Likewise, out-of-school services vary significantly state to state.

Subnational jurisdictions can be used as testing grounds for new ideas and policies, later to be mimicked if successful or abandoned if unworkable (Baumgartner and Jones 1993). For example, California's approach to providing out-of-school services for people with developmental disabilities bears recognition. Its structure relies upon 21 private, nonprofit regional centers contracted by California's Department of Developmental Services, which serve as middle men providing coordination between local service providers and clients and families who qualify for support – while offering some programming of their own (CA Department of Developmental Services 2020). Of particular note, the system is available to those who qualify from birth to death, providing a level of service continuity uncommon in many states, where transitioning between several service systems across the lifespan is common (Sullivan 2020).

Confronting Challenges and Cases of Success

The following case studies illustrate how different programs have responded to challenges in the system, finding innovative solutions to maximize success. The Cedarhurst School's Passage Program and the Uniquely Able Project aim to cultivate greater independence among autistic students while trying to provide the skills, experience, and training necessary to help them succeed in competitive employment. These are private, nonprofit programs striving to fill the gaps in the current transition system. Passage is a school-based service in Connecticut, while the Uniquely Able Project is a pre-employment program offered after high school in California.

The Cedarhurst School's Passage Program (Hamden, CT)

The Cedarhurst School is a private therapeutic junior and senior high school operated by Yale University, offering a structured, supportive learning environment to students identified as autistic,

emotionally disturbed, or having other health impairments, who are outplaced from school districts throughout Connecticut (Yale School of Medicine 2019). Cedarhurst's Passage Program specifically serves about a dozen special education students ages 18–21, creating an individualized pre-employment and independent living program attempting to support students in their transition to independence (Yale School of Medicine 2019). Student tuition is paid by the sending school district – with partial reimbursement from the state of Connecticut – and is estimated to be about \$75,000/year including school and transportation (Donovan 2020).

The transition-focused Passage Program, founded 10 years ago, was born out of a need among Cedarhurst students who were not ready to enter the real world, particularly when placed within a system that provided insufficient transition support after high school (Donovan and Clemens 2020). The program invests in on-site, community-based experiences, including daily part-time job placements at local businesses, and courses at nearby colleges including Southern Connecticut State University and Albertus Magnus College (Kannatt et al. 2020). These activities are supplemented by life skills and vocational classes at Cedarhurst, assistance in connecting students with state agencies that can provide additional transition services during and after Passage, and a weekly social skills group which develops peer-to-peer relationships between Passage students and a group of student volunteers at Yale (Kannatt et al. 2020).

Cedarhurst's expensive tuition and affiliation with Yale help enable the school's low student-to-faculty ratio and high standard of care, but unfortunately this makes the program quite difficult to replicate in public districts, which have fewer resources and struggle to provide adequate special education services. That being said, there are important lessons about transition to learn from Passage, including the program's:

- Individualization-heavy, community-centered model
- Daily job placements
- Private sector partnerships

- Efforts to coordinate services with local colleges and state agencies
- Peer-led social skills groups

The Uniquely Abled Project (Los Angeles, CA)

The Uniquely Abled Project (UAP) provides post-high school vocational opportunities to young adults with ASD, matching the unique skills of autistic individuals to high-demand career jobs, in a way that is scalable and free to parents. Founder Ivan Rosenberg, a management consultant and parent of two children on the spectrum, saw himself as a “business person dealing with a social problem” – confronting two major challenges he believed could become solutions for each other: (1) the soaring unemployment rate for millions of Americans with disabilities and (2) the fact that many businesses lack access to a skilled workforce (Rosenberg 2020).

Rosenberg identified several barriers related to the unemployment problem:

- Use of the word “disabled”
- Lack of understanding among employers and job developers regarding the potential of people with autism
- Insufficient affordable, vocational education programs for people with ASD
- Scarce efforts to match unique abilities to the specific needs of jobs (Rosenberg 2020)

UAP works within existing education and employment systems to create a structure of collaboration between different stakeholders – including autistic employees, educators at community colleges, job developers at state agencies, autism specialists, and private employers – under the banner of a single program. Its 16-week academy comprehensively trains, places, and provides ongoing support to individuals with ASD by (1) matching diagnoses to jobs, (2) offering vocational and soft skills training through community colleges, (3) teaching instructors how to train autistic students, (4) educating job developers to find specific manufacturing jobs students are being trained for, (5) coaching employers on how to work with autistic employees, and (6) providing post-hire support to academy students (Rosenberg 2020).

UAP provides consulting and facilitates collaboration to get programs started in different locales. The academy’s first major job match has been between people with high-functioning autism, who are often good at repetitive work with clear instructions, and entry-level computerized machine operator jobs in the manufacturing industry. To date, it has been quite successful, with an estimated 69 machine operator job placements out of 70 graduates since August 2016 (Rosenberg 2020). Because UAP works within the existing community college and employment agency systems, the cost of the program is low – about \$3,000–5,000 per student, which is subsidized by regional centers, state agencies, and banks (Rosenberg 2020). UAP plans to expand its approach and business model to assist more challenged autistic individuals while diversifying to other industries.

Several lessons can be learned from UAP’s:

- Comprehensive, collaborative training model within existing systems
- Identification of high-demand jobs that match with unique abilities of ASD individuals
- Desire to empower abilities of people with ASD within a culture of high expectations
- High placement rate, low cost, and potential for scalability

Relative success stories can often be attributed to resource access and money; however, innovative ideas, cross-sector collaboration, effective employer outreach, and a culture of high expectations also play a significant role. Although the autistic young adults participating in these two programs are predominantly “high-functioning” on the spectrum, several lessons and themes regarding the broader transition services system can be distilled. It is critical to generalize best practices from successful organizations to the larger autistic population – otherwise, only a select few will benefit.

Shaping the System from the Ground Up: Mobilization Among Parents

Parents are one of the stakeholders closest to the transition issue and most frustrated by its

challenges. However, the service system is set up in a way in which it can be difficult for them to effectively mobilize politically. Autism is a time-consuming and exhausting condition for both parents and individuals directly affected, which means there is often little energy for political advocacy and lobbying (Pitney 2015). Moreover, it can be hard to mobilize within schools because the entire nature of the IEP creation and implementation process is individual and private. But these factors haven't stopped many parents from advocating and creating service programs for this population, particularly with the rise of new electronic platforms like social media and mass mailing lists. The past 10–15 years has seen an increase in parent-initiated services that help fellow parents navigate the complex world of autism and create opportunities for individuals in need of vocational training or jobs. Additionally, many parents have leveraged their knowledge to become paid advocates, spawning a new employment sector providing a less expensive alternative to lawyers.

Future Directions

Policy Recommendations: Enhancing Autism Transition for a Brighter Future

1. *Further invest in quality of life research, and study transition program and employment outcomes, in order to better understand where major service gaps lie – and what programs actually work.*

Much of the confusion and uncertainty characterizing autism transition is the result of lacking adult outcome research and unanswered questions about the long-term impacts of programs, services, and other interventions. A 2012 review of nearly 150 articles on autism intervention research found less than 2% of participants over age 20, while another study assessing papers on adults with autism published from 2000 to 2010 found only 23 out of 11,000 studies directly addressing intervention services (Howlin 2014). Without this research it is difficult to measure the performance and enhance the quality of transition

services most effective at improving the lives of autistic people while creating policies that help to facilitate the creation thereof. While conducting long-term outcome and transition research, it is critical to include the input of autistic individuals.

2. *Require the federal government to fulfill its original obligation to cover 40% of a state's "excess costs" to educate children with disabilities through IDEA.*

Many of the money clashes and implementation battles challenging the transition services system – particularly within the education sphere – result from a lack of federal financial commitment to the Individuals with Disabilities Education Act. When the original legislation was signed in 1975, the drafters didn't anticipate the extent of the cost that would be placed on school districts or the complex services that would be needed to support students with more "invisible disabilities" like autism. Now that the government is aware of these challenges, it is time to reassert its commitment. A significant portion of this increased federal special education spending should be allocated to transition services and accompanied by more specific and ambitious guidelines related to the creation of IEPs and ITPs.

3. *Teach autistic individuals generalizable, community-based vocational and social skills – corresponding to their unique abilities and high demand jobs – both while in the education system and afterward.*

The education system and out-of-school service providers can learn lessons from case studies on the Cedarhurst School and the Uniquely Abled Project. Cedarhurst's Passage Program recognizes the need to find individualized, community-based job placements for students with special needs based on their interests and abilities – providing them with real-life skills that can be generalized to jobs after school ends. Meanwhile, UAP illustrates the importance of continuing to develop these vocational skills – within a culture of high expectations – once IDEA entitlements run out. UAP embodies the success that can

be associated with retro-engineering training programs to match the unique abilities of autistic individuals with the specific needs of high-turnover jobs. These programs recognize the need to accompany vocational training with the strengthening of social skills, in order to help overcome major barriers related to an autistic person's ability to be hired.

4. *Enhance collaboration between school districts, state and private programs, and employers in a way that reduces the post-IDEA services cliff, helps families navigate the system, and holds autistic adults to a higher set of expectations.*

A lack of collaboration between the various entities with a stake in the autism transition process heightens the presence of the services cliff and contributes to the challenges families face in navigating the complexities of the transition and adult service systems. Much of the confusion and clashes of values associated with insufficient collaboration are a result of political and policy fragmentation and stigma toward autistic adults. While the education system provides an infrastructure to support autistic individuals until age 21, no such logical system exists afterward. Ultimately, it may be beneficial for states to invest in autism-specific coordinating committees, aimed at helping to build partnerships between the business community, school districts, and state and private programs. Responsibilities could include the development of comprehensive lists of statewide programs available for people with autism and the creation of employer training modules to enhance autism awareness in the business community.

Transitioning to adulthood is not easy for anyone. It involves new, often daunting responsibilities, difficult decisions, and changes in environments. It is particularly challenging for the large group of autistic young adults aging out of the education system, facing a dizzying transition services system that is insufficient in supporting their potential and capitalizing on their unique abilities. The unexpected circumstances presented by the COVID-19 pandemic

pose particularly intense challenges for many on the spectrum, who do not deal well with a lack of structure or widespread uncertainty. With rising unemployment and looming budget cuts, there are legitimate concerns about what the future of an already-neglected autism transition service system will look like. For these reasons, it is particularly important to continue studying this problematic facet of autism politics and policy, in order to better understand how it works, why it is challenged, and how it can be improved.

See Also

- [Politics of Autism](#)
- [School to Work Transition Process](#)
- [Interventions to Support Transition to Adulthood for Individuals with Autism Spectrum Disorder](#)
- [US Social Policy and Autism Spectrum Disorders](#)

References and Reading

- Ardhanareeswaran, K., & Volkmar, F. (2015). Introduction. Focus: Autism spectrum disorders. *The Yale Journal of Biology and Medicine*, 88(1), 3–4.
- Autism Speaks. (2020). Autism statistics and facts. Retrieved 9 May 2020 from <https://www.autismspeaks.org/autism-statistics>
- Baumgartner, F., & Jones, B. (1993). *Agendas and instability in American politics*. Chicago: The University of Chicago Press.
- CA Department of Developmental Services. (2020). Regional centers. Retrieved 9 May 2020 from <https://www.dds.ca.gov/rc/>
- Centers for Disease Control and Prevention. (2020). Data & statistics on autism spectrum disorders. Retrieved 9 May 2020 from <https://www.cdc.gov/ncbddd/autism/data.html>
- Donovan, B. (2020, April 17). Director of the Cedarhurst School. *Online Correspondence*.
- Donovan, B., & Clemens, K. (2020, March 6). Director, associate director of the Cedarhurst School. *In-person Interview*.
- Gerhardt, P. F., Cicero, F., & Mayville, E. (2014). Employment and related services for adults with autism spectrum disorders. In F. R. Volkmar, B. Reichow, & J. McPartland (Eds.), *Adolescents and adults with autism spectrum disorders*. New York: Springer.

- Harwood, R., & Novotny, T. (2017, April 6). Youth with autism spectrum disorder transitioning to adulthood. U.S. Department of Health and Human Services. Retrieved 9 May 2020 from <https://www.hhs.gov/blog/2017/04/06/youth-autism-spectrum-disorder-transitioning-adulthood.html>
- Howlin, P. (2014). Outcomes in adults with autism spectrum disorders. In F. R. Volkmar, R. Paul, S. J. Rogers, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders*. Hoboken: Wiley.
- Kannatt, E., Bouldin, M., & Broatch, M. (March 25, 2020). Passage program teachers and employment specialists at the Cedarhurst School. *Video Interview*.
- Loomis, J. W. (2014). Supporting adult independence in the community for individuals with high-functioning autism spectrum disorders. In F. R. Volkmar, R. Paul, S. J. Rogers, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders*. Baltimore: Brookes.
- National Center for Education Statistics. (2009, 2019). *Digest of education statistics: 2018*. U.S. Department of Education. 2009: https://nces.ed.gov/programs/digest/d09/tables/dt09_050.asp, 2019: https://nces.ed.gov/programs/digest/d19/tables/dt19_204.30.asp.
- Pitney, J. (2015). *The politics of autism-navigating the contested spectrum*. Lanham: Rowman & Littlefield.
- Pitney, J. (2020, March 9). Roy P. Crocker professor of politics at Claremont McKenna College. *In-person Interview*.
- Rimland, B. (1970). In Pitney, J. (2015). *The politics of autism-navigating the contested spectrum*. Rowman & Littlefield. (p. 20).
- Rosenberg, I. (2020, March 19). President of the uniquely abled project. *Phone Interview*.
- Roux, A., Shattuck, P., Rast, J., Rava, J., & Anderson, K. (2015). *National autism indicators report: Transition into young adulthood*. Philadelphia: Life Course Outcomes Research Program/A.J. Drexel Autism Institute/Drexel University.
- Sullivan, M. (2020, March 19). Director, Lanterman Regional Center in Los Angeles, CA. *Phone Interview*.
- Yale School of Medicine. (2019). The passage program at Cedarhurst School. Retrieved 9 May 2020 from <https://medicine.yale.edu/psychiatry/cedarhurst/passage/>

Pondimin

- [Fenfluramine](#)

Population Norms

- [Normative Data](#)

Portugal and Autism

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Historical Background

At a time when Portuguese psychiatry was strongly dominated by the psychoanalytic school, parents' associations played a decisive role in the struggle for the implementation of clinical and pedagogical approaches to autism in Portugal.

As a matter of fact, it can be said that the popularization of autism recognition in Portugal was largely due to the commitment of parents.

In 1971, the first autism-specific institution, the Portuguese Association for the Protection of Autistic Children (APPCA), was founded in Lisbon, by a group of parents who did not find educational responses for their children with autism. This group was led by the physician and father, Dr. José Carlos de Almeida Gonçalves. APPCA's main objective was to build up a school where the specific needs of their children with autism could be met. The educational structure and model was created with the support from therapists of the British National Society for Autistic Children (NSAC), now the National Society for Autism (NSA), with whom the founders of APPCA had close contact since the beginning of the project. In 1972 the first clinical and therapeutic center for autism spectrum disorders was created by APPCA, in Lisbon. In 1984, the APPCA created a delegation in the north of Portugal, in Vila Nova de Gaia, where a second center was later to be opened. As a result of this process, and considering that the APPCA started to support not only children but adolescents and adults as well, the name was turned to

Portuguese Association for the Protection of Autistic Persons (APPDA), and the structure became decentralized: a national association with delegations in Lisbon and the North. Other delegations were later to be established in the center and south of the country and in Madeira and Azores islands. The APPDA is a member of the Autism-Europe International Association since 1988. The commitment of APPDA's President, Isabel Cottinelli Telmo, was pivotal for its development during that period. She was Vice-President of Autism-Europe from 1990 to 2008.

These associations were initially geared toward supporting people with very little autonomy and very low social skills. In 2003, the Portuguese Association of Asperger Syndrome (APSA) was created to try and meet the needs of people diagnosed with autism spectrum disorder (ASD) with greater functionality.

Some of these associations in the north, center, and south of the country have now special-needs schools, occupational centers, medical and psychology consultations, training centers, and residential support.

In 2002, the APPDA split up into as many autonomous associations as the existing regional delegations, under the common denomination of APPDA – Portuguese Association for Developmental Disorders and Autism. Together with a few other associations devoted to autism, these formed the Portuguese Autism Federation (FPDA), based in Lisbon.

Within the scope of the National Health Service, the diagnosis and intervention in Autism were carried out by the child and adolescent psychiatry and the neuropsychiatric and developmental pediatrics departments located in the main central hospitals. The first clinical and psychological interventions were also based in these same departments. In the late 1990s and early 2000s, with the pioneering drive of a few department directors, the creation of specific child development centers in the main central hospitals (in Lisbon, Porto, Coimbra, and Almada) was assured. These are centers within the National Health Service that provide services for prevention, diagnosis, and treatment of developmental disorders.

In 1999/2000, the first nationwide epidemiological study of autism was carried out on the population of Portuguese school children aged 6–9 years old (Oliveira et al. 2007).

Legal Issues, Mandates for Service

The specificity of autism spectrum disorders (ASD) was recognized by the Law decree 3/2008, which established the creation of “structured teaching units” which adapted specific methodologies and organizational practices to children and adolescents with autism. These units were to be created in public schools according to a policy of inclusive education.

Decree 21/2008 came afterward, recognizing the importance of special education schools for children with very little autonomy and severe behavioral problems. Referral to these later schools follows a case-by-case evaluation process.

With regard to the diagnosis and referral of children with alleged ASD, it is usually done by the pediatrician or the general practitioner. The initiative may also come from parents, teachers, or other therapists who attend the child. The referral is made to specialized services (Child and Adolescent Psychiatry, Neuropsychiatric, Developmental Pediatrics) or to the child development centers. These services are available both in the National Health Service and in private clinics.

Eligibility for support is achieved through evaluation by a Social Security Medical Board. The levels of support made available vary according to the degree of disability recognized. The level of support can vary throughout life, according to new assessments.

Overview of Current Treatments and Centers

Preschool intervention is ensured through National Health Service multidisciplinary teams (“early intervention” teams) linked to National Health Service centers or in private clinical centers specializing in intervention in developmental disorders. Some of these are nonprofit institutions.

During school age (6–7 years old), children with autism are mostly integrated in regular education schools. Whenever possible these children are referred to the so-called Structured Teaching Units (STU). These are special education units, based in regular schools, that implement teaching methodologies adapted to children with ASD, namely, the TEACCH (Treatment and Education of Autistic and Related Communication Handicapped Children) methodology. Its regulation was made by Law decree 3/2008. Pursuant to Article 30 of the Law decree 3/2008, partnerships were established with specialized resource centers, namely, to carry out specific programs, improve education strategies, and start preparing the transition to adulthood.

In cases where children have very little autonomy, or severe behavioral problems, they can be referred to special education schools.

Some centers offer specific intervention programs (e.g., DIR Floortime, applied behavior analysis (ABA)). Several other complementary and alternative treatments have been publicized and eventually used but generally with limited adherence.

After compulsory schooling, people with autism having good autonomy are, whenever possible, oriented toward professional employment (sometimes supported by protected employment or supported employment structures). People with reduced autonomy are oriented to specialized centers, where multidisciplinary interventions and occupational activities are implemented. Some of these have also associated residential structures.

Overview of Research Directions

The country's major research centers endorsing autism research programs are connected to universities or, in some cases, to neuroscience research centers inside private, nonprofit foundations. The main centers of research in autism are located in Coimbra and Lisbon.

The research developed at these centers has been oriented mainly toward basic genetic mechanisms (Coutinho et al. 2007; Correia et al. 2009)

and cell biology (Oliveira et al. 2005), neuropsychological characterization of ASD (Carmo et al. 2016, 2017), adaptive behavioral profiles (Mouga et al. 2015, 2016), and neurophysiology (Amaral et al. 2015; Tavares et al. 2016).

Some of these centers have also had an active participation in international consortia (Pinto et al. 2014; Salomone et al. 2016).

Overview of Training

The training is done in two different moments: undergrad and postgrad professional training. Different professional courses and university courses, namely, in psychology, psychomotricity, and teaching, consider in their curricula disciplines in which PEA is loomed. Posterior training is implemented in the centers devoted to autism that promote specific training and in-job training, under the guidance of supervisors with experience in Autism.

See Also

Portuguese Federation of Autism; <http://www.fpda.pt/>

Portuguese Neurodevelopmental Pediatrics Society; <http://www.spnd-spp.com>

References and Reading

- Amaral, C. P., Simões, M. A., & Castelo-Branco, M. S. (2015). Neural signals evoked by stimuli of increasing social scene complexity are detectable at the single-trial level and right lateralized. *PLoS One*, 25, 10(3).
- Carmo, J. C., Duarte, E., Pinho, S., Filipe, C. N., & Marques, J. F. (2016). Preserved proactive interference in autism Spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(1), 53–63.
- Carmo, J. C., Duarte, E., Souza, C., Pinho, S., & Filipe, C. N. (2017). Brief report: Testing the impairment of initiation processes hypothesis in autism Spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(4), 1256–1260.
- Correia, C., Coutinho, A. M., Almeida, J., Lontro, R., Lobo, C., Miguel, T. S., Martins, M., Gallagher, L., Conroy, J., Gill, M., Oliveira, G., & Vicente, A. M. (2009). Association of the alpha4 integrin subunit gene (ITGA4) with autism. *American Journal of Medical*

Genetics. Part B, Neuropsychiatric Genetics, 150B(8), 1147–1151.

- Coutinho, A. M., Sousa, I., Martins, M., Correia, C., Morgadinho, T., Bento, C., Marques, C., Ataíde, A., Miguel, T. S., Moore, J. H., Oliveira, G., & Vicente, A. M. (2007). Evidence for epistasis between SLC6A4 and ITGB3 in autism etiology and in the determination of platelet serotonin levels. *Human Genetics*, 121(2), 243–256.
- Mouga, S., Almeida, J., Café, C., Duque, F., & Oliveira, G. (2015). Adaptive profiles in autism and other neurodevelopmental disorders. *Journal of Autism and Developmental Disorders*, 45(4), 1001–1012.
- Mouga, S., Café, C., Almeida, J., Marques, C., Duque, F., & Oliveira, G. (2016). Intellectual profiles in the autism Spectrum and other neurodevelopmental disorders. *Journal of Autism and Developmental Disorders*, 46(9), 2940–2955.
- Oliveira, G., Ataíde, A., Marques, C., Miguel, T. S., Coutinho, A. M., Mota-Vieira, L., Gonçalves, E., Lopes, N. M., Rodrigues, V., Carmona da Mota, H., & Vicente, A. M. (2007). Epidemiology of autism spectrum disorder in Portugal: Prevalence, clinical characterization, and medical conditions. *Developmental Medicine and Child Neurology*, 49(10), 726–733.
- Oliveira, G., Diogo, L., Grazina, M., Garcia, P., Ataíde, A., Marques, C., Miguel, T., Borges, L., Vicente, A. M., & Oliveira, C. R. (2005). Mitochondrial dysfunction in autism spectrum disorders: A population-based study. *Developmental Medicine and Child Neurology*, 47(3), 185–189.
- Pinto, D., et al. (2014). Convergence of genes and cellular pathways dysregulated in autism spectrum disorders. *American Journal of Human Genetics*, 94(5), 677–694.
- Salomone, E., et al. (2016). Use of early intervention for young children with autism spectrum disorder across Europe. *Autism*, 20(2), 233–249.
- Tavares, P. P., Mouga, S. S., Oliveira, G. G., & Castelo-Branco, M. (2016). Preserved face inversion effects in adults with autism spectrum disorder: An event-related potential study. *Neuroreport*, 27(8), 587–592.

Positive Behavior (al) Interventions and Supports (PBIS)

- [Positive Behavioral Interventions and Supports \(PBIS\)](#)

Positive Behavior Intervention

- [Positive Behavioral Support](#)

Positive Behavior Support

- [Positive Behavioral Support](#)

Positive Behavior Support (PBS)

- [Positive Behavioral Interventions and Supports \(PBIS\)](#)

Positive Behavioral Interventions and Supports (PBIS)

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Synonyms

[Positive Behavior \(al\) Interventions and Supports \(PBIS\)](#); [Positive Behavior Support \(PBS\)](#); [Program-Wide Positive Behavior Supports \(PWPBS\)-early childhood programs](#); [School-Wide Positive Behavior \(al\) Interventions and Supports \(SWPBIS\)-for Schools K-12](#); [School-Wide Positive Behavior Support \(SWPBS\)-for Schools K-12](#)

Definition

Positive behavior support (PBS), also referred to as positive behavioral interventions and supports (PBIS), is a multitiered data-driven system of support, assessment, and intervention which has a potentially broad application base. Positive behavioral interventions and supports incorporate both academic and social interventions to promote overall competence. PBIS systems and procedures utilize empirically based decision-making

to support appropriate behaviors and successful student outcomes. Over the past decades, the research-based practices characteristic of PBIS have been shown effective in promoting a positive school climate and the prosocial behaviors which are important for success later in life (Gage et al. 2015). School-wide positive behavior support (SWPBS) has grown in popularity in both special and general education settings. Studies have shown improved outcomes for schools which implement this system with fidelity (Simonsen et al. 2012). More recently, data suggests that PBS could be helpful in other alternative settings (Simonsen and Sugai 2013; Farkas et al. 2012).

Positive behavioral interventions and supports uses a continuum approach and therefore contains systems and procedures which are flexible enough to meet the varying needs of individuals as well as provides an infrastructure of supports for larger organizations or groups. This increasingly popular collaborative and comprehensive prosocial model began as a non-aversive applied behavioral analysis-based special education intervention. PBS has been continuously evolving over the past 35 years.

Positive behavior support has recently been redefined by Kincaid et al. (2016):

PBS is an approach to behavior support that includes an ongoing process of research-based assessment, intervention, and data-based decision-making focused on building social and other functional competencies, creating supportive contexts, and preventing strategies that are respectful of a person's dignity and overall well-being and that are drawn primarily from behavioral, educational, and social services, although other evidence-based procedures may be incorporated. PBS may be applied within a multitiered framework at the level of the individual and at the level of larger systems (e.g., families, classrooms, schools, social service programs, and facilities) (p. 71).

Historical Background

Positive behavior support emerged in the 1980s as a direct result of the disability civil rights

movement. The concomitant deinstitutionalization of the disabled brought with it a need to manage behavioral issues at home, in the community, and at school. P.L. 94–142, the Education for All Handicapped Children Act (1975), mandated a free appropriate public education (FAPE) for all children regardless of disability. Consequently, families and local school districts were now required to figure out how to overcome behavioral challenges, comply with the new education laws, and accommodate children with disabilities.

At that time, applied behavioral analysis and its aversive therapies were the dominant approach for professionals to remediate problematic or challenging behaviors of students with disabilities. Negative reinforcement therapies, such as electric shock or other painful treatments, were controversial, but many medical professionals accepted these practices for their utility and effectiveness. In the 1960s, Lovaas's work demonstrated that the behavior of autistic children with severe communication issues could be modified by administering consequences. Lovaas advocated both positive reinforcement and more controversially contingent aversives, if necessary. He was an early advocate of using applied behavior analysis interventions for children with autism. His work showed that children with autism could be maintained at home or in a community setting (Smith and Eikeseth 2011).

The general public was not ready to tolerate harsh aversive practices in the community. Functional analysis was developed as an alternative to punishment and consequence-based behavior modification (Dunlap et al. 2009). This positive-based and non-aversive option to control difficult behaviors was welcomed by both parents and professionals to maintain disabled youth in both community and educational settings. Positive behavior supports uses functional analyses to examine and problem solve challenging behaviors. Evidence-based interventions and strategies are employed to support improved student outcomes and decrease incidences of inappropriate behavior. Functional behavioral analysis (FBA) has evolved into a process of understanding the primary function and underlying conditions of a problematic behavior. Data collection and

subsequent hypotheses help to determine the best way to reduce an undesirable behavior. Several recent studies have shown evidence that using the FBA process is an effective intervention for individuals with autism spectrum disorders from toddler through young adult (Fettig 2013).

The momentum of the positive behavior support movement was reinforced in the 1990s, with the amendment of P.L. 94–142, as the Individuals with Disabilities Education Act (IDEA) of 1997. The reauthorization of this special education law explicitly named positive behavioral interventions and supports (PBIS) as an effective school-wide approach to manage student behaviors (National Education Association 2014). More recently, P.L. 108–446, the Individuals with Disabilities Education Improvement Act of 2004, requires that all students receive free appropriate public education in the least restrictive environment (LRE).

Through the years, Congress has amended the IDEA (1997) and IDEIA (2004) with the goal of supporting students with both developmental and emotional difficulties. This legislation mandates schools use empirically supported practices to address social and academic behaviors which might interfere with a student's overall learning and success at school. The utilization of positive behavior support's preventative, prosocial, and empirically grounded elements, such as functional behavioral assessments (FBA) and behavioral intervention plans (BIP), and identification of measurable goals within an individualized education plan (IEP) is supported by this groundbreaking legislation. (www.pbis.org/school/pbis-and-the-law).

Federal grants have led to continued support for the development and evolution of the positive behavioral interventions and supports movement. The national positive behavior interventions and supports (PBIS) technical assistance center is maintained by the US Department of Education's Office of Special Education Programs (OSEP). This Center is staffed by researchers from the University of Oregon and the University of Connecticut. Professional training and implementation materials are free and readily available on the comprehensive website, PBIS.org. Procedures to monitor and support fidelity in the

implementation of positive behavior supports are provided by OSEP. The PBIS technical assistance center also maintains a computer application which provides a mechanism to support data collection in school-like environments for a nominal yearly fee. Organizations are also asked to complete surveys and continuously assess the progress and integrity with which they are adapting the PBIS framework. These assessment tools are available for organizations on the PBIS.org website. The fidelity of application is a key factor to consider when measuring the success and impact of the PBIS approach (Farkas et al. 2012).

Current Knowledge

Initially, positive behavior support was instituted in schools to address students with emotional and behavioral disorders. However, it was soon realized that positive behavior supports could be helpful for all students. The positive behavior preventative approach includes a strong interest in positive outcomes for individuals while maintaining respect for their dignity. Researched practices which promote social and academic success have been incorporated into the positive behavior approach framework. The current social service movement toward person-centered planning and self-determination works well within this approach. According to Horner (2013), more than 19,000 schools in the United States have trained their staff in school-wide positive behavior interventions and supports (SWPBIS) since 2000. The popularity of positive behavior support in schools does vary by state. For example, there are three states which have 60% of their schools following positive behavior support, and 16 states have greater 30% of their schools utilizing SWPBIS (Sugai and Simonsen 2012).

Positive behavioral interventions and supports focuses on supporting all students by creating a universally positive climate, utilizing research-based best practices both inside the classroom and outside the classroom, and examining the function of behaviors to identify appropriate individualized interventions. Positive behavior supports is data based, and decision-making is based

upon reported behaviors and systems for continued evaluation of outcomes. Mechanisms for the collection and analysis of behavioral data to assess the effectiveness of a selected approach are an important characteristic of positive behavior supports. The examination of student (s) response(s) to intervention(s) allows staff to modify their approach, when needed. The three-tiered continuum approach of positive behavior supports gives flexibility to address issues which impact an entire organization, a subgroup of the organization with a common behavioral issue, or, if needed, individual students. More support, intensive intervention, or skill development can be added, if deemed necessary. There are also several tools to aid organizations in monitoring and assessing their overall fidelity in implementing the positive behavior support approach. The positive behavior supports framework usually takes between 3 and 5 years to become fully functional in an organization.

The positive behavioral interventions and supports approach encompasses three integrated tiers which emphasize prevention by teaching, reteaching, and praising expected behaviors on a continuum. The tier one foundation of positive behavioral intervention and supports continuum approach supports the universal communication of a school-wide culture. This “code of conduct” clearly operationalizes positively stated prosocial values. Positive behavior supports seeks to teach all students how to behave by establishing standards of behavior across various settings. By teaching, reteaching, and reinforcing appropriate behaviors, schools improve the learning environment. By improving the school’s overall climate, the hope is to increase overall student achievement.

The connection between socio-emotional competence and academic achievement has been documented (Farkas et al. 2012). The explicit delineation of expected behaviors is particularly helpful to students with autism spectrum disorders who tend to have difficulty recognizing social cues or picking up on social norms in different settings. The concrete existence of positively stated expectations and rules guides students toward being better able to navigate toward more appropriate behaviors. The provision of

recognition and rewards to those students who meet or exceed stated standards serves to further reinforce appropriate behaviors. The consistency of language and predictable responses also works well for students with autism spectrum disorder. Common examples of this “code of conduct” might include “be responsible,” “be safe,” and/or “be respectful.” Specific descriptions, posters, or role plays around exactly “what this value looks like” are part of the PBIS teaching and reteaching component and are embedded into the curriculum across various settings.

The tier 2 of the positive behavioral interventions and supports continuum approach supports subgroups of students who need additional skill building in an area. A variety of interventions which can exist as part of the school’s available extra supports could be implemented. An example of such an intervention could be a social skill group, a check in/check out, or a study skills group. These groups would typically break down the steps of the required skill and provide opportunities for students to practice these skills. Staff would be trained in leading and monitoring these interventions. Data-based decision-making would determine the effectiveness and duration of participation in these interventions.

Tier 3 of the positive behavioral interventions and supports continuum approach provides more individualized plans for students who require more specific and intensive supports to meet behavioral expectations. At this level, functional behavioral assessments and behavioral intervention plans are important to identify which student needs, the inappropriate behavior is meeting (usually to obtain or to escape). A specific plan to discourage and replace the problematic behavior would be developed. The plan would need to be communicated and implemented across the whole organization for consistency. Family involvement and feedback can also be valuable.

Functional behavioral assessments consider behaviors from a more ecological perspective with an emphasis on prevention. Positive behavioral interventions and supports encourages the use of functional behavioral assessments at all tiers to analyze problems by understanding the

behavioral pathway. The behavioral pathway examines not only the problematic behavior but also the specific circumstances and environments under which the behavior occurs. By examining the behavioral pathway, interventions consider intrapersonal, interpersonal, environmental, and systemic factors. Preventative measures and additional supports can be instrumental in addressing factors leading up to the problematic behavior such as setting events, triggering antecedents, and maintaining consequences. The functional behavioral assessment shifts the focus from the behavior to the function of the behavior. The purpose is to identify a more acceptable replacement behavior which serves the same function as the unacceptable behavior.

Youth with autism and other developmental disorders may exhibit behaviors which interrupt the educational process and negatively impact their academic performance. The goal of positive behavior supports is to promote social competence, increase appropriate behaviors, and therefore encourage both social and academic achievement. The collaborative team and school-wide nature of this framework approach promote consistency and continuity and therefore would work well in meeting the needs of students with autism spectrum disorder (ASD) who benefit from structure, routine, and repetition. The transparency of expectations could also lower the stress level for individuals with ASD who do better when they can predict their environment (LaVoie 2005). Engaging families in the positive behavioral interventions and supports process is also an important component of setting students up for success.

As students progress through school and begin to transition toward adulthood, they are increasingly required to be accountable in managing their time and schedule. Positive behavioral interventions and supports usually promotes the prosocial value of being responsible. Research-based interventions which have been proven effective in promoting executive skill management, such as modeling, prompting, reinforcement, social skills training, etc., can all be embedded as interventions within the many tiers of the PBIS framework (Wong et al. 2013).

Future Directions

Positive behavioral interventions and supports is a very dynamic concept which is continually evolving. A wide array of literature provides empirical support for school-wide positive behavior supports as helpful to all students (Horner et al. 2010). Findings have associated school-wide positive behavioral interventions and supports with a more positive school environment, decrease in incidences requiring disciplinary actions, and improved academic achievements (Farkas et al. 2012).

States have been providing technical assistance to support the training and assistance to schools helping them to accurately implement all three tiers of PBIS. An integral part of positive behavior supports is the structure and designation of trained leadership teams who guide and support its adaption. School staff members are integral to the continuity and success of the implementation process. Data indicate significant positive outcomes when positive behavior supports is implemented correctly (Gage et al. 2015). There are several ways to evaluate the fidelity of implementation at each tier. A vast majority of states provide support for PBIS at either the district or state level to implement tier 1. However, the more specific training required and additional resources necessary to correctly implement tier 2 and tier 3 with fidelity make its successful adaptation more difficult (Pas and Bradshaw 2012). With so many variables to research within the positive behavior interventions and supports framework, the critical impact of each factor and stage of implementation on the effectiveness of PBIS need further research to be clarified (Childs et al. 2016).

The mandate for schools to use data-based methods to ensure a free and appropriate education for all has spurred research on best practices. The connection between academic achievement and social success is crucial. Positive behavior supports was originally developed for use in general education settings. Its success has led to consideration of instituting this framework in alternative organizations such as residential settings, psychiatric hospitals, mental health,

juvenile correction, day treatment programs, or transition programs. Implementation efforts in schools which only serve students with significant disabilities also need to be evaluated (Schelling and Harris 2016).

Several recent high-profile instances of violence in schools have expanded interest in addressing mental health issues for children in schools. According to Gage et al. (2015), PBIS has been associated with improved school climate and a reduction in disciplinary procedures such as out-of-school suspensions (Gage et al. 2015). The No Child Left Behind Act (2001) includes an emphasis on addressing issues of student well-being. Future directions to improve student well-being should look at the possible development of “wraparound” collaborations between the positive behavior support framework in educational settings and mental health organizations (Anello et al. 2016).

Future directions include the consideration of adapting positive behavior support systems within alternative programs (Simonsen and Sugai 2013). More studies and research are needed to establish the effectiveness of PBIS in alternative settings (Farkas et al. 2012). P.L. 110–315, the Higher Education Opportunity Act (HEOA) of 2008, establishes better access for students with intellectual disabilities to postsecondary education. Research should be conducted to consider how to adapt the positive behavior supports model in college settings.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Elementary and Secondary Education Act \(No Child Left Behind\)](#)
- [Free Appropriate Public Education](#)
- [Functional Analysis](#)
- [Functional Analysis: Assessment Procedures for Complex Challenging Behavior](#)
- [Higher Education Opportunity Act of 2008 \(HEOA\)](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Least Restrictive Environment \(LRE\)](#)

References and Reading

- Anello, V., Weist, M., Eber, L., Barrett, S., Cashman, J., Rosser, M., & Bazyk, S. (2016). Readiness for positive behavioral interventions and supports and school mental health interconnection: Preliminary development of a stakeholder survey. *Journal of Emotional and Behavioral Disorders*, 1–14. <https://doi.org/10.1177/1063426616630536>.
- Bradshaw, C., Pas, E. T., Bloom, J., Barrett, S., Hershfeldt, P., Alexander, A., McKenna, M., Chafin, A., & Leaf, P. J. (2012). A state-wide partnership to promote safe and supportive schools: The PBIS Maryland initiative. *Administration and Policy in Mental Health and Mental Health Services Research*, 39(4), 225–237. <https://doi.org/10.1007/s10488-011-0384-6>.
- Childs, K. E., Kincaid, D., George, H. P., & Gage, N. A. (2016). The relationship between school-wide implementation of positive behavior intervention and supports and student discipline outcomes. *Journal of Positive Behavior Interventions*, 18(2), 89–99. <https://doi.org/10.1177/1098300715590398>.
- Dunlap, G., Horner, R. H., & Sugai, G. (2009). Overview and history of positive behavior support. In W. Sailor, G. Dunlap, G. Sugai, & R. Horner (Eds.), *Handbook of positive behavior support* (pp. 3–16). New York: Springer. <https://doi.org/10.1007/978-0-387-09632-2>.
- Education for All Handicapped Children Act of 1975. RL. 94–142. 20 U.S.C. § 1400 et seq.
- Farkas, M. S., Simonsen, B., Migdole, S., Donovan, M. E., Clemens, K., & Cicchese, V. (2012). School-wide positive behavior support in an alternative school setting: An evaluation of fidelity, outcomes and social validity of tier 1 implementation. *Journal of Emotional and Behavioral Disorders*, 20(4), 275–288. <https://doi.org/10.1177/1063426610389615>.
- Fettig, A. (2013). *Functional behavior assessment (FBA) fact sheet*. Chapel Hill: The University of North Carolina, Frank Porter Graham Child Development Institute, The National Professional Development Center on Autism Spectrum Disorders.
- Gage, N. A., Sugai, G., Lewis, T., & Brzozowy, S. (2015). Academic achievement and school-wide positive behavior supports. *Journal of Disability Policy Studies*, 25(4), 199–209. <https://doi.org/10.1177/1044207313505647>.
- Higher Education Opportunity Act of 2008. P.L. 110–314, 20 U.S.C. § 1001 et seq.
- Horner, R. H. (2013). *Implementing SWPBIS with quality, equity and efficiency*. Retrieved 2 May 2016, from <http://www.pbis.org/resource/941>.
- Horner, R. H., Sugai, G., & Anderson, C. M. (2010). Examining the evidence base for school-wide positive behavior support. *Focus on Exceptional Children*, 42, 1–14.
- Individuals with Disabilities Education Act of 1997. P.L. 105–17, 20 U.S.C. § 1400 et seq.
- Individuals with Disabilities Education Improvement Act of 2004. P.L. 108–446, 20 U.S.C. § 1400 et seq.

- Kincaid, D., Dunlap, G., Kern, L., Lane, K., Bambara, L., Brown, F., et al. (2016). Positive behavior support: A proposal for updating and refining the definition. *Journal of Positive Behavior Interventions*, 18(2), 69–73. <https://doi.org/10.1177/1098300715604826>.
- Koegel, L. K., Robinson, S., & Koegel, R. L. (2009). Empirically supported intervention practices for autism spectrum disorders in school and community settings: Issues and practices. In W. Sailor, G. Dunlap, G. Sugai, & R. Horner (Eds.), *Handbook of positive behavior support* (pp. 149–176). New York: Springer. <https://doi.org/10.1007/978-0-387-09632-2>.
- LaVoie, R. (2005). *It's so much work to be your friend: Helping the child with learning disabilities find social success*. New York: Simon & Schuster.
- National Education Association. (2014). *Positive behavioral interventions and supports: A multi-tiered framework that works for every student*. http://www.nea.org/assets/docs/PB41A-Positive_Behavioral_Interventions-Final.pdf
- No Child Left Behind Act of 2001. P.L. 107–110, 20 U.S.C. § 6301 et seq.
- Pas, E. T., & Bradshaw, C. P. (2012). Examining the association between implementation and outcomes: State-wide scale-up of school-wide positive behavior intervention and supports. *Journal of Behavioral Health Services & Research*, 39(4), 417–433.
- Positive Behavioral Interventions & Supports – Office of Special Education Programs. (n.d.). Retrieved 11 May 2016, from <http://www.pbis.org/school/pbis-and-the-law>
- Russa, M. B., Matthews, A. L., & Owen-DeSchryver, J. S. (2015). Expanding supports to improve the lives of families of children with autism spectrum disorder. *Journal of Positive Behavior Interventions*, 17(2), 95–104. <https://doi.org/10.1177/1098300714532134>.
- Schelling, A. L., & Harris, M. L. (2016). School-wide positive behavioral interventions and supports: A snapshot of implementation in schools serving students with significant disabilities. *Journal of Positive Behavior Interventions*, 1–10. <https://doi.org/10.1177/1098300716632360>.
- Simonsen, B., & Sugai, G. (2013). PBIS in alternative education settings: Positive support for youth with high-risk behavior. *Education and Treatment of Children*, 36(3), 3–14.
- Simonsen, B., Britton, L., & Young, D. (2010). School-wide positive behavior support in an alternative school setting: A case study. *Journal of Positive Behavior Interventions*, 12(3), 180–191. <https://doi.org/10.1177/1098300708330495>.
- Simonsen, B., Eber, L., Black, A. C., Sugai, G., Lewandowski, H., Sims, B., et al. (2012). Illinois state-wide positive behavior interventions and supports: Evolution and impact on student outcomes across years. *Journal of Positive Behavior Interventions*, 14(1), 5–16. <https://doi.org/10.1177/1098300711412601>.
- Smith, T., & Eikeseth, S. (2011). O. Ivar Lovaas: Pioneer of applied behavior analysis and intervention for children with autism. *Journal of Autism and Developmental Disorders*, 41(3), 375–378.
- Strain, P. S., & Schwartz, I. (2009). Positive behavior support and early intervention for young children with autism: Case studies on the efficacy of proactive treatment of problem behavior. In W. Sailor, G. Dunlap, G. Sugai, & R. Horner (Eds.), *Handbook of positive behavior support* (pp. 107–123). New York: Springer. <https://doi.org/10.1007/978-0-387-09632-2>.
- Sugai, G., & Simonsen, B. (2012). *Positive behavioral interventions and supports: History, defining features, and misconceptions*. Center for PBIS & Center for Positive Behavioral Interventions and Supports, University of Connecticut. Retrieved from www.PBIS.org
- Wong, C., Odom, S. L., Hume, K., Cox, A. W., Fetting, A., Kucharczyk, S., et al. (2013). *Evidence-based practices for children, youth and young adults with autism spectrum disorder*. Chapel Hill: The University of North Carolina, Frank Porter Graham Child Development Institute Autism Evidence-Based Practice Review Group.

Positive Behavioral Support

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Synonyms

Positive behavior intervention; Positive behavior support

Definition

Positive behavioral support is both a values-based and an empirically verified system that uses specific nonaversive procedures to improve an individual's quality of life as well as reduce their problem behaviors in community settings. Positive behavioral support procedures were developed from the literature of applied behavioral analysis and operant conditioning and were created in response to the concern over the possible detrimental effects of aversive behavioral interventions on a person. Positive behavioral support

comprises varied treatment methods including pivotal response treatments; however, there are themes central to all techniques. One, functional analysis is used to identify both the context as well as the adaptive function of the problem behavior. Two, the intervention provides behavioral change while minimizing the use of punishers. Three, the use of the intervention is justified by the outcome. Four, the intervention considers the ecological context of the behavior. Five, the intervention is socially valid and respectful of a person's dignity.

References and Reading

- Carr, E. G., Dunlap, G., Horner, R. H., Koegel, R. L., Turnbull, A. P., Sailor, W., et al. (2002). Positive behavior support: Evolution of an applied science. *Journal of Positive Behavior Interventions*, 4(20), 4–16.
- Dunlap, G., & Fox, L. (1996). Early intervention and serious problem behaviors: A comprehensive approach. In L. K. Koegel, R. L. Koegel, & G. Dunlap (Eds.), *Positive behavioral support: Including people with difficult behavior in the community* (pp. 31–50). Baltimore: Paul H. Brookes Publishing.
- Horner, R. H., Dunlap, G., Koegel, R. L., Carr, E. G., Sailor, W., Anderson, J., et al. (1990). Toward a technology of “nonaversive” behavioral support. *The Journal of the Association for Persons With Severe Handicaps*, 15, 125–132.

Positive reinforcement is defined as the presentation of a stimulus contingent on a behavior that results in an increased frequency of that behavior in the future. The reinforcing stimulus is strongest if it is presented immediately following the behavior. For example, if a child with autism is told to sit in a chair and receives a desired treat such as a candy immediately after sitting, the likelihood of the child sitting down more frequently in the future increases. The effect of reinforcement depends on the strength of the reinforcer being used. However, procedures are also available for using delayed or partial reinforcement schedules. Strong reinforcers are idiosyncratic and depend on the individual as well as the environmental context, availability of other reinforcers, etc. Commonly used reinforcers include edibles, activities, praise, and tangibles such as toys or stickers.

See Also

- ▶ [Negative Reinforcement](#)
- ▶ [Reinforcement](#)
- ▶ [Schedule of Reinforcement](#)

Positive Reinforcement

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Definition

Positive reinforcement is a critical component of B. F. Skinner's behavioral theory of learning.

References and Reading

- Charlop-Christy, M. H., & Haymes, L. K. (1996). Using obsessions as reinforcers with and without mild reductive procedures to decrease inappropriate behaviors of children with autism. *Journal of Autism and Developmental Disorders*, 26(5), 527–546.
- Koegel, R. L., Vernon, T. W., & Koegel, L. K. (2009). Improving social initiations in young children with autism using reinforcers with embedded social interactions. *Journal of Autism and Developmental Disorders*, 39(9), 1240–1251.
- Koegel, L. K., Singh, A. K., & Koegel, R. L. (2010). Improving motivation for academics in children with autism. *Journal of Autism and Developmental Disorders*, 40(9), 1057–1066.
- Skinner, B. F. (1953). *Science and human behavior*. New York: MacMillan.
- Weeden, M., & Poling, A. (2011). Identifying reinforcers in skill acquisition studies involving participants with autism: Procedures reported from 2005 to 2009. *Research in Autism Spectrum Disorders*, 5(1), 388–391.

Positron-Emission Tomography

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Synonyms

[PET](#)

Definition

Positron-emission tomography (PET) is an imaging technique from the field of nuclear medicine. The technique produces a three-dimensional image or picture of functional processes in the body including the brain. The technique involves the use of a detector to measure pairs of gamma rays emitted indirectly by a positron-emitting radionuclide (tracer), which is introduced into the body on a biologically active molecule. Three-dimensional images of tracer concentration within the body are then constructed by computer analysis. With this technique, it is possible to image brain function and its disruption in neuropsychiatric and neurodevelopmental disorders. Because the technique involves ionizing radiation, it is not typically used in research studies of children.

See Also

► [Computed Tomography](#)

References and Reading

Zilbovicius, M., Boddaert, N., Belin, P., Poline, J. B., Remy, P., Mangin, J. F., et al. (2000). Temporal lobe dysfunction in childhood autism: A PET study. *The American Journal of Psychiatry*, 157(12), 1988–1993.

Posttraumatic Stress Disorder

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Synonyms

[Combat disorder](#); [Combat fatigue](#); [Combat neurosis](#); [Complete exhaustion](#); [Operational exhaustion](#); [Operational stress injury](#); [Shell shock](#); [Stress disorder](#)

Short Description or Definition

Posttraumatic stress disorder (PTSD) is a disorder characterized by specific symptoms which can develop after exposure to a potential traumatic event (PTE) or a number of traumatic events. PTEs in the general population may involve rape, motor vehicle accidents, assault, natural disasters, or terrorism. In the military, war zone or peacekeeping exposure – including imprisonment, torture, or witnessing atrocities or comrades being wounded or killed – might serve as a significant traumatic event. In PTSD, the individual will relive the traumatic event through intrusive thoughts and recollections during the day or disturbing dreams at night. When triggered by reminders, individuals often become emotionally upset and exhibit physical symptoms of anxiety. They will often avoid thinking and talking about their traumatic event or avoid specific reminders that trigger distress. Individuals may suffer from persistent negative

beliefs and emotions, lose interest in previously pleasurable activities, and feel detached from others. They generally also display hyperarousal symptoms including difficulty sleeping, poor concentration, irritability, hypervigilance, and being easily startled.

Categorization

PTSD is classified as a trauma-and-stressor-related disorder under the *Diagnostic and Statistical Manual of Mental Disorder* (American Psychiatric Association [APA] 2013). PTSD is unique among psychiatric diagnoses because of the requirement of a traumatic event in order to make the diagnosis. The traumatic event involves exposure to actual or threatened death, serious injury, or sexual violence. In order to meet the criteria for the “traumatic event,” exposure must occur by directly experiencing the traumatic event, witnessing the event occur to others, learning that the traumatic event occurred to a close family member or friend, and/or experiencing repeated or extreme exposure to aversive details of the traumatic event (Criterion A). Symptoms of PTSD are grouped in four main symptom clusters: reexperiencing, avoidance, negative alterations in cognition and mood, and hyperarousal. In addition to meeting the traumatic stressor criteria, the individual must present with at least one intrusive reexperiencing (Criterion B) symptom where the individual will relive the traumatic event in intrusive thoughts and recollections, one or both symptoms of avoidance (Criterion C), at least two symptoms of negative cognitions and moods (Criterion D), and at least two symptoms of hyperarousal (Criterion E). Finally, to make the diagnosis, the individual exposed to the traumatic event must experience significant impairment in social and/or occupational functioning as a result of their symptoms (Criterion G). According to DSM 5, the duration of the disturbance must be greater than one month (Criterion F) (American Psychiatric Association [APA] 2013) (Table 1).

Posttraumatic Stress Disorder, Table 1 Posttraumatic Stress Disorder, Diagnostic Criteria (excerpt from the DSM-5, pages 271–274). (Reprinted with permission from the Diagnostic and Statistical Manual of Mental Disorders. Copyright 2013. American Psychiatric Association)

Note: The following criteria apply to adults, adolescents, and children older than 6 years. For children 6 years and younger, see corresponding criteria below.

A. Exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways:

1. Directly experiencing the traumatic event(s).
2. Witnessing, in person, the event(s) as it occurred to others.
3. Learning that the traumatic event(s) occurred to a close family member or close friend. In cases of actual or threatened death of a family member or friend, the event (s) must have been violent or accidental.

4. Experiencing repeated or extreme exposure to aversive details of the traumatic event(s) (e.g., first responders collecting human remains; police officers repeatedly exposed to details of child abuse).

Note: Criterion A4 does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work-related.

B. Presence of one (or more) of the following intrusion symptoms associated with the traumatic event(s), beginning after the traumatic event(s) occurred:

1. Recurrent, involuntary, and intrusive distressing memories of the traumatic event(s).

Note: In children older than 6 years, repetitive play may occur in which themes or aspects of the traumatic event(s) are expressed.

2. Recurrent distressing dreams in which the content and/or affect of the dream are related to the traumatic event(s).

Note: In children, there may be frightening dreams without recognizable content.

3. Dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the traumatic event (s) were recurring. (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.)

Note: In children, trauma-specific reenactment may occur in play.

4. Intense or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).

5. Marked physiological reactions to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).

C. Persistent avoidance of stimuli associated with the traumatic event(s), beginning after the traumatic event (s) occurred, as evidenced by one or both of the following:

1. Avoidance of or efforts to avoid distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).

2. Avoidance of or efforts to avoid external

(continued)

Posttraumatic Stress Disorder, Table 1 (continued)

reminders (people, places, conversations, activities, objects, situations) that arouse distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).

D. Negative alterations in cognitions and mood associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:

1. Inability to remember an important aspect of the traumatic event(s) (typically due to dissociative amnesia and not to other factors such as head injury, alcohol, or drugs).

2. Persistent and exaggerated negative beliefs or expectations about oneself, others, or the world (e.g., "I am bad," "No one can be trusted," "The world is completely dangerous," "My whole nervous system is permanently ruined").

3. Persistent, distorted cognitions about the cause or consequences of the traumatic event(s) that lead the individual to blame himself/herself or others.

4. Persistent negative emotional state (e.g., fear, horror, anger, guilt, or shame).

5. Markedly diminished interest or participation in significant activities.

6. Feelings of detachment or estrangement from others.

7. Persistent inability to experience positive emotions (e.g., inability to experience happiness, satisfaction, or loving feelings).

E. Marked alterations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:

1. Irritable behavior and angry outbursts (with little or no provocation) typically expressed as verbal or physical aggression toward people or objects.

2. Reckless or self-destructive behavior.

3. Hypervigilance.

4. Exaggerated startle response.

5. Problems with concentration.

6. Sleep disturbance (e.g., difficulty falling or staying asleep or restless sleep).

F. Duration of the disturbance (Criteria B, C, D, and E) is more than 1 month.

G. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

H. The disturbance is not attributable to the physiological effects of a substance (e.g., medication, alcohol) or another medical condition.

Specify whether:

With dissociative symptoms: The individual's symptoms meet the criteria for posttraumatic stress disorder, and in addition, in response to the stressor, the individual experiences persistent or recurrent symptoms of either of the following:

(continued)

Posttraumatic Stress Disorder, Table 1 (continued)

1. **Depersonalization:** Persistent or recurrent experiences of feeling detached from, and as if one were an outside observer of, one's mental processes or body (e.g., feeling as though one were in a dream; feeling a sense of unreality of self or body or of time moving slowly).

2. **Derealization:** Persistent or recurrent experiences of unreality of surroundings (e.g., the world around the individual is experienced as unreal, dreamlike, distant, or distorted).

Note: To use this subtype, the dissociative symptoms must not be attributable to the physiological effects of a substance (e.g., blackouts, behavior during alcohol intoxication) or another medical condition (e.g., complex partial seizures).

Specify whether:

With delayed expression: If the full diagnostic criteria are not met until at least 6 months after the event (although the onset and expression of some symptoms may be immediate).

Posttraumatic Stress Disorder for Children 6 Years and Younger

A. In children 6 years and younger, exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways:

1. Directly experiencing the traumatic event(s).

2. Witnessing, in person, the event(s) as it occurred to others, especially primary caregivers.

Note: Witnessing does not include events that are witnessed only in electronic media, television, movies, or pictures.

3. Learning that the traumatic event(s) occurred to a parent or caregiving figure.

B. Presence of one (or more) of the following intrusion symptoms associated with the traumatic event(s), beginning after the traumatic event(s) occurred:

1. Recurrent, involuntary, and intrusive distressing memories of the traumatic event(s).

Note: Spontaneous and intrusive memories may not necessarily appear distressing and may be expressed as play reenactment.

2. Recurrent distressing dreams in which the content and/or affect of the dream are related to the traumatic event(s).

Note: It may not be possible to ascertain that the frightening content is related to the traumatic event.

3. Dissociative reactions (e.g., flashbacks) in which the child feels or acts as if the traumatic event(s) were recurring. (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.) Such trauma-specific reenactment may occur in play.

4. Intense or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).

5. physiological reactions to reminders of the traumatic event(s).

(continued)

Posttraumatic Stress Disorder, Table 1 (continued)

C. One or more of the following symptoms, representing either persistent avoidance of stimuli associated with the traumatic event(s) or negative alterations in cognitions and mood associated with the traumatic event(s), must be present, beginning after the event(s) or worsening after the event(s):

Persistent Avoidance of Stimuli

1. Avoidance of or efforts to avoid activities, places, or physical reminders that arouse recollections of the traumatic event(s).

2. Avoidance of or efforts to avoid people, conversations, or interpersonal situations that arouse recollections of the traumatic event(s).

Negative Alterations in Cognitions

3. Substantially increased frequency of negative emotional states (e.g., fear, guilt, sadness, shame, confusion).

4. Markedly diminished interest or participation in significant activities, including constriction of play.

5. Socially withdrawn behavior.

6. Persistent reduction in expression of positive emotions.

D. Alterations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:

1. Irritable behavior and angry outbursts (with little or no provocation) typically expressed as verbal or physical aggression toward people or objects (including extreme temper tantrums).

2. Hypervigilance.

3. Exaggerated startle response.

4. Problems with concentration.

5. Sleep disturbance (e.g., difficulty falling or staying asleep or restless sleep).

E. The duration of the disturbance is more than 1 month.

F. The disturbance causes clinically significant distress or impairment in relationships with parents, siblings, peers, or other caregivers or with school behavior.

G. The disturbance is not attributable to the physiological effects of a substance (e.g., medication or alcohol) or another medical condition.

Specify whether:

With dissociative symptoms: The individual's symptoms meet the criteria for posttraumatic stress disorder, and in addition, in response to the stressor, the individual experiences persistent or recurrent symptoms of either of the following:

1. **Depersonalization:** Persistent or recurrent experiences of feeling detached from, and as if one were an outside observer of, one's mental processes or body (e.g., feeling as though one were in a dream; feeling a sense of unreality of self or body or of time moving slowly).

2. **Derealization:** Persistent or recurrent experiences of unreality of surroundings (e.g., the world

(continued)

Posttraumatic Stress Disorder, Table 1 (continued)

around the individual is experienced as unreal, dreamlike, distant, or distorted).

Note: To use this subtype, the dissociative symptoms must not be attributable to the physiological effects of a substance (e.g., blackouts, behavior during alcohol intoxication) or another medical condition (e.g., complex partial seizures).

Specify whether:

With delayed expression: If the full diagnostic criteria are not met until at least 6 months after the event (although the onset and expression of some symptoms may be immediate).

Epidemiology

Exposure to a PTE is common: approximately 75.7% of individuals are exposed to a traumatic event sufficient to cause PTSD. However, not all individuals exposed to a traumatic event develop PTSD. The National Comorbidity Survey in the United States reported PTSD rates of 3.5% (12-month) and 6.8% (lifetime) (Kessler et al. 1995). PTSD rates are higher in women; the National Comorbidity Survey demonstrated prevalence rates of 10% in women and 5% in men (Kessler et al. 1995). In Canada, the lifetime prevalence of PTSD is similar to that found in the United States. In a community study in Canada, prevalence rates of PTSD were 2.4% (current and 1-month) and 9.2% (lifetime) (Van Ameringen 2003).

In veteran and military populations, the current and lifetime prevalence of PTSD among serving Canadian Armed Forces members has been estimated at 2.8% and 7.2%, respectively (Statistics Canada 2002). In a sample of Canadian peacekeeping veterans pensioned with a medical condition, 1-month prevalence of probable PTSD was reported as 10.3% (Richardson et al. 2006). In samples of US military members following deployment to Iraq and Afghanistan, the rates of PTSD were estimated between 11.2% and 17.1%, compared to a baseline rate of 5% before deployment (Hoge et al. 2004); while in a sample of UK military members, the reported rates were significantly lower at 4.8% (Iversen et al. 2009).

Natural History, Prognostic Factors, Outcomes

In general, the posttraumatic stress symptoms which might appear following exposure to a PTE are generally considered to be a normal response. In most individuals, the symptoms gradually subside and do not cause significant long-term impairment. However, if the symptoms persist and cause significant impairment in social and/or occupational functioning, then it is considered a disorder. Individuals who develop PTSD do not acquire new symptoms; rather, the initial posttraumatic stress symptoms which presented following the traumatic event do not subside.

Many risk factors have been identified for PTSD and are generally divided into pretraumatic, peritraumatic, and posttraumatic factors. Understanding risk factors is important in order to better identify and screen those who might be at increased risk of developing PTSD following exposure to a traumatic event. Pretraumatic risk factors include a family and/or personal history of psychiatric illness and past trauma including history of childhood abuse (Brewin et al. 2000; Ozer et al. 2003). Gender is another risk factor; although women are more likely to develop PTSD, men are more likely to be exposed to a traumatic event (Breslau et al. 1998; Kessler et al. 1995). Other pretraumatic risk factors include younger age, single marital status, and lower socioeconomic status (Breslau et al. 2006; Richardson et al. 2007). Peritraumatic risk factors include trauma severity and life threat (Brewin et al. 2000; Hoge et al. 2004; Richardson et al. 2007), and bodily injury (Koren et al. 2005). Peritraumatic risk factors specific to military-related PTSD include the number of operational deployments (Dohrenwend et al. 2006; Hoge et al. 2004; Richardson et al. 2007; Statistics Canada 2002). Finally, posttraumatic risk factors may include lack of access to treatment, stigmatization, ongoing life stressors, and lack of social support (Brewin et al. 2000; Ozer et al. 2003; Yehuda et al. 1998). Shame and guilt for developing PTSD symptoms are also posttraumatic risk factors (Yehuda et al. 1998).

Although the majority of people with posttraumatic stress symptoms will recover without treatment, a notable proportion will present with

significant persistent symptoms of PTSD, resulting in distress and/or impairment in functioning. As such, these individuals stand to benefit from treatment. Although not always possible, complete remission can be achieved in 30–50% of cases of PTSD (Friedman 2006). Pharmacological interventions, in addition to psychotherapy, can assist with symptom reduction and improve functioning and quality of life.

Clinical Expression and Pathophysiology

Clinically, PTSD rarely occurs on its own. Over 90% of individuals with PTSD meet criteria for at least one other mental disorder. Comorbidity is the rule rather than the exception. The most common comorbid disorders are major depressive disorder; anxiety disorders (social phobia, generalized anxiety disorder, obsessive-compulsive disorder, and panic disorder); and alcohol and substance use disorders (Kessler et al. 1995). Less common comorbid disorders are bipolar disorder and personality disorders, especially borderline (Pagura et al. 2010) and antisocial (Sareen et al. 2004). It is critical that these disorders be diagnosed as they complicate the clinical picture and can compromise treatment. There is also emerging evidence demonstrating a strong relationship between PTSD and physical health problems (Jakupcak et al. 2008; Sareen et al. 2007). The most common medical complaints associated with PTSD include chronic pain syndromes, mild traumatic brain injuries, asthma, gastrointestinal complaints, and cardiovascular disease (Sareen et al. 2005, 2007).

Although the exact neurophysiology of PTSD is not known, there are neurobiological alterations that have been implicated in PTSD including adrenergic hyperresponsiveness (Southwick et al. 1999), low cortisol levels, and enhanced negative feedback sensitivity of the hypothalamic-pituitary-adrenal axis (Yehuda et al. 1993; Yehuda et al. 2004), dysregulation of the immune response (Altemus et al. 2003; Raison and Miller 2003), and decreased hippocampal volume (Bonne et al. 2001; Bremner et al. 1995, 1997; Stein et al. 1997).

Evaluation and Differential Diagnosis

Important factors to consider in the differential diagnosis include prior exposure to psychological trauma and presence of any PTSD symptoms. As outlined above, patients with PTSD present with four symptom clusters: reexperiencing, avoidance, negative cognitions and moods, and hyperarousal symptoms (Canadian Psychiatric Association 2006).

PTSD symptoms and signs are generally non-specific, so initially the differential diagnosis of presenting symptoms may include both physical and mental disorders (Jetly and Cooper 2008). Check for physical, psychological, and social symptoms, inquire about exposure to psychological trauma, and then work through the differential diagnosis (Friedman 2006). While no physical health condition explains full-spectrum PTSD (Staab 2009), the condition may not be apparent initially, owing to avoidance or reluctance to discuss trauma-related events. Comorbid physical and mental health conditions are common in PTSD, and many have overlapping symptoms.

There are well-validated screens available for PTSD. Short screening instruments such as the Primary Care PTSD Screen, a four-item yes/no screening instrument, are designed for use by primary care practitioners. This screen has a sensitivity of 78% and specificity of 87% for PTSD in individuals who endorse three or more items (Friedman 2006). Individuals who screen positive should then be assessed for PTSD using the DSM 5 diagnostic criteria (Table 1), or using more elaborate screening instruments such as the Clinician Administered PTSD Scale (CAPS) (Blake et al. 1995) or a self-rating scale such as the PTSD Checklist (Military or Civilian Version) (Weathers et al. 1993).

Treatment

Treatment of PTSD may include several disciplines and approaches and can vary depending on the severity and chronicity of trauma symptoms and the presence of comorbid disorders. Most often, a phase-oriented approach to

treatment is utilized which includes behavioral stabilization, psychoeducation, anxiety management, focused trauma work, relapse prevention, and aftercare (Herman 1992).

Once a diagnosis has been established, psychoeducation in group or individual formats detailing diagnosis and treatment is critical for both patient and family (American Psychiatric Association [APA] 2004; Foa et al. 2000; Turnbull and McFarland 1996; Van der Kolk et al. 1996). Educating patients about the phases of treatment reassures those frightened by the notion of psychiatric medication and psychotherapy, as well as sets appropriate expectations for treatment. The main goal of stabilization is to manage acute symptoms and improve current functioning with psychoeducation, medication, and anxiety management training. Once symptoms stabilize, patients are more able to engage in psychotherapy (Van der Kolk et al. 1996).

Psychotherapy

Psychotherapy aims to reduce symptom severity and improve global functioning and quality of life resulting in enhanced functioning in social and occupational areas. Psychotherapeutic interventions for PTSD with the strongest empirical evidence are those that include components of prolonged exposure (PE) and/or cognitive processing therapy (CPT) (APA 2004; Resick and Schnicke 1993; US Department of Veterans Affairs/Department of Defense 2010). In PE, the patient experiences the trauma imaginally or in vivo during planned treatment sessions, with a goal of diminishing distress by forming new associations. CPT focuses on the appraisal of the traumatic event and the emotions resulting from the event (i.e., cognitive distortions). Common cognitive distortions include perceiving the world as dangerous, seeing oneself as powerless or inadequate, or feeling guilty for outcomes believed preventable (Friedman 2006). In CPT, the patient engages in writing narratives of the trauma with a goal of processing the emotions and restructuring the dysfunctional beliefs.

Eye Movement Desensitization and Reprocessing (EMDR) has received some empirical support and is generally considered to be an evidence-based

treatment for PTSD (APA 2004; Friedman 2006). In EMDR, patients are instructed to imagine the painful traumatic memories and associated negative cognition such as guilt and shame while visually focusing on the rapid movement of the clinician's finger (Friedman 2006).

Acceptance and Commitment Therapy (ACT) is an emerging treatment for PTSD, having shown promising outcomes in a limited number of trauma research studies (Bean et al. 2017). The goal of ACT is to increase psychological flexibility while engaging in valued activities; ACT has been demonstrated to be effective in treating several other disorders, including substance abuse, depression, and chronic pain (Hayes et al. 2012). Given the often high rates of co-morbidity in PTSD and ACT's transdiagnostic application, it may prove to be an ideal treatment for PTSD.

Behavioral Activation (BA) is a behaviorally based treatment for depression that promotes engagement in behaviors that positively reinforce consequences, promoting behavioral momentum and a collateral improvement in mood (Martell et al. 2010). There have been some studies demonstrating effectiveness for BA in treating trauma: notably, BA has relatively low dropout rates (Jakupcak et al. 2006, 2010).

Regardless of the psychotherapeutic treatment modality, prior behavioral stabilization is critical as some comorbid conditions may preclude the introduction of treatment (e.g., suicidality, self-injurious behavior, or psychosis). Further, initiating "trauma-focused psychotherapy" prior to stabilization may exacerbate preexisting comorbid symptoms of depression and substance abuse. Promising work in these areas of comorbidity, such as Cognitive Behavior Therapy for Insomnia (CBT-I), has application in enhancing psychotherapy for PTSD (Schwartz and Carney 2012). Group-based psychotherapy is also commonly used, focusing on psychoeducation, anger, depression, substance use, social and vocational skills, relaxation training, as well as other facets of PTSD (APA 2004; Foy et al. 2000).

Pharmacological Management

Selective serotonin reuptake inhibitors (SSRIs) have the most empirical evidence for efficacy in the treatment of all four PTSD symptom clusters

and are usually considered as a first-line treatment for PTSD (APA 2004; National Institute for Clinical Excellence 2005; Schoenfeld et al. 2004). SSRIs are also effective agents for the treatment of comorbid mood and anxiety disorders commonly associated with PTSD. Both paroxetine and sertraline have received FDA approval for the treatment of PTSD in the United States (APA 2004). In Canada, only paroxetine has Health Canada approval for the treatment of PTSD.

Second-generation, dual-acting antidepressants such as venlafaxine and mirtazepine are widely used in treating major depression and other anxiety disorders but have less empirical data demonstrating their efficacy for the specific treatment of PTSD (Chung et al. 2004; Connor et al. 1999; Davidson et al. 2003; Hopwood et al. 2000; Smajkic et al. 2001). As such, they are often considered as a second-line treatment in patients who have failed to respond to a trial of an SSRI. However, since SSRIs have not demonstrated their efficacy in the treatment of Vietnam or combat-related PTSD thus far (Friedman et al. 2007; Schoenfeld et al. 2004), second-generation antidepressants may be considered as first-line treatment. Tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs) have some limited data to support their use in the treatment of combat-related PTSD (Davidson et al. 1990; Kosten et al. 1991). However, they are not commonly used because of their side effect profile and potential for toxicity.

Benzodiazepines are not recommended as monotherapy for the treatment of PTSD (Friedman 2006) but are sometimes used as adjuncts in treating anxiety or insomnia (APA 2004). There is a risk of rebound insomnia when a benzodiazepine, used as a hypnotic, is discontinued especially after long-term use (Cooper et al. 2005). The revised Veterans Affairs/Department of Defense Clinical Practice Guidelines advise against the use of mood stabilizers such as topiramate, divalproex, and lamotrigine in the treatment of PTSD (US Department of Veterans Affairs/Department of Defense 2017). They also highlight that atypical antipsychotics are not recommended for the treatment of core PTSD symptoms; rather, they may be used for comorbid disorders.

See Also

- ▶ Antianxiety Medication
- ▶ Antidepressant Medications
- ▶ Antidepressants
- ▶ Anxiety
- ▶ Anxiety Disorders
- ▶ Anxiolytics
- ▶ Psychotherapy

References and Reading

- Altemus, M., Cloitre, M., & Dhabhar, F. S. (2003). Enhanced cellular immune response in women with PTSD related to childhood abuse. *American Journal of Psychiatry*, 160, 1705–1707. Retrieved 1 Sept 2003, from <http://ajp.psychiatryonline.org/cgi/content/abstract/160/9/1705>.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2001). *Diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2004). Practice guidelines for the treatment of patients with acute stress disorder and posttraumatic stress disorder. *American Journal of Psychiatry*, 161(Suppl. 11), 1–57.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.).
- Bean, R. C., Ong, C. W., & Twohig, M. P. (2017). Acceptance and Commitment Therapy for PTSD and trauma: An empirical review. *The Behavior Therapist*, 40, 145–150.
- Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Gusman, F. D., Charney, D. S., et al. (1995). The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress*, 8, 75–90.
- Bonne, O., Brandes, D., Gilboa, A., Gomori, J. M., Shenton, M. E., Pitman, R. K., et al. (2001). Longitudinal MRI study of hippocampal volume in trauma survivors with PTSD. *American Journal of Psychiatry*, 158, 1248–1251. Retrieved 1 Aug 2001, from <http://ajp.psychiatryonline.org/cgi/content/abstract/158/8/1248>.
- Bremner, J. D., Randall, P., Scott, T. M., Bronen, R. A., Seibyl, J. P., Southwick, S. M., et al. (1995). MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *American Journal of Psychiatry*, 152, 973–981. Retrieved 1 July 1995, from <http://ajp.psychiatryonline.org/cgi/content/abstract/152/7/973>.
- Bremner, J., Randall, P., Vermetten, E., Staib, L., Bronen, R. A., Mazure, C., et al. (1997). Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse: A preliminary report. *Biological Psychiatry*, 41, 23–32.
- Breslau, N., Kessler, R. C., Chilcoat, H. D., Schultz, L. R., Davis, G. C., & Andreski, P. (1998). Trauma and posttraumatic stress disorder in the community: The 1996 Detroit area survey of trauma. *Archives of General Psychiatry*, 55, 626–632.
- Breslau, N., Lucia, V. C., & Alvarado, G. F. (2006). Intelligence and other predisposing factors in exposure to trauma and posttraumatic stress disorder: A follow-up study at age 17 years. *Archives of General Psychiatry*, 63, 1238–1245.
- Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology*, 68(5), 748–766.
- Canadian Psychiatric Association. (2006). Posttraumatic stress disorder. In clinical practice guidelines management of anxiety disorders. *Canadian Journal of Psychiatry*, 51(Suppl. 2), 57–63.
- Chung, M. Y., Min, K. H., Jun, Y. J., Kim, S. S., Kim, W. C., & Jun, E. M. (2004). Efficacy and tolerability of mirtazapine and sertraline in Korean veterans with posttraumatic stress disorder: A randomized open label trial. *Human Psychopharmacology*, 19(7), 489–494.
- Connor, K. M., Davidson, J. R., Weisler, R. H., & Ahearn, E. (1999). A pilot study of mirtazapine in posttraumatic stress disorder. *International Clinical Psychopharmacology*, 14, 29–31.
- Cooper, J., Carty, J., & Creamer, M. (2005). Pharmacotherapy for posttraumatic stress disorder: Empirical review and clinical recommendations. *Australian and New Zealand Journal of Psychiatry*, 39(8), 674–682 (679).
- Davidson, J. R. T., & Van der Kolk, B. A. (1996). A pharmacological treatment of post-traumatic stress disorder. In B. Van der Kolk, A. McFarland, & L. Weisaeth (Eds.), *Traumatic stress: The effects of overwhelming experience and mind, body and society* (p. 521). New York: The Guilford Press.
- Davidson, J., Kudler, H., Smith, R., Mahorney, S. L., Lipper, S., Hammett, E., et al. (1990). Treatment of posttraumatic stress disorder with amitriptyline and placebo. *Archives of General Psychiatry*, 47, 259–266.
- Davidson, J. R., Weisler, R. H., Butterfield, M. I., Casat, C. D., Connor, K. M., Barnett, S., et al. (2003). Mirtazapine vs placebo in posttraumatic stress disorder: A pilot trial. *Biological Psychiatry*, 53, 188–191.
- Dohrenwend, B., Turner, J., Turse, N. A., Adams, B. G., Koenen, K. C., & Marshall, R. (2006). The psychological risks of Vietnam for U.S. veterans: A revisit with new data and methods. *Science*, 313(18), 979–982.
- Foa, E. B., Keane, T. M., & Friedman, M. J. (2000). Introduction. In E. Foa, T. Keane, & L. M. Friedman (Eds.), *Effective treatments for PTSD* (pp. 1–17). New York: The Guilford Press.
- Foy, W. F., Glynn, S. M., Schnurr, P. P., Jankowski, M. K., Wattenberg, M. S., Weiss, D. S., et al. (2000). Group therapy. In E. Foa, T. Keane, & M. Friedman (Eds.),

- Effective treatments for PTSD* (pp. 155–175). New York: The Guilford Press.
- Friedman, M. J. (2006). Posttraumatic stress disorder among military returnees from Afghanistan and Iraq. *American Journal of Psychiatry*, 163, 586–593. Retrieved 1 Apr 2006, from <http://ajp.psychiatryonline.org>.
- Friedman, M., Marmar, C., Baker, D. G., Sikes, C. R., & Farfel, G. M. (2007). Randomized, double-blind comparison of sertraline and placebo for posttraumatic stress disorder in a Department of Veterans Affairs setting. *Journal of Clinical Psychiatry*, 68(5), 711–720.
- Gradus, J. L., Qin, P., Lincoln, A. K., Miller, M., Lawler, E., Sørensen, H. T., et al. (2010). Posttraumatic stress disorder and completed suicide. *American Journal of Epidemiology*, 171(6), 721–727.
- Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (2012). *Acceptance and Commitment Therapy: The process and practice of mindful change* (2nd ed.). New York: Guilford Press.
- Herman, J. L. (1992). *Trauma and recovery: The aftermath of violence from domestic abuse to political terror*. New York: Basic Books.
- Hoge, C. W., Castro, C. A., Messer, S. C., McGurk, D., Cotting, D. I., & Koffman, R. L. (2004). Combat duty in Iraq and Afghanistan, mental health problems, and barriers to care. *New England Journal of Medicine*, 351(1), 13–22.
- Hopwood, M., Morris, P. L., Debenham, P., Bonwick, R., Parkin, I., Ignatiadis, S., et al. (2000). An open label trial of Venlafaxine in War veterans with chronic post traumatic stress disorder. *Australian and New Zealand Journal of Psychiatry*, 34, A31–A31.
- Iversen, A., van Staden, L., Hughes, J. H., Browne, T., Hull, L., Hall, J., et al. (2009). The prevalence of common mental disorders and PTSD in the UK military: Using data from a clinical interview-based study. *BMC Psychiatry*, 9(1), 68.
- Jakupcak, M., Roberts, L. J., Martell, C., Mulick, P., Michael, S., Reed, R., Balsam, K. F., Yoshimoto, D., & McFall, M. (2006). A pilot study of behavioral activation for veterans with posttraumatic stress disorder. *Journal of Traumatic Stress*, 19(3), 387–391.
- Jakupcak, M., Luterek, J., Hunt, S., Conybeare, D., & McFall, M. (2008). Posttraumatic stress and its relationship to physical health functioning in a sample of Iraq and Afghanistan war veterans seeking post-deployment VA health care. *Journal of Nervous and Mental Disease*, 196(5), 425–428.
- Jakupcak, M., Wagner, A., Paulson, A., Varra, A., & McFall, M. (2010). Behavioral activation as a primary care-based treatment for PTSD and depression among returning veterans. *Journal of Traumatic Stress*, 23(4), 491–495.
- Jetly, R., & Cooper, K. (2008). PTSD The symptoms and how to treat. *Canadian Journal of Continuing Medical Education*, 20(2), 43–46.
- Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, 52, 1048–1060.
- Koren, D., Norman, D., Cohen, A., Berman, J., & Klein, E. M. (2005). Increased PTSD risk with combat-related injury: A matched comparison study of injured and uninjured soldiers experiencing the same combat events. *American Journal of Psychiatry*, 162, 276–228. Retrieved 1 Feb 2005, from <http://ajp.psychiatryonline.org/cgi/content/abstract/162/2/276>.
- Kosten, T. R., Frank, J. B., Dan, E., McDougale, C. J., & Giller, E. L., Jr. (1991). Pharmacotherapy for post-traumatic stress disorder using phenelzine or imipramine. *Journal of Nervous and Mental Disease*, 179, 366–370.
- Martell, C. R., Dimidjian, S., & Herman-Dunn, R. (2010). *Behavioral activation for depression*. New York: Guilford Press.
- National Institute for Clinical Excellence. (2005). *Post-traumatic stress disorder (PTSD): The management of PTSD in adults and children in primary and secondary care* (NICE Clinical Guideline No. 26). London: National Institute for Clinical Excellence.
- Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Bulletin*, 129(1), 52–73.
- Pagura, J., Stein, M. B., Bolton, J. M., Cox, B. J., Grant, B., & Sareen, J. (2010). Comorbidity of borderline personality disorder and posttraumatic stress disorder in the US population. *Journal of Psychiatric Research*, 44(16), 1190–1198.
- Raison, C. L., & Miller, A. H. (2003). When not enough is too much: The role of insufficient glucocorticoid signaling in the pathophysiology of stress-related disorders. *American Journal of Psychiatry*, 160, 1554–1565. Retrieved 1 Sept 2003, from <http://ajp.psychiatryonline.org/cgi/content/abstract/160/9/1554>.
- Resick, P. A., & Schnicke, M. K. (1993). *Cognitive processing therapy for rape victims: A treatment manual*. Newbury Park: Sage Publications.
- Richardson, J. D., Elhai, J. D., & Pedlar, D. J. (2006). Association of PTSD and depression with medical and specialist care utilization in modern peacekeeping veterans in Canada with health-related disabilities. *Journal of Clinical Psychiatry*, 67, 1240–1245.
- Richardson, J. D., Naifeh, J. A., & Elhai, J. D. (2007). Posttraumatic stress disorder and associated risk factors in Canadian peacekeeping veterans with health-related disabilities. *Canadian Journal of Psychiatry*, 52(8), 510–518.
- Sareen, J., Stein, M., Cox, B. J., & Hassard, S. T. (2004). Understanding comorbidity of anxiety disorders and antisocial behavior: Findings from two large community surveys. *Journal of Nervous and Mental Disease*, 192, 178–186.
- Sareen, J., Cox, B., Clara, I., & Asmundson, G. J. (2005). The relationship between anxiety disorders and physical disorders in the U.S. National Comorbidity Survey. *Depression and Anxiety*, 21, 193–202.
- Sareen, J., Cox, B. J., Stein, M. B., Afifi, T. O., Fleet, C., & Asmundson, G. J. (2007). Physical and mental comorbidity, disability, and suicidal behavior associated with

- posttraumatic stress disorder in a large community sample. *Psychosomatic Medicine*, 69(3), 242–248.
- Schoenfeld, F. B., Marmar, C. R., & Neylan, T. C. (2004). Current concepts in pharmacotherapy for posttraumatic stress disorder. *Psychiatric Services*, 55, 519–531. Retrieved 1 May 2004, from <http://ps.psychiatryonline.org/cgi/content/abstract/55/5/519>.
- Schwartz, D., & Carney, C. E. (2012). Mediators of Cognitive-Behavioral Therapy for Insomnia: A Review of Randomized Controlled Trials and Secondary Analysis Studies. *Clinical Psychology Review*, 32(7), 664–675.
- Smajkic, A., Weine, S., Djuric-Bijedic, Z., Boskailo, E., Lewis, J., & Pavkovic, I. (2001). Sertraline, paroxetine, and venlafaxine in refugee posttraumatic stress disorder with depression symptoms. *Journal of Traumatic Stress*, 14, 445–452.
- Southwick, S., Paige, S., Morgan, C. A., 3rd, Bremner, J. D., Krystal, J. H., & Charney, D. S. (1999). Neurotransmitter alterations in PTSD: Catecholamines and serotonin. *Seminars in Clinical Neuropsychiatry*, 4, 242–248.
- Staab, J. (2009). *Posttraumatic stress disorder*. Philadelphia: Physicians' Information and Education Resource, American College of Physicians Observer, from <http://pier.acponline.org/physicians/public/d251/diagnosis/d251-s3.html>.
- Statistics Canada. (2002). *Canadian community health survey cycle 12 – Mental health and well-being (Canadian forces supplement)*. Ottawa: Statistics Canada.
- Stein, M. B., Koverola, C., Hanna, C., Torchia, M. G., & McClarty, B. (1997). Hippocampal volume in women victimized by childhood sexual abuse. *Psychological Medicine*, 27(4), 951–959.
- Turnbull, G., & McFarland, A. (1996). Acute treatments. In B. A. Van der Kolk, A. C. McFarland, & L. Weisaeth (Eds.), *Traumatic stress* (pp. 480–490). New York: The Guilford Press.
- US Department of Veterans Affairs/Department of Defense. (2010). *Clinical practice guideline for management of post-traumatic stress*. Washington, DC: Author.
- US Department of Veterans Affairs/Department of Defense. (2017). *Clinical practice guideline for management of post-traumatic stress*. Washington, DC: Author.
- Van Ameringen, M. A. (2003). The prevalence of PTSD in Canada. In *American Psychiatric Association Annual Meeting*. San Francisco: CA.
- Van der Kolk, B. A., McFarland, A., & Van der Hart, O. (1996). A general approach to treatment of post-traumatic stress disorder. In B. A. Van der Kolk, A. McFarland, & L. Weisaeth (Eds.), *Traumatic stress* (pp. 417–440). New York: The Guilford Press.
- Weathers, F. W., Litz, B. T., Herman, D., Huska, J., & Keane, T. (1993). The PTSD checklist: Reliability, validity, & diagnostic utility. In *Annual Meeting of the International Society for Traumatic Stress Studies*. San Antonio: International Society for Traumatic Stress Studies.
- Yehuda, R., Southwick, S. M., Krystal, J. H., Bremner, D., Charney, D. S., & Mason, J. W. (1993). Enhanced suppression of cortisol following dexamethasone administration in posttraumatic stress disorder. *American Journal of Psychiatry*, 150, 83–86.
- Yehuda, R., McFarlane, A. C., & Shalev, A. Y. (1998). Predicting the development of posttraumatic stress disorder from the acute response to a traumatic event. *Biological Psychiatry*, 44, 1305–1313.
- Yehuda, R., Golier, J. A., Halligan, S. L., Meaney, M., & Bierer, L. M. (2004). The ACTH response to dexamethasone in PTSD. *American Journal of Psychiatry*, 161, 1397–1403. Retrieved 1 Aug 2004, from <http://ajp.psychiatryonline.org/cgi/content/abstract/161/8/1397>.

Postural Control Assessment

► Posturography

Posturography

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Synonyms

Postural control assessment; Standing balance assessment

Definition

Posturography is the study of an individual's control of their upright body position and balance in standing. It typically includes the measurement of body alignment while standing quietly, or in response to visual, postural, or environmental perturbation, or while performing dynamic

tasks. Postural control in standing relies on the integration of multisensory information from the vestibular system, vision, and somatosensory information including cutaneous sensation (foot pressure against the floor) and proprioception (joint position sense). Posturography commonly includes conditions to test the function of these systems, such as closing the eyes, introducing conflicting visual information, or altering the angle of the support surface. This assesses the relative contribution and integration of these systems to regulate balance. The ability to maintain smooth control of an upright body position in standing is an essential part of everyday life and needed to successfully manage everyday tasks such as reaching into cupboards, bending down, or catching balls in standing.

Balance control in standing improves as developmental maturation occurs across early to middle childhood. Compared to age-matched peers, children with autism have been found to have reduced stability in standing, reflected by greater postural sway, especially in the sideways (mediolateral) direction. This has been shown in studies of children with low intelligence (Kohen-Raz et al. 1992) and normal intelligence (Chang et al. 2010; Minshew et al. 2004; Molloy et al. 2003). Postural deficits have been shown in both adults and children with autism, but the differences are larger in children (Minshew et al. 2004), suggesting that the development of the postural system in children with autism may be delayed (Fournier et al. 2010; Minshew et al. 2004). Children with autism may thus appear less stable or coordinated in standing activities than their age-matched peers.

These deficits in postural control may be due to difficulty in the integration of multiple sensory input or adaptation to altered sensory input. Individuals with autism have greater difficulty with standing with eyes closed (Gepner et al. 1995; Minshew et al. 2004; Molloy et al. 2003), or in conditions where the standing surface is angled (referenced to sway), or vision is altered (Minshew et al. 2004). This suggests that these children may have more difficulty with challenging balance activities in some outdoor situations where the ground may be soft or compliant, the level of light fluctuating, or when vision is

occupied by tracking the motion of balls or people. Children with impaired balance control in standing are likely to have comparable or greater difficulties with walking and running activities, where the demand for rapid and efficient sensory-motor integration is greater. There are currently no empirical studies which have investigated the functional significance of difficulties in standing balance; however, clinically, it is suspected that this may impact negatively on children's proficiency and ability in school and recreational physical activities such as sport.

References and Reading

- Chang, C., Wade, M., Stoffregen, T., Hsu, C., & Pan, C. (2010). Visual tasks and postural sway in children with and without autism spectrum disorders. *Research in Developmental Disabilities*, 31(6), 1536–1542.
- Fournier, K., Kimberg, C., Radonovich, K., Tillman, M., Chow, J., Lewis, M., et al. (2010). Decreased static and dynamic postural control in children with autism spectrum disorders. *Gait & Posture*, 32(1), 6–9.
- Gepner, B., Mestre, D., Masson, G., & de Schonen, S. (1995). Postural effects of motion vision in young autistic children. *Neuroreport*, 6(8), 1211–1214.
- Kohen-Raz, R., Volkmar, F., & Cohen, D. (1992). Postural control in children with autism. *Journal of Autism and Developmental Disorders*, 22(3), 419–432.
- Minshew, N., Sung, K., Jones, B., & Furman, J. (2004). Underdevelopment of the postural control system in autism. *Neurology*, 63(11), 2056–2061.
- Molloy, C., Dietrich, K. N., & Bhattacharya, A. (2003). Postural stability in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 33(6), 643–652.

Power of Attorney (Financial and Property Management)

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Definition

A Power of Attorney (POA) is a legal agreement that grants an agent the authority to act in the place

of a principal in matters of finances and property (American Law Institute 2006). Under a POA instrument, a principal authorizes an agent to perform a particular set of duties for the principal's benefit. A POA agent (historically referred to as an "attorney-in-fact") must act according to the terms and specifications set forth in the POA record (Garner 2019a).

A POA should be distinguished from a "Healthcare Power of Attorney," which is typically a separate and distinct legal instrument that grants an agent the specific authority to make decisions about the principal's healthcare and medical treatment (Nicolaidis et al. 2016).

Principles of a Power of Attorney

A POA can be a useful special needs planning tool for individuals with autism spectrum disorder (ASD) and their caregivers (Sharpe and Baker 2007). Establishing a POA can be beneficial in a variety of contexts, including: (1) personal convenience in managing financial and property affairs; (2) providing additional security in the event of an unexpected injury or other health condition; or (3) assisting a principal who has a progressively worsening health condition or legal incapacity (American Bar Association 2020). Unlike a legal guardianship or conservatorship, a POA appointment does not require a court order from a judge to be valid. Depending on a principal's individual circumstances, utilizing a POA agent may be preferable over a court-appointed conservator or guardian (Sitkoff and Dukeminier 2017). Because POAs do not require court orders for amendments, they can usually be swiftly revised or terminated at a principal's discretion.

Several nonprofit and for-profit organizations offer state-specific forms for POAs online; however, it is a best practice to consult with an attorney who is licensed in your state of residence when executing a POA (U.S. Government Services and Information 2020). As the National Council on Disability explains, "Although forms are often available online, powers of attorney are powerful documents that require careful consideration by the principal, who may benefit from a lawyer's advice and drafting experience, particularly in the area of

finances." (National Council on Disability 2018). Hiring an attorney to write your POA will ensure that your POA is valid in your home state and transferrable across state lines. It is strongly recommended to utilize an attorney to make certain that the terms of your POA for finances and property align with the specifications of your other legal documents and special needs planning instruments. The goals listed in your POA should coordinate with other long and short-term goals set forth in items such as your advanced medical directives, living will, special needs trust, and/or ABLE account (also referred to as a "529A account") (Ho and Lee 2019).

In some states, parents with children age 17 and younger may choose to enact a Minor POA document (also known as a "Child Power of Attorney"). This type of POA allows a parent to grant control over the decisions made for their child for a specified period of time, often between 6 and 12 months (e.g., Power of Attorney for Care of a Minor Child Act of 2003). A Minor POA may be used in situations where a parent is separated from and/or unable to assist the child because of employment-related travel, military service, incarceration, or a health condition.

Power of Attorney Authority and Timeframes

With a POA in place, an agent can take any action on behalf of a principal that is permitted by the POA agreement's language. The terms within a POA confirm both the scope of an agent's authority and the POA's timeframe. There are two primary types of POAs that differ according to the scope of the agent's authority: (1) General POA (granting extensive authority), and (2) Limited POA (also referred to as "Special Power of Attorney," granting specific authority) (American Jurisprudence 2020). See Table 1 "Agent Authority under a Power of Attorney." A General POA empowers an agent to make a broad range of business decisions – these may include signing checks to pay bills, managing all real estate property, and arranging financial affairs (*Zaubler v. Picone* 1984). Under a Limited POA agreement,

Power of Attorney (Financial and Property Management), Table 1 Scope of an agent’s authority under a power of attorney

Agent’s authority type	Agent’s scope of authority	Examples of tasks assigned to an agent
General power of attorney	An agent has powers apply to broad groups of issues.	1. Sign checks to pay all bills 2. Manage all real estate property 3. Manage the principal’s pension, retirement, annuity, or other income 4. Communicate all health care directives 5. Open a financial account 6. Conduct business operations
Limited power of attorney (also known as special authority)	An agent has specific powers that are limited to a certain act or type of act.	1. Complete a home closing sale 2. Purchase an automobile 3. Open a bank account 4. Refinance a mortgage or student loan 5. File last year’s tax return 6. Purchase a specific stock or bond

the agent’s authority is specifically stated and applies to a set number of activities (*In re Grubin 2012*). An agent with limited authority might perform the following duties: complete a home closing sale, purchase an automobile, open a bank account, arrange affairs to refinance a loan, or file the principal’s taxes (Requirements for filing power of attorney [2020](#)).

Unless a principal specifies that his or her POA should become effective upon a future date, event, or contingency, an agent’s authority under a POA is considered effective when the principal signs the POA document. There are four types of

Power of Attorney (Financial and Property Management), Table 2 Power of attorney timeframes

Length type	Definition
Permanent	Empowers the agent to act on the principal’s behalf throughout the principal’s lifetime and until the principal’s death.
Temporary	Empowers the agent to act on the principal’s behalf for limited time frame.
Springing	Empowers the agent to act on the principal’s behalf only in the event of a particular occurrence. This type of POA term can be designed as either permanent or temporary in scope. Without a triggering occasion, a springing power of attorney will never take effect.
Durable	Empowers the agent to act on the principal’s behalf after a principal becomes legally incapacitated. This type of POA term can be designed to be permanent, temporary, and/or springing in scope.

timeframes for a POA: permanent, temporary, springing, and durable (Andersen and Gary [2018](#)). See [Table 2](#) “Power of Attorney Timeframes.” A permanent term POA empowers the agent throughout the principal’s lifetime. A temporary term POA is written to last for a definite period of time; it can be written to take effect immediately or begin on a specified date. In contrast, a springing term POA will begin only upon the occurrence of particular future event. For example, a POA with springing terminology can be designed to take effect if a physician determines that the principal is unable to care for himself or herself because of an unforeseen accident or a decline in functional status. Lastly, a durable term POA empowers the agent to act on the principal’s behalf after a principal becomes “legally incapacitated” (Garner [2019b](#)). Depending on the principal’s preferences, a POA with a durable designation can be written as permanent, temporary, and/or springing in scope. In the majority of states, a POA is considered to be durable unless it explicitly provides that it is terminated by the legal incapacity of the principal. In a nondurable POA term, the agent’s authority will stop in the event the principal becomes legally incapacitated.

Creating a Power of Attorney

In the USA, POA agreements are largely governed by the probate laws of the individual states. The requirements for a valid POA can differ greatly among state laws. In most states, four primary elements are required to create a valid POA – the principal must: (1) be a legal adult, which is age 18 or older in most states; (2) have “legal capacity”; (3) sign the POA (or, in the event the principal is unable to make a signature, direct another individual to sign the principal’s name in the principal’s presence); and (4) ensure that at least one individual witnessed the signing of the POA, preferably a notary public (U.S. Consumer Financial Protection Bureau 2016). Though a notarization of the principal’s signature is not typically required to create a valid POA, it is a best practice to include a notary public’s authorization on all POAs to confirm the document authenticity. See Table 3 “Key Elements for a Power of Attorney.”

Ideally, a principal should appoint at least two individuals as agents and confirm the order of their authority. With a primary principal and a secondary individual in reserve, the latter will only receive authority under a POA in the event the primary principal is unavailable to serve the as the agent. It is also a best practice to provide copies of your POA document to friends, family members, or others who may have a need to know about its details. In some states, principals are required to file a record of their POA with the recorder’s office in their home county (also sometimes referred to as the “county clerk”).

Legal Capacity in Power of Attorney Matters

In the USA, the doctrine of “legal capacity” applies to the principal’s state of being when signing the POA agreement. For purposes of entering into a POA, a principal’s legal capacity is defined as the ability to understand the fundamental benefits, risks, and effect of entering into the agreement (Parker 2016). Historically, the term legal capacity was sometimes referred to as

Power of Attorney (Financial and Property Management), Table 3 Key Elements for a power of attorney

Element number	Element	Description
1	The principal must be a legal adult.	Age 18 is the legal threshold for adulthood in most states.
2	The principal does not have “legal capacity.”	Individuals lack legal capacity when they are unable to manage their property or business affairs because of the following: (1) they cannot receive and evaluate information; and/or (2) they cannot make or communicate decisions even with the use of technological assistance.
3	The principal must sign the POA agreement.	In the event the principal is unable to sign the POA agreement, another individual may sign on the principal’s behalf provided that the signer is acting on the principal’s direction and in the principal’s presence.
4	Other individuals witness the principal’s signature of the POA agreement	Witnesses serve to confirm and acknowledge the authenticity of the POA. As a best practice, a POA agreement’s signature should be certified by a notary public.

“legal competency.” Individuals do not have legal capacity when a judge rules that they are entirely incapable of managing financial or personal affairs. In these instances, the individual is unable to fully safeguard himself or herself against harm to self, wealth, and/or property. A judge will determine that a principal lacks legal capacity

based on evidence presented to the court, including medical records and testimony from treating physicians, mental health professionals, caregivers, family members, friends, and the individual under review for a legal capacity judgment (United States General Accounting Office 2004). As part of its guidance on best practices for state POA legislation and policy, the U.S. Uniform Law Commission offers the following clarification regarding legal capacity issues: “[I]n recognition that disability does not necessarily render an individual incapable of property and business management. . .definition of incapacity stresses the operative consequences of the individual’s impairment—inability to manage property and business affairs—rather than the impairment itself” (Uniform Law Commission 2006).

A principal’s legal capacity must be distinguished from his or her mental ability (Dudley and Edmonds 2018). Legal capacity embodies individual legal rights to have standing to bring and defend lawsuits and enter into/execute legally binding contracts. In contrast, mental or cognitive capacity issues in conservatorship matters focus on a person’s ability to express personal will and intentions (Kohn et al. 2013). Even in cases where the principal has been declared as lacking legal capacity, state laws usually require POA agents to take a principal’s preferences into account when making life-changing decisions about finances and property (National Council on Disability 2019).

Ending a Power of Attorney

There are six common reasons why a POA will end: (1) the principal passes away; (2) the principal chooses to revoke the POA; (3) the principal becomes legally incapacitated (and the POA is not designated as “durable” in length); (4) a court determines that the POA is invalid; (5) the agent can no longer carry out the required responsibilities; and (6) an agent has developed a conflict of interest that interferes with his or her obligations to the principal (U.S. Consumer Financial Protection Bureau 2015). See Table 4 “Common Reasons for Ending a Power of Attorney Agreement.”

Power of Attorney (Financial and Property Management), Table 4 Six common reasons for ending a power of attorney agreement

Reason number	Justification for invalidation, revocation, or termination
1	The principal passes away.
2	The principal chooses to revoke the POA agreement.
3	The principal becomes legally incapacitated and the POA agreement is explicitly designated as “nondurable.”
4	A court rules that the POA agreement is invalid due to fraud, the principal’s lack of legal capacity when entering into the agreement, or other fairness concerns.
5	The agent can no longer carry out the required responsibilities.
6	The agent has developed a conflict of interest that interferes with his or her obligations to the principal.

When POAs are challenged in court, they are often overturned based on the following considerations: a judge declares the POA as invalid based on evidence that the principal did not have legal capacity when entering into the POA; the agent has developed a conflict of interest against the principal; or some other fraud (also known as “misrepresentation”) occurred to induce the principal to enter into the POA.

In most jurisdictions, a principal can terminate a POA at any time and at his or her discretion. All types of POAs end after the principal passes away. A former POA agent can continue to act as the principal’s legal representative when he or she is also designated as the executor of the principal’s estate (Furrow et al. 2018). A POA agent will maintain the authority to pay the principal’s debts, manage estate affairs, and make funeral arrangements after the principal is deceased only when a principal appoints the same individual (or organization) as both POA agent and estate executor.

See Also

- [Conservatorship \(Full Conservatorship and Limited Conservatorship\)](#)
- [Guardianship](#)

- **Power of Attorney for Healthcare (Durable Healthcare Power of Attorney and Medical Power of Attorney)**
- **Special Needs Planning (SNP)**

References and Reading

- 3 Am. Jur. 2d § 20 *Powers of attorney: In general* (2020). American Bar Association. (2020). Estate planning info & FAQs: Power of Attorney. Retrieved from https://www.americanbar.org/groups/real_property_trust_estate/resources/estate_planning/power_of_attorney/
- American Law Institute. (2006). Restatement (third) of agency. Terminology § 1.04(7).
- Andersen, R., & Gary, S. (2018). *Understanding trusts and estates* (6th ed.). Durham: Carolina Academic Press.
- Dudley, K., & Edmonds, N. (2018). Psychology's role in guardianship and conservatorship evaluations. In S. S. Bush & A. L. Heck (Eds.), *Forensic geropsychology: Practice essentials* (pp. 257–279). American Psychological Association. <https://doi.org/10.1037/0000082-013>.
- Furrow, B. R., Greaney, T. L., Johnson, S. H., & Stoltzfus Jost, T. (2018). *Health law: Cases, materials and problems* (8th ed.). St. Paul, MN: West Academic Publishing.
- Garner, B. (Ed.). (2019a). *Power of attorney. Black's law dictionary* (11th ed.). St. Paul: Thomson/West.
- Garner, B. (Ed.). (2019b). *Incapacity. Black's law dictionary* (11th ed.). St. Paul: Thomson/West.
- Ho, L., & Lee, R. (Eds.). (2019). *Special needs financial planning: A comparative perspective*. Cambridge, UK/New York: Cambridge University Press.
- In re Grubin*, 476 B.R. 699, 707 (Bankr. E.D.N.Y. 2012).
- Kohn, N., Blumenthal, J., & Campbell, A. (2013). Supported decision-making: A viable alternative to guardianship. *Penn State Law Review*, 117(4), 1111. Retrieved from [http://www.pennstatelawreview.org/117/4/%20Final/4-Kohn%20et%20al.%20\(final\)%20\(rev2\).pdf](http://www.pennstatelawreview.org/117/4/%20Final/4-Kohn%20et%20al.%20(final)%20(rev2).pdf).
- National Council on Disability. (2018). Beyond guardianship: toward alternatives that promote greater self-determination. 145–152. Retrieved from <https://ncd.gov/publications/2018/beyond-guardianship-toward-alternatives>
- National Council on Disability. (2019). Turning rights into reality: How guardianship and alternatives impact the autonomy of people with intellectual and developmental disabilities. Retrieved from <https://ncd.gov/publications/2019/turning-rights-into-reality>
- Nicolaidis, C., et al. (2016). The development and evaluation of an online healthcare toolkit for autistic adults and their primary care providers. *Journal of General Internal Medicine*, 31(10), 1180–1189. <https://doi.org/10.1007/s11606-016-3763-6>.
- Parker, M. (2016). Getting the balance right: Conceptual considerations concerning legal capacity and supported decision-making. *Journal of Bioethical Inquiry*, 13(3), 381–393. <https://doi.org/10.1007/s11673-016-9727-z>.
- Requirements for filing power of attorney, 26 C.F.R. § 601.504 (2020).
- Sharpe, D. L., & Baker, D. L. (2007). Financial issues associated with having a child with autism. *Journal of Family and Economic Issues*, 28(2), 247–264. <https://doi.org/10.1007/s10834-007-9059-6>.
- Sitkoff, R. H., & Dukeminier, J. (2017). *Wills, trusts, and estates*. New York, NY: Wolters Kluwer Law & Business.
- U.S. Consumer Financial Protection Bureau. (2015). Managing someone else's money: Help for agents under a power of attorney. Retrieved from https://files.consumerfinance.gov/f/201310_cfpb_lay_fiduciary_guides_agents.pdf
- U.S. Consumer Financial Protection Bureau. (2016). What is a Power of Attorney (POA). Retrieved from <https://www.consumerfinance.gov/ask-cfpb/what-is-a-power-of-attorney-poa-en-1149/>
- U.S. General Accounting Office. (2004). Guardianships: collaboration needed to protect incapacitated elderly people. GAO-04-655. Retrieved from <https://www.gao.gov/products/GAO-04-655>
- U.S. Government Services and Information. (2020). Family legal issues: Power of attorney. Retrieved from <https://www.usa.gov/family-legal/item-213778>
- Uniform Law Commission. (2006). Uniform Power of Attorney Act. Retrieved from <https://www.uniformlaws.org/committees/community-home?CommunityKey=b1975254-8370-4a7c-947f-e5af0d6cb07c>
- Zaubler v. Picone*, 100 A.D.2d 620, 473 N.Y.S.2d 580 (N.Y. App. Div. 1984).

Power of Attorney for Healthcare (Durable Healthcare Power of Attorney and Medical Power of Attorney)

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Defined

A power of attorney for healthcare (also referred to as a healthcare power of attorney and hereafter as “HCPOA”) is a legal advance directive that grants an agent the authority to make decisions about the principal’s healthcare and medical treatment. Under a HCPOA instrument, a principal

authorizes an agent to perform a particular set of duties for the principal's benefit. A HCPOA agent (historically referred to as an "attorney-in-fact") must act according to the terms and specifications set forth in the HCPOA record (Garner 2019a).

HCPOA documents that are labeled as "durable" do not end in the event the principal becomes legally incapacitated (U.S. Department of Health & Human Services National Institute on Aging 2016). Agents under HCPOA agreements are often referred to as "patient advocates" because they are charged with understanding and carrying out principals' care preferences (Sabatino 2010). A HCPOA can be designed to be permanent or limited based on the principal's needs. For example, a HCPOA can be designed to commence only in the event the principal is unconscious or otherwise unable to communicate healthcare directives (Marks et al. 2019).

A HCPOA should be distinguished from a power of attorney (POA) for estate planning, which is a legal agreement that grants an agent the authority to act in the place of a principal in matters of finances and property (American Law Institute 2006).

Common Abbreviations and Acronyms in Healthcare Power of Attorney Matters

HCPOAs have many possible synonyms and abbreviations including medical power of attorney (MPOA), power of attorney for healthcare (POA-HC, POAHC, or alternatively HPOA), and advanced directive (AD). Because the majority of HCPOA documents are written to be "durable" in nature, these instruments are also commonly referred to as a durable healthcare power of attorney (DPOA-HC or DPOAHC) or durable power of attorney for healthcare (DHCPOA). Traditionally, a HCPOA agent was referred to as an "attorney in fact." Today, an agent acting under a HCPOA is more commonly referred to as a patient advocate, healthcare agent (HCA), healthcare proxy (HCP), healthcare surrogate, or surrogate decision-maker (e.g., Meisel 2015).

Principles of a Healthcare Power of Attorney

A HCPOA can be a useful special needs planning tool for individuals with autism spectrum disorder (ASD) and their caregivers (Satkoske et al. 2020). Establishing a HCPOA can be beneficial in a variety of contexts, including (1) personal convenience in managing health and personal wellness affairs; (2) providing additional security in the event of an unexpected injury or other health condition; or (3) assisting a principal who has a progressively worsening health condition or legal incapacity (American Bar Association 2011). Unlike a legal guardianship or conservatorship, a HCPOA appointment does not require a court order from a judge to be valid. Depending on a principal's individual circumstances, utilizing a HCPOA patient advocate may be preferable over a court-appointed conservator or guardian (U.S. National Council on Disability 2018). In most cases, HCPOAs can be revised or terminated quickly and conveniently at a principal's discretion. As a best practice, a principal should confirm his or her revocation of a HCPOA in a signed, notarized document. In limited circumstances – often when the wording in a HCPOA is unclear and a principal is unable to communicate his or her intentions – a judge will review a HCPOA and amend it with a court order.

Several nonprofit and for-profit organizations offer state-specific forms for HCPOAs online (e.g., Regents of the University of Michigan 2017). State-specific HCPOA forms are often available at no cost to the public online through state governments, state bar associations, or state medical societies (e.g., State of Illinois Power of Attorney Act Illinois Statutory Short Form: Power of Attorney for Healthcare, 2020 and State of Iowa Durable Power of Attorney for Healthcare Form 2020). Free, standardized forms are often limited in nature and provide a range of check-off options to try to suit a principal's needs – these lists can sometimes fail to fully communicate a principal's unique preferences and needs. HCPOA forms for the general public range in quality and is typically less comprehensive than individualized documents prepared after a consultation with a private attorney who specializes in special needs planning.

It is a best practice to consult with an attorney who is licensed in your state of residence when executing a HCPOA (U.S. Government Services and Information 2020). Hiring an attorney to write your HCPOA or review an existing document will ensure that your HCPOA is valid in your home state and transferrable across state lines. It is strongly recommended to utilize an attorney to make certain that the terms of your HCPOA align with the specifications of your other legal documents and special needs planning instruments. The HCPOA patient advocate should take care to understand the principal's entire special needs plan. The goals listed in your HCPOA should coordinate with other long- and short-term goals and spending priorities set forth in items such as your power of attorney for finance and property, special needs trust, and/or ABLE account (also referred to as a "529A account") (Ho and Lee 2019).

A HCPOA's instructions should be updated regularly and include wording that is detailed and specific. There are two primary reasons why HCPOA forms are sometimes found to be impractical or unhelpful: (1) the HCPOA language intended to guide the patient advocate is outdated and no longer relevant to the principal's current situation; and (2) the principal's directions for his or her patient advocate are too vague and ambiguous (e.g., subject to multiple reasonable interpretations) to provide a patient advocate with meaningful guidance on making tough decisions (Graber 2017). All in all, the overarching goal of a HCPOA is to clarify a principal's values and treatment preferences so that the patient advocate can work to implement those plans in collaboration with healthcare providers (Zerbo et al. 2015). Principals and their caregivers should review their HCPOA yearly to revisit whether certain values and treatment goals have changed in relation to their health and level of functioning.

Healthcare Power of Attorney Authority and Timeframes

With a HCPOA in place, a patient advocate can take any action on behalf of a principal that is permitted by the HCPOA agreement's language

(Elster and Parsi 2020). Unless a document expressly states otherwise, a HCPOA covers any healthcare-related service or procedure. HCPOAs typically cover all decision-making to maintain, diagnose, treat, or otherwise affect an individual's physical or mental condition (e.g., U.S. Department of Health & Human Services National Institute on Aging 2016). In some states, the scope of a HCPOA can also include wellness and personal care services. The terms within a HCPOA will confirm both the scope of the patient advocate's authority and the HCPOA's timeframe. There are two primary types of HCPOAs that differ according to the scope of the patient advocate's authority: (1) general HCPOA (granting extensive authority) and (2) limited HCPOA (also referred to as "special power of attorney," granting specific authority) (American Bar Association 2011). See Table 1, Scope of a Patient Advocate's Authority under a Healthcare Power of Attorney.

Unless a principal clearly specifies that his or her HCPOA should become effective upon a future date, event, or contingency, a patient advocate's authority under a HCPOA is considered effective when the principal signs the HCPOA document. Most HCPOA documents are designed to be "durable" in nature, meaning they do not end in the event the principal becomes "legally incapacitated" (Garner 2019b). In most states, a HCPOA is automatically considered to be durable unless the document specifically states that the principal wants a nondurable HCPOA. Under a nondurable HCPOA, a patient advocate's authority will stop in the event the principal becomes legally incapacitated (Palmer and Harmell 2016). A person's legal capacity must always be distinguished from his or her ability to communicate desires and intentions (Mitchell 2015). Legal capacity can only be officially determined by a judge – it embodies an individual's legal rights to have standing to bring and defend lawsuits and enter into/execute legally binding contracts.

There are four types of timeframes for a HCPOA: permanent, temporary, springing, and durable (American Bar Association 2009). See Table 2, Healthcare Power of Attorney Timeframes. A permanent term HCPOA empowers the patient advocate throughout the

Power of Attorney for Healthcare (Durable Healthcare Power of Attorney and Medical Power of Attorney), Table 1 Scope of a Patient Advocate’s Authority under a Healthcare Power of Attorney

Patient advocate’s authority type	Patient advocate’s scope of authority	Examples of tasks assigned to a patient advocates
General power of attorney for healthcare	Patient advocate has powers apply to broad groups of issues	1. Make all healthcare-related decisions 2. Manage all medical appointments 3. Make arrangements for surgeries, procedures, and treatments 4. Communicate all healthcare directives
Limited power of attorney for healthcare (also known as special authority for healthcare)	Patient advocate has specific powers that are limited to a certain act or type of act	1. Make healthcare-related decisions for a specific surgery, procedure, or treatment 2. Manage medical appointments related to a mental health only 3. Make arrangements for only physical and occupational therapies 4. Communicate healthcare directives related to end-of-life matters only

principal’s lifetime. A temporary term HCPOA is written to last for a definite period of time; it can be written to take effect immediately or begin on a specified date. In contrast, a springing term HCPOA will begin only upon the occurrence of particular future event. For example, a HCPOA with springing terminology can be designed to

Power of Attorney for Healthcare (Durable Healthcare Power of Attorney and Medical Power of Attorney), Table 2 Healthcare Power of Attorney Timeframes

Length type	Definition
Permanent	Empowers the patient advocate to act on the principal’s behalf throughout the principal’s lifetime and until the principal’s death.
Temporary	Empowers the patient advocate to act on the principal’s behalf for limited timeframe.
Springing	Empowers the patient advocate to act on the principal’s behalf only in the event of a particular occurrence. This type of HCPOA term can be designed as either permanent or temporary in scope. Without a triggering occasion, a springing HCPOA will never take effect.
Durable	Empowers the patient advocate to act on the principal’s behalf after a principal becomes legally incapacitated. This type of HCPOA term can be designed to be permanent, temporary, and/or springing in scope.

take effect if a physician determines that the principal is unable to care for himself or herself because of an unforeseen accident or a decline in functional status.

Creating and Ending a Healthcare Power of Attorney

In the USA, HCPOA agreements are largely governed by the contract laws of the individual states. The requirements for a valid HCPOA can differ greatly among state laws (e.g., Uniform Law Commission 1993, 2014). In most state jurisdictions, four primary elements are required to create a valid HCPOA – the principal must (1) be a legal adult, which is age 18 or older in most states; (2) have legal capacity; (3) sign the HCPOA (or in the event the principal is unable to make a signature, direct another individual to sign the principal’s name in the principal’s presence); and (4) ensure that at least one individual witnessed the signing of the HCPOA, preferably a notary public (American Bar Association

(2018); see also Moyer et al. 2013). Though a notarization of the principal’s signature is not typically required to create a valid HCPOA, it is a best practice to include a notary public’s authorization on all HCPOAs to confirm the document

Power of Attorney for Healthcare (Durable Healthcare Power of Attorney and Medical Power of Attorney), Table 3 Key Elements for Creating a Healthcare Power of Attorney

Element number	Element	Description
1	The principal must be a legal adult to appoint a patient advocate.	Age 18 is the legal threshold for adulthood in most states.
2	The principal must have legal capacity to agree to the terms of the HCPOA.	Individuals lack legal capacity when they are unable to manage their property or business affairs because of the following: (1) they cannot receive and evaluate information; and/or (2) they cannot make or communicate decisions even with the use of technological assistance.
3	The principal must sign the HCPOA agreement.	In the event the principal is unable to sign the HCPOA agreement, another individual may sign on the principal’s behalf provided that the signer is acting on the principal’s direction and in the principal’s presence.
4	Other individuals witness the principal’s signature of the HCPOA agreement.	Witnesses serve to confirm and acknowledge the authenticity of the HCPOA. As a best practice, a HCPOA agreement’s signature should be certified by a notary public.

authenticity. See Table 3, Key Elements for Creating a Healthcare Power of Attorney.

Ideally, a principal should appoint at least two individuals as patient advocates and confirm the order of their authority. With a primary principal and a secondary advocate in reserve, the latter will only receive authority under a HCPOA in the event the primary principal is unavailable to serve as the patient advocate. It is also a best practice to provide copies of your HCPOA document to your primary physician, treating physicians, friends, family members, or others who need to know its details (Nicolaidis et al. 2016).

In some cases, a principal’s healthcare provider, family member, or another individual will challenge the validity of a HCPOA in court. A judge may decide to order that a HCPOA is invalid based for one of the following reasons: (1) the judge determines that sufficient evidence exists to prove that the principal did not have legal capacity when entering into the agreement; (2) the patient advocate has developed a conflict of interest against the principal; (3) the patient advocate can no longer carry out the responsibilities required under the HCPOA; or (4) or some other fraud (also known as “misrepresentation”) occurred to induce the principal to enter into the HCPOA (Sitkoff and Dukeminier 2017).

As a general rule, a principal can terminate a HCPOA at any time and at his or her discretion. In some states, a HCPOA will remain effective following death of the principal so that the patient advocate can process organ donations, autopsy matters, and burial or cremation services (e.g., General Statutes of North Carolina 32A-19(b), 2020). Apart from these end-of-life matters, a HCPOA ends after the principal passes away.

See Also

- ▶ [Achieving a Better Life Experience Savings Account \(ABLE Savings Account, ABLE Account, 529A Savings Plan, and 529A Account\)](#)
- ▶ [Conservatorship \(Full Conservatorship and Limited Conservatorship\)](#)

- Guardianship
- Letter of Intent
- Power of Attorney for Healthcare (Durable Healthcare Power of Attorney and Medical Power of Attorney)
- Special Needs Planning (SNP)

References and Reading

- American Bar Association. (2009). Making medical decisions for someone else: A how-to guide. Retrieved from https://www.americanbar.org/content/dam/aba/administrative/law_aging/2011_aging_bk_proxy_guide_gen.pdf.
- American Bar Association. (2011). Giving someone a power of attorney for your health care: A guide with an easy-to-use, legal form for all adults. Retrieved from https://www.americanbar.org/content/dam/aba/administrative/law_aging/2011_aging_hcdec_univhcpaform.pdf.
- American Bar Association. (2018). Health care decision making: Resources for professionals and consumers. Retrieved from https://www.americanbar.org/groups/law_aging/resources/health_care_decision_making/.
- American Law Institute. (2006). Restatement (Third) of Agency. Terminology § 1.04(7).
- Elster, N., & Parsi, K. (2020). Transitioning from adolescence to adulthood with autism spectrum disorder: An overview of planning and legal issues. *Child and Adolescent Psychiatric Clinics*, 29(2), 399–408. <https://doi.org/10.1016/j.chc.2019.11.003>.
- Garner, B. (Ed.). (2019a). *Power of attorney*. *Black's law dictionary* (11th ed.). St. Paul: Thomson/West.
- Garner, B. (Ed.). (2019b). *Incapacity*. *Black's law dictionary* (11th ed.). St. Paul: Thomson/West.
- Graber, A. (2017). Autism, intellectual disability, and a challenge to our understanding of proxy consent. *Medicine, Health Care and Philosophy*, 20(2), 229–236. <https://doi.org/10.1007/s11019-016-9745-y>.
- Ho, L., & Lee, R. (Eds.). (2019). *Special needs financial planning: A comparative perspective*. Cambridge, UK \New York: Cambridge University Press.
- Marks, S., et al. (2019). Surgery without a surrogate: The low prevalence of healthcare power of attorney documents among preoperative patients. *Hospital Practice*, 47(1), 28–33.
- Meisel, A. (2015). Antecedent law and ethics of aid in dying. *Quinnipiac Law Review*, 34, 609. Retrieved from <https://www.quinnipiaclawjournals.com/content/dam/qu/documents/sol/law-journals1/law-review/volume-34/consolidated-pdf/quinnipiac-law-review-volume-34-issue-4.pdf>.
- Mitchell, M. (2015). Assessing patient decision-making capacity: It's about the thought process. *Journal of Emergency Nursing*, 41(4), 307–312. <https://doi.org/10.1016/j.jen.2014.10.014>.
- Moye, J., Sabatino, C., & Brendel, W. R. (2013). Evaluation of the capacity to appoint a healthcare proxy. *The American Journal of Geriatric Psychiatry*, 21(4), 326–336. <https://doi.org/10.1016/j.jagp.2012.09.001>.
- Nicolaidis, C., et al. (2016). The development and evaluation of an online healthcare toolkit for autistic adults and their primary care providers. *Journal of General Internal Medicine*, 31(10), 1180–1189. <https://doi.org/10.1007/s11606-016-3763-6>.
- Palmer, B., & Harmell, A. (2016). Assessment of healthcare decision-making capacity. *Archives of Clinical Neuropsychology*, 31(6), 530–540. <https://doi.org/10.1093/arclin/acw051>.
- Regents of the University of Michigan. (2017). Start the conversation: Making your health care wishes known Advance Directives and Durable Power of Attorney for Health Care. Retrieved from <http://www.med.umich.edu/1libr/AdvanceDirectives/ADbooklet.pdf>.
- Sabatino, C. (2010). The evolution of health care advance planning law and policy. *The Milbank Quarterly*, 88(2), 211–239. <https://doi.org/10.1111/j.1468-0009.2010.00596.x>.
- Satkoske, V., Migyanka, J., & Kappel, D. (2020). Autism and advance directives: Determining capability and the use of health-care tools to aid in effective communication and decision-making. *American Journal of Hospice and Palliative Medicine*, 37(5), 354–363. <https://doi.org/10.1177/1049909119888621>.
- Sitkoff, R., & Dukeminier, J. (2017). *Wills, trusts, and estates*. New York: Wolters Kluwer Law & Business.
- State of Illinois Power of Attorney Act Illinois Statutory Short Form: Power of Attorney for Health Care codified at 755 Illinois Compiled Statutes § 45 (2020). Retrieved from <http://dph.illinois.gov/sites/default/files/forms/forms-legal-power-attorney-040716.pdf>.
- State of Iowa Durable Power of Attorney for Health Care Form codified at Iowa Code § 144B(5) (2020). Retrieved from <https://www.legis.iowa.gov/docs/code/144B.5.pdf>.
- State of North Carolina Powers of Attorney: Extent of Authority; Limitations of Authority codified at General Statutes of North Carolina 32A-19(b) (2020). Retrieved from https://www.ncleg.gov/EnactedLegislation/Statutes/PDF/ByChapter/Chapter_32A.pdf.
- U.S. Department of Health & Human Services National Institute on Aging. (2016). End of life: Helping with comfort and care. NIH publication no. 16–603. Retrieved from https://order.nia.nih.gov/sites/default/files/2017-07/End_of_Life_508.pdf.
- U.S. Government Services and Information. (2020). Family legal issues: Power of attorney. Retrieved from <https://www.usa.gov/family-legal#item-213778>.
- U.S. National Council on Disability. (2018). Beyond guardianship: Toward alternatives that promote greater self-determination. 145–152. Retrieved from <https://ncd.gov/publications/2018/beyond-guardianship-toward-alternatives>.
- Uniform Law Commission. (1993). Health-Care Decisions Act. Retrieved from <https://www.uniformlaws.org/>

[committees/community-home?CommunityKey=63ac0471-5975-49b0-8a36-6a4d790a4edf](https://www.uniformlaws.org/committees/community-home?CommunityKey=63ac0471-5975-49b0-8a36-6a4d790a4edf).

Uniform Law Commission. (2014). Recognition of Substitute Decision-Making Documents Act. Retrieved from <https://www.uniformlaws.org/committees/community-home?CommunityKey=87d8d5a6-55e2-400e-887c-5fe978711cde>.

Zerbo, O., Massolo, M., Qian, Y., & Croen, L. (2015). A study of physician knowledge and experience with autism in adults in a large integrated healthcare system. *Journal of Autism and Developmental Disorders*, 45(12), 4002–4014. <https://doi.org/10.1007/s10803-015-2579-2>.

PPS

- [Revised Knox Preschool Play Scale \(RKPPS\)](#)

PPVT: Peabody Picture Vocabulary Test

- [PEAK Relational Training System](#)

Practice Guidelines in Autism

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Definition

Practice guidelines have two basic purposes. First, to promote effective care by reinforcing good practice. Second, by promoting change in professional practice where this does not comply with current best practice. In autism spectrum disorder (ASD), a significant number of practice guidelines have now been developed. This section focuses specifically on clinical guidelines, used in

healthcare, but also applicable in educational and other relevant settings.

Even as recently as 10 years ago, clinicians working in ASD assessment and intervention had little in the way of a “road map” advising them which approaches had a reliable and valid evidence base and which did not. The situation was even more confusing for people with ASD and their carers. Rapid developments over the last 10 years in ASD practice guideline development have reduced much of that uncertainty, but, as a corollary, successive guidelines have revealed the extensive gaps in the ASD evidence base and the research that is now required.

Clinical practice guidelines are developed by multiprofessional groups that are locality (region or state) or nationally representative. A systematic review is undertaken to identify and critically appraise the scientific literature. Guideline recommendations are then explicitly linked to (and graded according to the strength of) the supporting evidence. The guideline development group (GDG) needs to be representative of the key constituencies within the field under study. This is paramount in a politically sensitive field such as ASD. The ASD GDG should aim to represent all facets of the ASD multiagency, parent/carers, and voluntary network within the geographic area that the guideline pertains to. In order to build this consensual and representative approach, GDG composition needs to be carefully constructed.

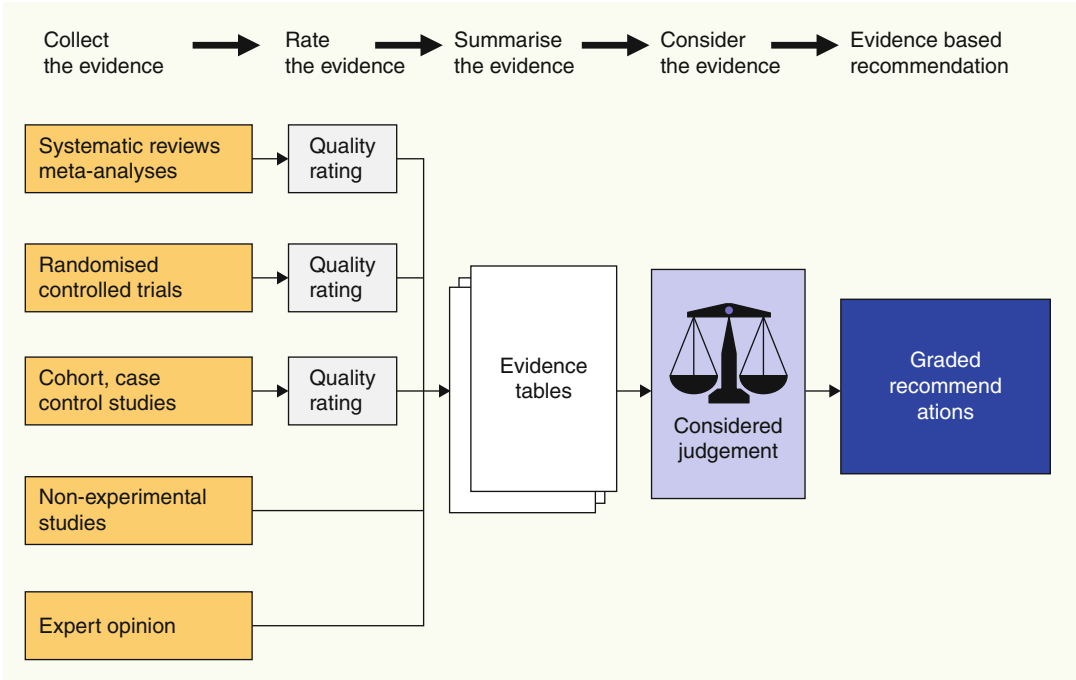
First, a chairman is recruited to lead the development of the practice guideline. Second, the guideline chair has to then recruit the GDG. The chair and GDG then have to clarify the guideline’s exact remit, a clue which is often to be found in the guideline title. For example, the guideline *Assessment, diagnosis and clinical interventions for children and young people with autism spectrum disorders* published by the Scottish Intercollegiate Guidelines Network (SIGN) in 2007 (SIGN 98 2007) explains the basic remit and range of its approach. Its emphasis on “clinical” interventions implies that it does not aim to cover primarily educational-based interventions, for example (although in ASD, it is hard to apply exact cutoff points in regard to types of interventions).

The GDG then needs to decide on key themes within the agreed remit. From these themes, “key questions” are generated. These questions aim to clarify and identify the field of evidence that needs to be sought. For example, in SIGN 98, key question 12 asked: “*Which conditions occur in association/comorbidly with ASD, and can their presence be specifically excluded or confirmed?*” (SIGN 98 2007). This question was then used to generate search terms in the literature search for that area of enquiry (i.e., comorbidity in ASD).

The process of evidence gathering and its subsequent analysis is varied, according to different guidelines. The highest standards of scientific validity and reliability tend to be found in those guidelines produced by national guideline organizations, such as the Scottish Intercollegiate Guidelines Network (SIGN) or the National Institute for Clinical Excellence (NICE) for England and Wales. Other guidelines may have somewhat more relaxed processes of evidence gathering and data analysis, especially relevant for a field such

as ASD where there is comparatively little quantitative “high-level” evidence, but where it is felt important to make recommendations across all aspects of the care agenda, in order to influence uni- or multiprofessional practice. This explains why, for example, that in regard to the recommendation levels in SIGN 98 (SIGN 98 2007) and the *New Zealand guideline on ASD* published in 2008 (Ministries of Health and Education 2008), there is a significant disparity of levels of evidence recommendation, despite the fact that the two guidelines conducted their international literature searches at approximately the same time. The SIGN guideline makes far less recommendations at the higher levels (i.e., A or B) than its New Zealand counterpart, due to its tighter evidence gathering, analysis, and rating criteria. Also the grades of recommendation systems are different, so that in SIGN 98 a level D recommendation denotes nonanalytic studies, for example, case reports, case series, or expert opinion, while in the NZG, expert opinion qualifies as a level C recommendation.

Practice Guidelines in Autism, Table 1 SIGN’s evidence rating process. (Copyright SIGN, reproduced by permission)



SIGN’s evidence gathering and analysis process are explained in Table 1.

This includes systematic reviews and meta-analyses, randomized controlled trials (RCTs), cohort and case control studies, nonexperimental studies and expert opinion. Second, the evidence gathered is rated according to its quality. Third, the evidence is summarized in evidence tables. Fourth, the guideline development group considers this evidence and rates it into considered judgment forms. Fifth, these forms then generate graded, evidence-based recommendations.

Table 2 shows the SIGN system of rating evidence levels, using a numerical scoring system and asterisks. Thus, an evidence level of 1⁺⁺ denotes high-quality meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias. At the other end of the evidence-rating spectrum, an evidence level of 4 denotes expert opinion (i.e., evidence that has not even been scrutinized within a nonanalytic study).

Table 3 shows how grades of recommendation are stratified in the SIGN system. The highest level of evidence-based recommendation (level A) denotes an evidence level that includes at

least one meta-analysis, systematic review, or RCT rates as 1⁺⁺ and that is directly applicable to the target population of the evidence-based guideline. Alternatively, this grade of recommendation can be achieved by the existence of a systematic review of RCTs or a body of evidence consisting principally of studies rated as 1⁺ that is directly applicable to the target population and that demonstrates overall consistency of results. At the other end of the spectrum of graded recommendations, level D denotes that the evidence level available is rated as 3 or 4, or that it is evidence extrapolated from studies rated as 2⁺⁺. An important point to remember about the resulting recommendation gradings is that, as the New Zealand ASD guideline states, “the attached grading reflects the rigour of the studies providing the evidence rather than an indication of the importance of the recommendation” (Ministries of Health and Education 2008).

Practice Guidelines in Autism, Table 2 SIGN’s levels of evidence. (Copyright SIGN, reproduced by permission)

SIGN levels of evidence	
1 ⁺⁺	High-quality meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias
1 ⁺	Well conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias
1 ⁻	Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias
2 ⁺⁺	High-quality systematic reviews of case-control or cohort studies
	High-quality case-control or cohort studies with a very low risk of confounding, bias, or chance and a high probability that the relationship is causal
2 ⁺	Well conducted case-control or cohort studies with a low risk of confounding, bias, or chance and a moderate probability that the relationship is causal
2 ⁻	Case-control or cohort studies with a high risk of confounding, bias, or chance and a significant risk that the relationship is not causal
3	Nonanalytic studies, e.g., case reports, case series
4	Expert opinion

Practice Guidelines in Autism, Table 3 SIGN grades of recommendation. (Copyright, SIGN, reproduced by permission)

SIGN grades of recommendation	
A	At least one meta-analysis, systematic review, or RCT rated as 1 ⁺⁺ , and directly applicable to the target population
	<i>or</i>
	A systematic review of RCTs or a body of evidence consisting principally of studies rated as 1 ⁺ , directly applicable to the target population, and demonstrating overall consistency of results
B	A body of evidence including studies rated as 2 ⁺⁺ , directly applicable to the target population, and demonstrating overall consistency of results
	<i>or</i>
	Extrapolated evidence from studies rated as 1 ⁺⁺ or 1 ⁺
C	A body of evidence including studies rated as 2 ⁺ , directly applicable to the target population and demonstrating overall consistency of results
	<i>or</i>
	Extrapolated evidence from studies rated as 2 ⁺⁺
D	Evidence level 3 or 4
	<i>or</i>
	Extrapolated evidence from studies rated as 2 ⁺

Historical Background

Practice guidelines in ASD are a fairly recent phenomenon. The main initial guideline development activity took place in the USA toward the end of the 1990s and early 2000s. These were either unidisciplinary national guidelines (Filipek et al. 2000; Volkmar et al. 1999) or multi-professional but state based (New York state Department of Health, Early Intervention program 2000; California Department of Developmental Services 2002). Thereafter, guideline development activity spread to Europe (Le Couteur 2003; SIGN 98 2007) and New Zealand (Ministries of Health and Education 2008). The difference with these later guidelines was that they were both national and multi-professional. Subsequent guideline development has been more targeted, using the work completed by previous GDGs which are relevant to universal geographical localities but focusing on specific aspects that are geographically particular. For example, in 2009, the National Institute for Clinical Excellence (NICE) for England and Wales decided to limit the remit of its forthcoming ASD guideline for children and young people to clinical assessment only. NICE decided not to undertake a more extensive evidence review to look at clinical interventions and treatments, because that work had been only recently done by SIGN, NICE's sister organization in Scotland. Therefore, subsequent national guideline approaches seem to be becoming more targeted, reflecting previous work undertaken and, perhaps, a more cost-conscious financial climate.

All of the published guidelines, whether by unidisciplinary, state, or national organizations, contain unique and interesting approaches which means that all of them repay close examination. For example, the New York State guideline (New York state Department of Health, Early Intervention program 2000) restricted its remit to 0–3 years and in this regard may be seen as one of the most detailed surveys of this crucial epoch regarding ASD. Information for “clinical clues” that should point the clinician to signs of possible autism in a child of this age range were carefully evidence based. For this reason, SIGN 98 (SIGN

98 2007) deliberately adopted this aspect of the New York guideline for its own recommendation, as being the best evidence base available. This is an example of a practice guideline becoming the source of the available evidence base for subsequent guidelines.

The *National Autism Plan for Children* for England and Wales (Le Couteur 2003) is unique in that it not only considered the evidence base for its various recommendations, but it also developed a consensus set of recommendations as regards what an ASD clinical service for pre-school and primary-school-aged children should consist of in terms of basic requirements. These suggestions are not, strictly speaking, evidence based, because they were the product of clinical consensus (i.e., they constitute level 4 evidence). However, given that no subsequent guideline group has developed as detailed a description of the different tiers of an ASD service, the NAPC recommendations have gained increasing importance over the years as a point of reference and, effectively, as evidence.

SIGN 98 produced perhaps the most robust and extensive survey of the international evidence regarding ASD up to its publication date. In addition, it was the first guideline to raise the important issue of validity and reliability of ASD diagnosis as regards subject recruitment for scientific studies. Simply put, SIGN's GDG asked, can researchers provide a reliable and valid guarantee that the subjects entered into their research studies, whom they claim have ASD, actually *do have* the condition (as defined by categorical classification systems)? The GDG argued that if researchers cannot do this, their research findings, no matter how “groundbreaking,” must be accordingly downgraded. To back up this approach, SIGN 98's GDG developed a sliding scale of reliability and validity for diagnosis as regards study subject recruitment (SIGN 98 2007) (see Table 4).

Cultural factors are a key parameter to consider in a condition such as ASD. The NZG (Ministries of Health and Education 2008) has provided a fascinating lead in this regard, with its sensitive consideration of the particular needs and interests of New Zealand's Maori community. Presumably any subsequent national multiprofessional

Practice Guidelines in Autism, Table 4 Criteria for assessing the reporting of the diagnosis of ASD in the literature. (Reproduced by permission of SIGN)

A. Components of diagnostic assessment		
1. A recognized process of obtaining information in necessary domains, usually by multidisciplinary or multiagency personnel		
2. Mapping of the resulting information into a recognized classification system such as DSM-IV or ICD-10 (see Sect. 2.2)		
3. Assessment using a recognized and published diagnostic instrument		
B. Components of a reliable diagnosis		
Increasing accuracy and reliability		Use of a process and diagnostic classification system, and an instrument (i.e., 1, 2, and 3, from A)
		1. Use of a process and a diagnostic classification system
		or
		2. Use of an instrument and a diagnostic classification system
		The use of a process, a diagnostic classification system, or an instrument, used singly
		Diagnosis simply stated

NB each component of the assessment should be explicitly stated in the study/report under consideration

guideline will need to seriously consider following this benchmark in its design, if cultural diversity is an issue pertaining to that population.

Current Knowledge

The most recent clinical practice guidelines have been those developed by the Scottish Intercollegiate Guidelines Network on *ASD in children and young people* published in June 2007 (SIGN 98 2007), which covers the epoch from 0 to 19 years and the New Zealand guideline on *Autism Spectrum Disorder*, published in March 2008, which considers ASD in the whole age range, including adults (Ministries of Health and Education 2008). This section will therefore consider the evidence base according to these two

guidelines. It is important to note, however, that these two guidelines “stand on the shoulders” of previous guidelines. For example, in the NZG, its section on diagnosis and assessment of young children is based on the *National Autism Plan for Children* (Le Couteur 2003) and “further reference was made” to the *Autistic Spectrum Disorders Best Practice Guidelines for Screening, Diagnosis and Assessment* that was developed by the California Department of Developmental Services (California Department of Developmental Services 2002).

As explained earlier, SIGN 98 and the NZG demonstrate different levels of evidence recommendation, with SIGN using tighter criteria, being less able to make recommendations generally or certainly higher-level (A or B) recommendations. In addition, the scope of the NZG is wider than SIGN 98, in that it covers the whole age range as opposed 0–19 years inclusive, and it considers educational interventions, for example, whereas SIGN 98 focuses on clinical interventions (or educational interventions that have a clinical application). If SIGN had considered the more general spread of educational interventions, it is unlikely that much of the evidence gathered would have been suitable for evidence-based recommendation, within SIGN’s evidence-rating system. This is an example of guidelines being pitched at the level for which they are required, either by a professional group or by a national government, to determine health and education policy. Theoretically, SIGN’s tighter criteria mean that its recommendations are more applicable internationally, which explains why they have been adopted in countries other than Scotland. A downside of this tighter approach, however, may be that important clinical areas (especially to patients and carers), for example, sensorimotor development, are left undirected in regard to the (lower) levels of evidence that are available. Sometimes, such gaps can be filled by the GDG stating “good practice points” (GPPs), and these can be found in both SIGN 98 and the NZG but, again, at possibly different criteria levels.

This section follows the structure of SIGN 98’s presentation of the evidence, with

slight modifications and therefore specifically limits its remit to “assessment, diagnosis and clinical interventions.” Readers who wish to consider the broader evidence base for educational interventions, for example, should look at the NZG.

Definitions and Concepts

SIGN 98 uses the term “autism spectrum disorders” (ASD) to cover the ICD-10 conditions termed autism, atypical autism, and Asperger’s syndrome (World Health Organization [WHO] 1993), and the NZG uses the term “ASD” to cover the DSM-IV-TR conditions of autistic disorder, Asperger’s disorder, and PDD-NOS (American Psychiatric Association 1994). Both guidelines consider both classification systems. SIGN 98 defers to ICD-10 in any difference of terminology between it and DSM-IV-TR. The NZ guideline follows DSM-IV-TR.

Recognition, Assessment, and Diagnosis

Recognition in Primary Care

Screening Both guidelines state that population screening for ASD is “not recommended” (SIGN at level C and the NZG at level B).

Surveillance SIGN 98 explains that, “as part of the core programme of child health surveillance,” clinicians can help to identify early those children who may have ASD or other developmental disorders. At level D recommendation, SIGN 98 states:

clinical assessment should incorporate a high level of vigilance for features suggestive of ASD, in the domains of social interaction and play, speech and language development and behaviour and that:

CHAT or M-CHAT can be used in young children to identify clinical features indicative of an increased risk of ASD, but should not be used to rule out ASD.

While the NZG does not make such specific recommendations, it recommends, at level C, that “health and education professionals should take regular opportunities (at least at 8–12 months, 2–3

years and 4–5 years) to discuss the child’s development with parents.”

SIGN 98 provides three useful tables which consider “warning signs” suggestive of possible ASD in the three age ranges of preschool and

Practice Guidelines in Autism, Table 5 Additional warnings of possible ASD in adolescents. (Copyright, SIGN. Reproduced by permission)

Warning signs
<i>General picture</i>
– Long-standing difficulties in social behaviors, communication, and coping with change, which are more obvious at times of transition (e.g., change of school, leaving school)
– Significant discrepancy between academic ability and “social” intelligence, most difficulties in unstructured social situations, e.g., in school or work breaks
– Socially “naïve,” lack common sense, not as independent as peers
<i>Language, nonverbal skills, and social communication</i>
– Problems with communication, even if wide vocabulary and normal use of grammar. May be unduly quiet, may talk at others rather than hold a to-and-fro conversation, or may provide excessive information on topics of own interest
– Unable to adapt style of communication to social situations, e.g., may sound like “a little professor” (overly formal), or be inappropriately familiar
– May have speech peculiarities including “flat,” unmodulated speech, repetitiveness, use of stereotyped phrases
– May take things literally and fail to understand sarcasm or metaphor
– Unusual use and timing of nonverbal interaction (e.g., eye contact, gesture, and facial expression)
<i>Social problems</i>
– Difficulty making and maintaining peer friendships, though may find it easier with adults or younger children
– Can appear unaware or uninterested in peer group “norms,” may alienate by behaviors which transgress “unwritten rules”
– May lack awareness of personal space or be intolerant of intrusions on own space
<i>Rigidity in thinking and behavior</i>
– Preference for highly specific, narrow interests or hobbies, or may enjoy collecting, numbering, or listing
– Strong preferences for familiar routines, may have repetitive behaviors or intrusive rituals
– Problems using imagination, e.g., in writing, future planning
– May have unusual reactions to sensory stimuli, e.g., sounds, tastes, smell, touch, hot or cold

school-age children and in adolescents. The pre-school table comes from the New York guideline (New York state Department of Health, Early Intervention program 2000) and the school-age table is from the NAPC (Le Couteur 2003). The NZG also uses the NAPC concepts in its similar tables. Table 5 from SIGN 98 was developed as expert consensus by the SIGN 98 guideline development group.

Screening of High-Risk Groups In addition, SIGN 98 considers screening of high-risk groups (so-called secondary screening) and recommends, at level C, that “the use of an appropriate structured instrument may be a useful supplement to the clinical process to identify children and young people at high risk of ASD.”

Timing of Diagnosis SIGN 98 also considers the evidence for the timing of diagnosis, explaining that “the evidence regarding the minimum age at which ASD can be reliably diagnosed is not clear.” It makes the level D recommendation that “ASD should be part of the differential diagnosis for very young (pre-school) children displaying absence of normal developmental features, as typical ASD behaviours may not be obvious in this age group.”

Methods of Assessment

Initial Assessment Both SIGN 98 and the NZG recognize that initial assessment will, in the NZG’s words, “be undertaken by an individual practitioner” (level B recommendation). SIGN 98 explains that “those (multiprofessional colleagues) involved in carrying out the initial assessment should be aware of the core features of ASD as well as of the wide range of different possible presentations.” Both guidelines advise that “if on the basis of initial assessment, it is suspected that a child or young person may have ASD, they should be referred for specialist assessment.”

Specialist Assessment SIGN 98 explains that, because a diagnosis of ASD may be seen as “a lifelong ‘label’,” “it is of equal importance that clinicians diagnose, and not diagnose, accurately.”

Therefore, both guidelines advise that specialist assessment should be undertaken by “a multi-disciplinary team of health care practitioners experienced in ASD” (level B recommendation). The multiprofessional approach is recommended because “it may identify different aspects of ASD and aid accurate diagnosis” and collaborative multiprofessional conclusions will enable informed intervention with “effective educational, behavioural, physical, emotional, social and communication strategies. . .to promote development.”

NZG also recommends that “diagnostic assessment of young people and adults should be comprehensive” and “involve the person concerned in interview and observation” and that “formal pathways for diagnostic assessment of young people and adults should be developed” (level C recommendation).

Components of Specialist Assessment

(a) History taking (parent/carer interview)

SIGN 98 explains that taking an “ASD-specific diagnostic history” is an important component of an ASD assessment because “without it, evidence of ASD-like behaviour cannot be put into context” (level D recommendation). Although ASD-specific history-taking instruments can be “useful” “as a means of improving the reliability of ASD diagnosis” (level C recommendation), SIGN 98 explains the paramount importance of clinicians keeping “a global perspective” of the patient’s circumstances in mind, “taking into consideration the possibility of comorbidities and the possible differential diagnoses.” In addition, the NZG advises that “if the person taking the developmental history is not medically trained, then the medical history and examination should be completed separately.”

(b) Clinical observation/assessment (child/young person assessment/interview)

SIGN 98 recommends that “healthcare professionals should directly observe and assess the child or young person’s social and communication skills and behaviour” (level D recommendation). Again, there is a recommendation that clinicians “should consider using ASD-specific observational instruments,

as a means of improving the reliability of ASD diagnosis” (level C recommendation). Such aids would include instruments like ADOS-G (Lord et al. 2000).

(c) Contextual and functional information

The NZG explains that “direct observation of the child’s behaviour in an unstructured setting is essential” and SIGN 98 states the good practice point that “information about children’s functioning outside the clinic setting, should be routinely obtained from as many sources as is feasible.”

Individual Profiling

Because people with ASD vary considerably as regards their individual difficulties and strengths, SIGN 98 recommends that “all children and young people with ASD should have a comprehensive evaluation of their speech and language and communication skills, which should inform intervention” (level D recommendation).

The NZG recommends in addition that “the assessment of intellectual, adaptive and cognitive skills” associated with ASD “should be seriously considered and, where possible and appropriate, formally assessed” (level B recommendation).

Biomedical Investigations

SIGN 98 makes a level D recommendation that “where clinically relevant” all children and young people with ASD should have their physical status examined “with particular attention to neurological and dysmorphic features”; undergo karyotyping, fragile X DNA analysis, and audiological examination; and be investigated to rule out recognized etiologies of ASD (such as tuberous sclerosis). In addition, SIGN 98 explains its awareness of the “considerable interest in the role of the immune system and the influence of bowel function” in ASD and that it conducted “an extensive search” for research into this area. SIGN 98 explains that its GDG “found no research evidence of an acceptable level in support of the clinical use” of investigations aimed at supporting such biomedical theories and that, therefore, in respect of this controversial area, it is “not possible at present to make a recommendation.”

Conditions Associated with ASD

SIGN 98 makes a level C recommendation that clinicians should “be aware of the need to routinely check for comorbid problems in children and young people with ASD” and that “where necessary, detailed assessment should be carried out to accurately identify and manage comorbid problems.”

The NZG makes three recommendations covering this area that “differential diagnosis must be covered during diagnostic assessment” and “must be thorough and cover all conditions commonly confused with ASD and those known to coexist with ASD” (level C). Thirdly, the NZG recommends that clinicians “must have a good understanding of the different forms of expression of ASD symptomatology across developmental stages and the symptomatology of common coexisting and alternative conditions” (level B).

Nonpharmacological Interventions

Communication Interventions

Support for Early Communication Skills SIGN 98 recommends that “interventions to support communication in ASD are indicated, such as the use of visual augmentation, e.g. in the form of pictures of objects” (level D).

Interventions for Social Communication and Interaction

SIGN 98 recommends that “interventions to support social communication should be considered for children and young people with ASD, with the most appropriate intervention being assessed on an individual basis” (level D).

Behavioral/Psychological Interventions

SIGN 98 explains that these types of interventions fall into three groups: intensive behavioral programs “aimed at improving overall functioning and altering outcome”; interventions which “aim to address specific behavioural difficulties associated with ASD,” for example, sleep disturbance; and interventions which do not fall into either of the first two categories.

Intensive Behavioral Programs SIGN 98 explains that “most intensive behavioural programmes for ASD are based on the principles of behaviour modification using applied behavioural analysis (ABA).” While the NZG does not comment on specific interventions in this category, SIGN 98 recommends that “the best known of the intensive ABA interventions,” the Lovaas program, “should not be presented as an intervention that will lead to normal functioning” (level A).

Interventions for Specific Behaviors The NZG recommends that “behaviour management techniques should be used to intervene with problem behaviours” (level A). SIGN 98 recommends that behavioral interventions “should be considered to address a wide range of specific behaviours. . . both to reduce symptom frequency and severity and to increase the development of adaptive skills” (level B).

Auditory Integration Training SIGN 98 recommends that “auditory integration training is not recommended” (level A).

Facilitated Communication SIGN 98 recommends that “facilitated communication should not be used as a means to communicate with children and young people with ASD” (level A).

Biomedical and Nutritional Interventions

Recognizing that this is an area of importance and focus for many parents and carers of people with ASD, SIGN 98 looked for evidence justifying or ruling out any such interventions, for example, exclusion diets. However, SIGN 98 states that there was “insufficient evidence” to meet “the criteria set for the review and therefore no recommendation can be made.”

Pharmacological Interventions

Risperidone

SIGN 98 recommends that risperidone is “useful for short term treatment of significant aggression, tantrums or self injury in children with autism”

(level B); however, it does not define “short term.” The NZG issues similar level B recommendation about risperidone, advising that “monitoring for side effects should be carried out on a regular basis.”

Methylphenidate

SIGN 98 recommends, at level B, that methylphenidate “may be considered for treatment of attention difficulties/hyperactivity in children or young people with ASD.” The NZG recommends, at level C, that methylphenidate “is effective for some children with ASD and co-morbid ADHD” but “should be used with caution because of the high risk of adverse effects.”

Fluoxetine

The NZG recommends, at level B, that SSRIs in general, including fluoxetine, “may be effective for some children with ASD and high anxiety and/or obsessive symptoms” but that, however, “in the absence of good evidence, these drugs should be used with caution and careful monitoring.”

Secretin

SIGN 98 recommends, at level A, that “secretin is not recommended for use in children and young people with ASD.”

Melatonin

At level B, the NZG recommends that melatonin “may be useful for improving sleep in children with ASD who have impaired sleep,” and at level D, SIGN 98 recommends that melatonin “may be considered for treatment of sleep problems which have persisted despite behavioural intervention.”

Other Treatments

The NZG recommends, at level B, that “typical” antipsychotics, such as haloperidol, “are effective in reducing motor stereotypies, temper tantrums and improving social relatedness,” but “these drugs have a high rate of adverse effects and are not recommended for first-line use.”

Service Provision

Training and Support for Parents

Information Provision SIGN 98 recommends that “professionals should offer parents good quality written information and an opportunity to ask questions when disclosing information about their child with ASD.” This information should be provided “in an accessible and absorbable form” (level D). The NZG issues similar guidance.

Meeting Support Needs SIGN 98 recommends that “education and skills interventions for parents of pre-school children with ASD should be offered” (level B).

Information for Discussion with Children, Young People, Parents, and Carers

Providing information and support

At the Time of Diagnosis

The NZG makes recommendations for this area, nearly all at level C, including that “formulation is the necessary next step from assessment” and that “clarity of diagnosis should be the goal of assessment and formulation.” The NZG also recommends that all diagnostic assessments “should include a detailed written report” that “disclosure of diagnosis of older teens and adults. . . should take into consideration the wishes of the person concerned” and that “sources of post-diagnosis support should be identified.” Finally, the NZG recommends that “information on ASD and support services should be available at all diagnostic disclosure interviews” (level B). SIGN 98 provides similar advice but at good practice point level only.

Recommendations for Research

SIGN 98 explains that further research is required “to address numerous areas where there is insufficient evidence to make a recommendation or to support existing clinical practice.” The guideline identifies several examples within each of four key areas:

- Recognition, assessment, and diagnosis
- Nonpharmacological interventions

- Pharmacological interventions
- Service provision

Future Directions

Given the cost of practice guideline development, it may be that the era of large, national, multi-professional guidelines such as those conducted by SIGN and in New Zealand (and the two guidelines soon to be published by NICE as regards both children and young people and adults in 2011–2012) is coming to an end. Instead, subsequent national guideline groups have elected to substantially follow the evidence-searching strategy of previous guidelines in order to save time and cost and to survey evidence from the dates of previous completed surveys (as is currently the case in Italy, which is using SIGN 98 in this way).

Thus, guideline organizations can assist each other, the international autism community, their carers, and clinicians to cultivate the evaluation of the evidence base as an ever-expanding tapestry of activity. Guideline development, in this way, provides fascinating and exciting opportunities for international collaboration. The information that will accrue from such collaboration may even generate its own contribution to the evidence base.

Furthermore, guideline organizations have been developing new ideas to spread the information available to the widest possible readership. For example, in 2009, the UK Academy of Medical Royal Colleges commissioned SIGN and the Royal College of Physicians and Surgeons of Glasgow (RCPSG) to publish two pilots of extant SIGN guidelines on the World Wide Web as resources for continuing professional development. The SIGN guideline on ASD was chosen as one of these initiatives and will be the first to be published (SIGN website; <http://www.sign.ac.uk> in 2012). This e-CPD module contains the SIGN 98 guideline itself and also contains embedded “think points” that help the reader develop their understanding of the information provided. Having read through this embellished version of the guideline, any clinician, from anywhere in the

world, can then complete the multiple choice questions that examine the clinician's understanding of the guideline.

Thus, evidence-based guidelines on ASD are now more widely available to the professional and lay public than ever. This hopefully will combat the bewildering effect of information overload that confronts the parent who, on receiving a diagnosis of ASD for their child, types “*autism*,” “*ASD*,” or “*autism treatments*” into a web search engine. There is no possible way that even the best informed lay person could reliably navigate the millions of hits that inevitably will result. For this reason alone, guideline organizations need to keep up with the possibilities inherent in information technology, to ensure that people with ASD, their carers, and the professionals who try to help them are provided with the most valid and reliable evidence base to date, in an accessible and understandable format.

See Also

- [CHAT](#)
- [Comorbidity](#)
- [M-CHAT](#)

References and Reading

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (DSM-IV)* (4th ed.). Washington, DC: American Psychiatric Association Press.
- California Department of Developmental Services. (2002). *Autistic spectrum disorders: Best practice for screening, diagnosis and assessment*. Sacramento: Author.
- Filipek, P. A., Accardo, P. J., Ashwal, S., Baranek, G. T., Cook, E. H., Dawson, G., et al. (2000). Practice parameter: Screening and diagnosis of autism. Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Child Neurology Society. *Neurology*, 55, 468–479.
- Le Couteur, A. (2003). *National autism plan for children*. London: National Autistic Society.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., Dilavore, P. C., et al. (2000). The Autism Diagnostic Observation Schedule-Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30(3), 205–223.
- Ministries of Health and Education. (2008). *New Zealand autism spectrum disorder guideline*. Wellington: Ministry of Health. [cited 26 Feb 2011]. Available from URL: <http://www.moh.govt.nz/autismspectrumdisorder>
- New York state Department of Health, Early Intervention program. (2000). Clinical practice guideline: Autism/pervasive developmental disorders: Assessment and intervention for young children (age 0–3 years). New York: Department of Health. [cited 20 May 2012]. Available from URL: http://www.health.state.ny.us/community/infants_children/early_intervention/autism/index.htm#contents
- Scottish Intercollegiate Guidelines Network (SIGN). (2007). *Assessment, diagnosis and clinical interventions for children and young people with autism spectrum disorders (The SIGN guideline)*. Edinburgh: Royal College of Physicians. [cited 26 Feb 2011]. Available from URL: <http://www.sign.ac.uk/guidelines/fulltext/98/index.html>
- Volkmar, F. R., Cook, E. H., Pomeroy, J., Realmuto, G., & Tanguay, P. (1999). Practice parameters for the assessment and treatment of children, adolescents and adults with autism and other pervasive developmental disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38(12), 32S–54S.
- World Health Organization (WHO). (1993). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research*. Geneva: Author.

Prader-Labhart-Willi Syndrome

- [Angelman/Prader-Willi Syndromes](#)

Pragmatic Communication

- [Pragmatics](#)

Pragmatic Communication Disorder

- [Autism: Social Communication Disorder](#)
- [Semantic Pragmatic Disorder](#)

Pragmatic Language

- ▶ [Discourse Management](#)
- ▶ [Pragmatics](#)

Pragmatic Language Disorder

- ▶ [Autism: Social Communication Disorder](#)
- ▶ [Pragmatic Language Impairment](#)

Pragmatic Language Impairment

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Synonyms

[Pragmatic language disorder](#); [Social \(pragmatic\) communication disorder](#); [Social communication disorder](#)

Short Description or Definition

Pragmatic language impairment (PLI) is a type of developmental language impairment in which there is disproportionate difficulty with pragmatics and social communication compared to the structural aspects of language such as grammar and vocabulary.

Categorization

PLI is not included as a category in DSM-V. PLI is a descriptive term that is used to identify the type of language problem present. PLI is still in clinical use but has been replaced in the research literature

and autism diagnostic practice by the term “Social Communication Disorder” or “Social (Pragmatic) Communication Disorder” (SPCD), principally by consensus (Swineford et al. 2014). DSM-V presented SCD as a new and distinct category from autism spectrum disorder (ASD) and specific language impairment (SLI). SCD is described as: a condition evident from early childhood, in which there is a persistent impairment in using language for social interactions (pragmatics). The impairment is accompanied by limitations in functional social participation that are not explained by low cognitive functioning or ASD. Nonverbal communication is also affected.

Epidemiology

There are no prevalence figures for PLI since it is not recognized as a category in diagnostic models. Precise estimates of prevalence of SCD are difficult to determine due to difficulties associated with differential diagnosis. Using DSM-V criteria for SCD in a Korean community sample of 7–12-year-old children, Kim et al. (2014) estimated the prevalence of SCD to be a 0.49%, commenting that this may be conservative. Ketelaars et al. (2009) found the prevalence of pragmatic language impairments to be 7.5% of children in a large community sample in the Netherlands, though it is not clear that these children would meet DSM-V criteria for SCD. Gender differences were found in this study; more boys were affected than girls in a ratio of 2.6:1. Botting and Conti-Ramsden (1999) identified 22% of elementary school-age children attending specialist language units in the UK as having PLI, indicating that amongst this severely affected group, a diagnosis of PLI or SCD is not uncommon.

Natural History, Prognostic Factors, Outcomes

Children with PLI/SCD show late emergence of language, including receptive language delay and an early history of autistic features (such as echolalia) with impaired social relationships (Adams

2015). By 5–6 years of age, there are relatively few problems with grammar and phonology and more fluent language has emerged, often with persistence of significant receptive language problems. A full profile of PLI/SCD emerges at this stage as the contrast between structural language and pragmatic competence becomes more marked.

Time spent in special educational provision by children with PLI/SCD is longer than that of children with other types of language impairment. Pragmatic difficulties in the primary school years may be related to emotional and behavior difficulties (Van Agt et al. 2011; Helland et al. 2014). Adults with a PLI history have relatively normal literacy and structural language skills, fewer autism symptoms than comparable ASD

individuals, and better work skills outcomes than adults with a history of SLI (Whitehouse et al. 2009).

Clinical Expression of Illness

(i) *Pragmatic behaviors in naturalistic social interactions:* Table 1 sets out pragmatic language behaviors observed in children who have PLI, typically after 4–5 years of age. These are present in conversational interactions or other unstructured talk. This account of pragmatic errors is based on the Gricean view of pragmatics as cooperative behavior between speakers. That is, talk should be relevant and informative.

Pragmatic Language Impairment, Table 1 Main pragmatic features observed pragmatic language impairment

Verbosity	The child talks at length about a topic and offers more than would be expected in cooperative talk
Loquacity	The child talks at length within one conversational turn; the impression is of a monologue rather than a genuine conversational exchange
Domination of interaction	The overall impression is that the child is dominating the interaction by being verbose/loquacious and not allowing the other speaker a turn
Initiations	The child initiates exchanges of information (a) more frequently than expected, by making many unsolicited statements or by asking many questions; or (b) the child is relatively passive or appears uninterested by making no or very few initiations
Responsiveness	The child does not respond to a conversational overture such as a question or a clear cue from the other speaker that it is his turn to talk; appears to ignore the other speaker
Turn-taking	There are problems with taking turns in interactions so that speakers talk at the same time. Signals (intonation, grammatical, and nonverbal signs) that are given by one speaker to another to indicate that the turn to talk is being passed over are not detected by the child or he is verbose and presses on with his talk over the other speaker.
Information management	The child offers too much information in the interaction and thus appears pedantic or odd. The same child may offer too little information, so that intended meaning is vague and the listener has difficulty following meaning
Reference	The child does not specify clearly objects, actions, or people who are being talked about; he does not take into account how much the listener knows already or could be expected to work out from the linguistic or physical context
Topic	The child changes topic suddenly (topic shift) or provides an unexpected link to a topic unrelated to the current exchange (topic drifting or linking). Shifting/drifting can occur to topics which are not relevant to the situation or may represent attempts to return to recurrent topics/favored interests
Coherence	The child's talk is disorganized, with unclear references and confused sequences in narratives, leading to loss of overall meaning of the discourse
Cohesion	The child does not use correct linguistic markers (e.g., pronouns) to indicate links between different utterances or turns resulting in confusion for the listener
Speech acts	Most children with PLI of school age show a normal range of speech act forms (e.g., questions, statements, denials) but may not always use these in optimal contexts

Pragmatic Language Impairment, Table 2 High-level language and comprehension deficits in PLI

Nonliteral interpretations	The child misinterprets, or interprets literally, nonliteral language such as metaphors, idioms, jokes, sarcasm
Inferential comprehension	Inference refers to the ability to retrieve unstated information from discourse and is fundamental to above-sentence level comprehension. The child with PLI typically shows limited ability to make appropriate inferences, particularly in naturalistic discourse, and may therefore misinterpret meanings
Comprehension of discourse	The child is unable to follow complex verbal interactions and may therefore not be able to contribute meaningful responses. Most children with PLI have some deficits in inference comprehension and in applying world knowledge to discourse comprehension
Misinterpretation of meanings in context	Words which have multiple meanings (e.g., bank, glasses) are misinterpreted by the child because he does not use the context (usually the sentence, but sometimes a physical context) to identify the correct meaning
Narrative organization	The child shows disorganized narratives (stories, accounts of recent events), leaving listeners confused as to actually happened. Many behaviors contribute to poor narrative organization such as inadequate reference, poor coherence, and cohesion as well as features of language impairment (e.g., poor vocabulary)

(ii) *High-level language difficulties* refer to the level of language processing at above-sentence level, for example, conversational discourse, understanding stories, narrated events, printed text in books. These are apparent on formal tasks and standardized testing but also pervade the child’s everyday functioning. Table 2 lists the main high-level language deficits seen in PLI and describes how these affect communication.

Semantic errors and immaturities do occur in children with PLI and are similar in nature and proportion to semantic behaviors seen in SLI. Use of semantically related substitutions or replacement of advanced vocabulary by earlier acquired words is common in all language impairments. Word-finding deficits may be present in PLI but are not universal in this group and are more common in SLI. Children with PLI may present with one or many other language deficits such as lag in sentence comprehension or expressive vocabulary (see evaluation below).

(iii) *Other communication features* of some children with PLI are similar to the social communication deficits in children with high-functioning ASD (HFASD). These are

(a) stereotyped language: the child inserts

“learned” sentences or phrases (e.g., “all of a sudden”) into interactive contexts where they have no meaning; (b) unusual or stereotyped intonation; (c) abnormalities of non-verbal communication: limited use of gesture to supplement communication, gaze aversion; and (d) associated characteristics such as social cognition deficits, difficulty with peer relations, behavioral difficulties, anxiety, and depression have been reported.

Heterogeneity in PLI

Children with PLI rarely demonstrate all of behaviors listed above. Some children may show mild pragmatic impairments and social interaction problems with relatively good language skills; others show multiple pragmatic, language, and social characteristics, frequently, and in all types of interactions.

Evaluation and Differential Diagnosis

Evaluation of the communication features of pragmatic language impairments in children follows three main principles:

- A comprehensive assessment must include assessment of language skills, pragmatic ability and social interaction.

- Since presentation of PLI is variable, evaluation should be individualized in order to pursue in-depth characteristics which should be targeted in intervention.
- Ideally evaluation should be carried out as part of a multidisciplinary assessment to address ASD and language characteristics.

Language, pragmatics, and social communication assessments should be carried out by a speech-language practitioner with experience in language disorders and autism. A combination of observational and formal assessment techniques will be used. The context of assessment is crucial. Functional deficits of social communication apparent in the child's everyday life may not be easy to observe in structured elicitation procedures or tests, even those dedicated to pragmatics, so test scores alone may overestimate ability. Evaluation should include interviews with carers and teachers.

Pragmatic skills shown in Table 1 are typically evaluated using observational checklists and naturalistic sampling such as classroom or family interactions or simple conversation in clinic. Before 4–5 years of age it may be difficult to identify problematic pragmatic behaviors, although some observational instruments of high quality are available (e.g., the Language Use Inventory (O'Neill 2007)).

There are no detailed normative data for the emergence of complex pragmatic behaviors in late pre-school to school-age children. All aspects of language skills including word and sentence level, in addition to the higher-level language skills shown in Table 2, need to be assessed by a specialist speech-language practitioner. Some standardized tests also have some pragmatic content, for example, Expression, Reception and Recall of Narrative Test (Bishop 2004), Test of Pragmatic Language-2 (Phelps-Terasaki and Phelps-Gunn 2007), and the Social Use of Language Test (Bowers et al. 2008).

An indication of the presence of PLI/SCD in children can be obtained using the Children's Communication Checklist (CCC-2) (Bishop 2003), a parent or teacher report instrument. Two summary CCC-2 scores can be derived: a General

Communication Composite which indicates the presence of communication impairment and a Social Interaction Deviance Composite which can indicate the presence of a disproportionate pragmatic impairment.

At the present time, differential diagnosis from other conditions is largely by consensus and convention rather than precise published criteria (Simms and Jin 2015). Differential diagnostic criteria are set out below alongside discussion of problematic areas.

Intellectual impairment is excluded as an explanatory factor in PLI using nonverbal IQ cut-off. PLI have nonverbal IQ >70; intellectual impairment nonverbal IQ <70. This is identical to the IQ cut-off used for SLI diagnosis, though narrower definitions of SLI use >85 cut-off. In clinical practice, children with PLI with nonverbal IQ in the range 70–85 are likely to present with a similar profile of language impairment as children with PLI who have nonverbal IQ >85.

PLI is differentiated from SLI by consideration of disproportionality of the impairment of pragmatics compared to the impairment in structural aspects of language such as grammar, vocabulary, and phonology. The Social Interaction Deviance Composite (SIDC) score of the CCC-2 can indicate the likely presence of PLI, but should not be used in isolation, as there is no precise cut-off between SLI and PLI. Caution is required, since SLI-type deficits (both expressive and receptive) may be perceived by CCC-2 respondents as affecting pragmatic competence. Significant receptive language difficulties, in particular, can result in the child adopting compensatory strategies in verbal interactions that appear bizarre and mimic some of the characteristics of PLI. Best diagnostic practice is to combine use of the CCC-2/SIDC with language assessment findings and specialist speech-language practitioner opinion.

It has been proposed that children with PLI/SCD can be differentiated in diagnosis from those with ASD (Bishop and Norbury 2002; Leyfer et al. 2008) since the former group demonstrate fewer repetitive behaviors and restricted interests dimension of ASD (Gibson et al. 2013). Reisinger et al. (2011) found relatively mild

autism characteristics (including restricted and repetitive behaviors) in a group of children with PLI compared to ASD children. However, Mandy et al. (2017) did not find evidence for SCD as a distinct category from ASD in a large sample of children and young people from a social communication disorders clinic. Bishop proposed that PLI might represent an intermediate state on a continuum between SLI and HFA, since children with PLI tended to move in and out of ASD diagnosis over time.

There remains a lack of confirmation of the differential status of ASD and SCD (Mazefsky et al. 2013; Foley-Nicpon et al. 2017). Additional instruments that focus on social communication and pragmatic deficits are still required 20 years after the first use of the term PLI. Contemporary research consensus is that PLI/SPCD is a condition on the borderlands of autism. The label of PLI or SCD may be useful where there is no diagnosis of autism provided but pragmatic problems requiring intervention persist. One difficulty of subsuming PLI/SCD under autism services is that children who require specialist language interventions may be assigned to generic autism behavioral programs and the effects of these are unknown with this population (Norbury 2014).

Treatments

Treatment for PLI is via a holistic, multi-disciplinary approach, taking into account priorities of children, carers, and other service users. Communication treatments for PLI are directed toward the language, pragmatic, and social interactional elements of the condition. Treatments can be direct (therapeutic activities and practice with a speech-language practitioner) and indirect (advice and training offered to teaching staff and parents). Ideally a combination of approaches should be instigated but specialist knowledge and expertise is essential. Children may also require other treatments for associated conditions at the same time as communication therapy.

Pre-school speech-language treatments are likely to be aimed at encouraging use of language

and establishing skills which underpin language comprehension, supplemented by advice on language facilitation to carers. Recognition is now given to the importance of early intervention for social use of language by increasing appropriate communicative intents and working in small groups (Parsons et al. 2017). These treatments should be delivered or supervised closely by speech-language practitioners.

Direct treatments for language impairment as the child enters the school years are diverse and will be matched to the individual child's profile. Language therapy techniques are used to target needs such as receptive language or word-finding difficulties. A substantial body of work on social communication interventions for children with language impairments is available (Brinton and Fujiki 1995; Timler et al. 2005). Pragmatic skills deficits are approached directly using metapragmatic therapy in which the child is encouraged to apply pragmatic rules in social interactions via self-monitoring. Social communication deficits are often treated in groups using a social skills approach, but this can lack individualization or targeting of specific skills. An integrated model of direct intervention, in which language, pragmatic and social communication therapies are matched to the individual needs of the child, is described in Adams and Gaile (2015). An expert summary of social language intervention principles and methods is provided by Fujiki and Brinton (2017).

Indirect support can be provided for classroom/home generalization of language goals and social communication via training of education support workers, teachers, and parents. Speech-language practitioners can provide advice on the level and complexity of language input and strategies for problem-solving difficult communication situations. Children may also require a number of personalized support strategies such as visual timetables.

Great care should be taken to distinguish between treatments that have a broad social communication function approach and those that focus on specific aspects of language pragmatics. There is a wide range of possible treatment approaches for broad social communication

needs; these are listed on the American Speech and Hearing Association website <https://www.asha.org>. However, not all of these treatments focus on language and pragmatics. The evidence for treatments of language pragmatic impair-

ments is relatively sparse (Gerber et al. 2012) compared to the vast literature on social skills interventions (Reichow and Volkmar 2010). Case reports of conversation treatments for children with SLI and/or PLI have demonstrated positive effects of intervention on pragmatic function and indicate appropriate methods of treatment (Brinton et al. 2004; Timler et al. 2005). Adams et al. (2012) showed some positive outcomes for individualized pragmatics and language intervention using the Social Communication Intervention Programme (SCIP). There is still a need for better evidence to support clinical practice in pragmatics intervention and a need for better outcome measures to support that research.

See Also

- ▶ [Autism: Social Communication Disorder](#)
- ▶ [Semantic Pragmatic Disorder](#)

References and Reading

- Adams, C. (2015). Assessment and intervention for children with pragmatic language impairment. *Social Communication Development and Disorders*, 1, 141–170.
- Adams, C., & Gaile, J. (2015). *Managing children's pragmatic and social communication needs in the early school years*. Cheshire: Napier Hill Press.
- Adams, C., Lockton, E., Freed, J., Gaile, J., Earl, G., McBean, K., et al. (2012). The Social Communication Intervention Project: A randomized controlled trial of the effectiveness of speech and language therapy for school-age children who have pragmatic and social communication problems with or without autism spectrum disorder. *International Journal of Language & Communication Disorders*, 47(3), 233–244.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Publishing.
- American Speech and Hearing Association. Social Communication: Treatment. <https://www.asha.org/PRPSpecificTopic.aspx?folderid=8589934980§ion=Treatment>. Accessed Apr 2020.
- Bishop, D. V. M. (2000). Pragmatic language impairment: A correlate of SLI, a distinct subgroup, or part of the autistic continuum? In D. V. M. Bishop & L. B. Leonard (Eds.), *Speech and language impairments in children: Causes, characteristics, intervention and outcome* (pp. 99–113). Hove: Psychology Press.
- Bishop, D. V. M. (2003). *The Children's Communication Checklist-2*. London: Harcourt Assessment.
- Bishop, D. V. M. (2004). *Expression, reception and recall of narrative instrument*. London: Harcourt Assessment.
- Bishop, D. V. M., & Norbury, C. F. (2002). Exploring the borderlands of autistic disorder and specific language impairment: A study using standardised diagnostic assessments. *Journal of Child Psychology and Psychiatry*, 43, 917–929.
- Botting, N., & Conti-Ramsden, G. (1999). Pragmatic language impairment without autism: The children in question. *Autism*, 3, 371–396.
- Bowers, L., Huisinigh, R., & LoGiudice, C. (2008). *Social language development test: Elementary*. Pro-Ed Inc. Austin, Tx.: LinguiSystems.
- Brinton, B., & Fujiki, M. (1995). Conversational intervention for children with specific language impairment. In M. Fey, J. Windsor, & S. F. Warren (Eds.), *Language intervention: Preschool through the intermediate years* (pp. 183–211). Baltimore: Paul Brookes.
- Brinton, B., Robinson, L., & Fujiki, M. (2004). Description of a program for social language intervention: “If you can have a conversation, you can have a relationship”. *Language, Speech, and Hearing Services in Schools*, 35, 283–290.
- Foley-Nicpon, M., Fosenburg, S. L., Wurster, K. G., & Assouline, S. G. (2017). Identifying high ability children with DSM-5 autism spectrum or social communication disorder: Performance on autism diagnostic instruments. *Journal of Autism and Developmental Disorders*, 47(2), 460–471.
- Fujiki, M., & Brinton, B. (2017). Pragmatics and social communication in child language disorders. In *Handbook of child language disorders* (pp. 441–460). New York: Psychology Press.
- Gerber, S., Brice, A., Capone, N., Fujiki, M., & Timler, G. (2012). Language use in social interactions of school-age children with language impairments: An evidence-based systematic review of treatment. *Language, Speech, and Hearing Services in Schools*, 43, 235–249.
- Gibson, J., Adams, C., Lockton, E., & Green, J. (2013). Social communication disorder outside autism? A diagnostic classification approach to delineating pragmatic language impairment, high functioning autism and specific language impairment. *Journal of Child Psychology and Psychiatry*, 54(11), 1186–1197.
- Helland, W. A., Lundervold, A. J., Heimann, M., & Posserud, M. B. (2014). Stable associations between behavioral problems and language impairments across childhood—the importance of pragmatic language problems. *Research in Developmental Disabilities*, 35(5), 943–951.

- Ketelaars, M. P., Cuperus, J. M., van Daal, J., Jansonius, K., & Verhoeven, L. (2009). Screening for pragmatic language impairment: The potential of the children's communication checklist. *Research in Developmental Disabilities, 30*(5), 952–960.
- Kim, Y. S., Fombonne, E., Koh, Y. J., Kim, S. J., Cheon, K. A., & Leventhal, B. L. (2014). A comparison of DSM-IV pervasive developmental disorder and DSM-5 autism spectrum disorder prevalence in an epidemiologic sample. *Journal of the American Academy of Child & Adolescent Psychiatry, 53*(5), 500–508.
- Leyfer, O. T., Tager-Flusberg, H., Dowd, M., Tomblin, B., & Folstein, S. E. (2008). Overlap between autism and specific language impairment: Comparison of Autism Diagnostic Interview and Autism Diagnostic Observation Schedule scores. *Autism Research, 1*, 284–296.
- Mandy, W., Wang, A., Lee, I., & Skuse, D. (2017). Evaluating social (pragmatic) communication disorder. *Journal of Child Psychology and Psychiatry, 58*(10), 1166–1175.
- Mazefsky, C. A., McPartland, J. C., Gastgeb, H. Z., & Minshew, N. J. (2013). Brief report: Comparability of DSM-IV and DSM-5 ASD research samples. *Journal of Autism and Developmental Disorders, 43*(5), 1236–1242.
- Norbury, C. F. (2014). Practitioner review: Social (pragmatic) communication disorder conceptualization, evidence and clinical implications. *Journal of Child Psychology and Psychiatry, 55*(3), 204–216.
- O'Neill, D. K. (2007). The language use inventory for young children: A parent-report measure of pragmatic language development for 18-to 47-month-old children. *Journal of Speech, Language, and Hearing Research, 50*(1), 214–228.
- Parsons, L., Cordier, R., Munro, N., Joosten, A., & Speyer, R. (2017). A systematic review of pragmatic language interventions for children with autism spectrum disorder. *PLoS One, 12*(4), e0172242.
- Phelps-Terasaki, T., & Phelps-Gunn, D. (2007). *The test for pragmatic language- 2*. Austin: Pro-Ed.
- Reichow, B., & Volkmar, F. R. (2010). Social skills interventions for individuals with autism: Evaluation for evidence-based practices within a best evidence synthesis framework. *Journal of Autism and Developmental Disorders, 40*, 149–166.
- Reisinger, L. M., Cornish, K. M., & Fombonne, É. (2011). Diagnostic differentiation of autism spectrum disorders and pragmatic language impairment. *Journal of Autism and Developmental Disorders, 41*(12), 1694–1704.
- Simms, M. D., & Jin, X. M. (2015). Autism, language disorder, and social (pragmatic) communication disorder: DSM-V and differential diagnoses. *Pediatrics in Review, 36*(8), 355–362.
- Swineford, L. B., Thurm, A., Baird, G., Wetherby, A. M., & Swedo, S. (2014). Social (pragmatic) communication disorder: A research review of this new DSM-5 diagnostic category. *Journal of Neurodevelopmental Disorders, 6*(1), 41.
- Timler, G., Olswang, L. B., & Coggins, T. E. (2005). “Do I know what I need to do?” A social communication intervention for children with complex clinical profiles. *Language, Speech, and Hearing Services in Schools, 36*, 73–85.
- van Agt, H., Verhoeven, L., van den Brink, G., & Koning, H. (2011). The impact on socio-emotional development and quality of life of language impairment in 8-year-old children. *Developmental Medicine and Child Neurology, 53*(1), 81–88.
- Whitehouse, A. J. O., Watt, H. J., Line, E. A., & Bishop, D. V. M. (2009). Adult psychosocial outcomes of children with specific language impairment, pragmatic language impairment and autism. *International Journal of Language and Communication Disorders, 44*, 511–528.

Pragmatic Language Skills Inventory

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Synonyms

PLSI

Description

The *Pragmatic Language Skills Inventory* (PLSI; Gilliam and Miller 2006) is a 45-item, standardized, norm-referenced, teacher-rating instrument designed to assess the pragmatic language abilities of children ages 5 through 12 years. It is based on long-standing theories of pragmatic language defined as use of language in context.

As an objective measurement scale, it allows educators to document progress and can also assist in targeting instruction and intervention goals for IEPs and Section 504 Plans. The PLSI can be used by teachers, speech-language pathologists, and early interventionists for further data collection and research.

Children are rated on the PLSI by a teacher or other professional (rater) who is familiar with his or her pragmatic skills compared to others of the same age and gender. The rater should, therefore, be familiar with the child's skill sets in different

school settings and as well as the abilities of average children of the same age and gender. The rater uses a 9-point scale to assess the child against three areas: personal interaction (initiating conversation, asking for help, participating in verbal games, and using appropriate nonverbal communicative gestures), social interaction (knowing when to talk and when to listen, understanding classroom rules, taking turns in conversations, and predicting consequences for one's behavior), and classroom interaction (using figurative language, maintaining a topic during conversation, explaining how things work, writing a good story, and using slang appropriately). Each of these areas represents a PLSI subscale. Each subscale consists of 15 items for a total of 45 items which can be completed in approximately 10 min.

PLSI scoring and interpretation is done by an examiner. This person may be a speech clinician, teacher, or education professional knowledgeable in assessment and basic psychometrics. Raw scores for each subscale can be converted to percentile ranks and standard scores using reference tables. The standards scores for each of the three subscales can also be summed to determine the child's pragmatic language index. The PLI is a standardized score ($M = 100$, $SD = 15$) and represents overall performance.

Historical Background

The PLSI is based on theories of pragmatic language by Bates (1976) and Snow (1994), which specify language in social context as a system of rules ensuring social appropriateness and the clarity of the speaker or author's intent. Snow specifies pragmatic language as a system of conversational rules. These rules include participating in conversation turn taking; telling the truth; offering new, relevant, and appropriate amounts of information to the listener; requesting appropriate information; providing the correct amount of background information in context; using unambiguous as well as figurative speech; as well as changing language appropriately to each social situation (Bates 1976). Gilliam and Miller developed the PLSI to assess school-age children in the above areas.

Psychometric Data

The PLSI was standardized on 1,175 school-age children, representative of population in regard to race, gender, rural or urban residence, ethnicity, family income, parent education, and disability.

The reliability of the PLSI was estimated using coefficient alpha (content sampling), test-retest (time sampling), and interrater reliability (correlation between raters). Coefficient alphas were calculated for each of the subscales by gender and disability status. All coefficient alphas for the PLSI's subscales exceed .95. Test-retest reliability was estimated to range between .78 and .91. Interrater reliability was estimated to range between .85 and .90.

The criterion-prediction validity of the PLSI was estimated by examining the relationship between the PLSI and the *Test of Pragmatic Language* (TOPL; Phelps-Terasaki and Phelps-Gunn 1992). Correlation coefficients ranged between .50 (large) and .86 (very large). Exploratory factor analysis indicated the items loaded as expected on both a single factor and on the three PLSI subscales. Correlation coefficients between the PLSI subscales and the total index score ranged from .75 to .86. Finally, the PLSI diagnostic subgroups were rated by teacher in expected ways. Individuals with intellectual disabilities exhibited the lowest scores and individuals identified as gifted and talented exhibited the highest scores.

The PLSI allows children's pragmatic language skills to be evaluated using norm-referenced scores. Scores can be used to determine if the child exhibits characteristics of a pragmatic language disorder and should be referred for a more comprehensive language assessment. Guidelines are provided to assist the examiner in interpreting the results. The PLSI can be used to identify children with pragmatic language disorders, document progress in ability, collect data for research, and target pragmatic language goals.

Clinical Uses

The PLSI is intended to be used in diagnosis of a pragmatic language disorder in conjunction with other assessment results, observations, case

histories, and parent interviews. It provides reliable and valid scores in order to differentiate children who have little likelihood of having a disorder.

The PLSI's objective scale of measurement allows educators to document a child's progress in pragmatic language ability. Administration is quick and simple enough to facilitate frequent use. The test can be used for special education and regular annual evaluations and in conjunction with other data in order to make transition and extended-year decisions.

The PLSI can help teachers and other professionals evaluate children's pragmatic language skills and assist in setting pragmatic language goals. Items from the PLSI can be utilized as specific targets in goal planning and intervention in Individualized Education Plans (IEPs) and Section 504 Plans.

References and Reading

- Bates, E. (1976). *Language and context: The acquisition of pragmatics*. New York: Academic Press.
- Gilliam, J. E., & Miller, L. (2006). *Pragmatic language skills inventory*. Austin: PRO-ED.
- Phelps-Terasaki, D., & Phelps-Gunn, T. (1992). *Test of Pragmatic Language*. Austin: PRO-ED.
- Snow, C. E. (1994). What is so hard about learning to read? A pragmatic analysis. In J. F. Duchan, L. E. Hewitt, & R. M. Sonnenmeier (Eds.), *Pragmatics: From theory to practice* (pp. 164–184). Englewood Cliffs: Prentice Hall.

Pragmatic Rating Scale

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Synonyms

[PRS-SA \(Pragmatic rating scale-school age\)](#)

Description

The Pragmatic Rating Scale-School Age (PRS-SA) is a assessment tool designed for documenting social communicative behaviors that violate pragmatic rules. It is designed for use with individuals aged 4 years through adulthood whose cognitive and linguistic functioning ranges from mild impairment through the gifted level. The definition of "pragmatics" used here is the set of rules governing the social use of language.

Pragmatics

Communication is a vital component of human interaction. It is one of the primary means of keeping people connected to each other. Communication may happen for a wide variety of purposes, such as informing someone (e.g., that you will be late), requesting permission, making a promise, teasing, showing something of interest, asking for information, greeting, negotiating, and so forth. To ensure that communication between people is coherent and socially appropriate, every culture has unwritten (tacit) rules for how communication is to transpire. The tacit rules that guide conversation, or social discourse, are known as the rules of pragmatic language behavior. Grice (1975) defined a set of maxims that, when adhered to, result in coherent conversation. These maxims are basic principles that enable conversational partners to organize and construct their communication behavior in ways that are interpretable and socially acceptable to each other, including such things as not saying more than is necessary, making sure that what you say is relevant (and that you indicate how and why it is relevant). When these maxims are systematically violated, people are signaling something to their conversational partner that serves to ensure the coherence of the communication. For example, when violating the maxim that requires speakers to say what they believe to be true, a speaker will signal through the tone of their voice or through facial expressions or gestures that their statement is not meant to be taken seriously (e.g., when we

exaggerate to make a point or to convey sarcasm). At times, violation of the Gricean maxims or pragmatic rules is permissible. For example, we may allow someone who is upset to monopolize a conversation, knowing that this is an extenuating circumstance. If, however, a particular person consistently monopolizes conversations, or if a person fails to systematically signal, in socially appropriate ways, their intentional violation of pragmatic rules, communication breakdown, and/or barriers to successful social interaction will arise. Such behavior may be indicative of a pragmatic language impairment.

Pragmatic Rating Scale-School Age

There are six scales within the PRS-SA: (1) Speech Acts; (2) Presupposition/Theory of Mind; (3) Discourse Management; (4) Speech and language behaviors; (5) Suprasegmental Speech Characteristics; and (6) Nonverbal Communicative Behaviors that affect Pragmatics. Within each of these scales, there are a number of items that provide a discrete set of communicative behaviors to be rated by the clinician. Ratings involve scores that range from 0 (unaffected; no evidence for pragmatic error involving this specific behavior or set of behaviors) to 2 (clear violation of pragmatic rules associated with the behavioral feature(s) being coded for this item). Within the Speech Acts scale, a checklist is provided on which clinicians record occurrences of the client's or patient's production of specific types of speech acts (communicative intents) and whether these were expressed in pragmatically appropriate or inappropriate ways. At the end of the PRS, two additional ratings are made that permit the clinician to provide an overall clinical judgment of the client's or patient's pragmatic behavior. The scores generated from the PRS include the summary scores for each scale, an overall composite score representing a sum of all the scale scores, and the clinical impression scores.

Scoring of the PRS-SA is based on a conversational sample that is designed to elicit a natural communicative exchange. Specific probes are

embedded into the conversation that provides opportunities to observe the full range of pragmatic behaviors rated within the PRS-SA. It may be used with already established behavior sampling procedures that include conversation segments between the clinician and child, such as the Autism Diagnostic Observation Scale – Generic, Modules 2 through 4 (Lord et al. 2001). It may also be used to rate pragmatic behavior over a number of interactive events so that clinicians may observe variability in pragmatic behavior linked to different types of contexts that involve a variety of social demands and supports, conversational partners, and social rules (e.g., playing games vs joint project development with peers; conversations with authority figures; conversations at lunchtime).

Historical Background

Pragmatic language disorders are not identifiable through existing cognitive assessment tools, nor through standard measures of grammatical and semantic language functioning. To assess pragmatic skill, an instrument is needed that enables clinicians and researchers to document pragmatic behavior in contexts that mimic true communicative exchanges. Pragmatic behavior, by definition, is dependent on context. In fact, it is the very ability to flexibly adapt one's communicative behavior to the context at hand, in socially appropriate ways, that defines healthy pragmatic functioning.

The PRS was originally developed in the early 1990s to document social discourse variations in a family study of autism. Soon after the PRS was developed, it began to be utilized to study pragmatic behavior in individuals with autism and to examine social discourse impairment as an endophenotype in other genetic disorders, including obsessive compulsive disorder. Next, the PRS was revised for use with school-aged children. In 2010, the PRS-School-Age version began to be used in a multi-site social skills intervention study focusing on elementary school-aged children with autism spectrum disorder (ASD). It is now being used to study the broader autism phenotype in

school-aged siblings of children with autism. Norms are also being established for children aged 4 through 12 years.

The PRS was developed because there was no other assessment instrument available for identifying individuals with social discourse difficulties. Many individuals with pragmatic impairments are viewed as socially odd and experience considerable social rejection, but because standard assessment tools do not assess pragmatic language functioning, these individuals often remain unidentified and, thus, untreated. The PRS was designed to facilitate identification of individuals with pragmatic impairment, providing a systematic method for defining the nature of their social communication difficulties and an objective basis for justifying intervention when needed. The PRS enables clinicians and researchers to document social discourse (pragmatic) variations or impairment, which is important for many types of endeavors such as identifying endophenotypes in genetic studies, identifying individuals with pragmatic disorders who are in need of intervention (or who may meet inclusion criteria for an intervention study), and educating family members, teachers, and employers about the nature of pragmatic difficulties that an individual is experiencing. Identifying and treating pragmatic impairment is important because such impairment may threaten success at the workplace, the ability to form and sustain friendships, the ability to gain access to social networks, and so forth.

Psychometric Data

At the time of preparation of this document (2011), the only psychometric data available on the PRS involves data presented in scientific manuscripts. Those data may be used to obtain general thresholds for PRS scores that fall in the “typical” range, though those studies focus primarily on adults and high-functioning individuals with autism spectrum disorders (including non-ASD controls). However, psychometric data are presently being compiled on children between the ages of 4 and 12 years. Thus, in the near future,

there will be empirical data on school-aged individuals with typical development, non-ASD delays, and ASD.

Clinical Uses

The PRS has a variety of clinical uses.

1. *Documenting the presence of a pragmatic language delay or impairment.* Elevated scores on the PRS, in combination with a speech-language pathologist’s clinical judgment of pragmatic language impairment, are likely to be indicative of impaired pragmatic ability and the need for intervention services. The PRS provides clinicians with a structured system for documenting communicative behaviors that may be contributing to breakdowns in social engagement and social communication. The operationally defined behaviors that are scored within the PRS provide clinicians with a systematic means of observing communicative behavior. The PRS items focus the clinician’s attention on dimensions of communication that otherwise may be missed or attributed to a different type of impairment. Documenting the types of communication behaviors that are problematic (based on the PRS items receiving a score of “1” or “2”), and the degree to which communicative behavior is disrupted for each PRS item, enables clinicians to glean information about whether a pragmatic impairment exists, and if so, how extensive and severe the impairment may be. The organization of communicative behaviors into different categories within the PRS (see “[Description](#)”) further enables clinicians to determine whether a child’s social communicative difficulties are primarily related to non-social processes (e.g., grammar, articulation), or are directly linked to difficulty with social inferencing, ability to link his/her ideas to the ideas of others through the use of linguistic devices, and so forth. Thus, the PRS is useful in clinical decision-making regarding need for intervention services and determination of intervention goals.

2. *Identifying specific behaviors that contribute to the pragmatic difficulties that an individual is displaying.* As indicated above, the specific types of items to be scored within the PRS permit documentation of the communicative behaviors that vary from the norm. For each PRS item, clinicians select a score ranging from 0 (within normal limits) to 2 (sign of abnormality). Clinicians may examine the items for which individuals received scores of 1 or 2, and look for patterns of communicative behavior that are problematic for their client or patient. By identifying specific aspects of pragmatic functioning that are difficult for an individual, clinicians may begin to define intervention goals and conversational supports, with the aim of improving the pragmatic language skills of the individual. Using the PRS, clinicians are able to objectively document specific pragmatic behaviors that are problematic as they help children or adults to develop greater levels of self-awareness and insight about their own communicative behavior. This is likely to enhance therapeutic response as individuals with pragmatic language difficulties often require explicit explanation of pragmatic rules, which are implicitly learned by most individuals. The operationally defined PRS items and objectively defined scoring system enable clinicians to discuss children's communication behavior with parents, helping them to develop a clearer understanding of their child's difficulties and how to develop strategies for helping their child achieve greater levels of success in communication.
3. *Documenting treatment response and progress.* The capacity of the PRS to document improvement in response to intervention has not been established, but is in the process of being evaluated through an intervention study. A short form of the PRS has been developed for use within treatment sessions to rate children's pragmatic behavior. This short PRS form is being studied to determine its utility for providing feedback to children on their advances in pragmatic behavior during and across intervention sessions, and for record-keeping purposes (e.g., progress notes).
4. *Communicating with teachers and family members about the client's needs.* As indicated above, the PRS provides a clear and objective means of describing the pragmatic behaviors and skills of an individual when providing test results to teachers and family members. This helps them to focus on specific behaviors rather than on a global impression of a socially challenged individual, thereby fostering the use of targeted skill-enhancing strategies.

See Also

- [Discourse Management](#)
- [Pragmatic Communication](#)
- [Pragmatic Language Impairment](#)
- [Pragmatics](#)
- [Presupposition](#)
- [Social Cognition](#)
- [Social Communication](#)

References and Reading

- Bailey, A., Le Couteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda, E., et al. (1995). Autism as a strongly genetic disorder: Evidence from a British twin study. *Psychological Medicine*, 25(1), 63–77.
- Cullen, B., Samuels, J., Grados, M., Landa, R., Bienvenu, O. J., Liang, K., et al. (1975). Logic and conversation. In P. Cole & J. Morgan (Eds.), *Syntax and semantics* (Vol. 3, pp. 41–58). New York: Academic.
- Grice, H. P. (1975). Logic and conversation. In P. Cole & J. Morgan (Eds.), *Syntax and semantics: Speech acts* (pp. 41–58). New York: Academic Press.
- Grossman, R. B., Carter, A., & Volkmar, F. (1997). Social behavior in autism. *Annals of the New York Academy of Sciences*, 807, 440–454.
- Grossman, R. B., Bemis, R. H., Skwerer, D. P., & Tager-Flusberg, H. (2010). Lexical and affective prosody in children with high-functioning autism. *Journal of Speech, Language & Hearing Research*, 53(3), 778–793.
- Harpur, J., Lawlor, M., & Fitzgerald, M. (2006). *Succeeding with interventions for Asperger's syndrome and adolescents*. London: Jessica Kingsley.
- Hurley, R. S., Losh, M., Parlier, M., Reznick, J. S., & Piven, J. (2007). The broad autism phenotype questionnaire. *Journal of Autism and Developmental Disorders*, 37(9), 1679–1690.
- Landa, R. (2000). Social language use in Asperger syndrome and high functioning autism. In A. Klin, F. F.

- Volkmar, & S. Sparrow (Eds.), *Asperger syndrome* (pp. 125–155). New York: Guilford.
- Landa, R., Piven, J., Wzorek, M., Gayle, J., Chase, G., & Folstein, S. (1992). Social language use in parents of autistic individuals. *Psychological Medicine*, 22(1), 245–254.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2001). *Autism Diagnostic Schedule-WPS (ADOS-WPS)*. Los Angeles: Western Psychological Services.
- Losh, M., & Piven, J. (2007). Social-cognition and the broad autism phenotype: Identifying genetically meaningful phenotypes. *Journal of Child Psychology and Psychiatry*, 48, 105–112.
- Paul, R., Orlovski, S., Marcinko, H. C., & Volkmar, F. (2009). Conversational behaviors in youth with high-functioning ASD and Asperger syndrome. *Journal of Autism and Developmental Disorders*, 39(1), 115–125.
- Piven, J., & Palmer, P. (1999). Psychiatric disorder and the broad autism phenotype: Evidence from a family study of multiple-incidence autism families. *The American Journal of Psychiatry*, 156(4), 557–563.
- Ruser, T. F., Arin, D., Dowd, M., Putnam, S., Winklosky, B., Rosen-Sheidley, B., et al. (2007). Communicative competence in parents of children with autism and parents of children with specific language impairment. *Journal of Autism and Developmental Disorders*, 37(7), 1323–1336.
- Saric, R., & Nestadt, G. (2008). Social and communication difficulties and obsessive-compulsive disorder. *Psychopathology*, 41(3), 194–200.

structure or form (phonology, morphology, syntax); content or meaning (semantics); and use of language in context (pragmatics; Bates 1976; Bloom and Lahey 1978). Difficulty with any of these language components – either singly or in combination – may result in a language disorder. Difficulty with pragmatics is one of the core features of autism spectrum disorder (American Speech-Language-Hearing Association [ASHA] n.d.).

Pragmatics is one aspect of a broader category known as *social communication* (Turkstra et al. 2016). Social communication is supported by an array of skills important to effective communication: social cognition (e.g., taking the perspective of another person, making inferences, understanding facial expressions); sociolinguistics (e.g., cultural influences, gender differences in communication, influences of different languages for multilingual speakers); and social interactions and relationships with peers (Adams 2005; Timler 2008; Timler et al. 2005). Pragmatics involves three main communication tasks: (a) using language for multiple purposes, (b) adapting language for different settings and communication partners, and (c) engaging in conversation and narrative discourse (Roth and Spekman 1984).

Pragmatics

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Synonyms

Language use; Pragmatic communication; Pragmatic language; Social cognition; Social communication; Social thinking; Use of language; Use of language in context

Definition

Effective communication requires mastery and integration of multiple components of language:

Use of Language for Different Purposes

Language is used for different reasons (e.g., greet, comment, ask, promise). By speaking certain utterances, certain actions are accomplished. Therefore, these purposes of speaking are known as *speech acts* (Austin 1962; Searle 1969).

Individuals with autism may use language in a more limited way. For example, they may not greet people, comment, or ask questions.

Adapting Language to Setting and Communication Partner

A pragmatic language disorder may be apparent when individuals do not use language appropriate to the setting or the communication partner. They may use the same language in all places and with all people. They may use a loud voice in a movie theater or restaurant. They may be impolite to their boss or abrupt with a grocery clerk.

Conversation and Narrative Discourse

Conversations require speakers to take turns; to appropriately initiate, maintain, and change conversational topics; to know when to interrupt; and to modify utterances following a misunderstanding. Nonverbal behaviors are also an integral part of conversations, such as using gestures and facial expressions and knowing how close to stand when speaking to another person. Speakers follow a set of conversational rules related to the quantity, quality, relevance, and manner in which they speak (Grice 1975).

Following conversation rules involves social cognition, which includes perspective-taking. The ability to take the perspective of another person is known as *theory of mind*. Speakers need to be aware of what their listener already knows. They need to know how much background information to provide.

Conversation problems, which may result from autism, may include difficulty following the rules for conversation-interrupting, not answering a question, bringing up topics unrelated to what is being discussed, or talking about only one subject. Speakers with autism also may experience problems in perspective-taking (ASHA n.d.). They may talk about people their communication partner does not know or omit important details.

Conversational use and narrative discourse vary across cultures. Therefore, it is critical to know the appropriate cultural expectations before determining that a particular pattern of conversation or storytelling is a pragmatic disorder.

Narrative discourse involves telling stories and talking about personal events. Speakers need to tell stories in a certain order and describe events in sequence.

Narrative discourse problems, which may result from autism, may involve telling a story out of sequence or omitting important parts.

Speech-language pathologists assess and treat pragmatic language disorders that may affect academics, self-esteem, behavior, and relationships.

Pragmatic language intervention is designed to capitalize on strengths and address weaknesses that affect language use and facilitate the individual's social activities and participation by assisting

the person to acquire new communication strategies (verbal and nonverbal). Intervention is expected to result in improved social functioning and participation (ASHA 2004).

See Also

- Pragmatic Communication
- Pragmatics
- Social Cognition
- Social Communication
- Social Thinking
- Speech-Language Pathologist (SLP)
- Theory of Mind

References and Reading

- Adams, C. (2005). Social communication intervention for school age children: Rationale and description. *Seminars in Speech and Language*, 26, 181–188.
- American Speech-Language-Hearing Association. (2004). *Preferred practice patterns for the profession of speech-language pathology*. Available from www.asha.org/policy
- American Speech-Language-Hearing Association (n.d.-a). *Autism* (Practice Portal). Available from www.asha.org/Practice-Portal/Clinical-Topics/Autism/
- American Speech-Language-Hearing Association. (n.d.-b). *Social communication disorders in childhood*. (Practice Portal). Available from www.asha.org/Practice-Portal/Clinical-Topics/Social-Communication-Disorders-in-School-Age-Children
- Austin, J. L. (1962). *How to do things with words*. Cambridge, MA: Harvard University Press.
- Bates, E. (1976). *Language and context: The acquisition of pragmatics*. New York: Academic Press.
- Bloom, L., & Lahey, M. (1978). *Language development and language disorders*. New York: Wiley.
- Grice, H. P. (1975). Logic and conversation. In P. Cole & J. L. Morgan (Eds.), *Speech acts* (pp. 41–58). New York: Academic Press.
- Roth, F. P., & Spekman, N. J. (1984). Assessing the pragmatic abilities of children: Part 1. Organizational framework and assessment parameters. *Journal of Speech and Hearing Disorders*, 49, 2–11.
- Searle, J. (1969). *Speech acts: An essay in the philosophy of language*. Cambridge, UK: Cambridge University Press.
- Timler, G. R. (2008). Social knowledge in children with language impairments: Examination of strategies, predicted consequences, and goals in peer conflict situations. *Clinical Linguistics & Phonetics*, 22, 741–763.

- Timler, G., Olswang, L., & Coggins, T. (2005). "Do I know what I need to do?" a social communication intervention for children with complex clinical profiles. *Language, Speech, and Hearing Services in Schools*, 36, 73–85.
- Turkstra, L. S., Clark, A., Burgess, S., Hengst, J. A., Wertheimer, J. C., & Paul, D. (2016). Pragmatic communication abilities in children and adults: Implications for rehabilitation professionals. *Disability and Rehabilitation*, 19, 1–14.

Praise

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Definition

Praise is the act of delivering positive statements by individuals in regard to a person, object, action, or idea. Praise can be delivered publicly so others have the ability to see or hear it or in private. Praise is often awarded after the completion of a task or an accomplishment. Praise can be delivered verbally, gesturally, and visually. Some examples of praise are saying "Good job" or "Way to go" and "I like how you are sitting so tall and steady," giving a thumbs-up, or providing a cue card that says "Nicely done."

See Also

- [Positive Reinforcement](#)

References and Reading

- Cooper, J., Heron, T., & Heward, W. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson Education.
- Lomas, J., Fisher, W., & Kelley, M. (2010). The effects of variable time delivery of food items and praise on problem behavior reinforced by escape. *Journal of Applied Behavior Analysis*, 43, 425–435.
- Malott, R., Malott, M., & Trojan, E. (2000). *Elementary principles of behavior* (4th ed.). Upper Saddle River: Prentice-Hall.

Praxis

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Synonyms

[Motor planning](#)

Definition

Praxis refers to the brain's ability to develop, organize, and carry out the necessary steps to complete an unfamiliar task or a series of tasks (Ayres 1973). Ayres (1972, 1979) noted that effective motor planning is dependent on an individual's ability to integrate information from the sensory systems, including visual, proprioceptive, vestibular, and tactile stimuli. Three components of praxis have been defined as ideation, planning, and execution (Ayres 1973, 1985). Ideation is defined as generating a plan of action (Parham and Mailloux 2001) and can impact an individual's ability to fluidly perform motor actions, particularly in new situations or with novel materials. Planning and execution further allow the individual to plan and anticipate body movements necessary for action and can incorporate sequencing a series of steps when necessary. Children with autism spectrum disorders may show difficulty within the domain of praxis, resulting in awkward motor execution, poor pretend and imaginative play skills, and compromised sequencing abilities for new and novel situations.

See Also

- [Ayres, A. Jean](#)
- [DeGangi-Berk Test of Sensory Integration](#)
- [Sensory Integration and Praxis Test](#)

References and Reading

- Ayres, A. (1972). *Sensory integration and learning disabilities*. Los Angeles: Western Psychological Services.
- Ayres, A. (1973). *Sensory integration and learning disorders*. Los Angeles: Western Psychological Services.
- Ayres, A. (1979). *Sensory integration and the child*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (1985). *Developmental dyspraxia and adult onset apraxia*. Torrance: Sensory Integration International.
- Parham, L. D., & Mailloux, Z. (2001). Sensory integration. In J. Case-Smith (Ed.), *Occupational therapy for children* (4th ed.). St. Louis: Mosby.

Pre-, During, and Posttest Design

► Qualitative Versus Quantitative Approaches

Precentral Gyrus

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Synonyms

[Brodmann's area 4](#); [Motor cortex](#); [Primary motor cortex](#)

Definition

The precentral gyrus (PCG), also known as the motor strip or primary motor cortex, is the part of the brain's neocortex responsible for executing voluntary movements. This gyrus is located in the most posterior portion of the frontal lobe, lying immediately anterior to (in front of) the central sulcus, and extending from the apex of the brain down to the sylvian fissure, which delineates the temporal lobe. Histologically, neurons in the precentral gyrus have a similar architecture and are denoted as Brodmann's area 4. The

functional organization of the PCG follows a topographical representation of an inverted homunculus (small human), such that the head and face regions are represented at the lowermost portion of the gyrus, the body and limbs extend toward the upper part of the gyrus, and the feet "dangle" over the apex of the brain. Areas of increased fine motor dexterity, especially the lips, tongue, and hands, have a far more extensive representation on the motor strip, while areas with little motor dexterity such as the torso have a minimal motor representation. In motor cortex, the left hemisphere controls the right side of the body and right brain controls the left body. Lesions to the motor cortex will result in an inability to move that portion of the body represented in the homunculus.

While there is little evidence that primary motor cortex plays a key role in autism, there is some evidence of motor cortex abnormalities. Behaviorally, many individuals with autism have motor deficits (e.g., Jansiewicz et al. [2006](#)), though other brain regions such as pre-motor cortex, cerebellum, and basal ganglia may contribute to these deficits. Several brain imaging studies in autism have found differences in precentral gyrus morphology including increased white matter (Mostofsky et al. [2007](#); Mengotti et al. [2011](#)) and decreased cortical thickness (Cauda et al. [2011](#); Scheel et al. [2011](#)). Decreased connectivity between motor cortex and cerebellum during motor performance (Mostofsky et al. [2009](#)) may indicate that motor abnormalities in autism may reflect a deficit in how brain areas involved in movement coordinate their activity.

See Also

- [Motor Control](#)
- [Motor Planning](#)

References and Reading

- Cauda, F., Geda, E., Sacco, K., D'Agata, F., Duca, S., Geminiani, G., & Keller, R. (2011). Grey matter abnormality in autism spectrum disorder: an activation

- likelihood estimation meta-analysis study. *Journal of Neurology, Neurosurgery, and Psychiatry*, 82(12), 1304–1313.
- Jansiewicz, E. M., Goldberg, M. C., Newschaffer, C. J., Denckla, M. B., Landa, R., & Mostofsky, S. H. (2006). Motor signs distinguish children with high functioning autism and asperger's syndrome from controls. *Journal of Autism and Developmental Disorders*, 36(5), 613–621.
- Mengotti, P., D'Agostini, S., Terlevic, R., De Colle, C., Biasizzo, E., Londero, D., Ferro, A., Rambaldelli, G., Balestrieri, M., Zanini, S., Fabbro, F., Molteni, M., & Brambilla, P. (2011). Altered white matter integrity and development in children with autism: A combined voxel-based morphometry and diffusion imaging study. *Brain Research Bulletin*, 84(2), 189–195.
- Mostofsky, S. H., Burgess, M. P., & Gidley Larson, J. C. (2007). Increased motor cortex white matter volume predicts motor impairment in autism. *Brain*, 130(8), 2117–2122.
- Mostofsky, S. H., Powell, S. K., Simmonds, D. J., Goldberg, M. C., Caffo, B., & Pekar, J. J. (2009). Decreased connectivity and cerebellar activity in autism during motor task performance. *Brain*, 132(9), 2413–2425.
- Scheel, C., Rotarska-Jagiela, A., Schilbach, L., Lehnhardt, F. G., Krug, B., Vogeley, K., & Tepest, R. (2011). Imaging derived cortical thickness reduction in high-functioning autism: Key regions and temporal slope. *NeuroImage*, 58(2), 391–400.
- on frequency data (Merbitz et al. 2004a). More specifically, precision teaching requires that instructors set a specific measureable and observable goal, select a method of instruction, conduct daily assessments of the learner's performance, and chart the number of correct and incorrect responses on a standard celeration chart. The core strength of precision teaching lies in the scientific rigor that it brings to the field of education (Kubina and Yurich 2009). Teachers have the opportunity when using this approach to observe, describe, record, and analyze a behavior, allowing them to make informed decisions concerning the effectiveness of the current method of teaching. This constant progress monitoring and charting provides the instructor with an easy-to-read, graphic representation of the learner's progress that can be used to develop an effective instructional program. If the student is learning at a rate that allows him or her to reach the set goal in the specified time frame, the instructor can conclude that the current instructional strategy is effective and continue with that program. If the learner is not progressing at the desired pace, the instructor can supplement or change the instructional strategy in an attempt to facilitate the learner's progress (Kubina and Yurich 2009). Precision teaching can be used with and is believed to enhance the efficacy of a wide variety of educational or treatment programs (Johnson and Street 2012; Kubina and Yurich 2009; Lindsley 1992).

Preciseness

► Perfectionism

Precision Teaching

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Definition

Precision teaching is a behavioral evaluation method for instruction and curricula that focuses

Historical Background

Ogden Lindsley and his colleagues began developing precision teaching in the 1960s (Lindsley 1964). At this time, behavioral techniques (e.g., schedules of reinforcement and punishment, response shaping, stimulus control) were commonly used in educational settings (Lindsley 1964). While generally effective, these strategies did not allow all students to reach the desired level of achievement.

Instead of focusing on specific behavioral techniques, Lindsley applied behavioral measurement and charting procedures to educational programs (Binder and Watkins 1990; Lindsley 1964).

More specifically, Lindsley and his colleagues used assessments of active and observable behavior, frequency counts, and charting to examine individual student progress. It was believed that using this behavioral measurement system would provide instructors with the information required to discover an effective instructional strategy for each learner.

Collecting frequency data enabled early precision-teaching researchers to discover the importance of rate of correct responding. Eric Haughton and his colleagues found that both accuracy and speed of responding influenced the acquisition of more advanced skills (Haughton 1980). Based on this finding, precision teaching came to emphasize the importance of learning each skill to fluency, or the accuracy and rate of responding required to make that skill functional (for more information on fluency, please see section “Goals and Objectives”).

Although not widely implemented, educators and researchers continue to develop precision-teaching manuals, tools, and fluency databases. Charting applications are also now available to allow teachers to electronically chart their student’s progress (Johnson and Street 2012). Research on precision teaching is published in *The Journal of Precision Teaching and Celeration* and other peer-reviewed journals.

Rationale or Underlying Theory

Precision teaching is based in the belief that all learners can succeed with the appropriate support. Unlike traditional educational programs, this perspective attributes failure to the program rather than the learner. If a learner is not succeeding, the instructor must change the environment (e.g., the type of instruction, intensity of instruction) to better support learner progress. A central component to the underlying theory of precision teaching is the “Try Again” principle, which states that when a method is not effective, the teacher needs to continue to modify or change the method until the learner is successful at the task (Kubina and Yurich 2009).

Creating appropriate learning environments means that the instructor must know when a

learner is struggling, the environmental variables that were in place, and what changes need to be made. Precision teaching provides a systematic framework for identifying and replacing ineffective teaching strategies. This is believed to facilitate the development of effective programs and learner progress.

Goals and Objectives

Precision teaching is intended to maximize learner progress by monitoring their response to intervention and modifying the instructional strategies as necessary. It is a content-free method of evaluating program effectiveness. As such, it can be and has been used to address a wide range of academic (e.g., reading words, solving addition problems), social (e.g., answering questions, making eye contact), and other (e.g., aggressive behaviors, welding) issues and skills (Kubina and Yurich 2009; Ramey et al. 2016).

Regardless of the program goal, precision teaching recognizes that both the learner’s ability to demonstrate a skill and the time it takes him or her to do so influence his or her ability to use this skill in real-world situations. Therefore, precision teaching emphasizes that the individual learn a skill to fluency, or gains a level of accuracy or quality and speed required to make a skill functional (Fabrizio and Moors 2003; Johnson and Street 2012). Individuals who learn a skill to fluency are more likely to retain the skill following intervention; engage in the skill long enough to complete real-world tasks; and demonstrate that skill in new situations, distracting environments, and in combination with related skills (Fabrizio and Moors 2003; Kerr et al. 2003).

Treatment Participants

Precision teaching has been used with a diverse range of learners. This educational method has been implemented in programs involving teaching skills to typically developing, gifted and developmentally disabled individuals (Cavallini et al. 2010; Johnson and Street 2012; Kubina et al. 2009). A focus of current research has

been on the implementation of precision teaching to interventions for individuals with autism. (Johnson and Street 2012; Kubina and Yurich 2009; Ramey et al. 2016).

Autism treatment programs have implemented precision teaching with students as a way to track each individual's progress and to evaluate the effectiveness of the students' current treatment plan. The daily assessment and charting of the learners progress allow for the teacher to be aware of any adjustments that need to be made in a timely manner (Kubina and Yurich 2009; Merbitz et al. 2004b). Individuals with ASD often fail to maintain the learned behaviors over time or apply them to different settings. Precision teaching's emphasis on fluency (see section "[Treatment Procedures](#)" for more detail) addresses these common issues (Kubina et al. 2002).

It has been suggested that it may be particularly important to use precision teaching with lower-functioning individuals (Merbitz et al. 2004b). By definition, these individuals are further behind their peers and are especially in need of effective instruction if they are to reach the normative level of achievement. More information is needed to identify individual characteristics associated with rate of learner progress.

Treatment Procedures

Precision teaching is an ongoing process of goal setting, assessment, charting, and behavioral skills training. The goals, assessments, charts, and program decisions are generally developed and completed by one or more instructors. However, when those receiving the treatment are able, they may assess and chart their own progress and contribute to the goal setting and program decisions (Lindsley 1992). Precision teaching requires that each individual learner has their own goals, assessments, and charts, but has been implemented to whole classes or treatment groups (Lindsley 1992). The main components of precision teaching (pinpointing, counting, and charting) are described below.

Pinpointing

Pinpointing refers to the identification of the targeted behavior. Regardless of the program goal, the instructor should identify an active and observable behavior that can be assessed to monitor learner progress (Kerr et al. 2003). For example, if a program is intended to increase a learner's addition skills, an appropriate measure would be the number of problems the learner correctly answers in 1 min. In other cases, the measureable behavior may be less obvious. For instance, the instructor may be trying to increase a learner's silent reading. The instructor cannot directly measure how many words a learner reads silently and will have to develop a less direct behavioral measure. The instructor may ask the learner to answer questions to determine his or her comprehension of the silently read passage (White 1986).

In pinpointing the targeted behavior, the instructor must select a learning channel. The learning channel refers to the type of instruction the individual receives and the type of response he or she is expected to demonstrate (Kerr et al. 2003). For example, an individual's program intended to increase the learner's vocabulary could use a hear/give or see/say learning channel. The learning channel hear/give could involve the instructor saying a word and the individual giving the instructor the corresponding picture, while the learning channel see/say would involve the instructor showing the individual the picture and the individual verbally labeling it. Other learning channels include hear/do, hear/say, see/do, and see/match (Fabrizio and Moors 2003; Kerr et al. 2003).

These different learning channels allow teachers to select the learning channel that best facilitates a particular individual's learning style. This may be especially relevant to the development of effective programs for individuals with autism, who do not always have the speech or academic skills required to say or write answers (Kerr et al. 2003).

Once the learning channel is established, the instructor must define the program goal and the correct and incorrect responses. Because of precision teaching's emphasis on fluency (see section

“Goals and Objectives”), goals are stated as a frequency aim (i.e., the number of correct responses within a unit of time) that is likely to lead to skill application, maintenance, endurance, stability, and generalization (Johnson and Street 2012). Researchers have compiled a database of frequency aims that have been associated with retention, endurance, stability, and application across a range of skills for individuals with autism (Fabrizio and Moors 2003). Although there is individual variability, instructors can consult this and other databases when developing frequency aims.

Counting

Progress is monitored by counting demonstrated behavior. The instructor selects a teaching strategy and a teaching time (e.g., discrete trial training for 15 min a day) and an assessment period (e.g., 1 min). The learner receives the instruction and then participates in an assessment. During the assessment period, the learner is given the opportunity to demonstrate the targeted behavior, and the instructor counts the number of correct and incorrect responses. Unlike traditional percent correct measures, counting the number of correct and incorrect responses allows the instructor to determine the accuracy and frequency of response.

Charting – The Standard Celeration Chart

Instructors monitor learner progress with standard celeration charts, which have days along the x-axis and frequency counts along the y-axis. In other words, a change from 10 to 20 correct responses looks the same as the change from 20 to 40. Each day, the instructor marks the number of correct and incorrect responses demonstrated during the assessment period.

The **standard celeration charts** differ from other measurement instruments because of the implementation of a ratio scale, as opposed to an equal interval scale, for measuring frequency (Johnson and Street 2012). Using a ratio scale allows for teachers to measure the learner’s relative change, or growth, over time (Johnson and Street 2012). On standard celeration charts, the frequency count axis is scaled to show changes

in the rate of progress instead of number of responses. This means that a change of 10–20 will look the same as a change of 20–40 because the individual has doubled their performance in both cases (Kubina et al. 2002).

Regularly charting learner performance in this way provides teachers with a clear picture of learner progress. If the chart shows a steep and steady rate of learning, the instructor can conclude that the existing program is sufficient. However, if the individual’s progress flattens or will not allow the individual to meet his or her goal by the established timeline, the instructor should modify the program to better facilitate learning.

Efficacy Information

There is evidence that precision teaching increases learner progress with typical, low-performing, and developmentally disabled students. A commonly referenced series of pivotal studies in the field were conducted in Great Falls, Montana. These studies found that typical and low-performing elementary and high school students who were taught with precision teaching tend to make more progress and score higher on measures of math and writing skills than assigned or matched control groups (White 1986).

Current research in the field has found similar results concerning the benefits of using precision teaching with participants ranging from elementary school students to graduate university students (Kubina et al. 2009; Lydon et al. 2017). A study conducted in Pennsylvania with over 200 students found a positive effect on the reading skill development of low-performing elementary school students when they were taught word reading skills through a direct instruction reading program combined with precision teaching (Kubina et al. 2009). Another study involved using precision teaching to enhance medical students’ procedural skills. The researchers found that the medical students who were taught with the simulation-based intervention that involved precision teaching demonstrated higher accuracy

with their performance of the skill compared to the control groups (Lydon et al. 2017).

There are a number of case studies indicating that individuals with disabilities including learning disabilities and ASD do make progress and meet set goals when their instructors use precision teaching strategies (Lolich et al. 2012; Schirmer et al. 2007; Travis et al. 2012). A few more extensive and methodologically rigorous research has also provided support for the potential of implementing precision teaching with individuals with ASD (Ramey et al. 2016; Weiss et al. 2008). Weiss et al. (2008) examined archival data on the skill maintenance and frequency building of 38 individuals with ASD. This study was the first published data set that supported the efficacy of skill interventions using precision teaching to increase the frequency of responses in children and adults with autism (Weiss et al. 2008).

Ramey et al. (2016) provided additional support for integrating precision teaching into interventions with individuals with autism and other developmental disabilities. This article provided a systematic review of 55 studies that all incorporated the use of precision teaching when targeting skill acquisition in individuals with developmental disabilities. The authors evaluated the empirical support of the precision teaching approach using the National Autism Center's 2009 (2015) National Standards Report guidelines, which has recently been updated to the National Standards Project, Phase II (2015). These guidelines classify treatment interventions into four categories: ineffective/harmful, unestablished, emerging, and established. Ramey et al. (2016) stated that according to these standards, precision teaching is currently classified as an emerging treatment for teaching a wide range of skills to individuals with autism. More high quality studies demonstrating the beneficial effects of precision teaching with this population are needed before it can be classified as an established treatment.

Outcome Measurement

Individual performance is measured via frequent assessments (Johnson and Street 2012; Kubina

and Yurich 2009). These assessments are specific to the targeted skill (e.g., if the targeted skill is labeling objects, the assessment will only measure the learner's labeling of objects). Data from each are charted on the previously described standard celeration charts and provide a graphic representation of the learner's progress (see section "Treatment Procedures" for more detail). For more general indicators of learner progress, instructors can use validated and appropriate assessment tools.

Qualifications of Treatment Providers

Precision teaching can be utilized by general and special education teachers, clinicians, para-professionals, or any other individual attempting to teach skills. There are a number of resources available to individuals interested in learning and implementing precision teaching, including treatment manuals, workshops, and fluency databases (e.g., Kubina and Yurich 2009; Lindsley 1992). There are no training, credentials, or licensure requirements for individuals implementing precision teaching.

See Also

- [Behavioral Assessment](#)
- [Behaviorism](#)
- [Education](#)
- [Educational Interventions](#)

References

- Binder, C., & Watkins, C. L. (1990). Precision teaching and direct instruction: Measurably superior instruction technology in schools. *Performance Improvement Quarterly*, 3(4), 74–96.
- Cavallini, F., Berardo, F., & Perini, S. (2010). Mental retardation and reading rate: Effects of precision teaching. *Life Span and Disability*, 13, 87–101.
- Fabrizio, M. A., & Moors, A. L. (2003). Evaluating mastery: Measuring instructional outcomes for children with autism. *European Journal of Behavior Analysis*, 4, 23–36.

- Haughton, E. C. (1980). Practicing practices: Learning by activity. *Journal of Precision Teaching*, 1, 3–20.
- Johnson, K., & Street, E. M. (2012). *Response to intervention and precision teaching: Creating synergy in the classroom*. New York: Guilford Publications.
- Kerr, K. P., Smyth, P., & McDowell, C. (2003). Precision teaching children with autism: Helping design effective programs. *Early Child Development and Care*, 173(4), 399–410.
- Kubina, R. M., & Yurich, K. K. L. (2009). Developing behavioral fluency for students with autism: A guide for parents and teachers. *Intervention in School and Clinic*, 44, 131–138.
- Kubina, R. M., & Yurich, K. K. L. (2012). *The precision teaching book*. Lemont: Greatness Achieved.
- Kubina, R. M., Morrison, R., & Lee, D. L. (2002). Benefits to adding precision teaching to behavioral interventions for students with autism. *Behavioral Intervention*, 17, 233–246.
- Kubina, R. M., Commons, M. L., & Heckard, B. (2009). Using precision teaching with direct instruction in a summer school program. *Journal of Direct Instruction*, 9, 1–12.
- Lindsley, O. R. (1964). Direct measurement and prosthesis of retarded behavior. *Journal of Education*, 147, 62–81.
- Lindsley, O. R. (1992). Precision teaching: Discoveries and effects. *Journal of Applied Behavior Analysis*, 25, 51–57.
- Lolich, E., McLaughlin, T. F., & Weber, K. P. (2012). The effects of using reading racetracks combined with direct instruction precision teaching and a token economy to improve the reading performance for a 12-year-old student with learning disabilities. *Academic Research International*, 3, 245–252.
- Lydon, S., Burns, N., Healy, O., O'Connor, P., McDermott, B. R., & Byrne, D. (2017). Preliminary evaluation of the efficacy of an intervention incorporating precision teaching to train procedural skills among final cycle medical students. *BMJ Simulation and Technology Enhanced Learning*. Published Online First: 13 February 2017. <https://doi.org/10.1136/bmjstel-2016-000154>
- Merbitz, C., Vieitez, D., Merbitz, N., & Pennypacker, H. S. (2004a). Precision teaching: Foundations and classroom applications. In D. J. Moran & R. W. Malott (Eds.), *Evidence-based educational methods* (pp. 47–61). San Diego: Elsevier/Academic Press.
- Merbitz, C., Vieitez, D., Merbitz, N. H., & Binder, C. (2004b). Precision teaching: Applications in education and beyond. In D. J. Moran & R. W. Malott (Eds.), *Evidence-based educational methods* (pp. 63–80). San Diego: Elsevier/Academic Press.
- National Autism Center (2009, 2015). National Standards Report. Randolph: National Autism Center
- Ramey, D., Lydon, S., Healy, O., McCoy, A., Holloway, J., & Mulhern, T. (2016). A systematic review of the effectiveness of precision teaching for individuals with developmental disabilities. *Review Journal of Autism and Developmental Disorders*, 3, 179–195.
- Schirmer, K., Almon-Morris, H., Fabrizio, M. A., Abrahamson, B., & Chevalier, K. (2007). Using precision teaching to teach storytelling to a young child with autism. *Journal of Precision Teaching and Celeration*, 23, 23–26.
- Travis, J., McLaughlin, T. F., Derby, K. M., & Carosella, M. (2012). The differential effect of race-track procedures for saying letter sounds by two first-grade students with learning disabilities. *Academic Research International*, 2, 372–382.
- Weiss, M. J., Fabrizio, M., & Bamond, M. (2008). Skill maintenance and frequency building: Archival data from individuals with autism spectrum disorders. *Journal of Precision Teaching and Celeration*, 24, 28–37.
- White, O. R. (1986). Precision teaching. *Exceptional Children*, 52, 522–534.

Predictive Value of Screening Measures

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Definition

The predictive value of a screening instrument refers to the accuracy of a screening measure. It measures whether an individual actually has a disease. The predictive value can be positive or negative. A positive predictive value (PPV) refers to the percentage of people who screen positive for the condition who actually have the condition. Whereas, the negative predictive value (NPV) is the percentage of people who screen negative for the condition and do not have the condition. The predictive value of screening instruments is determined by sensitivity, specificity, and prevalence of a condition.

See Also

- [Prevalence](#)
- [Sensitivity and Specificity](#)

Predictors at Toddlerhood for Long-Term Severity of ADHD Symptoms in Autism Spectrum Disorder

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Definition

Characteristics among toddlers with autism spectrum disorder that predict adolescent attention deficit/hyperactivity symptom severity.

Historical Background

Autism spectrum disorder (ASD) and attention deficit hyperactivity disorder (ADHD) are two of the most common neurodevelopmental disorders, affecting approximately 2% and 7%, respectively (Lyall et al. 2017; Willcutt 2012), of the general population. ASD and ADHD share an overlapping genetic load (Rommelse et al. 2010), and both disorders show a high occurrence in the family and have male predominance (Musser et al. 2014). ASD is defined by social communication deficits and restricted and repetitive behaviors (RRB) [American Psychiatric Association (APA) 2013], while ADHD is characterized by symptoms of inattention, impulsivity, and hyperactivity (APA 2013). Both disorders have significant long-term, multi-domain impairments; thus, early identification is critical for ASD and for ADHD.

ADHD is the most common comorbidity diagnosed in children and adolescents with ASD (59% to 83%) (van der Meer et al. 2012). Previous studies have examined the presence of ADHD symptoms in ASD, describing more severe ASD symptoms, higher rates of cognitive impairment,

more deficits in adaptive skills, a negative effect on family life, and lower quality of life in individuals with ASD and ADHD in comparison to ASD alone (Avni et al. 2018; Sikora et al. 2012; Sprenger et al. 2013).

Some researchers consider ADHD in the context of ASD as two different phenotypes (ASD alone vs ASD + ADHD), while others propose that within the ASD spectrum, ADHD is a less severe subtype (van der Meer et al. 2012).

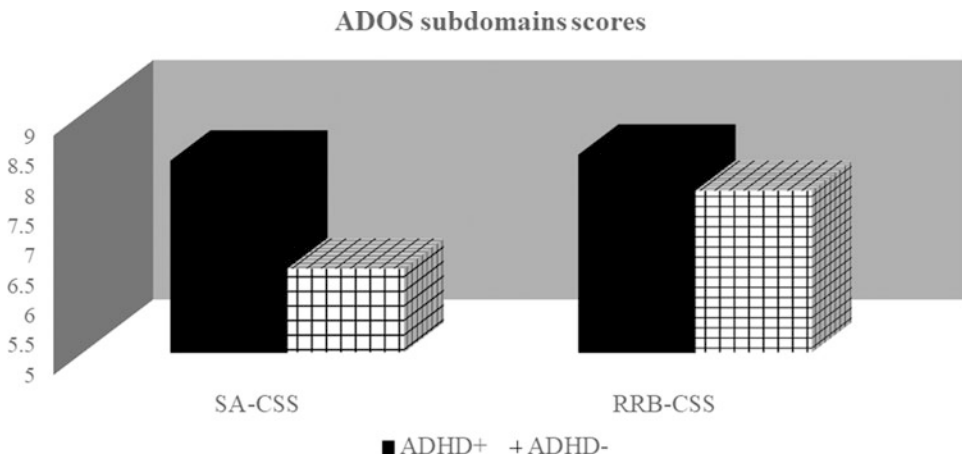
Recognizing significant ADHD symptoms in children and youth with ASD has important clinical implications. This allows for a variety of pharmacological and non-pharmacological interventions for ADHD that differ from those for ASD alone. When ADHD is identified in school-aged children with ASD, it allows for the implementation of psychoeducational rehabilitation for those with inattention and executive function deficits, which may affect academic achievement (Happé et al. 2006). For those with behavioral difficulties resulting from hyperactivity and impulsivity symptoms, behavioral interventions that are the mainstay intervention in ADHD can be applied to individuals with ASD to improve their functioning. Target areas can include psychoeducation – encouragement and structure, working memory and cognitive flexibility training, classroom modifications, and developing strategies to handle daily living challenges. In addition, classroom and behavioral modifications, along with self-regulation strategies, are all evidence-based elementary school interventions for managing ADHD and can be applied to ASD + ADHD. Targeted behavioral interventions for ASD and ADHD, such as parental management training to improve the severity of ADHD symptoms, had a positive correlation with the improvement of ASD symptom severity (Manohar et al. 2018). In contrast, failure to recognize or late recognition of ADHD can seriously impact educational and social functioning, worsen mood dysregulation, and increase the occurrence of disruptive behaviors (Biederman et al. 2014).

Thus, finding predictors at toddlerhood for the later occurrence of ADHD symptoms and early identification of ADHD in children diagnosed with ASD is of great importance.

Current Knowledge

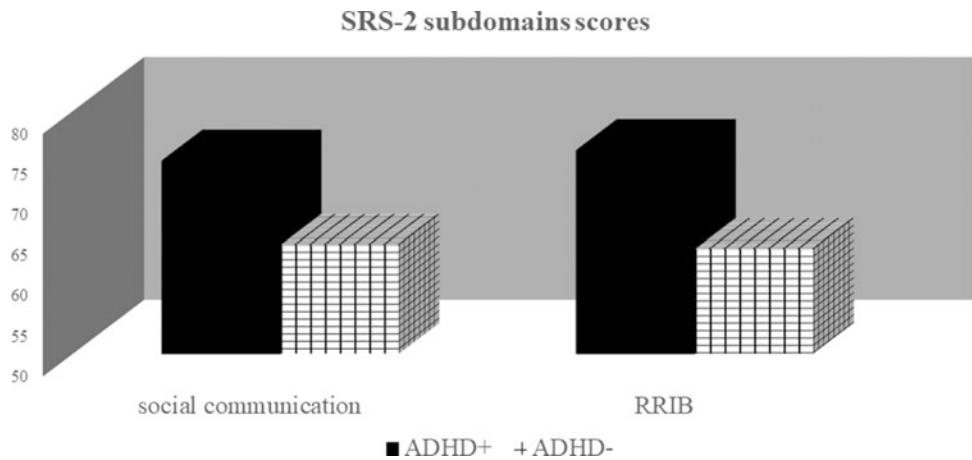
One long-term follow-up study on a population diagnosed with ASD at toddlerhood investigated the frequency of elevated ADHD symptoms and the characteristics of those with ASD and ADHD. In addition, the study searched for toddler characteristics that may predict ADHD symptom severity at adolescence (Zachor and Ben-Itzhak 2019). This study included 65 participants, 60 males and 5 females (mean age = 13:8y), diagnosed with ASD at toddlerhood (prior to age 38 months). At the time of diagnosis, the children underwent a comprehensive assessment that included behavioral, cognitive, and adaptive skills and neurological assessments. Diagnosis of ASD was based on results of standardized tests for ASD and on clinical judgment. At follow-up, after 8–16 years (mean, 11:7y), extensive evaluations of cognitive ability, adaptive skills, autism severity, and ADHD symptoms using standardized tests were performed. Participants were divided into two groups with the comorbidity (ADHD+; $n = 39$) and without (ADHD-; $n = 26$). The ADHD+ group included participants who met the cutoff scores according to both parents and teachers on a standardized ADHD questionnaire (Conners 3; Conners et al. 2011) on at least one scale and those who received medication for ADHD. This

longitudinal study found high frequency (60%) of elevated ADHD symptoms in adolescents diagnosed with ASD at toddlerhood as reported in previous studies on ADHD comorbidity in ASD (Avni et al. 2018; Murray 2010). This longitudinal study found that adolescents with elevated ADHD symptoms diagnosed at toddlerhood with ASD showed a more impaired clinical presentation. Having ASD and elevated ADHD symptoms was characterized by more severe ASD symptoms in social communication and RRB domains (Autism Diagnostic Observation Schedule-2 (ADOS-2); Lord et al. 2012), as assessed by professionals (Fig. 1) and reported by parents (Social Responsiveness Scale (SRS); Constantino and Gruber 2012) (Fig. 2). In addition, the group with ASD and elevated ADHD symptoms had lower cognitive ability overall and specifically lower verbal comprehension, perceptual reasoning, working memory, and processing speed (Wechsler Intelligence Scale for Children WISC-IV-; Wechsler 2010) (Fig. 3). It is worth noting that more than half of the participants with elevated ADHD symptoms had IQ scores in the intellectual disability (ID) range. The ASD and ID comorbidity may explain the more severe clinical presentation in autism severity and adaptive skills. In addition, having ASD and ADHD resulted in more impaired communication and

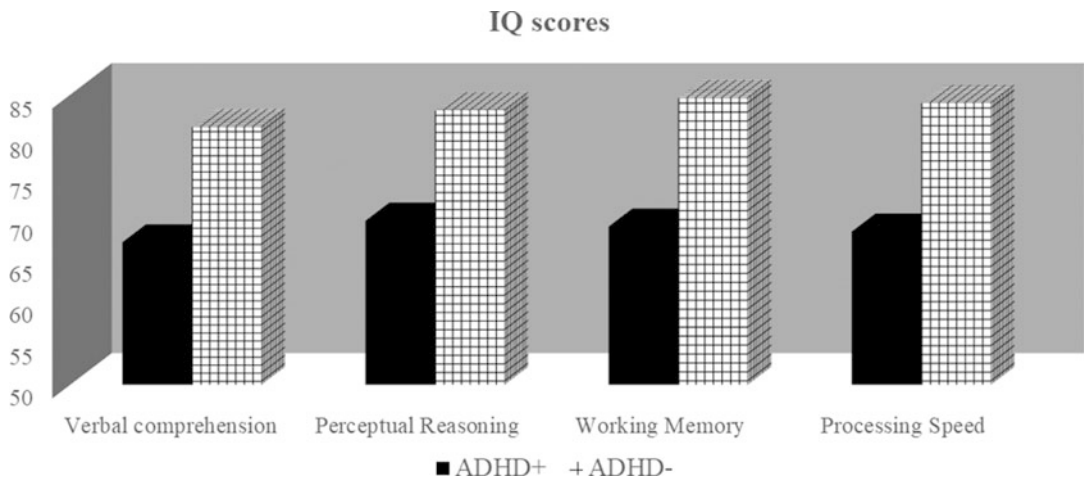


Predictors at Toddlerhood for Long-Term Severity of ADHD Symptoms in Autism Spectrum Disorder, Fig. 1 Autism severity measured by the Autism Diagnostic Observation Schedule (ADOS-2) calibrated severity

scores (CSS) in social affect (SA-CSS) and RRB (RRB-CSS) domains in adolescents with ASD with and without ADHD (assessed by professionals)



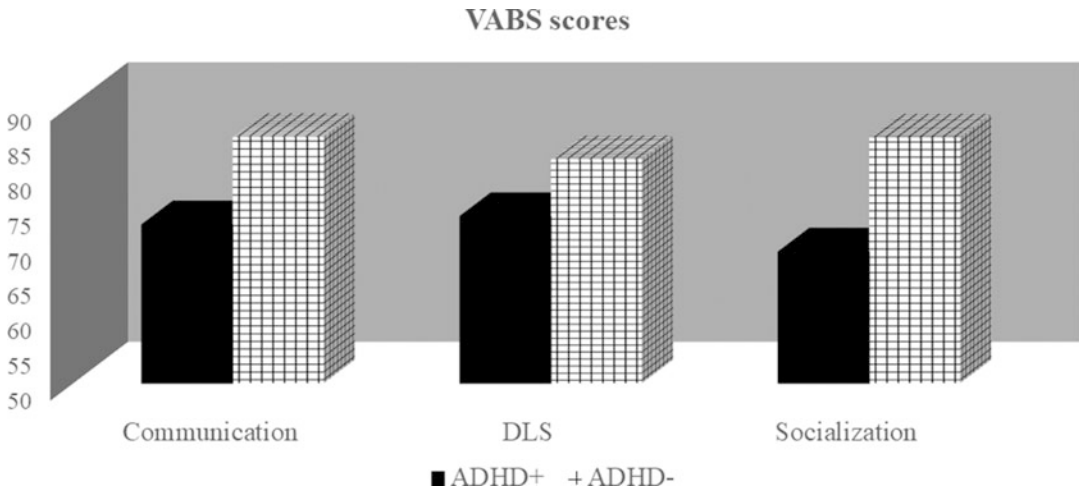
Predictors at Toddlerhood for Long-Term Severity of ADHD Symptoms in Autism Spectrum Disorder, Fig. 2 Autism severity as measured by the Social Responsiveness Scales (SRS-2) scores in social communication and repetitive and restricted interests behaviors (RRIB) in adolescents with ASD with and without ADHD (completed by parents)



Predictors at Toddlerhood for Long-Term Severity of ADHD Symptoms in Autism Spectrum Disorder, Fig. 3 IQ scores in adolescents with ASD with and without ADHD

social functioning (Vineland Adaptive Behavior Scales (VABS); Sparrow et al. 2005) (Fig. 4). These findings supported previous studies that compared individuals with ASD alone to those with ASD and ADHD. These studies reported greater autism severity and more impairments in verbal and nonverbal abilities, spatial working memory, response inhibition, executive functions, and adaptive functioning in all the VABS subdomains for those with ASD and ADHD (Rao

and Landa 2014). Overall, these findings suggest that having elevated ADHD symptoms is characterized by mild global deficits in functioning that affect individuals with ASD. In the above long-term follow-up study, a large portion (87%) of the population with ASD with elevated ADHD symptoms used medication regularly, mainly antipsychotics and stimulants, and about 20% used SSRIs regardless of the severity of the ADHD symptoms (Zachor and Ben-Itzhak



Predictors at Toddlerhood for Long-Term Severity of ADHD Symptoms in Autism Spectrum Disorder, Fig. 4 Adaptive skills scores measured by the Vineland

Adaptive Behavior Scales (VABS) in adolescents with ASD with and without ADHD

2019). This frequency is higher than the medication adherence rates of 57.1–71.4% for individuals diagnosed with ADHD without ASD in the general population (Gajria et al. 2014). It is possible that having both ASD and elevated ADHD symptoms that significantly impact functioning encourages the use of medication to improve certain behavioral symptoms.

In light of the high prevalence of ADHD in the general population and more so in ASD, an important research question is if there are early characteristics that may precede later occurrence of ADHD. Only a handful of studies have examined predictors in infants in the general population that may later be associated with an ADHD diagnosis. Early temperamental differences, such as reduced inhibitory control and attention, increased activity level, and more negative affect were documented in infants at risk for ADHD (Sullivan et al. 2015). Lower developmental quotient at 15- and 24-month follow-ups and a lower receptive language level at 36 months were associated with the reported severity of ADHD symptomatology in third grade (Arnett et al. 2013). Regulation and temperament challenges at 6–36 months of age were related to elevated parent-rated ADHD symptoms in first grade (Willoughby et al. 2017). However, since the diagnostic outcomes of participants in these studies were not known,

it was not clear which infant behaviors would predict a later diagnosis of ADHD. Another study identified several risk factors that were associated with the severity of ADHD symptoms in children with ASD with a mean age of 7:6 years. These variables included motor delay, history of enuresis and allergies, intellectual disability, and a family history of intellectual disability and psychosis (Lamanna et al. 2017).

Regarding ADHD in ASD, only one study so far has specifically examined variables in toddlers diagnosed with ASD that predict severity of ADHD symptoms in adolescence (Zachor and Ben-Itzhak 2019). This prospective study followed a cohort diagnosed with ASD at toddlerhood to adolescence and focused on the association between early cognitive and adaptive skills and ASD symptom severity in toddlerhood and the severity of ADHD symptoms in adolescence and searched for variables in toddlerhood that may predict the severity of ADHD symptoms in adolescence. More severe ASD symptoms in social affect and RRB domains as assessed by professionals (ADOS-2) and lower communication adaptive skills (VABS) in toddlerhood correlated with more severe inattention and hyperactivity/impulsivity symptoms (Conners 3) in adolescence. In addition, lower social adaptive behavior skills in toddlerhood were associated

with more severe hyperactivity/impulsivity symptoms in adolescence. When examining the contribution of cognitive ability, autism severity, and adaptive skills variables assessed during the toddler years for the prediction of later ADHD symptom severity, the severity of restricted and repetitive behaviors (RRB) predicted the severity of inattention and hyperactivity/impulsivity symptoms at adolescence. In addition, early overall adaptive skills predicted the severity of later hyperactivity/impulsivity symptoms.

Although ADHD is diagnosed routinely and more definitively in school-aged children, these findings of more severe RRB and poorer adaptive skills in toddlers diagnosed with ASD may represent early red flags for the later development of elevated ADHD symptoms.

Several studies found a strong association between hyperactive-impulsive ADHD traits and RRB at evaluation time. At the phenotypic level, inattention symptoms correlated with severity of RRB and with social interaction and communication deficits, whereas hyperactivity/impulsivity symptoms were more strongly associated with RRB (Ghirardi et al. 2019).

Anatomical and physiological changes in specific brain structures (basal ganglia, striatum), abnormal connectivity pathways, as well as neurotransmitter dysfunction may be associated with RRB in ASD (Abbott et al. 2018).

Several conclusions can be drawn from these studies. First, clinicians who diagnose ASD in toddlers should pay special attention to red flags such as severe RRB and poor functioning as early prognostic signs for later elevated ADHD symptoms, which should be followed closely. Second, the importance of evaluating symptoms of ADHD in ASD is emphasized as this comorbidity is very common in ASD. In particular, it is important to pay special attention to the presence of severe ADHD symptoms in populations with ASD and a more severe clinical presentation. Finally, there is a negative impact of having elevated ADHD symptoms in addition to ASD. Lower cognition, more debilitating ASD symptoms, and poorer adaptive skills in ASD with ADHD result in more challenging needs. Therefore, comprehensive medical, behavioral, and academic interventions and close follow-up are crucial for

adolescents with ASD who present with elevated ADHD symptoms. It will be important to implement interventions for those with ASD and ADHD to decrease inattention/impulsivity related difficulties, which may likely have a beneficial effect on social functioning. The same applies to the treatment of hyperactivity, which may lessen repetitive behaviors. Pharmacological and behavioral treatments that are known to improve ADHD symptomatology may also prove beneficial in ASD symptom severity and functioning.

Future Directions

The findings described above suggest abnormalities in specific brain regions and connectivity pathways, as well as neurotransmitter dysfunction, occur in both ADHD and RRBs. Combining this common neurobiological basis and the behavioral findings with the strong association between RRB and elevated ADHD symptoms may in the future create a basis for developing possible methods of intervention.

In light of the early predictors found at toddlerhood for later occurrence of elevated ADHD symptoms, it is important to develop early focused intervention to reduce RRB levels in toddlers with ASD and target adaptive skills deficits as early as possible. In future research, it will be important to examine whether these early interventions improve the severity of ADHD symptoms in adolescence.

In addition, it is important to explore early predictors for other comorbidities, such as anxiety and depression, two common disorders in ASD. It will also be important to search for other variables, including family characteristics and early behaviors such as emotion dysregulation and maladaptive internalizing/externalizing behaviors, which may predict or be associated with a later occurrence of elevated ADHD symptoms.

The high prevalence of ADHD in ASD, its impact on functioning, and the high adherence to medication require further examination of the effectiveness of known classes of drugs designed to treat ADHD is required. In addition, searching for a new, more effective drug for this special phenotype of ASD with elevated ADHD

symptoms and evaluating the effect on symptom severity of both disorders are highly needed.

In sum, ADHD symptoms are very common in ASD. Adolescents diagnosed at toddlerhood with ASD with elevated ADHD symptoms at adolescence have lower cognitive and adaptive skills and more severe autism symptoms. Early toddler behaviors, such as more severe restricted and repetitive behaviors (RRB) and lower overall functioning, predict the severity of ADHD symptoms in adolescence. Early awareness of red flags for ADHD in ASD and consequently providing focused interventions in various developmental and functional domains will improve the well-being and functioning of adolescents with ASD and ADHD.

References and Reading

- Abbott, A. E., Linke, A. C., Nair, A., Jahedi, A., Alba, L. A., Keown, C. L., et al. (2018). Repetitive behaviors in SRS are linked to imbalance of corticostriatal connectivity: A functional connectivity MRI study. *Social Cognitive and Affective Neuroscience*, 13, 32–42. <https://doi.org/10.1093/scan/nsx129>.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders, revised* (5th ed.). Washington, DC: Author.
- Arnett, A. B., Macdonald, B., & Pennington, B. F. (2013). Cognitive and behavioral indicators of ADHD symptoms prior to school age. *Journal of Child Psychology and Psychiatry*, 54(12), 1284–1294. <https://doi.org/10.1111/jcpp.12104>.
- Avni, E., Ben-Itzhak, E., & Zachor, D. A. (2018). The presence of comorbid ADHD and anxiety symptoms in autism spectrum disorder: Clinical presentation and predictors. *Frontiers in Psychiatry*, 9, 1–12. <https://doi.org/10.3389/fpsy.2018.00717>.
- Ben-Itzhak, E., Ben-Shachar, S., & Zachor, A. D. (2013). Specific clinical phenotypes in autism spectrum disorders are associated with gender representation. *Autism Research*, 6, 596–604.
- Biederman, J., Petty, C., Spencer, T. J., Woodworth, K. Y., Bhide, P. G., Zhu, J., et al. (2014). Is ADHD a risk for posttraumatic stress disorder (PTSD)? Results from a large longitudinal study of referred children with and without ADHD. *World Journal of Biological Psychiatry*, 15(1), 49–55.
- Conners, C. K., Pitkanen, J., & Rzepa, S. R. (2011). Conners 3rd edition (Conners 3; Conners 2008). In J. S. Kreutzer, J. DeLuca, & B. Caplan (Eds.), *Encyclopedia of clinical neuropsychology*. New York: Springer.
- Constantino, J. N., & Gruber, C. P. (2012). *Social responsiveness scale, second edition (SRS-2)*. Los Angeles: Western Psychological Services.
- Gajria, K., Lu, M., Sikirica, V., Greven, P., Zhong, Y., Qin, P., et al. (2014). Adherence, persistence, and medication discontinuation in patients with attention-deficit/hyperactivity disorder – A systematic literature review. *Neuropsychiatric Disease and Treatment*, 10, 1543.
- Ghirardi, L., Pettersson, E., Taylor, M. J., Freitag, C. M., Franke, B., Asherson, P., et al. (2019). Genetic and environmental contribution to the overlap between ADHD and ASD trait dimensions in young adults: A twin study. *Psychological Medicine*, 49(10), 1713–1721.
- Happé, F., Booth, R., Charlton, R., & Hughes, C. (2006). Executive function deficits in autism spectrum disorders and attention-deficit/hyperactivity disorder: Examining profiles across domains and ages. *Brain and Cognition*, 61, 25–39.
- Lamanna, A. L., Craig, F., Matera, E., Simone, M., Buttiglione, M., & Margari, L. (2017). Risk factors for the existence of attention deficit hyperactivity disorder symptoms in children with autism spectrum disorders. *Neuropsychiatric Disease and Treatment*, 13, 1559–1567.
- Lord, C., Rutter, M., DiLavore, P. C., Risi, S., Gotham, K., & Bishop, S. (2012). *Autism diagnostic observation schedule, second edition*. Torrance: Western Psychological Services.
- Lyall, K., Croen, L., Daniels, J., Fallin, M. D., Ladd-Acosta, C., Lee, B. K., et al. (2017). The changing epidemiology of autism spectrum disorders. *Annual Review of Public Health*, 38, 81–102.
- Manohar, H., Patnaik, P., Kandasamy, P., Chandrasekaran, V., & Philip, R. (2018). Implications of comorbid ADHD in ASD interventions and outcome: Results from a naturalistic follow up study from South India. *Asian Journal of Psychiatry*, 33, 68–73. <https://doi.org/10.1016/j.ajp.2018.03.009>.
- Murray, M. J. (2010). Attention-deficit/hyperactivity disorder in the context of autism spectrum disorders. *Current Psychiatry Reports*, 12(5), 382–388. <https://doi.org/10.1007/s11920-010-0145-3>.
- Musser, E. D., Hawkey, E., Kachan-Liu, S. S., Lees, P., Roullet, J. B., Goddard, K., et al. (2014). Shared familial transmission of autism spectrum and attention deficit/hyperactivity disorders. *Journal of Child Psychology and Psychiatry*, 55(7), 819–827.
- Rao, P., & Landa, R. J. (2014). Association between severity of behavioral phenotype and comorbid attention deficit hyperactivity disorder symptoms in children with autism spectrum disorders. *Autism*, 18(3), 272–280. <https://doi.org/10.1177/1362361312470494>.
- Rommelse, N. N. J., Franke, B., Geurts, H. M., Hartman, C. A., & Buitelaar, J. K. (2010). Shared heritability of attention-deficit/hyperactivity disorder and autism spectrum disorder. *European Child and Adolescent Psychiatry*, 19(3), 281–295. <https://doi.org/10.1007/s00787-010-0092-x>.
- Sikora, D. M., Vora, P., Coury, D. L., & Rosenberg, D. (2012). Attention-deficit/hyperactivity disorder symptoms, adaptive functioning, and quality of life in children with autism spectrum disorder. *Pediatrics*, 130 (Supplement), S91–S97.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales* (2nd ed.). Circle Pines: American Guidance Service.

- Sprenger, L., Bühler, E., Poustka, L., Bach, C., Heinzel-Gutenbrunner, M., Kamp-Becker, I., et al. (2013). Impact of ADHD symptoms on autism spectrum disorder symptom severity. *Research in Developmental Disabilities*, 34(10), 3545–3552.
- Sullivan, E. L., Holton, K. F., Nousen, E. K., Barling, A. N., Sullivan, C. A., Propper, C. B., et al. (2015). Early identification of ADHD risk via infant temperament and emotion regulation: A pilot study. *Journal of Child Psychology and Psychiatry*, 56(9), 949–957. <https://doi.org/10.1111/jcpp.12426>.
- van der Meer, J. M., Oerlemans, A. M., van Steijn, D. J., Lappenschaar, M. G., de Sonnevile, L. M., Buitelaar, J. K., et al. (2012). Are autism spectrum disorder and attention-deficit/hyperactivity disorder different manifestations of one overarching disorder? Cognitive and symptom evidence from a clinical and population-based sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(11), 1160–1172.
- Wechsler, D. (2010). *WISC-IV HEB. Wechsler intelligence scale for children-fourth edition. (Hebrew version)*. Psychtech: Jerusalem.
- Willcutt, E. G. (2012). The prevalence of DSM-IV attention-deficit/hyperactivity disorder: A meta-analytic review. *Neurotherapeutics*, 9(3), 490–499. <https://doi.org/10.1007/s13311-012-0135-8>.
- Willoughby, M. T., Gottfredson, N. C., & Stifter, C. A. (2017). Observed temperament from ages 6 to 36 months predicts parent- and teacher-reported attention-deficit/hyperactivity disorder symptoms in first grade. *Development and Psychopathology*, 29(1), 107–120.
- Zachor, D. A., & Ben-Itzhak, E. (2019). From toddlerhood to adolescence: Which characteristics among toddlers with autism spectrum disorder predict adolescent attention deficit/hyperactivity symptom severity? A long-term follow-up study. *Journal of Autism and Developmental Disorders*, 49(8), 3191–3202. <https://doi.org/10.1007/s10803-019-04042-9>.

Predictors of Clinician Certainty in the Diagnosis of Autism

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Definition

Clinician certainty is one aspect of diagnostic decision-making that refers to how sure clinicians

are that their diagnosis of autism spectrum disorder (ASD) was accurate. In other words, when clinicians decide to make a diagnosis of ASD, how sure are they that the diagnosis is correct and that the individual truly has autism?

Historical Background

Early and accurate diagnosis of autism spectrum disorder (ASD) is a critical healthcare priority, as earlier identification of ASD facilitates earlier entry into intervention services that increase the likelihood of positive developmental trajectories across the lifespan (Fountain et al. 2012). Earlier identification of ASD is also linked with increased family satisfaction with the diagnostic process (Crane et al. 2016). Thus, the need for timely diagnosis of ASD is imperative.

However, timely identification of ASD continues to be challenging, and the process of obtaining a diagnosis is fraught with difficulties for individuals and their families. For example, although symptoms of autism can be observed as early as infancy and reliable diagnoses can be made before age 3 (Guthrie et al. 2013), the majority of individuals are not identified with ASD until school age or later (Shattuck et al. 2009). Moreover, a growing number of individuals are receiving a diagnosis of ASD for the first time in adulthood, missing opportunities throughout their childhood and adolescence for adequate support and self-understanding. Importantly, there are disparities in access to early diagnosis, as child sex, socioeconomic status, and race/ethnicity all predict the timeliness of receiving an autism diagnosis (Nowell et al. 2015). Given these difficulties, it is unsurprising that the majority of caregivers and individuals seeking an ASD diagnosis report being highly dissatisfied with the diagnostic process.

Clinician difficulty in assessing and diagnosing ASD with certainty may be one factor contributing to challenges in the diagnostic process, as there is no singular “test” for ASD. Rather, ASD is a complex, heterogeneous neurodevelopmental disorder for which differential diagnosis can be difficult (Matson et al. 2012). Typically, best practice ASD assessment

involves integrating information from a number of sources that may yield conflicting results, including self-report, caregiver report, teacher report, observation, and performance on standardized tests (Kim and Lord 2012). Although standardized screening and assessment instruments have greatly improved our ability to identify and discriminate ASD, these measures do not have perfect sensitivity and specificity. Even very experienced clinicians using standardized assessment instruments may ultimately be uncertain regarding an individual's diagnosis, which could lead to delays in making a final diagnosis. The current entry first reviews current knowledge on clinician certainty, summarizes predictors of clinician certainty, and concludes with future directions for this critical area of study.

Current Knowledge

What is clinician certainty? Clinician certainty is one aspect of diagnostic decision-making that refers to how sure clinicians are that their diagnosis was accurate. In other words, when clinicians decide to make a diagnosis of ASD, how sure are they that the diagnosis is correct and that the individual truly has autism? Prior work has conceptualized clinician certainty using dichotomous (“I am absolutely certain about this diagnosis” versus not) and continuous (e.g., ranging from “absolutely certain not ASD” to “absolutely certain ASD”) rating scales (Hedley et al. 2016; McDonnell et al. 2019). Clinician certainty expands upon the construct of “frank presentations” of ASD, which refers to the phenomenon wherein clinicians can recognize distinctive behavioral presentations characteristic of ASD almost immediately (de Marchena and Miller 2017).

Levels of clinician certainty. Preliminary research suggests that a significant portion of ASD diagnoses are made with some level of uncertainty. Among a sample of young children referred for ASD diagnostic evaluations, 40% of diagnoses were made with some level of uncertainty (McDonnell et al. 2019). Such lack of certainty may negatively impact the diagnostic

process. For example, if clinicians are uncertain regarding the accuracy of an ASD diagnosis, there may be longer latencies to making a final diagnosis, and families may be less receptive to the diagnosis and its associated treatments. Thus, understanding factors that predict clinician certainty can identify individuals at risk for under-identification, prolonged diagnostic pathways, and negative diagnostic experiences and can also aid in targeting efforts designed to improve assessment processes.

Sex and clinician certainty. A range of demographic factors may relate to clinician certainty, including child sex, age, and broader family-level demographic factors. There has been much interest in understanding how to improve diagnostic practices among females given growing evidence that the disproportionate ratio of males to females diagnosed with ASD is partially due to a diagnostic sex bias (Loomes et al. 2017). Females with ASD may be under-identified due to difficulties identifying the female autism phenotype, which may be more subtle than presentations characteristic of males (Lai et al. 2017). Providers and parents consistently cite difficulties with the diagnostic process for females (Mademtzi et al. 2018; Penner et al. 2017), and an increasing number of women do not receive their diagnosis until late adolescence or adulthood (Bargiela et al. 2016). However, recent work has not found that clinician certainty relates to child sex (McDonnell et al. 2019). Future research is needed in this critical area of study to examine diagnostic decision-making for females presenting with symptoms of ASD.

Age and clinician certainty. Child age may also relate to clinician certainty in the diagnosis of ASD, although research findings are mixed. One study using a sample of youth under age 6 reported that clinicians were most certain when diagnosing the youngest children (McDonnell et al. 2019). It is possible that certainty is higher for the youngest children, because children who are already being seen for a diagnostic evaluation at a young age likely present with the most severe and/or obvious symptoms. Moreover, as children get older, differential diagnosis may become more complicated as clinicians increasingly need to consider a child's behavior in multiple settings and rule out

a range of behavioral, emotional, and developmental diagnoses. However, other research has not found an association between age and diagnostic certainty (Hedley et al. 2016), emphasizing the need for future research in this area.

Demographics and clinician certainty. Diagnostic certainty may also relate to broader sociodemographic characteristics. Strikingly, children's insurance status has been linked with clinician certainty, with diagnostic certainty over two times more likely for children on private compared to public (Medicaid) insurance in the United States (McDonnell et al. 2019). It is important to note that this finding held even when controlling for other demographic factors and the level of autism symptoms reported on gold standard measures such as the Autism Diagnostic Observation Schedule (ADOS), supporting that insurance status plays a significant role in diagnostic decision-making. This suggests that clinician judgment may be influenced by socioeconomic status (SES), as insurance status is one broad indicator of SES that relates to race, parental education, and family income. Thus, finding that Medicaid status was linked with lower diagnostic certainty extends a large body of work demonstrating disparities in autism identification among racially diverse and/or lower SES children (Nowell et al. 2015; Fountain et al. 2012). Future research aimed at explicating *why* diagnostic certainty relates to indicators of SES is essential for reducing healthcare disparities in this area.

Clinical characteristics and clinician certainty. Diagnostic certainty also relates to the ultimate diagnostic conclusion made by the diagnostician as well as to clinical characteristics of the individual presenting for an evaluation. Clinicians are much more likely to report being certain when they confirm that a diagnosis of ASD is present, compared to when they rule out ASD in favor of another or no diagnosis. In one study, 70% of diagnoses were classified as certain when a diagnosis of ASD was made, compared to only 30% when a diagnosis of ASD was not made (McDonnell et al. 2019). This indicates that it is easier for clinicians to positively identify ASD compared to ruling it out and suggests that clinicians are likely to detect some behaviors or

symptoms of autism even among children not diagnosed with ASD. Clinician certainty has also been related to level of autism symptoms, with certainty the lowest for those presenting with a moderate level of symptoms relative to those presenting with high or minimal symptoms (McDonnell et al. 2019). Higher clinician certainty also relates to lower IQ, suggesting that diagnostic decision-making may be more uncertain for children with higher levels of cognitive abilities (Hedley et al. 2016).

Parent report and clinician certainty. When considering a diagnosis of autism spectrum disorder, clinicians are advised to take information from multiple sources, such as parents, teachers, and clinical observation, into account. As a result, the degree to which informants agree on ASD symptoms is a relevant factor in the prediction of clinician diagnostic certainty. Evidence suggests only a moderate level of agreement between parents' report of ASD symptoms and clinicians' observed symptoms. Several studies find modest agreement between the ADOS, the gold standard clinician-administered diagnostic measure, and the ADI-R, a gold standard parent diagnostic interview. For example, de Bildt et al. (2004) note only fair agreement between the ADOS and the ADI-R, with agreement being higher for children ages 5–8 than older children. Similarly, in a study of 12-month-old infants at high- and low-risk for developing ASD, Macari et al. (2018) found a moderate agreement between parent report of ASD symptoms and clinician observation ($r = 0.34$). Another study found no relationship between the ADOS severity score and parent ratings on the Social Responsiveness Scale (SRS; Reszka et al. 2014). Given that clinician ratings and parent reports do not tend to be highly correlated, it is important to consider how clinician diagnostic certainty may be impacted by a contrasting parent report.

Teacher report and clinician certainty. It is also important to consider how teacher reports of ASD symptoms relate to parent or clinician accounts, given the importance of the school system in identifying and providing services for children with ASD. In a study of children ages 1–12 years by Mayes and Lockridge (2018),

mother, teacher, and psychologist reports on the Checklist for Autism Spectrum Disorders were compared. Although psychologists rated children with ASD as having more symptoms than did mothers or teachers, there was a high degree of correlation between mother and psychologist reports. However, mother-teacher and psychologist-teacher reports were not correlated. Similarly, another study found only a small to moderate correlation (weighted $r = 0.27$) between parent and teacher reports of social skills in children with ASD and/or intellectual disability (Stratis and Lecavalier 2015). Interestingly, a third study found a correlation of 0.418 between the teacher-rated SRS and the ADOS severity score, reflecting the necessity of additional work in this area (Reszka et al. 2014). It is important to consider the effect that informant discrepancy may have on clinician certainty. Disagreement between parents, teachers, and clinicians may impact the degree of uncertainty that clinicians feel during the diagnostic process. As such, it is critical that future research examines the effects of informant discrepancy in depth, with a particular emphasis on its relationship with clinician certainty.

Future Directions

Future research on the process of diagnostic decision-making in the context of ASD, including ongoing assessment of predictors of clinician certainty, has the exciting potential to advance our understanding of the nature of ASD as a disorder and also improve the diagnostic process. First, research on diagnostic certainty can advance our understanding of the nature of ASD. Recent debates about how to best conceptualize psychopathology have emerged in the adult and childhood psychopathology literatures (Kotov et al. 2017). For example, to what extent should psychological disorders be characterized as discrete categories versus continuous dimensions? Although autism has been minimally considered within this debate, ASD has increasingly been understood as a spectrum wherein characteristics of ASD are present to a certain extent even within

the general population (Lord and Jones 2012). Understanding how clinicians perceive and diagnose ASD has the potential to inform this ongoing debate. For example, finding that clinicians tend to be at least moderately certain when positively identifying ASD yet are very *uncertain* when ruling it out suggests that requiring a categorical decision (e.g., a child either has ASD or does not) may be quite difficult and may not adequately capture the nature of presenting autism symptoms.

Second, the limited body of work on diagnostic certainty thus far highlights the pressing need for ongoing research into improving the diagnostic process. It is alarming that children's health insurance status predicts clinician certainty even when clinicians have access to results from standardized assessment results such as the ADOS. Understanding how clinicians perceive autism symptoms in diverse cultural groups and *why* diagnostic decisions tend to be more uncertain for families on public-funded insurance programs such as Medicaid is essential for addressing disparities in diagnostic identification. Moreover, why is the overlap between parent and teacher report so small for some youth, and how should clinicians weight reports from different informants when attempting to summarize assessment results into one diagnostic conclusion? Future evaluations of the strength of associations between different assessment methods have important potential in this area. Lastly, much work remains to be done in the area of psychometrics, including developing empirically validated measures that can be disseminated for use by clinicians from all training backgrounds.

Third and finally, how can we improve clinician diagnostic certainty and their skills in differential diagnosis of ASD? Promising approaches have tested the value of providing mentoring and consultation to clinicians remotely over telecommunication, such as the ECHO programs (Mazurek et al. 2019). Expanding these types of training approaches into other healthcare settings, and integrating ASD assessment training universally into graduate training programs, could also prove instrumental in improving clinicians' ability to recognize and diagnose ASD with confidence. Understanding and improving clinician

decision-making in ASD has the potential to increase the timeliness of the diagnostic process and can result in a more positive experience for the youth or adult receiving the diagnosis of ASD. Thus, the overall goal of this research is to reduce disparities in the diagnostic process for underserved individuals and their families so that timely, confident, and more certain diagnoses can be provided equitably for all families.

See Also

- [Clinical Assessment](#)
- [Demographics and the Age of Autism Diagnosis](#)
- [Diagnosis and Classification](#)
- [Diagnostic Process](#)
- [Early Diagnosis](#)
- [Psychological Assessment](#)

References and Reading

- Bargiela, S., Steward, R., & Mandy, W. (2016). The experiences of late-diagnosed women with autism spectrum conditions: An investigation of the female autism phenotype. *Journal of Autism and Developmental Disorders*, 46(10), 3281–3294.
- Crane, L., Chester, J. W., Goddard, L., Henry, L. A., & Hill, E. (2016). Experiences of autism diagnosis: A survey of over 1000 parents in the United Kingdom. *Autism*, 20(2), 153–162.
- De Bildt, A., Sytema, S., Ketelaars, C., Kraijer, D., Mulder, E., Volkmar, F., & Minderaa, R. (2004). Interrelationship between autism diagnostic observation schedule-generic (ADOS-G), autism diagnostic interview-revised (ADI-R), and the diagnostic and statistical manual of mental disorders (DSM-IV-TR) classification in children and adolescents with mental retardation. *Journal of Autism and Developmental Disorders*, 34(2), 129–137.
- de Marchena, A., & Miller, J. (2017). “Frank” presentations as a novel research construct and element of diagnostic decision-making in autism spectrum disorder. *Autism Research*, 10(4), 653–662.
- Fountain, C., Winter, A. S., & Bearman, P. S. (2012). Six developmental trajectories characterize children with autism. *Pediatrics*, 129(5), e1112–e1120.
- Guthrie, W., Swineford, L. B., Nottke, C., & Wetherby, A. M. (2013). Early diagnosis of autism spectrum disorder: Stability and change in clinical diagnosis and symptom presentation. *Journal of Child Psychology and Psychiatry*, 54(5), 582–590.
- Hedley, D., Brewer, N., Nevill, R., Uljarević, M., Butter, E., & Mulick, J. A. (2016). The relationship between clinicians’ certainty and accuracy, and the influence of child characteristics, in the screening of autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(7), 2340–2348.
- Kim, S. H., & Lord, C. (2012). Combining information from multiple sources for the diagnosis of autism spectrum disorders for toddlers and young preschoolers from 12 to 47 months of age. *Journal of Child Psychology and Psychiatry*, 53(2), 143–151.
- Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., . . . Eaton, N. R. (2017). The hierarchical taxonomy of psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology*, 126(4), 454.
- Lai, M. C., Lombardo, M. V., Ruigrok, A. N., Chakrabarti, B., Auyeung, B., Szatmari, P., & MRC AIMS Consortium. (2017). Quantifying and exploring camouflaging in men and women with autism. *Autism*, 21(6), 690–702.
- Loomes, R., Hull, L., & Mandy, W. P. L. (2017). What is the male-to-female ratio in autism spectrum disorder? A systematic review and meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry*, 56(6), 466–474.
- Lord, C., & Jones, R. M. (2012). Annual research review: Re-thinking the classification of autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 53(5), 490–509.
- Macari, S. L., Wu, G. C., Powell, K. K., Fontenelle, S., Macris, D. M., & Chawarska, K. (2018). Do parents and clinicians agree on ratings of autism-related behaviors at 12 months of age? A study of infants at high and low risk for ASD. *Journal of Autism and Developmental Disorders*, 48(4), 1069–1080.
- Mademtzi, M., Singh, P., Shic, F., & Koenig, K. (2018). Challenges of females with autism: A parental perspective. *Journal of Autism and Developmental Disorders*, 48(4), 1301–1310.
- Matson, J. L., Beighley, J., & Turygin, N. (2012). Autism diagnosis and screening: Factors to consider in differential diagnosis. *Research in Autism Spectrum Disorders*, 6(1), 19–24.
- Mayes, S. D., & Lockridge, R. (2018). Brief report: How accurate is teacher report of autism symptoms compared to parent report? *Journal of Autism and Developmental Disorders*, 48(5), 1833–1840.
- Mazurek, M. O., Curran, A., Burnette, C., & Sohl, K. (2019). ECHO autism STAT: Accelerating early access to autism diagnosis. *Journal of Autism and Developmental Disorders*, 49(1), 127–137.
- McDonnell, C. G., Bradley, C. C., Kanne, S. M., Lajonchere, C., Warren, Z., & Carpenter, L. A. (2019). When are we sure? Predictors of clinician certainty in the diagnosis of autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49(4), 1391–1401.
- Nowell, K. P., Brewton, C. M., Allain, E., & Mire, S. S. (2015). The influence of demographic factors on the identification of autism spectrum disorder: A review

- and call for research. *Review Journal of Autism and Developmental Disorders*, 2(3), 300–309.
- Penner, M., King, G. A., Hartman, L., Anagnostou, E., Shouldice, M., & Hepburn, C. M. (2017). Community general pediatricians' perspectives on providing autism diagnoses in Ontario, Canada: A qualitative study. *Journal of Developmental and Behavioral Pediatrics*, 38(8), 593.
- Reszka, S. S., Boyd, B. A., McBee, M., Hume, K. A., & Odom, S. L. (2014). Brief report: Concurrent validity of autism symptom severity measures. *Journal of Autism and Developmental Disorders*, 44(2), 466–470.
- Shattuck, P. T., Durkin, M., Maenner, M., Newschaffer, C., Mandell, D. S., Wiggins, L., . . . Baio, J. (2009). Timing of identification among children with an autism spectrum disorder: Findings from a population-based surveillance study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 48(5), 474–483.
- Stratis, E. A., & Lecavalier, L. (2015). Informant agreement for youth with autism spectrum disorder or intellectual disability: A meta-analysis. *Journal of Autism and Developmental Disorders*, 45(4), 1026–1041.

Predictors of Emotion Regulation in Children with Autism Spectrum Disorder (ASD)

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Emotion regulation (ER) involves the ability to understand, monitor, and modulate emotion states in the service of goal-directed activity (Thompson 1994). A number of studies have identified significant challenges with ER in children with ASD relative to children without ASD, including a tendency to use less adaptive and more maladaptive regulatory strategies (Jahromi et al. 2012; Mazefsky et al. 2014; Nuske et al. 2017; Samson et al. 2012, 2015a, b). ER strategies also appear to be less effective in reducing observed negativity for children with ASD than for children with neurotypical development (Jahromi et al. 2012), suggesting problems with strategy selection, utilization, and implementation.

The growing evidence linking ER difficulties with comorbid emotional and behavioral problems in children with ASD (Mazefsky and White 2014; White et al. 2014) has underscored the importance of understanding factors that may predict increased risk for regulatory challenges in this population.

Multiple factors have been conceptualized as underlying problems with ER in children with ASD, though relatively few empirical investigations exist. Core symptoms of ASD as well as associated social-cognitive and executive functioning deficits may contribute to regulatory problems (Jahromi et al. 2013; Mazefsky and White 2014). Mechanisms of effect may be direct (e.g., difficulties with emotion understanding, weak central coherence, or cognitive and behavioral rigidity may challenge regulatory efforts) or indirect (e.g., social communication deficits may impede the delivery of co-regulatory supports from parents, teachers, and others in the child's environment). Interestingly, ASD symptoms have been linked more consistently to global ER quality (Berkovits et al. 2017; Fenning et al. 2018) than to ER strategies (Konstantareas and Stewart 2006; Patel et al. 2017). A recent study revealed ASD symptom severity to be the most significant correlate of observed global dysregulation in children with ASD, predicting above and beyond other child factors, including age, IQ, and sympathetic nervous system activation, and parental co-regulatory behavior (Fenning et al. 2018).

In addition to ASD-related symptoms, child intellectual functioning has been examined in relation to ER. Although most studies have not found significant links between IQ and ER in children with ASD (Berkovits et al. 2017; Samson et al. 2014, 2015a, b; Ting and Weiss 2017), some investigations employing observational ER measures have revealed associations with cognitive functioning (Fenning et al. 2018; Konstantareas and Stewart 2006). However, ER research has predominantly excluded children with ASD who exhibit co-occurring cognitive delays. Recent calls to action have advocated strongly for expanding inclusion criteria to ensure appropriate representation and understanding of the broader population of children with ASD (Fenning et al. 2018; Baker et al. 2019).

Maturation of the autonomic nervous system is believed to contribute directly to ER (Beauchaine et al. 2007). Respiratory sinus arrhythmia (RSA), an index of the parasympathetic nervous system, has emerged as a robust biomarker of ER in children without ASD (Beauchaine 2015). Several studies suggest the potential for altered RSA in children with ASD (see Benevides and Lane 2015 for a review). In addition, individual differences in RSA have been linked with variation in emotion recognition and social skills in this population (Bal et al. 2010; Patriquin et al. 2013; Van Hecke et al. 2009), but limited research has focused on ER specifically (Guy et al. 2014). Preliminary efforts to map certain sympathetic indices such as electrodermal activity to ER have not found in-the-moment associations (Fenning et al. 2018). Further research is needed, including consideration of sympathetic-parasympathetic interactions that may offer insight into complex, multi-system processes (Beauchaine et al. 2007; Buss et al. 2018; Fenning et al. 2019). Other biological factors, including neural circuitry and genetics, undoubtedly play a role as well (Mazefsky and White 2014).

Parenting has long been recognized as a powerful exogenous influence on child ER (Cole et al. 1994; Kopp 1982, 1989; Morris et al. 2007; Thompson and Meyer 2007), but surprisingly little research has examined parental influence on ER in children with ASD. The mere physical presence of parents may promote ER in young children with ASD (Jahromi et al. 2012), and greater observed parent-child reciprocity has been associated with reduced negative emotionality in this population (Hirschler-Guttenberg et al. 2015). Explicit parental co-regulatory efforts have also been linked to the quality of child ER but only when measured concurrently during dyadic parent-child tasks (Fenning et al. 2018; Gulrud et al. 2010; Ting and Weiss 2017). Further research utilizing carefully controlled longitudinal designs is needed to understand the potential influence of parenting on child ER.

Challenges with ER and associated emotional and behavioral problems are well documented in children with ASD. Emerging evidence suggests that these ER difficulties may be related to core

aspects of the disorder (Fenning et al. 2018; Mazefsky and White 2014). However, much remains to be learned about factors underlying risk for poor ER in this population. Understanding the developmental course of ER in children with ASD as well as factors predictive of individual differences in ER trajectories is critical to informing conceptualizations of ASD and to identifying and refining interventions tailored to address ER and related comorbid problems.

See Also

- [Internalization of Emotion Co-regulatory Support in Children with ASD](#)

References and Reading

- Baker, J. K., Fenning, R. M., & Moffitt, J. (2019). Brief report: A cross-sectional examination of the internalization of emotion co-regulatory support in children with ASD. *Journal of Autism and Developmental Disorders*. Advance online publication. <https://doi.org/10.1007/s10803-019-04091-0>.
- Bal, E., Harden, E., Lamb, D., Vaughan Van Hecke, A., Denver, J., & Porges, S. (2010). Emotion recognition in children with autism spectrum disorders: Relations to eye gaze and autonomic state. *Journal of Autism and Developmental Disorders*, 40, 358–370. <https://doi.org/10.1007/s10803-009-0884-3>.
- Beauchaine, T. P. (2015). Respiratory sinus arrhythmia: A transdiagnostic biomarker of emotion dysregulation and psychopathology. *Current Opinion in Psychology*, 3, 43–73. <https://doi.org/10.1016/j.copsyc.2015.01.017>.
- Beauchaine, T. P., Gatzke-Kopp, L., & Mead, H. K. (2007). Polyvagal theory and developmental psychopathology: Emotion dysregulation and conduct problems from preschool to adolescence. *Biological Psychology*, 74, 174–184. <https://doi.org/10.1016/j.biopsycho.2005.08.008>.
- Benevides, T., & Lane, S. (2015). A review of cardiac autonomic measures: Considerations for examination of physiological response in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45, 560–575. <https://doi.org/10.1007/s10803-013-1971-z>.
- Berkovits, L., Eisenhower, A., & Blacher, J. (2017). Emotion regulation in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 47(1), 68–79. <https://doi.org/10.1007/s10803-016-2922-2>.

- Buss, K. A., Jaffee, S., Wadsworth, M. E., & Kliever, W. (2018). Impact of psychophysiological stress-response systems on psychological development: Moving beyond the single biomarker approach. *Developmental Psychology*, 54, 1601–1605. <https://doi.org/10.1037/dev0000596>.
- Cole, P. M., Michel, M. K., & Teti, L. O. D. (1994). The development of emotion regulation and dysregulation: A clinical perspective. *Monographs of the Society for Research in Child Development*, 59, 73–102. <https://doi.org/10.2307/1166139>.
- Fenning, R. M., Baker, J. K., & Moffitt, J. (2018). Intrinsic and extrinsic predictors of emotion regulation in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48, 3858–3870. <https://doi.org/10.1007/s10803-018-3647-1>.
- Fenning, R. M., Erath, S. A., Baker, J. K., Messinger, D. S., Moffitt, J., Baucom, B. R., & Kaeppler, A. K. (2019). Sympathetic-parasympathetic interaction and externalizing problems in children with autism spectrum disorder. *Autism Research*. Advance online publication. <https://doi.org/10.1002/aur.2187>.
- Gulsrud, A. C., Jahromi, L. B., & Kasari, C. (2010). The co-regulation of emotions between mothers and their children with autism. *Journal of Autism and Developmental Disorders*, 40, 227–237. <https://doi.org/10.1007/s10803-009-0861-x>.
- Guy, L., Souders, M., Bradstreet, L., DeLussey, C., & Herrington, J. D. (2014). Brief report: Emotion regulation and respiratory sinus arrhythmia in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44, 2614–2620. <https://doi.org/10.1007/s10803-014-2124-8>.
- Hirschler-Guttenberg, Y., Golan, O., Ostfeld-Etzion, S., & Feldman, R. (2015). Mothering, fathering, and the regulation of negative and positive emotions in high-functioning preschoolers with autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 56, 530–539. <https://doi.org/10.1111/jcpp.12311>.
- Jahromi, L. B., Meek, S. E., & Ober-Reynolds, S. (2012). Emotion regulation in the context of frustration in children with high functioning autism and their typical peers. *Journal of Child Psychology and Psychiatry*, 53(12), 1250–1258. <https://doi.org/10.1111/j.1469-7610.2012.02560.x>.
- Jahromi, L. B., Bryce, C. I., & Swanson, J. (2013). The importance of self-regulation for the school and peer engagement of children with high-functioning autism. *Research in Autism Spectrum Disorders*, 7(2), 235–246. <https://doi.org/10.1016/j.rasd.2012.08.012>.
- Konstantareas, M. M., & Stewart, K. (2006). Affect regulation and temperament in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 36(2), 143–154. <https://doi.org/10.1007/s10803-005-0051-4>.
- Kopp, C. B. (1982). Antecedents of self-regulation: A developmental perspective. *Developmental Psychology*, 18, 199. <https://doi.org/10.1037/0012-1649.18.2.199>.
- Kopp, C. B. (1989). Regulation of distress and negative emotions: A developmental view. *Developmental Psychology*, 25, 343–354.
- Mazefsky, C. A., & White, S. W. (2014). Emotion regulation: Concepts & practice in autism spectrum disorder. *Child and Adolescent Psychiatric Clinics of North America*, 23(1), 15. <https://doi.org/10.1016/j.chc.2013.07.002>.
- Mazefsky, C. A., Borue, X., Day, T. N., & Minshew, N. J. (2014). Emotion regulation patterns in adolescents with high-functioning autism spectrum disorder: Comparison to typically developing adolescents and association with psychiatric symptoms. *Autism Research*, 7(3), 344–354. <https://doi.org/10.1002/aur.1366>.
- Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Social Development*, 16, 361–388. <https://doi.org/10.1111/j.1467-9507.2007.00389.x>.
- Nuske, H. J., Hedley, D., Woollacott, A., Thomson, P., Macari, S., & Dissanayake, C. (2017). Developmental delays in emotion regulation strategies in preschoolers with autism. *Autism Research*, 10, 1808–1822. <https://doi.org/10.1002/aur.1827>.
- Patel, S., Day, T. N., Jones, N., & Mazefsky, C. A. (2017). Association between anger rumination and autism symptom severity, depression symptoms, aggression, and general dysregulation in adolescents with autism spectrum disorder. *Autism*, 21(2), 181–189. <https://doi.org/10.1177/1362361316633566>.
- Patriquin, M. A., Scarpa, A., Friedman, B. H., & Porges, S. W. (2013). Respiratory sinus arrhythmia: A marker for positive social functioning and receptive language skills in children with autism spectrum disorders. *Developmental Psychobiology*, 55, 101–112. <https://doi.org/10.1002/dev.21002>.
- Samson, A. C., Huber, O., & Gross, J. J. (2012). Emotion regulation in Asperger's syndrome and high-functioning autism. *Emotion*, 12(4), 659. <https://doi.org/10.1037/a0027975>.
- Samson, A. C., Phillips, J. M., Parker, K. J., Shah, S., Gross, J. J., & Hardan, A. Y. (2014). Emotion dysregulation and the core features of autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44, 1766–1772. <https://doi.org/10.1007/s10803-013-2022-5>.
- Samson, A. C., Hardan, A. Y., Podell, R. W., Phillips, J. M., & Gross, J. J. (2015a). Emotion regulation in children and adolescents with autism spectrum disorder. *Autism Research*, 8(1), 9–18. <https://doi.org/10.1002/aur.1387>.
- Samson, A. C., Wells, W. M., Phillips, J. M., Hardan, A. Y., & Gross, J. J. (2015b). Emotion regulation in autism spectrum disorder: Evidence from parent interviews and children's daily diaries. *Journal of Child Psychology and Psychiatry*, 56(8), 903–913. <https://doi.org/10.1111/jcpp.12370>.
- Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development*, 59, 25–52. <https://doi.org/10.1111/j.1540-5834.1994.tb01276.x>.

- Thompson, R. A., & Meyer, S. (2007). Socialization of emotion regulation in the family. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 249–268). New York: Guilford Press.
- Ting, V., & Weiss, J. A. (2017). Emotion regulation and parent co-regulation in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(3), 680–689. <https://doi.org/10.1007/s10803-016-3009-9>.
- Van Hecke, A. V., Lamb, D., Lebow, J., Bal, E., Harden, E., Kramer, A., Denver, J., ... Porges, S. W. (2009). Electroencephalogram and heart rate regulation to familiar and unfamiliar people in children with autism spectrum disorders. *Child Development*, 80, 1118–1133. <http://www.jstor.org/stable/25592056>
- White, S. W., Mazefsky, C. A., Dichter, G. S., Chiu, P. H., Richey, J. A., & Ollendick, T. H. (2014). Social-cognitive, physiological, and neural mechanisms underlying emotion regulation impairments: Understanding anxiety in autism spectrum disorder. *International Journal of Developmental Neuroscience*, 39, 22–36. <https://doi.org/10.1016/j.ijdevneu.2014.05.012>.

Preemployment Skills for People with ASD

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Synonyms

Employment readiness skills; Job readiness; Life skills; Prevocational skills; Soft job skills

Definition

Preemployment skills are a set of skills that establish the foundation of employment skills. Conceptually, they are the building blocks of more advanced skills and are essential for an individual on the autism spectrum to master before he or she can be successful in the world of work. These skills are in direct contrast to employment skills which often are job specific. These are often referred to as soft job skills.

Generically, preemployment skills include behaviors such as bathing, grooming, and dressing appropriately for the particular work environment. Related to being able to present oneself professionally in a work environment with clean clothing is the skill of being able to do laundry including washing, folding, ironing, and hanging of clothing. Time management is another example of a generic skill individuals need to master in order to be successful in the work place. Time management for individuals on the spectrum is a cluster of behaviors such as setting an alarm clock or smartphone, waking up on time, arriving on time, and on-time task completion. Being able to navigate and use public transportation is another cluster of preemployment skills individuals on the spectrum should master prior to employment. The majority of individuals on the spectrum will probably not own their own car and learn how to drive. Consequently, learning how to get to and from home and work is paramount. In addition to learning how to get to and from work, the travel training should include how to get to the grocery store, medical provider offices, and retail outlets. The travel training should not be simply fixed route training, but rather, it should center on contingency management, i.e., what to do when things go wrong when using public transportation. Self-advocacy is another pre-employment soft skill that is important for individuals with ASDs to learn especially when dealing with supervisors and co-workers. Financial literacy especially money management, taxes, and budgeting also fall under the heading of preemployment skills.

With the passage of the Workforce Innovation and Opportunity Act (WIOA) in 2014, the federal government now requires state offices of vocational rehabilitation services to provide pre-employment transition services. Previously, state agencies refused to pay for such critical services and training. According to WINTACH (2017) preemployment transition services should include:

- Job exploration counseling
- Work-based learning experiences, which may include in-school or after school opportunities,

experiences outside of the traditional school setting, and/or internships

- Counseling on opportunities for enrollment in comprehensive transition or postsecondary educational programs
- Workplace readiness training to develop social skills and independent living
- Instruction in self-advocacy

Under WIOA 15% of vocational rehabilitation services budgets must be spent on transition services for youth with disabilities, thereby allowing youth with disabilities the opportunity to acquire preemployment skills. As per WINTACH, “Funds reserved for pre-employment transition services may be used for the required, authorized, and pre-employment transition coordination activities.”

Preemployment transition coordination consists of:

- Attending individualized education program meetings for students with disabilities, when invited
- Working with the local workforce development boards, one-stop centers, and employers to develop work opportunities for students with disabilities, including internships, summer employment and other employment opportunities available throughout the school year, and apprenticeships
- Working with schools, including those carrying out activities under section 614(d) of the IDEA, to coordinate and ensure the provision of preemployment transition services
- When invited, attending person-centered planning meetings for individuals receiving services under title XIX of the Social Security Act (42 U.S.C. 1396 et seq.) (WINTACH 2017)

See Also

- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Life Skills](#)
- [Workforce Innovations Opportunity Act](#)

References and Reading

- 20 U.S.C. § 1400 *et. seq.* Individuals with Disabilities Education Act (IDEA) of 1997.
- Federal Register Vol. 81, No 161, Part V, Pages 55791–56470. Department of Labor (20 CFR Parts 676, 677, and 678). Department of Education (34 CFR Part 361 and 463). 19 Aug 2016.
- The Workforce Innovations Opportunity Act of 2014 (WIOA). H.R. 803, 113th Cong. (2014).
- VanBergeijk, E. O. (2017, June). What are pre-employment skills and how does my child get them? *Parenting Special Needs*. <https://www.parentingspecialneeds.org/article/pre-employment-skills-child-get/>
- Workforce Innovation Technical Assistance Center (WINTACH). (2017). Pre-employment transition services. <http://www.wintac.org/topic-areas/pre-employment-transition-services>

Preference and Reinforcer Assessment

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Synonyms

[Forced-choice preference assessment](#); [Free-operant preference assessment](#); [Multiple stimulus with replacement \(MSW\) preference assessment](#); [Multiple stimulus without replacement \(MWSO\) preference assessment](#); [Paired-choice preference assessment](#); [Stimulus preference assessment](#)

Definition

Preference assessments yield information about stimuli that may function as motivators in behavioral programming (Pace et al. 1985). Assessments may include stimuli such as objects, activities, food, or social interactions and can be carried out through indirect and direct methods (Tullis et al. 2011). Examples of indirect methods include interviews, checklists, and rating scales (Green et al.

1988; Windsor et al. 1994) that could be completed by a caretaker or the individual. Direct methods involve structured activities that measure choice or engagement. Choice-based assessments measure preference by having the individual select between two or more stimuli followed by access to the selected stimulus. Engagement-based preference assessments, on the other hand, measure the duration that an individual may spend interacting with a given stimulus. Preference assessments provide a pool of potentially preferred stimuli for the individual, a comparative value or hierarchy of most-to-least preferred stimuli and, for more structured forms of preference assessment (e.g., forced-choice preference assessment), a relative value among the evaluated stimuli (Roscoe et al. 1999).

Reinforcer assessments expand beyond the concept of preference to assess whether a preferred stimulus possesses sufficient value for the individual to increase a specific behavior to contact the stimulus. In other words, reinforcer assessments determine if the individual will work to access a stimulus, thereby identifying the stimulus as a reinforcer rather than a preference. Oftentimes, stimuli assessed in the reinforcer assessment originate from preference assessments. The contrast between these two assessments is an important one for individuals with autism spectrum disorders: stimuli identified as preferred do not always function as reinforcers (DeLeon and Iwata 1996; Pace et al. 1985). That is, although an individual may demonstrate a preference for a given stimulus, it may not carry sufficient value to motivate an individual to complete demands to earn it.

An example of a preference and reinforcer assessment could be as follows: caretakers seek to identify potential reinforcers for a child's reading program. A preference assessment was conducted, which included a rating scale completed by caregivers, followed by direct assessment of the child's choices. Eight items were assessed: dolls, Play-Doh[®], a cartoon video, a coloring book, building blocks, a farm house toy, a hula hoop, and a Slinky[®]. In their rating scales, caregivers identified the Slinky[®], Hula

Hoop[®], and dolls as the most preferred items for their child, in that order. A paired-choice preference assessment conducted with the child yielded the greatest preference value for the coloring book, followed by the dolls and Hula Hoop[®]. Next, a reinforcer assessment was carried out with the child, where he was given 30-second access to each of the top four preferred stimuli after completing a reading task. The reinforcer assessment found that the child was most likely to read to access the Hula Hoop[®] and coloring book, but not the other two items. Based on these evaluations, these reinforcers were incorporated into the child's reading program.

See Also

- [Behavior Plan](#)
- [Positive Reinforcement](#)

References and Reading

- DeLeon, I. G., & Iwata, B. A. (1996). Evaluation of a multiple-stimulus presentation format for assessing reinforcer preferences. *Journal of Applied Behavior Analysis*, 29(4), 519–533.
- Green, C. W., Reid, D. H., White, L. K., Halford, R. C., Brittain, D. P., & Gardner, S. M. (1988). Identifying reinforcers for persons with profound handicaps: Staff opinion versus systematic assessment of preferences. *Journal of Applied Behavior Analysis*, 21(1), 31–43.
- Pace, G. M., Ivancic, M. T., Edwards, G. L., Iwata, B. A., & Page, T. J. (1985). Assessment of stimulus preference and reinforcer value with profoundly retarded individuals. *Journal of Applied Behavior Analysis*, 18(3), 249–255.
- Roscoe, E. M., Iwata, B. A., & Kahng, S. (1999). Relative versus absolute reinforcement effects: Implications for preference assessments. *Journal of Applied Behavior Analysis*, 32(4), 479–493.
- Tullis, C. A., Cannella-Malone, H. I., Basbigill, A. R., Yeager, A., Fleming, C. V., Payne, D., & Wu, P. F. (2011). Review of the choice and preference assessment literature for individuals with severe to profound disabilities. *Education and Training in Autism and Developmental Disabilities*, 46, 576–595.
- Windsor, J., Piché, L. M., & Locke, P. A. (1994). Preference testing: A comparison of two presentation methods. *Research in Developmental Disabilities*, 15(6), 439–455.

Prefrontal Cortex

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Definition

The term “prefrontal cortex” refers to the part of the brain’s frontal lobe that lies in front of the primary motor cortex and the areas immediately anterior to motor regions, called premotor cortex. The functions of the prefrontal cortex are vast but include the higher cognitive processes such as planning, decision-making, working memory, cognitive control, and response inhibition. These sophisticated brain functions are often called the “executive functions” reflecting the high level decision-making processes performed here. In humans, the prefrontal cortex is more highly developed in all other animals and constitutes the major brain differences between human and nonhuman primates. Damage to the prefrontal cortex will cause many cognitive, behavioral, and personality changes including impulsivity, difficulty in controlling and regulating emotion, problems making good decisions and integrating information, difficulty in organizing information, planning activities, keeping information in one’s mind, generating new thoughts and ideas, flexibility in thinking and behaving, difficulty retrieving information from memory, and problems in time awareness and future directedness. Thus, the prefrontal cortex is often thought of as the part of the brain that makes us most human.

See Also

- [Executive Function \(EF\)](#)
- [Frontal Lobe Findings in Autism](#)

References and Reading

- Fuster, J. M. (1980). *The prefrontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe*. New York: Raven Press.
Fuster, J. M. (2008). *The prefrontal cortex* (4th ed.). London: Academic.

Prelinguistic Autism Diagnostic Observation Schedule

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Synonyms

[PL-ADOS](#)

Description

The PL-ADOS (DiLavore et al. [1995](#)) is a semi-structured evaluation of play, social interaction, and communication skills in young children suspected of having an autism spectrum disorder. It was designed to generate a social context in which behaviors relevant for a diagnosis of ASD may be observed in a clinical setting. The PL-ADOS is intended for children under the chronological age of 6 years and under the developmental age of 3 years; it is aimed specifically toward children who are not yet using phrase speech. It includes 12 activities with 17 accompanying activity ratings along with 31 overall codes. There is an algorithm that includes a subset of codes and generates a binary classification (autism, non-spectrum) based on DSM-IV and ICD-10 criteria. Administration takes approximately 30 min and can be completed in a professional’s office or playroom, although a caregiver is present. Codes are completed immediately after administration and are based on all behaviors during the administration. Scores on

each code generally range from 0 to 2, with higher scores indicating greater abnormality.

Historical Background

The PL-ADOS was developed at a time when early diagnosis of ASD was an emerging field of study. Existing instruments, which were largely intended for older individuals, offered insufficient sensitivity for young nonverbal populations. The ADOS had proved to be a useful instrument to aid in the diagnostic process, and the PL-ADOS modified the ADOS for children with limited language skills. The tasks and administration were adapted to reduce the need for language skills and to make them more suitable for developmentally and chronologically younger children. Moreover, additional activities were created that addressed known areas of difficulty for young children with autism.

Psychometric Data

Instrument development involved both validity and reliability studies. The validity study included 21 children with autism and 42 children with non-autism developmental disorders (21 in a 3-year-old group and another 21 in a 2-year-old group). Nine of 17 activity ratings showed a group effect, successfully discriminating the autism sample from both groups with developmental disorders. The remaining items distinguished the group with autism from only one of the comparison samples or did not differ across group at all. Similar analyses on 31 overall summary codes revealed that the autism sample differed from both comparison samples on all items but five (*overall level of language, tantrums and aggression, functional play, imagination/creativity, showing*). However, the autism sample differed from the 3-year-old comparison sample on the latter three items. As expected, the group of children with autism had a higher average score on all items than the comparison samples.

An algorithm was generated by selecting items that met theoretical and empirical thresholds for optimal group classification. Algorithm items were grouped into two categories: social

interaction/communication and restricted, repetitive behaviors. Algorithm items in each category were summed, yielding two separate algorithm scores. Cutoff scores were then selected based on maximal sensitivity and specificity, with a final cutoff of 12 in social interaction/communication and a cutoff of 2 in restricted, repetitive behaviors.

The reliability study included a subsample of 20 children, 12 with autism, 4 with non-autism developmental disorders, and 4 typically developing children. Interrater reliability was evaluated using weighted kappas; weighted kappas of .60 or greater were considered to indicate acceptable reliability (Cicchetti and Sparrow 1981). A subsample of 20 children was included in these analyses; item ratings (associated with particular activities) on the PL-ADOS yielded weighted kappas between .63 and .95, while weighted kappas for the 31 overall codes ranged from .60 to 1.00.

Clinical Uses

The PL-ADOS was developed as a research measure and was not sold as a widely available clinical measure. It was eventually subsumed by ADOS (including the Module 1 and Toddler Module), which is currently offered for clinical and research use.

See Also

- [Autism Diagnostic Observation Schedule](#)
- [Autism Diagnostic Observation Schedule \(ADOS\): Toddler Module](#)

References and Reading

- Cicchetti, D. V., & Sparrow, S. A. (1981). Developing criteria for establishing interrater reliability of specific items: Applications to assessment of adoptive behavior. *American Journal of Mental Deficiency, 86*(2), 127–137.
- DiLavore, P., Lord, C., & Rutter, M. (1995). The prelinguistic autism diagnostic observation schedule. *Journal of Autism and Developmental Disorders, 25*(4), 355–379.

Prelinguistic Communication

► Preverbal Communication

Prelinguistic Communication Assessment

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Definition

Both theory and research focused on the early development of spoken language suggest an important causal relationship between prelinguistic or preverbal communication skills and the later emergence of the ability to communicate through spoken language. Thus, assessing the types of preverbal communication acts that a child with autism may use and the situations in which the child is most likely or motivated to communicate has become an important component of comprehensive assessments for young children with autism. Prelinguistic communication refers to a group of behaviors that children use, prior to the emergence of first words, to direct the attention and actions of other people. This category of behaviors is sometimes referred to as “joint attention” or “social communication” with behaviors in this category emerging between 9 and 14 months in typically developing children. Specifically, prelinguistic communication behaviors consist of a combination of gestures (shows, gives, reaches, points), eye gaze, and vocalizations which are used purposefully by children to send a message to a communicative partner. Thus, these behaviors are often referred to as preverbal “intentional communication.” Prelinguistic communication behaviors are used to convey two primary pragmatic functions: to comment on actions and events in the environment and to request an instrumental response from the

communicative partner. These two prelinguistic functions often are termed “initiating joint attention” and “initiating behavioral regulation.” The latter behavior also is referred to as “requesting.” One additional behavior that often is included within the umbrella category of joint attention skills as well as included in assessments measuring prelinguistic communication is termed “attention-following” or “responding to joint attention (RJA).” This behavior refers to the child’s ability to follow into the attentional focus of a communicative partner by responding to cues such as the adult’s point, head turns, and direction of eye gaze. Thus, there are some prelinguistic communication skills which the young child uses to initiate interaction with a social partner and some skills which are used to respond to the partner’s overtures to the child. For children with autism, research has indicated that children’s spontaneous use of prelinguistic communication behaviors are positive predictors of later language outcomes and may present an area of special challenge; that is, children with autism may initiate preverbal communication acts and respond to the adult’s attentional directives at lower rates than their typically developing peers. In addition, children with autism are likely to use a higher proportion of communicative initiations that serve as requests than they are to use initiations which function to comment or share attention with a social partner.

Historical Background

Many research studies conducted since the 1980s have examined and characterized the prelinguistic communication of preschool-aged children with autism. This research has indicated that children with autism most frequently communicate for purposes of behavior regulation and less frequently communicate to share attention and/or affect with another person. Children with autism who have mental ages under 20 months also show deficits in responding to the attentional directives of other people. Response to joint attention at the pretreatment period also has been shown to be a moderator of the relation between the amount of

intervention received by young children with autism and language gains 1 year later for a group of preschool-aged children with autism. Indeed, a deficit in joint attention is considered by many to be a core feature of ASD. Because the ability to initiate and respond to joint attention precedes the emergence of single-word spoken language in typical development, a deficit in joint attention may be considered a core impairment for young children with autism. In addition to characterizing deficits in social communication for young children with autism, other studies have detected robust associations between early prelinguistic communication behaviors and later language for these children. Theoretically, deficits in prelinguistic communication skills may impact language learning in several ways. One theory posits that prelinguistic initiations are foundational because they represent the child's motivation to engage in the kinds of shared interactions with social partners that support the development of socially transmitted skills. Once children develop expertise in initiating to social partners prelinguistically, they need only map spoken words onto the communicative functions they already possess. Another theory suggests that children who direct prelinguistic initiations will elicit language facilitating verbal input from responsive social partners, thus supporting language growth. Finally, theory suggests that children who are able to respond to the attentional directives of others will be more successful in learning language incidentally from the interactions of those around them and less likely to require explicit instruction to learn to speak. All of these theoretical approaches provide a rationale for understanding the emergence of prelinguistic communication skills in young children with autism and targeting these skills in interventions aimed at increasing early language development.

Current Knowledge

The background of research focusing on prelinguistic communication behaviors highlights the importance of assessing prelinguistic communication skills whether for diagnostic or

intervention purposes. To this end, several instruments have been constructed to provide this type of information. The most common method of assessing prelinguistic communication skills is by collecting a sample of these behaviors during an interaction with an examiner. The Early Social Communication Scales (ESCS) and the Communication and Symbolic Behavior Scales (CSBS) are two such instruments including activities that press for initiating joint attention, behavior regulation, and responding joint attention. Module 1 of the Autism Diagnostic Observation Schedule (ADOS) also includes activities designed to elicit prelinguistic communication skills (such as initiating joint attention, reaching, giving, and attention-following). Module 2 of the ADOS includes activities designed to sample attention-following as well as conventional, instrumental, and informational gestures that are used communicatively. In addition to tests which sample prelinguistic skills in an interactive format, parent report also is used to measure these behaviors; the words and gestures subscale of the MacArthur-Bates Communicative Development Inventory includes a section that queries the child's use of communicative gestures. Prelinguistic communication skills also have been included in measures developed for early autism screening. The Modified Checklist for Autism in Toddlers (MCHAT), for example, has an item querying the parent as to whether the child ever points or shows items to them or if the child can respond to the parent's point. The Screening Test for Autism in Two-Year-Olds (STAT) includes items that assess the child's ability to request using eye gaze and vocalization, and to direct attention.

Future Directions

Careful and accurate assessment of the prelinguistic communication skills of young children with autism will contribute to a more nuanced characterization of trajectories of development for these children. In addition, future studies should continue to examine whether interventions targeting early prelinguistic communication skills can improve spoken language outcomes for

children with autism relative to interventions that exclusively target spoken language goals. Finally, assessment of early prelinguistic communication skills may be informative to studies examining aptitude by treatment interactions. It may be that children with different profiles of prelinguistic communication skills would differentially benefit from different intervention approaches.

See Also

- [Joint Attention](#)
- [Preverbal Communication](#)
- [RJA/IJA \(Initiating/Responding to Joint Attention\)](#)

References and Reading

- Bakeman, R., & Adamson, L. (1984). Coordinating attention to people and objects in mother-infant and peer-infant interactions. *Child Development*, 55, 1278–1289.
- Baron-Cohen, S., Cox, A., Baird, G., Sweettenham, J., & Nighingale, N. (1996). Psychological markers in the detection of autism in infancy in a large population. *British Journal of Psychiatry*, 168, 158–163.
- Bono, M. A., Daley, T., & Sigman, M. (2004). Relations among joint attention, amount of intervention and language gain in autism. *Journal of Autism and Developmental Disorders*, 34, 495–505.
- Crais, E. R., Watson, L. R., & Baranek, G. T. (2009). Use of gesture development in profiling children's prelinguistic communication skills. *American Journal of Speech-Language Pathology*, 18, 95–108.
- Kleinman, J. M., Robins, D. L., Ventola, P. E., Pandey, J., Boorstein, H. C., Esser, E. L., et al. (2008). The modified checklist for autism in toddlers: A follow-up study investigating the early detection of autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38, 827–839.
- Mundy, P., Sigman, M., & Kasari, C. (1990). A longitudinal study of joint attention and language development in autistic children. *Journal of Autism and Developmental Disorders*, 20, 115–128.
- Paul, R., & Wilson, K. P. (2009). Assessing speech, language, and communication in autism spectrum disorders. In S. Goldstein, J. A. Naglieri, S. Ozonoff, S. Goldstein, J. A. Naglieri, & S. Ozonoff (Eds.), *Assessment of autism spectrum disorders* (pp. 171–208). New York: Guilford Press.
- Seibert, J. M., Hogan, A. E., & Mundy, P. C. (1982). Assessing interactional competencies: The early social-communication scales. *Infant Mental Health Journal*, 3, 244–258.
- Stone, W. L., Coonrod, E. E., Turner, L. M., & Pozdol, S. L. (2004). Psychometric properties of the STAT for early autism screening. *Journal of Autism and Developmental Disorders*, 34, 691–701.
- Toth, K., Munson, J., Meltzoff, A. N., & Dawson, G. (2006). Early predictors of communication development in young children with autism spectrum disorder: Joint attention, imitation, and toy play. *Journal of Autism and Developmental Disorders*, 36, 993–1005.
- Wetherby, A. (2006). Understanding and measuring social communication in children with autism spectrum disorders. In T. Charman & W. Stone (Eds.), *Social and communication development in autism spectrum disorders: Early identification, diagnosis, and intervention* (pp. 3–35). New York: Guilford Press.
- Wetherby, A. M., & Prizant, B. M. (2002). *Communication and symbolic behavior scales: Developmental profile* (1st ed.). Baltimore: Paul H Brookes.

Prelinguistic Sounds

- [Vocalization](#)

Preliteracy

- [Emergent Literacy](#)

Premack Principle

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Synonyms

[Grandma's rule](#)

Definition

The Premack principle is a principle of reinforcement which states that an opportunity to engage in more probable behaviors (or activities) will reinforce less probable behaviors (or activities). For

example, if a child enjoys playing computer games (more probable) and avoids completing math problems (less probable), we might allow her to play the computer after (contingent upon) completing 15 math problems. Prior to the introduction of the Premack principle, systems of reinforcement were viewed as the contingency between a stimulus and behavior. The Premack principle expanded the existing reinforcement contingency of stimulus behavior to include contingencies between two behaviors. This principle is often referred to as “grandma’s rule” because grandmothers (or any caregivers) often apply this principle: “you have to eat your vegetables (less probable) before you can have dessert (more probable)” or “you have to clean your room (less probable) before you can go outside and play (more probable).” In these example, the highly preferred (or probable) activity is used as reinforcement for the less preferred (or probable) activity. David Premack demonstrated this principle with Cebus monkeys and humans, including young children. For example, Premack and his colleagues demonstrated the power of the Premack principle by showing high-probability activities (i.e., playing pinball or eating candy) can be effective reinforcement for low-probability activities, but only if the child prefers the high-probability activity. In this study, highly preferred activities were effective as reinforcers for less preferred behaviors. This study also highlighted the concept that reinforcers are relative and defined in relation to the behaviors they reinforce. For example, eating candy functioned as reinforcement for playing pinball only for children who preferred eating candy to pinball. Conversely, playing pinball functioned as reinforcement for eating only for children who preferred pinball to candy.

See Also

- [Reinforcement](#)
- [Reinforcer](#)

References and Reading

Catania, A. C. (1998). *Learning*. Upper Saddle River: Prentice Hall.

Premack, D. (1959). Toward empirical behaviour laws: Positive reinforcement. *Psychological Review*, 66, 219–233.

Premack, D. (1963). Rate differential reinforcement in monkey manipulation. *Journal of the Experimental Analysis of Behavior*, 6, 81–89.

Prenatal Diagnosis of Autism

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Definition

The prenatal diagnosis of autism involves the potential identification of autism spectrum disorder (ASD) symptoms and/or characteristics during pregnancy and before birth (in utero).

Historical Background

Autism spectrum disorder (ASD) is a spectrum or continuum of complex, neuropsychiatric disorders incorporating autistic disorder, Asperger’s disorder, childhood disintegrative disorder, and pervasive developmental disorder (Med 2013). It was first clinically described by the psychiatrist Leo Kanner and pediatrician Has Asperger in the 1940s (Baron-Cohen 2015; Kanner 1943). Initially, geneticists were highly skeptical about a hereditary role in ASD etiology (Newschaffer et al. 2002). However, by the 1980s, there were strong empirical suggestions that ASD was indeed heritable (Newschaffer et al. 2002). ASD can unfortunately affect and hinder an individual’s social, cognitive, and communicative functioning and abilities (Med 2013). Aggression is often understood and noted as the most common problem among ASD individuals; in a 2011 study, Kanne and Mazurek reported that in a sample of 1,380 children and adolescents, 68% and 49% were reported by parents to have displayed aggression to a caregiver and non-caregiver, respectively (Kanne and Mazurek 2011). Thus, ASD has a large impact on the general population.

In a study examining ASD prevalence using 2016 data, Maenner et al. showed that the prevalence of ASD among 11 Autism and Developmental Disabilities Monitoring Networks (ADDMN) was 1 in 54 children aged 8 years old, with prevalence varying by the ADDMN site and state (Maenner 2020). ASD was also 4.3 times more prevalent among boys than girls but varied in prevalence across communities and socio-demographic groups (Maenner 2020). There is also evidence that this male predominance in ASD is partly due to the presence of protective factors reducing the risk of ASD in females (Jeste and Geschwind 2014). These varying aspects of ASD have further pushed for ASD research.

The prevalence of ASD has led to extensive research interests in the biological basis and genetic risk factors of ASD and in understanding the risk genes that facilitate ASD pathways and phenotypes (Jeste and Geschwind 2014). ASD has been shown to have a genetic heterogeneity and strong genetic component, in which rare genetic mutations play roles (Jeste and Geschwind 2014). Since the median age at earliest ASD diagnosis was 51 months or just over 4 years old for 2016, it would be interesting to consider if an earlier median diagnosis is plausible and beneficial (Maenner 2020). Shafali and Geschwind pointed out in a 2014 *Nature Reviews Neurology* review that genetic testing using chromosomal microarray analysis, a clinical diagnostic tool for detecting microdeletions or duplications related to neurodevelopmental and congenital disorders, is “warranted and clinically indicated for all suspected cases of ASD” (Batzir et al. 2015; Jeste and Geschwind 2014). This entry explores the hypothesis that there is a possibility for prenatally testing of ASD risk factors or ASD in utero.

Current Knowledge

Literature Review

ASD, Genetics, and Fetal Development

Researchers have been exploring the risk factors involved with and potential causes for ASD. During the later 1900s, researchers conducted many studies, including twin and family studies, which

suggested a large genetic component to ASD (Anderson et al. 2007). Studies have also pointed out an apparent polygenetic and heterogenetic nature to ASD and an unclear environmental influence on expression (Anderson et al. 2007). As Anderson et al. wrote in 2007, early disorder-associated phenotypes could help in identifying candidate genes, early screening or risk assessment for ASD (Anderson et al. 2007). Since this is a promising avenue for ASD research, many researchers have examined different genetically and biologically based causes for and molecular pathways implicated in ASD.

Researchers have noted several heritable and nonheritable risk factors for ASDs. Some hypotheses point to the following: postnatal changes in head size, brain volume anomalies, neuroanatomic defects (specifically in the amygdala), serotonergic system differences, immune system irregularities, and distinctive neuropeptides or neurotrophins in both ASD groups that could serve as detectable biomarkers (Newschaffer et al. 2002). There has also been suggestions that these anomalies are prenatal in origin, and with this knowledge and other studies, researchers believe that the “neuropathologic process underlying ASD begins in utero” (Newschaffer et al. 2002). In 2014, Stoner et al. found focal disruption of cortical laminar architecture in cortex regions of young children with autism, supporting a possible dysregulation of layer formation and neuronal differential during prenatal development (Stoner et al. 2014). Further work will help to verify these conclusions.

The Placenta and ASD

A study by Anderson et al. in 2007 found that placental trophoblast inclusions are associated with ASD. However, further research was needed to replicate the findings and determine if there was any utility in using these findings as a measure in the early detection of ASD (Anderson et al. 2007). In a more recent study also on the topic of placental gross shape, Park et al. compared placental shape features in a sample of younger siblings of ASD children (a high ASD risk group) and a sample of low-risk peers (Park et al. 2018). They found that in the siblings of ASD children group, there were moderately higher placental thickness

measurements and rounder, more regular in perimeter placentas (Park et al. 2018). These differences did persist for both males and females but were more pronounced in females (Park et al. 2018). The siblings of ASD children group also had reduced placental shape variability (Park et al. 2018). However, the researchers noted their study's limitations stating that future work was still needed to explore whether these placental morphological differences are actually associated with an ASD phenotype (Park et al. 2018).

T Cells and ASD

It has also been hypothesized that alterations in the mother's immune system, like a viral infection during pregnancy, during early periods of fetal neurodevelopment may play a role in ASD. In 2016, Choi et al. reported that the maternal interleukin-17a, the predominant T-helper 17 cell cytokine, pathway promotes autism-like phenotypes in murine offspring (Choi et al. 2016). Interleukin-17a administration to the fetal brain in mice promoted abnormal cortical development and ASD-like behavioral phenotypes (Choi et al. 2016). In pregnant mouse dams, Choi et al. showed that interleukin-17a-blocking antibody therapeutic treatment ameliorated maternal immune activation-associated behavioral abnormalities (Choi et al. 2016). The researchers proposed that therapeutic targeting of T-helper 17 cells in susceptible pregnant mothers could reduce the likelihood of having children with inflammation-induced ASD-like phenotypes (Choi et al. 2016).

Maternal Immune Response and ASD

Other studies have noted that although genetic factors are important for ASD, prenatal maternal immune system responses could also be associated with ASD pathogenesis. In 2009, Zimmerman et al. looked at maternal anti-brain antibodies in autism and identified specific serum antibodies that recognize prenatally expressed brain antigens in the mothers of autistic children (Zimmerman et al. 2007). They suggested that these autoantibodies, antibodies that are capable of binding to self-antigens and serving as disease markers, could transfer across the placenta to alter

fetal brain development (Zimmerman et al. 2007). They concluded that there were distinctive patterns of antibody reactivity to fetal brain in mothers and children, which could be relevant to autism pathogenesis (Zimmerman et al. 2007).

Future Directions

Detecting ASD In Utero

Although previous research efforts have shown that autism is detectable even in infancy, the question that still remains is whether or not prenatal ASD testing is plausible. In 2014, Deborah Brauser wrote an article on WedMD.com stating that in a small study looking at ultrasound scans from 40 children eventually diagnosed with ASD and 120 healthy children, children who eventually developed ASD had larger head and abdominal sizes at 20 weeks in utero than healthy children (Brauser 2014). The lead researcher Lois Salter of the University of Edinburgh had presented these preliminary, not peer-reviewed findings at the 2014 International Congress of the Royal College of Psychiatrists meeting (Brauser 2014). Salter claimed that "something about autism" was occurring at that 20-week mark, and that if further exploration is possible, this finding could "help with diagnosing [ASD] earlier and treating [ASD] earlier" (Brauser 2014). However, the study or its data could not be found online as of April 9, 2020.

There is currently no conclusive test that can predict the probability that pregnant mothers will have a child that will be diagnosed with ASD (Hollowood et al. 2018). However, Hollowood et al. showed in 2018 that among high-risk mothers, mothers who previously had a child with ASD and thus had an elevated risk of having another child with ASD, it was not possible to predict an autism pregnancy outcome (Hollowood et al. 2018). Based on the measurements, Hollowood et al. concluded that it was not possible to "determine during a pregnancy if a child will be diagnosed with ASD by age 3" despite the differences in folate-dependent transmethylation and transsulfuration metabolites being indicative of a mother's risk level for having a child with ASD (Hollowood et al. 2018).

There may be more promising avenues of research for a tool or methodology of detecting ASD in utero or at least at a time point earlier than the current standard for diagnosis. Researches in 2018 hypothesized that a saliva-based poly-“omic” RNA panel could distinguish children with ASD from children with neurotypical and non-ASD developmental delay (Hicks et al. 2018). Hicks et al. concluded that this salivary poly-omic RNA measurement method could be an accurate and non-invasive approach to improve referral specificity for ASD or provide objective support with an ASD diagnosis (Hicks et al. 2018).

Ethical Concerns

While researching this topic, it was clear that earlier diagnostics for ASD could be beneficial for both the mother and child, but the thought of prenatally testing for ASD also brought up ethical concerns. What would and should the pregnant mother do if she knew that she had a high risk of having a child who would develop ASD? Would it engage conversations on abortion or genetic editing using CRISPR-Cas9 for “designer babies” without a high risk for ASD? Would the family then tell their child at a young age that they have ASD and would doing so be beneficial for the family and/or the child?

During a guest genetics lecture by a Yale School of Medicine pediatrician and physician-scientist who studies ASD, on March 24, 2020, the doctor was asked about prenatal screening for ASD. They responded that prenatal, genetic testing is a standard form of care (e.g., Down syndrome and fragile X syndrome), so one can imagine that 1 day physicians could run potentially ASD screens for mothers with reliable risk factors and look for positive results. However, they posed that the question then becomes what does the provider, mother, and/or family do with that sensitive information. ASD, like previously mentioned, is a complex neuropsychiatric disorder, and there is no conclusive evidence that a particular gene “causes” autism. The doctor concluded that although it is controversial to predict ASD based on a gene panel, maybe in the future researchers will be more definitive when looking for mutations related to ASD.

Concluding Remarks

It seems too early to tell if a prenatal test for ASD or ASD testing in utero in general is possible. Although the prospect of such a technique is promising and scientifically interesting with real medical applications, one cannot neglect the ethical concerns and bioethical dilemmas that may be raised. More research is needed to establish conclusive genes or biological anomalies that are early, detectable hallmarks of or markers for ASD.

References and Reading

- Anderson, G. M., Jacobs-Stannard, A., Chawarska, K., Volkmar, F. R., & Klinman, H. J. (2007). Placental trophoblast inclusions in autism spectrum disorder. *Biological Psychiatry*, 61(4), 487–491.
- Baron-Cohen, S. J. T. L. (2015). Leo Kanner, Hans Asperger, and the discovery of autism, 386(10001), 1329–1330.
- Batzir, N. A., Shohat, M., & Maya, I. (2015). Chromosomal microarray analysis (CMA) a clinical diagnostic tool in the prenatal and postnatal settings. *Pediatric Endocrinology Reviews: PER*, 13(1), 448–454.
- Brauser, D. (2014). *Routine ultrasounds may detect autism in utero*. Medscape medical news. <https://www.webmd.com/brain/autism/news/20140627/ultrasounds-autism>
- Choi, G. B., Yim, Y. S., Wong, H., Kim, S., Kim, H., Kim, S. V., Hoeffler, C. A., Littman, D. R., & Huh, J. R. (2016). The maternal interleukin-17a pathway in mice promotes autism-like phenotypes in offspring. *Science*, 351(6276), 933–939.
- Hicks, S. D., Rajan, A. T., Wagner, K. E., Barns, S., Carpenter, R. L., & Middleton, F. A. (2018). Validation of a salivary RNA test for childhood autism spectrum disorder. *Frontiers in Genetics*, 9, 534.
- Hollowood, K., Melnyk, S., Pavliv, O., Evans, T., Sides, A., Schmidt, R. J., Hertz-Picciotto, I., Elms, W., Guerrero, E., & Kruger, U. (2018). Maternal metabolic profile predicts high or low risk of an autism pregnancy outcome. *Research in Autism Spectrum Disorders*, 56, 72–82.
- Jeste, S. S., & Geschwind, D. H. (2014). Disentangling the heterogeneity of autism spectrum disorder through genetic findings. *Nature Reviews Neurology*, 10(2), 74.
- Kanne, S. M., & Mazurek, M. O. (2011). Aggression in children and adolescents with ASD: Prevalence and risk factors. *Journal of Autism and Developmental Disorders*, 41(7), 926–937.
- Kanner, L. J. N. C. (1943). Autistic disturbances of affective contact, 2(3), 217–250.
- Maenner, M. J. (2020). Prevalence of autism Spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2016. *MMWR. Surveillance Summaries*, 69.
- Med, A. P. A. J. B. (2013). Diagnostic and statistical manual of mental disorders, 17, 133–137.

- Newschaffer, C. J., Fallin, D., & Lee, N. L. (2002). Heritable and nonheritable risk factors for autism spectrum disorders. *Epidemiologic Reviews*, 24(2), 137–153.
- Park, B. Y., Misra, D. P., Moye, J., Miller, R. K., Croen, L., Fallin, M. D., Walker, C., Newschaffer, C. J., Salafia, C. M., & Consortium, N. C. S. S. (2018). Placental gross shape differences in a high autism risk cohort and the general population. *PLoS One*, 13(8), e0191276.
- Stoner, R., Chow, M. L., Boyle, M. P., Sunkin, S. M., Mouton, P. R., Roy, S., Wynshaw-Boris, A., Colamarino, S. A., Lein, E. S., & Courchesne, E. (2014). Patches of disorganization in the neocortex of children with autism. *New England Journal of Medicine*, 370(13), 1209–1219.
- Zimmerman, A. W., Connors, S. L., Matteson, K. J., Lee, L.-C., Singer, H. S., Castaneda, J. A., & Pearce, D. A. (2007). Maternal antibrain antibodies in autism. *Brain, Behavior, and Immunity*, 21(3), 351–357.

Pre-post Design

► Qualitative Versus Quantitative Approaches

Pre-post-posttest Design

► Qualitative Versus Quantitative Approaches

Preschool

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Definition

Preschools are mandated to provide free and appropriate educational programming to children with autism. The communication, social/emotional, and cognitive delays that children with autism often display qualify them to receive a range of services under Parts B and C of the Individuals with Disabilities Education Act (Stahmer and Mandell 2007). Parts C and B of

IDEA require states to provide individualized educational programming to children with disabilities (P.L. 108–446, 2004).

A large number of children with autism attend preschool (Department of Education 2006). In 2006, 35,081 preschoolers with autism received individualized educational programming as a result of their autism diagnosis (Department of Education 2006). Most studies report the number of preschoolers served under the autism label is increasing (Bitterman et al. 2008; Department of Education 2006; White et al. 2007).

Standard preschool practices do not currently seem to optimize the development of preschoolers with autism. Many public preschool teachers report that they do not use “scientifically based” or “evidence-based” strategies (Hess et al. 2008; Stahmer et al. 2005; Stahmer et al. 2007). In one study, less than 10% of the strategies public school teachers reported using were scientifically based and 33% of the strategies had a limited evidence base (Hess et al. 2008). The most common strategies that public preschool teachers reported using were floor time, incidental teaching, visual schedules, social stories, sensory integration, and music therapy. In a similar study, approximately 33% of the strategies that preschool teachers reported using were evidence based and teachers acknowledged that there was no research regarding 30% of the strategies they used (Stahmer et al. 2005). The most common intervention techniques were applied behavior analysis, floor time, occupational therapy, PECS, sign language, and social stories.

Preschoolers with autism exhibit characteristic impairments in communication and social skills (DSM-IV 1994). Currently, most preschools do not effectively improve upon the communication and social skill deficits of children with autism. Teachers infrequently use validated strategies to promote the social communication of young children with autism. For example, despite that research suggests it is important to respond to the communicative gestures of children with autism (Smith et al. 1988), preschool teachers tend to respond to very few communicative gestures of young children with autism (Keen et al. 2005; Wong 2006). When teachers do respond to the communication of children with autism, they

typically do not respond in ways that will facilitate better social communication (McCathren 2000). Likewise, although children with autism benefit when adults use joint attention gestures (Smith et al. 1988), teachers of children with autism use few joint attention and behavior regulation gestures (McCathren 2000). Similarly, teachers of children with autism typically prompt for verbal communication much less than is recommended for children who are typically developing or developmentally delayed (Chiang 2009).

Preschool teachers also spend relatively little time engaging in pretend play with children who are typically developing or who have autism (Kontos and Keyes 1999; Wong 2006). This reality is troubling because pretend play is believed to be an important component of early childhood autism interventions targeting communication and social skill (Charman et al. 2003). Some contend that preschool programs devote less attention to pretend play than previous decades for several reasons: teachers failing to recognize the importance of play, teachers not knowing how to teach play, class schedules that do not allow time to properly play, as well as greater emphasis upon academic standards (Malone and Langone 1999).

There have been some peer-reviewed publications of preschool interventions targeting the communication and social skill deficits of young children with autism. Most of the experimental interventions report significant improvement in child performance, though they are overwhelmingly plagued by methodological weaknesses. Common shortcomings include a weak experimental design, lack of detail regarding participants, too little information regarding the subjects and experimental treatment, failure to include independent coders and/or assessors, as well as improper data analyses. Future work must correct these weaknesses so that public preschools become a context where the communication and social skill deficits of young children with autism can be effectively improved.

References and Reading

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

- Bitterman, A., Daley, T. C., Misra, S., Carlson, E., & Markowitz, J. (2008). A national sample of preschoolers with autism spectrum disorders: Special education services and parent satisfaction. *Journal of Autism and Developmental Disorders*, 38, 1509–1517.
- Charman, T., Baron-Cohen, S., Swettenham, J., Baird, G., Drew, A., & Cox, A. (2003). Predicting language outcome in infants with autism and pervasive developmental disorder. *International Journal of Language & Communication Disorders*, 38, 265–285.
- Chiang, H. M. (2009). Naturalistic observations of elicited expressive communication of children with autism: An analysis of teacher instructions. *Autism*, 13, 165–178.
- Department of Education. (2007, July 15). *Children and students served under IDEA, part B, in the U.S. and outlying areas by age group, year, and disability category: Fall 1997 through fall 2006*. Retrieved 10 June 2009 from http://www.ideadata.org/tables30th/ar_1-11.htm
- Hess, K. L., Morrier, M. J., Heflin, J., & Ivey, M. L. (2008). Autism treatment survey: Services by children with autism spectrum disorders in public school classrooms. *Journal of Autism and Developmental Disorders*, 38, 961–971.
- Keen, D., Sigafoos, J., & Woodyatt, G. (2005). Teacher responses to the communicative attempts of children with autism. *Journal of Developmental and Physical Disabilities*, 17, 19–33.
- Kontos, S., & Keyes, L. (1999). An ecobehavioral analysis of early childhood classrooms. *Early Childhood Research Quarterly*, 14, 35–50.
- Malone, M. D., & Langone, J. (1999). Teaching object-related play skills to preschool children with developmental concerns. *International Journal of Disability, Development and Education*, 46, 325–336.
- McCathren, R. B. (2000). Teacher-implemented prelinguistic communication intervention. *Focus on Autism and Other Developmental Disabilities*, 15, 21–29.
- Smith, C. B., Adamson, L. B., & Bakeman, R. (1988). Interactional predictors of early language. *First Language*, 8, 143–156.
- Stahmer, A. C., & Mandell, D. S. (2007). State infant/toddler program policies for eligibility and services provision for young children with autism. *Administration and Policy in Mental Health and Mental Health Services Research*, 34, 29–37.
- Stahmer, A. C., Collings, N. M., & Palinkas, L. A. (2005). Early intervention practices for children with autism: Descriptions from community providers. *Focus on Autism and Other Developmental Disabilities*, 20, 66–79.
- White, S. W., Scahill, L., Klin, A., Koenig, K., & Volkmar, F. R. (2007). Educational placements and service use patterns of individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 1403–1412.
- Wong, C. S. (2006). *Play and joint attention of children with autism in the preschool classroom*. Retrieved from ProQuest Information and Learning Company. (UMI: 3226028).

Pre-school Autism Communication Therapy (PACT)

► [Autism Family Experience Questionnaire \(AFEQ\)](#), The

Pre-school Autism Communication Trial (PACT Trial)

► [Autism Family Experience Questionnaire \(AFEQ\)](#)

Preschool Curricula

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Definition

Preschool curriculum provides a guide for *what* to teach young children. Usually organized in a developmental sequence, preschool curriculum traditionally refers to the content of what is taught, which can be comprehensive or focus on specific domains. For children with autism, preschool curriculum typically addresses the following target areas: social-communication skills, problem behaviors, sensory and motor skills, pre-academic skills, and adaptive behavior.

Beyond content, preschool curriculum can also include assessment procedures for setting goals and monitoring progress as well as intervention strategies for guiding *how* to teach young children. Practitioners working with young children

with autism can follow a published preschool curriculum or draw from multiple sources to design a more specific curriculum.

Curriculum Design/Selection

Autism is a spectrum disorder in which there is a great deal of heterogeneity. Therefore, curricular decisions must be individualized for young children with autism as well as their families. Following is a brief summary of issues to consider when selecting and adapting preschool curriculum for young children with autism.

Target Early Core Characteristics of Autism

In designing or selecting curriculum for a young child with autism, the child's skills must be considered in relation to the early core characteristics of autism: social-communication and repetitive behavior or restricted interests. Specifically, young children with autism have difficulty with engagement, joint attention, and symbolic play, all of which affect learning and development (Kasari et al. 2008).

Engagement. If a child is not purposefully engaged in activities or interactions, the opportunities for learning are dramatically reduced (Iovannone et al. 2003). In addition to active engagement, an important curricular goal for young children with autism is to increase the amount of time children with autism are *jointly* engaged in activities while shifting their attention between the activity and their interaction partners (Kasari et al. 2010).

Joint Attention. Joint attention refers to the ability to shift attention between objects or events and other people in order to share interest and plays an important role in language development (Loveland and Landry 1986). Specific skills include pointing and showing with coordinated joint looks which involves a child looking at a target object, then making eye contact with another person, and then back to the target object. For example, to share interest about a toy, a child may be looking at the toy, then glance up to look at the caregiver, and then look back at the toy. Compared to typically developing children and children with intellectual disabilities matched on mental age, children with autism have specific

deficits in initiating and responding to joint attention (Mundy et al. 1986). Children with autism are more likely to use pointing and attention skills to regulate others' behaviors rather than to share interest.

Symbolic Play. Symbolic or pretend play involves the representation of objects as something else. Children typically progress developmentally from playing with toys functionally, such as in constructive and manipulative play, to playing with toys symbolically (Lifter et al. 1993). However, in comparison to typically developing peers, children with autism at the same mental ages have significant delays in the development of symbolic play (Jarrold et al. 1993). Children with autism tend to manipulate toys or objects in a rigid or stereotyped manner rather than spontaneously initiate creative symbolic play activities (Libby et al. 1998).

Functionality

In addition to targeting the early core characteristics of autism, a well-designed preschool curriculum for young children with autism must also include content that serves a functional purpose for the child (Schertz et al. 2010). It is critical to focus on skills that are required for the child to be functional in a less restrictive or natural environment. For example, reducing aggressive behaviors and tantrums will allow the child to engage socially with others and, thus, may serve a more functional purpose than being able to color a picture within the lines.

Generalizability. A key concept regarding functionality is generalizability. Generalizing refers to extending what has been learned in one context to new and different contexts. Care must be taken so that children with autism can perform particular acts beyond the specific setting and manner they were taught. For curricular goals to be truly functional, they should have meaning in the child's everyday activities. Therefore, preschool curriculum must extend beyond interactions with only one person, setting, and/or activity. Furthermore, because generalizing may be particularly difficult for young children with autism, learning may be more meaningful and effective when specific skills are taught in natural settings.

Developmentally Appropriate

Preschool curricula are usually organized in a developmental sequence beginning with more basic skills and leading to more complex skills within a domain. In deciding where to begin within a curriculum, the individual goals or targets for the young child with autism must be developmentally appropriate, meaning that they are just above the child's level (zone of proximal development; Vygotsky 1978). Content that is too easy may not be stimulating enough while content that is too difficult may lead to frustration. Therefore, it is important to target a child's emerging skills and then guide or scaffold the child to master that skill.

Socially and Culturally Relevant

Finally, preschool curriculum needs to be socially and culturally relevant to children with autism, their families, and their communities. In addition to considering developmental level, the curriculum and materials must also reflect what is socially appropriate for the child's age (age appropriateness; Schertz et al. 2010). Although children with autism often show an uneven pattern of development, it is important to note the general characteristics of typically developing preschool-aged children in the community. While strengths and special interests should be nurtured and integrated within a child's curriculum, especially to help increase skills in areas of weakness, special attention must be paid to social norms in order to better facilitate independent functioning in natural environments. Indeed, in preschool, play is the natural setting for learning and interacting with others. The involvement of a professional familiar with typically developing preschoolers (e.g., preschool teacher) is essential to validating social relevance.

Even more critical is the inclusion of the family in determining social and cultural relevance. Input from the family will determine the curricular goals that are most valued and should be prioritized. It is the responsibility of the professionals to provide information to the families to help empower them to make informed decisions and make their preferences known.

Curriculum materials that reflect children's cultural and ethnic backgrounds will also promote generalization and positive ethnic identity (Hall 2009).

Assessment

Many of the issues identified previously in designing and/or selecting preschool curriculum require extensive knowledge of the individual child with autism, thereby requiring assessments in order to appropriately individualize preschool curriculum. Assessment is a process of collecting information in order to make decisions. In this case, assessment of a child is needed to provide data for making decisions about what to teach the child with autism. This process is important initially for deciding upon the curriculum and then adapting the curriculum as appropriate in order to monitor progress on an ongoing process (Schertz et al. 2010). The most helpful type of assessments for curricular decisions is usually curriculum-based assessments.

Curriculum-based assessments (also known as criterion-referenced tests) include a list of skills and behaviors (usually linked to a specific curriculum) that can be measured and recorded. To be useful, the assessment measure must be sensitive to changes. Performance on these objectives is scored which provides information on the child's mastered and emerging skills. In this way, a developmentally appropriate curriculum can be specified for the individual child with autism. The skills are then measured at regular intervals in order to track progress with objectives. This monitoring provides data to determine if curricular content and sequence and/or intervention strategies need to be modified. Most published preschool curriculum guides will include a curriculum-based assessment that parallels the curriculum content.

Intervention

Although the strict definition of preschool curriculum only refers to the *content* of what is taught, many published curricula today often include intervention strategies that guide *how* to teach the specified skills and behaviors. Intervention guidelines may range from providing a general approach or practices to offering specific activities and materials for facilitating the target skills. Although interventions within a curriculum may be based off of different theoretical models, it is imperative to acknowledge the same key issues for making curricular decisions: targeting the early core characteristics of autism and ensuring

functionality, developmental appropriateness, and social and cultural relevance.

Commonly Used Preschool Curricula

Published preschool curricula that can be used for young children with autism include those designed for typically developing children, children with special needs, and specifically for autism. Following is a brief description of commonly used published curricula for preschoolers with autism. This list is presented in alphabetical order and is not intended to be comprehensive:

- *Assessment, Evaluation, Programming System for Infants and Children, Second Edition* (AEPS; Bricker 2002). The AEPS is a 4-volume set of assessment and curriculum materials for use with young children at birth through age 6. Assessment and curriculum items are organized into six key domains: social, social-communication, fine motor, gross motor, adaptive, and cognitive.
- *Assessment of Basic Language and Learning Skills-Revised* (ABLLS-R; Partington 2006). The ABLLS-R includes a curriculum-based assessment, curriculum guide, and skills tracking system for use with children with autism or other developmental disabilities between the ages of 3 and 9. With a particular emphasis on language skills, the ABLLS-R also includes information for developing an Individualized Education Program (IEP) for the child.
- *The Carolina Curriculum for Preschoolers with Special Needs, Second Edition* (CCPSN; Johnson-Martin et al. 2004). The CCPSN is designed for assessing and teaching children with mild to severe special needs from 2 to 5 years' developmental age. Organized into 22 teaching sequences spanning five developmental domains, each curricular item includes suggested teaching procedures, materials, routine integration strategies, and sensorimotor adaptations.
- *The Creative Curriculum for Preschool, Fourth Edition* (Dodge et al. 2006) provides suggestions for arranging educational environment so that goals and objectives can be embedded within a daily schedule while balancing both teacher-directed and child-

initiated learning. The Creative Curriculum includes an assessment instrument as well as specific intervention strategies.

- *Hawaii Early Learning Profile 3–6* (HELP 3–6; Teaford et al. 2010). HELP 3–6 is a family-centered, comprehensive, and developmentally sequenced curriculum-based assessment that also offers play-based activities and intervention strategies for the behaviors and skills. Covering 622 skills in six domains, HELP 3–6 is designed for typically developing children as well as children who may have developmental delays.
- *More Than Words* (Sussman 1999). Drawn from *More Than Words – The Hanen Program for Parents of Children with Autism Spectrum Disorder*, this resource book was developed by speech-language pathologists and provides practical, research-based strategies using everyday activities and includes charts and checklists. Designed for parents, *More Than Words* specifically focuses on promoting communication and social skills in children with autism spectrum disorder.
- *The SCERTS Model: A Comprehensive Educational Approach for Children with Autism Spectrum Disorders* (Prizant et al. 2006). The SCERTS model stands for social-communication, emotional regulation, and transactional supports to improve the communication and social-emotional abilities in individuals with autism spectrum disorders. This resource includes two volumes: Volume I targets assessment and volume 2 targets program planning and intervention.
- *Selecting Teaching Programs* (Taylor and McDonough 1996, chapter in Maurice, Green, & Luce (Eds.), *Behavioral intervention for young children with autism: A manual for parents and professionals*). This chapter provides detailed teaching programs and a simple curriculum guide for implementing a behavioral program for children with autism.
- *Teaching Individuals with Developmental Delays: Basic Intervention Techniques* (Lovaas 2003). Guided by a behavioral approach and targeted towards the education of individuals with autism, this book provides detailed descriptions of current programs

within the following sections: basic concepts, transition to treatment, early learning concepts, expressive language, strategies for visual learners, and programmatic consideration.

- *A Work in Progress: Behavior Management Strategies and a Curriculum for Intensive Behavioral Treatment of Autism* (Leaf and McEachin 1999). A Work in Progress is a comprehensive and detailed curriculum guide that presents behaviorally based intervention strategies for families and/or professionals working with children with autism.
- *The Ziggurat Model – A Framework for Designing Comprehensive Interventions for Individuals with High-Functioning Autism and Asperger Syndrome* (Aspy and Grossman 2011). The Ziggurat Model provides a process for designing interventions for individuals with autism spectrum disorder. Included in the book is the Underlying Characteristics Checklist for Early Intervention which identifies characteristics associated with autism across multiple domains.

Historical Background

Although Public Law 94–142 gave students with disabilities the right to a free and appropriate public education in 1975, it was not until 1986 when those provisions were extended to preschool-aged children with disabilities (P.L. 99–857). Additionally, while autism was first described in 1943, autism was not added as a distinct federal disability category until 1990 (P.L. 101–476). Paralleling the recognition of autism as a separate category is the rising prevalence of autism. In the 1980s, prevalence rates for autism were estimated to be between 2 and 5 per 10,000. The Centers for Disease Control and Prevention (2009) now report current prevalence rates to be 1 in 110 (Schertz et al. 2010). Furthermore, while symptoms of autism appear before the age of 3, diagnosis often occurred later. However, with the broadening of the autism diagnostic criteria, greater public awareness of autism, and improved screening and diagnostic assessment tools and procedures, the majority of children with autism are diagnosed during the preschool years (Howlin and Moore 1997; Wiggins et al. 2006). In fact, the U.S. Department of Education, Special

Education Programs and Data Analysis System (2007) reported that 35,081 preschoolers received special education services under the autism category in 2006.

Different theoretical models of intervention have guided the development of distinct curriculum for children with autism (Olley 1999). Specific intervention for children with autism began in the 1970s with the structured teaching approach that has now become the TEACCH program and the applied behavioral approach (Schertz et al. 2010). Developmental approaches produced curricula based upon typical developmental sequences of learning (Mirenda and Donnellan 1987), while behavioral approaches tended to target eliminating or reducing interfering behaviors while promoting more positive behaviors. Current preschool curriculum for children with autism tends to provide a more integrated approach and now includes content for each of the major developmental domains. However, specific emphasis may be given to communication, social skills, and reduction of interfering behaviors, consistent with the core symptoms of autism. At this time, there are no national curricular standards for educating young children with autism (Olley 1999).

Rationale or Underlying Theory

Children with autism who participate in preschool programs with curriculum that targets core domains of impairment tend to have improved outcome and prognosis than those who do not. Curriculum provides a guide for systematic intervention. Regardless of intervention approach, the critical factor seems to be providing instruction that is thoughtfully planned and systematically delivered (Schertz et al. 2010). According to Iovannone et al. (2003) "Systematic instruction involves carefully planning for instruction by identifying valid educational goals, carefully, outlining instructional procedures for teaching, implementing the instructional procedures, evaluating the effectiveness of the teaching procedures, and adjusting instruction based on data." (p. 157). Therefore, preschool curriculum plays a critical role in the education of young children with autism in being linked systematically with assessment and intervention.

Treatment Participants

The preschool years are typically defined as ages 3–5. In the United States, local school systems provide education for children with special needs beginning at age 3, while Part C early intervention programs provide services under age 3.

Individuals responsible for preschool curriculum planning will include all members of the instructional or Individualized Educational Planning (IEP) team. This team includes family members and an intervention specialist or teacher as well as other specific service providers such as a speech-language pathologist, occupational therapist, adaptive physical education teacher, physical therapist, and/or school psychologist. A general education teacher can arrange for adaptations and supports needed for access to the general preschool curriculum as well as to promote successful inclusion with typically developing peers. Autism specialists may also be included to help the team design curriculum for young children with autism (Hall 2009).

Efficacy Information

Although research shows that intervention at young ages can improve developmental outcomes and trajectories for children with autism (National Research Council 2001), there is less consensus regarding specific intervention approaches. As a result of this controversy, the importance of curriculum has been overlooked in the literature (Olley 1999). Therefore, at this time, there is no standard curriculum for young children with autism as no single curriculum has been shown to be universally effective.

Overall, because of the focus on intervention and establishing evidence-based practices, little research has been conducted on determining what is important to teach and the sequence in which content should be taught. Reviews of the literature have identified domains of functioning that warrant intervention, strategies often used to target those domains, and a brief description of existing programs (Dawson and Osterling 1997; National Research Council 2001; Odom et al. 2010). Indeed, there have been many well-

designed studies evaluating intervention programs with well-developed curricula but few have evaluated the curriculum guides themselves. Research findings typically address outcomes as a result of the entire treatment package which address both content and intervention strategy, thus making it difficult to determine the specific effects of the curricular component versus the instructional component (Olley 1999).

Future research should look more closely at preschool curriculum to determine the scope and sequence of skills in order to determine the content most critical for young children with autism to learn for promoting later development.

See Also

- Curriculum
- Education
- PL94-142
- Preschool

References and Reading

- Aspy, R., & Grossman, B. G. (2011). *The Ziggurat Model: A framework for designing comprehensive interventions for individuals with high-functioning autism and Asperger syndrome*. Shawnee Mission: Autism Asperger Publishing.
- Bricker, D. (2002). *AEPS: Assessment, evaluation, and programming system for infants and children, Second edition, volume 4, Curriculum for three to six years*. Baltimore: Paul H. Brookes Publishing Co..
- Dawson, G., & Osterling, J. (1997). Early intervention in autism. In M. J. Guralnick (Ed.), *The effectiveness of early intervention* (pp. 307–326). Baltimore: Paul H. Brookes.
- Dodge, D. T., Colker, L. J., & Heroman, C. (2006). *The creative curriculum for early childhood* (4th ed.). Washington, DC: Teaching Strategies.
- Hall, L. J. (2009). *Autism spectrum disorders: From theory to practice*. Upper Saddle River: Merrill Prentice Hall.
- Hobson, R. P. (1989). Beyond cognition: A theory of autism. In G. Dawson (Ed.), *Autism: Nature, diagnosis, and treatment* (pp. 22–48). New York: Guilford Press.
- Howlin, P., & Moore, A. (1997). Diagnosis in autism: A survey of over 1200 patients in the UK. *Autism, 1*, 135–162.
- Iovannone, R., Dunlap, G., Huber, H., & Kincaid, D. (2003). Effective educational practices for students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities, 18*, 150–165.
- Jarrold, C., Boucher, J., & Smith, P. (1993). Symbolic play in autism: A review. *Journal of Autism and Developmental Disorders, 23*, 281–307.
- Johnson-Martin, N. M., Hacker, B., & Attermeier, S. M. (2004). *Carolina Curriculum for Preschoolers with Special Needs* (2nd ed.). Baltimore: Paul H. Brookes Publishing.
- Kasari, C., Paparella, T., Freeman, S. F., & Jahromi, L. B. (2008). Language outcome in autism: Randomized comparison of joint attention and play interventions. *Journal of Consulting and Clinical Psychology, 76*, 125–137.
- Kasari, C., Gulsrud, A., Wong, C., Kwon, S., & Locke, J. (2010). Randomized controlled caregiver mediated joint engagement intervention for toddlers with autism. *Journal of Autism and Developmental Disorders, 40*, 1045–1056.
- Leaf, R., & McEachin, J. (1999). *A work in progress: Behavior management strategies and a curriculum for intensive behavioral treatment of autism*. New York: DRL Books, L.L.C.
- Libby, S., Powell, S., Messer, D., & Jordan, R. (1998). Spontaneous play in children with autism: A reappraisal. *Journal of Autism and Developmental Disorders, 28*, 487–497.
- Lifter, K., Sulzer-Azaroff, B., Anderson, S., & Cowdery, G. E. (1993). Teaching play activities to preschool children with disabilities: The importance of developmental considerations. *Journal of Early Intervention, 17*, 139–159.
- Lovaas, O. I. (2003). *Teaching individuals with developmental delays: Basic intervention techniques*. Austin: Pro-Ed.
- Loveland, K. A., & Landry, S. H. (1986). Joint attention and language in autism and developmental language delay. *Journal of Autism and Developmental Disorders, 16*, 335–349.
- Mirenda, P. L., & Donnellan, A. M. (1987). Issues in curriculum development. In D. J. Cohen & A. M. Donnellan (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 211–226). New York: Wiley.
- Mundy, P., Sigman, M. D., Ungerer, J., & Sherman, T. (1986). Defining the social deficits of autism: The contribution of non-verbal communication measures. *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 27*(5), 657–669.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Odom, S. L., Boyd, B. A., Hall, L. J., & Hume, K. (2010). Evaluation of comprehensive treatment models for individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 40*, 425–436.
- Olley, J. G. (1999). Curriculum for students with autism. *School Psychology Review, 28*, 595–607.
- Partington, J. W. (2006). *The assessment of basic language and learning skills- Revised (The ABLLS-R): An assessment, curriculum guide, and skills tracking system for children with autism or other developmental disabilities*. Pleasant Hill: Behavior Analysts.
- Prizant, B. M., Wetherby, A. M., Rubin, E., Laurent, A. C., & Rydell, P. J. (2006). *The SCERTS model: A comprehensive*

- educational approach for children with autism spectrum disorders*. Baltimore: Paul H. Brookes.
- Schertz, H., Wong, C., & Odom, S. L. (Eds.). (2010). *Young exceptional children monograph no 12: Autism spectrum disorders*. Missoula: Division for Early Childhood.
- Sussman, F. (1999). *More than words: Helping parents promote communication and social skills in children with autism spectrum disorder*. Toronto: The Hanen Centre.
- Taylor, B. A., & McDonough, K. A. (1996). Selecting teaching programs. In C. Maurice, G. Green, & S. C. Luce (Eds.), *Behavioral intervention for young children with autism: A manual for parents and professionals* (pp. 63–177). Austin: Pro-Ed.
- Teafor, P., Wheat, J., & Baker, T. (2010). *Hawaii early learning profile 3–6*. Palo Alto: Vort Corporation.
- U.S. Department of Education, Office of Special Education Programs, Data Analysis System (2007, July 15). *Children with disabilities receiving special education under Part B of the Individuals with Disabilities Education Act 1997–2006*. Retrieved September, 2011. http://www.ideadata.org/tables30th/ar_1-11.htm
- Vygotsky, L. S. (1978). *Mind in society: The development of higher psychological processes*. Cambridge, MA: Harvard University Press.
- Wiggins, L. D., Baio, J., & Rice, C. (2006). Examination of the time between first evaluation and first autism spectrum diagnosis in a population-based sample. *Journal of Developmental and Behavioral Pediatrics*, 27, S79–S87.

child has receptive or expressive language delay/disorder or combination of both as well as to determine eligibility for early intervention or speech/language services. Item responses are obtained through a combination of direct testing, observation of spontaneous interactions, and through caregiver report. It is comprised of two subscales: auditory comprehension and expressive communication. Areas covered in the receptive and expressive subscales include preverbal skills, language content and structure, integrative language skills (thinking using language), and emergent literacy. Standard scores, percentile ranks, and age equivalents are derived for each subscale as well as an overall total language score. It also provides three supplemental assessments: language sample checklist, articulation screener, and caregiver questionnaire. Criterion scores are derived for the articulation screener for children 2 years 6 months to 7 years 11 months of age.

Historical Background

The Preschool Language Scale was first developed in 1969 as a measure of language development in young children. It was revised several times, and in 1992, the PLS-3 was published using normative data for standard scores and percentile ranks; the PLS-IV appeared in 2002, with a Spanish language version. The PLS-5, published in 2011, is the latest edition, which also includes a Spanish version.

Psychometric Data

From 45 states in the USA, 1,400 children participated in the standardization normative sample, collected in more than 45 states in the United States. The standardization sample matches US Census figures (March 2008 update) for age, sex, geographic region, race/ethnicity, and primary caregiver's education. Norms are reported for 3-month intervals for children from birth to 11 months and 6-month intervals for ages 12 months to 7 years 11 months. Clinical studies include a developmental delay study and three language disorder

Preschool Edition

► Sensory Processing Measure

Preschool Language Scale – 5

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Synonyms

PLS-5

Description

The Preschool Language Scale-Fifth Edition (PLS-5) was developed to identify whether a

studies (children with receptive language disorder, expressive language disorder, and both receptive and expressive disorder); case studies include children identified as high risk (environmental risk) and children identified with autism. Multiple reviews for bias were conducted throughout the test development. Nationally recognized experts in assessment and bias issues conducted a thorough review of test items and picture stimuli. An additional sample of African American and Hispanic children was tested to conduct the statistical analysis of bias. Split-half reliabilities range from 0.80 to 0.97. Sensitivity for the total language score is 0.83; specificity is 0.80.

Clinical Uses

It may be administered by a variety of qualified users such as speech-language pathologists, early childhood specialists, and psychologists as well as professionals trained to administer the assessment. Paraprofessionals may also administer the assessment; however, it should be scored and interpreted by experienced professionals. The purpose of the assessment is to identify delays inexpressive and/or receptive language development.

See Also

- [Expressive Language](#)
- [Receptive Language](#)

References and Reading

- Harris, S. L., Handleman, J. S., Gordon, R., Kristoff, B., & Fuentes, F. (1991). Changes in cognitive and language functioning of preschool children with autism. *Journal of Autism and Developmental Disorders*, 21, 281–290.
- Thurm, A., Lord, C., Li-Ching, L., & Newschaffer, C. (2006). Predictors of language acquisition in preschool children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 1721–1734.
- Zimmerman, I. L., Steiner, V. G., & Pond, E. R. (2011). *Preschool language scale* (5th ed.). San Antonio: Pearson.

Present Level of Growth or Knowledge

- [Annual Review](#)

Pressure/Squeeze Machine

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Definition

The squeeze machine, otherwise known as Grandin's Hug Machine, is a device that allows self-administered deep pressure along the sides of the body. This device was developed by Temple Grandin, a woman who has autism, to reduce her level of anxiety and arousal. The machine allows an individual to be positioned prone in the machine with the head supported outside of the machine, which looks like a large box. The box is open on each end with your head and forearms out one end and your lower legs out the other end. The individual is then able to control and grade the amount of pressure using a switch. An investigation using two groups of children, all diagnosed with autism, demonstrated the effectiveness of using Grandin's Hug Machine to decrease the level of arousal measured by galvanic skin response and the level of anxiety measured by the Connors Parent Rating Scale. Children who used Grandin's Hug Machine twice a week for 6 weeks displayed reduction in the level of arousal and reduced anxiety using these measures and when compared to children who were positioned in the Hug Machine but without the availability of receiving the deep pressure.

References and Reading

- Edelson, S. M., Edelson, M. G., Kerr, D. C. R., & Grandin, T. (1999). Behavioral and physiological effects of deep pressure on children with autism: A pilot study evaluating the efficacy of Grandin's Hug Machine. *American Journal of Occupational Therapy*, 53, 145–152.

- Inamura, K. N., Wiss, T., & Parham, D. (1990a). The effects of Hug Machine usage on the behavioral organization of children with autism and autistic-like characteristics: Part 1. *Sensory Integration Quarterly*, 18, 1–5.
- Inamura, K. N., Wiss, T., & Parham, D. (1990b). The effects of Hug Machine usage on the behavioral organization of children with autism and autistic-like characteristics: Part 2. *Sensory Integration Quarterly*, 18, 1–5.

Presupposition

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Synonyms

[Theory of mind](#)

Definition

Presupposition is an aspect of pragmatic or social language use. It entails an implicit assumption about the background knowledge relating to an utterance whose truth is taken for granted in discourse. A presupposition must be mutually known or assumed by the speaker and listener for the utterance to be considered appropriate in context.

Example of presupposition includes:

- Jim no longer teaches children with ASD.
 - Presupposition: Jim once taught children with ASD.

Presuppositional knowledge allows speakers to provide the correct amount of information necessary to get their message across to a listener. For example, it is not necessary to say “Jim once taught children with ASD. Jim no longer teaches children with ASD.” The second sentence presupposes the first, so the first does not need to be stated explicitly.

Many individuals with ASD have difficulty with presuppositional knowledge. They may provide too much or too little information because they have difficulty taking into account what their listener can reasonably be expected to know. For example, a speaker with ASD may start a conversation by saying, “WOW is my favorite game,” without determining whether it is reasonable to expect that the listener will know that WOW refers to World of Warcraft. Difficulties in the use of presuppositional knowledge can result in communication breakdowns and affect others’ perceptions of the speaker and their willingness to engage in conversation with him. Presuppositional skills are closely related to the “theory of mind” skills that are thought to be impaired in individuals with ASD.

See Also

- [Pragmatic Language](#)
- [Theory of Mind](#)

References and Reading

- Paul, R., Orlovski, S. M., Marcinko, H. C., & Volkmar, F. (2009). Conversational behaviors in youth with high-functioning ASD and Asperger syndrome. *Journal of Autism and Developmental Disorders*, 39, 115–125.
- Thurm, A., Lord, C., Lee, L. C., & Newschaffer, C. (2007). Predictors of language acquisition in preschool children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 1721–1734.
- Winner, M. G. (2007). *Thinking about YOU, thinking about ME: Teaching perspective taking and social thinking to persons with social cognitive learning challenges* (2nd ed.). San Jose: Think Social Publishing.

Pretend Play

- [Play](#)

Pretense

- [Imagination](#)

Prevalence

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Synonyms

[Period prevalence](#); [Point prevalence](#)

Definition

Prevalence is the percent of persons in a population with a certain condition. Prevalence combines both preexisting cases and incident cases together to form a numerator, which is then divided by the total population being studied. Like incidence, prevalence must be calculated for a specified time frame. If that time frame spans a broad range of time (i.e., a year, 10 years, etc.), then prevalence over that time frame is referred to as a “period prevalence.” If the time frame is relatively short, then the prevalence measure is often called “point prevalence.”

An example of when point prevalence would be measured would be visiting a clinic and counting the number of children with and without ASD on that day. Cases of ASD could be children who were diagnosed with ASD on that day, as well as children who had been previously diagnosed. Point prevalence is often compared to a photograph: data are collected all at once (or over a brief time period), and the majority of cases are preexisting cases, as there is little to no time for incident cases to occur. Period prevalence, on the other hand, has a broader time frame for measurement of preexisting and new cases, which may allow more new cases of disease to develop. An example of period prevalence could involve following the children in the prior example for a year. Those who were diagnosed with ASD within that year would be combined with children with prior ASD diagnoses to form a numerator, and the denominator would be the total number of children being followed in the cohort.

See Also

► [Incidence](#)

References and Reading

- Rothman, K. J., Greenland, S., & Lash, T. L. (2008). *Modern epidemiology* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
- Szklo, M., & Nieto, J. (2006). *Epidemiology: Beyond the basics* (2nd ed.). Boston: Jones and Bartlett.

Preverbal Communication

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Synonyms

[Early social communication](#); [Prelinguistic communication](#)

Definition

The term “preverbal communication” describes a group of social communication behaviors that are used to send an intentional, or purposeful, message to a communicative partner. Rather than being verbal in form (i.e., composed of words or word combinations), preverbal communication acts consist of some combination of eye gaze, gesture, and/or vocalization. The core feature of a preverbal communication act is that the child purposefully directs this behavior toward a communicative partner. Preverbal communication acts can take the form of conventional gestures such as showing, giving, open-handed reaching, pointing, nodding or shaking the head, and waving. Leading the adult by the hand toward a desired item also is a common preverbal communication strategy observed in children with autism. The most common pragmatic functions of preverbal communication acts are commenting and requesting.

See Also

- [RJA/IJA \(Initiating/Responding to Joint Attention\)](#)

References and Reading

- Brady, N. C., Marquis, J., Fleming, K., & McLean, L. (2004). Prelinguistic predictors of language growth in children with developmental disabilities. *Journal of Speech, Language, and Hearing Research*, 47, 663–677.
- Mundy, P., & Crowson, M. (1997). Joint attention and early social communication: Implications for research on intervention with autism. *Journal of Autism and Developmental Disorders*, 27, 653–676.
- Mundy, P., Sigman, M., & Kasari, C. (1994). Joint attention, developmental level, and symptom presentation in autism. *Development and Psychopathology*, 6, 389–401.
- Shumway, S., & Wetherby, A. M. (2009). Communicative acts of children with autism spectrum disorders in the second year of life. *Journal of Speech, Language, and Hearing Research*, 52, 1139–1156.
- Toth, K., Munson, J., Meltzoff, A. N., & Dawson, G. (2006). Early predictors of communication development in young children with autism spectrum disorder: Joint attention, imitation, and toy play. *Journal of Autism and Developmental Disorders*, 36, 993–1005.
- Warren, S. F., Yoder, P. J., Gazdag, G. E., & Kim, K. (1993). Facilitating prelinguistic communication skills in young children with developmental delay. *Journal of Speech and Hearing Research*, 36, 83–97.

Prevocational Skills

- [Preemployment Skills for People with ASD](#)

Primacy Effects

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Synonyms

[Cognitive bias](#); [Recency effect](#); [Serial position effect](#)

Definition

Primacy effect is the tendency to remember the first few things presented when given a list of items to recall. An individual will also have a tendency to assume that those things presented at the beginning of a list are of greater importance than those presented in the middle or end of the list. The first item or first few items may be transferred to long-term memory by the time of recall, while items at the end of the list are still in short-term memory. This has implications for learning, memorizing information, and making selections.

This effect was first described by Asch (1946) when he requested that participants judge a person based upon a list of descriptive attributes. Two lists, containing identical positive and negative descriptors, were presented. In one instance, the positive descriptors were presented at the beginning of the list, and in the other instance, the negative descriptors were presented at the beginning of the list. Those participants who were presented with the list of positive descriptors first rated the person more highly than those participants who were presented with the list of negative descriptors first.

See Also

- [Recency Effect](#)
- [Short-Term Memory](#)

References and Reading

- Anderson, N. H., & Hubert, S. (1963). Effects of concomitant verbal recall on order effects in personality impression formation. *Journal of Verbal Learning and Verbal Behavior*, 2, 379–391.
- Asch, S. E. (1946). Forming impressions of personality. *Journal of Abnormal and Social Psychology*, 41, 258–290.
- Bowler, D., Limoges, E., & Mottron, L. (2009). Different verbal learning strategies in autism spectrum disorder: Evidence from the Rey auditory verbal learning test. *Journal of Autism and Developmental Disorders*, 39, 910–915.
- Deese, J., & Kaufman, R. (1957). Serial effects in recall of unorganized and sequentially organized verbal material. *Journal of Experimental Psychology*, 54, 180–187.
- Ebbinghaus, H. (1913). *On memory: A contribution to experimental psychology*. New York: Teachers College.

- Murdock, B. B., Jr. (1962). The serial position effect of free recall. *Journal of Experimental Psychology*, 64, 482–488.
- Rosnow, R. (1966). Whatever happened to the “law of primacy”. *Journal of Communication*, 16, 10–31.
- Tager-Flusberg, H. (1991). Semantic processing in the free recall of autistic children: Further evidence for a cognitive deficit. *British Journal of Developmental Psychology*, 9, 417–430.

Primary Motor Cortex

► [Precentral Gyrus](#)

Primary Sensory Areas

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Synonyms

[Auditory cortex](#); [Somatosensory cortex](#); [Visual cortex](#)

Definition

Areas of the cerebral cortex that receive the first signals from sensory receptors are called primary sensory areas. These areas are separate for each sensory modality, but the major primary sensory regions are those for vision, hearing, and sensation.

Vision: Primary visual cortex lies in the lingual gyrus of the occipital lobe on both sides of the calcarine sulcus. Histologically termed Brodmann’s area 17, primary visual cortex is organized topographically, such that each part of the cortex represents a specific part of one’s visual field.

Hearing: Primary auditory cortex lies along the upper bank of the temporal lobe in the transverse temporal gyri, also termed Heschl’s gyrus. Histologically, the neurons comprising primary auditory cortex (A1) are labeled Brodmann’s areas 41 and 42. Auditory information from both ears

is processed in both the right and left hemispheres, and the organization is tonotopic: Different areas of cortex respond preferentially to different sound frequencies.

Sensation: The brain region that processes tactile sensations is called primary somatosensory cortex. Located on the postcentral gyrus in the parietal lobe just behind motor cortex in Brodmann’s areas 2, 1, and 3, the “sensory strip” has a topographic representation in the form of a homunculus, with exaggerated representations of sensitive areas such as the hands, lips, tongue, and genitals.

See Also

- [Auditory Cortex](#)
► [Visual Cortex](#)

References and Reading

- Kaas, J. H. (2011). Reconstructing the areal organization of the neocortex of the first mammals. *Brain, Behavior and Evolution*, 78(1), 7–21.
- LeBlanc, J. J., & Fagiolini, M. (2011). Autism: A “critical period” disorder? *Neural Plasticity*, 2011, 921680.

Primary Visual Cortex

[Visual Cortex](#)

Primate Prosocial Behaviors

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Definition

Cooperation: Umbrella term for social interactions among conspecifics which provide a benefit

to another individual (recipient) or to both the actor and the recipient. Cooperative behaviors thus comprise both altruistic and mutualistic interactions (Melis and Semmann 2010).

Altruistic or investing behaviors: Social behaviors in which only the recipient obtains immediate benefits, and actors' motivation is to intervene toward the recipient's goal or need (Warneken and Melis 2012).

Collaborative behaviors: Mutually beneficial behaviors in which two or more individuals coordinate their actions to produce outcomes from which both individuals benefit immediately, such as obtaining a common resource or producing an effect that one individual would not be able to produce on her or his own (Warneken and Melis 2012).

Historical Background

Human cooperative behavior exceeds that of all other species with regard to the scale and range of cooperative activities. Specifically, how did humans become so collaborative and skilled at maintaining cooperation in large groups of unrelated individuals? Which cooperation skills do we share with other species and which are unique to humans? Comparative studies of humans and chimpanzees as well as bonobos as our two closest living primate relatives can help us to identify those cooperative skills that already characterized the last common ancestor and those derived skills unique to the human lineage.

For a long time, natural observations have accumulated evidence that nonhuman primates behave cooperatively across many different contexts such as food sharing, allo-maternal care, territorial and predator defense, grooming, coalitionary support and aggression, and cooperative hunting (Kappeler and van Schaik 2006). However, it is only in the last decade that experimental studies have started to investigate the psychological mechanisms that underlie these cooperative behaviors.

Here, we will focus mainly on the experimental work conducted with chimpanzees and bonobos, since they are our closest living evolutionary

relatives, and a significant body of empirical evidence has accumulated in recent years, occasionally even allowing direct comparisons between humans and mainly chimpanzees.

Current Knowledge

We will first summarize the available evidence for altruistic behaviors in chimpanzees, focusing on both food-sharing behavior and instrumental helping. Altruistic behaviors are particularly interesting from a motivational point of view, since individuals act on behalf of others, without obtaining any immediate benefit from it, and potentially incurring serious costs. Although altruistic behaviors in humans are often maintained via reciprocation, there is no good evidence suggesting that nonhuman primates understand the long-term benefits of alternating favors or that they engage in altruistic behavior motivated by the prospect of a future selfish reward. From a cognitive point of view, altruistic behaviors require that individuals attend to and can infer what others need or desire.

Second, we will summarize the current knowledge on chimpanzees' and bonobos' ability to collaboratively solve problems. Collaborative behaviors are from a motivational perspective easier to explain, since individuals should be generally selfishly motivated to collaborate (unless they can free-ride profiting from the work of others without making any effort themselves, as in large-scale forms of cooperation). The challenge of collaborative behaviors is instead cognitive, since individuals need to understand how the different roles are interrelated with each other and how best to coordinate their behavior to that of the partner(s) in order to achieve the joint goal.

Altruistic Behaviors in Chimpanzees

Natural observations show that chimpanzees occasionally act on behalf of others. Interestingly, the beneficiaries of these helping acts are not just the helper's relatives, as is mostly the case among other nonhuman primates, but also nonrelatives (Langergraber et al. 2007). Several anecdotal

reports have shown that chimpanzees occasionally assist others, as is the case of the chimpanzee who rescued another nonrelated female from drowning in the moat of their enclosure (Fouts and Mills 1997) or the reported cases of adoptions by chimpanzee males (Boesch et al. 2010). In addition, there is evidence that chimpanzees regularly engage in behaviors such as food sharing, coalitionary support, and consolation (e.g., Boesch and Boesch-Achermann 2000; de Waal and Aureli 1996; Muller and Mitani 2005). However, the difficulty with many of these observations is that it is not always possible to rule out a selfish motivation behind actors' actions, since often these behaviors also lead to immediate benefits for the actors. For example, food-sharing behavior has also been explained by the "harassment" or "sharing-under-pressure" hypothesis (Gilby 2006). According to this hypothesis, chimpanzees share meat after group hunts not out of a concern for the welfare of others and to fulfill the others' needs and desires, but they share under duress. That is, beggars' insistent behavior interferes with the possessor's ability to feed and/or increases the risk that the possessor will get injured if beggars become increasingly aggressive. Therefore, individuals are better off if they give a piece to the beggars and can then continue feeding alone.

Experimental studies that present chimpanzees with situations in which subjects can potentially share resources and help others – at the same time that helpers' selfish gains can be ruled out – are starting to reveal chimpanzees' ability and propensity to act on behalf of others, as well as the circumstances under which these tendencies are expressed.

Food Sharing in Chimpanzees

Food sharing is an important aspect in the social life of chimpanzees. It occurs regularly and more often than among other nonhuman primate species (with the exception of the Callitrichidae, one family of New World monkeys). Characteristic of chimpanzee food sharing (in particular meat sharing after hunting monkeys or other mammals) is that it occurs not only among mother-offspring

dyads but also between unrelated individuals of different age and sex classes. Typically, chimpanzee food sharing is mostly "passive": food possessors tolerate beggars taking food from them. In general, very few instances of active sharing are observed, not even among mother-offspring pairs (Boesch and Boesch 1989; Ueno and Matsuzawa 2004). This suggests the possibility that food donors share to avoid being harassed by the beggars (Gilby 2006) and are not truly motivated to share food with others.

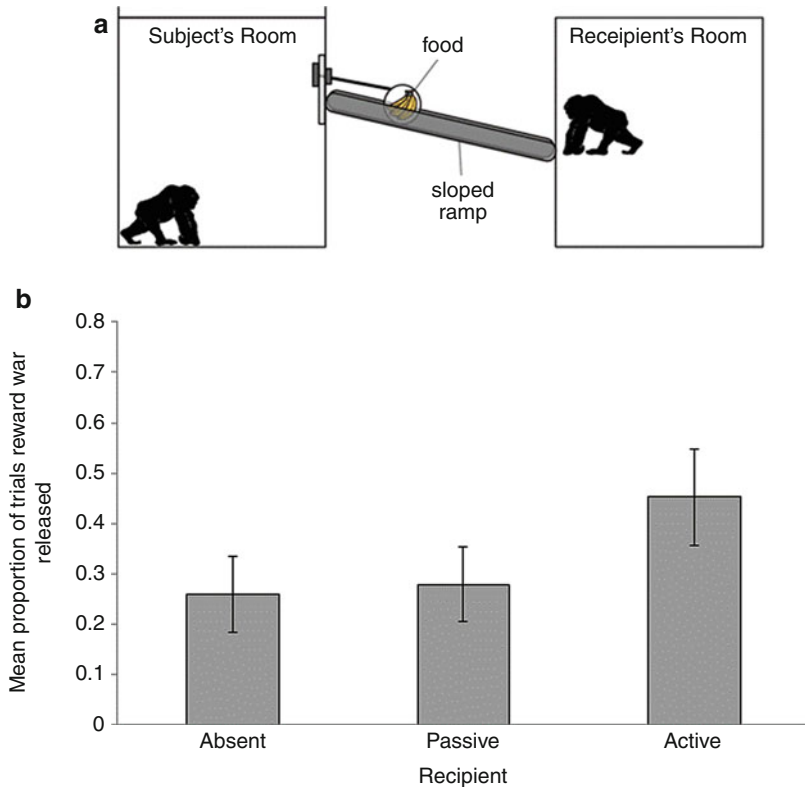
Several experimental studies investigating chimpanzees' propensity to provide food rewards to conspecifics have come to the same conclusion. The main method employed in these studies was to let chimpanzees choose between an option that would deliver a food piece to themselves and another food piece to a conspecific (mutualistic option) or an option that would deliver food only to themselves (selfish option). In all these studies chimpanzees chose randomly between the two options, even if the mutualistic choice would come at no cost to themselves. As chimpanzees did not deliver food to their partner, the general conclusion from these studies was that chimpanzees are indifferent to the welfare of their partners (Jensen et al. 2006; Silk et al. 2005; Vonk et al. 2008; Yamamoto and Tanaka 2010).

However, in a recent study chimpanzees did actively provide food to others (Melis et al. 2011). In this experimental setup, rather than choosing between a mutualistic and a selfish outcome, chimpanzees could help a conspecific obtain food that was out of reach by letting it slide down a ramp toward the recipient (see Fig. 1). The potential helper could never obtain the reward herself. Results showed that subjects released the food when the partner was actively communicating his goal by trying to get at the food or getting the helper's attention. This suggests that as long as partners actively signal their goals and/or desires, and the altruistic act does not interfere with the actor's selfish goals, there are situations in which chimpanzees are willing to provide food to others.

Importantly, active provision of food seems to occur only if the opportunity to monopolize

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Fig. 1 Experimental setup and main results of Melis et al. (2011): (a) subjects can help a conspecific by releasing a peg that stops a bag with food from sliding down the ramp; (b) subjects released the bag significantly more often when the recipient communicated his goal (“active”) than when he was passive or was not in the room



resources is removed. These results thus do not challenge the conclusion that our nearest living primate relatives are not particularly inclined to actively share resources with others when they could have it for themselves (e.g., Warneken and Tomasello 2009).

Instrumental Helping in Chimpanzees

A series of recent experimental studies have shown that under certain circumstances chimpanzees act on others' behalf assisting them to achieve their goals. This has been called “instrumental” or “targeted” helping (Warneken and Tomasello 2006). Warneken and Tomasello (2006) found that human-raised chimpanzees helped their human caregiver by handing her objects she was unsuccessfully reaching for even in the absence of an external reward. Moreover, a further study by Warneken et al. 2007, showed that a group of semi-free ranging chimpanzees also helped by handing the out-of-reach object to a human who had not interacted with them before

the experiment. The chimpanzees did so irrespective of being rewarded and when they had to actively climb toward the out-of-reach object (Warneken et al. 2007, Experiment 2). This shows that chimpanzees are willing and able to help human strangers obtain objects, even when helping is made effortful and they receive no immediate benefit for themselves.

However, the most ecologically valid question is whether chimpanzees would help other chimpanzees. This has now been demonstrated in three additional experimental paradigms. First, Warneken et al. (2007), Experiment 3, Melis et al. 2008) created a novel situation in which chimpanzees could help an unrelated conspecific to enter a room with food by releasing a chain that was blocking the door. Chimpanzees released the chain significantly more often when the recipient was unsuccessfully trying to enter the room, than in control conditions in which releasing the chain would either not help the recipient (because he was trying to go somewhere else) or no recipient

was present. Probably, recipients' unsuccessful attempts to open the door provided the subjects with the necessary cues to help them detect the recipients' goal.

In a second paradigm by Yamamoto et al. (2009), chimpanzees altruistically helped a conspecific partner by transferring a tool the other needed to access food. This occurred almost exclusively in situations preceded by recipients' communicative cues, such as reaching for the tool (e.g., arm poking) or getting the potential helper's attention (e.g., clapping hands, looking at helper). Chimpanzees virtually never handed over the tool proactively without such a cue, providing further evidence for the notion that recipient cues are critical to elicit helping.

In a third paradigm (Melis et al. 2011), the subject could release a bag with a reward so that it would slide down a ramp toward a recipient. Chimpanzees released the bag more often when the recipient was actively trying to access the reward (e.g., by pulling a rope attached to the bag) or communicate toward the potential helper than when the recipient remained passive. Thus, the main factor predicting helping was again the communicative cues (intentional or not) of the recipient.

This series of studies show that under certain circumstances, chimpanzees act on behalf of others by helping them to achieve their goals, even if it involves accessing food. However, as in the case of food sharing, they seem to engage in instrumental helping in a (re)active rather than proactive way (Melis et al. 2010; Warneken and Tomasello 2008). That is, in all these cases helping was preceded by the recipients' cues or communicative signals toward their goals. One possible explanation is that they might lack some cognitive skills necessary to infer what others need or desire in the absence of such communicative signals. This hypothesis finds further support in the results of comparable studies with children.

Specifically, when Brownell and colleagues (Brownell et al. 2009) adapted the apparatus designed by Silk et al. (2005) for children, they found that 18-month-old children (like chimpanzees) chose randomly between a purely selfish and a mutualistic option. Interestingly, 25-month-old

children chose the mutualistic option that rewarded both themselves and the recipient, but only in a condition in which the recipient had verbalized her desire for the food. This suggests that in certain situations explicit communication from the recipient is critical to elicit sharing behaviors, and that a child's failure might not be necessarily due to a lack of motivation, but due to the lack of social-cognitive skills.

Further evidence for the role of social-cognitive skills in helping others comes from three other studies. Although 14-month-old children already show spontaneous, unrewarded helping behavior in simple situations such as handing over out-of-reach objects, they do not help in other type of tasks which might be more complex in terms of the goal-structure and type of intervention necessary to help fulfill the other person's goals and needs. However, throughout their second year of life and after their second birthday, children become increasingly skilled at inferring what others want or need and intervene in a wider range of situations (Warneken and Tomasello 2006, 2007; Svetlova et al. 2010). Therefore, chimpanzees, similar to human children younger than two, might be constrained in helping contexts not due to a lack of motivation to act on the others' behalf but due to an inability to infer the others' needs and desires in the absence of very salient cues.

Collaborative Interactions

In this entry, we review cooperative behavior in which two or more individuals coordinate their behavior to produce outcomes from which all individuals can benefit. Chimpanzees coalitionary support in inter- and intragroup aggressive encounters or cooperative hunts are only some of the examples in which individuals work together to achieve something that is mutually beneficial (Muller and Mitani 2005; Boesch and Boesch 1989). Thus, the motivational basis of collaboration can be totally selfish: agents should be willing to collaborate if the only possibility to achieve an otherwise unobtainable goal is to work together. However, from a cognitive perspective, the question arises to what extent individuals are able to identify the opportunity to combine efforts with a

partner, using different behavioral and communicative means to guarantee successful coordinated actions. Furthermore, in the case of group-level collaboration, temptations for defection arise, and thus cognitive capacities to select trustworthy partners and avoid cheaters are key to maintain collaboration. Thus, differences in the cognitive skills across species might in part explain the differences observed in the flexibility with which species engage in mutualistic collaboration.

Pioneering experiments on chimpanzees found only very limited collaboration skills. Chimpanzees mostly failed to retrieve food rewards when collaboration was required (Crawford 1937; Chalmeau 1994; Povinelli and O'Neill 2000; see Melis et al. 2010 for more details on these initial studies). One interpretation of these results was that cognitive constraints prevented individuals from coordinating actions with the partner (e.g., Povinelli and O'Neill 2000). However, research conducted with other primate species suggested that successful cooperation might not only require a certain level of social cognition, but also tolerance between potential cooperative partners (e.g., Tonkean macaques: Petit et al. 1992; capuchin monkeys: de Waal and Davis 2003). Furthermore, a study by Chalmeau and colleagues showed that the cooperative behavior of their chimpanzees was limited by social constraints on the subordinates (Chalmeau 1994). Specifically, the most dominant individual monopolized the apparatus, thereby preventing others from potentially cooperating. This suggested that in cooperative problem-solving experiments, chimpanzees treated the situation as a competitive one, and that low levels of social tolerance could be affecting chimpanzees' ability to cooperate with others – especially in food retrieval contexts.

Building upon these findings, Melis and colleagues (Melis et al. 2006a) systematically varied tolerance levels by pairing individuals either with a low-tolerance or a high-tolerance partner. Pairs of chimpanzees were presented with an out-of-reach baited tray, which required that they both pull simultaneously (see Fig. 2a). Strikingly, when subjects were paired with partners they could not share food with; they never succeeded in pulling the tray. This was even the case when the food

rewards on the tray were in two separated dishes and thus each individual had a fair chance to obtain food. However, all subjects immediately succeeded when they were paired with a tolerant partner they could share food and co-feed with. However, when food items were clumped in one position and could thus be easily monopolized, chimpanzee cooperation started falling apart. Interestingly, this does not appear to be the case with bonobos, who succeed in the same pulling task equally often whether the rewards are clumped in one location or dispersed (Melis et al. 2006a; Hare et al. 2007). When the same study was conducted with 3-year-old human peers, results showed that peers collaborate and share at very high levels even when rewards are clumped, suggesting that tolerance constraints in children are not as severe as among chimpanzees (Warneken et al. 2011). The results of these studies suggest that we differ from chimpanzees but share with bonobos a more social tolerant nature over food (see also Hare and Kwetuenda 2010).

Taken together, these studies show that under the right circumstances, chimpanzees are able to work together toward mutualistic outcomes. But what level of understanding do chimpanzees bring to the situation? In other words, do individuals intentionally coordinate their actions with those of their partner or is success the by-product of uncoordinated individual efforts? In Melis et al. (2006b) most individuals learned within a few trials to wait for each other in the plank-pull task, delaying their own pulling of the rope until the partner was in position. The task required true synchronization, because otherwise the rope slipped out of the apparatus. In addition, in a more difficult version of the test, individuals could choose to recruit a partner by opening a door for the other to enter the testing area. Subjects recruited the partner significantly more often when the task required cooperation than when the task could be solved individually, showing an understanding of the role of their partner to succeed. Furthermore, when given the choice between two different collaborative partners, they preferentially recruited the more skillful one (See Fig. 2). Taken together, these results

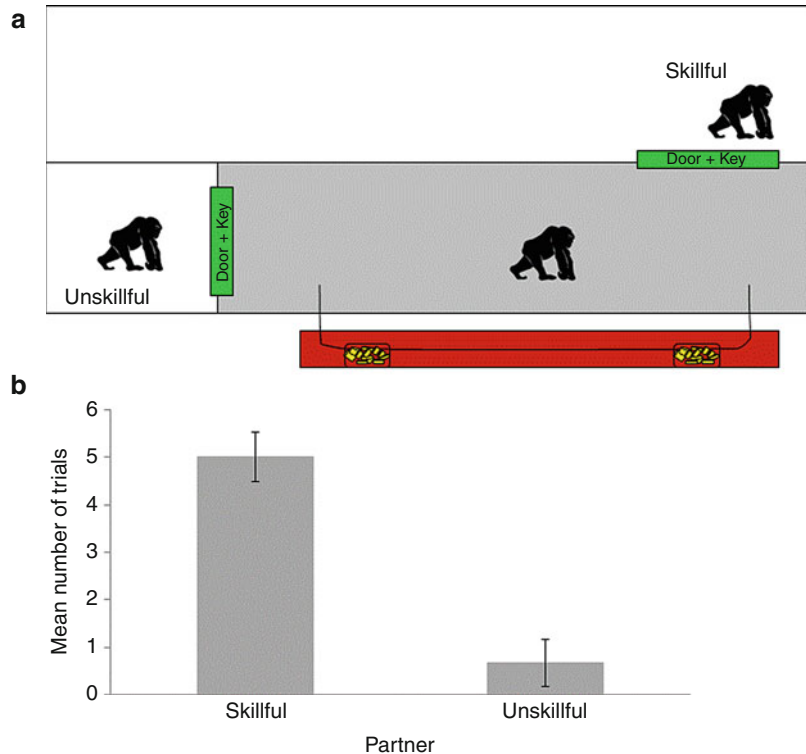
demonstrate that chimpanzee cooperation is not just the by-product of independent individual actions that happen to converge, but that individuals do intentionally coordinate their behavior with that of a partner and choose collaboration as a means to reach goals they cannot achieve individually.

Tomasello et al. (2005) proposed that human collaboration differs fundamentally from chimpanzee collaboration because the former is based upon shared intentions. Shared intentionality is characterized not only by cognitive skills that allow individuals to coordinate actions in pursuit of the shared goal, but by a unique motivation and commitment to collaborate with each other in pursuit of that goal. Studies with children have operationalized this motivation and commitment to collaborate in different ways. For example, if among two collaborating partners one individual stops performing her role, individuals will try to communicatively reengage the partner (Ross and

Lollis 1987; Warneken et al. 2006). Furthermore, children are more likely to reengage the partner if she has previously expressed the commitment to collaborate (“Let’s play together”), which suggests that they treat the other person as an agent with intentions to collaborate or not, and not just as a social tool (Gräfenhain et al. 2009). Second, Hamann et al. (2012) have found that children are not only committed to engage in a collaborative task until one individual is successful, but that there is actually a mutual commitment to ensure that the partner also reaches her goal. In this study pairs of children worked jointly lifting a bar so that each kid could access her own reward. When one of the kids was able to access her reward before the second one could access hers, 3-year-olds (but not 2-year-olds) were more likely to provide support so that the partner could also access hers, and they did this more than in a control condition without prior collaboration. This indicates that 3-year-olds appreciate the commitment for mutual support

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Fig. 2 Experimental setup and results of Experiment 2 of Melis et al. (2006b): (a) subjects were given the choice between recruiting a skillful and an unskillful collaborative partner. Subjects needed a partner to pull within reach the baited tray. (b) Subjects preferentially recruited the skillful partner



that characterizes collaboration. Last but not least, children seem to enjoy the collaborative activity in and of itself since they insist on the social nature of the game, reengaging a social partner even when the other person's involvement is not necessary for the child to succeed with the game (Warneken et al. 2012).

Recent studies with chimpanzees do not seem to suggest that chimpanzees' collaborative interactions are based upon joint intentions. Rather, the data so far is rather consistent with the hypothesis that chimpanzees view their partner as a social tool to reach their own individual goals. Specifically, one study by Warneken et al. (2006) used an "interruption method" with human children and chimpanzees: a human experimenter who had previously worked successfully on a mutualistic task stopped playing her role during certain predetermined interruption periods. Chimpanzees consistently attempted to solve the task by their own means or disengaged completely from the task, and never produced any communicative signals to influence their partner's behavior. However, in the same study children regularly employed different communicative means to reengage the partner. An experiment with conspecific dyads provided similar results, as subjects did not communicate with a recalcitrant conspecific partner (Hirata and Fuwa 2007).

Second, Greenberg and colleagues, using a method similar to Hamann et al. (2012), investigated whether chimpanzees appreciate the commitment for mutual support that characterizes collaboration (Greenberg et al. 2010). This study investigated whether partners in a collaborative activity are mutually committed to ensure that both partners succeed in obtaining their goal. Specifically, in a variation of the plank-pull task, two chimpanzees had to pull together to access food rewards. In a collaborative condition, both chimpanzees pulled a board with two rewards, but one "lucky" individual was able to access the reward before the partner could access his. In a non-collaboration condition, one individual could retrieve the reward without prior collaboration ("lucky"), whereas the "unlucky" chimpanzee needed the other's help to pull the board to the

end. Interestingly, results showed that the "lucky" individual helped the "unlucky" individual in approximately a third of trials by jointly pulling the board with her toward the end, even though she had already accessed hers and she would not receive any further reward for pulling to the end. However, this help was provided equally often in both the collaborative and the non-collaborative condition. Thus, chimpanzees were willing to help the other chimpanzee, but in contrast to human children, collaboration did not seem to enhance helping.

Last but not least, even if chimpanzees are willing and able to join efforts when the situation requires it, they do not seem particularly motivated to act together with a partner unless this is the only way to access their goal. In another study which also used the plank-pull task chimpanzees were given the choice between working either individually or collaboratively with a tolerant partner (with both options resulting in equal payoffs for the subjects). Chimpanzees preferably chose the nonsocial option and only shifted their preference to the social-collaborative option when the payoffs of this latter option were higher. This shows that chimpanzees are strategic and cognizant of their collaborative options, but do not seem to be particularly inclined to collaborate with others (Bullinger et al. 2011).

Taken together, the results from these different studies suggest that chimpanzees are able to engage in quite sophisticated mutualistic collaborative activities, including an understanding of when a partner is needed and which partner might best be suited to solve a mutualistic task. However, the current evidence supports the hypothesis that they view their partner as a social tool rather than forming "joint" goals with him and developing joint intentions and a commitment to pursue that goal.

Future Directions

- What enforcing mechanisms sustain altruistic and collaborative behaviors in nonhuman apes?

- To what extent do empathy and sympathy play a role in the observed altruistic behaviors of chimpanzees?
- What are the similarities and differences in the cooperative behaviors of chimpanzees and bonobos as our two closest living primate relatives?

References and Reading

- Boesch, C., & Boesch, H. (1989). Hunting behavior of wild chimpanzees in the Tai National Park. *American Journal of Physical Anthropology*, 78, 547–573.
- Boesch, C., & Boesch-Achermann, H. (2000). *The chimpanzees of the Tai Forest*. Oxford: Oxford University Press.
- Boesch, C., Bolé, C., Eckhardt, N., & Boesch, H. (2010). Altruism in forest chimpanzees: The case of adoption. *PLoS One*, 5, e8901. <https://doi.org/10.1371/journal.pone.0008901>.
- Brownell, C., Svetlova, M., & Nichols, S. (2009). To share or not to share: When do toddlers respond to another's needs? *Infancy*, 14, 117–130.
- Bullinger, A. F., Melis, A. P., & Tomasello, M. (2011). Chimpanzees (*Pan troglodytes*) prefer individual over cooperative strategies toward goals. *Animal Behaviour*, 82, 1135–1141.
- Chalmeau, R. (1994). Do chimpanzees cooperate in a learning task? *Primates*, 35(3), 385–392.
- Crawford, M. P. (1937). The cooperative solving of problems by young chimpanzees. *Comparative Psychology Monographs*, 14, 1–88.
- de Waal, F. B. M., & Aureli, F. (1996). Consolation, reconciliation, and a possible cognitive difference between macaques and chimpanzees. In A. E. Russon, K. A. Bard, & S. T. Parker (Eds.), *Reaching into thought: The minds of the great apes* (pp. 80–110). Cambridge: Cambridge University Press.
- de Waal, F. B. M., & Davis, J. M. (2003). Capuchin cognitive ecology: Cooperation based on projected returns. *Neuropsychologia*, 41, 221–228.
- Fouts, R. S., & Mills, S. T. (1997). *Next of Kin: My conversations with chimpanzees*. New York: William Morrow.
- Gilby, I. C. (2006). Meat sharing among the Gombe chimpanzees: Harassment and reciprocal exchange. *Animal Behaviour*, 71, 953–963.
- Gräfenhain, M., Behne, T., Carpenter, M., & Tomasello, M. (2009). Young children's understanding of joint commitments. *Developmental Psychology*, 45, 1430–1443.
- Greenberg, J., Hamann, K., Warneken, F., & Tomasello, M. (2010). Chimpanzee helping in collaborative and non-collaborative contexts. *Animal Behaviour*, 80, 873–880.
- Hamann, K., Warneken, F., & Tomasello, M. (2012). Children's developing commitments to joint goals. *Child Development*, 83, 137–145.
- Hare, B., & Kwetuenda, S. (2010). Bonobos voluntarily share their own food with others. *Current Biology*, 20, R230–R231.
- Hare, B., Melis, A. P., Woods, V., Hastings, S., & Wrangham, R. (2007). Tolerance allows bonobos to outperform chimpanzees in a cooperative task. *Current Biology*, 17, 619–623.
- Hirata, S., & Fuwa, K. (2007). Chimpanzees (*Pan troglodytes*) learn to act with other individuals in a cooperative task. *Primates*, 48, 13–21.
- Jensen, K., Hare, B., Call, J., & Tomasello, M. (2006). What's in it for me? Self-regard precludes altruism and spite in chimpanzees. *Proceedings of the Royal Society B*, 273, 1013–1021.
- Kappeler, P. M., & van Schaik, C. (2006). *Cooperation in primates and humans: Mechanisms and evolution*. New York: Springer.
- Langergraber, K. E., Mitani, J. C. C., & Vigilant, L. (2007). The limited impact of kinship on cooperation in wild chimpanzees. *Proceedings of the National Academy of Sciences of the United States of America*, 104, 7786–7790.
- Melis, A. P., & Semmann, D. (2010). How is human cooperation different? *Philosophical Transactions of the Royal Society B*, 365, 2663–2674.
- Melis, A. P., Hare, B., & Tomasello, M. (2006a). Engineering cooperation in chimpanzees. *Animal Behaviour*, 72, 275–286.
- Melis, A. P., Hare, B., & Tomasello, M. (2006b). Chimpanzees recruit the best collaborators. *Science*, 311, 1297–1300.
- Melis, A. P., Hare, B., & Tomasello, M. (2008). Do chimpanzees reciprocate received favors? *Animal Behaviour*, 76, 951–962.
- Melis, A. P., Warneken, F., & Hare, B. (2010). Collaboration and helping in chimpanzees. In E. V. Lonsdorf, S. R. Ross, & T. Matsuzawa (Eds.), *The mind of the chimpanzee: Ecological and experimental perspectives* (pp. 265–282). Chicago: University of Chicago Press.
- Melis, A. P., Warneken, F., Jensen, K., Schneider, A.-C., Call, J., & Tomasello, M. (2011). Chimpanzees help conspecifics obtain food and non-food items. *Proceedings of the Royal Society B*. <https://doi.org/10.1098/rspb.2010.1735>.
- Muller, M., & Mitani, J. C. (2005). Conflict and cooperation in wild chimpanzees. *Advances in the Study of Behavior*, 35, 275–331.
- Petit, O., Desportes, C., & Thierry, B. (1992). Differential probability of “coproduction” in two species of macaque (*Macaca tonkeana*, *M. mulatta*). *Ethology*, 90, 107–120.
- Povinelli, D. J., & O'Neill, D. K. (2000). Do chimpanzees use their gestures to instruct each other? In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds.), *Understanding other minds: Perspectives from developmental cognitive neuroscience* (2nd ed., pp. 459–487). Oxford: Oxford University Press.
- Ross, H. S., & Lollis, S. P. (1987). Communication within infant social games. *Developmental Psychology*, 23, 241–248.

- Silk, J., Brosnan, S., Vonk, J., Henrich, J., Povinelli, D., Richardson, A. S., et al. (2005). Chimpanzees are indifferent to the welfare of unrelated group members. *Nature*, 437, 1357–1359.
- Svetlova, M., Nichols, S., & Brownell, C. (2010). Toddlers' prosocial behavior: From instrumental to empathic to altruistic helping. *Child Development*, 81(6), 1814–1827.
- Tomasello, M., Carpenter, M., Call, J., Behne, T., & Moll, H. (2005). Understanding and sharing intentions: The ontogeny and phylogeny of cultural cognition. *Behavioral & Brain Sciences*, 28(5), 675–691.
- Ueno, A., & Matsuzawa, T. (2004). Food transfer between chimpanzee mothers and their infants. *Primates*, 45, 231–239.
- Vonk, J., Brosnan, S. F., Silk, J. B., Henrich, J., Richardson, A. S., Lambeth, S. P., et al. (2008). Chimpanzees do not take advantage of very low cost opportunities to deliver food to unrelated group members. *Animal Behaviour*, 75, 1757–1770.
- Warneken, F., & Melis, A. P. (2012). The ontogeny and phylogeny of cooperation. In J. Vonk & T. K. Shackelford (Eds.), *The Oxford handbook of comparative evolutionary psychology*. New York: Oxford University Press.
- Warneken, F., & Tomasello, M. (2006). Altruistic helping in human infants and young chimpanzees. *Science*, 311, 1301–1303.
- Warneken, F., & Tomasello, M. (2007). Helping and cooperation at 14 months of age. *Infancy*, 11, 271–294.
- Warneken, F., & Tomasello, M. (2008). Roots of human altruism in chimpanzees. *Primate eye 96* (Special issue: Abstracts of the XXII Congress of IPS, Edinburgh), 16.
- Warneken, F., & Tomasello, M. (2009). Varieties of altruism in children and chimpanzees. *Trends in Cognitive Sciences*, 13, 397–482.
- Warneken, F., Chen, F., & Tomasello, M. (2006). Cooperative activities in young children and chimpanzees. *Child Development*, 77, 640–663.
- Warneken, F., Hare, B., Melis, A. P., Hanus, D., & Tomasello, M. (2007). Spontaneous altruism by chimpanzees and young children. *PLoS Biology*, 5, 1414–1420.
- Warneken, F., Lohse, K., Melis, A. P., & Tomasello, M. (2011). Young children share the spoils after collaboration. *Psychological Science*, 22, 267–273.
- Warneken, F., Gräfenhain, M., & Tomasello, M. (2012). Collaborative partner or social tool? New evidence for young children's understanding of joint intentions in collaborative activities. *Developmental Science*, 15(1), 54–61.
- Yamamoto, S., & Tanaka, M. (2010). The influence of kin relationship and reciprocal context on chimpanzees' other-regarding preferences. *Animal Behaviour*, 79, 595–602.
- Yamamoto, S., Humle, T., & Tanaka, M. (2009). Chimpanzees help each other upon request. *PLoS One*, 4, e7416. <https://doi.org/10.1371/journal.pone.0007416>.

Primitive Reflexes

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Synonyms

Innate reflexes

Definition

Primitive reflexes are patterns of movement that are evoked by specific sensory input. They are complex, automatic movements controlled by the brainstem that can be seen as early as the 25th week of gestation. The primitive reflexes are considered a normal part of infant movement (Zafeiriou 2004). The primitive reflexes include the Moro (a distinctive startle response), the walking/stepping, the rooting, the sucking, the tonic neck (arm straightening in response to turning of the head), the palmar grasp, the plantar or Babinski (toe curling when the sole of the foot is stimulated), the Galant (sideward flexing of the back toward stimulation), the swimming, and the Babkin (head and facial responses to pressure to both palms) reflexes. During the first year of life, the reflexes typically disappear or are more difficult to elicit and their persistence or appearance after infancy is thought to be due to reduced inhibition of brainstem reflexes by the cortex and subcortical regions. Primitive reflexes in older children and adults have been associated with severe neurological problems including

cerebral palsy, acquired brain damage, and stroke (Go and Konishi 2008).

Primitive reflexes, including the snout, sucking, grasp, and rooting reflexes, have been studied in children and young adults with autism (Minderaa et al. 1985; De Bildt et al. 2012). Two thirds of the individuals with autism and none of the control subjects displayed the visual rooting reflex (VRR) to a waving reflex hammer. Neurologists and developmentalists have thoroughly studied the rooting reflexes (Ingram 1962), and they are defined as an orientation toward touch in the area around the mouth region or toward visual stimulation near the face (Schott and Rossor 2003). Although termed a primitive reflex, the VRR has not been described in infants. Rather, most prior reports of the VRR have to do with assessing its presence in adults with various types of neurological dysfunction (Turner and Schon 2006). Further research is needed to characterize more completely the presence and persistence of primitive reflexes in autism and related disorders.

References and Reading

- De Bildt, A., Mulder, E. J., Van Lang, N. D. J., De With, S. A. J., Minderaa, R. M., & Anderson, G. M. (2012). The visual rooting reflex in individuals with autism spectrum disorders and co-occurring intellectual disability. *Autism Research*, 5(1), 67–72.
- Go, T., & Konishi, Y. (2008). Neonatal oral imitation in patients with severe brain damage. *PLoS One*, 3(11), e3668.
- Ingram, T. T. S. (1962). Clinical significance of the infantile feeding reflexes. *Developmental Medicine & Child Neurology*, 4, 159–169.
- Minderaa, R. B., Volkmar, F. R., Hansen, C. R., Harcherik, D. F., Akkerhuis, G. W., & Cohen, D. J. (1985). Brief report: Snout and visual rooting reflexes in infantile autism. *Journal of Autism and Developmental Disorders*, 15(4), 409–416.
- Schott, J. M., & Rossor, M. N. (2003). The grasp and other primitive reflexes. *Journal of Neurology, Neurosurgery & Psychiatry*, 74(5), 558–560.
- Turner, C., & Schon, F. (2006). Visually-evoked rooting, a fascinating primitive reflex. *Practical Neurology*, 6, 358–359.
- Zafeiriou, D. I. (2004). Primitive reflexes and postural reactions in the neurodevelopmental examination. *Pediatric Neurology*, 31(1), 1–8.

Princeton Child Development Institute (PCDI)

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Definition

The Princeton Child Development Institute (PCDI) is a research-based intervention program for persons with autism of all ages. Located in Princeton, New Jersey, the PCDI is a private, nonprofit organization which collaborates with the New Jersey Department of Education and the Division of Developmental Disabilities to provide a variety of programs, including early intervention, accredited preschool- and school-aged programs, residential services, and adult skill-building programs. Training, dissemination, outreach, and intervention science are integrated within the professional activities of PCDI. Faculty of the institute maintain academic affiliations with Queens College of the City University of New York, the University of Kansas, and the University of North Texas, enabling PCDI to serve as a training site for professionals in the field of developmental disabilities and intervention science.

Historical Background

The PCDI was founded in 1970 by Peggy W. Pulleyn and Pamela Machold. Both women were family members of a young boy with autism and were frustrated by the lack of evidence-based intervention available in their community. Committed to providing educational programs that were based in applied behavior analysis, the institute established the first community-based program

to serve children with autism in the state of New Jersey. In 1975, two leaders in the field of applied behavior analysis – Patricia J. Krantz, Ph.D. and Lynn E. McClannahan, Ph.D. – became codirectors of the Institute and worked to expand its mission to include parent support and education, home-based intervention, and unique residential programs with a “family-like” atmosphere for young people with autism. Under the current leadership of Edward C. Fenske, Ed.S. and Gregory MacDuff, Ph.D., programs based on the PCDI model are growing both nationally and internationally.

Rationale or Underlying Theory

The PCDI provides interventions that are based on the principles of applied behavior analysis (ABA). Data-based instruction, systematic teaching approaches, opportunities for frequent practice of new skills, and active engagement of the learner in highly motivating learning opportunities are the cornerstones of this educational approach. Teaching is dynamic, with close monitoring of the learner’s behaviors so that quick adaptation of teaching techniques, settings, prompts, and reinforcement schedules encourages continual educational progress. A family-focused approach to care is foundational to practice and outreach efforts are directed towards community awareness and integration for persons with autism.

Goals and Objectives

The mission statement of the PCDI (as stated in their website at <http://www.pcdi.org/AboutUs/index.asp>, accessed February 21, 2011) is: “The mission of the Princeton Child Development Institute is to provide effective science-based intervention for children and adults with autism and, through research and dissemination, to extend treatment resources to people with autism, both nationally and internationally.”

Treatment Participants

Individuals with autism across the age range (and their family members) participate in services

provided by the PCDI. Toddlers (i.e., children younger than 2 years of age) can access home-based and/or center-based services. Preschool-aged children and those in elementary and secondary schools receive educational programming through a collaboration with the New Jersey Department of Education. Adults can participate in supported employment and life skills educational programs provided by PCDI and funded by the New Jersey Division of Developmental Disabilities. PCDI currently operates the Family Focus Residential Programs, which provide family-style group home supports to persons with autism of all ages. Parent and Sibling Support Programs are also offered to participating families.

Treatment Procedures

Interventions being implemented and studied at the PCDI are based in applied behavior analysis. Publications by the lead faculty are focused upon developing positive behavioral routines, providing visual and physical structure to encourage independence and success, systematically fading supports to promote mastery, and actively planning for generalization of skills across settings, tasks, and people. Positive behavior supports, direct instruction, naturalistic instruction, and data-based decision-making are all practiced. Programming is individualized to fit the needs of a particular student and family.

Efficacy Information

Several interventions practiced at PCDI are listed as meeting the criteria for evidence-based practice by the National Professional Development Center on Autism Spectrum Disorders, which is funded by the Department of Education to investigate and describe evidence-based procedures (see <http://autismpdc.fpg.unc.edu>).

The PCDI has been recognized for significant contributions to the field by the Journal of Applied Behavior Analysis, Senate of the State of New Jersey (1988, for commendable service to persons with autism), National Teaching-Family

Association (1989, Outstanding Contributions in Education), Division 25 of the American Psychological Association (Fred S. Keller Award for Distinguished Contributions to Behavioral Education, 1992), and the Association for Behavior Analysis (1999, Award for Enduring Programmatic Contributions in Behavior Analysis).

Qualifications of Treatment Providers

Treatment providers are well trained in applied behavior analysis and hold degrees in education, speech and communication sciences, and the behavioral sciences. Faculty are affiliated with several universities, and training opportunities are available for qualified professionals.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Visual Supports](#)

References and Reading

- Birkan, B., Krantz, P. J., & McClannahan, L. E. (2011). Teaching children with autism spectrum disorders to cooperate with injections. *Research in Autism Spectrum Disorders*, 5(2), 941–948.
- Buffington, D. M., Krantz, P. J., McClannahan, L. E., & Poulson, C. L. (1998). Procedures for teaching appropriate gestural communication skills to children with autism. *Journal of Autism and Developmental Disorders*, 28(6), 535–545.
- Fenske, E. C., Zalski, S., Krantz, P. J., & McClannahan, L. E. (1985). Age at intervention and treatment outcome in autistic children in a comprehensive intervention program. *Analysis and Intervention in Developmental Disabilities*, 5(1–2), 49–58.
- Fenske, E. C., Krantz, P. J., & McClannahan, L. E. (2001). Incidental teaching: A-not-discrete-trial teaching procedure. In C. Maurice, G. Green, & R. M. Simpson (Eds.), *Making a difference: Behavioral intervention for autism* (pp. 75–82). Austin: Pro-Ed.
- Gena, A., Krantz, P. J., McClannahan, L. E., & Poulson, C. L. (1996). Training and generalization of affective behaviors displayed by youth with autism. *Journal of Applied Behavior Analysis*, 29(3), 291–304.
- Hall, L. J., McClannahan, L. E., & Krantz, P. J. (1995). Promoting independence in integrated teaching aides to use activity schedules and decreased prompts. *Education and Training in Mental Retardation and Developmental Disabilities*, 30(3), 208–217.
- Krantz, P. J. (2000). Commentary: Interventions to facilitate socialization. *Journal of Autism and Developmental Disorders*, 30(5), 411–413.
- Krantz, P. J., & McClannahan, L. E. (1993). Teaching children with autism to initiate to peers: Effects of a script-fading procedure. *Journal of Applied Behavior Analysis*, 26(1), 121–132.
- Krantz, P. J., & McClannahan, L. E. (1994). *Preschool education programs for children with autism*. Austin: Pro-Ed.
- Krantz, P. J., & McClannahan, L. E. (1998). Social interaction skills for children with autism: A script-fading procedure for beginning readers. *Journal of Applied Behavior Analysis*, 31(2), 191–202.
- Krantz, P. J., Zalski, S., Hall, C. J., Fenske, E. C., & McClannahan, L. E. (1981). Teaching complex language to autistic children. *Analysis and Intervention in Developmental Disabilities*, 1(3–4), 259–297.
- Krantz, P. J., MacDuff, G. S., Wadstrom, O., McClannahan, L. E., & Dowrick, P. W. (1991). *Using video with developmentally disabled learners* (Practical guide to video in the behavioral sciences, pp. 256–266). Oxford, UK: Wiley.
- Krantz, P. J., MacDuff, G. S., & McClannahan, L. E. (1993). Programming participation in family activities in children with autism: Parents' use of photo activity schedules. *Journal of Applied Behavior Analysis*, 26(1), 137–138.
- MacDuff, G. S., Krantz, P. J., MacDuff, M. A., & McClannahan, L. E. (1988). Providing incidental teaching for autistic children: A rapid training procedure for therapists. *Education and Treatment of Children*, 11(3), 205–217.
- MacDuff, G. S., Ledo, R., McClannahan, L. E., & Krantz, P. J. (2007). Using scripts and script-fading procedures to promote bids for joint attention by young children with autism. *Research in Autism Spectrum Disorders*, 1(4), 281–290.
- McClannahan, L. E., & Krantz, P. J. (1993a). Some next steps in rights protection for the developmentally disabled. *School Psychology Review*, 14(2), 143–149.
- McClannahan, L. E., & Krantz, P. J. (1993b). On systems analysis in autism intervention programs. *Journal of Applied Behavior Analysis*, 26(4), 589–596.
- McClannahan, L. E., & Krantz, P. J. (1997a). Princeton Child Development Institute. *Behavior and Social Issues*, 7(1), 65–68.
- McClannahan, L. E., & Krantz, P. J. (1997b). In search of solutions to prompt dependence: Teaching children with autism to use photo activity schedules. In D. Baer & E. M. Pinkston (Eds.), *Environment and behavior* (pp. 271–278). Boulder: Westview Press.
- McClannahan, L. E., & Krantz, P. J. (2010). *Activity schedules for children with autism: Teaching independent behavior* (2nd ed.). Bethesda: Woodbine House. 147 pp.
- McClannahan, L. E., Krantz, P. J., & McGee, G. G. (1982). Parents as therapists for autistic children: A model for effective parent training. *Analysis and Intervention in Developmental Disabilities*, 2(2–3), 223–252.
- McClannahan, L. E., MacDuff, G. S., & Krantz, P. J. (2002). Behavior analysis and intervention for adults with autism. *Behavior Modification*, 26(1), 9–26.

- McClannahan, L. E., Krantz, P. J., Briggs, H. E., & Rzepnicki, T. L. (2004). *Using evidence in social work practice*. Chicago: Lyceum Press.
- McGee, G. G., Krantz, P. J., Mason, D., & McClannahan, L. E. (1983). A modified incidental teaching procedure for autistic youth: Acquisition and generalization of receptive labels. *Journal of Applied Behavior Analysis*, 16(3), 329–338.
- McGee, G. G., Krantz, P. J., & McClannahan, L. E. (1986). An extension of incidental teaching procedures to reading instruction for autistic children. *Journal of Applied Behavior Analysis*, 19(2), 147–157.
- van der Gaag, J. & Jee-Peng, T. (1998). *The benefits of early child development programs: An economic analysis*. Washington, DC: The World Bank. <http://www.siteresources.worldbank.org>
- Website: <http://www.pcdi.org/aboutUs/index.asp>
- Young, J. M., Krantz, P. J., McClannahan, L. E., & Poulson, C. L. (1994). Generalized imitation and response-class formation in children with autism. *Journal of Applied Behavior Analysis*, 27(4), 685–697.

Principal Component Analysis (PCA)

- [Glasgow Sensory Questionnaire \(GSQ\)](#)

Private Speech

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Synonyms

[Self-talk](#); [Speech-for-the-self](#)

Definition

Private speech refers to a group of vocalized utterances, typically seen in children from 2 to 7 years old, used as a form of self-regulation. Eventually, these utterances, which are intended for oneself and not others, are internalized and become inner speech. Children with autism use private speech as

a tool for behavioral and cognitive self-regulation as well as thinking and self-organization.

See Also

- [Expressive Language](#)
- [Information Processing Speed](#)
- [Verbal Communication](#)

References and Reading

- Lee, J. (2008). Gesture and private speech in second language acquisition. *Studies in Second Language Acquisition*, 30(02), 169–190.
- Lidstone, J., Meins, E., & Fernyhough, C. (2010). The roles of private speech and inner speech in planning during middle childhood: Evidence from a dual task paradigm. *Journal of Experimental Child Psychology*, 107(4), 438–451.
- Winsler, A., Abar, B., Feder, M., Schunn, C., & Rubio, D. (2007). Private speech and executive functioning among high-functioning children with autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(9), 1617–1635.

Proactive Inhibition

- [Proactive Interference in ASD](#)

Proactive Interference in ASD

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Synonyms

[Proactive inhibition](#)

Definition

Proactive Interference is a well-known and classic phenomenon in the study of human memory and learning. The effects of both prior or subsequent learning observed in the retention of new material not only challenged previous theories and explanations of how one forgets (e.g., decay theory) but turned out to be generally accepted as major determinants of forgetting (Baddeley 1990). Proactive interference refers to the fact that learning which occurs prior to a subsequent event affects the ability to recall it, by decreasing the retention rates. Classical experimental demonstrations can be found in the work of Underwood and colleagues (e.g., Keppel and Underwood 1962), both in short- and long-term memory, where the greater the amount of lists of words (or single words) that are previously learnt by the participants, the greater the forgetting. Some variables are known to modulate this effect, that is, variables that have the ability to reduce or boost forgetting. One of these demonstrations comes from Wickens and colleagues' work (e.g., Wickens 1970). In this experimental study, participants were presented with a triad of words of the same category, over several trials. Of interest is the fact that, besides the expected decrease of recall over trials of the same semantic category (e.g., food words) (build up effect), when the category of belonging of the word triads changed (e.g., to house-part words) there was an observable increase in recall reaching the initial levels of retention (release effect).

Drawing an analogy between, on the one hand, the pattern of preserved and intact functions within the domain of memory that is commonly found in amnesic patients and, on the other hand, the characterization of memory functioning in individuals with ASD, Boucher and Warrington (1976) put forward the hypothesis that autism could be described as an amnesic disorder. Since then, a great deal of research has been carried out regarding mnesic processes in ASD, namely, in the evaluation of proactive interference effects. In a set of two experiments, Carmo and colleagues (Carmo et al. 2016) replicated the paradigm used by Wickens (1970) with a group of high-functioning individuals with ASD to directly test

the presence of proactive interference. These authors found a robust and consistent proactive interference build up effect, with a steady decrease in the amount of recall over trials, similar to that of matched control participants. The release effect when shifting between semantic categories was also present, and thus, proactive interference seems intact in this population and cannot account for the overall reduction in recall rates described by some authors.

See Also

- [Auditory Verbal Learning](#)
- [Episodic Memory](#)
- [High-Functioning Autism \(HFA\)](#)
- [Memory](#)
- [Memory Assessment](#)
- [Semantic Memory](#)

References and Reading

- Baddeley, A. (1990). *Human memory: Theory and practice*. London, UK: Lawrence Erlbaum Associates, Publishers.
- Boucher, J., & Warrington, E. K. (1976). Memory deficits in early infantile autism: Some similarities to the amnesic syndrome. *British Journal of Psychology*, 67(1), 73–87.
- Carmo, J. C., Duarte, E., Pinho, S., Filipe, C. N., & Marques, J. F. (2016). Proactive interference in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(1), 53–63.
- Keppel, G., & Underwood, B. J. (1962). Proactive inhibition in short-term retention of single items. *Journal of Verbal Learning and Verbal Behavior*, 1, 153–161.
- Wickens, D. D. (1970). Encoding categories of words: An empirical approach to meaning. *Psychological Review*, 77(1), 1–15.

Problem Behavior

- [Conduct Disorder](#)

Problem Behaviors

- [Maladaptive Behavior](#)

Problem Sexual Behavior

► [Sexuality and Problem Behaviors](#)

Procedural Adherence

► [Procedural Fidelity](#)

Procedural Fidelity

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Synonyms

[Fidelity of implementation](#); [Independent variable measurement](#); [Procedural adherence](#); [Procedural integrity](#); [Procedural reliability](#)

Definition

Specific term for monitoring, collecting, and documenting data on the independent variable so that it results in a measure of treatment integrity. It provides individuals with information about the conditions that need to be met for the treatment to produce reliable effects. Typically, procedures are provided in written format, such as a manual. It is critical that procedures are assessed for procedural fidelity for consistency and standardization in replication as well as reducing the possibility that a third variable is responsible for the findings. Furthermore, procedural fidelity data can provide practitioners with useful information regarding implementation of research-based interventions.

See Also

► [Treatment Fidelity](#)

References and Reading

- Symes, M. D., Remington, B., Brown, T., & Hastings, R. P. (2006). Early intensive behavioral intervention for children with autism: Therapists' perspectives on achieving procedural fidelity. *Research in Developmental Disabilities, 27*, 30–42.
- Wolery, M. (1994). Procedural fidelity: A reminder of its functions. *Journal of Behavioral Education, 4*(4), 381–386.

Procedural Integrity

► [Procedural Fidelity](#)

Procedural Memory

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Definition

Procedural learning refers to the process by which skills are acquired implicitly (without conscious recall) through repeated exposure to and practice of a task (Squire 1986).

Historical Background

The discovery of the disassociation between memory that could be expressed verbally and accessed consciously (i.e., declarative memory) and experience-based brain adaptation that occurred implicitly (i.e., procedural memory) developed principally from the study of amnesic patients in the 1970s (Squire 1986). As discussed in Current Knowledge, in the intervening decades, a number of paradigms have been developed to

assess various manifestations of procedural learning. The number of studies explicitly investigating procedural learning in autism has increased over the last few years.

Current Knowledge

Introduction

Procedural learning forms the basis for the development of many cognitive and behavioral skills and is understood to result from plasticity in action-oriented brain systems (Gidley Larson and Mostofsky 2006). Motor/premotor circuits in particular have been shown to play a role in the development of many types of skills via procedural learning. Motor skill development is the focus of much procedural learning research, both because motor learning represents the “classic” understanding of procedural learning and because motor responses can be measured reliably. Nevertheless, there is also strong evidence that demonstrates the role of procedural learning in communicative (Ullman 2004) and social skill development (Lieberman 2000).

Autism is characterized by impairments in a number of skilled behaviors necessary for effective social and communicative functioning. Nearly all individuals with autism show substantial delays in adaptive functioning, even in the face of normal general intelligence. Additionally, clinical experience reveals that many children with autism show relative strength in ability to memorize facts and “scripts” (i.e., through separate, declarative learning systems), yet they have difficulty implementing experience-dependent, contextually based (i.e., procedurally based) strategies to deal with social situations and activities of daily living. One potential source of this functional disassociation is a cognitive disassociation between impaired procedural learning and spared (or even superior) declarative learning in autism. The discrepancy between intelligence and adaptive functioning suggests that autism may be specifically characterized by problems with learning “how to” engage in a wide range of skills necessary for social, communicative, and motor functioning, namely, impairments in procedural learning.

Given known skilled motor deficits in autism as well as the fact that much of motor skill development is associated with procedural learning, the study of motor impairment in autism may be critical to understanding the procedural learning basis of the autistic phenotype. Substantial and accumulating evidence demonstrates that a strong preponderance of children with autism display impairments in motor function, with particular impairments in the performance of *skilled* motor gestures (Dziuk et al. 2007; Mostofsky et al. 2006). As autism is a developmental disability, it follows that the impaired performance of skilled motor gestures results from impairments in the procedural learning of those motor skills (Gidley Larson and Mostofsky 2006).

These alterations in the development of motor, social, and communicative skills make the examination of procedural learning a desirable target of research. Several investigators have begun to examine procedural learning in autism. Although a variety of tasks used for the study of procedural learning have failed to show group differences between individuals with autism and controls, approaches that focus specifically on motor learning have shown robust effects. A picture is emerging that demonstrates the role of altered skill learning in the autism phenotype.

Procedural Learning: Phenomenology

The concept of procedural learning refers to brain adaptation that is responsive to repeated exposure to and practice of a task and occurs without the individual’s awareness (Squire 1986). This is sometimes referred to as “learning how,” whereas declarative learning, which consists of the acquisition of knowledge that occurs with awareness and can often be expressed linguistically, is sometimes referred to as “learning what.” When considering the complex relationship between procedural and declarative learning, it is important to consider that “procedures” that can be expressed by the individual as semantic concepts (i.e., “first do this, then do that”) exist in the brain as products of *declarative* learning rather than procedural learning. This consideration is particularly critical when studying tasks that purport to examine procedural

learning, as reliance on declarative mechanisms may produce a significant confusion.

There is a complex literature on declarative learning in autism, some of which suggests normal or even superior skills. Romero-Munguía has proposed the not-yet-validated “mnestic imbalance” theory of autism, in which it is the discrepancy between procedural learning deficits and spared declarative learning skill that produces the core features of autism (Romero-Munguía 2008). Specifically, the theory suggests that core impairments in behavioral flexibility are caused by deficits in the development of automatic actions and in prototype formation, which subsequently leads to impaired social and communicative skills. A preserved or even superior declarative memory allows for preferred activities, such as memorizing encyclopedias. Although there is not much experimental evidence yet to accept or reject this hypothesis, the underlying rationale suggests ways in which altered procedural learning could result in the core features of autism.

Motor Deficits in Autism: A Key to Recognizing Procedural Learning Impairments

Evidence that impairment in procedural learning may be affected in autism comes from the study of the motor system. Motor deficits have been recognized since Kanner’s initial description of autism (Kanner 1943) and have been the focus of increased investigation over the past few decades. While abnormalities of basic motor control have been reported (Jansiewicz et al. 2006; Rinehart et al. 2006), it is impairment in performance of *learned*, complex motor skills which has been most consistently described (Dowell et al. 2009; Dziuk et al. 2007; MacNeil and Mostofsky 2012; Mostofsky et al. 2006). Parents often report that their children with autism showed normal acquisition of early basic motor milestones such as sitting up and walking, but then showed delays in the acquisition of complex motor skills that rely on intact mechanisms of procedural learning. Impaired performance of complex motor skills appears to be specific to autism, even when compared with conditions such as attention deficit/

hyperactivity disorder (ADHD), in which there is known impairment of basic motor control (MacNeil and Mostofsky 2012; Mostofsky et al. 2010).

It is further possible that impaired procedural learning is directly relevant to impaired development of social and communicative skills in autism. On a theoretical level, but one that is informed by experimental data from healthy subjects and a range of clinical groups, it is thought that procedural learning mechanisms are critical for both language development (Ullman 2004) and development of social competency (Lieberman 2000). In research specific to autism, it has been demonstrated that there is a correlation between degree of impairment in complex motor function and degree of impairment in core social and communicative skills (Dziuk et al. 2007). The acquisition of social and communicative skills by procedural learning mechanisms may be further impaired in autism by deficiencies of the systems which are believed to generate the inputs and feedback for the learning of these skills. Specifically, “embodied cognitive science” suggests that social and communicative skills are acquired at least in part through an individual’s ability to implicitly model another’s actions in order to understand intent and emotional state (Klin et al. 2003); these skills are known as theory of mind, which is impaired in autism (Baron-Cohen et al. 1985; Williams 2008). This intention understanding has been linked to the so-called mirror neuron system (MNS) (Williams 2008).

Neurobiology of Procedural Learning

Procedural learning depends on frontal (motor and premotor)-parietal circuits important to establishing sensory-motor representations, as well as input from the basal ganglia and cerebellum, which provide reward-based and error-based feedback, respectively, both of which are critical to skill-based learning (Keele et al. 2003; Shadmehr and Krakauer 2008). There is considerable evidence that frontal-parietal connectivity (and therefore frontal-parietal network function) is decreased in autism (Just et al. 2007; Mostofsky and Ewen 2011). Connections between premotor and posterior parietal cortex are implicated not

only in procedural learning but also in the subsequent expression of previously learned skilled gestures (i.e., praxis) (Geschwind 1965; Wheaton and Hallett 2007) and in the imitation of both meaningful and novel gestures (described as comprising a “mirror neuron system”) (Iacoboni and Mazziotta 2007). The topographical overlap among circuitry involved in procedural learning, praxis, and motor imitation suggests that examination of procedural learning is key to understanding the developmental basis for well-documented autism-associated impairments in imitation and praxis (Dziuk et al. 2007; Mostofsky et al. 2006; Williams 2008). Socialization and communication are largely dependent on both the execution of these skilled actions as well as the ability to interpret these actions when performed by others. Procedural learning may thereby also be important to understanding the processes by which the core features of the autism phenotype develop.

Direct Examination of Procedural Learning in Autism

There have been direct experimental investigations of procedural learning in autism using a range of approaches. The concept of procedural learning encompasses a number of different capabilities, and the cognitive and neurobiological similarities and differences of these capabilities have not been fully resolved. There are experimental paradigms commonly used to examine many aspects of procedural learning, including motor adaption and sequence learning as well as category learning. The SRTT is perhaps the most commonly used experimental task for assessing implicit sequence learning (Robertson 2007). To perform the SRTT, the participant observes a screen that displays a sequence of stimuli and then presses the corresponding buttons in order. Reaction time and error rates are recorded. The stimuli are presented with a pattern imbedded in the order of presentation, so implicit learning of the pattern results in a progressive decrease in reaction time, as well as error rate. One early study of the SRTT in autism (Mostofsky et al. 2000) showed a typical pattern of progressively decreasing response times in control children but a stable pattern of response times in children with

high-functioning autism, suggesting impaired implicit sequence learning in autism. In several training periods that occurred over a 6-week period, Gordon and Stark (2007) demonstrated a decreased learning effect using the SRTT in children with low-functioning autism. On the other hand, there have been a number of studies which have found no difference between children with autism and controls (Barnes et al. 2008; Brown et al. 2010; Muller et al. 2004; Nemeth et al. 2010; Travers et al. 2010). It is likely that methodological concerns are responsible for the differing conclusions. A criticism of the SRTT approach is that it may measure sequence learning rather than motor learning (Robertson 2007) and therefore may not tax the systems that are altered in autism.

Two examinations of a contextual cueing task also showed no group differences (Barnes et al. 2008; Brown et al. 2010). Contextual cueing is a visual search task, where implicit patterns of distractors give a “hint” as to whether the target is present. An artificial grammar task, in which strings of letters with an implicit “grammatical” pattern are memorized, failed to show group differences (Brown et al. 2010). Similarly, a probabilistic classification learning task, where aspects of the stimuli (in this case, the clothing of a cartoon character) had a probabilistic relationship to the correct classification of the character, showed no differences by diagnosis (Brown et al.).

Approaches to Examining Motor Learning in Autism

Perhaps the greatest insights into the association between procedural learning and the autism phenotype have come from examination of motor learning. Recent theoretical perspectives of motor learning specify “internal models” as motor programs that encode discrete motor actions (Iacoboni and Mazziotta 2007; Mostofsky and Ewen 2011; Shadmehr and Krakauer 2008). As described, the formation of internal models of action is critical to learning skilled movements, as well as to learning to interpret others’ movements. Children with autism display impairments in both these domains, with well-documented difficulty in performing skilled actions (i.e., dyspraxia), as well as with recognizing and correctly identifying

those actions when performed by others (Baron-Cohen et al. 1985; Dowell et al. 2009). This suggests that anomalous formation of internal models of action may be important to understanding the patterns of motor and social deficits associated with autism. Consistent with this, children with autism show a strong association between the severity of dyspraxia and the severity of core impairments in social and communicative behavior (Dziuk et al. 2007).

Investigators have begun to examine patterns of action model formation in autism, initially focusing on tasks that test motor adaptation, or the ability to learn a novel pattern of movement based in response to a discrete sensory perturbation. In a particularly revealing study, Haswell et al. (2009) used a motor adaptation task in which participants used a joy stick to move a cursor on a computer screen situated horizontally in front of them so that it obscured the joy stick from their view. In a series of trials, participants used the joy stick to make a reaching motion, extending their arm toward a target. During most trials, a force perpendicular to the participant-controlled direction of movement was applied to the joy stick so that participants had to learn to adapt their arm motion in order to hit the target. After this learning phase of the task, the screen and controller were moved. The relationship of the controller movement to the cursor movement was changed in one of two ways. In the first, the coordinate system relative to the visual input (Cartesian coordinate) was maintained; in the second, the coordinate system relative to proprioceptive input (joint position) was maintained. The findings revealed that, as compared with control subjects, children with autism spectrum disorders showed markedly increased generalization when the proprioceptive coordinate system was maintained and showed decreased generalization (persistence of motor adaptation) when the visual coordinate system was maintained. Furthermore, the strength of increased reliance on proprioceptive input robustly correlated with impairments in imitation, praxis, and social competence, suggesting that alterations of motor adaptation may share a neurobiological basis with the core symptoms of autism (Haswell et al. 2009). Moreover, these

alterations of patterns of motor adaptation generalization were specific to autism and were not seen in children with ADHD (Izawa et al. 2012).

The findings from these studies of motor adaptation suggest that when learning a novel action, children with autism show a bias toward relying on proprioceptive input from their own “internal” world rather than visual input from the external world around them. Indeed, previous investigators had suggested that children with autism respond more effectively to proprioceptive rather than visual input (Masterton and Biederman 1983). Furthermore, these observations are in keeping with recent influential connectivity theories (Horwitz et al. 1988; Mostofsky and Ewen 2011), in which autism is postulated to be associated with an overgrowth of short-range connections (including those between primary somatosensory and motor cortices that encode proprioceptive feedback) and an undergrowth of longer-range connections (including those between inferior parietal and premotor cortices that encode visual feedback).

The correlation between motor learning ability and social/communicative competence (Dziuk et al. 2007; Dowell et al. 2009; Haswell et al. 2009; Izawa et al. 2012), as well as the fact that similar frontal-parietal networks are understood to underlie both types of skill, suggests that the brain systems necessary to acquiring and maintaining motor skills (praxis) may parallel (and perhaps overlap) those systems necessary to acquiring and maintaining social/communicative skills. In this light, autism could be viewed as a “developmental dyspraxia” of social and communicative function (Mostofsky and Ewen 2011), such that anomalous patterns of internal action model formation contribute to both impaired development of motor/social/communicative skills, as well as impaired ability to understand these actions when performed by others.

Summary

In summary, there is a compelling theoretical basis, both neurobiological and cognitive/behavioral, to suggest that impairments of procedural

learning are both present in autism and causally related to the core symptoms. Evidence to date using standard paradigms has, to a large degree, failed to demonstrate these impairments. Novel approaches that target specific known deficits in autism, however, may hold the key to relating alterations of procedural learning to the autism phenotype. This course of investigation could lead to crucial insights into the brain basis of autism and provide a foundation for advancing therapeutic strategies targeting autism-associated difficulties with social, communicative, and motor skills.

Future Directions

Findings generated from studies of procedural learning could lay the foundation for novel approaches for therapeutic interventions targeting improvements in motor, social, and communicative skills. This approach has the potential to provide crucial advances, as current therapeutic techniques (e.g., speech-language therapy, behavioral therapy, educational interventions) often depend on procedural skill learning. One approach under investigation involves “playing to the strength” of children with autism by teaching skills such as handwriting or sign language to children with autism using increased haptic (proprioceptive) input rather than the typical approach that relies on imitating others’ actions (Feder and Majnemer 2007). This approach of increasing proprioceptive input may help children with autism improve specific motor skills; however, it is unlikely to help these children learn social skills given that social interaction involves modeling others’ visually observed behavior. It will therefore be crucial to also explore both behavioral and electrophysiologic methods for enhancing visual-motor connectivity in children with autism.

See Also

- [Apraxia](#)
- [Behavior Therapy](#)
- [Cerebellum](#)

- [Declarative Memory](#)
- [Developmental Coordination Disorder](#)
- [Dysgraphia](#)
- [Dyspraxia](#)
- [Grammar](#)
- [Inferior Parietal Area](#)
- [Language Acquisition](#)
- [Language Interventions](#)
- [Mirror Neuron System](#)
- [Motor Control](#)
- [Motor Planning](#)

References and Reading

- Barnes, K. A., Howard, J. H., Jr., Howard, D. V., Gilotty, L., Kenworthy, L., Gaillard, W. D., et al. (2008). Intact implicit learning of spatial context and temporal sequences in childhood autism spectrum disorder. *Neuropsychology*, 22, 563–570.
- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a “theory of mind”? *Cognition*, 21, 37–46.
- Brown, J., Aczel, B., Jimenez, L., Kaufman, S. B., & Grant, K. P. (2010). Intact implicit learning in autism spectrum conditions. *Quarterly Journal of Experimental Psychology (Colchester)*, 63, 1789–1812.
- Dowell, L. R., Mahone, E. M., & Mostofsky, S. H. (2009). Associations of postural knowledge and basic motor skill with dyspraxia in autism: Implication for abnormalities in distributed connectivity and motor learning. *Neuropsychology*, 23, 563–570.
- Dziuk, M. A., Gidley Larson, J. C., Apostu, A., Mahone, E. M., Denckla, M. B., & Mostofsky, S. H. (2007). Dyspraxia in autism: Association with motor, social, and communicative deficits. *Developmental Medicine and Child Neurology*, 49, 734–739.
- Feder, K. P., & Majnemer, A. (2007). Handwriting development, competency, and intervention. *Developmental Medicine and Child Neurology*, 49, 312–317.
- Geschwind, N. (1965). Disconnection syndromes in animals and man II. *Brain*, 88, 585–644.
- Gidley Larson, J., & Mostofsky, S. (2006). Motor deficits in autism. In R. Tuchman & I. Rapin (Eds.), *Autism: A neurological disorder of early brain development*. London: Mac Keith Press for the International Review of Child Neurology Series.
- Gordon, B., & Stark, S. (2007). Procedural learning of a visual sequence in individuals with autism. *Focus on Autism & Other Developmental Disabilities*, 22, 14–22.
- Haswell, C. C., Izawa, J., Dowell, L. R., Mostofsky, S. H., & Shadmehr, R. (2009). Representation of internal models of action in the autistic brain. *Nature Neuroscience*, 12, 970–972.
- Horwitz, B., Rumsey, J. M., Grady, C. L., & Rapoport, S. I. (1988). The cerebral metabolic landscape in autism.

- Interrelations of regional glucose utilization. *Archives of Neurology*, 45, 749–755.
- Iacoboni, M., & Mazziotta, J. C. (2007). Mirror neuron system: Basic findings and clinical applications. *Annals of Neurology*, 62, 213–218.
- Izawa, J., Pekny, S., Marko, M., Haswell, C., Shadmehr, R., & Mostofsky, S. (2012). Motor learning relies on integrated sensory inputs in ADHD, but over-selectively on proprioception in autism spectrum conditions. *Autism Research*, 5(2), 124–136.
- Jansiewicz, E. M., Goldberg, M. C., Newschaffer, C. J., Denckla, M. B., Landa, R., & Mostofsky, S. H. (2006). Motor signs distinguish children with high functioning autism and Asperger's syndrome from controls. *Journal of Autism and Developmental Disorders*, 36, 613–621.
- Just, M. A., Cherkassky, V. L., Keller, T. A., Kana, R. K., & Minshew, N. J. (2007). Functional and anatomical cortical underconnectivity in autism: Evidence from an fMRI study of an executive function task and corpus callosum morphometry. *Cerebral Cortex*, 17, 951–961.
- Kanner, L. (1943). Autistic disturbances of affective conduct. *The Nervous Child*, 2, 217–250.
- Keele, S. W., Ivry, R., Mayr, U., Hazeltine, E., & Heuer, H. (2003). The cognitive and neural architecture of sequence representation. *Psychological Review*, 110, 316–339.
- Klin, A., Jones, W., Schultz, R., & Volkmar, F. (2003). The enactive mind, or from actions to cognition: Lessons from autism. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 358, 345–360.
- Lieberman, M. D. (2000). Intuition: A social cognitive neuroscience approach. *Psychological Bulletin*, 126, 109–137.
- MacNeil, L. K., & Mostofsky, S. H. (2012). Specificity of praxis impairments in children with autism. *Neuropsychology*, 26(2), 165–171.
- Masterton, B. A., & Biederman, G. B. (1983). Proprioceptive versus visual control in autistic children. *Journal of Autism and Developmental Disorders*, 13, 141–152.
- Mostofsky, S. H., & Ewen, J. B. (2011). Altered connectivity and action model formation in autism is autism. *The Neuroscientist*, 17, 437–448.
- Mostofsky, S. H., Goldberg, M. C., Landa, R. J., & Denckla, M. B. (2000). Evidence for a deficit in procedural learning in children and adolescents with autism: Implications for cerebellar contribution. *Journal of International Neuropsychological Society*, 6, 752–759.
- Mostofsky, S. H., Dubey, P., Jerath, V. K., Jansiewicz, E. M., Goldberg, M. C., & Denckla, M. B. (2006). Developmental dyspraxia is not limited to imitation in children with autism spectrum disorders. *Journal of International Neuropsychological Society*, 12, 314–326.
- Mostofsky, S., Izawa, J., Marko, M., Pekny, S., Dowell, L., & Shadmehr, R. (2010). *Evidence for specificity of anomalous motor learning in autism*. Paper presented at the ninth international meeting for autism research.
- Muller, R. A., Cauch, C., Rubio, M. A., Mizuno, A., & Courchesne, E. (2004). Abnormal activity patterns in premotor cortex during sequence learning in autistic patients. *Biological Psychiatry*, 56, 323–332.
- Nemeth, D., Janacek, K., Balogh, V., Londe, Z., Mingesz, R., Fazekas, M., et al. (2010). Learning in autism: Implicitly superb. *PLoS One*, 5, e11731.
- Rinehart, N. J., Tonge, B. J., Iansek, R., McGinley, J., Brereton, A. V., Enticott, P. G., et al. (2006). Gait function in newly diagnosed children with autism: Cerebellar and basal ganglia related motor disorder. *Developmental Medicine and Child Neurology*, 48, 819–824.
- Robertson, E. M. (2007). The serial reaction time task: Implicit motor skill learning? *Journal of Neuroscience*, 27, 10073–10075.
- Romero-Munguia, M. A. (2008). Mnestic imbalance: A cognitive theory about autism spectrum disorders. *Annals of General Psychiatry*, 7, 20.
- Shadmehr, R., & Krakauer, J. W. (2008). A computational neuroanatomy for motor control. *Experimental Brain Research*, 185, 359–381.
- Squire, L. R. (1986). Mechanisms of memory. *Science*, 232, 1612–1619.
- Travers, B. G., Klinger, M. R., Mussey, J. L., & Klinger, L. G. (2010). Motor-linked implicit learning in persons with autism spectrum disorders. *Autism Research*, 3, 68–77.
- Ullman, M. T. (2004). Contributions of memory circuits to language: The declarative/procedural model. *Cognition*, 92, 231–270.
- Wheaton, L. A., & Hallett, M. (2007). Ideomotor apraxia: A review. *Journal of Neurological Sciences*, 260, 1–10.
- Williams, J. H. (2008). Self-other relations in social development and autism: Multiple roles for mirror neurons and other brain bases. *Autism Research*, 1, 73–90.

Procedural Reliability

► Procedural Fidelity

Procedural Safeguards

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Synonyms

Parent protections

Definition

Procedural Safeguards are protections afforded by the Individuals with Disabilities Education Improvement Act of 2004 (IDEIA) to each student with a disability and to his or her parents to

ensure that the student receives a free and appropriate public education. The Procedural Safeguards ensure that parents (or the student if the student has reached the age of majority within the state) are notified of their rights to and the procedures for disagreeing with the special education process.

The safeguards afforded parents: the rights to examine all of the student's records, to participate in meetings regarding the identification, evaluation, and placement of the student, to consent to evaluations and the provision of special education services, to obtain an independent educational evaluation when needed, and to present and resolve a complaint. The Procedural Safeguards also grant parents the right to written notice within a reasonable time frame when the local education agency (typically the school) wants to evaluate a student, determine whether a student is eligible for special education services, change a student's evaluation, educational placement, or individualized education program (IEP), or refuse a parent's request to evaluate a student or change a student's IEP or placement. While some changes in placement are obvious (for example, a change from a regular education classroom placement to an autism support classroom), the IDEIA specifies certain less common situations that may constitute a change of placement, including removal to an alternate educational setting due to violations of a code of student conduct.

To help educate parents about their rights, schools must provide parents with a procedural safeguards notice at least once a year. Additional notices must be provided whenever a student is referred for an initial evaluation, when a parent requests a new evaluation, at the time a due process complaint is filed, or whenever a parent requests a copy. The notice must detail the process by which a parent may present and resolve complaints related to the special education process, including the option of voluntary mediation. Because states are at liberty to develop ways to implement the IDEIA, the procedural safeguards notices vary from state to state. The actual protections contained within the notice, however, may not confer fewer rights to parents and students than what is provided for by the IDEIA.

See Also

- ▶ Due Process
- ▶ Individuals with Disabilities Education Act (IDEA)

References and Reading

- Individuals with Disabilities Education Improvement Act of 2004, 20 U.S.C. §§ 1400 et seq.
- National Center for Learning Disabilities. (2006). *IDEA parent guide*. Retrieved from <http://www.ncld.org/images/stories/Publications/AdvocacyBriefs/IDEA2004ParentGuide/idea2004parentguide.pdf>
- U.S. Department of Education, Office of Special Education Programs. (2006a). *Procedural safeguards: Mediation*. Retrieved from <http://idea.ed.gov/explore/view/p/%2Croot%2Cdynamic%2CTopicalBrief%2C21%2C>
- U.S. Department of Education, Office of Special Education Programs. (2006b). *Procedural safeguards: Resolution meetings and due process hearings*. Retrieved from <http://idea.ed.gov/explore/view/p/%2Croot%2Cdynamic%2CTopicalBrief%2C16%2C>
- U.S. Department of Education, Office of Special Education Programs. (n.d.). *Procedural safeguards video clip*. Retrieved from <http://idea.ed.gov/explore/view/p/%2Croot%2Cdynamic%2CVideoClips%2C7%2C>

Processing Speed Index

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Synonyms

Processing speed quotient

Definition

Processing speed refers to the speed of cognitive processes and response output. The Processing Speed Index (PSI) is one of four indices that make up the full scale intelligence quotient (FSIQ) derived from The Wechsler Adult Intelligence Scale-4th edition (WAIS-IV) and The Wechsler Intelligence Scale for Children-4th

edition (WISC-IV), primary standardized clinical instruments used to measure intelligence. The tasks included in the scales that comprise the PSI, (Coding, Symbol Search), are timed and require attending to visual material, visual perception and organization, visual scanning, and hand-eye coordination. Performance on the Coding subtest also requires paired associative learning. Anxiety and fine motor problems may interfere with performance on the PSI. Coding, in particular, which requires copying simple shapes, may be affected by motor output problems. This tends to be an area of relative weakness for children with ASD (Calhoun and Mayes 2005).

See Also

- [Attention](#)
- [Processing Speed Quotient](#)
- [Wechsler Preschool and Primary Scale of Intelligence](#)

References and Reading

- Brock, J., Xu, J. Y., & Brooks, K. R. (2011). Individual differences in visual search: Relationship to autistic traits, discrimination thresholds, and speed of processing. *Perception*, 40(6), 739–742.
- Calhoun, S. L., & Mayes, S. D. (2005). Processing speed in children with clinical disorders. *Psychology in the School*, 42(4), 333–343.
- Faja, S., Webb, S. J., Merkle, K., Aylward, E., & Dawson, G. (2008). Brief report: Face configuration accuracy and processing speed among adults with high-functioning Autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 532–538.
- Mayes, S. D., & Calhoun, S. L. (2008). WISC-IV and WIAT-II profiles in children with high-functioning Autism. *Journal of Autism and Developmental Disorders*, 38, 428–439.
- Scheuffgen, K., Happe, F., Anderson, M., & Frith, U. (2000). High “intelligence,” low “IQ” Speed of processing and measured IQ in children with Autism. *Development and Psychopathology*, 12, 83–90.
- Wallace, G. L., Anderson, M., & Happe, F. (2009). Brief report: Information processing speed is intact in Autism but not correlated with measured intelligence. *Journal of Autism and Developmental Disorders*, 39, 809–814.

Processing Speed Quotient

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Synonyms

[Processing speed index](#)

Definition

The Processing Speed Quotient is one of the four indices of the Wechsler Preschool and Primary Scale of Intelligence-Third Edition (WPPSI-III). The other three are Verbal, Performance, and Full Scale Intelligence Quotients. The Processing Speed Quotient measures mental and visual-motor processing speed and accuracy for individuals aged 4 through 7 years. The Processing Speed Quotient is made of two subtests: Symbol Search and Coding and provides an estimate of a child’s ability to quickly and correctly scan, sequence, and discriminate simple visual information. This tends to be an area of relative weakness for children with ASD (Calhoun and Mayes 2005).

See Also

- [Processing Speed Index](#)
- [Wechsler Preschool and Primary Scale of Intelligence](#)

References and Reading

- Brock, J., Xu, J. Y., & Brooks, K. R. (2011). Individual differences in visual search: Relationship to autistic traits, discrimination thresholds, and speed of processing. *Perception*, 40(6), 739–742.
- Calhoun, S. L., & Mayes, S. D. (2005). Processing speed in children with clinical disorders. *Psychology in the School*, 42(4), 333–343.
- Faja, S., Webb, S. J., Merkle, K., Aylward, E., & Dawson, G. (2008). Brief report: Face configuration accuracy and processing speed among adults with high-

- functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 532–538.
- Scheuffgen, K., Happe, F., Anderson, M., & Frith, U. (2000). High “intelligence,” low “IQ”? Speed of processing and measured IQ in children with autism. *Development and Psychopathology*, 12, 83–90.
- Wallace, G. L., Anderson, M., & Happe, F. (2009). Brief report: Information processing speed is intact in autism but not correlated with measured intelligence. *Journal of Autism and Developmental Disorders*, 39, 809–814.

Profile of Intelligences

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Definition

A profile of intelligence is a way of presenting multiple scores earned on a cognitive assessment in relation to one another. The scores may include specific subtests, composite indices, and a full-scale intelligence quotient score (FSIQ). The profile of intelligence also depicts the scatter, or the pattern, of scores on a cognitive assessment.

Historical Background

In the past, psychologists analyzed profiles of intelligence to diagnose individuals with particular learning styles or disabilities. However, these diagnoses were not always clinically or psychometrically sound, as subtest scores are not as reliable as index scores, and the subscales of cognitive assessments often assess intersecting cognitive abilities (Sattler and Ryan 2009). Due in part to concerns about relying on limited data, the American Psychological Association [APA] (2002) stated that psychologists should not make diagnoses based on data from a single test, though they recognize that a profile of intelligence scores can provide practitioners with a great deal of information about an individual’s distribution of cognitive abilities, or relative strengths and weaknesses.

Current Knowledge

Today, psychologists use profiles of intelligence to create hypotheses about an individual’s cognitive strengths and weaknesses, which they can examine further with additional cognitive evaluations and qualitative interviews. Specific assessments often used with individuals with Autism Spectrum Disorders (ASD) include the Wechsler Preschool and Primary Scale of Intelligence – Third Edition (WPPSI-III; Wechsler 2002) for children 2 years 6 months to 7 years 3 months old, the Wechsler Intelligence Scale for Children-Fourth Edition (WISC-IV; Wechsler 2003) for children 6–16 years old, and the Wechsler Adult Intelligence Scale-Fourth Edition (WAIS-IV; Wechsler 1997) for individuals 16–90 years old. The Mullen Scales of Early Learning (Mullen 1995) are also used with very young children.

The WPPSI-III consists of seven core subtests, a composite of which creates the FSIQ. The seven core subtests are Information (test of ability to retain and retrieve knowledge), Vocabulary (test of verbal knowledge), Word Reasoning (test of verbal comprehension and reasoning ability), Block Design (test of visual perception and organization), Matrix Reasoning (test of visual information processing), Picture Concepts (test of abstract reasoning ability), and Coding (test of visual perception and short-term memory). The Verbal IQ score (VIQ) is the composite of Information, Vocabulary, and Word Reasoning scores. The Performance IQ score (PIQ) is the composite of Block Design, Matrix Reasoning, and Picture Concepts. The Processing Speed Quotient (PSQ) is based solely on the Coding score.

The WISC-IV consists of ten core subtests that are used to calculate the VIQ, PIQ, and FSIQ. The ten core subtests are Similarities (test of abstract reasoning), Vocabulary (test of verbal knowledge), Comprehension (test of general principles and social situations), Block Design (test of visual perception and organization), Picture Concepts (test of abstract reasoning ability), Matrix Reasoning (test of visual information processing), Digit Span (test of working memory), Letter-Number Sequencing (test of mental manipulation and short-term memory), Coding (test of visual perception and short-

term memory), and Symbol Search (test of processing and psychomotor speeds and visual short-term memory). Scores on the WISC-IV subtests create four composite index scores: Verbal Comprehension Index (VCI), Perceptual Reasoning Index (PRI), Working Memory Index (WMI), and Processing Speed Index (PSI). VCI is a composite score of Similarities, Vocabulary, and Comprehension; PRI is a composite score of Block Design, Picture Concepts, and Matrix Reasoning; WMI is a composite score of Digit Span and Letter-Number Sequencing; and PSI is a composite score of Symbol Search and Coding.

The WAIS-IV consists of ten core subtests that are used to calculate the VIQ, PIQ, and FSIQ. Nine of the subtests are similar to those previously described in the WISC-IV, but the WAIS-IV also includes Arithmetic (test of working memory and numeric reasoning). Similar to the WISC-IV, scores on the WAIS-IV create four composite index scores: Verbal Comprehension Index (VCI), Perceptual Reasoning Index (PRI), Working Memory Index (WMI), and Processing Speed Index (PSI). VCI is a composite score of Similarities, Vocabulary, Information, and Comprehension; PRI is a composite score of Block Design and Matrix Reasoning; WMI is a composite score of Digit Span and Arithmetic; and PSI is a composite score of Symbol Search and Coding.

Clinical Uses: Practitioners can assess an individual's cognitive strengths and weaknesses by conducting ipsative (i.e., intra-individual) comparisons of an individual's scores or by conducting comparisons to standardized scores (i.e., inter-individual). By taking the difference between index scores, scaled subtest scores, subtests of scaled subtest scores within a broader composite score, and the individual's respective average scores (i.e., ipsative comparisons), practitioners can learn which cognitive skills pose relative difficulty or relative areas of strength for the individual. Considering an individual's scores among those of a standardized group, practitioners can decipher which cognitive skills pose difficulty or areas of strength for this particular individual with respect to the norm group.

By doing thorough profile analyses, and comparing the spread of a person's profile of

intelligence to that of a standardized group, practitioners determine the frequency with which such variability of scores occurs. Knowing how often certain significant differences in index or scaled subtest scores occur in a standardized population, practitioners can begin to create hypotheses about an individual's specific abilities and areas of struggle. For example, if a person earned a PRI score significantly greater than his/her VCI score, practitioners might wonder if the individual's visual-spatial processing and visual-discrimination processing are more developed than his/her verbal processing or auditory-vocal processing. A significant difference between these two index scores could also be a sign of more developed nonverbal problem-solving skills than verbal retrieval of information skills. Although these discrepancies do not establish the presence of a cognitive disability, they do indicate a possible disparity in a person's cognitive abilities and identify cognitive skills that should be further evaluated.

Similarly, significant differences between an individual's scaled subtest scores, especially if they are uncommon in the standardized group, could signify particular cognitive abilities or difficulties. Moreover, comparing scaled scores of two subtests within the same index could reveal important information about the way in which an individual processes information or completes tasks. For example, Vocabulary and Similarities are both subtests of the VCI that measure abstract thinking ability. A significantly greater Vocabulary score than Similarities score might reveal that the individual's knowledge of words is greater than his/her ability to categorize and vice versa if the Similarities score were significantly greater than the Vocabulary score. Again, finding significant differences in scaled subtest scores that are rarely seen within the standardized group is not enough evidence to make declarative statements about a person's cognitive limitations. However, such discrepancies encourage a practitioner to direct attention to these particular cognitive skills, to assess further to determine if a problem exists.

Although it provides little information about variability across subtests, practitioners might

also find it helpful to examine the difference between an individual's highest and lowest scaled subtest scores. A significant range that is rarely found within the standardized group might be an indication of a specific cognitive aptitude or a particular cognitive deficit.

Lastly, analyzing profiles of intelligence in this in-depth manner is essential for determining if the FSIQ is an accurate depiction of a person's overall intelligence. If, in fact, there is a great deal of scatter between scaled subtest scores, then the FSIQ is not interpretable. When examining a profile of intelligence with significant variability, practitioners should administer further cognitive assessments to determine whether the assessment revealed a genuine portrayal of a person's cognitive abilities and to gather information about those cognitive tasks that caused the examinee particular difficulty.

It is critical that practitioners analyze profiles of intelligence after performing cognitive assessments because the pattern of scores provides substantial information about an individual's specific cognitive abilities (e.g., indices, subtests) and areas of struggle; this knowledge allows practitioners to assess more deeply a person's particular areas of, and possible reasons for, difficulty. Practitioners can then synthesize all of the information they have gathered through cognitive assessments with knowledge about the individual that has been collected via medical history and in-person interviews to create personalized suggestions for ways to improve or manage certain cognitive weaknesses. This thorough examination and comprehension of a person's cognitive abilities is essential because, if aid is needed, it allows for the development of individualized plans for support.

Clinical Uses for Individuals with Autism Spectrum Disorders

Practitioners have used profile analysis to determine profiles of intelligence that can be observed among groups of individuals with ASD; researchers have claimed that across the life span, individuals with ASD diagnoses had similar Wechsler Intelligence Scale profiles that were characterized by a lower VIQ than PIQ.

Comprehension was often the lowest subtest score and Digit Span was the highest subtest score on the VIQ composite, while Coding/Digit Symbol was the lowest subtest score and Block Design was the highest subtest score on the PIQ composite (Yirmiya and Sigman 1991). Furthermore, researchers distinguished between individuals with high functioning autism (HFA) and Asperger's Syndrome (AS) using profiles of intelligence: researchers found that individuals with AS had higher VIQ than PIQ, whereas those with HFA were more likely to have a profile similar to those with ASD, with a VIQ lower than their PIQ. Siegel et al. (1996), however, reported on findings from 16 studies of people with ASD and found that while this profile was common among people with ASD, many participants across studies had intelligence profiles that revealed higher VIQ than PIQ. Moreover, the profile of intelligence suggested to be associated with ASD has also been observed among individuals without ASD as well as individuals with language disorders. Siegel et al. continued to suggest that although certain aspects of a profile of intelligence might be common among individuals with ASD, profiles of intelligence are dependent upon an individual's particular abilities, not his/her ASD diagnosis.

Future Directions

As tests are increasingly co-normed, it will be easier to compare scores not only within a single cognitive tool, but across instruments and domains (e.g., intellectual functioning and memory capacities).

See Also

- ▶ [Picture Arrangement](#)
- ▶ [Processing Speed Index](#)
- ▶ [Psychological Assessment](#)
- ▶ [Verbal Comprehension Index](#)
- ▶ [Wechsler Memory Scale \(All Versions\)](#)
- ▶ [Wechsler Test of Adult Reading](#)

References and Reading

- American Psychological Association. (2002). Ethical principles of psychologists and code of conduct. *American Psychologist*, 57, 1060–1073.
- Lichtenberger, E. O., & Kaufman, A. S. (2009). *Essentials of WAIS-IV assessment*. Hoboken: Wiley.
- Mullen, E. M. (1995). *Mullen scales of early learning*. Circle Pines: American Guidance Service.
- Sattler, J. M., & Ryan, J. J. (2009). *Assessment with the WAIS-IV*. La Mesa: Jerome M. Sattler.
- Siegel, D. J., Minshew, N. J., & Goldstein, G. (1996). Wechsler IQ profiles in diagnosis of high-functioning autism. *Journal of Autism and Developmental Disorders*, 26(4), 389–406. <https://doi.org/10.1007/BF02172825>.
- Wechsler, D. (1997). *Wechsler adult intelligence scale-revised*. New York: Psychological Corporation.
- Wechsler, D. (2002). *Wechsler primary and preschool scale of intelligence* (3rd ed.). San Antonio: Harcourt Assessment.
- Wechsler, D. (2003). *Wechsler intelligence scale for children* (4th ed.). San Antonio: Harcourt Assessment.
- Yirmiya, N., & Sigman, M. (1991). High functioning individuals with autism: Diagnosis, empirical findings, and theoretical issues. *Clinical Psychology Review*, 11, 669–683. [https://doi.org/10.1016/0272-7358\(91\)90125-E](https://doi.org/10.1016/0272-7358(91)90125-E).

Profiling Elements of Prosody in Speech-Communication (PEPS-C)

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Synonyms

[PEPS-C](#)

Definition

Profiling Elements of Prosody in Speech-Communication (PEPS-C) is a semiautomated test battery for assessing receptive and expressive prosody skills. It has been used in several studies

of the speech and understanding of children with high-functioning autism/Asperger's syndrome (see section "References and Readings"). It is intended for use by clinicians and researchers in assessing prosody in any conditions in both children and adults. A demonstration of the test can be found on www.peps-c.com.

PEPS-C comprises 12 tasks, addressing receptive and expressive skills in parallel. The tasks are at two levels, examining prosodic function and prosodic form, respectively. PEPS-C defines four main linguistic functions conveyed by prosody, with a receptive and an expressive task for each:

1. Turn end: indicating whether an utterance requires an answer or not (question/statement)
2. Affect: indicating mood/emotions/opinions – in this test, signaling liking or reservation with respect to food items
3. Chunking: prosodic phrase boundaries indicating how speech can be verbally "chunked," as in the difference between "fruit, salad, and milk" and "fruit salad and milk"
4. Contrastive stress or focus: emphasizing one word in an utterance to focus attention on it, e.g., "white COW" as opposed to "WHITE cow"

Prosodic form processing requires non-cognitive skills. The tasks are:

- Two auditory discrimination tasks, essentially same/different tasks. Stimuli exemplify the prosodic variations that convey the different meanings used in the receptive prosodic function tasks.
- Two imitation tasks requiring the production of the types of prosodic variation needed for completing the expressive function tasks.

The assessment of skills at two levels helps to determine the level at which a client has a problem with prosody, thus enabling better targeting of intervention. The test is not standardized, but a limited amount of normative data is available.

See Also

- [Paralinguistic Communication Assessment](#)
- [Prosody](#)

References and Reading

- Hesling, I., Dilharreguy, B., Peppé, S., Amirault, M., Bouvard, M., et al. (2010). The integration of prosodic speech in high functioning autism: A preliminary fMRI study. *PLoS One*, 5(7), e11571. <https://doi.org/10.1371/journal.pone.0011571>.
- Järvinen-Pasley, A., Peppé, S., King-Smith, G., & Heaton, P. (2008). The relationship between form and function level receptive prosodic abilities in autism. *Journal of Autism and Developmental Disorders*, 38, 1328–1340.
- McCann, J., Peppé, S., Gibbon, F., O'Hare, A., & Rutherford, M. (2007). Prosody and its relationship to language in school-aged children with high-functioning autism. *International Journal of Language & Communication Disorders*, 42(6), 682–702.
- Peppé, S., & McCann, J. (2003). Assessing intonation and prosody in children with atypical language development: The PEPS-C test and the revised version. *Clinical Linguistics & Phonetics*, 17(4/5), 345–354.
- Peppé, S., McCann, J., Gibbon, F., O'Hare, A., & Rutherford, M. (2007). Receptive and expressive prosodic ability in children with high-functioning autism. *Journal of Speech, Language, and Hearing Research*, 50, 1015–1028.
- Peppé, S. J. E., Martínez-Castilla, P., Coene, M., Hesling, I., Moen, I., & Gibbon, F. (2010). Assessing prosodic disorder in five European languages. *International Journal of Speech-Language Pathology*, 12(1), 1–7.
- Peppé, S. J. E., Cleland, J., Gibbon, F. E., O'Hare, A. E., & Martínez-Castilla, P. (2011). Expressive prosody in children with autism spectrum conditions. *Journal of Neurolinguistics*, 24, 41–53.

Profound Hearing Impairment

- [Deafness](#)

Program-Wide Positive Behavior Supports (PWPBS)-Early Childhood Programs

- [Positive Behavioral Interventions and Supports \(PBIS\)](#)

Progressive Applied Behavior Analysis

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Definition

Leaf et al. (2016a, d) described the scientific nature of applied behavior analysis (ABA) and that ABA, as a science, and interventions based upon the principles of this science should be *progressive*. A progressive approach to ABA-based interventions contrasts with *conventional* approaches in several ways. Some hallmarks of a conventional approach to ABA are (1) the implementation of interventions by adhering to protocols with little to no variation, (2) routinely conducting a formal functional analysis prior to intervention, (3) routinely using continuous data to measure behavior, (4) having a limited curricular scope (e.g., only focusing on either reduction of aberrant behavior, language development, or academics), (5) and heavy reliance on formal assessments to determine potential reinforcing events. In essence, a conventional approach to ABA-based intervention can be described as protocol driven in that technicians are governed by rules as opposed to being responsive to the individuals with whom they work with.

Contrasting with a conventional approach, the goal of a progressive approach is to utilize a systematic plan during each session but, also, having the freedom to deviate from that plan at any given moment based upon the needs of the client. To do this, the behavior analyst is constantly making in-the-moment assessments to inform changes to the predetermined systematic plan. There are many variables that contribute to what has been described as clinical judgment which include, but are not limited to, (a) moment-to-moment changes in the function of the client's behavior, (b) interfering or aberrant

behaviors, (c) determination of whether behavior is primarily operant or respondent, (d) current levels of engagement, (e) the client's receptivity to learning, (f) the emotional state of the client, (g) current and past performance, (h) ongoing evaluation of reinforcer effectiveness, and (i) the physical health of the client. Furthermore, a progressive approach includes a formal, analogue functional analysis sparingly, if ever (i.e., the behavior analyst is constantly assessing function throughout the session making this type of functional analysis unnecessary) data collection systems are streamlined, curricular targets are comprehensive, and formal preference assessments are rendered unnecessary through in-the-moment reinforcer analysis (IMRA; Leaf et al. 2015, 2016c). In essence, a progressive approach to ABA is one that is characterized by structured flexibility in that the behavior analyst has a systematic plan but has the flexibility to alter this plan through responsiveness to the client (i.e., the behavior analyst is governed by the client's behavior instead of solely by a protocol).

Historical Background

ABA, as a practice, is rooted in the science of behavior analysis which is interested in the effect of environmental variables on behavior. Specifically, ABA, as a science and practice, is interested in the manipulations of these variables to improve socially significant behavior. The history of the science of behavior analysis has been marked by many important contributions that led to the development of the field of ABA. These events have included, but are not limited to, Edward Thorndike's contribution of the law of effect, Ivan Pavlov's contribution of respondent conditioning, John B. Watson's identification of behaviorism as a methodology, and B. F. Skinner's formulation of operant conditioning (see Moore 2008 for a detailed history of behavior analysis). These and many more contributions resulted in Baer et al. (1968) writing a seminal article which defined some important dimensions of ABA. Within their seminal article, Baer and colleagues described ABA as involving at least seven

dimensions which included applied, behavioral, analytic, technological, conceptually systematic (referred to as a conceptual system), effective, and generalizable (referred to as generality). Since 1968, there have been thousands of studies embodying the dimensions Baer et al. put forth to improve socially significant behavior. Today, interventions based upon the principles of ABA are in widespread use for treatment of individuals diagnosed with autism spectrum disorder (ASD) and have the strongest body of empirical evidence demonstrating its effectiveness (Smith and Iadarola 2015).

In the pioneering days of behavior analytic intervention, it was common for behavior analysts to utilize a structured yet flexible approach to intervention. This may have been a result of the dearth of established protocols during these initial years, which required interventionists to be innovative and pay close attention to what is happening in front of them. This flexible approach to intervention is exemplified by many of the early behavior analytic studies such as Wolf et al. (1963) who explored the use of a shaping procedure with a young boy with autism. The shaping procedure was used to successfully decrease tantrums, help with bedtime problems, and increase eyeglass wearing for the participant (Dickey). In another seminal study, Ayllon and Azrin (1965) evaluated the use of a token economy to improve the behavior of patients at a mental hospital ward. Targeted skills included serving meals, filling water bottles, and answering the telephone. Tokens were delivered contingent upon completion of the tasks, and these tokens were exchanged for a presumed reinforcing item. To identify these potential reinforcers, "No a priori decisions were made about what should be an effective reinforcer. Instead, patients' behavior was used to discover reinforcers" (Ayllon and Azrin 1965, p. 358). Thus, the researchers used in-the-moment assessment to determine what may or may not function as a reinforcer as opposed to conducting a formal preference assessment.

A progressive approach to ABA was also demonstrated by researchers evaluating comprehensive behavioral intervention for individuals diagnosed with ASD. For instance, Lovaas et al. (1973)

and Lovaas (1987) provided comprehensive intervention using a variety of behavioral techniques (e.g., discrete trial teaching, incidental teaching, and shaping) to improve the participants' cognitive skills, language skills, play skills, and reduction of aberrant behavior. The intervention in both studies was comprehensive in scope and intensive (e.g., providing an average of 40 h of intervention per week). The results of these studies demonstrated that comprehensive and intensive behavioral intervention resulted in children making significant gains on standardized assessments (e.g., IQ scores) and improvements on global behaviors (e.g., educational progress). While professionals may suggest that the approach was conducted in a rigid/conventional manner, those involved in the project described what aligns closer to a progressive approach (Leaf and McEachin 2016). This is illustrated by the lack of set protocols on what to teach, how to teach, the prompting systems used within teaching, or the way data was taken. Thus, two seminal and commonly cited studies in the behavioral treatment of ASD were done using a progressive approach.

Since the Lovaas (1987) report, the number of studies evaluating behavioral interventions for individuals diagnosed with ASD and the number of individuals receiving behavioral services have increased (Dorsey et al. 2009). Unfortunately, this increase has resulted in the manualization of ABA and a de-emphasis on the need for intensive and quality training. As such, many behavior analysts adhere to the latter rather than the intent of manuals and protocols (Leaf et al. 2016d). Today, it is more common to observe a conventional approach to ABA, which contrasts with the early years of ABA during which remarkable outcomes were achieved. In the last 10 years, however, researchers and clinicians have started to evaluate and describe how the progressive approach can successfully be implemented for individuals diagnosed with ASD.

Current Knowledge

There are many examples in the research of evaluations and descriptions of a progressive

approach to ABA-based interventions. In the text below, we will describe some of the current knowledge of the progressive approach and briefly contrast it to a more conventional model.

Reinforcement identification. In a conventional approach, behavior analysts typically conduct formal preference assessments (e.g., multiple stimulus without replacement, paired stimulus, single stimulus) in which one potentially reinforcing item is compared to another or to an array of stimuli. As previously stated, in a progressive approach, the necessity for formal and a priori preference assessment is eliminated. This is done by the frequent use of in-the-moment assessments to determine what stimuli to use as potential reinforcers. Within these in-the-moment assessments, the behavior analyst evaluates multiple variables including affect, verbal and nonverbal behavior, and engagement.

Leaf and colleagues (2015) compared a paired stimulus preference assessment to IMRA on the rate of responding with several children diagnosed with ASD during a simple sorting task. A paired stimulus preference assessment was conducted during which the participants selected among ten stimuli that would be used during the sorting task. In the paired stimulus condition, the researcher could only use the top three stimuli identified from the paired stimulus preference assessment, and in the IMRA condition the researcher, who was blind to the results of the paired stimulus preference assessment, could use any of the ten stimuli. The results of the study showed equivalent rates of sorting across the two conditions, but using IMRA, as opposed to the paired stimulus preference assessment, was more efficient with respect to time spent conducting the formal preference assessment. Leaf and colleagues (2016c) extended the findings of Leaf et al. (2015) by comparing the paired stimulus preference assessment to IMRA when teaching expressive labels with two children diagnosed with ASD. The methods employed were identical to those of Leaf et al. (2015) utilized; however, Leaf et al. (2016c) evaluated the two conditions with respect to the acquisition of a new skill (i.e., expressive labels). The results were similar to Leaf et al.'s (2015) study in that the participants

acquired the skills in both conditions, but the IMRA condition was again more efficient with respect to time spent in the formal assessment.

Reinforcement systems. Once potentially reinforcing events have been identified for use during intervention, reinforcement systems (e.g., token system) are typically developed to help improve pro-social behavior. Within a progressive approach, the behavior analyst is not limited to providing reinforcement on a predetermined schedule (e.g., FR1, VR3) or for predetermined behaviors (e.g., always provide access to candy following X response). Instead, the interventionist has the discretion to make in-the-moment adjustments to the reinforcement schedule as needed (e.g., when it is determined that a high level of response effort is required due to transient circumstances). In one recent example of a responsive, flexible reinforcement system, Leaf and colleagues (2017) used a level reinforcement system as part of a behaviorally based social skills group for children diagnosed with ASD. Within this level system, the children could move up and down within and across various levels, and each level represented a different contingency (e.g., the top indicated access to a treasure chest with various toys, the bottom indicated a brief time out from the activity). During the social skills group, the interventionists had the flexibility to provide reinforcement (i.e., movement up) and punishment (i.e., movement down) based upon his or her in-the-moment assessment of child performance. Thus, the rate of reinforcement was not predetermined and, instead, was responsive to child performance on an individual basis. Overall, the results of the randomized control trial showed significant gains in the social domain across a variety of standardized assessments for all participants.

Discrete trial teaching. One of the most commonly implemented behavioral techniques for individuals diagnosed with ASD is discrete trial training (DTT). The common implementation of DTT has led to descriptions and best practice recommendations of how to implement DTT (e.g., Grow and LeBlanc 2013; Leaf et al. 2016a). Some recommendations that align closer to a conventional approach have included only

using counterbalancing and an array of three stimuli with teaching receptive labels, only using simplified instructions (e.g., saying “ball” as opposed to “find the ball”) and not varying the instruction from trial to trial (Grow and LeBlanc 2013). These recommendations align with a conventional approach as they are followed without assessing and responding to a particular child, and instead the interventionist is responsive to a set of rules. Leaf et al. (2016a) outlined how this conventional approach contrasts with a progressive approach to DTT. Across the guidelines provided for a progressive approach to DTT, a general theme emerged. Each guideline described assessing the conditions under which an interventionist should or should not alter certain variables such as complexity of language, stimulus presentation, and selection of prompting systems. Ultimately, within a progressive approach to DTT, interventionists are not bound by inalterable rules and make in-the-moment assessments to determine what, if any, changes should be made from trial to trial, session to session, and child to child.

In a recent study, Leaf et al. (in press) compared elements of a conventional to a progressive approach to DTT for the five participants diagnosed with ASD. Specifically, Leaf and colleagues evaluated the recommendation to counterbalance the stimulus array when teaching receptive labels by comparing it to two conditions, one in which the stimuli were not moved across trials and another in which the interventionist had the flexibility to move or not move the stimuli across trials. The results were idiosyncratic across the five participants and stimulus sets. That is, there was no substantial difference when stimulus rotation was prescribed to move or not move on every trial. While this study had limitations and further research is needed, these preliminary findings show the importance of evaluating claims of best practice recommendations.

Prompting. One component which is usually a commonly included component within DTT is the provision of prompts. Within a conventional approach, a protocol typically controls when the interventionist prompts, fades a prompt, and what prompts to provide. A more progressive approach, on the other hand, utilizes what has been

described as flexible prompt fading (FPF). This prompting system relies on in-the-moment assessment to guide interventionist's selection of prompts and prompt fading. Instead of strict rules, FPF is guided by in-the-moment assessment and a goal to maintain the child's success rate at around 80% of opportunities.

Several studies have evaluated the use of FPF to teach various skills for individuals diagnosed with ASD. For instance, Soluaga and colleagues (2008) compared FPF to a constant time delay prompt to teach a variety of receptive labels to five children diagnosed with ASD. The results of a parallel treatments design showed that both prompting procedures were effective at teaching the various skills, but FPF was more efficient. Leaf et al. (2014) compared FPF to an error correction procedure to teach expressive labels to four children diagnosed with ASD. The results of a parallel treatments design again showed that both procedures were effective and FPF was more efficient. More recently, Leaf and colleagues (2016b) compared FPF to most-to-least prompting for teaching expressive labeling to four children diagnosed with ASD. Once again, both procedures were effective, but FPF was more efficient for three out of the four participants. Furthermore, FPF resulted in higher rates of independent correct responding during teaching. Taken together, these evaluations of a more progressive approach to prompting show that responding to child behavior, instead of to a protocol, can be effective and more efficient than a protocol-driven conventional approach.

Data collection. A cornerstone of any science is data-based decisions, and ABA is no exception. Specifically, ABA relies on objective data to make decisions about progress during treatment and to continue or discontinue certain procedures. Conventionally, data is taken continuously (e.g., frequency counts), discontinuously (e.g., time interval), or some combination of the two for all behavior in all environments. In a progressive approach to ABA, data is critical; however, the type of data system varies based upon several variables such as the task, frequency of the behavior, duration of the behavior, how informative the data is for demonstrating progress or informing

changes to the intervention, and the extent to which data collection might interfere with learning opportunities. Thus, continuous measurement may be indicated in some instances, while discontinuous measurement is indicated in other instances, and, at times, estimation data (Taubman et al. 2013) may be appropriate. Therefore, one type of measurement system is not used exclusively over others, unless that is what is required for the child and staff.

Global outcomes. To date, there have been two comprehensive studies that have evaluated a progressive approach to ASD intervention. Leaf and colleagues (2011) provided a programmatic description (i.e., an observational study without the use of an experimental design) of a clinic-based progressive approach to ASD intervention for individuals diagnosed with ASD. This description included an analysis of data for 64 individuals diagnosed with ASD who received services in 4 clinics based in the United States, Hong Kong, Australia, and the United Kingdom. The results of the analysis showed that 39% of the participants reached "best outcome" based on the criteria Lovaas (1987) outlined. Furthermore, an additional 31% would be considered best outcome by a revised definition (i.e., the same criterion used in Lovaas (1987) with the caveat that clients could receive minimal school support). Taken together, 70% of the clients served across these clinics providing a progressive approach to ASD intervention reached best outcome measures.

More recently Leaf et al. (2016) used a progressive approach to ASD intervention as part of a 16-week behaviorally based social skills group intervention. The interventionists in this group implemented a range of techniques (e.g., discrete trial teaching, shaping, cool vs. not cool, role play). The curriculum was not prespecified, was based upon several factors (e.g., child deficits, parent's desires of what to teach their child, or age typical social behaviors), and was individualized. The results of a randomized control trial showed that the intervention was successful at achieving significant gains in social behavior that were maintained over time. These two evaluations provide a small sample of progressive approaches to ASD intervention that result in

significant, meaningful gains, for individuals with diagnosed ASD.

Future Directions

Despite the recent increase in research and descriptions of a progressive approach to ASD intervention, more research is needed. First, future researchers should evaluate a progressive approach with a wider body of techniques and approaches to treatment (e.g., functional analyses or using consumer-friendly language as opposed to technical jargon). Second, researchers should continue to compare elements of a progressive approach to other approaches to identify the most efficacious techniques. Third, researchers should evaluate training professionals in a progressive approach. Fourth, researchers and professionals should continue to identify the characteristics of a progressive approach. Finally, research needs to be conducted across a wider variety of professionals and research labs. Continuing these lines of research would extend the knowledge of a progressive approach to ABA and ASD intervention and may lead to better outcomes for individuals diagnosed with ASD.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behaviorally Based Social Skill Groups](#)

References and Reading

- Ayllon, T., & Azrin, N. H. (1965). The measurement and reinforcement of behavior of psychotics. *Journal of the Experimental Analysis of Behavior*, 8(6), 357–383.
- Baer, D. M., Wolf, M. M., & Risley, T. R. (1968). Some current dimensions of applied behavior analysis. *Journal of Applied Behavior Analysis*, 1(1), 91–97.
- Dorsey, M. F., Weinberg, M., Zane, T., & Guidi, M. M. (2009). The case for licensure of applied behavior analysts. *Behavior Analysis in Practice*, 2(1), 53–58.
- Ghaemmaghami, M., Hanley, G. P., Jin, S. C., & Vanselow, N. R. (2016). Affirming control by multiple reinforcers via progressive treatment analysis. *Behavioral Interventions*, 31(1), 70–86.
- Grow, L., & LeBlanc, L. (2013). Teaching receptive language skills: Recommendations for instructors. *Behavior Analysis in Practice*, 6(1), 56–75.
- Hanley, G. P., Iwata, B. A., & McCord, B. E. (2003). Functional analysis of problem behavior: A review. *Journal of Applied Behavior Analysis*, 36(2), 147–185.
- Iwata, B. A., Dorsey, M. F., Slifer, K. J., Bauman, K. E., & Richman, G. S. (1982). Toward a functional analysis of self-injury. *Analysis and Intervention in Developmental Disabilities*, 2(1), 3–20.
- Leaf, R., & McEachin, J. (2016). The Lovaas model: Love it or hate it, but first understand it. In R. G. Romanczyk & J. McEachin (Eds.), *Comprehensive models of autism spectrum disorder treatment* (pp. 7–43). Seal Beach: Springer.
- Leaf, R. B., Taubman, M. T., McEachin, J. J., Leaf, J. B., & Tsuji, K. H. (2011). A program description of a community-based intensive behavioral intervention program for individuals with autism spectrum disorders. *Education and Treatment of Children*, 34(2), 259–285.
- Leaf, J. B., Leaf, R., Taubman, M., McEachin, J., & Delmolino, L. (2014). Comparison of flexible prompt fading to error correction for children with autism spectrum disorder. *Journal of Developmental and Physical Disabilities*, 26(2), 203–224.
- Leaf, J. B., Leaf, R., Alcalay, A., Leaf, J. A., Ravid, D., Dale, S., et al. (2015). Utility of formal preference assessments for individuals diagnosed with autism spectrum disorder. *Education and Training in Autism and Developmental Disabilities*, 50(2), 199–212.
- Leaf, J. B., Cihon, J. H., Leaf, R., McEachin, J., & Taubman, M. (2016a). A progressive approach to discrete trial teaching: Some current guidelines. *International Electronic Journal of Elementary Education*, 9(2), 361–372.
- Leaf, J. B., Leaf, J. A., Alcalay, A., Kassardjian, A., Tsuji, K., Dale, S., et al. (2016b). Comparison of most-to-least prompting to flexible prompt fading for children with autism spectrum disorder. *Exceptionality*, 24(2), 109–122.
- Leaf, J. B., Leaf, R., Leaf, J. A., Alcalay, A., Ravid, D., Dale, S., et al. (2016c). Comparing paired-stimulus preference assessments with in-the-moment reinforcer analysis on skill acquisition: A preliminary investigation. *Focus on Autism and Other Developmental Disabilities*, 1–11.
- Leaf, J. B., Leaf, R., McEachin, J., Taubman, M., Ala'i-Rosales, S., Ross, R. K., et al. (2016d). Applied behavior analysis is a science and, therefore, progressive. *Journal of Autism and Developmental Disorders*, 46(2), 720–731.
- Leaf, J. B., Leaf, J. A., Milne, C., Taubman, M., Oppenheim-Leaf, M., Torres, N., et al. (2017). An evaluation of a behaviorally based social skills group for individuals diagnosed with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(2), 243–259.
- Leaf, J. B., Cihon, J. H., Ferguson, J. L., McEachin, J., Leaf, R., & Taubman, M. (2018). Evaluating three

methods of stimulus rotation when teaching receptive labels. *Behavior Analysis in Practice*. Advance online publication. <https://doi.org/10.1007/s40617-018-0249-5>

Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55(1), 3–9.

Lovaas, O. I., Freitag, G., Gold, V. J., & Kassorla, I. C. (1965). Experimental studies in childhood schizophrenia. *Journal of Experimental Child Psychology*, 2, 67–84.

Lovaas, O. I., Koegel, R., Simmons, J. Q., & Long, J. S. (1973). Some generalization and follow-up measures on autistic children in behavior therapy. *Journal of Applied Behavior Analysis*, 6(1), 131–165.

Moore, J. (2008). *Conceptual foundations of radical behaviorism*. Cornwall-on-Hudson: Sloan.

Smith, T., & Iadarola, S. (2015). Evidence base update for autism spectrum disorder. *Journal of Clinical Child & Adolescent Psychology*, 44(6), 897–922.

Soluaga, D., Leaf, J. B., Taubman, M., McEachin, J., & Leaf, R. (2008). A comparison of flexible prompt fading and constant time delay for five children with autism. *Research in Autism Spectrum Disorders*, 2(4), 753–765.

Taubman, M. T., Leaf, R. B., McEachin, J. J., Papovich, S., & Leaf, J. B. (2013). A comparison of data collection techniques used with discrete trial teaching. *Research in Autism Spectrum Disorders*, 7(9), 1026–1034. <https://doi.org/10.1016/j.rasd.2013.05.002>.

Wolf, M., Risley, T., & Mees, H. (1963). Application of operant conditioning procedures to the behaviour problems of an autistic child. *Behaviour Research and Therapy*, 1(2), 305–312.

HTP	House-Tree-Person Test
KFD	Kinetic Family Drawing
RISB	Rotter’s Incomplete Sentence Blank
RIT	Rorschach Inkblot Test
R-PAS	Rorschach Performance Assessment System
SAT	Senior Apperception Test
TAT	Thematic Apperception Test

Nature of Projective Tests

Projective tests, also commonly called projective techniques, encompass a class of psychological measures characterized by two key features. First, they present a relatively unstructured or ambiguous stimulus (e.g., an inkblot) to which a person responds. Second, they allow for great latitude in the person’s response. Projective tests are contrasted with objective or structured measures which present a simple, clear stimulus (e.g., a statement such as “I often feel strange”) accompanied by a selected-response method of responding (e.g., Yes/No).

The rationale underlying use of a projective technique is the “projective hypothesis” which asserts that when faced with an ambiguous stimulus and given great freedom in responding, an individual’s personality dynamics will determine the response; and, therefore, the projective technique may reveal dynamics not detected by objective measures.

Projective Testing

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Abbreviations

ASD	Autism Spectrum Disorders
CAT	Children’s Apperception Test
CS	(Exner’s) Comprehensive System
DAP	Draw-A-Person
DAP	Draw-a-Person: Screening
SPED	Procedure for Emotional Disturbance
HFD	Human Figure Drawing

Extent of Use

Surveys show that projective techniques are very widely used in clinical assessment. This has been true over a long period of time and across many subfields including clinical, counseling, school, and forensic psychology, and neuropsychology (Hogan 2015). In several surveys identifying the most widely used psychological tests, various projective techniques usually garner four or five of the top ten spots. Doctoral work in clinical psychology also typically requires training in use of projective techniques such as the Rorschach. Some uses of projective techniques involve formal scoring, as described later. However, some

uses are informal, serving an “ice breaker” function: presentation of a nonthreatening stimulus to facilitate initial discussion and establishing rapport with a client without formal scoring.

Indicators for Use of Projective Techniques

Certain circumstances may suggest that use of projective techniques is particularly appropriate. First, most projective techniques require little or no literacy skills and, therefore, may be useful with persons of low educational level. Second, projective techniques, with their ambiguous stimuli, are less susceptible to faking (good or bad) than more objective measures. Third, projective techniques may be especially useful for hypothesis generation when the clinician has little initial notion regarding the source of a person’s difficulty.

The following sections describe the most widely used projective techniques, including basic description of the techniques and their typical applications.

Rorschach

The Rorschach Inkblot Test (RIT) is clearly the most widely used projective technique. The standard version of the RIT consists of 10 bilaterally symmetrical inkblots, five with mixtures of black and various shades of gray, two with black and gray with splotches of red, and three with mixtures of pastels. Each inkblot appears in the middle of a rigid piece of cardboard measuring about 6×9 inches.

Based on appearance of inkblots in the popular media, some people may think that administration and interpretation of the inkblots is a simple, intuitive matter: Show an inkblot, get a response, and, presto, you know a person’s innermost thoughts and feelings. That is certainly not the case with formal use of the Rorschach in contemporary clinical practice. Following Hermann Rorschach’s (1921) publication of these inkblots, several systems for administering, scoring, and

interpreting the RIT emerged. In the early 1970s, John Exner began development of the Comprehensive System (CS) for administration and scoring the RIT, selecting what he judged the best and most promising features of previous systems. The CS is now the industry standard for formal use of the RIT. Exner’s three volumes (Exner 2003; Exner and Erdberg 2005; Exner and Weiner 1995) serve, in effect, as the test manual for the RIT-CS.

Administration of the RIT-CS involves two phases. First, in the response phase, the examiner hands one of the inkblot cards to the client and asks “What might this be?” If necessary, the examiner encourages responses with statements such as “Most people see more than one thing.” The examiner records responses verbatim, as nearly as feasible. The examiner presents each card in turn with the latter directions. Then, in the inquiry phase, the examiner reintroduces each card, reminds the client of his or her response(s), and asks for elaboration on the responses, saying, for example, “Show me where you saw ____.” The examiner also records these responses verbatim.

Scoring the RIT-CS involves, first, application of an elaborate set of codes to the responses and, second, conversion of these codes into a host of ratios, percentages, and derivations. Codes occur in several major categories, for example, location, form quality, and determinants. The codes within the location category tell, for example, whether the response was to the entire blot (W), a common detail (D), an unusual detail (Dd), or to a space (S) rather than to the inkblot itself. Determinants deal with what features of the blot determined the response, for example, an animal figure, human figure, or movement. The host of coded responses are then converted into a variety of derivations (e.g., ideation, affect) and “constellations” (e.g., depression index, coping deficit index), eventuating in the Structural Summary, which serves as the primary basis for interpretation in the RIT-CS. Interpreting the Structural Summary requires advanced training. Computer programs aid in development of the Structural Summary after the initial coding, and computer-generated narrative reports are also available to aid interpretation.

Following Exner's death in 2006, several of his understudies, plus others, developed the Rorschach Performance Assessment System (R-PAS). This system is similar to but not identical to Exner's CS. R-PAS maintains a very active website (r-pas.org), program of research, and series of training opportunities. R-PAS now rivals CS in usage (Gurley 2017). See also Groth-Marnat and Wright (2016) and Weiner and Greene (2017) for comparisons of CS and R-PAS. Mihura and Meyer (2018) provide examples of the use of R-PAS in a variety of applications.

More so than for other categories of psychological assessments, exceptional controversy surrounds the use of projective techniques, with particularly vigorous debate centered on the Rorschach. Some authorities believe even the better known projectives, to say nothing of clearly discredited techniques, generally lack acceptable psychometric quality. Other authorities maintain that selected projective techniques, properly administered and interpreted, compare favorably with widely used measures of cognitive ability and objective measures of personality traits. See Krishnamurthy et al. (2013) for a summary of the arguments pro and con. Meta-analyses of validity studies for the RIT by Mihura et al. (2013) and Wood et al. (2015) are particularly helpful in showing that previously contending factions now agree that at least some Rorschach scores have respectable validity.

Thematic Apperception Test and Other Story-Telling Techniques

Another widely used projective technique is the *Thematic Apperception Test* (TAT). The TAT consists of 30 cards, 9 × 12 inches, each containing a photo deliberately selected to present an ambiguous situation; one card is entirely blank. Directions vary but generally ask the respondent to tell a story, including what is happening, what led up to the situation, and what the outcome may be. Not all cards are used with all respondents; some are meant only for boys, others only for men, others for girls, and others for women. Some examiners may select their own subsets of

cards. In clinical practice, clients usually respond orally. Research applications, with literate respondents, often employ a written response.

The TAT was developed by Murray (1943) to help measure his personality theory based on sets of psychological needs (e.g., affiliation, achievement) and presses (environmental forces). In contemporary practice, the TAT has numerous scoring systems (Jenkins 2008; Teglasi 2010). Typically, the systems use only 8–10 of the 30 cards, with exact selections varying among the systems. Bellak's system (Bellak and Abrams 1997) is one of the more widely cited. The diversity of systems militates against the development of research-based generalizations for the TAT, likely explaining the decline in its usage.

The TAT has two offshoots: The *Children's Apperception Test* (CAT) and *Senior Apperception Test* (SAT, not to be confused with the SAT used for college admissions), both similar to the TAT in purpose and general structure but using pictures more appropriate for their respective target groups. Bellak's work covers the CAT and SAT as well as the TAT.

Research with the TAT shows that it has some limited utility in measuring a few of the traits originally postulated by Murray, for example, achievement motivation, need for affiliation, and aggression. However, its use in ordinary clinical practice has declined in recent years primarily due to unsystematic administration and scoring procedures and a lack of rigorous psychometric work (Dana 1996).

Another story-telling projective technique is the *Roberts-2* (Roberts and Gruber 2005), a revision of the *Roberts Apperception Test for Children*, intended for ages 6–18. Like the prototype TAT, the test presents a series of pictures and asks the respondent to create a story with a beginning, middle, and end, and with specific reference to feelings evoked by the picture. The test aims to measure what it refers to as social understanding. It applies a set of codes for responses in a manner similar to that described earlier for the RIT-CS. Application of the codes results in 18 Developmental/Adaptive scales (e.g., Limit Setting, Constructive Resolution) and 10 Clinical scales (e.g., Aggression, Maladaptive Outcome). Although

the manual claims the test is not a projective technique, it clearly is projective by any conventional definition. An accompanying “casebook” provides one case of a child with autism and one with Asperger’s disorder, as well as a host of other types of cases. However, the manual presents no normative information for autism or other developmental disorders. The manual provides data for a general standardization sample and for a sample of undifferentiated referred clinical cases. Normative information for groups of cases such as those included in the casebook would enhance usefulness of the *Roberts-2*, particularly for research on social understanding in children with autistic spectrum disorders.

Sentence Completion Tests

The sentence completion technique presents the respondent with a few words in a sentence stem (e.g., I feel . . . or Most people . . .) and the respondent completes the sentence in his or her own words. The sentence completion technique is the only widely used projective technique that requires writing; it, therefore, depends on some degree of literacy and is not typically used below the high school level.

Numerous specific sentence completion tests (SCTs) are available; Holaday et al. (2000) described 15 SCTs. The most widely used is *Rotter’s Incomplete Sentences Blank* (RISB; Rotter et al. 1992). It consists of 40 incomplete sentences. There are three forms: high school, college, and adult. RISB attempts to measure only one psychological construct: maladjustment. The RISB manual provides specific guidance for rating each completed sentence on a seven-point scale for the degree of adjustment/maladjustment reflected in the wording. These ratings on individual sentences then sum to a total maladjustment score.

Torstrick et al. (2015) and Weis et al. (2008) provided favorable evidence about the validity of the RISB as a measure of maladjustment. Because sentence completion techniques require both reading, albeit minimal, and writing, they are not typically used with children.

Human Figure Drawings

Human figure drawings (HFD), also known as draw-a-person (DAP) tests, encompass a generalized technique as well as several specific tests. As a general technique, the procedure usually calls for the respondent, first, to “draw a picture of yourself,” followed by “draw a picture of a person of the opposite sex,” and may also include “draw a picture of your mother (or some other named person).”

Many clinicians “score” the drawings in only the most impressionistic manner. Alternatively, a variety of specific scoring systems have been proposed. The earliest use of the procedure aimed to provide a nonverbal measure of intelligence and the method still experiences some use for that purpose (Naglieri 1988), although it is much more frequently used as a projective measure of personality. Machover (1949) offered a widely cited but largely discredited system for interpreting human figure drawings. Naglieri et al. (1991) developed the *Draw-a-Person: Screening Procedure for Emotional Disturbance* (DAP: SPED) as an objectively scored, psychometrically sound version of the technique. The DAP: SPED manual provides comparative data for several special education samples in the age range 10–12 years but with no apparent representation of autistic syndrome disorder (ASD) cases.

Other drawing-based techniques include the *House-Tree-Person* test, with directions calling for, as suggested by the title, drawing a house, tree, and person; and the *Kinetic Family Drawing*, with directions calling for drawing a picture of a family doing something.

Despite the widespread use of human figure drawings in clinical practice, they have not fared well in psychometric research (Sattler 2014; Weiner and Greene 2017). At best, they may reveal some aspects of gross ideational or emotional aberration, and they may serve the purpose of hypothesis generation for later follow-up or as an ice-breaker.

Bender Visual-Motor Gestalt Test

The *Bender Visual-Motor Gestalt Test-II* (Brannigan and Decker 2003), usually referred to simply as the

Bender, consists of a series of geometric configurations presented on individual cards (e.g., two intersecting wavy lines). Administration proceeds in two phases: copy and recall. First, the examiner presents each card in turn and asks the client to copy the figure. After all cards have been presented, the examiner asks the client to draw as many of the designs as the client can recall. Each response is scored on a five-point scale from no-resemblance to near-perfect resemblance to the original figure.

The Bender's original purpose and most frequent uses are for neuropsychological evaluation. However, some clinicians use it as a projective technique, scoring responses for "indicators" of ideational aberration or emotional disturbance.

Clinical Uses of Projective Techniques for Autism Spectrum Disorders

Psychological tests receive use for initial diagnosis of a condition, treatment planning, follow-up evaluation of treatment outcomes, and for research to better understand a condition. Projective techniques appear to have little relevance for initial diagnosis for ASD cases. Initial diagnosis typically occurs at an age below the functional range for projective techniques. Furthermore, several well-established objective tests and methods exist for evaluating communication skills and social relationships relevant to autism spectrum disorders (Bishop et al. 2008; Ibanez et al. 2014; Lord et al. 2014). Projective techniques may be useful at older ages for treatment planning and follow-up evaluation, especially for comorbid conditions. However, any clinical use of projective measures with this population will require careful attention to the developmental level of the individual being assessed, and, in the absence of appropriate normative data for ASD samples, interpretations will necessarily be quite tentative.

Projective techniques may be of use for a variety of research purposes to develop broader understanding of the emotional and social lives of individuals with autism spectrum disorders. To date, uses of projective techniques with ASD cases have been characterized by isolated studies

with quite limited samples. However, some promising lines of research have developed. Emotion researchers have noted the importance of self-report information in understanding the emotional experiences of children and adolescents (Zeman et al. 2007), and projective measures can help elicit such information. An example with high-functioning ASD is found in the work of Bauminger et al. (2004), who studied loneliness, perceptions of friendships, and understanding of social interactions in school-aged children using, among other measures, thematic picture-based procedures with open-ended questioning. The Friendship Picture Recognition Interview (see Bauminger et al. 2004), described by the authors as a projective test, consists of a single color drawing of two children engaged in a close social interaction. Examiners ask all children a set of three questions and, depending on response to the third question, some children are also asked a fourth. The authors provide the procedure for scoring responses along with some information about interrater agreement. Informative results have been obtained from this and the group's other picture-based measures, but there is no indication in the literature of use of the measures by other researchers. Kenworthy (2010) provides another example of the use of the story-telling method, specifically the *Roberts-2*, in this instance to track changes in social perception in an ASD case.

In a study on theory of mind, Beaumont and Newcombe (2006) note that important information about high-functioning individuals' understanding of others' mental states can be obtained from measures that include spontaneous speech and narratives. That is, open-ended measures can add to the information obtained from more structured, objective measures. They included an "open-ended TAT narrative task" in a comparison of 20 individuals with high-functioning autism or Asperger's disorder and 20 controls matched on IQ, sex, and age. Six of Murray's TAT cards were used and the directions given to participants were the same as specified in the manual. Responses were not coded for traditional personality themes but for theory of mind constructs, using categories based on previous narrative studies with children by theory of mind researchers. Hypothesized differences were

found between the ASD and control groups on “mental state causal statements.” The results add interesting information about specific aspects of mental state understanding in this group.

Projective measures might also have utility for research on emotions and social perceptions in lower-functioning adolescents and adults. Dykens et al. (2007) have called for research into the rich internal lives of individuals with intellectual disabilities (ID) and suggested “semi-projective” measures as a way to obtain self-reports from these individuals. They recently used sentence completion and three-wishes tasks to obtain information about emotions and aspirations from individuals with ID associated with genetic syndromes. Content analysis was utilized and high intraclass correlations reported. The measures might prove useful for similar purposes with individuals with ASD and ID.

Several studies have employed the Rorschach with ASD cases, in all instances using Exner’s CS to report scores. Dykens et al. (1991) used the Rorschach to examine thought disorder in 11 high-functioning ASD cases and compared results with the CS schizophrenia reference norms. Ghaziuddin et al. (1995) also used the CS to compare 12 Asperger syndrome cases with 8 high-functioning autism cases and found differences on a few of the structural summary variables. It should be noted that Exner’s structural summary provides a score labeled ALOG, which originally stood for Autistic Logic but was later renamed Inappropriate Logic, although the acronym ALOG was retained. One should not assume that ALOG relates to autism in any special way.

Holaday et al. (2001) tested hypothesized differences in Rorschach protocols between 24 boys with Asperger’s disorder, 24 with “other emotional or behavioral disorders,” and the normative data. Several expected differences were found on social variables and, importantly, the authors provided group means from both of their sample groups for all of the variables in Exner’s normative tables, with a stated goal of improving the Rorschach’s potential utility for difficult differential diagnosis between Asperger’s and other conditions.

Yalof (2006) found several scores in Exner’s CS for the Rorschach useful in a detailed case study of a difficult differential diagnosis related

to nonverbal learning disability and Asperger’s syndrome. Yalof also reported using figure drawings and the TAT in this case study but appeared to use these two measures only impressionistically rather than in terms of formal scoring.

Several reports have considered the use of human figure drawings with ASD cases. Klin et al. (2005) suggested that informal use of drawings may have some use in understanding an autistic child’s thinking. Lee and Hobson (2006) obtained intriguing differences between 14 autistic cases and 14 learning disability cases matched on age and verbal ability in certain characteristics of human figure drawings but not on house drawings. Lim and Slaughter (2008) also used human figure, house, and tree drawings to compare 29 Asperger’s syndrome cases with 28 typically developing children, matched on gender, age, and nonverbal IQ, and found differences in the human figures but not on the house or tree drawings.

Roman (2005) proposed a free-play based projective technique (the *Projective Kit for Early Childhood*) for children aged 6 months to 4 years, suggesting that it might help “... to identify milestones in autistic vs. nonautistic mechanisms of relationship to the world through the children’s play” (Roman et al. 2011, p. 249). To date, this relatively new technique has not garnered much research attention.

Research literature on use of projective techniques with autistic syndrome disorder cases is not large but it is growing and it has suggested some possibly fruitful areas for further development. There is a particular need for normative studies of projective indexes with sizable groups of ASD cases. Such information may provide a useful adjunct to the currently established diagnostic instruments. Studies reviewed here also suggest that projectives may help to expand our understanding of the cognitive and social interaction features of ASD.

See Also

- [Clinical Assessment](#)
- [Diagnostic Instruments in Autistic Spectrum Disorders](#)

- Diagnostic Process
- Human Figure Drawing Tests
- Projective Tests of Personality

References and Reading

- Bauminger, N., Shulman, C., & Agam, G. (2004). The link between perceptions of self and social relationships in high-functioning children with autism. *Journal of Developmental and Physical Disabilities, 16*, 193–214.
- Beaumont, R., & Newcombe, P. (2006). Theory of mind and central coherence in adults with high-functioning autism or asperger syndrome. *Autism, 10*, 365–382.
- Bellak, L., & Abrams, D. M. (1997). *The thematic apperception test, the children's apperception test, and the senior apperception test in clinical use* (6th ed.). Boston: Allyn and Bacon.
- Bishop, S. L., Luyster, R., Richler, J., & Lord, C. (2008). Diagnostic assessment. In K. Chawarska, A. Klin, & F. R. Volkmar (Eds.), *Autism spectrum disorders in infants and toddlers: Diagnosis, assessment, and treatment* (pp. 23–49). New York: Guilford.
- Brannigan, G. G., & Decker, S. L. (2003). *Bender visual-motor gestalt test II examiner's manual*. Itasca: Riverside.
- Dana, R. H. (1996). The thematic apperception test (TAT). In C. S. Newmark (Ed.), *Major psychological assessment instruments* (2nd ed., pp. 166–205). Boston: Allyn and Bacon.
- Dykens, E., Volkmar, F., & Glick, M. (1991). Thought disorder in high-functioning autistic adults. *Journal of Autism and Developmental Disorders, 21*, 291–301.
- Dykens, E., Schwenk, K., Maxwell, M., & Myatt, B. (2007). The sentence completion and three wishes tasks: Windows into the inner lives of people with intellectual disabilities. *Journal of Intellectual Disability Research, 51*, 588–597.
- Exner, J. E., Jr. (2003). *The Rorschach: A comprehensive system, Basic foundations and principles of interpretation* (Vol. 1, 4th ed.). New York: Wiley.
- Exner, J. E., Jr., & Weiner, I. B. (1995). *The Rorschach: A comprehensive system, Assessment of children and adolescents* (Vol. 3, 2nd ed.). New York: Wiley.
- Exner, J. E., Jr., & Erdberg, P. (2005). *The Rorschach: A comprehensive system, Advanced interpretation* (Vol. 2, 3rd ed.). New York: Wiley.
- Ghaziuddin, M., Leininger, L., & Tsai, L. (1995). Brief report: Thought disorder in Asperger syndrome: Comparison with high-functioning autism. *Journal of Autism and Developmental Disorders, 25*, 311–317.
- Groth-Marnat, G., & Wright, A. J. (2016). *Handbook of psychological assessment* (6th ed.). Hoboken: Wiley.
- Gurley, J. R. (2017). *Essentials of Rorschach assessment: Comprehensive system and R-PAS*. Hoboken: Wiley.
- Hogan, T. P. (2015). *Psychological testing: A practical introduction* (3rd ed.). Hoboken: Wiley.
- Holaday, M., Smith, D. A., & Sherry, A. (2000). Sentence completion tests: A review of the literature and results of a survey of members of the Society for Personality Assessment. *Journal of Personality Assessment, 74*, 371–383.
- Holaday, M., Moak, J., & Shipley, M. A. (2001). Rorschach protocols from children and adolescents with asperger's disorder. *Journal of Personality Assessment, 76*, 482–495.
- Ibanez, L. V., Stone, W. L., & Coonrod, E. E. (2014). Screening for autism in young children. In F. R. Volkmar, R. Paul, K. A. Pelphrey, & S. J. Rogers (Eds.), *Handbook of autism and pervasive developmental disorders, assessment, interventions, policy, the future: Assessment, interventions, and policy* (4th ed., pp. 585–608). Hoboken: Wiley.
- Jenkins, S. R. (Ed.). (2008). *A handbook of clinical scoring systems for thematic apperceptive techniques*. New York: Lawrence Erlbaum Associates.
- Kenworthy, L. (2010). Asperger disorder. In J. E. Morgan, I. S. Baron, & J. H. Ricker (Eds.), *Casebook of clinical neuropsychology*. New York: Oxford University Press.
- Klin, A., Saulnier, C., Tsatsanis, K., & Volkmar, F. R. (2005). Clinical evaluation in autism spectrum disorders: Psychological assessment within a transdisciplinary framework. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders, vol 2: Assessment, interventions, and policy* (3rd ed., pp. 772–798). New York: Wiley.
- Krishnamurthy, R., Archer, R. P., & Groth-Marnat, G. (2013). The Rorschach and performance-based assessment. In T. M. Harwood, L. E. Beutler, & G. Groth-Marnat (Eds.), *Integrative assessment of adult personality* (3rd ed.). New York: Guilford.
- Lee, A., & Hobson, R. P. (2006). Drawing self and others: How do children with autism differ from those with learning disabilities. *British Journal of Developmental Psychology, 24*, 547–565.
- Lim, H. K., & Slaughter, V. (2008). Brief report: Human figure drawings by children with Asperger's syndrome. *Journal of Autism and Developmental Disorders, 38*, 988–994.
- Lord, C., Corsello, C., & Grzadzinski, R. (2014). Diagnostic instruments in autistic spectrum disorders. In F. R. Volkmar, R. Paul, K. A. Pelphrey, & S. J. Rogers (Eds.), *Handbook of autism and pervasive developmental disorders, assessment, interventions, policy, the future: Assessment, interventions, and policy* (4th ed., pp. 609–660). Hoboken: Wiley.
- Machover, K. (1949). *Personality projection in the drawing of the human figure*. Springfield: Charles C. Thomas.
- Mihura, J. L., & Meyer, G. J. (Eds.). (2018). *Using the Rorschach performance assessment system (R-PAS)*. New York: Guilford.
- Mihura, J. L., Meyer, G. J., Dumitrascu, N., & Bombel, G. (2013). The validity of individual Rorschach variables: Systematic reviews and meta-analyses of the

- comprehensive system. *Psychological Bulletin*, 139(3), 548–605.
- Murray, H. A. (1943). *Thematic apperception test*. Cambridge, MA: Harvard University Press.
- Naglieri, J. A. (1988). *Draw a person: A quantitative scoring system*. San Antonio: Psychological Corporation.
- Naglieri, J. A., McNeish, T. J., & Bardos, A. N. (1991). *Draw a person: Screening procedure for emotional disturbance*. San Antonio: Psychological Corporation.
- Roberts, G. E., & Gruber, C. (2005). *Roberts-2 manual*. Los Angeles: Western Psychological Services.
- Roman, P. (2005). La Mallette Projective. Première Enfance (M.P.P.E): un outil clinique pour l'évaluation de la personnalité du jeune enfant. *Devenir*, 17(3), 233–259.
- Roman, P., Dublneau, M., & Saboia, C. (2011). Projective kit for early childhood (P. K. E. C.): A projective tool for research and clinical assessment. *Rorschachiana*, 32(2), 223–251.
- Rorschach, H. (1921). *Psychodiagnostik*. Berne: Bircher.
- Rotter, J. B., Lah, M. I., & Rafferty, J. E. (1992). *Manual: Rotter incomplete sentences blank* (2nd ed.). San Antonio: Psychological Corporation.
- Sattler, J. M. (2014). *Foundations of behavioral, social, and clinical assessment of children* (6th ed.). La Mesa: Jerome M. Sattler.
- Teglasi, H. (2010). *Essentials of TAT and other storytelling assessments* (2nd ed.). Hoboken: Wiley.
- Torstrick, A., McDermut, W., Gokberk, A., Bivona, T., & Walton, K. E. (2015). Associations between the Rotter Incomplete Sentences Blank and measures of personality and psychopathology. *Journal of Personality Assessment*, 97(5), 494–505.
- Weiner, I. B., & Greene, R. L. (2017). *Handbook of personality assessment* (2nd ed.). Hoboken: Wiley.
- Weis, R., Toolis, E. E., & Cerankosky, B. C. (2008). Construct validity of the Rotter Incomplete Sentences Blank with clinic-referred and nonreferred adolescents. *Journal of Personality Assessment*, 90(6), 564–573.
- Wood, J. M., Garb, H. N., Nezworski, M. T., Lilienfeld, S. O., & Duke, M. C. (2015). A second look at the validity of widely used Rorschach indices: Comment on Mihura, Meyer, Dumitrascu, and Bombel (2013). *Psychological Bulletin*, 141(1), 236–249.
- Yalof, J. (2006). Case illustration of a boy with nonverbal learning disorder and Asperger's features: Neuropsychological and personality assessment. *Journal of Personality Assessment*, 87, 15–34.
- Zeman, J., Klimes-Dougan, B., Cassano, M., & Adrian, M. (2007). Measurement issues in emotion research with children and adolescents. *Clinical Psychology: Science and Practice*, 14, 377–401.

Projective Tests of Personality

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Abbreviations

CAT	Children's apperception test
DAF	Draw-a-family
DAM	Draw-a-man
DAP	Draw-a-person
HTP	House-tree-person
RATC	Roberts apperception test for children
SCT	Sentence completion test
TAT	Thematic apperception test

Synonyms

[Projective tests](#); [Projectives](#)

Description

Comment

This is only a partial list of the projective instruments that might be administered, and projectives are only one kind of personality test which might be utilized in psychological evaluation (Adams and Culbertson 2009). And while the projective tests are described separately here, they are commonly given along with other psychological instruments to describe cognitive and processing skills as well as social and emotional functioning. In addition, psychological testing is commonly supplemented by a good deal of other information, including different kinds of rating scales, in order to make diagnostic as well as descriptive comments. The projective tests are given to provide information to professionals and parents regarding the “inner workings” of complex patients, and to help them make diagnoses and

Projective Tests

► [Projective Tests of Personality](#)

design interventions in complicated cases, including autism. The actual diagnosis of Autism Spectrum Disorder (ASD) is made according to strict historical and behavioral criteria described in the Diagnostic and Statistical Manual of Mental Disorders (DSM). All the same, projective testing can be useful in supporting the diagnosis of autism or exploring alternative or additional diagnoses. Regardless of diagnosis, projective testing can be helpful in describing individual functioning, thereby enhancing the process of making recommendations for any specific client, including those with autism.

Overview

There are times when parents and professionals ask “Why?” a child is responding as they do or “What?” is going on. Beyond asking for a label to be applied to their youngster, they are asking for a comprehensive description of their functioning and recommendations to make things better. In these cases, projectives may enhance the overall evaluation of an individual with autism.

“Projective tests” refer to a range of psychological instruments focused on the assessment of personality and personal functioning. There is no real “right” answer. Instead, it is anticipated that the patients will “project” their own thoughts and feelings about themselves and life onto the external world. These projective tasks can include describing the inner life of an imaginary individual, completing a variety of sentences about life, telling stories about common and uncommon situations, and saying what amorphous inkblots look like.

Although the focus of interpretation of the tests is often on personality issues, the projective tests provide opportunities to assess a range of interacting and overlapping cognitive/processing and social/emotional functions. It is anticipated that the answers on projectives will reveal something about the unique style and perspective of each client. While in general practice projective tests are often undertaken to help clarify psychiatric diagnosis, in the context of autism, they are more often performed to describe psychological and interpersonal

functioning and to help individualize understanding and therefore intervention.

Projective assessment can enrich the evaluation of an individual with autism by focusing on the challenges they face from their own point of view and by helping professionals to be specific about intervention strategies. This allows mental health specialists to move beyond “making referrals” to professionals or even treatments; it helps parents and professionals to interact with patients and intervene around difficulties in a more fine-tuned way.

While there is no set agenda or inquiry, questions about patients might include the following: Do they function better in some situations than others? What kinds of situations or stressors seem to throw them? Is the problem more with how they understand things or with how they regulate emotion? What should professionals be aware of as they try to intervene in clinical and treatment settings? What can caretakers do to support and teach them so that they can be more adaptive on an ongoing basis, including at home and in school? Or, does the data suggest some diagnosis in addition to (or other than) autism which the treatment team should be aware of?

Projective testing can provide insights around individuals with autism which can help caretakers care for them better.

Instruments

The projective instruments utilized can include the full range of projective tests used with children, adolescents, and adults. What is appropriate will depend on a number of developmental features, including the intellectual level and language facility of any patient. Because psychological evaluation around autism more commonly involves children and adolescents than adults, the following discussion reflects this fact. While the use of projective testing with individuals with possible autism is not routine, when it is done, it frequently involves a battery of tests that fairly systematically assesses different ways/levels of organizing (structuring) information as they interact with different kinds of input (visual and

auditory) and output (motor and verbal). While the psychologist is interested in how the patient conceptualizes their social relationships and experiences their emotional life, he or she is also interested in the cognitive and processing infrastructure (underpinnings) which facilitates or impedes adaptive and appropriate social and emotional functioning (Jura and Humphrey 2017). Particularly in the case of developmental disabilities, the psychologist considers whether responding on a task or item is developmentally appropriate, immature, or deviant. The psychologist assesses whether any disruption is associated with intellectual challenge, skills deficit, or personal difference. The range of tasks presented involves varying demands on graphic ability (including the ability to draw a person or perhaps write a response), but particularly language facility; this can include not only receptive and expressive functions in oral and written form but also pragmatic communication skills as they are depicted or evident during testing. In analyzing responding, the psychologist tries to parse out the various intellectual, processing, social, and emotional factors that might have influenced a client's unusual test protocol. In this analysis, the psychologist remains aware of not only client responding but also test demands. Further, the psychologist tracks client behavior as well as actual answers. Some tests/items go more smoothly than others. This gives the psychologist an opportunity to observe the client's response to facility in some situations, in contrast to cognitive or social challenge in others, and perhaps ensuing frustration. Below, the tests will be described in the order in which they might be administered (basically from most to least structured, with increasing ambiguity and leeway over the session/s). Included in the descriptions are comments about the test characteristics and their utility. While this battery of tests is most likely to be administered during the assessment of autism, the same instruments might be given to an individual who has been diagnosed with autism, but professionals feel the situation is particularly complex and in need of clarification. The interpretation of any projective results is based on the context of the assessment and any guidelines associated with each

instrument, as well as the clinical experience of the examining psychologist. Based on the referral issues and goals of the assessment, the examiner may vary the test administration and therefore the analysis, in order to enhance the usefulness of the evaluation as a whole to any particular patient. What follows is a list of the types of projective instruments most often used in exploring autism.

Figure Drawings

On the *DAP*, the examiner asks the client to "Draw a person." There are variations on this direction which allow the rendering to be scored for general intellectual maturity ("Draw a picture of a man, and the very best picture you can.": *DAM*). There are other variations which potentially involve members of the family ("Draw a family": *DAF*) or people and objects ("Draw a house, a tree, and a person": *HTP*). The advantage of the simple *DAP* is that subsequent to asking the patient to draw the person, the examiner can then ask a number of questions about the individual's behavior, thoughts, and emotions, "as if" this were a real person. The psychologist subsequently notes whether the figure is well drawn or not, and possible cognitive and/or psychological reasons for a limited or disorganized presentation. The examiner also notes any personal themes that are hinted at in comments about how the figure might act, think, or feel and whether the client has the general capacity to imagine the internal life of another person.

Sentence Completion

On the *SCT*, the examiner asks the patient to complete a series of incomplete sentences. There are a number of variations on this, with different sets of sentences provided for different genders and ages. The client can be asked to hear or read each sentence (e.g., asking about family, fears, peers, or dreams) and then to respond by saying or writing their response. The administration allows for adjustments around level of development and purpose of evaluation and can provide opportunities for both oral and written language samples. The products can be assessed for formal expressive competence and thought coherence, as well as personal concerns. The examiner most

commonly reviews the responses for patterns of responding which suggest the personal approach of the client toward the people around them (including parents and friends), as well as the particular hopes or conflicts which are evident around ordinary life activities.

Thematic Approaches

On the *TAT*, the psychologist asks the patient to tell stories to black-and-white pictures, commonly of people, and usually in ordinary settings. Designed for adults and children, the TAT can be given to youngsters starting at about the age of 7–8 years, depending on a number of developmental and cultural factors. Often in the case of the populations addressed here, five to ten pictures (out of set of 31) are presented. The pictures may depict children or adults by themselves or in groups, perhaps in a rural scene or within an ordinary house. Some situations are more ambiguous or fanciful. Stories are assessed for cognitive factors such as coherence, as well as social/emotional concerns. The themes are seen in basically dynamic (psychological/personality) terms. A less arousing and challenging test has been designed for children: the Children's Apperception Test. It can involve ten scenes with animals (*CAT/CAT-A*) or people (*CAT-H*). Traditionally, the approach has been seen as based on psychoanalytic theory (a theory of psychological development focusing on characteristic themes at different ages/stages). Currently, the analysis is usually based on more generic psychodynamic understandings (general themes of psychological/interpersonal functioning). It is the premise of the thematic instruments that the patient will project their own views and concerns onto the scenes and individuals depicted. For example, the scenes might show a child with a musical instrument or might suggest a parent disciplining a child. The client has a wide variety of choices regarding how they see these situations and tell stories about them. Thematic material presents opportunities for the psychologist to identify patterns of perception and relating which might characterize the client in real life. While the CAT and TAT can be looked at systematically in order to understand the patient's approach, they are not (strictly speaking or very

commonly) scored. However, another thematic test can be scored, the Roberts test/s (*RATC, Roberts-2*). In fact, because of its methodical procedures, it is denied the Roberts-2 is a "projective test" (e.g., Parolin et al. 2019). It is noted the test uses the client's expressive language as a way of measuring the child's social cognitive skills and is standardized on nonreferred children. All the same, because it involves telling stories which reveal aspects of the client's social perception and emotional approach, it is included here. Currently on the Roberts-2, 16 pictures (with parallel versions, depicting Hispanic, White, or Black characters) are presented; all pictures in a set must be provided because of scoring issues. These are the thematic instruments which are most likely to be seen in the evaluation of a range of clients, including those with possible autism, although there are other tests available which might potentially be utilized.

Unstructured Material

On the *Rorschach*, the examiner asks the patient to view ten simple to complex inkblot designs; they are commonly black but sometimes involve red or pastel colors. While this instrument theoretically can be given to younger children (down to 5 years), it is typically administered to individuals about 6–7 years and up. Because of the potential for scoring, it is given according to strict directions and rules, and responses are recorded verbatim for later scoring and analysis. On each blot, the examiner asks the client what the inkblots look like to them ("What might this be?"). This is a test of perception, where the patient first gives one to several responses to each blot and then is asked to explain or justify their percept. The responses can subsequently be scored for factors based on appropriateness of the response (goodness of fit of percept to blot) and the determinants of the percept (e.g., the shape or color of the design), as well as other features of the response (including thought process or subject matter). Different scoring systems have been utilized over the years. According to the more current Exner comprehensive system, the pattern of responding is assessed according to formal response characteristics, importantly providing

information around cognitive integration (the ability to pull together information) as well as social and emotional functioning. While the Rorschach can be scored, some psychologists under some circumstances may interpret the client's responding without formally scoring the performance, but with knowledge of what scoring might mean to the individual's psychological functioning. From the Rorschach, information can be provided regarding the client's reality testing as well as accessibility to human experience, including relationships and emotions. Data can also be provided around the subject's ability to deal with stress and their capacity for disorganization. For example, the psychologist commonly looks at the pattern of responding to learn how much structure the client requires to see the world as others do or to get a feel for how susceptible the client is to dysphoric affect (anxiety/depression).

Interpretation

While comments might be made about the significance of responding on individual tests, the test analysis is usually based on the battery as a whole.

Pattern

It is the assumption of the psychologist that the client's responding on these tasks reflects the ways they habitually perceive situations and organize their experience and, therefore, suggests their typical thoughts, concerns, and approach. No one instrument, or response on any instrument, is definitive. It is the pattern of responding which suggests the way a patient is apt to handle situations and conflicts and to cope in life. Further, the pattern can be understood in cognitive/processing as well as social/emotional terms and levels, with implications for adaptive functioning. For example, the instruments used in a battery of tests can require increasing verbal and conceptual skills and thus have increasing potential for revealing how the client understands their experience. In this context, a patient may have the skill or motivation to provide the short response required on the DAP or SCT, but not to tell or write a more extensive response to the TAT. Further, the SCT may provide only a glimpse of the concerns reflected in more elaborate form on the TAT or

on the Rorschach. When the psychologist considers both cognitive/processing and social/emotional reasons for disruption, projective testing can help parents and professionals to better understand "why" the child proceeds as they do in life and help the treatment team to be more specific about the kinds of strategies which might benefit the patient.

Utility

Even when the projective instruments are scored, there is a large discrepancy between the information to be gleaned and any diagnosis to be made. And regardless of scoring, interpretation of projective tests can remain largely subjective. Further, most diagnoses are made based on psychiatric history and current presentation. Projective tests can only be viewed as consistent with the diagnoses being considered, and not as definitive of any particular diagnosis. Projectives provide only one (or several) data point/s among many in the process of assessing any child, adolescent, or adult. This is particularly true in the case of autism, where the diagnosis is made according to criteria falling within specific spheres and is based on history and behavior. All the same, when the patient cannot speculate about what the DAP figure is doing, thinking, or feeling; has difficulty telling a coherent story on the TAT/CAT which includes human motivations or interpersonal interactions; and struggles to identify ambiguous inkblot designs on the Rorschach in the most ordinary ways, one has a richer understanding of how the client approaches life and how challenging he or she finds it. To a certain extent, projective testing can be an assessment of severity – the performance suggests to what extent the client has difficulty making sense of their experience and therefore responding appropriately. Taken together, the test instruments can reflect difficulties with information processing which are seen on intellectual tasks (perhaps tasks which require creating a cartoon narrative or assembling a schematic puzzle) and also on projective instruments (which challenge the client to "make meaning" of a variety of scenes or blots). Together, the projective and cognitive tests can provide a fair assessment of the individual's

ability to understand their experience and deal with the passing social scene. They can help to identify specific difficulties that suggest “what” to do and target interventions in the context of the treatment plan.

Appropriateness

It needs to be noted that this discussion about the use of projective tests in assessing autism is being undertaken at a time when there are doubts about the validity of projective tests in the diagnosis and evaluation of psychological problems and psychiatric illness at all. In this context, a distinction needs to be made between clinical description and clinical diagnosis. Clinical diagnosis according to the Diagnostic and Statistical Manual of Mental Disorders (DSM) tends to be made based on a constellation of criteria, noted in history and presentation. In the case of some diagnoses, while some intellectual or achievement testing might be relevant (and rating scales might be helpful), even in the case of more emotional disorders, projective testing tends not to be necessary. Clinical description is another matter. While not necessary, projectives might be very relevant in assessing affect, thinking, and relationships in a variety of diagnostic contexts. This can include autism, although the situation is complex. In the case of a developmental disorder like autism, it is legitimate to ask whether it is appropriate to use projective tests which have been developed, understood, and in some cases normed, based on neurotypical populations. It needs to be emphasized that projectives may not be relevant/necessary to making the diagnosis of autism, but they may be very useful in assessing the autistic individual, so long as developmental issues are taken into account. In diagnosing and evaluating autism, prudent practice can involve a range of specific assessments (e.g., adaptive functioning or communication skills), which further help to spell out the affected individual’s strengths and weaknesses and to identify areas for intervention. As an extension of this evaluative and descriptive process, projective assessment can help to clarify the individual’s functional profile, either as part of an original assessment or as part of a subsequent evaluation. To effectively

intervene, it is not sufficient to label the patient as “autistic.” The individual is not simply autistic – he or she may manifest their autism in personal as well as characteristic ways – and they may be responding to their own unique history in the context of their developmental disability. In a “comprehensive” evaluation of a complex patient, their unique presentation needs to be understood in order to intervene effectively. Projective assessment can help with this.

Historical Background

Projective testing was developed over the last century for use with adults and subsequently adapted for children. Over time, it has become apparent that the testing technique can help in the understanding of children with atypical as well as typical development, including individuals with autism.

Projective Technique

Any projective test is based on the projective hypothesis: given an ambiguous stimulus with no specific correct answer, the subject’s response will reflect their personal interests and concerns. The most commonly used projective instruments were actually designed for adults, with versions more appropriate for children and adolescents being developed later. Formal projective tests date to the early 1900s, when Herman Rorschach, a Swiss psychiatrist, developed the Rorschach technique. Henry Murray, a Harvard psychologist, developed the TAT in the 1930s and 1940s (Murray 1943). The initial CAT was developed in the late 1940s, using animals rather than people as characters in human situations based on the theory that children would readily (better) identify with them. In 1965, the human version (CAT-H) was produced. In keeping with the time in which it was developed, the Children’s Apperception Test scenes pulled for specific issues psychoanalytically conceived. Currently, the pictures are seen according to more general interpersonal and developmental frameworks. The RATC has followed from the early 1980s, providing a more modern appearance and effective scoring. The

Roberts-2 published in 2005 involved the updating of pictures and norms. The test approaches focusing on figure drawing were developed over the last century. Early on, Goodenough (1926) designed a system for scoring a male figure for intellectual maturity on the DAM. Over time, and especially since the 1940s, figure drawings have been of more interest as a projective technique (Koppitz 1968) than as a cognitive test (Harris 1963). Elaborations have included the DAP (where the individual is free to draw either sex or any age), DAF (where the drawing need not be the client's family), and HTP (where the objects requested are drawn on three pages, or as a variation, one page). The drawings might be supplemented by questions posed to the client. Over time, many versions of the SCT have been developed for different purposes, not only based on age and gender but also clinical population. In general, interpretation of projectives is based on the totality of the situation, including the patient's demeanor as well as what they saw, drew, or said. While early on projective interpretations may have been filtered through psychoanalytic understandings, currently interpretation typically involves more general, dynamic, and cognitive approaches. It should be noted that in a reciprocal development around thinking (and particularly relevant to autism), some psychological tests/tasks address social cognition or perception (e.g., subtests on theory of mind and affect recognition on the NEPSY-II) (Jura and Humphrey 2017).

Research Data

Over the years, and even early on, there has been considerable debate about the value of projective techniques in psychological research and clinical practice (Blatt 1975), with particular focus on the Rorschach and its scoring (Wood et al. 2001). It is not possible to review or evaluate the extensive literature here, beyond noting that projectives are the subject of controversy. However, as an example of this debate, a range of projective techniques and their scoring (Rorschach and TAT, as well as figure drawings such as the DAP) have been attacked (Lilienfeld et al. 2000) and defended (Hibbard 2003). The debate about projectives

has raged over the years, with a focus on the valid and ethical use of personality testing in general and the Rorschach in particular (Board of Trustees of the Society for Personality Assessment 2005). However, psychologists have continued to use projective tests in clinical practice and have continued to explore their usefulness in research. Some of this research has been done around autism, and much of the research around autism has involved the Rorschach. This is probably because more than other projective tests, the Rorschach can be scored, allowing measurements to be compared across groups. Some of the research has involved the similarities and differences: (1) between autistic individuals and controls with similar intellectual levels (Ishisaka et al. 1997), (2) between students with Asperger's Disorder and other emotional or behavioral problems (Holaday et al. 2001), (3) between individuals with Asperger's Disorder and "high-functioning autism" (Ghaziuddin et al. 1995; Nihei and Nihei 2008), as well as (4) between adults and older adolescents with "high-functioning autism" and a reference group with schizophrenia (Dyken et al. 1991). Because this list is not exhaustive and these studies tend to involve small numbers and focused issues, no attempt is made to review the findings here. However, taking the data in their entirety, they suggest individuals with ASD compared to controls have difficulties with cognitive integration as well as coping deficits, less interpersonal responsiveness, and more thinking problems. It is worth mentioning that in one study (Dyken et al. 1991), the schizophrenia index score was elevated among individuals with "high-functioning autism," although there was no reason (based on history or symptoms) to believe this was their diagnosis. In this study, lower PIQ scores were related to greater perceptual distortions. These data argue for the necessity of taking all information into account in assessing any case, but particularly around issues of autism. These studies also suggest that larger and more complete comparative studies around Rorschach scoring may provide useful information in understanding many diagnostic groups, including individuals with autism. However, regardless of the usefulness of the Rorschach (or other projective

tasks) in distinguishing autism from other conditions (normative or diagnostic), projective techniques can still be useful in (a) helping to understand complications of individual cases of autism, and (b) this understanding can involve identifying comorbid conditions, such as depressed or disorganized psychological states. For example, the Rorschach may be useful in assessing disorganized thinking in “higher-functioning autistic individuals” (Klin et al. 2005). The use of the Rorschach is described in specific cases of potential autism: including that of a complex youngster in a community setting to help clarify diagnostic issues (Fogler et al. 2019) and that of young adult to better understand possible symptom overlap (Frigaux et al. 2018). It should be noted that despite continuing controversy around the use of projectives in general and the Rorschach in particular, interest remains in the use of this instrument not just for descriptive but for diagnostic purposes in individuals along the autism spectrum (Crucitti et al. 2015; Kishimoto et al. 2016). Kleiger (2017) reviews some data, research, and concepts potentially relevant to Rorschach responding in individuals along the spectrum. However, the complications introduced by the diagnostic complexity of this spectrum disorder, along with small sample sizes focused on particular diagnostic issues, make assessing the utility of projective tests in autism difficult. As a result, projectives are apt to be most useful for descriptive rather than diagnostic purposes in autism for the foreseeable future.

Psychometric Data

Most projective tests are not literally scored. While some tests may claim to “measure” aspects of personality, often this involves systematic notations and analysis, and perhaps some coding (e.g., the CAT), rather than actual scoring. The notable exceptions are the Rorschach (Exner 2001) and the Roberts (Roberts and Gruber 2005). However, because the instruments involve many scales and scores, the issues of reliability and validity are particularly complex. As a result, psychologists should exercise some care in using these

instruments, particularly with unusual populations, such as individuals with autism. Just to add to the complications, readers should be aware that new instruments are constantly being introduced and old instruments (including scoring) updated.

Rorschach Method

Psychologists are probably most familiar with Exner’s Rorschach Comprehensive System (RCS), formulated in 1974. According to this system, each response to every blot is scored according to response location, quality, determinants, contents, organization, and special scores. All scores across the ten blots are tallied and compared to normative samples. Ultimately, any interpretation might reflect the patient’s capacity to think in an organized way, their psychological approach around personal concerns and the human sphere, in addition to their basic reality testing. Based on the response scores, a structural summary is compiled which reflects different aspects of the overall performance. These data are used to formulate many interpretive postulates concerning psychological characteristics and functioning. Once the measurements are obtained (which can appear as frequencies, ratios, or percentiles and can include derived scores and special indices), they are compared to tables of descriptive statistics organized by different factors, most importantly age. It is a complex system. Fortunately, the results can be analyzed by computer. Because of the complexity of the scoring and analysis, some clinicians (perhaps especially those seeing particular child populations) may at times forgo formal scoring. They do this with full knowledge of Rorschach notation and interpretation, as well as relevant clinical experience. They use the Rorschach as part of a battery of tests, not so much to support a diagnosis as to enrich their psychological understanding of the individual client. Readers should be aware that since Exner’s death in 2006, there have been ongoing efforts to sustain his scoring system, and there is a computer scoring program for it (Rorschach Interpretation Assistance Program: Version 5 [RIAP5]). However, some professionals have begun to develop an alternative system. Readers should know that

Exner's RCS (Rorschach Comprehensive System) has been revised and reformulated in an essentially overlapping and coexisting but separate scoring system designated R-PAS (Rorschach Performance Assessment System, manual published 2011). R-PAS has been developed by a group which includes professionals who worked on Exner's earlier research council, and who believe R-PAS significantly extends the work undertaken by that previous group. Both systems are evidence-based, with the main validating evidence for the Rorschach coming from research based on the RCS (Mihura et al. 2013). Both systems have access to international norms (Composite International Reference Values, or CIRVs) based on the RCS (Meyer et al. 2007). However, it should be noted the adult samples from around the world are more stable than the child and adolescent samples. An online feature of R-PAS may allow for updating norms and data as new information becomes available. While there are currently no data speaking to the utility of R-PAS in the assessment of autism, it might be expected to perform similarly to the Exner RCS and require a good deal of clinical experience and judgement to be helpful.

Roberts Test

On the Roberts (RATC, Roberts-2), each story is scored separately on a number of rating categories. Check marks are made indicating that certain psychological factors (on developmental/adaptive and clinical scales) have been present in the stories. These data are summed and analyzed. Profiles (according to age group) are plotted based on these scores, reflecting, for example, reliance on others, basic human emotions, or even atypical responding. Together, the data are thought to reflect the child's feelings and reactions in the interpersonal sphere. The Roberts describes ways for both quantitative analysis (using T-scores) and qualitative interpretation (using clinical judgment). The manual provides descriptive statistics, as well as comments on reliability and validity studies. Because of the scoring, repeat testing can be particularly informative in assessing progress over time. It is acknowledged that only very general clinical inferences can be

made and that clinicians must use skill and judgment in interpreting results.

Other Instruments

It is worth noting that other projective instruments might be subject to scoring or systematic analysis. For example, the DAM can be scored for intellectual maturity. And Rotter et al. (1992) have published scorable version of the SCT. Various scoring systems for the TAT have been developed. However, for the most part, the thematic tests have not been made amenable to strategic scoring or normative data of the sort which would allow for specific data analysis or diagnostic comparisons. All the same, professionals remain interested in more systematic approaches to projective testing, including the scoring of thematic stories (Jenkins 2008).

Other Issues

Professionals have been concerned not only about the subjective aspect of the analysis of projective data but also the lack of racial sensitivity reflected in the thematic stimuli. Some of the more recently developed instruments have provided procedures and pictures which recognize ethnic/racial/cultural diversity (including the Roberts-2). Professionals tend to assume that the disruptions in relating and communication in the case of autism are so basic that they transcend racial/ethnic considerations. All the same, it would always be prudent to consider whether cultural tensions have exacerbated or distorted social issues as they present in testing and in life.

General Comment

It is often lamented that normative (objective) data are not provided for all projective instruments. However, even when such normative data are available, one still needs to ponder their use. Norms are usually provided on large national samples of typical children and adults. While such general normative data might facilitate the comparison of a client to population norms, when their data deviate from that of "typical" individuals, one still needs to account for differences. Differences might be associated with autism, or any number of other possibilities, all of which need to be considered. Further, data describing diagnostic groups or their psychological features

are not easily accessible, including data for autism. Generally speaking, even when features of a diagnostic group might be identified (and even through scoring), the final diagnostic determination is not necessarily clear. It is still up to the clinician to make a decision whether a client meets criteria for a specific diagnosis or not, based on the entirety of the available data. The diagnostic process involving projectives remains largely descriptive and supportive – perhaps particularly in the case of children and adolescents, and especially in the case of autism.

Clinical Uses

In the context of a comprehensive assessment of autism, projectives are potentially a useful addition. However, much needs to be considered.

Diagnostic Clarity: It is worth reiterating that projective testing may supplement and enrich the understanding of clients, including those along the autistic spectrum, but it may not always be directly relevant to the making of an actual diagnosis. All the same, the profile suggested by projective testing can be seen as consistent with a diagnosis of autism or perhaps can suggest some other (alternative or comorbid) condition which needs further evaluation. These comorbid conditions can include psychosis, depression, or anxiety, all of which may require medical as well as behavioral interventions (e.g., cognitive behavioral therapy). Testing may help to identify the need for attention to particular issues within more common treatments, such as parent training or social skills group. For example, projectives can help make very real the fact the child often does not know what is going on – and how confused, distressed, and disruptive they can become as a result. In this context, caretakers may need to focus on a child's social misperceptions and/or social anxiety (along with general skills deficits) before they can expect compliant or adaptive behavior.

Complicated Situation: According to DSM, diagnoses are clinical entities and, for the most part,

are made based on criteria related to history and presentation. Autism screening and diagnostic tools have evolved over time. In this context, it needs to be acknowledged that autism rating scales may or may not detect autism, and even observations and interviews have limitations. Parent report is dependent on parent impression, and behavioral observation relies on current behavior. Ultimately, the diagnosis must be made based on a constellation of information. Projective testing provides a way of assessing the unique features of any client, regardless of diagnosis; projectives will always be relevant to the kinds of unusual cognition, social disruption, and emotional approach which are characteristic of autism.

Case Selection: While it is tempting to say that projective assessment is most likely to be relevant in the cases of “high-functioning” individuals who have the capacity to deal with the directions and responding involved in projective testing, in fact projectives are given to individuals of many ages, including young children, chronologically and/or developmentally (Jura and Sigman 1985). In determining whether a patient along the spectrum might benefit from projective testing, professionals and parents should think in terms of developmental and skill level/s, and whether the child can understand and respond to the actual tasks being proposed. Where an individual (autistic or otherwise) might be dealing with intellectual challenges, the concept of mental age also comes into the interpretative process. The psychologist needs to ask to what extent the client's mental age accounts for his or her projective performance long before they consider other diagnostic possibilities.

Beyond Diagnosis: More recently, in the assessment of diagnosis, the emphasis has been on evidence-based instruments and identifiable treatment outcomes (Ozonoff et al. 2005). At the same time, the basic theory behind best practices has been to provide as comprehensive and relevant an evaluation as practically possible in order to individualize and maximize treatment. While Klin et al. (2005) have noted that projective testing may typically be

more optional than other assessments in autism, they identify projective approaches as useful with some patients. Particularly based on the battery of projectives described here, the psychologist can comment on an individual's cognitive comprehension, interpersonal style, and personal issues as they impact adjustment. For example, projective testing can help to focus caretakers on issues of pressure, and the need to increase structure and reduce stress. In general, projective testing can provide an opportunity for the adults to begin to comprehend a youngster in his or her own terms and to become sympathetic with their child's dilemma given how they understand the world in which they live. Projectives can help caretakers as well as patients themselves to make better sense of their experience. Sometimes, simply knowing what has been going on in a more specific cognitive, social, and/or emotional way allows caretakers and patients to respond differently and more effectively. Sometimes, very specific features of the testing can prompt further evaluation or specific intervention. At times in extreme cases, particularly where medication is involved, projective testing can be repeated after an appropriate interval of time in order to document whether progress is being made.

It needs to be acknowledged that autism is a complex and heterogeneous condition involving social and communication impairments and deficits, as well as restricted, repetitive patterns of interests, behaviors, or activities. Much needs to be done beyond diagnosing autism in some general way. A full workup often involves a number of assessments (including intellectual and academic achievement tests) in addition to specific evaluations (e.g., with a speech/language pathologist or occupational therapist or neuropsychologist). Certain medical consultations and procedures may also be in order. Any psychological testing, projective or otherwise, is deeply embedded in an elaborate evaluation process in order to assess the needs of any individual with autism and to make appropriate behavioral and medical recommendations.

See Also

- [Human Figure Drawing Tests](#)
- [Norm-Referenced Assessment](#)
- [Perceptual Development](#)
- [Personality, Clinical Assessment](#)
- [Psychological Assessment](#)

References and Reading

- Adams, R., & Culbertson, J. (2009). Personality assessment: Adults and children. In B. Sadock, V. Sadock, & P. Ruiz (Eds.), *Comprehensive textbook of psychiatry* (Vol. 1, 9th ed., pp. 951–973). New York: Lippincott, Williams & Wilkins.
- Blatt, S. J. (1975). Validity of projective techniques and their research and clinical contribution. *Journal of Personality Assessment*, 39(4), 327–343.
- Board of Trustees of the Society for Personality Assessment. (2005). The status of the Rorschach in clinical and forensic practice: An official statement by the Board of Trustees. *Journal of Personality Assessment*, 85(2), 219–237.
- Crucitti, M., Dritto, I. P., Di Perri, C., Gallo, G., Conti, C., Bruno, A., Muscatello, M. R. A., & Zoccali, R. A. (2015). A Rorschach investigation of autism spectrum disorders in adulthood. *Mediterranean Journal of Clinical Psychology*, 3(3).
- Dykens, E., Volkmar, F., & Glick, M. (1991). Thought disorder in high-functioning autistic adults. *Journal of Autism and Developmental Disorders*, 21(2), 291–301.
- Exner, J. (2001). *A Rorschach workbook for the comprehensive system* (5th ed.). Ashville: Rorschach Workshops.
- Fogler, J., Kuhn, J., Prock, L., Radesky, J., & Gonzalez-Heydrich, J. (2019). Diagnostic uncertainty in a complex young man: Autism versus psychosis. *Journal of Developmental & Behavioral Pediatrics*, 40, 72–74.
- Frigaux, A., Evrard, R., & Demogeot, N. (2018). Au carrefour des spectres: problèmes de diagnostic différentiel entre autisme et schizotypie, autour du cas d'un jeune adulte. *L'Évolution psychiatrique*, 83(1), 161–181.
- Ghaziuddin, M., Leininger, L., & Tsai, L. (1995). Thought disorder in Asperger syndrome: Comparison with high-functioning autism. *Journal of Autism & Developmental Disorders*, 25(3), 311–317.
- Goodenough, F. (1926). *Measurement of intelligence by drawings*. New York: World Book Company.
- Harris, G. (1963). *Children's drawings as measures of intellectual maturity*. New York: Harcourt, Brace & World.
- Hibbard, S. (2003). A critique of Lilienfeld et al.'s (2000) "The scientific status of projective techniques". *Journal of Personality Assessment*, 80(3), 260–271.

- Holaday, M., Moak, J., & Shipley, M. A. (2001). Rorschach protocols from children and adolescents with Asperger's disorder. *Journal of Personality Assessment*, 76(3), 482–495.
- Ishisaka, Y., Murasawa, T., Muramatsu, Y., Kamio, Y., & Toichi, M. (1997). Cognitive deficits of autism based on results of three psychological tests. *Japanese Journal of Child and Adolescent Psychiatry*, 38(3), 230–246.
- Jenkins, S. R. (Ed.). (2008). *A handbook of clinical scoring systems for thematic apperception techniques*. New York: Taylor and Francis Group.
- Jura, M. B., & Humphrey, L. A. (2017). Neuropsychological and cognitive assessment of children. In B. J. Sadock, V. A. Sadock, & P. Ruiz (Eds.), *Kaplan & Sadock's comprehensive textbook of psychiatry* (10th ed., pp. 3413–3436). Philadelphia: Wolters Kluwer.
- Jura, M., & Sigman, M. (1985). Evaluation of emotional disorders using projective techniques with mentally retarded children. In M. Sigman (Ed.), *Children with emotional disorders and developmental disabilities* (pp. 229–248). Orlando: Grune & Stratton.
- Kishimoto, N., Yamamuro, K., Iida, J., Ota, T., Tanaka, S., Kyo, M., Kimoto, S., & Kishimoto, T. (2016). Distinctive Rorschach profiles of young adults with schizophrenia and autism spectrum disorder. *Neuropsychiatric Disease and Treatment*, 12, 2403–2410.
- Kleiger, J. H. (2017). *Rorschach assessment of psychotic phenomena: Clinical, conceptual, and empirical developments*. London/New York: Routledge.
- Klin, A., Saulnier, C., Tsatsanis, K., & Volkmar, F. R. (2005). Clinical evaluation in autism spectrum disorders: Psychological assessment within a transdisciplinary framework. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and developmental disorders* (Vol. 2, 3rd ed., pp. 772–798). Hoboken: Wiley.
- Koppitz, E. M. (1968). *Psychological evaluation of children's human figure drawings*. New York: Grune & Stratton.
- Lilienfeld, S. O., Wood, J. M., & Garb, H. N. (2000). The scientific status of projective techniques. *Psychological Science in the Public Interest*, 1, 27–66.
- Meyer, G., Erdberg, P., & Shaffer, T. (2007). Toward international normative reference data for the comprehensive system. *Journal of Personality Assessment*, 89 (Suppl 1), S201–S216.
- Mihura, J., Meyer, G., Dumitrascu, N., & Bombel, G. (2013). The validity of individual Rorschach variables: Systematic reviews and meta-analyses of the comprehensive system. *Psychological Bulletin*, 139(3), 548–605.
- Murray, H. A. (1943). *Thematic apperception test*. Cambridge, MA: Harvard University Press.
- Nihei, S., & Nihei, Y. (2008). Contrasting Rorschach test results in Asperger's syndrome and high-functioning autism. *Tohoku Psychologica Folia*, 67, 6–9.
- Ozonoff, S., Goodlin-Jones, B. L., & Solomon, M. (2005). Evidence-based assessment of autism spectrum disorders in children and adolescents. *Journal of Clinical Child and Adolescents*, 34, 523–540.
- Parolin, L., De Carli, P., & Locati, F. (2019). The Roberts-2: Italian validation on a sample of children and adolescents. *Journal of Personality Assessment*, 1–15. <https://doi.org/10.1080/00223891.2018.1546713>.
- Roberts, G. E., & Gruber, C. (2005). *Roberts-2 (manual)*. Los Angeles: Western Psychological Services.
- Rotter, J. B., Lah, M. I., & Rafferty, J. E. (1992). *Rotter incomplete sentences, manual* (2nd ed.). New York: Psychological Corporation.
- Wood, J. M., Nezworski, M. T., Garb, H. N., & Lilienfeld, S. O. (2001). The misperception of psychopathology: Problems with the norms of the comprehensive system for the Rorschach. *Clinical Psychology: Science and Practice*, 8(3), 350–373.

Projectives

► Projective Tests of Personality

Proline-Rich Synapse-Associated Protein 2

► SHANK 3

ProM

► Prospective Memory in Autism Spectrum Disorder

Prompt Dependence

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Definition

Prompt dependence refers to a situation in which a child or adult does not perform a behavior until they are prompted. Prompt dependency is a common problem for individuals with autism spectrum

disorders (ASDs). Individuals with ASDs frequently only perform skills and behaviors they have acquired when asked to do so by an adult, regardless of whether or not natural prompts are present. For example, you might have a child that knows all of the steps of how to brush their teeth independently, but needs an adult to prompt them before beginning each step. Because prompt dependency is undesirable (we want to teach individuals to perform behaviors independently), it is essential to systematically fade prompts and thin reinforcement schedules to ensure that stimulus control is transferred to naturally occurring stimuli and reinforcers.

See Also

- [Fading](#)
- [Prompt Fading](#)
- [Prompts](#)

References and Reading

McClannahan, L. E., & Krantz, P. J. (1997). In search of solutions to prompt dependence: Teaching children with autism to use photographic activity schedules. In E. M. Pinkston & D. M. Baer (Eds.), *Environment and behavior* (pp. 271–278). Austin: PRO-ED.

from a physical prompt, a gesture, a verbal prompt, or no prompt at all where the individual is able to complete a task independently. A physical prompt can be lightly guiding the child into an activity or a more intense prompt where the child is completely guided in a hand over hand. A verbal prompt also has a range of cues, such as telling the child what to do or say, or just reminding the child, but not directly telling them what is expected. Prompts should be systematically decreased until the child is able to independently complete activities, so that the child does not become prompt dependent (McDonnell and Ferguson 1989).

References and Reading

- Hourcade, J. J. (1988). Effectiveness of gestural and physical guidance prompts as a function of type of task. *Education and Training in Mental Retardation*, 23(1), 38–42.
- McDonnell, J. (1987). The effects of time delay and increasing prompt hierarchy strategies on the acquisition of purchasing skills by students with severe handicaps. *The Journal of the Association for Persons With Severe Handicaps*, 12(3), 227–236.
- McDonnell, J., & Ferguson, B. (1989). A comparison of time delay and decreasing prompt hierarchy strategies in teaching banking skills to students with moderate handicaps. *Journal of Applied Behavior Analysis*, 22, 85–91.

Prompt Fading

- [Fading](#)

Prompt Hierarchy

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Definition

A prompt hierarchy is the range of cues necessary for an individual to complete a task. It can range

PROMPT System

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Definition

Prompts for Restructuring Oral Muscular Phonetic Targets (PROMPT; Hayden-Chumpelik, 1984) is a speech therapy technique that utilizes tactile, visual, and positioning cues through manipulation of the tongue, lips, and jaw as well as other structures responsible for speech. The PROMPT

technique is done by PROMPT-certified speech-language pathologists who physically guide the speech structures to elicit typical movement patterns for more intelligible speech. For children with ASD who are sometimes thought to have difficulty with motor-speech production (e.g., apraxia), the use of PROMPTs is hypothesized to improve control over the speech structures, thereby enhancing functional communication skills.

Historical Background

PROMPT was first developed in the late 1970s for children who exhibited difficulties with motor-speech production and who were not responding to traditional therapies. The PROMPT technique stemmed from work done in the fields of neurobiology, cognitive-linguistics, and social aspects which form the basis of this approach. The focus of PROMPT treatment has recently shifted from emphasis on motor placement and planning to broader social-communicative and language goals. However, no empirical support for these broader goals has as yet been provided.

Rationale or Underlying Theory

PROMPT's use of tactile, visual, and positioning cues stemmed from work examining the tactile system (e.g., visual, auditory) in both normal and abnormal brains. It is based on the hypothesis that apraxic difficulties can be addressed by concentrated practice in articulatory postures and movements.

Goals and Objectives

The goal of PROMPT therapy is to improve speech intelligibility and to enhance overall functional communication skills.

Treatment Participants

Originators claim that PROMPTs can be used with infants as young as 6 months old through adulthood. Empirical evidence for this claim is lacking.

Treatment Procedures

PROMPTs are tactile, visual, and positioning cues done by a PROMPTs-certified clinician who guides a participant's speech structures through the movements necessary to produce intelligible speech. The focus is on both the sequence and transitions of movements for speech production through practice.

Efficacy Information

Little empirical evidence to currently support this therapeutic approach.

Outcome Measurement

Increased speech-sound production and intelligibility for functional communication.

Qualifications of Treatment Providers

PROMPT-trained speech-language pathologists use this therapeutic technique. In addition, there is a parent training program taught by the child's clinician with the focus on helping parents with strategies for working with their own child.

See Also

- ▶ [Articulation Disorders](#)
- ▶ [Childhood Apraxia of Speech \(CAS\)](#)
- ▶ [Dyspraxia](#)

References and Reading

- Chumpelik (Hayden), D., & Sherman, J. (1983). *Treatment comparisons for developmental apraxia of speech*. Unpublished research, Thistletown Regional Centre, Toronto.
- Paul, R., & Sutherland, D. (2005). Enhancing early language in children with autism spectrum disorders. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. II, pp. 946–1002). Hoboken: Wiley.

- Rogers, S., Hayden, D., Hepburn, S., Charlifue-Smith, R., Hall, T., & Hayes, A. (2006). Teaching young nonverbal children with autism useful speech: A pilot study of the Denver Model and PROMPT interventions. *Journal of Autism and Developmental Disorders*, 36, 1007–1024.
- Square, P. A., Chumpelik (Hayden), D., Morningstar, D., & Adams, S. G. (1986). Efficacy of the PROMPT system of therapy for the treatment of apraxia of speech: A follow-up investigation. In R. H. Brookshire (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 221–226). Minneapolis: BBK.
- The PROMPT Institute Treatment, Education, Research. (2010). History, mission & goals. In <https://promptinstitute.com/>. Retrieved October 19, 2010, from <https://promptinstitute.com/index.php?page=history-mission-goals>

Prompting

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Synonyms

Prompts

Definition

A prompt is something an adult does to help someone perform a behavior (MacDuff et al. 2001; Wolery et al. 1992). The purpose of a prompt is “to cause students to perform the target behavior so that it can be reinforced when the target stimulus is present” (Wolery et al. 1992, pp. 42–43). Prompts give someone clues about how to perform a behavior and varying levels of assistance on how to perform it (e.g., verbally telling a child to say wave good-bye, providing a motor model of waving good-bye, placing your hand behind a child’s hand and assisting them in performing the actions of waving good-bye). Prompts are not cues or task directions; cues or task directions inform the child that it is time to perform a behavior, whereas a prompt provides assistance to the child that helps them do the behavior (Wolery et al. 1992). There

are many different types of prompts that can be classified in multiple ways (Reichow and Reichow 2012; Wolery et al. 1992). One way we can describe and classify prompts is with respect to the method of presenting the prompt and how it looks. A second way to classify prompts is along a continuum of how much assistance they provide to an individual (e.g., a gestural prompt provides less assistance than a hand-over-hand prompt). A third way that we can classify prompts is with respect to the behavior of the individual being prompted – i.e., whether or not they perform the correct behavior after the prompt. Additional information on prompts and prompting can be found in the Autism Internet Module website (<http://www.autisminternetmodules.org/>) on prompting (Neitzel and Wolery 2010).

See Also

- Prompt Dependence
- Prompt Hierarchy

References and Reading

- MacDuff, G. S., Krantz, P. J., & McClannahan, L. E. (2001). Prompts and prompt-fading strategies for people with autism. In C. Maurice, G. Green, & R. M. Foxx (Eds.), *Making a difference: Behavioral intervention for autism* (pp. 37–50). Austin: Pro-Ed.
- Neitzel, J., & Wolery, M. (2010). Prompting for children and youth with autism spectrum disorders: Online training module. (Chapel Hill: National Professional Development Center on Autism Spectrum Disorders, FPG Child Development Institute, UNC-Chapel Hill). In Ohio Center for Autism and Low Incidence (OCALI), *Autism Internet Modules*, www.autisminternetmodules.org. Columbus: OCALI.
- Reichow, B., & Reichow, T. (2012). Teaching children with autism spectrum disorders: Skill acquisition and fluency. In E. E. Barton & B. Harm (Eds.), *Educating young children with autism spectrum disorders* (pp. 127–149). Thousand Oaks: Corwin Press.
- Wolery, M., Ault, M. J., & Doyle, P. M. (1992). *Teaching students with moderate to severe disabilities: Use of response prompting strategies*. New York: Longmann.

Prompts

- Prompting

Pronoun Errors

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Definition

Pronouns are noun phrases that depend on the context for interpretation. For example, first (*I, we*) and second (*you*) person pronouns are defined by the speaker and the addressee, which shifts from one situation to the next. Third person pronouns (e.g., *he, she, they, it*) are also dependent on the context and are typically used to refer to things that are salient in the discourse or environment. Because pronouns interact with many aspects of language knowledge, there are several types of potential errors.

Although grammatical errors are possible (e.g., saying *me* for *I*, or *he* for *she*), most of the literature on the language of Autism Spectrum Disorder (ASD) has focused on pragmatic pronoun errors of several types. One commonly reported error is the use of third person referential expressions (like the speaker's own name, or a pronoun *he* or *she*) instead of the pronouns *I* or *you*. Research has also shown that speakers with ASD sometimes use over-specific expressions, like a name or description when a pronoun would be understandable in context. Another type of error is the use of a pronoun when the referent is not clear in the context. Such ambiguous pronouns are not frequently attested, but have sometimes been found to occur in the speech of individuals with ASD.

An often-cited type of error is pronoun reversal, which occurs when the words *I* and *you* (or *we* and *you*) are switched. For example, Lee, Hobson, and Chiat (1994) report a child saying to his teacher "I'm better now" when the teacher had been sick. This type of error was reported by Kanner (1943) in his seminal case study and since then has been frequently cited as a language characteristic associated with autism. However, contrary to this widespread attention, pronoun reversals are neither a defining characteristic of autism nor even a frequent one (e.g., Jordan 1989; Lee et al. 1994).

See Also

- [Language](#)
- [Pragmatics](#)
- [Pronoun Reversal](#)
- [Pronoun Use](#)

References and Reading

- Arnold, J. E. (2008). Reference production: Production-internal and addressee-oriented processes. *Language and Cognitive Processes*, 23, 495–527.
- Baltaxe, C. A. M., & D'Angiola, N. (1996). Referencing skills in children with autism and specific language impairment. *European Journal of Disorders of Communication*, 31, 245–258.
- Colle, L., Baron-Cohen, S., Wheelwright, S., & van der Lely, H. K. J. (2008). Narrative discourse in adults with height-functioning autism or Asperger syndrome. *Journal of Autism Developmental Disorders*, 38, 28–40.
- Hobson, R. P., Lee, A., & Hobson, J. A. (2010). Personal pronouns and communicative engagement in autism. *Journal of Autism and Developmental Disorders*, 40, 653–664.
- Jordan, R. R. (1989). An experimental comparison of the understanding and use of speaker-addressee personal pronouns in autistic children. *British Journal of Disorders of Communication*, 24, 169–172.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Lee, A., Hobson, R. P., & Chiat, S. (1994). I, you, me and autism: An experimental study. *Journal of Autism and Developmental Disorders*, 24, 155–176.

Pronoun Reversal

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Definition

Pronoun reversal errors occur when the speaker switches the words *I* and *you*, or *we* and *you*. For example, Lee, Hobson, and Chiat (1994) report a child saying to his teacher "I'm better now" when the teacher had been sick. This type of error was reported by Kanner (1943) in his seminal case study, and since then has been frequently cited

as a language characteristic associated with autism. However, contrary to this widespread attention, pronoun reversals are neither a defining characteristic of autism, nor even a frequent one (e.g., Jordan 1989; Lee et al. 1994).

See Also

- [Language](#)
- [Pragmatics](#)
- [Pronoun Reversal](#)
- [Pronoun Use](#)

References and Reading

- Jordan, R. R. (1989). An experimental comparison of the understanding and use of speaker-addressee personal pronouns in autistic children. *British Journal of Disorders of Communication*, 24, 169–172.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Lee, A., Hobson, R. P., & Chiat, S. (1994). I, you, me and autism: An experimental study. *Journal of Autism and Developmental Disorders*, 24, 155–176.

Pronoun Use

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Definition

Pronouns (e.g., *I, you, she, he, it, they, us, me, him, her, them, this, that*) are noun phrases that typically refer to people or things that are easily understood in context. Pronouns contain very little explicit lexical information. For example, personal pronouns in English encode gender (e.g., *she* vs. *he*), number (e.g., singular *I* vs. plural *we*), and person (e.g., first person *I* vs. second person *you* vs. third person *she*). Some languages (like Spanish) use null reference forms that are absent completely (e.g., *canto* = *I sing*).

Pronouns therefore depend on the physical context, discourse context, or background information for interpretation. First and second person pronouns (*I, we, you*) change their referents depending on who is speaking. Third person pronouns (e.g., *he, they, it*) also depend on the context for appropriate usage and interpretation, and are generally considered to refer to the most salient or accessible entity in the context that matches the grammatical properties of the pronoun. This situational dependence requires pragmatic knowledge, making pronoun use an area of potential difficulty for those with Autism Spectrum Disorder (ASD).

Historical Background

In Kanner's (1943) initial description of the characteristics of autism, he noted that some children with autism used pronouns inappropriately, for example, using *you* instead of *I*. For example, his first case study, Donald, was reported to say "you did not fall down" at age 5, to describe a situation where he himself nearly fell. Since then, similar errors have been widely attested. However, despite the salience of these errors, systematic studies of pronoun use and understanding have found that pronoun reversals are neither ubiquitous nor as frequent as Kanner's focus on them would suggest (e.g., Jordan 1989; Lee et al. 1994).

Current Knowledge

Pronoun Use in Typical Speech

The ability to use pronouns correctly requires both (1) grammatical knowledge and (2) pragmatic knowledge about their function. Grammatical knowledge is required because pronouns in languages like English mark properties of the referent (e.g., person, number, gender), as well as its syntactic (i.e., structural) role, for example, using *we* in subject position and *us* in object position. Speakers use this grammatical knowledge to select the correct pronoun when speaking, for example, *he* versus *she*, and to successfully interpret pronouns during language comprehension.

Pronouns are also unusual in the degree to which their interpretation is relative to the speaker and context. This requires language users to understand the pragmatic function of pronouns in order to successfully establish reference. That is, the intended referent of a pronoun changes from one situation to the next. It is this aspect of pronouns that has received the most attention in the literature about language and ASD.

The relativity of pronouns is especially salient for *I/we* and *you*, which have different referents depending on who is speaking. But the context is also important for the appropriate use of third person pronouns, like *he*, *they*, or *it*. First, the context determines whether a pronoun is appropriate at all, as opposed to some other form of reference. For example, I could say *he*, *my cat*, *Julio*, *the brown and white cat*, or *that furry creature* to refer to the same entity. Speakers must use their pragmatic knowledge about appropriate language use to choose the best expression for a particular linguistic and social context. A too explicit expression sounds awkward and redundant (e.g., *Julio ate kibble for breakfast. Julio went outside. Julio took a nap*), whereas a too general expression is ambiguous (e.g., *My cat and my son were playing. He fell down.*) Listeners also must rely on pragmatic knowledge to interpret pronouns. All forms of reference are ambiguous at some level (e.g., there are many Julios in the world), but pronouns are especially ambiguous. One view is that appropriate production and comprehension of pronouns relies on representations of the knowledge and attention of one's interlocutor, which are related to theory of mind (e.g., Bard and Aylett 2004; Bard et al. 2000; Gundel et al. 1993). It is also well established that pronoun use is highly constrained by the discourse context, which does not necessarily require theory-of-mind representations. For example, pronouns tend to be used for reference to recently and prominently mentioned things (Arnold 2008).

Pronouns thus participate in a complex set of grammatical and pragmatic knowledge about language use. Some of this knowledge is absolute, like the fact that *I* is used for the speaker and *you* is used for the addressee. Thus, errors of pronoun

reversal can be unambiguously identified if the speaker's intended meaning is known. But much of this knowledge is gradient. For example, in the passage above about Julio, the repeated mention of his name is awkward, but not ungrammatical. As an example of a too general expression, consider a quote from Kanner (1943): "The father made a special point of mentioning that Donald even failed to pay the slightest attention to Santa Claus in full regalia" (p. 218). If *Donald* were replaced by *he*, the referent would be somewhat ambiguous, but still understandable in context. Thus, assessments of an individual's ability to use pronouns need to take into consider the range of possible alternatives.

Pronoun Use in Autism Spectrum Disorder

Individuals with ASD often use language differently than their peers. These differences can relate to pronouns and the broader system of reference in several ways. Three potential areas of difference are discussed here: (a) Pronoun reversal, (b) treating first person references as third person, and (c) the pragmatically appropriate use of pronouns within the referential system more broadly, with a focus on third person reference (e.g., *he*, *she*, *it*, *they*).

Pronoun reversal. Much attention has been given to reports that individuals with ASD sometimes make errors referring to themselves. One such error is pronoun reversal, using *I* to refer to one's addressee, and *you* to refer to oneself. Lee et al. (1994) described an incident where a boy's teacher had just returned from sick leave, and he said to her "I'm better now," and another case where a 19-year-old subject said "Thank you for seeing you, Tony" at the end of the experimental session. These utterances are notable because they are readily identified as errors. Pronoun reversal errors also attested for typically developing toddlers, but are relatively rare, and tend to disappear by around 3 years of age (Clark 1978). However, one should not conclude that these errors occur in all instances, nor even that they occur for all children with ASD.

For example, Jordan (1989) found that the children with ASD in her study had some difficulty producing, but not understanding, pronouns.

However, the most common errors were a name instead of you or I, or using the wrong case pronoun, and not the reversal of you and I. Even the three subjects (out of ten with ASD) in her study who did produce at least one reversal did not do so all the time. Similarly, Lee et al. (1994) found a few cases of pronoun reversal in their ASD group, but not their developmental delay group. However, this was not the most frequent type of pronoun error, and there were almost no errors in comprehension of personal pronouns.

Nevertheless, pronoun reversals are attested more often for individuals with ASD than other groups. Several explanations have been proposed. One view is that reversals reflect a difficulty representing one's social identity, or distinguishing oneself from others (Charney 1980; Lee et al. 1994). Another possibility is that it results from the use of echolalia, that is, the tendency to repeat others' utterances, especially the ends of utterances where "you" is more commonly found than "I" (Bartak and Rutter 1974). Other authors have suggested that the correct use of personal pronouns is related to joint attention (Loveland and Landry 1986), or attention to the linguistic exchanges of others (Oshima-Takane and Benaroya 1989).

Treating you and I as a third person. Another pronoun error often associated with ASD is the use of proper names or third person pronouns instead of *I* or *you*. This type of error emerged in Lee et al.'s (1994) study, which used questions to elicit answers that would most naturally use *you* or *I*. A frequent type of error was the use of the experimenter's name or the participant's own name instead of a pronoun. However, it is worth noting that these were equally frequent in both the ASD and the comparison group of children with mild intellectual disability. Jordan (1989) also found that children with ASD frequently used their own name or the experimenter's name instead of *you* or *I*, and in this regard differed significantly from both the group with intellectual disability and the typically developing group.

The use of a proper name or third person pronoun is not semantically incorrect, in that it does communicate the intended referent. However, it does not follow the pragmatic rules of language use, that is, the appropriate use of language in context. Thus, using a third person reference

fails to adopt the convention of marking the speaker and addressee as the participants in the conversation, which is an important part of marking one's perspective in discourse.

Pragmatically appropriate third person reference. Pronouns are an important part of language because they allow us to refer to people and things, but they do not merely carry information about referential identity. The use of a pronoun also communicates something about the speaker's perspective on the referent or the situation. Pronouns tend to be reserved for things that are highly salient in the situation, whereas names and descriptions tend to be used for reference to less prominent entities.

This means that learning how to use pronouns appropriately includes learning that they are meant to indicate salient referents, and learning how to select the best reference form for the current situation. This choice applies mostly to third person references (e.g., *Mia* vs. *she*). By contrast, the discourse participants (*I*, *you*, *we*) are naturally salient in the context, and thus always should be referred to with pronouns.

Analysis of typical language use has identified numerous discourse properties that pattern with the use of pronouns. Speakers and writers typically introduce a character or object with a full name or noun phrase. Subsequent references can be pronominal, depending on a variety of factors stemming from the discourse context and the speaker's judgment about how interpretable the pronoun is. For example, pronouns are used more often for things mentioned recently, and especially in prominent syntactic positions, like grammatical subject (see Arnold 2008).

The question is whether individuals with ASD produce third person reference differently than other groups of speakers. Differences could emerge as a tendency to produce over-ambiguous speech, for example, using a pronoun in a context where the referent is not clear. Alternatively, differences could reflect a tendency to use overspecific forms, avoiding pronouns when they would be appropriate. Evidence has demonstrated that highly verbal individuals with ASD are generally sensitive to discourse constraints, and observed differences are a matter of degree, not complete failure to understand the referential system. Where

differences emerge, they are primarily in the direction of overspecification, but some studies have also reported increased ambiguity.

For example, Arnold, Bennetto, and Diehl (2009) analyzed spoken narratives produced by children and adolescents with ASD, and a well-matched group of their typically developing peers. They found that pronoun use was driven by the discourse context for both the younger (age 9–12) and older (age 13–17) groups, both with and without ASD. That is, pronouns were used most often when the referent had been last mentioned as the grammatical subject of the preceding utterance, and new introductions were almost always with a full name or description. But at the same time, the younger children with ASD displayed a slight bias toward overspecific forms. This is consistent with the observation of Baltaxe (1977) that many adults with ASD used referential expressions that were more specific than needed, and with evidence of using names instead of first and second person pronouns (see above). This tendency emerged only in those cases where the discourse context was less constraining, which highlights the importance of assessing referential behavior within the context of a fine-grained discourse analysis. As another example, Colle, Baron-Cohen, Wheelwright, and van der Lely (2008) examined referential choices made by adults in a story-telling task. The adults in both the ASD and control groups were similar in their use of explicit expressions to introduce or reintroduce characters, and for the most part used pronouns to maintain reference. At the same time, the autism group was relatively less likely to use pronouns for maintenance than the control group, again showing a bias toward overspecific reference.

Some studies have also reported that speakers with ASD may use ambiguous reference more than comparison groups. For example, the participants in Colle et al.'s (2008) study were more likely to use ambiguous pronouns for reference to one of the characters. Similarly, Tager-Flusberg (1995) asked children to tell a story from a picture book. Children in the ASD group were more likely to introduce a new character for the first time with a pronoun.

Our understanding of how reference production is related to ASD also comes from studies on

related aspects of reference, such as producing additional description when necessary. When the referent is salient or identifiable, a simple expression is sufficient (e.g., *the cup*), whereas additional description is necessary when a second possible referent is in the context (e.g., *the short cup*). Nadig, Vivanti, and Ozonoff (2009) found that the children with autism in their sample displayed individual differences. Some, who had higher structural language levels, behaved just like the typically developing group, adapting their expressions to both their own and their partner's perspectives. However, some failed to adapt their expressions at all, and some only adapted to their own perspective.

In sum, sometimes ASD is associated with errors in pronoun use, in particular the overuse of names or labels instead of the pronouns *you* and *I*, and on occasion the reversal of first and second pronouns. At the same time, many individuals with ASD have demonstrated sophisticated abilities to use pronouns appropriately within the discourse context.

Future Directions

Research has established that the pragmatic use of pronouns and other referential expressions is sometimes affected by ASD. Yet it appears that pronoun use strategies are not the same for all individuals with ASD. Future research is needed to better understand individual differences in referential strategies, and the range of both successes and difficulties with reference associated with ASD. To this end, detailed linguistic analyses are needed in order to precisely identify the contexts in which speakers with ASD may use pronouns and other forms of reference differently than other groups.

Further research is also needed to better understand whether ASD impacts the comprehension of pronouns. Question-answering studies have found good pronoun comprehension among individuals with and without ASD, but these say little about moment-by-moment processing. Further work is needed to assess whether individuals with ASD are sensitive to the appropriateness of particular types of

referential forms in a given context, and how referential processing proceeds in real time.

See Also

- [Echolalia](#)
- [Language](#)
- [Pragmatics](#)
- [Pronoun Errors](#)
- [Pronoun Reversal](#)

References and Reading

- Arnold, J. E. (2008). Reference production: Production-internal and addressee-oriented processes. *Language and Cognitive Processes*, 23, 495–527.
- Arnold, J. E., Bennetto, L., & Diehl, J. J. (2009). Reference production in young speakers with and without autism: Effects of discourse status and processing constraints. *Cognition*, 110, 131–146.
- Baltaxe, C. A. M. (1977). Pragmatic deficits in the language of autistic adolescents. *Journal of Pediatric Psychology*, 2, 176–180.
- Bard, E. G., & Aylett, M. P. (2004). Referential form, word duration, and modeling the listener in spoken dialogue. In J. C. Trueswell & M. K. Tanenhaus (Eds.), *Approaches to studying worldsituated language use: Bridging the language-as-product and language-as-action traditions* (pp. 173–191). Cambridge, MA: MIT Press.
- Bard, E. G., Anderson, A. H., & Sotillo, C. (2000). Controlling the intelligibility of referring expressions in dialogue. *Journal of Memory and Language*, 42(1), 1–22.
- Bartak, L., & Rutter, M. (1974). The use of personal pronouns by autistic children. *Journal of Autism and Childhood Schizophrenia*, 4(21), 7–222.
- Charney, R. (1980). Pronoun errors in autistic children: Support for a social explanation. *British Journal of Disorders of Communication*, 15(1), 39.
- Clark, E. V. (1978). From gestures to word: On the natural history of deixis in language acquisition. In J. S. Bruner & A. Garton (Eds.), *Human growth and development: Wolfson college lectures 1976*. Oxford: Clarendon Press.
- Colle, L., Baron-Cohen, S., Wheelwright, S., & van der Lely, H. K. J. (2008). Narrative discourse in adults with height-functioning autism or Asperger syndrome. *Journal of Autism Developmental Disorders*, 38, 28–40.
- Gundel, J. K., Hedberg, N., & Zacharski, R. (1993). Cognitive status and the form of referring expressions in discourse. *Language*, 69, 274–307.
- Hobson, R. P., Lee, A., & Hobson, J. A. (2010). Personal pronouns and communicative engagement in autism. *Journal of Autism Developmental Disorders*, 40, 653–664.
- Jordan, R. R. (1989). An experimental comparison of the understanding and use of speaker-addressee personal pronouns in autistic children. *British Journal of Disorders of Communication*, 24, 169–172.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Lee, A., Hobson, R. P., & Chiat, S. (1994). I, you, me and autism: An experimental study. *Journal of Autism and Developmental Disorders*, 24, 155–176.
- Loveland, K. A., & Landry, S. H. (1986). Joint attention and language in autism and developmental language delay. *Journal of Autism and Developmental Disorders*, 16, 335–349.
- Nadig, A., Vivanti, G., & Ozonoff, S. (2009). Adaptation of object descriptions to a partner under increasing communicative demands: A comparison of children with and without autism. *Autism Research*, 2, 1–14.
- Oshima-Takane, Y., & Benaroya, S. (1989). An alternative view of pronominal errors in autistic children. *Journal of Autism and Developmental Disorders*, 19, 73–85.
- Tager-Flusberg, H. (1995). ‘Once upon a rabbit’: Stories narrated by autistic children. *British Journal of Developmental Psychology*, 13, 45–59.

Pronunciation

- [Articulation](#)

PROSAP2

- [SHANK 3](#)

Prosocial Behavior

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Definition

Prosocial behaviors include a range of positive and friendly behaviors that individuals display toward others. In children, these behaviors include sharing, helping, and taking turns with others, cooperating with others to achieve a common goal, and negotiating with others to reach a mutually acceptable

compromise when a conflict occurs (Bierman 2004; Grusec et al. 2002; Ladd 2005). Prosocial behaviors are important to children's success in peer interactions. Children whose words and actions are perceived as prosocial are viewed more favorably by their peers than those whose words and actions are viewed as indifferent, passive, hostile, or irrelevant (Coie et al. 1990). Most social communication interventions aim to increase prosocial behaviors and may include specific activities to teach children to produce polite requests, compliment each other, respond to other's comments and questions, and resolve conflicts by staying calm and suggesting a compromise.

See Also

- [Circle of Friends](#)
- [Peer-Mediated Intervention](#)
- [Pragmatic Language Impairment](#)
- [Pragmatic Language Skills Inventory](#)
- [Pragmatics](#)
- [Social Communication](#)
- [Social Language Development Test](#)
- [Social Skill Interventions](#)
- [Social Skills Improvement System](#)
- [TRIAD Social Skills Assessment](#)

References and Reading

- Bierman, K. (2004). *Peer rejection: Developmental processes and intervention strategies*. New York: The Guilford Press.
- Coie, J., Dodge, K., & Kupersmidt, J. (1990). Peer group behavior and social status. In S. Asher & J. Coie (Eds.), *Peer rejection in childhood* (pp. 17–59). Cambridge, UK: Cambridge University Press.
- Grusec, J., Davidov, M., & Lundell, L. (2002). Prosocial and helping behavior. In P. K. Smith & C. H. Hart (Eds.), *Blackwell handbook of childhood social development* (pp. 457–474). Malden: Blackwell.
- Ladd, G. (2005). *Children's peer relations and social competence: A century of progress*. New Haven: Yale University Press.

Prosocial Behaviors

- [Social Skills Improvement System](#)

Prosocial Skills

- [Social Skills Improvement System](#)

Prosody

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Definition

Prosody refers to the rhythm and melody of the voice, including intonation, stress, and pauses. Prosody can provide cues to lexical meaning, (e.g., CONtract vs. conTRACT), grammatical structure, emphasis (e.g., I want THIS cookie), affect, and structure in discourse (e.g., asking a question or making a statement), among other functions.

P

Historical Background

In their initial descriptions of the disorder, both Leo Kanner and Hans Asperger noted the presence of differences in tone of voice used by children with autism spectrum disorder (ASD). Asperger noted that the children with this disorder could sound too soft and far away, shrill, and have intonation with a singsong quality or that did not go down appropriately at the end of a declarative statement. Descriptions of prosody have ranged from flat or monotonous to variable, pedantic, and/or having a singsong quality. Because of this variability in symptom presentation across the autism spectrum, it has been historically difficult to identify which characteristics of prosody production are “autistic-like.” As a result, it has been difficult to conduct

research on the topic, and more notably, few researchers have attempted to develop interventions to directly address prosodic differences.

Early attempts to characterize prosodic performance in individuals with ASD divided prosody skills into broad categories such as “linguistic” prosody (i.e., types of prosody that communicate sentence structure, discourse structure, word meaning, among others) and “affective” prosody, based on the theory that children with ASD would have difficulty with judging emotions in tone of voice. More recently, researchers have focused on the nuanced nature of prosody comprehension and production, and its integration with words, grammatical rules, gestures, and facial expression. Notably, researchers have focused on profiles of performance across pragmatic communication skills and its potential as an early diagnostic marker for ASD.

It should be noted that knowledge about prosody has historically lagged behind other areas of language research (such as syntax or semantics) in both typical and disordered populations. This lag in research has primarily been because of its complexity as a construct, difficulties in assessment, and its variable presentation in communication. Whereas syntax follows a set of rules that are consistently observable by most native speakers of a language, prosodic cues can be interpreted differently by different people, especially when the prosody is in conflict with other semantic or contextual information (e.g., irony or sarcasm). Similarly, research on prosodic performance in individuals with ASDs has been hindered by the relatively small (amongst communication domains) amount of research on normative samples.

Current Knowledge

Prosody refers to the patterns of rhythm, intonation, and stress of the voice. Prosody is considered to be a *suprasegmental* element of speech, meaning that one prosodic pattern can include more than just one sound or word, but potentially an entire sentence or conversation. Prosody has many functions in language and can be used to

clarify linguistic, pragmatic, and affective aspects of an utterance.

At the word level, prosody marks lexical and syntactic distinctions. *Lexical stress* can affect the meaning of the word, depending on which syllable is emphasized. For example, the word “CONtract” refers to a legally binding document, whereas “conTRACT” is a verb meaning “to shrink.” Individually stressed words within a sentence can also function emphatically (e.g., “It was a REALLY big fish!”), or contrastively, to draw attention to a difference from a word in a previous clause or sentence (e.g., “I have a blue car and she has a GREEN one.”) or repair a miscommunication (e.g., “Did you order chocolate cake?” “No, I ordered chocolate ICE CREAM.”). These uses are sometimes called “linguistic” or “grammatical” prosody to differentiate them from prosodic patterns that express affect (or “affective” prosody).

At the sentence level, prosody can have both grammatical and affective/pragmatic functions. Grammatically, prosodic cues can be used to identify clausal boundaries, clarifying sentences that might otherwise be ambiguous. For example, by altering the rhythmic pattern of pauses in a sentence, we can change “The man (pause) tapped the woman (pause) with the cane.” (i.e., the man used a cane to tap a woman) to “The man tapped (pause) the woman with the cane.” (i.e., the man tapped a woman who uses a cane).

Sentence-level prosody can also convey social and affective information. Intonation patterns often indicate the pragmatic function of a sentence: whether it is a statement or a question, and whether it is intended literally or sarcastically. Further, prosodic cues can communicate the speaker’s affective state, providing additional subtext to an utterance. For example, “My sister will be in town next week” could be said with excitement or with dismay, with each version shedding light on the relationship between the speaker and his sibling, and suggesting what the appropriate response might be. Listeners can also use prosody to identify a speaker’s accent, which can provide information about regional origins and social status.

Prosodic Development

While most adults can use prosody for all of these purposes, our knowledge of the developmental trajectory of these skills is in its infancy. Research on prosody is particularly difficult to conduct due to the wide variety of acoustic cues that can be used to indicate stress, such as pitch (fundamental frequency) and intensity changes, vocalic lengthening, and pauses. Further, it is impossible to completely separate prosodic cues from the lexical content of an utterance, and thus prosody must be considered in context.

In typically developing children, prosody is one of the earliest (if not the earliest) social abilities to develop. Fetuses can hear and respond to vocal patterns in utero. Within the first few days after birth, children can differentiate between their mother's voice and other voices. Moreover, newborns are able to differentiate between the prosodic patterns of their native language and a foreign language.

Prosodic features of language are present very early in language production. Even at the babbling stage, children produce sentence-like strings of sounds that conform to the intonation patterns of their native language. As children incorporate words into their vocabulary and begin to combine them into sentences, these utterances tend to have not only the word order but also the prosodic patterns of adult speech. In some areas of language acquisition, as in the case of forming questions, the use of prosodic cues precedes that of syntactic cues, with children using a rising intonation to indicate a question ("You go store?") before they are able to invert the subject and object ("Is he happy?") or insert auxiliaries ("Do you like ice cream?").

In the domain of comprehension, typically developing children are able to glean affective information from prosodic cues from infancy. Even before they have the vocabulary to understand their mother's speech, they are able to decipher whether she is happy or angry from her tone of voice. Later, children are able to use lexical stress cues to differentiate between words that contrast on stress patterns alone. By school age, children can use intonation patterns to

interpret syntactically ambiguous sentences. Finally, around age 8, children begin to be able to understand the more complex pragmatic prosodic cues, such as sarcasm, a meaning in which prosody contrasts with lexical meaning and/or contextual meaning to provide an alternate mental state. Prior to this, most children will interpret all statements literally, regardless of tone of voice.

Prosody in Autism Spectrum Disorders

Prosodic impairments are common across individuals with ASDs, including individuals who have intact structural linguistic abilities. In production, individuals with autism are often noted for having unusual prosody in their speech, either for speaking in a flat, monotone, or robotic manner or for having an exaggerated or otherwise odd style of speaking. In studies examining stress patterns, participants with ASD have been shown to be more likely than controls to misassign stress or to accent multiple elements in a sentence, rather than highlighting only key words.

Individuals with ASD seem to have an uneven profile with respect to comprehension of prosody. Several studies have shown impairments in using prosodic cues to understand affect and mental states. Studies have been more mixed with respect to other linguistic or pragmatic uses of prosody, with findings generally suggesting more impairment in sentence-level processing, with more spared prosodic comprehension for word stress and question contours. Findings for differences in prosodic production have been surprisingly limited, despite the pervasive nature of clinical reports of prosodic differences, and there is a need for more studies of subtle acoustic differences in prosodic patterns produced by individuals with ASD.

Assessment and Treatment of Prosodic Disorders

Prosodic disorders are notoriously difficult to assess objectively and to treat. Clinically, prosodic impairments are often diagnosed based on the

therapist's subjective judgments of "oddness," and a determination of whether any atypical speech patterns can be accounted for by other articulatory or phonological disorders. A few standardized measures have been developed to test prosodic abilities in children, including the Prosody-Voice Screening Profile (PVSP) and the Profiling Elements of Prosodic Systems for Children (PEPS-C). Generally, prosodic disorders are identified in the domain of language production. To this end, the PVSP analyzes samples of a child's natural speech and compares them to a normative database to find atypical prosodic patterns. However, to score this test, one must be able to transcribe prosody, which requires a great deal of time and training, making it less practical for typical clinical settings. The PEPS-C is a more structured assessment, which makes it possible to elicit language that uses prosody in a variety of functions such as phrasing, focus, and affect. This test includes both a production and a comprehension component using audio CDs and thus can be used to test receptive as well as expressive prosodic abilities.

There is currently no standard treatment for prosodic disorders. Speech therapists might model appropriate speech patterns for their clients or use Melodic Intonation Therapy, which first presents pitch patterns and rhythms with humming, and progressively approximates natural speech. Other therapies have been developed that focus on specific areas of prosody such as stress placement as well. Future research should focus on developing and testing interventions for prosodic impairments as this is an area that has been somewhat neglected in treatment plans. For high-functioning children with autism, odd prosody can be an obstacle in gaining social acceptance with peers.

Future Directions

Future research should utilize acoustic characteristics of the speech signal to characterize some of the subtle differences in prosody production that children with ASD exhibit. Future studies should focus on developing assessment techniques and

interventions for prosodic impairment. Furthermore, automatic analysis of the acoustic signal is promising for evaluation and instant feedback during therapy. In all areas of research, assessment, and treatment, it will be very important to be sensitive to individual differences in prosodic abilities and deficits, as these differences might be informative (i.e., a "bellwether") for an individual's specific cognitive/linguistic strengths and weaknesses.

See Also

- [Communication Assessment](#)
- [Communication Disorder/Communication Impairment](#)
- [Intonation](#)
- [Monotone](#)
- [Paralinguistic Communication Assessment](#)
- [Pragmatic Communication](#)
- [Pragmatics](#)
- [Prosody-Voice Screening Protocol](#)
- [Social Communication](#)

References and Reading

- Baltaxe, C. A. M. (1984). Use of contrastive stress in normal, aphasic, and autistic children. *Journal of Speech and Hearing Research*, 27, 97–105.
- Baltaxe, C. A. M., & D'Angiola, N. (1996). Referencing skills in children with autism and specific language impairment. *European Journal of Disorders of Communication*, 31(3), 245–258.
- Chevallier, C., Noveck, I., Happe, F., & Wilson, D. (2009). From acoustics to grammar: Perceiving and interpreting grammatical prosody in adolescents with Asperger syndrome. *Research in Autism Spectrum Disorder*, 3(2), 502–516.
- Diehl, J. J., & Berkovits, L. (2010). Is prosody a diagnostic and cognitive bellwether of autism spectrum disorders? In A. Harrison (Ed.), *Speech disorders: Causes, treatments, and social effects* (pp. 159–176). New York: Nova Science.
- Diehl, J. J., Watson, D., Bennetto, L., McDonough, J., & Gunlogson, C. (2009). An acoustic analysis of prosody in high-functioning autism. *Applied PsychoLinguistics*, 30, 1–20.
- Diehl, J. J., Friedberg, C., Paul, R., & Snedeker, J. (2015). The use of prosody during syntactic processing in children and adolescents with autism spectrum disorders. *Development and Psychopathology*, 27(03), 867–884.

- Eigsti, I. M., Schuh, J. M., Mencl, E., Schultz, R. T., & Paul, R. (2012). The neural underpinnings of prosody in autism. *Child Neuropsychology*, 18(6), 600–617. <https://doi.org/10.1080/09297049.2011.639757>.
- Fusaroli, R., Lambrechts, A., Bang, D., Bowler, D. M., & Gaigg, S. B. (2016). Is voice a marker for Autism spectrum disorder? A systematic review and meta-analysis. *Autism Research*, 10(3), 384–407.
- Ghaziuddin, M., & Gerstein, L. (1996). Pedantic speaking style differentiates Asperger syndrome from high-functioning autism. *Journal of Autism and Developmental Disorders*, 26, 585–595.
- Grossman, R. B., Edelson, L. R., & Tager-Flusberg, H. (2013). Emotional facial and vocal expressions during story retelling by children and adolescents with high-functioning autism. *Journal of Speech, Language, and Hearing Research*, 56(3), 1035–1044.
- Hobson, R. P., Ouston, J., & Lee, A. (1988). Emotion recognition in autism – Coordinating faces and voices. *Psychological Medicine*, 18, 911–923.
- Lindner, J. L., & Rosén, L. A. (2006). Decoding of emotion through facial expression, prosody and verbal content in children and adolescents with Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 36(6), 769–777.
- McCann, J., & Peppé, S. (2003). Prosody in autism spectrum disorders: A critical review. *International Journal of Language & Communication Disorders*, 38(4), 325–350.
- Paul, R., Augustyn, A., Klin, A., & Volkmar, F. R. (2005). Perception and production of prosody by speakers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 35(2), 205–220.
- Peppé, S. (2009). Why is prosody in speech-language pathology so difficult? *International Journal of Speech-Language Pathology*, 11(4), 258–271.
- Peppé, S., McCann, J., Gibbon, F., O'Hare, A., & Rutherford, M. (2007). Receptive and expressive prosodic ability in children with high-functioning autism. *Journal of Speech, Language, and Hearing Research*, 50(4), 1015–1028.
- Rosenblau, G., Kliemann, D., Dziobek, I., & Heekeren, H. R. (2016). Emotional prosody processing in autism spectrum disorder. *Social Cognitive and Affective Neuroscience*, 12(2), 224–239. <https://doi.org/10.1093/scan/nsw118>.
- Rutherford, M. D., Baron-Cohen, S., & Wheelwright, S. (2002). Reading the mind in the voice: A study with normal adults and adults with Asperger syndrome and high functioning autism. *Journal of Autism and Developmental Disorders*, 32(3), 189–194.
- Schoen, E., Paul, R., & Chawarska, K. (2011). Phonology and vocal behavior in toddlers with autism spectrum disorders. *Autism Research*, 4(3), 177–188. <https://doi.org/10.1002/aur.183>.
- Shriberg, L. D., Paul, R., McSweeney, J. L., Klin, A., Cohen, D. J., & Volkmar, F. R. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning autism and Asperger syndrome. *Journal of Speech, Language, and Hearing Research*, 44(5), 1097–1115.
- Tager-Flusberg, H., Edelson, L., Luyster, R., Amaral, D., Dawson, G., & Geschwind, D. (2011). Language and communication in autism spectrum disorders. *Autism spectrum disorders*, 172–185.
- Thurber, C., & Tager-Flusberg, H. (1993). Pauses in the narratives produced by autistic, mentally-retarded, and normal children as an index of cognitive demand. *Journal of Autism and Developmental Disorders*, 23, 309–322.
- Van Santen, J. P. H., Prud'hommeaux, E. T., Black, L. M., & Mitchell, M. (2010). Computational prosodic markers for autism. *Autism*, 14(3), 215–236.
- Warren, S. F., Gilkerson, J., Richards, J. A., Oller, D. K., Xu, D., Yapanel, U., et al. (2010). What automated vocal analysis reveals about the vocal production and language learning environment of young children with autism. *Journal of Autism and Developmental Disorders*, 40(5), 555–569.

Prosody-Voice Screening Protocol

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Synonyms

PVSP

Definition

The Prosody-Voice Screening Profile (PVSP) is a test of prosody production. The PVSP collects spontaneous speech in order to assess the prosodic and vocal characteristics of the speaker. Phrasing, rate, stress, pitch loudness, and vocal quality are transcribed and judged for appropriateness. It is suitable for use with children and adults; assesses expressive but not receptive prosodic ability; and has normative data to which comparisons can be made.

See Also

- Intonation
- Paralinguistic Communication Assessment
- Prosody

References and Reading

- McSweeney, J. L., & Shriberg, L. D. (2001). Clinical research with the prosody-voice screening profile. *Clinical Linguistics & Phonetics*, 15, 505–528.
- Shriberg, L. D., Kwiatkowski, J., & Rasmussen, C. (1990). *The prosody-voice screening profile*. Tucson: Communication Skill Builders.
- Shriberg, L., Kwiatkowski, J., Rasmussen, C., Lof, G. L., & Miller, J. F. (1992). *The prosody-voice screening profile (PVSP): Psychometric data and reference information for children*. Madison: Waisman Center on Mental Retardation and Human Development, University of Wisconsin-Madison.
- Shriberg, L. D., Paul, R., McSweeney, J. L., Klin, A., Cohen, D. J., & Volkmar, F. R. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning autism and asperger syndrome. *Journal of Speech, Language & Hearing Research*, 44(5), 1097–1115.

Prosopagnosia

► Pareidolic Faces

Prospective

► Longitudinal Research in Autism

Prospective Memory in Autism Spectrum Disorder

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Synonyms

Everyday memory; Memory for future actions; Memory for intentions; PM; ProM; Remembering to remember

Definition

“Prospective memory” (PM) is a form of memory that involves remembering in the future – i.e., “remembering to remember.” More specifically, PM engages many parallel cognitive functions to plan a future goal (task, thought, or action; “intention formation”), to be carried out (“intention initiation”) at a defined future point (“intention execution”), usually after a delayed period of time (“retention interval”). Successful PM not only depends on correctly recalling “when” to act, but also recalling appropriate information (“what” to remember) about the planned goal or action (“intention retrieval”) in the appropriate context (“how” to act on the intention). PM intentions are usually carried out during the course of unrelated ongoing activities and, therefore, draw on executive function resources, such as working memory, attention monitoring, planning, and switching between the ongoing and PM activities. Although PM involves similar cognitive mechanisms to episodic future thinking and retrospective memory, such as encoding and retrieving information over time, it contrasts with standard episodic or working memory. This is because PM depends not only remembering “when” to remember, but also on correctly remembering “what” should be remembered and “how” that information should be acted on. Although PM cannot be regarded as “process pure” because of the many cognitive functions it involves, it is a multiprocess framework drawing on a specific combination of processes that distinguish it as a unique type of memory with dissociable functions that balance automatic and strategic cognitive mechanisms (see McDaniel and Einstein 2000; Ball and Aschenbrenner 2018).

The most commonly investigated PM processes focus on time-based or event-based remembering. Time-based PM is centered around time-sensitive remembering, such as attending an appointment at 2:00 p.m. next Wednesday or turning off the oven after 30 min. Accordingly, time-based PM depends on self-monitoring and effective internal time-keeping to perform the PM action at the appropriate time. It also relies on self-

regulated inhibition to stop an ongoing activity so that the PM action can be completed. Therefore, time-based PM places substantial competing demands on cognitive resources (Ball and Aschenbrenner 2018). By contrast, event-based PM involves remembering to carry out an intention in response to an external cue or event, such as taking medication with breakfast or posting a letter when you pass the post office. Often, supportive strategies, such as using a diary or memo note, are helpful in serving as reminders or cues. Additionally, event-based PM can be prompted by other events or circumstances that occur simultaneously. For instance, seeing a postbox might serve as a reminder to post the letter. In this way, event-based PM can be considered less demanding on executive functioning/working memory processes.

The effective use of PM is vital for everyday functioning, autonomous living, and self-management of one's healthcare and daily living skills (e.g., remembering to take medication; pay bills on time; or attend an appointment next week). It is also a primary reason for everyday forgetting and is one of the most sensitive factors in age-related cognitive decline (Woods et al. 2012). Previous research has shown variable PM performance in typically developed individuals, depending on the types of tasks and PM cues (e.g., routine/non-routine; regular/irregular; important/not important; focal/non-focal), depending as well as on the valence of cues, and cognitive load of any ongoing tasks (e.g., Ball and Aschenbrenner 2018). In the context of ageing, PM ability depends on the context of the task and whether it is carried out in a naturalistic environment or laboratory-type setting. In particular, research has shown that older typically developed adults tend to perform as well as younger adults on event-based tasks, but have greater difficulties on time-based tasks (mirroring the performance seen in autism; see below). By contrast, in naturalistic settings, several studies have observed the reverse pattern of abilities (see Henry et al. 2004 for a review). In turn, PM failures are closely linked with increased cognitive decline in older age and poor physical and mental health.

Consequently, research suggests PM ability underpins the ability to function well, with abilities also predicting health-related quality of life, particularly in older age (Woods et al. 2012).

Prospective Memory and Autism

Individuals with autism spectrum disorder (ASD) demonstrate a unique profile of cognitive strengths and difficulties. A large body of research suggests a profile of impairments in autism that includes some aspects of executive function (e.g., inhibition of prepotent responses, complex planning), as well as difficulties recalling memories of personally experienced past events (episodic recollection), particularly on tasks that involve self-initiated recall of information (Bowler et al. 2015). However, relatively few studies have explored PM abilities in ASD and little is known of how PM contributes to everyday functioning and quality of life of autistic individuals (see Roestorf 2018).

In general, studies have explored distinctions between event-based and time-based PM in ASD, with varied findings, depending on task type and PM function (see Landsiedel et al. 2017 for a recent review). One difficulty with interpreting some studies of PM ability in autism is that several studies have assessed both event-based and time-based PM abilities concurrently, in the same task. This is problematic, as impairments in one type of PM may well confound performance on other aspects of the task (see Landsiedel et al. 2017 for a wider discussion). However, when PM abilities are examined in studies that assess time-based and event-based performance in separate tasks, results suggest that autistic individuals have difficulties completing time-based PM tasks in particular (e.g., Williams et al. 2013, 2014). By contrast, impairments on event-based PM tasks appear considerably smaller in autism. In general, time-based PM ability in autism seems largely related to executive function ability, and one possible explanation for this pattern of PM abilities is that more general impairments in executive function in autism seem to broadly underpin difficulties on time-based PM

task. This is likely a result of time-based PM relying more heavily on monitoring and self-initiated retrieval of PM intentions, both of which place high demands on executive function, whereas the executive loads posed by event-based tasks is considerably less, as event-based PM is typically supported by cued retrieval. Alternatively, poor time-based PM performance may also be the result of diminished self-projection ability (see Lind et al. 2014) and impairments in episodic future thinking (see Williams et al. 2013, for a wider discussion).

Recent research exploring PM abilities in both laboratory and naturalistic settings (Roestorf 2018) has also suggested that, whereas younger autistic adults presented similar difficulties to those observed in older typically developed adults (demonstrating greater impairments on time-based tasks than event-based PM tasks), this was not the case for older autistic adults. By contrast, older autistic individuals demonstrated more accurate PM abilities in event-based and time-based tasks in both laboratory and naturalistic settings, compared to matched typically developed adults. Overall, these findings present an emerging picture of PM function in ASD and suggest that PM difficulties may present different challenges for autistic adults at various life stages. However, substantially more research is needed to understand developmental influences on PM and how individual differences in time-based and event-based PM affect everyday functioning and quality of life in autistic individuals across the lifespan.

See Also

- [Executive Functioning and Martial Arts Training in Children with Autism Spectrum Disorder](#)

References and Reading

- Ball, H. B., & Aschenbrenner, A. J. (2018). The importance of age-related differences in prospective memory: Evidence from diffusion model analyses. *Psychonomic Bulletin & Review*, 25, 1114–1122. <https://doi.org/10.3758/s13423-017-1318-4>.
- Bowler, D. M., Gaigg, S. B., & Gardiner, J. M. (2015). Brief report: The role of task support in the spatial and temporal source memory of adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(8), 2613–2617.
- Landsiedel, J., Williams, D. M., & Abbot-Smith, K. (2017). A meta-analysis and critical review of prospective memory in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(3), 646–666. <https://doi.org/10.1007/s10803-016-2987-y>.
- McDaniel, M. A., & Einstein, G. O. (2000). Strategic and automatic processes in prospective memory retrieval: A multiprocess framework. *Applied Cognitive Psychology*, 14(7), S127–S144.
- Roestorf, A. (2018). *Ageing, cognition and quality of life in autism spectrum disorder: Cross-sectional and longitudinal studies*. Part 2, 207–305. Doctoral dissertation, City, University of London. <https://openaccess.city.ac.uk/id/eprint/22094/>

Additional References

- Henry, J. D., MacLeod, M. S., Phillips, L. H., & Crawford, J. R. (2004). A meta-analytic review of prospective memory and aging. *Psychology and Aging*, 19(1), 27–39. <https://doi.org/10.1037/0882-7974.19.1.27>.
- Lind, S. E., Williams, D. M., Bowler, D. M., & Peel, A. (2014). Episodic memory and episodic future thinking impairments in high-functioning autism spectrum disorder: An underlying difficulty with scene construction or self-projection?. *Neuropsychology*. <https://doi.org/10.1037/neu0000005>.
- Sheppard, D. P., Bruineberg, J. P., Kretschmer-Trendowicz, A., & Altgassen, M. (2018). Prospective memory in autism: Theory and literature review. *The Clinical Neuropsychologist*, 32(5), 748–782. <https://doi.org/10.1080/13854046.2018.1435823>.
- Williams, D., Boucher, J., Lind, S., & Jarrold, C. (2013). Time-based and event-based prospective memory in autism spectrum disorder: The roles of executive function and theory of mind, and time-estimation. *Journal of Autism and Developmental Disorders*, 43(7), 1555–1567.
- Williams, D. M., Jarrold, C., Grainger, C., & Lind, S. E. (2014). Diminished time-based, but undiminished event-based, prospective memory among intellectually high-functioning adults with autism spectrum disorder: Relation to working memory ability. *Neuropsychology*, 28(1), 30.
- Woods, S. P., Weinborn, M., Velnoweth, A., Rooney, A., & Bucks, R. S. (2012). Memory for intentions is uniquely associated with instrumental activities of daily living in healthy older adults. *Journal of the International Neuropsychological Society*, 18(1), 134–138.

Protection and Advocacy System (P&A)

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Synonyms

[Client assistance program](#)

Definition

The Protection and Advocacy (P&A) System is comprised of a nationwide network of organizations that has been mandated by the Congress to protect and advocate for the rights of people with disabilities. These organizations provide legal representation and other advocacy services. The goal of P&A is to provide people with disabilities, including children and adults on the autistic spectrum, equal access to employment opportunities, education, public services, healthcare, housing, and public accommodations. P&A organizations investigate abuse, neglect, and barriers that prevent disabled people from becoming productive and independent members of their community. Protection and Advocacy agencies are located in every state and are the primary nonfederal enforcers of disability rights statutes. These organizations may be part of the government or private not-for-profit organizations.

See Also

- [Advocacy](#)
- [Disability](#)
- [Employment](#)
- [Employment in Adult Life](#)
- [Employment Specialist](#)

References and Reading

National Disability Rights Network. Home page. Retrieved 7 Aug 2012 from <http://pandasc.org/>
World Work, Employment Support Institute, School of Business, Virginia Commonwealth University. Retrieved 7 Aug 2012 from <http://www.workworld.org/webhelp/basic.htm>

Protodeclarative

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Synonyms

[Comment](#); [Communicative act](#)

Definition

A primitive speech act used to establish social interaction and direct a caregiver's attention to an object, action, or entity. It is referred to as a protodeclarative because the directed attention to an object, action, or entity by the child acts as a comment in a communicative exchange. A protodeclarative may take several gestural forms including pointing to, showing, or giving of objects. The gesture may or may not be accompanied by a ritualized vocalization. The protodeclarative appears in typically developing children between 8 and 9 months of age. It is thought to demonstrate a shift in cognitive-linguistic development in that its use reflects the child's conscious intent to initiate social interaction and establish joint attention to an object, action, or entity with a caregiver.

Children with autism engage less often in the intentional use of protodeclarative gestures such as pointing, showing, and giving, meant to establish or maintain social interaction and joint attention. There is evidence that the lack of intentional

communication and bids for social interaction are related to a failure of various underlying mechanisms supporting development of preverbal communication.

See Also

- [Communicative Functions](#)
- [Gestures](#)
- [Joint Attention](#)

References and Reading

- Bates, E., Camaioni, L., & Volterra, V. (1975). The acquisition of performatives prior to speech. *Merrill-Palmer Quarterly*, 21, 205–226.
- Camaiono, L., Perucchini, P., Muratori, F., & Milone, A. (1997). Brief report: A longitudinal examination of the communicative gestures deficit in young children with autism. *Journal of Autism and Developmental Disorders*, 27, 715–725.
- Landy, S. H., & Loveland, K. A. (1988). Communication behaviors in autism and developmental language delay. *Journal of Child Psychology and Psychiatry*, 29, 621–634.
- Mundy, P., Sigman, M., & Kasari, C. (1990). A longitudinal study of joint attention and language development in autistic children. *Journal of Autism and Developmental Disorders*, 20, 115–128.
- Owens, R. E. (2008). The social and communicative bases of early language and speech. In R. E. Owens (Ed.), *Language development: An introduction* (7th ed., pp. 113–149). New York: Pearson.
- may take the form of a vocalization, a conventional gesture, or a combination of both. The protoimperative, as a ritualized request, appears in typically developing children between 8 and 9 months of age and is thought to demonstrate a shift in cognitive-linguistic development in that its appearance reflects the child's conscious intent to communicate to the caregiver to act as an agent to attain an object or perform an action. The protoimperative often takes the form of a pointing gesture or a whole-hand or swiping motion toward the desired object, frequently accompanied by a ritualized vocalization, such as a whine or grunt. Eye contact is usually established between the child and the caregiver during or following the ritualized request act. The intentional gestural communication and vocalization occur away from the object or action and are considered to be distal in nature.
- Children with autism frequently do not develop a protoimperative communicative function characterized by the use of distal communicative gestures, such as pointing while vocalizing. Rather, a request function may be accomplished via a contact gesture, such as leading a caregiver by the hand toward the desired object/action, or placing the caregiver's hand on the object. Protoimperatives using distal signaling via conventional gestures, vocalizations, and eye contact are often absent.

See Also

- [Communicative Functions](#)
- [Gestures](#)
- [Joint Attention](#)

Protoimperative

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Synonyms

[Communicative act](#); [Intentional](#), [Ritualized request](#)

Definition

A protoimperative is a primitive speech act used as a request for objects or actions. A protoimperative

References and Reading

- Bates, E. (1979). *The emergence of symbols: Cognition and communication in infancy*. New York: Academic.
- Bates, E., Camaioni, L., & Volterra, V. (1975). The acquisition of performatives prior to speech. *Merrill-Palmer Quarterly*, 21, 205–226.
- Camaiono, L., Perucchini, P., Muratori, F., & Milone, A. (1997). Brief report: A longitudinal examination of the communicative gestures deficit in young children with autism. *Journal of Autism and Developmental Disorders*, 27, 715–725.
- Chawarska, K., & Volkmar, F. R. (2005). Autism in infancy and early childhood. In D. J. Cohen & F. R. Volkmar

- (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 223–246). New York: Wiley.
- Mundy, P., Sigman, M., & Kasari, C. (1990). A longitudinal study of joint attention and language development in autistic children. *Journal of Autism and Developmental Disorders*, 20, 115–128.
- Owens, R. E. (2008). The social and communicative bases of early language and speech. In R. E. Owens (Ed.), *Language development: An introduction* (7th ed., pp. 113–149). New York: Pearson.

- Ferguson, C. A. (1976). Learning to pronounce: The earliest stages of phonological development in the child. *Papers and Reports on Child Language Development*, 11, 1–27.
- Menyuk, P., & Menn, L. (1979). Early strategies for the perception and production of words and sounds. In P. Fletcher & M. Garman (Eds.), *Language acquisition: Studies on first language development*. Cambridge, MA: CUP.
- Vihman, M., & McCune, L. (1994). When is a word a word? *Journal of Child Language*, 21, 317–342.

Protowords

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Synonyms

Phonetically consistent form; Quasi-words;
Vocable

Definition

Protowords are word-like forms produced by young children as they transition from prelinguistic to linguistic communication (i.e., the time at which children produce intelligible single words that approximate conventional pronunciation). In typical development, protowords emerge between 10 and 12 months of age and are made up of the types of sounds that children use when babbling. Common examples of protowords are mama, dada, and baba. Protowords differ from repetitive babbling in two critical ways: (a) protowords are limited to one to two syllables in length and (b) protowords are used consistently by the child to refer to a corresponding object, person, or event in the child's immediate context. Protowords also have been referred to as repeatedly occurring phonetic units loosely bound to specifiable contexts.

References and Reading

- Dore, J., Franklin, M., Miller, R., & Ramer, A. (1975). Transitional phenomena in early language acquisition. *Journal of Child Language*, 3, 13–28.

Protriptyline

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Synonyms

Vivactil

Definition

Protriptyline is in an older class of antidepressants known as tricyclics. The term “tricyclic” refers to the three-ring structure of this class of antidepressant medications. These medications are not used as commonly as in the past as they have been largely replaced by the SSRIs. Protriptyline is predominantly a norepinephrine reuptake inhibitor.

The tricyclic antidepressants have several adverse effects in common including dry mouth, urinary retention, constipation, nausea, increased heart rate, dizziness, and, at higher doses, confusion. The tricyclic antidepressants also carry some risk of altering the electrical conduction in the heart. They are well known to be fatal on overdose due to their potential for causing cardiac arrhythmia. Because of their known toxicity at higher doses, treatment with tricyclic antidepressants

requires blood-level monitoring and electrocardiogram monitoring as well. Finally, the tricyclic antidepressants are also vulnerable to drug-drug interaction. For example, some medications such as SSRIs or certain antibiotics may interfere with the breakdown of tricyclic antidepressant medications. The interference of metabolism of the tricyclic can cause a sharp increase in the blood levels of the tricyclic antidepressants and increase the vulnerability to toxic effects. The tricyclic medications have not been well studied in children or adults with autism.

See Also

► Antidepressant Medications

References and Reading

- Martin, A., Scahil, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.
- McDougle, C., & Posey, D. J. (2011). Assessment and treatment of autistic and other pervasive developmental disorders. In A. Martin, L. Scahill, & C. Kratochvil (Eds.), *Pediatric psychopharmacology: Principles and practice* (pp. 547–560). New York: Oxford University Press.

Provider Training

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Definition

Provider training is the professional development or technical assistance offered to or participated in by providers to develop competencies and proficiencies on a particular topic or in a specific area necessary for ensuring services, supports, and programming are delivered appropriately and

with fidelity. Provider training often includes a variety of topics, such as the characteristics of autism (including diagnostic, cognitive, sensory, and attentional features), functional assessment (including behavior and adaptive skills), instructional strategies (including applied behavior analysis or ABA techniques such as discrete trial training, structured teaching, and incidental teaching), data collection, teaching functional communication and social skills development, and transitioning into employment settings. Provider training also extends to higher education and other agencies preparing preservice professionals.

A provider is someone whose business is to supply a particular service or commodity or a person or group of people who plans and delivers a program. In the area of autism, providers may include but are not limited to the following domains: health care (medical, dental, physical, mental health, pediatric), special education, related services, school personnel, developmental services, rehabilitation, birth to three, community-based staff, post-secondary disabilities units, developmental specialists, and parents or legal guardians. Further, federal requirements under the Individuals with Disabilities Education Act call for qualified providers in the area of special education and related services. Related service providers can include, in addition to the aforementioned areas, professionals in audiology, early intervention, physical/occupational therapy, speech-language pathologists, recreation, school health/nursing, and social work. Providers can deliver direct services, usually referring to hands-on interactions between the provider and client or student, or indirect services, which may involve consulting with or supervising other staff in the delivery of services.

Historical Background

Though *ASD* is a familiar term in the early twenty-first century, it was only recognized in the 1940s as a severe disability. Since that time, there has been extensive interest in and professional activity concerning autism.

Current Knowledge

It is well understood among the experts developing and delivering provider training in the area of autism spectrum disorders that those receiving the training are prepared on the nature of *ASD* (etiology, incidence, range of symptoms, possible medical concerns), early identification, intensive programming, comprehensive curricula, systematic instruction, objective assessment, providing structured and predictable environments, delivering evidence-based practices and interventions for *ASD*, strategies and tools for implementing best practices across a multitude of settings (classroom, home, workplace, community, etc.), transition planning, and knowledge of facilitating peer relationships. Moreover, providers need to be familiar with terms and definitions used by professionals and consultants developing programs and supports in other service areas in order to best plan for and support the individual with *ASD*.

School-based personnel need training in understanding the characteristics of *ASD* and providing supports for individuals with the disorder. Further, it is recommended that school administrators should understand the educational needs of students with Autism so they can provide a quality program using a variety of service delivery models for children across the age span. Students with autism benefit from individualized and often intense educational services beginning early in life, services that require specialized training for those providing educational interventions with the following characteristics: (1) behaviorally based; (2) carefully planned and monitored instruction involving task analyses of skills, individualized incentives, goals embedded in routines and activities, and adequate intensity and quality; (3) ongoing, planned opportunities for interaction with typical peers; (4) need-based supports and intervention for families; (5) services delivered in many different settings to meet support needs and promote generalization; (6) broad curricular content that addresses all developmental needs; and (7) proactive use of positive behavior support for challenging behavior. Children with autism typically require the services of special educators,

general educators, and speech and language pathologists; occupational or physical therapists often address children's movement and sensory limitations. A collaborative team approach is necessary to plan, problem-solve, implement, and monitor the individualized education programs (IEPs) of these students; as part of a collaborative team, those participating on behalf of an individual with *ASD* should receive training in how best to support the individual with *ASD* given deficits in the areas of communication, behavior, and social interactions.

In the area of early screening for *ASD*, clinicians engaged in developmental screening in health care, community, and school settings are in a unique position to promote children's developmental health, and the Centers for Disease Control strongly suggests clinicians participate in training through the American Pediatric Association in the importance of early childhood development, early intervention, the screeners, appropriate referrals, and billing information; clinicians, in turn, should train other staff and personnel in the practice, particularly in using the screening instruments. Other service providers who may or may not be a part of an individual's educational team require training in how to meet the needs of people with *ASD* in a variety of areas and settings including:

- Consultation for families, educators, and service providers
- Evaluations for eligibility determination
- Training for supporting professionals and families
- Treatment planning and planning for families
- Service coordination
- Early intervention services
- Genetic evaluation, treatment, and counseling services
- Stipends for day care (through ABC block grant) and transportation
- Individualized summer services and camps
- Adult companion services
- Adult dental services
- Adult vision services
- Audiology Services
- Behavioral support services

- Environmental modifications
- Personal care services
- Nursing services
- Occupational therapy services
- Physical therapy services
- Prescribed drugs
- Private vehicle modifications
- Psychological services
- Respite care
- Specialized medical equipment, supplies, and assistive technology

Future Directions

Numerous legislative blue-ribbon commissions and statewide needs assessment initiatives are adding to the literature on the dissemination and implementation of best practices. In the 2008 session, the Connecticut General Assembly passed Special Act 08-5, *An Act Concerning the Teaching of Children with Autism and Other Developmental Disabilities*, and legislated a study group comprised of designees from four state agencies to complete an assessment of school-based personnel needs regarding service delivery to this population of learner and their families. The study group gathered, analyzed, and interpreted new and existing data from seven public forums, three online surveys, policy documents, and information from state data systems to generate recommendations and assemble a statewide plan addressing the methods of teaching children with autism in accordance with requirements of the Act. Findings included several themes confirmed by other states in their own investigations of provider needs including inconsistent quality assurance procedures for ensuring existing statewide training opportunities provided to school personnel and families reflect evidence-based practices, specifically around content, delivery, expertise, results, and alignment with national ASD competencies. Another example is the California Task Force on Education and Professional Development which delivered a set of best practice guidelines for those serving persons with ASD concerning the design and delivery of effective programs as well as a credentialing system for preservice professionals and teacher aides seeking

to work with children and youth with ASD in educational settings. Further, California has proposed that all teachers, including general educators, receive training in ASD as part of their preparation programs. The Alabama Autism Task Force and the University of Alabama Birmingham listed provider training as a top priority in its 2008 statewide assessment across agencies serving individuals from birth through adulthood, covering screening and early intervention through supportive employment opportunities. The Pennsylvania Department of Public Welfare and its state autism task force made training a top priority for its Bureau of Autism Services and has instituted a free, online virtual training site/resource center for its providers in state in order to build capacity across all regions. Many states are beginning to develop and/or implement community and direct service provider trainings tailored for staff involved in autism insurance waiver programs. Waiver provider trainings in most states supporting this effort include focuses on supports coordinators, level of care assessors, behavioral specialist interventions, individualized service plan development, and navigating systems of care. Additionally, several states are reviewing and revising state statute regarding language around coursework or training necessary for providers in meeting the needs of individuals with ASD as well as for those delivering training to the providers across multiple areas.

See Also

- [Related Services](#)
- [Special Education](#)

References and Reading

- Atwood, T. (2001). *Asperger's syndrome: A guide for parents and professionals*. Philadelphia: Jessica Kingsley.
- California best practice guidelines for ASD providers. <http://www.featt.org/LinkClick.aspx?fileticket=u6pwKw%2BLV38%3D&tabid=78&mid=413>
- California Departments of Education and Developmental Services. (1997). *Best practices for designing and delivering effective programs for individuals with autistic spectrum disorders*. Sacramento: Author.

- Center for Disease Control Autism Screening. <http://www.cdc.gov/ncbddd/autism/hcp-screening.html>
- Coleman, J. G. (1993). *The early intervention dictionary*. Bethesda: Woodbine House.
- Committee on Children and Disabilities, American Academy of Pediatrics. (2001). Developmental surveillance and screening for infants and young children. *Pediatrics*, 108(1), 192–195.
- Connecticut Birth to Three System Service Guideline #1: Autism spectrum disorder – Intervention guidance for service providers and families of young children with autism spectrum disorders. (2008, January). Retrieved 25 Feb 2011 from <http://www.birth23.org/providers/SG1-Autism.pdf>
- Connecticut Special Act 08-5 Study Group. (2009). An act concerning the teaching of children and youth with autism spectrum disorders. Retrieved 1 Feb 2011 from http://www.sde.ct.gov/sde/lib/sde/PDF/DEPS/Special/Updates/Minutes_3209.pdf
- Connecticut State Department of Education. (2005). Guidelines for identification and education of children and youth with autism spectrum disorders. Retrieved 30 Dec 2010 from http://www.sde.ct.gov/sde/lib/sde/PDF/DEPS/Special/Guidelines_Autism.pdf
- Council for Exceptional Children Best Practice Standards. <http://www.cec.sped.org/Content/NavigationMenu/ProfessionalDevelopment/ProfessionalStandards/PracticeStandards/default.htm>
- Eren, R., & Kelleher, J. P. (2011). School-based personnel training and support: An often-overlooked IEP requirement. *Autism Spectrum Quarterly*, 9, 53–56.
- Gerlach, E. K. (1996). *Autism treatment guide*. Eugene: Four Leaf Press.
- Koegel, R., & Koegel, L. (1995). *Teaching children with autism. Strategies for initiating positive interactions and improving learning opportunities*. Baltimore: Paul Brookes.
- Maurice, C., Green, M., & Luce, S. (1996). *Behavioral intervention for young children with autism*. Austin: Pro-Ed.
- Mental Health Consumer Connections. <http://www.athealth.com/consumer/disorders/Autism.html>
- Myles, B. S., & Simpson, R. L. (1990). A clinical/prescriptive method for use with students with autism. *Focus on Autistic Behavior*, 4(6), 1–14.
- National Academy of Sciences and Committee on Educational Interventions for Children with Autism. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- National Association of Special Education Teachers. (2009). Assessment in special education series. Retrieved 1 Feb 2011 from <http://www.naset.org/2960.0.html>
- Rogers, S. J. (1996). Brief report: Early intervention in autism. *Journal of Autism and Developmental Disorders*, 26(2), 243–246.
- Schopler, E., & Mesibov, G. (Eds.). (1995). *Learning and cognition in autism*. New York: Plenum Press.
- Squires, J., Nickel, R. E., & Eisert, D. (1996). Early detection of developmental problems: Strategies for monitoring young children in the practice setting. *Journal of Developmental and Behavioral Pediatrics*, 17, 420–427.
- Stahmer, A. C., Collings, N. M., & Palinkas, L. A. (2005). Early intervention practices for children with autism: Descriptions from community providers. *Focus on Autism and Other Developmental Disabilities*, 20(2), 66–79. Read Full item.
- U.S. Department of Education. (2006). *26th annual report to congress on the implementation of the IDEA: Vol. 2*. Washington, DC: Author. Retrieved 20 April 2009 from www.ed.gov/about/reports/annual/osep/2004/index.html.
- U.S. Department of Education, Office of Special Education Programs. (2006, April 10). Building the legacy: IDEA 2004. <http://idea.ed.gov/explore/view/p/%2Croot%2Cdynamic%2CTopicalArea%2C1%2C>
- Vernon, A. (1989). *Thinking. Behaving – An emotional education curriculum for children: Feeling*.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know*. Hoboken: Wiley.
- Volkmar, F. R., Paul, R., Klin, A., & Cohen, D. (2005). *The handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.
- Wing, L. (2002). *The autistic spectrum: A guide for parents and professionals*. London: Robinson Publishing.
- Wright, P., & Wright, P. (2007). *Wrightslaw: Special education law* (2nd ed.). Hartfield: Harbor House Law Press.

Prozac

► Fluoxetine

Prozac Weekly (Once-Weekly Dosing)

► Fluoxetine

Prozac™ (Fluoxetine)

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Synonyms

Fludac; Fluoxin; Flutine; Fluxil; Fontex; Lovan; Philozac; Sarafem; Zactin

Definition

Fluoxetine is a selective serotonin reuptake inhibitor (SSRI) commonly used to treat major depressive disorder (MDD), obsessive-compulsive disorder (OCD), bulimia nervosa, panic disorder, and premenstrual dysphoric disorder (PMDD), among other disorders (Wenthur et al. 2013). Some research suggests that fluoxetine can be used to treat repetitive behaviors in individuals with autism spectrum disorder (ASD); however, findings are inconsistent and placebo-controlled studies are scarce (Accordino et al. 2016).

Fluoxetine (molecular formula: $C_{17}H_{18}F_3NO$) is an oral formulation commonly known by the brand name Prozac. It was discovered by Eli Lilly and Company in 1972 and was the first SSRI to be approved for medical use by the United States Food and Drug Administration (FDA) in 1987. The introduction of fluoxetine represented a medical breakthrough in the treatment of depression and offered greater efficacy and lower side effects compared with tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs). Indications for fluoxetine include adult and pediatric major depressive disorder, adult and pediatric obsessive-compulsive disorder, premenstrual dysphoric disorder, panic disorder, and depressive episodes in bipolar I disorder. Common side effects of fluoxetine include sexual dysfunction, nausea, headache, drowsiness, diarrhea, weight loss, and tremors. Individuals taking fluoxetine also may experience increased risk of suicidality, violent thoughts or behaviors, and aggression (Wenthur et al. 2013).

Selective serotonin reuptake inhibitors (SSRIs) block the reabsorption of serotonin (5-hydroxytryptamine; 5-HT) neurotransmitters in the brain, causing increased levels of serotonin to be available. Some researchers hypothesize that serotonin dysregulation plays a role in the pathophysiology of ASD. According to this theory, dysregulation of serotonin in early periods of development leads to cortical abnormalities and alters neurotransmission of serotonin (Hollander et al. 2012). Additionally, the similarities in clinical presentation between compulsions in OCD (for which SSRIs are a first-line treatment) and

stereotyped behaviors in ASD lead some researchers to believe SSRIs could be used in the treatment of ASD (Accordino et al. 2016; Hollander et al. 2012).

Currently, no medications are approved by the FDA for the treatment of the core symptoms of autism spectrum disorder (Hollander et al. 2005). However, some research suggests that fluoxetine may be used as a treatment to reduce restricted and repetitive behaviors (RRBs) in individuals with ASD. Open-label and retrospective studies generally suggest SSRIs may be successful in treating some symptoms of ASD in both adults and children (Accordino et al. 2016; Hollander et al. 2005). However, few placebo-controlled trials have been undertaken (Accordino et al. 2016).

Studies of SSRIs have shown significant improvements in adults with ASD. Currently, there is only one published double-blind placebo-controlled study exploring the use of fluoxetine for adults with ASD. In this study, researchers examined the use of fluoxetine to treat repetitive behaviors in 37 adults diagnosed with ASD. Hollander and colleagues found that – compared with placebo – individuals given fluoxetine showed greater improvement in repetitive behaviors on the compulsion subscale of the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), on the obsessive-compulsive symptoms rating on the Clinical Global Impression (CGI) scale, and on the CGI rating of overall improvement. Participants received a moderately high dose of fluoxetine (mean dose 64.76 mg/day) over the 12-week trial and experienced only mild or moderate side effects (Hollander et al. 2012). A placebo-controlled study of a different SSRI, fluvoxamine, for adults with ASD also found significant improvements in a variety of symptoms, including repetitive thoughts and behaviors, language, and maladaptive behaviors (Accordino et al. 2016).

Placebo-controlled trials of SSRIs in children have shown inconsistent results. Unlike findings with adults, a study of fluvoxamine for children with ASD showed low tolerability and a low success rate (only 1 in 18 participants showed improvement in target symptoms). Additionally, in a large-scale placebo-controlled study of the SSRI citalopram, children with ASD showed no

difference in improvement between the SSRI and placebo. While placebo-controlled studies of fluvoxamine and citalopram have not found positive results in treating children with ASD, some research supports the use of fluoxetine for children with ASD. A study exploring the use of low-dose liquid fluoxetine found the medication to be successful in treating repetitive behaviors in children and adolescents with ASD. In this placebo-controlled crossover trial, 45 children with ASD were randomized and 39 participants were included in the analysis. At the end of the 8-week treatment period of low-dose fluoxetine (mean final dose 9.9 mg), children showed improvement in repetitive behaviors as measured by the compulsions subscale of the Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS) with a moderate to large effect size (Hollander et al. 2005). However, an unpublished multicenter placebo-controlled study examining the use of fluoxetine to treat repetitive behaviors in 158 children with ASD found no significant improvements when compared with placebo. Some researchers suggest that the differences in efficacy and tolerability of SSRIs in adults versus children with ASD may be due to changes in serotonin functioning throughout development (Accordino et al. 2016).

See Also

- [Antidepressants](#)
- [Repetitive Behavior](#)
- [Serotonin Reuptake Inhibitors \(SRIs\)](#)

References and Reading

- Accordino, R. E., Kidd, C., Politte, L. C., Henry, C. A., & McDougale, C. J. (2016). Psychopharmacological interventions in autism spectrum disorder. *Expert Opinion on Pharmacotherapy*, 17(7), 937–952. <https://doi.org/10.1517/14656566.2016.1154536>.
- Hollander, E., Phillips, A., Chaplin, W., Zagursky, K., Novotny, S., Wasserman, S., & Iyengar, R. (2005). A placebo controlled crossover trial of liquid fluoxetine on repetitive behaviors in childhood and adolescent autism. *Neuropsychopharmacology*, 30(3), 582.
- Hollander, E., Soorya, L., Chaplin, W., Anagnostou, E., Taylor, B. P., Ferretti, C. J., . . . & Settiani, C. (2012).

A double-blind placebo-controlled trial of fluoxetine for repetitive behaviors and global severity in adult autism spectrum disorders. *American Journal of Psychiatry*, 169(3), 292–299.

- Wenthur, C. J., Bennett, M. R., & Lindsley, C. W. (2013). Classics in chemical neuroscience: fluoxetine (Prozac). *ACS Chemical Neuroscience*, 5(1), 14–23.

PRS-SA (Pragmatic Rating Scale-School Age)

- [Pragmatic Rating Scale](#)

PRT[®]

- [Pivotal Response Training](#)

PSAP2

- [SHANK 3](#)

Pseudoscience

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Synonyms

[Bad science](#); [Bogus therapy](#); [Quack science](#)

Definition

Pseudoscience is a term used to describe proposed theories and therapies that are described as evidence based but actually have limited or no scientific support. Examples of therapies for autism

whose proponents describe them as scientifically based, but, in the judgment of most experts, are currently not supported by scientific evidence include the following: biofeedback/neurofeedback, “energy” therapies, animal-assisted therapies, electroconvulsive therapy, withholding vaccinations, and facilitated communication (Thyer and Pignotti 2010). Other such therapies are elimination diets, vitamin therapy, and anti-yeast therapy. The evidence provided to support these therapies meets many of the criteria listed below, which can serve as red flags for recognizing pseudoscience:

- *Rationale or underlying theory is not based on existing data* (Lilienfeld 1998): The intervention is not connected to other bodies of scientific knowledge and does not build upon existing research. A treatment may be pseudoscience if it is recommended without research that points to a link between the variable being manipulated (e.g., yeast, vitamins, gluten, etc.) and the symptoms of autism. In place of this research, the authors of pseudoscience may instead use complicated, scientific-sounding language to explain why the intervention works.
- *Reversed burden of proof* (Lilienfeld 1998): Scientists typically place the burden of proof on themselves. That is, if they propose a relationship, it is their responsibility to try to find evidence to support this relationship. Pseudoscientists may place the burden of proof on others and require someone else to prove that the relationship *does not* exist. For example, proponents of anti-vaccination insist that others refute the hypothesis that autism is linked to vaccines.
- *When studies have been done, they suffer from methodological weaknesses* (Offit 2008): Strong studies will have multiple levels of control including large sample sizes, random assignment, and isolation of a single variable, while other variables are held constant. These characteristics make the results of a study more reliable. Studies without these levels of control that lead to generalizations and unqualified conclusions may be pseudoscience.
- *Reliance on anecdotal evidence and testimonial* (Offit 2008): Instead of referring to clinical trials as evidence that their interventions work, proponents of pseudoscientific interventions often give anecdotal examples of children and families treated with great results. Aside from the obvious problem of verifying these testimonials, anecdotal reports are problematic because the effects of an intervention on one child cannot be generalized to the entire population of children and adults with autism. Additionally, without isolating a single variable, there is no way to attribute improvement to the intervention. Improvements may be explained by another therapy or by normal growth and maturation.
- *Overinterpretation of results* (Offit 2008): At this time, there is no single known cause or cure for autism. An intervention that is touted as a “cure” or describes “recovery” may be pseudoscience. Additionally, the causes of autism are numerous & complex, a study singling out one “cause” of autism, without reference to complex and interacting factors should raise a red flag.
- *Results have not been replicated; replications may be difficult or impossible* (Offit 2008): Offit warns against reacting to a single study, especially if the study is the first of its kind or “appears to break new ground.” A reliable intervention will be supported by many studies replicating the same positive results. Furthermore, a good study will have a detailed methods section, allowing other researchers to replicate the study. Vague descriptions of the methods may prevent replication and indicate pseudoscience.
- *Unchanged by disconfirming research* (Bunge 1984): Although the original study that led to a proposed relationship between autism and vaccines has been discredited and several large studies have found no link, many anti-vaccination proponents continue to believe in the relationship. Similarly, specific

interventions (e.g., elimination diets) may still be encouraged after several studies fail to find evidence for their support.

- *Conflict of interest* (Offit 2008): Most peer-reviewed journals require authors to report funding source and potential conflicts of interest. An example of a conflict of interest is a scientist receiving income from a drug company to show that a drug manufactured by that company is effective; the scientist's financial interest (i.e., income) may compete with his scientific interests and cause falsification or omission of data. Another example is an intervention that requires families to pay for a product or service, outside of an established medical or educational system.

See Also

- [Facilitated Communication](#)
- [Gluten-Free Diet](#)
- [Nutritional Interventions](#)
- [Vaccinations and Autism](#)

References and Reading

- Bunge, M. (1984). What is pseudoscience? *The Skeptical Inquirer*, 9, 36–46.
- Jacobson, J. W., Foxx, M., & Mulick, J. A. (Eds.). (2005). *Controversial therapies for developmental disabilities: Fad, fashion, and science in professional practice*. Mahwah: Lawrence Erlbaum.
- Lilienfeld, S. O. (1998). Pseudoscience in contemporary clinical psychology: What it is and what we can do about it. *The Clinical Psychologist*, 51, 3–9.
- Offit, P. A. (2008). *Autism's false prophets*. New York: Columbia University Press.
- Thyer, B. A., & Pignotti, M. (2010). Science and pseudoscience in developmental disabilities: Guidelines for social workers. *Journal of Social Work in Disability & Rehabilitation*, 9, 110–129.

Psychiatric Problems and Care in Older Adults with Autism

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Although autism spectrum disorders (ASDs) are often conceived as lifelong disabilities, little is known about their impact on individuals, families, and society as the affected persons grow old. Research in ASD is still only seldom concerned with middle-aged or older people. There are several follow-up studies showing that these disorders, which in all cases are present at an early age, are relatively stable from childhood to adolescence, and, especially if accompanied by intellectual disability (ID), into adulthood. Although two studies of the same Swedish cohort with normal IQ ASD ($n = 47$) showed that “caseness” slowly diminished (Helles et al. 2015), at mean age 30 years 76% still met criteria for an ASD.

In a review of 23 follow-up studies, Howlin and Moss (2012) found that the adult outcome of ASD in individuals with IQs in the normal range was described as unfavorable in the majority of cases. Only two of the 23 studies in the Howlin and Moss review comprised any individuals older than 50 years, and the larger part of all research in ASD is still conducted on children. Thus very little is known about ASD in people older than around 50 years (Bennett 2016), and only recently has work been commenced in this area, primarily in the UK, the USA, and the Netherlands. However, the knowledge concerning this group is still very limited, and there are many unanswered questions concerning the prevalence of ASD in these age groups, symptom persistence and variance, life circumstances and quality of life, concomitant illness and disorders, as well as services and care needs. It can be assumed that ASD in older adults to a large part is undiagnosed, since there is little reason to believe that the

PSI Test

- [Pediatric Speech Intelligibility Test](#)

prevalence of autism in older adults is much lower than 0.5–1% – the lower number if the Swedish follow-up studies mentioned above is taken into account (Brugha et al. 2011; Helles et al. 2015).

In the most recent years, ASD advocates have been pointing to the scarcity of knowledge concerning autism and aging, and efforts have been made to elucidate the problem. Several studies concerning neuropsychology, for example, by Dutch researchers, have been made. Other authors have investigated concomitant somatic and psychiatric diagnoses and use of services and care. These latter studies are usually made as register studies, with the pros and cons that are inherent to this methodology.

A number of nationwide register studies comprising a cohort of all Swedish elderly (born 1957 or earlier) community service users with intellectual disability (ID) and/or ASD have recently been made by Swedish researchers (Nylander et al. 2018). Of all registered individuals ($n = 7936$), the number with ASD diagnoses was 601, 64% males and 36% females. Concomitant ID diagnoses were registered in 256 cases. Fifty percent of the ASD group had any other psychiatric diagnosis, most often affective or anxiety disorder, and 63% had been in contact with specialist psychiatric care during the 10 years studied. Male gender, concomitant ID, and especially an Asperger syndrome diagnosis raised the OR for psychiatric care. Prescriptions of psychotropic drugs, in particular antipsychotics, were very common (more than 70%, in contrast to 12% having a diagnosis of psychotic disorder), and 24% had four or more psychotropic medications.

In summary, the so far scarce research into ASD in the elderly suggests that ASD is underdiagnosed and probably under-recognized in many cases and that older adults with ASD, or at least some of them, are a vulnerable group whose needs may be unmet.

A Swedish psychologist who made a pioneering work interviewing a group of elderly persons with ASD found that some of them expressed gratefulness for not having been diagnosed and “interfered with” when they were children. This implies that we should be careful to see the person and not only the diagnosis and,

consequently, offer only services that are wanted by the person and which we are certain would make a positive difference.

Definition

Autism in older adults is not yet well-known or researched.

References and Reading

- Bennett, M. (2016). “What is life like in the twilight years?” A letter about the scant amount of literature on the elderly with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46, 1883–1884.
- Brugha, T. S., McManus, S., Bankart, J., Scott, F., Purdon, S., Smith, J., Bebbington, P., Jenkins, R., & Meltzer, H. (2011). Epidemiology of autism spectrum disorders in adults in the community in England. *Archives of General Psychiatry*, 68(5), 459–465.
- Helles, A., Gillberg, I. C., Gillberg, C., & Billstedt, E. (2015). Asperger syndrome in males over two decades: Stability and predictors of diagnosis. *Journal of Child Psychology and Psychiatry*, 56, 711–718.
- Howlin, P., & Moss, P. (2012). Adults with autism spectrum disorders. *Canadian Journal of Psychiatry*, 57, 275–283.
- Nylander, L., Axmon, A., Björne, P., Ahlström, G., & Gillberg, C. (2018). Older adults with autism spectrum disorders in Sweden: A register study of diagnoses, psychiatric care utilization and psychotropic medication of 601 individuals. *Journal of Autism and Developmental Disorders*, 48(9), 3076–3085.

Psychiatrist

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Definition

A physician specializing in the evaluation and treatment of mental disorders. In the USA and Canada, these specialists have received M.D. or

D.O. degrees. Following specialty training, board certification is available in general psychiatric and a variety of subspecialties including child and adolescent psychiatry. The latter group often has the most experience with individuals with disabilities (of all ages), and all child and adolescent psychiatrists in the USA have received training in adult psychiatry as well. Forensic psychiatrists specialize in work with courts and the legal system. Training systems differ somewhat in other countries. Psychiatrists work to evaluate, diagnose, and treat a range of disorders with a range of methods including pharmacological (drug) and other treatments. For individuals with autism, a variety of comorbid psychiatric conditions may require such treatment, and a handful of medications have now been specifically approved for use in autism. These individuals also have special expertise in working with other medical professionals around other conditions the individual might exhibit, and this can be particularly important in situations where the person exhibits a medical problem with behavioral aspects such as epilepsy. It is important that the psychiatrist has experience in the evaluation and assessment of individuals with autism (Ghaziuddin et al. 1992; McCracken et al. 2002; Scahill and Martin 2005; Tuchman and Rapin 2002).

See Also

► [American Psychiatric Association](#)

References and Reading

- Ghaziuddin, M., Tsai, L., & Ghaziuddin, N. (1992). Comorbidity of autistic disorder in children and adolescents. *European Child & Adolescent Psychiatry*, 1(4), 209–213.
- McCracken, J. T., McGough, J., Shah, B., Cronin, P., Hong, D., Aman, M. G., et al. (2002). Risperidone in children with autism and serious behavioral problems. *New England Journal of Medicine*, 347(5), 314–321.
- Scahill, L., & Martin, A. (2005). Psychopharmacology. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, pp. 1102–1122). Hoboken: Wiley.
- Tuchman, R., & Rapin, I. (2002). Epilepsy in autism. *Lancet Neurology*, 1(6), 352–358.

Psychoanalyst

► [Psychodynamic Theories](#)

Psychodynamic Theories

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Synonyms

[Psychoanalyst](#)

Definition

Psychoanalysis

In their classic book on the *Language of Psychoanalysis*, Laplanche and Pontalis define psychoanalysis as a therapeutic approach developed by Sigmund Freud with a characteristic method of investigation (“free association”) that Freud claimed identified emotions and emotional conflict in the psychoanalytic relationship, a theory that attributes experience and behavior to conflicts between emotional impulses or instincts of which we are not directly conscious and the efforts that we make to block them from influencing us, and a psychology that is based on the theory and justified by the investigative method.

Psychodynamics

The psychology developed within psychoanalysis has also been called dynamic psychology, or psychodynamics. A basic premise of psychodynamics is that actions and reactions need energy and that this is derived from a well of energy, or drive, of which a person is not directly conscious or, as psychoanalysts would say, that are “in the unconscious.” These drives, like the hot fluids from the earth’s mantle, may break through a crust of repression and come into the light of consciousness,

albeit changed. An easy and nondisruptive discharge of this energy on a social appropriate object is healthy: blocked discharge or a discharge that causes damage through its force or its trajectory is unhealthy. Psychodynamic theories of autism are therefore based on the vicissitudes of drive energy supplemented in the post-Freudian era by energy arising from a new source (corresponding to a more recent theory of motivation) – the need for another person.

Freud originally supposed that it was sexual desire that was the most important drive (at least in people who were not starving or parched with thirst). So his psychodynamics concentrates on the vicissitudes of sexual desire during development. The open expression of sexual desire is inhibited by the primary carer, he thought, at least until the child develops some personal awareness (Freud attributes this to the creation of an “intrapsychic structure,” the Ich; Lacan calls this a new order of representation, l’Imaginaire, ushered in by the growing awareness of the father) and later some social awareness (again, attributed to an “intrapsychic structure,” the uber-Ich; Lacan calls this order, le Symbolique, and considers that it is ushered in by the name of the father). Freud supposed that there was sexual desire from birth, but at birth, it was directed toward the baby’s own skin. This development is easily derailed and so fails to develop normally, getting stuck or “fixated.” Early derailment was supposed to be associated with psychosis (no evidence was given for this assertion nor for the apparently normal development of people who developed psychoses in adulthood). Lacan thought that normal development required the acquisition of the name of the father, i.e., of the “name of the father,” and that a failure to acquire this led to a persistent susceptibility to imaginary rather than symbolic representation.

Lacan’s theory seemed to provide a neat explanation of the lack of language development in children with Kanner’s syndrome although it was not clear why it did not also apply to children who were normally socialized but had severe speech and language disorder. Freud’s theories were equally facile, but this time, in explaining the touching, sucking, poking, and self-stimulatory stereotypies associated with children

with Kanner’s syndrome. Again, it was not explained why it applied so well to children with intellectual disability and neglected children if it was to be a theory of ASD.

Autoerotic

The earliest phases of emotional development were the ones linked by Klein, a follower of Freud, to “psychosis” (never a clearly defined term in psychoanalytic literature) and to autism, which was considered to be a kind of psychosis. Freud had termed the earliest phase of sexual development when the infant was supposed to be erotically gratified by its own body, “autoerotic.”

“Autism”

Bleuler had, like Sigmund Freud, studied with Charcot, at Salpêtrière. He wrote a favorable book review (Bleuler 1896) of the monograph by Freud and his mentor Breuer that described their use of the hypnotic method to treat anxiety-related disorders and which launched psychoanalysis as a treatment. His assistant, Jung, and he had corresponded with Freud and had devised a word association experiment to test Freud and Breuer’s theory of repression which led Jung to formulate the concepts of “complexes,” for words that were delayed in recall, as against “simplexes” which were repeated with minimal delay. Bleuler moved away from Freud from about 1911 on, just after the publication of his textbook in which he introduced the word “autism” to a wider audience (Bleuler 1911). Bleuler gave his reasons for the distance in 1913 (Bleuler 1913), but he remained supportive of psychoanalysis, and in 1914, Freud could cite him (Freud 1914) *as an influential sympathizer*: “After this it was impossible for psychiatrists to ignore psycho-analysis any longer. Bleuler’s great work on schizophrenia (1911), in which the psychoanalytic point of view was placed on an equal footing with the clinical systematic one, completed this success” (Bleuler 1911, p. 28).

The term “autism” was coined by Bleuler in 1907 and was derived from Freud’s term “autoeroticism.” Freud wrote to Jung (Freud and Jung 1974) that “Bleuler still misses a clear definition of autoeroticism and its specifically psychological

effects. He has, however, accepted the concept for his Dem[entia] pr[aecox] contribution to Aschaffenburg's Handbook. He doesn't want to say autoerotism (for reasons we all know), but prefers 'autism' or 'ipsism'" (Freud and Jung 1974, pp. 44–45).

What Freud's correspondents would "all have known" is now forgotten, but there was no doubt that Bleuler used the word "autism" knowing that it implied a psychological cause for schizophrenia. Indeed, his treatment method, and that of his son, Manfred, attempted to link the psychological approaches of Freud and Jung with the organic approach of Kraepelin.

Historical Background

The psychoanalytic roots of the autistic spectrum disorders (ASDs) lie in the development of schizophrenia, or rather of dementia praecox, at the end of the nineteenth century and some 40 years before ASD was first described. That century had seen a conflict between the Romantic view of psychiatric disorder that attributed it to hidden mental problems and the organic approach that attributed it to pathological changes in the brain (Ellenberger 1970). It was widely recognized in the nineteenth century that the reasons for many actions were "unconscious" but determined by learned associations (Thomson 1964). The Romantics sought to correct these associations by rewards and punishments, believing that in this way mental illness could be cured. Paradoxically, this could lead to the development of coercive treatment that was often cruel and abusive, although not always. Samuel Gaskell, physician superintendent of Lancaster Moor Hospital in the UK, gave "disappointment in love" as the most common reason for admission of women to the asylum but also advocated conditions that would enhance the dignity and autonomy of his patients as much as possible (Tantam 1990).

The same paradox has reasserted itself in ASD a century later, where expectations of cure have sometimes led to cruelty and harm, although this has not been restricted to the proponents of a psychological cause for ASD.

The opponents of the Romantic approach, the biological psychiatrists, were much more negativistic, believing that mental illness was due to degeneration of the brain, with a number of them concluding that this degeneration was both hereditary and progressive. Enlightened biological psychiatrists thought that as their patients were incurable, they should be given the conditions most likely to mitigate their condition. This could be interpreted benignly, as it was by Gaskell, but also led to the development of the "institutions" where people were simply sequestered with minimal attention to their health and safety, and none to the possibility of recovery. Institutions became a byword for neglect, environmental poverty, and a lack of social stimulation that contributed so much to the clinical presentation of older children and adults with an ASD who had been "kept" in these conditions. However, there was an alternative approach that stressed what we would now call "normalization," but would then have been called "moral treatment."

In the midst of this confusion, it was difficult to make any scientific progress on which treatments worked for who – if any did – or what the course of a person's particular condition or problem would be. Griesinger, the father of the biological psychiatry approach, pinned his faith in pathological studies of the brain and that proved well founded for "general paralysis of the insane" a psychosis caused by syphilitic infection. But the pathology of other psychoses, the so-called functional psychoses, remained elusive, as it does today.

Again, there are parallels with ASD. There are some genetic conditions that are known to have a high risk of conferring ASD, and the neuropathology of these is increasingly well understood. But the pathology or pathologies characteristic of most of the ASDs remain obscure. As a result, the future of an individual with an ASD, and therefore knowing what would best help that person, remains unclear.

Emil Kraepelin's answer to this situation was to undertake a kind of case-control study, selecting the records of patients with a poor outcome and comparing previous notes made of their presentation and course with those in the records

of patients with a good prognosis to see if there were features that could predict the outcome. He coined the term “dementia praecox” for this group, a term that did not find favor in the UK or the USA. Anglophone psychiatrists accepted Kraepelin’s concept of the remitting “manic-depressive” psychoses but thought that Kraepelin had been too nihilistic about the other group of functional psychoses, for which they much preferred Eugen Bleuler’s term “schizophrenia,” introduced by Bleuler in 1911 (Bleuler 1911).

It is at this point that the history of psychoanalysis begins, as well as the history of the term “autism” which psychoanalysis inspired.

Current Knowledge

Kanner, in applying Bleuler’s term “autism” to children in 1944, did so knowing that it evoked a psychological causation. But by this time, Freud’s specifically sexual description of infant development, and the centrality of the vicissitudes of sexual energy or libido, had changed with the impact of Melanie Klein and the object relations theorists. She had, confusingly, adopted a term developed by the Scottish psychoanalyst and medical anthropologist Fairbairn – “schizoid” – to describe the stage of development shortly after the stage that Freud called “autoerotic.” This term had also replaced “autism” in the second edition of Bleuler’s textbook on schizophrenia. It is not clear whether Bleuler read Fairbairn, or Fairbairn read Bleuler. Bleuler’s textbook was only translated into English in the 1950s, and Fairbairn’s only published in the 1950s, too although he had given his paper about schizoid personality in the 1920s. But the shared etymology – both are derived from the ancient Greek for split – suggested to many commentators that there was a fundamental connection.

Muddying the Waters: Schizoid Personality, Autism, and Schizophrenia

Inferring a connection of things from their resemblance has a long history in medicine. The use of mandrake (*Mandragora* sp.) as a “sympathetic” treatment comes, for example, from its forked root

that was said to resemble a pair of legs. Mandrake looked to the credulous eye like a homunculus and so was assumed to have some human qualities (see, e.g., the references to it “screaming” when uprooted in the Harry Potter novels). Freud himself characterized such metaphorical or “iconic” reasoning to be characteristic of “the hysteric” and the dreamer (Freud 1965), but it has been a fallacy into which many psychoanalytic theorists have slipped.

The resemblance of autism in schizophrenia and early childhood autism created a connection between the two that led to ASD being rechristened early childhood schizophrenia for a number of years. Although some research groups do identify childhood-onset schizophrenia in children as young as age 7, they also consider that there are key symptoms can distinguish between the two and that the outcomes are different. So more narrowly defined schizophrenia and autism do not overlap, as Kanner indicated in his first description although they may occur together.

Psychoanalysts might also refer to schizophrenia but on the basis of the presence of schizoid traits. One of the splits in the British Psychoanalytic Society that Melanie Klein provoked was that she would use the term schizophrenia in such a loose way that medically qualified analysts, notably Edward Glover, wanted nothing to do with her. He reportedly said to Hannah Segal at her assessment for a training there that “In that case [Segal had begun her analysis with Klein] I have nothing to do with you! They’ve got their people; we’ve got our people. Goodbye” (<http://www.melanie-klein-trust.org.uk/segalinterview2001.htm>).

German psychiatrists used the term “schizoid” to include sensitivity, “introversion” (the term coined by the follower of Freud, CG Jung), and emotional detachment (Kretschmer), although there were many differences in detail and these are now reflected in the confusing DSM-IV descriptions of schizoid and schizotypal personality. They attributed it to temperament that was an expression of the broad phenotype of a genetic disposition to schizophrenia. This link has never been adequately established, however, and in clinical practice, depression seems to be a more commonly linked mental illness. Analysts attributed

schizoid personality to a disorder of object relations, and the research evidence points to that being the case (Lenzenweger 2010) although it also suggests that there is a hereditary, temperamental component, too (Cohen et al. 2010).

The term “schizoid” would not be relevant if it were not for the persisting notion that Asperger syndrome and schizoid personality disorder in children are different descriptions of the same condition (Wolff 2000). I have argued that people with AS may be more at risk of developing schizoid traits but that the two are quite distinct: One is a developmental disorder of brain development; the other is an emotional disorder, a disorder of mental development (Tantam 1988). However, the analytic stance on schizoidia may have reinforced the notion that autism, too, was psychogenic.

Getting information about outcome is particularly difficult for clinicians and researchers who see children or adults once or only at one stage of their development. This is commonly the situation for psychotherapists, including psychoanalysts. It is an even greater problem when their observations are confined to a few, albeit intensively studied, individuals – again a problem for psychoanalytic theorists.

Psychogenic Theories of ASD

Freud’s original formulation of an inner, autochthonous sexual drive continues to find some support as an explanation of deviant sexuality, but it was otherwise quickly replaced by post-Freudian accounts, except in the work of the influential French psychoanalyst, Jacques Lacan, who combined it with a semiological theory. He argued that drive had to be coupled to signification, in order to fill the “lack,” *béance*, that unsatisfied drive created. The final stage of signification, when drives emerge into our conscious awareness and we can find conscious ways of satisfying them, is its linkage to language and the symbolic order or “*le nom du père*.” Autism, as Lacan thought, occurs because this does not happen. He is credited as saying that the child with autism is too taken up with talking to himself that he cannot talk to others and also that when a child with autism hears his speech, it sounds like a hallucination, and he covers his ears to protect himself

from it (Tendlarz). It is tempting to think that Lacan may have been aware of Sir Michael Rutter’s briefly held view that autism was a language disorder. The linkage of desire to language is, in Lacanian terms, the “fourth stage of the cycle of the drive” that is the stage when, according to the solipsistic Freudian theories of infant development, the infant discovers the reality of other people (Lacan 1977). Some Lacanians attribute autism to a failure at the third cycle of the drive, leading to a developmental arrest at the stage before other people are recognized (Laznik 1993).

Developmental arrest is a very common psychoanalytic theme. Psychoanalysts suggest that development stops either because it “is fixated” because desire is too fully gratified (Freud postulated a series of sexually gratifying nursery nurses unknowingly fixating precociously prurient little boys) or because it is thrown back or regressed to an earlier stage by a disappointment, of which the type is the “Oedipal situation.”

In the 1920s, Freud began to consider that there was also a death instinct or rather an instinct to kill, as well as a sexual instinct, which he broadened into an affiliative or cooperative instinct. In his answer to Einstein’s question, “Why war?,” Freud thought that this destructive instinct might be overcome by an increase in the positive instincts as actions, Freud now thought, were motivated by the balance between these often opposing instincts. Kurt Lewin, who worked at the Institute of Social Relations in Frankfurt before moving to the USA, would have been familiar with these ideas, as the “Frankfurt school” was strongly influenced by psychoanalysis, and his formulation of the “approach-avoidance conflict” demonstrates this. For a short while, ASD was attributed to an irresolvable approach-avoidance conflict.

Freud’s idea of the protective effect of social solidarity did not apply to the Austria from which he fled in 1938 or any of the other Fascist powers which seem to have experienced an increase in in-group social harmony in proportion to their out-group violence. It may not be unrelated to this that the differences of children with autism from neurotypical children were first noted by both Asperger and Kanner in 1938, although

their observations were not widely published until just before the end of the war when some of the terrible civilian consequences of the war were becoming all too apparent.

The casualties of war included children orphaned, women bereft, men broken in the stress of battle, and people of all ages dehumanized. Psychoanalysts were involved in dealing with these crises. Fairbairn, who had described schizoid personality, worked with men who went absent without leave, for example. These war experiences changed the thinking of many health professionals dealing with children, and several of them saw a similarity between children with ASD and children who were traumatized in the war. Bruno Bettelheim, for example, who claimed to have been psychoanalyzed in Vienna and used Freudian concepts in his work, likened children with ASD to prisoners in a concentration camp and wrote that the mothers of these children were like the SS guards (Bettelheim 1967). His work did much to fuel the stigmatization of parents by professionals, particularly those that were psychoanalytically orientated, and in the end brought psychoanalytic approaches into disrepute, particularly after Bettelheim committed suicide and some of his ex-patients accused him of physical abuse of them.

Stress-Diathesis

Fairbairn's approach to men who went AWOL was that the stress of war brought out a latent overdependence on the mother and led to incapacitating separation anxiety. This is an example of what two other analysts called the "stress-diathesis" model (Grinker and Spiegel 1945): that a hidden vulnerability acquired in early development could lead to disorder if disclosed by a later stress. Over subsequent years, the most commonly postulated diatheses have been neurological and not, as originally proposed, psychological but the notion that stress can disclose ASD – an originally psychoanalytic concept – is still being propounded (Pfaff et al. 2011).

Few people would now discount Adolf Meyer's idea that circumstances, environment, and brain interact. So the stress-diathesis seems completely in harmony with this holistic

approach. But psychoanalysts are often normative thinkers, and any deviation from statistical normality has often been considered to be a diathesis that could lead to disorder. The evidence from neuroscience or genetics does not support this. Diversity in the brain and in the genes has survival value for the group because it allows for rapid evolution and for role differentiation within the group that confers adaptability.

Maternal Deprivation and Affective Contact

Curiously, the same 1948 issue of the *Journal of Mental Science* that contained a review of *Men Under Stress* (Issue 94) also contains review of a book on the psychology of the unwanted children by Agatha M. Bowley.

The war substantially increased the number of children without families: unwanted children, orphaned children, displaced children, evacuated children, all of them were more vulnerable to distress. In 1949, the World Health Organization, recognizing this, commissioned a psychoanalyst and director of the child psychotherapy training program at the Tavistock Clinic in London to report on these children. He concluded that there was a characteristic psychological reaction to what came to be called "maternal deprivation." The idea received powerful support from the cruel experiments of Harry Harlow who raised rhesus infants in isolation providing them with a series of inanimate mother surrogates. The maternal deprivation hypothesis was effectively laid to rest by Rutter (1981) but by this time had already been succeeded by Bowlby's much more nuanced attachment theory, developed in conjunction with Mary Ainsworth, among others. Attachment theory was partly inspired by *King Solomon's Ring*, a book of essays by the naturalist Konrad Lorenz (later joint recipient with Nico Tinbergen of a Nobel Prize for his part in creating ethology). Lorenz was brought up in a remarkable family who seem to have kept a menagerie in their Vienna flat. He was adopted by a family of goslings after their mother had died and postulated that an infant gosling imprinted on the first animal that they saw moving after they had hatched. Bowlby thought that this was an example of a more general process of "bonding" for which

there was intrinsic disposition – now thought to involve oxytocin and perhaps vasopressin surges in the mother – leading to grooming of the infant and a disposition to respond to a species-specific cry in the infant. Grooming by the mother changes the infant’s amygdala so that the infant treats the mother as a unique source of comfort from anxiety or threat and draws near to her protection under these conditions. Oxytocin probably mediates the proverbial savagery of a mother’s aggression when protecting her young, too. Bonding and oxytocin remain active areas of research in ASD.

Bowlby’s work was anticipated by Kanner. Kanner (1943) reported on 11 children, the first of whom, Donald, he saw in 1938. Many of these children would now be diagnosed as having Asperger syndrome.

Kanner’s descriptions are uncannily good. He noted that the 11 children had previously been diagnosed as having intellectual disability or schizophrenia but, as Kolvin later confirmed (Kolvin 1971), that schizophrenia could not apply as there was no period of normal development (although early development is still an important differentiator of ASD and schizophrenia, a period of early development is no longer considered unusual in ASD).

Kanner considered that the children that he was describing had a condition not previously described (he did not know of Asperger’s work), and he wrote that it was an “autistic disturbance of affective contact.”

Affective contact corresponds to Lorenz’ imprinting, and Bowlby’s attachment, but unlike Freud’s ideas of a sexual instinct which created a bond only when it had been shifted by learned experience from being directed to the self to being directed toward the mother, affective contact was purely biological. Kanner wrote that “... these children have come into the world with innate inability to form the usual, biologically provided affective contact with people.”

It is not clear why Kanner chose the words affective contact. They had been used in the 1920s in relation to schizophrenia <http://bjp.rcpsych.org/cgi/reprint/75/310/526-b> but also in relation to religious experience <http://www.jstor.org/pss/3745867>. (Flower 1929), but I could not

find any other reference to their use except, possibly, by the psychoanalyst and originator of the sandbox technique of play therapy, Margaret Lowenfeld, who began her career as a research scientist studying mother’s milk before becoming a child psychotherapist.

Blaming the Mother

Psychoanalytic approaches to autism have included one or more of five assumptions (see Textbox).

Textbox

Psychoanalytic assumptions leading to a psychogenic theory of ASD

That autism is a normal developmental stage

That autistic disorder is either an arrest or a regression to an earlier developmental stage as a result of some kind of stress, shock, or trauma

That the child has a diathesis to arrest or regression in the face of this stress, shock, or trauma that is psychological

That the stress, shock, or trauma most likely to be involved is separation from the mother

That rejection by the mother without physical separation may be enough stress, shock, or trauma to cause autism

Kanner’s 1943 description gave credence to assumption 5 (see Textbox), by his choice of the term “autistic” in the first place and by suggesting that emotional deprivation played a part in the “autistic disturbance”: “One other fact stands out prominently,” Kanner writes “In the whole group, there are very few really warmhearted fathers and mothers.... The question arises whether or to what extent this fact has contributed to the condition of the children.” Kanner strengthened his view that ASD could be psychogenic in a later paper (Kanner 1949) in which he first used the term “refrigerator parent” and gave credence to both elements 2 and 5 (Textbox) in his thinking. He wrote of children with ASD that “Their withdrawal seems to be an act of turning away from such a situation to seek comfort in solitude,” the situation being that “They were the objects of observation and experiment and not of genuine warmth and enjoyment.” Kanner thus gave his authority to a psychogenic account of the etiology

of autism and opened the way for the extremes of those like Bettelheim (1967) who would accuse the parents of children with ASD of being unfit, remove their children from them, and then, sometimes – or so some of those children have alleged – treat those children abusively. Bettelheim was not an analyst but was strongly influenced by Freudian theory.

Kanner's formulation of the children regressing in the face of some kind of trauma was entirely consistent with psychoanalytic theories of ASD at the time and indeed up to the 1980s. Frances Tustin's work was particularly influential in the UK and Lacan in France. Tustin had trained in a program directed by John Bowlby but remained resolutely Kleinian in her outlook, and she included all of the five elements mentioned in her early theories. She thought that the infant had to deal with both the life and the death instincts toward the mother. This was consistent with the object relations school of psychoanalysis, championed in the UK by Melanie Klein in opposition to more traditional approaches championed by Freud's youngest daughter, Anna. Klein's analysis of "Dick" described in *The Psychoanalysis of Children* (1932). In *The Writings of Melanie Klein*, Vol. 2. London: Hogarth Press 1975 is often taken as the starting point for psychoanalytic approaches to the treatment of ASD. Klein had previously analyzed her younger son, Erich, and was just completing her own analysis with Abraham. "Dick" was suffering from a psychological disorder that had been diagnosed as schizophrenia, but Klein considered that his mother had rejected him from birth and that as a result his "...symbolism had not developed. This was partly because of the lack of any affective relation to the things around him, to which he was almost entirely indifferent" (Klein 1975, pp. 223–224). Klein felt that this incapacity to symbolize was related to the abnormality of his "object relations," and what appeared to be a failure to understand people as people, toward whom he could show "opposition and obedience" or be "intelligible" (Klein 1975). She interpreted this impairment as being a result of "anxieties rooted in the child's relations with others..." and as "a manifestation of defenses

against his own destructive impulses toward the mother's body" (Morra 2002, p. 287).

Tustin's original formulation (in *Autism and Childhood Psychosis*) was that birth and bodily separation from the mother could lead to such negativity toward the mother that development was arrested at the "autistic" stage. Later, she detected "encapsulated autism" in children with many other conditions (*Autism and Childhood*). Toward the end of her career, she thought that autism was not a normal stage of development, a conclusion that another psychoanalyst who treated children with an ASD, Margaret Mahler, had come to much earlier. Mahler, unlike Tustin, had also concluded that children with an ASD had a biologically based disorder of affective contact and routinely included the mother along with the child in her analyses.

War Brings People Together

The impact of the Second World War was not confined to a front line of fighting men. Rapidly advancing armor and airplanes could spread it much more widely. As I noted earlier, many professionals were familiar with the civilian trauma that was a consequence of this. Children were often at much at risk as adults, and the image of the desolate and traumatized child has rarely been out of the newspapers, and out of our consciousness, since. The images of children traumatized by war have been joined by those of children beaten or neglected by carers, or institutionalized in Romanian, or Russian, orphanages. Psychoanalysts made important contributions to this awareness. Spitz wrote of institutionalism in children, and Bowlby of the consequences of "maternal deprivation." Maternal deprivation led, in Bowlby's view, to a lack of empathy and of emotional response that he called "affectionless psychopathy." He may have been influenced in this description by the interposition of a nanny or series of nannies between mother and child that was typical of an English upper class upbringing as well as by the wealth of research that he had reviewed for his WHO monograph (later published as Bowlby, J. (1951). *Maternal care and mental health* (p. 89). New York: Schocken).

The basic assumption in psychoanalysis that developmental disorders, like psychoses, were mental and not biological led many of its practitioners to reject Bowlby's idea that maternal deprivation affected the brain directly. They preferred Klein's idea that the child imagined what to expect of a carer and felt hostility toward a carer who did not come up to these expectations. Bowlby was ostracized in the British Psychoanalytic Society following three seminars that he had on attachment to the society, although he was rehabilitated after his appointment as Freud Professor of Psychotherapy at University College, London, in 1980. Since then, attachment theory has become mainstream.

Psychoanalysts and other psychotherapists often assumed that maternal deprivation could cause ASD. There were superficial similarities between children with an attachment disorder and children with an ASD although follow-up studies of neglected and traumatized children often withdrew from social interaction. They rocked, mumbled unintelligibly, masturbated or self-stimulated (echoes of autoeroticism), and related to objects but not to people. Follow-up studies of neglected children from Romanian and Russian orphanages provide strong evidence against these children with "attachment disorders" having the same course as children with an ASD, and therefore against the two conditions having a shared etiology. However, these findings could only have been made after a substantial number of children from Romania and Russian being adopted in Western Europe and North America, thus providing a natural experiment of the effects of early neglect. This only happened recently. So psychoanalysts could and, as we have seen, did fall into the same fallacy as Freud himself in thinking that similar symptoms meant a common cause.

Given the assumptions that the effects of maternal deprivation were purely psychological, and that maternal deprivation could cause ASD, it was not surprising that many psychoanalysts took the view that a child with ASD had been made that way by their parents. It was not the parents' objection to being so stigmatized that changed this, but

psychoanalysts' acceptance that Bowlby had been right, if not about maternal deprivation but about its successor, attachment theory. Most psychoanalysts now accept that there is a biological basis to ASD although continuing to believe – as indeed do most practitioners – that there are familial influences that might affect its expression. Some try to bolt on Kleinian concepts almost in their entirety to this biology, however, as if reluctant to yield the possibility that autism is really a mental disorder.

Recent Developments in Psychoanalysis

Bowlby's rehabilitation and the incorporation of attachment theory into mainstream psychoanalysis have been followed, either coincidentally or synchronously, by a reversed trend from the previous years. Scientific theories about ASD have been adopted into psychoanalysis, rather than psychoanalytic theories being applied to autism. Psychoanalysts are now taking developments from research in ASD and applying them to their own theories of mental functioning, applying these to neurotypical as well as people with ASD.

"Theory of mind" has been particularly susceptible since it is already a cognitive rather than a brain-based theory. Theory of mind sounds almost like "mindfulness," and in the way that thinking in this area seems to proceed, the two have been subsumed into a new kind of psychoanalytic approach that is targeted at a political priority, so-called borderline personality disorder (Fonagy and Bateman 2006).

Neuroscientists have also suggested that the mirror neuron theory of contagious emotion might also be applicable in psychoanalysis (Scalzone 2005), but this brain-based theory has met with much less response.

Future Directions

Alexithymia

Alexithymia was first posited by two Bostonian psychotherapists, Sifneos and Nemiah. They were struck that some people with psychosomatic disorders who got worse with psychoanalytic

treatment, rather than better. They attributed this to the inability of these people to put their emotions into words, that is, a lack of words for feelings, or alexithymia (Sifneos 1972). Frith suggested that alexithymia might also affect people with Asperger syndrome (Frith 2004). Like impaired theory of mind in anorexia nervosa, there is evidence that alexithymia might be particularly likely to occur as a result of disorder (Tantam et al. 1982) rather than as a cause of disorder. So, although it is important for psychotherapists to bear in mind that people with an ASD may be uncomfortable dealing with emotion words, it should not be assumed that this discomfort has led to their ASD but may be a consequence of a lack of previous narrative with other people about emotions. Psychoanalytic treatment relies heavily on analyst and participating in a discourse with the analyst about the emotional connotations of the words that they use. Alexithymia blocks this, meaning that analytic interpretations based on these connotations may be experienced by the analyst and as mysterious and threatening. The negative effects of psychoanalytic treatment of adults with an ASD, like psychoanalysis of people with schizophrenia, may be a consequence of this. Other person-centered or existentially orientated methods are more successful since they emphasize practical solutions.

Intersubjectivity

Relational psychoanalysis has emerged as an influential approach (Stolorow et al. 2002), especially in the USA. Its proponents sometimes locate its origins in the work of Guntrip and his two analysts, Winnicott and Fairbairn, already mentioned as the first psychoanalyst to use “schizoid” in psychoanalysis. Other influences are Laing, himself influenced by the Scottish school begun by Fairbairn (Sutherland 1989), and also Kohut. Existential analysis (van Deurzen 2002) was a strong influence on Laing and through him, on relational psychoanalysis, too.

A key concept for relational psychoanalyst is “intersubjectivity,” a term coined by Husserl (1973, originally published 1905). Stolorow,

Atwood, and Orange define this “. . . a field theory or systems theory in that it seeks to comprehend psychological phenomena not as products of isolated intrapsychic mechanisms but as forming at the interface of reciprocally interacting worlds of experience.”

Intersubjectivity as used by Husserl was purely mental, but it has been taken up by developmental psychologists who use it to describe the shared world of mother and infant (Trevvarthen and Aitken 2001) that they argue is based on interacting biological systems. In a recent book, I have described these interactions as evidence of an “interbrain” network, mediated by nonverbal communication (Tantam 2009). Trevvarthen and Aitken trace intersubjectivity as a development concept to work on interactional synchrony in the early 1970s. Daniel Stern, a psychoanalyst, was one of these researchers (Stern 1971) and is often considered to be one of the originators of relational psychoanalysis (Stern 1985). Like Sullivan (1953) and Foulkes and Anthony (1957), he rejected the conventional analytic view that the infant is born in a state of autistic self-preoccupation from which he or she needed to be awakened although he still took the view that a failure of “attunement” by the mother to the infant could lead to deviant development; he considered that the inability of the mother to attune to a child with autism was due to the child’s inability to make affective contact.

Autistic Psychopathy

“Lust” is a disturbing but gripping narrative of a woman who feels devoured by the sexuality and aggression of an alien species – men – who hold her in thrall. It is written by Elfriede Jelinek, a Nobel Laureate who had been referred to Hans Asperger as a child (Delantaye 2005), is also reputed to have been a patient of Hans Asperger. It is, perhaps, the life experience of someone with Asperger syndrome. The focus of this entry has, so far, been on the Kanner and his description of early childhood autism. However, Hans Asperger had almost simultaneously with Kanner published (Asperger 1944) his habilitation on children that

he described as having “autistic personality disorder” and that he had first reported in 1938 (Asperger 1938).

Although Asperger worked in the cradle of psychoanalysis, Vienna, he does not seem to have been influenced by the approach. He was sure that the condition that he described was a disorder of the brain (Hippler and Klicpera 2005) that could be partly or wholly circumvented by education. He wrote (2005) of children with Asperger syndrome learning rules of etiquette in the same way that they learned sums and of conflict-ridden intellectual training leading to assimilation to the community.

What is notable about Asperger’s work is the focus on peer relationships, rather than parenting relationships; his notion that the symptoms of children with ASD are an attempt to overcome their difficulties, and not a kind of pathology and that children with an ASD are not merely helpless victims: In fact, he wrote that they can sometimes be malicious. Psychoanalysis with its emphasis on pathology rather than resilience, on parents rather than peers, and on unconscious motives rather than effort and training is poles apart from this kind of thinking.

These children can take note of “rules of etiquette” given to them in a down-to-earth kind of way, which they then can fulfill – like they would a sum. The more “objective” such a law is – maybe in a form of schedule, which includes all possible variations of daily routines and which must be stuck to by both parties in the most pedantic kind of way – the better it will be. So it is not through a habit, which unconsciously and instinctively grows by itself but through conscious, intellectual training, in years of difficult and conflict-ridden work, that one will achieve the best possible assimilation to the community, which will be more and more successful with growing intellectual maturity.

Conclusion

For all the fascination of psychoanalysis, it has perhaps created too much of a focus on parent-

child relationships in the study of people with ASD and detracted from peer relationships. It has also led us to think too often of the symptoms of ASD as symptoms and to obscure the possibility that the actions of people with ASD are the expression of a uniqueness and resilience in the face of a different brain and therefore a different participation in the connectedness of society.

It is regrettable that psychoanalysis also contributed to a victimization of parents, especially mothers, that increased the burden on already heavily burdened families. On the plus side, psychoanalysis has focused on the development of thought and language in children with ASD that might still lead to a better understanding of the subjective experience of having an ASD. That it has not yet done so may be because, as Bowlby pointed out many years ago, psychoanalysis has prioritized the child’s imagination of threat at the expense of real danger and loss. Most of us do assimilate our life experience to an emotionally charged narrative about who and what has made us the way we are, and this is the stuff of psychoanalysis, at least new paradigm psychoanalysis (Rudnytsky 2008). People with an ASD lack the narrative facility of many neurotypicals (Losh and Capps 2003) and do not usually dip into and out of autobiographical memory. On the other hand, people with ASD, even those who are gifted, often experience victimization, exclusion, and loneliness that are all too based in reality and that are not just in the past but continue in the present (Wainscot et al. 2008). These experiences, along with the sometimes condemnatory attitudes that people with ASD may develop about neurotypicals, are often a more productive focus for psychotherapy than the presumed parental failure.

References and Reading

- Asperger, H. (1938). Das psychisch abnormale kind. *Wiener Klinische Wochenschrift*, 51, 1314–1317.
- Asperger, H. (1944). Die “Autistischen Psychopathen” in Kindersalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Bettelheim, B. (1967). *The empty fortress: Infantile autism and the birth of the self*. New York: Free Press.

- Bleuler, E. (1896). Buchanzeige über Breuer-Freuds "Studien über Hysterie". *Münchener Medizinische Wochenschrift*, 22, 524–525.
- Bleuler, E. (1911). *Dementia Praecox oder Gruppen der Schizophrenien*. Leipzig/Wein: Franz Deuticke.
- Bleuler, E. (1913). Die Kritik der Freudschen Theorie. *Allgemeine Zeitschrift für Psychiatrie und psychisch gerichtliche Medizin*, 52, 665–718.
- Cohen, A. S., Emmerson, L. C., Mann, M. C., Forbes, C. B., & Blanchard, J. J. (2010). Schizotypal, schizoid and paranoid characteristics in the biological parents of social anhedonics. *Psychiatry Research*, 178(1), 79–83.
- de la Delantaye, L. (2005). On Cynicism, dogs, hair, Elfriede Jelinek and the Nobel Prize. *Harvard Review*, 29, 32–39.
- Ellenberger, H. (1970). *The discovery of the unconscious: The history and evolution of dynamic psychiatry*. New York: Basic Books.
- Flower, J. C. (1929). Emotion, feeling and religion. *Philosophy*, 4(14), 192–204.
- Fonagy, P., & Bateman, A. W. (2006). Mechanisms of change in mentalization-based treatment of BPD. *Journal of Clinical Psychology*, 62(4), 411–430. <https://doi.org/10.1002/jclp.20241>.
- Freud, S. (1914). *On the history of the psychoanalytic movement* (Vol. 14). London: Hogarth.
- Freud, S. (1965). *The interpretation of dreams*. New York: Avon Books.
- Freud, S., & Jung, C. (1974). *The Freud-Jung letters: The correspondence between Sigmund Freud and C. G. Jung (1906–1913)*. Princeton: Princeton University Press.
- Grinker, R. R., & Spiegel, J. (1945). *Men under stress*. Philadelphia: Blakiston.
- Hippler, K., & Klicpera, C. (2005). Hans Asperger and his patients – A retrospective examination of the spectrum of autistic disorders. *Zeitschrift für Kinder- und Jugendpsychiatrie und Psychotherapie*, 33(1), 35–47.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kanner, L. (1949). Problems of nosology and psychodynamics of early infantile autism. *American Journal of Orthopsychiatry*, 19, 416–426.
- Kolvin, I. (1971). Studies in the childhood psychoses: Diagnostic criteria and classification. *The British Journal of Psychiatry*, 118(545), 381–384.
- Kretschmer, E. *Physique and character*. London.
- Lacan, J. (1977). *The four fundamental concepts of psychoanalysis*. London: Hogarth Press.
- Laznik, M.-C. (1993). Pour une théorie lacanienne des pulsions. *Le Discours Psychanalytique Revue de l'Association Freudienne*, 10, 221.
- Lenzenweger, M. F. (2010). A source, a cascade, a schizoid: A heuristic proposal from the longitudinal study of personality disorders. *Development and Psychopathology*, 22(4), 867–881.
- Losh, M., & Capps, L. (2003). Narrative ability in high-functioning children with autism or Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 33(3), 239–251.
- Pfaff, D. W., Rapin, I., & Goldman, S. (2011). Male predominance in autism: Neuroendocrine influences on arousal and social anxiety. *Autism Research*, 4(3), 163–176. <https://doi.org/10.1002/aur.191>.
- Rutter, M. (1981). *Maternal deprivation reassessed* (2nd ed.). Harmondsworth: Penguin.
- Scalzone, F. (2005). Notes for a dialogue between psychoanalysis and neuroscience 1 [Article]. *International Journal of Psychoanalysis*, 86(Part 5), 1405–1423.
- Sifneos, P. (1972). *Short-term psychotherapy and emotional crises*. Cambridge, MA: Harvard University Press.
- Stern, D. (1971). A micro-analysis of mother-infant interaction. *Journal of the American Academy of Child and Adolescent Psychiatry*, 10(3), 501–517.
- Stolorow, B., Atwood, G., & Orange, D. (2002). *Worlds of experience: Interweaving philosophical and clinical dimensions in psychoanalysis*. New York: Basic Books.
- Sullivan, H. S. (1953). *The interpersonal theory of psychiatry*. New York: W. W. Norton.
- Tantam, D. (1990). Samuel Gaskell, distinguished psychiatrist, and his family. *Transactions of the Unitarian Historical Society*, 19 IS, 228–237.
- Tantam, D. (2009). *Can the world afford autistic spectrum disorder? Nonverbal communication, Asperger syndrome and the interbrain*. London: Jessica Kingsley.
- Tantam, D., Kalucy, R., & Brown, D. (1982). Sleep, scratching and dreams in eczema: A new approach to alexithymia. *Psychotherapy and Psychosomatics*, 37, 26–85.
- Tendlar, E. (1995). Object and image in autistic children. *Journal of the Centre for Freudian Analysis and Research*, 6, 63–71.
- Thomson, A. (1964). The philosophy of J. F. Ferrier. *Philosophy*, 39(147), 46–62.
- Trevarthen, C., & Aitken, K. J. (2001). Infant intersubjectivity: Research, theory, and clinical applications [Review]. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, 42(1), 3–48. [621 refs].
- van Deurzen, E. (2002). *Existential counselling in practice*. London: SAGE.
- Wainscot, J., Naylor, P., Sutcliffe, P., Tantam, D., & Williams, J. (2008). Relationships with peers and use of the school environment of mainstream secondary school pupils with Asperger syndrome (high-functioning autism): A case-control study. *International Journal of Psychology and Psychological Therapy*, 8, 1–25.
- Wolff, S. (2000). Schizoid personality in childhood and Asperger syndrome. In A. Klin, F. Volkmar, & S. Sparrow (Eds.), *Asperger syndrome* (pp. 278–308). New York: Guilford.

Psychoeducational Profile – Revised (PEP-3)

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Synonyms

PEP-3

Description

The Psychoeducational Profile: TEACCH Individualized Psychoeducational Assessment for Children with Autism Spectrum Disorders – Third Edition (PEP-3; Schopler et al. 2005) is the most current version of an assessment developed by professionals at TEACCH to evaluate the skills and behaviors of 2- to 7- year-old children with suspected autism spectrum disorders. Similar to its predecessors, the PEP-3 was designed to (a) measure developmental strengths, weaknesses, and learning style to inform educational programming; (b) assist in the autism diagnostic process by obtaining information from caregivers and by providing opportunities to observe social, communication, and play behaviors; and (c) provide developmental ages and other scores for diagnostic decision-making, educational planning, and/or assessing change in behavior and development over time. As such, the PEP-3 has utility in clinical, educational, and research contexts.

Developmental subtests on the PEP-3 assess early cognitive abilities, language/communication, and motor skills. Subtests included are cognitive verbal/preverbal, expressive language, and receptive language, which together comprise the communication composite, and fine motor, gross

motor, and visual-motor imitation, which together comprise the motor composite. Each developmental subtest item is scored as passing (the child can execute the task successfully without needing a demonstration), emerging (the child demonstrates some knowledge of how to complete the task but needs demonstration or practice), or failing (the child does not attempt or is unable to complete any aspect of the task). Raw scores are converted to developmental ages, percentiles, and developmental levels.

Maladaptive behavior subtests on the PEP-3 assess behavior areas related to an autism spectrum diagnosis. Subtests included are affective expression, social reciprocity, characteristic motor behaviors, and characteristic verbal behaviors, which together comprise the maladaptive behavior composite. Ratings for the maladaptive behavior subtests are based on observations throughout the assessment, and each item is scored as appropriate (the behavior is appropriate for the child's chronological and overall mental age), mild (the behavior is slightly or mildly unusual for the child's chronological and overall mental age), and severe (the behavior is unequivocally unusual or dysfunctional). Raw scores are converted to percentiles and adaptive levels. New PEP-3 software can be used to assist with scoring, statistical analyses, graphing, charting, and report generation.

The caregiver report component is a questionnaire completed by a caregiver, such as a parent, guardian, or teacher, based on his or her daily observations of the child. Two sections ask caregivers to provide estimates of the child's developmental level in several areas and rate the applicability and severity of several diagnostic labels. This information provides the examiner with a broader perspective of the child's overall strengths and weaknesses and helps the examiner understand the caregiver's perceptions of the child. The caregiver report also has subtests asking caregivers to rate behavioral problems associated with autism, skills in personal self-care, and adaptability and responses to the environment. Caregiver ratings are scored as appropriate, mild,

or severe, and raw scores are converted to percentiles and developmental/adaptive levels. A developmental age equivalent can also be computed for personal self-care skills.

The item content and administration procedures of the PEP-3 were designed to maximize its utility in children with autism spectrum disorders. Test materials are manipulative and interesting to young children, and with the exception of the language subtests, items require minimal language ability. Items are not timed, and tasks can be administered in an alternative order if doing so will make the assessment more engaging or appealing for the child. Further, given the scattered skills often shown by children with autism spectrum disorders, basals and ceilings are not used, though examiners may credit lower level skills when higher level skills are demonstrated. Finally, performance on test items readily translates into educational planning goals, and observation of the child during the assessment provides information about his or her learning style and the best way to begin teaching those goals.

Historical Background

The development of the PEP-3 (and its two earlier versions) (Schopler and Reichler 1979; Schopler et al. 1990, 2005) parallels the understanding, assessment, and treatment of autism from the 1940s through the present. The early formulations of autism were based on psychogenic principles attributing the atypical behaviors to profound emotional disturbances caused by faulty parenting. The apparent intellectual deficits shown by the majority of cases were considered pseudo-mental retardation, a mask covering up normal intelligence. The “islets of intelligence” noted by Kanner were offered as evidence of normal abilities. The children who were tested failed to score anywhere near the normal range and also were difficult to manage in the assessment setting. The concept of “untestable” was used to describe these children. It was in this context that Schopler and Reichler, in their pioneering research in autism in the 1960s and early 1970s, determined

that an appropriate assessment instrument, developmentally-based and geared to the unique characteristics of children with autism, would demonstrate that not only could these children be fairly tested but also meaningful individualized information could be gathered to help with diagnosis and program planning.

At that time, the main tests used were the Stanford-Binet and Wechsler Scales, designed for language-proficient children with relatively even patterns of skills and adequate competence, motivation, and relating. Only the brightest of children with autism could be expected to successfully take these tests, and, even then, their responses and scattered skill profiles might be misinterpreted. The introduction of the PEP, an autism-specific assessment tool, addressed the long-standing problems inherent in testing based on the misunderstanding of the children. By using an instrument based on developmental principles and administrative flexibility, examiners not only were able to obtain accurate information, but the understanding of the nature of autism and how it affected children was better appreciated.

Another insight Drs. Schopler and Reichler highlighted was the distinction between diagnosis and individualized assessment, particularly relevant to autism. Diagnosis involves the grouping of cases based on a set of common characteristics, while individualized assessment pertains to the specific profile of skills, deficits, interests, and behaviors. In their research, Schopler and Reichler developed both the Childhood Autism Rating Scale (originally the Childhood Psychosis Rating Scale) and the PEP as companion scales, with the former addressing diagnosis and the latter individualized assessment, with both emphasizing direct observation. The PEP was also unique in its integration of behavioral and developmental data in one instrument, emphasizing the importance of linking the atypical features with the developmental delays and irregularities. Autism was meant to be understood as a disorder of development, not an emotional or primarily behavioral disturbance, and the PEP was a means of documenting this.

The first published edition of the PEP was in 1979, although a working version had been available within the TEACCH program during that

decade. The first revision, the PEP-R, was published in 1990 in response to the increased numbers of younger children being diagnosed with autism and the passage of PL 99-457 expanding public school services for exceptional children under the age of five. The revised PEP added many items developmentally at the three and under age range. Other changes included expanding the language functioning area, streamlining sections dealing with the assessment of behavior problems, and modifying terminology to meet current definitions and usage. The second revision, the Psychoeducational Profile – Third Edition (PEP-3) was published in 2004. In addition to improving psychometric properties (e.g., larger normative sample, more reliability and validity data), the PEP-3 also updated behavioral items to be more consistent with current research on characteristics of autism and added a caregiver form to gather information on self-care skills, autism characteristics, and adaptive behavior.

Psychometric Data

The PEP-3 was developed using a normative sample comprised of 407 children and adolescents with ASDs and 148 children with typical development from around the United States. Development data were collected by professionals who had experience with the PEP-R and/or work as psychologists within the TEACCH program. Overall, analysis of reliability and validity indicates that the PEP-3 demonstrates sound psychometric properties (Schopler et al. 2005).

The PEP-3 demonstrates high reliability (Schopler et al. 2005). Internal consistency was assessed by calculating coefficient alphas for all subtests and composites in a sample of individuals with ASDs at 11 age intervals (ages 2 through 12). Average coefficients indicated high reliability and for the developmental subtests ranged from 0.92 to 0.97, for the maladaptive behavior subtests ranged from 0.90 to 0.93, and for the caregiver report subtests ranged from 0.84 to 0.90. Test-retest reliability was assessed over a period of 2 weeks in a sample of 33 children with ASDs. Raw scores were used to calculate correlation

coefficients for each subtest, and all indicate high reliability: developmental subtest correlation coefficients ranged from 0.97 to 0.99, maladaptive behavior subtest correlation coefficients ranged from 0.94 to 0.98, and caregiver report subtest correlation coefficients ranged from 0.98 to 0.99. Interrater reliability for the caregiver report subtests was assessed for 31 children with ASDs and 9 children with typical development. Two parents for each child completed the caregiver report form. Polychoric correlation coefficients were calculated for each item due to the ordered, categorical nature of the data. Coefficients ranged from 0.70 to 0.91 for the problem behaviors subtest, from 0.65 to 1.00 for the personal self-care subtest, and from 0.52 to 0.90 for the adaptive behavior subtest. One additional item on the adaptive behavior subtest was excluded from the calculation ranges due to its low reliability.

Criterion-prediction validity was assessed in a series of studies correlating scores from the PEP-3 with scores from other developmental and behavioral assessments used to measure similar constructs. Overall, the vast majority of correlations were large (0.50 and above) and in the expected direction, thus supporting the validity of the PEP-3 as a measure of development and autism characteristics (Schopler et al. 2005). In the first study, correlations between the PEP-3 subtests and subtests from the Vineland Adaptive Behavior Scales (VABS) were computed for a sample of 45 children and adolescents with ASDs. The VABS is a parent report measure of child adaptive behavior and self-care skills. As expected, positive and significant correlations were found between the VABS and the PEP-3 for subtests related to developmental skills, self-care skills, and behaviors related to an autism spectrum diagnosis. Lower correlations were found between PEP-3 caregiver report of problem behaviors and VABS scores of motor and daily living skills, though these scales were not predicted to correlate highly. In the second study, correlations between the PEP-3 subtests and total scores from the Childhood Autism Rating Scales, a measure of autism symptom severity, were computed for a sample of 68 children and adolescents with

ASDs. Because higher scores on the PEP-3 reflect a more typical pattern of behavior and higher scores on the CARS reflect a more impaired or autistic pattern of behavior, significant negative correlations were predicted and found. Similarly, the third study examining the relation between the PEP-3 and the total score on the Autism Behavior Checklist – Second Edition in 316 individuals with ASDs yielded significant negative correlations between the measures except for the PEP-3 characteristic verbal behavior subtest.

The PEP-R and the PEP-3 have been translated into several languages including Chinese, Japanese, French, Portuguese, Dutch, Italian, and Estonian. Although some versions may benefit from further study, evidence available in English indicates that translated versions continue to show strong reliability and validity, and the PEP is a valuable instrument internationally for assessing children with autism (De Leon 2005; Fu et al. 2010, 2011; Kikas and Häidkind 2003; Steerneman et al. 1997; Villa et al. 2010).

Clinical Uses

The PEP-3 has multiple clinical and educational purposes including gathering developmental data for program planning and informing diagnostic decision-making, although it should not be used as a stand-alone measure for making a diagnosis. However, the PEP-3 collects information on the behavioral characteristics associated with ASD. It has normative data based on a large sample of children with autism, particularly useful in clarifying the extent to which the child being tested shows features shared by the sample of children with ASD. The PEP-3 can be used as the primary test in the evaluation process, but it should be supported by other information such as parent report, observations in a natural setting (e.g., home or school), previous history, and collateral reports. When diagnostic clarification is a primary goal of the evaluation, it is preferable if the PEP-3 is used in conjunction with the Childhood Autism Rating Scale, the Autism Diagnostic Observation Schedule, or another scale specifically designed for diagnostic classification.

Perhaps the greatest utility of the PEP-3 is in generating developmental information across multiple function areas. A strength of the test is that it was designed specifically for children with autism; it can be flexibly administered, contains materials of interest to children, and does not depend on language for instructions. The nonverbal domains and items do not require language processing for the child to complete them. The test covers a wide range of important developmental functions. Examples of test flexibility include the options of alternating easy and challenging activities, conducting the assessment using work systems or routines familiar to the student, and teaching and scaffolding to get complete information.

Developmental age scores can be derived and can be converted into a developmental quotient (Delmolino 2006). However, more important to program planning is the breakdown of skills into patterns as well as item analysis. In reviewing the data, the clinician is able to consider multiple levels of analysis including examining the child's overall pattern of strengths and weaknesses, analyzing the individual items that were passed or where an "emerging" score was obtained, reviewing the pattern of maladaptive behaviors, and considering the caregiver information. A careful analysis provides insight about appropriate developmental expectations useful in planning goals and curriculum.

Each of the developmental functions or domains is made up of many items that tap a variety of skills. For example, the Cognitive Verbal/Preverbal Scale measures the skills of problem-solving (nonverbal), matching, sorting and categorizing, and visual-motor integration. In addition, the examiner can observe characteristics of social communication (e.g., directing attention or commenting, seeking help or praise, sharing enjoyment), cognitive style (e.g., organization, flexibility, ability to transition between materials, persistence, cognitive areas of strength and weakness), and use of and response to materials (e.g., appropriate or inappropriate, unusual sensory responses, rigid or flexible). Results from performance on the different developmental domains can be used to develop instructional goals, curriculum, and behavioral strategies for

implementation at school and home. Integrating the information from the developmental and behavioral scales leads to a well-rounded approach to intervention and program planning. Other resources are available that show examples of how skills can be taught in a visual, structured way (Boswell et al. 2005; Eckenrode et al. 2003, 2009).

See Also

- ▶ Age Equivalents
- ▶ Autism Diagnostic Observation Schedule
- ▶ Expressive Language
- ▶ Psychological Assessment
- ▶ Receptive Vocabulary
- ▶ Schopler, Eric
- ▶ Vineland Adaptive Behavior Scales (VABS)
- ▶ Visual-Motor Function

References and Reading

- Boswell, S., Reynolds, B., Faulkner, R., & Benson, M. (2005). *Let's get started*. North Carolina: Lulu.
- De Leon, C. (2005). Psychoeducational profile revised (PEP-R): The Brazilian version elaboration. *Autism*, 9(4), 450–451.
- Delmolino, L. M. (2006). Brief report: Use of DQ for estimating cognitive ability in young children with autism. *Journal of Autism and Developmental Disorders*, 36(7), 959–963.
- Eckenrode, L., Fennell, P., & Hearsey, K. (2003). *Tasks Galore, Tasks Galore*. Raleigh.
- Eckenrode, L., Fennell, P., Hearsey, K., & Reynolds, B. (2009). *Tasks Galore: Let's play: Structured steps to social engagement and symbolic play*. Raleigh: Tasks Galore.
- Fu, C. P., Hsieh, C. L., Tseng, M. H., Chen, Y. L., Huang, W. T., Wu, P. C., et al. (2010). Inter-rater reliability and smallest real difference of the Chinese psychoeducational profile-third edition for children with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 4(1), 89–94.
- Fu, C. P., Chen, K. L., Tseng, M. H., Chiang, F. M., & Hsieh, C. L. (2011). Reliability and validity of the psychoeducational profile-third edition caregiver report in children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6, 115–122.
- Hogan, K., & Marcus, L. M. (2009). From assessment to intervention. In S. Goldstein, J. A. Naglieri, & S. Ozonoff (Eds.), *Assessment of autism spectrum disorders* (pp. 318–338). New York: Guilford.
- Kikas, E., & Häidkind, P. (2003). Letter to the editor: Developing an Estonian version of the psychoeducational profile revised (PEP-R). *Journal of Autism and Developmental Disorders*, 33(2), 217.
- Naglieri, J. A., & Chambers, K. A. (2009). Psychometric issues and current scales for assessing autism spectrum disorders. In S. Goldstein, J. A. Naglieri, & S. Ozonoff (Eds.), *Assessment of autism spectrum disorders* (pp. 55–90). New York: Guilford.
- Portoghese, C., Buttiglione, M., Pavone, F., Lozito, V., De Giacomo, A., Martinelli, D., et al. (2009). The usefulness of the revised psychoeducational profile for the assessment of preschool children with pervasive developmental disorders. *Autism*, 13(2), 179–191.
- Schopler, E., & Reichler, R. (1979). *Individualized assessment and treatment of autistic and developmentally disabled children: vol 1. Psychoeducational Profile (PEP)*. Austin: Pro-Ed.
- Schopler, E., Reichler, R., Bashford, A., Lansing, M., & Marcus, L. (1990). *Psychoeducational profile-revised (PEP-R)*. Austin: Pro-Ed.
- Schopler, E., Lansing, M., Reichler, R., & Marcus, L. (2005). *Psychoeducational profile: Third edition (PEP-3)*. Austin: Pro-Ed.
- Steerneman, P., Muris, P., Merckelbach, H., & Willems, H. (1997). Brief report: Assessment of development and abnormal behavior in children with pervasive developmental disorders: Evidence for the reliability and validity of the revised psychoeducational profile. *Journal of Autism and Developmental Disorders*, 27(2), 177–185.
- Villa, S., Micheli, E., Villa, L., Pastore, V., Crippa, A., & Molteni, M. (2010). Further empirical data on the psychoeducational profile-revised (PEP-R): Reliability and validation with the Vineland Adaptive Behavior Scales. *Journal of Autism and Developmental Disorders*, 40(3), 334–341.

Psychological Assessment

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Synonyms

Intelligence tests (see specific tests); IQ test

Definition

Assessment is the process by which a professional meets with and evaluates an individual, family, or family subsystem for a determined need. There

are many different types of assessments. The presenting concerns of the individual, family members, or family system should determine the type, focus, and amount of testing. Most often, assessment is conducted with individuals. Depending on the age of the individual, in addition to a diagnostic evaluation, assessment of the domains of neurocognitive, academic, language, adaptive, and social-emotional functioning and behavior problems is routinely recommended for individuals who have been identified as having, or as at risk for an, autism spectrum disorder (ASD).

Assessment can be both formal and informal. However, all methods of assessment should be completed with the aim of gaining information about an individual that will aid in making informed decisions. There are many purposes of assessment: screening, problem solving, diagnosis, counseling and rehabilitation, and evaluating treatment progress. In some circumstances, it may be possible to complete one assessment that can inform several of these purposes. However, in other circumstances, multiple specialized assessments may be required to obtain the information necessary to develop an accurate picture of the individual and the most complementary treatment plan (Sattler 2001).

Screening assessments are brief evaluations designed to signal initial risk for a particular disorder, condition, or problem. Examples of autism screeners include the Screening Tool for Autism in Toddlers and Young Children (STAT; Stone and Ousley 2008) and the Social Communication Questionnaire, Revised (SCQ-R; Rutter et al. 2003a). These brief observation and questionnaire tools are generally quite sensitive, thus ensuring that children who are at risk get the additional assessment needed to determine or rule out an ASD diagnosis. Another highly sensitive screener of emotional and social functioning is the Infant Toddler Social Emotional Assessment (ITSEA) (Briggs-Gowan and Carter 2006). This screener may be a good first tool in assessing general social and emotional competence before deciding on using an autism specific screener.

A problem-solving assessment is a detailed evaluation of a specific concern, or area of functioning. These assessments are often used to

determine whether or not an individual has a specific diagnosis, such as an ASD. The Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2002) and the Autism Observation Scale for Infants (Bryson et al. 2007) are two examples of problem-solving assessments often used as part of an evaluation designed to diagnose ASDs.

A comprehensive psychological assessment requires a detailed evaluation of an individual's functioning in more than one area. This type of assessment often results in a profile of an individual's strengths and weaknesses in areas such as academics, cognition, language, and social functioning. These types of assessments allow for accurate diagnoses and treatment plans to be made that best aid the individual in achieving at their highest ability. One important addition to the comprehensive evaluation is intelligence testing. This is especially important for individuals with an ASD given that intellectual ability has been linked to severity of autism symptoms, ability to acquire skills, level of adaptive functioning, and overall trajectory (Ozonoff et al. 2005). Commonly used cognitive tests for individuals with an ASD include the Wechsler Intelligent Scale for Children, Fourth Edition (WISC-IV; Wechsler 2003), the Differential Ability Scales-II (DAS-II; Elliott 2007), the Mullen Scales of Early Learning (MSEL; Mullen 1995), and the Leiter-Revised (Leiter-R; Roid and Miller 1997). The Leiter-R is an especially useful tool for individuals with limited language. Additionally, language assessments are also imperative to assessments of individuals with an ASD. Expressive language level is the second best predictor of overall trajectory (Ozonoff et al. 2005). Common, relatively brief receptive and expressive vocabulary tests used with individuals with an ASD include the Peabody Picture Vocabulary Test (PPVT; Dunn and Dunn 1997) and the Expressive One Word Vocabulary Test (EOWPVT; Martin and Brownell 2000), respectively.

Counseling, rehabilitation, and adaptive assessments focus on the individual's abilities, rather than their weaknesses. These assessments evaluate the individual's abilities to adjust and successfully complete daily responsibilities. A common measure that may be used with

individuals with an ASD to assess adaptive behavior is the Vineland Adaptive Behavior Scales II (Vineland II; Sparrow et al. 2005). This Vineland-II is a semi-structured parent interview that assesses an individual's daily living skills. Additionally, the TEACCH Transition Assessment Profile (TTAP; Mesibov et al. 2007) could also be utilized as a direct assessment of an individual's ability to complete daily tasks such as answering the phone, counting money, and sorting. The TTAP also includes parent and teacher interviews as well.

Progress evaluation assessments occur periodically after initial assessments to track an individual's changes in development and to evaluate the effectiveness of treatment.

Historical Background

Modern psychological testing originated a little less than one hundred years ago with the development of some basic sensory and motor measures by British scholar, Francis Galton. Following Galton's initial development of testing, American psychologist, James McKeen Cattell continued this work throughout his academic career, revolutionizing testing, beginning with his paper "Mental Tests and Measurements," published in 1890.

Moving forward to 1943, Kanner offered the first description of autism when he identified a subset of individuals with intellectual disability. These individuals suffered deficits of social and communication skills that predominantly occurred in boys (Kanner 1943). Assessment of autism has long been left to the clinical interview with the aid of the Diagnostic and Statistical Manual of Mental Disorders (DSM). Autism was first mentioned in DSM-I under Schizophrenic reaction, childhood type (Author 1952). It was not until DSM-III, launched in 1980, that Autism was considered a unique diagnosis (Author 1980). While several diagnostic questionnaires and clinical interviews have been in use for many years, it was not until the ADOS was developed that clinicians were able to assess autism through the process of provoking behavioral

symptoms through presses during a semi-structured interaction (Lord, Rutter, and DiLavore 2001). The ability to assess autism symptoms from a behavioral observational perspective has provided assessors with a first-hand method of assessing and evaluating an individual's behaviors as opposed to relying on second-hand accounts in isolation.

Current Knowledge

While there have certainly been historical missteps in the application of psychological assessment throughout the evolution of the practice, including the damaging role of prejudicial psychological assessment in the Immigration Restriction Act of 1924, assessment remains a useful and critical tool of understanding one's unique individual profile (Gregory 2004). Today, there are clearly defined standards and general protocol to follow. Additionally, psychologists today should practice with the understanding that while general profiles can be created for different diagnoses, cultural influences are important and should be considered within each unique case.

An important distinction exists between psychological testing and psychological assessment. The emphasis on psychological testing is rooted in data collection, whereas psychological assessment is not purely focused on data collection through tests, but is concerned with a more global evaluation of an individual. Current theory dictates that there are four important "pillars" of psychological assessment.

The four pillars of assessment include norm-referenced tests, interviews, observations, and informal assessment procedures (or, more generally, tests). An assessment that incorporates each of the four pillars is considered to be a good representation of an individual's unique profile (Sattler 2001).

Norm-referenced tests are considered to be critical in conducting psychological assessments (Carter, Godoy, and Marakovitz 2009). These tests are standardized on a clearly defined group and are scaled so that each score indicates an individual's rank within the normed group.

Norm-referenced tests are most commonly used to assess intellectual ability, academic achievement, adaptive behavior, and fine/gross motor skills (Sattler 2001). Norm-referenced tests allow for quantification of an individual's behavior. Quantification can be helpful in determining where an individual ranks in comparison to his/her chronological peers, determining the location of specific deficits/strengths, and providing a baseline to track progress. It is important to select standardized tests that have a sufficient number of items at the level at which an individual is functioning so that there is sufficient variation in functioning to observe relative strengths and weaknesses, or sufficient sensitivity to develop a cognitive profile that is not subject to floor effects.

Norm-referenced rating scales are often used given that they are inexpensive and can be administered flexibly (i.e., online, through the mail, in person). Additionally, norm-referenced rating scales can be used to enrich a comprehensive assessment of an individual by asking teachers, employers, daycare providers, and others in an individual's daily life to complete questionnaires as well (Carter et al. 2009).

Interviews with multiple informants are another key component to a sound assessment. This is especially true for individuals with ASDs, as symptom severity may depend largely on the environment (Ozonoff et al. 2005). For example, high-functioning individuals with an ASD may appear to be astute and connected with their schoolwork and peers in a class that they both are interested in and excel in. However, that same individual may have severe difficulties maintaining a part-time job as a cashier due to the many social demands of the position and a lack of interest. Interviewing a child's parents, teachers, and other caregivers provides critical insight into the child's functioning in several different environments. Interviews can be structured, semi-structured, or unstructured. While all interviews provide the opportunity for information to be delivered in a conversational matter, structured interviews are the most rigid with a set list of questions and often result in a comprehensive caricature of the individual and are usually designed to aid in diagnosis. The Autism

Diagnostic Interview-Revised (ADI-R; LeCouteur et al. 2003) and the Childhood Autism Rating Scale, Second Edition (CARS2; Schopler et al. 2010) are popular semi-structured parent interviews used to contribute to a diagnosis of ASD. Both the ADI-R and the CARS 2 review, with parents, critical information regarding the affected individual's communication, social, and behavioral development. Assessment of these three domains is an important piece of a comprehensive assessment of an individual with an ASD (Ozonoff et al. 2005). Semi-structured and unstructured interviews provide less structure but are also important to the assessment of an individual because they give the opportunity for information to be transmitted that may have been lost in a more structured and formal setting. While structured and semi-structured interviews provide an in-depth amount of information they are more commonly utilized in research settings (Carter et al. 2009).

Observations are another critical component of psychological assessment. Observations can be made both formally, through observational assessment tools such as the ADOS (Lord, Rutter, and DiLavore 2001), and/or informally throughout the evaluation and/or in other environments (e.g., school, work, community). Observations are important because they allow a clinician to identify factors that may be contributing to, or reinforcing, specific problem behaviors. Through making these direct observations, clinicians are then better able to create effective, personalized, intervention plans.

Assessments of individuals with an ASD should also strive to be multidisciplinary whenever possible. Professionals from psychology, psychiatry, pediatrics, and neurology and speech and language specialists are often imperative members of the treatment team. While interdisciplinary coordination is essential to promoting quality care it is important that one professional take the lead as the evaluation coordinator. The evaluation coordinator should communicate with the parent and the affected child/adult in order to ensure that all information is equally shared and that the family only needs to speak with one professional who will then disseminate the

information. Utilizing an evaluation coordinator is essential to a successful evaluation and ensures accurate transmission of information (Ozonoff et al. 2005).

In regard to informal assessment procedures, or tests, there are many different options for clinicians to choose from. It is important to choose tests that assess for the information that is pertinent to the individual's needs. It is important to use tests that have sound reliability and validity. If a test does not have either of these qualities, it should be used cautiously (Sattler 2001). Additionally, measures of sensitivity and specificity are especially important psychometric issues to evaluate when considering which test to administer. High percentages of sensitivity and specificity ensure that a test is sensitive enough to capture enough of a population for screening purposes, but restrictive enough to not overwhelm service systems (Carter et al. 2009).

Although the four pillars of assessment do not operate independently, they are unique. Each pillar contributes to the overall assessment and creates a vivid picture of the individual's strengths, weaknesses, and overall level of functioning. A quality assessment should be derived from a number of sources, assessment methods, and cover multiple domains of functioning (e.g., intelligence, memory, oral language, adaptive behavior, etc.). Following completion of the assessment, results should be calculated, substantial evidence in the form of observations should contribute to sound clinical impressions, and recommendations should then be made to facilitate treatment (Sattler 2001).

When completing an evaluation it is especially important to interpret the scores by considering what they suggest about the individual's strengths and weaknesses. Clinicians should be careful to not interpret scores at face value as they diminish the qualitative data gathered in the assessment period and may overgeneralize an individual's profile. For example, an individual may have a low adaptive behavior score but may be capable of completing high-level tasks such as preparing a simple meal. If only a low score was interpreted from an adaptive behavior scales interview, a recommendation might be made to focus on self-help skills, such as preparing simple meals. However,

given that the individual has already mastered this skill, the recommendation is not useful in facilitating adaptive growth.

When assessing individuals for ASDs, two levels of screening and evaluation exist. Level 1 screening involves observation by routine service providers, such as pediatricians. Level 2 evaluations involve comprehensive diagnostic assessments provided by an experienced clinician (Ozonoff et al. 2005). Comprehensive diagnostic assessments should take into consideration the developmental component of ASD. Generally, children are diagnosed with ASDs in early childhood and continue to fit criteria through the life span.

When considering psychological assessment of young children at risk for a diagnosis of an ASD it is important to understand the challenges of assessing infants and toddlers. Often psychological assessments of young children focus on social-emotional problem behaviors, without equal focus placed on social-emotional competencies. While it is understandable that externalizing behaviors often drive parental concern and therefore prompt assessment, it is arguable that focusing on a child's competencies allows clinicians to gain more insight into the overall profile of the child. Namely, recognizing a delay in social and emotional competencies indicates risk for the emergence of additional behavior problems. Further, competencies in areas of social-emotional functioning generally indicate continued mastery. Knowledge of both social-emotional problems and competencies contribute to better treatment plans that capitalize on a child's strengths (Carter et al. 2009).

It is also important to take into consideration the contexts of race, ethnicity, and culture when completing an assessment. Often, race, ethnicity, and culture are used interchangeably as different ways of stating the same thing, when they are distinctly different constructs. Race, is a socially constructed concept that has been created based on physical characteristics. Culture is distinct in that it describes the shared values, beliefs, and practices of a group that are transmitted across generations. Lastly, ethnicity indicates a specific culture and is usually associated with a common geographic or national origin (Carter et al. 2004). Assessments that incorporate sensitive understanding of these

three domains contribute to a comprehensive understanding of the affected individual that takes into account factors other than test scores and addresses potential clinician biases.

Future Directions

While psychological assessment has certainly made advancements since the 1890s, both in developing the sophistication of the testing instruments and in considering cultural issues, assessment is not a perfect science. Certainly, the four pillars of assessment and integration of empirical knowledge of specific disorders and profiles contribute to a useful conceptualization of an individual's functioning. However, as assessment tools continue to develop, it is important to consider the imperfections that currently exist and work toward rectifying them in an effort to further advance the field. For example, it may be helpful to develop diagnostic specific norms for different measures of assessment. Developing norms that are specific to different diagnostic groups allows clinicians to make quantitative impairments within groups, thus allowing for more specific recommendations and expectation.

See Also

- Adaptive Behavior Scales
- Autism Diagnostic Interview-Revised
- Autism Diagnostic Observation Schedule
- Peabody Developmental Motor Scales (PDMS)
- Vineland Adaptive Behavior Scales (VABS)
- Wechsler Preschool and Primary Scale of Intelligence

References and Reading

- American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (1st ed., text rev.). Washington, DC: Author.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed., text rev.). Washington, DC: Author.
- Briggs-Gowan, M. J., & Carter, A. S. (2006). *Brief infant toddler social and emotional assessment*. San Antonio: Pearson.
- Brownell, R. (2000). *Expressive one-word picture vocabulary test-2000*. Novato: Academic Therapy.
- Bryson, S. E., Zwaigenbaum, L., McDermott, C., Rombough, V., & Brian, J. (2007). The autism observation scale for infants: Scale development and reliability data. *Journal of Autism and Developmental Disabilities*, 38(4), 731–738. <https://doi.org/10.1007/s10803-007-0440-y>.
- Carter, A. S., Briggs-Gowan, M. J., & Ornstein Davis, N. (2004). Assessment of young children's social-emotional development and psychopathology: Recent advances and recommendations for practice. *Journal of Child Psychology and Psychiatry*, 45(1), 109–134.
- Carter, A. S., Godoy, L. L., Marakovitz, S. E., & Briggs-Gowan, M. J. (2009). Parent reports and infant-toddler mental health assessment. In C. H. Zeanah (Ed.), *Handbook of infant mental health* (pp. 233–252). New York: Guilford.
- Dunn, L. M., & Dunn, L. M. (1997). *Peabody picture vocabulary test* (3rd ed.). Circle Pines: American Guidance Service.
- Elliott, C. D. (2007). *Differential ability scales – II*. San Antonio: Pearson.
- Gregory, R. J. (2004). *Psychological testing: History, principles and applications* (4th ed.). Boston: Pearson Education.
- Kanner, L. (1943). Autistic disturbances of affective content. *The Nervous Child*, 2, 217–250.
- LeCouteur, A., Lord, C., & Rutter, M. (2003). *Autism diagnostic interview – Revised*. Torrance: Western Psychological Services.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2002). *Autism diagnostic observation schedule*. Torrance: Western Psychological Services.
- Martin, N. A., & Brownell, R. (2000). *Expressive one word picture vocabulary test* (4th ed.). Torrance: Western Psychological Services.
- Mesibov, G., Thomas, J. B., Chapman, S. M., & Schopler, E. (2007). *TEACCH transition assessment profile* (2nd ed.). Austin: PRO-ED.
- Mullen, E. M. (1995). *Mullen scales of early learning*. Circle Pines: American Guidance Service.
- Ozonoff, S., Goodlin-Jones, B. L., & Solomon, M. (2005). Evidence-based assessment of Autism spectrum disorders in children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, 34(3), 523–540. https://doi.org/10.1207/s15374424jccp3403_8.
- Roid, G., & Miller, L. (1997). *Leiter international test of intelligence-revised*. Chicago: Stoelting.
- Rutter, M., Bailey, A., Berument, S. K., Lord, C., & Pickles, A. (2003a). *Social communication questionnaire*. Torrance: Western Psychological Services.
- Rutter, M., LeCouteur, A., & Lord, C. (2003b). *Autism diagnostic interview – Revised (ADI-R)*. Torrance: Western Psychological Services.
- Sattler, J. M. (2001). *Assessment of children: Cognitive applications* (4th ed.). San Diego: Jerome M. Sattler.

- Schopler, E., Van Bourgondien, M. E., Wellman, G. J., & Love, S. R. (2010). *Childhood Autism rating scale* (2nd ed.). San Antonio: Pearson.
- Sparrow, S. S., Chicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales* (2nd ed.). San Antonio: Pearson.
- Stone, W. L., & Ousley, O. Y. (2008). *Screening tool for autism in toddlers and young children*. Nashville: VueInnovations.
- Wechsler, D. (2003). *Wechsler intelligent scale for children* (4th ed.). San Antonio: Pearson Education.

Psychologist

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Synonyms

Analyst; Clinician; Psychotherapist; Social scientist; Therapist

Definition

Academic or clinical professional who holds a doctoral degree in psychology from an organized, sequential, and regionally accredited school or university; an expert or specialist in psychology who carries out research in and/or practices psychology; a person who provides psychological services that include diagnostic evaluations, functional assessments, interventions, preventative and ameliorative care, consulting, program development and/or administration, education, and supervision.

References and Reading

- American Psychological Association. What is APA's definition of a "psychologist"? Retrieved from <http://www.apa.org/support/about/apa/psychologist.aspx#answer>
- Board of Professional Affairs, Committee on Professional Standards. (1987). General guidelines for providers of psychological services. *The American Psychologist*, 42(7), 712–723.

Psychomotor

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Synonyms

Psychomotor development

Definition

As typically used in psychology, psychomotor skills are those abilities that are related to motor activity in association with cognitive/mental activity. A number of psychomotor skills must be learned by the young infant including coordination, movement of the body, coordination of movement with thought/intention, and so forth. Complex psychomotor skills include activities like dancing, pitching, or playing a musical instrument.

Early investigators like Arnold Gesell (1949) charted the progress of early psychomotor skills and noted that, in general, there was a cephalocaudal ("head to tail") and proximodistal ("inner to outer") pattern of motor control, e.g., head control before trunk control and shoulder control before hand control. Other investigators like Piaget (1952) were more interested in the way that the development of thinking interrelated to motor control with coordination of early motor and perceptual abilities and simple reflexes into much more cognitively and motorically complex action. Some of the early work on charting the normative acquisition of skills also highlighted patterns of delay and developmental deviation. For example, delays in imitation skills in autism appear to reflect a fundamental lack of social orientation and interest rather than deficits in motor skills as such and may be important implications for treatment (Ingersoll et al. 2007; McDuffie et al. 2007).

See Also

- [Imitation](#)
- [Occupational Therapy \(OT\)](#)
- [Physical Therapy](#)

References and Reading

- Gesell, A. (1949). Human infancy and the ontogenesis of behavior. *American Scientist*, 37(4), 529–553.
- Ingersoll, B., Lewis, E., & Kroman, E. (2007). Teaching the imitation and spontaneous use of descriptive gestures in young children with autism using a naturalistic behavioral intervention. *Journal of Autism and Developmental Disorders*, 37(8), 1446–1456.
- McDuffie, A., Turner, L., Stone, W., Yoder, P., Wolery, M., & Ulman, T. (2007). Developmental correlates of different types of motor imitation in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(3), 401–412.
- Piaget, J. (1952). *The origins of intelligence in children*. New York: Norton.

involve some aspects of conceptual or psychological functioning. The term Psychomotor Developmental Index (PDI) is most closely associated with the Bayley Scales of Infant Development (BSID), first published by Nancy Bayley in 1969 and revised in 1993 as the BSID-II. The BSID-II includes the following three scales, mental, motor, and behavior, with the motor scale referred to as the Psychomotor Developmental Index (PDI). Test items for the PDI include motor skills such as rolling, crawling, grasp, and use of utensils.

Assessment of developmental functions emerged in the twentieth century with the advent of assessments such as the Gesell System of Developmental Diagnosis (1925), the Cattell Infant Intelligence Test (1940), and the original Bayley Scales of Infant Development (1969). These assessments focused on documenting normative expectations and were aimed at identifying a child's developmental progressions. Over time, these assessments also led to efforts to identify developmental delays for the purpose of planning appropriate early interventions.

Psychomotor Development

- [Psychomotor](#)

Psychomotor Development Index

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Synonyms

[Motor development assessment](#)

Definition

Psychomotor Developmental Index generally refers to a measure of motor skills that also

References and Reading

- Bayley, N. (1969). *Manual for the Bayley scales of infant development*. San Antonio: The Psychological Corporation.
- Bayley, N. (1993). *Manual for the Bayley scales of infant development*. San Antonio: The Psychological Corporation.
- Cattell, P. (1940). *The measurement of intelligence of infants and young children*. New York: The Psychological Corporation.
- Gessell, A. (1925). *The mental growth of the pre-school child*. New York: MacMillan.
- Glenn, S. M., Cunningham, C. C., & Dayus, B. (2001). Comparison of the 1969 and 1993 standardizations of the Bayley mental scales of infant development for infants with Down's syndrome. *Journal of Intellectual Disability Research*, 45(1), 55–62.
- Provost, B., Heimeryl, S., McClain, C., Kim, N., Lopez, B., & Kodituwakku, P. (2000). Concurrent validity of the Bayley scales of infant development II motor scale and the Peabody developmental motor scales in two-year-old children. *Physical & Occupational Therapy in Pediatrics*, 20(1), 5–18.

Provost, B., Lopez, B., & Heimerl, S. (2006). A comparison of motor delays in young children: Autism spectrum disorder, developmental delay, and developmental concerns. *Journal of Autism and Developmental Disorders*. Retrieved on 30 Mar 2011 from <http://cdd.unm.edu/Ec/pubs/pdfs/comparisonofmotordelays.pdf>.

Psychopathology

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Definition

The study of developmental aspects mental illness is a term used in the field of mental health in a way equivalent to the use of the term pathology (illness) in other parts of medicine. The area of psychopathology is concerned with mental disorders and maladaptive/abnormal aspects of behavior or development. This area is one of concern to many different disciplines including psychiatry, psychology, and social work. Research work in the area ranges from the level of the brain and gene to the expression of maladaptive patterns of behavior/development in the individual and to cultural and epidemiological studies.

The most widely used guides to descriptive psychopathology are the DSM (*Diagnostic and Statistical Manual*) of the American Psychiatric Association and the ICD (International Classification of Diseases) manual; the former exists in one form for both researchers and clinicians and the latter in two different guides: one for researchers and another more general one for clinician. It is important to note that of itself difference does not imply psychopathology and that the cultural, family, and other factors may greatly impact the way mental disorders are expressed. Developmental factors are also particularly important for

disorders, like autism, which arise in the first years of life and may impact many aspects of subsequent development and functioning. Having one disorder may also increase the risk for others (what is termed “comorbidity”). In addition to the study of specific patterns of distress/impairment at the level of disorders, the term psychopathology can also be used to refer to specific symptoms or behaviors suggestive of mental illness, e.g., delusions or hallucinations.

See Also

- [Comorbidity](#)
- [DSM-IV](#)
- [ICD 10 Research Diagnostic Guidelines](#)

References and Reading

- Bernier, R., Mao, A., et al. (2010). Psychopathology, families, and culture: Autism. *Child and Adolescent Psychiatric Clinics of North America*, 19(4), 855–867.
- Horovitz, M., Matson, J. L., Sipes, M., Shoemaker, M., Belva, B., & Bamberg, J. W. (2011). Incidence and trends in psychopathology symptoms over time in adults with severe to profound intellectual disability. *Research in Developmental Disabilities*, 32(2), 685–692.
- Rutter, M., & Sroufe, L. (2000). Developmental psychopathology: Concepts and challenges. *Development and Psychopathology*, 12(3), 265–296.
- Rutter, M., Kim-Cohen, J., et al. (2006). Continuities and discontinuities in psychopathology between childhood and adult life. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 47(3–4), 276–295.
- Smith, K. R. M., & Matson, J. L. (2010). Psychopathology: Differences among adults with intellectually disabled, comorbid autism spectrum disorders and epilepsy. *Research in Developmental Disabilities*, 31(3), 743–749.
- Volkmar, F. R., & Martin, A. (2011). *Essentials of child and adolescent psychiatry*. Philadelphia: Lippincott, Williams, and Wilkins.

Psychophysiology

- [Neurophysiology](#)

Psychosis

► Psychotic Disorder

Psychosocial

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Definition

This term refers to the combination of psychological and social factors as relevant to the development of the individual. The term was used by Erikson in his elaboration of life span model of development characterized by tensions typical of each age level. In some ways, research on psychosocial factors in autism was inhibited by the early focus on (and blaming of) parents and parental psychopathology in the etiology of autism (see Riddle 1987 for a review). As it became apparent that autism was a strongly brain-based and highly genetic disorder, work on psychosocial factors diminished substantially. It has increased in recent years with an awareness of the many and varied factors that can enhance (or inhibit) the optimal development of children with autism. This work has now included family factors including aspects of parenting (Rodrigue et al. 1992) and of sibling development (Rodrigue et al. 1992) as well as the importance of interventions in this area (Lord et al. 2005). The perspective of various relevant adults on the impact of psychosocial factors (Foley Nicpon et al. 2010) as well as the interaction of biological factors and psychosocial risk (Rutter and Casaer 1991) has also been explored.

See Also

► Adaptive Behavior

References and Reading

- Foley Nicpon, M., Doobay, A. F., & Assouline, S. G. (2010). Parent, teacher, and self perceptions of psychosocial functioning in intellectually gifted children and adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 40(8), 1028–1038.
- Lord, C., Wagner, A., Rogers, S., Szatmari, P., Aman, M., Charman, T., et al. (2005). Challenges in evaluating psychosocial interventions for autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 35(6), 695–708; discussion 709–611.
- Riddle, M. A. (1987). Individual and parental psychotherapy in autism. In D. J. Cohen & A. Donnellan (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 528–544). New York: Wiley.
- Rodrigue, J. R., Morgan, S. B., & Geffken, G. R. (1992). Psychosocial adaptation of fathers of children with autism, Down syndrome, and normal development. *Journal of Autism and Developmental Disorders*, 22(2), 249–263.
- Rutter, M., & Casaer, P. (Eds.). (1991). *Biological risk factors for psychosocial disorders*. New York: Cambridge University Press.

Psychosocial Effects of Soft Skills Training for Young Adults with Autism

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Definition

Work-related social skills interventions designed and developed to help transition-age individuals (14–24 years old), including young adults with autism, improve their soft skills through a manualized curriculum delivered in school- or community-based settings. Soft skills, which are intra- and interpersonal qualities that are transferable across contexts (e.g., social-communication skills, positive attitude, responsibility, problem-solving, flexibility, time and stress management, networking, teamwork, work ethic, professionalism, etc.) are important for workplace success.

Historical Background

Autism spectrum disorder (ASD) is a lifelong neurodevelopmental disorder characterized by persistent impairments in social communication and interaction, repetitive patterns of behaviors, restrictive interests or activities, and sensory processing abnormalities (APA 2013). According to Centers for Disease Control and Prevention, there has been a dramatic rise in the prevalence of ASD over the past decade, with approximately 1 in 54 children diagnosed with ASD in the United States (Maenner et al. 2020). In addition to the rapid increase in prevalence, approximately 60,000 youth with ASD turn 18-years-old each year in the United States (Anderson et al. 2018; Roux et al. 2013), and close to half a million youth with ASD will enter adulthood and seek employment over the next decade (Roux et al. 2015).

Social, Vocational, and Psychological Challenges Faced by Young Adults with ASD

While individuals on the autism spectrum exhibit a broad range of functioning, symptom severity, and intelligence, impaired social communication and interaction are hallmarks of ASD, leading to difficulties understanding nonverbal and abstract verbal interactions with resultant problems in forming relationships and social networks, as well as obtaining and maintaining employment. While many young adults with ASD affirm interest in and possess the talent and capacity for long-term, competitive, and integrated employment, their characteristic challenges with social communication and adaptive functioning skills often significantly impact their ability to gain employment or live independently (Anderson et al. 2014; Seaman and Cannella-Malone 2016). Specifically, socially awkward behaviors, poor eye contact, sensory processing abnormalities, difficulty with perspective taking, literalism, repetitive behaviors, restricted interests, and difficulty reading social cues all have the potential to disrupt social interaction, particularly in workplace settings where awareness of ASD may be limited or misunderstood.

In addition to the social skills deficits inherent to the diagnosis, individuals with ASD experience

disproportionate levels of anxiety and depression. Vickerstaff et al. (2007) found that older age and higher IQ predicted lower self-perceived social competence and higher levels of anxiety and depressive symptoms among children with ASD. In fact, many individuals with ASD, especially those with normal intelligence (IQ), are cognizant of their social difficulties. Additionally, as children with ASD transition into adulthood, social demands become more complex and their social deficiencies become more noticeable as a result of the loss of structured social supports they are often provided during the school years (Hillier et al. 2011; White and Roberson-Nay 2009). Consequently, young adults with ASD may choose to socially withdraw rather than risk repeated social failure.

The social skills deficits (Howlin and Moss 2012) and mental health challenges (White and Roberson-Nay 2009) of young adults with ASD complicate their participation in transition activities. Due, in part, to these persistent struggles, their postsecondary education and employment outcomes have been very poor, creating a tremendous extended burden on their psychosocial well-being. Results from the National Longitudinal Transition Study ([NLTS-2]; Newman et al. 2011) indicate that young adults with ASD experience lower employment rates (37.2%) than any other disability category and markedly disparate employment rates when compared to their peers without disabilities (54%). Compounding the problem, those with ASD who manage to obtain employment are underemployed, typically receiving low pay and limited working hours (Taylor and Seltzer 2011; Roux et al. 2013). Furthermore, the majority of those with ASD are not participating in employment training or postsecondary education in the critical first 2 years after high school (Anderson et al. 2014; Shattuck et al. 2012). For example, a secondary analysis of the NLTS-2 data indicated that, among 680 youth with ASD, 28% had attended a 2-year college; 1% a 4-year college; 9.3% a vocational or technical education program; and 33% had not participated in any postsecondary training (Shattuck et al. 2012).

Additionally, there are high prevalence rates of comorbid mental health conditions among young

adults with ASD, with an estimation of nearly 50% of the population having comorbid anxiety and/or depression (White and Roberson-Nay 2009). Croen et al. (2015) found that adults with ASD have a 117% higher incidence of anxiety, a 123% higher incidence of depression, and an astounding 433% higher incidence of suicide attempts than their peers. Another study found that 66% of a sample of adults with ASD self-reported suicidal ideation, and 35% had plans or attempts at suicide (Cassidy et al. 2014). Based on these data, the mental health challenges that young adults with ASD experience are not only prevalent but alarmingly severe. Hillier et al. (2011) further suggest that anxiety and depression may be closely related to some of the central difficulties of ASD, including poor social communication, difficulty with unexpected changes in routine, and narrow and repetitive interests. As a result of repeatedly uncomfortable and frequently failed interactions, the individual may withdraw from social engagement, thereby losing social networks and supports, and in turn experience more anxiety and depression. The situation can become even more complicated when symptoms of anxiety and depression among the ASD population do not present as they do in the general population, as they can be masked by ASD characteristics such as flat affect and social withdrawal (Hillier et al. 2011).

Current Knowledge

Relationship of Social Skills, Employment, and Well-being

The relationship between social functioning, employment, and psychological well-being can be seen as cyclical. Late adolescence and young adulthood are particular times of vulnerability to mental health challenges, as social demands increase and individuals with ASD are prone to short tenure of employment partly due to poor social and adaptive functioning. Specifically, poor social functioning may present barriers to obtaining and sustaining employment, leading to increased social isolation and decreased opportunities to practice social competence in work,

resulting in increased anxiety and depression (Connor et al. 2019). Pragmatically, poor employment outcomes also present a barrier to independent living and long-term psychological effects and well-being (Howlin et al. 2005).

Indeed, up to 90% of job losses in individuals with disabilities are due to social communication difficulties (Elksnin and Elksnin 2001). Barriers to successful employment in the ASD population include socially inappropriate behaviors, poor hygiene or adaptive behaviors, deficits in theory of mind, executive functioning impairments, difficulties with sensory processing, and inappropriate interactions with individuals of the opposite sex (Howlin et al. 2005). These barriers lead to a disproportionately high unemployment rates among individuals with ASD (Howlin and Moss 2012; Strickland et al. 2013). Even when employed, individuals with ASD are often underemployed, work less hours, receive lower wages, and have fewer responsibilities and opportunities for promotion given their educational backgrounds and hard skills (Taylor and Seltzer 2011). The damage to self-efficacy and mental health implications that result from unemployment and underemployment can be devastating.

Self-efficacy is an action oriented, future-focused construct that gives individuals the confidence to pursue interests, move forward with goals, and manage tasks (Bandura 1997). Self-efficacy is tied to both work and social performance and is inversely correlated with high levels of social anxiety and depression among individuals (Lorenz et al. 2016). Career success leads to increased self-efficacy and ultimately life satisfaction, whereas repeated job failures negatively impact career self-efficacy and consequently life satisfaction. Further, the high rates of co-occurring mental health conditions (e.g., anxiety and depression) among those with ASD are linked to decreased social and employment participation. Nonetheless, the psychological effects of long-term under- and unemployment have not been extensively studied in the ASD population.

Although the literature has yet to identify the causal relationships between social functioning, employment, and psychological well-being in

the ASD population, it is likely that the effects are bidirectional (White and Roberson-Nay 2009), where interventions targeting any one aspect may impact others. Additionally, these factors make adolescence not only a time of vulnerability but also a critical time for intervention to improve psychosocial and vocational outcomes. Thus, the need for interventions and supports to improve postschool outcomes for this population is extremely crucial during the transition years. In particular, soft skills training is especially vital, as social communication and interpersonal skills have been identified as a significant predictor to employment success for persons with ASD (Chen et al. 2015).

Needs for Evidence-Based Employment-Related Interventions

Successful employment not only increases an individual's social status and financial independence but also positively impacts physical and psychological health and improves overall quality of life. However, current trends indicate that young adults with ASD are not often prepared to meet the varying social demands of the work environment due to limited soft skills training and development (Wehman et al. 2015). This has continued to impact chronic under- and unemployment for this population (Seaman and Cannella-Malone 2016). While programs and services have been created to support the growing number of children with ASD, there has not yet been a concerted effort to ensure adequate support for youth with ASD as they transition into adulthood.

Recent legislation, the Workforce Innovation and Opportunity Act (WIOA 2014), calls for "research related to meeting the education and employment needs of eligible youth" and specifically mandates "the development of alternative, evidence-based programs and other activities that enhance the choices available to eligible youth" (WIOA 2014; Sect. 129). While a dramatically large increase in demand for vocational rehabilitation services for young adults with ASD have been well-described (Chen et al. 2015; Shattuck et al. 2012), less scholarly emphasis has been placed on intervention research as it relates to

employment-related training needs. Until recent years, there has been an expansion in the number of employment-related intervention studies, focusing on improving employment-readiness among transition-age individuals with ASD. Specifically, a total of 42 distinct intervention studies have been identified in recent systematic literature reviews (e.g., Anderson et al. 2017; Hedley et al. 2016; Hotton and Coles 2016; Seaman and Cannella-Malone 2016). Despite this expansion of research in employment-related intervention studies for the ASD population, interventions appear to be disproportionately trending towards specific job skills, or hard skills, with interventions in soft skills being less emphasized.

Based on these systematic reviews, employment-readiness training can be further broken into two skill development categories, hard skills and soft skills, which are both necessary components of obtaining and maintaining employment. *Hard skills* are technical skills and abilities that allow an individual to perform a particular task or activity within a specific context (e.g., developing a resume, using technology to apply for jobs, creating a spreadsheet, organizing merchandise, using computer software, performing clerical tasks, etc.); *soft skills* are intra- and interpersonal qualities that are transferable across contexts (e.g., social-communication skills, positive attitude, responsibility, problem-solving, flexibility, time and stress management, networking, teamwork, work ethic, professionalism, etc.).

Work-related social skills, or soft skills, are facilitators to employment acquisition and central to long-term workplace success (Seaman and Cannella-Malone 2016). While social and psychological difficulties are central to ASD (Hillier et al. 2011), recent research studies reveal that soft skills training and development is a recognized need by both individuals with on the autism spectrum and stakeholders (Seaman and Cannella-Malone 2016) to mitigate the employment situations and potentially improve the psychological well-being among the population. Yet, such intervention studies are scant and only two vocational social skills programs that specifically address both vocational and psychological functioning

for young adults with ASD have been identified (Hillier et al. 2011; Connor et al. 2019) to date. Further, there is limited research describing the relationship between soft skills training, psychological well-being, and employment outcomes for the population (Connor et al. 2019; Sung et al. 2018). Therefore, interventions designed to address workplace social functioning may be particularly relevant to addressing the psychosocial and employment needs of young adults with ASD (Chen et al. 2014).

Existing Interventions that Targets Employment-Related Social Skills

In a systematic review of the ASD literature, Spain and Blainey (2015) found preliminary and promising evidence that group-based social skills interventions for adults with ASD are “effective for enhancing social knowledge and understanding, improving social functioning, reducing loneliness and potentially alleviating comorbid psychiatric symptoms” (p. 874). Below is a summary of some existing intervention programs targeting social skills and/or work-related social skills for young adults with ASD.

Program for the Education and Enrichment of Relational Skills (PEERS)

Laugeson et al. (2015) studied the effectiveness of a social skills curriculum, PEERS for Young Adults, which is built on the manualized PEERS for Adolescents Program, with additional content focusing on dating etiquette, managing peer pressure, and using social goals to improve motivation and compliance. The program is designed for young adults with ASD between 18- and 23-years-old who attend with their social coach (e.g., parent or caregiver) Small groups of 6–8 participants attend 16 weekly group sessions for 90-min per session. Participants are taught social skills through didactics, role-play, and skill practicing during group socialization activities, while social coaches attend separate sessions simultaneously to learn how to provide coaching to young adults in handling social situations. Topics in the curriculum include: developing and maintaining friendships, conversational skills,

entering and exiting conversations, appropriate use of humor, electronic communication, dating skills, organizing get-togethers, handling bullying, handling disagreements, and handling dating pressure.

Gantman et al. (2012) demonstrated that social skills training using the PEERS for Young Adults curriculum resulted in significant increases in social skills knowledge and behaviors, empathy, and social contacts, as well as decreased ASD symptoms and loneliness for participants with ASD. More recently, in a replication study, McVey et al. (2016) found that, in addition to the improvements revealed by Gantman et al., participants also showed significant reductions in social anxiety. However, programs like PEERS focus broadly on general social functioning, including friendships, social gatherings, and effective conversation, which precludes an exclusive focus on developing work-related social skills.

Aspirations Program

Hillier et al. (2007, 2011) studied the effectiveness of the Aspirations program, which is a social and vocational support group consisting of eight weekly sessions for 1 h per session with small groups of between 5 and 7 participants. The curriculum focuses on improving social and vocational skills and covered topics include: introductions, social communication, relationships, independent living, independence and college, and employment. The program uses a discussion-based format designed to be directed by the group members, with the group facilitators simply guiding the discussion and ensuring participants remained on topic. Each session began with a group facilitator introducing the topic for that session and areas to be covered and ended with a review of topics covered and a reflection of learned content.

Hillier et al. (2007, 2011) found that the Aspirations program had positive impacts on empathy, attitudes towards peers, improvements in autistic features, and reductions in feelings of anxiety and depression in participants with ASD. The group members learned and gained greater understanding by sharing personal experiences and listening to those of others, giving each other advice, and

creating problem solving strategies as a group. However, the program was more of a support group rather than a structured learning approach targeting employment or soft skill development through skills training.

Job-Based Social Skills (JOBSS) Program

Gorenstein et al. (2020) evaluated the effectiveness of the JOBSS intervention, which is a manualized, 15-week, group-delivered intervention for small groups of 5–6 young adults with ASD that merges job training and social skills intervention and aims to increase the social-pragmatic skills necessary to obtain and maintain employment. The JOBSS curriculum is fully manualized and includes session-by-session scripts and accompanying worksheets and visual aids. Topics in the curriculum include: active listening, nonverbal communication, theory of mind, making a good impression, identifying emotions, managing stress, problem solving and teamwork, talking about our weaknesses, conversation skills, communication and workplace hierarchy, before and during the interview, ending the interview and afterwards, disclosure, and the hidden curriculum. The curriculum was created to be accessible and easy for those without specialized training to implement using a cognitive-behavioral therapy treatment modality (e.g., conducting structured sessions, connecting cognition to behavior, focusing on the present).

Gorenstein et al. (2020) showed that after completing the JOBSS curriculum, the participants with ASD, as reported by caregiver, demonstrated significantly improved social cognitive skills, and approximately half of participants reported a job obtainment 6 months later. All participants were satisfied with the intervention and were highly likely to recommend the program. The authors suggest that the JOBSS curriculum has potential to improve social skills of adults with ASD, thereby increasing successful employment.

Assistive Soft Skills and Employment Training (ASSET) Program

Sung et al. (2018) examined the preliminary efficacy of ASSET, a soft skills intervention, on

social functioning, self-efficacy, and psychological wellness for young adults with ASD. ASSET is a manualized, group-based intervention that is delivered in school or community-based settings with small groups of 6–8 participants. The originally piloted program was 8 weeks, one session per week, at 90-min each. However, the current program consists of 12 weeks, given added topics per stakeholder's recommendations. The curriculum covers six core work-related social skills, or soft skills: communication, attitude and enthusiasm, teamwork, networking, problem solving and critical thinking, and professionalism, as well as additional topics on mental health and stress management, awareness of self and others, time management, and organization and planning. Ten primary teaching strategies are integrated across sessions, including: didactics, modeling, group discussion, video examples, experiential activities, role-play, self-reflection, performance feedback, positive reinforcement, and generalization. Moreover, ASD-specific pedagogies are used throughout the curriculum, including structured-learning approaches, social performance training, and video-based instruction. The intervention is consistently held in conference rooms (as opposed to classrooms or clinics), and group socialization opportunities are created for participants to practice skills through interacting with peers under the guidance of group facilitators to promote generalization of learning.

Sung et al. (2018) revealed that young adults with ASD not only showed significant improvements in work-related social skills knowledge and social cognition, but their social and empathy self-efficacy improved through experiences of successful, scaffolded learning during the ASSET program. The participants and group facilitators also reported high satisfaction with program activities, training modalities, frequency, and duration of the intervention. Subsequently, Connor et al. (2019) extended the study and indicated that participants' social skill acquisition and augmented self-confidence result in improved psychological wellness, with significant reductions in anxiety and a trend toward lessening depressive symptoms among young adults with ASD.

Virtual Reality Training Programs

Kandalafi et al. (2013) investigated the feasibility and effectiveness of Virtual Reality Social Cognitive Training (VR-SCT). Young adults with ASD who completed the training showed improved theory of mind and emotion recognition. In another study applying virtual reality approaches to teaching interview skills, young adults with ASD who completed the Virtual Reality Job Interview Training program (VR-JIT) showed improved interview skills and self-confidence (Smith et al. 2014). While these studies highlight the effectiveness of using technology to teach skills, as training is individually completed on a computer, neither intervention is integrated in the community, where interviews and employment most often occur.

Future Directions

Research underscores the prevalence and magnitude of issues related to employment-related social competence among young adults with ASD, as well as the potential to utilize group interventions to partially address psychological wellness in addition to the more traditional, individual, clinical, and/or pharmaceutical approaches. The group-based training format offers individuals with ASD not only the opportunity for vicarious skills acquisition and practices with peers, but establishes the sense of belongingness that comes from being part of a group with shared experiences (Connor et al. 2019). The opportunity to share experiences and strategies and to develop relationships with peers who encounter similar challenges may have psychosocial impacts on the ASD population (Hillier et al. 2011). Thus, practitioners working with this population should keep in mind the secondary psychological benefits associated with soft skills interventions, particularly those that are group-based. In addition to more targeted mental health approaches, soft skills training programs may be considered an integral part of promoting, maintaining, and improving psychosocial functioning for young adults with ASD.

While interventions targeting employment-related social skills have been increasingly

prevalent for addressing the pressing soft skills and mental health needs of the larger ASD population, only very few programs are found to be theory-driven and/or possess empirical rigor such as using random assignment and experimental control. Additionally, to date, research has not systematically examined soft skills interventions and its psychosocial benefits as evidence-based practices for young adults with ASD. To better understand the vocational and psychosocial impacts of novel soft skills interventions, rigorous research on the efficacy and effectiveness is essential to ensuring that programs are suited to the population in natural contexts for improving the employment, independent living, and mental health disparities experienced by this population. In addition, continued research should focus on manualized soft skills training curricula. The provision of an empirically validated manual is particularly important because it provides reassurance to practitioners that the intervention can be delivered with in a standardized approach with ease and consistency. Furthermore, manualization not only aids practitioners in the delivery of the intervention, but also aids researchers by providing additional assurance that the intervention is delivered with fidelity. These steps all contribute to ensuring safe, efficacious, and effective programs.

Although randomized controlled trials have been conducted in some of the employment-related social skills interventions, they are largely tested within a single site. Future studies using multisite randomized controlled trials guided by implementation science (Locke et al. 2016) are critical to determine the most efficient and effective ways of implementing soft skills training and psychosocial supports in various environments, such as school or community-based settings. This way, young adults with ASD can benefit from the opportunities for practicing skills within natural contexts to generalize social skill performance. Implementation science emphasizes the need to use evidence-based practices to embed an established practice within an existing entity (e.g., school or community-based settings). It examines the factors, processes, and strategies that influence the uptake, use, and sustainability

of empirically supported interventions and/or practice innovations in routine practice settings. Integrating implementation science into intervention research can advance our efforts to bring research and practice closer together. Further, implementation science can be combined with the recent recommendations for ASD interventions to collectively inform the development process of a usable and acceptable intervention that is designed for implementation and scaling with fidelity by a variety of educational and vocational professionals in a wide range of academic, community, and clinical settings.

Despite the growing number of intervention research focusing on employment-related social skills, there is sparse research on validated measures for assessing specific soft skills, and even fewer instruments have been designed for and/or validated with individuals with ASD (Hedley et al. 2016; Strickland et al. 2013). Perry and Felce (2015) conducted a thorough review and identified 21 published instruments that measure employment-related social skills. Only one instrument is dedicated for vocational social skills assessment, whereas the rest are either too broad with limited focus on specific employment-related social skills or only applicable to children. Thus, research is warranted to develop measures that capture changes in work-related social function among the transition age population. Moreover, many of the current social skills measures are subjective self- or informant-report. Use of video data collection should be further explored as a potentially more direct approach to subjective observational rating.

In sum, the transition from secondary education to higher education, work, and independent living is a critical period in the development of all youth, and one that elicits unique challenges for young adults with ASD. Social isolation and co-occurring mental health problems make this population particularly at risk for service disengagement after high school. The research literature is replete with descriptions of disparate postsecondary outcomes for young adults with ASD, yet evidence-based soft skills interventions are lacking. As a result, it is imperative to develop a larger and stronger evidence base of transition

practice by validating manualized, group-based soft skills interventions that can be easily implemented by practitioners, embedded into multidisciplinary community contexts, and made accessible to young adults with ASD who may otherwise not receive services to address their social and emotional needs. Recognizing the potential bidirectional relationships between work-related social functioning and psychological wellness, more research is needed to inform how soft skills training will address psychosocial adaptation and functional and employment outcomes for young adults with ASD.

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Publishing.
- Anderson, D. K., Liang, J. W., & Lord, C. (2014). Predicting young adult outcome among more and less cognitively able individuals with autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 55 (5):485–494. <https://doi.org/10.1111/jcpp.12178>
- Anderson, A., Moore, D. W., Rausa, V. C., Finkelstein, S., Pearl, S., & Stevenson, M. (2017). A systematic review of interventions for adults with autism spectrum disorder to promote employment. *Review Journal of Autism and Developmental Disorders*, 4, 26–38. <https://doi.org/10.1007/s40489-016-0094-9>.
- Anderson, K. A., Sosnowy, C., Kuo, A. A., & Shattuck, P. T. (2018). Transition of individuals with autism to adulthood: A review of qualitative studies. *Pediatrics*, 141(Suppl 4):S318–S327. <https://doi.org/10.1542/peds.2016-43001>
- Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York: Freeman.
- Cassidy, S., Bradley, P., Robinson, J., Allison, C., McHugh, M., & Baron-Cohen, S. (2014). Suicidal ideation and suicide plans or attempts in adults with Asperger's syndrome attending a specialist diagnostic clinic: A clinical cohort study. *The Lancet Psychiatry*, 1(2), 142–147. [https://doi.org/10.1016/s2215-0366\(14\)70248-2](https://doi.org/10.1016/s2215-0366(14)70248-2).
- Chen, J. L., Sung, C., & Pi, S. (2015). Vocational rehabilitation service patterns and outcomes for individuals with autism of different ages. *Journal of Autism and Developmental Disorders*, 45, 3015–3029. <https://doi.org/10.1007/s10803-015-2465-y>.
- Connor, A., Sung, C., Strain, A., Zeng, T., & Fabrizi, S. (2019). Building skills, confidence, and wellness: Psychosocial effects of soft skills training for young adults with autism. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03962-w>. Advance online publication.

- Croen, L. A., Zerbo, O., Qian, Y., Massolo, M. L., Rich, S., Sidney, S., & Kripke, C. (2015). The health status of adults on the autism spectrum. *Autism, 19*(7), 814–823. <https://doi.org/10.1177/1362361315577517>.
- Elksnin, N., & Elksnin, L. K. (2001). Adolescents with disabilities: The need for occupational social skills training. *Exceptionality, 9*(1–2), 91–105. https://doi.org/10.1207/S15327035EX091&2_7.
- Gantman, A., Kapp, S. K., Orenski, K., & Laugeson, E. A. (2012). Social skills training for young adults with high-functioning autism spectrum disorders: A randomized controlled pilot study. *Journal of Autism and Developmental Disorders, 42*(6), 1094–1103. <https://doi.org/10.1007/s10803-011-1350-6>.
- Gorenstein, M., Giserman-Kiss, I., Feldman, E., Isenstein, E. L., Donnelly, L., Wang, A. T., & Foss-Feig, J. H. (2020). Brief report: A job-based social skills program (JOBSS) for adults with autism spectrum disorder: A pilot randomized controlled trial. *Journal of Autism and Developmental Disorders, 50*(1), 1–13. <https://doi.org/10.1007/s10803-020-04482-8>.
- Hedley, D., Uljarevic, M., Cameron, L., Halder, S., Richdale, A., & Dissanayake, C. (2016). Employment programmes and interventions targeting adults with autism spectrum disorder: A systematic review of the literature. *Autism, 21*, 1–13. <https://doi.org/10.1177/1362361316661855>.
- Hillier, A., Fish, T., Cloppert, P., & Beversdorf, D. Q. (2007). Outcomes of a social and vocational skills support group for adolescents and young adults on the autism spectrum. *Focus on Autism and Other Developmental Disabilities, 22*, 107–115. <https://doi.org/10.1177/10883576070220020201>.
- Hillier, A., Fish, T., Siegel, J. H., & Beversdorf, D. Q. (2011). Social and vocational skills training reduces self-reported anxiety and depression among young adults on the autism spectrum. *Journal of Developmental and Physical Disabilities, 23*, 267–276. <https://doi.org/10.1007/s10882-011-9226-4>.
- Hotton, M., & Coles, S. (2016). The effectiveness of social skills training groups for individuals with autism spectrum disorder. *Review Journal of Autism and Developmental Disorders, 3*(1):68–81. <https://doi.org/10.1007/s40489-015-0066-5>.
- Howlin, P., & Moss, P. (2012). Adults with autism spectrum disorders. *The Canadian Journal of Psychiatry, 57*(5), 275–283. <https://doi.org/10.1177/070674371205700502>.
- Howlin, P., Alcock, J., & Burkin, C. (2005). An 8-year follow-up of a specialist supported employment service for high-ability adults with autism or Asperger's syndrome. *Autism, 9*(5), 533–549. <https://doi.org/10.1177/1362361305057871>.
- Interagency Autism Coordinating Committee (IACC). (2011). *IACC strategic plan for autism spectrum disorder research*. Washington, DC: US Department of Health and Human Services.
- Kandalafi, M. R., Didehban, N., Krawczyk, D. C., Allen, T. T., & Chapman, S. B. (2013). Virtual reality social cognition training for young adults with high-functioning autism. *Journal of Autism and Developmental Disorders, 43*(1), 34–44. <https://doi.org/10.1007/s10803-012-1544-6>.
- Laugeson, E. A., Gantman, A., Kapp, S. K., Orenski, K., & Ellingsen, R. (2015). A randomized controlled trial to improve social skills in young adults with autism spectrum disorder: The UCLA PEERS® program. *Journal of Autism and Developmental Disorders, 45*(12), 3978–3989. <https://doi.org/10.1007/s10803-015-2504-8>.
- Lindsay, S., Adams, T., McDougall, C., & Sanford, R. (2012). Skill development in an employment-training program for adolescents with disabilities. *Disability and Rehabilitation, 34*(3), 228–237. <https://doi.org/10.3109/09638288.2011.603015>.
- Locke, J., Beidas, R. S., Marcus, S., Stahmer, A., Aarons, G. A., Lyon, A. R., . . . Mandell, D. S. (2016). A mixed methods study of individual and organizational factors that affect implementation of interventions for children with autism in public schools. *Implementation Science, 11*(1), 135. <https://doi.org/10.1186/s13012-016-0501-8>.
- Lorenz, T., Frischling, C., Cuadros, R., & Heintz, K. (2016). Autism and overcoming job barriers: Comparing job-related barriers and possible solutions in and outside of autism-specific employment. *PLoS One, 11*(1), e0147040. <https://doi.org/10.1371/journal.pone.0147040>.
- Maenner, M. J., Shaw, K. A., Baio, J., Washington, A., Patrick, M., DiRienzo, M., . . . Dietz, P. (2020). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2016. *Morbidity and Mortality Weekly Report Surveillance Summaries, 69*(SS-4), 1–12. <https://doi.org/10.15585/mmwr.ss6904a1>.
- McVey, A. J., Dolan, B. K., Willar, K. S., Pleiss, S., Karst, J. S., Casnar, C. L., Caiozzo, C., Vogt, E. M., Gordon, N. S., & Van Hecke, A. V. (2016). A replication and extension of the PEERS® for young adults social skills intervention: Examining effects on social skills and social anxiety in young adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 46*(12), 3739–3754. <https://doi.org/10.1007/s10803-016-2911-5>.
- Newman, L., Wagner, M., Knokey, A. M., Marder, C., Nagle, K., Shaver, D., et al. (2011). *The post-high school outcomes of young adults with disabilities up to 8 years after high school: A report from the National Longitudinal Transition Study-2* (NCSE no. 2011-3005). Menlo Park: SRI International.
- Perry, J., & Felce, D. (2015). Assessing work-related social skills: Existing approaches and instruments. Retrieved from: <https://www.researchgate.net/publication/255652550>
- Roux, A. M., Shattuck, P. T., Cooper, B. P., Anderson, K. A., Wagner, M., & Narendorf, S. C. (2013). Post-secondary employment experiences among young adults with an autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 52*(9):931–939. <https://doi.org/10.1016/j.jaac.2013.05.019>

- Roux, A. M., Shattuck, P. T., Rast, J. E., Rava, J. A., & Anderson, K. A. (2015). *National autism indicators report: Transition into young adulthood*. Philadelphia: Life Course Outcomes Research Program, A.J. Drexel Autism Institute, Drexel University.
- Seaman, R. L., & Cannella-Malone, H. I. (2016). Vocational skills interventions for adults with autism spectrum disorder: A review of the literature. *Journal of Developmental and Physical Disabilities*, 28(3), 479–494. <https://doi.org/10.1007/s10882-016-9479-z>.
- Shattuck, P., Narendorf, S., Cooper, B., Sterzing, P., Wagner, M., & Taylor, J. T. (2012). Postsecondary education and employment among youth with an autism spectrum disorder. *Pediatrics*, 129, 1042–1049. <https://doi.org/10.1542/peds.2011-2864>.
- Smith, T., Scahill, L., Dawson, G., Guthrie, D., Lord, C., Odom, S., . . . Wagner, A. (2007). Designing research studies on psychosocial interventions in autism. *Journal of Autism and Developmental Disorders*, 37(2), 354–366. <https://doi.org/10.1007/s10803-006-0173-3>.
- Smith, M. J., Ginger, E. J., Wright, K., Wright, M. A., Taylor, J. L., Boteler Humm, L., Olsen, D., Bell, M. B., & Fleming, M. F. (2014). Virtual reality job interview training in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44, 2450–2463. <https://doi.org/10.1007/s10803-014-2113-y>.
- Spain, D., & Blainey, S. H. (2015). Group social skills interventions for adults with high-functioning autism spectrum disorders: A systematic review. *Autism*, 19(7):874–886. <https://doi.org/10.1177/1362361315587659>
- Strickland, D. C., Coles, C. D., & Southern, L. (2013). JobTIPS: A transition to employment program for individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(10), 2472–2483. <https://doi.org/10.1007/s10803-013-1800-4>.
- Sung, C., Connor, A., Chen, J., Lin, C.-C., Kuo, H.-J., & Chun, J. (2018). Development, feasibility, and preliminary efficacy of an employment-related social skills intervention for young adults with high-functioning autism. *Autism*. <https://doi.org/10.1177/1362361318801345>. Advance online publication.
- Taylor, J. L., & Seltzer, M. M. (2011). Employment and postsecondary educational activities for young adults with autism spectrum disorders during the transition to adulthood. *Journal of Autism and Developmental Disorders*, 41, 566–574. <https://doi.org/10.1007/s10803-010-1070-3>.
- Vickerstaff, S., Heriot, S., Wong, M., Lopes, A., & Dossetor, D. (2007). Intellectual ability, self-perceived social competence, and depressive symptomatology in children with high-functioning autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 1647–1664. <https://doi.org/10.1007/s10803-006-0292-x>.
- Wehman, P., Sima, A. P., Ketchum, J., West, M. D., Chan, F., & Luecking, R. (2015). Predictors of successful transition from school to employment for youth with disabilities. *Journal of Occupational Rehabilitation*, 25, 323–334.
- White, S. W., & Roberson-Nay, R. (2009). Anxiety, social deficits, and loneliness in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 1006–1013. <https://doi.org/10.1007/s10803-009-0713-8>.
- Workforce Innovation and Opportunity Act, P.L. 113–128, 29 U.S.C. § 3101 (2014).

Psychostimulants

► Stimulant Medications

Psychotherapist

► Psychologist

Psychotherapy

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Definition

Barker provides a concise and pragmatic conceptualization of psychotherapy, defining the process as a psychological treatment of life problems. Of interest are four added caveats: the treatment must be provided by a trained

practitioner; treatment must occur as part of a professional relationship between provider and patient; treatment must attempt to remove, attenuate or change the life problems; and the process must involve the genesis or nurturance of adaptive behavior and/or personal growth (Barker 1999).

While there are multiple theoretical orientations within the realm of psychotherapy, treatments based upon behavioral and cognitive-behavioral theories have emerged as having the strongest evidence base. These treatments are provided under an array of titles (e.g., self-instructional training, problem-solving therapy, social skills training, and exposure therapy). However, a common thread among all of these psychotherapy programs is their grounding in empirically derived models which explain problem behaviors by way of focusing upon cognitive, emotional, and/or behavioral responses.

In and of itself, a diagnosis of ASD indicates the presence of persistent deficits in communication and social interaction, as well as restricted and/or repetitive patterns of behavior, interests, or activities (American Psychiatric Association [DSM-V] 2013). As such, psychotherapy protocols have been modified to accommodate the need for foundational skill building in these areas. However, a growing literature indicates that patients with ASD are also at high risk for comorbid mental health conditions, including anxiety, depression, bipolar disorder, aggressive behavior, and eating disorders (e.g., Anderson and Morris 2006; Brereton et al. 2006; Gilliot et al. 2001). Notably, such conditions are often more problematic to the child and his/her family than are the core ASD symptoms per se. Because of this, psychotherapy protocols have been developed targeting the specific elements of comorbid conditions.

Historical Background

Studies examining the effectiveness of various types of psychotherapy in typically developing youth with various presenting disorders are numerous (e.g., Abbass et al. 2006; Churchill et al. 2001; Haby et al. 2006; Leichsenring et al.

2004; Smith et al. 1980). In the case of ASD, however, there are multiple issues limiting extrapolation from these studies to youth with ASD and the respective co-occurring condition. Specifically, changes over time in how the etiology of ASD has been conceptualized – from willful withdrawal by the child and poor parenting practices to the contemporary position of a neurobiological etiology – affect the perceived need for psychotherapy, as well as the ultimate goals of such treatment.

Perhaps the first example of such change, and its impact upon psychotherapy as treatment, lies with Kanner's appropriation of the term "autism." Bleuler (1911) used the term to describe a pathognomonic feature of childhood schizophrenia; specifically, a willful withdrawal by the child into their fantasy world. In contrast, Kanner (1943) emphasized the inability of children with autism to use and/or recognize situational, environmental, or social cues.

Kanner went further by linking autism to a lack of familial warmth, although this did not account for asymptomatic siblings of children with autism. The subtle shift from motivational deficit to skills deficit takes on significance when juxtaposed against the prevailing Freudian theory of the 1940s and 1950s, wherein the etiology of ASD was conceptualized as a fundamental lack of parental love and attention, such that healthy interpersonal relationships were unable to be formed or developed by the child. Accordingly, psychotherapeutic treatment consisted of removing the child to a foster home for recovery.

While Freud's theory had little empirical or heuristic value, the notion of parental causation as a determinant in ASD etiology continued into the 1960s and 1970s. It is at this juncture that Bettelheim (1967) compared autism to being imprisoned in a concentration camp and directly linked indifferent mothers to its etiology. This conceptual shift did not affect the core belief that autism is a skills deficit; instead, Bettelheim asserted that these deficits were inflicted by the mother rather than via passive family dynamics. For the next two decades, therapeutic intervention continued to involve removal of children from

their homes; the children were increasingly sent to Bettelheim's institutions to receive "intensive" treatment, typically involving pharmacotherapy (including untested pharmacological agents such as D-lysergic acid diethylamide [LSD]), electric shock therapy, and aversive behavioral conditioning.

Interestingly, it was aspects of behavioral-based psychotherapy used to address problem behaviors among youth with ASD which began to show positive treatment outcomes. The first such techniques were pioneered by Lovaas and colleagues (e.g., Lovaas et al. 1973) and consisted of a comprehensive functional behavior analysis to identify problematic behaviors, task analysis to generate an incremental learning path, and use of shaping to reward progress toward displaying the desired behavioral routines. Taken as a whole, this treatment package represents the first known instance of early intensive psychotherapeutic interventions, wherein behavioral methods were used to instill and develop positive behaviors needed by the child, while simultaneously decreasing or extinguishing negative or challenging behaviors. Although more than 40 years have elapsed since these techniques were introduced for children with autism, they remain highly effective for skill-building and management of undesirable behaviors (e.g., Matson and Smith 2008; Sofronoff and Beaumont 2009).

Beyond changes in conceptualization, the issue of psychotherapy effectiveness is further clouded by the observation that the social, emotional, and behavioral skills commonly found to be impaired among children with ASD (e.g., Brereton et al. 2006; Cohen and Volkmar 1997; Myers et al. 2007) are the foundational skills for most forms of psychotherapy (Cashin 2008). As a result, the magnitude of such deficits is a key prognostic indicator for children (Mackay et al. 2007) and is commonly integrated into psychotherapy addressing higher-level skills and/or comorbid deficits.

In addition to aggressive and/or disruptive behavior management, attempts to address social skills deficits in children with ASD are numerous (e.g., Kamps et al. 1992; Koegel et al. 1992;

Krasny et al. 2003; Ozonoff and Miller 1995; Parsons and Mitchell 2002; Paxton and Estay 2007); however, the findings from most evaluative studies indicate that traditional social skills training shows relatively limited outcomes for children with ASD. Specific criticisms include the following: most programs are intended for application with various populations of neurotypical children (i.e., not children with ASD); a fundamental lack of agreement as to what constitutes the group of behaviors considered necessary for social skills training; lack of techniques to promote or monitor generalization of acquired skills throughout the course of the curriculum and beyond; and a failure to consider and account for variability in cognitive functioning of target children (e.g., Rao et al. 2008; Reichow and Volkmar 2010; White et al. 2007).

More recent studies have addressed comorbid anxiety and problems with social functioning in children with ASD, typically through using specific techniques (e.g., small group exercises, increasing structure within all activities, and parent training) within a more typical cognitive-behavioral therapy framework. Examples include "Exploring Feelings: Cognitive Behavior Therapy to Manage Anxiety" (Attwood 2004), "Facing your Fears" (Reaven et al. 2012), and "Behavioral Interventions for Anxiety in Children with Autism" (BIACA; Wood and Drahota 2006; Wood et al. 2009a). While maintaining a focus similar to psychotherapy for typically developing children (e.g., exposure, emotion recognition, cognitive therapy, and relaxation), these programs also include specific strategies designed to address skill deficiencies common among children with ASD (e.g., nonverbal communication, prosocial behaviors, emotional regulation, and perspective taking).

Current Knowledge

Relative to the general pediatric population, children with ASD are at a heightened risk for certain comorbid mental health conditions, such as anxiety, depression, externalizing behaviors,

eating disorders, and/or bipolar disorder (e.g., Anderson and Morris 2006; Brereton et al. 2006; Gilliot et al. 2001). As mentioned earlier, cognitive-behavioral psychotherapy shows strong empirical support for the treatment of symptoms used as diagnostic markers for ASD, as well as for associated comorbid conditions (e.g., Chambless and Hollon 1998; Kazdin and Weisz 2003). As such, it became necessary to modify traditional cognitive-behavioral techniques to address the particular combination of diagnostic symptoms and comorbid conditions in children diagnosed with ASD (e.g., Attwood 2003, 2004).

Examination of treatment elements of cognitive-behavioral therapy for neurotypical youth reveals some commonality across targeted problems. For example, cognitive-behavioral therapy for anxiety disorders (e.g., Wood and McLeod 2008; Wood et al. 2006) includes affective education, exposure therapy, cognitive restructuring, contingency management, and problem-solving. Cognitive-behavioral treatments commonly used for most presenting concerns contain some or all of these elements (e.g., Anderson and Morris 2006). Therefore, any necessary modification to such programs for children with ASD is related to previously mentioned social, emotional, and cognitive deficits rather than to the comorbid conditions (Paxton and Estay 2007; Shapiro 2009).

Extending this idea can provide guidelines for determining the appropriateness of psychotherapeutic intervention. For example, the psychotherapy protocol must account for the child's language and cognition skills; specifically, that their skills are sufficiently advanced to allow for meaningful discourse within session. If these skills are not sufficiently developed, then the protocol must include contingencies for building and generalizing such skills. A reluctance to speak or participate may be addressed through improving rapport and information sharing with the parents such that more information is gained about the child for use in session (e.g., increasing relevance of materials, rapport building through common interests). Similarly, positive reinforcement methods (i.e., shaping) may be used to address reduced language skills. Further, a mechanism for increasing the child's awareness

and self-reflection must be included to allow for generalization of new skills to novel situations (Shapiro 2009).

To date, there are relatively few randomized clinical trials examining the use of various forms of psychotherapy among children with ASD; however, the studies available are promising including a recent large-scale study (Wood et al. 2019). For example, a meta-analysis of studies using manualized psychotherapy protocols for children with ASD and comorbid anxiety reported significant reductions in anxiety symptoms, increased independent living skills, increased usage of adaptive coping strategies, and improved quality of life (Sukhodolsky et al. 2013). A preliminary examination of intensive (daily) psychotherapy addressing comorbid obsessive-compulsive symptoms among youth with ASD reported similar positive responses and gains related to adaptive functioning (Iniesta-Sepulveda et al. 2018). While generally similar to psychotherapy for children without ASD, these protocols typically are more intensive with respect to foundational skill building to address presumed deficits in cognitive, social, and emotional functioning (e.g., Wood et al. 2009a).

Future Directions

Although a growing research base exists supporting the utility of psychotherapy for social and behavioral problems among children with ASD, there remains a need for investigation into the efficacy of psychological treatments for adolescents with similar comorbid diagnoses. Though preliminary, a few studies provide support for effectively addressing comorbid anxiety symptoms among adolescents with ASD (e.g., Storch et al. 2015; Wood et al. 2015). These protocols place an emphasis upon social situations and basic skills unique to adolescent populations (e.g., social coaching to address adolescent peer relationship building, emotional regulation with respect to peer harassment/bullying, and independent living skills) in addition to core CBT elements (e.g., exposure).

Ultimately, the true measure of success for psychotherapy in children and adolescents with

ASD will be the degree to which treatment outcomes are clinically meaningful and translate into changes in functioning and quality of life. Some preliminary results suggest that psychotherapy incorporating in vivo exposure is superior to waitlist controls with respect to improvement in parent-reported social responsiveness, and that psychotherapeutic gains are durable over the short term (e.g., Wood et al. 2009b). As well, it remains to be seen how evidence-based forms of psychotherapy fare in randomized clinical trials against both pharmacological and exclusively behavioral techniques.

See Also

- [Anxiety Disorders](#)
- [Asperger Syndrome](#)
- [Autism](#)
- [Autism Spectrum Disorders](#)
- [Behavior Modification](#)
- [Cognitive Behavioral Therapy \(CBT\)](#)

References and Reading

- Abbass, A. A., Hancock, J. T., Henderson, J., & Kisely, S. (2006). Short-term psychodynamic psychotherapies for common mental disorders. *Cochrane Database of Systematic Reviews*, 4, CD004687.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association.
- Anderson, S., & Morris, J. (2006). Cognitive behavior therapy for people with Asperger syndrome. *Behavioural and Cognitive Psychotherapy*, 34, 293–303.
- Attwood, T. (2003). Frameworks for behavioral interventions. *Child and Adolescent Psychiatric Clinics of North America*, 12, 65–86.
- Attwood, T. (2004). Cognitive behaviour therapy for children and adults with Asperger's syndrome. *Behaviour Change*, 21, 147–161.
- Barker, R. L. (1999). *The social work dictionary* (4th ed.). Washington, DC: NASW Press.
- Bettelheim, B. (1967). *The empty fortress: Infantile autism and the birth of the self*. New York: Free Press.
- Bleuler, E. (1911). Dementia praecox oder gruppe der schizophrenien. In G. Anshaffenburg (Ed.), *Handbuch der psychiatrie*. Leipzig: Deuticke.
- Brereton, A., Tonge, B., & Einfeld, S. (2006). Psychopathology in children and adolescents with autism compared to young people with intellectual disability. *Journal of Autism and Developmental Disorders*, 36, 863–870.
- Cashin, A. (2008). Narrative therapy: A psychotherapeutic approach in the treatment of adolescents with Asperger's disorder. *Journal of Child and Adolescent Psychiatric Nursing*, 21, 48–56.
- Chambless, D. L., & Hollon, S. D. (1998). Defining empirically supported therapies. *Journal of Consulting and Clinical Psychology*, 66, 7–18.
- Churchill, R., Hunot, V., Corney, R., Knapp, M., McGuire, H., ... Wessely, S. (2001). A systematic review of controlled trials of the effectiveness and cost-effectiveness of brief psychological treatments for depression. *Health Technology Assessment*, 5, 1–173.
- Cohen, D. J., & Volkmar, F. (1997). Conceptualization of autism and intervention practices: International perspectives. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 947–950). New York: Wiley.
- Gilliot, A., Furniss, F., & Walter, A. (2001). Anxiety in high functioning children with autism. *Autism*, 5, 277–286.
- Haby, M. M., Donnelly, M., Corry, J., & Vos, T. (2006). Cognitive behavioural therapy for depression, panic disorder and generalized anxiety disorder: A meta-regression of factors that may predict outcome. *Australian and New Zealand Journal of Psychiatry*, 40, 9–19.
- Iniesta-Sepulveda, M., Nadeau, J. M., Ramos, A., Kay, B., Riemann, B. C., & Storch, E. A. (2018). An initial case series of intensive cognitive-behavioral therapy for obsessive-compulsive disorder in adolescents with autism spectrum disorder. *Child Psychiatry & Human Development*, 49, 9–19.
- Kamps, D. M., Leonard, B. R., Vernon, S., Dugan, E. P., Delquadri, J. C., ... Folk, L. (1992). Teaching social skills to students with autism to increase peer interactions in an integrated first-grade classroom. *Journal of Applied Behavior Analysis*, 25, 281–88.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kazdin, A. E., & Weisz, J. R. (Eds.). (2003). *Evidence-based psychotherapies for children and adolescents*. New York: Guilford Press.
- Koegel, L. K., Koegel, R. L., Hurley, C., & Frea, W. D. (1992). Improving social skills and disruptive behavior in children with autism through self-management. *Journal of Applied Behavior Analysis*, 25, 341–353.
- Krasny, L., Williams, B. J., Provencal, S., & Ozonoff, S. (2003). Social skills interventions for the autism spectrum: Essential ingredients and a model curriculum. *Child and Adolescent Psychiatric Clinics of North America*, 12, 107–122.
- Leichsenring, F., Rabung, S., & Leibing, E. (2004). The efficacy of short-term psychodynamic psychotherapy in specific psychiatric disorders: A meta-analysis. *Archives of General Psychiatry*, 61, 1208–1216.
- Lovaas, O. I., Koegel, R., Simmons, J. Q., & Long, J. S. (1973). Some generalization and follow-up measures

- on autistic children in behavior therapy. *Journal of Applied Behavior Analysis*, 6, 131–166.
- Mackay, T., Knott, F., & Dunlop, A. (2007). Developing social interaction and understanding in individuals with autism spectrum disorders: A groupwork intervention. *Journal of Intellectual and Developmental Disability*, 32, 279–290.
- Matson, J. L., & Smith, K. R. M. (2008). Current status of intensive behavioural interventions for young children with autism and PDD-NOS. *Research in Autism Spectrum Disorders*, 2, 60–74.
- Myers, S. M., Plauche-Johnson, C., & Council on Children with Disabilities. (2007). Management of children with autism spectrum disorders. *Pediatrics*, 120, 1162–1182.
- Ozonoff, S., & Miller, J. N. (1995). Teaching theory of mind: A new approach to social skills training for individuals with autism. *Journal of Autism and Developmental Disorders*, 25, 415–433.
- Parsons, S., & Mitchell, P. (2002). The potential of virtual reality in social skills training for people with autistic spectrum disorders. *Journal of Intellectual Disability Research*, 46, 430–443.
- Paxton, K., & Estay, I. A. (2007). *Counselling people on the autism spectrum: A practical manual*. London: Jessica Kingsley.
- Rao, P., Beidel, D., & Murray, M. (2008). Social skills interventions for children with Asperger syndrome or high functioning autism: A review and recommendations. *Journal of Autism and Developmental Disorders*, 38, 353–361.
- Reaven, J., Blakeley-Smith, A., Culhane-Shelburne, K., & Hepburn, S. (2012). Group cognitive behavioral therapy for children with high-functioning Autism Spectrum Disorder and anxiety: A randomized control. *Journal of Child Psychology and Psychiatry*, 53, 410–419.
- Reichow, B., & Volkmar, F. (2010). Social skill interventions for individuals with autism: Evaluation for evidence-based practices within a best evidence synthesis framework. *Journal of Autism and Developmental Disorders*, 40, 149–166.
- Shapiro, T. (2009). Psychotherapy for autism. *Journal of Infant, Child, and Adolescent Psychotherapy*, 8, 22–31.
- Smith, M. L., Glass, G. V., & Miller, T. I. (1980). *The benefits of psychotherapy*. Baltimore: Johns Hopkins University Press.
- Sofronoff, K., & Beaumont, R. (2009). The challenges of working with young people diagnosed with Asperger syndrome. In D. McKay & E. A. Storch (Eds.), *Cognitive-behavior therapy for children: Treating complex and refractory cases* (pp. 421–433). New York: Springer.
- Storch, E. A., Lewin, A. B., Collier, A. B., Arnold, E., De Nadai, A. S., Dane, B. F., . . . Murphy, T. K. (2015). A randomized controlled trial of cognitive-behavioral therapy versus treatment as usual for adolescents with autism spectrum disorders and comorbid anxiety. *Depression & Anxiety*, 32, 174–81.
- Sukhodolsky, D. G., Bloch, M. H., Panza, K. E., & Reichow, B. (2013). Cognitive-behavioral therapy for anxiety in children with high-functioning autism: A meta-analysis. *Pediatrics*, 132, e1341–e1350.
- White, S., Keonig, K., & Scahill, L. (2007). Social skill development in children with autism spectrum disorder: A review of intervention research. *Journal of Autism and Developmental Disorders*, 37, 1858–1868.
- Wood, J. J., & Drahota, A. (2006). *Cognitive behavioral therapy for anxiety and social problems in children with autism: Intervention manual*. Los Angeles: UCLA.
- Wood, J. J., & McLeod, B. M. (2008). *Child anxiety disorders: A treatment manual for practitioners*. New York: Norton.
- Wood, J. J., Piacentini, J. C., Southam-Gerow, M., Chu, B. C., & Sigman, M. (2006). Family cognitive behavioral therapy for child anxiety disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45, 314–324.
- Wood, J. J., Drahota, A., Sze, K., Har, K., Chiu, A., & Langer, D. A. (2009a). Cognitive behavioral therapy for anxiety in children with autism spectrum disorders: A randomized, controlled trial. *Journal of Child Psychology and Psychiatry*, 50, 224–234.
- Wood, J. J., Drahota, A., Sze, K., van Dyke, M., Decker, K., . . . Spiker, M. (2009b). Brief report: Effects of cognitive behavioral therapy on parent-reported autism symptoms in school-age children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 39, 1608–12.
- Wood, J. J., Ehrenreich-May, J., Allessandri, M., Fujii, C., Renno, P., . . . Storch, E. A. (2015). Cognitive behavioral therapy for early adolescents with autism spectrum disorders and clinical anxiety: A randomized, controlled trial. *Behavior Therapy*, 46, 7–19.
- Wood, J. J., Kendall, P. C., Wood, K. S., Kerns, C. M., Seltzer, M., Small, B. J., Lewin, A. B., & Storch, E. A. (2019). Cognitive Behavioral Treatments for Anxiety in Children with Autism Spectrum Disorder: A Randomized Clinical Trial. *JAMA Psychiatry*. <https://doi.org/10.1001/jamapsychiatry.2019.4160>

Psychotic Disorder

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Synonyms

Psychosis; Schizophrenia

Definition

Psychotic disorders are a group of psychiatric disorders in which distortion of reality and severe disorganization are present. Reality distortions include hallucinations, which include sensory experiences in the absence of external stimulation that appear real, and delusions, false beliefs that persist, despite evidence to the contrary. Disorganization can be reflected in speech, thought, or in behavior. According to the DSM-IV-TR (American Psychiatric Association 2000), the psychotic disorders include schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, brief psychotic disorder, and shared psychotic disorder. In addition, psychotic disorder can be diagnosed due to a general medical condition or as a substance-induced disorder. A diagnosis of psychotic disorder not otherwise specified indicates that there is not enough information to make a more specific diagnosis or that there are insufficient symptoms to meet the criteria of one of the specific diagnoses. The lifetime prevalence of psychotic disorders in the general population is estimated to be about 2%.

To receive a diagnosis of schizophrenia, an individual must present with two or more of five characteristic symptoms including delusions, hallucinations, disorganized speech, disorganized or catatonic behavior, and negative symptoms. Negative symptoms include dulling of affect and avolition which is a lack of self-directed behavior. The onset of schizophrenia can be slow and gradual or can be acute. There must be social or occupational dysfunction, and the duration of the symptoms must be at least 6 months. The course of the illness can present as a single episode, multiple episodes, or continuous.

There are several subtypes of schizophrenia that are defined based on the prominence of specific symptoms. The subtypes include paranoid, disorganized, catatonic, undifferentiated, and residual type. The committee for the development of the DSM-V has considered removing the subtype distinction due to relative instability of categorization across time and questionable validity (American Psychiatric Association 2010). If there is a history of autistic disorder or pervasive

developmental disorder, the DSM-IV-TR states that a diagnosis of schizophrenia can be made only if there are prominent delusions or hallucinations that are present for at least a month (or less if it is successfully treated). To receive a diagnosis of schizophreniform disorder, the individual must have symptoms of schizophrenia without a functional decline, and the episode is of short duration from 1 to 6 months.

In schizoaffective disorder, there are characteristic symptoms of schizophrenia along with either a depressive, manic, or mixed mood episode. The mood disorder must be present during much of the illness.

In delusional disorder, there are nonbizarre delusions of at least 1-month duration. Unusual behavior is directed toward the delusions. Hallucinations, if they occur, are related to the delusions. The individual's functioning is not significantly impaired.

In brief psychotic disorder, the symptoms last between 1 day and 1 month. Full recovery is the typical outcome.

Shared psychotic disorder occurs when one individual shares a delusion that is maintained by another individual. The symptoms are usually grandiose or paranoid.

Infantile psychosis and early childhood schizophrenia at one time were terms used to describe autism. Kanner (1949) stated that autism would likely be determined to be an early type of schizophrenia. In later years, Rutter (1972) distinguished between autism and schizophrenia based on a number of factors such as age of onset and the course of the disorders. Case reports of individuals diagnosed with autism in childhood and found to be diagnosed with psychotic disorders as adults raised the question of whether autism in childhood increases the likelihood of psychosis in adult life (e.g., Clarke et al. 1999). Based on a larger study group of well-documented cases, Volkmar and Cohen (1991) found that the frequency of schizophrenia in autism is comparable to the general population (about 1%).

Individuals with ASD may be misdiagnosed as psychotic if there is an appearance of a thought disorder. The presence of a thought disorder is evidenced by bizarre, disorganized, or

idiosyncratic speech. Individuals with ASD may have unusual interests and ways of expressing themselves verbally. They use language concretely and have difficulty describing abstract or emotional concepts (Howlin 1997).

Significant impairments in language and cognition occur commonly in autism spectrum disorders which poses a challenge to the accurate diagnosis of psychiatric disorders. The DM-ID was developed jointly by the American Psychiatric Association and the National Association for the Dually Diagnosed (2007) to address the difficulties inherent in making psychiatric diagnoses with persons with intellectual disability. The DM-ID suggests adaptations to the DSM-IV-TR. The adaptations include using reports from others as a substitute for self-report and reducing the number of required symptoms for making a diagnosis if the individual is nonverbal. In the case of the psychotic disorders, the DM-ID notes that engaging in self-talk is relatively common in individuals with intellectual disability and it should not be interpreted as a symptom of psychotic disorder (p. 150).

See Also

► [Schizophrenia](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed.). Washington, DC: Author. Text Rev.
- American Psychiatric Association. (2010). *Proposed draft revisions to DSM disorders and criteria*. Retrieved 23 Feb 2011 from <http://www.dsm5.org>
- Clarke, D., Baxter, M., Perry, D., & Prasher, V. (1999). The diagnosis of affective and psychotic disorders in adults with autism: Seven case reports. *Autism*, 3(2), 149–164.
- Fletcher, R., Loschen, E., Stavrakaki, C., & First, M. (Eds.). (2007). *Diagnostic manual – Intellectual disability (DM-ID): A clinical guide for diagnosis of mental disorders in persons with intellectual disability*. Kingston: NADD Press.
- Howlin, P. (1997). *Autism: Preparing for adulthood*. London: Routledge.
- Kanner, L. (1949). Problems of nosology and psychodynamics of early infantile autism. *American Journal of Orthopsychiatry*, 19, 416–426.
- Rutter, M. (1972). Childhood schizophrenia reconsidered. *Journal of Autism and Childhood Schizophrenia*, 2, 315–337.
- Smith, A. H. W., Haut, F., & Fleisher, M. D. (2007). Schizophrenia and other psychotic disorders. In R. Fletcher, E. Loschen, C. Stavrakaki, & M. First (Eds.), *Diagnostic manual – Intellectual disability (DM-ID): A clinical guide for diagnosis of mental disorders in persons with intellectual disability* (pp. 143–156). Kingston: NADD Press.
- Volkmar, F. R., & Cohen, D. J. (1991). Comorbid association of autism and schizophrenia. *American Journal of Psychiatry*, 148(12), 1705–1707.

Psychotic Symptoms in Autism

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Definition

Autism spectrum disorders are neurodevelopmental disorders that in DSM-V are characterized by impaired social interaction and communication, as well as restricted interests and repetitive behaviors. In addition, individuals diagnosed with ASD may exhibit broader phenomenology including psychotic symptoms. The presence of psychotic symptoms in ASD is a substantial source of morbidity, often being a reason for both hospitalization and the prescription of antipsychotic medications. The relationship between autism spectrum disorders and psychosis is both complex and interesting. Differentiation of these conditions, accurate diagnosis, and appreciation of the comorbidity is often challenging in the light of significant clinical overlap.

ASD and psychotic disorders have overlapping clinical phenomenology, neurobiology, and genetic risk factors. Further, studies have found higher prevalence rates of psychotic disorders, in addition to schizophrenia, in individuals with ASD as compared to the general population. Overall, research in the field of childhood neurodevelopmental disorders has identified evidence for a connection between developmental

disorders that manifest in childhood and the subsequent risk of psychotic experiences in adolescence.

Historical Background

There has been significant appreciation of the complexity and distinctive states of psychotic disorders and autism leading to a shift from how they were historically conceptualized. Bleuler categorized autism as a core feature of schizophrenia (Bleuler 1950) and Bender described autism as childhood onset schizophrenia (Bender 1947). Kraepelin (1971) had also recognized that many children who went on to present with dementia praecox “exhibited a quiet, shy, retiring disposition, made no friendships, lived only for themselves.” In adulthood, these individuals often received the diagnosis of schizoid or schizotypal personality disorders which themselves are associated with the risk of later development of schizophrenia. In the 1970s, Autism began to emerge as a separate condition, the distinction based more so in terms of an early age of onset for ASD and led to its official acknowledgement as a separate disorder in DSM-III. In 1972, Rutter addressed the confusion between these conditions and the way the term childhood schizophrenia was applied to various other conditions leading to the change in terminology.

Clinically, the neural deficits of schizophrenia are present earlier during development; however, symptoms of schizophrenia such as behavioral disorganization, delusions and hallucinations, apathy, and amotivation often present later in adolescence or in early adulthood (Lieberman 1999). Even in the case of childhood onset schizophrenia, formative accounts described a psychotic syndrome with a clear age of onset (Kolvin et al. 1971; Russell et al. 1989). However, symptoms of autism spectrum disorders are thought to manifest themselves from the very early periods of development and have a more persistent course (Kanner 1949).

Despite this important distinction, there are nevertheless challenges in differentiating these disorders. A number of adults with psychotic illness have some history of ASD-like symptoms in

childhood, a finding that some have described as indicating “a non-specific marker of severe early abnormal neurodevelopment” (Sporn et al. 2004). Shared genetic and neurodevelopmental processes underlying both conditions were highlighted in the evidence reviewed by Rapaport et al. (2009). In support of this perspective, a study by Sullivan et al. (2013) reports on a large cohort where a diagnosis of ASD was found to predict future psychotic experiences. It also highlights that the relationship between these conditions is further complicated by the hypothesis that there may be “an artificial overlap” between COS and ASD due to the “similarities in descriptive phenomenology.” Particularly, the social deficits in ASD may appear similar to negative symptoms of schizophrenia, and furthermore, recent work has found that psychotic experiences can co-occur with ASD, though this may also reflect concrete responses to probes regarding psychiatric symptomatology (Sullivan et al. 2013). Other areas where this overlap is evident include theory of mind problems, behavioral problems, and language difficulties (Fitzgerald 2012). The heterogeneity and spectrum of etiology and presentation of these disorders are a very important consideration when drawing and in the future redrawing diagnostic boundaries between these conditions.

Current Knowledge

There are unique challenges involved in attempting to diagnose psychotic symptoms in autism spectrum disorders. Experimental evidence highlights meaningful distinctions and consistencies between psychotic disorders and ASD. There is evidence for disruption of embryonic brain development in both ASD and psychosis (Delacy and King 2013). The NMDA receptor function and glutamate signaling pathway disruptions have been implicated in the pathophysiology of schizophrenia and ASD. There are particular areas of neurodevelopmental deficits that are shared between ASD and psychosis, such as deficits in theory of mind, functional deficits in the mirror neuron system, and abnormalities in the functional connectivity of the brain. The study by Walter et al. (2010) highlights theory of

mind, mirror neuron, and functional connectivity deficits in a sample of healthy volunteer subjects identified via the presence of the psychosis risk allele variant of the ZNF804A gene. The genetic overlap between the two continues to be explored, as evident in the two large case-control studies which have shown that parental schizophrenia is a significant risk factor for autism (Larsson et al. 2005; Daniels et al. 2008). Rapoport et al. (2009) also highlight the growing weight of evidence from candidate gene, linkage, and expression studies, and copy number of variants that appear to be shared in both autism and schizophrenia. The velocardiofacial syndrome (chromosome 22q11) deletion shows high rates of childhood ASDs (30 [50%] of 60) and psychotic symptoms (16 [26.7%] of 60). Although a study of common single-nucleotide polymorphisms found only a low genetic correlation between ASD and schizophrenia (Vorstman et al. 2013). Deletions, disruptions, and missense mutations in neurexin 1 (NRXN1) have been reported recently in several cases with autism (Marshall et al. 2008; Kim et al. 2008; Zahir et al. 2008; TAGP Consortium 2007; Feng et al. 2006; Weiss et al. 2008) and two cases with schizophrenia (Kirov et al. 2008; Zoghbi 2003). Additionally, certain risk factors are also shared by ASD and psychosis such as high paternal age, obstetric complications, and maternal infections.

Brain imaging studies in autism show increased head size/total brain volume seen in the first 3 years of age based on cross-sectional studies (Hazlett et al. 2005). By contrast, COS studies demonstrate loss of cortical gray matter in early ages that progresses through adolescent years, which seems to be an exaggeration of the normal pattern of cortical brain development. It may be surmised that in ASD there is an acceleration of early postnatal brain development when compared to COS demonstrating exaggerated brain maturation in childhood and adolescence. With respect to normal brain development “both patterns may be seen as an abnormal shift to the left with autism showing initial overgrowth and COS showing greater pruning down of cortex in early and middle part of the trajectory, both accelerations normalizing with age” (Rapoport et al. 2009).

In regard to pharmacological treatment, antipsychotics are the most commonly prescribed class of medications for ASD (Hsia et al. 2013). While the use of antipsychotics has been the mainstay of treatment of psychotic disorders, the FDA-approved medications for treatment of aggression associated with ASD are also antipsychotic agents such as risperidone and aripiprazole. There is a growing interest in the modulation of the glutamatergic signaling pathways by these agents and their efficacy in both ASD and treatment of psychotic symptoms.

However, the presentation of psychotic symptoms can be highly variable in ASD. A recent study of a dually affected cohort of patients with ASD (Larson et al. 2017) found significant differences in the autistic phenotype of patients who had comorbid psychotic illness compared to those with ASD alone. Those with comorbid psychotic illness showed fewer lifetime stereotyped, repetitive, or restrictive interests/behaviors, with greater diagnosis of psychosis NOS and lower rates of diagnosis of schizophrenia.

The evidence around the phenomenology of psychotic symptoms in ASD remains limited and unsystematic pointing to the possibility of further subtypes of individuals with ASD with psychotic comorbidity. One possibility proposed is the subtype of ASD that carries a higher risk of psychotic illness mediated by common genetic variants. In this case, significant differences would be anticipated in presentation of ASD features in patients with ASD with psychosis than ASD patients without psychosis, and that the psychotic symptoms would be similar across people with ASD (Larson et al. 2017; King and Lord 2011). The other consideration is of misdiagnosis of features of ASD as psychosis (Van Schalkwyk et al. 2015; Lai and Baron-Cohen 2015) given difficulties with theory of mind and reading other people's intentions that may resemble paranoia, expressive communication difficulties that can be mistaken for thought disorder and “melt downs” that resemble catatonia. It can be hypothesized that individuals with ASD may experience some degree of delusions without the presence of any additional impairment, but that hallucinations are likely indicative of a distinct process (Van Schalkwyk et al. 2017).

Diagnosing psychotic symptoms in an ASD patient also is complicated by factors such as deficits in language and a tendency towards concrete responses. Furthermore, “these psychopathological alternatives might simply be epiphenomena of different constructs and language from separate literatures applied to the same mental phenomena” (Larson et al. 2017), such that the experiences of the individuals with ASD can fit as part of both categories of ASD and psychosis.

Finally and very importantly, there are additional psychosocial stressors that are compounded by the stress resulting from the impairments associated with ASD that can elevate the risk for developing psychotic symptoms, such as social exclusion, which is common in ASD, has been hypothesized to be an important risk factor for nonaffective psychotic disorders (Selten et al. 2015; Gevonden et al. 2014). During adolescence, many psychiatric illnesses are likely to have their initial presentation, increasing the pertinence of this consideration. As these individuals undergo the transition from late adolescence to early adulthood there is an additional risk of substantial change as they may lose the support of entitlements and treatment networks that were available to them at younger ages (Volkmar et al. 2014). The effects of these changes may be that these individuals are seen by adult psychiatrists with comparatively less understanding of their condition, and perhaps more importantly, must interact with a treatment system that is not well-oriented to their needs (Volkmar et al. 2014). Individuals with behavioral problems are at increased risk of being admitted to inpatient units (Tsakanikos et al. 2007). As Kanner himself pointed out, adolescence is a common time during which some individuals with autism would experience deterioration (Kanner 1971) and this is also the typical age of onset of psychotic disorders.

Future Directions

It is evident that individuals with ASD have a higher risk of developing psychotic symptoms and will be treated in medical and mental health settings. It is often the case that the diagnosis of

ASD is made much later or has been overlooked due to several reasons prior to this, leaving the burden of diagnostic clarity and treatment on the shoulders of the clinical team to tease apart the symptoms based on etiology, phenomenology, and appreciation of overlapping presentations. The development of a multidimensional model that helps understand the complexity of these two conditions and expands to encompass affective, psychotic, and autism spectrum characteristics (Larson et al. 2017) will be important in addressing this diagnostic quandary. Further research in genetic markers, clarification of genetic and syndrome heterogeneity, and the population samples that have closer correlation to those seen more commonly in clinical practice will be significant in providing insight into early diagnosis and treatment of this comorbid condition.

References and Reading

- Bender, L. (1947). Childhood schizophrenia. *The American Journal of Orthopsychiatry*, 17, 40–56.
- Bleuler, E. (1950). *Dementia praecox or the group of schizophrenias*. New York: International Universities Press.
- Daniels, J. L., Forssen, U., Hultman, C. M., et al. (2008). Parental psychiatric disorders associated with autism spectrum disorders in the offspring. *Pediatrics*, 121(5), e1357–e1362.
- de Lacy, N., & King, B. H. (2013). Revisiting the relationship between autism and schizophrenia: Toward an integrated neurobiology. *Annual Review of Clinical Psychology*, 9, 555–587.
- Feng, J., Schroer, R., Yan, J., et al. (2006). High frequency of neurexin 1 beta signal peptide structural variants in patients with autism. *Neuroscience Letters*, 409, 10–13.
- Fitzgerald, M. (2012). Schizophrenia and autism/Asperger's syndrome overlap and difference. *Clinical Neuropsychiatry*, 171–176.
- Gevonden, M. J., Booij, J., van den Brink, W., Heijtel, D., van Os, J., & Selten, J. P. (2014). Increased striatal dopamine release in young adults with hearing impairment and its relevance for the social defeat hypothesis of schizophrenia. *JAMA Psychiatry*, 71(12), 1364–1372.
- Hazlett, H. C., Poe, M., Gerig, G., et al. (2005). Magnetic resonance imaging and head circumference study of brain size in autism: Birth through age 2 years. *Archives of General Psychiatry*, 62(12), 1366–1376.
- Hsia, Y., Wong, A. Y. S., Murphy, D. G. M., et al. (2013). Psychopharmacological prescriptions for people with

- autism spectrum disorder (ASD): A multinational study. *Psychopharmacology*, 231, 999–1009.
- Kanner, L. (1949). Problems of nosology and psychodynamics of early infantile autism. *American Journal of Orthopsychiatry*, 19, 416–426.
- Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of Autism and Childhood Schizophrenia*, 1(2), 119–145.
- Kim, H. G., Kishikawa, S., Higgins, A. W., et al. (2008). Disruption of neurexin 1 associated with autism spectrum disorder. *American Journal of Human Genetics*, 82, 199–207.
- King, B. H., & Lord, C. (2011). Is schizophrenia on the autism spectrum? *Brain Research*, 1380, 34–41.
- Kirov, G., Gumus, D., Chen, W., et al. (2008). Comparative genome hybridization suggests a role for NRXN1 and APBA2 in schizophrenia. *Human Molecular Genetics*, 17, 458–465.
- Kolvin, I., Ounsted, C., Humphrey, M., & McNay, A. (1971). Studies in the childhood psychoses. II. The phenomenology of childhood psychoses. *British Journal of Psychiatry*, 118(545), 385–395.
- Kraepelin, E. (1971). *Dementia praecox and paraphrenia* (p. 1236). New York, NY: Robert E Krieger publishing.
- Lai, M.-C., & Baron-Cohen, S. (2015). Identifying the lost generation of adults with autism spectrum conditions. *Lancet Psychiatry*, 2, 1013–1027.
- Larson, F. V., Wagner, A. P., Jones, P. B., Tantam, D., Lai, M.-C., Baron-Cohen, S., & Holland, A. J. (2017). Psychosis in autism: Comparison of the features of both conditions in a dually affected cohort. *The British Journal of Psychiatry*, 210, 269–275.
- Larsson, H. J., Eaton, W. W., Madsen, K. M., et al. (2005). Risk factors for autism: Perinatal factors, parental psychiatric history, and socioeconomic status. *American Journal of Epidemiology*, 161(10), 916–925.
- Lieberman, J. A. (1999). Pathophysiologic mechanisms in the pathogenesis and clinical course of schizophrenia. *The Journal of Clinical Psychiatry*, 60(Suppl 1), 9–12.
- Marshall, C. R., Noor, A., Vincent, J. B., et al. (2008). Structural variation of chromosomes in autism spectrum disorder. *American Journal of Human Genetics*, 82, 477–488.
- Rapoport, J., Chavez, A., Greenstein, D., Addington, A., & Gogtay, N. (2009). Autism spectrum disorders and childhood-onset schizophrenia: Clinical and biological contributions to a relation revisited. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(1), 10–18.
- Russell, A. T., Bott, L., & Sammons, C. (1989). The phenomenology of schizophrenia occurring in childhood. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28(3), 399–407.
- Schalkwyk, G. I. V., Peluso, F., Qayyum, Z., McPartland, J. C., & Volkmar, F. R. (2015). Varieties of misdiagnosis in ASD: An illustrative case series. *Journal of Autism and Developmental Disorders*, 45, 911–918.
- Schalkwyk, G. I. V., Volkmar, F. R., & Corlett, P. R. (2017). A predictive coding account of psychotic symptoms in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47, 1323–1340.
- Selten, J.-P., Lundberg, M., Rai, D., & Magnusson, C. (2015). Risks for non-affective psychotic disorder and bipolar disorder in young people with autism spectrum disorder: A population-based study. *JAMA Psychiatry*, 72, 483–489.
- Sporn, A. L., Addington, A. M., Gogtay, N., Ordoñez, A. E., Gornick, M., Clasen, L., et al. (2004). Pervasive developmental disorder and childhood-onset schizophrenia: Comorbid disorder or a phenotypic variant of a very early onset illness? *Biological Psychiatry*, 55(10), 989–994.
- Sullivan, S., Rai, D., Golding, J., Zammit, S., & Steer, C. (2013). The association between autism spectrum disorder and psychotic experiences in the Avon longitudinal study of parents and children (ALSPAC) birth cohort. *Journal of the American Academy of Child & Adolescent Psychiatry*, 52(8), 806–814.e2.
- TAGP Consortium. (2007). Mapping autism risk loci using genetic linkage and chromosomal rearrangements. *Nature Genetics*, 39, 319–328.
- Tsakanikos, E., Costello, H., Holt, G., Sturmey, P., & Bouras, N. (2007). Behavior management problems as predictors of psychotropic medication and use of psychiatric services in adults with autism. *Journal of Autism and Developmental Disorders*, 37(6), 1080–1085.
- Volkmar, F., Reichow, B., & McPartland, J. (2014). *Adolescents and adults with autism spectrum disorders*. New York: Springer.
- Vorstman, J. A. S., Anney, R. J. L., Derks, E. M., et al. (2013). No evidence that common genetic risk variation is shared between schizophrenia and autism. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 162B(1), 55–60.
- Walter, H., Schnell, K., Erk, S., et al. (2010). Effects of a genome-wide supported psychosis risk variant on neural activation during a theory-of-mind. *Molecular Psychiatry*, 16(4), 462–470.
- Weiss, L. A., Shen, Y., Korn, J. M., et al. (2008). Association between microdeletion and microduplication at 16p11.2 and autism. *The New England Journal of Medicine*, 358, 667–675.
- Zahir, F. R., Baross, A., Delaney, A. D., et al. (2008). A patient with vertebral, cognitive and behavioural abnormalities and a de novo deletion of NRXN1 alpha. *Journal of Medical Genetics*, 239–243.
- Zoghbi, H. (2003). Postnatal neurodevelopmental disorders: Meeting at the synapse? *Science*, 302, 826–830.

PT

► Physical Therapy

Public Law 94-142

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Definition

Public Law 94-142, also known as the Education for All Handicapped Children Act (EAHCA) of 1975, is the landmark federal legislation pertaining to the education of children with disabilities. The law guaranteed a “free, appropriate public education” to all children and young adults aged 3–21. PL 94-142 was an amendment to the Education of the Handicapped Act (EHA) of 1970 and served as Part B of the EHA. Congress has since replaced PL 94-142 with the Individuals with Disabilities Education Act and the Individuals with Disabilities Education Improvement Act, but the tenets of the EAHCA – namely to improve the educational opportunities of students with disabilities – remain intact.

Historical Background

Although considered groundbreaking, PL 94-142 was not the first piece of federal legislation pertaining to the education of students with disabilities. Many pieces of federal legislation beginning in the 1950s had touched on educational issues of students with disabilities, including several portions of bills (e.g., PL 85-926, PL 86-158, PL 87-276, PL 87-715, and PL 88-164), which provided training to teachers of deaf students and students with mental retardation (now called intellectual disability). Additionally, the Elementary and Secondary Education Act of 1965 (PL 89-10) is considered a significant bill pertaining to education because it was the first law to provide federal funding to states for the education of students. (It funded the education of students in specialized state schools for the blind, deaf, and mentally retarded and also provided federal funding to help educate impoverished students.)

The Education of the Handicapped Act of 1970 (EHA) (PL 91-230) was the first federal law to exclusively address the education of children with disabilities. It provided grants to institutions of higher learning to develop programs to train teachers of students with disabilities and funded regional resource centers to deliver technical assistance to state and local school districts. Its first amendment in 1974 mentioned the term “appropriate education” for the first time in federal legislation and added a requirement that states which received federal funds adopt the goal of full educational opportunities for students with disabilities.

Despite the EHA, however, as of 1975, over half of the students with disabilities were not receiving appropriate educational services, including over a million children who were not receiving any public education at all. Many of the students who were receiving educational services received them in institutions far away from their families, at great cost to the families or through the generosity of charities.

Spurred by the civil rights movement and by *Brown v. Board of Education* (1954), which ruled that states must provide equal educational opportunities to children regardless of race, beginning in the 1960s, advocates for students with disabilities began to file lawsuits against their home states and school districts. These lawsuits claimed that exclusion, segregation, and inappropriate educational services for students with disabilities violated the students' rights under the 14th Amendment of the United States Constitution. Cases like *Pennsylvania Association for Retarded Citizens (PARC) v. Commonwealth of Pennsylvania* (1972) were filed by parents of children with disabilities asserting their children's right to a public education.

By 1973, 27 right-to-education lawsuits had been filed on behalf of students with disabilities across 21 states. As a result, the policies being developed across the country varied significantly. While many of the early lawsuits resulted in expanded educational opportunities for children with disabilities, not all lawsuits were won by the students petitioning. Indeed, the combination of district court losses like *Harrison v. Michigan*

(1972) and the United States Supreme Court decision in *San Antonio v. Rodriguez* (1973), a school finance case which held that the Constitution did not establish a right to public education, caused many disability advocates to worry about how the Supreme Court might decide the right to education cases for students with disabilities. Rather than continue to take their chances in the courts, these advocates chose to pursue a legislative agenda.

In addition to the EHA, Congress had already begun to pass several pieces of progressive social legislation, including the Occupational Safety and Health Act of 1970, the Child Development Act of 1971, and Section 504 of the Vocational Rehabilitation Act of 1973, which outlawed discrimination on the basis of disability in programs receiving federal assistance. The climate for an expanded bill related to the education of children with disabilities seemed favorable. Disability and child advocates persuaded Senator Harrison Williams to introduce an amendment to the EHA to ensure that children with disabilities receive a free and appropriate public education (FAPE), to protect the rights of students and their parents, and to assist states in providing educational services to students with disabilities. The legislation was supported by the Council of Chief State School Officers and the National School Boards Association. Indeed, not a single person or organization testified against the bill in Congressional hearings. The bill, which came to be known as the Education for All Handicapped Children Act, or PL 94-142, passed by wide margins. Despite initial resistance by former President Gerald Ford, the President signed the act into law in 1975.

Since the bill's passing in 1975, there have been several amendments to the law. In fact, the laws pertaining to the education of children with disabilities are changed somewhat regularly because portions of the legislation must be reauthorized periodically and because Congress must routinely approve funding. The most significant changes to PL 94-142 include: the Infants and Toddlers with Disabilities Act of 1986 (PL 99-457), which required states to provide programs and services from birth (PL 94-192 only required programs for ages 3–21); the

Individuals with Disabilities Education Act of 1990 (PL 101-476), which changed the name of the law from the Education for All Handicapped Children Act to the Individuals with Disabilities Education Act (IDEA), added two categories of disability – including autism, and supported initiatives for transition to adulthood; the Individuals with Disabilities Education Act Amendments of 1997 (PL 105-17), which strengthened the role of parents, emphasized student progress toward meaningful educational goals, encouraged non-adversarial mediation to resolve disputes, added disciplinary provisions, and made extensive changes to the individualized education plan (IEP); and the Individuals with Disabilities Education Improvement Act of 2004 (PL 108-446), which required the use of scientifically based instructional practices and interventions whenever possible, required special education teachers to be “highly qualified,” revised the discipline procedures, revised dispute resolution procedures, created “early intervening services” to assist students who are struggling but who do not require special education services, reduced paperwork burdens on states and school districts, emphasized student progress and progress monitoring and reporting, and made other changes to the IEP process.

It is important to note that the federal laws pertaining to the education of students with disabilities are subject to state implementation. States are at liberty to provide more rights to students, but cannot provide less than those guaranteed by the federal statute. Each state has its own regulations interpreting and executing the federal law, and thus implementation will vary from state to state.

Current Knowledge

As indicated by the legislative history, the impetus for PL 94-142 was a national recognition of the poor outcomes of children with disabilities due to their unequal educational opportunities and a desire to significantly improve access to education for these children. The law addressed how children with disabilities were identified, how they

would be educated, how to evaluate the success of educational efforts, and how to protect the rights of children with disabilities and their parents. As an improvement on the EHA of 1970, the law authorized significant financial incentives for complying with PL 94-142.

Specifically, the law mandated that “all children with disabilities [must] have available to them . . . a free appropriate public education which emphasizes special education and related services designed to meet their unique needs.” It required that special education and related services for children between the ages of 3 and 21: (1) be provided at public expense; (2) include an appropriate preschool, elementary, and secondary school education in the home state of the student; and (3) be provided in accordance with an Individualized Education Program (IEP) uniquely designed for each student. The federal law also required states to set standards to implement the federal law. Thus, with the passage of PL 94-142, the federal government and the states became partners in the education of students with disabilities.

The concepts arising from PL 94-142 – in particular, the paradigm of a free and appropriate public education, the creation of the individualized education program, and the creation of a federal/state partnership in education – have been expanded upon greatly since 1975. Yet despite frequent litigation over the principles and years of state and federal regulations, PL 94-142 remains the most significant piece of federal legislation pertaining to the education of students with disabilities.

Future Directions

The most current legislation pertaining to the education of individuals with disabilities is Public Law 108-446, commonly referred to as the “Individuals with Disabilities Education Improvement Act of 2004” or “IDEIA.” Other legislation, such as the No Child Left Behind Act of 2001 (PL 107-110) and its subsequent revisions, have also influenced the development of laws pertaining to students with disabilities.

See Also

- [Free Appropriate Public Education](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- Brown v. Board of Education*, 347 U.S. 483 (1954).
 Education for All Handicapped Children Act of 1975, Pub. L. No. 94-142, 89 Stat. 773.
 Education of the Handicapped Act Amendments of 1986, Pub. L. No. 99-457, 100 Stat. 1145.
 Education of the Handicapped Act of 1970, Pub. L. No. 91-230, 84 Stat. 175.
 Elementary and Secondary Education Act Amendments of 1966, Pub. L. No. 89-750, §161, 80 Stat. 328.
 Elementary and Secondary Education Act of 1965, Pub. L. No. 89-10, 79 Stat. 27.
Federal Education Policy and the States, 1945–2009. Retrieved from http://www.archives.nysed.gov/edpolicy/research/res_essay_ford_pl94_142.shtm
 Handicapped Children’s Protection Act of 1986, Pub. L. No. 99-372, 100 Stat. 796.
Harrison v. Michigan, 350 F. Suppl. 846 (E.D. Mich. S.D. 1972).
 Individuals with Disabilities Education Act Amendments of 1997, Pub. L. No. 105-17, 111 Stat. 37.
 Individuals with Disabilities Education Act of 1990, Pub. L. No. 101-476, 104 Stat. 1142.
 Individuals with Disabilities Education Improvement Act of 2004, 20 U.S.C. §§ 1400 *et seq.*, Pub. L. No. 108-446, 118 Stat. 2803.
 Katsiyannis, A., Yell, M. L., & Bradley, R. (2001). Reflections on the 25th anniversary of the Individuals with Disabilities Education Act. *Remedial and Special Education*, 22(6), 324–334.
 Library of Congress. *Bill Summary & Status, Education for All Handicapped Children Act*. Retrieved from <http://thomas.loc.gov/cgi-bin/bdquery/z?d094:SN00006:@@D&summ2=m&#locshare/print>
 No Child Left Behind Act of 2001, 20 U.S.C. §§ 6301 *et seq.*
Pennsylvania Association for Retarded Citizens (PARC) v. Commonwealth of Pennsylvania, 343 F. Suppl. 279 (E.D. Pa. 1972).
 PL 94-142: policy, evolution, and landscape shift (2007). Retrieved May 2, 2011, from <http://www.thefreelibrary.com/PL+94-142%3a+policy%2c+evolution%2c+and+landscape+shift.-a0173465140>
San Antonio v. Rodriguez, 411 U.S. 1 (1973).
 The Infants and Toddlers with Disabilities Act of 1986, 20 U.S.C. §§1471–1485.
 US Office of Special Education Programs. (2000). *Twenty-five years of progress in educating children with disabilities through IDEA*. Retrieved May 2, 2011, from

<http://www2.ed.gov/policy/speced/leg/idea/history.html>

Yell, M. L., Shriner, J. G., & Katsiyannis, A. (2006). Individuals with Disabilities Education Improvement Act of 2004 and IDEA regulations of 2006: Implications for educators, administrators, and teacher trainers. *Focus on Exceptional Children*, 39(1), 1–24.

Public Perception of Autism Treatments: Science Versus Pseudoscience in the Age of Mass Media

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Definition

Autism treatments: Strategies aimed at improving outcomes for individuals with autism spectrum disorder (ASD) by teaching compensatory skills, ameliorating the core behavioral characteristics associated with ASD, and/or minimizing the impact of these behaviors on an individual's functioning. Numerous treatments and/or instructional strategies have been presented as plausible approaches to treating autism-related behaviors. The quality and quantity of research supporting a given practice or treatment vary. Strategies deemed *evidence-based practices* are supported by multiple, methodologically rigorous, and empirical research studies. In contrast, *pseudoscientific practices* are unsupported by credible evidence, yet consumers view them as viable treatment options.

Mass media: Technology that is intended to reach a broad audience. The most common platforms for mass media are newspapers, magazines, radio, television, and the Internet.

Historical Background

Autism spectrum disorder (ASD) is a lifelong disorder. Although there is no cure for ASD, behaviors that characterize the disorder can be

amenable to intervention. Education is believed to be “the best hope for improving outcomes for individuals with ASD” (McIntyre et al. 2013). The concept of evidence-based instruction is central to the provision of educational services to children and youth with ASD in the United States. Federal mandates such as the Every Student Succeeds Act (Pub. L. No. 114-95 § 114 Stat. 1177) and Individuals with Disabilities Education Act (20 U.S.C. § 1400 *et seq*) as well as ethical guidelines for special education professionals emphasize that instruction and intervention for students with disabilities be research based (Council for Exceptional Children 2015). Professional guidelines for special education professionals are summarized in Fig. 1.

Evidence-based practices. Multiple workgroups have conducted systematic reviews of autism intervention research to identify effective interventions and discriminate new and promising approaches from the deceptive and improbable. Most notably are the large-scale reviews conducted by the National Standards Project (National Autism Center 2015) and the National Professional Development Center on ASD (NPDC, Wong et al. 2015). The review process includes screening intervention studies for methodological rigor based on criteria established by panels of experts (Horner et al. 2005; Gersten et al. 2005) and design standards from the What Works Clearinghouse (U.S. Department of Education). Studies with strong methodological rigor are then subjected to content analysis in which researchers identify specific components of the intervention. Interventions that have multiple studies that support its effectiveness are deemed *evidence-based practices* (EBPs). The key distinction here is that EBPs have multiple, methodologically rigorous experimental studies, that have been conducted across different research groups.

Although there are differences in how each work group conceptually organized and labeled specific instructional strategies, the majority of the EBPs for individuals with ASD are based on foundational applied behavior analytic concepts (e.g., prompting, reinforcement, task analysis) or organized into multicomponent interventions that

The Council for Exceptional Children (CEC, 2015) identified 12 ethical principles to guide special educators' professional practice. The principles highlighted in bold specifically relate to the use of evidence-based practices:

1. Maintaining challenging expectations for individuals with exceptionalities to develop the highest possible learning outcomes and quality of life potential in ways that respect their dignity, culture, language, and background.
2. **Maintaining a high level of professional competence and integrity and exercising professional judgement to benefit individuals with exceptionalities and their families.**
3. Promoting meaningful and inclusive participation of individuals with exceptionalities in their schools and communities.
4. Practicing collegiality with others who are providing services to individuals with exceptionalities.
5. Developing relationships with families based on mutual respect and actively involving families and individuals with exceptionalities in educational decision making.
6. **Using evidence, instructional data, research, and professional knowledge to inform practice.**
7. **Protecting and supporting the physical and psychological safety of individuals with exceptionalities.**
8. **Neither engaging in nor tolerating any practice that harms individuals with exceptionalities**
9. Practicing within the professional ethics, standards, and policies of CEC; upholding laws, regulations and policies that influence professional practice; and advocating improvements in the laws, regulations, and policies.
10. **Advocating for professional conditions and resources that will improve learning outcomes of individuals with exceptionalities.**
11. Engaging in the improvement of the profession through active participation in professional organizations.
12. Participating in the growth and dissemination of professional knowledge and skills.

Public Perception of Autism Treatments: Science Versus Pseudoscience in the Age of Mass Media, Fig. 1 CEC Ethical principles & practice standards for special educators

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incorporate foundational concepts (e.g., functional communication training, pivotal response training, naturalistic intervention). In addition, there are a set of practices that have been routinely incorporated into a package of positive behavior intervention and support (e.g., functional assessment, extinction, redirection, antecedent interventions, direct reinforcement of other behavior). It should be noted that the primary target of EBPs are specific skills that will lend to improve outcomes and/or overall functioning. These practices are intended as key elements of instruction, to be implemented consistently and over a period of time. The general goal of any EBP is not to eradicate symptoms or serve as a quick "cure."

Pseudoscientific treatments. Treatments with a basis in pseudoscience offer solutions that are in stark contrast to scientific principles. These treatments typically do not have a foundation based on past scientific findings or controlled studies, are not refined over time as new evidence is disseminated, and either do not contribute to the extant research and/or are incompatible with existing and accepted theories (Mugaloglu 2014). Defining characteristics of pseudoscientific approaches includes the following: a lack of testability, self-correction, and/or peer review, an emphasis placed on confirmation and anecdotal evidence, correlating practices with the past as a means of arguing correctness, overly technical language,

and the presence of extraordinary claims related to the practice in question (Boudry et al. 2015; Lilienfeld et al. 2012). A table contrasting characteristic features of science and pseudoscience can be located in Table 1.

The field of ASD has been concerned with the spread of misinformation for years (Ganz et al. 2018). Facilitated communication (FC), for instance, was a practice that gained popularity during the 1990s through early 2000. FC is an intervention designed to address the complex communication needs impacting many students with disabilities, including ASD. A facilitator provides physical support to assist an individual in selecting words and/or phrases on an augmentative and alternative communication (AAC) device (Ganz et al. 2018). Methodologically rigorous studies that attribute that the supposed effects of improved communication to the facilitator rather than the individual with ASD have been used to debunk the practice. Several professional

organizations have since released position statements opposing the use of FC, including the American Academy of Pediatrics (1998), American Association on Mental Retardation (1994), American Psychological Association (1994), Association for Behavior Analysis (1995), and American Speech-Language-Hearing Association (1995). The use of FC has not been completely eradicated, however, with research documenting that FC use with children with ASD may be as high as 9.8% in some areas. A resurgence believed to be a variation of FC has emerged in recent years, gaining popularity under the guise of rapid prompting methods (Mostert 2001, 2010; Travers et al. 2014). Other popular pseudoscientific ASD treatments include, but are not limited to, sensory integration interventions (e.g., pressure and weighted vests, brushing techniques, vestibular swinging; Barton et al. 2015), learning styles (Pashler et al. 2008), auditory integration training (Simpson 2005), and DIR/Floortime (National Autism Center 2015).

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Table 1 Contrasted characteristics of science and pseudoscience

Science	Pseudoscience
Evidence obtained via experimentation informs beliefs; belief in a claim is withheld if evidence is not available; relies on the entire body of evidence	Beliefs are formed first, and evidence is sought to support; relies on credulity; disconfirming evidence is rejected to preserve belief
Makes conservative and tentative claims based on evidence; beliefs change with new evidence; open-minded	Makes sensational claims without evidence; rejects new evidence against belief; clog-minded
Uses precise terminology to aid understanding and independent verification; rejects unverifiable claims	Uses vague language and jargon to avoid criticism and inhibit verification; accepts unverified claims
Knows, understands, and applies the rules of logic with body of evidence to make claims	Uses logical fallacies and cherry-picks evidence to make claims
Treats critics as colleagues and values criticism from a community of scientists; engages in honest debate	Does not value criticism and condemns dissent; works in isolation and dishonestly engages in debate

Travers (2017) Published with permission

Current Knowledge

Pseudoscience is neither a new phenomenon nor unique to ASD. There are, however, factors unique to ASD that may make families and practitioners especially vulnerable to accepting and adopting pseudoscientific practices (Metz et al. 2016). One factor that makes ASD susceptible to attracting pseudoscientific treatments is the absence of a definitive or singular cause of ASD. ASD is identified based on the presence – or absence – of behavioral features, specifically deficits in social communication combined with the presence of restricted, repetitive behaviors and stereotyped interests (American Psychiatric Association 2013). There is no single assessment that clinicians use to diagnose ASD; rather decisions are based on information accumulated through a number of sources including parent interviews, developmental history, standardized assessments of cognitive, communication, adaptive, and social skills, and direct observation. Another factor that increases susceptibility to pseudoscientific information relates to the

heterogeneous presentation of ASD characteristics. As an example, deficits in social communication range from a complete absence of spoken ability to fluent speech (Tager-Flusberg et al. 2005). Some individuals with ASD demonstrate a lack of interest, awareness, and even avoidance of social interactions, while others are highly motivated by and seek social interaction but do so in socially inappropriate or awkward ways (Wing 1988). This heterogeneity necessitates that treatment or instruction be highly individualized based on the individuals' skills and areas of need. The educational programming for one individual with ASD will differ from his peer with ASD. There is, therefore, no single approach to treating ASD.

The lack of a definitive, universal answer regarding the cause, assessment technique, and treatment of ASD leads to perceived evidence gaps which are being filled with misinformation and ineffective interventions (Smith and MacDonald 2017). Pseudoscientific beliefs are commonly accepted because they play into the innate human desire to create order, and meaning, from information and experiences encountered. Individuals will attribute goals to events and perceive goal directedness in random noise, spot patterns and correlations in the environment, and overdetect meaning in vague or meaningless statements (Boudry et al. 2015). This, coupled with a weak understanding of the nature of science, aids in the spread of pseudoscientific practices. Many pseudoscientific practices gain popularity because consumers erroneously believe that they are backed by scientific evidence (Losh and Nzekwe 2011). In the case of facilitated communication, one study reported that 100% of undergraduate and 83% of graduate students studying speech pathology believed that FC was supported by scientific research (Ganz et al. 2018).

Role of media. Public awareness about ASD has increased over the past decade in large part due to campaign efforts such as the Center for Disease Control's Learn the Signs Campaign, Autism Speaks, and First Signs (<http://firstsigns.org/>) organizations. Media has supported these efforts by drawing attention to various fundraisers

such as the annual "Night of Too Many Stars," hosted by HBO (HBO, 2008 to present) and document series such as a ten-part series on autism featured on the Today Show in February 2005 (The Today Show 2005). Public search data on the Google search engine peak yearly in April during autism awareness month (DeVilbiss and Lee 2014), an indication that public interest and investigation of the condition coincide with the active push for awareness.

Mass media allows for rapid dissemination to a broad consumer audience. It plays an integral role in improving public awareness of ASD, facilitating early screening, and improving general awareness and advocacy for individuals with ASD while also having a notable influence on the spread of pseudoscientific beliefs and practices. Consumers' openness to media and their media preferences significantly relate with their acceptance of pseudoscientific beliefs (Losh and Nzekwe 2011). Pseudoscientific ASD practices are disproportionately represented in mass media publications. More than 75% of treatments mentioned in print media lack scientific support, while stories on the scientifically validated approach, applied behavior analysis, steadily decreased over the past decade. Many of the stories disseminated through media platforms feature anecdotal or personal stories that elicit an emotional response from readers. Accordingly, articles focused on pseudoscientific practices elicited greater positive comments compared to those presenting on evidence-based practices (Schreck et al. 2013). Consumers who rely upon mass media as their primary source of information are likely to encounter repeated messaging from a restricted range of perspectives.

Consumers' perceptions of ASD treatments.

A growing body of literature has focused on understanding knowledge that results from reading printed texts about autism and its treatments. Fleury and colleagues conducted a series of survey studies to evaluate perceptions of ASD treatments based on evidentiary status among different constituent groups, specifically members of the general public (Fleury et al. 2019), parents of children with ASD (Fleury & Chaxiong, under review), and special education teachers (Fleury

& Kemper, under review). In each study, participants were instructed to read four descriptions of pseudoscientific ASD treatments (i.e., chelation, pressure/weighted vests, brushing techniques, gluten-free casein-free diets) and evidence-based practices (i.e., Picture Exchange Communication System, discrete trial training, self-management, social narratives). The credibility of the source providing information about the treatments varied in the text (high vs. low credibility). The researchers created two versions of the survey to experimentally manipulate the credibility of the source providing information of the practice. For example, survey 1 consisted of a professor (high credibility) providing information about discrete trial training. The same information about discrete trial training was presented in survey 2 but from a website blog (low credibility). After reading each description, participants answered questions regarding their familiarity with the practice, the extent to which they accepted or approved of the practice, and the likelihood they would use or recommend the practice.

The extent to which participants were familiar with ASD practices differed across groups. The general public and parents of children with ASD were more familiar with pseudoscientific treatments compared to evidence-based ASD practices. The inverse was found for the special education teacher population. Special education teachers were more familiar with evidence-based practices compared to pseudoscientific treatments. After reading descriptions of different treatment options, however, participants representing all constituent groups provided higher approval ratings of evidence-based practices compared to pseudoscientific treatments. The participants did not differentiate between high-credibility and low-credibility sources. That is, information about a treatment given by a low-credibility source was not perceived differently from that provided by a high-credibility source. In a subsequent study, Fleury et al. (2019) examined the influence of direct instructions that made the credibility of the source that provided the information more salient. Specifically, the researchers asked participants to pay attention to source credibility as they read the texts. The

results showed that while the high-credibility condition continued to be effective, the low-credibility condition resulted in lower scores. These findings suggest that readers will attend to the credibility of the source when instructed, but do not naturally engage in sourcing under normal reading conditions.

Future Directions

Information posted on the Internet is not regulated for scientific accuracy. While mainstream media outlets may preserve their accountability via corrections and/or retractions, no such mechanisms for correcting the spread of misinformation exist within social media (Eckberg et al. 2018). This means that consumers can readily access information that reflects and reinforces their own interests regardless of empirical soundness (Losh and Nzekwe 2011). Confirmation bias may then lead individuals on social media to seek out and accept information that supports their personal beliefs and are likely to retain faulty beliefs even in the face of conflicting evidence. Individual stories are able to go viral with the help of social media (regardless of source credibility), which has led to the rapid spread of misinformation in the modern world. Compounding this issue is that audiences that are likely to use social media, students from middle school through college, lack the necessary skills for evaluating the accuracy of online claims (Eckberg et al. 2018). The following are specific areas that professionals can use to support consumers in thinking critically about ASD treatment information they encounter through popular media.

Attend to source credibility. Source credibility – how believable a source of information is perceived to be – plays a particularly important role when individuals are confronted with either new or discrepant information (Braasch et al. 2012). Being able to distinguish between credible and noncredible sources has important implications for individuals' comprehension of the information they encounter, especially when faced with making decisions about which texts to read in order to learn about a new subject (Strømsø

et al. 2010). Evaluating source credibility, also known as “sourcing,” is not a natural process for readers, particularly novices (Britt and Aglinksi 2002; Goldman et al. 2012; Strømsø et al. 2010). Evidence suggests that readers do not routinely attend to the credibility of characters in narratives unless they are being instructed to do so (Van Boekel et al. 2017); however, explicitly instructing consumers to attend to the source providing the information does influence their evaluation of treatments in the intended direction with information from noncredible sources being viewed less favorably than treatment descriptions from credible sources (Fleury et al. 2019). Consumers should verify the effectiveness of the promoted treatment in question with credible sources including current special education textbooks, special education technical assistance centers, and reputable nonprofit organizations such as the National Professional Development Center on Autism Spectrum Disorder, National Standards Project, the Association for Science in Autism Treatment, and the Organization for Autism Research (Travers 2017). The website links for the nonprofit organizations can be found in the See Also section at the conclusion of this entry.

Address deficits in background knowledge.

Teachers are a particularly influential population in the spread of information as they are commonly a trusted source for families, particularly for children receiving special education services (Adams and Christenson 1998). The concept of EBPs is central to the provision of special education services. The teaching licensure structure varies across states with many states mandate specific endorsements or licenses specifically for teachers who educate students with ASD. These regulations have been enacted to better prepare educators to provide effective, evidence-based instruction for their students with ASD. The extent to which these preservice teacher preparation changes will have on student outcomes, however, is yet to be established.

Engage in the conversation. The general public is susceptible to pseudoscience, particularly those who use mass media as their primary source of information. Pseudoscientific beliefs and treatments are frequently disseminated through

different media outlets such as movies, news publications, and the Internet, while the evidence discrediting them is published by researchers in journals whose primary audience is other researchers. This leaves consumers without access to scientific evidence, which may leave them unaware that it even exists (Ganz et al. 2018). This points to a pressing need for researchers to disseminate their work to broader public audiences. Researchers should be encouraged and supported in publishing their work in accessible language in outlets commonly used by broader public audiences.

Optimize public awareness campaign efforts. Take public awareness campaigns to the next level by tailoring professional development programs to reach a broader audience. The 4C framework (Betsch et al. 2015) stresses that messaging content should differ depending on consumers’ level, and type, of hesitancy toward science. The model was originally developed to explain vaccination hesitancy but may have applications for attitudes toward pseudoscientific ASD treatments. Consumers are classified as belonging to different psychological profiles: complacency, convenience, confidence, and calculation. Messaging for changing attitudes differs across profiles. A figure in which the motivational profiles and outreach strategies can be found in Fig. 2. The content of messaging could contain a combination of strategies including the following: emphasize the importance of EBP for individuals with ASD, warn about the dangers of pseudoscientific practices, debunk misconceptions, make additional training available and accessible to different stakeholder audiences (i.e., general public, caregivers, educators), and incentivize the use of EBPs.

Conclusion. Public awareness campaign efforts and mass media platforms play a significant role in increasing public awareness and interest in ASD. The field has benefited in several ways from increased public awareness including, but not limited to, improved conversations between parents and health-care professionals leading to earlier detection, increased funding for autism research, and greater understanding of the needs of this population. The field has also experienced

Motivation Profile	Strategies							
	Raise perceived risk of unsupported treatment	Stress social benefit of EBPs	Stress that EBPs are the norm in professional communities	Strengthen positive attitudes of EBPs	Change structure to facilitate EBP uptake	Strengthen self-control and implementation	Debunk myths	Incentivize EBP use
Complacency Complacent people passively omit science, rather than actively decide against it. They perceive risks associates with pseudoscientific treatments to be low and demonstrate low levels of knowledge and awareness.	✓	✓	✓	✓				
Convenience Convenience people agree that science is important but other issues are of greater importance. Implementation of EBPs is blocked by structural barriers (i.e., difficult access, costs)				✓	✓	✓		
Confidence Trust in the effectiveness of pseudoscientific treatments, the system that delivers it, and motivation of influencers who decide on its necessity							✓	
Calculation Engage in extensive information search for pros and cons of selected treatment. No pre-conceived attitudes, but instead base decisions on utility maximization	✓	✓					✓	✓

Public Perception of Autism Treatments: Science Versus Pseudoscience in the Age of Mass Media, Fig. 2 Strategies for tailoring outreach efforts based on motivation profiles. Note: The table gives suggestions regarding which strategy can address potential determinants of pseudoscience adoption based on non-vaccination

work by Betsch et al. (2015). This information is presented for theoretical purposes. It has not been tested whether these interventions are especially suitable for the suggested types of pseudoscience adopters. Future research should address this question

growth in the quality and quantity of ASD research, which has yielded a body of EBPs that have ample scientific evidence to support their effectiveness in improving outcomes for individuals with ASD. Despite the existence of EBPs, autism remains a “fad magnet” attracting many consumers to entertain pseudoscientific treatments that either lack ample empirical evidence or have empirical evidence that document a lack of effectiveness. Pseudoscientific beliefs thrive when individuals within a society are willing to both accept and transmit them. Much research has been dedicated to studying the factors that influence people’s acceptance of pseudoscientific beliefs; however, far fewer resources have been dedicated to understanding the willingness of individuals to transmit these beliefs to one another (Mercier et al. 2018). Solely focusing on EBPs is an insufficient tactic to counter the impact of pseudoscience (Vyse 2016).

Instead, consumers who adhere to an evidence-based model for intervention must also be prepared to detect and avoid using unproven and pseudoscientific strategies (Travers 2017). This is an area of much needed research, with important implications for the quality of services required to promote optimal outcomes for individuals with ASD.

References

Adams, K. S., & Christenson, S. L. (1998). Differences in parent and teacher trust levels: Implications for creating collaborative family-school relationships. *Special Services in the Schools*, 14(1–2), 1–22.

American Academy of Pediatrics. (1998). Auditory integration training and facilitated communication for autism. *Pediatrics*, 102, 431–433.

American Association on Mental Retardation. (1994). AAMR board approves policy on facilitated communication. *AAMR News & Notes*, 7(1), 1.

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental health disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- American Psychological Association. (1994). Resolution on facilitated communication by the American Psychological Association. Adopted in council, August 14, 1994. Los Angeles.
- American Speech-Language-Hearing Association. (1995). Position statement on facilitated communication. *ASHA*, 37, 22.
- Association for Behavior Analysis. (1995). Statement on facilitated communication. *ABA Newsletter*, 18(2).
- Barton, E. E., Reichow, B., Schnitz, A., Smith, I. C., & Sherlock, D. (2015). A systematic review of sensory-based treatments for children with disabilities. *Research in Developmental Disabilities*, 37, 64–80.
- Betsch, C., Böhm, R., & Chapman, G. B. (2015). Using Behavioral insights to increase vaccination policy effectiveness. *Policy Insights From the Behavioral and Brain Sciences*, 2, 61–73. <https://doi.org/10.1177/2372732215600716>.
- Boudry, M., Blancke, S., & Pigliucci, M. (2015). What makes weird beliefs thrive? The epidemiology of pseudoscience. *Philosophical Psychology*, 28, 1177–1198. <https://doi.org/10.1080/09515089.2014.971946>.
- Braasch, J. L. G., Rouet, J., Vibert, N., & Britt, A. (2012). Readers' use of source information in text comprehension. *Memory and Cognition*, 40, 450–465.
- Britt, A., & Aglinksi, C. (2002). Improving students' ability to identify and use source information. *Cognition and Instruction*, 20(4), 485–522.
- Council for Exceptional Children. (2015). *What every special educator should know: Professional ethics and standards*. Arlington: CEC. Retrieved from <https://www.cec.sped.org/Standards/Ethical-Principles-and-Practice-Standards>.
- DeVilbiss, E. A., & Lee, B. K. (2014). Brief report: Trends in U.S. national autism awareness from 2004–2014: The impact of national autism awareness month. *Journal of Autism and Developmental Disorders*, 44, 3271. <https://doi.org/10.1007/s10803-014-2160-4>.
- Eckberg, D. A., Densley, J., & Dexter, K. (2018). When legend becomes fact, tweet the legend: Information and misinformation in the age of social media. *Journal of Behavioral and Social Sciences*, 5, 148–156.
- Fleury, V. P., Trevors, G., & Kendou, P. (2019). Public perceptions of autism treatment: The role of credibility and evidence. *Journal of Autism and Developmental Disabilities*, 49(5), 1876–1886. <https://doi.org/10.1007/s10803-018-03868-z>.
- Ganz, J. B., Katsiyannis, A., & Morin, K. L. (2018). Facilitated communication: The resurgence of a disproven treatment for individuals with autism. *Intervention in School and Clinic*, 54, 52–56. <https://doi.org/10.1177/1053451217692564>.
- Gersten, R., Fuchs, L. S., Compton, D., Coyne, M., Greenwood, C., & Innocenti, M. S. (2005). Quality indicators for group experimental and quasi-experimental research in special education. *Exceptional Children*, 71(2), 149–164.
- Goldman, S., Braasch, J., Wiley, J., Graesser, A., & Brodowska, K. (2012). Comprehending and learning from internet sources: Processing patterns of better and poorer learners. *Reading Research Quarterly*, 47(4), 356–381.
- HBO Night of Too Many Stars. (2008–Ongoing). <http://www.cc.com/shows/night-of-too-many-stars>
- Horner, R., Carr, E., Halle, J., McGee, G., Odom, S., & Wolery, M. (2005). The use of single subject research to identify evidence-based practice in special education. *Exceptional Children*, 71, 165–180.
- Lilienfeld, S. O., Ammirati, R., & David, M. (2012). Distinguishing science from pseudoscience in school psychology: Science and scientific thinking as safeguards against human error. *Journal of School Psychology*, 50, 7–36. <https://doi.org/10.1016/j.jsp.2011.09.006>.
- Losh, S. C., & Nzekwe, B. (2011). The influence of education major: How diverse preservice teachers view pseudoscience topics. *Journal of Science Education and Technology*, 20, 579–591. <https://doi.org/10.1007/s10956-011-9297-0>.
- McIntyre, N., Mundy, P. C., Solomon, M., Hatt, N. V., Gwaltney, M., Jarrold, W., & Kim, K. (2013). Reading and oral communication in students with ASD. Paper presented at the International Meeting on Autism Research, San Sebastian.
- Mercier, H., Majima, Y., & Miton, H. (2018). Willingness to transmit and the spread of pseudoscientific beliefs. *Applied Cognitive Psychology*, 32, 499–505. <https://doi.org/10.1002/acp.3413>.
- Metz, B., Mulik, J. A., & Butter, E. M. (2016). Autism: A twenty-first century fad magnet. In R. M. Foxx & J. A. Mulick (Eds.), *Controversial therapies for autism and intellectual disabilities: Fad, fashion, and science in professional practice* (pp. 169–195). New York: Routledge.
- Mostert, M. P. (2001). Facilitated communication since 1995: A review of published studies. *Journal of Autism and Developmental Disorders*, 31, 287–313.
- Mostert, M. P. (2010). Facilitated communication and its legitimacy—twenty-first century developments. *Exceptionality*, 18, 21–41.
- Mugaloglu, E. Z. (2014). The problem of pseudoscience in science education and implications of constructivist pedagogy. *Science & Education*, 23, 829–842. <https://doi.org/10.1007/s11191-013-9670-x>.
- National Autism Center. (2015). *Findings and conclusions: National standards project, phase* (p. 2). National Autism Center: Randolph.
- Pashler, H., McDaniel, M., Rohrer, D., & Bjork, R. (2008). Learning styles concepts and evidence. *Psychological Science in the Public Interest*, 9, 105–119.
- Schreck, K. A., Russell, M., & Vargas, L. A. (2013). Autism treatments in print: Media's coverage of scientifically supported and alternative treatments.

- Behavioral Intervention*, 28, 299–321. <https://doi.org/10.1002/bin.1370>.
- Simpson, R. L. (2005). Evidence-based practices and students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 20, 140–149.
- Smith, I. M., & MacDonald, N. E. (2017). Countering evidence denial and the promotion of pseudoscience in autism spectrum disorder. *Autism Research*, 10, 1334–1337. <https://doi.org/10.1002/aur.1810>.
- Strømso, H., Bråten, I., & Britt, A. (2010). Reading multiple texts about climate change: The relationship between memory for sources and text comprehension. *Learning and Instruction*, 20, 192–204.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 335–364). Hoboken: Wiley.
- The Today Show. (2005). Autism – The hidden epidemic? <http://www.nbcnews.com/id/6844737/>
- Travers, J. C. (2017). Evaluating claims to avoid pseudo-scientific and unproven practices in special education. *Intervention in School and Clinic*, 52, 195–203. <https://doi.org/10.1177/1053451216659466>.
- Travers, J. C., Tincani, M., & Lang, R. (2014). Facilitated communication denies people with disabilities their voice. *Research and Practice in Severe Disabilities*, 39, 195–202.
- U.S. Department of Education, Institute of Education Sciences, National Center for Education Evaluation and Regional Assistance, What Works Clearinghouse *Standards Handbook*, version 4.0. https://ies.ed.gov/ncee/wwc/Docs/referenceresources/wwc_standards_handbook_v4.pdf
- Van Boekel, M., Lassonde, K., O'Brien, E. J., & Kendeou, P. (2017). Source credibility and the processing of refutation texts. *Memory & Cognition*, 45, 168–181.
- Vyse, S. (2016). Where to fads come from? In R. M. Foxx & J. A. Mulick (Eds.), *Controversial therapies for autism and intellectual disabilities: Fad, fashion, and science in professional practice* (pp. 3–16). New York: Routledge.
- Wing, L. (1988). The continuum of autistic characteristics. In *Diagnosis and assessment in autism* (pp. 91–110). Boston: Springer.
- Wong, C., Odom, S. L., Hume, K., Cox, A. W., Fettig, A., Kucharczyk, S., Brock, M. E., Plavick, J. B., Fleury, V. P., & Schultz, T. R. (2015). Evidence-based practice for children, youth, and young adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(7), 1951–1966. <https://doi.org/10.1007/s10803-014-2351-z>.
- and standards. Arlington: CEC. Retrieved from <https://www.cec.sped.org/Standards/Ethical-Principles-and-Practice-Standards>.
- Fleury, V. P., Trevors, G., & Kendou, P. (2019). Public perceptions of autism treatment: The role of credibility and evidence. *Journal of Autism and Developmental Disabilities*, 49(5), 1876–1886. <https://doi.org/10.1007/s10803-018-03868-z>.
- Foxx, R. M., & Mulick, J. A. (2016). *Controversial therapies for autism and intellectual disabilities*. New York: Routledge.
- National Autism Center. (2015). *Findings and conclusions: National standards project, phase (p. 2)*. National Autism Center: Randolph.
- National Professional Development Center on ASD. <https://autismpdc.fpg.unc.edu/evidence-based-practices>
- National Standards Project NSP conducted by the National Autism Center. <https://www.nationalautismcenter.org/national-standards-project/>
- Organization for Autism Research. <https://researchautism.org/>
- Shermer, M. (2002). *Why people believe weird things: Pseudoscience, superstition, and other confusions of our time*. New York: Macmillan.
- Smith, I. M., & MacDonald, N. E. (2017). Countering evidence denial and the promotion of pseudoscience in autism spectrum disorder. *Autism Research*, 10, 1334–1337. <https://doi.org/10.1002/aur.1810>.
- Travers, J. C. (2017). Evaluating claims to avoid pseudo-scientific and unproven practices in special education. *Intervention in School and Clinic*, 52, 195–203. <https://doi.org/10.1177/1053451216659466>.
- Wong, C., Odom, S. L., Hume, K., Cox, A. W., Fettig, A., Kucharczyk, S., Brock, M. E., Plavick, J. B., Fleury, V. P., & Schultz, T. R. (2015). Evidence-based practice for children, youth, and young adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(7), 1951–1966. <https://doi.org/10.1007/s10803-014-2351-z>.

See Also

- Association for Science in Autism Treatment. <https://asatonline.org/>
- Council for Exceptional Children. (2015). *What every special educator should know: Professional ethics*

Public Understanding of Autism

► Politics of Autism

Pull-Out Room

► Resource Room

Punishment

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Definition

Punishment is defined as a contingency that results in a decrease in the future rate of a response. The contingent stimuli or events used are referred to as punishers. Punishers fall within two categories, unconditioned or conditioned. *Unconditioned* punishers are stimuli that are effective in reducing behavior without having been previously paired with any other punisher. Examples of such would include electric shock or time-out from positive reinforcement. *Conditioned* punishers are stimuli that, in the past, have been paired with previously established punishers, gain their “punitive power” from such pairings, and, as a result, demonstrate a reductive effect on behavior they follow. Examples would include loss of money (i.e., fining) and reprimands.

There are two different types of punishment procedures, Type I and Type II. Positive punishment (Type I) is the presentation of a stimulus (presumed to be aversive or unpleasant) following a behavior that decreases its future probability of occurrence. For example, if a child bites other children, the parent could deliver a stern reprimand after each biting episode. If the child viewed the reprimand as unpleasant or aversive, then in the future, the child would bite less often. Negative punishment (Type II) is the removal of a stimulus contingent upon a target behavior that reduces its future frequency of occurrence. Examples of negative punishment techniques include time-out from positive reinforcement (loss of

access to reinforcement for certain amount of time), planned ignoring (loss of attention), and response cost (loss of earned reinforcement). For example, time-out from positive reinforcement could be used with the child biting his peers. In this case, the parent could place the child in a chair for a set time period (e.g., 3 min) and access to preferred items prevented. The child would learn that biting leads to loss of access to social and tangible rewards. Future occurrences of biting should decrease.

Historical Background

Punishment has been used over the past several decades as an intervention for behavior deemed inappropriate. It is a technique used in behavior modification based on operant behavior/conditioning principles developed by B.F. Skinner in the 1950s. Operant behavior is any behavior whose future frequency is influenced by its consequences; the fundamental description of the influences on operant behavior is known as the three-term contingency. The three-term contingency consists of the discriminative stimulus (antecedent or environmental cue), operant response (specific behavior), and the consequence (action following the response). For example, a student is sitting in class during school. The teacher asks a question (discriminative stimulus), and immediately the student yells out the answer without raising his hand (operant response). The teacher then verbally reprimands the student for shouting out the answer and not raising his hands (consequence). If the reprimand functions as a punisher, then in the future, the student is less likely to yell out without raising his hand. Over time, the student’s behavior (not raising hand and shouting the answer) would be influenced by its past history of consequences (being verbally reprimanded). It is important to recognize that all operant behavior is influenced by the consequences that immediately follow it. Punishment is a consequence that leads to a reduction in rates of behavior.

Historically, some of the earliest research in this area was conducted in laboratory settings using animals such as rats or pigeons as subjects. Skinner and other researchers used different forms of punishment to influence the occurrence of the animal's behavior. For example, researchers exposed rats to brief electrical shocks contingent upon lever pressing; lever pressing reduced in rate. The earliest studies examined various components of punishment such as the intensity, immediacy, and schedule. Numerous experiments have shown the effectiveness of punishment in decreasing behaviors. This basic research led to the discovery that human behavior was influenced via the same mechanisms.

These earliest studies provided the foundation for further research on punishment with different clinical populations. Beginning in the late 1950s and early 1960s, researchers began using punishment with individuals with autism and other developmental disorders. Many of these individuals displayed a variety of challenging behaviors, such as physical aggression, self-injurious behavior (hitting, kicking, biting self), noncompliance, property destruction, sexual assault, pica (ingesting nonedible items), and stereotypy (repetitive behaviors – rocking, hand flapping, grinding teeth, etc.). Practitioners in schools were responsible for managing these behaviors in order to ensure the safety of individuals, in addition to providing an adequate education. Initially, the goal in many of these situations was to control the individual's behavior and immediately reduce the occurrence of the problematic behavior. A quick reduction in the targeted behavior was often necessary and desired due to the potential for injury if left untreated. Research, in both laboratory and applied settings, showed that punishment provided an immediate suppression of challenging behavior. Providers used a variety of punishment techniques to achieve these reductions.

In the 1970s, ethical concerns about the use of punishment began to surface. Although the research clearly showed that punishment could terminate or reduce targeted behaviors, there often were abuses of its use. Reports of staff and other providers abusing individuals with

developmental disabilities were made public. One of the earliest scandals was in 1971 at a residential facility for persons with developmental disabilities. Individuals were subjected to punishments without consent or supervision. Many were placed in seclusion for long hours, deprived of food, physically restrained, and subjected to cruel and inhumane treatments. This scandal led to the creation of a Blue Ribbon Panel that advocated for more stringent rules regarding the treatment of individuals with developmental disabilities. The concerns about the unethical and illegal use of punishment led to legislation at both the state and federal levels to ensure that future abuses would not occur. This resulted in a marked decrease in the study of punishment and the types of punishers examined. Additionally, the number of studies using potentially harmful aversive stimuli (electric shock, restraint, etc.) decreased as well. There became a greater awareness and need to find safer, more acceptable, and effective interventions. With the passing of the Individual with Disabilities Education Act in 1990, the federal government provided guidelines that caregivers perform functional behavior assessments and rely on more positive practices. The technology of functional assessment became more widespread and focused on the cause of the challenging behavior. By identifying the maintaining cause, caregivers concentrated on interventions directed at the function and relied less on punishment. This approach differed considerably from the goal of previous research on punishment, which was to control the behavior, rather than study the reasons for the challenging behavior occurring. Punishment was still explored, but there were far more stringent ethical guidelines regarding its use.

Punishment has continued to be studied, although the focus of this research has changed. Many of the punishment studies in recent years have used relatively mild aversive stimuli that dramatically reduced the potential for physical or emotional harm. Oftentimes, these studies have paired mild aversive stimuli with reinforcement or some other form of positive strategies. Typically, researchers and practitioners have used punishment as a last resort, and more specifically,

punishment has, in fact, become the default technology that is only recommended to be used when all other interventions have failed. The use of punishment has been highly regulated in both federal and state legislation, with many agencies providing oversight of its use. The general template describes three or more levels of approved aversive stimuli that can be used with varying levels of consent. For example, procedures such as verbal reprimands, response cost, and planned ignoring tend to be much lower level (and thus, more acceptable requiring less levels of consent) of aversive stimuli when compared to over-correction, noxious stimuli (foul smells, water mist, shocks, etc.). Most often, the less aversive stimuli are used. Only in special cases when people are putting themselves or others in significant danger are the more aversive stimuli seriously considered.

Current Knowledge

Behavioral researchers have used a wide range of aversive stimuli as Type I punishment to decrease problem behaviors in students with autism. It is important to recognize that there are drastic differences in the qualitative properties of the aversive stimuli that have been used in the research. These range from relatively mild to severely aversive stimuli. Mildly aversive stimuli include reprimands and response blocking. The potential risk of either physical or psychological harm from these mildly aversive stimuli is considered minimal. In contrast, there is a much greater risk for harm to the individual when more severely aversive stimuli are used, such as physical or mechanical restraint, ammonia capsules, loud noises, air blasts, noxious tastes (lemon juice), facial mist, and contingent electric shocks. Studies have shown that both the mild and more severe aversive stimuli can function as successful punishers to reduce a wide range of challenging behavior.

Type II (negative) punishment involves the removal or loss of a stimulus contingent upon a response that leads to a reduction of that response. An example of negative punishment often used is

“planned ignoring.” The general assumption is that attention is a positive reinforcer for most individuals (however, best practice would require a confirmation of this hypothesis for each individual). Planned ignoring, then, is a punishment procedure in which attention is purposefully denied or removed contingent upon the targeted undesirable behavior. Planned ignoring could include the withholding of social reinforcers including attention, verbal interaction, and physical contact for a brief period of time. Another example of negative punishment is called “response cost.” This is a form of punishment where a specific amount of reinforcement previously earned by the individual is lost or removed contingent upon problematic behavior. This loss of reinforcement results in a decreased probability of the future frequency of the behavior. This procedure includes “fines” or loss of money, tokens, and time with preferred activities contingent upon the target behavior.

There is individual variation with regard to what functions as effective punishment. Some people will find particular stimuli or consequences punitive, while other people will not. Just as individuals vary in terms of what functions as positive reinforcement for them, so it is with punishment. Knowledge of what is and is not aversive or punitive to an individual is essential in determining how and what type of punishment is to be applied.

Once a consequence has been determined to be an effective punisher, there are a number of variables that must be taken into account in order to assure that the most effective and least restrictive procedure is being used. One factor that must be considered is the immediacy of the application of the punisher. The quicker (in time) the punishment follows the occurrence of the undesired behavior, the more effective the punisher will be in reducing future rates of that behavior. For example, a punisher applied within a few seconds of the occurrence of the target behavior should result in quicker and greater progress than delivering the punisher 1 min after the target behavior occurred. Another important factor that will influence the effectiveness of a punisher is its intensity or magnitude (i.e., how much of the punisher is applied). There is a potential danger in applying a

punisher at a low intensity. “Habituation” occurs when the punishing consequence loses its reductive effect due to its application at a low intensity. When using punishment, it is better to use a higher intensity of a punisher to assure that the stimulus will achieve the reductive effect. For example, a student misbehaves in the classroom and the teacher uses the punishment of sitting at desk being quiet with head down. A 5-s duration would be considered potentially less effective than 3 min. A third important factor to consider procedurally is the schedule with which the punisher is applied. Schedule refers to how often and when a punishment is to be used. The typical procedure is to implement the punishment contingent upon each occurrence of the targeted problem behavior. This “continuous” schedule of punishment is considered to provide the most suppressive effect. However, another option is for an “intermittent” schedule of punishment, in which the person applying the contingency does not punish each target response, but rather punishes on a fixed or random schedule (e.g., every third target response). A denser schedule is more effective in rapidly reducing a problem behavior; however, a variable schedule can be used should the continuous application of the consequence require too much time or resources to effectively implement. This schedule of intermittent punishment is not recommended as the effects may take longer to show, and it is not always true that a variable schedule is effective.

Another issue related to the implementation of a punishment procedure focuses on the simultaneous use of reinforcement strategies. A punishment procedure should always be implemented with a positive reinforcement procedure targeting the increase of incompatible or appropriate behavior that will replace the target problem. For example, if a student is hitting herself in the head to gain the attention of a teacher (assuming a level of severity that requires a punishment procedure), a treatment may be put into place that would punish this behavior while simultaneously teaching the student how to request attention appropriately and reinforcing this new behavior. In addition to the reinforcement for the replacement behavior, it is important that there

is an attempt to remove all reinforcement that is being received for the problem behavior that is being punished. Another and a more recent advance in the field is to conduct a punishment assessment to determine the effectiveness of the treatment prior to its application. This requires a clinician, usually in a controlled “test” environment, to expose an individual to a variety of potential punishers to see which has the most suppressive effect.

There are two components that are essential and required before attempting to implement any punishment procedure. First, there should be a definitive plan that describes all of the components of the punishment procedure. The plan should clearly define the behavior that is targeted for punishment, what the punisher is and its intensity, how often it is or is not to be applied, and a standard by which progress will be assessed, including conditions under which the procedure will be removed for health and safety. These details should be scripted clearly so that the training of staff is most efficient, and continued monitoring of the staff employing this procedure can be done most efficiently. Secondly, prior to the application of any punishment procedure, the service provider must gain parental consent. It is recommended that the parents be exposed to the punishment procedure so that they can experience what their child will be experiencing.

Finally, current best practice dictates that the implementer of a punishment procedure take regular data (i.e., per use, daily, etc.) on both the use of punishment and the rate of the undesired behavior both before (i.e., baseline or preintervention) and during the application of the punishment program. With the inclusion of frequent collection and regular review of the data, it is possible to assure clinicians will not only know if their treatment is effective, but, more importantly, they will know when it is not. This is important to discern, since it may be unethical to continue a punishment program if it is not reducing the targeted behavior. If the treatment team confirms, through data review, that the targeted undesired behavior is failing to reduce, the team will likely stop the punishment so that it is not being inappropriately applied.

Before implementing punishment procedures, one must be aware of its potential side effects. The first and most likely is a possible negative reaction, either emotionally or physically, from the individual being punished. In some cases, the use of a punisher could elicit reactions such as crying, avoiding the person who delivers the punishment, or aggressing toward that person. Other possible side effects are that the individual could begin to exhibit escape and avoidant behavior. This could be demonstrated by the individual refusing to do work, lying, cheating, and staying away from the person(s) who administer the punishment or the environments in which the punishment was delivered. An additional side effect of punishment is termed “behavioral contrast.” This means that a behavior punished and suppressed in one context could increase in rate in a different context in which that same behavior is *not* punished. For example, if a particular response is punished in school, but not at home, then there could be an increase in the unwanted response in the home. In addition to the possibility that there may be increases in the behavior under different conditions, there is also the chance that the use of punishment may in fact serve as a model for the punished child for how to deal with undesired behavior in the future. For example, a child on whom punishment was used could be more likely to use punishment in the future, as that was the type of discipline that was used with the child. Although this is not always true, it is possible that spanking a child could result in teaching that individual aggressive behavior, regardless of the intention of the original intervention, is tolerated and a way to deal with unwanted behavior. Finally, the act of punishing a behavior has the potential to reinforce the individual providing the punishment. Since punishment is often effective in obtaining an immediate effect of stopping the punished behavior, the person using the punishment will see an immediate effect, which could reinforce the use of this approach. If one loudly reprimands a child because she is spilling something and the spilling immediately stops, it is likely that punishment will be used again in the future. To prevent this from occurring, it is important that a strict schedule and set of rules be

determined on a case by case basis, carefully describing when, how long, and how often a punisher will be applied to assure that an individual is not overexposed or harmed by the unnecessary application of a punisher.

Future Directions

While previous research has shown that punishment has been successful in reducing problems behaviors, there remain several different areas that require further exploration. One relates to the magnitude of punishment. The magnitude of a punisher refers to size or intensity of the aversive stimulus used contingent upon behavior. For example, if parents were to place their child in a time-out chair for 10 min, then the magnitude of the punishment would be 10 min. These results could be compared to the effects that 15 min have on the rate of the child’s behavior. This research would be significant to the applied field because it would provide practitioners the ability to identify and use the least intense or smallest magnitude of aversive stimuli that is effective in reducing the behavior. With this information, practitioners could implement an aversive stimulus that was just “strong enough” to decrease the behavior. This would help minimize the risk to individuals in that only the necessary magnitude of the stimulus that was needed to reduce behavior would be used.

The schedule of punishment is another area that needs further study. Research in this area has shown that punishment is most effective when it is implemented after every occurrence of the targeted behavior. Oftentimes, implementing the punisher after every occurrence can become cumbersome and difficult for practitioners. The goal of most interventions including reinforcement and punishment is to ultimately fade or gradually remove the intervention. A select number of studies in the late 1970s examined this topic and showed that an intermittent schedule of punishment may be sufficient in some circumstances to reduce problem behavior to a socially acceptable level. However, further research is needed to provide a better understanding of the conditions

under which an intermittent schedule could be effective. This knowledge would ultimately help practitioners completely remove the intervention over time.

The identification of stimuli that will function as an effective punisher is another area in need of investigation. There are only a few studies in the literature that describe procedures to systematically identify stimuli that would potentially serve as effective punishers. These punisher assessments typically involve presenting various potentially aversive stimuli and measuring levels of compliance, resistance, and negative affect. The development of these assessments may give practitioners a way to determine the most effective punisher for an individual, and allow for quicker identification of possibly aversive stimuli that could then be implemented, should the need arise.

A related area of research would be to investigate if more mild and socially acceptable stimuli could function as effective punishers. Punishment does not necessarily need to involve physically painful or noxious stimuli. Research could be conducted to identify if daily activities that an individual found unpleasant (or not preferred), but not physically uncomfortable, could serve as a punisher. For example, if a child did not enjoy coloring and found it aversive, then would coloring serve as an effective punisher when implemented contingent upon a targeted behavior? If these activities were effective, then this may allow practitioners to use more socially acceptable and presumably safer stimuli as punishers should caregivers need to resort to procedures more intrusive than reinforcement.

Punishment is a complex process. The majority of research regarding punishment was conducted many years ago in laboratory settings. Such research allowed for the tight control of numerous variables that is nearly impossible to achieve in applied real-life situations. Clinicians have recognized a need to examine the practical applications of punishment in field settings and with populations such as persons with autism. It is important to determine how effective punishment techniques can be used in educational settings where environmental variables may not be as clear or under as much control. Although

legislation does afford needed protection of persons with whom punishment may be used, the abolishment of punishment may not be the answer for safe-guarding individuals receiving behavior support. One key solution to preventing abuse through improper punishment is to understand the conditions and guidelines under which punishment has been proven both safe and effective. These standards have been researched and are well understood, and provide a guide by which punishment can be an appropriate and useful treatment option for those in need of behavior modification.

Punishment should be studied further and its components understood to allow for a richer knowledge of the factors that influence its effectiveness, and the conditions under which the clinical use of punishment is warranted. With this information, clinicians and providers will have the knowledge to use these interventions most effectively. An increased understanding will help assure that individuals with autism receive the safest and most effective treatment possible. It is important that researchers continue to explore the use of punishment in an effort to provide practitioners with the most effective interventions for their clinical use.

See Also

- [Behavior Modification](#)
- [Challenging Behavior](#)
- [Consequence-Based Interventions](#)
- [Informed Consent](#)
- [Overcorrection](#)
- [Response Cost](#)
- [Response Interruption/Redirection](#)
- [Time-Out](#)

References and Reading

- Anderson, J. (2011). Abatement of intractable vocal stereotypy using an overcorrection procedure. *Behavioral Interventions*, 26(2), 134–146.
- Athens, E. S., Vollmer, T. R., & Sloman, K. N. (2008). An analysis of vocal stereotypy and therapist fading. *Journal of Applied Behavior Analysis*, 41, 291–297.

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Ernst, M. (2000). Commentary: Considerations on the characterization and treatment of self-injurious behavior. *Journal of Autism and Developmental Disorders*, 30(5), 447–450.
- Foxx, R. M. (2005). Severe aggressive and self-destructive behavior: The myth of the nonaversive treatment of severe behavior. In J. Jacobson, R. M. Foxx, & J. Mulick (Eds.), *Controversial therapies for developmental disabilities: Fads, fashion, and science in professional practice* (pp. 295–310). Mahwah: Lawrence Erlbaum.
- Foxx, R. M., & Garito, J. (2007). The long term successful treatment of the very severe behaviors of a preadolescent with autism. *Behavioral Interventions*, 22, 69–82.
- Lerman, D. C., & Vorndran, C. M. (2002). On the status of knowledge for using punishment: Implications for treating behavior disorders. *Journal of Applied Behavior Analysis*, 35(4), 431–464.
- Mayer, G. R., Sulzer-Azaroff, B., & Wallace, M. (2012). *Behavior analysis for lasting change* (2nd ed.). Cornwall-on-Hudson: Sloan Publishing.
- Moore, T. R., & Symons, F. J. (2009). Adherence to behavioral and medical treatment recommendations by parents of children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 1173–1184.
- Schilling, D. L., & Schwartz, I. S. (2004). Alternating seating for young children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 34, 423–432.

Purdue Pegboard

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Description

The Purdue Pegboard test measures two types of dexterity: one involving finger dexterity and the other involving the movement of arms, hands, and fingers. The test itself is small, can be administered quickly (about 10 min including all necessary instructions and modeling), and is relatively simple to administer and perform, making it a useful instrument for a wide variety of populations and clinical and industrial settings.

The test consists of a pegboard with 2 rows of 24 holes and 4 cups at the top of the board for storage of the pegs. The test is comprised of four elements: dominant hand, nondominant hand, both hands, and assembly. For the dominant hand portion, the subject inserts small pegs into holes on the board using only the dominant hand. The examiner instructs the subject to only pick up one peg at a time. The examiner then times the subject for 30 s and counts the number of inserted pegs. For the nondominant hand aspect, the procedure is the same, but the subject is asked to only use the nondominant hand. Regarding the both hands element, the subject places two pegs simultaneously, one with each hand, in two rows on the pegboard. The subject is told to only pick up one peg at a time with each hand. The examiner times the subject and counts the number of pairs of pegs inserted after 30 s. For assembly, using both hands, the subject assembles sequences of peg, collars, and washers. The examiner instructs the subject to utilize an alternating approach, placing the peg in the hole with the dominant hand while using the nondominant hand to pick up the collar and then, while placing the collar, using the dominant hand again to place the washer. The examiner times the subject for 60 s and records the number of parts placed.

After all three conditions are completed, the examiner compares the raw score (number of pegs or assembly parts) for each element to the normative data in order to derive the subject's standardized scores.

Historical Background

The Purdue Pegboard test was first developed in 1948 by Joseph Tiffin, Ph.D., industrial psychologist, of Purdue University. He developed the test as a means of measuring manual dexterity for the purpose of selecting employees for industrial and assembly line jobs. In the seminal paper on the Purdue Pegboard test, Tiffin and Asher (1948) wrote that one of the most important aspects of the test was that it was standardized under conditions in which groups of employees could be tested together; one examiner using a

battery of 10 peg boards could test 50 people an hour, making it an efficient means of measuring the manual dexterity of potential assembly line and factory workers. The initial normative data for the Purdue Pegboard was obtained from thousands of students and employees working a variety of industrial jobs and divided into five groups: college men and women, veterans, and industrial men and women. Tiffin and Asher (1948) also reported on the reliability and validity of the instrument. The test-retest reliability was high. The validity was calculated against industrial job performance, and the validity coefficients varied indicating the importance of determining the validity of the Purdue Pegboard separately for each job its use was being considered.

Since its development in 1948, the Purdue Pegboard test has become one of the preeminent tests of dexterity in clinical, research, and vocational settings. It has been utilized to investigate the relationship between cognitive constructs and motor dexterity as well as to examine the relationship between motor dexterity and pathology. For example, the initial studies on the Purdue Pegboard indicated a significant relationship between intelligence as measured using the Wechsler Adult Intelligence Scale (WAIS) and manual dexterity in individuals with cognitive impairment (Phillips and Holden 1967). Additionally, the Purdue Pegboard test was able to discriminate between individuals with brain injuries and controls, indicating its potential use as a screen for brain injury (Vega 1969). Performance on the Purdue Pegboard test also discriminated between children with reading disabilities and typically developing children (Leslie et al. 1985). Lastly, performance on the Purdue Pegboard test was related to future factory workshop capability in individuals with moderate to severe levels of mental retardation (Presnall 1979). More recently, the Purdue Pegboard test has been used as a measure of motor functioning in a variety of clinical groups, including autism, Tourette's syndrome, schizophrenia, and Parkinson's disorder. Refer to the Clinical Uses section for further information.

Psychometric Data

Normative Data

Tiffin and Asher (1948) collected the initial normative data on college students, veterans, and factory workers. Additional normative data on a variety of populations has also been collected. Gardner and Broman (1979) collected normative data on over 1,300 school-age children. Hamm and Curtis (1980) also reported normative data on 340 adolescent/adult candidates for vocational rehabilitation who were suspected of having a cognitive or physical disability. Wilson et al. (1982) presented normative data on over 20 preschool-age children (2–5 years).

Reliability

The reliability of the Purdue Pegboard test has been widely studied and is generally considered to be adequate to strong. Test-retest reliabilities from the first edition of the Purdue Pegboard manual ranged from 0.60 to 0.71 for one trial and 0.82 to 0.91 for three trials. For the three-trial approach, each subject was administered the conditions three times, and the means of the scores were obtained (Tiffin 1968; Tiffin and Asher 1948). More recently, Buddenberg and Davis (2000) evaluated the test-retest reliability of the Purdue Pegboard with 1 week between administrations. For the one-trial administration, reliabilities ranged from poor to adequate (0.37–0.70) with the highest reliability for the combined score. The reliability was higher with a three-trial administration, with all of the reliability scores being excellent (>0.80).

Validity

Tiffin and Asher (1948) initially reported on the validity of the Purdue Pegboard test based on a compilation of several studies that compared the performance on the test with factory job performance. The validity coefficients ranged drastically from 0.07 to 0.76 depending on the type of job to which performance on the pegboard was compared, indicating that the validity of the Purdue Pegboard should be individually determined based on the job for which its use was being considered.

A study on the intercorrelation of the Purdue Pegboard tests (dominant hand, nondominant hand, both hands, and assembly) found that the peg placement elements were more strongly correlated with each other than with the assembly element. These results led the authors to conclude that the Purdue Pegboard measured two factors: finger dexterity or the “ability to make rapid, skillful, controlled manipulative movements of small objects, where the fingers were primarily involved” and manual dexterity as defined as the “ability to make skillful, controlled arm-hand manipulations of larger objects” (Fleshman and Ellison 1962).

The Purdue Pegboard also has strong discriminative validity, as for example, when using criteria established by Costa et al. (1963), performance on the pegboard discriminated between patients with brain damage and controls with about 90% accuracy, and it was also possible to indicate the laterality or diffuseness of brain damage in about 70% of cases. Costa et al. (1963) criteria are published in the examiners manual.

The Purdue Pegboard also discriminated between typically developing children and children with a wide range of psychological/neurological conditions (Rapin et al. 1966). In this seminal validity study, almost all of the typically developing children achieved scores within the normal range, but just over 20% of the children with brain injury or intellectual disability achieved scores in the normal range. The Purdue Pegboard test was also reported to be sensitive to “non-motor brain damage, including the syndrome of clumsiness, hyperactivity, and visual motor dysfunction.”

Clinical Uses

The Purdue Pegboard was initially designed as an industrial psychological instrument for the purpose of assessing the manual dexterity of factory workers as a means of predicting productivity. Since its inception in 1948, it has been widely used in employment settings but also as a measure of motor functioning in clinical research.

The Purdue Pegboard has been used to measure motor functioning in a wide variety of clinical populations. For example, the Purdue Pegboard has been instrumental in demonstrating the subtle motor impairments associated with individuals with Tourette’s syndrome (TS) and tic disorders, and performance on the Purdue Pegboard has been used to predict outcome in individuals with TS (Bloch et al. 2006). Impaired motor skills in children with attention deficit hyperactivity disorder (ADHD) has also been demonstrated using the Purdue Pegboard test, and performance on the Purdue Pegboard is correlated with attention, more generally, in typically developing individuals (Strenger et al. 2002; Sukhodolsky et al. 2010). Furthermore, children with ASD often have poor motor skills, and this finding has been confirmed in studies using the Purdue Pegboard test (Mandelbaum et al. 2006). Lastly, the Purdue Pegboard test has been used extensively in schizophrenia research to assess motor functioning, as motor deficits are commonly seen in individuals with schizophrenia.

Clinical researchers also have widely used the Purdue Pegboard as a measure of motor functioning in treatment studies. Notably, Aman et al. (2008) used the Purdue Pegboard test as part of a battery to assess the cognitive effects of risperidone in children with ASD. Similarly, the Purdue Pegboard test has been used to measure the cognitive impact of antipsychotic medications in schizophrenia, and it has been utilized as an outcome measure to assess the efficacy of treatments for Parkinson’s disease.

Overall, the Purdue Pegboard was developed to assess dexterity in factory workers, but over its 60-year history, it has proved to be a valuable tool for assessing manual motor dexterity more broadly and has been utilized extensively in clinical research with a wide variety of clinical populations.

See Also

- [Fine Motor Development](#)
- [Motor Control](#)
- [Psychomotor](#)
- [Visual-Motor Function](#)

References and Reading

- Aman, M. G., Hollway, J. A., McDougle, C. J., Scahill, L., Tierney, E., McCracken, J. T., et al. (2008). Cognitive effects of risperidone in children with autism and irritable behavior. *Journal of Child and Adolescent Psychopharmacology*, 18, 227–236.
- Bloch, M. H., Sukhodolsky, D. G., Leckman, J. F., & Schultz, R. T. (2006). Fine-motor skill deficits in childhood predict adulthood tic severity and global psychosocial functioning in Tourette's syndrome. *Journal of Child Psychology and Psychiatry*, 47, 551–559.
- Buddenberg, L. A., & Davis, C. (2000). Test-retest reliability of the Purdue Pegboard test. *American Journal of Occupational Therapy*, 54, 555–558.
- Costa, L. D., Vaughan, H. G., Jr., Levita, E., & Farber, N. (1963). Purdue Pegboard as predictor of the presence and laterality of cerebral lesions. *Journal of Consulting Psychology*, 27, 133–137.
- Fleshman, E. A., & Ellison, G. D. (1962). A factor analysis of fine motor manipulation tests. *Journal of Applied Psychology*, 46, 96–105.
- Gardner, R. A., & Broman, M. (1979). The Purdue Pegboard: Normative data on 1334 school children. *Journal of Clinical Child Psychology*, 8, 156–162.
- Hamm, N. H., & Curtis, D. (1980). Normative data for the Purdue Pegboard on a sample of adult candidates for vocational rehabilitation. *Perceptual and Motor Skills*, 50, 309–310.
- Leslie, S. C., Davidson, R. J., & Batey, O. B. (1985). Purdue pegboard performance of disabled and normal readers: Unimanual versus bimanual differences. *Brain and Language*, 24, 359–369.
- Mandelbaum, D. E., Stevens, M., Rosenberg, E., Wiznitzer, M., Steinschneider, M., Filipek, P., et al. (2006). Sensorimotor performance in school-age children with autism, developmental language disorder, or low IQ. *Developmental Medicine and Child Neurology*, 48, 33–39.
- Phillips, B. K., & Holden, R. H. (1967). Relationship between fine motor manipulative ability and intelligence in a vocational rehabilitation setting. *Vocational Guidance Quarterly*, 15, 213–216.
- Presnall, D. M. (1979). The relationship of manual dexterity and skill acquisition factors to workshop productivity. *Education and Training of the Mentally Retarded*, 14, 11–17.
- Rapin, I., Tourke, L. M., & Costa, L. D. (1966). Evaluation of the Purdue Pegboard as a screening test for brain damage. *Developmental Medicine and Child Neurology*, 8, 45–54.
- Streng, H., Niederberger, U., & Seelhorst, U. (2002). Correlation between tests of attention and performance on grooved and Purdue pegboards in normal subjects. *Perceptual and Motor Skills*, 95, 507–514.
- Sukhodolsky, D. G., Landeros-Weisenberger, A., Scahill, L., Leckman, J., & Schultz, R. T. (2010). Neuropsychological functioning in children with Tourette syndrome with and without attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49, 1155–1164.
- Tiffin, J. (1968). *Purdue Pegboard: Examiner manual*. Chicago: Science Research Associates.
- Tiffin, J., & Asher, E. J. (1948). The Purdue Pegboard: Norms and studies of reliability and validity. *Journal of Applied Psychology*, 32, 234–247.
- Vega, A. (1969). Use of Purdue Pegboard and finger tapping performance as a rapid screening test for brain damage. *Journal of Clinical Psychology*, 25, 255–258.
- Wilson, B. C., Iacoviello, J. M., Wilson, J. J., & Risucci, D. (1982). Purdue Pegboard performance of normal preschool children. *Journal of Clinical Neuropsychology*, 4, 19–26.

Purines and Related Compounds

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Definition

Purines and pyrimidines are compositional units of DNA and RNA. They play important role not only for genetic information but also for many metabolic steps as formation of coenzyme and intermediates.

Disorders of purine metabolism such as Lesch-Nyhan syndrome are well known and described elsewhere, but defect of pyrimidine metabolism is known comparatively recently. The final product of purine metabolite in human is uric acid, which is easily detected and measureable, but there is no equivalent compound in pyrimidine metabolism. So, many patients with pyrimidine metabolism defect may be followed as unexplained diseases.

Deficiency of dihydropyrimidine dehydrogenase (DPD) which is the first enzyme of pyrimidine degradation pathway causes increase levels of uracil and thymine in blood and urine. The patients of this deficiency show very wide spectrum of clinical symptoms. Almost half of the patients have convulsion, mental and/or motor retardation and growth retardation, microcephaly,

and dysmorphism, and ocular abnormalities are seen in smaller part. Autism is also observed 18% of the patients with DPD deficiency. Etiology is unknown, but DPD deficiency is one of the related disorders of autism.

See Also

- [Intellectual Disability](#)
- [Neurochemistry](#)

References and Reading

Webster, D. N., Becroft, D. M. O., van Gennip, A. H., & van Kuilenburg, A. B. P. (2001). Hereditary orotic aciduria and other disorders of pyrimidine metabolism. In *The metabolic & molecular bases of inherited diseases* (pp. 2663–2702). New York: McGraw-Hill.

Purkinje Cells

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Definition

Purkinje cells are large neuronal nerve cells that occupy the middle layer of the cerebellar cortex (also known as the Purkinje layer). These cells were discovered in 1837 by Czech anatomist Jan Evangelista Purkinje who identified and described them. Structurally, Purkinje cells are characterized by a long axon and numerous and intricate dendritic spines branching from the cell soma. Purkinje cells regulate coordinated motor activity by releasing the neurotransmitter GABA (gamma-aminobutyric acid) projecting to deeper cerebellar nuclei and exerting inhibitory influences upon the receiving cells. Purkinje cells have a large deal of control over the refinement of motor activities as they have an essential part

in the cerebellum's role in receiving information from the cerebrum and planning coordinated activity in response. Damage to Purkinje cells can result in certain neurological diseases. The loss of Purkinje cells has been observed in children with autism.

References and Reading

- Arin, D. M., Bauman, M. L., & Kemper, T. L. (1991). The distribution of Purkinje cell loss in the cerebellum in autism. *Neurology*, *41*(Suppl. 1), 307.
- Bailey, A., Luthert, P., Dean, A., Harding, B., Janota, I., Montgomery, M., et al. (1998). A clinicopathological study of autism. *Brain*, *121*, 889–905.
- Bauman, M. L., & Kemper, T. L. (1994). Neuroanatomic observations of the brain in autism. In M. L. Bauman & T. L. Kemper (Eds.), *The neurobiology of autism* (pp. 119–145). Baltimore: Johns Hopkins University Press.
- Bauman, M. L., & Kemper, T. L. (2005). Neuroanatomic observations of the brain in autism: A review and future directions. *International Journal of Developmental Neuroscience*, *23*(2–3), 183–187.
- Gitvo, E. R., Freeman, B. J., Scheibel, A. B., Duong, T., Robinson, H., Guthrie, D., et al. (1986). Lower Purkinje cell counts in the cerebella of four autistic subjects: Initial findings of the UCLA-NSAC autopsy research report. *The American Journal of Psychiatry*, *143*, 862–866.

Putamen

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Structure

The putamen is a subcortical structure situated at the base of the forebrain between the lateral medullary lamina of the globus pallidus and the external capsule. The putamen, caudate nucleus, globus pallidus, and amygdala form the basal ganglia. The rostral portion of the putamen is continuous with the caudate nucleus, forming the dorsal striatum.

Function

The putamen and other structures of the basal ganglia play an important role in many motor and cognitive functions, such as initiating, regulating, and monitoring movements. The putamen also plays a role in learning and memory, specifically in the acquisition of stimulus–response associations over time.

Pathophysiology

Putamen dysfunction is thought to be associated with repetitive behaviors in individuals with ASD (Hollander et al. 2005; Estes et al. 2011). While repetitive behaviors are reportedly correlated with basal ganglia dysfunction, studies of putamen volume in ASD report no difference between typically-developing individuals and individuals with ASD (Hardan et al. 2003; Sears et al. 1999; Langen et al. 2007; Estes et al. 2011).

References and Reading

- Carpenter, B. M. (1979). *Human neuroanatomy* (7th ed.). Baltimore: Williams & Williams.
- Estes, A., Shaw, D. W. W., Sparks, B. F., Friedman, S., Giedd, J. N., et al. (2011). Basal ganglia morphometry and repetitive behavior in young children with autism spectrum disorder. *Autism Research*, 4, 212–220.
- Hardan, A. Y., Kilpatrick, M., Keshavan, M. S., & Minshew, N. J. (2003). Motor performance and anatomic magnetic resonance imaging (MRI) of the basal ganglia in Autism. *Journal of Child Neurology*, 18, 317–324.
- Hollander, E., Anagnostou, E., Chaplin, W., Esposito, K., Mehmet Haznedar, M., et al. (2005). Striatal volume on magnetic resonance imaging and repetitive behaviors in Autism. *Biological Psychiatry*, 58, 226–232.
- Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (1991). *Principles of neural science* (3rd ed.). Norwalk: Appleton & Lange.
- Langen, M., Durston, S., Staal, W. G., Palmen, S. J. M. C., & van Engeland, H. (2007). Caudate nucleus is enlarged in high-functioning medication-naïve subjects with Autism. *Biological Psychiatry*, 62, 262–266.
- Packard, M. G., & Knowlton, B. J. (2002). Learning and memory functions of the basal ganglia. *Annual Review of Neuroscience*, 25, 563–593.
- Sears, L. L., Vest, C., Mohamed, S., Bailey, J., Ranson, B. J., & Piven, J. (1999). An MRI study of the basal ganglia in Autism. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 23, 613–624.

PVL

- [Periventricular Leukomalacia](#)

PVSP

- [Prosody-Voice Screening Protocol](#)

Pyramidal System

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Definition

Pyramidal cells are neurons with a pyramid-shaped cell body, one large apical dendrite, and several smaller dendrites at their base. They are mainly located in the gray matter of the cerebral cortex, corticospinal tract, and the hippocampus. Their axons may have local collaterals, but they are also able to project outside their cortical region. The development of normal motor control is dependent upon the development of connections between the axons of the pyramidal cells in the corticospinal tract and the spinal cord.

See Also

- [Cerebral Cortex](#)

References and Reading

- Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (2000). *Principles of neural science* (4th ed., p. 324). New York: McGraw-Hill.

Qatar and Autism

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Historical Background

Similar to most countries within the region, Autism Spectrum Disorder (ASD) awareness and programming in Qatar have been changing rapidly throughout the past years. Despite ASD being recognized by medical professionals and cases are detected at an estimated similar rate to international trends, ASD was not documented as a separate disability category in Qatar's national statistics until 2012 (Ministry of Development Planning and Statistics 2013). Nonetheless, the country's leadership has shown special interest in ASD as there is a recognition of its growing prevalence and the need to provide services for the affected individuals and their families. Hence, on April 2, 2008, Qatar officially joined the United Nations in announcing April 2nd as the world's "Autism Awareness Day," an event which was proposed to the UN by Her Highness Sheikha Moza Bint Nasser (United Nations Meetings Coverage and Press Releases 2008). Yet, ASD and general special needs services were operational years before this event. In fact, the history of ASD services and policy developments in

Qatar are all linked back to initiatives launched by HH Sheikha Moza Bint Nasser.

The first unit designated to serve children, adults, and elderly with special needs was opened in 1982 at the country's main healthcare provider; Hamad Medical Corporation (HMC) – Rumailah Hospital. However, the buildings and organizational structure changed rapidly until 2012, when the Child Development Center was opened to provide services for children with different disabilities including ASD. Children with ASD (3–6 years) started receiving early intervention by the multidisciplinary team of professionals within the Autism Program; assessment, diagnosis, and early intervention.

In 1999, a significant development in Qatar for ASD service provision was the establishment of the Shafallah Center for Children with Disabilities (formerly known as the Shafallah Center for Children with Special Needs). It was established under the guidance of HH Sheikha Moza Bint Nasser to meet the increasing demands for services to children with special needs. The Shafallah Center is currently servicing a large number of individuals with ASD in the country. Most recently, in 2016, Qatar Foundation opened the "Renad Academy" to join in the venture of providing services to children with ASD and their families. Many private centers' programs have also been developed during these years to meet the increasing demands. These centers are all licensed by the Ministry of Public Health and the Ministry of Education.

Programs to assist in transitioning children with ASD into mainstream education were not functional until the Additional Educational Support Needs (AESN) policy was passed by the Ministry of Education (formerly known as the Supreme Education Council) in June of 2009 (Supreme Education Council 2009). To support and enhance the process of academic and social inclusion, the Ministry of Education also established the Evaluation Institute in 2009. This institute started offering individual student evaluations to serve the needs of diverse learners, including children with ASD who are attending mainstream schools. To further support this process of inclusion, Qatar Assistive Technology Center (known as “MADA”), a nonprofit organization was established in 2010 by the Ministry of Information and Communication Technology to provide assistive technology support to children, adults, and families of individuals with special needs.

“Best Buddies – Qatar chapter” was founded in 2007 under the umbrella of the Shafallah center. It is a nonprofit organization dedicated to enhancing the lives of individuals with intellectual disabilities by providing opportunities for one-to-one friendships and integrated employment. This organization is another example of the inclusion movement and change in societal attitudes and perspectives towards individuals with disabilities.

Legal Issues, Mandates for Service

In Qatar, Autism was not reported as a separate disability category until 2012, as it used to be reported within the intellectual disability classification (Ministry of Development Planning and Statistics 2013). In 2004, the first law that regulates the rights of persons with disabilities was issued (Law No. 2) (Qatar Legal Portal 2004). The 14 articles within this law reinforced the social view of disabilities and paved the way for the succeeding mandates in the years to follow. Rights of persons with disabilities are also included in the sustainable development plan, which is part of Qatar National Vision 2030 (General Secretariat for Development Planning 2008).

ASD in Qatar has affected families from all ethnic backgrounds and different socioeconomic status; it became a disability that has increasing awareness and concern in Qatar and the region. As mentioned previously, in 2007, Qatar joined the UN member states in marking April 2nd as the world’s “Autism Awareness Day.” This event came in conjunction with increased involvement of Qatari authorities to expand healthcare and social services provision to those affected with ASD. In 2007, the social security act provided monthly benefits to persons with disabilities, which included a monthly cash allowance for domestic help (Committee on the Rights of Persons with Disabilities 2015). In 2008, Qatar ratified the United Nations Convention on the Rights of Persons with Disabilities (UNCRPD) and adopted all the rights contained within this convention by April 2015 (Committee on the Rights of Persons with Disabilities 2015).

On the educational and health care level, the country continues to make more government-funded services available for both nationals (Qataris) and expatriates (other nationalities). This includes evaluation clinics to diagnose and assess children with special needs that come in line with the country’s decision on inclusion of children with disabilities into the public school system, through either full or partial integration.

In terms of ASD specifically, it has been recognized within the health, education, and labor sectors. Thus, individuals with ASD are protected by the local and international laws on the rights of individuals with special needs. Autism remains as a separate category in the mandates of the Ministry of Education, and the support system specified involves individualized planning tailored to each students’ abilities and areas of need. In line with this, a total of 63 of the country’s independent (government-funded) schools include a department for special education and inclusion services for students with ASD and have support staff who have the necessary qualifications to support the student’s learning on a daily basis. Moreover, it has created two new schools for children with autism (Al-Hedaya school)

approximately between ages 5 to 17 years. The purpose of Al-Hedaya school is to prepare as many students through early intervention to be ready for entry into mainstream educational setting; through providing intervention in a physical environment that is similar to a school. Nonetheless, as all individuals with ASD differ in response to early intervention and may not all eventually reach mainstream education system, Al-Hedaya school still fills another gap in early intervention for ASD in Qatar. Lastly, the establishment of Rou'a Assessment, Advice, and Support Center has provided schools with a specialized referral entity for detected cases, as well as for progress monitoring and educational program modifications as found suitable for each individual student with ASD.

At the postsecondary educational level, Qatar University established a center known as the "Inclusion and Special Needs Support Center," to ensure that the proper accommodations are provided based on each student's needs to succeed and fully participate in all aspects of university life.

In 2016, the World Innovation Summit for Health (WISH) issued a report on autism with recommendations for improvement of service provision internationally (Munir et al. 2016). In 2017, the National Autism Plan was launched in collaboration with the World Health Organization (WHO). The working group included stakeholders from around the country. This group was charged with creating a National Autism Plan based on six pillars: awareness; early recognition and screening; diagnosis and assessment; interventions; school services; and transition to adulthood. In 2017, the World Innovation Summit for Education (WISE) developed a report, which reviewed ASD services provision in Qatar and highlighted the areas of need. On June 9, 2019, Qatari stakeholders had a significant presence during a panel discussion at the United Nations Headquarters in New York (11–13 June); Qatar Biomedical Research Institute (QBRI), Shafallah Center, MADA, Hamad Bin Khalifa University Health and Human Technology Department, and Qatar Foundation

for Social Work. The delegation partook in panel discussions held on the sidelines of the 12th session of the Conference of the States Parties to the Convention on the Rights of Persons with Disabilities. The panel discussions were about the role of technology in assisting people with autism in education and the workplace to ensure nondiscrimination and facilitate their full and effective participation.

This brief review of the trends in Qatar's legal recognition and support service provision for ASD demonstrates a continuing strive to improve the lives of individuals affected with ASD.

Overview of Current Treatments and Centers

The diagnostic services usually involve a multidisciplinary team of professionals that includes a developmental pediatrician, child psychiatrist, psychologist, and other relevant specialists. The main diagnostic tools used are the DSM-5 criteria, ADOS-2, and ADI-R, in addition to the use of the main ASD screening tools such as the Red Flags Checklist (RFC) in well-baby clinics which is recommended by the Ministry of Public Health, M-CHAT, SCQ (which has been translated into Arabic language and validated by a research team in Qatar (Aldosari et al. 2019)), and others. ASD diagnostic services are available mainly at Rumailah Hospital, HMC's Behavioral Clinic, Shafallah Center for Children with Disabilities, and Sidra Medical & Research Center.

Intervention services which include:

- Child and family medical and social support services
- Multidisciplinary early intervention services
- Family training and counseling
- Mainstream education inclusion support
- Transition planning; independent living and employment services

These intervention services are offered at a public and private sector basis. The first government-funded and the country's largest center servicing more than 150 individuals with

ASD is the Shafallah Center for Children with Disabilities. Meanwhile, early intervention programs for ASD are mainly offered by Rumailah Hospital as well as some other private centers such as the Child Development Center which provide a comprehensive multidisciplinary early intervention programs that include; behavioral, speech, occupational, and physical therapy, and other relevant services. Recently, Qatar Foundation has joined this venture with the opening of Renad Academy. All of the intervention service centers foster a curriculum based on evidence-based practices. These practices include Applied Behavior Analysis, Pivotal Response Training, Verbal Behavior, Discrete Trial Training, Treatment and Education of Autistic and Communication related handicapped Children (TEACCH), etc.

The Shafallah Center for Children with Disabilities is the only center offering services from early intervention to employment and independent living training. The Ministry of Education also provides curriculums and programs consisting of 2-year preparation and then a 6-year vocational training. It also provides 4 years of vocational qualification training such as “carpentry, book binding, painting, agriculture, and poetry for boys; sewing, embroidery, and home economics for girls” (Ministry of Education 2007). All of the public and private programs offered are conducted by a multidisciplinary team of professionals that require the licensing of the Ministry of Public Health and the Ministry of Education.

The National Autism Plan (2017–2021) includes 44 recommendations to be implemented by 2021. It contains some short-term targets which should be applied within the first year of the plan. It aims at improving the quality of the offered services and continues the development of care, education, and community participation. These initiatives will further enhance the quality of the present services and align all services with Universal Best Practices for ASD intervention programming and education in Qatar. A taskforce created and monitored by the Ministry of Public Health has been delegated to follow up on the implementation of the National Autism Plan.

Overview of Research Directions

Despite minimal publications about ASD in Qatar, it has been increasing in the past few years. To date, ASD research included investigations about: the quality of life of caregivers (Kheir et al. 2012); social robotics for ASD (Qatar University 2016); clinical case reports (Al-Sarraj et al. 2014; Ahram et al. 2017); and an overview of Autism and Applied Behavior Analysis in the GCC (Kelly et al. 2016). Moreover, QBRI published the first paper about clinical characteristics of ASD in Qatar with factors including consanguinity, gender, socioeconomic status, co-morbidities, etc. The study was titled “ASD in Qatar: Profiles and Correlates of a Large Clinical Sample” which described the clinical characteristics of ASD for 171 cases enrolled in Shafallah Center (Alshaban et al. 2017). Most recently, the Autism research team at QBRI concluded a country-wide prevalence study which started in 2013. This research involved the collection of data about all diagnosed cases from 2013 to 2017, as well as screening more than 100 mainstream primary schools in Qatar (both public and private schools). The screening process included using the Arabic translated and validated Social Communication Questionnaire (SCQ) in detecting cases of ASD. Those who scored above the cut-off were invited for assessment to confirm or exclude the ASD diagnosis through using; clinical observation, DSM-5 criteria, and ADOS-2 diagnostic tools. The prevalence rate of ASD in Qatar was estimated at 1.14% (Alshaban et al. 2019), which is consistent with international research findings. Moreover, QBRI conducted a research project in collaboration with Cleveland Clinic (USA), in which the team created and validated the Arabic version of the English stimuli paradigm to be used in eye tracking device. Efforts are being made to incorporate it with the English algorithm software at Cleveland Clinic so that it could be used as one of the early objective screening and diagnostic tools for ASD. This is considered as an ambitious and pioneering project for Qatar and the region.

Furthermore, an Autism Genetic Research Program in Qatar started in 2007 through joining

the Autism Genome Project, an International Consortium for Autism Genetic research. The Shafallah Medical Genetics Center (now part of QBRI) took the lead with more than 160 families participating in that project. QBRI continues to lead the Autism genetic research program together with Sidra Medical & Research Center, in collaboration with other Qatari and International institutions. QBRI scientists will be undertaking another QNRF funded research project titled “Autism Spectrum Disorder; Epigenetics; Environmental exposure; DNA and chromatin; Gene Expression from Clinical samples.” Moreover, the research team also investigated the clinical characteristics of all ASD cases diagnosed through the prevalence project, examining the effect of parental age as a risk factor in ASD prevalence. One of the key findings was that consanguinity was not found to be a significant risk factor for ASD, nor to familial reoccurrence of ASD; nonetheless, it was found to predict the severity of ASD (Alshaban et al. 2019). More notably, the QBRI prevalence study yielded the first national ASD registry, which contains 110 demographic, medical, developmental, and educational variables for all cases diagnosed with ASD. This registry will serve the country’s future ASD research and service planning. Currently, QBRI Autism research team plans to conduct a study about the prevalence of grown-up individuals (13 years and above) with ASD in Qatar and to create an ASD registry for this age group, meanwhile another team from WISH will evaluate service availability and quality of life for those individuals.

Overview of Training

Professional training for individuals working with children with ASD in Qatar occurs at different levels; post-secondary training, ongoing professional training, and training workshops, conferences, and symposiums by both the public and private stakeholders. Regarding postsecondary training, Qatar University’s college of education offers three programs: Bachelor, Diploma, and Master programs in Special Education. The

programs have US accreditation, and the curriculum ensures that graduates leave with knowledge about the best universal practices in ASD intervention. These programs include mandatory supervised field training.

Ongoing training of teachers and specialists is offered by two entities: the Evaluation Institute (known as Ro’a center) and the National Center for Educator Development (NCED) at Qatar University. These institutions provide training workshops as well as ongoing support through field visits to the mainstream schools.

In 2014, QBRI organized ADOS-2 and ADI-R training course for healthcare professionals in association with BeginningwithA Autism Consultancy and Training; a total of 24 professionals from the Shafallah Center, Rumailah Hospital, QBRI, and Al Tamakun school were trained and licensed in administering the ADOS-2 and ADI-R diagnostic tools. In 2017, the same training was organized by QBRI and another 21 professionals from similar service providers were trained and licensed in the aforementioned diagnostic tools.

Lastly, training conferences, symposiums, and workshops take place throughout the year by different organizations and centers. These training events are offered either free of charge, or for a cost accompanied with Professional Development (PD) hours or Continuing Medical Education (CME) hours. The target audience is usually professionals, healthcare providers, and families. Some centers such as the Child Development Center (a private center offering early intervention services for children with ASD and other developmental delays) host an annual training symposium targeting professionals and families. Meanwhile, HMC, Sidra Medical & Research Center, QBRI, and other private centers have training workshops throughout the year, to raise awareness and knowledge about ASD and other related disabilities. For instance, in May 2017, QBRI organized its first Autism symposium which hosted professionals from the gulf countries and the United States, Cleveland Clinic, Oregon Health and Science University, Autism Speaks, etc. The second Autism symposium is currently being planned to be held in April 2020.

Social Policy and Current Controversies

ASD awareness is evidently higher within the Qatari society. With the government's support for outreach events, the impact is noticeable during each annual Autism Awareness Month of April, as more organizations take the initiative, come together to raise awareness, and collaborate to offer different activities for individuals with ASD and their families. Moreover, local nonprofit organizations such as Qatar Autism Families Association (QAFA) and the Qatar Autism Society are examples of social movements that serve as a unified voice of the families' demands and offer ongoing community support. Past and current debates about ASD continue regarding the need for the increase in the provision of diagnostic and intervention services. Nonetheless, the government's efforts continue to expand ASD services provision to accommodate the increasing number of diagnosed cases. Meanwhile, some of the private centers compete to provide quality services at a high cost that some families cannot afford. Debates also revolve around the need for more educators and other specialists to successfully include students with ASD and other disabilities into the mainstream schools. Some families seek to hire a private therapist, tutor, or instructional assistant (shadow teacher) to help in the provision of certain needed services. Support in transitioning an individual with ASD through the education to employment to independent living is a process which has been improving with the existing services offered by the adult training unit in Shafallah Center, Ro'a Center (Evaluation Institute), and the Inclusion and Special Needs Support Center at Qatar University. However, the center at Qatar University is only equipped with staff and resources to serve specific physical and learning disabilities, and ASD is not listed as one of the disabilities that the center can provide services for. Moreover, the adult training unit at Shafallah Center cannot cope with the needed transition services for all ASD cases within Qatar.

References and Reading

- Ahram, D. F., Al-Sarraj, Y., Taha, R. Z., Elhag, S. F., Al-Shaban, F. A., El-Shanti, H., & Kambouris, M. (2017). A chromosomal microdeletion of 15q in a female patient with epilepsy, ID, and autism spectrum disorder: A case report. *Clinical Case Reports*, 5(6), 1013–1017. <https://doi.org/10.1002/ccr3.945>.
- Aldosari, M., Fombonne, E., Aldhalaan, H., Ouda, M., Elhag, S., Alshammari, H., Ghazal, I., Alsaleh, A., Alqadoumi, T., Thomson, R., Al Khasawneh, M., Tolefat, M., & Alshaban, F. (2019). Validation of the Arabic version of the social communication questionnaire. *Autism*. <https://doi.org/10.1177/1362361318816065>
- Al-Sarraj, Y., Abul Al-Khair, H., Taha, R. Z., Khattab, N., El-sayed, Z. H., Elhusein, B., & El- Shanti, H. (2014). Distal trisomy 10q syndrome, report of a patient with duplicated q24.31 – Qter, autism spectrum disorder and unusual features. *Clinical Case Reports*, 2(5), 201–205.
- Alshaban, F., Aldosari, M., El Sayed, Z., Tolefat, M., El Hag, S., & Al Shammari, H. et al. (2017). Autism spectrum disorder in Qatar: Profiles and correlates of a large clinical sample. *Autism & Developmental Language Impairments*, 2. 239694151769921.
- Alshaban, F., Aldosari, M., Al-Shammari, H., El-Hag, S., Ghazal, I., Tolefat, M., Ali, M., Kamal, M., Abdel Aati, N., Abeidah, M., Saad, A. H., Dekair, L., Al Khasawneh, M., Ramsay, K., & Fombonne, E. (2019). Prevalence and correlates of autism spectrum disorder in Qatar: A national study. *Journal of Child Psychology and Psychiatry*. <https://doi.org/10.1111/jcpp.13066>.
- Committee on the Rights of Persons with Disabilities. (2015). *Committee on the rights of persons with disabilities considers initial report of Qatar*. Ohchr. org. Retrieved from: <http://www.ohchr.org/EN/NewsEvents/Pages/DisplayNews.aspx?NewsID=16353&LangID=E>
- General Secretariat for Development Planning. (2008). *Qatar National Vision 2030*. Retrieved from: <http://www.mdps.gov.qa/en/qnv1/Pages/default.aspx>
- Kelly, M., Alireza, I., Busch, H., Northrop, S., Al-Attrash, M., Ainsleigh, S., & Bhuptani, N. (2016). An overview of autism and applied behavior analysis in the Gulf Cooperation Council in the Middle East. *Review Journal of Autism and Developmental Disorders*, 3(2), 154–164. 4048901600731.
- Kheir, N., Ghoneim, O., Sandridge, A., Al-Ismael, M., Hayder, S., & Al-Rawi, F. (2012). Quality of life of caregivers of children with autism in Qatar. *Autism*, 16(3), 293–298. <https://doi.org/10.1177/1362361311433648>.
- Ministry of Development Planning and Statistics. (2013). Chapter IX: Disabilities. Retrieved from: http://www.mdps.gov.qa/en/statistics/Statistical%20Releases/Social/SpecialNeeds2013/9_Disabilities2013.pdf.
- Ministry of Education. (2007). Education Regarding Human Rights and Fundamental Freedoms in the

- State of Qatar. Human Rights Education in Asian Schools, Vol. 11, pp. 49–55. Doha. Retrieved from: <https://www.hurights.or.jp/pub/hreas/11/06HRE%20in%20Schools%20-%20Qatar.pdf>
- Munir, K. M., Lavelle, T. A., Helm, D. T., Thompson, D., Prestt, J., & Azeem, M. W. (2016). *Autism: A global framework for action: Report of the WISH autism forum*. Doha: World Innovation Summit for Health.
- Prime Minister Launches National Autism Plan. (2017). The Peninsula: Qatar's Daily Newspaper. Retrieved from: <https://thepeninsulaqatar.com/article/17/04/2017/Prime-Minister-Launches-National-Autism-Plan>.
- Qatar Legal Portal. (2004). *Law no. 2 of 2004 in respect of people with special needs*. AlMeezan: Qatar Legal Portal. Retrieved from: <http://www.almeezan.qa/LawArticles.aspx?LawTreeSectionID=2138&lawId=246&language=en>.
- Qatar University. (2016). Social Robots at Center of Autism Therapy Research. Retrieved from: <http://www.qu.edu.qa/newsroom/Engineering/Social-robots-at-center-of-autism-therapy-research>
- Supreme Education Council. (2009). *Additional educational support needs: Learning difficulties, disabilities, behavior support. A pack of policies, guidance documents, and support materials for schools*. Doha: The Education Institute. Retrieved from: http://www.sec.gov.qa/CS/Additional_Education/AE_English.pdf.
- United Nations Meetings Coverage and Press Releases. (2008). Press Conference by Permanent Mission of Qatar on First World Autism Awareness Day. [Un.org](http://www.un.org). Retrieved from http://www.un.org/press/en/2008/080320_Autism.doc.htm

Quack Science

► Pseudoscience

Qualitative Versus Quantitative Approaches

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Synonyms

Case study; Cause and effect; Correlational; Descriptive research; Ethnography;

Experimental research; Group research; Phenomenology; Pre-, during, and posttest design; Pre-post design; Pre-post-posttest design; Quasi-experimental; Single case; Single-subject design; Within-subject design

Definition

Qualitative approaches refer to research that is both descriptive and systematic. It allows the researcher to examine relationships between activities, situations, and materials (Fraenkel and Wallen 2006 in Gast 2010). It is a systematic approach to studying phenomenon within a particular context (Gast 2010). In qualitative research approaches, the researcher is a participant observer who goes from specific to more general by process of inductive analysis and does not focus on generalization of results. Qualitative methods focus on groups, in-depth interviews, and reviews; they are an inductive process used to formulate theory. Qualitative methods do not rely on statistics or numbers and are less generalizable than quantitative methods. Qualitative research approaches are subjective in nature. Brantliger et al. (2005) in Gast and Ledford (2014) noted, qualitative research is concerned “with understanding qualities or the essential nature within a phenomenon within a particular context (p. 195). “Qualitative research provides “detailed, in-depth descriptions of case phenomena under study” (Gast and Ledford 2014, p. 10).

Quantitative approaches to research are based on formal, objective, and systematic processes in which data are numerically quantified. Another hallmark of quantitative research is replicability. Quantitative approaches are objective, deductive, and based on numeric quantification and generalization of results. Quantitative methods are used to test prespecified concepts, constructs, and hypotheses that make up a theory. Most commonly used quantitative methods include group design research and single-subject (within subject/single case) research designs; the latter is theoretical and based on specific research questions rather than

hypotheses. Both group research designs and single-case designs are more generalizable. In group design, the generalization comes in larger numbers in the sample; in single-subject designs, generalization is accomplished through replication of findings. Quantitative research approaches are objective in nature and rely on replication either in group numbers or by independent researchers as is the case with single case research designs (Gast and Ledford 2014).

References and Reading

- Bailey, J. S., & Burch, M. R. (2002). *Research methods in applied behavior analysis*. Thousand Oaks: Sage.
- Barlow, D. H., & Hersen, M. (1984). *Single case experimental designs strategies for studying behavior change* (2nd ed.). New York: Pergamon Press.
- Brantlinger, E., Jimene, R., Klingner, J., Pugach, M., & Richardson, V. (2005). Qualitative studies in special education. *Exceptional Children*, 71, 195–207.
- Fraenkel, J. R., & Wallen, N. E. (2006). *How to design and evaluate research in education* (6th ed.). New York: McGraw-Hill.
- Gast, D. L. (2010). *Single subject research methodology in behavioral sciences*. New York: Routledge.
- Gast, D. L., & Ledford, J. R. (2014). *Single case methodology: Applications in special education and behavioral sciences* (2nd ed. pp. 10–16). New York: Routledge.
- Huck, S. W., & Cormier, W. H. (1996). *Reading statistical research* (2nd ed.). New York: Harper Collins College Division.
- Kennedy, C. (2005). *Single case designs for educational research* (1st ed.). New York: Pearson.
- <http://www.fortunecity.com/greenfield/grizzly/432/rra2.htm>

Quality of Life for Transition-Age Youth with ASD

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Definition

Quality of life is a multidimensional and complex concept addressing the health, well-being,

satisfaction, and comfort of individuals (Rapley 2003). In addition to being multidimensional, researchers have identified the importance of both subjective and objective indicators of quality of life. Objective features consist of those that can be measured through observation, such as employment, daily activity patterns, and maladaptive behavior. Subjective features exist in the private thoughts and perspectives of individuals and can be measured through either self-report or proxy report (Cummins 2005). The multidimensional nature of quality of life is difficult, as there is no agreement on which dimensions comprise the concept. For example, in a review of quality of life literature, Felce and Perry (1995) identified five core dimensions of the concept: physical well-being, material well-being, social well-being, emotional well-being, and development and activity. Schalock (2000) proposed a definition that includes eight core dimensions: emotional well-being, interpersonal relationships, material well-being, personal development, self-determination, social inclusion, and rights.

For transition-age youth with autism, quality of life is especially important to consider as many changes and challenges – physical, interpersonal, educational, mental – can emerge during this time period (Biggs and Carter 2016; Clark et al. 2015; Wei et al. 2015). While navigating these transitions can be difficult for anyone, young people with autism may experience even more elevated challenges. Thus, considerable attention should focus on the understanding and promoting strong quality of life for transition-age youth with autism. Young people with autism and their families need to be connected to the services and supports – both natural and formal – they need to experience a high quality of life.

Historical Background

During the 1970s, quality of life began to be widely studied across a number of diverse disciplines, and in the 1980s disability-related fields began to embrace the concept (Schalock 2000). However, during this early stage, researchers and service providers were embracing a concept that

was not well-understood or well-defined. Therefore, the later decades – throughout the 1990s, 2000s, and up until today – researchers have been attempting to address salient questions about the concept of quality of life for individuals with disabilities (Schalock 2000; Van Heijst and Geurts 2015). This includes research addressing the concept and quality of life specifically for different groups of individuals who share characteristics, such as transition-age youth with autism.

Current Knowledge

In the last decade, recent research has significantly contributed to understanding issues around the quality of life for individuals with disabilities. This section will summarize current knowledge specifically related to the quality of life of transition-age youth with autism in three areas: (a) measurement of the concept, (b) differences in quality of life between young people with and without autism, and (c) predictors of higher quality of life for this population.

First, the assessment of quality of life for young people with autism presents with a variety of challenges. The measures most frequently used in published papers to assess the quality of life of youth with autism include the PedsQL (Varni et al. 1999), Kidscreen (The KIDSCREEN Group Europe 2006), and Child Health Questionnaire (Landgraf et al. 1996). Each of these three measures uses Likert-type scales and has options for using self-report and proxy report. Although there are examples of the use of self-report to assess the subjective quality of life of youth with autism in the literature (e.g., Clark et al. 2015; Cottenceau et al. 2011; Kamp-Becker et al. 2010), these studies typically focus on high-functioning youth with average or above average measures of intelligence. There are additional challenges that arise when considering the use of self-report with young people with more complex support needs, including co-occurring intellectual disability and complex communication needs. Many have discussed how the difficulties with communication and cognition that can be associated with autism complicate being able to acquire reliable

self-report on subjective indicators of quality life (Biggs and Carter 2016; Clark et al. 2015). Thus, proxy-based responses are a popular alternative to self-report of quality of life, even though these reports have limitations. A majority of studies involving both self-report and proxy report for youth with autism suggest parents may rate quality of life lower than the youth themselves (Clark et al. 2015; Shipman et al. 2011; Van Heijst and Geurts 2015).

Second, findings from a number of studies indicate transition-age youth and young adults with autism have lower ratings of quality of life when compared with ratings of typically developing young people. In a meta-analysis of quality of life in individuals with autism across the life-span, van Heijst and Geurts (2015) found overall quality of life is lower for individuals with autism compared to individuals without autism. Furthermore, these differences were found consistently across the life-span. More specifically for transition-age youth, Kamp-Becker et al. (2010) found self-reported quality of life was lower for adolescents and young adults with high-functioning autism when compared with young people without a disability. Biggs and Carter (2016) found parent-proxy ratings of quality of life of their transition-age sons and daughters with autism (both with and without co-occurring intellectual disability) were significantly lower than normative ratings in the areas of social support, physical well-being, and psychological well-being. Collectively, these findings emphasize the importance of addressing aspects of quality of life as outcome measures for treatments and interventions for young people with autism. Moreover, across a number of studies focused on this population of young people, the greatest differences between ratings of quality of life when compared with typically developing peers are often found related to domains focused on social well-being and social support (e.g., Biggs and Carter 2016; Clark et al. 2015; Kamp-Becker et al. 2010). These findings further emphasize the importance social-related intervention and support strategies for this group of youth. Social-related intervention and support strategies for youth with autism can include communication interventions, social support and social skill

interventions, as well as high-quality inclusive learning opportunities (Biggs and Carter 2016).

Third, recent research has begun to understand the different factors that might predict or influence quality of life for transition-age youth with autism. Generally, however, what has dominated this area of research has been a focus on the contributions of demographic- and disability-related characteristics, such as age, gender, race/ethnicity, socioeconomic status, disability severity, adaptive behavior, and cognitive functioning (Schalock 2004). Each of these factors is related to fairly stable rather than malleable characteristics of youth, and the findings are mixed related to whether these different characteristics influence quality of life. For example, while some have found significant associations between quality of life and disability-related characteristics (e.g., support needs, functional abilities, or challenging behaviors), others have not (e.g., Biggs and Carter 2016; Kamp-Becker et al. 2010).

While there is limited and mixed knowledge about associations between these demographic- and disability-related characteristics with quality of life, even less is known about other, more malleable factors. Such factors that may be associated with quality of life – but also are more likely to be able to be positively influenced by intervention efforts – include young people's access to support-related resources (e.g., social resources and support, relationships, spiritual faith), their dispositions or personal characteristics (e.g., positive character strengths, self-determination), and their community involvement (e.g., participation in out-of-school activities). Research is only beginning to explore whether these types of factors might be associated with higher quality of life for young people with autism. For example, Biggs and Carter found greater involvement in out-of-school activities (e.g., athletics, volunteer experiences, community activities) and strength of religious faith were both associated with quality of life ratings within the domain related to social well-being. Furthermore, the character strengths of young people (e.g., courage, empathy, gratitude, humor, resilience) emerged as a strong predictor of higher quality

of life across all domains. Although other studies have linked factors like community involvement to various positive outcomes for young people with and without disabilities (e.g., Mahoney et al. 2006), few studies have directly explored whether these types of malleable factors have predictive relationships with quality of life.

Future Directions

A number of important areas related to quality of life for this population need further investigation, pointing the way for future research directions. First, a number of questions remain related to measurement of quality of life for transition-age youth with autism. Few efforts have focused on integrating subjective and objective indicators of quality of life into a single measurement tool, which may be well-suited to assessing the complex and multidimensional concept. The field also needs more information to better understand differences between using self-report and proxy report to assess subjective indicators of quality of life, and the influence of who does the reporting in proxy reports (e.g., mother versus father) has not yet been thoroughly investigated. Finally, additional research is needed to identify and develop ways to reliability and validly capture the views of youth with significant co-occurring cognitive and communication impairments through self-report on their own quality of life.

Second, current studies provide snapshots of quality of life of young people with autism at single points in time. While these cross-sectional investigations are important, future research is needed that also addresses longitudinally how quality of life for individuals with autism changes over time.

Third, more research is needed to further explore factors that may be associated with higher quality of life for young people with autism. Identifying the significant predictors of quality of life can be a critical step in developing effective interventions, supports, and services to help individuals with autism live a life of quality. This may be especially true if investigators are

successful in isolating the influences of more malleable factors in a young person's life, rather than focusing exclusively on rather fixed factors such as disability- and demographic-related characteristics. At the present time, few studies have explored these types of factors that may be targeted by intervention, such as community involvement, access to supportive relationships, involvement in faith communities, and positive characteristics such as humor, empathy, resilience, and self-determination.

Educators, therapists, and other service providers are charged with the task of providing services and supports that lead to quality outcomes for individuals with autism. Focusing on issues around quality of life can help these and other stakeholders support individuals with autism in living a life of quality throughout the life-span, including during this critical period of transition.

See Also

- [Adulthood, Transition To](#)
- [Friendships](#)
- [Inclusion](#)
- [Stigmatization](#)
- [Transition Planning](#)

References and Reading

- Biggs, E. E., & Carter, E. W. (2016). Quality of life for transition-age youth with autism or intellectual disability. *Journal of Autism and Developmental Disorders*, 46, 190–204.
- Clark, B. G., Magill-Evans, J. E., & Koning, C. J. (2015). Youth with autism spectrum disorders: Self- and proxy-reported quality of life and adaptive functioning. *Focus on Autism and Other Developmental Disabilities*, 30, 57–64.
- Cottenceau, H., Roux, S., Blanc, R., Lenoir, P., Bonnet-Brilhault, F., & Barthelemy, C. (2011). Quality of life of adolescents with autism spectrum disorders: Comparison to adolescents with diabetes. *European Child Adolescent Psychiatry*, 21, 289–296.
- Cummins, R. A. (2005). Moving from quality of life concept to a theory. *Journal of Intellectual Disability Research*, 49, 699–706.
- Felce, D., & Perry, J. (1995). Quality of life: Its definition and measurement. *Research in Developmental Disabilities*, 16, 51–74.
- Kamp-Becker, I., Schroder, J., Remschmidt, H., & Bachmann, C. J. (2010). Health-related quality of life in adolescents and young adults with high functioning autism-spectrum disorder. *GMS Psychosocial Medicine*, 7, 1–10.
- Landgraf, J. M., Abetz, L., & Ware, J. E. (1996). *Child health questionnaire (CHQ): A user's manual*. Boston, MA: The Health Institute, New England Medical Center.
- Mahoney, J. L., Harris, A. L., & Eccles, J. S. (2006). Organized activity participation, positive youth development, and the over-scheduling hypothesis. *Social Policy Report*, 20, 3–32.
- Rapley, M. (2003). *Quality of life research: A critical introduction*. London: SAGE.
- Schalock, R. L. (2000). Three decades of quality of life. *Focus on Autism and Other Developmental Disabilities*, 15, 116–127.
- Schalock, R. L. (2004). The concept of quality of life: What we know and do not know. *Journal of Intellectual Disability Research*, 48, 203–216.
- Shipman, D., Sheldrick, S., & Perrin, E. C. (2011). Quality of life of adolescents with autism spectrum disorders: Reliability and validity of self-reports. *Journal of Developmental and Behavioral Pediatrics*, 32, 85–89.
- The KIDSCREEN Group Europe. (2006). *The KIDSCREEN questionnaires: Quality of life questionnaires for children and adolescents handbook*. Lengerich: Pabst Science Publishers.
- Van Heijst, B., & Geurts, H. M. (2015). Quality of life in autism across the lifespan: A meta-analysis. *Autism*, 19, 158–167.
- Varni, J., Seid, M., & Rode, C. (1999). The PedsQL™: Measurement model for the pediatric quality of life inventory. *Medical Care*, 37, 126–139.
- Wei, X., Wagner, M., Hudson, L., Yu, J. W., & Shattuck, P. (2015). Transition to adulthood: Employment, education, and disengagement in individuals with autism spectrum disorders. *Emerging Adulthood*, 3, 37–45.

Quasiexperimental

- [Qualitative Versus Quantitative Approaches](#)

Quasi-words

- [Protowords](#)

Quetiapine

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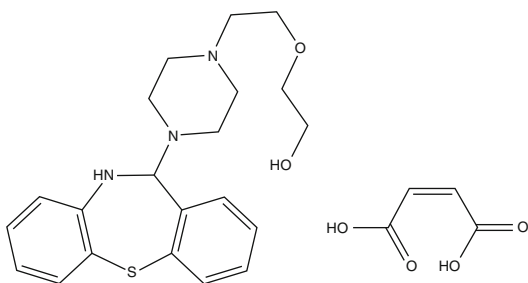
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Synonyms

Quetiapine fumarate; Seroquel; Seroquel XR

Definition



Quetiapine is a prescription drug in the group of atypical antipsychotics initially FDA-approved for medical use in the year 1997 whose active ingredient, quetiapine fumarate, has the chemical formula $C_{25}H_{31}N_3O_6S$. This drug, marketed by AstraZeneca, is FDA-approved for the treatment of global symptoms, positive symptoms, negative symptoms, cognition, and aggression in

schizophrenia, the treatment of bipolar disorder, and as an adjunctive treatment of major depressive disorder. Quetiapine has also been used in open-label trials in autism spectrum disorders (ASDs) targeting irritable behaviors marked by aggression, self-injury, and severe tantrums. Observed side effects include agitation, sedation, weight gain, aggression, and sialorrhea, and this drug has a low risk of anticholinergic effects, orthostasis, and increased liver enzyme levels and a very low risk of extrapyramidal symptoms, neuroleptic malignant syndrome, seizures, and hematologic effects.

See Also

- [Antipsychotics: Drugs](#)
- [Atypical Antipsychotics](#)

References and Reading

- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001). *Principles and practice of psychopharmacotherapy* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
- Printz, D. J., & Lieberman, J. A. (2006). Quetiapine. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 263–275). Washington, DC: American Psychiatric Publishing.
- Stahl, S. M. (2000). Antipsychotic agents. In *Essential psychopharmacology: Neuroscientific basis and clinical applications* (pp. 401–458). Cambridge, MA: Cambridge University Press.
- U.S. Food and Drug Administration. (2011). *Drugs@FDA*. Retrieved from: <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm>.

Quetiapine Fumarate

- [Quetiapine](#)

R

RAADS-R

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Synonyms

[Ritvo autism asperger diagnostic scale – revised](#)

Description

The RAADS-R is an 80-item scale specifically designed to assist clinicians diagnosing AD in adults 18 years and older. It contains a total of 80 questions. Sixty-four short questions identify symptomatic behaviors that are scored on a four-point Likert scale that takes into account developmental factors (true now and when I was young = 3, true only now = 2, true only when I was younger than 16 years old = 1, and never true = 0). It also contains 17 non-symptomatic questions that are scored in reverse order (true now and when I was young = 0, true only now = 1, true only when I was younger than 16 years old = 2, and never true = 3). These 16 questions are identified on the scale by an asterisk next to their question number.

Historical Background

The original version of the Ritvo Autism Asperger Diagnostic Scale (the RAADS) was developed to assist clinicians diagnosing autistic disorder in adults (18 years and older). It contained 78 items based on DSM-IV TR symptom criteria. After extensive field testing, the scale was modified by the elimination of three questions and the addition of five new questions. The revised 80-question scale is called the RAADS-R. A Swedish version of this scale has been published, and translations into other languages are being conducted Andersen et al. (2011).

Psychometric Data

The RAADS-R was validated in an international study conducted at nine medical centers on three continents (North America, Europe, and Australia). It involved 201 autistic disorder adults and 578 comparison cases (276 who never had a DSM-IV TR diagnosis and 302 who had a DSM-IV TR diagnosis other than autistic disorder). Results revealed the following: sensitivity = 97% (proportion of actual positives correctly identified), specificity = 100% (proportion of negatives correctly identified), concurrent validity = 96% when compared with other diagnostic instruments (ADOS Module 4, SRS), and test retest reliability $r = .987$.

Clinical Uses

To administer the RAADS-R, a diagnostician reads each question to the subject and marks the answer sheet. This insures that the questions are understood, the answers are properly coded, and the score can be quickly determined. Most importantly, however, it gives the diagnostician a chance to identify and discuss specific symptoms during administration. The average time to complete and score the RAADS-R is 30 min.

A total RAADS-R score of 64 or higher is consistent with the diagnosis of AD and supports the clinician's diagnosis. However, if there is difference between the clinician's diagnosis and the RAADS-R diagnostic assignment, the clinician's diagnosis should take precedence. This is because symptoms may be revealed only during an interview. Also, the RAADS-R standardization study reported that many AD subjects, particularly those in their late teens and early twenties, failed to acknowledge the presence of symptoms that their families said were present and which were readily observed by the diagnostician.

See Also

- ▶ [Autism Diagnostic Interview-Revised](#)
- ▶ [Autism Screening Instrument for Educational Planning \(ASIEP-2\)](#)
- ▶ [Behavior Observation Scale](#)
- ▶ [Behavior Rating Instrument for Autistic and Atypical Children \(BRIAC\)](#)
- ▶ [Behavior Rating Scale \(BRS\)](#)
- ▶ [Diagnosis and Classification](#)

References and Reading

- Andersen, L. M. J., Näswall, K., Manouilenko, I., Nylander, L., Edgar, J., Ritvo, R. A. et al. (2011). The Swedish version of the Ritvo autism and asperger diagnostic scale: revised (RAADS-R). A validation study of a rating scale for adults. *Journal of Autism Developmental Disorders*. 41(12):1635–1645.
- Ritvo, R. A., Ritvo, E. R., Guthrie, D., Ritvo, M. J., Hufnagel, D. H., McMahon, W. et al. (2011). The Ritvo autism asperger diagnostic scale-revised (RAADS-R). A scale to assist the diagnosis of autism

spectrum disorder in adults: An international validation study. *Journal of Autism Developmental Disorders*. 41(8):1076–1089 (Springer link, <http://www.springerlink.com/content/fhj14075h450547q/>)

Racing Thoughts, Distractibility, and Psychomotor Agitation

▶ Mania

Rank, Beata

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Names and Degrees

Beata Rank (Minzer): February 16, 1886–April 11, 1967

Major Appointments (Institution, Location, Dates)

- 1918–1926: Psychoanalytic training with Sigmund Freud in Vienna, Austria
- 1926–1936: College training in France
- 1936–1967: Child psychoanalyst in Boston Mass

Landmark Clinical, Scientific, and Professional Contributions

A child psychoanalyst who trained with Freud (among others), Beata Rank was an honorary Professor of Psychiatry at Boston University School of Medicine as well as a consultant/supervisor at the Judge Baker Guidance Center in Boston. Along with Marian C. Putnam, she both cofounded and codirected the James Jackson

Putnam Children's Center (1943). This Center was unusual in its emphasis on work with young (preschool) children and their parents. In the field of autism, her publication on unusual patterns of development (what she termed atypical development) was an early and highly original contribution prefiguring, in some ways, the notion of atypical PDD in DSM-III (1980).

Short Biography

A native of Poland, Beata Rank had an early interest in psychology and met her husband Otto, a psychoanalyst and student of Freud's, and wed him shortly before the conclusion of World War I. The couple moved to Vienna where they were very much part of the psychoanalytic circle centered around Sigmund Freud. She trained in psychoanalysis and worked on issues related to women's development and childhood. As her husband's relationship with Freud began to deteriorate, the couple moved to Paris for a time before Otto moved onto the USA. She continued her study of women's issue and eventually joined him in the USA. Within the Boston psychoanalytic community, she was accepted as a psychoanalyst and worked at Boston University as well as at the Judge Baker Child Guidance Center. At the Putnam Center, which she cofounded, she began a day treatment program for younger children and began to write about the emotional and cognitive problems of this age group. Her papers on atypical development (which prefigured, in some ways, the concept of PDD-NOS) were published during this time.

See Also

- [Pervasive Developmental Disorder Not Otherwise Specified](#)

References and Reading

American Psychiatric Association. (1980). *Diagnostic and statistical manual* (3rd ed.). Washington, DC: APA Press.

Rank, B. (1949). Adaptation of the psychoanalytic technique for the treatment of young children with atypical development. *The American Journal of Orthopsychiatry*, 19, 130–139.

Rank, B. (1955). Intensive study and treatment of preschool children who show marked personality deviations, or atypical development and their parents. In G. Caplan (Ed.), *Emotional problems of early childhood: Proceedings of the international institute of child psychiatry* (pp. 491–501). New York: Basic.

Raphe Nuclei

- [Midbrain Raphe](#)

Rapid Motor Imitation Approach (RMIA)

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Definition

The rapid motor imitation approach (RMIA) involves the quick performance of a massed set of large and/or small motor imitative responses prior to vocal imitation opportunities. This procedure was designed to facilitate transfer of imitation from motor to vocal modes of responding. In research applications of RMIA, a sequence of low effort motor imitation trials have been used to prime imitation of vocal responses in children with autism or developmental disabilities with no vocal behavior. There have only been a few examples of this approach applied in research, and the phenomenon responsible for inducing imitation across response modes is unknown. Generalized imitation training, behavioral momentum, and teaching arrangements that increase the value of consequences for vocal imitation have been offered as possible explanations.

See Also

- [Behavioral Momentum](#)
- [Establishing Operations](#)

References and Reading

- Nevin, J. A., & Grace, R. C. (2000). Behavioral momentum and the law of effect. *Behavioral and Brain Sciences*, 23, 73–130.
- Ross, D. E., & Greer, R. D. (2003). Generalized imitation and the mand: Inducing first instances of speech in young children with autism. *Research in Developmental Disabilities*, 24, 58–74.
- Tsiouri, I., & Greer, R. D. (2003). Inducing vocal verbal behavior in children with severe language delays through rapid motor imitation responding. *Journal of Behavioral Education*, 12(3), 185–206.
- Tsiouri, I., & Greer, R. D. (2007). The role of different social reinforcement contingencies in inducing echoic tacts through motor imitation responding in children with severe language delays. *Journal of Early and Intensive Behavior Intervention*, 4, 629–647.
- Williams, G., & Greer, R. D. (1993). A comparison of verbal behavior and linguistic communication curricula for training developmentally delayed adolescents to acquire and maintain vocal speech. *Behaviorology*, 1, 31–46.
- Young, J. M., Krantz, P. J., McClannahan, L. E., & Poulson, C. L. (1994). Generalized imitation and response-class formation in children with autism. *Journal of Applied Behavior Analysis*, 2, 685–697.

Rapid Prompting

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Definition

The Rapid Prompting method (aka RPM, Rapid Prompting, Soma[®] RPM, Informative Pointing, Spelling to Communicate, and S2C) is a teaching, therapeutic, and communication technique attributed to Soma Mukhopadhyay of the Halo-Soma Institute (Austin, TX) intended to establish independent, cognitively sophisticated pointing-,

typing-, or writing-based communication in people who are nonverbal due to autism (Friðriksson 2009; Iversen 2006, 2007; Mukhopadhyay 2008). Ostensibly based on Jean Piaget's developmental psychology (e.g., Piaget 1930) and A. Jean Ayres's theory of sensory integration (Ayres 1979), Rapid Prompting is similar to the discredited intervention Facilitated Communication (FC; Todd 2015; Tostanoski et al. 2014) in regarding autism as being substantially an expressive disorder (Crossley 1994), but superficially resembles in implementation the discrete trials methods of applied behavior analysis (ABA; Maurice et al. 1996). The subject, who is supposedly prevented by autism from displaying potentially considerable hidden literacy, selects a written response to a question or statement from a set of alternatives in a series of "teach-ask" trials that increase in complexity as the intervention proceeds. In the standard implementation, choosing a single item from a small set of choices expands to spelling out verbal responses by pointing to individual letters on a printed letter board. Thus, in practice, Rapid Prompting differs from FC in that the aide holds and moves the letter board in front of the subject rather than using physical and other prompts to guide the subject's pointing. However, as in FC, a verbally skilled individual physically or otherwise intervenes the production of the output attributed to the nonspeaking person – whether by moving the subject relative to the letters or the letters relative to the subject (see Todd 2015). Some reports suggest that the aide's assistance is not motivational as claimed by Rapid Prompting advocates; accurate responding may not occur unless the aide knows the answers (see Iversen 2006, pp. 242–248, 2007, p. 54). Also as in FC, the collection of ongoing performance data and the objective evaluation of authorship are very strongly discouraged (Halo-Soma n.d.; Iversen 2006, pp. 242–248; Mukhopadhyay 2008, pp. 16–17). To date, there are no published, methodologically sound studies of the effectiveness of Rapid Prompting for establishing reliable, independent communication in nonverbal people with autism (Lang et al. 2014; Schlosser et al. 2019).

Historical Background

Rapid Prompting was developed by Soma Mukhopadhyay, whose main professional training is in chemistry, from a collection of informal methods she claims to have used to teach her son Tito to communicate via handwriting and typing despite him having severe autism and little functional speech, (Iversen 2006; Mukhopadhyay 2008). Earning notice in India and the United Kingdom in the late 1990s, Mukhopadhyay and her son were profiled in the BBC documentary, “Tito’s Story,” in which her abilities as a teacher and his status as an avatar of hidden literacy were enthusiastically endorsed by autism professionals (see, e.g., Wing 2000/2003). In 2001, Mukhopadhyay was brought to the United States by Portia Iversen and Jon Shestack’s Cure Autism Now (CAN) Foundation. As chronicled in Iversen’s (2006) book, *Strange Son*, more endorsements followed, along with considerable mainstream media attention. Features by *60 Minutes II* (Kohn 2009), PBS (How does the autistic brain work? n.d.), *The New York Times* (Blakeslee 2002), *National Geographic* (Mind Tree Poems 2005), and *Scientific American* (Mukerjee 2004) represent just some of that early coverage. Supplementing the mainstream features were books of poetry, essays, and stories attributed to Tito Mukhopadhyay (2000, 2003), descriptions of Rapid Prompting in popular autism guidebooks (e.g., Sicile-Kira 2004), and a large number of items on the Internet.

Mukhopadhyay was based in California until 2004 when she moved her operation to what is now the Halo-Soma Institute in Austin, Texas. From Halo-Soma, Mukhopadhyay provides clinical services, presents internationally, and runs workshops. Halo-Soma does not conduct evaluation research, and Mukhopadhyay has published several works on Rapid Prompting including an instructional guide: *Understanding Autism Through Rapid Prompting Method* (Mukhopadhyay 2008), and a lesson supplement, *Curriculum Guide For Autism Using Rapid Prompting Method: With Lesson Plan Suggestions* (Mukhopadhyay 2011). An expansive website sponsored by Halo-Soma is its only other

comprehensive source on Rapid Prompting proper, featuring both free and subscription-restricted multimedia content (Halo-Soma n.d.). Iversen, who no longer collaborates with Mukhopadhyay, developed her own version of Rapid Prompting called “The Informative Pointing Method,” which she disseminates electronically from her “Strange Son” website (Iversen 2007). Another variant, “Alphabet Therapy” from Vanderbilt University, was said to combine ABA and Rapid Prompting procedures and is directed at people with Angelman Syndrome (Valle et al. 2008).

Attention to Rapid Prompting in the media, popular press, and on the Internet continues to expand. This includes not only more profiles of Mukhopadhyay and her son but a growing number of books and other accounts of individuals said to have achieved full literacy through the method (e.g., Bonker and Breen 2011; Burns and Wambua 2009; Sicile-Kira 2010). In 2009, both Soma Mukhopadhyay and Rapid Prompting were prominently featured in the Icelandic documentary “Sólskinsdrengrurinn” (Friðriksson 2009; literally, “The Sunshine Boy”; retitled “A Mother’s Courage: Talking Back to Autism” for distribution in English by Home Box Office). “Sólskinsdrengrurinn”/“A Mother’s Courage” followed Margrét Dagmar Ericsdóttir as she traveled to England and then the United States in search of help for her son’s condition, with approximately the final third of the movie devoted to the supposedly successful implementation of Rapid Prompting. FC advocates, who share Mukhopadhyay’s assumption of hidden intelligence in people with autism, increasingly reference Tito Mukhopadhyay as an example of someone whose hidden intelligence was allegedly revealed by alternative treatment methods – while generally not saying much about the Rapid Prompting technique itself (see, e.g., Biklen 2005).

Rationale or Underlying Theory

Rapid Prompting procedures were developed through trial and error, and acquired a theoretical rationale later. According to Mukhopadhyay (2008,

pp. 33–53), autism is the result of the child’s potentially more advanced cognitive abilities being undermined by altered versions of the early Piagetian stages. The cognitive difficulties are further exacerbated by poor integration of multiple sensory inputs, as suggested by Ayres (1979). However, Rapid Prompting is said not to be primarily based on psychology, but “upon how the brain works” (Halo-Soma n.d.; Mukhopadhyay, personal communication, May 6, 2009). Thus, references to neuroanatomy and physiology appear throughout Mukhopadhyay’s 2008 book, the Halo-Soma website, and workshop presentations. Over- and under-activity of the mirror neurons lead to echolalia and withdrawal, respectively (pp. 42–44). “Underconnectivity” creates poor “central coherence” (p. 26). A functional laterality assumption suggests teaching from the subject’s right side to “activate the student’s left-brain for reasoning” (p. 185). Troublesome behaviors such as hair pulling and perseverations are said to be “instinctive” and arise in the caudate nucleus (pp. 211–215). Voluntary or “deliberate” actions arise in the putamen and ultimately work their way to the motor cortex (pp. 215–220). Obsessive behaviors are said to be associated with “extreme emotions” and potentially lead to aggression because “the caudate nucleus is very close to the amygdala” (p. 216). The general solution to the communication and behavior problems associated with autism is to “activate the reasoning part of the brain so that the student becomes distracted by and engaged in learning” (Halo-Soma n.d.). This is done by providing a series of cognitive challenges of graduated difficulty through the “dominant sensory channel” to repair the defective sensorimotor and preoperational cognitive structures and then continue to provide input that will force the advancement into the concrete and formal operational stages.

At a pragmatic level, Rapid Prompting shares with FC the “presumption of competence”: the assumption that nonverbal people with autism are likely to possess considerable hidden knowledge that they cannot express due to hypothesized motor planning impediments (Biklen 2005; Iversen 2006; Mukhopadhyay 2008). In other words – and in apparent contradiction to other

elements of Rapid Prompting theory – autism is a disorder that interferes with expression and cognitive integration, not necessarily the ability to absorb and process knowledge itself (Mukhopadhyay 2008, p. 132). The assumption of hidden literacy proscribes that Rapid Prompting training should begin with age-typical materials and concepts, such as multiplication and division for a 12-year-old, and abstract ideas rather than foundational ones, such as color and number. As in FC, the hidden literacy assumption also seems to explain the sudden appearance of linguistically sophisticated responding in otherwise nonverbal, profoundly affected subjects who have never participated in formal classroom instruction, sometimes the first time Rapid Prompting is used, sometimes at a much higher level than predicted by the subject’s age, and sometimes in a language other than the one the subject has been primarily exposed to (see, e.g., Bonker and Breen 2011; Burns and Wambua 2009; Friðriksson 2009; Kohn 2009). Skeptics attribute such performances to the teachers rather than the students (Spitz 1997).

Aside from seating position implications of laterality and the link between the hidden literacy assumption and teaching level, there is little clear relationship between Mukhopadhyay’s neurological conceptualizations and the elements of the Rapid Prompting training protocol. For instance, the solution to the problem of “instinctive actions” such as hair pulling does not seem particularly specific to the caudate nucleus. It is to simply ask the student whether the cause of the problem is being “tired” or “mad,” writing those words on separate pieces of paper, and finally telling a social story related to the word pointed to (Mukhopadhyay 2008, p. 185). This disconnect between the methods employed and their putative or actual empirical basis is particularly true of Rapid Prompting’s use of learning-based techniques of stimulus fading across match-to-sample trials of increasing complexity – a kind of errorless stimulus control procedure. Neither Mukhopadhyay’s multivolume book series (e.g., *Understanding Autism Through Rapid Prompting Method*) nor the Halo-Soma website references any relevant, modern procedural, empirical, or

theoretical work in learning theory, particularly on the subject of stimulus control and stimulus transfer (see, e.g., Wolery and Gast 1984).

Goals and Objectives

The goal of Rapid Prompting and its variants is to establish functional independent pointing-based communication in people who are otherwise non-verbal due to severe autism or other developmental disabilities: “RPM aims to develop the skill of pointing to spell and/or write” (Mukhopadhyay 2008, p. 209). In contrast to the comprehensive educational, social, and functional scope of applied behavior analysis and some other interventions, Rapid Prompting is not intended to directly remediate the behavior problems associated with autism, teach activities of daily living, or prepare the subject for independence (Mukhopadhyay 2008, pp. 206–209). However, proponents assert that Rapid Prompting establishes functional communication, which is expected to reduce some of the problems associated with autism because with it the subject can express needs and ideas. To the extent, the subject cannot control overt problem behaviors or is unable to exhibit normal reactions to social situations, he or she is expected to be able to communicate accurately about the expected reactions (p. 208).

Treatment Participants

Rapid Prompting is directed primarily at people with autism who have little or no effective verbal communication ability, who can select items by pointing. Although difficult-to-manage behaviors such as tantrums, self-injury, or aggression might practically preclude a person’s participation in Rapid Prompting, there are no specific contraindications based on age, gender, or other aspects of behavioral profile (Mukhopadhyay 2008, pp. 86–89). A willing and able teacher with good verbal and organizational skills – typically a parent – is needed to provide the training and ongoing communication support. Alphabet Therapy, while

directed at people with Angelman syndrome, did not seem to have any features that would limit its use to that condition.

Treatment Procedures

Rapid Prompting training consists of a series of “teach-ask” multiple-choice or written-response trials that increase in difficulty as treatment progresses. The style of teaching is supposed to be dictated by the “open sensory channel” as indicated by behaviors such as self-stimulation (cf. Pashler et al. 2009). But the basic features of the training appear to be the same regardless of which sensory channel is presumed to be dominant. In a typical early training trial, the subject is given an item of information, then immediately queried about it. The subject might be told, “A triangle has three sides.” Then, as the teacher provides a set of response alternatives including the correct answer, asked “How many sides does a triangle have?” Early in the training, the response required could include selecting a single object, picture, letter, digit, or written word from two or more alternatives placed in front of the child. Later, it would include composing verbal statements by sequentially choosing letters from a letter board held by the teacher. Responding is prompted by specific actions of the teacher, such as putting a pencil (used as a pointer) in the subject’s hand after the question is asked. The pencil is removed from the subject’s hand to signal the end of each trial (see Mukhopadhyay 2008, pp. 127–178). In some implementations, the pencil is pushed through the holes associated with letters and numbers in plastic alphanumeric stencils – the holes said to make selecting easier. The stencils may also be used to guide letter formation if printing is to be taught. However, the stencils are usually replaced by a laminated letter board that the subject points to either with the pencil or sometimes with just the finger (see, e.g., Nonverbalautismmom 2010).

Mukhopadhyay encourages teachers to use words rather than pictures or objects and to write each of the response options by hand on pieces of paper torn from larger sheets during the trials.

(The use of torn paper is so fundamental to Rapid Prompting that it is depicted on the cover of Mukhopadhyay's 2008 and 2011 books.) Thus, in a typical teaching trial the teacher would:

1. Make a statement.
2. Ask a question related to the statement.
3. Tear the needed number of sheets of paper from a larger piece of paper.
4. Quickly write the choices on paper pieces; one correct, the others distractors.
5. Place the choices in front of the subject.
6. Hand the subject a pencil for pointing, prompting to initiate as necessary.
7. Take the pencil from the student following the selection.
8. Collect all the paper, taping the torn pieces to a larger piece.

The sound of the paper tearing is said to focus the subject on the task and prompt immediate responding. The pieces may be slapped down loudly to add to the prompting effect (see Kohn 2009). Two to three trials per minute might be achieved if the subject responds immediately, and the answers do not take a long time to write. Explicit verbal directives to pay attention and respond quickly are also common. It is not unusual to see a teacher speak continuously and gesture frequently (i.e., give "air prompts") during the trials. The constant talking is said to help the subjects learn to attend to speech, particularly aiding those who "are more comfortable listening to environmental sounds than spoken language" (Halo-Soma n.d.). Hand-over-hand guidance of responding is not normally used except when it is needed to initially establish pointing or writing (Mukhopadhyay 2008, p. 129).

Escape or other disruptive behavior is dealt with physically. Subjects are kept in place by positioning them between the table and walls such that the teacher can block the subject's only escape route and is able to move away if the subject's behavior becomes unmanageable (see p. 185). Errors are corrected by verbal reprimands, trial termination, physical redirection using the stencil or letter board to push the subject's hands away. This can include slapping or shaking the

letter board against the subject's face or chest (see Friðriksson 2009; Iversen 2007, p. 25). Explicit reinforcement of correct responses is not considered necessary, although incidental verbal praise embedded in feedback about accuracy is common (Ochs et al. 2005). Collecting ongoing performance data is strongly discouraged (Mukhopadhyay 2008), although video recordings are sometimes made (see Ochs and Solomon 2010), and stimulus materials from sessions may be retained (Mukhopadhyay 2008). According to Mukhopadhyay (2008, pp. 16–17), data collection is unnecessary and stigmatizing. The Halo-Soma website reports: "Soma does not use data sheets. She feels taking down hash marks interrupts the flow of instruction and inhibits the student's performance and success."

Despite its very heavy dependence on multiple forms of prompting, Rapid Prompting includes no procedures to either prevent the development of or test for "prompt dependency" – responding by the subject influenced by conscious or unconscious cues from teacher such as looking at the answer or frequently placing the correct choice in the same relative position. Such protections are more than justified by the use of prompt-fading procedures. But they are essential because: (1) facilitator influence is strongly suggested by reports of astonishing literacy suddenly appearing in people who were previously entirely nonverbal and showed no previous signs of such talents (Bonker and Breen 2011; Burns and Wambua 2009; Friðriksson 2009; Iversen 2006; Kohn 2009; Mukhopadhyay 2008); (2) there are several reported instances in which correct answers were not given unless the teacher was aware of the needed information – including a message-passing test in which Mukhopadhyay and her son were unable to correctly answer a single question about a story Mukhopadhyay had not heard (Iversen 2006; see especially pp. 242–248; 2007, p. 54); (3) video recordings of Rapid Prompting with letter boards seem to show the person holding the board subtly moving each letter toward the subject's finger, with the subject responding by moving his or her finger toward the letter (e.g., Friðriksson 2009; Nonverbalautismmom 2010); (4) research on facilitator influence in FC clearly

demonstrates that teachers can be unaware of authoring responses even when they are physically holding the subjects' hands (Shane 1994); (5) over a century of research on communication, particularly studies on the phenomenon of automatic writing, has revealed how the unconscious composition of complex verbal output can be engendered and maintained by Rapid Prompting-like situations (Spitz 1996); and (6) the procedures used in Rapid Prompting – fading verbal and gestural prompts for selecting among a small number of discrete items and then expanding the number of choices – very closely replicate the Pinchbeck Technique, a method conjurers have used for centuries to create the illusion of letter-by-letter communication (Pinchbeck 1805). Pinchbeck himself warned of the development of unconscious cueing of letter selections by the conjurer (Pinchbeck 1805, p. 26).

Efficacy Information

Mukhopadhyay claims to have treated hundreds of individuals, presumably successfully (Mukhopadhyay 2008, p. 2). Over 300 clients, mostly children, were pictured and partially identified on the Halo-Soma website. Other Rapid Prompting providers make similar claims. Yet, there are no published, methodologically sound, peer-viewed experimental studies examining the overall effectiveness of Rapid Prompting or its variants, nor are there any published objective evaluations of the validity of communications attributed to individual subjects (Lang et al. 2014; Schlosser et al. 2019). Indeed, the principal advocates of Rapid Prompting range from being dismissive of to expressly hostile to ongoing data collection and scientific tests of authorship (Iversen 2006, pp. 242–248; Mukhopadhyay 2008, pp. 16–17). Iversen, for instance, described the objective evaluation of authorship as a “crime” (Iversen 2006, pp. 242–248). Two published studies are said to demonstrate hidden cognitive competence based on Rapid Prompting (Chen et al. 2012; Jaswal et al. 2020). Those claims remain unfounded primarily due to the failure to the procedures used to clearly establish

that the subjects (rather than the aides) authored the output (Tostanoski 2014; Vyse 2020). Nevertheless, the efficacy of the method is all but assured by advocates. University of California San Francisco Emeritus neurologist Michael Merzenich, for instance, stated on the television program *60 Minutes II*, “I think it’s almost certain that this method can be used on many autistic children. In fact, the initial indication from these studies is that it might apply to the substantial majority of these children” (in Kohn 2009). Head of the Autism Research Institute and advocate of the DAN! biomedical protocol, Barry Edelson (Halo-Soma n.d.), has stated, “If my child had a limited verbal skills, I would look into the Rapid Prompting Method.” No objective evidence was provided to support these claims and recommendations, nor have the “studies” referred to by Merzenich appeared in the scientific literature (see Iversen 2006). According to Mukhopadhyay, the appropriate measures of the efficacy of Rapid Prompting are standard educational assessments – although doing such assessments with Rapid Prompting would necessarily raise the same questions of independence and authorship as would be raised by the use of FC.

Due to the lack of empirical support for Rapid Prompting, and its many procedural and conceptual similarities to Facilitated Communication, the American Speech Language and Hearing Association (ASHA) has issued a statement against its use (ASHA 2018). The statement begins:

It is the position of the American Speech-Language-Hearing Association (ASHA) that use of the Rapid Prompting Method (RPM) is not recommended because of prompt dependency and the lack of scientific validity. Furthermore, information obtained through the use of RPM should not be assumed to be the communication of the person with a disability.

Outcome Measurement

The validity of Rapid Prompting output could be assessed by effectively blinding the aide to the information asked of the subject. No such assessments have been published. There are no other standardized evaluations of Rapid Prompting.

Essentially, Rapid Prompting is presented as an augmentative or alternative communication (ACC) method, the use of which will make it possible to administer other kinds of standardized evaluations. Mukhopadhyay's first book (2008) contains a full chapter on administering tests using Rapid Prompting. However, those are not guidelines for formal evaluations, but rather advice on generating multiple-choice questions. There are no established standardized tests or assessments that have been normed for use with Rapid Prompting. The responsible and ethical administration of standardized tests would preclude use of scientifically discredited and unestablished communication interventions such as Facilitated Communication, Rapid Prompting, and their many variants. The direct physical intervention of the aide in producing test responses would call the validity of the test results into question.

Qualifications of Treatment Providers

There are no publicly recognized standards, regulations, or laws for registering, licensing, or certifying Rapid Prompting treatment providers. There are no listed university degree or certificate programs on Rapid Prompting as a clinical profession. The Halo-Soma Institute itself sponsors no recognized credentialing and seems to have published no professional or ethical standards of its own—although it appears to bestow a variety of titles suggesting formal certification (Halo-Soma n.d.). Thus, status of Rapid Prompting providers as professional therapists, psychologists, teachers, or aides is, at best, uncertain. The Halo-Soma organization has offered extended training workshops for selected application. However, most RPM training is appears to be provided directly to employees and clients through centers and in training workshops.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Early Intensive Behavioral Intervention \(EIBI\)](#)
- [Facilitated Communication](#)

- [Prompting](#)
- [Sensory Integration \(SI\) Therapy](#)

References and Reading

- American Speech-Language-Hearing Association. (2018). Rapid prompting method [Position statement]. Available online at www.asha.org/policy/; <https://doi.org/10.1044/policy.PS2018-00351>.
- Ayres, A. J. (1979). *Sensory integration and the child*. Los Angeles: Western Psychological Services.
- Biklen, D. (2005). *Autism and the myth of the person alone*. New York: New York University Press.
- Blakeslee, S. (2002, November 19). A boy, a mother and a rare map of autism's world. *New York Times*. Retrieved August 8, 2011, from <http://www.nytimes.com/2002/11/19/science/a-boy-a-mother-and-a-rare-map-of-autism-s-world.html>
- Bonker, E. M., & Breen, V. G. (2011). *I am in here: The journey of a child with autism who cannot speak but finds her voice*. Grand Rapids: Revell.
- Burns, M. P., & Wambua, D. M. (2009). *Danson. The extraordinary discovery of an autistic child's innermost thoughts and feelings*. Pittsburgh: St. Lynn's Press.
- Chen, G. M., Yoder, K. J., Ganzel, B. L., Goodwin, M. S., & Belmonte, M. K. (2012). Harnessing repetitive behaviours to engage attention and learning in a novel therapy for autism: An exploratory analysis. *Frontiers in Psychology*, 12(3), 1–16.
- Crossley, R. (1994). *Facilitated communication training*. New York: Teachers College Press.
- Friðriksson, F. Þ. (Director). (2009). *Sólskinsdrengrinn* (Documentary movie; aka: "The sunshine boy," "A Mother's courage: Talking back to autism"). Frontier Filmworks in Association with Klikk Productions: Reykjavik: Iceland.
- Halo-Soma. (n.d.). Rapid prompting method for autism. Retrieved November 20, 2011, from <http://www.halo-soma.org>
- How does the autistic brain work?. (n.d.). Retrieved August 19, 2011, from http://www.pbs.org/kcet/closertotruth/explore/show_03.html
- Iversen, P. (2006). *Strange son: Two mothers, two sons, and the quest to unlock the hidden world of autism*. New York: Riverhead Books.
- Iversen, P. (2007). The informative pointing method. Unpublished manuscript. Retrieved August 8, 2011, from http://www.strangeson.com/files/Informative_Pointing_Method.pdf
- Jaswal, V. K., Wayne, A., & Golino, H. (2020). Eye-tracking reveals agency in assisted autistic communication. *Sci Rep*, 10, 7882.
- Kohn, D. (2009, February 11). Breaking the silence. (CBS 60 Minutes II; video originally broadcast 2003). Retrieved August 8, 2011, from <http://www.cbsnews.com/stories/2003/01/14/60II/main536416.shtml>
- Lang, R., Tostanoski, A. H., Travers, J., & Todd, J. (2014). The only study investigating the rapid prompting

- method has serious methodological flaws but data suggest the most likely outcome is prompt dependency. *Evid Based Commun Assessment Interv*, 8, 40–48.
- Maurice, C., Green, G., & Luce, S. C. (1996). *Behavioral intervention for young children with autism: A manual for parents and professionals*. Austin: Pro-Ed Incorporated.
- Mind Tree Poems. (2005, March). Retrieved August 19, 2011, from http://ngm.nationalgeographic.com/ngm/0503/feature1/online_extra.html
- Mukerjee, M. (2004). A transparent enigma. *Scientific American*, 299(6), 24–25.
- Mukhopadhyay, T. R. (2000). *Beyond the silence*. London: Central.
- Mukhopadhyay, T. R. (2003). *The mind tree. A miraculous child breaks the silence of autism*. New York: Arcade.
- Mukhopadhyay, S. (2008). *Understanding autism through rapid prompting method*. Denver: Outskirts Press.
- Mukhopadhyay, S. (2011). *Curriculum guide for autism using rapid prompting method: With lesson plan suggestions*. Denver: Outskirts Press.
- Nonverbalautismmom. (2010, March 8). Non-verbal autism – letter of thanks. Retrieved August 11, 2011, from <http://www.youtube.com/watch?v=Wvn7kYJyOFM>
- Ochs, E., & Solomon, O. (2010). Autistic sociality. *Ethos*, 38(1), 69–92.
- Ochs, E., Solomon, O., & Sterponi, L. (2005). Limitations and transformations of habitus in child-directed communication. *Discourse Studies*, 7(4–5), 547–583.
- Pashler, H., McDaniel, M., Rohrer, D., & Bjork, R. (2009). Learning styles: Concepts and evidence. *Psychological Science in the Public Interest*, 9(3), 105–119.
- Piaget, J. (1930). The child's conception of physical causality. (M. Gabain, Trans.) New York: Harcourt, Brace and Company.
- Pinchbeck, W. F. (1805). *The expositor*. Wichita, KS: Stevens.
- Shane, H. C. (Ed.). (1994). *Facilitated communication: The clinical and social phenomenon*. San Diego: Singular Press.
- Sicile-Kira, C. (2004). *Autism spectrum disorders: The complete guide to understanding autism, Asperger's syndrome, pervasive developmental disorder, and other ASDs*. New York: Perigee Books.
- Sicile-Kira, J. (2010). How the rapid prompting method gave me a voice. *Autism File*, 34, 64–65.
- Spitz, H. H. (1997). *Nonconscious movements: From mystical messages to facilitated communication*. Mahwah: Lawrence Erlbaum Associates.
- Todd, J. T. (2015). Old horses in new stables: Rapid prompting, facilitated communication, science, ethics, and the history of magic. In R. M. Foxx & J. A. Mulick (Eds.), *Controversial therapies for developmental disabilities: Fad, fashion, and science in professional practice* (2nd ed.). Mahwah: Routledge.
- Tostanoski, A., Lang, R., Raulston, T., Carnett, A., & Davis, T. (2014). Voices from the past: Comparing the rapid prompting method and facilitated communication. *Developmental Neurorehabilitation*, 17, 219–223.
- Valle, C., Johnson, M., Huffsmith, B., Rossi, L. C., Freitas, L., O'Dowd, L., Rabbitt, C., & Bichell, T. J. (2008). *Alphabet therapy: A novel way to teach academic skills to children with Angelman syndrome*. Chicago: Workshop presented at the convention of the Association for Behavior Analysis International.
- Vyse, S. (2020). Of eye movements and autism: The latest chapter in a continuing controversy. *Skeptical Inquirer*. Retrieved June 26, 2020 from <https://skepticalinquirer.org/exclusive/of-eye-movements-and-autism-the-latest-chapter-in-a-continuing-controversy/>
- Wing, L. (2000/2003). Foreword. In T. R. Mukhopadhyay (Ed.), *The mind tree. A miraculous child breaks the silence of autism* (pp. ix–xii). New York: Arcade.
- Wolery, M., & Gast, D. L. (1984). Effective and efficient procedures for the transfer of stimulus control. *Topics in Early Childhood Special Education*, 4(3), 52–77.

Rapiflux

► Fluoxetine

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2012–2017: Professor Emerita of Neurology and Pediatrics (Neurology), Albert Einstein College of Medicine, Bronx, NY

Major Honors and Awards

Dr. Rapin has been recognized as an early leader in child neurology by most major professional societies. She served as an officer of the Child Neurology Society, the International Child Neurology Association, the American Academy of Neurology, the American Neurological Association, and the International Neuropsychological Society. Her work was supported by research grants from the National Institutes of Health, the March of Dimes, and autism foundations. She has received multiple honors and awards from US and international organizations for her work on genetic diseases of children; childhood deafness; and causes of intellectual disability, autism, and childhood epilepsy. She has received lifetime awards for her teaching of medical students, residents, and fellows; in 2008 she received the Lifetime Achievement Award from the International Society for Autism Research and in 2010 the President's Award from the American Academy of Neurology. She served on the editorial board of multiple major journals in neurology and neuropsychology.

Landmark Clinical, Scientific, and Professional Contributions

Dr. Rapin authored one book and coedited 10 books on child neurology, including two volumes on Child Neuropsychology for the Handbook of Neuropsychology. She published about 100 chapters and 183 articles in scientific journals (including *Science*). These have ranged from identification of progressive genetic and other diseases of the brain in her patients to methods of testing neurological function, to elucidations of the functioning of the auditory, visual, and motor systems, to issues of classification in developmental and psychiatric illnesses, to normal and abnormal development of higher cortical functions in children. One unique contribution is her important

theoretical and empirical papers about the relationship between sensory stimulation and development of higher cortical functions, including those affecting autism (see References).

Dr. Rapin has been a strong advocate for families with affected children and has won the devotion of these families with her reputation for direct and clear communication about all aspects of their child's condition. Dr. Rapin thinks broadly and deeply across disciplinary lines, promotes the careers of colleagues in her own and related disciplines, allowing her to formally and informally train many US and international colleagues in neurology, pediatrics, neuropsychology, language and communication disorders, and the nosology of disease.

Short Biography

Dr. Rapin was born in Lausanne, Switzerland, the eldest of three children, to a Swiss father and an American mother. Her mother, who belonged to a close-knit Connecticut family, met a young professor of French when she spent a summer in Lausanne as a Vassar student. They were engaged during the winter she spent at the Sorbonne in Paris after graduation. Thus, Dr. Rapin grew up in a bilingual Swiss home lined with books, where reading aloud in French or English, listening to music, and talking to interesting guests from many countries invited to tea or dinner were commonplace. Dr. Rapin remembers the war years in Switzerland as hated freezing winters with a sad mother completely cut off from her American family. Dr. Rapin decided to become a doctor when she was 10 years old, her interest having been piqued by the differences between active and passive immunity; despite literary parents, her sister became a research biologist and her brother a professor of computer science. She started at the Lausanne medical school after the war and graduated as a physician in 1952. Fascinated by neurology from the start, she discovered child neurology during a 1951 rotation in Paris; however, training was nonexistent in Switzerland or in fact anywhere. After completing the archival

work for a clinical neuropathology doctoral thesis, she moved to NY, where she became a Diplomate (Neurology), of the American Board of Psychiatry and Neurology in 1960, with Special Competence in Child Neurology in 1968. She served on the faculty of Columbia College of Physicians and Surgeons and Albert Einstein School of Medicine until her retirement from active practice in 2012, having trained many generations of child neurologists and evaluated and treated several thousand children. She lives in upstate New York and continues her scholarly work in writing and reviewing as Professor Emerita in Neurology and Pediatrics (Neurology). She is married to Harold Oaklander, Ph.D., and has four children and four grandchildren.

After this biosketch of Dr. Rapin was written, she passed away after a short illness, on May 24, 2017. She is deeply mourned by her family, colleagues, trainees, and patients.

References and Reading

Selected Papers on Sensory Input and Development of Higher Cortical Functions

- Jure, R., Rapin, I., & Tuchman, R. F. (1991). Hearing impaired autistic children. *Developmental Medicine and Child Neurology*, 33, 1062–1072.
- Jure, R., Pogonza, R., & Rapin, I. (2016). Autism Spectrum Disorders (ASD) in blind children: Very high prevalence, potentially better outlook. *Journal of Autism and Developmental Disorders*, 46(3), 759–769. <https://doi.org/10.1007/s10803-015-2612-5>.
- Rapin, I. (1964). Evoked responses to clicks in a group of children with communication disorders. *Annals of the New York Academy of Sciences*, 112, 182–203.
- Rapin, I. (1978). Consequences of congenital hearing loss – A long term view. *Canadian Journal of Otolaryngology*, 7, 473–483.
- Rapin, I. (1979a). Effects of early blindness and deafness on cognition. In R. Katzman (Ed.), *Congenital and acquired cognitive disorders* (pp. 189–245). New York: Raven Press.
- Rapin, I. (1979b). Conductive hearing loss: Effects on children's language and scholastic skills. A review of the literature. *The Annals of Otolaryngology, Rhinology, and Laryngology*, 88(Suppl. 60), 3–12.
- Rapin, I. (n.d.). Why the high prevalence of autism in children with congenital blindness? (Submitted for publication).
- Rapin, I., & Graziani, L. J. (1967). Auditory evoked responses in normal, brain damaged, and deaf infants. *Neurology*, 17, 881–894.
- Wilson, J. J., Rapin, I., Wilson, B. C., & Van Denburgh, F. V. (1975). Neuropsychologic function of children with severe hearing impairment. *Journal of Speech and Hearing Research*, 18, 634–652.
- ### Other Recent Autism Papers
- Goldman, S., Wang, C., Salgado, M. W., Greene, P. E., Kim, M., & Rapin, I. (2013a). Motor stereotypies in children with autism and other developmental disorders. *Developmental Medicine and Child Neurology*, 51(1), 30–38.
- Goldman, S., O'Brien, L. M., Filipek, P. A., Rapin, I., & Herbert, M. R. (2013b). Motor stereotypies and volumetric brain alterations in children with autistic disorder. *Research in Autism Spectrum Disorders*, 7(1), 82–92.
- Graf, W. D., Miller, G., Epstein, L. G., & Rapin, I. (2017). The autism “epidemic”: Ethical, legal, and social issues in a developmental spectrum disorder. *Neurology*, 88(14), 1371–1380. ID#: NEUROLOGY/2016/764498.
- Pfaff, D. W., Rapin, I., & Goldman, S. (2011). Male predominance in autism: Neuroendocrine influences on arousal and social anxiety. *Autism Research*, 4(3), 163–176.
- Rapin, I. (2006). Language heterogeneity and regression in the autistic spectrum disorders – Overlaps with other childhood language regression syndromes. *Clinical Neuroscience Research*, 6, 209–218.
- Rapin, I. (2014). Classification of behaviorally-defined disorders: Biology vs. the DSM. *Journal of Autism and Developmental Disorders*, 44(10), 2661–2666.
- Rapin, I. (2016). Dyscalculia and the calculating brain. *Journal of Pediatric Neurology*, 61, 11–20. <http://authors.elsevier.com/sd/article/S0887899416000679>
- Shetreat-Klein, M., Shinnar, S., & Rapin, I. (2014). Abnormalities of joint mobility and gait in children with autism spectrum disorders. *Brain & Development*, 36(2), 91–96.
- Silver, W. G., & Rapin, I. (2012). Neurobiological basis of autism. *Pediatric Clinics of North America*, 59(1), 45–61.
- Tuchman, R. F., Moshé, S. L., & Rapin, I. (2009). Convulsing toward the pathophysiology of autism. *Brain & Development*, 31(2), 95–103.173.
- Valicenti-McDermott, M., McVicar, K., Rapin, I., Wershil, B. K., Cohen, H., & Shinnar, S. (2006). Frequency of gastrointestinal symptoms in children with autistic spectrum disorders and association with family history of autoimmune disease. *Journal of Developmental and Behavioral Pediatrics*, 27, S128–S136.
- Weidenheim, K. M., Escobar, A., & Rapin, I. (2012). Brief report: Life history and neuropathology of a gifted man with Asperger syndrome. *Journal of Autism and Developmental Disorders*, 42(3), 460–467.

Ras/MAPK Pathway Syndromes

- [Noonan and Ras/MAPK Pathway Syndromes](#)
-

Rating Scale

- [Standardized Behavior Checklists](#)
-

Ratio-Haloperidol

- [Haloperidol](#)
-

Rational Demand Avoidance

- [Pathological Demand Avoidance \(PDA\)](#)
-

RAVLT

- [Rey Auditory Verbal Learning Test \(Rey AVLT\)](#)
-

RDoC and ASD

- [RDoC and Autism](#)
-

RDoC and Autism

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Synonyms

[RDoC and ASD](#); [Research domain criteria and ASD](#)

Definition

The Research Domain Criteria (RDoC) project was initiated by the National Institute of Mental Health in 2009 to develop a novel classification of psychopathology. It represents a framework that explicates genes, brain, and behavior dimensions across heterogeneous categories of mental disorders (Insel and Wang 2010). As opposed to the symptom-based, categorical approach used to diagnose mental health conditions, the RDoC framework considers psychopathology based on disorders of brain circuits using genetics and neuroscience research tools to discover the biosignatures that can inform diagnoses and treatment of mental illness. The RDoC constructs are organized in a matrix containing five primary domains: negative valence, positive valence, cognitive systems, social processes, and arousal/regulatory systems. Each domain includes constituent constructs and seven units of analysis (e.g., genes, circuits, behavior).

The RDoC framework offers a new way of characterizing features of autism spectrum disorder (ASD) given the phenotypic heterogeneity in ASD (e.g., verbal abilities, cognitive functioning, social interests) representing continua that extend into normative ranges. For example, the social processes domain includes constructs that bridge social behavior with circuits of attachment, social dominance, and perception/understanding of self and others. The neural underpinnings of social behaviors, such as eye gaze and emotion recognition, have been studied with various neuroimaging and electrophysiological methods in order to establish biomarkers of ASD. In addition, the RDoC framework may be valuable in understanding the underlying mechanisms of co-occurring conditions in ASD. For example, anxiety is prevalent in individuals with ASD. However, using the current categorical approach, it is challenging to discern whether anxiety represents a true comorbidity or is unique to autism and underpinned by a distinct pathophysiological mechanism.

Current research using the RDoC framework in ASD has suggested conceptualizing ASD symptoms along positive, negative, and

cognitive dimensions, similar to a dimensional approach traditionally used for schizophrenia. Positive features could include behaviors infrequent in typically developing (TD) individuals (e.g., repetitive behaviors, echolalia, and circumscribed interests); negative features could reflect the absence of behaviors expected in TD individuals (e.g., eye contact or reciprocal social interactions); and cognitive features could include patterns of thinking and behavior common in ASD (e.g., rigidity of thinking, impaired attention).

The use of dimensional measures to capture variation in traits across individuals may leverage the discovery of neural correlates of ASD symptom-dimensions or brain-behavior associations in ASD. Thus, findings from functional MRI studies suggest that anxiety in ASD could be associated with aberrant activity of the amygdala, implicated in emotion processing, under the negative valence domain (i.e., acute threat or fear) (Herrington et al. 2017) as well as with aberrant activity of the nucleus accumbens, implicated in reward processing, under the positive valence domain (i.e., reward learning) (Richey et al. 2014). Within the social processes domain, the perception of self and others construct has been used to examine brain regions involved in social cognition in individuals with ASD and with schizophrenia using tasks of social-cognitive processing such as perspective taking (Eack et al. 2017).

Another overarching goal of the RDoC approach is to accelerate research supporting precision medicine that produces treatments tailored to the individual patient's pathophysiology of the disorder and predictors of response to a given intervention. For example, an investigation of neural-systems-level targets of behavioral therapy for associated symptoms in ASD, such as irritability and aggression under the frustrative nonreward construct, can shed light on neural markers and predictors of treatment response (Sukhodolsky et al. 2016). Further, integration of information from neuroimaging and genetics could be helpful in determining a dimensional, biologically driven classification of mental illness. Next steps for the RDoC project include

the need to develop new constructs and domains. For example, emotion regulation is currently considered an implicit component in many RDoC constructs (e.g., control of fear or impulsive behaviors), while there is emerging evidence that emotion regulation may be transdiagnostic and require an explicit role in the RDoC matrix (Aldao et al. 2016).

See Also

► Dimensional Versus Categorical Classification

References and Reading

- Aldao, A., Gee, D. G., De Los Reyes, A., & Seager, I. (2016). Emotion regulation as a transdiagnostic factor in the development of internalizing and externalizing psychopathology: Current and future directions. *Development and Psychopathology*, 28(4pt1), 927–946. <https://doi.org/10.1017/s0954579416000638>.
- Eack, S. M., Wojtalik, J. A., Keshavan, M. S., & Minshew, N. J. (2017). Social-cognitive brain function and connectivity during visual perspective-taking in autism and schizophrenia. *Schizophrenia Research*, 183, 102–109. <https://doi.org/10.1016/j.schres.2017.03.009>.
- Herrington, J. D., Maddox, B. B., McVey, A. J., Franklin, M. E., Yerys, B. E., Miller, J. S., & Schultz, R. T. (2017). Negative valence in autism Spectrum disorder: The relationship between amygdala activity, selective attention, and co-occurring anxiety. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 2, 510.
- Insel, T. R., & Wang, P. S. (2010). Rethinking mental illness. *JAMA*, 303(19), 1970–1971.
- Richey, J. A., Rittenberg, A., Hughes, L., Damiano, C. R., Sabatino, A., Miller, S., . . . Dichter, G. S. (2014). Common and distinct neural features of social and non-social reward processing in autism and social anxiety disorder. *Social Cognitive and Affective Neuroscience*, 9(3), 367–377. <https://doi.org/10.1093/scan/nss146>.
- Sukhodolsky, D. G., Vander Wyk, B. C., Eilbott, J. A., McCauley, S. A., Ibrahim, K., Crowley, M. J., & Pelphrey, K. A. (2016). Neural mechanisms of cognitive-behavioral therapy for aggression in children and adolescents: Design of a Randomized Controlled Trial within the National Institute for Mental Health Research domain criteria construct of Frustrative non-reward. *Journal of Child and Adolescent Psychopharmacology*, 26(1), 38–48. <https://doi.org/10.1089/cap.2015.0164>.

Reaction Time

- ▶ [Information Processing Speed](#)
- ▶ [Response Latency](#)

Reactive Attachment Disorder

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Synonyms

[Attachment disorder](#); [Disordered attachment](#)

Short Description or Definition

Children with reactive attachment disorder (RAD) or disinhibited social engagement disorder (DSED) exhibit abnormal social and attachment-related behaviors before the age of 5 years. According to the *Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5)*, and the *International Classification of Diseases, 10th revision (ICD-10)*, these are stress-related disorders, and the symptoms occur in consequence, i.e., following, or in reaction to, pathogenic caretaking of the child in the early years of life.

In the DSM-IV, the RAD diagnosis included two subtypes, characterized by different behavioral patterns. In the inhibited or withdrawn type, children avoid comfort, seem afraid to seek it, do not respond to it, and even resent comfort offered to them by caregivers. Failure to develop attachments to any caregivers is the essence of this disorder. In the disinhibited or indiscriminate type, children are unselectively attached to familiar as well as unfamiliar figures. This disinhibited subtype is now considered in the DSM-5 as a separate diagnosis, the disinhibited social engagement disorder (DSED). Socially disinhibited behavior with strangers is the core feature of these disorders. Both disorders are caused by

social neglect in childhood, presenting with distinct behavioral manifestations, prognosis, and vulnerability factors, and were therefore redefined as distinct disorders.

Some children who are diagnosed with autism spectrum disorders (ASD) may present behavioral symptoms that resemble the clinical picture of RAD or DSED. However, it is well established that autism does not result from, nor is a reaction to, inadequate caretaking. Therefore, these diagnoses are exclusive, and children diagnosed with an ASD cannot receive an additional diagnosis of RAD or DSED, even if they manifest resembling symptoms. Moreover, RAD and DSED can be diagnosed only if symptoms do not meet criteria for ASD and if symptoms cannot be accounted for solely by developmental delay.

Categorization

RAD first appeared in the third version of the DSM in 1980. In this early version of the disorder, growth failure, lack of social responsivity, and evidence of physical and emotional neglect were the central features, with onset by the age of 8 months. The DSM-III definition did not include the need to evaluate children for ASD or developmental delay. In the DSM-III-R, the link to growth failure was excluded, and the age of onset was changed to the first 5 years of life. In 2003, a task force of the American Academy of Child and Adolescent Psychiatry (AACAP) proposed a set of empirically based diagnostic criteria for RAD. These modified criteria include the requirement for pathogenic caregiving, as stipulated in the *DSM-IV* (e.g., persistent disregard of the child's basic physical or emotional needs or repeated changes of primary caregiver). The two behavioral subtypes were first introduced in this version and persisted in the DSM-IV and DSM-IV-TR. The new behavioral criteria for the two RAD types focused more on specific attachment behaviors. The DSM-5 and the ICD-10 now include RAD and DSED as separate disorders (Zeanah et al. 2016).

The diagnostic criteria for RAD and DSED usually refer to toddlers and young children, and

the majority of research conducted on RAD and DSED has been conducted on young children. Moreover, the attachment behaviors that are used for RAD and DSED diagnoses are not developmentally adapted for older children or adults. Therefore, diagnosing RAD or DSED in older children is more complicated, and less is known about the manifestation of these disorders in older children and adolescents. Additionally, inasmuch as the *DSM* requires that the symptoms are present before the age of 5, a reliable history is necessary but in many cases of older children is unavailable (Boris and Zeanah 2005).

Epidemiology

It is rather difficult to estimate the true prevalence of RAD and DSED because there is paucity of systemic data derived from the general population. Moreover, clinically validated data are not always available on neglected, abused, or institutionalized children. The prevalence of RAD has been estimated at 1% of all children under the age of 5 (Richters and Volkmar 1994). Obviously, the prevalence of RAD and DSED is elevated among high-risk populations, and several researchers reported that 30–40% of children, who entered foster care or were institutionalized at a young age, manifest significant symptoms pertaining to RAD or DSED (Rutter et al. 2004; Smyke et al. 2002; Zeanah et al. 2002, 2004, 2016).

Natural History, Prognostic Factors, and Outcomes

Children who are abused or neglected early in life are at risk for RAD and DSED. These risk groups may include children whose parents are alcohol or substance abusers or mentally ill because these parents are most likely not able to respond to the child's physical or emotional needs in a reliable and sensitive way. Those young children who are most severely neglected, physically or sexually abused, or abandoned are usually placed in foster care. These children are

at an even greater risk for the development of RAD or DSED. Earlier timing, increased severity, and prolonged duration of the period of maltreatment or institutionalization (specifically beyond the age of 6 months) all increase the risk for RAD or DSED. Yet, not necessarily all children who are neglected or abused will present this disorder.

Not much literature is available on the course of symptoms in older children and adolescents or on the outcomes and natural course over time. The only systematic data source is from the Bucharest Early Intervention Project. In this clinical trial, children in institutions were randomly selected to be placed in foster care and followed throughout childhood. The data from this study suggests that the recovery paths for the two disorders appear to differ. Institutionalized children who were later placed into more normative settings (adoption or foster care) frequently exhibit difficulties in attachments with the new caregivers, but they almost never meet the criteria for RAD. Nevertheless, indiscriminate social behaviors associated with DSED tend to persist over the years, even after significant improvement in the caregiving environment, the establishment of preferred and even secured attachment with new caretakers, and adequate functioning levels in everyday life (O'Connor and Rutter 2000; Rutter et al. 2007a; Zeanah 2000; Zeanah and Smyke 2008).

It might be the case that pathogenic caregiving that occurs at a critical time point leads to the development of DSED, and once it has developed, it becomes inherent in the child, even if later on, secure attachment is formed with specific caregivers who are now available to the child. Rutter et al. (2004) suggested that a form of biological programming, altering brain structure and functioning as an adaptive reaction to the environmental circumstances, occurs in children who experienced severe deprivation at a sensitive period of development (before the age of 6 months). The strong persistence of these unique behavioral patterns into middle childhood/early adolescence, as well as the effect of the timing and duration of deprivation, supports this assumption.

Clinical Expression and Pathophysiology

RAD and Attachment Classifications

As opposed to RAD or DSED, attachment classifications (secure vs. insecure) within the attachment literature (Ainsworth et al. 1978; Bowlby 1988) are not considered clinical entities or psychopathologies but rather developmental risk or protective factors. Attachment classifications for young infants and children are available in the context of a relationship with a primary caregiver. In contrast, RAD and DSED are clinical entities describing children's personality traits, uncoupled from a relationship with the primary caregiver (Zeanah and Smyke 2008).

RAD and DSED are the only diagnostic entities in the *DSM-5* that specifically relate to attachment. However, limiting possible disordered attachment patterns to the rather specific diagnostic criteria of a disorder may underestimate the clinical conditions related to attachment disturbances in general. There are diverse behavioral manifestations and diagnoses in addition to RAD or DSED that may be associated with, and at times attributed to, attachment disturbances (e.g., conduct disorder, anxiety disorders) and can be effectively treated with attachment-based therapeutic approaches. The current focus on rather narrow diagnostic criteria, which does not include conceptualization of attachment-related issues, leads to treatments that may address, and temporarily reduce, symptoms rather than provide treatment for the underlying pathology and deficits associated also with the underlying attachment history (Hardy 2007).

Evaluation and Differential Diagnosis

A childhood history of severe adversity is associated with elevated risk for psychopathology and developmental delays; therefore the comorbidity of RAD/DSED with other disorders is highly likely to occur. However, some conditions are exclusive and should be distinguished from RAD/DSED.

Developmental delays and language disorders that are also associated with early environmental

deprivation may be experienced by children diagnosed with RAD/DSED. Similar to RAD, some of these delays are treatable, and after improvement in the environment, some of the gaps are bridged (Boris and Zeanah 2005). Developmental or language delay and RAD/DSED are not exclusive diagnoses, but RAD/DSED diagnosis should not be assigned if the clinical picture can be alternatively explained by developmental or language delays. An important distinction is that children with delays do not usually present with difficulties in emotion regulation and social relatedness.

RAD and DSED may co-occur with medical conditions that are associated with neglect and maltreatment such as failure to thrive, growth disturbances, and other undiagnosed medical conditions that require treatment.

The possible association between institutionalization and autism was introduced by the Romanian adoption studies. Rutter and his team reported on an autistic behavioral pattern found in more than 10% of the children adopted out of Romanian institutions (Rutter et al. 1999, 2007b). This behavioral pattern resembled the behavioral characteristics of autism: social and communicational difficulties or abnormalities and stereotyped behaviors. However, the behavioral characteristics in this group of children changed and sometimes diminished over time and therefore were defined as "quasi-autism." These children were also reported to present disinhibited attachment behaviors, emotional problems, peer problems, inattention/overactivity, and cognitive impairment. Rutter et al. reported that as the children who were diagnosed with quasi-autism grew older, some of their difficulties seemed to have more in common with DSED than with autism. However, although there was a substantial association between quasi-autism and DSED, and for some children the latter dominated at age 11, the autistic features still persisted to some degree. Rutter et al. suggested that the early deprivation might cause alterations and deficits in social cognition, manifesting in disinhibited social behavior and in "quasi-autism," and emphasize the potential benefit of interventions aimed to improve social skills and develop healthy, close relationships

for post-institutionalized children in general (Rutter et al. 2007a, 2010). RAD, DSED, and ASD are exclusive diagnoses. ASD usually occurs in adequate caregiving environments and is not a result of pathogenic care, whereas RAD and DSED do not typically occur in adequate caregiving environments. Symptoms of RAD tend to dramatically improve after improving the environment, whereas ASD symptoms are more resistant. Additionally, the deficits in imaginative play, deviant language development (e.g., echolalia), deficient initiation, or response to joint attention do not typically appear in RAD/DSED but are rather common in ASD (Boris and Zeanah 2005).

DSED, characterized by deficits as social inhibitory control, is associated with attention deficit and hyperactivity disorder (ADHD) symptoms and deficits in cognitive inhibitory control, but these disorders usually occur independently of each other with different underlying deficits (Gleason et al. 2011). In ADHD, the impulsivity is usually behavioral and/or cognitive, but not always social, whereas in DSED it is always social.

Posttraumatic stress disorder (PTSD) has also been documented in maltreated and institutionalized children. However, the association between PTSD and RAD has not been officially documented. It has been suggested that RAD overlaps with and perhaps might be better conceptualized as PTSD or acute stress disorder (Boris and Zeanah 2005).

Assessment Methods

The AACAP published a practice parameter with guidelines for the diagnosis and treatment of RAD (Boris and Zeanah 2005). This parameter was updated in 2016 and includes guidelines for assessment of children with suspected RAD/DSED (Zeanah et al. 2016). First, screening for RAD and DSED in children with a history of extreme neglect or foster care is recommended. There is no valid screening measure, and the guideline for assessing risk is to inquire about the child's attachment behaviors with familiar and non-familiar adults and about the child's history.

An evaluation for RAD or DSED must include structured observations of the child interacting with primary caregivers, as well as with strangers. The detailed history of the child's early attachment behavior and caregiving environment, obtained from several resources, is necessary as well. It is also important to gain information about the child's behavior in different settings (i.e., home, school/early learning setting). An interaction with a stranger is necessary for assessing DSED. If any suspicion regarding current or previous maltreatment arises, it should be reported to the responsible authorities. Additionally, children with RAD or DSED need further evaluation for developmental delays and language disorders as well as a pediatric examination for medical conditions that might be undiagnosed and untreated. A psychiatric evaluation is necessary in order to assess comorbid conditions.

Marvin and Whelan (2003) suggest a complementary approach for an assessment protocol of RAD. In their work, they combine terminology of the four attachment patterns with the *DSM* criteria and *ICD-10* coding. This protocol includes standardized assessment tools, clinical open-ended interviews with the parents and child, and clinical observation of the parent-child interaction in free play and in the strange situation (Ainsworth and Bell 1970). The revised practice parameter also includes an outline for a possible clinical observational assessment of attachment that may be used in the evaluation of children with RAD/DSED (Zeanah et al. 2016).

Treatment

The most important intervention for children with RAD and DSED is to provide the child with emotionally available attachment figures. Unfortunately, adoption by a healthy, good-enough new family is not always possible. However, two alternative strategies toward achieving this goal were reported to effectively improve signs of RAD in institutionalized children. The first approach includes changes and interventions within institutions, and the other includes placement of the

children into foster care, accompanied with supervision and guidance for the caregivers.

It has been demonstrated in two studies that within-institution changes and interventions can result in significant improvement of attachment disorders. In one study by McCall and colleagues (as reviewed in Zeanah and Smyke 2008), an intervention designed to change the quality of institutional caregiving was conducted. Two interventions were implemented: staff training focused on more sensitive caregiving as well as structural changes to decrease the number of caregivers per child. Three institutions were included in the study: in one, both staff training and structural changes were implemented; in a second, only staff training was provided; and in the third, no intervention was implemented. Results indicated that children whose institution received the two interventions displayed substantially more proximity-seeking and contact-maintaining attachment behavior and less avoidant attachment behavior with their caregivers compared to children in the other two groups.

Smyke et al. (2002) conducted a similar interventional study in an institution in Romania. Usually, in these institutions, the staff turnover is high and children have many different caregivers during the week. In the “pilot” units, structural changes were made in order to reduce the number of caregivers working with each child, without changing the overall number of caregivers. The researchers examined signs of RAD and DSED in children receiving the “standard care” versus children on a “pilot” unit as well as children living with their parents and attending childcare facilities. Smyke et al. reported significant differences among the groups, with consistently more signs of both RAD and DSED in children on the standard unit. Interestingly, the staff’s behavior on the “pilot” units changed as well in that the staff demonstrated more personalized and affective attitudes and was more psychologically invested in the children in their group compared to caregivers on the standard care unit. This suggests that focusing caregivers on a specific group of children may have positive effects on both caregivers and children.

As abovementioned, a randomized controlled trial study of foster care as an alternative to institutional care was implemented in the Bucharest Early Intervention Project (Zeanah et al. 2003). This project included 136 children with half of the children randomly assigned to foster care and 68 children to continued institutional care. The children were followed longitudinally through 54 months of age. Seventy-two family-reared children recruited from pediatric clinics in Bucharest were also included in the study as a comparison group. Local foster parents were recruited through advertisement and were trained using a manual that had been developed by Romanians and for Romanians. Three project social workers were trained to provide a variety of services to foster parents and the children they cared for, focusing on the needs of the children for stable, consistent, and emotionally available caregivers. The aim was to have the foster parent become emotionally invested in the child as if he or she was their own child. Results of this study indicated that signs of RAD were reduced substantially by placement in foster care, and the response to placement was early and sustained. Over time, the symptoms of RAD in the foster care group became indistinguishable from those in the community comparison group. However, signs of DSED responded to placement more modestly.

Most of the studies and documentations of RAD/DSED include children who were institutionalized. In the community or in situations in which there are parents or defined caregivers who can be considered as emotionally available, a systemic approach is needed. Parenting style and its “goodness of fit” with the child’s temperament and needs should be assessed. The recommended treatment will be attachment-based therapy, aiming to enhance positive child-caregiver interactions and to coach and teach the caregiver to reflect on the child’s feelings and needs and to respond sensitively. Dyadic therapy with the caregiver and child together is recommended as well, if possible. A family-based wider approach that involves other family members can enhance and broaden the positive achievements accomplished in dyadic therapy. Additional interventions

targeting comorbid conditions as behavioral difficulties and developmental and language delays are also recommended (Zeanah et al. 2016).

Basically, individual therapy with the child without collaboration with the caregivers is insufficient for the treatment of RAD/DSED (Boris and Zeanah 2005). It has been clearly stated by the American Psychiatric Association and the AACAP that nontraditional coercive treatments, based on noncontingent physical restraint or coercion (“holding,” “rebirthing therapy,” and more), do not have any empirical support and have been associated with serious harm and even death. Therefore, these treatments for children with RAD, DSED, or other attachment or behavioral disorders are contraindicated or even considered to be maltreatment (Boris and Zeanah 2005; Chaffin et al. 2006; Zeanah et al. 2016).

There is no specific pharmacological treatment for RAD. Psychopharmacologic agents may be used to address secondary difficulties and problems such as hyperactivity and difficulty focusing or sleeping but are not indicated for the core features of RAD or DSED.

See Also

- [Attachment](#)
- [Attachment Disorder](#)

References and Reading

- AACAP. (2003). AACAP Task Force on Research Diagnostic Criteria: Infancy Preschool Age. Research diagnostic criteria for infants and pre-school children: the process and empirical support. *Journal of the American Academy of Child and Adolescent Psychiatry*, 42, 1504–1512.
- Ainsworth, M. D., & Bell, S. M. (1970). Attachment, exploration, and separation: Illustrated by the behavior of one-year-olds in a strange situation. *Child Development*, 41(1), 49–67.
- Ainsworth, M. D., Blehar, M., Waters, E., & Wall, S. (1978). *Patterns of attachment*. Hillsdale: Lawrence Erlbaum Associates.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR* (4th ed.). Washington, DC: Author.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Balbernie, R. (2010). Reactive attachment disorder as an evolutionary adaptation. *Attachment & Human Development*, 12(3), 265–281.
- Boris, N. W., & Zeanah, C. H. (2005). Practice parameter for the assessment and treatment of children and adolescents with reactive attachment disorder of infancy and early childhood. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44(11), 1206–1219.
- Bowlby, J. (1988). *A secure base*. London: Routledge.
- Chaffin, M., Hanson, R., Saunders, B. E., Nichols, T., Barnett, D., Zeanah, C., et al. (2006). Report of the APSAC task force on attachment therapy, reactive attachment disorder, and attachment problems. *Child Maltreatment*, 11(1), 76–89.
- Gleason, M. M., Fox, N. A., Drury, S., Smyke, A., Egger, H. L., Nelson, C. A., et al. (2011). Validity of evidence-derived criteria for reactive attachment disorder: Indiscriminately social/disinhibited and emotionally withdrawn/inhibited types. *Journal of the American Academy of Child and Adolescent Psychiatry*, 50(3), 216–231.
- Hardy, L. T. (2007). Attachment theory and reactive attachment disorder: Theoretical perspectives and treatment implications. *Journal of Child and Adolescent Psychiatric Nursing*, 20(1), 27–39.
- Marvin, R. S., & Whelan, W. F. (2003). Disordered attachments: Toward evidence-based clinical practice. *Attachment & Human Development*, 5(3), 283–288.
- Minde, K. (2003). Assessment and treatment of attachment disorders. *Current Opinion in Psychiatry*, 16(4), 377–381.
- O'Connor, T. G., & Rutter, M. (2000). Attachment disorder behavior following early severe deprivation: Extension and longitudinal follow-up. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(6), 703–712.
- Richters, M. M., & Volkmar, F. R. (1994). Reactive attachment disorder of infancy or early childhood. *Journal of the American Academy of Child and Adolescent Psychiatry*, 33(3), 328–332.
- Rutter, M., Andersen-Wood, L., Beckett, C., Bredenkamp, D., Castle, J., Groothues, C., et al. (1999). Quasi-autistic patterns following severe early global privation. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 40(4), 537–549.
- Rutter, M. L., Kreppner, J. M., & O'Connor, T. G. (2001). Specificity and heterogeneity in children's responses to profound institutional privation. *The British Journal of Psychiatry*, 179(2), 97.
- Rutter, M., O'Connor, T. G., & English and Romanian Adoptees Study Team. (2004). Are there biological programming effects for psychological development? Findings from a study of Romanian adoptees. *Developmental Psychology*, 40, 81–94.

- Rutter, M., Colvert, E., Kreppner, J., Beckett, C., Castle, J., Groothues, C., et al. (2007a). Early adolescent outcomes for institutionally-deprived and non-deprived adoptees. I: Disinhibited attachment. *Journal of Child Psychology and Psychiatry*, 48(1), 17–30.
- Rutter, M., Kreppner, J., Croft, C., Murin, M., Colvert, E., Beckett, C., et al. (2007b). Early adolescent outcomes of institutionally deprived and non-deprived adoptees. III. Quasi-autism. *Journal of Child Psychology and Psychiatry*, 48(12), 1200–1207.
- Rutter, M., Sonuga-Barke, E., Beckett, C., Castle, J., Kreppner, J., Kumsta, R., et al. (2010). Deprivation-specific psychological patterns: Effects of institutional deprivation. *Monographs of the Society for Research in Child Development*, 75(1), 1–252.
- Smyke, A. T., Dumitrescu, A., & Zeanah, C. H. (2002). Attachment disturbances in young children. I: The continuum of caretaking casualty. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(8), 972–982.
- World Health Organization. (1992). *International statistical classification of diseases and related health problems*. Geneva: Author. 10th revision.
- Zeanah, C. H. (2000). Disturbances of attachment in young children adopted from institutions. *Journal of Developmental and Behavioral Pediatrics*, 21(3), 230–236.
- Zeanah, C. H., & Smyke, A. T. (2008). Attachment disorders in family and social context. *Infant Mental Health Journal*, 29(3), 219–233.
- Zeanah, C. H., Smyke, A. T., & Dumitrescu, A. (2002). Attachment disturbances in young children. II: Indiscriminate behavior and institutional care. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(8), 983–989.
- Zeanah, C., Nelson, C. A., Fox, N. A., Smyke, A. T., Marshall, P. J., Parker, S., et al. (2003). Effects of institutionalization on brain and behavioral development: The bucharest early intervention project. *Development and Psychopathology*, 15, 885–907.
- Zeanah, C. H., Scheeringa, M., Boris, N. W., Heller, S. S., Smyke, A. T., & Trapani, J. (2004). Reactive attachment disorder in maltreated toddlers. *Child Abuse & Neglect*, 28(8), 877–888.
- Zeanah, C. H., Cheshier, T., Boris, N. W., Walter, H. J., Bukstein, O. G., Bellonci, C., . . . Hayek, M. (2016). Practice parameter for the assessment and treatment of children and adolescents with reactive attachment disorder and disinhibited social engagement disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 55(11), 990–1003.
- Zero to Three. (2005). *Diagnostic Classification of mental health and developmental disorders of infancy and early childhood: (DC:0-3R)*. Washington, DC: Author. Revised edition.

Readiness to Learn

► Optimal Arousal

Reading

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Definition

Reading is a complex skill by which one extracts meaning from print. Reading research has identified successful understanding of what is read or reading comprehension, as largely requiring two fundamental skills: decoding (i.e., word recognition) and language comprehension. Referred to as the simple view of reading by Gough and Tunmer (1986), reading comprehension is considered to be the product of decoding and language comprehension. If either or both of these skills are weak, text comprehension suffers; that is, neither alone is sufficient to support reading comprehension. The success of reading comprehension is further determined by the coordination of these two components so that the process is automatic and accurate (i.e., fluent) (Scarborough 2001).

Historical Background

Over the last century, our understanding of reading has developed from a narrow view to one that recognizes the numerous factors that converge in reading. Early in the twentieth century, reading was thought to be simply the oral recitation of printed words; the ability to read was discussed in terms of systematic phonics, while little attention was given to comprehension skills. A few years later, a shift in educational pedagogy occurred in which learning to read by knowing

the sounds associated with letters (phonics) transitioned into expectations for whole-word reading (sight words); yet there still was little attention to reading as more than that. Approaching the middle of the twentieth century, it became more apparent to educators that reading development required knowledge beyond word reading alone, such as language, experience, attention, vocabulary, thinking, and even instruction. Today, it is agreed that reading requires being actively engaged in the convergence of processes such as phonological awareness (i.e., the ability to recognize and manipulate the sounds in words), memory, and retrieval with language and comprehension strategies.

Current Knowledge

Reading Components

Reading is a process that requires decoding and language comprehension with a degree of fluency. With instruction, practice, and motivation, the typical learner builds skills upon skills, gradually moving toward being an independent, proficient reader of multiple genres.

Decoding

Decoding is the ability to read words, including both real and nonsense words. It requires the application of knowledge of letter-sound relationships (i.e., phonics), including knowledge of letter patterns (i.e., orthography). Decoding skills account for most of the success in comprehension skills, especially in learning to read and early reading. Phonological processing skills account for most of the development of decoding skills. Phonological processing skills include phonological awareness, phonological memory, and phonological retrieval (naming). Phonological awareness is the ability to recognize that words are made of sounds (analysis) and to be able to manipulate those sounds in tasks such as rhyming words and blending sounds into words (synthesis). Translating the letters of a printed word into corresponding sounds (phonological codes) that are then stored with meaning in

memory is referred to as phonological memory. Phonological naming is the quick retrieval of these phonological codes from permanent memory, for example, names of common objects, colors, digits, or letters. When reading, efficient retrieval of phonological codes associated with letters, word segments, and whole words affects the success with which phonological information may be used in decoding (Wagner et al. 1997).

At about the same time that a child develops early phonological awareness skills, the alphabetic principle is recognized, that is, letter-sound associations are learned (i.e., phonics). With instruction and practice, a sight word bank is then developed using phonological memory, that is, coding information (making letter to sound associations) for temporary storage of that information so that the reader maintains an accurate representation of the phonemes associated with letters or parts of words while devoting cognitive resources to ongoing decoding and comprehension processes (Wagner et al. 1997). Sight words then are not words that are memorized through visual skills but instead are words that have been rooted in memory through initial decoding (analyzing the letters and attaching sounds to those letters) (Ehri 1995). Thus, in sight word reading, the words are not read by sounding out letters but instead have been anchored to their pronunciation in memory so they are quickly and accurately read (i.e., retrieved from memory) (Ehri 1998).

Well-developed knowledge of phonics enables the reader to decode unfamiliar words, thus ever increasing the store of available sight words. Children move through phases of sight word learning: prealphabetic phase in which a word is recognized by purely visual cues, partial alphabetic phase in which a word is accessed by remembering letter-sound associations of salient letters in that word, full alphabetic phase in which all letters and sounds are matched in a word, and consolidated alphabetic phase in which multi-letter patterns (e.g., “igh”) are also matched.

Decoding skills should be measured in tasks that require reading both real and nonsense words; however, measuring the ability to read nonsense

words provides a truer assessment of phonological skills and phonics because the meaning/recognition of words is removed. Decoding must not only be accurate but also automatic. That is, letter-sound associations must be quick. Oral reading of word lists and connected passages allows for insight in a student's word identification strategies and degree of fluency. Through oral reading, for example, the student may become aware of word reading problems and seek to correct any errors using phonological awareness and/or phonics skills.

Language Comprehension

Language comprehension or the ability to construct meaning from words whether spoken or written requires two kinds of knowledge: linguistic knowledge and background knowledge, each is somewhat dependent on the other. Linguistic knowledge is the knowledge of the formal structures of a language, while background knowledge is the knowledge of the world, including content and procedural knowledge acquired through exchanges with one's surroundings. Together, these knowledge bases enable one to draw inferences, thus allowing for interpretations beyond the literal.

Linguistic knowledge may be described as an understanding of those domains upon which competency in a language depends: phonology, semantics, and syntax. Phonology is defined as the sound structure of a language, semantics as the meaning component of a language (words and their meaningful parts, i.e., vocabulary), and syntax being the rules of language that dictate how to combine words to form sentences.

Differently, background knowledge is the understanding of how events work based on prior interactions with the environment; this background knowledge provides for the development of schemata. A schema is a structure that one uses both to organize current knowledge and to understand new information and experiences. In communicating through language, a well-developed schema in a particular domain of knowledge allows for understanding of even new information relevant to that domain because there is already a meaningful structure in place for interpreting the information.

Reading comprehension requires making sense of the printed words that have been translated into language (decoding). That is, reading comprehension requires language comprehension to draw upon both linguistic information and background knowledge so that in reading one may go beyond what is explicitly stated, link up ideas within the text, and derive meaning from what has been read. Of final importance though is that comprehension of print also requires literacy knowledge, that is, a familiarity with story structures and genres (Scarborough 2001).

Language comprehension is classically measured through oral language tasks. These include but are not limited to measures of receptive vocabulary, grammatical understanding, and discourse comprehension. Specific to the last, it is often suggested that this be assessed by a listening comprehension task using age-appropriate passages from an informal reading inventory. After listening to spoken passages, the student is asked questions targeting both information that was explicitly presented in the passage and that which was only inferred to in the passage.

Fluency

Although reading comprehension is determined largely by abilities in decoding and language comprehension, fluency is also a necessary component. That is, once a child can decode and has the necessary language skills, fluency should follow. To be fluent means decoding is fast and accurate (i.e., automatic), and reading is delivered with appropriate intonation. Fluency may be compromised in one or several ways. An individual may be fast but inaccurate. Occasional errors are realistic (at 99% accuracy, one is considered to be at an independent level, 95–98% instructional reading level). Although errors are acceptable even for fluent reader, too many errors if not caused by an attentional difficulty are typically caused by a lack of automaticity in decoding. Lack of automaticity in decoding may be due to a lack of skills in phonological awareness (e.g., a difficulty blending sounds even if one knows the letter-sound associations) or a lack of letter-sound knowledge (what sound does/g/make). An

individual can be slow but accurate. In terms of rate, for example, at the end of first grade, a child is in the 50th percentile if orally reading 53 correct words per minute; at the end of eighth grade, a student is in the 50th percentile if orally reading at 151 words per minute (Hasbrouck and Tindal 2006). Finally, an individual can be both slow and inaccurate. Particular to inflection and prosody, matching the stress and intonation of the text reflects correct on-line monitoring of what is being read, for example, use of a sad tone spoken by the character or a rising cadence as a reaction of the reader to a surprising statement.

Stages of Reading

Reading is a multifaceted process and as such requires systematic, organized instruction in the development of each of the stages of reading (Chall 1996): Stage 0 is referred to as the pre-reading stage, stage 1 as the initial reading or decoding stage, stage 2 as the confirmation stage, stage 3 as the reading for learning the new stage, stage 4 as multiple viewpoints stage, and stage 5 as the construction and reconstruction stage.

Beginning with stage 1, the stages of reading may be divided into those in which the child is learning to read and those in which the child is reading to learn. In learning to read, the characteristics of children reading in stages 1 and 2 (typically seen in grades 1, 2, and 3) include the ability to read simple, familiar texts and use the alphabetic principle to decode unfamiliar words. Differently beginning at stage 3 (typically grades 4–8), texts contain new words and ideas beyond the child's linguistic and background knowledge; therefore, there must be fluency in word recognition and critical thinking outside the child's knowledge. The transition from stage 2 to 3 is critical for academic success here on. In stage 4 (typically high school), students are expected to use strategies to be able to see different perspectives and to work with additional layers of facts and concepts beyond what was learned earlier. Reading in stage 5 (typically college) demands the construction of meaning at a high level of abstraction, integrating information across settings with a purpose.

Reading Disabilities

The simple view of reading suggests not only a way of defining reading ability but may also be used to classify a reading disability (Catts et al. 2006). The simple view presumes that once the printed matter is decoded, the reader applies to the text exactly the same mechanisms (linguistic knowledge and background knowledge) that would be used on its spoken equivalent. Gough, Hoover, and Peterson (1996) suggest that beyond the point of word recognition, both listening and reading appear to require essentially the same processes.

Using the simple view as a framework for classification of reading and reading disabilities, four types of readers emerge when decoding and language comprehension are measured:

1. Good readers or those with good decoding and good language comprehension
2. Dyslexic readers or those manifesting only a decoding difficulty
3. Poor comprehenders (or at the extreme, hyperlexic readers) or those exhibiting only a language comprehension difficulty
4. Mixed deficit poor readers or those demonstrating difficulties in both decoding and language comprehension

The use of this classification system which is based on the two fundamental components of reading has direct implications for both assessment and subsequently intervention. That is, effective intervention must be preceded by assessment of both decoding and language comprehension, neither of these should be assumed to be fully developed despite the age of the child. Then intervention focusing on improving specific areas of need, decoding and/or language comprehension should be carried out.

Heterogeneous Nature of Reading in Autism

There is a general belief that reading is a relative strength for individuals with autism spectrum disorders (ASD) (Nation et al. 2006). This assumption is based upon research and clinical observations which have reported many of those with ASD to have the ability to accurately read

aloud single words presented out of context; sometimes this ability is even beyond that expected by IQ. However, reading is not only this single process of word recognition but instead is the integration of two processes, decoding (which allows for word recognition and word identification) and language comprehension (Gough and Tunmer 1986). For many individuals with ASD, there is a disassociation between the two constructs of reading, decoding and language comprehension. This is because many of these individuals have language impairments, thus even with good word reading skills, their reading comprehension is poor.

Decoding in Individuals with ASD

Despite the notion that decoding is a relative strength in individuals with ASD, there is much variability in decoding accuracy. Decoding ability is measured by both word and nonword reading accuracy. Studies have found individuals with ASD to be able to read both real words and nonsense words, the first reflecting good word recognition, the latter, good underlying phonological skills. Yet it has also been found that some individuals with ASD, though relatively skilled at reading real words, are quite poor at reading nonsense words, perhaps considerably lower than the level expected given their ability in real word reading. This discrepancy suggests their real word reading is based on a visual strength (rote visual memory or association) as they lack the phonological skills (“sounding out” using letter-sound associations) to read nonwords. (Note that phonological skills are part of typical reading development.) Finally, there are those with ASD who do not evidence adequate measureable single word reading, either real or nonwords. Thus, within the population of individuals with ASD, some read both real words and nonwords accurately, some read real words but are unable to decode nonwords, and others are poor at both real and nonwords.

In discussing word reading accuracy, it is important to include mention of hyperlexia. Hyperlexia is a reading profile in which there is exceptionally well-developed word reading yet very poor reading comprehension in individuals

with marked cognitive and linguistic deficits; an early onset of preoccupation with reading is also evident. Strong skills in reading nonsense words as well as real words suggest that individuals with hyperlexia use phonological decoding strategies as typical readers do, and do not rely on visual memory or whole-word recognition (Newman et al. 2007). Yet despite this remarkable word reading, these individuals have significant difficulty understanding connected language (read or heard); thus, hyperlexia is a disorder of comprehension. In fact, though reading fluency in those with hyperlexia has been found to be excellent despite content, when it becomes necessary to stop and comprehend, fluency significantly decreases (Newman et al. 2007). As these reading, cognitive, and oral language characteristics tend to come together in individuals with autism, patterns of hyperlexic reading are therefore more frequent in this group (Nation et al. 2006).

Language Comprehension in Individuals with ASD

Despite adequate levels of word reading accuracy (i.e., decoding) in the population of individuals with ASD as a whole, poor reading comprehension is typical. Therefore, in keeping in mind the two components of reading, decoding (i.e., word reading) and language comprehension, it is the latter with which this population most often struggles. Specifically, impairments in vocabulary and oral language comprehension are typically measured and thus are associated with reading comprehension difficulties. However, as is the case with decoding, language abilities and difficulties vary in the population. There may be, for example, a general difficulty with integrating information read, difficulty understanding anaphoric references (linking pronouns to their antecedent nouns in the text), difficulty with using prior knowledge to make connections with new information read or difficulty with comprehension monitoring (Nation et al. 2006). Further, there may be a discrepancy between understanding explicit (i.e., literal) versus implicit (inferential) information: For those with ASD, the former being easier than the latter (a higher-level language skill). Additionally, difficult to comprehend

are words with multiple meanings figurative language, humor, as well as characters' motives, their goals, and their internal states (e.g., emotions).

Importantly, however, despite the large number of individuals with ASD who have relative strengths in word recognition and therefore reading comprehension difficulties stemming only from language difficulties, there are individuals with ASD for whom impairment in reading comprehension is due not only to language comprehension weaknesses but also, at least in part, to poor word reading accuracy. That is, these individuals are unable to recognize or decode the written word.

Reading Fluency in Children with ASD

As in typically developing children, the oral reading of those with ASD will vary as a function of word reading skills (decoding). That is, accuracy and rate will fall in line with decoding skills. However, for those with ASD, expression may be poor. Reading may lack appropriate pauses, changes in tone that reflect emotions of the characters, and/or necessary emphasis of words critical to meaning of the text. These deficits are thought to be associated with the more general deficits of social skills and pragmatic language development which are characteristic of ASD (Henry 2010).

Reading Assessment and Intervention in Children with ASD

Given that reading is primarily the product of decoding and language comprehension, assessment must comprehensively investigate each of these factors, that is, identifying both strengths and weaknesses in each area. Criterion-referenced instruments along with, as appropriate, standardized tests should be used to measure a learner's reading patterns.

Decoding refers to the word recognition processes that analyze the letters of a written word, transforming print to pronunciation of that word. Processes such as phonological awareness and knowledge of letter-sound associations (referred to as the alphabetic principle) are necessary for decoding unfamiliar words. Careful identification of decoding skills of the learner establishes areas

of need and sets the baseline for intervention in word reading.

However, good performance in decoding does not guarantee adequate reading comprehension. Especially when supporting those with ASD, dependence on assessments of word reading alone may overestimate reading comprehension as a strength. Autism and language are well connected, and language comprehension is necessary for reading; further, oral language abilities and reading development of students with ASD have been found to have a bidirectional relationship (i.e., improvement in one improves the other) (Lanter and Watson 2008). Therefore, the assessment of the learner's oral language skills using criterion-referenced instruments along with, as appropriate, standardized tests is necessary even if the individual has adequate or extraordinary single word reading (decoding). Keeping in mind that responses to comprehension questions should reflect the reader's relationship with the text, assessment and instruction should include not only answering literal questions (facts from the passage read) but questions that encourage inferencing, connecting prior knowledge with the author's meaning, and interpreting meaning.

Converging research has identified areas associated with reading achievement. These areas are decoding including phonological awareness, reading fluency, language, and comprehension strategies. Reading is not a single construct, and in those with ASD, reading components usually have dissociated. General reading instruction and intervention recommendations for children with ASD cannot be made; instead given the heterogeneous nature of reading skills in learners with ASD, individualized reading assessments should lead to appropriate reading instruction. It can be said, however, that best practice in reading instruction for children with ASD includes the use of rich and meaningful contextualized examples presented multiple times.

Future Directions

The National Reading Panel (2000) summarizes the future direction of reading as follows: Though

the literature in reading research is significant and converges into several key areas (e.g., phonological awareness is necessary for decoding), the need for continued research using rigorous experimental or quasi-experimental design that investigates reading instruction is clear if reading education is not to fall prey to educational fads. Additionally, today, technology is changing education. Research into its use as a tool in the teaching and learning of reading has been limited thus far and in much need.

See Also

- [Decoding Skills](#)
- [Emergent Literacy Skills of Preschoolers with Autism](#)
- [Literacy](#)

References and Reading

- Catts, H. W., Adlof, S. M., & Weismer, S. E. (2006). Language deficits in poor comprehenders: A case for the simple view of reading. *Journal of Speech Language and Hearing Research*, 49, 278–293. [https://doi.org/10.1044/1092-4388\(2006\)023](https://doi.org/10.1044/1092-4388(2006)023).
- Chall, J. S. (1996). *Stages of reading development* (2nd ed.). Fort Worth: Harcourt Brace.
- Ehri, L. C. (1995). Phases of development in learning to read words by sight. *Journal of Research in Reading*, 18, 116–125. <https://doi.org/10.1111/j.1467-9817.1995.tb00077>.
- Ehri, L. C. (1998). Research on learning to read and spell: A personal-historical perspective. *Scientific Studies of Reading*, 2, 97–114.
- Gough, P., & Tunmer, W. (1986). Decoding, reading, and reading disability. *Remedial and Special Education*, 7, 6–10.
- Gough, P. B., Hoover, W. A., & Peterson, C. L. (1996). Some observations on a simple view of reading. In C. Cornoldi & J. Oakhill (Eds.), *Reading comprehension difficulties* (pp. 1–13). Mahwah: Erlbaum.
- Hasbrouck, J., & Tindal, G. A. (2006). Oral reading fluency norms: A valuable assessment tool for reading teachers. *The Reading Teacher*, 59(7), 636–644.
- Henry, K. A. (2010). *How do I teach this kid to read?: Teaching literacy skills to young children with autism, from phonics to fluency*. Arlington: Future Horizons.
- Lanter, E., & Watson, L. R. (2008). Promoting literacy in students with ASD: The basics for the SLP. *Language, Speech, and Hearing Services in Schools*, 39, 33–43. [https://doi.org/10.1044/0161-1461\(2008\)004](https://doi.org/10.1044/0161-1461(2008)004).
- Nation, K., Clarke, P., Wright, B., & Williams, C. (2006). Patterns of reading ability in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 36, 911–919. <https://doi.org/10.1007/s10803-006-0130-1>. http://www.nationalreadingpanel.org/press/press_rel_langenberg.htm.
- Newman, T., Macomber, D., Naples, A. J., Babitz, T., Volkmar, F., & Grigorenko, E. L. (2007). Hyperlexia in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 760–774. <https://doi.org/10.1007/s10803-006-0206-y>.
- Scarborough, H. (2001). Connecting early language and literacy to later reading (dis)abilities: Evidence, theory, and practice. In S. B. Neuman & D. Dickinson (Eds.), *Handbook of early literacy research* (pp. 97–110). New York: Guilford Press.
- Wagner, R. K., Torgesen, J. K., Rashotte, C. A., Hecht, S. A., Barker, T. A., Burgess, S. R., et al. (1997). Changing relations between phonological processing, abilities and word level reading as children develop from beginning to skilled level readers: A 5-year longitudinal study. *Developmental Psychology*, 33, 468–479.

Reading Comprehension Disorder

- [Hyperlexia](#)

Reading Comprehension Impairments in School-Aged Children with HFA

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Definition

Children and adolescents with autism spectrum disorder (ASD) who are not affected by intellectual disability (ID) are often referred to as having higher-functioning autism (HFA). Many of these students spend substantial portions of their school day in general education settings where the ability to competently read, comprehend, and use written texts is critical for accessing the core curriculum

in science, social studies, language arts, and, increasingly, mathematics. Reading comprehension is a foundational learning mechanism and is intertwined with all literacy skills, including critical thinking, writing, listening, and following directions. Furthermore, reading can provide a portal into fictional worlds where relationships, emotions, and adventures are detailed and available for consumption at one's own pace and for rereading as often as desired. Therefore, developing proficient reading comprehension skills should be an essential goal for students with HFA.

Historical Background

Comprehending what one reads requires coordinated and efficient processing of many key components. The Simple View of Reading (SVR; Gough and Tunmer 1986) is a commonly used framework to explain the separate skills that underlie reading comprehension. According to this model, word recognition skills (decoding and sight word reading) and listening comprehension skills both make independent, and necessary, contributions to proficient reading comprehension. If a reader struggles with either of these main components, they will have difficulty accessing written texts. Developmentally, reading for meaning progresses over time and builds upon two key abilities already present in infancy: the ability to visually recognize objects and oral language skills. By the time children are 5 or 6 years old and entering elementary school, key visual recognition processes are well developed and most have expert oral language knowledge of phonology (sounds of language), basic grammar rules, and a vocabulary of several thousand words. As children learn to read, they develop letter-sound correspondence by connecting the sounds they hear in spoken language to the visual symbols, or letters, that represent them and apply that to decoding new words, progressing from the simple to the complex. Over time newly acquired words are stored in memory, and the pathway used to identify words by sight develops and progressively supplements the decoding pathway. Oral language processing creates meaning from the

words. These two processes may develop relatively independently, and the relationship between these factors and reading comprehension changes over time. For younger children decoding is more important because it is not yet automatic, but by secondary school decoding differences are generally small, and the relationship between reading and listening comprehension is very strong. Therefore, one should expect a person's reading comprehension to develop to the same level as their listening comprehension once word reading is fluent (Cain and Oakhill 2008). The SVR and the manner in which the skills develop over time will provide a framework for understanding reading comprehension challenges for students with HFA.

It is concerning that research has identified high rates of reading comprehension impairments in children and adolescents with HFA that are not consistent with their overall cognitive abilities (e.g., Jones et al. 2009; McIntyre et al. 2017). This learning difficulty not only affects current academic development; it also impacts future vocational outcomes such as full-time employment and earnings prospects and has been shown to predict earnings of workers across educational attainment levels and occupations. Impairments in comprehension skills can limit the ability to learn new information and communicate effectively, thereby further impacting independent living capacity in adulthood. Therefore, understanding and addressing the high rates of reading comprehension impairments in school-aged children and adolescents with HFA is an important endeavor.

Current Knowledge

The SVR framework supplies a foundation upon which to build an understanding of the general reading challenges that students with HFA may share with all students who struggle with reading. However, there is some evidence to suggest that in addition to the broad constructs of word recognition and listening comprehension, the comprehension difficulties experienced by many students with HFA may also be specific to their social communication and cognitive characteristics.

General Reading Challenges

Word recognition skill is dependent upon oral language skills such as phonological processing of speech sounds for word decoding and vocabulary development (National Reading Panel 2000). For many students with HFA, decoding skills develop adequately and word reading is a relative strength upon which to build. However, some do experience difficulties developing proficient word recognition skills, and those challenges are linked to phonological processing impairments similar to readers without ASD (Åsberg and Dahlgren Sandberg 2012). Of note, the word recognition impairments of readers with ASD have also been shown to be related to challenges in key aspects of oral language such as morphology (i.e., smallest parts of words that convey meaning such as prefixes and suffixes), syntax (i.e., word order in sentences), and vocabulary (Ricketts et al. 2013). This is important because oral language skills often develop atypically in children with ASD who display a wide variety of expressive and receptive language abilities that may develop at different rates and to different levels of competence (Eigsti et al. 2011). Therefore, the link between these language skills and word recognition is important to recognize, assess, and apply when targeting reading intervention services.

Listening comprehension skills such as narrative abilities and inference generation have been shown to be challenging for typically developing readers with poor comprehension (Cain and Oakhill 2008) and also for readers with HFA (McIntyre et al. 2017). In early elementary school, children are primarily exposed to academic and narrative content through read-alouds and oral discussions lead by caregivers and teachers. Listening comprehension skills develop in these contexts and should not only include an understanding of the individual words and sentences but also should lead to the construction of a mental model of the presented information that integrates one's related background knowledge. When the content is narrative in nature,

comprehension relies on understanding and utilizing story structure, the social-cognitive ability to understand the intentions and beliefs of characters in a story, as well as language skills more broadly, and research findings support a causal relation between narrative skills and reading comprehension development (Cain and Oakhill 2008). Children with ASD often display difficulties with narratives which may be due to difficulties with social cognition and oral language skills (Randi et al. 2010). Regardless of content area, the ability to generate links between different pieces of information as well as to connect to, and integrate, what one already knows about a topic is known as inference generation. Making inferences allows a listener or reader to fill in gaps and resolve ambiguities about what they are reading or hearing to attain deeper comprehension. Answering comprehension questions that require inferential thinking is much more challenging for most people than answering questions that depend on direct recall of facts and details, and research has reported that students with HFA have a relative strength for correctly answering direct recall questions (McIntyre et al. 2017). This could be the case because individuals with ASD may have difficulty accessing relevant background knowledge and integrating it with information in texts to make inferences, particularly when the content is highly social (Norbury and Nation 2011). Oral language skills have also been linked to inferential processing, and while individuals with ASD who have well-developed verbal skills have not displayed an overall problem with inference, they have demonstrated a particular difficulty with inferences about social and emotional information (Bodner et al. 2015; Happé 1995).

Finally, while both word recognition and listening comprehension skills are related to reading comprehension in readers with HFA, research has shown that they are not as tightly associated in students with ASD as they are in other groups of students (Jones et al. 2009; McIntyre et al. 2017). This is likely due to a higher incidence of a reading profile characterized by adequate word recognition skills alongside poor comprehension in many children

and adolescents with HFA, which is notable and likely specific to the language and greater social communication challenges in readers with ASD.

ASD-Specific Challenges

Beyond the components of the SVR, characteristics associated with a diagnosis of ASD impact reading comprehension for many students with HFA. Social communication, or the ability to use language and nonverbal communication in a purposeful way to convey a message to another person, plays a role in understanding that written texts are a form of communication with an intended meaning or purpose and are subject to different interpretations by different people. Specifically, the ability to attribute mental states such as desires and motivations to oneself, and to infer others' mental states, plays a key role in understanding and predicting their behavior and is often referred to as theory of mind (ToM). ToM helps one to understand that the purpose of oral and written language is to express thoughts and feelings and to aid in the interpretation of a speaker or writer's intended meaning. Not only is this relevant for texts read in school settings, it is increasingly important as computer access to social media, online forums, and news websites proliferates. Readers need to be able to discern the differing perspectives and motives of the online authors not only for accurate comprehension but also for their safety and well-being. For example, not understanding someone's intentions could lead to becoming the victim of fraud or becoming misled and upset by exaggerated viewpoints. ToM tasks have been shown to predict narrative text comprehension in all readers likely because the tasks capture inference making and complex social reasoning skills that are necessary for understanding characters' intentions and beliefs (Kim 2015). Impairments in ToM are reported across developmental stages and functioning levels in individuals with ASD (Baron-Cohen et al. 1997) and oral language skills have been linked to performance on ToM tasks (Happé 1995). Highly social texts have been shown to be more difficult to comprehend than those with

lower social content for children with ASD (Brown et al. 2013), and impairments in ToM explain differences in reading comprehension for children and adolescents with ASD that extend beyond the contributions of word recognition and listening comprehension skills (McIntyre et al. 2018; Ricketts et al. 2013). Taken together, there is growing evidence that comprehension challenges may also align with ASD-specific social-cognitive vulnerabilities.

A cognitive characteristic associated with ASD, restricted interests, can limit the breadth of background knowledge in long-term memory that is available for inference-making since an extensive focus on one or two topic areas diverts attention from broader learning experiences. Even when an individual has access to an extensive variety of experiences, a lack of interest or engagement may limit their uptake and benefit from these opportunities. Not only will this impact their general knowledge fund, it also affects their language and social development which in turn impacts reading comprehension. A related issue is inflexible and concrete learning seen in many students with ASD that may limit their ability to generalize and apply knowledge or skills learned in one context to new situations or settings. Readers with HFA might not realize when they have background knowledge that is relevant to a text, or fail to suppress irrelevant knowledge, and therefore miss opportunities for deeper comprehension. Another aspect of cognitive processing that is associated with, although not exclusive to, ASD is a tendency to focus on details rather than global meaning, which is often referred to as weak central coherence. This can impact reading comprehension because a focus on details leads to difficulty perceiving the larger meaning of a text which in turn leads to difficulty identifying main ideas and themes and integrating text meaning with background knowledge. This processing style also makes it more difficult to generalize and apply ideas and concepts to other texts. This becomes increasingly important in middle and high school when students are asked to do things such as extract themes from novels and compare or contrast them across other novels or

situations. If one is not able to identify global meanings and greater themes, it is difficult to recognize similarities between texts. Taken together, reduced breadth of background knowledge, contextualized learning, and a tendency to focus on text details rather than central meaning lead to difficulties building a comprehensive mental model of the text.

Building Reading Comprehension Skills

A body of reading comprehension intervention research with elementary through high school students with ASD is emerging. Specific intervention strategies and methods that have demonstrated promise include question generation (teaching students how to ask relevant questions about a text to facilitate monitoring and repair of comprehension), pronoun identification (determining the person or object to which the pronoun is referring), explicit or direct instruction techniques, making predictions about a text, cooperative learning groups, and peer tutoring. The use of visual supports in the form of graphic organizers, story element and word cards, and self-monitoring checklists has shown promise as well (El Zein et al. 2014). Teaching students to generalize text comprehension strategies outside the intervention materials to other texts is also an important intervention ingredient. Use of multiple exemplars while teaching, graphic organizers such as Venn diagrams, response-prompting strategies, cooperative learning instruction with typically developing peers, and computer-aided instruction all have shown some promise in promoting generalization across materials and exemplars for students with ASD (Sartini et al. 2018).

To expand on one instructional practice noted in both research reviews, reading interventions that also include a peer-mediated social component such as cooperative learning groups or peer tutoring can promote more active student engagement and increased opportunities to communicate and collaborate. Research on school-based peer-mediated interventions for children and adolescents with ASD has demonstrated that they are a promising approach to improving social competence along with academic functioning and access

to the general education curriculum for students with ASD (Bene et al. 2014; Ledford and Wehby 2015). A small body of prior research has demonstrated positive effects of peer-mediated reading comprehension interventions on both reading and social outcomes simultaneously for children and adolescents with ASD (Bene et al. 2014). Interventions of this type offer the possibility of a targeted approach to intervention for academic achievement that fits well with the curriculum and expertise of general education classrooms and may have a cascading positive impact on the social communication development of school-aged children with ASD.

Future Directions

While evidence is emerging in support of specific strategies or instructional practices, little is known about intervening comprehensively on challenges in oral language, global processing, social cognition (ToM), integration of background knowledge and inference making, and generalization to other texts and across contexts that may be specific to the social and cognitive characteristics of ASD. Neither do we know which of these skills are the most crucial to include in comprehensive reading instruction for students with HFA that are effective for varying age groups. Additionally, little work has been done with samples of students with HFA to examine other cognitive processing domains related to reading such as self-monitoring of comprehension and engaging in repair strategies, planning and organization, attention, inhibition of irrelevant information, memory, and how impairment in these skills impacts their text comprehension specifically. Finally, more research on academic achievement and reading instruction for individuals with HFA in the elementary and secondary grades that emphasizes the relationship between social and academic skills and simultaneously addresses both skill areas is warranted. As with other students, students with HFA have a variety of strengths and weaknesses that call for an individualized and flexible instructional approach. Therefore, a

curriculum that addresses multiple areas of concern (e.g., reading and social communication) and is adaptable may be the most feasible and effective for teachers to implement in general education settings and may ultimately serve the needs of many students in these settings with reading comprehension challenges.

See Also

- Academic Skills
- Learning Disability
- Narrative Assessment
- Narrative Language in ASD

References and Reading

- Åsberg, J., & Dahlgren Sandberg, A. (2012). Dyslexic, delayed, precocious or just normal? Word reading skills of children with autism spectrum disorders. *Journal of Research in Reading, 35*(1), 20–31.
- Baron-Cohen, S., Jolliffe, T., Mortimore, C., & Robertson, M. (1997). Another advanced test of theory of mind: Evidence from very high functioning adults with autism or Asperger syndrome. *Journal of Child Psychology and Psychiatry, 38*(7), 813–822.
- Bene, K., Banda, D. R., & Brown, D. (2014). A meta-analysis of peer-mediated instructional arrangements and autism. *Review Journal of Autism and Developmental Disorders, 1*(2), 135–142.
- Bodner, K. E., Engelhardt, C. R., Minshew, N. J., & Williams, D. L. (2015). Making inferences: Comprehension of physical causality, intentionality, and emotions in discourse by high-functioning older children, adolescents, and adults with autism. *Journal of Autism and Developmental Disorders, 45*(9), 2721–2733.
- Brown, H. M., Oram-Cardy, J., & Johnson, A. (2013). A meta-analysis of the reading comprehension skills of individuals on the autism spectrum. *Journal of Autism and Developmental Disorders, 43*(4), 932–955.
- Cain, K., & Oakhill, J. (2008). Reading comprehension difficulties: Correlate, causes, and consequences. In K. Cain & J. Oakhill (Eds.), *Children's comprehension problems in oral and written language: A cognitive perspective*. New York: Guilford Press.
- Eigsti, I.-M., de Marchena, A., Schuh, J., & Kelley, E. (2011). Language acquisition in autism spectrum disorders: A developmental review. *Research in Autism Spectrum Disorders, 5*(2), 681–691.
- El Zein, F., Solis, M., Vaughn, S., & McCulley, L. (2014). Reading comprehension interventions for students with autism spectrum disorders: A synthesis of research. *Journal of Autism and Developmental Disorders, 44*(6), 1303–1322.
- Gough, P. B., & Tunmer, W. E. (1986). Decoding, reading, and reading disability. *Remedial and Special Education, 7*(1), 6–10.
- Happé, F. G. (1995). The role of age and verbal ability in the theory of mind task performance of subjects with autism. *Child Development, 66*(3), 843–855.
- Jones, C. R., Happé, F., Golden, H., Marsden, A. J., Tregay, J., Simonoff, E., . . . & Charman, T. (2009). Reading and arithmetic in adolescents with autism spectrum disorders: Peaks and dips in attainment. *Neuropsychology, 23*(6), 718.
- Kim, Y. S. (2015). Language and cognitive predictors of text comprehension: Evidence from multivariate analysis. *Child Development, 86*(1), 128–144.
- Ledford, J. R., & Wehby, J. H. (2015). Teaching children with autism in small groups with students who are at-risk for academic problems: Effects on academic and social behaviors. *Journal of Autism and Developmental Disorders, 45*(6), 1624–1635.
- McIntyre, N. S., Solari, E. J., Gonzales, J. E., Solomon, M., Lerro, L. E., Novotny, S., . . . & Mundy, P. C. (2017). The scope and nature of reading comprehension impairments in school-aged children with higher-functioning autism spectrum disorder. *Journal of Autism and Developmental Disorders, 47*(9), 2838–2860.
- McIntyre, N. S., Oswald, T. M., Solari, E. J., Zajic, M. C., Lerro, L. E., Hughes, C., . . . & Mundy, P. C. (2018). Social cognition and reading comprehension in children and adolescents with autism spectrum disorders or typical development. *Research in Autism Spectrum Disorders, 54*, 9–20.
- National Reading Panel. (2000). *Report of the National Reading Panel: Teaching children to read: An evidence-based assessment of the scientific research literature on reading and its implications for reading instruction* (Report of the subgroups). Washington D.C.: NICHD Clearinghouse.
- Norbury, C., & Nation, K. (2011). Understanding variability in reading comprehension in adolescents with autism spectrum disorders: Interactions with language status and decoding skill. *Scientific Studies of Reading, 15*(3), 191–210.
- Randi, J., Newman, T., & Grigorenko, E. (2010). Teaching children with autism to read for meaning: Challenges and possibilities. *Journal of Autism and Developmental Disorders, 40*, 890–902.
- Ricketts, J., Jones, C. R., Happé, F., & Charman, T. (2013). Reading comprehension in autism spectrum disorders: The role of oral language and social functioning. *Journal of Autism and Developmental Disorders, 43*(4), 807–816.
- Sartini, E., Knight, V. F., Spriggs, A. D., & Allday, R. A. (2018). Generalization strategies to promote text comprehension skills by students with ASD in core content areas. *Focus on Autism and Other Developmental Disabilities, 33*(3), 150–159.

Reading Comprehension Strategies in Autism

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Synonyms

[Reading interventions](#)

Definition

Individuals with autism spectrum disorders (ASD) have significant variability of academic skills. For example, some students with autism may have age-appropriate decoding skills, but lack reading comprehension skills, while some students with ASD have deficits in both decoding and comprehension. Language, vocabulary, and decoding issues can all play a role in the lack of ability to understand text. In addition to these concerns, individuals with ASD also have impairments in three areas of cognition: theory of mind (Baron-Cohen 1995), central coherence (Frith 1989), and executive functioning (Luria 1966). Challenges in these areas can also impact their reading comprehension (Carnahan et al. 2011). Teachers can use the following strategies to target these areas of cognition and help support students with ASD when comprehending text.

Strategies to Support Theory of Mind

Theory of mind is the ability to see something from someone else's perspective (Baron-Cohen 1995). Individuals with ASD have a difficult time understanding perspective in social situations; additionally, they may have difficulty understanding perspectives of different characters in texts. This leads to a variety of implications with comprehending text (i.e., making

predictions, understanding emotions, understanding deception, determining fact from fiction, etc.)

This difficulty with perspective taking can be partly attributed to the students' inability to produce and understand pronouns (Finnegan and Accardo 2018). Anaphoric cueing teaches strategies on how to interpret pronouns by connecting pronoun references to specific characters in text. Teachers should target the visual area of strength for students with ASD by highlighting pronouns and character reference in the same color to assist in comprehension. Teachers should then use follow-up questions using pronouns to check for understanding. Supports and questioning can be faded over time.

Strategies to Support Central Coherence

Central coherence is the tendency to draw together diverse information to construct higher-level meaning in context (Frith 1989). Individuals with ASD have difficulty understanding the "big picture" in real-world situations, and they have the same difficulty when reading text. This can impact a student in the ability to identify the main idea and draw conclusions about what they have read.

Developing the schema for the students can help them generate prior knowledge about what they will be reading about. For example, before reading a text about a rainforest, show them pictures and videos about the rainforest and then visually brainstorm thoughts, questions, and facts about what they saw. They may also have an idiosyncratic focus of attention, difficulty making choices, and seeing connections within and across text. Teaching students to record important facts on post-it notes during reading can help students focus in on salient features in a visual way.

Strategies to Support Executive Functioning

Executive functioning skills relate to the ability to maintain an appropriate problem-solving set for attainment of a future goal (Luria 1966).

Individuals with ASD have impaired executive functioning skills and therefore may have difficulty with the following comprehension skills: organizing information within text, focusing on significant events in a story, and understanding alternate facts/opinions. Individuals with ASD often have rigidity in thinking which may lead to an inability to explore the topic by integrating new information, thoughts, or ideas. The use of graphic organizers will help students visually identify and organize what they have read (Finnegan and Accardo 2018). Graphic organizers depicting story elements are a way to target the visual strength of an individual with ASD while providing an organizational tool to target important text features (i.e., characters, setting, problem, solution, etc.).

See Also

- [Academic Skills](#)
- [Autism Theory](#)
- [Literacy](#)

References and Reading

- Baron-Cohen, S. (1995). *Mind blindness: An essay on autism and theory of mind*. Cambridge, MA: The MIT Press.
- Carnahan, C. R., Williamson, P. S., & Christman, J. (2011). Linking cognition and literacy in students with autism spectrum disorder. *Teaching Exceptional Children*, 43(6), 54–62. <https://doi.org/10.1177/004005991104300606>.
- Finnegan, E., & Accardo, A. (2018). Understanding character perspective: Strategies to support students with autism spectrum disorder. *The Reading Teacher*, 72(1). <https://doi.org/10.1002/trtr.1682>.
- Frith, U. (1989). *Autism: Explaining the enigma*. Oxford: Blackwell.
- Luria, A. R. (1966). *Human brain and psychological processes*. New York: Harper and Row.

Reading Skills

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Definition

Reading skills lead a person to interact and gain meaning from written language. There are several components one must master which lead to independently comprehending the intended message being relayed in the written content. First is phonemic awareness which is defined by the National Reading Panel as “recognizing and manipulating spoken words in language” (Whalon et al. 2009). Next is phonics defined by the same group as “understanding letter-sound correspondences in reading and spelling” then oral reading fluency which is “reading text with speed, accuracy, and expression.” The forth component is vocabulary defined as “understanding words read by linking the word to oral vocabulary” and lastly is comprehension defined as “directly teaching students to be aware of the cognitive processes involved in reading” (p. 4).

Typically developing children will start to acquire reading skills in preschool and continue to learn along a continuum until they reach an independent reading level. Students with ASD often acquire some reading skills in an uneven nature which may be indicative of the student’s language delays or rote memory skills. Despite strong decoding skills, students with ASD often have great difficulty becoming skilled readers which requires higher level skills such as making inferences from the text (Lanter and Watson 2008).

Historical Background

Since the early 1900s, theorists have acknowledged that reading is a complex task which requires a number of mastered skills. In 1985, the National Academy of Education held onto

Reading Interventions

- [Reading Comprehension Strategies in Autism](#)

the view supported by Gates in 1949 that reading is complex and requires a number of higher level skills to be manipulated together to achieve reading competence (Gough and Hoover 1990).

A differing view has developed, often referred to as the *Simple View of Reading*, and states “one central claim of the simple view is that reading only consists of two components, decoding and linguistic comprehension (Gough and Hoover 1990).

Current Knowledge

Research has proven that reading skills are acquired through systematic instruction and improve with practice and repeated instruction. Students with autism are a heterogeneous group which means one intervention cannot be prescribed to teach all students. Through reading assessments, an accurate profile of the student’s reading level can be determined and lead to appropriate reading interventions.

Future Directions

Research needs to continue in the area of developing reading skills for children with ASD. Whalon, Otaiba, and Delano recommend that researchers “evaluate the impact of comprehensive reading instruction on the reading development of children with ASD” (2009). As technology continued to evolve, its effectiveness along with computer-aided instruction should be evaluated as it applies to reading skills of all levels.

See Also

► [Reading](#)

References and Reading

- Afflerbach, P., Pearson, P. D., & Paris, S. G. (2008). Clarifying differences between reading skills and reading strategies. *The Reading Teacher*, 61(5), 364.
- Gough, P. B., & Hoover, W. A. (1990). The simple view of reading. *Reading and Writing: An Interdisciplinary Journal*, 2, 127–160.

- Huemer, S. V., & Mann, V. (2010). A comprehensive profile of decoding and comprehension in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(4), 485–493.
- King-Sears, M. E., Mainzer, L., & Swanson, C. (2011). TECHnology and literacy for adolescents with disabilities: A sound decision-making framework can assist teachers in adopting and embracing current technologies and looking toward Web 3.0 in order to create universally accessible learning environments to advance literacy learning for all students, and especially students with disabilities. *Journal of Adolescent & Adult Literacy*, 54(8), 569.
- Lanter, E., & Watson, L. R. (2008). Promoting literacy in students with ASD: The basics for the SLP. *Language, Speech, & Hearing Services in Schools*, 39, 33.
- Luyster, R., & Lord, C. (2009). Word learning in children with autism spectrum disorders. *Developmental Psychology*, 45(6), 1774–1786.
- Narkon, D. E., Wells, J. C., & Segal, L. S. (2011). E-word wall: An interactive vocabulary instruction tool for students with learning disabilities and autism spectrum disorders. *Teaching Exceptional Children*, 43(4), 38–45.
- Randi, J., Newman, T., & Grigorenko, E. L. (2010). Teaching children with autism to read for meaning: Challenges and possibilities. *Journal of Autism and Developmental Disorders*, 40(7), 890–902.
- Whalon, K., & Hart, J. E. (2011). Adapting an evidence-based reading comprehension strategy for learners with autism spectrum disorder. *Intervention in School and Clinic*, 46(4), 195–203.
- Whalon, K. J., Otaiba, S. A., & Delano, M. E. (2009). Evidence-based reading instruction for individuals with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 24(1), 3–16.

Reading the Mind in Eyes Test

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Synonyms

[Eyes Test](#); [RMET](#)

Description of the Instrument

The Reading the Mind in the Eyes Test (RMET) measures theory of mind or how well individuals can put themselves into the mind of another

person and attribute mental states to another person. The test itself takes approximately 20 min to complete and is relatively simple to administer and perform, making it a useful instrument for a variety of populations and settings.

The test consists of a series of 37 photographs of the eye region of the face of different actors and actresses. In addition, the test includes instructions, an eight-page word definitions list, a response form, and an answer form. There is one practice stimulus and 36 experimental stimuli. Each of the 37 images has four response options: emotion words that are defined in the provided word definition list. The response form, which is given to the subject, lists the four response options that are presented with the image. The subject is instructed to circle the word that best describes what the person in the picture is thinking or feeling. Further, the subject is told to choose the word that is most suitable for the image even if more than one word feels applicable. The task is not timed, but the subject is instructed to work as quickly and as accurately as possible.

The score on the RMET is calculated by counting the number of correct responses. While there are no normative data or scores, there are published averages for different populations.

History of the Instrument

The RMET was first developed in 1997 by Simon Baron-Cohen, Ph.D., and colleagues of the University of Cambridge (Baron-Cohen et al. 1997). He and his colleagues developed the test as a means of measuring theory of mind in adults with autism spectrum disorders (ASD). Previous theory of mind tests had been proposed for children with ASD but did not provide an opportunity for insight into theory of mind functioning in adults with ASD. In the seminal paper on the RMET, Baron-Cohen and colleagues (1997) suggested that deficits in theory of mind may underlie some of the difficulties seen in adults with ASD. In this initial study of the RMET, it was found that adults with ASD performed significantly worse (lower total score) than typically developing individuals and individuals with Tourette syndrome (clinical control group).

Further, they found that within the typically developing control sample, females performed significantly better than males; this was not tested in the clinical control group (individuals with Tourette syndrome) or in the experimental group (individuals with ASD). Performance on the task did not correlate with IQ, but all individuals had IQ scores within the average or above average range. This study concluded that theory of mind deficits existed in individuals with ASD, with an average or above average IQ, and that the RMET was successful in accurately measuring these subtle social difficulties. The authors also concluded that the observed theory of mind deficits in these individuals suggested that social cognition is independent of general intelligence (Baron-Cohen et al. 1997).

Since its development in 1997, the RMET has been revised (Baron-Cohen et al. 2001a). The authors identified three major psychometric problems with the first version of the RMET. First, the initial test had only two response options, which limited the range and restricted the interpretation of individual differences. Second, these response options were the basic emotions (happy, sad, anger, fear, and disgust) and were judged to be too easy, leading to ceiling effects. Finally, the two response choices were always semantic opposites; if the subjects were able to judge positive versus negative emotion, they would receive credit without having to utilize theory of mind principles. Thus, major modifications to the RMET appeared in the revised version, which was released in 2001. These modifications included increasing the number of response choices from 2 to 4 words, using only complex mental states as response choices rather than the basic emotions and including foil words that had the same emotional valence as the target word (i.e., target word, serious; foil words, ashamed, bewildered, and alarmed) instead of semantic opposites. It was found that with these modifications, individuals in the typical control group performed below ceiling, indicating that the revised test had more power to discriminate and identify meaningful individual differences. The results of the initial study (Baron-Cohen et al. 1997) were replicated; individuals with ASD were significantly impaired on the revised RMET as well. In addition to the

revised version of the adult RMET, a children's version of the test was also released in 2001 (Baron-Cohen et al. 2001b). The children's version of the RMET is very similar to the adult version; however, the response choices have been changed to be more suitable for children.

Psychometric Data

Normative Data

The RMET does not have normative data, but rather the total raw scores are used to compare between individual and group performance on the task. Baron-Cohen et al. (2001a) reported an expected distribution for typically developing adults, with adults with ASD performing significantly worse (lower total score).

Reliability

The reliability of the RMET has been broadly studied and is generally accepted as good. In a study of the Swedish version of the RMET, a group of students (mean age 23.9 years) completed the test at two time points separated by 3 weeks in order to evaluate test-retest reliability (Hallerbäck et al. 2009). There was no significant difference found between the mean score from time point 1 and time point 2 ($p = .35$). Further, there was a significant positive correlation between the scores from the first and second time points ($r = .60, p < .01$).

Validity

Baron-Cohen et al. (1997) initially reported on the validity of the RMET as a true test of theory of mind based on the idea that it did not involve executive functioning skills nor did it require a central coherence component (Ozonoff et al. 1991; Frith 1989). In order to validate the test, performance on the RMET was compared to performance on Happé's Strange Stories task (a known test of theory of mind) and two control tasks (Gender Recognition task and Basic Emotion Recognition task) (Happé 1994; Baron-Cohen et al. 1997). Individuals with ASD were significantly impaired on both the RMET and Happé's Strange Stories task as compared to a typically developing control group and a clinical

control group (individuals with Tourette syndrome) (Happé 1994; Baron-Cohen et al. 1997). Further, there were no differences between the groups on the either of the control tasks. Finally, Baron-Cohen et al. (1997) reported that because the target words were mental state terms and not just emotion terms, the RMET was testing theory of mind and not emotion recognition. Thus, it was concluded that the RMET was valid and indeed testing theory of mind.

The 2001 revision of the RMET provided direct evidence for the validity of the test (Baron-Cohen et al. 2001a). Results from the original study of the RMET (Baron-Cohen et al. 1997) were replicated in the study of the revised version, suggesting that the RMET is indeed a useful test for detecting individual differences and impairment in theory of mind functioning level in adults. Since its release, the revised version of the RMET has also been used in many studies of a wide range of psychological/neurological conditions. These studies have demonstrated that the RMET can reliably distinguish between nonclinical samples and clinical samples both in behavioral studies and fMRI studies (Baron-Cohen et al. 1999; Lee et al. 2005; Hallerbäck et al. 2009).

Clinical Research Uses

The RMET was initially developed as a behavioral test of theory of mind functioning level in adults with ASD. Since its release in 1997, it has been widely used in behavioral and functional magnetic resonance imaging (fMRI) studies with both children and adults.

The RMET has been instrumental in demonstrating theory of mind impairments associated with a variety of clinical populations including, but not limited to, ASD, depression, schizophrenia, and dementia and Alzheimer's disease. Lee et al. (2005) found that the ability to decode other's mental states was significantly impaired in severely depressed individuals and suggested that strategies that improve theory of mind reasoning may be useful in therapeutic interventions for severely depressed individuals. Furthermore, impaired theory of mind functioning in

individuals with schizophrenia has also been evaluated using the RMET. Bora et al. (2006) assessed mental state decoding in individuals with schizophrenia using the RMET and found that the RMET was successful in predicting level of social functioning. In addition, they determined that mental state decoding seems to be the most important cognitive mediator of social functioning in individuals with schizophrenia. Finally, the RMET has also been used in studies of dementia and Alzheimer's disease. Gregory et al. (2002) found that patients with frontal variant frontotemporal dementia were impaired on the RMET and that individuals with Alzheimer's disease were not significantly impaired. They concluded that theory of mind impairment may explain some of the deficits in interpersonal behavior experienced in frontal variant frontotemporal dementia and not in Alzheimer's disease.

The RMET has also been widely used in fMRI studies of ASD and other psychiatric disorders. Many studies have found that the deficits in theory of mind in individuals with ASD, as measured using the RMET, are substantiated by different brain activation patterns in response to social stimuli (Baron-Cohen et al. 1999; Gordon et al. 2013). Furthermore, Hirao et al. (2008) found that in individuals with schizophrenia, poor performance on the RMET was associated with reduced gray matter in the left ventrolateral prefrontal cortex (VLPFC). Thus, they suggested that this gray matter reduction may be important in understanding theory of mind deficits experienced by individuals with schizophrenia. Finally, clinical effects of oxytocin for individuals with ASD have been evaluated using the RMET. Performance on the RMET was enhanced after intranasal administration of oxytocin in individuals with ASD (Domes et al. 2007; Guastella et al. 2010; Gordon et al. 2013). These findings suggest that oxytocin may be involved in theory of mind deficits associated with ASD (Domes et al. 2007).

See Also

- Autism
- Functional Magnetic Resonance Imaging

- Psychological Assessment
- Social Cognition
- Theory of Mind

References and Reading

- Baron-Cohen, S., Jolliffe, T., Mortimore, C., & Robertson, M. (1997). Another advanced test of theory of mind: Evidence from very high functioning adults with autism or Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 38, 813–822.
- Baron-Cohen, S., Ring, H. A., Wheelwright, S., Bullmore, E. T., Brammer, M. J., Simmons, A., & Williams, S. C. (1999). Social intelligence in the normal and autistic brain: An fMRI study. *European Journal of Neuroscience*, 11, 1891–1898.
- Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001a). The “reading the mind in the eyes” test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry*, 42, 241–251.
- Baron-Cohen, S., Wheelwright, S., Spong, A., Scahill, V., & Lawson, J. (2001b). Are intuitive physics and intuitive psychology independent? A test with children with Asperger syndrome. *Journal of Developmental and Learning Disorders*, 5, 47–78.
- Bora, E., Eryavuz, A., Kayahan, B., Sungu, G., & Veznedaroglu, B. (2006). Social functioning, theory of mind and neurocognition in outpatients with schizophrenia; mental state decoding may be a better predictor of social functioning than mental state reasoning. *Psychiatry Research*, 145, 95–103.
- Domes, G., Heinrichs, M., Michel, A., Berger, C., & Herpertz, S. C. (2007). Oxytocin improves “mind-reading” in humans. *Biological Psychiatry*, 61, 731–733.
- Frith, U. (1989). *Autism: Explaining the enigma*. Oxford: Basil Blackwell.
- Gordon, I., Vander Wyk, B. C., Bennett, R. H., Cordeaux, C., Lucas, M. V., Eilbott, J. A., . . . & Pelphrey, K. A. (2013). Oxytocin enhances brain function in children with autism. *Proceedings of the National Academy of Sciences*, 110, 20953–20958.
- Gregory, C., Lough, S., Stone, V., Erzincinoglu, S., Martin, L., Baron-Cohen, S., & Hodges, J. R. (2002). Theory of mind in patients with frontal variant frontotemporal dementia and Alzheimer's disease: Theoretical and practical implications. *Brain*, 125, 752–764.
- Guastella, A. J., Einfeld, S. L., Gray, K. M., Rinehart, N. J., Tonge, B. J., Lambert, T. J., & Hickie, I. B. (2010). Intranasal oxytocin improves emotion recognition for youth with autism spectrum disorders. *Biological Psychiatry*, 67, 692–694.
- Hallerbäck, M. U., Lugnegård, T., Hjärthag, F., & Gillberg, C. (2009). The reading the mind in the eyes test: Test – retest reliability of a Swedish version. *Cognitive Neuropsychiatry*, 14, 127–143.

- Happé, F. G. (1994). An advanced test of theory of mind: Understanding of story characters' thoughts and feelings by able autistic, mentally handicapped, and normal children and adults. *Journal of Autism and Developmental Disorders*, 24, 129–154.
- Hirao, K., Miyata, J., Fujiwara, H., Yamada, M., Namiki, C., Shimizu, M., . . . & Murai, T. (2008). Theory of mind and frontal lobe pathology in schizophrenia: A voxel-based morphometry study. *Schizophrenia research*, 105, 165–174.
- Lee, L., Harkness, K. L., Sabbagh, M. A., & Jacobson, J. A. (2005). Mental state decoding abilities in clinical depression. *Journal of Affective Disorders*, 86, 247–258.
- Ozonoff, S., Pennington, B., & Rogers, S. (1991). Executive function deficits in high-functioning autistic children: Relationship to theory of mind. *Journal of Child Psychology and Psychiatry*, 32, 1081–1106.

Real-Life Rating Scale

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Synonyms

RLRS; Ritvo-Freeman real-life rating scale

Description

The scale is designed to assess changes in symptoms over time in children with autistic disorder. Observers rate rates 47 pathological behaviors on a Likert Scale. It is applicable in natural settings by nonprofessional raters who can be rapidly trained to achieve significant inter observer agreement, is rapid scored by hand, and can be repeated frequently without effecting inter rater agreement. It does not interfere with or alter the behaviors being rated.

Historical Background

The scale was developed by modifying the Behavior Observation Scale (BOS) (Freeman et al. 1996). This standardized instrument utilizes videotape made through a one-way mirror into an observation room in which there is a patient, an observer, and a uniform set of toys. In order to meet design

requirements for simplicity for this new scale, the laboratory setting was abandoned in favor of observing the patient in their real-life settings. Simplified objective definitions of behaviors were agreed upon, and a data sheet was developed that can be easily scored by hand.

Psychometric Data

High degrees of interrater reliability (novice vs. experienced observers) and test-retest reliability were achieved. Validity when compared to the Alpern-Boll Scale was highly significant ($p = <0.001$). Subscales (sensory motor, social relationship to people, affectual responses, sensory responses, language, and overall score) were highly correlated ($p = 0.001$).

Clinical Uses

The Ritvo-Freeman Real Life Rating Scale is particularly useful for measuring changes over time in the symptomatic behaviors of children with developmental disabilities, in particular autistic disorder. It has gained wide spread use in assessing the effects of behavioral and pharmacological treatments.

References and Reading

- Freeman, B. J., Ritvo, E. R., Yokoa, A., & Ritvo, A. (1996). A scale for rating symptoms of patients with the syndrome of autism in real life settings. *Journal of Autism and Developmental Disorders*, 25, 130–136.

Reasonable Accommodation

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Synonyms

Adjustment; Appropriate adaptation; Modification

Definition

A reasonable accommodation is an adaptation, modification, or change of the educational environment, the presentation of educational material, the method of response, or the educational content. Schools are expected to make the necessary changes that will allow students with disabilities to learn in spite of a fundamental weakness or deficiency. Reasonable accommodations are determined based on individual student needs and should be made based on an evaluation of obstacles or barriers that interfere with the student's access to the school facilities, the classroom, instruction, or student performance. Reasonable accommodations are intended to make it possible for students with disabilities to have an equal opportunity to education. There are several different types of accommodations; some examples include:

- Arranging location and or furniture for accessibility purposes
- Offering alternative ways of completing assignments or responding on assessments (e.g., oral vs. written)
- Curriculum or program modifications
- Test modifications (e.g., reduced number of items on tests)
- Time extensions to complete assignments and assessments
- Assistive technology equipment
- Document conversion (e.g., braille, large print, audiotape)
- Auxiliary aids and services (note takers, lab or library assistants, readers, interpreters)
- Captions for video material

See Also

- ▶ [Individual Education Plan](#)
- ▶ [Individuals with Disabilities Education Act \(IDEA\)](#)
- ▶ [Related Services](#)

References and Reading

Assistance to States for the Education of Children with Disabilities and Preschool Grants for Children with Disabilities; Final Rule, 2004, 4000-01-U Department of Education 34 CFR Parts 300 and 301, Part II.

ERIC Digest. (1993). *Including students with disabilities in general education classrooms (#E521)*. Reston: ERIC Clearinghouse on Disabilities and Gifted Education, ERIC Document Service No. ED358677.

Individuals with Disabilities Education Act of 2004, 20 U.S.C., et. seq.

Mondak, P. (2000). The Americans with disabilities act and information technology access. *Focus on Autism and Other Developmental Disabilities*, 15(1), 43–51.

Roberts, K. D. (2010). Topic areas to consider when planning transition from high school to postsecondary education for students with Autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 25(3), 158–162.

Simpson, R. L., Griswold, D. E., & Smith Myles, B. (1999). Educators' assessment accommodation preferences for students with autism. *Focus on Autism and Other Developmental Disabilities*, 14(4), 212–219.

Thompson, S. J., Morse, A. B., Sharpe, M., & Hall, S. (2005). *Accommodations manual: How to select, administer, and evaluate use of accommodations for instruction and assessment of students with disabilities* (2nd ed.). Washington, DC: The Council of Chief State School Officers.

Reasoning on the Autism Spectrum

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Definition

Autism spectrum disorder is associated with relative weaknesses in contexts that require intuition and relative strengths in contexts that require deliberation. Intuition involves rapid, effortless, parallel, nonconscious processing that is independent of working memory and cognitive ability. Deliberation, on the other hand, involves slower, effortful, sequential, conscious processing and is heavily dependent on working memory and related to individual differences in cognitive ability.

Historical Background

“Every fully functioning human adult shares a sense that the ability to think, to reason, is a part of their fundamental identity” (Holyoak and

Morrison 2012:1). Holyoak and Morrison argue that thinking and reasoning are the defining attributes of being human and that incapacity to think or reason has a far greater impact upon us than loss of language or sensory processing capabilities. There is relatively little literature examining thinking and reasoning in autism spectrum disorder (ASD), which is surprising as early reports have noted a capacity to hold forth, like a “little professor,” on their favorite subject or area of expertise, often failing to detect that their listener may have long since become bored of hearing more on the subject (e.g., Gillberg 1991). Thus, ASD has frequently been characterized in terms of relative strengths and weaknesses in different aspects of cognition. For those with ASD, their strengths (including their favorite subjects and areas of expertise) often fall within nonsocial domains and weaknesses within social domains, which may relate to the two diagnostic criteria of restricted, repetitive patterns of behavior, interests, or activities and deficits in social communication and interaction (respectively; APA 2013). These relative strengths and weaknesses in ASD can be understood within the framework of Dual Process Theory, which has been a dominant model within psychology for over 50 years.

Two types of thinking and reasoning are proposed within Dual Process Theory. Type 1 is also termed intuitive reasoning, and is autonomous and typically involves rapid, effortless, parallel, non-conscious processing that is independent of working memory and cognitive ability. Type 2 is also termed deliberative reasoning, and involves slower, effortful, sequential, conscious processing and is heavily dependent on working memory and related to individual differences in general cognitive ability. Intuitive processes are assumed to yield default responses in most situations, unless intervened upon by distinctive higher order deliberative processes. The idea that intuitive processing is the default and generally precedes deliberative processing is known as the ‘default-interventionist position’ (see Evans and Stanovich 2013; Kahneman 2011). Effective social communication and interaction is argued to be typically supported by more automatic, rapid and effortless

intuitive processing which helps to promote prosocial behaviour, such as cooperation (Rand et al. 2012).

Current Knowledge

The Dual Process Theory of Autism proposes that thinking and reasoning in ASD can be characterized as involving relative strengths in the typically slow and conscious deliberative processing, combined with a relative weakness in the typically rapid intuitive processing (Brosnan et al. 2016a, 2017). Luke et al. (2012) used self-report methodology to identify three core features of thinking and reasoning that were particularly problematic for people with ASD: (1) difficulties when talking to others; (2) problems related to changes in routine; and (3) difficulties when thinking or reasoning had to be undertaken quickly (e.g., quickly making a decision). While difficulties talking with others and changes in routine reflect the core diagnostic features of ASD, the difficulty with rapid thinking and reasoning for those with ASD does not currently reflect within the diagnostic criteria. Applying Dual Process Theory to autism (Brosnan et al. 2016a, 2017), difficulties making decisions that involve talking with others and rapid processing would reflect diminished intuitive processing, whereas difficulties making decisions that involve a change of routine would reflect relatively dominant deliberative processing. Consistent with this, behaviorally, people with ASD request more information prior to making a decision upon a probabilistic reasoning task compared to controls, a style of reasoning that has been termed a “circumspect reasoning bias” (Brosnan et al. 2014a). Intuitive processing is argued to be evidenced by “the framing effect” in which logical reasoning is influenced by the context of the reasoning task. De Martino et al. (2008: 10,746) report a decreased susceptibility to the framing effect in people with ASD, who demonstrate an “unusual enhancement in logical consistency.” Together, these research findings show a style of reasoning in ASD characterized by dominant deliberative processing alongside diminished intuitive processing.

One of the most widely used behavioral assessments of intuition and deliberation is the cognitive reflections test (CRT; Frederick 2005). The CRT comprises reasoning questions that have both an intuitive (incorrect) and deliberative (correct) response. An example includes the bat and ball problem: “A bat and ball together cost \$1.10. If the bat costs \$1 more than the ball, how much does the ball cost? . . .” People typically respond to the CRT questions with a majority of intuitive responses (Frederick 2005), which is theorized to reflect output from initial intuitive reasoning processes which have not been overridden by deliberative reasoning mechanisms. The overriding of initial intuitive reasoning by subsequent deliberative reasoning is demonstrated by achieving the correct answer. In the bat and ball problem, most people provide the intuitive response of 10 cents and a small proportion of people provide the deliberative response of 5 cents. There is evidence to suggest that those from the general population who respond with the deliberative response, do so by having overridden the intuitive response (Travers et al. 2016). Consistent with this, experimental manipulations designed to encourage participants to engage in deliberative reasoning (e.g., by taking longer time to respond) reduces intuitive responses (Evans and Curtis-Holmes 2005). Brosnan et al. 2016a, 2017 used the CRT and found that participants with ASD were significantly more likely to provide deliberative responses than the control participants, who instead were more likely to provide intuitive responses. This style of greater deliberative responding in ASD was not related to measures of IQ in those studies, showing enhanced deliberative thinking and reasoning in ASD is not just explained by differences in general ability. Importantly, the Dual Process Theory of Autism proposes a predisposition in ASD towards greater deliberative processing and less intuitive processing. However, while someone with ASD may have a reasoning style predisposed them to engage in more deliberative processing when thinking and reasoning, the output from the excessive deliberation may not necessarily be correct when attempting tasks, such as assessments of IQ. The Dual Process Theory of Autism proposes

a drive towards deliberation during thinking and reasoning, rather than greater deliberative ability per se.

Within the CRT, deliberative responses actually represent correct responses while the intuitive answers are wrong answers. Therefore, a drive towards deliberative reasoning can be seen as a potential advantage for those with ASD in such contexts. Since the intuitive responses are incorrect in the CRT, the extent to which the CRT can be viewed as a measure of intuitive ability has been questioned by Pennycook et al. (2016), who instead suggest it may better be viewed as a measure of intuitive preference and argue that the CRT is a unique predictor of susceptibility to biases. As biases occur through the application of automatic, typically rapid and effortless intuitive processes rather than engaging in further effortful deliberative processing, the CRT can be seen as a particularly potent measure of “miserly” cognitive processing (or “lazy thinking,” Kahneman 2011: 48). From this perspective, ASD is associated with not being cognitively miserly (or lazy thinking), or perhaps with a drive within thinking and reasoning towards being correct or having the correct response (see Brosnan et al. 2016b). Therefore, the bias towards deliberation is best characterized as being unbiased, as it is not influenced by context, at least within the context of the CRT. This has parallels with views of autism characteristics as differences in “style” of processing, rather than a deficit (e.g., Happé 1999). From this perspective, the thinking and reasoning style characteristic of ASD is biased towards deliberative processes, indicating that reasoning is less susceptible to potentially erroneous biases in ASD (Brosnan et al. 2016a, 2017; De Martino et al. 2008).

Despite the benefits of deliberative processing and the susceptibility to bias and error inherent to intuitive processing, intuition can be invaluable and indeed necessary in many instances requiring a quick decision. This is especially true in dynamic social situations in real life. Many social and emotional processes which support prosocial behavior are typically associated with intuitive processing because they involve rapidly changing situations requiring fast judgments and responses

(Evans and Stanovich 2013; Rand et al. 2012). A reasoning bias characterized by reduced intuitive processing may result in social difficulties consistent with those seen in ASD. For example, when recounting a story, someone might perceive quick and subtle cues of boredom in others, and so intuitively alter the story to suit the audience's mood. They might adapt quickly to the situation, either by making it more interesting or by ending it quickly. If someone with ASD (such as the "Little Professor" described above) noticed the same cues while recounting a story, they might not intuitively make such a fast and automatic alteration to the story. In fact, by utilizing deliberative processing while also trying to recount the story, it might actually lead to an even longer and more laborious story, which would exasperate the situation further. In this example, social communication and interaction difficulties in ASD can be related to difficulties with automatically utilizing the rapid and effortless intuitive processing, and instead relying more on the slower and more conscious deliberative processing. Consistent with this, Mendelson et al. (2016) suggest that a slower, inefficient, social information processing speed specifically underlies many social deficits commonly found in ASD, such as the ability to develop and maintain friendships. As Darius (2002) recounts: "There is no such thing as adequate delayed social reactions. One is either quick enough to keep up, or one is weird and socially disabled." Rapidly and automatically extracting emotional information from social environments is argued to be an intuitive process that feeds "downstream" empathy processes and related social-emotional functioning (Kahneman 2011). Thus, the unbiased use of deliberation proposed by the Dual Process Theory of Autism may relate to the social-emotional weaknesses which form part of the diagnostic criteria for ASD. Similarly, intuitive processing can produce a rapid and automatic sense of a "bad feeling" about someone else and lead to behaviors that minimize the risk of being bullied, exploited, or harmed. However, reduced intuitive processing in ASD might not automatically produce these "gut feelings" about others, which may be related to higher levels of bullying reported in ASD (see Maïano et al.

2016). In this situation, deliberative processing is less likely to produce this "bad feeling," resulting in greater vulnerability to harm in ASD.

A relative bias towards deliberative processing within the Dual Process Theory of Autism may also provide an account of the strengths associated with ASD in the literature, including pattern recognition and attention to detail (e.g., Baron-Cohen et al. 2009). Such capabilities are typically highly visual-spatial in nature and have been found to correlate with self-report and behavioral assessments of deliberative processing, such as the CRT (Brosnan et al. 2014b). It seems plausible to speculate that a greater attention to detail and appreciation of patterns within incoming data (e.g., visual-spatial information) would require more conscious processing resources (e.g., working memory) and therefore increase the likelihood of overriding of initial, potentially biased and erroneous, intuitive processing, resulting in a bias towards more deliberative processing. Although not causal, consistent with this, those with heightened attention to detail also have exceptional working memory capacity (i.e., non-autistic child prodigies, Ruthsatz and Urbach 2012). Weak Central Coherence accounts of ASD (e.g., Happé 1999) also embrace greater attention to detail to describe a local processing bias relative to processing the context of the global picture. Evans and Stanovich (2013) identify contextualized processing as a typical correlate of intuitive processing and decontextualized abstract reasoning is typical of deliberative processing, although this is not a defining feature as not all deliberative processing is context-free. The visual-spatial processing of global, gist, or gestalt stimuli has been found to be compromised in ASD (e.g., Brosnan et al. 2004), which may be relevant to typically rapid intuitive, contextualized visual processing. Within the Reverse Hierarchy Theory of visual perception, an initial rapid processing of low spatial frequency information for gist (vision at a glance) is proposed to precede a slower detailed processing of high spatial frequency (vision with scrutiny; see Vanmarcke et al. 2016). ASD may be characterized early on by a bias towards processing high-spatial visual information (Kéïta et al. 2014). Thus, there may be a

series of theoretical models that can be linked to provide an account of ASD characterized as vision with scrutiny leading to greater attention to detail, which in turn requires further processing that leads to a bias favoring deliberative processing. However, there are still outstanding questions. For example, whether Weak Central Coherence is a result of compromised global processing or enhanced local processing (see also Enhanced Perceptual Theory, Plaisted 2001). Consistent with the visual-spatial literature, there are at least two potential explanations for the pattern of a bias towards deliberative relative to intuitive processing in ASD: (1) those with ASD have impaired intuitive mechanisms and consequently deliberative reasoning is dominant; (2) intuitive mechanisms are intact but dominated by deliberative reasoning. It will be interesting to explore in future research if relative weakness in global visual-spatial processing and/or relative strengths in visual spatial local processing align with relative weakness in intuitive processing and/or relative strengths deliberative processing.

The Dual Process Theory of Autism would predict an increased propensity to engage in deliberative processing is situations that typically engage intuitive processing in the general population. For example, emotion recognition is often a rapid and effortless intuitive process in the general population, but people with ASD are reported to utilize more conscious and effortful processing to read the emotional expressions of others (e.g., “corners of mouth turned down + lowered eyebrows = sad”: Rutherford and McIntosh 2007; see also Brosnan et al. 2015). Dual Process Theory would predict that more intuitive tasks, such as emotion recognition, would be independent of working memory and cognitive abilities in a general population. The Dual Process Theory of Autism would predict that people with ASD would utilize deliberative reasoning over intuitive processes, such that emotion recognition capabilities in ASD would relate to working memory or general cognitive abilities (see Harms et al. 2010, for review of the evidence for this). We would predict that those with higher IQs within the ASD population would have greater deliberative capabilities, which may enable the development of

deliberative processing to overcome diminished intuitive processing in some contexts and tasks. However, it will not always be possible for those with ASD to utilize strengths in deliberative processing to overcome or compensate for weaknesses in intuition, such as in the example with the “Little Professor” above.

Future Directions

In terms of potential support or intervention, the Dual Process Theory of Autism would predict that structuring environments so they better support deliberative processing would be most beneficial for those with ASD. For example, physically slowing down incoming stimuli would enhance the performance of those utilizing deliberative reasoning strategies (Tardif et al. 2007). In addition, making content, processes, and communications both sequential in nature (rather than many things happening at once) and explicit (rather than implicit) to better enable conscious processing of relevant information would also enhance the performance of people with ASD by utilizing their deliberative reasoning strategies. Within the general population, the extent to which individuals engage in intuitive or deliberative reasoning has been found to be susceptible to manipulation. For example, being told to “think carefully” or to write down details of how you came to a decision have been found to elicit more deliberative responses, where as being instructed to go with a “gut-feeling” has been found to elicit more intuitive responses (Dijkstra et al. 2012). These are important future considerations, as if reasoning in ASD is characterized by diminished intuitive mechanisms, whether manipulations such as “go with your gut feeling” would affect those with ASD is an open question future research. Pennycook et al. (2016) suggest that “intuitive” individuals may or may not detect the need to think analytically about the problem, but they decide nonetheless to “go with their gut.” Knowing what your “gut is telling you” is a form of interoception. More formerly, interoception is defined as the integration of internal sensations of bodily arousal in the processing of emotional states. It has recently been suggested

that deficits in interoception may relate to many characteristics of autism (Ondobaka et al. 2017). Future research can establish the link between interoception, “gut-feelings,” and intuitive processing.

There are currently few if any measures of intuitive ability (Pennycook et al. 2016). The CRT index of intuitive preference is assessed through providing responses that are actually wrong, for example. Many assessments of IQ are strongly associated with assessments of deliberative ability (see Pennycook et al. 2016). Thus an assessment of intuition that is independent of deliberation would be welcome. Similarly measures that separate out drive and ability for both intuition and deliberation would allow for further exploration of the extent to which ASD is characterized by a drive towards deliberation rather than deliberative reasoning ability per se. Finally, if rapid, effortless, parallel, nonconscious processing (intuition) is not undertaken within social situations in autistic people, it would be fascinating to identify if rapid, effortless, parallel, non-conscious processing was undertaken in nonsocial situations – for example, having an “intuition” about how a mechanical system may be fixed. When thinking about systems, Baron-Cohen defines a “systemizing” as the drive to analyse or construct systems. What defines a system is that it follows rules, and when we systemize, we are trying to identify the rules that govern the system, in order to predict how that system will behave (Baron-Cohen 2003; Baron-Cohen et al. 2009). Within Baron-Cohen’s model, systemizing is distinct from social-emotional processing (termed empathizing). This would suggest that an “intuition” about how a mechanical system may be fixed, or a drive to “intuitively” systemize (e.g., Baron-Cohen 2003: 3), may be distinct from the intuition characterized by typical intuitive processing (see Brosnan et al. 2013, 2014b). Future research can explore the potential relationships between a drive to systemize and a bias towards deliberation. Initial data from both Baron-Cohen and our lab indicates that self-reported systemizing positively correlates with self-reported deliberation but only the latter correlates with actual reasoning performance. Speculatively, Singleton

et al. (2014) have found early evidence that the physiological arousal associated with social stimuli in the general population is associated with nonsocial stimuli of special interest in those with ASD. If such physiological responses relate to “gut feelings” and intuitive processing, the Dual Process Theory of Autism may develop in future to provide a detailed account of the different contexts within which people with and without ASD engage in intuitive and deliberative processing.

In summary, ASD can be characterized by a style of thinking involving greater deliberative over intuitive reasoning, which can be beneficial in some contexts, such as processing information in an unbiased manner. However, this reasoning style is more effortful and time-consuming and therefore not as effective within the fast moving social world. A bias away from intuitive processing and towards deliberative processing, relates to difficulties in social processing in real-time and the Dual Process Theory of Autism may not only account for thinking and reasoning in ASD but also relate to the social communication and interaction diagnostic criteria. The extent to which greater deliberative thinking and less intuitive thinking may relate to restricted and repetitive interests, activities, and behaviors can also be explored in future research.

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: APA.
- Baron-Cohen, S. (2003). *The essential difference. Male and female brains and the truth about autism*. New York City: Basic Books.
- Baron-Cohen, S., Ashwin, E., Ashwin, C., Tavassoli, T., & Chakrabarti, B. (2009). Talent in autism: Hyper-systemizing, hyper-attention to detail and sensory hypersensitivity. *Philosophical Transactions of the Royal Society B – Biological Sciences*, 364(1522), 1377–1383.
- Brosnan, M. J., Scott, F. J., Fox, S., & Pye, J. (2004). Gestalt processing in autism: failure to process perceptual relationships and the implications for contextual understanding. *Journal of Child Psychology and Psychiatry*, 45, 459–469.
- Brosnan, M., Ashwin, C., & Gamble, T. (2013). Greater empathizing and reduced systemizing in people who show a jumping to conclusions bias in the general

- population: Implications for psychosis. *Psychosis*, 5, 71–81.
- Brosnan, M., Chapman, E., & Ashwin, C. (2014a). Adolescents with autism spectrum disorder show a circumspect reasoning bias rather than 'jumping-to-conclusions'. *Journal of Autism and Developmental Disorders*, 44, 513–520.
- Brosnan, M., Hollinworth, M., Antoniadou, K., & Lewton, M. (2014b). Is empathizing intuitive and systemizing deliberative? *Personality and Individual Differences*, 66, 39–43.
- Brosnan, M., Johnson, H., Grawmeyer, B., Chapman, E., & Benton, L. (2015). Emotion recognition in animated compared to human stimuli in adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45, 1785–1796.
- Brosnan, M., Lewton, M., & Ashwin, C. (2016a). Reasoning on the autism spectrum: A dual process theory account. *Journal of Autism and Developmental Disorder*, 46, 2115–2125.
- Brosnan, M., Johnson, H., Grawmeyer, B., Chapman, E., Antoniadou, K., & Hollinworth, M. (2016b). Deficits in metacognitive monitoring in mathematics assessments in learners with autism spectrum disorder. *Autism*, 20, 463–472.
- Brosnan, M., Ashwin, C., & Lewton, M. (2017). Brief report: Intuitive and reflective reasoning in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47, 2595–2601.
- Darius. (2002). Darius's essay. In D. Prince-Hughes (Ed.), *Aquamarine Blue 5: Personal stories of college students with autism* (pp. 9–42). Athens: Swallow Press.
- De Martino, B., Harrison, N. A., Knafo, S., Bird, G., & Dolan, R. J. (2008). Explaining enhanced logical consistency during decision making in autism. *The Journal of Neuroscience*, 28, 10,746–10,750.
- Dijkstra, K. A., van der Pligt, J., van Kleef, G. A., & Kerstholt, J. H. (2012). Deliberation versus intuition: Global versus local processing in judgment and choice. *Journal of Experimental Social Psychology*, 48, 1156–1161.
- Evans, J. S. B., & Curtis-Holmes, J. (2005). Rapid responding increases belief bias: Evidence for the dual-process theory of reasoning. *Thinking & Reasoning*, 11, 382–389.
- Evans, J. S. B., & Stanovich, K. E. (2013). Dual-process theories of higher cognition advancing the debate. *Perspectives on Psychological Science*, 8, 223–241.
- Frederick, S. (2005). Cognitive reflection and decision making. *Journal of Economic Perspectives*, 19, 25–42.
- Gillberg, C. (1991). Clinical and neurobiological aspects of Asperger syndrome in six family studies. In U. Frith (Ed.), *Autism and Asperger syndrome* (pp. 122–146). Cambridge: Cambridge University Press.
- Happé, F. (1999). Autism: cognitive deficit or cognitive style? *Trends in Cognitive Sciences*, 3, 216–222.
- Harms, M. B., Martin, A., & Wallace, G. L. (2010). Facial emotion recognition in autism spectrum disorders: A review of behavioral and neuroimaging studies. *Neuropsychology Review*, 20, 290–322.
- Holyoak, K. J., & Morrison, R. G. (2012). Thinking and reasoning: A reader's guide. In K. J. Holyoak & R. G. Morrison (Eds.), *The Oxford handbook of thinking and reasoning*. New York: Oxford University Press.
- Kahneman, D. (2011). *Thinking, fast and slow*. London: Penguin.
- Kéïta, L., Guy, J., Berthiaume, C., Mottron, L., & Bertone, A. (2014). An early origin for detailed perception in autism spectrum disorder: biased sensitivity for high-spatial frequency information. *Scientific Reports*, 4, 5475.
- Luke, L., Clare, I. C. H., Ring, H., Redley, M., & Watson, P. (2012). Decision-making difficulties experienced by adults with autism spectrum conditions. *Autism*, 16, 612–621.
- Maïano, C., Normand, C. L., Salvat, M.-C., Moullec, G., & Aimé, A. (2016). Prevalence of school bullying among youth with autism spectrum disorders: A systematic review and meta-analysis. *Autism Research*, 9, 601–615.
- Mendelson, J. L., Gates, J. A., & Lerner, M. D. (2016). Friendship in school-age boys with autism spectrum disorders: A meta-analytic summary and developmental, process-based model. *Psychological Bulletin*, 142(6), 601–622.
- Ondobaka, S., Kilner, J., & Friston, K. (2017). The role of interoceptive inference in theory of mind. *Brain and Cognition*, 112, 64–68.
- Pennycook, G., Cheyne, J. A., Koehler, D. J., & Fugelsang, J. A. (2016). Is the cognitive reflection test a measure of both reflection and intuition? *Behavior Research Methods*, 48, 341–348.
- Plaisted, K. (2001). Reduced generalization in autism: An alternative to weak central coherence. In J. A. Burack, T. Charman, N. Yirmiya, & P. R. Zelazo (Eds.), *The development of autism: Perspectives from theory and research* (pp. 149–171). New Jersey: Lawrence Erlbaum Associates, Inc..
- Rand, D. G., Greene, J. D., & Nowak, M. A. (2012). Spontaneous giving and calculated greed. *Nature*, 489, 427–430.
- Rutherford, M. D., & McIntosh, D. N. (2007). Rules versus prototype matching: Strategies of perception of emotional facial expressions in the autism spectrum. *Journal of Autism and Developmental Disorders*, 37, 187–196.
- Ruthsatz, J., & Urbach, J. (2012). Child prodigy: A novel cognitive profile places elevated general intelligence, exceptional working memory and attention to detail at the root of prodigiousness. *Intelligence*, 40, 419–426.
- Singleton, C. J., Ashwin, C., & Brosnan, M. (2014). Physiological responses to social and nonsocial stimuli in neurotypical adults with high and low levels of autistic traits: Implications for understanding nonsocial drive in autism spectrum disorders. *Autism Research*, 7, 695–703.

- Tardif, C., Lainé, F., Rodriguez, M., & Gepner, B. (2007). Slowing down presentation of facial movements and vocal sounds enhances facial expression recognition and induces facial-vocal imitation in children with autism. *Journal of Autism and Developmental Disorders*, 37, 1469–1484.
- Travers, E., Rolison, J. J., & Feeney, A. (2016). The time course of conflict on the cognitive reflection test. *Cognition*, 150, 109–118.
- Vanmarcke, S., Van Der Hallen, R., Evers, K., Noens, I., Steyaert, J., & Wagemans, J. (2016). Ultra-rapid categorization of meaningful real-life scenes in adults with and without ASD. *Journal of Autism and Developmental Disorders*, 46, 450–466.

Recall

► Retrieval of Information

Recency Effect

► Primacy Effects

Recency Effects

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Definition

The *recency effect* is a phenomenon of memory in which individuals recall items presented at the end of a list better than items presented in the middle of the list and (sometimes) the beginning of the list. Moreover, when asked to recall events from a list, people have a tendency to start with words at the end of the list. The recency effect is one component of the serial position effect in which individuals tend to remember items from the beginning (primacy effect) or the end of a list better than the middle of the list. Unlike the primacy effect, the recency effect does not decrease

when the number of items on the list increases. Recency effects are strongest for verbally, rather than visually, presented items. The recency effect consistently occurs on immediate free recall tasks but is reduced on delayed free recall tasks when an individual is given a task to complete in the period between learning and recall.

Individuals with autism spectrum disorders (ASD) have different strengths and weaknesses in memory tasks depending on their verbal ability. Individuals with ASD who have higher verbal/intellectual abilities (sometimes called “high functioning”) perform just as well as typically developing individuals on free recall tasks and exhibit the expected primacy and recency effects. Furthermore, individuals with ASD with lower verbal/intellectual abilities are impaired in memory tasks; they do not display primacy effects but remember an overall similar amount of items due to an enhanced recency effect.

See Also

- Memory
- Primacy Effects

References and Reading

- Bjork, R. A., & Whitten, W. B. (1974). Recency-sensitive retrieval processes in long-term free recall. *Cognitive Psychology*, 6, 173–189. [https://doi.org/10.1016/0010-0285\(74\)90009-7](https://doi.org/10.1016/0010-0285(74)90009-7).
- Boucher, J. (1981). Immediate free recall in early childhood autism: Another point of behavioural similarity with amnesic syndrome. *British Journal of Psychology*, 72, 211–215. <https://doi.org/10.1111/j.2044-8295.1981.tb02177.x>.
- Bowler, D. M., Limoges, E., & Mottron, L. (2009). Different verbal learning strategies in autism spectrum disorder: Evidence from the Rey auditory verbal learning test. *Journal of Autism and Developmental Disorders*, 39, 910–915. <https://doi.org/10.1007/s10803-009-0697-4>.
- Mottron, L., Morasse, K., & Belleville, S. (2001). A study of memory functioning in individuals with autism. *The Journal of Child Psychology and Psychiatry*, 42, 253–260. <https://doi.org/10.1111/1469-7610.00716>.
- Murdock, B. B. (1962). The serial position effect of free recall. *Journal of Experimental Psychology*, 64, 482–488. <https://doi.org/10.1037/h0045106>.

Recent Developments in Understanding Friendship of Children and Adolescents with Autism Spectrum Disorders

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Definition

The conceptual definition of friendship can be summed up as “stable, frequent, and interconnected affective interactions that are manifested by certain classes of behavioural markers (e.g., sharing, play and conversational skills) that facilitate the functions of companionships, intimacy, and closeness” (Bauminger et al. 2008, p. 136).

Historical Background

The impairments in the areas of social interaction and communication, which are core features of autism spectrum disorder (ASD), and problems with emotional and social reciprocity (Fuentes et al. 2012) may result in difficulties in establishing peer relationships appropriate to the child’s developmental level. A growing body of literature has now documented some important differences and similarities in behavioral and affective manifestation of friendships, between typical children and those with ASD. This area of research is rapidly developing, and several recent developments in children and adolescents with ASD will be reviewed.

Current Knowledge

Congruency in Friendship Quality Perception

Friendship by its nature involves social exchanges that are voluntary, reciprocal, and mutual. The dyadic nature of friendship necessitates that the

viewpoints of both dyad members be taken into consideration. The majority of existing studies on friendship have looked at the pattern of friendship of children with ASD as compared to typical children but have not taken the perspectives of the friend into consideration. Two outcome measures have been used to analyze the dyadic nature of friendship, namely, level of dyadic reciprocity and congruency of the friendship.

Dyadic reciprocity is a measure of how each dyad member judges the other participants in terms of their level of friendship (e.g., best friends, friends, non-friend). Dyadic reciprocity has been explored in several studies with both typical children and children with ASD (Chamberlain et al. 2007; Kasari et al. 2011; Rotheram-Fuller et al. 2010). They demonstrated that children and adolescents with ASD had a consistently lower level of reciprocity across nominations of their top three friends and best friends as compared to typically developing matched peers. These results suggest that children with ASD may often perceive friendship relations differently to those they nominate as friends.

Friendship congruency is a comparative measure on how each individual in the dyad views his/her friendship. The analysis of congruency in friendship quality is deemed important since an individual’s perception of his/her friendship quality often impacts on both their own behavior toward their partner and also their evaluations of their friend’s behavior (Furman 1996). It is therefore possible that a difference in perceptions may place a relationship at risk of breakdown. Furthermore, a lack of congruence may also indicate that the perceptions of children with ASD may not accurately reflect the nature of the relationship.

The only study on friendship congruency in children with ASD was done by Petrina et al. (2016). In their study, perceptions of friendship quality of children with ASD were compared to their nominated friends within the same relationships. There were two groups of friendship types. The non-mixed group comprised those where the nominated friend had ASD and mixed group those where the child with ASD nominated a typically developing peer. The Friendship Quality Questionnaire (FQQ; Parker and Asher 1993) was administered to a total of 45 children with ASD

(mean age = 8.5; SD = 0.9) and their nominated friends.

Calculation of the absolute differences in scores between the members of the dyads gave an indication of the magnitude of differences across different dimensions of friendship quality (namely, companionship and recreation, conflict resolution, conflict and betrayal, help and guidance, intimate exchange, and validation and caring) across the dyad partners. In comparison with their nominated friends, children with ASD had substantial differences in perceptions of their friendship quality. Interestingly, the disability status of the dyad partner had no effect on the level of friendship congruency reported. However, when averaged across both groups (mixed and non-mixed dyads), the largest discrepancies were observed in the areas of conflict resolution and intimate exchange. This indicates that the greatest difference in perspectives centered around the concept of how disagreements were resolved and also differences in perspectives on the degree of intimate disclosure of personal information and feelings. This direct approach in quantifying the difference in the perspectives of friendship quality across the friends can give us clearer insight into which area of social skills can be improved for greater positive friendship experience.

Friendship Satisfaction

While extensive research has provided documentation of quantitative differences in the quality of friendships involving children with ASD (Petrina et al. 2014), there has been limited research explicitly examining the degree to which individuals are satisfied with friendships. Given that conceptualization of friendship may be different in children with ASD, it is possible that, while quantitatively different, their relationships may be perceived as satisfying in the sense that they meet individual needs and expectations (Calder et al. 2013; Petrina et al. 2017).

Calder et al. (2013) conducted research with children with ASD aged between 9 and 11 years. These children rated friendship quality as lower than typically developing peers, particularly affective dimensions, consistent with previous findings. In qualitative interviews, however, all but one of the children reported that they were satisfied with their friendships. In addition, Calder

et al. (2013) reported wide variation in the social motivation of children with ASD with regard to friendship formation.

While the level of satisfaction of children with ASD is of interest, the long-term maintenance of friendships is likely to be dependent on the level of satisfaction of both members of a dyad. Petrina et al. (2017) examined this possibility by investigating the friendship satisfaction of 77 children aged 5–10 years with ASD and their nominated friends. Children with ASD were either involved in mixed dyads with a typically developing friend or non-mixed dyads with another child with ASD. Across both types of dyads, friendship satisfaction ratings were relatively high. In addition, there was a relatively high level of agreement regarding satisfaction ratings within friendship dyads.

While existing research has been encouraging with regard to friendship satisfaction, it should be noted that it has been conducted with preadolescent children. It is possible that these findings may not hold for adolescents, where constructs such as intimacy and self-disclosure may be more important (Petrina et al. 2017).

Friendship Expectations

It has been previously noted that while friendships involving children with ASD are often qualitatively different from typical friendships, children with ASD often report that they are satisfied with their relationships. This raises the question of whether individuals with ASD have different expectations of their friendships, and this issue has been investigated, directly or indirectly.

Researchers have examined the issue of expectations indirectly by asking children to define or describe friendship (e.g., Bauminger and Kasari 2000) and by using assessments addressing understanding of friendship characteristics (e.g., Bauminger et al. 2004). While there have been variations, overall, individuals with ASD have tended to focus less on intimacy and affection and more on companionship. For example, O'Hagan and Hebron (2017) reported a small-scale qualitative study of friendship perceptions, involving three adolescents with ASD as well as a parent for each student and teachers. Key findings were that students did not provide a comprehensive definition of friendship, specifically failing to mention intimacy

and focusing on companionship and helping. O'Hagan and Hebron (2017) speculate that individuals with ASD may "place a higher value on specific aspects of friendship such as reciprocal help and support compared with other factors such as intimacy and duration" (p. 320).

An interesting recent example of an indirect approach providing insight into friendship expectations was provided by Bottema-Beutel et al. (2018). They examined differences in perceptions and responses to vignettes depicting friendship transgressions (e.g., revealing a secret or not returning phone calls) in mentally age-matched groups of third to fifth grade children with ASD ($n = 20$) or typical development ($n = 21$). Overall, there were relatively few statistically significant differences across perception of motivations, goals, or response types between the groups and no differences in ratings of the severity of transgressions. To the extent that perceptions and reactions to friendship transgressions reflect underlying expectations of friendships, the results of this study would suggest differences are relatively minor, but this study had limited statistical power and larger scale replication is needed.

Thus, most existing research providing insight into expectations relies on inference from such measures as friendship definitions or descriptions. An exception is the recent study of Bottema-Beutel et al. (2019), where expectations were more explicitly addressed. Bottema-Beutel et al. (2019) directly asked about 12 friendship expectations with the previously described sample. They found that expectations were very similar between groups with the exception that friends should express care, which was higher in the ASD group. A significant and moderate positive correlation was found between overall friendship quality and expectations in children with ASD but not for the typically developing group. Friendship expectation has also been associated with friendship satisfaction (Petrina et al. 2017), so understanding expectations, from both sides of a relationship, may be important in developing strategies to facilitate stable friendships.

Gender Difference(s) in Friendship

To date there has been little research on friendships of females with ASD, and even less

comparing friendships of males and females with ASD (Cook et al. 2018). It seems that generally females who are more intellectually able have better communication and social skills than males (Sturrock et al. 2019). This may translate into more typical friendship patterns for adolescent girls as they spend more time talking to friends, developing reciprocal relationships with one or two more intimate friends (Cook et al. 2018; Foggo and Webster 2017). Indeed, Sedgewick and colleagues believe gender is a larger determinant of friendship characteristics in adolescents with ASD than is the ASD diagnosis itself (Sedgewick et al. 2016, 2018). Although some researchers have found that the friendships of adolescent girls with ASD are similar to those of typically developing girls, they do have more difficulties with social communication and dealing with conflict. They may abandon a friendship, rather than resolving conflict. They may also be more prone to mask their ASD and remain more as observers, withdrawing from conflict (Moyses and Porter 2015). Other researchers have found that adolescent girls with ASD may be more likely to form friendships with boys with ASD, on the basis of shared interests and activities, or with other girls with ASD or other disabilities rather than with typically developing girls (Cook et al. 2018; Cridland et al. 2014).

Boys with ASD, on the other hand, like typically developing adolescent males, tend to focus more on shared activities, have a larger friendship group, with less emotional sharing and less conflict. They may thus be advantaged in developing more typical friendship patterns based on common activities (Cridland et al. 2014; Sedgewick et al. 2016). Some researchers believe males with ASD are different to both girls with ASD and typically developing males, in that they are less motivated to form emotionally close relationships and are more likely to avoid interactions (Sedgewick et al. 2016). Thus, although boys with ASD may fit into social groups, they are less likely to form deeper and intimate friendships (Mendelson et al. 2016).

Friendship Characteristics Across Developmental Stages

As noted above in the section on gender differences, the nature of friendship in people with ASD

changes over time in ways similar to those who are typically developing (O'Hagan and Hebron 2017). For boys with ASD, shared activities remain a focus, and for girls, intimacy and personal closeness become important. A small number of longitudinal studies with young children have found that ASD-linked difficulties with joint attention and symbolic play predicted aspects of friendships in 8–9 year olds. Those with better joint attention skills as preschoolers were more likely to have closer friendships with less conflict (see, e.g., Freeman et al. 2015).

Some studies have provided insights into changes in friendship patterns over time. Through the early and middle school years, where friendship is linked to shared activities, children with ASD may be well accepted even though they rely more on parental support to form and maintain friendships (Daniel and Billingsley 2010). From early adolescence on, many people with ASD will remain at the periphery of their social groups and experience difficulty making and maintaining friendships (O'Hagan and Hebron 2017; Rotheram-Fuller et al. 2010; Finke et al. 2019).

Future Directions

There has been substantial research comparing friendship characteristics between typically developing children and those with ASD. However, limited attention has been given to examining the perspectives from both dyad members. Given the reciprocal nature of friendship, the study of friendship warrants investigation involving both the dyad members' viewpoints. Recent research studies have also provided indication of similar expectations of friendship of children with ASD and typically developing children. Nevertheless, there are very few studies examining either congruence or expectations, so replication should be a high priority. While there may be quantitative differences in friendship characteristics, recent research has provided an indication that many children with ASD may be satisfied with their friendships. Again, very limited research has been conducted and existing studies have been mostly in preadolescents. Thus, further research to determine the robustness of these findings is a

priority. In typically developing children, the characteristics of friendship evolve predictably over time (Gilfford-Smith and Brownell 2003). We currently have very limited longitudinal data on how friendships in children with ASD differ in their development over extended time periods. While research of this type may be challenging and expensive to conduct, it has the potential to offer substantial insight into the nature of friendship in children and adolescents with ASD, including gender differences.

In addition to the specific suggestions offered to this point, there are some more general issues that should be considered by future researchers. The vast majority of extant friendship studies have provided examination of children in middle childhood who have typical intellectual ability. Many children with ASD have intellectual disabilities (Centers for Disease Control and Prevention 2016), and greater inclusion of this group in research would seem appropriate. In addition, given significant changes in friendship characteristics in typically developing children in the teenage years (Bell 2016), further research in adolescents would seem warranted. Finally, most data on friendship characteristics in current research has been gathered through either interview or written questionnaires. Future researchers should consider making greater use of objective data collection methods including direct observation of friendships in natural settings. Such information has the potential to either verify subjective data or indicate where perceptions differ from actual behavior.

References and Reading

- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development*, 71, 447–456. <https://doi.org/10.1111/1467-8624.00156>.
- Bauminger, N., Shulman, C., & Agam, G. (2004). The link between perceptions of self and of social relationships in high-functioning children with autism. *Journal of Developmental and Physical Disabilities*, 16, 193–214. <https://doi.org/10.1023/B:JODD.0000026616.24896.c8>.
- Bauminger, N., Solomon, M., Aviezer, A., Heung, K., Gazit, L., Brown, J., & Rogers, S. J. (2008). Children with autism and their friends: A multidimensional study of friendship in high-functioning autism spectrum disorder.

- Journal of Abnormal Child Psychology*, 36, 135–150. <https://doi.org/10.1007/s10802-007-9156-x>.
- Bell, B. T. (2016). Understanding adolescents. In L. Little, D. Fitton, B. T. Bell, & N. Toth (Eds.), *Human-computer interaction series. Perspectives on HCI research with teenagers* (pp. 11–27). Cham: Springer International Publishing.
- Bottema-Beutel, K., Malloy, C., Cuda, J., Kim, S. Y., & MacEvoy, J. (2018). Responses to vignettes depicting friendship transgressions: Similarities and differences in children with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 1–12. <https://doi.org/10.1007/s10803-018-3828-y>.
- Bottema-Beutel, K., Malloy, C., Cuda, J., Kim, S. Y., & MacEvoy, J. (2019). Friendship expectations may be similar for mental age-matched children with autism spectrum disorder and typically developing children. *Journal of Autism and Developmental Disorders*, 49, 4346–4354. <https://doi.org/10.1007/s10803-019-04141-7>.
- Calder, L., Hill, V., & Pellicano, E. (2013). ‘Sometimes I want to play by myself’: Understanding what friendship means to children with autism in mainstream primary schools. *Autism*, 17, 296–316. <https://doi.org/10.1177/1362361312467866>.
- Centers for Disease Control and Prevention. (2016). *Community report on autism 2016*. Retrieved from <https://www.cdc.gov/ncbddd/autism/documents/comm-report-autism-full-report.pdf>.
- Chamberlain, B., Kasari, C., & Rotheram-Fuller, E. (2007). Involvement or isolation? The social networks of children with autism in regular classrooms. *Journal of Autism and Developmental Disorders*, 37, 230–242. <https://doi.org/10.1007/s10803-006-0164-4>.
- Cook, A., Ogden, J., & Winstone, N. (2018). Friendship motivations, challenges and the role of masking for girls with autism in contrasting school settings. *European Journal of Special Needs Education*, 33, 302–315. <https://doi.org/10.1080/08856257.2017.1312797>.
- Cridland, E. K., Jones, S. C., Caputi, P., & Magee, C. A. (2014). Being a girl in a boys’ world: Investigating the experiences of girls with autism spectrum disorders during adolescence. *Journal of Autism and Developmental Disorders*, 44, 1261–1274. <https://doi.org/10.1007/s10803-013-1985-6>.
- Daniel, L. S., & Billingsley, B. S. (2010). What boys with an autism spectrum disorder say about establishing and maintaining friendships. *Focus on Autism and Other Developmental Disabilities*, 25, 220–229. <https://doi.org/10.1177/1088357610378290>.
- Finke, E. H., McCarthy, J. H., & Sarver, N. A. (2019). Self-perception of friendship style: Young adults with and without autism spectrum disorder. *Autism and Developmental Language Impairments*, 4, 1–16. <https://doi.org/10.1177/2396941519855390>.
- Foggo, R. S. V., & Webster, A. A. (2017). Understanding the social experiences of adolescent females on the autism spectrum. *Research in Autism Spectrum Disorders*, 35, 74–85. <https://doi.org/10.1016/j.rasd.2016.11.006>.
- Freeman, S. F. N., Gulsrud, A., & Kasari, C. (2015). Brief report: Linking early joint attention and play abilities to later reports of friendships for children with ASD. *Journal of Autism and Developmental Disorders*, 45, 2259–2266. <https://doi.org/10.1007/s10803-015-2369-x>.
- Fuentes, J., Bakare, M., Munir, K., Aguayo, P., Gaddour, N., Oner, O., & Mercadante, M. (2012). Autism spectrum disorders. In J. M. Rey (Ed.), *IACAPAP e-textbook of child and adolescent mental health* (pp. 1–27). Geneva: International Association for Child and Adolescent Psychiatry and Allied Professions. Retrieved from <http://iacapap.org/wp-content/uploads/C.2-AUTISM-SPECTRUM-072012.pdf>.
- Furman, W. (1996). The measurement of children and adolescent’s perceptions of friendships: Conceptual and methodological issues. In W. M. Bukowski, A. F. Newcomb, & W. W. Hartup (Eds.), *The company they keep: Friendships in childhood and adolescence* (pp. 41–65). Cambridge, MA: Cambridge University Press.
- Gillford-Smith, M., & Brownell, C. (2003). Childhood peer relationships: Social acceptance, friendships, and peer networks. *Journal of School Psychology*, 41, 235–284. [https://doi.org/10.1016/S0022-4405\(03\)00048-7](https://doi.org/10.1016/S0022-4405(03)00048-7).
- Kasari, C., Locke, J., Gulsrud, A., & Rotheram-Fuller, E. (2011). Social networks and friendships at school: Comparing children with and without ASD. *Journal of Autism and Developmental Disorders*, 41, 533–544. <https://doi.org/10.1007/s10803-010-1076-x>.
- Mendelson, J. L., Gates, J. A., & Lerner, M. D. (2016). Friendship in school-age boys with autism spectrum disorders: A meta-analytic summary and developmental, process-based model. *Psychological Bulletin*, 142, 601–622. <https://doi.org/10.1037/bul0000041>.
- Moyse, R., & Porter, J. (2015). The experience of the hidden curriculum for autistic girls at mainstream primary schools. *European Journal of Special Needs Education*, 30, 187–201. <https://doi.org/10.1080/08856257.2014.986915>.
- O’Hagan, S., & Hebron, J. (2017). Perceptions of friendship among adolescents with autism spectrum conditions in a mainstream high school resource provision. *European Journal of Special Needs Education*, 32, 314–328. <https://doi.org/10.1080/08856257.2016.1223441>.
- Parker, J. G., & Asher, S. R. (1993). Friendship and friendship quality in middle childhood: Links with peer group acceptance and feelings of loneliness and social dissatisfaction. *Developmental Psychology*, 29, 611–621. <https://doi.org/10.1037/0012-1649.29.4.611>.
- Petrina, N., Carter, M., & Stephenson, J. (2014). The nature of friendship in children with autism spectrum disorders: A systematic review. *Research in Autism Spectrum Disorders*, 8, 111–126. <https://doi.org/10.1016/j.rasd.2013.10.016>.
- Petrina, N., Carter, M., Stephenson, J., & Sweller, N. (2016). Perceived friendship quality of children with autism spectrum disorder as compared to their peers in mixed and non-mixed dyads. *Journal of Autism and Developmental Disorders*, 46, 1334–1343. <https://doi.org/10.1007/s10803-015-2673-5>.
- Petrina, N., Carter, M., Stephenson, J., & Sweller, N. (2017). Friendship satisfaction in children with autism spectrum disorder and nominated friends. *Journal of Autism and Developmental Disorders*, 47, 384–392. <https://doi.org/10.1007/s10803-016-2970-7>.

- Rotheram-Fuller, E., Kasari, C., Chamberlain, B., & Locke, J. (2010). Social involvement of children with autism spectrum disorders in elementary school classrooms. *Journal of Child Psychology and Psychiatry*, 51, 1227–1234. <https://doi.org/10.1111/j.1469-7610.2010.02289.x>.
- Sedgewick, F., Hill, V., Yates, R., Pickering, L., & Pellicano, E. (2016). Gender differences in the social motivation and friendship experiences of autistic and non-autistic adolescents. *Journal of Autism and Developmental Disorders*, 46, 1297–1306. <https://doi.org/10.1007/s10803-015-2669-1>.
- Sedgewick, F., Hill, V., & Pellicano, E. (2018). Parent perspectives on autistic girls' friendships and futures. *Autism and Developmental Language Impairments*, 3. <https://doi.org/10.1177/2396941518794497>.
- Sturrock, A., Yau, N., Freed, J., & Adams, C. (2019). Speaking the same language? A preliminary investigation, comparing the language and communication skills of females and males with high-functioning autism. *Journal of Autism and Developmental Disorders*. Advance online publication. <https://doi.org/10.1007/s10803-019-03920-6>.

Reception

- ▶ [Receptive Language](#)

Receptive Aphasia

- ▶ [Wernicke's Aphasia](#)

Receptive Language

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Synonyms

[Comprehension](#); [Language processing](#); [Language understanding](#); [Reception](#)

Definition

Receptive language is the ability to understand and interpret information that is being conveyed through speech. It also includes an individual's ability to attend/listen to language. Receptive language delay/disorder frequently co-occurs with other developmental delays including autism. Receptive language deficits related to autism may be secondary to cognitive or symbolic deficits, or to the deficits in attention to social cues and stimuli.

See Also

- ▶ [Expressive Language](#)
- ▶ [Listening Comprehension](#)
- ▶ [Verbal Comprehension](#)

References and Reading

- Thurm, A., Lord, C., Lee, L. C., & Newschaffer, C. (2007). Predictors of language acquisition in preschool children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 1721–1734.

Receptive Language Disorders

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Synonyms

[Language comprehension disorder/impairment/disability](#); [Receptive language impairment/disability](#)

Short Description or Definition

Receptive language disorder involves impairment in the ability to understand, or comprehend, language. Deficits may be observed in the

comprehension of oral, written, gestural, and/or symbolic language systems. All levels of language may be affected, including at the word, sentence, and discourse levels. Comprehension of social and nonliteral uses of language may be particularly impaired in individuals with autism spectrum disorders (ASD).

Categorization

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association 2000) defines mixed receptive-expressive language disorder according to four primary criteria (a specific category for receptive language disorders is not included in the DSM-IV-TR, presumably because receptive deficits are typically accompanied by expressive language impairments). The first criterion states that scores received on standardized tests of receptive and expressive language are substantially below those obtained on standardized tests of nonverbal intellectual ability (i.e., discrepancy criterion). Second, difficulties with language interfere with academic, occupational, and/or social functioning. Third, criteria are not met for expressive language disorder or a pervasive developmental disorder. Fourth, the language difficulties must be above and beyond those that are typically associated with environmental deprivation or conditions that may be present in the individual, such as mental retardation, speech-motor impairments, or sensory deficits.

Epidemiology

For individuals with ASD, impairments in social communication (including joint attention, social reciprocity, and language and related cognitive skills) are core features of the disorder (American Speech-Language-Hearing Association (ASHA) 2006). Deficits in social communication skills are related to impairments in receptive language ability. These deficits could range from inability to understand individual words to misinterpreting subtle nuances in a

speaker's intent (e.g., sarcasm, humor). Therefore, it seems reasonable to assume that many individuals with ASD have some degree of receptive language impairment. Although the definition of Asperger's disorder includes no clinically significant delay in language, several researchers question whether language is unaffected in this syndrome (cf. ASHA 2006).

Natural History, Prognostic Factors, Outcomes

The prerequisites for comprehending language begin to develop at birth, if not before. For example, at birth, infants prefer the voice of their mothers over other voices, presumably due to in utero exposure. They are also fascinated by facial expressions and respond to them appropriately.

In the first year of life, typically developing children understand a few words in context. By 18 months, children typically have a receptive vocabulary size of about 100–150 words. Subsequently, children's comprehension of syntactic structure develops as they understand utterances of increasing length and use word-order strategies to facilitate comprehension. Between the ages of 4–8 years old, children have receptive vocabularies of 3,000–8,000 words. Upon learning to read, children's receptive vocabularies increase by about 3,000 words per year, and they are able to comprehend oral and written discourse of increasing complexity (e.g., narratives, expository text).

The ultimate goal in comprehension is to understand the speaker's communicative intention, which (usually) is far more than interpreting an utterance literally. Research indicates that young children are able to infer a great deal about the communicative intention of a speaker before they are able to understand the words and sentences. This ability to use social knowledge to interpret a speaker's intentionality is critical for communicative effectiveness. For example, listeners must be able to understand the preparatory and sincerity conditions for speech acts (e.g., understanding a speaker's intention behind the utterance, "Is the Pope Catholic?"). We must also understand scripts for various communicative

situations. For example, when a waiter asks, “What would you like?” we know he is referring to what is on the menu. The ability to inference is also required given that a great deal of information is not explicitly stated in conversations or written texts (e.g., “I was hungry so I went to the restaurant,” infers that a meal was ordered and eaten). Understanding (or at least making educated guesses) about the psychology of other people helps us appropriately interpret the communicative intents of others.

Receptive language impairments may vary in their symptoms and level of severity. Some individuals may not understand even single words, while others only have difficulty comprehending more advanced forms of language, such as expository discourse or academic texts. Toddlers and preschoolers with early receptive language delays tend to have poorer outcomes than children with only expressive language delays. Receptive language ability is lower in children and individuals with intellectual disability, ASD, and various genetic syndromes where cognitive development is affected. In general, IQ is related to language outcomes (e.g., higher IQs are associated with better language skills). Individuals with ASD may have greater deficits with receptive language than expressive language (especially in the early years), and throughout development, they may show higher rates of improvement in their expressive language abilities compared to their receptive skills.

Clinical Expression and Pathophysiology

According to the American Speech-Language-Hearing Association (ASHA 1993), a language disorder is the “impaired comprehension and/or use of a spoken, written and/or other symbol system. The disorder may involve (1) the *form* of language (phonology, morphology, and syntax), (2) the *content* of language (semantics), and/or (3) the *function* of language in communication (pragmatics) in any combination.” In the case of individuals with receptive language disorders, the comprehension (i.e., reception, understanding) of language is impaired. All levels of language may

be affected, including at the word, sentence, and discourse levels. Most individuals with receptive language disorders also have expressive language impairments, with receptive skills superior to expressive abilities. In individuals with autism, however, receptive language may be more impaired than expressive language. For the entire autism spectrum, comprehension of social and nonliteral uses of language may be particularly impaired, given that the meaning of conversations and other discourse (usually involving language) is closely linked to nonverbal cues (e.g., facial expressions), paralinguistic features (e.g., intonation, emphatic stress, prosody), and other aspects of communication that require social awareness for accurate interpretation.

One of the first signs of ASD is a lack of responsiveness to social interactions. For example, infants later diagnosed with ASD were described by their parents as uninterested in human voices or faces. They rarely engaged in social games such as “peekaboo.” All of these behaviors are important precursors to developing receptive language abilities and effective communication skills. Parents of children with ASD report becoming seriously concerned about their children’s development during the toddler years, especially when their children exhibit a lack of responsiveness to words. In the second year of life, the receptive language abilities of children with ASD are depressed relative to their expressive abilities. The gap tends to narrow over time, with receptive and expressive language abilities becoming more similar by ages 3–4 years.

While a great deal of attention has been paid to the expressive language skills in individuals with ASD (e.g., echolalia, idiosyncratic language), less focus has been given to their receptive language and communication abilities. This is unfortunate given that an early lack of response to language is a strong indicator of autism in infants and toddlers. Persistent delays in language comprehension are a key differentiator between children with high-functioning autism and specific language disorders. Although older children with autism may score similarly on standardized tests of receptive and expressive language, they often appear to

be significantly more impaired in receptive language in terms of everyday functioning.

Individuals with ASD often have difficulty comprehending nonliteral uses of language, such as figurative language and indirect speech acts. Figurative language is language that is used in nonliteral ways in order to achieve a special effect or meaning. Similes, metaphors, idioms, proverbs, and slang are examples of figurative language. Upon hearing the expression “It’s raining cats and dogs,” an individual with ASD may think that the speaker means that cats and dogs are literally falling from the sky. Indirect speech acts (i.e., messages where the intended and literal meanings are different) may also be interpreted literally by individuals with ASD. For example, the utterance, “Can you pass the salt, please,” should not be answered with “yes.” Rather, it is an indirect (i.e., polite) way of asking someone to perform an action. The ability to comprehend and produce nonliteral language is an important area of linguistic development in school-age children. Figurative language is commonly used in a variety of communicative contexts, including casual conversation, teacher talk, and written texts. The meaning of figurative language and indirect speech acts may need to be explicitly taught to individuals with ASD. Many researchers believe that difficulties in understanding nonliteral language and social communication are related to deficits in “theory of mind,” or the ability to conceive of other people’s mental states and perspectives.

Some individuals with ASD, particularly those with normal range IQ, may develop grade-level reading skills, although their ability to decode text is superior to their ability to comprehend what they are reading. Often basic reading comprehension is present for topics that require little social knowledge (e.g., science) but is compromised when the text involves social topics such as emotions, character motivations, deception, and other topics that require social awareness and psychological understanding. Higher-functioning individuals with ASD have persistent deficits in comprehending social conversation, especially when sarcasm, humor, slang, and other nonliteral forms of language are involved.

Evaluation and Differential Diagnosis

Language comprehension in young children is assessed through informal observation, parent report/interview (e.g., *Receptive-Expressive Emergent Language Scale-3rd Edition*; Bzoch et al. 2003), or standardized assessments, although very few are available for children under 3 years of age (e.g., *Communication and Symbolic Behavior and Play Scales-DP*, Wetherby and Prizant 2003; *Peabody Picture Vocabulary Test-4th Edition*, Dunn and Dunn 2007; *Preschool Language Scale-5*, Zimmerman et al. 2011).

Language comprehension in young children tends to be highly context dependent. Children make use of comprehension strategies (e.g., “Do what you normally do”) and other contextual clues which can make them appear to understand more language than they actually do (Chapman 1978). For example, a parent gives her child a doll and a brush and says, “Brush the dolly’s hair.” When the child complies, the parent thinks the child understood the linguistic input, whereas the child may have simply done what comes naturally when given a doll and brush. Therefore, it is important to assess language comprehension without providing contextual clues. Sometimes this is done by asking children to perform unusual actions with objects (e.g., “Kiss the brush”). *The Clinical Assessment of Language Comprehension* (Miller and Paul 1995) is a useful resource which provides a variety of informal assessment procedures.

For older children, adolescents, and adults, numerous standardized tests are available for assessing language comprehension (e.g., *Test for Auditory Comprehension of Language-3rd Edition*, Carrow-Woolfolk 1999; *Clinical Evaluation of Language Fundamentals-4th Edition*, Semel et al. 2003). Additional assessment procedures include criterion referenced assessments (e.g., comprehension of passive sentences, ability to make inferences), informal assessment of complex discourse and written text, and informal observations of communicative ability (e.g., classroom and social situations). A few standardized assessments of pragmatic language skills

(i.e., social use of language) are available (e.g., *Comprehensive Assessment of Spoken Language*, Carrow-Woolfolk 1999; *Test of Language Competence*, Wiig and Secord 1989); however, higher-functioning individuals with ASD may perform well within a structured testing situation. For these individuals, observation within naturalistic communicative contexts may be necessary to identify deficits in the comprehension of social language.

Treatment

The receptive (and expressive) language development of young children with language delays is facilitated by a variety of evidence-based approaches, including prelinguistic milieu teaching, focused stimulation, and conversational recasting. In addition, approaches that focus on parent training, such as It Takes Two To Talk (Hanen Program for Parents), have been shown to be effective.

For individuals with ASD, research has demonstrated that a range of approaches are effective for promoting communication abilities and receptive language skills (cf. ASHA 2006). Common approaches range from naturalistic (e.g., Floor Time, Greenspan et al. 1998) to highly structured, behavioral interventions (Lovaas et al. (1989)). According to the National Research Council (2001), educational interventions for individuals with ASD should begin as early as possible, programming should be intensive with repeated and planned teaching opportunities, teacher-student ratios should be low, mechanisms for ongoing assessment and program evaluation should be in place, and family involvement and training is important. In addition, intervention for individuals with ASD should include spontaneous and functional communication, social skills, play skills, peer interactions, generalization of skills to natural contexts, mechanisms for addressing challenging behaviors, and promoting functional academic skills when appropriate.

For individuals who are literate, written language can be an effective means of communication and can be used for compensatory purposes (e.g., using a script or communication checklist

for functioning within various situations). For example, Social Stories (Gray 1993) use a story format using visual materials to improve an individual with ASD's social understanding of various situations. Technology (e.g., texting, social networking) may assist individuals with ASD in social communication without the stress of face-to-face interactions. Comprehension monitoring strategies for older, higher-functioning individuals with ASD (e.g., checklists) may also be effective. For nonverbal individuals with ASD, the use of augmentative and alternative communication (AAC) strategies is common for facilitating comprehension (and expression). AAC approaches include sign language, Picture Exchange Communication System (PECS), communication boards, and voice output devices.

See Also

- [Language](#)
- [Language Disorder](#)
- [Language Interventions](#)
- [Receptive Language](#)
- [Receptive Vocabulary](#)
- [Semantic Memory](#)
- [Semantic Pragmatic Disorder](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR*. Washington, DC: Author.
- American Speech-Language-Hearing Association (ASHA). (1993). Definitions of communication disorders and variations. *ASHA*, 35(Suppl. 10), 40–41.
- American Speech-Language-Hearing Association (ASHA). (2006). *Guidelines for speech-language pathologists in diagnosis, assessment, and treatment of autism spectrum disorders across the life span* [Guidelines]. Available from www.asha.org/policy. Accessed 18 Apr 2011.
- Bishop, D. V. M. (1997). *Uncommon understanding: Development and disorders of language comprehension in children*. East Sussex: Psychology Press.
- Bzoch, K., League, R., & Brown, V. (2003). *Receptive-expressive emergent language test* (3rd ed.). Circle Pines: American Guidance Service.
- Carrow-Woolfolk, E. (1999). *Test of auditory comprehension of language* (3rd ed.). Austin: Pro-Ed.

- Chapman, R. (1978). Comprehension strategies in children. In J. F. Kavanaugh & W. Strange (Eds.), *Speech and language in the laboratory, school, and clinic* (pp. 308–327). Cambridge, MA: MIT Press.
- Dunn, L. M., & Dunn, D. M. (2007). *Peabody picture vocabulary test* (4th ed.). Circle Pines: American Guidance Service.
- Frombonne, E. (2007). Epidemiological surveys of pervasive developmental disorders. In F. R. Volkmar (Ed.), *Autism and pervasive developmental disorders* (2nd ed., pp. 33–68). New York: Cambridge University Press.
- Graves, M. F. (2006). *The vocabulary book: Learning and instruction*. New York: Teachers College Press.
- Gray, C. (1993). *The social story book*. Arlington: Future Horizons.
- Greenspan, S. I., Wieder, S., & Simons, R. (1998). *The child with special needs: Encouraging intellectual and emotional growth*. Reading: Addison-Wesley.
- Justice, L. M. (2010). *Communication sciences and disorders* (2nd ed.). Boston: Allyn & Bacon.
- Long, S. H. (2005). Language and children with autism. In V. A. Reed (Ed.), *An introduction to children with language disorders* (3rd ed., pp. 253–275). Boston: Allyn & Bacon.
- Lovaas, I., Calouri, K., & Jada, J. (1989). The nature of behavioral treatment and research with young autistic persons. In C. Gillberg (Ed.), *Diagnosis and treatment of autism* (pp. 285–305). New York: Plenum Press.
- McCauley, R. J., & Fey, M. E. (2006). *Treatment of language disorders in children*. Baltimore: Paul H. Brookes Publishing.
- Miller, J. F., & Paul, R. (1995). *The clinical assessment of language comprehension*. Baltimore: Paul H. Brookes Publishing.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press, Committee on Educational Interventions for Children with Autism, Division of Behavioral and Social Sciences and Education.
- Nippold, M. A., & Scott, C. M. (2010). *Expository discourse in children, adolescents, and adults*. New York: Psychology Press.
- Paul, R. (2005). Assessing communication in autism spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, 3rd ed., pp. 799–816). Hoboken: Wiley.
- Paul, R. (2007a). Communication and its development in autism spectrum disorders. In F. R. Volkmar (Ed.), *Autism and pervasive developmental disorders* (2nd ed., pp. 129–155). New York: Cambridge University Press.
- Paul, R. (2007b). *Language disorders from infancy through adolescence: Assessment and intervention*. St. Louis: Mosby Elsevier.
- Paul, R., Chawarska, K., & Volkmar, F. (2008). Differentiating ASD from DLD in toddlers. *Perspectives on Language Learning and Education*, 15, 101–111.
- Semel, E., Wiig, E. H., & Secord, W. A. (2003). *Clinical evaluation of language fundamentals* (4th ed.). San Antonio: The Psychological Corporation.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 335–364). Hoboken: Wiley.
- The Hanen Centre. <http://www.hanen.org/>. Accessed 18 Apr 2011.
- Tomblin, J. B., Records, N. L., Buckwalter, P., Zhang, X., Smith, E., & O'Brien, M. (1997). Prevalence of specific language impairment in kindergarten children. *Journal of Speech, Language, and Hearing Research*, 40, 1245–1260.
- Wetherby, A., & Prizant, B. (2003). *Communication and symbolic behavior scales developmental profile*. Baltimore: Paul H. Brookes.
- Wiig, E. H., & Secord, W. (1989). *Test of language comprehension – expanded edition*. San Antonio: The Psychological Corporation.
- Zimmerman, I. L., Steiner, V. G., & Pond, R. E. (2011). *Preschool language scales* (5th ed.). Bloomington: Pearson.

Receptive Language Impairment/Disability

► Receptive Language Disorders

Receptive Lexicon

► Receptive Vocabulary

Receptive Vocabulary

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Synonyms

Receptive lexicon

Definition

Receptive vocabulary refers to the collection of words that is understood by an individual. Vocab-

ulary can be learned through spoken or written language, sign language (i.e., gestures), or through symbols. Words are learned by first recognizing patterns in the auditory or visual input and then attaching a corresponding meaning. Vocabulary development is dependent upon environmental, experiential, and individual factors. A child's vocabulary size and knowledge significantly impacts his or her comprehension of both oral and written language.

Research suggests that although overall vocabulary knowledge may be a relative strength in individuals with autism spectrum disorders (ASD), notable deficits may exist. For example, the acquisition of words that refer to mental state or emotional concepts may be especially impaired. Individuals with ASD may have difficulty generalizing the meanings of words to new contexts (e.g., understanding that the word "dog" can refer to many animals, not just the family pet). In addition, individuals with ASD often have difficulty comprehending vocabulary in nonliteral language (e.g., slang, figures of speech, proverbs, metaphors).

See Also

- [Peabody Picture Vocabulary Test](#)
- [Receptive Language](#)
- [Receptive Language Disorders](#)
- [Vocabulary](#)

References and Reading

- Capone, N. (2012). Language disorders in children. In H. D. Schwartz (Ed.), *A primer on communicative disorders* (pp. 96–115). Upper Saddle River: Pearson Education.
- Hegde, M. N., & Maul, C. A. (2006). *Language disorders in children: An evidence-based approach to assessment and treatment*. Boston: Allyn & Bacon.
- McAfee, J. G., & Shipley, K. G. (2009). *Assessment in speech language pathology a resource manual* (4th ed.). Clifton Park: Delmar Cengage Learning.
- Nicolosi, L., Harryman, E., & Kresheck, J. (1996). *Terminology of communication disorders: Speech-language-hearing* (4th ed.). Baltimore: Williams and Wilkins.
- Paul, R. (2007). *Language disorders from infancy through adolescence: Assessment and intervention*. St. Louis: Mosby Elsevier.

Receptive-Expressive Emergent Language Test (REEL-3)

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Synonyms

[REEL-3](#)

Description

The *Receptive-Expressive Emergent Language Test, Third Edition* (REEL-3; Bzoch et al. 2003) is a 66-item, standardized, norm-referenced test designed to help identify infants and toddlers who have language impairments or other disabilities that affect language development. It may be used as an assessment and planning instrument in early childhood intervention programs mandated under Education of the Handicapped Act Amendments of 1986 (P.L. 99-457). The REEL-3 has two core subtests, receptive language and expressive language, as well as a supplementary subtest, inventory of vocabulary words. Results are obtained from a caregiver interview. The test can be individually administered in approximately 20 min.

The REEL-3 is based on a tridimensional model of language acquisition regarding early language development as the intentional interaction content, form, and use of language. This model is further divided into four overlapping chronological phases: prelinguistic reflexive communication, first words and transition to intentional language use, rapid vocabulary development and initial sentence formation, and adultlike preschool register. The third dimension of the model divides language acquisition into receptive and expressive use.

Items in the REEL-3 are divided into reflective language and expressive language subtests. Both subtests are comprised of 66 items that are completed by a parent or caregiver. Reflective

language items measure a child's responses to sounds or language. Expressive language items measure a child's oral language production. Raw scores are tallied for each subtest then converted to standard scores. The receptive language ability score (RLAS) and the expressive language ability score (ELAS) may be combined into a single composite score, the language ability (LAS) score. The LAS can be considered an overall measure of language ability.

Historical Background

The REEL was originally published in 1971 to help identify problems in early language development of infants and toddlers aged birth to 3 years. The REEL was a 132-item checklist of developmental language milestones and corresponding age placement. Items on the REEL were developed from observational research of emergent language in a language laboratory and through early language research. The REEL was edited by the clinical work of multidisciplinary teams.

The REEL required the examiner to interview an informant, usually a parent or caregiver, who was familiar with behaviors consistently used by the child. Checklist items were limited to informant-observable behaviors directly related to auditory-oral language. Items on the REEL were grouped by chronological age. The REEL was comprised of a receptive subtest, based on listening responses, and an expressive subtest, based on vocalization. The subtests could be used independently or with a combined composite score as an indication of early language development. The REEL received positive acceptance as an assessment tool and was primarily used in clinical settings to identify children needing early intervention and to help design early intervention programs.

The revised edition of the REEL was published in 1991, responding to the expanded diagnostic need of practitioners due to the Education of the Handicapped Act Amendments of 1986 (P.L. 99-57), which emphasized special education service for children with disabilities. The REEL-2 was developed to assist in screening infants and

toddlers for early learning problems in clinical diagnosis and in planned intervention goals.

In response to reviews of the test, the REEL-2 reduced clinical language and jargon in the informant interviews, improved scoring and interpretation, and added an interview to help assess a child's language environment.

The REEL-3 improves on the theoretical and statistical base of the previous versions. In contrast to earlier versions of the test, the REEL-3 is based on a contemporary linguistic model for item selection and score interpretation. This model reflects research in the field of infant and toddler communication and language disorders in young children. The test has also included validity, reliability, and normative studies.

Psychometric Data

The REEL-3 was standardized on 1,112 infants and toddlers from 32 US states. The normative sample was stratified on the basis of age, gender, race, ethnic group membership, and geographic location following 2000 US census data.

The reliability of the REEL-3 was estimated using coefficient alphas (content sampling), test-retest (time sampling), and interrater reliability (interrater differences). Coefficient alphas were calculated on the REEL-3's subtests at 23 age intervals using the entire normative sample. Coefficient alphas for the language ability composite range from 0.74 to 0.98. Coefficient alphas for gender and ethnicity range from 0.95 to 0.98.

Test-retest reliability coefficients range from 0.78 to 0.80. Interrater reliability was estimated using the same test-retest sample. In this study, two raters independently scored mother's responses to the REEL-3 items. Kappa coefficients were then calculated using the Cohen (1960) method. The range of kappa coefficients for the expressive language subtest is 0.79–1.00 with a mean of 0.99.

The REEL-3 validity was estimated using content description, criterion-related, and constructed identification methods. Studies were conducted comparing the relationship between the REEL-3 and other tests of receptive and expressive

language. Correlation coefficients between the *Developmental Assessment of Young Children* (DAYC; Voress and Maddox 1998), communication subtest, the *Early Language Milestone Scale-Second Edition* ELM-2; Coplan 1993), *Cognitive Abilities Scale-Second Edition* (Bradley-Johnson and Johnson 2001), and the REEL-3 scores were calculated. The language ability composite scores on the REEL-3 yielded high correlations (0.60) with the above measures. Subtest standard scores of the REEL-3 are significantly intercorrelated (0.73).

Other validity studies, such as relationship to age, relationship to cognitive measures, and performance of various subgroups, were performed. The correlation between chronological age and the REEL-3 language ability composite was 0.90. The REEL-3 correlated moderately (0.49) with the general cognitive quotient (GCQ) of the *Cognitive Abilities Scale-Second Edition* (CAS-2; Bradley-Johnson and Johnson 2001). The performance of selected subgroups of gender, ethnic background, and speech or language impairment on the REEL-2 ranged from 90 to 110. Composite language ability scores for infants and toddlers with speech-language impairments were below average, as expected.

Clinical Uses

The REEL-3 can be used to help identify infants and toddlers who have language disorders. This is useful for identifying infants and toddlers who might benefit from early intervention by speech-language professionals. The REEL-3's norm-referenced scores can be used in reporting and determining eligibility data for special services. Scores may also assist in referral to medical, social, psychological, clinical, or disability services.

Reflective and expressive language subtests of the REEL-3 can be used to determine a significant discrepancy between comprehension process and production in early language acquisition. Significant differences can indicate a need for a specialist in early language development. The REEL-3 can also be used as a measure of a child's language development progress. As a norm-referenced,

standardized, informant based-test, the REEL-3 can be used in further research on early language acquisition.

References and Reading

- Bradley-Johnson, S., & Johnson, M. (2001). *Cognitive abilities scale* (2nd ed.). Austin: PRO-ED.
- Bzoch, K. R., League, R., & Brown, V. L. (2003). *Receptive-expressive emergent language test*. Austin: PRO-ED.
- Cohen, J. (1960). A coefficient of agreement for nominal scales. *Educational and Psychological Measurement*, 20, 37–46.
- Coplan, J. (1993). *Early language milestone scale-2*. Austin: PRO-ED.
- Lohman, D. F., & Hagen, E. P. (2001). *Cognitive abilities test* (2nd ed.). Rolling Meadows: Riverside.
- Voress, J., & Maddox, T. (1998). *Developmental assessment of young children (DAYC)*. Austin: PRO-ED.

Recessive Alleles

► Recessive Genes

Recessive Characteristics

► Recessive Genes

Recessive Genes

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Synonyms

[Recessive alleles](#); [Recessive characteristics](#);
[Recessive traits](#)

Structure

An allele is a particular form or “version” of a gene. Typically, this refers to the fact that a

variation in the genetic code is present that distinguishes one set of genetic instructions at the same locus from another. The term “recessive” describes a relationship among alleles of a gene; a trait or phenotype encoded by a recessive allele requires that two copies of that allele are present. In contrast, a trait that is coded for by only one of two alleles is called a dominant trait. An individual heterozygous (one copy each of different alleles) for the dominant and the recessive allele will appear identical to a person carrying two copies of the dominant alleles. The relationship of dominant and recessive alleles was first worked out experimentally by Gregor Mendel using traits in garden peas, such as color and surface texture; the theory of Mendelian inheritance has its origins in these experiments.

Function

One example of a Mendelian trait displaying a dominant-recessive allele relationship in humans would be the disease phenylketonuria: heterozygotes display no symptoms, one functional copy of the protein encoded by the *PKU* gene is sufficient for complete health. The same is true of the disease cystic fibrosis; one functional copy of the protein cystic fibrosis transmembrane conductance regulator, encoded by *CFTR*, is sufficient. However, two copies of the mutant allele would result in disease, making this a recessive disorder. ABO blood types, determined by identifiers on the outside of red blood cells, are another example; in this case, there are three alleles rather than two; while A and B are both dominant to O, they are codominant with each other, so that there are four blood type phenotypes: A (encoded by AA or AO), B (BB or BO), AB (encoded by AB), and O (only encoded by OO) (Griffiths et al. 2000; Miko 2008; OMIM).

Pathophysiology

Recessive alleles are of interest in autism, despite the consensus that autism is generally a

multifactorial disorder and not a single-gene Mendelian condition. Highly deleterious mutations, though not necessarily sufficient alone, may nonetheless account for a large proportion of the risk in individual cases, and thus homozygous mutations in patients with autism are of great interest. A homozygous deletion at *CNTNAP2* was implicated in several Old Order Amish families presenting with cortical dysplasia-focal epilepsy syndrome and autistic features (Strauss et al. 2006). Homozygosity mapping, a technique which specifically seeks out consanguineous families to look for regions that are identical in both copies in the offspring, due to each parent possessing at least one copy through shared ancestry, can be used to associate these homozygous regions with the phenotype of interest. A notable study applying this method to consanguineous families with children with autism demonstrated that this method can reveal rare inherited deletions that are likely to be causative, implicating several genes for which there was already evidence in autism (Morrow et al. 2008). Thus, despite autism’s status as a complex genetic disorder, methodology based on simple Mendelian principles can provide a way to gain insight into the genes involved.

References and Reading

- Cummings, M. R. (2011). *Human heredity: Principles and issues* (9th ed.). Belmont: Brooks/Cole.
- Griffiths, A. J. F., Miller, J. H., Suzuki, D. T., Lewontin, R. C., & Gelbart, W. M. (2000). *An introduction to genetic analysis* (7th ed.). New York: W. H. Freeman.
- Hartl, D. L. (2009). *Essential genetics: A genomics perspective* (5th ed.). Sudbury: Jones and Bartlett.
- Miko, I. (2008). Genetic dominance: Genotype-phenotype relationships. *Nature Education*, 1, 1.
- Morrow, E. M., Yoo, S. Y., Flavell, S. W., Kim, T. K., Lin, Y., Hill, R. S., et al. (2008). Identifying autism loci and genes by tracing recent shared ancestry. *Science*, 321(5886), 218–223.
- OMIM (Online Mendelian Inheritance in Man). <http://www.omim.org/>
- Strauss, K. A., Puffenberger, E. G., Huentelman, M. J., Gottlieb, S., Dobrin, S. E., Parod, J. M., et al. (2006). Recessive symptomatic focal epilepsy and mutant contactin-associated protein-like 2. *The New England Journal of Medicine*, 354(13), 1370–1377.

Recessive Traits

- [Recessive Genes](#)

Reciprocal Communication

- [Social Communication](#)

Reciprocal Communication/ Interaction

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Definition

Reciprocal communication/interaction is a type of communication in which children use verbal and nonverbal behaviors to engage in mutual, interactive dialogue. Characteristics include (but are not limited to) asking one's conversational partner a question, adding additional information to a statement they make, and/or guiding a conversation to elicit additional remarks from the conversant.

Impaired or absent reciprocity in communication is a diagnostic characteristic of Autism Spectrum Disorder.

See Also

- [Pragmatic Communication](#)

References and Reading

Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, R. Paul, A. Klin, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders: Diagnosis, development, neurobiology, and behavior* (Vol. I, pp. 335–364). Hoboken: Wiley.

Recognition

- [Retrieval of Information](#)

Recognition Memory

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Definition

Recognition memory involves the capacity to remember familiar stimuli when comparing a novel stimulus with one that is already stored in the memory. The seminal work on visual recognition memory conducted by Fantz (1956) and Fagan (1970) revealed that recognition memory develops in early infancy. When presented with a pair of stimuli (one novel and one familiar), infants look longer at the novel stimulus, encoding new information, and comparing this information to their stored mental representation of the familiar stimulus (Rose et al. 2004). Thus, by showing differential responses to novel and familiar stimuli, infants demonstrate their recognition of familiar stimuli. Factors that may influence one's recognition memory include the physical features of a stimulus, the number of stimuli being held in one's memory, and the amount of time that elapses between familiarization with a stimulus and recognition testing (Rose et al. 2004). Although the findings concerning recognition memory in children with autism have been somewhat mixed (e.g., Boucher 1981; Minshew and Goldstein 1993), there appears to be growing evidence of intact recognition memory among children with autism (Bennetto et al. 1996).

See Also

- [Declarative Memory](#)
- [Memory](#)
- [Memory Development](#)

References and Reading

- Bennetto, L., Pennington, B. F., & Rogers, S. J. (1996). Intact and impaired memory functions in autism. *Child Development*, 67, 1816–1835.
- Boucher, J. (1981). Memory for recent events in autistic children. *Journal of Autism and Developmental Disorders*, 11, 293–301.
- Fagan, J. F. (1970). Memory in the infant. *Journal of Experimental Child Psychology*, 9, 217–226.
- Fantz, R. L. (1956). A method for studying early visual development. *Perceptual and Motor Skills*, 6, 13–15.
- Minshew, M. J., & Goldstein, G. (1993). Is autism an amnesic disorder? Evidence from the California verbal learning test. *Neuropsychology*, 7, 209–216.
- Rose, S. A., Feldman, J. F., & Jankowski, J. J. (2004). Infant visual recognition memory. *Developmental Review*, 24, 74–100.

Recollection

- [Memory](#)

Reconcile

- [Fluoxetine](#)

Recovery in Autism

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Definition

While autism spectrum disorders (ASDs) are generally considered to be lifelong conditions, multiple studies have indicated that a small percentage of individuals who are diagnosed with an ASD in childhood experience a reduction in autism symptomatology to such a degree that they no longer meet diagnostic criteria for any ASD as they get older. The term “recovery” in this context

suggests a complete return to normal functioning; however, the limited available research examining this population suggests that some mild residual deficits are evident in some individuals in this group (Fein et al. 2013). For this and other reasons, the term “recovery” may not be the best way to describe this outcome in ASD (Helt et al. 2008; Sallows and Graupner 2005). Instead, the term “optimal outcome” has been proposed to describe individuals with a history of ASD diagnoses who no longer meet diagnostic criteria for ASD and who demonstrate average academic and adaptive functioning and receive minimal special education supports specific to autism symptoms.

Historical Background

Lovaas first formally used the term “recovery” in 1987, when reporting the results of a study that investigated an early, intensive, behavioral intervention program for ASDs. Lovaas reported that 47 % of his sample exhibited average cognitive functioning after receiving the intervention. Lovaas’ initial report of recovery has been supported by a number of more recent studies that explored the effect of early intensive behavioral treatment for ASDs. These studies reported that a small number of participants responded particularly well to treatment and performed in the average range on some outcome measures by the conclusion of the study (e.g., Cohen et al. 2007; Sallows and Graupner 2005; Zacher et al. 2007). The percentage of children who achieved this positive outcome varied considerably across studies, as did the measures used to assess outcomes.

Numerous longitudinal studies that followed children diagnosed with ASDs also reported that a small percentage of participants with ASDs within their cohort no longer met diagnostic criteria for ASD by the conclusion of the study (Sigman and Ruskin 1999; Stevens et al. 2000; Szatmari et al. 1989; Venter et al. 1992). In Szatmari and colleagues’ (1989) follow-up study of adults with ASD, 25 % of the sample no longer met diagnostic criteria for ASDs when reassessed between 11 and 27 years after initially being diagnosed with an ASD. Venter and colleagues (1992)

followed high-functioning children, adolescents, and young adults with autism over 8 years and reported that 22 % of the sample was included in regular education classrooms with limited or no supports. Sigman and Ruskin (1999) reported that 10 % of their sample lost their ASD diagnosis approximately 9 years after receiving the diagnosis. Similarly, Stevens and colleagues (2000) followed a group of preschool children with an ASD diagnosis for 8 years and found that 25 % of their sample exhibited few symptoms of autism and performed in the average range on measures of verbal and nonverbal reasoning.

Several important criticisms of the phenomenon of recovery from ASD have been voiced and have emphasized the need for a better definition of this concept. First, Piven et al. (1996) documented positive outcomes among individuals previously diagnosed with ASD and suggested that these individuals continue to present with subthreshold symptoms of ASDs. Specifically, Piven and colleagues (1996) reported that communication and socialization symptoms of ASD generally improve by adolescence and adulthood among high-functioning individuals with ASD. However, even among the 13 % of their sample who no longer met diagnostic criteria for ASD in adolescence and adulthood, all continued to present with subthreshold symptoms of ASD that caused substantial impairment in functioning. Seltzer et al. (2004) also argued that because the definition of recovery varied across the studies described above, it remained possible that these cases of recovery simply captured the expected improvement in ASD symptomatology as individuals aged. Additionally, because standardized diagnostic measures were not used regularly to ensure the accuracy of the early diagnosis in many of the studies mentioned above, it remained possible that cases of recovery were actually individuals who were inaccurately diagnosed with ASD when they were younger. Furthermore, many of the studies described above used average IQ scores and placement in regular education classrooms as indicators of recovery. However, high-functioning individuals with ASD would meet these criteria and still retain their ASD diagnosis. To address these criticisms, Helt and

colleagues (2008) reviewed previously published reports of individuals who eventually lost their ASD diagnosis and proposed a rigorous definition for this phenomenon to be used by future studies. The proposed definition described individuals who achieved “optimal outcomes (OOs)” and included having a diagnosis of ASD in early childhood made by an ASD specialist, but no longer meeting diagnostic criteria for any ASD and exhibiting average intellectual, academic, and adaptive functioning, as well as minimal special education support specific to ASD.

Current Knowledge

To understand this population further, several studies have focused on individuals who achieved OOs using a rigorous definition similar to that described by Helt et al. (2008). A study by Fein et al. (2005) described several children diagnosed with pervasive developmental disorder, not otherwise specified in early childhood, who lost their ASD diagnoses by middle childhood, instead meeting diagnostic criteria for attention deficit hyperactivity disorder (ADHD) at reevaluation. While these children no longer met diagnostic criteria for ASD, some of them continued to exhibit perseverative interests and occasional repetitive motor movements, as well as mild social difficulties. The authors suggested that attention difficulties may be a core feature of ASD that is more difficult to address with early behavioral intervention.

In a series of studies, Kelley et al. (2006) and Kelley et al. (2010) sought to uncover the presence of residual deficits among a group of children who were diagnosed with ASDs as toddlers, and at the time of the study exhibited average IQ scores, were mainstreamed into age-appropriate classrooms, functioned at age-appropriate levels according to the report of the children’s parents and teachers, and no longer met diagnostic criteria for any ASD. The first study evaluated the language abilities of 14 such children between the ages of 5 and 9 years and found residual difficulties on measures of pragmatic and semantic language, particularly in understanding the second-

order theory of mind, using mental state verbs, producing a narrative, and using inductive reasoning about animate objects (Kelley et al. 2006). In the second study, Kelley and colleagues (2010) described the functioning of an older OO group that ranged in age from 8 to 13 years. The OO group consisted of 11 children who were included in the first study along with two additional participants. The study employed two comparison groups: typically developing peers and high-functioning peers with a current ASD diagnosis. The three groups were compared on measures of cognitive, adaptive, social, and communicative functioning, ASD symptom severity, and expressive and receptive language. The results showed that the OO group exhibited more difficulties with attention than typically developing peers, but otherwise did not differ on any other measure included in the study, including pragmatic language ability.

Recently, a large retrospective study was conducted at the University of Connecticut that explored the functioning and residual weaknesses of a group of individuals who achieved OOs and documented the range of treatments they received (Fein et al. 2013). The study included 34 individuals who were diagnosed with ASD by a specialist in ASD before the age of 5 years. At the time of the study, participant in the OO group did not meet diagnostic criteria for any ASD by clinical judgment (based on DSM-IV criteria and ADOS), presented with average intellectual and adaptive functioning, and were included in a regular education classroom without one-on-one assistance and no special education services to address ASD-related deficits. Performance across measures was compared to 44 high-functioning individuals with a current ASD diagnosis (HFA group) and 34 typically developing peers (TD group). Groups were matched on age, gender, and nonverbal IQ, but differed significantly on verbal IQ, with the OO and TD groups scoring seven points higher than the HFA group. Parent reports of early development suggested the OO group exhibited milder symptoms of ASD than the HFA group within the social domain, but displayed equally severe difficulties in the communication domain (Fein et al. 2013). At the time

of the study, individuals in the OO group were functioning in the normal range on all measures of communication and social functioning, and, as expected, scored better than individuals in the HFA group in both domains. A comparison of specific behaviors within the social and communication domains across the OO and TD groups revealed that participants in the OO group were significantly more likely to ask for information and showed a trend to offer more information. A proportion of the OO group also displayed a subtle residual weakness in the level of insight into social relationships, and a very small proportion of OO participants still showed suboptimal eye contact (Orinstein et al. 2015a).

With respect to repetitive behaviors, parents of individuals in the HFA and OO groups reported similar levels of restricted and repetitive behaviors early in development, with the exception of milder levels of oversensitivity to noise and insistence on sameness in the OO group. While these behaviors were present in childhood, the OO group's restricted and repetitive behaviors appeared to resolve along with social and communication deficits and had disappeared almost entirely by the time of participation in this study. At the time of the study, restricted and repetitive behaviors were still present in the HFA group (Troyb et al. 2014b).

The cognitive functioning of the OO group was explored across a number of domains. Within the language domain, the OO group scored in the average range or above on all measures of expressive and receptive language and showed few differences from the TD group. The HFA group also performed within the average range, but scored significantly lower than the other groups on formulating sentences and a measure of overall language skills, even when verbal IQ was controlled for (Tyson et al. 2014). Narratives of a subsample of each group were also evaluated (Suh et al. 2014). Participants in the OO group displayed a subtle use of idiosyncratic language and speech disfluency that was not observed in the TD group. Participants in HFA group produced narratives with fewer central story elements and more ambiguous pronominal referents, idiosyncratic language, and

speech disfluency as compared to the other two groups (Suh et al. 2014). On a measure of auditory perceptual skills, the HFA group showed heightened pitch discrimination than both the OO and TD groups; the abilities of the OO did not differ from the TD group (Eigsti and Fein 2013). The ability to organize semantic categories was compared across the groups and revealed that both the OO and HFA groups were less consistent in acquiring and using category structures than the TD group, which may be related to with an underlying difficulty with generalization observed among individuals with ASD (Naigles et al. 2013). Scores on a verbal memory measure were similar across the OO and TD groups. The HFA group's overall score on the verbal memory measure was solidly average; however, more participants in this group received below average scores than in the other two groups (Tyson et al. 2014).

The academic functioning of the OO group fell in the average range, and the TD and OO group scored similarly across all measures of reading, mathematics, and writing. The HFA group also scored in the average range across academic domains, but scored significantly lower on subtests of reading comprehension and mathematical problem solving than the OO group. These findings indicated that the academic abilities of individuals who achieved OOs are similar to those of their typically developing peers, even in areas where individuals who have retained their ASD diagnoses exhibit some ongoing difficulty (Troyb et al. 2014a).

When directly assessed, executive functioning fell in the average range across the three groups, but the OO and HFA groups exhibited more impulsivity and less efficient planning and problem solving than the TD group. Parent report of everyday use of executive functioning revealed average abilities in both the OO and TD groups; however, the OO group exhibited more difficulty than the TD group on set shifting and working memory. Participants in the HFA group demonstrated more difficulty on all parent-reported executive functioning domains, with a clinically significant weakness in attention shifting (Troyb et al. 2014c).

An exploration of psychiatric functioning revealed higher rates of ADHD and specific phobias in the HFA and OO groups, as compared to the TD group. The OO and HFA groups were also more likely to meet diagnostic criteria for tic disorders. However, tics appeared to abate over time more frequently in the OO group as compared to the HFA group, and by the time of the study, tic disorders were no more prevalent in the OO group than in the TD group. The HFA group had the highest level of comorbid psychiatric symptoms both at the time of testing and earlier in their development (Orinstein et al. under 2015b).

Parent report of intervention histories of the OO and HFA groups was collected and examined. Results indicated that parents of participants in the OO group had earlier concerns about their child's development and received earlier referrals to specialists than did parents of participants in the HFA group. On average, the OO group received earlier and more intensive intervention than the HFA group, with more OO participants receiving applied behavior analysis. Children in the HFA group were more likely to have received medication, especially antipsychotics and antidepressants (Orinstein et al. 2014).

In 2014, Anderson et al. published a prospective study examining the range of outcomes among young adults with ASD, and their sample included eight participants who achieved a "very positive outcome." These eight individuals were diagnosed with ASD at age 2, were reevaluated at 3 years of age, and no longer met diagnostic criteria for ASD when they were seen for the third evaluation when they were 19 years old. At the age of 2 years, participants who achieved very positive outcomes showed similar degrees of repetitive behaviors, social delays, and adaptive functioning delays as did high-functioning participants who retained their ASD diagnoses. Participants who achieved a very positive outcome were also more likely to participate in individual treatment by age 3 and displayed a greater reduction in repetitive behaviors between the ages of 2 and 3 years than did other high-functioning participants in the sample who retained their ASD diagnoses. At the age of

19, participants in the very positive outcome group exhibited fewer symptoms of ASD in the socialization, communication, and repetitive behavior domains and had average verbal reasoning and academic abilities, as compared to high-functioning participants who retained their ASD diagnosis. In addition, participants in the very positive outcome group were also less likely to present with symptoms of irritability, hyperactivity, or depression. At the age of 19, of the eight participants, five were living away from home, seven were enrolled in college, five were employed, and none were taking psychotropic medication.

Future Directions

Questions that remain about optimal outcomes in ASD include how many children with ASD have the capacity to achieve these outcomes and what early factors predict these outcomes. A key question is what genetic or other biological factors mark the children who have the potential to achieve optimal outcome, even if this outcome is dependent of intense early intervention. Future prospective intervention studies should explore types of intervention to determine which is most likely to result in OOs following an ASD diagnosis. These studies would benefit from gathering data about fidelity with which intervention is delivered. In addition, future prospective studies should include frequent assessment intervals in order to explore the nature and timing of symptom reduction.

Another important question that remains unanswered is to what extent neurological characteristics have normalized in children who achieved OOs. Studies using structural and functional neuroimaging could help determine to what extent the neuroanatomical structures, functioning, and pathways have normalized in this population. Future research should also examine social functioning in more detail among individuals who achieved OO and specifically investigate peer interactions and the quality of friendships in order to determine whether subtle residual social deficits exist in this group.

See Also

- [Optimal Outcome](#)
- [Outcome](#)
- [Residual Autism](#)

References and Reading

- Anderson, D. K., Liang, J. W., & Lord, C. (2014). Predicting young adult outcome among more and less cognitively able individuals with autism spectrum disorders. *The Journal of Child and Psychology and Psychiatry*, 55, 485–494. <https://doi.org/10.1111/jcpp.12178>.
- Cohen, H., Amerine-Dickens, M., & Smith, T. (2007). Early intensive behavioral treatment: Replication of the UCLA model in a community setting. *Developmental and Behavioral Pediatrics*, 27, S145–S155. <https://doi.org/0196-206X/06/2702-0145>.
- Eigsti, I. M., & Fein, D. A. (2013). More is less: Pitch discrimination and language delays in children with optimal outcomes from autism. *Autism Research*, 6, 605–613. <https://doi.org/10.1002/aur.1324>.
- Fein, D., Dixon, P., Paul, J., & Levin, H. (2005). Brief report: Pervasive developmental disorder can evolve into ADHD: Case illustrations. *Journal of Autism and Developmental Disorders*, 35, 525–534. <https://doi.org/10.1007/s10803-005-5066-3>.
- Fein, D. A., Barton, M. L., Eigsti, I. M., Kelley, E. A., Naigles, L., Schultz, R. T., . . . Tyson, K. E. (2013). Optimal outcome in individuals with a history of autism. *The Journal of Child Psychology and Psychiatry*, 54, 195–205. <https://doi.org/10.1111/jcpp.12037>
- Helt, M., Kelley, E., Kinsbourne, M., Pandey, J., Boorstein, H., Herbert, M., . . . Fein, D. (2008). Can children with autism recover? If so, how? *Neuropsychology Review*, 18, 339–366. <https://doi.org/10.1007/s11065-008-9075-9>
- Kelley, E., Paul, J. J., Fein, D., & Naigles, L. R. (2006). Residual language deficits in OO children with a history of autism. *Journal of Autism and Developmental Disorders*, 36, 807–828. <https://doi.org/10.1007/s10803-006-0111-4>.
- Kelley, E., Naigles, L. R., & Fein, D. (2010). An in-depth examination of optimal outcome children with a history of autism spectrum disorders. *Research in Autism Spectrum Disorders*, 4, 526–538. <https://doi.org/10.1016/j.rasd.2009.12.001>.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9. <https://doi.org/10.1037/0022-006X.55.1.3>.
- Naigles, L. R., Kelley, E., Troyb, E., & Fein, D. (2013). Residual difficulties with categorical induction in children with a history of autism. *Journal of Autism and*

- Developmental Disorders*, 43(9), 2048–2061. <https://doi.org/10.1007/s10803-012-1754-y>.
- Orinstein, A., Helt, M., Troyb, E., Tyson, K. E., Barton, M. L., Eigsti, I. M., ... Fein, D. A. (2014). Intervention history of children and adolescents with high-functioning autism and optimal outcomes. *Journal of Developmental and Behavioral Pediatrics*, 35(4), 247–256. <https://doi.org/10.1097/DBP.0000000000000037>
- Orinstein, A., Suh, J., Porter, K., DeYoe, K. A., Tyson, K. E., Troyb, E., ... and Fein, D. A. (2015a). Social function and communication in optimal outcome children and adolescents with an autism history on structured test measures. *Journal of Autism and Developmental Disorders*, 45(8), 2443–2463. <https://doi.org/10.1007/s10803-015-2409-6>.
- Orinstein, A., Tyson, K. E., Suh, J., Troyb, E., Helt, M., Rosenthal, M., ... and Fein, D. A. (2015b). Psychiatric symptoms in youth with a history of autism and optimal outcome. *Journal of Autism and Developmental Disorders*, 45(11), 3703–3714. <https://doi.org/10.1007/s10803-015-2520-8>.
- Piven, J., Harper, J., Palmer, P., & Arndt, S. (1996). Course of behavioral change in autism: A retrospective study of high-IQ adolescents and adults. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35, 523–529. <https://doi.org/10.1097/00004583-199604000-00019>.
- Sallows, G. O., & Graupner, T. D. (2005). Intensive behavioral treatment for children with autism: Four-year outcome and predictors. *American Journal of Mental Retardation*, 110, 417–438. [https://doi.org/10.1352/0895-8017\(2005\)110\[417:IBTCFW\]2.0.CO;2](https://doi.org/10.1352/0895-8017(2005)110[417:IBTCFW]2.0.CO;2).
- Seltzer, M. M., Shattuck, P., Abbeduto, L., & Greenberg, J. S. (2004). Trajectory of development in adolescents and adults with autism. *Mental Retardation and Developmental Disabilities Research Reviews*, 10, 234–247. <https://doi.org/10.1002/mrdd.20038>.
- Sigman, M., & Ruskin, E. (1999). Continuity and change in the social competence of children with autism, down syndrome, and developmental delays. In *Monographs of the society for research in child development* (Serial No. 256) (Vol. 64). Chicago: University of Chicago Press.
- Stevens, M. C., Fein, D. A., Dunn, M., Allen, D., Waterhouse, L. H., Feinstein, C., ... Rapin, I. (2000). Subgroups of children with autism by cluster analysis: A longitudinal examination. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 346–352. <https://doi.org/10.1097/00004583-200003000-00017>
- Suh, J., Eigsti, I. M., Naigles, L., Barton, M., Kelley, E., & Fein, D. (2014). Narrative performance of optimal outcome children and adolescents with a history of an autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44, 1681–1694. <https://doi.org/10.1007/s10803-014-2042-9>.
- Szatmari, P., Bartolucci, G., Bremner, R., Bond, S., & Rich, S. (1989). A follow-up study of high-functioning autistic children. *Journal of Autism and Developmental Disorders*, 19, 213–225. <https://doi.org/10.1007/BF02211842>.
- Troyb, E., Orinstein, A., Tyson, K., Helt, M., Eigsti, I. M., Stevens, M. C., & Fein, D. A. (2014a). Academic functioning in children and adolescents with a history of autism spectrum disorders who have achieved an optimal outcome. *Autism*, 18, 233–243. <https://doi.org/10.1177/1362361312473519>.
- Troyb, E., Orinstein, A., Tyson, K. E., Eigsti, I. M., Naigles, L., & Fein, D. A. (2014b). Restricted and repetitive behaviors in individuals with a history of ASDs who have achieved optimal outcomes. *Journal of Autism and Developmental Disorders*, 44(12), 3168–3184. <https://doi.org/10.1007/s10803-014-2182-y>.
- Troyb, E., Rosenthal, M., Eigsti, I. M., Kelley, E., Tyson, K., Orinstein, ... Fein, D. A. (2014c). Executive functioning in children with ASDs who have achieved optimal outcomes. *Child Neuropsychology*, 20(4), 378–397. <https://doi.org/10.1080/09297049.2013.799644>
- Tyson, K. E., Kelley, E., Fein, D., Orinstein, A., Troyb, E., Barton, M. L., ... Rosenthal, M. (2014). Language and verbal memory in individuals with a history of autism spectrum disorders who have achieved optimal outcomes. *Journal of Autism and Developmental Disorders*, 44(3), 648–663. <https://doi.org/10.1007/s10803-013-1921-9>
- Venter, A., Lord, C., & Schopler, E. (1992). A follow-up study of high-functioning autistic children. *Journal of Child Psychology and Psychiatry*, 33, 489–597. <https://doi.org/10.1111/j.1469-7610.1992.tb00887.x>.
- Zachor, D. A., Ben-Itzhak, E., Rabinovich, A. L., & Lahat, E. (2007). Change in autism core symptoms with intervention. *Research in Autism Spectrum Disorders*, 1, 304–317. <https://doi.org/10.1016/j.rasd.2006.12.001>.

Redirection

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Definition

Redirection is the process of shifting one's focus or actions from one activity to another. As a guidance technique most often used to interrupt behavior, it orients an individual from one behavior or stimulus to another and acts to elicit the desired behavior or outcome. Redirections can occur in

subtle ways such as a change in topic within a conversation or the presentation of an instructional material or in a more intrusive manner such as physically positioning oneself between the individual and a certain item or location or by physically interrupting stereotypy and reengaging in the task at hand.

References and Reading

- Ahrens, E. A., Lerman, D. C., Kodak, T., Worsdell, A. S., & Keegan, C. (2011). Further evaluation of response interruption and redirection as treatment for stereotypy. *Journal of Applied Behavior Analysis, 44*, 95–108.
- Cassella, M. D., Sidener, T. D., Sidener, D. T., & Progar, P. R. (2011). Response interruption and redirection for vocal stereotypy in children with autism: A systematic replication. *Journal of Applied Behavior Analysis, 44*, 169–173.
- Cooper, J., Heron, T., & Heward, W. (2007). *Applied behavior analysis* (2nd ed.). Hoboken: Pearson Education.
- Malott, R., Malott, M., & Trojan, E. (2000). *Elementary principles of behavior* (4th ed.). Upper Saddle River: Prentice-Hall.

Reduced Prioritization of Facial Threat in ASD

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Synonyms

Threat detection; Threat perception; Threat superiority

Definition

Threat perception involves the rapid detection of potentially harmful stimuli within the environment. Typically developing (TD) individuals demonstrate a robust “threat superiority effect,” in which they detect threatening stimuli amongst nonthreatening stimuli faster and with greater

accuracy than vice versa (for a review, see LoBue and Rakison 2013). The prioritization of threatening stimuli is adaptive, facilitates biological readiness and self-protection, and occurs for dangers that are both ancient (e.g., snakes) and more modern (e.g., guns). People, of course, can also be a source of threat and not surprisingly threatening emotional expressions such as anger, and even threatening faces lacking emotion like those that are hypermasculine and perceived as aggressive (Shasteen et al. 2015) are detected more quickly and with more accuracy by TD individuals than are nonthreatening faces (Pinkham et al. 2010).

Autism spectrum disorder (ASD) is characterized by difficulties in social understanding, with many but not all on the spectrum exhibiting reduced abilities in face processing, emotion recognition, and mentalizing skill. A recent study examined whether these difficulties extend into the domain of social threat detection (Sasson et al. 2016). A failure by autistic adults to demonstrate a classic threat superiority effect for angry emotional expressions could indicate a reduced sensitivity to facial threat signals that could contribute to social misunderstanding, miscommunication, and under certain circumstances, increased susceptibility for victimization.

Sasson et al. (2016) compared performance between 21 autistic adults without intellectual disability and 28 TD controls on a “face in the crowd” task in which participants determine whether all faces within a 3×3 matrix are displaying the same emotional expression or whether one of the faces in the crowd is displaying a discrepant emotion. Unlike previous tests of the threat superiority effect in ASD, the task used here included veridical facial photographs and was constructed to mimic a true “crowd effect” by ensuring that no individual was presented twice within the same matrix. Results for TD participants replicated previous findings: they detected angry faces more rapidly than happy faces, both within crowds of happy distractor faces and neutral distractor faces. In contrast, autistic adults did not share this pattern, showing no response time advantage for detecting angry face targets relative to happy and neutral face targets. Furthermore,

whereas TD participants took longer to respond on “target absent” trials in which all faces in the crowd were angry compared to all happy or all neutral, autistic participants did not. This suggests that angry faces disproportionately captured attention for TD participants, but did not for autistic participants.

However, like TD participants, autistic adults were more accurate at detecting angry relative to happy faces. This suggests that autistic adults do assign some level of increased salience to signals of facial threat, but the lack of a concurrent response time advantage for detecting angry faces within a crowd – the finding that typically defines the presence of a threat superiority effect – suggests that autistic adults are characterized by a reduction in the adaptive prioritization of social threatening information. As such, this finding may reflect an additional domain in which social cognitive differences in autism relate to broader aspects of their social disability. Autistic adults are often more trusting, can be more vulnerable to manipulation, and experience high rates of victimization (Weiss and Fardella 2018). Whether the reduced prioritization of facial threat found in this study might relate to increased vulnerability for social harm is currently unclear. It is also unclear whether autistic adults would demonstrate a similar reduction in the “threat superiority effect” for sources of nonsocial threat (e.g., guns). If so, the findings here might reflect more general perceptual differences rather than a specific difficulty assessing aspects of social threat. If, however, autistic adults demonstrate an intact “threat superiority effect” for nonsocial stimuli, the discrepancy in threat assessment between social and nonsocial threat would parallel other aspects of the condition in which social cognitive difficulties are often dissociable from general cognitive ability.

References and Reading

- LoBue, V., & Rakison, D. H. (2013). What we fear most: A developmental advantage for threat-relevant stimuli. *Developmental Review*, 33(4), 285–303.
- Pinkham, A. E., Griffin, M., Baron, R., Sasson, N. J., & Gur, R. C. (2010). The face in the crowd effect: Anger

superiority when using real faces and multiple identities. *Emotion*, 10(1), 141.

- Sasson, N. J., Shasteen, J. R., & Pinkham, A. E. (2016). Brief report: Reduced prioritization of facial threat in adults with autism. *Journal of Autism and Developmental Disorders*, 46(4), 1471–1476.
- Shasteen, J. R., Sasson, N. J., & Pinkham, A. E. (2015). A detection advantage for facial threat in the absence of anger. *Emotion*, 15(6), 837.
- Weiss, J. A., & Fardella, M. A. (2018). Victimization and perpetration experiences of adults with autism. *Frontiers in Psychiatry*, 9, 203.

REEL-3

- [Receptive-Expressive Emergent Language Test \(REEL-3\)](#)

Reevaluation

- [Triennial Evaluation and Triennial Review](#)

Referent

- [Tact\(s\)](#)

Reflexive Behavior

- [Respondent Behavior](#)

Refrigerator Mother

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Definition

The term “refrigerator mother,” although commonly attributed to Bruno Bettelheim, appears to

derive instead from the following section of a 1949 paper by Leo Kanner: “Most of the patients were exposed from the beginning to parental coldness, obsessiveness, and a mechanical type of attention to material needs only. They were the objects of observation and experiment conducted with an eye on fractional performance rather than with genuine warmth and enjoyment. They were kept neatly *in refrigerators* [emphasis added] which did not defrost.”

See Also

- ▶ [Bettelheim, Bruno](#)
- ▶ [Kanner, Leo](#)

References and Reading

Kanner, L. (1973). *Childhood psychosis: New studies and new insights*. (p. 61). Washington, DC: V.H. Winston & Sons (Reprinted from Problems of nosology and psychodynamics in early infantile autism. *American Journal of Orthopsychiatry* [1949] 19, 416–426).

Regional Centers

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Definition

Centers that develop or provide service or training related to Autism Spectrum Disorder or ASD to a county or multicounty region. We distinguish regional centers from centers serving an area smaller than a county (e.g., a city or school district), and from other centers serving a larger area (e.g., a statewide programs).

Most regional centers are, at least partially, publicly funded. They are often formally mandated to serve a geographically defined area (e.g., a county or multicounty region), and a

subset of the overall population, as defined by age (e.g., between 3 and 21 years of age), or other characteristics of the person (e.g., those with ASD and significant intellectual disability or ID), or defined by a specific range of services provided (e.g., early intervention, behavioral health, vocational training, etc.). This mandate is usually dictated by the funding source. For example, many states allocate public funding for early intervention (Birth to 3) and behavioral health services by county, and may administer these services directly via a county-level program or contract out these services privately. Some of these centers were originally established when the institutionalization of persons with significant intellectual disabilities or psychiatric disorders was the standard of practice. As is the case of statewide service programs, regional centers providing direct service have been on the decline since the mid-1970s, with the increased emphasis on community-based educational and psychiatric services, and increased number of persons identified with ASD.

Some centers emerged because the designated population or need was too small to offer services locally. For example, individual school districts may not have enough students with ASD to create an ASD-specific program, but districts across a county may together designate a specific school district or another entity to operate a specialized program.

Other programs serve some of the functions of regional centers: University-based centers of excellence on developmental disabilities may assume regional leadership for training, specialized children’s hospital may develop satellite programs to extend services throughout a broader area, or private ASD centers serve as a hub for a range of services across a region (Handelman and Harris 2001, 2006). Such centers often have greater flexibility to develop new programs to fill critical gaps, and are not as vulnerable to the year-to-year fluctuations in levels of state and federal funding. Such centers are not held legally accountable, however, if they are not accessible to the entire population or fail to deliver critical services in a timely manner.

See Also

- [Douglass Development Disabilities Center](#)
- [Statewide Service Programs](#)

References and Reading

- Handelman, J., & Harris, S. L. (2001). *Preschool education programs for children with autism* (2nd ed.). Austin: Pro-Ed.
- Handelman, J., & Harris, S. L. (2006). *School-age education programs for children with autism*. Austin: Pro-Ed.

Register Variation

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Synonyms

[Inflection](#); [Language style](#); [Language variation](#)

Definition

Register variation includes the ability to vary one's language style based on the social context or communication partner. For example, adult speakers talk differently to babies than they do to other adults. Individuals with ASD often have difficulty with register shifts, using the same style of speech or vocabulary across communication partners regardless of age (e.g., same-aged peers vs. adults), status (peer vs. teacher), or context (home vs. work). Individuals with ASD also experience difficulty using register shifts flexibly to make requests in different contexts (e.g., favor vs. a right) or to persuade.

See Also

- [Pragmatic Language](#)

References and Reading

- Paul, R. (2005). Assessing communications in autism spectrum disorders. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. II, pp. 799–830). Hoboken: Wiley.
- Volden, J., Magill-Evens, J., Goulden, K., & Clarken, M. (2007). Varying language register according to listener needs in speakers with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37, 1139–1154.

Registered Behavior Technician (Definition)

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In the last few decades, there has been a significant increase in the prevalence of autism spectrum disorder (ASD). Today, 1 in 68 children is diagnosed with ASD (Centers for Disease Control and Prevention 2016) resulting in an increase in the demand of interventions that are evidence-based and shown to be effective in treating the symptoms of the disorder, therefore, leading to an increase in the demand of services based on the principles of applied behavior analysis (ABA). Behavioral interventions derived from these principles have been identified to be effective and efficient in improving the symptoms of ASD as well as teaching critical and socially significant skills to the consumers of this treatment (Myers and Johnson 2007; Howard et al. 2005; Eikeseth et al. 2007; Lovaas 1987). The increase in the demand of ABA services has also resulted an increase in the need for individuals who are adequately trained and qualified to implement the ABA interventions to the vulnerable population of individuals they serve (Hughes and Shook 2007).

In order to meet the need for qualified implementers of ABA, the Behavior Analysis

Certification Board (BACB[®]) responded by establishing the Registered Behavior Technician (RBT[®]) credential in 2014 for non-certified individuals (e.g., paraprofessionals and direct-care staff) that requires these individuals to complete specific training in the implementation of behavioral technology and meet other requirements established to obtain the credential. The training is to be delivered under the supervision of a Board Certified Behavior Analyst (BCBA[®]) and must adhere to the requirements established by the BACB. The RBT requirements established by the BACB include the following:

- (a) Be at least 18 years of age
- (b) Complete and pass a criminal background check
- (c) Have obtained a minimum of a high school diploma
- (d) Complete a 40-h, combined didactic and experiential training program that is aligned with the RBT task list
- (e) Pass a competency checklist (demonstrate the ability to implement behavioral interventions and tasks in a role-play or in vivo format) as evaluated by the trainer
- (f) Pass the RBT examination
- (g) Pay all of the fees associated with the RBT credentialing process (Forte et al. 2018)

The 40-h combined didactic and experiential training includes a review and demonstration of behavioral interventions commonly implemented by behavior technicians such as reinforcement, prompting, discrete trial teaching, task analysis, shaping, and data collection, to name a few. The BCBA delivering the training is responsible for developing the didactic training content (e.g., PowerPoint slides, worksheets, visuals) and experiential activities (e.g., role-play or in vivo training opportunities). Again, all training targets must be aligned with the 20-item RBT task list. Once the trainees have completed the 40-h training program, they must also complete a competency assessment that evaluates their performance when demonstrating behavioral interventions based on the RBT task list. The task list includes common tasks completed by RBTs and falls under

the categories of “Measurement, Assessment, Skill Acquisition, Behavior Reduction, Documenting and Reporting, and Professional Conduct and Scope of Practice” (BACB 2014, p. 1). The trainee is responsible for submitting verification indicating that they have completed all the necessary requirements in order to receive approval for taking the examination administered through the BACB.

To maintain the RBT credential, the individual must be supervised for a minimum of 5% of the hours they have implemented ABA interventions per month. For example, an RBT must receive at least 1.5 h of supervision for every 30 h they have engaged in the implementation of ABA interventions. The BACB has set up these requirements in order to ensure that the delivery of high-quality ABA services is implemented with integrity and it will have a direct impact on the consumers of ABA. At this time, the requirements do not require continuing education to maintain the credential. However, the BACB[®] has established a group of subject matter experts (SMEs) whose task is to evaluate the requirements of the RBT overtime (approximately every 5 years) and make revisions to the requirements based on research evaluating the efficacy of the training requirements. Further, the RBT credential is a relatively new credential, and the SMEs are continuing to work on *operationalizing* the task list items so that they can be appropriately measured by supervisors conducting the competency assessments (Carr et al. 2017).

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References and Reading

- Behavior Analyst Certification Board. (2014). *Behavior Analyst Certification Board RBT requirements*. Retrieved from <https://www.bacb.com/rbt/rbt-requirements/>
- Carr, J. E., Nosik, M. R., & DeLeon, I. G. (2017). The Registered Behavior Technician™ credential: A response to Leaf et al. (2017). *Behavior Analysis in Practice*. <https://doi.org/10.1007/s40617-017-0172-1>.
- Centers for Disease Control and Prevention. (2016). Prevalence and characteristics of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United

- States, 2012. Retrieved from <https://www.cdc.gov/mmwr/volumes/65/ss/ss6503a1.htm>
- Eikeseth, S., Smith, T., Jahr, E., & Eldevik, S. (2007). Outcome for children with autism who began intensive behavioral treatment between ages 4 and 7: A comparison controlled study. *Behavior Modification*, 31, 264–278.
- Forte, S., Dorsey, M. F., Weiss, M. J., Palmieri, M. J., & Powers, M. D. (2018). Exploring issues of generalization and maintenance in training instructional aides in a public school setting. *Journal of Behavioral Education*, 27(4), 435–460. <https://doi.org/10.1007/s10864-018-9304-0>.
- Howard, J. S., Sparkman, C. R., Cohen, H. G., Green, G., & Stanislaw, H. (2005). A comparison of intensive behavior analytic and eclectic treatments for young children with autism. *Research in Developmental Disabilities*, 26, 359–383.
- Hughes, J.C. & Shook, G.L. (2007). Training and certificate of behavior analyst in Europe: Past, present, and future challenges. *European Journal of Behavior Analysis* 8(2), 239–249.
- Lovaas, I. O. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- Myers, S. M., & Johnson, C. P. (2007). Management of children with autism spectrum disorders. *Pediatrics*, 120, 1162–1182.
- acquired and subsequently lost for at least a period of 3 months. Regression in ASD has been relatively poorly defined and has mainly been studied using retrospective parental reports after the individual has received an autism spectrum disorder (ASD) diagnosis. In her review, Rogers defined autistic regression as “loss of previously acquired language and social relatedness skills during the first 3 years of life” (Rogers 2004). Parr et al. (2011) recently used the term “early developmental regression” in the study of regression before age 3 years. In ASD, the most commonly reported regression is the loss of use of words or phrases, or developing language. Non-language regression (e.g., loss of sociability, loss of interest in play) is less clearly defined and may be difficult to distinguish from the recognition of onset of symptoms of ASD or variation in behavior and skills in ASD. Other regression (e.g., loss of motor skills) requires consideration of other diagnoses (see ► [“Rett Syndrome”](#) and ► [Childhood Disintegrative Disorder](#)).

Regression

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Definition

Regression is the loss of previously acquired skills. In a developmental context, a child is considered to have regressed when there is evidence that he/she has lost a definitely acquired skill that was previously used regularly for some time. The Autism Diagnostic Interview-Revised (ADI-R) requires skills previously

Historical Background

Early research about regression focused on word loss, timing, and the potential association with seizures and epilepsy (Kurita 1985; Tuchman et al. 1991). The subsequent link between the MMR vaccination, ASD, and regression led to considerable interest from researchers, both in terms of understanding regression better and investigating the possible link with MMR and other environmental factors (e.g., thimerosal). Investigating regression through retrospective reports has long been a methodological weakness in research methods. To date, most published research findings have used the ADI-R to characterize parental accounts of regression. Home videos have confirmed the reality of developmental regression in young children with ASD (Werner and Dawson 2005). However, the longitudinal prospective study of infants at increased risk (such as the younger siblings of children with ASD) is likely to shed most light on the process of

regression, both from a behavioral and neurobiological perspective (Landa et al. 2007; Werner et al. 2005).

Current Knowledge

Regression occurs in approximately 20–30% of children with ASD and is seen at equal rates in males and females with ASD. Early developmental regression is defined as onset before age 3 years, and onset is most often reported between 18 and 24 months of age. Some children over age 3 years have been reported to lose language, and nonlanguage skills (Parr et al. 2011). Regression occurs as frequently in families with one child with ASD (singleton) as families with two or more affected children (multiplex) (Parr et al. 2011), suggesting no genetic or familial basis to regression (Parr et al. 2008, 2011).

Developmentally, and in the general pediatric population, regression of skills is uncommon and suggests that some form of severe process is occurring in the brain. For example, children with early-onset epilepsy lose acquired skills and usually enter a period of developmental stasis, in which new skill acquisition stalls. One such condition is Landau-Kleffner syndrome – following typical development, children develop seizures, lose expressive language, and seem to have difficulties with understanding; other behavioral difficulties and cognitive impairments co-occur. An electroencephalogram (EEG) is abnormal.

Regression in ASD should be differentiated from Rett syndrome (a pervasive developmental disorder but not an ASD). Rett syndrome is seen in girls and very occasionally in boys. Following a period of normal development, loss of skills occurs, followed by microcephaly and the onset of seizures and other features (classically hand wringing, a sighing respiratory pattern, and scoliosis). The Rett phenotype is differentiated from regression in ASD through the clinical history and examination and, when necessary, genetic testing (for the MECP2 or CDLK5 mutations). Very rarely, a catastrophic regression of all skills is seen in children who have developed typically for at least the first 2 years. This presentation

may be a manifestation of childhood disintegrative disorder (CDD, formerly known as Heller syndrome) – children require investigation, but in CDD, no abnormality is found on laboratory testing or through EEG or cranial imaging.

Clinically, parents' descriptions of regression should always be taken seriously, and a search for a cause of regression made through history taking, examination, and investigations if necessary. While regression may be a presenting feature of ASD, often parents have already been concerned about their child's early social communication development and, in particular, the relatively slow acquisition of speech. In the clinical history, regression should be differentiated from an account of developmental skills gained and then not used for some period. This is particularly the case with word loss – children with typical development may use a word and then not use that word again for days or weeks. However, during this time, the child's language usually continues to develop – other words come and many become established (the so-called word explosion). Forward developmental progress is maintained at all times. This contrasts with the account of early regression in ASD, where parents report that their child loses the use of words and even phrases sometimes rapidly (some parents report “overnight”), or progressively over several weeks, without new language skills being gained. For all parents, any loss of their child's skills is extremely distressing. Recent data show that the loss of previously acquired speech in the early years is most frequently seen in association with ASD (Pickles et al. 2009); clinicians should specifically ask about features of ASD when regression is reported.

A regression history has usually been taken after the event. As increased knowledge is gained about the early behavioral manifestations of ASD in the first few years of life, and with new guidelines regarding ASD surveillance (Johnson et al. 2007), more contemporaneous data will become available. When seeing the parents of a child with a history of possible loss of skills, a thorough developmental history, together with a detailed account of the regression episode, including any coexisting illnesses or occurrences is required. In

particular, the presence or absence of a pre-regression ASD history should be sought. A detailed family history including relatives with ASD is required. The typical history is one of loss of skills, most commonly speech. Parents may report loss of sounds used communicatively or loss of words and phrases used with meaning. Parents may relate regression to an illness or other event such as vaccination. However, epidemiological and other studies have not identified a link between vaccinations (particularly the measles, mumps, and rubella (MMR) vaccination) (Fombonne and Chakrabarti 2001; Richler et al. 2006). Parents may also report loss of other skills, including social abilities, motor skills, and occasionally toileting skills. These problems have been much more difficult to quantify; however, any account of loss of skills in these latter areas should always lead clinicians to consider other diagnoses and possible causes. A general and neurological examination should always be undertaken. Some parents may bring video recordings of their child pre-, during, and postregression – mobile telephone videos have increased the frequency of this. This video evidence is always useful, and clinicians should invest time in watching them. Observational accounts from other settings (e.g., nursery staff) are also valuable. Based on the clinical presentation including the results of the history and physical examination, the clinician will make a judgment about whether to undertake further investigations. In the presence of neurodevelopmental history consistent with ASD, and a regression history without events of particular note, and in the absence of any noteworthy neurological findings, most clinicians do not investigate children with regression but either make an ASD diagnosis or assign the regression as “related to ASD.” However, if the regression is accompanied by a “non-ASD type” developmental history, symptoms or signs not usually seen in ASD, seizures, or abnormal findings on detailed neurological examination, investigations are required; when clinicians are not experts in ASD or pediatric neurology, onward referral to specialist services should be undertaken.

Parr et al. (2011) recently reported on the characteristics of regression in 124 children from a sample of 458 with ASD (27%). Early

developmental regression occurred in 23% of the children who lost skills. Late regression was more likely to be nonlanguage regression or a mix of language and nonlanguage regression. Social regression was the most frequently reported non language regression concern, but parents also described regression in other areas. In Parr’s study, most children with regression regained the skills they had lost and subsequently make developmental progress – the mean time from loss to skills being regained was 20 months. A small proportion of children with ASD and regression were reported by their parents to either experience a prolonged developmental plateau or not regain their skills for at least 2 years – these children had more evidence of developmental impairment than children who regained skills. In recent years, a number of studies have identified that language and cognitive outcomes are less good and that some autism symptoms are more severe in children with ASD who experience regression, compared with children with ASD with no history of regression (Baird et al. 2008; Parr et al. 2011). Data show that children with regression have a greater degree of social impairment (as measured by their ADI-R social domain score) and lower IQ scores and adaptive behavior scores. It is not yet known whether the long-term outcome differs for ASD children who lose skills, compared with those without regression.

While there are a lot of data showing what does not lead to regression, the cause of regression remains unknown. Lainhart et al. (2002) reported that regression was as common in families with the broader autism phenotype (BAP), as in those without the BAP, suggesting regression did not have a familial cause (a finding consistent with those of Parr et al. 2011). There were some early genetic findings of possible family association and genetic linkage, but this was not replicated by Parr and the International Molecular Genetic Study of Autism Consortium (Parr et al. 2006).

Treatment

When a medical cause of regression is found (e.g., an epileptic encephalopathy), this should be treated by a clinician with expertise in pediatric neurology. For most young children with ASD, the symptom profile in early development can be

very varied and although there is no evidence base for any specific treatment for regression, this presentation should inform the needs and skills based management plan (early intervention) for the child and family. Whether or not specific intervention aimed to improve speech and social communication alters developmental progress following regression is unknown. However, parents whose children regress and those professionals supporting the child and family should be encouraged to continue to use appropriate strategies to improve the child's social communication and monitor progress. Clinicians and community support staff should pay careful attention to the needs of parents and other family members at this time of increased stress and uncertainty for the family.

Future Directions

Prospective studies of infant siblings born in families with a child with ASD ("high-risk" studies) will be of particular value in documenting the course of regression and subsequent progress. Intervention studies with these young children with ASD may shed light on the utility of early intervention in an attempt to improve developmental progress and outcome following regression. However, it should be noted that such prospective studies will require large sample sizes to study enough siblings who will develop ASD and go on to lose skills (Ozonoff et al. 2010); thus, collaborative research strategies will be necessary to further improve our understanding of regression. Certain aspects of regression (e.g., skill loss after age 3 years) require systematic surveillance studies including very large populations. The prospective identification and early clinical referral of children who develop regression (either early or late) would also allow detailed investigation, including imaging, neurophysiology, and genetic and other laboratory testing.

See Also

- Aphasia
- Childhood Disintegrative Disorder

- Electroencephalogram (EEG)
- Rett Syndrome
- Thimerosal

References and Reading

- Baird, G., Charman, T., Pickles, A., Chandler, S., Loucas, T., Meldrum, D., et al. (2008). Regression, developmental trajectory and associated problems in disorders in the autism spectrum: The SNAP study. *Journal of Autism and Developmental Disorders*, 38, 1827–1836.
- Fombonne, E., & Chakrabarti, S. (2001). No evidence for a new variant of measles-mumps-rubella-induced autism. *Pediatrics*, 108, E58.
- Johnson, C. P., Myers, S. M., & The Council on Children with Disabilities. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120, 1183–1215. <https://doi.org/10.1542/peds.2007-2361>.
- Kurita, H. (1985). Infantile autism with speech loss before the age of thirty months. *Journal of the American Academy of Child Psychiatry*, 24, 191–196. [https://doi.org/10.1016/s0002-7138\(09\)60447-7](https://doi.org/10.1016/s0002-7138(09)60447-7).
- Lainhart, J., Ozonoff, S., Coon, H., Krasny, L., Dinh, E., Nice, J., et al. (2002). Autism, regression, and the broader autism phenotype. *American Journal of Medical Genetics*, 113, 231–237.
- Landa, R., Holman, K., & Garrett-Mayer, E. (2007). Social and communication development in toddlers with early and later diagnosis of autism spectrum disorders. *Archives of General Psychiatry*, 64, 853–864.
- Ozonoff, S., Iosif, A. M., Baguio, F., Cook, I. C., Hill, M. M., Hutman, T., et al. (2010). A prospective study of the emergence of early behavioral signs of autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 49, 256–266.e1-2.
- Parr, J. R., Lamb, J. A., Bailey, A. J., & Monaco, A. P. (2006). Response to paper by Molloy et al.: Linkage on 21q and 7q in autism subset with regression. *Molecular Psychiatry*, 11, 617–619.
- Parr, J. R., Le Couteur, A., Baird, G., Rutter, M., Pickles, A., Fombonne, E., et al. (2011). Early developmental regression in autism spectrum disorder: Evidence from an international multiplex sample. *Journal of Autism and Developmental Disorders*, 41, 332–340.
- Pickles, A., et al. (2009). Loss of language in early development of autism and specific language impairment. *Journal of Child Psychology and Psychiatry*, 50, 843–852. <https://doi.org/10.1111/j.1469-7610.2008.02032.x>.
- Richler, J., et al. (2006). Is there 'Regressive Phenotype' of autism spectrum disorder associated with the measles-mumps-rubella vaccine? A CPEA study. *Journal of Autism and Developmental Disorders*, 36, 299–316. <https://doi.org/10.1007/s10803-005-0070-1>.
- Rogers, S. J. (2004). Developmental regression in autism spectrum disorders. *Mental Retardation & Developmental Disabilities Research Reviews*, 10, 139–143.

- Tuchman, R. F., Rapin, I., & Shinnar, S. (1991). Autistic and dysphasic children. I: Clinical characteristics. *Pediatrics*, 88, 1211–1218.
- Werner, E., Dawson, G., Munson, J., & Osterling, J. (2005). Variation in early developmental course in autism and its relation with behavioral outcome at 3–4 years of age. *Journal of Autism and Developmental Disorders*, 35, 337–350. <https://doi.org/10.1007/s10803-005-3301-6>.

Regression Analysis

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Synonyms

[Linear regression](#); [Multiple regression](#)

Definition

Regression analysis is a statistical tool used to model the relationship between a dependent variable and one or more independent variables. Specifically, regression analysis describes how the typical value of the dependent variable changes when any one of the independent variables increases or decreases, while holding the other independent variables constant. For example, regression analysis has been utilized to demonstrate that sensory processing dysfunction significantly influences externalizing behavior among preschool-aged children with autism (Tseng et al. 2011).

See Also

- [Linear Regression](#)
- [Multiple Regression](#)

References and Reading

- Alderson-Day, B., & McGonigle-Chalmers, M. (2011). Is it a bird? Is it a plane? Category use in problem-solving

- in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 41, 555–565.
- Hilton, C. L., Harper, J. D., Kueker, R. H., Lang, A. R., Abbacchi, A. M., Todorov, A., & LaVesser, P. D. (2010). Sensory responsiveness as a predictor of social severity in children with high functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40, 937–945.
- Jones, A. P., & Frederickson, N. (2010). Multi-informant predictors of social inclusion for students with autism spectrum disorders attending mainstream school. *Journal of Autism and Developmental Disorders*, 40(9), 1094–1103.
- Tseng, M., Fu, C., Lu, L., & Shieh, J. (2011). Emotional and behavioral problems in preschool children with autism: Relationship with sensory processing dysfunction. *Research in Autism Spectrum Disorders*, 5(4), 1441–1450.
- Weismer, S. E., Lord, C., & Esler, A. (2010). Early language patterns of toddlers on the autism spectrum compared to toddlers with developmental delay. *Journal of Autism and Developmental Disorders*, 40, 1259–1273.

Regressive Autism

- [Acquired Autism](#)

Rehabilitation

- [Physical Therapy](#)

Rehabilitation Act of 1973

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Definition

The Rehabilitation Act of 1973 was a national strategy to improve the prospects for individuals with disabilities and ban judgment in employment against otherwise talented handicapped individuals. This Act was to substitute the Vocational Rehabilitation Act and to cover and revise the approval of awards to States for vocational rehabilitation services, with distinct weight on services to those with the most severe handicaps; to increase special Federal accountabilities and

teaching programs with respect to handicapped persons; to institute special responsibilities in the Secretary of Health, Education, and Welfare for organization of all programs with respect to handicapped individuals within the Department of Health, Education, and Welfare; and for other purposes. The Rehabilitation Act of 1973 had 11 different purposes:

1. Develop and implement complete State plans for meeting present and prospect needs for providing vocational rehabilitation services to handicapped individuals, serving first those with the most severe handicaps
2. Assess rehabilitation potential of handicapped persons
3. Conduct a study to develop approaches of providing rehabilitation services to meet the existing and forthcoming needs of handicapped persons for whom vocational aim is not likely or viable so that they may progress their capability to live with better independence and self-sufficiency
4. Help in the creation and enhancement of rehabilitation services
5. Advance novel and groundbreaking methods of applying the greatest advanced medical technology, scientific successes, and psychological and social information to resolve rehabilitation problems and develop new and innovative methods of providing rehabilitation services for handicapped persons through research, special projects, and demonstration
6. Start and enlarge services to groups of handicapped individuals who have been underserved in the past
7. Conduct many studies and tests to emphasis on long abandoned problem areas
8. Encourage and expand employment opportunities in the communal and private areas for handicapped persons and to place such individuals in employment
9. Create customer assistance pilot projects
10. Offer aid for the purpose of increasing the number of rehabilitation personnel and increasing their skills through training
11. Evaluate existing approaches to architectural and transportation barriers confronting handicapped persons and advance novel methods and solutions to current architectural and transportation barriers impeding handicapped individuals

See Also

- [Americans with Disabilities Act](#)
- [Department of Vocational Rehabilitation](#)
- [Vocational Evaluator](#)
- [Vocational Training](#)

References and Reading

- Americans with Disabilities Act of 1990; Pub L No. 101-336, 42 USC § 12101 (1990).
- David, A. L. (1986). What disabilities are protected under the Rehabilitation Act of 1973? *University of Memphis Law Review*, 16, 229.
- Horn, L., & Berkold, J. (1999). *Students with disabilities in postsecondary education: A profile of preparation, participation and outcomes*. Washington, DC: US Dept of Education, National Center for Education Statistic; NCES 1999-187. Available at: <http://nces.ed.gov/pubs99/1999187.pdf>
- Horn, L., Peter, K., & Rooney, K. (2002). *Profile of undergraduates in U.S. postsecondary institutions: 1999–2000*. Washington, DC: US Dept of Education, National Center for Education Statistics; NCES 2002-168. Available at: <http://nces.ed.gov/pubs2002/2002168.pdf>
- Rehabilitation Act of 1973, Pub.L. 93-112, 87 Stat. 355, H.R. 8070, enacted September 26, 1973.
- Sherr, M. A., & Babovich, W. M. (1997). Broadening the scope of the rehabilitation act of 1973. *Nursing Management*, 28(6), 41–42.

Rehabilitation Engineer

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Synonyms

[Biomedical engineer](#)

Definition

A rehabilitative engineer is a person who applies engineering principles to develop assistive technology to aid individuals with different kinds of disabilities. Rehabilitative engineering is considered a subspecialty of biomedical engineering.

As defined by the Rehabilitation Act of 1973, amended in 1998, rehabilitative engineering is the systematic application of engineering sciences to design, develop, adapt, test, evaluate, apply, and distribute technological solutions to problems confronted by individuals with disabilities in functional areas such as mobility, communications, hearing, vision, and cognition and in the activities associated with employment, independent living, education, and integration in the community (Engineers Australia 2011).

Rehabilitative engineers are the key to the development and delivery of “assistive technology,” a term which refers to technologies and principles that meet the needs of and address the barriers confronted by individuals with disabilities in a range of life areas. Assistive technology is often associated with education, rehabilitation, employment, transportation, independent living, and recreation (Engineers Australia 2011).

See Also

- ▶ [Augmentative and Assistive Technology](#)
- ▶ [Individuals with Disabilities Education Act \(IDEA\)](#)
- ▶ [Vocational Rehabilitation Act of 1973](#)

References and Reading

- Engineers Australia. (2011). *What is rehabilitative engineering?* Retrieved 25 July 2011, from http://www.engenhariadereabilitacao.net/arquivo/Brochura_CNERAustraliano.pdf
- National Assistive Technology Technical Assistance Partnership. (2012). Retrieved 18 June 2012, from <http://www.resnaprojects.org/nattap/>
- Rehabilitation Engineering Society of North America (RESNA). *begin_of_the_skype_highlighting*. <http://www.resna.org>

Reinforcement

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Synonyms

[Adding](#); [Reinforcing stimulus](#); [Strengthening](#); [Supporting](#)

Definition

Reinforcement is defined as strengthening a specific response (Skinner 1963). For example, imagine a scenario where a mother is attempting to teach her child with autism to say the word “bubble.” Every time the child attempts to say the word “bubble” on command, the mother might, strengthen the response by blowing bubbles, thus reinforcing the behavior. Moreover, reinforcement is responsible for the continued performance of the behavior that has been acquired (Skinner 1963). There are two types of reinforcement: positive reinforcement and negative reinforcement. Positive reinforcement is strengthening a behavior by rewarding an attempted response (Baltruschat et al. 2011; Fisher et al. 2005). The bubble scenario described above is an example of positive reinforcement. Negative reinforcement, on the other hand, is strengthening a behavior by removing a stimulus (Fisher et al.). For example, if a child with autism was engaging in self-injurious behavior by banging his head on a table during difficult tasks, and the teacher removed the difficult task, the teacher might negatively reinforce the child’s self-injurious behavior.

See Also

- ▶ [Negative Reinforcement](#)
- ▶ [Operant Conditioning](#)
- ▶ [Positive Reinforcement](#)

References and Reading

- Baltruschat, L., Hasselhorn, M., Tarbox, J., Dixon, D. R., Najdowski, A. C., Mullins, R. D., et al. (2011). Addressing working memory in children with Autism through behavioral intervention. *Research in Autism Spectrum Disorders*, 5, 267–276.
- Fisher, W. W., Adelinis, J. D., Volkert, V. M., Keeney, K. M., Neidert, P. L., & Hovanetz, A. (2005). Assessing preferences for positive and negative reinforcement during treatment of destructive behavior with functional communication training. *Research in Developmental Disabilities*, 26, 153–168.
- Skinner, B. F. (1963). Operant behavior. *American Psychologist*, 18(8), 503–515.

Reinforcement, Differential

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Definition

Differential reinforcement is a behavioral analytic term related to the principle of reinforcement and the process of extinction. Differential reinforcement involves providing reinforcement contingent on the display of specific responses, not all responses. Differential reinforcement serves to refine or shape certain responses and reduces or eliminates other responses that do not meet the criteria for reinforcement. Responses that are differentially reinforced are intended to increase and meet a prespecified criterion defined in terms of some response dimension, including frequency, topography, intensity, duration, latency, or magnitude. The responses that do not meet the criteria for reinforcement are not reinforced, put on extinction, and decreased. Differential reinforcement is a useful and naturally occurring process

for increasing some behavior while eliminating other behavior. The effectiveness of differential reinforcement to increase or eliminate specific targeted behaviors has been demonstrated both in laboratory studies with nonhuman organisms (e.g., rats, nonhuman primates) and in applied studies conducted with children with autism and other developmental disabilities, thus illustrating the clinical usefulness and the generality of the procedure. In the applied literature, differential reinforcement of other behavior has been demonstrated to reduce socially maintained aggression, self-injury, and property destruction; differential reinforcement of alternative behavior has been demonstrated to increase such behaviors as alternative communication behaviors and engagement in functional activities.

See Also

- [Differential Reinforcement Procedures of Other Behavior \(DRO\)](#)
- [Extinction Procedures](#)
- [Reinforcement](#)
- [Shaping of Behavior](#)

References and Reading

- Catania, A. C. (2013). *Learning* (5th ed.). Cornwall-on-Hudson: Sloan Publishing.
- Mazaleski, J. L., Iwata, B. A., Vollmer, T. R., Zarcone, J. R., & Smith, R. G. (1993). Analysis of the reinforcement and extinction components in DRO contingencies with self-injury. *Journal of Applied Behavior Analysis*, 26(2), 143–156. <https://doi.org/10.1901/jaba.1993.26-143>.
- Vollmer, T. R., & Iwata, B. A. (1992). Differential reinforcement as treatment for behavior disorders: Procedural and functional variations. *Research in Developmental Disabilities*, 13(4), 393–417.
- Vollmer, T., Roane, H., Ringdahl, J., & Marcus, B. (1999). Evaluating treatment challenges with differential reinforcement of alternative behavior. *Journal of Applied Behavior Analysis*, 32(1), 9–23. <http://doi.org/10.1901/jaba.1999.32-9>.
- Watts, A. C., Wilder, D. A., Gregory, M. K., Leon, Y., & Ditzian, K. (2013). The effect of rules on differential reinforcement of other behavior. *Journal of Applied Behavior Analysis*, 46(3), 680–684.

Reinforcer

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Synonyms

Reward

Definition

Reinforcement is demonstrated when a consequence is delivered following a behavior and subsequently that behavior is strengthened (e.g., occurs more in the future). When behavior increases (i.e., has been reinforced), the stimulus or event that followed the behavior (i.e., consequence) was a *reinforcer*. Thus, a reinforcer is any consequence for a response that has been shown to increase (or maintain) the future probability of that response. In other words, a consequence is only a reinforcer *if* the target behavior continues to occur or occurs more often. A common example could include praising a child for putting his/her toys away. A parent saying “way to go!” is a reinforcer if the child puts his/her toys away more in the future. If the child stops putting his/her toys away in the future, then praise, despite the parent’s best intentions, was *not* a reinforcer for the behavior of putting toys away. Research on operant behavior has consistently demonstrated the generality and importance of reinforcers in learning (i.e., increasing behavior; see Skinner 1938, 1953).

For a consequence to be a reinforcer, it must address some current motivation. For example, a high five will only increase a behavior (i.e., be a reinforcer) if the person currently wants a high five. On the other hand, if the person does not want a high five now, then the high five will not

reinforce current behavior (i.e., serve as a reinforcer). Similarly, a consequence will may be a reinforcer for some behaviors and not others. For example, a high five may be a reinforcer for the behavior of saying “thank you.” In contrast, a high five may not be a reinforcer for a different behavior, such as running 5 miles.

In working with individuals with autism spectrum disorders (ASDs), reinforcers will be needed to increase appropriate behaviors and, whenever possible, reinforcers should not be provided following inappropriate behaviors. Several assessment strategies are particularly important for identifying likely reinforcers for both appropriate and inappropriate behavior. Preference assessments can be used to identify likely reinforcers that may then be included in individualized interventions to increase appropriate behavior (e.g., DeLeon and Iwata 1996; Fisher et al. 1992). Alternatively, functional behavior assessments (FBAs) have been used to identify reinforcers for inappropriate behaviors (e.g., functional analysis; Iwata et al. 1994). Once reinforcers for inappropriate behaviors are identified, then interventions can be designed to increase an alternative, appropriate response and minimize or eliminate reinforcers for the inappropriate behavior (e.g., Carr and Durand 1985).

Reinforcers may be categorized as naturally occurring or contrived. In each case, the consequence is a reinforcer because it increased behavior. In the case of a naturally occurring reinforcer, the consequence is one that is commonly found in natural environments and situations. In contrast, a contrived reinforcer is a consequence that may not be common for that behavior in typical environments or situations. An example of a naturally occurring reinforcer is being called on after raising one’s hand in class. Alternatively, being given a token for raising one’s hand might be considered a contrived reinforcer. Naturally occurring consequences are generally preferable to contrived consequences because the behavior would be expected to continue in natural situations. However, for many individuals with ASDs, naturally occurring consequences may not reinforce their behavior. In other words, if the naturally occurring

consequence is the only consequence for their behavior, they will not continue to engage in that behavior. In these situations, contrived reinforcers will likely be needed before the learner will make progress. Over time, it may be possible to fade out (i.e., remove) the additional, contrived reinforcer (s), leaving only the naturally occurring consequences which may have become reinforcers.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Functional Analysis](#)
- [Functional Behavior Assessment](#)
- [Motivation](#)
- [Negative Reinforcement](#)
- [Positive Reinforcement](#)
- [Reinforcement](#)

References and Reading

- Carr, E. G., & Durand, V. M. (1985). Reducing behavior problems through functional communication training. *Journal of Applied Behavior Analysis*, 18, 111–126.
- DeLeon, I. G., & Iwata, B. A. (1996). Evaluation of a multiple-stimulus presentation format for assessing reinforcer preferences. *Journal of Applied Behavior Analysis*, 29, 519–533.
- Fisher, W. W., Piazza, C. C., Bowman, L. G., Hagopian, L. P., Owens, J. C., & Slevin, I. (1992). A comparison of two approaches for identifying reinforcers for persons with severe and profound disabilities. *Journal of Applied Behavior Analysis*, 25, 491–498.
- Iwata, B. A., Dorsey, M. F., Slifer, K. J., Bauman, K. E., & Richman, G. S. (1994). Toward a functional analysis of self-injury. *Journal of Applied Behavior Analysis*, 27, 197–209. (Reprinted from *Analysis and Intervention in Developmental Disabilities*, 2, 3–20, 1982).
- Skinner, B. F. (1938). *The behavior of organisms*. Acton: Copley Publishing Group.
- Skinner, B. F. (1953). *Science and human behavior*. New York: The Free Press.

Reinforcing Stimulus

- [Reinforcement](#)

Related Services

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Definition

Are supportive services provided by an educational agency such as a school district that are required to assist a child with a disability in obtaining benefit from special education. Related services are specified on a student's individual education plan. Included under the term are speech and language pathology and audiology services; interpreting services; psychological services; physical and occupational therapy; social work services; recreation, including therapeutic recreation; early identification and assessment of disabilities in children; counseling services, including rehabilitation counseling, orientation, and mobility services; and medical services for diagnostic or evaluation purposes. Related services also include school health services and school nurse services, social work services in schools, and parent counseling and training. The list of services provided here is not exhaustive; other services may need to be provided if an IEP deems that they are necessary for the student to obtain benefit from special education programming.

See Also

- [Augmentative and Assistive Technology](#)

References and Reading

- Assistance to States for the Education of Children with Disabilities and Preschool Grants for Children with Disabilities; Final Rule, 2004, 4000-01-U Department of Education 34 CFR Parts 300 and 301, Part II.
- Individuals with Disabilities Education Act of 2004, 20 U.S.C. et. seq.

Relationship Between Autism Stigma and Informal Caregiver Mental Health

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Definition

The relationship between autism stigma and informal caregiver mental health refers to the negative psychological consequences for informal caregivers when autism stigma is experienced. This has been evidenced to include depression, anxiety, psychological distress and well-being, psychological burden, and general mental health. “Autism stigma” refers to the negative social labeling and stereotyping ascribed by members of the general public toward autistic people (“public stigma” or “social stigma”) and/or their family and close associates (“courtesy stigma,” “associative stigma,” or “family stigma”). Autism stigma may also originate from health, social, educational, or legal professionals (“professional stigma” or “structural stigma”). “Self-stigma” (or “internalized stigma”) refers to autistic people accepting, agreeing, and incorporating negative stereotypes about autism into their own psychological identity. “Affiliate stigma” is a form of self-stigma. It occurs when informal caregivers of autistic people experience courtesy stigma and subsequently agree and incorporate the negative stereotypes directed toward them into their own psychological identity. The ways in which autism stigma is produced, manifested, and experienced are socially and culturally specific.

See Also

- [Autism Acceptance and Mental Health](#)
- [Stigmatization](#)

References and Reading

- Mak, W. W., & Cheung, R. Y. M. (2008). Affiliate stigma among caregivers of people with intellectual disability or mental illness. *Journal of Applied Research in Intellectual Disabilities*, 21(6), 532–445.
- Papadopoulos, C. (2016a). Self-stigma among carers of autistic people. *Network Autism*. <https://network.autism.org.uk/good-practice/evidence-base/self-stigma-among-carers-autistic-people>
- Papadopoulos, C. (2016b). Autism stigma and the role of ethnicity and culture. *Network Autism*. <https://network.autism.org.uk/knowledge/insight-opinion/autism-stigma-and-role-ethnicity-and-culture>
- Papadopoulos, C., Lodder, A., Constantinou, G., & Randhawa, G. (2018). Systematic review of the relationship between autism stigma and informal caregiver mental health. *Journal of Autism and Developmental Disorders*, 49(4), 1665–1685.

Relationship Development Intervention (RDI) Model

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Definition

The Relationship Development Intervention[®] (RDI) program is a comprehensive, parent-directed intervention designed to facilitate the social, emotional, and cognitive development of children, adolescents, and adults with autism spectrum disorders (ASD). The primary objective of the RDI program is to promote the development of dynamic intelligence through the restoration of a guided participation relationship (GPR) between the parent and individual with ASD. Gutstein (2009) suggests that dynamic intelligence is a set of mental processes, such as anticipating, evaluating, and integrating, which allow individuals to be flexible in their thinking, problem-solving, and coping with uncertain situations. It is the product of dynamic neural connectivity, which develops early on in childhood as a result of experiences provided by parents during guided participation

(Gutstein 2009). The concept of guided participation comes from the work of Vygotsky (1978) and Rogoff (1990). It is described as the process by which a more experienced adult scaffolds a child's competence and understanding by providing challenging experiences that are slightly above their current level of ability. Parents work in conjunction with a certified RDI consultant to establish a GPR through a parent-guiding curriculum and to facilitate the development of dynamic intelligence through a developmentally based dynamic intelligence curriculum (Gutstein 2009). Intervention objectives are targeted during daily activities, and the parent gradually assumes primary responsibility for implementation of the program with training from the RDI consultant.

Historical Background

The RDI program was developed by Dr. Steven Gutstein and Dr. Rachelle Sheely to “remediate information-processing deficits that are universal but by no means unique to persons on the autism spectrum” (Gutstein 2009, p. 23). The program came as a response to what Gutstein saw as a lack of intervention programs that addressed the core deficits of ASD and impacted the overall quality of life of individuals with ASD. The first book detailing the RDI program was published in 2000, followed by the publication of two RDI activity books, one aimed at young children (Gutstein and Sheely 2002a) and one aimed at children, adolescents, and adults (Gutstein and Sheely 2002b). In 2009, *The RDI Book: Forging New Pathways for Autism, Asperger's and PDD with the Relationship Development Intervention Program* was published and serves to replace previous works. The 2009 RDI book includes several modifications to the program including a systematized dynamic intelligence curriculum and a parent-guiding curriculum (Gutstein 2009). An RDI consultant training program, program for generalizing RDI in a school setting (Gutstein et al. 2007b), and an interactive Internet-based application have also been developed. The Internet-based

application, which was launched in 2007, is referred to as the RDI Learning System (RDILS). The RDILS allows parents, teachers, and RDI consultants in training to access information about the program, attend online seminars, and communicate with other individuals around the world. Additionally, parents may communicate with consultants through the RDILS, upload videos, and track their progress (Gutstein 2009). Gutstein (2009) indicates that currently, there are nearly 400 RDI consultants and consultants in training in 20 different countries.

Rationale or Underlying Theory

The RDI program was developed to remediate impairments in information processing that Gutstein (2009) suggests are the core deficits in individuals with ASD. Collectively, these information-processing impairments lead to challenges in “dynamic intelligence,” which allows individuals to be flexible thinkers, problem-solve effectively, collaborate with others, and cope with uncertain situations. This is contrasted with static intelligence, which is a representation of our knowledge, and includes abilities such as concept learning, categorizing, memorizing facts and procedures, and numerical computation (Gutstein 2009). Dynamic intelligence is the result of the elaborate neural integration that begins to develop in early childhood in response to exposure to cognitively challenging situations. Typically, the adult creates challenging learning opportunities through guided participation.

The concept of guided participation was first described by Rogoff (1991), who asserts that “both guidance and participation in culturally valued activities are essential to children's apprenticeship in thinking” (1991, p. 8). Guided participation occurs when the adult provides situations that are safe but slightly outside of the child's current independent capability, thus creating a cognitive challenge that requires problem-solving (Gutstein 2009). Participation in these uncertain situations facilitates the development

of complex and dynamic neural networks in the brain. Rogoff (1991) suggests that this relationship occurs naturally between caregivers and children and appears to be universal in some form across cultures.

According to Gutstein (2009), ASD is fundamentally a neurological disorder resulting from a paucity of dynamic neural interconnectivity in the brain. Recent research suggests that there is indeed evidence of neural underconnectivity in the brains of individuals with ASD (for a review, see Hughes 2007). Gutstein (2009) theorizes that a disruption of the guided participation relationship between parents and their children with ASD impedes the development of dynamic neural pathways while leaving static pathways generally intact. Thus, the RDI program was specifically designed to support the development of a guided participation relationship in order to facilitate dynamic intelligence development in individuals with ASD.

Goals and Objectives

As previously discussed, the fundamental goal of the RDI program is to facilitate the development of dynamic intelligence in individuals with ASD through guided participation. Dynamic intelligence is an overarching term used to refer to a set of skills, which typically develop in a sequential pattern. The RDI program employs a developmental approach to support the specific skills or objectives comprising dynamic intelligence. The dynamic intelligence curriculum utilized by the program currently consists of nearly 1,000 objectives that are targeted during intervention (Gutstein 2009). However, Gutstein (2009) indicates that the overarching goal of the RDI program is to enhance the quality of life of individuals with ASD. Thus, eight specific RDI goals, which represent characteristics determined to be essential to attaining quality of life, have been developed:

1. *Collaboration* – Refers to the ability to coordinate one's own thoughts and actions with a partner's to achieve a common goal.

Successful collaboration requires, among other things, negotiation, adaptation of communication, and monitoring of coordination.

2. *Deliberation* – Refers to the ability to approach unfamiliar situations and problems in a thoughtful and analytic manner. This may involve considering multiple approaches and perspectives before acting and anticipating the possible consequence of actions.
3. *Flexibility* – Refers to the ability to continually monitor and adjust actions based on past experiences, new information, and changing circumstances. It also involves the ability to manage problems that have no distinct right or wrong answer by considering multiple perspectives and thinking creatively.
4. *Fluency* – Refers to skills that are important to academic and employment success. This includes the ability to read for comprehension, summarize and synthesize information, write in an organized manner using appropriate syntax and spelling, communicate information through a variety of modes, and demonstrate competent use of computers, software, and other technical equipment.
5. *Friendship* – Refers to the ability to maintain relationships that are based on shared interests and experiences. Friendship requires empathy, loyalty, emotional coordination, and the ability to successfully manage conflict.
6. *Initiative* – Refers to the ability to independently initiate and complete tasks, set personal goals, and find alternate routes to success when things do not go as initially planned. It also involves the exploration of new ideas and skills even when they are challenging or unfamiliar.
7. *Responsibility* – Refers to the ability to act with integrity, take responsibility for one's own actions, honor commitments and promises, and care for items.
8. *Self-management* – Refers to, among other things, the ability to maintain healthy habits (e.g., hygiene, diet, sleep), effectively manage time, establish and maintain daily living routines that increase independence (e.g., paying bills on time), accurately evaluate personal strengths and limitations in relation to goals, and develop personal opinions, beliefs, and

values. Further, it involves the ability to effectively manage emotions and cope with negative emotions (e.g., stress, anger, frustration) in a productive manner.

Treatment Participants

The RDI program was designed specifically for individuals on the autism spectrum, including those with diagnoses of autism, Asperger's syndrome, and PDD-NOS. However, Gutstein (2009) indicates that consultants have reported success in working with individuals with other diagnoses such as reactive attachment disorder, attention deficit hyperactivity disorder, Tourette's syndrome, bipolar disorder, and seizure disorders. Because the program is highly individualized and flexible, treatment participants can vary greatly in terms of age and function. There are currently no set of age criteria. Participants may range in age from young children to adolescents and adults. Gutstein (2009) indicates that RDI consultants are trained to work closely with the family to individualize the program to be appropriate and effective for the participant. Additionally, the program is based on a developmental model, so no prerequisite skills are required to begin intervention. Instead, the intervention is tailored to fit the needs of participants with varying levels of social-emotional, language, and cognitive abilities.

Treatment Procedures

The RDI program is a parent-directed, developmentally based intervention, which aims to develop dynamic intelligence in individuals with ASD through guided participation. Parents are paired with an RDI-certified consultant in their area whose role is to customize the program based on the unique needs of the family and to facilitate parents in implementing the program. Consultants teach parents the tools that are essential to guided participation so that parents may become gradually more independent. Parents meet with consultants during live sessions to

practice assignments, discuss obstacles, and collaborate. Parents also communicate with consultants during the time between live sessions online through RDILS. There is no set time devoted to intervention each week. Instead, with the support of their consultant, parents learn how to effectively incorporate guided participation into daily activities so that this way of interacting with their child becomes an integral aspect of daily life.

There are four stages in the RDI program: Readiness (parent and child), Apprenticeship, Guiding, and Internalizing. The program utilizes the dynamic intelligence curriculum that was constructed over the past 10 years to represent the typical progression of skills and abilities contributing to dynamic intelligence (Gutstein 2009). The curriculum currently consists of nearly 1,000 developmentally sequenced objectives (Gutstein 2009). Each stage in the RDI program has a set of objectives, and each objective is further subdivided into lessons. Every lesson also has its own unique set of mastery criteria that the child must attain before moving to a more complex skill. The stages of the RDI program are briefly outlined below:

- *Readiness Stage* – During this stage, children are evaluated for obstacles to apprenticeship, which are addressed before full participation as an apprentice occurs. Potential obstacles include comorbid conditions such as seizure disorder, attention deficit disorder, anxiety, and sleep disorders and the failure to acquire prerequisite skills considered fundamental to successful involvement in a guided participation relationship (Gutstein 2009). At this stage, parents also learn to adjust their physical patterns of interaction and communication style and to identify, assess, and modify the expectations they hold for their child and for themselves.
- *Apprenticeship Stage* – A primary focus of this stage is the development of a trusting guide-apprentice relationship. Parents learn to carefully choose challenges that are appropriate for their child's unique needs and that are within their zone of proximal development while also developing an understanding that they cannot

necessarily predict how their child will react to the challenges they present. Parents also continue to adapt their communication style to support social reciprocity and mutual experience sharing.

- *Guiding Stage* – During this stage, children are introduced to the primary curriculum, which consists of several hundred objectives (Gutstein 2009). While previous stages focus primarily on scaffolding the child's active participation as an apprentice, the Guiding stage explicitly emphasizes the development of dynamic intelligence. Additionally, parents begin to assume greater responsibility in their role as a guide, as the fundamental skills for guided participation are transferred from the RDI consultant to the parent.
- *Internalizing Stage* – During this stage, parents assume primary responsibility for the tools of guided participation that they have learned from their consultant and learn to choose appropriate curriculum objectives for their child and create their own corresponding lessons. They also demonstrate greater independence, as time with their consultant is decreased. The curriculum objectives at this stage become increasingly complex, focusing primarily on internal processes, such as reflection and imagination, and older apprentices begin to take a more active role in the process of choosing objectives and creating lesson plans (Gutstein 2009). New communication partners and environments are introduced to facilitate the generalization of skills into real-world situations. For example, when the apprentice has mastered objectives with adults, a familiar peer may be introduced. Once competency with a peer has been demonstrated, the child may move on to a small group setting.

Efficacy Information

As of 2010, Gutstein and his colleagues have conducted two studies evaluating the efficacy of the RDI program. The initial study, which is outlined by Gutstein et al. (2007a), compared a group of children with ASD participating in the

RDI program for an average of 16 months and a group of children with ASD being treated with "more traditional intensive intervention methods" (p. 398). The authors report that more than 50% of children in the RDI group no longer met ADOS criteria for autism spectrum disorders and were more likely to participate in a typical classroom without support (Gutstein et al. 2007a). In comparison, the children participating in more traditional methods of intervention did not demonstrate significant improvements in core deficit areas, although they received greater direct therapist contact (Gutstein et al. 2007a).

In their follow-up study, Gutstein et al. (2007a) evaluated 16 children who participated in the RDI program between 2000 and 2005. Inclusion criteria for participants included a minimum of 30 months of participation in the RDI program between baseline and follow-up measures, an age of 20–96 months at the time treatment was initiated, a previous diagnosis of autism, Asperger's syndrome, or Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS), and a baseline IQ score of at least 70 (Gutstein et al. 2007a). Effectiveness of the program was based on the following measures: scores on the Autism Diagnostic Interview-Revised (ADI-R; Rutter et al. 2003) and the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2002), a semi-structured flexibility interview, and educational placement.

Of the 12 participants for which baseline ADOS scores were available, 10 met diagnostic criteria for autism and 2 met criteria for autism spectrum. Following treatment, six participants met diagnostic criteria for autism spectrum, while ten were placed in the "non-autism" diagnostic category (Gutstein et al. 2007a). Similarly, mean scores for the sum of the communication and social interaction domains on the ADI-R were 10.6 at baseline and 2.4 at follow-up, indicating a significant decrease in impairment over the course of treatment (Gutstein et al. 2007a). On the flexibility interview, parent ratings indicating age-appropriate flexibility on the 10-item questionnaire increased from a mean of 16% at baseline to a mean of 71% at follow-up. Finally, in regard to educational placement, only two

participants were placed in a mainstream classroom without an aide at baseline. However, at follow-up, 10 out of 16 children were placed in a mainstream classroom without an aide.

As Gutstein and colleagues (2007) points out, there are several limitations that impact the generalizability of the reported findings. These include the lack of a control group, a small number of participants, a narrow range of age and IQ scores for the participants, and the geographical homogeneity of participants. Nonetheless, this study, along with the initial study previously discussed, provides preliminary evidence for the efficacy of the RDI program. However, these results should be interpreted with caution, as they are only preliminary and lack generalizability due to study limitations. Future research including larger samples and control groups will be fundamental in determining the potential benefits of the RDI program for individuals with ASD.

Outcome Measurement

RDI consultants track the participants' progress as they move through the stages of the dynamic intelligence curriculum. Every objective in the curriculum is subdivided into lessons, which each have a unique and observable set of mastery criteria. Consultants work closely with parents to determine if mastery criteria for a particular lesson have been achieved.

Qualifications of Treatment Providers

Individuals interested in applying for RDI certification are required to read *The RDI Book* (2009), hold a bachelor's degree, and complete an application for certification. RDI consultants may have a variety of professional backgrounds, including speech and language pathologists, psychologists, teachers, social workers, autism specialists, occupational therapists, and physicians (Gutstein 2009). Additionally, parents of children participating in the RDI program may also apply for certification with a recommendation letter from their RDI consultant. The certification process,

which typically takes 18 months to complete, includes four steps: (1) Beginning Seminar, (2) Intermediate Seminar, (3) Professional Supervision, and (4) Advanced Seminar. The Beginning and Intermediate Seminars focus primarily on the Family Guided Participation Program, while the Advanced Seminar focuses mainly on the Dynamic Education Program. During the professional supervision step, consultants in training (CIT) are required to work with two families under the supervision of an RDI-certified consultant.

See Also

- [Neural Mechanisms in Autism](#)
- [Social Communication](#)
- [Zone of Proximal Development \(ZPD\)](#)

References and Reading

- Gutstein, S. E. (2001). *Autism Aspergers: Solving the relationship puzzle-A new developmental program that opens the door to lifelong social and emotional growth*. Arlington: Future Horizons.
- Gutstein, S. E. (2009). *The RDI book: Forging new pathways for autism, Asperger's and PDD with the relationship development intervention® program*. Houston: Connections Center.
- Gutstein, S., & Sheely, R. (2002a). *Relationship development intervention with young children: Social and emotional development activities for Asperger's, autism, PDD and NLD*. London: Jessica Kingsley.
- Gutstein, S., & Sheely, R. (2002b). *Relationship development intervention with children, adolescents and adults: A comprehensive program for social and emotional development in autism, PDD and NLD*. London: Jessica Kingsley.
- Gutstein, S. E., Burgess, A. F., & Montfort, K. (2007a). Evaluation of the relationship development intervention program. *Autism, 11*(5), 397–411.
- Gutstein, S., Gutstein, H., & Baird, C. (Eds.). (2007b). *The relationship development intervention program and education*. Houston: Connections Center.
- Hughes, J. R. (2007). Autism: The first finding = underconnectivity. *Epilepsy & Behavior, 11*(1), 20–24.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2002). *Autism diagnostic observation schedule*. Los Angeles: Western Psychological Services.
- Rogoff, B. (1990). *Apprenticeship in thinking: Cognitive development in social context*. New York: Oxford University Press.

- Rogoff, B. (1991). *Apprenticeship in thinking: Cognitive development in social context*. New York: Oxford University Press.
- Rutter, M., Le Couteur, A., & Lord, C. (2003). *ADI-R: The autism diagnostic interview-revised*. Los Angeles: Western Psychological Services.
- Vygotsky, L. (1978). Interaction between learning and development. In *Mind and society* (pp. 79–91). Cambridge, MA: Harvard University Press.

Relationship Enhancement Methods

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Definition

Relationship enhancement methods are typically regarded as interventions that use strategies aligned to a developmental or social learning theoretical framework. Some interventions used to promote the development of social interactions and relationships are derived from the principles of applied behavior analysis (Cooper et al. 2007). Relationship enhancement methods focus on the development of appropriate emotional responses and relationship building for individuals, in natural contexts, under the guidance of peers or adults, and through repetition in small, manageable steps. These methods incorporate the manipulation of the individual's environment to increase social interaction, social engagement, and both sensory and emotional regulation (Quinn and Malone 2000). The overarching aim is to address the early and profound disruption in a child with Autism Spectrum Disorders interpersonal-affective relatedness (Hobson, 1990 as cited in Simpson 2005). Originally designed to improve the quality of interaction between the parent and child with ASD (Greenspan 1998 as cited in Marcus et al. 2005), many of these methods have been extended to improve the quality of interaction

between the individual with ASD and other key members of their immediate social circle, such as siblings, peers within the school environment, and members of their community (coaches, team members, club group members, etc.).

Historical Background

Relationship enhancement methods that occur within natural contexts find their earliest basis in the developmental theoretical frameworks suggested by Bandura (1977), Piaget (1950), and Vygotsky (1978). Collectively, these frameworks suggest that the development of social interaction occurs across a developmental continuum, in the context of naturally provided support and responding from significant others, within multiple environments. All provide a basis for activities that may be used for methods of intervention to support the social relationship interactions of individuals with ASD.

Bandura (1977), as a social-learning theorist, supported the understanding that interaction is determined by interdependence of the factors of behavioral actions, personal knowledge and understanding, and environmental scenarios, with varying degrees of influence of each factor depending upon the situation or context. Those who develop approaches based upon Bandura emphasize increasing self-regulatory capacities for interaction and observational learning of others within the environment. This theory may be the basis for activities that include modeling of behavior, such as peer modeling and video modeling (Hall 2018).

Piaget (1950) emphasized the link between cognition and affect. He believed that positive affect during interactive learning facilitated memory, increasing cognition. It is possible that collectively these would support the development of long-term social relationships. His framework includes creating opportunities for individuals to engage in sensorimotor activities as a precursor to language, an integral component to the development of social interaction.

Vygotsky (1978) promoted learning through a sociocultural context, suggesting that most

learning takes place in a social context. His work supports the use of peers and group structure to develop social relationships. Those who use Vygotsky's work as a foundation to their approach emphasize knowledge and use of a students' "zone of proximal development," which is his/her true potential, paired with scaffolding to increase social interaction skills. He supports the Piaget axiom that social interaction is fundamental for intellectual development (Hall 2018 p. 124).

Developmental patterns of socio-emotional development have been outlined by Daniel Stern (1985) and L. Alan Sroufe (1995). Stern (1985) shares the development of one's sense of self (awareness of emotions, self-regulation of emotions, perspective taking, joint attention linked to knowledge of others, interpersonal interaction) and its process in social relatedness. He presents four development stages of emotion (emergent self, sense of core self, subjective self, and verbal self) that begin preverbally and later emerge through language or self-reflection. Approaches based upon Stern's work provide opportunities to increase this sense of self through activities such as those that encourage joint attention and imaginative play (Hall 2018, p. 125). Sroufe (1995) supports the understanding that emotion follows a developmental pattern, strongly linked to social development, known as socio-emotional development. He outlines the emergence of emotional development over the first 54 months of life (Hall 2018, p. 126) and notes the shift that occurs within the developmental process as one that moves from dyadic regulation (supported by another) toward self-regulation in the individual. Approaches based upon Stern emphasize both social and emotional development simultaneously and use of the support of adults and peers during a variety of fantasy and imaginary play activities (Hall 2018, p.126).

Rationale or Underlying Theory of Example Programs

Relationship enhancement methods are mostly grounded in the theoretical frameworks related to social learning theory, cognitive development,

and socio-emotional development. Some include techniques developed through applied behavior analysis as a means of encouraging the development of skills required for social interaction. Current published intervention programs exist that promote relationship enhancement and align to these theoretical frameworks. A few are included below; it is noted that all provide a manual or guide to direct means of implementation. The following provides a brief overview of each.

The Developmental Individual-Difference, Relationship-Based model (DIR)/Floortime Approach™, developed by Stanley Greenspan, M.D., reflects Piaget's and Sroufe's frameworks, respectively, with emphasis on sensori-motor activities and linking emotion to the growth of the mind and brain. (Hall 2018, p. 144). The primary focus is relationship-based, with primary caregivers and/or practitioners assisting the individual as they move through the phases of emotional development, capitalizing on these stages of emotion using activities of interest and motivation to the individual.

Relationship Development Intervention, developed by Steven Gutstein and Rachelle Sheely, also reflects Sroufe's framework, with emphasis on social-emotional development through guided participation with the caregiver (Hall 2018, p. 147). The caregiver follows a scope and sequence of socio-emotional development and implements activities designed to model and scaffold the identified set of skills outlined by the authors.

The Denver Model, developed by Sally Rogers, Ph.D. and Diane Osaki, OTR, reflects Stern's framework, with an emphasis on utilizing play for all therapies and activities to enhance social interaction, in a pleasurable manner (Hall 2018, p. 132). At the core of this program is a strong emphasis on social relatedness and social interaction, including parents as key to implementation. Additionally, this program incorporates techniques based on the principles of applied behavior analysis in an effort to further encourage the development of social interaction.

The Social Communication, Emotional Regulation, Transactional Supports (SCERTS) model, developed by Barry Prizant, Ph.D., Amy

Wetherby, Ph.D., Emily Rubin, M.S., Amy Laurent, Ed.M., OTR/L, and Patrick Rydell, Ed. D., reflects developmental theory, learning theory, and family systems theory. The emphasis placed upon emotional regulation relates to the work of Sroufe, with the connection of emotion on mind and brain development (Hall 2018, p. 126). There is a strong emphasis on the inclusion of a multi-disciplinary team to support the development of social interaction and relationships across multiple environments.

The Program for the Evaluation and Enrichment of Relational Skills (PEERS), initially developed by Elizabeth Laugeson and Fred Frankel (2010), reflects developmental theory related to how individuals form and maintain friendships. The emphasis is placed upon development of conversational management as well as key factors that influence relationship enhancement with peers.

Goals and Objectives

The goals and objectives written for these methods of intervention will address social interaction and emotional development, with emphasis upon natural contexts, including specific people within specific environments. A published program may include a specific scope and sequence meant to be followed; however, each emphasizes individuality and the necessity to establish goals and objectives relevant to the individual client. Potential goal areas will be based upon missed developmental or functional milestones and will likely highlight development of joint attention, intimacy, reciprocal communication, expression of emotions/feelings, problem solving, conversational management, and increased flexibility. There typically is an emphasis upon functional communication within natural settings, opposed to rote learning of specific, unrelated language concepts. Emotional regulation and availability of the individual to socially interact are paramount. Therefore, goals to increase the individual's ability to state current levels of regulatory behavior and that promote the ability to engage

in self-advocacy will be needed. Additional goals and objectives should focus upon strengthening social communication and social interaction through play and friendship development (Laugeson et al. 2014; Laugeson and Frankel 2010; Prizant et al. 2006; Gutstein 2000; Rogers et al. 2001).

Treatment Participants

Most individuals receiving programming based upon relationship enhancement methods are age 8 or younger. However, more recently, these approaches are being implemented with adolescents and adults (i.e., Laugeson et al. 2014). The authors of the programs indicated have developed activities for individuals of all ages with severe to mild Autism Spectrum Disorders, Asperger's Syndrome, and other developmental disabilities with varying cognitive ability levels. Family members and peers are integrated as key participants to insure success in generalization (Laugeson and Frankel 2010; Prizant et al. 2006; Rogers et al. 2001; Gutstein 2000; & Koegel and Koegel 1995).

Treatment Procedures

Generally, relationship enhancement methods follow the developmental level of the individual, utilizing activities in one to one and small group settings to increase social reciprocity for the purpose of developing appropriate social interactions, across all people, and all settings. The procedures and activities are driven by the particular program being implemented and based upon the strengths and weaknesses of each individual. The general, primary mode of delivery is through play based or natural contexts, with emphasis upon symbolic and pretend play. The practitioner follows the lead of the child or adult, guiding reciprocal turn-taking, responding to the individuals' initiations, matching the individuals' communication level, and modeling expected socially relevant responses. Scaffolding and embedded learning are key strategies, utilizing visual

supports, particularly for those individuals who have not yet developed adequate language or speech. Key implementers include Special Educators, Speech/Language Pathologists, School Psychologists, Clinical Psychologists, Occupational Therapists, other related disciplines, caregivers, siblings, and same age peers. The reader is referred to each specific method for more in-depth information regarding their various treatment procedures (Laugeson et al. 2014; Laugeson and Frankel 2010; Prizant et al. 2006; Rogers et al. 2001; Gutstein 2000; Koegel and Koegel 1995).

Efficacy Information

Given the complex nature of relationship enhancement methods, it is not surprising that one does not find empirical evidence for this broad category. Instead, it will be necessary for practitioners to conduct a review of literature regarding each published program. Additionally, it will be possible to conduct an analysis of each published program to identify discrete intervention strategies embedded within the programs then determine whether there is empirical evidence to support the discrete procedures that are part of the larger intervention model. Given the limited space for this article and the dearth of information available, the reader is directed to review the work conducted by the National Professional Development Center on Autism, The National Clearinghouse on Autism Evidence & Practice, and the National Standards Project for reviewed articles and currently regarded evidence-based practices for comparison. Additionally, the reader may locate detailed accounts of the listed programs found in this article within the Encyclopedia on Autism, the publisher of this article.

Outcome Measurement

Each method provides guidelines for outcome measurement. Many include case study notes, video diaries, individual progress notes, and completion of specific curricular goals and objectives.

Some include additional supplemental evaluations conducted by the Speech/Language Pathologist, the Occupational Therapist, and/or Clinical or School Psychologist working directly with the individual (Laugeson et al. 2014; Prizant et al. 2006; Rogers et al. 2001; Gutstein 2000; Koegel and Koegel 1995).

Qualifications of Treatment Providers

Each individual relationship enhancement method has its own list of qualifications for treatment providers. Generally, most of these methods support parents, family members, peers, and therapists as possible providers and provide a variety of workshops and trainings to assist treatment providers in implementing their approach. A few of the methods do require additional training to receive certification from their agency, indicating required knowledge and skill to implement the activities and strategies associated with the methodology (Laugeson et al. 2014; Prizant et al. 2006; Rogers et al. 2001; Gutstein 2000; Koegel and Koegel 1995).

See Also

- [DIR Model \(Developmental, Individual Difference, Relationship Based\): A Parent-Mediated Mental Health Approach to Autism Spectrum Disorders, The](#)
- [Early Start Denver Model](#)
- [Pivotal Response Training](#)
- [Relationship Development Intervention \(RDI\) Model](#)

References and Reading

- Bandura, A. (1977). *Social learning theory*. Upper Saddle River: Prentice-Hall.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Princeton: Prentice Hall.
- Gutstein, S., E. (2000). *Autism aspergers: Solving the relationship puzzle, a new developmental program that opens the door to lifelong social & emotional growth*. Future Horizons: Arlington.

- Hall, L. J. (2018). *Autism spectrum disorders: From theory to practice* (3rd ed.). Upper Saddle River: Pearson Education.
- Koegel, R., & Koegel, L. (1995). *Teaching children with autism: Strategies for initiating positive interactions and improving learning opportunities*. Maryland: Paul H. Brookes.
- Laugeson, E., & Frankel, F. (2010). *Social skills for teenagers with developmental and autism spectrum disorders: The PEERS treatment manual*. New York: Routledge.
- Laugeson, E. A., Ellingsen, R., Sanderson, J., Tucci, L., & Bates, S. (2014). The ABC's of teaching social skills to adolescents with autism spectrum disorder in the classroom: The UCLA PEERS^R program. *Journal of Autism and Developmental Disorders*. Retrieved from <https://doi.org/10.1007/s10803-014-2108-8>.
- Marcus, L., Kunc, L., & Schopler, E. (2005). *Working with families*. In F. Volkmar, R. Paul, A. Klin & D. Cohen (eds), *Handbook of autism and pervasive developmental disorders* (pp 1061–1064). Hoboken, NJ: John Wiley & Sons.
- Piaget, J. (1950). *The psychology of intelligence*. London: Broadway House.
- Prizant, B., Wetherby, A., Rubin, E., Laurent, A., & Rydell, P. (2006). *The SCERTS model: A comprehensive educational approach for children with autism spectrum disorders*. Baltimore: Paul H. Brookes.
- Quinn, B., & Malone, A. (2000). *Pervasive developmental disorder: An altered perspective*. London: Jessica Kingsley.
- Rogers, S. J., & DiLalla, D. L. (1991). A comparative study of the effects of a developmentally based instructional model on young children with autism and young children with other disorders of behavior and development. *Topics in Early Childhood Special Education*, 11(2), 29–47.
- Rogers, S. J., & Lewis, H. (1989). An effective day treatment model for young children with pervasive developmental disorders. *American Academy of Child and Adolescent Psychiatry*, 28(2), 207–214.
- Rogers, S. J., Herbison, J. M., Lewis, H. C., Pantone, J., & Reis, K. (1986). An approach for enhancing symbolic, communicative, and interpersonal functioning of young children with autism or severe emotional handicaps. *Journal of the Division for Early Childhood*, 10(2), 135–148.
- Rogers, S. J., Lewis, H. C., & Reis, K. (1987). An effective procedure for training early special education teams to implement a model program. *Journal of the Division of Early Childhood*, 11, 180–188.
- Rogers, S. J., Hall, T., Osaki, D., Reaven, J., & Herbison, J. (2001). The Denver model: A comprehensive, integrated educational approach to young children with autism and their families. In J. S. Handleman & S. L. Harris (Eds.), *Preschool education programs for children with autism* (pp. 95–133). Austin: Pro-Ed.
- Simpson, R. (2005). *Autism spectrum disorders*. California: Corwin Press.
- Volkmar, F., & Wiesner, L. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know*. Hoboken: Wiley.
- Vygotsky, L. (1978). *Mind in society: The development of higher psychological processes*. Cambridge, MA: Harvard University Press.

Relative Weakness in an Area of Functioning

► Disability

Relaxation Techniques

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Definition

Relaxation techniques consist of a variety of strategies that are integrated into behavioral therapies, with the common feature of decreasing physical tension within the affected individual, typically associated with their anxiety or other negative affective experiences (e.g., pain, traumatic memory).

Historical Background

Relaxation techniques have been used for centuries in Asia by physicians and healers in order to aid in the treatment of an assortment of health problems from infants to the elderly. The

technique, although used in western medicine, has not played as a central role in the treatment of major health problems and has generally been used as part of a larger treatment package or considered a type of complementary and alternative medicine (CAM). Relaxation training has been used in the treatment of physical, psychological, and emotional problems, and the methods range from different forms of massage and chiropractic treatments to the use of visualization techniques and virtual reality hypnosis. The most common form of relaxation therapy currently used is progressive muscle relaxation (PMR; Jacobson 1970), which involves alternately tensing and relaxing various muscle groups around the body. PMR originated from the theory that a psychobiological state, neuromuscular hypertension, is the basis for a variety of negative emotional states and psychosomatic diseases (Jacobson 1938). For individuals with autism, preliminary clinical trials have found massage therapy to be associated with decreased autism-specific behaviors such as stereotypies and aversion to certain stimuli as well as increasing on-task behavior and social relatedness in school and decreasing sleep problems at home (Escalona et al. 2001; Field et al. 1997).

Rationale or Underlying Theory

The modern rationale for relaxation therapy is directly related to the notion that people respond to situations perceived to be threatening in three specific modalities – physiologically, behaviorally, and subjectively (or cognitively) – each feeding on the other resulting in a cycle of increased negative reactions. In addition, an important element of psychological distress is an elicitation of a general stress response involving the sympathetic nervous system which is responsible for up- and downregulating numerous homeostatic systems. The resulting activation manifests by enervating the muscular, cardiovascular, and behavioral subsystems to name a few. Relaxation therapy aims to reverse the generalized stress response by deactivating arousal of one of its components, the muscular subsystem. By deactivating this

single subsystem, the patient may be able to reduce activation in other subsystems (Gellhorn and Kiely 1972). In addition, Jacobson (1938) asserted that the relaxation of muscles would lead to relaxation of the mind “because an emotional state fails to exist in the presence of complete relaxation of the peripheral parts involved” (Jacobson 1938, p. 218), an application of the James-Lange theory of emotion (Lang 1994). Therefore, relaxation therapy aims to inhibit the generation of anxious thoughts and emotions and undo the effects of muscular tension on related somatic processes.

Goals and Objectives

Relaxation therapy has been used to treat a variety of disorders ranging from hypertension to stress, insomnia, and headaches. The most prevalent use of these relaxation techniques, in particular muscle relaxation techniques, has been in the treatment of anxiety. The basic therapeutic notion of relaxation therapies is that people experiencing tension, stress, and anxiety can find relief from emotional distress and its physiological accompaniments by learning to reduce muscle tension (Conrad and Roth 2007).

Treatment Participants

Relaxation therapies have been shown to be beneficial to a wide range of individuals (from infants to the elderly) with varying treatment needs such as migraines, behavior problems, sleep problems, and autism-related behaviors, although the level of evidence varies widely across applications (Azrin et al. 1975; Escalona et al. 2001; Reese et al. 1998; Thompson et al. 2010). For example, there is a substantial evidence base for relaxation therapy in the treatment of chronic headache. Headaches are understood to be the result of a complex interaction between biology, environment, behavior, cognition, and emotions (Buse and Andrasik 2009). A muscular component plays a central role in the presence and severity of such headaches, thus providing the rationale for

relaxation therapy. While pharmacological treatments for headaches (and pain in general) are frontline treatment options, some individuals do not wish for, or respond well to, medication. In these cases, relaxation therapy has proved to be a viable alternative (Kroner-Herwig et al. 1998). Anxiety disorders are the most common conditions to be successfully treated with relaxation techniques, particularly muscle relaxation therapies. While there are a limited number of empirical studies that support the efficacy of relaxation therapy when compared with other evidence-based treatments, a number of well-designed randomized controlled trials have been conducted (Ost and Breitholtz 2000; Richter 1984). Relaxation techniques have also been tested for the treatment of specific behaviors in youth with autism spectrum disorders (ASD) (see section “[Efficacy Information](#)” below).

Treatment Procedures

Relaxation techniques typically take place in a clinic-based setting with a mental health professional on an individual basis. Treatment usually begins with an introduction of the rationale to the patient as well as several sessions of “training” on the relaxation process. While the first several sessions are heavily therapist-driven, as treatment progresses, the patient starts to take over the process of implementing the relaxation techniques with practice outside of the therapy room serving as a key component for generalization across settings (Mullins and Christian 2001; Ost 1987; Richter 1984). However, several studies have found that successful implementation of relaxation techniques by paraprofessionals and non-experienced parents can be achieved (Gasalberti 2006; Russell and Wise 1976). Treatment providers are typically seen as coaches in that the goal is to teach the individual strategies in order for patients to eventually recognize when to use relaxation techniques and effectively implement them when needed. Treatment is usually provided during several weeks of sessions with a combination of practicing in session and at home (Ost 1987). The specific number of treatment sessions

associated with successful remittance of problems is undetermined at this time, as studies have ranged from as few as one session a day for two weeks to as long as one time a week for 15 weeks. Relaxation therapies, regardless of which method is used, all focus on the systematic management and reduction of physical tension.

Given the long-standing history of progressive muscle relaxation training to address anxiety in youth, a general outline of the methods is provided (Jacobson 1938; Kendall 1994; Ost 1987). The basic procedures involve the client sitting in a comfortable chair as the therapist instructs them in alternating contracting and releasing of different muscle groups, typically beginning with the large muscle groups (e.g., arms and legs) and progressively moving to the smaller groups (e.g., face and hands). The client learns to recognize the contraction and relaxation in a certain order, simultaneously relaxing all parts that have been previously relaxed. After mastering the relaxation sequence, clients are taught to relax their muscles in real-life situations.

It should be noted that relaxation techniques are often provided as a component of a larger cognitive behavioral therapy treatment program (e.g., Kendall 1994; Wood and McLeod 2008).

Efficacy Information

Some difficulties arise when evaluating the efficacy of relaxation therapies due to the wide range of dependent variables used as well as the imprecise definition of relaxation strategies used in studies. Due to the variety of disorders that are targeted with relaxation therapy, measures vary greatly from study to study. Studies focusing on relaxation to decrease anxiety use measures of anxiety symptoms for their outcome measures, while studies using relaxation to decrease disruptive behavior problems may measure the total number of outbursts over a specific amount of time. In addition, several measures are used to capture the multiple features of a single disorder within a study, thus making it difficult to compare treatment efficacy across studies. However, findings from studies have shown that some relaxation

therapies may be as effective as other behavioral treatments for certain symptoms when they are accompanied by other supportive measures and maintained for an extended period of time (Ost and Breitholtz 2000; Richter 1984). Ost and Breitholtz (2000), in a randomized controlled experiment, compared cognitive therapy and an applied relaxation therapy in the treatment of adults with generalized anxiety disorder. At post-treatment, they found that patients from both groups improved on all independent assessor ratings and on eight of the nine self-report measures with no differences observed in either condition. In addition, a comparable number of participants in each group showed significant clinical improvement. On average, participants were able to maintain these improvements for an average of 13.7 months. Long-term follow-up studies have shown that gains made during treatment are typically maintained for an extended period of time. Other studies also have found that treatment outcomes were maintained for several months after treatment completion with one study of finding that positive results from relaxation therapy were maintained at a follow-up of 19 months (Ost 1987; Reese et al. 1998; Sherman et al. 2010). It should be noted that more evidence has accrued favorable to relaxation therapy for adults with psychological difficulties than with children.

Escalona and colleagues (Escalona et al. 2001) found that children with ASD who received massage therapy, when compared with a control group, exhibited fewer stereotypic behaviors and more on-task and social relatedness behaviors as well as fewer sleep problems posttreatment. Treatments with relaxation components focusing on the reduction of self-injurious and disruptive behaviors have been found to be successful in individuals with ASD as well (Mullins and Christian 2001; Reese et al. 1998). Mullins and Christian examined the effect of a progressive relaxation training program on disruptive behavior problems of a 12-year-old boy with autism. Training consisted of presenting the boy with the steps of the relaxation process along with a training book that provided visual cues while the researchers gave minimal verbal prompts throughout the process. Results from their study indicated that a

decrease in the duration of disruptive behaviors occurred when relaxation training preceded a leisure activity session (compared with no relaxation prior to activity). In addition, the percent of overt relaxed behaviors increased following training and results were acquired through minimal assistance after training. Reese et al. examined the effects of a multifaceted intervention program to reduce disruptive behaviors of individuals with autism and mental retardation living in a community group home. One component of the treatment was a relaxation breathing exercise taught to the participants as well as teaching them how to request a break to relax when exhibiting pre-disruptive behaviors. While the results are somewhat mixed as to the efficacy of the relaxation component, they did find that the participants could relax when prompted (although counselors could not always anticipate when they needed to be prompted) and were able to increase the number of times they requested a break to relax.

Reasons as to why and how relaxation therapy works have been difficult to come by (Conrad and Roth 2007), and research into identifying key elements of the techniques has been urged by researchers in this field (Sherman et al. 2010).

Outcome Measurement

Outcome measures vary with regard to the targeted behavior of the treatment. When treating individuals with anxiety, anxiety measures are typically used pre- and posttreatment in order to determine if the level of anxious symptoms decreased following treatment. Measures may be completed through diagnostic interviews with the patient such as the Anxiety Disorder Interview Schedule: Parent and Child Versions (ADIS; Silverman and Albano 1996) or through self-report measures such as the State-Trait Anxiety Inventory (STAI; Spielberger et al. 1970). During neurofeedback training, individuals are made aware of their brain activity through the use of electroencephalograph (EEG) with the goal being to control central nervous system activity (Hughes and Davis 1980; Thompson et al. 2010). One drawback to the use of relaxation techniques is

the lack of measurement of actual relaxation behaviors, making it difficult to determine if a relaxed state was achieved. In more recent studies, some researchers have attempted to address this limitation by using such measures as the Behavioral Relaxation Scale (Poppen 1988), an indicator of relaxation shown to have a positive correlation between those scores as well as electromyography recordings and self-report measures. Overall, there are a limited number of tools or instruments that are used to measure the level of relaxation obtained by the individual. Measures are typically focused on the behaviors that are the target of the relaxation therapy, which can vary widely due to the use of these techniques on an assortment of conditions.

Qualifications of Treatment Providers

Depending on the type of relaxation therapy used, the qualifications of the treatment providers differ. Most relaxation therapies are conducted by a licensed therapist who is able to help the individual recognize the distressing or uncomfortable feelings that need to be targeted as well as guiding the individual through relaxation techniques. For massage-based relaxation therapies, a licensed massage therapist, physical therapist, and even parents have been trained to use massage to reduce pain and promote relaxation (Gasalberti 2006). However, there have been some relaxation therapies that have been found to reduce problem behaviors, such as anxiety, through means that have yet to be understood and do not require a licensed therapist to provide the treatment (e.g., standard scripts, audiotaped instructions; see Kendall 1994). Researchers are beginning to investigate the mechanisms behind these therapies to understand efficacy and how to best implement them (Conrad and Roth 2007; Sherman et al. 2010).

See Also

- Cognitive Behavioral Therapy (CBT)
- General Anxiety

References and Reading

- Azrin, N. H., Gottlieb, L., Hughart, L., Wesolowski, M. D., & Rahn, T. (1975). Eliminating self-injurious behavior by educative procedures. *Behavior Research and Therapy*, 13, 101–111.
- Buse, D. C., & Andrasik, F. (2009). Behavioral medicine for migraine. *Neurologic Clinics*, 27(2), 445–465.
- Conrad, A., & Roth, W. T. (2007). Muscle relaxation therapy for anxiety disorders: It works but how? *Journal of Anxiety Disorders*, 21, 243–264.
- Escalona, A., Field, T., Singer-Strunck, R., Cullen, C., & Hartshorn, K. (2001). Brief report: Improvements in the behavior of children with autism following massage therapy. *Journal of Autism and Developmental Disorders*, 31(5), 513–516.
- Field, T., Lasko, D., Mundy, P., Henteleff, T., Kabot, S., Talpins, S., et al. (1997). Autistic children's attentiveness and responsivity improve after touch therapy. *Journal of Autism and Developmental Disorders*, 27, 333–338.
- Gasalberti, E. (2006). Alternative therapies for children and youth with special health care needs. *Journal of Pediatric Health Care*, 20, 133–136.
- Gellhorn, E., & Kiely, W. F. (1972). Mystical states of consciousness: Neurophysiological and clinical aspects. *Journal of Nervous and Mental Disease*, 154, 399–405.
- Hughes, H., & Davis, R. (1980). Treatment of aggressive behaviour: The effect of EMG response discrimination biofeedback training. *Journal of Autism and Developmental Disorders*, 10(2), 193–202.
- Jacobson, E. (1938). *Progressive relaxation* (2nd ed.). Oxford, UK: University of Chicago Press.
- Jacobson, E. (1970). *Modern treatment of tense patients*. Oxford, UK: Charles C. Thomas.
- Jacobson, E. (1987). Progressive relaxation. *The American Journal of Psychology*, 100(3/4), 522–537.
- Kendall, P. C. (1994). Treating anxiety disorders in children: Results of a randomized clinical trial. *Journal of Consulting and Clinical Psychology*, 62(1), 100–110.
- Kroner-Herwig, B., Mohn, U., & Pothmann, R. (1998). Comparison of biofeedback and relaxation in the treatment of pediatric headache and the influence of parent involvement on outcome. *Applied Psychophysiology and Biofeedback*, 23(3), 143–157.
- Lang, P. J. (1994). The varieties of emotional experience: A meditation on James-Lange theory. *Psychological Review*, 101(2), 211–221.
- Mullins, J. L., & Christian, L. (2001). The effects of progressive relaxation training on the disruptive behaviour of a boy with autism. *Research in Developmental Disabilities*, 22, 449–462.
- Ost, L. (1987). Applied relaxation: Description of a coping technique and review of controlled studies. *Behaviour Research and Therapy*, 25(5), 397–409.
- Ost, L., & Breitholtz, E. (2000). Applied relaxation vs. cognitive therapy in the treatment of generalized

- anxiety disorder. *Behaviour Research and Therapy*, 38, 777–790.
- Poppen, R. (1988). *Behavioral relaxation training and assessment*. New York: Pergamon Press.
- Reese, R. M., Sherman, J. A., & Sheldon, J. B. (1998). Reducing disruptive behavior of a group-home resident with autism and mental retardation. *Journal of Autism and Developmental Disorders*, 28(2), 159–165.
- Richter, N. C. (1984). The efficacy of relaxation training with children. *Journal of Abnormal Child Psychology*, 12(2), 319–344.
- Russell, R. K., & Wise, F. (1976). Treatment of speech anxiety by cue-controlled relaxation and desensitization with professional and paraprofessional counselors. *Journal of Counseling Psychology*, 23(6), 583–586.
- Sherman, K. J., Ludman, E. J., Cook, A. J., Hawkes, R. J., Roy-Byrne, P. P., Bentley, S., et al. (2010). Effectiveness of the therapeutic massage for generalized anxiety disorder: A randomized controlled trial. *Depression and Anxiety*, 27, 441–450.
- Silverman, W. K., & Albano, A. M. (1996). *The anxiety disorders interview schedule for DSM-IV*. San Antonio: Psychological Corporation.
- Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the STAI*. Palo Alto: Consulting Psychologists Press.
- Thompson, L., Thompson, M., & Reid, A. (2010). Neurofeedback outcomes in clients with Asperger's syndrome. *Applied Psychophysiology and Biofeedback*, 35, 63–81.
- Wood, J. J., & McLeod, B. (2008). *Child anxiety disorders: A treatment manual for practitioners*. New York: Norton.

Repertory Grid Process for Person-Centered Assessment of Social Cognition for Adolescents with Autism Spectrum Disorder

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Definition

A person-centered process for assessment and exploration of the social cognitive personal construct systems of adolescents with ASD with average or above-average intelligence and language skills. The process is modified from the typical flexible format presented in the repertory grid literature (see Fransella 2003; Fransella et al. 2004; Kelly 1955) to provide visual structure and a unique prompting hierarchy to help adolescents with ASD engage in the process.

Remembering to Remember

- [Prospective Memory in Autism Spectrum Disorder](#)

Remembrance

- [Memory](#)

Remeron

- [Mirtazapine](#)

Historical Background

Social pragmatic communication deficits are core to autism spectrum disorder (ASD; American Psychological Association 2013). Social skills groups designed to help adolescents with ASD learn and/or improve their ability to communicate with others in social environments are commonplace; however, the process of evaluating adolescents with ASD to determine their baseline social pragmatic communication skills, inform goals for therapy, and provide therapeutic interventions varies. Many standardized (and a few non-standardized) methods of evaluating social skills of such individuals typically entail gathering observational data of social behaviors an adolescent displays and those behaviors he or she does not display, then ranking those

social skills on Likert-type rating scales (Bellini 2006; Bowers et al. 2007, 2010; Carrow-Woolfolk 1999; Constantino and Gruber 2012; Garcia-Winner 2011; Gilliam and Miller 2006; Gresham and Elliott 2008; Kleiman 2003; Phelps-Terasaki and Phelps-Gunn 2007). Although such approaches can identify social skills demonstrated by the individual, they are limited to a “snapshot” of observable behaviors that arise as a function of the immediate environment and fail to access underlying cognitions and attitudes related to social interactions. Literature from personal construct psychology suggests that these underlying cognitions and attitudes are part of an organized system of personal constructs that can be accessed through a process called the repertory grid assessment (Fransella 2003; Fransella et al. 2004; Kelly 1955). Although repertory grids have been used by clinicians and researchers for many decades, they have only recently been used with individuals with ASD (Hare et al. 1999; Hess et al. 2018).

Current Knowledge

Hare et al. (1999) used repertory grids to explore the social root systems of adults with Asperger’s syndrome living in a group home. The residents demonstrated significant difficulties in their lives, including poor social interfacing with caretakers and with each other and some reportedly demonstrated suicidal ideation. After other attempts to evaluate and improve the social interaction skills and lives of the residents failed, a modified repertory grid process was attempted. Researchers found that, by engaging participants in the repertory grid process with some modifications to scaffold deficits in executive functioning seen in Asperger’s syndrome, the residents were able to engage in the repertory grid process and establish repertory grids. Data from the repertory grids provided researchers and caretakers with first-person insight into the residents’ social cognitive processes that could be used to enhance caretakers’ efficacy of caring for residents.

Hess et al. (2018) explored the potential utility of repertory grids with adolescents with ASD. In a study with five adolescents with ASD with average or above-average language and intelligence skills, Hess, Self, and DiLollo found that by providing visual structure and verbal prompting, these adolescents were able to successfully engage in the repertory grid process. Further, the modified repertory grid process provided a venue for participants to describe, via semi-structured conversational discourse within the structured framework of the process, social interactions with people over the course of their lives that shaped how they conceptualized others in social environments. The grid data delineated how each individual differentiated friend-from-foe and even how they thought of themselves and their hypothetical ideal selves and what behaviors/aspects of their current social selves they would change to become their ideal social selves. Beyond providing researchers with descriptions of the individuals’ social cognitive personal construct systems, the process also provided insight of surprising depth of each individual’s social cognitive awareness that helped researchers better understand why certain social behaviors were or were not observed, further distinguishing the repertory grid assessment from the standard approach.

Future Directions

The use of repertory grids with adolescents with ASD with average or above-average intelligence continues to be explored. Further research is needed to determine the utility of repertory grids in a variety of settings (e.g., public schools, group homes, family homes). It will be essential to determine how clinicians can best use the data collected during the repertory grid process to design effective therapeutic interventions for individuals who are receiving therapy services and to potentially advocate for treatment for those adolescents with ASD who are not receiving services.

See Also

- [Pragmatic Language](#)
- [Repertory Grid Process for Person-Centered Assessment of Social Cognition for Adolescents with Autism Spectrum Disorder](#)
- [Social Cognition](#)

References and Reading

- American Psychological Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Press.
- Bellini, S. (2006). *Building social relationships: A systematic approach to teaching social interaction skills to children and adolescents with autism spectrum disorders and other social difficulties*. Shawnee Mission: Autism Asperger Publishing.
- Bowers, L., Huisinigh, R., & LoGiudice, C. (2007). *Test of problem solving: Adolescent*. Austin, TX: PRO-ED.
- Bowers, L., Huisinigh, R., & LoGiudice, C. (2010). *Social language development test: Adolescent*. Austin, TX: PRO-ED.
- Carrow-Woolfolk, E. (1999). *Comprehensive assessment of spoken language*. Austin: PRO-ED.
- Constantino, J. N., & Gruber, C. F. (2012). *Social responsiveness scale* (2nd ed.). Torrance: WPS.
- Fransella, F. (2003). *International handbook of personal construct psychology*. West Sussex: Wiley.
- Fransella, F., Bell, R., & Bannister, D. (2004). *A manual for repertory grid technique* (2nd edn.). West Sussex: Wiley.
- Garcia-Winner, M. (2011). Social thinking-social communication profile. <http://www.socialthinking.com/Articles?name=Social%20Thinking%20Social%20Communication%20Profile>. Retrieved 25 Aug 2014.
- Gilliam, J. E., & Miller, L. (2006). *Pragmatic language skills inventory*. Austin: PRO-ED.
- Gresham, F. M., & Elliott, S. N. (2008). *Social skills improvement system (SSIS)*. Minneapolis: Pearson.
- Hare, D. J., Jones, J. P. R., & Paine, C. (1999). Approaching reality: The use of personal construct assessment in working with people with Asperger syndrome. *Autism*, 3(2), 165–176.
- Hess, S., Self, T., & DiLollo, A. (2018). Assessing personal constructs of adolescents with autism spectrum disorder: A person-centered measure of social cognition. *Journal of Autism and Developmental Disorders*, 48(2), 485–501.
- Kelly, G. A. (1955). *The psychology of personal constructs: Volume one: A theory of personality*. New York: W. W. Norton & Company.
- Kleiman, L. I. (2003). *Functional communication profile revised*. Austin: PRO-ED.
- Phelps-Terasaki, D., & Phelps-Gunn, T. (2007). *Test of pragmatic language*. Austin: PRO-ED.

Repetitive Behavior

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Definition

The term “repetitive behaviors” refers to abnormal behaviors that are characterized by repetition, rigidity, inappropriateness, and lack of adaptability (Bodfish 2007). They include motor stereotyped behaviors, self-stimulatory behaviors, self-injurious behaviors, compulsive or sameness behaviors, and verbal repetitive behaviors such as echolalia (Bodfish et al. 2000). Repetitive behaviors may become problematic when they occupy a significant portion of the individual’s waking hours, interfere with participation in other life activities, interfere with appropriate social interactions, disrupt the learning of more appropriate behaviors, or in the case of self-injurious behavior cause significant bodily damage. The specific function of repetitive behaviors in autism is unknown, but hypothesized functions include stress reduction, reward/gratification, and sensory stimulation (Mason 1991). Individuals with autism who engage in repetitive and restricted behaviors often experience anxiety, acute distress, or agitation if the behaviors are interrupted (Lam and Aman 2007).

Restricted and repetitive behaviors and interests are not exclusive to autism spectrum disorders and are often found in individuals with other developmental disorders such as mental retardation/intellectual disability or developmental delays, certain psychiatric disorders including schizophrenia and obsessive-compulsive disorder, several genetic syndromes, and medical conditions such as Parkinson’s disease and Tourette’s disorder (Bodfish et al. 2000; Moss et al. 2009; Richler et al. 2007) and in individuals with sensory disorders such as the visually impaired (Tröster et al. 1991). Repetitive behaviors are

frequently reported by mothers of typically developing children as well, particularly in early childhood (Leekam et al. 2007). However, there appear to be qualitative and quantitative differences between repetitive behaviors of children with autism and those who develop typically (Arnott et al. 2010; Richler et al. 2007), in the developmental pattern of such behaviors over time (Richler et al. 2010) and in the prevalence of repetitive behaviors even compared to children with intellectual disability (Bodfish et al. 1995).

The presence of restricted, repetitive, and stereotyped patterns of behavior, interests, and activities is one of the three core symptom clusters used to diagnosis autistic disorder according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revisions (American Psychiatric Association 2000). Within this domain, the DSM-IV-TR defines repetitive behaviors specifically as “(a) encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus; (b) apparently inflexible adherence to specific, non-functional routines or rituals; (c) stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole-body movements; (d) persistent preoccupation with parts of objects” (p. 75). The proposed DSM-V (American Psychiatric Association 2010) includes restricted or fixated behavior, interests, and activities as a required symptom for a diagnosis of autism spectrum disorder.

The DSM-IV-TR includes the diagnosis of stereotypic movement disorder that is defined as “repetitive, seemingly driven, and non-functional motor behavior (e.g., hand shaking or waving, body rocking, head banging, mouthing of objects, self-biting, hitting own body (p. 134).” The diagnosis includes a specifier of “with self-injurious behavior” if the repetitive behavior results in bodily damage that requires specific treatment or would require treatment if protective measures were not used. The diagnosis of stereotypic movement disorder also requires that the repetitive behavior is not a symptom of a pervasive developmental disorder such as autistic disorder, not a behavioral tic, not a compulsive behavior that is part of an

obsessive-compulsive disorder, and not trichotillomania or hair pulling that is considered an impulse-control disorder not elsewhere classified. The need to exclude or rule out other diagnoses that could better account for the observed repetitive or stereotyped behaviors illustrates the lack of specificity of repetitive behaviors to any one disorder.

The ICD-10 (International Classification of Diseases-Tenth Revision) is the current version of the international standard diagnostic classification for all general epidemiological purposes, for health management purposes, and for diagnostic clinical use. Childhood autism is included in the ICD-10 and is diagnosed on the basis of similar symptom clusters to the DSM-IV, one of which is the presence of restricted, stereotyped, and repetitive behavior (World Health Organization 2007). The ICD-10 also includes a diagnostic category called stereotyped movement disorders. These disorders are characterized by voluntary, repetitive, stereotyped, and nonfunctional movements (often rhythmic) that are not part of a diagnosed psychiatric or neurologic condition. Similar to the DSM-IV-TR, stereotyped movement disorders can be self-injurious or non-self-injurious.

A review of the research on the phenomenology of repetitive behaviors in autism provided useful operationalized definitions of subtypes of repetitive behavior (Lewis and Bodfish 1998). The authors were careful to try to distinguish among the different subtypes toward the goal of better defining and thus more accurately assessing for the presence of repetitive behaviors in autism (see Table 1). Restricted behavior and ritualistic behavior were later added as specific conceptual subtypes, and Akathisia and dyskinesia have since been removed as subtypes.

A five-conceptual groupings model of restricted repetitive behaviors proposed by Bodfish and colleagues that are assessed with the *Repetitive Behavior Scale-Revised* (RBS-R) (Bodfish et al. 2000) has received empirical support (Lam and Aman 2007; Bodfish et al. 2000).

The five factors that emerged in factor analysis are (1) *rituals/sameness*, (2) *self-injurious behavior*, (3) *stereotypic behavior*, (4) *compulsive behavior*, and (5) *restricted interests* (Lam and Aman 2007). The RBS-R has six subscales

Repetitive Behavior, Table 1 Subtypes of repetitive behaviors

Classification	Definition
Akathisia	Movement disorder that can manifest as repetitive and restless movements such as pacing or a hyperkinetic restless state such as the inability to remain still when sitting or standing
Compulsions	Repetitive intentional behaviors that appear to follow certain rules
Dyskinesia	Repetitive and involuntary movements that can be subdivided into three types: nonrhythmic and jerky movements (chorea) or slow, writhing movements (athetosis), or slow and sustaining muscle tensing (dystonia)
Echolalia	Repetitive use of speech immediately, such as repeating all or part of what was just heard or after a delay. Echolalia can be “parrot speech” defined as repeating whatever is heard and “perseverative speech” in which a small number of words or phrases is repeated in a ritualistic or persistent manner
Obsessions	Repetitive, persistent thoughts, impulses, or images that are experienced as intrusive or inappropriate and that caused marked anxiety or distress in the individual
Perseveration	Behavioral responses in which nonaberrant adaptive responses are repeated beyond what is necessary to reach a goal. This can include the perseveration of attention
Restricted behavior	Behavior with a limited range of focus, interest, or activity
Ritualistic behavior	Performing activities of daily living in a similar manner
Sameness	Involves overall repetitive routines and preferences rather than the discrete repeated acts or thoughts that define compulsions or obsessions
Self-injury	Repetitive motor movements that result in bodily injury or have the potential to result in bodily injury to the individual engaging in the behavior
Stereotypy	Repetitive and seemingly purposeless body movements, movements of body parts, or use of the body to generate movement in objects
Tics	Often considered involuntary movements, tics are repetitive behaviors that can be simple or complex and that are executed repeatedly in an “explosive” manner and out of context

because it assesses ritualistic behavior and sameness behavior separately although they load as a common factor.

Repetitive behaviors have also been subdivided into two conceptual categories referred to as “lower order” and “higher order” (Bodfish 2007). Lower order repetitive behaviors are repetitive motor movements such as repetitive manipulation of an object, body rocking, and hand flapping or a repetitive self-injurious behavior such as head banging. Higher order repetitive behaviors are more complex and are cognitive behaviors such as circumscribed/restricted interests, insistence on sameness, or compulsions.

An alternative two-factor categorization of repetitive behaviors has also been proposed for young children and has received research support (Richler et al. 2007). The two factors are described as a *repetitive sensory motor* factor (RSM) and an *insistence on sameness* factor (IS). The RSM factor includes behaviors such as hand/finger movements, complex body mannerisms, repetitive use of objects, and unusual sensory interests. The IS factor includes behaviors such as rituals and compulsions and resistance to or distress with change. It is suggested that RSM behaviors are more common in very young children than IS behaviors.

Historical Background

Repetitive behaviors as a core feature of autism were recognized as early as 1943 when Dr. Leo Kanner first described case studies of 11 children that he conceptualized as having an “autistic disturbance of affective contact” (Kanner 1943). These children had a “dread of change and incompleteness” (p. 246) that led to monotonous and repetitive behaviors and limitations in the range of spontaneous activity. Rituals had to be carried out from beginning to end without variation. Kanner also described the anxiety and distress that the children seemed to experience whenever the sameness of a chain of behaviors, including verbal behaviors, was broken. Kanner hypothesized that these repetitive behaviors and the insistence on

sameness were a part of the child's "good relation to objects" (p. 246) as opposed to their lack of interest in and poor relations to people.

In 1944, Dr. Hans Asperger, for whom Asperger's disorder is named, described four boys ages 6–11 years who had good language and cognitive skills, yet marked impairment in social interaction as having "autistic psychopathy" (Klin et al. 2000) or more accurately translated as "autistic personality disorder" (Volkmar and Klin 2000). Asperger also described that these four and a larger sample of children also had pre-occupations and unusual circumscribed interests that were the focus of much of their life and that interfered with the acquisition of necessary skills (Volkmar and Klin 2000). Asperger also noted that similar traits were often seen in other family members, particularly fathers.

Current Knowledge

Epidemiology of Repetitive Behaviors

Because the presence of restricted or circumscribed interests or repetitive or stereotyped behaviors is part of the diagnostic criteria for autism, it is understood that the prevalence of repetitive behaviors of some kind is 100% in this population which is generally supported by research (Bodfish et al. 2000; Campbell et al. 1990; Shay et al. 1993).

Research suggests the possibility of age-related differences in the patterns of particular restrictive repetitive behaviors (RRBs) in individuals with autism spectrum disorders, a possible relationship between level of intellectual functioning and patterns of RRBs, and possible interactions between age and level of functioning. However, research findings are inconsistent and seem to depend upon the age range of the sample, the specific subtypes of repetitive behaviors, and the instrument used to measure the behaviors.

Age. In general, RRBs seem to be less severe and less frequent in older compared to younger children with autism. Similarly, adults with autism display fewer repetitive behaviors than children, suggesting that RRBs may decrease over the

lifespan (Esbensen et al. 2009). Younger children with ASD may be more likely to exhibit motor and sensory repetitive behaviors, whereas older children may be more likely to exhibit more complex repetitive behaviors (Militeri et al. 2002). Among specific subclasses of RRBs, stereotyped movements and restricted range of interests appear to be less frequent among older individuals, whereas rituals/sameness appears to be more frequent in this group. Self-injurious behaviors and compulsive behavior subtypes do not show differences across age groups (Lam and Aman 2007).

Level of functioning. Research findings have also suggested that the expression of RRBs may be influenced by level of functioning (Bishop et al. 2006; Gabriels et al. 2005; Militeri et al. 2002). As described above, repetitive behaviors have been distinguished into two subcategories. The first is lower order behaviors that are characterized by repetition of body or part of body movement such as stereotyped movements, repetitive manipulations of objects, and self-injurious behavior. And the second, higher order repetitive behaviors that are more complex and are more "cognitive" such as object attachments, sameness, and circumscribed interests (Bodfish 2007). Evidence has been offered that lower functioning individuals such as those with a more severe or profound intellectual disability are more likely to engage in lower order repetitive behaviors, whereas higher functioning individuals are more likely to engage in higher order repetitive behaviors that are more cognitive in nature. However, lower order and higher order repetitive behaviors have been found to co-occur (Bodfish 2007), and the distinction between higher and lower order behaviors does not hold consistently.

In lower functioning individuals, suggested age-related differences in patterns of RRBs may be less pronounced. A comorbid diagnosis of intellectual disability is associated with demonstrating more repetitive behaviors than a diagnosis of autism alone as is engaging in more atypical behaviors (Dominick et al. 2007; Esbensen et al. 2009). For the majority of RRBs, intellectual ability seems more strongly negatively correlated to

the prevalence of RRBs in older compared to younger children. Other forms of RRB such as insistence on sameness or the need for routines do not appear to be related to level of intellectual functioning (Militeri et al. 2002).

Assessment Instruments for Repetitive Behavior

While multiple assessment instruments contain items or subscales that assess for the presence of specific repetitive behaviors, a few instruments have been developed specifically for this purpose. Some of them are briefly described here.

The *Repetitive Behavior Scale-Revised* (RBS-R) is a 43-item questionnaire that was designed to assess for the breadth and severity of restricted and repetitive behaviors in autism using a 0–3 point Likert scale (Bodfish et al. 1999). The empirically validated measure includes six subscales that measure six different dimensions or conceptual groupings of restrictive and repetitive behavior. These are (a) stereotyped behavior, (b) self-injurious behavior, (c) compulsive behavior, (d) ritualistic behavior, (e) sameness behavior, and (f) restricted behavior (Bodfish et al. (2000). The current scoring for the RBS-R recommends using five empirically derived subscales based on 38 of the 43 items (Lam and Aman 2007).

The *Repetitive Behavior Questionnaire-2* (RBQ-2) is a 20-item parent questionnaire based on a four-factor model of repetitive behaviors that includes unusual sensory interests, repetitive motor movements, rigidity/adherence to routine, and preoccupation with restricted patterns of interest (Arnott et al. 2010). There is also support that items can be grouped according to two factors with motor and sensory subscales combining and rigidity and insistence on sameness subscales combining. This measure has been used to investigate the frequency of autistic-like repetitive behaviors in typically developing children (Arnott et al. 2010; Leekam et al. 2007).

The *Behavior Flexibility Rating Scale-Revised* describes 16 situations that can be rated by a parent or caregiver of a child with developmental disabilities as problematic for an individual on a

scale of 0–2. Factor analysis suggests that three factors are assessed by the scale: flexibility toward objects, flexibility toward the environment, and flexibility toward persons (Peters-Scheffer et al. 2008).

The *Behavior Problems Inventory* (BPI-01), developed by Rojahn and colleagues (Rojahn et al. 2001), is a 52-item behavior rating instrument that assesses for self-injurious, aggressive/destructive, and stereotypic behavior in individuals with intellectual and other developmental disabilities. Behaviors are rated for both frequency and severity, and information is provided by care providers. The stereotyped behavior subscale contains 25 items.

The *Autism Diagnostic Interview-Revised* (ADI-R) has been used in research as well as for assessment, diagnosis, treatment, and educational planning for individuals with an autism spectrum disorder. The ADI-R is administered as a clinical interview and can be used with children and adults. The scale consists of 93 items that focus on three domains of functioning, one of which is restricted, repetitive, and stereotyped behaviors and interests (Lord et al. 1994).

Treatment

Repetitive behaviors are currently conceptualized as a complex interaction between biology and environment so that treatment models are generally integrated and can be a combination of medication and behavioral treatments or environmental manipulations (Bodfish 2007). Three general classes of medications have received some empirical support for the treatment of repetitive or stereotyped behaviors, medications that affect the neurotransmitters dopamine such as typical and atypical antipsychotics, those that affect the neurotransmitter serotonin such as the serotonin reuptake inhibitors or SSRIs, and those that affect the opiate system, most specifically naltrexone (Bodfish 2007).

Applied behavior analysis (ABA) is the most widely supported and scientifically validated treatment for core symptoms of autism spectrum disorders, including repetitive behaviors. ABA is the application of principles of learning and

motivation to obtain changes in behavior (Myers et al. 2007). It includes functional assessment, a systematic way of understanding the purpose or function of a behavior and its contingencies of reinforcement, as well as highly structured reinforcement-based intervention methods. Behavioral intervention can focus on increasing the frequency, intensity, or range of adaptive and prosocial behaviors; decreasing the frequency, intensity, and range of repetitive, stereotyped, or self-injurious behaviors; teaching new skills; and generalizing skills across situations. Reinforcement-based techniques are generally used in behavioral treatment as are changes to the environment to remove cues that trigger repetitive behaviors or to provide environmental cues that the behavior is appropriate (Bodfish 2007; Brusa and Richman 2008).

Future Directions

While the presence of repetitive or stereotyped behaviors or restricted range of interests will likely remain a diagnostic criteria for autism in the DSM-V (American Psychiatric Association 2010), it is not likely that the presence or absence of RRBs will become a key diagnostic indicator since most RRBs can be found in other subgroups of children and even in typically developing children. It is more likely that the frequency and intensity of RRBs, the degree of distress caused when the behavior is interrupted, and the amount of dysfunction or disruption to learning that occurs as a result of the RRBs will continue to be what distinguishes children with ASDs from typically developing children or from children with other disorders. What is clear is that RRBs are an underresearched core symptom of autism spectrum disorders and can have a significant detrimental effect on learning more adaptive behaviors and result in physical injury. Additional research on the specificity of RRBs in autism spectrum disorders, how specific repetitive behaviors may change over the lifespan, and how specific individual, demographic, or environmental factors might increase the incidence of RRBs in this population is needed.

See Also

- Echolalia
- Self-Injurious Behavior
- Stereotyped Movement Disorder
- Stereotypic Behavior

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed.). Washington, DC: APA Press. Text Rev.
- American Psychiatric Association. (2010). *Proposed draft revisions to DSM disorders and criteria*, APA DSM-5. Retrieved 10 Sept 2010 from <http://www.dsm5.org/ProposedRevisions/Pages/Default.aspx>
- Amott, B., McConachie, H., Meins, E., Fernyhough, C., LeCouteur, A., Turner, M., et al. (2010). The frequency of restricted and repetitive behaviors in a community sample of 15-month-old infants. *Journal of Developmental and Behavioral Pediatrics*, 31, 223–229.
- Bishop, S. L., Richler, J., & Lord, C. (2006). Association between restricted and repetitive behaviors and nonverbal IQ in children with autism spectrum disorders. *Child Neuropsychology*, 12, 247–267.
- Bodfish, J. W. (2007). Stereotypy, self-injury, and related abnormal repetitive behaviors. In J. W. Jacobson, J. A. Mulick, & J. Rojahn (Eds.), *Handbook of intellectual and developmental disabilities* (pp. 481–505). New York: Springer.
- Bodfish, J. W., Crawford, T. W., Powell, S. B., Golden, R. N., & Lewis, M. H. (1995). Compulsions in adults with mental retardation: Prevalence, phenomenology, and co-morbidity with stereotypy and self-injury. *American Journal on Mental Retardation*, 100, 183–192.
- Bodfish, J. W., Symons, F. J., & Lewis, M. H. (1999). The repetitive behavior scales. Western Carolina Center Research Reports.
- Bodfish, J. W., Symons, F. J., Parker, D. E., & Lewis, M. H. (2000). Varieties of repetitive behavior in autism: Comparisons to mental retardation. *Journal of Autism and Developmental Disorders*, 30, 237–243.
- Brusa, E., & Richman, D. (2008). Developing stimulus control for occurrences of stereotypy exhibited by a child with autism. *The International Journal of Behavioral Consultation and Therapy*, 4, 264–269.
- Campbell, M., Locascio, J., Choroco, M. C., Spencer, E. K., Malone, R. P., Kafantaris, V., et al. (1990). Stereotypies and tardive dyskinesia: Abnormal movements in autistic children. *Psychopharmacology Bulletin*, 26, 260–266.
- Dominick, K. C., Davis, N. O., Lainhart, J., Tager-Flusberg, H., & Folstein, S. (2007). Atypical behaviors in children with autism and children with a history of

- language impairment. *Research in Developmental Disabilities*, 28, 145–162.
- Esbensen, A. J., Mailick Seltzer, M., Lam, K. S. L., & Bodfish, J. W. (2009). Age-related differences in restricted repetitive behaviors in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 57–66.
- Gabriels, R. L., Cuccaro, M. L., Hill, D. E., Ivers, B. J., & Goldson, E. (2005). Repetitive behaviors in autism: Relationships with associated clinical features. *Research in Developmental Disabilities*, 26, 169–181.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nerv Child*, 2, 217–250.
- Klin, A., Volkmar, F. R., & Sparrow, S. S. (2000). Introduction. In A. Klin, F. R. Volkmar, & S. S. Sparrow (Eds.), *Asperger syndrome* (pp. 1–21). New York: The Guilford Press.
- Lam, K. S. L., & Aman, M. G. (2007). The repetitive behavior scale-revised: Independent validation in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 855–866.
- Leekam, S., Tandos, J., McConachie, H., Meins, E., Parkinson, K., Wright, C., et al. (2007). Repetitive behaviors in typically developing 2-year-olds. *Journal of Child Psychology and Psychiatry*, 48, 1131–1138.
- Lewis, M. H., & Bodfish, J. W. (1998). Repetitive behavior disorders in autism. *Mental Retardation and Developmental Disabilities Research Reviews*, 4, 80–89.
- Lord, C., Rutter, M., & Le Couteur, A. S. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24, 659–685.
- Mason, G. (1991). Stereotypies: A critical review. *Animal Behavior*, 41, 1015–1037.
- Militermi, R., Bravaccio, C., Falco, C., Fico, C., & Palermo, M. T. (2002). Repetitive behaviors in autistic disorder. *European Child and Adolescent Psychiatry*, 11, 210–218.
- Moss, J., Oliver, C., Arron, K., Burbidge, C., & Berg, K. (2009). The prevalence and phenomenology of repetitive behavior in genetic syndromes. *Journal of Autism and Developmental Disorders*, 39, 572–588.
- Myers, S. M., Johnson, C. P., & the Council on Children with Disabilities. (2007). Management of children with autism spectrum disorders. *Pediatrics*, 120, 1162–1182.
- Peters-Scheffer, N., Didden, R., Green, V. A., Sigafoos, J., Kozilius, H., Pituch, K., et al. (2008). The behavior flexibility rating scale-revised (BFRS-R): Factor analysis, internal consistency, inter-rater and intra-rater reliability, and convergent validity. *Research in Developmental Disabilities: A Multidisciplinary Journal*, 29, 398–407.
- Richler, J., Bishop, S. L., Kleinke, J. R., & Lord, C. (2007). Restricted and repetitive behaviors in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 73–85.
- Richler, J., Huerta, M., Bishop, S. L., & Lord, C. (2010). Developmental trajectories of restricted and repetitive behaviors and interests in children with autism spectrum disorders. *Development and Psychopathology*, 22, 55–69.
- Rojahn, J., Matson, J. L., Lott, D., Esbensen, A. J., & Smalls, Y. (2001). The behavior problems inventory: An instrument for the assessment of self-injury, stereotyped behavior, and aggression/destruction in individuals with developmental disabilities. *Journal of Autism and Developmental Disorders*, 31(6), 577–588.
- Shay, J., Sanchez, L., Cueva, J., Armenteros, J., Overall, J., & Campbell, M. (1993). Neuroleptic-related dyskinesia and stereotypies in autistic children. *Psychopharmacology Bulletin*, 29, 359–363.
- Tröster, H., Brambring, M., & Beelmann, A. (1991). Prevalence and situational causes of stereotyped behaviors in blind infants and preschoolers. *Journal of Abnormal Child Psychology*, 19, 569–590.
- Volkmar, F. R., & Klin, A. (2000). Diagnostic issues in Asperger syndrome. In A. Klin, F. R. Volkmar, & S. S. Sparrow (Eds.), *Asperger syndrome* (pp. 25–71). New York: The Guilford Press.
- World Health Organization. (2007). *International statistical classification of diseases and related health problems 10th revision*. Geneva: Author.

Repetitive Behavior

- [Stereotyped Movement Disorder](#)
- [Stereotypic Behavior](#)

Repetitive Movements

- [Stereotypic Behavior](#)

Replacement Behavior

- [Functionally Equivalent Alternative Behavior](#)

Report Form

- [Standardized Behavior Checklists](#)

Report of Annual Yearly Academic Progress

► [Annual Review](#)

Representation

► [Modeling](#)

Request

► [Mands](#)

Research Domain Criteria and ASD

► [RDoC and Autism](#)

Residual Autism

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Synonyms

[Residual infantile autism](#)

Definition

A concept introduced by, and limited to, the third edition of the *Diagnostic and Statistical Manual* (DSM-III) in 1980 to encompass individuals who once met criteria for infantile autism but no longer did so. The inclusion of this term reflected the lack

of developmental orientation in the first official definition of infantile autism in DSM-III and was meant to address the issue of developmental change. This term quickly proved unsatisfactory in many ways since the problems of most individuals with autism (or what now is termed autistic disorder) were often quite significant even if different than those they first exhibited as young children. In the following (1987) edition of DSM, the definition of autism was considerably changed to reflect a broader developmental orientation.

In some ways the concept might now seem more applicable to the small but growing number of individuals who no longer meet strict criteria for autistic disorder/autism spectrum disorder – a group that has also been termed “optimal outcome” (Fein et al. 2013). Such individuals often continue to exhibit subtle, or not so subtle, problems in social communication and learning (Suh et al. 2014; Troyb et al. 2014; Tyson et al. 2014).

See Also

- [Autism Spectrum Disorder](#)
- [DSM-III](#)
- [DSM-III-R](#)
- [DSM-IV](#)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Fein, D., Barton, M., Eigsti, I.-M., Kelley, E., Naigles, L., Schultz, R. T., Stevens, M., Helt, M., Orinstein, A., Rosenthal, M., Troyb, E., & Tyson, K. (2013). Optimal outcome in individuals with a history of autism. *Journal of Child Psychology and Psychiatry*, 54(2), 195–205.
- Suh, J., Eigsti, I.-M., Naigles, L., Barton, M., Kelley, E., & Fein, D. (2014). Narrative performance of optimal outcome children and adolescents with a history of an Autism Spectrum Disorder (ASD). *Journal of Autism and Developmental Disorders*, 44(7), 1681–1694.
- Troyb, E., Orinstein, A., Tyson, K., Helt, M., Eigsti, I.-M., Stevens, M., & Fein, D. (2014). Academic abilities in children and adolescents with a history of autism

spectrum disorders who have achieved optimal outcomes. *Autism*, 18(3), 233–243.

- Tyson, K., Kelley, E., Fein, D., Orinstein, A., Troyb, E., Barton, M., Eigsti, I.-M., Naigles, L., Schultz, R. T., Stevens, M., Helt, M., & Rosenthal, M. (2014). Language and verbal memory in individuals with a history of autism spectrum disorders who have achieved optimal outcomes. *Journal of Autism and Developmental Disorders*, 44(3), 648–663.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 5–41). Hoboken: Wiley.

Residual Infantile Autism

► Residual Autism

Resilience in Parents of Children with Autism Spectrum Disorder

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Definition

Resilience is defined as a “positive adaptation within the context of significant adversity” (Luthar et al. 2000, p. 545). Rutter (1987) described resilience as a response to risk where “some people succumb to stress and adversity whereas others overcome life hazards” (p. 317). Resilience also refers to “the ability to withstand hardship and rebound from adversity, becoming more strengthened and resourceful” (Walsh 1998, 2006, p. 263). These definitions share two similarities: (1) the presence of some type of adversity or risk factor and (2) positive outcomes after exposure to risk or adversity. Moreover, inherent in each definition is the conceptualization of resilience as a *process* as opposed to a personality characteristic that an individual has (Luthar et al. 2000).

Given the multidimensional nature of the construct of resilience, it is also important to define the various components of resilience. *Risk or adversity* refers to “negative life circumstances that are known to be statistically associated with adjustment difficulties” (Luthar and Cicchetti 2000, p. 858). In the context of parents raising a child with ASD, possible risk factors may be the severity of the child’s ASD symptoms, presence of comorbid behavioral or mental health problems, the total number of children with ASD, or the quality of the marital relationship. The outcome associated with process of resilience is that of *positive adaptation*, which is defined as “behaviourally manifested social competence or success at meeting stage-salient developmental tasks” (Luthar and Cicchetti 2000, p. 858). It is also possible that the reduction or absence of negative outcomes may be another outcome of resilience (Fergus and Zimmerman 2005). Possible indicators of positive adaptation include high levels of life satisfaction, psychological well-being, and positive affect. Similarly, lower levels of depressive symptoms, anxiety symptoms, and negative affect may also be considered evidence of positive adaptation.

A key component in the process of resilience is the presence of *protective factors*. A protective factor is generally considered to be an attribute that promotes positive adaptation. For parents of children with ASD, this includes, but is not limited to, factors such as social support, personality traits, cognitive appraisals, and religiosity/spirituality. Protective factors operate in a variety of ways to enhance positive adaptation. In the *compensatory* model of resilience, the protective factor has a direct effect on the outcome of interest (Fergus and Zimmerman 2005). For example, parents of children with ASD may report higher levels of stress compared to parents of neurotypical children, but the presence of a protective factor (e.g., spirituality, social support, etc.) may help compensate for the negative effects typically associated with raising a child with ASD. This model of resilience is typically tested using multiple regression models.

In the *protective* model of resilience, the protective factors are interactive effects wherein

individuals who possess the attribute, as compared to those without the attribute, are less affected by the risk or adversity (Luthar et al. 2000). In the context of ASD, parents of children with high levels of behavior problems and greater social support may report higher levels of life satisfaction as compared to parents with low levels of social support. There are two additional protective models: (1) *protective-stabilizing* and (2) *protective-reactive*. In a protective-stabilizing model, individuals with the protective attribute display consistent levels of positive adaptation despite adversity. The protective-reactive model, in contrast, states that when adversity increases, individuals with the protective attribute will show increasing levels of positive adaptation (Luthar et al. 2000). The final set of resilience models are referred to as *vulnerability* models (Luthar et al. 2000). A vulnerability factor is one in which individuals with the attribute show higher levels of negative outcomes when faced with adversity. Parents of children with more severe ASD symptoms may report higher levels of depressive symptoms when they also report higher levels of pessimism.

Historical Background

The construct of resilience has a long history in the study of children and adolescents; however, in the context of parenting children with autism spectrum disorder (ASD), the body of research is smaller. Raising a child with autism spectrum disorder (ASD) presents unique challenges and may negatively impact parental well-being (e.g., Davis and Carter 2008). Interviews with parents of children with ASD describe being dissatisfied with the diagnostic process (e.g., Goin-Kochel et al. 2006), having to go through a period of grief after the diagnosis (Fernández-Alcántara et al. 2016), and then continuing to experience mental health problems throughout their child's life (Benson 2014). The financial burden associated with caring for a child with ASD is also substantial (Buescher et al. 2014). In general, mothers of children with ASD report lower levels of psychological well-being compared to mothers

of children with other developmental disabilities (Abbeduto et al. 2004). Thus, for a long period of time, studies of parents of children with ASD were primarily focused on examining negative outcomes, such as parenting stress and mental health problems. For example, Ekas and Whitman (2010) found that higher levels of child ASD symptom severity were associated with poorer psychological well-being in mothers of children with ASD. Davis and Carter (2008) found that children's difficulties in the domain of social relatedness were associated with increased parenting stress. Children with ASD often display comorbid behavior problems (e.g., aggression), and the presence of these behaviors consistently predicts negative outcomes in parents (e.g., Davis and Carter 2008).

Characteristics of the parents may also exacerbate negative outcomes. For example, the broad autism phenotype (BAP) is a constellation of subclinical characteristics that indicates genetic liability to ASD (Wheelwright et al. 2010). Individuals with high levels of the BAP typically have social abnormalities, a rigid personality, and pragmatic language difficulties (Hurley et al. 2007; Sasson et al. 2013). Among mothers of children with ASD, BAP symptoms are associated with elevated depressive symptoms (Ingersoll and Hambrick 2011). Pruitt et al. (2018) found that each cluster of BAP symptoms was associated with increased depressive symptoms.

The quality of parents' social relationships may also be negatively impacted when raising a child with ASD. Parents of children with ASD reported less marital satisfaction (Brobst et al. 2009), lower marital quality (Saini et al. 2015; Sim et al. 2017), and a higher divorce rates (Hartley et al. 2010) compared to parents of neurotypical (NT) children. In addition, parents of children with ASD have more severe conflict (Hartley et al. 2017b) and fewer positive couple interactions when compared to parents of NT children (Hartley et al. 2017a).

Although the aforementioned body of research suggests that raising a child with ASD is challenging, there is considerable individual variability, with some parents reporting positive outcomes (e.g., Bayat 2007; Twoy et al. 2007). In a seminal

review paper, Hastings and Taunt (2002) called for more research examining positive factors associated with parenting a child with a developmental disability, including ASD. This includes identifying factors (i.e., protective factors) that lead to positive outcomes in the context of the challenges associated with raising a child with a disability. A focus on positive adaptation is consistent with the construct of resilience in which the outcomes are expected to index competence (i.e., increase in positive outcomes or a decrease in negative outcomes). Researchers have heeded this call, and the last 10–15 years have witnessed an increase in research devoted to studying protective factors and positive adaptation among parents of children with ASD.

Current Knowledge

Researchers have identified a variety of protective factors associated with positive adaptation in parents of children with ASD. As discussed above, a protective factor may have a direct or interactive effect on adaptation in the context of risk. These protective factors are varied and include social support, characteristics of the parent, religiosity/spirituality, coping styles, and cognitive appraisals. The current state of knowledge of each of these protective factors is discussed below.

Social Support. Social support refers to the formal (e.g., government agencies) and informal (e.g., friends, family, and social media) supports that assist parents in coping with the challenges associated with raising a child with ASD. A review of the literature by Boyd (2002) summarized research showing that greater social support was associated with less stress among parents of children with ASD. Similar associations have also been found among mothers of adolescent and adult children with ASD. For example, Smith et al. (2012) found that a larger social support network predicted improvements in psychological well-being over an 18-month period. In more recent years, Tobing and Glenwick (2007) found that mothers' *satisfaction* with social support was associated with lower levels of maternal distress.

Social support can be provided by a variety of sources. Ekas et al. (2010) examined support received by friends, family, and romantic partners. Higher levels of family support (e.g., grandparents, aunts, uncles, other children) were associated with lower levels of maternal depression, stress, and negative affect among mothers of children with ASD. Of particular interest to the construct of resilience, family support was also associated with higher levels of positive affect, life satisfaction, and psychological well-being via an increase in optimism.

Social support can also be formal in nature. Some research shows that formal support services may be unhelpful or have no effect on parent outcomes (e.g., Schwichtenberg and Poehlmann 2007; White and Hastings 2004). However, one study found that families receiving government-funded services (i.e., Medicaid waivers) reported having better emotional and physical health outcomes compared to families who were not receiving services (Eskow et al. 2011). Parent education and support groups are additional forms of formal support services that show promising effects on promoting positive adaptation among parents of children with ASD (e.g., Samadi et al. 2013). Taken together, this body of research demonstrates that social support serves as a protective factor for parents of children with ASD.

Parent Characteristics. Researchers have studied whether characteristics of the parent influences their positive adaptation. Characteristics that have received empirical attention include dispositional traits such as optimism and hardiness and cognitive ways of thinking (e.g., locus of control, self-efficacy). Among parents of children with ASD, optimism has emerged as a robust predictor of outcomes. Optimism refers to the tendency to expect positive outcomes in life (Scheier and Carver 1985). Ekas et al. (2010) found higher levels of optimism were associated with increased positive affect, life satisfaction, and psychological well-being in mothers of children with ASD. In a study of Hispanic parents, mothers and fathers reported similar levels of optimism and increased optimism predicted lower levels of depressive symptoms (Willis et al. 2016). Hardiness is a personality trait that

consists of high levels of commitment, control, and challenge. Thus, during stressful situations, hardy individuals perceive the situation as interesting, they focus on the opportunity for growth, and they attempt to control the situation (Kobasa 1982). Weiss (2002) found that high levels of hardiness were associated with lower levels of depression, anxiety, and depersonalization in mothers of children with ASD. Hope is another dispositional characteristic that may serve as a protective factor. Individuals with high levels of hope are able to generate pathways to reach goals and feel that they are able to utilize these pathways (Snyder et al. 2000). For mothers of children with ASD, high levels of hope are associated with less worry (Ogston et al. 2011), less depressive symptoms (Ekas et al. 2016; Faso et al. 2013), and lower levels of loneliness (Ekas et al. 2016). Finally, recent research has begun to explore gratitude as a possible protective factor for parents of children with ASD. Similar to hope, gratitude is considered to be a character strength and refers to the tendency to be thankful for something or someone in one's life (Watkins et al. 2003). While there are no studies examining trait levels of gratitude, a study implementing a gratitude intervention found that mothers who wrote letters of gratitude reported increases in psychological well-being across the course of the intervention (Timmons and Ekas 2018).

In addition to dispositional traits, parents' ways of thinking may also serve as protective factors. For example, mothers and fathers of children with ASD who had an internal locus of control (i.e., believing that one's behavior can control events) were better able to cope with parenting stress (Siman-Tov and Kaniel 2011). Self-efficacy is an individual's confidence in their ability to succeed at tasks, goals, and challenges (Bandura 2010). Kuhn and Carter (2006) found that mothers of children with ASD with greater self-efficacy reported lower levels of depressive symptoms and stress. Parenting self-efficacy refers to a person's confidence in their ability to succeed in the domain of parenting. Rezendes and Scarpa (2011) found high parenting self-efficacy predicted less depressive and anxiety symptoms in mothers of children with ASD.

Taken together, the available body of research suggests that characteristics of the parent may confer protection against the challenges associated with parenting a child with ASD.

Religiosity and Spirituality. Decades of research in the general population show that religiosity and spirituality are associated with better psychological and physical health (Cohen and Johnson 2017). In the context of raising a child with ASD, however, these associations are less studied. Coulthard and Fitzgerald (1999) found that parents receive comfort from their religious beliefs and engagement in prayer, and these were associated with better physical and mental health outcomes. In another study, researchers examined multiple dimensions of religiosity and spirituality and found that spirituality was a better predictor of positive outcomes (Ekas et al. 2009). This is consistent with research showing that spiritual wellness (i.e., reporting high levels of meaning and purpose in life) is associated with less depressive symptoms (Graybill and Esquivel 2012). Ekas et al. (in press) found that spirituality may be efficacious because it influences the ways in which mothers interpret their child's disability. Research in this area is relatively sparse; however, the limited research suggests that spirituality may be a particularly important protective factor for parents of children with ASD.

Coping Styles. Coping refers to an individual's behavioral and cognitive efforts to manage stressful situations that exceed the individual's personal resources (Folkman et al. 1986). In stressful situations, an individual can utilize either of two coping strategies: (1) problem-focused coping, wherein an individual engages in problem-solving to prevent the situation from happening again, and (2) emotion-focused coping, in which the person tries to regulate the emotions associated with the situation. In the process of resilience, an individual's coping style may be a protective factor that is utilized on a day-to-day basis to cope with the adversity being faced.

Among parents of children with ASD, Hastings et al. (2005) identified four categories of coping strategies: (1) active avoidance, (2) problem-focused, (3) religious denial, and

(4) positive coping. In this study, mothers who engaged in problem-focused coping (i.e., planning, active coping, using social support) reported less depressive symptoms, whereas mothers who utilized active avoidance coping (i.e., substance use, behavioral disengagement, venting, and distraction) were more likely to report increased depressive symptoms. Smith et al. (2008) also found the use of problem-focused coping to be associated with greater maternal well-being. Similarly, a study of Hispanic mothers and fathers of children with ASD found that the use of positive coping strategies (e.g., planning, reframing, active coping) was associated with less depressive symptoms, whereas avoidance coping predicted greater depressive symptoms (Willis et al. 2016). In sum, the use of problem-focused coping strategies appears to promote positive adaptation among parents of children with ASD.

Cognitive Appraisals. How an individual appraises the adversity they are experiencing may impact their adaptation. When faced with adversity, individuals may engage in positive reinterpretation or attempt to find meaning in the situation. Benefit finding is a form of positive reinterpretation wherein an individual is able to find positive contribution in negative or traumatic life events (Helgeson et al. 2006). Among parents of children with high-functioning ASD, higher levels of benefit finding were associated with greater positive affect (Samios et al. 2009). Benefit finding was associated with greater family cohesion (Ekas et al. 2016) and romantic relationship satisfaction (Ekas et al. 2015). Parents who were asked to write about the benefits of caregiving reported lower anxiety scores following an intervention (Lovell et al. 2016).

Parents may also make positive reinterpretations as it relates to the contributions their child with ASD makes to their lives. The positive contributions can include the child bringing the family closer together, contributing to the parent's personal growth, the child being a source of pride, and providing happiness and fulfilment to the parent (Behr et al. 1992). Mothers of preschool children with ASD reported more positive contributions compared to fathers, and these positive

contributions were associated with lower parenting stress for mothers (Kayfitz et al. 2010). Ekas et al. (in press) found that mothers who reported greater positive contributions also reported lower levels of anxiety. In sum, how a parent appraises their child's ASD diagnosis may help to promote more positive outcomes.

Future Directions

Parents of children with ASD experience higher levels of parenting stress, which can negatively impact their well-being in several domains. While many families face these challenges, protective factors may act as a buffer to provide parents of children with ASD with the capabilities to better cope with stressors that affect their well-being. However, the scope of resilience research is limited as most of these studies focus solely on mothers of children with ASD, employ a cross-sectional design, and rely exclusively on self-reports of parent well-being. Moreover, researchers have focused almost exclusively on compensatory models and have neglected additional resilience models that examine interactive effects. As such, when examining resilience in families of children with ASD, future researchers should aim to (1) include fathers in their samples, (2) examine resilience as a process that unfolds over time and employ longitudinal study designs, (3) use multi-method studies which utilize observations of parent-child interactions, and (4) specify protective models of resilience.

Studies examining the resilience process and outcomes for fathers are scarce. Fathers of children with ASD are often not included in parenting studies despite fathers playing an important role in their child's development (Braustein et al. 2013). As a result, not much is known about how protective factors operate for these fathers, as fathers may respond to situations differently than mothers. Understanding the resilience processes for fathers of children of ASD has important implications for parent-focused interventions, as improved interventions can lead to better outcomes for both parents and their child with ASD.

Additionally, when examining resilience in the context of ASD, researchers should focus on resilience as a process that unfolds over time and, thus, utilize longitudinal methodologies. As most current research employs a cross-sectional examination of resilience, it would be difficult to determine if protective factors are more effective at different points in their child's life. By exploring resilience longitudinally, researcher may come to a better understanding of when resilience has a greater impact on parent outcomes which may provide clinicians with insight as to the best times to provide services and interventions for families of children with ASD. Researchers have exclusively used parent report to examine resilience and protective factors in parents of children with ASD. However, multi-method research may provide further insights into how protective factors and positive adaptation impact other domains of parent well-being, including the quality of parenting. Thus, it would be beneficial for future research to include additional methods, such as observations, to determine how protective factors impact the quality of parent-child interactions.

Finally, the majority of research discussed above focused on the direct impact of protective factors on parent adaptation (e.g., compensatory models of resilience). However, several resilience models describe protective factors as providing a buffer against the effects of adversity. In these models, protective factors interact with adversity to impact outcomes. Statistically, these resilience models can be tested using moderation. For example, in a study of mothers of children with ASD, Ekas and Whitman (2011) found that daily positive affect buffered the negative impact of stress but only on days of low to moderate levels of stress. Similarly, social support and coping styles moderated the association between stressful life events and negative outcomes for mothers and fathers of children with ASD (Dunn et al. 2001). Lyons et al. (2010) found that certain coping styles buffered the negative effects of child ASD symptoms severity. Further research is needed with additional protective factors, such as optimism, hope, and cognitive appraisals.

See Also

- [DIR Model \(Developmental, Individual Difference, Relationship Based\): A Parent-Mediated Mental Health Approach to Autism Spectrum Disorders, The](#)
- [Parent Responsiveness to Children at Risk of ASD](#)
- [Parental Response to Diagnosis](#)

References and Reading

- Abbeduto, L., Seltzer, M. M., Shattuck, P., Krauss, M. W., Orsmond, G., & Murphy, M. M. (2004). Psychological well-being and coping in mothers of youths with autism, down syndrome, or fragile X syndrome. *American Journal on Mental Retardation*, 109(3), 237–254.
- Bandura, A. (2010). Self-efficacy. In *The Corsini encyclopedia of psychology* (pp. 1–3). Hoboken: Wiley.
- Bayat, M. (2007). Evidence of resilience in families of children with autism. *Journal of Intellectual Disability Research*, 51(9), 702–714.
- Behr, S. K., Murphy, D. L., & Summers, J. A. (1992). *User's manual: Kansas inventory of parental perceptions (KIPP)*. Lawrence: Beach Center on Families and Disability.
- Benson, P. R. (2014). Coping and psychological adjustment among mothers of children with ASD: An accelerated longitudinal study. *Journal of Autism and Developmental Disorders*, 44(8), 1793–1807.
- Boyd, B. A. (2002). Examining the relationship between stress and lack of social support in mothers of children with autism. *Focus on Autism and Other Developmental Disabilities*, 17(4), 208–215.
- Braustein, V. L., Peniston, N., Perelman, A., Cassano, M.C. (2013). The inclusion of fathers in investigations of autistic spectrum disorders. *Research in Autism Spectrum Disorders*, 7, 858–865.
- Brobst, J. B., Clopton, J. R., & Hendrick, S. S. (2009). Parenting children with autism spectrum disorders: The couple's relationship. *Focus on Autism and Other Developmental Disabilities*, 24(1), 38–49.
- Buescher, A. V., Cidav, Z., Knapp, M., & Mandell, D. S. (2014). Costs of autism spectrum disorders in the United Kingdom and the United States. *JAMA Pediatrics*, 168(8), 721–728.
- Cohen, A. B., & Johnson, K. A. (2017). The relationship between religion and well-being. *Applied Research in Quality of Life*, 12(3), 533–547.
- Coulthard, P., & Fitzgerald, M. (1999). In god we trust? Organised religion and personal beliefs as resources and coping strategies, and their implications for health in parents with a child on the autistic spectrum. *Mental Health, Religion and Culture*, 2(1), 19–33.

- Davis, N. O., & Carter, A. S. (2008). Parenting stress in mothers and fathers of toddlers with autism spectrum disorders: Associations with child characteristics. *Journal of Autism and Developmental Disorders*, 38(7), 1278.
- Dunn, M. E., Burbine, T., Bowers, C. A., & Tantleff-Dunn, S. (2001). Moderators of stress in parents of children with autism. *Community Mental Health Journal*, 37(1), 39–52.
- Ekas, N., & Whitman, T. L. (2010). Autism symptom topography and maternal socioemotional functioning. *American Journal on Intellectual and Developmental Disabilities*, 115(3), 234–249.
- Ekas, N. V., & Whitman, T. L. (2011). Adaptation to daily stress among mothers of children with an autism spectrum disorder: The role of daily positive affect. *Journal of Autism and Developmental Disorders*, 41(9), 1202–1213.
- Ekas, N. V., Whitman, T. L., & Shivers, C. (2009). Religiosity, spirituality, and socioeconomic functioning in mothers of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 39, 706–719.
- Ekas, N. V., Lickenbrock, D. M., & Whitman, T. L. (2010). Optimism, social support, and well-being in mothers of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 40(10), 1274–1284.
- Ekas, N. V., Timmons, L., Pruitt, M., Ghilain, C., & Alessandri, M. (2015). The power of positivity: Predictors of relationship satisfaction for parents of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(7), 1997–2007. <https://doi.org/10.1007/s10803-015-2362-4>.
- Ekas, N. V., Pruitt, M. M., & McKay, E. (2016). Hope, social relations, and depressive symptoms in mothers of children with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 29, 8–18.
- Ekas, N. V., Tidman, L., & Timmons, L. (in press). Religiosity/spirituality and mental health outcomes in mothers of children with autism spectrum disorder: The mediating role of positive thinking. *Journal of Autism and Developmental Disorders*.
- Eskow, K., Pineles, L., & Summers, J. A. (2011). Exploring the effect of autism waiver services on family outcomes. *Journal of Policy and Practice in Intellectual Disabilities*, 8(1), 28–35.
- Faso, D. J., Neal-Beevers, A. R., & Carlson, C. L. (2013). Vicarious futurity, hope, and well-being in parents of children with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 7(2), 288–297.
- Fergus, S., & Zimmerman, M. A. (2005). Adolescent resilience: A framework for understanding healthy development in the face of risk. *Annual Review of Public Health*, 26, 399–419.
- Fernández-Alcántara, M., García-Caro, M. P., Pérez-Marfil, M. N., Hueso-Montoro, C., Laynez-Rubio, C., & Cruz-Quintana, F. (2016). Feelings of loss and grief in parents of children diagnosed with autism spectrum disorder (ASD). *Research in Developmental Disabilities*, 55, 312–321.
- Folkman, S., Lazarus, R. S., Dunkel-Schetter, C., DeLongis, A., & Gruen, R. J. (1986). Dynamics of a stressful encounter: Cognitive appraisal, coping, and encounter outcomes. *Journal of Personality and Social Psychology*, 50(5), 992.
- Goin-Kochel, R. P., Mackintosh, V. H., & Myers, B. J. (2006). How many doctors does it take to make an autism spectrum diagnosis? *Autism*, 10(5), 439–451.
- Graybill, A., & Esquivel, G. (2012). Spiritual wellness as a protective factor in predicting depression among mothers of children with autism spectrum disorders. *Journal of Religion, Disability & Health*, 16, 74–87.
- Hartley, S. L., Barker, E. T., Seltzer, M. M., Floyd, F., Greenberg, J., Orsmond, G., & Bolt, D. (2010). The relative risk and timing of divorce in families of children with an autism spectrum disorder. *Journal of Family Psychology*, 24(4), 449.
- Hartley, S. L., DaWalt, L. S., & Schultz, H. M. (2017a). Daily couple experiences and parent affect in families of children with versus without autism. *Journal of Autism and Developmental Disorders*, 47(6), 1645–1658.
- Hartley, S. L., Papp, L. M., Mihaila, I., Bussanich, P. M., Goetz, G., & Hickey, E. J. (2017b). Couple conflict in parents of children with versus without autism: Self-reported and observed findings. *Journal of Child and Family Studies*, 26(8), 2152–2165.
- Hastings, R. P., & Taunt, H. M. (2002). Positive perceptions in families of children with developmental disabilities. *American Journal on Mental Retardation*, 107(2), 116–127.
- Hastings, R. P., Kovshoff, H., Brown, T., Ward, N. J., Espinosa, F., & Remington, B. (2005). Coping strategies in mothers and fathers of preschool and school-age children with autism. *Autism*, 9(4), 377–391.
- Helgeson, V. S., Reynolds, K. A., & Tomich, P. L. (2006). A meta-analytic review of benefit finding and growth. *Journal of Consulting and Clinical Psychology*, 74(5), 797–816. <https://doi.org/10.1037/0022-006X.74.5.797>.
- Hurley, R. S., Losh, M., Parlier, M., Reznick, J. S., & Piven, J. (2007). The broad autism phenotype questionnaire. *Journal of Autism and Developmental Disorders*, 37(9), 1679–1690.
- Ingersoll, B., & Hambrick, D. Z. (2011). The relationship between the broader autism phenotype, child severity, and stress and depression in parents of children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5(1), 337–344.
- Kayfitz, A. D., Gragg, M. N., & Orr, R. (2010). Positive experiences of mothers and fathers of children with autism. *Journal of Applied Research in Intellectual Disabilities*, 23, 337–343. <https://doi.org/10.1111/j.1468-3148.2009.00539.x>.

- Kobasa, S. C. (1982). The hardy personality: Toward a social psychology of stress and health. *Social Psychology of Health and Illness*, 4, 3–32.
- Kuhn, J. C., & Carter, A. S. (2006). Maternal self-efficacy and associated parenting cognitions among mothers of children with autism. *American Journal of Orthopsychiatry*, 76(4), 564–575.
- Lovell, B., Moss, M., & Wetherell, M. A. (2016). Assessing the feasibility and efficacy of written benefit-finding for caregivers of children with autism: A pilot study. *Journal of Family Studies*, 22(1), 32–42. <https://doi.org/10.1080/13229400.2015.1020987>.
- Luthar, S. S., & Cicchetti, D. (2000). The construct of resilience: Implications for interventions and social policies. *Development and Psychopathology*, 12(4), 857–885.
- Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development*, 71(3), 543–562.
- Lyons, A. M., Leon, S. C., Phelps, C. E. R., & Dunleavy, A. M. (2010). The impact of child symptom severity on stress among parents of children with ASD: The moderating role of coping styles. *Journal of Child and Family Studies*, 19(4), 516–524.
- Ogston, P. L., Mackintosh, V. H., & Myers, B. J. (2011). Hope and worry in mothers of children with an autism spectrum disorder or Down syndrome. *Research in Autism Spectrum Disorders*, 5(4), 1378–1384.
- Pruitt, M. M., Rhoden, M., & Ekas, N. V. (2018). Relationship between the broad autism phenotype, social relationships and mental health for mothers of children with autism spectrum disorder. *Autism*, 22(2), 171–180.
- Rezendes, D. L., & Scarpa, A. (2011). Associations between parental anxiety/depression and child behavior problems related to autism spectrum disorders: The roles of parenting stress and parenting self-efficacy. *Autism Research and Treatment*, 2011, 1.
- Rutter, M. (1987). Psychosocial resilience and protective mechanisms. *American Journal of Orthopsychiatry*, 57(3), 316–331.
- Saini, M., Stoddart, K. P., Gibson, M., Morris, R., Barrett, D., Muskat, B., ... & Zwaigenbaum, L. (2015). Couple relationships among parents of children and adolescents with autism spectrum disorder: Findings from a scoping review of the literature. *Research in Autism Spectrum Disorders*, 17, 142–157.
- Samadi, S. A., McConkey, R., & Kelly, G. (2013). Enhancing parental well-being and coping through a family-centred short course for Iranian parents of children with an autism spectrum disorder. *Autism*, 17(1), 27–43.
- Samios, C., Pakenham, K. I., & Sofronoff, K. (2009). The nature of benefit finding in parents of a child with Asperger syndrome. *Research in Autism Spectrum Disorders*, 3, 358–374. <https://doi.org/10.1016/j.rasd.2008.08.003>.
- Sasson, N. J., Lam, K. S., Childress, D., Parlier, M., Daniels, J. L., & Piven, J. (2013). The B road Autism Phenotype Questionnaire: Prevalence and Diagnostic Classification. *Autism Research*, 6(2), 134–143.
- Scheier, M. F., & Carver, C. S. (1985). Optimism, coping, and health: Assessment and implications of generalized outcome expectancies. *Health Psychology*, 4(3), 219.
- Schwichtenberg, A., & Poehlmann, J. (2007). Applied behaviour analysis: Does intervention intensity relate to family stressors and maternal well-being? *Journal of Intellectual Disability Research*, 51(8), 598–605.
- Sim, A., Cordier, R., Vaz, S., Parsons, R., & Falkmer, T. (2017). Relationship satisfaction and dyadic coping in couples with a child with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(11), 3562–3573.
- Siman-Tov, A., & Kaniel, S. (2011). Stress and personal resource as predictors of the adjustment of parents to autistic children: A multivariate model. *Journal of Autism and Developmental Disorders*, 41(7), 879–890.
- Smith, L. E., Seltzer, M. M., Tager-Flusberg, H., Greenberg, J. S., & Carter, A. S. (2008). A comparative analysis of well-being and coping among mothers of toddlers and mothers of adolescents with ASD. *Journal of Autism and Developmental Disorders*, 38(5), 876.
- Smith, L. E., Greenberg, J. S., & Seltzer, M. M. (2012). Social support and well-being at mid-life among mothers of adolescents and adults with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(9), 1818–1826.
- Snyder, C. R., Feldman, D. B., Taylor, J. D., Schroeder, L. L., & Adams, V. H., III. (2000). The roles of hopeful thinking in preventing problems and enhancing strengths. *Applied and Preventive Psychology*, 9(4), 249–269.
- Timmons, L., & Ekas, N. V. (2018). Giving thanks: Findings from a gratitude intervention with mothers of children with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 49, 13–24.
- Tobing, L. E., & Glenwick, D. S. (2007). Predictors and moderators of psychological distress in mothers of children with pervasive developmental disorders. *Journal of Family Social Work*, 10(4), 1–22.
- Twoy, R., Connolly, P. M., & Novak, J. M. (2007). Coping strategies used by parents of children with autism. *Journal of the American Academy of Nurse Practitioners*, 19(5), 251–260.
- Walsh, F. (1998). *The Guilford family therapy series. Strengthening family resilience*. New York: Guilford Press.
- Walsh, F. (2006). *Strengthening family resilience* (2nd ed.). New York: The Guilford Press.
- Watkins, P. C., Woodward, K., Stone, T., & Kolts, R. L. (2003). Gratitude and happiness: Development of a measure of gratitude, and relationships with subjective well-being. *Social Behavior and Personality: An International Journal*, 31(5), 431–451.
- Weiss, M. J. (2002). Hardiness and social support as predictors of stress in mothers of typical children, children

- with autism, and children with mental retardation. *Autism*, 6(1), 115–130.
- Wheelwright, S., Auyeung, B., Allison, C., & Baron-Cohen, S. (2010). Defining the broader, medium and narrow autism phenotype among parents using the Autism Spectrum Quotient (AQ). *Molecular Autism*, 1(1), 10.
- White, N., & Hastings, R. P. (2004). Social and professional support for parents of adolescents with severe intellectual disabilities. *Journal of Applied Research in Intellectual Disabilities*, 17(3), 181–190.
- Willis, K., Timmons, L., Pruitt, M., Schneider, H. L., Alessandri, M., & Ekas, N. V. (2016). The relationship between optimism, coping, and depressive symptoms in Hispanic mothers and fathers of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(7), 2427–2440.

Resource Distribution

- [Monotropism: An Interest-Based Account of Autism](#)

Resource Room

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Synonyms

[Pull-out room](#); [Send-out room](#)

Definition

A resource room is defined as a separate classroom for special education outside the mainstream classroom (Volkmar and Wiesner 2009). In a resource room, students with disabilities leave the general education classroom for a specified period of time to attend the resource room and receive specialized instruction as designated by the goals and objectives outlined in their Individual Education Plans (IEP) (Mastropieri and

Scruggs 2010). The instruction may be individualized or presented in a small group format.

References and Reading

- Jenkins, J. R., & Heinen, A. (1989). Students' preferences for service delivery: Pull-out, in-class, or integrated models. *Exceptional Children*, 55(6), 516–523.
- Lingo, A. S., Barton-Arwood, S. M., & Jolivet, K. (2011). Teachers working together. *Teaching Exceptional Children*, 43(3), 6–13.
- Ludlow, B. (2011). Collaboration. *Teaching Exceptional Children*, 43(3), 4.
- Mastropieri, M. A., & Scruggs, T. E. (2010). *The inclusive classroom: Strategies for effective differentiated instruction* (4th ed.). Upper Saddle River: Merrill.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know*. Hoboken: Wiley.

Respiratory Sinus Arrhythmia (RSA)

- [Physiological Measures of Parental Stress](#)

Respite Care

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Definition

Individuals with ASDs typically require more intensive, individualized attention to meet specific social and communication needs as well as address behavioral excesses. As a result, family members of individuals with ASDs often report having less time for other familial and individual responsibilities and needs and report experiencing higher levels of stress. Thus, respite care is a service designed to reduce caregiver stress by affording a period of time in which the primary caregiver's role is temporarily filled by a provider

who interacts with and meets the needs of the individual. This is not only beneficial for caregiver(s) but may further develop and generalize interpersonal skills for individuals with ASDs. Unfortunately, not everyone who needs respite services receives them. Funding for respite care is allocated through general federal government, federal Medicaid, state, and private grants. A developmental disability diagnosis, documentation of IQ score, an adaptive functioning assessment, and a psychosocial assessment are typically needed with further consideration given to financial need, enrollment in other services, and availability of funds. However, not all ASDs are weighted equally. Individuals with Asperger syndrome and pervasive developmental disorder not otherwise specified may be ineligible through certain funding sources. Additionally, many grant sources are aimed solely at assisting families caring for children with ASDs. However, individual state departments on aging can be helpful with linking resources for older adults with ASDs.

The Lifespan Respite Care Act of 2006 was enacted to establish a formal program to assist family caregivers in accessing affordable and high-quality respite care. Respite can be provided at home, a day center, or a residential program and may last for a few hours, a day, or longer. Standards of respite care include meeting with a family prior to the provision of care in order to address an individual's specific needs and routines. This is essential to maintaining continuity, inherent to positive outcomes when working with individuals with ASDs.

While standards of respite care are necessary for quality of care for an individual with an ASD, the primary objective of respite is a decrease in stress levels of the caregiver, so that the individual may resume and provide care-giving responsibilities at the highest level. This may be achieved through any variety of caregiver activities. Respite breaks may be used to attend to an emergency, responsibility, or task, focusing more time in a relationship with another child or family member, or recreation, such as going to see a movie, spending time with friends, or going for a walk. Benefits of respite care for family caregivers include reduced level of stress and/or depression,

greater sense of control, improved family functioning, feeling of rejuvenation from having time to self, and more opportunities for interpersonal connectedness through social outings (Chan and Sigafoos 2001a, b; Chou et al. 2008; MacDonald and Callery 2004; McConkey et al. 2004).

References and Reading

- Chan, J., & Sigafoos, J. (2001a). Adults with intellectual disability in long-term respite care: A qualitative study. *Journal of Intellectual and Developmental Disability*, 26, 339–344.
- Chan, J., & Sigafoos, J. (2001b). Does respite care reduce parental stress in families with developmentally disabled children? *Child and Youth Care Forum*, 30, 253–263.
- Chou, Y., Tzou, P., Pu, C., Kroger, T., & Lee, W. (2008). Respite care as a community care service: Factors associated with the effects of family carers of adults with intellectual disability in Taiwan. *Journal of Intellectual and Developmental Disability*, 33, 12–21.
- MacDonald, H., & Callery, P. (2004). Different meanings of respite: A study of parents, nurses and social workers caring for children with complex needs. *Child Care, Health and Development*, 30, 279–288.
- McConkey, R., Truesdale, M., & Conliffe, C. (2004). The features of short-break residential services valued by families who have children with multiple disabilities. *Journal of Social Work*, 4, 61–75.

Respondent Behavior

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Synonyms

[Reflexive behavior](#)

Definition

Respondent behavior is the term used to describe behavior which is elicited, or brought about, by preceding stimulus events in the environment (Cooper et al. 2019).

Best known by the work of Ivan Pavlov, respondent behavior can be conceptualized as a behavior in which an organism engages in as a result of a preceding event occurring in its environment (Pear and Eldridge 1984). Also referred to as a stimulus–response paradigm, respondent behaviors primarily refer to reflexive behavior, or innate behaviors which individuals come equipped with (Cooper et al. 2019). These behaviors are believed to have a protective value for the organism and its survival (Sturmey et al. 2020). A commonly used example is the behavior of eye blinking in response to a puff of air (Sturmey et al. 2020).

In his work, Pavlov determined that respondent behaviors can come under the control of preceding stimuli. He discovered that when a tone was repeatedly presented to a dog along with a piece of meat, the dog learned to associate the tone with the meat and eventually began to salivate to the sound of the tone alone. No longer requiring the meat to be present, the tone itself gained the ability to elicit salivation behavior. Pavlov’s work, along with the work of other early behaviorists, demonstrated that reflexive behaviors, or behaviors a species is born with, are both elicited by stimuli which preceded it, and can be conditioned to occur or not occur (Pierce and Cheney 2013).

Respondent behaviors are important to consider in the context of interventions and supports for all individuals including those with autism spectrum disorder (ASD). Consider the student who has an auditory sensitivity attending PE class. Upon the PE teacher blowing their whistle (stimuli) the student may cover their ears (respondent behavior) in response to the loud sound. Through the process of respondent conditioning (see ► [Respondent Behavior](#)), it is likely that the student will begin to cover their ears (respondent behavior) upon sight of the whistle (stimuli) without the teacher having to blow it. Aiding that individual to successfully navigate PE class and instances of the whistle blowing may be an important outcome for their education, but understanding the process by which ear covering occurs is an important first step.

Respondent behaviors are often the target of interventions to increase the health, happiness, and success of individuals with ASD as well. As an

example, systematic desensitization (see ► [“Desensitization”](#)) can increase health behaviors such as completion of dental procedures (Hernandez and Ikkanda 2011). When targeting the conditioned respondent behavior and the embedded learning process, interventionists can better support individual needs (Pierce and Cheney 2013).

See Also

- [Associative Learning](#)
- [Behavior](#)
- [Classical Conditioning](#)
- [Operant Behavior](#)
- [Pavlovian Conditioning](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2019). *Applied behavior analysis* (3rd ed.). Hoboken: Pearson Education.
- Hernandez, P., & Ikkanda, Z. (2011). Applied behavior analysis: Behavior management of children with autism spectrum disorders in dental environments. *The Journal of the American Dental Association*, 142(3), 281–287. <https://doi.org/10.14219/jada.archive.2011.0167>.
- Pear, J. J., & Eldridge, G. D. (1984). The operant–respondent distinction: Future directions. *Journal of the Experimental Analysis of Behavior*, 42(3), 453–467.
- Pierce, W. D., & Cheney, C. D. (2013). *Behavior analysis and learning* (5th ed.). New York: Psychology Press.
- Sturmey, P., Ward-Horner, J., & Doran, E. (2020). Respondent and operant behavior. In Sturmey, P. (Ed.), *Functional analysis in clinical treatment: Second edition* (pp. 25–56). London, UK: Academic Press. <https://doi.org/10.1016/B978-0-12-805469-7.00002-4>.

Response Cost

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Definition

Response cost is a procedure in which an individual loses a specified amount of a previously earned reinforcer contingent upon a behavior targeted for

reduction. It is often described in terms of penalties, fines, or fees and can be found in a behavior reduction plan for persons with autism.

The term and procedure originated in a basic laboratory study by Weiner (1962) to describe a contingency in which a person's response resulted in the removal of a point from an automated point counter. Since then, it has been examined in applied research and used to reduce or eliminate a large number of behavior problems across a large number of populations.

It is most frequently found as a component in token, point, and level systems, but has also been successfully used as a stand-alone procedure. Because it is a punishment procedure, it is vulnerable to the same unwanted side effects as most punishment (e.g., emotional reaction, behavior change that is situation-specific), but because it is almost always coupled with positive reinforcement, these risks can be reduced.

See Also

- [Punishment](#)
- [Token Economy](#)
- [Type II Punishment, Second Edition](#)
- [Token Economy](#)

References and Reading

- Falcomata, T. S., Roane, H. S., Hovanetz, A. N., Kettering, T. L., & Keeney, K. M. (2004). An evaluation of response cost in the treatment of inappropriate vocalizations maintained by automatic reinforcement. *Journal of Applied Behavior Analysis*, 37, 83–87.
- Keeney, K. M., Fisher, W. W., Adelinis, J. D., & Wilder, D. A. (2000). The effects of response cost in the treatment of aberrant behavior maintained by negative reinforcement. *Journal of Applied Behavior Analysis*, 33(2), 255–258.
- Matson, J. (2009). *Applied behavior analysis for children with autism spectrum disorders*. New York: Springer.
- Reese, M., Sherman, J., & Sheldon, J. (1998). Reducing disruptive behavior of a group-home resident with autism and mental retardation. *Journal of Autism and Developmental Disorders*, 28(2), 159–165.
- Weiner, H. (1962). Some effects of response cost upon human operant behavior. *Journal of the Experimental Analysis of Behavior*, 5, 201–208.

Response Effort

- [Effort \(Behavioral Assessment\)](#)

Response Interruption/Redirection

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Definition

Response interruption and redirection (RIRD) is an applied behavior analytic procedure commonly implemented to treat stereotypic behavior and other responses thought to be maintained by the sensory consequences of the response (i.e., automatic reinforcement; see Rapp and Vollmer 2005). RIRD entails interrupting each instance of the target behavior and redirecting to an appropriate response (Ahearn et al. 2007). For example, if a child emits stereotypic vocalizations, then a caregiver asks the child social questions (e.g., “what’s your name?” “where do you live?” “what’s your brother’s name?”) they have readily answered in the past. Once the child answers the questions in the absence of stereotypic vocalizations, the caregiver provides brief praise and ceases asking the child questions. RIRD and a related procedure, response blocking, has produced significant change for both motoric and vocal stereotypic responses as well as with automatically reinforced self-injurious behavior. However, RIRD is not always necessary to decrease stereotypy when appropriate behavior is promoted or when stereotypy is not interfering with social functioning. When RIRD is used it should be combined with instruction and reinforcement for appropriate behavior that occurs in situations in which stereotypy is interfering. Colón et al. (2012) directly introduced verbal operant training in an effort to increase appropriate vocalizations and decrease vocal stereotypy. Vocal stereotypy persisted at clinically unacceptable levels for two

of the three participants. RIRD was then introduced and was effective at decreasing stereotypy but was unnecessary for the other participant.

Love et al. (2012) offered noncontingent access to matched stimulation (toys that produced sound) as a means of providing an alternative activity while evaluating whether a decrease in vocal stereotypy would occur concomitantly. Matched stimulation and RIRD were then compared as a combined package and alone. RIRD was necessary to decrease vocal stereotypy to clinically significant levels and appropriate behavior continued to occur when RIRD was used while matched activities were present. Although RIRD has been shown to be an effective procedure, Duffy-Cassella et al. (2011) and Miguel et al. (2009) suggested that RIRD may require effortful treatment application particularly if an individual initially engages in high rates of stereotypy. However, two studies suggest that once RIRD has been implemented with high levels of integrity (i.e., correctly and for each instance during the times/situations in which stereotypy is being treated) that not every instances of stereotypy needs to be redirected for a treatment effect to be maintained (Ahrens et al. 2011; Colón and Ahearn 2019). Also, Sivaraman and Rapp (2019) examined the subsequent effects of RIRD when the treatment was no longer in place and found that longer exposure to the procedure, 20 min versus 5 min, was associated decreased stereotypy persisting for a short period of time.

References and Reading

- Ahearn, W. H., Clark, K. M., MacDonald, R. P. F., & Chung, B. I. (2007). Assessing and treating vocal stereotypy in children with autism. *Journal of Applied Behavior Analysis*, 40, 263–275. <https://doi.org/10.1901/jaba.2007.30-06>.
- Ahrens, E. N., Lerman, D. C., Kodak, T., Worsdell, A. S., & Keegan, C. (2011). Further evaluation of response interruption and redirection as treatment for stereotypy. *Journal of Applied Behavior Analysis*, 44, 95–108. <https://doi.org/10.1901/jaba.2011.44-95>.
- Colón, C. L., & Ahearn, W. H. (2019). An analysis of treatment integrity of response interruption and redirection. *Journal of Applied Behavior Analysis*, 52(2), 337–354. <https://doi.org/10.1002/jaba.537>.
- Colón, C. L., Ahearn, W. H., Clark, K. M., & Masalsky, J. (2012). The effects of verbal operant training and

response interruption and redirection on appropriate and inappropriate vocalizations. *Journal of Applied Behavior Analysis*, 45, 107–120. <https://doi.org/10.1901/jaba.2012.45-107>.

- Duffy-Cassella, M., Sidener, T. M., Sidener, D. W., & Progar, P. R. (2011). Response interruption and redirection for vocal stereotypy in children with autism: A systematic replication. *Journal of Applied Behavior Analysis*, 44, 169–173. <https://doi.org/10.1901/jaba.2011.44-169>.
- Love, J. J., Miguel, C. F., Fernand, J. K., & LaBrie, J. K. (2012). The effects of matched stimulation and response interruption and redirection on vocal stereotypy. *Journal of Applied Behavior Analysis*, 45, 549–564. <https://doi.org/10.1901/jaba.2012.45-549>.
- Miguel, C. F., Clark, K., Tereshko, L., & Ahearn, W. H. (2009). The effects of response interruption and redirection and sertraline on vocal stereotypy. *Journal of Applied Behavior Analysis*, 42, 883–888.
- Rapp, J. T., & Vollmer, T. R. (2005). Stereotypy I: A review of behavioral assessment and treatment. *Research in Developmental Disabilities*, 26, 527–547. <https://doi.org/10.1016/j.ridd.2004.11.005>.
- Sivaraman, M., & Rapp, J. T. (2019). Further analysis of the immediate and subsequent effect of RIRD on vocal stereotypy. *Behavior Modification*. <https://doi.org/10.1177/0145445519838826>.

Response Latency

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Synonyms

Latency; Latency of response; Reaction time

Definition

Response latency is the duration of time between the delivery of a stimulus and the response.

References and Reading

- Malott, R. W., & Suarez, E. A. (2004). *Elementary principles of behavior* (5th ed.). Upper Saddle River: Prentice Hall.

Response Time

► Latency, Second Edition

Restricted and Repetitive Behaviors and Sleep Disturbances in ASD

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Definition

Sleep problems may contribute to, and be exacerbated by, specific behavioral challenges in individuals with ASD, including restricted and repetitive behaviors.

Historical Background

Sleep problems are common in childhood and throughout the lifespan. Approximately 20–30% of typically developing children experience sleep difficulties, with the most common problems including difficulty falling asleep and staying asleep. Among adolescents and adults, sleep problems are more often labeled as insomnia (Meltzer and Mindell 2014). Population-based studies of adults conducted around the world suggest that approximately 30% of adults experience one or more symptoms of insomnia. Sleep problems occur even more frequently in children and adults with neurodevelopmental and psychiatric comorbidities (Krakowiak et al. 2008; Richdale and Schreck 2009).

Most of the research on sleep disturbance in Autism Spectrum Disorder (ASD) has been conducted on child and adolescent populations. Children with ASD are more likely to have sleep problems than are children in the general population, with prevalence estimates ranging from 50–80% (Couturier et al. 2005; Krakowiak et al. 2008; Richdale and Schreck 2009). Parents of children with ASD are more likely to report that their child resists going to bed, has problems falling asleep, has restless sleep, experiences nighttime or early morning awakenings, and sleeps less overall. Children with ASD also have higher rates of parasomnias, such as night terrors or talking and walking during sleep (Richdale and Schreck 2009). Sleep problems present significant and unique challenges for children with ASD and their families. As sleep is critical for normal brain development, sleep problems can reduce a child's ability to learn. They may also increase rates of challenging and repetitive behavior, making it more difficult for a child to cooperate with daily routines, participate in the community, and interact with others. The behavioral cascade of sleep disturbance may have long-term detrimental effects on a child's ultimate level of functioning and ability to develop relationships (Meltzer and Mindell 2014; Veatch et al. 2017). Sleep problems in children with ASD also contribute to maternal stress and maternal mental health (Hodge et al. 2013).

Current Knowledge

Causes of sleep abnormalities within ASD are assumed to be multifactorial, encompassing many possible biological, medical, psychological, and behavioral mechanisms. Biological correlates may include disruption in the sleep-wake cycle (circadian rhythms) and in melatonin production and regulation (Veatch et al. 2015). Medical contributors to sleep disturbance may include epilepsy, gastrointestinal distress, and pharmacological side-effects (Hollway and Aman 2011). Over the last two decades, investigators have examined a wide variety of developmental, emotional, and behavioral correlates of sleep

disturbance. Areas of interest have included the association of sleep disturbance to age, intellectual, language, and adaptive skills, psychiatric comorbidities, overall intensity of core features of ASD, and specific symptoms within ASD (Hollway et al. 2013).

Initial research on sleep problems in ASD used subjective measures, such as parent or self-report questionnaires and sleep diaries. Commonly used assessment tools have included: The Children's Sleep Habits Questionnaire (CSHQ; Owens et al. 2000), the Family Inventory of Sleep Habits (FISH; Malow et al. 2009), and the Behavioral Evaluation of Disorders of Sleep (BEDS; Schreck et al. 2003). Increasingly, investigators have paired parent- and self-reports with direct measures of sleep, including actigraphy, polysomnography, and other systems of recording sleep behaviors and physiologic activity (Meltzer et al. 2012). Studies using objective measures have largely confirmed parent's report of delayed sleep onset, fragmented sleep, reduced sleep efficiency, and decreased sleep length (Goodlin-Jones et al. 2008).

Cross-sectional and longitudinal designs have been used to better understand the onset and developmental course of sleep problems in ASD. As compared to their typically developing peers, children with ASD have shown sleep disturbance as young as 30 months of age, by parent-report (Humphreys et al. 2014). Longitudinal research has shown that sleep problems typically persist from childhood into adolescence and an emerging body of literature demonstrates that sleep problems may last into adulthood (Goldman et al. 2012; Baker and Richdale 2015). Results of self-report, sleep diaries, and actigraphy suggested that cognitively able adults with ASD take longer to fall asleep, have reduced sleep efficiency, and feel less refreshed after sleep than well-matched controls (Baker and Richdale 2015).

It is established that individuals with intellectual disability experience more sleep problems than their typically developing peers (Richdale et al. 2009); however, the association among sleep, IQ, adaptive skills, and language function in children with ASD appears complex and is not

yet well understood. In one review, approximately 40% of studies found associations between IQ and sleep and 60% did not (Hollway and Aman 2011). Similarly, findings for language and adaptive skills have been mixed (Veatch et al. 2017).

Severity of core ASD symptoms, including problems with social communication and the presence of restricted and repetitive behaviors, is clearly and significantly associated with sleep disturbance. Parent ratings of children with more severe total ASD symptoms indicated more sensitivity to nighttime sleeping environments, more frequent awakenings, and more overall sleep disturbance (Giannotti et al. 2008; Schreck et al. 2004; Mayes and Calhoun 2009). In a seminal review of the correlates of sleep disturbance in ASD, Hollway and Aman (2011) postulated a bidirectional theoretical framework in which core symptoms of ASD represented vulnerability factors that increased children's risk of insomnia when presented with environmental stressors. Features of ASD increased the possibility for maladaptive coping mechanisms, such as internalizing and externalizing behavioral problems, and decreased sleep. Specifically, as ASD symptoms increased across social communication and behavior domains, children were theorized to have higher likelihood of an anxiety-related response to stressors. Because reduced sleep has been shown to disrupt daytime behavior, the model maintained a bidirectional component.

Within diagnostic features of ASD, higher rates of social impairment have been associated with poor sleep quality and shorter sleep duration among children and adolescents (Phung and Goldberg 2017; Veatch et al. 2017). The association of sleep disturbance to specific problems in developing interpersonal peer relationships has been observed across studies. Peer relationships provide an important context for development for all children. As developing and maintaining social relationships is an area of increased vulnerability for children with ASD, interventions that improve sleep may have downstream effects on social outcomes. For example, children who are less irritable, or sleepy, will be able to make gains in therapy related to social interactions and to interact more positively with others.

Though restricted, repetitive behaviors (RRB) also represent a core symptom domain within ASD, a growing body of literature has demonstrated that challenges associated with RRB may contribute to sleep disturbance over and above general ASD severity. Poor sleepers with ASD have shown higher rates of stereotypy and screaming during the night (Schreck et al. 2004), repetitive sensory motor behaviors (Hundley et al. 2016), compulsive and ritualistic behavior (Goldman et al. 2009), and aggressive behavior (Park et al. 2012). Sensory over-responsivity has also been correlated with multiple sleep problems (Mazurek and Petroski 2015). Sensory problems at bedtime may manifest as hyperarousal and excessive or negative responses to aspects of the nighttime sleeping environment.

Best practices for treating sleep disturbance in children with ASD often begin by addressing the behavioral aspects of sleep (Malow et al. 2012; Souders et al. 2017). Behavioral interventions have been identified as a first-line approach in treating sleep problems. Studies encompassing parent training through a variety of formats including handbooks, group education, and in-person and telephone-based individual sessions have all been successful at reducing time to fall asleep and increasing total length of sleep. Training content has largely included principles of sleep hygiene, such as consideration of a calm and comfortable sleeping environment, provision of ample light in daytime – particularly in the early morning upon awakening, avoiding naps, reducing caffeine, and a consistent bedtime routine that is carefully planned to move more stimulating activities to earlier in the evening and calming activities closer to bedtime. Bedtime passes that are paired with rewards for remaining in bed have been useful with older children (Moore et al. 2007). Detailed information on behavioral treatment of sleep in ASD is provided in additional references (Katz and Malow 2014; Malow et al. 2014).

Medications are commonly used to improve sleep. Melatonin is the most studied and utilized choice for children with ASD (Hollway and

Aman 2011b; Malow et al. 2016). Multiple studies have documented an association between higher rates of repetitive behaviors and the use of medication for sleep (Abel et al. 2018; Malow et al. 2016).

Future Directions

As many individuals are diagnosed with ASD, it is increasingly important for medical and behavioral health providers to be aware of the high rates of sleep difficulties in children, adolescents, and adults with ASD, to assess for these problems, and to be facile with evidence based medical and behavioral interventions. As interventions for sleep are applied within homes and families, it is important to consider family-based, collaborative, and culturally sensitive approaches to treatment (Bellesheim et al. 2019). Encouraging family-driven goals and allowing for flexibility within standardized protocols may improve treatment adherence and results. While most research has centered on care provided to families in academic health centers, behavioral sleep education provided to parents by therapists in community settings is showing promise (MacDonald et al. 2019). Other approaches include the use of telehealth or mobile phone apps (Corkum et al. 2018). Future longitudinal research may seek to better understand the bidirectional association of sleep problems to development, health, and general well-being. Further intervention studies are needed to clarify the types of behavioral treatment that most effectively address specific sleep problems at different ages and developmental and language levels. Similarly, it should be recognized that individuals with ASD will have varying behavioral needs.

See Also

- [Medical Evaluation in Autism](#)
- [Repetitive Behavior](#)
- [Restricted Interest](#)

References and Reading

- Abel, E. A., Schwichtenberg, A. J., Broadhead, M. T., & Christ, S. L. (2018). Sleep and challenging behaviors in the context of intensive behavioral intervention for children with autism. *Journal of Autism and Developmental Disorders*, 48, 3871–3884.
- Baker, E., & Richdale, A. L. (2015). Sleep patterns in adults with a diagnosis of high-functioning autism spectrum disorder. *Sleep*, 28(11), 1765–1774.
- Bellesheim, K. R., Cole, L., Coury, D. L., Yin, L., Levy, S. E., Guinee, M. A., Klatka, K., Malow, B. A., Katz, T., Taylor, J., & Sohl, K. (2019). Family-driven goals to improve care for children with autism spectrum disorder. *Pediatrics*, 142(3), e20173225.
- Corkum, P. V., Reid, G. J., Hall, W. A., Godbout, R., Stremmler, R., Weiss, S. K., Gruber, R., Witmans, M., Chambers, C. T., Begum, E. A., Pantelis, A., & Rigney, G. (2018). Evaluation of an internet-based behavioral intervention to improve psychosocial health outcomes in children with insomnia (better nights, better days): Protocol for a randomized controlled trial. *JMIR Research Protocols*, 7(3), e76.
- Couturier, J. L., Speechley, K. N., Steele, M., Norman, R., Stringer, B., & Nicholson, R. (2005). Parental perception of sleep problems in children of normal intelligence with pervasive developmental disorders: Prevalence, severity, and pattern. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44, 815–822.
- Giannotti, F., Cortesi, F., Cerquiglini, A., Miraglia, D., Vagnoni, C., Sebastiani, T., & Bernabei, P. (2008). An investigation of sleep characteristics, EEG abnormalities and epilepsy in developmentally regressed and non-regressed children with autism. *Journal of Autism and Developmental Disorders*, 38, 1888–1897.
- Goldman, S. E., Surdyka, K., Cuevas, R., Adkins, K., Wang, L., & Malow, B. A. (2009). Defining the sleep phenotype in children with autism. *Developmental Neuropsychology*, 34, 560–573.
- Goldman, S. E., Richdale, A. L., Clemons, T., & Malow, B. A. (2012). Parental sleep concerns in autism spectrum disorders: Variations from childhood into adolescence. *Journal of Autism and Developmental Disorders*, 42, 531–538.
- Goodlin-Jones, B. L., Sitnick, S. L., Tang, K., Liu, J., & Anders, T. F. (2008). The children's sleep habits questionnaire in toddlers and preschool children. *Journal of Developmental and Behavioral Pediatrics*, 29, 82–88.
- Hodge, D., Hoffman, C. D., Sweeney, D. P., & Riggs, M. L. (2013). Relationship between children's sleep and mental health in mothers of children with and without autism. *Journal of Autism and Developmental Disorders*, 43, 956–963.
- Hollway, J. A., & Aman, M. G. (2011). Sleep correlates of pervasive developmental disorders: A review of the literature. *Research in Developmental Disabilities*, 32, 1399–1421.
- Hollway, J. A., & Aman, M. G. (2011b). Pharmacological treatment of sleep disturbance in developmental disabilities: A review of the literature. *Research in Developmental Disabilities*, 32, 939–962.
- Hollway, J. A., Aman, M. G., & Butter, E. (2013). Correlates and risk markers for sleep disturbance in participants of the autism treatment network. *Journal of Autism and Developmental Disorders*, 43, 2830–2843.
- Humphreys, J. S., Gringras, P., Blair, P. S., Scott, N., Henderson, J., Fleming, P. J., & Emond, A. M. (2014). Sleep patterns in children with autistic spectrum disorders: A prospective cohort study. *Archives of Disease in Childhood*, 99(2), 114–118.
- Hundley, R. J., Shui, A., & Malow, B. A. (2016). Relationship between subtype of restricted and repetitive behaviors and sleep disturbance in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46, 3448–3457.
- Katz, T., & Malow, B. A. (2014). *Solving sleep problems in children with Autism Spectrum Disorders*. Woodbine House: A Guide for Frazzled Families.
- Krakowiak, P., Goodlin-Jones, B., Hertz-Picotto, I., Croen, L. A., & Hansen, R. L. (2008). Sleep problems in children with autism spectrum disorders, developmental delays, and typical development: A population-based study. *Journal of Sleep Research*, 17(2), 197–206.
- MacDonald, L. L., Loring, W., Wyatt, A., Fawkes, D. B., Gray, L., & Malow, B. A. (2019). Behavioral sleep education for children with autism and insomnia: Partnership with community practices. Presented at the Associated Professional Sleep Societies Annual Meeting, San Antonio.
- Malow, B. A., Crowe, C., Henderson, L., McGrew, S. G., Wang, L., Song, Y., et al. (2009). A sleep habits questionnaire for children with autism spectrum disorders. *Journal of Child Neurology*, 24(1), 19–24.
- Malow, B. A., Byars, K., Johnson, K., Weiss, S., Bernal, P., Goldman, S. E., et al. (2012). A practice pathway for the identification, evaluation, and management of insomnia in children and adolescents with autism spectrum disorders. *Pediatrics*, 130(2), S106–S124.
- Malow, B. A., Adkins, K. W., Reynolds, A., Weiss, S. K., Loh, A., Fawkes, D., Katz, T., Goldman, S. E., Madduri, N., Hundley, R. J., & Clemons, T. (2014). Parent-based sleep education for children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(1), 216–228.
- Malow, B. A., Katz, T., Reynolds, A. M., Shui, A., Carno, M., Connolly, H. V., Coury, D., & Bennett, A. E. (2016). Sleep difficulties and

- medications in children with autism spectrum disorders: A registry study. *Pediatrics*, 137(S2), S61.
- Mayes, S. D., & Calhoun, S. (2009). Variables related to sleep problems in children with autism. *Research in Autism Spectrum Disorder*, 3, 931–941.
- Mazurek, M. O., & Petroski, G. F. (2015). Sleep problems in children with autism spectrum disorder: Examining the contributions of sensory over-responsivity and anxiety. *Sleep Medicine*, 16(2), 270–279.
- Meltzer, L. J., & Mindell, J. A. (2014). Systematic review and meta-analysis of behavioral interventions for pediatric insomnia. *Journal of Pediatric Psychology*, 39(8), 932–948.
- Meltzer, L. J., Montgomery-Downs, H. E., Insana, S. P., & Walsh, C. M. (2012). Use of actigraphy for assessment in pediatric sleep research. *Sleep Medicine Reviews*, 16, 463–475.
- Moore, B. A., Friman, P. C., Fruzzetti, A. E., & MacAleese, K. (2007). Brief report: Evaluating the bedtime pass program for child resistance to bedtime – A randomized controlled trial. *Journal of Pediatric Psychology*, 32(3), 283–287.
- Owens, J. A., Spirito, A., & Mcguinn, M. (2000). The children's sleep habits questionnaire (CSHQ): Psychometric properties of a survey instrument for school-aged children. *Sleep*, 23, 1043–1051.
- Park, S., Cho, S., Cho, I., Kim, B., Kim, J., Shin, M., et al. (2012). Sleep problems and their correlates and comorbid psychopathology of children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6, 1068–1072.
- Phung, J. N., & Goldberg, W. A. (2017). Poor sleep quality is associated with discordant peer relationships among adolescent with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 34, 10–18.
- Richdale, A. L., & Schreck, K. A. (2009). Sleep problems in autism spectrum disorders: Prevalence, nature and possible biopsychosocial aetiologies. *Sleep Medicine Reviews*, 13, 403–411.
- Richdale, A. L., Francis, A., Gavidia-Payne, S., & Cotton, S. (2009). Stress, behavior, and sleep problems in children with an intellectual disability. *Journal of Intellectual and Developmental Disability*, 25(2), 147–161.
- Schreck, K. A., Mulick, J. A., & Rojahn, J. (2003). Development of the behavioral evaluation of disorders of sleep scale. *Journal of Child and Family Studies*, 12(32), 349–359.
- Schreck, K. A., Mulick, J. A., & Smith, A. F. (2004). Sleep problems as possible predictors of intensified symptoms of autism. *Research in Developmental Disabilities*, 25(1), 57–66.
- Souders, M. C., Zavodny, S., Eriksen, W., Sinko, R., Connell, J., Kerns, C., Schaaf, R., & Pinto-Martin, J. (2017). Sleep in children with autism spectrum disorder. *Current Psychiatry Reports*, 19(6), 34.
- Veatch, O. J., Goldman, S. E., Adkins, K. W., & Malow, B. A. (2015). Melatonin in children with autism spectrum disorders: How does the evidence fit together? *Journal of Nature and Science*, 1(7), e125.

- Veatch, O. J., Sutcliffe, J. S., Warren, Z. E., Keenan, B. T., Potter, M. H., & Malow, B. A. (2017). Shorter sleep duration is associated with social impairment and comorbidities in ASD. *Autism Research*, 10, 1221–1238.

Restricted Interest

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Synonyms

[Limited interests](#); [Restrictive interests](#)

Definition

A limited set or limited number of interests and/or activities. Restricted and repetitive patterns of behaviors comprise one of the diagnostic criteria for autism spectrum disorders (APA 2013). The restricted interests may be simple motor stereotypies (i.e., lining up objects), echolalia (i.e., repetition of words or phrases), insistence on sameness (i.e., non-varied food or clothes), inflexible adherence to routines (i.e., same routes or schedules), highly restricted fixated interests (i.e., a narrow or limited range of items that hold the individual's interest), and hyper- or hypo-activity to sensory input (i.e., such as sounds, textures, lights, smell) (APA 2013).

Restrictive and repetitive behaviors (RRBs) in ASD can range from extreme and obvious to subtle and infrequent (Bernier 2014). Restricted interests can be seen in children as young as 17–37 months old (Matston et al. 2009). They are pervasive and can be associated with persistent interference with the social, communicative, and adaptive functioning in individuals with ASD. The interference with or interruption of the restricted interests can evoke challenging behaviors (i.e., outbursts, tantrums, severe anxiety, etc.) and can often cause significant challenges for the individual with ASD and his/her family (Stratis and Lecavalier 2013). Notwithstanding, by using

the individual's interest to improve task performance, RRBs can be used as intrinsic reinforcement and motivating rewards (Vismara and Lyons 2007). High levels of restricted interests were found to be associated with less severe symptoms of depression in individuals with ASD, suggesting that restricted interests may be an alleviating factor against the development of depression in ASD (Stratis and Lecavalier 2013). Furthermore, routines have been found to present an individual with ASD a sense of stability, predictability, and consistency, which can minimize their risk for adjustment problems (Stoppelbein et al. 2016).

Assessment of restricted behaviors: the Repetitive and Restricted Behavior Scale (RBS; Bodfish et al. 1999) and the Repetitive and Restricted Behavior Scale-Revised (RBS-R; Bodfish et al. 2000; Lams and Aman 2007) are scales specifically designed for the assessment of RRBs. The Autism Diagnostic Observation Schedule (ADOS; Lord et al. 1999) enables observation of RRBs during the variety of structured and play-based activities. The Autism Diagnostic Interview-Revised (ADI-R; Le Couteur et al. 2003) is used to gather information through parent interview and includes 13 questions that are grouped in the “interests and behaviors” category.

See Also

- Diagnostic Interviews
- DSM-5
- Repetitive Behavior
- Restrictive Interests

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual* (5th ed.). Washington, DC: APA Press.
- Bernier, R. (2014). Assessment of the core features of ASD. In J. Tarbox, D. Dixon, P. Sturmey, & J. Matson (Eds.), *Handbook of early intervention for autism spectrum disorders, Autism and Child Psychopathology Series* (pp. 65–86). New York: Springer.
- Bodfish, J. W., Symons, F. J., & Lewis, M. H. (1999). *The repetitive behavior scales (RBS)*. Western Carolina Center Research Reports.

- Bodfish, J. W., Symons, F. J., Parker, D. E., & Lewis, M. H. (2000). Varieties of repetitive behavior in autism: Comparisons to mental retardation. *Journal of Autism and Developmental Disorders*, 30, 237–243.
- Lams, K. S., & Aman, M. G. (2007). The repetitive behavior scale revised: Independent validation in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 855–866.
- Le Couteur, A., Lord, C., & Rutter, M. (2003). *The autism diagnostic interview – Revised (ADI-R)*. Los Angeles: Western Psychological Services.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (1999). *Autism diagnostic observation schedule – WPS (ADOS-WPS)*. Los Angeles: Western Psychological Services.
- Matston, J. L., Dempsey, T., & Fodstad, J. C. (2009). Stereotypies and repetitive/restrictive behaviours in infants with autism and pervasive developmental disorder. *Developmental Neurorehabilitation*, 12, 122–127.
- Stoppelbein, L., Biasini, F., Pennick, M., & Greening, L. (2016). Predicting internalizing and externalizing symptoms among children diagnosed with an autism spectrum disorder: The role of routines. *Journal of Child and Family Studies*, 25, 251–261. <https://doi.org/10.1007/s10826-015-0218-3>.
- Stratis, E. A., & Lecavalier, L. (2013). Restricted and repetitive behaviors and psychiatric symptoms in youth with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 7, 757–766.
- Vismara, L. A., & Lyons, G. L. (2007). Joint attention behaviors in young children with autism: Theoretical and clinical implications for understanding motivation. *Journal of Positive Behavior Interventions*, 9, 214–228.

Restrictive Interests

- Restricted Interest

Retrieval of Information

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Synonyms

Recall; Recognition

Definition

The process by which memories of events or information are recalled is known as retrieval of information. Retrieval of information is one of three processes involved in long-term memory; the other two processes are encoding and storage. Two kinds of information may be stored in the brain: episodic and semantic. Episodic memories are memories of personal events, whereas semantic memories contain general knowledge that a person has learned about the world. Together, episodic and semantic memories are known as explicit (or conscious) memories. The brain also stores unconscious memories (sometimes called implicit memories). These memories allow humans to perform tasks without having to think about how to do them (e.g., writing, bicycling). Explicit memories may be retrieved via recognition or recall. Recognition occurs when a present experience is automatically associated with a stored memory. For example, answering a multiple-choice question on a test may involve recognition. Recall involves re-accessing a memory that has not been directly triggered by a present experience. Responding to a short answer question on a test is an example of a task that requires recall.

Some individuals with autism appear to have certain memory-related deficits. For instance, impairments in working memory (the capacity to store information that the brain is actively processing) and short-term memory (the capacity to store information for a brief period of time) have been found. The pattern of deficits and strengths observed in individuals with autism may be similar to that of individuals with lesions in the frontal lobe of the brain. With regard to retrieval of information, individuals with autism may have deficits that are specific to certain types of information. For instance, individuals with autism may have difficulty retrieving information from episodic (or personal experience) memory but not from semantic (or general information) memory. They may also show deficits in comparison to non-autistic individuals in recall of semantic

information that contains contextual information (e.g., a sentence as opposed to a random string of words).

It is important to note that the profile of strengths and deficits in the ability to retrieve stored information will vary across individuals with different levels of functioning and different comorbidities. Intellectual disabilities may have a significant effect on memory functioning. In assessing recall and recognition in individuals with autism, researchers and clinicians must take note of the type of information involved (e.g., social vs. nonsocial material), as performance may differ based on the content of the information to be recalled. Retrieval of information may also be dependent on the context of learning or of assessment, with variation across individuals on the ideal context for obtaining optimal performance in each area of memory.

See Also

- [Episodic Memory](#)
- [Memory](#)
- [Memory Assessment](#)
- [Memory Development](#)
- [Recognition Memory](#)
- [Semantic Memory](#)
- [Short-Term Memory](#)

References and Reading

- Bennetto, L., & Pennington, B. F. (1996). Intact and impaired memory functions in Autism. *Child Development*, 67(4), 1816–1835. <https://doi.org/10.1111/1467-8624.ep9704041063>.
- Shalom, D. (2003). Memory in autism: Review and synthesis. *Cortex*, 39(4–5), 1129–1138. [https://doi.org/10.1016/S0010-9452\(08\)70881-5](https://doi.org/10.1016/S0010-9452(08)70881-5).
- Southwick, J. S., Bigler, E. D., Froehlich, A., DuBray, M. B., Alexander, A. L., Lange, N., & Lainhart, J. E. (2011). Memory functioning in children and adolescents with autism. *Neuropsychology*, 25(6), 702–710. <https://doi.org/10.1037/a0024935>.
- Toichi, M., & Kamio, Y. (2002). Long-term memory and levels-of-processing in autism. *Neuropsychologia*, 40(7), 964–969. [https://doi.org/10.1016/S0028-3932\(01\)00163-4](https://doi.org/10.1016/S0028-3932(01)00163-4).

Retrospective Infant Video Analysis

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Definition

Autism, once a relatively rare disorder, is now seen in as many as 1 in 88 children (Baird et al. 2006; Centers for Disease Control and Prevention CDC 2012). There is considerable interest in identifying the earliest emerging signs and symptoms of autism so that interventions can be developed to ameliorate or even prevent the disorder. Retrospective home videotape analysis is one methodology that has been used to explore the early behaviors of children who were later diagnosed with autism. This technique, which involves careful coding of behaviors from home videotapes, has been used to study communication, social, and sensory motor development in infants as young as the first 6 months of life. Findings have revealed impairments evident at a very early age in children who later meet criteria for an autism spectrum disorder (ASD; see Yirmiya and Charman 2010, for a review).

Historical Background

Autism spectrum disorder (ASD) is among the most complex and heritable of neurodevelopmental disorders. The phenotype comprises a wide range of behavioral syndromes and trajectories as diverse as the underlying risk factors and etiological mechanisms for ASD (Geschwind 2008). As such, it has been difficult to characterize a specific early symptom cluster for this disorder. Attempts to do so have accelerated over the past two decades with the aim of informing the development of early interventions that can ameliorate or even prevent the course of this disorder.

A number of novel approaches have been developed to augment retrospective parent report and shed light on the earliest emerging signs and symptoms of autism. One such approach has been to examine home videos taken prior to the child's diagnosis. The advantages of this methodology include the opportunity for trained observers blind to the child's later diagnosis to examine specific behaviors over time and across varying, naturally occurring contexts. However, there is a lack of standardization with this methodology and an inherent bias that occurs when only certain behaviors and contexts are chosen by families to be videotaped. Nevertheless, these studies have elucidated a number of behaviors that distinguish children later diagnosed with autism from children with delayed and typical development, as early as within the first 6 months of life.

Current Knowledge

The first studies of home videotapes in the early 1990s involved very small samples and typically developing children as controls. Impairments identified prior to 12 months of age included less goal-directed action (Losche 1990) and deficits in social interaction including reduced eye contact, social smiling, and facial expressions as compared to typically developing infants (Adrien et al. 1991, 1993). Following these studies was a group of studies that used first birthday parties to semi-standardize the context across participants. These studies found that less eye contact and less frequent response to name distinguished 12-month-old children later diagnosed with autism from 12-month-old infants with typical *and* delayed development (Osterling and Dawson 1994; Osterling et al. 2002) and that these same two indicators distinguished children later diagnosed with autism from typically developing infants as young as 8–10 months of age (Werner et al. 2000). Werner and Dawson (2005) also found early differences between children with and without regression who were later diagnosed with autism. Specifically, this study provided evidence of intact abilities at 12 months of age in

children with regression who were later diagnosed with autism, as is often reported by parents, as compared to children without regression who showed deficits in joint attention and communication. By 24 months, children both with and without regression showed deficits compared to typically developing children in use of eye contact, response to name, and communicative skills such as gesture, words, and vocalizations. Mars, Mauk, and Dowrick (1998) were the first to extend this technology to separately examine children later diagnosed with PDD-NOS as compared to children later diagnosed with autism. They found fewer joint attention deficits in the PDD-NOS group between 12 and 30 months of age.

Home video studies over the past decade have broadened to include a wider range of behaviors including pretend play, sensory behaviors, intersubjective behaviors, and motor behaviors. Maestro et al. (2001) examined early precursors to theory of mind abilities in infants in the first 6 months of life and found that children later diagnosed with autism showed deficits in anticipating another's aim and imitation compared to typically developing infants and poorer symbolic skills between 6 and 24 months. Maestro et al. (2002, 2005, 2006) went on to show that infants with autism over the first year of life demonstrated increasing attention away from social stimuli and toward objects, and this held true whether or not the children experienced a clinical regression. More recently, abnormalities in motor activity (more fidgety movements) distinguished children later diagnosed with autism vs. typically developing children (Phagava et al. 2008), and in the first 5 months of life infants with autism demonstrated lower levels of symmetry in motor movements as compared to infants with delayed and typical development (Esposito et al. 2009). However, Ozonoff et al. (2008) found more early abnormalities in motor behaviors in children with delayed development than in children with autism, indicating that movement abnormalities may be a consequence of general developmental delay rather than specific to autism.

In sum, while comparison groups, behaviors targeted, and age ranges vary across home videotape studies, taken together, results indicate the

presence of abnormalities in social communication functioning in the first year of life in children with ASD. More specifically, in the first 6 months of life, deficits in person-person interactions (dyadic, intersubjective) and reduced attention to social stimuli are apparent in infants with autism. By the end of the first year, triadic impairments (person-object-person) are evident, including decrements in joint attention, response to name, expression of emotions, and motor movement abnormalities. While retrospective home videotape methodology has provided information on early emerging behaviors and deficits in children later diagnosed with autism, it does not tell us how many children show these same early signs and do *not* go on to develop autism. For those data, a number of prospective studies that include children at risk for autism (e.g., infant siblings of children with autism) as well as large-scale pediatric screening studies have been conducted in recent years (see Yirmiya and Charman 2010, for a review).

Future Directions

To gain a better understanding of early markers specific to children who develop autism, future research will need to combine various methodologies as well as genetic, environmental, brain, and behavioral factors; this approach will shed light on the etiology of autism as well as the trajectories of the various phenotypes found in this complex neurodevelopmental disorder.

See Also

- [Developmental Delay](#)
- [Early Diagnosis](#)
- [Infants with Autism](#)
- [Joint Attention](#)
- [Orienting Response](#)
- [Pervasive Developmental Disorder Not Otherwise Specified](#)
- [Pretend Play](#)
- [Regression](#)
- [Sensorimotor Development](#)
- [Social Communication](#)

References and Reading

- Adrien, J., Perrot, A., Hameury, L., Martineau, J., Roux, S., & Sauvage, D. (1991). Family home movies: Identification of early autistic signs in infants later diagnosed as autistics. *Brain Dysfunction*, 4, 355–362.
- Adrien, J., Lenoir, P., Martineau, J., Perrot, A., Hameury, L., Larmande, C., et al. (1993). Blind ratings of early symptoms of autism based upon family home movies. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 617–626.
- Baird, G., Simonoff, E., Pickles, A., Chandler, S., Loucas, T., Meldrum, D., et al. (2006). Prevalence of the disorders of the autism spectrum in a population cohort of children in South Thames: The Special Needs and Autism Project (SNAP). *Lancet*, 368, 210–215.
- Centers for Disease Control and Prevention (CDC). (2012). Prevalence of autism spectrum disorders – autism and developmental disabilities monitoring network, 14 sites, United States, 2002. (Surveillance Summaries). *Morbidity and Mortality Weekly Report*, 56, 12–28.
- Esposito, G., Venuti, P., Maestro, S., & Muratori, F. (2009). An exploration of symmetry in early autism spectrum disorders: Analysis of lying. *Brain & Development*, 31, 131–138.
- Geschwind, D. (2008). Autism: Many genes, common pathways? *Cell*, 135, 391–395.
- Losche, G. (1990). Sensorimotor and action development in autistic children from infancy to early childhood. *Journal of Child Psychology and Psychiatry*, 31, 749–761.
- Maestro, S., Muratori, F., Barbieri, F., Casella, C., Cattaneo, V., Cavallaro, M., et al. (2001). Early behavioral development in autistic children: The first 2 years of life through home movies. *Psychopathology*, 34, 147–152.
- Maestro, S., Muratori, F., Cavallaro, M., Pei, F., Stern, D., Golse, B., et al. (2002). Attentional skills during the first 6 months of age in autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41, 1239–1245.
- Maestro, S., Muratori, F., Cavallaro, M. C., Pecini, C., Cesari, A., Paziente, A., et al. (2005). How young children treat objects and people: An empirical study of the first year of life in autism. *Child Psychiatry and Human Development*, 35, 383–396.
- Maestro, S., Muratori, F., Cesari, A., Pecini, C., Apicella, F., & Stern, D. (2006). A view to regressive autism through home movies. Is early development really normal? *Acta Psychiatrica Scandinavica*, 113, 68–72.
- Mars, A. E., Mauk, J. E., & Dowrick, P. W. (1998). Symptoms of pervasive developmental disorders as observed in prediagnostic home videos of infants and toddlers. *Journal of Pediatrics*, 132, 1–5.
- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders*, 24, 247–257.
- Osterling, J. A., Dawson, G., & Munson, J. A. (2002). Early recognition of 1-year-old infants with autism spectrum disorder versus mental retardation. *Development and Psychopathology*, 14, 239–251.
- Ozonoff, S., Young, G., Goldring, S., Greiss-Hess, L., Herrera, A., Steele, J., et al. (2008). Gross motor development, movement abnormalities, and early identification of autism. *Journal of Autism and Developmental Disorders*, 38, 644–656.
- Phagava, H., Muratori, F., Einspieler, C., Maestro, S., Apicella, F., Guzzetta, A., et al. (2008). General movements in infants with autism spectrum disorders. *Georgian Medical News*, 156, 100–105.
- Werner, E., & Dawson, G. (2005). Validation of the phenomenon of autistic regression using home videotapes. *Archives of General Psychiatry*, 62, 889–895.
- Werner, E., Dawson, G., Osterling, J., & Dinno, N. (2000). Brief report: Recognition of autism spectrum disorder before one year of age: A retrospective study based on home videotapes. *Journal of Autism and Developmental Disorders*, 30, 157–162.
- Yirmiya, N., & Charman, T. (2010). The prodrome of autism: Early behavioral and biological signs, regression, peri- and post-natal development and genetics. *Journal of Child Psychology and Psychiatry*, 51(4), 432–458.

Rett Syndrome

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Synonyms

Cerebroatrophic hyperammonemia; Rett's disorder

Short Description or Definition

Rett syndrome is a progressive X-linked dominant neurodevelopmental disorder which affects females almost exclusively. Rett syndrome displays a characteristic pattern of cognitive and functional stagnation and subsequent deterioration. Postnatal brain growth and neuronal development are highly impacted, and Rett syndrome represents one of the most common causes of

mental retardation in females second only to Down syndrome. Stereotypic hand movements, typically at midline, are one of the most prominent symptoms. Other characteristics of this disorder include a loss of spoken language and loss of or impaired ambulation (abducted gait). Seizure activity and scoliosis or kyphosis are common comorbid disorders.

Throughout the world teams of scientists have worked to gain a better understanding of Rett syndrome, attempting to identify the cause and pathophysiology of the disorder. A major breakthrough occurred in 1999 when Dr. Amir, a scientist at Baylor University (Huston, TX), discovered that Rett syndrome is caused by a mutation of the *methyl-CpG-binding protein 2 (MECP2)* gene. Rather than an inherited disorder that is passed on from one generation to the next, Rett syndrome is typically caused by a sporadic mutation of the *MECP2* gene. In most cases, the damaged copy of *MECP2* can be traced to the paternal allele. The incidence of recurrence in a family is less than 1%.

There are a number of atypical forms of Rett syndrome including individuals with developmental delays prior to regression or who lack the initial period of normal development (congenital variant) or who display an early psychomotor delay but without regression until school age (delayed onset variant), a group whose supportive characteristics do not appear until late childhood (atypical or “forme fruste”), those with a family recurrence (familial variant), and those who have retained some speech (“preserved speech variant”).

While Rett syndrome remains a clinical diagnosis, the link with *MECP2* has made DNA testing and confirmation possible for affected individuals and their families. To date, approximately 95% of girls exhibiting Rett symptoms have one of the over 200 currently identified *MECP2* mutations.

Rett syndrome is the first human disease found to be caused by defects in a protein involved in the regulation of gene expression through its interaction with methylated DNA. As such, Rett syndrome could hold the key to our understanding of a number of human disorders ranging from some forms of learning disability to autism.

Categorization

The diagnostic criteria for Rett syndrome initially developed in the mid-1980s (Hagberg et al. 1985; The Rett Syndrome Diagnostic Criteria Work Group 1988) and revised in 2002 (Hagberg et al. 2002) and have been recently revised once again (Neul et al. 2010). The fourth edition (revised) of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR) has included Rett syndrome (termed Rett’s disorder) as a subcategory of pervasive developmental disorder. The DSM-IV-TR diagnostic criteria for 299.80 Rett’s disorder generally coincide with those initially proposed by The Rett Syndrome Diagnostic Criteria Work Group (1988). The DSM-IV-TR criteria, however, differ in some significant ways from the most recent revision of the diagnostic criteria developed by members of the Rett Search Clinical Research Consortium, an international panel of experts convened by the International Rett Syndrome Association (2010). For example, the DSM-IV-TR indicates “apparently normal psychomotor development through the first 5 months after birth,” whereas Neul and his associates (2010) suggest that “mild generalized hypotonia and other previously reported subtle developmental alterations during the first 6 months of life are common in Rett syndrome.” Deceleration of head growth between 5 and 48 months of age is a necessary criterion within the DSM-IV-TR. The revised IRSA diagnostic criteria, on the other hand, have removed deceleration of head growth as a criterion. The members of the consortium simply indicate as a note that physicians should consider Rett syndrome as a diagnosis when postnatal deceleration of head growth is observed. They did so as an increasing number of individuals with Rett syndrome have a normal head size. The DSM-IV-R requires a loss of social engagement early in the course of the disorder (that may subside to some degree later). No such requirement is found in the new IRSA criteria.

The new IRSA criteria also provide diagnostic criteria for atypical or variant forms of Rett syndrome, while no such criteria are considered in the DSM-IV-R. Individuals must display at least 2 of the 4 main criteria for classical Rett syndrome and

5 of the 11 supportive criteria to qualify for the atypical diagnosis (see Table 1). The supportive criteria provided by the IRSA criteria are not required for a diagnosis of typical or classic Rett syndrome. The DSM-IV-TR includes EEG abnormalities, seizures, nonspecific brain imaging abnormalities, and severe or profound mental retardation (Axis II) as associated features and disorders. Thus, the two sources differ in regard to associated and supportive features of the disorder. Moreover, the current DSM-IV-TR criteria fail to indicate exclusionary criteria that are critical for differential diagnosis.

Epidemiology

Persons with Rett syndrome have been reported to exist in most countries of the world. Thus, Rett syndrome does not seem to be a particularly rare disorder, and it is more or less universal. The prevalence of the Rett syndrome has been studied, based upon the Swedish registry for mental retardation and surveys of neuropsychiatrists, in a part of southwestern Sweden (Hagberg 1985). The prevalence was about 1 per 15,000 live female births. In their study of 5,400 consecutive referrals to the pediatric neurology center in western Scotland et al. (1985) identified 19 cases of Rett syndrome. The resulting prevalence rate was very similar to that reported by Hagberg (1985), 1 per 12–13,000 females (Kerr and Stephenson 1986).

A prevalence study was conducted within the United States by a number of researchers at the Baylor College of Medicine (Kozinetz et al. 1993). The study employed the Texas Rett Syndrome Registry and explored females aged 2–18 years, and was the first with a large ethnic mix that would allow an exploration of racial/ethnic group-specific prevalence of Rett syndrome. A prevalence estimate of approximately 1 per 22,800 live female births was reported with no significant differences in prevalence estimates by race/ethnicity (African American, Caucasian, and Hispanic). This estimate is lower than that reported in earlier studies, suggesting the prevalence of Rett syndrome has perhaps been overestimated.

Natural History, Prognostic Factors, Outcomes

During normal human development, a large number of genes are expressed in a tissue-specific manner. That is, the gene must only function in the creation of specific cells. Many of these genes are needed during critical periods of central nervous system (CNS) development, and then their expression must be turned off. Other genes are required only after birth, and turning these genes on and off at appropriate times are critical for normal and proper development. The *MeCP2* gene encodes the development of the transcriptional silencing *methyl-CpG-binding protein 2*. This protein plays an important role in the “silencing” of various genes during CNS development. The mutations in the *MecP2* gene in Rett syndrome result in a failure to produce the *MecP2* protein. Thus, the genes that *MeCP2* would normally silence continue to engage in ongoing transcription. The number and nature of the genes for which *MeCP2* is meant to suppress throughout the genome is as yet unknown (Singer and Naidu 2001), although there is reason to believe these genes regulate the development and mature function of the brain and central nervous system (Shahbazian and Zoghbi 2002).

Numerous different mutations of the *MeCP2* gene have been identified in individuals with Rett syndrome. Some researchers suggest that the specific type of mutation in the *MECP2* gene affects the type and severity of symptoms of Rett syndrome. Studies are now underway to understand each mutation that may cause the features of Rett syndrome, and how these mutations might change the features of the syndrome. Investigators are trying to find other genetic switches that operate in a similar way to the *MeCP2* protein. Once they discover how the protein works and locate similar switches, they may devise therapies that can substitute for the malfunctioning switch. Another outcome might involve manipulating other biochemical pathways to compensate for the malfunctioning *MECP2* gene, thereby preventing progression of the disorder. Gene therapy to achieve regulated expression of a normal

Rett Syndrome, Table 1 Diagnostic criteria for Rett syndrome and Rett's disorder

Diagnostic criteria established by Neul et al. (2010)^a Consider diagnosis when postnatal deceleration of head growth is observed Required for typical or classical Rett syndrome 1. A period of regression followed by stabilization^c 2. All main criteria and all exclusion criteria 3. Supportive criteria are not required, although often present in typical RTT Required for atypical or variant RTT 1. A period of regression followed by recovery or stabilization^b 2. At least 2 of the main 4 criteria, 3. 5 out of 11 supportive criteria Criteria Main criteria 1. Partial or complete loss of purposeful hand skills 2. Partial or complete loss of acquired spoken language^d 3. Gait abnormalities: impaired (dyspraxic) or absence of ability 4. Stereotypic hand movements such as hand-wringing/squeezing, clapping/tapping, mouthing, and washing/rubbing automatisms Exclusion criteria for typical RTT 1. Brain injury secondary to trauma (peri- or postnatally), neurometabolic disease, or severe infection that causes neurological problems^e 2. Grossly abnormal psychomotor development in first 6 months of life^f Supportive criteria for atypical RTT^g 1. Breathing disturbances when awake 2. Bruxism when awake 3. Impaired sleep pattern 4. Abnormal muscle tone 5. Peripheral vasomotor disturbances 6. Scoliosis/kyphosis 7. Growth retardation 8. Small cold hands and feet 9. Inappropriate laughing/screaming spells 10. Diminished response to pain 11. Intense eye communication – “eye pointing”	DSM-IV-TR Rett's disorder^b Necessary criteria A. All of the following: 1. Apparently normal prenatal and perinatal development 2. Apparently normal psychomotor development through the first 5 months after birth B. Onset of all of the following after the period of normal development: 1. Deceleration of head growth between ages 5 and 48 months 2. Loss of previously acquired purposeful hand skills between ages 5 and 30 months with subsequent development of stereotyped hand movements (e.g., hand-wringing and hand-washing) 3. Loss of social engagement early in the course (although often social interaction develops later) 4. Appearance of poorly coordinated gait or trunk movements 5. Severely impaired expressive and receptive language development with severe psychomotor retardation Associated features and disorders 1. Severe or profound mental retardation (axis II) 2. Increased frequency of EEG abnormalities and seizure disorders 3. Nonspecific brain imaging abnormalities
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^aReproduced with permission from Wiley–Blackwell Publishing

^bReproduced with permission from the American Psychiatric Association, *Diagnostic and statistical manual of mental disorders* (4th ed. – Revised). Washington, DC: Author

^cBecause MECP2 mutations are now identified in some individuals prior to any clear evidence of regression, the diagnosis of “possible” RTT should be given to those individuals under 3 years old who have not lost any skills but otherwise have clinical features suggestive of RTT. These individuals should be reassessed every 6–12 months for evidence of regression. If regression manifests, the diagnosis should be changes to definite RTT. However, if the child does not show any evidence of regression by 5 years, the diagnosis of RTT should be questioned

^dLoss of acquired language is based on best-acquired spoken language skill, not strictly on the acquisition of distinct words or higher language skills. Thus an individual who had learned to babble but then lost this ability is considered to have a loss of acquired language

^eThere should be clear evidence (neurological or ophthalmological examination and MRI/CT) that the presumed insult directly resulted in neurological dysfunction

^fGrossly abnormal to the point that normal milestones (acquiring head control, swallowing, developing social smile) are not met. Mild generalized hypotonia or other previously reported subtle developmental alterations during the first 6 months of life is common in RTT and do not constitute an exclusionary criterion

^gIf an individual has or ever had a clinical feature listed it is counted as a supportive criterion. Many of these features have an age dependency, manifesting and becoming more predominant at certain ages. Therefore, the diagnosis of atypical RTT may be easier for older individuals than for younger. In the case of a younger individual (under 5 years old) who has a period of regression and >2 main criteria but does not fulfill the requirements of 5/11 supportive criteria, the diagnosis of “probably atypical RTT” may be given. Individuals who fall into this category should be reassessed as they age and the diagnosis revised accordingly

MECP2 gene is also under study in animal models.

Neuropathological studies have been conducted in Rett syndrome that may assist in gaining a better understanding of how ineffective *MeCP2* activity produces Rett syndrome. While individuals with Rett syndrome are typically small for their age, only the brain weighs less than expected for the height and weight of the individual. This supports the view that the *MeCP2* gene appears to have its greatest impact on “downstream” genes that code for the development and mature function of brain tissue. Accumulated autopsy studies suggest that the brain of the individual with Rett syndrome weighs significantly less than that of controls matched for both age and height. The decrease in brain size and weight appears to begin after birth (in most cases), starting at 3–4 months of age. Brain size and weight appear to stabilize in later childhood and the absence of markers for significant degenerative disorder supports the idea that the decreased brain size is the result of arrested brain development (Duncan Armstrong 2001).

The concentrations of two major brain gangliosides, GD1a in frontal gray matter and GD1b in temporal gray matter, appear to be lowered selectively in the cerebral cortex and cerebellum in girls with Rett syndrome (Lekman et al. 1991). The ganglioside GD1a is thought to play an important role in synaptogenesis since it is prominent in synaptic membranes, and high concentrations are reported during the time when nerve ending growth and synapse formation are most intense (25th fetal week until age 2 years). Ganglioside GD1b is rich in axons and accrues more slowly, reaching maximum concentration at age 20 years. The reduction in GD1a would help explain the pathogenic findings of decreased dendritic arborization reported by Armstrong (1992).

Synapses within the cerebral cortex, basal ganglia and brainstem responsible for movement and breathing employ the use of the excitatory amino acid neurotransmitter glutamate. Glutamate-mediated neurotransmission appears to be disrupted in persons with Rett syndrome (Lappalainen and Riikonen 1996).

Clinical Expression and Pathophysiology

Individuals with classical Rett syndrome exhibit a characteristic course of development. Prenatal and perinatal histories of these persons are generally unremarkable. While minor pre- and perinatal problems (e.g., mild hypotonia, tremulous neck movements, a low intensity of interpersonal contact, and abnormal hand use and language development) can be identified retrospectively in as many as 80% of the girls, it is unlikely that these mild symptoms would be detected as relevant even with detailed neurologic or developmental assessment. This apparently normal period of development is followed by a slowing or cessation of the acquisition of developmental milestones.

Hagberg and Witt-Engerstrom (1986) proposed a staging system to facilitate the characterization of the disorder patterns and profiles from infancy through adolescence. Their system suggests four clinical stages and was derived from a synthesis of clinical observations over the years in 50 Swedish cases of Rett syndrome. The purpose of the staging system is to provide average guidelines for stage patterns thought to be of use when confronted with the diagnostic problems resulting from the complex symptomatology and longitudinal profile of the condition. The four stages include early onset stagnation phase, rapid destructive stage, pseudostationary stage, and late motor degeneration stage. This staging system has gained general acceptance; however, the names assigned to each stage have been criticized (e.g., Trevathan and Naidu 1988), and the stages are commonly referred to simply by number. The symptoms and features of each of the four stages are as follows:

- *Stage 1* (onset 6–18 months) The clinical profile at this stage suggests a deterioration or at least a general slowing down (stagnation) of motor development. Hypotonia is typically noted. Deviation from normal development is often compensated for or hidden, in part, by the rapid developmental speed of infancy. Thus, additional gross motor abilities often are learned during this stage, even as others are lost, but are delayed in their appearance. The

symptoms of stage 1 are nonspecific and are not predictive of subsequent deterioration.

- *Stage 2* (onset 1–3 years) During this stage the syndrome becomes significantly more pronounced as the children lose previously acquired abilities. In most individuals, a relatively well-demarcated period of rapidly declining social interaction, stagnation or loss of acquired cognitive abilities, and loss of purposeful hand use and speech is evident. Stereotyped movements become a prominent symptom. The intellectual functioning of the girl with Rett syndrome during this stage is generally reported to fall within the severe to profound range of mental retardation. Ataxic/aprasic gait abnormalities are observed in ambulatory girls. Also, during the time the child is awake, she may display aberrant breathing patterns. Hyperventilation and respiratory pauses (generally lasting 30–40 s) are most common. Seizure activity is present in approximately one-fourth of the girls during this stage. Sleep abnormalities including delayed sleep onset and increased night awakenings are manifested by more than three-fourths of the girls.
- *Stage 3* (onset 2–10 years) Persons with Rett syndrome generally demonstrate diminishing autistic symptomatology and improved social interaction during this period. They appear to be more aware of their surroundings and seem to make attempts at using residual functional skills. Communication skills are reported to improve with better interaction. Some girls employ eye pointing, babbling, or even word pieces to signal communicative intent. Seizures occur in up to 80% of the girls with Rett syndrome. Spasticity or rigidity and scoliosis tend to progress, and “jerky,” truncal ataxia and apraxia become prominent.
- *Stage 4* (onset 10+ years) Progressive muscle wasting, scoliosis, spasticity, and rigidity frequently are displayed during this late stage. Decreasing mobility and a number of late stage second neuron abnormalities (e.g., drop foot abnormalities, remarkably plantar-flexed feet) may require the use of a wheelchair. Spinal cord dysfunction appears to act in

conjunction with extrapyramidal features to lessen mobility. Interestingly, this stage is marked by increased motor deterioration only. The individuals’ cognitive functioning remains stable, while social interaction (eye contact) and attentiveness improve. Seizure activity often becomes less problematic, allowing for a decrease in the anticonvulsant regimen for some girls.

Although persons over the age of 25 have been identified, the oldest being 76 years of age, little systematic research has been conducted with this group to provide information on the course of the disorder past adolescence. There is some evidence to suggest that the life expectancy of persons with Rett syndrome may be diminished; however, precise information is not available at this time.

Evaluation and Differential Diagnosis

The presentation of Rett syndrome differs considerably depending upon the stage and age of observation. For example, a child of 4 or 5 years of age with classical Rett syndrome can be correctly diagnosed with relative ease. Due to vague symptomology, diagnosis during infancy is frequently misinterpreted. Likewise, the late stage in adolescence displays a common, complex, picture of severe multiple disabilities with secondary contractures resembling any number of disorders and, therefore, often is misdiagnosed. The entire disease process must be recognized and considered to fully understand this condition.

The most common nonspecific diagnosis for children with Rett syndrome above age one is reported to be the early infantile autism (DSM-IV-TR – 299.00 autistic disorder). The behavioral patterns, progression, and prognosis of these two conditions, nevertheless, differ significantly. A basic distinction between the two disorders can be made on the basis of motor behavioral analysis (Olsson and Rett 1987; Percy et al. 1988). “Whereas autism represents a regression of verbal but not motor skills, Rett syndrome involves the simultaneous regression of both skills” (Percy et al. 1988, p. S67).

Stereotypic behavior associated with infantile autism is generally more complex and, unlike that in Rett syndrome, often involves the manipulation of an object with preservation of the pincer grasp. Persons with Rett syndrome demonstrate a very restricted repertoire of movements that appear monotonous in both form and speed.

Millichap (1987) has suggested that Rett syndrome might represent a variant of childhood disintegrative disorder (Heller's syndrome). Children with childhood disintegrative disorder develop their symptoms later (e.g., at least age 2 and typically age 3 or 4 years) than individuals with Rett syndrome, and normal neurological findings are reported in persons with the former disease.

Stage 2 developmental regressions often suggest neurodegenerative diseases. For example, the earliest stages of Rett syndrome are difficult to distinguish from infantile neuronal ceroid lipofuscinosis (INCL), an autosomal recessive disease especially frequent in the Finnish population. Both disorders cause a rapid regression of psychomotor development and the manifestation of hand and finger stereotypies at approximately the same age. As INCL progresses, however, myoclonus and retinal degeneration becomes apparent and differentiates the two disorders. Failure to designate retinopathy or optic atrophy as exclusionary criteria for Rett's disorder within the DSM-IV-TR may complicate the differential diagnosis of this disorder.

A report by Philippart (1993) links Rett syndrome with tuberous sclerosis, a neurocutaneous disorder. While this disorder may show initial similarities to Rett syndrome, close examination of the skin with a Wood's lamp and the presence of serial computed tomography abnormalities will distinguish this disorder. Chromosomal disorders, such as Angelman syndrome ("happy puppet syndrome") can display similar features to Rett syndrome. Children with Angelman syndrome, however, fail to display a period of normal development and subsequent rapid regression. Acute and chronic encephalitis may be distinguished by examination of the cerebrospinal fluid (CSF) and by a characteristic electroencephalography. The loss of language and the development of

seizures in preschool-aged children are similar in both Rett syndrome and the Landau-Kleffner syndrome. Head circumference growth and motor skills are preserved in the Landau-Kleffner syndrome.

In summary, the clinical identification of Rett syndrome rests on the careful exploration of clinical manifestations and the specific pattern of symptom progression. The differential diagnosis based upon clinical observation, while frequently difficult at presentation (especially during the earliest stages), becomes much easier after follow-up over several months to a few years. A diagnosis of Rett syndrome should involve a molecular genetic analysis to identify mutations in the *MECP2* gene.

Treatment

To date, there is no cure for the Rett syndrome, and therapeutic interventions directed at the fundamental mechanisms underlying Rett syndrome have failed to demonstrate any lasting or substantive improvements. At this time, we must be satisfied to provide suitable intervention aimed at symptomatic relief.

Seizure activity is reported in approximately 80% of persons with Rett syndrome. Selection of a specific medication should be based upon clinical seizure type and EEG pattern. Several clinicians (Budden 1986; Naidu et al. 1986) agree that standard dosages of Tegretol (C-carbamazepine) constitute the best seizure management program.

Communication abilities and subsequent programming for persons with Rett syndrome have been the topic of very limited empirical research. Prior to regression, most of the girls with this disorder are reported to have developed single spoken words and/or word combinations. Comprehension skills appropriate to the child's age also are typically noted (Budden et al. 1990; Woodyatt and Ozanne 1992). The loss of language skills during regression is sufficiently rapid to be mistaken for hearing loss. Following the regression phase of the disorder, speech and language skills are observed to be severely impaired. Verbalizations are typically nonexistent or limited to "nonfunctional" consonant-vowel

combinations, except in the estimated 2–4% of girls who fall within the preserved speech variant of Rett syndrome (Budden et al. 1990). Formal testing suggests that girls with Rett syndrome function at a pre-symbolic language level (Woodyatt and Ozanne 1992, 1993). Vocalizations, facial expressions, gestures, walking toward a desired item or activity, and eye gaze are common communicative behaviors displayed by persons with Rett syndrome.

Persons with Rett syndrome exhibit multiple orthopedic and motor movement disorders. The exact nature of these disorders can vary significantly across the different phases of the syndrome. Intensive therapy, while failing to alter the actual course of the disease, has been successful in addressing symptoms by maintaining or improving functional movement, mobility, preventing deformities, and keeping the girls in contingent contact with their environments. Therapeutic intervention is especially important, and should be more frequent, during the periods of regression associated with the disorder as transition skills are at risk.

Apraxia and *Ataxia* are frequently the earliest manifestations of motor problems in Rett syndrome. A number of therapeutic interventions have been found to be successful in the treatment of apraxia-ataxia. Tone reduction techniques similar to those used with patients afflicted with cerebral palsy or impaired through a stroke are appropriate. Interventions might include (1) use of the therapy ball, (2) balance stimulating floor activities, (3) segmental rolling, and (4) rotation and weight shift activities (Hanks 1990).

Stereotyped hand movements represent one of the most distinguishing characteristics of the Rett syndrome. Hand-wringing, hand-washing, hand tapping, and hand-to-mouth movements are common stereotypies resulting in the loss of purposeful hand function. Most researchers consider the stereotypic hand movements to represent primary circular reactions resulting from an underlying extrapyramidal disorder. Thus, they represent nonvolitional movements. Persons with Rett syndrome appear to require a great deal of concentration and effort to “break out” of the stereotypic movements even for brief attempts at purposeful

hand use. The suspected neurophysiological etiology of stereotypy in Rett syndrome has led researchers to caution practitioners attempting to modify these behaviors. As these behaviors appear to represent basically involuntary movements, attempts to change this behavior, especially through the use of aversive consequences, appear ill advised (Hanks 1990). Splinting has been found to be successful in interrupting hand-to-mouth and hand-wringing movements, thus allowing the girls to direct their attention to tasks and persons in their environment and to reduce the risk of skin breakdown related to these high-rate behaviors.

Ambulation remains one of the critical skills to develop and maintain in persons with Rett syndrome. Many of the girls fail to develop this skill prior to stage II, while others lose this ability as part of the rapid motor degeneration. As spasticity and apraxia increase, the girls often lose many of their functional gross motor skills they had previously achieved. Additionally, these girls often manifest spatial disorientation. Their perception of an upright posture results in a forward, backward, or lateral leaning. Ambulation may be lost, especially for girls with a backward lean orientation, as they are unable to initiate their forward weight shift. They fear falling when attempting weight shift in a forward direction. The abnormal gait pattern typically displayed in this syndrome results from a combination of spasticity, ataxia, apraxia, compensatory spinal rigidity, and spatial disorientation. Asymmetries may develop as one leg becomes stiffer or weaker. Weight shift is accomplished through lateral rocking, and trunk rotation is lost. More simply, because the center of balance is off, girls often think they are falling forward when they are standing normally. They try to compensate by leaning back, fall over, and over time may lose the ability to stand (Kerr et al. 1990).

Independent standing and ambulation represent a realistic therapy goal, especially in the early years. Many girls while unable to walk independently can do so with assistance. Such aided ambulation should be encouraged. Weight-bearing exercises, walking, and gait training have been successful (Hanks 1990). If

appropriate, activities designed to elicit righting and equilibrium responses might include use of the large therapy ball (prone positioning and then standing, leaning forward), weight shifting (seating the child on bench and then tipping the bench slightly backward), and ultimately active shift forward to come from sitting to standing.

Scoliosis (a side-to-side curvature of the spine) in individuals with Rett syndrome is well and kyphosis (“hunchback”) is not uncommon. Together, these deformities represent the primary musculoskeletal concern in Rett syndrome. Unfortunately, there are no rigid guidelines to predict deformity or to recommend treatment. Standard criteria (e.g., sex of patient, curve pattern, onset of menarche, Risser sign) typically useful in the determination of an appropriate intervention strategy do not appear to be of use with this syndrome (Hennessy and Haas 1988).

See Also

- [Angelman/Prader-Willi Syndromes](#)
- [Apraxia](#)
- [Ataxia](#)
- [Bruxism](#)
- [Childhood Disintegrative Disorder](#)
- [DSM-IV](#)
- [Scoliosis](#)
- [Seizure Disorder](#)
- [Stereotypic Behavior](#)
- [Tuberous Sclerosis Alliance](#)

References and Reading

- Armstrong, D. D. (1992). The neuropathology of the Rett syndrome. *Brain and Development*, 14(Suppl), S89–S100.
- Budden, S. S. (1986). Rett syndrome: Studies of 13 affected girls. *American Journal of Medical Genetics*, 24 (Suppl. 1), 99–109.
- Budden, S., Meek, M., & Henighan, C. (1990). Communication and oral-motor function in Rett syndrome. *Developmental Medicine and Child Neurology*, 32, 51–55.
- Duncan Armstrong, D. (2001). Rett syndrome neuropathology review 2000. *Brain and Development*, 23, S72–S76.
- Hagberg, B. (1985). Rett syndrome: Swedish approach to analysis of prevalence and cause. *Brain and Development*, 7(3), 277–280.
- Hagberg, B., & Witt-Engerstrom, I. (1986). Rett syndrome: A suggested staging system for describing impairment profile with increasing age towards adolescence. *American Journal of Medical Genetics*, 24(Suppl. 1), 47–59.
- Hagberg, B., Goutieres, F., Hanefeld, F., Rett, A., & Wilson, J. (1985). Rett syndrome: Criteria for inclusion and exclusion. *Brain and Development*, 7(3), 372–373.
- Hagberg, B., Hanefeld, F., Percy, A., & Skjeldal, O. (2002). An update on clinically applicable diagnostic criteria in Rett syndrome. *European Journal of Paediatric Neurology*, 6, 292–297.
- Hanks, S. (1990). Motor disabilities in the Rett syndrome and physical therapy strategies. *Brain and Development*, 12, 157–161.
- Hennessy, M. J., & Haas, R. H. (1988). The orthopedic management of Rett syndrome. *Journal of Child Neurology*, 3(Suppl), S43–S47.
- Kerr, A., & Stephenson, J. B. P. (1985). Rett’s syndrome in the west of Scotland. *British Medical Journal*, 291, 579–582.
- Kerr, A., & Stephenson, J. B. P. (1986). A study of the natural history of Rett syndrome in 23 girls. *American Journal of Medical Genetics*, 24, 77–83.
- Kerr, A., Southall, D., Amos, P., Cooper, R., Samuels, M., Mitchell, J., et al. (1990). Correlation of electroencephalogram, respiration, and movement in the Rett syndrome. *Brain and Development*, 12, 61–68.
- Kozinetz, C. A., Skender, M. L., MacNaughton, N., Almes, M. J., Schultz, R. J., Percy, A. K., et al. (1993). Epidemiology of Rett syndrome: A population-based registry. *Pediatrics*, 91, 445–450.
- Lappalainen, R., & Riikonen, R. S. (1996). High levels of cerebrospinal fluid glutamate in Rett syndrome. *Pediatric Neurology*, 15, 213–216.
- Lekman, A., Hagberg, B., & Svennerholm, B. A. (1991). Membrane cerebral lipids in Rett syndrome. *Pediatric Neurologist*, 7, 186–190.
- Millichap, J. G. (1987). Rett’s syndrome: A variant of Heller’s dementia? *Lancet*, 1, 440.
- Naidu, S., Murphy, M., Moser, H. W., & Rett, A. (1986). Rett syndrome: Natural history in 70 cases. *American Journal of Medical Genetics*, 24(Suppl. 1), 61–72.
- Neul, J. L., Kaufmann, W. E., Glaze, D. G., Christodoulou, J., Clarke, A. J., Bahi-Buisson, N., et al. (2010). Rett syndrome: Revised diagnostic criteria and nomenclature. *Annals of Neurology*, 68, 944–950.
- Olsson, B., & Rett, A. (1987). Autism and Rett syndrome: Behavioral investigations and differential diagnosis. *Developmental Medicine and Child Neurology*, 29, 429–441.
- Percy, A. K., Zoghbi, H. Y., Lewis, K. R., & Jankovic, J. (1988). Rett syndrome: Qualitative and quantitative differentiation from autism. *Journal of Child Neurology*, 3(Suppl), S65–S67.
- Philippart, M. (1993). Rett syndrome associated with tuberous sclerosis in a male and in a female: Evidence

- for arrested motor and mental development. *American Journal of Medical Genetics*, 48, 229–230.
- Shahbazian, M. D., & Zoghbi, H. Y. (2002). Rett syndrome and MeCP2: Linking epigenetics and neuronal function. *American Journal of Human Genetics*, 71, 1259–1272.
- Singer, H. S., & Naidu, S. (2001). Rett syndrome: “We’ll keep the genes on for you”. *Neurology*, 56(5), 611–617.
- The Rett Syndrome Diagnostic Criteria Work Group. (1988). Diagnostic criteria for Rett syndrome. *Annals of Neurology*, 23, 125–128.
- Trevathan, E., & Naidu, S. (1988). The clinical recognition and differential diagnosis of Rett syndrome. *Journal of Child Neurology*, 3(Suppl), S6–S16.
- Woodyatt, G. C., & Ozanne, A. E. (1992). Communication abilities and Rett syndrome. *Journal of Autism and Developmental Disorders*, 22, 155–173.
- Woodyatt, G. C., & Ozanne, A. E. (1993). A longitudinal study of cognitive skills and communication behaviors in children with Rett syndrome. *Journal of Intellectual Disability Research*, 37, 419–435.

Rett's Disorder

► [Rett Syndrome](#)

Reversal Learning

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Definition

Reversal learning refers to the ability to alter one's response after encountering just a few examples of a change in consequences for a behavior. Typically, reversal learning is assessed after training on simple discrimination tasks in which an individual is presented with two (or more) stimuli (such as pictures or objects), and responses to one stimulus are reinforced (i.e., followed by praise, snack foods, or access to a toy) whereas responses to the other stimulus are not. Once a learning criterion is met, the stimulus functions are reversed such that responses to the originally positive stimulus are no longer reinforced and

responses to the previously negative stimulus are reinforced. Reversals are often embedded in a variety of instructional tasks and are used as one measure of cognitive inflexibility in individuals diagnosed with a neurodevelopmental disability. Discrimination reversal procedures are also widely used in examining the neural mechanisms of behavioral flexibility.

Historical Background

There is a rich literature on the acquisition of discrimination reversals in humans from the 1950s through the 1970s, and much of this literature evolved in two different areas of psychology. In the developmental literature, researchers were interested in whether typically developing children showed developmental differences on different types of discrimination shift tasks. This work was paralleled by the work of other researchers who were interested in whether individuals with intellectual disabilities would acquire learning set (presenting new discriminations until the participant learns each discrimination in one trial), and whether they would acquire a variety of discrimination shifts, with a particular interest in reversal shifts. Both of these literatures advanced different theories for discrimination shift behavior, and they have laid the foundation for modern investigations of reversal acquisition by individuals with autism spectrum disorder (ASD). Below, these literatures will be briefly summarized.

Discrimination Shifts in Typically Developing Children and Adults

Much of the work in typically developing populations has been reviewed by Stevenson (1972), who points out that a major interest was to determine if there were developmental differences in discrimination-shift ability related to language acquisition. This area of research was heavily influenced by the work of Kendler and Kendler (1959) and their subsequent studies. There is mixed evidence for the idea that there are developmental differences regarding the ability to perform reversal shifts. Initially, groups of older children or adults were compared to groups

of younger children on two types of transfer tasks: reversal and nonreversal. The original discrimination was between stimuli that differed along two dimensions, such as color and size, and the problem could be solved by responding on the basis of a dimensional cue (e.g., objects of a particular color were always positive regardless of size). For participants given a reversal transfer task, the same dimension as in the original problem was still relevant, but the valences of the stimuli were reversed (e.g., the choices of the previously positive color were no longer reinforced and choices of the previously negative stimulus were). By contrast, for the nonreversal procedure, the relevant stimulus dimension changed (e.g., the positive stimulus was no longer defined by color, but by size). Some studies reported that older children and adults were more likely to acquire the reversal task quickly compared to the nonreversal, but other studies found no difference in the rate of acquiring the different shift tasks. Some studies investigated the role of verbal mediation on shift tasks by instructing some participants to verbalize the basis of their response and comparing them to participants who were not instructed to verbalize. On balance, these studies did not support the role of verbal mediation: the performance of groups instructed to verbalize did not differ from those not given such instructions and sometimes children would verbalize the correct response but simultaneously respond to the incorrect stimulus. Finally, giving participants overtraining on the original discrimination tended to facilitate acquisition of a reversal when visual stimuli were used but not when spatial stimuli were used. Moreover, it was found that a participant's dimensional preference exerted a strong influence on the rate of acquisition of a reversal.

Discrimination Shifts in Individuals with Intellectual Disabilities (ID)

The behavior of individuals with severe intellectual disability has long been described as rigid, perseverative, and inflexible and tests of discrimination reversals were thought to be an index of such rigidity. The reported studies tend to be group designs in which the comparison group was typically developing children matched on

mental age equivalent or IQ or a group of individuals with ID also matched on mental age equivalent or IQ. In comparison to typically developing peers, individuals with ID were reported to show deficits on discrimination reversal tasks in some studies, but not in others. Zeaman and House reported a substantial amount of work on the discrimination abilities of individuals with ID (e.g., Zeaman and House 1979). In these studies, acquisition of different types of discrimination-shift tasks were compared after learning an original discrimination to criterion. The individuals in these studies ranged in functioning level from moderate to profound disability, but specific diagnoses were rarely reported. Collectively, the results of this body of research indicated that these individuals were capable of acquiring discrimination reversal set (quicker acquisition of repeated reversal problems) of a spatial discrimination, that requiring differential responses to the two stimuli resulted in quicker acquisition of a reversal, and that prior training on a learning set task facilitated reversal acquisition. A novel finding from this literature, that contradicted the then-dominant learning theories of Hull and Spence, was the overtraining reversal effect – that providing overtraining on the original task facilitated subsequent reversal learning.

Current Knowledge

Relevance of Reversal Learning

In recent years, there has been a renewed interest in discrimination reversal learning in intellectual disabilities in general, and in autism specifically, for several reasons. First, discrimination reversals are a type of set-shifting, or the ability to alter behavior as a result of changes in the environment or context in which the behavior occurs. (Other examples of discrimination set-shifting include intra- and extradimensional shifts.) Set-shifting abilities in autism are of interest because they are part of a class of behaviors that are described as executive functioning abilities. Executive function is defined as a set of higher-order cognitive skills that require processing and integration of information from a range of both internal and

external sources. Other examples of executive functioning include planning, working memory, and inhibitory control. Some researchers have suggested that deficits in executive function may underlie the social and cognitive impairments frequently observed in individuals diagnosed with an ASD, but this does not appear to be a core deficit in the disorder. In addition, one of the three factors leading to an autism diagnosis is an excess of restricted, repetitive behaviors or interests, which may reflect deficits in inhibitory control. Performance on discrimination reversal shifts can be used to index deficits in inhibitory control because individuals are required to withhold (or inhibit) responses to stimuli when there is a history of responding to those stimuli being reinforced. Cognitive inflexibility is measured as the number of perseverative errors (i.e., repeated errors prior to switching one's selection) and regressive errors (i.e., a failure to maintain a switched response) during a reversal (Miller et al. 2015). Some researchers have suggested that an autism diagnosis may distinguishable from other diagnoses (such as attention-deficit hyperactivity disorder [ADHD] or Down syndrome) on the pattern on results across a variety of executive functioning tests, including set-shifting (e.g., Chantiluke et al. 2015). Moreover, there are reported positive correlations between set-shifting deficits and measures of repetitive and restricted interests (Yerys et al. 2009).

Performance by individuals with autism and/or intellectual disabilities on discrimination reversals is also of interest because rapid reversal responding is a prerequisite behavior for conditional discrimination tasks such as matching-to-sample (MTS) and for other tasks that tap relational learning (Dube and Serna 1998). Special education classrooms often use MTS procedures to teach students the relation between items (e.g., to teach the relation between a printed word and an object) as well as some communication skills. In addition, MTS procedures are used in standardized assessments (e.g., the Peabody Picture Vocabulary Test [Dunn and Dunn 2007] and Wechsler Intelligence Scale for Children [Wechsler 1991]). In MTS, a stimulus, the sample, is presented and after an observing period, two or

more additional stimuli, the comparisons, are presented. The individual is required to choose the comparison that goes with, or matches, the sample. Across teaching trials, the sample stimulus changes, but the same comparison options are presented. For example, on some trials, the sample may be the printed word CAT and on others it may be the printed word DOG, and the comparisons may be photographs of a cat and dog. Responses to the photo of the cat are reinforced when the printed word CAT is the sample, and responses to the photo of the dog are reinforced when the printed word DOG is the sample. In order to successfully solve this task, an individual is required to make rapid reversals across trials presented in a teaching session – whether a response to a given comparison stimulus will be reinforced changes multiple times depending on what sample is presented. Individuals with intellectual disability often have difficulty acquiring MTS discriminations, even when errorless teaching procedures are used, and the requirement for rapid, within-session reversals may be one contributing factor.

Reversal Learning Assessment

Matched comparisons. As a result of the differing contexts in which reversal learning is studied, there are several different methods that have been used to assess reversal shifts in individuals with autism and/or intellectual disability. One method has been the intradimensional/extradimensional (ID/ED) shift test from the Cambridge Neuropsychological Test Automated Battery (CANTAB; Cambridge Cognition 1996). The ID/ED shift test is presented on a computer and includes a series of nine stages in which discrimination problems of increasing difficulty are presented; four of the nine stages involve stimulus function reversals, whereas other stages involve other types of set-shifting. The dependent variables measured in this task include the number of errors to criterion per stage, the number of perseverative errors per stage, and the stage completed. Frequently, performance on this task by individuals with autism who do not have intellectual disability is compared to one or more matched comparison groups. Often, performance on the reversal stages of the task is not reported, but some studies have

included analysis from reversal stages; the data from these stages are mixed in that some studies have reported that individuals with autism make more errors on extradimensional reversals than do matched the comparison groups, whereas other studies have found no between-group differences.

Reversal shifts have also been assessed using a spatial discrimination task. In this task, an object, such as a toy, is hidden beneath one of two cups that are placed side-by-side on a table in front of the participant. On each trial, the object is hidden in the same location (e.g., always beneath the left cup). For reversal trials, the object is then switched to the alternative location without a clue to the participant. There have been mixed findings from this task; some studies have reported that children with autism make more preservative errors than comparison groups, whereas others have not found significant group differences. One contributing factor to this difference may be chronological age, as the participants in the studies that have not found deficits were younger than those in the studies that did report deficits.

Leung and Zakzanis (2014) conducted a meta-analysis of effect sizes of 72 studies investigating executive function in individuals with ASD that used a range of tests, many of which included discrimination reversals. In general, the only measure that exceeded their clinical marker threshold was an indirect measure (a behavior rating scale that was based on an informant response form assessing executive functioning in the home environment). One important difference between the rating scale and the more common direct measures (i.e., the variety of tasks that assess reversal learning) is that the former is able to capture the complexity of the natural environment in a way that the latter cannot. For example, reversal assessments conducted in a laboratory setting on a computer involve a level of instructional control that is not present when rapid reversal responding is required in the natural environment (e.g., during a conversation).

As noted above, when discrimination reversal learning is assessed in individuals with autism and no intellectual disability, there is mixed evidence for deficits relative to typically developing individuals. Recently, a few studies have been

reported in which discrimination training and reversal testing has been conducted under conditions in which not every correct response was reinforced (i.e., intermittent or probabilistic reinforcement conditions). Use of intermittent reinforcement in reversal tasks increases the overall complexity of both acquisition, initial reversal responding, and maintenance of reversal responding, which may render the task less susceptible to ceiling effects (D'Cruz et al. 2016). Because only a few studies have used this measure, it remains unclear as to whether individuals with ASD show consistent deficits; however, there are reports of deficits on intermittent reinforcement reversal tasks being linked to clinical measures of restricted and repetitive behavior.

Small-n designs. The ability of individuals with moderate to severe intellectual disability and/or autism to make rapid reversals has also been assessed in studies that did not include matched-group comparisons. In these studies, the focus was to teach behaviors that are prerequisite to MTS performance. Some studies involved within-session reversals of stimulus discriminations presented on a computer touchscreen and others studies involved between-session reversals of three-dimensional object stimuli. Despite the differences in procedures across the various studies, similar results have been found: a small number of individuals eventually are eventually able to make rapid reversals to the point of demonstrating reversal learning-set (i.e., faster acquisition of each successive reversal problem), whereas most are not (e.g., Lionello-DeNolf et al. 2008). Further, failures to observe reversal learning occurred even when errorless teaching procedures were used to teach both the original discrimination problem and its reversal. These studies can be contrasted with recent work with typically developing preschoolers (Postalli et al. 2015) and toddlers (de Sousa et al. 2015), the majority of whom acquired rapid reversal learning performances.

Finally, the acquisition of multiple reversals in the context of yoked-reversals in individuals with autism and intellectual disability is of interest because it may lead to the formation of functional equivalence (Sidman et al. 1989), one measure of

symbolic functioning. In a yoked-discrimination-reversal task, individuals are required to concurrently solve two or more simple simultaneous discriminations. Different pairs of stimuli appear on irregularly alternating trials (e.g., A1 and A2, B1 and B2, C1 and C2). Initially, one member of each pair is designated as positive (A1, B1, C1), and the other negative (A2, B2, C2). In reversal sessions, reinforcement contingencies are reversed for all stimulus pairs, and multiple yoked-reversals are conducted across sessions. On the first trial of a reversal session, individuals typically make an error (e.g., select stimulus A1 although now A2 is now the positive stimulus). Functional equivalence is indicated if, after experiencing an error with one stimulus set, the individual immediately selects the now positive (formerly negative) stimulus from all remaining stimulus pairs (e.g., B2, C2). While the number of studies reporting functional equivalence in individuals with autism and/or intellectual disabilities is small, the general pattern of results is similar to that of studies involving reversals of a single discrimination: a small number of individuals have developed functional equivalence (e.g., Santos et al. 2017), but most have not (e.g., Lionello-DeNolf et al. 2008).

Neurobiological Correlates

The neural correlates of reversal learning have been investigated in both typically developing individuals and individuals with neurodevelopmental disabilities, including ASD and ADHD. In these investigations, participants are asked to complete one or more tasks assessing reversal learning while inside an fMRI scanner, and activation of different neural systems is measured before, during, and/or after the task(s). For typically developing individuals during reversal tasks, brain regions associated with reward processing and acquisition of stimulus-response associations have been implicated (see Izquierdo et al. 2017, for a review). There tends to be increased activity in the medial orbitofrontal cortex (OFC), medial prefrontal cortex (mPFC), anterior cingulate cortex (ACC), ventral striatal regions, and the precuneus, but understanding of the roles of these (and other) areas is continuing to

evolve. The OFC may be related to the amount of discrimination training that has occurred prior to experiencing reversals and in learning about the value of trial outcomes, the ACC may be related to error detection, and the mPFC may be activated in situations that demand increased levels of attention (e.g., difficult discriminations or tracking multiple contingency reversals simultaneously).

A few studies have directly compared brain activation in (primarily male) individuals with ASD and matched controls. Reduced activation in a variety of brain areas related to decision-making and response planning in individuals with autism has been noted, including the ventral striatum, ACC, thalamus, premotor cortex, precuneus, dorsolateral prefrontal cortex, and the basal ganglia. There have also been noted increases in activation to the inferior frontal cortex and the parietal lobe (Chantiluke et al. 2015). Interestingly, individuals with autism may not differ from matched controls on behavioral measures of reversal learning even though the two groups may show different patterns of brain activation.

Finally, investigations of the neurochemical correlates of reversal learning have been conducted in nonhuman animals. These studies indicated that reduced levels of serotonin in the cortex is associated with increases in perseverative errors, and reduced levels of dopamine in the striatum is associated with increases in non-perseverative errors. The role of glutamate has also been investigated, but the data are conflicting. For all three neurotransmitters, there is some evidence that effects may be modulated by particular receptor subtypes.

Future Directions

Given the marked variability across investigations of reversal learning, more study is warranted in order to make more definitive statements about the capability of individuals with autism and/or intellectual disability on these tasks sorts of tasks. There are several variables that may contribute to the observed variability across studies, including the age of the

participants, the severity of intellectual disability, the level of verbal ability, task demands unrelated to reversals (e.g., higher-order cognitive skills), and the severity of symptoms in the domain of restricted, repetitive behavior. In particular, more studies that include younger individuals, female participants, and individuals with intellectual disability are needed. Given the ubiquity of stimulus reversals in teaching procedures and standardized assessments and the importance of stimulus reversals for communication and other forms of symbolic functioning, research is needed to determine which variables are critical for rapid reversal performance and the maintenance of that performance. Moreover, given the relative paucity of work investigating neural correlates of reversal learning in ASD, particularly in children and individuals with ID, additional work should be conducted. Finally, there have been a few reports of reversal learning in Pavlovian fear conditioning tasks (South et al. 2012), and performance has been correlated with restricted and repetitive behavior but not anxiety. Additional work in this area is needed before firm conclusions can be made. Collectively, work in these different areas could lead to interventions that encourage reversal learning and perhaps increase cognitive flexibility and symbolic communication in individuals with ASD.

See Also

- ▶ [CANTAB](#)
- ▶ [Executive Function \(EF\)](#)
- ▶ [Reinforcement](#)
- ▶ [Repetitive Behavior](#)
- ▶ [Symbolic Behavior](#)
- ▶ [Wechsler Preschool and Primary Scale of Intelligence](#)

References and Reading

Chantiluke, K., Barrett, N., Giampietro, V., Brammer, M., Simmons, A., Murphy, D. G., & Rubia, K. (2015). Inverse effect of fluoxetine on medial prefrontal cortex activation during reward reversal in ADHD and autism. *Cerebral Cortex*, 25, 1757–1770.

- Cognition, C. (1996). *CANTAB*. Cambridge: Cambridge Cognition.
- D'Cruz, A.-M., Mosconi, M. W., Ragozzino, M. E., Cook, E. H., & Sweeney, J. A. (2016). Alterations in the functional neural circuitry supporting flexible choice behavior in autism spectrum disorders. *Translational Psychiatry*, 6, 1–9.
- de Sousa, N. M., Gil, M. S., & McIlvane, W. J. (2015). Discrimination and reversal learning by toddlers aged 15–23 months. *The Psychological Record*, 65, 41–47.
- Dube, W. V., & Serna, R. W. (1998). Re-evaluation of a programmed method to teach generalized identity matching to sample. *Research in Developmental Disabilities*, 19, 347–379.
- Dunn, L. M., & Dunn, D. M. (2007). *Peabody picture vocabulary test* (4th ed.). Minneapolis: Pearson.
- Izquierdo, A., Brigman, J. L., Radke, A. K., Rudebeck, P. H., & Holmes, A. (2017). The neural basis of reversal learning: An updated perspective. *Neuroscience*, 345, 12–26.
- Kendler, T. S., & Kendler, H. H. (1959). Reversal and nonreversal shifts in kindergarten children. *Journal of Experimental Psychology*, 58, 56–60.
- Leung, R. C., & Zakzanis, K. K. (2014). Cognitive flexibility in autism spectrum disorders: A quantitative review. *Journal of Autism and Developmental Disorders*, 44, 2628–2645.
- Lionello-DeNolf, K. M., McIlvane, W. J., Canovas, D. S., de Souza, D. G., & Barros, R. S. (2008). Reversal learning set and functional equivalence in children with and without autism. *The Psychological Record*, 58, 15–36.
- Miller, H. L., Ragozzino, M. E., Cook, E. H., Sweeney, J. A., & Mosconi, M. W. (2015). Cognitive set shifting deficits and their relationship to repetitive behaviors in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45, 805–815.
- Postalli, L. M. M., Canovas, D. S., & de Souza, D. G. (2015). Simple discrimination and reversal learning sets in typically developing young children. *The Psychological Record*, 65, 411–423.
- Santos, E. A. L., Nogueira, C. B., Queiroz, L. L., & Barros, R. S. (2017). Equivalence class formation via class-specific consequences in children diagnosed with autism spectrum disorder. *Trends in Psychology*, 25, 831–842.
- Sidman, M., Wynne, C. K., Maguire, R. W., & Barnes, T. (1989). Functional classes and equivalence relations. *Journal of the Experimental Analysis of Behavior*, 52, 261–274.
- South, M., Newton, T., & Chamberlain, P. D. (2012). Delayed reversal learning and association with repetitive behavior in autism spectrum disorder. *Autism Research*, 5, 398–406.
- Stevenson, H. W. (1972). *Children's learning*. New York: Appleton-Century-Crofts.
- Wechsler, D. (1991). *Wechsler intelligence scale for children* (3rd ed.). San Antonio: The Psychological Corporation.

- Yerys, B. E., Wallace, G. L., Harrison, B., Celano, M. J., Giedd, J. N., & Kenworthy, L. E. (2009). Set-shifting in children with autism spectrum disorders: Reversal shifting deficits on the intradimensional/extradimensional shift test correlate with repetitive behaviors. *Autism: The International Journal of Research and Practice*, 13, 523–538.
- Zeaman, D., & House, B. J. (1979). A review of attention theory. In N. R. Ellis (Ed.), *Handbook of mental deficiency, psychological theory, and research* (2nd ed., pp. 63–120). Hillsdale: Erlbaum.

Reviva

► [Naltrexone](#)

Revised Knox Preschool Play Scale (RKPPS)

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administered in environments familiar to the child. The scale includes two 30-min observations of the child in different settings, one indoor and one outdoor. The test developer, S. Knox, has also indicated that two 15-min sessions may also be acceptable (see Jankovich et al. 2008). The scale consists of 4 dimensions and 12 categories of play behaviors, from which the therapist develops a “play profile.” This information is useful for assessing potential disabilities and planning appropriate interventions.

The four dimensions included in the RKPPS are the following: (1) space management, (2) material management, (3) pretense/symbolic, and (4) participation. Within these dimensions exist the 12 categories of play behaviors, also referred to as “factors.” Under “space management” exist the (1) gross motor and (2) interest factors. Within “material management” exist the factors of (3) manipulation, (4) construction, (5) purpose, and (6) attention. Under “pretense/symbolic” are the factors of (7) imitation and (8) dramatization and under “participation,” (9) type, (10) cooperation, (11) humor, and (12) language.

The RKPPS is scored in terms of ages: the 4 dimension ages and 12 category ages yield an overall play age. The developmental age estimates are divided into 6-month increments between the ages of 0 and 36 months (e.g., an estimate for a dimension age could be 6–12 months) and 12-month increments between the ages of 36–72 months (e.g., 48–60 months). Children are scored only according to demonstrated skills and not skills that are emerging, as per supplemental instructions from the authors.

Synonyms

[PPS](#)

Description

The RKPPS is a standardized instrument designed to evaluate play in children and correlate it to an approximate developmental age. It can be used to assess children aged birth to 72 months and is

Historical Background

Assessing play is simultaneously a complex and important task for service providers attempting to appropriately understand child-specific developmental vulnerabilities and skills in a manner that can be utilized to quantify current functioning and guide intervention services and techniques. Historically, practitioners in the field of occupational therapy have used a number of play assessments (e.g., Parten Social Play Hierarchy (Parten

1932), Play History (Takata 1969)) that examine certain components of play (i.e., imitation, creativity, rule organization) but do not yield results in terms of an overall developmental outcome or age. The Preschool Play Scale (Bledsoe and Shepherd 1982) was designed to examine play in a standardized manner and provide specific developmental markers of import. In 1997, this scale was revised by Knox to the current version, the Revised Knox Preschool Play Scale (RKPPS) (Knox 1997). Knox had, in 1974, developed “A Play Scale” (Knox 1974, 2005) to assist in determining developmental play ages, and this measure had several advantages: it assessed children in their natural environments and therefore catered to children who have difficulty conforming to standardized measures, all while delivering a developmental approximation. Knox’s revisions to the Preschool Play Scale included changes to the scoring scale and descriptor clarity. However, despite these modifications, the unrevised Preschool Play Scale continues to be used more frequently than the RKPPS due to a lack of reliability and validity data on the latter.

Psychometric Data

Extensive psychometric data regarding the use of this instrument within diverse samples of individuals with autism and other developmental disorders is not available. Recent interrater reliability analyses from Jankovitch et al. (2008) showed an 87% agreement between raters for the overall play age on the RKPPS when the scored ages were within 8 months of each other. In the same analysis, the interrater agreement rates for the play ages within fewer than 6 months of each other in the four domains ranged from 63.2% to 81.6%. Rates reached 91.7–100% agreement for the four domains when the age range expanded to a 12-month difference. When separated into age levels, the two raters agreed within the same range 81.8–100% of the time on all 12 RKPPS categories. In terms of construct validity, Jankovich et al. (2008) also addressed the RKPPS’s ability to measure the theoretical construct of “play.” By examining the scores of typically developing

children and comparing their chronological ages with the calculated play ages, they concluded that the construct validity of RKPPS was supported. They noted, however, that the older age groups were more accurate in the matches than the younger, 36–47-month age group, where 6 of the 14 children had play scores higher than their actual chronological ages.

Clinical Uses

The RKPPS is used clinically in the assessment of play in children ages 0–72 months in order to determine a developmental play age, screen for possible developmental concerns, and develop intervention programming. The measure is administered by trained examiners, generally occupational therapists. The recent reliability data suggest that scores up to 8 months below or above the child’s actual chronological age indicate that, without other factors or concerns, the child’s performance should be considered within typical ranges. The RKPPS has not yet been validated in a sample of children with a range of developmental disabilities or from varying socioeconomic, geographical, and cultural groups, although steps have recently been taken in that direction, notably in Brazil, where a Portuguese adaptation of RKPPS was preliminarily tested (see Pacciulio et al. 2010). Additionally, the RKPPS has been used in assessing differences in play between children with and without developmental coordination disorder (Kennedy-Behr et al. 2013).

See Also

- ▶ [Autism Diagnostic Observation Schedule](#)
- ▶ [Childhood Autism Rating Scale](#)
- ▶ [Communication and Symbolic Behavior Scale](#)

References and Reading

- Bledsoe, N., & Shepherd, J. (1982). A study of reliability and validity of a Preschool Play Scale. *American Journal of Occupational Therapy*, 36, 783–788.

- Jankovich, M., Mullen, J., Rinear, E., Tanta, K., & Deitz, J. (2008). Revised Knox Preschool Play Scale: Interrater agreement and construct validity. *American Journal of Occupational Therapy*, 62, 221–227.
- Kennedy-Behr, A., Rodger, S., & Mickan, S. (2013). A comparison of the play skills of preschool children with and without developmental coordination disorder. *OTJR: Occupation, Participation and Health*, 33, 198–208.
- Knox, S. (1974). A play scale. In M. Reilly (Ed.), *Play as exploratory learning* (pp. 247–266). Beverly Hills: Sage.
- Knox, S. (1997). Development and current use of the Knox Preschool Play Scale. In L. D. Parham & L. S. Fazio (Eds.), *Play in occupational therapy for children* (pp. 35–51). St. Louis: Mosby Year Book.
- Knox, S. (2005). Play. In J. Case-Smith (Ed.), *Occupational therapy for children* (5th ed., pp. 571–586). St. Louis: Mosby.
- Pacciulio, A. M., Pfeifer, L. I., & Santos, J. L. (2010). Preliminary reliability and repeatability of the Brazilian version of the Revised Knox Preschool PlayScale. *Occupational Therapy International*, 17, 74–80.
- Parten, M. B. (1932). Social participation among preschool children. *Journal of Abnormal and Social Psychology*, 28, 1243–1269.
- Takata, N. (1969). The play history. *American Journal of Occupational Therapy*, 23, 314–318.

Reward

► Reinforcer

Key Auditory Verbal Learning Test (Rey AVLT)

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Synonyms

Auditory verbal learning test; AVLT; RAVLT

Description

The Rey Auditory Verbal Learning Test (RAVLT) provides a standardized procedure to

evaluate a range of aspects of verbal learning and memory of supra-span lists of words. The test requires recalling as many words as possible in any order from a standard list of 15 unrelated words read out by the examiner. This procedure is repeated for four further trials, the words being presented in the same order on each trial. The words all have either one or two syllables, are high in imagery, and have a Thorndike-Lorge frequency of at least 50 per million. On each trial, the examiner records the words recalled, including repetitions and intrusions of words not present in the study list. Different authors have used a variety of procedures following Trial 5. In Rey's (1941) original version of the test, a short story containing the 15 studied words was presented, with a request to identify the studied words in the story. However, more recent versions of the test (e.g., Lezak et al. 2004) omit this step and proceed directly to the presentation and testing of a second list of words (Trial 6) followed in Trial 7 by a request to recall the original list. Many researchers also test delayed recall of the original list 20 min after the initial five learning trials (Trial 8) immediately followed by a 50-word recognition trial (Trial 9). Some authors (e.g., Vakil and Blachstein 1997) have added an additional trial in which the 15 words of the first-presented list were presented, written in a different order from that in which they were first presented. The task was to reorder the words according to the original sequence. A set of alternative word lists are available for use as parallel versions of the original list used in Trials 1–5, as well as of the second-learned list and the story or word list used to test recognition. These parallel forms permit repeated testing in order to measure changes in memory performance over time and can be found in Schmidt (1996). On all trials, the examiner keeps a detailed record of the words recalled or recognized, including repetitions, new words, and intrusions from earlier-learned lists.

The instructions for administering the test vary somewhat in different versions and experimental investigations, but all versions make explicit that the test is one of memory and learning and that the aim is the accurate recall (or recognition, where

appropriate) of as many as possible of the presented words in any order.

A wide range of measures can be obtained from the RAVLT. Norms are available for many of these, but the raw data can also be utilized to compare different groups of participants or to evaluate performance over time or in response to particular interventions. The following sections list the most widely used measures in the literature. The terms T1 through T9 refer to the total number of correct items in the nine testing trials described above.

A large number of *derived measures* can be obtained from the data obtained on the nine individual trials. *Learning rate* (T5–T1) provides an overall measure of how much additional material is retained on the four trials following the initial presentation. This measure can be further broken down by considering performance on the *learning curve*, which looks at the patterning of performance on each of the trials T1, T2, T3, T4, and T5. Summing the numbers of correct items across these five trials yields a measure of *total learning*. In order to control for the effect of initial memory performance on this measure, Ivnik et al. (1990) advocate correcting this measure by subtracting from it five times the score on T1 thereby yielding a *corrected total learning* score. An index of *best learning* is provided by the score on T5, and a *learning rate ratio* is obtained by dividing T1 by T5. An index of *delayed recall* is provided by the score on T8.

As well as measures of learning and recall, indices of underlying learning processes can also be obtained. Difference and ratio measures of *proactive interference* can be calculated by respectively subtracting T6 from T1 or by dividing T6 by T1. Similarly, difference and ratio measures of *retroactive interference* can be obtained by subtracting T7 from T5 and dividing T7 by T5, respectively. Difference and ratio measures of *forgetting rate* can also be obtained by subtracting T8 from T5 or by dividing T8 by T5. A measure of *freedom from distraction* is obtained by subtracting T6 from T5.

Further information on the dynamics of learning and memory in the test can be obtained from a

consideration of *additions*, which is the sum of new words in Trial N that are not recalled in Trial N-1, and *omissions*, which is the sum of words not recalled in Trial N that were recalled in Trial N-1. *Intrusions* – non-studied words that appear in recalled lists and *repetitions*, words that are repeated in the same recall trial – can also be recorded.

If a recognition memory trial (T9) is included, a simple measure of *recognition* is obtained by subtracting false alarms (FA, the number of non-studied words indicated as having been studied) from the number of hits (correctly recognized study words). Vakil and Blachstein (1997) have also suggested using a *nonparametric signal detection measure of recognition* using the formula $[5 (1 + \text{Hits} - \text{FA})]$. These authors also suggest computing difference and ratio versions of *retrieval efficiency* by subtracting T8 from T9 or dividing T8 by T9, respectively.

Researchers have also derived a number of other measures to evaluate the dynamics and organization of learning over the test. For example, Lezak (1983) suggests that Trial 1 performance usually matches an independently measured digit span score and that differences between the two may give clues to attentional, motivational, or emotional difficulties. The shape of the learning curve can indicate learning difficulties, especially when associated with a poor delayed recall score (Bishop et al. 1990). Altered serial position effects are also seen. Typical, healthy individuals recall more of the first and last items of a list and fewer of the middle items. These *primacy* and *recency* effects are maintained over trials for healthy individuals but are attenuated in individuals with lesions of the frontal lobes (Eslinger and Grattan 1994).

Some researchers have also investigated the *subjective organization* – the extent to which an individual reorganizes order of the items in a way that differs from the presented order – of recall across trials of the RAVLT. Diminished subjective organization has been reported in learning-disabled children (O'Donnell et al. 1988), children with specific language impairment (Records et al. 1995), and patients with frontal lobe damage

(Eslinger and Grattan 1994) but not in high-functioning adults with ASD (Bowler et al. 2009).

A potential limitation of the RAVLT in comparison with other verbal learning tests such as the California Verbal Learning Test (Delis et al. 2000) is that the studied words are not strongly semantically related. Although this results in the various measures described above being relatively uncontaminated by semantic organization, it does not permit measurement of semantic clustering. Tests, such as the CVLT, which contain lists made up of words from more than one category or different categories enable the extent to which semantically related material can be clustered in recall.

Historical Background

The evaluation of memory difficulties forms a major part of the work of clinicians involved in the assessment of individuals with brain damage, psychiatric or neuropsychiatric syndromes, or with developmental disorders. It was in this context that the Swiss psychologist, Édouard Claparède, first formalized a test involving the recall of 15 unrelated words, together with a procedure for administration of the test (Claparède 1919, see Boake 2000). As Boake points out, this makes the auditory verbal learning test one of the oldest mental measurements in continuous use. Another Swiss psychologist, André Rey, further developed the standardization of this test, which now bears his name. Rey's intention was to evaluate his neurological patients' memory performance in relation to the range of values that would typically be expected for similar individuals selected on the basis of parameters such as gender, age, educational level, socioeconomic status, ethnicity, etc. Since the development of Rey's original version of the test, it has been translated into several languages including English.

Psychometric Data

A wide range of norms are available for use with the RAVLT, which are comprehensively reviewed

in Schmidt (1996), as well as in Mitrushina et al. (1999) and Spreen and Strauss (1998). Schmidt is at pains to point out that different sets of norms have often utilized slightly different word lists and procedures. This means that care needs to be taken to ensure that the word lists and procedures used match those of the chosen normative data. It is also important to be aware that normative data are not always available for all possible measures that can be derived from the test. Schmidt (1996) also provides a set of metanorms obtained by pooling the data from methodologically sound normative studies published up to that date. In addition to the original French language norms published by Rey (1958), norms have also been published in other languages including Dutch (Van der Elst et al. 2005), Greek (Messinis et al. 2007), and Hebrew (Vakil et al. 2010). Several studies have also published data on age-related changes in RAVLT performance in both children and adults. In addition to those reviewed in Schmidt (1996) are those on Dutch children by van den Burg and Kingma (1999), on older English-speaking adults (Steinberg et al. 2005) and on Hebrew-speaking older adults (Vakil and Blachstein 1997).

Test-retest reliability has been assessed by a number of studies, and the existence of parallel forms of the test permits estimations that are relatively uncontaminated by practice effects. Schmidt (1996) reviews these studies and concludes that performance is more stable on later than on earlier trials and that reliability is higher for the recall than for the recognition trials. Good reliability is also reported for measures that are derived from performance on individual trials.

Given that it tests multi-trial recall of verbal material, the *face* and *content validity* of the RAVLT are undoubtedly high. Its *concurrent validity* as measured by how well it correlates with performance on other tests of memory is also high. Schmidt's (1996) review of validity studies concludes that factor analytic studies of memory that have included the RAVLT have generally shown that it loads with other measures of verbal memory as well as with measures of general verbal ability but not with measures of motor or visuospatial ability.

Several investigators have investigated the *factor structure* of the test, with different investigators identifying different numbers of factors, which in turn correlate with different subsets of the test items. Talley (1986) reports two factors (short-term memory and long-term memory), whereas Vakil and Blachstein (1993), using Hebrew-speaking participants, report three (acquisition, storage, and retrieval). More recently, Baños et al. (2005) reported three correlated factors (general verbal learning, auditory attention, and inaccurate recall) in a group of adults with spinal cord injury.

Clinical Uses

As befits a test whose origins lie in the area of clinical evaluation, the RAVLT has been utilized with a wide range of clinical conditions. These include memory impairments that result either from neurodegenerative disease, brain injury, or developmental disability. It has also been utilized to evaluate cognitive changes in healthy aging as well as at different stages of diseases, such as HIV infection, which are known to produce cognitive impairments. These studies are reviewed by Schmidt (1996) as well as by Lezak et al. (2004) and by Strauss et al. (2006). To date, only one study has used the test with individuals on the autism spectrum. Bowler et al. (2009) found no overall deficit in recall or learning in a group of high-functioning adults with autism spectrum disorder (ASD). Although these participants were found to have typical serial positions on Trial 1, by Trial 5 their primacy effect was found to lag significantly behind that of a typically developed comparison group. The authors speculate that this patterning of serial position effects over trials may result from a more item-specific strategy driven by greater attention to the perceptual rather than the conceptual aspects of the studied words.

See Also

► [Memory Assessment](#)

References and Reading

- Baños, J. H., Elliott, T. R., & Schmitt, M. (2005). Factor structure of the Rey auditory verbal learning test in adults with spinal cord injury. *Rehabilitation Psychology, 50*, 373–380.
- Bishop, J., Knights, R. M., & Stoddart, C. (1990). Rey auditory-verbal learning test: Performance of English and French children aged 5 to 16. *The Clinical Neuropsychologist, 4*, 133–140.
- Boake, C. (2000). Édouard Claparède and the auditory verbal learning test. *Journal of Clinical and Experimental Neuropsychology, 22*, 286–292.
- Bowler, D. M., Limoges, E., & Mottron, L. (2009). Different verbal learning strategies in autism spectrum disorder: Evidence from the Rey auditory verbal learning test. *Journal of Autism and Developmental Disorders, 39*, 910–915.
- Claparède, E. (1919). Percentilage de quelques tests d'aptitude. *Archives de Psychologie, 17*, 325–334.
- Delis, D. C., Kaplan, E., Kramer, J. H., & Ober, B. A. (2000). *California verbal learning test—Second edition (CVLT-II)*. San Antonio: Psychological Corporation.
- Eslinger, P. J., & Grattan, L. M. (1994). Altered serial position learning after frontal lobe lesions. *Neuropsychologia, 32*, 729–739.
- Ivnik, R. J., Malec, J. F., Tangalos, E. G., Peterson, R. C., Kokmen, E., & Kurland, L. T. (1990). The auditory-verbal learning test (AVLT): Norms for ages 55 and older. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 2*, 304–312.
- Lezak, M. D. (1983). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
- Messinis, L., Tsakona, I., Malefaki, S., & Papathanasopoulos, P. (2007). Normative data and discriminant validity of Rey's verbal learning test for the Greek adult population. *Archives of Clinical Neuropsychology, 22*, 739–752.
- Mitrushina, M. N., Boone, K. B., & D'Elia, L. S. (1999). *Handbook of normative data for neuropsychological assessment*. New York: Oxford University Press.
- Mitrushina, M., Boone, K. B., Razani, J., & D'Elia, L. F. (2005). *Handbook of normative data for neuropsychological assessment* (2nd ed.). New York: Oxford University Press.
- O'Donnell, J. P., Radtke, R. C., Leicht, D. J., & Caesar, R. (1988). Encoding and retrieval processes in learning-disabled, head-injured, and non-disabled young adults. *The Journal of General Psychology, 115*, 355–368.
- Records, N. L., Tomblin, J. B., & Buckwalter, P. R. (1995). Auditory verbal learning and memory in young adults with specific language impairment. *The Clinical Neuropsychologist, 9*, 187–193.
- Rey, A. (1941). L'examen psychologique dans le cas d'encephalopathie traumatique. *Archives de Psychologie, 28*, 286–340.

- Rey, A. (1958). *L'Examen clinique en psychologie*. Paris: Presses Universitaires de France.
- Schmidt, M. (1996). *The Rey auditory verbal learning test*. Los Angeles: Western Psychological Services.
- Spreen, O., & Strauss, E. (1998). *A compendium of neuropsychological tests: Administration, norms, and commentary* (2nd ed.). New York: Oxford University Press.
- Steinberg, B. A., Bieliauskas, L. A., Smith, G. E., Ivnik, R. J., & Malec, J. F. (2005). Mayo's older Americans normative studies: Age- and IQ-adjusted norms for the auditory verbal learning test and the visual spatial learning test. *The Clinical Neuropsychologist*, 19, 464–523.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). New York: Oxford University Press.
- Talley, J. L. (1986). Memory in learning disabled children: Digit span and the Rey auditory verbal learning test. *Archives of Clinical Neuropsychology*, 1, 315–322.
- Vakil, E., & Blachstein, H. (1993). Rey auditory verbal learning test: Structure analysis. *Journal of Clinical Psychology*, 49, 883–890.
- Vakil, E., & Blachstein, H. (1997). Rey AVLT: Developmental norms for adults and the sensitivity of different memory measures to age. *The Clinical Neuropsychologist*, 11, 356–369.
- Vakil, E., Greenstein, Y., & Blachstein, H. (2010). Normative data for composite scores for children and adults derived from the Rey auditory verbal learning test. *The Clinical Neuropsychologist*, 24, 662–677.
- Van den Burg, W., & Kingma, A. (1999). Performance of 225 Dutch school children on Rey's auditory verbal learning test (AVLT): Parallel test-retest reliabilities with an interval of 3 months and normative data. *Archives of Clinical Neuropsychology*, 14, 545–599.
- Van der Elst, W., Van Boxtel, M. P. J., Van Breukelen, G. J. P., & Jolles, J. (2005). Rey's verbal learning test: Normative data for 1,855 healthy participants aged 24–81 years and the influence of age, sex, education, and mode of presentation. *Journal of the International Neuropsychological Society*, 11, 290–302.

Reynell Developmental Language Scales

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Synonyms

NRDLS

Description

The New Reynell Developmental Language Scales (NRDLS) is the current edition of the Reynell Developmental Language Scales. The NRDLS is a norm-referenced assessment that measures expressive and receptive language skills in children 2–7 years of age. It contains two scales – Verbal Comprehension and Production. The Verbal Comprehension Scale measures language understanding of nouns, verbs, and prepositions. It also measures a child's ability to follow simple and complex directions and provides information on comprehension of basic inferences. The Production Scale measures spoken language in the areas of syntax, vocabulary, and content.

Historical Background

The RDLS was initially created in the United Kingdom with normative data collected in Britain. The British version is in its fourth revision (NRDLS: The University of Reading Edition; Edwards et al. 2011). There is also an American version, published in 1990 (Reynell and Gruber 1990).

Psychometric Data

The American version has a normative sample of 600 children with data collected across the United States. The sample is representative of the United States ethnic composition, parental education, and geographic regions.

Clinical Uses

The test is quick to administer, taking less than 30 min to complete. The NRDLS uses both manipulatives and pictures, making it a good choice for children with developmental disabilities. It provides standard scores, percentile ranks, and age equivalents for both expressive and receptive language skills.

See Also

► [Language Tests](#)

References and Reading

Edwards, S., Letts, C., & Sinka, I. (2011). *The new Reynell developmental language scales*. London: GL Assessment Limited.

Reynell, J., & Gruber, C. (1990). *Reynell developmental language scales*. Los Angeles: Western Psychological Services.

RG7314

► [Balovaptan](#)

Right-Hemisphere Deficit Syndrome

► [Right-Hemisphere Syndrome](#)

Right-Hemisphere Developmental Learning Disability

► [Right-Hemisphere Syndrome](#)

Right-Hemisphere Learning Disability

► [Right-Hemisphere Syndrome](#)

Right-Hemisphere Syndrome

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Synonyms

[Developmental right-hemisphere syndrome](#); [Non-verbal learning disabilities \(NLD\)](#); [Right-](#)

[hemisphere deficit syndrome](#); [Right-hemisphere developmental learning disability](#); [Right-hemisphere learning disability](#); [Social-emotional processing disorder](#)

Short Description or Definition

Compromised functioning of the right hemisphere is associated with impairments in attention; non-verbal memory; visual-perceptual and visual-spatial abilities; and the expression, recognition, and integration of emotion and affective states. In adults, the behavioral and neuropsychological sequelae of *acquired* injuries to the right hemisphere of the brain (e.g., through stroke, tumor, or traumatic brain injury) are well documented and described. Several researchers have also endeavored to examine and describe a similar syndrome on a developmental basis. The name of the proposed disorder has varied with each account (see above).

In these accounts, individuals with social difficulties, a characteristic neuropsychological profile, academic challenges, and neurological signs consistent with right-hemisphere damage or dysfunction have been identified. The syndrome has been described in children with documented perinatal or early childhood brain damage (e.g., Voeller 1986) as well as in children with no obvious evidence of brain damage (e.g., no structural lesions or seizure disorder) (Gross Tsur et al. 1995; Rourke 1989). Rourke (1989) isolated the syndrome, referred to as nonverbal learning disability (NLD), in his research of children with learning disabilities; Gross Tsur et al. (1995) described a specific set of impairments in children referred to a clinical center for social, behavioral, and/or academic problems. Findings have also been presented in adolescents and adults; those for whom neurodiagnostic examination suggested early brain injury affecting the right hemisphere and inadequate development of the functions it normally subserves (Manoach et al. 1995; Weintraub and Mesulam 1983) and those with no history of acquired cerebral injury or evidence of structural abnormality (Tranel et al. 1987). In the latter instance, the

neuropsychological evidence was suggestive of right-hemisphere dysfunction and the pattern of cognitive and behavioral functioning was life-long; the constellation of symptomatology was considered to represent a developmental learning disability of the right hemisphere.

In sum, this syndrome is predicated on a neurobehavioral and psychological profile seen in individuals with right-hemisphere damage, but its manifestation does not necessitate a demonstrable lesion. A developmental disorder of the right hemisphere is also proposed; that is, a pattern of results and behavioral phenotype suggestive of right-hemisphere dysfunction in children, adolescents, and or adults who have no obvious structural brain damage. Rourke's model is somewhat distinctive, both for being more fully developed and for encompassing three dimensions and progressions in neurodevelopment ("down-up," "back-front," as well as "right-left"); the principal working hypothesis of this model is that the behavioral phenotype of NLD will be manifest to the extent that white matter (long myelinated fibers) in the brain is underdeveloped, damaged, or dysfunctional (Rourke 1989, 1995).

Clinical Expression and Pathophysiology

The clinical expression of this disorder is based on case reports defining the core symptoms to include:

- Emotional difficulties (e.g., perception and interpretation of the emotional states of others, nonverbal expression of emotion, depression)
- Impaired interpersonal skills (e.g., shy, withdrawn, isolated, poor peer relationships)
- Paralinguistic communication deficits (e.g., atypical prosody, reduced eye contact, diminished or exaggerated facial expression and body gestures, literal interpretations)
- Poor visual-spatial ability often in the context of average or superior verbal abilities
- Academic deficits, especially in arithmetic with better developed reading and spelling skills
- Left-sided neurological findings

Attention deficit hyperactivity disorder is reported to be part of the clinical picture in several instances (e.g., Gross Tsur et al. 1995; Manoach et al. 1995; Voeller 1986). Graphomotor problems are generally noted; difficulties copying and drawing geometric and representational designs are consistent with right-hemisphere dysfunction. Rourke reports improvement in the handwriting of children with NLD over time, with repeated practice.

In the work on NLD, which has spanned several decades, the content and dynamics have been detailed (Rourke 1989) and classification rules derived for 9- to 15-year-old children (Pelletier et al. 2001) and 7- and 8-year-old children (Drummond et al. 2005). (Please see ► [“Nonverbal Learning Disabilities \(NLD\)”](#) entry in this Encyclopedia for more information.)

Although not identified in the case reports above, additional impairments associated with right-hemisphere damage include:

- Left-side neglect
- Difficulty identifying relevant information
- Problems with conversational rules
- Confabulation
- Disorientation in time and direction
- Anosognosia (failure to recognize the symptom's of one's own illness)
- Prosopagnosia (inability to recognize familiar faces)

Evaluation and Differential Diagnosis

Neither right-hemisphere syndrome nor any of the related syndromes listed above are recognized in the DSM-IV-TR. The terms *right-hemisphere learning disability*, *developmental learning disability of the right hemisphere*, and *social-emotional learning disabilities* (Denckla 1983; Weintraub and Mesulam 1983) emerged from the neurological field and *nonverbal learning disability* (Rourke 1989) from the field of neuropsychology. In some instances, individuals with Asperger syndrome may show a similar pattern of neuropsychological strengths and weaknesses as identified in these conditions (Klin et al. 1995).

A comprehensive neuropsychological examination is recommended, assessing cognitive, behavioral, social-emotional, academic, and adaptive functioning. The developmental history is also important, especially if the evaluation is occurring at a later developmental period (e.g., middle school or high school years).

Differential diagnoses include ADHD, mood disorders, and personality disorders. As noted above, in several of the case reports (Gross Tsur et al. 1995; Manoach et al. 1995; Voeller 1986), the clinical presentation was also significant for attention deficit disorder, with or without hyperactivity. A relationship between right-hemisphere involvement, attention deficit disorder, and depression has been hypothesized by others (e.g., Brumback and Staton 1982) and there is evidence that attentional processes are subserved by right-hemisphere pathways. Rourke (1989) offers a developmental perspective, that is, the diagnosis of ADD in children with NLD is usually based on inattentiveness to visual and tactile stimuli in the very early school years. Much of the “teaching” that takes place in the earliest school years is visually mediated and requires “hands on” exercises, tapping areas of deficit in children with NLD. The inability to complete such tasks or process such information may be misinterpreted as a primary failure in attention. From this perspective, inattention is thought to be a secondary deficit that stems from more basic deficits in primary visual and tactile perception.

Rourke and colleagues have identified the following pattern in individuals with NLD: children with NLD are characterized during early childhood as presenting with some type of acting-out or other “externalized” disorder but are at risk for the development of “internalized” forms of psychopathology, such as anxiety and depression, in adolescence and adulthood. Depression was reported in the adolescents and adults in Manoach et al.’s sample who were selected on the basis of meeting criteria for a social-emotional processing disorder (SEPD).

Grace and Malloy (1992) identified patients in an adult psychiatric population with evidence of a right-hemisphere learning disability (RHL),

seeking to examine the contribution of this disorder to adult psychiatric presentations. Many of the adults with RHL presented with depression; however, their developmental histories were distinct from other patients with clinical depression. The test performance of individuals with major depression may yield visual-spatial deficits and deficits secondary to psychomotor retardation on timed nonverbal tasks. However, for the RHL individuals with comorbid depression, the developmental history was significant for persistent problems in childhood with visual-spatial tasks, attention, social isolation, which predated onset of affective symptoms. Moreover, the pattern of neuropsychological deficits remained stable over time and did not resolve with improvement of psychiatric symptoms.

In Grace and Malloy’s psychiatric sample, episodic dyscontrol and psychotic disorders as well as several instances of psychosexual developmental issues were noted in their RHL group. Given the overlapping clinical features, these adolescents/adults may be identified with schizoid personality disorder or schizoaffective disorder; Rourke speaks to the issue of diagnostic overshadowing as concerns the diagnosis of disorders along the schizophrenia continuum in individuals with NLD (see www.nld-bprourke.ca Q&A).

Treatment

Recommendations for treatment include providing modifications to instructional materials, accommodations for areas of impairment, and support strategies for enhancing development of areas of deficiency. Comprehensive recommendations are provided by Rourke (1989, 1995) and Thompson (1997) as pertains to NLD. Some general recommendations identified for individuals with right-hemisphere syndrome include the following:

- Help the individual learn how to interpret social cues that occur in everyday interpersonal interactions.
- Provide explicit teaching of nonverbal cues (both interpretation and expression).

- Avoid sarcasm and/or metaphors when speaking to the individual.
- Provide a consistent routine.
- Make expectations explicit (no “reading between the lines”).
- Break down tasks into small steps and provide straightforward, sequential verbal descriptions.
- Repeat directions as needed.
- Decrease distractions when communicating.
- Consider that in many instances the behavior difficulties demonstrated by these children reflect situations where their processing capacities are overwhelmed or where they do not know the correct way to respond.

See Also

- [ADHD](#)
- [Asperger Syndrome](#)
- [Depressive Disorder](#)
- [Nonverbal Learning Disabilities \(NLD\)](#)
- [Rourke, Byron](#)

References and Reading

- Brumback, R. A., & Staton, R. D. (1982). An hypothesis regarding the commonality of right hemisphere involvement in learning disability, attentional disorder, and childhood major depressive disorder. *Perceptual Motor Skills*, 55, 1091–1097.
- Denckla, M. B. (1983). The Neuropsychology of social-emotional learning disabilities. *Archives of Neurology*, 40, 461–462.
- Drummond, C. R., Ahmad, S. A., & Rourke, B. P. (2005). Rules for the classification of younger children with nonverbal learning disabilities and basic phonological processing disabilities. *Archives of Clinical Neuropsychology*, 20, 171–182.
- Grace, J., & Malloy, P. (1992). Neuropsychiatric aspects of right hemisphere learning disability. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 5, 194–204.
- Gross Tsur, V., Shalev, R. S., Manor, O., & Amir, N. (1995). Developmental right-hemisphere syndrome: Clinical spectrum of the nonverbal learning disability. *Journal of Learning Disabilities*, 28, 80–86.
- Klin, A., Volkmar, F. R., Sparrow, S. S., Cicchetti, D. V., & Rourke, B. P. (1995). Validity and neuropsychological characterization of Asperger syndrome: Convergence with nonverbal learning disabilities syndrome. *Journal of Child Psychology and Psychiatry*, 36, 1127–1140.
- Manoach, D. S., Sandson, T., & Weintraub, S. (1995). The developmental social-emotional processing disorder is associated with right hemisphere abnormalities. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 8, 99–105.
- Pelletier, P. M., Ahmad, S. A., & Rourke, B. P. (2001). Classification rules for basic phonological processing disabilities and nonverbal learning disabilities: Formulation and external validity. *Child Neuropsychology*, 7, 84–98.
- Rourke, B. P. (1989). *Nonverbal learning disabilities: The syndrome and the model*. New York: Guilford Press.
- Rourke, B. P. (Ed.). (1995). *Syndrome of nonverbal learning disabilities: Neurodevelopmental manifestations*. New York: Guilford Press.
- Thompson, S. (1997). *The source for nonverbal learning disabilities*. East Moline: Lingui Systems.
- Tranel, D., Hall, L. E., Olson, S., & Tranel, N. N. (1987). Evidence for a right hemisphere developmental learning disability. *Developmental Neuropsychology*, 3, 113–120.
- Voeller, K. K. S. (1986). Right-hemisphere deficit syndrome in children. *American Journal of Psychiatry*, 143, 1004–1009.
- Weintraub, S., & Mesulam, M.-M. (1983). Developmental learning disabilities and the right hemisphere: Emotional, interpersonal, and cognitive components. *Archives of Neurology*, 40, 463–468.

Rilutek

- [Riluzole](#)

Riluzole

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Synonyms

[Rilutek](#); [Teglutik](#)

Definition

Riluzole is used for amyotrophic lateral sclerosis and prevention of ventilator dependence after a tracheostomy (Wink et al. 2011). Current pharmacological studies suggest riluzole modulates glutamate-induced excitotoxicity and inhibits the release of glutamate from presynaptic nerve

terminals (Wink et al. 2011). Common side effects of riluzole include headache, gastrointestinal distress, and decreased salivation. In recent years, several clinical studies and clinical trials have investigated riluzole's therapeutic efficacy for mood disorders, such as anxiety and depression (Wink et al. 2011). Among autism spectrum disorder patients (ASD), riluzole was effective in reducing repetitive behaviors and irritability (Wink et al. 2011). However, a single case report suggests riluzole may increase the concentration of other antipsychotic, antidepressant, or anticonvulsant drugs used for ASD patients through inhibition of CYP enzymes (Veenstra-VanderWeele 2010). Furthermore, a randomized placebo-controlled cross-over consisting of seven patients suggested that Riluzole was ineffective at reducing irritability despite being well-tolerated by ASD patients (Wink et al. 2018). However, a larger randomized study of 49 ASD patients showed a significant improvement in irritability, decreased social withdrawal, stereotypic behavior, and hyperactivity (Ghaleiha et al. 2013). Overall, riluzole shows promise for improving the social and cognitive deficits of ASD patients without significant adverse events.

See Also

- ▶ Midazolam
- ▶ Zyprexa

References and Reading

- Ghaleiha, A., Mohammadi, E., Mohammadi, M.-R., Farokhnia, M., Modabbernia, A., Yekehtaz, H., et al. (2013). Riluzole as an adjunctive therapy to risperidone for the treatment of irritability in children with autistic disorder: A double-blind, placebo-controlled, randomized trial. *Pediatric Drugs*, 15(6), 505–514. <https://doi.org/10.1007/s40272-013-0036-2>.
- Veenstra-VanderWeele, J. (2010). Increase in valproic acid levels during riluzole treatment in an adolescent with autism. *Journal of Child and Adolescent Psychopharmacology*, 20(2), 163–165. <https://doi.org/10.1089/cap.2009.0087>.
- Wink, L. K., Erickson, C. A., Stigler, K. A., & McDougle, C. J. (2011). Riluzole in autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, 21(4), 375–379. <https://doi.org/10.1089/cap.2010.0154>.
- Wink, L. K., Adams, R., Horn, P. S., Tessier, C. R., Bantel, A. P., Hong, M., et al. (2018). A randomized placebo-controlled cross-over pilot study of riluzole for drug-refractory irritability in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(9), 3051–3060. <https://doi.org/10.1007/s10803-018-3562-5>.

Rimland Diagnostic Form for Behavior-Disturbed Children (E-2)

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Synonyms

Rimland Diagnostic Form for Behavior-Disturbed Children (Form E-2)

Description

The Rimland Diagnostic Form for Behavior-Disturbed Children (Form E-1) was created in 1964 as a measure for identifying autism (Rimland 1964). The revised version, Form E-2, has 79 items related to the child's behavior and development since infancy and relies on retrospective information from the parents (Prior and Bence 1975). It includes characteristics such as cognitive, emotional, and perceptual development; speech development; obsessive behavior; and family background.

Historical Background

Form E-1 was the first diagnostic scale to be used to identify autism (Lord and Corsello 2005). It was created to systematically focus on a carefully selected range of symptoms rather than the inconsistently defined concepts relied upon by most measures at the time. The creation of the Rimland forms enabled clinicians to diagnose autism and

researchers to compare individuals objectively. It was originally designed to distinguish children with autism from those with schizophrenia, and the measure was revised to Form E-2 to differentiate between children with and without autism (Prior and Bence 1975; Rimland 1964). The scale was based on the current perspective on autism at the time, as described by Kanner in 1943 (Lord and Corsello 2005).

Psychometric Data

The original version of the Rimland, Form E-1, contained 76 questions to be answered by parents (1968). Rimland found that the parents' responses showed that the core symptoms of autism were more diffuse and idiosyncratic for children over 5½ years of age when compared to younger children (Morgan 1988). In response, he created the revised version, Form E-2, which addressed only children under the age of 5.

For each item on the checklist, a positive point was given for an answer that aligned with early infantile autism and a negative point for each behavior representative of a typically developing child, resulting in a total score (Morgan 1988). Rimland analyzed a sample of 2,218 completed forms and found total scores ranging from -42 to +45; the cutoff score that Rimland determined to be diagnostic of infantile autism was +20. He determined that only 10% of his sample scored +20 or higher, which was consistent with Kanner's belief that "true" autism is manifested in only 10% of children labeled as autistic (Parks 1983; Rimland 1971).

Several studies have demonstrated that the E-2 yields diagnoses that are different from those determined by most other diagnostic instruments (Lord and Corsello 2005). One example is the original validation study of the Childhood Autism Rating Scale (CARS; Schopler et al. 1980). Over 200 children who met the criteria for autism and 200 typically developing children were all rated on the E-2 form, and only 8 were considered to have autism by the E-2 form; of those 8, 3 were considered not to have autism on the CARS,

demonstrating a discrepancy between the two measures. Another study compared the E-2 to the Behavior Rating Instrument for Autistic and Atypical Children and found minimal diagnostic overlap (Cohen et al. 1978; Rutter et al. 1966). In addition, several studies have suggested that there are significant differences between parent and staff reports using the E-2 scale (Davids 1975; Prior and Bence 1975) and that the measure cannot differentiate between children with autism and children with other disorders (Lord and Corsello 2005). No interrater reliability, internal consistency, or test-retest reliability has been reported on the E-2 form (Parks 1983).

Other types of validity have been assessed for the E-2. The content validity of E-2 relies on Kanner's strict criteria for autism (1943) as well as on Rimland's (1964) review of relevant literature on the developmental and medical characteristics of children with autism (Morgan 1988). Several studies have examined the construct validity of the E-2 by comparing it to biological variables (Morgan 1988). Rimland (1971) and Boullin et al. (1971) reported that children with high levels of serotonin outflow had high E-2 scores. However, a later study (Yuwiler et al. 1975) was not able to replicate this finding. Rimland (1973) examined the relationship between E-2 scores and the response of children with autism and psychosis to megavitamin therapy. His results indicated that those children who showed a positive response to therapy had higher E-2 scores than those with no response, but this finding has not been replicated (Morgan 1988).

DeMyer, Churchill, Pontius, and Gilkey (1971) addressed discriminant validity when they found that both the E-1 and the E-2 could distinguish between psychotic (early schizophrenic and autistic children) and nonpsychotic children. Only the E-1 could discriminate between autistic and schizophrenic children.

Clinical Uses

Since the creation of the Rimland forms, the ways in which symptoms of autism are defined and

weighted have changed substantially, rendering the E-2 as an ineffective diagnostic tool (Lord and Corsello 2005). However, the E-2 forms may remain useful to parents starting to learn about behaviors to expect with autism. The E-2 form is currently scored without charge for parents by the Autism Research Institute in San Diego.

See Also

- [Behavior Rating Instrument for Autistic and Atypical Children \(BRIAAC\)](#)

References and Reading

- Boullin, D. J., Coleman, M., O'Brien, R. A., & Rimland, B. (1971). Laboratory predictions of infantile autism, based on 5-hydroxytryptamine efflux from blood platelets and their correlations with the Rimland E-2 score. *Journal of Autism and Childhood Schizophrenia*, 1, 63–71.
- Cohen, D. J., Caparulo, B. K., Gold, J. R., Waldo, M. C., Shaywitz, B. A., Rutterberg, B. A., et al. (1978). Agreement in diagnosis: Clinical assessment and behavior rating scales for pervasively disturbed children. *Journal of the American Academy of Child Psychiatry*, 17, 589–603.
- Davids, A. (1975). Childhood psychosis: The problem of differential diagnosis. *Journal of Autism and Childhood Schizophrenia*, 5(2), 129–138.
- DeMyer, M. K., Churchill, D. W., Pontius, W., & Gilkey, K. M. (1971). A comparison of five diagnostic systems for childhood schizophrenia and infantile autism. *Journal of Autism and Childhood Schizophrenia*, 1, 175–189.
- Lord, C., & Corsello, C. (2005). Diagnostic instruments in autistic spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 730–771). Hoboken: Wiley.
- Morgan, S. (1988). Diagnostic assessment of autism: A review of objective scales. *Journal of Psychoeducational Assessment*, 6, 139–151.
- Parks, S. (1983). The assessment of autistic children: A selective review of available instruments. *Journal of Autism and Developmental Disorders*, 13, 255–267.
- Prior, M. R., & Bence, R. (1975). A note on the validity of the Rimland Diagnostic Checklist. *Journal of Clinical Psychology*, 31(3), 510–513.
- Rimland, B. (1964). *Infantile autism: The syndrome and its implications for a neural theory of behavior* (2nd printing). New York: Appleton-Century-Crofts.
- Rimland, B. (1968). On the objective diagnosis of infantile autism. *Acta Paedopsychiatrica*, 35, 146–161.
- Rimland, B. (1971). The differentiation of childhood psychoses: An analysis of checklists for 2,218 psychotic children. *Journal of Autism and Childhood Schizophrenia*, 1(2), 161–174.
- Rimland, B. (1973). The effect of high dosage levels of certain vitamins on the behaviour of children with severe mental disorders. In D. R. Hawkins & L. Pauling (Eds.), *Orthomolecular psychiatry* (pp. 513–539). San Francisco: W. H. Freeman.
- Rutterberg, B. A., Dratman, M. L., Fraknoi, J., & Wenar, C. (1966). An instrument for evaluating autistic children (BRIAC). *Journal of the American Academy of Child Psychiatry*, 5, 453–478.
- Schopler, E., Reichler, R. J., DeVellis, R. F., & Daly, K. (1980). Toward objective classification of childhood autism: Childhood Autism Rating Scale (CARS). *Journal of Autism and Developmental Disorders*, 10, 91–103.
- Yuwiler, A., Ritvo, E., Geller, E., Glousman, R., Schneiderman, G., & Matsuno, D. (1975). Uptake and efflux of serotonin from platelets of autistic and non-autistic children. *Journal of Autism and Childhood Schizophrenia*, 5, 83–98.

Rimland Diagnostic Form for Behavior-Disturbed Children (Form E-2)

- [Rimland Diagnostic Form for Behavior-Disturbed Children \(E-2\)](#)

Rimland, Bernard

Fred R. Volkmar

Child Study Center, Irving B. Harris Professor of Child Psychiatry, Pediatrics and Psychology, Yale Child Study Center, School of Medicine, Yale University, New Haven, CT, USA

Name and Degrees

- Bernard Rimland
- B.A. Psychology, San Diego State University, San Diego, CA 1950

- M.A. Psychology, San Diego State University, San Diego, CA, 1951
- Ph.D. Psychology, Pennsylvania State University, PA, 1954

Major Appointments (Institution, Location, Dates)

- Director, Autism Research Institute, San Diego, CA

Landmark Clinical, Scientific, and Professional Contributions

A psychologist who worked for the US Navy as a researcher, Dr. Rimland's interest in autism arose when his son was diagnosed with the condition. At that time, the "refrigerator mother" notion of causation was still prominent (e.g., through the work of Bettelheim and others); Rimland conducted his own research, and in a groundbreaking book *Infantile Autism: The Syndrome and Its Implications for a Neural Theory of Behavior*, he provided a strong argument for the neurobiological cause of the condition. He also was the developer of one of the first rating scales for autism. His emphasis on the brain rather than caretaking as a cause of autism helped to focus the field and also led to a changed emphasis on teaching and intervention rather than psychotherapy for the condition. A tireless advocate for individuals with autism and their families, Rimland was quick to support the behavioral approaches developed for the condition and also was quick to rally support against facilitated communication. He had a leadership role in the Autism Society of America. He was the director of the Autism Research Institute in San Diego. In 1967, Dr. Rimland established the Autism Research Institute (ARI). This served as the base of his operation and the repository for data collected using his rating instrument. In the 1980s, after his retirement from the Navy, Dr. Rimland focused his efforts on new treatments, many of which remain controversial

(e.g., vitamin treatments, the role of vaccines in autism, and so forth). In 1985, after retiring from his day job as a Navy researcher, Dr. Rimland's research and ideas made him perhaps more prominent and took him outside the scientific mainstream. He published papers on the effects of high doses of vitamin B6 for autistic children. Tens of thousands of parents have said the vitamins helped their children, but most academic researchers have been skeptical.

Short Biography

Born in Cleveland, Ohio, Rimland relocated with his family to San Diego while he was a child. He attended San Diego State University earning a bachelor's degree in experimental psychology and a master's the following year. He completed doctoral studies at Pennsylvania State University in 1954. He then returned to work in San Diego for the US Navy as a researcher. His background in research fostered his work on the cause of autism.

See Also

- [Bettelheim, Bruno](#)

References and Reading

- Rimland, B. (1964). *Infantile autism: The syndrome and its implications for a neural theory of behavior*. New York: Appleton-Century-Crofts.
- Rimland, B. (1968). On the objective diagnosis of infantile autism. *Acta Paedopsychiatrica*, 35(4), 146–161.
- Rimland, B. (1971). The differentiation of childhood psychoses: An analysis of checklists. *Journal of Autism and Childhood Schizophrenia*, 1(2), 161–174.
- Rimland, B. (2004). Association between thimerosal-containing vaccine and autism: Comment. *JAMA: The Journal of the American Medical Association*, 291(2), 180.
- Rimland, B., & Ney, P. G. (1974). Infantile autism: Status of research. *Canadian Psychiatric Association Journal*, 19(2), 130–135.

Risk of ASD in Individuals with Down Syndrome

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Synonyms

Comorbid ASD; Co-occurring ASD

Definition

Individuals with Down syndrome (DS) are at increased risk for autism spectrum disorder (ASD). Current best estimates suggest that up to 19% of individuals with DS have co-occurring, or comorbid, ASD (Glennon et al. 2017). This prevalence rate is much higher than the ~2% ASD prevalence rate in the general population.

DS is a neurogenetic disorder caused by the triplication of chromosome 21, and it is the most common genetic cause of intellectual disability. A diagnosis of intellectual disability includes delayed cognitive development (i.e., lower than average IQ) and difficulties in adaptive functioning (e.g., practical skills or self-help skills) that impact daily function (AAIDD 2010). DS is also linked to a behavioral phenotype – a syndrome-specific profile of relative strengths and weaknesses – in cognitive, linguistic, and socio-emotional skills and behaviors that impact social communication and interaction. Compared to individuals with DS alone, those with DS who also have ASD are at risk of poorer outcomes – such as more severe cognitive impairment, fewer adaptive skills, and more challenging behaviors – that impact learning and independent living opportunities. Early identification of comorbid ASD is important because it leads to earlier intervention and treatment options and more favorable long-term outcomes (Glennon et al. 2017).

DS is usually diagnosed around birth and confirmed with genetic testing. It takes longer, however, for ASD symptoms to emerge, and the ASD diagnostic process requires clinical observation and behavioral testing. According to the *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition (DSM-5; APA 2013), a diagnosis of comorbid ASD in individuals with intellectual disability such as DS requires evidence that observed impairments in the domains of (a) social communication and interaction and (b) restricted, repetitive patterns of behavior cannot be explained by intellectual disability alone. Thus, when screening for ASD in individuals with DS, a practitioner must consider the individual's overall developmental level *and* the DS behavioral phenotype to determine whether observed impairments are typical for DS or signal comorbid ASD.

This can be a challenging task, especially because commonly used ASD screening tools were not designed for populations with intellectual disabilities. As a result, these tools may falsely identify ASD risk in individuals with DS based on behaviors that are more indicative of their intellectual disability (e.g., lack of flexibility; difficulty interpreting social cues) or the DS phenotype (Channell et al. 2015). For example, one of the most prominent features of the DS behavioral phenotype includes expressive language difficulties; these impact social communication and interaction, a domain also affected by ASD. Therefore, the practitioner must understand the symptoms that are typically associated with DS to determine if comorbid ASD is present beyond these symptoms.

On the other hand, there are certain maladaptive behaviors (e.g., inattention; social withdrawal; socially offensive behaviors) that could serve as red flags for comorbid ASD specifically in DS (Channell et al. 2019), and current screening tools may fail to capture these. Again, practitioners must identify when behavioral symptoms are atypical in DS and signal ASD. Researchers have begun to modify the algorithms for commonly used ASD screening tools to assist practitioners in screening individuals with DS (Magyar

et al. 2012), but more research is needed. Identification of ASD in DS is important because individuals with DS and comorbid ASD likely will need different treatment to support optimal development than those with DS alone.

See Also

- [American Association on Intellectual and Developmental Disabilities \(AAIDD\)](#)
- [Clinical Assessment](#)
- [Developmental Disabilities](#)
- [Diagnosis of Autism with Low Mental Age](#)
- [DSM-5](#)
- [Intellectual Disability](#)
- [Screening Instruments for ASD](#)
- [Screening Measures](#)

References and Reading

- American Association on Intellectual and Developmental Disabilities. (2010). *Intellectual disability: Definition, classification, and systems of supports* (11th ed.). Washington, DC: American Association on Intellectual and Developmental Disabilities.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Publishing.
- Channell, M. M., Phillips, B. A., Loveall, S. J., Conners, F. A., Bussanich, P., & Klinger, L. G. (2015). Patterns of autism spectrum symptomatology in individuals with Down syndrome without comorbid autism spectrum disorder. *Journal of Neurodevelopmental Disorders*, 7, 1866–1955. <https://doi.org/10.1186/1866-1955-7-5>.
- Channell, M. M., Hahn, L. J., Rosser, T. C., Hamilton, D., Frank-Crawford, M. A., Capone, G. T., Sherman, S. L., & the Down Syndrome Cognition Project. (2019). Characteristics associated with autism spectrum disorder risk in individuals with Down syndrome. *Journal of Autism and Developmental Disorders*, 49, 3543–3556. <https://doi.org/10.1007/s10803-019-04074-1>.
- Glennon, J. M., Karmiloff-Smith, A., & Thomas, M. S. C. (2017). Syndromic autism: Progressing beyond current levels of description. *Review Journal of Autism and Developmental Disorders*, 4, 321. <https://doi.org/10.1007/s40489-017-0116-2>.
- Magyar, C. I., Pandolfi, V., & Dill, C. A. (2012). An initial evaluation of the Social Communication Questionnaire for the assessment of autism spectrum disorders in children with Down syndrome. *Journal of Developmental and Behavioral Pediatrics*, 33, 134–145. <https://doi.org/10.1097/DBP.0b013e318240d3d9>.

Risperdal

- [Risperidone](#)

Risperdal TM (Risperidone)

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Synonyms

[Risperidone](#)

Definition

Risperdal TM (generic name risperidone) is an atypical antipsychotic medication. It has been approved by the US Food and Drug Administration (FDA) to treat schizophrenia, bipolar disorder, and irritability associated with ASD. Risperdal TM is a dopamine and serotonin antagonist. Dopamine is a neurotransmitter related to reward pathways in the brain, while serotonin is involved in mood regulation. Risperdal TM works by blocking postsynaptic dopamine and serotonin receptors. Specifically, Risperdal TM binds to D₂ dopamine receptors and 5-HT_{2A} serotonin receptors, decreasing the amount of dopaminergic and serotonergic activity in the brain.

In 2006, Risperdal TM was the first medication approved by the FDA for symptoms associated with ASD in children and adolescents. (The FDA subsequently approved aripiprazole, another atypical antipsychotic, for treatment of symptoms associated with ASD in 2009.) Risperdal TM treats irritability associated with ASD and has been shown to decrease aggression, tantrums, and self-injury in children with ASD. There are significant side effects associated with Risperdal TM use in ASD; the most common side effects include weight gain and somnolence, but

other side effects such as drooling and involuntary movements have also been reported.

approved for the treatment of irritability in children with autism.

See Also

► [Antipsychotics: Drugs](#)

References and Reading

- LeClerc, S., & Easley, D. (2015). Pharmacological Therapies for Autism Spectrum Disorder: A Review. *Pharmacy and Therapeutics*, 40(6), 389–397.
- Marder, S. R., & Meibach, R. C. (1994). Risperidone in the treatment of schizophrenia. *The American Journal of Psychiatry*, 151(6), 825.
- McCracken, J. T., McGough, J., Shah, B., Cronin, P., Hong, D., Aman, M. G., ... & McDougle, C. J. (2002). Risperidone in children with autism and serious behavioral problems. *New England Journal of Medicine*, 347(5), 314–321.
- McDougle, C. J., Holmes, J. P., Carlson, D. C., Pelton, G. H., Cohen, D. J., & Price, L. H. (1998). A double-blind, placebo-controlled study of risperidone in adults with autistic disorder and other pervasive developmental disorders. *Archives of General Psychiatry*, 55(7), 633–641.

Risperidone

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Department of Pediatrics, Emory University, Atlanta, GA, USA

Synonyms

[Risperdal](#)

Indications

Risperidone is approved for the treatment of tics, aggression, and agitation. It is also

Clinical Use (Including Side Effects)

Risperidone was introduced into the American marketplace in 1994. It soon found its way into the clinic for the treatment of agitation, tics, and aggression. There were several small studies conducted in children with autism suggesting that it could be effective for the treatment of agitation and aggression. In the late 1990s, a large-scale study was launched showing that risperidone was far superior to placebo in reducing tantrums, aggression, and self-injurious behavior in children with autism. The study showed that, in addition to the short-term benefits of risperidone, the beneficial effects lasted for 6 months of observation. Indeed, when the medication was withdrawn in the placebo-controlled discontinuation phase of the study, subjects randomly assigned to placebo showed prompt return of the tantrums, aggression, and self-injurious behavior. The findings of this study were replicated and another study, which led to the approval by the Food and Drug Administration, of risperidone for the treatment of irritability (tantrums, aggression, and self-injurious behavior) in children with autism, ages 5–17.

Although risperidone is clearly effective in reducing these serious behavioral problems in children with autism, it is not without adverse effects. These studies demonstrated that risperidone is not associated with the commonly observed neurological adverse effects of the typical antipsychotic medications such as haloperidol, which has also been studied in children with autism. However, weight gain and metabolic changes have emerged as important concerns over the past decade. These concerns are also present for other atypical antipsychotic medications to varying degrees. The weight gain appears to be directly related to increased appetite. In the study published by the Research Units on Pediatric Psychopharmacology, half of the parents described increased appetite treated with risperidone under double-blind conditions. When asked

whether this increase in appetite was a problem, 25% of the parents reported that the increase of appetite was a new problem. The increased appetite was associated with approximately a 6-lb weight gain in 8 weeks compared to approximately 1 lb in the placebo group. Over the 6 months of observation, children treated with risperidone gained 12 lb.

See Also

- [Atypical Antipsychotics](#)
- [Risperdal™ \(Risperidone\)](#)

References and Reading

- Martin, A., Scahill, L., Anderson, G. M., Aman, M., Arnold, L. E., McCracken, J., et al. (2004). Weight and leptin changes among risperidone-treated youths with autism: Six-month prospective data. *The American Journal of Psychiatry*, 161(6), 1125–1127.
- RUPP Autism Network, McCracken, J. T., McGough, J., Shah, B., Cronin, P., Hong, D., et al. (2002). Risperidone in children with autism and serious behavioral problems. *The New England Journal of Medicine*, 347, 314–321.
- RUPP Autism Network, McDougle, C. J., Scahill, L., Aman, M. G., McCracken, J. T., Tierney, E., et al. (2005). Risperidone for the core symptom domains of autism: Results from the study by the autism network of the research units on pediatric psychopharmacology. *The American Journal of Psychiatry*, 162, 1142–1148.
- Shea, S., Turgay, A., Carroll, A., Schulz, M., Orlik, H., Smith, I., et al. (2004). Risperidone in the treatment of disruptive behavioral symptoms in children with autistic and other pervasive developmental disorders. *Pediatrics*, 114(5), e634–e641.
- Troost, P. W., Lahuus, B. E., Steenhuis, M. P., Ketelaars, C. E., Buitelaar, J. K., van Engeland, H., et al. (2005). Long-term effects of risperidone in children with autism spectrum disorders: a placebo discontinuation study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44(11), 1137–1144.

Ritalin

- [Methylphenidate](#)

Ritualized Request

- [Protoimperative](#)

Ritvo Autism Asperger Diagnostic Scale – Revised

- [RAADS-R](#)

Ritvo, Edward

Ariella Riva Ritvo
Child Study Center, Yale School of Medicine,
Yale University, Los Angeles, CA, USA

Name and Degrees

Edward R. Ritvo, M.D.

Major Appointments (Institution, Location, Dates)

- B.A., Social Anthropology, Harvard University, Cambridge, MA, 1951
- M.D., Boston University School of Medicine, Boston, MA, 1955
- Internship, Massachusetts Memorial Hospital, Boston, MA, 1956
- Psychiatry Residency, Massachusetts Mental Health Center, Boston, MA, 1956–1958
- Teaching Fellow in Psychiatry, Harvard Medical School, Tufts Medical School, Boston, MA, 1957–1958
- Fellowship in Child Psychiatry, James Jackson Putnam Children's Center, Boston, MA, 1958–1959

Edward R. Ritvo: deceased.

- Chief, Closed Neuropsychiatric Section, US Army Brook General Hospital, Sam Houston, TX, 1958–1961
- Fellowship in Child Psychiatry, Reiss-Davis Clinic for Child Psychiatry, Los Angeles, CA, 1961–1962
- Instructor, Assistant Professor, Associate Professor, and Professor (now Emeritus), Neuropsychiatric Institute, UCLA School of Medicine, Los Angeles, CA 1962 to June, 2020

Major Honors and Awards

- The National Society of Autistic Children Annual Award for Scientific Achievement, 1974
- American Psychiatric Association, Blanche F. Ittleson Award, 1990
- American Academy of Child and Adolescent Psychiatry, George Tarjan Award, 1994
- International Society for Autism Research, Lifetime Achievement Award, 2010

Landmark Clinical, Scientific, and Professional Contributions

A prolific researcher and clinician, Dr. Ritvo has conducted pioneering studies in autism. His early work included new approaches to the definition of the condition, development of rating scales for autism, and studies of electrophysiology and neural systems. His work has also included epidemiological and genetic studies as well as pioneering work on neurochemical systems and potential pharmacological treatments. A member of the UCLA faculty for many decades, Dr. Ritvo has helped shape the careers of many researchers and clinicians.

Short Biography

Dr. Edward Ritvo graduated in Harvard College in 1951 and Boston University School of Medicine 1955 and completed Adult and Child Psychiatry training at Harvard Medical School. After serving as Chief of Fourth Army Psychiatry (Captain, US Army), he joined the faculty of

UCLA Medical School in 1960, where he pioneered multidisciplinary research on autism spectrum disorder in the areas of neurophysiology, neurobiochemistry, neuropathology, genetics, clinical diagnosis, and epidemiology. He is recognized as an international authority on autism spectrum disorder, has written three books and over 90 scientific articles in peer-reviewed journals, and has received awards for his research contributions from the American Psychiatric Association, the American Academy of Child Psychiatry, and the International Society for Autism Research. Edward R. Ritvo died on June 10, 2020.

See Also

- ▶ [Autistic Disorder](#)
- ▶ [Epidemiology](#)
- ▶ [Neurochemistry](#)
- ▶ [Serotonin](#)

References and Reading

- Frankel, F., Freeman, B. J., Ritvo, E., & Pardo, R. (1978). The effect of environmental stimulation upon the stereotyped behavior of autistic children. *Journal of Autism and Childhood Schizophrenia*, 8(4), 389–394.
- Freeman, B. J., Ritvo, E., & Miller, R. (1975). An operant procedure to teach an echolalic, autistic child to answer questions appropriately. *Journal of Autism and Childhood Schizophrenia*, 5(2), 169–176.
- Freeman, B. J., Ritvo, E. R., Guthrie, D., Schroth, P., & Ball, J. (1978). The behavior observation scale for autism: Initial methodology, data analysis, and preliminary findings on 89 children. *Journal of the American Academy of Child Psychiatry*, 17(4), 576–588.
- Garber, H. J., & Ritvo, E. R. (1992). Magnetic resonance imaging of the posterior fossa in autistic adults. *The American Journal of Psychiatry*, 149(2), 245–247.
- Geller, E., Ritvo, E., Freeman, B. J., & Yuwiler, A. (1982). Preliminary observations on the effects of fenfluramine on blood serotonin and symptoms in three autistic boys. *The New England Journal of Medicine*, 307, 165–169.
- Mason-Brothers, A., Ritvo, E. R., Guze, B., Mo, A., Freeman, B. J., Funderburk, S. J., & Schroth, P. C. (1987). Pre-, peri-, and postnatal factors in 181 autistic patients from single and multiple incidence families. *Journal of the American Academy of Child and Adolescent Psychiatry*, 26(1), 39–42.

- Ornitz, E. M., & Ritvo, E. R. (1968a). Neurophysiologic mechanisms underlying perceptual inconstancy in autistic and schizophrenic children. *Archives of General Psychiatry*, 19(1), 22–27.
- Ornitz, E. M., & Ritvo, E. R. (1968b). Perceptual inconstancy in early infantile autism. The syndrome of early infant autism and its variants including certain cases of childhood schizophrenia. *Archives of General Psychiatry*, 18(1), 76–98.
- Ritvo, E. R. (1977). Biochemical studies of children with the syndromes of autism, childhood schizophrenia and related developmental disabilities: A review. *The Journal of Child Psychology and Psychiatry*, 18(4), 373–379.
- Ritvo, E. R., & Freeman, B. J. (1977). National society for autistic children definition of the syndrome of. *Journal of Pediatric Psychology*, 2(4), 142–145.
- Ritvo, E. R., Ornitz, E. M., Eviatar, A., Markham, C. H., Brown, M. B., & Mason, A. (1969). Decreased post-rotatory nystagmus in early infantile autism. *Neurology*, 19(7), 653–658.
- Ritvo, E. R., Yuwiler, A., Geller, E., Ornitz, E. M., Saeger, K., & Plotkin, S. (1970). Increased blood serotonin and platelets in early infantile autism. *Archives of General Psychiatry*, 23(6), 566–572.
- Ritvo, E. R., Freeman, B. J., Scheibel, A. B., Duong, T., Robinson, H., Guthrie, D., & Ritvo, A. (1986). Lower Purkinje cell counts in the cerebella of four autistic subjects: Initial findings of the UCLA-NSAC Autopsy Research Report. *The American Journal of Psychiatry*, 143(7), 862–866.
- Ritvo, E. R., Freeman, B. J., Pingree, C., Mason-Brothers, A., Jorde, L., Jenson, W. R., et al. (1989). The UCLA-university of Utah epidemiologic survey of autism: Prevalence. *Journal of the American Psychiatric*, 146(2), 194–199.
- Ritvo, R. A., Ritvo, E. R., Guthrie, D., Yuwiler, A., Ritvo, M. J., & Weisbender, L. (2008). A scale to assist the diagnosis of autism and Asperger's disorder in adults (RAADS): A pilot study. *Journal of Autism and Developmental Disorders*, 38(2), 213–223.

Ritvo-Freeman Real Life Rating Scale

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Synonyms

Ritvo-Freeman RLRS; RLRS

Description

The Ritvo-Freeman Real Life Rating Scale (RLRS; Freeman et al. 1986) was originally designed as a direct observational measure for assessing treatment effects for individuals with autism in their natural setting. In this format, pairs of trained raters observed an individual with autism for 30 min in the same setting, at the same time of day, for 3 days, and reported the presence and frequency of a number of behaviors associated with autistic symptomatology in five different domains. The scale has been used to study treatment effects in both children with autism (e.g., Matese et al. 1994; McDougle et al. 2005; Ritvo et al. 1986) and in adults with autism (e.g., McDougle et al. 1996, 1998).

In the RLRS standardized procedure that was initially used by the Autism Network of the Research Units on Pediatric Psychopharmacology (RUPP), a room is set up with a parent chair that is surrounded by toys. The parent and child are escorted into the room, and the parent is provided with index cards that instruct the parent how they should interact with their child in specific ways. These instructions are designed to elicit reactions by the child with autism that can be used to code ratings for the 47 target behaviors. The parent and child are positioned about 10 ft. from one another, and the raters observe the interactions set up by the instructions. The raters typically observe behaviors from behind a one-way mirror (or from an observation suite), and the session is recorded.

During the 30-min observation period, the rater (s) notes the frequency of 47 individual target behaviors. At the end of the observation period, each item is rated on a 0–3 scale, reflecting the occurrence and frequency of the target behavior that was seen during the 30-min observation period: 0 = Never, 1 = Rarely (1–3 times), 2 = Frequently (4 or more times), and 3 = Almost always (occurred constantly throughout the observation period). Individual target behavior items are then grouped into the five scales: I. Sensory-Motor Behaviors (7 items), II. Social Relationship to People (9 items), III. Affectual Reactions (5 items), IV. Sensory Responses (16 items), and

V. Language (10 items). Scale scores are obtained by summing the scores of the individual items and dividing by the number of items on the scale. Scores on items reflecting appropriate behavior, e.g., item #3 on the Language scale (“initiates appropriate verbal communication”), are subtracted prior to the calculation of the mean score from scales II, IV, and V. The mean scores on the five scales are then summed to create an overall score.

Historical Background

As reported by Freeman et al. (1986), the RLRS was first developed as a measure of overall severity of autistic symptomatology. It was designed to be administered at repeated intervals both at baseline (i.e., before treatment is initiated) and then at subsequent intervals (i.e., after treatment is initiated).

Although two of the major goals in developing this instrument were that nonprofessional observers could be rapidly trained to use it and that interobserver agreement should be easily attainable, at least one multisite study (McDougle et al. 2005) reported unacceptable validity across different sites/raters. Due to concerns with unacceptable reliability across study sites, the RUPP network created a modified RLRS instrument that converted the original observational measure to a parent rating scale. The original five scales were retained in this parent instrument, and the individual items were rephrased so that parents could rate their child's behavior on a 0–3 questionnaire format.

Psychometric Data

Relatively little information was provided in the original (1986) article regarding the characteristics of the sample (e.g., age, IQ, gender) from which the psychometric data were generated. The authors state that the instrument is applicable to patients of varying symptom severity. Interrater reliability for experienced observers was assessed using correlational methods: Pearson correlations

ranged from .59 to .85 on the five scales and was .90 on the overall score; intraclass correlation ranged from .62 to .78 on the scales and was .83 on the overall score. Interrater reliability was predictably lower for novice observers.

In contrast, Matese et al. (1994) reported poor interrater reliability for Scale III (Affectual Reactions) and Scale IV (Sensory Response); they also reported that 14 out of 47 items had intraclass correlation coefficients below the acceptable level of .5. However, they also pointed out that reliability estimates may have been deleteriously affected by their small sample of children with autism ($n = 15$) and by their raters having had minimal practice using the RLRS with clients being rated.

Concerns about interrater reliability using direct observation method are particularly notable in multisite projects. As McDougle et al. (2005) report, despite extensive and repeated training of study personnel, as well as standardization of procedures, the RUPP network was unable to attain acceptable reliability across the five study sites on the original direct observational instrument.

Another potential psychometric concern with the RLRS is that because scores for appropriate behaviors are subtracted from subscales II, IV, and V, the resulting score on the subscale can be negative. Specifically, this situation can occur if the sum of the inappropriate behaviors (e.g., items 4–10 on the Language scale) is not higher than the sum of the appropriate behaviors (e.g., items 1–3 on the Language scale).

A particular strength of the RLRS is that the target behaviors that closely mirror core behaviors associated with autism. For instance, on Scale I: Sensory Motor Behaviors, the individual items include whirls, flaps, pacing, bangs/hits self, rocks, and toe walks. Another strength is that this autistic symptomatology can be assessed in a natural setting using the RLRS. However, given the concerns with interrater reliability across multiple sites/raters, to the extent possible, if the direct observation version of the RLRS is used, it may be advisable to employ the same rater across repeated administrations – a strategy employed by Tolbert et al. (2001).

Clinical Uses

This RLRS is not marketed commercially as a clinical measure. However, in addition to its use as a research instrument, its creators intended it to be used to assess response to intervention in a clinical setting – both within individual patients and among groups of patients. Ritvo et al. (1986) even reported anecdotally that parents had been trained to use the RLRS to track treatment response in the home setting.

The RLRS has been used to study a variety of medication and nonmedication treatments in individuals with autism, including risperidone (e.g., McDougle et al. 2005; Miral et al. 2008; Gencer et al. 2008), fluvoxamine (McDougle et al. 1996), fenfluramine (Ritvo et al. 1986), and casein-free/gluten-free diets (Hyman et al. 2010).

See Also

► [Direct Observation](#)

References and Reading

- Freeman, B. J., Ritvo, E. R., Yokota, A., & Ritvo, A. (1986). A scale for rating symptoms of patients with the syndrome of autism in real life settings. *Journal of the American Academy of Child Psychiatry*, 25, 130–136.
- Gencer, O., Emiroglu, F. N., Miral, S., Baykara, B., Baykara, A., & Dirik, E. (2008). Comparison of long-term efficacy and safety of haloperidol in children and adolescents with autistic disorder: An open label maintenance study. *European Journal of Child and Adolescent Psychiatry*, 17, 217–225.
- Hyman, S., Stewart, P. A., Smith, T., Foley, J., Cain, J., Peck, R., et al. (2010, May). *The gluten free and casein free (GFCF) diet: A double blind, placebo controlled challenge study (Poster #140.007)*. Presented at the international meeting for autism research (IMFAR), Philadelphia.
- Matese, M., Matson, J. L., & Sevin, J. (1994). Comparison of psychotic and autistic children using behavioral observation. *Journal of Autism and Developmental Disorders*, 24, 83–94.
- McDougle, C. J., Naylor, S. T., Cohen, D. J., Volkmar, F. R., Heninger, G. R., & Price, L. K. (1996). A double-blind, placebo-controlled study of fluvoxamine in adults with autistic disorder. *Archives of General Psychiatry*, 53, 1001–1008.
- McDougle, C. J., Holmes, J. P., Carlson, D. C., Pelton, G. H., Cohen, D. J., & Price, L. K. (1998). A double-blind, placebo-controlled study of risperidone in adults with autistic disorder and other pervasive developmental disorders. *Archives of General Psychiatry*, 55, 633–641.
- McDougle, C. J., Scahill, L., Aman, M. G., McCracken, J. T., Tierney, E., Davies, M., et al. (2005). Risperidone for the core symptom domains of autism: Results from the study by the autism network of the research units on pediatric psychopharmacology. *American Journal of Psychiatry*, 162, 1142–1148.
- Miral, S., Gencer, O., Inal-Emiroglu, F. N., Baykara, B., Baykara, A., & Dirik, E. (2008). Risperidone versus haloperidol in children and adolescents with AD: A randomized, controlled, double-blind trial. *European Journal of Child and Adolescent Psychiatry*, 17, 1–8.
- Ritvo, E. R., Freeman, B. J., Yuwiler, A., Geller, E. G., Schroth, P., Yokota, A., et al. (1986). Fenfluramine treatment of autism: UCLA collaborative study of 81 patients at nine medical centers. *Psychopharmacology Bulletin*, 22, 133–140.
- Tolbert, L., Brown, R., Fowler, P., & Parsons, D. (2001). Brief report: Lack of correlation between age of symptom onset and contemporaneous presentation. *Journal of Autism and Developmental Disorders*, 31, 241–245.

Ritvo-Freeman Real-Life Rating Scale

► [Real-Life Rating Scale](#)

Ritvo-Freeman RLRS

► [Ritvo-Freeman Real Life Rating Scale](#)

Rivotril

► [Clonazepam: Definition](#)

RJA/IJA (Initiating/Responding to Joint Attention)

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Definition

Before language develops, infants learn to communicate. One essential part of this involves learning to jointly attend to the same referent (object or event) with another person in social interactions. This development of the capacity for purposeful social attention coordination or *joint attention* develops slowly from early in the first year of life through childhood. Much like language, the development of joint attention in infancy is expressed in terms of both receptive and expressive forms (Mundy et al. 2009). *Responding to joint attention* (RJA) is the receptive form and refers to infants' ability to follow the direction of the gaze and gestures of others in order to share a common point of reference (Fig. 1a).

Once fully developed, RJA allows infants and toddlers to use others' gaze to identify the referent of their actions, such as language, or their intentions and feelings. Alternatively, *initiating joint attention* (IJA) is the expressive form and involves infants' use of gestures and eye contact to direct attention to objects, events, as well as to themselves. The function here is to use nonverbal bids to show or spontaneously seek to share a child's interests or pleasurable experience with others (Fig. 1). Both of these types of joint attention are behaviorally well developed by 9 months but continue to develop well into the second year. Their beginning point is less well understood but evidence suggests that the onset of RJA may be as early as 2–4 months of age (Mundy 2011).

Historical Background

The importance of joint attention in understanding autism stems in large part from its role in the social attention model of autism. This social attention model suggests that autism is characterized by atypical quantity or quality of attention to people in the first years of life. The attenuation of attention to people undermines the capacity of young children with autism to learn from and with other people. Hence, a paucity of social attention leads to an "early social learning disability." This contributes to deviations in the age-appropriate pace of social communication and cognitive development in children with this syndrome (see Fig. 2). Hence, social attention impairments have come to be considered a cardinal symptom domain of autism (e.g., Dawson et al. 2004; Mundy 1995; Schultz 2005). The causes of decreased early social attention are unclear. Nonetheless, they likely stem from atypical neural development and consequent differences in motivation, executive functions, or perceptual processes which typically serve to support or even promote attending to other people in early development.

Diminished social attention to people in autism is expressed in three domains: (1) impaired *social orienting* or the tendency to prioritize and deploy attention to people versus objects (Dawson et al. 2004; Klin 1991), (2) impaired *joint attention* or the capacity to readily coordinate visual, auditory, or mental attention with a social partner, which is often used to share affective experiences of objects of events with others (e.g., Mundy et al. 1986), and (3) impaired *face perception and processing* such as the capacity to process facial expressions of emotions (e.g., Schultz 2005). The study of all these domains has been vital to advancing the science of autism in the past two decades. However, perhaps the strongest direct evidence linking social attention deficits with social learning, social symptoms, and effective treatments for autism comes from studies of joint attention.



RJA/IJA (Initiating/Responding to Joint Attention), Fig. 1 Illustrations of different types of infant social attention coordination behaviors: (a) responding to joint attention – RJA, involving following another person's gaze and pointing gesture; (b) initiating joint attention – IJA, involving a conventional gesture "pointing" to share

attention regarding a room poster; (c_{1,2,3}) IJA, involving *alternating eye contact* to share attention with respect to a toy; (d) initiating behavior request, involving pointing to elicit aid in obtaining an out-of-reach object; and (e) responding to behavior requests, involving following an adult's open-palm "give it to me" gesture

The development of joint attention heralds a new stage of infant-caregiver interactions that facilitate social learning. For example, early language learning often takes place in unstructured, incidental situations where parents use an unknown word to refer to a new object. In such situations, infants are faced with a problem. How does an infant know which of the myriad potential referents in the field of view to attach to the word? Many processes help the infant to solve these problems, and one of these involves their use of

RJA and following the spatial direction of the gaze of their parent. This increases the likelihood of attending to the area of the environment that contains the correct word-object match, thus reducing referential mapping error in early word acquisition (Baldwin 1995). Infants' use of an IJA bid, such as a show, to spontaneously indicate interest in an object or event even more precisely delimits the referent for possible new word learning or information-gathering opportunities with caregivers. Thus, joint attention serves a

self-organizing role that optimizes social information processing in early, unstructured social learning interactions. Through joint attention, and other skills like imitation, young children play a vital and active or *constructivist* role in social learning.

Historically, it was Frank Curcio (1978) at Boston University who published the first observation that raised the hypothesis that children with autism may have a specific deficit in the use of joint attention which impedes their adoption of an active, constructivist role in social learning. Observing children in a classroom, Curcio noted that students with autism did not use eye contact and gestures to share the experience of an object or event with others (i.e., joint attention), even though they appeared to use eye contact and gestures for other forms of nonverbal communication such as requesting. Curcio's observation that children with autism exhibit a specific impairment in joint attention, relative to other preverbal communication behaviors, was ultimately replicated and extended by numerous studies. The first set of these studies indicated that joint attention impairments were characteristic of young children with autism *but not* young mental age-matched groups of children with other types of developmental disorders (Loveland and Landry 1986; Mundy et al. 1986).

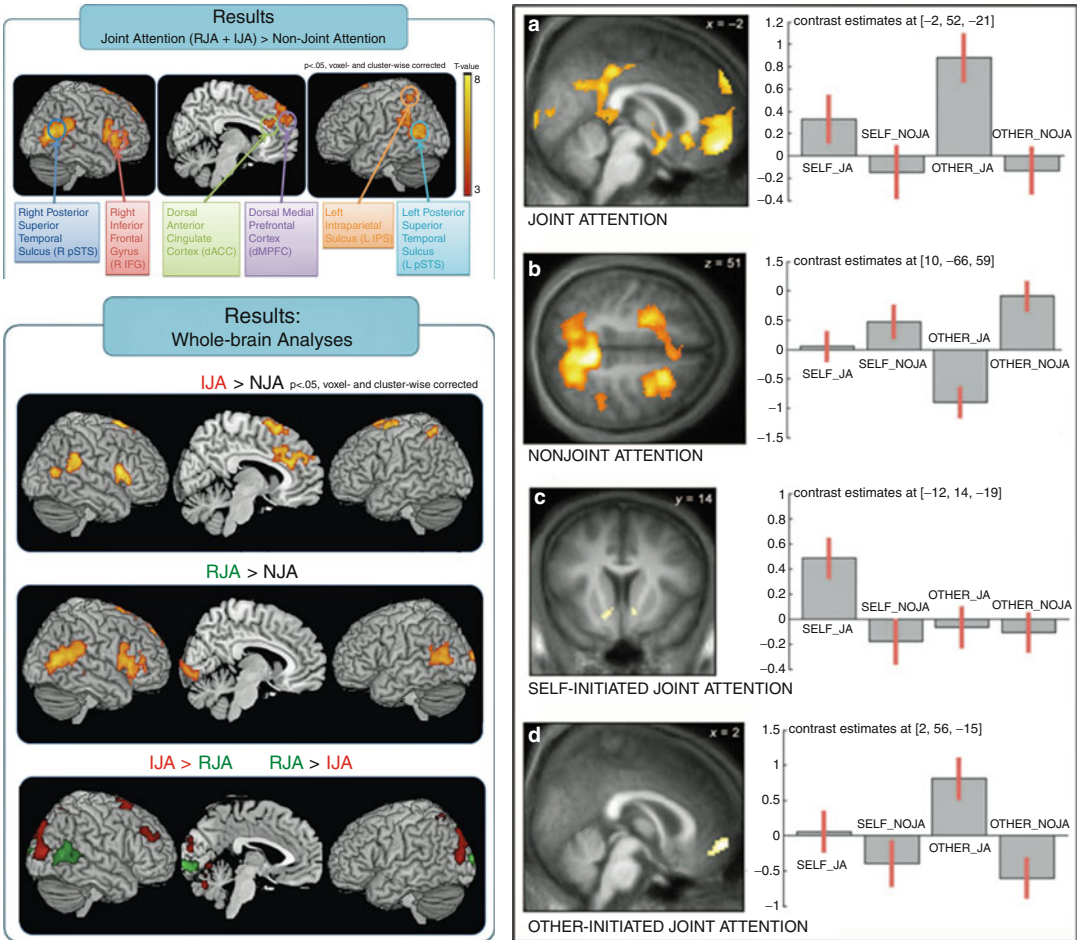
Later, the importance of the distinction between IJA and RJA became apparent. Clinical research indicates autism is characterized by pronounced impairments in *both* IJA and RJA in the first 24–48 months of life. However, sometime after about 30 months mental age, many children with autism begin to improve in RJA skills but remain relatively impaired in IJA (Mundy et al. 2009). Hence, many children with autism seem to improve in the face processing and gaze processing necessary to track and respond to gaze of others but continue to be relatively impaired in using gaze or gestures like showing to initiate episodes of shared attention and experience with others. This observation is incorporated into current diagnostic systems where IJA impairment or “the lack of spontaneous sharing experiences with others, such as showing. . .” is a primary symptom of social impairment in both American and

European psychiatric nosologies, but gaze following is not (Mundy et al. 2009). Moreover, gold-standard diagnostic measures, such as the Autism Diagnostic Observation Schedule, recognize that measures of both IJA and RJA are useful in diagnostic algorithms for the youngest children; but for older children on the cusp of language development, only IJA measures are included in the diagnostic criteria (Lord et al. 2000). Hence, one current important hypothesis about autism is that it involves a disturbance of the spontaneous generation of social behaviors, as much as or more than a disturbance of the perception and response to the social behaviors of others (Mundy 1995).

Current Knowledge

Advances in the current knowledge of the role of IJA and RJA impairments stem from at least three areas of science: cognitive neuroscience, developmental science, and intervention research. Cognitive neuroscience has begun to provide insights into the development of joint attention that tie joint attention to psychological and neurodevelopmental theory on the etiology of autism. Current evidence suggests that the development of RJA and IJA functions is supported by common as well as distinct elements of a distributed frontal and parietal neural network (e.g., Redcay et al. 2010; see Fig. 2). This is important because theory and research suggest that children with autism may be most vulnerable to complex cognitive developments and processes that require connectivity and the rapid transmission, comparison, and integration of information across distal frontal and cortical networks (e.g., Courchesne and Pierce 2005). Thus, it may be that joint attention impairments are an early expression of the developmental vulnerability in complex-distributed cognitive processes in autism.

It is also the case though that theory and research have long suggested that IJA impairments in autism may arise from disturbances in the motivation to attend to people or to spontaneously share experiences with other people (Mundy 1995). Recent cognitive neuroscience



RJA/IJA (Initiating/Responding to Joint Attention), Fig. 2 Illustrations of recent imaging data on the distributed cortical processing network involved in joint attention. (a) On the left-hand side of the figure are illustrations from a paper by Redcay et al. (2010) that depict the frontal and parietal systems involved in joint attention, as well as the patterns of cortical activation that distinguish initiating

and responding to joint attention behaviors. (b) On the right-hand side of the figure are illustrations from a recent paper by Leonhard Schilbach et al. (2010) that show the distributed joint attention network and raise the seminal possibility that network activation in the striatal cortex, associated with the processing of positive stimulus reward value, distinguishes the neural network of IJA from RJA

lends support to this hypothesis with the observation that one element of the neural network that distinguishes IJA and RJA is the contribution of frontal striatal reward networks to the former but not the latter (Schilbach et al. 2010, see Fig. 2).

The significance of IJA and RJA impairments in autism, however, may be best understood in the context of studies on the relations of joint attention to human learning and development. Indirect support for this connection has come from observations from developmental studies that indicate that frequency with which infants engage in joint

attention is positively and significantly related to their language acquisition, even when variance associated with general cognition is controlled (e.g., Mundy et al. 2009). Joint attention is also associated with the depth of information processing and recognition memory in infants, as well as with individual differences in childhood measures of IQ, self-regulation, and social competence (Mundy et al. 2009). In autism, IJA development has been observed to predict social development and RJA to predict language development into later childhood and even adolescence

(Sigman and McGovern 2005; Sigman and Ruskin 1999).

The connection between IJA, RJA, and learning in autism becomes even clearer in the context of information provided by early intervention research. Several experimental studies have reported observations that suggest that early intervention to improve the initiation of joint attention leads to a cascade of enhanced social learning effects in young children with autism (Kasari et al. 2008; Jones et al. 2006; Whalen et al. 2006). For example, Kasari et al. (2008) have reported a sequence of findings from a randomized control trial of an intervention to improve IJA development in 3-year-olds with autism. All the children in this study were enrolled in a comprehensive 1-year program of 30 h per week of intervention based on applied behavior analysis (ABA). Thirty-nine children in this program were randomly assigned to an additional brief 5- to 6-week course of 30 min of intervention to increase IJA development, and 17 children were assigned to an ABA-only treatment comparison group. The results indicated that the IJA treatment not only improved joint attention development in the treatment group but also boosted language learning and longer term outcomes for young children relative to the improvements children made in ABA alone.

This is not to say that RJA is not important in social learning. Another research group examined the degree to which language improved in a sample of 29 4-year-olds with autism who were in a variety of communication-based interventions (Bono et al. 2004). On average, children in this study received 24 h of intervention per week for 1 year. Children were enrolled in a variety of intervention modalities including ABA, speech and language therapy, occupational therapy, social skills interventions, sensory integration, and various combinations of these interventions. As a group, the 29 children displayed modest gains in language over the year, and language gains did not appear to be “dose related.” That is, greater language gains were not observed for children receiving more hours of intervention per week. However, on further inspection, Bono et al. (2004) observed that the group could be divided in

children with well-developed RJA and poorly developed RJA at the beginning of the study. Only the former group displayed significant language gains after 1 year and, within this group of children with more hours of intervention, displays greater gains in language. Thus, consistent with the social learning hypothesis of joint attention in autism, the presence or absence of RJA skill moderated language learning and sensitivity to intensity of intervention in this sample of children.

Future Directions

Social Cognition and Joint Attention

Sometime, people confuse joint attention with social cognition and neglect the role that joint attention plays in social learning. The social cognitive model of joint attention proposes that as infants monitor and represent their own goal-related intentional activity, they also monitor and represent the goal-related behavior of others (Tomasello et al. 2005). The early development of social cognition at about 9–12 months allows infants to integrate these two sources of information over time, and infants become able to impute the conditional proposition that if self-intentions lead to goal-related behavior, then goal-related behavior in others must follow from their intentions. Thus, this model suggests that social cognition is necessary for the development of functional joint attention in infancy. Furthermore, social cognition is thought to be equally common to all types of joint attention. Therefore, initiating and responding to joint attention should be highly correlated in development.

Research supports elements of the social cognitive model. In one revealing study, testers either turned their heads with their eyes open or turned their heads with eyes closed in gaze following trials presented to 9-, 10-, and 11-month-olds (Brooks and Meltzoff 2002). The task for the infants was to respond to joint attention and turn their attention in the direction of the gaze or head turn of the tester. All the children performed equally well in the “eyes open” condition. However, in the “eyes closed” condition, the 10- and 11-month-old infants rarely followed the testers’

head turns, but the 9-month-olds followed at a rate comparable to the eyes open condition. Thus, the 10-month-olds were able to inhibit following in this condition presumably based on a social cognitive awareness of the meaning of the referential intent of visual gaze. However, the 9-month-olds were not able to inhibit their responding behavior because they lacked this aspect of social cognition.

Of course, Brooks and Meltzoff's (2002) data also indicate that 9-month-olds engage in joint attention behavior *before* they fully develop aspects of social cognitive awareness. Indeed, 9-month responding to joint attention predicts 24-month language, attesting to the functional validity of its measurement at that age (Mundy et al. 2009). These observations challenge the social cognitive model. So do observations that initiating and responding to joint attention are not highly correlated in development, display different patterns of age-related growth in infancy, and have unique associations with subsequent development (Mundy et al. 2009). An alternative to the social cognitive perspective appears to be needed to account for these observations.

Mundy et al. (2009) have proposed one such alternative in which joint attention enables social cognitive development. Accordingly, joint attention is an outcome of two interacting attention regulation systems described by Michael Posner and his colleagues. One is the posterior orienting and perceptual attention system, and it plays a primary role in responding to joint attention development in infancy. This relatively involuntary system begins to develop in the first months of life and prioritizes the capacity to orient in the direction of biologically meaningful stimuli. It is supported by the parietal and superior temporal cortices (Fig. 2b), which serve aspects of representational development, imitation, and the perception of the eye and head orientations of others, as well as the perception of spatial relations between self, other, and the environment. The neural substrates and functions of this system are common to many primates. Shorthand for one goal-related cognitive output from this system is the understanding that "*where others' eyes go, behavior follows*" (Jellema et al. 2000).

Alternatively, initiating joint attention is supported by the later developing anterior attention system. This system controls volitional, goal-directed attention allocation that is constrained and corrected by reward-related self-appraisal of behavior. It involves a neural network that includes the frontal eye fields, prefrontal association cortex, orbital frontal cortex, and anterior cingulate. This system (e.g., anterior cingulate) also regulates the integrated activity across the anterior and posterior attention systems. This integration yields the distinct form of human attention we call joint attention (Mundy et al. 2009). As previously noted (Fig. 2), imaging data attest to the integrated activation of the anterior and posterior systems in human joint attention. Moreover, this joint attention network has much in common with the neural substrates of social cognition (Mundy et al. 2009).

Shorthand for an initial cognitive output from the anterior system is a *self-awareness* of intentional attention control and the understanding that "*where my eyes go, my successful behavior follows*." Mundy et al. (2009) suggested that an executive function of the anterior attention system integrates internal information about self-initiated goal-related attention deployment (where my eyes go, my behavior follows) with external information from posterior system processing of the attention and behavior in others (where others' eyes go others' behavior follows). As this integrative processing matures in social interaction, a fully functional adaptive human social cognitive system emerges. Thus, the social coordination of *overt* aspects of visual attention early in infancy provides information and experiences that nourish the development of the human capacity for social coordination of *covert* aspects of attention (i.e., social cognition), such as when social partners share attention to psychological phenomena including ideas, intentions, and emotions (Mundy et al. 2009).

Elements of this perspective are consistent with current social cognitive models. However, an explicit tenet here is that the rapid, real-time functional interactions and maturation of a distributed cortical attention network make a

fundamental contribution to the human development of social cognition. This notion owes much to Gerald Edelman's (2006) long-standing hypothesis that synchronous or "reentrant" firing of distal cortical neural groups is vital to consciousness and self-conscious systems. This model also recognizes that processes that affect the organization, accuracy, and speed of integrated processing, within or across the anterior and posterior attention systems, may contribute to phylogenetic or human developmental differences in joint attention and social cognition. Finally, to be fully realized, integrated processing of the anterior-posterior systems requires practice with sharing attention early in life. Thus, in this model, practice with joint attention in the first 9 months of life is thought of as a major *contributor to* rather than a *product of* the development of social cognition. Consequently, early joint attention impairments are thought to lead directly to a form of social learning disability which has a negative impact on language learning as well as social cognitive and social skills development in childhood (for more details see Mundy 2011; Mundy et al. 2009).

See Also

- [Joint Attention](#)
- [Social Cognition](#)
- [Social Communication](#)

References and Reading

- Baldwin, D. (1995). Understanding the link between joint attention and language. In C. Moore & P. Dunham (Eds.), *Joint attention: Its origins and role in development* (pp. 131–158). Hillsdale: Lawrence Erlbaum Associates.
- Bono, M., Daley, T., & Sigman, M. (2004). Joint attention moderates the relation between intervention and language development in young children with autism. *Journal of Autism and Related Disorders*, 34, 495–505.
- Brooks, R., & Meltzoff, A. (2002). The importance of eyes: How infants interpret adult looking behavior. *Developmental Psychology*, 38, 958–966.
- Courchesne, E., & Pierce, K. (2005). Why the frontal cortex in autism might be talking only to itself: Local over-connectivity but long distance disconnection. *Current Opinion in Neurobiology*, 15, 225–230.
- Curcio, F. (1978). Sensorimotor functioning and communication in mute autistic children. *Journal of Autism and Developmental Disorders*, 8, 281–292.
- Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., et al. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention in autism. *Developmental Psychology*, 40, 271–283.
- Edelman, G. M. (2006). *Second nature: Brain science and human knowledge*. New Haven: Yale University Press.
- Jellema, T., Baker, C., Wicker, B., & Perrett, D. (2000). Neural representation for the perception of intentionality of actions. *Brain and Cognition*, 44, 280–302.
- Jones, E., Carr, E., & Feeley, K. (2006). Multiple effects of joint attention intervention for children with autism. *Behavior Modification*, 30, 782–834.
- Kasari, C., Paparella, T., Freeman, S., & Jahromi, L. (2008). Language outcomes in autism: Randomized comparison of joint attention and play interventions. *Journal of Consulting and Clinical Psychology*, 76, 125–137.
- Klin, A. (1991). Young children's listening preferences in regard to speech: A possible characterization of the symptom of social withdrawal in autism. *Journal of Autism and Developmental Disorders*, 21, 29–42.
- Lord, C., Lambrecht, L., Cook, E., Leventhal, B., DiLavore, P., Leventhal, B. L., et al. (2000). The autism diagnostic observation schedule-generic: A standard measure of social communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30, 205–223.
- Loveland, K., & Landry, S. (1986). Joint attention and language in autism and developmental language delay. *Journal of Autism and Developmental Disorders*, 16, 335–349.
- Mundy, P. (1995). Joint attention and social-emotional approach behavior in children with autism. *Development and Psychopathology*, 7, 63–82.
- Mundy, P. (2011). The social behavior of autism: Parallel and distributed information processing perspective, Chapter 8. In D. Amaral, G. Dawson, & D. Geschwind (Eds.), *Autism spectrum disorders* (pp. 149–171). New York: Oxford University Press.
- Mundy, P., Sigman, M., Ungerer, J., & Sherman, T. (1986). Defining the social deficits of autism: The contribution of nonverbal communication measures. *Journal of Child Psychology and Psychiatry*, 27, 657–669.
- Mundy, P., Sullivan, L., & Mastergeorge, A. (2009). A parallel and distributed processing model of joint attention and autism. *Autism Research*, 2, 2–21.
- Redcay, E., Dodell-Feder, D., Pearrow, M. J., Mavros, P. L., Kleiner, M., Gabrieli, J. D. E., et al. (2010). Live face-to-face interaction during fMRI: A new tool for social cognitive neuroscience. *NeuroImage*, 50, 1639–1647.
- Schilbach, L., Wilms, M., Eickhoff, S., Romanzetti, S., Tepest, R., Bente, G., Bente, G., et al. (2010). Minds

made for sharing: Initiating joint attention recruits reward-related neurocircuitry. *Journal of Cognitive Neuroscience*, 22(12), 2702–2715.

Schultz, R. (2005). Developmental deficits in social perception in autism: The roles of the amygdale and fusiform area. *International Journal of Developmental Neuroscience*, 23, 125–141.

Sigman, M., & McGovern, C. (2005). Improvements in cognitive and language skills from Preschool to adolescence in autism. *Journal of Autism and Developmental Disorders*, 35, 15–23.

Sigman, M., & Ruskin, E. (1999). Continuity and change in the social competence of children with autism, Down syndrome, and developmental delays. *Monographs of the Society for Research in Child Development*, 64 (1, Serial No. 256), 1–113.

Tomasello, M., Carpenter, M., Call, J., Behne, T., & Moll, H. (2005). Understanding sharing intentions: The origins of cultural cognition. *Brain and Behavior Sciences*, 28, 675–690.

Whalen, C., Schreibman, L., & Ingersol, L. (2006). The collateral effects of joint attention training on social initiations, positive affect, imitation, and spontaneous speech for young children with Autism. *Journal of Autism and Developmental Disorders*, 36, 655–664.

RLRS

- ▶ [Real-Life Rating Scale](#)
- ▶ [Ritvo-Freeman Real Life Rating Scale](#)

RMET

- ▶ [Reading the Mind in Eyes Test](#)

RO 5028442

- ▶ [Balovaptan](#)

RO-5285119

- ▶ [Balovaptan](#)

Robot-Assisted Cognitive Behavioral Therapy for Young Children with Autism Spectrum Disorders

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Definition

Robot-assisted therapy is a promising area of translational neuroscience for children with autism spectrum disorders (ASD). The robot-assisted cognitive behavioral therapy (RA-CBT) is a robot-assisted therapy focused on cognitive behavioral therapy (CBT), which shows promising evidence of efficacy and represents a viable intervention for children with ASD. This type of treatment consists of a human-assisted social robot acting as an intervention mediator to increase the emotional competence and to improve the socio-emotional understanding skills in children with ASD. RA-CBT can be used as a complementary therapy for ASD children with the potential to reduce the workload of the therapist and to improve the social skills.

Historical Background

Heterogeneity in clinical symptoms is the main characteristic of children with ASD, including high rates of comorbid medical conditions (e.g., sleep disorders, gastrointestinal dysfunction, autoimmune disorders), neurological conditions (such as seizures and sensory and motor system abnormalities), and psychiatric disorders (such as attention deficit hyperactivity disorder and anxiety).

Deficits in social cognition and emotional regulation are common in children with ASD (Leekam 2016; Berkovits et al. 2017). Impairments in social awareness, perspective-taking, and implicit theory of mind are reported regardless of their cognitive and language abilities (Leekam 2016), as well as difficulties in metacommunication and in understanding social contexts or maintaining reciprocal conversation (Sharma et al. 2018). Emotional dysregulation provokes social and behavioral difficulties over time (Berkovits et al. 2017), as well as issues in social success, academic readiness, and prosocial behaviors.

Despite ASD is a neurobiological disorder, behavioral treatments are currently the main tool for reducing disability and comorbidity because these are usually focused on maximizing the ability in social skills and communication (Lai et al. 2014). Social situations in particular contain a great amount of information, overwhelming ASD children, who have difficulty deciding which pieces of information are relevant. It has been suggested that the perception of biological motion is impaired in ASD, generating a cascade of consequences for social development (Klin et al. 2009).

Generally, various behavioral approaches exist for ASD patients and are classified into (Lai et al. 2014) (i) comprehensive applied behavioral analysis-based intensive intervention; (ii) targeted skill-based intervention (training in joint attention, teaching social skills, and social skill training); and (iii) targeted behavioral intervention for anxiety and aggression (cognitive behavioral therapy).

In particular, the treatment approach named cognitive behavioral therapy (CBT) is a psychotherapeutic method used in clinical settings to treat ASD, as well as other mental health disorders. In particular, CBT protocols contribute to improve the core evidences of ASD, i.e., social-behavioral adaptations, social skills, social anxiety, emotional regulation and aggression, co-occurring emotional and behavioral symptoms, and others.

All the abovementioned treatments promote the development of intelligence, communication, and adaptive function, as well as language, daily living skills, and socialization. However, ASD patients show marked heterogeneity at genetic,

behavioral, etiological, and pathophysiological levels, which interfere with the efficacy of intervention programs (Maglione et al. 2012). Moreover, it is well known that ASD individuals engage more successfully in social interactions if social information is presented in an “attractive” manner (Quirnbach et al. 2009). Therefore, there is a critical need for tools capable of increasing the effectiveness of standard (cognitive-) behavioral therapies (as well as reducing their cost).

The last two decades have seen the emergence of some technology-based therapies, including computer programs, virtual reality, and robot-assisted protocols. Robot-assisted therapy has gained attraction in the past few years to become an emerging and promising application area where researchers and clinicians have developed models with a wide range of appearances, features, and functional capabilities that draw from expertise in fields such as engineering and clinical psychology (Sartorato et al. 2017). Robot-mediated intervention studies have shown positive outcomes in areas of research such as social communication, joint attention, imitation, and social behaviors (Pennisi et al. 2016), in teaching with robots as an emulated peer (Yun et al. 2017), and in learning of socio-emotional understanding skills (Marino et al. 2020). Again, the robot-assisted therapy approach has naturally become more attractive since this is based on that simple observation that a child that shows no eye contact (the most basic social behavior) with a therapist could be successfully stimulated by social robot (Pennisi et al. 2016).

This kind of behavioral approach has two fundamental advantages: (a) the ability of robot to adaptively “learn” both inter-individual differences at one time point and intraindividual differences over time, thus partially overcoming the limitations due to clinical heterogeneity, and (b) the opportunity to record objective data during therapy. This latter characteristic is vital to characterize the progress of the child providing quantitative data about the developmental process and to partially automate the currently manual analysis (Thill et al. 2012).

Recently, social robots are considered effective tools to support interventions with group-based CBT for young children with ASD aimed at the development of higher-order emotional behavior

and cognition. In a first randomized controlled trial, a RA-CBT method was applied within an emotional understanding and social cognition treatment protocol for children with ASD (Marino et al. 2020). The RA-CBT study reported positive results on the efficacy of a robot-assisted therapy as effective augmentative mediator in CBT protocols for socio-emotional understanding, through a social robot able to provide cues, prompts, and reinforcements. Significantly, such a study has shown an increased ability in the experimental group to understand the beliefs, emotions, and thoughts of others.

Current Knowledge

Robot-assisted intervention demonstrated clear evidence in motivating the child in social situations (Pennisi et al. 2016), as other nonhuman agents supporting the therapy with children with ASD. Preliminary evidence of efficacy in assisting in the diagnostic process is reported. Other robot-assisted intervention studies have shown reduced repetitive, stereotyped behaviors, and more spontaneous language in children with ASD engaged in social interactions with a robot (Thill et al. 2012; Pennisi et al. 2016), showing better performances with a robot partner rather than a human one. Meaningfully, it has been demonstrated that robot-assisted intervention provides therapists and researchers a means to connect with ASD subjects in an easier way improving social communication skills, such as eye contact, joint attention, imitation, turn-taking, self-initiated interactions, emotion recognition, and triadic interactions (Bird et al. 2007; Cabibihan et al. 2013; Kim et al. 2013; Pennisi et al. 2016).

Anyway, clinical validation studies of robot-assisted protocols in enough heterogeneous clinical samples are still undergoing. Currently, the feasibility study of Mengoni et al. (2017) study clinically tested the effectiveness of a humanoid robot to support social skills in young children on the autism spectrum. The RCT study of Yun et al. (2017) administered a robot-assisted discrete trial teaching protocol to improve basic social skills. The RCT study of

Marino et al. (2020) administered a RA-CBT to improve socio-emotional skills in children with ASD. In term of emotional comprehension, this study has shown how a short robot-assisted cognitive behavioral protocol of only 15 h may shift the children's skills to the normative range (Marino et al. 2020). Only preliminary but promising studies are applying neuroimaging tools for extracting neural hallmarks of robot-assisted treatment with ASD subjects (Jung et al. 2016; Chaminade et al. 2012; Goulart et al. 2019). Preliminary evidence with portable EEG system (Chaminade et al. 2012) has shown significant brain activity changes in ASD child's brain before, during and after social robot interaction.

The most widely used artefacts for robot-assisted intervention with ASD are listed in Table 1.

Future Directions

RA-CBT can effectively be adopted in boosting learning of socio-emotional understanding skills. Actually, in order to strengthen the clinical validation of RA-CBT, the sample size must be increased. It is worth mentioning that in the robot-assisted state-of-the-art just the attentional engagement with the robot is measured. A future direction must be dedicated to stimulating quantitatively comparison of other variables, i.e., the number of exchanges, the contingency of reinforcements, the consistency of the stimuli, the engagement of a human therapist, and other measurements of administration reliability. Moreover, effectiveness of RA-CBT in studies dedicated to inferring differences in gender engagement and gender socio-emotional understanding skills should be envisaged. Moreover, it should be interesting to investigate any possible protective effect of RA-CBT-based emotional skills training against the negative social feedback experienced by almost all children with ASD during childhood.

From a neuroscientific point of view, significant progress has been made in understanding the multidimensional nature of ASD and the possible trajectories of neural progression (Masi et al. 2017). In particular, one of the targets to unravel

Robot-Assisted Cognitive Behavioral Therapy for Young Children with Autism Spectrum Disorders,
Table 1 Most used artifacts for robot-assisted intervention with ASD

Name	Company	Type	Characteristics
NAO	Aldebaran robotics company	Humanoid	Body in plastic with 25 DoF (4 joints for each arm; 2 for each hand; 5 for each leg; 2 for the head and one to control the hips). This is able to capture a lot of information about the environment using sensors and microphones. Nao can speak and assure a certain degree of non-verbal communication
QTrobot	LuxAi	Humanoid	QTrobot is an expressive little humanoid robot, designed and built based on scientific approaches to assist in teaching new skills to learners with autism and special educational needs. QTrobot supports learners to practice cognitive, social, communication, and emotional skills and helps them benefit more from educational sessions.
KASPAR	University of Hertfordshire's adaptive systems research group	Humanoid	Minimally expressive with 6 DoF on the head and neck, 6 on the arms, 2 in the eyes. Its face is a silicon-rubber mask. KASPAR's face can show a range of simplified expressions but in a less complex way than real human face. It is able to respond to the touch of children and can move its arms, head, and eyes
FACE	University of Pisa	Humanoid	It is a passive body with an active head. It has 32 motors to simulate and modulate six basic emotions (happiness, sadness, surprise, anger, disgust, and fear). FACE is not able to speak; it has microphones and cameras by which it can analyze the emotional reactions of individuals, react to them, and store all data
Tito	Wikifactory	Humanoid	It uses wheels to move, has two arms that can be moved up and down; it can turn its head right and left and up. It has two eyes and a mouth by which it can smile. Tito can speak by prerecorded vocal messages. Tito has a small microphone camera and can also be controlled by a wireless remote control
Bioid robot	ROBOTIS	Humanoid	Bioid robot can be assembled in various configurations (scorpion, spider, dinosaur, puppy, and humanoid robot). It can have 18 or 16 DoF; thus it has a wide body mobility, but its face is inexpressive. It does not speak
Pleo	Ugobe Inc	Animal-like	Dinosaur pet toy with 16 DoF, a camera-based vision system for light detection and navigation, microphones, touch sensors, ground foot sensors, force feedback sensors, an orientation tilt sensor for body position. It is able to move itself autonomously and can express emotions by motions and sounds in response to children's touch or various interactions such as caresses or giving food
LEGO	NXT	Toy	This is a programmable robotics kit developed by LEGO. The main component (NXT brick) can receive input from a maximum of 4 sensors and controls 3 motors. It can reproduce sampled sounds

the etiopathogenesis of ASD is the identification of potential neuro-endophenotypes (Gottesman et al. 2003), i.e., measurable neural system related subclinical heritable biological markers or behavioral traits that are internal phenotypic expressions

of a genotype. The advent of functional and structural neuroimaging techniques has provided a very powerful method to mapping the neural correlates of ASD-related subtypes, developmental trajectories, or progressive degeneration (Lau

et al. 2019). Through neuroimaging studies, a lot of neuro-endophenotypes have been proposed for ASD patients (Mahajan and Mostofsky 2015; Ruparel et al. 2017).

The introduction into clinical practice of robot-assisted therapy with ASD will lead to the adoption of effective applied neuroimaging tools able to identify the neural effects of such a treatment in different ASD-related subtypes while contributing to understand neural ASD profiles and pathophysiological mechanisms. Although the endophenotype-oriented neuroimaging research approach applied to robot-assisted therapy is in its relative infancy, it is definitely promising for future directions.

References and Reading

- Berkovits, L., Eisenhower, A., & Blacher, J. (2017). Emotion regulation in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 47(1), 68–79.
- Bird, G., Leighton, J., Press, C., & Heyes, C. (2007). Intact automatic imitation of human and robot actions in autism spectrum disorders. *Proceedings of the Royal Society B: Biological Sciences*, 274(1628), 3027–3031. <https://doi.org/10.1098/rspb.2007.1019>.
- Cabibihan, J. J., Javed, H., Ang, M., Jr., & Aljunied, S. M. (2013). Why robots? A survey on the roles and benefits of social robots in the therapy of children with autism. *International Journal of Social Robotics*, 5, 593–618. <https://doi.org/10.1007/s12369-013-0202-2>.
- Chaminade, T., Da Fonseca, D., Rosset, D., Lutchter, E., Cheng, G., & Deruelle, C. (2012). fMRI study of young adults with autism interacting with a humanoid robot. In *Proceedings of the 21st IEEE international symposium on robot and human interactive communication* (pp. 380–385). Paris: IEEE.
- Gottesman, I. I., Gould, T. D. T., Manschreck, T. C. T., & Pogue-Geile, M. F. M. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *The American Journal of Psychiatry*, 160(4), 636–645.
- Goulart, C., Valadão, C., Caldeira, E., & Bastos, T. (2019). Brain signal evaluation of children with autism Spectrum disorder in the interaction with a social robot. *Biotechnology Research and Innovation*, 3, 60–68.
- Jung, C. E., Strother, L., Feil-Seifer, D. J., & Hutsler, J. J. (2016). Atypical asymmetry for processing human and robot faces in autism revealed by fNIRS. *PLoS One*, 11(7), e0158804. <https://doi.org/10.1371/journal.pone.0158804>.
- Kim, E. S., Berkovits, L. D., Bernier, E. P., Leyzberg, D., Shic, F., Paul, R., & Scassellati, B. (2013). Social robots as embedded reinforcers of social behavior in children with autism. *Journal of Autism and Developmental Disorders*, 43(5), 1038–1049. <http://doi.org/10.1007/s10803-012-1645-2>.
- Klin, A., Lin, D. J., Gorrindo, P., Ramsay, G., & Jones, W. (2009). Two-year-olds with autism orient to non-social contingencies rather than biological motion. *Nature*, 459(7244), 257–261.
- Lai, M. C., Lombardo, M. V., & Baron-Cohen, S. (2014). Autism. *Lancet*, 383(9920), 896–910.
- Lau, W. K. W., Leung, M. K., & Lau, B. W. M. (2019). Resting-state abnormalities in autism spectrum disorders: A meta-analysis. *Scientific Reports*, 9(1), 3892.
- Leekam, S. (2016). Social cognitive impairment and autism: what are we trying to explain? *Phil. Trans. R. Soc. B*, 371(1686). <https://doi.org/10.1098/rstb.2015.0082>.
- Maglione, M. A., Gans, D., Das, L., Timbie, J., & Kasari, C. (2012). Nonmedical interventions for children with ASD: Recommended guidelines and further research needs. *Pediatrics*, 130(Suppl 2), S169–S178.
- Mahajan, R., & Mostofsky, S. H. (2015). Neuroimaging endophenotypes in autism spectrum disorder. *CNS Spectrums*, 20(4), 412–426.
- Marino, F., Chilà, P., Trusso Sfrassetto, S., Carrozza, C., Crimi, I., Failla, C., Busà, M., Bernava, G., Tartarisco, G., Vagni, D., Ruta, L., & Pioggia, G. (2020). Outcomes of a robot-assisted social-emotional understanding intervention for young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 50(6), 1973–1987. <https://doi.org/10.1007/s10803-019-03953-x>.
- Masi, A., DeMayo, M. M., Glozier, N., & Guastella, A. J. (2017). An overview of autism spectrum disorder, heterogeneity and treatment options. *Neuroscience Bulletin*, 33(2), 183–193. <https://doi.org/10.1007/s12264-017-0100-y>.
- Mengoni, S. E., Irvine, K., Thakur, D., Barton, G., Dautenhahn, K., Guldberg, K., Robins, B., Wellsted, D., & Sharma, S. (2017). Feasibility study of a randomised controlled trial to investigate the effectiveness of using a humanoid robot to improve the social skills of children with autism spectrum disorder (Kaspar RCT): A study protocol. *Paediatrics, BMJ Open*, 7, e017376.
- Nikopoulos, C. K., & Keenan, M. (2003). Promoting social initiation children with autism using video modeling. *Behavioral Interventions*, 18(2), 87–108.
- Pennisi, P., Tonacci, A., Tartarisco, G., Billeci, L., Ruta, L., Gangemi, S., & Pioggia, G. (2016). Autism and social robotics: A systematic review. *Autism Research*, 9(2), 165–183.
- Quimbach, L. M., Lincoln, A. J., Feinberg-Gizzo, M. J., Ingersoll, B. R., & Andrews, S. M. (2009). Social stories: Mechanisms of effectiveness in increasing game play skills in children diagnosed with autism spectrum disorder using a pretest posttest repeated measures randomized control group design. *Journal of Autism and Developmental Disorders*, 39(2), 299–321.

- Ruparelia, K., Manji, K., Abubakar, A., & Newton, C. R. (2017). Investigating the evidence of Behavioral, cognitive, and psychiatric Endophenotypes in autism: A systematic review. *Autism Research and Treatment*, 2017, 6346912. <https://doi.org/10.1155/2017/6346912>.
- Sartorato, F., Przybylowski, L., & Sarko, D. K. (2017). Improving therapeutic outcomes in autism spectrum disorders: Enhancing social communication and sensory processing through the use of interactive robots. *Journal of Psychiatric Research*, 9, 1–11. <https://doi.org/10.1016/j.jpsychires.2017.02.00>.
- Sharma, S. R., Gonda, X., & Tarazi, F. I. (2018). Autism spectrum disorder: Classification, diagnosis and therapy. *Pharmacology and Therapeutics*. <http://doi.org/10.1016/j.pharmthera.2018.05.007>.
- Thill, S., Pop, C. A., Belpaeme, T., et al. (2012). Robot-assisted therapy for autism spectrum disorders with (partially) autonomous control: Challenges and outlook. *Paladyn*, 3, 209–217.
- Yun, S. S., Choi, J., Park, S. K., Bong, G. Y., & Yoo, H. (2017). Social skills training for children with autism spectrum disorder using a robotic behavioral intervention system. *Autism Research*, 10(7), 1306–1323.

Role of Relationship Satisfaction in Parental Coping

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Definition

Autism Spectrum Condition (ASC) is considered one of the developmental disorders with the most pervasive and enduring impact on the whole family system. Studies have revealed a number of factors that are predictive of positive and negative family adaptation outcomes. Among these, the influences of sociodemographic and child characteristics on adaptation have been established. However, part of the family outcomes are not

explained by these factors but by parental and couple variables (García-López et al. 2016a, b).

Among parent resources and characteristics, studies highlight the importance of coping strategies and cognitions, such as sense of coherence, parental self-efficacy, or perception of positive contributions of disability in predicting positive adaptation outcomes. In the general population, couples' thoughts, feelings, and behaviors spread from one partner to the other; in the context of a stressful situation, these reciprocal effects might become even stronger. Couples raising individuals with ASC experience heightened levels of stress compared with controls and have to confront vital decisions for their children; therefore, it is expected that the couple relationship becomes a crucial factor affecting parents' psychological adaptation (García-López et al. 2016b). From a family systems perspective, the couple relationship is regarded as the nucleus around which the family functions and it has a pivotal role in creating a positive family environment (Minuchin 1985). Paradoxically, raising an individual with ASC can be both a challenging and rewarding experience. Despite reported challenges, many couples adapt positively to raising a child with ASC. This is consistent with family resilience theories, which postulate that adverse circumstances can lead to personal and relational transformations through the discovery of untapped resources and strengths.

Traditionally, the literature on family adaptation has focused on the negative impact of ASC on the couple relationship, with numerous studies describing relationship dissatisfaction and higher divorce or separation rates when compared with families without individuals with ASC (Hartley et al. 2010). However, research is increasingly highlighting the role of the couple relationship as a resource in families managing the challenges associated with raising an individual with ASC. This situation can create new opportunities for the couple to work together and share experiences that bring them closer and help them find new purposes, points of view, or values in life. In this context, the role of relationship satisfaction emerges as a key factor in the process of parental coping among families raising individuals with ASC.

Historical Background

From the general theories of stress and coping (Lazarus and Folkman 1984) and from models of family adaptation (McCubbin and Patterson 1983), different studies have reported robust evidence that shows that coping strategies play a protective role in families of individuals with ASC adaptation outcomes (Dabrowska and Pisula 2010; Hastings et al. 2005a). Coping strategies include behavioral or cognitive efforts aimed at reducing stress levels (Lazarus and Folkman 1984). Many classifications of coping strategies have been proposed. Among these, individual coping and dyadic coping have been two of the more explored methods of coping with stress.

Individual coping has been widely researched and is usually divided into problem or emotion-focused strategies. In the general population, emotion-focused coping (e.g., avoidance, engaging in ruminating, blaming oneself) has been positively correlated with health problems (e.g., anxiety and depression), whereas problem-focused coping (e.g., time management and obtaining social support) has been negatively correlated with these conditions. Within families raising children with ASC, the same pattern has been found, as parents who adopt active avoidance coping strategies, such as denial, distraction, or guilt, report more stress than those who implement positive and problem-focused strategies.

In contrast, dyadic coping has been relatively neglected, even though the literature on ASC-related parental adaptation recognizes the couple relationship as a primary source of support. In particular, in couples parenting individuals with ASC, the partner becomes a key source of both emotional and instrumental support for addressing the additional parenting demands associated with the disorder (García-López et al. 2016a). From a systemic-transactional perspective, dyadic coping involves coping strategies aimed at maintaining or restoring the structural, functional, behavioral, emotional, and social balance of the whole dyadic system, as well as the equilibrium of each partner. This theoretical framework is based on two key assumptions: first, stress and coping represent a dyadic

(systemic) phenomenon; second, dyadic coping with stress includes both stress expression and dyadic support. Two types of stress can be differentiated: relationship stress and external stress. The former is derived from the relationship as a result of differing goals or desires, whereas the latter starts outside the relationship due to work strains or conflicts. Within this framework, dyadic coping is considered one partner's attempt to help reduce the external stress perceived by their partner and a common endeavor to cope with stress that originates inside the relationship. The stress coping process is regarded as a sequence consisting of stress expression by the stressed person, the perception of stress by the partner, and the partner's coping reaction to the stressed person's behavior (Bodenmann 2000).

Dyadic coping can be either positive or negative. Positive forms of dyadic coping refer to supportive dyadic coping (such as providing practical advice or offering empathic understanding), common dyadic coping (such as joint problem solving, sharing feelings, or relaxing together), and delegated dyadic coping (such as one partner explicitly asking the other for support and consequently agreeing a new division of tasks). Negative dyadic coping includes hostile dyadic coping (such as distancing, mocking, or minimizing the seriousness of the partner's stress), ambivalent dyadic coping (such as one partner supporting the other reluctantly, as if their contribution was unnecessary), and superficial dyadic coping (such as asking questions about the partner's feelings without listening or providing support that lacks empathy) (Bodenmann and Cina 2005). The type of dyadic coping strategies used depends on variables such as the partner's individual and dyadic competences, motivational factors, and the characteristics of the situation in which the stress occurs. Dyadic coping is associated with the partner's level of relationship quality (Bodenmann 2000), relationship stability, and communication behavior (Bodenmann and Cina 2000). Effective dyadic coping not only reduces stress and improves partnership well-being but also strengthens a feeling of "we-ness" in the partners.

The concept of dyadic coping has been argued to be especially relevant to the prevention of

relationship deterioration and marital therapy. This idea has been supported by dyadic coping being strongly associated with partnership satisfaction. According to Bodenmann (2005), the relationship between dyadic coping and marital satisfaction can be facilitated through two separate mechanisms. First, dyadic coping works as a moderator between stress and partnership satisfaction. Second, dyadic coping strengthens feelings of mutual trust and intimacy, and the cognitions that the relationship is helpful and supportive.

In the general population, stress, dyadic coping, and the couple's relationship are related in multiple ways. First, stress reduces the time partners spend together, which implies having less time for dyadic coping. Second, stress decreases the quality of dyadic communication. Under stress, couples use more negative communication patterns, which are in turn predictive of poor relationship functioning and divorce. Third, chronic stress increases the risk of physical and mental health problems, limiting the opportunities for pleasant activities, confronting equity of roles between the partners, thus reducing marital satisfaction. Chronic stress can also originate outside the couple and spill over the relationship, decreasing the quality of dyadic coping. So, the more effectively each partner copes with external stress, the more they can reduce the probability of spill-over, protecting the couple's relationship from the impact of external stress. Consequently, both individual and partnership coping are considered to be protective factors of relationship satisfaction (Bodenmann 2005).

In summary, there are many ways of coping with stress. Dyadic coping, rooted in the transactional stress and coping theory, and nourished by family adaptation models and empirical studies, is one of these methods. This type of coping involves managing stress as a couple and is strongly associated with relationship satisfaction.

Relationship Satisfaction and Coping throughout its Lifespan

In the general population, a U-shaped pattern of partnership happiness over its duration has been described: younger and older partners are more

satisfied with their relationship when compared to those who are middle-aged. This decrease in relationship quality in non-ASC families is usually linked to the arrival of children, which often involves spending less time together, experiencing more conflicts, or having to make more decisions for couples (Landis et al. 2013; Hock et al. 2012).

Having a child with special needs complicates the transition to parenthood by increasing parental stress and anxiety. This transition sets a period characterized by an increased focus on the needs of the child, to the detriment of the needs of the couple. The couple's relationship often suffers due to the intense demands and energy required to meet the special child's needs. This change in focus away from the relationship may put the couple at risk for problems, as they might not share enough time to grow together as a couple and so experience increased stress in their lives.

Couples raising an individual diagnosed with ASC may experience high levels of stress, which is linked to lower relationship satisfaction. The increase in stress levels begins at the initial diagnosis stage and continues throughout the different stages of the life cycle, since each stage presents different challenges (Pozo and Sarriá 2015). As a result, it is not expected that the U-shaped pattern that occurs in the general population will be reproduced in families raising individuals with ASC. Evidence suggests a continued decline in relationship satisfaction through childhood and across the child's transition to adulthood (Hartley et al. 2012), a period when other parents are usually enjoying an upturn in satisfaction.

In contrast, some authors have regarded ASC as a crucible for parents, with three different phases in the course of the couple's relationship: the ASC crucible, tag team, and deeper intimacy and commitment. The first phase (ASC crucible) is considered a test for the couple's relationship as the child is regarded as a stressor and parents describe a dichotomous reaction: either fight for the relationship or end it. During the second phase (tag team), parents are focused on their parenting role, which requires reorganizing family and professional roles, and the couple's relationship is regarded as secondary. The last phase (deeper

intimacy and commitment) involves renovating the marital commitment as parents realize that looking after the relationship is beneficial to the ASC child (Hock et al. 2012).

Current Knowledge

Findings regarding the role of relationship satisfaction on parental coping in families raising individuals with ASC draw a paradoxical picture. Taking care of an individual with ASC places an additional stress on the couple's relationship, which challenges its stability and capacity to cope with the situation. Relationship satisfaction may serve as a protective factor that enhances positive parental coping and adaptation.

In addition to the usual relationship and external stress that characterizes any couple, parents raising individuals with ASC have to address new sources of stress derived from managing the practical, emotional, and financial demands associated with ASC. Thus, parents of individuals with ASC experience numerous challenges both as individuals and as couples that may change their everyday life and long term outlook. Parents raising individuals with ASC present more psychological problems such as stress, anxiety, or depression, lower dyadic consensus, and lower levels of relationship satisfaction compared with parents of typically developing children (Hayes and Watson 2013).

The cumulative stress posed on a relationship could ultimately lead to its dissolution. However, findings are mixed when it comes to divorce rates, as some studies show it is higher than in the general population (Hartley et al. 2010), whereas other studies have found no significant differences (Freedman et al. 2012). Conversely, relationship satisfaction may not be as drastically undermined by the challenge of rearing a child with ASC as has commonly been supposed. Rather than considering relationship satisfaction as an outcome measure that might be compromised as a consequence of dealing with ASC, the quality of the relationship is increasingly being regarded as a protective factor mediating the link between the child's disability and parents' emotional well-

being. Particularly, couples who report relationship satisfaction also report less parenting stress, greater use of positive dyadic coping strategies, and less use of negative dyadic coping strategies than couples who are dissatisfied in the relationship with their partner. Satisfied couples are more likely to engage in positive rather than negative dyadic coping strategies than dissatisfied couples (Sim et al. 2017). Conversely, positive dyadic coping contributes to maintaining relationship satisfaction, which in turn positively impacts parental adaptation (García-López et al. 2016a). Consequently, relationship satisfaction and positive dyadic coping are bidirectionally associated and act as protective factors in the process of parents' psychological adaptation.

Future Directions

Despite the importance of relationship satisfaction in family and child outcomes, few studies have explored its role as a coping strategy in the process of parental adaptation. Consequently, there are several implications regarding relationship satisfaction and coping in parents of individuals with ASC for the future at both the research and clinical levels.

Future research should prioritize family-centered approaches, since they are considered to be best practices. Special attention should be paid to the relationship as a protective factor in future empirical studies, as it has been associated with improved child behaviors, reduced stress, and positive adjustment in families raising individuals with ASC (Benson and Kersh 2011; Hartley et al. 2012). Another field of research that merits further exploration refers to sex differences regarding the role relationship satisfaction on parental coping. Findings so far suggest that mothers use different individual coping strategies to manage the stress derived from ASC when compared to fathers (Hastings et al. 2005b), but it remains unclear what role sex plays when the couple relationship is regarded as a method to cope with stress.

On the clinical level, as couples raising individuals with ASC are considered to be at high risk

of poor relationship satisfaction, it is crucial for clinicians to evaluate and monitor the quality of the couple's relationship, providing specific psychoeducation programs for couples. Professionals should raise awareness about the importance of maintaining the couple relationship and enhancing dyadic coping during stressful times. Specifically, the programs should be aimed at training dyadic coping strategies and strengthening relationship satisfaction, which are both variables that can be modified through intervention. Special attention should be paid to gender differences and needs when designing and implementing these family-centered interventions. In summary, in an era when family involvement in treatment programs is pivotal, looking after the partnership well-being should be a prerequisite to implementing any parent-mediated intervention.

Overall, despite an increased risk of poor relationship satisfaction in couples raising individuals with ASD, evidence shows that many couples overcome this situation and grow stronger, taking advantage of the couple relationship as a coping strategy.

See Also

► Autism Speaks

References and Reading

- Benson, P. R., & Kersh, J. (2011). Marital quality and psychological adjustment among mothers of children with ASD: Cross-sectional and longitudinal adjustment. *Journal of Autism and Developmental Disorders*, 41, 1675–1685. <https://doi.org/10.1007/s10803-011-1198-9>.
- Bodenmann, G. (2000). *Stress und coping bei paaren* (Stress and coping in couples). Göttingen: Hogrefe.
- Bodenmann, G. (2005). Dyadic coping and its significance for marital functioning. In T. A. Revenson, K. Kayser, & G. Bodenmann (Eds.), *Couples coping with stress: Emerging perspectives on dyadic coping* (pp. 33–49). Washington, DC: American Psychological Association.
- Bodenmann, G., & Cina, A. (2000). Stress und Coping als Prädiktoren für Scheidung: Eine prospektive Fünf-Jahres-Längsschnittstudie [Stress and coping as predictors of divorce: A 5-year prospective study]. *Zeitschrift für Familienforschung*, 12, 5–20.
- Bodenmann, G., & Cina, A. (2005). Stress and coping among stable-satisfied, stable-distressed and separated/divorced Swiss couples: A 5-year prospective longitudinal study. *Journal of Divorce and Remarriage*, 44(1), 71–89. https://doi.org/10.1300/J087v44n01_04.
- Dabrowska, A., & Pisula, E. (2010). Parenting stress and coping styles in mothers and fathers of pre-school children with autism and down syndrome. *Journal of Intellectual Disability Research*, 54(3), 266–280. <https://doi.org/10.1111/j.13652788.2010.01258.x>.
- Freedman, B. H., Kalb, L. G., Zablotzky, B., & Stuart, E. A. (2012). Relationship status among parents of children with autism spectrum disorders: A population-based study. *Journal of Autism and Developmental Disorders*, 42, 539–548. <https://doi.org/10.1007/s10803-011-1269-y>.
- García-López, C., Sarriá, E., Pozo, P., & Recio, P. (2016a). Supportive dyadic coping order: The role of relationship satisfaction. *Journal of Autism and Developmental Disorders*, 46, 3434–3447. <https://doi.org/10.1007/s10803-016-2883-5>.
- García-López, C., Sarriá, E., & Pozo, P. (2016b). Parental self-efficacy and positive contributions regarding autism spectrum condition: An actor-partner interdependence model. *Journal of Autism and Developmental Disorders*, 46(7), 2385–2398. <https://doi.org/10.1007/s10803-016-2771-z>.
- Hartley, S. L., Barker, E. T., Seltzer, M. M., Floyd, F., Greenberg, J., Orsmond, G., & Bolt, D. (2010). The relative risk and timing of divorce in families of children with an autism spectrum disorder. *Journal of Family Psychology*, 24(4), 449–457. <https://doi.org/10.1037/a0019847>.
- Hartley, S. L., Barker, E. T., Baker, J. K., Seltzer, M. M., & Greenberg, J. S. (2012). Marital satisfaction and life circumstances of grown children with autism across 7 years. *Journal of Family Psychology*, 26, 688–697. <https://doi.org/10.1037/a0029354>.
- Hastings, R. P., Kovshoff, H., Ward, N. J., Espinosa, F. D., Brown, T., & Remington, B. (2005a). Systems analysis of stress and positive perceptions in mothers and fathers of pre-school children with autism. *Journal of Autism and Developmental Disorders*, 35(5), 635–644. <https://doi.org/10.1007/s10803-005-0007-8>.
- Hastings, R. P., Kovshoff, H., Brown, T., Ward, N. J., degli Espinosa, F., & Remington, B. (2005b). Coping strategies in mothers and fathers of preschool and school-age children with autism. *Autism*, 9, 377–391.
- Hayes, S. A., & Watson, S. L. (2013). The impact of parenting stress: A meta-analysis of studies comparing the experience of parenting stress in parents of children with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43, 629–642. <https://doi.org/10.1007/s10803-012-1604-y>.
- Hock, R. M., Timm, T. M., & Ramisch, J. L. (2012). Parenting children with autism spectrum disorders:

- A crucible for couple relationships. *Child & Family Social Work*, 17(4), 406–415. <https://doi.org/10.1111/j.1365-2206.2011.00794.x>.
- Landis, M., Peter-Wight, M., Martin, M., & Bodenmann, G. (2013). Dyadic coping and marital satisfaction of older spouses in long-term marriage. *GeroPsych*, 26, 39–47. <https://doi.org/10.1024/1662-9647/a000077>.
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.
- McCubbin, H. I., & Patterson, J. M. (1983). The family stress process: The double ABCX model of adjustment and adaptation. In H. I. McCubbin, M. B. Sussman, & J. M. Patterson (Eds.), *Social stress and the family: Advances and developments in family stress theory and research* (pp. 7–37). New York: The Haworth Press.
- Minuchin, P. (1985). Families and individual development: Provocations from the field of family therapy. *Child Development*, 56, 289–302. <https://doi.org/10.2307/1129720>.
- Pozo, P., & Sarriá, E. (2015). Still stressed but feeling better: Wellbeing in autism spectrum disorder families as children become adults. *Autism*, 19(7), 805–813. <https://doi.org/10.1177/1362361315583191>.
- Sim, A., Cordier, R., Vaz, S., Parsons, R., & Falkmer, T. (2017). Relationship satisfaction and dyadic coping in couples with a child with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47, 3562–3573. <https://doi.org/10.1007/s10803-017-3275-1>.

Role Release

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Synonyms

Child and family-centered intervention; Collaborative consultation; Collaborative intervention; Collaborative teaming; Cross-training; Interdisciplinary assessment and intervention; Intervention activity; Multi-skilling; Transdisciplinary assessment and intervention; Transfer of function

Definition

Role release is the transfer or sharing of a specific task, activity, or intervention practice usually

performed by a discipline-specific member of the educational or therapeutic team to another person. The task or activity delegated should meet certain conditions: the task or activity does not require discipline-specific training or licensure to perform, the task or activity is trained and the appropriate implementation of the activity is supervised by the responsible team member, and the task or activity is done on behalf of a specific child under the direction of the professional. Examples of areas of role release or cross-training include (1) management and administrative tasks related to programmatic activities such as scheduling and quality improvement; (2) nonclinical skills and activities including parent education, case management, and technical writing; (3) basic care activities including activities of daily living, monitoring of medications and vital signs, and noninvasive medical activities such as suctioning; and (4) clinical specific activities including intervention activities that can be cross-trained and monitored by the professional such as parent-led language stimulation and cross-disciplinary implementation of activities that facilitate and support a discipline-specific goal.

The concept of role release or cross-training of other personnel to manage or provide aspects of management and intervention activities has its roots in both health care and education. Various terms are utilized by the different disciplines to describe the collaborative effort in sharing responsibility for a child's therapeutic or educational needs including *collaborative consultation*, *collaborative intervention*, *collaborative teaming*, *interdisciplinary assessment and intervention*, *transdisciplinary assessment and intervention*, and *child and family-centered intervention*.

See Also

► Transactional Support (SCERTS) Model

References and Reading

- American Speech-Language-Hearing Association. (1997). Position statement: Multi-skilled personnel. *ASHA*, 39.

- Hunt, P., Soto, G., Maier, J., Liboiron, N., & Bae, S. (2004). Collaborative teaming to support pre-schoolers with severe disabilities who are placed in general education early childhood programs. *Topics in Early Childhood Special Education, 24*, 123–142.
- Prelock, P. (2000). Multiple perspectives for determining the roles of speech-language pathologists in inclusionary classrooms. *Language, Speech, and Hearing Services in Schools, 31*, 213–218.
- Prelock, P., Beatson, J., Bitner, B., Broder, C., & Ducker, A. (2003). Interdisciplinary assessment of young children with autism spectrum disorder. *Language, Speech, and Hearing Services in Schools, 34*, 194–202.
- Rainforth, B., & York-Barr, J. (1997). *Collaborative teams for students with disabilities: Integrating therapy and educational services*. Baltimore: Paul H. Brookes.
- Villa, R. A., Thousand, J. S., Nevin, A. I., & Malgeri, C. (1996). Instilling collaboration for inclusive schooling as a way of doing business in public schools. *Remedial and Special Education, 17*, 169–181.

Romanian Adoptive Children

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Definition

Some of the thousands of children (about 10%) who were adopted by Western families from destitute and overcrowded Romanian institutions during the 1990s revealed autistic-like symptoms pertaining to the triad of core autism deficits in social interaction, communication, and interests/activities. Although it is well known today that autism has a strong genetic component and is not considered a common consequence of severe early deprivation, researchers reported a high prevalence of autistic-like symptoms in these adopted children who had been institutionally reared and had experienced prolonged severe neglect in their early years (Hoksbergen et al. 2005; Rutter et al. 1999, 2007). Indeed, researchers attributed these autistic-like behaviors directly to the children's impoverished early environmental factors (e.g., extreme neglect, prolonged institutional care), and the provision of caring conditions for these impoverished children

led to significant developmental progress and even to the disappearance of their autistic-like deficits and delays (Rutter and Team 1998). This progress starkly contrasts with the progress trajectories of children with autism diagnoses.

Some of the adopted Romanian children showed typical features of autism as well as atypical features of autism in the preschool years. They exhibited difficulties in communication such as few reciprocal interchanges or poor conversational flow. However, their spontaneous usage of social bids and language was more flexible than that of children with autism. Adopted children who showed features of autism demonstrated inadequate social responsiveness and indiscriminate friendliness to strangers, which are both associated with reactive attachment disorder and disinhibited attachment style (Rutter et al. 2007). They also revealed repetitive and stereotyped behaviors such as intense interests and abnormal preoccupations, as well as stereotyped motor behaviors. Interestingly, as these children grew older, their typical autism-symptom manifestations became milder but were still notable. Neither the autistic symptoms' different quality nor their transient course is common in children with autism; therefore, this syndrome was termed "quasi-autistic" or "postinstitutional autistic syndrome" (Hoksbergen et al. 2005; Rutter et al. 1999, 2007).

Historical Background

Many psychological theories and clinical studies (e.g., Jean Piaget, Lev Vygotsky, John Bowlby, and René Spitz) have emphasized the importance of infants' early experiences in the first years of life for psychological development and personality characteristics. The availability of physical and social stimulation, the warm, intimate, and continuous relationship with the primary caregiver, is considered a necessary condition for infants' psychological well-being and social/emotional growth. Early studies on institutional rearing revealed that deprivation in early experiences and emotional relatedness renders a substantial impact on the intellectual, emotional, and social

development of infants and children. Institutional environments, which offer inadequate conditions for acquiring and practicing new skills, limited opportunities for reinforcement and praise, and few prospects for variation and adaptation to individual needs or differences have been associated with children's developmental delays, social/emotional unresponsiveness, and long-term psychological effects (Colvert et al. 2008; Fisher et al. 1997; Kreppner et al. 2007; Marcovitch et al. 1995; Morison et al. 1995; Rutter and Team 1998).

Due to the Romanian government's policy of outlawing birth control and abortions for women of childbearing age with fewer than five children, an estimated 100,000–300,000 children were separated from their parents who were unable to support and care for them. Even from birth, children were placed in long-term state-care institutions across Romania, which were usually impoverished and overcrowded, with a low caregiver-to-child ratio and rapid turnover in caregivers. This early deprived environment offered children little opportunity for social stimulation and personal contact in the first years of life and rendered a profound effect on children's attachment relationships and psychological development. During the early 1990s, after Western media exposure to these orphanages, large numbers of Romanian children were adopted by parents in Western countries, both for humanitarian reasons and for parents who were unable to have children of their own.

The first studies to examine the adjustment and development of Romanian adoptees, conducted shortly after adoption, reported medical problems (e.g., malnutrition, infections), eating problems, sleeping difficulties, developmental delays (e.g., in speech, language, gross and fine motor skills), stereotypical behaviors, attachment difficulties (e.g., clinging behaviors, indiscriminate approaches to strangers, overfriendliness), tantrums, hyperactivity, anxieties, and difficulties interacting with peers (Fisher et al. 1997; Marcovitch et al. 1995; Morison et al. 1995; Rutter and Team 1998). Later follow-up studies showed, importantly, that the majority of children made remarkable progress after spending time

with their adoptive families, and most of the reported difficulties tended to improve over time (Kreppner et al. 2007; Beckett et al. 2006). However, some adopted children revealed long-term effects of deprivation on psychosocial development (as will be discussed next). Most of the adoptive parents in these early studies pinpointed the importance of supportive professionals and programs like infant/child development clinics, adoption support groups, and networks of other families adopting Romanian children (Fisher et al. 1997).

Current Knowledge

Several research groups in Canada, the UK, and the Netherlands followed the development of children adopted from Romanian orphanages (Chisholm 1998; Rijk et al. 2010; Rutter et al. 2010; The St. Petersburg-USA Orphanage Research Team 2008). The English and Romanian Adoptees (ERA) study (Rutter et al. 2010) followed 165 young children adopted from profoundly deprived Romanian institutions who moved to England in the early 1990s. This group was compared to a sample of 52 nondeprived UK-born children who were adopted before the age of 6 months. So far, the children participating in this project were examined at the ages of 4, 6, 11, and 15 years, and those with autism-like symptoms were also seen at age 18–20. The children's cognitive, social, behavioral, and emotional functioning was evaluated at each time point, including specific diagnostic measures for assessing autism such as Autism Diagnostic Interview-Revised (ADI-R; Rutter et al. 2003) and Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2002).

Findings indicated a major catch-up during the preschool years; however, for some adoptees, cognitive and psychosocial difficulties were found during childhood and adolescence. The rate of deficit was significantly higher in children who stayed in deprived institutions for more than 6 months, and it persisted even more than 10 years after adoption. The Romanian adoptees' head circumference, as a measure of brain

growth, was below population norms at all ages, suggesting its possibly important role in mediating the effect of institutional deprivation on cognitive and psychosocial functioning. Interestingly, poor nutrition seemed to play a rather marginal role regarding effects on later development, whereas even very minimal language skills at the time of adoption were strongly associated with higher verbal and nonverbal IQ scores, suggesting language as some kind of a protective factor (Rutter et al. 2010).

Among these adoptees, Rutter et al. (2010) found evidence for specific outcome patterns, or a “syndrome,” associated with early deprived environment comprising quasi-autism symptoms as well as disinhibited attachment, inattention/overactivity problems, and cognitive impairments. About 10% of the Romanian adoptees who experience impoverished institutional care revealed quasi-autism symptoms compared to none of the UK adoptees. The Romanian adoptees also revealed greater deficits in executive functions and especially in theory of mind compared to the control group, both strongly associated with autism. It was suggested that these two deficits may play a significant role as mediators between institutional deprivation and quasi-autism symptoms and that institutional deprivation often led to impaired theory-of-mind abilities even when the quasi-autism symptoms were not present (Colvert et al. 2008). The longitudinal analyses of the quasi-autism patterns revealed that some of the autistic features faded over time, and in adulthood, some of the individuals with quasi-autism symptoms revealed emotional and/or behavioral disturbances such as anxieties, aggressive behaviors, as well as occurrences of outstanding skills, talents, and achievements. Furthermore, it was found that all adoptees with quasi-autism symptoms received either mental health or special education services.

The effect of early deprivation on brain structure was recently examined in a small sample of adolescents who participated in the ERA study. Using magnetic resonance imaging (MRI), significantly reduced brain volumes as well as larger relative amygdala volumes were found in the Romanian adoptee group compared

to a nonadopted matched control group, indicating possible dysfunctions in affective processing. The examination of the hippocampal and corpus callosum areas, which also play an important role in basic emotional processing and social engagement, did not reveal significant differences between these two groups, reflecting the possibility that such effects may emerge later in life (Mehta et al. 2009). Evidence for increased cortisol levels found in Romanian adoptees also supports the role of the limbic-hypothalamic-pituitary-adrenal axis in this population, reflecting the effects of prolonged stress and adversity on alterations in neuroendocrine functioning (Gunnar et al. 2001). More evidence regarding the effects of early deprivation on brain structure is coming from animal models. Severe stereotyped motor behaviors are believed to be caused by the restricted sensory environment with limited typical input. It was suggested that stereotyped motor behaviors in deprived populations serve significant functions of self-stimulating, self-soothing, or expressions of emotions such as frustration or anxiety and are associated with alterations in the cortical-basal ganglia circuitry (Bos et al. 2010).

Studies regarding gene-environment interactions have demonstrated that the association between severe deprived institutional care in the early years and the presence of long-term psychiatric problems and emotional and behavioral difficulties is mediated by genotype. More specifically, the gene coding of one of the key regulators of serotonin function in the brain – the serotonin transporter protein (5-HTT) – was detected as an important candidate to moderate the effect of adverse life stressors on emotion regulation and stress responsiveness (Kumsta et al. 2010). In addition, the dopamine transporter gene (DAT1) was also found as a possible candidate to moderate the effect of severe early deprivation and ADHD symptoms (Stevens et al. 2009). Yet, no studies have investigated brain structure or candidate genes that may moderate the associations between deprived early child-rearing environments and autistic-like symptoms.

Future Directions

The study of children who were raised in deprived institutions is considered a “natural experiment” that allows empirical scrutiny of the effects of extreme early deprivation on the development of cognitive and social/emotional functioning including the presence of autistic features. More investigations are needed to explore and identify moderating variables and factors that are responsible for the substantial heterogeneity in outcomes. For example, further genetic and biological studies using structural and functional brain imaging are needed to explore relevant genes and brain mechanisms as possible mediators for specific outcome characteristics of these adopted children, alongside analyses of variations in the quality of adoptive child-rearing environments.

See Also

- [ADHD](#)
- [Attachment Disorder](#)
- [Cognitive Skills](#)
- [Reactive Attachment Disorder](#)

References and Reading

- Beckett, C., Maughan, B., Rutter, M., Castle, J., Colvert, E., Groothues, C., et al. (2006). Do the effects of early severe deprivation on cognition persist into early adolescence? Findings from the English and Romanian adoptees study. *Child Development*, 77(3), 696–711.
- Bos, K. J., Zeanah, C. H., Smyke, A. T., Fox, N. A., & Nelson, C. A. (2010). Stereotypes in children with a history of early institutional care. *Archives of Pediatrics & Adolescent Medicine*, 164(5), 406–411.
- Chisholm, K. (1998). A three year follow-up of attachment and indiscriminate friendliness in children adopted from Romanian orphanages. *Child Development*, 69(4), 1092–1106.
- Colvert, E., Rutter, M., Beckett, C., Castle, J., Groothues, C., Hawkins, A., et al. (2008a). Emotional difficulties in early adolescence following severe early deprivation: Findings from the English and Romanian adoptees study. *Development and Psychopathology*, 20(2), 547–567.
- Colvert, E., Rutter, M., Kreppner, J., Beckett, C., Castle, J., Groothues, C., et al. (2008b). Do theory of mind and executive function deficits underlie the adverse outcomes associated with profound early deprivation? Findings from the English and Romanian adoptees study. *Journal of Abnormal Child Psychology*, 36(7), 1057–1068.
- Fisher, L., Ames, E. W., Chisholm, K., & Savoie, L. (1997). Problems reported by parents of Romanian orphans adopted to British Columbia. *International Journal of Behavioral Development*, 20(1), 67–82.
- Gunnar, M. R., Morison, S. J., Chisholm, K., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages. *Development and Psychopathology*, 13(3), 611–628.
- Hoksbergen, R., ter Laak, J., Rijk, K., van Dijkum, C., & Stoutjesdijk, F. (2005). Post-institutional autistic syndrome in Romanian adoptees. *Journal of Autism and Developmental Disorders*, 35(5), 615–623.
- Kreppner, J. M., Rutter, M., Beckett, C., Castle, J., Colvert, E., Groothues, C., et al. (2007). Normality and impairment following profound early institutional deprivation: A longitudinal follow-up into early adolescence. *Developmental Psychology*, 43(4), 931–946.
- Kumsta, R., Stevens, S., Brookes, K., Schlotz, W., Castle, J., Beckett, C., et al. (2010). 5HTT genotype moderates the influence of early institutional deprivation on emotional problems in adolescence: Evidence from the English and Romanian Adoptee (ERA) study. *Journal of Child Psychology and Psychiatry*, 51(7), 755–762.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2002). *Autism diagnostic observation schedule* (WPS edn.). Los Angeles: Western Psychological Services.
- Marcovitch, S., Cesaroni, L., Roberts, W., & Swanson, C. (1995). Romanian adoption – parents’ dreams, nightmares, and realities. *Child Welfare*, 74(5), 993–1017.
- Mehta, M. A., Golembo, N. I., Nosarti, C., Colvert, E., Mota, A., Williams, S. C. R., et al. (2009). Amygdala, hippocampal and corpus callosum size following severe early institutional deprivation: The English and Romanian Adoptees Study Pilot. *Journal of Child Psychology and Psychiatry*, 50(8), 943–951.
- Morison, S. J., Ames, E. W., & Chisholm, K. (1995). The development of children adopted from Romanian orphanages. *Merrill-Palmer Quarterly-Journal of Developmental Psychology*, 41(4), 411–430.
- Rijk, C., Hoksbergen, R. A. C., & ter Laak, J. (2010). Development of behavioural problems in children adopted from Romania to the Netherlands, after a period of deprivation. *The European Journal of Developmental Psychology*, 7(2), 233–248.
- Rutter, M., & English and Romanian Adoptees (ERA) Study Team. (1998). Developmental catch-up, and deficit, following adoption after severe global early privation. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 39(4), 465–476.
- Rutter, M., Andersen-Wood, L., Beckett, C., Bredenkamp, D., Castle, J., Groothues, C., et al. (1999). Quasi-autistic patterns following severe early global privation. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 40(4), 537–549.
- Rutter, M., Le Couteur, A., & Lord, C. (2003). *ADI-R: autism diagnostic interview-revised: Manual*. Western Psychological Services.

- Rutter, M., Colvert, E., Kreppner, J., Beckett, C., Castle, J., Groothues, C., et al. (2007a). Early adolescent outcomes for institutionally-deprived and non-deprived adoptees. I: Disinhibited attachment. *Journal of Child Psychology and Psychiatry*, 48(1), 17–30.
- Rutter, M., Kreppner, J., Croft, C., Murin, M., Colvert, E., Beckett, C., et al. (2007b). Early adolescent outcomes of institutionally deprived and non-deprived adoptees. III. Quasi-autism. *Journal of Child Psychology and Psychiatry*, 48(12), 1200–1207.
- Rutter, M., Sonuga-Barke, E. J., Beckett, C., Castle, J., Kreppner, J., Kumsta, R., Schlotz, W., Stevens, S., & Bell, C. A. (2010). Deprivation-specific psychological patterns: Effects of institutional deprivation. *Monographs of the Society for Research in Child Development*, 75(1), 1–252.
- Spitz, R. A. (1945). Hospitalism: An inquiry into the genesis of psychiatric conditions in early childhood. *The Psychoanalytic Study of the Child*, 1, 53–74.
- St. Petersburg-USA Orphanage Research Team. (2008). The effects of early social-emotional and relationship experience on the development of young orphanage children. *Monographs of the Society for Research in Child Development*, 73(1), 1–297.
- Stevens, S. E., Kumsta, R., Kreppner, J. M., Brookes, K. J., Rutter, M., & Sonuga-Barke, E. J. S. (2009). Dopamine transporter gene polymorphism moderates the effects of severe deprivation on ADHD symptoms: Developmental continuities in gene-environment interplay. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 150B(6), 753–761.

Rossetti Infant-Toddler Language Scale

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Synonyms

[Rossetti Infant-Toddler Language Scale \(The Rossetti\)](#)

Description

The *Rossetti Infant-Toddler Language Scale* (Rossetti 2006) was designed as a comprehensive measure of the communication skills of children, birth to age 3 years. Using a combination of

observation, direct child assessment, and caregiver report, the scale is intended to tap preverbal and verbal communication and interaction skills in six domains. These include the following: Interaction-Attachment (qualities of the caregiver-infant interaction and relationship), Pragmatics (use of language in social situations), Gesture (use of gestures to communicate), Play (use of representational thought and symbolism), Language Comprehension (understanding of language), and Language Expression (production of language).

The results of the *Rossetti* are intended to reflect the child's mastery of skills in each domain and are assessed according to 3-month age intervals. The number of items for each subtest varies by age, and only those domains for which reliable and discriminating items were deemed available appear at each interval. For example, for the age interval 0–3 months, all domains are represented except Gesture (because none are expected). By contrast, only the domains of Play, Language Comprehension, and Language Expression are tapped between 33 and 36 months because only items for these domains are believed to discriminate on the basis of age.

Materials provided in the Rossetti (2006) include a score sheet, caregiver questionnaire (in English and Spanish), and Examiner's Manual. A lengthy list of additional items (e.g., spoons, balls, dolls, picture stimuli) required to administer the *Rossetti* is given in the Appendix of the Examiner's Manual.

Only professionals with knowledge of child development and communication skills are to administer the *Rossetti*. To begin the evaluation, caregivers are asked to complete the caregiver questionnaire. A parental interview guide is provided in the manual, which elaborates on the questionnaire. The practitioner may use the questionnaire and/or whatever portions of the parental interview are deemed appropriate. Completion of the caregiver questionnaire and/or interview is followed by an observation of a caregiver-child interaction during which the examiner may collect a language sample or a list of the child's vocalizations and communicative attempts. Following observation, the examiner interacts directly with the child in an attempt to elicit the remaining items

on the scale. The manual instructs the examiner to begin with the Interaction-Attachment item that corresponds to 6 months below the child's chronological age or suspected developmental level. If a behavior is neither directly observed nor elicited, the caregiver is asked to report on the child's mastery of the behavior. For example, item 17 ("Imitates stirring with a spoon") requires a cup and a spoon. To elicit the behavior, the examiner models stirring a spoon in a cup. The examiner then gives the spoon and the cup to the child with a prompt to imitate. If the child does not imitate the action, the caregiver is asked "Does your child imitate stirring a spoon in a cup?"

Items are scored as "passed" if the behavior in question is noted through observation, elicitation, or caregiver report, and each of these is given equal weight in the scoring procedure. Little guidance is provided about how to score observed behaviors which will vary as a function of the precise procedures the examiner chooses to conduct the observation. For the elicited and caregiver report scoring procedures, several guidelines, recommended questions for caregivers, and testing tips are provided for each item.

The goal of the *Rossetti* is to establish the basal and ceiling level of performance in each of the six developmental domains. Global basal and ceiling levels that provide information about the child's overall communicative skills are also available. Because the number of items differs by age, administration time varies depending on the child's chronological or expected developmental level. Testing time is also affected by variation in administration procedures (e.g., whether the caregiver fills out the questionnaire prior to the testing session or whether the examiner conducts the caregiver interview in whole, in part, or not at all).

Historical Background

The *Rossetti* was first published in 1990. Minor revisions of some items and additional information regarding scoring and interpretation were included in a later publication (Rossetti 2005). The most recent version of the measure (Rossetti 2006) is similar to the earlier ones except that it

includes a Spanish version of the caregiver interview and report questions.

The *Rossetti* was originally developed in response to the need for a measure to assess preverbal and verbal skills of children. At that time, there were a variety of measures that existed to assess motor, cognitive, and language domains. However, Rossetti (2005) noted a paucity of measures to assess the communicative skills of children under age 3 years. The focus on early language and interaction skills was justified on the grounds that communicative skills are "the single best indicator of developmental performance" (Rossetti 2005, p. 7) with noteworthy correlations with subsequent academic performance.

One feature that distinguishes the *Rossetti* from many formal measures of communicative skills is that it is a criterion-referenced measure, meaning that scores are interpreted in relation to a functional performance standard. The value of many criterion-referenced tests has been affirmed for diagnosis, assessment, and intervention. In particular, there is a common recommendation that criterion-referenced measures be used when norm-referenced interpretation of test performance are unavailable or inappropriate (McCauley 1996). There are also recent reports of the use of the *Rossetti* (Hwa-Froelich 2009) and *Rossetti* adaptations (Glennen and Masters 2002) for the evaluation of linguistically and culturally diverse children.

Psychometric Data

As Rossetti (2005) described, "only items that were considered discriminating and representative of a skill at an age were included in the scale" (p. 10). Unfortunately, no psychometric data are provided to evaluate the degree to which items *actually* discriminate between performance levels. Guidelines for the evaluation of criterion-referenced measures are offered in the Standards for Educational and Psychological Testing (American Educational Research Association [AERA], American Psychological Association [APA], and National Council of Measurement in Education [NCMI] 1999). Although the *Rossetti* appears to achieve a few of the proposed guidelines (e.g., detailed description of test user

qualification), no evidence of the measure's reliability and validity is offered in the Examiner's Manual. This is a serious shortcoming of the *Rossetti*.

Clinical Uses

The *Rossetti* was not developed to screen for, or identify children with, autism spectrum disorder (ASD). On the other hand, the evaluation of communicative skills is important in the assessment of young children with ASD who often demonstrate qualitative communicative impairments and developmental delays. In this light, the *Rossetti* may have some utility as a clinical tool in ASD as one component in an evaluation or assessment battery. As noted above, it uses a combination of observation, direct child assessment, and caregiver report, which may make it an attractive clinical choice. Because the *Rossetti* involves caregivers as major sources of information in the assessment process, the measure might also be desirable from a family-centered perspective.

Rossetti (2005) suggests that the measure can aid in early detection and intervention for children who may be at risk for developmental delay and poor academic performance. It might be used to describe the severity of a delay (i.e., mild, moderate, severe) by providing information about a child's overall communicative skills. In addition, the *Rossetti* might be used to describe a child's communicative strengths and weaknesses to guide intervention efforts and document the child's development over time. However, evidence of meaningful intervention goals and objectives that could be derived from the test is lacking at present.

As noted above, the *Rossetti* is a criterion-referenced measure and it does not yield norm-referenced scores. For this reason, it has gained some traction for use with culturally and linguistically diverse children (e.g., Hwa-Froelich 2009) who are often not represented in the normative samples of norm-referenced tests. Nevertheless, users are cautioned that no systematic empirical evidence is available to support the reliability and validity of the *Rossetti*. At present, it does not achieve industry standards (see AERA, APA, and NCMI 1999) that could justify a formal

recommendation for clinical use. As such, professionals are encouraged to consider alternative measures of early communicative skills that have been well validated (e.g., the *MacArthur Communicative Development Inventories* [CDIs]; Fenson et al. 2007). For those who choose the *Rossetti* for clinical use, careful consideration of appropriate and inappropriate interpretation of *Rossetti* scores is recommended (see Andersson 2004).

See Also

- ▶ Attachment
- ▶ Communication and Symbolic Behavior Scale
- ▶ Communication Assessment
- ▶ Developmental Delay
- ▶ Direct Observation
- ▶ Early Language Milestone Scale
- ▶ Expressive Language
- ▶ Family-Centered Care, Second Edition
- ▶ Imitation
- ▶ Infant/Toddler Checklist
- ▶ Infant-Toddler Social and Emotional Assessment (ITSEA)
- ▶ Language Tests
- ▶ MacArthur-Bates Communicative Development Inventories, Second Edition
- ▶ Mullen Scales of Early Learning
- ▶ Observational Assessments
- ▶ Play
- ▶ Pragmatic Communication
- ▶ Pragmatics
- ▶ Prelinguistic Communication Assessment
- ▶ Preverbal Communication
- ▶ Reciprocal Communication/Interaction
- ▶ Screening Measures
- ▶ Symbolic Play
- ▶ Symbolic Thought

References and Reading

- American Educational Research Association, American Psychological Association, & National Council of Measurement in Education. (1999). *Standards for educational and psychological testing*. Washington, DC: American Psychological Association.
- Andersson, L. L. (2004). Appropriate and inappropriate interpretation and use of test scores in early intervention. *Journal of Early Intervention*, 27(1), 55–68.

- Fenson, L., Marchman, V. A., Thal, D. J., Dale, P. S., Reznick, J. S., & Bates, E. (2007). *MacArthur-Bates communicative development inventories* (2nd ed.). Baltimore: Paul H. Brookes.
- Glennen, S., & Masters, M. G. (2002). Typical and atypical language development in infants and toddlers adopted from Eastern Europe. *American Journal of Speech-Language Pathology*, 11, 417–433.
- Hwa-Froelich, D. A. (2009). Communication development in infants and toddlers adopted from abroad. *Topics in Language Disorders*, 29(1), 32–49.
- McCauley, R. (1996). Familiar strangers: Criterion-referenced measures in communication disorders. *Language, Speech, and Hearing Services in Schools*, 27, 122–131.
- Rossetti, L. (1990). *The Rossetti infant-toddler language scale: A measure of communication and interaction*. East Moline: LinguiSystems.
- Rossetti, L. (2005). *The Rossetti infant-toddler language scale: A measure of communication and interaction*. East Moline: LinguiSystems.
- Rossetti, L. (2006). *The Rossetti infant-toddler language scale: A measure of communication and interaction*. East Moline: LinguiSystems.

Rossetti Infant-Toddler Language Scale (The Rossetti)

- [Rossetti Infant-Toddler Language Scale](#)

Rote Learning

- [Rote Memory](#)

Rote Memory

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Synonyms

[Rote learning](#)

Definition

Rote memory is associated with recalling factual information or data. Rote memory generally entails memory for material without much reference to the meaning, emotions, or to the context to which it is associated. The major practice in rote memorization is learning by repetition or routine, without full comprehension or attention to what is being memorized. Rote memory is often prompted by external cues. Many individuals with autism spectrum disorders (ASD) have intact, if not strong, rote memory abilities. They can store vast lists of information (dinosaurs, periodic tables, bus timetables, movie scripts, etc.) for long periods of time and can recall or reproduce them accurately. However, individuals with autism typically do not use organization strategies or context to support memory. Rote memory can be viewed as an asset to individuals with ASD as they often do well on spelling bees, geography, and other contests or exams that require a large amount of information on one topic. However, rote memory can also be misleading by giving the impression that someone has higher level comprehension skills, when in fact the individual only comprehends at the factual level. The Wide Range Assessment of Memory and Learning, Second Edition (WRAML 2), is one common psychological assessment that measures memory functions and can be used across the life span.

See Also

- [Hyperlexia](#)
- [Memory](#)
- [Memory Assessment](#)
- [Memory Development](#)
- [Wechsler Memory Scale \(All Versions\)](#)
- [Wide Range Assessment of Memory and Learning \(WRAML\)](#)

References and Reading

- Bennetto, L., Pennington, B. F., & Rogers, S. J. (1996). Intact and impaired memory functions in autism. *Child Development*, 67, 1816–1835.

- Bowler, D. M., Gardiner, J. M., & Grice, S. J. (2000). Episodic memory and remembering in adults with Aspergers syndrome. *Journal of Autism and Developmental Disorders*, 30(4), 295–304.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Williams, D. L., Goldstein, G., & Minshew, N. J. (2006). The profile of memory function in children with autism. *Neuropsychology*, 20, 21–29.
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11, 115–129.

Rourke, Byron

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Name and Degrees

- Rourke, Byron (1939–2011)
- Secondary School – Assumption College School, Windsor, Graduation, June, 1956
- University – Windsor, Windsor Hons. B.A., June, 1962
- Graduate School – Fordham, New York, M.A., February, 1964; Ph.D., February, 1966

Major Appointments (Institution, Location, Dates)

Rank, Institution, Date

- Lecturer (part-time), University of Windsor, Windsor, Summer 1962, 1963
- Lecturer (part-time), Manhattan College, New York, 1963–1964
- Lecturer (part-time), Hunter College, New York, 1963–1964
- Lecturer (part-time), Fordham University, New York, Summer 1964, 1965
- Lecturer, University of Windsor, Windsor, Sept. 1965–Feb. 1966
- Assistant Professor, University of Windsor, Windsor, Feb. 1966–Aug. 1969
- Associate Professor, University of Windsor, Windsor, Sept. 1969–June 1974
- Professor, University of Windsor, Windsor, July 1974–Sept. 2004

- University Professor, University of Windsor, Windsor, June 1986–2004
- Clinical Professor, Yale University School of Medicine, July 1993–June 2009
- Professor Emeritus, University of Windsor, Windsor, Sept. 2004–2011

Major Honors and Awards

Major Predoctoral Awards

- Board of Governors Gold Medal in Psychology, University of Windsor Graduation, 1962
- Woodrow Wilson Fellowship, 1962–1963
- Fordham University Scholarship, 1963–1964
- Canada Council Pre-Doctoral Fellowships, 1963–1964 and 1964–1965
- Sigma Xi, 1964
- Phi Beta Kappa, 1966

Major Postdoctoral Awards

- Research Award for Distinguished Contributions to Clinical Neuropsychology, Ontario (now Canadian) Psychological Foundation, 1985
- University Professor, University of Windsor, 1986
- Award for Distinguished Contributions to Psychology as a Profession, Canadian Psychological Association, 1994
- Fellow of the Royal Society of Canada, 1997
- Who's Who in Canada, 1998
- Donald O. Hebb Award for Distinguished Contributions to Psychology as a Science, Canadian Psychological Association, 1999
- Innis-Gerin Medal for a Distinguished and Sustained Contribution to the Literature of the Social Sciences, Royal Society of Canada, 2001
- University of Windsor Award for Excellence in Scholarship and Research, 2002
- University of Windsor Faculty of Arts and Social Science Career Achievement Award, 2003
- Doctor of Laws, honoris causa, University of Windsor, 2004
- Canadian Psychological Association Gold Medal for Distinguished and Enduring Lifetime Contributions to Canadian Psychology, 2007

- International Neuropsychological Society Distinguished Career Award, 2008
- Member, Order of Canada, 2009

Landmark Clinical, Scientific, and Professional Contributions

Scientific Boards

- Member (1973–1979) and Chair (1977–1979), Fellowships and Sundry Awards Committee, the Ontario Mental Health Foundation
- Member, Research Committee, and Member, Advisory Board, Ontario Mental Health Foundation (1977–1979)
- Member (1988–1994), Scientific Advisory Board, Learning Disabilities Association of Canada

Scientific Societies and Committees

- Member of Governing Board (1976–1983), Secretary (1976–1979), President-Elect (1980), President (1981), Past-President (1982), International Neuropsychological Society
- Member, Executive Committee (1980–1982; 1983–1986), President-Elect (1986–1987), President (1987–1988), Past-President (1988–1989), Division 40, American Psychological Association
- Member (1986–1993; trial termination), Data, Safety, and Quality Review Group of the Diabetes Control and Complications Trial, National Institute of Diabetes and Digestive and Kidney Diseases, United States National Institutes of Health

Professional Boards

- Charter Member (1981), Governing Board Member (1981–1995), Secretary (1987–1993), President (1993–1995), American Board of Clinical Neuropsychology
- Charter Member (1995), President (1995–1996), American Academy of Clinical Neuropsychology

Editorial

- Co-Founder, Co-Editor, *Journal of Clinical Neuropsychology* (1978–1984)/*Journal of Clinical and Experimental Neuropsychology* (1985–1998)
- Co-Founder, Co-Editor, *The Clinical Neuropsychologist* (1985–2002)
- Co-Founder, Co-Editor, *Aging and Cognition* (1992–1995)
- Co-Founder, Co-Editor, *Child Neuropsychology* (1993–1998)
- Member, Review Board, *Journal of Learning Disabilities* (1982–1999)
- Consulting Editor, *Journal of Child Neurology* (1984–1991)
- Member, Editorial Board, *Canadian Psychology* (1986–1990)
- Member, Editorial Advisory Board, *Advances in Child Neuropsychology* (1989–1995)
- Member, Editorial Board, *Clinical Child Psychology* (1997–1999)

Key Books Authored, Coauthored, Edited, or Coedited

- Rourke, B. P., Bakker, D. J., Fisk, J. L., & Strang, J. D. (1983). *Child neuropsychology: An introduction to theory, research, and clinical practice*. New York: Guilford Press.
- Rourke, B. P. (Ed.). (1985). *Neuropsychology of learning disabilities: Essentials of subtype analysis*. New York: Guilford Press.
- Rourke, B. P., Fisk, J. L., & Strang, J. D. (1986). *Neuropsychological assessment of children: A treatment-oriented approach*. New York: Guilford Press.
- Rourke, B. P. (1989). *Nonverbal learning disabilities: The syndrome and the model*. New York: Guilford Press.
- Rourke, B. P. (Ed.). (1991). *Neuropsychological validation of learning disability subtypes*. New York: Guilford Press.
- Rourke, B. P., & Fuerst, D. R. (1991). *Learning disabilities and psychosocial functioning: A neuropsychological perspective*. New York: Guilford Press.

- Adams, K. M., & Rourke, B. P. (Eds.). (1992). *The TCN guide to professional practice in clinical neuropsychology*. Lisse, The Netherlands: Swets & Zeitlinger.
- Rourke, B. P., Costa, L., Cicchetti, D. V., Adams, K. M., & Plasterk, K. J. (Eds.). (1992). *Methodological and biostatistical foundations of clinical neuropsychology*. Lisse, The Netherlands: Swets & Zeitlinger.
- Rourke, B. P., & Del Dotto, J. E. (1994). *Learning disabilities: A neuropsychological perspective*. Thousand Oaks, CA: Sage.
- Rourke, B. P. (Ed.). (1995). *Syndrome of nonverbal learning disabilities: Neurodevelopmental manifestations*. New York: Guilford Press.
- Rourke, B. P., van der Vlugt, H., & Rourke, S. B. (2002). *Practice of child-clinical neuropsychology: An introduction*. Lisse, The Netherlands: Swets & Zeitlinger.
- Cicchetti, D. V., & Rourke, B. P. (Eds.). (2004). *Methodological and biostatistical foundations of clinical neuropsychology and medical and health disciplines* (Vol. 2). Lisse, The Netherlands: Taylor & Francis.

Short Biography

Byron Rourke devoted his professional life to teaching, training, and mentoring; clinical research; and developing the field of pediatric neuropsychology. He was a cofounding editor of the *Journal of Clinical and Experimental Neuropsychology*, *The Clinical Neuropsychologist* as well as *Child Neuropsychology*. He served as president of the International Neuropsychological Society and Division 40 of the American Psychological Association. He established the clinical neuropsychology program at the University of Windsor and fostered the careers of many in the field. He was the author, coauthor, or editor of numerous books and articles on child neuropsychology and is perhaps most well known for his pivotal work on the nonverbal learning disability syndrome and model. Byron received high honors including being the recipient of a membership as a

Fellow in the Royal Society of Canada, a Gold Medal Award from the Canadian Psychological Association's Award for Distinguished Contributions to the Practice of Psychology (1994) and, more recently, investiture into the Order of Canada (2009). He is remembered as one of the preeminent child clinical neuropsychologists in the field, devoted to his students and family. He is survived by his wife Carolyne of 47 years, his sons Phil, Sean, Damon, and Bernie and nine grandchildren.

References and Reading

Books

- Adams, K. M., & Rourke, B. P. (Eds.). (1992). *The TCN guide to professional practice in clinical neuropsychology*. Lisse: Swets & Zeitlinger.
- Cicchetti, D. V., & Rourke, B. P. (Eds.). (2004). *Methodological and biostatistical foundations of clinical neuropsychology and medical and health disciplines* (Vol. 2). Lisse: Taylor & Francis.
- Rourke, B. P. (Ed.). (1985). *Neuropsychology of learning disabilities: Essentials of subtype analysis*. New York: Guilford.
- Rourke, B. P. (1989). *Nonverbal learning disabilities: The syndrome and the model*. New York: Guilford.
- Rourke, B. P. (Ed.). (1991). *Neuropsychological validation of learning disability subtypes*. New York: Guilford.
- Rourke, B. P. (Ed.). (1995). *Syndrome of nonverbal learning disabilities: Neurodevelopmental manifestations*. New York: Guilford.
- Rourke, B. P., & Del Dotto, J. E. (1994). *Learning disabilities: A neuropsychological perspective*. Thousand Oaks: Sage.
- Rourke, B. P., & Fuerst, D. R. (1991). *Learning disabilities and psychosocial functioning: A neuropsychological perspective*. New York: Guilford.
- Rourke, B. P., Bakker, D. J., Fisk, J. L., & Strang, J. D. (1983). *Child neuropsychology: An introduction to theory, research, and clinical practice*. New York: Guilford.
- Rourke, B. P., Fisk, J. L., & Strang, J. D. (1986). *Neuropsychological assessment of children: A treatment-oriented approach*. New York: Guilford.
- Rourke, B. P., Costa, L., Cicchetti, D. V., Adams, K. M., & Plasterk, K. J. (Eds.). (1992). *Methodological and biostatistical foundations of clinical neuropsychology*. Lisse: Swets & Zeitlinger.
- Rourke, B. P., van der Vlugt, H., & Rourke, S. B. (2002a). *Practice of child-clinical neuropsychology: An introduction*. Lisse: Swets & Zeitlinger.

Selected Chapters in Edited Books

- Rourke, B. P. (2002). My odyssey in child-clinical neuropsychology. In A. Y. Stringer, A. L. Cooley, & A. L. Christensen (Eds.), *Pathways to prominence in neuropsychology: Reflections of twentieth century pioneers* (pp. 239–251). New York: Psychology Press.
- Rourke, B. P., Ahmad, S. A., Collins, D. W., Hayman-Abello, B. A., Hayman-Abello, S. E., & Warriner, E. M. (2002b). Child-clinical/pediatric neuropsychology: Some recent advances. *Annual Review of Psychology*, 53, 309–339.
- Rourke, B. P., Hayman-Abello, B. A., & Collins, D. W. (2003). Learning disabilities: A neuropsychological perspective. In R. S. Schiffer, S. M. Rao, & B. S. Fogel (Eds.), *Neuropsychiatry* (2nd ed., pp. 630–659). New York: Lippincott, Williams, & Wilkins.
- Tsatsanis, K. D., & Rourke, B. P. (2003). Syndrome of nonverbal learning disabilities: Effects on learning. In A. H. Fine & R. Kotkin (Eds.), *Therapists guide to learning and attention disorders* (pp. 109–145). New York: Academic.
- Tsatsanis, K. D., & Rourke, B. P. (2008). Syndrome of nonverbal learning disabilities in adults. In L. Wolf, H. Schreiber, & J. Wasserstein (Eds.), *Adult learning disorders: Contemporary Issues* (pp. 159–190). New York: Psychology Press.
- Rourke, B. P. (1995c). The science of practice and the practice of science: The scientist-practitioner model in clinical neuropsychology. *Canadian Psychology*, 36, 259–287.
- Rourke, B. P. (2000). Neuropsychological and psychosocial subtyping: A review of investigations within the University of Windsor laboratory. *Canadian Psychology*, 41, 34–50.
- Rourke, B. P. (2005). Neuropsychology of learning disabilities: Past and future. *Learning Disabilities Quarterly*, 28, 111–114.
- Rourke, B. P. (2008a). Neuropsychology as a (psycho) social science: Implications for research and clinical practice. *Canadian Psychology*, 49, 35–41.
- Rourke, B. P. (2008b). Is neuropsychology a (psycho) social science? *Journal of Clinical and Experimental Neuropsychology*, 30, 691–699.
- Rourke, B. P., Young, G. C., & Leenaars, A. (1989). A childhood learning disability that predisposes those afflicted to adolescent and adult depression and suicide risk. *Journal of Learning Disabilities*, 21, 169–175.
- Rourke, B. P., Del Dotto, J. E., Rourke, S. B., & Casey, J. E. (1990). Nonverbal learning disabilities: The syndrome and a case study. *Journal of School Psychology*, 28, 361–385.

Selected Journal Articles

- Collins, D. W., & Rourke, B. P. (2003). Learning-disabled brains: A review of the literature. *Journal of Clinical and Experimental Neuropsychology*, 25, 1011–1034.
- Drummond, C. R., Ahmad, S. A., & Rourke, B. P. (2005). Rules for the classification of younger children with nonverbal learning disabilities and basic phonological processing disabilities. *Archives of Clinical Neuropsychology*, 20, 171–182.
- For a complete listing: <http://www.nld-bprourke.ca/>
- Fuerst, D. R., & Rourke, B. P. (1993). Psychosocial functioning of children: Relations between personality subtypes and academic achievement. *Journal of Abnormal Child Psychology*, 21, 597–607.
- Klin, A., Volkmar, F. R., Sparrow, S. S., Cicchetti, D. V., & Rourke, B. P. (1995). Validity and neuropsychological characterization of Asperger syndrome: Convergence with nonverbal learning disabilities syndrome. *Journal of Child Psychology and Psychiatry*, 36, 1127–1140.
- Pelletier, P. M., Ahmad, S. A., & Rourke, B. P. (2001). Classification rules for basic phonological processing disabilities and nonverbal learning disabilities: Formulation and external validity. *Child Neuropsychology*, 7, 84–98.
- Rourke, B. P. (1987). Syndrome of nonverbal learning disabilities: The final common pathway of white-matter disease/dysfunction? *The Clinical Neuropsychologist*, 1, 209–234.
- Rourke, B. P. (1995b). Identifying features of the syndrome of nonverbal learning disabilities in children. *Perspectives: The Orton Dyslexia Society*, 21, 10–13.

Routine Events

► Daily Routines

RSH Syndrome

► Smith-Lemli-Opitz Syndrome

Rubella

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Synonyms

3-Day measles; German measles

Definition

Rubella is a virus whose only natural host is humans. It is an RNA Rubivirus in the Togaviridae family. It is spread by droplets in the air and close contact. It infects a person through the respiratory tract and spreads to other sites in the body through the bloodstream. One-quarter to one-half of infected people are asymptomatic. Symptoms may occur 1–3 weeks after exposure. The infected person is contagious from 3 to 8 days before the typical rash appears and remains infectious for another 1–2 weeks. The rash itself starts on the face and spreads to the trunk and extremities lasting approximately 3 days. Symptoms of infection vary by age. The common symptoms experienced by children in addition to the rash are fever, chills, sore throat, headache, eye inflammation, and enlarged lymph nodes at the base of the skull (suboccipital). Adults and older children may also have joint and muscle pain, temporary problems with blood clotting (transient thrombocytopenic purpura), and itching. Rare sequelae include inflammation of the brain. Treatment is supportive. Both serology (blood measures of the antibody that the body makes to infection) and viral culture can be used to diagnose infection. Infection of a pregnant mother is much more serious than postnatal infections. Congenital infection of a fetus may result in stillbirth, fetal death, and sequelae that include deafness, visual impairment, intellectual disabilities, autism, cerebral palsy, and heart disease. Prior to the license of rubella vaccine in 1969, epidemics occurred in 6- to 9-year cycles. Between 1962 and 1965, there were 20,000 cases of congenital rubella. With high immunization rates of children in the USA, congenital rubella has all but disappeared. Rubella vaccine is combined with measles and mumps with administration recommended at 12–15 months and 4–6 years of age. Women and girls of child-bearing age are assessed for rubella susceptibility and advised regarding immunization prior to becoming pregnant. The rubella vaccine is a live attenuated vaccine and does not contain thimerosal as a preservative.

See Also

► [Measles-Mumps-Rubella \(MMR\) Vaccination](#)

References and Reading

- American Academy of Pediatrics. (2009). In L. K. Pickering, C. J. Baker, D. W. Kimberlin, & S. S. Long (Eds.), *Red book: 2009 report of the committee on infectious diseases* (28th ed., pp. 574–579). Elk Grove Village: Author.
- <http://aapredbook.aappublications.org/cgi/content/extract/2009/1/3.116>
- <http://emedicine.medscape.com/article/968523-overview>
- <http://www.cdc.gov/vaccines/vpd-vac/rubella/default.htm>
- <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0002541/>

Rumination

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Synonyms

[Merycism](#); [Rumination disorder](#)

Definition

Rumination refers to the habit of regurgitating food and chewing it again. While occasionally seen in infants who are otherwise developing normally, it is more frequent in children with developmental delays (sometimes with autism). It can also be seen in older individuals without developmental delays. The behavior can lead to various difficulties including dental problems, delays in growth, and food aspiration (inhalation of food into the lungs). Sometimes, gastroesophageal reflux contributes to the problem. Particularly if medical complications are present, interventions may be employed. Behavioral interventions can

include a range of techniques. Treatment of any associated medical problems may be appropriate.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Rumination Disorder](#)

References and Reading

- Birmingham, C. L., & Firoz, T. (2006). Rumination in eating disorders: Literature review. *Eating and Weight Disorders: EWD*, 11(3), e85–e89.
- Rhine, D., & Tarbox, J. (2009). Chewing gum as a treatment for rumination in a child with autism. *Journal of Applied Behavior Analysis*, 42(2), 381–385.
- Volkmar, F., & Wiesner, L. (2009). *A practical guide to autism*. Hoboken: Wiley.

Rumination Disorder

Fred R. Volkmar

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Synonyms

[Merycism](#); [Rumination](#)

Short Description or Definition

Rumination refers to the habit of regurgitating food and chewing it again. While occasionally seen in infants who are otherwise developing normally, it is more frequently seen in children with developmental delays (sometimes with autism). It can also be seen in older individuals without developmental delays. Rarely, it is seen in otherwise typically developing individuals as an isolated phenomenon.

Typically food is rechewed and swallowed, but sometimes is spit out. Rumination may begin

immediately after eating/feeding or for up to an hour or so thereafter. Rumination disorder is diagnosed if the problem is persistent and a focus of treatment.

Ruminative behavior can lead to various difficulties including dental problems, delays in growth, and food aspiration (inhalation of food into the lungs). Sometimes, gastroesophageal reflux contributes to the problem. Particularly, if medical complications are present, interventions may be employed. Behavioral interventions can include a range of techniques. Treatment of any associated medical or dental problems arising from the condition may be appropriate.

Categorization

Rumination disorder has typically been defined on the basis of repeated rumination (regurgitation and rechewing of food) occurring for a period of a month or more after some time of normative functioning. The diagnosis is not made in the presence of some general medical condition that could account for its presence.

Rumination disorder is observed in several different contexts. Sometimes, it is seen in an otherwise typically developing infant or young children. More frequently is observed in association with intellectual deficiency (with or without ASD).

Epidemiology

Since much of the information on rumination is derived from case reports or small case series, available data are limited in important ways. As a practical matter, the condition is probably relatively rare. In infants (otherwise typically developing) the condition has its onset during the first year; for adults with ID (intellectual deficiency) the onset can be variable – even extending into adulthood. It is unclear if there is a gender predominance. Rumination can be associated with a number of other problems in eating/feeding.

Various factors may be involved in pathogenesis. Although typically there is an assumption that underlying GI problems are not present, this

may oversimplify a complex issue. Behavioral psychology has proposed various models that might account for rumination. It does appear that in infants, risk for rumination increased in under-stimulating environments.

Natural History, Prognostic Factors, Outcomes

The course of the conditions is highly variable. Sometimes it resolves quickly. At other times, it is persistent and then associated with various medical problems and complications, e.g., aspiration or nutritional problems may arise, GI complaints are frequent, and dental problems are common.

Clinical Expression and Pathophysiology

As noted, rumination disorder can be observed in rather different contexts – under-stimulated infants, individuals with significant cognitive delay, and sometimes in otherwise typically developing persons. The condition may be self-limited or become more chronic. Physical symptoms/associated findings can include abdominal pain (with or without constipation) as well as nausea and sometimes diarrhea and/or bloating. Dental problems are frequent if the condition persists as the acid regurgitated contributes to tooth decay.

Evaluation and Differential Diagnosis

It is important to be aware of various factors (medical and psychosocial) that might increase risk for rumination. Any associated or potentially contributing conditions should be documented. These might include medical factor such as reflux and psychosocial factors.

Treatments

A range of behavioral approaches have been used in the treatment of rumination. Early work focused on aversive methods but more recently

other approaches have been used including provision of high starch diets, chew toys, and positive behavioral techniques. Although the literature is mostly comprised of single case reports and case series, reasonably high rates of success are noted.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Dental Care](#)
- [Eating Disorders](#)
- [Rumination](#)

References and Reading

- Birmingham, C. L., & Firoz, T. (2006). Rumination in eating disorders: Literature review. *Eating and Weight Disorders: EWD*, 11(3), e85–e89.
- Nicholls, D., & Bryant-Waugh, R. (2009). Eating disorders of infancy and childhood: Definition, symptomatology, epidemiology, and comorbidity. *Child and Adolescent Psychiatric Clinics of North America*, 18(1), 17–30.
- Rhine, D., & Tarbox, J. (2009). Chewing gum as a treatment for rumination in a child with autism. *Journal of Applied Behavior Analysis*, 42(2), 381–385.
- Volkmar, F., & Wiesner, L. (2009). *A practical guide to autism*. Hoboken: Wiley.
- Woolston, J. L., & Hasbani, S. M. (2005). Eating and growth disorders in infants and children. In A. Martin & F. Volkmar (Eds.), *Lewis's child and adolescent psychiatry: A comprehensive textbook* (4th ed., pp. 583–592). Philadelphia: Wolters Kluwer.

Russia and Autism

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Historical Background

The history of autism as a separate condition in Russia began in 1926–1927 when G. Sukhareva published two chapters which are believed to be early descriptions of the Asperger syndrome in boys and girls (Ssucharewa and Wolff 1996;

Simmonds and Sukhareva 2019). Several years later, Simson described a similar condition for preschool children (Simson 1929). These were accurate accounts of autism manifestations that the authors called schizoid psychopathy, and the clinical elaboration of autistic features was seen as formation of schizophrenic personality.

For many years, autism had been considered as a part of the schizophrenic complex of symptoms until the first classification of autism disorders was developed in a mental health research center in 1997. Its publication coincided with the decision of the Russian Ministry of Public Health to adopt the International Classification of Diseases (ICD-10). On the one hand, the national classification and ICD-10 have regularized the diagnosis of autism and other pervasive developmental disorders. On the other hand, patients with psychotic symptoms fell into three diagnostic groups: atypical childhood psychosis (F84.1) and two categories introduced in the Russian adaptation of ICD-10 (1999) – childhood psychosis (F84.02) within the group childhood autism (F84.0) and childhood schizophrenia (F20.8xx3). Especially differentiation between F84.1 and F20.8xx3 remains diagnostically problematic; thus, it is likely that many patients on the spectrum are diagnosed with schizophrenia. Moreover, autism and autism spectrum disorders are rarely diagnosed in adults and remain a child phenomenon in clinical contexts and public understanding. This will change since the Ministry of Health issued a letter that lifted age restrictions for the diagnosis of autism in 2017. A detailed account of the clinical tradition of autism understanding in Russia is given in Bashina (1999), Tiganov and Bashina (2005), and Psychiatry (2011). The situation may also change after official adoption of the ICD-11.

Since 2003, a specialized open-access Russian language journal *Autizm i narusheniya razvitiya* [Autism and Development Disorders] has been published in Moscow. It appears four times a year and covers a wide range of educational and clinical topics addressed to professionals and parents/caregivers. Along with original materials, it occasionally includes translations of articles and educational resources published abroad (<http://psyjournals.ru/en/autism/>).

Recently there have been some noticeable efforts to raise the public awareness of autism and break the understanding of autism as a solely psychiatric problem. Since 2011 the independent not-for-profit organization *Tsentr problem autisma* [“Autism Challenge” Center] has been promoting ABA intervention, organizing trainings for professionals, and starting the first pilot project of inclusive education designed for children with ASD (<http://autismchallenge.ru>). In April 2013, the Center organized the first international conference “Autism: Challenges and Solutions.” The conference has become the central annual event for autism-related topics that unite researchers, professionals, and parents in Moscow. In 2012 the first specialized foundation *Vykhod* [Way Out] (<http://outfund.ru>) was established. It focuses on promoting autism awareness, introducing services in several regions, and supporting evidence-based interventions. By 2019, the foundation organized three scientific conferences. In recent years many medical and educational conferences and congresses included a section devoted to autism research.

In the mid-2010s, the Federal Resource Center for Comprehensive Support to Children with Autism Spectrum Disorders (FRC) comprised the research and educational facility that existed over 20 years in Moscow, as a department of the Moscow State University of Psychology and Education (MSUPE, <http://autism-frc.ru/en>). Its activities are coordinated with the autism-related projects of the Ministry of Education and have been instrumental in research, outreach, and regional development.

The public awareness of autism grew considerably in recent years, especially after the success of the Russian documentary *Anton Tut Ryadom* [Anton’s Right Here] by Lyubov Arkus (2012). Every April, many cities organize events similar to the Autism Speaks *Light It Up Blue* initiative. Since 2017, a festival *Lyudi kak lyudi* [Just Like Everyone] takes place in Moscow on April 2. It was initiated by the regional charitable organization *Kontakt* [Contact] that provides legal and psychological help to families with autistic children (https://contact-autism.ru/about/who_we_are). In 2019 the

festival added numerous regional partners all over Russia (<http://2апреля.рф>).

Legal Issues, Mandates for Service

The Constitution of the Russian Federation grants the right to education for every citizen, and the Education Law of 2012 (<http://kremlin.ru/acts/bank/36698>) states that every individual with disabilities (literally, individual with restricted health abilities) should get high-quality education including early correction aimed to develop social skills. It mentions creating conditions for inclusive education. The development might accelerate after the UN Convention on the Rights of Persons with Disabilities was formally ratified in 2012. The diagnosis of autism or other developmental disorders does not automatically entitle the individuals or their families to benefits; they must apply for the disability status and its renewal.

The regional boards of psychology, medicine, and education (so-called PMPK) give recommendations about the conditions which would best serve the child, but the parents/caregivers are not legally bound to follow them and enroll the child into a school of their choice. However, the recommendations can be useful for individual provisions at schools, such as later the beginning of secondary education or special conditions during exam periods.

Overview of Current Treatments and Centers

The healthcare system sees autism as an illness that must be treated by psychiatrists. Prescription of antipsychotic and other drugs is not uncommon (Simashkova et al. 2019), often in in-clinic settings. A hospital admission can be also a necessary step when confirming the disability status. Several psychiatric hospitals have specialized units for children with ASD. In many regions medical help and rehabilitation are also provided by the Department of Labor and Social Security.

Most schools for children with disabilities have merged with general education schools that should be able to accommodate children with autism and other conditions through individualized educational plans and additional staffing, including paraprofessionals and tutors. The Ministry of Education developed and published Federal Educational Standards for Elementary Schools, four of which regulate education of children with ASD (<https://docs.edu.gov.ru/document/b903f8ab3dee1dc5e0835ee9f10b59a9/>). They differ in the number of years a child should learn in elementary school and the overall learning goals. For children who are not expected to reach the general goals even with additional years of instruction, a special individual developmental program should be developed and implemented. Inefficiency of this system in specific schools and regions can force parents/caregivers to home-school their children with ASD.

Behavioral interventions only randomly exist in official school systems, usually through support of parent organizations and foundations. Several private not-for-profit rehabilitation centers play a significant role in the regions where they exist. They are often the only institutions which provide help for individuals with autism of all ages and give access to complementary treatment approaches. Adults with ASD, who do not receive support from their families, may be institutionalized into neuropsychiatric homes, whose reform is long overdue.

Overview of Research Directions

The first publication about autism from Russia in a Western scientific journal appeared in 1993 (Lebedinskaya and Nikolskaya 1993). Most scientific and clinical books and articles are published in Russian language and because of that are not readily available for international audience. This applies to extensive literature dedicated to the differentiation of psychotic disorders, autism, and schizophrenia in children (for a recent commentary in English, see Simashkova et al. 2019). Several research groups have been publishing in international peer-reviewed

journals; they include research on chromosomal abnormalities (Vorsanova et al. 2010; Yurov et al. 2007; Zelenova et al. 2019), oxidative stress (Porokhovnik et al. 2015), and perception in autism (Stroganova et al. 2007, 2012; Sysoeva et al. 2016). Until recently comparability of experimental data with other countries was problematic because of a different diagnostic tradition. This is changing after Russian adaptations of ADOS-2 and ADI-R, which can be used for validating the clinical diagnosis, have been published. The funding of research remains insufficient: since 2012 fewer than 20 autism-related projects were supported by the Russian Foundation for Basic Research and by the Russian Science Foundation.

Overview of Training

As with other developmental disorders, only physicians and, more precisely, psychiatrists diagnose autism officially. They are trained at medical schools without pediatric specialization, unlike most doctors in other fields. A wide range of specialists provide educational and rehabilitation services. Many university departments of psychology grant degrees in special and clinical psychology. Pedagogical universities train specialists in special education and speech pathologists. No undergraduate courses are specifically dedicated to ASD, and until recently the training of special education teachers and psychologists did not address autism as a stand-alone condition. In 2014, the MSUPE introduced a 2-year master's degree program in Psychology and Education of Individuals with ASD (<http://sp.mgppu.ru/education/ras.html>). Teachers and psychologists can attend professional development courses organized by the FRC and other organizations, such as the Naked Heart Foundation (nakedheart.org), Center for Curative Pedagogics (<http://ccp.org.ru/en>), Early Intervention Institute (<http://eii.ru>), and Nash Solnechny Mir (<http://www.solnechnymir.ru/>), among others. A webinar has become a popular professional development format for professionals who cannot attend in-person trainings. The Saint Petersburg State University developed a 26-hour introductory course Autism Spectrum Disorders for

professionals and general public that is available online (<http://www.coursera.org/learn/rasstrojstva-autisticheskogo-spektra>).

Social Policy and Current Controversies

The awareness of autism grew significantly in the 2010s, and it has translated into greater acceptance. Many cultural organizations are more open and try to attract more visitors with autism with accessible programming or special provisions. For example, major art museums offer gallery and studio programs, as well as special materials for children and adults with ASD and other developmental disorders (e.g., <https://pushkinmuseum.art/museum4all/index.php?lang=en>, https://garagemca.org/en/visit/disabled_visitors/disabilities).

Main challenges to be resolved in the next years include the fire wall between providers of educational and medical services, the discrepancy between the declared inclusivity of education and available resources, as well as lack of support and inadequate possibilities for employment and education of adults with ASD.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Childhood Psychosis](#)
- [Childhood Schizophrenia](#)
- [ICD 10 Research Diagnostic Guidelines](#)
- [Inclusion](#)

References and Reading

- ADI-R. (2014). Autism diagnostic interview – Revised (in Russian, Раттер М., Кутто Э., Лорд К. ADI-R. Интервью для диагностики аутизма: руководство / Пер. на русский язык и адаптация О. Донец, А. Моховикова, Д. Переверзевой, А. Сорокина. Под общ. ред. А. Сорокина. [Б. м.]: Western Psychological Services; Giunti O.S., 2014. 122 с.).
- ADOS-2. (2016). *Autism diagnostic observation schedule* (2nd ed.) (in Russian, Лорд К., Раттер М., ДиЛаворе П., Ризи С., Готэм К., Бишоп С., Лайстер Р., Гатри У. ADOS-2. План диагностического обследования при аутизме. Изд. 2-е: руководство / Пер. на

- русский язык и адаптация А. Сорокина, Е. Давыдовой, К. Салимовой при участии Е. Пшеничной. [Б. м.]: Western Psychological Services; Giunti O.S., 2016. 544 с.).
- Bashina, V. M. (1999). *Autism in childhood*. Moscow (in Russian, Башина В.М. Аутизм в детстве. М 1999).
- International Classification of Diseases (10th Rev). (1994). Nuller, Y. L., & Tsirkkin, S. Y. (Ed: trans), *Classification of mental and behavioral disorders: ICD-10 clinical descriptions and diagnostic guidelines*. Saint-Petersburg: WHO. (in Russian, Международная классификация болезней (10-й пересмотр). Классификация психических и поведенческих расстройств: МКБ-10 Клинич. описания и указания по диагностике. ВОЗ; Пер. на рус. яз. под ред. Ю. Л. Нуллера, С. Ю. Циркина СПб 1994).
- Lebedinskaya, K. S., & Nikolskaya, O. S. (1993). Brief report: Analysis of autism and its treatment in modern Russian defectology. *Journal of Autism and Developmental Disorders*, 23(4), 675–679.
- Porokhovnik, L. N., Passekov, V. P., Gorbachevskaya, N. L., Sorokin, A. B., Veiko, N. N., & Lyapunova, N. A. (2015). Active ribosomal genes, translational homeostasis and oxidative stress in the pathogenesis of schizophrenia and autism. *Psychiatric Genetics*, 25(2), 79–87. <https://doi.org/10.1097/YPG.0000000000000076>.
- Psychiatry: National Handbook. (2011). *Russian society of psychiatrists*. (in Russian: Психиатрия: Национальное руководство. Российское общество психиатров. М 2011).
- Simashkova, N. V., Boksha, I. S., Klyushnik, T. P., et al. (2019). Diagnosis and management of autism spectrum disorders in Russia: Clinical-biological approaches. *Journal of Autism and Developmental Disorders*, 49, 3906–3914. <https://doi.org/10.1007/s10803-019-04071-4>.
- Simmonds, C., & Sukhareva, G. E. (2019). The first account of the syndrome Asperger described? Part 2: The girls. *European Child & Adolescent Psychiatry*. <https://doi.org/10.1007/s00787-019-01371-z>.
- Simson, T. P. (1929). *Neuropathies, psychopathies and psychogenies of infancy*. Moscow-Leningrad (in Russian, Симсон Т.П. Невропатии, психопатии и реактивные состояния младенческого возраста. М—Л 1929).
- Ssucharewa, G. E., & Wolff, S. (1996). The first account of the syndrome Asperger described? Translation of a paper entitled “Die schizoiden Psychopathien im Kindesalter” by Dr. G. E. Sucharewa; scientific assistant, which appeared in 1926 in the Monatsschrift für Psychiatrie und Neurologie 60: 235–261. *European Child and Adolescent Psychiatry*, 5(3), 119–132.
- Stroganova, T. A., Orekhova, E. V., Prokofyev, A. O., Posikera, I. N., Morozov, A. A., Obukhov, Y. V., & Morozov, V. A. (2007). Inverted event-related potentials response to illusory contour in boys with autism. *Neuroreport*, 18(9), 931–935.
- Stroganova, T. A., Orekhova, E. V., Prokofyev, A. O., Tsetlin, M. M., Gratchev, V. V., Morozov, A. A., & Obukhov, Y. V. (2012). High-frequency oscillatory response to illusory contour in typically developing boys and boys with autism spectrum disorders. *Cortex*, 48(6), 701–717.
- Suchareva, G. E. (1926). Schizoid psychopathies of childhood. In *Issues of child psychoneurology* (No. 2). Moscow (in Russian, Сухарева Г.Е. Шизоидные психопатии детского возраста. В сб.: Вопросы детской психоневрологии. Вып 2. М 1926).
- Syssoeva, O. V., Davletshina, M. A., Orekhova, E. V., Galuta, I. A., & Stroganova, T. A. (2016). Reduced oblique effect in children with autism spectrum disorders (ASD). *Frontiers in Neuroscience*, 9, 512. <https://doi.org/10.3389/fnins.2015.00512>.
- Tiganov, A. S., & Bashina, V. M. (2005). Current approaches to understanding of autism in childhood. *Zhurnal Nevrologii i Psikhiiatrii Imeni S.S. Korsakova*, 105(8), 4–13. (in Russian, Современные подходы к пониманию аутизма в детстве. Журнал неврологии и психиатрии им.С.С.Корсакова, 105(8): 4–13), (2005) abstract in English. <http://www.mediasphera.ru/journals/korsakov/102/eng/1078/>.
- Vorsanova, S. G., Voinova, V. Y., Yurov, I. Y., Kurinnaya, O. S., Demidova, I. A., & Yurov, Y. B. (2010). Cytogenetic, molecular-cytogenetic, and clinical-genealogical studies of the mothers of children with autism: A search for familial genetic markers for autistic disorders. *Neuroscience and Behavioral Physiology*, 40(7), 745–756.
- World Health Organization. (1993). *The ICD-10 classification of mental and behavioral disorders*. Geneva: World Health Organization.
- Yurov, Y. B., Vorsanova, S. G., Iourov, I. Y., Demidova, I. A., Beresheva, A. K., Kravetz, V. S., Monakhov, V. V., Kolotii, A. D., Voinova-Ulas, V. Y., & Gorbachevskaya, N. L. (2007). Unexplained autism is frequently associated with low-level mosaic aneuploidy. *Journal of Medical Genetics*, 44(8), 521–525. Epub 2007 May 4.
- Zelenova, M. A., Yurov, Y. B., Vorsanova, S. G., et al. (2019). Laundering CNV data for candidate process prioritization in brain disorders. *Molecular Cytogenetics*, 12, 54. <https://doi.org/10.1186/s13039-019-0468-7>.

Rutter, Michael

Fred R. Volkmar

Child Study Center, Irving B. Harris Professor of Child Psychiatry, Pediatrics and Psychology, Yale Child Study Center, School of Medicine, Yale University, New Haven, CT, USA

Name and Degrees

- Michael Rutter
- MRCS (Eng), LRCP (London), 1955 MB

- ChB (Birmingham: Distinction in Pharmacology and Therapeutics), 1955
- MRCP (London), 1958
- Academic Diploma in Psychological Medicine (London: Distinction), 1961
- MD (Birmingham: Honors), 1963
- FRCPsych, 1971
- FRCP, 1972

Major Appointments (Institution, Location, Dates)

- Professor of Child Psychiatry, Institute of Psychiatry, University of London (1973–1998)
- Honorary Consultant Psychiatrist, Joint Bethlem and Maudsley Hospitals, London (1966–)
- Honorary Director, Medical Research Council Unit in Child Psychiatry (1984–1998)
- Honorary Director, Social, Genetic, and Developmental Psychiatry Research Centre (1994–1998)
- Professor of Developmental Psychopathology, Institute of Psychiatry (1998–)

Major Honors and Awards

- Foreign Associate Member, US National Academy of Education, 1990
- Knight Baronet, 1992
- Commander of the Order of the British Empire (C.B.E.), 1985
- Fellow of the Royal Society, 1987
- Founding Fellow, Academy of Medical Sciences, 1998
- Foreign Associate Member, Institute of Medicine of the National Academy of Sciences, USA, 1988
- Honorary Fellow, British Academy, 2002
- Founding Member, Academia Europaea, 1988
- Fellow, King's College London, 1998
- Helmut Horten Award in 1997
- International Society for Autism Research, Lifetime Achievement Award, 2002
- C. Anderson Aldrich Award, American Academy of Pediatrics, 1981

- Foreign Honorary Member, American Academy of Arts and Sciences, 1989
- Adolf Meyer Award Lecturer, American Psychiatric Association, 1985
- Blanche F. Ittleson Award, American Psychiatric Association, 1991
- Honorary Degrees: University of Leiden, 1985; Catholic University, Louvain, 1990; University of Birmingham, 1990; University of Edinburgh, 1990; University of Chicago, 1991; University of Minnesota, 1993. Doctor of Science: University of Ghent, 1994; University of Jyväskylä, Finland, 1996; University of Warwick, 1999; University of East Anglia, 2000; University of North London, 2000; University of Amsterdam, 2001; Yale University, 2010; University of Oxford, 2005; University of York, 2005; University of Dublin, 2007; London University, 2008; Helsinki University, 2010

Landmark Clinical, Scientific, and Professional Contributions

The preeminent autism researcher in the world, Professor Sir Michael Rutter's contributions have spanned over four decades of seminal research. His earliest work on classification of autism helped establish the validity of the condition apart from childhood schizophrenia. His early work also illustrated the importance of structured educational interventions rather than less structured psychodynamic approaches to treatment. His early approaches to definition and his work on diagnostic nosology helped establish the recognition, and definition, of autism in the 1980 edition of the American Psychiatric Association's DSM-III. His interest in issues of classification and nosology has continued, and he was a major contributor to the revision of the current definition of autistic disorder in DSM-IV and ICD-10. Over the last decades, his interest in autism has continued and his publication record is remarkable. He has been involved in many of the most important research enterprises including, most recently, the search for genes in autism, medical correlates of

autism, and diagnosis. His work on severe deprivation in children has been path-breaking in the field.

In addition to his work in autism, other areas of interest have included resilience in relation to stress, links between development in childhood and adult, schools, psychiatric epidemiology and genetics, schools, and behavioral difficulties. With over 400 publications (books, chapters, and papers), he has had a tremendous influence on the field of child psychiatry and child development. He has taught and collaborated with many individuals who themselves have become leaders in the field.

Short Biography

Born in Lebanon to British parents, Michael Rutter lived in the USA as a child during World War II. He completed his basic training in medicine at the University of Birmingham, England, qualifying in 1955. He assumed an academic position at the University of London's Institute of Psychiatry and Maudsley Hospital in 1996 where he has since remained. He became professor of child psychiatry and head of the department of child and adolescent in 1975 (until 1995). He was knighted in 1992 and was made an honorary member of the British Academy, a fellow of the Royal Society, and founding fellow of the Academia Europaea and the Academy of Medical Sciences. The Michael Rutter Centre for Children and Adolescents at the Maudsley Hospital, London, is named in his honor.

See Also

- [Autistic Disorder](#)
- [Childhood Schizophrenia](#)
- [DSM-III](#)
- [DSM-IV](#)
- [Epidemiology](#)
- [Genetics](#)
- [Reactive Attachment Disorder](#)

References and Reading

- Autism Genome Project Consortium, Szatmari, P., Paterson, A. D., Zwaigenbaum, L., Roberts, W., Brian, J., et al. (2007). Mapping autism risk loci using genetic linkage and chromosomal rearrangements. *Nature Genetics*, 39(3), 319–328.
- Bartak, L., Rutter, M., et al. (1977). A comparative study of infantile autism and specific developmental receptive language disorders III. Discriminant function analysis. *Journal of Autism and Childhood Schizophrenia*, 7(4), 383–396.
- Bolton, P., & Rutter, M. (1990). Genetic influences in autism. *International Review of Psychiatry*, 2(1), 67–80.
- Bolton, P., Rutter, M., et al. (1989). Females with autism and the fragile X. *Journal of Autism and Developmental Disorders*, 19(3), 473–476.
- Bolton, P. F., Murphy, M., et al. (1997). Obstetric complications in autism: Consequences or causes of the condition? *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(2), 272–281.
- Bolton, P. F., Pickles, A., et al. (1998). Autism, affective and other psychiatric disorders: Patterns of familial aggregation. *Psychological Medicine*, 28(2), 385–395.
- Bolton, P. F., Carcani-Rathwell, I., et al. (2011). Epilepsy in autism: Features and correlates. *The British Journal of Psychiatry*, 198(4), 289–294.
- Cantwell, D. P., Baker, L., et al. (1989). Infantile autism and developmental receptive dysphasia: A comparative follow-up into middle childhood. *Journal of Autism and Developmental Disorders*, 19(1), 19–31.
- Folstein, S., & Rutter, M. (1977). Infantile autism: A genetic study of 21 twin pairs. *Journal of Child Psychology and Psychiatry*, 18(4), 297–321.
- Howlin, P., Goode, S., et al. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 45(2), 212–229.
- Kolvin, I. (1999). The contribution of Michael Rutter. *British Journal of Psychiatry*, 174, 471–475.
- le Couteur, A., Rutter, M., et al. (1989). Autism diagnostic interview: A standardized investigator-based instrument. *Journal of Autism and Developmental Disorders*, 19(3), 363–387.
- Lord, C., Pickles, A., et al. (1997). Diagnosing autism: Analyses of data from the autism diagnostic interview. *Journal of Autism and Developmental Disorders*, 27(5), 501–517.
- Parr, J. R., Le Couteur, A., Baird, G., Rutter, M., Pickles, A., Fombonne, E., & Bailey, A. J. (2011). Early developmental regression in autism spectrum disorder: Evidence from an international multiplex sample. International Molecular Genetic Study of Autism Consortium (IMGSAC) Members. *Journal of Autism and Developmental Disorders*, 41(3), 332–340.
- Rutter, M. (1968). Concepts of autism: A review of research. *The Journal of Child Psychology and Psychiatry*, 9(1), 1–25.

- Rutter, M. (1970). Autism: Educational issues. *Special Education*, 59(3), 6–10.
- Rutter, M. (1972). *Maternal deprivation reassessed*. Oxford: Penguin.
- Rutter, M. (1974). The development of infantile autism. *Psychological Medicine*, 4(2), 147–163.
- Rutter, M. (1978). Diagnosis and definition of childhood autism. *Journal of Autism and Childhood Schizophrenia*, 8(2), 139–161.
- Rutter, M. (1983). Cognitive deficits in the pathogenesis of autism. *The Journal of Child Psychology and Psychiatry*, 24(4), 513–531.
- Rutter, M. (1989). Isle of Wight revisited: Twenty-five years of child psychiatric epidemiology. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28(5), 633–653.
- Rutter, M. (1996). Autism research: Prospects and priorities. *Journal of Autism and Developmental Disorders*, 26(2), 257–275.
- Rutter, M. (1998). Autism: The two-way interplay between research and clinical work. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 40(2), 169–188.
- Rutter, M. (2006). Autism: Its recognition, early diagnosis, and service implications. *Journal of Developmental and Behavioral Pediatrics*, 27(2 Suppl), S54–S58.
- Rutter, M., Bailey, A., et al. (1994). Autism and known medical conditions: Myth and substance. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 35(2), 311–322.
- Volkmar, F. R., & Rutter, M. (1995). Childhood disintegrative disorder: Results of the DSM-IV autism field trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, 34(8), 1092–1095.
- Volkmar, F. R., Klin, A., et al. (1994). Field trial for autistic disorder in DSM-IV. *The American Journal of Psychiatry*, 151(9), 1361–1367.

SAD

► Separation Anxiety Disorder

Safety

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Definition

Accidents are a leading cause of death in autism (Gillberg et al. 2010; Pickett et al. 2006; Shavelle et al. 2001). The risk for accidents in autism likely reflects the role of several factors including associated cognitive disabilities, impulsivity, and behavior problems sometimes combined with relatively good motor abilities. Unusual restricted and sensory interests may also contribute to this problem, e.g., a child who likes spinning things may try to manually explore a spinning fan. While individuals with autism can be nervous about

many new situations, at other times, they may seem driven to explore things that are intensely interesting to them.

In some ways the problem is not, of course, confined to children with autism, i.e., injuries are the leading cause of death in children and youth in the USA. For every injury that is fatal, about 200 other injuries result in emergency room visits. Some steps can be taken to insure safety for the individual both in- and outside of the home. This includes basic awareness of safety in the context of the age and level of functioning of the child. Obvious hazards should be attended to. Guns should not be in the home, and careful security should be provided for poisons and dangerous substances (including medications). Similarly, access to flammable materials, knives, and electrical cords should be limited. Within the home, careful attention should be paid to kitchen and bathroom areas and any areas that present obvious hazards (e.g., swimming pools). Given that drowning is a potential concern, children with autism should be taught to swim. Explicit teaching of safety should also occur. Various accommodations for individuals of differing levels of ability and cognitive understanding can be made. This can include use of visual support for teaching as well as special stickers that indicate that something is harmful (see Volkmar and Wiesner 2009). Parents should be aware of plants in and near the

home that may be poisonous. Both at home and school, the Poison Control Center number should be posted near the phone.

Some children present special issues that raise safety concerns. For children who mouth objects, a careful check of sources of lead-based paint should be made. Such children should not be given access to toys with small parts (i.e., that could be a choking hazard). Other children can have problems with bolting or wandering. Wandering can be presented with use of special locks, monitoring devices, and so forth. Bolting can be a problem for less cognitively able children, e.g., jumping out away from a parent on a busy street or jumping out of a car while it is in motion. Appropriate steps to insure safety should be made. Children who wander may be induced to wear a special bracelet that identifies them. Helper dogs have also been used. For children whose impulsivity is a major problem, medications and behavioral interventions can be used. In one study of bolting (Anderson et al. 2012), in over 1200 children, nearly 50% of families reported at least 1 episode of bolting in the child with ASD after age 4 years. In about a quarter of cases, the length of the absence was sufficient to cause concern. Risk for bolting increased with severity of autism.

Both at home and at school, outside areas can present special hazards. At school, the presence of other children may complicate monitoring of a child with a social disability. Accidents are probably more frequent in areas where children have less supervision and where social demands are greater, e.g., recess, gym, and cafeteria. Explicit teaching of safety concepts (including dealing with fire drills) should be part of the student's program in school. School staff should be prepared to prevent accidents and also have sufficient training in simple first aid that they can immediately respond. In some areas, e.g., where there is access to a pool or potentially dangerous materials, children should not be left without appropriate supervision. As with other aspects of teaching, a structured approach with explicit rules and routines can help teach safety concepts. This task becomes easier as children acquire language, but even for less verbal children, use of picture

schedules and visual cues can help to teach safety issues. As children get older, explicit teaching about common situations, such as crossing the street or use of simple appliances, can be included. Some computer aids are now available, and it is likely that more will be available in the future. The literature on behavioral interventions, e.g., for bolting, has also increased (Roane and DeRosa 2014; Stevenson et al. 2016).

References and Reading

- Anderson, C., Law, J., Daniels, A., Rice, C., Mandell, D. S., Hagopian, L., & Law, P. A. (2012). Occurrence and family impact of elopement in children with autism spectrum disorders. *Pediatrics*, 130(5), 870–877.
- Fancher, V. K. (1991). *Safe kids: A complete child-safety handbook and resource guide for parents*. New York: Wiley.
- Gillberg, C., Billstedt, E., Sundh, V., & Gillberg, I. C. (2010). Mortality in autism: A prospective longitudinal community-based study. *Journal of Autism and Developmental Disorders*, 40(3), 352–357.
- Marotz, L. R., Cross, M. Z., & Rush, J. M. (2005). *Health, safety, and nutrition for the young child* (6th ed.). Clifton Park: Thompson Delmar.
- Pickett, J. A., Paculdo, D. R., Shavelle, R. M., & Strauss, D. J. (2006). 1998–2002 update on “causes of death in autism” [comment]. *Journal of Autism and Developmental Disorders*, 36(2), 287–288.
- Reich, J. B. (2007). *Babyproofing bible: The exceedingly thorough guide to keeping your child safe from crib to kitchen to car to yard*. Beverly: Fair Winds Press.
- Roane, H. S., & DeRosa, N. M. (2014). Reduction of emergent dropping behavior during treatment of elopement. *Journal of Applied Behavior Analysis*, 47(3), 633–638.
- Rodgers, G. C., Jr., & Matyunas, N. J. (1994). *Handbook of common poisonings in children* (3rd ed.). Elk Grove Village: American Academy of Pediatrics.
- Shavelle, R. M., Strauss, D. J., & Pickett, J. (2001). Causes of death in autism. *Journal of Autism and Developmental Disorders*, 31(6), 569–576.
- Shaw, E. (2001). *Keep kids safe: A parent's guide to child safety*. Appleton: Quality Life Resources.
- Stevenson, M. T., Ghezzi, P. M., & Valenton, K. G. (2016). Fct and delay fading for elopement with a child with autism. *Behavior Analysis in Practice*, 9, 169–173.
- Strickland, D. C., McAllister, D., Coles, C. D., & Osborne, S. (2007). An evolution of virtual reality training designs for children with autism and fetal alcohol spectrum disorders. *Topics in Language Disorders*, 27(3), 226–241.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism*. Hoboken: Wiley.

Sainsbury, Clare

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Short Biography

Born in 1974, Clare Sainsbury – a British woman with Asperger’s syndrome – studied at Oxford University in the UK. For many years, she ran an online support group for university students on the autistic spectrum. Her highly acclaimed book – *Martian in the Playground: Understanding the Schoolchild with Asperger’s Syndrome*, published in 2000 – won the TES/NASEN award for best academic book. She has spent several years working part time with children and teenagers with severe autism.

References and Reading

Sainsbury, C. (2000). *An Anthropologist in the playground: Understanding the schoolchild with Asperger’s syndrome*. Bristol: Lucky Duck Publishing.

Sameness, Insistence on

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Definition

Insistence on sameness (IS) is a component/variant of restricted repetitive behavior, a core feature of autistic disorder. IS behaviors are characterized by extreme behavioral rigidity, where things must be “just so” and change is not tolerated. Perhaps some of the most richly detailed and clinically astute descriptions of the need for sameness found in autistic disorder can be found in Leo Kanner’s seminal paper

“Autistic Disturbances of Affective Contact” (Kanner 1943):

There is a marked limitation in the variety of his spontaneous activities. The child’s behavior is governed by an anxiously obsessive desire for the maintenance of sameness that nobody but the child may disrupt on rare occasions. Changes of routine, of furniture arrangement, of a pattern, of the order in which every day acts are carried out, can drive him to despair.

As noted above, the need for sameness in autistic disorder can manifest itself in various ways. These can include extremely rigid eating habits, insisting on taking a particular route to school or work, requiring objects to be placed a certain way, or elaborate rituals around activities of daily living. These behaviors can be excessive and may cause significant impairment to individuals with autism. Preventing or stopping such behavior is often quite difficult, as affected individuals may become anxious, agitated, or aggressive if they are interrupted. As noted by Wahlberg and Jordan (2001), people with autism seem almost incapable of adjusting their behavior to manage the many interruptions and unexpected events that can happen on a daily basis. Given the significant problems these behaviors can pose (for both the individuals with autism as well as their families), relatively little is known about their phenomenology and treatment.

Historical Background

Insistence on sameness behaviors have traditionally been subsumed under the restricted repetitive behavior (RRB) core domain of autism. In the Diagnostic and Statistical Manual of Mental Disorders – 4th Edition (DSM-IV-TR; APA 2000), criteria for RRB can be met by a person exhibiting at least one of the following: “(a) encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus; (b) apparently inflexible adherence to specific, nonfunctional routines or rituals; (c) stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting or complex whole-body movements); or

(d) persistent preoccupation with parts of objects.” What becomes clear upon examination of these criteria is that they are very broad, ranging from repetitive movements of the body to more cognitively mediated symptoms such as intense interests or the need for sameness. There has been growing interest in studying the structure of RRB in autism, as identifying distinct types of behavior may help guide treatment and aid researchers in subsetting groups, as one of the major obstacles in the field has been the tremendous phenotypic variability found in the disorder.

To date, at least six studies (Bishop et al. 2006; Cuccaro et al. 2003; Lam et al. 2008; Mooney et al. 2009; Shao et al. 2003; Szatmari et al. 2006) have examined the structure of repetitive behavior in autism empirically using factor analysis of the RRB domain of the Autism Diagnostic Interview-Revised, a standardized assessment used to inform diagnosis (ADI-R, Rutter et al. 2003). An additional study examined the factor structure of RRB in more detail using the Repetitive Behavior Scale-Revised (Lam and Aman 2007). All of these studies independently verified the presence of an insistence on sameness factor in individuals with autistic disorder. This provides evidence that insistence on sameness is a distinct subtype of repetitive behavior in autism, which may aid treatment research and work examining the neurobiological and genetic underpinnings of the disorder.

Current Knowledge

Explaining Insistence on Sameness in Autistic Disorder

It is not yet understood exactly *why* individuals with autistic disorder crave sameness and loathe change. However, several theories have been offered to try to explain these sameness behaviors in autism; this has been covered comprehensively by Turner (1999). A brief summary of each of these hypotheses is below.

1. Homeostasis. This theory postulates that IS and other repetitive behaviors in autism are the result of too much stimulation from the environment, which the individual finds

aversive (Hutt and Hutt 1970). If novel situations are too arousing, then insisting on sameness in activities and the environment could help maintain homeostasis.

2. Weak central coherence. This hypothesis posits that people with autism are characterized by preferential processing of local rather than global processing of the environment (Frith and Happe 1994). This can lead to a focus on seemingly insignificant details and an inability to understand the wider context of a situation.
3. Executive dysfunction. Problems in executive functioning can lead to difficulties generating, planning, and controlling behavior. These deficits are consistent with the presence of sameness behaviors in autism; the inability to initiate novel behavior thus leads to a persistence on a restricted set of behaviors (e.g., Hughes et al. 1993).
4. Sameness behaviors as a form of compulsive behavior. Researchers have questioned whether sameness behaviors in autism are related to compulsive behaviors found in obsessive-compulsive disorder (OCD, DSM-IV; APA, 2000). Compulsions are defined as repetitive behaviors that serve to reduce anxiety or distress, rather than generate pleasure (American Psychiatric 2000). Therefore, insisting on sameness may reduce anxiety elicited by changes in the environment. This theory has also guided some medication treatment approaches in autism (described below).
5. Sensory reinforcement. Here, it is postulated that there is something intrinsically reinforcing about sameness behaviors: that the behavior is self-stimulatory (Lovaas et al. 1987).

It is important to note that it is unlikely that there is any single explanation that can account for insistence on sameness behaviors in autism. These theories are not mutually exclusive, and it is possible that individual instances may have different substrates.

Measurement of Insistence on Sameness

The Sameness Questionnaire (Prior and MacMillan 1973). This 28-item informant-based measure was developed to compare children with

autism to children with childhood schizophrenia. This questionnaire includes several items that are designed to assess the child's resistance to change (e.g., Does he insist on furniture remaining in the same place? Does he object to visiting new places?) but also includes items that assess motor stereotypies and compulsions.

The Behavior Flexibility Rating Scale (BFRS; Green et al. 2006). Intended for use in individuals with developmental disabilities, this questionnaire has 16 items that serve to identify specific situations related to a resistance to change and to rate the extent to which individuals show problems with environmental changes. Three dimensions of flexibility are assessed: flexibility toward objects, flexibility toward the environment, and flexibility toward persons.

Repetitive Behavior Scale-Revised (RBS-R; Bodfish et al. 1999; Lam and Aman 2007). The RBS-R is an informant-completed questionnaire intended to assess the variety of RRBs observed in individuals with autism spectrum disorders. The RBS-R has 5 empirically derived subscales, one of which is called "Ritualistic/Sameness Behaviors" that has 12 items tapping insistence on sameness behaviors.

The Yale-Brown Obsessive-Compulsive Scale (Y-BOCS; Goodman et al. 1989) and *the Children's Yale-Brown Obsessive-Compulsive-Scale (CY-BOCS;* Scahill et al. 2006). Both the Y-BOCS and CY-BOCS were originally intended to assess obsessive-compulsive disorder in typically developing individuals. Due to the communication limitations in autism, usually the Compulsion section of the Y-BOCS/CY-BOCS is used. This clinician-administered rating tool does probe for some elements of sameness behavior; however, it also queries other types of RRB and provides an overall severity score for all repetitive behaviors combined.

The Childhood Routines Inventory (CRI; Evans et al. 1997). The CRI is a 23-item parental report questionnaire that assesses repetitive and perfectionistic behaviors in the context of normative development. Questions cover many aspects of insistence on sameness, such as "prefers the same household activities every day," "repeats actions over and over," and "prefers to have things done in a particular order or way."

Insistence on Sameness: Specific to Autism?

Although the need for sameness is characteristic of autistic disorder, such behavioral rigidity is also found in other populations. Repetitive, ritualistic, and "just so" behaviors are highly common among typically developing children, around the ages of 2–3 years (Gesell et al. 1974). For example, children may line up objects, have elaborate bedtime routines, and have very rigid rules around food presentation. If these demands are not met, tantrums can result; indeed, the characterization of "the terrible twos" may reflect an aspect of this normative development (Evans et al. 1997). As many as 85% of children aged 2–3 exhibit some of these "just so" behaviors, and by the age of 6 years, these behaviors begin to wane, giving way to more flexible behaviors that are necessary to negotiate the next phases of development (Evans et al. 1997).

The need for sameness is also found in other clinical populations, such as obsessive-compulsive disorder (DSM-IV, APA 1994), Prader-Willi syndrome (Wigren and Hansen 2005), and Down syndrome (Evans and Gray 2000). Research in this area is in its infancy, however. More work needs to be done to examine the specificity of sameness behaviors in autism and how (or if) they differ from other disorders in frequency, severity, and typography. Such knowledge could guide research to the neurobiological underpinnings of the disorder (Militeri et al. 2002).

Treatment

There is some debate in the literature as to whether a person with autism's desire for sameness should be accommodated (by reducing change) or be a target for treatment (by teaching the individual to cope with change) (Mesibov et al. 2005). However, for at least some individuals, the need for sameness is so extreme that it can intensify social difficulties, lead to aggression and/or anxiety, and severely limit the day-to-day activities of the entire family. In these cases, IS may be a reasonable target for intervention.

1. *Behavioral treatments.* To date, there are no well-established behavioral treatments that have focused on the reduction of IS in autism.

However, there are several established approaches in psychological literature that could be applied to reducing IS behaviors in autism, including replacement-based interventions, graduated stimulus change, and the use of visual schedules. For a review, please see Green et al. (2007). Clearly, further research is needed to fully develop and empirically validate these treatment approaches.

2. *Psychopharmacological treatments.* Because of the potential link between the compulsive behaviors found in OCD and the repetitive behaviors found in autism, selective serotonin reuptake inhibitors (SSRIs) have been of interest to researchers and practitioners alike. There have been at least seven randomized controlled trials of SSRIs in autism to date (for a review, see Williams et al. 2010). The largest, well-controlled study of citalopram in children with autism ($n = 149$) showed no reduction of repetitive behaviors in general with active treatment (King et al. 2009). At this point, there is not a great deal of empirical support for the use of SSRIs to treat repetitive behavior in autism. However, in these studies, repetitive behaviors were measured more generally; it is unknown whether extreme IS behaviors in particular would be more responsive to treatment with an SSRI. More research in this area may be warranted.

Future Directions

As stated previously, one of the largest obstacles in the study of autistic disorder is the extreme variability and heterogeneity in the phenotype. One promising line of research is to use insistence on sameness to subset groups. Although insistence on sameness is characteristic of autistic disorder, not *all* people with autism display this class of behavior. Identifying individuals with high levels of IS may be a way to identify groups of people with autistic disorder that are more behaviorally homogeneous, which would be of tremendous use to workers in the field who are trying to delineate the neurobiological and genetic causes of autism.

Although research in this area is in its infancy, there is growing evidence that insistence on sameness in autism may have, at least in part, a genetic

basis. Several groups have looked at sibling pairs with autism to see how insistence on sameness aggregates in families (Cuccaro et al. 2003; Lam et al. 2008; Shao et al. 2003; Szatmari et al. 2006). Three of these studies (Shao et al. 2003; Szatmari et al. 2006; Lam et al. 2008) provided evidence that insistence on sameness behaviors are familial (whereas another forms of repetitive behaviors, Repetitive Motor Behaviors, are not). Further evidence for a genetic basis for IS behaviors is provided by recent study of the relationship between sameness behaviors in individuals with autism and obsessive-compulsive symptoms in their parents (Abramson et al. 2005). Briefly, they found that insistence on sameness was positively correlated with obsessive-compulsive behaviors in parents. Further, when individuals with autism were grouped on the basis of parental Y-BOCS scores (clinically significant versus nonclinically significant), individuals whose parents had clinically significant Y-BOCS scores had higher ADI-R insistence on sameness factor scores.

It is recognized that although autism is a strongly genetic disorder, its phenotypic presentation is so complex that the identification of candidate genes or other etiological mechanisms is unbelievably complex. These studies provide preliminary data that subsetting groups of individuals with autism by severity of insistence on sameness may be a useful way to reveal phenotypically and potentially etiologically distinct subgroups of individuals with autism. More research in this area is needed.

References and Reading

- Abramson, R., Ravan, S., Wright, H., Wieduwilt, K., Wolpert, C., Donnelly, S., et al. (2005). The relationship between restrictive and repetitive behaviors in individuals with autism and obsessive compulsive symptoms in parents. *Child Psychiatry and Human Development*, 36, 155–164.
- Association American Psychiatric. (2000). *Diagnostic and statistical manual* (4th ed.). Washington, DC: APA Press. Text Rev.
- Bishop, S. L., Richler, J., & Lord, C. (2006). Association between restricted and repetitive behaviors and nonverbal IQ in children with autism spectrum disorders. *Child Neuropsychology*, 12(4), 247–267.
- Bodfish, J. W., Symons, F. W., & Lewis, M. H. (1999). *The repetitive behavior scale*. Morganton: Western Carolina Center Research Reports.

- Cuccaro, M. L., Shao, Y., Grubber, J., Slifer, M., Wolpert, C. M., Donnelly, S. L., et al. (2003). Factor analysis of restricted and repetitive behaviors in autism using the autism diagnostic interview-R. *Child Psychiatry and Human Development*, 34(1), 3–17.
- Evans, D. W., & Gray, F. L. (2000). Compulsive-like behavior in individuals with Down syndrome: Its relation to mental age level, adaptive and maladaptive behavior. *Child Development*, 71, 288–300.
- Evans, D. W., Leckman, J., Reznick, J., Carter, A., Henshaw, D., King, R., et al. (1997). Ritual, habit and perfectionism: The prevalence and development of compulsive-like behavior in normal young children. *Child Development*, 68, 58–68.
- Frith, U., & Happe, F. (1994). Autism: Beyond theory of mind. *Cognition*, 50, 115–132.
- Gesell, A., Ames, L. B., & Ilg, F. L. (1974). *Infant and the child in the culture today*. New York: Harper & Row.
- Goodman, W. K., Price, L. H., Rasmussen, S. A., et al. (1989). The Yale-Brown obsessive-compulsive scale. I. Development, use, and reliability. *Archives of General Psychiatry*, 46, 1006–1011.
- Green, V., Sigafoos, J., Pituch, K., Itchon, J., O'Reilly, M., & Lancioni, G. (2006). Assessing behavioral flexibility in individuals with developmental disabilities. *Focus on Autism and Other Developmental Disabilities*, 21, 230–236.
- Green, V., Sigafoos, J., O'Reilly, M., Pituch, K., Didden, R., Lancioni, G., et al. (2007). Behavioral flexibility in individuals with autism: Theory, assessment and intervention. In L. B. Zhao (Ed.), *Autism research advances* (pp. 63–77). New York: Nova.
- Hughes, C., Russell, J., & Robbins, T. W. (1993). Evidence for executive dysfunction in autism. *Neuropsychologica*, 32, 477–492.
- Hutt, C., & Hutt, S. J. (1970). Stereotypies and their relation to arousal: A study of autistic children. In C. Hutt & S. J. Hutt (Eds.), *Behaviour studies in psychiatry* (pp. 175–204). Oxford: Pergamon Press.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- King, B., Hollander, E., Sikich, L., McCracken, J., Scahill, L., Bregman, J., et al. (2009). Lack of efficacy of citalopram in children with autism spectrum disorders and high levels of repetitive behavior: Citalopram ineffective in children with autism. *Archives of General Psychiatry*, 66(6), 583–590.
- Lam, K. S. L., & Aman, M. G. (2007). The repetitive behavior scale-revised: Independent validation in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 855–866.
- Lam, K. S. L., Bodfish, J., & Piven, J. (2008). Evidence for three subtypes of repetitive behavior in autism that differ in familiarity and association with other symptoms. *Journal of Child Psychology and Psychiatry*, 49, 1193–1200.
- Lovaas, O. I., Newsom, C., & Hickman, C. (1987). Self-stimulatory behavior and perceptual reinforcement. *Journal of Applied Behavior Analysis*, 20, 45–68.
- Mesibov, G. B., Shea, V., & Schopler, E. (2004). *The TEACCH approach to autism spectrum disorders*. New York: Springer.
- Mesibov, G. B., Shea, V., & Schopler, E. (2005). *The TEACCH Approach to autism spectrum disorders*. New York: Kluwer Academic/Plenum Publishers.
- Militeri, R., Bravaccio, C., Falco, C., Fico, C., & Palermo, M. T. (2002). Repetitive behaviors in autistic disorder. *European Child & Adolescent Psychiatry*, 11, 210–218.
- Mooney, E., Gray, K., Tonge, B., Sweeney, D., & Taffe, J. (2009). Factor analytic study of repetitive behaviours in young children with pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 39, 765–774.
- Prior, M., & MacMillan, M. B. (1973). Maintenance of sameness in children with Kanner's syndrome. *Journal of Autism and Childhood Schizophrenia*, 3, 154–167.
- Rutter, M., Le Couteur, A., & Lord, C. (2003). *ADI-R: The autism diagnostic interview-revised*. Los Angeles: Western Psychological Services.
- Scahill, L., McDougle, C. J., Williams, S., Dimitripoulos, A., Aman, M. G., McCracken, J., et al. (2006). The children's Yale-Brown obsessive-compulsive scale modified for pervasive developmental disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45(9), 1114–1123.
- Shao, Y., Cuccaro, M. L., Hauser, E. R., Raiford, K. L., Menold, M. M., Wolpert, C. M., et al. (2003). Fine mapping of autistic disorder to chromosome 15q11-q13 by use of phenotypic subtypes. *American Journal of Human Genetics*, 72(3), 539–548.
- Szatmari, P., Georgiades, S., Bryson, S., Zwaigenbaum, L., Roberts, W., Mahoney, W., et al. (2006). Investigating the structure of the restricted, repetitive behaviours and interests domain of autism. *Journal of Child Psychology and Psychiatry*, 47(6), 582–590.
- Turner, M. (1999). Annotation: Repetitive behaviour in autism: A review of psychological research. *Journal of Child Psychology and Psychiatry*, 40, 839–849.
- Wahlberg, T., & Jordan, S. (2001). A case study in the dynamics of autism. In T. Wahlberg, F. Obiakor, S. Burkhardt, & A. F. Rotatori (Eds.), *Autistic spectrum disorders* (pp. 53–65). New York: JAI Press.
- Wigren, M., & Hansen, S. (2005). ADHD symptoms and insistence on sameness in Prader-Willi syndrome. *Journal of Intellectual Disability Research*, 49, 449–456.
- Williams, K., Wheeler, D., Silove, N., & Hazell, P. (2010). Selective serotonin reuptake inhibitors (SSRIs) for autism spectrum disorders (ASD). *Cochrane Database of Systematic Reviews*, 8, CD004677.

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Savant Skills (in Autism)

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Definition

Savant syndrome is a rare but remarkable condition in which persons with developmental disabilities, including autism, have some spectacular, exceptional, and unexpected abilities (“islands of genius”) that stand in marked, jarring juxtaposition to overall limitations. These special abilities are generally music, art, calendar calculating, lightning calculating, or mechanical visual-spatial skills. Whatever the special ability, it is always coupled with massive memory which is exceedingly deep, but limited within very narrow confines.

Savant syndrome occurs in approximately one in ten persons with autistic disorder, ranging in ability from “splinter skills” to “talented” to “prodigious.” That latter term is a high threshold category in which, in absence of a disability, the person would be classified as a prodigy or genius; there are probably fewer than 100 known, living prodigious savants in that extremely high-level performance category. Savant syndrome can be congenital (present from birth) or acquired (a neurotypical person in whom unexpected savant abilities emerge following head injury, stroke, dementia, or other CNS disease).

Savant syndrome is not a disease or disorder by itself. The special abilities seen in the savant are always grafted on to some underlying disability, including autistic disorder.

Theories to Explain Savant Syndrome

There is no single theory which can explain all cases of savant syndrome. Neuropsychologists have concentrated on “weak central coherence,” “mind blindness,” “executive dysfunction,” and “hypersystemizing” as core defects in both autism and savant syndrome. These ideas are discussed at length in the 2010 book *Autism and*

Talent by Happe and Frith which addresses the question of why savant syndrome is overrepresented in autistic disorders compared to other developmental disabilities. Rimland (1964) and Treffert (2010) postulate that in the savant, there is a process of “recruitment,” “rewiring,” and “release” of available and still intact brain capacity as a compensatory mechanism for damage to other brain areas (most often left hemisphere) and damage to the higher level semantic memory circuits which produces a reliance on lower level, habit or procedural memory circuits. This produces the typical clinical picture of right brain skills coupled with “automatic” memory seen most often in the savant. Casanova and Trippe (2010) propose that differences in connectivity, favoring short-range connections compared to long-range connections in minicolumns of the brain, are responsible both for autism itself as well as the novel abilities of savants. Powell (2006) and others have been exploring how “quantum mechanics” and the mechanism of “entanglement” might account for savant abilities including even the extrasensory perception or “psi” capacities in some savants. This theory suggests that suppression of the “dominance” of one area of the brain permits both the release of non-dominant skills and the “without reckoning” memory of savants. The brain area release and substitution phenomenon has been called “paradoxical functional facilitation” and is applicable in both congenital and acquired savants (Kapur 1996).

Historical Background

The first account of savant syndrome is a scientific paper that appeared in the German psychology journal *Gnothi Sauton* in 1873, describing lightning calculator with an extraordinary memory (Moritz 1783). Rush (1789) reported the case of Thomas Fuller, a lightning calculator “who could comprehend scarcely anything, either theoretical or practical, more complex than counting.” But the word “savant” did not appear until 1887 when Down described eleven patients with “special faculties” among the hundreds of patients with severe

cognitive disabilities he had seen during his career at Earlswood Asylum in London. These skills included art, music, mathematics, precise time-keeping, and memory. One boy had memorized “The Rise and Fall of the Roman Empire” and could recite it backward or forward. Down named this condition “idiot savant” since at that time “idiot” was an accepted scientific term for IQ below 25. He combined that with the term “savant” derived from the French word *savoir* meaning “learned person.” It is not clear how many of Down’s patients were autistic, as distinct from mental retardation, but in that same article Down described what he called “developmental retardation” as separate from “congenital” and “accidental” retardation. In so doing, it is clear his “developmental retardation” category would include what is now classified as early-onset and late-onset autistic disorder (Treffert 2006).

In his 1914 *Mental Deficiency (Amentia)* textbook, Tredgold devoted an entire chapter to the “idiot savant” with a succinct, colorful, and capturing description of the idiot savant. That term endured until 1988 when Treffert suggested “savant syndrome” be substituted as the name for this remarkable condition because of the negative connotation of “idiot” and because not all persons with savant syndrome have low IQ. Then in 1988, the popular, award-winning movie *Rain Man* made “autistic savant” household words. That was an extremely useful public education tool but with the caveat that not all persons with autism are savants and not all savants are autistic.

Current Knowledge

The Condition Is Rare but One in Ten Autistic Persons Show Some Savant Skills

In Rimland’s (1978) survey of 5,600 children with autism, 531 (9.8%) were reported by parents to have special abilities, and a 10% incidence of savant syndrome in autistic disorder has become the generally accepted figure in autistic disorder. However, Hermelin (2001) estimated the incidence to be as low as “one or two in 200.” Bolte and Poustka (2004) identified savant skills in 13% of 243 individuals with autism based on at

least one special skill as assessed on the Autism Diagnostic Interview-Revised (ADI-R). Howlin et al. (2010) reported that in a sample of 137 persons with autism involved in long-term follow-up, 28.5% of individuals showed some savant skills based on either cognitive testing or parental reports. That number is probably higher than in other studies because the presence of savant skills, in some cases, was based solely on cognitive scores, particularly block design, on formal IQ testing.

The generally accepted figure for prevalence of savant skills in autistic disorder remains at about one in ten (10%). That figure may be adjusted upward somewhat as larger series of such cases are assembled.

But savant syndrome is not limited just to autistic disorder. In a survey of an institutionalized population with a diagnosis of mental retardation, Hill (1977) reported the incidence of savant syndrome was 1:2,000 (.06%). A more recent survey of 583 facilities found a prevalence rate of 1.4 per 1,000 (Saloviita et al. 2000). Whatever the exact figures, mental retardation and other forms of developmental disability are more common than autistic disorder, so a reasonable estimate is that approximately 50% of persons with savant syndrome have autism as the underlying disorder and the other 50% have other forms of developmental disability, mental retardation, or other CNS injury or disease as the underlying disorder. Thus, not all autistic persons have savant syndrome, and not all persons with savant syndrome have autistic disorder.

Interestingly, in Kanner’s (1971) follow-up of his original 11 cases, 6 had outstanding savant skills in music or memory. In Miller’s (1998) report on 45 individuals with savant abilities, 22 had autism or symptoms highly suggestive of that disorder.

Savant Skills Typically Occur in Particular Categories of Abilities

Considering all the skills in the human repertoire, it is interesting that savant skills narrow to five general categories: *music, art, calendar calculating, lightning (rapid) calculation, and mechanical and/or spatial skills* such as measuring distances

with precise accuracy, the ability to construct complex models or structures, map-making, or direction finding. These special skills tend to be those associated with right hemisphere function. Other skills are also reported from time to time such as polyglot (language acquisition); unusual sensory discrimination in smell, touch, or vision including synesthesia; perfect appreciation of passing time without benefit of a clock; and outstanding knowledge in specific fields such as neuropsychology, statistics, or navigation.

Generally, a single skill exists, but in some instances several skills exist simultaneously. Whatever the special skill, it is always accompanied by prodigious memory. Some observers rank memory as a separate special skill. However, prodigious memory within the area of the special skill cuts across all the special ability areas as a shared, integral part of the syndrome itself.

There Is a Spectrum of Savant Skills

The most common form of savant skills are “*splinter skills*,” which include obsessive preoccupation with, and memorization of, music and sports trivia, license plate numbers, maps, historical facts, or obscure items such as vacuum cleaner sounds, for example. *Talented savants* are those whose special skills are more highly honed and are very conspicuous when viewed in contrast to overall limitations. *Prodigious savants* are those persons whose skills are at such a spectacular level that if no disability was present, they would be classified as prodigy or genius (Treffert 1988).

The Special Skills Are Always Accompanied by Massive Memory

Whatever the special abilities, a remarkable memory of a unique and uniform type yields the condition together. Terms such as automatic, mechanical, concrete, and habit-like have been applied to this extraordinary memory. Down (1887) used the term “verbal adhesion”; Critchley (1979) used the term “memory without reckoning,” and Tredgold (1914) used the term “automatic.” Such unconscious memory suggests what Mishkin et al. (1984) referred to as nonconscious “habit” formation rather than a “semantic”

memory system. They proposed two different types of memory: a higher level corticolimbic circuit for semantic memory and a lower level corticostriatal circuit for the more primitive habit memory, which is sometimes referred to as procedural or implicit memory. Savant memory is characteristically very deep, but exceedingly narrow within the confines of the accompanying special skill.

Males Outnumber Females in Both Autism and Savant Syndrome

Males outnumber females in savant syndrome by an approximate of 6:1 ratio compared to an approximate of 4:1 ratio in autistic disorder. In explaining that finding, Geschwind and Galaburda (1987) point out in their work on cerebral lateralization that the left hemisphere normally completes its development later than the right hemisphere and is thus subjected to prenatal influences, some of which can be detrimental, for a longer period of time. In the male fetus particularly, circulating testosterone, which can reach very high levels, can slow growth and impair neuronal function in the more vulnerably exposed left hemisphere, with actual enlargement and shift of dominance favoring skills associated with the right hemisphere. A “pathology of superiority” was postulated, with a compensatory growth in the right brain as a result of impaired development or actual injury to the left brain.

This finding explains why the savant skills described above tend to be “right brain” abilities. It may account as well for the high male/female ratio in other disorders, including autism itself, since left hemisphere dysfunction is often seen in autism (Rimland 1964; Treffert 2010). Other conditions, such as dyslexia, delayed speech, and stuttering, also have a high male predominance in incidence, which may be a manifestation of the same left hemisphere growth interference in the prenatal period described above.

Intelligence and Savant Skills

When Down coined the term “idiot savant,” it was immediately linked with mental retardation since at that time “idiot” referred to an IQ below 25. As it turns out, that was not only a regrettable term for

present day usage, but it was also in error since almost all reported cases have occurred in persons with an IQ above 40. That early association with mental retardation has led to the erroneous impression that savant syndrome is always associated with cognitive impairment. That is not the case. *Low IQ is not a requisite for savant syndrome.*

IQ levels in savant syndrome can range from below 40, which is quite rare, to as high as 140. The majority of savants have IQ levels between 40 and 70, but as many as 25% have IQ scores above 70. Bolte and Poustka (2004), for example, compared performance of 33 savant and 26 nonsavant autistic subjects on the Wechsler Adult Intelligence Scale-Revised (WAIS-R). Full-scale IQ scores for the savants ranged from 38 to 128, with a mean of 83.3; the nonsavant autistic control group IQ levels were somewhat lower (71.4) but that was not statistically significant. Miller (1998), in a review of studies where detailed IQ information was available, found that the mean overall IQ/IQ estimate for savants with autism was 71 (range 40–99) with the mean verbal IQ of 77 (range 52–114) and mean nonverbal IQ of 75 (range 47–92). Howlin et al. (2010) found, in their 10-year follow-up on 137 autistic savants, that nonverbal ability, using a variety of tests appropriate to each individual, gave a mean performance level of 69.9 (range 28–135). Verbal ability scores in that group showed a mean verbal IQ/or ratio IQ estimate (based on any test) of 52.9 (range 7–134).

Thus, although savant syndrome *can* occur in persons with autism with very low IQ scores, in the majority of cases IQ falls within the mild learning disability range.

Some autistic savants do have IQ levels in the superior range, and wide scatter on intelligence testing is a consistent characteristic of this group. Because of the wide scatter on such testing, some investigators have concluded that savant syndrome argues for “multiple intelligence” theory against “general intelligence” and the validity of the g factor (Treffert 2010). Whatever the case, while some savants do have IQ scores below 70, having an IQ score above 70 does not “disqualify” someone from having savant syndrome.

“Treatment” and “Training the Talent”

Savant syndrome is a combination of special skills grafted on to some underlying disability such as autism. Any formal “treatment” therefore would be directed at the underlying disability. With respect to the special skills, appropriate intervention is called “training the talent.”

In 1930, Phillips framed the question of dealing with special skills this way: “The problem of treatment comes next. ...is it better to eliminate the defects or train the talent?” Experience since that time shows that question need not be either/or. Instead, by “training the talent,” one can actually help ameliorate the “defect.” Supporting and nurturing whatever the special skill – music, art, and math – leads eventually to improved language, social, and daily living skills *without trade-off or loss of those special skills* as progress continues in other areas of functioning and learning. The special skills are a way of engaging the savant. They are not frivolous or interfering. They are a form of expression and can be a “conduit toward normalization,” with the eventual goal of channeling those abilities more usefully.

Clark (2001) developed a savant skill curriculum using a number of successful strategies currently employed in the education of gifted children (enrichment, acceleration, and mentorship) and autism education (visual supports and social stories) in an attempt to channel and apply, usefully, the often nonfunctional obsessive savant and splinter skills of a group of students with autism. This special curriculum did prove highly successful in the functional application of savant skills and an overall reduction in the level of autistic behaviors in many subjects. Improvements in behavior, social skills, and academic self-sufficiency occurred, along with better communication skills in some subjects. These strategies are woven into a number of successful approaches now in various autism treatment centers and classrooms.

Simultaneously, some specialized schools are developing, such as Soundscape Centre in England which is the only specialized educational facility in the world uniquely dedicated to the needs and potential of persons with sight loss

and special musical abilities, including musical savants. Some savants with special graphic abilities have now graduated from prestigious art schools, and some with special musical abilities have likewise graduated from equally prestigious music schools. There are also a number of savants with a Ph.D. degree in mathematics or other fields.

Grandin and Duffy's book – *Developing Talents: Careers for Individuals with Asperger Syndrome and High-Functioning Autism* (2005) – is an excellent, practical resource which outlines methods for helping children “develop their natural talents” using “drawing, writing, building models, programming computers,” and similar skills to help in the search for eventual work experience.

The Case of Nadia and the “Dreaded Trade-off” Versus Progression of Skills

In 1978, Selfe reported the case of Nadia who lost her special art skills when exposed to traditional schooling. This raised the question whether there might be a “dreaded trade-off” of special skills for acquisition of better language, social, and daily living skills. Experience has shown this to be not the case, and Nadia's experience is the exception rather than the rule.

Rather, based on following certain savants over a number of years, a pattern of progression of savant skills emerges first with *replication*, then *improvisation*, and finally, in some cases, the capacity to be *creative*. A fuller description of this pattern of progress in a number of savants can be found in Treffert's, 2010 *Islands of Genius: The Bountiful Mind of the Autistic, Acquired and Sudden Savant* book or his chapter on savant syndrome in *Autism and Talent* (Happé and Frith 2010).

Family History and Savant Syndrome

The role of family history in savant syndrome is an unsettled one. Two studies, one with 25 savants and another with 51 subjects, showed relatives with special skills in *some*, but not all, cases (Duckett 1976; Young 1995). Young traveled to a number of countries and met with 51 savants and

their families. There was a family history of similar skills in some, but not all, cases, but even in the absence of a history of a specific skill, there was a familial predisposition toward high achievement.

Might there be a gene for savant syndrome? Here, results are also mixed. Nurmi et al. (2003), using parental reports on special skills, compared 21 multiplex autism families (multiple affected members, mainly affected sibling pairs and their parents) which he identified as “savant skills positive” with 74 similar families that he identified as “savant skills negative.” Results showed “the subset of 21 savant skills positive families yielded significantly increased evidence for linkage to 15q11-q13.” The 73 families in which the individuals with autism had few or no savant skills showed no evidence of such linkage. Ma et al. (2005) attempted to replicate those findings using the savant skill factor (SFF) from the Autism Diagnostic Interview-Revised (ADI-R) instead of parental reports. Their results “failed to demonstrate linkage to 15q-11-q13” in their sample. But the differing definitions for savants in these two studies make for a difficult comparison.

The exploration of family history in savant syndrome, based on experience thus far, suggests that “family history” needs to extend beyond first-degree relatives to uncles, aunts, and cousins, for example, and beyond the present generation to prior generations in the family. Only with that more extensive and careful exploration will the contribution of family history to savant syndrome become more clear.

Future Directions

There has been more progress toward better understanding savant syndrome in the past 25 years than in the prior 100 since Down's first description of this extraordinary condition in 1887. Many unanswered questions still remain but interest in exploring those is accelerating, particularly with the rapid emergence of newer technologies that allow the study of brain

function, not just brain *structure*. Savant syndrome provides a unique window into the brain, using those new techniques, to learn more talent, memory, and creativity itself, not only in savants but in everyone else as well. Future direction of research to better understand the savant and services to better help him or her to reach their full potential will include what follows.

New Technologies

The brain sits well protected in the rigid box surrounding it. It cannot be “scoped” like other organs and biopsies are problematical. Therefore, various imaging techniques are the least intrusive way of studying brain structure and organization. CT and MRI scans permit high-resolution images of brain architecture, including deep structures. Even more illuminating though are functional studies such as PET and SPECT. Those require isotopes however, so functional MRI (fMRI) is more often used to look at the brain at work. And it is study of brain function that will ultimately provide keys to better understanding not just savant syndrome but autism itself.

Some theorize that autism, and savant syndrome, is a result of abnormal “connectivity” in the brain. Diffusion tensor imaging (DTI) measures water flow in the complex neuronal network, and diffusion tensor tracing (DTT) traces those actual connections between the hemispheres, within the hemispheres, and between the upper and lower brain structures, projecting tracings of the actual fibers and the pathways that connect those fiber tracts. Casanova and Trippe (2010) speculate that minicolumn organization (or disorganization) may be vital in autism itself, and Mottran et al. (2006) and colleagues extend changes in synaptic plasticity to account for some unusual synaptic connectivity in savant syndrome. Studies of such connectivity patterns are also underway.

One of the problems with studying savant syndrome with these technologies is that it requires virtual immobilization in the MRI and similar machines. It is impossible of course to play a piano, or sculpt, in an MRI machine. So in order

to truly view the brain while the savant is “at work” with their special skill, methods need to be devised to allow brain function studies with less immobilization. Near-infrared spectroscopy (NIRS) measures hemoglobin flow using a scalp cap and does not require the individual to be immobile and will be applied in such studies. Also new magnetoencephalography technology provides a great deal of information beyond traditional EEG in terms of sensitivity and magnification, and it can provide extremely detailed information about the electrical activity in both surface and deep areas in the autistic and savant brain.

A Savant Syndrome Registry and Broad-Based, Controlled Studies

The relative rarity of savant syndrome, contrasted with the attention-getting extraordinary nature of some abilities, has led to largely anecdotal reporting of cases through the years. The lack of a standardized definition of “savant syndrome,” the absence of uniform neuropsychological testing of savants, and the lack of quantification of savant skills into distinct categories has hampered research efforts with large sample, control groups. To that end, a *worldwide savant syndrome registry* has been recently organized by Treffert and Rebedew collecting cases largely from the savant syndrome web site at www.savantsyndrome.com along with professional journal reports, personal correspondence, and media descriptions. To date, 309 cases of savant syndrome from 33 countries have been entered into that registry. Those cases have been categorized by ability, disability (formal diagnosis) age, gender, family history, medical history, congenital or acquired type, geographic location, and a number of other variables. From that analysis, now underway, there can be determination, on a large sample basis, of the distribution of autism versus other diagnoses as the underlying disability, the frequency of various abilities, a breakdown of congenital cases versus acquired cases, gender distribution of savant skills, age of savants, family history, medical history, educational level, vocational and

socioeconomic status, and a variety of other parameters. Cases will be added to that registry as they are discovered and reported. A second phase of that “registry” will consist of gathering information from an even more detailed questionnaire completed by those savants, families, and caregivers willing to do so.

Having savants registered by geographic location should help those researchers hoping to find willing subjects in their particular countries to participate in these larger case studies including control groups.

Exploring the Interface Between Prodigy, Genius, and Savant Syndrome and Other Quandaries

As more savant cases come to attention, both congenital and acquired, the interface between prodigy, genius, and savant syndrome becomes a natural area for exploration. These conditions likewise provide a fertile field for investigation regarding the nature of talent and the role of “innate” versus “learned” abilities, the age old nature versus nurture argument. They also raise the question of “multiple” intelligences versus a “general” intelligence, or “g” factor. Savants, whether congenital or acquired, seem to “know things they never learned,” which gives entry to the area of genetic or ancestral memory for exploration as well. Finally, savant syndrome, both congenital and acquired, provides a striking confirmation of brain plasticity and is a natural workshop to explore those mechanisms as well. Research into these areas is already underway, and more will follow.

Development of Methods of “Training the Talent” and Resources to Use Those Special Skills

A better understanding of, and appreciation for, savant skills has taken them out of the “Gee whiz, look at that” category and cast them into a vigorous treatment – “training the talent” – effort with the hope those special abilities can be used vocationally in many cases fostering increasing independence and community acceptance. Studies are

underway, and will increase in the future, as to what are the best methods to use in education efforts: special classrooms, regular classrooms, or hybrid approaches. Already, some programs have successfully placed savant and neurotypical “gifted and talented” students together in combined programs. Private schools specifically designed for, and dedicated to elementary, middle school and high school students with savant syndrome and high-functioning autism have likewise emerged. Those provide particular populations for research on program effectiveness and outcome.

But while there has been the development of an increasingly rich network of educational services for K-12 students with savant syndrome, autism, and Asperger’s, until recently those students, now adults, have faced a very barren landscape of similar services for the over-18 age group. But that is changing for the better, and there is a solid effort underway to incorporate more adult savants, with their special talents, into the regular workforce. Grandin and Duffy (2005) have provided a good blueprint for developing talents as a pathway to careers that utilize the unique skills of the savant. There are now employment agencies in several countries that successfully specialize just in the matching of individuals with Asperger’s with willing employers. The years ahead will see more and more such efforts as the network of services for the over-18-year-old autism and savant populations expands to provide a comparable network of services available to them during their regular school years. A very comprehensive Transition Tool Kit is available from *Autism Speaks* either as a printed version or downloaded at the *Autism Speaks* web site (request Autism Speaks Transition Tool Kit). It fills a need for practical information for persons with savant syndrome, and their families, in making the transition to adulthood.

A Challenge to the Capabilities of All of Us

Until savant syndrome can be fully understood and explained, there can be no explanation and understanding of the full intricacy and capacity of the human brain. For no model of brain function,

including memory, will be complete until it can fully incorporate and elucidate how it is possible to have such a jarring juxtaposition of ability and disability within the same individual. While that question has intrigued all the researchers approaching it, this past century and a quarter since first described by Down, newer technologies now make it possible to more clearly unravel this remarkable condition, with all the implications it has for better understanding not just the savant but in better understanding brain function in everyone in terms of talent, skills, learning, memory, and creativity itself.

But there is more to savant syndrome than just neurons, circuits, and the brain's marvelous intricacy. Future research findings will surely be of great *scientific* interest. But there are many lessons to be learned as well of *human* interest from these remarkable people and their equally remarkable families, caregivers, teachers, and therapists that surround them. These people that care *about* the savant, as they care lovingly *for* them, provide valuable examples of the power of unconditional positive regard, support, encouragement, patience, and optimism that benefit anyone “differently abled” (as it is sometime called) with savant syndrome and autism. The investigation of both the scientific interest of the savant, as well as the human interest involved, can provide valuable and unique insights into actualizing and maximizing human potential in whomever, and in whatever quantity, it exists within all persons.

See Also

- [Autistic Disorder](#)
- [Autistic Savants](#)
- [Weak Central Coherence](#)

References and Reading

Bolte, S., & Poustka, F. (2004). Comparing the profiles of savant and non-savant individuals with autistic disorder. *Intelligence*, 32, 121–131.

- Casanova, M., & Trippe, J. (2010). Radial cytoarchitecture and patterns of cortical connectivity in autism. In F. Happe & U. Frith (Eds.), *Autism and talent*. London: Oxford University Press.
- Clark, T. R. (2001). *The application of savant and splinter skills in the autistic population through curriculum design: A longitudinal multiple replication case study unpublished doctoral dissertation*. Sydney: University of New South Wales.
- Critchley, M. (1979). *The divine banquet of the brain*. New York: Raven.
- Down, J. L. (1887). *On some of the mental affections of childhood and youth*. London: Churchill.
- Duckett, J. (1976). *Idiot savants: Superspecialization in mentally retarded persons*. Doctoral Dissertation, Department of Special Education, University of Texas, Austins.
- Geschwind, N., & Galaburda, A. M. (1987). *Cerebral lateralization: Biological mechanisms, associations and pathology*. Cambridge, MA: MIT Press.
- Grandin, T., & Duffy, K. (2005). *Developing talents: Careers for individuals with asperger syndrome and high functioning autism*. Shawnee Mission: Autism Asperger Publishing.
- Happe, F., & Frith, U. (2010). *Autism and talent*. London: Oxford University Press.
- Hermelin, B. (2001). *Bright splinters of the mind*. London: Jessica Kingsley.
- Hill, A. L. (1977). Idiot savants: Rate of incidence. *Perceptual and Motor Skills*, 44, 161–162.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2010). Savant skills in autism: Psychometric approaches and parental reports. In F. Happe & U. Frith (Eds.), *Autism and talent*. London: Oxford University Press.
- Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of Autism and Childhood Schizophrenia*, 1, 119–145.
- Kapur, N. (1996). Paradoxical functional facilitation in brain-behavior research. *Brain*, 119, 1775–1790.
- Ma, D. Q., Jaworski, J., Menoid, M. M., Donnelly, S., Abramson, R. K., Wright, H. H., et al. (2005). Ordered-subset analysis of savant skills in autism for 15q-11-q13. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 135B, 38–41.
- Miller, L. (1998). Defining the savant syndrome. *Journal of Developmental Physiology*, 10, 73–85.
- Mishkin, M., Malamut, B., & Bachevalier, J. (1984). Memories and habits: Two neural systems. In G. Lynch, J. L. MacGaugh, & N. M. Weinberger (Eds.), *Neurobiology of learning and memory*. New York: Guilford Press.
- Moritz, K. P. (1783). *Gnothi Sauton oder Magazin der Erfahrungsseelenkunde als ein Lesebuch für Gelehrte und Ungelehrte*. Berlin: Mylius.
- Mottran, L., Dawson, M., Soulieres, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36, 27–43.

- Nurmi, E. L., Dowd, M., Tadevosyan-Leyer, O., Haines, J., Folstein, S., & Sutcliffe, J. S. (2003). Exploratory subsetting of autism families based on savant skills improves evidence of genetic linkage to 15q11-q13. *Journal of the Academy of Child Adolescent Psychiatry*, 42, 856–863.
- Powell, D. (2006). We are all savants. *Shift: At the Frontiers of Consciousness*, 9, 14–17.
- Rimland, B. (1964). *Infantile autism: The syndrome and its implications for a neural theory of behavior*. New York: Appleton-Century-Crofts.
- Rimland, B. (1978). Savant characteristics and their cognitive implications. In G. Serban (Ed.), *Cognitive defects in the development of mental illness*. New York: Brunner/Mazel.
- Rush, B. (1789). Account of a wonderful talent for arithmetical calculation in an African slave, living in Virginia. *American Museum*, 5, 62–63.
- Saloviita, T., Ruusila, L., & Ruusila, U. (2000). Incidence of savant syndrome in Finland. *Perceptual and Motor Skills*, 91, 120–122.
- Tredgold, A. F. (1914). *Mental deficiency (amentia)*. New York: William Wood.
- Treffert, D. A. (1988). The idiot savant: A review of the syndrome. *The American Journal of Psychiatry*, 145, 563–572.
- Treffert, D. A. (2006). Dr. Down and developmental disorders. *Journal of Autism and Developmental Disorders*, 36, 965–966.
- Treffert, D. A. (2010). *Islands of genius: The bountiful mind of the autistic. Acquired and Sudden Savant*. London: Jessica Kingsley.
- www.daroldtreffert.com background information on Darold A. Treffert, M.D.
- www.savantsyndrome.com a comprehensive web site for both text and video on savant syndrome in general, and specific savants.
- Young, R. (1995). *Savant syndrome processes underlying extraordinary abilities*. Unpublished doctoral dissertation. University of Adelaide.

condition is rare, but because of the striking skills that some savants have, it has captured the attention of the general public and researchers. Savant talents vary from exceptional mathematical abilities, to prodigious musical skills, to extraordinary artistic talent.

Historical Background

The first documented case of savant syndrome appeared in the literature in the late eighteenth century. In 1887, Dr. J. Langdon Down, well-known for his identification of Down's syndrome, described savant syndrome as its own condition. In his lectures he described ten different cases that had been identified (Treffert 2014). Until several decades ago, savants were called “idiot savants.” This title is not only jarring to the modern ear, but entirely inaccurate. “Idiot” was formerly used to describe those with an IQ under 25. However, almost all individuals with savant syndrome have IQs above 40 (Treffert 2009). In the late twentieth century, the condition was renamed savant syndrome (Rimland and Hill 1984). In 1980, autism was first listed in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) under infantile autism (Camulli and Goh 2018). In 2013, the diagnosis in the DSM was generalized to autism spectrum disorder. Although some symptoms of savant syndrome are included in the DSM-V, it is not categorized as a distinct diagnosis.

Various definitions of savant syndrome have been proposed since it was first identified in 1887. Due to immense variation in both the special skills and underlying developmental disabilities associated with it, characterizations of the condition have focused mainly on the juxtaposition between the talent and the underlying impairments (Bennett and Heaton 2017).

Savant Syndrome

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Definition

Savant syndrome is a disorder characterized by a niche area of immense talent that contrasts with a general intellectual impairment. Many people with savant syndrome also have autism. The

Current Knowledge and Research

While savant syndrome is more common in individuals with autism than in the general population, it is still extraordinarily rare. According to

the Centers for Disease Control and Prevention, between 1% and 2% of the global population has autism spectrum disorder (Baio et al. 2018). Of that number, about 10% of individuals with autism have some form of savant syndrome (Camulli and Goh 2018). Due to the rareness of the disorder, research into its mechanisms is sparse. The research that does exist relies primarily on case studies rather than on experimental studies. This presents challenges in drawing significant conclusions about the basis of the disorder.

It is a common misconception that savants are only found among those with autism. Only 50% of people with savant syndrome have a diagnosis of autism. The other 50% have some other underlying developmental disability or CNS disorder, including Prader-Willi syndrome or Smith-Magenis syndrome (Corrigan et al. 2012). Savant syndrome appears far more frequently in males than in females. Although this is true of autism as well, the imbalance is greater in savant syndrome. Whereas autism is four times more likely to occur in males than in females, savant syndrome is six times more likely to occur in males (Treffert 2009). Autistic savants and autistic non-savants are similar in that they both show deficits in communication abilities and other social skills (Bennett and Heaton 2017). There are also no significant differences in IQ in verbal, performance, or processing speed; however, savants tend to score more highly on working memory.

The core symptoms of savant syndrome include a developmental disability, a specific skill, and interest in that specific area (Camulli and Goh 2018). Researchers and clinicians describe savants as falling on a spectrum, depending on the nature and level of accomplishment within their skillset. Talented savants typically have a narrowed skillset in music, art, or some other area that contrasts with their disability (Treffert 2009; Camulli and Goh 2018). At the extreme end of the spectrum, prodigious savants have abilities that would be considered extraordinary by any measure. This form of savant syndrome is extremely rare. It has been estimated that there are only about 25 prodigious savants in the world presently (Camulli and Goh 2018).

Most commonly, savants' skills arise or are identified at a very young age. Nevertheless, there have been a number of documented cases of savant syndrome being acquired at a later age, typically following a brain injury (Lythgoe et al. 2005; Treffert 2009). With the exception of one report, in which the savant's skills deteriorated over time, savants are able to improve their skills through training and practice (Hou et al. 2000; Treffert 2009).

Among the unusual range of talents of savants, several special abilities are more commonly seen. The most frequent is calendar calculations, in which an individual can rapidly determine a specific day of the week that a date fell on, sometimes going back centuries, and often correctly accounting for leap years (Brink 1980). While a typical individual could learn this skill, it comes more effortlessly to a subset of people with savant syndrome. Other documented abilities include artistic expression, mechanical abilities, or musical skills (Brink 1980; Corrigan et al. 2012). Perfect pitch, while rare in the general population, is far more common among savants. Additionally, mastering several languages (Smith et al. 1993) has been documented. People with savant syndrome tend to have a very narrow range of skills, although in a few cases, individuals have multiple talents (Treffert 2009). Accompanying their remarkable talent, people with savant syndrome often have exceptional memory.

Some researchers have described people with savant syndrome as having exceptional memory and the ability to learn within their skillset quickly, but lacking the creativity to improvise or compose new music or art. This, however, is untrue. There have been a number of documented cases of savants showing creativity in their area of expertise. In one case, a man named Leslie who was very skilled musically began by reproducing songs on the piano. This in itself took a high level of skill, as he could replicate very advanced and long pieces after hearing them only once. After further developing his talent, he could later improvise or compose his own pieces (Treffert 2009). This demonstrates the complicated nature of the abilities of those with savant syndrome and the uniqueness of each case.

Some characteristic physical phenotypes are more likely to manifest in individuals with savant syndrome. For example, visual impairment is more common in savants with significant musical skills (Treffert 2014). There have also been several cases of savants with epilepsy or seizure disorders.

The most carefully documented cases are of prodigious savants, despite their rarity. The first reported case in 1783 described a man named Jedediah Buxton as a “lightning calculator” because of his exceptional mathematical abilities (Treffert 1988). Several artistic savants have been described, including Gottfried Mind, born in 1768, who was exceptionally skilled at drawing cats from memory (Hou et al. 2000). Kiyoshi Yamashita, born in Japan in 1922, was called the “van Gogh of Japan” (Hou et al. 2000). Stephen Wiltshire is a modern example of an artistic savant. He became well-known because of his accuracy in drawing cities after seeing them only once (Höfer and Röckenhaus 2015).

Musical savants are also well documented. Blind Tom, a former slave, became well-known in the nineteenth century for his remarkable piano playing abilities. His extraordinary memory allowed him to replicate a song perfectly after only hearing it once. Derek Paravicini, a modern-day musical savant, is also blind and autistic. Similar to Blind Tom, Derek can play a song flawlessly after hearing it once (Moss 2007). He can also improvise and create music on the spot. Derek’s musical genius would be remarkable in a typically developing person. However, Derek is particularly notable in that he has significant cognitive disabilities, in addition to being blind (Moss, 2007). When he first played the piano at the age of 3, he did not realize that the piano is supposed to be played using one’s fingers. Until he was formally taught, he played with his elbows or the backs of his hands (Moss 2007). Now, he displays his talents widely and is celebrated for them.

Other prodigious savants are renowned for their great memory. Perhaps the most well-known savant is the fictional character, Raymond Babbitt, from the movie *Rain Man*. The character was based on a real individual named Laurence Kim Peek, who has memorized thousands of

books and has an excellent memory for geography. He is able to read very quickly, as he reads one page with his left eye and one with his right eye and has no corpus callosum (Brogaard 2012).

While many reports of savant syndrome describe cases in which the individual was born with notable skills or developed talents at a young age, several cases have been described of “acquired” savant syndrome. In acquired savant syndrome, significant talents emerge after a traumatic brain injury. After a gunshot wound to the head, Mr. Z was reported to have lost the ability to speak, read, or write. However, he gained significant skills in mechanics, geography, and art (Brink 1980). Researchers theorized that the injury to his left hemisphere led to further development of the right hemisphere, which resulted in the emergence of skills in these areas.

No single cognitive or biological mechanism has been proposed that can account for all cases of savant syndrome. One historical explanation was that savants’ exceptional skills were the result of a tendency to practice them intently or even excessively, due to more rigid behavioral patterns. However, several studies have shown this not to be the case. Although savants are more obsessive than neurotypical control groups, there is no difference in obsessive behavior between savants and non-savants with autism (Bennett and Heaton 2017). Given that the skills of savants are remarkable when compared to typical children who practice seriously, a purely behavioral explanation of this kind is unlikely. Rather, the disorder is ultimately more likely to be explained in terms of neuroanatomical abnormalities and/or dysfunctional neurobiological mechanisms.

There have been several case studies in which brain structure has been studied in savant syndrome. A 63-year-old musical and artistic savant with a diagnosis of ASD was found to have striking asymmetries in the amygdala, with the right side 24% larger than the left, as well as an asymmetrical caudate nucleus and putamen. Magnetic resonance spectroscopy revealed concentrations of the neurotransmitters GABA and glutamate more than two standard deviations below controls. Diffusion tensor imaging (DTI) showed greater white matter densities in most neural

structures as compared to controls with the greatest disparity occurring in the hippocampus (Corrigan et al. 2012). Kim Peek (see above) had no corpus callosum and no anterior or posterior commissures. Additionally, the morphology of the cerebellum and left hemisphere was distorted (Brogaard 2012; Corrigan et al. 2012). A number of other cases have been examined with MRI, fMRI, and PET, and although the abnormalities have not been consistent across patients, most savants imaged had some anatomical abnormalities or altered activation of neuronal pathways when performing their talents (Corrigan et al. 2012). Which of these abnormalities, if any, contributed to the specific areas of genius or to the developmental disabilities in these individuals is unclear.

Evidence from the case studies described above, that specific brain structures show exuberant growth, is at its face, consistent with the view that savant syndrome derives from increased development of certain structures and the connections between them, at the expense of connectivity in other areas of the brain. This theory was initially proposed to account for the striking overlap of structural and functional features between individuals with ASD and individuals with high IQ. For example, researchers have identified a statistically significant overlap between genes that are associated with autism and genes that are associated with high IQ (Crespi 2016). Similarly, both people with autism and those with higher IQs tend to have larger brain volumes and more brain growth (Crespi 2016).

These findings are somewhat paradoxical given that individuals with autism on average tend to have lower IQs than the population at large. Crespi (2016) seeks to resolve this paradox by suggesting that people with autism may have higher than average IQs in certain areas. These areas of functional gain could result from higher levels of local neuronal connectivity as compared to controls. While support for this theory in the realm of autism is constrained by limited evidence of discrepancies on IQ subtests (Charman et al. 2010), it may be more applicable to savant syndrome, where these kinds of discrepancies are a hallmark of the condition. DTI studies as well as

resting state fMRI studies on a larger number of savants would be helpful in testing this hypothesis further.

Greater neuronal connectivity in the savant brain could support the theory that savants have enhanced perceptual functioning (Mottron et al. 2009). Many savant skills, including skills in music, art, math, and reading, follow a series of consistent patterns. Superior pattern recognition, therefore, may underlie some savants' exceptional skills. For example, Derek Paravicini, a musical savant with perfect pitch, is far better at recognizing musical cords than a non-savant, which likely contributes to his prodigious musical talents. Superior pattern completion, or the ability to recall a missing component of a known signal, could also contribute to some savants' areas of greater ability (Mottron et al. 2009). For example, Stephen Wiltshire can complete a two-dimensional drawing of the three-dimensional city after seeing it only once. He achieves this by beginning with notable buildings and structures and then filling in details (Höfer and Röckenhaus 2015). Prodigious talents could also be accounted for by an enhanced ability to detect deviations from a usual pattern, as has been documented in some cases of autism (Mottron et al. 2009).

Genetic linkage studies in savant populations have been limited due to small sample sizes. However, one study found that there may be evidence to suggest that the region 15q11-q13 contributes to the development of savant skills (Nurmi et al. 2003). Previous studies have indicated that duplications in this region may produce the autism phenotype, and deletions in the paternal chromosome or maternal chromosome result in Prader-Willi syndrome or Angelman syndrome, respectively (Nurmi et al. 2003). A significant difference was found in HLOD scores, a statistic that denotes evidence of genetic linkage, for 21 families positive for savant traits compared to 73 autistic families without the trait. The most significant HLOD score within the savant skills group was associated with the GABRB3 gene, which codes for a subunit of GABA receptors (Nurmi et al. 2003). GABA is the primary inhibitory neurotransmitter in the brain. Variations in

this gene have previously been indicated in individuals with Prader-Willi syndrome as well as other conditions. A second study, however, failed to replicate these findings (Ma et al. 2005).

Future Directions

Although research in this area has progressed significantly since savant syndrome was first described, many questions remain unanswered and the condition is still largely unexplained. Researchers still seek to understand the biological basis for the disorder as a whole and the specific mechanism or mechanisms that result in stark disparities between areas of prodigious talents and otherwise low intellectual abilities. The greatest benefit will come from studies that integrate analyses of brain morphology, connectivity, and function.

Like autism, savant syndrome is a heterogeneous disorder. Hence, no single explanation can explain all manifestations. Researchers need to investigate not only general mechanisms underlying savant syndrome but also variations in the disorder. For example, what causes one savant to be a gifted musician and another to be a gifted artist? The answer to this question may relate to environmental factors as much as individual predisposition.

The principle challenge to addressing these questions is the rareness of the disorder. Small sample sizes have been the norm of past studies and will likely continue to be so in the future. Despite its rareness, however, the exceptional nature of this disorder warrants our attention. Not only may this endeavor provide insight into the etiology of the disorder, but it may also shed light on mechanisms underlying the development of extraordinary abilities in the broader population.

See Also

- Autistic Savants
- High-Functioning Autism (HFA)
- Verbal Intelligence

References and Reading

- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., . . . & Dowling, N. F. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summaries*, 67(6), 1–23. <https://doi.org/10.15585/mmwr.ss6706a1>.
- Bennett, E. A., & Heaton, P. (2017). Defining the clinical and cognitive phenotype of child savants with autism spectrum disorder. *Current Pediatric Research*, 21(1), 140–147.
- Brink, T. L. (1980). Idiot savant with unusual mechanical ability: An organic explanation. *The American Journal of Psychiatry*, 137(2), 250–251.
- Brogaard, B. (2012). Kim Peek, the Real Rain Man. Retrieved from <https://www.psychologytoday.com/us/blog/the-superhuman-mind/201212/kim-peek-the-real-rain-man>.
- Camulli, J. E., & Goh, L. A. (2018). Re-conceptualising autistic savantism as a spectrum syndromic disorder: A sequel to the case study of a young adult savant artist. *European Journal of Special Education Research*, 3(4), 185–204.
- Charman, T., Pickles, A., Simonoff, E., Chandler, S., Loucas, T., & Baird, G. (2010). IQ in children with autism spectrum disorders: Data from the Special Needs and Autism Project (SNAP). *Psychological Medicine*, 41(3), 619–627. <https://doi.org/10.1017/S0033291710000991>.
- Corrigan, N. M., Richards, T. L., Treffert, D. A., & Dager, S. R. (2012). Toward a better understanding of the savant brain. *Comprehensive Psychiatry*, 53, 706–717.
- Crespi, B. J. (2016). Autism as a disorder of high intelligence. *Frontiers in Neuroscience*, 10, 300.
- Höfer, P., & Röckenhaus, F. (2015). Beautiful minds. Retrieved from <https://www.amazon.com/Beautiful-Minds-Petra-Höfer/dp/B015RRQR20>. Accessed 27 Sept 2019.
- Hou, C., Miller, B. L., Cummings, J. L., Goldberg, M., Mychack, P., Bottino, V., & Benson, F. (2000). Artistic savants. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 13(1), 29–38.
- Lythgoe, M. F., Pollak, T. A., Kalmus, M., Haan, M. D., & Chong, W. K. (2005). Obsessive, prolific artistic output following subarachnoid hemorrhage. *Neurology*, 64(2), 397–398. <https://doi.org/10.1212/01.wnl.0000150526.09499.3e>.
- Ma, D., Jaworski, J., Menold, M., Donnelly, S., Abramson, R., Wright, H., . . . & Cuccaro, M. L. (2005). Ordered-subset analysis of savant skills in autism for 15q11-q13. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 135B(1), 38–41. <https://doi.org/10.1002/ajmg.b.30166>.
- Moss, S. (2007). He attacked the keyboard. *The Guardian*.
- Mottron, L., Dawson, M., & Soulières, I. (2009). Enhanced perception in savant syndrome: Patterns, structure and

- creativity. *Philosophical Transactions of the Royal Society B*, 364, 1385–1391.
- Nurmi, E. L., Dowd, M., Tadevosyan-Leyfer, O., Haines, J., Folstein, S. E., & Sutcliffe, J. S. (2003). Exploratory subsetting of autism families based on savant skills improves evidence of genetic linkage to 15q11-q13. *Journal of the American Academy of Child and Adolescent Psychiatry*, 42(7), 856–863.
- Rimland, B., & Hill, A. L. (1984). Idiot savants. In *Mental retardation and developmental disabilities* (pp. 155–169). Boston: Springer.
- Smith, N. V., Tsimpli, I.-M., & Ouhalla, J. (1993). Learning the impossible: The acquisition of possible and impossible languages by a polyglot savant. *Lingua*, 91(4), 279–347. [https://doi.org/10.1016/0024-3841\(93\)90002-e](https://doi.org/10.1016/0024-3841(93)90002-e).
- Treffert, D. A. (1988). The idiot savant: A review of the syndrome. *American Journal of Psychiatry*, 145(5), 563–572.
- Treffert, D. A. (2009). The savant syndrome: An extraordinary condition. A synopsis: Past, present, future. *Philosophical Transactions of the Royal Society B*, 364, 1351–1357.
- Treffert, D. A. (2014). Savant syndrome: Realities, myths and misconceptions. *Journal of Autism and Developmental Disorders*, 44, 564–571.

SB5

- [Stanford-Binet Intelligence Scales and Revised Versions](#)

SCAN-A (Adults)

- [Screening Test for Auditory Processing Disorders](#)

SCAN-C (Children)

- [Screening Test for Auditory Processing Disorders](#)

Scavenging

- [Pica](#)

Schaffer v. Weast (Burden of Proof in Moving a Child to Another Placement)

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Synonyms

[Schaffer v. Weast and the Burden of Persuasion](#)

Definition

The party seeking relief bears the burden of persuasion under the Individuals with Disabilities Education Act (IDEA). A burden of persuasion is “a party’s duty to convince the fact finder to view the facts in a way that favors the party.”

In *Schaffer v. Weast*, the United States Supreme Court ruled that the party challenging the status quo bears the burden of persuasion under the IDEA. In *Schaffer*, the plaintiff, Brian Schaffer, suffered from learning disabilities and speech-language impairments. Believing that Brian required more intensive attention, Brian’s parents initiated a due process hearing challenging the public school’s Individual Education Plan (IEP). The Supreme Court ruled that Brian’s parents, as the party seeking relief from the school’s IEP, must prove that the school’s proposed IEP was inadequate to meet Brian’s needs.

The Court reached this decision for various reasons. First, the Court noted that, with few exceptions, under Anglo-American law, the burden of persuasion is traditionally placed on the party challenging the status quo. Second, there was no reason to believe that Congress intended to place the burden of persuasion on defendants. In addition, the Administrative Procedure Act (APA) governs initial IEP challenges. The APA places the burden of persuasion on plaintiffs. By incorporating the APA into the IDEA, Congress intended to place the burden of persuasion on plaintiffs. Third, pragmatically, placing the

burden on school districts as a matter of course would squander public funds because school districts would be forced to litigate every IEP prior to implementation. Congress has repeatedly amended the IDEA to reduce the expense of enforcement. Thus, Congress did not intend school districts to incur great costs litigating each IEP before implementation. Fourth, Congress provided for equal access to information for parents challenging an IEP. This includes parents' rights to review all records on their child, an independent evaluation of the child, and options rejected by the school board. Thus, Congress provided parents with some resources necessary to challenge an IEP.

Implications for ASD Students

Customarily, parents challenge IEPs. A school district challenging an IEP, however, would also assume the burden of persuasion.

The IDEA currently requires school districts to provide voluntary mediation sessions. These sessions attempt to resolve disputes without litigation. Additionally, parents are encouraged to play a major role in the design of services to meet the student's particular needs.

Litigation Strategies

Practitioners should be aware that school districts need not disclose evidence of how other similarly situated children with ASD have performed in either analogous or dissimilar placements. Parents challenging an IEP should obtain trained legal and psychological counsel as many practitioners report using knowledge and language beyond that of a lay person's understanding to their advantage. If parents cannot afford professional help, however, their complaint will not be dismissed because "parents have a recognized legal interest in the education . . . of their child." Parents who meet their burden are entitled to reimbursement for expenditures if the school district has not made a "free appropriate public education available to the child."

See Also

- [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- Caruso, D. (2010). Autism in the U.S.: Social movement and legal change. *American Journal of Law and Medicine*, 36, 483–514. Retrieved July 5, 2012, from Business Source Complete, Westlaw. http://web2.westlaw.com/find/default.wl?fn=_top&rs=WLV11.01&rp=%2ffind%2fdefault.wl&mt=208&vr=2.0&sv=Split&cite=36+AMJLM+483.
- Freed, L. G. (2009). Cooperative federalism post-Schaffer: The burden of proof and preemption in special education. *Brigham Young University Education and Law Journal*, 103, 109–110. Retrieved July 5, 2012, from Business Source Complete, Westlaw. http://web2.westlaw.com/find/default.wl?fn=_top&rs=WLV11.01&rp=%2ffind%2fdefault.wl&mt=208&vr=2.0&sv=Split&cite=2009+BYUELJ+103.
- Garner, B., Jackson, T., & Newman, J. (Eds.). (2004). *Black's law dictionary* (8th ed.). St. Paul: West.
- Harvard Law Review Association. (2007). The Supreme Court, 2006 term leading cases III. Federal statutes and regulations D. Individuals with disabilities education act 121. *Harvard Law Review*, 365, 367–370. Retrieved July 5, 2012, from Business Source Complete, Westlaw. http://web2.westlaw.com/find/default.wl?fn=_top&rs=WLV11.01&rp=%2ffind%2fdefault.wl&mt=208&vr=2.0&sv=Split&cite=121+HVLRL+365.
- Individuals with Disabilities Education Act. 20 U.S.C. § 1400.
- Individuals with Disabilities Education Act. 20 U.S.C. § 1412.
- Pierce v. Society of the Sisters of the Holy Names of Jesus and Mary, 268 U.S. 510, 534–535 (1925).
- Schaffer v. Weast, 546 U.S. 49, 49–67 (2005).
- Wasserman, L. (2009). The rights of parentally-placed private school students under the individuals with disabilities education improvement act of 2004 and the Need for Legislative Reform 2009. *Brigham Young University Education and Law Journal*, 131, 136. Retrieved July 5, 2012, from Business Source Complete, Westlaw. http://web2.westlaw.com/find/default.wl?fn=_top&rs=WLV11.01&rp=%2ffind%2fdefault.wl&mt=208&vr=2.0&sv=Split&cite=2009+BYUELJ+131.
- Wilson, J. (2007). Missing the big idea: The Supreme court loses sight of the policy behind the individuals with disabilities education act in Schaffer v. Weast. *Houston Law Review*, 44, 161–186. Retrieved July 5, 2012, from Business Source Complete, Westlaw. http://web2.westlaw.com/find/default.wl?fn=_top&rs=WLV11.01&rp=%2ffind%2fdefault.wl&mt=208&vr=2.0&sv=Split&cite=44+HOUCLR+161.

Schaffer v. Weast and the Burden of Persuasion

► [Schaffer v. Weast \(Burden of Proof in Moving a Child to Another Placement\)](#)

Schedule of Reinforcement

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Definition

General Definition

A schedule of reinforcement specifies the relation between one or more responses (i.e., response classes) and one or more reinforcing consequences. In addition to describing when a reinforcing consequence is available, different schedules of reinforcement have been shown to result in different patterns of responding (Ferster and Skinner 1957). Schedules of reinforcement should be considered during the planning phase for interventions for individuals with autism spectrum disorders (ASDs). In addition, schedules of reinforcement may need to be revised after an intervention has been implemented to ensure continued desirable outcomes and overall long-term success.

Schedules of reinforcement can be particularly important in various applied situations. Careful design and implementation of an appropriate schedule of reinforcement may increase the likelihood of learning a new response (i.e., skill acquisition). For example, an otherwise effective educational intervention may have limited effect if the consequence does not occur often enough, with sufficient immediacy, or with sufficient magnitude or quality. The design of interventions to reduce inappropriate behaviors and increase appropriate alternative behaviors should include

careful planning of appropriate schedules of reinforcement to result in desirable short-term and long-term effects.

Historical Background

Much of the earliest work on schedules of reinforcement is credited to the work of B. F. Skinner (Ferster and Skinner 1957; Green et al. 1959; Skinner 1953). Ferster and Skinner conducted extensive research on the effects of varying the relation between a particular response or pattern of responses and access to reinforcing consequences (e.g., food). Their research showed that the several parameters consistently effected patterns of responses, even when overall amount of reinforcement accessed was similar across conditions. For example, they found that reinforcement could be arranged to result in relatively fast or slow responding with or without pauses after reinforcement depending on how food deliveries were arranged. Research on schedules of reinforcement has continued to show reliable relations between schedules of reinforcement and particular patterns of behavior in both animals and humans.

Current Knowledge

Simple Versus Complex Schedules

Schedules of reinforcement may be separated into two broad categories, simple schedules and complex schedules. Simple schedules arrange a single contingency, such as reinforcement for every fifth response. In a complex schedule of reinforcement, more than one schedule is arranged and more than one schedule may be in effect at a given time. It is likely that real-life situations are associated with many schedules of reinforcement impacting an individual's behavior at any given time.

Simple Schedules

There are two general types of simple schedules of reinforcement, continuous reinforcement schedules and intermittent reinforcement schedules. In a continuous reinforcement schedule (CRF), each occurrence of a response is reinforced. An

example of a CRF schedule is the child's parent praises the child every time a child correctly places a puzzle piece. In contrast to CRF reinforcement schedules, intermittent reinforcement schedules arrange a reinforcer for some but not all occurrences of the target behavior. To use the puzzle example again, an intermittent schedule of reinforcement would mean the child's parent providing praise after some but not all correct placements of puzzle pieces.

Recommendations often include using CRF schedules when beginning a new intervention, such as to increase or improve communication (Cooper et al. 2007). The rationale for using a CRF schedule during the initial phases of teaching has to do with increasing the likelihood the learner will contact the contingency between the target behavior and a reinforcing consequence. By increasing the probability of this behavior-consequence relation, the behavior may be more likely to increase (i.e., be reinforced). After the behavior begins to increase, it is often recommended that the schedule be shifted from a continuous schedule to an intermittent schedule.

Intermittent reinforcement schedules can be based on either a *ratio* or *interval* schedule. In a ratio schedule, reinforcement occurs after a specified number of behaviors have occurred (e.g., reinforcement will occur after 5 sit-ups are completed). In an interval schedule, reinforcement occurs after the first response after a specified time period passes (e.g., the first request that occurs after 30 min have passed will be reinforced).

In addition to the ratio or interval schedule arrangement, intermittent schedules will also be either *fixed* or *variable*. In a fixed schedule, the criterion for reinforcement remains exactly the same for each opportunity (e.g., reinforcement will occur after every fifth sit-up). However, in a variable schedule, the specific criterion for reinforcement will vary for each opportunity and the schedule is usually expressed as the mean (e.g., reinforcement may occur after anywhere between 2 sit-ups and 10 sit-ups but will occur after a mean of 5 sit-ups).

Taken in combination, interval schedules are often described as fixed ratio (FR), fixed interval

(FI), variable ratio (VR), or variable interval (VI) schedules. Each schedule, the associated pattern of responding, and examples will be presented below.

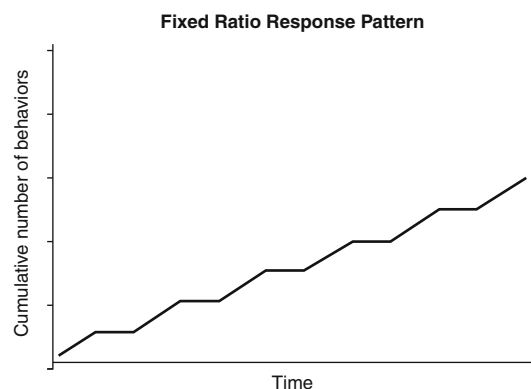
Fixed Ratio (FR) Schedules

Fixed ratio schedules of reinforcement specify that reinforcement will occur after a constant number of occurrences of the target behavior. For example, if the target behavior is correctly spelling one's name and an FR3 schedule is used, then the reinforcer would be presented after every third correctly spelled name.

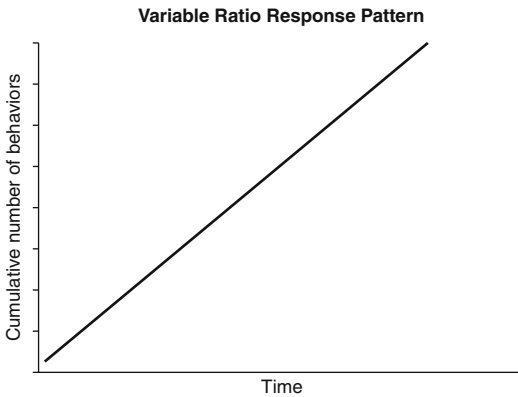
Researchers have shown that FR schedules maintain a relatively high rate of responding. However, FR schedules are also associated with a post-reinforcement pause: After accessing a reinforcer, there is often a pause before the person again begins to engage in the target behavior (see Fig. 1).

Variable Ratio (VR) Schedules

Variable ratio schedules of reinforcement specify the mean number of responses that will occur to access reinforcement. Thus, across subsequent reinforcer deliveries, sometimes the number of target behaviors will be greater or less than the mean. To again use the example of correct name spelling, the behavior could be reinforced on a VR 3 schedule. Under this schedule, the reinforcer



Schedule of Reinforcement, Fig. 1 Illustration of characteristic pattern of responding under a FR schedule, note the stable rate of responding (indicated by the constant increasing slope) and the post-reinforcement pause (indicated by the flat sections of the data path)



Schedule of Reinforcement, Fig. 2 Illustration of characteristic pattern of responding under a VR schedule, note the stable and relatively high rate of responding and no post-reinforcement pause; also note rate of responding is generally higher in a VR schedule than a VI schedule

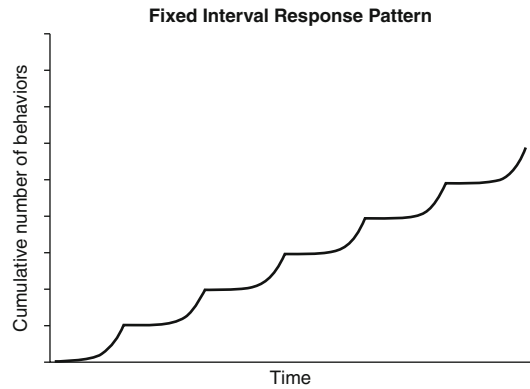
might be delivered after 1, 5, 2, and 4 correct instances of name writing, the mean of which is 3.

Researchers have consistently found that VR schedules of reinforcement result in a relatively high rate of responding. Variable ratio schedules do not result in post-reinforcement pauses, which is in contrast to FR schedules (see Fig. 2).

Fixed Interval (FI) Schedules

Fixed interval schedules of reinforcement specify reinforcement will be delivered for the first response after a predetermined amount of time has elapsed. Reinforcement on a FI schedule is still contingent on the target behavior, so while the fixed interval may be 5 min, reinforcement will not be delivered until after the target behavior occurs. For example, on a FI 5-min schedule, reinforcement for requests for a break may not be granted until after 5 min have passed, so a request at 4 min would not be reinforced. However, if the person requests a break after another 4 min have passed (i.e., a total of 8 min have elapsed), then the request would be reinforced.

Researchers have often found that FI schedules of reinforcement result in a characteristic pattern. Immediately following reinforcement, there is usually period of time with no responding (a pause similar that which is seen in FR schedules); the behavior then begins to occur at a low to moderate rate before achieving a high rate just

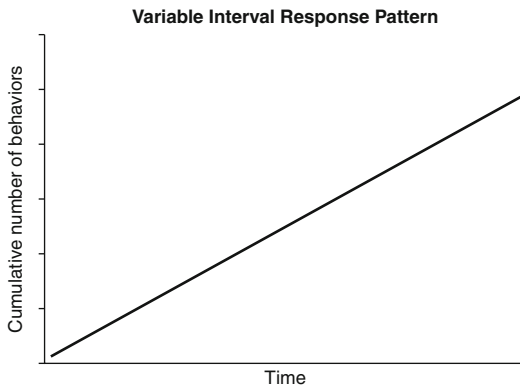


Schedule of Reinforcement, Fig. 3 Illustration of characteristic pattern of responding under a FI schedule, note the FI scallop comprised of increasing rate of responding leading to high rate responding (indicated by the progressively increasing slope) and the post-reinforcement pause (indicated by the flat sections of the data path following the steep/high-rate responding)

before the interval ends. This pattern of slow responding at the beginning of the interval and fast responding at the end has been called the FI scallop due to the unique shape when performance is graphed on a cumulative record (see Fig. 3). While this pattern of responding has been consistently found with research using animal subjects, the FI scallop pattern of responding with human participants has been less reliable (See Chance 2009).

Variable Interval (VI) Schedules

Variable interval schedules of reinforcement arrange reinforcement for the first behavior that occurs after a certain amount of time has passed. However, the length of each reinforcement interval may differ from the preceding and subsequent intervals. VI schedules are arranged such that individual intervals will vary around a mean interval. For example, a VI 10-min reinforcement schedule could be selected for the behavior of requesting a break. Under this schedule, reinforcement might be delivered for the first response after 6 min for one interval and after 15 min for a different interval. Over subsequent intervals, reinforcement would become available after 10 min had passed, on average. Again, it is important to remember that reinforcement does not occur after



Schedule of Reinforcement, Fig. 4 Illustration of characteristic pattern of responding under a VI schedule, note the stable and moderate rate of responding (as compared to the VR schedule) and no post-reinforcement pause

the interval time passes; instead, reinforcement occurs following the first target behavior observed after the interval time has passed.

Researchers have consistently found that VI schedules of reinforcement result in moderate rate behavior. In addition, the VI schedule is not associated with a post-reinforcement pause (see Fig. 4).

Additional Simple Schedules

Differential Reinforcement

Several schedules of reinforcement have been considered differential reinforcement schedules. These schedules of reinforcement are designed to increase a particular behavior relative to other behaviors or increase the occurrence of a particular pattern of behavior. Differential reinforcement schedules include differential reinforcement of other behavior, differential reinforcement of incompatible behavior, differential reinforcement of alternative behavior, differential reinforcement of high-rate behavior, and differential reinforcement of low-rate behavior.

Differential Reinforcement of Other Behavior (DRO)

Differential reinforcement of other behavior schedules arrange reinforcement for the

nonoccurrence of a particular target behavior. DRO schedules are often arranged using a time-based criterion for reinforcement, for example, a child might be reinforced if they have not engaged in disruptive behavior for 10 min. In this example, the goal of the DRO schedule is to increase the occurrence of other behaviors (i.e., any behavior that is not the disruptive behavior).

Differential Reinforcement of Incompatible (DRI) and Alternative Behavior (DRA)

Differential reinforcement of incompatible behavior schedules arrange reinforcement for a target behavior that is incompatible with another behavior (the second behavior is often an inappropriate behavior). A DRI schedule, for example, could arrange reinforcement for having hands folded on one's lap. In this example, placing folded hands on one's lap is incompatible with hitting peers and may therefore contribute to reducing the occurrence of hitting peers and increasing hand folding. In other words, folding hands and hitting others cannot occur at the same time.

Similar to DRI, differential reinforcement of alternative behavior arranges reinforcement for a particular target behavior and is often designed to help reduce an inappropriate behavior. In contrast to DRI, the reinforced behavior in a DRA schedule is not incompatible with the other behavior, so the two behaviors could, in theory, occur at the same time. However, the behavior targeted for reinforcement in the DRA schedule can be selected, in part, because it is unlikely to occur at the same time as the behavior targeted for reduction. A DRA could be arranged to increase a child's hand-raising as part of an intervention to reduce hitting peers. In this case, it is possible that the child could hit a peer with one hand while raising other hand, but this may be unlikely.

Functional communication training (FCT) is a special case of DRA. In FCT, an appropriate, communicative response is reinforced as part of an intervention to reduce the occurrence of an inappropriate response (see Carr and Durand 1985 for early research on FCT). In the case of FCT, the appropriate response is a communicative

response that accesses the same reinforcer as the inappropriate behavior. For example, a child's hitting might be maintained by removal of work, so FCT could be used to reinforce asking for a break from work.

Differential Reinforcement of Low-Rate Behavior (DRL)

Differential reinforcement of low-rate behavior arranges reinforcement for behavior occurring below a specific rate criterion. The goal of a DRL schedule is to reduce the rate of the target behavior without completely eliminating it. DRL might be used, for example, to reduce the rate of repeating requests to play with a particular toy. The DRL schedule could arrange reinforcement for a request only when a sufficient amount of time passed since the previous request. There are several ways in which the rate of behavior can be calculated, but the outcome is similar regardless of the method used to calculate the rate, and reinforcement only occurs when behavior occurs at or below a target rate.

Differential Reinforcement of High-Rate Behavior (DRH)

Differential reinforcement of high-rate behavior arranges reinforcement for behavior occurring above a specific rate criterion. The goal of DRH schedules is to increase the rate of a particular behavior. DRH could be used to increase the rate of behaviors such as correctly typed words per minute. In this example, reinforcement could be arranged only for correct typing that occurs above the criterion level for words per minute.

Time-Based Simple Schedules

While schedules of reinforcement are typically based on the occurrence of target behavior, they may also be used to arrange reinforcement at particular times, regardless of behavior. In time-based schedules, a stimulus or event with demonstrated reinforcing properties is delivered at specific times whether or not the target behavior has occurred. These time-based schedules are

sometimes called *noncontingent reinforcement* (NCR) because the consequence is not contingent on a particular response.

Time-based schedules can be arranged as either fixed-time or variable-time schedules. In a fixed-time (FT) schedule, the reinforcer is delivered at a specific time, such as every 30 min (e.g., FT 30-min schedule). Reinforcers can also be arranged on a variable-time (VR) schedule, such that the reinforcer is delivered after differing amounts of time, varying around an average. For example, reinforcement could occur after 10 min, 15 min, and 20 min for an average of 15 min (which would be a VR 15-min schedule).

Time-based reinforcement schedules have been used in interventions to reduce inappropriate behavior. Researchers have shown that when reinforcers are delivered every so often (i.e., on a FT or VT schedule), the target behavior may decrease (e.g., Vollmer et al. 1993). This reduced level of inappropriate behavior has often been attributed to a decrease in motivation for the reinforcer for the target behavior. For example, if a child engaged in hitting because it has resulted in attention in the past (i.e., attention is the reinforcer), using a VT schedule, an adult might provide attention every 5 min on average. With the VT schedule in place, the child may receive sufficient access to adult attention and therefore not engage in hitting others.

Complex Schedules

There are a variety of ways in which several schedules of reinforcement can be combined. When two or more simple schedules are combined, the resulting schedule is called a *complex schedules*. Complex schedules can include two simple schedules operating simultaneously, called a concurrent schedule. Complex schedules can also include two or more simple schedules that occur in a particular order, such as multiple schedules or mixed schedules. Furthermore, complex schedules can include a combination of schedules occurring at the same time at some times and in a particular order at other times (e.g., concurrent chains schedules). These complex

schedules can be analogous to common situations in which a person must choose between two or more behaviors and reinforcement contingencies.

Future Directions

Research on schedules of reinforcement can still be seen today, though it may not be discussed as such. An important outcome for individuals with ASDs is increasing and maintaining appropriate behaviors while decreasing and eliminating inappropriate behaviors. Researchers and practitioners should continue to look at the relation between reinforcement and acquisition of appropriate behaviors (e.g., social skills, academic behaviors, personal care) with a particular focus on potential relations between reinforcement schedules during learning and extent to which learned behaviors maintain when some or all supports are removed. Recent research in behavioral momentum (see Nevin and Grace 2000 for a review of behavioral momentum) has illustrated that overall reinforcement in a context may play an important role in maintenance of behavior (i.e., behavioral persistence). Other researchers have used progressive ratio schedules to examine maintenance of behavior (see Roane 2008 for a discussion). Ultimately, much of the success of individuals with autism will be based on their acquisition of appropriate behaviors, and schedules of reinforcement play a key role in both the speed at which behaviors may be acquired and the extent to which the learning will be demonstrated independently after interventions are removed or reduced.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavior Analysis](#)
- [Negative Reinforcement](#)
- [Positive Reinforcement](#)
- [Reinforcement](#)

References and Reading

- Carr, E. G., & Durand, V. M. (1985). Reducing behavior problems through functional communication training. *Journal of Applied Behavior Analysis*, 18, 111–126.
- Chance, P. (2009). *Learning and behavior: Active learning* (6th ed.). Belmont: Wadsworth.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Prentice Hall.
- Ferster, C. B., & Skinner, B. F. (1957). *Schedules of reinforcement*. New York: Appleton.
- Green, E. J., Sanders, R. M., & Squire, R. W., Jr. (1959). Schedules of reinforcement and discrimination learning. *Journal of the Experimental Analysis of Behavior*, 2, 293–299.
- Nevin, J. A., & Grace, R. C. (2000). Behavioral momentum and the law of effect. *Behavioral and Brain Sciences*, 23, 73–130.
- Roane, H. S. (2008). On the applied use of progressive-ratio schedules of reinforcement. *Journal of Applied Behavior Analysis*, 41, 155–161.
- Skinner, B. F. (1953). *Science and human behavior*. New York: The Free Press.
- Vollmer, T. R., Iwata, B. A., Zarcone, J. R., Smith, R. G., & Mazaleski, J. L. (1993). The role of attention in the treatment of attention-maintained self-injurious behavior: Noncontingent reinforcement and differential reinforcement of other behavior. *Journal of Applied Behavior Analysis*, 26, 9–21.

Schedules

- [Daily Routines](#)

Schizophrenia

- [Psychotic Disorder](#)

Scholarships

- [FAFSA](#)

Scholz's Disease

- [Metachromatic Leukodystrophy](#)

School Harassment

► Bullying

National Association of School Psychologists. http://www.nasponline.org/about_sp/whatis.aspx
 School Psychology. (n.d.). School psychology – Division 16 of APA [Power Point Slides]. Retrieved from <http://www.apa.org/ed/graduate/specialize/school.aspx>

School Psychologist

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School to Work Transition Process

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Definition

School psychology is the specialization of the professional practice of psychology that is concerned with children, youth, and families and the schooling process. The field of school psychology combines a unique knowledge base with that of related fields including clinical and educational psychology in order to focus on the individual study of children's learning and adjustment primarily in educational settings.

School psychologists primarily administer psychological and educational tests and conduct observations and interviews in order to better understand areas of concern and abilities that may be present for a student. School psychologists also consult with parents and educators and develop behavioral and teaching-based strategies to improve academic and socioemotional outcomes and support students with learning and behavioral disabilities.

Definition

'Transition' refers to a change in status from behaving primarily as a student to assuming emergent adult roles in the community. These roles include employment, participation in post-secondary education, maintaining a home, being appropriately involved in the community, and experiencing satisfying personal and social relationships. (Council for Exceptional Children's Division of Career Development and Transition 1994)

The transition process from school to adulthood, specifically employment, involves the application of educational experiences into successful employment. The educational experiences that are applied in adulthood involve more than employment: both community living and experiencing personal and social relationships impact successful quality of life as an adult.

See Also

- Educational Psychology
- Educational Testing

References and Reading

International School Psychology Association. <http://www.ispaweb.org/>
 Lee, S. W. (2005). *Encyclopedia of school psychology*. Thousand Oaks: SAGE.

Historical Background

If one looks back 20 years, students with autism were being educated either in generic disability classrooms or in residential institutions. There were no discussions regarding adults with autism, as many were institutionalized or living at home with their aging parents.

As the awareness of the unique needs of individuals with autism has increased, evidence-based practices have been established and are being integrated into the public school environment, planning for the school to work transition can now be achieved.

Current Knowledge

In 1975, PL 94:142 authorized the Education of All Handicapped Children Act. This Act mandated a free and appropriate public education for all children regardless of disability. In 1990, the Individuals with Disabilities Education Act, PL 101:475 not only reauthorized the PL-94:142 but extended the requirement for education of children with disabilities and required transition services for children with disabilities. The amendment specifically outlined that special education needed to prepare students for employment and independent living.

Even with this law and clear attempts made by educators to offer educational services that impact success as an adult, two-thirds of Americans with disabilities between the ages of 16 and 64 are not employed (Wehman 2001). Specifically, many individuals with autism spectrum disorders have completed high school and/or college but are unable to make the transition to employment. For individuals with autism spectrum disorders, particular reasons for this failure may include difficulty with social competencies, communication deficits, behavioral issues, and potential comorbid mental health issues.

Difficulty with social competencies is core to the diagnosis of autism spectrum disorders. Individuals with autism spectrum disorders often have difficulty understanding the nuances of typical social behavior and as such struggle with interacting with others. Individuals with autism spectrum disorders also have difficulty with problem solving and coping, which can all translate to difficulty in the workplace. Teachers and vocational rehabilitation specialists often focus on job competencies rather than social competencies. For individuals with autism spectrum disorders, the job competencies may be secondary and the social competencies need to be a primary focus during the school age years.

In addition to challenges with social competencies, individuals with autism spectrum disorders often struggle with communication. They may have difficulty initiating or maintaining a conversation, often will not use facial expressions, body language, or other nonverbal

behaviors to complement their social communication interaction. Effective communication is paramount to success in a job placement, as that is what allows for clarification of job requirements, performance, and working effectively with coworkers.

Another area that may negatively impact success in a job placement is behavioral issues. While behavioral issues vary, many individuals with autism spectrum disorders may insist on sameness, regardless of the job at hand, and intense preoccupation with topics or subjects may also interfere with job requirements, as both lead to off-task behavior. Supporting the individual with autism spectrum disorder to self-monitor and control behavioral issues, as well as educating the community to increase understanding and tolerance of these behaviors, is necessary for successful employment of individuals with autism spectrum disorders.

Individuals with autism spectrum disorders may also exhibit comorbid mental health issues. Specifically related to autism in adolescents, there is an increased vulnerability to anxiety disorders and depression (Wehman et al. 2009). It is essential that individuals with autism spectrum disorders are afforded the appropriate mental health support services to treat such disorders.

Research points to the fact that individuals with autism spectrum disorders can work and obtain a quality of life, but they need support to do so (Wehman et al. 2009). A significant factor to this success will be effective transition planning during the school years. Ideally, transition planning should begin in early intervention, when a young child with autism spectrum disorder begins to be taught important skills that impact social skills, behavior, communication, and independence. These educational themes should be featured throughout a young student's Individualized Education Plan (IEP).

The IEP is the legal document that is a guideline for all transitions. Transition IEPs should include assessments, goals and objectives, and intervention strategies that target priorities for skills that are necessary for success as an adult. The transition IEP team is typically made up of the student, parents, special education teacher,

general education teacher, community agency personnel, potential employers, and/or vocational rehabilitation counselors. This team is charged with assessing and developing goals and objectives that will meet transition to employment goals as well as effective implementation strategies to do so.

Prior to developing IEP goals and objectives, there needs to be some level of career awareness for the student with autism spectrum disorder. Typically, career awareness begins in the elementary school years when children are exposed to a variety of “community helpers.” In the middle school years, students are exposed to career awareness through the content of some academic subjects such as science or English through which they learn about various careers, for example, scientists or authors. In high school, there is a more specific focus on career awareness as focus is placed on preparation for post-secondary education or vocational and technical training. Individuals with autism spectrum disorders should receive some type of vocational assessment through which potential jobs can be matched to the individual’s strengths and interests. These students should also be exposed to job sampling, which allows them to try out a variety of jobs for brief periods of time to actually experience that job. Another avenue for vocational assessment is performance sampling, which is simulated work done in the classroom. This type of assessment results in a more cursory review for job matching.

For students with autism spectrum disorders, there clearly needs to be goals and objectives in the IEP that focus on social competencies, adaptive skills, and work habits. In the area of social competencies, goals and objectives must effectively address the challenges of social interactions, problem solving, coping, and self-management. Adaptive skills should focus on grooming, mobility, health and safety, and money management. Work habits should be addressed through goals and objectives that highlight productivity, attendance, and accuracy in a job.

Once appropriate goals and objectives have been developed and accepted by the IEP team, focus needs to be placed on successful

achievement of these goals and objectives. When preparing a student with autism spectrum disorders for employment, it will be necessary for knowledgeable staff to oversee the implementation of goals. The school environment must either have personnel with expertise in autism spectrum disorders or have access to this expertise through consultation. Successful achievement of goals and objectives will only be reached if strategies meet the unique needs of students with autism spectrum disorders. In addition, for successful achievement of transitional IEP goals, there clearly has to be practice of skills beyond the self-contained classroom.

Educational inclusion allows for an individual with disabilities to be appropriately supported to learn in a general education environment. In inclusion, the student with autism spectrum disorder can gain general knowledge and improve their social interactions through peer modeling and practice. Inclusion provides students with autism spectrum disorder the opportunity to be members of the school community and to improve their independence. Achievement of IEP goals and objectives will be partly based on the appropriate balance of specialized direct instruction and inclusion opportunities.

Alongside educational inclusion as a critical component of the school to work transition process is community-based instruction. Community-based instruction is the systematic teaching of students with autism spectrum disorders within the natural community setting. Community-based instruction enhances the transition of a student with autism spectrum disorder to employment, as skills are taught and reinforced in the actual environment where the student will live and work as an adult (Wehman et al. 2009).

When IEP goals and objectives are implemented through community-based instruction, skills are taught where they are expected to occur. This also allows for immediate generalization into the natural environment. Community-based instruction can be implemented at a community college, work site, stores and restaurants, and recreational facilities. When community-based instruction is not possible, it is important that places outside of the classroom in the school

environment be used to foster this natural setting. Places such as the cafeteria, library, office, or school grounds can be used for natural environment instruction.

When community-based instruction is focused specifically on employment, it is essential that the IEP team assess the local community in regard to the availability of jobs. The IEP team will also have to match the job to student interests and capabilities and determine whether instruction needs to begin before the job placement or if on-the-job training can be implemented from the beginning.

There are supports beyond the IEP that are positive factors leading to successful employment for young adults with autism spectrum disorders. It is important that, as students with autism spectrum disorders approach their 21st birthday and enter a world with no entitlements, community supports are secured. Vocational rehabilitation services are available in all states in the USA. It is important that a relationship is developed with vocational rehabilitation services in order to prepare for the transition from school supports to post-21 supports. This may specifically be noted in transitioning from school staff to a job coach who can be available for the individual with autism spectrum disorder once they have graduated and are at a specific job placement.

Another important support that needs to begin prior to the 21st birthday of an individual with autism spectrum disorder and should be part of the transition from school to work is the development of appropriate supports on the job site. The good news in 2011 is that the general public is much more familiar with autism spectrum disorders than it was in 2000. As part of this general public awareness, coworkers, supervisors, and employers have the potential to have some knowledge base regarding autism spectrum disorders before the student is employed. It is important that this awareness be expanded upon and include specific information about a particular student with autism spectrum disorder so that appropriate supports can be provided by these coworkers, supervisors, and employers. Not only will this continue to increase understanding and tolerance

for the unique characteristics of autism spectrum disorders, but it will also allow a coworker or supervisor to provide support that potentially a job coach would have provided. Job coaches need to be faded, and as such, if the natural supports in the work site can support the individual with autism spectrum disorder, the more successful they will be.

Future Directions

Increased awareness of the supports that individuals with autism spectrum disorders need to achieve successful employment can be viewed as a positive prognostic indicator for the future. It is evident that the payoff of appropriate education of students with autism spectrum disorders will be a new generation of successfully employed individuals. The pressure must be felt during an individual with autism spectrum disorder's entitlement years of education to effectively prepare that individual for adulthood.

See Also

- [Individualized Transition Plan \(ITP\)](#)
- [Transition Planning](#)
- [Transitional Living](#)

References and Reading

- Altwood, T. (2006). *The complete guide to Asperger's syndrome*. London: Jessica Kingsley.
- Individuals with Disabilities Education Act (IDEA) of 1990. PL101-476, 20. USC-SS 1400 et seq.
- Luce, S., & Dyer, K. (1995). Providing effective transitional programming to individuals with autism. *Behavior Disorders*, 21(1), 36–52.
- Persson, B. (2000). Brief report: A longitudinal study of quality of life and independence among adult men with autism. *Journal of Autism and Developmental Disorders*, 30(1), 61–66.
- Rusch, F. R., & Minch, K. E. (1988). Identification of co-worker involvement in supported employment: A review and analysis. *Research in Developmental Disabilities*, 9(3), 247–254.
- Schaller, J., & Yang, N. K. (2005). Competitive employment for people with autism: Correlates of successful

- closure of competitive and supportive employment. *Rehabilitation Counseling Bulletin*, 49(1), 4–6.
- Volkmar, F., Paul, R., Klin, A., & Cohen, D. (Eds.). (2006). *Handbook of autism and pervasive developmental disorders: Diagnosis, development, neurobiology and behavior* (Vol. 1, 3rd ed.). Hoboken: Wiley.
- Wehman, P. (2001). Postsecondary education and disability. *Journal of Vocational Rehabilitation*, 16(3/4), 141, 1.
- Wehman, P. (2006). *Life beyond the classroom: Transition strategies for young people with disabilities* (4th ed.). Baltimore: Paul H Brookes.
- Wehman, P., Smith, M. D., & Schall, C. (2009). *Autism and the transition to adulthood: Success beyond the classroom*. Baltimore: Paul H Brookes.
- Weiner, J. S., & Zivolich, S. (2003). A longitudinal report for three employees in a training consultant model of natural support. *Journal of Vocational Rehabilitation*, 18, 199–202.

School-Aged Children

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Definition

The school-aged period encompasses the years between early childhood and adolescence, during which many children step out into the world outside the family for the first time. The characteristic symptoms and clinical signs of autism spectrum disorders are typically detected prior to the school-aged period, during early childhood. The diagnostic criteria for autistic disorder listed in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR 2000) specify that the onset of symptoms occurs prior to the age of 3 years (DSM-IV-TR 2000). There is significant focus on the identification of children on the autism spectrum during the early childhood years. However, not all children with autism spectrum disorders are identified prior to school enrollment. Moreover, the school-aged years present children with autism spectrum disorders with ongoing challenges. The presence of an autism spectrum disorder affects the trajectory and completion of

the key developmental tasks of the school-aged years and often requires specialized attention in a number of domains, including mental health, medical, and educational settings.

Historical Background

The school-aged years were once conceptualized by Freud and others as a period of latency, during which sexual drives are suppressed and energies are diverted to the skill mastery challenges of school and peer relationships. Psychologist Erik Erikson identified the actualization of “Industry versus Inferiority” as the critical developmental accomplishment of the school-aged years. Industry refers to a child’s increasing competence in a variety of realms that is expected over the course of childhood. Though brain size does not change significantly after the age of 7, the school-aged period is a time of continued maturation of brain processes. Typically developing children become capable of higher level cognitive tasks. They become more adept at mathematics and concrete reasoning, they begin to develop a fund of knowledge about the world, they learn to read and to write, and they begin to develop moral reasoning as well. Their motor skills become more precise and they are able to complete more intricate motor tasks. Social development is marked by the growing importance of peers and the capacity to develop friendships. School-aged children are eventually able to follow rules and to engage in team-based play. The completion of developmental achievements in the cognitive, motor, and social domains allows for a greater independence from parents. If these developmental tasks are not completed, feelings of inferiority and self-doubt may emerge (Combrink-Graham and Fox 2007).

Autism spectrum disorders characteristically impact all the developmental domains. Significant impairments in social interactions and language development are pathognomonic of classic autistic disorder and occur with varying severity in other spectrum disorders. Intellectual disabilities are also common in children with autism spectrum disorders as are unusual body movements and subtle motor abnormalities such as toe walking

and poor coordination. Children with spectrum disorders may require additional supports and interventions to navigate the developmental tasks of childhood (DSM IV-TR 2000).

In the context of the civil rights movements of the mid-twentieth-century United States, advocacy for more supports and services for children with developmental disabilities intensified, leading to federal legislation supportive of these aims. In particular, the Individuals with Disabilities Education Act (IDEA) of 1975 is a landmark piece of legislation that has had major implications for the educational opportunities available to children with autism. Prior to the enactment of this legislation, many children with developmental disabilities were excluded from the school system. IDEA stipulates that all children should have access to a free public education that is appropriate to their needs. IDEA was amended in 2004 to promote the use of evidence-based strategies to improve outcomes. IDEA entitles children to an individualized educational plan (IEP) and promotes placement in the least restrictive educational environment possible to meet a child's needs (Bryan 2010; Steedman 2007).

Current Knowledge

The particular psychiatric, medical, and educational needs of children with autism spectrum disorders often become evident during the school-aged years.

Mood and Anxiety Disorders in School-Aged Children on the Autism Spectrum

It may be a challenge to detect mood disorders such as depression in children with impaired language skills. However, there is some research that suggests that depression may occur frequently in autistic persons (Skokauskas and Gallagher 2010). Comorbid anxiety is common in children with autism spectrum disorders. Despite the presence of social impairment, some children are aware of and distressed by difficulties connecting with others. Anxiety can lead to avoidance behaviors and further isolation from peers and

community. There is wide variation in the estimates of the prevalence of anxiety disorders in children on the autism spectrum. As many as 40–50% of children in this population may meet criteria for anxiety disorders, including generalized anxiety disorder, separation anxiety, and simple phobias. It can be difficult to determine whether symptomatology represents a separate and coexisting anxiety disorder rather than a manifestation of the autistic disorder itself. Higher functioning children with autism spectrum disorders may be more likely to experience anxiety. There is some evidence that cognitive behavioral therapy and medications (such as the selective serotonin reuptake inhibitors) may be helpful for the treatment of anxiety disorders in children on the autistic spectrum (White et al. 2009).

Behavioral and Social Problems

Aggression and other self-injurious behaviors are also common in children with autism spectrum disorders and can be particularly disruptive in school environments. These behaviors can also pose a safety risk to the children, peers, families, and other caregivers. Aggression is often a target of psychopharmacologic treatment. The atypical antipsychotic medication risperidone is approved by the Food and Drug Administration for the treatment of aggression in children with autism. Other frequently used medications include other atypical antipsychotic medications, alpha adrenergic agonists such as clonidine, mood stabilizers, and stimulants (Parikh et al. 2008).

Since hyperactivity and inattention can be associated symptoms of autism, the DSM-IV-TR stipulates that a separate diagnosis of attention deficit hyperactivity disorder (ADHD) should not be given if autistic disorder is present. Attentional symptoms are nonetheless frequent targets of psychopharmacologic treatments, and there is some evidence that children on the autism spectrum respond differently to medications typically used for ADHD (Reiersen and Todd 2008). Children with spectrum disorders may also have deficits in executive functions such as planning and mental flexibility, though they tend to perform comparably to controls on tests of inhibition (Hill 2004).

Social skills do not improve automatically with age in children with autism spectrum disorders. In fact, impairment may become more pronounced over time with the increasing social demands of the school-aged period. Children may become more acutely aware of their social limitations, resulting in feelings of loneliness and distress. Examples of problems with social interactions exhibited by children with spectrum disorders include fixation on restricted interests and inability to read the interest of peers, difficulty with reciprocal conversation, and abnormalities of expressive language. Social skills training (SST) programs may be available to school-aged children with autism. More research is needed to determine the most effective methods for social skills building (Williams White and Keonig 2007).

Medical Disorders

In addition to developmental, behavioral, and mood symptoms, children with autism and autism spectrum disorders appear to be at increased risk for a number of medical problems that can occur during the school-aged years. Diagnosis of these medical issues is often a challenge in this population, as children may present with symptoms that are not typical. More time and effort may be needed to arrive at an accurate diagnosis, particularly for children who are non-verbal or who have significant behavioral disruptions. Common medical disorders seen in children with autism spectrum disorders include seizures, sleep disorders, and gastrointestinal disorders (Goldson and Bauman 2007). As many as one third of children with autism will have a seizure at one point in their lives. Children with co-occurring intellectual disabilities appear to be more likely to have seizures. Though the incidence of seizures is highest in early childhood and in adolescence, seizures can occur at any time during childhood (Volkmar and Nelson 1990). While a sizable minority of typically developing children will experience sleep problems, two thirds of children on the autism spectrum have sleep difficulties, including insomnia, hypersomnia, enuresis, and nightmares. Poor

sleep has been linked to behavioral difficulties and poor school performance (Richdale and Schreck 2009). Gastrointestinal disorders – such as constipation, diarrhea, and abdominal pain – occur frequently in the general child population; the relative prevalence of these issues in children with autism spectrum disorders has not been clearly established. Gastrointestinal problems may present with behavioral and sleep disturbances in school-aged children with autism (American Academy of Pediatrics [AAP] 2010).

Educational Needs and Accommodations

The educational environment presents a number of opportunities and challenges for children with autism spectrum disorders. Educational programs can provide the foundations for continued education, the development of life and independent living skills, the cultivation of social skills, and employment. Collaboration between parents, teachers, school administrators, and medical and mental health providers is paramount, particularly when developing the individualized educational plan (IEP). The IEP should define specific goals for the academic year and identify a system of modifications and supports to help the child achieve those goals. There are a variety of educational approaches available; interventions should be targeted toward children's specific and individual needs. Evidence-based interventions in schools might include applied behavioral analysis, positive behavioral supports, and assistive devices and technology. The child should be placed in the least restrictive educational environment, and there has been a move toward the inclusion classroom, in which special education services are embedded in the regular classroom. Parents, teachers, and school administrators must work together to determine the appropriate balance of inclusion instruction and separate special education instruction for each child (Noland et al. 2007). Children with autism may process information differently and may benefit from the use of pictures and concrete teaching tools. They also frequently benefit from structured environments and clear expectations (Mukuria and Obiakor 2008).

Future Directions

Children with autism spectrum disorders often require a number of specialized services during the school-aged years in order to meet their medical, psychiatric, and educational needs. More data about appropriate interventions in these realms are needed. Care coordination between primary care and specialty providers may facilitate accurate diagnosis and effective treatment of medical conditions. Further efforts to clarify the prevalence and presenting symptoms of certain medical disorders may help inform future diagnostic and treatment guidelines for children with autism spectrum disorders. In particular, the relationship between autism and gastrointestinal and neurologic disorders is an area of active research. Many psychopharmacologic agents are commonly used to treat psychiatric symptoms in this population, but definitive evidence of benefit is not yet available for many medications. Management of long-term medication side effects such as obesity and metabolic derangements should also be a focus of clinical and research attention. More information about the impact of non-pharmacologic interventions such as social skills training would be valuable. There has been a growing emphasis on using scientifically based treatments in the classroom setting, and more data on the implementation and effectiveness of these interventions is also needed.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Atypical Antipsychotics](#)
- [Individual Education Plan](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Latency](#)

References and Reading

American Academy of Pediatrics. (2010). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125(Suppl), S1–S18.

- American Psychiatric Association. (2000). Pervasive developmental disorders. In *Diagnostic and statistical manual of mental disorders* (Rev. 4th ed.). Washington, DC: Author.
- Bryan, W. V. (2010). *Sociopolitical aspects of disabilities* (2nd ed.). Springfield: Charles C. Thomas.
- Combrink-Graham, L., & Fox, G. S. (2007). Development of school-age children, Chapter 3.1.3. In A. Martin & F. Volkmar (Eds.), *Lewis's child and adolescent psychiatry: A comprehensive textbook* (4th ed., pp. 267–278). Philadelphia: Lippincott Williams & Wilkins.
- Goldson, E., & Bauman, M. (2007). Medical health assessment and treatment issues in autism, Chapter 2. In R. L. Gabriels & D. E. Hill (Eds.), *Growing up with autism: Working with school age children and adolescents*. New York: The Guilford Press.
- Hill, E. L. (2004). Executive dysfunction in autism. *Trends in Cognitive Sciences*, 8(1), 26–32.
- Lopez, B., Hill, D., Shaw, S., & Gabriels, R. (2007). School consultation and interventions for middle school and high school students with autism, Chapter 12. In R. L. Gabriels & D. E. Hill (Eds.), *Growing up with autism: Working with school age children and adolescents*. New York: The Guilford Press.
- Mukuria, G. M., & Obiakor, F. E. (2008). Curriculum innovation to educate students with autism in general education. In A. F. Rotatori, F. E. Obiakor, & S. Burkhardt (Eds.), *Autism and developmental disabilities: Current practices and issues* (Advances in special education, Vol. 18, pp. 25–40). Bingley: Emerald Group.
- Noland, R., Cason, N., & Lincoln, A. (2007). Building a foundation for successful school transitions and educational placement, Chapter 10. In R. L. Gabriels & D. E. Hill (Eds.), *Growing up with autism: Working with school age children and adolescents*. New York: The Guilford Press.
- Parikh, M. S., Kolevzon, A., & Hollander, E. (2008). Psychopharmacology of aggression in children and adolescents with autism: A critical review of efficacy and tolerability. *Journal of Child and Adolescent Psychopharmacology*, 18(2), 157–178.
- Reiersen, A. M., & Todd, R. D. (2008). Co-occurrence of ADHD and autism spectrum disorders: Phenomenology and treatment. *Expert Review of Neurotherapeutics*, 8(4), 657–669.
- Richdale, A. L., & Schreck, K. A. (2009). Sleep problems in autism spectrum disorders: Prevalence, nature, & possible biopsychosocial aetiologies. *Sleep Medicine Reviews*, 13(6), 403–411.
- Skokauskas, N., & Gallagher, L. (2010). Psychosis, affective disorders and anxiety in autism spectrum disorder: Prevalence and nosological considerations. *Psychopathology*, 43(1), 8–16.
- Steedman, W. (2007). Advocating for services, Chapter 7. In R. L. Gabriels & D. E. Hill (Eds.), *Growing up with autism: Working with school age children and adolescents*. New York: The Guilford Press.

- Volkmar, F. R., & Nelson, D. S. (1990). Seizure disorders in autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 29(1), 1007–1008.
- White, S. W., Oswald, D., Ollendick, T., & Scahil, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229.
- Williams White, S., & Keonig, K. (2007). Social skills development in children with autism spectrum disorders: A review of the intervention research. *Journal of Autism and Developmental Disorders*, 37, 1858–1868.

School-Clinic Care Coordination for Youth with ASD

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Definition

School-clinic care coordination is the interprofessional communication and collaboration between providers in the educational (i.e., schools) and medical (i.e., primary/specialty care clinics) homes to promote streamlined and integrated care for individuals with special health care needs, such as autism spectrum disorder (ASD).

Current Knowledge

ASD is a complex and multifaceted neurodevelopmental disorder that often requires interprofessional care for behavioral, medical, psychiatric, and learning-related issues. For example, many children with ASD have co-occurring GI problems, neurological disorders (e.g., seizures), genetic syndromes, feeding and sleep issues, academic difficulties, mood disorders (e.g., anxiety, depression), disruptive behaviors (e.g., ADHD), and intellectual disability, among others. As a result,

many youth with ASD often receive care from providers (e.g., school nurses, school psychologists, primary care clinicians, developmental behavioral pediatricians) across school and community settings.

Many families with ASD report feeling overwhelmed and marginalized when attempting to coordinate the care their child receives from providers across multiple settings. This often occurs when providers from multiple disciplines do not discuss their service delivery plans with one another, leading to fragmentation or duplication of care. Streamlining services across these providers and settings is critical to increase the efficiency and quality of care as well as to promote family satisfaction and their experience of care (i.e., family centered care).

Streamlined services can be accomplished through care coordination whereby providers collaborate interprofessionally through information sharing and collaborative decision-making around unified approaches to evaluation and management that address behavioral, medical, psychiatric, and learning-related issues. For example, school-based academic and behavior performance data can be shared by school psychologists with a specialty ASD clinic in order to enhance diagnostic clarity and treatment planning. Similarly, a physician can share information with a school professional about a co-occurring medical condition and its potential impact on learning in order to inform educational programming and related supports. Because many youth with ASD are prescribed psychotropic medications for co-occurring mental health conditions (e.g., ADHD), school psychologists are often well positioned to undertake collaborative medication-related roles. For example, they can collect behavioral performance data as well as side effect checklists and share these data with prescribers (child-adolescent psychiatrists, developmental behavioral pediatricians, nurse practitioners, pediatricians) for dosage titration and progress monitoring purposes (pending parental consent for information sharing).

There are several barriers that prevent effective school-clinic collaboration among professionals. These include, but are not limited to, lack of knowledge of roles and functions of service providers in

other settings; differences between medical diagnosis eligibility criteria and special education criteria; difficulty with sharing information given Family Educational Rights and Privacy Act (FERPA) and Health Insurance Portability and Accountability Act (HIPAA) privacy restrictions; and provider time constraints. Providers are encouraged to engage in behaviors that decrease barriers and facilitate effective interprofessional collaboration, for example, understanding both HIPAA and FERPA laws and determining what steps and legal documents are required prior to information sharing; respecting, understanding, and having awareness of other service providers and their roles; valuing interprofessional collaboration and team-based care; engaging in shared decision-making; sharing and discussing relevant information; collaboratively determining a communication plan at the outset (e.g., when, about what, with what frequency, and via what medium); and ensuring support from caregivers and administrators.

To increase engagement in school-clinic care coordination for children with ASD, there will likely need to be changes to administrative support structures in schools and clinics/health systems that allow for shared decision-making and team-based care (e.g., HIPAA/FERPA compliant web-based portals for information sharing and collaboration). Changes to training structures in professional programs are also needed to better emphasize crosscutting skills associated with interprofessional collaboration and team-based care. It is strongly recommended that providers receive training specific to interprofessional care, both at the preservice and professional development levels.

autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 45(3), 636–644.

- Farmer, J. E., Clark, M. J., Mayfield, W. A., Cheak-Zamora, N., Marvin, A. R., Law, J. K., & Law, P. A. (2014). The relationship between the medical home and unmet needs for children with autism spectrum disorders. *Maternal and Child Health*, 18(3), 672–680.
- Golnik, A., Scal, P., Wey, A., & Gaillard, P. (2012). Autism-specific primary care medical home intervention. *Journal of Autism and Developmental Disorders*, 42(6), 1087–1093.
- Green, B. N., & Johnson, C. D. (2015). Interprofessional collaboration in research, education, and clinical practice: Working together for a better future. *Journal of Chiropractic Education*, 29(1), 1–10.
- Kogan, M. D., Strickland, B. B., Blumberg, S. J., Singh, G. K., Perrin, J. M., & van Dyck, P. C. (2008). A national profile of the health care experiences and family impact of autism spectrum disorder among children in the United States, 2005–2006. *Pediatrics*, 122(6), e1149–e1158.
- McClain, M. B., Shahidullah, J. D., Mezher, K. R., Havercamp, C. R., Benallie, K. J., & Schwartz, S. E. (2019). School-clinic care coordination for youth with ASD: A national survey of school psychologists. *Journal of Autism and Developmental Disorders*. Online first.
- Shahidullah, J. D., Azad, G. F., Mezher, K., McClain, M. B., & McIntyre, L. L. (2018). Linking the medical and educational home to support children with autism spectrum disorder: Practice recommendations. *Clinical Pediatrics*, 57, 1496–1505.
- Shahidullah, J. D., Forman, S. G., Palejwala, M. H., Chaudhuri, A., Pincus, L. E., Lee, E., Shafrir, R., & Barone, C. (2019). National survey of chief pediatric residents' attitudes, practices, and training in collaborating with schools. *Journal of Interprofessional Education and Practice*, 15, 82–87.
- Slusser, M., Garcia, L., Reed, C. R., & McGinnis, Q. (2018). *Foundations of interprofessional collaborative practice in healthcare*. St. Louis: Elsevier.

References and Reading

- Carbone, P. S., Behl, D. D., Azor, V., & Murphy, N. A. (2010). The medical home for children with autism spectrum: Parent and pediatrician perspectives. *Journal of Autism and Developmental Disorders*, 40, 317–324.
- Cheak-Zamora, N. C., & Farmer, J. E. (2015). The impact of the medical home on access to care for children with

School-Wide Positive Behavior (a) Interventions and Supports (SWPBIS)-for Schools K-12

- [Positive Behavioral Interventions and Supports \(PBIS\)](#)

School-Wide Positive Behavior Support (SWPBS)-for Schools K-12

► [Positive Behavioral Interventions and Supports \(PBIS\)](#)

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Major Appointments (Institution, Location, Dates)

- Professor: Department of Psychiatry, University of North Carolina, Chapel Hill, 1964–2005
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- Editor, *Journal of Autism and Developmental Disorders*, 1974–1998
- Editor, *Current Issues in Autism Series*, Plenum, 1983–1998

Major Honors and Awards

- 2006 American Psychological Foundation Gold Medal Award for Life Achievement in the Application of Psychology
- 2006 International Society for Autism Research Lifetime Achievement Award
- 2005 Autism Society of North Carolina Lifetime Achievement Award
- 1993 The North Carolina Award: the highest honor that is awarded citizens of NC
- 1985 American Psychological Association Distinguished Professional Contributions to Public Service
- 1985 O. Max Gardner Award from the UNC Board of Governors for great contributions to the welfare of the human race
- 1977 American Psychological Association Award for Distinguished Contributions to the advancement of knowledge and service
- 1972 American Psychiatric Association Gold Achievement Award (with Robert Reichler, M.D.)

Landmark Clinical, Scientific, and Professional Contributions

Dr. Schopler conducted some of the first research to use scientific methodology in the study of autism. He continued to promote the importance of quality research in the field of autism throughout his career, especially in his role as editor of the *Journal of Autism and Developmental Disorders*. He insisted that intervention strategies were founded upon sound research so that individuals with autism and their families could select effective interventions and avoid being manipulated by unsupported claims (Reichler and Schopler 1971; Schopler 1965, 1966, 2001).

Dr. Schopler was a lifelong advocate for incorporating the parent perspective in autism research and treatment. His research was central to the transition from seeing autism as psychogenic in nature (the refrigerator mother theory) to a scientifically based understanding that autism is

developmental in nature (Schopler 1971, Schopler and Reichler 1971).

Dr. Schopler along with Robert Reichler, M.D. directed the 5-year NIMH-funded Child Research Project beginning in 1967. This was a landmark study demonstrating that parents are a central component to the treatment of children with autism (Schopler and Reichler 1971). Results of the study became a model for parent-professional collaboration at a level that was unheard of at the time.

Dr. Schopler was one of the first researchers to document the positive response of children with autism to structured intervention approaches. His research led the field of autism away from unstructured psychodynamic interventions that were predominant at the time and toward interventions based on an understanding of the learning styles of individuals with autism (Schopler et al. 1971, 1994).

Dr. Schopler was the cofounder of Division TEACCH along with Robert Reichler, M.D. TEACCH (an acronym for Treatment and Education of Autistic and related Communication handicapped CHildren) was the first comprehensive statewide program for the treatment of individuals with autism and their families. Under his leadership, Division TEACCH provided accessible services to families in the state of North Carolina. TEACCH integrated provision of a range of services including assessment, parent training, and community-based school, vocational, and residential programs (Mesibov et al. 2005).

Dr. Schopler was the cocreator of the Childhood Autism Rating Scale (CARS) (Schopler et al. 1988), one of the first standardized diagnostic assessment instruments for autism. Since its original publication, the CARS has been a mainstay of diagnostic assessment. Dr. Schopler began his career with a commitment to evidence-based clinical practice and closed his career by continuing this pursuit, contributing to the development of the most recent version of the CARS, the CARS-2-HF (Schopler et al. 2010).

Dr. Schopler contributed to the development of the Psychoeducational Profile (PEP) (Schopler

and Reichler 1979) and the Adolescent and Adult Psychoeducational Profile (AAPEP) (Mesibov et al. 1988). These two assessment instruments were based on the premise that understanding an individual's pattern of strengths and weaknesses is essential to selecting successful intervention goals and strategies. The PEP is now in its third edition (Schopler et al. 2005), and the AAPEP has been updated as the TEACCH Transition Assessment Profile (Mesibov et al. 2007). Both instruments have been translated into several languages and are used by autism programs worldwide.

Throughout his career, Dr. Schopler was devoted to increasing the accessibility of services in North Carolina and around the world. He never tired in his efforts to support parents who have often been the driving force behind service development. His ability to develop collaborative relationships with parents and professionals has led to multiple replications of the TEACCH model that continue today (Schopler 2000).

Short Biography

With no doubt, Eric Schopler was an individual who had a lasting impact on the field of autism research and intervention. Dr. Schopler was the author of over 200 books and articles about autism. He was the cofounder of the internationally recognized autism treatment program, Division TEACCH. He was the recipient of numerous awards and honors, but above all else, he valued his collaborations with parents and their children with autism. This collaboration between parent and professional was the hallmark of his career. It set him apart from most of the respected professionals of his day who espoused a psychodynamic approach to autism. Dr. Ruth Christ Sullivan, parent and first president of the Autism Society of America commented on his uniqueness as a professional, "Here was a young professional mingling with parents as equals. At the time you could be chastised or ostracized for mingling with parents" (Sullivan 2005).

Dr. Schopler's sensitivity to the dangers of marginalizing groups began with his childhood in Germany. He was born in Furth, Germany, in 1927. In 1938, his parents, Ernst Schopler and Erna Oppenheimer Schopler, escaped Nazi persecution by emigrating to the United States with Eric and his siblings. A child at the time, Dr. Schopler did not recall direct abuse by the Nazi government, but he recalled friends and teachers who suddenly left or disappeared. Out of his struggle to understand what had happened to his family and all European Jews, a cognitive framework developed that made him ever watchful for abuses of power, particularly within members of his own field, psychology.

He observed just such an abuse with regard to the parents of children with autism and other severe disorders. As a social worker at the Bradley child psychiatric hospital in Providence, RI, Dr. Schopler observed how parents were blamed for their child's disorder. Dr. Schopler found this conclusion to be unsupported by empirical observation. His own observation of the children at Bradley and at the University of Chicago's Research Center for Childhood Schizophrenia made clear that their unusual and frustrating behavior was due to developmental phenomenon such as the way in which they used their senses (Schopler 1965, 1966). He could identify no way in which the children's behavior was associated with parenting. In his research on parents, he found no evidence that parents of children with autism had higher rates of thought disorders than parents of typical children (Schopler and Loftin 1969). His conclusion, therefore, was that blaming parents served a purpose unrelated to a true understanding of autism. In his article *Parents of Psychotic Children as Scapegoats* (Schopler 1971), Dr. Schopler used the theories of Gordon Allport to identify how mental health professionals had marginalized and undermined the parents of psychotic children (Allport 1954). Parents of children with autism were, in Dr. Schopler's view, being treated in much the same way that Allport described the treatment of the Jewish community and African-Americans

during the 1950s. To Dr. Schopler, the purpose of scapegoating parents was to blame an innocent and powerless group for the failures and frustration of professionals who were unsuccessful in treating autism.

Dr. Schopler's experiences from childhood through graduate school initiated two complementary threads in his career: (1) commitment to empowering disenfranchised parents by establishing the validity of their perspective and their usefulness in treatment, and (2) the means to accomplish the first goal was to ensure that intervention was based on sound empirical observation. The role of the parent was to be a lens through which research could be translated into useful practice rather than treatments devised by researchers which were often irrelevant to the lives of children with autism (Schopler 2005a). With this mindset, Dr. Schopler arrived at the University of North Carolina at Chapel Hill and joined the Department of Psychiatry where he met a like-minded psychiatry resident, Robert Reichler, M.D. The pair initiated the Child Research Study which demonstrated the efficacy of structured treatments in autism and the need to include parents as cotherapists in their child's treatment. When the study was completed, Drs. Schopler and Reichler had established the empirical evidence to begin the TEACCH program. Dr. Schopler would be the first to remind admirers that TEACCH was not his idea. The idea was generated by the parents participating in the Child Research Study. Parents provided that "lens" which would guide the application of this important research. They also provided the energy and commitment to convince the North Carolina legislature to fund TEACCH. But collaboration was still at the heart of this effort. It is unlikely that TEACCH could have been created without the evidence provided to the legislature by Dr. Schopler, Dr. Reichler, and even the endorsement of Dr. Leo Kanner.

Under his leadership, TEACCH continued to grow, but Dr. Schopler's efforts had an impact far beyond the border of North Carolina and Division TEACCH. In 1974, he took on the position as

editor of the *Journal of Autism and Childhood Schizophrenia* (later the *Journal of Autism and Developmental Disorders*) at the request of its outgoing editor, Leo Kanner. He was committed to developing this fledgling journal into a major outlet for soundly completed research. His research standards were high. Dr. Schopler refused, however, to lower the standards of scientific study simply to fill the pages of a journal devoted to a young field. Dr. Schopler used this new role to continue his efforts at marrying research to family needs. He initiated a “parents speak” section in which parents would occasionally weigh in on issues important to the journal. He also added the “Ask the Editor/Expert” section of the journal which invited parents to raise practical questions to be addressed by the editor (Schopler 1974).

Dr. Schopler’s commitment to high scientific standards and awareness of the needs of parents and their children were the foundation for many debates within the scientific and clinical literature. Having begun his career by challenging the leaders of his field, he did not shy from an argument now that he was one of the leaders. In the early days, psychodynamic theories and treatments of autism were an obvious focus of criticism for a researcher endorsing empirical methods. Later controversial topics such as facilitated communication were the object of swift critiques by Dr. Schopler (1992). He was watchful for research claims that exceeded the scope of intervention and played upon the hopes of parents by offering the hope of incredible gains where no evidence for gains of this magnitude could be found (Schopler et al. 1989). As the field developed, however, his critiques often took a different tone. Instead of invalidating a proposed intervention, he became a voice for moderation. He often weighed in on arguments by reminding researchers and clinicians of the need for individualization. He recognized from both his professional and personal history the dangers of dogmatically adhering to one approach (Schopler 1989). He endorsed research that explored the mechanisms that could predict which individuals with autism might respond to

an intervention and which might not. He commented on the complexity of this type of discourse and the need to gather many types of data including data that was not easy to quantify (Schopler 2005b).

Long past his death in 2006, Dr. Schopler’s influence continues to grow. His participation in teaching and mentoring young professionals has had an impact on many of the leading researchers and clinicians of our time. The authorship of “over 200 books and articles” is often quoted. It is less frequently noted that his writings have been cited nearly 10,000 times, clear evidence that his work will continue to be read far into the future. His commitment to training others in the TEACCH model has led to replications of the program worldwide. But one of his most lasting contributions is probably one of the least noticed. In large part because of his work, no parent (whose child is seen by a competent professional) will be treated as a scapegoat for their child’s autism. Encouraging parent participation in their child’s treatment is now the norm. Today, most parents of young children are unaware that that the position of parents was ever different than it is now. This is a gift parents today receive from Dr. Schopler and the parents with whom he collaborated. As mentioned earlier, Dr. Schopler’s most valued professional experiences were with individuals with autism and their families. The thanks of parents who knew him most eloquently express Dr. Schopler’s commitment to individuals with autism. In a remembrance offered at Dr. Schopler’s memorial, Mary Lou Warren recounted when she and her husband first met Dr. Schopler as participants in the Child Research Study in 1967 (Warren 2006). In just a few sentences, she captures Dr. Schopler as an individual and his core contribution to the field of autism:

I remember the first time that Frank and I saw Eric. He came out of his office to greet us on our first visit to Trailer 18, just outside the doorway of the Psychiatry wing. He was dressed in a plaid shirt and khaki pants and had mud on his shoes. And I remember asking myself, “Could this be the doctor?!” I was not quite prepared for this,

because what I'd been used to was white coats, stiff upper lips, and blank stares. Later that day as we walked down the sidewalk to our car, I had tears in my eyes as I looked at Frank and said, "My God! We have finally found someone who wants to help us."

See Also

- ▶ [American Psychological Association](#)
- ▶ [Childhood Autism Rating Scale](#)
- ▶ [Mesibov, Gary](#)
- ▶ [Refrigerator Mother](#)
- ▶ [Structured Teaching](#)

References and Reading

- Allport, G. (1954). *The nature of prejudice*. Cambridge, MA: Perseus Books.
- Mesibov, G., Schopler, E., Schaffer, B., & Landrus, R. (1988). *Individualized assessment and treatment for autistic and developmentally disabled children: Vol. 4, Adolescent and adult psychoeducational profile (AAPEP)*. Austin: PRO-ED.
- Mesibov, G. B., Shea, V., & Schopler, E. (2005). *The TEACCH approach to autism spectrum disorders*. New York: Springer.
- Mesibov, G., Thomas, J., Chapman, M., & Schopler, E. (2007). *The TEACCH transition assessment profile (TTAP)*. Austin: PRO-ED.
- Reichler, R., & Schopler, E. (1971). Observations on the nature of human relatedness. *Journal of Autism and Childhood Schizophrenia*, 1, 283–296.
- Rutter, M., & Schopler, E. (1987). Autism and pervasive developmental disorders: Concepts and diagnostic issues. *Journal of Autism and Developmental Disorders*, 17, 159–186.
- Schopler, E. (1965). Early infantile autism and receptor processes. *Archives of General Psychiatry*, 13, 327–335.
- Schopler, E. (1966). Visual versus tactual receptor preferences in normal and schizophrenic children. *Journal of Abnormal Psychology*, 71, 108–114.
- Schopler, E. (1971). Parents of psychotic children as scapegoats. *Journal of Contemporary Psychotherapy*, 4, 17–22.
- Schopler, E. (1974). New publisher, new editor, expanded editorial policy – Goal: An improved Journal. *Journal of Autism and Childhood Schizophrenia*, 4, 91–92.
- Schopler, E. (1978). On confusion in the diagnosis of autism. *Journal of Autism and Childhood Schizophrenia*, 8, 137–138.
- Schopler, E. (1989). Excesses of the normalization concept. *American Psychologist*, 44(2), 447–448.
- Schopler, E. (1992). Editorial commentary. *Journal of Autism and Developmental Disorders*, 22, 337–338.
- Schopler, E. (Guest Editor). (2000). International priorities for developing autism services via the TEACCH model [2-part special issue]. *International Journal of Mental Health*, 29(1–2), 1–183.
- Schopler, E. (2001). Treatment for autism: From science to pseudoscience or anti-science. In E. Schopler, N. Yirmiya, L. Marcus, & C. Shulman (Eds.), *The research basis of autism intervention* (pp. 9–21). New York: Kluwer Academic/Plenum.
- Schopler, E. (2005a). A video tribute to Eric Schopler.
- Schopler, E. (2005b). Comments on "challenges in evaluating psychosocial intervention for autism spectrum disorders" by Lord, et al. *Journal of Autism and Developmental Disorders*, 35, 709–711.
- Schopler, E., & Loftin, J. (1969). Thinking disorders in parents of psychotic children. *Journal of Abnormal Psychiatry*, 74, 271–287.
- Schopler, E., & Olley, J. G. (1982). Comprehensive educational services for autistic children: The TEACCH model. In C. R. Reynolds & T. B. Gutkin (Eds.), *Handbook of school psychology* (pp. 626–643). New York: Wiley.
- Schopler, E., & Reichler, R. J. (1971). Parents as cotherapists in the treatment of autistic children. *Journal of Autism and Childhood Schizophrenia*, 1, 87–102.
- Schopler, E., & Reichler, R. (1979). *Individualized assessment and treatment for autistic and developmentally delayed children. Volume I, psychoeducational profile*. Baltimore: University Park Press.
- Schopler, E., Brehm, S., Kinsbourne, M., & Reichler, R. (1971). Effect of treatment structure on development in autistic children. *Archives of General Psychiatry*, 24, 415–421.
- Schopler, E., Reichler, R. J., DeVellis, R. F., & Daly, K. (1980). Toward objective classification of childhood autism: Childhood autism rating scale (CARS). *Journal of Autism and Developmental Disorders*, 10, 91–103.
- Schopler, E., Mesibov, G. B., Shigley, R. H., & Bashford, A. (1984). Helping autistic children through their parents: The TEACCH model. In E. Schopler & G. B. Mesibov (Eds.), *The effects of autism on the family* (pp. 65–81). New York: Plenum.
- Schopler, E., Reichler, R. J., & Renner, B. R. (1988). *The childhood autism rating scale (CARS), revised*. Los Angeles: Western Psychological Services.
- Schopler, E., Short, A., & Mesibov, G. (1989). Relation of behavioral treatment to "normal functioning": Comment on Lovaas. *Journal of Consulting and Clinical Psychology*, 57, 162–164.
- Schopler, E., Mesibov, G. B., & Hearsey, K. (1994). Structured teaching. In E. Schopler & G. B. Mesibov (Eds.), *Behavioral issues in autism* (pp. 195–207). New York: Plenum.

- Schopler, E., Lansing, M., Reichler, R. J., & Marcus, L. M. (2005). *Psychoeducational profile third edition (PEP-3)*. Austin: PRO-ED.
- Schopler, E., Van Bourgondien, M. E., Wellman, G. J., & Love, S. R. (2010). *Childhood autism rating scale* (2nd ed.). Los Angeles: Western Psychological Services.
- Sullivan, R. C. (2005). A video tribute to Eric Schopler.
- Warren, M. L. (2006). Remembrance of Eric Schopler. Text can be found at <http://ericschopler.blogspot.com/2006/07/remembering-eric.html>

Scoliosis

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Synonyms

[Curvature of the spine; Spinal curvature](#)

Definition

Scoliosis is an orthopedic condition characterized by a lateral curvature of the spine. It can be congenital (present at birth) or develop with growth. It may be secondary to malformations of the vertebrae (bones in the spine) or ribs and may be associated genetic syndromes. Scoliosis can also develop with neuromuscular conditions like cerebral palsy, spina bifida, and muscular dystrophy where there is imbalance of the muscular forces on the spine. The most common type of scoliosis is idiopathic. This means it occurs for unknown reasons most commonly with growth spurts as occur in adolescence. Clinically significant idiopathic scoliosis is more common in girls. It seems to run in families. On physical examination, one shoulder or hip may appear higher than the other, and there may be asymmetry with examination of the back while the

patient is bending forward at the waist. It may be associated with leg length discrepancy. Specialized x-rays are necessary to document both the curvature and if there is compensatory curvature in the opposite direction (S curve). Treatment depends on the age of the patient and the cause of the scoliosis. Idiopathic scoliosis with curves with less than a 20° angle on x-ray measurement is monitored during growth. Angles over 25° typically are managed with bracing to prevent further curvature. Braces can be taken off for sports. Bracing is much less helpful for scoliosis in younger children and due to neuromuscular disorders. Curves over 40° may require surgical correction. There are several different surgical approaches that involve fusing the bones of the spine together and/or using rods to help straighten the back.

Milder scoliosis can lead to back discomfort over time. More significant scoliosis associated with neuromuscular disorders may be lead to other significant symptoms associated with mobility, positioning, and orthopedic health. Severe scoliosis with curves approaching 100° may impact breathing. Therapies such as chiropractic manipulation and electrical stimulation have not been demonstrated to prevent progression of scoliosis. While physical therapy may be helpful for other symptoms of neuromuscular disorders, it does not prevent progression of scoliosis.

See Also

► [Rett Syndrome](#)

References and Reading

- <http://www.mayoclinic.com/health/scoliosis/DS00194>
<http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0002221/>
<http://www.nlm.nih.gov/medlineplus/scoliosis.html>
<https://healthychildren.org/English/health-issues/conditions/orthopedic/Pages/Scoliosis.aspx>. 25 Aug 2017.
https://www.niams.nih.gov/Health_Info/Scoliosis/. 25 Aug 2017.

Scotland and Autism

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History

It is perhaps fair to say that, as a comparatively small country in terms of population, Scotland has played a leading role in many aspects of autism practice and research.

Scotland's autism story possibly began a long time ago. While this article will not focus on people with autism, one of the earliest presentations which has been claimed to be a case of autism is that of a Scottish minor aristocrat, Hugh Blair of Borgue (1708–1760s) (Hugh Blair of Borgue – Wikipedia 2016). Blair is described to have presented with various difficulties compatible with the diad (or triad) of impairments, such as obliviousness to social cues, bizarre collecting interests, and repetitive routines. However, Scotland's prominence in terms of autism history predominantly occupies the last 50 years. The pioneering work of Leo Kanner in the USA led to increasing awareness among Scottish parents about autism and their search for clinicians and educationalists who were interested in helping their affected children in their own country.

On the clinical side, individual child psychiatrists in Glasgow and Edinburgh began to investigate the needs of children with autism and develop services for them. Fred Stone (1921–2009) (Moore and Stone 2009) included autism in his wide ranging approach to the mental health needs of children and adolescents following his appointment to only the second consultant child and adolescent psychiatry post in Scotland in 1954. Over the next decade, Stone's work led, inter alia, to the founding of the Scottish Centre for Autism, which pioneered

multidisciplinary assessment and preschool intervention for ASD. On the east coast, Sula Wolff (1924–2009), consultant child psychiatrist at Edinburgh, began to study children with autism in the 1960s, culminating in her seminal work *Loners* (1995). Wolff preferred to use the term “schizoid” to classify socially dysfunctional children. This lack of clear linkage to the work of other emerging UK experts on autism, such as Michael Rutter, possibly rendered Wolff's work insufficiently recognized (Watts and Sula 2009). This emerging clinical interest in autism in Scotland was underpinned by observational research into infant-parent interaction led by Colwyn Trevarthen in Edinburgh, from the 1970s onwards, which generated primary psychological theories about intersubjectivity, with autism identified as a paradigm of intersubjective dysfunction.

In the 1990s, the pioneering work of Stone, Wolff, Trevarthen, and others fostered an emerging cohort of clinicians from all the key disciplines who began to develop services across Scotland, not just in the major teaching centers (McClure et al. 2010).

Following the ground breaking development of DSM-III in 1980 and increasing clarification and consensus about the nature of autism through the ensuing decade, what became increasingly clear throughout the 1990s was the lack of consensus about the autism evidence base. In 1971, writing about the 11 child subjects of his classic 1943 paper *Autistic Disturbances of Affective Contact*, Leo Kanner stated (1971):

We must keep in mind that they were studied before the days when a variety of therapeutic methods were inaugurated, based on a variety of theoretical premises: psychoanalytically oriented, based on operant conditioning, psychopharmacological, educational, via psychotherapy of parents, and combination of some of them. Sufficient time has not elapsed to allow meaningful long-range follow up evaluations. At any rate, no accounts are as yet available that would afford a reasonably reliable idea about the more than temporary or fragmentary effects of any of these procedures intended for amelioration.

Scottish clinicians took an active interest in international developments in the autism field

throughout the 1980s and early 1990s and, through a combination of individual efforts, answered Kanner's call for an overview of the evidence base in autism. This issue achieved a particularly Scottish focus with the publication of the Public Health Institute of Scotland's *Needs Assessment Report* in 2001 (Autistic Spectrum Disorder – Needs Assessment Report 2012). One of its key recommendations was that the Scottish Intercollegiate Guidelines Network (SIGN) should develop a practice guideline in autism. At the time, this was a novel suggestion, because hitherto, medical guidelines had focused on conditions amenable to quantitative, as opposed to qualitative research. SIGN's foresight in commissioning what became guideline 98 (2016) meant that Scotland led the way in producing the first national evidence based guideline for ASD. SIGN 98 pioneered use of guideline versions for children and young people, as well as parents and carers and produced an e-CPD module accessible via the SIGN website to anyone in the world. Awareness of autism, and its implications for clinical services, had come of age and one of the key clinical challenges for the next decade is how clinical services are going to address autism needs in a fair and equitable approach across the age range.

In parallel, and sometimes in partnership with academic and clinical pioneers, the powerful parent lobby in Scotland has also been instrumental in driving forward autism friendly services, since the Scottish Society for Autistic Children (SSAC) formed in 1968 (Feinstein 2010). Feinstein outlines the history of the parent experience in the 1960s and 1970s, when parent organizations grew but were only accessible to a small number of families. The 1990s saw the rise of many local parent groups affiliated to the larger National Autistic Society (2016) such as the Strathclyde Autistic Society and the Lothian Autistic Society (2016). These were formed in the same way as the older organizations – by parents in need. In 1992, Rhona Gallagher recalls that she and two other mothers of children with autism met up for a coffee in a local hotel and that was the start of the Lothian Autistic Society (LAS). It just got

bigger and bigger and eventually in mid-1990s there were over 200 members between parents and professionals:

Although I was a member of NAS and SSAC at the time, it was really nice to meet up face to face with other mums for support and sharing experiences as ASD wasn't as commonly known then as it is now. We were all concerned about educational placements and the lack of knowledge and understanding within the city (of Edinburgh). I think that once the LAS was formed and it was a local society, then parents felt that they had "a voice" and were able to make a lot of their concerns and frustrations known to various professional bodies. There was an enormous issue at the time where whole families were suffering due to the behaviour of the child with autism and there was no respite provision available. From this issue, the LAS voucher system was formed and this gave parents the respite that a lot of them were desperately in need of in order to function. The LAS is now more than 20 years old and still going strong and has become a huge support to parents, siblings, play schemes, social clubs, and respite. It has changed and evolved over the years and now is a professional charity with over 700 family members and professionals.

Parents like Rhona lobbied for parent/carer support and for new educational provision for the growing number of children being diagnosed with autism. They were supported in this by professionals, who also saw the need to develop new ways of working. In the 1980s, most autism schools in Scotland were residential, such as the charity funded schools Struan House and Camphill. One of the first local authority funded autism provisions, Middlefield Residential School in Glasgow, opened in 1988. This school was set up by Strathclyde Regional Council (SRC) as a national resource (for Scotland) and admitted pupils from Lothian, Ayrshire, Inverness, Dumfries, as well as Glasgow and outlying areas.

The staff at the Department of Child & Family Psychiatry (DCFP), which later became the Scottish Centre for Autism at the Royal Hospital for Sick Children at Yorkhill, Glasgow, and the parents they worked with were instrumental in raising the need for appropriate educational facilities for the children who were attending their intervention program (now the Scottish Centre for Autism and parent programme). Most of the

original pupils were those known to DCFP. This was one catalyst for the improvement of local provision and the development of professional parent partnerships.

The 1990s brought a significant shift in educational provision and practice for children with autism in Scotland, when other local authorities across Scotland began to open language unit provisions in mainstream schools and classes in special schools, specifically for children with communication disorders or autism. These provisions were staffed by Education and therapy staff, working in collaboration.

By the 1990s, the importance of early intervention was also increasingly being highlighted by parents and this also led to professional/parent partnerships in developing new models of service delivery and support. In Edinburgh, the Changing Children's Services Fund was used in 1996 to set up *Spectrum* – a multiagency intervention team providing support for preschool children and their families from the point of concern that autism may explain the child's difficulties (not from the time of diagnosis, which may be much later). The Scottish Centre for Autism early intervention programme published evidence that 10 h of intervention was as effective as some of the 40 h a week private intervention programs being advertised in the USA. Achievable and sustainable models of early intervention were being developed by statutory services.

Interventions and teaching methods in use internationally began to be adopted locally such as use of TEACCH (Mesibov et al. 2004), the Hanen parent programmes (Sussman 2006, 2012; Carter et al. 2011), both of which emerged in the 1970s in North America and Canada but came to Scotland in the early 1990s. While intensive interaction (Nind and Hewett 2012) and Makaton signing (Mirenda and Teresa 2009) were widely used in Scotland with children with learning or language difficulties, there was minimal use of either approach or of visual and symbol supports for children with autism. The advent of technology (e.g., laminators, computer access to symbols) supported the developing use of visual supports for children with autism (Howlin

et al. 2007; PECS UK 2016) and growing awareness that the environment was crucial in supporting children with autism. The Hanen programme advocated the importance of the impact of parents and carers in using everyday interactions to enhance development in children with communication impairments and then specifically for those with autism (Carter et al. 2011).

Since 2000, there has been a further shift and growth in educational practice and thinking with a move towards increased inclusion of pupils with autism and other additional needs in local schools, with "the presumption of mainstream" being adopted (Standards in Scotland's Schools Act, 2000) and the requirement of schools to adapt to the needs of all children (Education (Additional Support for Learning) Acts 2004 and 2009; The Equality Act 2010). Practice guidance for practitioners became more widely available, for example, due to the Autism Toolbox (2016). While this process engenders new challenges, the context in Scotland is increasingly positive and statutory services are working in partnership with parent groups and the third sector, researchers and professionals, to work towards improving recognition and models of service provision across the country.

Overview of Assessment and Supportive Intervention and Social Policy/Training Initiatives

Scotland has produced several centers of excellence in terms of educational and clinical practice in autism over the last 50 years, which have been the subject of international attention. This productivity stems in part from the strong tradition of relatively well-resourced, public-funded health and education services in Scotland. However, third sector/charitable organizations have also pioneered service development, often leading the way when public services have failed to keep up with emerging needs. Thus the Steiner movement in Camphill (Aberdeen) and Ochil Tower School (Auchterarder), the New School at Struan (Scottish Society for Autism), and the Scottish

Centre for Autism have all produced in-house approaches to autism intervention and support which have been adopted and adapted by services at home and abroad.

The work of the Scottish Government's Autism Reference Group (ARG) (2002 to date, now called the Scottish Autism Strategy Governance Group) has helped to frame many concepts in the area of autism assessment (The Quality Diagnostic Standard) and educational support and intervention (The Autism Toolbox) (2016) which have begun to generate a standardized quality and approach to services nationally, across disciplines and agencies. The Scottish Government's work on autism has been formulated within a 10-year Autism Strategy, funded by £14.5 million (2016), meaning that the Scottish Government is, arguably, one of the leading national legislatures in terms of developing autism services.

Autism assessment has gradually developed a standardized approach throughout the country and its improvement has, arguably, been one of Scotland's key successes in comparison with other nations. NHS Education for Scotland (NES) funded the development of an ADOS Training network from the mid 2000s onwards, which has steadily increased the numbers of professionals from all agencies offering this skill. Training in other diagnostic instruments such as DISCO and 3di has been supported by the Scottish Government, via the ARG. Thus autism assessment services are now in a much healthier state than they were in the early 1990s. However, there are still major challenges. Autism assessment waiting lists for children are growing, capacity is insufficient, and services for adults (especially those for adults without a learning disability) are nonexistent in some parts of Scotland. Recently, the Scottish Government funded the Autism ACHIEVE Alliance (AAA) to survey the state of autism assessment services across the age range, across the country and these findings have been published as an Executive Summary report (Autism Spectrum Disorders – Waiting For Assessment & Executive Summary 2014) as well as peer reviewed publications (Mckenzie et al. 2015; Rutherford et al. 2016a, b). This is the first time any national government has

attempted to establish the state of a country's autism assessment services and the AAA findings will provide a framework for action, in terms of how to improve the situation, over the next decade.

Overview of Research Directions

Several pioneers of early autism research have been based in Scotland, such as Stone, Wolff, and Trevarthen. The SIGN guideline helped to demonstrate key gaps in the autism evidence base affecting Scotland and many other countries (Guideline 2016). However, mainstream psychiatric research in Scotland has only recently begun to take an interest in autism, perhaps reflecting the relatively weak base of research into child and adolescent mental health in Scotland, compared to the historical emphasis on schizophrenia and other conditions which mainly affect adults. The work of the Scottish Government and other agencies, over the last decade, has helped to galvanize autism research and bring local researchers into contact with international pioneers, to hopefully stimulate and assist a new generation of autism researchers.

See Also

- ICD 10 Research Diagnostic Guidelines
- Practice Guidelines in Autism

References and Reading

- Autism Spectrum Disorders – Waiting For Assessment, Executive Summary. Autism ACHIEVE Alliance/ Scottish Government. (2014).
- Autistic Spectrum Disorder – Needs Assessment Report. Public Health Institute of Scotland. (2002).
- Carter, A. S., Messinger, D. S., Stone, W. L., Celimli, S., Nahmias, A. S., & Yoder, P. (2011). A randomized controlled trial of Hanen's "More Than Words" in toddlers with early autism symptoms. *Journal of Child Psychology and Psychiatry*, 52, 741–752.
- Feinstein, A. (2010). *A history of autism: Conversations with the pioneers*. John Wiley & Sons.
- Guideline 98. (2016). Assessment, diagnosis and clinical interventions for children and young people with autism spectrum disorders. sign.ac.uk. (<http://www.sign.ac.uk/guidelines/fulltext/98/>)

- Howlin, P., Gordon, K. R., Pasco, G., et al. (2007). The effectiveness of picture exchange communication system (PECS) training for teachers of children with autism: A pragmatic, group randomised controlled trial. *Journal of Child Psychology and Psychiatry*, 48, 473–481.
- Hugh Blair of Borgue – Wikipedia, the Free Encyclopedia. (2016). en.wikipedia.org. (http://en.wikipedia.org/wiki/Hugh_Blair_of_Borgue)
- Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of autism and childhood schizophrenia*, 1(2), 119–145.
- Lothian Autistic Society. (2016). (<http://www.lothianautistic.org/>)
- McClure, I., Mackay, T., Mamdani, H., & McCaughey, R. (2010). A comparison of a specialist autism spectrum disorder assessment team with local assessment teams. *Autism*, 14, 589–603.
- McKenzie, K., Forsyth, K., O'Hare, A., McClure, I., Rutherford, M., Murray, A., & Irvine, L. (2015). Factors influencing waiting times for diagnosis of Autism Spectrum Disorder in children and adults. *Research in developmental disabilities*, 45, 300–306.
- Mesibov, G. B., (null), Shea, V., Schopler, E., et al. (2004). *The TEACCH approach to autism spectrum disorders*. New York: Springer.
- Mirenda, P., & Teresa, I. (2009). *Autism spectrum disorders and AAC*. Baltimore: Paul Hamlyn.
- Moore, W. (2009). Fred Stone. *BMJ*, 339, 807.
- National Autistic Society. (2016). (<http://www.autism.org.uk/>)
- Nind, M., & Hewett, D. (2012). *Access to communication: Developing the basics of communication with people with severe learning difficulties through intensive interaction*. Brighton: Routledge.
- PECS UK. (2016). (<http://www.pecs-unitedkingdom.com/>)
- Rutherford, M., McKenzie, K., McClure, I., Forsyth, K., O'Hare, A., McCartney, D., & Finlayson, I. (2016a). A national study to investigate the clinical use of standardised instruments in autism spectrum disorder assessment of children and adults in Scotland. *Research in Autism Spectrum Disorders*, 29, 93–100.
- Rutherford, M., McKenzie, K., Forsyth, K., McCartney, D., O'Hare, A., & McClure, I. (In press 2016b). Why are they waiting? Exploring professional perspectives and developing solutions to delayed diagnosis of Autism Spectrum Disorders in adults and children. *Research in Autism Spectrum Disorders*.
- Scottish Government SAHRE3T058CSGGU. (2016). The Scottish Strategy for Autism. *scotlandgov.uk*.
- Wolff, S. (1995). *Loners: The life path of unusual children*. Psychology Press.
- Sussman, F. (2006). *TalkAbility: People skills for verbal children on the autism spectrum: A guide for parents*. Toronto: Hanen Program.
- Sussman, F. (2012). *More than words: A guide to helping parents promote communication and social skills*. Toronto: Hanen Centre.
- The Autism Toolbox. (2016). (<http://www.scotland.gov.uk/Resource/Doc/278458/0083689.pdf>)
- Watts, G. (2009). Sula Wolff. *The Lancet*, 374(9703), 1740

SCQ

► Social Communication Questionnaire

Screening Instruments for ASD

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Definition

This entry focuses on screening instruments for autism spectrum disorders (ASD). By screening instruments, we are referring to two different purposes of instruments: Level 1 (Universal Screening or General Population Screening) and Level 2 (often tailored to other instruments for ASD screening in high risk cohorts, such as clinical samples). Level 1 screening instruments are often designed as parental questionnaires, while Level 2 screening instruments are typically designed to probe for ASD specific symptoms utilizing parent-endorsement and/or observations by a clinician. In terms of Level 1 screening instruments, the Modified Checklist for Autism in Toddlers (M-CHAT)(R/F) (Ibanez et al. 2014; Robins et al. 2001, 2014) is the most widely used instrument for ASD-specific screening. The M-CHAT is designed to be completed in a primary care provider setting (Robins et al.

2001). Among Level 2 screening instruments, the most frequently used examinations/assessments are the Childhood Autism Rating Scale (CARS/CARS2) (Schopler and Reichler 1988; Schopler et al. 1980), the Social Communicative Questionnaire (SCQ) (Rutter et al. 2003), and the Screening Tool for Autism in Toddlers (STAT) (Stone et al. 2004). These screening instruments are tailored for identification of ASD, usually primarily in toddlers and young children. Both Level 1 and Level 2 instruments aim to identify symptoms within the categories of symptom descriptions (social, communication, and RRB), more specifically behaviors related to sharing, perspective-taking, joint attention, language development, and unusual/encompassing behaviors and interests.

To increase cost efficiency and decrease the threshold for utilization, these instruments are most often designed as brief parent endorsed questionnaires that can be completed out swiftly in, e.g., the waiting room of a pediatrician. The ideal aim of screening instruments is to identify as many (if not all) of the children with the condition that are in the population screened.

Background

Over the past 30 years an increasing amount of studies on early identification of ASD have emerged. A wide range of screening instruments have been developed and further modified as the criteria for ASD have changed across diagnostic manuals (American Psychiatric Association 2000, 2013; Banach et al. 2009; World Health Organization 1992). Baron-Cohen et al. (1992) conducted one of the first studies to utilize an ASD-specific screening instrument, which assessed the performance of the Checklist for Autism in Toddlers (CHAT). The checklist was a nine-item parent endorsed list of behaviors and in addition to these items, it included three observational-based items intended for the general practitioner to complete during the health visit (Baron-Cohen et al. 1992). The result of screening approximately 16,000 18 months old

children was that almost all of the screen positive children were diagnosed with ASD when assessed. The screening criteria was defined as failing the three predefined key items of the nine endorsed by parents and confirmed through the general practitioner's observations. The researchers also conducted a 6-year follow-up study of the same population that was screened at 18 months. The 6-year follow-up revealed that a large proportion of the children with an ASD diagnosis at follow-up were not identified by CHAT at 18 months at the health visit (Baird et al. 2000). These were false negative cases on the CHAT.

Some of the false negatives in this study may have been due to the widening of diagnostic criteria for the various autism spectrum diagnoses that occurred during the 1990s. In 2001, Robins and Colleagues (Robins et al. 2001) published a validation study on a modified version of the CHAT, including a range of new items to the screener. The aim of the Modified Checklist for Autism in Toddlers (M-CHAT) and arguments for including a range of new items was to identify the broader autism spectrum now included in both the ICD-10 (World Health Organization 1992) and the DSM-IV-TR (American Psychiatric Association 2000). None of the studies conducted utilizing the M-CHAT during the early 2000s included diagnostic assessment or prospective follow-up of all screen negative cases (Kleinman et al. 2008; Robins et al. 2001). Many of the studies using M-CHAT reported excellent psychometric properties with both high sensitivity and specificity. This contributed to M-CHAT as the recommended (preferred) screening instruments in most parts of the world, and it has been validated in various languages and cultures (Robins 2018). The M-CHAT has received some criticism for the high rates of false positives, identifying children as probable ASD in 10% of the unselected population without receiving the diagnosis upon assessment. This resulted in the development of the M-CHAT R/F (Robins et al. 2014) with the explicit goal of reducing the number of false positives. However, the instrument has yet to be examined in terms of also reducing the number of false negatives.

Current Knowledge

In recent years, the recommendation relating to Level 1 screening (i.e., universal or general population screening) have ranged from recommending it (Committee on Practice and Ambulatory Medicine, Bright Futures Periodicity Schedule Workgroup 2016; McPheeters et al. 2016) to not recommend using it in the general population due to lack of evidence for the efficacy of screening entire populations (Siu et al. 2016; UK National Screening Committee 2012). In terms of the latter, researchers have not presented evidence whether existing screening instruments are able to detect ASD across the whole spectrum of symptom patterns in children who develop ASD even though it seems like most instruments pick up on the most severe children (Beuker et al. 2014; Kleinman et al. 2008; Robins et al. 2001; Stenberg et al. 2014; Øien et al. 2018). Another critical issue of screening in the general populations is the costs, both in terms of resources used to assess screen positive children and also in terms of concerns and unnecessary worries in parents of false positive children. Screening in general populations comes with a high price as the high number of false positives most probably would prolong the time taken before the assessment of children who truly require evaluation for ASD and can result in increased healthcare cost (Yuen et al. 2018).

The American Academy of Pediatrics recommends universal screening for ASD for all children at 18- and 24-month well-visits (Committee on Practice and Ambulatory Medicine, Bright Futures Periodicity Schedule Workgroup 2016; Zwaigenbaum et al. 2015). However, the lack of studies utilizing prospective general population data with long-term follow-up demonstrates a knowledge gap in the literature. As few studies have diagnostically assessed the children who were screened – either they were true positive, false positive, true negative, or false negative – the true performance of a given instrument cannot be calculated from the majority of screening studies. There are however some exceptions: the CHAT study utilizing a 6-year follow-up (Baird et al. 2000; Baron-Cohen et al. 2000) and

Stenberg and colleagues study utilizing a prospective pregnancy cohort in the Norwegian Mother and Child Study (with yearly patient registry linkage as children grow older) (Stenberg et al. 2014). The lack of prospective studies with follow-up limits our current understanding of how well a screening instrument performs in identifying the broad spectrum of children with ASD and to what extent only a subset of children with ASD are captured.

Many of the different screening instruments used have faced criticism due to its low specificity by capturing many children with other types of developmental problems as well as children with severe intellectual impairment, but with no ASD (Chlebowski et al. 2013; Pandey et al. 2008). In addition, research in general population samples show that screening at 18 months misses a large proportion of children who later were identified with ASD (false negatives), even when the parents, at the same time, rate their children with atypicalities on other instruments (Øien et al. 2018).

Future Directions

This entry has highlighted that screening for ASD in general populations is difficult, while use of screening instruments in high-risk samples potentially benefit children not only with ASD, but also children on the developmental path to other diagnosis. Changes to cut-offs would not improve the rate of false negatives as they most often show no actionable concern on these instruments (screen negatives with very low scores). This should encourage clinicians to not rely solely on the instrument, but utilize parental concern, surveillance of developmental milestones, and provide good clinical judgment (Øien et al. 2018).

More problematic is the fact that some children on the path of developing ASD most probably do not show the range of ASD-like symptoms that, to our current knowledge, should be indicators for raising actionable concern at 18 and/or 24 months of age. While this indicates that we, using existing screening instrument, will not be able to identify all children with ASD at 18 and/or 24 months of

age, this should still be the goal for future screening studies. A renewed focus is needed on both improving already developed screening instruments and, foremost, providing more research on how to capture children with milder general developmental delay, possibly with more spared functions linked to language and mental abilities. We know far less of the symptom patterns in these children when they were 18 or 24 months of age. It is important to take other factors into account as well in future research. The wording of items can influence how they are rated, and more research is needed focusing how this and the grading of response options on individual items influences parents/professionals rating.

When developing new screening instruments aiming to identify children also from the broader autism spectrum, it is necessary that the field looks toward prospective general population studies such as the Norwegian MoBa. Such samples will allow researchers to examine trajectories of development and behavior even when diagnosed later in life. Such studies could potentially contribute to our understanding of symptoms specific to subgroups of children, or even symptoms that we do not usually consider core symptoms. This approach should ultimately lead to a better understanding of what phenomena to probe for in the development or modification of screening instruments.

The heterogeneity in ASD increased with the introduction of the DSM-IV(-TR) (American Psychiatric Association 2000) and ICD-10 (World Health Organization 1992), as the criteria also increased the number of children within the normal range IQ and complex language. Due to the heterogeneous nature of ASD, designing brief screening instruments to detect the broader autism spectrum is associated with great difficulties. Heterogeneity over time and patterns of symptom onset as well as different developmental pathways contributes to the complexities of identifying children using only one screening instrument at a given age. Designing a flawless instrument might be impossible, but improvements in the design or modification of instruments should be a focus for future studies. It might be questioned whether a child is missed by current screening

instruments due to the fact that it does not show the amount of symptoms to raise concern at the time of screening, or if current screening instruments do not contain the relevant behavioral markers to capture children across the whole spectrum and are therefore not able to identify all children with ASD due to the heterogeneity.

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Vol. 1). Arlington: American Psychiatric Publishing. <https://doi.org/10.1176/appi.books.9780890423349>.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. Arlington: American Psychiatric Publishing.
- Baird, G., Charman, T., Baron-Cohen, S., Cox, A., Swettenham, J., Wheelwright, S., & Drew, A. (2000). A screening instrument for autism at 18 months of age: A 6-year follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(6), 694–702. <https://doi.org/10.1097/00004583-200006000-00007>.
- Banach, R., Thompson, A., Szatmari, P., Goldberg, J., Tuff, L., Zwaigenbaum, L., & Mahoney, W. (2009). Brief report: Relationship between non-verbal IQ and gender in autism. *Journal of Autism and Developmental Disorders*, 39(1), 188–193. <https://doi.org/10.1007/s10803-008-0612-4>.
- Baron-Cohen, S., Allen, J., & Gillberg, C. (1992). Can autism be detected at 18 months?: The needle, the haystack, and the CHAT. *The British Journal of Psychiatry*, 161(6), 839–843. <https://doi.org/10.1192/bjp.161.6.839>.
- Baron-Cohen, S., Wheelwright, S., Cox, A., Baird, G., Charman, T., Swettenham, J., et al. (2000). Early identification of autism by the CHecklist for autism in toddlers (CHAT). *Journal of the Royal Society of Medicine*, 93(10), 521–525. <https://doi.org/10.1177/014107680009301007>.
- Beuker, K. T., Schjølberg, S., Lie, K. K., Swinkels, S., Rommelse, N. N. J., & Buitelaar, J. K. (2014). ESAT and M-CHAT as screening instruments for autism spectrum disorders at 18 months in the general population: Issues of overlap and association with clinical referrals. *European Child & Adolescent Psychiatry*, 23(11), 1081–1091. <https://doi.org/10.1007/s00787-014-0561-8>.
- Chlebowski, C., Robins, D. L., Barton, M. L., & Fein, D. (2013). Large-scale use of the modified checklist for autism in low-risk toddlers. *Pediatrics*, 131(4), e1121–e1127. <https://doi.org/10.1542/peds.2012-1525>.
- Committee on Practice and Ambulatory Medicine, Bright Futures Periodicity Schedule Workgroup. (2016). 2016

- recommendations for preventive pediatric health care. *Pediatrics*, 137(1), e20153908. <https://doi.org/10.1542/peds.2015-3908>.
- Ibanez, L. V., Stone, W. L., & Coonrod, E. E. (2014). Screening for autism in young children. In F. R. Volkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, 4th ed., pp. 585–608). Hoboken: Wiley. <https://doi.org/10.1002/9781118911389.hautc24>.
- Kleinman, J. M., Robins, D. L., Ventola, P. E., Pandey, J., Boorstein, H. C., Esser, E. L., et al. (2008). The modified checklist for autism in toddlers: A follow-up study investigating the early detection of autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(5), 827–839. <https://doi.org/10.1007/s10803-007-0450-9>.
- McPheeters, M. L., Weitlauf, A., Vehorn, A., Taylor, C., Sathe, N. A., Krishnaswami, S., et al. (2016). *Screening for autism spectrum disorder in young children*. Rockville, MD: Agency for Healthcare Research and Quality.
- Oien, R. A., Schjølberg, S., Volkmar, F. R., Shic, F., Cicchetti, D. V., Nordahl-Hansen, A., et al. (2018). Clinical features of children with autism who passed 18-month screening. *Pediatrics*, e20173596. <https://doi.org/10.1542/peds.2017-3596>
- Pandey, J., Verbalis, A., Robins, D. L., Boorstein, H., Klin, A., Babitz, T., et al. (2008). Screening for autism in older and younger toddlers with the modified checklist for autism in toddlers. *Molecular Autism*, 12(5), 513–535. <https://doi.org/10.1177/1362361308094503>.
- Robins, D. L. (2018). M-CHAT. Retrieved October 22, 2018, from <https://mchatscreen.com/mchat-rf/translations/>
- Robins, D. L., Fein, D., Barton, M. L., & Green, J. A. (2001). The modified checklist for autism in toddlers: An initial study investigating the early detection of autism and pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 31(2), 131–144. <https://doi.org/10.1023/A:1010738829569>.
- Robins, D. L., Casagrande, K., Barton, M., Chen, C.-M. A., Dumont-Mathieu, T., & Fein, D. (2014). Validation of the modified checklist for autism in toddlers, revised with follow-up (M-CHAT-R/F). *Pediatrics*, 133(1), 37–45. <https://doi.org/10.1542/peds.2013-1813>.
- Rutter, M., Bailey, A., & Lord, C. (2003). SCQ. The Soical Communication Questionnaire. Torrance, CA: Western Psychological Services.
- Schopler, E., & Reichler, R. J. (1988). *The childhood autism rating scale (CARS)*. Los Angeles: Western Psychological Services.
- Schopler, E., Reichler, R. J., DeVellis, R. F., & Daly, K. (1980). Toward objective classification of childhood autism: Childhood autism rating scale (CARS). *Journal of Autism and Developmental Disorders*, 10(1), 91–103. <https://doi.org/10.1007/BF02408436>.
- Siu, A. L., Bibbins-Domingo, K., Grossman, D. C., Baumann, L. C., Davidson, K. W., Ebell, M., et al. (2016). Screening for autism spectrum disorder in young children: US preventive services task force recommendation statement. *JAMA*, 315(7), 691–696. <https://doi.org/10.1001/jama.2016.0018>.
- Stenberg, N., Bresnahan, M., Gunnes, N., Hirtz, D., Hornig, M., Lie, K. K., et al. (2014). Identifying children with autism spectrum disorder at 18 months in a general population sample. *Paediatric and Perinatal Epidemiology*, 28(3), 255–262. <https://doi.org/10.1111/ppe.12114>.
- Stone, W. L., Coonrod, E. E., Turner, L. M., & Pozdol, S. L. (2004). Psychometric properties of the STAT for early autism screening. *Journal of Autism and Developmental Disorders*, 34(6), 691–701. <https://doi.org/10.1007/s10803-004-5289-8>.
- UK National Screening Committee. (2012, November 13). Screening for autistic spectrum disorders in children under the age of five policy position statement and summary. Retrieved July 3, 2017, from <https://legacyscreening.phe.org.uk/autism>
- World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders*. Geneva: World Health Organization.
- Yuen, T., Carter, M. T., Szatmari, P., & Ungar, W. J. (2018). Cost-effectiveness of universal or high-risk screening compared to surveillance monitoring in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 1–12.
- Zwaigenbaum, L., Bauman, M. L., Fein, D., Pierce, K., Buie, T., Davis, P. A., et al. (2015). Early screening of autism spectrum disorder: Recommendations for practice and research. *Pediatrics*, 136(Supplement), S41–S59. <https://doi.org/10.1542/peds.2014-3667D>.

Screening Measures

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Definition

Autism screening is the process of administering a brief assessment to determine the level of risk for

autism. This is usually done by giving parents a brief questionnaire or checklist about their child's development, including social, language, motor, and cognitive development, but can also be done by direct clinical observation. If concerns about a child's development arise from the screening instrument, the child is referred for a more in-depth, diagnostic, and developmental evaluation.

Historical Background

Autism spectrum disorders (ASDs) are disorders of development; the Centers for Disease Control reported prevalence rates as 1 in 59 in 2014 (Baio et al. 2018). Evidence of neurobiological abnormalities in the first year of life (Courchesne 2004) and retrospective evaluation of infant behavior (Baranek 1999; Osterling and Dawson 1994) suggest that symptoms of ASD may be present by 12 months. Parents first report concerns around 17–18 months on average, although recent data suggest that parents first express concerns around 14–15 months, with some stating concerns before 11 months (Chawarska et al. 2007). Despite parents' early concerns, most children are not diagnosed with ASD until age 4 or even later, especially children of urban and/or low socioeconomic status (Baio et al. 2018; Earls and Hay 2006; Fombonne et al. 2001; Howlin and Asgharian 1999; Mandell et al. 2002; Pinto-Martin et al. 2005; Tonge et al. 2006; Wiggins et al. 2006; Williams and Brayne 2006; Zwaigenbaum et al. 2007). Furthermore, diagnoses of ASD have been demonstrated to be stable and reliable in children as young as 14 months of age (Pierce et al. 2019), even in children with significantly delayed language and cognitive abilities (Hinnebusch et al. 2017). Published guidelines (Filipek et al. 2000; Glascoe 2005; Gray et al. 2006; Johnson and Myers 2007) report that the positive effects of early diagnosis far outweigh the negative effects and that families express the desire to be informed as early as possible. Early identification of children with autism enables families to access early intervention services, which have been shown to lead to the best outcomes

when accessed early in life (MacDonald et al. 2014; Rogers et al. 2014). Standardized universal screening reduces the delay in diagnosis experienced by low-resource and minority families (Herlihy et al. 2014). Screening instruments that alert doctors to the possibility of ASD are important and necessary and have a tremendous impact on public health and the welfare of affected children and their families. Pediatric screening is often the only evaluation children receive until they begin preschool or kindergarten (Glascoe 2005). Therefore, what is needed is a brief, but relatively sensitive, screening instrument for use in physicians' offices that indicate a need for further evaluation in children with early signs of autism. There is clear evidence that early detection of and intervention for ASDs can lead to substantially better prognosis; thus, the American Academy of Pediatrics currently recommends that all children be screened for autism at their 18- and 24-month well-child visits and that children who "fail" screening be referred for a diagnostic evaluation (Lipkin et al. 2020). Here we describe some commonly used screening measures; a review of screening tools available for use in low- and middle-income countries can be found in Marlow et al. (2019).

While the majority of the screening instruments described here are designed for infants and toddlers, several are also applicable for older children and adults. Recent work reporting on children who are not detected by early developmental screeners but are later diagnosed with autism has concluded that use of multiple screening tools reduces false-negative risk (Beacham et al. 2018) and screening at repeated time points (e.g., 18 months and 24 months) is beneficial (Dai et al. 2019; Øien et al. 2018; Robins 2019). For screening of older children and adults, we refer the reader to the following reviews: Baghdadli et al. (2017) and Hirota et al. (2018).

Current Knowledge

Autism screeners fall into two different levels. Level 1 screening instruments are broad-based and used to identify children at risk for ASDs in

the general population. Level 2 screening instruments are more specific tools used to screen children who are at increased risk for ASDs (e.g., children with previous developmental concerns, younger siblings of children with ASDs). Level 1 screening instruments are designed to be implemented by general providers, without specialized knowledge about ASDs. Typically, pediatricians conduct screening with level 1 instruments during well-child visits. Therefore, these screening instruments must be brief and easy to administer. Level 2 screening instruments generally take longer to complete and are typically utilized by practitioners with specific training on ASDs. Level 2 screening instruments include parent report and sometimes include direct observation of the child. Some screening measures have both level 1 and level 2 components and thus can be used in a variety of settings. When evaluating screening instruments, two constructs are important: sensitivity and specificity. Sensitivity is the proportion of children correctly identified by the screening instrument as having an ASD, while specificity is the proportion of children correctly identified by the screening instrument as not having an ASD. High sensitivity is important for level 1 screening instruments in order to maximally identify all children at risk for an ASD. Specificity may be less important for level 1 screening instruments because children who show concerns based on the screening instrument are likely to benefit from evaluation and intervention whether or not they have an ASD. However, for level 2 screening instruments, specificity is more highly valued since diagnosis becomes important, and distinguishing between ASD and other developmental disabilities is necessary. In addition to sensitivity and specificity, many instruments report their positive predictive value (PPVs). This figure is the proportion of children screening positive who turn out to be true cases. This figure is very sensitive to base rates. Conditions with low base rates tend to have low PPVs. In addition, there is always a trade-off between sensitivity and PPV; instruments that pick up most cases (high sensitivity) also tend to pick up more false-positive cases, resulting in lower PPVs. Because of the trade-off with

sensitivity, and because of the variation in PPV with base rate, PPV is not always reported. A recent meta-analysis of screening tools in toddlers (Sánchez-García et al. 2019) indicated that screening tools in toddlers are effective and accurate, and the authors include several recommendations for future screening studies.

Broad Developmental Screening Tools

Broad developmental screening tools are designed to assess risk for any developmental disorder and are not specifically designed to assess risk for ASDs. A thorough examination of all such measures is beyond the scope of this entry (see Glascoe 2000 or Macy 2012 for a review); however, three common broad developmental screening tools are discussed, all of which are recommended by the American Academy of Pediatrics (AAP) Screening Tool Finder. The Parents' Evaluation of Developmental Status (PEDS) (Glascoe 2001) is a screening instrument designed for use in children between birth and 8 years of age. The PEDS contains ten items that assess parent concern in developmental domains such as fine and gross motor skills, receptive and expressive language, and self-help skills. Based on the reported parental concerns, the child is classified as being at low, moderate, or high risk for developmental issues. The PEDS showed sensitivity ranging from 0.74 to 0.79 and specificity of 0.70 to 0.80 across a broad age range. The Ages and Stages Questionnaires, Third Edition (ASQ-3; Squires et al. 2009), is a screening instrument designed for use in children from 1 to 66 months. There are 21 different forms specific to the skills expected of the various age levels assessed. There are 30 items divided into 5 areas: communication, gross motor, fine motor, problem solving, and personal social. The ASQ-3 showed sensitivity of at least 0.86, and specificity has been measured at 0.85 for all age groups. There are questions on behavior and communication within this third edition that help identify parent concerns that may be related to ASD. Emerging data on whether the PEDS or the ASQ-3 is targeted enough for the identification

of children at risk for an ASD suggest that they may be useful as first-level screeners if fairly low thresholds are used, but current findings are limited and require further study (Wiggins et al. 2014; Hardy et al. 2015). A third screening measure used to identify a range of developmental concerns is the Survey of Wellbeing of Young Children (SWYC), designed for children under age 5½ years. There are 12 separate versions for children of different ages (designed to be administered at each well-child checkup until age 5 years). Autism-specific scales are included for children 16–36 months of age (Perrin et al. 2016). Early estimates of validity suggest the SWYC is comparable to other developmental screening tools, and the authors are continuing to evaluate validity compared to standardized developmental testing.

Autism-Specific Screening Tools

Checklist for Autism in Toddlers (CHAT; Level 1)

Developed in the United Kingdom (UK), the CHAT (Baron-Cohen et al. 1992, 1996, 2000) is a screening tool developed for pediatricians to use at the 18-month checkup for children. The CHAT consists of nine yes/no questions to be answered by the child's parent. These questions ask if the child exhibits specific behaviors, including social play, social interest in other children, pretend play, joint attention, pointing to ask for something, pointing to indicate interest in something, rough and tumble play, motor development, and functional play. The CHAT also includes observations of five brief interactions between the child and the examiner, which enable the clinician to compare the child's actual behavior with the parental reports. The specificity of the CHAT is excellent; however, the sensitivity is quite low (0.20–0.38 depending on criteria used).

Early Screening of Autistic Traits Questionnaire (ESAT; Level 1)

The ESAT (Dietz et al. 2006; Swinkels et al. 2006) screening tool consists of 14 yes/no items completed by parents of children age

0–36 months (although it has only been empirically demonstrated in children age 8–20 months). It covers the domains of pretend play, joint attention, interest in others, eye contact, verbal and nonverbal communication, stereotypes, preoccupations, reaction to sensory stimuli, emotional reaction, and social interaction. Children failing three or more items are considered at risk for ASD. Early estimates suggest that the ESAT's sensitivity is greater than 0.9, but specificity is considerably lower. However, these values were derived from a sample that was pre-screened and thus should be interpreted with caution, as they are not derived from the general population (Towle and Patrick 2016). Additionally, the ESAT has not been demonstrated in a prospective sample.

Communication and Symbolic Behavior Scales Developmental Profile Infant-Toddler Checklist (CSBS-DP-IT Checklist; Level 1)

The CSBS-DP-IT Checklist (Wetherby et al. 2002) is a 24-item parent-report questionnaire shown to have both high sensitivity and specificity. There are three subdomains: social and emotional communication, receptive and expressive speech, and symbolic behavior. Because the CSBS-DP-IT Checklist was designed to detect children with a variety of communication delays, it tends to be sensitive to autism as well. For clinical purposes, it has the potential to identify a wide range of children who may benefit from early treatment. A study by Pierce et al. (2011) reported a PPV of 0.75 for ASD and other developmental disorders compared to typical development when used in pediatric well-child visits at 12 months of age, but pediatricians were free to override screening results and fail to refer children for evaluation. Twenty percent of screen positives were found to have an ASD, 55% another developmental disorder, and 25% false positives (Pierce et al. 2011). While sensitivity for ASD in particular is unknown at this age, and it remains probable that a 12-month-old screening will not pick up the majority of ASD cases, it is promising as a general screening at 12 months, which would then need to be followed by additional ASD-specific screening.

Developmental Check-In (DCI; Level 1)

The DCI is a novel screening tool designed for use in underserved populations for whom literacy limitations may interfere with their engagement with other screening strategies (Janvier et al. 2019). The DCI presents 28 social, language, and developmental red flags in the form of images with very limited use of text. Caregivers responded to each yes/no item based on the child's behavior. Cutoffs were determined empirically for various age and gender groups; in the total sample, sensitivity was 0.66 and specificity was 0.76. Although there is currently limited empirical support for the DCI, it may be particularly useful in certain underserved groups, including families with limited education, and those whose English proficiency is limited.

Pervasive Developmental Disorders Screening Test, Second Edition (PDDST-II; Levels 1 and 2)

The PDDST-II (Siegel 2004) consists of three stages designed to be administered in different settings by clinicians of increasing knowledge about ASDs. The first stage consists of 22 yes/no items and is intended for use by pediatricians in primary care settings for children 12–48 months. Further evaluation is recommended for children with five or more items answered as “yes.” According to the author, using these criteria, sensitivity is 0.92 and specificity is 0.91 in an “at-risk” sample (Siegel 2004). The second stage of the PDDST-II is a 14-item measure that is intended for use in developmental clinics. The cutoff is still five items in the second stage, with a sensitivity of 0.73 and specificity of 0.49. The third and final stage consists of 12 items and is intended for use in autism clinics. The cutoff for this stage is a score of 8, with sensitivity of 0.58 and specificity of 0.60. Unfortunately, sensitivity and specificity have not yet been reported for the PDDST-II in a large population-based sample.

Modified Checklist for Autism in Toddlers-Revised with Follow-Up (M-CHAT-R/F; Levels 1 and 2)

One of the most commonly used autism screening measures in the United States is the M-CHAT-R/F (Robins et al. 2014), which is nationally

recommended by the AAP. The M-CHAT-R/F consists of two stages of screening. The first stage is a 20-item yes or no parent questionnaire that screens for signs of an ASD in children aged 16–30 months. This is a level 1 screening instrument and is widely used by pediatric offices across the nation and internationally; the M-CHAT has been translated into more than 50 languages. The M-CHAT-R is scored “Low Risk,” if parents respond in a way that indicates risk for ASD on 0–2 items, whereas parent responses are scored “Medium Risk” if they indicate risk for ASD on 3–8 items, or “High Risk” if parent responses indicate risk for ASD on 8–20 items. High-risk children can be referred immediately for evaluation. For medium-risk children, a second stage of screening is a follow-up interview (which can be done on the phone or in person). The interview is completed by following a script, although it involves some clinical judgment. If a child continues to indicate risk on two or more items on the M-CHAT-R/F, it is strongly recommended that the child be referred for early intervention and diagnostic assessment. While the M-CHAT-R/F detects ASD at a higher rate compared to the M-CHAT while also reducing the number of children needing follow-up, additional studies are still under way to get a firmer idea of sensitivity and specificity for this revised screening instrument.

Social Responsiveness Scale: Second Edition (SRS-2; Level 2)

The SRS-2 (Constantino and Gruber 2012) is a 65-item rating scale that measures the severity of autism spectrum symptoms as they occur in natural social settings. The SRS-2 is completed by a parent or teacher in just 15–20 min and provides a clear picture of a child's social impairments, assessing social awareness, social information processing, capacity for reciprocal social communication, social anxiety/avoidance, and autistic preoccupations and traits. It is appropriate for use with individuals aged 2.5 through adulthood. The SRS-2 can be used as a level 2 screener in clinical or educational settings, an aid to clinical diagnosis, or a measure of response to intervention. SRS-2 scores are particularly helpful in identifying the severity of social impairment based on

five treatment subscales: social awareness, social cognition, social communication, social motivation, and restricted interests and repetitive behavior. Raw scores are converted to T-scores for gender and respondent; a total T-score of 60–75 is considered a mild to moderate indication of deficient reciprocal social behavior reflecting autism spectrum disorder, while a T-score of 76 or higher is considered severe and strongly associated with a clinical diagnosis of autism spectrum disorder. For a cutoff raw score of 80, sensitivity is reported as 0.78 and specificity as 0.94 in the SRS-2 manual.

Screening Tool for Autism in 2-Year-Olds (STAT; Level 2)

The STAT (Stone et al. 2000) is an interactive measure that is designed to differentiate between autism and other developmental disorders and is therefore intended for children 24–35 months old already identified as at risk for an ASD, although it has been shown to be useful in samples as young as 14 months of age (Stone et al. 2008). The STAT is administered within the context of play and takes about 20 min to complete. The STAT consists of 12 items that were derived from three measures: the Play Assessment Scale (Fewell 1991), the Prelinguistic Communication Assessment (Stone et al. 1997b), and the Motor Imitation Scale (Stone et al. 1997a). Items assess behaviors in four social-communicative domains that do not require language comprehension: play, requesting, directing attention, and motor imitation. Sensitivity of the STAT is estimated to be 0.92 and specificity is 0.85 (Stone et al. 2004).

Childhood Autism Rating Scale: Second Edition (CARS2; Level 2)

The CARS2 (Schopler et al. 2010) is a behavioral rating scale designed for the identification of autism in children. There are two forms, the Standard Version (CARS2-ST) and High-Functioning version (CARS2-HF); each form consists of 15 items, each rated on a scale of 1–4. The CARS2 is based on a trained rater's clinical judgment of observed behavior and parent report.

A total score is calculated by adding the scores of the individual items. Scores of 30–36.5 on the ST form and 28–33.5 on the HF form suggest mild to moderate autism, and scores of 37–60 on the CARS2-ST form and 34–60 on the CARS2-HF form indicate severe autism. Chlebowski et al. (2010) found that a score of 25.5–30 is consistent with PDD-NOS. The CARS2-ST and CARS2-HF forms appear to be both sensitive and specific to behavioral problems associated with autism.

Gilliam Autism Rating Scale: Third Edition (GARS-3; Level 2)

The GARS-3 (Gilliam 2014) is a symptom severity behavioral checklist that assists teachers, parents, and clinicians in identifying and diagnosing autism in individuals ages 3 through 22. The 56 items on the GARS-3 are based on the definitions of autism adopted by the American Psychological Association and the 2013 *Diagnostic and Statistical Manual of Mental Disorders: Fifth Edition* (DSM-V) and are grouped into 6 subscales: restrictive/repetitive behaviors, social interaction, social communication, emotional responses, cognitive style, and maladaptive speech. Raw scores combine to give standard scores, percentile ranks, severity level, and binary probability of autism called the autism quotient, with higher scores reflecting greater severity of impairment. Using the new cutoff scores, the estimated diagnostic sensitivity and specificity of the GARS-3 are 0.97.

Social Communication Questionnaire (SCQ; Level 2)

The SCQ (Berument et al. 1999) is a 40-item screening questionnaire designed for parents to complete about their child aged 4–40 years, with a mental age of at least 2 years. There are both current and lifetime versions available to detect signs of an ASD in children. A cutoff score of 22 is used to differentiate autism from other ASDs, and a cutoff score of 15 is used to differentiate ASDs from other developmental disorders. Sensitivity of the SCQ is 0.85 and specificity is 0.75.

Future Directions

A controversy about the advisability of universal early ASD screening has been raised by the United States Preventive Services Task Force. They concluded that early screening was sufficiently accurate and that early intervention had demonstrated efficacy in improving outcomes, but concluded that a randomized controlled trial was needed to assess outcomes for children who participated in early universal screening versus those who did not (Siu and US Preventive Services Task Force 2016). This position has been rebutted by many organizations and research teams (Robins et al. 2016; Fein and Baby Sibs Research Consortium 2016; Fein et al. 2017).

Continued research to refine the screening instruments for increased accuracy, including better sensitivity and specificity, in identification of children at risk for ASDs is warranted. Furthermore, additional research regarding the utility of broad developmental screening in detecting children at risk for ASDs is necessary to determine whether an autism-specific screening instrument is necessary. Finally, future research will help clarify the most appropriate time points for autism screening.

See Also

- [Ages and Stages Questionnaire, Second Edition](#)
- [Checklist for Autism in Toddlers \(CHAT\)](#)
- [Childhood Autism Rating Scale](#)
- [Gilliam Autism Rating Scale \(GARS\)](#)
- [Infant/Toddler Checklist](#)
- [Modified Checklist for Autism in Toddlers \(M-CHAT\)](#)
- [Pervasive Developmental Disorders Screening Test \(PDDST\)](#)
- [Screening Tool for Autism in Two-Year-Olds \(STAT\)](#)
- [Social Communication Questionnaire](#)
- [Social Responsiveness Scale](#)

References and Reading

- Baghdadli, A., Russet, F., & Mottron, L. (2017). Measurement properties of screening and diagnostic tools for autism spectrum adults of mean normal intelligence: A systematic review. *European Psychiatry, 44*, 104–124.
- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., . . . Dowling, N. F. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summaries, 67*(6), 1–23.
- Baranek, G. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders, 29*(3), 213–224.
- Baron-Cohen, S., Allen, J., & Gillberg, C. (1992). Can autism be detected at 18 months? The needle, the haystack, and the CHAT. *The British Journal of Psychiatry, 161*, 839–843.
- Baron-Cohen, S., Cox, A., Baird, G., Swettenham, J., Drew, A., Morgan, K., Nightingale, N., & Charman, T. (1996). Psychological markers in the detection of autism in infancy in a large population. *The British Journal of Psychiatry, 168*, 158–163.
- Baron-Cohen, S., Wheelwright, S., Cox, A., et al. (2000). Early identification of autism by the checklist for autism in toddlers (CHAT). *Journal of the Royal Society of Medicine, 93*, 521–525.
- Beacham, C., Reid, M., Bradshaw, J., Lambha, M., Evans, L., Gillespie, S., . . . Richardson, S. S. (2018). Screening for autism spectrum disorder: Profiles of children who are missed. *Journal of Developmental and Behavioral Pediatrics, 39*(9), 673–682.
- Berument, S. K., Rutter, M., Lord, C., Pickles, A., & Bailey, A. (1999). Autism screening questionnaire: Diagnostic validity. *The British Journal of Psychiatry, 175*, 444–451.
- Chawarska, K., Paul, R., Klin, A., Hannigen, S., Dichtel, L. E., & Volkmar, F. (2007). Parental recognition of developmental problems in toddlers with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 37*, 62–72.
- Chlebowski, C., Green, J., Barton, M., & Fein, D. (2010). Using the Childhood Autism Rating Scale to diagnose autism spectrum disorders. *Journal of Autism and Developmental Disorders, 40*(7), 787–799.
- Constantino, J. N., & Gruber, C. P. (2012). *Social Responsiveness Scale* (2nd ed.). Los Angeles: Western Psychological Services.
- Courchesne, E. (2004). Brain development in autism: Early overgrowth followed by premature arrest of growth. *Mental Retardation and Developmental Disabilities Research Reviews, 10*, 106–111.
- Dai, Y. G., Miller, L. E., Ramsey, R. K., Robins, D. L., Fein, D. A., & Dumont-Mathieu, T. (2019).

- Incremental utility of 24-month autism spectrum disorder screening after negative 18-month screening. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03959-5>
- Dietz, C., Swinkels, S., van Daalen, E., van Engeland, H., & Buitelaar, J. K. (2006). Screening for autistic spectrum disorder in children aged 14–15 months. II: Population screening with the early screening of autistic traits questionnaire (ESAT) design and general findings. *Journal of Autism and Developmental Disorders*, 36, 713–722.
- Earls, M. F., & Hay, S. S. (2006). Setting the stage for success: Implementation of developmental and behavioral screening and surveillance in primary care practice – The North Carolina Assuring Better Child Health and Development (ABCD) Project. *Pediatrics*, 118(1), 183–188.
- Fein, D., & Baby Sibs Research Consortium. (2016). Commentary on USPSTF final statement on universal screening for autism. *Journal of Developmental & Behavioral Pediatrics*, 37(7), 573–578.
- Fein, D., Barton, M., & Dumont-Mathieu, T. (2017). Optimizing outcome in autism spectrum disorders. *Policy Insights from the Behavioral and Brain Sciences*, 4(1), 71–78.
- Fewell, R. (1991). *Play Assessment Scale* (5th revision). Unpublished manuscript, University of Miami School of Medicine.
- Filipek, P. A., Accardo, P. J., Ashwal, S., Baranek, G. T., Cook, E. H., Dawson, G., et al. (2000). Practice parameter: Screening and diagnosis of autism: Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Child Neurology Society. *Neurology*, 55(4), 468–479.
- Fombonne, E., Simmons, H., Ford, T., Meltzer, H., & Goodman, R. (2001). Prevalence of pervasive developmental disorders in the British Nationwide Survey of Child Mental Health. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40, 820–827.
- Gilliam, J. E. (2014). *Gilliam Autism Rating Scale* (3rd ed.). Austin: PRO-ED.
- Glascoe, F. (2000). Early detection of developmental and behavioral problems. *Pediatric Review*, 21(8), 272–280.
- Glascoe, F. (2001). *Parents' evaluation of developmental status*. Nashville: Ellsworth and Vandermeer, LLC.
- Glascoe, F. (2005). Screening for developmental and behavioral problems. *Mental Retardation and Developmental Disabilities Research Reviews*, 11(3), 173–179.
- Gray, K., Tonge, B., & Brereton, A. (2006). Screening for autism in infants, children, and adolescents. *International Review of Research in Mental Retardation*, 32, 197–227.
- Hardy, S., Haisley, L., Manning, C., & Fein, D. (2015). Can screening with the ages and stages questionnaire detect autism? *Journal of Developmental and Behavioral Pediatrics*, 36(7), 536–543.
- Herlihy, L. E., Brooks, B., Dumont-Mathieu, T., Barton, M. L., Fein, D., Chen, C. M., & Robins, D. L. (2014). Standardized screening facilitates timely diagnosis of autism spectrum disorder in a diverse sample of low-risk toddlers. *Journal of Developmental and Behavioral Pediatrics*, 35, 85–92.
- Hinnebusch, A. J., Miller, L. E., & Fein, D. A. (2017). Autism spectrum disorders and low mental age: Diagnostic stability and developmental outcomes in early childhood. *Journal of Autism & Developmental Disorders*, 47, 3967–3982.
- Hirota, T., So, R., Kim, Y. S., Leventhal, B., & Epstein, R. A. (2018). A systematic review of screening tools in non-young children and adults for autism spectrum disorder. *Research in Developmental Disabilities*, 80 (May), 1–12.
- Howlin, P., & Asgharian, A. (1999). The diagnosis of autism and Asperger's syndrome: Findings from a survey of 770 families. *Developmental Medicine and Child Neurology*, 41, 834–839.
- Janvier, Y. M., Coffield, C. N., Harris, J. F., Mandell, D. S., & Cidav, Z. (2019). The Developmental Check-In: Development and initial testing of an autism screening tool targeting young children from underserved communities. *Autism*, 23(3), 689–698.
- Johnson, C. P., & Myers, S. M. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120(5), 1183–1215.
- Lipkin, P. H., Macias, M. M., & Council on Children with Disabilities Section on Developmental and Behavioral Pediatrics. (2020). Promoting optimal development: Identifying infants and young children with developmental disorders through developmental surveillance and screening. *Pediatrics*, 145(1), 1–19.
- MacDonald, R., Parry-Cruwys, D., Dupere, S., & Ahearn, W. (2014). Assessing progress and outcome of early intensive behavioral intervention for toddlers with autism. *Research in Developmental Disabilities*, 35(12), 3632–3644.
- Macy, M. (2012). The evidence behind developmental screening instruments. *Infants and Young Children*, 25(1), 19–61.
- Mandell, D., Listerud, J., Levy, S., & Pinto-Martin, J. (2002). Race differences in the age of diagnosis among Medicaid-eligible children with autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(12), 1447–1453.
- Marlow, M., Servili, C., & Tomlinson, M. (2019). A review of screening tools for the identification of autism spectrum disorders and developmental delay in infants and young children: Recommendations for use in low- and middle-income countries. *Autism Research*, 12(2), 176–199.
- Øien, R. A., Candpsych, S. S., Volkmar, F. R., Shic, F., Cicchetti, D. V., Nordahl-Hansen, A., . . . Chawarska, K. (2018). Clinical features of children with autism who passed 18-month screening. *Pediatrics*, 141(6), 1–10.

- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders*, 24(3), 247–257.
- Perrin, E. C., Sheldrick, C., Visco, Z., & Mattern, K. (2016). The survey of well-being of young children (swyc) user's manual. Boston, MA: Floating Hospital for Children at Tufts Medical Center.
- Pierce, K., Carter, C., Weinfeld, M., Desmond, J., Hazin, R., Bjork, R., & Gallagher, N. (2011). Detecting, studying and treating autism early: The one year well-baby check-up approach. *Journal of Pediatrics*, 159(3), 458–465. Accessed 29 April 2011.
- Pierce, K., Gazestani, V. H., Bacon, E., Barnes, C. C., Cha, D., Nalabolu, S., . . . Courchesne, E. (2019). Evaluation of the diagnostic stability of the early autism spectrum disorder phenotype in the general population starting at 12 months. *JAMA Pediatrics*, 173(6), 578.
- Pinto-Martin, J. A., Dunkle, M., Earls, M., Fliedner, D., & Landes, C. (2005). Understanding and addressing the challenges of disability: Developmental stages of developmental screening: Steps to implementation of a successful program. *American Journal of Public Health*, 95(11), 1928–1932.
- Robins, D. L. (2019). How do we determine the utility of screening tools? *Autism*. <https://doi.org/10.1177/136236131989417>.
- Robins, D. L., Casagrande, K., Barton, M., Chen, C. M., Dumont-Mathieu, T., & Fein, D. (2014). Validation of the modified checklist for autism in toddlers, revised with follow-up (M-CHAT-R/F). *Pediatrics*, 133(1), 37–45.
- Robins, D. L., Adamson, L. B., Barton, M., Connell, J. E., Dumont-Mathieu, T., Dworkin, P. H., Fein, D., Greenstein, M. A., Hsu, H., Kerns, C., Newschaffer, C., Plumb, J., Shattuck, P., Turchi, R., & Vivanti, G. (2016). Universal autism screening for toddlers: Recommendations at odds. *Journal of Autism and Developmental Disorders*, 46(5), 1880–1882.
- Rogers, S. J., Vismara, L., Wagner, A. L., McCormick, C., Young, G., & Ozonoff, S. (2014). Autism treatment in the first year of life: A pilot study of infant start, a parent-implemented intervention for symptomatic infants. *Journal of Autism and Developmental Disorders*, 44(12), 2981–2995.
- Sánchez-García, A. B., Galindo-Villardón, P., Nieto-Librero, A. B., Martín-Rodero, H., & Robins, D. L. (2019). Toddler screening for autism spectrum disorder: A meta-analysis of diagnostic accuracy. *Journal of Autism and Developmental Disorders*, 49(5), 1837–1852.
- Schopler, E., Van Bourgondien, M. E., Wellman, G. J., & Love, S. R. (2010). *The Childhood Autism Rating Scale* (2nd ed.). Los Angeles: Western Psychological Services.
- Siegel, B. (2004). *Pervasive developmental disorders screening test-II (PDDST-II): Early childhood screeners for autistic spectrum disorders*. San Antonio: Harcourt Assessment.
- Siu, A. L., & US Preventive Services Task Force. (2016). Screening for autism Spectrum disorder in young children, US Preventive Services Task Force recommendation statement. *Journal of the American Medical Association*, 315(7), 691–696.
- Squires, J., Bricker, D., & Potter, L. (2009). *Ages & stages questionnaires, third edition (ASQ-3) user's guide*. Baltimore: Paul H. Brookes Publishing.
- Stone, W. L., Ousley, O. Y., & Littleford, C. (1997a). Motor imitation in young children with autism: What's the object? *Journal of Abnormal Child Psychology*, 25, 475–485.
- Stone, W. L., Ousley, O. Y., Yoder, P. J., Hogan, K. L., & Hepburn, S. L. (1997b). Nonverbal communication in 2- and 3-year old children with autism. *Journal of Autism and Developmental Disorders*, 27, 677–696.
- Stone, W., Coonrod, E., & Ousley, O. (2000). Brief report: Screening tool for autism in two-year olds (STAT): Development and preliminary data. *Journal of Autism and Developmental Disorders*, 30, 607–612.
- Stone, W. L., Coonrod, E. E., Turner, L. M., & Pozdol, S. L. (2004). Psychometric properties of the STAT for early autism screening. *Journal of Autism and Developmental Disorders*, 34(6), 691–701.
- Stone, W. L., McMahon, C. R., & Henderson, L. M. (2008). Use of the screening tool for autism in two-year-olds (STAT) for children under 24 months: An exploratory study. *Autism*, 12(5), 557–573.
- Swinkels, S. H., Dietz, C., van Daalen, E., Kerkhof, I. H., van Engeland, H., & Buitelaar, J. K. (2006). Screening for autistic spectrum in children aged 14–15 months. I: The development of the early screening of autistic traits questionnaire (ESAT). *Journal of Autism and Developmental Disorders*, 36, 723–732.
- Tonge, B., Brereton, A., Kiomall, A., Mackinnon, A., King, N., & Rinehart, N. (2006). Effects on parental mental health of an education and skills training program for parents and young children with autism: A randomized controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45(5), 561–569.
- Towle, P. O., & Patrick, P. A. (2016). Autism spectrum disorder screening instruments for very young children: A systematic review. *Autism Research and Treatment*, 2016, 1–29.
- Wetherby, A., Allen, L., Cleary, J., & Kublin, K. (2002). Validity and reliability of the communication and symbolic behavior scales developmental profile with very young children. *Journal of Speech, Language, and Hearing Research*, 45, 1202–1218.
- Wiggins, L. D., Baio, J., & Rice, C. (2006). Examination of the time between first evaluation and first autism spectrum diagnosis in a population-based sample. *Journal of Developmental and Behavioral Pediatrics*, 27, 79–87.
- Wiggins, L., Piazza, V., & Robins, D. (2014). Comparison of a broad-based screen versus disorder-specific screen in detecting young children with an autism spectrum

disorder. *Autism: The International Journal of Research and Practice*, 18(2), 76–84.

Williams, J., & Brayne, C. (2006). Screening for autism spectrum disorders: What is the evidence? *Autism*, 10(1), 11–35.

Zwaigenbaum, L., Thurm, A., Stone, W., Baranek, G., Bryson, S., Iverson, J., Kau, A., Klin, A., Lord, C., Landa, R., Rogers, S., & Sigman, M. (2007). Studying the emergence of autism spectrum disorders in high-risk infants: Methodological and practical issues. *Journal of Autism and Developmental Disorders*, 37(3), 466–480.

Screening of Autism Spectrum Disorders in Older Adults

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Definition

HAP: The hetero-anamnestic personality questionnaire can be used as a screener for ASD features in older adults.

People on the autism spectrum experience more psychiatric problems than neurotypical people. When they turn to mental health care facilities for help, it is important to detect (features of) an autism spectrum disorder (ASD) to provide suitable care. In the field of elderly care or geriatric psychiatry however, there is a lack of knowledge about how to detect and diagnose ASD and how to care for autistic older adults. Detecting ASD (features) in an early stage of diagnosis and treatment is of great benefit to autistic older adults and their families. It is important that they get appropriate care that takes into account age-related factors. However, there is a lack of research on the topic, even though the subject is getting more attention and there is gradually more awareness and understanding about ASD and aging.

ASD in older adults can easily be overlooked. First, older adults and their families often seek help because of other complaints, such as depression or anxiety. Second, the features of ASD might be different in older adults, for example, as a result of a lifetime of social camouflaging. It

can take long before a diagnosis of ASD is confirmed and appropriate care is provided.

To help clinicians detect these features and be able to screen for ASD in older adults, a study was conducted with the Hetero-Anamnestic Personality Questionnaire (HAP): an informant-based personality questionnaire, developed and validated for older adults. This measure has several advantages. First, it can be administered as a general screener. It is aimed at personality traits, and research has shown that autistic people score differently on certain scales of various personality questionnaires. Even if a clinician initially does not consider ASD, the results can still indicate in that direction. Second, because it is informant based, it is not dependent on co-occurring psychiatric or cognitive impairments of the older adult and can therefore be administered at the beginning of diagnosis and treatment.

A group of older adults (age > 60) with ($N = 40$) and without ($N = 43$) ASD, all receiving psychiatric or psychological care because of various psychiatric or cognitive impairments (with the exception of personality disorders), were compared on the scores of the HAP. The ASD group had significantly higher scores, with large effect sizes, on the HAP scales: “Socially avoidant behavior,” “Rigid behavior,” and “Unpredictable and impulsive behavior.” These scales were also capable of discriminating between persons with and without ASD. Cutoff points were determined at 5 for “Socially avoidant behavior” and 4 for “Rigid behavior” and “Unpredictable and impulsive behavior.”

Further research is needed to determine whether the HAP can also discriminate between autistic people and people with a personality disorder. Because the study was executed in the Netherlands, in a small population (albeit with sufficient statistical power for this study), with no additional data on severity of ASD or severity of co-occurring psychiatric symptoms, further research is also needed to determine whether these results are replicable and to obtain more knowledge on the subject.

Nevertheless, the HAP turns out to have feasibility in care settings and mental health institutions as a screener for ASD in older adults, even if there are co-occurring conditions. It can be

administered at the beginning of a diagnostic process when ASD is not even considered as a diagnosis. This can save time in starting up appropriate care for older autistic adults and their families.

See Also

- ▶ [Accuracy of the ADOS-2 in Identifying Autism Among Adults with Complex Psychiatric Conditions, The](#)
- ▶ [Autism Behavior Checklist](#)
- ▶ [Autism Diagnostic Interview-Revised](#)
- ▶ [Autism Diagnostic Observation Schedule](#)
- ▶ [Autism Family Experience Questionnaire \(AFEQ\), The](#)
- ▶ [Behavior Observation Scale](#)
- ▶ [Behavior Rating Scale \(BRS\)](#)
- ▶ [Camouflaging Autistic Traits Questionnaire \(CAT-Q\)](#)
- ▶ [Diagnosis and Classification](#)
- ▶ [Diagnostic Interview for Social and Communication Disorders](#)
- ▶ [Gilliam Autism Rating Scale \(GARS\)](#)
- ▶ [Netherlands and Autism, The](#)
- ▶ [Personality, Clinical Assessment](#)
- ▶ [Psychiatric Problems and Care in Older Adults with Autism](#)
- ▶ [Screening Measures](#)
- ▶ [Self-Report Autism Scales for Adults](#)

References and Reading

- Barendse, H. P. J., & Thissen, A. J. C. (2006). *Manual of the hetero-anamnestic personality questionnaire (HAP)*. Schijndel: Barendse & Thissen.
- Heijnen-Kohl, S. M. J., Kok, R. M., Wilting, R. M. H. J., Rossi, G., & van Alphen, S. P. J. (2017). Screening of autism spectrum disorders in geriatric psychiatry. *Journal of Autism and Developmental Disorders*, 47(9), 2679–2689.
- Hull, L., Petrides, K. V., Allison, C., et al. (2017). “Putting on my best normal”: Social camouflaging in adults with autism spectrum conditions. *Journal of Autism and Developmental Disorders*, 47(8), 2519–2534.
- Michael, C. (2016). Why we need research about autism and ageing. *Autism*, 20(5), 515–516.
- Wright, S. D. (Ed.). (2016). *Autism spectrum disorder in mid and later life*. Philadelphia: Jessica Kingsley Publishers.

Screening Test for Auditory Processing Disorders

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Synonyms

SCAN-A (adults); SCAN-C (children)

Description

The Screening Test for Auditory Disorders (SCAN) is a screening tool used to determine if a child (or adult) should be evaluated for central auditory processing (CAP) disorders. The SCAN-C is a CAP screening instrument for children (Keith 1986, 2000a, b), while the SCAN-A is used for adolescents and adults (Keith 1994). The revised SCAN-C was designed for children from 5 to 11 years of age and consists of four subtests: Filtered Words, Auditory Figure Ground (speech-in-noise), Competing Words, and Competing Sentences (Keith 2000a, b). The Filtered Words subtest consists of 1,000-Hz low-pass filtered monosyllabic words with a filter roll-off of 32 dB/octave. The listener must repeat the word she/he perceives. The Auditory Figure Ground subtest consists of monosyllabic words recorded at +8 signal-to-noise ratio with a multi-talker babble background. The listener must repeat the stimulus words in the presence of the background noise. The Competing Words subtest consists of monosyllabic word pairs presented to both ears (dichotically) with simultaneous onset times (Keith et al. 1989). As a directed listening task, the listener is first asked to repeat both words that are perceived, repeating the word heard in the right ear first. Then a second test list is given and the listener must repeat both words that are perceived, repeating the word heard in the left ear first. The Competing Sentences subtest consists of sentence pairs that are unrelated in topic which are presented to both ears (dichotically) with

simultaneous onset times (Keith 2000b). The listener is asked to repeat only the sentence heard in the right ear. Then a second test list is given and the listener is asked to repeat only the sentence heard in the left ear. These subtests are pre-recorded and can be administered under headphones in a quiet room. The raw scores on each of the SCAN subtests are converted to standard scores, percentile ranks, and confidence intervals. The screening is easy to administer and score. Total test time is approximately 20 min.

Historical Background

The SCAN was developed to be a screening test for CAP in children and was first introduced in 1986. The goal of this screening test was to help assess auditory maturation and identify children who may benefit from more involved CAP testing and specific auditory rehabilitation. The original SCAN-C was developed for children between the ages of 3 and 11 years and included three subtests: Filtered Words, Auditory Figure Ground, and Competing Words. Keith et al. (1989) demonstrated that the SCAN Competing Words subtest was highly correlated with other tests of dichotic listening (Staggered Spondaic Words (SSW) and Competing Sentences Test (CST)). These researchers also demonstrated that language abilities may affect performance on the SCAN. Subsequent research indicated that the original SCAN-C test-retest reliability may have been poor, with children performing better on the second administration of the screener than the first administration (Amos and Humes 1998). In addition, researchers showed that children performed more poorly in a quiet room compared to an audiologic booth (Emerson et al. 1997). These, along with other factors, led to the development of the revised SCAN-C which included an additional subtest (Competing Sentences), new test instructions to make them easier for young children to understand, test materials recorded on compact discs rather than audiocassettes, and fewer word pairs for the Competing Words subtest to make the

test more efficient. Research completed by Keith (2000b) indicated improved test-retest reliability and no significant differences in performance between test environments (quiet room vs. audiologic booth).

Psychometric Data

Normative data exist on a wide age range (5–11 years) with a total sample size of 650 children. Normative data indicate a systematic improvement in performance with increase in age. Test-retest reliability was high for children between the ages of 5 and 7 years. Children included in the normative sample were all able to take the test in English without test modifications, had normal and symmetric hearing from the octave frequencies between 500 and 4,000 Hz, and had intelligible speech with few articulation errors. Children were excluded from the standardization sample if they were receiving special education services or services related to gifted and talented programs (Keith 2000b).

Like other tests of central auditory processing, early research completed on the SCAN included children who spoke English as their primary language, were in regular education classrooms (without special education services), and demonstrated normal hearing and normal middle ear function (Amos and Humes 1998; Emerson et al. 1997; Keith 2000b; Keith et al. 1989). Furthermore, it should be noted that researchers have found that children with language problems might score poorly on the SCAN (Keith et al. 1989). This is likely due to the fact that the test stimuli on the SCAN have a high linguistic load.

Clinical Uses

The SCAN-C is often used in school systems as well as audiological practices to determine if a more thorough CAP evaluation is warranted. It is often administered by a speech-language pathologist in a quiet room in the school setting,

and referrals are made to audiologists for further CAP testing as necessary. Clinically, the person administering the SCAN should take into consideration the cognitive, hearing, speech, and language abilities of the individual being tested as these factors may affect screening outcomes. Thus, the SCAN-C is not typically administered to children with autism, and performance on this screener should be interpreted with caution in this population.

References and Reading

- Amos, N., & Humes, L. (1998). SCAN test-retest reliability for first and third-grade children. *Journal of Speech, Language, and Hearing Research*, 41, 834–845.
- Bellis, T. (1996). *Assessment and management of Central Auditory Processing Disorders in the educational setting: From science to practice*. San Diego: Singular Publishing Group.
- Emerson, M., Crandall, K., Seikel, J., & Chermak, G. (1997). Observations on the use of the SCAN to identify children at risk for central auditory processing disorder. *Language, Speech, and Hearing Services in Schools*, 28, 43–49.
- Keith, R. (1986). *SCAN: A screening test for auditory processing disorders*. San Diego: The Psychological Corporation.
- Keith, R. (1994). *SCAN-A: A test for auditory processing disorders in adolescence and adults*. San Antonio: The Psychological Corporation.
- Keith, R. (2000a). *SCAN-C: Test for auditory processing disorders in children-revised*. San Antonio: The Psychological Corporation.
- Keith, R. (2000b). Development and standardization of SCAN-C Test for Auditory Processing Disorders in children. *Journal of the American Academy of Audiology*, 11(8), 438–445.
- Keith, R., Rudy, J., Donahue, P., & Katbamna, B. (1989). Comparison of SCAN results with other auditory and language measures in a clinical population. *Ear and Hearing*, 10(6), 382–386.

Screening Tool for Autism in Toddlers

► Screening Tool for Autism in Two-Year-Olds (STAT)

Screening Tool for Autism in Two-Year-Olds (STAT)

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Synonyms

Screening tool for autism in toddlers; STAT

Description

The STAT is an interactive, play-based screening instrument for autism that contains 12 items tapping the core deficit areas of socialization and communication. Items are organized into four domains: play (two items), requesting (two items), directing attention (four items), and motor imitation (four items). The items selected for inclusion are those that were found to discriminate between 2-year-old children with autism and developmentally matched comparison samples (Stone et al. 2000). Although the STAT was designed for use with children between the ages of 24 months and 36 months, preliminary evidence for its utility with children as young as 14 months has been obtained (Stone et al. 2008), and ongoing research is being conducted to evaluate its use with 3-year-old children.

The STAT was designed for use by a broad range of community service providers working with young children, such as early intervention providers, birth-to-three personnel, preschool teachers, psychologists, speech-language pathologists, and health-care professionals. It can be used in clinical, educational, and home settings. It takes about 20 min to administer and was designed to be scored live. Training on the STAT is available through participation in 1-day workshops (see <http://uwreadilab.com/our-workshops/>) or completion of a web-based tutorial (see <http://vkc.mc.vanderbilt.edu/vkc/triad/training/stat/>).

STAT training workshops have been conducted in countries around the world, and it has been translated into Spanish, Mandarin, and Polish.

The STAT User's Manual contains information about the purpose and use of the STAT, describes its development and psychometric properties, and provides general information, guidelines, and tips regarding the administration and scoring of each item. The STAT Materials Kit contains all of the play materials needed for the screening, such as a ball, toy car and truck, puppet, baby doll, bubbles, balloons, and many others. The STAT Test Protocol contains detailed instructions regarding the number of trials allowed for each item and the specific wording of verbal directions and prompts, along with scoring criteria and examples for each item.

Administration of the STAT involves setting up situations to elicit key social and communication behaviors. The play items on the STAT assess the child's participation in a turn-taking game and his/her functional play with a doll. The requesting items involve presenting desired objects in closed containers and observing whether and how the child asks for help or more, either verbally or nonverbally. The directing attention items involve creating unexpected events and observing whether and how the child shares interest or enjoyment in the event with the examiner. The imitation items assess the extent to which the child attempts or succeeds in imitating simple actions demonstrated by the examiner. Multiple trials can be given for most items, and the child's item score reflects his/her best performance on the item. Specific scoring criteria are used to determine whether the child's performance on each item is a pass, fail, or refuse. The average number of fails for each of the four domains is summed and compared to a cutoff score to identify the child's risk for autism.

Administration of the STAT was designed to be developmentally sensitive and to optimize the child's interest and attention. Language demands are minimal. Although instructions, prompts, and materials are standardized, the sequence in which items are administered can be adapted to the needs of the child. Likewise, items can be administered on the floor or at a table as needed to engage the child and elicit his/her best performance.

Historical Background

Development of the STAT began in the mid-1990s, after a growing empirical literature at the time indicated that (a) autism could be diagnosed accurately at age 2 and (b) behaviors in the social and communication domains were the most prominent characteristics at that age. It was developed at Vanderbilt University by Wendy Stone, PhD, a professor of pediatrics, and Opal Ousley, PhD, who was a psychology graduate student at the time. The purpose of the STAT was to facilitate the early identification of autism by directing children in need of autism-specialized services to assessment centers with the necessary experience and expertise to diagnose autism at young ages. The STAT was designed as a second-stage screener for use in referral (vs. primary care) settings. That is, the purpose of the STAT was to identify children at specific risk for autism from more heterogeneous groups of children for whom there were developmental concerns.

The first STAT Training Workshop was conducted in 1999, and the first published study describing the STAT appeared in 2000 (Stone et al. 2000). Subsequent publications have provided additional information about its psychometric properties for 2-year-olds (Stone et al. 2004) and for children under 24 months (Stone et al. 2008). The web-based STAT Training Tutorial (Kobak et al. 2011) became available in 2009.

The development and refinement of the STAT is ongoing. Current projects are examining its utility for screening in 3-year-olds, the use of a continuous scoring system for each item, and the inclusion of ratings for social engagement and atypical behaviors.

Psychometric Data

Use of the STAT with 2-Year-Olds

The psychometric properties of the STAT were reported in 2004 (Stone et al. 2004). The results, described below, demonstrate strong psychometrics with respect to screening properties, test-retest reliability, interobserver agreement, and concurrent validity with the ADOS.

Screening Properties. The screening properties of the STAT (i.e., sensitivity, specificity, positive predictive value [PPV], and negative predictive value [NPV]) were evaluated on a sample of 52 children, 26 with autism and 26 with developmental delay and/or language impairment (DD/LI). Participants were referred for diagnostic evaluations from a variety of clinical settings between 1997 and 2000. Eligibility criteria were the following: chronological age between 24 and 36 months (i.e., between 2 years, 0 months, 0 days and 2 years, 11 months, 29 days), absence of severe sensory or motor impairments, and absence of identified genetic disorders. All data were collected during the course of the child's diagnostic evaluation, which was conducted by a team of clinicians that included a licensed psychologist and a licensed speech-language pathologist. Autism diagnoses were made by the team psychologist, based on criteria provided in DSM-IV (APA 1994). The STAT was administered by trained examiners who were independent from the diagnostic team and were blind to the results of the diagnostic evaluation. Likewise, the team clinicians were blind to the results of the STAT screening.

Development and validation samples were created to include equal numbers of children with autism and children with DD/LI. There were no diagnostic group differences for chronological age, mental age, or maternal level of education in the development or validation sample. Signal detection using the development sample identified a score of 2 or higher as the optimal cutoff for autism risk. Applying this cutoff score to the validation sample resulted in a sensitivity of 0.92, specificity of 0.85, PPV of 0.86, and NPV of 0.92, providing strong support for its screening properties using this scoring algorithm.

Concurrent validity was assessed by comparing STAT risk category (at risk vs. not at risk) with diagnostic classification (autism vs. nonspectrum) on the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 2000) in a sample of 82 children. The STAT and ADOS were administered by different examiners who were blind to the results of the other's evaluation. Cohen's kappa for agreement between STAT risk category and

ADOS classification was 0.95. Only two children were identified incorrectly by the STAT; both were identified as high risk by the STAT but did not meet ADOS criteria for autism (i.e., false positives).

Interobserver agreement for STAT risk category was evaluated by having two examiners score the STAT independently for 29 children (14 with autism, 2 with PDD-NOS, and 13 with DD/LI). Interobserver agreement was 1.00 using Cohen's kappa.

Test-retest reliability for STAT risk category was examined by having 21 children (nine with autism, six with PDD-NOS, and six with DD/LI) participate in two STAT screenings approximately 2 weeks apart (mean delay = 20 days, range = 4–44 days). Different examiners administered the STAT at each time point to reduce any potential familiarity bias. Test-retest reliability was 0.90 using Cohen's kappa.

Use of the STAT with Children Under 2 Years Old

A preliminary investigation of the utility of the STAT for children under 24 months of age was published in 2008 (Stone et al. 2008). This study included 71 children (44 male, 27 female) who received the STAT between 12 and 23 months of age and a follow-up diagnostic evaluation after 24 months. The average length of time between the initial and follow-up visits was 15 months. All children were at elevated risk for autism by virtue of having an older sibling with an autism spectrum diagnosis ($n = 59$) or being referred for evaluation for concerns about autism ($n = 12$).

The STAT was administered and scored in the same manner as for the 2-year-old samples (Stone et al. 2004), without any procedural or scoring changes. Signal detection analysis with the entire sample revealed that the STAT cutoff score of 2 demonstrated high sensitivity (1.0) but low specificity (0.40) for this age group. The optimal cutoff score for this young sample was 2.75; this cutoff yielded a sensitivity of 0.95, specificity of 0.73, PPV of 0.56, and NPV of 0.97. However, it was also observed that false positives were highest (38%) for the 12–13-month-old age group. When the sample was limited to children

14 months and older, STAT screening properties for a 2.75 cutoff improved to a sensitivity of 0.93, specificity of 0.83, PPV of 0.68, and NPV of 0.97. These results provide preliminary support for the use of the STAT in children between 14 months and 23 months of age, using a cutoff score of 2.75. Replication with an independent sample is currently under way.

Clinical and Community Use

The STAT can be used by a variety of service providers in a diverse array of settings to facilitate early identification and early intervention for children with autism. A “screen-positive” result on the STAT can indicate the need for referral to professionals who have specialized training and experience working with young children with autism. For example, in state birth-to-three systems, service providers or coordinators can screen children with the STAT to identify those at risk for autism, so that timely referrals can be made to diagnosticians and interventionists with specialized experience and training. In community-based intervention settings, such as speech-language clinics, clinicians can use the STAT to screen children on their caseload to determine their need for further diagnostic evaluation for autism. In diagnostic assessment settings, children on the waiting list can be screened with the STAT so that they can be routed to the clinicians with the most experience and training in assessment and early diagnosis of autism.

Several community-based research projects have examined the utility of the STAT as a component of a larger assessment battery focused on early autism detection of autism. For example, the STAT is the centerpiece of a program designed to train pediatricians in community practice settings how to conduct autism assessments (i.e., the STAT-MD program). In this program, pediatricians learn how to administer and score the STAT, as well as how to conduct developmentally sensitive parent interviews and use incidental observations to form diagnostic impressions within a DSM-based framework (Swanson et al. 2014; Warren et al. 2009). More recently, the

STAT has been integrated into Project ECHO, which provides training to primary care providers through a technology-based mentorship model. Participants in ECHO Autism STAT receive training in the screening and diagnosis of children at highest risk for autism (Mazurek et al. 2019).

The STAT has also been used in conjunction with Level 1 (i.e., population-based) screening tools to identify children who may benefit from a more in-depth autism evaluation. One study of this two-tiered screening model in pediatric practices found evidence supporting the combined use of the M-CHAT and the STAT for reducing false positive results (Khowaja et al. 2018). A 2-tiered screening model was also used in a statewide early intervention system in South Carolina, where children who screened positive on general developmental screens received the STAT (Rotholz et al. 2017). Results revealed that this approach led to increased detection and earlier access to behavioral intervention for young children with autism. The potential use of a two-threshold STAT scoring system, which identifies children at highest and lowest autism risk, was demonstrated within an early intervention system (Roberts et al. 2019). In addition, the STAT was found to be suitable for use by community providers as a second-stage case confirmation tool for epidemiological risk factor research in prospective cohorts (Newschaffer et al. 2017).

In addition to providing a cutoff score to identify autism risk, the interactive nature of the STAT affords the opportunity for other clinical uses. The STAT provides a standard setting for obtaining a rich sample of behaviors that represent core social and communicative deficits in autism. During a 20-min STAT screening, clinicians can obtain important information about the child’s strengths and needs in the areas of object play, turn-taking, communication, and imitation. This information can then be used to identify specific intervention goals and to design targeted teaching activities. For example, special educators have used the STAT to prepare preliminary educational plans for children who will be entering their school system at age 3. Similarly, children’s performance on the STAT can be used to formulate and initiate

individualized teaching activities during the often-long waits between the suspicion of autism and the child's formal diagnostic evaluation. A workbook for this purpose has recently been developed.

Research projects have also capitalized on the standardized yet brief behavioral sample afforded by the STAT. For example, the STAT Total and Directing Attention scores have been found to differentiate between groups of toddlers at high and low risk for autism (Stone et al. 2007). The Imitation domain score was used in a study examining predictors of language development in toddlers at elevated familial risk for autism (Edmunds et al. 2017). The STAT has also been used successfully as a context for coding social-communicative behaviors such as initiating joint attention, positive affect sharing, and repetitive behavior (McDuffie et al. 2005; Yoder et al. 2009). In addition, the STAT has been used to screen children under 24 months for eligibility for participation in research projects (Carter et al. 2011).

See Also

- Early Diagnosis
- Screening Measures
- Social Communication

References and Reading

- American Psychiatric Association (APA). (1994). *Diagnostic and statistical manual of mental disorders: DSM-IV*. Washington, DC: American Psychiatric Association.
- Carter, A. S., Messinger, D. S., Stone, W. L., Celimli, S., Nahmias, A. S., & Yoder, P. (2011). A randomized trial of more than words in toddlers with early autism symptoms. *Journal of Child Psychology and Psychiatry*, 52, 741–752.
- Edmunds, S. R., Ibanez, L. V., Warren, Z., Messinger, D. S., & Stone, W. L. (2017). Longitudinal prediction of language emergence in infants at high and low risk for ASD. *Development and Psychopathology*, 29, 319–329.
- Khowaja, M., Robins, D. L., & Adamson, L. B. (2018). Utilizing two-tiered screening for early detection of autism spectrum disorder. *Autism*, 22(7), 881–890.
- Kobak, K. A., Stone, W. L., Ousley, O. Y., & Swanson, A. R. (2011). Web-based training in early autism screening: Results from a pilot study. *Telemedicine and e-Health*, 17, 640–644.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Leventhal, B. L., DiLavore, P. C., Pickles, A., & Rutter, M. (2000). The autism diagnostic observation schedule-generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30(3), 205–223.
- Mazurek, M. O., Curran, A., Burnette, C., & Sohl, K. (2019). ECHO autism STAT: Accelerating early access to autism diagnosis. *Journal of Autism and Developmental Disorders*, 49(1), 127–137.
- McDuffie, A., Yoder, P. J., & Stone, W. L. (2005). Prelinguistic predictors of vocabulary in young children with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, 48, 1080–1097.
- Newschaffer, C. J., Schriver, E., Berrigan, L., Landa, R., Stone, W. L., Bishop, S., Burkom, D., Golden, A., Ibanez, L., Kuo, A., Lakes, K. D., Messinger, D. S., Paterson, S., & Warren, Z. E. (2017). Development and validation of a streamlined autism case confirmation approach for use in epidemiological risk factor research. *Autism Research*, 10, 485–501.
- Roberts, M. Y., Stern, Y., Hampton, J. H., Grauzer, J. M., Miller, A., Levin, A., Kornfield, B., Davis, M. M., Kaat, A., & Estabrook, R. (2019). Beyond pass-fail: Examining the potential utility of two thresholds in the autism screening process. *Autism Research*, 12(1), 112–122.
- Rotholz, D. A., Kinsman, A. M., Lacy, K. K., & Charles, J. (2017). Improving early identification and intervention for children at risk for autism spectrum disorder. *Pediatrics*, 139, e20161061. Advance online publication.
- Stone, W. L., Coonrod, E. E., & Ousley, O. Y. (2000). Brief report: Screening tool for autism in two-year-olds (STAT): Development and preliminary data. *Journal of Autism and Developmental Disorders*, 30, 607–612.
- Stone, W. L., Coonrod, E. E., Turner, L. M., & Pozdol, S. L. (2004). Psychometric properties of the STAT for early autism screening. *Journal of Autism and Developmental Disorders*, 34, 691–701.
- Stone, W. L., McMahon, C. R., Yoder, P. J., & Walden, T. A. (2007). Early social-communicative and cognitive development of younger siblings of children with autism spectrum disorders. *Archives of Pediatrics & Adolescent Medicine*, 161, 384–390.
- Stone, W. L., McMahon, C., & Henderson, L. M. (2008). Use of the screening tool for autism in two-year-olds (STAT) for children under 24 months: An exploratory study. *Autism*, 12, 573–589.
- Swanson, A., Warren, Z., Stone, W. L., Vehorn, A., Dohrmann, E., & Humberd, Q. (2014). The diagnosis of autism in community practice settings: Does advanced training facilitate practice change? *Autism*, 18, 555–561.

- Warren, Z., Stone, W. L., & Humberd, Q. (2009). A training model for the diagnosis of autism in community pediatric practice. *Journal of Developmental and Behavioral Pediatrics, 30*, 442–446.
- Yoder, P. J., Stone, W. L., Walden, T. A., & Malesa, E. E. (2009). Predicting social impairment in younger siblings of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 39*, 1381–1391.

Scripting

- [Echolalia](#)
- [Movie Talk](#)

Scripts

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Synonyms

[Social scripts](#)

Definition

Scripts are written, audio, and/or pictorial examples of phrases or sentences that children with disabilities can use in specific social or academic situations. The individual is explicitly taught the script through modeling, prompting, and reinforcement and then prompted to use the script in the specific situation for which it was developed. Following successful use of the script, a script-fading procedure should be implemented to fade the use of the script over time. Scripts can be used to increase social initiations (e.g., “what do you want to play?”), provide suggestions to improve conversational skills (e.g., “my favorite movie is”), or request assistance (e.g., “help please”).

The use of scripts has improved the communication and social interaction skills that are prevalent in individuals with autism spectrum disorder (ASD; Ganz et al. 2008). Individuals with ASD benefit from the use of scripts in both academic and nonacademic settings, including home, workplace, and community. Scripts have been shown to be effective with children who have minimal language and reading skills (Krantz and McClannahan 1998), for nonreaders using an audio taped script and script-fading procedure (Stevenson et al. 2000), as well as for those with extensive verbal skills, but poor social skills (Krantz and McClannahan 1993). Social scripts can reduce the stress associated with social interactions and assist the child by helping them understand the perspective of others and by providing them with age-appropriate social language. Including informal language, slang, or child-specific terms in the social script may help the conversational exchange appear more natural (Kamps et al. 2002). Although script-fading procedures have demonstrated an increase in the number of unscripted social interactions emitted by research participants (Krantz and McClannahan 1993, 1998; Stevenson et al. 2000; Wichnick et al. 2010), few studies have demonstrated stimulus generalization until recently. Brown et al. (2008) demonstrated a systematic increase in unscripted interactions, and these interactions generalized to untrained stimuli. Further, Wichnick-Gillis et al. (2016) demonstrated the effectiveness of the script-fading procedure in increasing interactions of children with autism towards peers and were able to incorporate cues in the natural environment that served as discriminative stimuli for social interactions. This shift to embedding cues within the natural environment will likely lead to greater generalization when using scripts to promote language and social interaction skills for individuals with autism.

See Also

- [Social Stories](#)

References and Reading

- Brown, J.L., Krantz, P.J., McClannahan, L.E., & Poulson, L. (2008). Using script fading to promote natural environment stimulus control of verbal interactions among youths with autism. *Research in Autism Spectrum Disorders* 2(3), 480–497. <https://doi.org/10.1016/j.rasd.2007.08.006>
- Ganz, J., Kaylor, M., Bourgeois, B., & Hadden, K. (2008). The impact of social scripts and visual cues on verbal communication in three children with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 23, 79–94.
- Kamps, D., Royer, J., Dugan, E., Kravits, T., Gonzalez-Lopez, A., Garcia, G., et al. (2002). Peer training to facilitate social interaction for elementary students with autism and their peers. *Exceptional Children*, 68, 173–187.
- Krantz, P. J., & McClannahan, L. E. (1993). Teaching children with autism to initiate to peers: Effects of a script-fading procedure. *Journal of Applied Behavior Analysis*, 26, 121–132.
- Krantz, P. J., & McClannahan, L. E. (1998). Social interaction skills for children with autism: A script-fading procedure for beginning readers. *Journal of Applied Behavior Analysis*, 31, 191–202.
- Stevenson, C. L., Krantz, P. J., & McClannahan, L. E. (2000). Social interaction skills for children with autism: A script-fading procedure for nonreaders. *Behavioral Intervention*, 15, 1–20.
- Wichnick, A. M., Vener, S. M., & Poulson, C. L. (2010). The effects of a script-fading procedure on unscripted social initiations and novel utterances among young children with autism. *Research in Autism Spectrum Disorders*, 4, 51–64.
- Wichnick-Gillis, A. M., Vener, S. M., & Poulson, C. L. (2016). The effect of a script-fading procedure on social interactions among young children with autism. *Research in Autism Spectrum Disorders*, 26, 1–9.

SCZD15

► SHANK 3

SDA

► Structured Descriptive Assessment

Season of Birth in Autism

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Synonyms

Seasonality of autism

Definition

Seasonal variations in rates of autism might reflect the operation of environmental factors in pathogenesis; however, empirical support for seasonality is decidedly mixed. Landau et al. (1999) carefully investigated eight studies that had served as support for the seasonality hypothesis as an etiological factor in the development of autism. More specifically, the authors of these studies have reported a higher autistic birth rate in spring (March) and in Autumn (August) with a significant deficit of autistic births in the fall and winter months. A detailed examination of each of the eight investigations revealed a number of methodological problems that served to call into question, the validity of these claims of support for the seasonality theory of the genesis of autism.

Major methodological and bio-statistical flaws are noted as were how they were controlled in the Landau et al. investigation:

- Each of the eight studies utilized the general “live population” from which the autistic sample was drawn, thereby potentially biasing the reported results (Fombonne 1989).
- *The author and collaborators utilized ID non-Autistic subjects as a control.*

The comparison population in two of the eight studies defined the general population to contain live births only (i.e., Barak et al. 1995). As noted by Dalen (1975) and Hare (1975), this sampling bias does not take into account infant mortality, which can potentially produce later skewing of the monthly distribution of births

- *The ID non-Autistic control group applies to this methodological flaw as well.*

Relative to the statistical procedures that were employed, the sample sizes in many of the studies were not sufficiently large (e.g., the study by Gillberg 1990).

- *The sample sizes for autism and comparison groups varied markedly.*

As correctly noted by Bartik (1981), the repeated application of the chi-square (d) test without controlling for Type 1 error will lead eventually to confusing statistically significant results with those that have occurred on the basis of chance alone.

- *The earlier study utilized chi-square (d) appropriately as well as introducing a novel statistic that appears very well designed to test the seasonality hypothesis in autism.*

In order for a seasonal finding to be plausible in a biological sense, birth rates should not be restricted to 1 month only; this is because adjacent months would presumably also be affected.

- *This methodological concern was addressed in a twofold manner: first, the seasonality hypothesis was tested by examining the numbers of Autistic births both at the beginning and middle of March and August; and secondly, by determining the highest birth month for each of the study groups.*

In addition, the number of study groups was strongly influenced by the results of research

investigations showing a robust correlation between cognitive impairments and neurobiological deficits among children (Gillberg 1988; Rutter 1983). The implication of these two studies is that children who are more cognitively impaired are more likely to experience a seasonality-of-birth effect. Therefore, in order to test the seasonality hypothesis in autism, it required four study groups: (1) Autistic-Mute (AM); (2) Autistic-Verbal (AV); (3) Intellectual Disability-Mute (IDM); and (4) Intellectual Disability-Verbal (IDV). The prediction was that, in accord with the seasonality hypothesis in autism, the groups would order themselves, for both March and August births as: AM, AV, IDM, and IDV. Put in clinical research terms, the investigators were testing for an ordinal trend with a dichotomous outcome. The statistic that seemed most appropriate was Jonckheere's 1970 test for analyzing ordinal trends with a dichotomous outcome. The authors utilized a computer program designed by Cicchetti et al. (1992). The statistic was utilized in this investigation to study the correlation between the severity of neuropsychological symptomatology to differences in verbal and performance IQ. It was used more recently to examine the relationship between age and the effects of mercury exposure among dentists and controls (Duplinsky and Cicchetti 2012).

Results not only indicated no support for the seasonality hypothesis, some of the findings, somewhat paradoxically, showed results that trended in a direction that was opposite the hypothesis.

More Recent Test of the Seasonality Hypothesis in Autism

Published in 2012, Mazumdar et al. performed another test of the seasonality hypothesis in autism. The main rationale for the study was the argument that date of birth (DOB) is biased, because factors associated with the development of autism have occurred prenatally. As a consequence, the authors therefore argued for utilizing

time of conception, rather than DOB, for testing the seasonality hypothesis. There are four reasons for arguing against this position: The first is a problem of definition. DOB is arguably a very accurate concept. Conception, however, as the authors define it, is not nearly as accurate or valid a measurement. Thus, Mazumdar et al. (2012, p. 4) define it as “The Birth Master File (BMF) also contains the date of the mother’s last menses to which we added a fortnight to obtain an approximate date of conception.”

The second problem is that the authors do not explain why DOB should, of necessity, be considered a biasing factor when whatever occurred prenatally would, at DOB or shortly thereafter, manifest as autism, etiologically. The third problem is that Mazumdar and co-authors also reported that seasonality of autism has been “disappearing” over recent years, in California, where the study took place. The scientific question here is why would this occur if the etiology of the problem was truly related to the genesis of seasonality in autism? It should be stressed that the title of the Mazumdar et al. publication is: *The Disappearing Seasonality of autism Conceptions in California*. In conclusion, the interpretation of the findings of this study appear to be flawed. It should finally be noted that the authors of a very well-designed study in Germany have argued convincingly that research focusing on conception should be prospective rather than retrospective (Gnoth et al. 2003). This is once again, because of the inaccuracy of estimates of precisely when conception has taken place.

See Also

► [Environmental Risk Factors for Autism](#)

References and Reading

Barak, Y., Ring, A., Sulkes, J., & Gabbay, U. (1995). Season of birth and autistic disorder in Israel. *American Journal of Psychiatry*, 152(5), 798–800.

- Bartik, B. D. (1981). Monthly variations in births of autistic children in North Carolina. *Journal of the American Medical Women's Association*, 36, 363–368.
- Cicchetti, D. V., Showalter, D., Fuerst, D., & Rourke, B. P. (1992). A computer program for analyzing ordinal trends with dichotomous outcomes. Application to neuropsychological research. *Clinical Neuropsychologist*, 6, 458–463.
- Dalen, P. (1975). *Season of birth: A study of schizophrenia and other mental disorders*. Amsterdam: Elsevier/North Holland.
- Duplinsky, T. G., & Cicchetti, D. V. (2012). The health status of dentists exposed to mercury from silver amalgam tooth restorations. *International Journal of Statistics in Medical Research*, 1, 1–15.
- Fombonne, E. (1989). Season of birth and childhood psychosis. *British Journal of Psychiatry*, 155, 655–661.
- Gillberg, C. (1988). The neurobiology of infantile autism. *Journal of Child Psychology and Psychiatry*, 29, 257–266.
- Gillberg, C. (1990). Do children with autism have March birthdays? *Acta Psychiatrica Scandinavica*, 82, 152–156.
- Gnoth, C., Godehardt, D., Godehardt, E., Frank-Hermann, P., & Freundl, G. (2003). Time to pregnancy: Results of the German prospective study and impact on the management of infertility. *Human Reproduction*, 18, 1959–1966.
- Hare, E. H. (1975). Manic-depressive psychosis and season of birth. *Acta Psychiatrica Scandinavica*, 52, 69–79.
- Jonckheere, A. R. (1970). Techniques for ordered contingency tables. In J. B. Riemersma & H.C. van der Meer (Eds.), *Proceedings of the NUFFIC International Summer Session in Science “Het Oude Hof”*, The Hague.
- Landau, E., Cicchetti, D. V., Klin, A., & Volkmar, F. R. (1999). Season of birth in autism: A fiction revisited. *Journal of Autism and Developmental Disorders*, 29, 385–393.
- Mazumdar, S., Liu, K., Susser, E., & Bearman, P. (2012). The disappearing seasonality of autism conception in California. *PLoS One*, 7, 1–9.
- Rutter, M. (1983). Cognitive deficits in the pathogenesis of autism. *Journal of Child Psychology and Psychiatry*, 24, 513–531.

Seasonality of Autism

► [Season of Birth in Autism](#)

Secondary and Postsecondary Students with Autism Spectrum Disorder

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Definition

This entry addresses topics regarding students with autism spectrum disorder (ASD) without intellectual disabilities (ID) in secondary and postsecondary education. This is a group of individuals on the autism spectrum that is often referred to as having a *higher functioning autism* (Zeliadt 2016) or has been diagnosed with Asperger syndrome according to The International Classification of Diseases and Related Health Problems (ICD-10) (World Health Organization 1992). One of the hallmarks of ASD is to have challenges when it comes to social communication, thus making students with ASD at risk of being socially excluded in school settings. In this entry, we address issues pertaining to students with ASD without ID's school – and student life, especially issues regarding social experiences and mental well-being.

Historical Background

Over the past several decades, there has been an increasing number of those diagnosed with ASD with average or above average intellectual ability, many of whom are likely to attend mainstream

high school and subsequently pursue a postsecondary education (Jackson et al. 2018b). Furthermore, in line with the global ideology of inclusion on social and educational policy, students with ASD are more likely to attend mainstream schools and enroll into colleges than before (Nilsen 2017; Øien and Nordahl-Hansen 2018). Students with ASD without ID may receive special education support depending on their needs. In a more long-term perspective, many adults with ASD without ID struggle when it comes to issues such as working life, independent living, and establishing intimate relationships (Koegel et al. 2012).

Current Knowledge

Studies show that individuals with ASD without ID experience an increased feeling of loneliness and isolation during adolescence (Lasgaard et al. 2010). Demands on social skills accentuate during teenage years, adding more pressure on students with ASD who already have challenges with social communication (White and Robinson-Nay 2009; Øien et al. 2019). Similarly, many adults with ASD experience that the feeling of loneliness grows stronger as they become older (Müller et al. 2008). Approximately a third of individuals with ASD without ID suffer from depression and anxiety (Koegel et al. 2012). Locke et al. (2010) conducted a study with the objective to research socioemotional aspects of students with high-functioning ASD in mainstream high schools. Even though the students with ASD were in an inclusive school setting, they experienced a higher degree of loneliness than their typically developed (TD) peers. Also, findings from a systematic review of literature unfolding friendship among children and youth with high-functioning autism (Petrina et al. 2014) showed that friendship can be a difficult aspect of school. The review showed that these children and youths had less friends and had more fragile friendships than their TD peers. The students with ASD also had fewer interactions with peers outside the school setting. Correspondingly, over three quarters of 56 college students with ASD reported having regular

struggles of feeling of loneliness and isolation (Jackson et al. 2018b). Students with ASD are also at risk of school refusal behavior. A Norwegian study showed that school refusal behavior was significantly higher in students with ASD without ID, as compared to TD students (Munkhaugen et al. 2017). In addition, the study showed that students with ASD without ID were more at risk of school refusal behavior than students with ASD and ID. Likewise, college students with ASD are at risk of dropping out before graduating (Jackson et al. 2018b) thus finishing their degree at a lower rate than their TD peers (Jackson et al. 2018b). Bullying is also a topic of great concern when it comes to students with ASD. Research show that students with ASD both with and without ID are more at risk of being bullied than their neurotypical peers and subsequently suffer from depression and anxiety (Williams et al. 2017; Weinstock 2018). Although bullying often decreases when students become of age, autistic college students may still experience being bullied in their college years (DeNigris et al. 2018). In fact, being bullied may increase suicidal behavior in individuals with ASD (Weinstock 2018). In a recent study of 56 college students with ASD, as many as three quarters of the participants reported lifetime suicidal behaviors (Jackson et al. 2018b). Further, some studies show that individuals with ASD without ID are particularly at risk of suicide (Richa et al. 2014).

Findings from a metasynthesis of 31 qualitative studies by Williams et al. (2017) proposed that inclusive mainstream settings may heighten many pupils with ASD's sense of being "different" from TD peers in a negative way, thus increasing their risk for developing low self-esteem and mental health problems. Most of the participants in the metasynthesis were in their teenage years. Further, in a study by Elias and White (2018) of the needs of college students with ASD from their parents' perspective, the results suggested that college students struggle with social tasks and independent living. The results further suggest that these difficulties are impairing when it comes to finishing their postsecondary degree (Elias and White 2018). Additionally, school transition is a topic known to be challenging for

students with ASD (Nuske et al. 2018). However, a recent study showed that school transitions may be perceived as a new and positive start for some (Skafle et al. 2019). The students in this study, all diagnosed with Asperger syndrome, expressed that they felt included by their high school peers and teachers and appreciated leaving their former classmates in order to start "fresh" and entering a new role with new people. These students also expressed that their social skills improved by being social with their peers in school. Previous research also shows that TD students can be of good support and become role models for ASD students (Koegel et al. 2012). In order to support ASD students, there are different types of interventions especially targeted at educating social skills, with the objective of subsequently improving these students' social life in schools and colleges. Interventions may be short term and long term and focus on one specific social skill or several at the time (Ke et al. 2018). Moreover, different people surrounding the ASD student, such as parents, friends, clinicians, teachers, or special education teachers, can be responsible for the interventions (Mundy and Mastergeorge 2012).

Future Directions

In order to support the needs of students with ASD without ID in high schools and colleges, more research about the unique needs of students with ASD must to be undertaken, as there are a limited number of previous studies. It is likely that the rate of individuals with autism who graduate will rise with better support and thus finish their degree at the same level as their TD peers. In the USA, only 40% of students with ASD finished their post-secondary degree compared to 60% of TD students (Kuder and Accardo 2018). Moreover, in order to understand the needs of high school students and college students with ASD, it is necessary to investigate what works from their own perspective. So, in addition to investigating challenges, one needs to look further at positive outcomes and also listens to the ASD community itself.

References and Reading

- DeNigris, D., Brooks, P. J., Obeid, R., et al. (2018). Bullying and identity development: Insights from autistic and non-autistic college students. *Journal of Autism and Developmental Disorders*, 48, 666–678. <https://doi.org/10.1007/s10803-017-3383-y>.
- Elias, R., & White, S. W. (2018). Autism Goes to college: Understanding the needs of a student population on the rise. *Journal of Autism and Developmental Disorders*, 48, 732–746. <https://doi.org/10.1007/s10803-017-3075-7>.
- Jackson, S. L. J., Hart, L., & Volkmar, F. R. (2018a). Preface: Special issue – College experiences for students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48, 639–642. <https://doi.org/10.1007/s10803-018-3463-7>.
- Jackson, S. L. J., Hart, L., Brown, J. T., & Volkmar, F. R. (2018b). Brief report: Self-reported academic, social, and mental health experiences of post-secondary students with autism Spectrum disorder. *Journal of Autism and Developmental Disorders*, 48, 643–650. <https://doi.org/10.1007/s10803-017-3315-x>.
- Ke, F., Whalon, K., & Yun, J. (2018). Social skill interventions for youth and adults with autism Spectrum disorder: A systematic review. *Review of Educational Research*, 88(1), 3–42. <https://doi.org/10.3102/0034654317740334>.
- Koegel, L. K., Koegel, R. L., Chin, J. K., & Koegel, B. L. (2012). Ch. 4: Improving educational interventions for school age children with autism without intellectual disabilities. In P. Mundy & A. Mastergeorge (Eds.), *Educational interventions for students with autism*. San Francisco: Jossey-Bass. Retrieved from <https://www.adlibris.com>.
- Kuder, S. J., & Accardo, A. (2018). What works for college students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48, 722–731. <https://doi.org/10.1007/s10803-017-3434-4>.
- Lasgaard, M., Nielsen, A., Eriksen, M. E., et al. (2010). Loneliness and social support in adolescent boys with autism Spectrum disorders. *Journal of Autism and Developmental Disorders*, 40, 218–226. <https://doi.org/10.1007/s10803-009-0851-z>.
- Locke, J., Ishijima, E. H., Kasari, C., & London, N. (2010). Loneliness, friendship quality and the social networks of adolescents with high-functioning autism in an inclusive school setting. *Journal of Research in Special Educational Needs*, 10, 74–81. <https://doi.org/10.1111/j.1471-3802.2010.01148.x>.
- Müller, E., Schuler, A., & Yates, G. B. (2008). Social challenges and supports from the perspective of individuals with Asperger syndrome and other autism spectrum disabilities. *Autism*, 12(2), 2.173–2.190.
- Mundy, P., & Mastergeorge, A.M. (Red). (2012). *Educational interventions for students with autism*. San Francisco, CA, USA: UC Davis MIND Institute/WILEY.
- Munkhaugen, E. K., Gjevik, E., Pripp, A. H., Sponheim, E., & Diseth, T. H. (2017). School refusal behaviour: Are children and adolescents with autism Spectrum disorder at a higher risk? *Research in Autism Spectrum Disorders*, 41–42, 31–38. <https://doi.org/10.1016/j.rasd.2017.07.001>.
- Nilsen, S. (2017). Special education and general education—coordinated or separated? A study of curriculum planning for pupils with special educational needs. *International Journal of Inclusive Education*, 21(2), 205–217.
- Nuske, H. J., Hassrick, E. M., Bronstein, B., Hauptman, L., Aponte, C., Levato, L., Stahmer, A., Mandell, D. S., Mundy, P., Casari, C., & Smith, T. (2018). Broken bridges—new school transitions for students with autism spectrum disorder: A systematic review on difficulties and strategies for success. *Autism*, 1–20. <https://doi.org/10.1177/1362361318754529>.
- Øien, R. A., & Nordahl-Hansen, A. (2018). Norway and autism. In F. R. Volkmar (Ed.), *Encyclopedia of autism Spectrum disorders*. Springer. ISBN 978-1-4614-6435-8. https://doi.org/10.1007/978-1-4614-6435-8_102106-2.
- Øien, R. A., Nordahl-Hansen, A., & Schjølberg, S. (2019). Mental health and ASD. In F. R. Volkmar (Ed.), *Encyclopedia of autism Spectrum disorders*. Springer. https://doi.org/10.1007/978-1-4614-6435-8_102050-1.
- Petrina, N., Carter, M., & Stephenson, J. (2014). The nature of friendship among children with ASD: A systematic review. *Research in Autism Spectrum Disorders*, 8, 111–126. <https://doi.org/10.1016/j.rasd.2013.10.016>.
- Richa, S., Fahed, M., Khoury, E., & Mishara, B. (2014). Suicide in autism Spectrum disorders. *Archives of Suicide Research*, 18(4), 327–339. <https://doi.org/10.1080/13811118.2013.82483>.
- Skafle, I., Nordahl-Hansen, A., & Øien, R. A. (2019). Short report: Social perception of high school students with ASD in Norway. *Journal of Autism and Developmental Disorders*, 50, 670–675. <https://doi.org/10.1007/s10803-019-04281-w>.
- Van Schalkwyk, G., Smith, I. C., Silverman, W. K., et al. (2018). Brief report: Bullying and anxiety in high-functioning adolescents with ASD. *Journal of Autism and Developmental Disorders*, 48, 1819–1824. <https://doi.org/10.1007/s10803-017-3378-8>.
- Weinstock, C.P. (2018). The hidden danger of suicide in autism. *Spectrum*. Retrieved from <https://www.spectrumnews.org/features/deep-dive/hidden-danger-suicide-autism/>
- White, S. W., & Roberson-Nay, R. (2009). Anxiety, social deficits, and loneliness in youth with autism Spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 1006–1013. <https://doi.org/10.1007/s10803-009-0713-8>.
- Williams, E. I., Gleeson, K., & Jones, B. E. (2017). How pupils on the autism spectrum make sense of themselves in the context of their experiences in a mainstream school setting: A qualitative metasynthesis. *Autism*, 1–21. <https://doi.org/10.1177/1362361317723836>.
- World Health Organization. (1992). *The ICD-10 classification of mental and Behavioural disorders*. Switzerland: WHO Library Cataloguing in Publication Data.
- Zeliadt, N. (2016). The measure of a life. *Spectrum*. Retrieved from <https://spectrumnews.org/features/deep-dive/the-measure-of-a-life/>

Secondary Handicapping Conditions

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Synonyms

Comorbidity

Definition

Secondary handicapping conditions involve additional characteristics of other disabilities or disorders that may affect an individual to a lesser degree than the primary disability or disabling conditions, leaving marked impairment on the lives of individuals. Secondary conditions have been defined as “those physical, medical, cognitive, emotional, or psychosocial consequences to which persons with disabilities are more susceptible by virtue of an underlying condition, including adverse outcomes in health, wellness, participation, and quality of life” (Hough 1999, p. 186). Individuals on the autism spectrum are likely to experience additional deficit areas or secondary handicapping conditions as a result of comorbidity of one or more of the following conditions, disabilities, or disorders related to vision, depression, tuberous sclerosis, general anxiety disorder, Tourette’s syndrome, obsessive compulsive disorder, bipolar disorder, attention deficit hyperactivity disorder, Fragile X syndrome, intellectual/cognitive disabilities, seizure disorder, epilepsy, sensory integration, oppositional defiance disorder, depression, neuroinflammation disorders, immune disorders, nonverbal learning disorders, motor clumsiness, and gastrointestinal distress/bowel disease.

See Also

- Attention Deficit/Hyperactivity Disorder
- Comorbidity

- Course of Social Avoidance in Fragile X Syndrome, The
- Depressive Disorder
- Epilepsy
- Gastrointestinal Disorders and Autism
- Nonverbal Learning Disabilities (NLD)
- Obsessive-Compulsive Disorder (OCD)
- Seizure Disorder
- Tourette’s Syndrome

References and Reading

- Ehlers, S., & Gillberg, C. (1993). The epidemiology of Asperger’s syndrome. A total population study. *Journal of Child Psychology and Psychiatry*, 34(8), 1327–1350.
- Harrison, J. E., & Bolton, P. F. (1997). Annotation: Tuberous sclerosis. *Journal of Child Psychology and Psychiatry*, 38(6), 603–614.
- Hough, P. (1999). Center on health promotion research for persons with disabilities: Final report. Retrieved 1 Feb 2011 from http://www.ncpad.org/files/pdf/rimmer_chp.html
- Klin, A. (2006). Autism and Asperger’s syndrome: An overview. Retrieved 15 Jan 2011 from http://www.scielo.br/scielo.php?script=sci_arttext&pid=S1516-44462006000500002&lng=en&nrm=iso&tlng=en
- LoVullo, S. V., & Matson, J. L. (2009). Comorbid psychopathology in adults with autism spectrum disorders and intellectual disabilities. *Research in Developmental Disabilities*, 30, 1288–1296.
- Matson, J. L., & Nebel-Schwalm, M. S. (2007). Comorbid psychopathology with autism spectrum disorder in children: An overview. *Research in Developmental Disabilities*, 28(4), 341–352.
- Matson, J. L., Hess, J., & Boisjoli, J. A. (2010). Comorbid psychopathology in infants and toddlers with autism and pervasive developmental disorders – not otherwise specified (PDD-NOS). *Research in Autism Spectrum Disorders*, 4, 300–304.
- Reiersen, A. M., & Todd, R. D. (2008). Co-occurrence of ADHD and autism spectrum disorders: Phenomenology and treatment. *Expert Review of Neurotherapeutics*, 8(4), 657–669.
- Simonoff, E., Pickels, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(8), 921–929.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know*. Hoboken: Wiley.
- Volkmar, F. R., Paul, R., Klin, A., & Cohen, D. (2005). *The handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.

- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229.
- Zafeiriou, D. I., Ververi, A., & Vargiami, E. (2007). Childhood autism and associated comorbidities. *Brain & Development*, 29(5), 257–272.

Second-Generation Antipsychotics (SGAs)

► Atypical Antipsychotics

Second-Generation Sequencing

► Next-Generation Sequencing

SecreFlo

► Secretin

Secretin

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Synonyms

SecreFlo

Definition

Secretin, a digestive polypeptide hormone secreted in the upper part of the small intestine, stimulates the pancreas to secrete water and

bicarbonate, thus neutralizing hydrochloric acid that passes from the stomach into the duodenum. It is most commonly used in a synthetic form to evaluate pancreatic function.

In the late 1990s, extensive media coverage was given to a single case in which a 3-year-old child with autism who received intravenous secretion as part of a pancreatic assessment for gastrointestinal issues showed improvements in speech and behavior soon afterward. A subsequent case study series of three young children with autism who also received secretin as part of gastrointestinal evaluations reported improvements in language and social abilities in addition to the amelioration of gastrointestinal issues (Horvath et al. 1998). Based largely on the single case anecdotal report, the three-case series, and compelling anecdotal evidence, media reports publicized secretin as a treatment for autism. However, the case series had significant methodological limitations, and no empirical evidence supported the treatment. Concern over the widespread off-label use of secretin as a treatment for autism was expressed in the medical and research community (Volkmar 1999).

In response to the media frenzy and escalating off-label use of the hormone, a series of randomized, double-blind studies investigated the effects of secretin on children with autism. Sandler et al. (1999) randomly assigned 56 children diagnosed with either autism or PDD-NOS to receive either secretin or placebo saline infusions. They were assessed at baseline, immediately after infusion, and for 4 weeks postinfusion. The main findings were twofold: first, scores on the 16 outcome measures or assessment domains were not significantly improved with secretin treatment versus with placebo. Second, both secretin and placebo groups had significant decreases from baseline in autism symptoms over the 4-week period. Similarly, Coniglio et al. (2001) assessed 57 randomly assigned participants at baseline, 3 and 6 weeks postsecretin or placebo administration by parent report and clinical behavior testing. While a marginally significant improvement was seen at 3 weeks posttreatment for secretin, at 6 weeks neither group showed significant change in assessment from baseline.

In 2000, Chez et al. undertook a two-part study to further investigate the anecdotal claims of secretin treatment. First, 56 children were administered one dose of secretin and evaluated at baseline and several weeks postinfusion. Overall, the group had minimal improvement in behavior and language abilities, but not significant gains at a clinical level. Next, 17 of the most responsive subjects and an additional eight new subjects entered a double-blind trial of secretin and placebo with a 4-week crossover. They were evaluated by parent report at baseline, at 4 weeks, and at 8 weeks. Results of the second study did not demonstrate any significant behavioral differences between patients treated with placebo and secretin when the administration was double-blind and placebo controlled. Likewise, no significant improvement was seen in autism behaviors as assessed by parent and teacher report in a study of eight children who received secretin or placebo in a randomized double-blind study, also with a 4-week crossover (Carey et al. 2002). In fact, the majority of changes seen were behavioral improvement after placebo and deterioration after secretin. Taken together, these studies suggest a lack of evidence for secretin as a viable treatment option.

References and Reading

- Carey, T., Ratliff-Schaub, K., Funk, J., Weinle, C., Myers, M., & Jenks, J. (2002). Double-blind placebo-controlled trial of secretin: Effects on aberrant behavior in children with autism. *Journal of Autism and Developmental Disorders*, 32(3), 161–167.
- Chez, M. G., Buchanan, C. P., Bagan, B. T., Hammer, M. S., McCarthy, K. S., Ovrutskaya, I., et al. (2000). Secretin and autism: A two-part clinical investigation. *Journal of Autism and Developmental Disorders*, 30(2), 87–94.
- Coniglio, S. J., Lewis, J. D., Lang, C., Burns, T. G., Subhani-Siddique, R., Weintraub, A., et al. (2001). A randomized, double-blind, placebo-controlled trial of single-dose intravenous secretin as treatment for children with autism. *Journal of Pediatrics*, 138(5), 649–655.
- Horvath, K., Stefanatos, G., Sokolski, K. N., Watchel, R., Nabors, L., & Tildon, J. T. (1998). Improved social and language skills after secretin administration in patients with autistic spectrum disorders. *Journal of the Association for Academic Minority Physicians*, 9, 9–15.
- Sandler, A. D., Sutton, K. A., DeWeese, J., Girardi, M. A., Sheppard, V., & Bodfish, J. W. (1999). Lack of benefit of a single dose of synthetic human secretin in the treatment of autism and pervasive developmental disorder [see comments]. *The New England Journal of Medicine*, 341(24), 1801–1806.
- Volkmar, F. R. (1999). Lessons from secretin. *The New England Journal of Medicine*, 341(24), 1842–1844.

Section 504 of the Rehabilitation Act of 1973

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Definition

In 1973, President Carter engaged into law the Rehabilitation Act (P.L. 93-112; U.S. Congress 1973). This Act provided for a number of provisions subsequent in newfangled shields and rights for persons with disabilities. Section 504 of the Rehabilitation Act stated that “no otherwise qualified handicapped individual in the United States shall, solely by reason of his handicap, be excluded from participation in, be denied the benefit of, or be subjected to discrimination under the program or activity receiving Federal financial assistance.” According to Section 504, individual with disability means any person who:

1. Has a physical or mental impairment which substantially limits one or more major life activities
2. Has a record of such impairment
3. Is regarded as having such impairment

Students eligible for services under these provisions include some of the following groups:

1. Students with disabilities whose learning is impacted and those with attention deficit

disorder, attention deficit/hyperactivity disorder, immune disorders, allergies, learning disabilities, and hearing impairments

2. Those who have been misdiagnosed or labeled with a disability
3. Students with AIDS and other contagious diseases such as hepatitis B and tuberculosis
4. Students who experience temporary disabling circumstances such as those due to injuries

Section 504 essentially mandates that school districts must provide accommodations that include health related services that enables students to attend school. Schools must create a Section 504 accommodation plan to address how the school will insure that the student is not discriminated against upon the basis of their disability. Unlike IEPs under IDEA, Section 504 Accommodation Plans are unfunded.

See Also

- Individualized Plan for Employment (IPE)
- Individuals with Disabilities Education Act (IDEA)

References and Reading

- Betz, C. L. (2001). Use of 504 plans for children and youth with disabilities: Nursing application. *Pediatric Nursing*, 27(4), 374–375.
- Holler, R., & Zirkel, P. A. (2008a). Legally best practices in Section 504 Plans. *School Administrator*, 65(8), 38–40.
- Holler, R., & Zirkel, P. A. (2008b). Section 504 and public schools: A national survey concerning “Section 504-only” students. *NASSP Bulletin*, 92(1), 19–43.
- Katsiyannis, A., & Conderman, G. (1994). Section 504 policies and procedures: An established necessity. *Remedial and Special Education*, 15, 311–318.
- Moses, M., Gilcherst, C., & Schwab, N. C. (2005). Section 504 of the Rehabilitation act: Determining eligibility and implications for school districts. *The Journal of School Nursing*, 21(1), 48–58.
- U.S. Congress. (1973). Section 504 of the 1973 Rehabilitation Act, Pub. L. No. 93-112, 87 Stat. 394 (September 26, 1973), codified at 29 U.S.C. § 701 et seq.

Secure Employment

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Definition

Secure employment is an employment scheme designed for individuals that are not presently able to perform in competitive employment or supported employment settings. Secure employment is a facility-based program that utilizes a structured environment where individuals are guaranteed employment without the fear of losing employment due to intrusive behaviors. While in secure employment, trained staff work with the individuals to develop greater vocational independence and appropriate behavioral skills necessary for potential integration or reintegration into competitive or supported employment.

See Also

- Competitive Employment
- Supported Employment

References and Reading

- Gerhardt, P. F., & Holmes, D. L. (2005). Employment: Options and issues for adolescents and adults with autism spectrum disorders. In F. R. Volkmar et al. (Eds.), *Handbook of autism and developmental disorders* (Vol. 2, 3rd ed., pp. 1087–1101). Hoboken: Wiley.
- Holmes, D. L. (1998). *Autism through the lifespan: The Eden model*. Bethesda: Woodbine House.
- Holmes, D. L. (2011, May 31). *When the school bus stops coming: The employment dilemma for adults with autism*. Retrieved May 31, 2011, from Education. Com: <http://www.education.com/reference/article/employment-dilemma-adults-autism/>
- Ozonoff, S., Dawson, G., & McPartland, J. (2002). *A parent's guide to asperger syndrome and high functioning autism*. New York: Guilford Press.

Sedative Hypnotic Drugs

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Synonyms

Barbiturates; Benzodiazepines

Indications

Anxiety, Sedation, Sleep disturbances

Mechanisms of Action

Both barbiturates and benzodiazepines exert their primary mechanism of action by interacting with the GABA_A receptor. The GABA_A receptor is a heteromeric receptor containing five subunits, most commonly two alpha subunits, two beta subunits, and one gamma subunit. GABA_A receptor alpha, beta, and gamma subunits can be of several subtypes (alpha1-6, beta1-3, and gamma1-3). Different subunit compositions can lead to different types of GABA_A receptor that have different expression and function throughout the CNS.

Barbiturates

Barbiturates act by a mixture of several mechanisms dependent on the dose employed. The primary mechanism of action is by potentiating the action of the inhibitory neurotransmitter GABA by binding to GABA_A receptor alpha subunits (which are distinct from both the GABA and benzodiazepine binding sites). This leads to an increase in the duration of chloride ion channel opening at the GABA_A receptor which potentiates the action of GABA. Furthermore, barbiturates also block the action of the excitatory neurotransmitter glutamate by blocking glutamatergic

AMPA receptors. At higher doses, barbiturates also block the Ca²⁺-dependent release of neurotransmitters. Taken together, barbiturates exert potent CNS depressant effects via these mechanisms.

Barbiturates have also been shown to bind to other ligand-gated ion channel targets including the neuronal nicotinic acetylcholine receptor, the serotonin 5-HT₃ receptor, and the glycine receptor family, suggesting that their therapeutic and side effects may also depend on these other potential mechanisms of action.

Benzodiazepines

Benzodiazepines act primarily by potentiating the action of the inhibitory neurotransmitter GABA by acting on benzodiazepine binding sites which form part of GABA_A receptor complexes modulating GABAergic receptor function in the CNS. This serves to promote the binding of GABA to its binding sites on the GABA_A receptor which increases the conductance of chloride ions across the coupled ion channel leading to membrane hyperpolarization and inhibition. These benzodiazepine-binding sites are situated at the junction between alpha and gamma subunits on the GABA_A receptor. Interestingly, benzodiazepine binding can only occur at those GABA_A receptors expressing alpha subunits with a histidine amino acid residue (alpha 1, 2, 3, 5). For this reason, benzodiazepines have no effect on GABA_A receptors containing alpha 4 or alpha 6 subunits which have an arginine amino acid residue instead of a histidine one. It is notable that different benzodiazepines have differential effects of different GABA_A populations dependent on the subunit composition. For example, those with higher affinity for GABA_A receptor populations expressing alpha 2 and/or alpha 3 tend to show more marked anti-anxiety effects, while those with an affinity to alpha 1 containing GABA_A receptors demonstrate increased hypnotic activity.

Outside of the CNS, benzodiazepines also bind to peripheral benzodiazepine receptors that are not structurally or functionally coupled to the

GABA_A receptor but are expressed predominantly in the peripheral nervous system and glia, suggesting immunomodulatory actions. It has also been reported that benzodiazepines also act as weak antagonists at adenosine receptors which may play a role in their anxiolytic, muscle relaxant, and anticonvulsant effects.

Key sites of action for both barbiturates and benzodiazepines include binding sites in the spinal cord (exerting muscle relaxant effects), brainstem (accounting for anticonvulsant properties), cerebellum (inducing ataxia), and cortic limbic brain regions (regulating emotional and behavioral effects).

Specific Compounds and Properties

Barbiturates

Thiopentone and methohexital (very short duration of action) are employed as anaesthetic-induction agents. Secobarbital (short duration of action), butobarbital (medium duration of action), phenobarbital (long duration of action). Barbiturates are associated with drowsiness, tolerance, increased risk of overdose, and possible physical and psychological dependence with severe withdrawal symptoms.

Benzodiazepines

Chlordiazepoxide (prototype of benzodiazepine class; introduced in the 1930s), diazepam (extensively used; rapid onset of action; *N*-desmethyldiazepam (nordiazepam) is the major active metabolite; long half-life (60 h)), lorazepam (rapid onset of action; half-life of 12 h), temazepam (half-life of 10–15 h), flunitrazepam (potent; short duration of action; half-life of 10–20 h), alprazolam (half-life of 9–16 h), oxazepam (half-life of 12 h), nitrazepam (long duration of action; half-life of 25–35 h), flurazepam (long duration of action (via metabolite)), lormetazepam (medium duration of action; half-life of 10–12 h), loprazolam (short half-life; slow absorption), triazolam (short duration of action; half-life of 3–4 h; sedation at higher doses), zopiclone (binds near but not at the benzodiazepine receptor; half-life of 5–8 h dependent

on age of subject; lower degree of side effects compared to other benzodiazepines while retaining sedative hypnotic actions), eszopiclone (*s*-enantiomer of zopiclone; approved in the USA for long-term insomnia treatment), zolpidem (rapid absorption; short half-life (0.7–3.5 h), low side-effect profile), zaleplon (very short half-life of 1 h; low side-effect profile; reduces sleep onset).

Clinical Use (Including Side Effects)

The term “sedative” originally pertained to therapeutics that allayed anxiety although its more current usage is more often associated with drowsiness or torpor. This originally included barbiturates as a drug class versus what was originally perceived as nonsedative properties of benzodiazepines. This distinction is artificial with both drug classes having similar clinical and pharmacological profiles with marked anxiolytic properties. Hypnotics pertain to the sleep-induced properties of these therapeutics, and as such, sedative hypnotics as a drug class best pertain to benzodiazepines, although other classes such as barbiturates, gamma-aminobutyric-acid (GABA) analogues, and anti-epileptic drugs have sedative hypnotic effects. However, the more severe side-effect profiles (including increased risk of death with overdose) of barbiturates have led to their replacement in routine clinical use by benzodiazepines.

The clinical utility of barbiturates has been surpassed by the adoption of benzodiazepines as safer sedative hypnotics. Barbiturates have been employed primarily in the past as anxiolytic and sedative agents with some such as phenobarbital still retaining clinical usage as anticonvulsants. Due to the risk of death with overdose, their clinical use must be carefully monitored particularly with regard to their CNS depressant effects which can mediate side effects as varied as respiratory depression, postural hypotension, confusion, fatigue, and impaired concentration/decision-making.

Benzodiazepines are most commonly employed in the symptomatic control of anxiety and stress-related conditions, although regulatory approval is usually for panic disorder and

generalized anxiety. Irrespective of origin, these anxiety symptoms are the primary clinical indication of these therapeutics. While anxiety disorders can be both short and long lasting, benzodiazepines are usually used for the symptomatic management regardless of symptom duration. Given the long half-life of some benzodiazepines such as diazepam, treatment can be taken once daily but patients often prefer the reassurance of twice-daily administration claiming that they perceive anti-anxiety effects directly after each dose. For episodic anxiety, short duration of action benzodiazepines such as lorazepam taken before exposure to the anxiety generating situation exert potent effects while also exerting anti-anxiety effects when taken during a panic attack. This preventive effect against panic attacks has also been claimed for other benzodiazepine such as alprazolam. However, discontinuation of benzodiazepine treatment often results in relapse or even rebound panic episodes. Benzodiazepines with a short duration of action are also employed adjunctively to relaxation therapy. Overdosage with benzodiazepines is common, but associated deaths are fortunately rare. However, extreme caution must be taken in terms of dose regulation in children, especially in those with preexisting respiratory complaints. Benzodiazepine effects are potentiated in the presence of other depressant drugs such that the consumption of alcohol while being treated with benzodiazepines is not advised. In situations where benzodiazepines and alcohol are concurrent, the patient often falls into a deep sleep but can be aroused following administration of the benzodiazepine antagonist flumazenil or similar drugs.

Hypnotic effects of both barbiturates and benzodiazepines are most commonly employed against sleep disturbances, notably insomnia. The cause of the sleep disturbance should be investigated first and if possible resolved through other means as the utility of long-term benzodiazepines may result in paradoxical sleep disturbances. Often, anxiety/stress can precipitate sleep disturbances, and resolution of these problems will reduce the need for long-term benzodiazepine use. If used for their hypnotic effect, those benzodiazepines with a shorter half-life are

preferred to avoid excessive sleeping through the 24-h cycle. Of note, 5–10 mg diazepam once at night or temazepam (which has a half-life of 10–15 h) can induce sleep onset without many residual side effects the next day, while nitrazepam with a longer half-life (25–35 h) is employed to both manage anxiety during the day and induce sleep at night. While the hypnotic use of most benzodiazepines are often utilized prophylactically prior to the patient going to bed, the newer drugs such as zolpidem and zaleplon can be taken on an “as needed” basis if natural sleep onset fails after retiring to bed. This reduces the risk of dependence on these drugs and given their short duration of action reduces the side-effect profile associated with their utility.

Tolerance to moderate doses of barbiturates or benzodiazepines can occur, necessitating an increase in dosage to achieve anti-anxiety effects. However, as the stress/anxiety resolves, stepwise decreases in dosage should also be considered. Long-term clinical studies of barbiturate and benzodiazepine usage in autistic populations are lacking, but withdrawal of medication often results in relapse, suggesting that maintenance ensures continuation of the anti-anxiety effects or at least an avoidance of rebound effects. Dependence can occur at higher doses with drug withdrawal precipitating relapse 2–4 days or 5–10 days post-discontinuation in the case of short- and long-acting benzodiazepines, respectively. Withdrawal symptoms often include anxiety, apprehension, dizziness, insomnia, confusional psychoses, and anorexia. Physical dependency can precipitate withdrawal symptoms such as muscle weakness, postural hypotension, convulsions, tremor, and nausea and vomiting. As such, it is best to reduce the dose gradually in order to minimize the risk of withdrawal symptoms which often ensue following abrupt termination of benzodiazepine use.

Oversedation of subjects results in the most commonly reported side effects of barbiturates and benzodiazepines: drowsiness, tiredness and torpor which are both dose- and time dependent. Drowsiness is typically observed within the first week of treatment, an effect to which the subject becomes tolerant to rapidly. During the initial phase of benzodiazepine treatment, subjects are

advised not to drive given the impairment to both cognitive and psychomotor coordination caused by benzodiazepines. These cognitive impairments are a sustained feature of chronic benzodiazepine treatment with memory function being notably affected. Patients taking benzodiazepines can also encounter paradoxical behavioral responses such as increased aggression, uncharacteristic behavior, acute rage episodes, and intermittent crying. In autistic children, paradoxical anxiogenic effects have been reported. These paradoxical effects are not unique to benzodiazepines and usually resolve spontaneously after a week of treatment or following dose adjustment. Other undesirable side effects associated with benzodiazepine use include weight gain, skin rash, menstrual irregularities, impairment of sexual function, and respiratory depression. Rare side effects such as blood dyscrasias are also sometimes seen.

References and Reading

- Allgulander, C. (1986). History and current status of sedative-hypnotic drug use and abuse. *Acta Psychiatrica Scandinavica*, 73, 465–478.
- Chouinard, G. (2004). Issues in the clinical use of benzodiazepines: Potency, withdrawal and rebound. *The Journal of Clinical Psychiatry*, 65, 7–12.
- Coupey, S. M. (1997). Barbiturates. *Pediatrics in Review*, 18, 260–265.
- Fineberg, N., & Drummond, L. M. (1995). Anxiety disorders. Drug treatment or behavioural cognitive psychotherapy? *CNS Drugs*, 3, 448–466.
- Garreau, B., Herry, D., Zilbovicius, M., Samson, Y., Gue-
rin, P., & Lelord, G. (1993). Theoretical aspects of the study of benzodiazepine receptors in infantile autism. *Acta Paedopsychiatrica*, 56, 133–138.
- Johns, M. W. (1975). Sleepy and hypnotic drugs. *Drugs*, 9, 448–478.
- Johnson, G. A. (1996). GABAA receptor pharmacology. *Pharmacology and Therapeutics*, 69, 173–198.
- Lader, M. (2008). Effectiveness of benzodiazepines: Do they work or not? *Expert Review of Neurotherapeutics*, 8, 1189–1191.
- Michellini, S., Cassano, G. B., Frare, F., et al. (1996). Long-term use of benzodiazepines: Tolerance, dependence and clinical problems in anxiety and mood disorders. *Pharmacopsychiatry*, 29, 127–134.
- Stevens, J. C., & Pollack, M. H. (2005). Benzodiazepines in clinical practice: Consideration of their long-term use and alternative agents. *The Journal of Clinical Psychiatry*, 66, 21–27.

Seeing Essential English (SEE I)

- [Manual Sign](#)
- [Sign Language](#)

Segment

- [Phonemes](#)

Seizure

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Synonyms

[Convulsion](#); [Epilepsy](#)

Short Description or Definition

Seizures (sometimes called epileptic seizures) are the stereotypical clinical manifestations (signs and symptoms) of excessive and/or hypersynchronous, usually self-limited, abnormal activity of neurons situated in the cerebral cortex. Epilepsy is defined as two or more unprovoked afebrile seizures (Commission on Classification and Terminology of the International League Against Epilepsy [1981](#)).

Categorization

Although children with autism and epilepsy have many types of movements, the clinical

semiology of *seizures* is similar to those in children without autism (i.e., children with pediatric epilepsy alone). The most common type of seizures during childhood, a *partial seizure*, occurs in two categories. *Simple partial seizures* are those in which the first clinical signs and electroencephalographic (EEG) signatures initiate focally in one area of the brain without impairment of consciousness. Simple partial seizures show focal neurological signs such as focal jerking of one hand/arm, sensory change or pain in one limb, or a unilateral contraction of the face. *Partial complex seizures* (*psychomotor seizures*) are the more common of two types of partial seizures that manifest as focal neurological signs plus impairment of consciousness. Commonly, for instance, in temporal lobe epilepsy, patients may experience a gustatory sensation, rising epigastric feeling, or some other aura followed by a behavioral arrest. The child does not respond and stare off. Then the patient is tired. Sometimes the children develop jerking movements of limbs contralateral to the seizure focus. *Secondary generalization of partial complex seizures* occurs when seizures spread to the opposite hemisphere and is manifested clinically as *generalized tonic-clonic seizures*.

Clinical Expression and Pathophysiology

Partial epilepsies in childhood with autism often manifest as a frontal lobe or temporal lobe epilepsy. The interictal epileptiform activity until age 10–11 years is unilateral focal or multifocal epileptiform discharges. The ictal manifestations on EEG usually include evolving focal sharp-wave or spike and slow-wave discharges. As children become older (age 10–11 years), epileptiform discharges on EEGs are more frequent in the frontal or centrofrontal regions. Adolescents can have multifocal epilepsy in which the predominant seizure type is a partial complex seizure. However, a majority of seizures in the adolescent are generalized seizures. The generalized tonic-clonic seizures are the most common type of generalized seizures. The seizure may have a prodrome in which a change in behavior

is seen. Most seizures began without warning when the patient falls to the floor, cries out, eyes roll back in the head, and the limbs have a rhythmic tonic-clonic-tonic pattern of jerking. The patient may lose bowel or bladder function at the end of the ictal phase. Cyanosis can develop but is usually transient. The generalized tonic-clonic seizures are typical of frontal lobe epilepsy which occurs in adolescents. The EEG correlate is a buildup of low-voltage fast activity which evolves into high-amplitude spike/poly-spike or polyspike and wave discharges. Patients are typically sleepy or confused for some period of time after the seizure.

The second most common type, *absence seizures*, is characterized clinically by lapses in consciousness in which one can see a motionless stare, eyelids may droop, or eyes may briefly roll backwards and lasting 10–15 s in children with autism. The children usually resume their full activity after the spell or maybe briefly confused (<30 s). Only rarely, absence seizures are associated with other activity including automatisms, brief clonical movements of arms or eyelids, or loss of postural tone. The onset of absence seizures is associated with EEG onset of regular bilaterally frontal predominant generalized 3-Hz spike and wave discharges which begin and end suddenly in the setting of the background EEG for the child. With either absence or GTC seizures, children can have fragments of discharges interictally which include bilaterally frontal predominant generalized spike and slow-wave discharges.

Atypical absence seizures are lapses in consciousness in which one can see a motionless stare but are associated more with motor signs, particularly changes in tone, and can be more pronounced than in typical absence seizures. These seizures can have monitor focal or lateralizing signs. The onset and cessation of these seizures are less clear and last longer, typically 15–60 s with variable postictal confusion. These patients are more likely to have absence status epilepticus. The clinical onset is associated with similar generalized spike-wave discharges but usually at a frequency of <2.5 Hz. Although atypical absence seizures can be seen in the setting

of Lennox-Gastaut syndrome, these seizures are rarer in autism patients with Lennox-Gastaut syndrome.

Tonic and atonic seizures are more common in autism patients but not necessarily always associated with Lennox-Gastaut syndrome. *Tonic seizures* are more common in childhood and represent a continuum of the atonic-tonic seizures. The seizures have tonic spasms of the face or chest/trunk with tonic flexion of the upper extremities and flexion or extension of the lower extremities. Along with impairment of consciousness, patients can have papillary dilation, tachycardia, apnea/cyanosis, and urinary incontinence followed by a period of post-ictal confusion. Ictal EEG is low-amplitude very fast activity. Atonic seizures (usually called drop attacks) consist of a sudden loss of postural tone. In some patients, the drop attacks are preceded by one or more clonic jerks. In mild forms, the child may just have a head drop. In severe forms, the patient's whole body may drop to the floor and require a seizure helmet. The atonic seizure usually lasts only a few seconds and has little postictal period. An occasional patient may be out several minutes on the floor. Atonic seizures on the ictal EEG exhibits either generalized polyspike and wave discharges or a sudden electrodecrement (suppression) of the EEG. The synchronization of discharges between hemispheres is important for these seizure types because a corpus callosotomy can be a treatment to abolish these seizures.

Myoclonic seizures (epileptic myoclonus) are relatively rare in autism and usually seen in the most profoundly affected autism-epilepsy patients. The broader term myoclonus refers to certain quick involuntary muscle jerks involving any part of the neuroaxis. Myoclonic seizures in autism can be differentiated both semiology wise and neurophysiologically from movement disorders, hyperreflexia, and rare cases of spasticity. Myoclonic seizures (although can be unifocal, multifocal, or unilateral) are usually bilateral generalized jerks which are either sporadic or rhythmic in nature. Action or sensory

myoclonus rarely occurs in autism-epilepsy patients. Commonly, the myoclonic seizures are rapid rhythmic bilateral synchronous jerks (2–8 Hz) of the upper extremities with occasional lower extremity/whole body involvement. The ictal EEG is generalized polyspike and slow-wave discharges associated with the quick jerks.

Status epilepticus is commonly defined as repeated seizures/events without a return to consciousness lasting longer than 30 min in duration. Most types of epileptic seizures can be manifested as status epilepticus. The two major types of status epilepticus, generalized convulsive status epilepticus (major motor seizures, recurrent generalized tonic-clonic convulsions) and nonconvulsive status epilepticus (recurrent nonconvulsive seizures including absence status, partial complex status, and simple partial status) are recognized clinically. Convulsive status epilepticus is the most common medical emergency from neurological disease in autism because brain damage and death can result from the systemic consequences of repeated generalized tonic-clonic seizures. Most persons with generalized tonic-clonic status epilepticus have localized cerebral disturbances as a cause and therefore have secondary generalized partial seizures. Repeated cerebral epileptic activity can disrupt brain structures or otherwise cause permanent neurological or intellectual deficits.

Evaluation and Differential Diagnosis

See ► [“Neurologist.”](#)

Treatment

Therapy must be directed at suppressing all ictal activity on EEG. Ictal EEG activity can have a progression from (1) discrete seizures, (2) merging of seizures with waxing and waning of amplitude and frequency with variable locations, (3) continuous ictal activity, (4) continuous ictal

activity intermixed with periods of isoelectric EEG, and (5) the PLEDS or GPEDS pattern. The recurrence of frequent generalized tonic-clonic seizures in a row creates a life-threatening systemic condition of hyperpyrexia, failure of cerebrovascular autoregulation, acidosis, and severe hypoxia, causing hypotension, hypoperfusion of the brain, pulmonary edema, electrolyte disturbances, and eventual circulatory collapse. Even after cessation of status epilepticus and correction of systemic abnormalities, sepsis from aspiration pneumonia can be late but life-threatening complication of status epilepticus. Treatment of status epilepticus in autism, consisting of ABC's, correction of glucose/electrolyte disturbances, control of BP, and oxygenation, benzodiazepines, and a series of routine anticonvulsants, is not different from treatment of status epilepticus in other conditions.

See Also

► [Epilepsy](#)

References and Reading

Commission on Classification and Terminology of the International League Against Epilepsy. (1981). Proposal for revised clinical and electroencephalographic classification of epileptic seizures. *Epilepsia*, 22, 489–501.

Seizure Disorder

► [Epilepsy](#)

Seizures

► [Medical Conditions Associated with Autism](#)

Selective Attention

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Definition

Selective attention refers to the ability to pay attention to a limited array of all available sensory information. Selective attention, as a filter to help prioritize information according to its importance, is adaptive. If attention is too selective, however, it is maladaptive. Excessively selective attention has become known as “stimulus overselectivity,” which is prevalent in autism. Its cause or causes are assumed to be brain-organic. Because overselectivity has serious implications for impairment of learning at many levels, including social, emotional, and language learning (all key features of autism), it is suggested that an evidence-based treatment should focus on the normalization of attention patterns as early as possible to take advantage of the young brain's plasticity. Behavior analysis can provide such evidence-based treatments. Until a true cure for autism is found, behavior analysis remains the treatment of choice.

Historical Background

The term of “stimulus overselectivity” was coined by Lovaas et al. (1971) in their pioneering study investigating abnormal attention patterns in children with autism. Even though some degree of selectivity in attention is considered adaptive, Lovaas et al. (1971) felt that the degree of selectivity observed in their study was excessive, thus their term “overselectivity.” In their study, children with autism, with mental retardation, or

with typical development were first trained with so-called compound stimuli comprising visual (flood light), auditory (tone), and tactile (blood-pressure cuff) stimulus components that were presented simultaneously at predictable time intervals – in effect adding a fourth, temporal, component. When the child responded in the presence of the compound stimulus, a reward was given but not when the child responded in the absence of it. (Technically speaking, this constituted a successive discrimination paradigm.) During testing, the stimulus components were then presented successively in isolation and random order. The question of interest was whether the children would respond to the isolated stimulus components, after they had responded reliably to the compound stimuli. Generally, the typical children responded to all three isolated components, the children with mental retardation to two, and the children with autism to only one. (No child responded to the temporal element.) Thus, the children with autism were more selective in their attention to isolated stimulus components than the children in the other two groups. No consistent preference for any particular sensory modality was observed for these three groups.

Over the next four decades, this study by Lovaas et al. (1971) has stimulated a slew of follow-up research studies that employed a variety of different training and testing paradigms, different sensory modalities and stimulus types, and various schemes of experimental control and matching procedures (e.g., by diagnosis, chronological age, mental age, verbal skills, functioning level, and severity of autistic traits). No consistent pattern has emerged though (cf. Ploog 2010). For example, even though stimulus overselectivity is clearly prevalent in autism, it also occurs in other diagnostic categories (e.g., Down Syndrome); even though there appears to be a correlation between mental age and degree of overselectivity, there is evidence that overselectivity is also independent of mental age; and even though overselectivity is more likely to occur with increased stimulus complexity, overselectivity can also be shown in relatively simple two-stimulus situations. In some cases, it can even

be shown that the atypical attention consists of a *lack* of focus and *not of too narrow a focus*. In other words, processing of sensory information appears simply disorganized or haphazard and quite the opposite to the original conceptualization of overselective attention. Despite such inconsistencies, however, there is consensus that children with autism somehow differ from their typically developing peers in their attention patterns. This has lead researchers to propose different conceptualizations of attention patterns in autism, which will be discussed in the following section. After that, the implications of atypical attention patterns will be discussed. Finally, possible treatment approaches to remediate or normalize such atypical attention patterns will be discussed.

Alternative Conceptualizations

Table 1 is an attempt to provide a way to compare and delimit several of the most common

Selective Attention, Table 1 Comparison of theories in their conceptualizations of atypical attention patterns in ASD

	Approach			
Regarding atypical attention, the theory/ hypothesis . . .	SO ^a	PD ^b	WCC ^c	PPF ^d
distinguishes between local versus global information	x	x	x	x
assigns a role to “meaning”		x	x	
assumes bias/ undirected focus rather than true deficit	x	x	x	
sees atypical attention as cause for other abnormalities	x	x		
posits diminished movement perception				x
attempts to account for all sensory modalities	x	x	x	

^aStimulus Overselectivity Hypothesis

^bPrioritization Deficit Hypothesis

^cWeak Central Coherence Theory

^dEnhanced Perceptual Functioning Theory

approaches to understanding the atypical attention patterns seen in autism: stimulus overselectivity hypothesis, prioritization deficit hypothesis, weak central coherence theory, and enhanced perceptual functioning theory. The stimulus overselectivity hypothesis (SO) was introduced in the previous section.

The prioritization deficit hypothesis (PD) is really an extension of SO, except that instead of limiting the attentional abnormalities strictly to “overselectivity,” it includes unfocused, unattended, misdirected, or chance attention to isolated stimulus features. This extension seemed justified based on studies that implied that, surprisingly, the attention of children with autism was *less* selective than that of typically developing children. In Ploog et al. (2009), for example, children with autism and with typical development were shown to accurately discriminate between spoken sentences on the basis of *what* was being said (content) and *how* it was being said (prosody: question vs. statement). So there was no deficit in perception or attention. However, when content was pitched against prosody, the autistic children chose both, content, and prosody, with equal probability whereas typical children focused their attention strongly on content. (There was some evidence, albeit not statistically significant, that the children with autism slightly outperformed the typically developing children on prosody discrimination.) Such a finding of reduced selectivity does not seem to be restricted to language stimuli (e.g., Ploog and Kim 2007, with tactile stimuli, even though a point was being made in their study that, depending on interpretation, the children with autism did in fact exhibit restricted attention). In the language study in particular, it appeared as if the children with autism, in the context of testing, indiscriminately responded to any stimulus dimension whereas the typical children assigned higher priority to content, even though all children were perfectly capable of perceiving differences in both content and prosody. In other words, the children with autism did not prioritize the information embedded in the stimuli according to what seemed to be the most relevant information to the typical children – such prioritization being an

example of *adaptive* selective attention. Thus, PD essentially broadens the SO notion to encompass maladaptive undirected attention. Early work by Etzel et al. (1981) raised the notion of “relevancy” in the development of stimulus control, which is a behavior analytical term to operationalize “attention” (e.g., Reynolds 1961). In PD, this concept of “relevancy” incorporates the notion of “meaning” when responding to language stimuli. The children with autism appear to select their choices of stimulus components more on a sensory-perceptual basis, whereas the typical children do so based on a higher cognitive level. This hypothesis was put to a test in a series of studies by Ploog and colleagues (Brooks et al. 2018; Brooks and Ploog 2013; Ploog et al. 2014). The findings were mixed. For example, in Ploog et al. (2014), we used the same test paradigm as Ploog et al. (2009) but employed German speech stimuli with participants who did not speak or understand German. The rationale for using “unfamiliar” speech stimuli was to remove “meaning” for both the typically developing and the autistic children. However, overall, the typical children still continued to prioritize content over prosody. Another interesting finding was provided by Brooks and Ploog (2013) who employed affective stimuli: the preference for content versus prosody was moderated by the emotional valence. “Grouchy” stimuli made typically developing children decrease their preference for content (as if they avoided the negative tone) whereas “enthusiastic” stimuli made children with autism increase their preference for content (as if they were more attracted by the positive tone but ignored the negative tone). Brooks et al. (2018) found that children with autism did not exhibit a deficit in generalizing to an unfamiliar voice (from training male to testing female or vice versa). However, note, that all four studies showed clearly that there was no bona fide deficit in perception in the children with autism.

Another conceptualization of atypical attention in autism is captured in the weak central coherence theory (WCC; e.g., Happé and Frith 2006). The main idea of WCC is that the “cognitive style” in autism is to perceive and process sensory information in a “detail-focused” manner rather

than to integrate an array of information in a larger context. As such, WCC is consistent with SO. In contrast, however, WCC is based on the assumption that a deficit in central coherence leads to a failure to extract “meaning.” SO does not afford a special status to meaning per se, beyond the overall information that is embedded in the stimuli, whereas PD, as was discussed above, does not posit that atypical attention results in a failure to extract meaning but rather that a failure to extract meaning results in atypical attention. Happé and Frith (2006) pointed out three challenges to early accounts of WCC: First, WCC may result (but not necessarily so) in superior processing of some information possibly because of the sharper focus on local information and because of the exclusion of global, contextual, and semantic information, which might distract from a sharp focus on local information. SO is compatible with this notion; PD is, too, as a chance focus on some stimulus feature or piece of information may result in superior processing of it. This idea may explain the underlying mechanism of the “savant” phenomenon that has been reported in some, albeit rare, cases of autism. Second, WCC may be a bias in selecting some information rather than a true deficit of not attending to certain aspects of sensory information. This, again, is compatible with SO and PD as it has not been possible to show in the literature any modality-specific deficits or limit to the degree of complexity to account for atypical attention. Third, WCC may be a by-product rather than the cause for anomalies in social cognition. In contrast, SO and PD both provide convincing evidence that atypical attention may be the cause for a multitude of deficits seen in autism, without arguing that atypical attention is the cause for autism. In this respect, SO and PD are not compatible with WCC.

Enhanced perceptual functioning (EPF; e.g., Mottron et al. 2006), as the name implies, posits superior perception in individuals with autism compared to typically developing individuals. Superiority in perception is assumed to occur in both, “low-level” and in “complex” cognitive tasks. In elaborating on EPF, Mottron et al. (2006) proposed “eight principles of autistic

perception.” The three most important ones are: First, an enhanced detail perception is assumed. This puts EPF on equal footing with SO, WCC, and PD and can possibly account for superior perception in some areas. Second, EPF proposes diminished movement perception. None of the other theories considered here speak to this issue directly; it is also not clear how this notion might be linked to atypical attention with respect to information that is not characterized by movement per se (e.g., language, tactile perception, and still pictures of visual patterns). However, if one considers movement information as just one specific type of information, SO, WCC, and PD can account for diminished movement perception, just as for any other form of compromised information processing. Third, autistic individuals exhibit increased frequency of lateral glances—a behavior that may be involved in perception of movement. Again, it is not clear how lateral glances would be critical in the development of attention to information other than the visual modality. The other theories considered here do not address this behavior. In conclusion, it seems that EPF is focusing on visual attention, whereas the other theories can incorporate attention within all sensory modalities.

Implications for Autism Spectrum Disorders

In the following discussion of the implications of abnormal attention in autism, the focus will be on a limited selection of key characteristics of autism: impairments in social skills, language, and emotional behavior. In addition, a hypothesis with regard to a contributing cause of stereotypic behavior, also common in autism, will be proposed.

Assuming that a child with autism is not attending to contextual and situational information in the same manner as a typical child would, it should not be surprising if a number of problems develop as a result. Consider a situation in which a child with autism, Andy, would like to play soccer with another child, Brad. Andy sees the ball, likes it, runs towards it, and tries to kick it. But he may not have heard what Brad said

because his attention was on the ball, not on other contextual and situational information. Whether Brad said, “Hey, go away! I am waiting for my friend to play with!” or “Hey, that’s great! You like soccer, too! Let’s play!” represents a critical difference in the way Andy would experience the conclusion of this social interaction. In the first case, Brad would probably take the ball away from Andy and show anger or worse. In the second case, Brad might let Andy have the ball and try to play with him. From Andy’s perspective, however, it was impossible to predict what he was going to experience. In one case, the soccer ball will be associated with a pleasant social interaction, in the other case with an unpleasant one. Of course, a social situation does not rest on one piece of information (i.e., the soccer ball) but also on a multitude of other cues (e.g., what is being said, how it is being said, “body language” of the other person, facial expressions, whether only one additional person or several persons enter in the social situation). Even if Andy attended to some of the spoken language (e.g., “Hey”), it would not have been sufficient to allow Andy to negotiate the social interaction with Brad. Under these considerations, it should be obvious that Andy would be very unlikely to develop normal, social behavior.

The previous scenario may also serve as an example to discuss the development of appropriate emotional behavior. Brad may have said “Hey” while smiling or frowning. If Andy was indeed oblivious (not paying attention) to the different facial expressions, he would have missed out on an opportunity to learn that happy and angry faces, displayed by others, are usually correlated with running into an emotionally positive or negative situation, respectively.

Abnormal attention much earlier in life represents an even bigger problem: At birth, most children nurse reflexively but the efficiency with which they do so is subject to learning as the mother provides important feedback for effective suckling. Furthermore, whether the baby nurses correctly or perhaps bites the mother’s nipple produces different consequences. In the former case, the mother might rock the baby, hum soothingly, and the baby is naturally rewarded with milk.

In the latter case, the mother might yelp in pain, push the baby back, say “No!”, and nursing is interrupted. Typical children learn the consequences of their behavior quite early, readily, and quickly. (A 2-month-old or even younger baby, for example, can learn to move his limbs in order to make a mobile spin.) But learning the consequences of one’s behavior depends critically on attending to a number of cues (e.g., tasting milk in mouth, hearing “no” or the soothing humming, feeling being pushed back, or being rocked gently). If a child does not attend to a variety of cues, the direction of any social interaction will be unpredictable and it will not develop appropriately.

Acquisition of language depends critically on being able to attend to multiple cues as well. Either overselective attention (SO and WCC concept) or unfocused, undirected attention (PD concept) would result in an impairment of language development. In the example of the nursing baby, it should be obvious that the baby will not learn the meaning of “no” as a verbal cue not to repeat whatever the baby was doing before hearing “no.” (Technically speaking, this is an example of positive punishment.) Here is another example: An infant is on a stroll with his mother. The mother is pointing out a cat and says: “Look at the kitty!” A little bit later, they come across a dog and the mother says: “There’s a doggie!” An infant, who pays attention only to the dog and cat animals but not to what the mother says, will not acquire the correct labels. Maybe the infant is not even looking at the animals but instead at the parked cars or the trucks at a construction site next to the sidewalk. But even if the child was attending to the animals *and* to his mother’s speech, it would be possible to learn the proper labels only if the infant actually attends to the relevant sounds (i.e., “doggie” and “kitty”) in his mother’s speech. Such language learning can be explained well by so-called “statistical learning” (which is the term used by linguists to refer to language learning through classical conditioning; Pavlov 1927) but it only works if the listener is able to detect the sequential patterns of sounds in speech (e.g., “the” and “a” being followed with high probability by a noun referencing an

object in the listener's environment). Finally, with critical deficits in receptive language (i.e., listening), one would expect that development of expressive language (i.e., speaking) would be seriously compromised.

With all these examples, one can appreciate that the child, who suffers from abnormal attention (either not being able to process several paths of information simultaneously or not being able to focus on the relevant piece of information), lives in an essentially unpredictable world. Learning in general is severely impaired; social interactions cannot be understood; whatever happens as a result of one's actions is not predictably linked to one's own behavior; language is a meaningless garble of sounds instead of probabilistic patterns; and other people's emotions – with significant consequences for self – will appear random. At the same time, good and bad things will happen to the child without much chance to understand why. Experimental work (as early as Pavlov 1927) has shown that unpredictable consequences to one's behavior, or random sequences of events that occur independently of one's behavior, might result in erratic and bizarrely stereotyped behavior (experimental "neurosis," Pavlov 1927, or "superstition," Skinner 1948). Abnormal attention and the resulting unpredictable consequences and events may therefore contribute to stereotyped behavior that is quite common in autism. (This is not to deny the possibility that engaging in these stereotyped behaviors, such as self-stimulatory behavior, are in itself reinforcing to the child, either because of the sensory stimulation it provides or because of the child's ability to produce some predictable outcome.)

Treatment Based on Normalizing Attention Patterns in ASD

WCC and EPF do not directly suggest treatment or remediation strategies. SO and PD do. This is so because both SO and PD base their analyses on operational definitions of attention that then allow the atypical attention to be normalized directly. There are no claims that any of these treatments result in a removal of the original

cause of abnormal attention or other deficiencies (whether such deficiencies are secondary to abnormal attention or not). Rather, remediation strategies should be seen as "crutches" to overcome some of the abnormalities in a given situation. However, if remediation strategies are implemented from early on, chances are that the brain, when it still has great plasticity, can be trained to process information more adaptively despite putative brain-organic differences compared to the typical brain. At the very least, the rate of divergence between typical and abnormal attention and problems resulting from abnormal attention can be minimized while the autistic child is developing. (Note that *despite effective* treatment, IQ scores may still decrease just because the rate of increasing mental age and cognitive functioning, due to effective treatment, is not keeping pace with the rate of increasing chronological age.)

Some behavior analytical treatments focus specifically on so-called pivotal behaviors such as attention to multiple cues (Koegel et al. 1989). Pivotal behaviors are behaviors that once acquired result in changes in other behaviors even if those were not specifically trained. Training of pivotal behaviors is therefore a very efficient way of producing significant, positive, and broad behavior changes without having to train each target behavior individually. Responding to multiple cues is considered a pivotal behavior because an ability to simultaneously pay attention appropriately to multiple bits of information is the basis for learning almost anything (as alluded to before). Pivotal behavior training for changing a person's attention to multiple cues is based on the conceptual framework of SO, which can easily be expanded to incorporate PD accounts of situations when attention seems to be directed towards irrelevant cues – cues that should be low in priority for attention – and not to high-priority cues. Two strategies will be discussed: attentional shaping (also referred to as within-stimulus fading or transfer-along-a-continuum) and training on conditional discrimination tasks.

Sidman and Stoddard (1966) conducted a pioneering study to demonstrate the effectiveness of attentional shaping – resulting in attention to a

neglected aspect of a stimulus – by employing drawings of ellipses with varying widths that children with mental retardation initially found very difficult to discriminate. Attentional shaping is a fading procedure that focuses the child on a relevant stimulus dimension from the very beginning of learning a difficult discrimination task (e.g., the width of the ellipse) by first exaggerating the difference and then decreasing it gradually. Ploog and Williams (1995) conducted a pigeon study to assess theoretically the effectiveness of this procedure and contrasting it with another procedure using so-called extra-stimulus prompting (Schreibman 1975) and a trial-and-error procedure. The following serve as an example for attentional shaping: It may be difficult for a child with autism to learn to tell apart the letter “E” from the letter “F,” if the child only attends to any features of the letters other than the critical one – presence or absence of the horizontal line on the bottom of the letters. Attentional shaping would begin with drawing the child’s attention to the feature that is critical for distinguishing between the two letters (i.e., bottom horizontal line). A first step might involve presenting sequentially flash cards with either a big horizontal line on it or a blank card with nothing on it. Once the child mastered this first step (generally not a difficult task even for a child with atypical attention), the next steps are introduced. The exaggerated line is gradually reduced to realistic dimensions and moved towards the bottom of the flash card. Then, the letter “F” is faded in on both flashcards until both letters are presented realistically (“E” being “F” with line vs. “F” being “F” without line).

Advances in computer technology have made attentional shaping much more feasible – especially if embedded in a fun computer game – because the different steps in training can be automated and individually titrated (including correction procedures) while the child is enjoying playing a computer game. Attentional shaping can be implemented in any situation as long as the distinguishing (relevant) feature of a discrimination task can be identified. An alternative way of teaching a difficult discrimination task, but possibly more problematic and not based on

attentional shaping, is what is commonly called “prompting.” Applied to the previous example, the adult may point to the letter “E” and reward the child for saying “E” or for selecting it from several choices. Then the prompt (i.e., the pointing finger) may gradually be removed. The reason why such prompting may not be so effective with a child who does not attend to multiple cues is that the child may only attend to the pointing finger but not simultaneously to the finger *and* the letter “E.” The failure to learn about the relevant stimulus feature can be explained by the behavior-analytic concept of “stimulus blocking” (e.g., Ploog and Williams 1995). Intuitively, such failure is also not surprising because the child is probably unresponsive to multiple cues to begin with, thus will either not attend to the pointing finger or not to the letter “E.” The two techniques, attentional shaping and prompting in the manner described here, have been referred to as within- and extra-stimulus prompting (cf. Schreibman 1975, who also showed superiority of within-stimulus over extra-stimulus prompting). Generally, the attentional shaping approach has been more effective than other prompting techniques in teaching children with autism because it practically imposes from the very beginning the stimulus feature that was initially neglected or was not given attentional priority by the child. By conducting extended training with attentional shaping methods, the child can learn the pivotal behavior of responding to multiple cues, that is, acquire a generalized skill beyond simply learning a series of individual discrimination tasks (Stokes and Baer 1977).

The learning of conditional discrimination tasks poses major difficulties to children who are not responsive to multiple cues. However, extended training on these task results in a generalized, increased responsiveness because learning to attend to multiple cues is inherent in the task by implicitly providing reinforcement only for attending to multiple cues. Consider this example: A mother might say “Please, pick the red shirt and get dressed!” or “Please, pick the blue pants and get dressed!” Unless the child paid attention to color and the particular clothing item, the child would have difficulties to follow the mother’s

request and not earn praise. A training procedure to make the child attentive to multiple cues can be implemented conveniently on a computer. For example, the words “RED SHIRT” might flash on the screen (or a person or computer could present the spoken words). After a short delay, pictures of several items will be given as choices to the child: a red shirt, a blue shirt, red pants, and blue pants. If the child picks the correct item (i.e., red shirt), he earns a reward such as a point, later to be exchanged for some preferred activity, or being allowed to view a brief, engaging video clip. Training would proceed with many of similar stimulus sets. Responsiveness to multiple cues can easily be expanded to incorporate three, four, or more cues (e.g., red/blue, big/small, new/old, corduroy/canvas, pants/shorts). Finally, it should also be clear that conditional discrimination tasks can be incorporated in daily life without requiring explicit training sessions with the associated advantages (e.g., more enjoyable for the children; logistically more convenient for the parents than having to schedule separate therapy sessions; more affordable and efficient because therapy can be implemented 24/7). Thus, providing conditional discrimination training should be part of parent training by using daily life situations (e.g., red/blue shirt/pants when getting dressed; small/big spoon/fork when setting table; blue/white small/large frisbee/ball).

Summary

Selective attention is adaptive, but if it is too selective, disorganized, or undirected, it is considered an abnormal attention pattern that is maladaptive. Abnormal attention is prevalent in autism but not specific to autism. Abnormal attention has been conceptualized as stimulus overselectivity, a prioritization deficit, weak central coherence, or enhanced perceptual functioning. Regardless of conceptualization, abnormal attention has serious implications for normal functioning and learning deficits, including many of the landmark features of autism such as deficits in language development, social and emotional behavior, academic success, abstract

cognition, and even possibly stereotypic/self-stimulatory excessive behaviors. Therefore, treatments should focus on normalizing attention patterns in individuals with autism as early in life as possible. Because the ability of normal selective attention is considered pivotal, behavior analytical treatments have targeted a normalization of attention in individuals with autism. As a treatment, attentional shaping is effective in training to direct attention specifically to one or several aspects of the surroundings that were previously neglected. Training on a series of conditional discrimination tasks is effective as a treatment in increasing the overall responsiveness to multiple cues and conveniently can be implemented by the primary caregivers.

References and Reading

- Brooks, P. J., & Ploog, B. O. (2013). Attention to emotional tone of voice in speech perception in children with autism. *Research in Autism Spectrum Disorders*, 7, 845–857.
- Brooks, P. J., Gaggi, N. L., & Ploog, B. O. (2018). Generalization of content and emotional prosody across speakers varying in gender in youth with Autism Spectrum Disorder. *Research in Developmental Disabilities*, 83, 57–68.
- Etzel, B. C., LeBlanc, J. M., Schilmoeller, K. J., & Stella, M. E. (1981). Stimulus control procedures in the education of young children. In S. W. Bijou & R. Ruiz (Eds.), *Behavior modification: Contributions to education* (pp. 3–37). Hillsdale: Lawrence Erlbaum Associates.
- Happé, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 36, 5–25.
- Koegel, R. L., Schreibman, L., Good, A. B., Cerniglia, L., Murphy, C., & Koegel, L. K. (1989). *How to teach pivotal behaviors to autistic children: A training manual*. Santa Barbara: University of California.
- Lovaas, O. I., Schreibman, L., Koegel, R. L., & Rehm, R. (1971). Selective responding by autistic children to multiple sensory input. *Journal of Abnormal Psychology*, 77, 211–222.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: an update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36, 27–43.
- Pavlov, I. P. (1927). *Conditioned Reflexes: An Investigation of the Physiological Activity of the Cerebral Cortex*. New York: Dover Publications.

- Ploog, B. O. (2010). Stimulus overselectivity four decades later: A review of the literature and its implications for current research in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 40, 1332–1349.
- Ploog, B. O., & Kim, N. (2007). Assessment of stimulus overselectivity with tactile compound stimuli in children with autism. *Journal of Autism and Developmental Disorders*, 37, 1514–1524.
- Ploog, B. O., & Williams, B. A. (1995). Two methods of stimulus fading applied to a simultaneous flicker rate discrimination in pigeons. *Learning and Motivation*, 26, 161–182.
- Ploog, B. O., Banerjee, S., & Brooks, P. J. (2009). Attention to prosody (intonation) and content in children with autism and in typical children using spoken sentences in a computer game. *Research in Autism Spectrum Disorders*, 3, 743–758.
- Ploog, B. O., Brooks, P. J., Scharf, A., & Aum, S. (2014). Perception of the prosody and content of sentences in an unfamiliar language in children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 8, 775–787.
- Reynolds, G. S. (1961). Attention in the pigeon. *Journal of the Experimental Analysis of Behavior*, 4(3), 203–208.
- Schreibman, L. (1975). Effects of within-stimulus and extra-stimulus prompting on discrimination learning in autistic children. *Journal of Applied Behavior Analysis*, 8, 91–112.
- Sidman, M., & Stoddard, L. T. (1966). Programming perception and learning for retarded children. In N. R. Ellis (Ed.), *International review of research in mental retardation* (Vol. 2, pp. 151–208). New York: Academic.
- Skinner, B. F. (1948). “Superstition” in the pigeon. *Journal of Experimental Psychology*, 38, 168–172.
- Stokes, T. F., & Baer, D. M. (1977). An implicit technology of generalization. *Journal of Applied Behavior Analysis*, 10, 349–367.

Selective Mutism

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Synonyms

Elective mutism

Short Description or Definition

Selective mutism is a disorder characterized by a “persistent failure to speak in specific social

situations (e.g., school, with playmates) where speaking is expected, despite speaking in other situations” (American Psychiatric Association [APA] 2000, p. 125). The diagnostic criteria for selective mutism also require that the disturbance lasts for more than 1 month and interferes with social communication or educational or occupational achievement. Additionally, the failure to speak cannot be the result of a lack of knowledge or comfort with the language expected in the social situation. Selective mutism is also not diagnosed if the disturbance is due to a communication disorder or occurs exclusively in the context of a pervasive developmental disorder, schizophrenia, or other psychotic disorder (APA 2000).

Categorization

Selective mutism is listed under Other Disorders of Infancy, Childhood, and Adolescence in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA 2000). Some scholars have argued that it would be more appropriate to categorize selective mutism under anxiety disorders in the DSM or classify it as a behavioral symptom of certain anxiety disorders rather than a distinct disorder (Anstendig 1999). Children who develop selective mutism tend to have similar biological and family dynamic characteristics as those who develop anxiety disorders, and cognitive-behavioral and pharmacological treatments for anxiety disorders have been used successfully with children who exhibit selective mutism (Anstendig 1999). However, there are currently no plans to list selective mutism as an anxiety disorder in the next edition of the DSM (APA 2010).

Epidemiology

The disorder has been estimated to occur in 0.2–2.0% of youth and tends to be more common in girls than boys (Kearney 2010). Prevalence rates are often higher in school-based samples compared to clinic-based samples because

children with selective mutism may not necessarily be referred for mental health services, as they are not disruptive in school (Cleave 2009).

Natural History, Prognostic Factors, Outcomes

Selective mutism usually develops during the pre-school years but may not be diagnosed until the child is in elementary school (Viana et al. 2009). The disorder does not have a specific, identified cause and likely develops through multiple pathways (Viana et al.). It is associated with a slow-to-warm and shy temperament (Sharp et al. 2007). Parents of children with selective mutism tend to have higher rates of anxiety and other forms of psychopathology than control groups (Viana et al. 2009). Some researchers have found relatively high comorbidity between selective mutism and other communication disorders (Viana et al.). For some children, selective mutism may have a chronic course that can lead to problems with peer rejection, academic performance, or social skills (Kearney 2010). For other children, the disorder may only last a few months (Sharp et al. 2007). Adults are less likely to be diagnosed with selective mutism, probably because they have more control over their environment and can more easily avoid situations that require speaking (Sharp et al.). Many young adults who were diagnosed with selective mutism as children report difficulties with self-confidence, independence, achievement, and social communication skills (Sharp et al.).

Clinical Expression and Pathophysiology

The clinical expression of selective mutism is usually characterized by a child who speaks normally at home with family members and does not speak in other settings, such as school or restaurants. Many children with selective mutism will also present with symptoms of anxiety disorders, particularly social phobia, which may include

fearing and/or avoiding social or performance situations. Parents, teachers, and peers may compensate for the child's failure to speak by speaking for the child or allowing nonverbal forms of communication (Kearney 2010).

The pathophysiology of selective mutism has not been directly examined, although physical symptoms of anxiety (e.g., increased heart rate, shaking, sweating) may be present in some individuals with selective mutism (Kearney 2010).

Evaluation and Differential Diagnosis

Evaluations for selective mutism may begin with interviewing the child, his or her parents, and the child's teacher. Some children with selective mutism will respond nonverbally (e.g., shaking head) to yes/no questions, which can provide an opportunity to obtain information from the child's perspective (Kearney 2010). Interviews may include questions related to each of the specific diagnostic criteria for selective mutism, the settings that involve a failure to speak, the circumstances surrounding the failure to speak, whether the child can be encouraged to speak in certain public settings, symptoms that may surround the child's failure to speak, and how others respond to the child's mutism (Kearney 2010). Behavior scales specific to selective mutism, anxiety, depression, and oppositional behavior can also be useful for obtaining information from multiple informants. Evaluations for selective mutism can also include direct behavioral observations across settings (Kearney 2010). In terms of differential diagnosis, if there is reason to suspect that a child's failure to speak is better accounted for by a communication disorder (e.g., specific language impairment), it would be important to conduct a comprehensive speech/language evaluation.

Treatment

Interventions for selective mutism include behavioral strategies such as exposure-based practice,

stimulus fading, shaping and prompting, relaxation training, and self-modeling, as well as pharmacological treatments (Kearney 2010). Exposure-based practice involves a hierarchy of speaking situations in which the child is expected to gradually speak in increasingly difficult or anxiety-provoking situations (Kearney 2010). Stimulus fading can be incorporated into exposure-based practice by gradually introducing new stimuli (e.g., peers or teachers). Shaping and prompting involve the reinforcement of successive approximations of the desired behavior. Relaxation training focuses on teaching the child how to relax his or her muscles and breathe properly to manage physical symptoms of anxiety. Self-modeling techniques may involve videotaping the child speaking clearly at home and playing the video at school to model the desired behavior in a setting in which speaking does not currently occur (Kearney 2010). The research base on pharmacological treatments for selective mutism is somewhat limited, but selective serotonin reuptake inhibitors (SSRIs) have shown promise for treating cases of selective mutism that are not responsive to psychosocial or behavioral interventions (Carlson et al. 2008).

See Also

- Anxiety
- Anxiety Disorders
- Separation Anxiety Disorder
- Social Behaviors and Social Impairment

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author. Text revision.
- American Psychiatric Association. (2010). *Disorders usually first diagnosed in infancy, childhood, or adolescence*. Arlington: Author. Retrieved 20 Mar 2011 from <http://www.dsm5.org/ProposedRevisions/Pages/InfancyChildhoodAdolescence.aspx>.
- Anstendig, K. D. (1999). Is selective mutism an anxiety disorder? Rethinking its DSM-IV classification. *Journal of Anxiety Disorders*, 13, 417–434.
- Carlson, J. S., Mitchell, A. D., & Segool, N. (2008). The current state of empirical support for the pharmacological treatment of selective mutism. *School Psychology Quarterly*, 23, 354–372.
- Cleave, H. (2009). Too anxious to speak? The implications of current research into selective mutism for educational psychology practice. *Educational Psychology in Practice*, 25, 233–246.
- Kearney, C. A. (2010). *Helping children with selective mutism and their parents: A guide for school-based professionals*. Oxford: Oxford University Press.
- Sharp, W. G., Sherman, C., & Gross, A. M. (2007). Selective mutism and anxiety: A review of the current conceptualization of the disorder. *Journal of Anxiety Disorders*, 21, 568–579.
- Viana, A. G., Beidel, D. C., & Rabian, B. (2009). Selective mutism: A review and integration of the last 15 years. *Clinical Psychology Review*, 29, 57–67.

Selective Serotonin Reuptake Inhibitors (SSRIs)

- Antidepressants

Self-Advocacy

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Synonyms

Advocacy; Autonomy; Efficacy; Independence; Self-determination; Self-direction; Self-empowerment; Self-reliance

Definition

Self-advocacy refers to the skills of people from within a specific group to integrate knowledge

about their civil rights, relevant laws, and their relative adaptive strengths and weaknesses to effectively communicate their perspectives and needs to the overall community (on an individual level to broader social systems). With regard to autism spectrum disorders (ASD), self-advocacy movements developed in reaction to the impingement of civil rights experienced by people with developmental disabilities (Ward and Meyer 1999). A few advancements that have arisen from these movements include integration with mainstream society, access to education and independent living, and, overall, being recognized as a full and equal member of society. Contemporary notions of self-advocacy refer to people on the autism spectrum (including their families and caregivers) using and developing skills to effectively communicate their overall needs from personal (e.g., a student expressing a need for classroom accommodations to the teacher) to broader social issues (e.g., groups of people with ASD collaborating with organizations and lobbying to develop public policies to prevent discrimination; Turnbull and Turnbull 1985).

Self-advocacy requires complex organizational skills (e.g., personal decision-making, goal-setting) and understanding of social institutions (e.g., navigating the political system). These skills are seen as crucial to developing a strong sense of agency and empowerment within the ASD population. Direct training in self-advocacy to promote active life participation has been proposed by some as an integral aspect of improving the lives of people with ASD that should begin at a young age (Shogren and Turnbull 2006). In particular, receiving opportunities to make informed choices and exercise decision-making is regarded as the foundation for self-advocacy (Turnbull and Turnbull 2001). Some have also argued that self-advocacy requires direct instruction (e.g., workshops) and guided experience to develop necessary skills and enter related leadership roles (Caldwell 2010). It should be noted that there is some tension between notions of self-advocacy that involve disclosure of an ASD diagnosis to others (Davidson and Henderson 2010) and concepts of social inclusiveness that disparage the use

of labels as a means of oppression and social control (Bagatell 2010).

See Also

- [Adulthood, Transition to](#)
- [Advocacy](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Normalization](#)

References and Reading

- Bagatell, N. (2010). From cure to community: Transforming notions of autism. *Ethos*, 38(1), 33–55.
- Davidson, J., & Henderson, V. L. (2010). “Coming out” on the spectrum: Autism, identity and disclosure. *Social and Cultural Geography*, 11(2), 155–170.
- Joyner Hayne, R. E., Sibley, K., Shore, S. M., Meyer, R., & Schwartz, P. (2004). *Ask and tell: Self-advocacy and disclosure for people on the Autism spectrum*. Shawnee Mission: Autism Asperger.
- Shogren, K. A., & Turnbull, A. P. (2006). Promoting self-determination in young children with disabilities: The critical role of families. *Infants and Young Children*, 19(4), 338–352.
- Ward, M. J., & Meyer, R. N. (1999). Self-determination for people with developmental disabilities and autism: Two self-advocates’ perspectives. *Focus on Autism and Other Developmental Disabilities*, 14(3), 133–139.

Self-Care

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Definition

Self-care is a subset of adaptive skills and activities of daily living that encompass (a) eating/feeding/drinking, (b) personal hygiene/grooming, (c) toileting, and (d) health/medication management. Many individuals with autism need systematic, intensive teaching in self-care skills due to

deficits in language and attention skills, interfering behaviors, and/or sensory impairments. Teaching self-care skills should occur naturally during daily routines, in all environments. Assessments are done to determine current abilities, strengths, and likes as well as frustrators and limitations. Targeted skills may be designated by participants of an interdisciplinary team. A longitudinal and functional approach should be taken when planning a self-care skill program. The steps for each self-care skill need to be broken down and clearly defined: (a) specifying the target skill, (b) task-analyzing the skill, (c) systematically instructing the skills training, (d) evaluating progress of the learner, and (e) making program modifications as needed. To teach effectively, it is helpful to define the style in which the individual with autism learns best, whether it is visual, sounds, words, touch, and/or smell. The instructional design should also consider the use of physical and verbal cues, behavioral support, generalization and maintenance, and reinforcement.

Techniques to Assist in Learning Self-care Skills

Visual Aids

Remember kids on the autism spectrum usually learn better with both visual and verbal information. Try very simple illustrations using stick figures and show the step-by-step actions. While we take getting dressed for granted, putting a shirt on is actually a long sequence of complicated movements for any young child. Break the process into its smallest components and illustrate each one. For example, step-by-step illustrations for putting on shoes, brushing teeth, and toileting can be stuck to the relevant wall for your child to follow. You can tap each picture to prompt your child on each step.

Social Stories™

Try using these illustrations in a social story, a powerful technique for learning new skills. Social stories are an excellent way to introduce the

concept of a new skill, especially when a child dislikes disruption to routines.

Applied Behavior Analysis

One of the most effective interventions for autistic disorders is applied behavior analysis. It uses a simple ABC model for learning new behaviors:

- Antecedent – a request by the parent or therapist
- Behavior – the child's response (or lack of response) to the request
- Consequence – what happens as a result of the behavior, i.e., praise for success

First, the skill to be learned is broken down into the smallest units for easy learning. For example, a child learning to brush teeth independently may start with learning to unscrew the toothpaste cap. Once the child has learned this, the next step may be squeezing the tube, and so on.

See Also

- ▶ [Age Appropriate](#)
- ▶ [Daily Living Skills](#)
- ▶ [Self-help Skills](#)

References and Reading

- Anderson, S. R., Jablonski, A. L., Thomeer, M. L., & Knapp, V. M. (2007). *Self-help skills for people with autism*. Bethesda: Woodbine House.
- Baker, B. L., & Brightman, A. J. (2004). *Steps to independence: Teaching everyday skills to children with special needs* (4th ed.). Baltimore: Paul H. Brookes.
- Minshawi, N. F., Ashby, I., & Swiezy, N. (2009). Adaptive and self-help skills. In J. L. Matson (Ed.), *Applied behavior analysis for children with autism spectrum disorders*. New York: Springer.
- National Research Council. (2001). Committee on educational interventions for children with autism. In C. Lord & J. P. McGee (Eds.), *Educating children with autism*. Washington, DC: Division of Behavioral and Social Sciences and Education, National Academy Press.
- Shea, V., & Mesibov, G. B. (2005). Adolescents and adults with autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive*

developmental disorders, Vol. 1: Diagnosis, development, neurobiology, and behavior. Hoboken: Wiley.

Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales* (2nd ed.). Circle Pines: American Guidance Service.

Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know.* Hoboken: Wiley.

Self-Contained Classroom

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Synonyms

[Life skills classroom](#); [Separate classroom](#); [Special education classroom](#)

Definition

The term “self-contained classroom” refers to a classroom, where a special education teacher is responsible for the instruction of all academic subjects. The classroom is typically separated from general education classrooms but within a neighborhood school. A self-contained classroom is a special education placement that falls near the middle of a continuum of program options that range in restrictiveness, where the general education classroom is least restrictive and a hospital or a homebound placement is most restrictive. Student-to-teacher ratios in self-contained classrooms are usually smaller than in general education classrooms and other less restrictive special education placements such as resource classrooms. Children who are placed in self-contained classrooms often have multiple, intensive support needs and require a comprehensive and highly structured educational and/or behavioral program. Children who receive the majority of their instruction in self-contained classrooms may be

integrated with their peers for music, physical education, and art classes. Even though the concept of self-contained classrooms was derived from the Individuals with Disabilities Education Act (IDEA), there is no federal definition of self-contained classroom, and the term is not mentioned in the law.

Self-contained classrooms have been advocated for a number of different student groups. Many students with autism receive their instruction in self-contained classrooms because these classrooms are led by teachers who have specialized training to instruct this population. Similar arguments have been made for self-contained classrooms for students identified as gifted and talented and students with emotional and/or behavioral disorders.

An alternate definition of self-contained classroom exists. In some contexts, self-contained classrooms are more broadly defined as any classroom where a single teacher teaches all subjects. This definition applies to most elementary school classrooms but is distinguished from team teaching models, where each teacher of a grade teaches a single subject to each class in rotation.

See Also

- [Academic Supports](#)
- [Inclusion](#)
- [Special Education](#)

References and Reading

- Chen, G. (2009). Understanding self-contained classrooms in public schools. *Public School Review*. Retrieved from <http://www.publicschoolreview.com/articles/73>
- Heward, W. L. (2009). *Exceptional children: An introduction to special education* (9th ed.). Columbus: Pearson.
- Mastropieri, M. A., & Scruggs, T. E. (2004). *The inclusive classroom: Strategies for effective instruction*. Columbus: Pearson.
- Van Tassel-Baska, J., Willis, G. B., & Meyer, D. (1989). Evaluation of a full-time self-contained class for gifted students. *Gifted Child Quarterly*, 33, 7–10.
- White, S. W., Scahill, L., Klin, A., Koenig, K., & Volkmar, F. R. (2007). Educational placements and service use

patterns of individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 1403–1412.

Wright, W. D., & Wright, P. D. (2007). *Wrightslaw: Special education law* (2nd ed.). Hartfield: Harbor House Law Press.

Self-Determination

► Self-Advocacy

Self-Directed Support Corporation Supports

► Entrepreneurial Supports

Self-Direction

► Self-Advocacy

Self-Employment Supports

► Entrepreneurial Supports

Self-Empowerment

► Self-Advocacy

Selfemra

► Fluoxetine

Self-Help

► Daily Living Skills

Self-Help Skills

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Definition

Self-help skills are a subset of a larger repertoire of daily living skills, sometimes called activities of daily living (ADLs). Specifically, the term “self-help” usually refers to the following basic areas of independent behavior:

- *Eating/Feeding and Drinking*: Skills may include holding and using utensils properly, drinking without spilling, eating a variety of foods, proper use of a napkin, table manners, and other mealtime routines.
- *Grooming*: This area includes skills such as brushing hair, shaving, and dressing (e.g., selecting clothing, putting clothes on and off without assistance, and managing fasteners).
- *Personal Hygiene*: Skills include those such as bathing, brushing teeth, washing hair, and applying deodorant.
- *Toileting*: Skills related to toileting include managing clothing, cleaning oneself, as well as overall bowel and bladder management.

These may be considered the primary critical skill areas in the first 10 years of life and form the foundation for other skills to follow (Anderson et al. 2007); more complex skills will be required for adolescents and adults.

Self-help skills are a set of basic skills needed in order for a person to live independently (Volkmar and Wiesner 2009) and to succeed both in work and leisure as well as socially. Individuals with autism spectrum disorders (ASD) often have difficulty acquiring self-help skills and may need targeted instruction in this area of development. Critical areas of self-help and the importance of specific skills may

fluctuate with age and setting. For example, for a young child, it will be important to focus on basic feeding, eating, dressing, and toileting skills, whereas, a child in elementary school may be focused on lunchtime routines in several environments (e.g., home and school cafeteria) and independent use of a school backpack. Puberty and the teen years bring on new hygiene and self-care issues such as using deodorant, shaving, and managing monthly menstrual periods (Sicile-Kira 2006).

As young people with ASD transition beyond the school doors and into the community, self-help skills become more nuanced. For example, a person needs to go beyond just getting dressed to making the appropriate choice of clothing for a job interview. Grooming and personal hygiene routines become skill sets tied to one's self-awareness of needs and the community/work setting.

As demands fluctuate, the professionals who are identifying and assisting with the development of these skills (e.g., educators, psychologists, occupational therapists) may be multiple and will also change over time (Anderson et al. 2007).

The attainment of basic self-help skills is critical in order for individuals to participate as independently as possible in home, community, school, and work settings. Individuals with ASDs often show significant deficits in adaptive skills relative to their age and cognitive ability, and the gap between cognitive ability and adaptive skills frequently increases with age (Minshawi et al. 2009). Specific deficits have been noted in speaking, dressing, reading, the ability to comply with requests, socialization, play skills, and toileting (Scheuermann and Webber 2002). In addition to reducing dependence on caregivers, possessing adequate self-help skills is critical for maintaining health and social acceptance (Anderson et al. 2007; Scheuermann and Webber 2002). Scheuermann and Webber (2002) identified toileting, eating, dressing, personal grooming, and hygiene as the most essential self-help skills for increasing social acceptance, noting that peer rejection is often higher if individuals appear dirty or disheveled.

Instrumental Activities of Daily Living (IADLs)

While basic self-help skills (feeding, dressing, eating, toileting, and grooming) allow a person with ASD to function more independently in the home/school setting, additional life skills beyond this realm are also essential. These skills are often referred to as instrumental activities of daily living (IADLs) and go beyond the basics of getting up in the morning, dressing, and eating breakfast. They are the skills that support life in the home and community and may demand more complex interactions than basic activities of daily living (BADLs)/self-help skills (AOTA 2014). IADL categories for consideration when looking at functional independent living may include:

- *Care of Others*: This area of IADLs includes activities such as pet care, selecting and supervising caregivers and support personnel, and child rearing. Pets in the homes of children with ASD have fostered social engagement and interactions for these youth (Autism Speaks 2015). Pet care may be a first consideration in the *care of others* realm for children and adults with ASD. As youth with ASD age, the role of self-advocacy intertwines with IADL tasks related to caregiver/support personnel.
- *Communication Management*: This area of IADLs focuses on telephone use, communication devices, use of personal digital assistants, and cellphone use. With the continuous change in the area of assistive technology (AT), these skills and devices need frequent review and assessment for effective and efficient use. Being able to advocate for one's needs in this area is also a target for intervention as a young user of these tools moves into the increasingly more digital work world. Tools such as the Student Self-Evaluation Matrix (QIAT 2018) are available to help young technology users be better problem solvers and advocates for their AT in settings beyond the high school arena.
- *Community Mobility*: While mobility in the self-help area usually refers to transfers from place to place, community mobility focuses on pedestrian mobility and public transportation use. Considerations regarding driving via

appropriate driving assessment are also included in this IADL area and are important to explore as teens with ASD transition to adulthood (Anderson and Foden 2009)

- *Financial Management*: This IADL area covers the realm from basic money use to banking and financial decision-making. Topics in this area include the ability to make financial decisions beyond those taught in the classroom setting. Persons with ASD need real-life practice in the community to acquire these skills. This area includes personal finance management as well as information on Social Security income (in the USA) and daily money handling skills for purchases and bills (VT Transition Guide – Financial) and should include understanding of relevant legislation related to financial issues (e.g., ABLE Act of 2014).

All of the above areas are related to the concepts of *self-advocacy and self-determination*. Young people with ASD need to learn to assess their own needs, be involved in decision-making on their own behalf, and demonstrate self-determination skills. Components of self-determination and self-advocacy that can be taught to persons with ASD involve skills in (1) personal goal setting, (2) choice making, (3) self-regulation, and (4) decision-making (Wehmeyer et al. 2010). Young adults with ASD need to combine all these skills to make life choices regarding self-care (managing medical needs, healthy lifestyle) and positive social interactions (e.g., community clubs, safe online social experiences).

It is important to regard these skills as important and teachable tasks for youth with ASD in the high school setting. While research in the area of evidence-based transition intervention strategies is still limited, several key findings regarding high school experiences for youth with ASD have been noted (Wehman et al. 2014). Best practices for students with ASD in high school should include more rigorous student participation in his/her transition planning, actual training in self-determination skills, paid work experiences while in high school, and involvement in student

mentoring programs with peers (Wehman et al. 2014).

Historical Background

Deficits in adaptive behaviors such as self-help skills have historically been included in definitions of mental retardation/intellectual disability and developmental disabilities. One of the earliest definitions to allude to deficits in self-help skills was Tredgold's (1937) definition of mental deficiency, which he defined as "a state of incomplete mental development of such a kind and degree that the individual is incapable of adapting himself to the normal environment of his fellows in such a way to maintain existence independently of supervision, control or external support" (p. 5).

The American Association on Intellectual and Developmental Disabilities (AAIDD) has included a criterion of adaptive deficits in each of the iterations of the definition of mental retardation it has published since 1961 (Beirne-Smith et al. 2006). Similarly, the definition of mental retardation/intellectual disability in the most recent edition of the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)* (American Psychiatric Association 2013) also includes the criterion of adaptive deficits related to self-help skills. Interestingly, the current DSM definition of autism spectrum disorder does not require deficits in adaptive behavior other than social skills for diagnosis.

Additionally, the legal definition of a developmental disability (which includes autism spectrum disorders), as defined by the Developmental Disabilities Assistance and Bill of Rights Act of 2000 (P.L. 106–402, pp. 7–8), does include deficits in self-help skills:

(A) In general, the term "developmental disability" means a severe, chronic disability of an individual that:

- (i) Is attributable to a mental or physical impairment or combination of mental and physical impairments
- (ii) Is manifested before the individual attains age 22

- (iii) Is likely to continue indefinitely
- (iv) Results in substantial functional limitations in three or more of the following areas of major life activity:
 - (I) Self-care, (II) receptive and expressive language, (III) learning, (IV) mobility, (V) self-direction, (VI) capacity for independent living, and (VII) economic self-sufficiency
- (v) Reflects the individual's need for a combination and sequence of special, interdisciplinary, or generic services, individualized supports, or other forms of assistance that are of lifelong or of extended duration and are individually planned and coordinated

Given the inclusion of adaptive deficits in the legal definition of developmental disabilities as noted above, there has recently been a push to closely examine the adaptive functioning of individuals on the autism spectrum who do not also meet criteria for an intellectual disability (i.e., those with average cognitive ability). Research has shown that even those individuals with ASDs who have average cognitive ability often exhibit significant deficits in adaptive behaviors including self-help. Thus, there has been a movement to consider adaptive functioning in addition to cognitive ability when determining services for individuals with ASDs (Chawarska and Volkmar 2005; Shea and Mesibov 2005).

Changes in federal law regarding special education have recently acknowledged the importance of self-help skills as a part of the more global functional skills repertoire. These functional skills are needed for persons with ASD to function as independently as possible in life. The reauthorization in 2004 of the Individuals with Disabilities Education Act (IDEA) included new requirements that a student's Individualized Education Program (IEP) targets both functional and academic skills. Functional goals are those outside the academic realm that include everyday routines in life. Because functional skill needs will vary from child to child, self-help skills may be addressed in an IEP, depending on the unique needs of the individual child (Howey 2008).

Current Knowledge

Challenges to Skill Acquisition

While the majority of children learn self-help skills such as bathing and dressing with ease, as noted above, many children with autism have great difficulty with this process and often require formal instruction in the area of self-help (Scheuermann and Webber 2002). Adults and adolescents with ASDs frequently exhibit significant deficits in self-help skills compared to their cognitive skills. For example, standardized scores for daily living skills on measures of adaptive behavior typically average anywhere from 15 to 30 points lower than IQ scores for individuals with ASDs who have average cognitive ability (Shea and Mesibov 2005). When compared to nondisabled children who have similar cognitive skills, children with autism spectrum disorders often have significantly lower scores on standardized measures of adaptive behavior (Chawarska and Volkmar 2005).

That children with ASDs have difficulty learning self-help skills is not surprising when the specific characteristics associated with autism are taken into account. Sensory issues can negatively impact learning self-help skills such as bathing or eating. Additionally, difficulties with sequencing and understanding social expectations may make learning self-help skills more difficult or less motivating (Shea and Mesibov 2005). Furthermore, Volkmar and Wiesner (2009) suggest that because children with autism tend to be more rigid and dependent upon specific contexts for learning, they have difficulty generalizing skills to new situations (e.g., bathing and dressing at home is a different skill than bathing and dressing at grandparent's house or in gym class). Thus, whereas a neurotypical child may only have to learn a skill once, a child with autism may have to learn the same skill many times in many different situations.

This challenge with needing step-by-step instruction can often be due to issues with praxis. Praxis is the ability one has to think of and plan new motor acts; it is also called motor planning. Functional praxis is based on adequate foundation skills in the areas of sensory processing,

neuromuscular control, and motor control (Cook 1991). Cognition also plays a part in one's ability to conceive, sequence, and perform a new motor skill (Tomchek 2001). Impairments in praxis are often noted in individuals with autism. Persons with autism may have motor planning issues related to motor skill but may also have other, less clearly understood factors contributing to praxis issues (Tomchek).

Another challenge in learning self-help skills that may relate specifically to children with autism spectrum disorders is the role of language and socialization. Anderson et al. (2007) state that typically developing children are often motivated to learn new self-help skills in order to gain approval from parents, peers, or others. Because many children with autism may not seek social reinforcement, they may be less motivated to practice and learn these skills. Additionally, while neurotypical children will often learn self-help skills naturally through observation, interaction, and caregiver modeling, this may be ineffective with a child who has an ASD. Rather, children with autism are more likely to require step-by-step formal instruction and tangible reinforcement in order to learn basic self-help skills. Furthermore, studies have shown that even when adolescents with autism know how to perform certain self-help skills, when left to function independently, they often simply *do not* do them, perhaps because many individuals with autism are not motivated by factors such as social acceptance (Shea and Mesibov 2005).

Commonly Used Assessment Tools

It is clear that from an early age, adaptive behavior, including self-help skills, is considered a critical area of development. In fact, Part C of the IDEA Amendments of 2004 mandates that states must provide services to children under age 3 who need early intervention services if they experience delay(s) in cognitive, physical, communication, social or emotional, or adaptive development. To this end, many curriculum-based and criterion-based measures include adaptive behavior/self-

help skills as one of the primary developmental domains assessed.

For both children and adults, self-help skills are routinely included in broader measures of adaptive or independent behavior. Four commonly used assessment measures are highlighted below:

- *Adaptive Behavior Assessment Scale-Third Edition (ABAS-3)*: The ABAS-3 provides a complete assessment of adaptive skills for persons from birth to 89 years. It covers the broad domains of conceptual, social, and practical and examines 11 specific skill areas, including self-care (feeding, personal hygiene, dressing). The ABAS-3 has five rating forms, each of which is designed for a specific age range and respondent. A General Adaptive Composite score is provided in addition to domain and skill area scores (Harrison and Oakland 2015).
- *Vineland Adaptive Behavior Scale-Third Edition (Vineland-3)*: The Vineland-3 is a measure of adaptive behavior in individuals from birth to 90 years. The Vineland-3 examines personal and social skills considered necessary for everyday living across three domains including communication, daily living skills, and socialization; it offers optional motor skills and maladaptive behavior domains as well. Within the daily living skills domain, specific focus is on personal care such as brushing teeth, feeding, toileting, dressing, and bathing. The Vineland-3 forms include the interview, parent/caregiver rating, and teacher rating; the measure provides an overall Adaptive Behavior Composite and domain scores (Sparrow et al. 2016).
- *Scales of Independent Behavior-Revised (SIB-R)*: The SIB-R is a comprehensive, norm-referenced tool that assesses 14 areas of adaptive behavior and 8 areas of maladaptive or problem behavior. One of the primary domains is personal living, which allows examination of eating, toileting, dressing, and self-care. The measure can be administered via interview or by checklist procedure and is appropriate for individuals from infancy through 80+ years. There are three versions of

the SIB-R available for use: full scale, short form, and early development form. The SIB-R provides a broad independence score as well as a cluster score for personal living (Bruininks et al. 1996).

- *Pediatric Evaluation of Disabilities Inventory* (PEDI): The PEDI is a clinical assessment tool used to assess the functional skills and performances of children from the ages of 6 months to 7.5 years old. It can also be used to determine the skills of children older than this age whose functional skills may be below those expected of a 7.5-year-old child with no disabilities. The PEDI measures abilities in three domains: self-care, mobility, and social function. Specific to self-care, the PEDI focuses on mealtime skills and utensil use, dressing, basic hygiene skills, and bowel and bladder management. The PEDI also includes a caregiver assistance scale, which provides information regarding the level of help a child needs to complete a domain-specific activity or skill. Information for the PEDI may be obtained via structured interviews, observations of the child, or professional judgment of therapists and/or teachers (Haley et al. 1992). This measure has been revised to be available as a computer adaptive test, the PEDI-CAT for use from birth through 20 years of age (Hayley and Coster 2010).

Teaching Self-Help Skills and the Use of Evidence-Based Interventions

Though children with autism spectrum disorders do not appear to learn and generalize the use of self-help skills as naturally as typically developing children, with formal instruction, many individuals with ASDs learn these skills quite well. In fact, self-help skills can be acquired relatively quickly with appropriate instruction because these skills often play to the strengths (and are less dependent on the weaknesses) of many children with autism. Specifically, learning self-help skills may involve concrete thinking, fixed sequential actions, and little necessary social interaction (Anderson et al. 2007).

As mentioned before, children with autism may have greater difficulty generalizing skills to different contexts and settings. Therefore, whenever possible, self-help skills should be taught in the same way and same environment as they will be performed. Furthermore, it is important to involve teachers, parents, and any additional caregivers in self-help skill training in order to ensure the child is taught the skill in the same way across contexts (Scheuermann and Webber 2002). Because any delay in the acquisition of self-help skills will most likely become more significant with age, it is important to begin teaching adaptive skills such as dressing and undressing, personal care, and toileting as early as preschool in order to promote independence in adolescence and adulthood (Volkmar and Wiesner 2009). Self-help skill acquisition may be “put off” as teaching self-help skills takes time and effort on the part of the teachers (this may often be the individual’s parents) and the learner. Resources are available to assist parents and other caregivers in teaching self-help skills; for example, Baker and Brightman (2004) provide strategies designed specifically to improve independence skills, including self-help, for children ages 3 to preadolescence. Sicile-Kira (2006) lists several strategies that help adolescents with autism to acquire basic self-help skills including the use of schedules, picture icons, task analysis, and video modeling.

Selected strategies that may be effective for teaching self-help skills are highlighted below; these intervention techniques have been effective for teaching a variety of skills to individuals with ASD (National Professional Development Center on Autism Spectrum Disorders 2011). Clearly, when selecting an evidence-based intervention, the choice should be determined by the need (s) and abilities of the individual and the target issues.

Behavioral Approaches

One method of teaching self-help skills that has had well-documented success is applied behavior analysis (ABA). The Board Certified Behavior

Analysts (BCBAs) work with caregivers to develop structured programs to teach children necessary skills using principles of behavior analysis and reinforcement (Anderson et al. 2007). ABA utilizes many behavioral techniques, such as discrete trials to teach self-help skills to children with autism. Discrete trials involve breaking complex tasks or concepts into simpler, more concrete tasks. In this method, the therapist may give the child a prompt or cue that signals what the child should do. When the child performs the desired response correctly, she/he is given a reward, which in ABA is referred to as a reinforcer (Volkmar and Wiesner 2009).

Another behavioral method of teaching self-help skills is called chaining. Chaining is similar to discrete trials in that it involves breaking a complex task into many basic tasks. The main difference between discrete trials and chaining is that in chaining, each small task serves as the prompt or signal to begin the next task and that each subsequent task serves as a reinforcer for the task that preceded it. There are two types of chaining: backward chaining and forward chaining. In backward chaining, the last task in the complex sequence is the first basic task that is taught; thus, teaching starts at the end and moves backward to the beginning of the complex task. First, the child is physically guided through all of the steps of the chain. The next time, physical guidance will be used until the last step of the chain. At that point, the parent or therapist will wait for the child to perform the task independently or with reduced prompting. When the child completes the task, reinforcement is given. Once the child has mastered the final task independently, she/he is ready to learn the second to last task in the chain. The next time through the chain, physical guidance will be stopped just before the second to last task. Then the child will independently complete the final two tasks. This continues until the child has mastered the entire chain independently. In forward chaining, the opposite occurs. Training begins with the first “mini-task,” and subsequent tasks are completed with physical guidance. Once the child masters the first task in the chain, she/he is then required to complete the first two tasks in the chain before

receiving physical guidance and so on until the entire chain is learned (Anderson et al. 2007).

Visual Supports

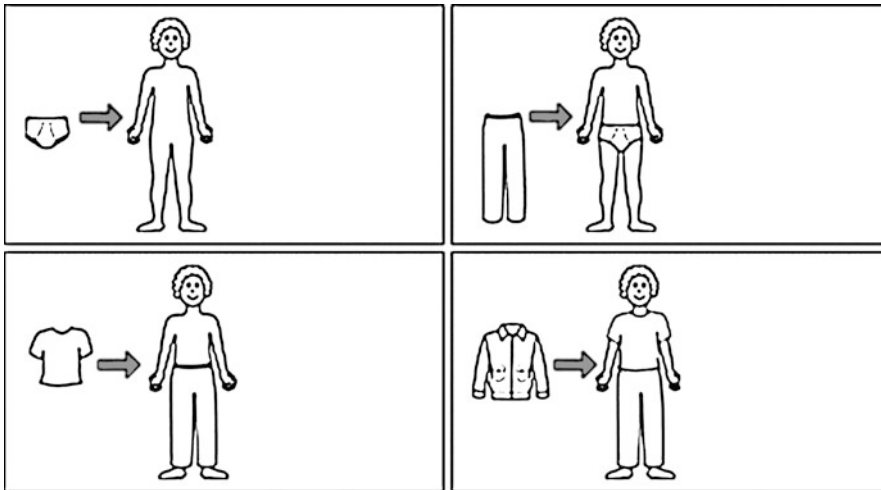
Progress can be further increased by using visual aids whenever possible in order to make the skills even less abstract (Kluth and Shouse 2009). Visual supports have a strong evidence base (Hume 2008) as practical, usable interventions for persons with ASD. Visual supports refer to objects, pictures, text, or a combination thereof provided to help a person understand and remember a concept or a task. Geis and Tomchek (2001) noted children with autism use their visual channels more effectively to process their world. Examples of visual supports include picture schedules, simple text checklists for routines, and charts combining words and symbols as behavioral reminders. Symbols for a dressing routine could include the following picture sequences for the daily routine (Fig. 1):

Sicile-Kara (2006) noted that picture icons were valuable in teaching routines to adolescents on the autism spectrum. Specific attention should be given to ensure that nonverbal teens know what the icons/symbols mean prior to their use in a dressing or self-care routine.

Social Narratives

Social narratives describe situations based on social and physical cues or teach specific social skills or behaviors. They can be individualized to meet the needs of the learner and may include pictures to assist the individual in gaining specific skills or responding appropriately (Collet-Klingenberg and Franzone 2008).

Perhaps the most widely known protocol, Carol Gray’s Social Story, is a comprehensive intervention used to explain a concept or skill to a person with autism using a specific format. A Social Story™ describes a situation, skill, or concept in terms of relevant social cues, perspectives, and common responses in a specifically defined style and format. The goal of a Social



Self-Help Skills, Fig. 1 Picture sequence of dressing routine. (Copyright © Do2Learn)

Story™ is to share accurate social information in a patient and reassuring manner that is easily understood by its audience; it is hoped that an individual's improved understanding of events and expectations may lead to more effective responses (the Gray Center, "What are Social Stories™?").

Video Modeling

Though it was noted above that children with ASDs often do not learn as readily from models as many typically developing children do, there are some types of modeling that are effective for teaching self-help skills to children with autism. One of these is video modeling (Charlop-Christy et al. 2000). Video modeling involves videotaping a model exhibiting a skill. The recording is then shown to the child with autism, who then learns to imitate the model. Children with autism are more likely to imitate actions they see in videos than those they see in person, perhaps because imitating live models has a greater social component. Additionally, the model in the video never alters his/her performance of the task as a live model will do, which can appeal to the child's preference for sameness. There is a particular type of video modeling that is gaining favor called video self-modeling. In video self-modeling, the child is

videotaped completing the task. The child can then watch the video and observe himself/herself completing the task; thus, the child serves as his/her own model. This technique is effective if the child is able to complete a task periodically, but is not consistent (Anderson et al. 2007). Additionally, it is theorized that by serving as his/her own model, the child may be more confident in his/her ability to correctly complete the task because the video serves as proof that she/he has been successful in it before (Buggey 2009).

Peer-Mediated Instruction and Intervention

Peer-mediated instruction and intervention (PMII) is an evidence-based practice that is best characterized by its use of competent peers to facilitate the learning and display of appropriate behaviors by the child with a less complex set of skills (Neitzel 2008). Peer-mediated strategies are built upon careful prompting and shaping of child behaviors by typical peers embedded in child-initiated interactions within natural contexts (Rogers 2000) and can be implemented with preschool-aged children as well as older students. PMII has been shown to have positive effects on academic, interpersonal, and personal-social

development (Maheady et al. 2001). Briefly, with PMII, once a particular skill is targeted as a need for the child with ASD, a skilled peer is selected and taught the necessary steps to model or teach the targeted skill with adult guidance and supervision. The peer then models the targeted skill in specific settings and situations.

- Reinforcement
- Self-Care
- Social Stories
- Structured Teaching
- Video Modeling/Video Self-Modeling
- Vineland Adaptive Behavior Scales (VABS)
- Visual Supports

Future Directions

Clearly, the acquisition of basic self-help skills should be viewed as a critical and necessary step in the overall development of individuals with autism spectrum disorders. An individual's ability to perform self-help skills has a strong influence on school, work, and social success. In addition, consideration of self-help skills, including more complex IADLs, may impact future decisions with respect to independent living arrangements as well as educational and vocational placements in adulthood. Thus, while it remains important to address an individual's needs in core areas of autism such as communication, behavior, and social skills, it is equally important to focus on developing self-help skills from a young age in order to allow children with ASDs to grow into independent adults with full lives.

See Also

- Adaptive Behavior
- Applied Behavior Analysis (ABA)
- Assessment of Functional Living Skills (AFLS)
- Behavior Analyst Certification Board
- Behavior Modification
- Behavior Plan
- Board Certified Associate Behavior Analyst
- Daily Living Skills
- Developmental Milestones
- Differential Reinforcement
- Functional Life Skills
- Guided Compliance
- Learning Styles
- Modeling
- Motivation
- Positive Behavioral Support

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed. (DSM-5)). Washington, DC: Author.
- Anderson, C., & Foden, T. (2009). Rules of the road: Driving and ASD. *Interactive Autism Network*. Retrieved 10 Jan 2017 from https://iancommunity.org/cs/adults/driving_with_asd
- Anderson, S. R., Jablonski, A. L., Thomeer, M. L., & Knapp, V. M. (2007). *Self-help skills for people with autism*. Bethesda: Woodbine House.
- AOTA. (2014). Companion to the AOTA classification codes for continuing education activities. Retrieved 4 Jan 2017 from <https://www.aota.org/~media/Corporate/Files/EducationCareers/APP/AOTAClassificationCodesforCE-Companion.pdf>
- Autism Speaks. (2015). More evidence that pets foster social skills in children with autism. Retrieved 4 Jan 2017 from <https://www.autismspeaks.org/science/science-news/more-evidence-pets-foster-social-skills-children-autism>
- Baker, B. L., & Brightman, A. J. (2004). *Steps to independence: Teaching everyday skills to children with special needs* (4th ed.). Baltimore: Paul H. Brookes Publishing.
- Beirne-Smith, M., Patton, J. R., & Kim, S. H. (2006). *Mental retardation: An introduction to intellectual disabilities* (7th ed.). Columbus: Pearson.
- Bruininks, R. H., Woodcock, R. W., Weatherman, R. F., & Hill, B. K. (1996). *Scales of independent behavior – Revised*. Rolling Meadows: Riverside Publishing.
- Buggey, T. (2009). *Seeing is believing: Video self-modeling for people with autism and other developmental disabilities*. Bethesda: Woodbine House.
- Charlop-Christy, M. H., Le, L., & Freeman, K. A. (2000). A comparison of video modeling with in-vivo modeling for teaching children with autism. *Journal of Autism and Developmental Disorders*, 30, 537–552.
- Chawarska, K., & Volkmar, F. R. (2005). Autism in infancy and early childhood. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Diagnosis, development, neurobiology, and behavior, Vol. 1). Hoboken: Wiley.
- Collet-Klingenberg, L., & Franzone, E. (2008). *Overview of social narratives*. Madison: The National Professional Development Center on Autism Spectrum Disorders, Waisman Center, University of Wisconsin.

- Cook, D. (1991). The assessment process. In W. Dunn (Ed.), *Pediatric occupational therapy: Facilitating effective service provision*. Thorofare: SLACK Incorporated.
- Developmental Disabilities Assistance and Bill of Rights Act. 45 U.S.C. 15001. (2000). Retrieved 16 Feb 2011 from <http://www.acf.hhs.gov/programs/add/adddocs/act.pdf>
- DO2Learn. (n.d.). Retrieved 11 Mar 2011 from www.do2learn.com
- Geis, R., & Tomchek, S. (2001). Integration of sensorimotor and speech language therapy. In R. Huebner (Ed.), *Autism: A sensorimotor approach to management*. Gaithersburg: Aspen.
- Gray Center. (n.d.). What are social stories™? Retrieved 11 Mar 2011 from www.thegraycenter.org
- "H.R. 647 – 113th Congress: ABLE Act of 2014". www.GovTrack.us. 2013. 12 Jan 2017. <https://www.govtrack.us/congress/bills/113/hr647>
- Haley, S. M., Coster, W. J., Ludlow, L. H., Haltiwanger, J. T., & Andrellos, P. J. (1992). *Pediatric evaluation of disability inventory (PEDI): Development, standardization, and administration manual*. Boston: Trustees of Boston University.
- Harrison, P. L., & Oakland, T. (2015). *Adaptive behavior assessment system, manual, and intervention planner* (3rd ed.). Torrance: Western Psychological Services.
- Hayley, S. M., & Coster, W. J. (2010). *PEDI-CAT: Development, standardization and administration manual*. Boston: CREcare, LLC.
- Howey, P. (2008). What you need to know about IDEA 2004: Present levels of functional performance & functional goals in IEPs. Wrightslaw. Retrieved 11 Mar 2011 from <http://www.wrightslaw.com/howey/iep.functional.perf.htm>
- Hume, K. (2008). *Overview of visual supports*. Chapel Hill: National Professional Development Center on Autism Spectrum Disorders, Frank Porter Graham Child Development Institute, The University of North Carolina.
- Kluth, P., & Shouse, J. (2009). *The autism checklist: A practical reference for parents and teachers*. San Francisco: Jossey-Bass.
- Maheady, L., Harper, G. F., & Mallette, B. (2001). Peer-mediated instruction and interventions and students with mild disabilities. *Remedial and Special Education*, 22(1), 4–14.
- Minshawi, N. F., Ashby, I., & Swiezy, N. (2009). Adaptive and self-help skills. In J. L. Matson (Ed.), *Applied behavior analysis for children with autism spectrum disorders*. New York: Springer.
- National Professional Development Center on Autism Spectrum Disorders. Retrieved 13 Apr 2011 from <http://autismpdc.fpg.unc.edu/>
- Neitzel, J. (2008). *Overview of peer-mediated instruction and intervention for children and youth with autism spectrum disorders*. Chapel Hill: National Professional Development Center on Autism Spectrum Disorders, Frank Porter Graham Child Development Institute, The University of North Carolina.
- Quality Indicators for Assistive Technology (QIAT) in Post Secondary Education. *Student self-assessment matrix*. Retrieved 21 Sept 2018 from <http://qiat-ps.org/collaborators/>
- Rogers, S. J. (2000). Interventions that facilitate socialization in children with autism. *Journal of Autism and Developmental Disorders*, 30(5), 399–409.
- Scheuermann, B., & Webber, J. (2002). *Autism: Teaching does make a difference*. Belmont: Wadsworth.
- Shea, V., & Mesibov, G. B. (2005). Adolescents and adults with autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Diagnosis, development, neurobiology, and behavior, Vol. 1). Hoboken: Wiley.
- Sicile-Kira, C. (2006). *Adolescents on the spectrum: A parent's guide to the cognitive, social, physical, and transition needs of teenagers with autism spectrum disorders*. New York: Penguin.
- Sparrow, S. S., Cicchetti, D. V., & Saulnier, C. A. (2016). *Vineland adaptive behavior scales* (3rd ed.). San Antonio: Pearson.
- Tomchek, S. D. (2001). Assessment of individuals with an autism spectrum disorder utilizing a sensorimotor approach. In R. Huebner (Ed.), *Autism: A sensorimotor approach to management*. Gaithersburg: Aspen.
- Tredgold, A. F. (1937). *A textbook of mental deficiency*. Baltimore: Woods.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know*. Hoboken: Wiley.
- VT Transition Guide for Young Adults with Autism & Developmental Disabilities – Financial. Retrieved on 10 Jan 2017 from <http://www.vttransitionguideasd.org/parents-resources/financial-parents-resources/>
- Wehman, P., Schall, C., Carr, S., Targett, P., West, M., & Cifu, C. (2014). Transition from school to adulthood for youth with autism spectrum disorder: What we know and what we need to know. *Journal of Disability Policy Studies*, 25(1), 30–40.
- Wehmeyer, M. L., Shogren, K. A., Zager, D., Smith, T. E. C., & Simpson, R. (2010). Research-based principles and practices for educating students with autism: Self-determination and social interactions. *Education and Training in Autism and Developmental Disabilities*, 45(4), 475–486.

Self-Injurious Behavior

- Stereotyped Movement Disorder
- Stereotypic Behavior

Self-Injurious Behavior (SIB)

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Definition

Self-injurious behavior (SIB) is behavior in which the individual inflicts injury on themselves. The range of potential self-injurious behavior is quite staggering and disconcerting. SIB may include head banging (the most common form) on walls, floors, or tables; finger, hand, or wrist biting; hair pulling; eye poking or gouging; hitting self; and scratching self. Many individuals display multiple types of SIB. SIB is “one of the most dangerous and debilitating behavior problems in the entire field of developmental disabilities” (Volkmar et al. 2009, p. 157).

SIB is a serious concern for caregivers of individuals with autism. The behavior may cause emotional reactions in families and service providers that are incompatible with reasoned practical intervention. Furthermore, SIB may lead to secondary health problems such as bruising, bleeding, infection, and head trauma that serve as setting events for more maladaptive behavior and in extreme cases may cause further neurological impairment. Compared with individuals with autism who do not exhibit SIB, individuals with autism who do exhibit SIB are more likely to be placed in residential treatment settings (Borthwick-Duffy et al. 1987).

SIB should not be confused with “non-suicidal self-injury” (NSSI), a syndrome recently proposed for inclusion in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association 2000). Whereas the purpose of neither syndrome of behaviors is resultant death, NSSI usually consists of repeated superficial self-injury with sharp objects done by typically developing adolescents between ages 15 and 19. SIB consists of the wide array of behaviors noted above done by individuals with developmental disabilities.

Current Knowledge

In the Diagnostic and Statistical Manual of Mental Disorders, SIB is not a defining feature of autism. It may not occur in an individual diagnosed with autism, and it is not limited to people with autism but may also present in individuals with mental retardation with or without a diagnosis of autism and in people with specific genetic disorders such as Lesch-Nyhan syndrome. Nonetheless, the prevalence of SIB in autism is much greater than in typically developing children (Dominick et al. 2007) and individuals with other developmental disabilities (Bodfish et al. 2000). SIB is more common in the severe and profound levels of mental retardation (Handen and Gilchrist 2006). Furthermore, the rate of SIB across individuals can range from thousands of times per day to once every few weeks. Generally, SIB seems to diminish as language development improves. Amelioration of SIB typically includes a variety of applied behavior analysis interventions and sometimes psychopharmacology.

Applied Behavior Analysis

The first step in applied behavior analysis is determination of variables controlling the behavior, a functional analysis. The controlling variables for SIB are as varied as the types of SIB, and there appears to be no causal relationship between a specific type of SIB and specific controlling variables. Many variables maintain problem behavior (Martin and Pear 2007), and these variables may develop, shape, or maintain SIB (Durand and Crimmins 1988; Minshawi 2008).

Functional Analysis

Some SIB is maintained by social positive reinforcement. For example, typical caregiver reactions might include attention in the form of hugging, getting close to the person, reprimands, or verbal attempts to sooth the person (Iwata et al. 1994). This is identified when functional analysis consistently shows that social attention follows behavior or when the person with autism smiles at, looks at, or approaches caregivers just behavior emitting SIB.

Some SIB is maintained by social negative reinforcement. In this scenario, antecedents to the SIB might be occasions where caregivers present demands that the person considers aversive. This may include instructions to complete a task or chore, say something or do schoolwork that is specifically difficult for the individual. Following this, the person may emit SIB that is reinforced (negative reinforcement) when the instruction or demand is withdrawn. Often, SIB is disruptive to the setting and results in such demands being withdrawn or postponed. SIB also causes some caregivers to stop making demands (Volkmar and Wiesner 2009). This is identified when functional analysis consistently shows SIB occurs when the person is presented with demands that are withdrawn following the SIB.

Some SIB is maintained by internal sensory positive reinforcement, sometimes referred to as automatic reinforcement. For example, SIB may lead to sensations or perceptions that include odd visual patterns, repetitive sounds, and tactile or kinesthetic sensations (Guess and Carr 1991). Some individuals with autism have sensory deficits that include high thresholds for pain, and there is speculation that some SIB is reinforced because it is experienced as pleasurable self-stimulation rather than aversive pain (Boucher 2009). This is identified when functional analysis consistently shows that SIB occurs when the individual with autism is alone, in the absence of external positive reinforcement or escape/avoidance conditions. Caregivers may report that the SIB is unrelated to any task or situation or that it occurs when there is a lack of external stimulation. The section on neurobiological research below briefly addresses such models for SIB.

Conversely, some SIB seems elicited by antecedent stimuli rather than maintained by reinforcing consequences. Although the sensory organs of individuals with autism are not compromised, some individuals with autism appear hypersensitive to stimulation (Dawson and Toth 2006). Sometimes this is referred to as sensory dysfunction that includes auditory, tactile, or visual defensiveness. Therefore, SIB may be

elicited by loud, high-pitched, or sudden noise (e.g., other children squealing, fire alarms, or loud music); certain textures of clothing or food; or lights that blink, shine directly in a person's face, or have high wattage. Such SIB may appear highly emotional in nature and is identified when functional analysis consistently shows that SIB occurs when specific stimuli or circumstances present or in settings paired with those stimuli. Conceptually, there are occasions when this is consistent with the logic of negative reinforcement described above. However, this is not social negative reinforcement but rather internal sensory negative reinforcement. For example, if playground noise at school recess elicits SIB by a child, it might be reasonable to assume that the noise is aversive to the child. Thus, when stimuli paired with going to playground are presented, such as after a specific class or walking down the hallway in line toward the playground, the child with autism may emit SIB that causes adults to disallow the "fun" playground opportunity, when in fact the SIB is negatively reinforced by escape/avoidance of the noisy playground.

Treatment

The second step in applied behavior analysis is manipulation of the variables that function to control the SIB and/or shaping desirable behavior that serves the same function, thereby decreasing the strength of SIB. The treatment is selected based on the functional assessment described above as well as the individual characteristics of the individual with autism, including IQ level. Typically, procedures are combined and highly individualized (Kahng et al. 2002; Volkmar et al. 2009).

If SIB is maintained by social positive reinforcement, then that reinforcement contingency is altered. One potential procedure is simply ignoring the person when they emit SIB, known as extinction. This is a deceptively complicated procedure because it is very difficult for caretakers, especially parents, to wholly ignore SIB. If caretakers occasionally give attention to SIB, whether or not it is intentional, then the SIB may become stronger because intermittent

reinforcement can be more powerful than continuous reinforcement. Iatrogenic effects may also include temporary increase in the rate of SIB at the outset of treatment (an extinction burst) and spontaneous recovery when the individual is not in the same setting where extinction was implemented. An alternative procedure allows caretakers to give attention to the person when they are not engaging in SIB, called differential reinforcement of zero responding (DRO). In this case, social attention is presented only if the SIB does not occur during a specified period. Typically, the length of time chosen is the average time interval between behavior occurrences. For example, if a child emits SIB four times per hour, the procedure is arranged to provide social attention every 15 min that the child does not emit SIB. Then, the schedule of reinforcement is thinned and social attention is presented every 20 min that the child does not emit SIB, and so forth. A disadvantage of extinction and DRO procedures is that they do not teach appropriate behavior, and sometimes the overall rate of reinforcement remains low if SIB continues. A third alternative is a procedure where caretakers give social attention for behavior that is incompatible with SIB, a response that cannot be emitted at the same time as SIB. This is differential reinforcement of incompatible behavior (DRI) or differential reinforcement of alternative behavior (DRA). For example, if functional analysis reveals that SIB is emitted when faced with challenging academic requirements, teachers may give social attention whenever the child requests help in that context. The hope is that as requests for help increase, SIB decreases.

If SIB is maintained by social negative reinforcement, once again the functional contingency between the behavior and consequence is altered. In some cases, caretakers simply insist, or persist, when presenting demands such that the SIB does not function to allow escape from demands. Sometimes, this procedure is impractical because the SIB compromises the individual's health. An alternative is similar to the DRI procedure described above. If a child exhibits SIB when demands are presented, shaping the child to sign

"no" and then withdrawing the demands contingent upon the signed "no" may reduce SIB because the function of "no" is identical to the function of SIB. The potential disadvantage is the remaining problem of a child who escapes demands. In some cases, cost-benefit analysis will reveal that the advantages of reduced SIB outweigh the disadvantages of escape from work. The DRI procedures described here are consistent with data illustrating the negative correlation between language development and SIB. Among individuals with autism, as functional communication improves in rate and quality, SIB decreases.

SIB maintained by internal sensory positive reinforcement can be challenging to manage because the functional relations between SIB and its consequences may be difficult to identify, particularly among individuals with communication deficits. Enriching the quality and intensity of surrounding environments may attenuate the function of SIB that is reinforced by auditory or visual outcomes. Blocking may disrupt the reinforcement relationship between SIB and tactile stimulation. For example, if functional analysis of a child's SIB determines that the reinforcer for scratching face is tactile stimulation in the absence of external positive reinforcement or escape/avoidance conditions, gloves on the child's hands may reduce scratching because it no longer functions to produce the tactile consequence.

If SIB is elicited by antecedent stimuli rather than maintained by reinforcing consequences, then structural changes in the environment may be warranted. Sometimes changes in the environments are practical and inexpensive. For example, if SIB is elicited by the sound of the old electric pencil sharpener, a cost-benefit analysis may reveal that the most parsimonious solution is a new sharpener with a less abrasive sound or moving the sharpener to a different location. Similar to functional communication training described above, SIB may be weakened by teaching the individual with autism an adaptive response such as signing, "I don't like that noise," and then eliminating the sound contingent upon that

communication. In this manner, the signed communication replaces the function of the SIB. Systematic desensitization incorporates gradual exposure to stimuli that elicit SIB. For example, if the hustle and bustle of the local mall elicits SIB, then numerous short trips to the mall, with incremental increases in the length of each visit, may help habituation to the mall.

Punishment

Sometimes, punishment is used when SIB is recalcitrant or severe, although there are disadvantages to punishment procedures. The presentation of punishing stimuli (positive punishment) contingent upon SIB has included water mists, visual screening, aversive tastes and odors, and electric shock. The removal of reinforcing stimuli (negative punishment) contingent upon SIB has included timeout and response cost (the removal of enjoyable items and tokens or opportunities to engage in preferred activities). One advantage of punishment is that it can be used even if functional analysis fails to determine the function of the SIB, particularly SIB maintained by internal sensory positive reinforcement. However, the disadvantages of punishment are well documented. Punishment does not teach appropriate behavior, so one form of SIB may be weakened only to be replaced by another form of SIB that serves the same function. Sometimes punishment elicits emotional responses (sad mood, fear, anger) that are incompatible with learning. Individuals who receive punishment may learn to avoid the people who administer the punishment and the settings where it is administered. Timeout may work for some children with autism, but timeout may be ineffective if the behavior of the individual with autism is negatively reinforced by escape from social interaction or positively reinforced by internal sensory events or the opportunity to engage in stereotypic behavior.

Neurobiological Research and Psychopharmacological Treatment

Neuroleptics have long been used for treatment of SIB, as well as aggression and stereotypical/repetitive behavior. Risperidone is the most commonly

used atypical antipsychotic that targets dopamine-blocking agents (Chavez and Chavez-Brown 2006). Potential side effects include fatigue/sedation, increased appetite, and corresponding weight gain and drooling. Recently, the efficacy of olanzapine indicates some improvement of SIB, but side effects such as dystonic reaction (distorted twisting/movement of part or all of the body) limits its usefulness in children (Handen and Gilchrist 2006).

Neurobiological models have evaluated the potential relevance of the endogenous opioid peptide hypothesis, which suggests that SIB results in increased levels of beta-endorphin levels. The outcome is elevated pain threshold and tolerance of, or reward by, pain. This is consistent with the suggestions that some SIB is maintained by internal sensory positive reinforcement wherein SIB is reinforced because it is experienced as pleasurable. Naltrexone is an opioid antagonist that may decrease SIB in some individuals (ElChaar et al. 2006). Potential side effects include drowsiness, decreased appetite, and aggression.

Some have suggested that SIB is really a manifestation of internal compulsive thoughts, consistent with obsessive-compulsive disorder (Boucher 2009). An outcome of this proposal is limited research indicating the efficacy of selective serotonin reuptake inhibitors (SSRIs) such as Zoloft and Prozac (Aman et al. 1999).

See Also

- [Challenging Behavior](#)
- [Neuroleptics](#)
- [Reinforcement](#)

References and Reading

- Aman, M. G., Arnold, L. E., & Armstrong, S. C. (1999). Review of serotonergic agents and perseverative behavior in patients with developmental disabilities. *Mental Retardation and Developmental Disabilities*, 5, 279–289.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, 4th ed.: Text revision*. Washington, DC: Author.

- Bodfish, J., Symons, F., & Parker, D. (2000). Varieties of repetitive behavior in autism: Comparisons to mental retardation. *Journal of Autism and Developmental Disorders*, 30, 237–243.
- Borthwick-Duffy, S. A., Eyman, R. K., & White, J. F. (1987). Client characteristics and residential placement patterns. *American Journal of Mental Deficiency*, 92, 24–30.
- Boucher, J. (2009). *The autistic spectrum: Characteristics, causes and practical issues*. Los Angeles: Sage.
- Chavez, B., & Chavez-Brown, M. (2006). Role of risperidone in children with autism spectrum disorder. *The Annals of Pharmacotherapy*, 40, 909–916.
- Dawson, G., & Toth, K. (2006). Autism spectrum disorders. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology, Vol. 3: Risk, disorder, and adaptation* (2nd ed., pp. 317–357). Hoboken: Wiley.
- Dominick, K., Davis, N. O., Lainhart, J., Tager-Flusberg, H., & Folstein, S. (2007). Atypical behaviors in children with autism and children with a history of language impairment. *Research in Developmental Disabilities*, 28, 145–162.
- Durand, V. M., & Crimmins, D. B. (1988). Identifying the variables maintaining self-injurious behavior. *Journal of Autism and Developmental Disorders*, 18, 99–117.
- ElChaar, G., Maisch, N., Augusto, L., & Wehring, H. (2006). Efficacy and safety of Naltrexone in pediatric patients with autistic disorder. *The Annals of Pharmacotherapy*, 40, 1086–1095.
- Guess, D., & Carr, E. (1991). Emergence and maintenance of stereotypy and self-injury. *American Journal on Mental Retardation*, 96, 299–319.
- Handen, B. L., & Gilchrist, R. H. (2006). Mental Retardation. In E. J. Mash & R. A. Barkley (Eds.), *Treatment of childhood disorders* (3rd ed., pp. 411–454). New York: Guilford Press.
- Iwata, B. A., Pace, G. M., Dorsey, M. F., Zarcone, J. R., Vollmer, T. R., Smith, R. G., et al. (1994). The functions of self-injurious behavior: An experimental epidemiological analysis. *Journal of Applied Behavior Analysis*, 27, 215–240.
- Kahng, S., Iwata, B. A., & Lewin, A. B. (2002). Behavioral treatment of self-injury, 1964–2000. *American Journal on Mental Retardation*, 107, 212–221.
- Martin, G. M., & Pear, J. (2007). *Behavior modification: What it is and how to do it*. Upper Saddle River: Pearson/Prentice Hall.
- Minshawi, N. F. (2008). Behavioral assessment and treatment of self-injurious behavior in autism. *Child and Adolescent Psychiatric Clinics of North America*, 17, 875–886.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: what every parent, family member, and teacher needs to know*. Hoboken: Wiley.
- Volkmar, T. R., Sloman, K. N., & Samaha, A. L. (2009). Self-injury. In J. L. Matson (Ed.), *Applied behavior analysis for children with autism spectrum disorders* (pp. 157–173). New York: Springer.

Self-Management Interventions

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Definition

Self-management interventions are multi-component behavioral strategies that emphasize the monitoring and regulation of behaviors and reinforcements (rewards). The core elements of self-management interventions involve (a) self-observation of a targeted behavior, (b) self-recording, and (c) delivery of a reinforcer. The identified person in treatment (e.g., client, child, or adult) is given a central role in managing behavior change. The client is taught to observe or monitor (self-monitor) and track (self-record or self-manage) his or her own behaviors by using a recording method (tallying the occurrence of a target behavior on paper or with a counter device). When treatment goals are met, the client is expected to obtain a reward (reinforcement). An emphasis on systematically promoting the client to independently engage in these skills is an integral aspect of the intervention. Although behaviors are referenced as the outcome of change, self-management interventions have been examined and used for therapeutic changes in mood and cognitions. A review of the literature suggests they are potentially efficacious for increasing adaptive behaviors and reducing unwanted behaviors.

Historical Background

Self-management interventions have a long history as a set of therapeutic methods to promote

and maintain behavior change. Self-management interventions also have been referred to as self-monitoring (e.g., Kazdin 1974), self-control procedures (Nelson and Hayes 1981), or self-reinforcement systems (e.g., Mahoney 1970). These interventions have been used to target changes in behaviors (verbal and nonverbal) as well as emotions and cognitions. However, the argument in the field has been that internal, unobservable thoughts and emotions may be difficult to self-monitor due to challenges with substantiating the accuracy of internal processes. Therefore, overt, observable behaviors have been proposed to be more amenable to self-management interventions.

Most of the instrumental research and development in self-management interventions began in the late 1960s to early 1970s (Kazdin 1974; Mahoney 1970; cf. Korotitsch and Nelson-Gray 1999). The principles and methodology of self-management interventions developed from the field of behavioral psychology. It is strongly rooted in behavior modification principles and, later, cognitive-behavioral theories (Mahoney 1970). Since then, it has evolved and widely been adapted as a psychotherapeutic method for a range of clinical populations and conditions. Self-management interventions are often used in clinical settings and can be used alone as the primary therapeutic tool or integrated within a larger treatment package (e.g., Cognitive-Behavioral Therapy).

Self-management procedures are grounded on the notion that the client takes on the role of the “behavioral engineer” (Mahoney 1970). Historically, self-monitoring has been used for both assessment or information gathering and intervention purposes. Self-management for assessment purposes emphasizes the accuracy of a client’s self-monitoring to obtain and provide data on targeted behaviors (e.g., for diagnostic or treatment development purposes). In contrast, self-management interventions are often based on a triad of skills – self-monitoring, self-recording, and self-reinforcement (or for some pediatric populations, parent- or teacher-delivered reinforcement) – for the explicit purposes of behavioral change. The emphasis is on discerning the

expression of target behaviors rather than accuracy of self-recording. Much of the early work on self-management interventions included not only clinical applications but component analyses of the various variables involved in self-management interventions (cf. Kazdin 1974; Mahoney 1974). Since then, more of the contemporary research on self-management interventions with people with autism spectrum disorder (ASD) involves direct clinical application (e.g., Lee et al. 2007).

Rationale or Underlying Theory

Mechanisms for Behavioral Change. Self-management interventions have been hypothesized to emphasize the active role of the client in promoting and maintaining change. The client’s role in directing change is pivotal and is an integral mechanism in this intervention (Mahoney 1972). Clients determine whether behaviors will be increased or decreased to obtain a desired outcome (reward). As a result, emphasis is placed on self-management procedures to be gradually transitioned from therapist management of contingencies to the client (Mahoney 1970). Particular emphasis is placed on the client to learn to discriminate when the target behaviors occurred to obtain the reward. By doing so, the client develops self-awareness through contingency management skills (i.e., the notion that reward is only obtained contingent upon the expression of target behaviors). Some clients with poor impulse control can learn this association even if parents and teachers remain in control of the reinforcement.

Initiating Behavioral Change. At the very foundation of self-management intervention principles is the notion that self-monitoring achieves significant changes in behaviors, referred to as the “reactivity effect” (Kazdin 1974; Korotitsch and Nelson-Gray 1999). The premise is that self-monitoring alone leads to reactive behavioral effects and is a widely accepted aspect of self-management interventions. The client’s enhanced self-awareness of behavioral expression is proposed to be pivotal to initiating behavioral

change. Kanfer (1970) asserted that self-regulation is achieved by the active process of the client's own observations, evaluations, and obtainment of rewards. Awareness of one's own behaviors is proposed to promote the acquisition of adaptive behaviors. Self-awareness helps people recognize and appreciate new contingency relationships within their environment to develop adaptive behaviors (Mischel 1973).

Maintenance and Generalization of Behavior Change. These instrumental concepts underlying self-management have led to subsequent hypotheses that self-management interventions facilitate clients to independently manage their symptoms while decreasing their reliance on the administration of intervention by clinicians (Harrower and Dunlap 2001; Koegel et al. 1992). Self-management procedures are considered to be economical and easy to transport, making it relatively easy to use the intervention across a variety of settings. These factors have been hypothesized to promote generalization of behavioral change in ASD (Koegel et al. 1992). Also, it has been speculated to increase self-regulation of symptoms and has been referred to as a pivotal area that can elicit change in a variety of other symptom domains related to ASD (Koegel et al. 2001). The emphasis on increased client self-sufficiency in managing the treatment of symptoms seems to be especially pertinent in using this intervention for children with ASD and their families.

Goals and Objectives

The primary goal of self-management interventions is the development, maintenance, and generalization of adaptive behavioral change. The goal is to facilitate an awareness of the interaction between one's behavior and the surrounding environment, including contingency management of rewards (Mahoney 1970). In the treatment of ASD, generalization of desired changes across contexts that extend beyond the treatment setting is also an aim (Koegel et al. 1992). Another goal is to increase independence of symptom management by children (and families of children) or adults.

Treatment Participants

Self-management intervention research has been conducted with a wide range of ages and for a variety of presenting issues in both typically and atypically developing populations. The youngest age group in which this intervention has been targeted in ASD has been with preschool children (Newman et al. 2000); however, it has been applied more frequently with school-aged children (Lee et al. 2007). Children as young as 3 years of age appear to accurately self-monitor behaviors with brief training provided by a clinician. The intervention has been used and appears to be effective across children and adolescents with varying ASD diagnoses (i.e., autism, Asperger's disorder, and pervasive developmental disorder-not otherwise specified), functioning levels, and for treating a range of social, communication, and behavioral impairments (Apple et al. 2005; Pierce and Schreibman 1994; Wilkinson 2005). Some targeted behaviors have included independence skills such as completing daily living tasks (e.g., schedules), school performance, social skills, conversation skills, play skills, and managing challenging behaviors such as disruptive and repetitive behaviors. For example, in youngsters with ASD, self-management intervention was used to successfully increase behavioral variability in the play-related behaviors of three young children (preschool to 6 years of age) diagnosed with autism and mild cognitive impairments (Newman et al. 2000). In another study, school-aged children with ASD (ages 7, 12, and 13 years) demonstrated an increase in independent, appropriate (i.e., functional) toy play and a decrease in self-stimulatory behaviors that maintained in unsupervised and new play settings with the implementation of self-management intervention (Stahmer and Schreibman 1992). Youngsters with ASD (ages 5 and 6) demonstrated appropriate school performance behaviors in their classrooms and decreased off-task behaviors (e.g., running out of the classroom; Koegel et al. 1999). Self-management intervention as part of a video modeling treatment package effectively increased initiations of compliment giving in three young children with ASD (ages 4–5) in

comparison to video self-modeling alone (Apple et al. 2005). Children with ASD (ages 6 and 11) demonstrated increased appropriate verbal responses and decreased disruptive behaviors and social-communicative responses across clinical, home, and community settings with self-management intervention (Koegel et al. 1992).

In contrast, there are only a few research studies examining self-management interventions in adults with ASD. In one study, nine young adults diagnosed with high-functioning ASD (IQ above 80) between the ages of 17–25 years demonstrated a significant improvement in appropriate, self-initiated question-asking skills with the implementation of a self-management treatment package (Palmen et al. 2008). Further research in the application and efficacy of self-management interventions across the age span is necessary.

Treatment Procedures

Self-management interventions often involve the following fundamental treatment elements: (a) identifying and explicitly defining goals for behavioral change (to promote self-monitoring), (b) developing skills for the client to monitor and record the occurrence or nonoccurrence of identified behaviors (self-recording), and (c) facilitating the client to obtain rewards once the targeted behaviors have been demonstrated (self-reward). There exists variability in the implementation of self-management interventions, but most incorporate the basic elements presented above to some degree. For example, Newman et al. (2000) used all three elements of self-management intervention by having the children self-monitor their play behaviors with the experimenters, “record” when they displayed flexible play by taking tokens from a jar, and then trading in these tokens to obtain a reward. In another study, children viewed appropriate social initiations on a video and then learned to monitor and self-record initiations in the classroom setting with their peers by placing a check mark in a box that was printed on laminated paper for one child or pressing a button on a wrist counter for the other children (Apple et al. 2005). The children selected their reward before

beginning the self-management session for the day and approached their teachers once they had accumulated the designated number of points. Traditionally, self-management interventions are provided in an individual therapy setting that involves the client and the clinician across a range of settings such as the clinic or naturalistic environments within the community (e.g., Koegel et al. 1999). There is some preliminary evidence that it can also be implemented in a group treatment setting (e.g., Palmen et al. 2008).

More specific procedures for self-management among youth involve (1) identifying a range of child-preferred rewards and allowing children to designate items they would like to earn; (2) presenting them with a clear, individualized description of the targeted behavior using child-friendly language; (3) informing children of the number of “points” (target behaviors) necessary to obtain the reward; (4) teaching them to use self-management recording materials and track points through modeling and role playing (i.e., Self-Monitoring Training); (5) presenting children with opportunities to engage in targeted behaviors; (6) prompting them to mark a point once the target behavior was demonstrated; and (7) immediately presenting them with the selected reward once the agreed-upon number of points are achieved. Self-monitoring training (i.e., discrimination training) is often provided within self-management interventions. This involves a clinician teaching the client to be aware of behaviors by distinguishing desired targeted behaviors from undesired behaviors. Both types of behaviors are usually modeled by the clinician, and the client is asked whether such behaviors would warrant a point on the self-management sheet. The length of time providing the training appears to vary based on functioning levels and complexity of targeted behaviors. Training can be brief and has been accomplished in a 1-h period provided twice a week (e.g., Stahmer and Schreibman 1992).

Promoting independence with self-management skills is an overarching goal of this intervention method. In combination with the basic therapeutic elements, most interventions emphasize gradually facilitating the individual to develop autonomy with the intervention. Clinicians incrementally

decrease their involvement in implementing the intervention as clients demonstrate reasonable success with both self-management skills and treatment goals. One primary method has been for clinicians to gradually fade prompts provided to the client. These prompts, which can be verbal (e.g., reminders to mark the occurrence of a target behavior immediately after it occurred) or nonverbal (e.g., physically helping the child track responses by placing a tally mark on a sheet of paper), eventually transition from direct (e.g., a verbal reminder to check off a point each time the target behavior occurred) to indirect prompts (e.g., a brief reminder provided once at the beginning of the session for the client to as many “points” as possible). The frequency of prompts provided by the clinician and the degree of involvement in implementing the intervention are eventually tapered off. In one study, children were initially coached in using wrist counters to track their points (or responses) and reminded to press the counter each time the children asked an appropriate question. Gradually, such reminders and assistance were decreased as the children demonstrated success with using the self-management tools and asking appropriate questions during conversations (Koegel et al. 1992). Also, the frequency or duration of the target behavior necessary to obtain a reward is gradually increased. The rationale behind this process is to incrementally increase the targeted behavior in a manageable way to promote feelings of success in the client, maintain rapport, and preserve treatment motivation. In addition, the process involves extending the length of time before self-reinforcement occurs in order to promote maintenance of targeted behaviors in a naturalistic manner. Stahmer and Schreibman (1992) initially had children receive a point for demonstrating appropriate play behaviors for 30 s, which incrementally increased to 20 min in order to obtain a point. Koegel et al. (1992) initially rewarded children for obtaining 1 point for asking an appropriate question at the beginning of treatment. Eventually, children needed to obtain 30 points to obtain the reward.

There is preliminary evidence that recording and tracking one’s *own* behaviors results in increased behavior change in comparison to

being provided with *external* prompts that targeted behaviors have occurred. A study by Newman and Ten Eyck (2005) demonstrated that children’s social initiations increased dramatically when they kept track of their initiations by taking a penny out of a bowl compared to a condition in which the children with ASD were externally provided with a penny by adults for initiating social behaviors. In fact, it seems that complex social behaviors in children with ASD such as social-communicative initiations are significantly improved using self-management intervention (e.g., Apple et al. 2005) in comparison to other methods such as video self-modeling. There is strong evidence that recording accuracy does not seem to be crucial for treatment effects to occur (Lee et al. 2007). Children may occasionally forget to self-record after engaging in the targeted behaviors. Some researchers have remarked that this phenomenon may be indicative of the targeted behaviors becoming integrated into the children’s natural behavioral repertoire (Koegel et al. 1992; Newman et al. 2000). Conversely, there did not seem to be significant instances of children purposefully recording behaviors when they did not occur in order to obtain a reward. Therefore, the expectation for self-recording seems to be that clients should demonstrate reasonable accuracy during treatment.

The identification and use of highly motivating rewards is an integral aspect of this treatment. In the literature, these have included access to highly desired snacks or drinks, toys, activities (e.g., playing video games), or small prizes such as stickers. Rewards are conducted through some form of a preference assessment. This process involves the clinician identifying child-preferred items or activities by conducting a direct observation or gathering input from caregivers or from the children themselves. For example, in one study, children were provided with a prize bag that contained a range of child-preferred prizes (e.g., stickers, figurines, small toys; Apple et al. 2005). Highly motivating rewards can be reserved for self-management intervention in order to ensure reinforcing properties. Reinforcement immediately following the self-management intervention appears to be successful particularly when self-

management skills are just developing. Most studies of youngsters with ASD used contingent rewards immediately following a self-management session (e.g., Apple et al. 2005; Koegel et al. 1999). In one study of an adult with mild cognitive impairment, delayed reinforcement by accumulating a certain number of points to exchange for motivating items at the end of the week was effective, but somewhat less powerful in initially maintaining complex behaviors (e.g., Moore 2009).

Materials used to monitor behaviors consist of visual cues such as checklists with questions related to the occurrence of the target behavior (e.g., Did I stay in my seat?) or check boxes printed on paper for children to mark the occurrence of the desired behavior. Materials to record points consist of writing instruments such as pens, markers, or crayons (e.g., Stahmer and Schreibman 1992). Other materials to designate points included stickers or tokens (e.g., Newman et al. 2000). Devices such as timers or wrist watch counters have been used in an attempt to provide more discrete self-monitoring and decrease the need for clinician-delivered prompts. For example, a young adult with ASD used a digital watch to keep track of the time intervals (e.g., 5 min) in which he engaged in appropriate on-task behaviors when completing classroom assignments (Moore 2009). After the watch beeped when 5 min had passed, he marked whether he demonstrated on-task behaviors during that time interval by placing tally marks on a sheet of paper. In many studies, wrist counters have been used for children to track their targeted behaviors (e.g., Koegel et al. 1999). Overall, self-management materials appear to be manageable and successfully used across ages and functioning levels and can be tailored to meet the unique needs of each person while still maintaining treatment efficacy.

Several variables contributing to enhanced treatment effects have been identified. Treatment effects were enhanced when motivation for change within the client was high (Lipinski et al. 1975); immediate self-recording after each target behavior occurred rather than after a long delay (Mahoney et al. 1973); clients were aware of explicit performance standards/goals and

provided with feedback (Kazdin 1974); and positive self-reinforcement by obtaining a reward for engaging in the targeted behavior rather than retracting rewards for the absence of behaviors was used (e.g., giving oneself a punitive consequence by removing access to preferred items as used in a cost-response strategy; Humphrey et al. 1978). Additionally, younger children may need more guidance and explicit rehearsal of behaviors, facilitation procuring and obtaining rewards, and support from adults or other support people to learn and successfully use self-management procedures (Lee et al. 2007). A combination of the above variables appears to have greater effects than self-monitoring alone or another single component of self-management intervention by itself (Kazdin 1974).

Efficacy Information

Much of the literature on self-management interventions in ASD has been conducted using single-case research design. Single-case research models use rigorous and systematic methodology to closely examine behaviors when specific variables are manipulated (e.g., implementation of treatment) with a small number of participants (more scientifically rigorous studies include at least three participants). At this time, no large group design studies exist examining this intervention in ASD. Positive treatment effects of self-management interventions have been long established as either the sole treatment intervention or within the context of other treatment methods across studies. Lee et al. (2007) conducted a meta-analysis on 11 single-case studies to examine the effectiveness of self-management interventions among preschool and school-aged children with ASD. They concluded that, overall, self-management interventions are effective in promoting and maintaining positive behaviors. The average effect size was approximately 82% based on single-case design meta-analysis using percentage of nonoverlapping data (PND). PND is one type of meta-analyses to measure the effect size to synthesize single-case research. Higher PND scores reflect greater

treatment efficacy with scores ranging from 0% to 100%. Efficacy criteria range from ineffective (scores below 50%), questionable (50–70%), effective (70–90%), to highly effective treatments (90% or greater). The percentage of data points in the treatment phase that do not overlap with the highest value of data in the baseline phase (pretreatment) is calculated. Treatments employing self-management strategies were effective regardless of age group (preschool children vs. school-aged children), emphasis on specific self-management components (self-monitoring vs. self-reward components), or self-management materials (paper sheet vs. tokens). A trend for increased efficacy was observed in programs that were individualized for the children, coparticipants (e.g., adults or peers) provided support in monitoring the children's target behaviors, and self-management was conducted in natural settings (e.g., the home). Most studies also demonstrated maintenance effects (behavioral gains maintained even with the removal of self-management procedures) and generalization effects (behavioral gains demonstrated in novel settings or activities). For example, Stahmer and Schreibman (1992) demonstrated that prior to treatment, the three children with ASD in their study engaged in approximately less than 40% of appropriate toy play during each session. With intervention, all three children increased the percentage in which they demonstrated appropriate play to an average of 88% for child 1, 80% for child 2, and 96% for child 3. Generalization effects were evident in a novel setting once self-management procedures were faded. Two of the three children demonstrated appropriate play at levels similar to that during treatment after a 1-month follow-up (greater than 80% of appropriate play each session). In another study, two children with ASD demonstrated appropriate schoolwork performance during baseline (prior to treatment) approximately for 55% across sessions (Koegel et al. 1999). During intervention, levels of performance increased to approximately 95% for both children and maintained at this high level even after clinician involvement had been completely removed.

Outcome Measurement

Outcome measurements vary across studies and depend on the treatment targets. In general, given that many of the self-management treatment studies in ASD are based on single-case research design paradigms, outcome variables tend to be based on observable behaviors that have been recorded using frequency- (event recording) or interval- (duration) based variables gathered through direct observation or videotaped recordings. Frequency data refer to measuring the number of times behaviors occurred during a designated period of time. Interval recording systems measure the total number of intervals during which the targeted (e.g., 10-s interval) behaviors occurred within a specified overall time period (e.g., 10 min). Data are based on either the entire session or representative timed samples or data probes (e.g., last 10 min of a session). The manner in which this data is calculated varies and has consisted of frequency counts, rates, or percentages. Behaviors are carefully and specifically operationalized to accurately capture changes over time. Reliability data are typically reported in the literature to ensure that raters (typically at least two raters for a specified portion of the data) were both accurate and reliable in measuring behaviors. In one study, outcome measures consisted of the percentage of play opportunities in which children demonstrated appropriate, varied play (Newman et al. 2000). Children with ASD were presented with ten opportunities to play during each session. Each instance of varied play was totaled for the session and divided by the total number of opportunities presented and then multiplied by 100 in order to obtain a total percentage of opportunities the children engaged in varied play per session. Koegel et al. (1992) examined the frequency of appropriate and inappropriate verbal responses using an event recording system. The total number of these responses was summed for each data probe. The presence of disruptive behaviors was measured using an interval recording system. Each instance of disruptive behavior that was present during a 30-sec interval (from a 15-min data probe) was calculated and then divided by the total number of intervals to

obtain the percentage of 30-sec intervals in which disruptive behaviors were observed during each session. Additionally, a few self-management intervention studies also measured the occurrence of targeted behaviors in typically developing children to serve as a marker for the expected frequency or percentage of behaviors expected (e.g., Koegel et al. 1999).

Qualifications of Treatment Providers

Self-management interventions with the ASD population are typically provided by trained professionals with advanced degrees in psychology or education and training in evidence-based practices with the ASD population. Also trained professionals provide ongoing supervision and training in self-management interventions to support staff (e.g., school aides or teachers), parents, or behavioral clinicians without advanced degrees in order to provide treatments that can be maintained by various people in the child or adult's life.

See Also

- Behavior Modification
- Cognitive Behavioral Therapy (CBT)
- Habit Reversal

References and Reading

- Apple, A. L., Billingsley, F., & Schwartz, I. S. (2005). Effects of video modeling alone and with self-management on compliment-giving behaviors of children with high-functioning ASD. *Journal of Positive Behavior Interventions*, 7(1), 33–46.
- Harrower, J. K., & Dunlap, G. (2001). Including children with autism in general education classrooms: A review of effective strategies. *Behavior Modification*, 25(5), 762–784.
- Humphrey, L. L., Karoly, P., & Kirschenbaum, D. S. (1978). Self-management in the classroom: Self-imposed response cost versus self-reward. *Behavior Therapy*, 9(4), 592–601.
- Kanfer, F. H. (1970). Self-monitoring: Methodological limitations and clinical applications. *Journal of Consulting and Clinical Psychology*, 35(2), 148–152.
- Kazdin, A. E. (1974). Reactive self-monitoring: The effects of response desirability, goal setting, and feedback. *Journal of Consulting and Clinical Psychology*, 42(5), 704–716.
- Koegel, L. K., Koegel, R. L., Hurley, C., & Frea, W. D. (1992). Improving social skills and disruptive behavior in children with autism through self-management. *Journal of Applied Behavior Analysis*, 25(2), 341–353.
- Koegel, L. K., Harrower, J. K., & Koegel, R. L. (1999). Support for children with developmental disabilities in full inclusion classrooms through self-management. *Journal of Positive Behavior Interventions*, 1(1), 26–34.
- Koegel, R. L., Koegel, L. K., & McNeerney, E. K. (2001). Pivotal areas in intervention for autism. *Journal of Clinical Child Psychology*, 30(1), 19–32.
- Korotitsch, W. J., & Nelson-Gray, R. O. (1999). An overview of self-monitoring research in assessment and treatment. *Psychological Assessment*, 11(4), 415–425.
- Lee, S., Simpson, R. L., & Shogren, K. A. (2007). Effects and implications of self-management for students with autism: A meta-analysis. *Focus on Autism and Other Developmental Disabilities*, 22(1), 2–13.
- Lipinski, D. P., Black, J. L., Nelson, R. O., & Ciminero, A. R. (1975). Influence of motivational variables on the reactivity and reliability of self-recording. *Journal of Consulting and Clinical Psychology*, 43(5), 637–646.
- Mahoney, M. J. (1970). Toward an experimental analysis of covert control. *Behavior Therapy*, 1(4), 510–521.
- Mahoney, M. J. (1972). Research issues in self-management. *Behavior Therapy*, 3(1), 45–63.
- Mahoney, M. J. (1974). Self-reward and self-monitoring techniques for weight control. *Behavior Therapy*, 5(1), 48–57.
- Mahoney, M. J., Moore, B. S., Wade, T. C., & Moura, N. G. (1973). Effects of continuous and intermittent self-monitoring on academic behavior. *Journal of Consulting and Clinical Psychology*, 41(1), 65–69.
- Mischel, W. (1973). Toward a cognitive social learning reconceptualization of personality. *Psychological Review*, 80(4), 252–283.
- Moore, T. R. (2009). A brief report on the effects of a self-management treatment package on stereotypic behavior. *Research in Autism Spectrum Disorders*, 3(3), 695–701.
- Nelson, R. O., & Hayes, S. C. (1981). Theoretical explanations for reactivity in self-monitoring. *Behavior Modification*, 5(1), 3–14.
- Newman, B., & Ten Eyck, P. (2005). Self-management of initiations by students diagnosed with autism. *Analysis of Verbal Behavior*, 21, 117–122.
- Newman, B., Reinecke, D. R., & Meinberg, D. L. (2000). Self-management of varied responding in three students with autism. *Behavioral Interventions*, 15(2), 145–151.
- Palmen, A., Didden, R., & Arts, M. (2008). Improving question asking in high-functioning adolescents with autism spectrum disorders: Effectiveness of small-group training. *Autism*, 12(1), 83–98.

- Pierce, K. L., & Schreibman, L. (1994). Teaching daily living skills to children with autism in unsupervised settings through pictorial self-management. *Journal of Applied Behavior Analysis*, 27(3), 471–481.
- Stahmer, A. C., & Schreibman, L. (1992). Teaching children with autism appropriate play in unsupervised environments using a self-management treatment package. *Journal of Applied Behavior Analysis*, 25(2), 447–459.
- Wilkinson, L. A. (2005). Supporting the inclusion of a student with asperger syndrome: A case study using conjoint behavioural consultation and self-management. *Educational Psychology in Practice*, 21(4), 307–326.

Self-Other Distinction and Social Cognition in ASD

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Definition

Autism spectrum disorder (ASD) is characterized by marked impairments in social interactions. One of the most basic elements constituting the ability to interact with other social agents is to form representations of the self and the other while also being able to distinguish these representations. How crucial the ability for self-other distinction is for social interactions becomes obvious in the motor domain. Recent theories have emphasized that perceiving the actions of other agents activates corresponding motor representations in the observer, by means of the mirror neuron system (MNS; Gallese and Goldman 1998). When we see somebody else acting, this activates a motor representation similar to the one that is active when planning and executing the action oneself. Such a common representational basis of observed and executed actions makes it necessary, however, to distinguish these representations. Without self-other distinction, externally triggered motor representations would lead to overt imitative behavior. Interestingly, such an imitative response tendency can be observed in specific social situations (the so-called chameleon effect, Chartrand and Bargh

1999) and in specific pathologies (Lhermitte et al. 1986; Brass et al. 2003).

In the context of ASD, the so-called “broken mirror hypothesis” has been proposed (Williams et al. 2001). This hypothesis suggests that ASD is characterized by an impairment of the mirror system, which leads to a deficit of mirroring other people’s behavior. However, this theory has been strongly contested. Automatic imitation (not requiring higher level cognitive processes) has been found to be intact or even enhanced in ASD (Sowden et al. 2016; Spengler et al. 2010), while electroencephalography (EEG) and functional magnetic resonance (fMRI) studies on MNS functioning have yielded rather inconsistent findings, with less, typical, or more MNS activity in ASD (e.g., Martineau et al. 2010; Raymaekers et al. 2009). Current approaches of shared representation of perception and action rather postulate an abnormality in modulating MNS activity in the process of self-other distinction. Importantly, self-other distinction is also required in other social-cognitive processes. Our ability to take different spatial perspectives requires that we can distinguish our own perspective from the perspective of someone else (Samson et al. 2010). Furthermore, theory of mind (ToM), the ability to attribute mental states (beliefs, intentions, desires) to oneself and others, and to understand that others have mental states that may differ from one’s own also requires self-other distinction. In situations where other people hold beliefs that differ from our own beliefs, we need to distinguish between self- and other-related beliefs. Indeed, many researchers in the domain of social neuroscience have argued that self-other distinction is a low-level neurocognitive mechanism that lies at the basis of many domains of social functioning (Brass et al. 2009; de Guzman et al. 2016; Sowden and Shah 2014; Uddin 2011). At the neural level, research has indicated the crucial involvement of the temporoparietal junction (TPJ) in self-other distinction (e.g., Spengler et al. 2009). The TPJ is a brain region at the intersection of the inferior parietal and the superior temporal cortex. While the specific functional role of the TPJ and its functional neuroanatomy is still controversial (Krall et al. 2014), there is ample evidence that

the TPJ plays a crucial role in social cognition. Interestingly, the TPJ is also one of the core brain areas of the so-called ToM network and studies have consistently implicated TPJ dysfunction in ASD during ToM and other socio-cognitive tasks (e.g., Lombardo et al. 2011; Nijhof et al. 2018; Spengler et al. 2010). Together these findings led to recent theories of ASD pointing to a potential deficit in self-other distinction that might underlie social cognitive impairments in ASD, associated with TPJ dysfunction (e.g., de Guzman et al. 2016; Lombardo et al. 2011; Spengler et al. 2010).

Historical Background

Already in the earliest writings on autism the problem of distinguishing self from other was recognized. In his seminal article, Kanner wrote: “She used to talk using ‘you’ for herself and ‘I’ for her mother or me, as if she were saying things as we would in talking to her.” (Kanner 1943, p. 228). In this entry, Kanner described 11 children, who seemed to be unable to relate to other people, were self-absorbed, and who showed a general lack of interest in other people. Kanner noticed that none of the eight children that could speak used personal pronouns properly; they referred to other people by using first person pronouns (e.g., calling their mother “I”) and contrariwise used pronouns referring to others, to refer to themselves (when wanting a candy, saying “You want candy”). While this peculiar use of pronouns might index self-other distinction difficulties, Kanner did not refer to it as such but explained it in terms of echolalia. In his theoretical work, Hobson (1990) argued that self- and other-awareness is essential for the comprehension and hence proper use of pronouns, and hypothesized that ASD involves a specific impairment in understanding “I” in relation to “Thou,” which may lead to echolalia as well as other ASD-related behavior. More recently, researchers have started to systematically investigate potential deficits in neurocognitive mechanisms of self-other distinction in ASD (Lombardo et al. 2011; Spengler et al. 2010).

Current Knowledge

In the motor domain, self-other distinction has been investigated with the imitation-inhibition task (Brass et al. 2000). In this task, participants have to carry out a movement in response to an imperative stimulus (e.g., a number) while observing congruent or incongruent finger movements. In incongruent trials, participants have to suppress the automatic tendency to imitate the movement in order to carry out the instructed movement. Based on the idea, that movement observation leads to an automatic activation of a corresponding motor representation in the observer, it has been argued that suppressing the observed movement in incongruent trials requires self-other distinction and activates the anterior medial prefrontal cortex (aFMC) and the TPJ (Brass et al. 2005). Furthermore, it has been shown that brain activation in the imitation-inhibition task overlaps with brain activation in a ToM task and in an agency task, supporting the idea of a common underlying mechanism of self-other distinction that is linked to the TPJ (Spengler et al. 2009). More recent neurostimulation studies have shown a causal role of the TPJ in self-other distinction (Bardi et al. 2017; Santiesteban et al. 2012; Sowden and Catmur 2015).

It is commonly known that individuals with ASD have difficulties representing others and the idea of altered self-representation is also rather old (Frith and Happe 1999; Kanner 1943). While earlier theoretical work has claimed that ASD may involve impairment in understanding “I” in relation to “Thou” (Hobson 1990), only recently a specific deficit in neurocognitive mechanisms of self-other distinction has been proposed for ASD (Lombardo et al. 2011; Spengler et al. 2010). The idea that self-other distinction is compromised in the motor domain is supported by clinical observations. Young children with ASD often show pronoun reversal, echopraxia, and echolalia (involuntary imitation of motor and speech respectively) (Kanner 1943; Uddin 2011), and studies have shown decreased ability to inhibit imitative motor actions in ASD (Spengler et al. 2010) (see, however, Sowden et al. 2016 for

failures to show this relationship). In addition, there is neuroimaging evidence for this claim. In a sample of high-functioning adults with ASD, it was demonstrated that self-other distinction in the imitation-inhibition task was related to reduced brain activation in the TPJ and the anterior medial frontal cortex (aMFC) in a ToM task, supporting the idea that self-other distinction might be compromised in ASD (Spengler et al. 2010). Atypical empathic behavioral and physiological responses in ASD have also recently been linked to self-other distinction (De Coster et al. 2018). Furthermore, a recent electroencephalography (EEG) study demonstrated that self-other distinction in a task where participants had to match observed behavior and tactile stimuli was compromised in ASD (Deschrijver et al. 2017). Here, participants observed a hand touching a surface, combined with a tap-like tactile sensation at their own hand that either matched or mismatched the tactile consequence of the observed movement. Neurotypicals showed modulation of the P3 component for incongruent trials, which was absent in the ASD group. Crucially, this effect reliably correlated with self-reported social and sensory difficulties in ASD. Taken together, there is evidence from different domains that problems in self-other distinction might play an important role in ASD. Such problems might also result in compensatory strategies leading to the avoidance of social stimuli more generally. If people with ASD have problems to distinguish self from other when interacting with other social agents, they might prevent such problems by avoiding social interactions.

Future Directions

While there is augmenting evidence that self-other distinction is impaired in ASD, further research is needed to determine how such a deficit relates to the various social and nonsocial problems in ASD. As argued above, Theory of Mind requires self-other distinction, hence ToM problems can be partly explained by self-other distinction deficits. The observation that people with ASD avoid

social interactions could be explained as a compensatory strategy as outlined above. However, the link between compromised self-other distinction and nonsocial difficulties, such as restricted repetitive behavior and atypical sensory processing is less well understood, and warrants further investigation. Future research also has to clarify the link between self-other distinction problems and atypical processing of self-related stimuli. There is increasing evidence of altered self-referential processing in ASD, incorporating mental aspects of self, such as autobiographical memory difficulties and atypical neural responding to hearing one's own name (Nijhof et al. 2018), but also physical aspects like own face processing (Uddin 2011). As briefly outlined above, an integral part of defining oneself is by distinguishing oneself from others. Hence, if self-other distinction is impaired, self-referential processing might be compromised as well. Although coming from separate research lines, ToM deficits and altered processing of self-related stimuli in ASD may be tightly linked as a disturbed mechanism of self-other distinction may underlie both domains of impairment.

At a more general level, it needs to be addressed how problems with self-other distinction relate to recent attempts to explain ASD within a predictive coding framework. The predictive coding framework states that the brain constantly makes predictions about the world and processes incoming sensory information in light of those predictions (priors). ASD has been argued to be associated with inflexible precision in regulating sensory expectations based on these priors (Goris et al. 2018; Van de Cruys et al. 2014). It is not yet known how this relates to altered representations of self and other in ASD. The concept of self-agency which lies at the heart of self-other distinction is however strongly linked to predictive processes. Hence, self-other distinction problems might be a special case of a general predictive coding deficit. Finally, a better understanding of the role of self-other distinction and the TPJ in ASD may provide important putative targets for future interventions, such as neurostimulation of the TPJ and training of self-

other distinction, to improve social-cognitive functioning in individuals with ASD. Further research is needed, however, to address the question how other parts of the ToM network such as the aFMC are related to self-other distinction problems.

See Also

- [Social Language Development Test](#)
- [Theory of Mind](#)

References and Reading

- Bardi, L., Gheza, D., & Brass, M. (2017). TPJ-M1 interaction in the control of shared representations: New insights from tDCS and TMS combined. *NeuroImage*, 146, 734–740.
- Brass, M., Bekkering, H., Wohlschläger, A., & Prinz, W. (2000). Compatibility between observed and executed finger movements: Comparing symbolic, spatial, and imitative cues. *Brain and Cognition*, 44, 124–143.
- Brass, M., Derrfuss, J., Matthes-von Cramon, G., & von Cramon, D. Y. (2003). Imitative response tendencies in patients with frontal brain lesions. *Neuropsychology*, 17(2), 265–271.
- Brass, M., Derrfuss, J., & von Cramon, D. Y. (2005). The inhibition of imitative response tendencies: A functional double dissociation of imitative and overlearned responses. *Neuropsychologia*, 43, 89–98.
- Brass, M., Ruby, P., & Spengler, S. (2009). Inhibition of imitative behaviour and social cognition. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 364, 2359–2367.
- Chartrand, T. L., & Bargh, J. A. (1999). The chameleon effect: The perception-behavior link and social interaction. *Journal of Personality and Social Psychology*, 76(6), 893–910.
- De Coster, L., Wiersema, J. R., Deschrijver, E., & Brass, M. (2018). The effect of being imitated on empathy for pain in adults with high-functioning autism: Disturbed self-other distinction leads to altered empathic responding. *Autism*, 22(6), 712–727.
- De Guzman, M., Bird, G., Banissy, M. J., & Catmur, C. (2016). Self-other control processes in social cognition: From imitation to empathy. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 371, 20150079.
- Deschrijver, E., Wiersema, J. R., & Brass, M. (2017). Action-based touch observation in adults with high functioning autism: Can compromised self-other distinction abilities link social and sensory everyday problems? *Social Cognitive and Affective Neuroscience*, 12(2), 273–282.
- Frith, U., & Happe, F. (1999). Theory of mind and self-consciousness: What it is like to be autistic. *Mind and Language*, 14, 1–22.
- Gallese, V., & Goldman, A. (1998). Mirror neurons and the simulation theory of mind-reading. *Trends in Cognitive Sciences*, 12, 493–501.
- Goris, J., Braem, S., Nijhof, A. D., Rigoni, D., Deschrijver, E., Van de Cruys, S., Wiersema, J. R., & Brass, M. (2018). Sensory prediction errors are less modulated by global context in autism spectrum disorder. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 3(8), 667–674.
- Hobson, R. P. (1990). On the origins of self and the case of autism. *Development and Psychopathology*, 2, 163–181.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Krall, S. C., Rottschy, C., Oberwelland, E., et al. (2014). The role of the right temporoparietal junction in attention and social interaction as revealed by ALE meta-analysis. *Brain Structure and Function*, 220, 587–604.
- Lhermitte, F., Pillon, B., & Serdaru, M. (1986). Human autonomy and the frontal lobes. Part I: Imitation and utilization behavior: A neuropsychological study of 75 patients. *Annals of Neurology*, 19(4), 326–334.
- Lombardo, M. V., Chakrabarti, B., Bullmore, E. T., & Baron-Cohen, S. (2011). Specialization of right temporo-parietal junction for mentalizing and its relation to social impairments in autism. *NeuroImage*, 56(3), 1832–1838.
- Martineau, J., Andersson, F., Barthélémy, C., Cottier, J. P., & Destrieux, C. (2010). Atypical activation of the mirror neuron system during perception of hand motion in autism. *Brain Research*, 1320, 168–175.
- Nijhof, A. D., Bardi, L., Brass, M., & Wiersema, J. R. (2018). Brain activity for spontaneous and explicit mentalizing in adults with autism spectrum disorder: An fMRI study. *NeuroImage*, 18, 475–484.
- Raymaekers, R., Wiersema, J. R., & Roeyers, H. (2009). EEG study of the mirror neuron system in children with high functioning autism. *Brain Research*, 1304, 113–121.
- Samson, D., Apperly, I. A., Braithwaite, J. J., Andrews, B. J., & Bodley Scott, S. E. (2010). Seeing it their way: Evidence for rapid and involuntary computation of what other people see. *Journal of Experimental Psychology: Human Perception and Performance*, 36, 1255–1266.
- Santiesteban, I., Banissy, M. J., Catmur, C., & Bird, G. (2012). Enhancing social ability by stimulating right temporoparietal junction. *Current Biology*, 22, 2274–2277.
- Sowden, S., & Catmur, C. (2015). The role of the right temporoparietal junction in the control of imitation. *Cerebral Cortex*, 25(4), 1107–1113.

- Sowden, S., & Shah, P. (2014). Self-other control: A candidate mechanism for social cognitive function. *Frontiers in Human Neuroscience*, 8, 1–5.
- Sowden, S., Koehne, S., Catmur, C., Dziobek, I., & Bird, G. (2016). Intact automatic imitation and typical spatial compatibility in autism spectrum disorder: Challenging the broken mirror theory. *Autism Research*, 9, 292–300.
- Spengler, S., von Cramon, D. Y., & Brass, M. (2009). Control of shared representations relies on key processes involved in mental state attribution. *Human Brain Mapping*, 30(11), 3704–3718.
- Spengler, S., Bird, G., & Brass, M. (2010). Hyperimitation of actions is related to reduced understanding of others' minds in autism spectrum conditions. *Biological Psychiatry*, 68(12), 1148–1155.
- Uddin, L. Q. (2011). The self in autism: An emerging view from neuroimaging. *Neurocase*, 17(3), 201–208.
- Van de Cruys, S., Evers, K., Van der Hallen, R., Van Eylen, L., Boets, B., De-Wit, L., & Wagemans, J. (2014). Precise minds in uncertain worlds: Predictive coding in autism. *Psychological Review*, 121(4), 649–675.
- Williams, J. H., Whiten, A., Suddendorf, T., & Perrett, D. I. (2001). Imitation, mirror neurons and autism. *Neuroscience and Biobehaviour Review*, 25, 287–295.

Self-Recognition

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Definition

Self-recognition refers to recognizing the “self” as separate from others. The term self encompasses various modalities (e.g., physical, emotional, psychological) which are unique and representative of an individual. Generally, attributes of self are categorized as either physical or psychological. Physical aspects of the self (e.g., face) typically refer to material embodiments of the self and awareness of personal agency, whereas psychological characteristics include multiple cognitive constructs such as personality traits and subjective perspectives (Gillihan and Farah 2005). The ability to self-recognize can be used as an index of self-awareness as well as an early developmental marker. A face is among the

earliest and the most common embodied representations of one's self. Self-face recognition occurs between 18 and 24 months and is considered crucial for the development of a self-concept and successful social behavior as it signals the emergence of sophisticated levels of self-awareness. The differentiation of self and others progresses continuously over the first 24 months, culminating in a clear identification of self-image and self-awareness (Lewis 1991). Impaired or inhibited emergence of self-recognition is linked with developmentally delayed disorders such as Autism Spectrum Disorder (ASD).

Historical Background

Self is foundational to the term Autism. The word Autism is derived from the Greek word “autos” meaning “self.” Leo Kanner first applied this term to children demonstrating complete self-focus and who were seemingly indifferent to social interaction (1943). Researchers have since considered individuals with Autism to have an “absent self” or reduced self-awareness in order to explain the observed impairments (Blakemore and Frith 2003).

Early research on visual self-recognition suggests children with ASD do not have impaired bodily self-recognition and are capable of acknowledging their own reflection (Dawson and McKissick 1984). Self-recognition is classically examined through a mirror paradigm originally developed by Gallup (1970) to investigate self-concept in nonhuman primates. Variations of this paradigm have been used to empirically study self-recognition in infants and children. The protocol involves secretly marking an infant's nose and observing their response upon placing the infant in front of a mirror. A response directed at the mark infers self-recognition. At approximately 2 years of age, young children are able to recognize themselves and exhibit a definite recognition of their reflection by touching their noses. Children with ASD have shown variable ability in this task compared to typically developing children (TD) (Dawson et al. 1984).

However, impaired ability in this task is not considered to be a deficit of the disorder since it has not been found to be related to ASD characteristics. Dawson and McKissick (1984) demonstrated that language, motor, and social deficits were not related to self-face recognition. Moreover, some studies that report significant self-recognition impairments in children with ASD also found that their sample exhibited mental ages below their chronological age. Therefore, variable or absence of self-recognition in the mirror task can be thought to be a reflection of a developmental delay and not a specific inability characteristic of ASD (Dawson and McKissick 1984; Ferrari and Matthews 1983). This suggests self-focused observations in ASD may be related to other “self” domains such as more abstract or cognitive representations of self.

A reduced sense of self in children with ASD is evident in cognitive aspects of self such as self-awareness and self-referencing. For example, while observations of embarrassment and coyness during the mirror task usually appear around 2 years of age in TD children (Amsterdam and Greenberg 1977), similar self-conscious reactions were reported missing in children in ASD who performed the same task. These findings are congruent with research reporting a reduction in the use of self-referencing pronouns in ASD children. Concurrent with the ability to recognize one’s self in a mirror is the onset of personal pronoun use (e.g., “I” and “me”; Preyer 1889). The use of personal pronouns in children with ASD has been shown to be reduced and is thought to be indicative of a failure to self-reference (Fay 1979). A lack of self-awareness reactions and use of personal pronouns points to an underdeveloped or impaired cognitive sense of self in ASD. Such impairments early in development are thought to be related to an increased difficulty distinguishing between self and other and subsequently increased social impairments (Kanner 1943). More recent research has focused on how the ability to distinguish between self and others differs in children with Autism.

Current Knowledge

The multidimensional nature of self-recognition lends itself to be examined from both a behavioral and neurological perspective. While some aspects of self-awareness are atypical in ASD, others appear to be intact. Abstract representations of self, such as identifying personal feelings and conceptual ideals, are more affected than awareness of one’s physical self. The formation of a secure and balanced self-representation is important to social understanding (Decety and Sommerville 2003). Researchers have investigated self-recognition in ASD at various stages of development at both the behavioral and neural level.

Behavioral

Studies implementing various self-referencing paradigms have reported aberrant self-recognition ability in children with ASD from a young age. A reduction in the use of self-reference has been well characterized. Studies examining verbal expressions of self have found that individuals with ASD have specific difficulties identifying personal experiences, mental states, and emotions (see Capps et al. 1995; Frith and Happé 1999). Retrospective studies on individuals with ASD and their siblings demonstrated a reduction in self-reference, such as name orientation, based on the examination of first birthday home videos. Longitudinal behavioral studies following infant siblings at high risk for ASD (i.e., siblings of children with ASD) have found orienting to one’s name in the videos was a behavioral marker that differentiated between those children who were later diagnosed with ASD and those who did not. The authors suggest these early observations are a sign of reduced social motivation and are unrelated to developmental delays alone (Zwaigenbaum et al. 2005). This attenuated tendency to self-reference is seen throughout development and into adulthood. In a study looking at self-reference and memory performance, adults with ASD did not perform as well during the memory task condition that involved words

presented in a self-related manner (i.e., “Does the word describe you?”) compared to the control group (Toichi et al. 2002). Additional longitudinal studies are needed to track participants throughout development.

Expanding upon the mirror task, a revised digital version of the paradigm uses previously recorded video to examine self-recognition. This delayed self-recognition (DSR) task tests whether children recognize their past self as opposed to their present self in a modified version of Gallup’s mirror paradigm. A sticker is placed on a child’s head in secret and is later shown a video of the sticker being placed on their head. The majority of children with ASD showed no difficulty realizing that a sticker was actually on their head nor did they significantly differ in prompts to acknowledge the sticker. The DSR task demonstrates children with ASD have no impairment recognizing between their current and past self (Lind and Bowler 2009). This finding is also congruent with research on self-recognition of agency in ASD. Agency refers to the person causing or generating an action. In an action-monitoring task, Williams and colleagues (2008) showed that children with ASD performed similarly to typically developing individuals by showing stronger self-reference when following and recalling their own actions compared to the actions of the experimenter. Both the DSR and agency monitoring studies suggest bodily self-recognition is unimpaired in ASD.

While physical attributes of self-recognition are intact in ASD, it is unclear if the neural bases of these processes are being employed the same way as in TD individuals. For example, individuals with ASD may employ alternative cognitive mechanisms, such as matching representations of like images to achieve the same results. Another caveat in many self-recognition studies is testing age. Due to the relatively older age at diagnosis, it is difficult to compare early developmental constructs in children with ASD that occur in the first few years of life. As of yet, no known self-recognition research has been reported on infants at high risk for Autism. Numerous neuroimaging studies, however, have examined the neural mechanisms in children and adults.

Neuroimaging

While specific brain region involvement may vary depending on different self-referential processes, the right prefrontal cortex plays a large role in self-recognition (Lou et al. 2004; Keenan et al. 2000). Self-face recognition research implicated the right hemisphere with various psychological aspects of the self (i.e., trait attributes, judgment of self, self-reflection) (Platek et al. 2006; Uddin et al. 2005). While it appears that self-referencing, particularly self-face recognition, is lateralized in the right hemisphere (RH), the left hemisphere (LH) is likely to play an important role. Several functional Magnetic Resonance Imaging (fMRI) studies have shown bilateral activation during different operationalizations of self (Kircher et al. 2000). Research on split-brain patients (where the corpus callosum is severed and communication between the two hemispheres has been lost) also report discrepant findings in regards to hemispheric differences when examining self-processing (Keenan et al. 2003). The relatively few self-face studies in ASD provide some evidence to suggest that self-face recognition tasks elicit activity in similar cortical regions as neurotypical individuals. However, the patterns of activity within these regions differ.

Neuroimaging research on self-recognition in ASD has largely focused on neural processes distinguishing between self- and other-faces. Self-face recognition paradigms typically included morphed versions of a participant’s face with that of a familiar or unfamiliar other’s face to varying degrees. Participants are asked to identify at what point the stimulus looks more like themselves than the other person’s face (Uddin et al. 2006). Several imaging techniques using a similar paradigm have supported RH specialization for self-face recognition in control participants (Keenan et al. 2000) and altered RH patterns of activity in individuals with ASD (Gunji et al. 2009). In particular, the right inferior frontal gyrus (IFG) appears to be involved in self and other face distinction and to show reduced activity in individuals with ASD. The right IFG is part of the Mirror Neuron System (MNS) and is thought to contribute to successful understanding of

other's intentions as well as self-recognition (Uddin et al. 2005). More specifically, in an fMRI study by Uddin et al. (2008) children with ASD exhibited different activation patterns in the right IFG, compared to the TD group. TD children exhibited almost equal activation to self and other face processing, while ASD only exhibited significant activation when viewing their own faces (Uddin et al. 2008). The authors suggest that this dissociation may point to a neural substrate for self-focused and decreased other focused social motivation. They also propose that the shared representation for self and others seen in TD children may be a neural mechanism by which we understand others and reduction of activity in the IFG during other-face processing may indicate reduced social understating in children with ASD. Kita et al. (2010) likewise reported right IFG activation in a self-recognition near-infrared spectroscopy study. Control and ASD participants exhibited higher levels of oxygenated hemoglobin in the right compared to left IFG when viewing self-face stimuli. They also reported that an increase in IFG activation was inversely related to ASD symptom severity, such that lower activity was related increased ASD symptom severity (Kita et al. 2010).

Neuroimaging studies have also found individuals with ASD have difficulties during cognitive self-referential tasks such as mentalizing. High-level inference-based mentalizing involves cognitive representations of self and other and recruits similar neural systems in regards to physical self and other representation. In an fMRI study by Lombardo (2010) TD and ASD participants made reflective judgments about themselves and a nonclose other (i.e., president). Neurotypical adults more strongly recruited the ventral medial prefrontal cortex (vMPFC) when mentalizing about themselves. In contrast, the ASD group activated the vMPFC equally when mentalizing about self and other. Further, the magnitude of self and other distinction in the vMPFC was negatively correlated with social impairments in the ASD participants. This study suggests that specific dysfunctions in cortical midline structures of the brain, such as MPFC, may alter patterns of

activity when mentalizing (Lombardo et al. 2007). Uddin et al. (2008) found the reverse dissociation during a face recognition task, suggesting that differences between neurotypical and ASD differ depending on the nature of the self-referential process.

Other studies have also reported a dissociation between self and other perception in individuals with pervasive developmental disorder (PDD) (i.e., ASD). In an electrophysiological study (Gunji et al. 2009), atypical patterns of self- and other-recognition were demonstrated in children with PDD compared with TD children and adults. The TD groups made a distinction between self and familiar stimuli, but the children with PDD did not (Gunji et al. 2009).

The dual nature of self, simultaneously being similar and different from other individuals is central to the development of social cognition (Lombardo and Baron Cohen 2010). Neural activation related to distinguishing between self and other appear to be atypical in ASD, suggesting that altered self- and other-recognition may be linked to deficits in social cognition in the disorder.

Future Directions

Self-recognition research in Autism has primarily focused on specific self-representation processes. Neuroimaging in particular have used task-related MRI to examine self-recognition mechanisms and networks. Future research should explore the basic mechanisms and the levels at which different domains of self (i.e., physical and psychological) relate to one another. The relationship between patterns and levels of activation that involve the MNS and higher mentalizing systems should also be explored in the same sample. Presently, there are few longitudinal studies, more are needed to examine developmental changes in self-referencing and recognition. It would also be interesting to investigate different cultural perspectives of self and how interdependent or independent views of self modulate self-recognition mechanisms.

References and Reading

- Amsterdam, B., & Greenberg, L. M. (1977). Self conscious behavior of infants: A videotape study. *Developmental Psychobiology*, 10(1), 1–6.
- Blakemore, S. J., & Frith, C. (2003). Self-awareness and action. *Current Opinion in Neurobiology*, 13(2), 219–224.
- Capps, L., Sigman, M., & Yirmiya, N. (1995). Self-competence and emotional understanding in high-functioning children with autism. *Development and Psychopathology*, 7(01), 137–149.
- Dawson, G., & McKissick, F. C. (1984). Self-recognition in autistic children. *Journal of Autism and Developmental Disorders*, 14(4), 383–394.
- Decety, J., & Sommerville, J. A. (2003). Shared representations between self and other: A social cognitive neuroscience view. *Trends in Cognitive Sciences*, 7(12), 527–533.
- Fay, W. H. (1979). Personal pronouns and the autistic child. *Journal of Autism and Developmental Disorders*, 9(3), 247–260.
- Ferrari, M., & Matthews, W. S. (1983). Self-recognition deficits in autism: Syndrome-specific or general developmental delay? *Journal of Autism and Developmental Disorders*, 13(3), 317–324.
- Frith, U., & Happé, F. (1999). Theory of mind and self-consciousness: What is it like to be autistic? *Mind and Language*, 14(1), 82–89.
- Gallup, G. G. (1970). Chimpanzees: Self-recognition. *Science*, 167(3914), 86.
- Gillihan, S. J., & Farah, M. J. (2005). Is self special? A critical review of evidence from experimental psychology and cognitive neuroscience. *Psychological Bulletin*, 131(1), 76.
- Gunji, A., Inagaki, M., Inoue, Y., Takeshima, Y., & Kaga, M. (2009). Event-related potentials of self-face recognition in children with pervasive developmental disorders. *Brain & Development*, 31(2), 139–147.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2(2), 217–230.
- Kaplan, J., Aziz-Zadeh, L., Uddin, L., & Iacoboni, M. (2008). The self across the senses: An fMRI study of self-face and self-voice recognition. *Social Cognitive and Affective Neuroscience*, 3(3), 218–223. <https://doi.org/10.1093/scan/nsn014>.
- Keenan, J. P., Freund, S., Hamilton, R. H., Ganis, G., & Pascual-Leone, A. (2000). Hand response differences in a self-face identification task. *Neuropsychologia*, 38, 1047–1053.
- Keenan, J. P., Wheeler, M., Platek, S. M., Lardi, G., & Lassonde, M. (2003). Self face processing in a callosotomy patient. *European Journal of Neuroscience*, 18(8), 2391–2395.
- Kircher, T. T. J., Senior, C., Phillips, M. L., Benson, P. J., Bullmore, E. T., Brammer, M., Simmons, A., Williams, S. C. R., Bartels, M., & David, A. S. (2000). Towards a functional neuroanatomy of self processing: Effects of faces and words. *Cognitive Brain Research*, 10(1–2), 133–144.
- Kita, Y., Gunji, A., Inoue, Y., Goto, T., Sakihara, K., Kaga, M., Inagaki, M., et al. (2010). Self-face recognition in children with autism spectrum disorders: A near-infrared spectroscopy study. *Brain & Development*, 33, 494–503.
- Lewis, M. (1991). Ways of knowing: Objective self-awareness or consciousness. *Developmental Review*, 11(3), 231–243.
- Lind, S. E., & Bowler, D. M. (2009). Delayed self-recognition in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 39(4), 643–650.
- Lombardo, M. V., & Baron Cohen, S. (2010). Unraveling the paradox of the autistic self. *Wiley Interdisciplinary Reviews: Cognitive Science*, 1(3), 393–403.
- Lombardo, M. V., Barnes, J. L., Wheelwright, S. J., & Baron-Cohen, S. (2007). Self-referential cognition and empathy in autism. *PLoS One*, 2(9), e883. <https://doi.org/10.1371/journal.pone.0000883>.
- Lou, H. C., Luber, B., Crupain, M., Keenan, J. P., Nowak, M., Kjaer, T. W., Sackeim, H. A., et al. (2004). Parietal cortex and representation of the mental self. *Proceedings of the National Academy of Sciences of the United States of America*, 101(17), 6827.
- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders*, 24(3), 247–257.
- Preyer, W. T. (1889). *The mind of the child: The development of the intellect* (Vol. 2). New York: Appleton.
- Toichi, M., Kamio, Y., Okada, T., Sakihama, M., Youngstrom, E. A., Findling, R. L., & Yamamoto, K. (2002). A lack of self-consciousness in autism. *The American Journal of Psychiatry*, 159(8), 1422.
- Uddin, L. Q., Kaplan, J. T., Molnar-Szakacs, L., Zaidel, E., & Iacoboni, M. (2005). Self-face recognition activates a frontoparietal. *NeuroImage*, 25(3), 926–935.
- Uddin, L. Q., Molnar-Szakacs, I., Zaidel, E., & Iacoboni, M. (2006). rTMS to the right inferior parietal lobule disrupts self–other discrimination. *Social Cognitive and Affective Neuroscience*, 1(1), 65.
- Uddin, L. Q., Davies, M. S., Scott, A. A., Zaidel, E., Bookheimer, S. Y., Iacoboni, M., & Dapretto, M. (2008). Neural basis of self and other representation in autism: An FMRI study of self-face recognition. *PLoS One*, 3(10), 1–9.
- Williams, D., Happe, F., & Jarrold, C. (2008). Intact inner speech use in autism spectrum disorder: Evidence from a short term memory task. *Journal of Child Psychology and Psychiatry*, 49(1), 51–58.
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, 23(2–3), 143–152.

Self-Recognition and Self-Referential Behavior

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In order to discuss self-recognition in children with autism spectrum disorder (ASD), we will first need to embed the interest in self-recognition into a more general theory of the development of the self-system, in particular that aspect of the system referred to as the self-reflecting aspect of the self. Having done so, the measurement of this aspect of the system will be presented along with data on its development and the relation to brain function. Following this, the research on the development of self-referential behaviors in children with ASD and its relationship to brain function will be discussed (portions of this manuscript have appeared in *The Rise of Consciousness and the Development of Emotional Life* (©2014 Michael Lewis, Published by The Guilford Press)).

The Self-System

The terms *self* and *nonself* in reference to plants and to cells, as well as to humans, are used. If the term self is confusing, consider several examples from the human literature. The term self-regulation is used when talking about newborn infants (Kopp 1982) and intersubjectivity in 6-month-olds (Rochat 2009; Stern 1985). Things do not get much better when we consider adult

humans, for example, the Western view of self as “I self” versus an Eastern view of self as “we self” (see Geertz 1984). Even dissociative identity disorder, the idea of multiple selves rather than a single self, has been identified (Ross 1989).

Even in everyday lives, people are confronted with explaining selves. Much of their motor action, although initially planned, is carried out by core processes of the body that include, by definition, self-regulation and self-other differentiation. The same, of course, is true of thinking. One level of self is necessary to formulate, at least sometimes, what it is that is wished to be thinking about, but another level of self does not appear to be involved in the processes that actually conduct the task of thinking. Consider this example: a person is given the problem of adding a 7 to the sum of 7s that precede it (e.g., $7 + 7 = 14 + 7 = 21 + 7 = 28$, etc.). It is clear that as subjects carry out this task, they cannot reflect on themselves doing the arithmetic. One aspect of the self has set up the problem, another solves it. These diverse examples from plants, cells, human newborns, and adults all address the topic of what it is that people mean when they use the term self.

It is important to remember that:

- All living systems self-regulate; in any living system, there needs to be communication between parts of that system. This can include a unit as small as a cell, a plant, or an animal or even a more complex organism. Self-regulation is a property of living matter. Self-regulation makes no assumptions about a mental state or self-awareness (McClelland et al. 2014).
- Some minimal differentiation between self and other is a necessary condition for action. How this differentiation is produced is unknown (see Butterworth 1992). What appears to be so is that all organisms, even such things as T cells, cannot act without at some level being able to distinguish between self and other. The ability to self-regulate or to distinguish self from other is part of the core processes of all living systems (von Bertalanffy 1967). It is not an extended consciousness as some believe (Damasio 1999).

- Even higher order functions such as perception, thinking, and complex actions, such as driving a car, can be performed by adult humans without a mental state or self-reflection; that is, without their being able to reflect on, look at, and observe the processes that allow these behaviors to be carried out.
- A unique aspect of some self-systems is self-reflection. By self-reflection we mean the capacity of a self to know it knows or to remember it remembers. It is this “meta” ability that is referred to as self-reflection. This reflective capacity of self-awareness may be uniquely human, although we may need to include the great apes who may be also capable of this.

Given the existence of the various levels of self, the question posed is: When I say “I know X,” is it the case that I *must* know that I know X? If it is the case that I can know that I know X, when is it the case that I do know that I know X? These kinds of epistemological questions require different levels of self. These epistemological questions are best addressed by the analysis of different levels of the self-system since from an epistemological point of view, knowledge can be bodily or reflective knowledge, but the knowledge of the knowledge is always a reflective self. Knowledge of the knowledge is the capacity of the self to reflect on itself. This ability to reflect on itself is what makes it so important to understand in the study of autism.

The Measurement of Self-Reflection

Measurement follows from the constructs that are made. If there is interest in the development of self, then the measurement of self-referential behavior is necessary. Because early imitation, intersensory integration, and coordination between infant and mother all are likely to reflect the core processes of self, they are not adequate measures of self-reflection.

The study of self-reflection in adults requires symbolic language capacity. In an adult, or typically developing child, it can be asked, “Who are

you?” “Tell me something about yourself,” or “Tell me something that you know that others don’t know.” Alternatively, following R. D. Laing (1970), we can see whether the child understands statements such as “I know, you know that I know where you put your teddy bear.” As is readily understood, all of these questions imply some idea the child has about itself, or its self-reflection, because the recursive knowledge about what others know about what you know explicitly implies a self referent.

Without language, however, the child with ASD cannot through language explain this idea to us. One alternative is to require, without using language, that children do certain tasks and see whether they can do them. If the child understands the task given, it is possible to demonstrate that the child has the idea, even though she/he does not have language. Thus, for example, in the work on deception (Allen and Lewis in preparation; Lewis et al. 1989) and in the research on theories of mind, Wellman (1990) and Moses and Chandler (1992), for example, have been able to show that the child can deceive and also place itself in the role of another. In each of these types of studies, there is an implicit theory of mind that includes self-reflection (see Carpendale and Lewis 2010; Chandler and Birch 2010).

Unfortunately, even these studies require that children understand complex language although they do not have to produce it or only need some early emerging capacities. A language measure, a bit less suspect, is that of personal pronoun usage. Because parents rarely use the label “me” or “mine” when referring to the child or to themselves (they say, “I am {or Mommy is} going to the store.”), the use of these terms by the child is likely to be a reasonable measure of self-reflection. This appears even more the case when children’s use of the terms and how they behave when using them are observed. In a typically developing child, it can be observed that as the child says “mine,” it also pulls the object away from another child and toward itself. Because moving the object toward oneself does not move the object as far away from the other as possible, the placement of the object next to the body, together with the use of the term “me” or

“mine,” appears to reference self-reflection. Typically developing children begin to use personal pronouns including “me” and “mine” by the latter part of the second year of life, which can provide a linguistic demonstration of the emerging mental state (Harter 1983; Hobson 1990).

Another procedure that can be used to measure self-reflection is self-recognition. Lewis has studied self-recognition in infants and young children in detail (Lewis 1992, 2003a; Lewis and Brooks-Gunn 1979a; Lewis and Ramsay 2004). The procedure is simple. Unknown to the child, their nose is marked with rouge and then the child is placed in front of a mirror, where it is possible to observe whether the child, looking in the mirror, touches its marked nose or whether it touches the image in the mirror. The data from a variety of studies indicate that typically developing infants even as young as 2 months, when placed in front of mirrors, show interest and respond to the mirror image. Children will smile, coo, and try to attract the attention of the child in the mirror, although they do not behave as if they recognize that it is they in the mirror; this is an evolutionarily derived adapted action pattern produced in the context of *babyiness*. Indeed, even dogs, cats, and older children and adults are attracted by babyishness. At older ages, when locomotion appears, on occasion, infants have been observed going behind the mirror to see whether they can find the child in the mirror. In addition, they often strike the mirror as if they are trying to touch the other. Somewhere around 15–18 months of age, they appear to begin to know that the images are themselves because they touch their noses or comment about their noses when looking in the mirror. Self-reflection is captured by the children’s use of self-directed referential behaviors. The touching of their noses when they look in the mirror seems to reveal that they know that it is “me” there.

The ability to use the mirror to reference herself has been mistaken for the child’s understanding of the property of mirrors. There is ample evidence that although children are able to produce self-referential behavior through the use of the mirror-mark technique, they do not know many of the properties of reflected surfaces; for example, they cannot use the mirror to find an

object only seen in its surface (Butterworth 1992). What is important about the self-referential behaviors in the mirror is that they need not be a marker of general knowledge about reflected surfaces but rather a marker for the child’s knowledge about itself. They are the equivalent of the phrase “that’s me.” This recognition, if put into words, says, “That is me over there; this is me here.”

Measuring other aspects of self-reflection is possible, pretend play in particular. From a variety of theoretical perspectives (Huttenlocher and Higgins 1978; Leslie 1987; McCune-Nicolich 1981; Piaget 1962/1951), it is apparent that pretense is an early manifestation of the ability to understand mental states including one’s own and others’. Pretense involves double knowledge or dual representation of the literal and pretend situation. The dissociable relation between the two allows the child to distinguish between appearance and reality. Research by Piaget (1962/1951) and subsequent investigators (Fein 1975; Lowe 1975; McCune 1995; Nicolich 1977) indicates that pretense emerges in children by the middle to latter part of the second year of life. The capacity for pretense not only marks self-reflection but also the beginning of a theory of mind that is based on mentalism, not that which is based on evolutionarily derived adapted action patterns such as gaze behavior, joint attention, social referencing, and preverbal communication abilities (Bretherton 1991; Carpenter et al. 1999; Leslie 1987).

There are studies that have examined the relation between verbal measures of self-recognition (including the use of the personal pronoun “me” and one’s name) and the mark-directed behavior (Bertenthal and Fischer 1978; Lewis and Ramsay 2004; Pipp et al. 1987). These studies have generally found that verbal measures appear after the mark-directed behavior (Harter 1983). It also is apparent that self-referential and pretense behaviors emerge at approximately the same point in development (see Lewis and Ramsay 2004). A mental state of self-reflection is taken for granted in Leslie’s (1987) model on the relation between pretend play and theory of mind. That pretend play emerges as soon as the onset of self-

recognition would support the belief that both reflect the emergence of self-referential behavior, as well as a source for a theory of mind.

In a series of studies on self-referential behavior, Lewis and his colleagues have established that self-recognition starts to emerge by 15 months and that, for the most part, all typically developing children demonstrate it by 24 months, thus showing an onset as well as offset (Lewis and Brooks-Gunn 1979b). Moreover, children with Down syndrome need a mental age of 15–18 months in order to show mirror recognition (Cicchetti and Sroufe 1978). Children who show self-recognition show self-conscious emotions such as embarrassment, while those who do not show recognition do not show embarrassment (Lewis et al. 1989).

Self-recognition, personal pronoun use, and pretend play all indicate self-referential behavior. It is apparent that, with development, self-representation increasingly becomes a more complex and multifaceted phenomenon that progressively includes other cognitive and evaluative aspects of self-knowledge (Lewis 2014). These results suggest that in terms of emergent time, self-recognition is earliest in the formation of a complex self-reflection. Of the three self-referential abilities assessed, self-recognition in mirrors was the one most likely to emerge first in development, suggesting that physical self-referential action may provide the core aspect of self-representation that continues to develop beyond the second year of life.

Consistent with the present findings is work that indicates children's emerging understanding of a theory of mind by the middle of the second year of life. For example, Meltzoff (1995) reports that 18-month-old toddlers have the ability to understand the intentions of others. After observing adult models demonstrate the intention to act in a certain way by starting, but not completing, a given activity, the toddlers, when given the opportunity, performed the complete acts the adult intended. Similarly, Asendorpf and colleagues (Asendorpf and Baudonniere 1993; Asendorpf et al. 1996) found increases in imitative play linked to the presence of self-recognition in 20-month-old infants. Indeed, many studies link

self-recognition to other abilities that mark more broadly the emergence of self-representation. For example, self-recognition is related to children's self-conscious emotions, empathy (Bischof-Kohler 1994), as well as altruism (Zahn-Waxler et al. 1992). Self-recognition is related to autobiographical memories (Harley and Reese 1999) and is associated with imitation (Asendorpf 2002). The degree of correspondence between self-recognition, pretend play, language self-referents, and the emergence of ownership suggests, in the typically developing child, the emergence of self-reflection in the middle of the second year of life (Friedman 2011). The absence or delay of self-recognition, pretend play with others, and personal pronoun usage during this time frame has been associated with mental age delays and with ASD, and this will be considered in more detail, but first we will discuss the role of brain maturation in self-referential behavior.

Self-Reflection and the Development of Brain Function

While social coaction between the child and its social world may affect self-reflection abilities, it is likely it does so through its effect on the development of brain functioning. Lewis and colleagues have used self-recognition in mirrors, personal pronoun usage, and pretend play as measures of self-referential behavior and have used these measures to examine the relation between brain function development and self-reflection.

Given that there appears to be a developmental onset, for typically developing children, of these self-referential behaviors usually absent before 15 months, given that a mental age of 15–18 months is necessary for its display, and given that it is absent or delayed in children with autism, it is likely that developing biological processes play an important role in the development of them. Finally, given that monkeys cannot, but the great apes and humans can, suggests a phylogenetic as well as an ontogenetic pattern. These findings provide strong support for the idea that brain development may be associated with the onset of self-reflection. But to be clear, brain

development itself is not a split-off feature of an encapsulated brain; the brain develops according to processes of probabilistic epigenesis, through complex relational bidirectional coactions with other bodily processes and environmental contexts (Overton 2006).

This idea of the significance of brain development for self-reflection is further supported by the findings that specific brain region activation is associated with adult self-referential behaviors. The left superior temporal gyrus and the left medial frontal gyrus are activated when subjects engage in a theory-of-mind task relative to reading sentences (Fletcher et al. 1995; Gallagher et al. 2000; Mitchell et al. 2002). Activation of the left superior temporal cortex (Brodmann area 22), the left inferior parietal cortex, and the left and right occipital cortices (BA 18) occurs when subjects judge whether adjectives are relevant to themselves (Fossati et al. 2003; Macrae et al. 2004). In a study of brain activation to hearing one's own name, Carmody and Lewis (2006) found activation in the middle and superior temporal cortex and the left middle frontal cortex. In general, there is agreement that self-referential behaviors activate regions near the temporoparietal junction, although there are data suggesting activation of the medial frontal cortex as well (Fletcher et al. 1995; Kampe et al. 2003).

To measure brain activation developmentally as a function of the capacity to show self-reflection, one would need to study the relation between the emergence of this representational ability and changes in brain function. Obtaining functional magnetic resonance imaging (fMRI) or positron emission tomography (PET) scans in very young children has been shown to be difficult (Souweidane et al. 1999), and there are few, if any, published fMRI studies of children between 15 and 30 months of age, which is the critical age range for the development of self-referential behaviors (Paus 2005; Saxe et al. 2004).

One way to study brain development is to use magnetic resonance imaging (MRI) to measure the relative amounts of gray and white matter in different cortical brain regions (Toga et al. 2006). MR images show a gray-white matter contrast in

a sequence that reflects the time course of brain development (Barkovich 2000, 2005; Paus et al. 2001), and changes in MRI features are informative in determining the developmental changes in white and gray matter for normal and clinical cases (Barkovich 2002). Different MRI techniques are available to assess brain development and involve either qualitative judgments or quantitative measures. Although the qualitative descriptions help characterize brain development, computational analysis of MR images allows the detection of individual changes in white matter that signal the biological underpinnings of development of motor, sensory, cognitive, and perhaps social changes. To that end, quantitative measures may prove more valuable than the qualitative judgments of change.

Quantitative measures include volumetric analyses of gray and white matter (Thompson et al. 2007), development of white matter relative to gray matter (Carmody et al. 2004), and the more recent diffusion tensor imaging (DTI) techniques to assess white matter integrity (Anjari et al. 2007). DTI provides data on the anatomy and the density of white matter fibers and development (Dubois et al. 2006), as well as providing images of the cortical association tracts (Mori et al. 2002).

Lewis and Carmody were interested in using the standard clinical MRI scans to assess brain development in both clinical and nonclinical groups. Quantitative scores based on the difference between white matter and gray matter were obtained using quantitative assessments of T2-weighted MR images for specific brain regions, which generate age changes in white matter development (Barkovich 2002; Dietrich et al. 1988; McArdle et al. 1987). Given this technique, it is possible to measure individual differences in development by region and relate these individual regional differences in development to individual differences in children's self-representation.

To study this problem, Lewis and Carmody (2008) studied 15 infants from 15 to 30 months of age and related their development of particular brain regions to their scores on a self-referential

scale made up of mirror self-recognition, personal pronouns, and pretend play. The findings from this study indicate that the degree of brain development in a specific region, independent of age, is related to the emergence of children's self-representation. This held for the self-referential score, as well as the three components that make up the score. It is the degree of development in the left temporoparietal junction that is most related to self-representational behavior. This is consistent with other findings that implicate the temporoparietal junction in its role in self-representational behavior (Samson et al. 2004; Saxe et al. 2004; Saxe and Kanwisher 2003). This brain region, as well as regions located nearby, has been found to be activated during several types of self-representational behaviors, such as when subjects engage in a theory-of-mind task relative to reading sentences (Fletcher et al. 1995; Gallagher et al. 2000; Mitchell et al. 2002), when subjects judge whether adjectives are relevant to themselves (Fossati et al. 2003; Macrae et al. 2004), and when subjects hear their own name (Carmody and Lewis 2006).

However, it should be noted that studies have found evidence for both left- and right-hemisphere involvement in self-representation; the left hemisphere shows greater activation on tasks involving self-representation, whereas the right hemisphere shows activation in tasks involving self in comparison with others. For example, Ruby and Decety (2001), using PET, reported left parietal activation when subjects mentally simulated an action with a first-person perspective and right parietal activation when subjects simulated an action with a third-person perspective (i.e., imagining the action of the other). In addition, Turk et al. (2002) found in a study of face recognition in a split-brain patient that the left hemisphere in comparison with the right hemisphere showed more activation to self than to a familiar face, whereas the right hemisphere showed more activation to familiar faces of others than to the self.

This work on typically developing children has important implications when we study children with ASD, a topic discussed next.

Self-Reflection and Self-Referential Behavior in Children with ASD

This section will first examine the abilities of children with ASD to show self-referential behavior. Following this, the examination of brain differences in children with ASD and self-referential behavior will be considered.

Self-Referential Abilities in Children with ASD

The developmental markers of self-referential behavior are delayed or absent in children with ASD. Looking at self-referential behavior found in 2-year-old typically developing children, Lewis and Ramsay (2004) showed that mirror recognition, personal pronoun use, and other-directed pretend play were related and occurring at approximately the same time. Children who recognized themselves in mirrors also showed personal pronoun use and engaged in complex pretend play that involved themselves and another. The data on typically developing children show that between 15 and 24 months they come to recognize themselves in mirrors (Lewis and Brooks-Gunn 1979b; Lewis and Ramsay 2004) and that personal pronoun usage also develops in the second year (Howe et al. 2003; Pipp et al. 1987).

Regardless of age, children with ASD show deficits in personal pronoun use and in other-directed pretend play. Personal pronouns are not used or are used inappropriately by children with autistic disorder (Lee et al. 1994). For example, children diagnosed with autism with an average mental age of 5–6 years were compared on production of personal pronouns to a group of language-delayed children matched in mental age to the autistic group (Loveland and Landry 1986). The children with autism had more difficulties producing correct personal pronouns than the language-delayed group indicating that children with autism are different from typically developing children by more than mental age deficiency differences.

Children with autistic disorder tend to engage in self-directed play rather than play involving others (Charman and Baron-Cohen 1997; Sigman 1998). Although children with autistic disorder

have the capacity to copy symbolic play, to perform self-directed symbolic play, and to imitate pretend play when prompted, these children produce fewer novel and unprompted pretend play acts relative to typically developing children (Rutherford and Rogers 2003).

In a study, Carmody and Lewis (2012) compared self-referential behaviors of a group of children with ASD who were 4 years of age with a group of typically developing children of 2 years of age. By 24 months, 100 % of the typically developing children showed self-recognition in mirrors compared to only 55 % of the 4-year-old children with ASD. Eighty-three percent of the typically developing 2-year-olds had personal pronoun usage, while only 60 % of the 4-year-olds with ASD did. Finally, while 80 % of the typically developing 2-year-olds showed pretense, only 35 % of the 4-year-olds with ASD showed pretense. Thus, these data are consistent with other studies which indicate that in children with ASD, self-referential behavior is either delayed or absent in comparison to typically developing children. It does emerge for some children with ASD at 4–6 years. However, there are a sizable number of children with ASD who are more severely affected who do not show these self-referential behaviors at all.

Several reports have suggested that mental age is the determining factor in the emergence of self-recognition and that once the mental age of 2 years is achieved, children with ASD will have self-recognition (Dawson and McKissick 1984; Ferrari and Matthews 1983; Spiker and Ricks 1984). The belief that mental age is the major factor in the inability of children with ASD to show self-recognition is still widely held. For example, Lind and Bowler in 2009 stated that for the mirror recognition task that "...a number of studies (Dawson and McKissick 1984; Ferrari and Matthews 1983; Neuman and Hill 1978; Spiker and Ricks 1984) have demonstrated that children with ASD are capable of mirror self-recognition at a mental age of 18 months (p. 643)."

That this capacity emerges later has been used to suggest that the lack of self-referential behavior in children with ASD is a function of mental age. For example, Dawson and McKissick (1984),

using the mirror recognition task, make such an argument. However, mental age alone cannot account for the failure to recognize oneself in the mirror. For example, Ferrari and Matthews (1983) found that the mirror self-recognizers having ASD had on average a mental age of 38 months, while non-recognizers had an average mental age of 22 months. Given repeated demonstrations that show that 80 % of typically developing children with a mental age of 21 months show self-recognition, the failure of the children with ASD to show self-recognition with an average mental age of 22 months does not support the idea that mental age alone accounts for the failure of children with ASD to show self-recognition. Given that social skills in children with ASD are more than two standard deviations below those predicted by their mental age and that deficits in social skills in children with ASD have been reported as early as 12 months (Bryson et al. 2007; Volkmar et al. 1993; Zwaigenbaum et al. 2005), it may be the case that mental age is not central to the failure of children with ASD in developing self-referential behavior.

In order to look at this question, Carmody and Lewis (2012) looked at the mental age of those 4-year-old children with ASD who passed and failed the three self-referential tasks – mirror recognition, personal pronouns, and pretense. For the latter two, there were no mental age differences, while only mirror recognition showed a mental age effect. Even so, the mental ages of the children with ASD who failed the mirror recognition task had mental ages well above 24 months (in fact, 35 months). Given these findings, mental age alone cannot account for these differences.

The nature of the origins of self-representational behavior remains to be determined. Many have argued for experiential factors in this development for typically developing children, arguing that social interactions give rise to self-referential behavior, and as a result of impoverished experiences of the child in relation to other, children with ASD fail to develop (Cooley 1902; Duval and Wicklund 1972; Hobson et al. 2006; Keller et al. 2004). Another view is that the emergence of self-representational behavior in typically developing children is a

brain maturational event (Leslie 2002; Lewis 2003b). Self-representation appears to emerge within an age window across many cultures (Keller et al. 2004; Priel and de Schonen 1986; Vyt 2001). In typically developing children, there is no mirror recognition before 15 months, but by 24 months all children exhibit it. Lewis and Carmody (2008) have shown that self-representation is related to the maturation of left temporoparietal junction (TPJ) in typically developing children. It is likely, therefore, that the cause of the deficits in self-representation in ASD is multiply determined. While both views of the origins of deficits in children with autism have compelling features, it is likely the case that brain dysfunctions affect both parent–child interactions and self-referential behavior in some epigenetic way to produce the defects we see in the 2–5-year period.

Brain Function Differences Between Typically Developing Children and Children with ASD

There has been considerable study of brain differences in typically developing children and children with ASD. There is too extensive a literature to include, and therefore only some general comments on brain size and function as they relate to self-referential behavior will be discussed here.

Brain imaging studies of children with autism have revealed the atypical growth patterns of the brain relative to typically developing children. For example, by two or three years of age, autistic boys have brain volumes larger than normal average in the cerebrum and cerebellum. This early excessive growth fails to be sustained as their pace of growth falls significantly below normal development in middle childhood (Amaral et al. 2008; Bauman and Kemper 2005). Not only are these overall differences indicative of different brains in the children with autism relative to those with typical development but specific brain region differences also have been found. There is some evidence that the frontal lobes are different in children with autism. It should be noted that Lewis and Carmody (2008) found that the temporal lobes and some frontal regions are associated with self-referential ability in typical development (see also Samson et al. 2004; Saxe et al. 2004).

Studying brain region development in children with ASD, Carmody and Lewis used similar MRI procedures as in earlier studies (Carmody et al. 2007; Carmody and Lewis 2006, 2010). In the 2010 paper, they looked at myelination differences by region, those including the medial frontal cortex, the temporal parietal junction, and temporal poles. Data based on typically developing children served as a reference for age-expected values of maturation for each region, and the results showed that for both the left and right medial frontal cortex, children with ASD showed significantly greater maturation than the typically developing children. However, the left temporal parietal junction of the children with ASD showed significantly less maturation relative to the typically developing group; the right temporal parietal junction showed no group differences. These data support their contention that children with ASD show specific brain region maturation differences relative to the non-ASD children such that the children with ASD show maturation in the frontal region which is overdeveloped, while they show underdevelopment in the left temporal parietal junction region. These findings of overdevelopment in the medial frontal cortex compliment the reports of deviant growth in the frontal region as reported by others (Amaral et al. 2008). The underdevelopment in the left temporal parietal junction in children with autism is of interest given the relation of this region with the emergence of self-reflection. These brain regions that show over- and underdevelopment in the autism group are those involved in various tasks of self-representation and the self in comparison to others (Frith 2001). More recently Carmody and Lewis compared the scores on self-referential behavior with some selected brain region volumes in children with ASD and found a significant association with the left temporal pole, the left temporal cortex, and the left medial prefrontal cortex, such that the greater the volume of white matter, the lower the self-referential scores (Carmody and Lewis in preparation). This demonstration in children with ASD of the relation between the left temporal region with self-recognition, personal pronouns, and pretend play adds further support for the brain

maturation hypothesis. The findings of brain region overdevelopment in frontal regions, while at the same time underdevelopment in the left temporal parietal region, suggest that autism is not a generalized delay of brain maturation but rather a variation in the timing of maturation that is specific to regions related to self-reflection.

Conclusion

Children with ASD have difficulties with self-referential behavior which is likely due to multiple causes, chiefly brain function difficulties. The failure of self-referential behavior development, either its delay or absence, has serious consequences for their social and emotional behavior, for without the self-referential abilities, a wide variety of skills which require them will be absent (Lewis 2014).

It also should be noted that the absence of these self-referential abilities – mirror recognition, personal pronoun usage, and pretense – can be used as markers which can aid in the diagnosis of autism. Given that the national average for the diagnosis of autism is still about 4 years and given that typically developing children gain these skills by 2 years, the measurement of the presence of self-referential ability can be used as an aid to early diagnosis.

References

- Allen, J. W. P., & Lewis, M. (in preparation). Who peeks and who lies: Biological, cognitive, emotional, and socialization correlates.
- Amaral, D. G., Schumann, C. M., & Nordahl, C. W. (2008). Neuroanatomy of autism. *Trends in Neurosciences*, 31(3), 137–145.
- Anjari, M., Srinivasan, L., Allsop, J. M., Hajnal, J. V., Rutherford, M. A., Edwards, A. D., et al. (2007). Diffusion tensor imaging with tract-based spatial statistics reveals local white matter abnormalities in preterm infants. *NeuroImage*, 35(3), 1021–1027.
- Asendorpf, J. B. (2002). Self-awareness, and secondary representation. In A. N. Meltzoff & W. Prinz (Eds.), *The imitative mind: Development, evolution, and brain bases. Cambridge studies in cognitive perceptual development* (pp. 63–73). New York: Cambridge University Press.
- Asendorpf, J. B., & Baudonniere, P. M. (1993). Self-awareness and other-awareness: Mirror self-recognition and synchronic imitation among unfamiliar peers. *Developmental Psychology*, 29(1), 88–95.
- Asendorpf, J. B., Warkentin, V., & Baudonniere, P. M. (1996). Self-awareness and other-awareness II: Mirror self-recognition, social contingency awareness, and synchronic imitation. *Developmental Psychology*, 32(2), 313–321.
- Barkovich, A. J. (2000). Concepts of myelin and myelination in neuroradiology. *American Journal of Neuroradiology*, 21(6), 1099–1109.
- Barkovich, A. J. (2002). Magnetic resonance imaging: Role in the understanding of cerebral malformations. *Brain Development*, 24(1), 2–12.
- Barkovich, A. J. (2005). Magnetic resonance techniques in the assessment of myelin and myelination. *Journal of Inherited Metabolic Disease*, 28(3), 311–343.
- Bauman, M. L., & Kemper, T. L. (2005). Neuroanatomic observations of the brain in autism: A review and future directions. *International Journal of Developmental Neuroscience*, 23(2–3), 183–187.
- Bertenthal, B. L., & Fischer, K. W. (1978). Development of self-recognition in the infant. *Developmental Psychology*, 14, 44–50.
- Bischof-Kohler, D. (1994). Self-objectification and other-oriented emotions: Self-recognition, empathy, and prosocial behavior in the second year. *Zeitschrift für Psychologie*, 202, 349–377.
- Bretherton, I. (1991). *Intentional communication and the development of an understanding of mind*. Hillsdale: Erlbaum Associates.
- Bryson, S. E., Zwaigenbaum, L., Brian, J., Roberts, W., Szatmari, P., Rombough, V., & McDermott, C. (2007). A prospective case series of high-risk infants who developed autism. *Journal of Autism and Developmental Disorders*, 37, 12–24.
- Butterworth, G. (1992). Origins of self-perception in infancy. *Psychological Inquiry*, 3, 103–111.
- Carmody, D. P., & Lewis, M. (in preparation). Self representation and brain enlargement in children with Autism Spectrum Disorder.
- Carmody, D. P., & Lewis, M. (2006). Brain activation when hearing one's own and others' names. *Brain Research*, 1116, 153–158.
- Carmody, D. P., & Lewis, M. (2010). Regional white matter development in children with autism spectrum disorders. *Developmental Psychobiology*, 52(8), 755–763.
- Carmody, D. P., & Lewis, M. (2012). Self representation in children with and without Autism Spectrum Disorders. *Child Psychiatry and Human Development*, 43(2), 227–237.
- Carmody, D. P., Dunn, S. M., Boddie-Willis, A. S., DeMarco, J. K., & Lewis, M. (2004). A quantitative measure of myelination development in infants, using MR images. *Neuroradiology*, 46, 781–786.
- Carmody, D. P., Moreno, R., Mars, A., Seshadri, K., Lambert, G., & Lewis, M. (2007). Brain activation to social

- words in a sedated child with autism. *Journal of Autism and Developmental Disorders*, 37, 1381–1385.
- Carpendale, J. I. M., & Lewis, C. (2010). The development of social understanding: A relational perspective. In W. F. Overton & R. M. Lerner (Eds.), *The handbook of life-span development, Vol. 1: Cognition, biology, and methods* (pp. 584–627). Hoboken: Wiley.
- Carpenter, M., Nagel, K., & Tomasello, M. (1999). Social cognition, joint attention, and communicate competence from 9 to 15 months of age. *Monographs of the Society for Research in Child Development*, 63, 1–212.
- Chandler, M. J., & Birch, S. A. J. (2010). The development of knowing. In W. F. Overton & R. M. Lerner (Eds.), *The handbook of life-span development, Vol. 1: Cognition, biology, and methods* (pp. 671–719). Hoboken: Wiley.
- Charman, T., & Baron-Cohen, S. (1997). Brief report: Prompted pretend play in autism. *Journal of Autism and Developmental Disorders*, 27, 325–332.
- Cicchetti, D., & Sroufe, L. A. (1978). An organizational view of affect: Illustration from the study of Down's syndrome infants. In M. Lewis & L. A. Rosenblum (Eds.), *The development of affect* (Vol. 1, pp. 309–350). New York: Plenum Press.
- Cooley, C. H. (1902). *Human nature and social order*. New York: Scribner's.
- Damasio, A. (1999). *The feeling of what happens: Body and emotion in the making of consciousness*. New York: Harcourt Brace.
- Dawson, G., & McKissick, F. C. (1984). Self-recognition in autistic children. *Journal of Autism and Developmental Disorders*, 14, 383–394.
- Dietrich, R. B., Bradley, W. G., Zaragoza, E. J. T., Otto, R. J., Taira, R. K., Wilson, G. H., et al. (1988). MR evaluation of early myelination patterns in normal and developmentally delayed infants. *American Journal of Roentgenology*, 150(4), 889–896.
- Dubois, J., Hertz-Pannier, L., Dehaene-Lambertz, G., Cointepas, Y., & Le Bihan, D. (2006). Assessment of the early organization and maturation of infants' cerebral white matter fiber bundles: A feasibility study using quantitative diffusion tensor imaging and tractography. *NeuroImage*, 30(4), 1121–1132.
- Duval, S., & Wicklund, R. A. (1972). *A theory of objective self-awareness*. San Diego: Academic.
- Fein, G. G. (1975). A transformational analysis of pretending. *Developmental Psychology*, 11, 291–296.
- Ferrari, M., & Matthews, W. S. (1983). Self-recognition deficits in autism: Syndrome-specific or general developmental delay? *Journal of Autism and Developmental Disorders*, 13, 317–324.
- Fletcher, P. C., Happe, F., Frith, U., Baker, S. C., Dolan, R. J., Frackowiak, R. S., et al. (1995). Other minds in the brain: A functional imaging study of "theory of mind" in story comprehension. *Cognition*, 57(2), 109–128.
- Fossati, P., Hevenor, S. J., Graham, S. J., Grady, C., Keightley, M. L., Craik, F., et al. (2003). In search of the emotional self: An fMRI study using positive and negative emotional words. *American Journal of Psychiatry*, 160(11), 1938–1945.
- Friedman, O. (2011). *Principles guiding young children's reasoning about ownership*. Presentation at the October 11 Colloquium at the Rutgers University Center for Cognitive Science, New Brunswick.
- Frith, U. (2001). Mind blindness and the brain in autism. *Neuron*, 32(6), 969–979.
- Gallagher, H. L., Happe, F., Brunswick, N., Fletcher, P. C., Frith, U., & Frith, C. D. (2000). Reading the mind in cartoons and stories: An fMRI study of 'theory of mind' in verbal and nonverbal tasks. *Neuropsychologia*, 38(1), 11–21.
- Geertz, C. (1984). On the nature of anthropological understanding. In R. A. Shweder & R. A. Levine (Eds.), *Cultural theory: Essays on mind, self, and emotion*. Cambridge, England: Cambridge University Press.
- Harley, K., & Reese, E. (1999). Origins of autobiographical memory. *Developmental Psychology*, 35, 1338–1348.
- Harter, S. (1983). Developmental perspectives on the self-system. In E. M. Hetherington (Ed.), *Handbook of child psychology: Vol. 4. Socialization, personality and social development* (pp. 275–385). New York: Wiley.
- Hobson, R. P. (1990). On the origins of self and the case of autism. *Development and Psychopathology*, 2, 163–181.
- Hobson, P. R., Chidambi, G., Lee, A., & Meyer, J. (2006). Foundations for self-awareness: An exploration through autism. *Monographs of the Society for Research in Child Development*, 71(2), vii–166.
- Howe, M. L., Courage, M. L., & Edison, S. (2003). When autobiographical memory begins. *Developmental Review*, 23, 471–494.
- Huttenlocher, J., & Higgins, E. T. (1978). Issues in the study of symbolic development. In W. Collins (Ed.), *Minnesota symposia on child psychology* (Vol. 11, pp. 98–140). Hillsdale: Erlbaum.
- Kampe, K. K., Frith, C. D., & Frith, U. (2003). "Hey John": Signals conveying communicative intention toward the self-activate brain regions associated with "mentalizing", regardless of modality. *Journal of Neuroscience*, 23(12), 5258–5263.
- Keller, H., Yovai, R., Borke, J., Kartner, J., Jensen, H., & Papaligoura, Z. (2004). Developmental consequences of early parenting experiences: Self-recognition and self-regulation in these cultural communities. *Child Development*, 75, 1745–1760.
- Kopp, C. B. (1982). Antecedent of self-regulation: A developmental perspective. *Developmental Psychology*, 18, 199–214.
- Laing, R. D. (1970). *Knots*. New York: Pantheon.
- Lee, A., Hobson, R. P., & Chiat, S. (1994). I, you, me, and autism: An experimental study. *Journal of Autism and Developmental Disorders*, 24, 155–176.
- Leslie, A. M. (1987). Pretense and representation: The origin of "Theory of Mind". *Psychological Review*, 94, 412–426.

- Leslie, A. M. (2002). Pretense and representation revisited. In N. L. Stein & P. J. Bauer (Eds.), *Representation, memory and development: Essays in honor of Jean Mandler* (pp. 103–114). Mahwah: Erlbaum.
- Lewis, M. (1992). *Shame: The exposed self*. New York: Free Press.
- Lewis, M. (2003a). The development of self-consciousness. In J. Roessler & N. Eilan (Eds.), *Agency and self-awareness: Issues in philosophy and psychology* (pp. 275–295). New York: Oxford University Press.
- Lewis, M. (2003b). The emergence of consciousness and its role in human development. *Annals of the New York Academy of Science*, 1001, 104–133.
- Lewis, M. (2014). *The rise of consciousness and the development of emotional life*. New York: Guilford.
- Lewis, M., & Brooks-Gunn, J. (1979a). Toward a theory of social cognition: The development of self. In I. Uzgiris (Ed.), *New directions in child development: Social interaction and communication during infancy* (pp. 1–20). San Francisco: Jossey-Bass.
- Lewis, M., & Brooks-Gunn, J. (1979b). *Social cognition and the acquisition of self*. New York: Plenum.
- Lewis, M., & Carmody, D. (2008). Self representation and brain development. *Developmental Psychology*, 44(5), 1329–1334.
- Lewis, M., & Ramsay, D. (2004). Development of self-recognition, personal pronoun use, and pretend play during the 2nd year. *Child Development*, 75(6), 1821–1831.
- Lewis, M., Stanger, C., & Sullivan, M. W. (1989a). Deception in three-year-olds. *Developmental Psychology*, 25(3), 439–443.
- Lewis, M., Sullivan, M. W., Stanger, C., & Weiss, M. (1989b). Self-development and self-conscious emotions. *Child Development*, 60, 146–156.
- Lind, S. E., & Bowler, D. M. (2009). Delayed self-recognition in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 39, 643–650.
- Loveland, K. A., & Landry, S. H. (1986). Joint attention and language in autism and developmental language delay. *Journal of Autism and Developmental Disorders*, 16, 335–349.
- Lowe, M. (1975). Trends in the development of representational play in infants from one to three years – An observational study. *Journal of Child Psychology and Psychiatry*, 16, 33–47.
- Macrae, C. N., Moran, J. M., Heatherton, T. F., Banfield, J. F., & Kelley, W. M. (2004). Medial prefrontal activity predicts memory for self. *Cerebral Cortex*, 14(6), 647–654.
- McArdle, C. B., Richardson, C. J., Nicholas, D. A., Mirfakhraee, M., Hayden, C. K., & Amparo, E. G. (1987). Developmental features of the neonatal brain: MR imaging. Part I. Gray-white matter differentiation and myelination. *Radiology*, 162, 223–229.
- McClelland, M. M., Geldhof, G. J., Cameron, C. E., & Wanless, S. B. (2014). Development and self-regulation. In W. F. Overton & P. C. Molenaar (Eds.), *Theory and Method. Vol. 1: Handbook of child psychology and developmental science* (7th ed., Editor-in-Chief: Richard M. Lerner). Hoboken: Wiley.
- McCune, L. (1995). A normative study of representation play at the transition to language. *Developmental Psychology*, 31, 198–206.
- McCune-Nicolich, L. (1981). Toward symbolic functioning: Structure of early pretend games and potential parallels with language. *Child Development*, 52(3), 785–797.
- Meltzoff, A. N. (1995). Understanding the intentions of others: Re-enactment of intended acts by 18-month-old children. *Developmental Psychology*, 31, 838–850.
- Mitchell, J. P., Heatherton, T. F., & Macrae, C. N. (2002). Distinct neural systems subserve person and object knowledge. *Proceedings of the National Academy of Sciences of the USA*, 99(23), 15238–15243.
- Mori, S., Kaufmann, W. E., Davatzikos, C., Stieltjes, B., Amodei, L., Fredericksen, K., et al. (2002). Imagining cortical association tracts in the human brain diffusion-tensor-based axonal tracking. *Magnetic Resonance in Medicine*, 47(2), 215–223.
- Moses, J., & Chandler, M. J. (1992). Traveler's guide to children's theories of mind. *Psychological Inquiry*, 3, 285–301.
- Neuman, C. J., & Hill, S. D. (1978). Self-recognition and stimulus preference in autistic children. *Developmental Psychobiology*, 11, 571–578.
- Nicolich, L. (1977). Beyond sensorimotor intelligence: Assessment of symbolic maturity through analysis of pretend play. *Merrill-Palmer Quarterly*, 23, 89–102.
- Overton, W. F. (2006). Developmental psychology: Philosophy, concepts, methodology. In R. M. Lerner & W. Damon (Eds.), *Handbook of child psychology. Vol. 1: Theoretical models of human development* (6th ed., pp. 18–88). Hoboken: Wiley.
- Paus, T. (2005). Mapping brain maturation and cognitive development during adolescence. *Trends in Cognitive Sciences*, 9(2), 60–68.
- Paus, T., Collins, D. L., Evans, A. C., Leonard, G., Pike, B., & Zijdenbos, A. (2001). Maturation of white matter in the human brain: A review of magnetic resonance studies. *Brain Research Bulletin*, 54(3), 255–266.
- Piaget, J. (1962). *Play, dreams, and imitation in childhood* (trans: Gatlegno, C., & Hodgson, F. M.). New York: Norton. (Original work published 1951 in French)
- Pipp, S., Fischer, K. W., & Jennings, S. (1987). Acquisition of self- and mother knowledge in infancy. *Developmental Psychology*, 23, 86–96.
- Priel, B., & de Schonen, S. (1986). Self-recognition: A study of a population without mirrors. *Journal of Experimental Child Psychology*, 41, 237–250.
- Rochat, P. (2009). *Others in mind: Social origins of self-consciousness*. New York: Cambridge University Press.

- Ross, C. A. (1989). *Multiple personality disorder*. New York: Wiley.
- Ruby, P., & Decety, J. (2001). Effect of subjective perspective taking during simulation of action: A PET investigation of agency. *Nature Neuroscience*, 4(5), 546–550.
- Rutherford, M. D., & Rogers, S. J. (2003). Cognitive underpinnings of pretend play in autism. *Journal of Autism and Developmental Disorders*, 33, 289–302.
- Samson, D., Apperly, I. A., Chiavarino, C., & Humphreys, G. W. (2004). Left temporoparietal junction is necessary for representing someone else's belief. *Nature Neuroscience*, 7, 499–500.
- Saxe, R., & Kanwisher, N. (2003). People thinking about thinking people. The role of the temporo-parietal junction in "theory of mind". *NeuroImage*, 19(4), 1835–1842.
- Saxe, R., Carey, S., & Kanwisher, N. (2004). Understanding other minds: Linking developmental psychology and functional neuroimaging. *Annual Review of Psychology*, 55, 87–124.
- Sigman, M. (1998). The Emanuel Miller Memorial Lecture 1997. Change and continuity in the development of children with autism. *Journal of Child Psychology and Psychiatry*, 39, 817–827.
- Souweidane, M. M., Kim, K. H., McDowall, R., Ruge, M. I., Lis, E., Krol, G., et al. (1999). Brain mapping in sedated infants and young children with passive-functional magnetic resonance imaging. *Pediatric Neurosurgery*, 30(2), 86–92.
- Spiker, D., & Ricks, M. (1984). Visual self-recognition in autistic children: Developmental relationships. *Child Development*, 55, 214–225.
- Stern, D. (1985). *The interpersonal world of the infant*. New York: Basic Books, Inc.
- Thompson, D. K., Warfield, S. K., Carlin, J. B., Pavlovic, M., Wang, H. X., Bear, M., et al. (2007). Perinatal risk factors altering regional brain structure in the preterm infant. *Brain*, 130, 667–677.
- Toga, A. W., Thompson, P. M., & Sowell, E. R. (2006). Mapping brain maturation. *Trends in Neurosciences*, 29(3), 148–159.
- Turk, D. J., Heatherton, T. F., Kelley, W. M., Funnell, M. G., Gazzaniga, M. S., & Macrae, C. N. (2002). Mike or me? Self-recognition in a split-brain patient. *Nature Neuroscience*, 5, 148–159.
- Volkmar, F. R., Carter, A., Sparrow, S. S., & Cicchetti, D. V. (1993). Quantifying social development in autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 627–632.
- Von Bertalanffy, L. (1967). *Robots, men, and minds*. New York: Brazillies.
- Vyt, A. (2001). Processes of visual self-recognition in infants: Experimental induction of mirror experience via video self-image presentation. *Infant and Child Development*, 10, 173–187.
- Wellman, H. M. (1990). *The child's theory of mind*. Cambridge, MA: MIT Press.
- Zahn-Waxler, C., Radke-Yarrow, M., Wagner, E., & Chapman, M. (1992). Development of concern for others. *Developmental Psychology*, 28, 126–136.
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, 23, 143–152.

Self-Regulation

► Mutual Regulation

Self-Reliance

► Self-Advocacy

Self-Report Autism Scales for Adults

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Synonyms

Adult Repetitive Behaviors Questionnaire-2-Revised (RBQ-2A-R); Autism spectrum condition (ASC); Autistic Spectrum Quotient (AQ-9); Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5); Systemizing Quotient (SQ-7)

Description

While diagnosticians make individual diagnoses of the autism spectrum condition (ASC) in clinical settings based on direct observations of the patients and reports by caregivers, empirical researchers seeking to gauge autistic-like tendencies in the nonclinical adult population often rely on self-

report measures, such as the *AQ-50* (Baron-Cohen et al. 2001), *RBQ-2A* (Barrett et al. 2015), and *SQ-40* (Baron-Cohen et al. 2003). These self-report autism scales and their abbreviated versions have been used as screening tools as well as in empirical research.

Despite their popularity in empirical work, they have presented either factorial validity issues (*AQ* and *SQ*), needed updating for a modern context (*SQ*), or have never been further tested using confirmatory factor analysis (*RBQ-2A*). Given their influence in the literature, it is important to ensure their psychometric validity and refine them as needed to provide rigorous and consistent measurement for future empirical researchers.

Based on three rounds of development and testing using different US and global samples from general adult populations, Ronnie Jia, PhD, Zachary Steelman, PhD, and Heather Jia, PhD reported in the *Journal of Autism and Developmental Disorders* (2019) refined versions of these self-report instruments that are psychometrically sound, parsimonious, and generalizable across nonclinical adult populations. The resulting *AQ-9*, consisting of two factors, *social communication* and *attention to detail*, now mirrors the current dual diagnostic criteria in the *DSM-5*. The *RBQ-2A-R* has now been refined through CFA for the first time. The new *SQ-7* scale also has updated content. All three refined scales demonstrate satisfactory psychometric validity and parsimony and now provide evidence of their appropriateness for empirical research.

Historical Background

To be appropriate for empirical survey-based research, a measurement instrument must possess satisfactory *psychometric validity*, such as reliability and factorial validity (Hair et al. 1998). While most researchers follow the same guidelines on scale reliability (e.g., Cronbach's $\alpha \geq 0.70$, Nunnally 1978), there has been less attention and consistency in the types of evidence required to demonstrate factorial validity (e.g.,

convergent validity, discriminant validity, measurement invariance), especially when evaluating these specific autism-related scales.

Many studies have relied on exploratory techniques, such as exploratory factor analysis (EFA) and principal component analysis (PCA), which often lead to significant over-factoring (Frazier and Youngstrom 2007), rather than confirmatory factor analysis (CFA), which can evaluate alternative a priori factor structures for the best model fit and effectively establish factorial validity (e.g., MacCallum et al. 1992). In addition to overall model fit, items should also have high loadings on the intended factors (convergent validity) and low loadings on other factors (discriminant validity). Thresholds for individual item loadings have also been recommended (e.g., 0.71 = excellent, 0.63 = very good, 0.55 = good, 0.45 = fair, and 0.32 = poor; Tabachnick and Fidell 2007). While it is critical for a factor to have at least three items (Kline 2010), it is desirable for a factor to have at least four loadings of 0.60 or higher (Guadagnoli and Velicer 1988).

Besides psychometric validity, another characteristic of a desirable measurement scale for empirical survey-based research is *parsimony*. In survey studies that include a large number of measurement items, researchers may be rightfully concerned about participants' response burden, which can lead to decreased data quality and response rates (Veale and Williams 2015). While researchers need valid measures with strong loadings and domain coverage of their focal constructs, scale parsimony is also necessary to encourage respondent participation and reduce response burden and bias (e.g., Dillman 2000).

Based on the above two key criteria (i.e., psychometric validity and parsimony), the *AQ*, *RBQ-2A*, and *SQ* are examined for their appropriateness for empirical survey-based research.

Autistic Spectrum Quotient (AQ)

The original *AQ* scale (Baron-Cohen et al. 2001) consists of 50 items in 5 factors: *social skill*, *attention switching*, *attention to detail*, *communication*, and *imagination*. Subsequent research (e.g., Austin 2005; Hurst et al. 2007; Kloosterman

et al. 2011) has recommended a three-factor model, including *social skills*, *attention to detail*, and *communication/mindreading*. However, the consistent lower reliability and convergent validity in the third factor across these studies provide cumulative evidence for a two-factor *AQ* model (*social skills* and *attention to detail*), which would be consistent with the current two-factor ASC diagnostic criteria specified in *DSM-5*: “persistent deficits in social communication and social interaction” and “restricted, repetitive patterns of behavior, interests, or activities” (American Psychiatric Association 2013). Therefore, continued evaluation and refinement of the *AQ* is needed in keeping with the current *DSM-5* criteria, which will allow for the further alignment between academic research and clinical practice.

Another noteworthy effort to achieve a parsimonious *AQ* measure is Allison et al.’s (2012) *AQ-10*, which was proposed as a rapid screening tool and was constructed by choosing the two items with the highest discrimination index values from each of the five *AQ-50* scales (Baron-Cohen et al. 2001). However, evidence for its factorial validity was not reported, and its five-factor structure is unlikely to hold in view of findings in Austin (2005), Hurst et al. (2007), and Kloosterman et al. (2011). Additionally, since each of the subscales consists of only two items, rather than the recommended thresholds of at least three items per factor (Kline 2010), they may suffer from low reliability as well as model estimation problems (Kline 2010), which can hinder their value when used in survey-based research.

Since the factorial validity of the *AQ-10* has never been reported in the literature, and the scale is frequently used in research (e.g., Jackson et al. 2018), it is necessary that its psychometric soundness be empirically tested. Despite its potential shortcomings, it has the distinct advantage of being the briefest *AQ* measure. In view of its parsimony, it was still used as a starting point for constructing a parsimonious and psychometrically sound *AQ* measure that is also theoretically linked to the *DSM-5* diagnostic criteria. (The *AQ-50* was not chosen because it is neither parsimonious nor psychometrically sound.)

Adult Repetitive Behavior Questionnaire (RBQ-2A)

The *RBQ-2A* (Barrett et al. 2015) focuses on a specific set of autism-related behaviors, i.e., restricted and repetitive behaviors. Developed using principal component analysis (PCA), it consists of 14 items in two subscales: *repetitive motor behavior* (RMB) and *insistence on sameness* (IoS). However, there is limited evidence of its factorial validity as three items have “poor” loadings ($\lambda < 0.45$, Tabachnick and Fidell 2007) and another three item loadings are in the “fair” range ($\lambda < 0.55$, Tabachnick and Fidell 2007). Given its relative newness, no follow-up research using this scale has retested and reported its psychometric properties using CFA. It is therefore necessary to further validate and refine the scale for future empirical research.

Systemizing Quotient (SQ)

The 40-item *SQ* scale (*SQ-40*, Baron-Cohen et al. 2003) was proposed as a measure of autistic tendency based on the empathizing-systemizing theory (Baron-Cohen 2002). Based on CFA results, Ling et al. (2009) found that a single-factor model for *SQ-40* has poor fit and recommended a 4-factor, 18-item solution, including *technicality*, *topography*, *DIY*, and *structure*. However, the *topography* and *DIY* subscales each had less than 3 items with “good” loadings, and overall, 8 of the 18 items have loadings that are less than “good” ($\lambda < 0.55$, Tabachnick and Fidell 2007), indicating limited convergent validity in these two subscales and the possibility that a 2-factor *SQ* model (including *technicality* and *structure*) is a better fit.

Veale and Williams (2015) tested a single-factor, eight-item measure (*SQ-8*). However, most items have lower loadings ($\lambda < 0.55$, Tabachnick and Fidell 2007). Therefore, further development of the *SQ* scale will likely require the higher loading items from the *SQ-8* to be supplemented by additional items from the *SQ-18* (Ling et al. 2009) to enhance scale validity.

In sum, though the existing *AQ*, *RBQ-2A*, and *SQ* measures have been used in many empirical studies, they still exhibit significant psychometric

issues and require additional refinement and testing to be used in future research.

Building on these prior efforts, Jia et al. (2019) reported three consecutive studies to rigorously evaluate, develop, and refine these three frequently used scales to improve their psychometric validity and parsimony as well as ensure applicability to the general adult populations. Study 1 performs a baseline examination by testing the briefest existing versions of these measures with a sample of US college students before they are refined in Study 2 through adding or removing items using a worldwide sample of adults. The scales are finally validated in Study 3 with a sample of US adult participants. These consecutive evaluations assess scale reliability, convergent and discriminant validity, factorial invariance, and nomological validity, with the aim of achieving a set of psychometrically valid, parsimonious, and consistent measures in line with established standards in the literature.

Current Knowledge

Study 1: Initial Evaluation

Method

An anonymous online survey consisting of the most parsimonious versions of the three measures, i.e., *AQ-10* (Allison et al. 2012), the *RBQ-2A* (Barrett et al. 2015), and the *SQ-8* (Veale and Williams 2015), was administered with 207 undergraduate students from 2 large public universities in the United States. All items were measured on a 7-point scale from “strongly disagree” to “strongly agree.”

Results

As expected, all five *AQ-10* subscales exhibited low internal consistency ($\alpha_{\text{Attention to Detail}} = 0.21$, $\alpha_{\text{Attention Switching}} = 0.57$, $\alpha_{\text{Communication}} = 0.50$, $\alpha_{\text{Imagination}} = 0.21$, and $\alpha_{\text{Social}} = 0.43$). When all items were evaluated in a single factor, internal consistency remained low ($\alpha = 0.48$). Both *RBQ-2A* subscales, *repetitive motor behavior* and *insistence on sameness*, achieved adequate internal consistency ($\alpha_{\text{RMB}} = 0.80$ and $\alpha_{\text{IoS}} = 0.83$), so did the single-factor *SQ-8* ($\alpha = 0.75$). Having

demonstrated adequate internal consistency (i.e., $\alpha \geq 0.70$), the *RBQ-2A* and *SQ-8* were therefore further tested for validity both within and between the scales through CFA.

When testing the *RBQ-2A* and *SQ-8* using CFA, the two scales were first examined separately. Results show that the two-factor *RBQ-2A* exhibits less than satisfactory model fit ($\chi^2 = 162.98$, $df = 76$, $CFI = 0.94$, $RMSEA = 0.074$ with 90% CI: 0.058–0.090) with the CFI below the recommended thresholds of 0.95 and $RMSEA$ exceeding 0.06 for acceptable model fit (Hu and Bentler 1999). The single-factor *SQ-8* also indicates poor model fit ($\chi^2 = 71.27$, $df = 20$, $CFI = 0.90$, $RMSEA = 0.11$ with 90% CI: 0.08–0.14), and only a single item loading exceeded 0.60.

After testing each scale individually, the two scales were examined together in a single model to evaluate their convergent and discriminant validity. The combined model also provided less than satisfactory fit ($\chi^2 = 373.19$, $df = 206$, $CFI = 0.92$, $RMSEA = 0.063$ with 90% CI: 0.053–0.073). Factor loadings indicate that four *RBQ-2A* items (RMB #1, IoS #1, #2 and #7) have “poor” loadings ($\lambda < 0.45$, Tabachnick and Fidell 2007), which are similar to those reported by Barrett et al. (2015). Thus, these four items should be removed or refined in future steps of scale development.

The *SQ-8* item loading matrix also has low loadings (items #2, #3R, and #4R) that are similar to those reported in prior research (e.g., Veale and Williams 2015), necessitating their removal or refinement in future steps. As also observed by Veale and Williams, Item 1 (“maps”) has significant conceptual overlap with Item 6 (“motorways”) and thus could be removed to increase parsimony. Since only one item has a loading exceeding 0.60, further development of the *SQ* scale requires additional items from the larger *SQ-18* to strengthen its psychometric properties.

Results from Study 1 indicate that, as expected, the *AQ-10* has low internal consistency in all of its five subscales, which likely resulted from its small number of items. Also as expected, the *SQ-8* has low item loadings and poor overall model fit. Thus, neither the *AQ-10* nor the *SQ-8* possesses

satisfactory psychometric properties for empirical survey-based research at this time. Further development and refinement of these two scales require incorporating additional *AQ-26* and *SQ-18* items to enhance their psychometric properties.

Though the *RBQ-2A* scale also exhibits less than satisfactory model fit, its psychometric properties may be improved by simply removing items with low loadings as the number of items in each factor (6 and 8) still far exceeds the recommended minimum of three items per scale (Kline 2010). Such item removal can also further improve its parsimony.

Based on the above results from Study 1, the focus in Study 2 was to improve scale validity by removing unsatisfactory items from the *RBQ-2A* and incorporating and testing additional *AQ* and *SQ* items.

Study 2: Further Refinement

Method

Data were collected from a larger, more diverse adult sample ($N = 355$ participants from 44 countries) recruited on Amazon's Mechanical Turk (MTurk) so as to enhance scale applicability and generalizability to a variety of settings. The use of MTurk data has seen significant growth in recent years in psychology, psychiatry, and other behavioral fields as a way to recruit participants that are more diverse than college students (e.g., Gosling and Mason 2015).

The survey included additional *AQ* and *SQ* items to enhance scale validity. The 6 items from the 3 retained subscales of the *AQ-10*, including *social skills* (#36 and #45), *attention to detail* (#5 and #28), and *communication/mindreading* (#27 and #31), were supplemented by 13 items from these 3 subscales of the larger *AQ-26* that had loadings of 0.40 or higher in prior research (Hurst et al. 2007). The four remaining *SQ-8* items from Study 1 (#5, #6, #7, and #8) were supplemented by six items with loadings over 0.40 from the *technicity* and *structure* subscales of the larger *SQ-18* (Ling et al. 2009). No items from its *topography* or *DIY* subscales were considered as neither factor had at least three items with sufficiently high loadings (Ling et al. 2009).

Additionally, since reverse worded items can cause respondent confusion and mistakes while failing to prevent inattentive or acquiescent answering (van Sonderen et al. 2013), three such *SQ* items were positively restated to increase clarity and improve internal consistency (#11 from “rarely” to “often”, #43 from “I would not” to “I would”, and #51 from “I do not think” to “I think”). Due to satisfactory loadings, the ten *RBQ-2A* items from Study 1 were retained.

Results

Based on the overall CFA factor loading matrix for the three measurement scales, eight *AQ* items (#5, #26, #27, #28, #31, #35, #36, and #45) with loadings in the “poor” range ($\lambda < 0.45$, Tabachnick and Fidell 2007) were dropped, before separately testing each measure. In each individual goodness-of-fit test, modification indices were used to identify and remove items that have high error covariance with other items or load onto non-intended factors (Kline 2010). This process led to the removal of another six items (*AQ* #11 and #38; *RMB* #3; *SQ* #11, #30, and #51) to further increase the psychometric properties of each scale.

After this initial refinement, the goodness-of-fit test results indicate that all three resulting scales achieved satisfactory model fit in their individual CFA tests (*AQ*: $\chi^2 = 58.29$, $df = 26$, $CFI = 0.98$, $RMSEA = 0.059$ with 90% CI : 0.039–0.079; *RBQ-2A*: $\chi^2 = 40.03$, $df = 26$, $CFI = 0.99$, $RMSEA = 0.039$ with 90% CI : 0.008–0.062; *SQ*: $\chi^2 = 19.03$, $df = 13$, $CFI = 0.99$, $RMSEA = 0.037$ with 90% CI : 0.000–0.069), along with acceptable levels of internal consistency (all $\alpha \geq 0.70$), except for *AQ attention to detail* ($\alpha = 0.67$), which is near the threshold. Further, when assessing the three scales together in a holistic model, the model fit remained satisfactory ($\chi^2 = 499.85$, $df = 260$, $CFI = 0.97$, $RMSEA = 0.051$ with 90% CI : 0.044–0.058), indicating adequate psychometric validity and parsimony with the refined scales. The three instruments, hereinafter referred to as the *AQ-9*, *RBQ-2A-R*, and *SQ-7*, were subject to a final test of their convergent and discriminant validity, nomological validity, and factorial invariance.

Study 3: Final Validation

Method

Similar to Study 2, to achieve greater scale applicability and generalizability in Study 3, a sample of 442 US adults were recruited on MTurk. In addition to the *AQ-9*, *RBQ-2A-R*, and *SQ-7* items from Study 2, scales for both neuroticism and extraversion from the Big Five Inventory (BFI, John and Srivastava 1999), which are known correlates of the *AQ* and *SQ* in prior research (e.g., Austin 2005; Schwartzman et al. 2016), were also administered for the purpose of demonstrating scale nomological validity and further evidence of convergent and divergent validity when examining the autism scales with additional constructs.

Factorial invariance tests were conducted to assess the extent to which each of these three scales is equivalent and consistent across different groups (e.g., males and females). We examined configural, metric, scalar, and complete invariance, which provide increasing levels of model strictness across groups to indicate measurement consistency (Longo et al. 2018). Though higher levels of invariance are often hard to achieve “as metric equivalence and, particularly, scalar equivalence are frequently rejected in social science research” (Cheung and Rensvold 2002, p. 601), metric invariance is considered a prerequisite for meaningful cross-group comparison (Bollen 1989).

In this study, invariance tests were conducted between male and female participants, which enable us to link our findings with existing knowledge on ASC, such as higher autistic tendencies in men than in women (e.g., Austin 2005). However, such known gender difference was also expected to make higher levels of invariance (e.g., scalar, complete) unlikely. Thus, our goal was to provide evidence of configural and metric invariance between males and females with a pattern of gender differences that are in keeping with prior literature.

Results

The CFA loading matrix for the three autism scales suggests that each scale exhibits

satisfactory convergent and discriminant validity with items loading primarily on their focal constructs and less so on the others in the model (Kline 2010). Results from three separate goodness-of-fit tests also provide evidence of satisfactory model fit:

- *AQ-9*: social communication (items #15R, #17R, #22, #44R, #47R, $\alpha = 0.92$) and attention to detail (items #6, #12, #19, and #23, $\alpha = 0.80$), with an overall model fit ($\chi^2 = 64.78$, $df = 26$, $CFI = 0.99$, $RMSEA = 0.058$, 90% CI : 0.041–0.076)
- *RBQ-2A-R*: repetitive motor behavior (items #2, #3, #4, #5, and #6, $\alpha = 0.83$) and insistence on sameness (items #3, #4, #5, and #6, $\alpha = 0.84$), with an overall model fit ($\chi^2 = 76.35$, $df = 26$, $CFI = 0.98$, $RMSEA = 0.066$, 90% CI : 0.049–0.084)
- *SQ-7*: technicity (items #5, #20, #33, and #43, $\alpha = 0.82$) and structure (items #13, #37, and #49, $\alpha = 0.73$), with an overall model fit ($\chi^2 = 20.24$, $df = 13$, $CFI = 0.99$, $RMSEA = 0.036$, 90% CI : 0.000–0.064).

Invariance tests between male and female participants indicate that all three scales showed satisfactory model fit and met the ΔCFI threshold for configural and metric invariance, but as anticipated, not scalar or complete invariance due to known gender differences also reported in prior autism research. (See Jia et al. 2019 for details.)

Also as expected, many factors within the three scales are significantly correlated with one another. As further evidence for nomological validity, they are also significantly linked to their known Big Five correlates (e.g., Austin 2005; Schwartzman et al. 2016). For example, *AQ social communication* is negatively related to extroversion ($r = -0.86$), and *RBQ-2A repetitive motor behavior* is positively related to neuroticism ($r = 0.51$).

In sum, despite the frequent use of the *AQ*, *SQ*, and *RMB-2A* in the literature, there has been limited in-depth evaluation of their psychometric properties and appropriateness for empirical

research. Three rounds of testing and refinement using heterogeneous samples from general adult populations result in three revised self-report autism scales, *AQ-9*, *RBQ-2A-R*, and *SQ-7*, that have demonstrated evidence of factorial validity (through exhibiting satisfactory convergent and discriminant validity), factorial invariance (through establishing scale configural and metric invariance across genders), nomological validity (through demonstrating intercorrelations among the three scales and with Big Five traits), as well as parsimony (all scales contain less than ten items). The resulting *AQ-9* now mirrors the current dual diagnostic criteria in the *DSM-5* and thus better aligns academic research and clinical practice. The *RBQ-2A-R* has been refined through CFA for the first time. The *SQ-7* scale consists of two factors, including *technicity* and *structure*, and its content has also been updated to remain applicable. These parsimonious and psychometrically satisfactory measures can provide efficient and consistent measurement of autistic tendencies in the general adult population in future survey research that examine relationships between autism-like symptoms and other constructs.

Future Directions

Future research should further test these instruments using new samples and in different settings. For example, these scales can be administered to the same subjects over time to assess their test-retest reliability. Though these self-report measures are not intended or designed to be diagnostic instruments for ASC, future research can employ both neurotypical and autistic samples to assess their discriminant ability and effectiveness as screening tools.

See Also

- [Comparison of Diagnostic Instruments for ASD](#)
- [Screening Measures](#)

References and Reading

- Allison, C., Auyeung, B., & Baron-Cohen, S. (2012). Toward brief "Red Flags" for autism screening: The Short Autism Spectrum Quotient and the Short Quantitative Checklist for Autism in toddlers in 1,000 cases and 3,000 controls. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(2), 202–212. <https://doi.org/10.1016/j.jaac.2011.11.003>.
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Arlington: American Psychiatric Publishing.
- Austin, E. J. (2005). Personality correlates of the broader autism phenotype as assessed by the Autism Spectrum Quotient (AQ). *Personality and Individual Differences*, 38, 451–460. <https://doi.org/10.1016/j.paid.2004.04.022>.
- Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences*, 6(6), 248–254.
- Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from Asperger syndrome/high-functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders*, 31(1), 5–17.
- Baron-Cohen, S., Richler, J., Bisarya, D., Gurunathan, N., & Wheelwright, S. (2003). The systemizing quotient: An investigation of adults with Asperger syndrome or high-functioning autism, and normal sex differences. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 358, 361–374. <https://doi.org/10.1098/rstb.2002.1206>.
- Barrett, S. L., Uljarević, M., Baker, E. K., Richdale, A. L., Jones, C. R. G., & Leekam, S. R. (2015). The Adult Repetitive Behaviours Questionnaire-2 (RBQ-2A): A self-report measure of restricted and repetitive behaviours. *Journal of Autism and Developmental Disorders*, 45(11), 3680–3692. <https://doi.org/10.1007/s10803-015-2514-6>.
- Bollen, K. A. (1989). *Structural equations with latent variables*. New York: Wiley.
- Cheung, G. W., & Rensvold, R. B. (2002). Evaluating goodness-of-fit indexes for testing measurement invariance. *Structural Equation Modeling*, 9(2), 233–255. https://doi.org/10.1207/S15328007SEM0902_5.
- Dillman, D. A. (2000). *Mail and internet surveys: The tailored design method*. New York: Wiley.
- Frazier, T. W., & Youngstrom, E. A. (2007). Historical increase in the number of factors measured by commercial tests of cognitive ability: Are we overfactoring? *Intelligence*, 35, 169–182.
- Gosling, S. D., & Mason, W. (2015). Internet research in psychology. *Annual Review of Psychology*, 66, 877–902.
- Guadagnoli, E., & Velicer, W. (1988). Relation of sample size to the stability of component patterns. *Psychological Bulletin*, 103, 265–275.

- Hair, J. F., Black, W. C., Babin, B. J., Anderson, R. E., & Tatham, R. L. (1998). *Multivariate Data Analysis*, 5(3), 207–219. Upper Saddle River: Prentice Hall.
- Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*, 6(1), 1–55. <https://doi.org/10.1080/10705519909540118>.
- Hurst, R., Mitchell, J., Kimbrel, N., Kwapil, T., & Nelson-Gray, R. (2007). Examination of the reliability and factor structure of the Autism Spectrum Quotient (AQ) in a non-clinical sample. *Personality and Individual Differences*, 43(7), 1938–1949. <https://doi.org/10.1016/j.paid.2007.06.012>.
- Jackson, S. L. J., Hart, L., Brown, J. T., & Volkmar, F. R. (2018). Brief Report: Self-Reported Academic, Social, and Mental Health Experiences of Post-Secondary Students with Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders*, 48(3), 643–650. <https://doi.org/10.1007/s10803-017-3315-x>.
- Jia, R., Steelman, Z. R., & Jia, H. H. (2019). Psychometric assessments of three self-report, adult autism scales (AQ, RBQ-2A and SQ) in general adult populations. *Journal of Autism and Developmental Disorders*, 49(5), 1949–1965. <https://doi.org/10.1007/s10803-019-03880-x>.
- John, O. P., & Srivastava, S. (1999). The big five trait taxonomy: History, measurement, and theoretical perspectives. In L. A. Pervin & O. P. John (Eds.), *Handbook of personality: Theory and research* (2nd ed., pp. 102–138). New York: Guilford.
- Kline, R. B. (2010). *Principles and practice of structural equation modeling*. New York: The Guildford Press.
- Kloosterman, P., Keefer, K., Kelley, E., Summerfeldt, L., & Parker, J. (2011). Evaluation of the factor structure of the Autism-Spectrum Quotient. *Personality and Individual Differences*, 50(2), 310–314.
- Ling, J., Burton, T. C., Salt, J. L., & Muncer, S. J. (2009). Psychometric analysis of the systemizing quotient (SQ) scale. *British Journal of Psychology*, 100, 539–552. <https://doi.org/10.1348/000712608X368261>.
- Longo, Y., Coyne, I., & Joseph, S. (2018). Development of the short version of the scales of general well-being: The 14-item SGWB. *Personality and Individual Differences*, 124, 31–34.
- MacCallum, R., Roznowski, M., & Necowitz, L. B. (1992). Model modifications in covariance structure analysis: The problem of capitalization on chance. *Psychological Bulletin*, 111(3), 490–504.
- Nunnally, J. C. (1978). Assessment of reliability. In *Psychometric theory* (2nd ed., pp. 245–246). New York: McGraw-Hill.
- Schwartzman, B. C., Wood, J. J., & Kapp, S. K. (2016). *Journal of Autism and Developmental Disorders*, 46, 253–272. <https://doi.org/10.1007/s10803-015-2571-x>.
- Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics* (5th ed.). Boston: Allyn and Bacon.
- van Sonderen, E., Sanderman, R., & Coyne, J. C. (2013). Ineffectiveness of reverse wording of questionnaire items: Let's learn from cows in the rain. *PLoS One*, 8(9). <https://doi.org/10.1371/annotation/af78b324-7b44-4f89-b932-e851fe04a8e5>.
- Veale, J. F., & Williams, M. N. (2015). The psychometric properties of a brief version of the systemizing quotient. *European Journal of Psychological Assessment*, 33(3), 173–180. <https://doi.org/10.1027/1015-5759/a000283>.

Self-Reported Temperament in Children and Adolescent with High Functioning Autism

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Definition

An examination of the use and efficacy of self-reported temperament to index transdiagnostic differences in reactivity and self-regulation in children and adolescents with ASD.

Historical Background

In the early twentieth century, developmental psychologists observed large groups of neurotypical children in order to describe normative sequences of motor and cognitive development. Although clear patterns emerged, what was perhaps more striking was the large amount of variability between children. Shirley (1933) described temperament as the core or nucleus of later personality. Over the ensuing decades, temperament came to be defined as early-appearing, biologically based, relatively stable individual differences in reactivity and self-regulation (Rothbart and Derryberry 1981). Individuals vary along dimensions of Negative Affectivity (e.g., fear, sadness, frustration, discomfort in response to sensory stimulation), Surgency (e.g., activity level,

enjoying novel activities, displaying positive affect, positive excitement), and Effortful Control (e.g., focusing attention, inhibiting reactions; Rothbart 2007). These three dimensions are related to the Big Five personality factors used to describe adult personality (Evans and Rothbart 2007). Individual differences in temperament act as a filter for experienced social and nonsocial environments and as such impact all aspects of a child's emerging personality including cognitions about self, others, and the surrounding physical and social world (Rothbart 2007). Furthermore, a child's temperament affects their perception of the world and, in a transactional way, the world's perception of them (Henderson et al. 2018). For example, parent perceptions of children's temperamental "difficulty" are related to differences in parent-child interactions and children's attachment representations (Seifer et al. 1996). The interplay between temperament, social perception, and social interaction forms the foundation for later personality (Rothbart 2007).

For most of the twentieth century, temperament research and theory were applied to the study of neurotypical children. However, within the last 30 years, there has been increasing attention paid to the assessment, characterization, and meaning of temperament in relation to a variety of neurodevelopmental disorders, including autism spectrum disorders (ASD). Importantly, temperament is a transdiagnostic factor that may be critical for understanding heterogeneity in symptom expression and developmental outcomes for individuals with ASD (Burrows et al. 2016; Chetcuti et al. 2019; Schwartz et al. 2009). Further, being able to quantify these individual differences opens the door to more individualized and tailored approaches to intervention, as certain strategies may be differentially successful depending on the nature of the child's temperament.

Historically, studies examining temperament in children relied on detailed and lengthy observational protocols and/or informant (e.g., parent, teacher) reports, due to concerns that children lacked the introspection and insight to accurately report on their typical behavioral and emotional reaction tendencies. There is no doubt that these approaches are highly informative. For example,

studies of the impact of observed and parent-reported early behavioral inhibition on lifecourse development have been highly influential in the fields of developmental psychology, clinical psychology, and developmental neuroscience (Asendorpf 1990; Perez-Edgar and Fox 2018). Furthermore, youth below age 8 do have significant difficulties reporting on their own abilities, due to their cognitive and language development. However, there is growing consensus that youth self-reports provide an important and unique perspective on their own internal processes (e.g., Achenbach et al. 1987; Burrows et al. 2018; Gotham et al. 2014; Moss et al. 2015). Furthermore, as youth enter adolescence, parents and teachers have less opportunity to observe the individual's behavior, and the youth themselves may have the best insight into their temperament (Achenbach et al. 1987). Importantly, there are only small to moderate associations between informant report and self-reports (De Los Reyes and Kazdin 2005; Lerner et al. 2012), highlighting the unique perspective youth self-reports contribute to the assessment of temperament.

Concerns about the psychometrics of self-reports are mirrored in the developmental disabilities literature due to a misconception that individuals with ASD do not possess sufficient language skills or insight to reflect on their own traits (e.g., Mundy et al. 2007; Tantam 2000). Thus it is critical to carefully examine the reliability and the concurrent and predictive validity of self-report measures of temperament in youth with and without ASD.

Current Knowledge

The spectrum hypothesis (Van Leeuwen et al. 2007) provides a useful model for conceptualizing and studying quantitative and qualitative similarities and differences in self-reported temperament between youth with and without ASD. Specifically, this model provides a framework for investigating whether clinical and non-clinical groups differ in (a) the psychometric properties of measures (i.e., factor structure, reliability), (b) their overall levels of temperament (i.e., mean-level

differences between groups), and (c) the nature and strength of covariation with other constructs of interest.

While the study of self-reported temperament in ASD is still nascent, several investigations have explored how individuals with ASD report on their own temperament and personality. To date, studies of self-reported temperament have found quantitative (e.g., mean-level differences) but not qualitative (e.g., psychometrics, associations with outcomes) differences in self-reported temperament in youth and adults with ASD compared to typically developing (TD) samples. Burrows et al. (2016) reported that self-reports on the Early Adolescent Temperament Questionnaire – Revised (EATQ-R), the primary measure of temperament in children, showed similar psychometric properties in youth with ASD compared to an age- and gender-matched sample of TD youth (Burrows et al. 2016). Specifically, for the broadscale domains of temperament, youth with ASD showed moderate to high levels of reliability (Cronbach's alphas ranging from 0.64 to 0.76), which was similar to TD youth levels of reliability (Cronbach's alphas ranging from 0.66 to 0.83). The two groups' estimates of reliability also overlapped on all domains, indicating that they were not reporting at differing levels of reliability. Similarly, Hesselmark et al. (2015) administered the Revised NEO Personality Inventory (NEO-PI-R) to a group of adults with ASD (with no intellectual disability) and a control group of adults without ASD or other developmental disorders. The ASD and control groups displayed comparable internal consistencies and factor structures on the NEO-PI-R. There is also evidence supporting the validity of self-reports of temperament in youth with ASD. Self-reported temperament showed similar associations with parent-reported internalizing and externalizing problems across groups of youth with and without ASD, indicating high and comparable levels of external validity of self-reported temperament (Burrows et al. 2016; Schwartz et al. 2009).

When examining quantitative differences in self-reported temperament, studies find mean-level differences across many domains of temperament. One consistent finding is that groups of

youth with ASD endorse lower Surgency and higher Negative Affect than their TD peers (Burrows et al. 2016; Samyn et al. 2011; Schwartz et al. 2009). Differences were found in all sub-dimensions of the Surgency factor (Burrows et al. 2016), indicating that youth with ASD show lower enthusiasm for high-intensity activities, have higher levels of fear/worry, and are more shy when meeting new people. Youth with ASD also self-reported higher Negative Affect in all domains, including a higher propensity for frustration, depressive mood, and frustration. There appear to be some subtle differences in Effortful Control between youth with ASD and TD peers, with Samyn et al. (2011) finding overall differences that were driven by group differences in inhibitory control and attentional control. Burrows et al. (2016) by contrast found no group differences in effortful control. These mean-level differences in temperament likely capture meaningful differences within the autism population that may partially account for the large degree of heterogeneity in comorbid psychological functioning in youth with ASD. As a transdiagnostic risk factor for a host of outcomes (e.g., anxiety, depression, ADHD), temperament may be an important early marker for later psychopathology. Quantifying differences in self-reported temperament early in development may help identify individuals who could benefit from prevention or intervention services to help reduce risk for, and impairment associated with, psychiatric comorbidities.

Another issue that has garnered considerable attention is the question of overlap between temperament and symptoms of ASD. For example, social communication symptoms core to ASD may influence an individual's levels of Surgency, with shared behavioral overlap in reduced social interest. While there may be some conceptual overlap, measures of self-reported temperament do not generally correlate with assessments of symptoms of ASD. For example, Schwartz et al. (2009) found no significant correlations between any domains of temperament and multiple measures of autism symptoms in youth with ASD. Self-reported Effortful Control also appears to be more strongly related to symptoms of attention-deficit/hyperactivity disorder (ADHD) than ASD

(Samyn et al. 2011), indicating differentiation between temperament and ASD symptoms. Certain temperamental characteristics increase risk for later behavior problems comparably in TD and ASD youth. For example, in both TD and ASD samples, behavioral inhibition and physiological reactivity to novelty are temperamental precursors to later social anxiety (SA) disorders (Bellini 2006; Clauss and Blackford 2012). In general, within samples of youth with ASD, temperament and ASD traits appear to vary widely and independently and to independently contribute to later outcomes. Understanding the unique contributions of ASD and temperament traits, as well as how they interact over time, may help explain varying developmental trajectories for children with ASD.

Future Directions

Looking forward, we believe the most theoretically and clinically relevant questions have to do with how self-reports of temperament can help explain *within-group* differences in symptom expression, psychiatric comorbidity, and adaptive functioning. Given the relatively low time and cost demands of administering self-reports of temperament, they should be a standard component of research protocols and clinical assessments. It will be particularly important to incorporate such measures into prospective, longitudinal studies to examine how and when in development self-reported temperament affects symptom expression and adaptive functioning. Similarly, it will be important to employ multimethod and multi-informant approaches to assessing temperament to identify points of agreement versus discrepancy. No study to date has investigated agreement and discrepancy between youth self- and parent-reports of temperament or identified factors that predict patterns of agreement. Investigations of this type may help determine when in development self report versus parent reports are most reliable or clinically relevant and how patterns of informant agreement/disagreement affect outcomes.

It will be important to continue designing and refining self-report temperament measures in a

way that minimizes measurement overlap with ASD symptom severity. Given the overlap between some core dimensions of temperament and ASD symptom domains (e.g., social withdrawal and avoidance), it is important to delineate between these constructs both theoretically and quantitatively so as not to artificially inflate estimates of concurrent or longitudinal associations. Some promising new directions in assessing temperament and emotion include the use of experience sampling methodologies to get self-reports of reactions to specific daily contexts, events, and stressors rather than asking participants to provide global judgments of their general reaction tendencies. Such moment-to-moment sampling techniques tend to be less susceptible to global biases and may be more manageable for youth with ASD.

Finally, conceptualizing temperament as the lens through which individuals experience their social and non-social worlds suggests that temperament may play a critical role in designing and selecting intervention approaches optimized to meet the heterogeneous needs of youth with ASD. For example, youth with ASD who are extremely high in Surgency will face significantly different challenges in the classroom than will youth with ASD who are extremely low in Surgency. Self-reported temperament could also hold great promise as a tool to assist with transition planning for youth with ASD, given the accumulating data in other fields that life course outcomes, including career satisfaction and success, are optimized for individuals who experience a good fit between temperament and environmental demands (e.g., Denissen et al. 2018).

References and Reading

- Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, 101(2), 213. <https://doi.org/10.1037/0033-2909.101.2.213>.
- Asendorpf, J. B. (1990). Development of inhibition during childhood: Evidence for situational specificity and a two-factor model. *Developmental Psychology*, 26(5), 721–730.

- Bellini, S. (2006). The development of social anxiety in adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 21(3), 138–145. <https://doi.org/10.1177/10883576060210030201>.
- Burrows, C. A., Usher, L. V., Becker-Haimes, E. M., McMahon, C. M., Mundy, P. C., Jensen-Doss, A., & Henderson, H. A. (2018). Profiles and correlates of parent–child agreement on social anxiety symptoms in youth with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(6), 2023–2037. <https://doi.org/10.1007/s10803-018-3461-9>.
- Burrows, C. A., Usher, L. V., Schwartz, C. B., Mundy, P. C., & Henderson, H. A. (2016). Supporting the spectrum hypothesis: Self-reported temperament in children and adolescents with high functioning autism. *Journal of Autism and Developmental Disorders*, 46(4), 1184–1195. <https://doi.org/10.1007/s10803-015-2653-9>.
- Chetcuti, L., Uljarević, M., & Hudry, K. (2019). Editorial perspective: Furthering research on temperament in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 60(2), 225–228. <https://doi.org/10.1111/jcpp.12957>.
- Clauss, J. A., & Blackford, J. U. (2012). Behavioral inhibition and risk for developing social anxiety disorder: A meta-analytic study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(10), 1066–1075.e1061.
- De Los Reyes, A., & Kazdin, A. E. (2005). Informant discrepancies in the assessment of childhood psychopathology: A critical review, theoretical framework, and recommendations for further study. *Psychological Bulletin*, 131(4), 483–509. <https://doi.org/10.1037/0033-2909.131.4.483>.
- Denissen, J. J., Bleidorn, W., Hennecke, M., Luhmann, M., Orth, U., Specht, J., & Zimmermann, J. (2018). Uncovering the power of personality to shape income. *Psychological Science*, 29(1), 3–13. <https://doi.org/10.1177/0956797617724435>.
- Evans, D., & Rothbart, M. K. (2007). Developing a model for adult temperament. *Journal of Research in Personality*, 41, 868–888. <https://doi.org/10.1016/j.jrp.2006.11.002>.
- Gotham, K., Bishop, S. L., Brunwasser, S., & Lord, C. (2014). Rumination and perceived impairment associated with depressive symptoms in a verbal adolescent–adult ASD sample. *Autism Research*, 7(3), 381–391. <https://doi.org/10.1002/aur.1377>.
- Henderson, H. A., Green, E. S., & Wick, B. L. (2018). The social world of behaviorally inhibited children: A transactional account. In K. Perez-Edgar & N. Fox (Eds.), *Behavioral inhibition: Integrating theory, research, and clinical perspectives* (pp. 135–155). New York: Springer.
- Hesselmark, E., Eriksson, J. M., Westerlund, J., & Bejerot, S. (2015). Autism spectrum disorders and self-reports: Testing validity and reliability using the NEO-PI-R. *Journal of Autism and Developmental Disorders*, 45(5), 1156–1166. <https://doi.org/10.1007/s10803-014-2275-7>.
- Lerner, M. D., Calhoun, C. D., Mikami, A. Y., & De Los Reyes, A. (2012). Understanding parent–child social informant discrepancy in youth with high functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(12), 2680–2692. <https://doi.org/10.1007/s10803-012-1525-9>.
- Moss, P., Howlin, P., Savage, S., Bolton, P., & Rutter, M. (2015). Self and informant reports of mental health difficulties among adults with autism findings from a long-term follow-up study. *Autism*. <https://doi.org/10.1177/1362361315585916>.
- Mundy, P. C., Henderson, H. A., Inge, A. P., & Coman, D. C. (2007). The modifier model of autism and social development in higher functioning children. *Research and Practice for Persons with Severe Disabilities*, 32(2), 124. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2773555/>.
- Perez-Edgar, K., & Fox, N. A. (2018). *Behavioral inhibition: Integrating theory, research, and clinical perspectives*. Cham: Springer International Publishing. <https://doi.org/10.1007/978-3-319-98077-5>.
- Rothbart, M. K. (2007). Temperament, development, and personality. *Current Directions in Psychological Science*, 16(4), 207–212. <https://doi.org/10.1111/j.1467-8721.2007.00505.x>.
- Rothbart, M. K., & Derryberry, D. (1981). Development of individual differences in temperament. In M. E. Lamb & A. Brown (Eds.), *Advances in developmental psychology* (Vol. 1, pp. 37–86). Hillsdale: Erlbaum.
- Samyn, V., Roeyers, H., & Bijttebier, P. (2011). Effortful control in typically developing boys and in boys with ADHD or autism spectrum disorder. *Research in Developmental Disabilities*, 32(2), 483–490. <https://doi.org/10.1016/j.ridd.2010.12.038>.
- Schwartz, C. B., Henderson, H. A., Inge, A. P., Zahka, N. E., Coman, D. C., Kojkowski, N. M., & Mundy, P. C. (2009). Temperament as a predictor of symptomatology and adaptive functioning in adolescents with high-functioning autism. *Journal of Autism and Developmental Disorders*, 39(6), 842–855. <https://doi.org/10.1007/s10803-009-0690-y>.
- Shirley, M. M. (1933). *The first two years a study of 25 babies*. Minneapolis: University of Minnesota Press.
- Seifer, R., Schiller, M., Resnick, S., Riordan, K., & Sameroff, A. J. (1996). Attachment, maternal sensitivity, and infant temperament during the first years of life. *Developmental Psychology*, 32, 12–25. <https://doi.org/10.1037/0012-1649.32.1.12>.
- Tantam, D. (2000). Psychological disorder in adolescents and adults with Asperger syndrome. *Autism*, 4(1), 47–62. <https://doi.org/10.1177/136236130004001004>.
- Van Leeuwen, K. G., Mervielde, I., De Clercq, B. J., & De Fruyt, F. (2007). Extending the spectrum idea: Child personality, parenting and psychopathology. *European Journal of Personality*, 21(1), 63–89. <https://doi.org/10.1002/per.598>.

Self-Stimulatory Behavior

- ▶ Stereotyped Movement Disorder
- ▶ Stereotypic Behavior

Self-Talk

- ▶ Private Speech

Self-Understanding of Academic Competencies in ASD

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Definition

Self-understanding or self-perception is the ability to understand and reflect upon one's competency to perform in the world (Harter 2012). It has been established that young children overestimate their competencies (Bjorklund and Green 1992) and that this overestimation can lead to a sense of self-efficacy (Bandura 1991). As children age, they become more aware of their abilities due to an increase in acquiring cognitive abilities and social experiences that are necessary for an accurate view of the self (Harter 2012).

Research has demonstrated that children and adolescents with Autism Spectrum Disorders (ASD) overestimate their competencies above and beyond what is normative in the general population. Individuals with ASD tend to have inaccurate views of their abilities when asked about their symptomatology (e.g., Johnson et al. 2009); social functioning (e.g., Nicpon et al. 2010); and academic competencies (Furlano and Kelley 2019; Furlano et al. 2015). It is possible that deficits in specific cognitive abilities can

affect how individuals with ASD think of themselves.

Historical Background

Conceptualizations of the self have greatly evolved throughout the years and date back to the ancient Greeks. Historic philosophers (for review see Baumeister 1987) and contemporary researchers (e.g., Harter 2012) have offered different conceptualizations of the self. Of importance is James' (1890) distinction between the concepts of the "I-Self," defined as "the actor or the knower," and the "Me-Self," which refers to "the object of one's knowledge" and is a more conscious process that is related to self-esteem.

Current Knowledge

Self-Understanding Throughout the Lifespan

As previously mentioned, young children hold inaccurate views of the self (Bjorklund and Green 1992). Parenting in the first years of life may influence how children come to view their abilities. For example, caregivers often encourage young children to boast about their performances (e.g., clapping when accomplishing a task), and this acts as a means of nurturing positive views of the self (Butler 2005). Unrealistically positive views of the self continue in the preschool years (Bjorklund and Green 1992), as children at this age think of themselves using simple and observable features and have difficulty creating more general and larger views of the self (Bandura 1991). Preschool-aged children lack some of the cognitive abilities and social experiences that influence self-understanding (Harter 2012). For example, preschoolers lack the ability to engage in social comparison, which allows individuals to compare their abilities to others' (Harter 2012). Lacking the ability to engage in social comparison does not allow children to account for other people's performances, which can cause them to overestimate their competencies (Ruble et al. 1980).

Self-understanding becomes more accurate in middle to late childhood, as children at this age are able to differentiate between abilities across domains (for review see Harter 2012). For example, older children will often understand that they are better at one academic subject compared to another. Children and adolescents also have the ability to engage in abstract thinking and self-understanding, which allows them to describe their abilities as higher-order representations of the self in form of trait labels (Harter 2012). For example, older children and adolescents are able to understand that doing well in academic courses such as math, science, and geography fall under the trait label of “smart” (Harter 2012). Self-understanding continues to be mostly realistic in late adolescence and adulthood; however, there is evidence suggesting that most adults tend to think highly of themselves (e.g., Taylor and Brown 1988). Social psychology research has demonstrated two well-known phenomena related to self-understanding in adults. First, there is the “better-than-average” effect, where individuals evaluate themselves as being more skilled when comparing themselves to an average target (Alicke and Govorun 2005). Individuals also display an optimism bias where they believe that they are less likely than others to experience a negative event and have unrealistic optimism about the future (Alicke and Govorun 2005).

Self-Understanding in ASD

Self-referential behaviors, which can be viewed as developmental prerequisites for self-understanding, are examined in young children using mirror recognition tasks, personal pronoun use, and other-directed pretend play (for review see Lewis et al. 2016). While typically developing (TD) children display these behaviors around 2 years of age (Lewis and Ramsay 2004), self-referential behaviors tend to be absent or delayed in children with ASD (Lewis et al. 2016). Children with ASD have difficulty using personal pronouns (e.g., Lee et al. 1994) and also tend to choose to participate in self-directed play rather than playing with others (Charman and Baron-Cohen 1997).

Research has begun to examine how individuals with ASD understand their competencies. The majority of research has focused on examining how children and adolescents with ASD think of their own symptomatology and social functioning. For example, Johnson et al. (2009) found that children and adolescents with ASD described themselves as having fewer autistic traits and more empathy as compared to parental reports. Research has also demonstrated that individuals with ASD are not aware of the scope of their social deficits and tend to overestimate their social competence compared to parental and teacher reports (Vickerstaff et al. 2007).

Self-Understanding of Academic Competency in ASD

Previous research in the field has focused on examining self-understanding in areas in which individuals with ASD inherently have difficulty. Very few studies to date have examined self-understanding of academic abilities. Basic academic skills (i.e., reading, math, and writing) in children and adolescents with high-functioning ASD (full-scale IQ above 70) tend to be intact and relative to IQ scores (Mayes and Calhoun 2008). Academic difficulties in individuals with ASD mainly lie in areas related to organization, attention, and complex processing, and children and adolescents with ASD have been found to have deficits in listening and reading comprehension (Whitby and Mancil 2009).

Recently, research has demonstrated that individuals with ASD tend to overestimate their academic competencies when they were asked how they performed on basic academic tasks. Furlano et al. (2015) assessed how self-understanding of academic abilities in children and adolescents with ASD, aged 12–18 years, differed from TD peers. In order to test self-understanding, two objective academic tasks, a verbal and a math task, were used (Furlano et al. 2015). Participants were asked to predict how they thought they would perform before the task and how they thought they performed after the task (Furlano et al. 2015). Self-understanding was examined by comparing the participant’s perceived score on the tasks

to their actual score (Furlano et al. 2015). Using the participant's actual score on the task as a criterion measure reduced any bias associated with parental report, which is typically used in self-understanding research, and also allowed participants to self-evaluate their competencies on concrete tasks that they encounter on a daily basis in school (Furlano et al. 2015). The results of the study demonstrated that while there were no differences between the ASD and TD groups' performance on the actual task, meaning that their academic abilities did not differ, the ASD group overestimated their competencies both before and after completing the task compared to the TD group (Furlano et al. 2015).

Furlano and Kelley (2019) replicated and built upon these results by examining whether self-understanding of academic abilities in children with ASD differ compared to TD controls and whether estimations of competency change after providing feedback on the task. Feedback is regularly used in academic settings and has been shown to increase self-efficacy and academic achievement (Pajares and Schunk 2001). The ability to process feedback is important as it helps individuals monitor their strengths and weaknesses, which in turn allows them to focus on the areas that they may have difficulty in (Grainger et al. 2014). Individuals with ASD have been shown to be able to process external and concrete feedback similarly to TD peers; however, they have difficulty in understanding more social and abstract feedback, such as a high five (Ingersoll et al. 2003). As such, Furlano and Kelley (2019) sought to examine if individuals with ASD would be better able to track and update their estimations of their own competencies when they were given feedback on how they performed on each question of an academic task. The results of the study demonstrated that compared to the TD group, the ASD group overestimated their performance on academic tasks when they were asked to predict their performance before and after each task, except when they were given feedback (Furlano and Kelley 2019). The ASD group's estimations of their performance became more accurate when they were given feedback compared to when they were not, which suggests

that they are able to process feedback and track and update their evaluations (Furlano and Kelley 2019). Overall, the results of these two studies (Furlano and Kelley 2019; Furlano et al. 2015) suggest that children and adolescents with ASD overestimate their academic competencies even after completing concrete tasks that they encounter on a daily basis in school, and this overestimation of competency is above and beyond what is normative in the general population. This suggests that individuals with ASD have deficits in the self-understanding of their academic competencies, a domain in which they do not have inherent deficits.

Factors Influencing Self-Understanding in ASD

Harter (2012) suggests that both cognitive abilities and social experiences shape how an individual thinks of their competencies. Although very little empirical research has been conducted on how specific factors relate to self-understanding in ASD, there are some potential theoretical explanations that may account for why individuals with ASD tend to overestimate their academic competencies.

Cognitive abilities. Cognitive abilities play an important role in shaping how individuals think of themselves. The overestimations of competency seen in young children have been attributed to cognitive limitations (Harter 2012). Young children lack certain cognitive abilities that are required to process an accurate view of the self (Harter 2012). As such, it is possible that cognitive ability deficits seen in individuals with ASD may cause them to lack understanding of their own abilities.

Theory of mind. It has been well-established that many individuals with ASD, including those with high-functioning ASD, experience difficulties in theory of mind, which is the ability to attribute mental states to others (Baron-Cohen et al. 2001). Researchers have suggested that the same cognitive mechanisms that underlie theory of mind understanding are same as those that are required for attributing mental states to the self (Frith and Happé 1999). Theory of mind understanding allows for individuals to utilize opinions

that other people have of them in order to create a view of the self (Selman 2003). The inability to understand that others may be critical of you can lead to individuals to have overly positive views of the self (Harter 2012).

Metacognitive monitoring. Metacognitive processes are inherently related to understanding the self. Flavell (1979) broadly defined metacognition as “cognition about cognition” or “thinking about thinking,” and his original model was comprised of two components: metacognitive knowledge and metacognitive monitoring. Metacognitive knowledge is acquired world knowledge that relates to cognitive matters, whereas metacognitive monitoring refers to how an individual manages their knowledge and is used to control cognition to meet a goal (Flavell 1979). It has been suggested that children with ASD have difficulties with more complex academic tasks due to deficits in metacognitive monitoring (Grainger et al. 2014). Deficits in metacognitive abilities would make it difficult for individuals to introspect and activate mental representations about the self (Lyons and Zelazo 2011). More specifically, it would be difficult for individuals with metacognitive monitoring deficits to keep track of their performance during academic tasks, which would affect how they evaluate their achievements.

Executive functioning. Executive functioning is an umbrella term for cognitive processes that control behavior, such as planning, working memory, attention, problem-solving, verbal reasoning, and inhibition (Ozonoff et al. 1991). Research has suggested that individuals with ASD have difficulties with cognitive flexibility (Ozonoff et al. 1991) and inhibitory control (Russell et al. 1996). Both of these processes may affect how individuals think of themselves. Deficits in cognitive flexibility, which is the ability to switch back and forth between concepts, can make it difficult for individuals to conceptualize errors during a task (Ozonoff et al. 1991). Having poor inhibitory control may make it difficult to control responses when answering self-evaluations (Owens et al. 2007).

Episodic memory. Episodic memory is memory for a specific event that happened at a certain

time and place (Tulving 1972). Individuals with ASD have generally been shown to excel at tasks that involve semantic memory (i.e., rote memorization; Bennetto et al. 1996) but display impairments in episodic memory (Lind and Bowler 2009). It has been suggested that there is a dynamic relation between episodic memory and the self where the events that individuals recall shape how they view themselves (Tessler and Nelson 1994). For example, when individuals are asked how they are at math, they generally attempt to recall their performance on a previous math task. Researchers have challenged the link between episodic memory and self-understanding by arguing that individuals have self-schemas that are retrieved without having to reference memories (Sedikides and Spencer 2007). However, these self-schemas typically exist in adults, who have more stable views of the self, compared to children who are constantly updating and building their view of the self (Wheeler et al. 1997). As such, it is possible that deficits in episodic memory may contribute to individuals with ASD inaccurately reporting their abilities.

Social experiences. While it has been suggested that the deficits possessed by individuals with ASD have in self-understanding are due to difficulties in specific cognitive abilities, it is also important to consider the role of social experiences. One socially motivated factor that may influence how an individual reports on their abilities is self-enhancement. The “better-than-average” effect and optimism bias seen in adults are said to be influenced by self-enhancement motives (Taylor and Brown 1988). Individuals may overestimate their competencies when asked about their abilities in order to hide difficulties and display confidence (Harter 2012). While self-enhancement motives may underlie self-understanding in TD individuals, overestimations seen in ASD may not be deliberate and are instead linked to impairments in specific cognitive abilities that are necessary for accurate self-understanding.

Self-Understanding in ADHD

A closely related population that has consistently displayed deficits in self-understanding is

individuals with attention deficit hyperactivity disorder (ADHD; Owens et al. 2007). Research has previously shown that 31% of children with ASD meet diagnostic criteria for ADHD and 24% meet subthreshold clinical ADHD symptoms (Yerys et al. 2009). The *DSM-5* now allows for a co-diagnosis of ADHD and ASD (APA 2013), and it has been suggested that there is a degree of overlapping traits between the ASD and ADHD diagnostic criteria (Mazefsky et al. 2012). Much like children with ASD, children with ADHD display difficulties in social deficits and behavior problems (Pelham and Bender 1982). Children with ADHD tend to have fewer close friendships and are more rejected in the classroom compared to their TD peers (Pelham and Bender 1982). A large body of literature suggests that despite having chronic difficulties in academics, many children and adolescents with ADHD tend to overestimate their academic competencies in comparison with other criteria, such as parental report, teacher report, and other more objective criteria of competency (for review see Owens et al. 2007).

There are several theoretical explanations for why children and adolescents with ADHD tend to overestimate their competencies in a variety of domains. Firstly, it is possible that some of the inherent deficits that exist in both the ASD and ADHD diagnosis contribute to overestimations of competency. For example, both children with ADHD and ASD have difficulty with planning, attention, problem-solving, and inhibition (Ozonoff et al. 1991). Another possible explanation is the self-protective hypothesis, which suggests that when children with ADHD are threatened by a task, they attempt to hide their incompetence by exaggerating their abilities (Owens et al. 2007). This hypothesis suggests that children with ADHD use these overestimations of competency as defense mechanisms that helps to protect their self-esteem (Evangelista et al. 2008). In sum, it is possible that both of these populations are overestimating their competencies due to overlapping cognitive deficits that are inherent in both diagnoses, or it is possible that individuals with ASD have impaired self-understanding due to unique cognitive deficits,

while individuals with ADHD do not have impaired self-understanding and overestimate their abilities to protect their self-esteem.

Future Directions

Future research on self-understanding of academic competencies in individuals with ASD should focus on two main questions. First, researchers should empirically examine factors that underlie this phenomenon. As mentioned, it is possible that individuals with ASD are unaware of their competencies and overestimate their abilities because they lack certain cognitive abilities that are necessary for accurate views of the self (Harter 2012). This is further evidenced by the fact that children and adolescents with ASD overestimate their competencies even when they are asked to reflect on their performance on concrete academic tasks that they experience in school (Furlano and Kelley 2019). Future research should empirically investigate how cognitive abilities, which have been theoretically linked to self-understanding (i.e., theory of mind, metacognition), relate to the overestimations of competency. Different methodology to examine self-understanding of academic competencies should be used. It is possible that more concrete and simple estimations of academic competency (i.e., asking participants how many questions they answered correctly on a task; Furlano and Kelley 2019) may be more related to metacognition, while more abstract broader questions of the self (i.e., asking participants to select a description that fits their abilities; Harter 1985) may be more related to episodic memory.

It is also vital to understand the influence that holding overly positive views of the self can have over time. There exists conflicting evidence on whether having enhanced views of the self is beneficial or detrimental. In childhood, having a positive view of the self is beneficial as it can help children to persevere through difficult tasks regardless of outcome (Taylor and Brown 1988). This is especially helpful in an academic domain, as it promotes children's willingness to try even after failing to succeed (Taylor and Brown 1988).

However, some research suggests that having overly positive views of the self can have negative implications in clinical populations. For example, overestimations of competency have been found to lead to conduct problems in children with ADHD (McQuade et al. 2011). Children with ADHD tend to react negatively when they are given realistic feedback that does not correspond to how they view themselves (McQuade et al. 2011). Research has also shown that individuals with Obsessive Compulsive Disorder who have greater insight into their symptoms show more readiness for treatment (Storch et al. 2008). It is therefore possible that having a positive view of the self in terms of academic competencies may be beneficial in children with ASD, as it can help them continue to be motivated on academic tasks even if they have not previously done well on a similar task. However, this overestimation of academic competency may not be helpful past childhood. For example, if adolescents and adults are not aware of their strengths and weaknesses, it may cause them to make poor choices about their education and future career path. Overall, future research should continue to examine self-understanding in ASD and should examine the consequences of overestimating academic competencies throughout the lifespan using longitudinal studies.

See Also

- Academic Skills
- Self-Other Distinction and Social Cognition in ASD
- Self-Recognition and Self-Referential Behavior

References and Reading

Alicke, M. D., & Govorun, O. (2005). The better-than-average effect. In M. D. Alicke, D. A. Dunning, & J. I. Krueger (Eds.), *The self in social judgment* (pp. 85–106). New York: Psychology Press.

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- Bandura, A. (1991). Social cognitive theory of self-regulation. *Organizational Behavior and Human Decision Processes*, 50, 248–287. [https://doi.org/10.1016/0749-5978\(91\)90022-L](https://doi.org/10.1016/0749-5978(91)90022-L).
- Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The ‘reading the mind in the eyes’ test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry*, 42, 241–252. <https://doi.org/10.1111/1469-7610.00715>.
- Baumeister, R. F. (1987). How the self became a problem: A psychological review of historical research. *Journal of Personality and Social Psychology*, 52(1), 163. <https://doi.org/10.1037/0022-3514.52.1.163>.
- Bennetto, L., Pennington, B. F., & Rogers, S. J. (1996). Intact and impaired memory functions in autism. *Child Development*, 67(4), 1816–1835. <https://doi.org/10.1111/j.1467-8624.1996.tb01830.x>.
- Bjorklund, D. F., & Green, B. L. (1992). The adaptive nature of cognitive immaturity. *American Psychologist*, 47, 46–54. <https://doi.org/10.1037/0003-066X.47.1.46>.
- Butler, J. (2005). *Giving an account of oneself*. New York: Fordham University Press.
- Charman, T., & Baron-Cohen, S. (1997). Brief report: Prompted pretend play in autism. *Journal of Autism and Developmental Disorders*, 27, 325–332. <https://doi.org/10.1023/A:1025806616149>.
- Evangelista, N. M., Owens, J. S., Golden, C. M., & Pelham, W. E. (2008). The positive illusory bias: Do inflated self-perceptions in children with ADHD generalize to perceptions of others? *Journal of Abnormal Child Psychology*, 36, 779–791. <https://doi.org/10.1007/s10802-007-9210-8>.
- Flavell, J. H. (1979). Metacognition and cognitive monitoring: A new area of cognitive–developmental inquiry. *American Psychologist*, 34(10), 906. Retrieved from <https://psycnet.apa.org/fulltext/1980-09388-001.pdf>
- Frith, U., & Happé, F. (1999). Theory of mind and self-consciousness: What is it like to be autistic? *Mind & Language*, 14, 1–22. <https://doi.org/10.1111/1468-0017.00100>.
- Furlano, R., & Kelley, E. A. (2019). Do children with autism spectrum disorder understand their academic competencies? *Journal of Autism and Developmental Disorders*, 1–13. <https://doi.org/10.1007/s10803-019-03988-0>.
- Furlano, R., Kelley, E., Hall, L., & Wilson, D. (2015). Self-perception of competencies in adolescents with autism spectrum disorders. *Autism Research*, 8, 761–770. <https://doi.org/10.1002/aur.1491>.
- Grainger, C., Williams, D. M., & Lind, S. E. (2014). Metacognition, metamemory, and mindreading in high-functioning adults with autism spectrum disorder.

- Journal of Abnormal Psychology*, 123(3), 650. <https://doi.org/10.1037/a0036531>.
- Harter, S. (1985). *Manual for the Self-Perception Profile for Children*. Unpublished Manuscript. University of Denver.
- Harter, S. (2012). *The construction of the self: A developmental perspective*. New York: Guilford Press.
- Hoza, B., Waschbusch, D. A., Pelham, W. E., Molina, B. S. G., & Milich, R. (2000). Attention deficit/hyperactivity disorder and control boys' responses to social success and failure. *Child Development*, 71, 432–446. <https://doi.org/10.1111/1467-8624.00155>.
- Ingersoll, B., Schreibman, L., & Tran, Q. H. (2003). Effect of sensory feedback on immediate object imitation in children with autism. *Journal of Autism and Developmental Disorders*, 33(6), 673–683. <https://doi.org/10.1023/B:JADD.0000006003.26667.f8>.
- James, W. (1890). The consciousness of self. In James, W., Burkhardt, F., Bowers, F., & Skruskelis, I. K. (Eds.) *The principles of psychology* (Vol. 1, No. 2). London: Macmillan.
- Johnson, S. A., Filliter, J. H., & Murphy, R. R. (2009). Discrepancies between self- and parent-perceptions of autistic traits and empathy in high functioning children and adolescents on the autism spectrum. *Journal of Developmental Disorders*, 39, 1706–1714. <https://doi.org/10.1007/s10803-009-0809-1>.
- Lee, A., Hobson, R. P., & Chiat, S. (1994). I, you, me, and autism: An experimental study. *Journal of Autism and Developmental Disorders*, 24, 155–176. <https://doi.org/10.1007/BF02172094>.
- Lewis, M., & Ramsay, D. (2004). Development of self-recognition, personal pronoun use, and pretend play during the 2nd year. *Child Development*, 75(6), 1821–1831. <https://doi.org/10.1111/j.1467-8624.2004.00819.x>.
- Lewis, M., Stoicescu, L., Matthews, T., & Seshadri, K. (2016). Self-recognition and self-referential behavior. In F. Volkmar (Ed.), *Encyclopedia of autism spectrum disorders*. New York: Springer. <https://doi.org/10.1007/978-1-4614-6435-8>.
- Lind, S. E., & Bowler, D. M. (2009). Recognition memory, self-other source memory, and theory-of-mind in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 39(9), 1231–1239. <https://doi.org/10.1007/s10803-009-0735-2>.
- Lyons, K. E., & Zelazo, P. D. (2011). Monitoring, meta-cognition, and executive function: Elucidating the role of self-reflection in the development of self regulation. *Advances in Child Development and Behavior*, 40, 379–412. <https://doi.org/10.1016/B978-0-12-386491-8.00010-4>.
- Mayes, S. D., & Calhoun, S. L. (2008). WISC-IV and WIAT-II profiles in children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 38(3), 428–439. <https://doi.org/10.1007/s10803-007-0410-4>.
- Mazefsky, C. A., Oswald, D. P., Day, T. N., Eack, S. M., Minshew, N. J., & Lainhart, J. E. (2012). ASD, a psychiatric disorder, or both? Psychiatric diagnoses in adolescents with high-functioning ASD. *Journal of Clinical Child & Adolescent Psychology*, 41(4), 516–523. <https://doi.org/10.1080/15374416.2012.686102>.
- McQuade, J. D., Tomb, M., Hoza, B., Waschbusch, D. A., Hurt, E. A., & Vaughn, A. J. (2011). Cognitive deficits and positively biased self-perceptions in children with ADHD. *Journal of Abnormal Child Psychology*, 39, 307–319. <https://doi.org/10.1007/s10802-010-9453-7>.
- Nicpon, M. F., Doobay, A. F., & Assouline, S. G. (2010). Parent, teacher, and self perceptions of psychosocial functioning in intellectually gifted children and adolescents with Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders*, 40, 1028–1038. <https://doi.org/10.1007/s10803-010-0952-8>.
- Owens, J. S., Goldfine, M. E., Evangelista, N. M., Hoza, B., & Kaiser, N. M. (2007). A critical review of self-perceptions and the positive illusory bias in children with ADHD. *Clinical Child and Family Psychology Review*, 10, 335–351. <https://doi.org/10.1007/s10567-007-0027-3>.
- Ozonoff, S., Pennington, B. F., & Rogers, S. J. (1991). Executive function deficits in high-functioning autistic children: Relationship to theory of mind. *Journal of Child Psychology and Psychiatry*, 32, 1081–1105. <https://doi.org/10.1111/j.1469-7610.1991.tb00351.x>.
- Pajares, F., & Schunk, D. H. (2001). Self-beliefs and school success: Self-efficacy, self concept, and school achievement. In R. J. Riding & S. G. Rayner (Eds.), *International perspectives on individual differences: Perception* (pp. 239–266). London, UK: Ablex Publishing.
- Pelham, W. E., & Bender, M. E. (1982). Peer relationships in hyperactive children: Description and treatment. *Advances in Learning & Behavioral Disabilities*, 1, 365–436. Retrieved from <https://psycnet.apa.org/record/1983-06085-001>
- Ruble, D. N., Boggiano, A. K., Feldman, N. S., & Loeb, J. H. (1980). Developmental analysis of the role of social comparison in self-evaluation. *Developmental Psychology*, 16(2), 105. <https://doi.org/10.1037/0012-1649.16.2.105>.
- Ruffman, T. (1999). Children's understanding of logical inconsistency. *Child Development*, 70(4), 872–886. <https://doi.org/10.1111/1467-8624.00063>.
- Russell, J., Jarrold, C., & Henry, L. (1996). Working memory in children with autism and with moderate learning difficulties. *Journal of Child Psychology and Psychiatry*, 37, 673–686. <https://doi.org/10.1111/j.1469-7610.1996.tb01459.x>.
- Sedikides, C., & Spencer, S. (2007). *The self: Frontiers in social psychology*. New York: Psychology Press.

- Selman, R. L. (2003). *Promotion of social awareness: Powerful lessons for the partnership of developmental theory*. New York: Russell Sage Foundation.
- Storch, E. A., Merlo, L. J., Larson, M. J., Marien, W. E., Geffken, G. R., Jacob, M. L., et al. (2008). Clinical features associated with treatment-resistant pediatric obsessive compulsive disorder. *Comprehensive Psychiatry*, 49(1), 35–42. <https://doi.org/10.1016/j.comppsy.2007.06.009>.
- Taylor, S. E., & Brown, J. D. (1988). Illusions and well-being: A social psychological perspective on mental health. *Psychological Bulletin*, 103, 193–210. <https://doi.org/10.1037/0033-2909.103.2.193>.
- Tessler, M., & Nelson, K. (1994). Making memories: The influence of joint encoding on later recall by young children. *Consciousness and Cognition*, 3(3), 307–326. <https://doi.org/10.1006/ccog.1994.1018>.
- Tulving, E. (1972). Episodic and semantic memory. *Organization of Memory*, 1, 381–403. Retrieved from http://alumni.media.mit.edu/~jorkin/generals/papers/Tulving_memory.pdf
- Vickerstaff, S., Heriot, S., Wong, M., Lopes, A., & Dossetor, D. (2007). Intellectual ability, self-perceived social competence, and depressive symptomatology in children with high-functioning autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(9), 1647–1664. <https://doi.org/10.1007/s10803-006-0292-x>.
- Wheeler, M. A., Stuss, D. T., & Tulving, E. (1997). Toward a theory of episodic memory: The frontal lobes and autonoetic consciousness. *Psychological Bulletin*, 121, 331. <https://doi.org/10.1037/0033-2909.121.3.331>.
- Whitby, P. J. S., & Mancil, G. R. (2009). Academic achievement profiles of children with high functioning autism and Asperger syndrome: A review of the literature. *Education and Training in Developmental Disabilities*, 551–560. Retrieved from http://daddcec.org/portals/0/cec/autism_disabilities/research/publications/education_training_development_disabilities/2009v44_journals/etdd_200912v44n4p551-560_academic_achievement_profiles_children_with_high_functioning.pdf
- Yerys, B. E., Wallace, G. L., Sokoloff, J. L., Shook, D. A., James, J. D., & Kenworthy, L. (2009). Attention deficit/hyperactivity disorder symptoms moderate cognition and behavior in children with autism spectrum disorders. *Autism Research*, 2, 322–333. <https://doi.org/10.1002/aur.103>.

Semantic Fluency

► Verbal Fluency

Semantic Memory

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Definition

The concept of semantic memory, along with episodic memory, forms part of the declarative memory system, the operation of which is open to conscious awareness. It encompasses an individual's stock of general knowledge. Although traditionally thought of as storing information in an abstract, propositional manner that transcends the perceptual features of the information it represents, more recent formulations acknowledge some enduring representation of the original sensory or perceptual information. Semantic memory is organized in a way that permits flexible goal-directed retrieval and utilization of the stored information, and its operation is accompanied by a particular state of conscious awareness that does not involve the self reexperiencing the past. In this respect, it contrasts both with episodic memory, in which conscious awareness involves the self, and with procedural memory, which is not accessible to conscious awareness.

Historical Background

Most authorities now agree that human memory can be divided into “non-declarative” systems, such as sensory or procedural memory, whose operation is relatively inaccessible to conscious awareness, and “declarative” systems, which represent the conscious act of “remembering something.” A major distinction within the declarative memory system is that between semantic and episodic memory (see Gardiner 2008 for review). The term “semantic memory” emerged from the literature on experimental, laboratory-based studies of memory in the 1960s and 1970s and was systematically elaborated in a chapter by Tulving

(1972). Tulving has become the scientist most famously associated with the term “semantic memory” and its relation to other memory systems, in particular episodic and procedural memory (see Tulving 1985 for review). Although dividing memory along these lines is not universally accepted (see Squire 1987 and Jacoby 1988 for alternative accounts), it is largely to the work of Tulving that we owe the idea of a separate, semantic memory system that handles and organizes our store of factual general knowledge about the world.

Tulving (1972) defined semantic memory as “. . . the memory necessary for the use of language. It is a mental thesaurus, organized knowledge a person possesses about words and other verbal symbols, their meaning and referents, about relations among them and about rules, formulas, and algorithms for the manipulation of these symbols, concepts and relations” (p. 386). Tulving (1985) contrasts this semantic memory system with the episodic memory system, which stores events as situated in time, rich in perceptual and contextual detail and related to a sense of the experiencing self. In a later account, Tulving (2001) refers to semantic memory as a store of “timeless facts” that are accompanied by *noetic conscious awareness*. This awareness is not tied to a particular time or place and does not involve a sense of the self as knower even though it may contain propositions about the self (I am a psychologist, I have a cat, etc.). Episodic memory, by contrast, involves “mental time travel” where the self travels back in time to recreate the temporal and spatial context of the recollected episode and is accompanied by *autonoetic conscious awareness*, which is associated with a specific place and time and involves a sense of self as the knower of the recollected information.

The reasoning that led Tulving (1972) to argue for separate semantic and episodic memory systems rests on a number of observations and distinctions that are still relevant to attempts to characterize the structure of the human information processing system and which are also relevant to an understanding of autism spectrum disorder (ASD). A key distinction between

episodic and semantic memory lay in the way information was stored in the two systems. Whereas Tulving argued that episodic information is stored in terms of the perceptual attributes of items information and the relation of directly experienced items to each other in space and time, information in semantic memory is more abstract and propositional in nature. Although a representation in semantic memory may contain elements of the original, perceptual features of the information on which the memory is based (e.g., registering that water is a liquid), the way this information is represented is biased more toward the logical implications of the proposition “water is a liquid” than to any perceptual experience of wetness or fluidity that might be encountered in a direct experience of drinking a glass of water.

In the four decades since the term became current in discussions of memory, research has been aimed at further characterizing the structure and function of the semantic memory system. Some investigators have attempted to confirm or challenge the semantic/episodic distinction at both a behavioral and a neural level. Of more relevance to the study of ASD, however, is research that has concentrated on how information in the semantic memory system is organized.

It is of considerable advantage to an organism if its store of knowledge (facts, propositions, etc.) can be organized and linked together in various ways. Such organization means that each stimulus, object, or event is not seen in isolation but that groups of stimuli can be treated similarly, thus achieving savings in information processing load. One form such organization can take is for objects and events to be grouped into categories or concepts, which in turn can be further grouped hierarchically. For example, HAT, COAT, SHOE, and SCARF can all be thought of as “items of clothing,” just as CAR, BICYCLE, BOAT, and TRAIN can be grouped under the heading “modes of transport.” These two superordinate categories further contrast with “animals” and “fruit” in being artifacts rather than natural kinds. Words can also be related associatively, for example, by tending to occur together. So BED, PILLOW, BLANKET, SNOOZE, and NIGHT can be

thought of as “going together” because they are all strongly associated with the word “SLEEP.”

Attempts to formulate theories of concept formation and representation have come from philosophy, neurology, experimental psychology, and neuroscience (see Murphy 2002, Chap. 1 for review). *Classical concept theory* proposes that all higher-order concepts can be fully defined by feature combinations that are individually necessary and jointly sufficient to define an exemplar of that concept. Accordingly, we might say that birds are warm-blooded, have feathers and wings, lay eggs, and can fly. However, classical concept theory encounters a number of difficulties, both at a logical and a psychological/neuropsychological level. For example, penguins are birds, yet they do not fly, raising the question of what constitute necessary defining features of the category “bird.” Also, researchers have grappled with the question of how an abstract, multidimensional concept such as “bird” can be represented in the brain, given that it derives from an aggregate of different perceptual features from different sensory modalities (appearance, sound, feel, etc.). In an attempt to overcome some of these objections, *prototype theory* argues that concepts comprise averaged-out representations of a range of instances of members of the concept or category, weighted by their frequency of occurrence in the real world. Thus, a prototype of a bird might add “can fly” to the properties listed earlier but not posit this as a necessary condition for being a bird (e.g., think of ostriches or penguins). More recently, theorists such as Barsalou (2008) have argued that the organization of representations of the world is grounded in our physical, embodied experience of that world and that this organization will ultimately reflect the features of the situated, embodied groundedness that engendered them. For example, Marques (2006) asked participants to verify statements such as (1) *a dog can bark*, (2) *a bee can buzz*, and (3) *a horse has hooves*. They found that verification time increased when a statement followed 1 in a different modality (statements 1 and 3 involve a switch from audition to vision) than when it followed 1 in the same

modality (statements 1 and 2 both involve audition). They concluded that this demonstrated the lasting effects of perceptual modality on conceptual reasoning.

Although much of the discussion around prototype and grounded theories of conceptual representation does not explicitly consider how concepts are actually represented in the brain, the question of how such representations are implemented is a growing area of research. Of particular relevance to ASD is research into the extent to which representations in semantic memory are amodal and completely abstracted from their initial sensory and perceptual origins or unimodal, tied to modality-specific representational systems or multimodal, retaining features of all modalities involved in the laying down of the representations (see Gainotti 2006 for review).

Some researchers such as Lambon Ralph and Patterson (2008) argue for there being an “amodal semantic hub” at which categories are represented across all modalities that contributed to the generation of that category. Other researchers, such as Damasio (1989) argue that the multimodal, multisensory activations generated when an object is encountered are coded hierarchically in the nervous system in a way that generates higher-order representations that continue to retain some of the original sensory/perceptual information. What Damasio terms “higher-order convergence zones” become more or less hierarchically organized in ways that reflect the organism’s experience of objects in the real world.

From this brief overview, we can see that the original notion of semantic memory, that of a consciously accessible, organized set of pieces of information about the world that is separate from but interacts with our recollection of the personally experienced past, is still current, if not entirely uncontroversial. The information it contains is, to a greater or lesser extent, abstracted from the sensory information that initially gave rise to it, and when it is retrieved, it is accompanied by a state of conscious awareness that does not involve the self in reliving past episodes.

Current Knowledge

One of the most enduring discoveries in the science of autism spectrum disorder (ASD) is the observation by Hermelin and O'Connor (1970) that the disorder consists of a difficulty with the meaningful encoding of stimuli. That observation was made at a time when the concept of semantic memory was beginning to emerge in the mainstream experimental psychology literature. In the intervening four decades, much more has been learned both about the organization and functioning of human memory in general and about the patterning of other psychological functions in ASD, yet the core of Hermelin and O'Connor's observation – that being on the autism spectrum implies a difficulty in keeping an abstract, verbally mediated, systematically organized store of knowledge that can be drawn upon to guide action – continues to have considerable validity.

The three aspects of semantic memory outlined in the *Historical Background* section each have their implications for the operation of the system in individuals with ASD. There is now a considerable weight of evidence to support the idea that individuals from all parts of the autism spectrum experience some difficulties with the structural organization of the semantic memory system. Although early studies of categorization abilities in children with intellectual disability (ID) and ASD revealed intact basic and superordinate categorization skills, electrophysiological signatures of superordinate categories were found to be atypical. Moreover, whereas children with ASD and ID have been shown to be unimpaired in sorting a set of shapes under appropriate shape headings, they were impaired in sorting a set of objects into the categories of trees, beds, human figures, animals, tools, and vehicles. Moreover, their ASD group was impaired on a class-inclusion task where they had to answer questions like “are all the circles red?” (See Bowler 2007, Chap. 7 for a more detailed review of this work).

A further set of findings have shown that memory in ASD is less influenced by meaningful relations among studied items. This is especially true

in tasks such as free recall, where the test procedure provides few clues to the studied material. One measure of sensitivity to the semantic structure of studied material is the extent to which words from the same category are clustered together at recall even though they were randomly distributed through the study list. The overwhelming majority of studies of children and adults with ASD show diminished categorical clustering of items in free recall. Children with ASD and significant intellectual disability (ID) also make less use of sentence structure and categorical relations to aid their free recall of lists of words. For example, children without autism show better recall of the sentence *where is the ship* than the random string *wall long cake sand*. Recall by children with ASD, matched with the non-ASD children on digit span, has shown no difference between the two types of material. Studies have also shown that children without ASD recall more words from a list where each word is the name of an animal than from lists where each word came from a different category. Children with ASD matched with the typical children on verbal mental age showed no advantage for the animals list, an observation that also holds for higher-functioning adults with ASD. This difficulty in exploiting categorical semantic relatedness also extends to the recall of incidentally encoded context. When high-functioning adults with ASD are asked to study words inside a rectangle when there are also conceptually related or unrelated words outside the rectangle, they do not show enhanced free recall of words that are studied in conjunction with semantically related words. Comparison participants without ASD, by contrast, show enhanced recall when both words (inside or outside the rectangle) are related (see Bowler 2007, Chap. 7 for a detailed review of these studies).

Difficulties with exploiting structural semantic relations to aid recall appear at first sight to be contradicted by demonstrations of intact illusory memories in individuals with ASD (see Bowler et al. 2011 for review). These studies typically ask participants to study lists of words that are strong associates of a non-studied word (such as the

BED, PILLOW, BLANKET... example given earlier) and then test recognition of studied words and non-studied words including the strong associate SLEEP. However, as Bowler et al. (2011) observe, tasks requiring the exploitation of hierarchical semantic relations differ in their computational demands from those that require a grasp of associations between pairs of items. Bowler et al. draw a parallel between the kind of reasoning involved in illusory memories and the processing of what Halford (1992) has termed *binary relations*, in which two items of information must be processed simultaneously. Exploiting hierarchical semantic relations to aid recall involves *ternary relations*, in which successive studied words (say APPLE, HORSE, PEAR) need not only to be compared to each other but also to the higher-order categories under which they are subsumed.

One reason why relational complexity may be difficult for individuals with ASD is that it may result from a bias or preference for item specific rather than relational processing. When adults with ASD and typical participants are asked to recall lists of items made up of categories, each of which is represented by between 2 and 16 exemplars, ASD participants' recall of the smaller categories is impaired, suggesting that they are less likely to notice the links between items from categories represented by small numbers of items widely spaced in the study list (imagine studying a list of 42 words and encountering *apple* among the first few words and *pear* among the last few). Orienting participants to relations among items by sorting them into categories at study aided recall in both groups of participants but was less effective for the ASD group. Orienting them to specific features of items by asking participants to rate each item's pleasantness was more effective in enhancing recall in the ASD group. These findings suggest a bias in ASD toward processing individual item features and away from relations among items. This in itself will militate against the kinds of complex relational strategies needed to recruit hierarchical category structure in the aid of recall (see Bowler et al. 2011 for further elaboration).

The explanation of difficulties in recruiting semantic relatedness to aid recall in terms of

processing complexity helps to flesh out the observation by Minshew and colleagues (2001) that individuals with ASD experience greater difficulties with complex but not simple memory tasks. But both accounts beg the question of how and why such difficulties with complexity might come about. An answer to this question may well be provided by a reconsideration of how Tulving (1972) originally described the laying down of semantic memory traces. He emphasized the abstract, propositional nature of semantic traces, which were not closely tied to the original sensory-perceptual information that gave rise to them. We have already seen that more recent theorists have taken different perspectives on the extent to which semantic memories are distanced from their sensory, perceptual, and embodied origins, but all acknowledge some level of abstraction from the sensory data (see Gainotti 2011). The bias seen in ASD toward item-specific processing is consistent with the view that the principles governing the organization of semantic memory in this population may well reflect a greater contribution from sensory and perceptual data.

One theory of conceptual representation that has been researched in the context of ASD is prototype theory, which, as we have seen, proposes that abstract representations of semantic categories are built up as averages of the features of the examples of members of that category. The balance of existing experimental evidence suggests that at least a subset of individuals on the autism spectrum have some ability to abstract perceptual prototypes (Molesworth et al. 2005) but can take longer to do it. This last finding can be explained in terms of the *enhanced perceptual functioning* (EPS, Mottron et al. 2006) account of ASD, which argues that individuals with ASD, although as capable as typical individuals of generating global, abstract representations of stimuli, continue to hold lower-level perceptual representations of learned material. The presence of these lower-level representations is argued to affect both the speed and extent to which prototypical representations are abstracted. The EPS may also help to account for why individuals with ASD appear to organize stored information in a

different way from typically developed individuals. Bowler et al. (2008) asked participants with and without ASD to recall a list of 16 unrelated words presented over 15 trials, with the words presented in a different order on each trial. One of the principal aims of the study was to ascertain the amount of *subjective organization* engaged in by participants, i.e., the extent to which the structure of each participant's recall became similar across trials and, as a consequence, differed from the order of the presented words. An index was also calculated of the degree to which subjective organization converged among individuals across trials. Bowler et al. (2008) found that whereas their ASD participants showed only slightly diminished subjective organization, the extent to which this organization converged across individuals over trials was significantly less than in the comparison participants. This suggests that as a group, the ASD participants were organizing the studied material differently from each other, whereas the typical group was showing increasing similarity in their organization of the material. Taken together with the EPS theory and with the observations on diminished clustering in recall of categorized lists mentioned earlier on, this finding suggests that the way that ASD individuals organize items in memory may be more diverse and not as biased toward abstract, semantic aspects as in typical individuals. Memory organization in ASD might rely to a greater extent on perceptual features of studied material, such as rhyme, number of syllables, or form of the word in the case of written studied material. Such conjectures have yet to be tested empirically.

A final, important aspect of semantic memory system centers on the kind of conscious awareness that accompanies its operation. Both familiarity and the noetic conscious awareness or experience of "timeless facts" referred to by Tulving can be measured in recognition memory experiments by asking participants to make an additional "remember/know" judgment once they have identified a test word as having been encountered in the study phase. Participants are told to make "remember" responses when they have the experience of reliving the studied episode (reflecting autonoetic consciousness) and "know" responses when they

have a strong sense of having encountered the word previously (reflecting noetic awareness) but without distinct recollection of the episode. A large number of studies have now demonstrated diminished episodic "remembering" accompanied by undiminished semantic "knowing" in high-functioning adults with ASD. Moreover, a series of manipulations that are known to enhance or diminish the level of "remember" or "know" responses in the typical population yielded similar effects in an ASD group, suggesting that processes underlying these responses, although quantitatively different, are qualitatively similar in the two groups. These findings suggest that individuals with ASD utilize their semantic memory system to compensate for episodic difficulties, an observation that is borne out by studies of autobiographical memory, where individuals with ASD characteristically report autobiographical events in terms that suggest items of factual knowledge rather than personally experienced, relived events (see Bowler et al. 2011 for review). However, the research reviewed earlier, showing enhanced perceptual involvement in semantic memory representations, suggests that the semantic memories of ASD and non-ASD individuals may be qualitatively similar only up to a point.

Insofar as semantic memory consists of a stock of knowledge about the world, individuals with ASD without accompanying intellectual impairment appear to have an intact store of factual material. Where they appear to differ is in the way that this information is recorded and manipulated. Semantic memories in ASD appear to be tagged with more of the sensory and perceptual information from the original source of the memories, which seems to promote a more item specific rather than a relational approach to processing the material. This has important consequences in any task that requires the flexible deployment of semantic relations for its successful resolution.

Future Directions

Future research needs to address the following points:

1. Establish the basis on which individuals with ASD organize information in semantic memory.
2. Determine the brain bases of such organization both in terms of processing at encoding and retrieval and in terms of storage.
3. Work out the implications of atypical encoding, storage, and retrieval on the way semantic information is utilized in adaptive functioning.
4. Investigate ways to overcome any negative consequences of atypical operation of the semantic memory system.

See Also

- [Declarative Memory](#)
- [Enhanced Perceptual Functioning](#)
- [Episodic Memory](#)
- [Explicit Memory](#)
- [Free Recall](#)
- [Memory](#)
- [Memory Assessment](#)
- [Memory Development](#)
- [Procedural Memory](#)
- [Recognition](#)

References and Reading

- Barsalou, L. W. (2008). Grounded cognition. *Annual Review of Psychology*, 59, 617–645.
- Bowler, D. M. (2007). *Autism spectrum disorders: Psychological theory and research*. Chichester: Wiley.
- Bowler, D. M., Gaigg, S. B., & Gardiner, J. M. (2008). Subjective organisation in the free recall learning of adults with Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 38, 104–113.
- Bowler, D. M., Gaigg, S. B., & Lind, S. E. (2011). Memory and autism: Binding self and brain. In I. Roth & P. Rezaie (Eds.), *Researching the autism spectrum: Contemporary perspectives* (pp. 316–346). Cambridge: Cambridge University Press.
- Damasio, A. R. (1989). Time-locked multiregional retroactivation: A systems-level proposal for the neural substrates of recall and recognition. *Cognition*, 33, 25–62.
- Gainotti, G. (2006). Anatomical, functional and cognitive determinants of semantic memory disorders. *Neuroscience and Biobehavioral Reviews*, 30, 577–594.
- Gainotti, G. (2011). The organization and dissolution of semantic-conceptual knowledge: Is the 'amodal hub' the only plausible model? *Brain and Cognition*, 75, 299–309.
- Gardiner, J. M. (2008). Concepts and theories of memory. In J. Boucher & D. Bowler (Eds.), *Memory in autism: theory and evidence*. Cambridge, UK: Cambridge University Press.
- Halford, G. S. (1992). *Children's understanding: The development of mental models*. Hillsdale: Lawrence Erlbaum Associates.
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. Oxford: Pergamon Press.
- Jacoby, L. L. (1988). Memory observed and memory unobserved. In U. Neisser & E. Winograd (Eds.), *Remembering reconsidered: Ecological and traditional approaches to the study of memory* (pp. 145–177). New York: Cambridge University Press.
- Lambon Ralph, M. A., & Patterson, K. (2008). Generalization and differentiation in semantic memory: Insights from semantic dementia. *Annals of the New York Academy of Sciences*, 1124, 61–76.
- Marques, J. F. (2006). Specialization and semantic organization: Evidence for multiple semantics linked to sensory modalities. *Memory and Cognition*, 34, 60–67.
- Minshew, N. J., Johnson, C., & Luna, B. (2001). The cognitive and neural basis of autism: A disorder of complex information processing and dysfunction of neocortical systems. *International Review of Research in Mental Retardation*, 23, 111–138.
- Molesworth, C. J., Bowler, D. M., & Hampton, J. A. (2005). Memory for modal prototypes in high-functioning children with autism. *Journal of Child Psychology and Psychiatry*, 46, 661–672.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36, 27–43.
- Murphy, G. (2002). *The big book of concepts*. Cambridge, MA: MIT Press.
- Squire, L. R. (1987). *Memory and brain*. New York: Oxford University Press.
- Tulving, E. (1972). Episodic and semantic memory. In E. Tulving & W. Donaldson (Eds.), *Organization of memory* (pp. 381–403). New York: Academic Press.
- Tulving, E. (1985). How many memory systems are there? *American Psychologist*, 40, 385–398.
- Tulving, E. (2001). Episodic memory and common sense: How far apart? *Philosophical Transactions of the Royal Society, B*, 356, 1505–1515.

Semantic Pragmatic Deficit Disorder

- [Autism: Social Communication Disorder](#)
- [Semantic Pragmatic Disorder](#)

Semantic Pragmatic Deficit Syndrome

- [Autism: Social Communication Disorder](#)
- [Semantic Pragmatic Disorder](#)

Semantic Pragmatic Disorder

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Synonyms

[Pragmatic communication disorder](#); [Pragmatic language impairment](#); [Semantic pragmatic deficit disorder](#); [Semantic pragmatic deficit syndrome](#); [Semantic pragmatic language disorder](#); [Social communication disorder](#)

Short Description or Definition

“Semantic pragmatic disorder” is a term that describes individuals with a communicative profile characterized by relatively intact structural language (phonology, morphology, syntax) but with abnormalities in language content and use. Communicative behaviors commonly observed in semantic pragmatic disorder are listed below (Rapin 1996):

- Verbosity
- Comprehension deficits for connected speech
- Word finding deficits
- Atypical word choices
- Phonology and syntax unimpaired
- Inadequate conversational skills
- Speaking aloud to no one in particular
- Poor maintenance of topic
- Answering besides the point of the question

Categorization

In the mid-1980s, two taxonomies of developmental language disorders were independently published, one in the USA (Rapin and Allen 1983) and the other in the UK (Bishop and Rosenbloom 1987). Both described a subtype of developmental language disorder in which the primary impairment was in language content and use. Rapin and Allen termed this language profile “semantic pragmatic deficit syndrome,” and Bishop and Rosenbloom used the term “semantic pragmatic disorder.” Since the early 2000s, there has been a transition to the alternate label of pragmatic language impairment (or PLI), particularly in the UK, after evidence that semantic and pragmatic deficits do not always occur in combination (Bishop 1998).

Semantic pragmatic disorder contrasts with specific language impairment, in which there is primary impairment in the structural aspects of language, and with autism spectrum disorders, which includes a raft of behavioral difficulties such as social impairments and restricted and repetitive behaviors. However, a number of studies have shown that the diagnostic boundaries between these conditions are not clear-cut; some children with SLI exhibit pragmatic difficulties, and some children with semantic pragmatic disorder present with one or more autistic features (Bishop and Norbury 2000). Although semantic pragmatic disorder is not included in either the DSM-IV-TR or ICD-10 classification systems, it remains a relatively common diagnosis for language-impaired children in North America, Europe, and Australasia. The DSM-V proposes a new diagnostic entity, Social Communication Disorder, with diagnostic criteria that is highly similar to descriptions of Semantic Pragmatic Disorder.

Epidemiology

There has been no epidemiological study of semantic pragmatic disorder in the general population, and therefore, the incidence and prevalence of the condition remain unclear. However, it is

estimated that these children make up a significant minority of the language disordered population, with a large study in the UK finding that 25 of 242 children (10.3%) displayed characteristics of semantic pragmatic disorder (Conti-Ramsden and Botting 1999).

Natural History, Prognostic Factors, and Outcomes

One study has chartered the adult outcome of individuals diagnosed with semantic pragmatic disorder in childhood (Whitehouse et al. 2009a, b). The pattern of communication difficulties observed in childhood was found to persist to adulthood, and the adults with semantic pragmatic disorder had enduring difficulties with language use but presented with relatively intact language and literacy skills. The adults with semantic pragmatic disorder were more academically able than a comparison group of adults with specific language impairment, and all had obtained some form of tertiary education. However, while the vast majority of these adults demonstrated a range of independent behaviors, few had experienced close friendships or romantic relationships.

Clinical Expression and Pathophysiology

There is considerable debate as to whether semantic pragmatic disorder is best viewed as a subtype of developmental language disorder or an extension of pervasive developmental disorder (Boucher 1998). Evidence that some children have poor pragmatic language ability without additional autistic symptomatology (Botting and Conti-Ramsden 1999) has led to suggestions that semantic pragmatic disorder may fit a profile intermediate between specific language impairment and autism (Bishop and Norbury 2002).

Little research has investigated underlying biological mechanisms that may be associated with a diagnosis of semantic pragmatic disorder. Woodhouse et al. (1996) examined head circumference in 11 children with semantic pragmatic disorder and found that all had a head

circumference that exceeded the 90th percentile for their age, a finding that draws parallels with the widely observed acceleration in head circumference growth among children with autism (Redcay and Courchesne 2005).

An increasing number of studies have investigated biological associations with pragmatic language skills either among individuals with autism and their relatives or within the general population. Family studies indicate that pragmatic difficulties appear to be a heritable trait and are often observed among relatives of autism probands to the exclusion of any other autistic-like trait (Landa et al. 1992; Whitehouse et al. 2007, 2010a). Molecular genetic studies have started to pinpoint loci associated with pragmatic language ability, such as the short arm of chromosome 5, which was found to be associated with scores on the social communication subscales of the Children's Communication Checklist in a general population sample of 10 years of children in the UK (St. Pourcain et al. 2010). Recent studies have also indicated an association between exposure to increased levels of prenatal testosterone and pragmatic language difficulties among otherwise typically developing children (Whitehouse et al. 2010a), though this finding was not replicated in a different cohort (Knickmeyer et al. 2005).

Evaluation and Differential Diagnosis

Three methods are commonly utilized to diagnose semantic pragmatic disorder.

Standardized Assessments: There are a small number of standardized assessments of childhood pragmatic language ability. The Test of Pragmatic Language (Phelps-Terasaki and Phelps-Gunn 1992) and Test of Pragmatic Skills (Shulman 1985) are two of the more widely used assessments, requiring children to formulate appropriate utterances in relation to pictorial and/or verbal prompts. Ability is then compared to age-relevant norms. However, it is been argued that standardized assessments of pragmatic language ability lack true ecological validity for assessing communication in context (Bishop 1998).

Observation in Naturalistic Contexts:

A number of procedures have been developed for the observation and assessment of an individual's pragmatic function in context. These range from a broad assessment, in which a stretch of conversation is rated on several aspects of communicative function (Prutting and Kirchner 1987), to a more fine-grained analysis, which involves coding each utterance in a conversation (Bishop and Adams 1989). Inter-rater reliability for both approaches is known to be high for trained raters. However, this procedure is limited by the often time-consuming process of sample collection, transcription, and analysis. Furthermore, the “snapshot” approach creates uncertainty as to whether the absence of a particular behavior means that this behavior does not occur or whether it occurs rarely but was not observed at the time of sampling.

Ratings: A third assessment method is to obtain ratings of pragmatic language ability from someone who has observed the behavior of the individual over a prolonged period. This approach holds the advantages of being relatively brief and providing a more representative account of an individual's typical ability, including behaviors that occur rarely or are difficult to elicit in test situations. However, ratings are often prone to subjective interpretation, and findings should necessarily be viewed with this in mind. One widely used assessment for evaluating semantic pragmatic disorder is the Children's Communication Checklist (Bishop 1998) and its revised version, the Children's Communication Checklist 2nd edition (Bishop 2003), which have good validity in differentiating between children with specific language impairment, autism, and semantic pragmatic disorder. Adult- (Whitehouse and Bishop 2009) and self-report (Bishop et al. 2009) versions of this checklist have been developed. Other rating scales of pragmatic language include the verbal pragmatic rating scale (Bloom et al. 1999) and the adolescent pragmatics screening scale (Brice 1992). However, these scales do not provide norms and have not been validated for differential diagnoses of semantic pragmatic disorder.

Treatment

Communication difficulties are central to SPD, and thus, the primary intervention strategy is speech-language pathology. Single-case studies have provided the bulk of empirical data in this area, and, while not without the limitations, these studies suggest that speech-language pathology is effective in remediating pragmatic language difficulties, including poor speech prosody, limited communicative intent, verbosity, impaired turn taking, and reduced nonverbal communication. Findings from these studies emphasize the importance of a thorough assessment of communicative strengths and weaknesses and the provision of intervention targeted to the specific areas of need (Adams 2001; Adams et al. 2006; Adams and Lloyd 2007). Findings suggest that intensive therapy (i.e., multiple sessions per week) over a period of at least 8 weeks may provide the most significant benefit. While there is some indication that improvement in pragmatic language following speech-language therapy may endure beyond the period of direct intervention, there have been no long-term follow-up studies.

See Also

- Autism
- Pragmatic Language
- Social Communication Disorder
- Specific Language Impairment

References and Reading

Book

- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.). Washington, DC: APA Press.
- Bishop, D. V. M. (2003). *Children's Communication Checklist – 2 (CCC-2)*. London: Psychological Corporation.
- Bishop, D. V. M., Whitehouse, A. J. O., & Sharp, M. (2009). *Communication Checklist – Self Report (CC-SR)*. London: Pearson.
- Phelps-Terasaki, D., & Phelps-Gunn, T. (1992). *Test of pragmatic language*. Los Angeles: Western Psychological Services.

- Shulman, B. B. (1985). *Test of pragmatic skills*. Arizona: Communication Skills Builders.
- Whitehouse, A. J. O., & Bishop, D. V. M. (2009). *Communication Checklist – Adult (CC-A)*. London: Pearson.

Chapter in a Book

- Bishop, D. V. M. (2000). Pragmatic language impairment: A correlate of SLI, a distinct subgroup, or part of the autistic continuum? In D. V. M. Bishop & L. B. Leonard (Eds.), *Speech and language impairments in children: causes, characteristics, intervention and outcome*. Hove: Psychology Press.
- Rapin, I., & Allen, D. (1983). Developmental language disorders: Nosologic considerations. In U. Kirk (Ed.), *Neuropsychology of language, reading, and spelling* (pp. 155–184). New York: Academic.

Article in a Journal

- Adams, C. (2001). Clinical diagnostic and intervention studies of children with semantic-pragmatic language disorder. *International Journal of Language & Communication Disorders*, 36(3), 289–305.
- Adams, C., & Lloyd, J. (2007). The effects of speech and language therapy intervention on children with pragmatic language impairments in mainstream school. *British Journal of Special Education*, 34(4), 226–233.
- Adams, C., Lloyd, J., Aldred, C., & Baxendale, J. (2006). Exploring the effects of communication intervention for developmental pragmatic language impairments: A signal generation study. *International Journal of Language & Communication Disorders*, 41(1), 41–65.
- Bishop, D. V. M. (1998). Development of the Children's Communication Checklist (CCC): A method for assessing qualitative aspects of communicative impairment in children. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 39(6), 879–891.
- Bishop, D. V. M., & Adams, C. (1989). Conversational characteristics of children with semantic-pragmatic disorder. II: What features lead to a judgement of inappropriacy? *British Journal of Disorders of Communication*, 24, 241–263.
- Bishop, D. V. M., & Norbury, C. F. (2002). Exploring the borderlands of autistic disorder and specific language impairment: A study using standardised diagnostic instruments. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 43(7), 917–929.
- Bishop, D. V. M., & Rosenbloom, L. (1987). Classification of childhood language disorders. In W. Yule & M. Rutter (Eds.), *Language development and disorders: Clinics in developmental medicine [double issue]*. London: MacKeith Press.
- Bloom, R. L., Pick, L. H., Borod, J. C., Rorie, K. D., Andelman, F., Obler, L. K., et al. (1999). Psychometric aspects of verbal pragmatic ratings. *Brain and Language*, 68(3), 553–565.
- Botting, N., & Conti-Ramsden, G. (1999). Pragmatic language impairment without autism. *Autism*, 3(4), 371–396.
- Boucher, J. (1998). SPD as a distinct diagnostic entity: Logical considerations and directions for future research. *International Journal of Language & Communication Disorders*, 33(1), 71–108.
- Brice, A. E. (1992). The adolescent pragmatics screening scale. *The Howard Journal of Communications*, 3(3–4), 177–193.
- Conti-Ramsden, G., & Botting, N. (1999). Classification of children with specific language impairment: Longitudinal considerations. *Journal of Speech Language and Hearing Research*, 42(5), 1195–1204.
- Knickmeyer, R., Baron-Cohen, S., Raggatt, P., & Taylor, K. (2005). Foetal testosterone, social relationships, and restricted interests in children. *Journal of Child Psychology and Psychiatry*, 46(2), 198–210.
- Landa, R., Piven, J., Wzorek, M. M., Gayle, J. O., Chase, G. A., & Folstein, S. E. (1992). Social language use in parents of autistic individuals. *Psychological Medicine*, 22(01), 245–254.
- Prutting, C. A., & Kirchner, D. M. (1987). A clinical appraisal of the pragmatic aspects of language. *The Journal of Speech and Hearing Disorders*, 52(2), 105–119.
- Rapin, I. (1996). Practitioner review: Developmental language disorders: A clinical update. *Journal of Child Psychology and Psychiatry*, 37(6), 643–655.
- Redcay, E., & Courchesne, E. (2005). When is the brain enlarged in autism? A meta-analysis of all brain size reports. *Biological Psychiatry*, 58(1), 1–9.
- St. Pourcain, B., Wang, K., Glessner, J. T., Golding, J., Steer, C., Ring, S. M., et al. (2010). Association between a high-risk autism locus on 5p14 and social communication spectrum phenotypes in the general population. *American Journal of Psychiatry*, 267, 1363–1372.
- Whitehouse, A. J. O., Barry, J. G., & Bishop, D. V. M. (2007). The broader language phenotype of autism: A comparison with specific language impairment. *Journal of Child Psychology and Psychiatry*, 48(8), 822–830.
- Whitehouse, A. J. O., Line, E. A., Watt, H. J., & Bishop, D. V. M. (2009a). Qualitative aspects of developmental language impairment relate to language and literacy outcome in adulthood. *International Journal of Language & Communication Disorders*, 44(4), 489–510.
- Whitehouse, A. J. O., Watt, H. J., Line, E. A., & Bishop, D. V. M. (2009b). Adult psychosocial outcomes of children with specific language impairment, pragmatic language impairment and autism. *International Journal of Language & Communication Disorders*, 44(4), 511–528.
- Whitehouse, A. J. O., Coon, H., Miller, J., Salisbury, B., & Bishop, D. V. M. (2010a). Narrowing the broader autism phenotype A study using the Communication

Checklist – Adult Version (CC-A). *Autism*, 14(6), 559–574.

Whitehouse, A. J. O., Maybery, M. T., Hart, R., Mattes, E., Newnham, J. P., Sloboda, D. M., et al. (2010b). Fetal androgen exposure and pragmatic language ability of girls in middle childhood: Implications for the extreme male-brain theory of autism. *Psychoneuroendocrinology*, 35(8), 1259–1264.

Semantic Pragmatic Language Disorder

- [Autism: Social Communication Disorder](#)
- [Semantic Pragmatic Disorder](#)

Send-Out Room

- [Resource Room](#)

Sensation Avoiding

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Synonyms

[Sensory avoiding](#)

Definition

Sensation avoiding is the tendency to avoid sensory stimulation. According to Dunn's Model of Sensory Processing, persons who tend to avoid sensation have a low neurological threshold for sensation (meaning that they notice stimuli very easily), and actively attempt to manage the stimulation (Dunn 2001). Thus, sensation avoiders may be very rigid, ritualistic, and rule-bound in an attempt to control and limit stimulation in the context of daily life and reduce the amount of unpredictable stimulation. Sensation avoiders may also actively

attempt to terminate a sensation, for example, by covering one's ears, leaving a room, or avoiding crowded places. Dunn's conceptualization of sensation avoiding has been supported with standardized measures of people's responses to sensory events in daily life (Infant/Toddler Sensory Profile, Sensory Profile, Adolescent/Adult Sensory Profile), and physiological responsiveness to stimuli. On the Sensory Profile, children with autism spectrum disorders demonstrate patterns of sensation avoiding more than their peers without autism (Ben-Sasson et al. 2009).

Persons with autism who are sensation avoiding have a low tolerance for stimulation. This means that even very-low-intensity levels of sensation may be overwhelming and may interfere with everyday functioning. For example, a sensation avoider may have a negative emotional response to sensory input that someone may not normally find bothersome or even notice. Although sensation avoiding can occur for any and all sensory systems in persons with autism, extreme sensitivities to auditory stimuli are commonly reported based on parental observation and first-hand accounts from persons with autism. For example, many persons with an autism spectrum disorder have reported hypersensitivity to the "buzz" of florescent lights or "whir" of ceiling fans. Selective feeding ("picky eater"), thought to be due to extreme sensitivities to tactile stimuli and texture, is also commonly reported in persons with autism.

Interventions do not typically target changing one's way of processing information. Rather, strategies are used that enable improved performance in daily activities regardless of sensory processing patterns. Strategies to support someone who demonstrates sensation avoiding include creating environments with decreased stimulation, reducing unpredictable stimuli that occur during activities, and respecting one's need for control over their environment, perhaps through routines and rituals.

See Also

- [Sensation-Seeking](#)
- [Sensory Impairment in Autism](#)

References and Reading

- Ben-Sasson, A., Hen, L., Fluss, R., Cermak, S. A., Engel-Yeger, B., & Gal, E. (2009). A meta-analysis of sensory modulation symptoms in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 1–11.
- Dunn, W. (2001). The sensations of everyday life: Empirical, theoretical, and pragmatic considerations. *American Journal of Occupational Therapy*, 55(6), 608–620.
- Dunn, W. (2007). Supporting children to participate successfully in everyday life by using sensory processing knowledge. *Infants & Young Children*, 20(2), 84–101.
- Huebner, R. A., & Dunn, W. (2001). Introduction and basic concepts. In R. A. Huebner (Ed.), *Autism: A sensorimotor approach to management* (pp. 3–40). Austin: Pro-ed.

Sensation-Seeking

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Synonyms

[Sensory seeking](#)

Definition

Sensation-seeking is the tendency to pursue sensory stimulation and excitement. According to Dunn's Model of Sensory Processing (2001), sensation seekers generally have a high threshold for sensation (meaning that they require a lot of stimuli to notice), and actively seek out stimulation. Thus, sensation seekers can be very active and fidgety, can appear excitable and engaged, and can enjoy a variety of stimuli in their environment. For example, a child who is sensation-seeking might wiggle excessively in his chair at school to provide extra movement, hum, or make other noises to add auditory stimuli to his environment, or be excited and engaged by the variety of decorations, music, and smells of a holiday party. Differences in physiological responses (e.g., heart rate) to new stimuli have been reported in

sensation seekers versus non-sensation seekers in the literature.

Individuals with Autism Spectrum Disorders (ASDs) are often reported to have unusual responses to sensory stimulation, including sensation-seeking behaviors. Persons with autism who are sensation-seeking have a high tolerance for stimulation and derive pleasure from sensation. Thus, they demonstrate a craving for stimulation, and an interest in sensory experiences that are more intense or prolonged than what might be typically expected. Sensation seekers are typically under-responsive to environmental stimuli compared to others; thus, they require increased sensation to maintain an optimal level of arousal to participate in daily activities.

The empirical evidence for sensation-seeking in persons with ASD is mixed. Studies using caregiver questionnaires show lower or similar occurrence of sensation-seeking behaviors in toddlers with autism spectrum disorders compared to typically developing peers. However, older children, youth, and adults with autism tend to experience increased sensation-seeking behaviors compared to typically developing peers. While previous research indicated that sensory processing patterns are relatively stable across the life span, a recent meta-analysis indicated that sensory-seeking behaviors may peak in early to mid-childhood (6–9 years). Also some evidence exists that sensory-seeking behaviors may be more prominent in persons with autism (“autistic disorder”) than in persons with a diagnosis on the broader spectrum (i.e., ► [“Asperger Syndrome”](#), ► [“Pervasive Developmental Disorder Not Otherwise Specified”](#)). Overall, the literature in this area is sparse and is less consistent in sample selection, diagnostic information, and the methods used. There is also a dearth of information on adolescents and adults with autism spectrum disorder.

Interventions do not typically target changing one's way of processing information. Rather, strategies are typically used that enable improved performance in daily activities regardless of sensory processing patterns. Strategies to support someone who demonstrates sensation-seeking include creating environments that provide

increased stimulation. Providing opportunities for increased sensory experiences within the context of everyday activities is important so that the sensation seeker does not have to stop engaging in the activity to obtain the extra sensory input they crave.

See Also

- [Sensation Avoiding](#)
- [Sensory Impairment in Autism](#)

References and Reading

- Ben-Sasson, A., Hen, L., Fluss, R., Cermak, S. A., Engel-Yeger, B., & Gal, E. (2009). A meta-analysis of sensory modulation symptoms in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 1–11.
- Dunn, W. (2001). The sensations of everyday life: Empirical, theoretical, and pragmatic considerations. *American Journal of Occupational Therapy*, 55(6), 608–620.
- Dunn, W. (2007). Supporting children to participate successfully in everyday life by using sensory processing knowledge. *Infants & Young Children*, 20(2), 84–101.
- Huebner, R. A., & Dunn, W. (2001). Introduction and basic concepts. In R. A. Huebner (Ed.), *Autism: A sensorimotor approach to management* (pp. 3–40). Austin: Pro-ed.

Sense of Balance

- [Vestibular Function in Children with Autism](#)

Sensitivity and Specificity

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Synonyms

[True negative rate](#); [True positive rate](#)

Definition

Sensitivity is the proportion of true positives that are correctly identified by a test or measure (e.g., the percentage of children with autism who are correctly identified as having autism). Sensitivity is calculated by:

True Positives/(True Positives + False Negatives)

*A false negative rate (β) is calculated by $1 - \text{sensitivity}$, or False Negatives/(True Positives + False Negatives)

Specificity is the proportion of true negatives that are correctly identified by a test or measure (e.g., the percentage of children without autism who are correctly identified as not having autism). Specificity is calculated by:

True Negatives/(True Negatives + False Positives)

*A false positive rate (α) is calculated by $1 - \text{specificity}$, or False Positives/(False Positives + True Negatives)

See Also

- [Epidemiology](#)
- [Screening Measures](#)

References and Reading

- Coonrod, E. E., & Stone, W. L. (2005). Screening for Autism in young children. In F. Volkmar, A. Klin, A. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed.). New York: Wiley.
- Fombonne, E. (2005). Epidemiological studies of pervasive developmental disorders. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of Autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 42–69). Hoboken: Wiley.
- Snow, A. V., & Lecavalier, L. (2008). Sensitivity and specificity of the modified checklist for autism in toddlers and the Social communication questionnaire in preschoolers suspected of having pervasive developmental disorders. *Autism: The International Journal of Research and Practice*, 12(6), 627–644.
- Stone, W. L., Coonrod, E. E., Turner, L. M., & Pozdol, S. L. (2004). Psychometric properties of the STAT for early autism screening. *Journal of Autism and Developmental Disorders*, 34(6), 691–701.

Sensorimotor Development

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Historical Background

Arguably the most influential figure in the field of developmental psychology, Jean Piaget authored seminal work on the development of children's intelligence. He posited that cognitive and intellectual changes are the result of development, an active process that the child brings about through interactions with her environment. This process encompasses a series of successive qualitative changes of cognitive structures or schemata created by the child through experience. Piaget's background as a biologist shaped his thinking about the process as a continuous exchange between the individual and the physical world; in other words, "intelligence finds itself entangled in a network of relations between the organism and the environment" (Piaget 1952, p. 19). Piaget's body of work emerged based on extensive, meticulously detailed documentation of his own three children's behavior from birth through adolescence (Piaget 1951, 1952).

The Sensorimotor Stage (Birth to 2 Years)

Piaget (1952) described four stages of cognitive development, the first of which is sensorimotor development. This is the period from birth to approximately 24 months of age that begins with a few reflexes and ends with the appearance of language and symbolic representation of the world. Knowledge in this stage can only be derived from interactions with the physical world, and development proceeds from this exploration. Infants' sensorimotor intelligence proceeds through six increasingly complex stages, each one building on the achievements of the last. In this stage, infants and toddlers do not yet possess the full power of symbolic representation, or language, so must rely on their direct experiences with objects. By around age 2, the child's capacity to represent action in the mind increases, allowing

for conceptual thought, thus lessening the dependence on physical and sensory experience.

Substage I: The Use of Reflexes (0–1 Month)

Infants are born with several reflexes, or general action patterns such as sucking, grasping, and looking. Each reflex constitutes an "organized totality" that includes perceptions, coordinated movements, and a need; it is more than just a "summation of movements" (Piaget 1952, p. 38). These reflexes differ from simple reflexes (i.e., blinking) in that they are modified with experience. At first, there is no differentiation among stimuli; for example, newborn infants will suck on a blanket and on a nipple with equal vigor. After several weeks, infants adjust their sucking to the specific object in their mouth.

Substage II: Primary Circular Reactions (1–4 Months)

When action schemes incorporate new objects, infants move into the second substage of sensorimotor development. In this period, reflexes expand into larger coordinated behaviors, primarily centered on the infants' own bodies. Piaget described systematic thumb sucking and tongue protrusion; looking, hearing, and phonation; and prehension in this substage. Out of the single behaviors of grasping objects and sucking objects grows the integration of these two actions, grasping an object and bringing it to the mouth to suck. The ability to thumb suck at will is one of the *first acquired adaptations* that develops during this period. Turning to find the source of a voice is another primary circular reaction, reflecting coordination between hearing and vision. The term *primary circular reactions* refer to such behaviors. Reactions are primary because they focus on the infants' own bodies, and they are circular because they form "cycles of movements that repeat an interesting sensation discovered by chance" (Muller 2009, p. 208).

Substage III: Secondary Circular Reactions, or the Procedures Destined to Make Interesting Sights Last (4–8 Months)

One of the notable changes in this period is that the child's behavior becomes increasingly

oriented toward the environment, beyond her own body, including people, animals, objects, and events. At this time, infants begin to reproduce interesting actions, such as banging, shaking, and waving, especially when these actions cause an interesting visual effect or sound. “Everything thus goes back to movements of legs or feet, arms or hands, and it is these ‘circular’ movements of prehension which become differentiated in movements directed at shaking, swinging, displacing, rubbing, etc.” (Piaget 1952, p. 178). Infants begin to understand that the interesting events are related to their physical activity and thus reproduce the actions. Another characteristic of this period is the shift toward intentional, goal-directed behavior. During this period, infants still lack the concept of object permanence (see Substage IV).

Substage IV: Coordination of Secondary Schemata (8–12 Months)

Infants begin to use means to attain ends that are not attainable directly. For example, they will push aside a pillow to reach a toy, or pull a string to obtain an object attached to one end. The main achievement of this period is *object permanence*, the awareness that objects exist when they are no longer in sight. During this period, infants begin to search for objects that disappear, indicating that they understand that objects still exist after they disappear from view. The fragility of this mental representation, however, is exemplified in the *A-not-B error*. Once infants have searched for a hidden object at one location (A), when the location is moved (B) they continue to search for it at location A. Not until around the first birthday is the infant able to inhibit search at the previous location and look for the object at the new location.

Substage V: Tertiary Circular Reactions, or the Discovery of New Means Through Active Experimentation (12–18 Months)

Beginning at 12 months of age, infants begin to explore their worlds more actively, “experimenting” with objects, intent on seeing how they behave in new situations. Once making such discoveries, they are repeated, but these

tertiary circular reactions differ from the previous kinds in that the infant varies the actions slightly each time. In this stage, rather than applying the same schemata to problems, they now find new means through trial and error. In contrast to secondary circular reactions, in which infants attempt to recreate a certain event, tertiary circular reactions aim to “ferret out new phenomena” (Piaget 1952, p. 274) in the process.

Substage VI: Invention of New Means Through Mental Combinations (18–24 Months)

In this final stage, the child begins to invent as well as discover. Rather than relying on trial-and-error experimentation, the child begins to be able to devise solutions to simple problems through representation (thinking), independent of immediate experience. In effect, the experimentation happens in thought. This development of mental representation fosters another hallmark of this period, *deferred imitation*, whereby children form mental representations of events so enduring that they can repeat others’ behavior hours or even days after it was first observed.

Current Knowledge

Sensorimotor Development in Autism

Measurement of sensorimotor development in autism was first undertaken in order to assess intellectual functioning in severely affected non-verbal children (Curcio 1978). The Ordinal Scales of Psychological Development had recently been published (Uzgiris 1975) based on Piagetian stages of sensorimotor development in the first 2 years, and was the instrument of choice for many researchers conducting studies in this domain. The resulting studies reported no deficits in children with autism in most areas of sensorimotor development (with the notable exception of gestural imitation) compared to mental-age-matched peers. Furthermore, a relative strength in object permanence skills was often observed in the children with autism (Dawson and McKissick 1984; Morgan et al. 1989; Sigman and Ungerer 1981, 1984; Wetherby and Gaines 1982).

A hypothesis from the literature on typical infant development posited that intentional communication arises in the first year concurrent to the development of Stage IV sensorimotor skills. In other words, the discovery that goals can be met by way of intermediate actions and that other people can act on behalf of the child is necessary before the infant can engage in intentional communication, including actions such as protodeclarative (i.e., showing, pointing to share interest) and protoimperative gestures (i.e., pointing to request) (Bates et al. 1979). Thus, a number of studies were conducted to examine the relationship between sensorimotor skills and communication abilities in children with autism. While some studies found a positive correlation between them (Abrahamsen and Mitchell 1990; Curcio 1978), others reported cognitive skills well beyond the children's language skills (Sigman and Ungerer 1981; Wetherby and Gaines 1982) and thus no clear relationship between the two. Relatively small samples of children of varying ages and ability level as well as different measures of language or prelinguistic functioning may have contributed to the disparate findings.

See Also

- [Object Permanence](#)
- [Piagetian Stages](#)

References and Reading

- Abrahamsen, E. P., & Mitchell, J. R. (1990). Communication and sensorimotor functioning in children with autism. *Journal of Autism and Developmental Disorders*, 20(1), 75–85.
- Baranek, G. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders*, 29(3), 213–224.
- Bates, E., Benigni, L., Bretherton, L., Camaioni, L., & Volterra, V. (1979). *The emergence of symbols: Cognition and communication in infancy*. New York: Academic Press.
- Curcio, F. (1978). Sensorimotor functioning and communication in mute autistic children. *Journal of Autism and Childhood Schizophrenia*, 8(3), 281–292.
- Dawson, G., & McKissick, F. C. (1984). Self-recognition in autistic children. *Journal of Autism and Developmental Disorders*, 14(4), 383–394.
- De Myer, M. K., Mann, M. A., Tilton, J. R., & Loew, L. H. (1967). Toy play behavior and use of body by autistic and normal children as reported by mothers. *Psychological Reports*, 21, 973–981.
- Lösche, G. (1990). Sensorimotor and action development in autistic children from infancy to early childhood. *Journal of Child Psychology and Psychiatry*, 31(5), 749–761.
- Morgan, S. B., Cutrer, P. S., Coplin, J. W., & Rodrigue, J. R. (1989). Do autistic children differ from retarded and normal children in piagetian sensorimotor functioning? *Journal of Child Psychology and Psychiatry*, 30(6), 857–864.
- Muller, U. (2009). Infancy. In U. Müller, J. I. M. Carpendale, & L. Smith (Eds.), *The Cambridge companion to Piaget* (pp. 200–228). Cambridge: Cambridge University Press. Cambridge Collections Online.
- Ozonoff, S., Macari, S., Young, G. S., Goldring, S., Thompson, M., & Rogers, S. J. (2008). Atypical object exploration at 12 months of age is associated with autism in a prospective sample. *Autism*, 12(5), 457–472.
- Piaget, J. (1951). *Play, dreams, and imitation in childhood*. New York: WW Norton.
- Piaget, J. (1952). *The origins of intelligence in children*. New York: International Universities Press.
- Sigman, M., & Ungerer, J. (1981). Sensorimotor skills and language comprehension in autistic children. *Journal of Abnormal Child Psychology*, 9(2), 149–165.
- Sigman, M., & Ungerer, J. (1984). Cognitive and language skills in autistic, mentally retarded, and normal children. *Developmental Psychology*, 20(2), 293–302.
- Stone, W. L., Lemanek, K. L., Fischel, P. T., Fernandez, M. C., & Altemeier, W. A. (1990). Play and imitation skills in the diagnoses of autism in young children. *Pediatrics*, 86, 267–272.
- Uzgiris, I. (1975). *Assessment in infancy: Ordinal scales of psychological development*. Urbana: University of Illinois Press.
- Wetherby, A. M., & Gaines, B. (1982). Cognition and language development in autism. *The Journal of Speech and Hearing Disorders*, 47(1), 63–70.
- Williams, E. (2003). A comparative review of early forms of object-directed play and parent-infant play in typical infants and young children with autism. *Autism*, 7(4), 361–374.

Sensorimotor Play

- [Play](#)

Sensory Aphasia

► Wernicke's Aphasia

Sensory Avoiding

► Sensation Avoiding

Sensory Diet

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Definition

The term “sensory diet” was coined by an occupational therapist, Patricia Wilbarger, following the work of Dr. A. Jean Ayres’ theory of sensory integration. This term refers to the need for an individual to have varied sensory experiences throughout the day, similar to a nutritional diet, to maintain an optimal level of arousal or alertness to meet environmental and task demands.

Historical Background

For well over 40 years occupational therapists providing intervention for children with sensory processing disorders (SPD) or sensory integrative disorders (SID) have advocated for the use of varied sensory experiences to help a child develop more functional and adaptive skills (Ayres 1971, 1973). Characteristically, children who display SPD or SID have very limited variety in their responses to sensory experiences and have difficulties when faced with the need to produce a graded response to a specific situation. Ayres (1973) described a group of children with SID who responded in a defensive manner to seemingly non-noxious or nonirritating sensory

experiences. These children, in particular, seemed to need graded sensory experiences to help them produce functional responses. An example can be seen when a young girl becomes overly distressed to the point of crying when having her hair combed or a boy starts gagging when he smells raw carrots. Children with SPD or SID benefit from varied sensory experiences to help produce a functional and graded adaptive response to a situation. Ayres’ concept of providing graded sensory experiences coupled with child-directed play activities formed the foundation for the concept of a sensory diet.

The term “sensory diet” has been attributed to the occupational therapist Patricia Wilbarger (1984) and then further described by Patricia and her daughter, also an occupational therapist, Julia Wilbarger (1991). They used the term “sensory diet” to describe the use of varied sensory experiences with a graded and child-directed approach to foster organized responses to situations. Following Ayres’ concepts of sensory defensiveness, they postulated that a “sensory diet” would help a “child feel calm, alert, and organized most of the time using special activities” in a manner similar to eating well-balanced meals on a regularly scheduled basis (Wilbarger and Wilbarger 1991, p. 6). These sensory experiences help the child maintain an organized response to functional activities. Although originally applied primarily to children with a sensory defensive profile, the concept of a sensory diet was expanded and used with other children (Williams and Shellenberger 1994) and with adults (Kinnealey et al. 1995).

Rationale or Underlying Theory

For an individual to maintain an optimal level of alertness to effectively respond to environmental situations requires regular and varied sensory experiences or a sensory diet (Wilbarger 1995). The rationale offered includes research addressing deprivation studies and resultant behavioral issues. The concept of a “diet” reflects the need for a variety of sensory experiences and for those experiences to be provided throughout the day in a manner similar to a nutritional diet. The sensory

diet is not the same for each person but instead is tailored to meet the unique needs of the individual (Williams and Shellenberger 1994). Specific areas of the brain respond selectively to various forms of sensory information, but if the brain is deprived of a specific form of sensory information, that portion of the brain will undergo neuroplastic changes to process different information (Merabet and Pascual-Leone 2010). The sensory diet is provided to enrich the brain and foster appropriate processing of sensory information to allow the individual to be alert and adaptive within his or her environment. Individuals who are overly aroused or defensive to a specific form of sensory information often present with maladaptive responses or avoidance.

Goals and Objectives

The goals of a sensory diet follow the overarching goals of sensory integrative therapy, to help the individual produce functional and adaptive responses to the environment (Dunn 2007). To meet this goal, the first concern would be to have the individual achieve and then maintain an optimal level of arousal or alertness needed for the activity (Williams and Shellenberger 1994). From Wilbarger's (1984) concept of a sensory diet, the first sensory systems addressed are proprioceptive, tactile, and vestibular. These follow Ayres' (1972a) classic approach to sensory integrative therapy. The sensory diet includes activities that are designed and structured for the client throughout the day to enhance the level of alertness or arousal needed to meet the functional task. During play activities, the level of alertness or arousal would tend to be higher than during naptime or during meals. The goals are individually developed and reflect a client-centered approach (Pfeiffer et al. 2011; Wilbarger 1995). Instead of prescribing the same sensory experience for every client, the occupational therapist determines what pertinent goals the client or, in the case of very young children and infants, the family wants to achieve. A careful analysis of the individual's

response to current sensory systems and variations in levels of alertness and arousal throughout the day is completed through an interview, and then goals are mutually established. Individuals vary substantially in sensory preferences, and assuming that everyone would benefit from the same sensory experience would be similar to assuming everyone enjoys dark chocolate. Following the interview process, the sensory diet is developed to meet the agreed upon goals. An example can be seen with a child who had difficulties going to bed at night and became very distressed, hyperverbal, and active. Using a weighted blanket to provide deep pressure and then adding slow rocking in a rocking chair helped this child develop the routine of being calm before the event of going to bed instead of viewing this transition as a challenge. The parents had requested occupational therapy services to help with the daily routines related to preparation for going to bed, and the use of the sensory diet was an effective means to meet this specific goal.

Treatment Participants

The sensory diet was initially used for infants and children who presented with a defensive response style to non-noxious sensory experiences (Ayres 1972a; Wilbarger 1984, 1995) but was then extended to children with other SPD or SID profiles (Williams and Shellenberger 1994) and to adults (Kinnealey et al. 1995). The sensory diet has also been used with individuals who are diagnosed with autism spectrum disorders (ASD). The recognition of unusual responses to sensory stimuli for individuals who have ASD has been included in the diagnostic criteria in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR American Psychiatric Association 2000). Evidence of SPD in individuals diagnosed with ASD has been provided by several authors (Barenek 2002; Dunn et al. 2002a; Kern et al. 2006; Kientz and Dunn 1997; Minshew and Hobson 2008; Tomchek and Dunn 2007), and the incidence of SPD for

this population varies from 42% to 88% (Barenek 2002).

Treatment Procedures

The first step is identification of the individual's responses to various sensory experiences and the person's level of alertness or arousal throughout the day. There are reliable instruments available to assist with the identification of sensory responses as similar or dissimilar to peers such as the Sensory Profile (Dunn 1999), Infant/Toddler Sensory Profile (Dunn 2002), Adolescent/Adult Sensory Profile (Brown and Dunn 2002), School Companion Sensory Profile (Dunn 2006), and the Sensory Processing Measure (Miller-Kuhanek et al. 2008). An interview would then identify the patterns of alertness or arousal during the day. The types of task during that portion of the day would be compared to the typical level of arousal needed to meet the task demands and to assess the fit of these factors (Nackley 2001; Wilbarger 1995). For example, a child who is very active and disorganized at the beginning of the school day would have difficulties with the social expectations of sharing toys or playing an interactive game with peers. This child may need additional sensory experiences to help his level of arousal to slightly diminish to a more appropriate level to allow social participation with peers. Information about the individual's ability to transition between various activities should be included in the interview process (Wilbarger 1995).

The second step in the treatment procedures is identification of beneficial sensory experiences that either increase or decrease the level of arousal for the individual as needed to meet task demands (Wilbarger 1995; Williams and Shellenberger 1994). These sensory experiences can be grouped into large categories of sensory input, but the form of the sensory experience may be different for each individual. The three general categories of sensory experiences used in the sensory diet are deep pressure (proprioceptive), movement

(vestibular), and touch (tactile). The sensations of vision, sound, taste, and smell are also used when creating a sensory diet with careful consideration for individual differences. This concept is illustrated with the sense of smell and how a specific odor may be very calming for one individual but distressing to another individual. When looking at the form of the sensory input, it is important to consider the use of this sensory input within the individual's daily life (Nackley 2001). For example, deep pressure (proprioceptive input) may be helpful in modifying the level of alertness to allow individuals to meet task demands, but the form of the deep pressure is often different. For an individual, it is best provided through the use of a weighted vest that is snugly fitted (VandenBerg 2001). Another individual, who also finds the use of proprioceptive input helpful, prefers the use of squeezing a resistive squeeze ball throughout the day to assist with level of alertness (Nackley 2001).

The third step in developing the sensory diet is developing a plan where the preferred sensory experiences can be provided throughout the day (Nackley 2001; Wilbarger 1995). These have been referred to as a "sensory tune-up" and reflect the need to provide the individual with opportunities throughout the day to use sensory input to adjust the level of alertness in preparation for the task demands. Wilbarger (1995) discussed the need to use preferred sensory input prior to transitions between activities in addition to regularly scheduled periods. Specific forms of sensory experiences tend to have longer lasting impact on the level of arousal or alertness. Vestibular-based activities that are alerting, spinning in a revolving chair or on a scooter board, can have effects lasting several hours, and this form of sensory input should only be used following recommendations of a trained occupational therapist. Proprioceptive input can also produced longer lasting results of 1–2 h in duration. Changes in noise and lighting can produce an immediate response in the level of alertness, but the duration tends to be shorter. Wilbarger (1995) notes the visual and auditory sensory

input can also evoke emotional responses and that should be considered when completing the interview process. Nackley (2001) provided a description of how the sensory diet could be structured throughout the school day and coupled with daily tasks.

Efficacy Information

Large randomized controlled trials using a sensory diet have not been completed. The current research addresses use of sensory integration treatment from which a sensory diet was derived. Although numerous studies exist using sensory integration strategies to produce changes, the results have been mixed (May-Benson and Koomar 2010). Often the heterogeneity and small numbers of participants resulted in potential type II errors, and the use of inappropriate measures did not reflect the foundation of sensory integration and the use of a sensory diet, that being an individually designed intervention to help the person respond adaptively and appropriately to task and environmental demands. Recent literature using Goal Attainment Scaling (GAS) as an outcome measure has demonstrated significant improvements when using sensory integration treatment (Mailloux et al. 2007; Pfeiffer et al. 2011). Goal Attainment Scaling (GAS) is a method to foster client participation in the goal setting process that is meaningful and relevant (Pfeiffer et al.). In an investigation comparing the use of sensory integration services to fine motor intervention services for children with ASD, superior gains were reported by parents on GAS scores for those children receiving sensory integration services. Additionally, gains were identified in reduced autistic mannerisms, as measured by the Social Responsiveness Scale for the children receiving sensory integration treatment.

When a sensory diet has been used in conjunction with other sensory strategies, positive results were reported (Hall and Case-Smith 2007). A small study of 10 school-aged children with SPD using a combination of a sensory diet with a therapeutic listening program produced

significant gains in sensory processing scores and handwriting skills.

Outcome Measurement

Recent literature has provided a clearer direction for research on the efficacy of sensory integration treatment of which the sensory diet is derived (Parham et al. 2011). The fidelity measure for Ayres Sensory Integration intervention was developed to insure that therapeutic methods used in an investigation accurately represent the philosophy and principles of this approach. The sensory integration approach described by Ayres (1969, 1971, 1972a, b, 1973, 1982) served as the foundation for the sensory diet (Wilbarger 1984). This fidelity measure identified the need to engage in collaborative goal setting with family members and teachers along with discussing the influence of sensory integration on the individual's participation in functional activities. The use of GAS has shown promise in supporting the principles outlined by Ayres and the fidelity measure. Investigations using GAS have reported improvements following sensory integration (Pfeiffer et al. 2011; Schaaf and Nightlinger 2007).

Qualifications of Treatment Providers

The fidelity measure of Ayres Sensory Integration intervention stipulates that the therapist receive post-professional training in sensory integration with a minimum of 50 educational hours in theory and practice. The sensory diet was developed from the theory of sensory integration, and these standards should be applied. Occupational therapists are the professionals most frequently pursuing advanced training in the theory and practice of sensory integration beyond the post-professional basis. The entry-level occupational therapist has completed courses within the professional preparation to understand the neuroanatomy, neurophysiology, and occupation base of sensory integration theory and practice.

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders-text revision*. Washington, DC: Author.
- Ayres, A. J. (1969). Deficits in sensory integration in educationally handicapped children. *Journal of Learning Disabilities*, 2, 160–168.
- Ayres, A. J. (1971). Characteristics of types of sensory integrative dysfunction. *American Journal of Occupational Therapy*, 25, 329–334.
- Ayres, A. J. (1972a). Types of sensory integrative dysfunction among disabled learners. *American Journal of Occupational Therapy*, 22, 13–18.
- Ayres, A. J. (1972b). Improving academic scores through sensory integration. *Journal of Learning Disabilities*, 5, 338–343.
- Ayres, A. J. (1973). *Sensory integration and learning disorders*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (1982). *Sensory integration and the child*. Los Angeles: Western Psychological Services.
- Barenek, G. T. (2002). Efficacy of sensory and motor interventions for children with autism. *Journal of Autism and Developmental Disorders*, 32, 397–422.
- Brown, C., & Dunn, W. (2002). *Adolescent/adult sensory profile: User's manual*. San Antonio: Psychological Corp.
- Dunn, W. (1999). *Sensory profile: User's manual*. San Antonio: Psychological Corp.
- Dunn, W. (2002). *Infant/toddler sensory profile: User's manual*. San Antonio: Psychological Corp.
- Dunn, W. (2006). *School companion sensory profile: User's manual*. San Antonio: Psychological Corp.
- Dunn, W. (2007). Supporting children to participate successfully in everyday life by using sensory processing knowledge. *Infants and Young Children*, 20, 84–101.
- Dunn, W., Myles, B. S., & Orr, S. (2002a). Sensory processing issues associated with Asperger syndrome: A preliminary investigation. *American Journal of Occupational Therapy*, 56, 97–102.
- Dunn, W., Saiter, J., & Rinner, L. (2002b). Asperger syndrome and sensory processing: A conceptual model and guidance for intervention planning. *Focus on Autism and Other Developmental Disabilities*, 17, 172–185.
- Hall, L., & Case-Smith, J. (2007). The effect of sound-based intervention on children with sensory processing disorders and visual-motor delays. *American Journal of Occupational Therapy*, 61, 209–215.
- Kern, J. K., Trivedi, M. H., Garver, C. V., Grannemann, B. D., Andrews, A. A., Salva, J. S., et al. (2006). The pattern of sensory processing abnormalities in autism. *Autism*, 10, 480–494.
- Kientz, M. A., & Dunn, W. (1997). A comparison of the performance of children with and without autism on the sensory profile. *American Journal of Occupational Therapy*, 51, 530–537.
- Kinnealey, M., Oliver, B., & Wilbarger, P. (1995). A phenomenological study of sensory defensiveness in adults. *American Journal of Occupational Therapy*, 49, 444–451.
- Mailloux, Z., May-Benson, T. A., Summers, C. A., Miller, L. J., Brett-Green, B., Burke, J. P., et al. (2007). The issue is – Goal attainment scaling as a measure of meaningful outcomes for children with sensory integration disorders. *American Journal of Occupational Therapy*, 61, 254–259.
- May-Benson, T. A., & Koomar, J. A. (2010). Systematic review of the research evidence examining the effectiveness of interventions using a sensory integrative approach for children. *American Journal of Occupational Therapy*, 64, 403–414.
- Merabet, L. B., & Pascual-Leone, A. (2010). Neural reorganization following sensory loss: The opportunity of change. *Nature Reviews: Neuroscience*, 11, 44–52.
- Miller-Kuham, H., Henry, D., Glennon, T., Parham, L. D., Ecker, C., & Burke, J. P. (2008). *The sensory processing measure*. Los Angeles: Western Psychological Services.
- Minshew, N. J., & Hobson, J. A. (2008). Sensory sensitivities and performance on sensory perceptual tasks in high-functioning individuals with autism. *Journal of Autism and Developmental Disorders*, 38, 1485–1498.
- Nackley, V. L. (2001). Sensory diet application and environmental modifications: A winning combination. *Sensory Integration Special Interest Section Quarterly*, 24, 1–4.
- Parham, L. D., Roley, S. S., May-Benson, T. A., Koomar, J., Brett-Green, B., et al. (2011). Development of a fidelity measure for research on the effectiveness of the Ayres Sensory Integration Intervention. *American Journal of Occupational Therapy*, 65, 133–142.
- Pfeiffer, B. A., Koenig, K., Kinnealey, M., Sheppard, M., & Henderson, L. (2011). Effectiveness of sensory integration interventions in children with autism spectrum disorders: A pilot study. *American Journal of Occupational Therapy*, 65, 76–85.
- Schaaf, R. C., & Nightlinger, K. M. (2007). Occupational therapy using sensory integrative approach: A case study of effectiveness. *American Journal of Occupational Therapy*, 61, 239–246.
- Tomchek, S. D., & Dunn, W. (2007). Sensory processing in children with and without autism: A comparative study using the Short Sensory Profile. *American Journal of Occupational Therapy*, 61, 190–200.
- VandenBerg, N. L. (2001). The use of a weighted vest to increase on-task behavior in children with attention difficulties. *American Journal of Occupational Therapy*, 55, 621–628.
- Wilbarger, P. (1984, September). Planning an adequate sensory diet-application of sensory processing theory during the first year of life. *Zero to Three*, 7–12.
- Wilbarger, P. (1995). The sensory diet: Activity programs based on sensory processing theory. *Sensory Integration Special Interest Section Quarterly*, 18, 1–4.

- Wilbarger, P., & Wilbarger, J. L. (1991). *Sensory defensiveness in children aged 2–12*. Santa Barbara: Avanti Educational Programs.
- Wilbarger, J., & Wilbarger, P. (2002). Wilbarger approach to treating sensory defensiveness and clinical application of the sensory diet. Sections in alternative and complementary programs for intervention. In A. C. Bundy, E. A. Murray, & S. Lane (Eds.), *Sensory integration: Theory and practice* (2nd ed.). Philadelphia: F. A Davis.
- Williams, M. S., & Shellenberger, S. (1994). *How does your engine run? A leader's guide to the ALERT program for Self-regulation*. Albuquerque: TherapyWorks.

Sensory Experiences Questionnaire

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Synonyms

SEQ

Description

The Sensory Experiences Questionnaire (Version 3.0; SEQ; Baranek 2009; Ausderau et al. 2014b) is a caregiver report assessment intended to be used by researchers and clinicians to characterize the sensory features in children ages 2–12 years with autism spectrum disorder (ASD) and/or other developmental disabilities (DD). The questionnaire typically requires about 15–20 minutes for a parent or caregiver to complete. If needed, the SEQ (Version 3.0) can also be administered in a structured interview format. The SEQ (Version 3.0) has 105 items that measure the frequency of

sensory features across four sensory response patterns (i.e., Hyporesponsiveness; Hyperresponsiveness; Sensory Interests, Repetitions and Seeking behaviors (SIRS), and Enhanced Perception), which reflect items across five sensory modality categories (i.e., auditory; visual; tactile; gustatory/olfactory; and vestibular/proprioceptive), and across two contexts (i.e., social or non-social). The first 97 items measure the frequency of the child's sensory behaviors using a 5-point Likert scale ranging from 1 (almost never) to 5 (almost always). The last eight items ask broader questions about the child's sensory experiences and allow the caregiver to elaborate with a qualitative response. The summary scores for the four sensory response patterns are calculated based on the 97 quantitative items, excluding control items, with the qualitative answers providing further contextual information. A higher score indicates a greater frequency or intensity of sensory features.

Historical Background

The Sensory Experiences Questionnaire, formerly known as the Sensory Supplement Questionnaire (SSQ), was developed by Grace Baranek in 1999. It has been through four revisions (Versions 1.0, 2.0, 2.1, and 3.0) to better meet research needs and the evolving conceptual model. The revisions have included refining items for a broader age range, expanding the number of items, adding control items, increasing the types of sensory patterns represented, and further psychometric studies. The items included in the SEQ (Version 3.0) were developed from a review of existing literature on sensory features in children with ASD including empirical studies (Ayres and Tickle 1980; Baranek et al. 1997, 2005, 2014; Ben-Sasson et al. 2009; Dahlgren and Gillberg 1989; Iarocci and McDonald 2006; Lord et al. 1994; Rogers and Ozonoff 2005; Stone and Hogan 1993), parental report studies (Kientz and Dunn 1997; Lord 1995; Ornitz et al. 1977; Rogers et al. 2003; Stone and Hogan 1993), expert clinical reports (Greenspan and Wieder 1997), conceptual models of sensory processing (Ayres 1989;

Baranek et al. 2001; Dunn 1997; Miller et al. 2001), and consideration of neuropsychological theories of ASD describing core features (Mundy and Neal 2001; Ozonoff et al. 2005; Rajendran and Mitchell 2007; Volkmar et al. 2004; Waterhouse et al. 1996).

Psychometric Data

Throughout the development of the SEQ, the psychometric properties of the versions have been assessed on an ongoing basis to ensure its reliability and validity. The overall internal consistency of the SEQ (Version 1.0) (21-item version) was 0.80 (Cronbach's alpha) (Baranek et al. 2006) and the test-retest reliability was 0.92 (intraclass correlation coefficient) (Little et al. 2011). Reliability analysis (internal consistency) of the subscales for the SEQ (Version 2.1) yielded the following psychometrics (Cronbach's alpha): Hyperresponsiveness = 0.73; Hyporesponsiveness = 0.75; Sensory Seeking = 0.80; Social = 0.69; Nonsocial = 0.78 (Little et al. 2011).

A known-groups validity study with 258 caregivers of children ages 5–80 months found that the SEQ (Version 1.0) discriminated well between children with ASD and those with other DD, as well as those with typical development (Baranek et al. 2006). Prevalence of overall sensory features for the ASD group was 69% (versus 38% for the DD group) based on the items sampled. Sensory symptoms were inversely related to mental age such that more developmentally mature children across the groups demonstrated fewer sensory features. Children with ASD in this study had increased levels of overall sensory features as well as increased patterns of Hyporesponsiveness compared to children with other DD in both social and nonsocial contexts. Children in any of the clinical groups (those with ASD as well as those with DD) showed increased levels of Hyperresponsiveness compared to typically developing children. This study demonstrated the SEQ's ability to discriminate unique pan-modal sensory response patterns in children with ASD from comparison groups.

Walz and Baranek (2006) using the SEQ (Version 1.0) demonstrated the SEQ's validity for characterizing sensory response patterns in children with Angelman syndrome through 18 years of age. In a longitudinal analysis of sensory processing, Baranek et al. (2008) demonstrated that the SEQ (Version 1.0) was sensitive to maturational changes when examining developmental trajectories of sensory processing in 13 boys with fragile X syndrome from ages 9 months through 5 years.

Two studies showed the utility of the SEQ to predict to restricted and repetitive behaviors (RRB). Boyd et al. (2010) used the SEQ (Version 2.1) in combination with other sensory measures to examine the association between three sensory response patterns and RRB in children with ASD ($n = 67$, mean age = 51.69 months) and DD ($n = 42$, mean age = 49.45 months). The sensory measures used in this study included the SEQ and were used to validate three sensory constructs (i.e., Hyporesponsiveness; Hyperresponsiveness; and Sensory Seeking) through a factor analysis. Model fit was assessed using standard fit measures: Chi-square = 32.4 (31), RMSEA = 0.021, and CFI = 0.997. A major finding of the study was the significant association between Hyperresponsiveness and the presence of RRB in children with ASD and DD and primarily nonsignificant associations with the other sensory response patterns. A more recent study with 87 toddlers at elevated risk for ASD showed that the SEQ (Version 2.1) Hyperresponsiveness predicted significantly to later RRB symptoms at 3–5 years of age (Grzadzinski et al. 2020).

To further assess the relationship between the sensory response patterns (i.e., Hyporesponsiveness; Hyperresponsiveness, and Sensory Seeking) and social communication in children with ASD ($n = 72$, mean age = 52.3 months) and DD ($n = 44$, mean age = 48.1 months), Watson et al. (2011) used the SEQ (Version 2.1) in combination with other sensory measures to determine children's sensory response pattern scores and other measures of social-communicative symptoms of ASD, as well as language, social, and communication skills. A primary finding of the study was a

positive association between Hyporesponsiveness and social-communicative symptom severity for both the ASD and the DD groups. Hyperresponsiveness was not associated with social-communicative symptom severity for either groups, and Sensory Seeking was only associated with social-communicative symptom severity in the ASD group. The SEQ (Version 2.1) was able to be used in combination with sensory measures to assess their unique association with social communication as well as other language and communication skill measures.

Using a national sample of 1,307 children with ASD, Ausderau et al. (2014) empirically validated the factor structure the SEQ (Version 3.0). A confirmatory factor analytic model with 10 latent variables: four substantive factors of hypothesized sensory response patterns (i.e., Hyporesponsiveness; Hyperresponsiveness; SIRS; and Enhanced Perception), five method factors of sensory modalities (i.e., auditory, visual, tactile, gustatory/olfactory, and vestibular/proprioception), and one social context were tested, resulting in good model fit.

Correlations between the substantive factors were freed, but fixed to zero between the method factors, as well as between the method factors and the substantive factors. Model fit for this confirmatory factor model was assessed using standard fit measures: Chi-square = 16,724.18 (3984) ($p < 0.01$), RMSEA = 0.051, and SRMR = 0.07. Factor loadings for the items on the latent variables were generally strong and provided support for each of the hypothesized sensory content factors. Between-factor correlations ranged from 0.44 to, suggesting that these factors were distinct and they covaried significantly. The study confirmed the factor structure for the SEQ (Version 3.0), and tested measurement invariance for age and sex. To understand the impact of the four sensory response patterns on autism severity, this study also conducted a mixed model regression analysis, controlling for key child (i.e., chronological age, IQ, sex) and family characteristics (e.g., education and income). All four sensory response patterns

significantly predicted autism severity as measured by the Social Responsiveness Scale (SRS).

Two subsequent large sample studies demonstrated the utility of the SEQ (Version 3.0) for subtyping ASD sensory features. One study with 1294 children with ASD found four sensory subgroups of children with ASD (i.e., Mild; Attenuated-Preoccupied; Sensitive-Distressed; and Extreme) (Ausderau et al. 2014a) and these subtypes were found to be highly stable over a 1-year period based on a latent profile transition analysis. In addition, a follow-up study (Ausderau et al. 2016) found that the four subtypes measured by the SEQ (Version 3.0) were found to predict differentially to outcomes in adaptive behavior, parenting stress, and autism severity 1 year later in a sample of 884 children with ASD.

Baranek et al. (2019) conducted a study to look at the stability of sensory features in a longitudinal study with 55 children with ASD and 35 with DD. Regression analyses revealed that for both groups SEQ (versions 2.1 and 3.0) sensory response pattern scores early in development (preschool period) were significantly strong predictors of scores 3 years later (on average). Correlation coefficients between the two time points were statistically significant (Hyporesponsiveness 0.63–0.64; Hyperresponsiveness 0.55–0.60; SIRS 0.63–0.73) for the two groups. Mean scores were found to decline for SIRS in both groups and for Hyporesponsiveness in the ASD group. Therapeutic services received between the two time points were positively associated with the severity of sensory features, suggesting that children with more severe sensory processing problems may fail to improve over time.

Clinical Uses

The SEQ is meant to be used alone or in combination with other diagnostic tools by researchers and clinicians. It can be used to characterize sensory features in children by sensory response pattern, modality, and social context. The SEQ can also be used to assist in discrimination of sensory

features that are specific to children with ASD versus those with DD or typical development. Clinicians can identify and quantify the frequency of a range of sensory features in children that may require further evaluation and/or intervention. The characterization of the sensory features or sensory subtypes may also assist clinicians in treatment planning. Information gleaned from the SEQ (Version 3.0) can be used to develop targeted, personalized interventions. Clinicians interested in using a strengths-based approach to working with children with ASD may find the SEQ (Version 3.0) particularly relevant, as the Enhanced Perception construct may enable clinicians to address an area of particular strength in some individuals. The SEQ is a reliable and valid tool that may be useful in monitoring changes in sensory features over time as a result of maturation or intervention.

The SEQ can be used by researchers to screen for inclusion into certain research studies, to characterize sensory features in children with ASD or DD, and to assess the stability or change in sensory features over time as related to sensory response pattern, modality, and context. It also provides an assessment tool to relate sensory features of children to a variety of outcome variables such as children's autism severity, adaptive skills, or parenting stress. The SEQ (Version 2.1) has been used in at least two randomized controlled trials (Baranek et al. 2015; Watson et al. 2017).

See Also

- [Enhanced Perceptual Functioning](#)
- [Hyperresponsiveness](#)
- [Hypo-arousal](#)
- [Hyporesponsiveness](#)
- [Sensation Avoiding](#)
- [Sensation-Seeking](#)
- [Sensory Impairment in Autism](#)
- [Stereotyped and Restrictive Behaviors and Sensory Interests \(SRS\)](#)
- [Sensory Processing](#)
- [Tactile Defensiveness](#)

References and Reading

- Ausderau, K. K., Furlong, M., Sideris, J., Bulluck, J., Little, L. M., Watson, L. R., . . . , & Baranek, G. T. (2014a). Sensory subtypes in children with autism spectrum disorder: Latent profile transition analysis using a national survey of sensory features. *Journal of Child Psychology and Psychiatry*, 55(8), 935–944.
- Ausderau, K., Sideris, J., Furlong, M., Little, L. M., Bulluck, J., & Baranek, G. T. (2014b). National survey of sensory features in children with ASD: Factor structure of the Sensory Experience Questionnaire (3.0). *Journal of Autism and Developmental Disorders*, 44(4), 915–925.
- Ausderau, K. K., Sideris, J., Little, L. M., Furlong, M., Bulluck, J. C., & Baranek, G. T. (2016). Sensory subtypes and associated outcomes in children with autism spectrum disorders. *Autism Research*, 9(12), 1316–1327.
- Ayres, A. J. (1989). *Sensory Integration and Praxis Tests (SIPT)*. Los Angeles: Western Psychological Services.
- Ayres, A. J., & Tickle, L. S. (1980). Hyper-responsivity to touch and vestibular stimuli as a predictor of positive response to sensory integration procedures by autistic children. *American Journal of Occupational Therapy*, 34, 375–381.
- Baranek, G. T. (1999). *Sensory Experiences Questionnaire (SEQ, version 2.1)*. University of North Carolina at Chapel Hill.
- Baranek, G. T. (2002). Efficacy of sensory and motor interventions for children with autism. *Journal of Autism and Developmental Disorders*, 32, 397–422.
- Baranek, G. T. (2009). *Sensory Experiences Questionnaire (SEQ, version 3.0)*. University of North Carolina at Chapel Hill.
- Baranek, G. T., Foster, L. E., & Berkson, G. (1997). Sensory defensiveness in persons with developmental disabilities. *Occupational Therapy Journal of Research*, 17(3), 173–185.
- Baranek, G. T., Reinhartsen, D., & Wannamaker, S. (2001). Play: Engaging children with autism. In R. Heubner (Ed.), *Sensorimotor interventions in autism* (pp. 311–351). Philadelphia: F. A. Davis.
- Baranek, G. T., Parham, L. D., & Bodfish, J. W. (2005). Sensory and motor features in autism: Assessment and intervention. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Assessment, interventions, and policy) (Vol. 2, 3rd ed., pp. 831–857). Hoboken: Wiley.
- Baranek, G. T., David, F. J., Poe, M. D., Stone, W. L., & Watson, L. R. (2006). Sensory Experiences Questionnaire: Discriminating sensory features in young children with autism, developmental delays, and typical development. *Journal of Child Psychology and Psychiatry*, 47(6), 591–601.
- Baranek, G. T., Roberts, J., David, F., Sideris, J., Mirrett, P., Hatton, D., et al. (2008). Developmental trajectories

- and correlates of sensory processing in young boys with fragile X syndrome. *Physical & Occupational Therapy in Pediatrics*, 28(1), 79–98.
- Baranek, G. T., Watson, L. R., Boyd, B. A., Poe, M., David, F. J., & McGuire, L. (2013). Hypo-responsiveness to social and nonsocial sensory stimuli in children with autism, children with developmental delays, and typically developing children. *Development and Psychopathology*, 25(2), 307–320.
- Baranek, G. T., Little, L. M., Parham, L. D., Ausderau, K. K., & Sabatos-DeVito, M. (2014). Sensory features in autism spectrum disorders. In F. R. Volkmar, R. Paul, S. J. Rogers, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders*, 4e (pp. 378–408). Hoboken: Wiley.
- Baranek, G. T., Watson, L. R., Turner-Brown, L., Field, S. H., Crais, E. R., Wakeford, L., Little, L. M., & Reznick, J. S. (2015). Preliminary efficacy of adapted responsive teaching for infants at risk for autism spectrum disorder in a community sample. *Autism Research and Treatment*. <https://doi.org/10.1155/2015/386951>. PMID: PMC4306223.
- Baranek, G. T., Carlson, M., Sideris, J., Kirby, A. V., Watson, L. R., Williams, K. L., & Bulluck, J. (2019). Longitudinal assessment of stability of sensory features in children with autism spectrum disorder or other developmental disabilities. *Autism Research*, 12(1), 100–111. <https://doi.org/10.1002/aur.2008>.
- Ben-Sasson, A., Hen, L., Fluss, R., Cermak, S. A., Engel-Yeger, B., & Gal, E. (2009). A meta-analysis of sensory modulation symptoms in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(1), 1–11.
- Boyd, B. A., Baranek, G. T., Sideris, J., Poe, M. D., Watson, L. R., Patten, E., et al. (2010). Sensory features and repetitive behaviors in children with autism and developmental delays. *Autism Research*, 3, 78–87.
- Dahlgren, S. O., & Gillberg, C. (1989). Symptoms in the first two years of life: A preliminary population study of infantile autism. *European Archives of Psychiatry and Neurological Science*, 238(3), 169–174.
- Dunn, W. (1997). Impact of sensory processing on the daily lives of young children and their families: A conceptual model. *Infants and Young Children*, 9(4), 23–35.
- Greenspan, S., & Wieder, S. (1997). Developmental patterns and outcomes in infants and children with disorders in relating and communicating: A chart review of 200 cases of children with autistic spectrum diagnoses. *Journal of Developmental and Learning Disorders*, 1(1), 87–141.
- Grzadzinski, R., Donovan, K., Truong, K., et al. (2020). Sensory reactivity at 1 and 2 years old is associated with ASD severity during the preschool years. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-020-04432-4>.
- Iarocci, G., & McDonald, J. (2006). Sensory integration and the perceptual experience of persons with autism. *Journal of Autism and Developmental Disorders*, 36(1), 77–90.
- Kientz, M. A., & Dunn, W. (1997). A comparison of the performance of children with and without autism on the sensory profile. *American Journal of Occupational Therapy*, 51(7), 530–537.
- Kirby, A. V., Little, L. M., Schultz, B., Watson, L. R., Zhang, W., & Baranek, G. T. (2016). Development and pilot of the caregiver strategies inventory. *The American Journal of Occupational Therapy: Official Publication of the American Occupational Therapy Association*, 70(4), 6.
- Little, L. M., Freuler, A. C., Houser, M. B., Guckian, L., Carbine, K., David, F. J., et al. (2011). Brief report: psychometric validation of the sensory experiences questionnaire. *American Journal of Occupational Therapy*, 65, 207–210.
- Lord, C. (1995). Follow-up of two-year-olds referred for possible autism. *Journal of Child Psychology and Psychiatry*, 36(8), 1365–1382.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(5), 659–685.
- Miller, L. J., Reisman, J. E., McIntosh, D. N., & Simon, J. (2001). An ecological model of sensory modulation: Performance of children with fragile X syndrome, autistic disorder, attention-deficit/hyperactivity disorder, and sensory modulation dysfunction. In S. Smith-Roley, E. I. Blanche, & R. Schaaf (Eds.), *Understanding the nature of sensory integration with diverse populations* (pp. 57–88). San Antonio: Therapy Skill Builders.
- Mundy, P., & Neal, R. (2001). Neural plasticity, joint attention, and a transactional social-orienting model of autism. In L. M. Glidden (Ed.), *International review of research in mental retardation* (Autism) (Vol. 23, pp. 139–168). San Diego: Academic Press.
- Ornitz, E. M., Guthrie, D., & Farley, A. H. (1977). The early development of autistic children. *Journal of Autism and Childhood Schizophrenia*, 7(3), 207–229.
- Ozonoff, S., South, M., & Provençal, S. (2005). Executive functions. In F. R. Volkmar, R. Paul, A. Klin, & Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Assessment, interventions, and policy) (Vol. 2, 3rd ed., pp. 606–627). Hoboken: Wiley.
- Rajendran, G., & Mitchell, P. (2007). Cognitive theories of autism. *Developmental Review*, 27, 224–260.
- Rogers, S. J., & Ozonoff, S. (2005). Annotation: What do we know about sensory dysfunction in autism? A critical review of the empirical evidence. *Journal of Child Psychology and Psychiatry*, 46, 1255–1268.
- Rogers, S. J., Hepburn, S., & Wehner, E. (2003). Parent reports of sensory symptoms in toddlers with autism and those with other developmental disorders. *Journal of Autism and Developmental Disorders*, 33(6), 631–642.

- Stone, W. L., & Hogan, K. L. (1993). A structured parent interview for identifying young children with autism. *Journal of Autism and Developmental Disorders*, 23(4), 639–652.
- Volkmar, F. R., Lord, C., Bailey, A., Schultz, R. T., & Klin, A. (2004). Autism and pervasive developmental disorders. *Journal of Child Psychology and Psychiatry*, 45(1), 135–170.
- Walz, N. C., & Baranek, G. T. (2006). Sensory processing patterns in persons with Angelman syndrome. *American Journal of Occupational Therapy*, 60(4), 472–479.
- Waterhouse, L., Fein, D., & Modahl, C. (1996). Neurofunctional mechanisms in autism. *Psychological Review*, 103(3), 457–489.
- Watson, L. R., Patten, E., Baranek, G. T., Poe, M. D., Boyd, B. A., Freuler, A. C., et al. (2011). Differential associations between sensory response patterns and language, social, and communication measures in children with autism or other developmental disabilities. *Journal of Speech, Language, and Hearing Research*, 54(6), 1562–1576.
- Watson, L. R., Crais, E. R., Baranek, G. T., Turner-Brown, L., Sideris, J., Wakeford, L., Kinard, J., Reznick, J. S., Martin, K. L., & Nowell, S. W. (2017). Parent-mediated intervention for one-year-olds screened as at-risk for autism spectrum disorder: A randomized controlled trial. *Journal of Autism and Developmental Disorders*, 47(11), 3520–3540. <https://doi.org/10.1007/s10803-017-3268-0>.

multisensory integration and sensorimotor functions such as motor planning (Brandwein et al. 2013; Foxe et al. 2013; Russo et al. 2010). Parents of children with ASD report that these sensory features are among the most challenging that they face in managing their children's behavior and their daily life (Schaaf et al. 2011). Parents rate intervention for sensory difficulties as one of their top priorities and most helpful interventions (Myers et al. 2009; Peacock 2012). Studies show that up to 95% of children with ASD exhibit these sensory features (Baker et al. 2008; Ben-Sasson et al. 2009) and that sensory features may help differentiate ASD from children with other clinical disorders (Baranek et al. 2006).

Despite the prevalence of sensory features in ASD and their potential impact on function and behavior, there is a lack of consensus regarding the value of sensory features in the autism phenotype. Some argue that sensory features have important diagnostic value, whereas others suggest that they are not a distinguishing characteristic and thus should not be included in the DSM-5 as a diagnostic characteristic. This chapter will explore sensory features in ASD with a focus on their value as a core phenotypic marker of ASD.

Sensory Features as a Marker of Autism Spectrum Disorders

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Definition

Sensory features are included as one of the diagnostic markers of autism spectrum disorder (ASD) under the restrictive and repetitive behaviors, interests, or activities diagnostic criteria in the DSM-5 (American Psychiatric Association [APA] 2013). Sensory features are defined as hypo- and hyper-reactivity to sensation and unusual interests in sensory aspects of the environment in the DSM-5 but have also been shown to include difficulties with

Historical Background

Sensory features were first noted as part of the autism phenotype by Kanner in 1943. Later, Wing (1969) argued that sensory abnormalities are an underlying impairment in autism. Thus, in the early years of autism, sensory features were considered a key aspect of the autism phenotype. However, as more studies were conducted on the cognitive and social aspects of autism, a more social cognition view of autism became predominant in the field (e.g., Rutter 1983). This view purported that cognition and language impairments are the core features of ASD and sensory symptoms were viewed as by-products of primary deficits in social cognition. Based on this perspective, sensory features were subsequently removed from the DSM as a key diagnostic marker of ASD. Over the subsequent years, the key role of sensory

features in ASD has been debated. In 2013, findings related to the prevalence and ubiquitous nature of sensory features in ASD resulted in sensory features again appearing in the DSM-5 (APA 2013). Sensory features are now a component of the ASD diagnostic criteria in the DSM-5 under the restricted and repetitive behaviors' diagnostic marker. They are included as one of the four potential markers of the restrictive and repetitive behaviors, interests, or activities diagnostic criteria as shown below:

Restricted and repetitive patterns of behaviors, interests, or activities as manifested by at least two of the four symptoms (APA 2013):

- Stereotyped or repetitive speech, motor movements, or use of objects
- Excessive adherence to routines, ritualized patterns of verbal or nonverbal behavior, or excessive resistance to change
- Highly restricted, fixated interests that are abnormal in intensity or focus
- **Hyper- or hyporeactivity to sensory input or unusual interest in sensory aspects of the environment**

Current Knowledge

Sensory Features as a Diagnostic Marker of ASD

As stated above, there is a continued debate about whether sensory features are a distinguishing diagnostic feature of ASD. In support of their value as a diagnostic marker, Robertson and Baron-Cohen (2017) find strong evidence that sensory symptoms are a “core, primary characteristic of the neurobiology of autism, and that sensory differences are a core phenotypic marker of ASD” (p. 674). They base this conclusion on the evidence that sensory symptoms are present early in development (as early as 6 months of age), are predictive of later diagnostic status, affect every sensory modality, and are estimated to occur in as many as 90% of autistic individuals (as cited in Robertson and Baron-Cohen 2017). Genetic and neuroimaging studies (summarized below) also provide additional evidence of the unique contribution of sensory features to the autism phenotype.

Genetic evidence suggests that there is some genetic heritability of sensory features in ASD with greater sensory symptoms in families that are believed to have a higher genetic inclination for autism (multiplex families) than in families with a single individual diagnosed with autism (simplex families). Further, there is some evidence showing that parents and siblings of individuals with ASD show higher levels of self-reported sensory traits relative to the general population (Donaldson et al. 2017). Research showing that sensory features may distinguish those with ASD from other genetically based neurodevelopmental conditions supports this position. For example, Bizzell et al. (2019) found that sensory features differentiate ASD from other clinical disorders in a genetically homogeneous group of boys with 46,XXX syndrome. Thirty percent of these boys with XXX meet the criteria for ASD, and those with XXX + ASD showed sensory features comparable to boys with ASD. This finding suggests that sensory features may be a distinguishing characteristic for the ASD phenotype. Further, Robertson and Baron-Cohen (2017) suggest that data showing that sensory patterns in neurodevelopmental disorders such as Rhetts syndrome and dyslexia are distinctly different than those with ASD support the view that sensory features may serve as a “selective, objective marker of autism” (p. 676).

Further supporting the view that sensory features are a key distinguishing characteristic of ASD is neurological evidence. For example, imaging studies show alterations in sensory-dedicated neural circuitry at the molecular and anatomical levels in primary sensory regions of the brain associated with sensory features in autism and that these findings are primary rather than secondary consequences of alterations in higher-order cognitive processes. Robertson and Baron-Cohen (2017) summarize these findings and conclude that the pattern of findings, regarding atypical sensory responses in primary sensory cortices as well as the alterations in the functional architecture of sensory cortex, is compatible with the hypothesis that sensory differences are a core phenotypic marker of autism. Another key finding here is that there is disruption in the excitatory-

inhibitory balance in the brain of individuals with ASD that may lead to differences in the perception and processing of sensory information and that difference in GABA may mediate these differences (Robertson and Baron-Cohen 2017).

In contrast, some researchers suggest that sensory features are not a phenotypic characteristic of ASD. For example, Grapel et al. (2015) found that adolescents and adults with ASD (including PDD) did not show differences in parent-reported sensory-related issues in comparison to others with developmental or behavioral difficulties (not ASD). They question the rationale for including them as a diagnostic criterion for ASD: While sensory symptoms are “relatively common in ASD, they do not discriminate autism nearly as strongly as the core social dysfunction and they are frequent in other developmental problems” (p. 71). They conclude that sensory features are not a marker of ASD and question the rationale for including them as a diagnostic criterion for ASD. In support of this view, McCormick et al. (2016) conducted a longitudinal study comparing sensory symptoms in ASD, developmental delay, and typically developing children ages 2–8 ($n = 79$) at three time points and found that there were not significant differences in sensory symptoms between the two clinical groups (ASD and DD). This finding supports the notion that sensory symptoms are not a diagnostic feature of ASD. One confound to this study is that they used the Short Sensory Profile (McIntosh et al. 1999) to evaluate sensory features which is biased toward reporting hyper-reactivity over other types of sensory features.

Although this evidence is persuasive, it is confounded by age, autism severity, and assessment strategies used to identify sensory features. Regarding age, given that there is evidence that sensory features are stable through the preschool years (from ages 3 to 6) but then tend to decrease with age, the age range included in a given study will impact findings. Further, severity of autism tends to impact sensory symptoms, whereby the greater severity of ASD is associated with great sensory features (Leekam et al. 2007). Another confound to these findings is that terminology patterns have varied across studies and, over the years, as have assessments used to identify

sensory features making it challenging to compare findings. Finally, few longitudinal studies have tracked sensory features in ASD over time which further limits the ability to interpret the data and make strong conclusions about the role of sensory features in the autism phenotype.

Early Sensory Symptoms Impact the Neurodevelopmental Trajectory

Regardless of their unique contribution to the autism phenotype, it is clear that sensory features impact future development. One promising area of research is the idea that early, atypical sensory processing and integration has cascading effects on social, communicative, and emotional development (Hannant et al. 2016). Studies suggest that low-level sensory-perceptual alterations (i.e., perception of sensory cues) may lead to downstream effects such as altered neural connectivity that impact higher-level deficits in cognition, language, praxis, and socialization in individuals with ASD (Cascio et al. 2016). A central tenet of this line of investigation is that early atypical sensory perceptions and experiences set the stage for an abnormal neurodevelopmental trajectory that further affects development and elaboration of functional brain networks that are key in autism symptomatology. Future work in this area will likely provide insight into the unique contributions of early sensory processing, perception, and integration on the autism neurodevelopmental trajectory and subsequently on the expression of autism behaviors.

Relationship of Sensory Features to Autism Severity and Other Symptoms

Additional evidence for the impact of sensory features in ASD comes from data showing that sensory features influence other ASD behaviors. Sensory features are shown to predict deficits in social and cognitive function and explain independent variance in social and communication symptoms in diagnostic assessments (Frazier et al. 2012; Turner-Brown et al. 2013). Further, sensory features are predictive of social deficits and repetitive behaviors (Boyd et al. 2010) as well as eventual diagnostic status (Donaldson et al. 2017). Large-scale studies demonstrate covariance between questionnaire-based measures of

sensory sensitivities and autistic traits (see, e.g., Mayer (2017)). These correlations present a strong argument for a relationship between sensory and social-cognitive processing in autism.

Further, sensory symptoms are associated with decreased participation in daily living and leisure activities for children with ASD and their families (Schaaf et al. 2011) as well as caregiver strain (Kirby et al. 2019). Dunn et al. (2016) conducted a scoping review to examine the impact of sensory features on daily living in children. They found a link between sensory processing (measured by a variety of sensory measures) and participation in many areas including daily living skills, play/leisure activities, and social participation. Although this study was not specific to ASD, it shows the potential impact of sensory features on daily lives. Similarly, Jasmin et al. (2009) found high correlations between sensorimotor deficits and daily living skills in ASD.

To explore the impact of intervention designed to address sensory features on daily life function and participation in children with ASD, Schaaf et al. (2014) conducted an intervention study designed to assess the impact of occupational therapy intervention using Ayres Sensory Integration[®] on functional skills. They found that Ayres Sensory Integration[®] intervention improved function and participation in children with ASD in those who received the intervention in comparison to those who continued treatment as usual (Schaaf et al. 2014). This study provided the first solid evidence that an intervention targeting sensory features had a value for improving function and participation in children with ASD and resulted in Ayres Sensory Integration[®] meeting the criteria for an evidence-based intervention for ASD (Schoen et al. 2019). Currently, Molholm, Schaaf, Foxe, and colleagues are conducting a large, NIH-funded comparative effectiveness trial comparing ASI and behavioral intervention/discrete trial training on functional skills (1R01HD082814-01A1). This trial is underway and will provide further insight into the value of interventions that target sensory features and their impact on function and participation. Further, this study will evaluate the impact of the interventions on sensory features directly,

including neural and behavioral markers, an area that has not been investigated but that is needed.

Sensory Subtypes in ASD

Given that sensory features in ASD are heterogeneous and ubiquitous, the identification of specific subtypes of sensory features in ASD holds a promise for reducing heterogeneity and for guiding future research and tailoring interventions. However, it is likely that subtypes of sensory features will vary by person, by age, and by severity of autism. However, to date, researchers have not found a consistent set of subtypes of sensory features that are universal to ASD. Rather the data show that sensory features are heterogeneous. The interpretation of this literature is complicated by variations in terminology, differences in operational definitions of sensory features, differences in assessment measures used to classify sensory features, variability in the statistical procedures used to subtype data (e.g., latent class analysis, factor analysis, cluster analysis), as well as the age group of the sample. Common patterns identified include hypo-reactivity (decreased or delayed response to sensory inputs); hyper-reactivity (exaggerated response to sensory stimuli); and sensory seeking behaviors, also termed extreme sensory interests or repetitive sensory behaviors (fascination with or physically active behaviors to obtain sensory input) (APA 2013). Subgroups of sensory features also include enhanced perception (superior sensory acuity)/decreased sensory perception (lack of sensory acuity); mild to severe sensory features (Ausderau et al. 2014); hypo-, hyper-, and multisensory patterns (Lane et al. 2014); and uniformly elevated sensory symptoms or raised sensory avoiding and sensitivity using the Short Sensory Profile-2 (Simpson et al. 2019). Thus, the subtyping effort has not yielded specific universally accepted patterns, but rather an acceptance that sensory features are heterogeneous and that further efforts to identify subtypes may be useful to tailor interventions more specifically to the sensory needs of each individual and to guide future research into the sensory features of ASD.

Studies of Specific Sensory Modalities in ASD

Researchers have found sensory differences in many sensory systems in ASD, as well as

differences in multisensory integration. Findings point to both reduced and enhanced perception and reactivity, with personal accounts often differing from physiological or neurological findings. For example, studies of auditory processing have shown reduced temporal processing and delayed responses (Kwakye et al. 2011), whereas first-person accounts often report heightened sensitivity and avoidance responses (Grandin and Panek 2013). Studies of tactile processing have shown both superior (Blakemore et al. 2006) and reduced (Puts et al. 2014; Tavassoli et al. 2016; Marco et al. 2012) tactile threshold detection (see Mikkelsen et al. (2018) for a review). However, more recent evidence from Puts' lab is showing that tactile reactivity may be related to an attentional issue rather than autism. They showed that children with ASD have normal reaction times to tactile stimuli but that children with ASD + ADHD and ADHD only have slower reaction times (unpublished data). This finding suggests that ADHD may be the confounding factor regarding tactile detection thresholds in ASD. Further, Puts' team is showing evidence of abnormal sensory gating in ASD suggesting impaired feedforward inhibition such that children with ASD do not show the typical suppression (gating) of tactile stimuli following subthreshold stimuli (Puts et al. 2017). These findings show a promise in terms of understanding the mechanisms of sensory features in ASD and furthering our understanding of the unique aspects of sensory features in ASD.

In the area of proprioception, Mostofsky and Ewen (2011) show evidence of enhanced reliance on proprioceptive cues for movement in motor learning tasks, while Smith-Roley et al. (2015) showed signs of reduced proprioceptive perception in behavioral tasks designed to measure proprioceptive awareness, perception, and accuracy.

Visual processing and perception in ASD is characterized by strength in detail focused visual percept and difficulty with global percept (Robertson and Baron-Cohen 2017). Further, there appears to be faster detection of single visual targets in a cluttered visual display among distractors; greater detection or discrimination thresholds for static stimuli with deficits in visual motion processing (ability to identify global

direction of a visual signal). These deficits in visual motion are predictive of severity of autism symptoms.

To test the hypothesis that differences in visual perception and processing in ASD are related to lack of excitation/inhibition balance, Robertson conducted binocular rivalry experiments. Binocular rivalry is a "basic visual function [experiment] that depends on the strength of inhibitory interactions in the visual cortex" (Robertson and Baron-Cohen 2017, p. 676). Binocular rivalry tests the brain's ability to suppress alternating back and forth perception in each eye in the visual cortex. They found that persons with ASD show reduced strength of perceptual suppression and this deficit is associated with reduced GABAergic action in early visual cortex.

Pain – There have been reports of both hypo- and hyperresponses to pain in ASD (Grandin and Panek 2013; Yasuda et al. 2016). Failla et al. (2018) found an intact early response to pain in adults with ASD with a reduction in response as the stimulation continued. They suggest that alterations in pain perception reported by individuals with ASD may be due to altered coping strategies or top-down modulation of sustained pain; however, this hypothesis needs to be further studied and validated and points to the need to study both immediate and sustained reactions of pain but also of all sensory modalities.

In summary, the data on single sensory modalities processing in ASD has not reached consensus although there appears to be an emerging theme of lack of inhibition or modulation/gating of sensory input in ASD that underlies hyper-reactivity to sensation in the tactile and visual systems. Further, there is some evidence that reduced GABA may be the mechanism underlying hyper-reactivity, but more research is needed to confirm this hypothesis. There is very little research on sensory perception in ASD as there appears to be a tendency to study sensory reactivity over perception, and more research is needed in the area of perception and discrimination of sensation in ASD. The nature and extent of sensory features in ASD has proven to be a challenging area of research that requires more attention and focus in ASD (Grandin and Panek 2013).

Multisensory Integration

There is evidence that multisensory integration, the ability to perceptually bind two or more sensory inputs in time and space, is altered in ASD. Studies show that the temporal binding of two concurrent sensations from different sensory modalities (i.e., auditory and visual or visual and somatosensory) is slower with an elongated temporal binding window (Stevenson et al. 2016; Wallace and Stevenson 2014; Brandwein et al. 2013). This delay in temporal binding impacts the ability to integrate sensory stimuli in “real” time. Functionally, this lack of multisensory integration may manifest as decreased or slow to respond to multisensory inputs. Given that most activities in the everyday world require multisensory integration, it is easy to imagine the impact that decreased multisensory integration would have on function and behavior. Responses may be slow, absent, or unusual. Delay in multisensory integration is a highly replicated finding, and it has been associated with autism severity (Brandwein et al. 2013). However, its ability to distinguish autism from other clinical disorders has not been established.

Future Directions

Further research is needed to fully understand sensory features in ASD and their contribution to the autism phenotype. Research should extend beyond sensory reactivity (hypo-hyper) to include sensory perception and integration. Research extending the concepts of detail versus global perception, central coherence, and immediate versus sustained responses is needed across all sensory systems. Further exploration of sensory gating across sensory systems will also shed light on the potential mechanisms of hypo- and hyper-reactivity in ASD and the potential role of GABA in sensory reactivity. Further, to allow for comparison of studies across study sites and protocol, a specific assessment protocol that has universal acceptability and that combine performance-based and proxy report (Schaaf and Lane 2015) will improve our understanding of sensory features in ASD and allow for more

accurate subtyping to reduce heterogeneity and improve precision in research and practice.

See Also

- ▶ Autism Spectrum Disorder
- ▶ Autistic Disorder
- ▶ DSM-5
- ▶ DSM-IV

References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: APA Press.
- Ausderau, K. K., Furlong, M., Sideris, J., Bulluck, J., Little, L. M., Watson, L. R., & Baranek, G. T. (2014). Sensory subtypes in children with autism spectrum disorder: Latent profile transition analysis using a national survey of sensory features. *Journal of Child Psychology and Psychiatry*, 55(8), 935–944.
- Baker, A. E. Z., Lane, A., Angley, M. T., & Young, R. L. (2008). The relationship between sensory processing patterns and behavioural responsiveness in autistic disorder: A pilot study. *Journal of Autism and Developmental Disorders*, 38(5), 867–875. <https://doi.org/10.1007/s10803-007-0459-0>.
- Baranek, G. T., David, F. J., Poe, M. D., Stone, W. L., & Watson, L. R. (2006). Sensory experiences questionnaire: Discriminating sensory features in young children with autism, developmental delays and typical development. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 47(6), 591–601. <https://doi.org/10.1111/j.1469-7610.2005.01546.x>.
- Ben-Sasson, A., Hen, L., Fluss, R., Cermak, S. A., Engel-Yeger, B., & Gal, E. (2009). A meta-analysis of sensory modulation symptoms in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(1), 1–11. <https://doi.org/10.1007/s10803-008-0593-3>.
- Bizzell, E., Ross, J., Rosenthal, C., Dumont, R., & Schaaf, R. (2019). Sensory features as a marker of autism spectrum disorders. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03948-8>.
- Blakemore, S.-J., Tavassoli, T., Calò, S., Thomas, R. M., Catmur, C., Frith, U., & Haggard, P. (2006). Tactile sensitivity in Asperger syndrome. *Brain and Cognition*, 61(1), 5–13. <https://doi.org/10.1016/j.bandc.2005.12.013>.
- Boyd, B. A., Baranek, G. T., Sideris, J., Poe, M. D., Watson, L. R., Patten, E., & Miller, H. (2010). Sensory features and repetitive behaviors in children with autism and developmental delays. *Autism Research*:

- Official Journal of the International Society for Autism Research*, 3(2), 78–87. <https://doi.org/10.1002/aur.124>.
- Brandwein, A. B., Foxe, J. J., Butler, J. S., Russo, N. N., Altschuler, T. S., Gomes, H., & Molholm, S. (2013). The development of multisensory integration in high-functioning autism: High-density electrical mapping and psychophysical measures reveal impairments in the processing of audiovisual inputs. *Cerebral Cortex*, 23(6), 1329–1341. <https://doi.org/10.1093/cercor/bhs109>.
- Cascio, C. J., Woyanowski, T., Baranek, G. T., & Wallace, M. T. (2016). Toward an interdisciplinary approach to understanding sensory function in autism spectrum disorder. *Autism Research: Official Journal of the International Society for Autism Research*, 9(9), 920–925. <https://doi.org/10.1002/aur.1612>.
- Donaldson, C. K., Stauder, J. E. A., & Donkers, F. C. L. (2017). Increased sensory processing atypicalities in parents of multiplex ASD families versus typically developing and simplex ASD families. *Journal of Autism and Developmental Disorders*, 47(3), 535–548. <https://doi.org/10.1007/s10803-016-2888-0>.
- Dunn, W., Little, L., Dean, E., Robertson, S., & Evans, B. (2016). The state of the science on sensory factors and their impact on daily life for children: A scoping review. *OTJR: Occupation, Participation and Health*, 36(2 Suppl), 3S–26S. <https://doi.org/10.1177/1539449215617923>.
- Failla, M. D., Moana-Filho, E. J., Essick, G. K., Baranek, G. T., Rogers, B. P., & Cascio, C. J. (2018). Initially intact neural responses to pain in autism are diminished during sustained pain. *Autism: The International Journal of Research and Practice*, 22(6), 669–683. <https://doi.org/10.1177/1362361317696043>.
- Foxe, J. J., Molholm, S., Del Bene, V. A., Frey, H. P., Russo, N. N., Blanco, D., et al. (2013). Severe multisensory speech integration deficits in high-functioning school-aged children with autism spectrum disorder (ASD) and their resolution during early adolescence. *Cerebral Cortex*, 25(2), 298–312.
- Frazier, T. W., Youngstrom, E. A., Speer, L., Embacher, R., Law, P., Constantino, J., et al. (2012). Validation of proposed DSM-5 criteria for autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(1), 28–40.e3. <https://doi.org/10.1016/j.jaac.2011.09.021>.
- Grandin, T., & Panek, R. (2013). *The autistic brain: Thinking across the spectrum*. New York: Houghton Mifflin Harcourt Publishing.
- Grapel, J. N., Cicchetti, D. V., & Volkmar, F. R. (2015). Sensory features as diagnostic criteria for autism: Sensory features in autism. *The Yale Journal of Biology and Medicine*, 88(1), 69–71.
- Hannant, P., Cassidy, S., Tavassoli, T., & Mann, F. (2016). Sensorimotor difficulties are associated with the severity of autism spectrum conditions. *Frontiers in Integrative Neuroscience*, 10, 28. <https://doi.org/10.3389/fnint.2016.00028>.
- Jasmin, E., Couture, M., McKinley, P., Reid, G., Fombonne, E., & Gisel, E. (2009). Sensori-motor and daily living skills of preschool children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(2), 231–241. <https://doi.org/10.1007/s10803-008-0617-z>.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kirby, A. V., Williams, K. L., Watson, L. R., Sideris, J., Bulluck, J., & Baranek, G. T. (2019). Sensory features and family functioning in families of children with autism and developmental disabilities: Longitudinal associations. *The American Journal of Occupational Therapy*, 73(2), 7302205040p1–7302205040p14. <https://doi.org/10.5014/ajot.2018.027391>.
- Kwakye, L. D., Foss-Feig, J. H., Cascio, C. J., Stone, W. L., & Wallace, M. T. (2011). Altered auditory and multisensory temporal processing in autism spectrum disorders. *Frontiers in Integrative Neuroscience*, 4, 129. <https://doi.org/10.3389/fnint.2010.00129>.
- Lane, A. E., Molloy, C. A., & Bishop, S. L. (2014). Classification of children with autism spectrum disorder by sensory subtype: A case for sensory-based phenotypes. *Autism Research*, 7(3), 322–333. <https://doi.org/10.1002/aur.1368>.
- Leekam, S. R., Nieto, C., Libby, S. J., Wing, L., & Gould, J. (2007). Describing the sensory abnormalities of children and adults with autism. *Journal of Autism and Developmental Disorders*, 37(5), 894–910. <https://doi.org/10.1007/s10803-006-0218-7>.
- Marco, E. J., Khatibi, K., Hill, S. S., Siegel, B., Arroyo, M. S., Dowling, A. F., et al. (2012). Children with autism show reduced somatosensory response: An MEG study. *Autism Research: Official Journal of the International Society for Autism Research*, 5(5), 340–351. <https://doi.org/10.1002/aur.1247>.
- Mayer, J. L. (2017). The relationship between autistic traits and atypical sensory functioning in neurotypical and ASD adults: A spectrum approach. *Journal of Autism and Developmental Disorders*, 47(2), 316–327. <https://doi.org/10.1007/s10803-016-2948-5>.
- McCormick, C., Hepburn, S., Young, G. S., & Rogers, S. J. (2016). Sensory symptoms in children with autism spectrum disorder, other developmental disorders and typical development: A longitudinal study. *Autism: The International Journal of Research and Practice*, 20(5), 572–579. <https://doi.org/10.1177/1362361315599755>.
- McIntosh, D. N., Miller, L. J., Shyu, V., & Dunn, W. (1999). *Overview of the short sensory profile sensory profile—User's manual*. Minneapolis: Pearson.
- Mikkelsen, M., Wodka, E. L., Mostofsky, S. H., & Puts, N. A. J. (2018). Autism spectrum disorder in the scope of tactile processing. *Developmental Cognitive Neuroscience*, 29, 140–150. <https://doi.org/10.1016/j.dcn.2016.12.005>.
- Mostofsky, S. H., & Ewen, J. B. (2011). Altered connectivity and action model formation in autism is autism. *The Neuroscientist*, 17(4), 437–448. <https://doi.org/10.1177/1073858410392381>.
- Myers, B. J., Mackintosh, V. H., & Goin-Kochel, R. P. (2009). “My greatest joy and my greatest heart ache:”

- Parents' own words on how having a child in the autism spectrum has affected their lives and their families' lives. *Research in Autism Spectrum Disorders*, 3(3), 670–684. <https://doi.org/10.1016/j.rasd.2009.01.004>.
- Peacock, E. (2012). Top 8 autism therapies – Reported by parents|autism speaks. Retrieved from <https://www.autismspeaks.org/blog/top-8-autism-therapies-reported-parents>.
- Puts, N. A. J., Wodka, E. L., Tommerdahl, M., Mostofsky, S. H., & Edden, R. A. E. (2014). Impaired tactile processing in children with autism spectrum disorder. *Journal of Neurophysiology*, 111(9), 1803–1811. <https://doi.org/10.1152/jn.00890.2013>.
- Puts, N. A., Wodka, E. L., Harris, A. D., Crocetti, D., Tommerdahl, M., Mostofsky, S. H., & Edden, R. A. (2017). Reduced GABA and altered somatosensory function in children with autism spectrum disorder. *Autism Research*, 10(4), 608–619. <https://doi.org/10.1002/aur.1691>.
- Robertson, C. E., & Baron-Cohen, S. (2017). Sensory perception in autism. *Nature Reviews. Neuroscience*, 18(11), 671–684. <https://doi.org/10.1038/nrn.2017.112>.
- Roley, S. S., Mailloux, Z., Parham, L. D., Schaaf, R. C., Lane, C. J., & Cermak, S. (2015). Sensory integration and praxis patterns in children with autism. *American Journal of Occupational Therapy*, 69(1), 6901220010p1–6901220010p8. <https://doi.org/10.5014/ajot.2015.012476>.
- Russo, N., Foxe, J. J., Brandwein, A. B., Altschuler, T., Gomes, H., & Molholm, S. (2010). Multisensory processing in children with autism: High-density electrical mapping of auditory-somatosensory integration. *Autism Research: Official Journal of the International Society for Autism Research*, 3(5), 253–267. <https://doi.org/10.1002/aur.152>.
- Rutter, M. (1983). Cognitive deficits in the pathogenesis of autism. *Journal of Child Psychology and Psychiatry*, 24, 513–531.
- Schaaf, R. C., & Lane, A. E. (2015). Toward a best-practice protocol for assessment of sensory features in ASD. *Journal of Autism and Developmental Disorders*, 45(5), 1380–1395. <https://doi.org/10.1007/s10803-014-2299-z>.
- Schaaf, R. C., Toth-Cohen, S., Johnson, S. L., Outten, G., & Benevides, T. W. (2011). The everyday routines of families of children with autism: Examining the impact of sensory processing difficulties on the family. *Autism: The International Journal of Research and Practice*, 15(3), 373–389. <https://doi.org/10.1177/1362361310386505>.
- Schaaf, R. C., Benevides, T., Mailloux, Z., Faller, P., Hunt, J., van Hooydonk, E., et al. (2014). An intervention for sensory difficulties in children with autism: A randomized trial. *Journal of Autism and Developmental Disorders*, 44(7), 1493–1506. <https://doi.org/10.1007/s10803-013-1983-8>.
- Schoen, S. A., Lane, S. J., Mailloux, Z., May-Benson, T., Parham, L. D., Smith Roley, S., & Schaaf, R. C. (2019). A systematic review of ayres sensory integration intervention for children with autism. *Autism Research: Official Journal of the International Society for Autism Research*, 12(1), 6–19. <https://doi.org/10.1002/aur.2046>.
- Simpson, K., Adams, D., Alston-Knox, C., Heussler, H. S., & Keen, D. (2019). Exploring the sensory profiles of children on the autism spectrum using the Short Sensory Profile-2 (SSP-2). *Journal of Autism and Developmental Disorders*, 49(5), 2069–2079. <https://doi.org/10.1007/s10803-019-03889-2>.
- Stevenson, R. A., Segers, M., Ferber, S., Barense, M. D., Camarata, S., & Wallace, M. T. (2016). Keeping time in the brain: Autism spectrum disorder and audiovisual temporal processing. *Autism Research*, 9(7), 720–738.
- Tavassoli, T., Bellesheim, K., Tommerdahl, M., Holden, J. M., Kolevzon, A., & Buxbaum, J. D. (2016). Altered tactile processing in children with autism spectrum disorder. *Autism Research: Official Journal of the International Society for Autism Research*, 9(6), 616–620. <https://doi.org/10.1002/aur.1563>.
- Turner-Brown, L. M., Baranek, G. T., Reznick, J. S., Watson, L. R., & Crais, E. R. (2013). The first year inventory: A longitudinal follow-up of 12-month-old to 3-year-old children. *Autism: The International Journal of Research and Practice*, 17(5), 527–540. <https://doi.org/10.1177/1362361312439633>.
- Wallace, M. T., & Stevenson, R. A. (2014). The construct of the multisensory temporal binding window and its dysregulation in developmental disabilities. *Neuropsychologia*, 64, 105–123.
- Wing, L. (1969). The handicaps of autistic children—A comparative study. *Journal of Child Psychology and Psychiatry*, 10(1), 1–40.
- Yasuda, Y., Hashimoto, R., Nakae, A., Kang, H., Ohi, K., Yamamori, H., et al. (2016). Sensory cognitive abnormalities of pain in autism spectrum disorder: A case-control study. *Annals of General Psychiatry*, 15, 8. <https://doi.org/10.1186/s12991-016-0095-1>.

Sensory History

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Definition

As part of a comprehensive occupational therapy evaluation for a child with underlying sensory processing challenges, the examiner may perform standardized assessments to evaluate motor performance and motor planning abilities. Using such instruments at the SIPT (Sensory Integration and Praxis Tests), the examiner will be able to identify additional deficits in sensory processing.

The examiner will also obtain a sensory history with the caregivers or client to evaluate the impact of sensory processing deficits on activities of daily living, leisure skills, and social interactions. This will allow the examiner to more fully understand sensory processing abilities and behavioral consequences to proceed with an intervention plan appropriate to the individual.

See Also

- [Evaluation of Sensory Processing](#)
- [Sensory Experiences Questionnaire](#)
- [Sensory Integration and Praxis Test](#)
- [Sensory Processing](#)
- [Tactile Defensiveness](#)
- [Touch Sensitivity](#)

References and Reading

- Mailloux, Z., & Smith Roley, S. (2011). Sensory integration. In H. M. Kuhaneck & R. Watling (Eds.), *Autism: A comprehensive occupational therapy approach* (pp. 469–507). Bethesda: AOTA Press.
- Tomchek, S. D. (2010). Sensory processing in individuals with an autism spectrum disorder. In H. M. Kuhaneck & R. Watling (Eds.), *Autism: A comprehensive occupational therapy approach* (pp. 135–161). Bethesda: AOTA Press.

Sensory Impairment

- [Deaf-Blind](#)

Sensory Impairment in Autism

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Definition

Sensory impairments are used in two ways in the context of autism. They may indicate a dual

disability of autism plus a sensory impairment as, for example, autism and visual impairment, autism and hearing loss, or autism and a sensory-motor impairment. There is nothing in the etiology of autism that protects against comorbidities, and clearly the sensory impairment will interact with the autism to produce a person-specific range of difficulties and a particular developmental path. Individuals with dual or even multiple diagnoses of this kind will not only have more difficulties to overcome but may have fewer compensatory strategies available to them. In addition, it will be harder to find appropriate support in education, social care, and vocational training, where key staff need to be knowledgeable about autism as well as the sensory impairment.

However, a separate sensory disorder is fairly uncommon in autism. The term “sensory impairment” is more commonly used to apply to sensory issues that many believe are central to autism itself. These are sensory processing problems, which are not severe enough to reach criteria as a separate sensory disorder. Sensory processing disorder (SPD) is currently still not accepted as a separate diagnostic category in its own right in DSM V (APA 2013) so it still cannot be considered as a co-morbidity in autism. Nevertheless, associated sensory features were accepted as part of the autism diagnosis in DSM-V with two broad characterizations of ‘sensory seeking’ (sensory under-responsivity) versus ‘sensory avoiding’ (sensory over-responsivity) being used as part of the characterization of the child at diagnosis. Sensory processing differences have been identified with autism since it was first categorized, but they have never before featured in the diagnostic criteria. Although there has been growing research interest in this issue, and the recognition of the key role sensory processing problems may have in autism (Baker et al. 2008), it had been the consensus that sensory problems were neither universal in, nor unique to, autism.

All forms of sensory input processing have been suggested as being impaired in autism, although only a limited range of senses has been studied. The nature of the difficulties has been defined as hyposensitivity or hypersensitivity in particular sensory domains although there are also

said to be issues with the latency and timing of responses to sensory input. The most studied have been auditory and visual processing, although some research suggests the proximal senses (touch, taste, smell) and motor senses are most affected in autism. There has also been considerable debate about the existence of a problem with multisensory integration in autism. Pain is a sense, the processing of which is also said to be disturbed in autism, and clearly this is very important in development and learning. However, there has been little research and some equivocal results.

There are a plethora of anecdotal reports of sensory processing difficulties in autism but not a great deal of scientific evidence. One key issue is to establish whether there are sensory processing difficulties that are universal in, and/or unique to, autism and, if so, to investigate the nature and possible causes for them. It is important, for the understanding of autism, to know whether sensory issues are prime, leading to other problems, for example, in perception, or whether they are secondary to other problems such as in attention. It is also important to understand these difficulties in order to develop the most helpful remediation and compensation strategies and to evaluate them.

Historical Background

Dual Autism Plus Sensory Impairment Diagnoses

Hearing Impairment The sensory processing problems associated with autism sometimes mean that infants may be unresponsive to sounds – especially social sounds like speech. As a result, it is common for hearing impairment to be first investigated as a possible cause for the differences in development noted by parents. This may arise from a genuine confusion of the symptoms of autism with deafness or it may be a simple precaution to rule out this sensory impairment before an autism diagnosis. If the child is found to have a significant hearing impairment but also has autism, clinicians and parents may believe the hearing loss is the cause of all the child's problems, and it may be many years before the dual

disability is fully acknowledged. This can mean that the child misses out on the most appropriate forms of education and support. The teaching of communication forms such as sign or symbol language may be less successful than in children with a sole hearing impairment because parents and staff have not recognized that the child will not learn to communicate just by being given the means to do so; in autism, the child needs to be taught directly about communication first. In such situations, it is common for the child to become frustrated and to develop secondary problems of social isolation and challenging behavior, making developmental progression even more problematic.

As long as the autism is recognized, and strategies put in place to teach and support the child in "autism-friendly" ways, there are many symbiotic features in hearing impairment and autism that mean that placement in services for the hearing impaired can be successful for the child with the dual problem. In countries like the UK, schools for the deaf, and peripatetic teachers of the deaf, were one of the earliest special educational services developed, and parents get regular in-home support. The growth of inclusion has been very successful for most children with hearing impairment, however, and cochlear implants have meant many now receive education through oral means. Much support has switched to mainstream schools, where the majority of such children are now educated. The children who have been least successfully integrated, and remain in the special schools, are those with multisensory impairments and comorbid conditions. Increasing numbers of children in these schools now have autism. Initially, they were children who had dual diagnoses, but, as schools developed expertise in working with children with autism, numbers of children with autism alone attend these schools. Staff expertise in using visual methods of instruction, having a clear and calm structure and concentrating on teaching communication, have all been helpful for these pupils with autism.

Visual Impairment The symptoms of autism are often missed in children with the dual disability because of some overlap in the development of autism and blindness. In children who are

congenitally blind, it is clear that visual social signals (like eye contact, visual joint attention, imitative smiling, and facial expressions) will be absent. This can lead to an asocial pattern of development that is akin to that of autism (Pring 2004), and even the repetitive stereotypies are often seen as what are called “blindisms.” Children who are just blind, however, will develop compensatory behaviors such as auditory and tactile joint attention, but this does not happen if they also have autism. Once again, services for the visually impaired have been the earliest to develop in many countries because the differences in development are marked and it is clear that parents and teachers will need knowledge, training, and support to help the child develop in the best way for him/her. No one expects the blind child to act as if she/he can see; it is accepted that education and other services must adapt to the needs of the child.

In that way, the child with the dual disability of autism and visual impairment in a setting for the visually impaired will at least have the advantage of staff who accept the difference and foster the child's attempts to do what best suits him/her, but in other respects, this is not a good environment for the child with autism. Auditory processing and tactile difficulties mean that most children with autism learn best from visually mediated structure and do not do well where touch guidance or verbal instruction (speech) is used, without visual support. Specific (e.g., nonsocial tactile) alternatives to visual mediation need to be developed. Technological aids are beginning to transform the teaching of communication and other academic skills to people with visual impairment (especially if there is some residual sight), and these have been helpful for those with autism as well. However, it is best if staff are trained to distinguish blind from autistic development and can develop strategies to support the dual disability.

Sensory-Motor Impairment and Cerebral Palsy Severe forms of cerebral palsy such as spasticity and athetosis will usually be diagnosed early, and so differences in development caused by autism may at first be missed in the

concentration on the physical problems of controlling movement. Parents may attribute all difficulties to the cerebral palsy, and it is not until the child joins groups of others who have cerebral palsy without autism that the autistic differences are recognized. In services that rely on methods of physical manipulation to engender movement in children with cerebral palsy, the autism may make it harder for the child to benefit from the therapy because of difficulties with being touched and the continual need for others to assist with everyday life. If the methods used are more functionally based (as in Conductive Education), then the physical arrangement of the environment to encourage independent movement may suit the child with autism so the dual disability is not so much of a problem. Cerebral palsy, however, will make it harder for the child to show independent actions and so the child is more dependent on communication to get needs met. This is a problem in the dual disability in that communication is a core difficulty in autism. Yet, the fact that the child needs to communicate may provide extra communicative pressure to help the child acquire communication skills, as long as staff are skilled in preventing frustration and the development of challenging behavior. The core difficulty in cerebral palsy is control of motor movements, but there are also often additional cognitive problems that interfere with development. The interaction with autism adds to the complexity and makes it even harder to determine and meet individual needs.

In less severe forms of sensory-motor impairment, the effects may be subtler but also more difficult to understand and allow for. Ataxia (where the child is generally clumsy and has problems in balance) may exacerbate sensory processing problems characteristic of autism. However, the child with a dual disability may benefit from the support of a skilled physiotherapist or occupational therapist, whereas the “minor” motor problems of the child with autism alone may be neither understood nor treated.

Dyspraxia (a problem in the planning and control of voluntary movements) is often found as comorbid with autism. Unless dyspraxia is understood, the child who can perform actions when

they are “triggered,” but not when directed to (because that involves planning), is likely to be misunderstood as deliberately noncooperative. In the context of autism, especially, dyspraxic difficulties are often seen as a matter of motivation and will, and so are never addressed as a motor problem, where help is required.

Sensory Processing Impairments in Autism

The Personal Perspective Problems in sensory integration in autism have been long recognized, and many people with autism themselves describe their condition almost solely in terms of these sensory difficulties. In this view, supported by some professionals, (e.g., Shanker 2004), it is because of problems in integrating senses, and hypersensitivity to certain sensory domains, that individuals with autism quickly become overwhelmed by sensations, making it difficult to make sense of the world around them. In other words, sensory problems lead to perceptual problems. This does not account for cases of apparent hyposensitivity – a lack of responsiveness to sensory stimuli. One suggestion is that when thresholds are low, there is a biological reaction to overstimulation of “shutdown,” leading to very high thresholds. Eventually, a balance is reached, and the person can function again, until whatever hormonal or neural response, causing the lowering of the sensory threshold, begins again. This would mean fluctuating sensory reactions. If such a cycle of over- to under-sensitivity is a reality, it would be difficult to adjust to the environment itself but necessary to give the person as much proximal control as possible (headphones to wear to block out or dampen unpleasant noise, hats or colored lenses in glasses to reduce glare, soft clothing to wear, and the freedom to remove shoes when necessary) so they are able to manage their own environment as far as possible. All this comes from anecdotal accounts of people with autism themselves, albeit compellingly and vividly described. Even accepting the validity of the experiences, the mechanisms underlying them are

speculative, and some people seem to be relatively permanently over- or under-sensitive with respect to certain stimuli, rather than going through such cycles.

An alternative view of what is occurring in autism is that the central core difficulties are at the psychological level, making it a failure in perception that causes individuals with autism to perceive all the overwhelming details of sensory stimuli instead. There are no easy answers to this “chicken and egg” dilemma. It may rest with neurological testing eventually to make this decision. What is clear at a practical level is that both processes are important. When the environment is adapted to reduce sensory overload, the person with autism seems better able to think and make sense of it. Alternatively, when people with autism are taught to be better at chunking information and at forming concepts and categories, they are better able to make sense of their world and not be so overwhelmed by sensory stimuli.

The Beginnings of a Scientific Approach Attempts have been made to classify the sensory responses in autism so they can be quantified and two important questions addressed: (1) Is there a pattern of stimuli responsiveness that is characteristic of all individuals on the autism spectrum? (2) Do individuals on the autism spectrum differ from typical peers or individuals with other disorders in their responses to sensory stimuli? Dunn (1999) produced a sensory profile from a questionnaire to parents or carers, where responses are given on a Likert scale of frequency (always, sometimes, seldom, never) to questions about particular reactions to everyday sensory stimuli. Dunn measures 125 items grouped into 9 factors (10, if the category “other” is included) and 14 sections. Scores are compared with a standardized “regular” mean and responses are classified as:

- “Typical” – at or above 1 standard deviation below the mean
- “Probable difference” – between 1 and 2 standard deviations below the mean
- “Definite difference” – more than 2 standard deviations below the mean

Sensory Impairment in Autism, Table 1 Sensory sections (Adapted from Dunn 1999)

Sensory processing	Modulation	Behavioral/emotional
1. Auditory processing	1. Sensory processing related to time/endurance	1. Emotional/social
2. Visual processing	2. Modulation re body position and movement	2. Behavioral outcomes of sensory processing
3. Vestibular processing	3. Modulation of movement re activity level	3. Thresholds for responding
4. Touch processing	4. Modulation of sensory input affecting emotions	
5. Multisensory	5. Modulation of visual input affecting emotions and activity level	
6. Oral-sensory		

The sections are further grouped into “sensory processing,” “modulation,” and “behavioral/emotional” (Table 1).

Alongside these are 9 (10) factors:

- 1. Sensation-seeking
- 2. Emotionally reactive
- 3. Low endurance/time
- 4. Oral sensory sensitivity
- 5. Inattention/distractibility
- 6. Poor registration
- 7. Sensory sensitivity
- 8. Sedentary
- 9. Fine-motor/perceptual
- 10. Other

The purpose of the sensory assessment is to build a sensory profile of the individual. This scale is relatively objective (reporting norms, levels of reliability, and validity) allowing comparison of individuals with autism to others and, within the scale, to identify particular patterns of sensory disturbance. This profile is based on a broad range of sensory processing that might be affected in autism although it does not include proprioceptive sensations, which allow one to get feedback on actions and monitor the position of one’s body in space. People with autism describe difficulties in this kind of processing, and it would help account for some of the problems in making goal-directed actions in some individuals with autism and in being aware of one’s own intentionality.

Nor does it mention the processing of pain, for which there is conflicting evidence. Anecdotal reports, of a high pain threshold or an extreme delay in registering pain, are not supported by the little scientific evidence that exists (Nader et al. 2004). An absence of, or delay in experiencing, pain would make it very difficult for the child to connect the actions that lead to an injury with the experience of pain, and so the child might well repeat dangerous actions regardless of consequences. However, failure to report pain may be due to social or communication differences or fear of the “sensory and social onslaught” that comes from comforting. There is certainly anecdotal evidence of recklessness in some individuals with autism but the lack of responsiveness to social restraint may be key to that. It is possible that there is a synesthesia effect in autism in that other sensations, which are typically benign, may cause pain. Thus, some taste, touch, and auditory sensations have been reported as painful by adults with autism.

There are other profiles, used by practitioners to help their understanding of what may be happening in individuals, to plan effective programs for learning and to help avoid challenging behavior. Bogdashina’s (2003) sensory profile includes as additional items “literal” perception, “Gestalt” perception (inability to distinguish background from foreground information), inconsistent/fragmented/distorted perception, sensory agnosia, delayed perception, and vulnerability to sensory overload. These categories have good

face validity for those who live and work with those with autism, but the items are often hard to operationalize, so comparisons across scorers and individuals are hard to establish. Bogdashina describes different perceptual styles as “monoprocessing,” “peripheral,” “system shut-down,” “compensatory,” “resonance,” and “day-dreaming.” She goes on to describe different cognitive styles as “subconscious,” “unconscious,” and “preconscious.” Finally, Bogdashina talks of some particular sensory processing issues which have been said to characterize (or at least be common within) autism: these are “synesthesia,” “central auditory processing disorder” (CAPD), “scotopic sensitivity” or “Irlen syndrome” (a disorder whose existence is controversial), and “sensory integration dysfunction.” Many of these supposed sensory disabilities are tied to particular treatment approaches, so caution is needed to establish their validity as concepts before evaluation of treatments is undertaken.

Current Knowledge

Dual Diagnoses of Sensory Impairment and Autism

More children with sensory impairments are receiving a dual diagnosis of autism with a sensory disorder, although the confusion over early signs remains. Children with “pure” sensory impairment continue to be more likely to be educated in mainstream environments, and special schools retain those with multiple, more complex disorders. As Jordan (2011) has argued, specialist education for children with autism is not about location but primarily about the knowledge and skills of staff. More teachers of the visually and hearing impaired are recognizing their need for additional training in understanding and working with children with autism, when faced with the dual disability. There has been no evaluation of the different placements for such dual-diagnosed children; it might be expected that where a hearing impairment is involved, the school for hearing impaired children (with additional training and resources) would be successful, whereas there would need to be greater adjustments in the

teaching of children with the dual diagnosis of autism and visual impairment compared to those with visual impairment alone. However, this is an empirical question that has not yet been addressed. A method of communicative assessment and treatment for children with severe autism (COMFOR: Noens, Van Berckalaer-Onnes, Verpoorten & Van Duijn (2006) has been developed and the same team are now developing a version for children with an additional visual impairment).

Sensory Dysfunction Within Autism

Evidence for a Sensory Dysfunction as Part of Autism Early studies suggested that children with autism could be distinguished from children with developmental delay and typically developing children on the basis of abnormalities in sensory functioning. Some showed differences in early sensory-motor behavior (under 1 year) that could distinguish those who were later typically developing, intellectually impaired, or had autism (Baranek 1999). Most of the studies have compared the scores of children with autism with those of typically developing children or children with other disorders (or both) on Dunn’s Sensory Profile. Most have found significant differences, on at least some categories. However, many of the findings are contaminated by using children from a wide age range, when there are known age differences in sensory modulation with age. Participants also need to be matched for developmental age (or there need to be two comparison groups) since those with intellectual impairment also show differences in sensory modulation from the typical. Rogers and Ozonoff (2005) review the evidence and conclude that there are some differences between children with autism and typical children (and sometimes other groups as well) but little consistency on the nature and extent of these differences. The most consistent finding is of greater under-responsiveness in children with autism spectrum disorders. Miller et al. (2005) examined sensory processing in children with a diagnosis of Asperger syndrome or High Functioning Autism. They classified their sample into three different responsivity groupings: 1.

Sensory over-responsivity; 2. Sensory under-responsivity; 3. Sensory seeking. They also found they could be divided into 'low' versus 'high' arousal groups and 'sensory habituators' versus 'non-habituators'. However, recent studies have queried the value of such grouping across different sense domains. Ben-Sasson et al. (2009) conducted a meta-analysis of studies of sensory disturbances in autism and established that such disturbances were both multi-modal and variable. They also found that sensory disturbances were universal across the autism spectrum. Young children with autism were found to be more likely to be hypo-responsive or non-responsive but not sensory seeking. Older children and adults were more likely to be hypersensitive but were also more likely to be sensory seeking. This suggests a variable pattern of responsiveness to sensory stimuli across different domains and no simple relationship between sensory sensitivity and sensory seeking or avoidance.

The full form of the sensory profile contains items that pertain to core areas of autism so it is not surprising that differences in these areas would occur. The short form is better in that it excludes social, communication, and motor responses. Dunn and Daniels (2002) have produced a short form specific to toddlers and infants, and this has been used, with other measures, in a comprehensive study by Ben-Sasson et al. (2007), which sought to address some of the methodological weaknesses of the earlier studies. They used comparison groups matched for chronological and mental age and not only the Dunn toddler profile but other parent-report scales and direct observations. They found consistency in parent reports across different measures but a poor correlation between parental report and direct observations. The most consistent finding was in the extremity of the responses in the autism group compared with controls with a high frequency of unresponsiveness to sensory stimuli and high levels of avoiding behavior, as well as low levels of sensory-seeking behavior. In interpreting their findings, however, they point to the fact that many of the scales, as with the full sensory profile, use social stimuli (e.g., avoids eye contact) to judge sensory responses,

and, by definition, these will be different in autism.

Crane et al. (2009), using the Adult Adolescent Sensory Profile (AASP), a shorter self-report questionnaire, found that the scores of adults with autism differed significantly from the typical in four areas. Those with autism scored more highly on "low registration," "sensory sensitivity," and "sensory avoiding" but had lower scores on "sensory seeking." The most extreme differences were in "sensory avoidance." There were correlations between the scores and IQ but no correlation with age or degree of autism. Thus, there appears to be some support for sensory differences in autism that persist in time, but there is little evidence that the sensory differences lead to autism in any way.

A recent comprehensive study of sensory processing in autism by Lane, Young, Baker and Angley (2010) used a model-based cluster analysis approach to examine sensory processing subtypes in 54 children with autism and to relate them to different levels and kinds of adaptive functioning. They claim to have revealed three subtypes of sensory functioning which predicted communicative competence and maladaptive behaviour. While admitting limitations of the study in that small numbers were involved and the measures came from a parental questionnaire, the authors suggest that the results from the model they used allow more specific questions to be posed, which should yield more scientific data in the future. They found overall significant differences between the children with autism and typically developing children in the degree of under-responsiveness, the number who were seeking sensation and (in 92% of the children) those with problems in auditory filtering. They used measures across different sensory domains and compared specific data rather than producing generalized 'sensitivity' classifications.

The classifications found in the Lane et al. study (op cit) were based on sensitivities in particular domains: 1. Sensory-based inattention and sensory seeking (SBIS); 2. Sensory modulation difficulties in movement (SMMS); 3. Sensory modulation difficulties in taste/smell (SMTS). The SBIS group only had mild sensory processing

problems and these were related to the behavioral issues of inattention, distractibility, over-focused attention and impulsivity. The SMSS group had wide sensory modulating difficulties across most domains; there were cases of both over and under sensitivity. They had problems with motor sensitivity and extremely low scores in the low energy/weak domain. The behavioral relationships were with low muscle tone, weak grasp and easily tiring. Finally, the SMTS group has sensory modulation difficulties in taste/ smell particularly but across all domains except the motor. This group was associated with the greatest problems in communication. There was a general relationship found between the degree of sensory function and challenging behavior with taste/smell sensitivity, auditory filtering and movement sensitivity all being related to particular patterns of challenging behavior.

Interventions for Sensory Dysfunction in Autism Dawson and Watling (2000) reviewed research evidence of the effectiveness of different treatments to address the sensory problems in autism. They examined the three areas where most treatment has been undertaken: (1) auditory integration therapy, (2) sensory integration therapy, and (3) occupational therapy for sensory dysfunction (as distinct from occupational therapy directed at building functional skills). Auditory integration therapy is directed at the presumed deficits in auditory processing in autism. The aim is to get the child to “relearn” how to listen by taking him/her through specially selected sounds presented through earphones several times a day over a period of a few weeks. Many claims have been made about the effects on behavior and even reduction (sometimes “elimination”) of autistic symptoms. However, as Dawson and Watling report, in all but one of the studies, the children who had undergone AIT did no better than “placebo” controls. The one study showing beneficial effects was the least satisfactory in its methodology.

Sensory integration therapy is a program of controlled sensory experiences involving vestibular, proprioceptive, and somatosensory activities. The activities themselves include swinging, deep pressure touch, and tactile stimulation. There were few methodologically sound studies of sensory

integration therapy, most reports being of very small case studies over short time frames. In addition, the more rigorous the research design, the smaller the beneficial effect noted.

Finally, although occupational therapists are heavily involved in treating children with autism for sensory integration problems, Dawson and Watling could find no scientific evaluation of their work.

Neurophysiological Support for Sensory Dysfunction in Autism If sensory dysfunction is a core part of autism, one would expect to find it in studies of the neurology of sensory processing as well as in behavior (Table 2). Maro et al. (2011) have recently reviewed these findings. They look at studies that have concerned auditory, tactile, visual, and multisensory stimuli and used measures such as EEG (electroencephalography), MEG (magnetoencephalography), and fMRI (functional magnetic response imaging).

These findings, therefore, suggest that behavioral differences in responses to sensory information are reflected in neurophysiological differences. The results are not consistent enough for a definitive picture to emerge but there is evidence of some differences. Maro et al. (2011) interpret these findings with caution. They point out that other mechanisms, such as attention, which are known to be affected in autism, may influence these findings. There is evidence of problems with attention shift in autism but only with those who have additional learning difficulties. It also appears that individuals with autism can overcome their difficulty in switching attention to speech tones away from other complex tones by direct instruction to attend to them. Similarly, problems with selectively attending to certain stimuli in autism vary according to the intensity and complexity of the stimuli, and it is only in complex situations that individuals with autism are less able than others to respond to a sound source. Thus, the difficulty may lie with inhibiting reactions to other stimuli as much as with attention to the target stimulation.

Conclusions Regarding Sensory Dysfunction and Autism This is an area of great mismatch between practice and established science. There

Sensory Impairment in Autism, Table 2 Summary of findings of sensory processing in autism at neurophysiological level

Auditory	Tactile	Visual	Multisensory	Higher multisensory
There are many inconsistent findings, but there is good evidence of significant differences in speech sound processing. Some evidence of atypical processing in the primary auditory cortex	The results suggest some specific hypersensitivities in autism. There were enhanced responses to low-level vibration. Some hypersensitivity to vibration and heat but not to light touch	Better responses to detail when stimuli are simple but general impairment with complex stimuli. Also some problems with spatial discrimination. Face recognition is impaired but depends on familiarity, attention, gaze direction, fixation, and type/complexity of stimuli. Children with autism better than typically developing at processing detailed high spatial frequency stimuli and worse at rapid low-frequency processing. Differed in emotional responses to faces in amygdala and cortex	Some differences in the processing of visual auditory illusions, which suggests individuals with autism may be less efficient at multisensory integration. Problems in autism when auditory and visual stimuli presented together – leading to delay in processing at the second stage	Most significant difference found is in the integration of audio and visual information in the reception of speech. Individuals with autism appear to depend more on the auditory perception of phonemes, divorced from their lip patterns, and training has little effect on this. Practically this could lead to problems in speech perception in noisy environments where lip patterns would normally serve as a discriminatory cue. Findings suggestive of problems in autism in the connections between cortical and subcortical regions of the brain

is a wealth of anecdotal evidence of sensory processing problems in autism but very few scientific “facts” have been established. It appears that sensory processing is neither uniformly good nor bad in autism, but rather depends on the nature and complexity of the signal, the background stimulation, and the directions given. The uncertain picture at the behavioral level is mirrored at the neurophysiological level, although specific difficulties in connecting vision with sound, especially in relation to speech, do seem to be firmly established.

Future Directions

Dual Sensory Impairments and Autism

The current situation, described above, where children with “pure” sensory impairments are increasingly educated within the mainstream is

likely to continue. With technological advances in the treatment of sensory impairments and in providing tools to access the mainstream curriculum, however, those with more complex multisensory impairments and/or dual diagnoses may also come to be included in the mainstream, so that the existence of schools and services specific to particular sensory impairments becomes problematic. There will still be a need for qualified staff to support and educate families, vocational, and social care staff and to provide peripatetic support to schools, but it may become difficult for staff to gain experience (and therefore, expertise) in the sensory impairment. This in turn will affect the services available to individuals with autism and a comorbid sensory impairment. It will no longer be a matter of adding autism training to staff already experts in sensory disability but perhaps a need for “de novo” training in such dual disabilities. Since such dual disabilities are

relatively rare, such services may need to be centralized and with that will come accessibility issues and the possible reemergence of residential segregated schooling. Lifelong support may also deteriorate as relevant expertise becomes scarce.

Sensory Processing Disorder and Autism

The present dearth of scientific evidence on the nature and extent of sensory dysfunction as part of autism is likely to change in line with the changes in DSM-V. Diagnoses, even of just “associated features” of autism, cannot just rely on anecdotal evidence. The current sensory profile assessment tools will need to be adapted so they have greater validity, especially when some research suggests that parents of children with autism are not accurate reporters of their child’s sensory experience. Adult profiles circumvent this problem but have the additional problem of only being accessible to those above a certain level of intellect and ability to communicate. Some research already suggests that low- and high-functioning individuals with autism (in terms of adaptive skills) differ in sensory responsiveness, and those who have least sensory problems are liable to do best in acquiring academic and adaptive skills, including communication. It may be that the validity of behavioral sensory profiles will need to be validated against neuropsychological “direct” testing of sensory processing in individuals with autism and others.

Whatever profile is used, the terms need to be operationalized to give greater reliability. A priority will be areas where there is already some good evidence of sensory difficulties: auditory and tactile stimuli. Once valid and reliable tools for measuring sensory processing are available, the validity (or otherwise) of sensory processing differences in autism will need to be established by good scientific comparison studies across the senses of vision, hearing, taste, touch, smell, proprioception, vestibular sensation, and pain. There may also need to provide data across different domains since it appears that generalized “sensitivity” scores are not as useful as measures of sensitivity within different sense domains. Multisensory integration will also need to be studied, through carefully constructed tasks that clearly depend on such integration, as will unusual phenomena such as synesthesia.

However, finding (or refuting) differences in sensory processing in autism is only the start. Any such differences will need to be interpreted and investigated in terms of their relationship to other possible confounding variables: the role of attention shift and selective attention differences in autism, the differences in communication, emotion processing, social referencing, and processing of cognitive complexity. Once a clearer picture emerges of the nature of the differences in autism processing, there needs to be an analysis of the extent to which “differences” can be equated with “difficulties,” which might then be candidates for remediation. Only once the clear targets for treatment have been set does it make sense to evaluate treatments (or perhaps develop new treatments for evaluation). Treatments could then be evaluated in terms of their research rationale as well as efficacy, in line with good practice (Jones and Jordan 2008). Occupational therapy needs to develop as a profession to include this kind of evaluation of practice if it is to demonstrate its value beyond the level of anecdote.

See Also

- ▶ [Auditory Integration Therapy](#)
- ▶ [Auditory Processing](#)
- ▶ [Blindness](#)
- ▶ [Deafness](#)
- ▶ [Dyspraxia](#)
- ▶ [Evaluation of Sensory Processing](#)
- ▶ [Face Perception](#)
- ▶ [Sensory Diet](#)
- ▶ [Sensory Processing Measure](#)
- ▶ [Touch Sensitivity](#)

References and Reading

- Baranek, G. T. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders*, 29, 213–224.
- Ben-Sasson, A., Cermak, S. A., Orsmond, G. I., Tager-Flusberg, H., Carter, A.S., Kadlec, M.B., & Dunn, W. (2007) Extreme sensory modulation behaviors in toddlers with autism spectrum disorders. *The American Journal of Occupational Therapy*, 61(5), 584–592.

- Bogdashina, O. (2003). *Sensory perceptual issues in autism and Asperger syndrome*. London: Jessica Kingsley.
- Crane, L., Goddard, L., & Pring, L. (2009). Sensory processing in adults with autism spectrum disorders. *Autism: The International Journal of Research & Practice*, 13(3), 215–228.
- Dawson, G., & Watling, R. (2000). Interventions to facilitate auditory, visual and motor integration in autism: A review of the evidence. *Journal of Autism & Developmental Disorders*, 30(5), 415–421.
- Dunn, W. (1999). *The sensory profile manual*. San Antonio: The Psychological Corporation.
- Dunn, W., & Daniels, D. B. (2002). Initial development of the Infant/Toddler Sensory Profile. *Journal of Early Intervention*, 25(1), 27–41.
- Jones, G., & Jordan, R. (2008). Research base for interventions in autism spectrum disorders. In E. McGregor, M. Núñez, K. Williams, & J. Gómez (Eds.), *An Integrated View of Autism*. Oxford: Oxford University Press, p. 281–302.
- Jordan, R. (2011). Special education. In G. O'Brien & M. Bax (Eds.), *Evidence-based therapy in childhood neurodisability*. London: McKeith Press.
- Maro, E. J., Hinkley, L. B., Hill, S. S., & Nagarajan, S. S. (2011). Sensory processing in autism: A review of neurophysiological findings. *Pediatric Research*, 69(2), 48R–54R.
- Nader, R., Oberlander, T. F., Chambers, C. T., & Craig, K. D. (2004). Expressions of pain in children with autism. *Clinical Journal of Pain*, 20(2), 88–97.
- Pring, L. (Ed.). (2004). *Autism and blindness: Research and reflections*. Chichester: Wiley.
- Rogers, S. J., & Ozonoff, S. (2005). Annotation: What do we know about sensory dysfunction in autism? A critical review of the empirical evidence. *Journal of Child Psychology and Psychiatry*, 46, 1255–1268.
- Shanker, S. G. (2004). Autism and the dynamic developmental model of emotions. *Philosophy, Psychiatry & Psychology*, 11(3), 219–233.

Sensory Integration (SI) Therapy

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Definition

Sensory integration therapy, guided by sensory integration theory originated by Dr. A. Jean

Ayres (1979), is commonly utilized by occupational therapy practitioners (additional information found under A. Jean Ayres in the “[See Also](#)” section below). Although many professionals outside the field of occupational therapy have declared that their intervention techniques are “sensory integration,” their practices do not correspond to the theoretical principles researched by Dr. Ayres and her occupational therapy colleagues, nor do they result in an adaptive or purposeful response and improved engagement in occupation (Roley et al. 2007). As a result of this encroachment on the term sensory integration, the successor trustee of Dr. Ayres estate (Baker/Ayres Trust) trademarked the term Ayres Sensory Integration® (ASI) thus allowing consumers to be assured that they are acquiring the sensory integration services intended. In order for occupational therapists to identify their use of ASI, the term OT/SI is recommended.

Sensory processing difficulties are noted in many diagnostic categories, as well as in children with no other clinical or developmental concerns. However, there is a high incidence of sensory processing impairments in children on the autism spectrum. For this specific population of individuals, Ayres Sensory Integration intervention is different from simply providing sensory activities as a reward for appropriate behavior or desired performance during discrete trial programs, moving an individual into a “time-out” room which houses sensory equipment, or providing strategies throughout the day to keep the child calm and focused (i.e., sensory diet). In fact, it is well understood by occupational therapy practitioners that ASI is done within the context of play and that engagement in the play experience is its own reward because the play is meaningful to the child. As the aim of occupational therapy is to support an individual’s participation in life through engagement in occupation (American Occupational Therapy Association [AOTA] 2008), the therapeutic intervention outlined by the occupational therapy practitioner using an Ayres Sensory Integration approach is child specific based on the child’s sensory processes and how these processes are barriers to, or facilitators of, participation and engagement in life activities identified as priorities by the family and team.

Two specific areas of attention with this population include:

- **Praxis:** the process of action performance requiring ideation, planning, execution, and sequencing (May-Benson 2004)
- **Modulation:** maintaining a functional state of alertness and regulating the degree, intensity, and nature of responses to a sensory event (Miller 2006)

It is also important to note that occupational therapy practitioners utilize an ASI approach in combination with other theoretical frameworks. An official statement by the American Occupational Therapy Association (AOTA 2010, p. 4) regarding intervention for individuals on the autism spectrum cites: “Effective interventions also address contextual factors such as structure, consistency of routine, sensory environments that optimize attention and arousal, and caregiver skills that contribute to occupational performance. Research evidence indicates that the occupational therapy intervention process should be individualized, intensive, and comprehensive; include the family; and facilitate active engagement of the individual” (Tomchek and Case-Smith 2009). This document goes on to highlight the underlying sensory processes that should be assessed for this population: “sensory modulation, self-regulation, praxis, and motor imitation” (p. 4). Additionally, Ben-Sasson et al. (2007) emphasize that in order to accurately identify the needs of very young children on the spectrum, occupational therapists need to assess multiple components of the child’s sensory functioning across varying contexts in order to fully understand and support the impact on the child’s performance and the family’s life.

Historical Background

Ayres original theory of sensory integration (Ayres 1972, 1979) described what was referred to as a scientific theory. That is to say that as more research is conducted, technology

becomes available to scientifically measure the underlying theoretical constructs, and outcome information is obtained, the theory would evolve to inform practice. This concept has also been described as the scientific evolution of the sensory integration frame of reference (Schaaf and Davies 2010).

Ayres began her work in this area in the 1950s, coined the term sensory integration in the 1960s, and culminated her work with the publication of the Sensory Integration and Praxis Tests (SIPT; 1989). While there has been some change in the terminology used to describe the diagnostic categories identified through this comprehensive assessment tool (refer to the Sensory Integration and Praxis Tests in the “[See Also](#)” section for full review), the intervention strategies utilizing Ayres basic tenets have not changed. The theory espouses that improving sensory integrative functions leads to adaptive responses (purposeful actions in response to an environmental demand) allowing the child to engage more fully in everyday life tasks or occupations (i.e., play, learning, daily living tasks).

Rationale or Underlying Theory

Dr. Ayres work, based on neuroscience, psychology, education, biology, and occupational therapy, hypothesized that underlying difficulties with integrating sensory information can lead to learning and behavior difficulties (Ayres 1972). The three major postulates of sensory integration theory are:

1. Learning is dependent on the ability to take in and process sensation from movement and the environment, and use it to plan and organize behavior.
2. Individuals who have a decreased ability to process sensation also may have difficulty producing appropriate actions, which, in turn, may interfere with learning and behavior.
3. Enhanced sensation, as a part of meaningful activity that yields an adaptive interaction,

improves the ability to process sensation, thereby enhancing learning and behavior.

The basic assumptions of the theory are that the nervous system is plastic, sensory integration develops, the brain functions as an integrated whole, adaptive interactions are critical to sensory integration, and people have an inner drive to develop sensory integration through participation in sensorimotor activities. Lane and Schaaf (2010), in their article examining the neuroscience evidence supporting or refuting the implementation of OT/SI, stated (p. 375): “OT/SI is based on the belief that engagement in individually tailored activities, rich in the needed sensory stimuli, will improve the ability of the brain and nervous system to process sensory information, enhance the organization and integration of sensation, and, as a result, have a positive impact on the child’s ability to participate in daily life activities (Ayres 1972, 1979).” After reviewing the neuroscience literature, Lane and Schaaf go on to state: “There is little question that the nervous system is plastic and that sensory input is an important mediator of this plasticity. Motor activity and interest in task also appear to be important contributors, and active engagement is seen to enhance the effects. Moreover, these studies indicated that neuroplastic changes were developmental, dynamic (reactive), and task specific. In this regard, these data provide indirect support for the use of OT/SI, which is built on the premise that active engagement in meaningful, sensorimotor activities at the just-right challenge and in a playful or meaningful context has a positive impact (by means of neuroplasticity) on processing in the nervous system (Ayres 1972).”

Goals and Objectives

Despite the intervention chosen, the goals and objectives of occupational therapy intervention are the same. According to the AOTA’s statement on the scope of services for individuals on the autism spectrum (2008, p. 4):

Assessing the outcomes of service is an integral part of the occupational therapy process and is important for determining future actions and for evaluating occupational therapy services provided for the individual, organization, or population. This assessment involves monitoring the client’s responses to intervention, reevaluating and modifying the intervention plan, and measuring intervention success through outcomes that are important to the client within the dynamic physical and social environments and cultural contexts where functioning occurs. Progress is noted through improvement in the client’s occupational performance, adaptation, participation in desired activities, satisfaction, role competence, health and wellness, and quality of life and through prevention of further difficulties and facilitation of effective transitions. Occupational therapy practice for individuals with an ASD is consistent with the WHO’s (2001) *ICFg* and the National Research Council’s 2001 recommended practices for educating individuals with an ASD.

For OT/SI specifically, the intent of therapeutic intervention is to assist the individual in receiving, processing, correctly interpreting, and integrating sensory information as a foundation for improving functional participation in life’s tasks. It is important to note that these underlying skills, rather than simply training of a child’s functional difficulties, are the focus of this intervention approach. The maturation and development of these underlying substrates of dysfunction are then measured via praxis (motor planning) and modulation abilities which impact functional participation, learning, and behavior. In addition, since the sensory processes of a child on the spectrum also affect the family’s routines and social participation (Bagby et al. 2012), this area also needs to be supported by the occupational therapy practitioner. For example, Bagby, Dickie, and Baranek’s research identified that chosen activities for the family are often influenced by the child’s tolerance to sensory experiences, the amount of planning that needs to occur prior to participation in life’s activities is significant, and the ability to create family memories is often compromised as parents need to split up during family outings.

Treatment Participants

Occupational therapy practitioners, with advanced training in ASI, will often refer to their intervention as OT/SI to indicate utilization of this specialty approach. This approach is a dynamic exchange between the OT/SI practitioner and the child/individual with sensory processing concerns. Additionally, the collaboration with family and school personnel is critical for the generalization of treatment outcomes.

Treatment Procedures

In addition to Ayres writings (Ayres 1972, 1979), sensory integrative interventions have been described and discussed in texts intended to improve the clinical reasoning skills of clinicians (Bundy et al. 2002; Fisher et al. 1991) and numerous articles in the occupational therapy literature. Schaaf and Miller (2005) highlight how astute observation of the child's ability to process and utilize sensory information is a key skill of the well-trained therapist.

In order to clearly delineate the components of this intervention approach, a group of occupational therapy researchers (Sensory Integration Research Collaborative [SIRC]) detailed the required elements of intervention (Parham et al. 2007, 2011). While the original intent was to provide a mechanism to assure fidelity (truthfulness) to the theoretical principles during the intervention phase of research studies, the secondary benefit was that occupational therapy practitioners were provided with a framework to understand what is and what is not Ayres Sensory Integration intervention.

The Ayres Sensory Integration Fidelity Measure (Parham et al. 2011, pp. 135–136) outlines the following components for this occupational therapy approach:

- The therapist should have post-professional training in sensory integration which includes a minimum of 50 education hours of SI theory and practice (e.g., post-professional SI or SIPT certification or university course) and ongoing

supervision (minimum of 1 h per month) by an expert with at least 5 years experience providing occupational therapy using SI intervention.

- The occupational therapy assessment report should include information-related occupational therapy principles (AOTA 2008) including historical information, reason for referral, an occupational profile, and performance patterns.
- The occupational therapy assessment report should include information related to specific sensory patterns and processes, including sensory modulation and discrimination, postural/ocular control, visual-perceptual skills, fine and gross motor skills, motor coordination, praxis, organizational skills, and the influence of sensory integration and multisensory processing on performance, as well as an interpretive summary describing the effects of sensory integration and praxis on the referring difficulties.
- The physical environment in which the ASI intervention should occur includes adequate space for the flow of the intervention including vigorous physical activity; equipment and materials which can be flexibly arranged to adjust to the rapid changes which occur during the course of intervention and the varying size of the child; no less than three hooks for suspended equipment (with adequate spacing for full orbit); availability of rotational devices (to allow for 360° of rotation) and bungee cords; mats/cushions/pillows for safety; a variety of equipment for sensory experiences, e.g., bouncing equipment, therapy balls, a variety of swings, scooters, ramps, climbing equipment, weighted objects, ball pit, barrel, spandex fabric, vibrating toys, tactile material, visual targets, and props to engagement in play; and a quiet space.
- Communication with parents and teachers to support goal setting as well as to allow for discussion of the potential influence of sensory integration and praxis on performance in valued activities; the child and family's participation in home, school, and community; and how the therapist defines the areas to be addressed that will improve engagement.

- During the intervention process, the therapist should:
 - Ensure the child's physical safety by anticipating physical hazards and be in close physical proximity during activities.
 - Present the child with tactile, vestibular, and proprioceptive opportunities to support the development of self-regulation, sensory awareness, and/or movement in space.
 - Help the child attain and maintain appropriate levels of alertness and an affective state that supports engagement in activities.
 - Collaborate on activity choices with the child.
 - Support and challenge postural control, ocular control, and/or bilateral development through the use of postural, resistive whole-body, ocular-motor, oral, or bilateral challenges, as well as projected action sequences.
 - Challenge praxis (ability to conceptualize and plan novel motor tasks) and organization of behavior in time and space.
 - Tailor the activity to the just-right challenge by increasing the complexity of the challenge when the child responds successfully.
 - Ensure that activities are successful and, thus, the child is making an adaptive response to the presented challenges in sensory modulation or discrimination; postural, ocular, or oral control; or praxis.
 - Support the child's intrinsic motivation to play by creating a setting which supports play as a way to fully engage the child in the intervention.
 - Establish a therapeutic alliance which promotes and establishes a connection with the child and conveys a sense of working together in a mutually enjoyable partnership (vs. basic pleasantries or feedback on performance such as praise or instruction).

Efficacy Information

Unequivocal evidence on the effectiveness of Ayres Sensory Integration remains elusive for a

variety of reasons. This is not to say that occupational therapy practitioners do not see improvement in the children they serve, as there are numerous reports of progress via parental observations, improvement and mastery of identified goals and objectives, and small-scale studies with positive outcomes. However, gold-standard research outcomes have not been documented.

One difficulty with performing high-quality, empirically sound research is the confusion over what is and what is not Ayres Sensory Integration intervention. Several meta-analyses have been performed to review the literature on this topic, yet the actual interventions did not align consistently with the principles of ASI intervention. An early review by Ottenbacher (1982) found significant positive effects in motor outcomes for children with learning disabilities and mental retardation as compared to no treatment; Polatajko et al. (1992) found that the sensory integration intervention showed the best gains in motor performance for children with learning disabilities but that the SI intervention was no more effective than other interventions; Vargas and Camilli (1999) reported moderate effects in motor performance compared to no treatment but little difference as compared to other more traditional occupational therapy approaches (i.e., perceptual-motor approach); Hoehn and Baumeister (1994) reexamined some of the Polatajko, Kaplan, and Wilson's studies and found some positive outcomes but did not substantiate an SI approach as providing a more positive outcome than alternative approaches; and Baranek (2002), who looked specifically at sensory-based interventions for children on the autism spectrum, found that the small sample sizes and weak designs need to be considered when viewing the positive outcomes. In the most recent review, May-Benson and Koomar (2010) systematically reviewed, evaluated, and synthesized the findings of 27 articles on the effectiveness of sensory integration interventions found in the literature from 1972 through 2007. "Outcome areas examined were as follows: motor performance, sensory processing, behavioral outcomes, academic and psychoeducational outcomes, and occupational performance" (p. 406).

Since the last systematic review, several small studies related to the population of children on the autism spectrum have occurred. For example, a group of 37 children on the spectrum was studied in a design comparing sensory integration (SI) intervention to a fine motor intervention (Pfeiffer et al. 2011). While both groups improved on Goal Attainment Scaling, the SI group showed more significant improvement in the three domains of sensory processing, motor skills, and social functioning. Additionally, the SI group demonstrated fewer autistic mannerisms (as measure by a subscale of the *Social Responsiveness Scale*) than the fine motor group after the interventions. In another small study, Smith et al. (2005) reported positive effects of a sensory integrative approach on decreasing self-stimulating behaviors. Despite these efforts, large-scale, randomized control effectiveness studies have yet to be completed.

Another difficulty in designing research studies on the effectiveness of ASI is the underlying concept that the process is dynamic exchange between child and practitioner with the intervention specific to each child's particular combination of difficulties, thus operationalizing the variables within a research design presents a significant challenge. In an effort to address this and other methodological challenges, as well as definitively outline what are the core components of ASI, a team of researchers developed a fidelity measure for sensory integration research (Parham et al. 2007, 2011). This measure outlines both structural and process elements necessary for the intervention to be congruent with Ayres theory and thus designated as Ayres Sensory Integration. For research purposes, this measure allows for the manualization of the intervention – i.e., the precise description of the intervention components which are then consistently administered in order to have confidence that the outcome is the result of the intervention and the intervention can be reproduced in subsequent studies.

The Ayres Sensory Integration Fidelity Measure, which outlines the following components of providing ASI, has been found to have strong

content validity for the structural and process elements, thus representing the essential features of ASI intervention (Parham et al. 2011). Additionally, the process components are reliable and valid when scored by experts in ASI, but not when scored by practitioners without advanced training.

Structural elements (Parham et al. 2011, p. 135):

1. Therapist qualifications
2. Components of the evaluation/assessment report
3. Physical environment
4. Communication with the parent or teacher

Process elements (Parham et al. 2011, p. 136):

1. Ensure physical safety.
2. Present sensory opportunities.
3. Help the child attain and maintain appropriate levels of arousal.
4. Challenge postural, ocular, oral, and bilateral motor control.
5. Challenge praxis and organization of behavior.
6. Collaborate in activity choice.
7. Tailor the activity to present the just-right challenge.
8. Ensure that activities are successful.
9. Support the child's intrinsic motivation to play.
10. Establish a therapeutic alliance.

With this ASI Fidelity Measure, along with a manual outlining ASI's philosophies, principles, and strategies which is currently under development, it is hoped that more scientifically sound research on the efficacy and effectiveness of Ayres Sensory Integration is in the near future.

Outcome Measurement

See “Goals and Objectives” as functionally related outcomes are child specific.

Qualifications of Treatment Providers

There has been much discussion related to the qualifications of the treatment provider as it relates to Ayres Sensory Integration. As this theoretical approach was created by an occupational therapist for occupational therapists, the model of intervention is presented in all entry-level curricula for occupational therapists. However, there is variability between academic institutions in how the information is presented, to what extent the information is discussed and/or practiced, and the qualifications of the teacher within this area of practice. Additionally, as some physical therapists with a pediatric specialty also utilize this approach, the qualifications of the treating practitioner needed to be highlighted. The qualifications, while originally designed for research purposes, serve as a guideline for practitioners aspiring to provide professionally responsible ASI intervention. The Ayres Sensory Integration Fidelity Measure outlines the therapist qualifications to include (Parham et al. 2011, p.135):

- Post-professional training in sensory integration – certification in SI/SIPT: minimum of 50 education hours of SI theory and practice (e.g., post-professional SI or SIPT certification or university course)
- Supervision by an expert with at least 5 years experience providing occupational therapy using SI intervention (minimum of 1 h per month)

Years ago, there was a specialty certification that was offered by Sensory Integration International, the organization originally created for the advancement and dissemination of Dr. Ayres' work. After the passing of Dr. Ayres, and some internal difficulties, this organization ceased offering these courses. Currently, there is only one training series in order for therapists to be certified in the administration and interpretation of the Sensory Integration and Praxis Tests (SIPT), offered jointly by the University of

Southern California's Division of Occupational Science and Occupational Therapy and Western Psychological Services (USC/WPS) and approved by the Successor Trustee of Dr. Ayres' estate (holder of the trademark term Ayres Sensory Integration®).

This certification includes a series of four, 5-day courses:

- Theoretical foundations of sensory integration
- Technical and logistical administration of the SIPT as well as other commercially available measures of sensory processing
- Interpretation of the SIPT's standardized information and interfacing that information with other nonstandardized observations and reports of the child's performance in order to develop appropriate intervention plans
- Intervention planning based on the theory of sensory integration, including practical applications and clinical reasoning strategies

Please note, however, that the USC/WPS website does not list therapists who were certified by the original organization sanctioned to offer the Certification of the Administration and Interpretation of the SIPT. As a result, parents and professionals should not limit themselves to only those therapists registered with USC/WPS organization.

See Also

- ▶ [Ayres, A. Jean](#)
- ▶ [DeGangi-Berk Test of Sensory Integration](#)
- ▶ [Occupational Therapy \(OT\)](#)
- ▶ [Sensory Diet](#)
- ▶ [Sensory Integration and Praxis Test](#)
- ▶ [Sensory Processing](#)
- ▶ [Sensory Processing Assessment](#)
- ▶ [Sensory Processing Measure](#)
- ▶ [Sensory Processing Measure: Preschool \(SPM-P\)](#)
- ▶ [Test of Sensory Functioning in Infants](#)

References and Reading

- American Occupational Therapy Association. (2008). Occupational therapy practice framework: Domain and process (2nd ed.). *American Journal of Occupational Therapy*, 62, 625–688.
- American Occupational Therapy Association. (2010). *AOTA statement: Scope of occupational therapy services for individuals with an autism spectrum disorder across the lifecourse*. Bethesda: Author.
- Ayres, A. J. (1972). Improving academic scores through sensory integration. *Journal of Learning Disabilities*, 5, 338–343.
- Ayres, A. J. (1979). *Sensory integration and the child*. Los Angeles: Western Psychological Services.
- Bagby, M. S., Dickie, V. A., & Baranek, G. T. (2012). How sensory experiences of children with and without autism affect family occupations. *American Journal of Occupational Therapy*, 66, 78–86. <https://doi.org/10.5014/ajot.2012.000604>.
- Baranek, G. T. (2002). Efficacy of sensory and motor interventions for children with autism. *Journal of Autism and Developmental Disorders*, 32, 397–422. <https://doi.org/10.1023/A:1020541906063>.
- Ben-Sasson, A., Cermak, S. A., Orsmond, G. I., Tager-Flusberg, H., Carter, A. S., Kadlec, M. B., et al. (2007). Extreme sensory modulation behaviors in toddlers with autism spectrum disorders. *American Journal of Occupational Therapy*, 61, 584–592. <https://doi.org/10.5014/ajot.61.5.584>.
- Bundy, A. C., Lane, S. J., & Murray, E. A. (Eds.). (2002). *Sensory integration theory and practice* (2nd ed.). Philadelphia: F. A. Davis.
- Fisher, A., Murray, E., & Bundy, A. (1991). *Sensory integration theory and practice*. Philadelphia: F. A. Davis.
- Hoehn, T. P., & Baumeister, A. A. (1994). A critique of the application of sensory integration therapy to children with learning disabilities. *Journal of Learning Disabilities*, 27, 338–350.
- Lane, S. J., & Schaaf, R. C. (2010). Examining the neuroscience evidence for sensory-driven neuroplasticity: Implications for sensory-based occupational therapy for children and adolescents. *American Journal of Occupational Therapy*, 64, 375–390. <https://doi.org/10.5014/ajot.2010.09069>.
- May-Benson, T. A. (2004). Praxis is more than just motor planning: Clinical reasoning for understanding intervention for praxis. *OT Practice*, 9, CE1–CE7.
- May-Benson, T. A., & Koomar, J. A. (2010). Systematic review of the research evidence examining the effectiveness of interventions using a sensory integrative approach for children. *American Journal of Occupational Therapy*, 64, 403–414. <https://doi.org/10.5014/ajot.2010.09071>.
- Miller, L. J. (2006). *Sensational kids: Help and hope for children with sensory processing disorder*. New York: Putnam.
- Miller, L. J., Coll, J. R., & Schoen, S. A. (2007). A randomized controlled pilot study of the effectiveness of occupational therapy for children with sensory modulation disorder. *American Journal of Occupational Therapy*, 61, 228–238. <https://doi.org/10.5014/ajot.61.2.228>.
- Ottenbacher, K. (1982). Sensory integration therapy: Affect or effect. *American Journal of Occupational Therapy*, 36, 571–578.
- Parham, L. D., Cohn, E. S., Spitzer, S., Koomar, J. A., Miller, L. J., Burke, J. P., et al. (2007). Fidelity in sensory integration intervention research. *American Journal of Occupational Therapy*, 61, 216–227. <https://doi.org/10.5014/ajot.61.2.216>.
- Parham, L. D., Roley, S. S., May-Benson, T. A., Koomar, J., Brett-Green, B., Burke, J. P., et al. (2011). Development of a fidelity measure for research on the effectiveness of the Ayres Sensory Integration intervention. *American Journal of Occupational Therapy*, 65, 133–142. <https://doi.org/10.5014/ajot.2011.000745>.
- Pfeiffer, B. A., Koenig, K., Kinnealey, M., Sheppard, M., & Henderson, L. (2011). Research scholars initiative – Effectiveness of sensory integration interventions in children with autism spectrum disorders: A pilot study. *American Journal of Occupational Therapy*, 65, 76–85. <https://doi.org/10.5014/ajot.2011.09205>.
- Polatajko, H. J., Kaplan, B. J., & Wilson, B. N. (1992). Sensory integration treatment for children with learning disabilities: Its status 20 years later. *OTJR: Occupation, Participation and Health*, 12, 323–341.
- Roley, S. S., Mailloux, Z., Miller-Kuhaneck, H., & Glennon, T. (2007). Understanding Ayres Sensory Integration. *OT Practice*, 12, CE1–CE8.
- Schaaf, R. C., & Davies, P. L. (2010). Evolution of sensory integration frame of reference. *American Journal of Occupational Therapy*, 64, 363–367. <https://doi.org/10.5014/ajot.2010.090000>.
- Schaaf, R. C., & Miller, L. J. (2005). Occupational therapy using a sensory integrative approach for children with developmental disabilities. *Mental Disabilities and Developmental Disabilities Research Reviews*, 11, 143–148.
- Smith, S. A., Press, B., Koenig, K. P., & Kinnealey, M. (2005). Effects of sensory integration intervention on self-stimulating and self-injurious behaviors. *American Journal of Occupational Therapy*, 59, 418–425. <https://doi.org/10.5014/ajot.59.4.418>.
- Tomchek, S. D., & Case-Smith, J. (2009). *Occupational therapy practice guidelines for children and adolescents with autism*. Bethesda: AOTA Press.
- Vargas, S., & Camilli, G. (1999). A meta-analysis of research on sensory integration treatment. *American Journal of Occupational Therapy*, 53, 189–198. <https://doi.org/10.5014/ajot.53.2.189>.

Websites with Additional Information

www.siglobalnetwork.org
www.spdfoundation.net/
www.thespiralfoundation.org/

Sensory Integration and Praxis Test

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Synonyms

SIPT

Description

The *Sensory Integration and Praxis Tests* (SIPT; Ayres 1989) is a series of 17 subtests designed to measure sensory integration processes that underlie learning and behavior in children from 4 through 8 years, 11 months. This comprehensive, standardized assessment tool is considered the gold standard tool for evaluating sensory integration and praxis (motor planning) functions. Administration of the entire test generally takes 2½ h to complete, the examiner must follow specific procedures when administering the test (including what instructions can be provided to the child), and the child must be able to attend for long periods of time and follow the verbal directions. As a result, it may not be an appropriate testing instrument for all children on the autism spectrum.

There are 17 subtests within the SIPT, each yielding a standardized score by which the child's score is compared to those of typically developing children on praxis abilities and a variety of sensory processes, including tactile (touch), vestibular (movement/balance), proprioceptive/kinesthetic (where are the body parts in space and how do they work together), and visual (Table 1). The test focuses on how the child recognizes, discriminates, and perceives the sensory experience. It does not, however, have a specific scoring mechanism to identify if the child over- or underresponds to various sensory

experiences (referred to as sensory modulation, sensory underreactivity, and sensory overreactivity). For modulation type of information, please see the links below in the "See Also" section for the *Sensory Processing Measure*, *Sensory Processing Measure – Preschool*, and/or soon to be published *Sensory Processing Assessment*.

All SIPT results are scored via computer program, and while administering all 17 subtests is ideal, any combination can be implemented and still maintain the integrity of the standardized score. Following scoring, the computer generates a detailed report explaining the SIPT results. However, as the secondary purpose of the SIPT is to provide a foundational understanding of the child in order to outline appropriate intervention approaches, the ability to correctly implement and interpret the information provided by the SIPT is vital. Specialized training in the SIPT is not only strongly suggested and professionally responsible, but the therapist's name and professional qualifications are required in order to purchase the SIPT. Years ago, this specialty certification was offered by Sensory Integration International which was the organization originally created for the advancement and dissemination of Dr. Ayres' work. After the passing of Dr. Ayres, and some internal difficulties, this organization ceased offering these courses. Currently, there is only one training series in order for therapists to be certified in the administration and interpretation of the SIPT, offered jointly by the University of Southern California's Division of Occupational Science and Occupational Therapy and Western Psychological Services (USC/WPS) and approved by the Successor Trustee of Dr. Ayres' estate (holder of the trademark term Ayres Sensory Integration®).

This certification includes a series of four, 5 day courses:

- Theoretical foundations of sensory integration
- Technical and logistical administration of the SIPT as well as other commercially available measures of sensory processing
- Interpretation of the SIPT's standardized information and interfacing that information with other nonstandardized observations and

Sensory Integration and Praxis Test, Table 1 The 17 SIPT subtests

Test name	Description
Space Visualization	This test evaluates visual space perception and mental manipulation of objects in space. Motor performance is not required – therefore, deliberately visual and not visual-motor
Figure Ground	This test requires the child to select a foreground figure from a competing background
Standing and Walking Balance	This test evaluates the child's ability to balance on one or both feet, both statically and dynamically, with eyes open and closed
Design Copying	This section evaluates visuopractic and visuoconstruction skills. It assesses both accuracy and approach in copying designs
Postural Praxis	This part of the test evaluates the child's ability to imitate positions or postures demonstrated by the examiner
Bilateral Motor Coordination	This test measures the ability of the child to move both arms and both feet together in a smoothly integrated pattern
Praxis on Verbal Command	This test measures the child's ability to motor plan body postures from verbal directions without visual cues
Constructional Praxis	This test measures the child's ability to relate objects to each other in three-dimensional space. It involves visual spatial understanding and motor planning a course of action to replicate simple and complex block structures Constructional Praxis involves ideation, conceptualization, space perception, and planning
Postrotary Nystagmus	This test evaluates the integrity of a relatively discrete vestibular-ocular reflex following rotation. It is the only test of the SIPT that is reflexive rather than performance based
Motor Accuracy	This test is a visuomotor test which assesses eye-hand coordination in a wide variety of positions relative to the body, including crossing the body's midline
Sequencing Praxis	This test is designed to measure the child's ability to repeat a series of hand and finger movements following demonstration by the examiner
Oral Praxis	This test measures the child's ability to plan and execute tongue, jaw, and lip movements following demonstration by the examiner
Manual Form Perception	Part I – involves identifying the visual counterpart of a geometric form held and manipulated in the hand Part II – involves feeling a shape, without the aid of vision or visual cues with one hand and finding the matching shape among a line of blocks manipulated with the other hand, without the aid of vision and visual cues
Kinesthesia	This test assesses the capacity to perceive joint position and movement
Finger Identification	This test measures the ability to identify which finger or fingers are touched by the examiner with vision occluded
Graphesthesia	This test evaluates the ability to translate tactile input into a motor response. It assesses the integration between tactile and visual input and fine motor planning
Localization of Tactile Stimuli	This test assesses the child's ability to localize a specific tactile stimulus, applied to the hand or arm, with vision occluded

reports of the child's performance in order to develop appropriate intervention plans

- Intervention planning based on the theory of sensory integration, including practical applications and clinical reasoning strategies

Please note, however, that the USC/WPS website does not list therapists who were certified by the original organization sanctioned to offer the Certification of the Administration and Interpretation of the SIPT. As a result, parents and

professionals should not limit themselves to only those therapists registered with the USC/WPS organization.

Historical Background

The SIPT, years in development, was intended to help clinicians clinically understand the aspects of sensory processing and praxis abilities which contributed to irregularities in learning and behavior.

The theoretical model of sensory integration was the basis of the tool. This model highlighted how there are neurological processes which organize sensation from within one's own body and from the environment upon which an individual relies in order to use the body effectively to meet the demands of a given situation. It was theorized at the time that the body's receptors not only needed to accurately receive sensory stimuli but needed to efficiently process and organize this sensory information – i.e., select the needed information to make a response, inhibit information not needed, compare and synthesize the current information with past information stored within the brain, and relate the information to the current demand for action. The ability to flexibly and constantly perform this type of integration of information is necessary to perform functionally within an ever-changing environment. More specifically, the ability of a child to correctly receive, recognize, and integrate sensations from the body allows the child to “know” his/her body and develop plans for how to use the body for successful engagement in life's developmental tasks. When the child successfully plans and carries out an action, we call this action an “adaptive response” for a given situation or environmental demand.

The SIPT was designed to assess how the child perceives sensory information (tactile, proprioceptive/kinesthetic, vestibular, and visual) and how the body is able to plan or “do” requested motor actions.

Psychometric Data

In addition to the intent to create a reliable measurement and analysis of data by accepted statistical processes, the guiding principle in determining the procedures ultimately outlined in the SIPT was the “capacity of each measure to discriminate between dysfunctional and non-dysfunctional children of normal intelligence” (Ayres 1989, p. 161).

The standardization procedures began by ensuring a nationally representative sample based on the 1980 U.S. Census Bureau data,

including age, sex, ethnicity, rural/urban, and geographic location within the USA. Approximately 2,000 children were part of the standardization sample and testing occurred during the 1984–1985 school year. No children were included if there was an identified motor impairment (i.e., cerebral palsy) or severe visual handicap, and a number of children from Canada were included as it was expected that the tool would be utilized in that country.

Preliminary analyses examined age- and gender-related differences (multivariate analysis of variance [MANOVA]). Additionally, item data were examined to determine appropriate points to discontinue or stop the test without changing the outcome if the entire test had been completed (Pearson product-moment correlations and multiple discriminant analyses). Thereafter, statistical computation of group means and standard deviations was completed whereby separate norms were developed for each of the 12 age groups for girls and boys. Next, major SIPT scores were determined based on the extent by which they could discriminate between normal and dysfunctional performance.

A wide variety of studies were conducted to investigate construct, content, and criterion-related validity:

- Construct validity: the extent to which the test assesses the theoretical construct
- Content validity: the extent to which the test items provide a representative sampling of performance on important aspects of the construct
- Criterion-related validity: the extent to which performance on the test can be used to predict the child's current or future performance on important aspects of the construct (Ayres 1989, p. 181)

A detailed analysis of the SIPT's construct occurred via factor analyses (tests are grouped together with one underlying, common theme) and cluster analysis (children are grouped if they are alike). This initial information, as well as subsequent analyses, are presented in Table 2.

This information is important as the utility of a tool is whether the test can successfully and

Sensory Integration and Praxis Test, Table 2 SIPT factor and cluster analyses

Factor analyses (Ayres 1989)	Factor analysis (Mulligan 1998, p. 258)
• Bilateral integration and sequencing • Praxis on verbal command • Somatosensory and oral praxis • Visuopraxis • Somatopraxis	• Dyspraxia • Visual-perceptual deficit • Somatosensory deficit • Bilateral integration and sequencing deficit “Mulligan demonstrated that these four factors were highly correlated with one another, and proposed that they all relate to one underlying construct she names general praxis dysfunction.”
Cluster analyses (Ayres 1989)	Cluster analyses (Mulligan 2000, p. 258)
• Group 1: Low Average Bilateral Integration and Sequencing • Group 2: Generalized Sensory Integrative Dysfunction • Group 3: Visuo- and Somatodyspraxia • Group 4: Low Average Sensory Integration and Praxis • Group 5: Dyspraxia on Verbal Command • Group 6: High Average Sensory Integration and Praxis	• Cluster 1: Generalized Sensory Integration Dysfunction and Dyspraxia – Severe • Cluster 2: Dyspraxia • Cluster 3: Generalized Sensory Integration Dysfunction and Dyspraxia – Moderate • Cluster 4: Low Average Bilateral Integration and Sequencing • Cluster 5: Average Sensory Integration and Praxis

accurately identify clinically important groups of children who would be in need of different types of remediation or support.

Mulligan’s (1998) confirmatory factor analysis utilizing the SIPT scores of more than 10,000 children and cluster analyses (2000) provide clinicians with additional information useful in identifying deficits and planning interventions. Additionally, Davies and Tucker (2010) and Mailloux et al. (2011) provided additional perspectives on these analyses for practitioners to plan specific intervention strategies.

Clinical Uses

The SIPT is the only standardized assessment tool specifically designed to identify various types of sensory dysfunction. As stated above, however, sensory modulation issues are not specifically identified. As sensory modulation is an important component of sensory processing within the population of children identified as on the autism spectrum, other assessments might be more useful. Additionally, if the child to be evaluated is unable to follow only the stated directions or

attend for a long period of time, this assessment tool might not be appropriate.

See Also

- ▶ [Ayres, A. Jean](#)
- ▶ [Evaluation of Sensory Processing](#)
- ▶ [Occupational Therapy \(OT\)](#)
- ▶ [Sensory Diet](#)
- ▶ [Sensory Integration \(SI\) Therapy](#)
- ▶ [Sensory Processing](#)
- ▶ [Sensory Processing Assessment](#)
- ▶ [Sensory Processing Measure](#)
- ▶ [Sensory Processing Measure: Preschool \(SPM-P\)](#)
- ▶ [Test of Sensory Functioning in Infants](#)

References and Reading

Ayres, A. J. (1989). *The sensory integration and praxis tests*. Los Angeles: Western Psychological Services.

Davies, P. L., & Tucker, R. (2010). Evidence review to investigate the support for subtypes of children with difficulty processing and integrating sensory information. *American Journal of Occupational Therapy*, 64, 391–402.

- Mailloux, Z., Mulligan, S., Roley, S. S., Blanche, E., Cermak, S., Coleman, G. G., et al. (2011). Verification and clarification of patterns of sensory integrative dysfunction. *American Journal of Occupational Therapy*, 65, 143–151.
- Mulligan, S. (1998). Patterns of sensory integration dysfunction: A confirmatory factor analysis. *American Journal of Occupational Therapy*, 52, 819–828.
- Mulligan, S. (2000). Cluster analysis of scores of children on the Sensory Integration and Praxis Tests. *Occupational Therapy Journal of Research: Occupation, Participation, and Health*, 20, 256–270.

Sensory Integration and Praxis Tests (SIPT)

► Touch Sensitivity

Sensory Overresponsivity as a Predictor of Amplitude Discrimination Performance in Youth with ASD

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Definition

The term “sensory overresponsivity” has been defined as an exaggerated, rapid-onset, or prolonged reaction to sensory stimulation (Ben-Sasson et al. 2008). It is estimated that 56 to 70 percent of youth with autism spectrum disorder (ASD) demonstrate sensory overresponsivity (Baranek et al. 2006). Sensory overresponsivity has also been associated with greater anxiety, social impairment, and functional impairment in individuals with ASD (Cascio et al. 2016; Green and Ben-Sasson 2010; Liss et al. 2006), making it a clinical construct of relevance for

this population. Though sensory overresponsivity has been documented in parent reports across the sensory modalities, atypical responses to touch are particularly highlighted in self- and parent reports and have been the focus of a substantive amount of experimental research (Cascio et al. 2012; Kaiser et al. 2015; Marco et al. 2012).

In particular, tactile processing in those with ASD has been investigated using psychophysical methods, which help to provide further insight into the neurobiological bases of sensory reactivity via objective, quantitative metrics. Tactile processing can be delineated into several components, including detection, discrimination, and adaptation. Tactile detection can be measured by flutter, vibration, and contact stimulation and indicates the point at which an individual is able to perceive the stimulus itself. Tactile discrimination refers to the ability to differentiate sensory information among various incoming stimuli. Finally, adaptation encompasses the impact of repetitive stimulation on a subsequent stimulus (Kohn 2007). Research with typically developing (TD) adults has indicated that the presence of an adapting stimulus increases the threshold for tactile discrimination of a subsequent stimulus (Tannan et al. 2008; Tommerdahl et al. 2010). That is, the adapting stimulus tends to reduce the perceived intensity of the later stimuli, making it more difficult to detect. Puts et al. (2014) investigated tactile processing using a tactile psychophysical battery in a group of TD children and children with ASD. Puts et al. (2014) found that performance on an amplitude discrimination task significantly worsened after single-site adaptation compared with a no-adaptation condition for TD children, whereas an increasing subthreshold stimulus did not have a significant effect on the performance of children with ASD on the same task. Such results imply that individuals with ASD demonstrate difficulties in their ability to habituate to incoming sensory information in comparison with TD individuals, such that they do not adjust as readily to prior sensory input.

Testing the effect of repetitive or long-duration stimuli on tactile processing thus provides a window into the ability to habituate to sensory information (Mikkelsen et al. 2018). Habituation is a

process by which repeated application of a stimulus results in a progressive decrease of a response over time. In general, habituation allows us to filter out irrelevant stimuli while focusing on important stimuli (Rankin et al. 2009). Several studies have suggested that habituation is reduced in individuals with autism (Guiraud et al. 2011; Kemner et al. 2002). In a study of habituation to repeated sounds in infants at high risk for autism, Guiraud et al. (2011) posited that reduced habituation may result in an inability to discriminate novel from repeated sounds, which may produce an increased response to repeated sensory input.

Given that a reduced ability to habituate may result in experiencing repeated sensory information as novel, this repeated stimulation may give rise to a behavioral experience of sensory overload. Consequently, it is possible that behavioral performance on tactile processing tasks is related to the construct of sensory overresponsivity. McKernan et al. (2019) used a set of amplitude discrimination tasks from the same tactile psychophysical battery as Puts et al. (2014) in a group of TD children and children with ASD. McKernan et al. (2019) found that sensory overresponsivity, as measured by caregiver report, predicted amplitude discrimination performance with an adapting stimulus as well as the effect of adaptation in the cohort of youth with ASD. It was also found that performance on the amplitude discrimination with adaptation task significantly predicted sensory overresponsivity scores among children with ASD, but not TD children, suggesting a bidirectional relationship between sensory overresponsivity and the effect of adaptation. Consequently, it is possible that sensory overresponsivity is the mechanism underlying the previously observed lack of an adaptation effect among children and adults with ASD.

Historical Background

Sensory sensitivities have long been reported in individuals with ASD, dating back to the 1940s with Leo Kanner's initial description of autism. Children, adolescents, and adults with ASD have been found to have significantly elevated levels of

sensory symptoms as compared to individuals with typical development and general developmental delay (Ben-Sasson et al. 2009; Failla et al. 2017; Rogers and Ozonoff 2005). Further, the severity of these sensory features is related to adaptive functioning among children with ASD (Rogers et al. 2003). Differences in sensory processing can be framed in terms of an increased or adverse response (hyperreactivity) or a decreased or lessened response (hyporeactivity) to sensory stimuli (Dunn and Brown 1997; Siper et al. 2017). Importantly, the degree and profile of sensory symptoms are highly varied in those with ASD (Cascio 2010), with many of these patterns co-occurring in the same individual, such that he or she may demonstrate hyporesponsivity in one sensory modality and hyperresponsivity in another (Ausderau et al. 2014; Iarocci and McDonald 2006; Lane et al. 2011). Sensory overresponsivity has been previously described as an exaggerated, rapid-onset, or prolonged reaction to sensation (Ben-Sasson et al. 2008), with behavioral features including avoidance of or prolonged negative responses to sensory stimuli (Liss et al. 2006). Traditionally, sensory overresponsivity has been measured by a combination of caregiver report (e.g., the Sensory Profile; Dunn 1999) and direct observation (Crane et al. 2009; Tavassoli et al. 2014; Wodka et al. 2016). In addition, specific measures of sensory overresponsivity have been developed, including the Sensory Overresponsivity Inventory (Schoen et al. 2008) and the Sensory Experience Questionnaire (Baranek et al. 2006). Importantly, however, questionnaire measures that assess sensory reactivity do not always converge with psychophysical thresholds indexing tactile sensitivity. In part, this may be due to the wide range of questions posed on questionnaire measures, many of which extend beyond the realm of pure sensory experience into perceptual, emotional, and cognitive responses associated with sensation. Conversely, psychophysical measures may provide a more specific measurement of individuals' behavioral responses to tactile input.

Use of psychophysical methods in the examination of tactile processing stems from the work of Weber and Fechner in the nineteenth century,

which gave rise to the investigation of detection thresholds and the just-noticeable difference. It is only in the last two decades, however, that psychophysical methods have been used to investigate tactile processing in individuals with ASD. A variety of metrics, including tactile detection thresholds, adaptation, and textures, have been used to expand inquiry yet have often produced conflicting results.

With regard to detection thresholds, a small body of literature has found no difference between adults with ASD and TD adults in contact detection threshold, as measured by Von Frey hairs (O’Riordan and Passetti 2006) and flutter stimulation (Blakemore et al. 2006), as well as children with and without ASD, as measured by flutter and vibration stimulation (Guclu et al. 2007). On the other hand, however, decreased flutter perception has also been reported (Puts et al. 2014; Tavassoli et al. 2016). It is likely that small sample sizes and differences in methodology contribute to the lack of clear conclusions in this regard (Mikkelsen et al. 2018). In addition, the large attentional demands inherent to such tasks make psychophysical studies more difficult, and consequently rarer, in samples of children.

In terms of behavioral response to textures, Cascio et al. (2016) investigated tactile defensiveness in children with typical development, ASD, and other developmental disabilities. Participants received passive touch from unpleasant, pleasant, and social textures (plastic mesh, soft fleece, and an experimenter’s finger, respectively) at three sites on the upper body. Children with ASD had significantly higher tactile defensiveness reactions and lower pleasantness ratings compared to the TD group, with the most pleasant material and the social touch conditions distinguishing the children with ASD and other developmental disabilities from TD children on tactile defensiveness. These results are in fitting with previous research with a group of adults with and without ASD by Cascio et al. (2012), which used functional magnetic resonance imaging (fMRI) to investigate responses to somatosensory stimulation with three textures spanning a range of roughness and pleasantness. Adults with ASD exhibited less activation in the primary somatosensory cortex for

neutral and pleasant stimuli but demonstrated greater response for unpleasant stimuli. Cascio et al. (2012) noted that these results point to the possibility of a predisposition to hyperresponsiveness to unpleasant tactile stimulation and hyporesponsiveness to positive or neutral tactile stimuli in those with ASD.

Current Knowledge

Across multiple studies, the effect of adaptation – presumed to be an increase or sharpening in contrast between stimuli – has been observed to be absent in both children and adults with ASD (Tommerdahl et al. 2007, 2008; Puts et al. 2014). A strong body of literature has proposed that GABA, an inhibitory neurotransmitter, may explain altered tactile processing in individuals with ASD. Indeed, previous research has found evidence for reduced GABA levels within sensory regions in children with ASD (Gaetz et al. 2014; Rojas et al. 2014). In addition to impairments in the inhibitory system in individuals with ASD, another potential contributor to the lack of an adaptation effect may be behavioral, as a result of overactive sensory responses.

McKernan et al. (2019) investigated the relationship between sensory overresponsivity, defined as an exaggerated response to sensory stimulation, and performance on three vibrotactile tasks: amplitude discrimination without adaptation at a single site, amplitude discrimination without adaptation at two sites, and amplitude discrimination with single-site adaptation. Participants included 25 TD children and 25 children with ASD between the ages of 6 and 17 years. Sensory overresponsivity was measured via a composite of items on the Sensory Profile, a caregiver questionnaire assessing children’s responses to various sensory experiences. The effect of adaptation was measured by the difference in performance on tasks with and without an adapting stimulus. Sensory overresponsivity did not significantly predict the difference on any of the amplitude discrimination tasks for TD youth. In contrast, sensory overresponsivity significantly predicted performance on the single-site

adaptation task, as well as the difference between adaptation and non-adaptation, for youth with ASD. The reverse relationships were also tested to determine whether performance on the amplitude discrimination tasks was predictive of sensory overresponsivity scores. None of the three tasks significantly predicted sensory overresponsivity in the TD group; however, the single-site adaptation task was found to significantly predict sensory overresponsivity in the ASD cohort. Taken together, these results imply that sensory overresponsivity has a unique and differential impact on tactile processing in children with ASD, such that the level of sensory overresponsivity may be a significant contributor to the reduction of adaptation effects among those with ASD.

Other research into the phenomenon of sensory overresponsivity has used neuroscience methods, such as fMRI, to investigate the brain's response to sensory stimulation. Green et al. (2019) exposed children and adolescents with ASD and typical development to two consecutive paradigms: an initial exposure phase, during which they experienced auditory, tactile, and joint (simultaneous auditory and tactile) stimulation, followed by a generalization phase, in which they experienced novel auditory, tactile, and joint stimulation. Tactile stimuli consisted of two scratchy sponges rubbed on participants' inner arms at a rate of one stroke per second. The ASD group was divided into youth with high and low levels of sensory overresponsivity. Green et al. (2019) found that participants with ASD in the high sensory overresponsivity group demonstrated a reduced ability to maintain habituation in the relevant sensory cortices and amygdala and to maintain inhibition in irrelevant sensory cortices. In this manner, it appears that children and adolescents with ASD with high levels of sensory overresponsivity were unable to maintain downregulation of brain responses across time and with a novel stimulus. Taken together, these results indicate that sensory habituation is a particular difficulty for those with ASD with high overresponsivity, which may also help to explain the lack of adaptation effect seen in previous psychophysics studies.

Future Directions

Although sensory overresponsivity has been shown to be predictive of performance on an amplitude discrimination task among children with ASD, its relationship to other tactile psychophysical tasks has yet to be investigated. As such, it is unknown which specific aspects of tactile processing are most associated with the construct of sensory overresponsivity and whether this relationship is unique to amplitude discrimination or is common among other psychophysical tasks. Further research regarding how these constructs relate across different sensory modalities, such as audition or vision, is also needed.

Previous research has also linked differences in tactile processing among individuals with ASD to altered GABA function in this population (Puts et al. 2011, 2017; Tavassoli et al. 2016). Much remains unknown regarding to what extent altered GABA functioning may explain the construct of sensory overresponsivity. Further exploration of this relationship may uncover a common mechanism responsible for observed differences in tactile processing among those with ASD.

Future research may also wish to consider the influence of sex differences upon the construct of sensory overresponsivity, as well as performance on tactile psychophysics tasks. Given the discrepancy in sex ratio in population estimates of ASD prevalence (Baio et al. 2018), many studies include a greater number of male than female participants. To date, no studies have explicitly examined the impact of sex differences on the relationship between sensory overresponsivity and the effect of adaptation. Further exploration into sex differences in sensory overresponsivity, as well as performance on tactile psychophysics tasks in general, is needed.

In addition, the measurement of the construct of sensory overresponsivity itself remains imperfect, as no one objective metric currently exists. The combination of caregiver and/or self-report measures of sensory processing, along with the use of a clinician-rated behavioral measure, may help to provide a thorough and more valid assessment of sensory overresponsivity; however, some degree of subjectivity is inherent in each of these

approaches. Innovative approaches, including the use of neuroscience methods such as fMRI, may provide an objectively empirical measurement of sensory overresponsivity.

Finally, given the substantial impact of sensory overresponsivity on quality of life, including symptoms of anxiety and functional impairment, in those with ASD (Green and Ben-Sasson 2010; Liss et al. 2006), future research would do well to develop and test specialized interventions related to sensory overresponsivity. Interventions may be at both an individual and a systems level, with a focus on increasing coping strategies in individuals with ASD who have high sensory overresponsivity, as well as modifying external home, school, and work environments to accommodate and support the needs of those with sensory processing differences. Distinctive interventions may be created for specific sensory modalities, in fitting with the heterogeneous profile of sensory processing differences among those with ASD. Such interventions will enable individuals with sensory overresponsivity and ASD to participate more fully in their communities, while respecting the unique way in which they perceive their sensory worlds.

See Also

- ▶ [Hyperresponsiveness](#)
- ▶ [Hyporesponsiveness](#)
- ▶ [Sensory Processing](#)
- ▶ [Sensory Stimuli](#)
- ▶ [Tactile](#)
- ▶ [Tactile Temporal Resolution](#)

References and Reading

- Ausderau, K., Sideris, J., Furlong, M., Little, L. M., Bulluck, J., & Baranek, G. T. (2014). National survey of sensory features in children with ASD: Factor structure of the sensory experience questionnaire 3.0. *Journal of Autism and Developmental Disorders*, 44, 915–925. <https://doi.org/10.1007/s10803-013-1945-1>.
- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., et al. (2018). Prevalence of autism spectrum disorder among children aged 8 years—Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summaries*, 67(SS-6), 1–23. <https://doi.org/10.15585/mmwr.ss6706a1>.
- Baranek, G. T., David, F. J., Poe, M. D., Stone, W. L., & Watson, L. R. (2006). Sensory experiences questionnaire: Discriminating sensory features in young children with autism, developmental delays, and typical development. *Journal of Child Psychology and Psychiatry*, 47(6), 591–601. <https://doi.org/10.1111/j.1469-7610.2005.01546.x>.
- Ben-Sasson, A., Cermak, S. A., Orsmond, G. I., Tager-Flusberg, H., Kadlec, M. B., & Carter, A. S. (2008). Sensory clusters of toddlers with autism spectrum disorders: Differences in affective symptoms. *Journal of Child Psychology and Psychiatry*, 49, 817–825. <https://doi.org/10.1111/j.1469-7610.2008.01899.x>.
- Ben-Sasson, A., Hen, L., Fluss, R., Cermak, S. A., Engel-Yeger, B., & Gal, E. (2009). A meta-analysis of sensory modulation symptoms in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 1–11. <https://doi.org/10.1007/s10803-008-0593-3>.
- Blakemore, S. J., Tavassoli, T., Calo, S., Thomas, R. M., Catmur, C., Frith, U., & Haggard, F. (2006). Tactile sensitivity in Asperger syndrome. *Brain Cognition*, 61, 5–13. <https://doi.org/10.1016/j.bandc.2005.12.013>.
- Cascio, C. J. (2010). Somatosensory processing in neurodevelopmental disorders. *Journal of Neurodevelopmental Disorders*, 2, 62–69. <https://doi.org/10.1007/s11689-010-9046-3>.
- Cascio, C. J., Moana-Filho, E. J., Guest, S., Nebel, M. B., Weisner, J., Baranek, G. T., & Essick, G. K. (2012). Perceptual and neural response to affective tactile texture stimulation in adults with autism spectrum disorders. *Autism Research*, 5(4), 231–244. <https://doi.org/10.1002/aur.1224>.
- Cascio, C. J., Lorenzi, J., & Baranek, G. T. (2016). Self-reported pleasantness ratings and examiner-coded defensiveness in response to touch in children with ASD: Effects of stimulus material and bodily location. *Journal of Autism and Developmental Disorders*, 46, 1528–1537. <https://doi.org/10.1007/s10803-013-1961-1>.
- Crane, L., Goddard, L., & Pring, L. (2009). Sensory processing in adults with autism spectrum disorders. *Autism*, 13(3), 215–228.
- Dunn, W. (1999). *Sensory profile: User's manual*. San Antonio: Psychological Corporation.
- Dunn, W., & Brown, C. (1997). Factor analysis on the sensory profile from a national sample of children without disabilities. *American Journal of Occupational Therapy*, 51(7), 490–495.
- Failla, M. D., Peters, B. R., Karbasforoushan, H., Foss-Feig, J. H., Schauder, K. B., Heflin, B. H., & Cascio, C. J. (2017). Intra-insular connectivity and somatosensory responsiveness in young children with ASD. *Molecular Autism*, 8, 25. <https://doi.org/10.1186/s13229-017-0143-y>.
- Gaetz, W., Bloy, L., Wang, D. J., Port, R. G., Blaskey, L., Levy, S. E., & Roberts, T. P. (2014). GABA estimation

- in the brains of children on the autism spectrum: Measurement precision and regional cortical variation. *NeuroImage*, 86, 1–9. <https://doi.org/10.1016/j.neuroimage.2013.05.068>.
- Green, S. A., & Ben-Sasson, A. (2010). Anxiety disorders and sensory overresponsivity in children with autism spectrum disorders: Is there a causal relationship? *Journal of Autism and Developmental Disorders*, 40, 1495–1504. <https://doi.org/10.1007/s10803-010-1007-x>.
- Green, S. A., Hernandez, L., Lawrence, K. E., Liu, J., Tsang, T., Yeargin, J., et al. (2019). Distinct patterns of neural habituation and generalization in children and adolescents with autism with low and high sensory overresponsivity. *The American Journal of Psychiatry*, xx. <https://doi.org/10.1176/appi.ajp.2019.18121333>.
- Gucul, B., Tanidir, C., Mukaddes, N. M., & Ünal, F. (2007). Tactile sensitivity of normal and autistic children. *Somatosensory and Motor Research*, 24(1–2), 21–33. <https://doi.org/10.1080/08990220601179418>.
- Guiraud, J. A., Kushnerenko, E., Tomalski, P., Davies, K., Ribeiro, H., Johnson, M. H., & BASIS Team. (2011). Differential habituation to repeated sounds in infants at high risk for autism. *Neuroreport*, 22(16), 845–849.
- Iarocci, G., & McDonald, J. (2006). Sensory integration and the perceptual experience of persons with autism. *Journal of Autism and Developmental Disorders*, 36, 77–90. <https://doi.org/10.1007/s10803-005-0044-3>.
- Kaiser, M. D., Yang, D. Y. J., Voos, A. C., Bennett, R. H., Gordon, I., Pretzsch, C., et al. (2015). Brain mechanisms for processing affective (and nonaffective) touch are atypical in autism. *Cerebral Cortex*, 26(6), 2705–2714. <https://doi.org/10.1093/cercor/bhv125>.
- Kemner, C., Orange, B., Verbaten, M. N., & Van Engeland, H. (2002). Normal P50 gating in children with autism. *Journal of Clinical Psychiatry*, 63, 214–217. <https://doi.org/10.4088/jcp.v63n0307>.
- Kohn, A. (2007). Visual adaptation: Physiology, mechanisms, and functional benefits. *Journal of Neurophysiology*, 97, 3155–3164.
- Lane, A. E., Young, R. L., Baker, A. E. Z., & Angley, M. T. (2011). Sensory processing subtypes in autism: Association with adaptive behavior. *Journal of Autism and Developmental Disorders*, 40(1), 112–122. <https://doi.org/10.1007/s10803-009-0840-2>.
- Liss, M., Saulnier, C., Fein, D., & Kinsbourne, M. (2006). Sensory and attention abnormalities in autistic spectrum disorders. *Autism*, 10(2), 155–172. <https://doi.org/10.1177/13623613060062021>.
- Marco, E. J., Khatibi, K., Hill, S. S., Siegel, B., Arroyo, M. S., Dowling, A. F., et al. (2012). Children with autism show reduced somatosensory response: An MEG study. *Autism Research*, 5, 340–351. <https://doi.org/10.1002/aur.1247>.
- McKernan, E. P., Wu, Y., & Russo, N. (2019). Sensory overresponsivity as a predictor of amplitude discrimination performance in youth with ASD. *Journal of Autism and Developmental Disorders*, xx, 1–9. <https://doi.org/10.1007/s10803-019-04013-0>.
- Mikkelsen, M., Wodka, E. L., Mostofsky, S. H., & Puts, N. A. J. (2018). Autism spectrum disorder in the scope of tactile processing. *Developmental Cognitive Neuroscience*, 29, 140–150. <https://doi.org/10.1016/j.dcn.2016.12.005>.
- O’Riordan, M., & Passetti, F. (2006). Discrimination in autism within different sensory modalities. *Journal of Autism and Developmental Disorders*, 36, 665–675. <https://doi.org/10.1007/s10803-006-0106-1>.
- Puts, N. A. J., Edden, R. A. E., Evans, C. J., McGlone, F., & McGonigle, D. J. (2011). Regionally specific human GABA concentration correlates with tactile discrimination thresholds. *Journal of Neuroscience*, 31(46), 16556–16560. <https://doi.org/10.1523/JNEUROSCI.4489-11.2011>.
- Puts, N. A. J., Wodka, E. L., Tommerdahl, M., Mostofsky, S. H., & Edden, R. A. E. (2014). Impaired tactile processing in children with autism spectrum disorder. *Journal of Neurophysiology*, 111, 1803–1811. <https://doi.org/10.1152/jn.00890.2013>.
- Puts, N. A. J., Wodka, E. L., Harris, A. D., Crocetti, D., Tommerdahl, M., Mostofsky, S. H., & Edden, R. A. E. (2017). Reduced GABA and altered somatosensory function in children with autism spectrum disorder. *Autism Research*, 10(4), 608–619. <https://doi.org/10.1002/aur.1691>.
- Rankin, C. H., Abrams, T., Barry, R. J., Bhatnagar, S., Clayton, D. F., Colombo, J., et al. (2009). Habituation revisited: An updated and revised description of the behavioral characteristics of habituation. *Neurobiology of Learning and Memory*, 92(2), 135–138. <https://doi.org/10.1016/j.nlm.2008.09.012>.
- Rogers, S. J., & Ozonoff, S. (2005). Annotation: What do we know about sensory dysfunction in autism? A critical review of the empirical evidence. *Journal of Child Psychology and Psychiatry*, 46(12), 1255–1268. <https://doi.org/10.1111/j.1469-7610.2005.01431.x>.
- Rogers, S. J., Hepburn, S., & Wehner, E. (2003). Parent reports of sensory symptoms in toddlers with autism and those with other developmental disorders. *Journal of Autism and Developmental Disorders*, 33, 631–642. <https://doi.org/10.1023/B:JADD.0000006000.38991.a7>.
- Rojas, D. C., Singel, D., Steinmetz, S., Hepburn, S., & Brown, M. S. (2014). Decreased left perisylvian GABA concentration in children with autism and unaffected siblings. *NeuroImage*, 86, 28–34. <https://doi.org/10.1016/j.neuroimage.2013.01.045>.
- Schoen, S. A., Miller, L. J., & Green, K. E. (2008). Pilot study of the sensory over-responsivity scales: Assessment and inventory. *American Journal of Occupational Therapy*, 62(4), 393–406. <https://doi.org/10.5014/ajot.62.4.393>.
- Siper, P. M., Kolevzon, A., Wang, A. T., Buxbaum, J. D., & Tavassoli, T. (2017). A clinician-administered observation and corresponding caregiver interview capturing DSM-5 sensory reactivity symptoms in children with ASD. *Autism Research*, 10(6), 1133–1140. <https://doi.org/10.1002/aur.1750>.

- Tannan, V., Holden, J. K., Zhang, Z., Baranek, G. T., & Tommerdahl, M. A. (2008). Perceptual metrics of individuals with autism provide evidence for disinhibition. *Autism Research*, 1(4), 223–230. <https://doi.org/10.1002/aur.34>.
- Tavassoli, T., Hoekstra, R. A., & Baron-Cohen, S. (2014). The sensory perception quotient (SPQ): Development and validation of a new sensory questionnaire for adults with and without autism. *Molecular Autism*, 5(1), 29. <https://doi.org/10.1186/2040-2392-5-29>.
- Tavassoli, T., Bellesheim, K., Tommerdahl, M., Holden, J. M., Kolevzon, A., & Buxbaum, J. D. (2016). Altered tactile processing in children with autism spectrum disorder. *Autism Research*, 9, 616–620. <https://doi.org/10.1002/aur.1563>.
- Tommerdahl, M., Tannan, V., Cascio, C. J., Baranek, G. T., & Whitsel, B. L. (2007). Vibrotactile adaptation fails to enhance spatial localization in adults with autism. *Brain Research*, 1154, 116–123. <https://doi.org/10.1016/j.brainres.2007.04.032>.
- Tommerdahl, M., Tannan, V., Holden, J. K., & Baranek, G. T. (2008). Absence of stimulus-driven synchronization effects on sensory perception in autism: Evidence for local underconnectivity? *Behavioral and Brain Functions*, 4(19). <https://doi.org/10.1186/1744-9081-4-19>.
- Tommerdahl, M., Favorov, O. V., & Whitsel, B. L. (2010). Dynamic representations of the somatosensory cortex. *Neuroscience and Biobehavioral Reviews*, 34, 160–170. <https://doi.org/10.1016/j.neubiorev.2009.08.009>.
- Wodka, E. L., Puts, N. A., Mahone, E. M., Edden, R. A. E., & Mostofsky, S. H. (2016). The role of attention in somatosensory processing: A multi-trait, multimethod analysis. *Journal of Autism and Developmental Disorders*, 46, 3232–3241. <https://doi.org/10.1007/s10803-016-2866-6>.

Sensory Processing

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Definition

Sensory processing is a general term that describes the way that the central and peripheral nervous systems manage incoming sensory stimuli and allows for an organized and adaptive response to the presented sensory information (Lane et al. 2000). Through sensory registration,

the peripheral nervous system detects the input and relays the specific information to the brain. Sensory integration allows the organization of input from the different sensory systems to lead to an appropriate, adaptive response (Tomchek 2010). Sensory integration incorporates sensory modulation which allows us to notice important sensory information and to ignore irrelevant sensory stimuli. Deficits in sensory modulation may result in inattentiveness, sensory seeking behaviors, or avoidance of sensory input. Additionally, individuals with deficits in sensory modulation may also exhibit difficulty regulating their state of alertness and arousal, and fluctuations within these states may be evident. Maintaining a regulated emotional state may be challenging for some individuals with sensory processing deficits as well. Emotional lability may be noted or even a flattened affect. Adults with sensory processing difficulties may ultimately display sensory defensiveness as well as difficulties with discrimination, motor planning, and motor performance (May-Benson 2011).

Individuals with sensory processing deficits exhibit difficulty managing input from one or more of the sensory systems. The visual system within the context of sensory processing refers to the way the brain interprets visual information, separate from visual activity. Individuals with deficits in visual sensory processing may exhibit difficulty with distinguishing relevant visual information embedded in a rival background, such as reading from a chalkboard or whiteboard. Some individuals who may seek more intensive visual information may look at objects from an unusual perspectives or angles or engage in finger gazing.

Auditory (sensory) processing refers to the way in which the brain manages auditory stimuli, though separate from hearing. Individuals with deficits in auditory sensory processing may exhibit difficulty filtering out background noise while trying to listen to a classroom lecture. Exaggerated reactions to loud or unexpected noises may also be evident. Some individuals will cover their ears to such noises and may even avoid settings in which loud noises naturally occur. Other children with auditory sensory

processing difficulties may seek noises and may even engage in repetitive noise making to provide themselves with this type of input.

The olfactory and gustatory systems refer to the systems of taste and smell, respectively. Deficits in either one of these systems may affect food selectivity, an area of concern for some children with autism spectrum disorders. Some children may seek out intensive smells or tastes that are not typically part of a child's diet. Others may avoid smells or tastes that can limit an overall adequate food repertoire with a balanced diet. Some individuals may avoid environments that offer a distinct or unusual smell, ultimately impacting social participation if an individual avoids a cafeteria, restaurant, or family kitchen.

The tactile system refers to the sense of touch. Tactile perception allows us to have knowledge of tools and materials through touch alone. The sense of touch allows us to know where we were touched, an important component of maintaining social boundaries. Individuals who seek tactile stimuli may crave materials with a heightened or unusual texture or tactile surface. Some individuals may display inefficient tactile awareness and may ultimately exhibit difficulty with motor performance and tool usage. They may require visual support to assist with their performance. Individuals who are overreactive to tactile input may display exaggerated or heightened responses to tactile input and may avoid materials with an unusual tactile surface. These individuals may be extremely selective about what clothes to wear and having their hair combed or washed. Additionally, some individuals will avoid social situations in which unexpected touch opportunities may occur.

The vestibular system refers to the system of balance, reacting to head movement in relation to gravity. With head movement, eye, head, and postural reactions are integrated. Individuals who seek out vestibular input may crave more intensive movement opportunities, including spinning, rocking, and jumping. Some individuals may use repetitive rocking as a means of calming or self-regulation. Individuals who are hyper-responsive to vestibular input may dislike movement in which their heads are inverted, avoid swings, and get carsick easily. Some individuals

display gravitational insecurity in which they are uncomfortable if their feet leave the ground or their balance is displaced.

The proprioceptive system is triggered by movement or stretch and provides information on body-in-space awareness. Individuals with deficits in proprioceptive functioning may "crash" onto furniture or the floor, trail their hand along the walls and furniture as they are walking, or seek deep hugs. They might not notice if they are sitting too close to someone and may also exhibit grading motor force and pressure while performing motor actions. Their movements can be clumsy as they bump into furniture and objects in their environment. Individuals with deficits in proprioceptive functioning may use their vision as a means of providing increased feedback for body-in-space awareness and overall motor performance.

Overall motor planning and performance is dependent on adequate processing of sensory information and deficits in any of these areas can impact motor execution. Some children may display difficulty with motor imitation, tool usage, and overall initiation.

Historical Background

A. Jean Ayres, the clinician who first postulated sensory integration theory, described sensory and motor challenges in children with learning disabilities (Ayres 1963, 1965). Her early work expanded to identify different patterns of sensory integration dysfunction to conceptualize ways to understand sensory responses. Ultimately, sensory integration theory purports that motor performance, learning, and adaptive behavior are impacted by adequate processing and overall organization of sensory input (Baranek et al. 2008). Sensory integration therapy involves child-guided play addressing responsiveness to sensory input (Cascio 2010). Particular emphasis on tactile, vestibular, and proprioceptive input is incorporated into the interventions. Sensory integration therapy has been highly critiqued in the literature as an effective method of intervention (Hoehn and Baumeister 1994).

Current Knowledge

Further studies on sensory processing and how to conceptualize its influence on behavior continue to advance our understanding of this topic. Brown and Dunn (2010) outline four potential patterns of sensory processing based on Dunn's Model of Sensory Processing (Dunn 1997) that examines the relationship between neurological thresholds and behavioral responses. Dunn's model addresses a continuum and a level of interaction between the neurological thresholds and the behavioral responses as a way of understanding an individual's response to sensory information around them. The neurological thresholds are described as low or high, and the following four patterns of behavioral responses are identified: registration, seeking, sensitivity, and avoiding. *Registration* refers to the extent to which an individual misses sensory information. *Seeking* refers to the extent that an individual acquires sensory information. *Sensitivity* refers to the degree that an individual is aware of sensory information, and *avoiding* refers to the degrees by which an individual is bothered by sensory information.

Some individuals with a high neurological threshold may crave more intensive sensory experiences and may look to such heightened sensory input as frequent movement experiences to satisfy their need to acquire sensory input. These individuals may be perceived as risk taker or impulsive with poor personal boundaries. Some individuals with hyporesponsiveness may display sluggish or delayed reactions to sensory input. These individuals may exhibit difficulty recognizing or attending to sensory input in their environment and ultimately exhibit a more passive or even delayed response. Some individuals may use visual feedback to provide themselves with increased body-in-space awareness or may even engage in self-stimulatory behaviors to provide themselves with increased input.

Children and adults with a low neurological threshold may exhibit hyperresponsive reactions to sensory information and may display exaggerated and prolonged responses to sensory stimuli. Individuals may learn to avoid situations that provide sensory input that may be perceived as

unpleasant or display unusual responses to these types of sensory input. Engaging in repetitive actions may be evident and used as a means of calming an overaroused nervous system. Fluctuations in alertness and arousal may also be noted, impacting focus and attention and even general social interactions. Ayres (1972) described fight-flight-or-fright responses to sensory input as well, resulting in exaggerated responses to sensory stimuli, freezing in place to a specific sensory trigger with delayed responses, or even aggressive behaviors in the presence of sensory input.

Sensory processing is addressed by many different disciplines as difficulties in this area can manifest itself in different domains, including social participation, learning, play, and activities of daily living. Occupational therapists, physical therapists, speech and language pathologists, social workers, psychologists, and physicians may all work together to facilitate the child's performance in a variety of environments for successful participation. Environmental modifications may be necessary to promote participation in different settings, including headphones for a loud cafeteria or a study carrel in a visually stimulating classroom.

While environmental modifications and activity adaptations offer a way to facilitate participation for those individuals who demonstrate deficits in sensory processing, some clinicians address changes within the individual child. Sensory integration is based on the premise that activities that offer needed sensory stimulation will promote the brain's ability to process sensory information more effectively (Ayres 1972, 1979). Lane and Schaaf (2010) examined neuroplasticity and found that sensory input, motor performance, and motivation all affect neuroplasticity and subsequently support occupational therapy/sensory integration interventions.

Future Directions

Continued focus on evidence-based practice has prompted practitioners seek to demonstrate the overall effectiveness of interventions for sensory processing difficulties, particularly in regard to

sensory integration therapy. Psychophysical, neuroanatomical, and neurophysical measures have yielded much information on somatosensory processing (Cascio 2010) and may provide additional methods of effective intervention strategies, particularly in relation to tactile deficits. Miller et al. (2007) noted a reduction in the amplitude of electrodermal responses to sensory stimuli for children with sensory modulation disorders when a sensory integrative approach was incorporated into occupational therapy intervention. There is general agreement that the interventions aimed at sensory processing require further examination and research (Baranek 2002) and may ultimately show positive effects (Benson and Koomar 2010). Efforts to investigate empirically those specific sensory intervention procedures used with those with autism (e.g., weighted vests) and those used as part of multielement component interventions (e.g., brushing program) will continue to shed light on both the process of intervention and the potential outcomes that are predicted by those treatments. Additional work investigating the functional relationship between particular sensory deficits and the prediction and attainment of specific outcomes will also support the evidence-based use of these procedures.

See Also

- Ayres, A. Jean
- DeGangi-Berk Test of Sensory Integration
- Evaluation of Sensory Processing
- Hyperresponsiveness
- Low Registration
- Occupational Therapy (OT)
- Praxis
- Tactile Defensiveness
- Touch Sensitivity

References and Reading

- Ayres, A. J. (1963). The development of perceptual-motor abilities: A theoretical basis for treatment of dysfunction. (Eleanor Clarke Slagle Lecture). *American Journal of Occupational Therapy*, 17, 221–225.
- Ayres, A. J. (1965). Patterns of perceptual-motor dysfunction in children: A factor analytic study. *Perceptual and Motor Skills*, 20, 335–368.
- Ayres, A. J. (1972). *Sensory integration and learning disabilities*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (1973). *Sensory integration and learning disorders*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (1979). *Sensory integration and the child*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (1985). *Developmental dyspraxia and adult onset apraxia*. Torrance: Sensory Integration International.
- Baranek, G. T. (2002). Efficacy of sensory and motor interventions for children with autism. *Journal of Autism and Developmental Disorders*, 32, 397–422.
- Baranek, G. T., Foster, L. G., & Berkson, G. (1997). Tactile defensiveness and stereotypic behaviors. *American Journal of Occupational Therapy*, 51, 91–95.
- Baranek, G. T., Wakeford, L., & David, F. J. (2008). Understanding, assessing and treating sensory-motor issues. In K. Chawarska, A. Klin, & F. R. Volkmar (Eds.), *Autism spectrum disorders in infants and toddlers* (pp. 104–140). New York: Guilford Press.
- Benson, T. A., & Koomar, J. A. (2010). Systematic review of the research evidence examining the effectiveness of interventions using a sensory integrative approach for children. *American Journal of Occupational Therapy*, 64, 403–414.
- Brown, N. B., & Dunn, W. (2010). Relationship between context and sensory processing in children with autism. *American Journal of Occupational Therapy*, 64, 474–483.
- Cascio, C. (2010). Somatosensory processing in neurodevelopmental disorders. *Journal of Neurodevelopmental Disorders*, 2, 62–69.
- Dunn, W. (1997). The impact of sensory processing abilities on the daily lives of young children and their families: A conceptual model. *Infants and Young Children*, 9, 23–25.
- Hoehn, T. P., & Baumeister, A. A. (1994). A critique of the application of sensory integration therapy to children with learning disabilities. *Journal of Learning Disabilities*, 27, 338–350.
- Lane, S. J., Miller, L. J., & Hanft, B. (2000). Towards a consensus in terminology in sensory integration theory and practice: Part two. Sensory integration: Patterns of function and dysfunction. *Sensory Integration Special Interest Section Quarterly*, 23(2), 1–3.
- Lane, S. J., & Schaaf, R. C. (2010). Examining the neuroscience evidence for support for sensory-driven neuroplasticity: Implications for sensory-based occupational therapy for children and adolescents. *American Journal of Occupational Therapy*, 64, 375–402.
- May-Benson, T. (2011). Understanding the occupational needs of adults with sensory processing disorder. *OT Practice*, 16, 13–18.
- Miller, L. J., Coll, J. R., & Schoen, S. A. (2007). A randomized controlled pilot study of the

effectiveness of occupational therapy for children with sensory modulation disorder. *American Journal of Occupational Therapy*, 61, 228–238.

Parham, L. D., & Mailloux, Z. (2001). Sensory integration. In J. Case-Smith (Ed.), *Occupational therapy for children* (4th ed.). St. Louis: Mosby.

Tomchek, S. D. (2010). Sensory processing in individuals with an autism spectrum disorder. In H. M. Kuhanek & R. Watling (Eds.), *Autism: A comprehensive occupational therapy approach* (pp. 135–161). Bethesda: AOTA Press.

Sensory Processing Assessment

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Synonyms

[SPA](#)

Description

The *Sensory Processing Assessment* (Baranek 2010), currently in revision/development, is an observational assessment which provides objective data regarding a “child’s approach, avoidance, and/or seeking behaviors with novel sensory toys, orienting and habituation responses to social and non-social sensory stimuli, and generation of novel action strategies with toys” (p. 2). This 15-min, play-based tool is currently being utilized in several research projects but is not yet published in final format. The design, for children between 9 months and 6 years of age, was intended for use with children on the autism spectrum and other developmental delays to assist with identifying patterns of sensory processing. It was thought that this tool could be utilized as a counterpart of the Sensory Experiences Questionnaire (SEQ; refer to the “[See Also](#)” section below) which is a parent report assessment tool.

There are four parts to this assessment tool with identified descriptors as to what the examiner should observe in order for the child to obtain a specific score:

- Novel Sensory Toys – rates hyper-responsiveness and sensory seeking
- Sensory Habituation – observes responses to repeated stimuli
- Sensory Orienting Trials – rates hyporesponsiveness
- Stereotyped Behaviors Checklist – items marked as observed or not

Historical Background

The author, Grace Baranek, Professor and Associate Chair of Research at the University of North Carolina – Chapel Hill’s Division of Occupational Science and Occupational Therapy, has a long-standing and very well-respected history of research in the area of autism spectrum disorders. The Sensory Experiences Project (SEP), funded by the National Institute for Child Health and Human Development, focuses on the research on the sensory features/processes of children on the autism spectrum, developmental delay, and/or typical development for children 2 through 12 years old. This research is conducted under the University’s PEARLS project (Program for Early Autism Research, Leadership, and Service) whose intent is improve early identification markers and contribute to the evidence with regard to best practices in intervention.

Psychometric Data

Preliminary studies of reliability and validity have been completed. As this assessment tool is not yet published, full detail of the psychometric data is not yet available.

Clinical Uses

The intended use of this assessment tool is to augment traditional diagnostic and developmental

assessment in order to identify patterns of sensory processing which may have implications for diagnosis or intervention. As this tool is not yet published, a full description of its clinical use is not yet available.

See Also

- [Ayres, A. Jean](#)
- [Evaluation of Sensory Processing](#)
- [Occupational Therapy \(OT\)](#)
- [Sensory Diet](#)
- [Sensory Integration and Praxis Test](#)
- [Sensory Integration \(SI\) Therapy](#)
- [Sensory Processing](#)
- [Sensory Processing Assessment](#)
- [Sensory Processing Measure](#)
- [Sensory Processing Measure: Preschool \(SPM-P\)](#)
- [Test of Sensory Functioning in Infants](#)

BUS	School bus
CAF	Cafeteria
DC:0-3R	Diagnostic classification of mental health and developmental disorders of infancy and early childhood, revised
DMIC	Diagnostic manual for infancy and early childhood
HEA	Hearing
MUS	Music class
PHY	Physical education class
PLA	Planning and ideas
REC	Recess/Playground
SES	Socioeconomic status
SI	Sensory integration
SOC	Social participation
SPD	Sensory processing disorder
TOT	Total sensory systems
TOU	Touch
U.S.	United States
VIS	Vision

Synonyms

[Preschool edition](#); [SPM](#); [SPM-P](#)

References and Reading

- A full list of Dr. Baranek's research publications produced as a result of the SEP can be found at: <http://www.med.unc.edu/ahs/ocsci/sep/research-articles>.
- Baranek, G. (2010). Sensory processing assessment for young children (SPA)©1999: Version 2.2: Research Version (3/15/2010).

Sensory Processing Measure

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Abbreviations

ART	Art class
ASD	Autism spectrum disorder
BAL	Balance and motion
BOD	Body awareness

Description

The *Sensory Processing Measure* (SPM) is a standardized assessment tool published by Western Psychological Services (www.wpspublish.com) that provides information about the sensory and environmental issues that may impact a child's performance in school, home, and community settings (Parham et al. 2007). The design of the SPM allows a practitioner to consider whether a child's performance is being hindered by sensory processing or sensory integration difficulties. The SPM's three integrated rating scales (Home Form, Main Classroom Form, and School Environments Form) are designed to elicit information about the sensory processing, praxis, and social participation of primary school children ages 5–12 years (Miller Kuhaneck et al. 2007a, b; Parham and Ecker 2007). With the SPM in part being grounded in Ayres Sensory Integration (SI) theory (Ayres 1972, 1979, 2005), it provides norm-referenced standard scores for two higher

level integrative functions (praxis and social participation) and five sensory systems; those being visual, auditory, tactile, proprioceptive, and vestibular functioning (see Table 1) (Parham et al. 2007). Within each sensory system, the SPM offers clinical information on a number of processing SI vulnerabilities including under-responsiveness, over-responsiveness, sensory-seeking behavior, and perceptual problems (Parham et al. 2007).

The SPM Home Form (made up of 75 items) (Parham and Ecker 2007) is completed by a child's parent or caregiver while the SPM Main

Classroom Form (consisting of 62 items) (Miller Kuhaneck et al. 2007a) is filled out by a child's main classroom teacher (see Table 1). The SPM School Environments Form (containing 10–15 items per school environment) (Miller Kuhaneck et al. 2007b) is completed by other school staff who work with and know a child in those relevant school contexts (e.g., school bus driver, physical education teacher, librarian). The SPM Home and Main Classroom Forms each take approximately 15–20 min to be completed by respondents and about 5–10 min to be scored by practitioners (Parham et al. 2007). The SPM forms are written at an 8th grade reading level, but in the event that parents have poor reading skills, it is possible to complete the Home Form by interviewing the respondent(s).

All forms of the SPM have items that are answered by respondents using a 4-point Likert-type scale (Never = 4, Occasionally = 3, Frequently = 2, Always = 1) (Parham et al. 2007). The SPM AutoScore™ feature (forms containing a sheet of carbon paper) permits the transfer of respondents' answers to a scoring worksheet that allows for easy calculation of the subscale scores. The clinician adds up and transfers the raw subscale scores to the SPM Profile Sheet. Prior to giving the SPM AutoScore™ proforma to the parent or teacher, the Profile Sheet must be detached so that it can be used later in scoring the scale.

The eight sensory processing scores (Social Participation [SOC], Vision [VIS], Hearing [HEA], Touch [TOU], Body Awareness [BOD] (proprioception), Balance and Motion [BAL] (vestibular function), Planning and Ideas [PLA] (praxis), and Total Sensory Systems [TOT]) generated by both the SPM Home and Main Classroom Forms are converted from raw scores into T-scores as well as their corresponding percentile ranks (Miller Kuhaneck et al. 2007a; Parham and Ecker 2007). The T-scores for these two forms are then identified for interpretive purposes as falling into one of three categories: the *Typical* range (T-score of 40–59 indicating a child's behavioral and sensory functioning are similar to that of typically developing peers), the *Some Problems* range (T-score of 60–69 indicating that a child

Sensory Processing Measure, Table 1 Sensory Processing Measure (SPM) forms and scales

SPM subscales	Number of items
<i>Home Form</i>	
Social participation (SOC)	10
Vision (VIS)	11
Hearing (HEA)	8
Touch (TOU)	11
Body awareness (BOD)	10
Balance and motion (BAL)	11
Planning and ideas (PLA)	9
Total sensory systems (TOT)	56
<i>Main Classroom Form</i>	
Social participation (SOC)	10
Vision (VIC)	7
Hearing (HEA)	7
Touch (TOU)	8
Body awareness (BOD)	7
Balance and motion (BAL)	9
Planning and ideas (PLA)	10
Total sensory systems (TOT)	42
<i>School Environments Form</i>	
Art class (ART)	15
Music class (MUS)	15
Physical education class (PHY)	15
Recess/playground (REC)	15
Cafeteria (CAF)	15
School bus (BUS)	10

Note: The SPM Home and Main Classroom scales yield norm-referenced standard scores. The SPM School Environments scales yield Total Scores that are interpreted by means of a cutoff criterion. The TOT scales include VIS, HEA, TOU, BOD, and BAL items, plus several items representing taste and smell processing
Source: Parham et al. (2007, p. 4). (Reproduced with permission from publisher)

exhibits mild-to-moderate difficulties in his or her behavioral or sensory functioning), or the *Definite Dysfunction* range (T-score of 70–80 indicating that a child is presenting with significant sensory processing problems that are likely having an impact on his/her daily functioning) (Miller Kuhaneck et al. 2007a; Parham and Ecker 2007).

“The VIS, HEA, TOU, BOD, and BAL scales are referred to as the *sensory systems scales* because they address a child’s ability to process direct sensory inputs. The SOC and PLA scales, on the other hand, represent higher level integration functions that are strongly influenced by sensory inputs while encompassing other cognitive and contextual factors. The TOT scale is a composite of all the sensory system scales, plus additional items that reflect taste and smell inputs” (Parham et al. 2007, p. 19). One advantage of using the SPM is that for the first time, an Environment Difference score permits direct comparison of a child’s sensory functioning at home and at school. While the scales on the SPM Home and Main Classroom Forms are identical, the items themselves are specific to each specific environment. Individual item responses indicate how sensory difficulties present themselves in these two different settings.

With the SPM, the School Environments Form is provided on an unlimited-use CD. This allows the SPM user to look at the child’s functioning in six specific school environments outside of the main classroom: Art Class (ART), Music Class (MUS), Physical Education Class (PHY), Recess/Playground (REC), Cafeteria (CAF), and School Bus (BUS) (see Table 1) (Miller Kuhaneck et al. 2007b). Each school environment has its own Rating Sheet, which can be printed from the CD provided with the purchase of a test manual (Miller Kuhaneck et al. 2007b). Each rater can complete his or her 15-item Rating Sheet (10 items for the School Bus setting) in less than 5 min.

The SPM School Environments Forms yield no T-scores. Instead, these forms yield a total score for each relevant school environment that is interpreted against an established cutoff criterion. Scores at or above the cutoff point indicate that the child is experiencing a high number of sensory processing problems in that specific

school environment (Miller Kuhaneck et al. 2007b). Whether a practitioner uses one or all six Rating Sheets, the SPM School Environments Form must always be administered in conjunction with the SPM Main Classroom Form (Parham et al. 2007). In other words, the SPM School Environments Form should not be used in isolation. As reported in the SPM manual, the SPM School Environments Form items include behavioral indicators of sensory modulation dysfunction, difficulties with regulation of arousal, and praxis deficits that may be influencing educational performance, as well as the ability to participate fully in the educational environment and maintain relationships with peers (Miller Kuhaneck et al. 2007b).

Historical Background

The SPM came about with the cooperation of two groups of occupational therapy researchers in the United States who were each completing separate studies with the goal of generating a scale that would provide a link between home and school environments in the identification of SI problems. The SPM Home Form originated from the *Evaluation of Sensory Processing* by Parham and Ecker (2002), while the SPM Main Classroom and School Environments Form evolved from the *School Assessment of Sensory Integration* by Miller Kuhaneck et al. (2007a). “The goals underlying this combination of assessment instruments were to maximize the clinical utility of the measures and share a common standardization effort. By so doing, it was the test authors’ intent to create an integrated collection of parallel test materials regarding sensory processing and sensory integration that could be used across multiple settings” (Watson and Woodin 2007, p. 3). The test authors set out to reformat the scales so that they were more efficient and user friendly. A research version of each instrument was created to be used in the standardization of the SPM.

The theoretical basis of the SPM was derived from Ayres’s theory of SI (Ayres 1972). The SPM manual reports that disorders of SI are believed to

include the following: not using sensory information, sensory-seeking behaviors, being overwhelmed by sensory stimuli, difficulties with sensory discrimination, and sensorimotor problems. It is suggested that children who exhibit impediments with sensory processing may have difficulties in other areas of functioning such as planning and organizing their body movements.

Psychometric Data

Given that the SPM is a relatively new scale, limited psychometric data has been reported about it by external researchers and reviewers (Brown et al. 2010; Henry 2007; Henry et al. 2009; Kuhaneck and Henry 2009a, b). The information reported in this section focuses on the recent reliability and validity data reported in the SPM manual published in 2007 (Parham et al. 2007). The SPM Home Form and Main Classroom Form were standardized on a sample of 1,051 typically developing children ages 5–12 years in kindergarten through to grade 6 from approximately 76 sites across the United States (Henry 2007). All of the children in the sample were assessed with the SPM Home Form (completed by the child's parent or caretaker) and Main Classroom Form (completed by the child's primary school teacher). No children included in the standardization sample were attending full-time special education programs; however, "no attempt was made to exclude children with mild academic or behavioral difficulties" (Parham et al. 2007, p. 47).

Demographic data about the standardization sample were compared by age and against the 2001 American Census population estimates with regard to gender, race, geographical distribution, and educational attainment of parents (Henry 2007). In the SPM manual, it was noted that "whites are slightly overrepresented, whereas Blacks and Asians are slightly underrepresented. . . in terms of U. S. geographical distribution, the Midwest is overrepresented compared to Census figures, whereas the South and West are underrepresented" (Parham et al. 2007, p. 47). A subsample of 306 children from the SPM

standardization sample was used to develop scores for the School Environments Form (Miller Kuhaneck et al. 2007b).

Types of reliability data often reported include internal consistency, test-retest/time sampling reliability/temporal stability, inter-rater/inter-scorer reliability, intra-rater/intra-scorer reliability, alternate form reliability, and split-half reliability (American Educational Research Association [AERA], American Psychological Association [APA] and National Council on Measurement in Education [NCME] 1999). For the SPM Home Form subscale scores, internal consistency Cronbach alpha coefficients ranged from 0.77 to 0.95 (median = 0.85), while for the SPM Main Classroom Form subscale scores, internal consistency Cronbach alphas ranged from 0.75 to 0.95 (median = 0.86) (Parham et al. 2007). The SPM VIS, HEA, and TOU subscales had the lowest internal consistency estimates ranging from 0.75 to 0.84 for the total sample. "Although acceptable, this is concerning as vision, hearing and touch would be areas where the identification of sensory issues would be most observable. Social Participation, Planning and Ideas, and the Total Sensory Systems Score all demonstrate excellent reliability" (Cruce 2007, p. 2). The SPM School Environments scores yielded internal consistency values that ranged from 0.82 to 0.91 (median = 0.89) (Parham et al. 2007). "Internal consistency for the School Environments Form also was strong" (Cruce 2007, p. 2).

Test-retest reliability data were collected on 77 children ages 5–12 in a 2-week follow-up using both the SPM Home and Main Classroom Forms. As would be expected, temporal stability was high with all composites having correlation coefficients above 0.94 (Cruce 2007). For the SPM Home Form subscale scores, test-retest reliability coefficients ranged from 0.94 to 0.98 (median = 0.97), while for the SPM Main Classroom Form subscale scores, test-retest reliability coefficients ranged from 0.95 to 0.98 (median = 0.97) (Parham et al. 2007). "Additionally, standard errors of measurement and confidence intervals calculated at 95% appear to be quite acceptable" (Cruce 2007, p. 2).

Types of validity often reported for tests include face validity, content validity, criterion-related validity, and construct validity (AERA, APA, & NCME 1999; Anastasi and Urbina 1997). Two subtypes of criterion-related validity frequently included are concurrent validity and predictive validity, while subtypes of construct validity often reported include factor analysis validity, discriminant validity, convergent validity, divergent validity, diagnostic validity, and rating scale validity (Fawcett 2007; Wolfe and Smith 2007a). Many scales are now using a combination of Classical Test Theory methods (e.g., factor analysis) and Item Response Theory approaches (e.g., Rasch analysis) to examine the construct validity of a measure (Wolfe and Smith 2007b).

Limited evidence of validity was reported in the SPM manual. Content validity for the SPM forms was initially examined using the *Evaluation of Sensory Processing* (Johnson-Ecker and Parham 2000; Parham and Ecker 2002) and the *School Assessment of Sensory Integration* (Miller Kuhaneck et al. 2007a, b, c) in their original form. The items from the two original scales were examined and those items that were redundant or not aligned with Ayres's SI theory (Ayres 1972, 1979) were discarded. The item sets were then subjected to several rounds of expert review and were retained only if they were judged to adequately represent the intended sensory function construct, social participation, or praxis (Parham et al. 2007). "These earlier developmental phases generate confidence in the content validity of the current SPM items and scales" (Parham et al. 2007, p. 57).

A research version of the SPM was subjected to confirmatory and exploratory factor analyses to examine its construct validity. "Results of the factor analyses provided support for the basic scale structure of the SPM" (Watson and Woodin 2007, p. 3). Item-scale correlations are reported for the Home Form and the Main Classroom Form. According to Watson and Woodin (2007), the item-scale correlations provide "robust support for the Home Form as a measure that can be scored and interpreted according to its separate scales. In contrast, questionable results were

attained for the Main Classroom Form in terms of high interscale correlations between the BOD (proprioception) and BAL (vestibular) scales. This finding should be considered when interpreting results from these scales in individual cases" (p. 3). The standard errors of measurement varied between 1.29 and 4.40 points (Parham et al. 2007).

Convergent validity (a subtype of construct validity) was examined by comparing the SPM to the Sensory Profile (Dunn 1999), and the results indicated adequate overlap in areas where the tests displayed similar content. "Findings indicated that the SPM Home Form yielded strong and consistent relationships with the scores on the Sensory Profile" (Watson and Woodin 2007, p. 3). No comparable measure was available to investigate the convergent validity of the SPM Main Classroom Form. Therefore, the relationship between the SPM Main Classroom Form and the independently completed SPM School Environments Forms was examined. "Findings of strong relationships between these forms provide support for the stability of measurement of these forms" (Watson and Woodin 2007, p. 3). Brown et al. (2010) examined the convergent validity between the Home and Main Classroom Forms SPM and the Sensory Profile School Companion. The Sensory Profile and the SPM Home Form were significantly correlated ($\rho = 0.86$, $p < 0.01$). The Sensory Profile School Companion and SPM Main Classroom Form were also significantly correlated ($\rho = 0.74$, $p < 0.01$). The authors concluded that the two sensory processing scales exhibited moderate levels of convergent validity.

Discriminant validity (a subtype of construct validity) was evidenced by the SPM's ability to differentiate between two groups of children with known differences, those with typical development from those with formally diagnosed clinical disorders. It was reported in the SPM manual that a sample of 345 children (separate from the standardization sample) receiving occupational therapy intervention was used to verify that the SPM subscales were able to differentiate between typical children from those with clinical disorders (Parham et al. 2007).

The SPM authors used Rasch analysis (Bond and Fox 2001) to investigate its rating scale validity structure. With Rasch analysis, two sets of scores are generated, one for children (person ability scores) and the other for the SPM items (item difficulty scores), expressed by means of a common numerical metric called a logit score (Bond and Fox 2001). The use of a common logit allows instrument developers to examine the functioning of items with respect to different ability levels of participants. In other words, “the Rasch method illuminates how the categories of the SPM item rating scale operate together to measure vary amounts of sensory processing dysfunction” (Parham et al. 2007, p. 69). The rating scale validity results indicated that each of the SPM’s four rating scale categories (Never, Occasionally, Frequently, Always) “is the most probable response for a distinct level of the underlying sensory processing dysfunction. . .none of the categories are redundant. . .the category sequence of increasing behavioral frequency is properly aligned with the continuum of increasing sensory pathology” (Parham et al. 2007, p. 69).

Parham et al. (2007) investigated the moderating effects of demographic variables (including age, gender, ethnicity, and socioeconomic status [SES]) on the SPM scale scores. “An effect size metric was used to determine if the demographic moderator variables were associated with any clinically meaningful differences between groups in the SPM raw scale scores” (Parham et al. 2007, p. 47). The analysis of the demographic moderating effects did not yield any significant results. “Actual differences in SPM scores due to age, gender, ethnicity, and SES were small and not likely to have any meaningful impact on interpretation of [SPM] test results” (Parham et al. 2007, p. 49).

Lai et al. (2011) explored the psychometric properties of a Chinese version of the SPM referred to as the Sensory Processing Measure-HK Chinese (SPM-KHC). The original SPM Home Form and Main Classroom Forms were translated into Chinese using the forward and backward translation procedure. The equivalence of the translation was reviewed by a panel of eight experts and content validity (relevance and

representativeness) was assessed by a panel of 20 experts. Data was collected from two groups of participants: a group of typically developing children and a group of children presenting with ASD. The sample sizes were as follows: 542 healthy children for the SPM Home Form, 325 healthy children for the SPM Main Classroom Form, 100 children with ASD for the SPM Home Form, and 95 children with ASD for the Main Classroom Form (Lai et al. 2011). The internal consistency coefficients for the SPM-KHC Home Form scales ranged from 0.674 to 0.861 and from 0.779 to 0.933 for the SPM-KHC Main Classroom Form. Two week test-retest ICCs for the SPM-KHC Home Form ranged from 0.70 to 0.95 and from 0.82 to 0.97 for the SPM-KHC Main Classroom Form (Lai et al. 2011).

Convergent validity between the SPM-KHC Home Form and the Chinese Sensory Profile in the form of correlations between the scales of the instruments ranged from -0.357 to -0.673 (Lai et al. 2011). The SPM-KHC Home Form and the SPM-KHC Main Classroom Form subscales were able to discriminate between the two participant groups thus providing evidence of its discriminant validity. The construct validity of the SPM-KHC is examined by exploratory factor analysis. For the SPM-KHC Home Form, “principal component analysis extracted 22 factors with eigenvalue greater than 1” that accounted for 61.4% of the total variance (Lai et al. 2011, p. 2640). For the SPM-KHC Main Classroom Form, “principal component analysis extracted 12 factors with eigenvalue greater than 1” that accounted for 67.3% of the total variance (Lai et al. 2011, p. 2640). For both the SPM-KHC Home and Main Classroom Forms, the first factor included all the items of Vision, Hearing, Touch, Taste and Smell, Body Awareness, Balance and Motion, and Planning and Ideas scales.

Clinical Uses

As outlined in the SPM manual, it has three primary uses: (1) providing clinicians with information regarding a child’s sensory integrative functioning in order to create strategies that

enhance performance in school and at home; (2) evaluating a child's performance in a variety of different environments, allowing comparison and discussion regarding the differences; and (3) examining the behaviors indicative of sensory modulation dysfunction and dyspraxia, as well as the related areas of arousal, attention, and social participation (Parham et al. 2007). Watson and Woodin (2007) state that "the SPM will be useful in determining the types of interventions that would be most appropriate for a child as a means of measuring program effectiveness, building team collaboration regarding intervention strategies, fostering program planning, and developing Individualized Educational Plans (IEPs)" (p. 3). In summary, the SPM scales can be used in a range of educational, clinical practice, and research settings.

Dr. Ayres (1972, 1979) first coined the term *sensory integration dysfunction* as part of her theory that deficits in processing sensation from the body and the environment lead to sensorimotor and learning problems in children. More recently, the term *sensory processing disorder* (SPD) has been proposed. It is estimated that SPD affects the lives of 1 in 20 children and adults (Ahn et al. 2004). SPD is recognized by both the *Diagnostic Manual for Infancy and Early Childhood* (DMIC) of the Interdisciplinary Council on Developmental and Learning Disorders (2005) and the *Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood, Revised* (DC:0-3R) (Zero to Three 2005). Three primary diagnostic groups have been categorized within the SPD category to describe children who have difficulty regulating and organizing responses to sensory input: *sensory modulation disorder*, *sensory discrimination disorder*, and *sensory-based motor disorder* (Ayres 2005; Miller et al. 2007; Miller Kuhaneck et al. 2010).

Sensory modulation disorder has three subtypes: *sensory over-responsivity*, *sensory under-responsivity*, and *sensory seeking* (Roley et al. 2001; Dunn 2010). Children with *sensory over-responsivity* register sensations too intensely and for a longer duration than typically developing children and may react to sensory input by pulling

away, screaming, or avoiding input (Robinson and Magill-Evans 2009). Children with *sensory under-responsivity* tend not to respond to input and may be withdrawn or seem to be in their own world (Dunn 2010). *Sensory seeking* describes children who tend to crave intense or an unusual amount of sensory input. They may be constantly on the move, bang their heads against windows or walls, or fall repeatedly in an effort to gain appropriate sensory information (Miller Kuhaneck et al. 2010). The other two diagnostic groups include sensory discrimination disorder or difficulty deciphering or interpreting qualities of sensory information and sensory-based motor disorder, in which children tend to appear uncoordinated because of faulty processing of sensory input (Dunn 2010). A diagnosis of SPD is not made unless the condition significantly affects a child's daily life (Baranek et al. 2006).

The SPM forms can be used by a variety of health and education professionals (including occupational therapists, psychologists, teachers, social workers, counsellors, physiotherapists, speech-language pathologists, nurses, pediatricians, and physicians, among others) to evaluate the sensory processing issues of school-age children (Henry et al. 2009). For example, the SPM's unique multi-environment approach let practitioners investigate why a child who functions well in a highly structured classroom with a predictable daily routine may have problems at home or at a playground in the local community park where the environment is variable and the routine is not predictable. Cruce (2007) states that the SPM is "a good choice if one needs to identify sensory difficulties across environments, and the School Environments Form especially fills a gap seen in most rating scales as it looks at alternate settings where students are likely to struggle. This measure is best used comprehensively, and one should be guarded in interpreting the SPM Main Classroom Form in isolation without the use of the SPM Home Form for comparison" (p. 3).

The SPM can be used to determine whether a child is presenting with signs indicative of SPD. One study by the Sensory Processing Disorder Scientific Work Group (Ben-Sasson et al. 2009)

suggests that one in every six children experiences sensory symptoms that may be significant enough to have a negative impact on some aspect of everyday life functions. A significant number of children diagnosed with autism spectrum disorder (ASD) (and related disorders) present with signs of SPD (e.g., child who does not like to eat foods with certain textures or smells, does not like to wear clothes made of certain types of material, does not like to wear socks or shoes, does not like to be hugged, does not like to handle certain types of materials such as Playdoh™, and/or does not like having his or her hair brushed or washed) (Baranek et al. 2005; Kientz and Dunn 1997; Leekam et al. 2007; Tomchek and Dunn 2007; Watling 2000; Watling et al. 2001), and the SPD would be a measure suitable to assess this group of children. “Children with autism are expected to show elevated scores on the SPM SOC and PLA scales. . .these deficits in participation and praxis may be caused or exacerbated by processing deficits in specific sensory systems” (Parham et al. 2007, p. 33). Parham et al. also noted that “with moderate to severe autism, it is not uncommon to see all SPM scales elevated in the Definite Dysfunction range” (p. 33).

The SPM can be used to assess children presenting with suspected sensory processing challenges as well as children with confirmed diagnoses including genetic disorders, developmental disabilities, learning disabilities, neurological problems, behavioral issues, or psychosocial problems (Baker et al. 2008; Carrasco Koester et al. 2014; Hansen and Jirikowic 2013; Pfeiffer et al. 2015; Robinson and Magill-Evans 2009; Rosenberg et al. 2015; Spira 2014). It states in the SPM manual that it was not designed for use with students with severe sensory impairments such as blindness or deafness or children with severe motor impairments (Parham et al. 2007). It can be used in conjunction with other assessment tools to provide a comprehensive overview of a child’s daily functioning or can be used as a stand-alone tool. Similarly, the SPM can be used to document children’s progress or clinical change after being involved in an intervention program (e.g., pre-intervention and posttest intervention). Recently a number of studies have been published

where the SPM has been used to assess the sensory processing issues of children presenting with ASD (Chang et al. 2012; Fernández-Andrés et al. 2015; Gee et al. 2014; Gibbs and Toth-Cohen 2011; Sanz-Cervera et al. 2015; Smith Roley et al. 2015).

Cruce (2007) noted that “the SPM appears to be a usable, structurally sound instrument that attempts to quantify sensory problems in children. The SPM would be a good instrument for occupational therapists to use in planning appropriate empirically validated treatment options” (p. 3). In conclusion, the SPM appears to be a useful, informative, and psychometrically sound scale that clinicians can use with confidence to assess school-age children including those diagnosed with ASD. Watson and Woodin (2007) stated that the “SPM has an adequate normative base, provides data across multiple settings in home and school environments, is easy to administer and score, and provides adequate to moderate evidence of reliability, validity, factor structure, and discriminant validity” (p. 3).

See Also

- [Occupational Therapy \(OT\)](#)
- [Sensation Avoiding](#)
- [Sensation-Seeking](#)
- [Sensory Diet](#)
- [Sensory Experiences Questionnaire](#)
- [Sensory Impairment in Autism](#)
- [Sensory Integration \(SI\) Therapy](#)
- [Sensory Processing](#)
- [Sensory Processing Assessment](#)
- [Sensory Processing Measure: Preschool \(SPM-P\)](#)
- [Sensory Sensitivity Questionnaire: Revised](#)
- [Sensory Stimuli](#)
- [Standardized Tests](#)

References and Reading

- Ahn, R. R., Miller, L. J., Milberger, S., & McIntosh, D. N. (2004). Prevalence of parents’ perceptions of sensory processing disorders among kindergarten children. *American Journal of Occupational Therapy*, 58(3), 287–293.

- American Educational Research Association, American Psychological Association, & National Council on Measurement in Education. (1999). *Standards for educational and psychological testing*. Washington, DC: American Psychological Association.
- Anastasi, A., & Urbina, S. (1997). *Psychological testing*. Upper Saddle River: Prentice Hall International.
- Ayres, A. J. (1972). *Sensory integration and learning disorders*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (1979). *Sensory integration and the child*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (2005). *Sensory integration and the child, 25th anniversary edition*. Los Angeles: Western Psychological Services.
- Baker, A. E. Z., Lane, A., Angley, M. T., & Young, R. L. (2008). The relationship between sensory processing patterns and behavioral responsiveness in autistic disorder: A pilot study. *Journal of Autism and Developmental Disorders*, 38, 867–875.
- Baranek, G. T., Parham, L. D., & Bodfish, J. W. (2005). Sensory and motor features in autism: Assessment and intervention. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Assessment, interventions and policy, Vol. 2, pp. 831–857). Hoboken: Wiley.
- Baranek, G. T., David, F. J., Poe, M. D., Stone, W. L., & Watson, L. R. (2006). Sensory experiences questionnaire: Discriminating sensory features in young children with autism, developmental delays, and typical development. *Journal of Child Psychology and Psychiatry*, 47(6), 591–601.
- Ben-Sasson, A., Carter, A. S., & Briggs-Gowan, M. J. (2009). Sensory over-responsivity in elementary school: Prevalence and social-emotional correlates. *Journal of Abnormal Child Psychology*, 37, 705–716.
- Bond, T. G., & Fox, C. M. (2001). *Applying the Rasch model: Fundamental measurement in the human sciences*. Mahwah: Lawrence Erlbaum Associates.
- Brown, T., Morrison, I. C., & Stagnitti, K. (2010). The convergent validity of two sensory processing scales used with school-age children: Comparing the Sensory Profile and the Sensory Processing Measure. *New Zealand Journal of Occupational Therapy*, 57(2), 56–65.
- Carrasco Koester, A., Mailloux, Z., Geppert Coleman, G., Baltazar Mori, A., Paul, S. M., Blanche, E., & ... Cermak, S. A. (2014). Sensory integration functions of children with cochlear implants. *American Journal of Occupational Therapy*, 68(5), 562–569.
- Chang, M. C., Parham, L. D., Imperatore Blanche, E., Schell, A., Chih-Ping, C., Dawson, M., & Clark, F. (2012). Autonomic and behavioral responses of children with autism to auditory stimuli. *American Journal of Occupational Therapy*, 66(5), 567–576.
- Cruce, M. K. (2007). Test review of the Sensory Processing Measure. In R. A. Spies, J. F. Carlson, & K. F. Geisinger (Eds.), *The eighteenth mental measurements yearbook* (pp. 1–3). Lincoln: Buros Institute of Mental Measurements. Retrieved 15 Nov 2010, from the Buros Institute's Test Reviews Online Web site: <http://www.unl.edu/buros>.
- Dunn, W. (1999). *The Sensory Profile*. San Antonio: Psychological Corporation.
- Dunn, W. (2010). Sensory processing: A critical assessment area for autism spectrum disorders. In F. Volkmar (Ed.), *Autism and autism spectrum disorders: History, diagnosis, neurobiology, treatment and outcome*. London: The Biomedical & Life Sciences Collection, Henry Stewart Talks Ltd. Retrieved from <http://hstalks.com/bio>.
- Fawcett, A. L. (2007). *Principles of assessment and outcome measurement for occupational therapists and physiotherapists: Theory, skills and application*. Hoboken: Wiley.
- Fernández-Andrés, M. I., Pastor-Cerezuela, G., Sanz-Cervera, P., Tárraga-Mínguez, R., & Fernández-Andrés, M. I. (2015). A comparative study of sensory processing in children with and without autism spectrum disorder in the home and classroom environments. *Research in Developmental Disabilities*, 38, 202–212.
- Gee, B. M., Thompson, K., & St John, H. (2014). Efficacy of a sound-based intervention with a child with an autism spectrum disorder and auditory sensory over-responsivity. *Occupational Therapy International*, 21(1), 12–20.
- Gibbs, V., & Toth-Cohen, S. (2011). Family-centered occupational therapy and telerehabilitation for children with autism spectrum disorders. *Occupational Therapy in Health Care*, 25(4), 298–314.
- Hansen, K. D., & Jirikowic, T. (2013). A comparison of the Sensory Profile and Sensory Processing Measure Home Form for children with fetal alcohol spectrum disorders. *Physical & Occupational Therapy in Pediatrics*, 33(4), 440–452.
- Henry, D. A. (2007). Collecting data for the Sensory Processing Measure: Lessons learned. *OT Practice*, 12(3), 10–15.
- Henry, D. A., Ecker, C. E., Glennon, T. J., & Herzberg, D. (2009). Using the Sensory Processing Measure (SPM) in multiple practice areas. *OT Practice*, 14(10), 9–13.
- Interdisciplinary Council on Developmental and Learning Disorders. (2005). *Diagnostic manual for infancy and early childhood (ICDL-DMIC)*. Bethesda: Interdisciplinary Council on Developmental and Learning Disorders.
- Johnson-Ecker, C. L., & Parham, L. D. (2000). The Evaluation of Sensory Processing: A validity study using contrasting groups. *American Journal of Occupational Therapy*, 54(5), 494–503.
- Kientz, M. A., & Dunn, W. (1997). A comparison of the performance of children with and without autism on the sensory profile. *American Journal of Occupational Therapy*, 51, 530–537.
- Kuhaneck, H. M., & Henry, D. A. (2009a). The Sensory Processing Measure (SPM): Meeting the needs of school-based practitioners: Part one: Description and

- background. *Journal of Occupational Therapy, Schools & Early Intervention*, 2(1), 51–57.
- Kuhaneck, H. M., & Henry, D. A. (2009b). The Sensory Processing Measure (SPM): Meeting the needs of school-based practitioners: Part two: Case example and practical applications. *Journal of Occupational Therapy, Schools & Early Intervention*, 2(1), 58–63.
- Lai, C., Chung, J., Chan, C., & Li-Tsang, C. (2011). Sensory Processing Measure-HK Chinese version: Psychometric properties and pattern of response across environments. *Research in Developmental Disabilities*, 32(6), 2636–2643.
- Leekam, S. R., Nieto, C., Libby, S. J., Wing, L., & Gould, J. (2007). Describing the sensory abnormalities of children and adults with autism. *Journal of Autism and Developmental Disorders*, 37, 894–910.
- Miller Kuhaneck, H., Henry, D. A., & Glennon, T. J. (2007a). *Sensory Processing Measure (SPM) main classroom form*. Los Angeles: Western Psychological Services.
- Miller Kuhaneck, H., Henry, D. A., & Glennon, T. J. (2007b). *Sensory Processing Measure (SPM) school environments form*. Los Angeles: Western Psychological Services.
- Miller Kuhaneck, H., Henry, D. A., Glennon, T. J., & Mu, K. (2007c). Development of the Sensory Processing Measure – school: Initial studies of reliability and validity. *American Journal of Occupational Therapy*, 61(2), 170–175.
- Miller Kuhaneck, H., Ecker, C., Parham, L. D., Henry, D. A., & Glennon, T. J. (2010). *Sensory Processing Measure – Preschool (SPM – P): Manual*. Los Angeles: Western Psychological Services.
- Miller, L. J., Anzalone, M., Lane, S., Cermak, S. A., & Osten, E. (2007). Concept evolution in sensory integration: A proposed nosology for diagnosis. *American Journal of Occupational Therapy*, 61, 135–140.
- Parham, L. D., & Ecker, C. (2002). Evaluation of sensory processing: Research version 4. In A. C. Bundy, S. J. Lane, & E. A. Murray (Eds.), *Sensory integration: Theory and practice* (2nd ed., pp. 194–196). Philadelphia: F. A. Davis.
- Parham, L. D., & Ecker, C. (2007). *Sensory Processing Measure (SPM) home form*. Los Angeles: Western Psychological Services.
- Parham, L. D., Ecker, C., Miller Kuhaneck, H., Henry, D. A., & Glennon, T. J. (2007). *Sensory Processing Measure (SPM): Manual*. Los Angeles: Western Psychological Services.
- Pfeiffer, B., Daly, B. P., Nicholls, E. G., & Gullo, D. F. (2015). Assessing sensory processing problems in children with and without attention deficit hyperactivity disorder. *Physical & Occupational Therapy in Pediatrics*, 35(1), 1–12.
- Robinson, S., & Magill-Evans, J. (2009). Young children with autism spectrum disorder: Sensory processing and daily life skills. *Occupational Therapy Now*, 11(5), 11–13.
- Roley, S. S., Blanche, E. I., & Schaaf, R. C. (2001). *Understanding the nature of sensory integration with diverse populations*. San Antonio: Therapy Skill Builders.
- Rosenberg, L., Maeir, A., Yochman, A., Dahan, I., & Hirsch, I. (2015). Effectiveness of a cognitive – Functional group intervention among preschoolers with attention deficit hyperactivity disorder: A pilot study. *American Journal of Occupational Therapy*, 69(3), 1–8.
- Sanz-Cervera, P., Pastor-Cerezuela, G., Fernández-Andrés, M., & Tárraga-Mínguez, R. (2015). Sensory processing in children with Autism Spectrum Disorder: Relationship with non-verbal IQ, autism severity and Attention Deficit/Hyperactivity Disorder symptomatology. *Research in Developmental Disabilities*, 45, 188–201.
- Smith Roley, S., Mailloux, Z., Parham, L. D., Schaaf, R. C., Lane, C. J., & Cermak, S. (2015). Sensory integration and praxis patterns in children with autism. *American Journal of Occupational Therapy*, 69(1), 1–8.
- Spira, G. (2014). *A sensory intervention to improve sleep behaviors and social participation of children in Israel with sensory modulation disorder* (Unpublished doctoral dissertation). Fort Lauderdale: Nova Southeastern University.
- Tomchek, S. D., & Dunn, W. (2007). Sensory processing in children with and without autism: A comparative study using the short sensory profile. *American Journal of Occupational Therapy*, 61, 190–200.
- Watling, R. (2000). Interventions to facilitate auditory, visual, and motor integration in autism: A review of the evidence. *Journal of Autism and Developmental Disorders*, 30(5), 415–421.
- Watling, R. L., Deitz, J., & White, O. (2001). Comparison of Sensory Profile scores of young children with and without autism spectrum disorders. *American Journal of Occupational Therapy*, 55, 416–423.
- Watson, T. S., & Woodin, M. F. (2007). Test review of the Sensory Processing Measure. In R. A. Spies, J. F. Carlson, & K. F. Geisinger (Eds.), *The eighteenth mental measurements yearbook* (pp. 1–3). Lincoln: Buros Institute of Mental Measurements. Retrieved 15 Nov 2010, from the Buros Institute's Test Reviews Online Web site: <http://www.unl.edu/buros>.
- Wolfe, E. W., & Smith, E. V. (2007a). Instrument development tools and activities for measure validation using Rasch models: Part I – Instrument development tools. *Journal of Applied Measurement*, 8(1), 97–123.
- Wolfe, E. W., & Smith, E. V. (2007b). Instrument development tools and activities for measure validation using Rasch models: Part II – Validation activities. *Journal of Applied Measurement*, 8(2), 204–234.
- ZERO TO THREE. (2005). *Diagnostic classification of mental health and developmental disorders of infancy and early childhood, revised*. Washington, DC: ZERO TO THREE Press.

Sensory Processing Measure, Preschool Edition

► [Sensory Processing Measure: Preschool \(SPM-P\)](#)

Sensory Processing Measure: Preschool (SPM-P)

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Abbreviations

ASD	Autism spectrum disorder
BAL	Balance and motion
BOD	Body awareness
DIF	Environment Difference Early
ECIS	Childhood Intervention Service
HEA	Hearing Infant Toddler Sensory
IFSP	Profile
PLA SD	Planning and Ideas Standard Deviation
SI	Sensory integration
SOC	Social participation
SPD	Sensory processing disorder
TOT	Total sensory systems
TOU	Touch
VIS	Vision

Synonyms

[Sensory processing measure, preschool edition;](#)
[Sensory processing measure; SPM; SPM-P](#)

Description

The *Sensory Processing Measure – Preschool* (SPM-P) (Glennon et al. 2011; Miller Kuhaneck et al. 2010a, 2010b) is a set of scales that allows the assessment of praxis, social participation, and

sensory processing issues present in preschool-age children (2–5 years of age). The SPM-P consists of the Home Form (Ecker and Parham 2010) and the School Form (Miller Kuhaneck et al. 2010) that are published Western Psychological Services (www.wpspublish.com). The SPM-P is a companion instrument to the *Sensory Processing Measure* (SPM) (Parham et al. 2007) that is designed for use with children ages 5–12 years. The SPM-P has the same theoretical basis and subscale structure as the SPM. “These commonalities facilitate a seamless transition between the two assessments for children who need to be followed over the longer term” (Miller Kuhaneck et al. 2010, p. 3). It should be noted that the coverage of 5-year-olds is common to both the SPM and SPM-P; for 5-year-olds who have not yet started kindergarten, then the SPM-P should be used, whereas the SPM should be utilized for 5-year-olds who have already enrolled in kindergarten/grade one.

Similar to the SPM, the SPM-P is contextualized within Ayres sensory integration (SI) theory (Ayres 1972, 1979, 2005). “The theory holds that children with compromised sensory processing may be unable to learn efficiently, regulate their emotions, or function at an expected level in daily activities” (Miller Kuhaneck et al. 2010, p. 3). As well, sensory processing problems lead to difficulties in higher level integrative functions, such as praxis (the ability to plan and organize movement) and social participation (Dunn 2010). Ayres SI theory outlines assessment principles related to sensory function, three of which are incorporated into the SPM-P: (1) it includes *assessment of sensory systems* since it includes subscales that examine the tactile, visual, auditory, proprioceptive, and vestibular sensory systems; (2) it incorporates *assessment of sensory integration vulnerabilities* by providing information about the weaknesses in each sensory system including under-responsiveness, over-responsiveness, sensory-seeking behavior, and perceptual problems; and (3) it allows *assessment across multiple environments* since the clinician can compare a child’s sensory processing functioning across the home, preschool, and community contexts (Miller Kuhaneck et al. 2010).

The SPM-P Home Form consists of 75 items designed to be completed by a child’s parent or caregiver. The SPM-P School Form also contains 75 items meant to be completed by a child’s preschool teacher or child care provider. According to the SPM-P manual, a respondent “must have observed the child in the preschool or day care setting on a daily basis for at least 1 month prior to completing the School Form” (Miller Kuhaneck et al. 2010, p. 5). Both forms take approximately 15–20 min to complete and 5–10 min to score. The SPM-P forms are written at an 8th grade reading level, but in the event that parents have poor reading skills (e.g., English as a second language), it is possible to complete the Home Form by interviewing the respondent(s).

The SPM-P Home and School Forms have items that are answered by respondents using a 4-point Likert-type scale based on the frequency of a specific behavior occurring (never = 4, occasionally = 3, frequently = 2, always = 1). “Each form is an AutoScore™ Form that transfers the rater’s responses to a Scoring Worksheet via carbon paper. The Scoring Worksheet makes it easier to calculate the SPM-P scores” (Miller Kuhaneck et al. 2010, p. 3). Prior to giving the SPM-P Home or School Forms to respondents to fill out, the Summary Score Sheet must be removed and saved for later use when scoring the items. Based on the SPM-P scoring structure, “a higher raw score always indicates a higher level of problems or dysfunction than does a lower raw score” (Miller Kuhaneck et al. 2010, p. 14).

The SPM-P Home and School Forms generate eight norm-referenced standard subscale scores: social participation (SOC), vision (VIS), hearing (HEA), touch (TOU), body awareness (BOD) (refers to proprioception), balance and motion (BAL) (refers to vestibular function), planning and ideas (PLA) (refers to praxis), and Total Sensory Systems (TOT) (Miller Kuhaneck et al. 2010) (see Table 1). The SOC and PLA subscales have items that represent higher level SI functions that are impacted by sensory input but also include other cognitive and environmental factors. “The VIS, HEA, TOU, BOD, and BAL scales are referred to as the *sensory systems scales* because they address a child’s ability to process direct

sensory inputs. . .the TOT scale is a composite of all of the sensory systems scales, plus additional items that reflect taste and smell inputs” (Miller Kuhaneck et al. 2010, pp. 13–14). The SPM-P items that make up the SOC, VIS, HEA, TOU, BOD, BAL, PLA, and TOT scales (see Table 1) provide information about a number of processing SI vulnerabilities including under-responsiveness, over-responsiveness, sensory-seeking behavior, and perceptual problems (Miller Kuhaneck et al. 2010).

The standard scores (referred to as T-scores) for each subscale derived by both the SPM-P Home and Main Classroom Forms facilitates the classification of a child’s sensory processing functioning into one of three categories for clinical interpretation purposes: (1) the *typical* range (a T-score of 40–59) indicating a child’s behavioral and sensory functioning are similar to that of

Sensory Processing Measure: Preschool (SPM-P), Table 1 SPM-P forms and scales

SPM-P subscales	Number of items
<i>Home Form</i>	
Social participation (SOC)	8
Vision (VIS)	11
Hearing (HEA)	9
Touch (TOU)	14
Body awareness (BOD)	9
Balance and motion (BAL)	11
Planning and ideas (PLA)	9
Total sensory systems (TOT)	58
<i>School Form</i>	
Social participation (SOC)	10
Vision (VIC)	10
Hearing (HEA)	10
Touch (TOU)	10
Body awareness (BOD)	10
Balance and motion (BAL)	10
Planning and ideas (PLA)	10
Total sensory systems (TOT)	55

Source: Miller Kuhaneck, H., Ecker, C., Parham, L. D., Henry, D. A., & Glennon, T. J. (2010). *Sensory Processing Measure – Preschool (SPM-P): Manual*. Los Angeles, CA: Western Psychological Services, p. 4. Reproduced with permission from publisher

Note: The TOT scales include the VIS, HEA, TOU, BOD, and BAL items, plus several items representing taste and smell processing

typically developing peers; (2) the *some problems* range (a T-score of 60–69) indicating that a child exhibits mild-to-moderate difficulties in his/her behavioral or sensory functioning; or (3) the *definite dysfunction* range (a T-score of 70–80) indicating that a child is presenting with significant sensory processing problems that are likely having an impact on his/her daily functioning. “In addition, an Environment Difference (DIF) score allows direct comparison of the child’s sensory functioning between home and preschool/day care environments” (Miller Kuhaneck et al. 2010, p. 3).

Historical Background

The SPM was published in 2007 and the more recent companion SPM-P was published in 2010. When developing the SPM-P, the authors strived to develop items equivalent to the SPM. “The SPM-P authors selected items from the SPM and the *Evaluation of Sensory Processing* (Johnson-Ecker and Parham 2000) item set that could be adapted for use with preschoolers” (Miller Kuhaneck et al. 2010, p. 38). The two sensory processing instruments are designed to provide an assessment continuum for children ages 2–12 years who require longer term follow-up and monitoring.

Psychometric Data

Given that the SPM-P is a relatively new scale being published in 2010, limited psychometric data has been reported about it by external researchers and reviewers. The information reported here focuses on the reliability and validity data reported in the SPM-P manual published in 2010 (Miller Kuhaneck et al. 2010). The SPM-P Home Form and Main Classroom Form were standardized on an American sample of 651 (319 boys and 332 girls) typically developing children ages 2–5 years (Miller Kuhaneck et al. 2010). The sample group corresponded to percentage distribution of categories in relation to gender, age, race, parents’ education level, and

geographical regions from the American 2007 census. The SPM-P authors noted that in comparison to the 2007 census data on ethnicity, a larger percentage of Whites were included in the norming sample compared to Asians, Blacks, and Hispanics (who were underrepresented). In relation to the American Census geographical regions, the children in the sample from the Midwest were overrepresented, whereas the other three regions (North, South, and West) were underrepresented.

The norms for the SPM-P subscales are age-stratified to control for developmental differences between younger and older children with separate norms provided for 2-year-olds and 3–5-year-olds. Two-year-olds were included only if they attended a child care center where a staff member knew them well enough to complete the SPM-P School Form. Five-year-old children were only included in the standardization sample if they had not started kindergarten. None of the children included in the standardization group had special needs or were receiving early intervention services.

To investigate the potential moderating effects of the demographic features of the normative sample, the SPM-P authors used an effect size metric. This metric was used to investigate whether any of the “demographic moderator variables were associated with any clinical meaningful differences between groups in the SPM-P raw scores” (Miller Kuhaneck et al. 2010, p. 38). The effect size data supported the idea of using age-stratified norms and “differences in SPM-P scores due to gender, ethnicity, and SES were small and not likely to have any meaningful impact on the interpretation of test results” (Miller Kuhaneck et al. 2010, p. 40).

Types of reliability data often reported include internal consistency, test-retest/time sampling reliability/temporal stability, inter-rater/inter-scorer reliability, intra-rater/intra-scorer reliability, alternate form reliability, and split-half reliability (American Educational Research Association [AERA], American Psychological Association [APA], & National Council on Measurement in Education [NCME] 1999; Anastasi and Urbina 1997). For the SPM-P Home Form subscale

scores, internal consistency Cronbach alpha coefficients ranged from 0.75 to 0.93 (median = 0.92), while the for the SPM-P School Form subscale scores, internal consistency Cronbach alphas ranged from 0.72 to 0.94 (median = 0.82) (Miller Kuhaneck et al. 2010). “Five of eight Home scales and four of eight School scales have alphas of 0.80 or greater” (Miller Kuhaneck et al. 2010, p. 45). “The Home form resulted in reliability coefficients greater than or equal to 0.73 across all samples. The School form produced similar results across all samples with coefficients greater than or equal to 0.72. Given that there are separate norms provided for the 2- and 3- to 5-year-old samples it is unclear why reliability estimates were not provided for the 3- to 5-year age sample.” (Gokiart and Georgis 2010, p. 3).

Test-retest reliability data were collected on 49 healthy children (23 males and 26 females) ages 2–5 years within a 2-week follow-up period using both the SPM-P Home and School Forms. For the Home Form subscale scores, test-retest reliability coefficients ranged from 0.90 to 0.98 (median = 0.92), while for the Main Classroom Form subscale scores, test-retest reliability coefficients spanned from 0.90 to 0.96 (median = 0.97) (Miller Kuhaneck et al. 2010).

Types of validity often reported for tests include face validity, content validity, criterion-related validity, and construct validity (AERA, APA, & NCME, 1999). Two subtypes of criterion-related validity frequently included are concurrent validity and predictive validity. Two subtypes of criterion-related validity frequently included are concurrent validity and predictive validity, while subtypes of construct validity often reported include factor analysis validity, discriminant validity, convergent validity, divergent validity, diagnostic validity, and rating scale validity (Fawcett 2007; Wolfe and Smith 2007a). Many scales are now using a combination of classical test theory methods (e.g., factor analysis) and item response theory approaches (e.g., Rasch analysis) to examine the construct validity of a measure (Wolfe and Smith 2007b). The SPM-P manual reported a range of validity evidence.

Content validity for the SPM-P forms was ensured since the SPM-P items were derived

directly from the SPM. In turn, the SPM was developed based on items from two previously published scales: those being the evaluation of sensory processing (Johnson-Ecker and Parham 2000; Parham and Ecker 2002) and the School Assessment of Sensory Integration (Miller Kuhaneck et al. 2007). The SPM-P items were also reviewed by a panel of experts and revised based on their feedback. Finally, content validity was demonstrated since the SPM-P items (like those of the SPM) are based largely on the constructs of Ayres SI theory.

A research version of the SPM-P was subjected to confirmatory and exploratory factor analyses (a type of classical test theory approach to validity) to examine its construct validity. Findings of the factor analyses provided support for the basic scale structure of the SPM-P Home and Classroom Forms. Item-scale correlations are reported for the Home and Classroom Forms and provide evidence that supports the separate scoring and interpretation of the SPM-P scales.

The SPM-P authors used Rasch analysis (Bond and Fox 2001) to investigate its rating scale validity structure. With Rasch analysis, two sets of scores are generated, one for participants (person ability scores) and the other for the SPM items (item difficulty scores), expressed by means of a common numerical metric called a logit score (Bond and Fox 2001). The use of a common logit table allows instrument developers to examine the functioning of items with respect to the different ability levels of participants. In other words, the Rasch method provided insights into how the Likert-type scoring categories of the SPM-P item rating scale work together to measure differing amounts of sensory processing difficulties. The rating scale validity results indicated that each of the SPM-P's four rating scale categories (never, occasionally, frequently, always) “is the most probable response for a distinct segment of the underlying variable of sensory processing dysfunction. . .none of the categories are redundant. . .the category sequence of increasing behavioral frequency is properly aligned with the continuum of increasing sensory pathology” (Miller Kuhaneck et al. 2010, p. 55).

Convergent validity was examined by correlating the SPM-P Home Form with the Short Sensory Profile (Dunn 1999) using a sample of 105 children (74 boys and 31 girls) and the Infant/Toddler Sensory Profile (ITSP) (Dunn and Daniels 2002) using a sample of 62 children (42 boys and 20 girls). The SPM-P Home Form total score and the Short Sensory Profile total scores were moderately correlated with each other at $r = 0.62$. Subscale score correlations ranged from 0.06 to 0.59 with the majority of the interscale correlations being significant at $r > 0.20$ (Miller Kuhaneck et al. 2010). The correlations between the SPM-P Home Form and the ITSP were generally strong with correlations ranging from 0.04 to 0.53 with the majority of interscale correlations being $r > 0.25$ (Miller Kuhaneck et al. 2010).

Brown and Subel (2013b) explored the convergent validity the ITSP with the SPM-P Home and School Forms when completed by the parents of a group of 18 children aged between 23 and 36 months of age. Nine of the children were typically developing and nine of the children attended an early childhood intervention service (ECIS) since they were diagnosed with a developmental delay. The SPM-P School Forms were completed by an ECIS staff member for the children with a known developmental disability and a childcare teacher for the typically developing children. Spearman rho correlations were used to examine the associations between the ITSP and the SPM-P Home and School Form subscales. The ITSP and SPM-P Home Form had three of their subscales that were significantly correlated: ITSP Tactile and SPM-P Home Touch processing; ITSP Vestibular Processing and SPM-P Home Body Awareness; and ITSP Vestibular Processing and SPM-P Home Balance and Motion (Brown and Subel 2013b). Similarly, there were three pairs of scales from the ITSP and SPM-P School Form were significantly correlated: ITSP Auditory Processing and SPM-P School Balance; ITSP Vestibular Processing and SPM-P School Balance; and ITSP Oral Processing and SPM-P School Taste and Smell (Brown and Subel 2013b). The authors concluded that the ITSP and the SPM-P Home and

School Forms appear to assess several similar sensory processing factors.

The convergent validity of the SPM-P School Form was examined by correlating it with the Sensory Profile School Companion (Dunn 2006) using a sample of 20 children (16 boys and 4 girls). Correlation results were generally supportive of the convergent validity between the two sensory processing scales. Interscale correlation coefficients ranged from 0.07 to 0.64 with the majority of them being >0.44 (Miller Kuhaneck et al. 2010).

Discriminant validity was demonstrated for the SPM-P based on the data from a separate group (other than the standardization sample) of 242 children receiving occupational therapy intervention. The SPM-P subscales and items were able to differentiate between typically healthy from those with a known clinical diagnosis including autism spectrum disorder (ASD) (Miller Kuhaneck et al. 2010). "Even at the item level, the SPM-P shows robust capacity to differentiate between clinical and nonclinical groups" (Miller Kuhaneck et al. 2010, p. 63).

Brown and Subel (2013a) investigated the known-group validity of the Infant Toddler Sensory Profile and SPM-P Home and School Forms comparing two groups of children between the ages of 23 and 36 months (mean age 32 months, $SD = 2.55$). One group was typically developing and the second group was diagnosed with a developmental delay and was receiving services from an ECIS. The SPM-P School Forms were completed by an ECIS staff member for the children with a known developmental disability and a childcare teacher for the typically developing children. "Four of eight of the SPM-P Home Form subscales had statistically significant differences: touch, body awareness, balance and motion, and planning and ideas. On seven of eight of the SPM-P School Form subscales, statistically significant differences were identified: social participation, vision, hearing, touch, taste and smell, balance and motion, and planning and ideas" (Brown and Subel 2013a, p. 54).

In the SPM-P manual, results about the sensitivity and specificity of the Home and School Forms were reported. At the cutoff score of

$T = 60$, the SPM-P Home TOT scale had a sensitivity of 0.64 and specificity of 0.84 (Miller Kuhaneck et al. 2010). At the same cutoff score, the SPM-P School TOT scale had a sensitivity of 0.52 and specificity of 0.82 (Miller Kuhaneck et al. 2010).

In summary, the SPM-P Home and School Forms is a user-friendly scale that provides a detailed overview of a younger child's sensory processing weaknesses and strengths. Bain and Hunt (2010) commented that among the positive features of the SPM-P are "its quick and easy administration and scoring procedures. Information regarding interrater reliability and correlations across the Home and School forms are mostly lacking in the manual" (p. 3). Gokiart and Georgis (2010) report that the SPM-P "manual provides adequate reliability and validity evidence to support the inferences that are generated from the SPM-P eight scale scores and children's progress can be monitored into school using the companion SPM measure" (p. 3). However, Gokiart and Georgis (2010) also recommend that "additional construct evidence of validity, specifically for the separate age-based normative groups and in comparison to direct measures of function, would further enhance the utility of the tool" (p. 3).

Clinical Uses

The SPM-P provides invaluable information for practitioners related to the identification of and intervention with young children presenting with sensory processing difficulties. It can be used in a range of settings including clinical, community-based, child care, early intervention, early childhood education, hospital, rehabilitation, and research (Henry and McClary 2011). The SPM-P manual notes that it can be "used as either a quick screening instrument or as one component of a comprehensive diagnostic evaluation" (Miller Kuhaneck et al. 2010, p. 13).

The SPM-P forms can be used by a variety of health and education professionals (including occupational therapists, psychologists, early childhood education teachers, social workers, counselors, physiotherapists, speech-language

pathologists, nurses, pediatricians, physicians, and early intervention specialists among others) to evaluate the sensory processing issues of preschool-age children (Miller Kuhaneck et al. 2010). For example, the SPM-P's dual environment approach lets practitioners investigate why a child who functions well in a structured preschool classroom with a predictable daily routine may have problems in certain home environments. "The SPM-P can be administered by itself as a screening instrument, but the examiner should not use the results to make diagnostic or treatment decisions without first assembling the widest possible spectrum of information about the child" (Miller Kuhaneck et al. 2010, p. 4). It is recommended that other sources of information (e.g., medical records, clinical observations, interviews with parents and teachers) be accessed as well.

Olson et al. (2016) completed a multiple case study that investigated the effectiveness and usability of the Sensory Processing Measure-Preschool Quick Tips (SPM-P QT) by parents and teachers to implement an intervention program that was informed by results of the SPM-P. Four children between the ages of two and five who presented with apparent poor sensory modulation and self-regulation at school and at home took part in the study. The selection and application of sensory intervention strategies was guided by the SMP-P QT. When the children were re-assessed using the SPM-P, more improvements were noted on the School version than the Home version. After the study was completed, "feedback by both the parents and the school staff who had selected and implemented the SPM-P Quick Tips indicated progress in social participation and in activities of daily living dependent on sensory processing and praxis (such as tooth brushing, feeding, and sleeping)" (Olson et al. 2016, p. 142).

It has been documented that many children and adults with ASD exhibit sensory processing dysfunction (SPD) (Baker et al. 2008; Baranek et al. 2005, 2006; Dunn 2010; Kientz and Dunn 1997; Leekam et al. 2007; Rogers et al. 2003; Tomchek and Dunn 2007; Watling et al. 2001). The SPM-P manual states that "with moderate to severe

autism, it is not uncommon to see all SPM-P scales elevated in the Definite Dysfunction range” (Miller Kuhaneck et al. 2010, p. 25). It is typically expected that children diagnosed with ASD will have elevated scores on the SOC and PLA subscales. Difficulties in social functioning are one of the hallmark diagnostic criteria for ASD, so it is anticipated that children would have poor scores on the SOC subscale while the “deficits in participation and praxis may be caused or exacerbated by processing deficits in specific sensory systems” (Miller Kuhaneck et al. 2010, p. 25).

In summary, the SPM-P is a welcome addition to the range of tools that practitioners can utilize to assess children diagnosed with or suspected of having ASD. The SPM-P hones in issues indicative of SPD. It is a companion instrument to the SPM and allows monitoring of a child’s sensory processing from 2 to 12 years of age. The SPM-P is based on the same subscale structure and SI theory as the SPM, thus allowing continuity of assessment, intervention, and monitoring from preschool settings to primary school contexts. The SPM-P provides information about two different environments (home and early childhood education settings) based on the input and views of two different respondents (parent/caregiver and preschool teacher). This feature of the SPM-P also fits with the principles of family-centered practice since the opinions, input, and views of parents and care givers are solicited. The ease of scoring and interpreting the results is another attractive characteristic of the SPM-P. These features make it easier for practitioners to explain test results and engage parents in the intervention process.

Both the SPM and SPM-P can be used for evidence-based practice, scientifically based research, differentiated instruction, and monitoring of progress of a child receiving services (Miller Kuhaneck et al. 2010). The SPM-P also has extensive reliability and validity data reported about it in its manual. However, given that it is a relatively new measure, it is recommended that the SPM-P be investigated more closely by external reviewers and investigators so that a related body of psychometric knowledge can be developed about the scale. The SPM-P is highly

recommended for use by practitioners who work with children diagnosed with ASD.

See Also

- Occupational Therapy (OT)
- Sensation Avoiding
- Sensation-Seeking
- Sensory Diet
- Sensory Experiences Questionnaire
- Sensory Impairment in Autism
- Sensory Integration (SI) Therapy
- Sensory Processing
- Sensory Processing Assessment
- Sensory Sensitivity Questionnaire: Revised
- Sensory Stimuli
- Standardized Tests

References and Reading

- American Educational Research Association, American Psychological Association, & National Council on Measurement in Education. (1999). *Standards for educational and psychological testing*. Washington, DC: American Psychological Association.
- Anastasi, A., & Urbina, S. (1997). *Psychological testing*. Upper Saddle River, NJ: Prentice Hall International.
- Ayres, A. J. (1972). *Sensory integration and learning disorders*. Los Angeles, CA: Western Psychological Services.
- Ayres, A. J. (1979). *Sensory integration and the child*. Los Angeles, CA: Western Psychological Services.
- Ayres, A. J. (2005). *Sensory integration and the child, 25th anniversary edition*. Los Angeles, CA: Western Psychological Services.
- Bain, S. K., & Hunt, A. (2010). Test review of the Sensory Processing Measure-Preschool. In J. F. Carlson, K. F. Geisinger, & J. L. Jonson (Eds.), *The nineteenth mental measurements yearbook* (pp. 1–3). Lincoln, NE: Buros Institute of Mental Measurements. Retrieved February 8, 2017, from the Buros Institute’s Test Reviews Online Web site: <http://www.unl.edu/buros>.
- Baker, A. E. Z., Lane, A., Angley, M. T., & Young, R. L. (2008). The relationship between sensory processing patterns and behavioral responsiveness in autistic disorder: A pilot study. *Journal of Autism and Developmental Disorders*, 38, 867–875.
- Baranek, G. T., Parham, L. D., & Bodfish, J. W. (2005). Sensory and motor features in autism: Assessment and intervention. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Assessment,

- interventions and policy, Vol. 2, pp. 831–857). Hoboken, NJ: Wiley.
- Baranek, G. T., David, F. J., Poe, M. D., Stone, W. L., & Watson, L. R. (2006). Sensory experiences questionnaire: Discriminating sensory features in young children with autism, developmental delays, and typical development. *Journal of Child Psychology and Psychiatry*, 47(6), 591–601.
- Bond, T. G., & Fox, C. M. (2001). *Applying the Rasch model: Fundamental measurement in the human sciences*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Brown, T., & Subel, C. (2013a). Known-group validity of the Infant Toddler Sensory Profile and the Sensory Processing Measure-Preschool. *Journal of Occupational Therapy, Schools & Early Intervention*, 6(1), 54–72.
- Brown, T., & Subel, C. (2013b). Comparing the Infant Toddler Sensory Profile (ITSP) and the Sensory Processing Measure-Preschool (SPM-P) when used with preschool-aged children: A pilot study. *Irish Journal of Occupational Therapy*, 40(1), 11–21.
- Dunn, W. (1999). *Sensory Profile: User's manual*. San Antonio, TX: Psychological Corporation.
- Dunn, W. (2006). *Sensory Profile School Companion*. San Antonio, TX: Psychological Corporation.
- Dunn, W. (2010). Sensory processing: A critical assessment area for autism spectrum disorders. In F. Volkmar (Ed.), *Autism and autism spectrum disorders: History, diagnosis, neurobiology, treatment and outcome*. London: The Biomedical & Life Sciences Collection, Henry Stewart Talks Ltd. Retrieved from <http://hstalks.com/bio>.
- Dunn, W., & Daniels, D. (2002). Initial development of the Infant/Toddler Sensory Profile. *Journal of Early Intervention*, 25, 2–41.
- Ecker, C., & Parham, L. D. (2010). *Sensory Processing Measure – Preschool (SPM-P) home form*. Los Angeles, CA: Western Psychological Services.
- Fawcett, A. L. (2007). *Principles of assessment and outcome measurement for occupational therapists and physiotherapists: Theory, skills and application*. Hoboken, NJ: Wiley.
- Glennon, T. J., Miller Kuhaneck, H., & Herzberg, D. (2011). The Sensory Processing Measure-Preschool (SPM-P)-part one: Description of the tool and its use in the preschool environment. *Journal of Occupational Therapy, Schools & Early Intervention*, 4(1), 42–52.
- Gokiart, R., & Georgis, R. (2010). Test review of the Sensory Processing Measure-Preschool. In J. F. Carlson, K. F. Geisinger, & J. L. Jonson (Eds.), *The nineteenth mental measurements yearbook* (pp. 1–3). Lincoln, NE: Buros Institute of Mental Measurements. Retrieved February 8, 2017, from the Buros Institute's Test Reviews Online Web site: <http://www.unl.edu/buros>.
- Henry, D. A., & McClary, M. (2011). The Sensory Processing Measure-Preschool (SPM-P)-part two: Test-retest and collective collaborative empowerment, including a father's perspective. *Journal of Occupational Therapy, Schools & Early Intervention*, 4(1), 53–70.
- Johnson-Ecker, C. L., & Parham, L. D. (2000). The Evaluation of Sensory Processing: A validity study using contrasting groups. *American Journal of Occupational Therapy*, 54(5), 494–503.
- Kientz, M. A., & Dunn, W. (1997). A comparison of the performance of children with and without autism on the Sensory Profile. *American Journal of Occupational Therapy*, 51, 530–537.
- Leekam, S. R., Nieto, C., Libby, S. J., Wing, L., & Gould, J. (2007). Describing the sensory abnormalities of children and adults with autism. *Journal of Autism and Developmental Disorders*, 37, 894–910.
- Miller Kuhaneck, H., Henry, D. A., Glennon, T. J., & Mu, K. (2007). Development of the Sensory Processing Measure – School: Initial studies of reliability and validity. *American Journal of Occupational Therapy*, 61(2), 170–175.
- Miller Kuhaneck, H., Ecker, C., Parham, L. D., Henry, D. A., & Glennon, T. J. (2010a). *Sensory Processing Measure – Preschool (SPM-P): Manual*. Los Angeles, CA: Western Psychological Services.
- Miller Kuhaneck, H., Henry, D. A., & Glennon, T. J. (2010b). *Sensory Processing Measure – Preschool (SPM-P) school form*. Los Angeles, CA: Western Psychological Services.
- Olson, C. H., Henry, D. A., Klinner, A. P., Kylo, A., Richter, C. M., Charley, J., & . . . Walworth, J. (2016). Effectiveness and usability of the Sensory Processing Measure-Preschool quick tips: Data-driven intervention following the use of the SPM-Preschool in an early childhood, multiple-case study. *Journal of Occupational Therapy, Schools & Early Intervention*, 9(2), 142–162.
- Parham, L. D., & Ecker, C. (2002). Evaluation of sensory processing: Research version 4. In A. C. Bundy, S. J. Lane, & E. A. Murray (Eds.), *Sensory integration: Theory and practice* (2nd ed., pp. 194–196). Philadelphia: F.A. Davis.
- Parham, L. D., Ecker, C., Miller Kuhaneck, H., Henry, D. A., & Glennon, T. J. (2007). *Sensory Processing Measure (SPM): Manual*. Los Angeles: Western Psychological Services.
- Rogers, S. J., Hepburn, S., & Wehner, E. (2003). Parent reports of sensory symptoms in toddlers with autism and those with other developmental disorders. *Journal of Autism and Developmental Disorders*, 33, 631–642.
- Smith, E. V. (2001). Evidence for the reliability of measures and validity of measure interpretation: A Rasch measurement perspective. *Journal of Applied Measurement*, 2, 281–311.
- Smith, E. (2002). Detecting and evaluating the impact of multidimensionality using item fit statistics and principal component analysis of residuals. *Journal of Applied Measurement*, 3, 205–231.
- Tomchek, S. D., & Dunn, W. (2007). Sensory processing in children with and without autism: A comparative study using the Short Sensory Profile. *American Journal of Occupational Therapy*, 61, 190–200.

- Watling, R. L., Deitz, J., & White, O. (2001). Comparison of sensory profile scores of young children with and without autism spectrum disorders. *American Journal of Occupational Therapy*, 55, 416–423.
- Wolfe, E. W., & Smith, E. V. (2007a). Instrument development tools and activities for measure validation using Rasch models: Part I – Instrument development tools. *Journal of Applied Measurement*, 8(1), 97–123.
- Wolfe, E. W., & Smith, E. V. (2007b). Instrument development tools and activities for measure validation using Rasch models: Part II – Validation activities. *Journal of Applied Measurement*, 8(2), 204–234.

comparison to neurotypical peers (Talay-Ongan and Wood 2000, p. 203). This parent-completed questionnaire includes a total of 54 close-ended items, 9 in each of 6 sensory domains: auditory, visual, tactile, gustatory, vestibular, and olfactory. Items from each modality are randomly distributed within the questionnaire. Respondents rate each item *yes* or *no* based on behaviors typically displayed by the child. Descriptive comments can be recorded for each item at the discretion of the respondent. Example items include:

- My child seems oblivious to (often bad) odors.
- Certain sounds appear to be painful to my child.

Only one paper using the SSQ-R was published in the literature though May 2011.

Sensory Seeking

► Sensation-Seeking

Sensory Sensitivity Questionnaire (SSQ)

► Glasgow Sensory Questionnaire (GSQ)

Sensory Sensitivity Questionnaire: Revised

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Synonyms

SSQ-R

Description

The SSQ-R was developed as a preliminary tool with the specific aim of informing formal methods of assessing “the nature and extent of sensory hyper- and hypo-sensitivities across all modalities in children with autism” in

Historical Background

The Sensory Sensitivity Questionnaire-Revised (SSQ-R) was developed through a process involving a review of caregiver-report questionnaires of sensory-based performance and behavior by Bettison (1992, 1994), Edelson (1992), and Ayres (1979). Thirty-seven items were selected, revised, and grouped into auditory, visual, tactile, gustatory, olfactory, and vestibular domains. The resulting 37-item measure was pilot tested on a small sample of nine children with autism and age- and gender-matched controls in Australia. Twenty-two items on the initial version were found to discriminate between the children with and without autism in the sample. These 22 items were retained. The remaining items were revised based on qualitative comments provided by the pilot study participants. Additional items were developed through further analysis of the sources originally consulted during test development in order to have an equal number of test items in each sensory domain. The resulting 54-item questionnaire is titled the Sensory Sensitivity Questionnaire-Revised (SSQ-R).

Psychometric Data

Psychometric investigation of the SSQ-R is limited. Authors state that content validity is assumed because items describe behaviors believed to reflect sensory hyper- or hyposensitivities. Concurrent validity is also assumed due to the substantial similarities to other tools examining the same processes. The one published study that examined the SSQ-R reported that 45 of the 54 items were found to discriminate between children with autism and those with neurotypical development using a Cronbach's coefficient alpha criteria of 0.95 (Talay-Ongan and Wood 2000).

Clinical Uses

The close-ended, yes/no response for each item makes this tool likely easy and quick to use. However, it is currently available through the author only; therefore, it is unlikely to be encountered in clinical documentation, though it may be more heavily used in the region where the tool was developed.

See Also

- [Tactile Defensiveness](#)
- [Touch Sensitivity](#)

References and Reading

- Ayres, A. J. (1979). *Sensory integration and the child: Sensory history*. Los Angeles: Western Psychological Services.
- Bettison, S. (1992). Sound sensitivity in autism. In *Autism: The puzzle. Are the pieces starting to fit? National conference 1992 proceedings* (pp. 100–108). Melbourne: Victorian Autistic Children's and Adult's Association.
- Bettison, S. (1994). "Auditory training" as a treatment for sound sensitivity in autism: Preliminary results. *Special Education Perspectives*, 3, 1.
- Edelson, S. M. (1992). *Sensory problems questionnaire*. Newberg: Center for the Study of Autism.
- Talay-Ongan, A., & Wood, K. (2000). Unusual sensory sensitivities in autism: A possible crossroads. *International Journal of Disability, Development and Education*, 47, 201–212.

Sensory Stimuli

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Definition

A sensory stimulus is any event or object that is received by the senses and elicits a response from a person. The stimulus can come in many forms such as light, heat, sound, touch, as well as from internal factors. Sensory stimuli can be perceived by the auditory, tactile, visual, gustatory, proprioceptive, and vestibular systems. One characteristic of people with ASD is difficulty with perceiving stimuli as the information is often interpreted differently by the person's brain. Unusual responses to sensory stimuli are typically referred to as hypo- or hyper-sensitive reactions. "Dunn (1997) described the unusual sensory reactions of children by defining hyper-reactivity as having a lowered threshold to sensory input (sensory avoiding), or low tolerance to sensory input (sensory sensitivity), and hypo-reactivity as a higher threshold to sensory input (poor registration) or seeking out sensory input (sensory seeking)" (Jasmin et al. 2009).

See Also

- [Sensory Impairment in Autism](#)
- [Sensory Processing](#)
- [Stimulus](#)

References and Reading

- Dunn, W. (1997). The impact of sensory processing abilities on the daily lives of young children and their families: A conceptual model. *Infants and Young Children*, 9, 23–35.
- Engel-Yeger, B., Hardal-Nasser, R., & Gal, E. (2011). Sensory processing dysfunctions as expressed among children with different severities of intellectual developmental disabilities. *Research in Developmental Disabilities: A Multidisciplinary Journal*, 32(5), 1770–1775.

- Jasmin, E., Couture, M., McKinley, P., Reid, G., Fombonne, E., & Gisel, E. (2009). Sensori-motor and daily living skills of preschool children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(2), 231–241.
- Leekam, S. R., Nieto, C., Libby, S. J., Wing, L., & Gould, J. (2007). Describing the sensory abnormalities of children and adults with autism. *Journal of Autism and Developmental Disorders*, 37(5), 894–910.

Sensory System for Sense of Hearing

► Auditory System

Sentence Structure

► Syntax

Separate Classroom

► Self-Contained Classroom

Separation Anxiety Disorder

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Synonyms

SAD

Short Description or Definition

Separation anxiety disorder (SAD) is characterized by excessive fear of separation from a person to whom a child is attached (American Psychiatric Association [APA] 2000). Children with SAD usually worry that if separated from a significant person (usually a parent) something bad will happen to them, such as being kidnapped. Additionally, they may worry that something bad will happen to the attachment figure, such as injury or death. Physical symptoms when separation is anticipated coincide with such worries (e.g., stomachaches). The disturbance stemming from SAD symptoms causes significant interference in the child's home, social, or school life. For a diagnosis of SAD (APA 2000), individuals must experience these symptoms for at least 4 weeks and have an onset before 18 years of age. This area of anxiety is commonly experienced, at a clinical level, by children and youth with ASD.

Categorization

SAD is categorized as a childhood anxiety disorder in the DSM-IV-TR. It is one among a variety of child onset disorders.

Epidemiology

Rates of anxiety disorders in the ASD population have varied between 11% and 84% (Muris et al. 1998; Simonoff et al. 2008). Incidence rates of SAD in the ASD population vary from 0.5% to 19% (Leyfer et al. 2006; Mattila et al. 2010; Simonoff et al. 2008; Sukhodolsky et al. 2008). A review of prevalence rates in typically developing children indicates rates of SAD to be between 0.5% and 20.2% (Cartwright-Hatton et al. 2006). In a comparison study of school-age children with and without a pervasive developmental disorder (PDD), Gadow and colleagues (2005) found similar SAD symptom prevalence rates based on parent report in the PDD clinical sample and the non-PDD clinical sample.

Research indicates that SAD may occur more frequently in children with ASD who have higher cognitive functioning ($IQ > 70$; Sukhodolsky et al. 2008). Of children with ASD and greater cognitive ability, 19% were found to have a co-occurring SAD. SAD commonly presents comorbidly with other anxiety disorders such as generalized anxiety disorder and social phobia.

Natural History, Prognostic Factors, and Outcomes

Little is known about the natural history of SAD in the ASD population. In the typically developing population, many factors such as genetics, anxious temperaments, certain parenting behaviors, and stressful life events are thought to influence the development of anxiety disorders, particularly SAD (Craske 1999; Eaves et al. 1997; Rapee 2001; Wood 2006). One recent treatment demonstrated that improvements in a putative parenting mechanism linked with SAD (intrusiveness) over the course of the acute phase of intervention mediated a reduction of anxiety in school-age children at a 1-year follow-up assessment (Wood et al. 2009). It is hypothesized that when parents take over tasks that children could do for themselves (intrusiveness), it reduces children's self-efficacy for effective action when away from parents, heightening separation anxiety (Wood 2006). Interestingly, intrusive behaviors are associated specifically with SAD symptoms and not other anxiety disorders. The initial clinical evidence from this treatment study highlights the possibility of a dynamic (reversible) effect of intrusive parenting on SAD, but it does not imply that such parenting initially caused the anxiety. Little is known about prognostic factors for SAD in individuals with ASD.

For children with ASD, preliminary studies of CBT for the treatment of anxiety, including SAD, in individuals with ASD have shown positive outcomes with similar rates of anxiety remission as observed among typically developing children with anxiety disorders (Chalfant et al. 2007; Reaven et al. 2009; Sofronoff et al. 2005; Wood et al. 2009a, b). Although various studies have

shown positive responses to CBT, the studies have not specifically examined SAD separately. These studies have treated multiple anxiety disorders, given the finding that most children with ASD referred for treatment have multiple anxiety disorders, not just SAD. Further research is needed to determine what factors are associated with treatment outcomes for SAD symptoms, specifically.

Clinical Expression and Pathophysiology

Children with SAD experience specific cognitive, behavioral, and physiological manifestations of anxiety related to separation (APA 2000). While separation anxiety is normative in infants and toddlers, it typically declines once toddlers reach 30 months of age (Marks 1987; Ehrenreich et al. 2008). Separation anxiety past 30 months of age is atypical and can be SAD-related based on the severity of behaviors and worries. A core anxious belief in children with SAD is an excessive fear that something bad will happen to themselves or a significant person (e.g., parent) if separated, causing significant interference in family, school, and social functioning (e.g., school failure). As a result, children may demonstrate worry when they anticipate a separation (e.g., a child may cry or beg parents not to leave upon learning of an impending separation such as a parent going to run errands while leaving the child at home). Further, there may be places children refuse to go, for example, school, because they are scared to be separated. Children often want the parent near them at bedtime until they have fallen asleep or insist on sleeping with their parents (co-sleeping). It is, of course, important to disentangle whether co-sleeping is a deviation from cultural or family expectations in determining whether this behavior may reflect SAD. Children may be afraid to be alone in their house or in parts of their house even when family members are at home (e.g., child is afraid to be in the bedroom when family is in the living room). They may follow family members around the home or request to be accompanied to different rooms in the home. Children also often have bad dreams

about being separated from loved ones, such as being lost, or being caught in a natural disaster. These persistent fears and worries cause significant distress, and some children experience accompanying physical symptoms such as nausea, vomiting, and headaches.

Sze and Wood (2007) conducted a case study of an 11-year-old girl with concurrent ASD and SAD and described common fears that children with ASD and SAD experience. The youngster was reluctant to be separated from her parents due to worries that she would be kidnapped or that her parents would be killed while they were apart. She would repeatedly call her parents, up to 20 times a day, when they were away from her. Further, during the night, she would frequently join her parents to sleep in their bed. She would not stay over at her grandparents' house or engage in age-appropriate social activities such as sleepovers due to fear of separation from parents.

Evaluation and Differential Diagnosis

Evaluation of SAD in individuals with ASD is still largely conducted with instruments developed for use in the typically developing population. SAD is commonly assessed with semi-structured interviews based on DSM-IV criteria, which involve clinicians, parents, and children. In such semi-structured interviews, clinicians ask the parent and child questions concerning the child's anxiety and distress when separation is anticipated. Further questions focus on worries that the child might have prior to and during a separation, with particular focus on worries about harm befalling the child or parent during a separation. Additional questions focus on avoidance or refusal to go places without parents and avoidance or refusal to go to sleep without their parents. Interviews also include questions on physical symptoms that may occur in conjunction with these worries. After gathering information on specific symptoms, clinicians ask about the impact these anxiety symptoms have in home, social, or academic life. For example, parents may be unable to leave their

child with a babysitter or to be in a separate room from their child while in their house, often resulting in considerable conflict among multiple family members. Additionally, SAD-related symptoms can significantly interfere with children's social life if they avoid play dates or cry in front of friends at separation situations like school drop-off. Further, SAD-related symptoms such as intermittent or chronic school refusal can significantly interfere with a child's academic life. To meet DSM-IV-TR diagnostic criteria for SAD, children must exhibit three of the core symptoms for at least 4 weeks and demonstrate significant interference in child's home, social, or academic life or experience significant distress related to the anxiety symptoms.

In addition to clinical interviews, parent, teacher, and self-reports of these behaviors are useful in evaluating a child for SAD. Although there are often discrepancies among reporters and inter-rater agreement can be modest, such measures are useful in gaining a full understanding of the child's behaviors in different settings. Few measures have been specifically developed to assess SAD in the ASD population, although the Child and Adolescent Symptom Inventory (CASI) developed by Gadow and Sprafkin (1994, 1997) does contain a SAD subscale. Research is beginning to show, however, preliminary evidence for the validity of measures established in the typically developing population in the ASD population (Chalfant et al. 2007; Wood et al. 2009).

The Anxiety Diagnostic Interview Scale for DSM-IV – Child and Parent (ADIS C/P; Silverman and Albano 1996) is a frequently used semi-structured interview conducted separately with the child and parent in order to assess the child's level of anxiety with regard to several different anxiety disorders, including SAD, and has been used in ASD samples (e.g., Chalfant et al. 2007; White et al. 2009; Wood et al. 2009a). The ADIS has very favorable psychometric properties in the typically developing population (e.g., Wood et al. 2002) with preliminary evidence of acceptable inter-rater reliability and

treatment sensitivity in ASD (Chalfant et al. 2007; Wood et al. 2009a, b). From the interview, severity scores are generated for each anxiety diagnosis, ranging from 0 to 8 (with higher scores representing more anxiety and a 0 representing no diagnosis), for each anxiety disorder diagnosis. A score of 4 (moderate) or greater is deemed as clinically significant.

The Multidimensional Anxiety Scale for Children – Child and Parent (MASC C/P; March 1998) is another measure commonly used in the ASD population to assess anxiety. The MASC is a 39-item paper and pencil measure where children and parents rate the child's level of anxiety on a 4-point Likert scale. The MASC is composed of four subscales, including physical symptoms, social anxiety symptoms, harm avoidance, and separation. The MASC has robust psychometric properties in typically developing samples (March et al. 1997); however, its validity in ASD samples is still being determined (Bellini 2004; White et al. 2009).

Evaluation of SAD in the ASD population is complicated by the wide variability in ASD symptomology (Bellini 2004; Wood and Gadow 2010). Similar to other psychiatric conditions, it can be difficult to tease apart SAD symptoms from ASD symptoms. Characteristics associated with ASD such as social difficulties, adherence to routines, and lower adaptive functioning skills may complicate recognition of SAD in these individuals. For example, not partaking in play dates may be symptomatic of SAD if avoidance is due to worries about being separated from a parent and not just symptomatic of ASD-related social deficits. Additionally, adherence to particular bedtime routines may be symptomatic of SAD, and not just ASD, if the bedtime routine requires that the parent staying close to the child throughout the night. Parent assistance in daily living skills due to SAD-related fears (not wanting to separate from parent) also can be distinguished from parent assistance due to lower adaptive functioning skills. For example, a child may need assistance bathing due to lower adaptive skills (ASD-related) or the parent may be providing assistance because the

child insists that the parent be in the same room as him or her (SAD related). Diagnosticians must be skilled in distinguishing between ASD- and SAD-related symptoms to determine the possible impact of SAD on a child's functioning. In addition, there is still considerable controversy over whether "true" psychiatric syndromes occur in the ASD population or if they are an extension of the ASD diathesis (see, e.g., Wood and Gadow 2010). More research is needed to describe the relation between ASD, SAD, and other psychiatric conditions.

Treatment

Various treatments have been suggested for SAD in the ASD population. There is preliminary evidence for the efficacy of modified cognitive behavioral therapy (CBT) in treating SAD in children with high-functioning autism (HFA; Chalfant et al. 2007; Reaven et al. 2009; Sofronoff et al. 2005; Wood et al. 2009). Traditional CBT for typically developing children often consists of psychoeducation and coping skills training. Once new coping skills have been taught, sessions consist of practicing these new skills in increasingly feared situations (Barrett et al. 1996; Kendall et al. 1997).

Enhancements of CBT for individuals with ASD address common impairments associated with ASD in social communication, perspective taking, and understanding of emotions and social cues (Sze and Wood 2007, 2008). There is often an additional emphasis on increasing self-help skills, using cartoons to convey emotions and thoughts, and creating a reward system to increase motivation. Special interests have also successfully been incorporated into treatment to teach coping skills and increase motivation (Sofronoff et al. 2005; Sze and Wood 2007, 2008; Wood et al. 2009).

In the case study by Sze and Wood (2007), a modified CBT treatment plan was effective in treating SAD in an 11-year-old girl also diagnosed with ASD. Characteristic of CBT treatment began

with psychoeducation and coping skills training. To address some of the client's ASD-related symptoms, which can interfere with traditional CBT practices, the therapist used more concrete spoken language and placed a greater emphasis on role playing and visual materials. Additionally, she illustrated abstract concepts using simpler terms and the client's special interests as examples. Further, the therapist employed an interactive rather than didactic style of communication during sessions. A modification of CBT that may be particularly important for the treatment of SAD in the ASD population was the incorporation of work on independent living skills (also see Drahota et al. 2011). The therapist encouraged the parent to gradually reduce the level of assistance in self-help skills and offer choices to increase child's confidence and self-efficacy. After the psychoeducation and skills training components, the therapist developed an anxiety hierarchy with the family to serve as a treatment plan for the rest of the sessions. This hierarchy not only included items pertaining to the child's anxiety and fears but also ASD symptoms. Examples of items included on the participant's hierarchy for SAD were "staying at grandparents' house for 1 h without calling parents, staying at grandparents' house overnight with only one telephone call to parents, and sleeping in my own bed throughout the night without entering parents' bedroom." Additionally, items pertaining to ASD that were included on the hierarchy were "playing and eating with a target peer at recess and lunch, initiating a telephone call to a target peer, and inviting a target peer over for a play date, acting like a good host." The therapist and child discussed coping thoughts for these feared situations. The child was assigned homework to sleep independently, reduce the number of times she called her parents when they were away, and spend time at her grandparents' house gradually without calling her parents. Within 3 weeks of doing these SAD-related exposures, she no longer reported any anxiety concerning separation from her parents. SAD-related worries stayed in remittance throughout the rest of treatment, and she no longer met criteria for SAD at the posttreatment assessment.

Clinical trials that have included SAD as one of the targeted anxiety disorders have demonstrated positive outcomes with modified CBT in this population. Although specific information about the remittance rates for SAD have not been included in the trials, which include children with several types of anxiety disorders, the interventions were effective in relieving anxiety symptoms overall. Wood and colleagues (2009) conducted a randomized controlled trial to examine the efficacy of CBT in 40 children aged 7–11 years old. The treatment program included psychoeducation, coping skills training, and in vivo exposures. Additionally, therapists consulted with schoolteachers and assigned weekly homework for the child to practice at home. At the intake assessments, participants had an average of 4.18 psychiatric disorders, and at posttreatment, over half of the participants in the immediate treatment condition had remission of all anxiety disorders. Chalfant et al. (2007) also conducted a randomized controlled trial for 47 individuals aged 8–13 with HFA and anxiety disorders examining group CBT. At post-assessments, they found that significantly more children in the immediate treatment condition no longer met criteria for an anxiety disorder than the waitlist condition. Reaven et al. (2009) examined group CBT for individuals aged 8–14 with HFA and found significant decreases of anxiety in the immediate treatment versus waitlist group on parent reports of anxiety. Significant decreases in parent-reported SAD symptoms were observed in the immediate treatment group at posttreatment. These studies indicated that modified cognitive behavioral therapy can be an effective treatment modality for treating anxiety disorders in children and youth with HFA.

Medication, relaxation techniques, modeling, behavior modification, and a variety of other therapies have also been explored as treatments for SAD in the typically developing population. While it is likely these practices are used in community settings for children with ASD, there has been little empirical evidence for their efficacy in the ASD population. Selective serotonin reuptake inhibitor (SSRI) medications are frequently prescribed for anxiety in children with ASD, and while there is evidence for their use in the

treatment of anxiety disorders in typically developing children, no clinical trials have examined their efficacy for anxiety reduction in the ASD population. Research on effective treatments for SAD as well as other anxiety disorders in the ASD population is indicated.

See Also

► Cognitive Behavioral Therapy (CBT)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual* (4th ed., Text Rev.). Washington, DC: APA Press.
- Barrett, P. M., Dadds, M. R., & Rapee, R. M. (1996). Family treatment of childhood anxiety: A controlled trial. *Journal of Consulting and Clinical Psychology*, 64, 333–342.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development*, 71, 447–456.
- Bellini, S. (2004). Social skill deficits and anxiety in high-functioning adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 19, 78–86.
- Cartwright-Hatton, S., McNicol, K., & Doubleday, E. (2006). Anxiety in a neglected population: prevalence of anxiety disorders in pre-adolescent children. *Clinical Psychology Review*, 26, 817–833.
- Chalfant, A. M., Rapee, R., & Carroll, L. (2007). Treating anxiety disorders in children with high functioning autism spectrum disorders: A controlled trial. *Journal of Autism and Developmental Disorders*, 37, 1842–1857.
- Craske, M. G. (1999). *Anxiety disorders: Psychological approaches to theory and treatment*. Boulder: Westview Press.
- Drahota, A., Wood, J. J., Sze, K. M., & Van Dyke, M. (2011). Effects of cognitive behavioral therapy on daily living skills in children with high-functioning autism and concurrent anxiety disorders. *Journal of Autism and Developmental Disorders*, 41(3), 257–265.
- Eaves, L. J., Silberg, J. L., Maes, H. H., Simonoff, E., Pickles, A., Rutter, M., et al. (1997). Genetics and developmental psychopathology 2: The main effects of genes and environment on behavioral problems in the Virginia twin study of adolescent behavioral development. *Journal of Child Psychology and Psychiatry*, 38, 965–980.
- Ehrenreich, J. T., Santucci, L. C., & Weiner, C. (2008). Separation anxiety disorder in youth: Phenomenology, assessment and treatment. *Psicolog? A Conductual*, 16, 389–412.
- Gadow, K. D., & Sprafkin, J. (1994). *Child symptom inventory-4*. Stony Brook: Checkmate Plus.
- Gadow, K. D., & Sprafkin, J. (1997). *Adolescent symptom inventory-4 screening manual*. Stony Brook: Checkmate Plus.
- Kendall, P. C., Flannery-Schroeder, E., Panichelli-Mindel, S. M., Southam-Gerow, M., Hein, A., & Warman, M. (1997). Therapy for youths with anxiety disorders: A second randomized clinical trial. *Journal of Consulting and Clinical Psychology*, 65, 366–380.
- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., et al. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36, 849–861.
- March, J. (1998). *Manual for the multidimensional anxiety scale for children*. Toronto: Multi-Health Systems.
- March, J. S., Parker, J. D., Sullivan, K., Stallings, P., & Conners, C. K. (1997). The Multidimensional Anxiety Scale for Children (MASC): Factor structure, reliability, and validity. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 554–565.
- Marks, I. M. (1987). *Fears, phobias and rituals: Panic, anxiety and their disorders*. New York: Oxford University Press.
- Mattila, M., Hurtig, T., Haapsamo, H., Jussila, K., Kuusikko-Gauffin, S., Kielinen, M., et al. (2010). Comorbid psychiatric disorders associated with Asperger syndrome/high-functioning autism: A community- and clinic-based study. *Journal of Autism and Developmental Disorders*, 40(9), 1080–1093.
- Muris, P., Steerneman, P., Merkelbach, H., Holdrinet, I., & Meesters, C. (1998). Comorbid anxiety symptoms in children with pervasive developmental disorders. *Journal of Anxiety Disorders*, 12, 387–393.
- Rapee, R. M. (2001). The development of generalized anxiety. In M. W. Vasey & M. R. Dadds (Eds.), *The developmental psychopathology of anxiety*. New York: Oxford University Press.
- Reaven, J., Blakeley-Smith, A., Nichols, S., Dasari, M., Flanagan, E., & Hepburn, S. (2009). Cognitive-behavioral group treatment for anxiety symptoms in children with high-functioning autism spectrum disorders, a pilot study. *Focus on Autism and Other Developmental Disabilities*, 24, 27–37.
- Silverman, W. K., & Albano, A. M. (1996). *The anxiety disorders interview schedule for DSM-IV-child and parent versions*. San Antonio: Graywind.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(8), 921–929.
- Sofronoff, K., Attwood, T., & Hinton, S. (2005). A randomised controlled trial of CBT intervention

for anxiety in children with Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 46, 1152–1160.

- Sukhodolsky, D. G., Scahill, L., Gadow, K. D., Arnold, L. E., Aman, M. G., & McDougle, C. J. (2008). Parent-rated anxiety symptoms in children with pervasive developmental disorders: Frequency and association with core autism symptoms and cognitive functioning. *Journal of Abnormal Child Psychology*, 36(1), 117–128.
- Sze, K. M., & Wood, J. J. (2007). Cognitive behavioral treatment of comorbid anxiety disorders and social difficulties in children with high-functioning autism: A case report. *Journal of Contemporary Psychotherapy*, 37, 133–144.
- Sze, K. M., & Wood, J. J. (2008). Enhancing CBT for the treatment of autism spectrum disorders and concurrent anxiety: a case study. *Behavioural and Cognitive Psychotherapy*, 36, 403–409.
- White, S. W., Ollendick, T., Scahill, L., Oswald, D., & Albano, A. M. (2009). Preliminary efficacy of a cognitive-behavioral treatment program for anxious youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(12), 1652–1662.
- Wood, J. J. (2006). Parental intrusiveness and children's separation anxiety in a clinical sample. *Child Psychiatry and Human Development*, 37, 73–87.
- Wood, J. J., & Gadow, K. (2010). Exploring the nature and function of anxiety in youth with autism spectrum disorders. *Clinical Psychology: Science and Practice*, 17, 281–292.
- Wood, J. J., Piacentini, J. C., Bergman, R. L., McCracken, J., & Barrios, V. (2002). Concurrent validity of the anxiety disorders section of the anxiety disorders interview schedule for DSM-IV. Child and parent versions. *Journal of Clinical Child and Adolescent Psychology*, 31, 335–342.
- Wood, J. J., Drahota, A., Sze, K., Har, K., Chiu, A., & Langer, D. A. (2009a). Cognitive-behavioral therapy for anxiety in children with autism spectrum disorders: A randomized, controlled trial. *Journal of Child Psychology and Psychiatry*, 50, 224–223.
- Wood, J. J., Drahota, A., Sze, K., Van Dyke, M., Decker, K., Fujii, C., et al. (2009b). Brief report: Effects of cognitive behavioral therapy on parent-reported autism symptoms in school-age children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 39(11), 1608–1612.

Sequential Processing

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Synonyms

[Analytic processing](#); [Serial processing](#); [Successful integration](#); [Successive processing](#)

Definition

Sequential processing is the mental ability to take in, store, and process information in a serial and temporal manner. It is the ability to arrange separate elements into successive steps, when each step is an indicator for later steps. These elements are linked together in a chain-like way. For example, the ability to recall a series of digits, such as a telephone number, or the flow of words in speech, is dependent upon sequential processing (Angus 1985). Sequential processing refers to the manipulation of stimuli one at a time or feature by feature (i.e., performing a series of movements according to instructions, repeating numbers in a specific order) as opposed to simultaneous processing that emphasizes integrated input formed in holistic units (i.e., face processing tasks, analogies). Sequential processing enables the individual to start and complete a single task, in contrast to parallel, simultaneous processing or multitasking in which multiple information is integrated simultaneously. Inhibition of sequential processing is noticeable in responses to tasks in which participants are requested to locate the presence of a target figure embedded within a complex background (i.e., the Embedded Figures Test: Witkin 1971) in which, for most people, the observation of a familiar global pattern occurs faster than the observation of the component parts and therefore inhibits the perception of the component parts (Gerrard and Rugg 2009).

Individuals with autism are known for their uneven cognitive profiles (Happé 1994).

SEQ

► [Sensory Experiences Questionnaire](#)

A common profile is that of verbal, abstract reasoning, and sequential processing impairments alongside little visual–spatial or organizational deficits (Tonn and Obrzut 2005). A different typical cognitive profile in ASD is that of weak central coherence, in which individuals with ASD attend to specific local details at the expense of global features (Happe and Frith 2006), thereby showing a simultaneous processing deficit.

A disorder in sequential processing may impact various areas of functioning (i.e., tasks that require the concept of first, next, last). Individuals who have a sequential processing deficit may exhibit difficulty with word decoding skills and phonetics, grammar, breaking down arithmetic problems into their component parts, following a sequence of steps or rules, and following oral instructions (Powell et al. 1997).

The sequential processing scale of the Kaufman Assessment Battery for Children (K-ABC; Kaufman and Kaufman 1983) is a measure of problem-solving skills that emphasizes sequence and the processing of information in a serial order. The simultaneous processing scale measures problem-solving skills that involve the using of several processes at the same time.

See Also

- ▶ [Embedded Figures Test \(EFT\)](#)
- ▶ [Kaufman Assessment Battery for Children, Second Edition](#)

References and Reading

- Angus, J. (1985). The Luria model of information processing. *Australian Journal of Educational Technology*, 1(1), 59–67. <http://www.ascilite.org.au/ajet/ajet1/angus.html>.
- Gerrard, S., & Rugg, G. (2009). Sensory impairments and autism: A re-examination of causal modelling. *Journal of Autism and Developmental Disorders*, 39, 1449–1463. <https://doi.org/10.1007/s10803-009-0773-9>.
- Happe, F. (1994). Wechsler IQ profile and theory of mind in autism: A research note. *Journal of Child Psychology and Psychiatry*, 35, 1461–1471.
- Happe, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36(1), 5–25. <https://doi.org/10.1007/s10803-005-0039-0>.
- Kaufman, A. S., & Kaufman, N. L. (1983). *Kaufman assessment battery for children: Interpretive manual*. Circle Pines: American Guidance Service.
- Powell, L., Houghton, S., & Douglas, G. (1997). Comparison of etiology specific cognitive functioning profiles for individuals with fragile X and individuals with Down syndrome. *The Journal of Special Education*, 31, 362–376.
- Tonn, R. T., & Obrzut, J. E. (2005). The neuropsychological perspective on autism. *Journal of Developmental and Physical Disabilities*, 17, 409–419. <https://doi.org/10.1007/s10882-005-6623-6>.
- Witkin, H. A. (1971). *Manual for the embedded figures tests*. Palo Alto: Consulting Psychologists Press.

Serax

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Synonyms

[Oxazepam](#)

Definition

Oxazepam is a benzodiazepine. The mechanism of action is complicated, but it has been shown that the benzodiazepines bind to specific GABA receptors in the brain and enhance a GABA function. GABA is a primary inhibitory neurotransmitter in the brain. The effect of the benzodiazepines is to promote the influx of chloride ions in specific brain areas. This is the mechanism by which it enhances the inhibitory effects of GABA. Oxazepam is used primarily to treat anxiety disorders, but may also be used as a sleep medication.

See Also

- [Benzodiazepines](#)

References and Reading

Seahill, L. (2009). Antipsychotic medications. In M. K. Dulcan (Ed.), *Dulcan's textbook of child and adolescent psychiatry* (pp. 775–778). Arlington: American Psychiatric Publishing.

Serentil

- [Mesoridazine](#)

Serial Position Effect

- [Primacy Effects](#)

Serial Processing

- [Sequential Processing](#)

Seromycin

- [D-Cycloserine: Definition](#)

Seroplex

- [Escitalopram](#)

Seroquel

- [Quetiapine](#)

Seroquel XR

- [Quetiapine](#)

Serotonin

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Synonyms

5-HT; 5-Hydroxytryptamine

Definition

Serotonin is a central and peripheral neurotransmitter and neuromodulator that also acts as a growth factor in the central nervous system during development. Serotonin is produced from the essential amino acid tryptophan after hydroxylation and decarboxylation and is degraded to 5-hydroxyindoleacetic acid (5-HIAA).

Elevated levels of platelet serotonin have been consistently reported in autism; however, the cause of the elevation has remained elusive. It is also not clear whether brain serotonin production or function is altered in autism. However, alterations in type-2 serotonin receptors in brain and platelets have been reported.

See Also

- [CSF 5-HIAA](#)
- [Neurotransmitter](#)

References and Reading

Anderson, G. M., Horne, W. C., Chatterjee, D., & Cohen, D. J. (1990). The hyperserotonemia of autism. *Annals of the New York Academy of Sciences*, 600, 331–342.

- Cook, E. H. (1990). Autism: Review of neurochemical investigation. *Synapse*, 6(3), 292–308.
- Goldberg, J., Anderson, G. M., Zwaigenbaum, L., Hall, G. B., Nahmias, C., Thompson, A., et al. (2009). Cortical serotonin type-2 receptor density in parents of children with autism spectrum disorders. *Journal of Autism & Developmental Disorders*, 39(1), 97–104.
- Lam, K. S., Aman, M. G., & Arnold, L. E. (2006). Neurochemical correlates of autistic disorder: A review of the literature. *Research in Developmental Disabilities*, 27(3), 254–289.
- Mulder, E. J., Anderson, G. M., Kema, I. P., de Bildt, A., van Lang, N. D., den Boer, J. A., et al. (2004). Platelet serotonin levels in pervasive developmental disorders and mental retardation: Diagnostic group differences, within-group distribution, and behavioral correlates. *Journal of the American Academy of Child & Adolescent Psychiatry*, 43(4), 491–499.

although the others are an active ingredient in over-the-counter medications, drugs for which marketing has been discontinued, compounds that have not been marketed, compounds in herbs, and cocaine, whose mechanism of action consists of or includes the inhibition of the reuptake of serotonin (5-HT), most of which are primarily used for the treatment of major depressive disorder (MDD) in the United States, although some are also marketed for the treatment of these other conditions: obsessive-compulsive disorder (OCD), fibromyalgia (FM), generalized anxiety disorder (GAD), social anxiety disorder, diabetic peripheral neuropathic pain (DPNP), and chronic musculoskeletal pain.

Serotonin Behavioral Syndrome

► Serotonin Syndrome

Serotonin Reuptake Inhibitors (SRIs)

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Definition

Serotonin reuptake inhibitors are a class of compounds, most of which are prescription drugs,

Historical Background

The first psychiatric drugs whose mechanisms of action included the reuptake of 5-HT are most of the tricyclic and tetracyclic antidepressants (TCAs). The TCAs are doxepin, first marketed by Pfizer in 1969 as Sinequan; trimipramine, first marketed by Odyssey Pharmaceuticals in 1979 as Surmontil; amoxapine, first marketed by Lederle in 1980 as Asenden; maprotiline, first marketed by Novartis in 1980 as Ludiomil; clomipramine, first marketed by Tyco Healthcare in 1989 as Anafranil; imipramine, first marketed by Novartis in 1959 as Tofranil; amitriptyline, first marketed by AstraZeneca in 1961 as Elavil; nortriptyline, first marketed by Eli Lilly in 1964 as Aventyl Hydrochloride; protriptyline, first marketed by Odyssey Pharmaceuticals in 1967 as Vivactil; and desipramine, first marketed by Sanofi-Aventis U.S. in 1964 as Norpramin.

The development of this group of prescription drugs in the family of classical antidepressants began around the time tricyclic compounds were being developed for the treatment of schizophrenia. Consequently, these drugs were first tested for efficacy in the treatment of schizophrenia, although they were instead found to act as antidepressants in the patients in whom they were studied. The study of the pharmacology of the TCAs led to the development of the selective serotonin reuptake inhibitors (SSRIs), a family of

antidepressants that are generally clinically favorable to the TCAs due to having significantly fewer side effects.

The first SSRI developed was zimelidine; however, its production was discontinued due to its toxicity. The subsequent SSRIs to be developed have fewer side effects than the TCAs while showing similarities in the percent of patients who respond to SSRIs as compared to the TCAs. The six SSRIs that were developed after zimelidine are fluoxetine, sertraline, paroxetine, fluvoxamine, citalopram, and escitalopram. Fluoxetine was first marketed by Lilly in 1987 as brand-name drugs Prozac and Sarafem. Sertraline was first marketed by Pfizer in 1991 as Zoloft. Paroxetine was first marketed by GlaxoSmithKline in 1992 as Paxil. Fluvoxamine was first marketed by Solvay in 1994 as Luvox. Citalopram was first marketed by Forest Labs in 1998 as Celexa. Escitalopram was first marketed by Forest Labs in 2002 as Lexapro. Most of these drugs are marketed for the treatment of MDD among other conditions, although fluvoxamine is only marketed for the treatment of OCD in the United States and Europe.

Another family of SRIs developed beginning in the 1990s is the selective serotonin-norepinephrine reuptake inhibitors (SNRIs). The four SNRIs marketed in the United States are venlafaxine, first marketed by Wyeth Pharmaceuticals, Inc., in 1993 as Effexor; duloxetine, first marketed by Eli Lilly in 2004 as Cymbalta; desvenlafaxine, first marketed by Wyeth Pharmaceuticals, Inc., in 2008 as Pristiq; and milnacipran was first marketed by Cypress Bioscience in 2009 as Savella. Duloxetine and milnacipran are not yet available as generic drugs. Milnacipran is marketed in Japan as a treatment for MDD, although it is only FDA-approved for use in the treatment of FM in the United States.

Research on the pathophysiology of depression has led to the development of SRIs outside of these three families such as the serotonin-, norepinephrine-, dopamine-reuptake inhibitors (SNDRI) which inhibit the reuptake of 5-HT, norepinephrine (NE), and dopamine (DA). Although the SNDRI are not currently marketed in the United States, other SRIs that are not of the families of TCAs, SSRIs, or SNRIs such as ziprasidone, trazodone, and dextromethorphan are. One SRI that was used

for obesity management and weight loss, sibutramine with the brand name Meridia, was marketed by Abbott but has been discontinued.

Current Knowledge

Three families of drugs that act as SRIs are the SSRIs, the SNRIs, and the TCAs. Most of these SRIs are generally used to treat MDD, although some of these drugs have other indications such as OCD and other anxiety disorders. Trazodone, with the brand name Olepto, and nefazodone, formerly with the brand name Serzone, are SRIs with 5-HT_{2A} antagonist activity marketed in the United States for the treatment of depression. Other compounds that act as SRIs with 5-HT_{2A} antagonist activity have been developed but have not been marketed in the United States. Cyclobenzaprine, with the brand name Flexeril, is an SRI marketed in the United States as a short-term, adjunctive treatment to physical therapy and rest for muscle spasms associated with acute, painful musculoskeletal conditions. In addition, the opioids pethidine, tramadol, methadone, dextromethorphan, and propoxyphene may have a small amount of SRI activity. The active ingredient in over-the-counter medications dextromethorphan exhibits SRI activity as well as the compounds hyperforin and adhyperforin in St. John's wort and either mesembrine or its metabolite Δ^7 mesembrenone in *Scelletium tortuosum*. A relatively new group of compounds called SNDRI are reuptake inhibitors of 5-HT, NE, and DA; however, these compounds are still in relatively early stages of drug development. Additionally, compounds that act as SRIs and 5-HT_{2C} antagonists have been developed but are not currently marketed in the United States. Other variations of compounds with SRI activity have been reported in the patent literature but require further testing to determine their viability as medications.

The SSRIs are the only SRIs which affect 5-HT receptors without also affecting NE. The six SSRIs that are currently marketed in the United States are fluoxetine, sertraline, paroxetine, fluvoxamine, citalopram, and escitalopram. Fluoxetine, with the brand names Prozac and Sarafem, is marketed for the treatment of MDD and OCD. Sertraline, with the brand name Zoloft, is marketed for the

treatment of MDD, OCD, panic disorder, post-traumatic stress disorder (PTSD), premenstrual dysphoric disorder (PMDD), and social anxiety disorder. Paroxetine, with the brand names Paxil, Paxil CR, and Pexeva, is marketed for the treatment of MDD, OCD, panic disorder, social anxiety disorder, GAD, and PTSD. Fluvoxamine, with the brand names Luvox and Luvox CR, is marketed for the treatment of OCD in the United States and is marketed for the treatment of MDD in other countries. Citalopram, with the brand name Celexa, is marketed for the treatment of MDD. Escitalopram, with the brand name Lexapro, is marketed for the treatment of MDD and GAD. SoRI-6238 is another compound known to be an SSRI.

The SNRIs selectively inhibit the reuptake of both 5-HT and NE. These drugs are similar in clinical use to the SSRIs but have the additional effects of treating pain and physical symptoms, such as those of FM. Milnacipran, with the brand name Savella, is marketed just for the treatment of FM in the United States, although it is marketed for the treatment of MDD in Japan. Duloxetine, with the brand name Cymbalta, is marketed for the treatment of MDD, GAD, DPNP, FM, and chronic musculoskeletal pain. Venlafaxine, with the brand names Effexor and Effexor XR, is marketed for the treatment of MDD, GAD, social anxiety disorder, and panic disorder. Desvenlafaxine, with the brand name Pristiq, is marketed for the treatment of MDD. Other SNRI compounds have been developed but have not been marketed in the United States.

The TCAs are a family of SRIs which affect both 5-HT and NE as well as acting as anticholinergic or antimuscarinic agents, alpha-adrenergic antagonists, and antihistamines. Although these drugs seem to have similar efficacy to the SSRIs and SNRIs and may be more effective than those drugs, the TCAs are not as well tolerated and have more side effects than the SSRIs and the SNRIs. Clinically, the TCAs are rarely used due to their side effects. Although these compounds are named for their chemical rings, their side chains are believed to be more important to their functions. The ten TCAs currently marketed in the United States are clomipramine, doxepin, trimipramine, amoxapine, maprotiline, imipramine, amitriptyline, nortriptyline, protriptyline, and desipramine. Different compounds have different

degrees of 5-HT reuptake inhibition; for example, clomipramine exhibits a relatively high amount of SRI activity compared to other TCAs.

Clomipramine, with the brand name Anafranil, is marketed for the treatment of OCD. Doxepin, marketed under the brand name Sinequan, is labeled for use as a treatment for psychoneurotic individuals, alcoholic individuals, individuals with an organic disease with comorbid depression, anxiety, or both; and for individuals with psychotic depressive disorders with anxiety. A formulation of doxepin is also marketed with the brand name Silenor to treat insomnia, and a cream with doxepin hydrochloride as its active ingredient marketed with the brand name Zonalon is marketed for the short-term treatment of pruritus in adults with atopic dermatitis or lichen simplex chronicus. Trimipramine, with the brand name Surmontil; amoxapine, formerly with the brand name Asenden; maprotiline, with the brand name Ludiomil; imipramine, with the brand name Tofranil; a formulation combining amitriptyline hydrochloride with perphenazine, with the brand names Triavil 2–10, Triavil 2–25, Triavil 4–10, Triavil 4–25, and Triavil 4–50; nortriptyline, with the brand name Pamelor; protriptyline, with the brand name Vivactil; and desipramine, with the brand name Norpramin, are marketed for the treatment of depression. Additionally, a formulation combining amitriptyline hydrochloride with chlordiazepoxide, with the brand name Limbitrol, is marketed as a treatment for depression associated with anxiety.

Two important safety issues must be noted with the use of SRIs. The concomitant use of SRIs and monoamine oxidase inhibitors (MAOIs) is a known cause of serotonin syndrome which is potentially lethal. Also, the use of antidepressants in children, adolescents, and young adults may increase depressive symptoms or cause the onset of suicidal ideation; therefore, the appropriate discretion must be used when prescribing SRIs in children or adolescents with depression.

Future Directions

In recent years, the development of new, more efficacious antidepressants has been attempted by combining the effects of SRIs with other

pharmacological effects. The SNRIs are an example of a group of drugs whose development has begun relatively recently which combine SRI effects with NE reuptake inhibition in order to enhance antidepressant effects compared to SSRIs. Since dopamine is thought to be implicated in depression as well, research and development has begun for SNDRIs, compounds with SRI effects, NE reuptake inhibition, and DA reuptake inhibition. At least five SNDRI compounds have been developed. Other compounds being developed and investigated are SRIs and 5-HT_{2A} antagonists; at least 27 of these compounds have been developed to date. The treatment of depression with a combination of SRIs with atypical antipsychotics is another approach being investigated. Other compounds with SRI activity have reportedly been developed, but these compounds require further testing to determine their viability as medications.

See Also

- ▶ [Selective Serotonin Reuptake Inhibitors \(SSRIs\)](#)
- ▶ [Tricyclic Antidepressants \(TCAs\)](#)

References and Reading

- Andreasen, N. C., & Black, D. W. (2001). *Introductory textbook of psychiatry* (3rd ed.). Washington, DC: American Psychiatric Publishing.
- Gillman, P. K. (2005). Monoamine oxidase inhibitors, opioid analgesics and serotonin toxicity. *British Journal of Anaesthesia*, 95, 434–441.
- Greden, J. F. (2006). Duloxetine and milnacipran. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 171–180). Washington, DC: American Psychiatric Publishing.
- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001). Chapter 7: Treatment with antidepressants. In *Principles and practice of psychopharmacotherapy* (3rd ed., pp. 215–325). Philadelphia: Lippincott Williams & Wilkins.
- Moltzen, E. K., & Bang-Andersen, B. (2006). Serotonin reuptake inhibitors: The corner stone in treatment of depression for half a century—a medicinal chemistry survey. *Current Topics in Medicinal Chemistry*, 6, 1801–1823.
- Müller, W. E. (2003). Current St. John's wort research from mode of action to clinical efficacy. *Pharmacological Research*, 47, 101–109.
- Nelson, J. C. (2006). Tricyclic and tetracyclic drugs. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of*

- clinical psychopharmacology* (2nd ed., pp. 5–29). Washington, DC: American Psychiatric Publishing.
- Sadock, B. J., & Sadock, V. A. (2003). *Kaplan & Sadock's synopsis of psychiatry: Behavioral sciences/clinical psychiatry*. Philadelphia: Lippincott Williams & Wilkins.
- Sadock, B. J., & Sadock, V. A. (2005). *Kaplan & Sadock's pocket handbook of clinical psychiatry*. Philadelphia: Lippincott Williams & Wilkins.
- Smith, C. (2011). The effects of *Sceletium tortuosum* in an in vivo model of psychological stress. *Journal of Ethnopharmacology*, 133, 31–36.
- Stahl, S. M. (2000). Classical antidepressants, serotonin selective reuptake inhibitors, and noradrenergic reuptake inhibitors. In *Essential psychopharmacology: Neuroscientific basis and clinical applications* (pp. 199–243). Cambridge, MA: Cambridge University Press.
- U.S. Food and Drug Administration. (2011). *Drugs@FDA*. Retrieved from <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm>

Serotonin Syndrome

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Synonyms

[Hyperactivity syndrome](#); [Serotonin behavioral syndrome](#)

Short Description or Definition

Serotonin syndrome is a rare but life-threatening side effect of serotonin (5-HT)-enhancing substances in which overactivation of 5-HT receptors causes various physiological symptoms including diarrhea, agitation, autonomic instability, and cardiovascular collapse.

Categorization

It is a syndrome consisting of mental, autonomic, and neurological disorders.

Epidemiology

Serotonin syndrome is rare, and some reported cases of neuroleptic malignant syndrome (NMS) may actually have been serotonin syndrome since these two syndromes present with similar symptoms and physicians are less familiar with serotonin syndrome than NMS. Although little is known about the prevalence of serotonin syndrome, one retrospective study showed that 4 of the 17 patients using lithium and paroxetine concomitantly developed the syndrome.

This syndrome is more common in individuals taking selective serotonin reuptake inhibitors (SSRIs) than in the general population. Risk factors include dehydration, agitation, and organic cerebral disorders. Onset occurs within 24 h of the administration of the causal agent or agents for all ages and both sexes.

Natural History, Prognostic Factors, and Outcomes

Serotonin syndrome is thought to be caused by the overstimulation of 5-HT_{1A} receptors and potentially involves 5-HT₂ agonists, catecholamines, dopamine (DA), and/or tryptamine as well. Serotonin syndrome is caused by overdose of 5-HT precursors or agonists or the concomitant use of at least two compounds from these groups: serotonin precursors or agonists, serotonin-release stimulators, serotonin reuptake inhibitors (SRIs), and compounds that inhibit 5-HT metabolism in a nonspecific manner including monoamine oxidase inhibitors (MAOIs) and cocaine. Although the current theory regarding the pathophysiology of serotonin syndrome suggests that MAOI-B agents would not precipitate or contribute to serotonin syndrome, evidence suggests that any of the clinically available MAOIs can cause serotonin syndrome. Venlafaxine, trazodone, nefazodone, MAOIs, lithium, tryptophan, meperidine, sumatriptan, buspirone, amphetamines, and

SSRIs have been reported to have caused serotonin syndrome. Serotonin syndrome may also result from the concomitant use of an SRI and methylene blue. Onset of symptoms is abrupt and occurs less than 24 h after the causal agent enters the body. When in mild to moderately severe form, serotonin syndrome usually resolves within 24–72 h although symptoms may last weeks without progression and end when the serotonergic agents are removed. This syndrome may result in death.

Clinical Expression and Pathophysiology

The pathophysiology of serotonin syndrome is not well understood, but excessive activation of serotonin receptors, mainly the 5-HT_{1A} receptors, is thought by many to occur in serotonin syndrome. Other agents which may be involved in the pathophysiology of serotonin syndrome include 5-HT₂ agonists, catecholamines, dopamine (DA), and tryptamine. In serotonin syndrome, the following symptoms are experienced in the order listed: diarrhea; restlessness; agitation, hyperreflexia, autonomic instability; myoclonus, seizures, hyperthermia, shivering, rigidity; delirium, coma, status epilepticus; cardiovascular collapse; and death.

The main symptoms of serotonin syndrome are altered consciousness, autonomic instability, and neuromuscular abnormalities. Observed symptoms include agitation, tremor, myoclonus, hyperreflexia, tremulousness, dysarthria, incoordination, headache, abdominal cramping, bloating, diarrhea, sweating, flushing, delirium, hypomanic symptoms, racing thoughts, pressure of speech, elevated/dysphoric mood, confusion, disorientation, lethargy, diaphoresis, fever, hyperthermia, elevated blood pressure, tachycardia, hypotension, hypertension, and cardiovascular collapse resulting in death.

Evaluation and Differential Diagnosis

Since no single diagnostic test exists for serotonin syndrome, multiple approaches to the differential diagnosis of this syndrome exist. The three components of a differential diagnosis of serotonin syndrome are a history of the use of a serotonergic agent, the presence of signs or symptoms associated

with serotonin syndrome, and the absence of other similar conditions such as NMS. Physicians may consult the Radomski criteria for diagnostic criteria of serotonin syndrome (Radomski et al. 2000).

Both certain diseases and poisonings should be ruled out for a differential diagnosis of serotonin syndrome. The conditions that should be ruled out are catatonia, severe dystonic reaction, encephalitis, hyperthyroidism, malignant hyperthermia, meningitis, NMS, septicemia, stiff man syndrome, herpetic encephalopathy, myocardial necrosis, delirium tremens, and tetanus. The poisonings that should be ruled out are poisonings with adrenergics, anticholinergics, amphetamines, cocaine, 2,4-dichlorophenoxyacetic acid, dinitrophenol, lithium, lysergic acid diethylamide (LSD), MAOIs, pentachlorophenol, phencyclidine (PCP), salicylates, strychnine, and water hemlock. Ruling out NMS is important since it presents in a manner similar to serotonin syndrome. Laboratory tests are indistinguishable between these two syndromes. A drug history including prolonged exposure to neuroleptic agents or discontinuation of dopamine (DA) agonists, lead pipe rigidity, and absence of mydriasis would indicate a diagnosis of NMS as opposed to that of serotonin syndrome.

Treatment

Prevention of serotonin syndrome is attempted by avoiding the concomitant use of SRIs and MAOIs and allowing time between discontinuation of one of these types of drugs and the initiation of treatment with a drug of the other type. At least 2 weeks must be allowed before beginning an MAOI after discontinuing most SSRIs, although at least 5 weeks must be allowed before beginning an MAOI after discontinuing fluoxetine. At least a few days must be allowed before beginning and MAOI after discontinuing a tricyclic antidepressant (TCA).

Treatment consists of the discontinuation of offending agent and the use of intensive care measures as needed while waiting for the syndrome to resolve. Benzodiazepines, cyproheptadine, chlorpromazine, methysergide, propranolol, nitroglycerine, dantrolene, anticonvulsants, mechanical ventilation, paralyzing agents, and cooling blankets

may be utilized to aid in treatment. Supportive care is sufficient in all but severe cases. In severe cases, therapeutic agents can be used, although none of them have shown clear and consistent efficacy for the treatment of serotonin syndrome.

See Also

- ▶ Antidepressant Medications
- ▶ Serotonin Reuptake Inhibitors (SRIs)

References and Reading

- Andreasen, N. C., & Black, D. W. (2001). "Serotonin reuptake inhibitors." Somatic treatments. In *Introductory textbook of psychiatry* (3rd ed., pp. 727–730). Washington, DC: American Psychiatric Publishing.
- Birmes, P., Coppin, D., Schmitt, L., & Lauque, D. (2003). Serotonin syndrome: A brief review. *Canadian Medical Association Journal*, 168, 1439–1442.
- Gillman, P. K. (1998). Serotonin syndrome: History and risk. *Fundamental & Clinical Pharmacology*, 12, 482–491.
- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001). "Serotonin syndrome." Chapter 7: Treatment with antidepressants. In *Principles and practice of psychopharmacotherapy* (3rd ed., pp. 299–300). Philadelphia: Lippincott Williams & Wilkins.
- Lane, R., & Baldwin, D. (1997). Selective serotonin reuptake inhibitor-induced serotonin syndrome: Review. *Journal of Clinical Psychopharmacology*, 17, 208–221.
- Martin, T. G. (1996). Serotonin syndrome. *Annals of Emergency Medicine*, 28, 520–526.
- Ng, B. K. W., & Cameron, A. J. D. (2008). The role of methylene blue in serotonin syndrome: A systematic review. *Psychosomatics*, 51, 194–200.
- Radomski, J. W., Dursun, S. M., Reveley, M. A., & Kutcher, S. P. (2000). An exploratory approach to the serotonin syndrome: An update of clinical phenomenology and revised diagnostic criteria. *Medical Hypotheses*, 55, 218–224.
- Sadock, B. J., & Sadock, V. A. (2005). "Serotonin syndrome." Psychopharmacology and other biological therapies. In *Kaplan & Sadock's pocket handbook of clinical psychiatry* (p. 419). Philadelphia, PA: Lippincott Williams & Wilkins.
- Shim, J. J., & Yonkers, K. A. (2006). "Psychoactive drug side effects." Psychiatric emergencies. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 720–722). Washington, DC: American Psychiatric Publishing.
- Sun-Edelstein, C., Tepper, S. J., & Shapiro, R. E. (2008). Drug-induced serotonin syndrome: A review. *Expert Opinion on Drug Safety*, 7, 587–596.
- U.S. Food and Drug Administration. (2011). *Information for Healthcare Professionals: Selective Serotonin Reuptake Inhibitors (SSRIs), Selective Serotonin-*

Norepinephrine Reuptake Inhibitors (SNRIs), 5-Hydroxytryptamine Receptor Agonists (Triptans). Retrieved from <http://www.fda.gov/Drugs/DrugSafety/PostmarketDrugSafetyInformationforPatientsandProviders/DrugSafetyInformationforHealthcareProfessionals/ucm085845.htm>.

Sertraline

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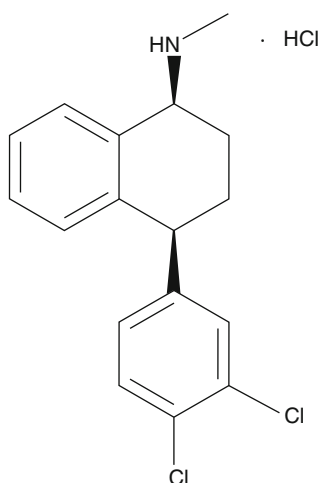
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Synonyms

Sertraline hydrochloride; Zoloft

Definition



Sertraline is a prescription drug in the group of selective serotonin reuptake inhibitors (SSRIs) in

the family of antidepressant medications whose active ingredient sertraline hydrochloride has the chemical formula $C_{17}H_{17}NCl_2 \cdot HCl$. This drug was initially FDA-approved for medical use in the year 1991. This drug is FDA-approved for the treatment of major depressive disorder, obsessive-compulsive disorder (OCD), panic disorder with or without agoraphobia, posttraumatic stress disorder (PTSD), premenstrual dysphoric disorder (PMDD), and social anxiety disorder. Risks include onset of suicidal ideation and increased depression in major depressive disorder. Observed side effects include nausea, diarrhea/loose stools, insomnia, somnolence, headache, dry mouth, ejaculation failure, and decreased libido.

See Also

► [Antidepressant Medications](#)

References and Reading

- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001). *Principles and practice of psychopharmacotherapy* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
- Shim, J. J., & Yonkers, K. A. (2006). Sertraline. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 47–57). Washington, DC: American Psychiatric Publishing.
- Stahl, S. M. (2000). *Essential psychopharmacology: Neuroscientific basis and clinical applications*. Cambridge, MA: Cambridge University Press.
- U.S. Food and Drug Administration. (2011). *Zoloft*. Retrieved from: <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm?fuseaction=Search.Overview&DrugName=ZOLOFT&CFID=58140906&CFTOKEN=dca5c88fa35d547e-32692DDE-BA03-1665-9B13C8D0AAD8102F>.

Sertraline Hydrochloride

► [Sertraline](#)

Service

► Employment

Service Delivery Model

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Definition

Service delivery model refers to a set of practices, organized around a conceptual framework, that promote the development, learning, or welfare of individuals with autism spectrum disorders (ASD) and their families (i.e., this entry will not address health services). “Model” implies that a clearly articulated organization of practices exist; “service” indicates that the treatment, intervention, or therapy practices that individuals with autism receive are specified, and “delivery” specifies that there is a method for implementing the service(s) and documenting the quantity and/or quality of the implementation. This definition may be construed broadly or in more discrete terms.

When construed broadly, service delivery model may refer to provisions of service specified by a governmental agency, insurance company, or other human service agency. For example, the Individuals with Disabilities Improvement Education Act (IDEIA) funds services to students with disabilities, including ASD, enrolled in public school programs and infants and toddler with disabilities enrolled in early intervention programs (<http://idea.ed.gov/>). Medicaid agencies within states create waivers that provide funds for service for Individuals with ASD (<http://www.cga.ct.gov/2007/rpt/2007-R-0319.htm>). Similarly, in some states, insurance companies fund services for individuals with ASD, such as speech and language therapy or early intensive behavioral intervention (Young et al. 2009).

Format for Service Delivery

Intervention and treatment services for children and youth with ASD may be delivered in different formats. *Clinic-based services* occur, as the name suggests, in a clinical setting, and often, a trained therapist delivers the service to the child or youth with ASD. For example, a speech pathologist may provide speech. At times, the therapists will also work with parents to show or teach them ways of working with their child at home. An example of a clinic-based service is when a child with ASD receives discrete trial training at a treatment center, as happens for part of the Lovaas Institute model (<http://www.lovaas.com/about.php>), or a child and family attend clinic-based sessions for developmental therapy as occurs with the DIR model (<http://www.icdl.com/dirFloortime/overview/GuidelinesforComprehensiveApproach.shtml>).

Services may also be delivered in the *home or community* setting. In such service delivery models, a therapist or home visitor may travel to the child’s home and work directly with the child, work with the parent to teach her or him to deliver the intervention, or a combination of the two. The content and purpose of these programs vary considerably. For example, Devlin and Harber (2004) had described home-based programs in which the parent delivers discrete trial training intervention for their children with ASD. In contrast, other home-based service models consist of home visitors who support families in promoting communication during natural routines and play (Wetherby and Woods 2006).

A third type of service delivery model is through the *school-based* programs. In this model, children with ASD attend and participate in classes that may be specially designed for students with ASD or that exist in inclusive general education settings. In these service delivery models, the class is led by a teacher and other professionals provide related service at the school. These are the types of services prescribed by the public schools and supported by IDEIA, mentioned previously. A variation on this public school service model occurs in high school, when adolescents with ASD are involved in

community independence and vocational training (Bambera et al. 2007).

A fourth type of service delivery model occurs for adults with ASD during the post-school years. The needs of adults with ASD vary and so do the services. In some communities, agencies provide support and options for residence outside of the family's home, and options may range from group homes to independent community residence with support (Stancliffe et al. 2004). In addition, a goal for many adults with ASD is supported or independent employment, and agencies may provide vocational skills training and supported employment in the community (Mank 2007).

In addition, often there are combinations of the service delivery model just noted. For example, Project DATA (Schwartz et al. 2004) is an early intervention program for children with ASD that operates in a classroom setting, but in which children with ASD also received individual discrete trial training and a home-based component in which staff visit the home to work with the parents on a regular, frequent basis.

Comprehensive Treatment Programs as Service Delivery Models

The more discrete definition of service delivery models refers to intervention or treatment programs that include specifically articulated practices, procedures for assessing implementation, evidence of replication (or use in more than one site), and often evidence of efficacy (Odom et al. 2010). These programs have been also called "comprehensive treatment models" (Odom et al. 2007) or "brand-name" programs (National Research Council 2001).

Historical Background

The initial mode of treatment services for children with ASD were institutional (Kanner 1971), often having a psychodynamic orientation (Ruttenberg 1971). A controversial program established by Bruno Bettelheim (1967) in the 1950s–1960s was based on removing children from the

purportedly damaging influence of their "refrigerator" mothers, with these services again delivered in a residential setting. Partly in reaction to the psychodynamic approach and also changing theoretical perspectives in the field, other models emerged in the 1960s. The two emerging treatment models of the time were the "Lovaas" model (Lovaas 1971) and the TEACCH (Treatment and Education of Autistic and Related Communication Handicapped Children) model (Mesibov et al. 2004). The Lovaas model, which first was known as the UCLA Young Autism Program (Lovaas 1996) and then the Lovaas Institute model, is based on the application of the principles of applied behavior analysis. Its most prominent feature is the use of discrete trial training, although the current Lovaas Institute model includes a wider range of practices. The TEACCH model follows a psychoeducational and social learning theory theoretical framework, incorporates a "structured teaching" approach (Schopler et al. 1971), and proposed, early on, to incorporate parents as the "therapists" for their own children with ASD (Schopler and Reichler 1971).

From the 1980s to early 2000s, comprehensive treatment models continued to develop and followed a broader array of theoretical and conceptual frameworks. For example, the Pivotal Response Treatment model (Koegel and Koegel 2006) and the Walden model (McGee et al. 1999) followed a more "naturalistic" application of applied behavior analysis. The LEAP (Learning Experiences: An Alternative Program for Preschoolers and their Parents) model (Hoyson et al. 1984) incorporated early childhood education orientation, typically developing children, and the application of applied behavior analysis. The DIR model developed by Greenspan and going by the popular name of Floortime followed a clinical, "relationship-based" process (Greenspan and Weider 2006). The Early Start Denver model, also known just as the Denver model, incorporate both developmental and behavioral principles in a clinical (Dawson et al. 2010) and also school-based format (Rogers et al. 2001).

In the early 2000, the National Academy of Sciences convened a committee to review

educational programs for students with ASD, and they identified models that they called “brand-name” programs because individuals often referred to them by the developer’s name or an acronym established for the program (e.g., “the Lovaas model,” “Walden,” TEACCH, LEAP, etc.). The committee concluded that although these service delivery models/comprehensive treatment programs were identifiable and clearly existed, with the exception of the UCLA Young Autism Project, studies addressing the efficacy of these programs were limited.

Current Knowledge

In the ensuing years, comprehensive treatment models continued to be developed and appeared in the literature. In a set of books edited by Handleman and Harris (2006, 2008), developers of comprehensive treatment models described approaches for preschool and school-age children with ASD and their families. To examine the cohesiveness of comprehensive treatment models for young children with ASD, Odom et al. (2010) searched the literature and identified 30 programs. They classified these programs as applied behavior analysis (ABA)-Clinic or Home (e.g., Lovaas Institute, Pivotal Response Treatment), ABA-Classroom Based (e.g., May Institute, Princeton Child Development Institute), ABA-Inclusive (e.g., LEAP, Walden model), Developmental and Relationship-Based (e.g., Early Start Denver model, SCERTS [Social Communication, Emotional Regulation, and Transaction Support]), and Idiosyncratic (e.g., TEACCH, Miller Method). Odom et al. concluded that as a group, programs were strongest in their specification of practices and content inherent in their models and replications by other sites. They were relatively weak on measurement of implementation and evidence of efficacy.

In a review of the literature that examined the evidence of efficacy of comprehensive treatment models, Rogers and Vismara (2008) found that the Lovaas Institute model has the strongest evidence of efficacy, and the Pivotal Response Treatment

has emerging evidence. Conducting one of the most comprehensive reviews of the research literature, the National Standards Project (2009) found evidence for “established” comprehensive treatment models and focused intervention practices. They identified Comprehensive Behavioral Treatment for Young Children and Pivotal Response Treatment as two comprehensive approaches that met their criteria for “established treatments.” Also, they identified developmental relationship-based treatment as an emerging treatment (i.e., some positive evidence, but not enough to meet the criteria for established treatment). A review issued by the What Works Clearinghouse (http://ies.ed.gov/ncee/wwc/reports/ece_cd/lovaas_model/index.asp), however, found that the Lovaas model had potentially positive effects on cognitive development, but no discernible effects on communication/language, social-emotional, or functional behavior.

Future Directions

The field of treatment service delivery has been pushed by the search for the most effective practices for children and youth with ASD and their families. In the past, efficacy research on comprehensive treatment models and services has been limited by funding opportunities. Efficacy studies are expensive, especially if they are school-based and require random assignment of classrooms or buildings rather than individual children and families. Currently more avenues for funding exist for efficacy and effectiveness studies (i.e., through NIH, HRSA, and the Institute for Education Science), and in the future, there will be more scientific evidence documenting the effects of comprehensive treatment models as well as comparison of the relative effects of different models.

With greater confidence in the efficacy of specific treatment and service models will come the desire to “scale up” the implementation of these models. Implementation science (Fixsen et al. 2005) has made great inroads into examining factors affecting the use of model features and quality of service in a variety of fields of human service

areas (Durlak and DuPre 2008). In the future, not only will the specific practices within models be critical for determining the impact of the services provided, other implementation factors such as professional development and training, coaching, and administrative support will separate the effective from the ineffective delivery of services to individuals with ASD and their families.

In the past, the majority of the research and development related to service delivery models has occurred at the early childhood and elementary levels. With the increasing awareness of ASD across the age span has come the recognition of the need for treatment and service delivery models for middle school, high school, and adulthood (Interagency Autism Coordinating Committee 2011). In their strategic plan, the Interagency Autism Coordinating Committee (2011) has identified high school programs for students with ASD as one critical area of need. Effective comprehensive treatment and service models will undoubtedly emerge in the future.

As in every area of life in the twenty-first century, technology will impact the future of treatment and service delivery for individuals with ASD. At the individual level, treatment models and services will design personal technology (e.g., iTouch, Pads, personal organizers) that fosters independence. For example, iPods have been used to prompt individuals through schedules for their school day (Cihak et al. 2010). For students, this may foster a shift from the interpersonal to the “technopersonal” (i.e., increased communication through electronic media). In professional development, there are already websites that display online courses and modules of evidence-based practices, which will be used in training and can be the basis for treatment and service delivery (National Professional Development Center on ASD 2011; <http://www.npdc.fpg.edu>). Following the principles of telemedicine, the easy use of videotelemetry allows supervision and coaching of professionals and parent located at remote sites (Vismara et al. 2009). Currently, technology is advancing faster than the treatment and service delivery science, and it will lead to great changes in the future.

See Also

- Applied Behavior Analysis (ABA)
- Early Start Denver Model
- Pivotal Response Training
- Supported Employment

References and Reading

- Bambera, L., Wilson, B. A., & McKenzie, M. (2007). Transition and quality of life. In S. Odom, R. Horner, M. Snell, & J. Blacher (Eds.), *Handbook of developmental disabilities* (pp. 371–389). New York: Guilford Press.
- Bettelheim, B. (1967). *The empty fortress: Infantile autism and the birth of the self*. New York: The Free Press.
- Cihak, D., Gahrenkrog, C., Ayres, K. M., & Smith, C. (2010). The use of video modeling via a video iPod and a system of least prompts to improve transitional behaviors for students with autism spectrum disorders in the general education classrooms. *Journal of Positive Behavior Interventions*, 12, 103–115.
- Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., et al. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The Early Start Denver Model. *Pediatrics*, 125(1), 17–23.
- Devlin, S. D., & Harber, M. M. (2004). Collaboration among parents and professionals with discrete trial training in the treatment for autism. *Education and Training in Developmental Disabilities*, 39, 291–300.
- Durlak, J. A., & DuPre, E. P. (2008). Implementation matters: A review of research on the influence of implementation on program outcomes and the factors affecting implementation. *American Journal of Community Psychology*, 41, 327–350.
- Felce, D., Perry, J., Lowe, K., & Jones, E. (2011). The impact of autism or severe challenging behavior on lifestyle outcome in community housing. *Journal of Research in Intellectual Disabilities*, 24, 95–104.
- Fixsen, D. L., Naoom, S. F., Blase, K. A., Friedman, R. M., & Wallace, F. (2005). *Implementation research: A synthesis of the literature* (FMHI Publication #231). Tampa: University of South Florida, Louis de la Parte Florida Mental Health Institute, The National Implementation Network.
- Greenspan, S., & Weider, S. (2006). *Engaging autism: Helping children relate, communicate, and think with the DIR Floortime approach*. New York: De Capo Lifelong Books.
- Handleman, J., & Harris, S. (Eds.). (2001). *Preschool education programs for children with autism* (2nd ed.). Austin: PRO-ED.
- Harris, S., & Handleman, J. (Eds.). (1994). *Preschool education programs for children with autism*. Austin: PRO-ED.

- Hoyson, M., Jamieson, B., & Strain, P. (1984). Individualized group instruction of normally developing and autistic-like children: The LEAP curriculum model. *Journal of the Division for Early Childhood*, 8, 157–172.
- Interagency Autism Coordinating Committee. (2011). *The 2011 Interagency Autism Coordinating Committee strategic plan for Autism Spectrum Disorder Research*. Bethesda: U.S. Department of Health and Human Services. Retrieved July 2, 2011, from <http://www.iacc.hhs.gov/strategic-plan/2011/future.shtml>.
- Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of Autism and Childhood Schizophrenia*, 1, 119–145.
- Koegel, R. L., & Koegel, L. K. (2006). *Pivotal response treatments for autism: Communication, social, & academic development*. Baltimore: Brookes.
- Lovaas, I. (1971). Considerations in the development of a behavioral treatment program for psychotic children. In D. Churchill, G. Alpern, & M. DeMyer (Eds.), *Infantile autism: Proceedings of the Indiana University Colloquium* (pp. 124–144). Springfield: Charles C. Thomas.
- Lovaas, I. (1996). The UCLA young autism model of service delivery. In C. Maurice, G. Green, & S. Luce (Eds.), *Behavioral intervention for young children with autism* (pp. 241–248). Austin: PRO-ED.
- Mank, D. (2007). Employment. In S. Odom, R. Horner, M. Snell, & J. Blacher (Eds.), *Handbook of developmental disabilities* (pp. 390–409). New York: Guilford Press.
- McGee, G., Morrier, M., & Daly, T. (1999). An incidental teaching approach to early intervention for toddlers with autism. *The Journal of the Association for Persons With Severe Handicaps*, 24, 133–146.
- Mesibov, G. B., Shea, V., & Schopler, E. (2004). *The TEACCH approach to autism spectrum disorders*. New York: Springer.
- National Professional Development Center on Autism Spectrum Disorders. (2011). *Autism internet modules*. Chapel Hill: Author. Retrieved July 2, 2011, from <http://www.autismmpdc.fpg.unc.edu/>.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- National Standards Project. (2009). *The National Standards Project's findings and conclusions*. Randolph: Author.
- Odom, S. L., Rogers, S., McDougle, C. J., Hume, K., & McGee, G. (2007). Early intervention for children with autism spectrum disorder. In S. Odom, R. Horner, M. Snell, & J. Blacher (Eds.), *Handbook of developmental disabilities* (pp. 199–223). New York: Guilford Press.
- Odom, S. L., Boyd, B., Hall, L., & Hume, K. (2010). Evaluation of comprehensive treatment models for individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40, 425–436.
- Rogers, S. J., & Vismara, L. A. (2008). Evidence-based comprehensive treatments for early autism. *Journal of Clinical Child and Adolescent Psychology*, 37, 8–38.
- Rogers, S. J., Hall, T., Osaki, D., Reaven, J., & Herbison, J. (2001). The Denver Model: A comprehensive integrated educational approach to young children with autism and their families. In J. Handleman & S. Harris (Eds.), *Preschool education programs for children with autism* (2nd ed., pp. 95–134). Austin: PRO-ED.
- Ruttenberg, B. A. (1971). A psychoanalytic understanding of infantile autism and its treatment. In D. Churchill, G. Alpern, & M. DeMyer (Eds.), *Infantile autism: Proceedings of the Indiana University Colloquium* (pp. 145–184). Springfield: Charles C. Thomas.
- Schopler, E., & Reichler, R. J. (1971). Parents as cotherapists in the treatment of psychotic children. *Journal of Autism and Childhood Schizophrenia*, 1, 87–102.
- Schopler, E., Brehm, S., Kinsbourne, M., & Reichler, R. J. (1971). Effect of treatment structure on development in autistic children. *Archives of General Psychiatry*, 20, 174–181.
- Schwartz, I., Sandall, S., McBride, B., & Boulware, G. (2004). Project DATA (Developmentally Appropriate Treatment for Autism): An inclusive school-based approach to educating young children with autism. *Topics in Early Childhood Special Education*, 23, 156–168.
- Stancliffe, R. J., Emerson, E., & Lakin, K. C. (2004). Residential supports. In E. Emerson, C. Hatton, T. Thompson, & T. Parmenter (Eds.), *The international handbook of applied research in intellectual disabilities* (pp. 459–478). Chichester: Wiley.
- Vismara, L. A., Young, G. S., Stahmer, A. C., Griffith, E. M., & Rogers, S. J. (2009). Dissemination of evidence-based practice: Can we train therapist from a distance. *Journal of Autism and Developmental Disorders*, 39, 1636–1651.
- Wainer, A., & Ingersol, B. (2010). The use of innovative computer technology for teaching social communication to individuals with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5, 96–107.
- Wetherby, A. M., & Woods, J. J. (2006). Early social interaction project for children with autism spectrum disorders beginning in the second year of life: A preliminary study. *Topics in Early Childhood Special Education*, 26, 67–82.
- Young, A., Ruble, L., & McGrew, J. (2009). Public vs. private insurance: Cost, use, accessibility, and outcomes of services for children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 3, 1023–1033.

Service Need

► Service Utilization in Autism

Service Receipt

► [Service Utilization in Autism](#)

Service Use

► [Service Utilization in Autism](#)

Service Utilization in Autism

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Synonyms

[Service need](#); [Service receipt](#); [Service use](#)

Definition

The provision of services to address health, social, and educational needs and goals. Service utilization has been described as a dynamic interaction between consumers and providers that can be operationalized by describing its continuity (e.g., the degree to which it is provided uninterrupted over time as needed), its comprehensiveness (the degree to which it recognized the full range of needs across time), its accessibility (the facilitation or barriers to receipt), and its productivity (the capacity to generate the sufficient volume of activity required to address needs) (Da Silva et al. 2011). Within the context of autism, service utilization can be driven by need and availability and will inevitably cross various sectors (social services, education, health) as a result of the multifaceted nature of the condition. Service utilization also varies in response to

demographic, clinical, and systemic factors over time. Some needs are acute, while others chronic.

There is considerable research on services utilized by children with autism (e.g., diagnostic, behavioral intervention, special education, speech and language therapy, financial supports, and supports for family), with home and education supports varying by school district or parent socioeconomic status. Given the high rate of physical and mental health comorbidities, several studies have documented the high volume of health service utilization specifically. Compared to children without autism, those with autism have higher rates of pediatrician, specialty care (including speech therapy, occupational or social skills therapy, physical therapy, psychotherapy, and neurology), hospitalization, and emergency department (ED) visits within a 1-year period (Cummings et al. 2016). Adults with autism are also more like to visit the family physician, pediatrician, psychiatrist, neurologist, and gastroenterologist and to visit the ED for psychiatric reasons and be hospitalized overall and for psychiatric reasons, compared to peers without developmental disabilities (Weiss et al. 2018). There is some question as to whether high rates of service use are specific to autism though, with recent large-scale studies highlighting higher rates of health use in adolescents and young adults with many different kinds of developmental disabilities (i.e., those with autism, with fragile X syndrome, or with other intellectual disabilities) relative to non-affected peers (McDermott et al. 2015). At the same time, large-scale administrative studies continue to highlight a higher volume of psychiatric-related health service use in adults with autism compared to those with other kinds of developmental disabilities (Weiss et al. 2018).

While service productivity may be high, accessibility, continuity, and comprehensiveness may be low, with many individuals reporting difficulties in receiving services that correspond to priority needs (Lai and Weiss 2017). Unmet needs have been described with regard to healthcare, therapeutic, and family support services. Individuals with autism often experience many barriers to service receipt across the lifespan and sectors, as

a result of wait lists, a lack of resources, and inadequate service provider skills ((Hodgetts et al. 2015). Unmet needs have been associated with clinical severity and demographic factors (e.g., age, race, socioeconomic status, or rurality). Systemic variables are also important to consider, as in some jurisdictions, only individuals with a certain severity of autism or with co-occurring intellectual disability are eligible for services. Service availability is also related to how services are funded and the capacity of providers. There are significant transition-based issues with service utilization, with gaps in provision being associated with moving from child to adult contexts. It is important to note that barriers to service receipt at one point in time can influence services needed and used in the future. For example, good continuity of care can result in a reduced use of emergency-related services in individuals with autism and other developmental disabilities (Wood et al. 2007). Continued research is needed to understand trajectories of service utilization over time.

See Also

- Autism Acceptance and Mental Health
- Benhaven Residential Services
- Community Services
- Emergency Department Utilization and Autism
- Health Disparities

References and Reading

- Cummings, J. R., Lynch, F. L., Rust, K. C., Coleman, K. J., Madden, J. M., Owen-Smith, A. A., et al. (2016). Health services utilization among children with and without autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46(3), 910–920. <https://doi.org/10.1007/s10803-015-2634-z>.
- Da Silva, R. B., Contandriopoulos, A.-P., Pineault, R., & Tousignant, P. (2011). A global approach to evaluation of health services utilization: Concepts and measures. *Healthcare Policy*, 6(4), e106–e117. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/22548101>.
- Hodgetts, S., Zwaigenbaum, L., & Nicholas, D. (2015). Profile and predictors of service needs for families of

- children with autism spectrum disorders. *Autism*, 19(6), 673–683. <https://doi.org/10.1177/1362361314543531>.
- Lai, J. K. Y., & Weiss, J. A. (2017). Priority service needs and receipt across the lifespan for individuals with autism spectrum disorder. *Autism Research*, 10(8), 1436–1447. <https://doi.org/10.1002/aur.1786>.
- McDermott, S., Hardin, J. W., Royer, J. A., Mann, J. R., Tong, X., Ozturk, O. D., & Ouyang, L. (2015). Emergency department and inpatient hospitalizations for young people with fragile X syndrome. *American Journal on Intellectual and Developmental Disabilities*, 120(3), 230–243. Retrieved from <http://www.aaiddjournals.org/doi/10.1352/1944-7558-120.3.230>.
- Weiss, J. A., Isaacs, B., Diepstra, H., Wilton, A. S., Brown, H. K., McGarry, C., & Lunsy, Y. (2018). Health concerns and health service utilization in a population cohort of young adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48, 36–44. <https://doi.org/10.1007/s10803-017-3292-0>.
- Wood, D., Hall, A., Hou, T., Wludyka, P., & Zhang, J. (2007). Continuity of care to prevent emergency room use among persons with intellectual and developmental disabilities. *Journal of Policy and Practice in Intellectual Disabilities*, 4(4), 219–228. <https://doi.org/10.1111/j.1741-1130.2007.00127.x>.

Set Shifting

- Cognitive Flexibility

Setting Events

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Definition

Setting events are prior events or conditions, internal or external to the individual, that influence the probability and form of behavior presented by the individual. Setting events are related to the concepts of discriminative stimulus and establishing operation. A discriminative stimulus is an antecedent stimulus in the presence of which a response is likely to occur

because in the past, the response has been more successful in the presence of that stimulus than in the absence of that stimulus. An establishing operation (sometimes referred to as motivating operation) is an environmental event that affects the individual by momentarily altering the reinforcing effectiveness of a consequence and altering the rate or intensity of behavior that has resulted in that consequence (Michael 1993). Typical stimulus control analyses focus on brief temporal associations between stimuli and response, focusing on stimuli contiguous with the behavior of interest. The distinction between setting events and the concepts of discriminative stimulus and establishing operation is that setting events do not refer to the simple presence or absence of discrete immediate objects or events. Setting events are more complex events that may overlap the behavior of interest or precede the behavior of interest by immediate or large temporal gaps, and setting events may include the individual's response to events. The key distinction is that the setting events might be temporally distant from the behavior of interest and that they could include stimuli within the individual. Thus setting events could occur in a wholly separate time and place, long before the behavior of interest, or they could occur while the behavior of interest is happening. The section "[Current Knowledge](#)" presents examples of setting events that may influence the behavior of individuals with autism. The events could have occurred hours or days before, or they include the behavior of the individual, yet the setting events still have an impact on the behavior of the individual (Wahler and Fox 1981).

Historical Background

Early research in applied behavior analysis focused on the immediate antecedents and consequences of behavior. Setting events emerged from efforts to understand the potential influence of environment interactions that were not immediate (Kantor 1959).

Current Knowledge

There are many setting events known to influence the behavior of individuals with autism. It is critical for those working and living with individuals diagnosed with autism to identify known setting events, the behavioral circumstances people recently or historically passed through. Knowledge of the individual is equally important as knowledge of autism because a specific setting event will likely be functional for some people and not for others. Parents and teachers who have a lot of experience with a specific person are the best sources for determining which setting events are influential. Knowing how a specific setting event might influence a specific behavior in the individual has the advantage of allowing time to prepare positive behavior supports (Lucyshyn et al. 2002).

Examples of antecedent interventions are presented below. The emphasis on understanding how setting events influence the actions of individual people cannot be overstated. The adage, "once you have seen one person with autism, you have seen one person with autism," is quite applicable. The details of the influences of setting events are highly individualized across people with autism.

Types of Setting Events

Environmental. Characteristics of setting events in the environment may include if the setting is loud and chaotic versus quiet and orderly, a crowd of people versus a few people, daily routines versus high-rate novelty and unpredicted schedule changes, difficult demands versus previously mastered demands, demands that require a long time to complete or concurrent demands presented at a high rate versus demands that can be completed quickly (Carr et al. 1996).

Physiological. Physiological setting events of greatest interest within the individual are those that make the person with autism less amenable to reinforcement, less tolerant of frustration, and more likely to engage in intense behavior

reinforced by attention or escape from stress. Such events may include pain (e.g., earaches, toothaches, sunburns, and constipation), illness (e.g., seasonal allergies), discomfort (disorienting medication), lack of sleep or fatigue, recent seizure activity (Carr and Smith 1995), and menstruation (Carr et al. 2003). In some cases, setting events are related to sensory dysfunction that includes auditory, tactile, or visual defensiveness. Therefore, individual behavior may be influenced by loud, high-pitched, or sudden noise (e.g., vacuum cleaners or electronic pencil sharpeners), clothes that are too tight or too loose, seat belts that are too tight, sensations that are too gentle such as a soft touch, or light that is too bright, fluorescent, or blinking.

Social. Social setting events may have occurred earlier in the day, on the bus to school, just before or during the time of interest. These events could include interpersonal conflict, novel people, or the absences of selected people typically present (the substitute teacher syndrome).

Immediate antecedents. Discrete, immediate events that may influence the subsequent behavior of individuals with autism include failure to pass contracts or earn rewards that include preferred activities or tangible reinforcers, the delay or loss of scheduled rewards or preferred activities due to unforeseen events (e.g., cannot go on the scheduled trip to the park because of inclement weather, cannot visit a friend because they are ill), or simple instructions, demands, or requests to engage in required activity versus opportunities to choose (Carr and Newsom 1985).

Psychological states. Finally, internal behavioral characteristics of the individual can serve as setting events that alter the reinforcing properties of consequences. Psychological states may include sad mood, stress, perseverative thinking or overfocused thoughts, specific fears, or the absence of specific stimuli. For example, a comfort item such as a blanket or a specific toy might set the occasion for desirable behavior. Conversely, the absence of the comfort item might set the occasion for intense anxious fear responses.

Functional Assessment

Knowledge of potential setting events highlights the importance of functional assessment. However, functional assessment needs to evaluate more than the current setting and the events immediately preceding the behavior. The functional assessment may need to assess events long preceding the immediate behavior of interest and behavior within the individual. In some cases, an indirect functional assessment that utilizes subjective data provided by caregivers may be more practical than an experimental functional analysis. If behavior is disrupted by global occurrences such as weather patterns (e.g., snow days that disrupt school routines), it might be systematically difficult to alter those events. However, an event that is not manipulable in an experimental sense may still affect the behavior.

Setting events are not unique to individuals with autism. What is unique is the wide range of idiosyncratic events that may serve to influence the behaviors of a person with autism and the wide range of maladaptive responses that might be evoked. The setting events described above can lead to behavior ranging from resistance to teaching to tantrums, aggression, self-injurious behavior or stereotypical repetitive behavior. It is important to avoid the temptation to seek rationale explanation for the impact of a setting event. If pencil sharpeners disturb a child, we may never know why, but that does not reduce the impact of the pencil sharpener.

Intervention

As suggested above, a functional analysis of setting events can lead directly to intervention strategies. Staff and parents may (1) prevent a stressful event from occurring or remove the person from that event before the person is exposed to the stressor, (2) ensure that specific stimuli that occasion success are present, (3) make alternate preparations to increase the likelihood of success (e.g., foreshadow an event with a student and then rehearse potential adaptive responses), or (4) program incremental exposure to setting events to increase opportunities to adapt and respond successfully.

There are advantages to interventions that focus on antecedent programming. Sometimes

reinforcement procedures do not work because they are difficult to consistently apply or because of difficulties identifying effective reinforcers. There are numerous concerns about the role of punishing consequences. In contrast, caretakers may be more willing to apply antecedent intervention. “Antecedent intervention has the purpose of preventing the occurrence of challenging behavior . . . an antecedent approach manipulates conditions to eliminate the probability of undesirable behavior” (Luiselli 2008, p. 394). With the help of parent and teacher input, indirect or descriptive functional assessments can lead to hypothesis statements about the strength of setting events or events that correlate with behavior. The outcomes may be hypotheses that are useful in a practical sense on a daily basis.

The Setting and the Schedule

For example, teachers could assess the presence of psychological or medical states. Upon arriving at school, personnel may ask questions such as “Is he overfocused on anything today? Did he talk about seeing insects on the bus ride to school?” or “How did he sleep last night? Is his nose still stuffy?” Knowledge of a person’s physiological or medical state or social interactions earlier that day may result in temporary curriculum changes (e.g., more choices, Cannella et al. 2005; preferred tasks, shorter tasks, slower pacing, Luiselli 2008).

Knowledge of sensory dysfunction can lead to simple solutions. For example, the noise occasioned when a classroom chair slides across the floor might evoke high-rate hand flapping and an emotional tantrum. An inexpensive solution observed in many classrooms is tennis balls attached to legs of chairs, so noise is suppressed. The importance of environmental structure leads to careful consideration of predictable scheduling.

Skill building. Functional communication (O’Reilly et al. 2006) can help teach adaptive skills when stressful settings are identified. A teacher could help a student prepare for a challenging lesson by teaching the student an adaptive response such as “I need help.” In turn, the teacher could provide assistance and praise contingent on the student’s request for help. If noisy settings elicit emotional reaction, in separate discrete trials, staff could gradually expose the student to

incremental increases in the noise, allowing for sensory adaptation (habituation). These hypothetical strategies can emerge directly from understanding how an environmental event influences the behavior of the individual child.

Future Directions

Setting events are important, but compared with discriminative stimuli, setting events are less likely to be a focus of experimental research or a functional analysis. One reason is that setting events can occur in remote time long before the behavior of interest, and functional analysis tends to focus on the current setting or stimuli that immediately precede the behavior of interest. Another reason setting events are less considered is that functional analysis most often focuses on reinforcing consequences, whereas some setting events affect behavior regardless of the consequences. Finally, since our original understanding of the law of effect, the majority of behavior modification research has focused on manipulating consequences of behavior. Nonetheless, the advantages and successes of antecedent interventions bode well for proportionately more research.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavioral Assessment](#)
- [Functional Analysis](#)

References and Reading

- Cannella, H. I., O’Reilly, M. F., & Lancioni, G. (2005). Choice and preference assessment research with people with severe to profound developmental disabilities: A review of the literature. *Research in Developmental Disabilities, 26*, 1–15.
- Carr, E. G., & Newsom, C. (1985). Demand-related tantrums: Conceptualization and treatment. *Behavior Modification, 9*, 403–426.
- Carr, E. G., & Smith, C. E. (1995). Biological setting events for self-injury. *Mental Retardation and Developmental Disabilities Research Reviews, 1*, 94–98.

- Carr, E. G., Reeve, C. E., & Magito-McLaughlin, D. (1996). Contextual influences on problem behavior in people with developmental disabilities. In L. K. Koegel, R. L. Koegel, & G. Dunlap (Eds.), *Positive behavioral support: Including people with difficult behavior in the community* (pp. 403–423). Baltimore: Paul H. Brookes.
- Carr, E. G., Smith, C. E., Giacini, T. A., Whelan, B. M., & Pancari, J. (2003). Menstrual discomfort as a biological setting event for severe problem behavior. *American Journal on Mental Retardation*, 108, 117–133.
- Kantor, J. R. (1959). *Interbehavioral psychology*. Granville: Principia Press.
- Lucyshyn, J. M., Kayser, A. T., Irvin, L. K., & Blumberg, E. R. (2002). Functional assessment and positive behavioral support at home with families. In J. M. Lucyshyn, G. Dunlap, & R. W. Albin (Eds.), *Families and positive behavior support* (pp. 97–132). Baltimore: Paul H. Brookes.
- Luiselli, J. K. (2008). Antecedent (preventive) intervention. In J. K. Luiselli, D. C. Russo, W. P. Christian, & S. M. Wilczynski (Eds.), *Effective practices for children with autism: Educational and behavioral support interventions that work* (pp. 393–412). New York: Oxford University Press.
- Michael, J. L. (1993). *Concepts and principles of behavior analysis*. Kalamazoo: Society for the Advancement of Behavior Analysis.
- Newsom, C., & Hovanitz, C. A. (2006). Autism spectrum disorders. In E. J. Mash & R. A. Barkley (Eds.), *Treatment of childhood disorders* (3rd ed., pp. 455–511). New York: Guilford Press.
- O'Reilly, M. F., Cannella, H. I., Sigafos, J., & Lancioni, G. (2006). Communication and social skills intervention. In J. K. Luiselli (Ed.), *Antecedent control: Innovative approaches to behavior support* (pp. 187–206). Baltimore: Paul H. Brookes.
- Wahler, R. G., & Fox, J. J. (1981). Setting events in applied behavior analysis: Toward a conceptual and methodological expansion. *Journal of Applied Behavior Analysis*, 14, 327–338.

Sex Chromosomes

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Synonyms

X and Y chromosomes

Definition

The sex chromosomes (X and Y) determine male (XY) and female (XX) genetic sex. The X chromosome is large and contains considerably more genetic material than the Y chromosome (155 megabases (Mb) of DNA vs. 60 Mb) (Jorde et al. 2010). Sex-linked disorders are caused by mutations on either the X or Y chromosome and demonstrate characteristic modes of inheritance that distinguish them from disorders caused by mutations on the autosomes, or the nonsex chromosomes. For example, X-linked mutations cannot be transmitted from father to son, unlike autosomal mutations, and are more likely to affect males, because males have only one X chromosome (Jorde et al. 2010).

Because autism is four times more likely to occur in males than females and is highly heritable, studies have examined the extent to which genes found on the X chromosome might contribute to the etiology of autism (Skuse 2000, 2007). That is, early studies questioned whether autism is an X-linked disorder, based on the male predominance. However, there is no evidence from genome-wide linkage scans to suggest a simple pattern of sex-linked inheritance (Skuse 2000, 2007). This is consistent with findings from recent large-scale genome-wide studies that are revealing considerable heterogeneity in the genetic architecture of autism such that multiple genes on different chromosomes likely interact to confer risk (State 2010).

Although idiopathic autism is highly heterogeneous and characterized by a complex pattern of inheritance, studies of rare monogenic syndromes that are associated with autism have been instrumental in identifying genes that are likely to confer risk (State 2010). While these susceptibility genes are found throughout the genome, a number of them are located on the X chromosome. These include *fragile X mental retardation protein*, the gene that causes fragile X syndrome, which is highly associated with autism (Moss and Howlin 2009). In addition, Rett syndrome, which occurs almost exclusively in females, is caused by mutations in the X-linked *MECP2* gene (Moss and Howlin). Another important X-linked

susceptibility gene is *neurexin 4X* (*NLGN4X*), which was identified in studies of two families with autism, one of which included individuals with autism and intellectual disability, carrying truncating mutations disrupting this gene (State 2010).

Given the considerable genetic heterogeneity and lack of evidence for a simple pattern of X-linked inheritance, the cause of the higher frequency of autism in males is not known. One hypothesis is that imprinting of genes on the X chromosome may lead to the expression of protective genes on the paternally inherited X chromosome in females (Skuse 2000). Another question is whether X-linked syndromes of intellectual disability, which are more common in males, account in part for the male predominance, yet severe intellectual disability occurs more often in females with autism (Skuse 2007). One recent model addresses the overlap of autism and intellectual disability in the context of emerging observations regarding pleiotropy, suggesting that susceptibility genes may confer risk for both autism and intellectual disability, and that the expression of one or the other is influenced by the combined effects of genetic, epigenetic, and environmental factors during development (State 2010).

See Also

- Chromosomal Abnormalities
- Pleiotropy
- Sex Hormones

References and Reading

- Jorde, L. B., Carey, J. C., & Bamshad, M. J. (2010). *Jorde: Medical genetics* (4th ed.). Philadelphia: Mosby.
- Moss, J., & Howlin, P. (2009). Autism spectrum disorders in genetic syndromes: Implications for diagnosis, intervention and understanding the wider autism spectrum disorder population. *Journal of Intellectual Disability Research*, 53(10), 852–873.
- Skuse, D. H. (2000). Imprinting, the X-chromosome, and the male brain: Explaining sex differences in the liability to autism. *Pediatric Research*, 47(1), 9–16.

Skuse, D. H. (2007). Rethinking the nature of genetic vulnerability to autistic spectrum disorders. *Trends in Genetics*, 23(8), 387–395.

State, M. W. (2010). The genetics of child psychiatric disorders: Focus on autism and Tourette syndrome. *Neuron*, 68(2), 254–269.

Sex Differences in ASD on Restricted and Repetitive Behaviors and Interests

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Definition(s)

Restricted and repetitive behaviors and interests (RRBI) is a symptom cluster of autism spectrum disorder (ASD), which include stereotyped or repetitive behaviors, sameness behaviors, restricted interests, self-injurious behaviors, and sensory processing differences (American Psychiatric Association 2013). The *Diagnostic and Statistical Manual of Mental Disorders Fifth Edition* defines these items as follows:

- Stereotyped and repetitive behaviors are behaviors that happen over and over again in an exact way (e.g., playing with the same toy in the same way, abnormal or excessive repetition of the same speech) or unusual motor movements such as hand-flapping or body rocking.
- Sameness behaviors are the insistence to perform actions in the same way including insistence on routines, anxiety surrounding changes in plans, and resulting difficulties with changes in schedules.
- Restricted interests are interests, hobbies, or preoccupations that are unusual in their intensity and in breadth that often occupy most of an individual's time and focus.
- Self-injurious behaviors are behaviors that result in bodily harm and are inflicted by oneself, including skin picking; hair pulling;

biting, hitting, or pinching oneself; head banging, etc.

- Sensory processing differences include a wide array of sensory features, such as hypo- or hyper-reactivity to sensory input (e.g., sounds, pain), sensory seeking behaviors (using senses to explore the world such as smelling objects or people), and sensory avoiding behaviors (e.g., textures, sounds, smells, tastes that may be aversive).

RRBI are commonly measured by three instruments:

1. ADOS-2 (Autism Diagnostic Observation Schedule-Second Edition; Lord et al. 2012) RRB subscale
2. ADI-R (Autism Diagnostic Interview-Revised; Lord et al. 1994) RRB subscale
3. RBS-R (Repetitive Behavior Scale-Revised; Bodfish et al. 2000).

Historical Background

RRBI is one of the two core symptom clusters used to diagnose ASD and has been a core symptom cluster of autism since its very conception as a pervasive developmental disorder (PDD) in 1980 in the *DSM-III*, although RRBI was then referred to as “bizarre responses to the environment” (APA 1980). However, RRBI has garnered attention for its presenting sex differences in ASD. In particular, RRBI is suggested to be less predictive of an ASD diagnosis for females relative to males. Thus, sex differences in the RRBI domain have been studied to better understand the sex discrepancy (i.e., larger male to female ratio) in current ASD diagnostic rates (Baio et al. 2018).

Current Knowledge

RRBI, when studied as a unitary construct, often reveals no significant sex differences in ASD. These results have held true in toddlers diagnosed at 24 months of age (Lawson et al.

2018), in older toddlers (4 years; Carter et al. 2007), in older children (Banach et al. 2009), and in older adolescents and adults after controlling for ASD severity (Harrop et al. 2015; Holtmann et al. 2007). However, looking into RRBI subscales indicates subtle sex differences with ASD that may not be detectable when looking at the RRBI domain as a whole.

Using the parent-reported RBS-R, previous work has noted that varying sub-domains of RRBI are not as predictive of an ASD diagnosis for females as for males (Antezana et al. 2018; Duvekot et al. 2016). Sex differences in RRBI items on the RBS-R emerged for eight different items, including “pulls hair/skin,” “fascination with movement of object,” “hand/finger mannerisms,” “hoarding/saving,” “rubs or scratches self,” “object usage,” “insists on sitting at the same place,” and “preoccupation with parts of an object.” Overall, females demonstrated fewer stereotyped and restricted behaviors, including restricted interests, but more compulsive, restricted, self-injurious, and sameness behaviors than males (Antezana et al. 2018). These results are particularly interesting as they indicate some RRBI areas where females have greater or fewer RRBI scores, which may explain why no sex differences emerge when reviewing RRBI as a unitary construct: these subtle differences are washed out. Specific subscales of RRBI are discussed below.

Stereotyped and Repetitive Behaviors

The RRBI domain includes many different types of behaviors and manifestations (see Definitions); however, across all behaviors, females demonstrate decreased stereotyped and repetitive behaviors. These differences in stereotyped and repetitive behaviors are present across development and include decreased rates of preoccupations, routines, lining up of objects, and stereotyped mannerisms (Antezana et al. 2018; Hattier et al. 2011; Hiller et al. 2014; Nicholas et al. 2008). Thus, across development and the range of stereotyped and repetitive behaviors, females appear to demonstrate fewer behaviors than males on the spectrum.

Sameness Behaviors

Little work has investigated sex differences in sameness behaviors. As mentioned above, RBS-R items indicate stronger female insistence on sameness than males, as indicated by responses to items such as “insists to sit in the same place” (Antezana et al. 2018). Other results have indicated that insistence on sameness (IS) is a distinct subscale from the other behaviors included in RRBI and may present with unique manifestations relative to other behaviors. As IS has been related to communication and cognitive performance in ASD (Szatmari et al. 2006), it is expected that sex differences in IS are present; however, this area is underexplored.

Restricted Interests

Females present with fewer parent- and teacher-reported restricted interests than males (Beggiato et al. 2016; Hiller et al. 2014) that are also lower in severity (McFayden et al. 2018). Not only are these interests different in severity and frequency, but females present their interests in a different fashion relative to males, which may make them harder to detect in females. Females demonstrate greater interests in living organisms, humans, and dolls, which appear social in nature, and may even discuss their interests differently by conveying them in more socially appropriate ways (e.g., writing letters to animal activists and engaging with others) and using less rote presentations of facts or figures relative to males (Mandy et al. 2012; McFayden et al. 2018). Taken together, females have fewer restricted interests, which are lower in severity, and also conveyed and discussed differently than those of males; all of which may make restricted interests more difficult to identify and diagnose in females with ASD.

Self-Injurious Behaviors

Sex differences in self-injurious behaviors are not detectable in toddlers or preschoolers (e.g., Schroeder et al. 2014 and Solomon et al. 2012), but sex differences begin to emerge in school-aged children and continue through adulthood (Beggiato et al. 2016). Females present with greater self-injurious behaviors in the forms of hair pulling, scratching, and cutting (Antezana

et al. 2018). When including non-suicidal self-injurious behaviors (NSSIs), the same pattern of sex differences remains: females endorse greater NSSIs than males starting in adolescence and continuing into adulthood (Maddox et al. 2016). These sex differences in ASD also reflect sex differences in typically developing populations, where females engage in self-injurious behaviors and NSSIs at a greater frequency than males (Bresin and Shoenleber 2015).

Sensory Processing Differences

Overall, little work has been done looking at sensory feature sex differences in autism. Generally, sensory features are similar between males and females. In one work, parents of males indicated greater difficulty with physical sensory features (“endurance/tone” on the Short Sensory Profile; Bitsika et al. 2018). However, this one sensory difference may, in fact, relate more to stereotyped motor movement sex differences in ASD, as discussed above. Sex differences in sensory features remain an underexplored area.

Neurophysiology

Sex differences in RRBI in autism have also been explored neurophysiologically by looking at brain structure, brain function, hormones, and neurotransmitters across males and females with autism spectrum disorder. Results indicate significant sex differences related to RRBI that are visible at the neural level. Structural MRI indicates sex differences in cortical thickness, intraparietal sulcus, gray matter, motor cortices, and the cerebellum, which are linked to RRBI (Supekar and Menon 2015; Van’t Westeinde et al. 2018). Hormonally, sex differences in arginine vasopressin and oxytocin were related to sex differences in RRBI (Miller et al. 2013). Together, there is clear evidence to suggest behavioral and neurophysiological sex differences in RRBI in ASD.

Summary

Subtle sex differences in RRBI in ASD are present. Females have fewer stereotyped/repetitive behaviors and restricted interests but have more self-injurious behaviors and insistence on sameness than males. At this point, not enough has

been conducted in the field of sensory features to be able to draw strong conclusions. When viewed as a whole, the sex differences in RRBI in autism mirror the sex differences in typically developing populations quite well: females present with more “internalizing” features, whereas males present with more “externalizing,” easily observable features. This generalization holds apart from self-injurious behaviors, which are observable behaviors and also more common in females than males.

Implications

Importantly, these differences have implications for diagnostic measures. The female presentation of stereotyped/repetitive behaviors and restricted interests is less severe and less noticeable than males and in fact may not be present to a detectable extent by select diagnostic measures. This work highlights important future directions for research and clinical assessment, discussed below.

Future Directions

Future research must investigate RRBI by looking into the sub-components instead of only reviewing RRBI as a unitary construct. There are several underexplored areas of RRBI sex differences including sensory processing features (e.g., hypo-/hyper-reactivity to sensory input), stereotyped speech (e.g., echolalia), and insistence on sameness. Measurement of RRBI should be better specified to indicate whether RRBI is measured by frequency of behaviors (e.g., how many times are they occurring?), severity or intensity of behaviors (e.g., how extreme or intense is this behavior?), or the behavior’s interference in family or social life (e.g., how much is this behavior getting in the way?). As comorbidities of ASD are becoming better understood, future directions also include studying how comorbid psychopathology impacts RRBI presentation of sex differences, especially disorders that already present as more common in one sex over the other (e.g., ADHD in males; anxiety in females). Lastly, future research on the social construct of gender self-identification and its impact on RRBI and other

ASD criteria will aid in furthering the field’s understanding of sex and gender differences in ASD, as well as understanding diagnostic specificity.

See Also

- [Repetitive Behavior](#)
- [Restricted Interest](#)
- [Treatment for Higher-Order Restricted, Repetitive Behaviors](#)

References and Reading

- American Psychiatric Association (APA). (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed., DSM-III). Washington, DC: American Psychiatric Association.
- American Psychiatric Association (APA). (2013). *Diagnostic and statistical manual of mental disorders* (5th ed., DSM-V). Arlington: American Psychiatric Publishing.
- Antezana, L., Factor, R. S., Condy, E. E., Strege, M. V., Scarpa, A., & Richey, J. (2018). Gender differences in restricted and repetitive behaviors and interests in youth with autism. *Autism Research*, 12, 274–283. <https://doi.org/10.1002/aur.2049>.
- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., et al. (2018). Prevalence of autism Spectrum disorder among children aged 8 years—Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summaries*, 67(6), 1–23. <https://doi.org/10.15585/mmwr.ss6706a1>.
- Banach, R., Thompson, A., Goldberg, J., Tuff, L., Zwaigenbaum, L., & Mahoney, W. (2009). Brief report: Relationship between non-verbal IQ and gender in autism. *Journal of Autism and Developmental Disorders*, 39, 188–193. <https://doi.org/10.1007/s10803-008-0612-4>.
- Beggiato, A., Peyre, H., Maruani, A., Scheid, I., Rastam, M., Amsellem, F., et al. (2016). Gender differences in autism spectrum disorders: Divergence among specific core symptoms. *Autism Research*, 10, 680–689. <https://doi.org/10.1002/aur.1715>.
- Bitsika, V., Sharpley, C. F., & Mills, R. (2018). Sex differences in sensory features between boys and girls with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 51, 49–55. <https://doi.org/10.1016/j.rasd.2018.04.002>.
- Bodfish, J. W., Symons, F. J., Parker, D. E., & Lewis, M. H. (2000). Varieties of repetitive behavior in autism:

- Comparisons to mental retardation. *Journal of Autism and Developmental Disorders*, 30, 237–243. <https://doi.org/10.1023/A:1005596502855>.
- Bresin, K., & Shoenleber, M. (2015). Gender differences in the prevalence of nonsuicidal self-injury: A meta-analysis. *Clinical Psychological Reviews*, 38. <https://doi.org/10.1016/j.cpr.2015.02.009>.
- Carter, A. S., Black, D. O., Tewani, S., Connolly, C. E., Kadlec, M. B., & Tager-Flusberg, H. (2007). Sex differences in toddlers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37, 86–97. <https://doi.org/10.1007/s10803-006-0331-7>.
- Duvekot, J., van der Ende, J., Verhulst, F. C., Slappendel, G., van Daalen, E., Maras, A., & Greaves-Lord, K. (2016). Factors influencing the probability of a diagnosis of autism spectrum disorder in girls versus boys. *Autism*, 21, 646–658. <https://doi.org/10.1177/1362361316672178>.
- Harrop, C., Gulsrud, A., & Kasari, C. (2015). Does gender moderate core deficits in ASD? An investigation into restricted and repetitive behaviors in girls and boys with ASD. *Journal of Autism and Developmental Disorders*, 45, 3644–3655. <https://doi.org/10.1007/s10803-015-2511-9>.
- Hattier, M. A., Matson, J. L., Tureck, K., & Horovitz, M. (2011). The effects of gender and age on repetitive and/or restricted behaviors and interests in adults with autism spectrum disorders and intellectual disability. *Research in Developmental Disabilities*, 32, 2346–2351. <https://doi.org/10.1016/j.ridd.2011.07.028>.
- Hiller, R. M., Young, R. L., & Weber, N. (2014). Sex differences in autism spectrum disorder based on DSM-5 criteria: Evidence from clinician and teacher reporting. *Journal of Abnormal Child Psychology*, 42, 1381–1393. <https://doi.org/10.1007/s10802-014-9881-x>.
- Holtmann, M., Bölte, S., & Poustka, F. (2007). Autism spectrum disorders: Sex differences in autistic behaviour domains and coexisting psychopathology. *Developmental Medicine & Child Neurology*, 49, 361–366. <https://doi.org/10.1111/j.1469-8749.2007.00361.x>.
- Lawson, L. P., Joshi, R., Barbaro, J., & Dissanayake, C. (2018). Gender differences during toddlerhood in autism spectrum disorder: A prospective community-based longitudinal follow-up study. *Journal of Autism and Developmental Disorders*, 48, 2619–2628. <https://doi.org/10.1007/s10803-018-3516-y>.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24, 659–685. <https://doi.org/10.1007/7814313>.
- Lord, C., Rutter, M., DiLavore, P. C., Risi, S., Gotham, K., & Bishop, S. (2012). *Autism diagnostic observation schedule* (2nd ed.). Torrance: Western Psychological Services.
- Maddox, B. B., Trubanova, A., & White, S. W. (2016). Untended wounds: Non-suicidal self-injury in adults with autism spectrum disorder. *Autism*, 21, 412–422. <https://doi.org/10.1177/1362361316644731>.
- Mandy, W., Chilvers, R., Chowdhury, U., Salter, G., Seigal, A., & Skuse, D. (2012). Sex differences in autism spectrum disorder: Evidence from a large sample of children and adolescents. *Journal of Autism and Developmental Disorders*, 42, 1304–1313. <https://doi.org/10.1007/s10803-011-1356-0>.
- McFayden, T. C., Albright, J. A., Muskett, A. E., & Scarpa, A. (2018). The sex discrepancy in an ASD diagnosis: An in depth look at restricted interests and repetitive behaviors. *Journal of Autism and Developmental Disorders*, 49, 1693–1699. <https://doi.org/10.1007/s10803-018-3839-9>.
- Miller, M., Bales, K., Taylor, S. L., Yoon, J., Hostetler, C. M., Carter, C. S., & Solomon, M. (2013). Oxytocin and Vasopressin in children and adolescents with autism spectrum disorders: Sex differences and associations with symptoms. *Autism Research*, 6, 91–102. <https://doi.org/10.1002/aur.1270>.
- Nicholas, J. S., Charles, J. M., Carpenter, L. A., King, L. B., Jenner, W., & Spratt, E. G. (2008). Prevalence and characteristics of children with autism-spectrum disorders. *Annals of Epidemiology*, 18, 130–136. <https://doi.org/10.1016/j.annepidem.2007.10.013>.
- Schroeder, S. R., Marquis, J. G., Reese, R. M., Richman, D. M., Mayo-Ortega, L., Oyama-Ganiko, R., et al. (2014). Risk factors for self-injury, aggression, and stereotyped behavior among young children at risk for intellectual and developmental disabilities. *American Journal on Intellectual and Developmental Disabilities*, 119, 351–370. <https://doi.org/10.1352/1944-7558.119.4.351>.
- Solomon, M., Miller, M., Taylor, S. L., Hinshaw, S. P., & Carter, C. S. (2012). Autism symptoms and internalizing psychopathology in girls and boys with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42, 48–59. <https://doi.org/10.1007/s10803-011-1215-z>.
- Supekar, K., & Menon, V. (2015). Sex differences in structural organization of motor systems and their dissociable links with repetitive/restricted behaviors in children with autism. *Molecular Autism*, 6. <https://doi.org/10.1186/s13229-015-0042-z>.
- Szatmari, P., Georgiades, S., Bryson, S., Zwaigenbaum, L., Roberts, W., Mahoney, W., Goldberg, J., & Tuff, L. (2006). Investigating the structure of the restricted, repetitive behaviors and interests domain of autism. *The Journal of Child Psychology and Psychiatry*, 47, 582–590. <https://doi.org/10.1111/j.1469-7610.2005.01537.x>.
- Van't Westeinde, A. V., Cauvet, E., Toro, R., Kuja-Halkola, R., Neufeld, J., Mevel, K., & Bolte, S. (2018). Sex differences in brain structure: An autism twin study on restricted and repetitive behaviors. *BioRxIV: The Preprint Server for Biology*. <https://doi.org/10.1101/334367>.

Sex Education

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Definition

Sexual awareness comes early for most children. By age 3 years, most children recognize that girls and boys are different. An understanding of where babies come from and later on of the changes in one's body at puberty develops through interactions with many people: siblings, parents, other family members, peers, and teachers. These interactions may be difficult for people with ASDs.

People used to think that the social problems associated with autism prevented sexual interest or relationships. We now know that is not true. Early intervention as well as an emphasis on programs in the community has helped more people with ASDs to have very meaningful relationships, including sexual relationships. Parents of adolescents with ASDs, like many parents, may find it difficult to view adolescents as sexual beings, and they may worry about the adolescent being taken advantage of.

Children learn early that there are different rules for private and public behavior and for who can touch what, where, and when. Adolescents need to cope with distinctions between love and sex and learn about the importance of contraception and prevention of sexually transmitted infections as well as issues of separating from their families and developing long-term relationships. These issues may be more difficult for children on the autism spectrum because of language and communication difficulties, social issues, and varying cognitive levels. Learning about sexuality may not occur at the same age or in the same ways for children with ASDs. Peer relationships may be lacking, and other avenues for socializing may be lacking. It may be harder for children with ASDs to learn the difference between what is okay to think and what is acceptable to say or do. The distinction between private and public behavior

may be poorly understood, and the tendency to be very literal may cause misunderstandings.

It is important to make the learning process about sexuality as positive as possible and to help the child learn what not to do as well as what to do. The ultimate goal is to help the adolescent form a more positive view of himself or herself.

In general, parents of adolescents and young adults with ASDs should help dealing with emerging sexuality by having open discussions, with information at a level that is appropriate for the individual. Socially acceptable behaviors should be made clear. Sex is only one part of what needs to be discussed. The role that relationships play in sexuality and the differences between friendship and a sexual relationship need to be taught.

Adolescents with Asperger's disorder or autism can at times be quite preoccupied with sex or with wanting a girlfriend or boyfriend. These can be age-appropriate concerns but sometimes cause awkward situations when approaches are made in an unconventional manner.

Privacy and modesty are important concepts that may be difficult for children and adolescents with ASDs to understand. Difficulties in generalization can make these hard to teach and understand. Bathroom use early on can be a place to begin this teaching.

Masturbation can be an issue with any child. This can be a source of discomfort for parents and teachers. The distinction between private and public behavior may be hard to learn at times. This at times can be a problem in school. Allowing the student opportunities for frequent movement or objects to manipulate with the hands as an alternative activity may be helpful. Exercise can also be helpful. The occupational therapist or specialized teacher may be helpful in coming up with strategies for teaching the adolescent or young adult with an ASD that masturbation is not appropriate at school.

Teaching about boundaries is another important issue. Parents are frequently worried about the possibility of sexual or physical abuse. Abuse or inappropriate sexual behavior can be issues. Most children with ASDs are closely supervised and that will hopefully prevent either of these

problems. For verbal children, teaching the “no, go, tell” strategy may be useful. This method is described by Schwier and Hinsburger (2000).

Occasionally individuals on the autism spectrum experience difficulties in relation to gender identity. Also occasionally inappropriate sexual comments or requests can lead to legal entanglements.

See Also

► Daily Living Skills

References and Reading

- Aston, M. C. (2003). *Aspergers in love: Couple relationships and family affairs*. London: Jessica Kingsley.
- Aston, M. C. (2009). *The Asperger couple's workbook: Practical advice and activities for couples and counselors*. London: Jessica Kingsley.
- Attwood, S. (2008). *Making sense of sex: A forthright guide to puberty, sex and relationships for people with Asperger's syndrome*. London: Jessica Kingsley.
- Banach, R., Thompson, A., Szatmari, P., Goldberg, J., Tuff, L., Zwaigenbaum, L., et al. (2009). Brief report: Relationship between non-verbal IQ and gender in autism. *Journal of Autism & Developmental Disorders*, 39(1), 188–193.
- Bentley, K., & Attwood, T. (2007). *Alone together: Making an Asperger marriage work*. London: Jessica Kingsley.
- Bolick, T. (2001). *Asperger syndrome and adolescence: Helping preteens and teens get ready for the real world*. Gloucester: Fair Winds Press.
- Buron, K. D. (2007). *A 5 is against the law! Social boundaries: Straight up! An honest guide for teens and young adults*. Shawnee Mission: Autism Asperger.
- Fegan, L., Rauch, A., & McCarthy, W. (1993). *Sexuality and people with intellectual disability* (2nd ed.). Baltimore: Paul H. Brookes.
- Henault, I., & Attwood, T. (2005). *Asperger's syndrome and sexuality: From adolescence through adulthood*. London: Jessica Kingsley.
- Hingsburger, D. (1995). *Just say know! Understanding and reducing the risk of sexual victimization of people with developmental disabilities*. Horizons: Diverse City Press.
- Hirschfeld, L., Bartmess, E., White, S., & Frith, U. (2007). Can autistic children predict behavior by social stereotypes? *Current Biology*, 17(12), R451–R452.
- Kraemer, B., Delsignore, A., Gundelfinger, R., Schnyder, U., & Hepp, U. (2005). Comorbidity of Asperger syndrome and gender identity disorder. *European Child & Adolescent Psychiatry*, 14(5), 292–296.
- Schwier, K. M., & Hinsburger, D. (2000). *Sexuality and your sons and daughters with intellectual disabilities*. Baltimore: Paul H. Brookes.
- Shore, S. (2003). *Beyond the wall: Personal experience with autism and Asperger syndrome* (2nd ed.). Shawnee Mission: Autism Asperger.
- Sicile-Kira, C. (2006). *Adolescents on the autism spectrum: A parent's guide to the cognitive, social, physical, and transition needs of teenagers with autism spectrum disorders*. New York: Penguin.

Sex Hormones

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Synonyms

Androgens (male sex hormones); Estrogens (female sex hormones)

Definition

Sex hormones, including testosterone and estrogen, are produced by the gonads, or sex organs. In addition to their role in the development of the reproductive system, these hormones serve important functions in brain development, such as controlling the growth and survival of nerve cells, or neurons (Keller et al. 2008). Both testosterone and estrogen exert their effects on neurons by binding to protein receptors within the cell, which, in turn, directly control the expression of specific genes in these cells (Keller et al. 2008).

The higher frequency of autism in males (4:1) has raised questions about the effect of sex hormones on the risk of developing autism (Keller et al. 2008). One possible explanation for the male predominance, given the genetic risk of autism, is that sex hormones have a modifying effect on an underlying genetic predisposition (Keller et al.). According to this theory, sex hormones may influence the penetrance of a risk gene such that males

carrying a particular mutation may be more likely to develop symptoms of autism than females with the same mutation due to their exposure to different sex hormones during brain development (Keller et al.). It has been proposed that sex hormones may exert this modifying effect via an epigenetic mechanism, that is, by modulating the levels and timing of gene expression by altering chromatin structure (Kaminsky et al. 2006).

One group of researchers led by Simon Baron-Cohen has proposed an “extreme male brain” theory of autism, which posits that traits associated with autism represent a more marked version of a male pattern of cognitive processing (Auyeung and Baron-Cohen 2008; Baron-Cohen et al. 2005). According to this theory, females have a greater tendency toward “empathizing,” or understanding another person’s emotions, while the tendency of males is toward “systematizing,” or analyzing a system based on rules. Baron-Cohen suggests that individuals with autism function in a more extreme manner than the average male in their propensity to “systematize” rather than “empathize” (Auyeung and Baron-Cohen 2008; Baron-Cohen et al. 2005). Moreover, he proposes that such sexual differences in cognitive processing, and by extension, traits associated with autism, may be related to levels of fetal testosterone, and that the ratio of the lengths of the second and fourth digits, which is lower in males, may serve as an approach to assess exposure to androgens during fetal development (Auyeung and Baron-Cohen 2008). However, experimental validation of this hypothesis, via studies analyzing the association of fetal testosterone levels and the occurrence of autism, is required (Auyeung and Baron-Cohen).

There are a limited number of studies of differences in sex hormone levels in individuals with autism, and no studies of therapeutic interventions involving the use of sex hormones (Anderson and Hoshino 2005). At this time, further research is required to elucidate how sex hormones establish sexual dimorphisms both in the developing brain, producing structural differences, and at the molecular level, resulting in sex-specific differences in gene expression.

References and Reading

- Anderson, G. M., & Hoshino, Y. (2005). Neurochemical studies of autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 453–472). Hoboken: Wiley.
- Auyeung, B., & Baron-Cohen, S. (2008). A role for fetal testosterone in human sex differences: Implications for understanding autism. In A. W. Zimmerman (Ed.), *Autism: Current theories and evidence* (pp. 185–208). Totowa: Humana Press.
- Baron-Cohen, S., Knickmeyer, R. C., & Belmonte, M. K. (2005). Sex differences in the brain: Implications for explaining autism. *Science*, 310(5749), 819–823.
- Kaminsky, Z., Wang, S. C., & Petronis, A. (2006). Complex disease, gender and epigenetics. *Annals of Medicine*, 38(8), 530–544.
- Keller, F., Panteri, R., & Biamonte, F. (2008). Interaction between genetic vulnerability and neurosteroids in purkinje cells as a possible neurobiological mechanism in autism spectrum disorders. In A. W. Zimmerman (Ed.), *Autism: Current theories and evidence* (pp. 209–231). Totowa: Humana Press.

Sexuality and Problem Behaviors

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Synonyms

Deviant sexual behavior; Paraphilias; Problem sexual behavior

Definition

There are numerous reasons why the sexual behavior of people with Autism Spectrum Disorder (ASD) may appear atypical when compared to

the general population. Adolescence is the developmental period when sexual expression emerges for most individuals. In addition to the usual information provided (albeit often inadequately) by parents and school programs, typically developing adolescents rely on friends to learn about cultural norms of sexual behaviors and relationships (Song et al. 2000). Most adolescents with ASD, in contrast, do not develop the close reciprocal friendships that so often lead to the sharing and vetting of important information about sexuality (Murphy and Elias 2006). Thus, they may not have an opportunity to discuss their interests and behaviors with friends and miss the opportunity to hear others' perspectives and may not internalize the full set of social norms. The social differences of people with ASD also increase the likelihood of sexual behavior, knowledge, identity, and experience that differ from typical, mainstream experience, and raise the likelihood of sexual behaviors that are perceived as deviant (Dewinter et al. 2013). Further, while typically developing people may feel the urge to engage in less common sexual behaviors, they are usually influenced by peer perception and social expectations, and are highly attuned to the potential social and legal ramifications of engaging in these types behaviors.

When a person with ASD engages in illegal sexual behavior, the behavior may result from a core characteristic related to the diagnosis, rather than to a sexually "deviant" impulse. The term "counterfeit deviance" is sometimes used in such situations to describe inappropriate behavior rooted in factors other than true deviance (Hingsburger and Griffiths 1986). "Counterfeit deviance" is most often used in the context of inappropriate sexual behavior, in which the observed behavior looks paraphilic but, upon closer examination, was the result of other factors. Stalking often falls into this category. The social challenges associated with ASD may result in a person with ASD missing social cues that indicate their romantic target is not interested, and, because the person with ASD has not perceived these cues, they continue to pursue the person to the point that law enforcement becomes involved (Post et al. 2014). Likewise, a lack of awareness of

social perceptions of behavior or even a lack of concrete understanding that certain things happen in certain places can lead to charges like indecent exposure or public masturbation.

Despite these challenges, it is important to keep in mind that in many ways sexual behaviors in ASD may not differ at all from general population norms. For instance, one investigation of teenagers with ASD found behaviors, interests, and attitudes of those with ASD were largely consistent with those of neurotypical individuals. The only significant difference was that the boys with ASD were significantly more open-minded about homosexuality (Dewinter et al. 2015).

As with any group of people, it is possible for a person with ASD to display a truly deviant sexual interest or drive. It is important to keep in mind, however, that there is no evidence that truly deviant interests or drives occur at a higher rate in ASD than in the general population.

See Also

- ▶ [Counterfeit Deviance](#)
- ▶ [Sex Education](#)
- ▶ [Sexuality in Autism](#)
- ▶ [Stalking](#)

References and Reading

- Dewinter, J., Vermeiren, R., Vanwesenbeeck, I., & Nieuwenhuizen, C. (2013). Autism and normative sexual development: A narrative review. *Journal of Clinical Nursing*, 22(23–24), 3467–3483.
- Dewinter, J., Vermeiren, R., Vanwesenbeeck, I., Lobbestael, J., & Van Nieuwenhuizen, C. (2015). Sexuality in adolescent boys with autism spectrum disorder: Self-reported behaviours and attitudes. *Journal of Autism and Developmental Disorders*, 45(3), 731–741.
- Hellemans, H., Colson, K., Verbraeken, C., Vermeiren, R., & Deboutte, D. (2007). Sexual behavior in high-functioning male adolescents and young adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37(2), 260–269.
- Hingsburger, D., & Griffiths, D. (1986). Dealing with sexuality in a community residential service. *Psychiatric Aspects of Mental Retardation Reviews*, 5, 63.

- Murphy, N. A., & Elias, E. R. (2006). Sexuality of children and adolescents with developmental disabilities. *Pediatrics*, 118(1), 398–403.
- Song, E. Y., Pruitt, B. E., McNamara, J., & Colwell, B. (2000). A meta-analysis examining effects of school sexuality education programs on adolescents' sexual knowledge, 1960–1997. *Journal of School Health*, 70(10), 413–416.
- Tullis, C. A., & Zangrillo, A. N. (2013). Sexuality education for adolescents and adults with autism spectrum disorders. *Psychology in the Schools*, 50(9), 866–875.

Sexuality and Romantic Relationships Among People with ASD

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Definition

A review of the current research on sexual and romantic relationships and behaviors in the ASD population includes recommendations about sexual education for individuals with ASD with the help of their caretakers/teachers.

Historical Background

Autism spectrum disorder (ASD) is characterized by persistent deficits in social communication and interaction as well as restricted, repetitive behavior patterns (5th ed.; DSM-5; American Psychiatric Association 2013). Individuals most often exhibit signs and symptoms of autism in early childhood. However, distinct deficits may not manifest fully until social demands exceed the individual's social abilities (DSM-5 2013). Children with ASD often struggle in school due to low verbal and/or nonverbal intelligence quotients (IQ) and experience difficulty making friends (Lai et al. 2013). Despite these challenges, an increasing number of individuals with ASD have found work, earned the opportunity to attend

college, and formed successful relationships (Newman et al. 2011). Marriage was originally thought to be impossible for almost all individuals with ASD. However, a growing number of people with the disorder, especially those who are high functioning, engage in marriage (Newman et al. 2011).

A growing body of work has found that individuals with ASD often experience sexual desires and participate in romantic relationships (Van Bourgondien et al. 1997; Dewinter et al. 2015; Fernandes et al. 2016; Strunz et al. 2017). Little work regarding this topic has appeared until recently, as research initially labeled people with ASD as sexually immature, asexual, or, in the case of those caught up in legal punishment, hypersexual (Kellaher 2015). Historically, it was thought that individuals with ASD used only masturbation as an outlet for sexual frustrations (Van Bourgondien et al. 1997). Individuals with ASD were seen as more likely to be irritable and aggressive if their sexual needs were not met through self-pleasure, even though these hypotheses were not supported with empirical studies (Van Bourgondien et al. 1997). Additionally, there is a lack of data comparing the sexual and romantic behavior of males with that of females. Both the nature and the frequency of these behaviors should be analyzed. Females with ASD are a highly underrepresented demographic in autism research due to the disparity in prevalence of the disorder between sexes. The rate of ASD is three to four times higher in males than in females (Loomes et al. 2017). There is also minimal research regarding gender dysphoria among those with ASD.

Current Knowledge

Since the recent boom in ASD and intimacy research, perspectives on the matter have changed drastically. Strunz and colleagues found that individuals with ASD participate in romantic relationships more often than not, with 73% of study participants reporting that they had engaged in a romantic relationship. It is important to note, however, that the study only focused on

individuals who did not possess any form of cognitive impairment. Additionally, more than half of the participants who were single stated that human contact was too exhausting for them, expressed fear of not fulfilling a romantic partner's expectations, and/or admitted to not knowing how to find or get involved with a romantic partner. Half of the participants conveyed uncertainty about how to navigate relationships or how to behave when committed to one. (Strunz et al. 2017).

It has been found that individuals with ASD have a greater propensity to be bisexual or homosexual than the general population (Dewinter et al. 2017). Furthermore, people with ASD have a higher frequency of tolerance for homosexuality than that which is found in the general population (Dewinter et al. 2013, 2015; Fernandes et al. 2016). Dewinter et al. (2017) surveyed a large sample of adolescents and adults and found that 18.4% of men with ASD and 43.4% of women with ASD identified as non-heterosexual, while only 9.6% of typically developing men and 13% of typically developing women identified as non-heterosexual. An increased prevalence of gender dysphoria has also been found among individuals with ASD when compared to the rate in the general population. Dewinter et al. (2017) found that 8% of males with ASD and 22% of females with ASD experienced feelings of gender nonconformity; in the general population, 5.7% of males and 4% of females have gender dysphoria. The explanation for the difference in rates of gender dysphoria and non-heterosexuality between the ASD and non-ASD population is as of yet unclear.

Individuals with ASD are disproportionately at risk of becoming victims of sexual assault. Several increased risk factors exist within the ASD population, making these individuals more susceptible to abuse. Some of these risks include social-emotional deficits in understanding their own feelings as well as those of others, dependence on service providers who often have to help in the bathroom where sexual assault could be misconstrued with helping, poor communication, and difficulty identifying sexual assault. Despite the strong evidence for increased risk factors, there is no data to support an increased rate of sexual assault against people with ASD. Parents

reported that approximately 12% of participants with ASD experienced at least one instance of sexual abuse. This number is not significantly different from the general population, which is 6–10% for men and 16–23% for women. Primary reasons that a disparity has not been found are underreporting and stigma, which prevent the uncovering of an increased prevalence of sexual assault against people with ASD as compared to the general population. Sexual assault is often reported by a parent or guardian rather than the victim, and individuals with ASD may not tell their caretakers about their assault or not even understand they have been sexually assaulted. For example, some evidence suggests that the rate of assault is higher among males with ASD than among neurotypical males. Moreover, because ASD presents in vastly different ways for different individuals, it is hard to generally help and educate the population on understanding sexual assault (Sevlever et al. 2013).

Additionally, those with ASD may engage in sexually offending behavior due to their diminished capacity for empathy, poor social reciprocity, and possible sexual frustration (Sevlever et al. 2013). Individuals with ASD may not understand how their actions make others feel or that their actions are inappropriate (Sullivan and Caterino 2008; Fernandes et al. 2016). People with ASD are also less likely to understand or reference another person's emotions when presented with romantic situations and instead tend to refer back to learned social rules and norms (McNaughtan 2019). This could result in the individual with ASD not understanding the effect they are having on their partner's experience. In a 2002 study by Hurlbutt and Chalmers, three high-functioning individuals with ASD were interviewed and asked questions about their life experiences. One participant admitted to scaring off girls he liked by frequently calling them, not understanding that this behavior was a form of harassment (Hurlbutt and Chalmers 2002). It was found that individuals with ASD, but without physical identification explaining their diagnosis, are more likely to engage in inappropriate behavior (Fernandes et al. 2016). One possible cause is that, without ID, others may not understand that socially

inappropriate behavior could be the result of ASD and, therefore, shun rather than support those engaging in the behavior. Additionally, Sevlever et al. (2013) found that individuals with ASD are often legally cited for sexually offending behavior due to their difficulty in discriminating between privately appropriate and publically appropriate behavior, such as masturbation or nudity. Lack of social understanding, for example, in the context of sarcasm and jokes, may also lead individuals with ASD to act out sexually through exposure of genitalia or engaging in unwanted sexual contact with others. These actions frequently result in conflict with the forensic and legal systems, including being charged with sexually related offenses (Sevlever et al. 2013).

Kellaher and colleagues describe the prevalence of deviant sexual behaviors in individuals with ASD, which includes fetishism, pedophilia, and masochism. The behaviors can be grouped into autoerotic behaviors, like transvestism and fetishism, and person-oriented behaviors such as frottage and pedophilia. Of the cases examined, autoerotic behaviors did not lead to legal infractions, as they are not considered taboo. Of those exhibiting person-oriented deviant sexual behaviors, legal ramifications were less likely to occur if authority figures such as administrators or teachers had been understanding of the individual's diagnosis. Kellaher also speculates that the prevalence of sexually deviant behaviors – specifically sadism, transvestism, masochism and fetishism – may be connected to sensory abnormalities related to ASD. These sexually deviant behaviors have a higher chance of being fostered in an individual strongly drawn to certain stimuli, such as the infliction of pain, while in a time of sexual naïvety (Kellaher 2015).

Because of differences associated with ASD, there has been a call to reorganize and reimagine sexual education for those on the spectrum. There is a large difference between the perceived and actual sexual knowledge of individuals with ASD, who were more frequently obtaining knowledge through trial and error than through sexual education (Kellaher 2015). Corona et al. (2016) collected input from parents of children with ASD and found that many believe that the current

sexual education is not adequate. Numerous parents reported that their children still performed inappropriate sexual acts in public, such as touching their own genitals or the genitals of others, after participating in a school-based sexual education program (Corona et al. 2016). Adequate sexual education is essential for controlling these inappropriate public behaviors, improving social deficits that translate to a higher susceptibility to being a victim of abuse, and providing an environment to foster healthy sexual habits (Travers and Tincani 2010). Therefore, sexual education, especially for those with ASD, should encompass topics such as self-awareness, personal hygiene, abuse awareness, self-esteem and assertiveness, stages of romantic relationships and how to maintain them, sexual responses and decisions, partnered sex, sexual anatomy, reproductive rights and health, contraception and STIs, and parenting (Travers and Tincani 2010; Davies and Dubie 2011; Tullis and Zangrillo 2013).

Sexual education curricula for ASD individuals are few and far between. Using the framework from curricula used to educate adults with developmental delays (DD) could provide a start for such programs due to similar difficulties between those with DD and those with ASD. Sexual education curriculums must be flexible to allow for updates, incorporate current research, and adapt to the students' age and mental abilities. Additionally, sexual education not should be limited to strictly teaching the mechanical skills involved but should focus on all levels of social interaction. Sexual education not only should include understanding of sexual interactions, sexual intercourse, and contraception but should incorporate skills in using menstrual products and checking for abnormalities in the body, such as a breast or testicular self-examination. These self-care tasks may require more particular and personalized teaching methods given the absence of readily available environmental signals that many individuals with ASD rely on (Tullis and Zangrillo 2013).

A question important to the design of sexual education for people with ASD is who the most effective sexual educators are and when and where such education should occur (Sevlever

et al. 2013). Someone who is familiar with the strategies that maximize the learning of students with ASD should lead sexual instruction (Davies and Dubie 2011). However, sexual education for individuals on the spectrum should not be limited to ASD experts. Regarding residential programs, there should be systematic training of all staff on how to teach their residents to appropriately express sexual feelings in order to better aid these individuals (Van Bourgondien et al. 1997). Moreover, parents who did not partake in sexual education or who had low sexual expectations for their children were more likely to only address basic sexual topics instead of using a comprehensive approach (Holmes et al. 2016). Not addressing important sexual factors, including but not limited to contraception, sexual abuse, and partnered relationships/sex, increases the risk of victimization and publically inappropriate sexual behaviors (Koegel et al. 2014). Because of this, it is critical that parents and caregivers are provided with appropriate and accurate information and educational strategies to support the sexual development of their children with ASD (Byers et al. 2013).

Future Directions

As knowledge about autism spectrum disorders becomes more vast in the coming years – and those on the spectrum are pushed farther into adult expectations – further opportunity and pressure to seek romantic or intimate relationships will emerge. As more research is conducted regarding the sexuality associated with ASD, the aforementioned reworking of the sexual education system should be considered, as it cannot lag behind other areas of ASD education and programs. Each individual with ASD requires unique and often personalized treatment, but research and surveys could improve personalized planning and public school curriculum regarding sexual and relationship education for those on the spectrum.

Another particular direction of research encapsulated by intimacy and ASD is genetic research. People with ASD are more likely to marry and/or have children with individuals who also have

differences (Newman et al. 2011). Speculation based on the strong evidence for genetic causality of ASD suggests that children born of those who are on the spectrum may also receive a diagnosis, but further studies are required to determine if this is the case. The implications of raising children if one or both parents have ASD and if both the parents and the children have ASD should be studied.

Technological advancements and consumerist economies ensure that education is not the sole source of information in modern day. Recent studies have delved into the accessibility of sexually explicit images accessed via the internet, with half of the adolescent males with ASD sampled having discussed sex on the internet within the 6 months prior to the survey. The vast majority had viewed the images on pornographic websites (Dewinter et al. 2015). Additionally, online dating and social forums provide an impersonal and anonymous environment that individuals with ASD might thrive in. This setting could also provide a medium for victimization (Koegel et al. 2014). Therefore, the effect of media on an ASD individual's perspective on sexuality and romance should be considered and studied. Media usage and its consequences should also be incorporated into sexual education curriculums.

Moreover, the research about romantic relationships within the ASD population has largely neglected to study low-functioning individuals with ASD. A majority of the studies referenced in this paper pertained exclusively to higher-functioning individuals. Current educational advice is also more focused on the higher-functioning population (Davies and Dubie 2011) and should be generalized or modified for the rest of the ASD population. It is important to investigate current sexual education curricula to better understand what kinds of programs work best for all individuals with ASD, both high and low functioning (Tullis and Zangrillo 2013).

Despite large strides in recent research, women with ASD are still consistently underrepresented in study pools, especially with regard to intimate topics. Very few of the studies referenced here included females or equally represented them.

Navigating sexual intimacy and romance would undoubtedly be a different experience for females with ASD and needs to be studied to a further extent.

With regard to all the above topics, the perspective of those with ASD should be considered. Many individuals have a desire to share their own experience in efforts to improve life for others who share the same diagnosis (Hurlbutt and Chalmers 2002). In addition to those who study ASD, the true experts, undoubtedly, are individuals who have personally lived with ASD and through this have developed opinions on how therapies, programs, and interactions can be improved drastically. Therefore, input from individuals with ASD from various age groups should be collected and considered.

See Also

- Friendships
- Social Language Development Test

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders (DSM-5®)*. Arlington: American Psychiatric Association Publishing.
- Byers, E. S., Nichols, S., & Voyer, S. D. (2013). Challenging stereotypes: Sexual functioning of single adults with high functioning autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 43(11), 2617–2627. <https://doi.org/10.1007/s10803-013-1813-z>.
- Corona, L. L., Fox, S. A., Christodulu, K. V., & Worlock, J. A. (2016). Providing education on sexuality and relationships to adolescents with autism spectrum disorder and their parents. *Sexuality and Disability*, 34(2), 199–214. <https://doi.org/10.1007/s11195-015-9424-6>.
- Davies, C., & Dubie, M. (2011). *Intimate relationships and sexual health: A curriculum for teaching adolescents/adults with high-functioning autism spectrum disorders and other social challenges*. Shawnee Mission: AAPC Publishing.
- Dewinter, J., Vermeiren, R., Vanwesenbeeck, I., & van Nieuwenhuizen, C. (2013). Autism and normative sexual development: A narrative review. *Journal of Clinical Nursing*, 22(23–24), 3467–3483. <https://doi.org/10.1111/jocn.12397>.
- Dewinter, J., Vermeiren, R., Vanwesenbeeck, I., Lobbestael, J., & Van Nieuwenhuizen, C. (2015). Sexuality in adolescent boys with autism spectrum disorder: Self-reported behaviours and attitudes. *Journal of Autism and Developmental Disorders*, 45(3), 731–741. <https://doi.org/10.1007/s10803-014-2226-3>.
- Dewinter, J., De Graaf, H., & Begeer, S. (2017). Sexual orientation, gender identity, and romantic relationships in adolescents and adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(9), 2927–2934. <https://doi.org/10.1007/s10803-017-3199-9>.
- Fernandes, L. C., Gillberg, C. I., Cederlund, M., Hagberg, B., Gillberg, C., & Billstedt, E. (2016). Aspects of sexuality in adolescents and adults diagnosed with autism spectrum disorders in childhood. *Journal of Autism and Developmental Disorders*, 46(9), 3155–3165. <https://doi.org/10.1007/s10803-016-2855-9>.
- Holmes, L. G., Himle, M. B., & Strassberg, D. S. (2016). Parental romantic expectations and parent–child sexuality communication in autism spectrum disorders. *Autism*, 20(6), 687–699. <https://doi.org/10.1177/1362361315602371>.
- Hurlbutt, K., & Chalmers, L. (2002). Adults with autism speak out: Perceptions of their life experiences. *Focus on Autism and Other Developmental Disabilities*, 17(2), 103–111. <https://doi.org/10.1177/10883576020170020501>.
- Kellaher, D. C. (2015). Sexual behavior and autism spectrum disorders: An update and discussion. *Current Psychiatry Reports*, 17(4), 25. <https://doi.org/10.1007/s11920-015-0562-4>.
- Koegel, L. K., Detar, W. J., Fox, A., & Koegel, R. L. (2014). Romantic relationships, sexuality, and autism spectrum disorders. In *Adolescents and adults with autism spectrum disorders* (pp. 87–104). New York: Springer. https://doi.org/10.1007/978-1-4939-0506-5_5.
- Lai, M. C., Lombardo, M. V., Chakrabarti, B., & Baron-Cohen, S. (2013). Subgrouping the autism “spectrum”: Reflections on DSM-5. *PLoS Biology*, 11(4), e1001544. <https://doi.org/10.1371/journal.pbio.1001544>.
- Loomes, R., Hull, L., & Mandy, W. P. L. (2017). What is the male-to-female ratio in autism spectrum disorder? A systematic review and meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry*, 56(6), 466–474. Retrieved from <https://www.sciencedirect.com/science/article/pii/S0890856717301521>
- McNaughtan, H. (2019). *Romantic consent in emerging adults with autism (ASD)* (Doctoral dissertation). Retrieved from <https://tspace.library.utoronto.ca/handle/1807/98169>
- Newman, L., Wagner, M., Knokey, A. M., Marder, C., Nagle, K., Shaver, D., & Wei, X. (2011). *The post-high school outcomes of young adults with disabilities up to 8 years after high school: A report from the*

- National Longitudinal Transition Study-2 (NLTS2). NCSE 2011-2005. National Center for Special Education Research. Retrieved from <https://files.eric.ed.gov/fulltext/ED524044.pdf>
- Sevlever, M., Roth, M. E., & Gillis, J. M. (2013). Sexual abuse and offending in autism spectrum disorders. *Sexuality and Disability*, 31, 189–200. <https://doi.org/10.1007/s11195-013-9286-8>.
- Stokes, M., Newton, N., & Kaur, A. (2007). Stalking, and social and romantic functioning among adolescents and adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37(10), 1969–1986. <https://doi.org/10.1007/s10803-006-0344-2>.
- Strunz, S., Schermuck, C., Ballerstein, S., Ahlers, C. J., Dziobek, I., & Roepke, S. (2017). Romantic relationships and relationship satisfaction among adults with Asperger syndrome and high-functioning autism. *Journal of Clinical Psychology*, 73(1), 113–125. <https://doi.org/10.1002/jclp.22319>.
- Sullivan, A., & Caterino, L. C. (2008). Addressing the sexuality and sex education of individuals with autism spectrum disorders. *Education and Treatment of Children*, 31, 381–394. Retrieved from https://www.jstor.org/stable/42899984?seq=1#metadata_info_tab_contents
- Travers, J., & Tincani, M. (2010). Sexuality education for individuals with autism spectrum disorders: Critical issues and decision making guidelines. *Education and Training in Autism and Developmental Disabilities*, 45, 284–293. Retrieved from https://www.jstor.org/stable/23879812?seq=3#metadata_info_tab_contents
- Tullis, C. A., & Zangrillo, A. N. (2013). Sexuality education for adolescents and adults with autism spectrum disorders. *Psychology in the Schools*, 50(9), 866–875. <https://doi.org/10.1002/pits.21713>.
- Van Bourgondien, M. E., Reichle, N. C., & Palmer, A. (1997). Sexual behavior in adults with autism. *Journal of Autism and Developmental Disorders*, 27, 113–125. <https://doi.org/10.1023/A:1025883622452>.

Sexuality in Autism

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Definition

Sexuality refers to the physiological processes, behaviors, impulses, and desires associated with reproduction, the genital organs, and mating.

Historical Background

Research has studied sexuality in autism over the last 30 years. Early thinking assumed that individuals with autism were generally asexual or that they expressed their sexuality in inappropriate ways. More recently, there have been a small number of studies examining the sexual behaviors of various samples of individuals with autism or Asperger's disorder, with more attention being paid to males. However, the challenges of studying a highly private aspect of human experience in a group with limited communication skills leave us with only a beginning understanding of how individuals with autism experience their sexuality.

Current Knowledge

There is a wide variation in the sexual functioning of individuals with autism, and so generalizations must be made with care. There are people who experience full sexuality with partners, entering into marriage and having children. There are others who never have any interest in sexual activities.

That being said, research has generally found few differences in the physiology and development of sexual functions between people with autism and others. A majority of individuals with autism, both men and women, demonstrate an interest in sexuality and masturbate or seek sexual stimulation in other ways. For some, particularly individuals with greater developmental challenges, the interest in sexuality may be delayed or may never emerge, but this is the minority. While there have not been extensive clinical studies of sexual functioning, it is thought that most individuals with autism enter puberty at the expected time, evidence the physical changes associated with this development, experience arousal and climax, and are capable of reproduction. Many exhibit behavior challenges and regression during puberty as they adjust to the changes in their bodies.

However, moving beyond the physiological aspects of sexuality, differences are found in the ways individuals with autism experience

attraction and arousal. Social challenges reduce the focus on romantic bonding with others and the power of sexual taboos or regulations. Consequently, for many, sexual fantasies are less likely to focus on an erotic situation with partners, and there is a greater frequency of attractions to non-human contents such as objects, animals, cartoon characters, undergarments, body parts, or particular sensory experiences.

There is also some suggestion in the literature that individuals with autism are more likely to experience homosexuality, bisexuality, or issues related to gender identity. While there is no strong empirical evidence to support this trend, it is consistent with the idea that the social challenges of autism lead to a sexual orientation and gender identity less bound by societal dictates or rules from the family and peer group. Furthermore, with autism, sexuality can develop more independently of relationships to others. For some high functioning individuals, struggles with their sense of gender are closely tied to more general questions about their identity as a young adult – what roles they can fill, how they should act, and what types of relationships they should form.

Challenges to sexual relationships: When trying to engage in sexual activities with partners, individuals with autism encounter several sets of obstacles. Their challenges with social skills, reduced social motivation, and problems following the “hidden curriculum” of social conventions often leave them isolated or withdrawn, the target of teasing or bullying, and without friendships or a peer network. They may not have regular access to and interactions with potential partners. Furthermore, competent courtship or sexual play requires subtle (verbal and nonverbal) communication, knowledge of complex social rules, and well-developed sensory processing, all areas of challenge for most people with autism. Many males experience extreme frustration that they cannot find partners for romantic or sexual relationships. For some of the highest functioning individuals, they are able to maintain friendships with women and to regularly socialize with other adults, yet do not have the skills to effectively engage in the behaviors needed to start a sexual relationship. In our culture, women often have

less difficulty finding partners who are seeking sexual interactions but may encounter a succession of liaisons that only include sexual activities without the establishment of a romantic or committed relationship. With their limited social judgment, some women with autism are vulnerable to engaging with serial partners in a dangerous and promiscuous manner.

Even when individuals with autism are able to find a partner, their experience of sexuality can be out of synch with the relationship. They may not enjoy the sensory aspects of lovemaking, may have difficulty focusing on the pleasure of their partner, or may engage in behaviors not affirming to their partner. The literature written by individuals with autism highlights the challenges of marriage or long-term sexual relationships.

Problems with sexual behaviors: While some individuals with autism experience pleasurable and satisfying sexual activities (autoerotic and with partners) that conform to the rules and expectations of the culture, there are a number of difficulties cited in the literature. One of the greatest concerns of families and caretakers is repetitive, compulsive masturbation for extended periods of time. In some cases, this may entail finding erotic materials on the Internet or calling sexual phone services. With their limited judgment and poor understanding of the implications of their actions, individuals may run up large bills or fall prey to computer viruses. An excessive interest in self-pleasuring sexual activities is most common when the individual is understimulated without the availability of other recreational activities or entertainment (such as for individuals in an institutional setting). Usually, providing alternative leisure activities and teaching them the skills to take advantage of such activities leads to a reduction in sexual self-stimulation.

Difficulties with not being able to effectively stimulate oneself to climax can cause problems. Poor technique can lead to hypermasturbation, or repetitive, nonorgasmic self-stimulation, as the individual does not experience the release and resolution of orgasm. In rare cases, individuals engage in atypical masturbatory activities with objects or materials that lead to self-mutilation or injury. There are also many cases where

psychotropic medication impacts sexual arousal or the ability to reach climax, and this can interfere with satisfying sexual practices.

Another key area of challenge relates to a lack of modesty. People with autism may not experience discomfort in front of other people when undressed, engaging in self-stimulation, looking at sexual materials, or talking about sexual topics. For some, self-stimulating sexual behaviors may occur compulsively in certain situations or in response to particular sensory experiences, even when they are in a public setting. Additionally, touching the genital area or other sexual gestures may be exhibited as part of a stereotypy without sexual stimulation being experienced. Whatever the motivation, these sexual behaviors in public lead to alienation of peers and others, trouble with school and community authorities, and embarrassment for family members.

Sexual impulses can also lead to inappropriate contact with strangers or people in the community. Some individuals become attracted to and stimulated by women's hair, legs, or other body parts. They may stare at people with the desired attribute or even attempt to touch them. Males with autism, accustomed to getting hugs from teachers and other caretakers when younger, may seek out hugs from women as an adolescent or adult and become noticeably aroused by such contact. Some of these behaviors may be more driven by sensory seeking than by sexual arousal, but they appear to be sexual violations and are responded to as such, leaving the individual vulnerable to extreme sanctions.

Sexual behaviors performed by individuals with autism in public are often misunderstood to be paraphilias or sexual disorders. They can be viewed as exhibitionistic when they disrobe, even though they are undressing because of sensory issues and a lack of understanding of social rules around nudity, rather than because it is sexually arousing. Their focus on objects or body parts can be described as fetishistic. An interest in looking at images of children can be seen to be pedophilic. While there are high functioning individuals with autism who present true paraphilias, careful assessment of these behaviors indicates that they are more frequently the product of

sensory processing issues, a lack of understanding of social rules, and deficits in perspective taking. As with any inappropriate sexual conduct, they require a strong response that will prevent their occurrence. Yet, unlike true paraphilias, when these behaviors are based in autistic functioning, they are responsive to education and behavioral intervention.

In severe cases, sexual interests and practices can lead to criminal behaviors such as stealing objects for sexual stimulation (e.g., women's undergarments), illegally using credit cards to access pornography, or making inappropriate sexual advances. Staring at or touching strangers in public can be understood to be stalking or sexual assault. There is a particularly troubling trend of male individuals, searching for erotic materials on the Internet, visiting child pornography sites. These individuals may have no capacity or desire to prey on children yet get caught and arrested when detected by law authorities. When sexual behaviors are viewed to be criminal, individuals have to deal with a justice system that often does not understand autism and can deal harshly and destructively with individuals.

In all these cases, thorough assessment of the pattern of behavior is needed. For example, in one case, a young man was making detailed, sexually threatening comments to women using seemingly precise jargon and terminology to describe what he intended to do. A comprehensive evaluation of his pattern of arousal and sexual knowledge revealed that he knew very little about sexuality and experienced no sexual arousal. He was just mimicking phrases he had heard and was reinforced by the reaction he received.

A final challenge related to sexuality is the vulnerability of people with autism to predatory individuals. With their difficulties with perspective taking, understanding social rules, judging intent, and anticipating outcomes, individuals are easy targets for people who are sexually abusive, assaultive, or predatory. This is particularly true for females. It is important to provide education and necessary levels of supervision to prevent victimization.

Facilitating sexual development: Building a positive sexual adjustment in people with autism

requires careful monitoring, individualized sex education, and behavioral intervention when problems occur. Caregivers need to consider all the situations confronting the individual and ensure that they will not be vulnerable to predators, exposed to sexual materials, or likely to act inappropriately. Access to the Internet or phone lines should be controlled. The amount of hugs and physical contact permitted with teachers and caregivers should be adjusted and eliminated when the child is in preadolescence. As the child develops and is more able to be independent, it is essential to teach him the skills he will need to handle potential sexual impulses or threats and remain safe and appropriate.

Effective sex education is the best way to prevent sexual problems. It should be provided as soon as the child exhibits sexual interest or behaviors and reviewed and revisited on a regular schedule. The information should be geared to the individual's needs and his ability to understand. For the people with the greatest level of challenge, this instruction might just focus on rules about acceptable sexual behaviors in public and in private, and on appropriate and inappropriate touching. It may be necessary as well to teach the individual effective ways to stimulate himself/herself and define where and when this can occur. With higher functioning people who are seeking sexual partners, education should cover more detailed information about sexual functioning, sexually transmitted diseases, pregnancy, and sexual abuse/assault. Depending on the level of understanding, there should also be instruction about dating relationships and the process of courtship. Teaching should be delivered in a way that is effective for people on the autism spectrum. This includes emphasizing rote instruction, using visual context cues and materials, providing rehearsal with scaffolded supports, fading prompts as they are no longer needed, and including generalization training. Teaching techniques known to be effective with individuals with autism, such as social stories, rule cards, priming, and social autopsies, should be utilized to ensure effective instruction.

When sexual problems occur, careful assessment is warranted. For many difficult situations, additional education may be all that is needed.

When problems persist, the incidents should be tracked and studied through a functional behavior analysis in order to identify triggers and situations that must be avoided, to teach needed skills and alternative responses to the inappropriate conduct, and to ensure that the target behaviors are not being inadvertently reinforced. As noted above, designing the most effective response may require careful assessment of the individual's sexual knowledge and pattern of arousal, sensory processing factors, and other secondary factors.

Future Directions

Research into sexuality and autism is just beginning to systematically assess the sexual functioning, development, interests, and behaviors of people on the autism spectrum. This work should be expanded to larger, more representative samples and should examine differences across gender, age, cognitive ability, level of autism, and other key variables. Particular attention should be paid to problematic sexual conduct/victimization and ways to prevent and effectively treat it. There is a developing literature on sexual education for people with autism, and this needs to expand to provide families and schools access to effective teaching materials that apply to a wide range of individuals. At the level of social and behavioral research, we need to better understand how high functioning individuals with autism can remain more integrated with peer groups and develop the skills needed for dating, courtship, and maintenance of sexual relationships.

See Also

► [Daily Living Skills](#)

References and Reading

- Helleman, H., Colson, K., Verbraeken, C., Vermeiren, R., & Deboutte, D. (2007). Sexual behavior in high-functioning male adolescents and young adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37, 260–269.

- Hellemans, H., Roeyers, H., Leplae, W., Dewaele, T., & Deboutte, D. (2010). Sexual behavior in male adolescents and young adults with autism spectrum disorder and borderline/mild mental retardation. *Sexuality and Disability*, 28, 93–104.
- Henault, I. (2006). *Asperger's syndrome and sexuality: From adolescence through adulthood*. London: Jessica Kingsley.
- Koller, R. (2000). Sexuality and adolescents with autism. *Sexuality and Disability*, 18(2), 125–135.
- Konstantareas, M. M., & Lunsy, Y. J. (1997). Sociosexual knowledge, experience, attitudes, and interests in individuals with autistic disorder and developmental delay. *Journal of Autism and Developmental Disorders*, 27(4), 397–413.
- Lawson, W., & Jones, G. (2005). *Sex, sexuality and the autism spectrum*. London: Jessica Kingsley.
- Newport, J., & Newport, M. (2002). *Autism-Asperger's and sexuality: Puberty and beyond*. Arlington: Future Horizons.
- Ousley, O. Y., & Mesibov, G. B. (1991). Sexual attitudes and knowledge of high-functioning adolescents and adults with autism. *Journal of Autism and Developmental Disorders*, 21, 471–481.
- Sullivan, A., & Caterino, L. (2008). Addressing the sexuality and sex education of individuals with autism spectrum disorders. *Education and Treatment of Children*, 31(3), 381–394.
- Tarnai, B., & Wolfe, P. (2008). Social stories for sexuality education for persons with autism/pervasive developmental disorder. *Sexuality and Disability*, 26, 29–36.

SHANK 3

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Synonyms

DEL22q13.3 (Entrez Gene, OMIM, Uniprot); KIAA1650; Proline-rich synapse-associated protein 2; PROSAP2; PSAP2; SCZD15; SPANK-2

Definition

SHANK3, the gene encoding the protein *SH3 and multiple ankyrin repeat domains 3*, belongs to the SHANK family of genes; the other members are SHANK1 and SHANK2. The members of the

SHANK family of genes are scaffolding proteins, comprising multiple domains, that are found in the postsynaptic density and connect membrane proteins, such as neurotransmitter receptors (e.g., NMDA-type and metabotropic glutamate) and ion channels, to the actin cytoskeleton and to intracellular signaling pathways. They are actively involved in synapse formation and dendritic spine development (EntrezGene, Uniprot, Durand et al. 2012). SHANK3 is located on chromosome 22.q13.3 and the canonical verified isoform consists of 1741 amino acids, with at least one other isoform (Uniprot).

SHANK3 is the site of a recurrent chromosomal breakpoint within 22q13.3 (Bonaglia et al. 2006), leading to a 22q13.3 deletion syndrome. Deletions and other disruptions in 22q13.3 were subsequently linked strongly to autism (Durand et al. 2007). Recent clinical reviews have stated that 22q13 abnormalities are estimated to be involved in roughly 1% of all autism diagnoses (Abrahams and Geschwind 2008; Buxbaum 2009). Functional studies also demonstrate the severe effects of SHANK3 disruption; mutations in SHANK3 were recently shown to affect dendritic spine development (Durand et al. 2012). A mouse SHANK3 knock-out (in which both copies of a gene have been removed) also demonstrated compulsive and repetitive behavior and social impairments, reminiscent of an autism phenotype (Peça et al. 2011). SHANK3 is one of a handful of genes thought to be conclusively associated with ASD. It has also recently been linked to schizophrenia (Gauthier et al. 2010).

See Also

► Synaptic Proteins

References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, 9(5), 341–355.
- Bonaglia, M. C., Giorda, R., Mani, E., et al. (2006). Identification of a recurrent breakpoint within the SHANK3

- gene in the 22q13.3 deletion syndrome. *Journal of Medical Genetics*, 43(10), 822–828.
- Buxbaum, J. D. (2009). Multiple rare variants in the etiology of autism spectrum disorders. *Dialogues in Clinical Neuroscience*, 11(1), 35–43.
- Durand, C. M., Betancur, C., Boeckers, T. M., et al. (2007). Mutations in the gene encoding the synaptic scaffolding protein SHANK3 are associated with autism spectrum disorders. *Nature Genetics*, 39(1), 25–27.
- Durand, C. M., Perroy, J., Loll, F., et al. (2012). SHANK3 mutations identified in autism lead to modification of dendritic spine morphology via an actin-dependent mechanism. *Molecular Psychiatry*, 17(1), 71–84.
- EntrezGene. <http://www.ncbi.nlm.nih.gov/gene>
- Gauthier, J., Champagne, N., Lafreniere, R. G., et al. (2010). De novo mutations in the gene encoding the synaptic scaffolding protein SHANK3 in patients ascertained for schizophrenia. *Proceedings of the National Academy of Sciences of the United States of America*, 107(17), 7863–7868.
- OMIM (Online Mendelian Inheritance in Man). <http://www.omim.org/>
- Peça, J., Feliciano, C., Ting, J. T., et al. (2011). Shank3 mutant mice display autistic-like behaviours and striatal dysfunction. *Nature*, 472(7344), 437–442.

SHANK3

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Synonyms

ProSAP2

Definition

Shank is a super scaffolding protein located at the glutamatergic synapses. Through interacting with other synaptic proteins, Shank plays a key role in orchestrating signalling molecules and spine morphology at the post-synaptic density. Shank protein is encoded by Shank 1, Shank 2, and Shank 3 genes. It consists of five important domains, including N-terminal ankyrin repeats, Src homology domain3 (SH3), PSD-95/Dlg/ZO-1 domain (PDZ domain), proline-rich region, and C-terminal sterile alpha motive. Meta-analysis shows that up to 1% of autism spectrum disorder

(ASD) patients have mutations in Shank genes. In particular, Shank 3 has been heavily studied. Patients with Shank 3 mutation are associated with more profound cognitive impairment, and it has been proposed that Shank 3 as the target of genetic screening in ASD. Shank 3 partial gene-deletion or gene-knockout rodents show abnormal social communication, repetitive rituals, and self-injurious behavior. Long-term potentiation, a process that is believed as the physiological basis of learning and memory, are impaired in these animals. Taken together, perturbation of Shank protein may lead to synaptic dysfunction, which has been proposed as the common mechanism among ASD heterogeneous etiologies.

See Also

► [Glutamate](#)

References and Reading

- Leblond, C. S., Nava, C., Polge, A., Gauthier, J., Huguet, G., Lumbroso, S., & Bourgeron, T. (2014). Meta-analysis of SHANK Mutations in Autism Spectrum Disorders: A gradient of severity in cognitive impairments. *PLoS Genetics*, 10(9), e1004580.
- Monteiro, P., & Feng, G. (2017). SHANK proteins: Roles at the synapse and in autism spectrum disorder. *Nature Reviews. Neuroscience*, 18(3), 147–157.
- Wang, X., McCoy, P. A., Rodriguez, R. M., Pan, Y., Je, H. S., Roberts, A. C., & Jiang, Y. H. (2011). Synaptic dysfunction and abnormal behaviors in mice lacking major isoforms of Shank3. *Human Molecular Genetics*, 20(15), 3093–3108.

Shaping of Behavior

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Definition

Shaping of behavior is a behavior modification technique that rewards successive approximations

to a desired target behavior. For individuals with autism, shaping can be used to teach new behaviors. The procedure involves defining what the target behavior should be and then choosing which initial response to reinforce. The initial response to reward should be something that the individual exhibits that is related to the target behavior. Approximations to the desired behavior are differentially reinforced as the shaping procedure is implemented. Each successive approximation that is closer to the target behavior is reinforced, while reinforcement is withheld for earlier approximations in order to continue moving toward the target behavior. For example, shaping was initially used to teach children with autism expressive words. Rather than only providing a reward when a desired word was produced, differential speech attempts that become closer and closer to the target word were rewarded in order to shape the target word and increase the individual's motivation to speak. Newer approaches found that shaping usually is not necessary for producing speech in nonverbal children, and reinforcing verbal attempts may be more effective (Koegel and Koegel 2012).

See Also

- Behavior Modification
- Differential Reinforcement
- Reinforcement

References and Reading

- Koegel, R. L., & Koegel, L. K. (2012). *The PRT pocket guide*. Baltimore: Paul H. Brookes Publishing.
- Koegel, R. L., O'Dell, M., & Dunlap, G. (1988). Producing speech use in nonverbal autistic children by reinforcing attempts. *Journal of Autism and Developmental Disorders*, 18(4), 525–538.
- Malott, R. W. (2008). *Principles of behavior* (6th ed.). Englewood Cliffs: Pearson Prentice Hall.
- Skinner, B. F. (1975). The shaping of phylogenetic behavior. *Journal of the Experimental Analysis of Behavior*, 24, 117–120.

Shared Book Reading with Preschoolers with Autism

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Definition

Shared book reading, also referred to as “interactive book reading,” includes a variety of strategies that adults use when reading to children. Shared reading activities are commonly conducted in small groups or in 1:1 arrangement. During shared reading, adults use a variety of strategies to actively engage young children in the act of reading and storytelling to promote children's language and early literacy skills. Systematic reviews of the evidence base on shared reading interventions reveal potentially positive effects on phonological processing and early reading/writing skills with mixed effects on comprehension and language development (US Department of Education). The majority of studies included in the evidence base for shared reading interventions involve typically developing children and children from at-risk populations. Within the past decade, researchers have extended the application of shared reading interventions to children with disabilities, specifically those with autism spectrum disorder (ASD).

Historical Background

Reading Readiness Versus Emergent Literacy

Reading instruction for children with developmental disabilities has historically been absent from educational programming or severely lacking in quality (e.g., sole focus on sight word instruction; Spector 2011). Excluding individuals with severe disabilities from reading instruction was justified according to the reading readiness approach to learning which suggested that

students' readiness to learn and their physical status were fundamental to beginning literacy instruction. Fundamental factors believed to be responsible for school failure or success included physical readiness (visual acuity, hearing, intellectual readiness (measured by IQ and "reading readiness" tests) and personal readiness indicators such as social maturity, language readiness skills, and perceptual readiness (Williams 1953). According to the reading readiness model, many children with developmental disabilities would not receive literacy instruction on the basis that they were not "ready," and formal reading instruction was not included as part of the educational plan. Within the reading readiness paradigm, literacy development fell under the domain of formal schooling; parents and early childhood educators had little involvement or influence in children's reading development. Work initiated by Marie Clay (e.g., Clay 1975) challenged this paradigm by arguing that children's early experiences with informal literacy activities support reading development. Whereas the reading readiness approach creates a clear distinction between pre-reading and reading, the emergent literacy paradigm conceptualizes literacy acquisition as a continuum beginning at birth. That is, children's literacy development begins long before children start formal instruction in elementary school. Young children gain a number of foundational skills that support future reading achievement through their informal interactions with literacy materials even though they are not able to read or write in the conventional sense (Whitehurst and Lonigan 1998). Accordingly, researchers studying emergent literacy have focused on understanding how children's environments and experiences support the development of skills, knowledge, and attitudes related to reading development.

Adults play an instrumental role in supporting young children's early literacy skill development by creating literacy-rich environments. The creation of supportive literacy environments includes (a) adult-directed activities that expose children to literacy, such as reading to children, (b) opportunities for children to independently explore books and writing instruments, and

(c) structuring the setting in a manner that allows both exposure and access to these aforementioned opportunities (e.g., displaying environmental print, creating book areas, writing centers; Smith and Dickenson 2002). Literacy-rich early childhood environments give young learners ample opportunities to engage in meaningful literacy activities. By engaging in literacy activities, by observing others engage in literacy activities, and through direct teaching, children gradually develop knowledge about language, print, and literacy (Teale and Sulzby 1986). It is now understood that reading, writing, and oral language develop in a complementary way. Adults play an important role in interacting with children and structuring their environment in ways that can help them learn these skills long before they begin formal schooling.

Literacy Development in ASD

The rate of reading improvement for individuals with autism spectrum disorder (ASD) is significantly slower than that of students with learning disabilities (Wei et al. 2011). The majority of literacy research that has been conducted with this population is focused on conventional reading skills, such as decoding, fluency, and comprehension (Huemer and Mann 2010; Mayes and Calhoun 2003; Nation et al. 2006). Although the research on early literacy development for children with ASD is rather limited (Westerveld et al. 2016), findings from emerging research suggest that many children with ASD demonstrate early signs of reading difficulty during the preschool years (Fleury and Lease 2018; Westerveld et al. 2017). Westerveld et al. (2017) found that 40–75% of preschool children with ASD performed within expected range in code-focused emergent literacy skills (i.e., alphabet knowledge, print knowledge, phonological awareness) compared to only 15% scoring within the expected range for meaning-focused emergent literacy skills (i.e., vocabulary knowledge, listening comprehension). Upon closer examination of specific code-focused component skills, children with ASD are particularly strong in one particular aspect of code-focused skills: alphabet knowledge. Multiple research groups have replicated

this finding of alphabet knowledge being a relative strength in young children with ASD (Davidson and Ellis Weismer 2014; Lanter et al. 2012; Westerveld et al. 2017). Other aspects of code-focused skills – print concept knowledge and phonological awareness – remain relatively weak for this group of children (Davidson and Ellis Weismer 2014; Dynia et al. 2014; Lanter et al. 2012). The marked variability of emergent literacy performance across studies is particularly noteworthy and appears to relate to language ability, nonverbal cognition, and autism symptom severity (Dynia et al. 2014; Westerveld et al. 2017).

Generally, children with ASD have considerably greater difficulty acquiring meaning-focused skills – the extent to which children understand language. Weakness in the ability to draw meaning from literacy and language is a consistent finding across studies examining emergent literacy profiles for young children with ASD (Davidson and Ellis Weismer 2014; Westerveld et al. 2017). This may not be surprising given the strong correlation between language ability and reading comprehension (Huemer and Mann 2010; Mayes and Calhoun 2003; Nation et al. 2006). It is well established that children's vocabulary knowledge as well as the ability to comprehend and produce language in discourse contributes to reading achievement (Justice and Sofka 2010; Lucas and Norbury 2018). Delayed verbal language is most commonly the first concern reported by caregivers of young children with ASD (Ozonoff et al. 2010). Most young children with ASD develop language later than their typically developing peers (Tager-Flusberg et al. 2005), and although it is currently estimated that between 75% and 90% of children with ASD eventually develop speech (Rogers 2006), they continue to struggle with pragmatics, often referred to as the social aspects of language (Leekam 2007; Tager-Flusberg and Joseph 2003). Difficulties in both language development and the broader domain of social communication have a negative impact on reading comprehension (Lucas and Norbury 2018). Given that social communication problems are a core feature of ASD, it is likely

that many children with ASD will experience reading comprehension failure when presented with school activities in which they are expected to read for meaning.

Shared Book Reading Activities

Experts advocate that early childhood special education programs include emergent literacy instruction as part of the curriculum (Carta et al. 2015) to support children's future reading achievement. A common literacy practice valued by educators and early childhood professionals is reading aloud to children. Reading books to children gained popularity during the 1980s in large part because early childhood policy makers publicized that reading aloud was an excellent, developmentally appropriate instructional practice (Bredenkamp 1986) and several research efforts focused on the topic of parent-child book reading in the home environment (e.g., Heath 1982; Teale and Sulzby 1986). Reading aloud to children emerged as a key facet of family literacy programs and the central focus of several public library outreach efforts (e.g., the Carnegie Library's Beginning to Read program, Reading is Fundamental, Washington Learning Systems [see Segel and Friedberg 1991; <http://www.rif.org/>; <http://www.walearnin.com>]). As a result of these efforts, adult-child shared reading practices (a parent reading a picture book with a toddler or a teacher reading a book to a class of preschoolers) gained popularity and continue to be widely recommended to promote language and other skills related to early literacy development.

The benefits of adult-child shared reading practices on children's language development are based on the premise that book reading allows children to hear adults use rich language and children are then provided ample opportunities to practice using language. Research conducted in the past few decades has stressed that the quality of book reading is more important than the quantity of book reading (e.g., Scarborough and Dobrich 1994). The manner in which children are read to affects their engagement during book readings and what they take away from the experience. A situation in which an adult simply

reads the text on the page positions the child as a passive listener, rather than an active participant in the reading. Compare this to a book reading interaction that involves the adult describing illustrations and asking the child questions about what he or she sees and understands about the story. In this second scenario, the child has the opportunity to and is expected to engage with the text. The child becomes an active participant with the adult, the book, and the entire process of reading. Through quality adult-child shared reading interactions, children not only gain access to a variety of books but are also provided opportunities to develop a number of emergent literacy skills, specifically oral language.

Current Knowledge

Children with ASD may have difficulty fully participating in shared book reading due to deficits in social communication that are characteristic of the disorder (Fleury and Schwartz 2017; Kluth and Chandler-Olcott 2008). Shared book reading requires that children are able to sustain social interaction regarding a particular topic (the book), including asking questions or making comments about what they notice, which can be challenging for many children with ASD due to their difficulties with social communication (Dawson et al. 2004; Tager-Flusberg and Joseph 2003). An important, but less obvious, difficulty that underlies social communication is joint attention, the visual sharing of attention with a social partner in reference to an object of event of mutual interest (Carpenter et al. 1998). Young children with ASD demonstrate lower levels of joint attention compared to typically developing children and age-matched peers with Down syndrome (Adamson et al. 2004). Because the design of many literacy activities relies heavily on social and communication ability, children who lack basic skills in these areas will have difficulty participating in, and benefiting from, these activities without instructional support (Kluth and Chandler-Olcott 2008).

Home Literacy Environment

Shared book reading during the emergent literacy stage of reading development can occur both in the home and at school. In the home, shared book reading frequency is an important aspect of the home literacy environment. Two studies have examined the home literacy environment of preschoolers with ASD. Lanter et al. (2012) found that caregivers of 41 preschool-aged children with ASD engaged in frequent book reading with their child starting at a young age. However, Dynia et al. (2014) found that 35 children with ASD participated in shared book reading less frequently than a matched sample of 35 typically developing children. Further, more frequent shared book reading was related to higher alphabet knowledge for both the children with ASD and their peers. One study that included 41 school-aged children with ASD found that children with ASD and concomitant language disorder participated in more frequent shared book reading than children with ASD and age-appropriate language skills and children who were typically developing; however, the shared book reading sessions were shorter in duration (Lucas and Norbury 2018).

Engagement During Shared Book Reading

Another set of studies has focused on children with ASD's behavior and engagement during shared book reading. When children with ASD participate in shared reading, they attend to the activity for less time compared to their peers (Watson et al. 1996). For instance, Thompson et al. (2018) measured the eye-tracking behavior of ten minimally verbal children with ASD aged 5–8 years old while reading an e-book and compared this eye-tracking performance with children who were typically developing. The children with ASD only attended to the e-book for approximately half of the time that it was presented and when they did attend to the e-book, they were minimally attentive to the literacy concepts (e.g., print). Similarly, Fleury and Hugh (2018) found that preschoolers with ASD ($n = 17$) looked at the book less and demonstrated more non-engaged behaviors during shared book reading with their caregivers than

their typically developing peers. Finally, one study by Bean et al. (2019) examined 22 children with ASD's book reading orientation during a whole-group book reading in a preschool classroom and found that children with ASD were less engaged during whole-group book reading than children with developmental language delay and their typically developing peers. Further, book reading orientation was found to be a significant predictor of gains in emergent literacy skills.

Shared Book Reading Interventions

There have also been several examinations into shared book reading interventions with preschool-age children with ASD. One series of studies has examined the Reading to Engage Children with Autism in Language and Learning (RECALL) dialogic reading intervention for children with ASD (Whalon et al. 2013). RECALL combines dialogic reading strategies and behavior supports for children with ASD (e.g., time delay, direct models, visual supports). Whalon et al. (2015) examined RECALL led by an interventionist with a pair of children (one child with ASD and a participating typically developing peer). Participation in RECALL, which was designed to facilitate joint attention during shared book reading, prompted more correct responses to fact and inference-based questions about the text and resulted in increased reading comprehension for the children with ASD. Parent-implemented RECALL has also been examined in a systematic case study with caregiver-child dyad (Whalon et al. 2016). Results indicated that the caregiver could implement RECALL with integrity and the child with ASD increased their spontaneous correct responding. Similarly, in a study by Fleury et al. (2014), the researcher engaged in one-on-one book reading sessions using a variety of dialogic reading question prompts (e.g., recall, open-ended questions, wh-questions) during shared book reading. Results indicated that all three children with ASD (ages 4–5 years old) participated in longer book reading sessions during the dialogic reading intervention sessions. In another study by Fleury and Schwartz (2017), paraeducators implemented one-on-one book

reading sessions with nine preschoolers with ASD. During these sessions, the children had higher levels of participation during dialogic intervention sessions as well as increases in book-specific vocabulary. The effect of a dialogic reading intervention using technology was examined for four preschoolers with ASD (Grygas Coogle et al. 2018). Results indicated that dialogic reading, using traditional books and technology-enhanced books, was a promising intervention for increasing the vocabulary skills of children with ASD. In a large study on the effects of different emergent literacy interventions, Hudson et al. (2017) examined the effects of an interactive book reading intervention in comparison to a phonological awareness intervention for 133 preschoolers with ASD. Results indicated that children with ASD had significantly greater gains in vocabulary and listening comprehension after participation in the interactive book reading intervention compared to the phonological awareness intervention and business as usual.

Finally, Boyle et al. (2019) completed a meta-analysis of 11 shared book reading interventions for children with ASD. The interventions in the meta-analysis were implemented with preschool- and school-age children with ASD. The six preschool interventions included studies reviewed above (Fleury et al. 2014; Fleury and Schwartz 2017; Whalon et al. 2015; Whalon et al. 2016) and two unpublished dissertations (Vogler-Elias 2009; Zimmer 2013). Results from the meta-analysis showed that shared book reading has positive effects on expressive language and listening comprehension for children with ASD.

Future Directions

Given that the findings on the frequency of shared book reading in the home by children with ASD is mixed, more studies are needed on the types and frequency of literacy activities that children with ASD are experiencing in their homes. In addition, more research is needed on whether the home literacy environment is related to

children with ASD's emergent literacy skills. Further, based on eye-tracking and observational measures, children with ASD seem to be less engaged during shared book reading than their typically developing peers. However, despite the mixed findings on shared book reading frequency and the limited engagement displayed during shared book reading, shared book reading interventions have found positive effects for children with ASD. This suggests that traditional measures of engagement may not be adequately capturing what it means to be engaged for children with ASD. Future studies should examine alternate ways of assessing engagement during shared book reading for children with ASD. As for shared book reading interventions, most of the extant literature is based on studies using single-subject design and small sample sizes. Future research should aim to include larger group design studies including rigorous randomized controlled trials.

See Also

- Academic Skills
- Emergent Literacy
- Literacy

References and Reading

- Adamson, L. B., Bakeman, R., & Deckner, D. F. (2004). The development of symbol-infused joint engagement. *Child Development*, 75(4), 1171–1187. Retrieved from <http://www.jstor.org/stable/3696533>.
- Bean, A., Perez, B., Dynia, J. M., Justice, L. M., & Kaderavek, J. (2019). Book-reading engagement in children with autism and language impairment: Associations with emergent-literacy skills. *Journal of Autism and Developmental Disorders*, Manuscript under review.
- Boyle, S. A., Mcnaughton, D., & Chapin, S. E. (2019). Effects of shared reading on the early language and literacy skills of children with autism spectrum disorders: A systematic review. *Focus on Autism and Other Developmental Disabilities*, 34(4), 205–214. <https://doi.org/10.1177/1088357619838276>.
- Bredenkamp, S. (1986). The reliability and validity of the early childhood classroom observation scale for accrediting early childhood programs. *Early Childhood Research Quarterly*, 1(2), 103–118.
- Carpenter, M., Nagell, K., Tomasello, M., Butterworth, G., & Moore, C. (1998). Social cognition, joint attention, and communicative competence from 9 to 15 months of age. *Monographs of the Society for Research in Child Development*, 63, i–174.
- Carta, J. J., Greenwood, C. R., Atwater, J., Goldstein, H., Kaminski, R. A., & McConnell, S. R. (2015). Identifying preschool children for higher tiers of language and early literacy instruction within a response to intervention framework. *Journal of Early Intervention*, 36(4), 281–291. <https://doi.org/10.1177/1053815115579937>.
- Clay, M. (1975). *What did I write?* Auckland: Heinemann.
- Davidson, M. M., & Ellis Weismer, S. (2014). Characterization and prediction of early reading abilities in children on the autism spectrum. *Journal of Autism and Developmental Disorders*, 24(4), 828–845. <https://doi.org/10.1007/s10803-013-1936-2>.
- Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., & Liaw, J. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology*, 40(2), 271.
- Dynia, J. M., Lawton, K., Logan, J. A., & Justice, L. M. (2014). Comparing emergent-literacy skills and home-literacy environment of children with autism and their peers. *Topics in Early Childhood Special Education*, 34(3), 142–153. <https://doi.org/10.1177/0271121414536784>.
- Fleury, V. P., & Hugh, M. L. (2018). Exploring engagement in shared reading activities between children with autism spectrum disorder and their caregivers. *Journal of Autism and Developmental Disorders*, 48(10), 3596–3607. <https://doi.org/10.1007/s10803-018-3632-8>.
- Fleury, V. P., & Lease, E. M. (2018). Early indication of Reading difficulty: A descriptive analysis of emergent literacy skills in children with autism spectrum disorder. *Topics in Early Childhood Special Education*, 34, 142–153. <https://doi.org/10.1177/0271121417751626>.
- Fleury, V. P., & Schwartz, I. S. (2017). A modified dialogic reading intervention for preschool children with autism spectrum disorder. *Topics in Early Childhood Special Education*, 37(1), 16–28. <https://doi.org/10.1177/0271121416637597>.
- Fleury, V. P., Miramontez, S. H., Hudson, R. F., & Schwartz, I. S. (2014). Promoting active participation in book reading for preschoolers with autism spectrum disorder: A preliminary study. *Child Language Teaching and Therapy*, 30(3), 273–288. <https://doi.org/10.1177/0265659013514069>.
- Grygas Coogle, C., Floyd, K. K., & Rahn, N. L. (2018). Dialogic reading and adapted dialogic reading with preschoolers with autism spectrum disorder. *Journal of Early Intervention*, 40(4), 363–379. <https://doi.org/10.1177/1053815118797887>.
- Heath, S. B. (1982). What no bedtime story means: Narrative skills at home and school. *Language in Society*, 11(1), 49–76.

- Hudson, R. F., Sanders, E. A., Greenway, R., Xie, S., Smith, M., Gasamis, C., . . . Hackett, J. (2017). Effects of emergent literacy interventions for preschoolers with autism spectrum disorder. *Exceptional Children*, 84(1), 55–75. <https://doi.org/10.1177/0014402917705855>.
- Huemer, S. V., & Mann, V. (2010). A comprehensive profile of decoding and comprehension in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(4), 485–493.
- Justice, L. M., & Sofka, A. E. (2010). *Engaging children with print: Building early literacy skills through quality read-alouds*. New York: Guilford Press.
- Kluth, P., & Chandler-Olcott, K. (2008). *A land we can share: Teaching literacy to students with autism*. Baltimore: Paul H Brookes Publishing.
- Lanter, E., Watson, L. R., Erickson, K. A., & Freeman, D. (2012). Emergent literacy in children with autism: An exploration of developmental and contextual dynamic processes. *Language, Speech, and Hearing Services in Schools*, 43(3), 308–324. [https://doi.org/10.1044/0161-1461\(2012/10-0083\)](https://doi.org/10.1044/0161-1461(2012/10-0083)).
- Leekam, S. (2007). Language comprehension difficulties in children with autism spectrum disorders. In K. Cain & J. Oakhill (Eds.), *Children's Comprehension Problems in Oral and Written Language: A Cognitive Perspective*, (pp. 104–127). New York: The Guilford Press.
- Lucas, R., & Norbury, C. F. (2018). The home literacy environment of school-aged children with autism spectrum disorders. *Journal of Research in Reading*, 41(1), 197–219. <https://doi.org/10.1111/1467-9817.12119>.
- Mayes, S., & Calhoun, S. (2003). Analysis of WISC-III, Stanford-Binet: IV, and academic achievement scores in children with autism. *Journal of Autism and Developmental Disorders*, 33, 329–339.
- Nation, K., Clarke, P., Wright, B., & Williams, C. (2006). Patterns of reading ability in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 36, 911–919. <https://doi.org/10.1007/s10803-006-0130-1>.
- Ozonoff, S., Iosif, A. M., Baguio, F., Cook, I. C., Hill, M. M., Hutman, T., . . . Young, G. S. (2010). A prospective study of the emergence of early behavioral signs of autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 49(3), 256–266.
- Rogers, S. (2006). Evidence-based interventions for language development in young children with autism. In *Social and communication development in autism spectrum disorders: Early identification, diagnosis, and intervention*. New York: Guilford.
- Scarborough, H. S., & Dobrich, W. (1994). On the efficacy of reading to preschoolers. *Developmental Review*, 14(3), 245–302.
- Segel, E., & Friedberg, J. B. (1991). "Is today Liberry day?" Community support for family literacy. *Language Arts*, 68(8), 654–657.
- Smith, M. W., & Dickenson, D. (2002). Early Language and Literacy Classroom Observation (ELLCO) Toolkit, Research Edition. Newton: Paul H. Brookes Publishing.
- Spector, J. E. (2011). Sight word instruction for students with autism: An evaluation of the evidence base. *Journal of Autism and Developmental Disorders*, 41(10), 1411–1422.
- Tager-Flusberg, H., & Joseph, R. M. (2003). Identifying neurocognitive phenotypes in autism. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, 358(1430), 303–314. <https://doi.org/10.1098/rstb.2002.1198>.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. *Handbook of Autism and Pervasive Developmental Disorders*, 1, 335–364.
- Teale, W., & Sulzby, E. (1986). *Emergent literacy: Writing and reading*. Norwood: Ablex Publishing Corporation.
- Thompson, J. L., Plavnick, J. B., & Skibbe, L. E. (2018). Eye-tracking analysis of attention to an electronic storybook for minimally verbal children with autism spectrum disorder. *The Journal of Special Education*, 53(1), 41–50. <https://doi.org/10.1177/0022466918796504>.
- Vogler-Elias, D. (2009). *A parent-implemented shared storybook reading intervention for preschoolers with autism spectrum disorders*. Available from ProQuest Dissertations & Theses A&I; ProQuest Dissertations & Theses Global. (305094109). Retrieved from <http://proxy.lib.ohio-state.edu/login?url=https://search.proquest.com/docview/305094109?accountid=9783>
- Watson L. R., Andrews, M. D., & Orovitz, J. (1996). Emergent literacy in children with autism versus typical development. Unpublished paper presented at the meeting of the American Speech-Language-Hearing Association, Seattle.
- Wei, X., Blackorby, J., & Schiller, E. (2011). Growth in reading achievement of students with disabilities, ages 7 to 17. *Exceptional Children*, 78, 89–106.
- Westerveld, M. F., Trembath, D., Shellshear, L., & Paynter, J. (2016). A systematic review of the literature on emergent literacy skills of preschool children with autism spectrum disorder. *The Journal of Special Education*. <https://doi.org/10.1177/0022466915613593>.
- Westerveld, M. F., Paynter, J., Trembath, D., Webster, A. A., Hodge, A. M., & Roberts, J. (2017). The emergent literacy skills of preschool children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(2), 424–438. <https://doi.org/10.1007/s10803-016-2964-5>.
- Whalon, K., Delano, M., & Hanline, M. F. (2013). A rationale and strategy for adapting dialogic reading for children with autism spectrum disorder: Recall. *Preventing School Failure: Alternative Education for Children and Youth*, 57(2), 93–101. <https://doi.org/10.1080/1045988X.2012.672347>.
- Whalon, K., Martinez, J. R., Shannon, D., Butcher, C., & Hanline, M. F. (2015). The impact of reading to engage children with autism in language and learning (RECALL). *Topics in Early Childhood*

- Special Education*, 35(2), 102–115. <https://doi.org/10.1177/0271121414565515>.
- Whalon, K., Hanline, M. F., & Davis, J. (2016). Parent implementation of RECALL: A systematic case study. *Education and Training in Autism and Developmental Disabilities*, 51(2), 211–220.
- Whitehurst, G. J., & Lonigan, C. J. (1998). Child development and emergent literacy. *Child Development*, 69(3), 848–872.
- Williams, G. (1953). What does research tell us about readiness for beginning reading? *The Reading Teacher*, 6, 34–40.
- Zimmer, K. (2013). *Efficacy of caregiver training to establish joint attention of children with autism*. Gainesville: University of Florida.

(e.g., “Where do you think he will look next?”). When parents scaffold children’s language during shared storybook reading, it becomes a context that can facilitate language development across all ages. Shared storybook reading interventions focus on teaching adults (i.e., parents, teachers, or other caregivers) scaffolding strategies to support language, emergent literacy, and social engagement during the shared storybook reading context. Scaffolding strategies support increased responsiveness to children’s communication attempts and engagement during shared storybook reading. The adult’s role shifts over time as the child gains skills and is able to take on a more equal role during the shared storybook reading (Kaderavek and Sulzby 1998).

Shared Storybook Reading

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Definition

Shared storybook reading is a naturalistic and interactive routine in which an adult and a child engage in dialogue and share joint attention on a storybook. During shared storybook reading, children are exposed to both oral and written language at the same time (Ezell and Justice 2005) because children both see and hear the words during the interaction. This routine promotes the development of language and social engagement (Kaderavek and Justice 2002), which are two of the core deficit areas for children with ASDs. Children are exposed to shared storybook reading early in life; many parents report reading to children by 6 months of age (Catts and Khami 1999). Many parents naturally scaffold their child’s development by gradually increasing the cognitive and language expectations during shared storybook reading to match children’s growing abilities (Catts and Khami 1999). For example, toddlers can learn to turn the pages in a book or identify new vocabulary words, whereas older children might learn to make inferences (e.g., “How do you think this boy feels?”) or predictions

Historical Background

Historically, children with disabilities have had reduced access to emergent literacy activities like shared storybook reading when they were not perceived to demonstrate signs of literacy readiness (e.g., the ability to comment about pictures in a book, the ability to answer questions during a story, the ability to sit still for an entire storybook). Children with disabilities begin their school careers with less than one-half the exposures to print as their typical peers (van Kleeck et al. 1997; van Kleeck and Vander Woude 2003).

Children with autism spectrum disorders may have unique risks for reduced exposure to emergent literacy activities, such as shared storybook reading. Firstly, children with ASDs are at risk for difficulty with literacy because literacy is a language-based skill (Catts and Khami 1999) and children with ASDs have difficulty acquiring language. Secondly, caregiver perceptions and practices (Koppenhaver et al. 1991) may contribute to reduced opportunity for literacy exposure. Caregivers may underestimate the abilities of children with disabilities because the children have been labeled as having delays and difficulties (Koppenhaver et al. 1991). In particular, parents of children with ASDs may feel overwhelmed by their child’s behavioral needs or resistance to social interactions. Furthermore, parents of

children with developmental disabilities often devote large portions of the day meeting educational, therapeutic, and medical needs, leaving little time for “extra” activities related to emergent literacy. It is important to recognize that parents of children with ASDs will likely require support to engage in early literacy experiences with their children.

Rationale or Underlying Theory

Children with disabilities have historically had reduced opportunity to participate in emergent literacy activities, which are the building blocks to later literacy achievement. The *interactive to independent* model of literacy development (Kaderavek and Rabidoux 2004) proposes the access to literacy for individuals with atypical communication. The model includes five levels of achievement drawn from the social interaction, participation, and situated pragmatics theories of language and literacy development. Vygotsky’s social interaction theory (Vygotsky 1978) suggests that literacy development is a product of children’s supportive social interactions surrounding literacy. Beukelman and Mirenda’s participation theory (Beukelman and Mirenda 1998) stresses the importance of removing barriers to literacy for individuals with disabilities. Finally, the situated pragmatics theory (Duchan et al. 1994) expands the definition of literacy contexts by stressing the importance of developing goals that allow children to participate in naturally occurring contexts. In the context of the *interactive to independent* model, literacy is not a static skill requiring achievement of specific predetermined criteria before moving onto more advanced steps. Instead, literacy is a dynamic skill made up of several smaller sets of subskills that can be achieved in a nonlinear fashion. The model focuses on five levels of development as children move from emergent to conventional literacy. The first level focuses on joint attention and responsiveness during early literacy interactions by helping the child maintain focus on storybooks, teaching the parent strategies to increase engagement, and increasing the duration of shared

literacy experiences. The second level focuses on balance and turn taking by helping the parent build on the child’s communicative attempts to increase the child’s communicative repertoire surrounding literacy materials. The first two levels of this model have a pragmatic focus in that they prioritize the social aspects of shared literacy experiences, such as turn taking, joint attention, and acceptance of a range of communicative behaviors. The third level of the model begins to move toward conventional literacy by helping the child to increase emergent literacy skills, such as print awareness, phonological awareness, and metalinguistic awareness. The fourth and fifth levels represent movement toward conventional independent literacy, which is defined as a complex language-based skill that involves the ability to read and write. Specifically, the fourth level focuses on achievement of conventional literacy with social support, whereas the fifth level targets the achievement of full conventional literacy without social support (i.e., independent literacy achievement).

Goals and Objectives

The long-term goal of shared storybook reading interventions is to increase literacy opportunity for children with autism spectrum disorders, thus, increasing their opportunity to participate in a literate society. The objectives of shared storybook reading interventions focus on increasing active participation during in shared storybook reading exchanges. Objectives should be child-centered, individualized, and measurable. Justice and Pence (2005) have suggested three features that are essential to effective shared storybook reading interactions: parent sensitivity and responsiveness, child engagement, and repeated reading. The first feature, parent sensitivity and responsiveness, refers to parents who allow children time to respond and show they value child contributions to the interaction. Behaviors of sensitive and responsive adults include observing, waiting, listening, and being face to face with the child. The second feature, child engagement, is facilitated by allowing the child to participate

during the storybook reading. Child participation might include labeling pictures, commenting, and asking questions. Children are more likely to be engaged when they are allowed to set the pace of the interaction. The third feature, repeated reading, allows for increased familiarity and facilitates vocabulary learning. These three features are ways for adults to intentionally scaffold language and engagement during shared storybook reading, thus, moving children to higher levels of performance within the interaction. Another tool, the Kaderavek-Sulzby Bookreading Observational Protocol (KSBOOP) (Kaderavek and Sulzby 1998), can be utilized to assess areas of strength and need during parent-child shared storybook reading exchanges, including parent scaffolding behaviors, child language attempts, and other variables that are contributing to the success of the interaction.

Treatment Participants

Shared storybook reading is an intervention context appropriate for any child, including children with autism spectrum disorders.

Treatment Procedures

Interventions using shared storybook reading focus on teaching adults scaffolding strategies to support language, emergent literacy, and social engagement. These strategies include teaching adults ways to increase responsiveness to children's communication attempts and support engagement during shared storybook reading. The adult's role shifts over time as the child gains skills and is able to take on a more equal role during the shared storybook reading (Kaderavek and Sulzby 1998). Specific strategies include ask WH questions, repeat or recast what the child says, praise and encourage, follow the child's interests or lead, ask open-ended questions, expand what the child says, use attention getters.

Often, the focus of shared storybook reading interventions is to change adult behaviors. That

said, it is important to note that some parents of children with disabilities are already responsive to their children's needs and already adjust their behavior and language accordingly. For example, Justice and Kaderavek (2003) analyzed the shared storybook interactions between 11 preschoolers with language impairment and their mothers. They found that some children with language impairment and their mothers often shared topic control and balance of contributions (although some had a more unequal experience), suggesting that interventions targeting strategies during book reading interactions should be well designed such that they do not decrease positive back-and-forth interactions that are already present in parent-child interactions. In other words, shared storybook interventions should enhance and encourage strategies that parents may already be doing as well as teach them new skills to facilitate language and social engagement.

Efficacy Information

Shared storybook reading contributes to early language and literacy development (Ezell and Justice 2005). Shared storybook reading interventions have been effective at increasing vocabulary development, social engagement, and emergent literacy awareness in at-risk groups, including children living in poverty (Whitehurst et al. 1994a, b) and preschoolers with language impairment (Crain-Thoreson and Dale 1999; Crowe et al. 2000). Clinicians, teachers, and parents were successful at learning to increase strategies to support children's language, social engagement, and emergent literacy skills during shared storybook reading.

To date, only one case study has examined the use of shared storybook reading as an intervention context for children with ASDs. Bellon et al. (2000) examined the use of scaffolding strategies during a clinician-implemented shared storybook reading intervention for a preschooler with high-functioning autism. A seven-week intervention resulted in an increase in the preschooler's spontaneous speech. This preliminary study indicated that shared storybook reading may be an effective

intervention context for young children with ASDs. Shared storybook reading has not yet been explored using a parent-implemented intervention paradigm for preschoolers with ASDs, although a number of other parent-implemented interventions have successfully been examined in children with ASDs.

Outcome Measurement

Outcome measures for shared storybook reading interventions include measures of parent behavior, child behavior, and parent-child variables. Parent focused measures include measuring changes in rate and frequency of scaffolding strategy use. Child focused measures include measuring changes in children's language (e.g., vocabulary measures, mean length of utterance) or emergent literacy skills (e.g., formal measure of phonological awareness and emergent literacy). Parent-child variables attempt to measure changes in social interaction, such as turn-taking ratios or duration of interaction.

Qualifications of Treatment Providers

Interventionists with specific expertise in shared storybook reading interventions include speech-language pathologists and educators.

See Also

► [Emergent Literacy](#)

References and Reading

- Bellon, M. L., Ogletree, B. T., & Harn, W. E. (2000). Repeated storybook reading as a language intervention for children with autism: A case study on the application of scaffolding. *Focus on Autism and Other Developmental Disabilities*, 15(1), 52–58.
- Beukelman, D. R., & Mirenda, P. (1998). *Augmentative and alternative communication: Management of severe communication disorders in children and adults* (2nd ed.). Baltimore: Paul H. Brookes.
- Catts, H., & Khami, A. (1999). *Language and reading disabilities*. Needham Heights: Allyn & Bacon.
- Crain-Thoreson, C., & Dale, P. S. (1999). Enhancing linguistic performance: Parents and teachers as book reading partners for children with language delays. *Topics in Early Childhood Special Education*, 19(1), 28–39.
- Crowe, L. K., Norris, J. A., & Hoffman, P. R. (2000). Facilitating storybook interactions between mothers and their preschoolers with language impairment. *Communication Disorders Quarterly*, 21(3), 131–146.
- Duchan, J., Sonnenmeier, R., & Hewitt, L. (Eds.). (1994). *Pragmatics: From theory to practice*. Englewood Cliffs: Prentice Hall.
- Ezell, H., & Justice, L. (2005). *Shared storybook reading: Building young children's language & emergent literacy skills*. Baltimore: Paul H. Brookes.
- Justice, L. M. (Ed.). (2006). *Clinical approaches to emergent literacy intervention*. San Diego: Plural.
- Justice, L. M., & Kaderavek, J. N. (2003). Topic controlling during shared storybook reading: Mothers and their children with language impairments. *Topics in Early Childhood Special Education*, 23(3), 137–150.
- Justice, L. M., & Pence, K. (2005). *Scaffolding with storybooks: A guide for enhancing young children's language and literacy achievement*. Newark: International Reading Association.
- Kaderavek, J., & Justice, L. M. (2002). Shared storybook reading as an intervention context: Practices and potential pitfalls. *American Journal of Speech-Language Pathology*, 11(4), 395–406.
- Kaderavek, J. N., & Ravidoux, P. (2004). Interactive to independent literacy: A model for designing literacy goals for children with atypical communication. *Reading & Writing Quarterly: Overcoming Learning Difficulties*, 20(3), 237–260.
- Kaderavek, J., & Sulzby, E. (1998). Parent-child joint book reading: An observational protocol for young children. *American Journal of Speech-Language Pathology*, 7(1), 33–47.
- Kluth, P., & Darmody-Latham, J. (2003). Beyond sight words: Literacy opportunities for students with autism. *The Reading Teacher*, 56(6), 532–535.
- Koppenhaver, D. A., Coleman, P. P., Kalman, S. L., & Yoder, D. E. (1991). The implications of emergent literacy research for children with developmental disabilities. *American Journal of Speech-Language Pathology*, 7(1), 38–44.
- van Kleeck, A. (Ed.). (2006). *Sharing books and stories to promote language and literacy*. San Diego: Plural.
- van Kleeck, A., & Vander Woude, J. (2003). Book sharing with preschoolers with language delays. In A. van Kleeck, S. A. Stahl, & E. Bauer (Eds.), *On reading to children: Parents and teachers* (pp. 58–92). Mahwah: Lawrence Erlbaum Associates.
- van Kleeck, A., Gillam, R. B., Hamilton, L., & McGrath, C. (1997). The relationship between middle-class parents' book-sharing discussion and their preschoolers' abstract language development. *Journal of Speech & Hearing Research*, 40(6), 1261–1271.

- van Kleeck, A., Stahl, S. A., & Bauer, E. B. (Eds.). (2003). *On reading books to children: Parents and teachers*. Mahwah: Lawrence Erlbaum Associates.
- Vygotsky, L. S. (1978). *Mind in society: The development of higher psychological processes*. Cambridge, MA: Harvard University Press.
- Whitehurst, G. J., Arnold, D. S., Epstein, J. N., Angell, A. L., Smith, M., & Fischel, J. E. (1994a). A picture book reading intervention in day care and home for children from low-income families. *Developmental Psychology*, 30(5), 679–689.
- Whitehurst, G. J., Epstein, J. N., Angell, A. L., Payne, A. C., Crone, D. A., & Fischel, J. E. (1994b). Outcomes of an emergent literacy intervention in head start. *Journal of Educational Psychology*, 86(4), 542–555.

Shell Shock

► Posttraumatic Stress Disorder

Sheltered Employment

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Definition

Sheltered employment refers to segregated programs designed to help individuals with disabilities who are not able to work in a competitive employment setting. Sheltered workshops, day treatment, and work activity centers are examples of sheltered employment programs. Usually, sheltered employment programs are run by private, not-for-profit organizations that receive funding through state and federal sources (Kregel and Dean 2002).

Historical Background

Sheltered employment programs can typically be categorized into two types: extended employment and transitional employment. Extended

employment placements are designed to allow the individual the opportunity to utilize his/her abilities to work and earn money in a segregated workshop setting. In transitional employment settings, the individual is provided with training in a segregated setting in order to develop skills that would be necessary to be successful in an integrated employment setting (Kregel and Dean 2002). However, as Taylor notes in 2002, only about 3.5% of individuals in sheltered employment move on to work in competitive employment settings. Taylor also notes that individuals working in a sheltered setting earn significantly less per week than their counterparts working in competitive employment.

The success rate of sheltered employment settings has often been questioned by critics who state that individuals in these settings are not provided with significant employment outcomes and are isolated which contributes to lowered expectations in the workplace and can spur negative attitudes from the rest of the community (Kregel and Dean 2002).

In response to the criticisms of sheltered employment, the U.S. Department of Education's Rehabilitation Services Administration changed the regulations that govern State Vocational Rehabilitation Programs in January of 2001. This change redefines the term "employment outcome" to mean an individual working in an integrated setting (Wehman et al. 2003). No longer would a sheltered, segregated setting be an acceptable, final employment outcome for an individual with a disability.

Current Knowledge

Since sheltered employment is no longer an acceptable final employment outcome for an individual with a disability, many individuals are placed into supported employment programs as an alternative (Kregel and Dean 2002). Individuals may still be placed into a sheltered employment settings as long as the goal is for the individual to build skills and gain experience in order to move into competitive, integrated employment.

See Also

- [Competitive Employment](#)
- [Sheltered Workshops](#)
- [Supported Employment](#)

References and Reading

- Kregel, J., & Dean, D. H. (2002). Sheltered vs. supported employment: A direct comparison of long-term earnings outcomes for individuals with cognitive disabilities. In J. Kregel, D. H. Dean, & P. Wehman (Eds.), *Achievements and challenges in employment services for people with disabilities: The longitudinal impact of workplace supports monograph*. Retrieved 29 May 2011 from <http://www.worksupport.com/resources/viewContent.cfm/151>
- Taylor, S. J. (2002, September 2). Disabled workers deserve real choices, real jobs. Retrieved from <http://www.accessiblesociety.org/topics/economics-employment/shelteredwksps.html>
- Wehman, P., Revell, W. G., & Brooke, V. (2003). Competitive employment; has it become the “first choice” yet? *Journal of Disability Policy Studies*, 14(3), 163–173.

Sheltered Workshops

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Synonyms

[Supported employment](#)

Definition

A sheltered workshop is a self-contained workplace that offers a supportive and protected work environment for people with disabilities. Sheltered workshops are funded by a combination of public vocational and rehabilitation programs and contracts from businesses. Workshops are private not-for-profit corporations. Each workshop must have a special certificate from the Department of Labor. The goal of a sheltered workshop is to provide meaningful vocational experience and job skills for

people with disabilities who cannot find work elsewhere. Employees receive payment; however, their hourly pay is usually below the market rate for similar work in private industry. The workshop may also provide such related vocational rehabilitation services as job interview training.

See Also

- [Supported Employment](#)

References and Reading

Disability.gov. https://www.disability.gov/home/about_us

Short Sensory Profile

- [Short Sensory Profile in Autism](#)

Short Sensory Profile 2

- [Short Sensory Profile in Autism](#)

Short Sensory Profile in Autism

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Synonyms

[Short Sensory Profile](#); [Short Sensory Profile 2](#); [SSP](#); [SSP-2](#)

Description

The Short Sensory Profile (SSP; McIntosh et al. 1999) is a caregiver report questionnaire used

in research and clinical settings to measure sensory processing abnormalities in children with and without autism spectrum disorder (ASD). The SSP is a shortened version of the Sensory Profile (SP; Dunn 1999), a 125-item survey that assesses sensory features according to the theoretical framework of Winnie Dunn (1997, 2001, 2007). The SSP questionnaire contains 38 items organized into 7 empirically derived subscales: tactile sensitivity (TAC; 7 items), taste/smell sensitivity (TSM; 4 items), movement sensitivity (MOV; 3 items), underresponsive/seeking sensation (USS; 7 items), auditory filtering (AFL; 6 items), low energy/weak (LEW; 6 items), and visual/auditory sensitivity (VAS; 5 items). All items are rated on a 1–5 Likert-type scale based on the frequency of the described behavior (1 = Always, 2 = Frequently, 3 = Occasionally, 4 = Seldom, 5 = Never). Scores are typically calculated by summing all items across a given subscale, and the SSP total score is often used as a measure of overall sensory symptomology. As lower item scores indicate more frequent atypical behaviors, lower SSP total and subscale scores are indicative of more pronounced sensory processing dysfunction. The SSP total score and the score on each subscale can be used to classify a child's level of sensory abnormality as "typical," "probable difference," or "definite difference" based on score percentiles from a large normative sample of children without disabilities 3–10 years of age ($N \approx 1200$).

In recent years, the second edition of the Sensory Profile (SP2; Dunn 2014) has been released, along with a shortened version similar to the SSP (SSP-2). The SSP-2 contains 34 items organized into subscales representing the four "sensory quadrants" of Dunn's theoretical model (sensory sensitivity, sensory avoiding, low registration, sensory seeking). A large number of items are shared between the SSP and SSP-2, but the latter measure has been modified to improve content coverage of specific sensory response patterns (such as hyporeactivity/low registration) that are poorly represented in the original SSP. The format of the SSP-2 is similar to that of the SSP, with behaviors being rated on a 1–5 Likert-type scale representing frequency ("almost never" to

"almost always"). However, unlike the previous version, *higher* scores on the SSP-2 represent increased frequency of the atypical behaviors. Scores on the SSP-2 and its subscales can also be compared to a large normative sample of children without disabilities aged 3–14 years ($N = 697$), allowing for a child's score to be characterized as abnormally high or low.

Historical Background

The Short Sensory Profile was developed as a short form of Dunn's Sensory Profile caregiver questionnaire, with the goal of maximally discriminating between typical children and those with clinically significant sensory processing dysfunction. The 38-items of the SSP were chosen based on psychometric analyses of SP questionnaires completed by the caregivers of 117 children aged 3–17 years: 21 with clinically diagnosed sensory modulation disorder (SMD); 24 with fragile X syndrome (FXS); 35 with other developmental disabilities, including but not limited to ASD; and 37 with typical development (TD). From the 125 original SP items, 27 were immediately eliminated based on item content outside the domain of sensory modulation issues. Sixty additional items were removed based on (a) poor discrimination between SMD and TD groups, (b) low item-total correlations with conceptually similar sections of the full-length SP, (c) low item loadings in a principal component analysis (PCA) using the large normative SP validation sample, and (d) increased subscale Cronbach's alpha upon removal of the item. The final 38 items were then subjected to another PCA in the large normative sample, from which the seven SSP subscales were derived.

As the SP and SSP were some of the first proxy-report measures of sensory processing available, they were quickly applied to the study of sensory features in ASD. In the first study of this sort, Kientz and Dunn (1997) compared SP item scores between 32 children with ASD and 64 TD controls, finding significant group differences in 84 of 99 items. The first study to employ

the SSP was that of Rogers and colleagues (2003), who found that scores on five of the SSP subscales (TAC, TSM, USS, AFL, and LEW) differentiated children with ASD or FXS from mental age-matched children with other developmental disabilities or TD. However, the measure remained fairly obscure in autism research until 2007, when the measure's creator published a large retrospective study comparing SSP item, subscale, and total scores between 281 children with ASD and 281 age-matched TD controls (Tomchek and Dunn 2007). In this seminal study, the ASD group exhibited a strikingly high incidence of sensory processing issues, with SSP total scores classifying 83.6% and 11.4% of ASD children as having a "definite difference" (83.6%) or "probable difference" (11.4%) in sensory processing, compared to 3.2% and 13.6% in the TD group. Substantial numbers of ASD children also scored within the "definite difference" range in each of the SSP subscales (23.1–86.1%). Mean differences between the groups were highly significant for all SSP subscales, with small to moderate effect sizes ($d = 0.219$ – 0.628) for five of the seven subscales (TAC, TSM, USS, AFL, and VAS). Smaller effects were found for group differences in the MOV subscale ($d = 0.039$), LEW subscale ($d = 0.109$), and SSP total score ($d = 0.115$). Lastly, 35 of the 38 items (all except "prefers long-sleeved clothing even when it is warm or short sleeves when it is cold," "dislikes activities where head is upside down," and "covers eyes or squints to protect eyes from light") showed significant between-group differences at the $p < 0.001$ level ($d = 0.243$ – 0.652). These figures form the basis for the oft-cited claim that greater than 90% of children with ASD exhibit sensory processing impairments.

After the publication of Tomchek and Dunn's study, the use of the SSP in autism research increased substantially. Based on a review of 93 studies assessing sensory processing in ASD, the SSP was the second most utilized sensory phenotyping measure (28.0% of studies) after the full-length SP (Burns et al. 2017). Additionally, the SSP has been the sole sensory measure employed in several large-scale autism phenotyping projects (Charman et al. 2017;

Lajonchere et al. 2012; Singer 2005). The SSP and its subscales are frequently used as dimensional measures of sensory features, serving to quantify the relationships between specific patterns of sensory reactivity and other clinical or neurophysiologic measures (e.g., Glod et al. 2015). A separate group of studies has investigated profiles of SSP subscale scores from children with ASD to define phenotypic subgroups using cluster analysis techniques (Hand et al. 2017, 2018; Lane et al. 2010, 2011, 2014; Simpson et al. 2019; Tomchek et al. 2018; Uljarević et al. 2016). The SSP total score is also frequently utilized in the development of newer sensory measures to establish convergent validity (Neil et al. 2017; Siper et al. 2017; Tavassoli et al. 2016). Although the SSP-2 has been available for a number of years, its use in autism research thus far has been very limited (Simpson et al. 2019). This phenomenon may be due to the increased numbers of sensory measures now available for use in research paradigms, the large amount of SSP data that has already been collected, the relative paucity of studies utilizing the SSP-2 in ASD, and the presence of psychometric validation studies of the SSP but not SSP-2 in the ASD population. Nevertheless, as more studies are published utilizing the SSP-2 in ASD, it is likely that this measure will come to replace the original SSP in research and clinical practice settings.

Psychometric Data

Despite the widespread use of the SSP in children with ASD, there have been relatively few studies assessing its psychometric properties in this population (McIntosh et al. 1999; Stevenson et al. 2019; Tomchek et al. 2014; Williams et al. 2018). As the SSP was developed using a large TD sample and a smaller mixed-diagnosis sample, the SSP test manual does not contain estimates of test reliability or validity that pertain directly to the ASD population. Nevertheless, the figures provided therein provide preliminary evidence of the measure's reliability and validity in a typical population. Internal consistency reliability was

estimated for the SSP subscales in the mixed-diagnosis sample of 117 children and found to be adequate in all cases ($\alpha = 0.82\text{--}0.89$). Construct validity was also assessed in a small study evaluating skin conductance responses to sensory stimuli in children with SMD ($n = 19$) and TD children ($n = 19$). Results showed that children with abnormal skin conductance responses (either greater than or less than the values of the TD group) scored lower on the SSP, indicating higher levels of sensory modulation difficulties than children with typical skin conductance (McIntosh et al. 1999). Known-groups validity was also established, both by the initial item-selection process (i.e., only choosing items that discriminated between children with SMD and TD; McIntosh et al. 1999) and by the ability of 92% of the SSP items to discriminate between ASD and TD groups with a small to moderate effect size (Tomchek and Dunn 2007).

To date, three studies have investigated the psychometric properties of the SSP in samples of children with ASD, focusing primarily on the questionnaire's latent structure. For much of the time that the SSP has been used in ASD, the seven subscales derived from the TD sample were assumed to represent the latent structure in this population. However, factor-analytic studies have generally disagreed with this assertion, suggesting that the latent structure of the questionnaire differs significantly in children with ASD (Tomchek et al. 2014; Williams et al. 2018). In the first study of this sort, Tomchek et al. (2014) carried out a PCA with varimax rotation on the 38 items of the SSP in a sample of 400 ASD children 3–6 years of age. The original 7-component solution of the SSP was not replicated in this sample, and the authors instead chose to report a 6-component structure based on interpretability. The TSM and LEW subscales from the original pattern were replicated, and the other four components were interpreted as (a) tactile and movement sensitivity, (b) auditory and visual sensitivity, (c) sensory seeking/distractibility, and (d) hypo-responsivity (Tomchek et al. 2014). More recently, Williams et al. (2018) utilized confirmatory factor analysis to test both the original 7-factor model and the revised 6-factor model of the SSP in a sample of

388 children with ASD aged 2–12 years. Based on widely accepted fit index guidelines (i.e., CFI/TLI > 0.95 , RMSEA < 0.06 , SRMR < 0.08 , and WRMR < 1.0 are indicative of good model fit; Hu and Bentler 1999; DiStefano et al. 2017), neither the 7-factor model (CFI = 0.922, TLI = 0.915, RMSEA = 0.071, SRMR = 0.086, WRMR = 1.603) nor the 6-factor model (CFI = 0.924, TLI = 0.918, RMSEA = 0.069, SRMR = 0.086, WRMR = 1.623) showed acceptable fit to the data. Furthermore, examination of the residual correlations showed multiple areas of local model misfit for both models, further suggesting they are inappropriate to describe the factor structure of the SSP in ASD children. Most recently, Stevenson et al. (2019) tested the original 7-factor SSP model in an independent ASD sample ($N = 361$, ages 1.88–21.89 years). Although these authors concluded that “fit indices indicated acceptable overall fit,” two of three reported model fit indices suggested that the model was not a good fit for the data (CFI = 0.865, RMSEA = 0.066, SRMR = 0.067). Together, these three studies indicate that the 7-factor model of the SSP derived in TD children is not valid in children with ASD and should be abandoned in favor of a structural model specific to this population.

After failing to confirm either of the proposed SSP models in an ASD sample, Williams and colleagues conducted an exploratory factor analysis, finding a 9-factor solution to best explain the covariance structure of the SSP. These factors were interpreted as (1) low energy/weakness, (2) taste/smell sensitivity, (3) hyperactivity/inattention, (4) tactile sensitivity, (5) movement sensitivity, (6) auditory distractibility, (7) hyporesponsiveness to speech, (8) visual sensitivity, and (9) noise distress. Seven of the 38 items (1, 2, 5, 15, 19, 21, 37) were found to be poor indicators of any factor and were thus dropped from the final model. Confirmatory factor analysis of the remaining 31 items indicated an acceptable fit of the 9 correlated factor model on all fit indices except the WRMR (CFI = 0.974, TLI = 0.969, RMSEA = 0.052, SRMR = 0.058, WRMR = 1.042). While this 9-factor model has yet to be replicated in independent samples, it

represents the most appropriate structural model proposed for the SSP to date. Moreover, the low energy/weak, taste/smell sensitivity, and movement sensitivity factors were unchanged from their original subscales, indicating that these three subscales in particular can be validly interpreted in children with ASD. The composition of each subscale is presented in Table 1.

Internal consistency reliability of the SSP and its subscales in children with ASD were reported by Williams et al. (2018). Reliability coefficients (coefficient ω) for the original seven SSP subscales were all acceptable, ranging from 0.76 (MOV) to 0.97 (LEW). However, the authors warned that these coefficients should only be interpreted for subscales that correspond to the underlying factor structure. Thus, the only interpretable ω coefficients are those for the TSM ($\omega = 0.94$), MOV ($\omega = 0.76$), and LEW ($\omega = 0.97$) subscales, which have equivalent factor structures in the ASD and TD populations. Based on their exploratory factor model, Williams et al. also estimated the internal consistency reliabilities (coefficient ω_s) for the nine empirically derived SSP subscales (Table 1). Internal consistency reliability for the various subscales varied substantially with the value for the “hypo-responsiveness to speech” indicating unacceptably low reliability ($\omega_s = 0.63$) but all other values surpassing the often-used acceptability

cutoff of 0.70 or greater (Nunnally and Bernstein 1994). Note that the values for TSM, MOV, and LEW differ somewhat from those mentioned previously, due to the prior values being calculated from unidimensional confirmatory factor models. Additionally, reliability of the summed SSP total score was found to be very high (coefficient $\omega_t = 0.96$), indicating that 96% of total score variance was attributable to the underlying latent factors. Internal consistency reliabilities of the SSP-2 quadrants in a sample of ASD children, as measured by Cronbach’s α , were reported to be adequate for three of the four scales (seeking $\alpha = 0.69$, avoiding $\alpha = 0.83$, sensitivity $\alpha = 0.75$, registration $\alpha = 0.75$; Simpson et al. 2019). No study to date has assessed other forms of reliability, such as test-retest and inter-rater reliability, for either the SSP or SSP-2.

Additionally, there has been little published work specifically assessing the validity of the SSP total and subscale scores in ASD. However, the aforementioned factor-analytic studies contribute somewhat to our understanding of the test’s structural validity in ASD. As four of the original seven SSP subscales (TAC, USS, AFL, and VAS) were not replicated in the best-fitting factor model (Williams et al. 2018), these scales do not seem to measure meaningful constructs in the ASD population and are thus not valid for use. Of the remaining scales, the TSM scale does seem to have some construct validity as a measure of food selectivity, supported by large correlations between TSM scores and other measures of food selectivity across samples of children with ASD (Williams et al. 2018). The construct validity of the two remaining subscales has not been assessed in this population, and it remains to be seen whether these sales are valid measures of their underlying constructs. The revised scales presented by Williams and colleagues also have not undergone any tests of construct validity. However, as several of these scales contain only two items, they likely do not represent all content areas of their underlying latent constructs and may have questionable content validity. Williams et al. (2018) also assessed the validity of the SSP total score as a measure of general sensory dysfunction, finding

Short Sensory Profile in Autism, Table 1 Empirically derived subscales of the Short Sensory Profile for children with ASD

Scale	SSP items	Reliability (ω_s)
Low energy/weakness	28–33	0.95
Taste/smell sensitivity	8–11	0.96
Hyperactivity/inattention	16–18, 20, 27	0.75
Tactile sensitivity	3, 4, 6, 7	0.78
Movement sensitivity	12–14	0.82
Auditory distractibility	22, 24, 25	0.81
Hypo-responsiveness to speech	23, 26	0.63
Visual sensitivity	36, 38	0.81
Noise distress	34, 35	0.79
Total score	1–38	$\omega_t = 0.96$

it to be a poor measure for this construct on multiple grounds. When modeled, a general sensory processing dysfunction factor explained a fairly low percentage of total score variance (coefficient $\omega_H = 0.68$). The scale's content validity was also called into question, as the SSP does not contain items assessing the full range of sub-constructs that contribute to general sensory processing dysfunction (Miller et al. 2007; Williams et al. 2018). Overall, it was recommended that the SSP total score not be utilized as a measure of general sensory processing dysfunction in children with ASD. To date, no study has assessed the validity of the SSP-2 or its subscales in children with ASD, although the form does seem to measure a wider range of sensory processing sub-constructs, thus improving its content validity.

Clinical Uses

The SSP has sometimes been utilized in clinical settings as part of a multidisciplinary assessment battery in young children with ASD and other developmental disabilities, helping to inform clinical recommendations for services such as physical or occupational therapy. The questionnaire was originally developed for clinical use by occupational therapists to screen for sensory processing dysfunction in children, and it is sometimes also used for this purpose in ASD. However, given the high likelihood of most children with ASD scoring in the abnormal range overall, some clinicians may prefer to use this test to examine which areas of sensory processing are contributing most to a child's difficulty. Some clinicians may also administer the SSP on repeated occasions to track a child's progress over time as he or she undergoes an intervention. However, no empirical data have been published on the suitability of the SSP or SSP-2 for informing clinical decision-making, guiding intervention choices, or reflecting the efficacy of ongoing interventions in children with ASD. Additional research is required to validate the SSP or the SSP-2 for clinical use in any of these domains.

See Also

- Clinical Assessment
- Perception
- Sensory Impairment in Autism

References and Reading

- Burns, C. O., Dixon, D. R., Novack, M., & Granpeesheh, D. (2017). A systematic review of assessments for sensory processing abnormalities in Autism Spectrum Disorder. *Review Journal of Autism and Developmental Disorders*, 4(3), 209–224. <https://doi.org/10.1007/s40489-017-0109-1>.
- Charman, T., Loth, E., Tillmann, J., Crawley, D., Wooldridge, C., Goyard, D., & Buitelaar, J. K. (2017). The EU-AIMS longitudinal European autism project (LEAP): Clinical characterisation. *Molecular Autism*, 8(1), 27. <https://doi.org/10.1186/s13229-017-0145-9>.
- DiStefano, C., Liu, J., Jiang, N., & Shi, D. (2017). Examination of the weighted root mean square residual: Evidence for trustworthiness? *Structural Equation Modeling*, 25(3), 453–466. <https://doi.org/10.1080/10705511.2017.1390394>.
- Dunn, W. (1997). The impact of sensory processing abilities on the daily lives of young children and their families: A conceptual model. *Infants & Young Children*, 9(4), 23–35. <https://doi.org/10.1097/00001163-199704000-00005>.
- Dunn, W. (1999). *Sensory profile: User's manual*. San Antonio: Psychological Corporation.
- Dunn, W. (2001). The sensations of everyday life: Empirical, theoretical, and pragmatic considerations. *American Journal of Occupational Therapy*, 55(6), 608–620. <https://doi.org/10.5014/ajot.55.6.608>.
- Dunn, W. (2007). Supporting Children to Participate Successfully in Everyday Life by Using Sensory Processing Knowledge. *Infants & Young Children*, 20(2), 84–101. <https://doi.org/10.1097/01.iyc.0000264477.05076.5d>.
- Dunn, W. (2014). *Sensory profile 2: User's manual*. San Antonio: Psychological Corporation.
- Glod, M., Riby, D. M., Honey, E., & Rodgers, J. (2015). Psychological correlates of sensory processing patterns in individuals with autism spectrum disorder: A systematic review. *Review Journal of Autism and Developmental Disorders*, 2(2), 199–221. <https://doi.org/10.1007/s40489-015-0047-8>.
- Hand, B. N., Dennis, S., & Lane, A. E. (2017). Latent constructs underlying sensory subtypes in children with autism: A preliminary study. *Autism Research*, 61, 110. <https://doi.org/10.1002/aur.1787>.

- Hand, B. N., Lane, A. E., De Boeck, P., Basso, D. M., Nichols-Larsen, D. S., & Darragh, A. R. (2018). Caregiver burden varies by sensory subtypes and sensory dimension scores of children with autism. *Journal of Autism and Developmental Disorders*, 48(4), 1133–1146. <https://doi.org/10.1007/s10803-017-3348-1>.
- Hu, L.-T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*, 6(1), 1–55. <https://doi.org/10.1080/10705519909540118>.
- Kientz, M. A., & Dunn, W. (1997). A comparison of the performance of children with and without autism on the Sensory Profile. *American Journal of Occupational Therapy*, 51(7), 530–537.
- Lajonchere, C., Jones, N., Coury, D. L., & Perrin, J. M. (2012). Leadership in health care, research, and quality improvement for children and adolescents with autism spectrum disorders: Autism treatment network and autism intervention research network on physical health. *Pediatrics*, 130(Supplement 2), S62–S68. <https://doi.org/10.1542/peds.2012-0900C>.
- Lane, A. E., Young, R. L., Baker, A. E. Z., & Angley, M. T. (2010). Sensory processing subtypes in autism: association with adaptive behavior. *Journal of Autism and Developmental Disorders*, 40(1), 112–122. <https://doi.org/10.1007/s10803-009-0840-2>.
- Lane, A. E., Dennis, S. J., & Geraghty, M. E. (2011). Brief report: Further evidence of sensory subtypes in autism. *Journal of Autism and Developmental Disorders*, 41(6), 826–831. <https://doi.org/10.1007/s10803-010-1103-y>.
- Lane, A. E., Molloy, C. A., & Bishop, S. L. (2014). Classification of children with autism spectrum disorder by sensory subtype: A case for sensory-based phenotypes. *Autism Research*, 7(3), 322–333. <https://doi.org/10.1002/aur.1368>.
- McIntosh, D. N., Miller, L. J., & Shyu, V. (1999). Development and validation of the Short Sensory Profile. In W. Dunn (Ed.), *Sensory profile: User's manual* (pp. 59–73). San Antonio: Psychological Corporation.
- Miller, L. J., Anzalone, M. E., Lane, S. J., Cermak, S. A., & Osten, E. T. (2007). Concept evolution in sensory integration: A proposed nosology for diagnosis. *The American Journal of Occupational Therapy*, 61(2), 135–140.
- Neil, L., Green, D., & Pellicano, E. (2017). The psychometric properties of a new measure of sensory behaviors in autistic children. *Journal of Autism and Developmental Disorders*, 47(4), 1261–1268. <https://doi.org/10.1007/s10803-016-3018-8>.
- Nunnally, J. C., & Bernstein, I. H. (1994). *Psychometric theory*. New York: MacGraw-Hill.
- Rogers, S. J., Hepburn, S., & Wehner, E. (2003). Parent reports of sensory symptoms in toddlers with autism and those with other developmental disorders. *Journal of autism and developmental disorders*, 33(6), 631–642. <https://doi.org/10.1023/B:JADD.0000006000.38991.a7>
- Simpson, K., Adams, D., Alston-Knox, C., Heussler, H. S., & Keen, D. (2019). Exploring the sensory profiles of children on the autism spectrum using the Short Sensory Profile-2 (SSP-2). *Journal of Autism and Developmental Disorders*, 49(5), 2069–2079. <https://doi.org/10.1007/s10803-019-03889-2>.
- Singer, E. (2005). “Phenome” project set to pin down subgroups of autism. *Nature Medicine*, 11(6), 583–583. <https://doi.org/10.1038/nm0605-583a>.
- Siper, P. M., Kolevzon, A., Wang, A. T., Buxbaum, J. D., & Tavassoli, T. (2017). A clinician-administered observation and corresponding caregiver interview capturing DSM-5 sensory reactivity symptoms in children with ASD. *Autism Research*, 10(6), 1133–1140. <https://doi.org/10.1002/aur.1750>.
- Stevenson, R. A., Parks, K. M., Schulz, S. E., Segers, M., Anagnostou, E., Nicolson, R., . . . Liu, X. (2019, May). Sensory processing in ASD and ADHD: A multi-groups approach. Poster presented at the annual meeting of the international society for autism research, Montreal.
- Tavassoli, T., Bellesheim, K., Siper, P. M., Wang, A. T., Halpern, D., Gorenstein, M., et al. (2016). Measuring sensory reactivity in autism spectrum disorder: Application and simplification of a clinician-administered sensory observation scale. *Journal of Autism and Developmental Disorders*, 46(1), 287–293. <https://doi.org/10.1007/s10803-015-2578-3>.
- Tomchek, S. D., & Dunn, W. (2007). Sensory processing in children with and without autism: A comparative study using the Short Sensory Profile. *American Journal of Occupational Therapy*, 61(2), 190–200.
- Tomchek, S. D., Huebner, R. A., & Dunn, W. (2014). Patterns of sensory processing in children with an autism spectrum disorder. *Research in Autism Spectrum Disorders*, 8(9), 1214–1224. <https://doi.org/10.1016/j.rasd.2014.06.006>.
- Tomchek, S. D., Little, L. M., Myers, J., & Dunn, W. (2018). Sensory subtypes in preschool aged children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(6), 2139–2147. <https://doi.org/10.1007/s10803-018-3468-2>.
- Uljarević, M., Lane, A., Kelly, A., & Leekam, S. (2016). Sensory subtypes and anxiety in older children and adolescents with autism spectrum disorder. *Autism Research*, 9(10), 1073–1078. <https://doi.org/10.1002/aur.1602>.
- Williams, Z. J., Failla, M. D., Gotham, K. O., Woytnarowski, T. G., & Cascio, C. (2018). Psychometric evaluation of the Short Sensory Profile in youth with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(12), 4231–4249. <https://doi.org/10.1007/s10803-018-3678-7>.

Short-Term Memory

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Definition

Short-term memory refers to the immediate or temporary storage of information.

Historical Background

The limited data that can be stored in short-term memory will be lost if not repeated or rehearsed. An individual's ability to temporarily store information is considered to be a function of a variety of factors, including how the information was encoded, whether it could be rehearsed, and even the psychological state of the individual, such as his or her anxiety level (e.g., Darke 1988). Early work on memory suggested that short-term memory was a unified temporary storage system (Atkinson and Shiffrin 1968). According to Baddeley and Hitch (1974), short-term memory was later proposed to have several components, collectively referred to as working memory, responsible for simultaneously storing and operating on information for relatively short periods of time when an individual performs cognitive tasks. These three components of working memory were referred to as the central executive, the visuospatial sketchpad, and the phonological loop. The central executive component is conceptualized as a control system with a key role in identifying issues on which to focus attention, decision-making, and response selection (Boller 1996). The visuospatial sketchpad is considered responsible for storing visual and spatial information, while the phonological loop is responsible for storing sounds, and is thought to be important for language development (Baddeley 1992). More recently, Baddeley (2000) proposed a revision to the Baddeley and Hitch (1974) model by conceptualizing the central executive as a domain general

component that integrates or coordinates information from visuospatial and verbal information. Moreover, the new system proposed by Baddeley (2000) includes the notion of an "episodic buffer" which is thought to be a short-term storage system that is controlled by the central executive, integrates information, and plays a role in the exchange of information to and from episodic long-term memory.

Several theories exist concerning potential short-term memory deficits in autism (see Poirier and Martin 2008 for a review). Some have suggested that because short-term memory tasks require individuals to recall stimuli in their order of presentation, individuals with autism should show a deficit due to deficits in temporal processing (Boucher 2001). Others argue that difficulties with retrieval on short-term memory tasks, in particular, should be apparent in autism due to impairments in remembering contextual and personally experienced aspects of events (Bowler et al. 2004). Finally, there is some evidence of a group-by-difficulty interaction such that short-term memory impairments for higher-functioning individuals with autism may appear as task difficulty increases (Craig and Anderson 1999).

Current Knowledge

Although previous empirical findings suggested an intact short-term memory system in individuals with autism (e.g., Hermelin and O'Connor 1967), these studies may have had some methodological limitations, including a reliance on span-type tasks and use of a smaller than ideal number of trials (Poirier and Martin 2008). With respect to working memory, which is commonly considered a dimension of executive function, individuals with autism appear to show performance consistent with the types of deficits one might see in patients with frontal lobe damage (Hill 2004), leading many in the field to argue that autism is inherently a disorder of executive function. However, empirical findings indicating a working memory deficit specific to autism are somewhat mixed (see Poirier and Martin 2008 for a review of

current findings concerning working memory in autism). For example, in a study of verbal working memory, Bennetto et al. (1996) found individuals with autism (11–25 years of age) to be impaired in working memory in comparison to a comparison group of individuals with learning disorders. However, Russell et al.'s (1996) study of working memory found no significant difference between children with autism and a moderate-learning-difficulties comparison group, although both of these groups performed significantly worse than a normally developing comparison group. Some studies have even found no working memory deficits in their samples of individuals with autism (Guerts et al. 2004; Ozonoff and Strayer 2001). Differences in these studies may be a function of the difficulty of the task or the level of support provided to retrieve information (Poirier and Martin 2008). Interestingly, when studies have differentiated between tasks that allow the use of inner speech to regulate behavior (e.g., tasks like the Luria hand game) and those that require a verbal response and therefore do not allow for inner speech (e.g., the day/night task), group differences were not found in the latter tasks, supporting the contention by Russell and colleagues that individuals with autism have a particular impairment in their ability to use inner speech to rehearse the rules of working memory tasks (Russell 1997).

Finally, an alternative account of working memory deficits in autism has been put forth by Minshew and Goldstein (1998), who suggest that such deficits are a function of information processing difficulties that overwhelm working memory resources. Studies supporting this approach reveal that differences in working memory performance appear as the working memory load increases (Joseph et al. 2005).

Future Directions

Given the mixed findings to date on short-term memory and working memory deficits in autism, there is a clear need for future work in this area. According to Poirier and Martin (2008), future research should address whether verbal working

memory and spatial working memory are both equally affected in ASDs. There is also a need for more research on whether observed deficits are due to complex information processing issues and whether these vary according to task complexity and difficulty, or task support for retrieval. Future work should also explore the implications of deficits in short-term memory, for example, the extent to which verbal short-term memory difficulties may impact language development (Alloway et al. 2009).

See Also

- ▶ [Executive Function \(EF\)](#)
- ▶ [Memory](#)
- ▶ [Memory Assessment](#)
- ▶ [Memory Development](#)

References and Reading

- Alloway, T. P., Rajendran, G., & Archibald, L. M. D. (2009). Working memory in children with developmental disorders. *Journal of Learning Disabilities*, 42, 372–382.
- Atkinson, R. C., & Shiffrin, R. M. (1968). Human memory: A proposed system and its control processes. In K. W. Spence & J. T. Spence (Eds.), *The psychology of learning and motivation: Advances in research and theory* (Vol. 2). New York: Academic Press.
- Baddeley, A. D. (1992). Working memory. *Science*, 255, 556–559.
- Baddeley, A. D. (2000). The episodic buffer: A new component of working memory? *Trends in Cognitive Science*, 4, 417–423.
- Baddeley, A. D., & Hitch, G. J. (1974). Working memory. In G. Bower (Ed.), *The psychology of learning and motivation* (pp. 47–90). San Diego: Academic Press.
- Bennetto, L., Pennington, B. F., & Rogers, S. J. (1996). Intact and impaired memory functions in autism. *Child Development*, 67, 1816–1835.
- Boller, K. (1996). Conceptualizing, describing, and measuring components of memory: A summary. In G. R. Lyon & N. A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 221–231). Baltimore: Paul H. Brookes.
- Boucher, J. (2001). “Lost in a sea of time”: Time parsing and autism. In C. Hoerl & T. McCormack (Eds.), *Time and memory: Issues in philosophy and psychology* (pp. 111–135). Oxford: Clarendon Press.
- Bowler, D. M., Gardiner, J. M., & Berthollier, N. (2004). Source memory in adolescents and adults with

- Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 34, 533–542.
- Craik, F. I. M., & Anderson, N. D. (1999). Applying cognitive research to problems of aging. In D. Gopher & A. Koriati (Eds.), *Attention and performance XVII* (pp. 583–615). Mahwah: Erlbaum.
- Darke, S. (1988). Anxiety and working memory capacity. *Cognition and Emotion*, 2, 145–154.
- Guerts, H. M., Verte, S., Oosterlaan, J., Roeyers, H., & Sergeant, J. A. (2004). How specific are executive function deficits in attention deficit disorder and autism? *Journal of Child Psychology and Psychiatry*, 45, 836–854.
- Hermelin, B., & O'Connor, N. (1967). Remember of words by psychotic and subnormal children. *British Journal of Psychology*, 58, 213–218.
- Hill, E. L. (2004). Evaluating the theory of executive dysfunction in autism. *Developmental Review*, 24, 189–233. <https://doi.org/10.1016/j.dr.2004.01.001>.
- Joseph, R. M., McGrath, L., & Tager-Flusberg, H. (2005). Executive dysfunction and its relationship to language impairment in school-age children with autism. *Developmental Neuropsychology*, 27, 361–378.
- Mayes, S. D., & Calhoun, S. L. (2008). WISC-IV and WIAT-II profiles in children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 38, 428–439.
- Minshew, N. J., & Goldstein, G. (1998). Autism as a disorder of complex information processing. *Mental Retardation and Developmental Disabilities Research Reviews*, 4, 129–136.
- Ozonoff, S., & Strayer, D. L. (2001). Further evidence of intact working memory in autism. *Journal of Autism and Developmental Disorders*, 31, 257–263.
- Poirier, M., & Martin, J. S. (2008). Working memory and immediate memory in ASDs. In J. Boucher & D. Bowler (Eds.), *Memory in autism: Theory and evidence* (pp. 231–248). Cambridge, NY: Cambridge University Press.
- Russell, J. (1997). How executive disorders can bring about an inadequate theory of mind. In J. Russell (Ed.), *Autism as an executive disorder* (pp. 256–304). Oxford: Oxford University Press.
- Russell, J., Jarrold, C., & Henry, L. (1996). Working memory in children with autism and with moderate learning difficulties. *Journal of Child Psychology and Psychiatry*, 37, 673–686.

Sibling Emotion

► Sibling Feelings About Their Brother or Sister with Autism Spectrum Disorder

Sibling Feelings About Their Brother or Sister with Autism Spectrum Disorder

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Synonyms

Sibling emotion; Sibling relationship

Definition

Siblings of people with autism spectrum disorder (ASD) often play a large role in their brothers' or sisters' lives. Siblings can be teachers, models, and social partners, and, in later life, they can provide emotional, financial, and other functional support for the individual with ASD.

In order to maintain the health and well-being of both the sibling and the individual with ASD throughout these interactions across the lifespan, the siblings must have a positive relationship with each other. Although many individuals with ASD do have healthy relationships with their typically developing siblings, there are several aspects of ASD that may make it difficult for siblings to deal with. First, the social communication deficits of people with ASD may make it difficult for siblings to communicate with their brother or sister with ASD. Second, if the individual with ASD has a lot of behavior problems, such as aggression, then the sibling relationship may be not as close.

Overall, siblings of individuals with ASD tend to have less positive relationships with their brother or sister, even when compared to siblings of individuals with other intellectual and developmental disabilities (Shivers et al. 2019). Siblings of individuals with ASD who exhibit high levels

of behavior problems also tend to have lower levels of warmth and closeness in the sibling relationships (Hastings and Petalas 2014).

An important part of the sibling relationship is how the typically developing siblings feel about their brother or sister with ASD. Siblings of individuals with ASD have, on average, more negative beliefs about people with disabilities in general (Shivers et al. 2019). That is, siblings of people with ASD are more likely to believe that their brother or sister is a “problem” for the family or the classroom. Additionally, compared to siblings of individuals without disabilities, siblings of individuals with ASD report more overall negative feelings toward their brother or sister (Shivers and McGregor 2019). Importantly, however, siblings of individuals with ASD do not report lower levels of positive feelings toward their brother or sister (Shivers and McGregor 2019).

Although general research shows that siblings of individuals with ASD may struggle to develop close, positive relationships with their brother or sister and may feel, on average, more anxious and frustrated about their brother or sister, individual reports show a lot of variability in sibling experiences. Parents and siblings report nurturing relationship, with the typically developing sibling showing patience and pride when interacting with or teaching the individual with ASD (Dansby et al. 2018). Siblings also report strong feelings of family, indicating a sense of togetherness and protectiveness (Pavlopoulou and Dimitriou 2019). However, siblings also report many challenges. While survey research shows that behavior problems among the individual with ASD are related to less closeness in the sibling relationships (Hastings and Petalas 2014), siblings report more complexity – that is, seeing behavior problems in their brother or sister can make the sibling feel sad or helpless (Pavlopoulou and Dimitriou 2019). Finally, a common theme in sibling reports is the feeling that the world does not understand either the individual with ASD or the sibling experience (Dansby et al. 2018). Siblings believe that society does not

understand autism, and thus, life is harder for both them and their brother or sister.

Sibling feelings about their brother or sister with ASD are complex and wide-ranging. Although siblings of individuals with ASD, on average, have more negative feelings toward their brother or sister and less close sibling relationships, these feelings in total involve more than just anger and irritation. Siblings worry about their brother or sister, showing concern for both how the world will treat them now and in the future. They also report pride, warmth, and love. Overall, siblings of individuals with ASD acknowledge many challenges that can make it difficult to have a close relationship with their brother or sister, but many siblings still report numerous positive experiences with the individual with ASD.

References and Reading

- Dansby, R. A., Turns, B., Whiting, J. B., & Crane, J. (2018). A phenomenological content analysis of online support seeking by siblings of people with autism. *Journal of Family Psychotherapy*, 29(3), 181–200.
- Hastings, R. P., & Petalas, M. A. (2014). Self-reported behaviour problems and sibling relationship quality by siblings of children with autism spectrum disorder. *Child: Care, Health and Development*, 40(6), 833–839.
- Pavlopoulou, G., & Dimitriou, D. (2019). ‘I don’t live with autism; I live with my sister’. Sisters’ accounts on growing up with their preverbal autistic siblings. *Research in Developmental Disabilities*, 88, 1–15.
- Shivers, C. M., & McGregor, C. M. (2019). Brief Report: Sibling feelings toward their brother or sister with or without autism or intellectual disability. *Journal of Autism and Developmental Disorders*, 49(1), 404–409.
- Shivers, C. M., et al. (2019). Functioning among typically developing siblings of individuals with autism spectrum disorder: A meta-analysis. *Clinical Child and Family Psychology Review*, 22(2), 172–196.

Sibling Relationship

- Sibling Feelings About Their Brother or Sister with Autism Spectrum Disorder

Sibling Support

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Definition

Sibling supports are any services, interventions, or programs focused on typically developing siblings of individuals with intellectual or developmental disabilities, including autism spectrum disorders (ASD), with the goal of improving sibling well-being or increasing siblings' participation in the lives of their brothers or sisters with a disability. Sibling supports most often take the form of peer support but can also be informational sessions aimed at increasing knowledge about their brother or sister.

Historical Background

Sibling supports were initially developed in reaction to extant research and anecdotal reports suggesting that having a brother or sister with an intellectual or developmental disability, including ASD, can be difficult for some siblings (Lobato 1983; Meyer and Vadasy 1994). Although supports and services are often available to the individual with the disability himself or herself, supports are far less common for family members, including siblings.

The number of sibling support programs and interventions has increased somewhat over the past 20 years; Sibshops (Meyer and Vadasy 1994) is perhaps the first and best-known example of a sibling support. Many of the sibling support programs that have followed either incorporate aspects of the Sibshop model or build on it. It is

important to note that support interventions and programs for siblings have typically been geared toward siblings of individuals (mostly children) with any intellectual or developmental disability, including siblings of those with ASD. With the exception of one program, none of these supports specifically targets siblings of individuals with ASD.

Rationale or Underlying Theory

Sibling support programs and interventions share two main rationales. The rationale for programs that mainly focus on fostering sibling peer support – such as Sibshops (event-based program for siblings of children with disabilities: Meyer and Vadasy 1994), Sibkids (an internet listserv for siblings of children with disabilities developed as part of the Sibling Support Project, the umbrella organization that also includes Sibshops), and support group for siblings of children with ASD based at the Thistletown Regional Centre in Toronto, Ontario (Smith and Perry 2004) – is that being a sibling of an individual with intellectual and developmental disabilities can be a primarily positive experience, primarily negative experience, or a combination of the two. Furthermore, these siblings face unique challenges and experiences not shared by children who do not have a brother or sister with disabilities. Thus, developing peer supports with others who have a brother or sister with intellectual or developmental disabilities will allow siblings to discuss their experiences with others who understand, leading to wellness and well-being.

The rationale for sibling supports that are more informational in nature – such as the Sunsibs program based at Sunfield residential school in the United Kingdom (described in Conway and Meyer 2008), the Siblink program (Lobato and Kao 2002), the “Intervention for Siblings: Experience Enhancement” program (ISEE; Williams et al. 2003), and an unnamed program focused on siblings from socioeconomically disadvantaged families living in the inner city (Phillips 1999) – is that increasing siblings’

knowledge about their brother or sister's disability, behaviors, and needs will be beneficial to the nondisabled siblings. Specifically, increasing knowledge might lower the stress experienced by these siblings, help them to develop a closer relationship with their brother or sister with disabilities, and prepare them for the possibility of providing care for their brother or sister in adulthood.

Goals and Objectives

Existing sibling support programs have three general goals/objectives: increasing peer support, sharing of information, and increasing involvement of the sibling with the brother or sister with the disability. The Sibling Support Project, which includes both Sibshops and the Sibkids/Sibnet listservs, has the primary objective of *fostering peer support*. The Sibshop guidebook (Meyer and Vadasy 1994) lays out five specific goals related to this objective: (1) "Provide brothers and sisters of children with special needs an opportunity to meet other siblings in a relaxed, recreational setting"; (2) "Provide brothers and sisters with opportunities to discuss common joys and concerns with other siblings of children with special needs"; (3) "Provide siblings with an opportunity to learn how others handle situations commonly experienced by siblings of children with special needs"; (4) "Provide siblings with an opportunity to learn more about the implications of their sibling's special needs"; and (5) "Provide parents and other professionals with opportunities to learn more about the concerns and opportunities frequently experienced by brothers and sisters of people with special needs." As can be seen from these specific goals, sharing of information from one sibling to another (and not necessarily from teacher to sibling) is also an important aspect of Sibshops. Although not as well defined, the internet listserv arm of the Sibling Support Project (Sibkids for children and Sibnet for adults) has the objective of providing peer support and discussion in a secure environment.

Two of the existing sibling support programs have the primary objective of *increasing knowledge about the brother's or sister's disability*. The Thistlethorn program (Smith and Perry 2004) has perhaps the most delineated specific goals from this group of programs, including the following: (1) increasing knowledge and understanding of ASD and related developmental disorders, (2) providing the opportunity for siblings to discuss their feeling in an accepting environment, (3) helping siblings to share ways of coping with difficult situations unique to having a sibling with autism, (4) enhancing siblings' self-concepts, and (5) encouraging siblings to have fun in a supportive environment. As can be seen from these goals, sibling peer support is also an objective of the Thistlethorn program. The ISEE program (Williams et al. 2003) has the specific objectives of improving sibling knowledge, as well as improving siblings' perceptions of and affective reactions to their brother's or sister's illness or disability (this program also included siblings of children with chronic illness).

The support program geared toward socioeconomically disadvantaged children (Phillips 1999) and the SibLink program (Lobato and Kao 2002) both promote *providing information and enhancing peer social support* as equal objectives. Specifically, the program for disadvantaged families has the goal of alleviating the stress of having a sibling with an intellectual disability by providing information about developmental disabilities to facilitate understanding and by creating a context that provides social support from peers and adults. The SibLink program has the goals of improving sibling knowledge, sibling adjustment to living with a brother or sister with a chronic illness or developmental disability, and sibling connectedness to others who have a brother or sister with a chronic illness or developmental disability.

Finally, the Sunsibs program (as described in Conway and Meyer 2008) has the primary objective of *increasing involvement of the sibling in the lives of his/her brother or sister with ASD or other disabilities*. In order to increase sibling involvement, Sunsibs has the specific goals of providing

opportunities for siblings of children in the Sunfield program to meet other siblings and build relationships with them, of keeping siblings in touch with the Sunfield facility, and of helping siblings feel an integral part of their brother's or sister's lives while that brother or sister is participating in the Sunfield programs.

As can be seen from the above descriptions, sibling support programs differ in the emphasis placed on facilitating increased peer support, sharing of information, and increased involvement of the sibling with the brother or sister with the disability. Furthermore, many of the programs incorporate two or three of these themes into their overall goals or objectives.

Treatment Participants

The majority of sibling supports focus on school-aged children. Sibshops were originally developed for siblings aged 8–13 years but had been adapted to older or younger school-aged children (Conway and Meyer 2008; Meyer and Vadasy 1994). The Sibkid listserv is geared toward siblings under 18 years of age. The Thistle town program included siblings aged 6–16 years (Smith and Perry 2004), ISEE included siblings aged 7–15 years (Williams et al. 2003), and the program for disadvantaged children included siblings aged 9–12 years (Phillips 1999). Further, the SibLink program included siblings aged 8–13 years (Lobato and Kao 2002). Two of the programs are open to adult siblings. The Sunsibs program allows adult siblings to participate, as long as their brother or sister with the disability is between 6 and 19 years of age (Conway and Meyer 2008). The Sibnet listserv is open to siblings who are 18 years of age or older.

Nearly all of the existing sibling supports focus on siblings of individual with intellectual or developmental disabilities more generally, including those with ASD. The sole program that included only siblings of individuals with ASD is the Thistle town program. Two of the sibling supports – the ISEE and SibLink programs – included siblings of children with chronic illness

in addition to siblings of children with developmental disabilities.

Treatment Procedures

Sibling supports take the form of workshops/events, one-on-one supports, and secure internet listservs. Specific information about the procedures and content for each program can be found in the References and Readings. Workshops and events are the most prevalent type of procedure. Sibshops (Meyer and Vadasy 1994) consist of events that contain a mix of information, discussion, games, activities, and guests. The Thistle town program is an 8-week, age-specific support group that includes exercises, games, and activities promoting group cohesion, informational session on autism and related disorders, and discussions of feelings and attitudes associated with living with a person with a developmental disability (Smith and Perry 2004). Similarly, the SibLink program consists of six 90-min sessions conducted over 6–8 weeks, focused on improving sibling knowledge and family communication, managing feelings about having a brother or sister with a disability, and discussing how to balance siblings' own needs (Lobato and Kao 2002). The SibLink program also includes recreational activities at each session. The ISEE program contains structured teaching about the brother's or sister's disability or illness and psychosocial sessions over the course of a 5-day summer camp and two follow-up booster sessions (Williams et al. 2003). The program for socioeconomically disadvantaged children consists of an after-school program that lasts for 15 weeks and involves discussion about developmental disabilities, recreational activities, and homework assistance (Phillips 1999). The commonalities among most of these programs are the following: (1) having a specific starting and ending point with a limited number of sessions (except for Sibshops, which is ongoing) and (2) inclusion of social, informational, and recreational components.

The other procedures are less common. The Sunsibs program involves more direct support and training to the sibling, whereas the Sibkid

and Sibnet listservs involve siblings in posting and responding to messages on a secure website.

Efficacy Information

All of the sibling supports except for Sunsibs and the Sibkids/Sibnet listservs have some information about efficacy. However, it is difficult to draw conclusions about efficacy as the authors were only able to find one study that tests efficacy for most of the programs. Specific information about efficacy for each program is available in the References and Readings. Four of the five studies included standardized, validated measures as indicators of efficacy. Two programs – the ISEE program and the program for socioeconomically disadvantaged children – included before and after treatment measures and a control group that did not receive the treatment. For the ISEE program, researchers found improvements in knowledge and attitudes, social support, and self-esteem, and to some extent, behavior problems for siblings in the full-treatment condition (structured teaching and psychosocial sessions at summer camp) and improvements in attitude, self-esteem, and social support in the partial-treatment condition (summer camp only). Researchers concluded that there were dosage effects, with the full-treatment siblings showing the most improvement and partial-treatment siblings showing more improvement than controls (Williams et al. 2003). Researchers testing the program for socioeconomically disadvantaged children collected data on sibling depression, anxiety, perceived social support, and sibling relationship. They found improvement in each of these measures from before to after intervention, with no improvement in the control group (Phillips 1999).

Efficacy evaluation of two of the interventions – the Thistle town and SibLink programs – included standardized measures taken before and after intervention but no control group. Siblings who participated in the Thistle town program reported increased self-concept and greater knowledge about their brother's or sister's disability (ASD) at the conclusion of the program, but there were no significant differences in coping

(Smith and Perry 2004). After treatment, participants in the SibLink program reported more accurate knowledge of their brother's or sister's disability, greater connectedness with that sibling, and fewer behavior problems relative to pre-treatment (Lobato and Kao 2002).

Finally, efficacy of Sibshops was not evaluated by standardized measures, but instead by asking 30 sibling participants about their feelings regarding the program (Johnson and Sandall 2005). Evaluators found that over 90% of surveyed siblings reported positive effects of Sibshops on their feelings toward their brother or sister, over 60% stated that Sibshops taught them coping skills, and 94% said that they would recommend Sibshops to others. In a study of 16 Sibshops participants, D'Arcy, Flynn, McCarthy, O'Connor, and Tierney (2005) found that 14 children reported that they liked the program, with 11 rating Sibshops as "excellent" or "very good," though the participants showed no significant improvements in self-esteem after attending Sibshops.

In sum, there is some evidence to suggest that sibling supports are effective in improving sibling knowledge about their brother or sister with disabilities and sibling psychosocial and behavioral outcomes. Further study is needed to determine whether benefits of each of these sibling supports are also found in other samples.

Outcome Measurement

A number of standardized measures used to determine efficacy were included in the studies. There were no common efficacy measures across studies. In other words, each instrument was used as an indicator of efficacy in only one of the interventions/support programs. Self-esteem and self-concept were measured using the Piers-Harris Children's Self-Concept Scale (Piers and Harris 1969), the Self-Perception Profile for Children (Harter 1985), and the Self-Esteem Questionnaire (DuBois et al. 1996). Sibling psychological well-being was measured using the Child Depression Inventory (Kovacs 1992) and the Children's Manifest Anxiety Scale-Revised (Reynolds and Richmond 1985). Sibling behavior problems

were measured using the Eyberg Child Behavior Inventory (Eyberg and Robinson 1983) and the Child Behavior Checklist (Achenbach and Edelbrock 1983). Sibling social support was measured with the Social Support Scale for Children (Harter 1985) and the Perceived Social Support Scale-Revised (Procidino and Heller 1983). A number of different measures of sibling knowledge of, attitudes about, and relationship toward their brother or sister with the disability were used to determine efficacy, many of which appear to be developed for that specific research project. Exceptions include the Autism Knowledge Measure for Young Children (Perry 1989), the Sibling Relationships Questionnaire (Furman and Buhrmester 1985), and the Sibling Perception Questionnaire (Hodapp et al. 1997; Sahler and Carpenter 1989).

Qualifications of Treatment Providers

Just over one-half of the sibling supports have no specified qualifications for treatment providers except for training in that specific support program. The exceptions are the ISEE program, which uses pediatric nurse clinicians to deliver treatment, and the program for socioeconomically disadvantaged children, which require team leaders to have a minimum of 3 years of experience working with families of children with developmental disabilities and at least a high school education. The SibLink program was delivered by two doctoral-level trainees in psychology or psychiatry for each sibling group.

See Also

- Family Burden
- Family Therapy

References and Reading

Achenbach, T. M., & Edelbrock, C. S. (1983). *Manual for the child behavior checklist and revised child behavior profile*. Burlington: University of Vermont, Department of Psychiatry.

- Conway, S., & Meyer, D. (2008). Developing support for siblings of young people with disabilities. *Support for Learning*, 23, 113–117.
- D'Arcy, F., Flynn, J., McCarthy, Y., O'Connor, C., & Tierney, E. (2005). Sibshops: An evaluation of an interagency model. *Journal of Intellectual Disabilities*, 9, 43–57.
- DuBois, D. L., Felner, R. D., Brand, S., & Phillips, R. S. C. (1996). Early adolescent self-esteem: A developmental-ecological framework and assessment strategy. *Journal of Research on Adolescence*, 6, 543–579.
- Eyberg, S., & Robinson, E. (1983). Conduct problem behavior: Standardization of a behavior rating scale with adolescents. *Journal of Clinical Child Psychology*, 12, 347–354.
- Furman, W., & Buhrmester, D. (1985). Children's perception of the qualities of sibling relationships. *Child Development*, 56, 448–461.
- Harter, S. (1985). *Manual for the social support scale for children*. Denver: University of Denver.
- Hodapp, R. M., Wijma, C. A., & Masino, L. L. (1997). Families of children with 5p- (cri du chat) syndrome: Familial stress and sibling reactions. *Developmental Medicine and Child Neurology*, 39, 757–761.
- Johnson, A. B., & Sandall, S. (2005). *Sibshops: A follow-up of participants of a siblings support program*. Seattle: University of Washington.
- Kovacs, M. (1992). *Manual for the children's depression inventory*. North Tonawanda: Multi-Health Systems.
- Lobato, D. (1983). Siblings of handicapped children: A review. *Journal of Autism and Developmental Disorders*, 13, 347–364.
- Lobato, D. J., & Kao, B. T. (2002). Integrated sibling-parent group intervention to improve sibling knowledge and adjustment to chronic illness and disability. *Journal of Pediatric Psychology*, 27, 711–716.
- Meyer, D. J., & Vadasy, P. F. (1994). *Sibshops: Workshops for siblings of children with special needs*. Baltimore: Paul H. Brookes.
- Perry, A. (1989). *Autism knowledge measure for young children*. Unpublished. Thistleton Regional Centre, Toronto.
- Phillips, R. S. C. (1999). Intervention with siblings of children with developmental disabilities from economically disadvantaged families. *Families in Society*, 80, 569–577.
- Piers, E. V., & Harris, D. B. (1969). *The Piers-Harris children's self-concept scale*. Nashville: Counselor Recordings and Tests.
- Procidino, M. E., & Heller, K. (1983). Measures of perceived social support from friends and from family: Three validation studies. *American Journal of Community Psychology*, 11, 1–24.
- Reynolds, C. R., & Richmond, B. O. (1985). *Revised children's manifest anxiety scale (RCMAS)*. Los Angeles: Western Psychological Services.
- Sahler, O. J. Z., & Carpenter, P. J. (1989). Evaluation of a camp program for siblings of children with cancer. *American Journal of Diseases of Childhood*, 143, 690–696.

- Smith, T., & Perry, A. (2004). A sibling support group for brothers and sisters of children with autism. *Journal on Developmental Disabilities, 11*, 77–88.
- Williams, P. D., Williams, A. R., Graff, C., Hanson, S., Stanton, A., Hafeman, C., et al. (2003). A community-based intervention for siblings and parents of children with chronic illness or disability: The ISEE study. *Journal of Pediatrics, 143*, 386–393.

Siemerling-Creutzfeldt Disease

- [Adrenoleukodystrophy](#)

Sign Language

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Synonyms

[American sign language \(ASL\)](#); [Conceptually accurate signed English \(CASE\)](#); [Linguistics of verbal English \(LOVE\)](#); [Manual sign](#); [Manually coded English \(MCE\)](#); [Seeing essential English \(SEE I\)](#); [Signed English](#); [Signing exact English \(SEE II\)](#)

Definition

Sign language is a system of communicating visually and spatially through signs created with the hands. The term “sign language” does not specify a particular sign system such as American Sign Language (ASL) or manual codes of English such as Seeing Essential English (SEE I), Signing Exact English (SEE II), Linguistics of Verbal English (LOVE), and Conceptually Accurate Signed English (CASE).

However, the word “language” denotes a rule-governed system of communication and not a code for a language. ASL is the natural language of the deaf community in the United States and much of Canada (Neidel et al. 2000). ASL is a distinct language from spoken English, while manual codes of English are based on spoken English and are an attempt to represent English on the hands. Typically, when signing is employed in the language intervention for children with autism, the type of signing systems that are used are manual codes of English rather than ASL. The main reason for this may be the hope that using sign language will lead to verbal communication, an outcome which has been supported by research (Schlosser and Wendt 2008).

See Also

- [American Sign Language \(ASL\)](#)
► [Manual Sign](#)

References and Reading

- Neidel, C., Kegl, J., MacLaughlin, C., Bahan, B., & Lee, R. G. (2000). *The syntax of American sign language*. Cambridge, MA: The MIT Press.
- Schlosser, R. W., & Wendt, O. (2008). Effects of augmentative and alternative communication intervention on speech production in children with autism: A systematic review. *American Journal of Speech-Language Pathology, 17*, 212–230.

Signed English

- [Manual Sign](#)
► [Sign Language](#)

Signing Exact English (SEE II)

- [Manual Sign](#)
► [Sign Language](#)

Siladryl Allergy [OTC]

► [Diphenhydramine](#)

Silenor

► [Adapin](#)

Silphen Cough [OTC]

► [Diphenhydramine](#)

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Name and Degrees

Simon Baron-Cohen, PhD, New College Oxford
BA and an MPhil in Clinical Psychology at the
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Major Appointments (Institution, Location, Dates)

Simon Baron-Cohen is Professor of Developmental
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low at Trinity College, Cambridge. He is Director,
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lege London (UCL), and an MPhil in Clinical

Psychology at the Institute of Psychiatry, London,
and has held lectureships in these departments.
Baron-Cohen is a Fellow of the British Academy.

Major Honors and Awards

Baron-Cohen has received many awards. These
include awards from the British Psychological
Society (BPS) (Spearman Medal and May
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Association (McCandless Award), and the Phila-
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Kanner-Asperger Medal from the German Society
for Research into Autism.

Baron-Cohen is a Fellow of the British and
American Psychological Associations and of the
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National Autistic Society, Autism Anglia, and
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Association and Vice-President, International
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Chair of the NICE Guideline Development Group
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Landmark Clinical, Scientific, and Professional Contributions

Among his many publications, Baron-Cohen is
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edited scholarly anthologies including *Under-
standing Other Minds*, *Synaesthesia*, and *The*

Maladapted Mind. He has written books for parents and teachers including *Autism and Asperger Syndrome: The Facts*, and *Teaching Children with Autism to Mind-read*. He has celebrated autism in *An Exact Mind*. He is author of the DVDs *Mind Reading* and *The Transporters*, to help children with autism learn emotion recognition, both nominated for BAFTA awards.

Short Biography

Baron-Cohen has a long history in the study of Autism Spectrum Conditions (autism) and is the author of more than 450 scientific articles. In 1985 he formulated and went on to test the “mindblindness” theory of autism in one of his many landmark papers. Baron-Cohen devised a puppet paradigm, the Sally-Anne test, to show that children with autism were impaired in the ability to attribute a false belief to others. This began a rich body of research, which demonstrated that children and adults with autism had fundamental difficulties with tasks that required a “theory of mind.” As part of this, he developed a number of measures of theory of mind as well as other dimensions of cognition, such as systemizing and empathizing, which have been used extensively in both research and clinical settings. In 1997, he formulated and went on to test the “fetal sex steroid” theory of autism. Among his other contributions to the field of autism, include studies of prevalence and screening, autism genetics, autism neuroimaging, autism and technical ability, typical cognitive sex differences, and synesthesia. As part of his research, Baron-Cohen has supervised more than 30 PhD theses.

Baron-Cohen also has a strong history in the clinical care of individuals with autism, with a particular focus on adults with Asperger Syndrome. In 1999 he created the first UK clinic for adults with suspected Asperger Syndrome, called the CLASS clinic (Cambridge Lifespan Asperger Syndrome Service), at a time when the National Health Service (NHS, UK) did not see the clinical need for this. This has helped over 1,000 patients to have their disability recognized, the “lost generation” of adults who had missed out on diagnosis in childhood, and has been used to create a

model for similar clinical services all over the UK. As part of this clinical work, he developed both a screening questionnaire (the Autism Spectrum Quotient, or AQ) and diagnostic instrument (the Adult Asperger Assessment, AAA). In 2016 he appeared in the BBC2 documentary *Employable Me*, revealing the remarkable strengths in people with autism and discussing how to promote inclusion. He has also written of the need to conceptualize autism as a difference rather than disability, a conceptual shift strongly supported by individuals with autism and their advocates, and he coined the term autism spectrum conditions, instead of autism spectrum disorders, to reflect this paradigm shift.

See Also

- [Brain Connectivity Theories of Autism](#)
- [Theory of Mind](#)
- [Uta Frith](#)

References and Reading

- Baron-Cohen, S. (1989). The autistic child's theory of mind: A case of specific developmental delay. *Journal of Child Psychology and Psychiatry*, 30(2), 285–297.
- Baron-Cohen, S., Wheelwright, S., Cox, A., Baird, G., Charman, T., Swettenham, J., Drew, A., & Doehring, P. (2000). The early identification of autism: The checklist for autism in toddlers (CHAT). *The Journal of Developmental and Learning Disorders*, 4, 3–30.
- Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The “reading the mind in the eyes” test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry*, 42(2), 241–252.
- Baron-Cohen, S., Knickmeyer, R. C., & Belmonte, M. K. (2005). Sex differences in the brain: Implications for explaining autism. *Science*, 310(5749), 819–823.
- Baron-Cohen, S., Lombardo, M. V., Auyeung, B., Ashwin, E., Chakrabarti, B., & Knickmeyer, R. (2011). Why are autism spectrum conditions more prevalent in males? *PLoS Biology*, 9(6), e1001081.
- Baron-Cohen, S., Johnson, D., Asher, J., Wheelwright, S., Fisher, S. E., Gregersen, P. K., & Allison, C. (2013). Is synaesthesia more common in autism? *Molecular Autism*, 4(1), 40.
- Baron-Cohen, S., Auyeung, B., Nørgaard-Pedersen, B., Hougaard, D. M., Abdallah, M. W., Melgaard, L.,

- Cohen, A. S., Chakrabarti, B., Ruta, L., & Lombardo, M. V. (2015). Elevated fetal steroidogenic activity in autism. *Molecular Psychiatry*, 20, 369–376.
- Chakrabarti, B., Kent, L., Suckling, J., Bullmore, E., & Baron-Cohen, S. (2006). Variations in the human cannabinoid receptor (CNR1) gene modulate striatal response to happy faces. *European Journal of Neuroscience*, 23, 1944–1948.
- Lai, M. C., Lombardo, M. V., Suckling, J., Ruigrok, A. N., Chakrabarti, B., Ecker, C., Deoni, S. C., Craig, M. C., Murphy, D. G., Bullmore, E. T., MRC AIMS Consortium, & Baron-Cohen, S. (2013). Biological sex affects the neurobiology of autism. *Brain*, 136(Pt 9), 2799–2815.
- Lai, M.-C., Lombardo, M., & Baron-Cohen, S. (2014). Autism. *The Lancet*, 383, 896–910.
- Schwarz, E., Guest, P., Rahmoune, H., Wang, L., Levin, Y., Ingudomnukul, E., Ruta, L., Kent, L., Spain, M., Baron-Cohen, S., & Bahn, S. (2011). Sex-specific serum biomarker patterns in adults with Asperger's syndrome. *Molecular Psychiatry*, 16, 1213–1220.
- Warrier, V., Baron-Cohen, S., & Chakrabarti, B. (2013). Genetic variation in GABRB3 is associated with Asperger syndrome and multiple endophenotypes relevant to autism. *Molecular Autism*, 4(1), 48.
- Welchew, D., Ashwin, C., Berkouk, K., Salvador, R., Suckling, J., Baron-Cohen, S., & Bullmore, E. (2005). Functional dysconnectivity of the medial temporal lobe in autism. *Biological Psychiatry*, 57, 991–998.

Simple Phobia

► Phobia

Simplified Chinese Psychoeducational Profile Third Edition

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Synonyms

CPEP-3: Chinese Psychoeducational Profile Third Edition

Description

The Simplified Chinese Psychoeducational Profile Third Edition (sCPEP-3) is a comprehensive

measurement tool developed for the assessment of Chinese children with autism spectrum disorder (ASD) in mainland China. The instrument can be used to assess children aged between 6 months and 7 years old. The sCPEP-3 has two major components: the Performance Test and the Caregiver Report. The 172-item Performance Test consists of ten subtests administered by professionals based on direct observation and testing. The first six subtests focus on a child's developmental abilities, among which three subtests measure communication ability, Cognitive Verbal/Preverbal (34 items), Expressive Language (25 items), and Receptive Language (19 items), and another three subtests measure motor ability: Fine Motor (20 items), Gross Motor (15 items), and Visual-Motor imitation (10 items). The remaining four subtests of the Performance Test measure maladaptive behaviors including Affective Expression (11 items), Social Reciprocity (12 items), characteristics motor behaviors (15 items), and characteristics verbal behaviors (11 items). The 38-item Caregiver Report is completed by parents or major caregivers (e.g., parents, teacher, or guardian) based on their daily observations of the child in natural settings. The Caregiver Report contains three subtests, which measure Problem Behavior (10 items), Personal Self-Care (13 items), and Adaptive Behaviors (15 items), respectively. The scoring of items has been quantified with "Fail" = 0, "Emerging" = 1, and "Pass" = 2.

The sCPEP-3 covers both key developmental areas and maladaptive behaviors related to the diagnosis of ASD and thus can be used to measure the developmental strengths, weaknesses, and learning style of a child with ASD. The results constitute a comprehensive evaluation profile of the child, which helps therapists and professionals set up individualized treatment strategies and educational programs according to the child's unique characteristics (Schopler et al. 2005). In particular, the Caregiver Report in the sCPEP-3 gathers information about a child's self-care skills, behavioral problems, and adaptive behaviors based on the caregiver's daily observation of the child's performance. Several advantages of the Caregiver Report need to be noted. First, caregivers' evaluation of the child concerned is based on real-life experience over

a long period of time, which can well supplement the results of the assessment conducted by professionals within a short period of time. Second, children's behavioral problems, personal self-care skills, and adaptive behaviors reported by caregivers serve as important information for researchers to gain a holistic understanding of children with ASD. Third, the gathered information about the child's daily routine is important for the development of individualized intervention plan (Shek and Yu 2015). The Caregiver Report is usually conducted before the administration of the assessment by therapists.

Historical Background

To design and implement effective and early treatment strategies for children with ASD, it is necessary to have a comprehensive and accurate evaluation of the child's developmental strengths and deficits. An ideal assessment should have the following critical components. First, the assessment should be addressing all of the major areas of the child's development. Second, the assessment should be age-normed for use in early childhood to indicate the progression of skill development. Third, the items should be directly linked to the program targets. Fourth, the assessment should yield results to guide the development of a structured treatment program. Last but not least, the assessment should be able to track and monitor the child's development in key areas over time (Gould et al. 2011).

To this end, Schopler and Reichler (1979) first developed the Psychoeducational Profile (PEP) to assess children with ASD from preschool age to elementary age (i.e., 2–7 years old). The PEP was also intended to be used as a tool for planning and developing individualized educational programs within the TEACCH program. This instrument incorporates children's behavioral and developmental information, emphasizes the connection between the developmental delays of children with ASD and their atypical features, and provides a useful framework for researchers and practitioners to assess the development of children with ASD (Coonrod and Marcus 2013). In 1990, PEP was revised into the PEP-Revised (PEP-R)

(Schopler et al. 1990). Compared with the PEP, the PEP-R added items related to the developmental features for underage children. The language functioning sections and items related to problem behaviors of children with ASD were also expanded and updated. Another main characteristic of PEP-R is its easy and flexible administration, which further facilitates practitioners to use the tool in developing personalized treatment strategies for children. The PEP-R has been translated into several languages and used in different cultures, such as Chinese (Ka-ting Lam and Rao 1993; Shek et al. 2005), Dutch (Steerneman et al. 1997), and Brazilian (De Leon et al. 2004). In 2005, the PEP-R was revised into the PEP-third (PEP-3), which further updated the behavioral items corresponding to the latest research findings. The PEP-3 utilized more simple and concrete materials and instructions than the previous version, which makes the instrument more suitable for children with ASD aged between 6 months to 7 years old. The newly added Caregiver Report, a questionnaire completed by caregiver, provides a more comprehensive picture for the special learning strengths and weakness of children with ASD and helps practitioners, professionals, and researchers to formulate suitable and tailored intervention strategies and plan (Schopler et al. 2005).

The PEP-3 was first translated into Mandarin Chinese in Taiwan based on the guidelines for cross-cultural adaptation of instruments (Wild et al. 2005). Researchers made some modifications of the items such as reading English letters, words, sentences, and stories based on the Chinese culture in Taiwan context (Fu et al. 2010). The psychometric properties of this version of the PEP-3 were examined based on relatively small samples of children in Taiwan (Chen et al. 2011; Fu et al. 2010). In 2013, Heep Hong Society, the biggest organization in Hong Kong that provides professional training and education to children with developmental and learning problems and their families, invited a group of experts consisting of psychologists, occupational therapists, special educators, and special child-care workers to translate the PEP-3 into Traditional Chinese and conducted a validation study to examine the psychometric properties of the

instrument (Shek and Yu 2014, 2015). Since then, the CPEP-3 has been widely adopted by helping professionals and researchers working with children with ASD in Hong Kong. So far, the CPEP-3 has been used as an important educational assessment in more than 50 child centers to guide practitioners' development and evaluation of individualized educational plans for children with ASD.

More recently, researchers further converted the CPEP-3 into a Simplified Chinese version (sCPEP-3) for its use in mainland China (Yu et al. 2019). Although Hong Kong and mainland China are all Chinese-speaking regions, they still have significant language, culture, and lived experiences differences. In particular, Mandarin and Simplified Chinese characters are widely used in most areas of mainland China, and Cantonese and Traditional Chinese characters are employed in Hong Kong. Not only the pronunciations of Mandarin and Cantonese but also the vocabulary and grammar of written Chinese used in these two regions are different. Considering the language and cultural differences, bilingual researchers first reviewed the converted sCPEP-3 and made minor modifications to the wordings of a few items. A few more culturally suitable objects were further adopted to replace the original ones. Finally, a bilingual expert team examined the sCPEP-3 and compared with the original CPEP-3 to ensure that the two versions are conceptually equivalent. To provide empirical evidence for the application of the sCPEP-3 in the context of mainland China, a validation study that examined the psychometric properties of the sCPEP-3 was conducted (Yu et al. 2019).

Psychometric Data

Given the advantages of CPEP-3, scholars have conducted validation studies in different Chinese contexts. In Taiwan, evidence on the reliability and validity of both the Performance Test and the Caregiver Report has been reported. Based on samples of children with ASD, researchers

reported high internal consistency (Cronbach's alpha coefficients ranged between 0.92 and 0.98), good to excellent inter-rater reliability, and test-retest reliability (Fu et al. 2010, 2012) of the Chinese Psychoeducational Profile Third Edition (CPEP-3).

In Hong Kong, several studies were carried out and published, which provided comprehensive psychometric data for the traditional Chinese version of the CPEP-3 (Shek and Yu 2014, 2015, 2018). Specifically, with a normative sample of 455 children with ASD and a comparison group of 281 children with typical development, researchers examined the construct validity of the CPEP-3. It was found that older children scored higher than younger children for both groups on different subsets of CPEP-3, while no gender difference was identified within the group with ASD. The results of the confirmatory factor analysis supported the three-factor theoretical model of the Performance Test. Shek and Yu (2018) further reported the test-retest reliability, inter-rater reliability, and criterion-related validity of CPEP-3 Performance Test. The Cronbach's alpha coefficients ranged from 0.89 to 0.97, the test-retest correlation coefficients were all above 0.82, and the inter-rater reliability ranged between 0.34 and 0.87. It was also found that the CPEP-3 Performance Test subtests were positively correlated with the Merrill-Palmer Revised Scales of Development (MPR) while negatively correlated with subscale scores of the Childhood Autism Rating Scale (CARS). These results suggest good criterion-prediction validity of the CPEP-3 Performance Test. With regard to the Caregiver Report, Cronbach's alpha coefficients ranged from 0.65 to 0.89, and the test-retest reliability was between 0.82 and 0.97 (Shek and Yu 2015). For validity, CPEP-3 Caregiver Report subscale scores were positively correlated with the Hong Kong Based Adaptive Behavior Scale (HABS), which measures similar social-cognitive and behavioral functioning constructs (Shek and Yu 2015). The findings suggest that CPEP-3 is a psychometrically sound instrument for the assessment of children with ASD in Hong Kong.

In 2018, a large-scale validation study on the sCPEP-3 was conducted in a representative sample of children in mainland China (Yu et al. 2019). A total of 554 Chinese children with ASD and 311 typically developing Chinese children participated in this study. The results showed that Cronbach's alpha coefficients ranged from 0.77 to 0.96 for the Performance Test subscales and from 0.80 to 0.86 for the Caregiver Report subtests. Mean inter-item correlation coefficients equaled to or exceeded 0.29 for all subscales, suggesting good internal consistency of the sCPEP-3. However, it was noted by the researchers that Cronbach's alpha coefficients for three subtests (Cognitive Verbal/Preverbal, Expressive Language, and Receptive Language) are 0.95 or higher, indicating that some items in these subscales may be redundant. All test-retest correlation coefficients were between 0.73 and 0.98, indicating good time sampling reliability of the instrument. The mean and median polychoric correlation coefficients for all subtests of the Performance Test ranged from 0.64 to 0.93, which provides support for the inter-rater reliability.

Convergent validity, construct validity, and factorial validity of the sCPEP-3 were examined in the study. First, correlation coefficients between children's scores on the sCPEP-3 subtests and three other questionnaires, Gesell Developmental Observation-Revised (GDO-R), Vineland Adaptive Behavior Scale (VABS), and Childhood Autism Rating Scale (CARS), were calculated. The findings support good convergent validity of the sCPEP-3. Second, no significant gender difference within the ASD group was found in the participants' scores of sCPEP-3, while children in the control group scored significantly higher than the children with ASD on all subtests, which provided solid evidence for the construct validity of the sCPEP-3. Third, a confirmatory factor analysis (CFA) was carried out to test the factorial validity of the sCPEP-3. Model fitting results support the three-dimensional theoretical model of the Performance subtest: communication, motor, and maladaptive behaviors. Based on these findings, it is concluded that the sCPEP-3 is a

psychometrically sound measure for researchers, practitioners, and professionals to assess and monitor the development of children with ASD in mainland China.

Clinical Uses

The sCPEP-3 is a promising tool that can be used by professionals to chart the development of Chinese children with ASD aged between 6 months and 7 years old in a comprehensive manner. The satisfactory psychometric properties of the sCPEP-3 suggest that this instrument can offer reliable and valid evaluative information for researchers and practitioners working with children with ASD in mainland China.

Several practical implications are worth noting. First, a comprehensive evaluation of children with ASD should base on the standardized observation scores from professionals, as well as the information provided by children's caregiver, such as parents, guardian, or teacher (Huerta and Lord 2012). The sCPEP-3, which contains a formal Performance Test and a Caregiver Report, enables the researchers and clinicians to integrate information from different sources; to obtain detailed data and an overall pattern of children's developmental strength, weakness, learning styles, maladaptive behaviors, and interests; and to set up personalized educational plan and educational strategies (Yu et al. 2019). Second, Chinese clinicians can use the sCPEP-3 in combination with other diagnostic instruments to improve the diagnostic accuracy of ASD and reduce the rate of unrecognized cases in mainland China. Although several ASD screening tools and diagnostic instruments have been utilized and validated for Mandarin-speaking children (Li et al. 2005; Yin et al. 2011; Zhou et al. 2017), validation studies are limited in the context of mainland China. The use of the validated sCPEP-3 can facilitate the development of structured treatment programs and promote the provision of individualized educational programs for Chinese children with ASD (Yu et al. 2019). Finally, the sCPEP-3 can serve as a credible and valid outcome measure

to evaluate the treatment effects of ASD program in China.

See Also

- [Childhood Autism Rating Scale](#)
- [Clinical Assessment](#)
- [Psychoeducational Profile – Revised \(PEP-3\)](#)

References and Reading

- Chen, K. L., Chiang, F. M., Tseng, M. H., Fu, C. P., & Hsieh, C. L. (2011). Responsiveness of the psychoeducational profile-for children with autism spectrum disorders. *Journal of autism and developmental disorders*, 41(12), 1658–1664.
- Coonrod, E., & Marcus, L. (2013). Psychoeducational Profile – Revised (PEP-3). In F. R. Volkmar (Ed.), *Encyclopedia of autism spectrum disorders* (pp. 2439–2444). New York: Springer.
- De Leon, V., Bosa, C., Hugo, C., & Hutz, C. S. (2004). Psychometric properties of the Psychoeducational Profile Revised: PEP-R. *Avaliação Psicológica*, 3(1), 39–52.
- Fu, C. P., Hsieh, C. L., Tseng, M. H., Chen, Y. L., Huang, W. T., Wu, P. C., & Chiang, F. M. (2010). Inter-rater reliability and smallest real difference of the Chinese psychoeducational profile-for children with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 4(1), 89–94.
- Fu, C. P., Chen, K. L., Tseng, M. H., Chiang, F. M., & Hsieh, C. L. (2012). Reliability and validity of the psychoeducational profile-caregiver report in children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6(1), 115–122.
- Gould, E., Dixon, D. R., Najdowski, A. C., Smith, M. N., & Tarbox, J. (2011). A review of assessments for determining the content of early intensive behavioral intervention programs for autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5(3), 990–1002.
- Huerta, M., & Lord, C. (2012). Diagnostic evaluation of autism spectrum disorders. *Pediatric Clinics of North America*, 59(1), 103.
- Lam, M., & Rao, N. (1993). Developing a Chinese version of the Psychoeducational Profile (CPEP) to assess autistic children in Hong Kong. *Journal of Autism and Developmental Disorders*, 23(2), 273–279.
- Li, J. H., Zhong, J. M., Cai, L. Y., Chen, Y., & Zhou, M. Z. (2005). Comparison of clinical application of three autism rating scale. *Chinese J Contemporary Pediatrics*, 7(1), 59–62.
- Schopler, E., & Reichler, R. J. (1979). *Individual assessment and treatment for autistic and developmental disabled children: Psychoeducational profile* (Vol. 1). Baltimore: University Park Press.
- Schopler, E., Reichler, R. J., & Renner, B. R. (1988). *The childhood autism rating scale (CARS)*. Los Angeles: Western Psychological Services.
- Schopler, E., Reichler, R. J., Bashford, A., Lansing, M. D., & Marcus, L. M. (1990). *Individualized assessment of autistic and developmentally disabled children: Psychoeducational Profile Revised (PEP-R)*. Austin: Pro-Ed.
- Schopler, E., Lansing, M. D., Reichler, R. J., & Marcus, L. M. (2005). *Psychoeducational profile: TEACCH individualized psychoeducational assessment for children with autism spectrum disorders* (3rd ed.). Austin: Pro-Ed.
- Shek, D. T. L., & Yu, L. (2014). Construct validity of the Chinese version of the Psycho-Educational Profile-(CPEP-3). *Journal of Autism and Developmental Disorders*, 44(11), 2832–2843.
- Shek, D. T. L., & Yu, L. (2015). Psychometric properties of the Chinese version of the psychoeducational profile—third edition—caregiver report. *Journal of Intellectual and Developmental Disability*, 40(4), 321–329.
- Shek, D. T. L., & Yu, L. (2018). Reliability and validity of the Chinese version of the psycho-educational profile performance test. *International Public Health Journal*, 10(1), 43–56.
- Shek, D. T. L., Tsang, S. K. M., Lam, L. L., Tang, F. L. Y., & Cheung, P. M. P. (2005). Psychometric properties of the Chinese version of the Psycho-Educational Profile-Revised (CPEP-R). *Journal of Autism and Developmental Disorders*, 35(1), 37–44.
- Steerneman, P., Muris, P., Merckelbach, H., & Willems, H. (1997). Assessment of development and abnormal behavior in children with pervasive disorders. Evidence for the reliability and validity of the revised psychoeducational profile. *Journal of Autism and Developmental Disorders*, 27(2), 177–185.
- Wild, D., Grove, A., Martin, M., Eremenco, S., McElroy, S., Verjee-Lorenz, A., & Erikson, P. (2005). Principles of good practice for the translation and cultural adaptation process for patient-reported outcomes (PRO) measures: Report of the ISPOR Task Force for Translation and Cultural Adaptation. *Value in Health*, 8(2), 94–104.
- Yin, Q. Y., Chen, J. M., Luo, X. R., & Li, X. R. (2011). Reliability and validity of two autism rating scales. *International Medicine and Health Guidance News*, 17(12), 1470–1475.
- Yu, L., Zhu, X., Shek, D. T., Zou, X. B., Deng, H. Z., & Yeung, P. W. A. (2019). Validation of the simplified Chinese psychoeducational profile third edition in Mainland China. *Journal of Autism and Developmental Disorders*, 49(4), 1599–1612.
- Zhou, H., Zhang, L., Luo, X., Wu, L., Zou, X., Xia, K., . . . & Fombonne, E. (2017). Modifying the autism spectrum rating scale (6–18 years) to a Chinese context: An exploratory factor analysis. *Neuroscience Bulletin*, 33(2), 175–182.

Simply Sleep™ [OTC]

► [Diphenhydramine](#)

Simpson-Angus Scale for Extrapyramidal Symptoms

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Definition

The Simpson-Angus Rating Scale is a 10-item scale used to rate adverse neurological effects of antipsychotic medications more broadly. It involves direct observation and a brief neurological examination.

For example, the rating requires the clinician to observe the patient's gait and check for tremor, excessive salivation, as well as rigidity in the arms, shoulder, and neck.

Each item is rated from 0 to 4 and a total score can be obtained.

See Also

► [Antipsychotics: Drugs](#)

References and Reading

Simpson, G. M., & Angus, J. W. S. (1970). A rating scale for extrapyramidal side effect. *Acta Psychiatrica Scandinavica*, 212(Suppl), 11–19.

Sinequan

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Synonyms

[Doxepin](#)

Definition

Sinequan (doxepin) is a tricyclic antidepressant medication that has largely fallen out of use as an antidepressant. It is approved for the treatment of adults with insomnia – especially midsleep awakening. It is associated with multiple adverse effects including poor coordination, confusion, and increased heart rate and also has a potential for cardiac arrhythmia. Other adverse effects include dry mouth, urinary retention, and constipation. There are no studies of the use of doxepin in children or adults with autism spectrum disorders.

See Also

► [Adapin](#)

References and Reading

Martin, A., Scahill, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford University Press.
McDougle, C., & Posey, D. J. (2011). Assessment and treatment of autistic and other pervasive developmental disorders. In A. Martin, L. Scahill, & C. Kratochvil (Eds.), *Pediatric psychopharmacology: Principles and practice* (pp. 547–560). New York: Oxford University Press.

Singapore and Autism Spectrum Disorder

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Singapore is a young island city-state in South-east Asia with a diverse population of approximately 5.7 million (<https://www.singstat.gov.sg/modules/infographics/population>). Ethnic Chinese constitute approximately 76% of the residents, followed by ethnic Malays (13.6%), Indians (9.4%), and others (3.4%). Its relatively brief history as an independent state has been characterized by considerable economical and capital growth, four official languages (English, Mandarin, Malay, and Tamil; English is the official language of business, administration, and school instruction), an internationally strong and competitive educational system, as well as governmental and community visions of Singapore as an inclusive society (Lee 2004). It is within this context that this paper seeks to present and summarize the history and current development of services, education, and other

initiatives for individuals with autism spectrum disorder (ASD) and their families.

Legal Issues/Mandates for Service

Singapore signed the UN Convention on the Rights of Persons with Disabilities in November 2012 and ratified it in August 2013. Service development and provisions for individuals with disabilities in Singapore have been guided by national-level committees and advisory groups. The Enabling Masterplans (EMs) for the disability sector (EM 2007–2011, EM 2012–2016, and EM 2017–2021) are comprehensive disability-specific policy documents designed to chart the development of programs and services for people with disabilities in the public sector and aimed to enhance the integration of persons with disabilities in Singapore and maximize their potential for independent living. The EMs have specifically included ASD under developmental disorders in their definition of disability. ASD is also stated as one of the seven disability types in the Singapore government's classification of disability.

The EMs identified national priorities and guided the development of services to address these priorities. Of relevance to individuals with ASD are the following recommendations of the first Enabling Masterplan: the development and maintenance of quality programs and staff, the empowerment of family caregivers through education and training, transition planning, and the integration of students with disabilities in the education system that may bridge the gap between special and mainstream curriculum. The second Enabling Masterplan (2012–2016) prioritized the early identification of disabilities in community settings, the introduction of an additional national child screening appointment with the pediatrician at 24–30 months, the development and evaluation of inclusion strategies within preschool settings, the improved coordination of services across the life-span, and the development of a national database of individuals with disabilities (Enabling Masterplan 2012–2016 Steering Committee 2012; see also Poon and Lim 2012). The most recent 2017–2021 Enabling Masterplan

acknowledges the increase in the number of individuals receiving a diagnosis of ASD and the consequential need to provide more supports and services. It also prioritizes the development of services for a more inclusive society with lifelong educational and employment opportunities for all persons with disabilities and increasing supports for their caregivers and for people with autism and other disabilities as they grow older.

While the EMs provide guidance for the general disability sector, the needs of different disability groups are not homogenous. As such, the Autism Resource Centre (Singapore), with support from the Autism Network Singapore (ANS), embarked on a project to develop an Autism Enabling Masterplan (AEM) in 2018. The AEM aims to identify needs and gaps that are specific to the autism community, prioritizing and proposing solutions thereof. The goal is to provide a directional roadmap to guide the work in Singapore with a focus on empowering and enabling persons on the autism spectrum to realize their potential and live a good quality life. High-priority areas include employment, residential living options, and future care planning. The AEM is targeted to be published by end 2020.

Historical Background

There are currently no formal published studies on the prevalence of disability in Singapore. However, it has been estimated that approximately 3.2% of young children (0–6 years), 2.5% of school-aged children (7–18 years), and 2.5% of adults have some form of disability in the city-state (Enabling Masterplan 2012–2016 Steering Committee 2012). Of the preschool children referred to the National Child Development Programme (which serves young children presenting with developmental concerns), about a quarter (20–25%) present with features of ASD and a third (30–35%) with speech and language delays and associated disorders (Ho 2018). With increasing numbers of children with special educational needs being identified within the national educational system, the incidence of older children and adults with disabilities is likely to increase.

The Child Psychiatric Clinic (now known as the Child Guidance Clinic) at the Woodbridge Hospital (now known as the Woodbridge Hospital/Institute of Mental Health (IMH)) first identified and clinically diagnosed cases of autism (then termed infantile psychosis) in the 1970s. The first ASD-specific special education program was established in 1989 at Margaret Drive Special School, subsequently run by Rainbow Centre Singapore, serving both preschool and school-aged children with ASD (Poon et al. 2016). In 2004, Pathlight School was established as an ASD-specific school for young people on the spectrum who are able to access the mainstream curriculum with appropriate specialist teaching and in a ASD-specific learning environment (see section on “Educational Provisions for School Going Children ” below for more details).

Overview of Current Services, Centers, and Interventions

Diagnostic and Clinical Services

Around 1989, Dr. Vera Bernard-Opitz, a Senior Lecturer, joined the Department of Psychology at the National University of Singapore and volunteered at Margaret Drive Special School (Bernard-Opitz et al. 2001a). Shortly after, the Developmental Assessment Clinic was established in 1991 at the Singapore General Hospital (Ho 2007). This tertiary-level diagnostic facility for preschool children subsequently led to the development of a nationwide Child Development Programme funded by the Ministry of Health in 2002 in collaboration with two of the leading public hospitals in Singapore (National University Hospital (NUH) and KK Women and Children's Hospital (KKH)), which currently diagnoses and manages the majority of children with disabilities (including ASD) under the age of 7 years in Singapore. Through this program, approximately 1000 new cases of ASD in preschool children are diagnosed yearly (Ho 2018). More than half of Singaporean parents are concerned about their child's early development by the age of 2 years. The average age of ASD diagnosis in Singapore is at 3 years of age, which is at least as early as, and possibly earlier

than, other developed countries (Moh and Magiati 2012; Bernard-Opitz et al. 2001a). Since 1995, the Ministry of Education (MOE) has employed psychologists to provide psychological assessments and advice for students with special educational needs in mainstream primary and secondary schools. The number of students with special needs seen by the MOE psychologists, including those suspected to have ASD, has increased in recent years. As from 2007, REACH teams (Response, Early Intervention and Assessment in Community Mental Health), a community-based multidisciplinary mental healthcare service, have been working collaboratively with school counselors employed in mainstream schools to identify and support students with emotional, social, and behavioral issues, including those with ASD.

The specialist outpatient Neurobehavioral Clinic (Autism Services) was established in 2006 and has been providing diagnostic assessment for 6–19-year-old children and adolescents with suspected ASD and intervention services for those children and adolescents with ASD who present with mental health or behavioral comorbidities. This service is available within the Child Guidance Clinic, at the country’s largest mental health organization, the Institute of Mental Health (IMH). Visits to the Child Psychiatric (now Child Guidance) Clinic for a query of ASD in 7–18-year-old children and young people have increased substantially in recent years (see Fig. 1).

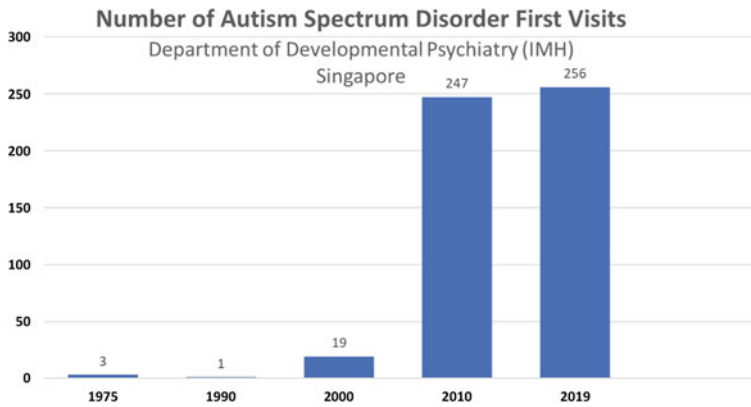
There were, until a few years ago, no specialist clinical services for the assessment, diagnosis, and support for adults with ASD. Since 2011, a

specialist multidisciplinary outpatient neurodevelopmental clinic for adults with intellectual disabilities or ASD and mental health or behavioral challenges (the Adult Neurodevelopmental Disorder Service) has been setup within IMH to support those individuals and their families and provide comprehensive assessment and intervention services. Although a minority of individuals are assessed and diagnosed in private settings, or in non-governmental voluntary welfare organizations (VWOs), the majority of children and young people with ASD are diagnosed at the earlier mentioned public organizations, where multidisciplinary teams (including pediatricians or child psychiatrists, nurses, psychologists, and other allied healthcare professionals) carry out comprehensive interdisciplinary developmental assessments. The Academy of Medicine Singapore-Ministry of Health Singapore Clinical Guidelines for Preschool Children with ASD (2010) recommends that assessment of suspected ASD be carried out by multidisciplinary teams through direct observation of the child; administration of ASD-specific instruments, such as the Autism Diagnostic Observation Schedule (ADOS) and the Autism Diagnostic Interview-Revised (ADI-R); and gathering of ASD-specific developmental information. Recommendations are then made for support, intervention, and management in the community.

Early Intervention

The Academy of Medicine Singapore-Ministry of Health Singapore Clinical Guidelines for Preschool Children with ASD (2010) recommends

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Fig. 1 Number of first visits to the Child Psychiatric/Child Guidance Clinic relating to possible autism/ASD



that all preschool children with ASD be referred at the earliest instance to an early intervention program that provides an individualized intervention plan with comprehensive and targeted interventions using visual strategies and emphasizes the use of augmentative communication systems and direct teaching of social skills.

Early intervention programs in Singapore are supported by the Early Childhood Development Agency (ECDA) from 1 April 2020. This signifies recognition that early intervention (EI) is an integral part of early childhood provision in Singapore. The early intervention provisions consist of a continuum of program types. The Early Intervention Programme for Infant and Children (EIPIC) supports children with moderate to severe developmental needs. There are approximately 3000 children including those with autism who are supported in 21 such centers. Some of these centers serve children with different developmental needs, while others provide specialized programs for children on the autism spectrum. At the other end of the spectrum, the Development Support-Learning Support (DS-LS) programs support children with mild developmental needs within their preschools. Therapists may work with the children and preschool teacher to enhance social communication and interactions, as well as attention and behavioral issues of students in the classroom.

To better support children with developmental needs and their families, EI services have been made more customized and affordable since July 2019. Two new programs have been introduced, namely, (a) EIPIC Under-2s and (b) Development Support (DS)-Plus. The EIPIC Under-2s program provides training to the parent/caregiver to carry out intervention strategies in the child's daily routines within their home setting even if the child does not have a formal diagnosis of ASD. This aims to help parents create learning opportunities throughout the child's daily life.

Development Support Plus (DS-Plus) on the other hand supports children with mild to moderate developmental needs within their preschools, rather than at a separate EI center. EI centers which have launched the enhanced EI programs will identify children for suitable EI programs that

are tailored to their developmental needs, through the use of an Early Intervention Benchmarking Framework. Parents will be kept up-to-date on their child's progress in EI and the child's suitability for a change in the level of intensity of EI, through the child's 6-monthly progress reviews.

Educational Provisions for School Going Children

Formal education in Singapore begins the calendar year as a child turns 7. Children with ASD can enroll in government-funded national mainstream schools or in special education ("SPED") schools. Approximately 26,000 students in mainstream schools are reported to have some form of special educational needs. However, this is based on the number of students who are accessing specialized services. The exact number of students with ASD in mainstream schools is currently not known. In order to support the inclusion of students with special educational needs in mainstream schools, the Ministry of Education (MOE) introduced the Support for Special Needs Initiative in 2005. As part of this effort, all primary and secondary schools are provided with Allied Educators – a learning and behavior support professional – who is trained in supporting the learning and behavioral needs of students with disabilities (such as dyslexia or other specific learning disorders as well as ASD in children without cognitive or verbal impairments) through direct and indirect approaches (i.e., in-class support and pull-out sessions for students, and/or training teachers). In addition, MOE has been providing in-service training to teachers to support students with special educational needs in mainstream schools. MOE is working towards equipping all teachers with baseline competencies to support students with SEN, and about 15% of teachers in every school have received training to equip them with deeper expertise in this area.

Complementing the educational services in mainstream schools are special education (SPED) schools for children who require higher levels of support. There are currently 19 government-funded SPED schools in Singapore, run by 12 Social Service Agencies (SSAs) serving approximately 6000 students. As mentioned, the provision of a

specialized ASD program within special education was first offered in 1989 at Margaret Drive Special School. Between 2004 and 2006, three SPED schools that provide specialized programs only for students with ASD diagnoses were established to meet rising demands in enrolment applications. These are the Pathlight School, the Eden School, and the St Andrew's Autism School. These schools complement the other SPED schools, most of which have ASD-specific programs or educational provisions for students with ASD. As part of the continual effort in enhancing accessibility to special education, more SPED schools are being established. Since 2018, three new ASD-specific SPED schools have begun operations, serving students with ASD with higher support needs, and another four ASD-specific SPED schools are planned from 2021 (Rajah 2019). There is considerable variation over the specific approaches or methods being implemented within the different SPED schools. However, some key characteristics that are shared include a customized structured curriculum, higher teacher-student ratio, collaboration with parents, specialized support from allied educational and health professionals, and a focus on developing communication, functional daily living, and social-emotional skills, with the aim of promoting life-long independence.

Specifically, the Pathlight School was set up by the Autism Resource Centre with government funding for school-going children with ASD who are able to access the mainstream National Curriculum with support, within a specialized learning environment. Apart from school-based education for its primary school students, the Pathlight School also offers a satellite school model for physical, social, and academic integration with mainstream school students for its secondary school students. Pathlight students attend school at the Satellite Partnership (Satellite Partnership program was introduced by the Ministry of Education to improve inclusivity and facilitate the interaction between students from SPED schools and students from 19 selected mainstream schools) mainstream schools daily (physical integration) and interact socially with their students

during recess and selected school events (social integration). Selected students, who have been assessed to be ready, join the mainstream classes for certain lessons (academic integration). This model provides for purposeful and meaningful, but gradual and individualized, participation in mainstream school and social integration.

SPED students in the other SPED schools also access meaningful participation and frequent interactions with their peers in mainstream schools, with the program targeted at vocational preparation, and transition to post-school independent living, learning, and working opportunities. The provision of Transition Planning Coordinators (TPCs) and Job Coaches to every SPED school, serving students aged 13 and above, supports the co-development and enactment of Individual Transition Plans together with the student, parents, and school personnel. The professional development for all staff, including that for TPCs, Job Coaches, and teachers, is funded by the government over and above the government's funding for all SPED school staff, operations, and infrastructure.

Services and Support for Adults with ASD

The exact number of adults with ASD in Singapore is currently not known, but based on an estimated rate of 1 in 167 Singaporeans having ASD, there are likely approximately 18,000 Singaporean adults with ASD (Tan 2011). However, only a small number of these individuals receive specialist support or training in adult centers. Similar to most other developed countries, it is increasingly being recognized that support and services for adults with ASD are very limited in Singapore. There is a current focus on the employment of adults with ASD. The Employability and Employment Centre (E2C) was established in 2012 to equip individuals with mild to moderate ASD with work skills and to support them in their efforts to secure and remain in employment. There is also a small but growing number of day activity centers providing specialized care for adults with ASD and intellectual disabilities, such as the St Andrew's Autism Centre, the Eden Centre for

Adults, and the Thye Hua Kwan Autism Centre. Aside from these ASD-specific initiatives, adults with ASD with comorbid intellectual disability can also access other day activity centers and sheltered workshops for individuals with intellectual disability.

Although adults with disabilities typically live with their families, some require residential support when their parents or family are unable to care for them (Poon 2013). Many autistic adults requiring residential facilities have typically been placed in homes for adults with intellectual disabilities. In 2018, the St Andrew's Adult Home, the first specialized residential facility with a capacity of 200 residents, began operations to meet the needs of adults with autism.

Research and Learning/Professional Development

Research

The first wave of research relating to ASD in Singapore in the 1990s and early 2000s focused on intervention approaches, including computer-aided interventions (Bernard-Opitz et al. 1999, 2001b, 2004; Chen and Bernard-Opitz 1993) and structured playgroups (Bernard-Opitz et al. 2004; Kok et al. 2002). Research in ASD in Singapore has grown considerably with strong collaborations forged between researchers from public organizations, clinicians, and leading academic institutions (i.e., National University of Singapore, NUS; National Institute of Education (NIE) at the Nanyang Technological University; Duke-NUS) to investigate a range of research areas and questions, including diagnosis (i.e., Sung et al. 2018; Wong and Koh 2016), early identification of children with ASD (i.e., Koh et al. 2014); early intervention (i.e., Poon and Bull 2016; Lim et al. 2007); the assessment of learning, functional level, and experiences of children with ASD in schools (Aljunied and Frederickson 2011; Aljunied and Frederickson 2013; Poon 2011; Poon et al. 2014); emotional and mental health comorbidity with a particular

focus on anxiety difficulties and psychopharmacology (i.e., Magiati et al. 2014, 2016; Ooi et al. 2011, 2014; Poon et al. 2014; Sung et al. 2010, 2011; Zainal et al. 2014; Zainal and Magiati 2019); the role and perspectives of families of individuals with ASD (i.e., Lai et al. 2015; Moh and Magiati 2012; Poon 2013; Poon et al. 2013a; Xue et al. 2014); the incorporation of technology in intervention and the role of screen time on behavioral difficulties (Cabibihan et al. 2013); the enlistment of autistic youth in National Service (Chay et al. 2020).

Training and Professional Development

In 2019, Singapore became the first Asian country to host the Asia Pacific Autism Congress (APAC) outside of Australia. This not only encapsulated the strong bilateral relationship between the two countries in terms of collaboration in research but also represented the maturity in research capabilities of the community in Singapore. The congress was organized by the Autism Resource Centre (Singapore) with the strong support from members of the Autism Network Singapore. The organizing and scientific committee members included partners from the local and international autism community including individuals on the autism spectrum. International and Asia-based speakers, autistic people and their families, researchers, educators, clinicians, and researchers shared their work and experiences in the congress. The conference theme “Thriving with Autism” (source: www.apac.autismcongress.org/2019/) aptly reflected the aspiration in research engagement towards maximizing the quality of life of all individuals with autism and their families in Singapore, and beyond.

Social Policy and Advocacy

Services for early intervention and special education are provided collaboratively between the government, VWOs, and the larger community, including corporations (i.e., “many helping hands” approach; Ho 2007). As such, volunteers play a large part in the service provision for

individuals with ASD in roles such as guidance in the VWO governance boards, assistance in organizing fund-raising and other events, and support in the day-to-day operations of various centers. The Autism Association of Singapore was established in 1992 by parents and currently operates two EIPIC centers, one special school and one Day Activity Center (DAC) for adults. Likewise, the Autism Resource Centre, established in 1999, has similar roots and currently operates an EIPIC center, a special school, the Employability and Employment Centre, an autism library, and an autism training and consultancy service that offers more than 2000 training seats per year. As from 2015, a small, but strong and growing, autistic community has emerged with several autistic adults taking leadership and advocacy roles and providing education, leadership, and much needed voices for autistic people in the country. Family members also feature prominently as advocates. For example, a parent who is also a journalist has published two collections of articles, one focusing on Singaporean families' perspectives and another on the independence of autistic adults, both with the aim of raising awareness of autism (Tan 2010, 2018). Parent advocates have also spearheaded various parent support groups in school communities, via online platforms and through social media.

Disability support offices (DSOs) have been established in publicly funded institutes of higher learning (institutes of higher learning in Singapore includes universities, polytechnics, the Institute of Technical Education, private education, and the arts institutions, under the purview of the Division of Higher Education Group of the Ministry of Education) since 2014 to support students with special needs, with services expanded to cater for an increasing number of autistic students entering these higher learning institutions. Institutes of higher learning have also incorporated modules specific to autism, such as for psychology undergraduates at the National University of Singapore and for educators at the National Institute of Education.

Current Issues and Future Directions

Notwithstanding the country's brief history, there has been considerable effort to develop services and supportive infrastructures for individuals with disabilities, including those with ASD, and their families. Although considerable progress has been made, there continues to be a number of areas for further growth. There is a need to further raise the bar for the quality of services for school-aged children, adolescents, and undiagnosed adults with ASD. With increasing numbers of students with ASD accessing the mainstream education system, there is a pressing need to improve on the inclusion and support for these individuals, including looking at ways to diversify the continuum of provisions available to students with ASD (Enabling Masterplan 2007). Caregivers and families continue to report long wait times, particularly for post-school services. More resources need to be injected to develop more and better quality services to support those in higher institutes of learning (such as the polytechnics and universities), those in the workplace, and those who may not be suitable for independent employment. More services need to be developed in the areas of diagnostic services in the adult population and in mental health and emotional support services throughout the life-span for the autistic individuals.

There is also a need to further align and synergize the efforts of schools, professionals, researchers, and other service providers in order to maximize the potential of the individuals with ASD and to enhance their quality of life. While the number of services and centers has increased considerably, the quality and impact of intervention and educational approaches remain under-evaluated. Few programs can clearly document the empirical evidence on which their development and implementation are based, with the exception of the structured teaching approach, which appears to be a component shared across most intervention settings. For this reason, there have been government-initiated and government-funded efforts to improve and standardize the

quality of early intervention services offered. These efforts have taken the form of a study to describe the state of services (i.e., EIPIC baseline study), a consultancy service to improve the quality of services (i.e., EIPIC Consultancy Project), and an ongoing evaluation study to systematically measure the outcomes in addition to the child, family, and intervention factors associated with the progress of the children receiving EIPIC interventions.

Separately, the Autism Resource Centre's Early Intervention Programme has embarked on an international accreditation by the United Kingdom's National Autistic Society, which provides a rigorous quality assurance service to autism-focused programs around the world. Finally, another area for development that has been highlighted includes providing greater support for the autistic individuals during transition phases across the developmental life-span (e.g., Poon and Lim 2012; EM Steering Committee 2012). To address this, a number of programs are currently in progress, such as a project seeking to support the transition of children with ASD from preschool to primary schools, as well as a hospital-based early intervention program for caregivers of recently diagnosed children with ASD (Temasek Cares 2013). One of the restructured hospitals, KKH, has initiated a home-based program for children with ASD and their families (SAFE; Support for Autism through Family Empowerment), which aims to provide families of newly diagnosed children with the knowledge of ASD, information on available services, and parenting/management strategies (Lim, personal communication).

The national approach spearheaded by the Enabling Masterplans has good potential to continue to set directions for the management and coordination of services, and the autism-specific Masterplan currently being developed will likely put autism more firmly and specifically on the national agenda, bringing about pertinent and sustainable improvements in the next 10 years. It is important that the voices and needs of the individuals with ASD and their families take a central

place in the implementation of the agenda set by the Enabling and Autism Masterplans. Finally, to ensure that the understanding of the needs of this population is current and robust, there is an increasing need to further continue to promote collaborations between autistic people, their families, schools, professionals, and researchers.

References and Reading

- Aljunied, M., & Frederickson, N. (2011). Cognitive indicators of different levels of special educational support needs in autism. *Research in Autism Spectrum Disorders*, 5(1), 368–376.
- Aljunied, M., & Frederickson, N. (2013). Does central coherence relate to the cognitive performance of children with autism in dynamic assessments? *Autism*, 17(2), 172–183.
- Bernard-Opitz, V., Sriram, N., & Sapuan, S. (1999). Enhancing vocal imitations in children with autism using the IBM speech viewer. *Autism*, 3(2), 131–147.
- Bernard-Opitz, V., Kwok, K. W., & Sapuan, S. (2001a). Epidemiology of autism in Singapore: Findings of the first autism survey. *International Journal of Rehabilitation Research*, 24(1), 1–6.
- Bernard-Opitz, V., Sriram, N., & Nakhoda-Sapuan, S. (2001b). Enhancing social problem solving in children with autism and normal children through computer-assisted instruction. *Journal of Autism and Developmental Disorders*, 31(4), 377–384.
- Bernard-Opitz, V., Ing, S., & Tan, Y. K. (2004). Comparison of behavioural and natural play interventions for young children with autism. *Autism*, 8(3), 319–333.
- Cabibihan, J. J., HifzaJaved, H., Ang, M., Jr., & Aljunied, M. (2013). Why robots? A survey on the roles and benefits of social robots for the therapy of children with autism. *International Journal of Social Robotics*, 5(4), 593–618. <https://doi.org/10.1007/s12369-013-0202-2>.
- Chay, P. Y., Yam, S. Y., Cheok, C. C. S., & Magiati, I. (2020). Getting ready for national service: A preliminary investigation of the perspectives of young autistic pre-enlistees and their caregivers on national service. *Research in Autism Spectrum Disorders*, 69, 101–451.
- Chen, S. H. A., & Bernard-Opitz, V. (1993). Comparison of personal and computer-assisted instruction for children with autism. *Mental Retardation*, 31(6), 368–376.
- Department of Statistics, Singapore. (2013). <http://www.singstat.gov.sg/statistics>
- Enabling Masterplan Steering Committee. (2007). *Enabling Masterplan, 2007–2011*. Singapore: Ministry of Community Development, Youth and Sports.
- Enabling Masterplan Steering Committee. (2012). *Enabling Masterplan 2012–2016: Maximising*

- potential, embracing differences*. Singapore: Ministry of Community Development, Youth and Sports.
- Ho, L. Y. (2007). Child development programme in Singapore 1988 to 2007. *Annals of the Academy of Medicine, Singapore*, 36, 898–910.
- Ho, L. Y. (2018). Building an inclusive early childhood intervention ecosystem in Singapore, 1988–2017. 16th Haridas Memorial Lecture, Singapore Paediatric Society, 2018. OCN 1044752631 | ISBN 978–981–11–7843–6 (paperback) | 978–981–11–9096–4 (e-book).
- Koh, H. C., Lim, S. H., Chan, G. J., Lin, M. B., Lim, H. H., ... Magiati, I. (2014). The clinical utility of the modified checklist for autism in toddlers with high risk 18–48 month old children in Singapore. *Journal of Autism and Developmental Disorders*, 44(2), 405–416.
- Kok, A. J., Tan, Y. K., & Bernard-Opitz, V. (2002). A comparison of the effects of structured play and facilitated play approaches on preschoolers with autism: A case study. *Autism*, 6(2), 181–196.
- Lai, W. W., Goh, T. J., Oei, T. P. S., & Sung, M. (2015). Coping and well-being in parents of children with autism spectrum disorders (ASD). *Journal of Autism and Developmental Disorders*, 45(8), 2582–2593.
- Lee, H. L. (2004). *Speech presented at the opening of the Spastic Children's Association of Singapore's Cerebral Palsy Centre [Speech]*. Singapore: Prime Minister's Office. <http://stars.nhb.gov.sg/stars/public/>
- Lim, S. B., & Academy of Medicine Singapore-Ministry of Health Clinical Practice Guidelines Workgroup in Autism Spectrum Disorders. (2010). Academy of Medicine Singapore-Ministry of Health clinical practice guidelines: Autism spectrum disorders in pre-school children. *Singapore Medical Journal*, 51(3), 255–263.
- Lim, S. M., Kattapuram, A., & Lian, W. B. (2007). Evaluation of a pilot clinic-based social skills group. *The British Journal of Occupational Therapy*, 70(1), 35–39.
- Magiati, I., Chan, J. Y., Tan, J. W. L., & Poon, K. K. (2014). Do non-referred young people with autism spectrum disorders and their caregivers agree when reporting anxiety symptoms? A preliminary investigation using the Spence Children's Anxiety Scale. *Research in Autism Spectrum Disorders*, 8(5), 546–558.
- Magiati, I., Ong, C., Lim, X. Y., Tan, J. W. L., Ong, A. Y. L., Patricia, F., ... Howlin, P. (2016). Anxiety symptoms in young people with autism spectrum disorder attending special schools: Associations with gender, adaptive functioning & autism symptomatology. *Autism*, 20(3), 306–320.
- Ministry of Health, Singapore. (2010). *Autism spectrum disorders in pre-school children. AMSMOH clinical practice guidelines 1/2010*. Singapore: Ministry of Health. Available at: <http://moh.gov.sg/cpg>
- Moh, T. A., & Magiati, I. (2012). Factors associated with parental stress and satisfaction during the process of diagnosis of children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6(1), 293–303.
- Ooi, Y. P., Tan, Z. J., Lim, C. X., Goh, T. J., & Sung, M. (2011). Prevalence of behavioural and emotional problems in children with high-functioning autism spectrum disorders. *Australian and New Zealand Journal of Psychiatry*, 45(5), 370–375.
- Ooi, Y. P., Rescorla, L., Sung, M., Fung, D. S., Woo, B., & Ang, R. P. (2014). Comparisons between autism spectrum disorders and anxiety disorders: Findings from a clinic sample in Singapore. *Asia-Pacific Psychiatry*, 6(1), 46–53.
- Poon, K. K. (2011). The activities and participation of adolescents with autism spectrum disorders in Singapore: Findings from an ICF-based instrument. *Journal of Intellectual Disability Research*, 55(8), 790–800.
- Poon, K. K. (2012). Challenging behaviors among children with autism spectrum disorders and multiple disabilities attending special schools in Singapore. *Research in Developmental Disabilities*, 33(2), 578–582.
- Poon, K. K. (2013). Parental expectations regarding post-school social attainments of adolescents with autism spectrum disorders in Singapore. *American Journal on Intellectual and Developmental Disabilities*, 118(2), 95–107.
- Poon, K. K., & Bull, B. (2016). The profile of and subgroups within young children with autism spectrum disorder receiving early intervention in Singapore. *Journal of Intellectual Disability Research*, 60(7–8), 720.
- Poon, K. K., & Lim, A. K. (2012). Current provision, recent developments and future directions for early childhood intervention in Singapore. *Infants and Young Children*, 25(4), 323–333.
- Poon, K. K., Koh, L., & Magiati, I. (2013a). Parental perspectives on the importance and likelihood of adult outcomes for children with autism spectrum disorders, intellectual disabilities or multiple disabilities. *Research in Autism Spectrum Disorders*, 7(2), 382–390.
- Poon, K. K., Musti-Rao, S., & Wettasinghe, C. M. (2013b). Special education in Singapore: History, trends, and future directions. *Intervention in School and Clinic*, 49, 59–64.
- Poon, K. K., Soon, S., Wong, M. E., Kaur, S., Khaw, J., Ng, Z., & Tan, C. S. (2014). What is school like? Perspectives of Singaporean youth with high-functioning autism spectrum disorders. *International Journal of Inclusive Education*, 18(10), 1069–1081.
- Poon, K. K., Tham-Toh, S. Y. J., & Lee, E. H. (2016). Educational provision for children with disabilities in Singapore. In J. Tan & K. E. Kuah-Pearce (Eds.), *Educating marginalized communities in East Asia: State, civil society and NGO partnerships* (pp. 120–135). Singapore: Routledge.
- Rajah, I. (2019). *Speech by Ms Indranee Rajah, Second Minister for Education at an extraordinary celebration [Speech]*. MOE Speeches/Interviews; Ministry of Education. <https://www.moe.gov.sg/news/speeches/speech-by-ms-indranee-rajah%2D%2Dsecond-minister-for-education%2D%2Ddat-an-extraordinary-celebration-concert>
- Sung, M., Fung, D. S., Cai, Y., & Ooi, Y. P. (2010). Pharmacological management in children and adolescents with pervasive developmental disorder.

- Australian and New Zealand Journal of Psychiatry*, 44(5), 410–428.
- Sung, M., Ooi, Y. P., Goh, T. J., Pathy, P., Fung, D. S., Ang, R. P., & Lam, C. M. (2011). Effects of cognitive-behavioral therapy on anxiety in children with autism spectrum disorders: A randomized controlled trial. *Child Psychiatry & Human Development*, 42(6), 634–649.
- Sung, M., Goh, T. J., Tan, B. L. J., Chan, J. S., & Liew, H. S. A. (2018). Comparison of DSM-IV-TR and DSM-5 criteria in diagnosing autism spectrum disorders in Singapore. *Journal of Autism and Developmental Disorders*, 48(10), 3273–3281.
- Tan, B. (2010). *Come into my world: 31 stories of autism in Singapore*. Singapore: Author.
- Tan, T. (2011, November 5). Changing face of special needs. *The Straits Times*, D2–D5. http://www.autism.org.sg/press/2011/111105-changing_face_of_special_needs-ST.pdf
- Tan, B. (2018). *My way: 31 stories of independent autism*. Singapore: Author.
- Temasek Cares. (2013). *Reflection 2012–2013: Initiating new programs to care better for Singaporeans*. Singapore: Author. <http://www.temasekcares.org.sg/downloads/FY201213/TC%20AR%20English.pdf>
- Wong, C. M., & Koh, H. C. (2016). Brief report: Investigating the implications of applying the new DSM-5 criteria for diagnosing autism spectrum disorder in a preschool population in Singapore. *Journal of Autism and Developmental Disorders*, 46(9), 3177–3182.
- Xue, J., Ooh, J., & Magiati, I. (2014). Family functioning in Asian families raising children with autism spectrum disorders: The role of capabilities and positive meanings. *Journal of Intellectual Disability Research*, 58(5), 406–420.
- Zainal, H., & Magiati, I. (2019). A comparison between caregiver-reported anxiety and other emotional and behavioral difficulties in children and adolescents with autism spectrum disorders attending specialist or mainstream schools. *Journal of Autism and Developmental Disorders*, 49(7), 2653–2663.
- Zainal, H., Magiati, I., Tan, J. W., Sung, M., Fung, D. S., & Howlin, P. (2014). A preliminary investigation of the Spence Children's Anxiety Parent Scale as a screening tool for anxiety in young people with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(8), 1982–1994.

Single-Nucleotide Polymorphism

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Synonyms

Single-nucleotide variation; SNP

Definition

A single-nucleotide polymorphism (SNP, pronounced “snip”) is the variation in a single base of DNA that is present in at least 1% of the population (i.e., a common variant). SNPs are a subset of single-nucleotide variants (SNVs); SNVs include both common (SNPs) and rare (<1% frequency in the population) variants of a single base pair. SNP usually refers to a substitution (when a different base pair is present, e.g., ATCCA instead of ATGCA) rather than a single base insertion (e.g., ATGACA) or a single base deletion (e.g., ATGCA). Confusingly, however, the abbreviation “SNP” is also sometimes used to describe insertions or deletions of a few bases of DNA or rare single-nucleotide polymorphisms (which are technically SNVs).

Every human has about three million SNPs (and an additional 150,000 rare SNVs). Therefore, every individual has a unique combination of SNPs (except for identical twins). This diversity means that SNPs can be used to identify a person from a forensic sample (DNA fingerprinting), to identify family relationships, and to examine the ancestral origins of an individual (e.g., North African, Central European, East Asian).

Human SNPs are recorded in several databases, including the NCBI “dbSNP” database (<http://www.ncbi.nlm.nih.gov/projects/SNP/>). Each SNP

Single Case

► Qualitative Versus Quantitative Approaches

has a unique identifier beginning with the letters “rs” (e.g., rs3572840). For many SNPs, ancestry information is given and a small number includes clinical information about known disease associations. There are various other resources that leverage SNPs to provide information about an individual’s traits or ancestry, including private companies that provide individualized sequencing services for customers.

SNPs may be found in coding or noncoding regions of the genome. Some SNPs play roles in regulating the expression of genes (expression quantitative trait loci or eQTLs) and the methylation of DNA (methylation quantitative trait loci or meQTLs). Particular SNPs may increase or decrease risk of a particular disease. Alternatively, a SNP may also act as a marker for a nearby variant that contributes risk or resilience. Therefore, SNPs are commonly used in genetic studies that look for common variants associated with disease, such as genome-wide association studies (GWAS).

To date, GWAS studies of ASD have been limited in their ability to definitively identify SNPs significantly associated with ASD risk. Given that 50% of ASD risk is estimated to be due to common inherited mutations, including SNPs, these negative findings are almost certainly due to a lack of statistical power. Therefore, as cohort sizes (and hence power) increase, significantly associated SNPs are expected to be identified.

See Also

- [Common Disease-Common Variant Hypothesis](#)
- [Copy Number Variation](#)
- [DNA](#)
- [Genetics](#)
- [Genome-Wide Association](#)

References and Reading

Andrews, S. V., Ellis, S. E., Bakulski, K. M., Sheppard, B., Croen, L. A., Hertz-Picciotto, I., et al. (2017). Cross-tissue integration of genetic and epigenetic data offers insight into autism spectrum disorder. *Nature*

Communications, 8(1), 1011. <https://doi.org/10.1038/s41467-017-00868-y>.

Autism Spectrum Disorders Working Group of The Psychiatric Genomics Consortium, Anney, R. J., Ripke, S., Anttila, V., Grove, J., Holmans, P., et al. (2017). Meta-analysis of GWAS of over 16,000 individuals with autism spectrum disorder highlights a novel locus at 10q24.32 and a significant overlap with schizophrenia. *Molecular Autism*, 8, 1–17. <https://doi.org/10.1186/s13229-017-0137-9>.

Cheng, Y., Quinn, J. F., & Weiss, L. A. (2013). An eQTL mapping approach reveals that rare variants in the SEMA5A regulatory network impact autism risk. *Human Molecular Genetics*, 22(14), 2960–2972. <https://doi.org/10.1093/hmg/ddt150>.

Single-Nucleotide Variation

- [Single-Nucleotide Polymorphism](#)

Single-Subject Design

- [Qualitative Versus Quantitative Approaches](#)

Single-Subject Study

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Definition

Single-subject research (sometimes referred to as single-case research) refers to a collection of experimental research designs (e.g., multiple

baseline, withdrawal, alternating treatment; see references for list of guides) that can be used to measure the effects of an intervention in individual participants. By manipulating the experimental conditions, each individual serves as their own control, enabling scientists to infer causal relations.

Historical Background

Early examples of single-subject research can be found in research in psychology and physiology conducted in the nineteenth and early twentieth centuries. Scientists such as Fechner, Ebbinghaus, and Pavlov all used individual research subjects and repeated measure over time in their experiments (Barlow et al. 2009). With this focus on the individual and frequent measurement, the researchers could identify variables related to changes in subject performance. These characteristics of frequent measurement, repeated over time at the individual level, are the hallmark of single-subject design because they allow each subject (or *participant* in studies with humans) to serve as their own control. The generality of findings relevant for individuals is then examined with additional subjects to identify fundamental variables responsible for the phenomena under study.

Burrhus Frederick Skinner's research was largely focused on single-subject methodology and resulted in an extensive body of findings related to learning and behavior. Skinner conducted thousands of experiments showing how certain variables resulted in consistent effects on learning and performance (see Ferster and Skinner 1957; Skinner 1938) and, more than any other person, is responsible for the development of the laws of behavior central to fields of the experimental analysis of behavior and applied behavior analysis (ABA; see Baer et al. 1968). Since Skinner and other early scientists pioneered single-subject research, others have continued to use and advocate for the use of single-subject research methodology as the primary way to identify specific variables related to individual's success and lack of progress.

The logic of single-subject design is focused on establishing a predictable trend in behavior, such that if any changes in behavior are noted, they can be attributed to changes made in interventions at the time of the change in behavior. By establishing this predictable trend, or baseline, an individual can serve as his/her own control, eliminating the need for a comparison group or other control procedures. To establish this baseline, single-subject design uses frequent data collection to track performance on target behaviors for a single individual, such as initiating conversation, on-task behavior, or occurrence of inappropriate behaviors. As data are collected, the data can then be summarized in graphical form, which provides a clear illustration of trends in the target behavior over time; trends can be improving, deteriorating, or stable (i.e., no change). Once these trends are established, then changes can be made, if necessary, to improve performance. Following changes, comparisons can be made between pre- and post-change levels in performance.

Current Knowledge

Research studies including individuals with autism spectrum disorders (ASDs and other populations) are often separated into two research paradigms, group-design and single-subject research. Notably, each design is appropriate for some research questions and not others. At times, it has been incorrectly noted that one research methodology is superior to another. This perspective fails to account for the nature of the questions being asked. Broadly speaking, group research is designed to answer what is the average effect of a procedure on a group of individuals. Single-subject research, on the other hand, is designed to ask to what extent will a given procedure result in clear effects observed with each individual. In other words, group-design research asks about the average effect of a treatment, and single-subject research asks about effects at the individual level. In relation to individuals with ASDs, single-subject research has been invaluable because it is sensitive to the wide variety of individual

strengths and challenges experienced by individuals with ASDs and is the only way to identify the key variables and interventions that result in increased success for each individual. In fact, intervention research on children with ASDs and single-subject designs is more frequently used than all other research paradigms (Reichow et al. 2007) and has laid the foundations for the identification of many evidence-based treatments for individuals with ASDs.

Relative to autism research, single-subject design has been invaluable in demonstrating important outcomes for individuals with ASDs and the careful assessment and planning that is necessary to appropriately individualize interventions for each person. In addition to basic and applied research, single-subject design has been central to educational and therapeutic interventions for individuals with ASDs. Single-subject design methodology is also the foundation of effective data-based decision making for individuals in educational and therapeutic settings, where ongoing data collection, progress monitoring, and using those data to identify the need for program revision is essential. The translation of this research paradigm into practice where it can be used to isolate intervention effects at the level of the individual is critical for ensuring that individuals receiving services have effective programs that are helping move them toward optimal outcomes.

Finally, it is important to note that single-subject design is not the same as case study research. Case study research is limited to an account of an individual's performance or experiences over the course of some intervention(s) or a retelling of an account of case(s) from a clinician (e.g., Kanner's original 1943 report of 11 children with early infantile autism could be considered a case study). When used to describe interventions, case study research is not experimental in that it does not allow for clear identification of functional relations between variables (i.e., causal relations), whereas single-subject designs, on the other hand, manipulate experimental conditions such that a functional relation (causal relation), if existing, can be identified.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavior Analysis](#)

References and Reading

- Baer, D. M., Wolf, M. M., & Risley, T. R. (1968). Some current dimensions of applied behavior analysis. *Journal of Applied Behavior Analysis*, 1, 91–97.
- Barlow, D. H., Nock, M. K., & Hersen, M. (2009). *Single case experimental designs: Strategies for studying behavior change* (3rd ed.). Boston: Allyn Bacon.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Prentice Hall.
- Ferster, C. B., & Skinner, B. F. (1957). *Schedules of reinforcement*. Englewood Cliffs: Prentice-Hall.
- Johnston, J. M., & Pennypacker, H. S. (1993). *Strategies and tactics of behavioral research*. Hillsdale: Lawrence Erlbaum.
- Kazdin, A. E. (2010). *Single-case research designs: Methods for clinical and applied settings*. New York: Oxford University Press.
- Reichow, B., Barton, E. E., Volkmar, F. R., & Cicchetti, D. V. (2007, May). *The status of research on interventions for young children with autism spectrum disorders*. Poster presented at the International Meeting for Autism Research, Seattle.
- Sidman, M. (1960). *Tactics of scientific research*. New York: Basic Books.
- Skinner, B. F. (1938). *The behavior of organisms: An experimental analysis*. Cambridge, MA: B.F. Skinner Foundation.

Singsong

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Definition

Odd patterns of vocal intonation can be referred to as “singsong,” a term which generally describes a

speech style with a wide pitch range with large rises and falls. It should be noted that this term is somewhat imprecise as it is also occasionally used to describe a very different style of speech with a particularly narrow pitch range, as in a poetry reading, or speech produced to the tune of a preferred song. In all cases, this term is used to indicate that speech has a quality that is different from the typical melodic patterns of conversation. Some individuals with ASD have been noted to have a “singsong” style of speaking, with exaggerated or theatrical-sounding prosodic patterns.

See Also

- [Intonation](#)
- [Monotone](#)
- [Pedantic Speech](#)
- [Prosody](#)

References and Reading

- Diehl, J. J., & Berkovits, L. (2010). Is prosody a diagnostic and cognitive bellwether of autism spectrum disorders? In A. Harrison (Ed.), *Speech disorders: Causes, treatments, and social effects* (pp. 159–176). New York: Nova Science.
- McCann, J., & Peppé, S. (2003). Prosody in autism spectrum disorders: A critical review. *International Journal of Language & Communication Disorders*, 38(4), 325–350.
- Peppé, S. (2009). Why is prosody in speech-language pathology so difficult? *International Journal of Speech-Language Pathology*, 11(4), 258–271.
- Shriberg, L. D., Paul, R., McSweeney, J. L., Klin, A., Cohen, D. J., & Volkmar, F. R. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning Autism and Asperger syndrome. *Journal of Speech, Language, and Hearing Research*, 44(5), 1097–1115.

SIPT

- [Sensory Integration and Praxis Test](#)

Situation Awareness

- [Driving Hazard Perception in ASD](#)

Situations-Options-Choices-Consequences-Strategies-Simulation (SOCCSS) Program

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Definition

Situations-Options-Choices-Consequences-Strategies-Simulation (SOCCSS) Program is a strategy developed by Jan Roosa according to Myles and Aderson (2001). It is a social decision-making process designed to help students on the autism spectrum and with other disabilities understand social situations. Individuals on the autism spectrum have difficulty in instances where they must understand and act upon the “unwritten rules” of a social situation. It is a step-by-step process by which individuals with ASDs learn to analyze and apply discrete skills in a given situation (Texas Statewide Leadership for Autism 2009). This is a particularly helpful technique because individuals with ASDs have difficulty generalizing skills across environments and situations. SOCCSS can be used analytically as an interpretive tool, or it can be used as an instructional tool to aid in learning how to handle future social situations. Gray’s (1994) *Comic Strip Conversations* has been described as a visual or graphic SOCCSS (Minnesota Para-professional Consortium).

The SOCCSS social skills strategy consists of the six steps that comprise the acronym:

Step 1 Situation: the teacher or therapist helps the individual analyze the problem by answering the five “W’s” (i.e., who, what, when, and why) of the situation.

Step 2 Options: involves brainstorming ways to respond.

Step 3 Consequences: includes a review of the possible consequences of each option generated in the previous step.

Step 4 Choices: consists of reviewing each options-consequences sequence and then prioritizing possible responses. The individual then selects his or her most likely response.

Step 5 Strategy: turns the previous four steps into a plan of action which may include a written script.

Step 6 Simulations: are practice sessions to deal with situation via guided imagery, reading written scripts, role plays, and other forms of behavioral rehearsal (Minnesota Paraprofessional Consortium).

Before the SOCCSS strategy is implemented with an individual with an ASD, he or she should be assessed for his or her abilities across a variety of dimensions. These dimensions include behavioral abilities and characteristics of the person with the ASD. SOCCSS requires some ability to:

- Answer who, what, when, where, and why questions
- Understand a simple cause-and-effect relationship
- Make choices
- Follow multistep directions
- Participate in social interactions
- Problem solve
- Identify or accept a socially appropriate outcome
- Generalize learning (Interactive Collaborative Autism Network)

Characteristics that need to be considered when assessing the appropriateness of SOCCSS or the need to modify the strategy include:

- Literal interpretation of information
- Poor perspective taking
- Inflexibility or a tendency to adhere strictly to a routine
- Difficulty predicting outcomes
- Lack of knowledge about social conventions such as turn-taking
- Lack of understanding of the cause-and-effect relationship of one’s behavior on others
- Difficulty generalizing learning
- Difficulty multichanneling (Lawson 2001), i.e., the ability to respond to more than one stimulus at a time (Interactive Collaborative Network)

See Also

- [Comic Strip Conversations](#)

References and Reading

- Gray, C. (1994). *Comic strip conversations* (pp. 7–12). Arlington: Future Education. <http://www.thegraycenter.org>
- Interactive Collaborative Autism Network. University of New Mexico Autism Center. Autism Courses at CDD. *Why teach social skills*. Retrieved 27 May 2011, from http://cdd.unm.edu/swan/autism_course/module/s/social/soccss/lecture01.html
- Lawson, W. (2001). *Understanding and working with the spectrum of Autism: An insider’s view* (pp. 13–17). London: Jessica Kingsley Publishers.
- Minnesota Para-professional Consortium. *Strategies to improve social understanding*. Retrieved 27 May 2011, from http://paraalink.org/asds3/asds3_4.html
- Myles, B. S., & Aderson, D. A. (2001). *Asperger syndrome and adolescence: Practical solutions for school success*. Shawnee Mission: Autism Asperger.
- Myles, B., Trautman, M. L., & Schelvan, R. L. (2004). *The hidden curriculum: Practical solutions for understanding understated rules in social situations* (pp. 20–23). Shawnee Mission: Autism Asperger. <http://www.asperger.net>
- Texas Statewide Leadership for Autism. (2009). *Target: Texas guide for effective teaching situations-options-choices-consequences-strategies-simulation (SOCCSS) program*. Retrieved 27 May 2011, from <http://www.txautism.net/docs/Guide/Interventions/SOCCSS.pdf>

Skill

► Target Behavior

Skinner's Verbal Behavior

► Skinnerian Categories of Verbal Behavior

Skinnerian Categories of Verbal Behavior

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Synonyms

[Skinner's verbal behavior](#); [Verbal behavior](#)

Definition

Skinner's view of language is that verbal behavior is under the control of consequences mediated by other people. He was less interested in the mental structures like knowledge or language competence but rather the functional relationships of the behavior in the environment in which it occurs. The child's response is conditioned by its consequence in a specified situation or what Skinner called the antecedent-behavior-consequence (ABC) structure. For example, a toddler says "bah" and his mother responds, "Sure! Here's the ball." If the child is rewarded for "emitting" a specific behavior (i.e., making a sound), the child will learn to make the sound again under the same circumstances to gain the same reward.

Skinner's categories of verbal behavior include echoic, mand, tact, and intraverbal. According to Skinner's theory, each has a different function and

will be produced under circumstances that elicit that function. An echoic is the repetition of a heard word or phrase for verbal learning and practice, or an imitation. It is elicited by a request for imitation and a reward of the imitation (e.g., caregiver says "apple," child repeats "apple," child then gets to eat a slice of the apple). A mand behavior functions as a request (e.g., child says "car" because he wants access to his toy car) to immediately benefit the speaker. A tact is a label (e.g., child says "duck" because he sees one in a pond). Intraverbals are responses to others' verbal behavior (e.g., responding to questions, taking conversational turns).

See Also

► [Operant Conditioning](#)

References and Reading

- Skinner, B. F. (1957). *Verbal behavior*. Acton: Copley Publishing Group.
- Sundberg, M., & Michael, J. (2001). The benefits of Skinner's analysis of verbal behavior for children with autism. *Behavior Modification*, 25, 698–724.

SLDT-A

► [Social Language Development Test](#)

SLDT-E

► [Social Language Development Test](#)

Sleep Bruxism

► [Bruxism](#)

Sleep Disorders

► Medical Conditions Associated with Autism

Sleep Disorders and Autism

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Synonyms

Sleep disturbance; Somnolence

Definition

Sleep disorder is a term used to describe problems with the quality, timing, and amount of sleep that can negatively impact the physical, emotional, behavioral, cognitive, and social development and daytime functioning of youth and cause stress for families. Research has suggested that youth with autism are particularly vulnerable to sleep disorders, with prevalence rates reported as high as 80%. There is also mounting evidence that sleep disorders that are associated with too little sleep or poor sleep quality can worsen certain symptoms associated with autism, such as aggression, somatosensory disturbances, and repetitive behaviors, which can, in turn, make sleeping even more difficult. Furthermore, sleep disorders in youth with autism tend to be more severe, persistent, and resistant to treatment with a higher relapse rate compared to youth with other developmental disorder such as attention deficit hyperactivity disorder and in typically developing youth.

The types of subjective sleep problems most commonly encountered in sleep in youth with autism include irregular sleep-wake patterns; short sleep duration; insomnia symptoms (difficulty falling and staying asleep, early morning awakenings); parasomnias such as sleep waking, night terrors, and bruxism (teeth grinding);

circadian rhythm abnormalities; and sleep complaints associated with medical disorders. Moreover, the prevalence of objectively measured sleep disorders such as obstructive sleep apnea and periodic limb movement disorder are significantly increased in youth with autism.

From a public health standpoint, given the high prevalence and persistence of sleep disorders in youth with autism, all children presenting with autism should be systematically screened for the presence of sleep disturbances and disorders and assessed for any potential co-occurring sleep disorder. If the clinical presentation suggests the risk for any sleep disorder, these youth should be evaluated using appropriate assessment techniques including thorough sleep history and sleep diaries and overnight sleep studies when warranted. Clinicians need to be aware of the impact of sleep disorders in youth with autism and provide targeted evidence-based interventions that include, but are not limited to, behavioral interventions and sleep hygiene techniques, cognitive behavioral therapy for insomnia, chronotherapy, and use of melatonin and prescription medication.

The etiologies of sleep disorders in youth with ASD are likely multifactorial with the interaction of biological mechanisms, psychosocial and environmental influences, and family factors including child-rearing practices that are not conducive to good sleep playing a role. Future research needs to be conducted on the causes of sleep disorders and effectiveness of treatment in youth with autism.

See Also

- Medical Conditions Associated with Autism
- Mental Health and ASD

References and Reading

- Cortesi, F., Gianotti, F., Ivanenko, A., & Johnson, K. (2010). Sleep in children with autism spectrum disorder. *Sleep Medicine, 11*, 659–664.
- Elrod, M. G., & Hood, B. S. (2015). Sleep differences among children with autism spectrum disorders and

typically developing peers: A meta-analysis. *Journal of Developmental and Behavioral Pediatrics*, 36(3), 166–177.

Maski, K., & Owens, J. (2018). Pediatric sleep disorders. *Continuum (Minneapolis Minn)*, 24(1 Child Neurology), 210–227.

Mazurek, M. O., Dovgan, K., Neumeyer, A. M., & Malow, B. A. (2019). Course and predictors of sleep and co-occurring problems in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49(5), 2101–2115.

Richdale, A. L., & Schreck, K. A. (2009). Sleep problems in autism spectrum disorders: Prevalence, nature, & possible biopsychological aetiologies. *Sleep Medicine Reviews*, 13, 403–411.

Sleep Disturbance

- ▶ [Sleep Disorders and Autism](#)

Sleep-ettes D [OTC]

- ▶ [Diphenhydramine](#)

Sleepinal® [OTC]

- ▶ [Diphenhydramine](#)

Sleep-tabs [OTC]

- ▶ [Diphenhydramine](#)

SLO Syndrome

- ▶ [Smith-Lemli-Opitz Syndrome](#)

SLOS

- ▶ [Smith-Lemli-Opitz Syndrome](#)

SLP (Acronym for Speech-Language Pathologist)

- ▶ [Speech-Language Pathologist \(SLP\)](#)

Small-Group Technology-Assisted Instruction Using Socially Assistive Robotics and Virtual Reality

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Definition

Small-group technology-assisted instruction (SGTAI) is an instructional paradigm, during which a group of students, typically two to four, are instructed by a teacher in a close spatial and temporal proximity. In the SGTAI paradigm, at least one of the students or the teacher is emulated via the usage of virtual characters or robots.

Historical Background

One-to-One Instruction

One-to-one instruction has been long established for instructing individuals with an autism spectrum disorder (ASD). In this format, a teacher uses systematic prompting procedures followed by corrective feedback or reinforcement to instruct chosen target behaviors to a single student. In this format, the instruction is delivered to the target student within a highly structured and well-planned format

and typically isolated from their peers in a contained setting. The efficacy and benefits of this instruction format in terms of behavior development of students with ASD are well-substantiated in the literature and practice. Within this format, the teacher's attention and instruction resources are dedicated to the target student, which reduces distracting stimuli and facilitates skill acquisition. Furthermore, this instructional format may be successfully implemented with minimal prerequisite skills from the student.

Despite the benefits, one-to-one (1:1) instruction consumes teacher's instructional time and, hence, demands copious resources for recruitment and training of qualified instructors. This imposes substantial strain on the educational system as data indicates there is a constant dearth of competent special education teachers (US Department of Education 2014). Besides, this instruction format disregards natural instruction environment and hinders instructional and social integration of such students as they are often separated from their peers during instruction. Relying merely on 1:1 format restricts peer interaction opportunities and may prevent acquisition of social interaction abilities essential for functioning in classroom settings where multi-student instruction is the standard.

Small-Group Instruction

Small-group instruction (SGI) is another instruction format, during which a group of students, typically two to four, are instructed together. Small-group instruction improves efficiency of instruction in terms of teacher's time since multiple students are instructed concurrently. It also more closely resembles inclusive environments and general classroom settings where students are in a close proximity and, thus, prepares an environment with higher chances of peer interaction. Within this arrangement, students may acquire appropriate student-to-student interaction skills and get prepared for functioning in less restrictive settings. Besides, students receive multiple and varied forms of the target behavior, which, in turn, facilitates skill generalization to other people and non-training settings.

Imitational Learning

As a result of exposure to other students' behaviors in the group, SGI provides a context in

which students can learn new responses and skills through imitation of peers. From a behavioral standpoint, imitation, which is a category of observational learning, is defined as an individual's behavior that is contiguous in time and similar to that of a model. For instance, if one shows a thumbs-up in front of a child for the first time and the child emits a similar gesture following the modeled behavior, it would be considered an imitative behavior. An imitative behavior may be strengthened through direct reinforcement.

Vicarious Learning

Another advantage of SGI format is that it provides a context in which students can learn novel skills or refine behaviors by viewing the "consequences" received by their peers for their correct or incorrect responses and behaviors. This type of learning, which is another category of observational learning, is called vicarious learning. Merriam-Webster dictionary defines vicarious as "experienced or felt by watching, hearing about, or reading about someone else rather than by doing something yourself." Vicarious learning involves an individual observing others come in contact with stimulus-response contingencies and, thus, learn without directly receiving consequences. A significant difference between imitational learning and vicarious learning is that the latter may cause behavior suppression as a result of observing non-preferred consequences received by a model due to a behavior. For instance, a child may vicariously learn not to throw a basketball at a peer after he sees negative reactions of other students to another peer doing so. Early studies done by Bandura (1965) demonstrated that children who observed a model received punishment following his aggressive behavior were less likely to match the response of the model and to engage in that kind of behavior. Imitation, on the other hand, can only include an observer's behavior matching that of a model.

SGI provides a context in which a student can vicariously learn new responses by watching teacher's instruction delivered to other students, their responses, and the subsequent teacher's reinforcement or feedback according to their accuracy of response. Therefore, simply by group participation, each student in the group has the opportunity to

acquire skills targeted to other students even when he/she is not directly reinforced for his/her learning.

Despite the aforementioned benefits, for a successful implementation, SGI requires a few prerequisite skills from students. For a successful inclusion and proper progress within an SGI format, students are required to possess certain prerequisite skills, such as contingent turn-taking (without disruptive and aberrant behavior, out of seat behavior, or verbalization) and compliance. Lacking such group-specific skills may adversely affect satisfactory progress of the student and that of his/her peers alike.

Current Knowledge

Computer-Assisted Instruction

Many individuals with ASD find interacting with computers more joyful and comfortable than with humans and feel an affinity with them, suggesting that computer-assisted instruction (CAI) can be an effective intervention method for them (see chapters ► [“Computer-Based Intervention Assistive Technology”](#) and ► [“Computer-Assisted Instruction to Teach Academic Skills”](#)). There have been several studies investigating the use and efficacy and efficiency of CAI for behavioral, academic, and social development of individuals with ASD (Pennington 2010). Although the reported positive gains vary, the overall results are quite favorable. CAI can provide learning environments that are individualized, highly controlled, and structured and can include a variety of modalities, such as text, sound, and images. CAI enhances motivational and attentional factors and increases engaged time and enjoyment in individuals with ASD. CAI also has the potential to reach people on the autism spectrum at a broader scale because of the savings due to automation.

Small-Group Technology-Assisted Instruction

Computer-assisted instruction has been also used in the context of SGI. For instance, Mechling et al. (2007) used PowerPoint slides presented on an interactive electronic whiteboard to instruct recognition of grocery words to students with intellectual disabilities within a small group arrangement. Their instructional procedure

involved a teacher advancing the PowerPoint slides, delivering prompts and directions, reading the target words, and providing reinforcement and feedback to students. This study is perhaps the first to demonstrate the efficacy of CAI in the context of an SGI arrangement.

Saadatzai et al. (2018a) developed an intelligent tutoring system which resembled an SGI arrangement, which featured a virtual character and a humanoid robot. The virtual character, displayed on a desktop screen, simulated a human teacher delivering sight word instruction, with meaningful and contingent gaze behavior, body gestures, and facial expressions. On the other hand, the humanoid robot adopted a peer metaphor and served as an emulated peer for students with ASD. The communication of the virtual teacher (VT) and the robot peer (RP) with students and each other was through synthesized speech and automatic speech recognition, in order to facilitate learning through natural language. In this study, the RP's behavior repertoire (e.g., meaningful body gesture as well as contingent social behavior) was designed to increase students' engagement. The RP demonstrated proper group-specific social behaviors, interacted with the students, and provided a context for the participants to rehearse social skills. The authors demonstrated the efficacy of this tutoring system with three children with ASD. In this study, the VT instructed three types of sight words to student-RP dyads including (i) target words unique to student, (ii) nontarget words unique to the RP, and (iii) target words shared between student and the RP. The results indicated that (i) the students acquired the target words directly instructed to them with 100% accuracy; (ii) they vicariously learned the majority of the nontarget words solely taught to the RP, merely by watching the instruction to the RP; and (iii) they learned the shared target words faster and with fewer errors. The participants demonstrated maintenance of their target words for 8 weeks after the instruction and generalized performance to words written on paper at their homes. They, furthermore, maintained and generalized the nontarget words, which were previously instructed to the RP, with a mean accuracy of 88.89%. In a follow-up study, Saadatzai et al. (2018b) replicated this study with two adults with ASD and reported similar outcomes. These two studies were the first to prepare a small-group

technology-assisted instruction (SGTAI) in an agent-robot-student format and to study the effects of such a learning environment on vicarious learning of nontarget stimuli.

Why SGTAI

National Research Council's Committee on Educational Interventions for Children with ASD recommended that developmentally appropriate SGI instruction should be included in the education of individuals with ASD as group skills are crucial for a student to thrive in inclusive and general classroom settings (National Research Council 2001). Individuals with ASD, however, are often unable to benefit from such instruction arrangements due to their impaired social interaction and communication deficiencies. Furthermore, they often struggle to generalize skills acquired during 1:1 intervention to novel settings and peers. They may become adept at a trained skill but fail to transfer that knowledge to everyday life. This makes development of interventions for these individuals highly challenging. Koegel and Rincover (1974) evaluated the consequences of transferring individuals with ASD from a 1:1 format to an SGI format. In this study, in a 1:1 arrangement, eight students with ASD were first instructed a number of behaviors required for group participation and were then transferred to a group arrangement. The results demonstrated that the students minimally and inconsistently generalized the skills they had acquired in the 1:1 arrangement to the group arrangement, even in a small group comprised of one teacher and two students. Additionally, even prolonged group participation did not seem to produce much change as the students failed to acquire new behaviors during a month-long participation in the group setting. Similarly, Graff, Green, and Libby (1998) conducted a study on the effects of two levels of behavioral intervention. The participant, a 5-year-old child with ASD, received 1:1 intervention for a year, which was changed due to resource limitations. When the student-to-teacher ratio increased, the participant's challenging behavior escalated, while his progress in skill acquisition either decreased or remained stable. In this case, the abrupt change in teacher-to-student ratio seemed to adversely affect his progress in the group arrangement.

Although group instruction can be an effective and efficient instructional arrangement for individuals with ASD, it seems necessary to pay careful attention to how to transit from 1:1 to group settings. Special procedures should be identified and be employed to ensure a smooth shift and avoid abrupt transition for many of these individuals. Furthermore, extra attention must be paid on developing instructional techniques that equip these students with behaviors necessary for multi-student contexts and that prepare them for group participation. Following this rationale, the Division of Early Childhood of the Council for Exceptional Children emphasized on the role and potential of technology to improve access to and participation in general education curriculum and inclusive environments (Stremmel 2005). In this context, SGTAI reflects a novel direction in tutoring systems in which students with ASD can learn and rehearse group-specific social and coping behaviors (such as perspective-taking, reciprocity, and imitational learning) in a controlled environment without negative feedback from and teasing of peers, unnecessary embarrassment, and fear of failure.

It is well-established that resemblance of a training setting to the natural environment (i.e., where behaviors are expected to occur) enhances generalization of skills (Koegel and Rincover 1977; Stokes and Baer 1977). Hence, SGTAI may properly prepare individuals with ASD for group participation by providing a naturalized learning environment that facilitates transition to natural situations. SGTAI provides an educational context where students with ASD learn some target skills (typically academic such as reading), but also certain desirable behaviors are elicited and practiced (e.g., eye contact, gaze following, joint attention, turn-taking, imitation, and attention to other students' responses). SGTAI provides a tightly controlled and reinforcing environment in which students are required to perform group-specific behaviors, facilitating generalization of those behaviors to classroom settings. Although SGTAI should not substitute learning scenarios that more closely resemble natural contexts, it may promote participation in them.

Future Directions

With all the present questions regarding how to include individuals with ASD in regular instructional settings, an enhanced understanding of how these individuals acquire skills in such arrangements is of paramount importance. For future research, the space of possibilities is quite complex. However, the following technologies have immense potential for immediate impact and are significant to explore.

Virtual Reality

Virtual reality learning environments are simulated educational platforms employing computer-generated 3D graphics, which typically incorporate animated characters with instructional roles, referred to as pedagogical agents. Pedagogical agents (PAs) are lifelike virtual characters that interface between the content of an educational environment and students. PAs engage with students through natural language, meaningful body gesture, and contingent facial expressions. In such learning environments, students learn the intended behaviors while interacting with a virtual character exhibiting social and emotional behaviors (such as eye gaze behavior, body gesture, and facial expressions). PAs can be programmed to demonstrate emotional states pertinent to the pedagogical circumstance, such as engagement, confusion, frustration, and curiosity. PAs can utilize eye gaze behavior to simulate joint attentional bids or regulate turn-taking. They can demonstrate head nods/shakes or emotive faces to communicate feedback to the student's responses and actions (Kandalafi et al. 2013; Saadatzi et al. 2017).

In VR learning environments, students can rehearse real-life scenarios through the comfort and safety of a simulated environment, in which physical proximity, sound level, and social interactions are tightly controlled. VR provides a predictable and customizable environment where students can learn and repetitively practice social behaviors through interaction with PAs that require fewer social skills and hence produce less anxiety. A favorable property of PAs is that, depending on the desired purpose, they can serve different educational roles in an SGTAI

environment, such as a simulated teacher or a simulated peer/classmate.

Application of VR for instruction of social and academic skills to students with ASD, particularly in the context of SGTAI, is currently underrepresented, calling for further research to reveal and realize its full potential. To date, most VR tutoring systems for individuals with ASD have been developed as desktop virtual environments (DVEs). DVEs visualize the learning environment on a computer display and utilize conventional devices to receive users' inputs (e.g., keyboard and mouse). As such, DVEs cannot completely surround their users into the synthetic learning environment. On the contrary, immersive virtual environments (IVEs) employ head-mounted displays or VR domes to surround their users in the synthetic environment. It has been suggested that IVEs have the potential to induce higher engagement and can generate an environment with high ecological validity, which are conducive to acquisition of skills and generalization thereof to real world. However, there are a number of concerns associated with the usage of IVEs for individuals with ASD, as they may cause sensory overstimulation, VR-induced symptoms (i.e., cyber-sickness), and irritation due to the head-mounted displays (Wallace et al. 2010). Although previous literature suggests that both desktop and immersive virtual environments are effective tools for instruction of individuals with ASD, their effects on user experience as well as their relative effectiveness are less known and warrant further investigation.

Socially Assistive Robotics

The origination of robotic therapy of ASD goes back to the 1976 through a seminal study conducted by Weir and Emanuel (Weir and Emanuel 1976), where an autistic boy was introduced to a remote-controlled turtle. The researchers investigating robotic intervention for ASD envisage design and construction of robotic systems that can guide these individuals from very simple forms of interaction towards more complicated ones, as those in social human-human interactions, by slowly increasing the system's behavior repertoire as well as its complexity,

duration, frequency, and unpredictability. At first, in a very simple context, the system demonstrates a small set of behavior plans, specifically for those individuals with intense deficiencies. After a while, in a gradual basis, the system features a larger behavior repertoire and plans its behavior, based on an agenda that is therapeutically relevant. The previous literature indicates that robots have the potential to serve as intervention means for individuals with ASD. They can implement multi-modal and embodied social interaction and can closely simulate dynamics of human-human interaction (see chapters ► [Robot-Assisted Cognitive Behavioral Therapy for Young Children with Autism Spectrum Disorders](#)).

Both virtual reality and social robots can create individualized and highly controlled learning experiences that are well reproducible and engaging. However, robots' physical presence and embodiment allow for touch and physical interactions, thereby amplifying their capacity to be employed in certain intervention scenarios. Furthermore, their physicality can aid students with ASD build a rapport and facilitate their joint attention with other entities in an SGTAI environment (i.e., virtual/robot/human teacher and peers). Previous literature suggests that, due to their appealing and salient nature, robots tend to evoke episodes of joint attention in students with ASD, draw their attention, and furnish a playful experience. These potentials may be utilized to steer these individuals' attention to instructional material and enhance their engagement with the SGTAI environment. The salience and novelty of robots may be harnessed as a reinforcer and, thereby, amplify these individuals' investment in the instruction. As individuals with ASD are known to exhibit short attention spans, robots can be employed to enhance students' engagement through their visual appeal and body motion, as well as their interactive behavior. In general, robots are engaging for children with ASD and can offer reduced complexities as compared to human interactions.

Adaptive SGTAI

Since ASD does not occur to the same degree and in the same form in all cases, individuals with ASD are a user group with substantial

heterogeneity. In efforts to justify why human teaching is so effective, the benefits have been attributed to their proficiency in customizing pedagogical strategies and in providing in-depth individualization of content. Against their long-standing promise, however, computerized tutoring systems for individuals with ASD rarely perform such individualization. Future SGTAI systems should adopt an adaptive instruction paradigm by creating and inferring from a dynamic student model, rather than simply using static rules and heuristics. Adaptation to personal learning styles will optimize the learning experience and render the instruction potentially more targeted when an individual is struggling. Future research should deploy and evaluate individualized instruction algorithms in SGTAI, enabling adaptation to individual students' (dis)abilities based on longitudinal observations of interactions with students.

Affect-Sensitive SGTAI

Learning is always accompanied and influenced by a set of affective states. For example, boredom and frustration are negatively correlated with learning gains, whereas confusion and engagement positively influence learning. In a classroom setting, expert human tutors are proficient at detecting the emotional state of students and taking actions accordingly in order to optimize their learning. A few recent tutoring systems, designed for typical students, have incorporated affect responsiveness into their pedagogical strategies. To date, however, little effort has been made to investigate this approach in CAIs developed for individuals with ASD. Given that ASD is associated with differences in emotional experiences and autonomic responses, there is no guarantee that individuals with ASD experience the stimuli in the same way as typical students and that the learning-affect link is the same. It has been suggested that affective and cognitive components are equally influential in educational procedures and that CAI systems with mechanisms for detecting and responding to students' affective states are highly beneficial.

In order for a tutoring system to respond to different affective states of students, it must first be endowed with a means to recognize those

states. Individuals with ASD often have communicative difficulties regarding the expression of affective states which limit traditional auditory and visual approaches in automatic detection of emotions. Future research should investigate what expert therapists observe in individuals with ASD to rate their affective states as well as how they decide upon a course of action in response to that observation.

SGTAI systems should be able to detect students' affective cues but also should intelligently customize their pedagogy accordingly. By considering affective information, SGTAI systems can begin to dynamically tailor their pedagogical strategies not just to the cognitive cues such as number or type of errors made by the student and time taken to fulfill criterion but also to their affective states (e.g., frustration, confusion, interest). As such, an affect-sensitive SGTAI system would not only detect whether students are making mistakes but also whether that is accompanied by curiosity and engagement or by frustration. Therefore, various reactions of the SGTAI system (from the virtual/robot/human teacher and peers) may be created and examined for these different circumstances (e.g., adjusting difficulty level, changing the pedagogical strategy).

See Also

- [Computer-Assisted Instruction to Teach Academic Skills](#)
- [Computer-Based Intervention Assistive Technology](#)
- [Robot-Assisted Cognitive Behavioral Therapy for Young Children with Autism Spectrum Disorders](#)

References and Reading

- Bandura, A. (1965). Vicarious processes: A case of no-trial learning. In *Advances in experimental social psychology* (pp. 1–55). New York: Academic.
- Graff, R. B., Green, G., & Libby, M. E. (1998). Effects of two levels of treatment intensity on a young child with severe disabilities. *Behavioral Interventions*, 13(1), 21–41.
- Kandalaf, M., Didehban, N., Krawczyk, D., Allen, T., & Chapman, S. (2013). Virtual reality social cognition training for young adults with high-functioning autism. *Journal of Autism and Developmental Disorders*, 43(1), 34–44.
- Koegel, R. L., & Rincover, A. (1974). Treatment of psychotic children in a classroom environment: Learning in a large group. *Journal of Applied Behavior Analysis*, 7(1), 45–59.
- Koegel, R. L., & Rincover, A. (1977). Research on the difference between generalization and maintenance in extra-therapy responding. *Journal of Applied Behavior Analysis*, 10, 1–12.
- Mechling, L. C., Gast, D. L., & Krupa, K. (2007). Impact of SMART Board technology: An investigation of sight word reading and observational learning. *Journal of Autism and Developmental Disorders*, 37(10), 1869.
- National Research Council. (2001). In C. Lord & J. P. McGee (Eds.), *Educating children with autism*. Washington, DC: National Academy Press.
- Pennington, R. C. (2010). Computer-assisted instruction for teaching academic skills to students with autism spectrum disorders: A review of literature. *Focus on Autism and Other Developmental Disabilities*, 25(4), 239–248.
- Saadatz, M. N., Pennington, R. C., Welch, K. C., Graham, J. H., & Scott, R. E. (2017). The use of an autonomous pedagogical agent and automatic speech recognition for teaching sight words to students with autism spectrum disorder. *Journal of Special Education Technology*, 32(3), 173–183.
- Saadatz, M. N., Pennington, R. C., Welch, K. C., & Graham, J. H. (2018a). Small-group technology-assisted instruction: Virtual teacher and robot peer for individuals with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(11), 3816–3830.
- Saadatz, M. N., Pennington, R. C., Welch, K. C., & Graham, J. H. (2018b). Effects of a robot peer on the acquisition and observational learning of sight words in young adults with autism spectrum disorder. *Journal of Special Education Technology*, 33(4), 284–296.
- Stokes, T. F., & Baer, D. M. (1977). An implicit technology of generalization. *Journal of Applied Behavior Analysis*, 10, 349–367.
- Stremmel, K. (2005). DEC recommended practices: Technology applications. In S. Sandall, M. L. Hemmeter, B. J. Smith, & M. E. McLean (Eds.), *DEC recommended practices: A comprehensive guide for practical application in early intervention/early childhood special education* (pp. 147–162). Missoula: DEC.
- U.S. Department of Education, Office of Postsecondary Education. (2014). Teacher shortage areas nationwide listing 1990–1991 thru 2013–2014. Retrieved 15 June 2017, from <https://www2.ed.gov/about/offices/list/oep/pol/tsa.html#list>
- Wallace, S., Parsons, S., Westbury, A., White, K., & Bailey, A. (2010). Sense of presence and atypical social judgments in immersive virtual environments:

Responses of adolescents with autism spectrum disorders. *Autism*, 14(3), 199–213.

Weir, S., & Emanuel, R. (1976). *Using LOGO to catalyze communication in an autistic child*. D. A. I. Research Report No. 15. University of Edinburgh. Department of Artificial Intelligence.

Smith v. Robinson (1984)

► [Smith v. Robinson \(1984\) Rights to Attorney Fees for Parents](#)

Smith v. Robinson (1984) Rights to Attorney Fees for Parents

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Synonyms

[Smith v. Robinson \(1984\)](#)

Definition

This case addressed a parent's entitlement to reimbursement of attorney's fees incurred as a result of legal action against a public school board for discrimination against the special needs of a mentally impaired student. In *Smith v. Robinson*, the Cumberland, Rhode Island, superintendent of schools denied continued funding, by the school committee, of the necessary special programs required by the petitioner, a child who suffers from cerebral palsy and other mental handicaps. The petitioner's parents filed an action in federal district court against the school committee and against various state school officials. The parents brought forth claims for declaratory and

injunctive relief based on state law, specifically the Education of the Handicapped Act (hereinafter "EHA" or the "Act"), on §504 of the Rehabilitation Act of 1973 and, with respect to certain federal constitutional claims, on 42 U.S.C. §1983. Although the district court originally found for the petitioner and awarded attorney's fees, on appeal, the Court of Appeals reversed the lower court's decision on the grounds that the petitioner's action, and subsequent relief granted, fell within the scope of the Act which does not provide for attorney's fees. The Court of Appeals went on to state that notwithstanding the petitioner's federal constitutional claims – which generally allow for the reimbursement of attorney's fees – the relevant decision was within the scope of the EHA and that the Act's omission of attorney's fees does not imply the court's use of federal law in awarding fees. The US Supreme Court thereafter upheld the Court of Appeals' decision.

Conclusion

In *Smith v. Robinson*, although the child's parents prevailed in their claims of discrimination and violation of due process against the school committee in federal court, the US Supreme Court held against reimbursement of attorney's fees for the entire proceedings.

The Supreme Court held that a petitioner availing himself of the EHA and being successful thereunder cannot collect attorney's fees for unsuccessful federal claims simultaneously filed. In addition, the court found that the specific simultaneous claims – §504 of the Rehabilitation Act of 1973 and on 42 U.S.C. §1983 – raised by the petitioners did not, in and of themselves, allow for attorney's fees.

See Also

► [Liability](#)

Smith, Tristram

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Name and Degrees

Tristram Smith, Ph.D.

Major Appointments (Institution, Location, Dates)

Haggerty-Friedman Professor of Developmental/
Behavioral Pediatric Research, University of
Rochester Medical Center (2016–2018)
Professor of Pediatrics, University of Rochester
Medical Center (2012–2016)
Associate Professor of Pediatrics, University of
Rochester Medical Center (2007–2012)
Assistant Professor of Pediatrics, University of
Rochester Medical Center (2000–2006)
Assistant Professor, Department of Psychology,
Washington State University (1995–2000)
Visiting Assistant Professor, Department of Psy-
chology, Drake University (1993–1995)
Lecturer, Department of Psychology, University
of California Los Angeles (1990–1993)

Major Honors and Awards

New York State Association for Behavior Analy-
sis Award for Significant Contributions to
Applied Behavior Analysis (2003)
Ruth A. Lawrence Academic Faculty Research
Service Award, University of Rochester
Medical Center (2009)
Endowed Chair: Haggerty-Friedman Professor-
ship of Developmental/Behavioral Pediatric
Research (2016)

Landmark Clinical, Scientific, and Professional Contributions

Dr. Smith's early research contributed significantly to our understanding that we can improve outcomes for individuals with ASD. His seminal research with Dr. O. Ivar Lovaas provided the first demonstration that children with autism spectrum disorder could be successfully engaged in behavioral interventions and that for some children, intervention resulted in significant gains on standardized assessment outcomes and clinical outcomes. This seminal work not only altered conventional wisdom regarding the possibility of interventions in autism, but it ignited advocacy efforts and provided both hope and a plan for many families. Subsequent to his work on early intensive behavioral interventions (EIBI), Dr. Smith conducted replications studies, assessed long-term outcomes, and specifically evaluated predictors of success in EIBI.

To extend his evaluation of existing interventions, Dr. Smith led and participated in numerous efforts to develop and evaluate novel interventions. Many of these focused on parent-mediated treatments to address concerns such as disruptive behavior and feeding difficulties. This research contributed support to parents' ability to deliver evidence-based interventions to address a variety of challenges and also established that parent-mediated treatment can result in similar or enhanced gains compared to other options, such as medication to address hyperactivity.

More recently, Dr. Smith's work has supported the successful dissemination and implementation of community-based interventions to enhance outcomes for individuals with ASD. His school-based interventions showed that academic and behavioral improvement was possible via interventions delivered in real-world classroom settings and that educators could reliably implement these strategies.

Dr. Smith was actively involved in the development and evaluation of interventions that targeted core difficulties in ASD, in addition to academic and behavioral outcomes. He

participated in multi-site network comparative effectiveness studies evaluating the relative benefits of applied behavior analytical strategies versus social-communication interventions for children with ASD and minimal verbal skills. Dr. Smith also led the development and implementation of a modular intervention to support student with ASD in classrooms that targeted both core and associated features of ASD.

Short Biography

An early friendship with a young man with autism inspired Dr. Smith's application into the doctoral program at the University of California Los Angeles, where in 1983 he was accepted to work with Dr. O. Ivar Lovaas on the UCLA Young Autism Project. With Dr. Lovaas, Dr. Smith engaged in seminal research on behavioral interventions to improve cognitive outcomes for young children with ASD. In 2000, Dr. Smith joined the faculty at the University of Rochester Medical Center, where an interdisciplinary, federally funded autism research center had been recently established under the leadership of Patricia Rodier, PhD. With his collaborators, Dr. Smith was awarded Studies to Advance Autism Research in Treatment (STAART) Center grant, thus beginning a very successful series of funded investigations based in Rochester. Dr. Smith demonstrated research expertise across myriad topics, including longitudinal evaluations of EIBI and assessing predictors of outcomes in EIBI. Acknowledging the significant role that families play in their children's treatment, Dr. Smith collaborated on several, multi-site projects on parent-mediated intervention related to disruptive behavior and feeding challenges. Always mindful of how his research translated into meaningful outcomes for families, Dr. Smith recently focused much of his work on the application of evidence-based interventions in real world settings. Through extensive collaborations with the community and researchers around the country, Dr. Smith led studies on implementation of interventions to support parents, educators, and other school personnel in helping children with autism access appropriate and high-quality

services in home and public school settings – often with an emphasis on families who are traditionally under-represented in research.

See Also

- [Early Intensive Behavioral Intervention \(EIBI\)](#)
- [Lovaas, O. Ivar](#)
- [Parent Training](#)

References and Reading

- Arnold, L. E., Ober, N., Aman, M. G., Handen, B., Smith, T., Pan, X., . . . & Rice Jr, R. (2018). A 1.5-year follow-up of parent training and atomoxetine for attention-deficit/hyperactivity disorder symptoms and noncompliant/disruptive behavior in autism. *Journal of Child and Adolescent Psychopharmacology*, 28, 322.
- Bearss, K., Johnson, C., Smith, T., Lecavalier, L., Swiezy, N., Aman, M., . . . & Scahill, L. (2015). Parent training for young children with autism spectrum disorder and disruptive behavior: A randomized clinical trial. *JAMA*, 313(15), 1524–1533. <https://doi.org/10.1001/jama.2015.3150>.
- Cohen, H., Amerine-Dickens, M., & Smith, T. (2006). Early intensive behavioral treatment: Replication of the UCLA model in a community setting. *Journal of Developmental and Behavioral Pediatrics*, 27, S145–S155.
- Handen, B. L., Aman, M. G., Arnold, L. E., Hyman, S. L., Tumuluru, R., Lecavalier, L., . . . & Smith, T. (2015). Atomoxetine, parent training and their combination in children with autism spectrum and ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, 54(11), 905–915. <https://doi.org/10.1016/j.jaac.2015.08.013>.
- Hyman, S. L., Stewart, P. A., Foley, J., Cain, U., Peck, R., Morris, D. D., & Smith, T. (2016). The gluten-free/casein-free diet: A double-blind challenge trial in children with autism. *Journal of Autism and Developmental Disorders*, 46(1), 205–220. <https://doi.org/10.1007/s10803-015-2564-9>.
- Iadarola, S., Shih, W., Dean, M., Reisinger, E., Harwood, R., Hetherington, S., Mandell, D., Kasari, C., & Smith, T. (2018). Implementing a manualized, classroom transition intervention for students with ASD in under-resourced schools. *Behavior Modification*, 42(1), 126–147. <https://doi.org/10.1177/0145445517711437>.
- McEachin, J. J., Smith, T., & Lovaas, O. I. (1993). Long-term outcome of children with autism who received early intensive behavioral treatment. *American Journal on Mental Retardation*, 97, 359–372.
- Mruzek, D. W., McAleavey, S., Engel, S., & Smith, T. (2016). Toilet training students with intellectual disability in a school setting with a novel enuresis alarm:

An initial evaluation. *Journal of Special Education Technology*, 31(4), 217–227. <https://doi.org/10.1177/0162643416673915>.

- Smith, T., & Antolovich, M. (2000). Parental perceptions of supplemental interventions received by young children with autism in intensive behavior analytic treatment. *Behavioral Interventions*, 15, 83–97.
- Smith, T., & Iadarola, S. (2015). Evidence update: Autism spectrum disorder. *Journal of Clinical Child and Adolescent Psychology*, 44(6), 897–922. <https://doi.org/10.1080/15374416.2015.1077448>.
- Smith, T., Buch, G. A., & Gamby, T. (2000a). Parent-directed, intensive early intervention for children with pervasive developmental disorder. *Research in Developmental Disabilities*, 21, 297–309.
- Smith, T., Groen, A., & Wynn, J. W. (2000b). Randomized trial of intensive early intervention for children with pervasive developmental disorder. *American Journal on Mental Retardation*, 104, 269–285.
- Smith, T., Klorman, R., & Mruzek, D. W. (2015). Predicting outcome of community-based early intensive behavioral intervention for children with autism. *Journal of Abnormal Child Psychology*, 43(7), 1271–1282. PMID: 25778537.
- Smith, T., Aman, M. G., Arnold, L. E., Silverman, L. B., Lecavalier, L., Hollway, J., ... & Handen, B. L. (2016a). Atomoxetine and parent training for children with autism and ADHD: 24-week extension study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 55(10), 868–876. <https://doi.org/10.1016/j.jaac.2016.06.015>.
- Smith, T., Jordan, A., & Tiede, G. (2016b). Early intensive behavioral intervention: Current status of the UCLA model and future directions. *Perspectives on Early Childhood Psychology and Education*, 1(2), 7–28.

Smith-Lemli-Opitz Syndrome

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Synonyms

7-Dehydrocholesterol reductase deficiency; RSH syndrome; SLO syndrome; SLOS

Short Description or Definition

Smith-Lemli-Opitz syndrome (SLOS) was described by the original authors as “RSH syndrome” based on the first initials of three individuals with SLOS (Smith et al. 1964). New cases were later described and additional characteristics were delineated. SLOS is an autosomal recessive disorder due to inborn error of cholesterol metabolism caused by mutations of the 7-dehydrocholesterol reductase gene (*DHCR7*) (Tint et al. 1994), and the gene was later identified at 11q12-q13 (Fitzky et al. 1998; Wassif et al. 1998; Waterham et al. 1998). In individuals with SLOS enzyme, activity of the protein coded by *DHCR7* is reduced or absent, resulting in insufficient cholesterol synthesis and the accumulation of precursor sterols, 7-dehydrocholesterol (7DHC) and its isomer 8-dehydrocholesterol (8DHC). This deficiency causes extremely variable clinical, physical, and behavioral manifestations: from presenting as a lethal disorder with multiple major congenital anomalies to presenting with minor physical stigmata, as well as behavioral and learning disabilities, autism spectrum disorders, aggression, self-injury, sleep problems, irritability, and attention deficit hyperactivity disorder.

Categorization

Smith-Lemli-Opitz-Syndrome (SLOS) is categorized as an autosomal recessive multiple malformation Mendelian disorder (Kelley 2000; Opitz 1969).

Epidemiology

SLOS has an estimated incidence among individuals of European ancestry in Canada and the USA of 1 in 15,000 to 1 in 60,000 births (Bzdúch et al. 2000; Lowry and Yong 1980; Opitz 1999a; Porter 2000; Ryan et al. 1998; Tint et al. 1994) and a carrier frequency of 1 in 30 to 1 in 50 for the most common SLOS mutant allele in North American populations (Battaile et al. 2001; Nowaczyk and

Waye 2001). In the Asian and African races, SLOS is less common and historically females were diagnosed less frequently due to a lower degree of visible genital anomalies (Porter 2000). In those affected with more severe mutations, there is increased incidence of intrauterine fetal deaths and neonatal deaths. Also, individuals with SLOS who have mild features are often not diagnosed and may come to the attention of clinicians due to the diagnosis of SLOS in a family member.

Individuals with SLOS have a deficiency of 3-beta-hydroxysterol delta-7-reductase activity due to mutation of the 3-beta-hydroxysterol delta-7-reductase gene (*DHCR7*). This enzymatic deficiency impairs the conversion of 7-dehydrocholesterol (7DHC) to cholesterol in the last step of cholesterol biosynthesis via the Kandutsch-Russel biosynthetic pathway. Therefore, total sterol and cholesterol levels are decreased and 7DHC and 8DHC levels are elevated (Porter 2000). In SLOS, the clinical (nonbehavioral) severity of abnormalities correlates with the ratio of the blood level of 7DHC (or the sum 7DHC plus 8DHC) expressed as a fraction of total sterols (total sterols = cholesterol plus 7DHC plus 8DHC) (Kelley and Hennekam 2000). The best biochemical predictor of clinical severity is the plasma cholesterol level, which decreased with increasing clinical severity (Cunniff et al. 1997). Independent of sterol synthesis, absolute blood sterols levels are subject to wide physiological variables, giving a spectrum of variability (Kelley and Hennekam 2000). In one study, approximately 10% of subjects with SLOS had normal cholesterol levels at the time of diagnosis, and the SLOS diagnosis would have been missed without specific quantification of 7DHC (Cunniff et al. 1997).

Natural History, Prognostic Factors, Outcomes

Smith-Lemli-Opitz Syndrome is characterized by major abnormalities of development at birth (Kelley 2000). At 6 weeks of gestation when the heart chambers and cerebral

ventricles are forming, there are disturbances in morphogenesis. Furthermore, at 12 weeks of gestation, more effects are noted during the development of terminal digits (K).

Clinical Expression and Pathophysiology

The clinical manifestations of SLOS are extremely variable and the phenotypic spectrum is broad. At the severe end of the spectrum, SLOS is a lethal disorder with multiple major congenital anomalies, and in mild cases SLOS combines minor physical stigmata with behavioral and learning disabilities. Phenotypic features include developmental delay, abnormal genitalia,



as well as hand and toe abnormalities that may include:

Polydactyly



and toe syndactyly.



Short Thumbs



In mild cases, toe syndactyly may be the only evident physical anomaly (Bukelis et al. 2007).

Hirschsprung disease, sexual ambiguity, or partial sex reversal in 46, XY males and structural defects of the heart, lungs, liver, and kidneys may occur (Kelley 2000). Facial and cranial abnormalities that may occur include hypertelorism, narrow forehead, short nose with upturned nares, prominent nasal bridge, soft cleft palate or bifid uvula, posteriorly rotated and low-set ears, micrognathia, high-arched hard palate, and microcephaly (Kelley and Hennekam 2000). Potential ocular abnormalities include cataracts, underdevelopment of the optic nerve, ptosis epicanthus, unequal palpebral fissures, and convergent strabismus. Other central nervous system (CNS) anomalies include agenesis or hypoplasia of the corpus callosum or cerebellum, increased ventricular size, and decreased size of frontal lobes (Nowaczyk and Tierney 2004). In the more severely affected individuals, there may be lethal brain defects such as holoprosencephaly (seen in about 5%) and incomplete myelination (Jira et al. 2003). Structural abnormalities include callosal abnormalities, a Dandy-Walker variant, an arachnoid cyst, and holoprosencephaly (Caruso et al. 2004). The individuals may have severe language impairment (Kelley 1996; Nwokoro and Mulvihill 1997; Tierney et al. 2000, 2001, Tierney et al. 2001; Tint et al. 1994), with greater receptive than expressive language abilities (Kelley 1996; Tierney et al. 2001). Nwokoro and Mulvihill (1997) described a sleep disturbance and Ryan et al. (1998) studied 23 biochemically confirmed subjects aged 6 months and older and found that 70% had a sleep cycle disturbance that usually did not respond to sedatives. Individuals with SLOS have also been described as “hyperactive” (Elias and Irons 1995; Opitz 1999b), and attention deficit hyperactivity disorder was diagnosed in one child, who was reported to have a positive clinical response to treatment with methylphenidate (Nowaczyk et al. 1998). Abnormal aggression occurred in 35 of 56 subjects (63%) in a repeated manner sometime in the past or present, and 89% of individuals with SLOS had repeated self-injury, 54% had self-biting, 48% had head banging, and 54% had opisthokinesis (a highly characteristic arched backward diving motion of the upper body often resulting in the

child's hitting an object). An additional 7 subjects (13%) did not demonstrate the characteristic opisthokinesis but did arch their neck backward frequently, while 17 (30%) did neither (Tierney et al. 2001). Over half of 17 subjects with SLOS meet Autism Diagnostic Interview (ADI-R) and DSM-IV diagnostic criteria for autism (Tierney et al. 2001), and in another study of 14 SLOS subjects, approximately three-fourths (71–86% depending upon the evaluation method) met the criteria for some variant of ASD, with 50% having met the criteria for autism (Sikora et al. 2006).

The characteristic irritability of SLOS continues throughout life, with aggression reported in both children and adults (Pauli et al. 1997; Ryan et al. 1998). Tierney et al. (2001) found that a group of 30 individuals with SLOS, ages 3.2–32.4 years, had auditory, oral, visual, and tactile processing difficulties (sensory hyper-reactivity/hypersensitivity) that was greater than 2 standard deviations from the mean observed with a group of typical subjects (Dunn and Westman 1997), and had statistically greater difficulties with these behaviors than typical subjects (Dunn and Westman 1997), subjects with attention deficit hyperactivity disorder (Bennett and Dunn 1996), Asperger disorder (W. Dunn, personal communication, 1999), autistic disorder (Kientz and Dunn 1997), and other developmental disorders (Ermer and Dunn 1998). Such injuries frequently present and was found upon interview that 89% of subjects had a history of self-injury at some time and that 68% had self-injury within the prior month including less injurious behaviors such as skin picking, and the Nisonger-CBRF scale demonstrated a self-injury rate of 83% that occurred within the 2 months prior to assessment (Tierney et al. 2001).

Evaluation and Differential Diagnosis

Professional organizations such as the American Academy of Neurology and the Child Neurology Society are recommending screening for biochemical and genetic disorders that are associated with ASDs (Filipek et al. 2000). A clinical diagnosis of SLOS or suspected mild SLOS can be

confirmed by biochemical testing. An elevated plasma 7DHC level relative to the cholesterol level establishes the diagnosis, although 7DHC levels can also be measured in other tissues. Furthermore, although the majority of individuals with SLOS have lower than normal cholesterol levels, approximately 10% of affected individuals diagnosed clinically and biochemically with SLOS have normal total cholesterol levels and only mildly elevated levels of 7DHC (Irons 2007), particularly those who have a milder phenotype or are older (Kelley 1995). Thus, a normal value for plasma cholesterol does not exclude the diagnosis of SLOS. Also, different laboratories may report the 7DHC results in either milligrams per deciliter, micrograms per milliliter, or millimoles per liter (Irons 2007).

Some neuroleptic medications for the treatment of behavioral and psychiatric disorders interfere with the activity of enzyme *DHCR7* and mildly lower the production of cholesterol. This may lead to false-positive test results. Molecular genetic testing and/or fibroblasts testing is needed to clarify the diagnosis in those individuals. More than 120 mutations in SLOS have been described (Correa-Cerro and Porter 2005; Yu and Patel 2005).

Genetic Counseling

Due to the autosomal recessive nature of the disorder, each sibling of an affected individual should be tested as there is a 25% chance that an offspring will be affected, a 50% chance of being an asymptomatic carrier, and a 25% chance of being unaffected and not a carrier (Irons 2007). Carriers are heterozygotes for SLOS and are not at risk of developing the disorder and do not have abnormal cholesterol levels. Biochemical testing of fibroblasts has been used for carrier detection (Shefer et al. 1997), as has molecular carrier testing if the *DHCR7* mutations in the family are known. For evaluation of the affected individual, in addition to biochemical testing, molecular genetic testing is used. Most affected individuals are compound heterozygotes for two different abnormal alleles, with an overall mutation detection rate of 96% (Witsch-Baumgartner et al. 2001). It has been hypothesized that the other

mutations that are not detected on molecular testing are due to mutations that regulate the transcription or function of the gene product (Correa-Cerro and Porter 2005). Biochemical testing or molecular genetic testing can be used for prenatal pregnancies (Irons 2007). Knowing the specific mutation mutations that the parents carry can help to assess for risks in the affected offspring. For pregnancies of carriers in families that may result in SLOS, diagnostic elevation of 7DHC levels may be assessed for in amniotic fluid obtained by amniocentesis (Abuelo et al. 1995; Dallaire et al. 1995; Rossiter et al. 1995) or in tissue obtained from chorionic villus samples (CVS) (Mills et al. 1996; Sharp et al. 1997). In fetuses who have a family history of a mild variant form of SLOS, the 7DHC enzyme deficiency will need to be demonstrated in cultured amniocytes (Irons 2007).

Treatment

Cholesterol is needed not only for myelination, neuroactive steroid synthesis, and cell wall structure in the body but is also needed for modulation of the oxytocin receptor, serotonin receptors, and the G-proteins that are necessary for other neurotransmitter function (Bukelis et al. 2007; Lee and Tierney 2011). The current standard treatment for SLOS is to begin dietary cholesterol supplementation as soon as this condition is diagnosed. Cholesterol is supplied in natural form (egg yolk, cream, liver) or as a purified cholesterol suspension; the dosing for the cholesterol suspension typically is started in the range of 40–50 mg/kg/day up to 150 mg/kg/day for maintenance treatment (Linck et al. 2000). Cholesterol supplementation improves the ratio of cholesterol to total sterols (Elias et al. 1997; Irons et al. 1997; Nwokoro and Mulvihill 1997) and can decrease 7DHC levels (Linck et al. 2000).

Tube feeding is often required in infants and younger children because of feeding difficulties. Medical and surgical management of gastroesophageal reflux may be needed. During severe acute illness (e.g., infections) or following major surgical procedures, patients with SLOS may

develop overt adrenal insufficiency requiring fresh frozen plasma as a source of cholesterol.

Treatment of individuals with SLOS with dietary cholesterol supplementation has resulted in limited clinical effects and benefits. Yet, clinical anecdotal reports and open-label studies with cholesterol treatment in children with SLOS have demonstrated reductions in the number of infections, decreased rashes (Elias et al. 1997), reduced photosensitivity (Azurdia et al. 2001; Elias et al. 1997; Starck et al. 2002), and improved speech articulation (Irons et al. 1997). In a study of six children with SLOS (from birth to 11 years) receiving cholesterol supplementation, Elias et al. (1997) reported that all of the six children had decreased problem behaviors, better tolerance of infection, improvement of gastrointestinal symptoms, as well as the improvement in photosensitivity, rashes, and infections noted above. After cholesterol supplement is begun, individuals had decreased irritability (Elias and Irons 1995; Nwokoro and Mulvihill 1997), a happier affect (Irons et al. 1995; Nwokoro and Mulvihill 1997; Pauli et al. 1997; Opitz 1999b), decreased hyperactivity with improved attention (Elias and Irons 1995), and decreased self-injury. Other behaviors reported to improve with cholesterol supplementation include aggressive behaviors (Nwokoro and Mulvihill 1997; Ryan et al. 1998), temper outbursts, trichotillomania, and tactile defensiveness (Nwokoro and Mulvihill 1997). In order to retrospectively compare the behaviors that occurred before and after cholesterol supplementation began, a Parenting Global Rating Form was completed by the parents of 44 subjects who began cholesterol supplementation after early infancy. The parents of 6 subjects (12%) said that there was no change in behavior following treatment with cholesterol. For the remaining 38 subjects (75%) for whom the parents saw behavioral change after cholesterol treatment was initiated, the mean score was 7.9 ± 1.7 on a scale of 1 (terrible) to 10 (excellent), indicating that they believed that cholesterol supplementation had a very positive effect on average (Tierney et al. 2001). Kelley and Hennekam (2000) also reported that irritability and sleep disorders improved rapidly after initiation of therapy in

contrast to other behavioral issues such as tactile sensitivity which improved over a longer period of time. Furthermore, consistent with what is described by many caretakers, they also reported that behavioral abnormalities return when dietary cholesterol therapy is interrupted. Martin et al. (2001), in a case report, described significant improvement in hyperactivity, aggression, and self-injurious behavior within 48 h of initiating dietary cholesterol therapy.

Elias et al. (1997) reported in six children that all demonstrated improved growth and more rapid progress in development after cholesterol supplementation was begun, with increased pubertal progression in the older subjects. In contrast, however, in a longitudinal study of cognitive, motor, and adaptive developmental progress in 14 youths with SLOS receiving cholesterol supplementation, Sikora et al. revealed that despite dietary cholesterol supplementation, developmental quotients did not improve over time (Sikora et al. 2004). Similarly, in a double-blinded placebo-controlled, crossover trial in ten individuals with SLOS, Tierney et al. (2010) found no short-term behavioral effects of dietary cholesterol supplementation. The disparities in clinical improvements demonstrated by these studies may be related to the absence of a direct cholesterol transport mechanism across the blood-brain barrier (Lee and Tierney 2011). It is possible that supplementation with cholesterol beginning in infancy or early childhood may improve cognitive outcomes in SLOS patients. But, to date, no positive effect of cholesterol supplementation on cognitive development has been found in a controlled prospective study. Although Sikora et al. (2004) found that the developmental status of SLOS did not improve over time with cholesterol supplementation, it is possible that supplementation with cholesterol beginning in infancy or early childhood may improve cognitive outcomes in individuals with SLOS.

Dietary cholesterol therapy may impact the development of or presentation of autistic features. Individuals with SLOS treated with cholesterol have been reported to be more sociable, including initiating hugs and being more interactive (Irons et al. 1995; Ryan et al. 1998). Of

subjects with SLOS who began cholesterol supplementation before the age of 5.0 years (9/17), 22% met the criteria for autism at ages 4.0–5.0 years while on supplementation, whereas, of the 8 subjects not supplemented with cholesterol before age 5.0 years, 88% met the criteria for autism at ages 4.0–5.0 years while not on supplementation (Tierney et al. 2001). Although these findings are retrospective and in a small population, they suggest that cholesterol supplementation might have decreased the severity ASD symptoms that would have occurred. Furthermore, no side effects have been reported with cholesterol treatment, which has been in use for about 15 years, serum cholesterol levels are rarely normalized, and elevated 7DHC levels persist. There is data that suggests 7DHC has some toxic effects in that 7DHC increases the degradation of HMG CoA reductase (resulting in lower sterol levels) (Fitzky et al. 2001) and that 7DHC impairs intracellular cholesterol transport (Wassif et al. 2002). O'Brien et al. (2002) suggested in a rat model that correction of the SLOS CNS biochemical defect is associated with return of a normal eye-blink response.

Multiple lines of evidence suggest that statins, a class of medications used to lower cholesterol in those with abnormally high levels, may improve DHCR7 activity, resulting in increased cholesterol levels in individuals with mild DHCR7 deficiency (Jira et al. 2000), in human fibroblasts from mildly affected individuals (Wassif et al. 2005), and in an SLOS mouse model (Correa-Cerro et al. 2006). The mechanism by which statins may increase plasma cholesterol is hypothesized to result from increased expression of a *DHCR7* allele that encodes a mutant enzyme with residual enzymatic function and is supported by in vitro experiments using SLOS fibroblasts (Wassif et al. 2005).

Mildly to moderately raised levels of 7DHC have been found in three psychiatric patients without SLOS who were treated with haloperidol (Nowaczyk and Tierney 2004), with 7DHC levels directly proportional to the dose of haloperidol. 7DHC levels decreased to normal upon haloperidol discontinuation, although it is unknown if an increase in the 7DHC level would have a

beneficial or a deleterious effect. However, the benefits of these medications for behavioral treatment may outweigh the potential risks of lowering cholesterol synthesis. If the behavioral and learning deficits reported in SLOS are related to alterations in sterol composition, rather than fixed developmental defects, then therapeutic interventions designed to alter brain sterol composition may be effective in improving behavioral symptoms (Aneja and Tierney 2008).

References and Reading

- Abuelo, D. N., Tint, G. S., Kelley, R., Batta, A. K., Shefer, S., & Salen, G. (1995). Prenatal detection of the cholesterol biosynthetic defect in the Smith-Lemli-Opitz syndrome by the analysis of amniotic fluid sterols. *American Journal of Medical Genetics*, 56, 281–285.
- Aneja, A., & Tierney, E. (2008). Autism: The role of cholesterol in treatment. *International Review of Psychiatry Journal*, 20(2), 165–170.
- Azurdia, R. M., Anstey, A. V., & Rhodes, L. E. (2001). Cholesterol supplementation objectively reduces photosensitivity in the Smith-Lemli-Opitz syndrome. *British Journal of Dermatology*, 144, 143–145.
- Battaile, K. P., & Steiner, R. D. (2000). Smith-Lemli-Opitz syndrome: The first malformation syndrome associated with defective cholesterol synthesis. *Molecular Genetics and Metabolism*, 71, 154–162.
- Battaile, K. P., Battaile, B. C., Merckens, L. S., Maslen, C. L., & Steiner, R. D. (2001). Carrier frequency of the common mutation IVS8-1G>C in DHCR7 and estimate of the expected incidence of Smith-Lemli-Opitz syndrome. *Molecular Genetics and Metabolism*, 72, 67–71.
- Bennett, D., & Dunn, W. (1996). *Comparison of sensory characteristics of children with and without attention deficit hyperactivity disorder*. Presentation at the American Occupational Therapy Association Conference, Boston.
- Bukelis, I., Porter, F. D., Zimmerman, A. W., & Tierney, E. (2007). Smith-Lemli-Opitz syndrome and autism spectrum disorder. *American Journal of Psychiatry*, 164, 1655–1661.
- Bzdúch, V., Behulova, D., & Skodova, J. (2000). Incidence of Smith-Lemli-Opitz syndrome in Slovakia. *American Journal of Medical Genetics*, 90, 260.
- Caruso, P. A., Poussaint, T. Y., Tzika, A. A., Zurakowski, D., Astrakas, L. G., Elias, E. R., et al. (2004). MRI and ¹H MRS findings in Smith-Lemli-Opitz syndrome. *Neuroradiology*, 46, 3–14.
- Correa-Cerro, L. S., & Porter, F. D. (2005). 3beta-hydroxysterol delta7-reductase and the Smith-Lemli-Opitz syndrome. *Molecular Genetics and Metabolism*, 84, 112–126.
- Correa-Cerro, L. S., Wassif, C. A., Kratz, L., Miller, G. F., Munasinghe, J. P., Grinberg, A., et al. (2006). Development and characterization of a hypomorphic Smith-Lemli-Opitz syndrome mouse model and efficacy of simvastatin therapy. *Human Molecular Genetics*, 15(6), 839–851.
- Cunniff, C., Kratz, L. E., Moser, A., Natowicz, M. R., & Kelley, R. I. (1997). Clinical and biochemical spectrum of patients with RSH/Smith-Lemli-Opitz syndrome and abnormal cholesterol metabolism. *American Journal of Medical Genetics*, 68, 263–269.
- Dallaire, L., Mitchell, G., Giguere, R., Lefebvre, F., Melancon, S. B., & Lambert, M. (1995). Prenatal diagnosis of Smith-Lemli-Opitz syndrome is possible by measurement of 7-dehydrocholesterol in amniotic fluid. *Prenatal Diagnosis*, 15, 855–858.
- Dunn, W. (1999). *Sensory profile caregiver questionnaire*. San Antonio: Psychological Corporation.
- Dunn, W., & Westman, K. (1997). The sensory profile: The performance of a national sample of children without disabilities. *American Journal of Occupational Therapy*, 51, 25–34.
- Elias, E. R., & Irons, M. B. (1995). Abnormal cholesterol metabolism in Smith-Lemli-Opitz syndrome. *Current Opinion in Pediatrics*, 7(6), 710–714.
- Elias, E. R., Irons, M. B., Hurley, A. D., Tint, G. S., & Salen, G. (1997). Clinical effects of cholesterol supplementation in six patients with the Smith-Lemli-Opitz syndrome (SLOS). *American Journal of Medical Genetics*, 68, 305–310.
- Ermer, J., & Dunn, W. (1998). The sensory profile: A discriminant analysis of children with and without disabilities. *American Journal of Occupational Therapy*, 52(4), 283–290.
- Filipek, P. A., Accardo, P. J., Ashwal, S., Baranek, G. T., Cook, E. H., Jr., Dawson, G., et al. (2000). Practice parameter: Screening and diagnosis of autism-report of the quality standards subcommittee of the American academy of neurology and the child neurology society. *Neurology*, 55, 468–479.
- Fitzky, B. U., Witsch-Baumgartner, M., Erdel, M., Lee, J. N., Paik, Y. K., Glossmann, H., et al. (1998). Mutations in the delta7-sterol reductase gene in patients with the Smith-Lemli-Opitz syndrome. *The Proceedings of the National Academy of Sciences Online (U.S.)*, 95, 8181–8186.
- Fitzky, B. U., Moebius, F. F., Asaoka, H., Waage-Baudet, H., Xu, L., Xu, G., et al. (2001). 7-Dehydrocholesterol-dependent proteolysis of HMG-CoA reductase suppresses sterol biosynthesis in a mouse model of Smith-Lemli-Opitz/RSH syndrome. *Journal of Clinical Investigation*, 108(6), 905–915.
- Irons, M. (1993–1998, Updated 2007, October 24). Smith-Lemli-Opitz syndrome. In R. A. Pagon, T. D. Bird, C. R. Dolan, et al. (Eds.), *GeneReviews* (Internet). Seattle: University of Washington.
- Irons, M., Elias, E. R., Abuelo, D., et al. (1995). Clinical features of the Smith-Lemli-Opitz syndrome and

- treatment of the cholesterol metabolic defect. *International Pediatrics*, 10, 28–32.
- Irons, M., Elias, E. R., Abuelo, D., Bull, M. J., Greene, C. L., Johnson, V. P., et al. (1997). Treatment of Smith-Lemli-Opitz syndrome: Results of a multicenter trial. *American Journal of Medical Genetics*, 68, 311–314.
- Jira, P. E., Wevers, R. A., De Jong, J., Rubio-Gozalbo, E., Janssen-Zijlstra, F. S., van Heyst, A. F., et al. (2000). Simvastatin. A new therapeutic approach for Smith-Lemli-Opitz syndrome. *Journal of Lipid Research*, 41, 1339–1346.
- Jira, P. E., Wevers, R. A., de Jong, J., Rubio-Gozalbo, E., Janssen-Zijlstra, F. S., van Heyst, A. F., et al. (2002). Simvastatin, A new therapeutic approach for Smith-Lemli-Opitz syndrome. *The Journal of Lipid Research*, 41, 1339–13346.
- Jira, P. E., Waterham, H. R., Wanders, R. J., Smeitink, J. A., Sengers, R. C., & Wevers, R. A. (2003). Smith-Lemli-Opitz syndrome and the DHCR7 gene. *Annals of Human Genetics*, 67(3), 269–280.
- Kelley, R. I. (1995). Diagnosis of Smith-Lemli-Opitz syndrome by gas chromatography/mass spectrometry of 7-dehydrocholesterol in plasma, amniotic fluid and cultured skin fibroblasts. *Clinica Chimica Acta*, 236(1), 45–58.
- Kelley, R. I. (1996). Smith-Lemli-Opitz syndrome. In P. J. Vinken, G. W. Bruyn, & H. W. Moser (Eds.), *Handbook of neurology, neurodystrophies, and neuropiloidoses* (pp. 581–587). Amsterdam: Elsevier.
- Kelley, R. I. (2000). Inborn errors of cholesterol biosynthesis. *Advances in Pediatrics*, 47, 1–53.
- Kelley, R. I., & Hennekam, R. C. (2000). The Smith-Lemli-Opitz syndrome. *Journal of Medical Genetics*, 37, 321–335.
- Kientz, M. A., & Dunn, W. (1997). A comparison of the performance of children with and without autism on the Sensory Profile. *American Journal of Occupational Therapy*, 51(7), 530–537.
- Lee, R. W. Y., & Tierney, E. (2011). Hypothesis: The role of sterols in autism spectrum disorder. *Autism Research and Treatment*, 2011, 1–7.
- Linck, L. M., Lin, D. S., Flavell, D., Connor, W. E., & Steiner, R. D. (2000). Cholesterol supplementation with egg yolk increases plasma cholesterol and decreases plasma 7-dehydrocholesterol in Smith-Lemli-Opitz syndrome. *American Journal of Medical Genetics*, 93, 360–365.
- Lowry, R. B., & Yong, S. L. (1980). Borderline normal intelligence in the Smith-Lemli-Opitz (RSH) syndrome. *American Journal of Medical Genetics*, 5, 137–143.
- Martin, A., Koenig, K., Scahill, L., Tierney, E., Porter, F. D., & Nwokoro, N. A. (2001). Smith-Lemli-Opitz syndrome. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40(5), 506–507.
- Mills, K., Mandel, H., Montemagno, R., Soothill, P., Gershoni-Baruch, R., & Clayton, P. T. (1996). First trimester prenatal diagnosis of Smith-Lemli-Opitz syndrome (7-dehydrocholesterol reductase deficiency). *Pediatric Research*, 39, 816–819.
- Nowaczyk, M. J. M., & Tierney, E. (2004). *Smith-Lemli-Opitz syndrome: Demystifying genetic syndromes* (pp. 207–223). Kingston: National Association for the Dually Diagnosed.
- Nowaczyk, M. J. M., & Wayne, J. S. (2001). The Smith-Lemli-Opitz syndrome: A novel metabolic way of understanding developmental biology, embryogenesis and dysmorphology. *Clinical Genetics*, 59, 75–76.
- Nowaczyk, M. J., Whelan, D. T., & Hill, R. E. (1998). Smith-Lemli-Opitz syndrome: Phenotypic extreme with minimal clinical findings. *American Journal of Medical Genetics*, 78(5), 419–423.
- Nwokoro, N. A., & Mulvihill, J. J. (1997). Cholesterol and bile acid replacement therapy in children and adults with Smith-Lemli-Opitz (SLO/RSH) syndrome. *American Journal of Medical Genetics*, 68(3), 315–321.
- O'Brien, W. T., Xu, G., Batta, A., Tint, G. S., Salen, G., Dyer, C. A., et al. (2002). Developmental sensitivity of associative learning to cholesterol synthesis inhibitors. *Behavioural Brain Research*, 129(1–2), 141–152.
- Opitz, J. M. (1969). The RSH syndrome. *Birth Defects*, 5, 167–169.
- Opitz, J. M. (1999a). RSH (so-called Smith-Lemli-Opitz) syndrome. *Current Opinion in Pediatrics*, 11(4), 353–362.
- Opitz, J. M. (1999b). The RSH syndrome: Paradigmatic metabolic malformation syndrome. In M. I. New (Ed.), *Diagnosis and treatment of the unborn child*. Reddick: Idelson-Gnocchi.
- Pauli, R. M., Williams, M. S., Josephson, K. D., & Tint, G. S. (1997). Smith-Lemli-Opitz syndrome: Thirty-year follow-up of “S” of “RSH” syndrome. *American Journal of Medical Genetics*, 68(3), 260–262.
- Porter, F. D. (2000). RSH/Smith-Lemli-Opitz syndrome: A multiple congenital anomaly/mental retardation syndrome due to an inborn error of cholesterol biosynthesis. *Molecular Genetics and Metabolism*, 71, 163–174.
- Rossiter, J. P., Hofman, K. J., & Kelley, R. I. (1995). Smith-Lemli-Opitz syndrome: Prenatal diagnosis by quantification of cholesterol precursors in amniotic fluid. *American Journal of Medical Genetics*, 56, 272–275.
- Ryan, A. K., Bartlett, K., Clayton, P., Eaton, S., Mills, L., Donnai, D., et al. (1998). Smith-Lemli-Opitz syndrome: A variable clinical and biochemical phenotype. *Journal of Medical Genetics*, 35, 558–565.
- Sharp, P., Haan, E., Fletcher, J. M., Khong, T. Y., & Carey, W. F. (1997). First-trimester diagnosis of Smith-Lemli-Opitz syndrome. *Prenatal Diagnosis*, 17, 355–361.
- Shefer, S., Salen, G., Honda, A., Batta, A., Hauser, S., Tint, G. S., et al. (1997). Rapid identification of Smith-Lemli-Opitz syndrome homozygotes and heterozygotes (carriers) by measurement of deficient 7-dehydrocholesterol-delta 7-reductase activity in fibroblasts. *Metabolism*, 46, 844–850.
- Sikora, D. M., Ruggiero, M., Petit-Kekel, K., Merckens, L. S., Connor, W. E., & Steiner, R. D. (2004). Cholesterol supplementation does not improve developmental

- progress in Smith-Lemli-Opitz syndrome. *Pediatrics*, 144(6), 783–791.
- Sikora, D. M., Pettit-Kekel, K., Penfield, J., Merkens, L. S., & Steiner, R. D. (2006). The near universal presence of autism spectrum disorders in children with Smith-Lemli-Opitz syndrome. *American Journal of Medical Genetics Part A*, 140, 1511–1518.
- Smith, D. W., Lemli, L., & Opitz, J. M. (1964). A newly recognized syndrome of multiple congenital anomalies. *Journal of Pediatrics*, 64, 210–217.
- Starck, L., Lovgren-Sandblom, A., & Bjorkhem, I. (2002). Cholesterol treatment forever? The first Scandinavian trial of cholesterol supplementation in the cholesterol-synthesis defect Smith-Lemli-Opitz syndrome. *Journal of Internal Medicine*, 252, 314–321.
- Tierney, E., Nwokoro, N. A., & Kelley, R. I. (2000). Behavioral phenotype of RSH/Smith-Lemli-Opitz syndrome. *Mental Retardation and Developmental Disabilities Research Review*, 6(2), 131–134.
- Tierney, E., Nwokoro, N. A., Porter, F. D., Freund, L. S., Ghuman, J. K., & Kelley, R. I. (2001). Behavior phenotype in the RSH/Smith-Lemli-Opitz syndrome. *American Journal of Medical Genetics*, 98, 191–200.
- Tierney, E., Conley, S. K., Goodwin, H., & Porter, F. D. (2010). Analysis of short-term behavioral effects of dietary cholesterol supplementation in Smith-Lemli-Opitz syndrome. *American Journal of Medical Genetics, Part A*, 152A, 91–95.
- Tint, G. S., Irons, M., Elias, E. R., Batta, A. K., Frieden, R., Chen, T. S., et al. (1994). Defective cholesterol biosynthesis associated with the Smith-Lemli-Opitz syndrome. *The New England Journal of Medicine*, 330, 107–113.
- Wassif, C. A., Maslen, C., Kachilele-Linjewile, S., Lin, D., Linck, L. M., Connor, W. E., et al. (1998). Mutations in the human sterol delta7-reductase gene at 11q12-13 cause Smith-Lemli-Opitz syndrome. *The American Journal of Human Genetics*, 63, 55–62.
- Wassif, C. A., Vied, D., Tsokos, M., Connor, W. E., Steiner, R. D., & Porter, F. D. (2002). Cholesterol storage defect in RSH/Smith-Lemli-Opitz syndrome fibroblasts. *Molecular Genetics and Metabolism*, 75(4), 325–334.
- Wassif, C. A., Krakowiak, P. A., Wright, B. S., Gewandter, J. S., Sterner, A. L., Javitt, N., et al. (2005). Residual cholesterol synthesis and simvastatin induction of cholesterol synthesis in Smith-Lemli-Opitz syndrome fibroblasts. *Molecular Genetics and Metabolism*, 85(2), 96–107.
- Waterham, H. R., Wijburg, F. A., Hennekam, R. C., Vreken, P., Poll-The, B. T., Dorland, L., et al. (1998). Smith-Lemli-Opitz syndrome is caused by mutations in the 7-dehydrocholesterol reductase gene. *American Journal of Human Genetics*, 63, 329–338.
- Witsch-Baumgartner, M., Löffler, J., & Utermann, G. (2001). Mutations in the human DHCR7 gene. *Human Mutation*, 17, 172–182.
- Yu, H., & Patel, S. B. (2005). Recent insights into the Smith-Lemli-Opitz syndrome. *Clinical Genetics*, 68, 383–391.

SNAP

- [Strong Narrative Assessment Procedure \(SNAP\), Second Edition](#)
-

SNE

- [Leigh Disease](#)
-

SNP

- [Single-Nucleotide Polymorphism](#)
-

Social

- [Play](#)
-

Social (Pragmatic) Communication Disorder

- [Pragmatic Language Impairment](#)
-

Social Behaviors and Social Impairment

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Definition

Social behaviors encompass all behaviors used to interact and communicate with other people. These comprise behaviors as simple as looking

at another and smiling at them to more complex behaviors such as entering into a crowded room with a group of strangers and beginning to interact with them. Many of these behaviors, from the most simple to complex, are challenging for people with an autism spectrum disorder (ASD). Impairments in the development of social behavior, or social impairments, lie at the heart of autism.

Historical Background

Impairments in social behaviors have been noted from Leo Kanner's (1943) original observations, on which he based his first detailed account of 11 cases of autism. He observed the failure of these children to interact with other people in the usual way, with many preferring to be alone. He wrote about their failure to look at other people and to even notice their presence/absence, such that "Comings and goings, even of the mother, did not seem to register" and "The father or mother or both may have been away for an hour or a month; at their homecoming, there is no indication that the child has been even aware of their absence." Impairments in social interaction and behavior have continued to be considered at the heart of autism, with the various iterations of the DSM (from 1980 onward) including persistent difficulties in social interaction as one of the core diagnostic features.

Current Knowledge

The lack of social reciprocity, considered a core underlying feature of ASDs, is evident from the period of infancy (Barbaro and Dissanayake 2009) and persists throughout development and into adulthood (Taylor and Seltzer 2010; Howlin et al. 2013). Early forms of social impairment are evident in the failure to engage in reciprocal face interactions with the caregiver(s) comprising behaviors such as eye gaze, affect sharing, and bouts of mutual facial and gestural imitation. These early dyadic behaviors culminate in social

routines like peek-a-boo-type games in typical development, which are mutually rewarding for both social partners.

The early deficits in dyadic interactions in autism during infancy lead to concomitant difficulties in triadic interactions, such as engaging in joint attention (shared gaze/affect with a social partner and an object/event of mutual interest) and social referencing. The use of social gestures, such as pointing and waving, typical in prelinguistic children, is also infrequent. Impairments in empathic responding and the early developing positive social behaviors like helping and sharing (collaborative prosocial behaviors) ensue. However, despite these early developing difficulties in social engagement and responding, it is important to note that children with autism develop attachments to their caregivers, which are functionally and qualitatively similar to those of their non-autistic peers (Chandler and Dissanayake 2014).

In early childhood, there is little initiation of social interactions with other people, which becomes clearly evident during the preschool years, in interacting with peers. Children with an ASD are usually found on the periphery of social activity with little social play evident (Macintosh and Dissanayake 2006). Solitary play is the norm unless the child is directly engaged in an interaction with a mature play partner. As a result, peer relationships are limited and, when evident, center around the more narrow interests of the children with an ASD. Thus, friendships are infrequent among these children, leading to missed opportunities to use and practice their social behaviors and skills. The capacity to form and maintain relationships with peers is a crucial developmental task for children, and impairment in this ability is still recognized as a core feature of ASDs (DSM 5, 2013).

The development of perspective-taking skills, critical to the development of social relationships, is compromised in autism. Turn taking in interactions is difficult. Taking another's affective and/or cognitive perspective is crucial to understanding other people as well as understanding the self in relation to others. Indeed, the development of selfhood in autism is atypical

which lies at the core of their social-cognitive difficulties (Hobson 1990; Lombardo et al. 2010). The development of social understanding and social responsiveness is deeply intertwined, and each is compromised in autism. It is important to note that it is not just a lack of social behaviors but also knowledge of how and when to use what social behavior and skills have been acquired that leads to the marked social impairment in autism.

The direct consequence of the social interaction difficulties, particularly found among more able children and adolescents with an ASD, is social isolation and peer rejection, leading to poor social support and reported loneliness (Bauminger et al. 2003). Often, these higher-functioning individuals report a desire for friendships but lack the necessary social skills to go about establishing them. The commonly reported mood (depression and anxiety) problems in these individuals, especially common during adolescence and adulthood (Hedley et al. 2017), are likely to be exacerbated by their social difficulties.

Future Directions

Given the centrality of social impairment in ASDs, the teaching of social behaviors and skills comprises an important component in any intervention program, regardless of age and ability level (White et al. 2007). In addition to teaching social behaviors and skills, a key component of any intervention program is to motivate affected individuals to interact socially with others. The development of social competence relies on this.

More research is sorely needed on the efficacy/effectiveness of intervention programs designed to enhance the social behaviors and skills of infants, children, adolescents, and adults with an ASD. These lifelong developmental disorders are likely to demand developmentally appropriate social intervention treatments that change to accommodate the particular life stage. As with all interventions, treatment efficacy will be enhanced if these begin at the earliest possible time in development.

See Also

- Attachment
- Friendships
- Imitation
- Social Cognition

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Barbaro, J., & Dissanayake, C. (2009). Autism spectrum disorders in infancy and toddlerhood: A review of the evidence on early signs, early identification and early diagnosis. *Journal of Developmental and Behavioral Pediatrics*, 30, 447–459.
- Bauminger, N., Shulman, C., & Agam, G. (2003). Peer interaction and loneliness in high-functioning children with autism. *Journal of Autism Developmental Disorders*, 33, 489–507.
- Chandler, F., & Dissanayake, C. (2014). An investigation of the security of caregiver attachment during middle childhood in children with high-functioning autistic disorder. *Autism*, 18(5), 485–492.
- Hedley, D., Uljarević, M., Wilmot, M., Richdale, A., & Dissanayake, C. (2017). Brief report: Social support, depression and suicidal ideation in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47, 3669–3677.
- Hobson, R. P. (1990). On the origins of self and the case of autism. *Development and Psychopathology*, 2, 163–181.
- Howlin, P., Moss, P., Savage, S., & Rutter, M. (2013). Social outcomes in mid- to later adulthood among individuals diagnosed with autism and average nonverbal IQ as children. *Journal of the American Academy of Child & Adolescent Psychiatry*, 52, 572–581.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Lombardo, M. V., Chakrabarti, B., Bullmore, E. T., Sadek, S. A., Pascoe, G., Wheelwright, S., Suckling, J., Baron-Cohen, S., & the MRC AIMS Consortium. (2010). Atypical neural self-representation in autism. *Brain*, 133, 611–624.
- Macintosh, K., & Dissanayake, C. (2006). A comparative study of the spontaneous social interactions and social skills of children with high-functioning autism and children with Asperger's disorder. *Autism*, 10, 199–220.
- Taylor, J. L., & Seltzer, M. M. (2010). Changes in the autism behavioral phenotype during the transition to adulthood. *Journal of Autism and Developmental Disorders*, 40, 1431–1446.
- White, S. W., Koenig, K., & Scahill, L. (2007). Social skills development in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37, 1858–1868.

Social Camouflaging in Adults with ASD

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Definition

A description of the construct of camouflaging (minimizing the appearance of autistic characteristics during social interactions) and current literature.

Historical Background

The earliest known record of the concept of camouflaging in the academic literature comes from the seminal work of Lorna Wing, who described autistic girls without intellectual disability, who appeared to have better social and communication abilities than comparable males, and so were likely to be missed in clinical assessments (Wing 1981a). As part of this “camouflaging hypothesis,” she also described examples of both males and females learning social rules, which might result in apparently typical social behavior despite underlying difficulties (Wing 1981b). However, the concept of some autistic individuals having superficially improved social skills was not considered in depth by academics for many years afterward.

Meanwhile, both clinicians and autistic individuals themselves described case studies of individuals learning to “pretend to be normal” (Holliday Willey 2015). These accounts often focused on females in particular, and suggested that using specific, learned strategies might mask the appearance of social difficulties in autistic females, and thus make it harder to identify their autism (Kopp and Gillberg 1992). Other clinicians identified or suggested specific camouflaging behaviors, such as suppressing repetitive hand movements, giving scripted answers while chatting, or staying close to others without actually

interacting (Gould and Ashton-Smith 2011; Kreiser and White 2014; Lai and Baron-Cohen 2015; Mandy and Tchanturia 2015), although none of these were tested in large-scale, high-quality studies. The implied consequence of these behaviors was that they made identification of difficulties harder by teachers or clinicians, even if the individual themselves or their parents may be aware of difficulties (Mandy et al. 2012).

Most of these studies focused on camouflaging in females, as part of the consideration of the female autism phenotype, or a female-specific behavioral expression of autism (Lai et al. 2011). It was suggested that autistic females might have more social reinforcements or expectations to adapt their behaviors depending on the social environment (Kreiser and White 2014). A complementary approach at the same time suggested that there may be genetic protective effects which allowed females to compensate for some autistic difficulties, with the possibility of not requiring an autism diagnosis (Dworzynski et al. 2012).

The first studies to explicitly examine the concept of camouflaging were qualitative, and mostly based around interviews with autistic girls, women, and their parents. More strategies were identified, such as mimicking peers’ social behaviors (Cridland et al. 2014), or developing coping strategies to attempt to deal with the exhausting social world (Tierney et al. 2016). Some autistic women suggested that their camouflaging or masking strategies made it harder for them to receive a diagnosis, as clinicians did not understand they were masking some of their symptoms (Bargiela et al. 2016; Milner et al. 2019). These studies all supported the idea that camouflaging may be a mostly female behavior, with gendered consequences for diagnosis and outcomes.

Lai and colleagues (2017) were some of the first to operationalize camouflaging, using a so-called “discrepancy” approach. Camouflaging can be measured as the discrepancy between an individual’s internal autistic status, and their external autistic presentation. By measuring camouflaging in this way in a mixed gender adult sample, Lai and colleagues demonstrated that camouflaging can occur in both males and

females and that there is substantial individual variation in the extent of camouflaging. However, camouflaging is on average greater (discrepancy scores were higher) in autistic adult females than males. A complementary measurement approach, used by Hull et al. (2017), operationalized camouflaging based on the self-reported experiences of autistic adults, known as an observational/reflective approach. In this large qualitative study, the vast majority of autistic adult males, females, and individuals of non-binary gender described using camouflaging strategies at some point in their lives. This analysis produced a conceptual model of camouflaging, including its motivations, strategies, and consequences, which has informed subsequent research.

Current Knowledge

Camouflaging is conceptualized as the process of “hiding behaviour that might be viewed as socially unacceptable or artificially ‘performing’ social behaviour deemed to be more neurotypical” (Lai et al. 2017). It can be deliberately learned, such as gradually reducing stimming behaviors, or automatically performed, for instance, mimicry of others’ speech patterns (Hull et al. 2017). As a relatively new topic of research interest, current knowledge is developing rapidly and the specific characteristics of camouflaging are still being determined.

Motivations for camouflaging include short-term or functional aims, such as performing well in a job interview or avoiding bullying from others, in addition to longer-term and relational aims, such as developing friendships (Cage and Troxell-Whitman 2019; Hull et al. 2017). It is still unclear to what extent these aims are achieved, and which abilities may be needed for successful camouflaging. However, individuals who camouflage deliberately may reflect on their camouflaging strategies in order to better achieve their aims (Hull et al. 2017), while those who are not aware of their use of camouflaging strategies may not be able to manipulate their behaviors.

Strategies for camouflaging have been identified or reported through a variety of mechanisms. Studies following the discrepancy approach described above have measured the extent of camouflaging as the discrepancy between mentalizing abilities and ADOS score (Lai et al. 2017, 2018; Schuck et al. 2019), between theory of mind and ADOS score (Livingston et al. 2018), and between parent-reported autistic traits and various behaviors associated with autism (Boorse et al. 2019; Parish-Morris et al. 2017; Ratto et al. 2018; Rynkiewicz et al. 2016). While this enables comparison of camouflaging between groups, it is harder to identify specific instances of camouflaging in real life.

Studies following an observational/reflective approach have identified specific camouflaging strategies, either through behavioral observation (Dean et al. 2017) or through self-reported camouflaging (Cage and Troxell-Whitman 2019; Hull et al. 2018). Examples of these strategies include playing next to, but not with, other children; developing a script for social situations; and learning social behaviors from others. Camouflaging can be measured using the psychometrically validated self-report Camouflaging Autistic Traits Questionnaire (CAT-Q) in adults.

Although there may be beneficial aims associated with camouflaging, most studies report negative consequences of camouflaging. Autistic individuals have self-reported experiencing exhaustion, mental health difficulties such as anxiety or loneliness, and feeling inauthentic (Hull et al. 2017; Milner et al. 2019; Tierney et al. 2016; Tint and Weiss 2018). Quantitative measures of camouflaging have shown positive associations with depression, anxiety, and suicidal ideation (Cassidy et al. 2018; Hull et al. 2018; Lai et al. 2017). In addition, camouflaging has been proposed as a factor preventing autistic girls and women in particular from receiving an accurate and timely diagnosis (Bargiela et al. 2016; Hull et al. 2017).

Researchers have recently begun to examine gender differences in autistic camouflaging. Discrepancy-based studies have generally found that females have a higher discrepancy between internal and external autistic experiences,

resulting in greater internal experience of autistic characteristics, but lesser external presentation of autism compared to males (Lai et al. 2017; Ratto et al. 2018). However, the proportion of autistic adult males and females who report having camouflaged does not appear to differ; the vast majority of autistic adults of all genders report having camouflaged at some point in their lives (Cage et al. 2018; Cassidy et al. 2018; Hull et al. 2017). Research using self-report measures reveals mixed findings so far (Cage and Troxell-Whitman 2019; Cassidy et al. 2018; Hull et al. 2019).

Future Directions

Although the field of camouflaging research has expanded dramatically since 2015, this is still a very new area with many future directions for research. All research has so far taken place in Western cultures, therefore estimates of the prevalence, consequences, and potential gender differences in camouflaging in other cultures need to be identified. Research has also been limited to autistic individuals without intellectual disability; although it has generally been assumed that individuals with intellectual disability or minimal language do not camouflage, this needs to be actively tested.

Camouflaging is also likely to vary across situations, and across developmental stages, within individuals. Longitudinal studies are necessary to identify when and how camouflaging first develops; most research has focused on adults but some studies have identified camouflaging in adolescents and younger children. Earlier identification of camouflaging can help researchers understand how camouflaging develops across time, and how consequences for mental health arise and, potentially, can be addressed.

Larger scale samples using a variety of methods are needed to systematically answer questions concerning gender differences in autism; once these studies are available, meta-analytic techniques will be useful to integrate multiple different studies to give a more definitive answer. Although some research into camouflaging has included non-binary people (Cage and

Troxell-Whitman 2019; Hull et al. 2017, 2019), there are still many gaps in our understanding of camouflaging by transgender and non-binary individuals, and how gender identity interacts with autistic identity.

Future directions for clinical research should focus on implications for diagnosis and interventions. Longitudinal studies identifying participants pre-assessment can help us understand the role camouflaging plays in the diagnostic process, particularly for females. Many social skill interventions, particularly those advocated for younger children, promote social strategies similar to camouflaging, such as forced eye contact. It is still unclear to what extent camouflaging overlaps with these strategies, but the mental health problems associated with camouflaging suggest that more research is needed to evaluate the longer-term implications of deliberately learned social strategies.

See Also

- CAT-Q
- Clinical Assessment
- Social Behaviors and Social Impairment

References and Reading

- Bargiela, S., Steward, R., & Mandy, W. (2016). The experiences of late-diagnosed women with autism spectrum conditions: An investigation of the female autism phenotype. *Journal of Autism and Developmental Disorders*, 46(10), 3281–3294. <https://doi.org/10.1007/s10803-016-2872-8>.
- Boorse, J., Cola, M., Plate, S., Yankowitz, L., Pandey, J., Schultz, R. T., & Parish-Morris, J. (2019). Linguistic markers of autism in girls: Evidence of a “blended phenotype” during storytelling. *Molecular Autism*, 1–12.
- Cage, E., Di Monaco, J., & Newell, V. (2018). Experiences of autism acceptance and mental health in autistic adults. *Journal of Autism and Developmental Disorders*, 48(2), 473–484. <https://doi.org/10.1007/s10803-017-3342-7>.
- Cage, E., & Troxell-Whitman, Z. (2019). Understanding the reasons, contexts and costs of camouflaging for autistic adults. *Journal of Autism and Developmental Disorders*, 49(5), 1899–1911. <https://doi.org/10.1007/s10803-018-03878-x>.

- Cassidy, S., Bradley, L., Shaw, R., & Baron-Cohen, S. (2018). Risk markers for suicidality in autistic adults. *Molecular Autism*, 9(42), 1–14.
- Cridland, E. K., Jones, S. C., Caputi, P., & Magee, C. A. (2014). Being a girl in a boys' world: Investigating the experiences of girls with autism spectrum disorders during adolescence. *Journal of Autism and Developmental Disorders*, 44(6), 1261–1274. <https://doi.org/10.1007/s10803-013-1985-6>.
- Dean, M., Harwood, R., & Kasari, C. (2017). The art of camouflage: Gender differences in the social behaviors of girls and boys with autism spectrum disorder. *Autism*, 21(6), 678–689. <https://doi.org/10.1177/1362361316671845>.
- Dworzynski, K., Ronald, A., Bolton, P., & Happé, F. (2012). How different are girls and boys above and below the diagnostic threshold for autism spectrum disorders? *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(8), 788–797. <https://doi.org/10.1016/j.jaac.2012.05.018>.
- Gould, J., & Ashton-Smith, J. (2011). Missed diagnosis or misdiagnosis? Girls and women on the autism spectrum. *Good Autism Practice (GAP)*, 12(1), 34–41.
- Hull, L., Petrides, K. V., Allison, C., Smith, P., Baron-Cohen, S., Lai, M.-C., & Mandy, W. (2017). "Putting on my best normal": Social camouflaging in adults with autism spectrum conditions. *Journal of Autism and Developmental Disorders*, 47(8), 2519–2534.
- Holliday Willey, L. (2015). *Pretending to be normal: Living with Asperger's syndrome* (Expanded Edition). London, UK: Jessica Kingsley Publishers.
- Hull, L., Mandy, W., Lai, M.-C., Baron-Cohen, S., Allison, C., Smith, P., & Petrides, K. V. (2018). Development and validation of the Camouflaging Autistic Traits Questionnaire (CAT-Q). *Journal of Autism and Developmental Disorders*, 49(3), 819–833.
- Hull, L., Lai, M.-C., Baron-Cohen, S., Allison, C., Smith, P., Petrides, K. V., & Mandy, W. (2019). Gender differences in self-reported camouflaging in autistic and non-autistic adults. *Autism*. <https://doi.org/10.1177/1362361319864804>.
- Kopp, S., & Gillberg, C. (1992). Girls with social deficits and learning problems: Autism, atypical Asperger syndrome or a variant of these conditions. *European Child & Adolescent Psychiatry*, 1(2), 89–99. <https://doi.org/10.1007/BF02091791>.
- Kreiser, N. L., & White, S. W. (2014). ASD in females: Are we overstating the gender difference in diagnosis? *Clinical Child and Family Psychology Review*, 17(1), 67–84. <https://doi.org/10.1007/s10567-013-0148-9>.
- Lai, M.-C., & Baron-Cohen, S. B. (2015). Identifying the lost generation of adults with autism spectrum conditions. *The Lancet Psychiatry*, 2(11), 1013–1027. [https://doi.org/10.1016/S2215-0366\(15\)00277-1](https://doi.org/10.1016/S2215-0366(15)00277-1).
- Lai, M.-C., Lombardo, M. V., Pasco, G., Ruigrok, A. N. V., Wheelwright, S. J., Sadek, S. A., ... Baron-Cohen, S. (2011). A behavioral comparison of male and female adults with high functioning autism spectrum conditions. *PLoS One*, 6(6), e20835. <https://doi.org/10.1371/journal.pone.0020835>
- Lai, M.-C., Baron-Cohen, S., & Buxbaum, J. D. (2015). Understanding autism in the light of sex/gender. *Molecular Autism*, 6(24), 1–5. <https://doi.org/10.1186/s13229-015-0021-4>.
- Lai, M.-C., Lombardo, M. V., Ruigrok, A. N. V., Chakrabarti, B., Auyeung, B., Szatmari, P., ... MA Consortium. (2017). Quantifying and exploring camouflaging in men and women with autism. *Autism*, 21(6), 690–702.
- Lai, M.-C., Lombardo, M. V., Chakrabarti, B., Ruigrok, A. N., Bullmore, E. T., Suckling, J., ... Baron-Cohen, S. (2018). Neural self-representation in autistic men and women and association with 'compensatory camouflaging'. *Autism*. <https://doi.org/10.1177/1362361318807159>.
- Livingston, L. A., & Happé, F. (2017). Conceptualising compensation in neurodevelopmental disorders: Reflections from Autism Spectrum Disorder. *Neuroscience and Biobehavioral Reviews*. <https://doi.org/10.1016/j.neubiorev.2017.06.005>.
- Livingston, L. A., Colvert, E., Bolton, P., & Happé, F. (2018). Good social skills despite poor theory of mind: Exploring compensation in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*. <https://doi.org/10.1111/jcpp.12886>.
- Mandy, W., & Tchanturia, K. (2015). Do women with eating disorders who have social and flexibility difficulties really have autism? A case series. *Molecular Autism*, 6(1), 6. <https://doi.org/10.1186/2040-2392-6-6>.
- Mandy, W., Chilvers, R., Chowdhury, U., Salter, G., Seigal, A., & Skuse, D. (2012). Sex differences in autism spectrum disorder: Evidence from a large sample of children and adolescents. *Journal of Autism and Developmental Disorders*, 42(7), 1304–1313. <https://doi.org/10.1007/s10803-011-1356-0>.
- Milner, V., McIntosh, H., Colvert, E., & Happé, F. (2019). A qualitative exploration of the female experience of Autism Spectrum Disorder (ASD). *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03906-4>.
- Parish-Morris, J., Liberman, M. Y., Cieri, C., Herrington, J. D., Yerys, B. E., Bateman, L., ... Schulz, R. T. (2017). Linguistic camouflage in girls with Autism spectrum Disorder. *Molecular Autism*, 8(1), 48.
- Ratto, A. B., Kenworthy, L., Yerys, B. E., Bascom, J., Trubanova, A., White, S. W., ... Anthony, G. (2018). What about the girls? Sex-based differences in autistic traits and adaptive skills. *Journal of Autism and Developmental Disorders*, 48(5), 1698–1711.
- Rynkiewicz, A., Schuller, B., Marchi, E., Piana, S., Camurri, A., Lassalle, A., & Baron-Cohen, S. (2016). An investigation of the 'female camouflage effect' in autism using a computerized ADOS-2 and a test of sex/gender differences. *Molecular Autism*, 7(10), 1. <https://doi.org/10.1186/s13229-016-0073-0>.
- Tierney, S., Burns, J., & Kilbey, E. (2016). Looking behind the mask: Social coping strategies of girls on the autistic spectrum. *Research in Autism Spectrum Disorders*, 23, 73–83.

- Tint, A., & Weiss, J. A. (2018). A qualitative study of the service experiences of women with autism spectrum disorder. *Autism*, 22(8), 928–937. <https://doi.org/10.1177/1362361317702561>.
- Wing, L. (1981a). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11, 115–129. <https://doi.org/10.1017/S0033291700053332>.
- Wing, L. (1981b). Sex ratios in early childhood autism and related conditions. *Psychiatry Research*, 5(2), 129–137. [https://doi.org/10.1016/0165-1781\(81\)90043-3](https://doi.org/10.1016/0165-1781(81)90043-3).

status undoubtedly has profound impacts on the diagnosis, severity, and treatment of autism, in which individuals from lower social classes tend to have later diagnoses, harsher symptoms, and less access to psychological and pharmacological treatments. As a result, social class is an important factor to consider when studying autism, especially when studying the efficacy of interventions.

Social Class and Autism

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Definition

Social class, also deemed socioeconomic status, is a categorization of a group or individual within an established hierarchical social structure based on factors such as education, income, occupation, financial stability, and location. In statistical analyses, social class can be used as an independent variable when analyzing large populations and is often reduced to a select few variables (e.g., net income, gross income, parental education, property value, nature of labor) for statistical purposes. Social class often plays a significant role in the etiology of a disease, and lower socioeconomic status tends to correlate with higher rates of incidence and severity. Autism spectrum disorder (ASD) has a more complex intersection with social class than most other diseases, likely attributed to its large variance of symptoms between different patients and the fact that mental illness is more difficult to track and study than physical.

There is no official consensus within the field concerning the prevalence of ASD in one social class over another, as multiple studies and statistical analyses have yielded contradictory results from their various populations. However, socioeconomic

Historical Background

Social class became an established concern surrounding autism when it was first introduced by Leo Kanner's hallmark study (1943) of 11 children exhibiting antisocial symptoms. Each one of these children came from a wealthy family with highly educated parents, and the public eye speculated that autism may be a "rich man's disease." Throughout the following decades, especially the 1960s, multiple reviews of different populations in America and Europe continued the narrative that autism diagnoses were more prevalent in wealthy and middle-class families than those of the lower classes.

In the following two decades, the narrative began to shift, where studies heralded results finding no differences in autism rates between the socioeconomic classes. This literature primarily emerged from the United States, which had varied healthcare policies across different states. More recently, landmark studies from Denmark (Larsson et al. 2005) and Sweden (Rai et al. 2012) explored the effects of a universal healthcare system on the prevalence of ASD. Universal healthcare bridges some (but not all) of the inequities that lower social classes face by providing equal access to healthcare regardless of socioeconomic position. By using a variety of variables that measured socioeconomic status, Larsson et al. (2005) concluded that social class played "little or no role in the etiology of autism in Denmark," whereas the equivalent study in Sweden (Rai et al. 2012) concluded that more children in lower social classes are diagnosed with ASD than those in higher classes.

To this day, there is still no consensus among psychologists and sociologists about ASD's prevalence in certain social classes over others and how it interacts with the disorder's etiology. This phenomenon is due to the fact that across separate studies and analyses, researchers used different methods when selecting eligible patients, categorizing the socioeconomic statuses of families, analyzing the data with various statistical tests, and interpreting those statistics in those publications. Further standardized research is required to obtain a clearer vision surrounding how social class can affect the onset of autism.

Current Knowledge

Social Class and ASD Diagnosis

There is an understanding within the field that families from lower social classes are proportionally given fewer ASD diagnoses than higher-class families due to a variety of factors. The most significant factor is that families from lower social classes in systems that do not provide universal healthcare often find themselves in positions where they cannot afford medical appointments. As a result, they do not address pressing or concerning health necessities because they fear the enormous costs. Proportionally, fewer individuals from low-income backgrounds actively seek out healthcare, and they are widely less confident that they could obtain healthcare if necessary (Kirby 2008). These individuals often consider free clinics or clinics for the uninsured as their sole accessible source of healthcare, and these clinics operate on lower budgets, fewer resources (i.e., language interpreters, appropriate legal/social services, specialized care), and fewer appropriately educated staff. Additionally, the primary function of these free services are not dedicated to psychological disorders, so diagnoses for ASD would be secondary to more noticeable medical concerns. Similar lack of access is seen with rural populations that live hours away from a healthcare professional and even further from someone with psychology expertise.

Another factor in the socioeconomic inequality for ASD diagnoses is expressed by the heavy stigma against mental health. For lower classes, psychological health is perceived as a luxury for those who can afford it (Corrigan 2004). These perceptions are further accentuated in non-Western populations, whose cultural identities accentuate weakness if someone is in need of mental health services. Even if this stigma did not exist, education surrounding autism is sparse, and receiving a diagnosis for ASD typically requires active, educated efforts from a patient's family. Parents of children from lower socioeconomic backgrounds are likely to be less educated, more likely to ingrain stigma around ASD, and less likely to believe that their child has a treatable disorder.

Yet another factor is implicit biases found within experts in child psychiatry, who may misread symptoms or dismiss patients due to their lower socioeconomic status. Child psychiatrists were more likely to give an ASD diagnosis to a child from a higher class, whereas they were more hesitant to give the same diagnosis to a child from a lower class with identical symptoms, whom they were more likely to diagnose with cultural deprivation or attentional deficits (Cuccaro et al. 1996). These implicit biases surrounding lower-class populations and resultant misdiagnoses erect barriers for poorer families to efficient treatments for ASD, causing further healthcare disparities between the different classes.

Social Class, ASD Severity, and Well-Being

A common priority throughout the field surrounding autism is early diagnosis for children. Later diagnoses lead to later interventions, which can progress to harsher symptoms and greater difficulty when attempting to integrate into society. Late diagnoses have also been shown to correlate with higher rates of nonverbal cases and greater antisocial tendencies post-adolescence. Socioeconomic status was a stronger predictor for the age of autism diagnosis than the severity of the symptoms when diagnosed (Fountain et al. 2011), and children with parents of higher education were

often diagnosed earlier than those of parents with lower education.

Due to the late diagnosis and resultant lack of preventative healthcare, children of parents from lower social classes often face less effective interventions resulting in harsher symptoms that are difficult to rectify in adulthood. Also, ASD frequently snowballs with various other mental disorders such as depression, anxiety, or attentional deficits (Liss et al. 2008), as well as physical diseases like obesity and malnutrition. The creation of an intervention that addresses not only autism but also a mix of mood, attentional, and physical complications may not be possible in many patients, especially with the extremely severe cases found in children who are diagnosed late. Early diagnosis is inconsequential if the resulting interventions are ineffective, and families from a lower social class are disadvantaged both in terms of age of diagnosis and intervention efficacy. (For focus on the latter, see section “[Social Class and ASD Treatment: Pharmaceutical and Behavioral.](#)”)

Accompanying low socioeconomic status are other trends that affect the severity of the disorder. For example, lower amounts of family involvement correlate with greater severity of the disorder (Dunlap 1999). Parents of lower-class families tend to work long hours, especially single parents with multiple children. As a result, the lack of parent involvement in a child’s life can increase the severity of his/her symptoms, which can also harm the development of self-determination and well-being of the adolescent (Dunlap 1999).

ASD also augments the severity of ailments in other, more specialized realms of health, a phenomenon exemplified by dental care. Dentistry is inherently invasive and requires a stationary patient, so children with autism are difficult patients to treat. Though research continues in order to make the dentist’s office a more accommodating place for individuals with ASD (Marshall et al. 2007), many of these patients, especially those with parents of low education, completely neglect dentistry. Similar “luxury” fields of health like optometry, physical therapy, and nutrition counseling are likewise

neglected, though they may be necessary for an improved quality of life for the patient. Many guardians who come from uneducated backgrounds fear bringing an individual with ASD into these offices given the stigma of the disorder. Even if they overcome this fear, locating educated, willing physicians and counselors who can administer these services to patients with ASD may also be difficult given the limited resources of a less educated guardian.

Ultimately, the diagnosis of ASD is a catalyst for important changes in a patient’s life. However, without effective interventions that address both mental and physical diseases that accompany the disorder and basic support systems like a stable family and home, ASD can increase in severity and actively compound with other health deficiencies to harm a child’s well-being. Families from a lower socioeconomic status lack the education, resources, and time to provide their child with these various support and treatment systems, resulting in disproportionately more severe cases attributed to low-income, less-educated families.

Social Class and ASD Treatment: Pharmaceutical and Behavioral

The treatment of autism can be expensive and cause significant financial losses as patients undergo various interventions to treat the disorder. Families from lower classes tend to be barred from empirically tested treatments due to high costs and inconveniences. Given the difficulties of transportation, location, and finding sufficient time, in addition to certain stigmas and unconscious bias from healthcare providers against poorer individuals, families from lower classes are at a disadvantage when it comes to actively treating an individual’s autism.

Specifically, some pharmaceuticals for behavioral disorders have been empirically tested to alleviate some of ASD’s symptoms (Ratey et al. 1987), and the costs of these pharmacological interventions are often too high for families of lower classes. Although these pharmaceuticals do not address the specific social disabilities of ASD, they can greatly improve the quality of life for both the child and surrounding family members. However, for families from a lower social class, issues of cost immediately come to mind as

they consider these kinds of treatments. Pharmacological interventions require not only the drugs themselves but also consistent appointments with child psychiatrists and pharmacists. Many families from lower classes simply cannot keep up with these regular expenses and forfeit their child's treatment before completing the regimen. Though paid trial sessions for experimental interventions could waive some of the accompanying costs, the field is bioethically critical of allowing populations from lower classes to take the brunt of the trauma from untested procedures.

On the other side of treatment is active behavioral therapy, which consistently demonstrates its efficacy in improving the symptoms of ASD (Reaven et al. 2012). Families of lower social class run into identical problems as those in pharmacological interventions (i.e., lack of transport, low availability of centers/clinics, significant debt from therapy sessions). These regular expenses immediately make individualized therapy inaccessible to most families in lower classes. Though specific insurance measures have been placed throughout Western countries to allow families from low-income backgrounds to receive healthcare (e.g., free universal healthcare in Europe, Medicaid in the United States), psychotherapy is still viewed as a luxury (Kataoka et al. 2002). Additionally, many private insurance companies do not make provisions for mental health in their coverage, so families from both lower and middle classes cannot afford psychiatric therapy for their child with ASD.

Aside from traditional therapy sessions, innovative programs like Ramapo in New York, Chapel Haven in Connecticut, and Camp Talisman in North Carolina are similarly financially inaccessible for low-income patients. These programs use camp or dormitory settings to promote teamwork and social interactions with others. Need-based scholarships are scarce within these programs, and the vast majority of the students who attend these programs are from wealthy socioeconomic backgrounds who can afford the cost of transitioning into a social world.

Combinations of these various treatments – pharmacological, behavioral, and nontraditional – can become extremely expensive, and families

who cannot initially afford such programs could develop severe debt. Unlike college educations, a child with ASD is not likely to return on these investments with a consistent job, so these interventions are administered with little chance of recouping finances. As a result, families from different socioeconomic backgrounds address the future of their children with ASD differently.

It is possible for therapy to continue through adulthood, and some individuals with ASD will be able to integrate themselves into a society with regular assistance from a mental health professional. However, many individuals from lower socioeconomic statuses will not improve, especially given the disadvantageous late diagnoses and lower-quality interventions that accompany lower social classes, and families must continue expensive care for the afflicted member. With resources for adults with ASD (e.g., care homes) costing multiple thousands of dollars a year (Carbone et al. 2010), ASD can consume the lives of families from lower social classes even through an afflicted individual's adulthood.

Future Directions

To better understand the intersection of ASD and social class, the field must conduct further research to understand how social class can affect the etiology of autism. In these future studies, standardization of eligible patients, variables calculated, and how these variables are interpreted is critical in order to better understand how socioeconomic status contributes to autism's symptoms in individuals.

Additionally, there is a consensus in the field that increasing mental health awareness in educational systems worldwide is absolutely critical, especially in low-income neighborhoods. Standardized earlier interventions for autism with routine screenings in all schools would allow for less prevalence of severe ASD in adulthood. Also, this kind of education will foster a culture against bullying toward those with mental disorders in schools (Van Roekel et al. 2010) and increase the quality of afflicted children's lives. As a result, further studies should explore the most effective

ways to raise awareness of ASD for parents of children, expert psychologists, healthcare professionals, teachers, and classmates in order to address mental health as a whole, especially in impoverished areas.

See Also

- Cognitive Behavioral Therapy (CBT)
- Diagnosis and Classification
- Early Diagnosis
- Time Trends in Diagnosis
- Verbal Behavior Interventions

References and Reading

- Carbone, P. S., Behl, D. D., Azor, V., & Murphy, N. A. (2010). The medical home for children with autism spectrum disorders: Parent and pediatrician perspectives. *Journal of Autism and Developmental Disorders*, 40(3), 317–324.
- Corrigan, P. (2004). How stigma interferes with mental health care. *American psychologist*, 59(7), 614.
- Cuccaro, M. L., Wright, H. H., Rownd, C. V., Abramson, R. K., Waller, J., & Fender, D. (1996). Brief report: Professional perceptions of children with developmental difficulties: The influence of race and socioeconomic status. *Journal of Autism and Developmental Disorders*, 26(4), 461–469.
- Dunlap, G. (1999). Consensus, engagement, and family involvement for young children with autism. *Journal of the Association for Parents with Severe Handicaps*, 24(3), 222–225.
- Fountain, C., King, M. D., & Bearman, P. S. (2011). Age of diagnosis for autism: Individual and community factors across 10 birth cohorts. *Journal of Epidemiology and Community Health*, 65(6), 503–510.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kataoka, S. H., Zhang, L., & Wells, K. B. (2002). Unmet need for mental health care among US children: Variation by ethnicity and insurance status. *American Journal of Psychiatry*, 159(9), 1548–1555.
- Kirby, J. B. (2008). Poor people, poor places and access to health care in the United States. *Social Forces*, 87(1), 325–355.
- Larsson, H. J., Eaton, W. W., Madsen, K. M., Vestergaard, M., Olesen, A. V., Agerbo, E., Schendel, D., Thorsen, P., & Mortensen, P. B. (2005). Risk factors for autism: Perinatal factors, parental psychiatric history, and socioeconomic status. *American Journal of Epidemiology*, 161(10), 916–925.
- Liss, M., Mailloux, J., & Erchull, M. J. (2008). The relationships between sensory processing sensitivity, alexithymia, autism, depression, and anxiety. *Personality and Individual Differences*, 45(3), 255–259.
- Marshall, J., Sheller, B., Williams, B. J., Mancl, L., & Cowan, C. (2007). Cooperation predictors for dental patients with autism. *Pediatric Dentistry*, 29(5), 369–376.
- Rai, D., Lewis, G., Lundberg, M., Araya, R., Svensson, A., Dalman, C., Carpenter, P., & Magnusson, C. (2012). Parental socioeconomic status and risk of offspring autism spectrum disorders in a Swedish population-based study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(5), 467–476.
- Ratey, J. J., Bemporad, J., Sorgi, P., Bick, P., Polakoff, S., O'Driscoll, G., & Mikkelsen, E. (1987). Brief report: Open trial effects of beta-blockers on speech and social behaviors in 8 autistic adults. *Journal of Autism and Developmental Disorders*, 17(3), 439–446.
- Reaven, J., Blakeley-Smith, A., Culhane-Shelburne, K., & Hepburn, S. (2012). Group cognitive behavior therapy for children with high-functioning autism spectrum disorders and anxiety: A randomized trial. *Journal of Child Psychology and Psychiatry*, 53(4), 410–419.
- Van Roekel, E., Scholte, R. H., & Didden, R. (2010). Bullying among adolescents with autism spectrum disorders: Prevalence and perception. *Journal of Autism and Developmental Disorders*, 40(1), 63–73.

Social Cognition

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Definition

The mental processes in which individuals are involved with when making sense of the social world, including the understanding of self, others, and the interplay between self and others, are what is called social cognition (Lewis 1999; Beer and Ochsner 2006). Social cognition broadly includes the cognitive processes that enables spontaneous decoding and encoding (provide correct interpretation) of verbal and nonverbal social and emotional cues, the ability to recognize central and peripheral social and emotional information, the knowledge of different social behaviors and their consequences in diverse social tasks (e.g., social

knowledge and understanding), and the ability to make an adequate attribution about another person's mental state (i.e., "theory of mind" abilities, Perner and Wimmer 1985), as well as about the self (Crick and Dodge 1994; Lewis 1999). Social cognition, thus, is including information processing about all people, including the self, and about the norms and procedures of the social world. The knowledge about social norms and procedures is both declarative and procedural. By large, declarative knowledge is what the individual knows about social concepts, scripts, relations, and phenomena, and procedural knowledge is the knowledge of rules, skills, and strategies that enables the use of declarative knowledge in order to react or respond efficiently in diverse social situations.

See Also

- [Attention](#)
- [Frontal Lobe Findings in Autism](#)
- [Pragmatics](#)
- [Theory of Mind](#)

References and Reading

- Beer, J. S., & Ochsner, K. N. (2006). Social cognition: A multi level analysis. *Brain Research*, 1079, 98–105.
- Crick, N. R., & Dodge, K. A. (1994). A review and reformulation of social information-processing mechanisms in children's social adjustment. *Psychological Bulletin*, 115, 74–101.
- Lewis, M. (1999). Social cognition and the self. In P. Rochart (Ed.), *Early social cognition: Understanding others in the first months of life* (pp. 81–100). Mahwah: Lawrence Erlbaum.
- Perner, J., & Wimmer, H. (1985). "John thinks that Mary thinks that...": Attribution of second-order false beliefs by 5- to 10-year-olds. *Journal of Experimental Child Psychology*, 39, 437–471.

Social Cognition Training

- [Cognitive Enhancement Therapy](#)

Social Cognitive Interventions

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Synonyms

[Emotion recognition](#); [Perspective taking](#); [Social learning](#); [Social skills](#); [Social thinking](#); [Theory of mind](#)

Definition

Social impairments are among the most recognizable features of autism spectrum disorder (ASD), presenting early in infancy and persisting through the lifespan. Reduced affect recognition and difficulty in predicting thoughts and feelings in others are critical features of ASD – particularly in verbal, cognitively able individuals. Advances in social neuroscience led to identification of key neural networks underlying social cognition with consistent findings of ASD-specific neural responses in regions associated with perception of biological motion, face and emotion recognition, and perspective taking.

The foundation of advanced social cognitive skills such as perspective taking and empathy lie in early social communication behaviors such as imitation and joint attention. Recently, several efficacious interventions have been established for critical early social communication behaviors in young children with ASD. Targeted behavioral interventions for social cognition are emerging and typically utilize social group formats. Results from randomized, comparative trials of social cognitive skills groups suggest moderate, short-term effects on informant reports of social behavior and quality of life. However, changes on neuropsychological measures of social cognition are less consistent and with small effects. Pilot, wait-list controlled trials utilizing theatrical and drama-based group curricula have also been studied with findings of moderate to small effects on behavioral and cognitive outcomes.

Technology-based interventions involving gaming and virtual reality are also emerging as potential treatments for social cognitive impairments in ASD. To date, game-based packages for facial processing, knowledge of emotional management strategies, and affect recognition/understanding have shown small effects on task-specific measures, with largely unknown effects on more distal measures, maintenance, or generalizability.

Taken together, promising targeted social cognitive group therapy are in the treatment development pipeline. Persistent concerns about size, generalizability, and durability of treatment effects remain to be addressed. In addition, the complexity and heterogeneity of ASD across development suggests interventions for higher-level skills such as social cognition must target multiple domains. Future research may consider multimodal interventions of empirically supported psychosocial interventions and promising pharmacotherapies to address complex social, behavioral, cognitive, and psychiatric needs of older children and individuals with ASD.

References and Reading

- Goods, K. S., Ishijima, E., Chang, Y. C., & Kasari, C. (2013). Preschool based JASPER intervention in minimally verbal children with autism: Pilot RCT. *Journal of Autism and Developmental Disorders*, 43(5), 1050–1056.
- Pelphrey, K. A., Shultz, S., Hudac, C. M., & Vander Wyk, B. C. (2011). Research review: Constraining heterogeneity: The social brain and its development in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 52(6), 631–644.
- Ramdoss, S., Lang, R., Mulloy, A., Franco, J., O'Reilly, M., Didden, R., & Lancioni, G. (2011). Use of computer-based interventions to teach communication skills to children with autism spectrum disorders: A systematic review. *Journal of Behavioral Education*, 20(1), 55–76.
- Soorya, L. V., Siper, P. M., Beck, T., Soffes, S., Halpern, D., Gorenstein, M., Kolevzon, A., Buxman, J., & Wang, A. T. (2015). Randomized comparative trial of a social cognitive skills group for children with autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 54(3), 208–216.
- White, S. W., Keonig, K., & Scahill, L. (2007). Social skills development in children with autism spectrum disorders: A review of the intervention research. *Journal of Autism and Developmental Disorders*, 37(10), 1858–1868.

Social Cognitive Learning Theory

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Definition

Social cognitive learning theory is a broad-ranging theoretical model of human behavior and is a major contribution to the social and behavioral sciences developed by Albert Bandura. Social cognitive learning theory posits that individuals are active agents in their development, not simply passive observers. In this model, there is a dynamic, continuous interrelationship between an individual's behaviors, the environment, and intrapersonal factors (i.e., cognitive, affective, and biological events) during development (Bandura 1986, 1999). The model seeks to explain the determinants of behavior as well as the dynamic conditions that can change behavior over time.

Historical Background

Social cognitive learning theory grew from Miller and Dollard's initial work in the area of social learning theory in 1941. Social learning theory provides a foundation for the development of social cognitive learning theory, embodying the idea that if an individual were motivated to learn a particular behavior, it would be learned through

observation and imitation of actions. Social cognitive learning theory expanded on this premise and is the product of decades of research and analysis of human functioning through the lens of learning frameworks. In 1963, Bandura and Walters wrote the seminal work *Social Learning and Personality Development*, which set forth the most comprehensive and complex account of social learning theory with its emphasis on observational learning and vicarious reinforcement. Social learning theory was a departure from prevalent models of development of the time, such as psychoanalysis and behaviorism (Grusec 1992). Psychoanalysts described behavior as driven by internal impulses, whereas behaviorists described behavior as completely shaped by the environment (Davis and Luthans 1980; Waller 2004). Social learning theory, while drawing on behaviorism in some ways, depicted behavior as a product of both internal factors and environmental factors and was the first major neo-behavior analytic theory to ascribe a role to cognition.

Bandura considered observational learning and imitation to be core components of social learning theory (Bandura 1969). Social learning theory posited that observational learning was a more effective method for behavioral change than direct learning or successive approximations (Grusec 1992). Social learning theory also conceptualized imitation differently than previous frameworks (Bandura and Walters 1963; Grusec 1992). In social learning theory, imitative responses did not need to be reinforced for observational learning to occur (Grusec 1992).

In 1986, Bandura renamed this framework from “social learning theory” to “social cognitive theory” with the publication of *Social Foundations of Thought and Actions: A Social Cognitive Theory*. The renaming of the theory demonstrated the new emphasis on cognitive processes. The new terminology helped to better represent the components and processes he had been advocating since the early 1960s (Grusec 1992). From 1986 on, Bandura consistently referred to the theory as social cognitive theory although the underlying framework remained the same.

This entry refers to the framework as “social cognitive learning theory” (SCLT) which combines the two names given by Bandura for the

framework and is used by some scholars (e.g., Money 1996). This term will be used throughout the remainder of this entry.

Current Knowledge

Core Concepts of SCLT

According to SCLT, there is a dynamic relationship between behavior, cognition, and other personal and environmental influences where each factor continually influences and impacts the others (i.e., triadic reciprocal determinism) (Bandura 1989). Individuals are both producers and products of their environment (Bandura 1989). That is, the way people interpret their behavior impacts and alters their beliefs and the environment they operate in, which then affects their future behaviors (Bandura 1986).

SCLT proposes that individuals are not simply passive observers but active agents in their development; therefore, it takes an agentic perspective to human functioning (Bandura 2005). That is, humans have control over their thoughts and behavior. They are active planners, forethinkers, self-regulators, and self-examiners by integrating their experiences to actively interact within their environment (Bandura 2005). The centrality of human agency is embodied by six core concepts that are fundamental to SCLT: the ability to symbolize and engage in forethought, vicarious learning, self-regulatory mechanisms, self-reflection, and self-efficacy (Bandura 1986). They place the individual as an active participant in the dynamic relationship between the environment, the individual’s cognitions, personal factors, and behaviors rather than a passive bystander of environmental variables or unconscious internal conflicts. These key concepts are described in more detail below.

Bandura noted that symbolization of thoughts allows people to store information that can be utilized for guiding future behavior (Bandura 1989). Symbols are mental representations of thoughts or images, which allow people to have structure, meaning, and continuity in their lives (e.g., language and gestures) to navigate their world, understand their environment, and problem solve (Malone 2002). For example, a child

develops a mental image of a smile and simultaneously assigns emotional and verbal meaning to this representation such as “a smile indicates that someone is friendly and approachable.” He later recalls and builds onto this “symbol” based on his experiences. The child’s internal representation of a smile influences him to respond positively to a smiling peer (e.g., by greeting or asking the peer to play). It is proposed that the use of symbols allows for individuals to model observed behavior (Bandura 1971).

The concept of forethought is that individuals can assess the likely consequences of their actions before they actually engage in an activity. Forethought utilizes symbolic representations in order to create future-directed plans (Bandura 2005). For example, when approaching a group of people engaged in a conversation, a person may mentally assess a range of strategies to enter the conversation. The choices can vary from appropriate such as making an on-topic comment (e.g., “Hi, that’s a funny story you just shared!”) to inappropriate (e.g., spontaneously dancing in front of the group to get their attention). The person assesses these choices and associated outcomes and then selects the strategy that would achieve the desired result (e.g., entering the conversation by commenting on the topic in order to be welcomed by the group). It is the mechanism individuals use to create a course of action, set goals, and predict the likely consequences of behavior, which motivate and influence future behavior (Bandura 2005).

Vicarious learning, also known as observational learning, is a departure from previous behavioral models of learning. Rather than depending solely on one’s own actions and experiences as a source of learning, Bandura suggested that individuals learn by observing and modeling others’ behavior. As such, individuals learn novel behaviors without the process of trial and error associated with direct learning (Bandura 1986); learning by example reduces error in behavior. Observational learning involves modeling behavior, particularly when the observed behavior is rewarded. It is guided by attention to the model’s actions, forming symbolic representations of these new behaviors in memory, performing the

observed behavior, and experiencing motivation to produce the actions. For example, a child may observe a friend successfully join a game by seeing how the friend first stands next to and watches other children play, waits for an appropriate pause, and finally expresses an interest in joining the game (e.g., “I want to play ball, too!”). The child observes that these behaviors were successful in entering a play situation. She later recalls and engages in similar behaviors when presented with a related social scenario. The developmental level of the learner may also influence learning and behavior, which has potential consequences for individuals on the autism spectrum (discussed in more detail in the next section). Vicarious learning facilitates the acquisition of information that guides behavior (Grusec 1992).

Self-reflection is the ability for people to evaluate their experiences. It is a mechanism that allows people to ultimately adapt behaviors and cognitions according to what they have seen, heard, felt, realized, and so forth. For example, after experiencing a difficult conversation with a friend, a teenager may recall the conversation. He evaluates whether the situation was handled appropriately by mentally reviewing his interactions as well as that of his friend. He assesses his feelings about the overall interaction and what actions he could have done differently to make the conversation more successful. Bandura identified self-reflection as the most “distinctly human” capability (Bandura 1986, p. 21).

Self-regulation refers to the ability to make adaptations to behavioral responses (Bandura 2003, 2005). Motivation and performance are not solely based on material incentives but social influences and personal standards as well (Bandura 2005). Personal standards are used as a yardstick for monitoring and comparing behaviors. If one’s behavior does not measure up to these standards, the individual will self-evaluate and adjust behaviors accordingly. For example, a girl sees her friend in a quiet library and enthusiastically greets her peer. The librarian at the library responds by exclaiming “shh!” The girl takes into account this information from her environment (i.e., the feedback from others that she needs to speak quietly) and adapts her behaviors

by speaking at a lower volume and standing closer to her peer to continue the interaction. The ability to engage in the self-regulation is shaped by the ability to accurately and reliably engage in self-observation and self-monitoring (Bandura 2003).

Self-efficacy, the belief that one is able to produce desired outcomes, is fundamental to SCLT. It is viewed as a foundation for motivation, confidence, emotional and psychological well-being, and understanding and explaining people's actions (Bandura 1977; Bandura et al. 1977). Bandura (1986) defines self-efficacy as "people's judgments of their capabilities to organize and execute courses of action required to attain designated types of performances" (p. 391); so, it is a belief about one's capabilities, rather than necessarily knowing what to do. For example, a person with high self-efficacy about his social skills will feel confident in his ability to successfully enter social conversations at a party. Consequently, he will be more likely to try entering new social situations and interact with unfamiliar people because he is sure that his strategies will lead to positive responses from others. Self-efficacy is essential for behavior change (Bandura and Walters 1963; Bandura 1977) because a person's belief in the capability of producing a desired outcome influences the incentive to act or change. Poor perceptions of self-efficacy will weaken motivation to persevere in the face of challenges. Self-efficacy beliefs are developed through successful experiences in particular situations (defined in SCLT as mastery of experiences), seeing a person similar to oneself succeed (vicarious experiences), being told that one is capable of succeeding (social persuasion), and positive interpretation of an event (reduction of stress reactions and negative affect) (Bandura 1994).

In summary, SCLT posits that specific cognitive processes mediate behaviors. In both direct and indirect learning experiences, the environment and thoughts affect one another in an intertwined fashion. These theoretical underpinnings provide an overarching understanding of human development. The theory has been highly influential in psychology, providing foundations for mid-level theories as well as intervention programs. It has provided a conceptual framework for

some mid-level cognitive behavioral theories because it embraces the combined importance of external factors (i.e., experience and social contexts) and internal factors (i.e., cognition) in understanding and predicting human behavior.

SCLT in Understanding Autism Spectrum Disorder

As outlined above, SCLT suggests that an individual's development and functioning relies on one's ability to symbolize, to engage in forethought, learn vicariously, self-regulate, self-reflect, and experience self-efficacy (Bandura 1986). Individuals with ASD may have limitations in a number of these areas, and these impairments likely have important consequences on their development, behavior, and capacity to benefit from intervention strategies.

Impairment in the ability to symbolize, for example, is consistently reported in ASD (e.g., Bernabei et al. 1999). These challenges are evidenced by lack of imagination or pretend play (Jarrold 2003; Varga 2010), deficits in theory of mind (Peterson et al. 2009), and limited use of gestures and other nonverbal means of communication (Shumway and Wetherby 2009). Such deficits impede one's ability to make sense of perceived information from people and their environment, create mental representations, and apply this to their behavioral repertoire. This can have detrimental consequences in terms of social cognitive development and understanding one's social milieu.

Evidence suggests that individuals with ASD exhibit atypical visual scanning and attention (Brenner et al. 2007), which impacts the ability to benefit from observational learning. For example, individuals with ASD may have difficulty processing visual information using a top-down strategy (Loth et al. 2011). Whereas typically developing individuals are able to use prior knowledge and context to perceive fragmented or degraded images as recognizable stimuli (e.g., faces), individuals with ASD often attend to local features of a stimulus when processing visual information (e.g., Nakahachi et al. 2008; Rondon and Deruelle 2007). In other words, individuals with ASD may attend to details rather than relying

on prior knowledge and experience and context to interpret ambiguous stimuli. In addition, evidence suggests that rather than attending to pertinent features of a face (e.g., eyes), individuals with ASD tend to devote attention to the mouth when viewing faces (e.g., Klin et al. 2002). It has been hypothesized that these patterns of attention contribute to face processing and recognition deficits associated with ASD (Golarai et al. 2006), demonstrating how atypical visual attention impacts interpretation of social stimuli. Atypical processing of social stimuli undoubtedly impacts the ability to garner important information from one's environment, impeding successful observational learning. It has also been demonstrated that individuals with ASD fail to orient to social stimuli (e.g., their name being called; Dawson et al. 1998), indicating they may not be motivated to attend to social stimuli and likely miss critical cues in their environment. Failure to attend to social cues suggests that individuals with ASD perceive, assimilate, and are shaped by a set of informational cues that differs from typically developing individuals. Studies of young children have also demonstrated that ASD is characterized by challenges with disengagement of attention, or difficulty disengaging from one stimulus and shifting eye gaze to a new stimulus (Landry and Bryson 2004; Zwaigenbaum et al. 2005). This impedes successful visual scanning of one's environment and ability to process salient stimuli, which are critical features of observational learning. Finally, it has consistently been reported that individuals with ASD have impairments in joint attention, or the sharing of attention between two people to reference a stimulus, and this finding is considered a token feature of ASD (e.g., Mundy et al. 2009). This skill is fundamental to learning about one's environment and developing symbolic thought. Within a SCLT framework, deficits in these areas may have serious implications in terms of one's ability to learn from their environment, develop appropriate social skills, and function in everyday life.

According to SCLT, individuals with ASD may experience further disadvantage given their

impaired imitation (i.e., modeling) skills (Williams et al. 2004). Evidence suggests that mirror neuron dysfunction may partially account for these difficulties (e.g., Williams 2008). Mirror neurons fire not only when an individual is actively imitating but also when observing others executing the same actions (e.g., Iacoboni and Dapretto 2006). Impairments in this neural system would suggest that in addition to the impact on the ability to imitate, individuals with ASD may not process another's actions when observing them. This can have detrimental consequences on the capacity to learn from one's environment and integrate social information into their schema.

Another aspect of autism is the failure to develop developmentally appropriate friendships (Bauminger and Kasari 2000; Chamberlain et al. 2006). This challenge may impact both motivation in observational learning and development of self-efficacy. Motivation, in observational learning, refers to the incentive to reproduce the observed behavior (Bandura 1986; Malone 2002). The incentive for observational learning behavior is often a form of social reinforcement or approval. Social reinforcement or approval may be less motivating for individuals with autism due to social deficits, such as social and emotional reciprocity and developmentally appropriate relationships with peers (Bauminger and Kasari 2000; Chamberlain et al. 2006).

These challenges associated with ASD have consequences on one's capacity to perceive and assimilate salient information from their environment, thereby impeding appropriate social development according to SCLT. Intervention strategies targeted at these specific deficits (e.g., visual attention, imitation) may provide individuals with ASD with the opportunity to more efficiently and successfully acquire information from their environment and from other people. For example, Faja and colleagues (Faja et al. 2008) developed a computerized face-training program to facilitate proper visual attention and improved face recognition in individuals with ASD. And, teaching joint attention to young children with

autism has led to improvements in related skills, including imitation and spontaneous speech (Whalen et al. 2006). These examples demonstrate how targeting skills related to SCLT can have broad effects on social functioning in ASD.

SCLT and Interventions for ASD

Video modeling is a form of behavioral intervention used to target numerous behaviors in individuals with autism. In video modeling, the model is shown on a video rather than live, in an attempt to change a behavior or help the individual with autism learn a new, appropriate behavior (Grant and Evans 1994). Generally, the individual views the video, observing the model engaging in the appropriate behavior, and then imitates the target behavior (Charlop-Christy et al. 2000). Video modeling has been shown to be an effective, and rapid, behavioral intervention in numerous single-case design studies. It has been shown to be effective for low- and high-functioning individuals with autism (Charlop-Christy et al. 2000). Video modeling incorporates many tenets of SCLT in order to promote behavioral changes in individuals with autism.

Primarily, video modeling incorporates the four processes required for observational learning, attention, retention, symbolic representation, and motivation, in order to enhance the effectiveness of observational learning for individuals with autism. Charlop-Christy et al. (2000) found that video modeling was a more effective behavioral intervention than in vivo modeling for individuals with autism. Video modeling improved the attention of individuals with autism as compared to in vivo modeling. Many individuals with autism have difficulty with stimulus overselectivity, failing to perceive the entire stimulus and its relevant cues. Video modeling can counteract this problem by focusing in on the specific stimulus cues necessary to learn the target behavior (Charlop-Christy et al. 2000). Video modeling minimizes attentional requirements, allows the individual to focus on the appropriate stimuli repeatedly, and thereby facilitates successful acquisition of the target

behavior and its maintenance in memory (Corbett and Abdullah 2005).

Video modeling reduces the amount of symbolic representation necessary for observational learning to occur by decreasing the amount of language present in the video. The individual with autism is only required to listen to and comprehend minimal, if any, language (Sherer et al. 2001), which reduces or eliminates the challenge of symbolically representing language.

Furthermore, motivation may enhance the effectiveness of video modeling as a behavioral intervention because video viewing is an easy, low-demand activity, which many individuals with autism engage in at home (Shipley-Benamou et al. 2002). This method of teaching new information is less structured than many behavioral interventions and creates a more casual learning environment, which may encourage successful acquisition of new information (Charlop-Christy and Daneshvar 2003). Studies by Charlop-Christy et al. (1998) and Schreibman (1988) found that individuals with autism often repeat phrases from videos, television, or commercials; watch the same video multiple times; or persevere on a certain television show or video. These findings suggest that video viewing is intrinsically reinforcing for some individuals with autism (Charlop-Christy et al. 2000).

Video modeling has been used to target a wide range of skills in individuals with autism. A review of 19 studies on video modeling and autism by Delano (2007) found that video modeling proved effective in addressing social-communicative skills, functional skills, perspective-taking skills, and problem behavior. Haring et al. (1987) used video modeling to promote the development of purchasing skills to individuals with autism. Video modeling was used by Charlop and Milstein (1989) to promote conversational speech to children with autism. Grant and Evans (1994) showed that video modeling enhanced social initiation and appropriate toy play in children with autism. Studies by Charlop-Christy (1993, 1994) demonstrated that

play behaviors, such as independent play, cooperative play, and pretend play, improved through a video modeling intervention. Video modeling also improved the level of verbal and motor play response in children with autism (D'Ateno et al. 2003) and reciprocal play skills (Nikopoulos and Keenan 2004). Lastly, a few studies have used video modeling to reduce disruptive and challenging behaviors (Shipley-Benamou et al. 2002; Buggey 2005).

SCLT is a core foundation of cognitive behavioral therapy (CBT). Interventions using CBT for individuals with autism (e.g., Chalfant et al. 2006; Reaven et al. 2009; Sofronoff et al. 2005; Wood et al. 2009) emphasize attention, motivation, self-reflection, self-regulation, and self-efficacy. In CBT, active participation by the child to develop and practice coping skills is key. Studies have shown CBT to be an effective intervention in reducing anxiety in children with high-functioning autism and Asperger's disorder (Chalfant et al. 2006; Reaven et al. 2009; Sofronoff et al. 2005; Wood et al. 2009).

A fundamental component of CBT with individuals with autism is developing awareness and identification of emotion feelings in order to promote greater self-regulation of behavior. Self-reflection and self-regulation are often challenging for individuals with autism; therefore, these are taught to participants in CBT by practicing the identification of cues of emotion (e.g., through body feelings), which is one method of self-reflection (Chalfant et al. 2006; Reaven et al. 2009; Wood et al. 2009). In addition, the interventions underscored teaching the connections between feelings and thoughts.

Another component of SCLT utilized in CBT with individuals with autism is the promotion of self-efficacy. The work by Wood et al. (2009) targeted improving self-efficacy as a fundamental component of the CBT intervention. Wood et al. (2009) and Drahota et al. (2011) discussed the importance of improving self-care in individuals with autism in order to increase individuals' mastery experiences and ultimately improve self-efficacy. The focus on improving self-care is important because researchers have found

adaptive skill deficits in individuals with autism (Rodrigue et al. 1991). As mentioned earlier, according to SCLT, one way of improving self-efficacy is through mastery experiences. CBT interventions also promote mastery experiences in feared situations. For example, Wood et al. (2009) and Reaven et al. (2009) used a hierarchy of feared situations where participants are rewarded as they try increasingly challenging (feared) situations. The use of a hierarchy allows the participants to start with less fearful situations and work their way up to more challenging feared situations, gaining confidence with small steps as they go. The successful achievement of the feared situations in vivo in CBT allows the participant to systematically confront and succeed in anxiety-provoking situations, which systematically increases the participants' self-efficacy.

Future Directions

SCLT provides an informative framework for understanding human functioning and may have relevance to the impact of ASD symptoms on learning and adaptive behavior in individuals with autism. Video modeling and CBT are two interventions that are influenced by SCLT in treating ASD symptoms, and each has evidence of efficacy. Future research with individuals with autism may consider incorporating SCLT principles to inform interventions in order to improve skill learning and comprehension. By creating interventions that consider how deficits in autism impact symbolic representations, forethought, vicarious learning, self-reflection, self-regulation, and self-efficacy, the interventions may be able to better achieve their goals and meet the needs of affected individuals.

See Also

- [Cognitive Behavioral Therapy \(CBT\)](#)
- [Joint Attention](#)
- [Video Modeling/Video Self-modeling](#)

References and Reading

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Bandura, A. (1969). Social-learning theory of identificatory processes. In D. A. Goslin (Ed.), *Handbook of socialization theory and research* (pp. 213–262). Chicago: Rand McNally.
- Bandura, A. (1971). Analysis of modeling processes. In A. Bandura (Ed.), *Psychological modeling: Conflicting theories*. Chicago: Aldine-Atherton.
- Bandura, A. (1977). *Social learning theory* (pp. 10–21). Englewood Cliffs: Prentice Hall.
- Bandura, A. (1986). *Social foundations of thought and action: A social cognitive theory*. Englewood Cliffs: Prentice Hall.
- Bandura, A. (1989). Social cognitive theory. *Annals of Child Development*, 6, 1–60.
- Bandura, A. (1994). Self-efficacy. In L. W. Levy, K. L. Karst, & A. Winkler (Eds.), *Encyclopedia of human behavior* (Vol. 4). New York: Academic Press.
- Bandura, A. (1999). Social cognitive theory: An agentic perspective. *Asian Journal of Social Psychology*, 2, 21–41.
- Bandura, A. (2003). Observational learning. In J. H. Byrne (Ed.), *Encyclopedia of learning and memory* (2nd ed., pp. 482–484). New York: Macmillan.
- Bandura, A. (2005). The evolution of social cognitive theory. In K. G. Smith & M. A. Hitt (Eds.), *Great minds in management* (pp. 9–35). Oxford: Oxford University Press.
- Bandura, A., & Walters, R. H. (1963). *Social learning and personality development*. New York: Holt, Rinehart & Winston.
- Bandura, A., Adams, N. E., & Beyer, J. (1977). Cognitive processes mediating behavioral change. *Journal of Personality and Social Psychology*, 35, 125–139.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development*, 71, 447–456.
- Bernabei, P., Palli, F. G., Levi, G., Mazzoncini, B., & Cannoni, E. (1999). Disturbance of imagination and symbolization in pervasive developmental disorders: Preliminary study utilizing the Rorschach Inkblot test. *Perceptual and Motor Skills*, 89, 917–930.
- Brenner, L. A., Turner, K. C., & Muller, R. A. (2007). Eye movement and visual search: Are there elementary abnormalities in autism? *Journal of Autism and Developmental Disorders*, 37, 1289–1309.
- Buggey, T. (2005). Video self-modeling applications with students with autism spectrum disorder in a small private school setting. *Focus on Autism and Other Developmental Disabilities*, 20, 52–63.
- Chalfant, A., Rapee, R., & Carroll, L. (2006). Treating anxiety disorders in children with high-functioning autism spectrum disorders: A controlled trial. *Journal of Autism and Developmental Disorders*, 33, 283–298.
- Chamberlain, B., Kasari, C., & Rotheram-Fuller, E. (2006). Involvement or isolation: The social networks of children with autism in regular classroom. *Journal of Autism and Developmental Disorders*, 37, 230–242.
- Charlop, M. H., & Milstein, J. P. (1989). Teaching autistic children conversational speech using video modeling. *Journal of Applied Behavior Analysis*, 22, 275–285.
- Charlop-Christy, M. H. (1993). *Using video modeling with autistic children*. Paper presented at the Annual Conference of the Northern California Association for Behavior Analysis, Berkeley.
- Charlop-Christy, M. H. (1994). *New procedures for treating the severe behavior problems of autistic children*. Paper presented at the Annual Conference of the Association for Behavior Analysis and Therapy/Southern California, Los Angeles.
- Charlop-Christy, M. H., & Daneshvar, S. (2003). Using video modeling to teach perspective taking to children with autism. *Journal of Positive Behavior Interventions*, 5, 12–21.
- Charlop-Christy, M. H., Schreibman, L., Pierce, K., & Kurtz, P. F. (1998). Childhood autism. In R. Morris & T. R. Kratochwill (Eds.), *The practice of child therapy* (3rd ed., pp. 271–302). Boston: Allyn and Bacon.
- Charlop-Christy, M. H., Le, L., & Freeman, K. A. (2000). A comparison of video modeling with in vivo modeling for teaching children with autism. *Journal of Autism and Developmental Disorders*, 30, 537–552.
- Corbett, B. A., & Abdullah, M. (2005). Video modeling: Why does it work for children with autism? *The Journal of Early and Intensive Behavior Intervention*, 2, 2–8.
- D'Ateno, P., Mangiapanello, K., & Taylor, B. A. (2003). Using video modeling to teach complex play sequences to a preschooler with autism. *Journal of Positive Behavior Interventions*, 5, 5–11.
- Davis, T. W., & Luthans, F. A. (1980). A social learning approach to organization behavior. *Academy of Management Review*, 5, 281–290.
- Dawson, G., Meltzoff, A. N., Osterling, J., Rinaldi, J., & Brown, E. (1998). Children with autism fail to orient to naturally occurring social stimuli. *Journal of Autism and Developmental Disorders*, 28, 479–485.
- Delano, M. E. (2007). Video modeling interventions for individuals with autism. *Remedial and Special Education*, 28, 33–42.
- Drahota, A., Wood, J. J., Sze, K. M., & Van Dyke, M. (2011). Effects of cognitive behavioral therapy on daily living skills in children with high-functioning autism and concurrent anxiety disorders. *Journal of Autism and Developmental Disorders*, 41, 257–265.
- Faja, S., Aylward, E., Bernier, R., & Dawson, G. (2008). Becoming a face expert: A computerized face-training program for high-functioning individuals with autism

- spectrum disorders. *Developmental Neuropsychology*, 33, 1–24.
- Golarai, G., Grill-Spector, K., & Reiss, A. L. (2006). Autism and the development of face processing. *Clinical Neuroscience Research*, 6, 145–160.
- Grant, L., & Evans, A. (1994). *Principles of behavior analysis*. New York: Harper Collins.
- Grusec, J. E. (1992). Social learning theory and developmental psychology: The legacies of Robert Sears and Albert Bandura. *Developmental Psychology*, 28, 776–786.
- Haring, I., Kennedy, C., Adams, M., & Pitts-Conway, V. (1987). Teaching generalization of purchasing skills across community settings to autistic youth using videotape modeling. *Journal of Applied Behavior Analysis*, 20, 89–96.
- Iacoboni, M., & Dapretto, M. (2006). The mirror neuron system and the consequences of its dysfunction. *Nature Reviews Neuroscience*, 7, 942–951.
- Jarrod, C. (2003). A review of research into pretend play in autism. *Autism*, 7, 379–390.
- Klin, A., Jones, W., Schultz, R. T., Volkmar, F. R., & Cohen, D. J. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59, 809–816.
- Landry, R., & Bryson, S. (2004). Impaired disengagement of attention in young children with autism. *Journal of Child Psychology and Psychiatry*, 45, 1115–1122.
- Loth, E., Gomez, J. C., & Happe, F. (2011). Do high-functioning people with autism spectrum disorder spontaneously use event knowledge to selectively attend to and remember context-relevant aspects of science? *Journal of Autism and Developmental Disorders*, 41, 945–961.
- Malone, Y. (2002). Social cognitive theory and choice theory: A compatibility analysis. *International Journal of Reality Therapy*, 22, 10–13.
- Miller, N. E., & Dollard, J. (1941). *Social learning and imitation*. New Haven: Yale University Press.
- Money, W. H. (1996). Applying group support systems to classroom settings: A social cognitive learning theory explanation. *Journal of Management Information Systems*, 12, 65–80.
- Mundy, P., Sullivan, L., & Mastergeorge, A. (2009). A parallel and distributed processing model of joint attention, social-cognition and autism. *Autism Research*, 2, 2–21.
- Nakahachi, T., Yamashita, K., Iwase, M., Ishigami, W., Tanaka, C., Toyonaga, K., et al. (2008). Disturbed holistic processing in autism spectrum disorders verified by two cognitive tasks requiring perception of complex visual stimuli. *Psychiatry Research*, 159, 330–338.
- Nikopoulos, C. K., & Keenan, M. (2004). Effects of video modeling on social initiations by children with autism. *Journal of Applied Behavioral Analysis*, 37, 93–96.
- Peterson, C. C., Garnett, M., Kelly, A., & Attwood, T. (2009). Everyday social and conversation applications of theory-of-mind understanding by children with autism spectrum disorders or typical development. *European Child & Adolescent Psychiatry*, 18, 105–115.
- Reaven, J., Blakeley-Smith, A., Nichols, S., Dasari, M., Flanigan, E., & Hepburn, S. (2009). Cognitive-behavioral group treatment for anxiety symptoms in children with high-functioning autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 24, 27–37.
- Rodrigue, J. R., Morgan, S. B., & Geffken, G. R. (1991). A comparative evaluation of adaptive behavior in children and adolescents with autism, Down syndrome, and normal development. *Journal of Autism and Developmental Disorders*, 21, 187–198.
- Rondan, C., & Deruelle, C. (2007). Global and congruent visual processing in adults with autism and Asperger syndrome. *Research in Developmental Disabilities*, 28, 197–206.
- Schreibman, L. (1988). *Autism*. Newberry Park: Sage.
- Sherer, M., Pierce, K. L., Paredes, S., Kisacky, K. L., Ingersoll, B., & Schreibman, L. (2001). Enhancing conversational skills in children with autism via video technology. Which is better, “self” or “other” as a model? *Behavior Modification*, 25, 140–158.
- Shipley-Benamou, R., Lutzker, J. R., & Taubman, M. (2002). Teaching daily living skills to children with autism through instructional video modeling. *Journal of Positive Behavior Interventions*, 4, 165–175.
- Shumway, S., & Wetherby, A. M. (2009). Communicative acts of children with autism spectrum disorders in the second year of life. *Journal of Speech, Language, and Hearing Research*, 52, 1139–1156.
- Sofronoff, K., Attwood, T., & Hinton, S. (2005). A randomized controlled trial of a CBT intervention for anxiety in children with Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 46, 1152–1160.
- Varga, S. (2010). Pretence, social cognition and self-knowledge in autism. *Psychopathology*, 44, 46–52.
- Waller, B. (2004). Comparing psychoanalytic and cognitive-behavioral perspectives on control. *Philosophy, Psychiatry, & Psychology*, 11, 125–128.
- Whalen, C., Schreibman, L., & Ingersoll, B. (2006). The collateral effects of joint attention training on social initiations, positive affect, imitation, and spontaneous speech for young children with autism. *Journal of Autism and Developmental Disorders*, 36, 655–664.
- Williams, J. H. (2008). Self-other relations in social development and autism: Multiple roles for mirror neurons and other brain bases. *Autism Research*, 1, 73–90.
- Williams, J. H., Whiten, A., & Singh, T. (2004). A systematic review of action imitation in autistic spectrum disorder. *Journal of Autism and Developmental Disorders*, 34, 285–299.
- Wood, J. J., Drahota, A., Sze, K., Har, K., Chiu, A., & Langer, D. A. (2009). Cognitive behavioral therapy for anxiety in children with autism spectrum disorders:

A randomized, controlled trial. *Journal of Child Psychology and Psychiatry*, 50, 224–234.

Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, 23, 143–152.

Social Communication

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Synonyms

[Reciprocal communication](#)

Definition

Social communication is a broad term that describes the vast amount of verbal and non-verbal behaviors used to interact with other people. Examples of the verbal and nonverbal behaviors are (but are not limited to) speech, prosody, gestures, and facial expressions. These behaviors can be used to initiate or respond to joint attention, to share emotion with others, or to signal when an individual wants the attention of another person, along with many other uses.

A deficit in social communication is a diagnostic characteristic of children with ASD.

See Also

- [Gestures](#)
- [Pragmatics](#)
- [Prosody](#)
- [Reciprocal Communication/Interaction](#)
- [Speech](#)

References and Reading

Wetherby, A. M. (1984). Profiles of communicative and cognitive-social abilities in autistic children. *Journal of Speech, Language, and Hearing Research*, 27(3), 364.

Social Communication and Interaction (SCI)

- [DiBAS-R Validation](#)

Social Communication Disorder

- [Pragmatic Language Impairment](#)
- [Semantic Pragmatic Disorder](#)

Social Communication Questionnaire

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Synonyms

[Autism screening questionnaire \(ASQ\); SCQ](#)

Description

The Social Communication Questionnaire (SCQ), formerly known as the Autism Screening Questionnaire (ASQ), is a screening measure that was developed to identify symptomology associated with autism spectrum disorder (ASD; Rutter et al. 2003a). It is widely recognized in the field as a recommended screening measure for ASD (Coonrod and Stone 2005; Hus and Lord 2011).

The SCQ contains 40 *yes/no* items that are completed by the parent or primary caregiver. It is applicable to individuals whose chronological age is over 4 years, provided that their mental age is over 2 years. A body of research is accumulating that aims to determine the utility of the SCQ in young children, however (e.g., Corsello et al. 2003).

There are two versions of the SCQ. The Lifetime version inquires about the individual's entire developmental history, and the items in the Current version refer to the individual's behavior in the most recent 3-month period. The Lifetime form should be used for diagnostic screening purposes, and the Current form is appropriate to use when the goal is to understand an individual's current level of ASD symptomology, such as when evaluating treatment or educational plans.

Scoring of the SCQ results in a total score that is compared to a cutoff score developed from the Lifetime form. The total score is calculated by summing the item responses as indicated on the scoring form that is provided as part of the SCQ protocol. For each item, a score of 1 is given for abnormal behavior, and a score of 0 is given for its absence. All items are given equal weight in determining the total score. Different scoring procedures are used for verbal and nonverbal individuals. For verbal individuals, six items asking about language abnormalities are included in the total score, whereas these items are disregarded for individuals who are nonverbal. A cutoff score of 15 or greater identifies individuals who are likely to have an ASD, indicating that more thorough evaluations are warranted. Subscores that correspond to the three symptom domains of ASD (social abnormalities, communication abnormalities, and restricted, repetitive, and stereotyped behavior) can also be calculated. However, it must be noted that due to insufficient research on these subscores, they should be used for research purposes only (Rutter et al. 2003).

Historical Background

The SCQ was originally developed as a companion to the Autism Diagnostic Interview-Revised (ADI-R; Rutter et al. 2003b). It was designed as a

shorter version of the ADI-R, which is a 93-item structured interview. Items from the ADI-R with discriminative diagnostic validity were chosen for the SCQ. Selected items assess the three symptom domains of ASD: reciprocal social interaction, communication, and restricted, repetitive, and stereotyped patterns of behavior. Items were also selected based on the extent to which the behavior in question was readily observable by the caregiver. The wording of the items was also modified to increase the clarity of the item for parents.

Psychometric Data

Standardization data for the SCQ were collected from 200 individuals between the ages of 4–40 years (Berument et al. 1999). The sample included 160 individuals with ASD and 40 with nonspectrum disorders. Validity of the SCQ was assessed by analyzing its factor structure, the ability of SCQ items and total score to discriminate ASD and non-ASD groups, and correlating SCQ scores with those from the ADI-R. Factor analysis of the SCQ supported a four-factor structure consisting of the following factors: social interaction, communication, abnormal language, and stereotyped behavior. Item analyses indicated that 85% of the items differentiated between ASD and non-ASD individuals at a statistically significant level. Similarly, total scores on the SCQ were significantly different between ASD and non-ASD groups. Examination of total scores indicated a cutoff score of 15 for discriminating ASD from non-ASD diagnoses. A cutoff of 22 was suggested to distinguish between autism and other ASDs. Comparisons to the ADI-R revealed significant correlations between all domains of the two instruments as well as the total scores. Two additional studies support the concurrent validity of the SCQ and ADI-R. Both found good agreement between the SCQ and the ADI-R diagnostic categories but found lower agreement between the SCQ and ADI-R at the item level (Rutter et al. 2003). In sum, the standardization data supported the validity of the SCQ and suggested strong psychometric properties.

A subsequent study of the diagnostic validity of the SCQ examined its performance in a sample

of 157 children ($n = 71$ autism, $n = 49$ ASD, $n = 37$ non-ASD) (Corsello et al. 2003). Total mean scores between diagnostic groups were in the expected direction, with the autism group earning the highest scores, followed by the ASD group, and the non-ASD group had the lowest mean total scores. The authors stated that these results suggest that the total SCQ score corresponds with diagnosis and is a reasonable index of symptom severity. However, there is also evidence that the diagnostic validity of the SCQ appears to be enhanced when it is used in conjunction with observational measures such as the Autism Diagnostic Observation Schedule (ADOS; Lord et al. 1999) (Corsello et al. 2007, in Hus and Lord 2011).

Whereas the literature agrees that the SCQ is a reliable and valid screening tool for individuals 4 years and older, adaptations may need to be made when using the SCQ in children under this age. Corsello and colleagues have suggested that when using the SCQ with children under the age of 5 years, the cutoff score should be lowered in order to improve the sensitivity of the measure (Corsello et al. 2007, in Bishop et al. 2008). Another option is to use the Current version of the SCQ, which can be used with children as young as 2 years (Hus and Lord 2011).

Clinical Uses

The authors of the SCQ cite three main uses of the instrument (Rutter et al. 2003). First, it can be used as a screening instrument to detect individuals who are in need of a clinical assessment for ASD. Second, SCQ scores can be used as a measure of overall ASD symptomology between different groups, such as children with other developmental disorders. Third, scores can be used as an index of severity of ASD symptoms between groups or in one group over time, as in the case of treatment studies.

When using the SCQ as a screening instrument for ASD, it must be noted that the SCQ does not replace a diagnostic assessment. It should not be used on its own as a diagnostic tool (Rutter et al. 2003).

See Also

- Autism Diagnostic Interview-Revised
- Screening Measures

References and Reading

- Berument, S. K., Rutter, M., Lord, C., Pickles, A., & Bailey, A. (1999). Autism screening questionnaire: Diagnostic validity. *British Journal of Psychiatry*, 175, 444–451.
- Bishop, S. L., Luyster, R., Richler, J., & Lord, C. (2008). Diagnostic assessment. In K. Chawarska, A. Klin, & F. R. Volkmar (Eds.), *Autism spectrum disorders in infants and toddlers* (pp. 23–49). New York: The Guilford Press.
- Coonrod, E. E., & Stone, W. L. (2005). Screening for autism in young children. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 707–729). Hoboken, NJ: Wiley.
- Corsello, C., Leventhal, B., & Cook, L. (2003). *A screening instrument for autism spectrum disorders*. Poster session presented at the Biennial meeting of the society for research in child development, Tampa Bay.
- Corsello, C., Hus, V., Pickles, A., Risi, S., Cook, E., Leventhal, B., et al. (2007). Between a ROC and a hard place: Decision making and making decisions about using the SCQ. *Journal of Child Psychology and Psychiatry*, 48, 932–940.
- Hus, V., & Lord, C. (2011). Evaluation and testing. In E. Hollander, A. Kolevzon, & J. T. Coyle (Eds.), *Textbook of autism spectrum disorders* (pp. 49–66). Arlington: American Psychiatric Publishing.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (1999). *Autism diagnostic observation schedule – WPS* (WPSth ed.). Los Angeles: Western Psychological Services.
- Rutter, M., Bailey, A., & Lord, C. (2003a). *The social communication questionnaire*. Los Angeles: Western Psychological Services.
- Rutter, M., LeCouteur, A., & Lord, C. (2003b). *Autism diagnostic interview – Revised manual*. Los Angeles: Western Psychological Services.

Social Emotional Skills

- SSIS SEL

Social Gaze

- Mutual Gaze

Social Inclusion

- [Inclusion of Adolescents with ASD in Community Sporting Clubs](#)

Social Initiation

- [Initiation of Communication](#)

Social Interventions

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Definition

A core feature of Autism Spectrum Disorder (ASD), social disability, does not remit with development (Sigman and Ruskin 1999) and may in fact worsen during adolescence when social demands are heightened (Picci and Scherf 2015). “Social interventions” are a diverse group of therapeutic efforts intended to improve social functioning, including ability to make friends, achieve interpersonal intimacy, and navigate novel social situations. Social interventions target improved social competence in numerous ways, such as directly teaching specific prosocial skills, reducing competing or socially inappropriate behaviors, or training in social cognition believed to contribute to improved social functioning (e.g., emotion recognition, theory of mind). There are multiple clinical approaches to social interventions stemming from varied theoretical

orientations. Social interventions are also sometimes referred to as social skills training programs or relationship-based interventions.

Historical Background

Since Kanner’s (1943) original description of autism, social difficulty has been thought of as a prominent, and arguably unifying, characteristic of ASD. About three decades later, clinical scientists began to investigate the possibility of social interventions to improve these problems (Bemporad 1979; Stokes 1977). Social interventions have historically been firmly rooted in behaviorism, primarily operant conditioning in which behaviors are learned via repeated pairing with reinforcement or reward. Although often a component of treatment for people with ASD, social interventions are also often useful for individuals who struggle with other problems, such as Attention-Deficit/Hyperactivity Disorder (ADHD), depression, and shyness (e.g., de Boo and Prins 2007; Greco and Morris 2001; Segrin 2000).

Gary Mesibov (1984) extracted key elements (i.e., modeling, coaching, role playing with feedback) from established social skills training programs and applied them to develop a group social skills treatment model for verbal adolescents and adults (age 14–35 years) with ASD. Shortly thereafter, Williams (1989) implemented a similar group format for an intervention using modeling, role-playing, and playing interactive games. Ten children and adolescents (age 9–16 years) with ASD completed 4 years of a weekly 45-minute social skills training session. These early reports sparked a scientific and clinical curiosity that still persists to this day.

Lovaas (1987) was the first to describe behavioral modification treatment for young children with ASD, which is based on operant theory and entails intensive therapy across settings. This intensive instruction requires the child to actively engage with his or her social environment, while being provided with consistent reinforcement for adaptive behaviors and interactive play with peers. As a follow-up to this early study, McEachin et al. (1993) looked specifically at social functioning

and found that treatment group participants significantly improved in the area of socialization, based on parent report, relative to control group participants.

Despite these early and informative research studies, a lack of readily available social intervention programs for ASD existed well into the 1990s (Klin and Volkmar 2000). Toward the end of the twentieth century, a broader range of social skills interventions was being developed and scientifically investigated, including self-management techniques (Koegel et al. 1992) and sociodramatic play-based approaches (Thorp et al. 1995). The science was also maturing, as evidenced by more rigorous data collection efforts and recognition of the need for well-characterized samples along with manual-based programs to allow for replication.

Current Knowledge

Several published meta-analyses on social interventions for individuals with ASD (e.g., Bellini et al. 2007; Wang et al. 2013), along with the National Professional Development Center (NPDC), which is dedicated to promotion of evidence-based practices with individuals with ASD, have sought to determine the impact of social interventions for people with ASD. Reichow and Volkmar (2010), in their review, determined that social skills groups could be considered an established, evidence-based intervention. Indeed, group-based interventions are arguably the most common modality of social intervention. Although group-based interventions have been found to be modestly effective, gains do not necessarily materialize at the level of social behaviors in naturalistic settings (e.g., school) (Gates et al. 2017). In terms of differential impact of constituent aspects of intervention, it appears that video modeling (i.e., using self or other to model skill or behavior being taught via video), a technique employed in many social interventions, is one of the most effective components (e.g., Wang and Spillane 2009). Perhaps the most consistently reported finding from the applied studies on social interventions for ASD is that there

is considerable variability in terms of treatment response across participants.

At the time of this publication, there are several social intervention curricula for ASD. As an exhaustive review is beyond the scope of this entry, we will provide a brief overview of research on selected curricula. Readers who wish more in-depth or detailed information are encouraged to read information provided in the suggested selected readings. Groups that apply a cognitive-behavioral approach to train social competence are commonly used. The *Program for the Education and Enrichment of Relational Skills* (PEERS), a group-based program rooted in cognitive-behavioral therapy, was initially developed for adolescents with ASD (Laugeson et al. 2012) and has since been extended to both preschool-age children and adults (Gantman et al. 2012). Results from several randomized controlled trials (RCTs) have supported the efficacy of PEERS (Laugeson and Park 2014). The *summerMAX* program is an intensive, 5-week long, behaviorally based treatment for school-age children. It has demonstrated feasibility and efficacy in both university (Lopata et al. 2010) and community agency settings (Thomeer et al. 2016).

Theatrical, or drama-based interventions, have also been developed to improve social competence in youth with ASD. Theoretically, such interventions not only promote skill generalization (perhaps via enhanced interaction with typical peers) but also increase perspective-taking and social cognition (e.g., Corbett et al. 2016). The *Socio-Dramatic Affective-Relational Intervention* (SDARI; Lerner et al. 2011) is a brief (6-week) curriculum, associated with modest improvement in ability to detect emotions. The *Social Emotional NeuroScience Endocrinology Theatre* (SENSE) Theatre (Corbett et al. 2011) combines peer mediation (i.e., training similar-age peers to engage with, and help, the learners with ASD), behavioral skills training, and drama to improve social skills. In an RCT, participants who completed SENSE Theater showed significant improvements, relative to wait-list control participants, on several indicators of social behavior and cognition (Corbett et al. 2016).

There are several other types of social interventions that show considerable promise as being effective for some individuals with ASD. Social Stories (Gray 1998) are a straightforward approach used to explain social situations and behaviors to people with ASD. Social stories are often used to promote appropriate social skills and decrease problem behaviors. Results from several single subject and small sample studies have been quite positive (e.g., Dodd et al. 2008; Sansosti and Powell-Smith 2008). Pivotal Response Treatment (PRT) is another type of intervention, derived from the principles of applied behavioral analysis (ABA), for people with ASD (Koegel et al. 2006). Instead of targeting distinct individual behaviors, PRT focuses on pivotal areas of a child's development. The goal of PRT is to provide learning opportunities within the context of the child's natural environments.

Future Directions

The research base on social interventions has matured tremendously over the last decade. We have a better appreciation for the differential impact of various treatment components, and consumers and practitioners have considerable choice in terms of available packaged curricula. Nevertheless, there are many as-yet unanswered questions related to the clinical impact of social interventions.

Recently, research has been undertaken that will inform our understanding of predictors of treatment response. This work is critical for personalized treatment planning. Clinical science has long-appreciated the fact that response to even our best treatments is not uniform across clients. This is true also for targeting social improvement in people with ASD. By understanding both predictors and moderators of response, we can begin to tailor approaches to fit the client (cf, personalized medicine).

Unfortunately, the vast majority of the research in this area has been with preadolescent children. By comparison, research on the feasibility and efficacy of social interventions for very young children or adults, as well as adolescents to some

degree, is limited. It will be imperative that we begin to explore how to effectively intervene with older adolescents and adults especially, given the pervasiveness of social disability and its relationship to later impairment as well as co-occurring problems (e.g., anxiety). This will not be a straightforward "upward" extension given that some of the best-supported strategies (e.g., peer-video modeling and mediation) rely on availability of similar age peers and other support systems. Additionally, the nature of adult social interaction is incredibly variable across contexts, and there are fewer opportunities for face to face interaction practice with unfamiliar people after exit from secondary school.

Emerging directions for social interventions are diverse. As described by Wieckowski and White (2017), there is growing evidence for the feasibility and utility of technology-based interventions. On the whole, these interventions have been found to be acceptable to end-users (e.g., children with ASD) and target a host of measurable mechanisms underlying change in social function (e.g., facial emotion recognition). For example, improved ability to detect others' emotions may contribute to more empathic responding and prosocial behavior. Examples of such interventions include *Mind Reading: The Interactive Guide to Emotions* (Golan and Baron-Cohen 2006), the *Junior Detective Training Program* (Beaumont and Sofronoff 2008), and *Let's Face It!* (Tanaka et al. 2010). More research is needed to understand the clinical impact and generalizability of technology-based social interventions.

As the field continues to mature with respect to effective social interventions with ASD, we need to focus simultaneously on expansion as well as refinement. Regarding the former, it is critically important that novel approaches be developed (e.g., technology) as this will allow greater choice to consumers and, ultimately, aid dissemination. We do not expect even a well-established treatment to work for every client or patient. The heterogeneity in cause and presentation of ASD mandates diversity in availability of treatment approach. Regarding refinement, as we continue to deepen our understanding of the processes that underlie social impairments, ability to target key

mechanisms will be improved (Lerner et al. 2012) and, likewise, there is greater appreciation of the importance of dismantling research to identify the “critical elements” of efficacious interventions. In the future, we will see more equivalence testing of interventions, in order to evaluate relative superiority, and adaptive trials in which participants are assessed at regular intervals, in order to determine how allocation (treatment assignment) will proceed. In summary, the field has made great strides with regard to social interventions for people with ASD, and we are hopeful about the future with respect to clinicians’ ability to adopt a scientifically informed approach to determining what type of intervention works best for whom, across the lifespan, for people with ASD.

See Also

- Social Behaviors and Social Impairment
- Social Communication
- Social Skills Improvement System
- Social Skill Interventions

References and Reading

- Beaumont, R., & Sofronoff, K. (2008). A multi-component social skills intervention for children with asperger syndrome: The junior detective training program. *Journal of Child Psychology and Psychiatry*, 49(7), 743–753. <https://doi.org/10.1111/j.1469-7610.2008.01920.x>.
- Bellini, S., Peters, J. K., Benner, L., & Hopf, A. (2007). A meta-analysis of school-based social skills interventions for children with autism spectrum disorders. *Remedial and Special Education*, 28, 153–162.
- Bemporad, J. R. (1979). Adult recollections of a formerly autistic child. *Journal of Autism and Developmental Disorders*, 9, 179–197.
- de Boo, G. M., & Prins, P. J. M. (2007). Social incompetence in children with ADHD: Possible moderators and mediators in social-skills training. *Clinical Psychology Review*, 27, 78–97.
- Corbett, B. A., Gunther, J. R., Comins, D., Price, J., Ryan, N., Simon, D., . . . Rios, T. (2011). Brief report: Theatre as therapy for children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41(4), 505–511. <https://doi.org/10.1007/s10803-010-1064-1>.
- Corbett, B. A., Key, A. P., Qualls, L., Fecteau, S., Newsom, C., Coke, C., & Yoder, P. (2016). Improvement in social competence using a randomized trial of a theatre intervention for children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46, 658–672. <https://doi.org/10.1007/s10803-015-2600-9>.
- Dodd, S., Hupp, S. D. A., Jewell, J. D., & Krohn, E. (2008). Using parents and siblings during a social story intervention for two children diagnosed with PDD-NOS. *Journal of Developmental and Physical Disabilities*, 20, 217–229.
- Gantman, A., Kapp, S. K., Orenski, K., & Laugeson, E. A. (2012). Social skills training for young adults with high-functioning autism spectrum disorders: A randomized controlled pilot study. *Journal of Autism and Developmental Disorders*, 42(6), 1094–1103. <https://doi.org/10.1007/s10803-011-1350-6>.
- Gates, J., Kang, E., & Lerner, M. D. (2017). Efficacy of group social skills interventions for youth with autism spectrum disorder: A systematic review and meta-analysis. *Clinical Psychology Review*, 52, 164–181. <https://doi.org/10.1016/j.cpr.2017.01.006>.
- Golan, O., & Baron-Cohen, S. (2006). Systemizing empathy: Teaching adults with Asperger syndrome or high-functioning autism to recognize complex emotions using interactive multimedia. *Development and Psychopathology*, 18(02), 591–617.
- Gray, C. A. (1998). Social stories and comic strip conversations with students with asperger syndrome and high-functioning autism. In E. Schopler, G. B. Mesibov, & L. J. Kunce (Eds.), *Asperger syndrome or high-functioning autism?* (pp. 167–198). New York: Plenum.
- Greco, L. A., & Morris, T. L. (2001). Treating childhood shyness and related behavior: Empirically evaluated approaches to promote positive social interactions. *Clinical Child and Family Psychology Review*, 4, 299–318.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Klin, A., & Volkmar, F. R. (2000). Treatment and intervention guidelines for individuals with Asperger syndrome. In A. Klin, F. R. Volkmar, & S. S. Sparrow (Eds.), *Asperger syndrome* (pp. 340–366). New York: Guilford Press.
- Koegel, L. L., Koegel, R. L., Hurley, C., & Frea, W. D. (1992). Improving social skills and disruptive behavior in children with autism through self-management. *Journal of Applied Behavior Analysis*, 25(2), 341–353. <https://doi.org/10.1901/jaba.1992.25.341>.
- Koegel, R. L., Openden, D., Fredeen, R., & Koegel, L. K. (2006). The basics of pivotal response treatment. In R. L. Koegel & L. K. Koegel (Eds.), *Pivotal response treatments for autism* (pp. 3–30). Baltimore: Paul H. Brookes Publishing.
- Laugeson, E. A., & Park, M. N. (2014). Using a CBT approach to teach social skills to adolescents with autism spectrum disorder and other social challenges: The PEERS method. *Journal of Rational-Emotive Cognitive-Behavior Therapy*, 32, 84–97. <https://doi.org/10.1007/s10942-014-0181-8>.

- Laugeson, E. A., Frankel, F., Gantman, A., Dillon, A. R., & Mogil, C. (2012). Evidence-based social skills training for adolescents with autism spectrum disorders: The UCLA PEERS program. *Journal of Autism and Developmental Disorders*, 42, 1025–1036. <https://doi.org/10.1007/s10803-011-1339-1>.
- Lerner, M. D., Mikami, A. Y., & Levine, K. (2011). Socio-dramatic affective-relational intervention for adolescents with Asperger syndrome and high functioning autism: Pilot study. *Autism*, 15(1), 21–42. <https://doi.org/10.1177/1362361309353613>.
- Lerner, M. D., White, S. W., & McPartland, J. C. (2012). Mechanisms of change in psychosocial interventions for autism spectrum disorders. *Dialogues in Clinical Neuroscience*, 14(3), 307–318.
- Lopata, C., Thomeer, M. L., Volker, M. A., Toomey, J. A., Nida, R. E., Lee, G. K., et al. (2010). RCT of a manualized social treatment for high-functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40, 1297–1310.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- McEachin, J. J., Smith, T., & Lovaas, O. I. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. *American Journal on Mental Retardation*, 97, 359–372.
- Mesibov, G. B. (1984). Social skills training with verbal autistic adolescents and adults: A program model. *Journal of Autism and Developmental Disorders*, 14, 395–404.
- Picci, G., & Scherf, K. S. (2015). A two-hit model of autism: Adolescence as the second hit. *Clinical Psychological Science: A Journal of the Association for Psychological Science*, 3(3), 349–371. <https://doi.org/10.1177/2167702614540646>.
- Reichow, B., & Volkmar, F. R. (2010). Social skills interventions for individuals with autism: Evaluation for evidence-based practices within a best evidence synthesis framework. *Journal of Autism and Developmental Disorders*, 40, 149–166.
- Sansosti, F. J., & Powell-Smith, K. A. (2008). Using computer-presented social stories and video models to increase the social communication skills of children with high-functioning autism spectrum disorders. *Journal of Positive Behavior Interventions*, 10, 162–178.
- Segrin, C. (2000). Social skills deficits associated with depression. *Clinical Psychology Review*, 20, 379–403.
- Sigman, M., & Ruskin, E. (1999). *Continuity and change in the social competence of children with autism, down syndrome, and developmental delays*. With commentary by Carolyn B. Mervis and Byron F. Robinson (Monographs of the Society for Research in Child Development, Vol. 61, no. 1, p. 256). Malden: Blackwell.
- Stokes, K. S. (1977). Planning for the future of a severely handicapped autistic child. *Journal of Autism and Childhood Schizophrenia*, 7, 288–298.
- Tanaka, J. W., Wolf, J. M., Klaiman, C., Koenig, K., Cockburn, J., Herlihy, L., ... & Schultz, R. T. (2010). Using computerized games to teach face recognition skills to children with autism spectrum disorder: the Let's Face It! program. *Journal of Child Psychology and Psychiatry*, 51(8), 944–952.
- Thomeer, M. L., Lopata, C., Donnelly, J. P., Booth, A., Shanahan, A., Federiconi, V., ... Rodgers, J. D. (2016). Community effectiveness RCT of a comprehensive psychosocial treatment for high-functioning children with ASD. *Journal of Clinical Child & Adolescent Psychology*. Advance online publication. <https://doi.org/10.1080/15374416.2016.7359>.
- Thorp, D. M., Stahmer, A. C., & Schreibman, L. (1995). Effects of sociodramatic play training on children with autism. *Journal of Autism and Developmental Disorders*, 25(3), 265–282.
- Wang, P., & Spillane, A. (2009). Evidence-based social skills interventions for children with autism: A meta-analysis. *Education and Training in Developmental Disabilities*, 44, 318–342.
- Wang, S., Parrila, R., & Cui, Y. (2013). Meta-analysis of social skills interventions of single-case research for individuals with autism spectrum disorders: Results from three-level HLM. *Journal of Autism and Developmental Disorders*, 43(7), 1701–1716. <https://doi.org/10.1007/s10803-012-1726-2>.
- Wieckowski, A. T., & White, S. W. (2017). Emerging social skills interventions for individuals with autism. In J. Leaf (Ed.), *Handbook of social skills and autism Spectrum disorder – Assessment, curricula, and intervention*. New York: Springer.
- Williams, T. I. (1989). A social skills group for autistic children. *Journal of Autism and Developmental Disorders*, 19, 143–155.

Social Language Development Test

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Synonyms

SLDT-A; SLDT-E; Social language development test-adolescent; Social language development test-elementary

Description

The tests of social language development—elementary and adolescent (Bowers et al. 2008, 2010) are diagnostic tests of social language skills. They are designed to determine the role language development plays in the acquisition of social understanding for students from the ages of 6.0–17.11 years from the following educational settings: regular education (no active IEP) and special education (active IEP). Students with a diagnosis of autism and delayed language development were included in the item pool and standardization studies.

The testing materials are as follows:

The social language development test—elementary (SLDT-E) includes an examiner's manual, the scoring standards and example responses book, a picture stimuli book, and test forms. Ages include 6.0–11.11 years.

The SLDT-E is comprised of four subtests designed to differentiate how students with language impairments and autism differ from their typically developing peers in social cognitive processing, in identifying feelings of participants in a conflict, in identifying and evaluating strategies to overcome obstacles, and in knowing when a conflict is resolved. The subtests are the following: (a) making inferences, (b) interpersonal negotiation, (c) multiple interpretations, and (d) supporting peers. Each subtest has 12 items.

Many children with language impairments (LI) exhibit poor social interaction with others, and these social differences may appear as early as preschool and continue or intensify as these children mature (Brinton et al. 2004; Cohen et al. 1998; Craig 1993; Fujiki et al. 1997; Hadley and Rice 1991). A tool that would pinpoint areas of strengths and weaknesses would guide speech-language pathologists (SLPs) in determining appropriate intervention goals and objectives. Tasks in this test focus on taking someone else's perspective, making correct inferences, negotiating conflicts with peers, being flexible in interpreting situations, and supporting friends diplomatically.

The social language development test—adolescent (SLDT-A) includes an examiner's manual, the scoring standards and example responses book, a picture stimuli book, test forms, and an audio CD used with subtest E: interpreting ironic statements. Ages include 12.0–17.11 years.

The SLDT-A is comprised of five subtests designed to differentiate typically developing elementary children from typically developing adolescents, typically developing adolescents from language-impaired adolescents, and language-impaired adolescents from adolescents identified with high-functioning autism. The subtests are the following: (a) making inferences, (b) interpreting social language, (c) problem solving (stating and justifying solutions), (d) social interaction, and (e) interpreting ironic statements. Each subtest has 12 items each. Three of the subtests have two questions per item.

Language is the skill that is central to engaging in all aspects of peer relationships. Students with limited language skills are susceptible to misinterpretations and have more difficulty participating in the highly verbal, rapidly delivered social processes used in peer group formation (Gallagher 1993). They often have difficulty maintaining friendships, negotiating conflict, and getting along with others. Tasks in this test focus on taking someone else's perspective, making correct inferences, solving problems with peers, interpreting social language, and understanding idioms, irony, and sarcasm.

These tests do not address all aspects of social language or pragmatic skills. Rather, they focus on social interpretation and interaction with peers and/or friends. For both tests, the items are not arranged in order of difficulty because, as of yet, no hierarchy is established for these tasks. For this reason, there are no basals or ceilings; each subtest is administered in its entirety to every student.

The tests begin with the first item in subtest A. Explicit directions for how to administer the items are provided. Demonstration items are provided for each subtest. These items may be altered or explained to show the student how to respond.

Following the demonstration item, each subtest begins with item 1 regardless of the student's age. Each item is presented verbally. The only exception is subtest E: interpreting ironic statements on the SLDT-A. An audio CD is used to administer this subtest.

Allowable prompts, used only as worded, are listed on the test forms. These are used if the student's response is unclear. These prompts are not used to give the student "a second chance" after a clear, complete, but incorrect response.

More than one testing session is allowable if the break between sessions occurs upon completion of a subtest and all of its questions.

The test examiner must be a trained professional familiar with language disorders (e.g., speech-language pathologist, psychologist) as they require careful interpretation of responses by the examiner.

Subtests A in both tests tap the student's ability to infer what someone in a photo is thinking. The student also states the visual clues that facilitated making the inference. Skills assessed in these subtests include the ability to:

- Detect nonverbal and context clues in a picture of a person or people
- Assume the perspective of a specific person in a picture
- Infer what the person is thinking
- Express the person's thought as a relevant, direct quotation
- State the visual clue that suggests what the person is thinking

A score of 1 or 0 is assigned to each response based on the relevancy and quality of the response. For the SLDT-A, the student must provide an appropriate response for both questions to earn a score of 1.

SLDT-E Subtest B: Interpersonal Negotiation asks the student to imagine being involved in a given conflict with a friend. The student states the problem (task a), proposes an appropriate solution (task b), and explains why that would be a good solution (task c). No pictures or photos are used for this subtest. Skills assessed in this subtest include the ability to:

- Understand a short passage about a conflict with a friend
- Infer the perspective of each person in the conflict
- State the problem clearly
- Propose a solution
- Explain why that would be a good solution

A score of 3, 2, 1, or 0 is assigned to each response based on relevancy and quality.

SLDT-A Subtest B: Interpreting Social Language asks the student questions about how people communicate. The student is asked to give an example and an appropriate context in which the type of communication would be used. Skills assessed include the ability to:

- Demonstrate actions
- Tell an appropriate reason or use for an action
- Think/talk about language
- Interpret figurative language including idioms

A score of 1 or 0 is assigned to each response based on relevancy and quality.

SLDT-E Subtest C: Multiple Interpretations asks the student to provide two distinctively different, plausible interpretations of the same photo. Skills assessed in this subtest include the ability to:

- Logically interpret a situation in a photo in two different ways

A score of 1 or 0 is assigned to each response, based on relevancy and quality.

SLDT-A Subtest C: Problem Solving (Stating and Justifying Solutions) asks the student to solve a problem by stating and justifying a logical solution. The student must give both a solution and a justification. Skills assessed include the ability to:

- State an appropriate solution to a problem in a situation with a peer
- Justify the solution
- Negotiate conflicts with peers
- Take the perspective of the other person in the situation
- State the conflict from a mutual perspective

A score of 1 or 0 is assigned to each response based on relevancy and quality.

SLDT-E Subtest D: Supporting Peers asks the student to take the perspective of someone involved in a situation with a friend. The student tells what to say in reaction to the friend's situation. Responses receive credit based on the degree of support they offer to the friend, not on the truthfulness.

Being truthful is a basic maxim of interpersonal communication (Grice 1980), yet speakers are also expected to help, not hurt, their communicative partners (Lakoff 1973; Sweetser 1987). Telling white lies successfully requires reconciling these apparently contradictory communication rules and knowing when and how to adapt them to suit various social situations (Talwar et al. 2007).

A score of 4, 3, 2, 1, or 0 is assigned to each response based on relevancy and quality of support for a friend.

SLDT-A Subtest D: Social Interaction asks the student to listen to situations and answer questions about them. In some situations, an appropriate response may include a dishonest or rude remark. Skills assessed include the ability to:

- Listen to short passages
- Understand social interactions with peers
- Provide an appropriate, supportive response
- Ignore the situation (when doing nothing is the best option)

A score of 1 or 0 is assigned to each response based on the relevancy and quality of support for the situation.

SLDT-A Subtest E: Interpreting Ironic Statements asks the student to listen to some situations on a CD. The narrator reads the situations and asks what someone means at the end of each one. Research on intonation features associated with irony is inconsistent, and ironic speech does not necessarily include "signature acoustic features" (Bryant and Fox Tree 2005; Nakassis and Snedeker 2002). For this reason, the assumed features of irony (e.g., exaggerated pitch) were "toned down" so that the student must rely on context discrepancies and other context clues to

determine the ironic meaning of a statement. Our research proved that including exaggerated intonations made the task too easy for the age range of this test. Skills assessed included the ability to:

- Understand common idioms used in everyday language
- Reject the literal meaning of the statement
- Understand the speaker's belief
- Judge the speaker's attitude
- Recognize sarcasm and interpret irony

A score of 1 or 0 is assigned to each response based on the relevancy and quality.

Historical Background

In 1983, we published a test of pragmatics called the interpersonal language skills assessment (ILSA) by speech-language pathologists, Carolyn Blagden (now LoGiudice) and Nancy McConnell. This test of pragmatic behaviors looked at interaction of 8–13-year-olds while playing a board game. The authors wanted to determine if there was age progression and reverse age progression on such behaviors as advising, commanding, accusing, deprecating, justifying, and supporting. They found that in general, as students got older, their outright negative comments decreased while their sarcasm increased. They also found out that comments with grammar or semantic errors decreased with age. Although some speech pathologists were curious about the skills assessed in the ILSA, there was a general lack of interest from the field. By 1989, this test was out of print.

Between 1990 and 2008, we continued to scour the research on pragmatic/social language skills. Our hypothesis was that children develop social "governors" as they mature. Over time, research articles about peer support, negotiating, making inferences, and interpreting facial expression and body language appeared in the literature. We also read research about the development of these skills in children on the autism spectrum and on children who had language disorders but who were not classified as autistic.

Despite a growing body of research, as of yet, there is no well-recognized, research-based developmental model for children learning social interaction skills. That may be because the following:

- Adults seem to have mastered social interaction without any conscious effort or formal instruction.
- Much of social language is abstract and covert and cannot be observed directly. We can only assume what someone else is thinking based on nonverbal cues, background knowledge, and the person's words and actions.

We do know that children with language impairments exhibit poor social interaction with others. We decided that if speech pathologists had an effective, standardized tool to assess the social interaction skills of students that would also pinpoint areas of strengths and weaknesses, it would guide the SLP in qualifying students for therapy and help in determining appropriate intervention goals and objectives.

We began writing and rewriting items and testing and retesting children. Our item pool results showed us the need for a scoring system that was sensitive to subtle changes in verbal behavior. The standardization studies proved the scoring standards were correct but that further refining was necessary. These tests represent the important data we gathered and we believe are a good start toward identifying children with social language differences.

Psychometric Data

Item pool and standardization studies were conducted for both tests. The items and subtests for both tests were subjected to rigorous statistical analyses in these studies.

The item pool and standardization studies for each test included subjects from regular education, special education, all socioeconomic levels, and from various racial groups, including White, Black, Hispanic or Latino, and other mixed racial group. Subjects with IEPs for special services but who attended regular education classes were also

included. Subjects who did not use English proficiently at school, were nonverbal, had any degree of hearing loss, or resided outside the United States were excluded from the standardization studies for each test. Subjects were from all geographic regions of the United States. The sample population reflected the national school population demographics from the 2004 national census for race, gender, age, and educational placement for the item pool and standardization studies.

For both tests, the item pool items were administered to random samples of subjects at yearly age intervals. Subjects included in the item pool studies were not included in the standardization studies.

A team of expert speech-language pathologists determined credit levels for item pool responses during a response analysis session. Items that did not show statistical age progression, showed bias, or were not deemed fair for an ethnic group were eliminated. Item difficulty indexes and item discrimination indexes were computed for each item at each of the yearly age levels. Items retained for the final versions of the test met two empirical criteria:

1. Demonstrate age progression in terms of increasing percents of subjects passing at successive age levels
2. Demonstrate significant discrimination between high and low scorers on the subtest at each age level

The test examiners were speech-language pathologists who held a master's degree or higher from an accredited university.

SLDT-E

Item Pool

1. Four subtests, 68 items.
2. $N = 390$ subjects ages 6 years, 0 months to 11 years, 11 months.
3. Data was gathered at yearly age intervals.

Standardization

1. Four subtests, 48 items.
2. $N = 1104$ subjects ages 6 years, 0 months to 11 years, 11 months.

3. Data was gathered at half-year age intervals. The item pool analysis showed that significant changes in social language development could be shown by grouping subjects in this manner.
4. Test examiners were required to make value judgments regarding the appropriateness of responses and to score them as indicated on the scoring standards provided to them.
5. The final version of the test has four subtests, 12 items per subtest for a total of 48 items.

SLDT-A

Item Pool

1. Five subtests, 89 items.
2. $N = 500$ subjects ages 12 years, 0 months, to 17 years, 11 months.
3. Data was gathered at yearly age intervals.

Standardization

1. Five subtests, 69 items.
2. $N = 834$ subjects ages 12 years, 0 months, to 17 years, 11 months.
3. Data was gathered at half-year age intervals. The item pool analysis showed that significant changes in social language development could be shown by grouping subjects in this manner.
4. Test examiners were required to make value judgments regarding the appropriateness of responses and to score them as indicated on the scoring standards provided to them.
5. The final version of the test has five subsets, 12 items per subtest for a total of a 60 items.

Mean and median raw score values and standard deviations for each subtest and the total test score were computed for both tests. There were no significant gender differences which supported the use of combined male-female norms for the elementary and adolescent tests.

For reporting purposes, three types of scores are used: age equivalents, percentile ranks, and standard scores.

Reliability was established by both the use of test-retest and internal consistency methods. Table 1 represents the internal consistency results

for the SLDT-E, and Table 2 represents the internal consistency results for the SLDT-A.

Empirical validity for each test was established with contrasted groups. Validity was established by comparing the test performances of randomly selected subjects from the sample population with a matched sample of subjects with language disorders and autism spectrum disorders receiving special services. The SLDT-E and the SLDT-A significantly discriminate between these clinical groups at most age levels. The exception is on the SLDT-E for ages 6–0 through 6–5. In this age group, the normative sample and the language-disordered group did not show a significant difference for the total test score.

Construct validity was established with point biserial correlations. Inspection of these correlations revealed highly satisfactory levels of item consistency for both tests (88% for the SLDT-E and 97% for the SLDT-A). The subtest intercorrelations and the correlations of individual subtests with the total test suggest that the subtests do assess separate social language functions but also measure a common general dimension. Overall, internal consistency estimates were satisfactory.

Race/socioeconomic group differences were conducted at the subtest level for both tests. Random samples of White, Black, and Hispanic or Latino student performances at the item level were compared by analyzing the proportion of students passing each item and at the subtest level by:

- Chi-square analyses to determine if significant relations existed between race and test performance
- Two-factor analysis of variance (ANOVA) analyses

In the SLDT-E, there were race differences in only 11% of the total number of the possible statistical tests for race and socioeconomic status (SES) group differences in only 18% of the statistical tests for that factor. In the SLDT-A, there were race and SES group differences in only 5% of the total number of the possible statistical tests for race and SES differences in only 15% of the statistical tests for that factor.

Social Language Development Test, Table 1 Test-retest reliability coefficients and standard errors of measurement for each task, each subtest, and total test by age

Chronological age	N	Making inferences						Interpersonal negotiation						Multiple interpretations		Supporting peers		Total test			
		Task a			Task b			Task a			Task b									Task c	
		Making inferences total			Task a			Task b			Task a			Task b			Task c		Interpersonal negotiation total		
		r	SEM	r	SEM	r	SEM	r	SEM	r	SEM	r	SEM	r	SEM	r	SEM	r	SEM	r	SEM
6-0 ... 6-5	9	.94	0.79	.46	2.35	.87	1.98	.64	6.12	.25	6.30	.40	6.82	.59	15.21	.70	1.73	.66	5.86	.59	23.11
6-6 ... 6-11	3	.50	2.46	1.00	0.00	.99	0.66	-.40	12.24	1.00	0.26	.99	0.82	1.00	1.16	.98	0.43	.99	0.95	1.00	1.44
7-0 ... 7-5	3	.97	0.52	.94	0.71	.98	0.81	-.20	7.76	.80	9.05	1.00	0.40	.94	4.74	.93	0.76	.97	1.87	.98	4.81
7-6 ... 7-11	11	.75	1.63	.42	2.22	.67	3.02	.87	2.50	.70	2.95	.72	3.59	.81	7.19	.59	1.90	.81	4.02	.91	8.64
8-0 ... 8-5	12	.91	1.09	.86	1.16	.91	1.70	.80	2.78	.91	1.74	.84	2.65	.87	5.89	.53	1.91	.86	3.19	.88	9.75
8-6 ... 8-11	8	.14	3.08	.53	1.85	.58	3.44	.66	4.11	.82	2.93	.86	2.59	.92	5.06	.59	1.84	.65	5.40	.76	14.54
9-0 ... 9-5	7	.83	1.22	.74	1.01	.87	1.58	.57	4.28	.72	2.89	.94	1.43	.80	6.55	.75	1.41	.92	2.41	.90	7.69
9-6 ... 9-11	8	.67	1.67	.77	0.97	.83	1.75	.05	4.86	.63	2.98	.69	3.10	.56	8.48	.78	1.30	.86	2.95	.66	12.64
10-0 ... 10-5	6	.44	2.08	.82	1.02	.72	2.53	.90	1.94	.28	4.87	.05	5.92	.12	14.89	.67	1.59	.53	5.06	.53	17.21
10-6 ... 10-11	9	.36	2.47	.69	1.03	.48	3.03	.48	2.69	.80	2.07	.28	4.54	.57	6.88	.86	0.99	.58	4.30	.58	11.34
11-0 ... 11-5	10	.72	1.82	-.30	2.99	.58	3.53	.39	5.56	.68	3.81	.53	4.98	.84	7.77	.88	1.04	.89	2.77	.78	15.27
11-6 ... 11-11	10	.35	2.35	.88	0.70	.70	2.32	.69	2.53	.69	3.20	.90	1.94	.94	3.35	.57	1.67	.30	6.75	.87	8.71
Median		.63	1.77	.65	1.33	.77	2.20	.45	4.78	.56	3.59	.68	3.23	.75	7.26	.74	1.38	.75	3.79	.79	11.26

r reliability coefficient, SEM standard error of measurement

Social Language Development Test, Table 2 Reliability based on item homogeneity: Kuder-Richardson (KR20) coefficients for each task, each subtest, and total test by age

Chronological age	Making inferences			Interpersonal negotiation subtest					Multiple interpretations	Supporting peers	Total test
	Task a	Task b	Making inferences total	Task a	Task b	Task c	Interpersonal negotiation total				
6-0 ... 6-5	.75	.73	.84	.84	.76	.83	.93	.74	.77	.95	
6-6 ... 6-11	.78	.76	.86	.86	.77	.83	.93	.71	.77	.96	
7-0 ... 7-5	.70	.74	.82	.75	.71	.78	.89	.68	.76	.94	
7-6 ... 7-11	.75	.73	.82	.81	.67	.75	.90	.70	.74	.93	
8-0 ... 8-5	.78	.77	.86	.80	.66	.75	.88	.65	.66	.93	
8-6 ... 8-11	.76	.74	.84	.83	.73	.78	.91	.68	.77	.95	
9-0 ... 9-5	.71	.62	.80	.83	.52	.75	.87	.68	.77	.93	
9-6 ... 9-11	.70	.63	.79	.76	.56	.68	.85	.67	.73	.92	
10-0 ... 10-5	.69	.74	.83	.78	.70	.76	.90	.67	.62	.93	
10-6 ... 10-11	.76	.60	.80	.38	.46	.69	.77	.65	.57	.87	
11-0 ... 11-5	.80	.78	.88	.89	.78	.82	.94	.74	.83	.97	
11-6 ... 11-11	.73	.67	.80	.45	.44	.72	.75	.65	.65	.89	
Median KR20	.74	.71	.83	.75	.65	.76	.88	.68	.72	.93	

For both tests, the differences were usually small with most of them in the magnitude of one or two raw scores. Collectively, the results indicate that neither race nor SES group has a major impact on possible race or SES group bias for either the elementary or adolescent test. Further study is warranted however.

Clinical Uses

The results obtained from diagnostic testing should help the examiner to:

1. Identify the student's strengths and weaknesses
2. Make recommendations for additional testing
3. Make well-founded, educationally significant recommendations for remediation
4. Help teachers, parents, guardians, and the student understand the nature of the student's social language functioning
5. Make well-founded recommendations for boosting social success in school and at home

A student's performance on either of these tests may relate to his academic performance and peer interaction. A student may perform poorly on these tests because he has below average vocabulary skills, difficulty recognizing and interpreting facial expressions and body language, does not know the steps involved in processing a photo of someone, etc.

Performance on the individual subtests and total test performance, along with additional diagnostic tests and parent/teacher report, will give the SLP a framework for developing a remediation program that addresses the student's social language weaknesses and builds on the student's strengths.

For each subtest on the SLDT-E and SLDT-A, the authors identified error patterns and, based on those, identified remediation strategies for the SLP. These are described in the examiner's manuals.

As previously stated, these tests do not address all aspects of social language or pragmatic skills. They focus on social interpretation and interaction with peers and/or friends and are the first tests to

have normative data on which to base therapeutic decisions. The authors encourage further research into social language skill development.

See Also

- [Asperger Syndrome](#)
- [High-Functioning Autism \(HFA\)](#)
- [Norm-Referenced Testing](#)

References and Reading

- Agaliotis, I., & Goudiras, D. (2004). A profile of interpersonal conflict resolution of children with learning disabilities. *Learning Disabilities: A Contemporary Journal*, 2, 15–29.
- American Psychological Association (APA). (1974). *Standards for educational and psychological tests*.
- American Speech-Language-Hearing Association. (2004). *Preferred practice patterns for the profession of speech-language pathology*.
- American Speech-Language-Hearing Association. (2006). *Guidelines for speech-language pathologists in diagnosis, assessment, and treatment of autism spectrum disorders across the life span*.
- Baron-Cohen, S. (1995). *Mind blindness: An essay on autism and theory of mind*. Cambridge, MA: MIT Press/Bradford Books.
- Baron-Cohen, S., Allen, J., & Gillberg, C. (1992). Can autism be detected at 18 months? The needle, the haystack, and the CHAT. *The British Journal of Psychiatry*, 161, 839–843.
- Baron-Cohen, S., Cox, A., Baird, G., Swettenham, J., Nightingale, N., Morgan, K., et al. (1996). Psychological markers of autism at 18 months of age in a large population. *The British Journal of Psychiatry*, 168, 158–163.
- Bauminger, N. (2002). The facilitation of social-emotional understanding and social interaction in high-functioning children with autism: Intervention outcomes. *Journal of Autism and Developmental Disorders*, 32(4), 283–298.
- Bauminger, N., Schorr Edelsztein, H., & Morash, J. (2005). Social information processing and emotional understanding in children with LD. *Journal of Learning Disabilities*, 38(1), 45–61.
- Bavelas, J. V., & Chovil, N. (2000). Visible acts of meaning: An integrated message model of language in face-to-face dialogue. *Journal of Personality and Social Psychology*, 19, 163–194.
- Bishop, D. V. M., & Adams, C. (1989). Conversational characteristics of children with semantic-pragmatic disorder. *British Journal of Disorders of Communication*, 24, 241–263.

- Bishop, D. V. M., & Adams, C. (1992). Comprehension problems in children with specific language impairment: Literal and inferential meaning. *Journal of Speech and Hearing Research*, 35, 119–129.
- Black, K. A. (2000). Gender differences in adolescents' behavior during conflict resolution tasks with best friends. *Adolescence*, 35(139), 499–512.
- Blakemore, S. (2008). The social brain in adolescence. *Neuroscience*, 9, 267–277.
- Bliss, L. S. (1992). A comparison of tactful messages by children with and without language impairment. *Language, Speech, and Hearing Services in Schools*, 23, 343–347.
- Bok, S. (1978). *Lying: Moral choice in public and private life*. London: Quartet Books.
- Bowers, L., Huisinigh, R., & LoGuidice, C. (2007). *Tasks of problem solving adolescent*. East Moline: LinguSystems.
- Bowers, L., Huisinigh, R., & LoGuidice, C. (2008). *Social language development test elementary*. East Moline: LinguSystems.
- Brady, C. (2008). *Tease-proof your preteen with ADHD: Practicing social skills at home will make school a much friendlier place for your child with ADHD*. Retrieved October 13, 2009, from <http://www.additudemag.com>
- Bremer, C. D., & Smith, J. (2004). Teaching social skills. *Addressing Trends and Developments in Secondary Education and Transition*, 3(5). Available online at www.ncset.org
- Bretherton, I., & Beeghly, M. (1982). Talking about internal states: The acquisition of an explicit theory of mind. *Developmental Psychology*, 18, 906–921.
- Bretherton, I., McNew, S., & Beeghly-Smith, M. (1981). Early person knowledge as expressed in gestural and verbal communication: When do infants acquire a “theory of mind”? In M. E. Lamb & L. R. Sherrod (Eds.), *Infant social cognition: Empirical and theoretical considerations* (pp. 333–373). Hillsdale: Lawrence Erlbaum Associates.
- Brinton, B., & Fujiki, M. (2002). Social development in children with specific language development and profound hearing loss. In P. K. Smith & C. H. Hart (Eds.), *Blackwell handbook of childhood social development* (pp. 588–603). Malden: Blackwell.
- Brinton, B., Fujiki, M., & McKee, L. (1998). Negotiation skills of children with specific language impairment. *Journal of Speech, Language, and Hearing Research*, 41, 927–940.
- Brinton, B., Robinson, L. A., & Fujiki, M. (2004). Description of a program for social language intervention: “If you can have a conversation, you can have a relationship”. *Language, Speech, and Hearing Services in Schools*, 35, 283–290.
- Brinton, B., Spackman, M. P., Fujiki, M., & Ricks, J. (2007). What should Chris say? The ability of children with specific language impairment to recognize the need to dissemble emotions in social situations. *Journal of Speech, Language, and Hearing Research*, 50, 798–811.
- Brown, D. S. (2000). Social skills: Finding friends and persuading people, Chapter 5. In D. S. Brown (Ed.), *Learning a living, a guide to planning your career and finding a job for people with learning disabilities, attention deficit disorder and dyslexia*. Bethesda: Woodbine House.
- Bryan, T. H. (1977). Learning disabled children's comprehension of nonverbal communication. *Journal of Non-verbal Communication*, 10, 36–41.
- Bryant, G. A., & Fox Tree, J. E. (2005). Is there an ironic tone of voice? *Language and Speech*, 48(3), 257–277.
- Burgess, S., & Turkstra, L. S. (2006). Social skills intervention for adolescents with autism spectrum disorders: A review of the experimental evidence. *EBP Briefs*, 1(4), 1–21.
- Carpendale, J. I. M., & Lewis, C. (2004). Construction and understanding of mind: The development of children's social understanding within social interaction. *The Behavioral and Brain Sciences*, 27, 79–151.
- Carpendale, J., & Lewis, C. (2006). *How children develop social understanding*. Malden: Blackwell.
- Carroll, L. (2007). Psst! Gossip may be good for you. MSNBC.com, Mental Health. Retrieved July 25, 2007, from <http://www.msnbc.msn.com/id/199748142/>
- Cohen, N. J., Menna, R., Vallance, D. D., Barwick, M. A., Im, N., & Horodezky, N. B. (1998). Language, social cognitive processing, and behavioral characteristics of psychiatrically disturbed children with previously identified and unsuspected language impairments. *Journal of Child Psychology and Psychiatry*, 39, 853–864.
- Cooper, D., & Anderson-Inman, L. (1988). Language and socialization. In M. Nippold (Ed.), *Later language development: Ages nine through nineteen* (pp. 225–245). Boston: Little, Brown.
- Craig, H. (1993). Social skills of children with specific language impairment: Peer relationships. *Language, Speech, and Hearing Services in Schools*, 24, 206–215.
- Craig, H. K., & Gallagher, T. M. (1986). Interactive play: The frequency of related verbal responses. *Journal of Speech, Language, and Hearing Research*, 29, 375–383.
- Crais, E. R., & Chapman, R. S. (1987). Story recall and inferencing skills in language/learning-disabled and nondisabled children. *The Journal of Speech and Hearing Disorders*, 52, 50–55.
- Crick, N. R., & Dodge, K. A. (1994). A review and reformulation of social information processing mechanisms in children's social adjustment. *Psychological Bulletin*, 115(1), 74–101.
- Crone, E. A., Bullens, L., Van Der Plas, E. A. A., Kijkuit, E. J., & Zelazo, P. D. (2008). Developmental changes and individual differences in risk and perspective taking in adolescence. *Development and Psychopathology*, 20, 1213–1229.
- Crooke, P. J., Hendrix, R. E., & Rachman, J. Y. (2007). *Teaching social thinking to children with ASD: An effectiveness study*. Presentation at ASHA, November, 2007, Boston.

- Crooke, P. J., Hendrix, R. E., & Rachman, J. Y. (2008). Brief report: Measuring the effectiveness of teaching social thinking to children with Asperger syndrome (AS) and high functioning autism (HFA). *Journal of Autism and Developmental Disorders*, 38, 581–591.
- Davis, P. N. (2006). Social skills intervention for children diagnosed with autism spectrum disorders. Seminar presented at the ASHA Convention, November.
- Decety, J., Michalska, K. J., & Akitsuki, Y. (2008). Who caused the pain? An fMRI investigation of empathy and intentionality. *Neuropsychologia*, 46, 2607–2614.
- Denham, S. A. (1986). Social cognition, prosocial behavior, and emotion in preschoolers: Contextual validation. *Child Development*, 57, 194–201.
- DePaulo, B. M., & Bell, K. L. (1996). Truth and investment: Lies are told to those who care. *Journal of Personality and Social Psychology*, 71, 703–716.
- DePaulo, B. M., & Kashy, D. A. (1998). Everyday lies in close and casual relationships. *Journal of Personality and Social Psychology*, 74, 63–79.
- Dews, S., & Winner, E. (1997). Attributing meaning to deliberately false utterances. In C. Mandell & A. McCabe (Eds.), *The problem of meaning: Behavioral and cognitive perspectives*. Amsterdam: Elsevier Science.
- Dimitrovsky, L., Spector, H., Levy-Shiff, R., & Vakil, E. (1998). Interpretation of facial expressions of affect in children with learning disabilities with verbal or nonverbal deficits. *Journal of Learning Disabilities*, 31, 286–312.
- Dunn, J., Brown, J., Slomkowski, C., Tesla, C., & Youngblade, L. (1991). Young children's understanding of other people's feelings and beliefs: Individual differences and their antecedents. *Child Development*, 62, 1352–1366.
- Durkin, K., & Conti-Ramsden, G. (2007). Language, social behavior, and the quality of friendships in adolescents with and without a history of specific language impairment. *Child Development*, 78(5), 1441–1457.
- Elksnin, L. K., & Elksnin, N. (2000). *Teaching parents to teach their children to be prosocial*. Retrieved November 10, 2009, from www.ldonline.org/article/6036
- Faigin, G. (1990). *The artist's complete guide to facial expression*. New York: Watson-Guptill.
- Filippova, E., & Astington, J. W. (2008). Further development in social reasoning revealed in discourse irony understanding. *Child Development*, 79(1), 126–138.
- Flin, R., & Dziurawiec, S. (1989). Developmental factors in fact processing. In A. W. Young & H. D. Ellis (Eds.), *Handbook of research on face processing* (pp. 335–378). New York: Elsevier.
- Ford, J. A., & Milosky, L. M. (2003). Inferring emotional reactions in social situations: Differences in children with language impairment. *Journal of Speech, Language, and Hearing Research*, 46, 21–30.
- Franklin, J. (2009). *Social and emotional: Educating the whole child*. Retrieved November 10, 2009, from www.thesouthern.com
- Fujiki, M., Brinton, B., Robinson, L. A., & Watson, V. (1997). The ability of children with specific language impairment to participate in a group decision task. *Journal of Childhood Communication Development*, 18, 1–10.
- Fujiki, M., Brinton, B., Hart, C. H., & Fitzgerald, A. (1999a). Peer acceptance and friendship in children with specific language impairment. *Topics in Language Disorders*, 19, 34–38.
- Fujiki, M., Brinton, B., Morgan, M., & Hart, C. (1999b). Withdrawn and social behavior of children with language impairment. *Language, Speech, and Hearing Services in Schools*, 39, 183–195.
- Fujiki, M., Spackman, M. P., Brinton, B., & Hall, A. (2004). The relationship of language and emotion regulation skills to reticence in children with specific language impairment. *Journal of Speech, Language, and Hearing Research*, 47, 637–646.
- Fujiki, M., Brinton, B., Jones, E., Quist, N., Stott, D., Gardner, V., et al. (2008). *The ability of children with LI to hide emotions in hypothetical scenarios and naturalistic contexts*. Poster session presented at the ASHA Convention, Chicago.
- Gallagher, T. M. (1993). Language skill and the development of social competence in school-age children. *Language, Speech, and Hearing Services in Schools*, 24, 199–205.
- Gertner, B. L., Rice, M. L., & Hadley, P. A. (1994). Influence of communicative competence on peer preference in a preschool classroom. *Journal of Speech and Hearing Research*, 37, 913–923.
- Gresham, F. M., Sugai, G., & Horner, R. H. (2001). Interpreting outcomes of social skills training for students with high-incidence disabilities. *Exceptional Children*, 67, 331–344.
- Grice, H. P. (1980). *Studies in the way of words*. Cambridge, MA: Harvard University Press.
- Gutstein, S. E., & Shelly, R. K. (2002). *Relationship development intervention with young children: Social and emotional development activities for Asperger syndrome, autism, PDD and NLD*. London/New York: Jessica Kingsley.
- Hadley, P. A., & Rice, M. L. (1991). Conversational responsiveness in speech and language-impaired preschoolers. *Journal of Speech and Hearing Research*, 34, 1308–1317.
- Hadwin, J., & Perner, J. (1991). Pleased and surprised: Children's cognitive theory of emotion. *British Journal of Developmental Psychology*, 9, 215–234.
- Hawkins, J. D., Kosterman, R., Catalano, R. F., Hill, K. G., & Abbott, R. D. (2008). Effects of social development intervention in childhood 15 years later. *Archives of Pediatrics & Adolescent Medicine*, 162(12), 1133–1141.
- Henry, F. M., Reed, V. A., & McAllister, L. L. (1995). Adolescents' perceptions of the relative importance of selected communication skills in their positive peer relationships. *Language, Speech, and Hearing Services in Schools*, 26, 263–272.

- Jackson, S., Enright, R., & Murdock, J. (1987). Social perception problems in learning disabled youth: Developmental lag versus perceptual deficit. *Journal of Learning Disabilities*, 20, 361–364.
- Janusz, J., Kirkwood, M., Yeates, K., & Taylor, H. (2002). Social problem-solving skills in children with traumatic brain injury: Long-term outcomes and prediction of social competence. *Child Neuropsychology*, 8, 179–194.
- Kessler, M., & Donahue, M. (2008). *Language and learning in school-age children and adolescents: Reading comprehension and social inferencing in middle school students with learning disabilities*. Poster session presented at the ASHA Convention, Chicago.
- Kleinhaus, N., Richards, T., Sterling, L., Stegbauer, K., Mahurin, R., Clark Johnson, L., et al. (2008). Abnormal functional connectivity in autism spectrum disorders during face processing. *Brain*, 131(4), 1000–1012.
- Kuebli, J., Butler, S., & Fivush, R. (1995). Mother-child talk about past emotions: Relations of maternal language and child gender over time. *Cognition and Emotion*, 9, 265–283.
- Lakoff, R. (1973). The logic of politeness: Or minding your P's and Q's. In *Papers presented at the ninth regional meeting of the Chicago Linguistic Society* (pp. 292–305). Chicago: Chicago Linguistics Society.
- Lapadat, J. D. (1991). Pragmatic language skills of students with language and/or learning disabilities: A quantitative synthesis. *Journal of Learning Disabilities*, 24, 147–158.
- Lavoie, R. (1994). *Last one picked . . . first one picked on: Learning disabilities and social skills with Rick Lavoie (DVD)*. Arlington: Public Broadcasting Service.
- Leadbeater, B. J., Hellner, I., Allen, J. P., & Aber, J. L. (1989). The assessment of interpersonal negotiation strategies in multi-problem youth. *Developmental Psychology*, 25, 465–472.
- Leadbeater, B., Ohan, J., & Hoglund, W. (2006). How children's justifications of the "best thing to do" in peer conflicts relate to their emotional and behavioral problems in early elementary school. *Merrill-Palmer Quarterly*, 52, 807–821.
- Limperopoulos, C., Bassan, H., Sullivan, N. R., Soul, J. S., Robertson, R. L., Jr., Moore, M., et al. (2008). Positive screening for autism in ex-preterm infants: Prevalence and risk factors. *Pediatrics*, 121(4), 758–765.
- Lleras, C. (2008). Do skills and behaviors in high school matter? The contribution of noncognitive factors in explaining differences in educational attainment and earnings. *Social Science Research*, 37, 888–902.
- LoGuidice, C., & LaQuay, K. (2009). *No-glamour idioms*. East Moline: LinguiSystems.
- LoGuidice, C., & Warner, M. (2003). *The nonverbal language kit*. East Moline: LinguiSystems.
- McClellan, D., & Katz, L. (1993). *Young children's social development: A checklist* (p. ED356100). Urbana: ERIC Clearinghouse on Elementary and Early Childhood Education.
- McConnell, N., & LoGiudice, C. (1998). *That's life! Social language*. East Moline: LinguiSystems.
- McKinley, N. L., & Lord-Larson, V. (1985). Neglected language-disordered adolescent: A delivery model. *Language, Speech, and Hearing Services in Schools*, 34, 117–127.
- Mehrabian, A. (1971). *Silent messages*. Belmont: Wadsworth.
- Meltzoff, A., & Decety, J. (2003). What imitation tells us about social cognition: A rapprochement between developmental psychology and cognitive neuroscience. *The Royal Society*, 358, 491–500.
- Mishna, F. (2003). Learning disabilities and bullying: Double jeopardy. *Journal of Learning Disabilities*, 36, 336–347.
- Mize, J., & Abell, E. (1996). Encouraging social skills in young children: Tips teachers can share with parents. *Dimensions of Early Childhood*, 24, 15–23.
- Moore-Brown, B., Sanger, D., Montgomery, J., & Nishida, B. (2002). Communication and violence: New roles for speech-language pathologists. *The ASHA Leader*, 7 (4–5), 14.
- Nakassis, C., & Snedeker, J. (2002). Beyond sarcasm: Intonation and context as relational cues in children's recognition of irony. In A. Greenhill, M. Hughes, H. Littlefield, & H. Walsh (Eds.), *Proceedings of the twenty-sixth Boston University conference on language development*. Somerville: Cascadilla Press.
- Nakkula, M. J. (2001). Negotiation training and interpersonal development: An exploratory study of early adolescents in Argentina. *Adolescence*, 36, 1–20.
- National Association of School Psychologists. (2002). *Social skills: Promoting positive behavior, academic success, and school safety*. Retrieved December 30, 2005, from http://www.naspcenter.org/factsheets/socialskills_fs.html
- National Center for Technology Innovation (NCTI) and Center for Implementing Technology in Education (CITED). (2007). *Practicing social skills: How to teach your student social interactions*. Retrieved November 10, 2009, from www.ldonline.org/article/21025
- National Network for Child Care (NNCC), Asher, S. R., & Williams, G. (1993). Children without friends, part 1: Their problems. In C. M. Todd (Ed.), *Day care center connections* (pp. 3–4). Urbana-Champaign: University of Illinois Cooperative Extension Service.
- Nelson, K. (1996). *Language in cognitive development*. Cambridge: Cambridge University Press.
- Ninio, A., & Snow, C. (1999). The development of pragmatics: Learning to use language appropriately. Invited chapter. In T. K. Bhatia & W. C. Ritchie (Eds.), *Handbook of language acquisition*. New York: Academic.
- Nippold, M. A. (1993). Developmental markers in adolescent language: Syntax, semantics, and pragmatics. *Language, Speech, and Hearing Services in Schools*, 24, 21–28.
- Nippold, M. A., Mansfield, T. C., & Billow, J. L. (2007). Peer conflict explanations in children, adolescents, and

- adults: Examining the development of complex syntax. *American Journal of Speech-Language Pathology*, 16, 179–188.
- Oliva, A., & La Greca, A. (1988). Children with learning disabilities: Social goals and strategies. *Journal of Learning Disabilities*, 21, 301–306.
- Paul, R. (2001). *Language disorders from infancy through adolescence: Assessment and intervention* (2nd ed.). St. Louis: Mosby-Yearbook.
- Pearl, R., Bryan, T., Fallon, P., & Herzog, A. (1991). Learning disabled students' detection of deception. *Learning Disabilities Research and Practice*, 6, 12–16.
- Peterson, C., & Slaughter, V. (2003). Opening windows into the mind: Mothers' preferences for mental state explanations and children's theory of mind. *Cognitive Development*, 18, 399–429.
- Place, K. S., & Becker, J. A. (1991). The influence of pragmatic competence on the likeability of grade-school children. *Discourse Processes*, 14, 227–241.
- Possel, P., Seemann, S., Ahrens, S., & Hautzinger, M. (2006). Testing the causal mediation component of Dodge's social information processing model of social competence and depression. *Journal of Youth and Adolescence*, 35, 849–859.
- Project for School Innovation. (2004). *Making inferences from text*. Project for school innovation. Retrieved July 1, 2007, from <http://www.psinnovation.org/files/documents/LSGIntro.doc>
- Quinn, K. A., Macrae, C. N., & Bodenhausen, G. V. (2003). Social cognition. In L. Nadel (Ed.), *Encyclopedia of cognitive science* (Vol. 4, pp. 66–73). London: Nature.
- Raffaelli, M., & Duckett, E. (1989). "We were just talking . . .": Conversations in early adolescence. *Journal of Youth and Adolescence*, 18(6), 567–582.
- Raskind, M. (2005). *Research trends: Social information processing and emotional understanding in children with LD*. Retrieved November 10, 2009, from <http://www.greatschools.net/cgi-bin/showarticle/2974>
- Reed, V. A., McLeod, K., & McAllister, L. (1999). Importance of selected communication skills for talking with peers and teachers: Adolescents' opinions. *Language, Speech, and Hearing Services in Schools*, 30, 32–49.
- Rice, M. L., Sell, M. A., & Hadley, P. A. (1991). Social interactions of speech- and language-impaired children. *Journal of Speech and Hearing Research*, 34, 1299–1307.
- Rossetti, L. (2006). *The Rossetti infant-toddler language scale*. East Moline: LinguiSystems.
- Rothenberg, S. (1998). *Nonverbal learning disabilities and social functioning*. Retrieved March 28, 2007, from NLD on the web: www.nldontheweb.org/Rothenberg-1.htm
- Ruble, L., & Gallagher, T. (2004). *Autism spectrum disorders: Primer for parents and educators. Special needs*. Bethesda: National Association of School Psychologists.
- Ruby, P., & Decety, J. (2003). What you believe versus what you think they believe: A neuroimaging study of conceptual perspective-taking. *European Journal of Neuroscience*, 17, 2475–2480.
- Ruby, P., & Decety, J. (2004). How would you feel versus how do you think she would feel? A neuroimaging study of perspective-taking with social emotions. *Journal of Cognitive Neuroscience*, 16(6), 988–999.
- Santrock, J. W. (2005). *Child development* (10th ed.). New York: McGraw-Hill.
- Schultz, L. H., & Selman, R. L. (1999). *The meaning and measurement of social competence from a developmental perspective*. Cambridge, MA: Harvard University Graduate School of Education. Manuscript submitted for publication.
- Schumaker, J., & Deshler, D. (1995). Social skills and learning disabilities. Newsbriefs, *Learning Disabilities Association of America*, March–April, pp. 8–11.
- Selman, R., & Demorest, A. (1984). Observing troubled children's interpersonal negotiation strategies: Implications of and for a developmental model. *Child Development*, 55, 288–304.
- Selman, R. L., & Schultz, L. H. (1990). *Making a friend in youth: Developmental theory and pair therapy*. Chicago: University of Chicago Press.
- Selman, R., Beardslee, W., Schultz, L., Krupa, M., & Podorefsky, D. (1986). Assessing adolescent interpersonal negotiation strategies: Toward the integration of structural and functional models. *Developmental Psychology*, 22, 450–459.
- Shamay-Tsoory, S. G., Tomer, R., & Aharon-Peretz, J. (2005). The neuroanatomical basis of understanding sarcasm and its relationship to social cognition. *Neuropsychology*, 19(3), 288–300.
- Shatz, M., Wellman, H. J., & Silber, S. (1983). The acquisition of mental verbs: A systematic investigation of the first reference to mental state. *Cognition*, 14, 301–321.
- Sillars, A. L. (1991). Behavior observation. In B. M. Montgomery & S. Duck (Eds.), *Studying interpersonal interaction*. New York: Guilford Press.
- Smith, I. M., & Bryson, S. E. (2007). Gesture imitation in autism: II. Symbolic gestures and pantomimed object use. *Cognitive Neuropsychology*, 24(7), 679–700.
- Stevens, L. J., & Bliss, L. S. (1995). Conflict resolution abilities of children with specific language impairment and children with normal language. *Journal of Speech and Hearing Research*, 38, 599–611.
- Sweet Nichols, C. (1998). Encoding of nonverbal social cues by children with differing profiles of learning disabilities. *Dissertation Abstracts International*, 59 (5-B), 2489.
- Sweetser, E. (1987). The definition of "lie." An examination of the folk models underlying a semantic prototype. In D. Holland & N. Quinn (Eds.), *Cultural models in language and thought*. New York: Cambridge University Press.
- Talwar, V., & Lee, K. (2002). Emergence of white lying in children between 3 and 7 years of age. *Merrill-Palmer Quarterly*, 48, 160–181.

- Talwar, V., Murphy, S. M., & Lee, K. (2007). White lie-telling in children for politeness purposes. *International Journal of Behavioral Development*, 31, 1–11.
- Timler, G. R. (2008). Social knowledge in children with language impairments: Examination of strategies, predicted consequences, and goals in peer conflict situations. *Clinical Linguistics & Phonetics*, 22, 741–763.
- Timler, G., Olswang, L., & Coggins, T. (2005). Do I know what I need to do? *Language, Speech, and Hearing Services in Schools*, 36, 73–85.
- Tur-Kaspa, H., & Bryan, T. (1994). Social information-processing skills of students with learning disabilities. *Learning Disabilities Research and Practice*, 9, 12–23.
- Turkstra, L. S. (2005). Looking while listening and speaking: Eye-to-face gaze in adolescents with and without traumatic brain injury. *Journal of Speech, Language, and Hearing Research*, 48, 1429–1441.
- Turkstra, L. (2007). Pragmatic communication disorders: New intervention approaches. *The ASHA Leader*, 12, 16–17.
- Turkstra, L., Ciccio, A., & Seaton, C. (2003). Interactive behaviors in adolescent conversation dyads. *Language, Speech, and Hearing Services in Schools*, 34, 117–127.
- University of Illinois at Urbana-Champaign. (2008). *10 years on, high school social skills predict better earnings than test scores*. Retrieved November 10, 2009, from <http://www.sciencedaily.com/releases/2008/10/081015120749.htm>
- Vaughn, S., Elbaum, B., & Boardman, A. G. (2001). The social functioning of students with learning disabilities: Implications for inclusion. *Exceptionality*, 9, 47–65.
- Wang, A. T., Lee, S. S., Sigman, M., & Dapretto, M. (2007). Reading affect in the face and voice. *Archives of General Psychiatry*, 64, 698–708.
- Wang, J., Iannotti, R. J., & Nansel, T. R. (2009). School bullying among adolescents in the United States: Physical, verbal, relational, and cyber. *Journal of Adolescent Health*, 45(4), 368–375.
- Ward, K. (2007). *Autism: General information & practical suggestions for the classroom*. Retrieved November 10, 2009, from <http://www.autism.ca/teach.htm>
- Wiener, J. R., & Schneider, B. H. (2002). A multi-source exploration of the friendship patterns of children with and without learning disabilities. *Journal of Abnormal Child Psychology*, 30, 127–141.
- Williams White, S., Keonig, K., & Scahill, L. (2007). Social skills development in children with autism spectrum disorders: A review of the intervention research. *Journal of Autism and Developmental Disorders*, 37, 1858–1868.

Social Language Development Test-Adolescent

- [Social Language Development Test](#)

Social Language Development Test-Elementary

- [Social Language Development Test](#)

Social Learning

- [Social Cognitive Interventions](#)

Social Learning Disability/Difference

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Synonyms

[Autism spectrum disorder](#)

Definition

The term social learning disability/differences was coined by Volkmar (2016) as a way to more simply explain the nature of autism/Asperger's disorder/autism spectrum disorder. It was also chosen as a non-prerogative term that readily conveyed way to play problems/differences in social learning in the broader context of learning disabilities. This has some practical advantages in countries like the USA which strongly emphasize inclusion of individuals with learning problems in ongoing community life and occupation.

Terms like learning disability or learning disorder have commonly been used in English-speaking countries for effete to a range of problem in various areas such as reading, writing, math,

spelling, organization (executive functioning), and so forth (O'Brien and Pearson 2004). The literature on these disabilities/differences is relatively large and complex and the ways in which these problems have been defined both diagnostically and legally. It is often the case that a diagnosis of learning disability is made in the context of an individual with average (or even above average) intellectual potential. In the USA Section 504 of the Rehabilitation Act of 1973 had granted certain rights to individuals in occupation and educational (college) settings. This act differs from the Individuals with Disabilities Education Act that applies to children attending schools. Similar regulations exist in Canada and the UK while differing at times in specific definitions employed. The effects of learning disabilities broadly defined can be significant with lowered academic performance, poor self-esteem, and social problems. The term also (implicitly) recognizes the broad range of problem referred to typically as the broader autistic phenotype (Ingersoll et al. 2014).

For autism spectrum disorder, it is increasingly clear, as Kanner himself first suggested (1943) that the marked problems in social engagement, often from the beginning of life, have profound consequences for learning and may result in significant long-term social, communicative, and cognitive problems and additionally may complicate organization of executive functioning skills (NRC 2001; McPartland et al. 2014). There is an increasing large literature noting problems in areas such as joint attention, face perception, and so forth, and it is presumed that these problems pose great challenges for the individual acquiring knowledge in more typical ways (McPartland et al. 2014). Put another way without the "social frame" within which to view the world individuals with social learning differences/disorders may learn idiosyncratic ways often employing unusual strategies such as gestalt learning (Brosnan et al. 2004).

Practical advantages of the term include the focus on the social learning aspects of autism. It also may simplify understanding of lay audiences. As with other learning difficulties/differences, it also emphasizes the importance of inclusion and a commitment to helping individuals with learning differences.

See Also

- Autism Spectrum Disorder
- Autistic Disorder
- Pervasive Developmental Disorder

References and Reading

- Brosnan, M. J., Scott, F. J., Fox, S., & Pye, J. (2004). Gestalt processing in autism: Failure to process perceptual relationships and the implications for contextual understanding. *Journal of Child Psychology & Psychiatry*, 45(3), 459–469.
- Ingersoll, B., Wainer, A., Volkmar, F. R., Paul, R., Rogers, S. J., & Pelphrey, K. A. (2014). The broader autism phenotype. In *Handbook of autism and pervasive developmental disorders* (4th ed.). Wiley.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- McPartland, J. C., Tillman, R. M., Yang, D. Y. J., Bernier, R. A., Pelphrey, K. A., Volkmar, F. R., . . . , & Pelphrey, K. A. (2014). The social neuroscience of autism spectrum disorder. In *Handbook of autism and pervasive developmental disorders* (4th ed.). Wiley.
- National Research Council. (2001). *Educating young children with autism*. Washington DC: NRC Press.
- O'Brien, G., & Pearson, J. (2004). Autism and learning disability. *Autism*, 8(2), 125–140.
- Section 504, Rehabilitation Act of 1973 (29 U.S.C. § 701).
- Volkmar, F. R. (2016). *The changing face of autism. The Birgit Olsson lecture*. Gothenburg: University of Gothenburg, November 16, 2016.

Social Motivation and Friendship Experiences of Autistic Adolescents

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Definition

Social motivation is defined as the intrinsically rewarding nature of interacting with other people.

Friendships are defined as reciprocal relationships between two people, characterized by

communication over time and a degree of emotional closeness and trust.

We use “identify-first” language (“autistic person”) rather than person-first language (‘person with autism’), because it is the preferred term of autistic activists (Sinclair 1999) and many autistic people and their families (Kenny et al. 2016) and is less associated with stigma (Gernsbacher 2017).

Historical Background

Having stable reciprocal friendships is held to be a key indicator of a “good adult outcome” (Goldfarb et al. 1976). It is unsurprising, then, that many adolescents and adults diagnosed with autism – defined in part by “deficits in developing, maintaining, and understanding relationships” (American Psychiatric Association 2013) – rarely achieve such good outcomes, as defined in this way. Long-term longitudinal studies have found that autistic people have few, if any, friends, are less likely to be married or in a long-term romantic relationship (Howlin 2000; Howlin et al. 2013), and are more likely to rely on their parents for social support than on same-age peers or colleagues (Howlin et al. 2004) than their neurotypical counterparts. These findings have fueled a wealth of research on the social connections of autistic children and young people (review: Petrina et al. 2014) and generated numerous interventions and supports designed to help autistic children and young people develop and maintain long-lasting friendships with others (review: Chang and Locke 2016).

There is increasing recognition, however, that traditional, normative markers of what a successful (social) outcome is for an autistic person all too often have been derived on the basis of *non-autistic* researchers and clinicians’ perspectives, rather than on the views of young autistic people themselves (Cribb et al. 2019; Ruble and Dalrymple 1996). In this review, we provide an overview of research, which focuses on young autistic people’s own subjective experiences of their friendships, identifies key factors that contribute to variation in those definitions and outcomes,

and seeks to develop effective interventions to support them in achieving their goals.

Current Knowledge

Friendships on the Autism Spectrum

It has been repeatedly demonstrated that young autistic people have fewer friendships (Bauminger and Shulman 2003) and higher levels of loneliness (Bauminger and Kasari 2000), and experience greater social exclusion than both neurotypical young people and those with other neurodevelopmental conditions (Kasari, Locke, Gulsrud and Rotheram-Fuller 2011; Locke, Ishijima, Kasari, and London 2010; Rowley et al. 2012). Difficulties with developing friendships and social relationships have often been attributed to “diminished social motivation” (Chevallier et al. 2012) suggesting that autistic people cannot, and do not *want*, to form friendships (for critical review, see Jaswal and Akhtar 2019). Yet, anecdotal reports combined with long-standing empirical work suggest, however, that this is not always, or even usually, the case.

Research has demonstrated that autistic people are interested in forming secure and supportive friendships and are emotionally invested in the friendships they have (Calder et al. 2013; Sumiya et al. 2018). They report having friends and best friends (Mendelson et al. 2016; Sedgewick et al. 2016) and have a desire to play with, and chat to, their non-autistic peers (Travis et al. 2001). Autistic children tend to have fewer friends than non-autistic children, see those friends less often, and tend to focus on shared activities rather than emotional sharing – but are highly satisfied with their friendships regardless of these differences (Petrina et al. 2014). This is the case regardless of whether their nominated friend is also autistic, and the non-autistic friends of autistic children are just as happy with the friendship as the autistic partner (Petrina et al. 2017). Importantly, a recent study has shown that mental age-matched autistic and non-autistic children (estimated average 10 years old) had similar expectations of their friendships (Bottema-Beutel et al. 2018).

The extent and nature of young autistic people's friendships could be influenced by several factors, including (1) homophily, (2) others' acceptance and inclusion of young autistic people, (3) an individual's motivation to make and keep friends, and (4) gender.

Homophily. Homophily – similar interests, beliefs, and attributes – is a strong element in the friendships of non-autistic young people (Shrum et al. 1988), and the same is true for young autistic people. Recent research has shown that they take similar features into consideration when making and maintaining friendships as their non-autistic counterparts, such as shared interests and wanting similar types and levels of interaction (Finke et al. 2019).

Research has also demonstrated that young autistic people who are involved in extra-curricular activities like sports and hobby clubs have more friends than those who do not take part in these activities, often facilitated by the shared interest (Dovgan and Mazurek 2019). Shared interests can also help to build social skills, thereby improving social relationships (Nabors et al. 2017). These shared interests do not have to be in-person activities. Many autistic people make genuine friendships online, for example, through gaming or social media (Brownlow et al. 2015; Ringland et al. 2016). Online interactions require less decoding of complex social information, such as tone of voice and body language, and come with a ready-made set of emotional indicators (emojis) and non-verbal reactions (e.g., gifs, memes) with recognized meanings. Adults on the autism spectrum have expressed that communicating online is good for them as it gives them time to plan what they want to say and because it removes the challenges of face-to-face talking (Benford and Standen 2009; Burke et al. 2010). Social media use has been shown to be associated with higher friendship quality for autistic adolescents, though high levels of anxiety had similar negative effects on offline and online friendships (van Schalkwyk et al. 2017). That online friendships should be considered a central part of our definition of friendship is important in recognizing the reality of social engagement in the modern world.

Acceptance and Inclusion. Another aspect which has been found to be key in building positive friendships for young autistic people is acceptance. Research in this area is relatively new, although there is a multitude of first-person accounts of the stigma experienced by autistic people and the difference an accepting community can make to wellbeing and quality of life.

Among autistic people, greater acceptance of their autism and associated needs is linked to better relationships, better mental health, and greater life satisfaction (Cage et al. 2018). Positive self-perceptions also mediate the relationship between increased self-esteem and better mental health (Cooper et al. 2017). Young autistic people are often anxious about “getting things wrong” socially, so finding friends who are accepting of their differences and allow them to be their authentic selves is important, and these friendships are stronger than relationships where they feel they must pretend not to be autistic (Sosnowy et al. 2019). Indeed, positive correlations have been found between good peer interactions and friendships and increased self-esteem among young autistic people (Johnson 2019). This research highlights the importance of young autistic people building the friendships they want to have, rather than being encouraged to have friendships like those of young people who are not on the autism spectrum.

Motivation to make and keep friends. The well-known social motivation theory posits that autistic people have lower levels of social motivation, such that they find social interaction less intrinsically rewarding and do not seek out relationships (Chevallier et al. 2012). Recent work thoroughly critiques this conception of autistic people's social lives (Jaswal and Akhtar 2019). Indeed, in a study of autistic and non-autistic children's social networks, Calder et al. (2013) found that it was autistic children's social motivation that appeared to account for the significant variation in the nature and extent of their friendships. While some young autistic people desperately wanted friends, others had limited social connections but preferred things this way: “I am happy with my life right now. I am not friendly and talkative, but

I am not not friendly. I am somewhere in the middle” (p. 12).

One potential source of individual differences in young autistic people’s motivation for making and keeping friends is gender. Just as there are recognized gender differences in the nature and extent of friendships among people who are not autistic (Aukett et al. 1988; Caldwell and Peplau 1982; De Goede et al. 2009), there is growing evidence for gender differences in the friendships, social skills, and social motivation of young autistic people. Research on autistic friendships and social relationships has almost exclusively focused on male participants, largely based around the historical assumption that autism is a mostly male condition and/or the belief that those few girls who received a diagnosis were so rare as to be unrepresentative and not worth including (Gould and Ashton-Smith 2011; Haney 2016). As there is increased recognition of the differences between males and females on the autism spectrum (Lai et al. 2015), understanding the differences in their friendships, and what causes these differences, is important for being able to support young autistic people to have positive social relationships.

Autistic girls are more likely to have apparently typical social development (Bauminger et al. 2010) than autistic boys. This is in part because autistic girls tend to be more motivated toward, and skilled at, imitating the social behaviors of their peers, such as cooperative and pretend play (Kopp and Gillberg 2011). Autistic girls engage in complex imitation (Hiller et al. 2014), have language skills similar to those of non-autistic female peers (Goddard et al. 2014; Kauschke et al. 2016), and are more socially cooperative (Mandy et al. 2012) than autistic boys. Potentially driving these differences, autistic girls have consistently been found to have higher levels of social reciprocity and social motivation than autistic boys (Head et al. 2014; Mandic-Maravic et al. 2015; Sedgewick et al. 2016, 2018).

These skills, combined with higher levels of “camouflaging” behaviors aimed at helping autistic girls to hide their social challenges and appear “normal” so they can try to fit in (Dean et al. 2017;

Hull et al. 2017; Milner et al. 2019), mean that autistic girls are likely to have very different social experiences to autistic boys. The social isolation of autistic girls is often less obvious, being more akin to neglect than active rejection (Dean et al. 2014) and means that parents and teachers may not notice and intervene to help them make friends.

Gender Differences. There are significant differences in the ways in which adolescent autistic boys and girls spend time with their friends, despite spending similar amounts of time socializing overall (Kuo et al. 2013). Autistic boys tended to play games with their friends, whereas autistic girls were more likely to chat with theirs. Another study found that autistic girls aged 10–16 years scored significantly higher on the Friendship Questionnaire – with higher scores representing better friendships – than autistic boys and, furthermore, scored similarly to boys who were *not* autistic (Head et al. 2014). This finding was supported by parental reports of the children’s relationships, suggesting that autistic girls have better social skills and higher social motivation than autistic boys.

Research suggests that, as girls face similar expectations regardless of diagnostic status, being female may be more important in determining social experiences than being autistic, and this needs to be considered when supporting autistic girls (Carpenter et al. 2019). For example, studies of social motivation and friendships among autistic adolescents in both special (Sedgewick et al. 2016) and mainstream (Sedgewick et al. 2018) education settings found gender differences consistent with those of non-autistic adolescents. Autistic girls rated their best friendships as closer and more supportive than autistic boys, at similar levels to non-autistic girls. Those best friendships were also characterized by similar behaviors to non-autistic girls, such as emotional sharing and talking about other people, rather than being focused on shared actions or objects (i.e., games or lessons).

Where there were important differences between autistic and non-autistic girls was in the form their friendships took. While non-autistic girls tended to have several best friends plus a

wider group of friends and acquaintances, autistic girls had one or two best friends without a wider group, and the best friendships of autistic girls were very intense and could be an all-consuming preoccupation. Nevertheless, autistic girls often spent time with their friends in the same ways as non-autistic girls, such as chatting and going to the cinema, and saw their friendships as just as important and positive as non-autistic girls did.

Supporting Friendships

The research reviewed above demonstrates that a range of factors influence the friendship experiences of young autistic people. Any interventions or strategies that seek to support the formation and maintenance of positive friendships for young autistic people must consider shared interests, non-autistic peers' attitudes toward the young autistic person, and young people's preferences for making and keeping friends – allowing them to form their own vision of a “good” social life. One key area where specific support is likely to be needed is in navigating the negative aspects of social relationships, both bullying and conflict within friendships, which are all too common for young autistic people.

Young autistic people are more likely to be bullied than their peers due to their social vulnerabilities (Cappadocia et al. 2012; Sofronoff et al. 2011), with up to 94% of young autistic people reporting being bullied (Hebron and Humphrey 2014; van Roekel et al. 2010). Being bullied is associated with being more likely to have mental health issues in young autistic people, though those with good friends and higher self-esteem are somewhat protected (Cook et al. 2016).

Although there is a wealth of work on bullying of young autistic people (for review, see Hong and Espelage 2012), little research has been conducted on the impact of conflict *within* their friendships and peer relationships. These behaviors can have negative impacts similar to those of more overt bullying but, critically, take place within relationships typically categorized as “friendships.” This categorical (friends, who are usually nice to you) and behavioral (someone being unpleasant or bullying) mismatch can lead to difficulties for any adolescent who must try to make space for mean

behaviors within the concept of a “friend” but may be especially puzzling for teenagers on the autism spectrum. Indeed, autistic adolescents can often have a “fixed” and “active” definition of friendship, focused on doing things with someone – “someone you hang out with” (Bauminger and Kasari 2000; Calder et al. 2013) – rather than emotional closeness, which may leave autistic adolescents vulnerable to social manipulation.

Importantly, this fixed definition of friendship can also lead to fixed ideas about what represents a breach of that friendship. One study showed that young autistic people view “betrayal” as the most severe transgression in a friendship (Bottema-Beutel et al. 2018). The perspective-taking difficulties often experienced by young autistic people might mean that they can misinterpret the actions of others as a transgression when they were not intended as such (van Roekel et al. 2010). They may then react more easily (Mazefsky et al. 2013) and in a more emotionally extreme manner than non-autistic young people would (Gomez and Baird 2005; Volker et al. 2010). They are also less likely to forgive a friend who upsets them (Rogé and Mullet 2011) due to a sense of having been done an injustice. This emotional reaction, however, is likely to result in further teasing from peers rather than reducing the bullying.

Just as young autistic people generally are more likely to be bullied than non-autistic young people, autistic girls are more likely to be bullied than autistic boys (Sedgewick et al. 2018). The challenges of friendship formation and maintenance may be especially significant for autistic girls, who are more likely to experience relational conflict than autistic boys, just as non-autistic girls experience more relational conflict than non-autistic boys (Bowie 2007). In light of these gender differences, research has started to explore how autistic girls experience and manage relational conflict. Several studies have found that autistic girls experience high levels of this conflict and often fall out with friends as a result of it (Milner et al. 2019; Sedgewick et al. 2018). One study found that autistic girls are more likely to have an “all or nothing” response to conflict with a friend, seeing it either as entirely their fault and therefore entirely their responsibility to ‘fix’ or as

entirely the other person's fault and entirely for them to resolve (Sedgewick et al. 2018). Other work has shown that autistic girls do have conflict resolution strategies but can struggle to predict the expectations of their peers and so face difficulties effectively deploying these approaches (Vine Foggo and Webster 2017). Indeed, research has shown that difficulties with developing and using social problem-solving strategies are common for young autistic people (Hochhauser et al. 2015), even when they are highly motivated to repair the damage and want to maintain the friendship.

Future Directions

There is a huge amount still to learn about friendships among young autistic people. There is a need for future research to examine the experiences of those young people who are non-binary and transgender; on the ways that social relationships change over time, especially during times of life transition; on the reciprocal influences of mental health and friendships on the well-being of young autistic people; and on the differences between mixed autistic/non-autistic and non-mixed friendships.

The friendships young autistic people choose to make may not look exactly like those of non-autistic young people. But they are valid and genuine, and appear to have similar beneficial long-term impacts as all friendships. Understanding that these friendships are possible for young autistic people, and that they can enjoy these friendships on their own terms, is a critically important message, which should be shared with young people, their families, and the people supporting them.

See Also

There are a range of programs designed to support social skills and relationships among autistic young people. Some examples include:

The Program for the Education and Enrichment of Relational Skills (PEERS; Laugeson et al. 2014) – a 16-week program of lesson plans to help autistic

young people develop social skills and practice social interaction

Social Stories (Gray 1994) – a manualized approach to designing individualized stories to help autistic young people predict and practice social interactions
The Hidden Curriculum (Myles, Trautman and Schelvan 2004) – a book that gives practical tips and tutorials for teachers and parents to support autistic young people in learning unwritten social rules

The Secret Agent Society (Beaumont et al. 2015) – a social-emotional skills training program for school-aged autistic children that includes child group sessions, parent workshops, and teacher tip sheets, home missions, using a multilevel computer game and a system to reward and monitor progress at home and school

The Transporters (Golan et al. 2010) – an animated series which aims to teach autistic young children about reading facial expressions and emotions displayed on the face

Girls Night Out (Jamison and Schuttler 2017) – a social skills and self-care program designed with autistic adolescent girls in mind, aimed at helping them understand and engage with their peers more successfully

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5*. Washington, DC: American Psychiatric Association.
- Aukett, R., Ritchie, J., & Mill, K. (1988). Gender differences in friendship patterns. *Sex Roles*, 19(1–2), 57–66. <https://doi.org/10.1007/BF00292464>.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development*, 71(2), 447–456. <https://doi.org/10.1111/1467-8624.00156>.
- Bauminger, N., & Shulman, C. (2003). The development and maintenance of friendship in high-functioning children with autism: Maternal perceptions. *Autism*, 7, 81–97. <https://doi.org/10.1177/1362361303007001007>
- Bauminger, N., Solomon, M., & Rogers, S. J. (2010). Externalizing and internalizing behaviors in ASD. *Autism Research*, 3(3), 101–112. <https://doi.org/10.1002/aur.131>.
- Beaumont, R., Rotolone, C., & Sofronoff, K. (2015). The secret agent society social skills program for children with high-functioning autism spectrum disorders: A comparison of two school variants. *Psychology in*

- the Schools, 52(4), 390–402. <https://doi.org/10.1002/pits.21831>.
- Benford, P., & Standen, P. (2009). The internet: a comfortable communication medium for people with Asperger syndrome (AS) and high functioning autism (HFA)? *Journal of Assistive Technologies*, 3(2), 44–53. <https://doi.org/10.1108/17549450200900015>.
- Bottema-Beutel, K., Malloy, C., Cuda, J., Kim, S. Y., & MacEvoy, J. (2018). Responses to vignettes depicting friendship transgressions: Similarities and differences in children with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 1–12. <https://doi.org/10.1007/s10803-018-3828-y>.
- Bowie, B. H. (2007). Relational aggression, gender, and the developmental process. *Journal of Child and Adolescent Psychiatric Nursing*, 20(2), 107–115. <https://doi.org/10.1111/j.1744-6171.2007.00092.x>.
- Brownlow, C., Bertilsdotter Rosqvist, H., & O'Dell, L. (2015). Exploring the potential for social networking among people with autism: Challenging dominant ideas of 'friendship'. *Scandinavian Journal of Disability Research*, 17(2), 188–193. <https://doi.org/10.1080/15017419.2013.859174>.
- Burke, M., Kraut, R., & Williams, D. (2010). Social use of computer-mediated communication by adults on the autism spectrum. In *Proceedings of the 2010 ACM Conference on Computer Supported Cooperative Work – CSCW'10* (Vol. 425). <https://doi.org/10.1145/1718918.1718991>.
- Cage, E., Di Monaco, J., & Newell, V. (2018). Experiences of autism acceptance and mental health in autistic adults. *Journal of Autism and Developmental Disorders*, 48(2), 473–484. <https://doi.org/10.1007/s10803-017-3342-7>.
- Calder, L., Hill, V., & Pellicano, E. (2013). 'Sometimes I want to play by myself': Understanding what friendship means to children with autism in mainstream primary schools. *Autism*, 17(3), 296–316. <https://doi.org/10.1177/1362361312467866>.
- Caldwell, M., & Peplau, L. (1982). Sex differences in same-sex friendship. *Sex Roles*, 8(7), 721–732. <https://doi.org/10.1007/BF00287568>.
- Cappadocia, M. C., Weiss, J. A., & Pepler, D. (2012). Bullying experiences among children and youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(2), 266–277. <https://doi.org/10.1007/s10803-011-1241-x>.
- Carpenter, B., Happé, F., & Egerton, J. (2019). *Girls and autism: educational, family and personal perspectives*.
- Chang, Y.-C., & Locke, J. (2016). A systematic review of peer-mediated interventions for children with autism spectrum disorder. *Research in Autism Spectrum Disorders*, 27, 1–10. <https://doi.org/10.1016/J.RASD.2016.03.010>.
- Chevallier, C., Kohls, G., Troiani, V., Brodtkin, E. S., & Schultz, R. T. (2012). The social motivation theory of autism. *Trends in Cognitive Sciences*, 16(4), 231–239. <https://doi.org/10.1016/J.TICS.2012.02.007>.
- Cook, A., Ogden, J., & Winstone, N. (2016). The experiences of learning, friendship and bullying of boys with autism in mainstream and special settings: A qualitative study. *British Journal of Special Education*, 43(3), 250–271. <https://doi.org/10.1111/1467-8578.12143>.
- Cooper, K., Smith, L. G. E., & Russell, A. (2017). Social identity, self-esteem, and mental health in autism. *European Journal of Social Psychology*, 47(7), 844–854. <https://doi.org/10.1002/ejsp.2297>.
- Cribb, S., Kenny, L., & Pellicano, E. (2019). 'I definitely feel more in control of my life': The perspectives of young autistic people and their parents on emerging adulthood. *Autism*, 23(7), 1765–1781. <https://doi.org/10.1177/1362361319830029>.
- De Goede, I. H. A., Branje, S. J. T., & Meeus, W. H. J. (2009). Developmental changes and gender differences in adolescents' perceptions of friendships. *Journal of Adolescence*, 32(5), 1105–1123. <https://doi.org/10.1016/J.ADOLESCENCE.2009.03.002>.
- Dean, M., Kasari, C., Shih, W., Frankel, F., Whitney, R., Landa, R., . . . Harwood, R. (2014). The peer relationships of girls with ASD at school: Comparison to boys and girls with and without ASD. *Journal of Child Psychology and Psychiatry*, 55(11), 1218–1225. <https://doi.org/10.1111/jcpp.12242>.
- Dean, M., Harwood, R., & Kasari, C. (2017). The art of camouflage: Gender differences in the social behaviors of girls and boys with autism spectrum disorder. *Autism*, 21(6), 678–689. <https://doi.org/10.1177/1362361316671845>.
- Dovgan, K. N., & Mazurek, M. O. (2019). Relations among activity participation, friendship, and internalizing problems in children with autism spectrum disorder. *Autism*, 23(3), 750–758. <https://doi.org/10.1177/1362361318775541>.
- Finke, E. H., McCarthy, J. H., & Sarver, N. A. (2019). Self-perception of friendship style: Young adults with and without autism spectrum disorder. *Autism and Developmental Language Impairments*, 4, 2396941519855390. <https://doi.org/10.1177/2396941519855390>.
- Gernsbacher, M. A. (2017). Editorial perspective: The use of person-first language in scholarly writing may accentuate stigma. *Journal of Child Psychology and Psychiatry*, 58(7), 859–861. <https://doi.org/10.1111/jcpp.12706>.
- Goddard, L., Dritschel, B., & Howlin, P. (2014). A preliminary study of gender differences in autobiographical memory in children with an autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(9), 2087–2095. <https://doi.org/10.1007/s10803-014-2109-7>.
- Golan, O., Ashwin, E., Granader, Y., McClintock, S., Day, K., Leggett, V., & Baron-Cohen, S. (2010). Enhancing emotion recognition in children with autism spectrum conditions: An intervention using animated vehicles with real emotional faces. *Journal of Autism and Developmental Disorders*, 40(3), 269–279. <https://doi.org/10.1007/s10803-009-0862-9>.

- Goldfarb, R. H., Hermelin, B., Lansing, M., Lotter, V., Lovaas, O. I., Menyuk, P., . . . Rutter, M. (1976). International symposium on autism: A reappraisal of concepts and treatment. *Journal of Autism and Childhood Schizophrenia*, 6(1), 107–108.
- Gomez, C. R., & Baird, S. (2005). Identifying early indicators for autism in self-regulation difficulties. *Focus on Autism and Other Developmental Disabilities*, 20(2), 106–116. <https://doi.org/10.1177/10883576050200020101>.
- Gould, J., & Ashton-Smith, J. (2011). *Missed diagnosis or misdiagnosis? Girls and women on the autism spectrum*. Retrieved from <https://www.ingentaconnect.com/content/bild/gap/2011/00000012/00000001/art00005>.
- Gray, C. (1994). *Social stories*. Arlington: Future Horizons.
- Haney, J. L. (2016). Autism, females, and the DSM-5: Gender bias in autism diagnosis. *Social Work in Mental Health*, 14(4), 396–407. <https://doi.org/10.1080/15332985.2015.1031858>.
- Head, A. M., McGillivray, J. A., & Stokes, M. A. (2014). Gender differences in emotionality and sociability in children with autism spectrum disorders. *Molecular Autism*, 5(1), 19. <https://doi.org/10.1186/2040-2392-5-19>.
- Hebron, J., & Humphrey, N. (2014). Exposure to bullying among students with autism spectrum conditions: A multi-informant analysis of risk and protective factors. *Autism*, 18(6), 618–630. <https://doi.org/10.1177/1362361313495965>.
- Hiller, R. M., Young, R. L., & Weber, N. (2014). Sex differences in autism spectrum disorder based on DSM-5 Criteria: Evidence from clinician and teacher reporting. *Journal of Abnormal Child Psychology*, 42(8), 1381–1393. <https://doi.org/10.1007/s10802-014-9881-x>.
- Hochhauser, M., Weiss, P. L., & Gal, E. (2015). Negotiation strategies of adolescents with high-functioning autism spectrum disorder during social conflicts. *Research in Autism Spectrum Disorders*, 10, 7–14. <https://doi.org/10.1016/J.RASD.2014.10.022>.
- Hong, J. S., & Espelage, D. L. (2012). A review of research on bullying and peer victimization in school: An ecological system analysis. *Aggression and Violent Behavior*, 17(4), 311–322. <https://doi.org/10.1016/J.AVB.2012.03.003>.
- Howlin, P. (2000). Outcome in adult life for more able Individuals with autism or asperger syndrome. *Autism*, 4(1), 63–83. <https://doi.org/10.1177/1362361300004001005>.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45(2), 212–229. <https://doi.org/10.1111/j.1469-7610.2004.00215.x>.
- Howlin, P., Moss, P., Savage, S., & Rutter, M. (2013). Social outcomes in mid- to later adulthood among individuals diagnosed with autism and average nonverbal IQ as children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(6), 572–581.e1. <https://doi.org/10.1016/J.JAAC.2013.02.017>.
- Hull, L., Mandy, W., & Petrides, K. (2017). Behavioural and cognitive sex/gender differences in autism spectrum condition and typically developing males and females. *Autism*, 21(6), 706–727. <https://doi.org/10.1177/1362361316669087>.
- Jamison, T. R., & Schuttler, J. O. (2017). Overview and preliminary evidence for a social skills and self-care curriculum for adolescent females with autism: The girls night out model. *Journal of Autism and Developmental Disorders*, 47(1), 110–125. <https://doi.org/10.1007/s10803-016-2939-6>.
- Jaswal, V. K., & Akhtar, N. (2019). Being versus appearing socially uninterested: Challenging assumptions about social motivation in autism. *Behavioral and Brain Sciences*, 42, e82. <https://doi.org/10.1017/S0140525X18001826>.
- Johnson, K. (2019). Friendship: Understanding the differing perspectives of young adults with autism and their peers without autism. In *Annual Graduate Student Symposium*. Retrieved from <https://scholarworks.uni.edu/agss/2019/all/1>.
- Kasari, C., Locke, J., Gulsrud, A., & Rotheram-Fuller, E. (2011). Social networks and friendships at school: Comparing children with and without ASD. *Journal of Autism and Developmental Disorders*, 41, 533–544. <https://doi.org/10.1007/s10803-010-1076-x>.
- Kauschke, C., van der Beek, B., & Kamp-Becker, I. (2016). Narratives of girls and boys with autism spectrum disorders: Gender differences in narrative competence and internal state language. *Journal of Autism and Developmental Disorders*, 46(3), 840–852. <https://doi.org/10.1007/s10803-015-2620-5>.
- Kenny, L., Hattersley, C., Molins, B., Buckley, C., Povey, C., & Pellicano, E. (2016). Which terms should be used to describe autism? Perspectives from the UK autism community. *Autism*, 20(4), 442–462. <https://doi.org/10.1177/1362361315588200>.
- Kopp, S., & Gillberg, C. (2011). The Autism Spectrum Screening Questionnaire (ASSQ)-Revised Extended Version (ASSQ-REV): An instrument for better capturing the autism phenotype in girls? A preliminary study involving 191 clinical cases and community controls. *Research in Developmental Disabilities*, 32(6), 2875–2888. <https://doi.org/10.1016/J.RIDD.2011.05.017>.
- Kuo, M. H., Orsmond, G. I., Cohn, E. S., & Coster, W. J. (2013). Friendship characteristics and activity patterns of adolescents with an autism spectrum disorder. *Autism*, 17(4), 481–500. <https://doi.org/10.1177/13623613111416380>.
- Lai, M.-C., Lombardo, M. V., Auyeung, B., Chakrabarti, B., & Baron-Cohen, S. (2015). Sex/gender differences and autism: Setting the scene for future research. *Journal of the American Academy of Child and Adolescent Psychiatry*, 54(1), 11–24. <https://doi.org/10.1016/J.JAAC.2014.10.003>.
- Laugeson, E. A., Ellingsen, R., Sanderson, J., Tucci, L., & Bates, S. (2014). The ABC's of teaching social skills to adolescents with autism spectrum disorder in the classroom: The UCLA PEERS® program. *Journal of Autism and Developmental Disorders*, 44(9), 2244–2256. <https://doi.org/10.1007/s10803-014-2108-8>.

- Locke, J., Ishijima, E. H., Kasari, C., & London, N. (2010). Loneliness, friendship quality and the social networks of adolescents with high-functioning autism in an inclusive school setting. *Journal of Research in Special Educational Needs*, 10, 74–81. <https://doi.org/10.1111/j.1471-3802.2010.01148.x>
- Mandic-Maravic, V., Pejovic-Milovancevic, M., Mitkovic-Voncina, M., Kostic, M., Aleksic-Hil, O., Radosavljev-Kircanski, J., . . . Lecic-Tosevski, D. (2015). Sex differences in autism spectrum disorders: Does sex moderate the pathway from clinical symptoms to adaptive behavior? *Scientific Reports*, 5(1), 10418. <https://doi.org/10.1038/srep10418e>.
- Mandy, W., Chilvers, R., Chowdhury, U., Salter, G., Seigal, A., & Skuse, D. (2012). Sex differences in autism spectrum disorder: Evidence from a large sample of children and adolescents. *Journal of Autism and Developmental Disorders*, 42(7), 1304–1313. <https://doi.org/10.1007/s10803-011-1356-0>.
- Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(7), 679–688. <https://doi.org/10.1016/J.JAAC.2013.05.006>.
- Mendelson, J. L., Gates, J. A., & Lerner, M. D. (2016). Friendship in school-age boys with autism spectrum disorders: A meta-analytic summary and developmental, process-based model. *Psychological Bulletin*, 142(6), 601–622. <https://doi.org/10.1037/bul0000041>.
- Milner, V., McIntosh, H., Colvert, E., & Happé, F. (2019). A qualitative exploration of the female experience of autism spectrum disorder (ASD). *Journal of Autism and Developmental Disorders*, 1–14. <https://doi.org/10.1007/s10803-019-03906-4>.
- Myles, B.S., Trautman, M.L. & Schelvan, R.S. (2004). *The Hidden Curriculum: Practical Solutions for Understanding Unstated Rules in Social Situations*. Shawnee Mission, Kan.: Autism Asperger Publishing Company.
- Nabors, L., Hawkins, R., Yockey, A. R., Booker, S., & Tipkemper, A. (2017). Adolescents with autism spectrum disorder: Friendships and social interactions. *Advances in Neurodevelopmental Disorders*, 1(1), 14–20. <https://doi.org/10.1007/s41252-016-0001-5>.
- Petrina, N., Carter, M., & Stephenson, J. (2014). The nature of friendship in children with autism spectrum disorders: A systematic review. *Research in Autism Spectrum Disorders*, 8(2), 111–126. <https://doi.org/10.1016/J.RASD.2013.10.016>.
- Petrina, N., Carter, M., Stephenson, J., & Sweller, N. (2017). Friendship satisfaction in children with autism spectrum disorder and nominated friends. *Journal of Autism and Developmental Disorders*, 47(2), 384–392. <https://doi.org/10.1007/s10803-016-2970-7>.
- Ringland, K. E., Wolf, C. T., Faucett, H., Dombrowski, L., & Hayes, G. R. (2016). Will I always be not social? In *Proceedings of the 2016 CHI Conference on Human Factors in Computing Systems – CHI’16* (pp. 1256–1269). <https://doi.org/10.1145/2858036.2858038>.
- Rogé, B., & Mullet, E. (2011). Blame and forgiveness judgements among children, adolescents and adults with autism. *Autism*, 15(6), 702–712. <https://doi.org/10.1177/1362361310394219>.
- Rowley, E., Chandler, S., Baird, G., Simonoff, E., Pickles, A., Loucas, T., & Charman, T. (2012). The experience of friendship, victimization and bullying in children with an autism spectrum disorder: Associations with child characteristics and school placement. *Research in Autism Spectrum Disorders*, 6, 1126–1134. <https://doi.org/10.1016/j.rasd.2012.03.004>.
- Ruble, L. A., & Dalrymple, N. J. (1996). An alternative view of outcome in autism. *Focus on Autism and Other Developmental Disabilities*, 11(1), 3–14. <https://doi.org/10.1177/108835769601100102>.
- Sedgewick, F., Hill, V., Yates, R., Pickering, L., & Pellicano, E. (2016). Gender differences in the social motivation and friendship experiences of autistic and non-autistic adolescents. *Journal of Autism and Developmental Disorders*, 46(4), 1297–1306. <https://doi.org/10.1007/s10803-015-2669-1>.
- Sedgewick, F., Hill, V., & Pellicano, E. (2018). ‘It’s different for girls’: Gender differences in the friendships and conflict of autistic and neurotypical adolescents. *Autism*, 136236131879493. <https://doi.org/10.1177/1362361318794930>.
- Shrum, W., Cheek, N. H., & Hunter, S. M. (1988). Friendship in school: Gender and racial homophily. *Sociology of Education*, 61(4), 227. <https://doi.org/10.2307/2112441>.
- Sinclair, J. (1999). *Don't mourn for us*. Autistic Rights Movement UK.
- Sofronoff, K., Dark, E., & Stone, V. (2011). Social vulnerability and bullying in children with Asperger syndrome. *Autism*, 15(3), 355–372. <https://doi.org/10.1177/1362361310365070>.
- Sosnowy, C., Silverman, C., Shattuck, P., & Garfield, T. (2019). Setbacks and successes: How young adults on the autism spectrum seek friendship. *Autism in Adulthood*, 1(1), 44–51. <https://doi.org/10.1089/aut.2018.0009>.
- Sumiya, M., Igarashi, K., & Miyahara, M. (2018). Emotions surrounding friendships of adolescents with autism spectrum disorder in Japan: A qualitative interview study. *PLOS ONE*, 13(2), e0191538. <https://doi.org/10.1371/journal.pone.0191538>.
- Travis, L., Sigman, M., & Ruskin, E. (2001). Links between social understanding and social behavior in verbally able children with autism. *Journal of Autism and Developmental Disorders*, 31(2), 119–130. <https://doi.org/10.1023/A:1010705912731>.
- van Roekel, E., Scholte, R. H. J., & Didden, R. (2010). Bullying among adolescents with autism spectrum disorders: Prevalence and perception. *Journal of Autism and Developmental Disorders*, 40(1), 63–73. <https://doi.org/10.1007/s10803-009-0832-2>.
- van Schalkwyk, G. I., Marin, C. E., Ortiz, M., Rolison, M., Qayyum, Z., McPartland, J. C., . . . Silverman, W. K.

(2017). Social media use, friendship quality, and the moderating role of anxiety in adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(9), 2805–2813. <https://doi.org/10.1007/s10803-017-3201-6>.

Vine Foggo, R. S., & Webster, A. A. (2017). Understanding the social experiences of adolescent females on the autism spectrum. *Research in Autism Spectrum Disorders*, 35, 74–85. <https://doi.org/10.1016/J.RASD.2016.11.006>.

Volker, M. A., Lopata, C., Smerbeck, A. M., Knoll, V. A., Thomeer, M. L., Toomey, J. A., & Rodgers, J. D. (2010). BASC-2 PRS profiles for students with high-functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(2), 188–199. <https://doi.org/10.1007/s10803-009-0849-6>.

Social Pragmatic Communication Disorder

► Autism: Social Communication Disorder

Social Responsiveness Scale

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Synonyms

SRS

Abbreviations

AUC	Area under curve
ADHD	Attention deficit hyperactivity disorder
ADI-r	Autism diagnostic interview-revised
ADOS	Autism diagnostic observation scale
ASD	Autism spectrum disorder
BPVS	British picture vocabulary scale
CBCL	Child behavior checklist
CCC	Children’s communication checklist
OCD	Obsessive compulsive disorder
PDD-NOS	Pervasive developmental disorders-not otherwise specified

ROC	Receiver operating characteristics
SCDC	Social and communication disorders checklist
SCQ	Social communication questionnaire
VABS	Vineland adaptive behavior scales

Description

The Social Responsiveness Scale (SRS) is a 65-item rating scale that measures the severity of autistic symptomatology as a quantitative trait, among children clinically affected by autism spectrum conditions as well as among children in the general population. It is particularly useful for characterizing milder autistic syndromes that lie at the boundary between the normal population distribution and clinical-level affectation. The SRS can be completed by a parent, a teacher, a spouse (in the case of the adult version of the SRS), or another adult informant. The validity of self-report is still under study. The SRS involves ratings of children in their natural social contexts and reflects what has been consistently observed over weeks or months of time rather than in a single clinical or laboratory observation. In this way, it capitalizes on both direct observation and on the accumulated history of behaviors observed by the informant over time. The SRS generates quantitative scores for the severity of autistic traits and symptoms, and distinguishes Autism Spectrum Disorder (ASD) from other psychiatric conditions (Constantino et al. 2000). Norms have been published by gender and rater type (parent versus teacher) in order to standardize ratings, which otherwise differ as a function of these parameters. SRS scores are highly heritable (Constantino and Todd 2003), stable over time (Constantino et al. 2009), exhibit high inter-rater reliability (Constantino et al. 2007a), are continuously distributed in the general population (Constantino and Todd 2003), are nonsignificantly correlated with IQ among children representing the normal range of IQ in the general population (Constantino et al. 2007a), and exhibit a unitary factor structure (Constantino et al. 2004), which supports the use of a single index score as a quantitative measure of autistic severity. SRS scores greater than 75 *T* (98.8th percentile)

indicate a level of autistic social impairment that is generally highly clinically significant.

Historical Background

Over the course of some 25 studies involving over 10,000 individuals (including Constantino et al. 2006, 2007a, 2007b, 2009; Constantino et al. 2003b; Gruber 2005; Constantino et al. 2003b; Constantino and Todd 2000; Lee et al. 2010; Levitt and Campbell 2009; Pine et al. 2006; Virkud et al. 2009), research involving the SRS has yielded information about the distribution of autistic traits and symptoms in children and families affected by autism, as well as children and families in the general population. Complementary research involving an array of other quantitative rating scales has converged with findings from studies involving the SRS in demonstrating that quantitative autistic traits *exhibit a continuous distribution in nature* (from very mild to very severe, see Fig. 1 panel A below) and are substantially heritable (estimates on the order of 0.60–0.80). The traits and symptoms captured by the SRS are extremely stable over time (5-year test-retest correlations on the order of 0.60–0.70 in clinical samples and 0.70–0.80 in nonclinical populations, as depicted in panel B below), yet capable of fluctuating within individuals over time and in response to intervention. The SRS is currently in use in a broad range of settings, including schools, clinical services, and research programs. It offers the capability of feasible and reliable quantitative characterization of core components of the autistic syndrome across informants and across environmental contexts, and has been translated into over 20 foreign languages at the time of this writing.

Psychometric Data

Each version of the SRS can be completed in 15 min and generates both scale scores for specific symptom domains relevant to the characterization and treatment of autistic syndromes, as well as a singular total score for autistic social impairment,

empirically validated via factor, cluster, and latent class analysis (Constantino et al. 2004, 2007a). Higher total scores on the SRS indicate greater severity of social impairment; and inter-rater reliability is high (parent-teacher correlation 0.72, $n = 1,200$). The SRS exhibits nonsignificant correlations with IQ, and substantial agreement with the Autism Diagnostic Interview (ADI-r) and the Autism Diagnostic Observation Scale (ADOS) (Constantino et al. 2003, Constantino et al. 2004, Constantino et al. 2007a; Lee et al. 2010). Parent-report scores on the SRS distinguish children with ASDs (including Autistic Disorder, Asperger Disorder, and Pervasive Developmental Disorders [PDD-NOS]) from those with other child psychiatric conditions (Constantino et al. 2004; Constantino et al. 2000; Constantino et al. 2007b; Constantino and Gruber 2005). Autistic traits and symptoms measured by the SRS are continuously distributed not only in clinical populations but in the general population (Constantino and Todd 2003). Furthermore, traits measured by the SRS aggregate in the male first-degree relatives of subjects with ASD in multiple-incidence families (Constantino et al. 2006; Virkud et al. 2009). In addition, there is evidence from molecular genetic studies that SRS scores map to specific autism susceptibility loci (Duvall et al. 2007; Levitt and Campbell 2009; Coon et al. 2010).

Internal Consistency

Internal consistency is a form of reliability that can be estimated from a single administration. Studies using the SRS have reported very high estimates. The earliest study on the SRS (Constantino et al. 2000) assessed internal consistency in regular education children aged 4–7 years ($N = 197$). An alpha .97 was obtained. In a report on a clinical sample using the German translation, Bolte et al. (2011) obtained an alpha .94 in a mixed clinical group ($N = 255$) and alpha .96 in an ASD group ($N = 148$).

Retest Reliability/Temporal Stability Construct Validity

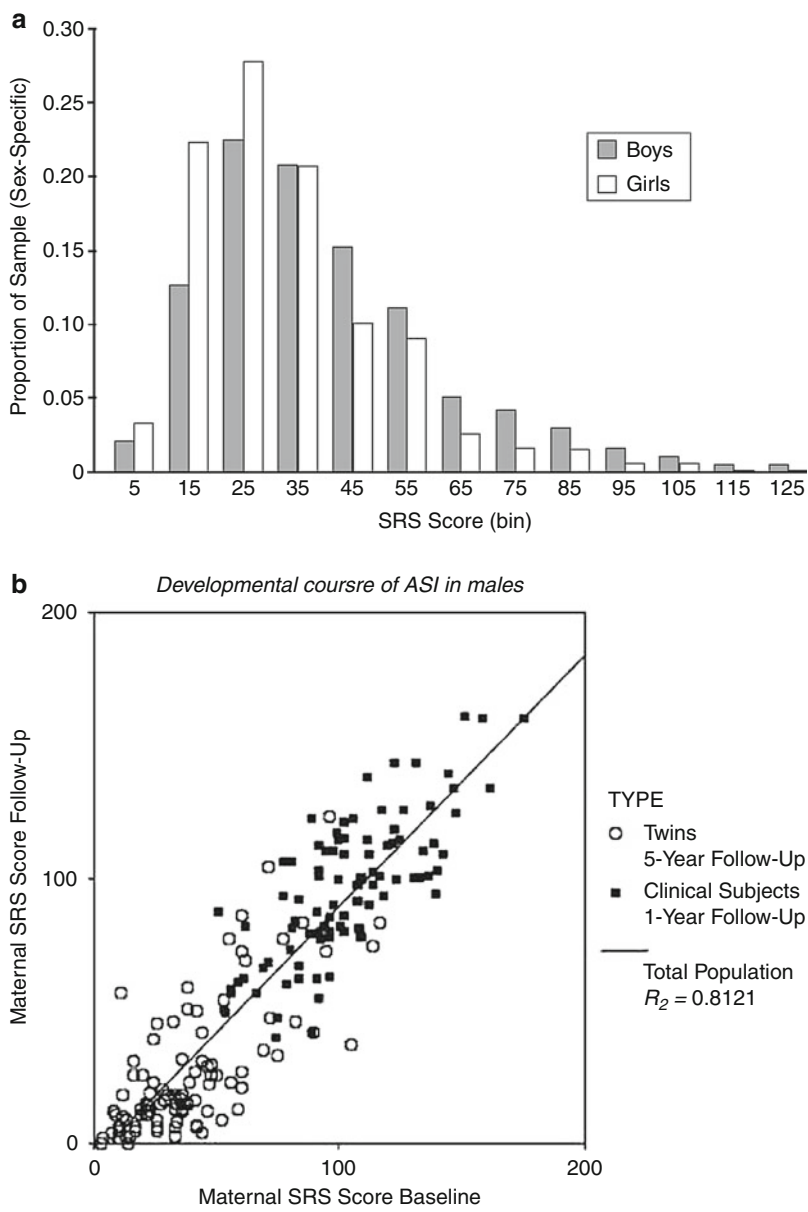
Retest reliability and temporal stability are closely related psychometric constructs. While the actual

study principle is the same – an individual is asked to fill in an instrument twice so that results can be compared after time delay – the meaning of the result changes substantially when the two procedures are separated over increasingly long time periods. When the interval is short, say a few weeks to a few months, the study is said to address test-retest reliability and findings are attributed narrowly to the reliability of the instrument.

When the interval is longer however, say on the order of 6 months to several years, then the study is said to address temporal stability and the results are attributed to the validity of the underlying construct (autism in the case of the SRS) and of the instrument.

Research interest has focused on longer intervals in clinical populations. It is a testimony to both the high validity of the autism construct and

Social Responsiveness Scale, Fig. 1 *School-age SRS studies: (a) General population distribution (b) 5-year Stability.* (Constantino et al. 2009)



to the SRS in measuring that construct that correlations have been very high. Constantino et al. (2000) reported on a clinical group ($N = 30$) with a retest interval averaging 137 days and found a correlation $r = .88$. Bolte et al. (2009) assessed a clinical group ($N = 49$), with a 3–6 month retest interval and found a retest correlation of $r = .95$ was obtained. Constantino et al. (2009) tested 95 male twin pairs ASD affected and 95 males typically developing (total $N = 285$) longitudinally over a 5-year period and found that retest correlations maintained around $r = .90$ in both groups, as shown in Fig. 1 Panel B.

In general, the reliability of an instrument is seen as a strong constraint on its validity. Thus, the evidence of longer interval construct stability above, with correlations on the order of 0.80–0.90, tends to obviate the need to put resources into documenting short interval retest reliability. Bolte et al. (2008) did evaluate retest reliability in conjunction with the development of the German edition, but only in the normative group. With a retest interval ranging from 3 weeks to 4 months, they found retest reliability was $r = .80$ for mothers and $r = .72$ for fathers.

Inter-rater Reliability/Convergent Validity

Inter-rater report comparisons serve two functions. In a narrow sense they are an aspect of reliability, since they involve applying the same items to the same child. Yet inter-rater comparisons also go beyond that to provide a broader aspect of validity, since they involve passing the items through different experience bases. Thus, mother and father, even while living in the same home and sharing many family experiences, do each have separate personal experiences with a child and, moreover, a unique prior personal history that can influence how each will interpret the meaning of the 65 test items. And this sense of difference in background, experience, and perspective expands even more when a parent report is compared to that of a teacher; for one a teacher and a parent have virtually no overlapping observations of a child's behavior and for another a teacher, while seeing the child in a more limited setting than the parent, can draw on professional training and direct experience with scores and

even hundreds of other children when judging the child's behavior.

Despite these differences in perspective, studies reporting on different observers have found highly concordant reports. With regard to mother-father inter-rater agreement, Constantino et al. (2003a) collected mother and father reports in a sample of ASD-diagnosed individuals ($N = 61$). They found mother-father reports correlated $r = .91$. Bolte et al. (2008), using the German edition, reported on 527 mixed clinical cases (160 with ASDs) and also found mother-father reports correlated $r = .91$. In the normative sample, Bolte et al. (2009) obtained a mother-father correlation $r = .61$. Prior comments regarding results in normative or other less-affected groups apply again here.

Parent reports have also been compared to teacher reports in clinical samples. Regarding parent-teacher comparisons, Constantino et al. (2003a) found mother-teacher reports correlating $r = .82$ and father-teacher reports correlating $r = .75$ (both $N = 61$). In a later study Constantino et al. (2007a) compared teacher- and parent-report SRS scores in a larger sample ($N = 577$) of clinically referred children and found parent-teacher reports correlated $r = .72$. As noted in the introductory comments, differences in experience bases are much larger between teachers and parents than between parents. These finding on parent-teacher comparisons are also highly indicative of strong reliability and validity.

Comparisons with Other Instruments: Concurrent Validation

Two broad issues can be addressed from reports where the SRS has been used with other instruments. Of these, *convergent validity* compares the instrument to others that have the same focal purpose. For the SRS, this involves comparisons with other instruments that are designed to evaluate symptoms and behaviors on the Autism Spectrum. In these comparisons, higher correlations are desirable. In contrast, *divergent/discriminant validity* addresses the instrument's performance in contexts where a broader variety of symptoms and behavior problems need to be addressed. For the SRS, this could involve comparison to

instruments that assess other commonly identified psychological disorders and behavioral problems – for example, behavioral expressions of internalizing problems of anxiety, depression, and Obsessive Compulsive Disorder (OCD) or behavioral expressions of externalizing problems such as Attention Deficit Hyperactivity Disorder (ADHD), conduct disorders, and Bipolar disorder – or those that assess less specifically linked problems such as developmental delay. In these comparisons, moderate- to lower- or even zero-order correlations are desirable, demonstrating that the SRS explains variance that is unique to the social behavior associated with ASDs and not strongly overlapping with that due to behavioral difficulties associated with other disorders and identified by other instruments.

Convergent Validity

Parent and Teacher Report Behavior Assessments. Comparison with other ASD-directed measures have largely involved instruments using a similar approach to assessment: multiple item objective questionnaires with a multipoint Likert-scale or a true-false response format that is completed by informants familiar with the child's behavior in natural living contexts such as parents and teachers. Good agreement has been reported with the several instruments that have been investigated.

The most widely investigated comparison has been with the Social Communication Questionnaire (SCQ; Rutter et al. 2003), an instrument that uses a true-false response format with 40 items derived from the ADI-r. Findings with the SRS have been relatively consistent. In studies with mixed clinical samples as cited earlier, Charman et al. (2007) report a correlation $r = .68$ ($n = 119$); Bolte et al. (2008) report $r = .58$ ($n = 107$). In addition, also in a mixed clinical sample, (Pine et al. 2008) found $r = .65$ ($n = 352$) and in a small sample of children affected by epilepsy Granader et al. (2010) report $r = .61$ ($n = 21$). A slightly lower value was reported by Bolte et al. (2011), $r = .50$ ($n = 480$), in a sample that included typically developing as well as clinical subjects.

There have also been several studies correlating SRS scores with those from the Children's

Communication Checklist (CCC; Bishop 1998), a 70-item instrument with a Likert scale response format. Pine et al. (2008) reports correlations or $r = -.49$ and $-.72$ with the two ASD-focused (and positively valanced) subscales. Similarly, Charman et al. (2007) report a SRS to CCC correlation $r = -.75$.

The SRS has also been compared to the Social and Communication Disorders Checklist (SCDC, (Skuse et al. 2005)) a brief, 12-item scale using a 3-point Likert scale response format. Using German translations, Bolte et al. (2011) found the instruments moderately correlated $r = .49$.

The ADI-r and the ADOS. The SRS has also been compared to instruments that are less similar in design, in particular the Autism Diagnostic Interview, revised (► ADI-r; Lord et al. 1994) and the Autism Diagnostic Observation Schedule (► ADOS; Lord et al. 2000).

The design characteristics of these instruments are quite different from that of the SRS and other behavioral parent and teacher reports cited in the previous section. The ADI-r is a long structured psychiatric interview from which a subset of questions are selected and coded; the ADOS is a structured observation of behavior conducted by a trained professional under standard conditions from which pre-identified behavior is identified and coded. Both instruments were designed to facilitate categorical, diagnostic decision making and, consequently, do not produce the same kind of dimensional results produced by the objective questionnaires. It has long been recognized that differences in method tend to produce evidence of validity at lower levels of correlation (e.g., Campbell and Fiske 1959). Given the differences, results provided clear support for the validity of the SRS.

ADI-r results include “domain scores,” sums of coded item subsets. Constantino et al. (2003a) reported an early investigation of SRS validity, comparing SRS to ADI-r lifetime scores in a sample ASD-diagnosed individuals ($N = 61$). We note that ADI-r domain scores characterize level of severity around the age of 4 years, at a relative peak in manifestations of autistic symptomatology from the standpoint of developmental history. Despite this fundamental difference between the

SRS (which measures current dysfunction) and the ADI-r (which indexes historic symptomatology in early childhood), correlations between parent-report SRS scores and ADI-r domain scores ranged from .65 to .77 for mother reports, from .52 to .70 for teacher reports, and from .60 to .74 for father reports. In a subsequent report on a larger sample, somewhat lower, though highly statistically significant correlations were reported between SRS Parent reports and ADI-r domain scores (range of $r = .31-.36$) and SRS Teacher reports (range of $r = .26-.40$) (Constantino et al. 2007a). These correlations were on the order of what was observed for correlations between ADI-r and ADOS domain scores. In the study associated with the development of the German edition, Bolte et al. (2008) collected 133 clinical cases and report more specifically on parent reports for the separate domains: Social Interaction Domain score $r = .46$, Communication Domain Score $r = .40$, Stereotypic Behavior Domain score $r = .38$. Charman et al. (2007) used an ADI-r results that combined scores across domains and found a correlation $r = .59$. As noted in the introductory paragraph, the ADI-r was designed to facilitate categorical analyses and its brief domain scores do not provide a strong basis for correlational analyses, for example, Bolte et al. (2009) used standard adjustments to compensate for attenuated ranges in short scales. The relatively stronger finding in the Charman et al. (2007) study may in part reflect the combination of domains into a single, more dimensionalized score.

The same sets of investigators produced parallel findings on comparisons to the ADOS. Constantino et al. (2007a) found ADOS domain scores correlated with SRS Parent reports (range of $r = .37$ to $.58$) and SRS Teacher reports (range of $r = .15-.43$). The Bolte et al. (2008) study reports Communications/Social Deficits score $r = .35$. In a subsequent study Bolte et al. (2011) found correlations in the $r = .32-.35$ range with the ADOS Domain scores. Again, using a single combined domain score, Charman et al. (2007) found an SRS to ADOS correlation $r = .48$. The earlier comments regarding brief domain scores

and Charman et al.'s use of a combined domain score also apply here.

Divergent/Discriminant Validity

The task of differentiating diagnostic groups is addressed by studies cited in a later section. This section provides a more general sense of overall validity from comparing correlations with different instruments and their scales. The comparison scales reported here are directed at a wider variety of mental health problems and diagnoses, some of which may have overlapping symptoms with ASD and others thought or known to have no systematic relation to ASDs. Reported correlations will reflect a validation of the SRS by showing moderate- to low- or zero-order correlations.

As noted earlier, differences in methods can have an impact on correlational evidence (e.g., Campbell and Fiske 1959). Most of the reports in the following discussion involve parent and teacher behavioral reports, similar in design to convergent measures like the SCQ, CCC, and SCDC. The appropriate comparison in this section is with those reported on the parent and teacher report behavioral measures in the previous section.

Studies using the Child Behavior Checklist (CBCL; [Achenbach and Ruffle 2000]) support the view that the SRS is more sensitive to behaviors that can sometimes be associated with ASD-related problems, less sensitive to behavior seldom seen in ASDs, and – perhaps most critically – sufficiently independent of the CBCL to indicate sensitivity to behavioral problems not assessed by the CBCL. Two studies report correlations between the SRS and CBCL in clinical samples, with Constantino et al. (2000) reporting on 84 clinical cases and Bolte et al. (2008) reporting on 119 clinical cases from the German validation studies. Despite the differences in location and language, the findings were quite parallel. Both studies found moderate correlations for the SRS with CBCL subscales that have some overlap with the kinds of symptoms seen with ASDs: Social Problems, Thought Problems, and Attention Problems (correlations range $r = .48$ to $.64$). As expected, results were somewhat mixed but

generally lower for correlation with CBCL subscales directed at less related behavior: Withdrawn, Delinquent Behavior, and Aggressive Behavior (correlations range $r = .34-.54$). Both studies found zero-order correlations for the SRS with the CBCL Somatic Complaints subscale ($r = .11$ and $.12$ ns). In another study involving a general population twin sample, Constantino et al. (2003b) examined overlap in the constructs captured by the scales, suggesting about 16% total shared variance.

Using the Vineland Adaptive Behavior Scales (VABS; Sparrow et al. 1984) studies have reported on the relation of development to SRS scores. Charman et al. (2007) report a correlation of $-.44$ for the positively valenced composite VABS scores with the SRS and Bolte et al. (2008) found a correlation $r = -.36$ for the composite correlations ranging from $-.34$ to $-.43$ for the subscales. This level of correlation appears reasonable for a developmental disorder like ASD. Regarding narrower cognitive ability, Charman et al. (2007) reported no significant correlation of the SRS to the British Picture Vocabulary Scale (BPVS; Dunn et al. 1997).

Clinical Uses

The first question for a clinical assessment is whether affected individuals show scores elevated enough for the assessment to have clinical utility. Independent reports providing information on both typically developing children with those diagnosed as autistic have consistently shown a statistically significant and clinically meaningful separation. Children not affected by ASD or any other disorder are typically reported to have SRS Total Scores in the narrow range from 0 to 35. This is true in studies where children have been drawn to be representative of typically developing populations (Bolte et al. 2008, 2011; Coon et al. 2010) and in studies where matched controls have been drawn (Reiersen et al. 2007; Pine et al. 2008). In contrast, results for groups of children with PDD-NOS, ASD, or autistic disorder find SRS Total Score group means in the range

86–116 (Charman et al. 2007; Constantino et al. 2000; Coon et al. 2010; Kalb et al. 2010). Even given the rather large standard deviations reported for autistic groups (ranging 27–33), the findings indicate a separation of two standard deviations or more. These differences are substantial and have a clear practical utility.

Clear and useful separation can also be seen when contrasting autism with other non-autistic diagnoses. Studies that have presented SRS scores for mixed or specific non-ASD diagnosis clinical samples have reported group SRS Total Score means in the range 40–75, that is, consistently higher than those reported for typically developing groups and lower than those reported for autism-affected groups (Bolte et al. 2008, 2011; Charman et al. 2007; Constantino et al. 2000; Pine et al. 2008; Puleo and Kendall 2011; Reiersen et al. 2007; Towbin et al. 2005). It must be noted that studies will produce different results depending on the specific symptoms associated with the contrast diagnosis and on the severity of clinically affected subjects who are ascertained or recruited.

Screening and Receiver Operating Characteristics (ROC) Reports

The SRS has had a number of clinical applications: qualifying subjects for research studies, providing support for clinical diagnoses, assessment of treatment effects, etc. Wide international concern about autism, however, has also led to an interest in developing and applying screening tools, and the SRS has been studied for this purpose. These studies evaluate an instrument's sensitivity (the proportion of actually affected individuals who are correctly identified) and specificity (the proportion of nonaffected individuals who are correctly identified). In general, there is a trade-off so that increasing specificity degrades sensitivity and vice versa.

The studies reported here reflect the complexity of the screening task (e.g., differing contrast diagnoses, population pools, comparison instruments, and cut points that may favor sensitivity in some cases and specificity in others). Even with the variation, however, the findings to be reported

indicate that the SRS has a useful power to properly identify affected children and to accurately discriminate children with ASD conditions from typically developing children and also from those with varied non-ASD disorders. For a psychiatric sample of 133 children with ASD diagnoses and 126 mixed non-ASD diagnoses (sample described in Constantino et al. (2004)), a ROC analysis was reported. With the standard recommended research cutoff of 75, sensitivity was reported as .85 and specificity as .75. Charman et al. (2007) reported on a sample of children previously identified with developmental or special education risk factors, and contrasted 49 children with confirmed ASD diagnoses with 70 children with confirmed non-ASD diagnoses. Under these conditions, Area Under Curve (AUC) was .77, sensitivity was .78, and specificity was .67.

A larger and more diverse study by using the German translation involved a sample of 480 children, including 148 with ASD diagnoses, 255 with non-ASD clinical diagnoses, and 77 typically developing children (Bolte et al. 2011). Results were reported based on the recommended research cutoff for the SRS of 75 that was used in the prior two reports. Contrasting ASD cases with typically developing children they report $AUC = .98$ with sensitivity = .80, specificity = 1.0. Contrasting ASD cases with the non-ASD clinical group, they report $AUC = .81$ with sensitivity = .80, specificity = .69. Contrasting ASD with a clinical sub-sample of ADHD affected children, they report $AUC = .86$ with sensitivity = .80, specificity = .78. And finally, contrasting ASD with a clinical sub-sample of children with anxiety diagnoses, they report $AUC = .82$ with sensitivity = .81, specificity = .74.

The findings across the three studies and groups are fairly consistent with regard to sensitivity, ranging from .78 to .85 and clustering around .80. With regard to specificity, findings are more varied and depend on the nature of the contrast group, ranging from .69 to 1.0, with perhaps .75 as an adequate single estimate in clinical settings where there are mixed diagnoses in the contrast group. Co-equal high rates, as seen for the SRS, are generally desirable, but there can be applications where favoring one or the other

many be preferred, for example, an instrument with higher sensitivity for use in early, broad population studies or one with higher specificity for use in aiding differential clinical diagnosis. The SRS is highly versatile in this sense; given the fact that it is fundamentally a quantitative trait measure, users can adjust the cutoff values used for screening to optimize sensitivity or positive predictive value.

The results above are reported on the conventional procedure, where a single administration of an instrument is used to identify children with potential problems. The SRS has two features that make it relatively easy to add to screening power. As a behavioral report, it does not require clinician time to conduct the assessment and as a report with several forms, it is relatively easy to collect more than one score on a child, for example, a parent and a teacher or a mother and a father. Taking advantage of these features, Constantino et al. (2007a) compared teacher- and parent-report SRS scores in a large sample ($N = 577$) of clinically referred children. A PDD-affected sample ($n = 271$) was compared to nonaffected siblings ($N = 119$). Parent and teacher forms were both administered and the screening power reported when both reports on a given child were elevated to the diagnostic cut point. Under these conditions the ROC analyses showed an $AUC = .95$ with sensitivity 0.75 and specificity 0.96.

Results in Other Clinical and Behavioral Research

While the SRS has the identification and characterization of ASDs as its central purpose, it has been used in studies of other behavior difficulties and clinical diagnoses. In all of the studies reported below, children with non-ASD clinical diagnoses are seen to have clinically relevant weakness in the kinds of social behavior assessed by the SRS. As noted by one of the teams (Pine et al. 2008), this can raise the question of whether ASDs are being under-assessed when there are comorbid conditions. More broadly, however, all of the findings show that the behavioral and social problems associated with ASD can have relevance in other areas. The SRS measures these problems in a way that is sensitive to their

presence, differentiated in elevation from that seen in actual ASD diagnoses, and may be highly sensitive to treatment effects (Puleo and Kendall 2011).

Heritability/Genetic Epidemiology

In an extensive line of research employing the SRS in large genetically informative samples of twins (Constantino and Todd 2000, 2005; Ho et al. 2005), siblings (Constantino et al. 2006; Schwichtenberg et al. 2010), and families (Virkud et al. 2009), representing both the general population Constantino and Todd (2003) and clinically affected families (Constantino et al. 2010), including studies involving molecular genetic markers (Duvall et al. 2007; Campbell et al. 2010; Coon et al. 2010), it has been shown that the quantitative traits measured by the SRS are highly heritable across the entire range of severity in which they occur in nature (from mild to severe), and that subclinical autistic traits characterized by mild elevations SRS scores constitute candidate endophenotypes, that is, genetically related to the cause(s) of autism itself.

Brain Imaging

Several neuroimaging studies have elucidated brain imaging phenotypes that relate closely to variation in SRS scores, both in the general population and in clinically affected samples (Assaf et al. 2010; Di Martino et al. 2009; Kaiser et al. 2010; Paul et al. 2010).

Longitudinal Course

Constantino et al. (2009) tested $N = 285$ (95 male twin pairs ASD affected and 95 male typical developing) longitudinally over a 5-year period, and found that test retest correlations maintained around $r = .90$ in both groups. Individual trajectories varied as a function of severity at baseline. Moderate improvement with age was noted, underscoring the instrument's potential utility for ascertaining incremental response to intervention. Ongoing research on the longer-term longitudinal course of autistic traits and symptoms among clinically affected children and their siblings is underway in NIH-funded research.

See Also

- ▶ Attention Deficit/Hyperactivity Disorder
- ▶ Obsessive-Compulsive Disorder (OCD)
- ▶ Pervasive Developmental Disorders
- ▶ Vineland Adaptive Behavior Scales (VABS)

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References and Reading

Suggested Reading

- Achenbach, T. M., & Ruffle, T. M. (2000). The child behavior checklist and related forms for assessing behavioral/emotional problems and competencies. *Pediatric Review, 21*(8), 265–271.
- Assaf, M., Jagannathan, K., Calhoun, V. D., Miller, L., Stevens, M. C., Sahl, R., et al. (2010). Abnormal functional connectivity of default mode sub-networks in autism spectrum disorder patients. *NeuroImage, 53*(1), 247–256.
- Bishop, D. V. (1998). Development of the children's communication checklist (CCC): A method for assessing qualitative aspects of communicative impairment in children. *Journal of Child Psychology and Psychiatry, 39*(6), 879–891.
- Bolte, S., Dziobek, I., & Poustka, F. (2009). Brief report: The level and nature of autistic intelligence revisited. *Journal of Autism and Developmental Disorders, 39*(4), 678–682.
- Bolte, S., Poustka, F., & Constantino, J. N. (2008). Assessing autistic traits: Cross-cultural validation of the social responsiveness scale (SRS). *Autism Research, 1*(6), 354–363.
- Bolte, S., Westerwald, E., Holtmann, M., Freitag, C., & Poustka, F. (2011). Autistic traits and autism spectrum disorders: The clinical validity of two measures presuming a continuum of social communication skills. *Journal of Autism and Developmental Disorders, 41*(1), 66–72.
- Campbell, D. T., & Fiske, D. W. (1959). Convergent and discriminant validation by the multitrait-multimethod matrix. *Psychological Bulletin, 56*(2), 81–105.
- Campbell, D. B., Warren, D., Sutcliffe, J. S., Lee, E. B., & Levitt, P. (2010). Association of MET with social and communication phenotypes in individuals with autism spectrum disorder. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 153B*(2), 438–446.
- Charman, T., Baird, G., Simonoff, E., Loucas, T., Chandler, S., Meldrum, D., et al. (2007). Efficacy of three screening instruments in the identification of autistic-

- spectrum disorders. *The British Journal of Psychiatry*, 191, 554–559.
- Constantino, J. N. (2011). The quantitative nature of autistic social impairment. *Pediatric Research*, 69(5Pt2), 55R–62R.
- Constantino, J. N., Abbacchi, A. M., Lavesser, P. D., Reed, H., Givens, L., Chiang, L., et al. (2009). Developmental course of autistic social impairment in males. *Development and Psychopathology*, 21(1), 127–138.
- Constantino, J. N., Davis, S. A., Todd, R. D., Schindler, M. K., Gross, M. M., Brophy, S. L., et al. (2003a). Validation of a brief quantitative measure of autistic traits: Comparison of the social responsiveness scale with the autism diagnostic interview-revised. *Journal of Autism Developmental Disorders*, 33(4), 427–433.
- Constantino, J., & Gruber, C. P. (2005). *The social responsiveness scale (SRS) manual*. Los Angeles: Western Psychological Services.
- Constantino, J. N., Gruber, C. P., Davis, S., Hayes, S., Passanante, N., & Przybeck, T. (2004). The factor structure of autistic traits. *Journal of Child Psychology and Psychiatry*, 45(4), 719–726.
- Constantino, J. N., Hudziak, J. J., & Todd, R. D. (2003b). Deficits in reciprocal social behavior in male twins: Evidence for a genetically independent domain of psychopathology. *Journal of the American Academy of Child and Adolescent Psychiatry*, 42(4), 458–467.
- Constantino, J. N., Lajonchere, C., Lutz, M., Gray, T., Abbacchi, A., et al. (2006). Autistic social impairment in the siblings of children with pervasive developmental disorders. *The American Journal of Psychiatry*, 163(2), 294–296.
- Constantino, J. N., Lavesser, P. D., Zhang, Y., Abbacchi, A. M., Gray, T., & Todd, R. D. (2007a). Rapid quantitative assessment of autistic social impairment by classroom teachers. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46(12), 1668–1676.
- Constantino, J. N., Przybeck, T., Friesen, D., & Todd, R. D. (2000). Reciprocal social behavior in children with and without pervasive developmental disorders. *Journal of Developmental Behavior Pediatrics*, 21(1), 2–11.
- Constantino, J. N., & Todd, R. D. (2000). Genetic structure of reciprocal social behavior. *The American Journal of Psychiatry*, 157(12), 2043–2045.
- Constantino, J. N., & Todd, R. D. (2003). Autistic traits in the general population: A twin study. *Archives of General Psychiatry*, 60(5), 524–530.
- Constantino, J. N., & Todd, R. D. (2005). Intergenerational transmission of subthreshold autistic traits in the general population. *Biological Psychiatry*, 57(6), 655–660.
- Constantino, J. N., Yang, D., Gray, T. L., Gross, M. M., Abbacchi, A. M., Smith, S. C., et al. (2007b). Clarifying the associations between language and social development in autism: A study of non-native phoneme recognition. *Journal of Autism and Developmental Disorders*, 37(7), 1256–1263.
- Constantino, J. N., Zhang, Y., Frazier, T., Abbacchi, A. M., & Law, P. (2010). Sibling recurrence and the genetic epidemiology of autism. *The American Journal of Psychiatry*, 167(11), 1349–1356.
- Coon, H., Villalobos, M. E., Robison, R. J., Camp, N. J., Cannon, D. S., Allen-Brady, K., et al. (2010). Genome-wide linkage using the social responsiveness scale in Utah autism pedigrees. *Molecular Autism*, 1(1), 8.
- Di Martino, A., Shehzad, Z., Kelly, C., Roy, A. K., Gee, D. G., Uddin, L. Q., et al. (2009). Relationship between cingulo-insular functional connectivity and autistic traits in neurotypical adults. *The American Journal of Psychiatry*, 166(8), 891–899.
- Dunn, L. M., Dunn, L. M., Whetton, C., & Burley, J. (1997). *British Picture Vocabulary Scale* (2nd ed.). Windsor/Berks: NFER-Nelson.
- Duvall, J. A., Lu, A., Cantor, R. M., Todd, R. D., Constantino, J. N., & Geschwind, D. H. (2007). A quantitative trait locus analysis of social responsiveness in multiplex autism families. *The American Journal of Psychiatry*, 164(4), 656–662.
- Granader, Y. E., Bender, H. A., Zemon, V., Rath, S., Nass, R., & Macallister, W. S. (2010). The clinical utility of the social responsiveness scale and social communication questionnaire in tuberous sclerosis complex. *Epilepsy & Behavior*, 18(3), 262–266.
- Ho, A., Todd, R. D., & Constantino, J. N. (2005). Brief report: Autistic traits in twins vs. non-twins – a preliminary study. *Journal of Autism Developmental Disorders*, 35(1), 129–133.
- Kaiser, M. D., Hudac, C. M., Shultz, S., Lee, S. M., Cheung, C., Berken, A. M., et al. (2010). Neural signatures of autism. *Proceedings of the National Academy of Science USA*, 107(49), 21223–21228.
- Kalb, L. G., Law, J. K., Landa, R., & Law, P. A. (2010). Onset patterns prior to 36 months in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(11), 1389–1402.
- Lee, H., Marvin, A. R., Watson, T., Piggot, J., Law, J. K., Law, P. A., et al. (2010). Accuracy of phenotyping of autistic children based on internet implemented parent report. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 153B(6), 1119–1126.
- Levitt, P., & Campbell, D. B. (2009). The genetic and neurobiologic compass points toward common signaling dysfunctions in autism spectrum disorders. *Journal of Clinical Investigation*, 119(4), 747–754.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., DiLavore, P. C., et al. (2000). The autism diagnostic observation schedule-generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30(3), 205–223.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(5), 659–685.
- Paul, L. K., Corsello, C., Tranel, D., & Adolphs, R. (2010). Does bilateral damage to the human amygdala produce

autistic symptoms? *Journal of Neurodevelopmental Disorders*, 2(3), 165–173.

- Pine, D. S., Guyer, A. E., Goldwin, M., Towbin, K. A., & Leibenluft, E. (2008). Autism spectrum disorder scale scores in pediatric mood and anxiety disorders. *Journal of American Academy of Child and Adolescent Psychiatry*, 47(6), 652–661.
- Pine, E., Luby, J., Abbacchi, A., & Constantino, J. N. (2006). Quantitative assessment of autistic symptomatology in preschoolers. *Autism*, 10(4), 344–352.
- Puleo, C. M., & Kendall, P. C. (2011). Anxiety disorders in typically developing youth: autism spectrum symptoms as a predictor of cognitive-behavioral treatment. *Journal of Autism and Developmental Disorders*, 41(3), 275–286.
- Reiersen, A. M., Constantino, J. N., Volk, H. E., & Todd, R. D. (2007). Autistic traits in a population-based ADHD twin sample. *Journal of Child Psychology and Psychiatry*, 48(5), 464–472.
- Rutter, M., Bailey, A. J., Berument, S. K., LeCouteur, A., Lord, C., & Pickles, A. (2003). *Social communication questionnaire (SCQ)*. Los Angeles: Western Psychological Services.
- Schwichtenberg, A. J., Young, G. S., Sigman, M., Hutman, T., & Ozonoff, S. (2010). Can family affectedness inform infant sibling outcomes of autism spectrum disorders? *Journal of Child Psychology and Psychiatry*, 51(9), 1021–1030.
- Skuse, D. H., Mandy, W. P., & Scourfield, J. (2005). Measuring autistic traits: Heritability, reliability and validity of the social and communication disorders checklist. *The British Journal of Psychiatry*, 187, 568–572.
- Sparrow, S., Balla, D., & Cicchetti, D. (1984). *Vineland adaptive behavior scales*. Circle Pines: American Guidance Service.
- Towbin, K. E., Pradella, A., Gorrindo, T., Pine, D. S., & Leibenluft, E. (2005). Autism spectrum traits in children with mood and anxiety disorders. *Journal of Child and Adolescent Psychopharmacology*, 15(3), 452–464.
- Virkud, Y. V., Todd, R. D., Abbacchi, A. M., Zhang, Y., & Constantino, J. N. (2009). Familial aggregation of quantitative autistic traits in multiplex versus simplex autism. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 150B(3), 328–334.

Social Scientist

► Psychologist

Social Scripts

► Scripts

Social Security Amendments of 1965 (or “Medicare Act of 1965” and/or the “Medicaid Act of 1965”)

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Definition

The Social Security Amendments of 1965 (the “Amendments”) are the milestone laws that established the U.S. Medicare and Medicaid programs (Pub. L. No. 89–97). Collectively, these federal statutes have four major provisions: (1) a hospital insurance program for qualifying adults aged 65 and older (Medicare Part A, Title XVIII); (2) a medical insurance program to offset out-of-pocket healthcare costs for qualifying adults aged 65 and older (Medicare Part B, Title XVIII); (3) increased government benefits through the Social Security program (formerly called the “Old-Age, Survivors, and Disability Insurance” system in Medicare Part B, Title XVIII); and (4) improved the federal-state public assistance programs for qualifying adults with low incomes (Medicaid, Title XIX).

Congress’ expansion of the Social Security Act with the Amendments of 1965 marked a pivotal moment in U.S. healthcare policy. Federal Medicare and state Medicaid systems have evolved over time based on the legal foundations set forth in 1965 (Martin and Weaver 2005). Millions of Americans have received government benefits from one or both of the programs since their inception.

Individuals with autism spectrum disorder (ASD) and their caregivers should be knowledgeable about the government-sponsored benefits that are available under the Amendments of 1965 and their legal precedent (Bowen 2014).

Medicare Legislative Details

The Amendments of 1965 added Title XVIII (18) to the Social Security Act to create the first regulations for the Medicare program (Cohen and Ball 1965). Medicare helps qualified individuals to access healthcare services. In its first iteration, Medicare was a welfare program that only served adults age 65 and older who received government financial aid. Over time, federal lawmakers have expanded the Amendments of 1965 so that Medicare also covers individuals with disabilities of all ages (as defined by Social Security Administration (SSA) rules) as well as individuals with end-stage renal disease (DeWitt 2010).

Medicare is administered by the Centers for Medicare & Medicaid Services (CMS), which is a government agency within the U.S. Department of Health & Human Services (DHHS). Since 1965, Congress has worked with CMS to provide additional benefits (DHHS CMS 2020a). The first version of Medicare included part A for hospital insurance and part B for medical insurance. These two parts are often referred to as “Original Medicare.” Today, Medicare services are divided into four primary areas: part A for inpatient/hospital coverage, part B for outpatient/medical coverage, part C for supplemental healthcare benefits, and part D for prescription drug coverage.

Per the Amendments of 1965, Medicare coverage is limited to items and services that are medically “reasonable and necessary” for the diagnosis or treatment of an individual’s illness or injury. Because healthcare technology, medical services, and healthcare economics are continuously evolving, CMS updates its guidelines regularly to help providers and Medicare beneficiaries navigate program requirements.

Medicaid Legislative Details

Title XIX (19) in the Amendments of 1965 established the first regulations for the Medicaid program. Medicaid provides state governments with the opportunity to receive federal funds for a portion of the services that it provides to people with limited incomes (DHHS CMS 2020b).

Whereas Medicare is a federal program, Medicaid programs are largely administered by their respective state governments. Under the Amendments of 1965, each Medicaid program is jointly funded by both federal and state budgets. The amount of money that each state receives from the federal government for its Medicaid program varies under Federal Medical Assistance Percentage (FMAP) rules (Mitchell 2018).

Each state’s Medicaid system must comply with federal statutes and administrative regulations from CMS. These federal rules set the minimum criteria for enrollee qualifications, billing procedures, and benefit categories. The nature and extent of Medicaid services varies widely between state programs. As long as they comply with federal legal standards as a baseline, state Medicaid agencies can design and implement additional procedures and policies to best serve their residents.

Legislative Details for Other Key Provisions

Titles XIX and XVII created several policies that protect the sensitive health information of program enrollees. These laws were among the first of their kind to govern collecting, using, and disclosing personal health information. The US lawmakers have used the guidelines from the Amendments of 1965 to create other milestone laws that safeguard medical records, such as the Health Insurance Portability and Accountability Act of 1996 (“HIPAA,” Pub. L. No. 104–191).

The 1965 Amendments also enacted new legal requirements for Social Security Disability Insurance (SSDI). Whenever a person is able to collect workers’ compensation payments, SSDI benefits must be reduced in accordance with SSA guidelines (Reno et al. 2005). Under the 1965 Amendments, a person’s total earnings from SSDI and workers compensation cannot result in a reduced income – the total amount of reduced SSDI and workers’ compensation payments can never be less than what the individual received in SSDI before he or she became eligible for workers compensation. These policies are often referred to as

the “workers’ compensation offset” for SSDI payments.

Legislative History and Background

The 1965 Amendments’ provisions were heavily debated in the 89th Congress (U.S. Social Security Administration [n.d.](#)). The House of Representatives approved the Amendments on April 8, 1965. On July 9, 1965, it was approved in the Senate. President Lyndon B. Johnson signed the Amendments into law on July 30, 1965.

The momentous Medicare and Medicaid statutes were the result of years of national deliberation about the need for government-sponsored healthcare coverage programs. In passing these Amendments in 1965, federal lawmakers recognized that a significant population of Americans could not access medical goods and services because they could not obtain health insurance from private providers. Specifically, expert reports to 89th Congress concluded that two key issues prevented many individuals from accessing private health insurance: either they could not afford to make out-of-pocket payments to a private insurer or they were unable to work and obtain employer-sponsored health coverage (Berkowitz [2005](#)).

Findings from the 1950 American Census were one of the driving forces behind the Amendments of 1965. Statistics in the 1950 Census established that the United States had a steadily aging population with individuals living longer lifespans than ever before (U.S. Department of Commerce Bureau of the Census [1975](#)). At the same time, the census also found that the majority of adults age 65 and older, as well as individuals with disabilities, were either uninsured or had only minimal health insurance coverage (often referred to as “underinsured”) (U.S. National Archives and Records Administration [2020](#)).

Since 1965, a large body of health law and policy scholars have praised Medicare and Medicaid while recommending program expansions (Moore and Smith [2005](#)). At the same time, a

different cohort of scholars contend that the systems’ financing structures are overly broad, among other concerns. Though Medicare and Medicaid policies have advanced since their establishment in 1965, many of the fundamental program goals and baseline benefits remain the same today. Lawmakers have expanded on the foundational ideas in the 1965 Amendments to provide more Americans with access to healthcare through Medicaid and Medicare. Over time, Congress has also increased the types of services available through the programs. In 1973, Congress made a ground-breaking modification to the Amendments of 1965 that allows people of all ages with long-term disabilities to access Medicare.

Over the years, judicial court opinions from the U.S. Supreme Court and case decisions from other lower courts have established guidance for interpreting the 1965 Amendments’ various provisions (often called “legal precedent”). Federal and state administrative rules – typically from CMS and state health departments – also provide legal insight about Medicare and Medicaid program requirements in specific circumstances. This information is available online in the form of agency regulations, registers, manuals, and memoranda (e.g., DHHS CMS [2020c](#)) and State of Michigan DHHS [2020](#)).

Though Congress and government agencies have made numerous modern developments to Medicare and Medicaid laws, many of the central provisions from the 1965 Amendments are still in use today – these include the goal to reduce barriers to medical care for America’s most economically vulnerable citizens.

See Also

- ▶ [Americans with Disabilities Act](#)
- ▶ [Autism Collaboration, Accountability, Research, Education, and Support \(CARES\) Act of 2019 \(Also Referred to as the “Autism CARES Act of 2019”\)](#)
- ▶ [Civil Rights Act of 1964](#)
- ▶ [Confidentiality](#)
- ▶ [Medicaid and Autism Spectrum Disorder](#)

References and Reading

- Berkowitz, E. (2005). Medicare and Medicaid: The past as prologue. *Health Care Financing Review*, 27(2), 11–23. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4194925/>
- Bowen, S. (2014). *Autism spectrum disorders (ASD): State of the states of services and supports for people with ASD*. L & M Policy Research. Report No. HHSM-500-2006-000091/HHSM-500-T0002. Retrieved from <https://autismsocietyofhawaii.org/wp-content/uploads/2015/06/asd-state-of-the-states-report.pdf>
- Cohen, W. & Ball, R. (1965). Bulletin: Social security amendments of 1965: Summary and legislative history. *Social Security Bulletin*, 28(9), 3–21. Retrieved from <https://www.ssa.gov/policy/docs/ssb/v28n9/v28n9p3.pdf>
- DeWitt, L. (2010). The development of social security in America. *Social Security Bulletin*, 70(1). Retrieved from <https://www.ssa.gov/policy/docs/ssb/v70n3/v70n3p1.html#:~:text=The%20First%20Social%20Security%20Payments,to%20qualify%20for%20monthly%20benefits>
- Health Insurance Portability and Accountability Act of 1996, Pub. L. No. 104–191, 110 Stat. 1936, codified as amended at 42 U.S.C. § 1320d *et seq.* (1996). Retrieved from <https://www.govinfo.gov/content/pkg/PLAW-104publ191/pdf/PLAW-104publ191.pdf>
- Martin, P., & Weaver, D. (2005). Social security: A program and policy history. *Security Bulletin*, 66(1). <https://www.ssa.gov/policy/docs/ssb/v66n1/v66n1p1.pdf>
- Mitchell, A. (2018). *Medicaid's Federal Medical Assistance Percentage (FMAP)*. U.S. Congressional Research Service. Report No. 7-5700. Retrieved from <https://fas.org/sgp/crs/misc/R43847.pdf>
- Moore, J. & Smith, D. (2005). Legislating Medicaid: Considering Medicaid and its origins. *Health Care Financing Review*, 27(2), 45–52. Retrieved from <https://www.cms.gov/Research-Statistics-Data-and-Systems/Research/HealthCareFinancingReview/downloads/05-06Winpg45.pdf>
- Reno, V., Williams, C., & Sengupta, I. (2005). Workers' compensation, social security disability insurance, and the offset: A fact sheet. *Social Security Bulletin*, 65(4). Retrieved from <https://www.ssa.gov/policy/docs/ssb/v65n4/v65n4p3.html>
- Social Security Amendments of 1965, Pub. L. No. 89–97, 79 Stat. 286, codified as amended at 42 U.S.C. § 426a and 42 U.S.C. § 1396b. Retrieved from <https://www.aging.senate.gov/imo/media/doc/reports/rpt565.pdf>
- State of Michigan Department of Health & Human Services. (2020). *Medicaid provider manual*. Retrieved from <http://www.mdch.state.mi.us/dch-medicare/manuals/MedicaidProviderManual.pdf>
- U.S. Department of Commerce Bureau of the Census. (1975). *Historical statistics of the United States: Colonial times to 1970*. Retrieved from <https://www.census.gov/history/pdf/histstats-colonial-1970.pdf>
- U.S. Department of Health & Human Services Centers for Medicare & Medicaid Services. (2020a). *About CMS: History*. Retrieved from <https://www.cms.gov/About-CMS/Agency-Information/History>
- U.S. Department of Health & Human Services Centers for Medicare & Medicaid Services. (2020b). *About us: Program history*. Retrieved from <https://www.medicare.gov/about-us/program-history/index.html>
- U.S. Department of Health & Human Services Centers for Medicare & Medicaid Services. (2020c). *Regulations & guidance*. Retrieved from <https://www.cms.gov/Regulations-and-Guidance/Regulations-and-Guidance>
- U.S. National Archives and Records Administration. (2020). *Social Security Act Amendments (1965): Document Info*. Retrieved from <https://www.ourdocuments.gov/doc.php?flash=false&doc=99>
- U.S. Social Security Administration. (n.d.). *Legislative history: Vote Tallies for passage of Medicare in 1965: Actions in congress*. Retrieved from <https://www.ssa.gov/history/tally65.html>

Social Skill Interventions

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Definition

Social skill interventions attempt to remediate deficits in the set of social skills that are used to interact and communicate with others. For individuals with autism spectrum disorders (ASD), this means difficulties in initiating interactions, sharing enjoyment, sustaining reciprocity, taking the perspective of another, and making inferences about the interests of others. Social skill deficits are a central feature of ASD. There are many different programs established to treat these social skill deficits for individuals with ASD; however, many programs are not well researched and do not have an established evidence base.

Historical Background

The social skill deficits noted in autism spectrum disorder (ASD) were first described in papers published in 1943, one in English and one in German. Leo Kanner (1943) described 11 children

with “early infantile autism” in his paper “Autistic disturbances of affective contact” and highlighted poor social relatedness as a key component of the disorder. Hans Asperger (1944) in Vienna, Austria, submitted a thesis on “Autistic psychopathy in childhood” and described four children with “autistic psychopathy.” He identified these children with “deficient social behavior.” Since this time, significant developments have been made to help us understand the neuropsychological underpinnings of social cognition. The notable work of researchers in the area of social cognition and theory of mind, including Dr. Baron-Cohen in England and Dr. Tony Attwood in Australia, has helped the profession and community gain a better understanding of the core social cognitive processes involved in the social interaction deficits characteristic of individuals with ASD. In addition, Dr. Temple Grandin, an individual with autism, has provided invaluable insight into the neurological strengths and challenges in autism, with her book “Thinking in Pictures” (1995), including difficulties understanding the social rules of interaction and developing relationships with other people. In recent years, there has been an increase in the awareness of the benefits of early intervention in ASD and the need for opportunities for social engagement. With this awareness has come a growing interest in developing early social communication skills including joint attention and symbolic play. Current researchers in this area include Dr. Peter Hobson, Dr. Connie Kasari, Dr. Peter Mundy, Dr. Barry Prizant, Dr. Sally Rogers, Dr. Laura Schreibman, and Dr. Amy Wetherby, among others. Practitioners, like Michelle Garcia Winner (2006), have described specific social cognition intervention strategies and developed social skills programming to assist professionals in intervention.

Rationale or Underlying Theory

Deficits in social interaction are one of the three core characteristics of autism spectrum disorder (ASD). Difficulties in engagement and socialization have far-reaching implications throughout childhood and into adulthood. Deficits in social

cognition impact the development of relationships with others, academic and vocational success, as well as mental health and quality of life. Based on current research, early intervention that focuses on building joint attention, a pivotal skill leading to later development of language and social communication, is key to fostering and developing social engagement with others. Social skills intervention is an important part of any intervention program for children and adolescents/adults with ASD.

Goals and Objectives

Social skills intervention is intended to teach both pragmatics (the social use of language) and social cognition (theory of mind, perspective taking), the latter being a critical component for children with ASD. Teaching pragmatic language skills can entail the (1) use of language (greeting, informing, demanding, promising, requesting), (2) changing language according to the needs of the listener or situation, and (3) following rules for conversation and storytelling (taking turns, introducing topics, staying on topic, rephrasing, using verbal/nonverbal signals, facial expressions/eye contact, proximity). Social cognitive aspects are addressed by teaching individuals to take the perspective and to perceive the thoughts and intentions of others. These skills are often best taught by making the abstract as “concrete” as possible, using visuals and pictures to represent abstract concepts.

Treatment Participants

Participants in social skills intervention can vary in age, skill development, and diagnosis. Children with social skill challenges span the range of developmental disabilities from those with specific learning disability and specific language impairment to those with ASD. For young children with ASD, the focus of intervention is establishing joint attention and sharing positive affect/engagement with others during play and people games/routines. During elementary and middle school, social skills intervention may be

more focused on teaching specific pragmatic language skills as well as how to engage in group activities. For children that have Asperger syndrome or high-functioning autism, a focus on teaching social cognition skills begins early as these social cognitive differences are a hallmark of these disorders.

Treatment Procedures

Social skills intervention for young children with autism spectrum disorder (ASD) often occurs during parent-child interactions. Teaching parents to engage their young children with ASD has positive outcomes on skill development and can allow for parents to help their children engage with neurotypical peers. Small group settings are often utilized to teach children with autism how to play cooperatively with others. These groups are often facilitated by an educator or speech-language pathologist. For school-age children, social skills intervention occurs in groups of similarly aged peers and can include neurotypical peers as models of prosocial behavior. These groups are often facilitated by an educator, speech-language pathologist, and/or a psychologist and can occur within the school setting or privately. Social skills intervention for the older child or adolescent with ASD entails a focus on social cognition. Often a cognitive behavioral therapy (CBT) approach is utilized to address deficits in theory of mind and perspective taking. Typically, those social skills taught include turn-taking, sharing, initiating, playing with friends, having a conversation, suggesting play ideas, giving compliments, understanding jokes and idiomatic expressions, showing affection, and perspective taking. There are also a range of intervention strategies that support social skill development including peer mediation, social stories and comic strip conversations, video modeling, integrated playgroups, ILAUGH model, etc. Evidence-based programs utilize similar strategies for teaching social skills. Skillstreaming (McGinnis and Goldstein 1997) utilizes modeling, role-playing, specific feedback, and skill transfer with homework activities to develop

competence in interpersonal conflict resolution and to learn self-control of behavior. The Second Step program (Borch 2002) contains in-school curricula, parent training, and skill development to teach socioemotional skills. Teaching strategies include modeling, coaching, group decision making, and direct skill practice. Cognitive behavioral techniques (CBTs) have been applied to the remediation of social deficits and involve the use of “self-talk” strategies to modify overt behaviors (Mahoney 1974; Meichenbaum 1977). Utilizing these techniques in social skills intervention, according to Kendall (1993), involves cognitive, behavioral, developmental, and emotive strategies that include modeling, role-playing, reinforcement, and self-evaluation. CBTs have been utilized by specific practitioners to develop social skills intervention for individuals with autism spectrum disorder (ASD), including the social thinking curriculum (Winner 2006).

Efficacy Information

Based on a review of four recent, critical reviews of social skills intervention research (Bellini et al. 2007; Matson et al. 2007; Rao et al. 2008; White et al. 2006), there is currently limited evidence for the effectiveness of group social skills intervention. Current research lacks control groups for comparison and lacks blind observers to evaluate results. All studies lack generalization techniques to demonstrate use of skills across settings. Further, there is limited longitudinal research to determine maintenance of skill acquisition, and, in many cases, there is a lack of consensus on a common definition of what comprises social skills. Of particular note is the study by Bellini et al. (2007) reviewing school-based social skills group intervention. Overall, there is a lack of evidence that children with ASD are receiving effective social skills intervention in the school setting. Recommendations based on these reviews include the need for a manualized social skills curriculum, the use of rigorous research designs to assess the effectiveness of intervention, the consistency in measurement, and the implementation of generalization measures.

Outcome Measurement

There are few assessment instruments that are able to adequately measure social skills across a variety of contexts. Standardized tests are typically not adequate in sensitivity measures to assess these skills. However, standardized measures exist that consist of parent, teacher, and individual rating scales and provide a view of social skills development that considers the impact on communication, academic performance, and behavior. Measures such as the *Social Skills Rating System* (SSRS; Gresham and Elliott 1990) and the *Vineland Adaptive Behavior Scales*, Second Edition (Vineland II; Sparrow et al. 2005) are often utilized as standardized measures of social skill development. Other standardized measures specific to measuring skills in autism spectrum disorder (ASD) include the *Autism Social Skills Profile* (ASSP; Bellini 2006), the *Social Responsiveness Scale* (SRS; Constantino and Gruber 2005), and the *Social Communication Questionnaire* (SCQ; Rutter et al. 2003). These standardized measures should be paired with observations of social interactions in natural settings (home, school, group activities). A focus in the literature at this time is the limited generalization of social skills across environments and contexts. More rigorous research designs and targeted evaluation of social skills are needed to determine the efficacy of social skills intervention.

Qualifications of Treatment Providers

Individuals providing social skills intervention should have training and experience in working with individuals with autism spectrum disorders. These licensed professionals may include psychologists, speech-language pathologists, social workers, and educators. Individuals with some form of psychological training can be well versed in teaching skills to adolescents and can address some of the concomitant behavioral and psychological issues that can be present (depression, anxiety, etc.).

See Also

- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Social Cognition
- ▶ Social Communication
- ▶ Social Interventions

References and Reading

- Asperger, H. (1944). Autistic psychopathy in childhood (Firth, U. (1991), Trans. and annotated). In U. Frith (Ed.), *Autism and Asperger syndrome* (pp. 37–92). Cambridge, MA: Cambridge University Press.
- Bellini, S. (2006). *Building social relationships: A systematic approach to teaching social interaction skills to children and adolescents with autism spectrum disorders and other social difficulties*. Shawnee Mission: Autism Asperger.
- Bellini, S., Peters, J., Benner, L., & Hope, A. (2007). A meta-analysis of school-based social skills interventions for children with autism spectrum disorders. *Remedial and Special Education*, 28(3), 153–162.
- Borch, P. (2002). *Second Step staff training video: Grades 1–5 (VHS)*. Seattle: Committee for Children.
- Constantino, J., & Gruber, C. (2005). *The social responsiveness scale manual*. Los Angeles: Western Psychological Services.
- Grandin, T. (1995). *Thinking in pictures: And other reports from my life with autism*. New York: Doubleday Dell.
- Gresham, F., & Elliott, S. (1990). *Social skills rating system*. Circle Pines: American Guidance Service.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kendall, P. (1993). Cognitive-behavioral therapies with youth: Guiding theory, current status, and emerging developments. *Journal of Consulting and Clinical Psychology*, 61, 235–247.
- Koegel, R., & Frea, W. (1993). Treatment of social behavior in autism through the modification of pivotal social skills. *Journal of Applied Behavior Analysis*, 26, 369–377.
- Mahoney, M. (1974). *Cognition and behavior modification*. Cambridge, MA: Ballinger.
- Marans, W., Rubin, E., & Laurent, A. (2005). Addressing social communication skills in individuals with high-functioning autism and asperger syndrome: Critical priorities in educational programming. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, pp. 977–1002). Hoboken: Wiley.
- Matson, J., Matson, M., & Rivet, T. (2007). Social skills treatments with children with autism spectrum disorders. *Behavior Modification*, 31, 682–707.
- McConnell, S. (2002). Interventions to facilitate social interaction for young children with autism: Review of available research and recommendations for

- educational intervention and future research. *Journal of Autism and Developmental Disorders*, 32(5), 351–372.
- McGinnis, E. & Goldstein, A. (1997). *Skillstreaming the elementary school child: New strategies and perspectives for teaching prosocial skills* (Rev. ed.). Champaign: Research Press.
- Meichenbaum, D. (1977). *Cognitive behavioral modification: An integrative approach*. New York: Plenum.
- Rao, P., Beidel, D., & Murray, M. (2008). Social skills interventions for children with Asperger's syndrome or high-functioning autism: A review and recommendations. *Journal of Autism and Developmental Disorders*, 38(2), 353–361.
- Rutter, M., Bailey, A., & Lord, C. (2003). *Social communication questionnaire-WPS (SCQ-WPS)*. Los Angeles: Western Psychological Services.
- Sparrow, S., Cicchetti, D., & Balla, D. (2005). *Vineland II: A revision of the vineland adaptive behavior scales: I. survey/caregiver form*. Circle Pines: American Guidance Service.
- White, S., Keonig, K., & Scahill, L. (2006). Social skills development in children with autism spectrum disorders: A review of the intervention research. *Journal of Autism and Developmental Disorders*, 37(10), 1858–1868.
- Winner, M. (2006). *Think social! A social thinking curriculum for school-age students*. San Jose: Think Social.

Social Skills

- [Social Cognitive Interventions](#)
- [SSIS SEL](#)

Social Skills Improvement System

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Synonyms

[Prosocial behaviors](#); [Prosocial skills](#); [Social-emotional skills](#)

Description

The Social Skills Improvement System (SSiS; Gresham and Elliott 2008) Rating Scales assist professionals in screening and classifying students ages 3–18 years suspected of having significant social skills deficits. The SSiS Classwide Intervention Program and SSiS Intervention Guide are manualized treatment programs for eliminating or reducing social skill deficits identified on the SSiS Rating Scales. The SSiS uses a multirater (parents, teachers, and students with at least third-grade reading ability) approach that provides a comprehensive examination of seven areas of prosocial skills (communication, cooperation, assertion, responsibility, empathy, engagement, and self-control) and five areas of problem behaviors (internalizing, externalizing, bullying, hyperactivity/inattention, and autism spectrum). The instrument yields norm-referenced scores based on a national sample representative of the 2006 US Census.

Given that autism is characterized by impaired social interactions, problems with verbal and nonverbal communication, and repetitive/stereotyped behaviors, the SSiS Rating Scales and related intervention manuals offer professionals a focused screener for autism spectrum disorders within a comprehensive content of social behavior. The SSiS Autism Spectrum Subscale is comprised of 15 items, 8 from the Social Skills Scale (e.g., interacts well with other children, makes eye contact when talking, uses gestures or body appropriately with others) and 7 items from the Problem Behavior Scale (e.g., is preoccupied with object parts, becomes upset when routines change, repeats the same thing over and over). The items on both the Social Skills Scale and Problem Behavior Scale are rated on a 4-point scale (0 = Never, 1 = Seldom, 2 = Often, 3 = Almost Always). Low-frequency ratings on the eight social skills items, whereas high-frequency ratings on the problem behavior items are indicative of a possible autism spectrum disorder and thus should be followed up with a more comprehensive assessment.

Historical Background

The SSiS Rating Scales originally was published in 1990 as the *Social Skills Rating System* (SSRS; Gresham and Elliott 1990). The SSRS was reportedly used by many professionals as part of their autism screening efforts; however, the SSRS was not designed with autism or autism spectrum disorders in mind. When the revision of the SSiS was being conceptualized, the authors consult with educators serving autistic children and the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., 1994) of the American Psychiatric Association and the National Research Council's report (2001) on *Educating Children with Autism* to ensure they were sensitive to critical identifying behaviors and conditions.

Psychometric Data

The SSiS-RS manual provides extensive validity evidence based on test content, internal structure, intercorrelations among scales and subscales, item-total correlations, and relations with other variables (Gresham and Elliott 2008). Item development of the SSiS-RS was based on a broad review of the empirical literature on social skills deficits in special populations, reviews of published empirical studies using an earlier version of the scale (Social Skills Rating System, Gresham and Elliott 1990), and research on the relationship between specific social behaviors and important social outcomes for children and youth. Intercorrelations among scales and subscales for each form are moderate to high for the social skills and problem behavior scales. Item-total correlations across forms by age tend to be moderate to high, many of which exceed 0.70–0.80.

Correlations between the SSiS-RS and the Behavior Assessment System for Children, Second Edition (BASC-2; Reynolds and Kamphaus 2004), are moderate to high, depending on the scales and subscales. For example, the median correlations between the SSiS-RS total social skills score and the teacher form of the BASC-2 social skills score are 0.78 and 0.69 for the teacher and parent forms, respectively. Correlations

between the SSiS-RS total social skills scores and the socialization scores of the Vineland Adaptive Behavior Scales, Second Edition (Vineland II; Sparrow et al. 2005), are 0.65 and 0.44 for the teacher and parent forms, respectively.

Using a sample of 50 students with an autism spectrum disorder as diagnosed by the DSM-IV-TR, a known group comparison analysis with a nonclinical sample was conducted. On the teacher and parent forms of the SSiS, all the mean score differences were statistically significant. The Social Skills Scale means of 74.6 and 75.8 were at least 1.5 standard deviations lower than the nonclinical reference sample of students. Conversely, the scale means for the problem behaviors on both forms were more than 1 standard deviation higher than for the nonclinical reference group. The greatest differences were on the Autism Spectrum Subscale, where the scores of 23.9 and 22.6 were nearly 2 standard deviations higher than the nonclinical reference group mean. These results are consistent with expectations that individuals with autism spectrum disorder have major deficiencies in social skills and tend to exhibit more problem behaviors, thus lending substantial support for the validity of the scores of this subscale as a measure of behaviors typically exhibited by individuals diagnosed with autism.

Clinical Uses

The SSiS Rating Scales – teacher, parent, and student forms – are intended to be used as a comprehensive measure of children's social skills and a useful screener of children's problem behaviors. In particular, these rating scales are designed to identify social skills acquisition and performance deficits in areas of communication, cooperation, assertion, responsibility, empathy, engagement, and self-control.

The Autism Spectrum Subscale is designed to facilitate a time-efficient and contextualized screening measure. By using the nationally representative norming sample for comparison, it is possible to get a clear comparison between a student suspected of an autism spectrum disorder to a

same age and sex comparison group of nonclinical students.

Scores on the SSIS Rating Scale items can be analyzed using the Social Skills Analysis of Behavior to identify strengths, performance deficits, and acquisition deficits. Based on the analysis, items can be aligned or linked to a skill unit in the SSIS Classwide Intervention Program (CIP) to be taught using a manualized lesson plan. The SSIS CIP is a highly effective intervention program as document in several recent randomized control trial (RCT) investigations with first, second, and third grade students.

See Also

- [Social Behaviors and Social Impairment](#)
- [Social Communication Questionnaire](#)
- [Social Language Development Test](#)
- [Social Skill Interventions](#)
- [Stereotypic Behavior](#)
- [Structured Descriptive Assessment](#)

References and Reading

- DiPerna, J. C., Lei, P., Bellinger, J., & Cheng, W. (2015). Efficacy of social skills improvement system Classwide intervention program (SSIS-CIP) primary version. *School Psychology Quarterly*, 30(1), 123–141. <https://doi.org/10.1037/spq0000079>.
- Elliott, S. N., & Gresham, F. M. (2007). *Social skills improvement system: Performance screening guides*. Bloomington: Pearson Assessments.
- Elliott, S. N., & Gresham, F. M. (2008). *The SSIS ASSIST: Scoring, interpretation, and report writing program*. Bloomington: Pearson Assessments.
- Gresham, F. M., & Elliott, S. N. (1990). *Social skills rating system*. Minneapolis: NCS Pearson.
- Gresham, F. M., & Elliott, S. N. (2008). *Social skills improvement system: Rating scales*. Bloomington: Pearson Assessments.
- Reynolds, C., & Kamphaus, R. W. (2004). *Behavior assessment system for children* (2nd ed.). Minneapolis: NCS Pearson.
- Sparrow, S. S., Cichetti, D. V., & Balla, D. A. (2005). *Vineland Adaptive behavior scales: Parent/caregiver rating form* (2nd ed.). Minneapolis: NCS Pearson.
- The SSIS is published by Pearson Clinical Assessments. 19500 Bulverde Road, San Antonio, TX 78359. www.PsychCorp.com.

Social Stigma

- [Stigmatization](#)

Social Stories

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Definition

A social story is a brief story that describes a social situation including cues and appropriate responses.

Historical Background

Social Stories™ were first developed, introduced, and trademarked in 1991 by Carol Gray. They are generally regarded as a positive intervention strategy with some components of priming. Social Stories™ were developed so that students with autism spectrum disorders (ASD) could learn social skills. They are written and tailored to the individual with ASD to help him or her understand and respond appropriately in various social situations.

Originally, social stories were written using three types of sentences: descriptive, directive, and perspective taking. Descriptive sentences provided an overview of a social situation in terms of relevant social cues. Directive sentences specified an appropriate response, and perspective sentences described the feelings and responses of both the individual with ASD and others represented in the target social situation. Ms. Gray specified a specific ratio of sentences to be contained within each Social Story™. Also, initially, Social Stories™ were presented as text,

and the individual with ASD was encouraged to read them by himself or herself.

Over the years, in recognition that not all students with ASD were able to read and comprehend Social Stories™, the process for writing these stories has changed dramatically. Ms. Gray originally discouraged the inclusion of illustrations as she felt that a student may be distracted by them or misinterpret the situation based on an illustration. Today, it is commonplace to supplement social story text with pictures in the forms of photographs or Mayer-Johnson™ Picture Communication Symbols or to use PowerPoint and computer-assisted presentation of Social Stories™. Social Stories™ also have been presented in the form of Comic Strip Conversations™, news articles, and research articles. Ms. Gray emphasizes presenting Social Stories™ in a format that is motivating to the individual with ASD. Other changes to the development and presentation of social stories are the revision of the types of sentences used within a social story; social stories now include the following: descriptive, directive, perspective, affirmative, control, cooperative, and partial sentences. As described by Ms. Gray, affirmative sentences may express a commonly shared value or feeling, and they serve to enhance the meaning of statements. Control sentences are written by the individual after he or she reviews the social story and are used to identify personal strategies that will help him or her recall and implement skills that are outlined in the social story. Cooperative sentences are sentences that point out ways that others will assist the individual. Finally, partial sentences promote prediction and encourage the person with ASD to guess the next step in the situation. It should be noted that, at this time, control and cooperative sentences are optional when writing Social Stories™. There is a recommended ratio of zero to one directive or control sentences and between two and five descriptive and/or perspective sentences in a Social Story™. Despite these expansions of the form of a social story, the intent remained to use it as an antecedent condition to teach students with ASD to recognize and respond to a wide array of social contexts.

Rationale or Underlying Theory

Some have described the rationale for using Social Stories™ as one that teaches persons with autism spectrum disorder to develop theory of mind (perspective-taking skills). Carol Gray does not present a theory but rather describes the goal of Social Stories™ is to share accurate social information in a patient and reassuring a manner that is easily understood by its audience. Half of all Social Stories™ developed should affirm something that an individual does well. Although the goal of a Story™ should never be to change the individual's behavior, that individual's improved understanding of events and expectations may lead to more effective responses.

An alternative explanation for the use of social stories is that it involves a shared schema or background knowledge and allows one to “build a scaffold of understanding for a schema (mental representation) that an individual does not yet possess” (Reynhout and Carter 2006). Myles and Simpson (2001) suggested that Social Stories™ provide access to the “hidden curriculum” (the dos and don'ts understood and adhered to by everyone except those with ASD) inherent in social situations (Reynhout and Carter 2006).

Goals and Objectives

As previously mentioned, the goal of a social story is to share important information about a social context. Information is shared in a way that is age appropriate to the student with ASD and ideally allows him or her to read about and be more “comfortable” in social situations. Social Stories™ have been used to teach a variety of skills including following routines, academic skills such as math, social situations such as playing at recess, having a conversation, and respecting personal space. As noted by Gray and Garand (1993), “Generic Social Stories™ can be used to describe social situations frequently experienced by children with autism which can be individualized and adapted as the need arises.”

Treatment Participants

Traditionally, persons with ASD have participated in the use of Social Stories™. Initially, Social Stories™ were recommended for use with students who have ASD and who have the ability to read and who are considered individuals with “high-functioning” autism.

Treatment Procedures

Social Stories™ are written according to the guidelines published by Carol Gray. Several considerations are necessary when writing Social Stories™. These considerations include the need to maintain the perspective of the student for whom the story is written, keep the story within the student’s comprehension level, and include appropriate vocabulary and size of font for their reading ability. Behavioral responses should be stated in positive terms, that is, label what you want the student to do, rather than what they should not do, and Gray and Garand (1993) recommend that students demonstrate intellectual ability in the range of 35–55 on a standardized IQ test (Reynhout and Carter 2006). Additionally, it is important to determine whether text will be supplemented with pictures and if pictures will be photographs, Picture Communication Symbols™ (Mayer Johnson), or embedded in a PowerPoint presentation.

Once social stories are developed, they are to be read to the child who is unable to read them independently. Reading of the Social Story™ should happen in advance of entering into the social situation, and there should be a time to check for comprehension. In summary, social stories are written according to the guidelines set forth by Carol Gray, and then read to or with the individual with ASD, after which, a comprehension check is made. Comprehension can be checked either orally or in writing by having the student answer a series of questions about the social story. There currently are no hard-and-fast guidelines about the frequency of using Social Stories™; however, it is noted that they can be faded over time.

Efficacy Information

To date, there is limited data on the efficacy of social stories. Studies that have been conducted have yielded mixed results. For example, Social Stories™ have rarely been evaluated exclusively; usually, they incorporate some other form of treatment such as role play, modeling, rehearsal, written checklists, and token rewards. The implementation of Social Stories™ also has varied from study to study. For example, those that have used social stories have varied the frequency of presentation as well as the setting for presentation (home, in class, outside of a classroom). They also have used Social Stories™ to follow up with a situation if there is a misperception by the student. In this situation, social stories are used as an antecedent and consequence, and, during the consequence portion, the reader highlights the information that was misinterpreted.

Variations in presentation, use, and style of Social Stories™ make it difficult to interpret the data. Of studies that have been conducted using single-subject research methodology, results have been mixed. Some researchers have noted initial gains using Social Stories™, yet the gains are short lived, and there is lack of maintenance and generalization. Efficacy of Social Stories™ may also have been compromised. There is literature to suggest that there has been inconsistency in conforming to the guidelines for writing social stories. At best, current research data for Social Stories™ does not support them as an evidence-based practice; however, they continue to be used due to clinical popularity and the belief that they represent best practice strategies for students with ASD (Smith 2001 in Reynhout and Carter 2006).

Outcome Measurement

Currently, outcome measurement for Social Stories™ is limited. There are a few studies that have been conducted, and many of these studies have some methodological flaw(s). There are limited studies and/or review of the literature that address the efficacy of social stories; of those available, analysis of the results remains varied

and inconclusive, suggesting that more research is needed.

Qualifications of Treatment Providers

There are no hard-and-fast qualifications for treatment providers; rather, one simply needs to be able to follow the guidelines set forth by Carol Gray. Treatment providers can be teachers, speech-language pathologists, parents, counselors, and other significant adults who work with or are familiar with the person with ASD.

References and Reading

- Ali, S., & Frederickson, N. (2006). Investigating the evidence base of social stories. *Educational Psychology in Practice*, 22(4), 355–377. <https://doi.org/10.1080/02667360600999500>.
- Delano, M., & Snell, M. E. (2006). The effects of Social Stories™ on the social engagement of children with autism. *Journal of Positive Behavior Intervention*, 8(1), 29–42.
- Dev, P. C. (2014). Using social stories for students on the autism spectrum: Teacher perspective. *Pastoral Care in Education*, 32(4), 284–294. <https://doi.org/10.1080/02643944.2014.974662>.
- Gray, C. A., & Garand, J. D. (1993). Social stories: Improving responses of students with autism with accurate social information. *Focus on Autism and Other Developmental Disabilities*, 8, 1–10.
- Gray, C. (1994a). *Comic strip conversations*. Arlington: Future Horizons.
- Gray, C. (1994b). *The new social story book* (illustrated ed.). Arlington: Future Horizons.
- Gray, C. (2010). *The new social story book* (revised and expanded 10th anniversary edition). Arlington: Future Horizons.
- Lorimer, P. A., Simpson, R. L., Myles, B. S., & Ganz, J. B. (2002). The use of Social Stories™ as a preventative behavioral intervention in a home setting with a child with autism. *Journal of Positive Behavior Intervention*, 4(1), 53–60.
- McAfee, J. (2002). *Navigating the social world* (pp. 114, 120, 145, 155, 185, 187–188, 194, 200). Arlington: Future Horizons.
- Myles, B. S., & Simpson, R. (2001). Understanding the hidden curriculum: An essential social skill for children and youth with Asperger syndrome. *Intervention in School and Clinic*, 36, 279–286.
- Reynhout, G., & Carter, M. (2006). Social Stories™ for children with disabilities. *Journal of Autism and Developmental Disorders*, 36(4), 445–469.
- Santosti, F. J., & Powell-Smith, K. A. (2006). Using Social Stories™ to improve the social behavior of children with Asperger syndrome. *Journal of Positive Behavior Intervention*, 8(1), 43–57.
- Santosti, F. J., & Powell-Smith, K. A. (2008). Using computer-presented social stories and video models to increase the social communication skills of children with high-functioning autism spectrum disorders. *Journal of Positive Behavior Intervention*, 10(3), 162–178.
- Schneider, N., & Goldstein, H. (2010). Using Social Stories™ and visual schedules to improve socially appropriate behaviors in children with autism. *Journal of Positive Behavior Intervention*, 12(3), 149–160.
- Smith, C. (2001). Using social stories to enhance behavior in children with autistic spectrum difficulties. *Educational Psychology in Practice*, 17, 337–345.

Social Thinking

- [Pragmatics](#)
- [Social Cognitive Interventions](#)

Social Tools And Rules for Teens (START) Program

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Definition

The Social Tools And Rules for Teens (START) socialization program for adolescents with autism spectrum disorder (ASD) is a peer-facilitated intervention that combines experiential learning opportunities with an interactive social curriculum. It is a 20-session program consisting of weekly 90-min group meetings. Each meeting consists of an individual check-in session, a free socialization period, a social topic discussion, a structured social activity, and a checkout

session. Structured and unstructured free socialization periods are embedded within each group session to provide a supportive, natural context for social experimentation, self-management, and skill building. The interactive didactic social lessons are intended to provide opportunities to promote social insight and effective social skill use. The START program was developed at the University of California, Santa Barbara.

Historical Background

By the nature of their diagnosis, adolescents with ASD often experience limited social success due to challenges with social skills, insight, and motivation (Orsmond et al. 2004). These limited social competencies can leave these individuals vulnerable to social rejection or exclusion (Schroeder et al. 2014), limiting their access to the peer interaction needed to actually improve their social skillset. Many adolescents with ASD experience lower perceived social status, friendship quality, and social motivation, along with greater levels of loneliness than typically developing peers (Locke et al. 2010; Mazurek 2013). Fortunately, there has been a recent increase in empirically supported social skill interventions that attempt to address these challenges and generate crucial social momentum (Miller et al. 2014).

A supportive network of peers is a crucial, yet often overlooked, component in socialization efforts. Fortunately, the use of peer-mediated interventions is growing in popularity (Watkins et al. 2015). Typically-developing peer models are valued for their ability to connect with same-aged participants and model, teach, and constructively evaluate social performance. They are also immersed in the same unique peer social culture as the target population and can provide much more ecologically appropriate instruction than a generic social skills curriculum taught by adult clinicians. Through these positive interactions, individuals with ASD also gain exposure to unconditional social acceptance – exchanges with receptive peers who are friendly, willing to converse, and forgiving of social missteps. Having positive experiences with peers allows

adolescents with ASD to build social momentum that increases both confidence and competence to interact.

The initial pilot of the START program was developed to improve social competence in adolescents with ASD through the use of immersive experiential learning techniques (Vernon et al. 2016). This process of learning operates through exposure to and processing of real-world experience (Kolb 2014). A socialization intervention that incorporates experiential learning allows participants to engage with peers and experiment with social strategies firsthand, reflect on these experiences, and learn from their mistakes. Instructional social skills lessons also have value; however, many of the social nuances associated with live interactions are unable to be replicated when teaching a didactic lesson. Individuals often learn best by doing. This process is analogous to learning a sport or musical instrument. Some gains can be made through professional instruction, tutoring, and practice exercises, but one can only master a skillset through repeated live, real-world performances. By actively engaging in a naturalistic social setting with typically developing peers, individuals can benefit from the social complexities inherent in real-world interaction. Moreover, these active learning opportunities allow individuals to experience the natural incentives of positive peer interactions, such as discussion of favorite topics, responses to humor, and opportunities to jointly engage in enjoyable activities. By practicing social interactions in a safe, welcoming environment, participants can begin to associate social engagement with rewarding positive experiences and outcomes. The efficacy of the START program has been further established through the outcomes of a randomized clinical trial (Ko et al. 2018; Vernon et al. 2018).

Rationale or Underlying Theory

In order to better understand the design and conceptualized mechanism of change of the START program, it may be helpful to explore its theoretical underpinnings. The model is grounded

in an experiential model of social learning. Experiential learning theory is conceptualized as a four-stage learning process (Kolb et al. 2001) in which (1) an experience serves as the stimulus for (2) reflective observation on what is functionally working or failing to work in the learning context, which (3) solidifies one's abstract conceptualization of a given phenomenon. Based on this accrual of knowledge, the individual is able to engage in (4) active experimentation to hone one's knowledge base and associated skill set, which is then applied to future experiences as the cycle repeats.

When applied to socialization and conversational competence, experiential learning is heavily reliant on sustained immersion in an appropriate social environment (Baker et al. 2002). Mapped onto the four-stage experiential learning process, interactions with others provide an experiential stimulus that allow individuals to reflect on their interpersonal successes and failures, improve their understanding of successful social strategies, and modify subsequent social bids to improve the likelihood of success in future engagements. For the general adolescent population, the accumulation of these interactions appears to be crucial for establishment of social competence and seems to be predicated upon two interrelated factors: (a) the willingness (or motivation) of the individual to engage with available social partners and (b) the willingness of these partners to be responsive to the social bids of that individual.

Goals and Objectives

The START program is designed to improve the social readiness skills of adolescents with ASD. Social readiness can be conceptualized as the combination of two essential components needed for optimal interpersonal functioning: social motivation and social competency. In turn, social competency can be further broken down into mastery of social insight and social skills (Vernon et al. 2016). The START socialization curriculum was intentionally designed to integrate both experiential and didactic training components

within a single peer-facilitated intervention to target these components.

Social Motivation

Social motivation is the desire to voluntarily engage in social pursuits and the anticipation that one will derive pleasure from such interactions, serving as a popular conceptualization of autism-related vulnerabilities (Chevallier et al. 2012; Dawson et al. 2005). Social motivation is a prerequisite that makes social immersion and experiential learning possible. At its foundation, the START program was intended to maximize the motivation of individuals who are interpersonally vulnerable. A safe, accepting space was fostered for practicing social skills without the fear of rejection or judgment. Group cohesion and unconditional acceptance were emphasized. By creating a supportive experiential context, the participants were granted the opportunity to become fully immersed and accepted by a peer group, which then became the ideal forum to practice and master critical social competencies. Over the course of 20 weeks, they accumulated frequent, reoccurring experiences of social success to establish both confidence and valuable social momentum.

Social Insight

Social insight is the understanding of underlying principles that govern successful social behavior, encompassing various concepts of social cognition (i.e., empathy, theory of mind, problem-solving capacities, and executive functioning skills; Schmidt et al. 2011). Several program components were specifically incorporated into the program to foster social insight. The check-in and checkout phases of each group provided a private forum to intentionally reflect on one's social experiences and process perceived success and failure. Participants were also encouraged to think through role-plays and videos and describe the reasoning behind certain social decisions. Finally, during group topic discussions, the rationale underlying the use of certain social strategies was explored and explicit links were made to principles of empathy, courtesy, respect, and constructive problem solving.

Social Skills

Finally, social skills describe specific behaviors that are employed in the proper context at the proper time to facilitate a successful person-to-person exchange. The hands-on activities and self-management components of the START program facilitated the development and mastery of this concrete social skillset. Individual skills were modeled, practiced, and critiqued each session. Intentional practice redundancies were also used to promote eventual automaticity of skill use. Specifically, individualized target skills were practiced during check-in, self-managed during group, and assigned as homework in between group sessions. Similarly, weekly skills highlighted in the group curriculum were critically analyzed during role-play sessions and video examples, demonstrated, practiced in pairs, and assigned as weekly social homework.

Treatment Participants

The inclusion criteria for START program participation includes an age between 12 and 17 years, a diagnosis of autism spectrum disorder, a verbal IQ score above 70, and the ability to comprehend and speak in full sentences. To date, only an English language version of the START program has been evaluated. Because the program relies on the use of conversation, discussion of social issues, and self-reflection, it is likely not suitable for individuals with more limited language skills or intellectual impairment (although an adapted version of the START program for individuals with higher support needs is currently in development).

Treatment Procedures

The START program consists of 20 weekly sessions that are 90 min in length. Each meeting consists of a 5-min individual check-in session, a 20-min free socialization period, a 40-min social topic discussion, a 20-min structured social activity, and a 5-min checkout session that parents are invited to join.

Pre-group Preparation

Prior to beginning the program, participants complete an initial 90-min intake session, which consists of obtaining consent/assent from parents and adolescents, obtaining basic demographic information, and completing all required intake measures. Trained undergraduate-level research assistants conducted all intakes and subsequent progress meetings. During the pre-intervention session, an assessment of individual social vulnerabilities was conducted. Participants and parents were provided with a list of common social skill difficulties/vulnerabilities and also had the option to write in additional concerns that were not listed (Vernon et al. 2016). Based on a rank order of specific social skill difficulties completed separately by each adolescent, their parent, and an intake clinician, consensus was reached on an individual social skill to serve as the initial focus of self-management. The primary objective was to directly target the social skill deficit that was identified as having the largest negative impact on each adolescent's level of social success.

The primary facilitator discussed the target skill with the adolescent and they jointly practiced self-management procedures (monitoring and tracking one's use of a particular social skill). Specifically, the skill was operationally defined for the participant, modeled by the social facilitator, and then practiced in a brief conversational exchange while the participant tracked their skill use using a small digital tally counter. After the participant could verbally describe the target social skill and successfully demonstrate accurate self-management in conversation, they were encouraged to continue self-managing use of this skill during each START group session. Individual goals were reexamined every 5 weeks and a new primary skill was introduced as participants either (a) demonstrated adequate mastery of a previous target skill or (b) exhibited a more significant challenge in another skill domain that was identified as a more notable source of social difficulty.

START Program Session Format

START program sessions: All of the adolescents began participation in the START program within

2 weeks of completing all pre-intervention measures. The 90-min weekly program consisted of the following phases: an individual therapeutic check-in session, a group unstructured socialization time, a structured group activity, a group discussion and practice of a social skill topic, and an individual checkout session with parent involvement. Participants, two to four college-aged social facilitators, and one to two high school peers attended each group.

Individual Check-in Session

Adolescents first completed an individual check-in session with a college-aged social facilitator. This time period was allotted to provide a private forum to discuss the previous week's social challenges and successes, review social homework, practice self-management of the current individualized target behavior, and become oriented to the activities of the upcoming session. Facilitators used this time to prime individuals of session content and answer questions.

Free Socialization Phase

All participants joined the group's free socialization time with the facilitators and high school peers. This time was allowed to unfold without a predetermined agenda and was intended to create a natural, comfortable social environment. Topics were brought up organically by the participants and often included video games, favorite foods and places to eat, school and current events, vacation and weekend plans, and memorable personal experiences. Food and refreshments were provided for each group to aid in the creation of a casual, club-like atmosphere. While conversing with one another, the participants and social facilitators discretely tracked their use of individual target skills using self-management. The high school peer models and the college-aged facilitators also participated in this self-management process to ensure that every group member was held to the same expectations and to minimize perceived differences between session attendees.

Social Topic Discussion and Practice

After the free socialization phase, the social facilitators then introduced the week's social skill topic, which was explored for the next 40 min. After a brief introduction of the target skill, the topic was then modeled by the social facilitators in a series of two brief role-plays – one “bad” example demonstrating poor implementation of the skill and a follow-up “good” example depicting proper use of that particular skill. Popular television and movie clips were also used to highlight good and bad examples of the skill in question. Social facilitators discussed the main points of the topic and recalled relevant personal experiences. The adolescent participants were also encouraged to contribute to the topic – discussing their experiences related to that topic and providing their own suggestions regarding the successful use of a particular social skill. Finally, for the last 5 min of the group, all participants practiced the related skill with a partner. A manualized curriculum of key points and sample stories and scripts were used to structure and guide these discussions. This portion of the group was intended to increase understanding of a social skills topic and provide opportunities to both observe and practice the skill. Social skills topics covered included making introductions, maintaining a conversation, respectfully disagreeing, and group interactions.

Social Activity Phase

For the final 20 min, the group transitioned into a structured social activity. These activities varied each week, but generally resembled commonly used team-building activities and party games. Activities were also selected to be highly enjoyable and motivating to increase the engagement of the group participants. This phase was intended to foster sharing of personal information, encourage learning about peer interests, increase comfort in the group, and promote cooperation and teamwork. Other benefits included opportunities to work on effective communication, compromise, and good sportsmanship skills.

Checkout Session

At the end of each group session, individual checkout sessions were conducted with each participant, a parent, and their assigned social facilitator. The participants discussed their experiences in the group, reviewed the group socialization topic with their parent, and set two weekly homework assignments – one based on their individual self-management target and the other based on the weekly group curriculum. Finally, a written summary of the current topic was provided to the family as a visual reference.

Efficacy Information

The efficacy of the START program has been evaluated through three peer-reviewed research articles. The first article summarized the results of pilot study with six participants (Vernon et al. 2016). The study used a combination of a multiple baseline and a pre-post design to establish preliminary evidence of efficacy by examining improvements on parent-report measures, adolescent self-report measures, and increases in coded social skill use on videotaped social conversation probes.

A randomized clinical trial was then conducted to evaluate the impact of the START model on social performance. All participants were randomly assigned to one of two groups: Those assigned to the START treatment group immediately received 20 weeks of the START program. Those assigned to the waitlist control group were permitted to continue any preexisting therapy efforts but were not exposed to the experimental socialization treatment package. Both groups were reevaluated after a 20-week time period.

The first START RCT article detailed the results of the parent and adolescent self-report social survey measures (Vernon et al. 2018). Results of the mixed MANOVA analyses revealed the presence of significant Group $\times$ Time effects for the social outcome variables (Wilks' Lambda = 0.53; $F(5,29) = 5.22, p = .002$). Group $\times$ Time interaction reached statistical significance for three outcome measures: Parent SRS, $F(1,33) = 7.68, p = .009$, partial $\eta^2 = .189$; Parent SMCS, F

$(1,33) = 13.29, p = .001$, partial $\eta^2 = .287$; and Adolescent SSIS, $F(1,33) = 7.29, p = .010$, partial $\eta^2 = .181$. All parents endorsed social validity ratings indicating that their adolescent highly enjoyed being a part of the social skills group (mean rating of 8.14 out of 10, $SD = 0.97$). Additionally, they endorsed that their child's social skills and competence improved through participation in the group (mean of 7.50 out of 10, $SD = 0.82$). The adolescent participants also indicated that they enjoyed their time in the groups (mean rating of 8.41 out of 10, $SD = 1.83$). Likewise, they endorsed that their social skills and competence improved through participation in the group (mean of 8.34 out of 10, $SD = 1$).

The next peer-reviewed publication examined real-world social skill use during live peer conversations (Ko et al. 2018). Three observable social behaviors were coded: questions asked, directed positive facial expressions, and mutual engagement. Results revealed a significant Group $\times$ Time interaction effect for Questions Asked, $F(1, 33) = 4.86, p = .035$. Participants in the treatment group significantly increased the percentage of questions asked from pre- to post-intervention, whereas the percentage of questions asked by the waitlist group remained relatively unchanged. Effect size was medium-large (partial $\eta^2 = .128$). Additionally, analyses revealed the presence of a significant Group $\times$ Time effect on the percentage of Positive Facial Expressions, $F(1, 33) = 7.73, p = .009$. The treatment group demonstrated significant improvements in the percentage of directed positive facial expressions during social conversations compared to the waitlist group. The Effect size was large (partial $\eta^2 = .190$). Group $\times$ Time interaction did not reach statistical significance for Mutual Engagement, $F(1,33) = 0.32, p = .576$.

Outcome Measurement

As described above, the efficacy of the START program has been evaluated using standardized survey measures (parent report and adolescent self-report measures; Vernon et al. 2018) and behaviorally coded social skill use derived from

live social conversations with unfamiliar peers (Ko et al. 2018).

Survey-Based Outcome Measures

Social Skills Improvement System Rating Scales (SSIS-RS): The SSIS-RS is a 75–83 item revised version of a widely used rating scale measuring several aspects of social skills, including communication, cooperation, assertion, responsibility, empathy, engagement, and self-control (Gresham and Elliott 2008). Internal consistency alpha reliability coefficients for the parent and self-report forms are reported to be in the mid to upper .90s, with moderate to high correlations to other social and behavioral scales.

Social Responsiveness Scale, Second Edition (SRS-2): The SRS-2 is a 65-item rating scale that covers various dimensions of interpersonal behavior, communication, and stereotypic behavior associated with ASD (Constantino and Gruber 2005). Internal consistency alpha reliability coefficients for the parent forms were reported to be above .90 and strong correlations ($r = .52-.74$) with subscales of the ADI-R. This measure was used as an indicator of ASD symptom severity, with score reductions associated with a decrease in observable symptoms.

Social Motivation & Competencies Scale (SMCS): The SMCS is an unpublished rating scale that was developed by the current researchers for use in this study. It was designed with corresponding parent and adolescent self-report versions. Items pertaining to comfort in social interaction, conversation skill use, empathy, friendships, appropriate behavior, social contact, and social interest were rated on a 5-point Likert scales. This measure was used as an indicator of social motivational factors and concrete skill competencies.

Social Validity Survey: Parents and participants were both asked to provide ratings about the acceptability of the START program. Specifically, they were asked to provide separate ratings on a 0–10 Likert scale on both (a) enjoyment of the adolescent's time in the group and (b) the extent to which the adolescent's social skills and competence improved as a result of participation.

Social Conversation Probe Procedures

Social conversation data were collected during the pre-intervention intake session and after program completion (i.e., after 20 weeks of the START program or waitlist participation). At these time points, participants engaged in 5-min conversations with unfamiliar peers whom they had never met. These peer conversational partners were not trained or coached prior to their interactions with the participants. They also possessed no knowledge of the purpose of the research study and did not know the diagnostic status of the participants. They were randomly assigned to participants and never completed more than one conversation with the same participant. They were provided with the following instructions: "You will be having a conversation with another person that you have never met before. You will have five minutes to get to know each other."

Social Conversation Outcome Measures

Dependent measures were selected to provide representative data associated with improvements to the core social deficits of ASD (APA 2013). Video-recorded conversations, pre- and post-intervention, were systematically coded for each dependent measure. The three dependent measures coded were questions asked, positive facial expressions, and mutual engagement.

Questions Asked: Social inquiries have been identified as a crucial interpersonal strategy, but compared to typically developing adolescents, adolescents with ASD are known to make far fewer social initiations to their peers. A question was defined as a verbal query that is intended to elicit a response from the conversational partner. Trained coders, who were blind to the treatment status of the participants, counted each question asked in the video clips by both the participant and the conversational partner. In order to take into account the number of questions asked by the conversational partner, Questions Asked was defined as the percentage of questions asked by the participant during conversation.

Positive Facial Expressions: Individuals with ASD commonly have deficits in appropriate affective expression. Smiling and laughter have long been established as important

nonverbal indicators of social attunement in conversation and are generally associated with friendliness and positive impressions. Nonverbal social engagement was measured through observed efforts to convey interest in a social partner's conversation (positive facial expressions). A five-second partial interval coding scheme was implemented to code for the presence or absence of a positive facial expression (defined as visible smiling or laughing). A percentage was calculated to determine the percentage of time the participant was displaying positive facial expressions during each 5-min clip.

Mutual Engagement: Challenges with social reciprocity are a hallmark characteristic of ASD. To measure changes in reciprocity, mutual engagement, or the extent to which both social partners were jointly engaging in conversation, was examined to explore the balance of conversational contributions. This measure specifically examined the contributions of a participant relative to their conversational partners. Coders examined five-second intervals and focused on whether (a) the participant was the primary speaker, (b) the conversational partner was the primary speaker, (c) both partners contributed equally to the conversational volley, or (d) no one spoke. Mutual engagement was defined as the percentage of intervals in which both individuals contributed equally to the conversation.

Qualifications of Treatment Providers

Trained undergraduate research assistants served as the social facilitators for the START program. High school volunteers were also recruited from local schools to serve as peer mentors. All facilitators received an initial 10-h training on basic group facilitation techniques, covering basic group facilitation skills, methods for fostering rapport, and exposure to practice group sessions. The social facilitators also participated in weekly 1-h supervision meetings for ongoing clinical training purposes. One undergraduate was assigned as the designated primary social facilitator for each participant and was responsible for all check-in and checkout sessions, along with all progress meetings

with that individual. Advanced clinical psychology doctoral students and a licensed clinical psychologist jointly conducted all training and supervision sessions.

See Also

- [Social Interventions](#)
- [Social Responsiveness Scale](#)
- [Social Skills Improvement System](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5*. Washington, DC: American Psychiatric Association.
- Baker, A. C., Jensen, P. J., & Kolb, D. A. (2002). *Conversational learning: An experiential approach to knowledge creation*. Portsmouth: Greenwood Publishing Group.
- Chevallier, C., Kohls, G., Troiani, V., Brodtkin, E. S., & Schultz, R. T. (2012). The social motivation theory of autism. *Trends in Cognitive Sciences*, 16(4), 231–239.
- Constantino, J., & Gruber, C. (2005). *Social responsiveness scale*. Los Angeles: Western Psychological Services.
- Dawson, G., Webb, S. J., & McPartland, J. (2005). Understanding the nature of face processing impairment in autism: Insights from behavioral and electrophysiological studies. *Developmental Neuropsychology*, 27(3), 403–424.
- Gresham, F. M., & Elliott, S. N. (2008). *Social Skills Improvement System (SSIS) rating scales*. Minneapolis: Pearson.
- Ko, J. A., Miller, A. R., & Vernon, T. W. (2018). Social conversation skill improvements associated with the START program for adolescents with ASD: Results of a randomized controlled trial. *Autism*. Online First Publication.
- Kolb, D. A. (2014). *Experiential learning: Experience as the source of learning and development* (2nd ed.). Upper Saddle River: Pearson Education.
- Kolb, D. A., Boyatzis, R. E., & Mainemelis, C. (2001). Experiential learning theory: Previous research and new directions. In R. J. Sternberg & L. F. Zhang (Eds.), *Perspectives on thinking, learning, and cognitive styles* (pp. 227–247). Mahwah: Lawrence Erlbaum.
- Locke, J., Ishijima, E. H., Kasari, C., & London, N. (2010). Loneliness, friendship quality and the social networks of adolescents with high-functioning autism in an inclusive school setting. *Journal of Research in Special Educational Needs*, 10(2), 74–81.
- Mazurek, M. O. (2013). Loneliness, friendship, and Well-being in adults with autism spectrum disorders. *Autism*. <https://doi.org/10.1177/1362361312474121>.

- Miller, A. R., Vernon, T. W., Wu, V., & Russo, K. (2014). Social skill group interventions for adolescents with autism spectrum disorders: A systematic review. *Review Journal of Autism and Developmental Disorders*, 1(4), 254–265.
- Orsmond, G. I., Krauss, M. W., & Seltzer, M. M. (2004). Peer relationships and social and recreational activities among adolescents and adults with autism. *Journal of Autism and Developmental Disorders*, 34(3), 245–256.
- Schmidt, C., Stichter, J. P., Lierheimer, K., McGhee, S., & O'Connor, K. V. (2011). An initial investigation of the generalization of a school-based social competence intervention for youth with high-functioning autism. *Autism Research and Treatment*, 2011, 1–11.
- Schroeder, J. H., Cappadocia, M. C., Bebko, J. M., Pepler, D. J., & Weiss, J. A. (2014). Shedding light on a pervasive problem: A review of research on bullying experiences among children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(7), 1520–1534.
- Vernon, T. W., Miller, A. R., Ko, J. A., Barrett, A., & McGarry, E. (2018). A randomized controlled trial of the Social Tools and Rules for Teens (START) program: An immersive socialization intervention for adolescents with ASD. *Journal of Autism and Developmental Disorders*, 48(3), 892–904.
- Vernon, T. W., Miller, A. R., Ko, J. A., & Wu, V. (2016). Social Tools and Rules for Teens (The START program): Program description and preliminary outcomes of a multi-component socialization intervention for adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(5), 1806–1823.
- Watkins, L., O'Reilly, M., Kuhn, M., Gevarter, C., Lancioni, G. E., Sigafoos, J., & Lang, R. (2015). A review of peer-mediated social interaction interventions for students with autism in inclusive settings. *Journal of Autism and Developmental Disorders*, 45(4), 1070–1083.

Social Validity

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Definition

The assessment of social validity involves measures that evaluate the social acceptability of an intervention or program (Kazdin 1977). As it applies to the application of techniques derived

from the science of applied behavior analysis (ABA), the assessment of social validity is defined as measuring the social acceptability of goals, social appropriateness of procedures, and the social importance of the effects of a treatment or intervention (Wolf 1978). Measures of social validity are not meant to be the primary dependent measures of an intervention, nor used to evaluate the effectiveness of a treatment or intervention (Schwartz and Baer 1991). Instead, the measurement of social validity is intended to provide supplemental data to assess the acceptability of an intervention by relevant consumers (Wolf 1978).

Social validity assessment is a two-part process involving (1) obtaining accurate information about consumers' opinions on an intervention or program and (2) using that information to sustain or change an intervention or program (Schwartz and Baer 1991). Based upon this process, there are several ways to assess and measure social validity. Some general techniques to assess social validity are through the use of interviews, questionnaires, surveys, rating scales, discussions with consumers, direct observation, consumer choice of intervention, and comparison of behavior to a normative population (Carter 2010; Schwartz and Baer 1991). Interviews, questionnaires, surveys, rating scales, and discussions are more subjective measures of social validity. Direct observation of behavior, consumer choice of intervention, and comparison of behavior to a normative population are more objective forms of assessing social validity. These methods of measuring social validity may provide data on all three aspects of the social validity of an intervention that Wolf (1978) outlined (i.e., goals, procedures, effects) or only one or two aspects of an intervention (e.g., assessing the social validity of the treatment effects). Selecting the consumer who will respond to assessments of social validity (e.g., interviews, surveys) is another important aspect of the social validation process. Consumers who could be asked about the social validity of an intervention could be direct consumers (i.e., the individual participating in the intervention), indirect consumers (i.e., those individuals who are strongly impacted by the intervention), members

of the immediate community (i.e., individuals who interact with either direct or indirect consumers), or members of the extended community (i.e., those who live in the community with the direct or indirect consumers; Schwartz and Baer 1991).

Historical Background

ABA is the scientific application of the principles of behavior analysis to socially significant problems, as was defined by the authors of the first seminal article in the *Journal of Applied Behavior Analysis* (JABA; Baer et al. 1968). Within this seminal article, the authors outlined seven dimensions of the science of ABA; the first two described being applied and behavioral (Baer et al. 1968). To uphold the applied dimension of ABA the authors stated that the science would address problems that are socially significant to society. To uphold the behavioral dimension of ABA, our science seeks to change behavior that is observable and measurable. Baer et al. (1968) stated that the behavioral dimension was of critical importance to the science of ABA and urged the field to avoid the use of subjective measures of behavior, such as verbal reports of behavior and feelings. Since ABA is a science of behavior, Montrose Wolf understood the importance of observable and measurable behavior and grew concerned as the editors of JABA began to publish studies which included subjective measurements in addition to objective and measurable changes in behavior (Wolf 1978). But, as the journal progressed, more and more publications included subjective measures (Wolf 1978). Wolf grew concerned but found solace in the applied dimension of ABA. If the goal of JABA was to publish studies that address problems of social importance, how would the field decide which behaviors, procedures, and results were socially important to the consumers? Wolf noted that in order for ABA to uphold the applied dimension of our science, behavior analysts would need to use subjective measurement to assess social validity (Wolf 1978). In 1987, Baer, Wolf, and Risley wrote another seminal paper entitled “Some still

current dimensions of applied behavior analysis” which reiterated the seven dimensions outlined in 1968. In this paper, social validity was mentioned several times as a way to evaluate if ABA, as a science, is supporting the dimensions of practice. The authors further stated, having “valid social-validity assessments will soon become crucial to survival [of our science]” (Baer et al. 1987, p. 323).

Current Knowledge

Currently, there have been two literature reviews assessing the use of social validity within JABA. Kennedy (1992) analyzed articles published in JABA from 1968 to 1990 and articles published in *Behavior Modification* from 1977 to 1990 for the frequency and types of social validity measurements used within the two journals. Kennedy found that the measurement of social validity rarely occurred within JABA between the years 1968 and 1975, but increased in the mid-1970s. The measurement of social validity within JABA peaked in 1983 and then decreased to a steady rate (i.e., 20% of articles reporting social validity measures) throughout the late 1980s. Similar trends in the journal *Behavior Modification* were also found. Carr et al. (1999) extended Kennedy’s work and evaluated social validity trends in JABA through 1998 (Carr et al. 1999). Carr et al. found similar trends through the 1990s, with approximately 25% of articles published in JABA reporting measures of social validity. Taken together, the results of these reviews show that there has been a lack of research reporting social validity measures within behavior analytic research.

Research on the process of social validity has mainly focused on how to assess the social appropriateness of behaviorally based treatments (Carter 2010). The majority of the research on this process has occurred around the development and evaluation of the Treatment Evaluation Inventory (TEI; Kazdin 1980). The TEI has 16 items which are rated on a Likert type scale from 1 to 7 (Kazdin 1980). Originally, this tool was evaluated with 88 participants and each participant was

given case descriptions which she/he then rated using the TEI. Kazdin found that when several different treatment scenarios were described to the evaluators, they rated the interventions differentially across several aspects including questions about how likely they would be to implement the treatment, how unfair they thought the treatment was, and how acceptable they thought the treatment was for the participants. Analyses were conducted in order to evaluate the strength of the relationship between the acceptability ratings and the treatment scenarios (Kazdin 1980). The TEI was found to account for 61% of the differences in the acceptability ratings across treatments and the author concluded that the strength of the treatment effect was relatively large (Kazdin 1980). The TEI has the most research to support the validity of its use and continues to be evaluated and utilized today (e.g., Diller et al. 2013).

Future Directions

Given the paucity of research on the assessment of social validity, the future directions are not that different from those outlined by Schwartz and Baer in 1991. Schwartz and Baer (1991) stated that the future directions of social validity were threefold, and these directions are still applicable today. First, more researchers and clinicians need to conduct social validity assessments and report the results. Based upon previous reviews, social validity measures within research published in JABA has been reported in about 20% of articles across the past 30 years (Carr et al. 1999; Kennedy 1992). Furthermore, these reviews found that the overall trend of articles publishing measures of social validity is not increasing in our flagship journal (i.e., JABA). Even though the probability of research published in JABA reporting social validity measures is low, it may not mean research within the treatment of ASD as a whole is not. JABA is a small sample with respect to research conducted on the treatment of ASD. In addition to striving to increase the assessment and reports of social validity within research, behavior analysts could also measure

and report social validity measures in our clinical practice and report the findings outside of research journals.

The second future direction stated by Schwartz and Baer (1991) was that a greater number and types of consumers should be asked to report on social validity. Currently, direct consumers and indirect consumers closely involved with the participants of the intervention are the ones commonly involved in the assessment of social validity. Less commonly included consumers are members of the immediate and extended community. Including immediate and extended community members in the assessment of the social validity of our interventions could have potential benefits for how our science is perceived by others and how our procedures become mainstream (Friman 2014).

Third, Schwartz and Baer (1991) suggested that future research on the topic of social validity should evaluate objective precursors associated with the social invalidity and validity of a program. This suggestion remains relevant today. More research is required evaluating the predictors of the social validity or invalidity of a program or intervention because the effectiveness of an intervention is not enough for the widespread adoption of behavior analytic programs. The lack of the adoption of Direct Instruction within public schools after the program was evaluated through Project Follow Through (Lindsley 1992; Morrell 1998; Watkins 1997) is a prime example. Although Direct Instruction and the behavior analytic teaching models were found to be the most effective forms of education, these procedures were not adopted by the schools and the procedures are rarely implemented by teachers (Watkins 1997). Why would these procedures not be adopted and widely used if they were proven to be the most effective? One reason is that the social acceptability of these procedures was not initially assessed. Assessing the social validity of Direct Instruction prior to nationwide implementation could have provided much needed information on how the model was perceived by teachers, administrators, and students. Stallings (1974) found that the teachers who implemented Direct Instruction were the least satisfied with the program. This critical information

could have been utilized to inform improvements to increase the social acceptability of Direct Instruction. The assessment of social validity is vital to the survival of our science. To date, there continues to be a lack of reporting on social validity measures within applied behavior analytic research. If the techniques derived from our science are to become a mainstay of our society, we need to increase both the assessment and reporting of social validity measures.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Ecological Validity](#)
- [Generalization and Maintenance](#)

References and Reading

- Baer, D. M., Wolf, M. M., & Risley, T. R. (1968). Some current dimensions of applied behavior analysis. *Journal of Applied Behavior Analysis*, 1(1), 91–97.
- Baer, D. M., Wolf, M. M., & Risley, T. R. (1987). Some still-current dimensions of applied behavior analysis. *Journal of Applied Behavior Analysis*, 20(4), 313–327.
- Carr, J. E., Austin, J. L., Britton, L. N., Kellum, K. K., & Bailey, J. S. (1999). An assessment of social validity trends in applied behavior analysis. *Behavioral Interventions*, 14(4), 223–231.
- Carter, S. L. (2010). *The social validity manual: A guide to subjective evaluation of behavior interventions*. London: Elsevier.
- Diller, J. W., Brown, R. M., & Patros, C. H. G. (2013). Revisiting Kazdin (1980): Contemporary treatment acceptability for problem behavior in children. *International Journal of Psychology & Psychological Therapy*, 13(2), 225–231.
- Friman, P. C. (2014). Publishing in journals outside the box: Attaining mainstream prominence requires demonstrations of mainstream relevance. *The Behavior Analyst*, 37, 73–76.
- Kazdin, A. E. (1977). Assessing the clinical or applied importance of behavior change through social validation. *Behavior Modification*, 1(4), 427–451.
- Kazdin, A. E. (1980). Acceptability of alternative treatments for deviant child behavior. *Journal of Applied Behavior Analysis*, 13(2), 259–273.
- Kennedy, C. H. (1992). Trends in the measurement of social validity. *The Behavior Analyst*, 15(2), 147–156.
- Lindsley, O. R. (1992). Why aren't effective teaching tools widely adopted? *Journal of Applied Behavior Analysis*, 25(1), 21–26.
- Morrell, R. F. (1998). Project follow through: Still ignored. *American Psychologist*, 53(3), 318–318.
- Schwartz, I. S., & Baer, D. M. (1991). Social validity assessments: Is current practice state of the art? *Journal of Applied Behavior Analysis*, 24(2), 189–204.
- Stallings, J. A. (1974). An implementation study of seven follow through models for education. Paper Presented at the Annual Meeting of the American Educational Research Association, Chicago
- Watkins, C. L. (1997). *Project follow through: A case study of contingencies influencing instructional practices of the educational establishment*. Cambridge, MA: Cambridge Center for Behavioral Studies.
- Wolf, M. M. (1978). Social validity: The case for subjective measurement or how applied behavior analysis is finding its heart. *Journal of Applied Behavior Analysis*, 11(2), 203–214.

Social-Emotional Behavior and Autism Spectrum Disorder

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Definition

Social-emotional (SE) development refers to the capacity to experience, express, and manage one's emotions, as well as the ability to understand and respond to the emotional states of others. In the first few years of life, children rapidly develop social and emotional competencies that allow them to navigate increasingly complex social interactions, including establishing and maintaining relationships with others. These skills are important contributors to emotional well-being and psychological health in adulthood (Frijda 1986; Ekman 1992; Saarni et al. 2006). Many of these SE skills, however, are challenging for children with autism spectrum disorder

(ASD). That is, children with ASD often experience difficulties in SE reciprocity – such as emotional expressiveness, regulation, and responsiveness – contributing to their difficulties in engaging appropriately in social situations (American Psychiatric Association 2013; Sigman and Capps 1997).

Emotional and social development are intertwined and affect how one makes social connections and responds to others (Frijda 1986; Ekman 1992; Saarni et al. 2006; Darwin et al. 1998; Shariff and Tracy 2011; Cole and Moore 2015). Emotions can be understood as dynamic, social processes, both created by and resultant from social relationships with others (Salovey 2003). During typical development, the expression of emotions is regarded as the first social-communicative action of infants, with important implications for later competence. For example, emotional expressiveness in infancy evokes responses from caregivers and invites them to share emotional states, resulting in SE reciprocity. By the second year of life, intentional communication skills develop, allowing toddlers to engage in more complex SE behavior to share experiences with others (Mundy 1995). As infants and toddlers are particularly attuned to social and emotional stimulation early in life, the degree of scaffolding and responsiveness by caregivers help to regulate these emotions and greatly impact subsequent SE growth and development (Carter et al. 2004; Feldman 2007).

SE capacities develop from both intra- and interpersonal processes. Intrapersonal SE behavior includes the ability to identify and understand one's own emotions, to manage and communicate emotions, and to regulate one's own behavior. Interpersonal aspects of SE behavior involve the ability to accurately identify and interpret the emotional states of others, as well as the capacity to empathize (Harris 1989; Frith 1994; Denham 1998; Thompson and Lagattuta 2006). In building these SE skills, children are learning how to establish and maintain relationships with family, peers, and the community to promote successful participation in reciprocal social interactions and navigation of complex social environments (Skinner 2018).

Historical Background

Difficulties in SE and affective behavior have been noted in Leo Kanner's (1943) original observations of 11 cases of autism, where he defined ASD as a biological impairment in the capacity for social relatedness. Kanner described children with ASD as "hav[ing] come into the world with innate inability to form the usual, biologically provided affective contact with people, just as other children come into the world with innate physical or intellectual handicaps." He noted that these children were emotionally detached, aloof, and unresponsive – many of whom were unable "to relate themselves in the ordinary way to people and situations from the beginning of life." Kanner's (1943) original account highlights the role of SE behavior in the emerging ASD phenotype, pointing to a failure of mechanisms to perceive, express, respond to, and understand social and emotional events.

Evidence of the ASD-related SE impairments in ASD is largely confined to retrospective parental reports and early home video review due to the later age of diagnosis (still at the age 4–5 mark; Brett et al. 2014; Christensen et al. 2016). As many as 50% of parents recall SE difficulties within the first year of life, including marked displays of temperament and behavioral reactivity and lack of responsiveness to attempts of social interaction and play (Ohta et al. 1987; Dahlgren and Gillberg 1989; De Giacomo and Fombonne 1998). Moreover, during the second year, parents have retrospectively reported a pattern of regression involving loss of speech and/or SE connectedness in approximately 20–50% of children with ASD (Kurita 1985; Hoshino et al. 1987; Rutter and Lord 1987; Werner and Dawson 2005). Similarly, analyses of early home videotapes have revealed that children later diagnosed with ASD show atypical emotional development around the time of their first birthday. Findings include increased negative affect, ambiguous emotional expressions, and atypical affect regulation (Adrien et al. 1991; Baranek 1999; Maestro et al. 2005).

Current Knowledge

Early difficulties in SE behavior play a role in the emerging phenotype of ASD. Many of the first studies examining SE development in children with ASD have focused primarily on emotional expressiveness and responsiveness. These retrospective studies indicated that although children with ASD did not appear less emotionally expressive than children with typical development (TD) or mental retardation (Ricks and Wing 1975), they did experience higher levels of negative emotions and lower levels of positive emotions (Capps et al. 1993). Other studies investigated whether emotional expressions and responses of children with ASD were socially and contextually appropriate. Compared to children with TD or mental retardation, children with ASD showed deficits in emotional expression and sharing of emotional experience, such as reduced positive affect and coordination of gaze with emotion expression to share experiences (Yirmiya et al. 1989; Kasari et al. 1990, 1993). Furthermore, prospective studies of children later diagnosed with ASD have noted that the paucity of SE reciprocity, combined with delays in verbal and non-verbal communication, contributes to increased negative affect and distress, reduced positive affect, and low levels of regulation (Gomez and Baird 2005; Zwaigenbaum et al. 2005; Bryson et al. 2007; Clifford et al. 2007; Watson et al. 2007; Garon et al. 2009, 2016). These atypicalities are apparent in the first years of life of children who go on to be diagnosed with ASD, with upward of 30% of parents reporting SE concerns by their child's first birthday (De Giacomo and Fombonne 1998; Sacrey et al. 2015).

The recent application of prospective studies of infant siblings of children diagnosed with ASD, the so-called "high-risk" sibling (having a higher probability of also being diagnosed with ASD, at about 18% (Ozonoff et al. 2011), compared to the general population at 1–2% (Baio et al. 2018)) has allowed a more in-depth examination of SE behavior as it develops. A large study of

331 high-risk infant siblings and 133 infants of families without a history of ASD used the Infant Toddler Social-Emotional Assessment (ITSEA; described below) to collect information on SE behavior when the children were 18 months of age (Raza et al. 2019). The HR infant siblings who were diagnosed with ASD at age 3 showed a higher prevalence of internalizing (depression, anxiety) and dysregulation (negative emotionality) concerns and a lower number of competencies (empathy, prosocial peer relations) compared to other high-risk infant siblings who were not diagnosed with ASD and comparison infants. Furthermore, these behaviors at 18 months were associated with total severity scores on the Autism Diagnostic Interview and the Autism Diagnostic Observation Scale at age 3 (Raza et al. 2019). Using a Brief version of the ITSEA (BITSEA; described below), Kiss et al. (2017) compared SE behavior in preschool children with and without ASD and developed three new scales for use in ASD screening (the ASD Problems, ASD-Competence, and ASD-Total subdomains). The ASD-Competence scale best discriminated ASD from TD and other developmental delays (0.91 for sensitivity, 0.80 for specificity).

In line with these findings, examination of SE behavior in school-aged children diagnosed with ASD also highlight the higher prevalence of SE atypicalities compared to TD peers. Intellectually delayed children with ASD have more neutral or idiosyncratic expressions (e.g., smiling when not appropriate to the context) compared to TD peers (Capps et al. 1993) and a higher prevalence of emotional disorders than children with an intellectual disability without ASD (71% vs. 42% for emotional problems and 65% vs. 46% for conduct problems, respectively; Totsika et al. 2011). In contrast, intellectually able children with ASD show a similar range of expressiveness as TD peers, yet they also display less positive affect (Capps et al. 1993) and have higher levels of emotional problems (74% vs. 18%, respectively) and conduct problems (64% vs. 22%, respectively) than their TD peers (Totsika et al. 2011).

Impaired SE behavior has been implicated in a range of co-occurring and/or comorbid problem behavior in children with ASD. It is estimated that up to 50% of children with ASD meet clinical criteria for two or more SE disorders, even after controlling for the social-communication and repetitive behavior/restrictive interest core diagnostic criteria (Leyfer et al. 2006; Simonoff et al. 2008). Depression and anxiety are often reported to be the most frequent SE comorbid diagnosis (Howlin 2000). A recent meta-analysis (Lugo-Marin et al. 2019) of 47 studies examining the prevalence of SE disorders in adults with ASD found that 18.8% were also diagnosed with a mood disorder (depression and bipolar most prevalent), 17.8% were also diagnosed with an anxiety disorder (social anxiety disorder, obsessive compulsive disorder, and adjustment disorder most prevalent), and 54.8% were diagnosed with any psychiatric disorder (including substance use disorder, schizophrenia spectrum disorder, mood disorder, anxiety disorder, eating disorder, personality disorder, and attention deficit hyperactivity disorder).

Thus, it is fitting that deficits in SE reciprocity are included in the diagnostic criteria of ASD in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association 2013) and the International Classification of Diseases-11 (World Health Organization 2018). Because SE impairments appear early in life and impact developing social and language skills (Carpenter and Tomasello 2000; Mundy and Sigman 2006; Woods and Wetherby 2003), assessment tools that probe behaviors characterizing ASD, such as the Autism Diagnostic Interview-Revised (ADI-R; Rutter et al. 2003) and the Autism Diagnostic Observation Scale-2 (ADOS-2; Lord et al. 2000), also include probes for SE behavior.

Measuring Social-Emotional Behavior

Over the past few decades, researchers have emphasized the importance of identifying young children at risk for delays in SE behavior. To fully understand SE development in infants and

toddlers, assessments of both problem behavior and competencies are crucial (Briggs-Gowan and Carter 1998; Carter et al. 2003; Carter and Briggs-Gowan 2006). Competencies include behavior that reflect achievement of age-appropriate skills in SE development – such as attention, empathy, emotional awareness, and prosocial peer interactions, to name a few (Radke-Yarrow and Zahn-Waxler 1984; Saarni 1988; Eisenberg and Mussen 1989; Zahn-Waxler et al. 1992; Briggs-Gowan and Carter 1998). Assessments of SE competencies are important for understanding a child's ability to meet new developmental demands, the likelihood of later competence, and the risk for subsequent SE difficulties (Denham et al. 1990; Cicchetti 1993; Cicchetti and Cohen 1995; Hay et al. 1995; Masten and Coatsworth 1995, 1998; Carter 2002). SE problems, on the other hand, are comprised of behavior that reflects a failure to achieve age-appropriate SE competencies and may interfere with aspects of a child's functioning. This includes behavior that occurs in TD but has become problematic (i.e., atypical expression, frequency, and/or intensity), as well as behavior that deviates from the normative developmental course. Behavior traditionally characterized as externalizing and internalizing problems (Achenbach 1966), regulatory problems, and maladaptive behavior encompass SE problems (Briggs-Gowan and Carter 1998; Carter et al. 2003; Carter and Briggs-Gowan 2006).

With these constructs in mind, the ITSEA and BITSEA were developed to measure SE behavior in young children. The ITSEA is a parent-reported questionnaire for children between 12 and 36 months of age that profiles problem behavior and competencies (Carter and Briggs-Gowan 2006). The ITSEA is comprised of 190 questions querying four domains of SE behavior: externalizing (e.g., aggression), internalizing (e.g., anxiety), dysregulation (e.g., negative emotionality), and competence (e.g., empathy). The ITSEA was shortened to the BITSEA (Briggs-Gowan and Carter 2002). This version is comprised of 42 questions covering SE problem behavior and competencies, as well as a new ASD domain that

is designed to identify children with SE difficulties that may require additional follow-up for ASD.

Other assessments that are not specific for SE behavior but have been used to collect information on SE development and related atypicalities include the Battelle Personal-Social Domain (Newborg et al. 1984), the Child Behavior Checklist (CBCL2/3; Achenbach et al. 1987), the Infant-Toddler Developmental Assessment (IDA; Provence et al. 1995), the Toddler Behavior Checklist (Mouton-Simien et al. 1997), and the Vineland Adaptive Behavior Scales (VABS; Sparrow et al. 1984).

Future Directions

Substantial progress has been made in the conceptualization of SE development in children with ASD. Growing evidence suggests that impairments in SE development may limit functional outcomes for children with ASD and give rise to a range of co-occurring and/or comorbid problem behavior (Gadow et al. 2004; Brereton et al. 2006; Witwer and Lecavalier 2008). Up to 50% of children with ASD meet clinical criteria for two or more SE disorders, the underpinnings of which appear very early in childhood (Raza et al. 2019; Kiss et al. 2017). As such, early detection of emerging SE atypicalities is warranted to promote healthy developmental outcomes and successful school adaptation.

To this end, a greater emphasis on exploring the associations between SE behavior and ASD symptom presentation is crucial. Investigating this relationship can improve our understanding of the mechanisms underlying ASD etiology, phenotypic overlap between core diagnostic symptoms, and comorbid psychopathologies, as well as clarify the role SE development plays in functional outcomes of individuals with ASD. This may contribute to early identification and diagnosis of ASD and inform the development of SE interventions and treatment approaches, with broader implications for

policies and mental health services that aim to improve the quality of life for individuals with ASD.

What can we do to achieve these goals? Adequately powered longitudinal studies are needed to elucidate how SE atypicalities increase the risk of comorbid psychiatric disorders and outcomes in individuals with ASD. Additionally, cohort studies following both high-risk infants and early diagnosed children into adolescence and beyond may provide a clearer picture of SE trajectories, as well as identify clusters of behaviors that are predictive of specific comorbid diagnoses (e.g., anxiety, ADHD). Finally, integrating information across several methodologies and assessments (i.e., combinations of behavioral observation, physiological measurement, and parent-report) could provide a holistic picture of SE processes to promote healthy development and positive mental health and establish a strong foundation for school readiness and future success.

See Also

- ▶ [Affective Development](#)
- ▶ [Brief Infant-Toddler Social and Emotional Assessment \(BITSEA\)](#)
- ▶ [Emotional Processing in ASD](#)
- ▶ [Emotional Regulation](#)
- ▶ [Infant-Toddler Social and Emotional Assessment \(ITSEA\)](#)
- ▶ [Mutual Regulation](#)
- ▶ [Predictors of Emotion Regulation in Children with Autism Spectrum Disorder \(ASD\)](#)
- ▶ [Temperament](#)

References and Reading

- Achenbach, T. M. (1966). The classification of children's psychiatric symptoms: A factor-analytic study. *Psychological Monographs*, 80, 615.
- Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, 101, 213–232.

- Adrien, J. L., Faure, M., Perrot, A., Hameury, L., Garreau, B., Barthelemy, C., & Sauvage, D. (1991). Autism and family home movies: Preliminary findings. *Journal of Autism and Developmental Disorders*, 21(1), 43–49.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Publishing.
- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., et al. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 Sites, United States, 2014. *Surveillance Summaries*, 67(6), 1–23.
- Baranek, G. T. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders*, 29, 213–224.
- Brereton, A., Tonge, B., & Einfeld, S. (2006). Psychopathology in children and adolescents with autism compared to young people with intellectual disability. *Journal of Autism Developmental Disorders*, 36, 863–870.
- Brett, D., Warnell, F., McConachie, H., & Parr, J. R. (2014). Factors affecting age at ASD diagnosis in UK: No evidence that diagnosis age has decreased between 2004 and 2014. *Journal of Autism and Developmental Disorders*, 46(6), 1974–1984.
- Briggs-Gowan, M. J., & Carter, A. S. (1998). Preliminary acceptability and psychometrics of the infant-toddler social and emotional assessment (ITSEA): A new adult-report questionnaire. *Infant Mental Health Journal*, 19, 422–445.
- Briggs-Gowan, M. J., & Carter, A. S. (2002). *Brief infant-toddler social and emotional assessment (BITSEA) manual (version 2.0)*. New Haven: Yale University.
- Bryson, S., Zwaigenbaum, L., Brian, J., Roberts, W., Szatmari, P., Rombough, V., & McDermott, C. (2007). A prospective case series of high-risk infants who developed autism. *Journal of Autism and Developmental Disorders*, 37, 12–24.
- Capps, L., Kasari, C., Yirmiya, N., & Sigman, M. (1993). Parental perception of emotional expressiveness in children with autism. *Journal of Consulting and Clinical Psychology*, 61, 475–484.
- Carpenter, M., & Tomasello, M. (2000). Joint attention, cultural learning, and language acquisition: Implications for children with autism. In A. M. Wetherby & B. M. Prizant (Eds.), *Autism spectrum disorders: A transactional developmental perspective* (Communication and language intervention series, pp. 21–54). Baltimore: Brookes Publishing.
- Carter, A. S. (2002). Assessing social-emotional and behavior problems and competencies in infancy and toddlerhood: Available instruments and directions for application. In B. Zuckerman, A. Lieberman, & N. Fox (Eds.), *Emotion regulation and developmental health: Infancy and early childhood* (pp. 277–299). New York: Johnson & Johnson Pediatric Institute.
- Carter, A. S., & Briggs-Gowan, M. J. (2006). *The infant-toddler social and emotional assessment (ITSEA) manual*. San Antonio: Harcourt Assessment.
- Carter, A. S., Briggs-Gowan, M. J., Jones, S. M., & Little, T. D. (2003). The infant-toddler social and emotional assessment (ITSEA): Factor structure, reliability, and validity. *Journal of Abnormal Child Psychology*, 31, 495–514.
- Carter, A. S., Briggs-Gowan, M. J., & Davis, N. O. (2004). Assessment of young children's social-emotional development and psychopathology: Recent advances and recommendations for practice. *Journal of Child Psychology and Psychiatry*, 45(1), 109–134.
- Christensen, D. L., Baio, J., Van Naarden Braun, K., Bilder, D., Charles, J., Constantino, J. N., et al. (2016). Prevalence and characteristics of autism spectrum disorder among children aged 8 years-autism and developmental disabilities monitoring network, 11 Sites, United States, 2012. *Morbidity and Mortality Weekly Reports Surveillance Summaries*, 65(3), 1–23.
- Cicchetti, D. (1993). Developmental psychology: Reactions, reflections, and projections. *Developmental Review*, 13, 471–502.
- Cicchetti, D., & Cohen, D. J. (1995). Perspectives on developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Vol. 1. Theory and methods* (pp. 3–20). New York: Wiley.
- Clifford, S., Young, R., & Williamson, P. (2007). Assessing the early characteristics of autistic disorder using video analysis. *Journal of Autism and Developmental Disorders*, 37, 301–313.
- Cole, P. M., & Moore, G. A. (2015). About face! Infant facial expression of emotion. *Emotion Review*, 7(2), 116–120.
- Dahlgren, S. O., & Gillberg, C. (1989). Symptoms in the first two years of life. A preliminary population study of infantile autism. *European Archives of Psychiatry and Neurological Sciences*, 238, 169–174.
- Darwin, C., Ekman, P., & Prodger, P. (1998). *The expression of the emotions in man and animals*. Oxford: Oxford University Press.
- De Giacomo, A., & Fombonne, E. (1998). Parental recognition of developmental abnormalities in autism. *European Child & Adolescent Psychiatry*, 7(3), 131–136.
- Denham, S. (1998). *Emotional development in young children*. New York: Guilford.
- Denham, S. A., McKinley, M., Couchoud, E. A., & Holt, R. (1990). Emotional and behavioral predictors of preschool peer ratings. *Child Development*, 61, 1145–1152.
- Eisenberg, N., & Mussen, P. H. (1989). *The roots of pro-social behavior in children*. New York: Cambridge University Press.
- Ekman, P. (1992). Are there basic emotions? *Psychological Review*, 99, 550–553.

- Feldman, R. (2007). Parent-infant synchrony: Biological foundations and developmental outcomes. *Current Directions in Psychological Science*, 16(6), 340–345.
- Frijda, N. H. (1986). *The emotions*. Cambridge: Cambridge University Press.
- Frith, U. (1994). Autism and theory of mind in everyday life. *Social Development*, 3(2), 108–124.
- Gadow, K., DeVincent, C., Pomeroy, J., & Azizian, A. (2004). Psychiatric symptoms in preschool children with PDD and clinic and comparison samples. *Journal of Autism and Developmental Disorders*, 34, 379–393.
- Garon, N., Bryson, S., Zwaigenbaum, L., Smith, I. M., Brian, J., Roberts, W., & Szatmari, P. (2009). Temperament and its relationship to autistic symptoms in a high-risk infant sib cohort. *Journal of Abnormal Child Psychology*, 37, 59–78.
- Garon, N., Zwaigenbaum, L., Bryson, S., Smith, I. M., Brian, J., Roncadin, C., Valliancourt, T., Armstrong, V., Sacrey, L. R., & Roberts, W. (2016). Temperament and its association with autism symptoms in a high-risk population. *Journal of Abnormal Child Psychology*, 44, 757–769.
- Gomez, C., & Baird, S. (2005). Identifying early indicators for autism in self-regulation difficulties. *Focus on Autism and Other Developmental Disabilities*, 20, 106–116.
- Harris, P. L. (1989). *Children and Emotion: The development of psychological understanding*. Oxford: Blackwell.
- Hay, D. F., Castle, J., Stimson, C. A., & Davies, L. (1995). The social construction of character in toddlerhood. In M. Killen & D. Hart (Eds.), *Morality in everyday life: Developmental perspectives* (pp. 23–51). New York: Cambridge University Press.
- Hoshino, Y., Kaneko, M., Yashima, Y., Kumashiro, H., Volkmar, F. R., & Cohen, D. J. (1987). Clinical features of autistic children with setback course in their infancy. *Japanese Journal of Psychiatry and Neurology*, 41(2), 237–245.
- Howlin, P. (2000). Outcome in adult life for more able individuals with autism or Asperger syndrome. *Autism*, 4(1), 63–83.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kasari, C., Sigman, M., Mundy, P., & Yirmiya, N. (1990). Affective sharing in the context of joint attention interactions of normal, autistic, and mentally-retarded children. *Journal of Autism and Developmental Disorders*, 20(1), 87–100.
- Kasari, C., Sigman, M. D., Baumgartner, P., & Stipek, D. J. (1993). Pride and mastery in children with autism. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 34(3), 353–362.
- Kiss, I. G., Feldman, M. S., Sheldrick, R. C., & Carter, A. S. (2017). Developing autism screening criteria for the brief infant toddler social emotional assessment (BITSEA). *Journal of Autism and Developmental Disorders*, 47, 1269–1277.
- Kurita, H. (1985). Infantile autism with speech loss before the age of thirty months. *Journal of the American Academy of Child and Adolescent Psychiatry*, 24(2), 191–196.
- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., Tager-Flusberg, H., & Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36(7), 849–861.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., DiLavore, P. C., et al. (2000). The autism diagnostic observation schedule-generic: A standard measure of social and communicative deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 30, 205–223.
- Lugo-Marin, J., Magan-Maganto, M., Rivero-Santana, A., Cuellar-Pompa, L., Alviani, M., Jenaro-Rio, C., et al. (2019). Prevalence of psychiatric disorders in adults with autism spectrum disorder: A systematic review and meta-analysis. *Research in Autism Spectrum Disorders*, 59, 22–33.
- Maestro, S., Muratori, F., Cesari, A., Cavallaro, M. C., Paziente, A., Pecini, C., Grassi, C., Manfredi, A., & Sommaro, C. (2005). Course of autism signs in the first year of life. *Psychopathology*, 38(1), 26–31.
- Masten, A. S., & Coatsworth, J. D. (1995). Competence, resilience, and psychopathology. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology: Risk, disorder and adaptation* (Vol. 2, pp. 715–752). New York: John Wiley.
- Masten, A. S., & Coatsworth, J. D. (1998). The development of competence in favorable and unfavorable environments. *Lessons from research on successful children*. *American Psychologist*, 53, 205–220.
- Mouton-Simien, P., McCain, A. P., & Kelley, M. L. (1997). The development of the toddler behavior screening inventory. *Journal of Abnormal Child Psychology*, 25, 59–64.
- Mundy, P. (1995). Joint attention and social-emotional approach behavior in children with autism. *Development and Psychopathology*, 7, 63–82.
- Mundy, P., & Sigman, M. (2006). Joint attention social competence and developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Theory and method* (pp. 293–332). Hoboken: John Wiley and Sons.
- Newborg, J., Stock, J., Wnek, L., Guidubaldi, J., & Svinicki, J. (1984). *Battelle developmental inventory: Examiner's manual*. Allen: DMLINC Associates.
- Ohta, M., Nagai, Y., Hara, H., & Sasaki, M. (1987). Parental perception of behavioral symptoms in Japanese autistic children. *Journal of Autism and Developmental Disorders*, 17, 549–563.
- Ozonoff, S., Young, G. S., Carter, A., Messinger, D., Yirmiya, N., Zwaigenbaum, L., et al. (2011). Recurrence risk for autism spectrum disorders: A baby siblings research consortium study. *Pediatrics*, 128(3), e488–e495.

- Provence, S., Erickson, J., Vater, J., & Palmeri, S. (1995). *The infant-toddler developmental assessment (IDA)*. Chicago: The Riverside Publishing Company.
- Radke-Yarrow, M., & Zahn-Waxler, C. (1984). Roots, motives, and patterns and children's prosocial behavior. In E. Staub, D. Bar-Tal, J. Karylowski, & J. Reykowski (Eds.), *The development and maintenance of prosocial behavior: International perspectives on positive morality* (pp. 81–99). New York: Plenum.
- Raza, S., Sacrey, L. R., Zwaigenbaum, L., Bryson, S., Brian, J., Smith, I. M., Roberts, W., Szatmari, P., Vaillancourt, T., Roncadin, C., & Garon, N. (2019). Relationship between early social-emotional behavior and autism spectrum disorder: A high-risk sibling study. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03977-3>.
- Ricks, D. M., & Wing, L. (1975). Language, communication, and the use of symbols in normal and autistic children. *Journal of Autism and Childhood Schizophrenia*, 5, 191–221.
- Rutter, M., & Lord, C. (1987). Language disorders associated with psychiatric disturbance. In W. Yule & M. Rutter (Eds.), *Language development and disorders* (pp. 206–233). Philadelphia: JB Lippincott.
- Rutter, M., Le Couteur, A., & Lord, C. (2003). *Autism diagnostic interview—revised (ADI-R) manual*. Los Angeles: Western Psychological Services.
- Saarni, C. (1988). Emotional competence: How emotions and relationships become integrated. In R. A. Thompson (Ed.), *Nebraska symposium on motivation: Socio-emotional development: Current theory and research in motivation* (pp. 115–182). Lincoln: University of Nebraska Press.
- Saarni, C., Campos, J. J., Camras, L. A., & Witherington, D. (2006). Emotional development: Action, communication, and understanding. In W. Damon, R. M. Lerner, & N. Eisenberg (Eds.), *Handbook of child psychology: Social, emotional, and personality development* (6th ed., pp. 226–299). New York: John Wiley.
- Sacrey, L. R., Zwaigenbaum, L., Bryson, S., Brian, J., Smith, I. M., Roberts, W., Szatmari, P., Roncadin, C., Garon, N., Novak, C., Vaillancourt, T., McCormick, T., MacKinnon, B., Jildera, S., & Armstrong, V. (2015). Can parents' concerns predict autism spectrum disorder? A prospective study of high-risk siblings from 6 to 36 months of age. *Journal of the American Academy of Child & Adolescent Psychiatry*, 54, 470–478.
- Salovey, P. (2003). Introduction: Emotion and social processes. In R. Davidson, H. H. Goldsmith, & K. Scherer (Eds.), *The handbook of affective science* (pp. 747–751). Oxford: Oxford University Press.
- Shariff, A. F., & Tracy, J. L. (2011). What are emotion expressions for? *Current Directions in Psychological Science*, 20(6), 395–399.
- Sigman, M., & Capps, L. (1997). *Children with autism: A developmental perspective*. Cambridge, MA: Harvard University Press.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(8), 921–929.
- Skinner, E. (2018). Children's developmental needs during the transition to kindergarten: What can research on social-emotional, motivational, cognitive, and self-regulatory development tell us? In A. Mashburn, J. LoCasale-Crouch, & K. Pears (Eds.), *Kindergarten transition and readiness*. Cham: Springer.
- Sparrow, S. S., Balla, D. A., & Cicchetti, D. (1984). *Vineland adaptive behavior scales: Expanded form manual*. Circle Pines: American Guidance Service.
- Thompson, R. A., & Lagattuta, K. (2006). Feeling and understanding: Early emotional development. In K. McCartney & D. Phillips (Eds.), *The Blackwell handbook of early childhood development* (pp. 317–337). Oxford: Blackwell.
- Totsika, V., Hastings, R. P., Emerson, E., Lancaster, G. A., & Berridge, D. M. (2011). A population-based investigation of behavioural and emotional problems and maternal mental health: Associations with autism spectrum disorder and intellectual disability. *Journal of Child Psychology and Psychiatry*, 52(1), 91–99.
- Watson, L. R., Baranek, G. T., Crais, E. R., Reznick, S., Dystra, J., & Perryman, T. (2007). The first year inventory: Retrospective parent responses to a questionnaire designed to identify one-year olds at risk for autism. *Journal of Autism and Developmental Disorders*, 37, 49–61.
- Werner, E., & Dawson, G. (2005). Validation of the phenomenon of autistic regression using home videotapes. *Archives of General Psychiatry*, 62(8), 889–895.
- Witwer, A. N., & Lecavalier, L. (2008). Examining the validity of autism spectrum disorder subtypes. *Journal of Autism and Developmental Disorders*, 38, 1611–1624.
- Woods, J. J., & Wetherby, A. M. (2003). Early identification of and intervention for infants and toddlers who are at risk for autism spectrum disorder. *Language, Speech and Hearing Services in Schools*, 34, 180–193.
- World Health Organization. (2018). *International statistical classification of diseases and related health problems* (11th Revision). Retrieved from <https://icd.who.int/browse11/l-m/en>.
- Yirmiya, N., Kasari, C., Sigman, M., & Mundy, P. (1989). Facial expressions of affect in autistic, mentally retarded and normal-children. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 30(5), 725–735.
- Zahn-Waxler, C., Radke-Yarrow, M., Wagner, E., & Chapman, M. (1992). Development of concern for others. *Developmental Psychology*, 28, 126–136.
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, 23, 143–152.

Social-Emotional Inhibition of Return

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Definition

The term “inhibition of return (IOR)” refers to a cognitive mechanism that biases visual attention against previously explored locations in order to favor exploration of new areas. “Social-emotional IOR” specifically refers to alterations in the IOR mechanism by social-emotional cues, in that the saliency of such information is hypothesized to capture and hold attention to a greater degree, thus diminishing the IOR effect in the neurotypical population.

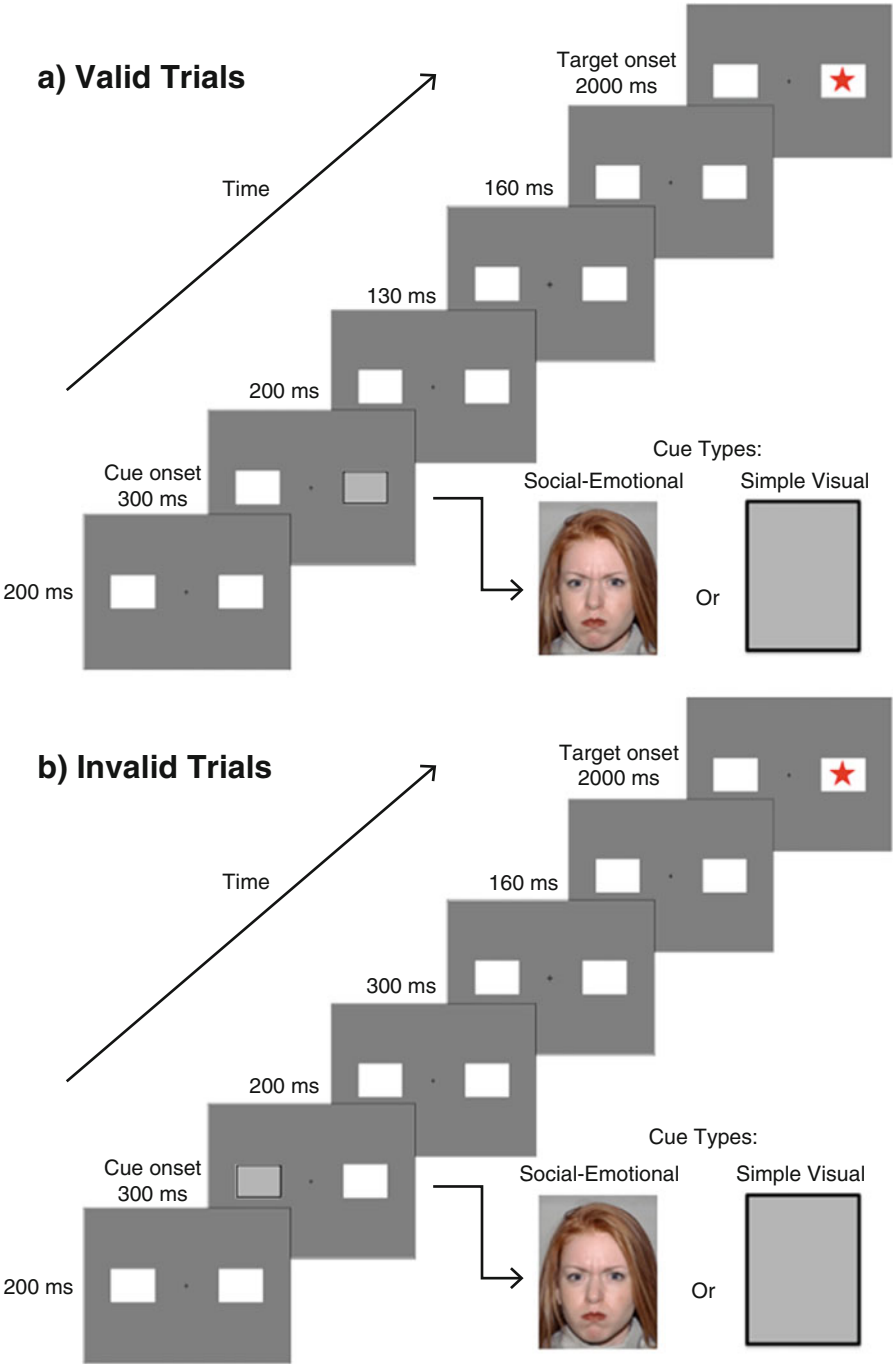
Historical Background

Visual attention is one modality in which individuals navigate their world and learn from it. Social-emotional signals are prioritized in visual orienting from an early age in neurotypical children, as this information can capture and hold attention to a greater degree than other information due to its saliency (Johnson et al. 1991; Klin et al. 2002; Nakano et al. 2010; Riby et al. 2012; Rice et al. 2012; Yerys et al. 2012). Multiple processes comprise visual attention, including the orienting process, which allows for disengagement, shifting, and reengagement of attention (Posner and Petersen 1990). The IOR mechanism specifically reflects an adaptive bias in visual

attention orienting, such that previously inspected spatial locations are inhibited in order to prioritize exploration of novel spatial locations. Further, this mechanism has been linked to facilitation of visual search, with alterations in IOR resulting in repetitive search of already inspected areas (Itti and Koch 2001; Klein 2000; Tipper et al. 1996; Wang and Klein 2010). Salient information, such as social-emotional cues, can override or diminish the IOR effect in order to prioritize this type of information for processing (Fox et al. 2002).

The IOR effect can be elicited and measured using a peripheral spatial cueing task in which participants are first presented with a cue (e.g., a flashing box or picture of a face to the left or right of center) followed by a target (e.g., an asterisk ‘*’) on either the same (Valid) or opposite side (Invalid) of the cue’s original location (Fig. 1). Previous work has demonstrated that the IOR effect is apparent when the interval between the cue and target (cue-target onset asynchrony; CTOA) is longer than 300 ms, such that participants make slower manual responses to validly cued trials than invalidly cued trials (IOR effect = Valid response time – Invalid response time; Posner et al. 1984).

Fox et al. (2002) demonstrated that angry schematic faces diminish the IOR effect in neurotypical young adults, such that the difference in response time between the validly cued trials and invalidly cued trials for angry faces is less than that for neutral faces. Similar findings have been replicated with ecologically valid social-emotional face cues (Pérez-Dueñas et al. 2014) and other nonsocial emotionally evocative cues (i.e., spiders, emotionally valenced words; Pérez-Dueñas et al. 2009; Rutherford and Raymond 2010). Further, greater anxiety and worry traits have been linked to a diminished social-emotional IOR effect (Verkuil et al. 2009). There is mixed evidence as to whether social-emotional signals diminish IOR (Stoyanova et al. 2007), though these conflicting results may be driven by task variability, which has been



Social-Emotional Inhibition of Return, Fig. 1 Peripheral spatial cueing paradigm. This paradigm is used to measure the IOR effect. Two trial types are depicted here: (a) Valid trials in which the cue and target appear on the same side and (b) Invalid trials in which the cue and target appear on opposite sides. CTOA and stimuli type may vary

demonstrated to influence the presence of the IOR effect when using social-emotional cues (Silvert and Funes 2016). As social-emotional cues may capture attention to a greater degree, making it difficult to inhibit and disengage from, the use of social-emotional cue stimuli in a spatial cueing paradigm provides an opportunity to test how this type of information may be prioritized via the IOR effect in visual attention orienting.

Current Knowledge

Altered visual attention has been frequently reported in ASD (Keehn et al. 2013; Sacrey et al. 2014), which may be linked to the noted strengths (i.e., attention to detail; Happé 1994; Mottron et al. 2006) and weaknesses (i.e., diminished social attention; Klin et al. 2002; Riby et al. 2012) in this condition. Visual orienting is one of the mechanisms proposed by the social motivation hypothesis to contribute to the cascading difficulties in reciprocal social communication in ASD over the lifespan (Chevallier et al. 2012; Clements et al. 2018). For example, seminal work by Swettenham et al. (1998) using a naturalistic task found that 20 month old infants with ASD demonstrated more shifts between objects and less spontaneous shifts toward faces compared to both typically developing infants and infants with developmental delay. These reports of atypical visual orienting have been replicated with spatial attention tasks (i.e., gap overlap tasks), such that when compared to controls, infants at high-risk for developing ASD, youth with ASD, and adults with ASD have demonstrated difficulties orienting toward social stimuli compared to controls (Elsabbagh et al. 2009; Kikuchi et al. 2011; Landry and Bryson 2004; Sacrey et al. 2013; Zwaigenbaum et al. 2005). Considering the visual attention strengths in ASD, the social impairments that characterize ASD, and the important role that social-emotional information has on visual attention, measuring IOR with social emotional cues can aid in understanding the prioritization of this type of information in ASD.

To date, six studies have examined IOR in ASD, with two out of six studies using peripheral social or emotional cues (Antezana et al. 2015;

Zalla et al. 2016) and four out of six studies using peripheral simple visual cues (i.e., flashing box; Marotta et al. 2013; Pieron et al. 2014; Rinehart et al. 2008; Song et al. 2018). Of note, one study additionally used central social cues to measure IOR (Marotta et al. 2013).

In regards to IOR with simple nonsocial visual cues, Rinehart et al. (2008) were the first to explicitly test the IOR effect in ASD using a spatial cueing task with peripheral cues (i.e., gray rectangle that flashed white) in 12 children with autism without intellectual disability, 12 children with Asperger's syndrome, and 12 typically developing controls. Although the autism and control groups exhibited similar IOR effects, the Asperger's syndrome group trended towards a stronger IOR effect compared to the control group, demonstrating that the IOR effect may capture a potential alteration in visual attention orienting in ASD. Marotta et al. (2013) examined the IOR effect in 14 children with Asperger's syndrome compared to 14 typically developing controls using peripheral visual cues (i.e., a flashing '+' sign) and central eye gaze cues. No group differences were found in IOR for peripheral visual cues, but the autism group demonstrated absence of IOR for central eye gaze cues, in that they responded faster to gaze valid stimuli than gaze invalid stimuli (Marotta et al. 2013). IOR in the original spatial cueing task (Posner et al. 1984) is only measured with peripheral cues, not central cues, thus it is difficult to compare IOR in this context to results from other studies.

When testing multiple CTOAs with a spatial cueing task, one study found a stronger IOR effect emerged for adults with ASD compared to controls at 200 and 300 ms CTOA, but no group differences at 500 and 700 ms CTOA (Pieron et al. 2014). Thus, suggesting an accelerated time course for IOR in ASD, which may be linked to the superior search abilities noted in ASD (Mottron et al. 2006). A recent study by Song et al. (2018) examined visual IOR in 16 children with ASD and 16 typically developing controls; contrary to previous findings of null group differences (Marotta et al. 2013) or potentially stronger IOR effects in ASD (Pieron et al. 2014; Rinehart et al. 2008), they found that the ASD group demonstrated a diminished IOR effect compared to the control group.

Extending findings of potential alterations in visual IOR for ASD, Antezana et al. (2015) examined social-emotional IOR using peripheral neutral and angry face cues in 41 children with ASD and 25 typically developing children. Although there were no group differences for Angry IOR versus Neutral IOR, findings of a stronger IOR effect for all social-emotional cues emerged for the ASD group compared to the control group. Thus, the ASD group took a greater amount of time in responding to valid social-emotional cues (i.e., area was inhibited for longer) than invalid social-emotional cues, indicating presence of an orienting alteration. Interestingly, IOR was specifically linked to ASD symptomology, such that those with a stronger IOR effect also demonstrated greater ASD symptomology. The IOR effect was not linked to anxiety or ADHD symptomology.

Zalla et al. (2016) examined IOR in 16 adults with autism and 16 controls who were matched on age, gender, IQ, and educational level. In this study, the authors used inverted versus upright face stimuli, with averted gaze faces as peripheral cues, and semitransparent gray squares over the face stimuli as targets. They found that both groups demonstrated an IOR effect to the inverted face condition, though the ASD group displayed diminished IOR for the upright face condition. As gaze cues were used, this alteration may be linked to reflexive exogenous orienting in ASD, which are important for development of social skills, i.e., joint attention (Dawson et al. 2004).

Currently, a number of studies point to potential alterations in the magnitude and timing of visual IOR in ASD (Pieron et al. 2014; Rinehart et al. 2008; Song et al. 2018), with accumulating evidence for social and emotional cues playing a role in alterations of the IOR effect in ASD (Antezana et al. 2015; Zalla et al. 2016). Future work is needed in understanding both the task variability (i.e., CTOA, cue and target stimuli type) and developmental contexts (i.e., age), which may influence IOR in ASD.

Future Directions

As there is some evidence for alterations in the IOR mechanism in ASD, it is important for future

work to disentangle the role of social-emotional cues (and other cue types) on the IOR effect across development. Examining the IOR effect with social-emotional cue types within typical and atypical development may reveal critical periods for orienting, which may have cascading effects on social outcomes in ASD. Currently, no studies exist examining the IOR effect longitudinally or cross-sectionally to uncover developmental trajectories or age differences in IOR. In ASD, IOR has been mostly examined in youth (Antezana et al. 2015; Marotta et al. 2013; Rinehart et al. 2008; Song et al. 2018), though two studies have examined the IOR effect in adults (Pieron et al. 2014; Zalla et al. 2016). The mixed findings in directionality of magnitude may partially be related to differences in age of samples; for example, a stronger IOR effect has been found with social-emotional cues in youth with ASD (Antezana et al. 2015), while diminished IOR with social gaze cues in adults with ASD (Zalla et al. 2016). Future work examining the IOR effect developmentally can aid in understanding of risk and resilience factors for social outcomes in ASD.

It is known that task variability influences whether an IOR effect is evoked with social-emotional cues (Silvert and Funes 2016), thus it will be important to understand the task contexts in which IOR is altered for ASD. More specifically, CTOA, stimuli type, and whether peripheral or central cues are used may greatly influence the magnitude of an IOR effect and whether group differences exist. As one study in adults found that the ASD group had an accelerated onset for IOR compared to controls (Pieron et al. 2014), examining multiple CTOAs may be important in understanding the IOR mechanism in ASD. Currently, only a few studies have examined multiple CTOAs in ASD (Pieron et al. 2014; Rinehart et al. 2008; Song et al. 2018), while others have used a fixed CTOA across the task (Antezana et al. 2015; Marotta et al. 2013; Zalla et al. 2016). Future work can examine whether this accelerated onset is also present with social-emotional cue types and across development.

Task variables such as the spatial location of cues and type of stimuli used in the spatial cueing task may further impact the magnitude of IOR

effect in ASD. For example, visual orienting is differentially affected by peripheral versus central cues (Posner and Cohen 1984). Although most of the work examining the IOR effect in ASD has used peripheral cues, there is a potential for a diminished IOR effect in ASD when using central social gaze cues (Marotta et al. 2013), though more work is needed in this area.

The type of stimuli used as cues and targets may further influence orienting based on saliency and valence. For example, the use of salient versus simple visual *targets* (i.e., face vs. ‘*’) may impact the rate of return to inhibited areas. In regard to cues, simple nonsocial visual stimuli (i.e., flashing square) have been primarily used in ASD, though more work is needed in understanding how the complex social, nonsocial, and emotionally evocative stimuli impact the IOR effect. Currently, there is use of social gaze and social-emotional cues, though these cue types may have distinct influences on IOR for ASD, as the former relates to social information processing, while the latter relates to threat-based processing. Additionally, as there is evidence that nonsocial information is prioritized over social information in ASD (Bos et al. 2019; Kohls et al. 2018; Sasson and Touchstone 2013), it will be important for future work to examine the IOR effect with positively valenced nonsocial images (i.e., restricted interest or object stimuli) which autistic individuals may find harder to disengage from. Moreover, considering the high rates of anxiety disorders (i.e., phobias, social anxiety) in ASD (Lai et al. 2019), the use of negatively valenced emotionally evocative stimuli (i.e., spiders, angry faces) with the peripheral spatial cueing paradigm can unveil how orienting mechanisms may be altered in relation to co-occurring anxiety symptoms in ASD. The peripheral spatial cueing paradigm can be extended to examine how the saliency of different types of information may impact prioritization of information via orienting in ASD.

Eye-tracking can reveal more granular IOR effect results than manual responses. This type of methodology can measure how long information was visually inhibited versus current methods of response time only allowing for processing speed of the target. Additionally, as repetitive

foraging has been suggested as being a failure of IOR effect, eye-tracking can elucidate as to whether alterations in IOR are due to repetitive orienting toward inhibited areas or delayed orienting overall. Finally, examining how the IOR effect interacts with visual search capabilities in ASD can further aid in understanding the perceptual strengths in ASD. If alterations in IOR exist for ASD, it will be useful to examine linkages of these effects with other cognitive and social outcomes across development to reveal potential markers for risk.

Altogether, current work suggests that potential alterations in IOR exist, with magnified effects with social and emotionally evocative cues (Antezana et al. 2015; Marotta et al. 2013; Zalla et al. 2016), future work is needed in disentangling task effects, such as CTOA, stimuli type for cues and targets, and the role of peripheral and central cues on the IOR effect. Eye-tracking methods may reveal fine-grained behavioral patterns that lead to group differences. It is important for future work to examine the IOR effect as it relates to cognitive and social outcomes to understand how alterations in this mechanism are linked to strengths and weaknesses in ASD across development.

See Also

► [Attention](#)

References and Reading

- Antezana, L., Mosner, M. G., Troiani, V., & Yerys, B. E. (2015). Social-emotional inhibition of return in children with autism spectrum disorder versus typical development. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-015-2661-9>.
- Bos, D. J., Silverman, M. R., Ajodan, E. L., Martin, C., Silver, B. M., Brouwer, G. J., . . . Jones, R. M. (2019). Rigidity coincides with reduced cognitive control to affective cues in children with autism. *Journal of Abnormal Psychology*. <https://doi.org/10.1037/abn0000423>.
- Chevallier, C., Kohls, G., Troiani, V., Brodtkin, E. S., & Schultz, R. T. (2012). The social motivation theory of autism. *Trends in Cognitive Sciences*, 16(4), 231–239. <https://doi.org/10.1016/j.tics.2012.02.007>.

- Clements, C. C., Zoltowski, A. R., Yankowitz, L. D., Yerys, B. E., Schultz, R. T., & Herrington, J. D. (2018). Evaluation of the social motivation hypothesis of autism: A systematic review and meta-analysis. *JAMA Psychiatry*. <https://doi.org/10.1001/jamapsychiatry.2018.1100>.
- Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., & Liaw, J. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology*, 40(2), 271–283. <https://doi.org/10.1037/0012-1649.40.2.271>.
- Elsabbagh, M., Volein, A., Holmboe, K., Tucker, L., Csibra, G., Baron-Cohen, S., ... Johnson, M. H. (2009). Visual orienting in the early broader autism phenotype: Disengagement and facilitation. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 50(5), 637–642. <https://doi.org/10.1111/j.1469-7610.2008.02051.x>.
- Fox, E., Russo, R., & Dutton, K. (2002). Attentional bias for threat: Evidence for delayed disengagement from emotional faces. *Cognition & Emotion*, 16(3), 355–379. <https://doi.org/10.1080/02699930143000527>.
- Happé, F. G. (1994). Wechsler IQ profile and theory of mind in autism: A research note. *Journal of Child Psychology and Psychiatry*, 35(8), 1461–1471.
- Itti, L., & Koch, C. (2001). Computational modelling of visual attention. *Nature Reviews Neuroscience*, 2, 194–203.
- Johnson, M. H., Dziurawiec, S., Ellis, H., & Morton, J. (1991). Newborns' preferential tracking of face-like stimuli and its subsequent decline. *Cognition*, 40, 1–19. [https://doi.org/10.1016/0010-0277\(91\)90045-6](https://doi.org/10.1016/0010-0277(91)90045-6).
- Keehn, B., Müller, R.-A., & Townsend, J. (2013). Atypical attentional networks and the emergence of autism. *Neuroscience and Biobehavioral Reviews*, 37(2), 164–183. <https://doi.org/10.1016/j.neubiorev.2012.11.014>.
- Kikuchi, Y., Senju, A., Akechi, H., Tojo, Y., Osanai, H., & Hasegawa, T. (2011). Atypical disengagement from faces and its modulation by the control of eye fixation in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41(5), 629–645. <https://doi.org/10.1007/s10803-010-1082-z>.
- Klein, R. M. (2000). Inhibition of return. *Trends in Cognitive Sciences*, 4(4), 138–147. [https://doi.org/10.1016/S1364-6613\(00\)01452-2](https://doi.org/10.1016/S1364-6613(00)01452-2).
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809–816.
- Kohls, G., Antezana, L., Mosner, M. G., Schultz, R. T., & Yerys, B. E. (2018). Altered reward system reactivity for personalized circumscribed interests in autism. *Molecular Autism*, 9, 9. <https://doi.org/10.1186/s13229-018-0195-7>.
- Lai, M.-C., Kasse, C., Besney, R., Bonato, S., Hull, L., Mandy, W., ... Ameis, S. H. (2019). Prevalence of co-occurring mental health diagnoses in the autism population: A systematic review and meta-analysis. *The Lancet Psychiatry*. [https://doi.org/10.1016/S2215-0366\(19\)30289-5](https://doi.org/10.1016/S2215-0366(19)30289-5).
- Landry, R., & Bryson, S. E. (2004). Impaired disengagement of attention in young children with autism. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 45(6), 1115–1122. <https://doi.org/10.1111/j.1469-7610.2004.00304.x>.
- Marotta, A., Pasini, A., Ruggiero, S., Maccari, L., Rosa, C., Lupiáñez, J., & Casagrande, M. (2013). Inhibition of return in response to eye gaze and peripheral cues in young people with Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 43(4), 917–923. <https://doi.org/10.1007/s10803-012-1636-3>.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36(1), 27–43. <https://doi.org/10.1007/s10803-005-0040-7>.
- Nakano, T., Tanaka, K., Endo, Y., Yamane, Y., Yamamoto, T., Nakano, Y., ... Kitazawa, S. (2010). Atypical gaze patterns in children and adults with autism spectrum disorders dissociated from developmental changes in gaze behaviour. *Proceedings Biological Sciences/The Royal Society*, 277(1696), 2935–2943. <https://doi.org/10.1098/rspb.2010.0587>.
- Pérez-Dueñas, C., Acosta, A., & Lupiáñez, J. (2009). Attentional capture and trait anxiety: Evidence from inhibition of return. *Journal of Anxiety Disorders*, 23(6), 782–790. <https://doi.org/10.1016/j.janxdis.2009.03.002>.
- Pérez-Dueñas, C., Acosta, A., & Lupiáñez, J. (2014). Reduced habituation to angry faces: Increased attentional capture as to override inhibition of return. *Psychological Research*, 78(2), 196–208. <https://doi.org/10.1007/s00426-013-0493-9>.
- Pieron, M., Seassau, M., Leboyer, M., & Zalla, T. (2014). Accelerated time course of saccadic inhibition of return in individuals with autism spectrum disorders. *Experimental Brain Research*. <https://doi.org/10.1007/s00221-014-4152-1>.
- Posner, M. I., & Cohen, Y. (1984). Components of visual orienting. *Attention and Performance X*, 32, 531–556.
- Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, 13, 25–42. <https://doi.org/10.1146/annurev.ne.13.030190.000325>.
- Posner, M. I., Walker, J. A., Friedrich, F. J., & Rafal, R. D. (1984). Effects of parietal injury on covert orienting of attention. *Journal of Neuroscience*, 4(7), 1863–1874.
- Riby, D. M., Brown, P. H., Jones, N., & Hanley, M. (2012). Brief report: Faces cause less distraction in autism. *Journal of Autism and Developmental Disorders*, 42(4), 634–639. <https://doi.org/10.1007/s10803-011-1266-1>.
- Rice, K., Moriuchi, J. M., Jones, W., & Klin, A. (2012). Parsing heterogeneity in autism spectrum disorders: Visual scanning of dynamic social scenes in school-aged children. *Journal of the American Academy of*

- Child & Adolescent Psychiatry*, 51(3), 238–248. <https://doi.org/10.1016/j.jaac.2011.12.017>.
- Rinehart, N. J., Bradshaw, J. L., Moss, S. A., Brereton, A. V., & Tonge, B. J. (2008). Brief report: Inhibition of return in young people with autism and Asperger's disorder. *Autism*, 12(3), 249–260. <https://doi.org/10.1177/1362361307088754>.
- Rutherford, H. J. V., & Raymond, J. E. (2010). Effects of spatial cues on locating emotional targets. *Visual Cognition*, 18(3), 389–412. <https://doi.org/10.1080/13506280902787043>.
- Sacrey, L.-A. R., Bryson, S. E., & Zwaigenbaum, L. (2013). Prospective examination of visual attention during play in infants at high-risk for autism spectrum disorder: A longitudinal study from 6 to 36 months of age. *Behavioural Brain Research*. <https://doi.org/10.1016/j.bbr.2013.08.028>.
- Sacrey, L.-A. R., Armstrong, V. L., Bryson, S. E., & Zwaigenbaum, L. (2014). Impairments to visual disengagement in autism spectrum disorder: A review of experimental studies from infancy to adulthood. *Neuroscience & Biobehavioral Reviews*, 47, 559–577. <https://doi.org/10.1016/j.neubiorev.2014.10.011>.
- Sasson, N. J., & Touchstone, E. W. (2013). Visual attention to competing social and object images by preschool children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 1–9. <https://doi.org/10.1007/s10803-013-1910-z>.
- Silvert, L., & Funes, M. J. (2016). When do fearful faces override inhibition of return? *Acta Psychologica*, 163, 124–134. <https://doi.org/10.1016/j.actpsy.2015.11.002>.
- Song, H., Kwon, M.-K., Park, M., & Chung, H. (2018). Basic auditory processing in the children with autistic features. *Applied Neuropsychology: Child*. <https://doi.org/10.1080/21622965.2018.1532293>.
- Stoyanova, R. S., Pratt, J., & Anderson, A. K. (2007). Inhibition of return to social signals of fear. *Emotion (Washington, D.C.)*, 7(1), 49–56. <https://doi.org/10.1037/1528-3542.7.1.49>.
- Swettenham, J., Baron-Cohen, S., Charman, T., Cox, A., Baird, G., Drew, A., . . . Wheelwright, S. (1998). The frequency and distribution of spontaneous attention shifts between social and nonsocial stimuli in autistic, typically developing, and nonautistic developmentally delayed infants. *Journal of Child Psychology and Psychiatry*, 39(5), 747–753. <https://doi.org/10.1111/1469-7610.00373>.
- Tipper, S. P., Weaver, B., & Watson, F. L. (1996). Inhibition of return to successively cued spatial locations: Commentary on Pratt and Abrams (1995). *Journal of Experimental Psychology: Human Perception and Performance*, 22(5), 1289–1293. <https://doi.org/10.1037/0096-1523.22.5.1289>.
- Verkuil, B., Brosschot, J. F., Putman, P., & Thayer, B. F. (2009). Interacting effects of worry and anxiety on attentional disengagement from threat. *Behaviour Research and Therapy*, 47(2), 146–152. <https://doi.org/10.1016/j.brat.2008.11.003>.
- Wang, Z., & Klein, R. M. (2010). Searching for inhibition of return in visual search: A review. *Vision Research*, 50(2), 220–228. <https://doi.org/10.1016/j.visres.2009.11.013>.
- Yerys, B. E., Ruiz, E., Strang, J., Sokoloff, J., Kenworthy, L., & Vaidya, C. J. (2012). Modulation of attentional blink with emotional faces in typical development and in autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 54(6), 636–643. <https://doi.org/10.1111/jcpp.12013>.
- Zalla, T., Fernandez, L. G., Pieron, M., Seassau, M., & Leboyer, M. (2016). Reduced saccadic inhibition of return to moving eyes in autism spectrum disorders. *Vision Research*, 127, 115–121. <https://doi.org/10.1016/j.visres.2016.07.008>.
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, 23(2–3), 143–152. <https://doi.org/10.1016/j.ijdevneu.2004.05.001>.

Social-Emotional Learning Disability

► Nonverbal Learning Disabilities (NLD), Second Edition

Social-Emotional Learning Skills

► SSIS SEL

Social-Emotional Processing Disorder

► Right-Hemisphere Syndrome

Social-Emotional Skills

► Social Skills Improvement System

Socialization and High-Functioning Autism

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Definition

The processes related to acquisition of social skills high-functioning autism include contributions from theory of mind, executive functioning, and pragmatics.

Historical Background

Literature shows the persistent difficulties in communication and social interaction experienced by children with autism spectrum disorder (ASD) across multiple contexts. They affect a large number of children with ASD, to the extent that 87% and 47.8% of them score in the borderline/abnormal range on scales measuring respectively peer problems and prosocial behavior (Helland and Helland 2017). The impairments are expressed in behaviors such as inappropriate affect, social isolation, and failure to initiate interactions with peers, cooperate, share, make friends, express empathy, or provide emotional support (Russell et al. 2012; Schwenck et al. 2012). These social disabilities may cause daily difficulties, become more pronounced with age, and can lead to negative long-term outcomes. Despite these significant social and communication challenges, scant empirical research has been dedicated to the study of the variables mediating socialization impairments in children with high-functioning autism (HFA).

Different sources of social communication deficits in ASD have been proposed. One of the most known explanations refers to “theory of mind” (ToM) deficits, which implies difficulties

being aware that others have a mind with mental states, information, and motivations that may differ from one’s own (Baron-Cohen 1995). Therefore ToM allows an individual to effectively communicate and interact socially. Individuals with ASD obtain significantly worse results on ToM measures than people with typical development (TD) (Baron-Cohen 1989), and the lack of ToM competences in children with autism impairs their social behaviors (Mazza et al. 2017). In particular, Bishop-Fitzpatrick et al. (2017) determined that performance on first- and second-order false belief tasks was significantly associated with better social functioning, materialized as higher levels of socially adaptive behavior and lower levels of social problems. Nevertheless, it is important to underline the inconsistent results found in research, with some studies failing to detect significant associations between ToM and social functioning (Jones et al. 2017).

Pragmatic competence (PC) has also been linked to social problems in ASD. Pragmatics is defined as the range and frequency of communicative functions, discourse skills, and flexibility to modify speech for different listeners and social situations. It requires picking up cues from the language and behavior of others and an understanding of the rules which govern behaviors in context. Deficits in pragmatic skills are highly prevalent in individuals with ASD, especially in domains related to discourse management (skills to start, maintain, and end a conversation, taking turns, prosody), conversational repairs, and presuppositions (assumptions about the speaker and the specific social context, ToM) (Schoen Simmons et al. 2014). Compared to their peers with TD, they use fewer facial expressions and gestures, have difficulty taking turns in conversation, and can insist on topics in which they are especially interested. The structural language and pragmatics abilities have been related to social functioning in children with ASD, contributing with significant unique variance to the prediction of social behavior (Volden et al. 2009). Moreover, children and adolescents with HFA with more social symptoms of autism have been reported to present lower performance on pragmatic

competence measures (Orinstein et al. 2015). These pragmatic deficits affecting social relations tend to persist in adolescence and adulthood and lead to feelings of loneliness and social exclusion.

Other potential source of the social difficulties encountered by individuals with ASD is the deficit in executive functioning (EF). Executive functions encompass a broad spectrum of inter-related processes that are in charge of “guiding, directing, and controlling cognitive, emotional, and behavioral functions, especially during the active solution of novel problems” (Gioia et al. 2000). The role of the executive profile in autism requires more thorough analysis, but research have suggested a specific pattern of executive dysfunction in components such as planning and flexibility that distinguishes autism from other disorders (Hill 2004; Wallace et al. 2016). A review of the relationships between EF and the social domain of impairment in ASD revealed inconsistent results (Jones et al. 2017). Some studies failed to report significant relations (Cantio et al. 2016), but other investigations pointed out that poorer EF is associated with increased playground isolation and less engagement with peers (Freeman et al. 2017). Moreover, significant associations and longitudinal predictive power have been found between sub-domains of the Behavior Rating Inventory of Executive Function (BRIEF) and adaptive functioning on the Vineland Adaptive Behavior Scales, with executive problems significantly contributing to less adaptive behavior across domains (Pugliese et al. 2015). In addition, the executive functions have demonstrated to predict both emotional and behavioral school engagement, and more specifically, emotion regulation has predicted prosocial peer engagement (Jahromy et al. 2013). Studies have also shown that the pattern of associations between adaptive social behaviors and the EF is different in ASD compared to TD (Leung et al. 2016). The executive processes of behavioral regulation, including inhibition, emotional regulation, and shift, predicted social functioning in children with ASD and TD. However, the metacognitive executive processes of initiation, working memory, monitoring, planning, and organization were

predictors of social adaptation only in children with ASD.

Current Knowledge

Understanding the relationships between the aforementioned constructs and the role they play in the socialization deficits of children with ASD may shed light into the nature of these impairments in order to design suitable interventions. Pragmatic comprehension seems to depend on the inference of the speaker’s beliefs, which is the theory of mind (ToM) ability. In fact, some accounts of ASD conceptualize pragmatic language deficits as reflecting a broader impairment in ToM and empathy. A significant correlation has been found in children with autism – but not in a group with developmental delay – between the performance on ToM tasks and the capacity to respond to a conversational partner with new and relevant contingent information (Capps et al. 1998). In another pioneer study by Tager-Flusberg and Sullivan (1995), theory of mind performance was significantly correlated with a number of different narrative measures only for the group with autism. In addition, the individuals with autism gave significantly fewer appropriate explanations for the emotional states of the story characters, which is linked to the capacity to understand other minds. In this regard, the tendency of people with autism to interpret speech literally might suggest that they do not understand the mind of the speaker. Consequently, difficulties in inferring another person’s beliefs may limit the ability to make predictions about his/her behavior, intentions, and feelings, and this affects the ability to communicate and interact socially. But, even successfully completing the ToM tasks, children with autism seem to have more problems with everyday mindreading than younger children with TD. In fact, children and adolescents with HFA can perform advanced ToM tasks as well as their TD peers, and they seem to be able to master the theoretical principles of advanced mental states. However, they may still fail to apply these theoretical principles during everyday social interactions. As Frith et al. (1994) suggested,

some children with HFA learn to resolve explicit ToM problems in experimental contexts in a non-mentalist way. They can use nonsocial strategies that allow them to respond correctly to false belief questions, but they do not generalize the solutions to real life, which also requires the flexible processing of changing emotional and social signals. Therefore, measures of explicit/conceptual ToM competence and measures of applied ToM competence expressed in real world must be considered in research that tries to describe the relationships between the ToM and adaptive social behavior (Hutchins et al. 2016). Results are enhanced when both explicit social cognition performance and implicit social cognition performance in terms of spontaneous perspective taking are included.

Although ToM deficits may contribute to the interpretation of social communication impairments in ASD, many symptoms are not reflections of an underlying deficit in interpreting people from a mentalistic perspective. Indeed, ToM does not appear to be sufficiently highly correlated with broad measures of social functioning to offer a full explanation of this aspect of ASD (Bottema-Beutel et al. 2018). Other factors related to social development, such as executive functioning and its relationship with ToM, should also be considered. Some studies with children with ASD have revealed that EF-planning skills and EF-cognitive shifting significantly contributed to the explained variance of ToM measures (Kimhi et al. 2014). It has also been demonstrated that ToM deficits is associated with EF impairments in ASD, suggesting attipicity in multiple cognitive domains (Pellicano 2007). A possible explanation of this association is that false belief tasks used to assess ToM abilities impose executive demands and, therefore, EF affects ToM expression. Consequently, it is likely that the functioning in one domain be critical for the emergence of the functioning in another area. Specifically, Perner (1998) highlighted that the meta-representative ability underlying ToM must be reached before children self-regulate their behavior. Alternatively, Russell (1996) proposed a causal relation in the opposite direction, suggesting that the abilities to monitor actions and direct our own

behaviors are critical to be able to reflect about our own and others' mental states. In this regard, in a longitudinal study conducted over a 3-year period, early EF and central coherence skills predicted developmental changes in ToM skills, regardless of age, language, and nonverbal intelligence (Pellicano 2010). Accordingly, impaired EF may have a cascading impact in the development of ToM.

Lastly, when considering the relationships between theory of mind, executive functioning, and pragmatic competence and comparing the profile of children with high-functioning autism (HFA) and children with typical development (TD), the results of the group of children with HFA are clearly lower on explicit and applied ToM skills, the main EF indexes, and PC (Berenguer et al. 2018). In addition, it seems that in the relationship between the socialization domain and ToM, EF, and pragmatic competence as mediators, both the ToM skills applied to everyday life and pragmatic competence had a direct and indirect influence on the socialization of children with HFA. This finding suggests the negative impact of deficits in ToM and pragmatic language competence on the social functioning of children with HFA. Similarly, relationships were found between FE (the metacognitive index of the BRIEF) and the socialization abilities of children with HFA, although they did not reach a significant predictive value. However, these findings should be taken as tentative, because the EF-ToM and pragmatic competence relation in autism is complex. It will be important to establish clearly how poor executive abilities affect the development of ToM and how these cognitive processes affect qualitatively the final stages of social cognition development.

Future Directions

Knowledge of underlying factors of social functioning in children with HFA is a priority for designing effective intervention programs. It has especial interest for parents, teachers, and other social agents involved in optimizing the development of children and adolescents with HFA, to

promote evidence-based interventions that minimize isolation and improve social functioning. Particularly, it is useful the evidence that links symptoms of ASD and general social functioning through mediation of pragmatic and ToM abilities, including also aspects of metacognition.

Longitudinal studies will be essential for mapping the developmental trajectories of EF and ToM abilities and for pointing whether EF and pragmatic skills are in fact crucial domains for the later development of ToM in autism and how it affects social behavior. Furthermore, more sophisticated theoretical models of social functioning need to be developed and tested to enhance our understanding of the contributions and interactions between these and other constructs. More research is needed in order to identify profiles of individuals with ASD, for whom different cognitive and developmental mechanisms are in play in regard to social functioning (Bottema-Beutel et al. 2018).

Moreover, it would be necessary to go beyond techniques based on applied behavior analysis to adopt multicomponent programs that take different aspects into account. This approach appears to encourage social perception and problem-solving capabilities, promoting a more advanced ability to define and recognize emotions in social situations. Specifically, it has proved to be effective in enhancing the capabilities for group peer interaction and for improving awareness of others and social cognition among children with HFA. The implementation of a school-based, teacher-facilitated social skills intervention known as Program for the Education and Enrichment of Relational Skills (PEERS) has also shown significant improvements in responsiveness and social cognition in children with autism (Laugeson et al. 2014). The participants demonstrated overall improvement in social responsiveness, particularly in the areas of social motivation, social awareness, social communication, and decreased autistic mannerisms. The results of PEERS on the importance of active participation of the child and teachers as significant mediators of the program effect are in line with the conclusion of a recent meta-analyses underlining the valuable role of the

parents in pragmatic language interventions (Parsons et al. 2017).

In addition to considering the natural contexts in the interventions of children with autism, it should not be forgotten that the association with positive emotional experiences may be able to promote spontaneous orientation toward social communication, increase socialization opportunities, and facilitate the learning and practice of mentalist and pragmatic skills.

See Also

- [Executive Function \(EF\)](#)
- [PEERS](#)
- [Pragmatic Language](#)
- [Social Skills](#)
- [Theory of Mind](#)

References and Reading

- Baron-Cohen, S. (1989). The autistic child's theory of mind: A case of specific developmental delay. *Journal of Child Psychology and Psychiatry*, 30, 285–297.
- Baron-Cohen, S. (1995). *Mindblindness*. Cambridge, MA: MIT Press.
- Berenguer, C., Miranda, A., Colomer, C., Baixauli, I., & Rosello, B. (2018). Contribution of theory of mind, executive functioning, and pragmatics to socialization behaviors of children with high functioning autism. *Journal of Autism and Developmental Disorders*, 48, 430–441. <https://doi.org/10.1007/s10803-017-3349-0>.
- Bishop-Fitzpatrick, L., Mazefsky, C. A., Eack, S. M., & Minshew, N. J. (2017). Correlates of social functioning in autism spectrum disorder: The role of social cognition. *Research in Autism Spectrum Disorders*, 35, 25–34.
- Bottema-Beutel, K., Kim, S. Y., & Crowley, S. (2018). A systematic review and meta-regression analysis of social functioning correlates in autism and typical development. *Autism Research*, 12, 152–175.
- Cantio, C., Jepsen, J. R., Madsen, G. F., Bilenberg, N., & White, S. J. (2016). Exploring “The autisms” at a cognitive level. *Autism Research*, 9, 1328–1339.
- Capps, L., Kheres, J., & Sigman, M. (1998). Conversational abilities among children with autism and children with developmental delays. *Autism*, 2, 325–344.
- Freeman, L. M., Locke, J., Rotheram-Fuller, E., & Mandell, D. (2017). Brief report: Examining executive and social functioning in elementary-aged children with autism. *Journal of Autism and Developmental Disorders*, 47(6), 1890–1895.

- Frith, U., Happé, F., & Siddons, F. (1994). Autism and theory of mind in everyday life. *Social Development*, 3, 108–124.
- Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2000). Test review behavior rating inventory of executive function. *Child Neuropsychology*, 6, 235–238.
- Helland, W. A., & Helland, T. (2017). Emotional and behavioral needs in children with specific language impairment and in children with autism spectrum disorder: The importance of pragmatic language impairment. *Research in Developmental Disabilities*, 70, 33–39. <https://doi.org/10.1016/j.ridd.2017.08.009>.
- Hill, E. L. (2004). Executive dysfunction in autism. *Trends in Cognitive Sciences*, 8, 26–32.
- Hutchins, T. L., Prelock, P. A., Morris, H., Benner, J., LaVigne, T., & Hoza, B. (2016). Explicit vs applied theory of mind competence: A comparison of typically developing males, males with ASD, and males with ADHD. *Research in Autism Spectrum Disorder*, 21, 94–108.
- Jahromy, L. B., Bryce, C. I., & Swanson, J. (2013). The importance of self-regulation for the school and peer engagement of children with high-functioning autism. *Research in Autism Spectrum Disorders*, 7, 235–246.
- Jones, C. R. G., Simonoff, E., Baird, G., Pickles, A., Marsden, A. J., Tregay, J., . . . Charman, T. (2017). The association between theory of mind, executive function, and the symptoms of autism spectrum disorder. *Autism Research*, 11(1), 95–109.
- Kimhi, Y., Shoam-Kugelmas, D., Agam Ben-Artzi, G., Ben-Moshe, I., & Bauminger-Zviely, N. (2014). Theory of mind and executive function in preschoolers with typical development versus intellectually able preschoolers with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44, 2341–2354.
- Laugeson, E., Ellingsen, R., Sanderson, J., Tucci, L., & Bates, S. (2014). The ABC's of teaching social skills to adolescents with autism spectrum disorder in the classroom: The UCLA PEERS (©) Program. *Journal of Autism and Developmental Disorders*, 44, 2244–2256.
- Leung, R. C., Vogan, V. M., Powell, T. L., Anagnostou, E., & Taylor, M. J. (2016). The role of executive functions in social impairment in autism spectrum disorder. *Child Neuropsychology*, 22, 336–344.
- Mazza, M., Mariano, M., Peretti, S., Masedu, F., Pino, M. C., & Valenti, M. (2017). The role of theory of mind on social information processing in children with autism spectrum disorders: A mediation analysis. *Journal of Autism and Developmental Disorders*, 47, 1369–1379.
- Orinstein, A. J., Suh, J., Porter, K., De Yoe, K. A., Tyson, K. E., Troyb, E., et al. (2015). Social function and communication in optimal outcome children and adolescents with an autism history on structured test measures. *Journal of Autism and Developmental Disorders*, 45, 2443–2463.
- Parsons, L., Cordier, R., Munro, N., Joosten, A., & Speyer, R. (2017). A systematic review of pragmatic language interventions for children with autism spectrum disorder. *PLoS ONE*, 12(4), e0172242.
- Pellicano, E. (2007). Links between theory of mind and executive function in young children with autism: Clues to developmental primacy. *Developmental Psychology*, 43(4), 974.
- Pellicano, E. (2010). Individual differences in executive function and central coherence predict developmental changes in theory of mind in autism. *Developmental Psychology*, 46(2), 530–544.
- Perner, J. (1998). The meta-intentional nature of executive functions and theory of mind. In P. Carruthers & J. Boucher (Eds.), *Language and thought* (pp. 270–283). Cambridge: Cambridge University Press.
- Pugliese, C. E., Anthony, L., Strang, J. F., Dudley, K., Wallace, G. L., & Kenworthy, L. (2015). Increasing adaptive behavior skill deficits from childhood to adolescence in autism spectrum disorder: Role of executive function. *Journal of Autism and Developmental Disorders*, 45, 1579–1587.
- Russell, G., Golding, J., Norwich, B., Emond, A., Ford, T., & Steer, C. (2012). Social and behavioural outcomes in children diagnosed with autism spectrum disorders: A longitudinal cohort study. *Journal of Child Psychology and Psychiatry*, 53, 735–744.
- Schoen Simmons, E., Paul, R., & Volkmar, F. (2014). Assessing pragmatic language in autism spectrum disorder: The Yale in vivo Pragmatic Protocol. *Journal of Speech Language and Hearing Research*, 57, 2162–2173. https://doi.org/10.1044/2014_JSLHR-L-14-0040.
- Schwenck, C., Mergenthaler, J., Keller, K., Zech, J., Salehi, S., Taurines, R., et al. (2012). Empathy in children with autism and conduct disorder: Group-specific profiles and developmental aspects. *Journal of Child Psychology and Psychiatry*, 53, 651–659.
- Tager-Flusberg, H., & Sullivan, K. (1995). Attributing mental states to story characters: A comparison of narratives produced by autistic and mentally retarded individuals. *Applied PsychoLinguistics*, 16, 241–256.
- Volden, J., Coolican, J., Garon, N., White, J., & Bryson, S. (2009). Brief report: Pragmatic language in autism spectrum disorder: Relationships to measures of ability and disability. *Journal of Autism and Developmental Disorders*, 39, 388–393.
- Wallace, G. L., Yerys, B. E., Peng, C., Dlugi, E., Anthony, L. G., & Kenworthy, L. (2016). Chapter three-assessment and treatment of executive function impairments in autism spectrum disorder: An update. *International Review of Research in Developmental Disabilities*, 51, 85–122.

Soft Job Skills

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Somnolence

- [Sleep Disorders and Autism](#)

Son-Rise Program

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Synonyms

[Options method](#)

Definition

The Son-Rise Program was first described by Kaufman in the 1970s as a treatment for children with autism whereby the parent or therapist increases child motivation for social interaction

by “joining in” or imitating child-led behavior. Kaufman based his therapy on the approach he developed to treat his young son with autism – an approach that Kaufman described as successful. The intensive one-to-one therapy is conducted in a distraction-free playroom, and the parent or therapist follows the child’s lead by imitating the child’s repetitive behavior and routines, or “isms.” This showing of acceptance of the child’s behavior is thought to increase the child’s motivation to interact with others so that the therapist can build on the shared activity by encouraging social interaction and communication. The Son-Rise Program is implemented by parents and volunteer therapists in the home environment; parents coordinate and supervise the program following training through the Options Institute. No formal research has been conducted to assess the effectiveness of the Son-Rise Program. In addition to treating individuals with autism, the Son-Rise Program has been used to treat children and adults with other developmental disabilities such as Down syndrome.

References and Reading

- Jordan, R., & Powell, S. (1993). Reflections on the option method as a treatment for autism. *Journal of Autism and Developmental Disorders*, 23, 682–685.
- Kaufman, B. N. (1976). *Son rise*. New York: Warner Books.
- Kaufman, B. N. (1981). *A miracle to believe in*. New York: Fawcett Crest.
- Williams, K. R., & Wishart, J. G. (2003). The Son-Rise Program intervention for autism: An investigation into family experiences. *Journal of Intellectual Disability Research*, 47, 291–299.

Sotos Syndrome and ASD

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Synonyms

[Cerebral gigantism](#)

Short Description or Definition

Sotos syndrome is a congenital overgrowth disorder caused by haploinsufficiency of the NSD1 gene. The cardinal features of the syndrome are overgrowth (defined as height and/or head circumference > 97th percentile) with advanced bone age, macrocephaly, characteristic facial appearance, and intellectual disability. Although these features are consistent within the Sotos syndrome population, the severity of the phenotype is variable. The majority of individuals with Sotos syndrome have mild-moderate intellectual disability or borderline intellectual functioning. In addition, increased likelihood of autism spectrum disorder (ASD), attention deficit hyperactivity disorder (ADHD), anxiety and aggression/tantrums have been reported in individuals with Sotos syndrome.

Categorization

The identification of a genetic abnormality responsible for Sotos syndrome was first established in a Japanese sample (Kurotaki et al. 2002). The authors identified that Sotos syndrome is caused by haploinsufficiency of the NSD1 (nuclear receptor binding SET domain protein 1) gene. The NSD1 gene encodes SET domain-containing histone methyltransferases and is located at chromosome 5q35 (Tatton-Brown and Rahman 2013). Sotos syndrome is caused by intragenic mutations of the NSD1 gene or 5q35 microdeletions encompassing NSD1 and these abnormalities result in loss of function. Subsequent research investigated the prevalence of NSD1 abnormalities in a sample of 266 individuals with a clinical diagnosis of Sotos syndrome. The findings from this study identified that abnormalities of the NSD1 gene were present in more than 90% of individuals with a clinical diagnosis of Sotos syndrome (Tatton-Brown et al. 2005).

As well as having macrocephaly, individuals with Sotos syndrome typically display distinctive neurological abnormalities. Schaefer et al. (1997) investigated structural brain abnormalities in 40 participants with Sotos syndrome, using magnetic resonance imaging (MRI). The findings

indicated that participants displayed a characteristic pattern of abnormalities. Specifically, participants displayed abnormality of the corpus callosum, ventricular abnormalities, midline abnormalities, and delayed or disturbed maturation of the brain.

Epidemiology

The estimated incidence of Sotos syndrome is approximately 1 in 14,000 live births (Tatton-Brown and Rahman 2004). In the majority of cases, the NSD1 abnormalities which cause Sotos syndrome are *de novo*, meaning that they occur spontaneously (Kurotaki et al. 2002). However, the syndrome has an autosomal dominant inheritance pattern, meaning that a child of an individual with Sotos syndrome has a 50% chance of also having the syndrome. A small number of familial cases arising from autosomal dominant transmission have been reported in the literature (Tatton-Brown et al. 2005; Tatton-Brown and Rahman 2007). The syndrome is one of several single-gene disorders associated with overgrowth and intellectual disability and has recently been identified as by far the most prevalent of this category of disorder (Tatton-Brown et al. 2017).

Natural History, Prognostic Factors, Outcomes

Sotos syndrome was initially identified by Sotos et al. (1964) who observed five patients with similar clinical features: excessively rapid growth, acromegalic features, and a nonprogressive cerebral disorder with mental retardation. This combination of features was considered to be attributable to a specific syndrome which was termed “Sotos syndrome.” Subsequent research has confirmed these cardinal features (overgrowth, macrocephaly, characteristic facial appearance, and intellectual disability) in larger samples of individuals with Sotos syndrome (Cole and Hughes 1994; Tatton-Brown et al. 2005). As Sotos syndrome is associated with macrocephaly, initial research often used the terms “cerebral gigantism” and “Sotos syndrome” interchangeably

to refer to the same condition but “cerebral gigantism” is no longer used to describe individuals with Sotos syndrome.

Overgrowth (defined as height and/or head circumference > 97th percentile) is an easily identifiable feature as children with Sotos syndrome are large for their age and tend to be considerably taller than their peers. However, increased height usually becomes less apparent after puberty and in many cases, adults with Sotos syndrome are not significantly taller than those in the general population (Tatton-Brown and Rahman 2007). In contrast, macrocephaly is a stable feature of the syndrome and usually persists throughout adulthood (Tatton-Brown and Rahman 2007). The facial appearance is characterized by a prominent forehead, high anterior hairline, prominent chin, and downslanting palpebral fissures (Dodge et al. 1983). The characteristic facial appearance is most recognizable between the ages of 1 and 6 years, and the facial features often become less apparent in adulthood (Tatton-Brown and Rahman 2007). As the clinical features tend to be more prominent and recognizable in childhood, it is important for individuals displaying this combination of features to be referred for evaluation as early as possible.

A number of medical issues are associated with Sotos syndrome. Approximately 70% of infants with Sotos syndrome develop neonatal jaundice and/or have difficulty with feeding. In addition, cardiac and renal anomalies, seizures, and scoliosis occur in 15–30% of individuals with Sotos syndrome but the type and severity of these issues is variable (Tatton-Brown and Rahman 2007).

To date, most of the published literature reporting data on cognition and behavior in Sotos syndrome has focused on children with the syndrome, and therefore, there is limited understanding of outcomes in adulthood for individuals with Sotos syndrome (Lane et al. 2016). However, research investigating cognition in a sample of 52 adults and children with Sotos syndrome indicated that intellectual disability persists throughout adulthood and that the cognitive profile is

consistent in both adults and children with Sotos syndrome (Lane et al. [under review](#)).

Clinical Expression and Pathophysiology of Sotos Syndrome

Intellectual disability is one of the cardinal features of Sotos syndrome and the majority of individuals have mild-moderate intellectual disability or are in the borderline range (Lane et al. 2016). However, significant variability in intellectual functioning has been reported within the literature, ranging from severe intellectual disability to average intellectual ability. Broadly, the syndrome is associated with relative strength in verbal IQ and relative weakness in performance IQ (Lane et al. 2016). Research investigating the cognitive profile in more detail has identified that individuals with Sotos syndrome display a clear and consistent profile of relative cognitive strengths and weaknesses. Specifically, the Sotos syndrome cognitive profile is characterized by relative strength in verbal ability and visuospatial memory but relative weakness in nonverbal reasoning ability and quantitative reasoning (Lane et al. [under review](#)). Although language and communication delays have been reported in several studies, in general, language abilities have been found to be consistent with overall level of intellectual functioning (Finegan et al. 1994). Fine and gross motor skills are often delayed in childhood and this can be attributable to the large size of the child, as well as the associated hypotonia and poor coordination which are common in children with Sotos syndrome (Tatton-Brown and Rahman 2007).

In terms of the behavioral phenotype, ASD is the most researched behavioral issue within the Sotos syndrome population. Social communication impairment and difficulties with restricted interests and repetitive behaviors have been reported in 68% (Sheth et al. 2015) and 83% (Lane et al. 2017) of individuals with Sotos syndrome. Other behavioral issues such as ADHD, anxiety, and aggression/tantrums have been

reported in the literature, but to date, these behaviors have not been systematically investigated in large samples of individuals with Sotos syndrome so the prevalence of these behaviors is unknown (Lane et al. 2016). As children with Sotos syndrome are often large for their age, they may be mistaken as older and more able than their actual developmental level and this assumption may result in frustration and temper outbursts (Cole and Hughes 1994). This may account for the aggression/tantrums that have been reported in the literature.

Evaluation and Differential Diagnosis

Until the identification of the genetic abnormality associated with Sotos syndrome was identified in 2002 (Kurotaki et al. 2002), the syndrome was diagnosed on the basis of clinical features. However, genetic screening is now widely available. Therefore, individuals who meet the clinical criteria and are suspected of having Sotos syndrome can be screened to check for abnormality of the NSD1 gene.

Other examples of single-gene disorders associated with overgrowth and intellectual disability include Weaver syndrome (Weaver et al. 1974) and Tatton-Brown Rahman syndrome (Tatton-Brown et al. 2014). As these syndromes are also associated with overgrowth and intellectual disability, there is some overlap in the phenotypes of these syndromes. However, each of these disorders is caused by a distinct genetic abnormality and therefore molecular testing is crucial for differentiating between these overgrowth syndromes and for ascertaining the accurate diagnosis for an individual.

Research has identified that the majority of individuals with Sotos syndrome display clinically significant behavioral symptomatology associated with ASD (Lane et al. 2017). In addition, the severity of ASD symptomatology does not differ between males and females with Sotos syndrome. However, severity of ASD symptomatology has been found to be affected by age, with

less severe behaviors observed in young children (2.5–5 years) and adults (20+ years), when compared to children (5–19 years) with Sotos syndrome (Lane et al. 2017). This indicates that ASD symptomatology may be more apparent in children with Sotos syndrome and that severity of ASD symptomatology may decrease in adulthood. Therefore, individuals with a diagnosis of Sotos syndrome who experience social communication impairment and difficulties with repetitive behaviors may require a comprehensive assessment for ASD. A comorbid diagnosis of ASD may be warranted for some individuals with Sotos syndrome.

Treatment

Sotos syndrome is a congenital disorder and is therefore a lifelong condition, which does not require treatment. Within the Sotos syndrome population, there is significant variability in the severity of the phenotype. It is therefore important to consider the needs of the individual when considering appropriate support and issues should be treated symptomatically. To date, syndrome-specific support and interventions have not been established for individuals with Sotos syndrome.

Within the Sotos syndrome population, individuals should be monitored for medical issues such as cardiac and genitourinary anomalies, neonatal jaundice, neonatal hypotonia, seizures and scoliosis, which are common in Sotos syndrome (Tatton-Brown and Rahman 2004) and appropriate treatment should be provided for these issues if they arise. For example, children may develop scoliosis and require a back brace or in some cases, surgery. In addition, seizures are common and some individuals may need to take medication to control the seizures (Tatton-Brown and Rahman 2004).

A cognitive assessment may be useful to ascertain the intellectual ability of a child with Sotos syndrome and to consider the appropriate educational setting for the child. Approximately 50% of children with Sotos syndrome attend special

educational needs schools, while the remaining 50% attend mainstream schools but often require additional support. As children with Sotos syndrome have developmental delay, speech and language therapy may be useful in early childhood to support language development. Similarly, children with Sotos syndrome often have fine and gross motor delays so occupational therapy might be appropriate in early childhood. As the phenotype of Sotos syndrome has not yet been clearly defined, a comprehensive assessment of each individual with Sotos syndrome will inform effective and appropriate strategies and support which can be targeted to the needs of the individual.

See Also

- [Developmental Delay](#)
- [Intellectual Disability](#)

References and Reading

- Cole, T. R. P., & Hughes, H. E. (1994). Sotos syndrome – A study of the diagnostic criteria and natural history. *Journal of Medical Genetics*, 31(1), 20–32. <https://doi.org/10.1136/jmg.31.1.20>.
- Dodge, P. R., Holmes, S. J., & Sotos, J. F. (1983). Cerebral gigantism. *Developmental Medicine and Child Neurology*, 25(2), 248–252.
- Finegan, J. A. K., Cole, T. R. P., Kingwell, E., Smith, M. L., Smith, M., & Sitarenios, G. (1994). Language and behaviour in children with Sotos syndrome. *Journal of the American Academy of Child and Adolescent Psychiatry*, 33(9), 1307–1315. <https://doi.org/10.1097/00004583-199411000-00013>.
- Kurotaki, N., Imaizumi, K., Harada, N., Masuno, M., Kondoh, T., Nagai, T., . . . , Matsumoto, N. (2002). Haploinsufficiency of NSD1 causes Sotos syndrome. *Nature Genetics*, 30(4), 365–366.
- Lane, C., Milne, E., & Freeth, M. (2016). Cognition and behaviour in sotos syndrome: A systematic review. *PloS One*, 11(2), e0149189.
- Lane, C., Milne, E., & Freeth, M. (2017). Characteristics of autism spectrum disorder in Sotos syndrome. *Journal of Autism and Developmental Disorders*, 47(1), 135–143.
- Lane, C., Milne, E., & Freeth, M. (under review). The cognitive profile of Sotos syndrome.
- Schaefer, G. B., Bodensteiner, J. B., Buehler, B. A., Lin, A., & Cole, T. R. P. (1997). The neuroimaging findings in Sotos syndrome. *American Journal of Medical Genetics*, 68(4), 462–465. [https://doi.org/10.1002/\(sici\)1096-8628\(19970211\)68:4<462::aid-ajmg18>3.0.co;2-q](https://doi.org/10.1002/(sici)1096-8628(19970211)68:4<462::aid-ajmg18>3.0.co;2-q).
- Sheth, K., Moss, J., Hyland, S., Stinton, C., Cole, T., & Oliver, C. (2015). The behavioral characteristics of Sotos syndrome. *American Journal of Medical Genetics Part A*, 167(12), 2945–2956.
- Sotos, J. F., Dodge, P. R., Muirhead, D., Crawford, J. D., & Talbot, N. B. (1964). Cerebral gigantism in childhood. A syndrome of excessively rapid growth. *The New England Journal of Medicine*, 271, 109–116.
- Tatton-Brown, K., & Rahman, N. (2004). Features of NSD1-positive Sotos syndrome. *Clinical Dysmorphology*, 13(4), 199–204. <https://doi.org/10.1097/00019605-200410000-00001>.
- Tatton-Brown, K., & Rahman, N. (2007). Sotos syndrome. *European Journal of Human Genetics*, 15(3), 264–271. <https://doi.org/10.1038/sj.ejhg.5201686>.
- Tatton-Brown, K., & Rahman, N. (2013). The NSD1 and EZH2 overgrowth genes, similarities and differences. *American Journal of Medical Genetics Part C-Seminars in Medical Genetics*, 163C(2), 86–91. <https://doi.org/10.1002/ajmg.c.31359>.
- Tatton-Brown, K., Douglas, J., Coleman, K., Baujat, G., Cole, T. R. P., Das, S., . . . , Childhood Overgrowth, C. (2005). Genotype-phenotype associations in Sotos syndrome: An analysis of 266 individuals with NSD1 aberrations. *American Journal of Human Genetics*, 77(2), 193–204. <https://doi.org/10.1086/432082>.
- Tatton-Brown, K., Seal, S., Ruark, E., Harmer, J., Ramsay, E., del Vecchio Duarte, S., . . . , Aksglaede, L. (2014). Mutations in the DNA methyltransferase gene DNMT3A cause an overgrowth syndrome with intellectual disability. *Nature Genetics*, 46(4), 385–388.
- Tatton-Brown, K., Loveday, C., Yost, S., Clarke, M., Ramsay, E., Zachariou, A., . . . , Rittinger, O. (2017). Mutations in epigenetic regulation genes are a major cause of overgrowth with intellectual disability. *The American Journal of Human Genetics*, 100(5), 725–736.
- Weaver, D. D., Graham, C. B., Thomas, I., & Smith, D. W. (1974). A new overgrowth syndrome with accelerated skeletal maturation, unusual facies, and camptodactyly. *The Journal of Pediatrics*, 84(4), 547–552.

Sound Sensitivity

- [Misophonia](#)

South Africa and Autism

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Introduction

In this entry, an attempt is made to provide an overview of autism spectrum disorders (ASD) in South Africa. To this end, attention will be drawn to the historical context, current treatment options, legal mandates for service, research efforts, and relevant controversies. Compiling such a description presents a methodologically complex task – very little systematic data are available about ASD in South Africa, at least in part attributable to the absence of centralized bodies tasked with the monitoring of available services. There are even less data available in peer-reviewed journals, in line with the findings of Tomlinson and Swartz (2003), who demonstrate that in a survey of 764 journal articles related to ASD, only 4 % were outside the USA or Europe. A systematic review of “autism” and “Africa” in the Web of Science over the 10-year period of 2002–2012 revealed 33 papers about or from Africa, in rather stark contrast to >18,000 peer-reviewed papers on autism over the same period in the rest of the world (personal communication, Prof P J de Vries). This entry therefore represents a careful balance between relying on robust data sources and providing sufficient detail such that a helpful picture can be drawn. Our data sources therefore include websites of specific organizations,

estimates of service delivery based on available databases, and perspectives of key role players – including managers of NGOs, clinicians in public and private practice, and academic psychiatrists. Every effort has made to be as transparent as possible in describing data sources, and when no citation is presented, the perspective can be assumed to be the product of the accumulated experience of the authors.

Historical Background

As it is for much of sub-Saharan Africa, the history of autism in South Africa is brief and poorly documented. A survey of six African countries conducted in 1978 by Viktor Lotter (1978), a pediatrician working in the UK at the time, was probably the first systematic study to show that there were children in Africa (Ghana, Nigeria, Kenya, Zimbabwe, Zambia, and South Africa) who displayed patterns of behavior consistent with the construct of autism as understood at the time. Lotter was particularly interested in looking for autism in “indigenous African children,” defined by him as “black African children raised by black African parents.” He therefore took great care to exclude so-called mixed race or non-African black children. He also compared his African sample to a group of children from Middlesex in London identified with autism. Out of 1300 African children with intellectual disability, he identified 30 with autism-related behaviors; nine children met the “western criteria” for autism of the time. Interestingly, he found verbal and nonverbal children with autism and rates of epilepsy, gender ratios, and social backgrounds to be relatively similar to those of his Middlesex sample. On direct comparison of the African and Middlesex samples, he described differences in specific autistic characteristics. For instance, he found no flapping, ritualistic play, or self-injury in the African sample, but did report an overrepresentation of “other marked movements” and “carrying, banging, or twisting of objects.” He argued that there may be less autism in Africa

and that some of the phenomena typically associated with autism may be uncommon in African settings (Lotter 1978).

Subsequent studies have confirmed that autism has similar gender ratios in Africa as the rest of the world (Ametepee and Chitiyo 2009), and at least one subsequent study has highlighted the comparative rarity of certain movements (like rocking) that occur more commonly in western populations (Mankoski et al. 2006). Additionally, it has been noted that as in Kanner's original description of children with autism, published cases in Africa tend to be of children from higher socioeconomic backgrounds (Ametepee and Chitiyo 2009; Kanner 1943). The most likely explanation is that wealthier individuals live in closer proximity to medical centers and are therefore more likely to be able to access services. This finding of SES as a correlate for access to services maps well onto similar observations in the USA (Durkin et al. 2010). It has now become accepted that autism occurs across Africa. To date there have been no systematic investigations into the prevalence of autism in Africa. There may be many potential reasons for this lack of epidemiological data on autism in Africa, including the fact that communicable diseases have dominated health priorities in Africa over the last century, combined with the lack of systematic research programs in autism over the last number of decades, and the lack of appropriate screening and diagnostic tools for autism in African languages (Grinker et al. 2012).

In response to a growing recognition of the existence of autism, combined with very limited available services, the nongovernmental organization (NGO) Autism South Africa was founded in 1989 (Autism South Africa 2014). The history of autism in South Africa has been influenced in a significant way by the growth and the development of this organization. The group initially consisted of concerned parents and professionals that were primarily engaged in lobbying government, but, given increasing requests for the provision of support and assistance to families, the group expanded in scope and established permanent, full-time offices in 1997. Today, Autism South Africa serves as a resource for parents and families, providing regular parent training and

facilitating referral of both families and individuals with autism to relevant educational and supportive resources. Autism South Africa has branches and staff in a number of South African Provinces. There are also other ASD NGOs, including Autism Western Cape, based in Cape Town, and Action in Autism, based in KwaZulu-Natal. Despite the growth of organizations like Autism South Africa and other NGOs, there continues to be a significant shortage in services for both family members and individuals with autism, with an estimated 135,000 individuals with ASD in South Africa not receiving appropriate services (Bateman 2013).

Legal Issues: Mandates for Service

There are currently no legal mandates for ASD-specific services in South Africa. Although the South African constitution is internationally renowned for enshrining equal rights for all, significant resource shortages prevent implementation of this ideal. For example, although all children in South Africa have a right to education, with only five government-funded schools tailored to ASD individuals, many children are left underserved. As a member of the WHO, South Africa adopted the resolution on autism spectrum disorders (World Health Organization 2013) (http://www.who.int/mental_health/action_plan_2013/eb_resolution_childhood/en/), which urges member states to expand capacity for services, develop and implement policies, promote research and awareness, and provide focus on community-based (rather than long-term institutional) treatment along with several other goals. The resolution is focused on an implementation period from 2013 to 2020. To date there are no published reports of progress currently available for South Africa.

Overview of Current Treatments and Centers

The South African healthcare system is for the most part sharply divided between a well-resourced private sector, serving around 20 % of

the population, and a relatively resource-limited state sector, which serves the remaining 80 %. However, even in the private sector, services for ASD are of variable quality, being for the most part unmonitored and not always employing evidence-based practices. In addition to being non-standardized, current services are remarkable for being of greatly insufficient capacity. Representatives from the University of Cape Town's Autism Research Group note that ASD-specific schools in South Africa have several year waiting lists, often leading to children being accepted to these facilities and receiving appropriate intervention 3 or 4 years after the time of diagnosis (UCT Department of Psychology Autism Research 2014). These children are nevertheless more fortunate than a majority who are unable to receive specialized education altogether (Bateman 2013). Available schools, diagnostic centers, and residential treatment facilities are discussed below.

Schools

There are only nine schools specifically set up for individuals with ASD in South Africa (Autism South Africa 2014). Four of these are private schools that offer very comprehensive programs, including pre- and after-school care, holiday programs, and access to a wide range of recreational facilities. The remaining five are state funded, often with support from NGOs. The first government school for children with ASD was the Vera School, in Cape Town, founded in 1970. It is regarded as a specialist resource center in the management of autism spectrum disorder. The school has 145 pupils and can accommodate 35 on site in hostels. It has 74 members of staff and is publicly funded through the Western Cape Education Department (Charity SA 2014).

The Alpha School was established in 1974 by what was then known as the Society for the Autistic Children. It was originally set up in a living room of a private home in Athlone, Cape Town, and after various relocations, it moved to its current premises in Woodstock, Cape Town, in 1995. It has traditionally served the less privileged sectors of the Western Cape population. At present it has approximately 76 learners up to the age of 17, in ten classrooms with a variety of volunteers and students assisting in the activities of the

teachers and supplementary staff – which includes speech therapy, occupational therapy, and psychology. The children are occupied with classwork, swimming, and various other activities including a peace garden, which is an initiative that involves the community and the children taking ownership in cultivating a patch of a vegetable garden (Scanza 2014).

As an example in the private sector, “The DreamTree School” in the relatively wealthy Western Cape Province of South Africa is a well-resourced center with occupational and speech therapists, experienced special education teachers, and psychologists on site (The Dream Tree School 2014). This facility cites evidenced-based practices such as applied behavioral analysis (ABA) and social skills training as a basis for their approach. Although there are fewer schools in other provinces, one exemplar in the non-private sector is the UNICA School in Menlo Park, Gauteng. This facility is operated as a public benefit organization (PBO), in partnership with the government, donors, and nominal fees payable by the parents of attendees (around \$1000 per year in 2014 for tuition). The UNICA School attracts students from across South Africa, Swaziland, and neighboring countries and cites the TEACCH curriculum as being central to their approach (The Unica School for Autism 2014).

Six of the ASD-specific schools are in the Western Cape Province of South Africa, with the other three in the Gauteng and Eastern Cape provinces. Given the small number of schools in the context of a population of 52 million people, clearly many children with ASD are unable to attend these facilities. Other sources of education include special programming within regular schools and attendance of schools that target children with special educational needs in a much broader sense. Unfortunately, there are no systematic data on the quality and suitability of the education obtained by ASD individuals at these organizations, and it is assumed to be highly variable across different regions.

While the existing educational programs listed above often refer to ABA, TEACCH, Makaton, and PECS, it is unclear whether more contemporary, relationship-based intervention approaches

such as the Early Start Denver Model, JASPER, Pivotal Response Training, etc., are utilized in schools. It is also worth bearing in mind that the statutory school-going age is 7 years and that no governmental preschools exist. No child with autism under the age of 6 or 7 will therefore have access to public sector early intervention or educational programs.

Assessment and Diagnosis

It is assumed that university hospitals in South Africa provide diagnostic services for individuals with symptoms suggestive of ASD. However, with only eight medical schools in South Africa, the capacity is insufficient to ensure that a majority of individuals receive an early diagnosis. There are only nine qualified neurodevelopmental pediatricians in South Africa, with a national training capacity of two candidates per year, and only around 46 specialist child and adolescent psychiatrists in South Africa (de Vries et al. 2013; Skosana 2014). In the Western Cape, which is a home to three relatively large centers (Tygerberg Hospital, Lentegour Hospital, Red Cross Children's Hospital), around ten individuals are evaluated for ASD per week. In addition, the organization Autism South Africa runs an assessment clinic once a month in Johannesburg, targeting individuals who do not have access to academic medical centers.

The availability of accurate diagnostic services within the private sector is unclear. There are a very small number of appropriately experienced pediatricians and child and adolescent psychiatrists in South Africa, and anecdotal evidence suggests that general practitioners (who form a significant majority of overall providers) are often uncomfortable with diagnosing neurodevelopmental disabilities (Bateman 2013).

Residential Treatment

Available residential treatment is provided by NGOs and includes the Horizon Farm in the province of KwaZulu-Natal (Horizon Farm Trust 2014) and Lethabo Le Khutso Adult Care center in Pretoria, Gauteng province (Association For

Autism 2014). The Horizon Farm is operated by the Horizon Farm trust, has been in operation since 1994, and is situated on a 30-acre farm, facilitating an outdoor lifestyle for 30 individuals with intellectual disabilities, including but not limited to ASD. This facility reports an ever-increasing waiting list. The Lethabo Le Khutso Adult Care center is a facility that provides residential care for adults with ASD and is an initiative of the Association For Autism, an NGO. In most parts of South Africa, there appear to be no residential treatment facilities available specifically set up for adults with autism with/without intellectual disability.

Other Services

The bulk of additional supports and services are provided by NGOs. These include day programs, like the CARE Day School in Johannesburg, Gauteng province (Centre For Autism Research and Education 2014), and the Star Academy with locations in Gauteng and KwaZulu-Natal (The Star Academy 2014). These organizations provide a range of services with a prominent focus on applied behavioral analysis. The organization Autism South Africa, highlighted previously, acts broadly in providing screening clinics and helping parents get connected with available treatment while serving as advocates for the ASD community at the governmental level (Autism South Africa 2014). The Els for Autism Foundation was founded in 2009 by the well-known South African golfer Ernie Els and his wife Liezl, who have a son affected with ASD (Ernie Els Foundation 2014). The goal of this NGO is to support an e-learning platform that provides access to ASD education and therapy, as well as online training at no cost. These and other similar organizations are a significant part of the ASD treatment landscape in South Africa.

Overview of Research Directions

Prior to 2012, there was no systematic research programs on autism anywhere in Africa,

including in South Africa. In 2012 Prof Petrus de Vries founded the Center for Autism Research in Africa at the University of Cape Town. The goals of this initiative are to develop an interdisciplinary center for ASD and related disabilities across themes of awareness, training, clinical work, and research (Malcolm-Smith et al. 2013). Current programs within the center include:

1. The translation and validation of screening tools for ASD such as the social communication questionnaire (SCQ) and modified checklist for autism in toddlers (M-CHAT) into Afrikaans, isiXhosa, and Kiswahili. In addition to validation of psychometric properties, efforts are being made to ensure that these measures are culturally appropriate using mixed-method research approaches.
2. The translation and validation of diagnostic tools like the Autism Diagnostic Observation Schedule (ADOS) and Autism Diagnostic Interview-Revised (ADI-R) into Afrikaans, isiXhosa, and Kiswahili.
3. Development of post-diagnostic programs, including programs for psychoeducation and parent training. Tied to the development of these programs is the evaluation of existing programs developed in the UK, with assessment of their suitability for the South African context.
4. Identification of evidence-based interventions and, in particular, parent-/caregiver-led interventions, which are particularly salient given resource limitations and the limited number of expert therapists available.
5. The training of staff in primary, secondary, and tertiary level care centers in early detection, diagnosis, and treatment of ASD.
6. Evaluation and development of service models. Given the limited existing services, even in better-resourced parts of South Africa, evaluation of existing educational services (including needs and gap analysis) is crucial for the development of sustainable, integrated services for individuals with ASD and their families.
7. Investigating the potential for use of technology in low-resource environments to facilitate the abovementioned aims and improve early detection, diagnosis, and treatment.

This group therefore has an ambitious research agenda, which is likely to be highly impactful in improving understanding of ASD in South Africa. However broader involvement from multidisciplinary clinical and academic colleagues across all academic medical centers remains an important need.

Cultural Factors and Controversies

There is a significant lack of awareness surrounding ASD, particularly in rural areas of South Africa (Autism South Africa 2014). This, in combination with the dearth of available evidence-based treatments for autism, has maintained widespread misconceptions about the nature of the disorder, as well as propagation of pseudoscientific diagnostic and treatment practices. In certain communities, ASD continues to be viewed as evidence of demonic possession, or a sign of punishment from the individuals' ancestors (Autism South Africa 2014). As reported by Grinker et al. (2012), parents of children with ASD in rural communities reported taking their children to traditional healers for consultations about treatment. However, it is of interest that these parents also reported the utility of having the "western" diagnosis of ASD provided to their children, as it reduced their concerns about a potential spiritual cause for the child's difficulties. This study also noted an interesting fact about child-care centers or crèches operating in these communities. Staff working at crèches reported the expectation that after the age of 2, direct eye contact with elders would be discouraged and potentially seen as disrespectful – an interesting finding suggesting the possibility of differential cultural expectations regarding certain social communication behaviors that are used as red flags for autism.

In wealthier, urban areas, a different set of challenges exist, with a number of expensive, private practices offering nonevidenced-based explanations and treatments for individuals with ASD. These practices are run by individuals with a broad range of qualifications and most often offer treatments centered on viewing autism as a consequence of gastrointestinal dysfunction. These interventions may include restrictive diets, herbal medications, homeopathic remedies, and supplementation. Many practitioners operating in this paradigm report their alignment with the now-defunct Defeat Autism Now! Program. The surprising prominence of such practitioners is likely a consequence of the limited availability of adequately trained child and adolescent psychiatrists and neurodevelopmental pediatricians in South Africa.

References and Reading

- Ametepee, L., & Chitiyo, M. (2009). What we know about autism in Africa: A brief research synthesis. *The Journal of the International Association of Special Education*, 1(1), 11–14.
- Association For Autism. (2014). Retrieved October 31, 2014, from <http://www.afa.org.za/>
- Autism South Africa. (2014). Retrieved October 31, 2014, from <http://www.aut2know.co.za>
- Bateman, C. (2013). Autism – Mitigating a global epidemic. *South African Medical Journal*, 103(5), 276–277. <https://doi.org/10.7196/samj.6915>.
- Centre For Autism Research and Education. (2014). Retrieved October 31, 2014, from <http://www.thecarecentre.co.za>
- Charity SA. (2014). Vera school. Retrieved October 31, 2014, from <http://www.charitysa.co.za/vera-school-for-autistic-learners.html>
- de Vries, P. J., Venter, A., Jacklin, L., & Oliver, C. (2013). Behavioural phenotypes and neurodevelopmental disorders in Africa. *Journal of Intellectual Disability Research*, 57(9), 793–795.
- Durkin, M. S., Maenner, M. J., Meaney, F. J., Levy, S. E., DiGuseppi, C., Nicholas, J. S., ... Schieve, L. a. (2010). Socioeconomic inequality in the prevalence of autism spectrum disorder: Evidence from a U.S. cross-sectional study. *PloS One*, 5(7), e11551.
- Ernie Els Foundation. (2014). Els for Autism. Retrieved October 31, 2014, from <http://www.elsforautism.com>
- Grinker, R. R., Chambers, N., Njongwe, N., Lagman, A. E., Guthrie, W., Stronach, S., ... Wetherby, A. M. (2012). “Communities” in community engagement: Lessons learned from autism research in South Korea and South Africa. *Autism Research : Official Journal of the International Society for Autism Research*, 5(3), 201–210.
- Horizon Farm Trust. (2014). Retrieved October 31, 2014, from <http://www.horizonfarmtrust.org.za>
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Lotter, V. (1978). Childhood autism in africa. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 19(3), 231–244.
- Malcolm-Smith, S., Hoogenhout, M., Ing, N., Thomas, K. G. F., & de Vries, P. J. (2013). Autism spectrum disorders – Global challenges and local opportunities. *Journal of Child & Adolescent Mental Health*, 25(1), 1–5.
- Mankoski, R. E., Collins, M., Ndosi, N. K., Mgalla, E. H., Sarwatt, V. V., & Folstein, S. E. (2006). Etiologies of autism in a case-series from Tanzania. *Journal of Autism and Developmental Disorders*, 36(8), 1039–1051.
- Scanza. (2014). Alpha school for autism. Retrieved October 31, 2014, from www.scanza.dk/en/projects/alpha-school-for-autism/
- Skosana, I. (2014, April 4). Autism and its uncommon angels. *Mail and Gaurdian*. Retrieved October 31, 2014, from <http://mg.co.za/article/2014-04-03-autism-and-its-uncommon-angels>
- The Dream Tree School. (2014). The Dream Tree School – The school for children with Autism. Retrieved October 31, 2014, from <http://www.thedreamtreeschool.co.za>
- The Star Academy. (2014). Retrieved October 31, 2014, from www.thestaracademy.co.za
- The Unica School for Autism. (2014). Retrieved October 31, 2014, from www.unicaschool.co.za
- Tomlinson, M., & Swartz, L. (2003). Imbalances in the knowledge about infancy: The divide between rich and poor countries. *Infant Mental Health Journal*, 24(6), 547–556. <https://doi.org/10.1002/imhj.10078>.
- UCT Department of Psychology Autism Research. (2014). UCT autism research. Retrieved October 31, 2014, from <http://www.uctautism.com>
- World Health Organization. (2013). WHA resolution on “Comprehensive and Coordinated Efforts for the Management of Autism Spectrum Disorders.” Retrieved October 31, 2014, from http://www.who.int/mental_health/action_plan_2013/eb_resolution_childhood/en/

SPA

► Sensory Processing Assessment

Spain and Autism

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Historical Background

Much of the data that allows us to reconstruct the history and the current situation of autism in Spain has been taken from the compilation prepared by Adam Feinstein, principally through Mercedes Belinchón, Doctor in Psychology and professor of the Autonomous University of Madrid, who has played an important role in that institution and promoted different initiatives in relation with education and research of special importance (Feinstein 2016).

The support services specifically for people with ASD (autism spectrum disorder) that began to emerge in Spain were promoted by professionals, principally with links to the health services, many holding a clear psychodynamic orientation that characterized their early training. The most important references in those first phases are the Centro TAURE (Madrid) that opened in 1973 and the Centro Carrilet (Barcelona), founded in 1974. In these first years of life, the professional life of Doctor Ángel Díez Cuervo (Neuro-pediatrics) may be highlighted. He has been a leading light in Spain, supporting the launch and the growth of institutions. His important contributions have added to our knowledge of scientific evidence-based practices and their development.

This first phase in the development of specific support services extended up until the mid-1970s, a time at which a family-centered model began to gain in popularity, through associations that characterize the reality in which we now live. They have managed to generate the majority of resources specific to our country. In 1976, two of the associations were launched that supported the successive formation of other organizations of parents of people living with autism; APNA

(Madrid) and La Garriga (Barcelona), managed by Isabel Bayonas and Joan Roca, respectively, which have for decades contributed to the development of policies of support for the collective and the launch of associations in other geographical areas of the country.

Before the end of the 1970s, other entities were set up that continue to be nationwide references to this day, such as the Asociación Guipuzcoana de Autismo (Gautena). It enjoys the technical support of the child psychiatrist Joaquín Fuentes Biggi, a reference for autism both within and outside Spain. In 1979, the Association New Horizons (Madrid) was born, one of the first entities that has developed services for adults with ASD in Spain.

The establishment of these organizations went on to generate cooperative alliances between families and professionals, which were shaping a model of services where education came to be seen as the best development tool for people with ASD. The psychodynamic perspective has gradually lost ground at centers and support services, except in Cataluña, a regional community where it is still very popular. The mothers and fathers who pioneered it, together with professionals such as Ángel Díez Cuervo and Ángel Rivière Gómez, contributed the philosophical and conceptual groundwork that still serves as a reference today for many associations and has assisted them in their development throughout Spain. Their contributions reinforced the biological factor as the origin of autism, burying the myths that blamed the families, and the value of integral education as a better tool with which to promote the development for people with ASD.

It had special importance at the I International Symposium on Autism, organized in Madrid in 1978, by APNA and SEREM (*Servicio de Recuperación y Rehabilitación de Minusválidos Físicos y Psíquicos*) [Recovery and Rehabilitation Service for the Physically and Mentally Disabled] under the catchphrase “Help me to know who I am.” This meeting, directed at professionals, families, public authorities, and society in general, had many repercussions throughout Spanish territory. As from that point, people began to

hear of a different approach to autism, unlike the erroneous theories and interpretations that had up until the present been prevalent. This initiative was crucial, as it placed the different models that coexisted at that time in our country on the table: psychodynamic, behaviorist, and the perspective that defined the biological origin of autism and interventions from the cognitive behavioral point of view through educational processes of intervention. In that symposium, the families reaffirmed the need and the responsibility to play an active role in setting up services and demands for support. Another of the important consequences of the Symposium was the launch on 4 May 1978 of a Committee composed of families of nine different nationalities (Australia, France, Germany, Holland, Ireland, Norway, Portugal, Spain, and Sweden), for the organization of the International Coordinating Office for Parents of Autistic Children and Adults, with the objective of lobbying for people with autism and recognition that each autistic person needs education, treatment, and specialized and individualized care; has the right to develop his or her potential to a maximum as human beings; and to have a decent place in society (Martínez et al. 2010).

Moreover, in the field of the associations, one initiative that has profoundly affected the development of autism in Spain has been the birth of the AETAPI, now the *Asociación Española de Profesionales del Autismo* [Spanish Association of Autism Professionals], driven by the professionals themselves as a space of exchange, development, and education. This organization has contributed to generating a consensual model of intervention, based on good practice with scientific evidence, and at present supports professionals, people living with ASD, families, and organizations in the defense of rights and a strategy to confront unscientific practices that are still sold today as “miracles.” Every 2 years, AETAPI organizes a congress on autism and promotes the development of good practice and research, through the Angel Rivière Prizes. This organization has over the years counted on the support of professionals such as Javier Tamarit, who began his links with autism through his work at CEPRI, at the end of the 1970s,

and contributed in a very significant way to the development of knowledge on ASD in Spain.

Over time, associations have been extending across national territory and have been grouping themselves together in federations, in different autonomous communities and, at a national level, in confederations. The FESPAU Confederation, born in 1994, at present represents 25 organizations, and the Spanish Autism Confederation, born in the same year, groups together 77 entities comprising families, people with ASD, and professional linked to the collective. In 2004, the Asperger Spain Federation was launched. At present, the three organizations jointly collaborate on some initiatives, especially those oriented towards the promotion of state policies for assistance to people with ASD.

In 2001, the *Grupo de Estudios de los Trastornos del Espectro del Autismo* (GETEA) [Study Group on Autism Spectrum disorders] was formed, as a part of CISAT (*Centro de Investigación sobre el Síndrome del Aceite Tóxico*) [Research Center on the Toxic Oil Syndrome], the forerunner to the present-day *Instituto de Salud Carlos III* [Institute of Health Carlos III] with the objective of conducting a project to gain greater knowledge of the reality of autism in Spain and to recommend ways of improving assistance, training, and research into ASD. This group knew how to harness the necessary interest, knowledge, and experience to confront the present-day challenges of research and practice related with autism, consisting in the study of the causes of the increased prevalence and etiology of these disorders, as well as the aspects related with the detection, diagnosis, and intervention directed at people who present it. As a result of the work of this group, four guides of good practice were published for the early detection, diagnosis, treatment, and investigation of autism spectrum disorders in the *Revista Española de Neurología* [Spanish Journal of Neurology].

At present, the IIER continues to be involved in the development of ASD-related projects in both the state sector and the European context and collaborates at an international level in epidemiological research into these types of disorders.

Legal Issues, Mandates for Service

This historic moment coincided with the transition to democracy in Spain, which was accompanied by greater involvement and initiative in civil society to transform reality, generating a significant movement of associations in the 1970s and 1980s. Moreover, legislation and initiatives were advanced that favored the recognition of the rights of people with disability and their inclusion in education, society, and the workplace, as well as the consolidation of entities responsible for the development of actions directed at these collectives, among the most important of which are the following:

Ley General de Educación [General Law on Education] of 1970 that recognizes the right to education of people with autism, adopting the term Special education and recognizing it as a specific modality.

Decreto de 23 de mayo de 1975 [Decree of 23 May, 1975], establishing the Instituto Nacional de Educación Especial (INEE) [National Institute of Special Education] as an autonomous organism, dependent on the Ministry of Education and Science, which will be a key piece in the progress and the development of attention to students with special educational needs through specialized centers.

Approval of the Royal Board of Special Education in *Real Decreto de abril de 1976* [Royal Decree of April 1976], chaired by H.M. Queen Sophia, the forerunner of the present-day Real Patronato sobre Discapacidad [Royal Board on Disability]. In addition to the Ministry of Education and Science, the Ministries of the Treasury, Territorial Government (now defunct), Justice, Employment, and Social Security all formed part of it, as well as other personalities of recognized prestige, appointed by H.M. Queen Sophia.

The Spanish Constitution (1978) dedicated its article 49 to recognizing the rights of the collective of people with disability, mentioning that the public authorities will uphold a policy

of prevention, treatment, rehabilitation, and integration, and will ensure the delivery of the specialized attention that they require and will give them special help for full enjoyment of the rights that this provision grants to all of its citizens.

The *Ley de Integración Social de los Minusválidos, de 7 de abril de 1982* (LISMI) [Law of Social Integration of the Disabled, of 7 April 1982] is a norm of special importance in Spain, because it explicitly regulates the right of disabled people to inclusion in social, educational, and employment activities. In the area of education, under the third article of Title VI, their inclusion in the ordinary system of general education is contemplated, receiving in each case the support and the resources that the law envisages.

The *Real Decreto del 6 de marzo de 1985* [Royal Decree of 6 March 1985] regulates Special Education in a specific way as an option that forms part of the national educational system, contemplating the inclusion in ordinary centers with the support that each person requires, and where serious difficulties exist, schooling will take place in specific units or centers.

The *Real Decreto 2377/1985, por el que se aprueba el Reglamento de Normas Básicas sobre conciertos educativos* [Royal Decree 2377/1985, in approval of the Rule of Basic Norms on educational agreements], supported the creation of many of the specific educational centers, the majority promoted and managed by families, and subsidized with public funds.

The consolidation of educational integration came in 1990, with the *Ley de Ordenación General del Sistema Educativo* [Law on the General Organization of the Educational System], which incorporates the terms of pupils with special educational needs and determines that they will have access to the necessary supporting resources and the curricular adaptations that they may require.

Ley 39/2006, de 14 de diciembre, de Promoción de la Autonomía Personal y Atención a las personas en situación de dependencia [Law 39/2006, of 14 December, on the Promotion of Personal Independence and Attention to

people in a situation of dependency], which regulated the universal right to different modalities of services and their delivery to people living with disabilities. On that basis, the professional figure of the Personal Assistant was defined as personalized support in the fields and times at which the disabled person may most require so.

On 13 December 2006, the Convention on the Rights of Persons with Disability was adopted in New York and its Optional Protocols, international legal instruments that were ratified by Spain and entered into force on 3 May 2008, having since then formed part of our legal order.

Real Decreto Legislativo 1/2013, de 29 de noviembre, por el que se aprueba el Texto Refundido de la Ley General de Derechos de las Personas con Discapacidad y de su Inclusión Social [Royal Legislative Decree 1/2013, of 29 November, in approval of the Consolidated Text of the General Law of the Rights of People with Disabilities]. This regulation was based on the proposal in the Convention of the Rights of People with Disability (ONU 2006).

In 2013, the rights of people with disability advanced in Spain, with the recognition, among others, of the right to full participation and inclusion, initiated at the earliest possible stage through the enactment of the *Ley General de Derechos de las Personas con Discapacidad y de su Inclusión Social* [General Law of the Rights of Persons with Disability and of their Social Inclusion] published in the BOE of 3 December 2013. Its drafting was based on the proposal advanced at the International Convention of the Rights of Persons with Disability, approved at the United Nations General Assembly in 2006 (UNO 2006). Article 26 of that Convention specifically refers to the right to education in the context of “full inclusion and participation in all aspects of life.” Both initiatives are based on the principle of nondiscrimination towards persons with disability, and they understand education as part of comprehensive habilitation and rehabilitation services. It is stressed that education should start at the

earliest possible stage and should be based on a “multidisciplinary assessment” of the “individual needs and strengths” of the person with disability, as well as opportunities in their own community. Appropriate habilitation and rehabilitation services and support will be oriented towards decision-making and measures to attain full independence. It also considers that the public administrations should promote the maintenance of effective and appropriate attention, through the coordination of resources and habilitation and rehabilitation services in the areas of health, employment, education, and social services, with the purpose of guaranteeing that people with disability are offered appropriate and varied services and programs in their own communities, in both rural and urban zones.

The most recent initiative in matters of autism promoted by the State has been the preparation of the Spanish Strategy on Autism Spectrum Disorders, approved by the Council of Ministers and sponsored by the Ministry of Health (2015). It is at present in a phase of development. This initiative seeks, among other questions, to encourage measures that promote the detection and diagnosis of ADS, appropriate educational attention, including the stages prior to the obligatory schooling of pupils with ADS, and evidence-based practices, especially in the case of professionals linked to early attention and infant education. The strategy also promotes the strengthening of a varied, appropriate, and specialized network of educational centers in all the territories that have the necessary means to provide individualized and quality education to pupils with ADS (professional profiles, ratios, resources, etc.). At the same time, it supports specialized, inclusive, and quality education for pupils with ADS, which is flexible and innovative in relation to the existing educational offer (procedures and choice of schools: combined education, ordinary education with support, specific classrooms in ordinary education and special education), in such a way that it responds to the individual needs and maximizes educational success,

personal development, and social inclusion. It moreover highlights the need to promote resources and support systems for people with ADS in adult life, guaranteeing their inclusion in working life, the possibility of developing an independent life outside the family home, legal protection of their rights, and full social participation in their own communities.

Overview of Current Treatments and Centers

At present, the map of ADS organizations in Spain is still incomplete, with provinces that have no specialized services, although associations have been created in some of them. In these situations, the majority of people with ADS with greater needs for support attend generalist centers for people with disabilities.

The proposal of family organizations in our country is to generate networks of support services that cover the needs of people with ADS throughout the life cycle and in all areas (diagnosis, early attention, schooling stage, training services, and support for the socioeconomic inclusion of adult persons in the workplace, at home, in leisure activities, etc.). In a parallel although not in a generalized manner, support services related to access to higher education and university especially have been established for people with ADS without the associated intellectual disability.

In relation to the models of intervention, there is quite widespread consensus over the need to incentivize actions based on educational objectives and the promotion of the strengths of people with ADS throughout their life.

The perspective of intervention is flexible, integrating the contributions of different psychoeducational models and, especially, those that offer greater scientific evidence: the TEACCH method, positive behavioral support, and different sociocommunicative methods.

Guaranteeing the quality of life for people with ADS and their families constitutes a clear objective for the majority of organizations and professionals, in such a way that the services are

oriented towards inclusion and the development of people in their own communities, as well as considering fundamental dimensions such as emotional and physical well-being, self-determination, social relations, etc.

If we center on the educational stage, the modalities of schooling that exist in Spain at present are as follows (Alonso and Cuesta 2017):

Ordinary Educational Centers

Ordinary classroom: In general, nowadays, in the field of Infant and Primary Education, a majority of children with ADS attend ordinary schools.

ADS classrooms located in ordinary educational centers: some centers, in their majority dependent on family associations, have classrooms located at ordinary centers (the number of specialized professionals at the center remaining unchanged).

Stable classrooms, an inexistent option in some autonomous regions. There is no official norm that regulates this approach to educational inclusion. The initiatives that have developed emerged from collaborative agreements between the provincial educational administrations, associations of autism, and ordinary centers where those classrooms are located. Under this modality, the pathway to inclusion in the ordinary classroom may be adapted in accordance with the strengths of each pupil. We can find stable classrooms in the three communities that are the object of our study. Stable classrooms may be found in the three communities that are studied in this paper.

Preferential centers of rehabilitation, in which the inclusion of the pupils with ASD is prioritized, because these centers have specialized support resources.

Combined or Mixed Schooling: permits the pupil to attend two different educational centers, generally one for special education and another for ordinary education, which makes it possible to combine the contribution of both centers. The times of permanence at each one will be established in an individual plan for each pupil, agreed between both centers.

Specific Special Education Centers: those in which all the classrooms are specifically for pupils with ASD.

General Centers of Special Education: welcome children with different types of disability.

With respect to adults, there are many organizations that have promoted support services, such as:

Day centers for adults, at which training programs are developed and the social and labor inclusion of people with ASD is promoted.

Employment support services, in which professional training is facilitated, intermediation with firms, labor practices, and incorporation in the workplace. Inclusion in the work place through supported employment has allowed the real incorporation of people with ASD in the world of work.

Different modalities of **housing services:** protected housing for people who require intermittent support, housing with permanent support, residences.

Leisure programs.

Programs that promote the practice of sports.

Support for access to higher education.

Many of the autism organizations have also specialized diagnostic services, which generally cooperate with the public services that are involved and that are complemented with programs of early attention.

The Spanish Autism Confederation (2009, 2016) has proposed a catalogue of support services for persons with ADS throughout their life, on the basis of the needs identified by organizations, professionals, and people with ASD. This document is called “Calidad de vida, hoy [Quality of life, today]” (Vidriales et al. 2015).

Overview of Research Directions

After the first large-scale investigation on autism in our country, conducted by GETEA Group

(Instituto de Salud Carlos III), another relevant initiative took place to dynamize this sector in Spain. This was the celebration of the *I Encuentro de Investigadores sobre Autismo* [First Meeting of Researchers on Autism], organized by the Centre of Applied Psychology of the Autonomous University of Madrid (Belinchón 2010). As follow-up to this meeting, a further initiative led to an up-to-date review of the different investigations that have contributed to greater understanding of autism from the perspective of neurobiology and genetics. The work also offers relevant information with practical implications for diagnosis and intervention (Alonso 2014).

Güemes et al. (2009) completed an investigation that evaluated the efficiency of psychoeducational interventions in the ASD.

Moreover, various investigations have been completed in relation to the needs of people with ASD and their families, with the purpose of promoting knowledge and raising awareness of the relevance of specialized support (Belinchón 2001; Belinchón et al. 2008; Saldaña et al. 2006).

One of the most relevant collaborative investigations into the quality of life of people with ASD has been promoted by Autism Spain in the project *Red para la Calidad de Vida* [Network for the Quality of Life]. This initiative in which 73 families, 70 people with ASD, and professionals have participated generated various results, among which is the design of an evaluation methodology of the specific quality of life for people with ASD, which contemplates the design of an information tool, ICalidad, that facilitates the evaluation process and includes a specific questionnaire (Vidriales et al. 2015).

Other investigations have referred to the needs emerging in relation to the support services: aging (Vidriales et al. 2016; Ruggieri et al. 2019), women with ASD, through the European project Autism in Pink, and the situation on the access of people with ASD to higher education and the university, in the framework of the European project Autism and Uni.

Participation in the European project ASDEU may also be highlighted, the objective of which is to analyze the social and economic costs of

autism; to review the existing programs on early detection and to develop proposals for their introduction and improvement; to prepare a plan for the improvement of professional training; to validate biomarkers associated with ASD; and to improve knowledge on diagnosis, comorbidity, and the effectiveness of care and support in adult life and in the elderly with a diagnosis of autism.

Finally, one of the more recent and relevant initiatives is the Bebe-Miradas Project, promoted by the Burgos Autism Association and the Miradas Foundation, consisting in the identification of signs in babies that warn of the presence of an Autism Spectrum Disorder (ASD), by means of “eye tracking,” referring them to specialized and specific intervention that prevents and minimizes the impact of the disorder in the development of the child.

Overview of Training

In parallel to the creation of groups of families and people with ASD, training and dissemination initiatives have started to emerge that to a great extent raise the awareness of public administrations and the development of professional practices that may have distanced themselves from psychoanalytic perspectives for the development of models based on a cognitive behavioral approach. A group of young professionals, linked to the first associations as technical advisors, were deeply involved in this process, such as Ángel Díez Cuervo and Ángel Riviére Gómez.

At present, the association of AETAPI professionals has been turned into a reference for training in autism care, through the biannual congresses that it organizes as well as thanks to other online training initiatives and one-day conferences on specialization that it has offered. The refresher course that this body promotes has seen over 250 participants in the three editions that it has celebrated up until today.

Likewise, AETAPI subscribes to and supports the training and specialization of professionals through collaborative agreements with different Universities (Autónoma de Madrid, Burgos, Sevilla, Zaragoza, and the Universidad Nacional a Distancia), and it collaborates in initiatives such

as the European IPA+ project *Inclusion of People with Autism in Europe. Towards a specialized training model for professionals*, in collaboration with other countries, in order to design a training curriculum that responds to the needs of professionals and the specialized organizations.

Social Policy and Current Controversies

The edition of the DSMV generated broad debate on the diagnosis of ASD among specialized professionals and the creation of a working group within the framework of AETAPI that conducts an exhaustive analysis of the implications that this new model had in the field of autism. One of the principal controversies was generated around the suppression of the diagnostic categories established in earlier editions, and especially one related with Asperger syndrome, because of its implications for the identification and the social recognition of the people who form part of this collective. At this time, agreement exists to continue gradually to include the terminology proposed in this new edition, without sidelining the concept of Asperger syndrome.

There are many challenges in the area of autism: increased prevalence generates new needs and the planning of different support services, from a flexible perspective, which contemplates diversity of demand in a collective characterized by great variability of degrees and manifestations of symptoms.

It is necessary to continue working to define, on the basis of the evidence contributed by science and experience, the most appropriate model to understand and to intervene in autism, burying interpretative proposals or “miracle cures” which are a cause of great concern to people with ASD and their families (AETAPI 2011; Ruggieri and Cuesta 2017).

The initiative of the government to promote the Spanish Strategy on Autism Spectrum Disorders has awakened great hope. It implies a step forward in the recognition of the rights of people who form part of this group and in the visibility of ASD as a condition in its own right, which generates specific needs and that makes the development

of specific support systems essential for the people who present them, throughout all stages of the life cycle.

References and Reading

- Alonso, J. R. (2014). *Investigaciones recientes sobre el autismo*. Valencia: Psycicom.
- Alonso, J. & Cuesta J. L. (2014). Necesidad de planes de formación interdisciplinar. Atención sanitaria en personas con Trastorno de Espectro Autista. *International Journal of Developmental and Educational Psychology*, N^o 1, 2, 547–553.
- Asociación Española de Profesionales del Autismo (AETAPI). (2011). *Propuesta para la planificación de servicios y programas para personas con trastornos del espectro del autismo y sus familias*. Retrieved from: http://aetapi.org/documentos_aetapi.htm
- Belinchón, M. (Coord.). (2001). *Las personas con Trastornos del Espectro Autista en la Comunidad de Madrid*. Madrid: Ediciones Martín & Macías
- Belinchón, M. (Coord.). (2010). *Investigaciones sobre el autismo en español. Problemas y perspectivas*. Madrid: Universidad Autónoma de Madrid. Centro de Psicología Aplicada.
- Belinchón, M., Hernández, J. M., & Sotillo, M. (2008). *Personas con Síndrome de Asperger. Funcionamiento, detección y necesidades*. Madrid: Fundación ONCE.
- Confederación Autismo España. (2009). *Autismo y Calidad de Vida, Hoy*. Madrid: Confederación Autismo España. Retrieved from http://www.sobretodo personas.org/phocadownload/Bibliografia_Discapacidad/Trastorno_del_espectro_autista_TEA/Autismo,%20calidad%20de%20vida:%20hoy.pdf.
- Confederación Autismo España. (2016). *Envejecimiento y Trastorno del Espectro del Autismo*. Madrid: Confederación Autismo España. Retrieved from http://www.autismo.org.es/sites/default/files/envejecimiento_informe_ejecutivo_version_web.pdf.
- Feinstein, A. (2016). *Historia del autismo. Conversaciones con los pioneros*. Ávila: Autismo Ávila.
- Guemes, I., Martín, M. C., Canal, R., & Posada, M. (2009). *Evaluación de la eficacia de las intervenciones psicoeducativas en los trastornos del espectro autista*. Madrid: Ministerio de Ciencia e Innovación. Instituto de Salud Carlos III.
- Martínez, M. A., Cuesta, J. L., Esteban, N., Lezcano, F., Casado, R., Arnaiz, J., & Pérez, L. (2010). Historia y situación actual del movimiento asociativo para las personas con trastornos del espectro autista en Castilla y León. *Siglo Cero*, 41(4)(236), 78–96.
- Ministerio de Sanidad. (2015). *Estrategia Española en Trastornos del Espectro del Autismo*. Retrieved from: http://www.autismo.org.es/sites/default/files/blog/adjuntos/estrategia_espanola_tea_ministerio_sanidad.pdf
- ONU. (2006) Convención Internacional de los Derechos de las Personas con Discapacidad. Disponible en <https://www.un.org/esa/socdev/enable/documents/tccconvs.pdf>
- Ruggieri, V., & Cuesta, J. (2017). *Autismo. Cómo Intervenir, desde la Infancia a la Vida Adulta*. Buenos Aires: Paidós.
- Ruggieri, V., Cuesta, J., Merino, M., & Arberas, C. (2019). Aging and autism: Understanding, intervention, and proposals to improve quality of life. *Current Pharmaceutical Design*, 25. <https://doi.org/10.2174/1381612825666191204165117>.
- Saldaña, D., Álvarez, R., Moreno, M., López, A. M., Lobatón, S., & Rojano, M. A. (2006). *Vida Adulta y trastornos del espectro autista Calidad de vida y empleo en Andalucía*. Sevilla: Federación Autismo Andalucía. Retrieved from http://sid.usal.es/idos/F8/FDO22145/estudio_vida_adulta.pdf.
- Vidriales, R., Cuesta, J. L., Plaza, C., & Hernández, M. (2015). Personas con Trastorno del Espectro del Autismo con necesidades intensas y generalizadas de apoyo: Estrategias para mejorar su calidad de vida. *Revista Española de Discapacidad*, 3(2), 101–115.
- Vidriales, R., Hernández, C., & Plaza, M. (2016). *Envejecimiento y Trastorno del Espectro del Autismo. Una etapa vital invisible*. Edita: Autismo España. Retrieved from: http://www.autismo.org.es/sites/default/files/envejecimiento_informe_completo_version_web.pdf.

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► SHANK 3

Sparrow, Sara

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Name and Degrees

Sparrow, Sara S. M.A. Ph.D. (May 9, 1933–June 10, 2010)

Major Appointments (Institution, Location, Dates)

- 2003 Professor Emerita, Yale University, Child Study Center and Department of Psychology; Senior Research Scientist, Yale Child Study Center
- 1989–2003 Professor of Psychology, Chief Psychologist, The Child Study Center, School of Medicine; Professor, Department of Psychology, Yale University
- 1986–1989 Professor of Psychology, Chief Psychologist, The Child Study Center, School of Medicine; Lecturer, Department of Psychology, Yale University
- 1977–1986 Associate Professor of Psychology, Chief Psychologist, The Child Study Center, School of Medicine; Lecturer, Department of Psychology, Yale University
- 1977–2002 Director, Psychology Internship Program, The Child Study Center, Yale University
- 1974–1977 Assistant Professor, The Child Study Center and the Department of Psychology; Chief Psychologist, The Child Study Center, Yale University
- 1974–1975 Visiting Professor, Child Development Department, Connecticut College, New London, Connecticut
- 1972–1974 Research Associate and Lecturer, Department of Psychology and The Child Study Center, Yale University
- 1972–1973 Acting Chief Psychologist, The Yale Child Study Center, Yale University
- 1970–1972 Research Associate, Department of Psychology, Yale University
- 1969–1970 Research Staff, Department of Psychology, Yale University
- 1968–1969 Postdoctoral Fellow, Department of Psychology, Yale University
- 1965 Director, Summer Speech Clinic, Speech Department, University of Florida
- 1962–1964 Coordinator, Speech and Hearing Program, Orange County Schools, Orlando, Florida
- 1958–1961 Speech Therapist, Orange County Schools, Orlando, Florida

Major Honors and Awards

- 2010 Lifetime Child Study Center Service Award
- 2009 Edgar Doll Award for “her outstanding research and sustained contributions to the understanding of intellectual and developmental disabilities,” APA Toronto
- 2009 Distinguished Career Award, International Neuropsychology Society, Atlanta, GA
- 2008 Outstanding Alumnus of the Year, University of Florida, College of Public Health and Health Professions, Gainesville, FL
- 2006 Distinguished Alumnus Award, University of Florida, College of Public Health and Health Professions, Gainesville, FL
- 2004–2005 President, Division 33, Mental Retardation and Developmental Disabilities, American Psychological Association
- 2003–2004 President-Elect, Division 33, Mental Retardation and Developmental Disabilities, American Psychological Association
- 2002–2003 President-Elect Designate, Division 33, Mental Retardation and Developmental Disabilities, American Psychological Association
- 2002 Career Scientist Award, The American Academy on Mental Retardation
- 2000–2002 National Research Council, Committee on Disability Determination for Mental Retardation, Washington, DC
- 1998 Invited Participant, NICHD Workshop on Measurements of Clinical Outcome, Surrogate Endpoints, and Diagnostic Markers in Pediatric Drug Trials. September 24–25, 1998 Rockville, MD
- 1998 Professional Achievement Award, 2nd Annual Learning Disorders Symposium, H.E.L.P. Group. September 18–19, 1998. Sherman Oaks, CA
- 1994–1997 Elected to the Board of Governors, International Neuropsychological Society
- 1993–1999 Coeditor, Journal of Child Neuropsychology
- 1993 Cochairperson, Human Resources Development Task Force, Center for Health and Human Services
- 1992 National Advisory Board: “Development and Implementation of a Plan to Assess the

Neurodevelopment of Infants and Children Exposed to Drugs in Utero." NIMH

1990 Facilitator: National Conference on Implementing Public Academic Linkages for Clinical Training in Psychology. NIMH and APA, Dec. 10–12, 1990

1990 Project Review Committee, National Institute of Mental Health Training Grants

1989 "National Conference on Clinical Training for Multidisciplinary Services for Seriously Emotionally Disturbed Children and Adolescents." Georgetown University Leavey Conference Center, Washington, DC

1989 Member Consensus Development Panel, National Institutes of Health, Treatment of Destructive Behaviors in Persons with Developmental Disabilities

1988 Fellow, American Psychological Association (Div. 33)

1987 Review Panel Member, Research Project "The Higher Cerebral Function Disorders in Children," NICHD, Washington, DC, 1987, 1988, Chairman 1989–1994

1985–1986 Chairman, Quality Review Committee, Connecticut State Planning Council on Developmental Disabilities

1984 Fellow, American Psychological Association (Div. 12)

1984 First Recipient, Connecticut Psychological Association "Award for Distinguished Effort by a Connecticut Psychologist, Utilizing Psychological Knowledge and Skills, Which Has Made Contribution to the Welfare of the Public," for development and publication of revised Vineland Adaptive Behavior Scales (1984, 1985), at Fairfield University, December 7, 1984

1984 Project Review Committee, National Institute of Mental Health Training Grants

1982 Elected to American Academy of Mental Retardation

1980–1986 Member, Connecticut State Planning Council on Developmental Disabilities (appointed by Governor).

1971–1972 Committee on Book Reading, International Reading Association

1970 Who's Who in American Women

1968 American Men of Science

Landmark Clinical, Scientific, and Professional Contributions

Vineland Adaptive Behavior Scales, Second Edition (Vineland-II)

Short Biography

Dr. Sara S. Sparrow was a North American psychologist, whose work focused on children and their developmental trajectories, ranging from intellectual giftedness to severe mental retardation. Dr. Sparrow made contributions to the fields of child neuropsychology and developmental disabilities across a wide range of diagnostic groups of children and also across cultures. She was particularly engaged with children with autism spectrum disorders, intellectual disabilities, and learning disabilities. Dr. Sparrow trained many mental health professionals at the doctoral and postdoctoral levels; she was the founder of the child psychology internship program at the Yale Child Study Center. She was also an influential and highly valued teacher and clinical supervisor within various Yale University programs. Several generations of her former trainees work all over the world, leading clinical science programs for children with special needs.

Dr. Sparrow's landmark contribution was the Vineland Adaptive Behavior Scales, Second Edition (Vineland-II), which is one of the most broadly used psychological assessment instruments used throughout the world. She was the senior author and a devoted promoter of this instrument. The Vineland is used extensively in clinical, educational, and research settings as a means of evaluating individuals' personal and social skills in everyday living. It was the first life-span, norm-referenced instrument measuring adaptive behavior in multiple domains. The Vineland is a particularly critical diagnostic tool in the assessment of individuals with intellectual and developmental disabilities, including autism spectrum disorders.

A native of Minneapolis, Minnesota, Dr. Sparrow graduated summa cum laude from

Montclair State College in New Jersey in 1958 and received her professional degrees from the University of Florida, M.A., in speech pathology in 1962 and Ph.D. in clinical psychology/clinical neuropsychology in 1968. She went to Yale in 1968 as a postdoctoral fellow in the Department of Psychology. She joined the department's research staff the following year, then served as acting chief psychologist at the Child Study Center in 1972–1973. She was appointed to the rank of assistant professor in 1974, associate professor in 1977, and full professor in 1986.

Dr. Sparrow's contributions to psychology included more than 120 articles and book chapters in the fields of child neuropsychology, learning disabilities, test development, and adaptive behavior in a broad range of child psychiatric conditions. Additionally, Dr. Sparrow maintained active clinical practice within the Yale Child Study Center, where she was Chief Psychologist from 1997 to 2002, and then continued working with patients and conducting research as Professor Emerita of Psychology and Advisor to the Yale Academic Skills Clinic until her untimely death. Beyond Yale, Dr. Sparrow assumed leadership roles in psychology at the national level. She served as President of Division 33 of the American Psychological Association (Intellectual and Developmental Disabilities), was a member of the National Research Council (National Academy of Sciences) Committee on Disability Determination for Mental Retardation, and was cofounder of the *Journal of Child Neuropsychology*.

Dr. Sparrow's scientific contributions have been recognized through numerous awards including Alumnus of the Year by the College of Public Health and Health Professions at the University of Florida; she and her husband, Dr. Domenic Cicchetti, shared the first Scientific Achievement Award given by the Connecticut Psychological Association; she was a recipient of the Edgar Doll Award from Division 33 of the APA which is the division's highest recognition of outstanding scientific contributions to the field of intellectual and developmental disabilities; and in

2002 Dr. Sparrow received the Career Scientist Award from the American Association of Mental Retardation for her lifetime contributions to the field.

See Also

- [Vineland Adaptive Behavior Scales \(VABS\)](#)

References and Reading

- Sparrow, S. S., & Cicchetti, D. V. (1987). Adaptive behavior and the psychologically disturbed child. *The Journal of Special Education*, 21, 89–100.
- Sparrow, S. S., & Davis, S. M. (2000). Recent advances in the assessment of intelligence and cognition. *Journal of Child Psychology and Psychiatry*, 41, 117–131.

Spastic Diplegia

- [Cerebral Palsy](#)

Spastic Paralysis

- [Cerebral Palsy](#)

Speaker Versus Listener-Oriented Disfluency

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Definition

Deficits in pragmatic language and social communication are characteristic of verbal individuals with autism spectrum disorders (ASD). Disfluencies are pauses and interruptions, which

break the continuity of speech outputs. Disfluencies are often classified into different categories, primarily including pauses, repetitions, and repairs. These disfluencies are produced by all people in the normal course of speaking, and estimates place their occurrence at 6–10 per 100 words produced. Crucially, these disfluencies are distinguished from “dysfluency,” which refers to pathological fluency issues (i.e., stuttering or stammering). However, it is important to note that several reports have indicated stuttering-like disfluency in some individuals with ASD (see Scott 2015).

Disfluencies may serve pragmatic purposes in that they cue problems within the speaker for the benefit of the listener. Speaker-oriented disfluencies are thought to be due to speaker-internal factors (i.e., difficulties associated with the process of planning and producing language). In contrast, listener-oriented disfluencies are believed to be produced in interactive dialogue, specifically for the listener and for the benefit of communicative exchanges/efficiency. This entry provides a review of existing evidence relating to speaker- and listener-oriented disfluency in ASD. Evidence for speaker-oriented (or speaker-internal causes of) disfluency could come from different sources. However, the pattern one would expect is that there should be negative correlations between rates of disfluency and speaker-internal variables. In short, higher rates of disfluency would be associated with lower ability individuals, including individual differences variables, such as executive functions, intelligence, and also higher ASD symptom severity. In contrast, listener-oriented disfluency should show positive correlations between higher social-communication abilities, and also higher abilities in individuals differences variables. The debate between speaker- and listener-oriented disfluency has been applied to ASD, given that individuals with ASD tend to take an egocentric approach to dialogue and as such should be less likely to produce disfluencies that are specifically helpful for listeners in conversation.

Historical Background

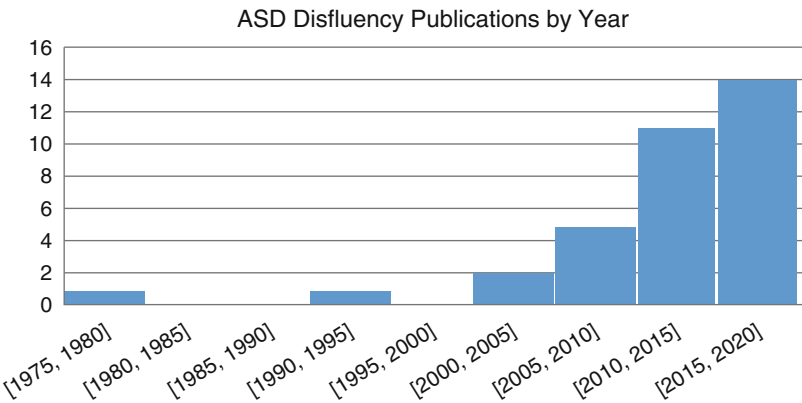
It has long been known that language impairments are fundamental to ASD (e.g., Rutter 1970). There has been a great deal of recent interest in the production of disfluency by individuals with high-functioning forms of ASD. However, this line of research has its beginnings in the 1970s, and the first report of excessive disfluency production in ASD was provided by Simmons and Baltaxe (1975). Those authors found that approximately half of their sample showed hesitations, repetitions, prolongations, and nonfluencies. (Definitions and examples of these categories were not provided.) Abnormalities in the fluency of speech outputs in the 1970s were classified as faults in rhythm, and tightly linked with abnormalities in prosody. To provide some historical and current context, a histogram is provided showing the number of studies examining disfluent speech in ASD over time (see Fig. 1). This includes PhD dissertations, conference presentations, and published research studies. As can be seen in the figure, a clear uptick occurred approximately 15 years ago.

Current Knowledge

This review follows the standard disfluency classification scheme from psycholinguistics (i.e., filled pauses, unfilled pauses, repetitions, repairs, and “other”) and presents results of each type of disfluency separately. The main focus is the debate about which forms of disfluency are speaker-oriented and which are listener-oriented. The latter (i.e., communicative purposes) are the type hypothesized to be produced less often in individuals with ASD, due to their impairments in socially oriented pragmatic language use (De Marchena and Eigsti 2016; Tager-Flusberg et al. 2005). These impairments, specified in the DSM-5, include failure of normal back-and-forth conversation and failure to initiate or respond to social interactions. However, the criteria also specify that individuals with deficits in social communication but not

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Fig. 1 Histogram showing frequency of studies related to disfluent speech in ASD



otherwise meeting the criteria for ASD should be screened for social (pragmatic) communication disorder, a recent addition to the DSM-5, which overlaps substantially with ASD diagnostic criteria. The conversational profile for social-communication disorder includes difficulties following social rules of communication in conversation, which leads to problems (1) communicating effectively and engaging in a social manner, (2) matching speech outputs to context, (3) rephrasing when misunderstood, (4) making appropriate inferences, and (5) understanding non-literal language. These kinds of issues are all related to socially oriented *pragmatic* language use.

With these general pragmatic language issues in mind, it is important to situate disfluency production within a theoretical context. The first theoretical context is the process of language production. Many prominent models of production assume a stage-based architecture. For example, Levelt (1989) proposed a three-stage model consisting of conceptualization, formulation, and articulation. The basis of any model of production is the conversion of nonlinguistic thoughts into the articulated words and syntax structures of speech output. Most models also assume some type of monitoring mechanism, which is responsible for detecting faults or problems in pre-articulated speech plans. As mentioned previously, speaker-oriented disfluencies are likely related to difficulties arising in one or more stages of production. Difficulties could arise from a number of causes (e.g., planning

problems, memory retrieval difficulty, lapses of attention, or individual difference variables). For example, if lemma selection (choosing which words to convey the intended message) involves inhibitory control, then we should observe individual differences in inhibition relating to rates of disfluency. The second theoretical context involves theories of ASD, which are surprisingly not often considered in disfluency studies. If interactive dialogue relies on taking the listeners' needs into account or being overtly helpful to the listener via disfluency, then Theory of Mind has implications for listener-oriented issues (Baron-Cohen et al. 1985). In contrast, if disfluency production is more related to individual differences in executive functions, then theories of Executive Control in ASD become relevant for speaker-oriented (or speaker-internal) issues (e.g., Hill 2004).

With this theoretical context in mind, a couple of caveats should be noted. The first is that despite existing disfluency studies examining children, adolescents, and adults, there is currently not sufficient evidence to determine how disfluency production changes over the course of development. Therefore, in the following review, there is no discussion of developmental effects. The second is that studies have used both controlled and more naturalistic speaking situations, and again, there are limited studies of each type, which limits firm conclusions about how task differences affect disfluency production. Thus, task differences are only highlighted in the event of conflicting and/or mixed findings.

Filled Pauses

Many studies have focused on the production of “fillers,” which in English, primarily constitute “UMs” and “UHs.” The production of filler-type disfluencies is believed to arise from difficulties experienced by the speaker, for example, in thinking or planning what one wants to say (Clark 1994). In short, the speaker experiences a problem, they know it, and in response, produce *UM* or *UH* to signal the listener. Therefore, fillers serve communicative purposes in interactive dialogue. Past studies (e.g., Clark and Fox Tree 2002) have suggested that speakers produce fillers as a collateral signal to the listener that they are not finished speaking, want convey some uncertainty, or signal an upcoming delay (i.e., they need more planning time).

Research has shown that individuals with high-functioning autism (HFA) produce fewer fillers compared to controls, and given the social-pragmatic deficits in ASD, fillers are thought to provide evidence of listener-oriented disfluency. *UMs* tend to occur at the beginning of prosodic phrases and are typically followed by longer pauses than are *UHs*. Some studies (e.g., Lake et al. (2011) and MacFarlane et al. (2017)) did not differentiate *UM* and *UH* but did report fewer filled pauses in ASD. In contrast, Suh et al. (2014) did not find significant group differences (ASD vs. control) in a monologue story telling task. However, in a key study, Irvine et al. (2016) found that *UM* production differentiated groups, but *UH* did not. Moreover, *UM* rate was correlated with ASD symptom severity, but did not correlate with other key individual difference variables (age, verbal IQ, nonverbal IQ, language ability, or executive function). Further studies have confirmed this dissociation between *UM* and *UH* in ASD (e.g., Gorman et al. 2016). A key advancement in filler production was the calculation of *UM* to *UH* ratio (i.e., $UM/(UM + UH)$). On the basis of the evidence, it has been concluded that *UM* production reflects social-communication skills and is therefore a clear listener-oriented type of disfluency (see Table 1, for mean *UM-UH* ratios across studies). Moreover, low *UM* production has even been postulated as clinical-diagnostic marker of ASD, specifically

Speaker Versus Listener-Oriented Disfluency, Table 1 Results for *UM/UH* ratio across studies

Study	Control	ASD
Gorman et al. (2016) (estimated from Fig.)	.77	.40
Heeman et al. (2010) (turn initial)	.69	.47
Heeman et al. (2010) (utterance initial)	.63	.30
Heeman et al. (2010) (utterance medial)	.80	.40
McGregor and Hadden (2018)	.92	.70
Parish-Morris et al. (2017) (boys)	.78	.56
Means	.77	.47

in boys with the disorder (McGregor and Hadden 2018). Females with ASD, in contrast, were not different than controls in *UM-UH* ratio. However, this gender finding should be replicated before firm conclusions are drawn.

Two additional findings are worth highlighting. The first finding relates to the location of filled pauses. Heeman et al. (2010) analyzed the location of filled pauses under the assumption that location may reveal information about their function. Filled pauses were coded as to whether they occurred turn initially, utterance initially, or mid-utterance. *UH* rates were similar across all three positions. However, participants with ASD used *UM* less frequently across all three positions. *UMs* were half as frequent at turn-initial positions, one-fifth as frequent at utterance-medial positions, and utterance-initial position fell in between. The second finding relates to speaking task. The highest rates of filled pauses occurred in conversation, as compared to a describing task and a play task. Crucially, *UMs* seemed to vary by position and task type, but the differences between ASD and controls remained significant, whereas *UHs* showed no difference between groups despite some variance in position and task type. It is currently unknown how much of the task differences are associated with the face-to-face nature of the speaking situation, but filled pauses are typically not produced in monologue speaking situations. Interestingly, Heeman et al. also reported no significant differences in the length of pauses following fillers

between the two groups. In the psycholinguistic literature, it has been argued that *UM* is used as a signal of an upcoming long delay and *UH* an upcoming short delay (Clark and Fox Tree 2002).

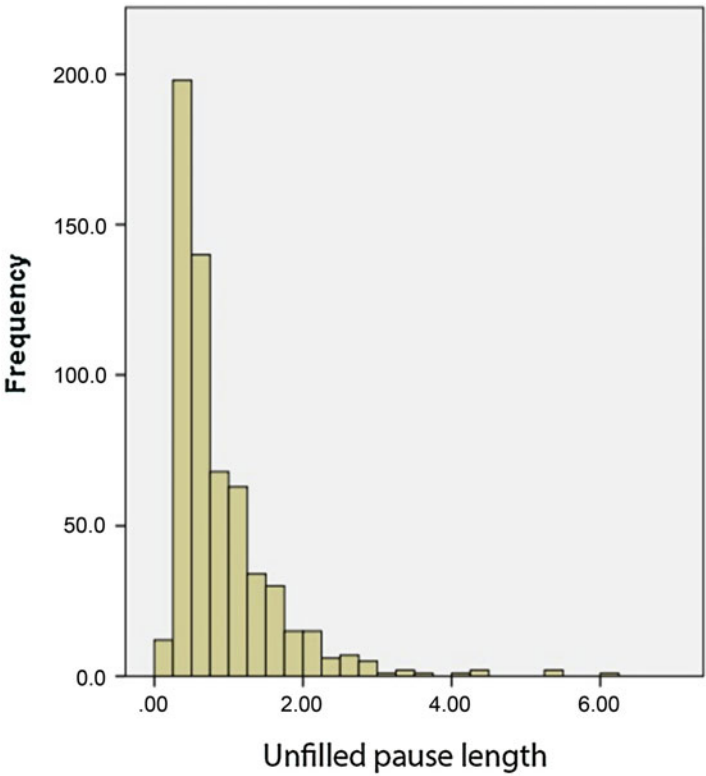
In summary, individuals with ASD do not produce filled pauses, in particular, *UMs* as frequently as do controls. Estimates suggest that *UMs* are produced approximately 2-to-1 over *UHs* in controls, but approximately 1-to-1 in ASD. Thus, it seems that individuals with ASD tend to produce unfilled pauses instead of filling pauses with *UM*. Moreover, differences in filled pause production do not seem to be related to age, more general language abilities, IQ, or executive functions. Several studies have examined whether *UM* usage is correlated with ASD symptom severity. Two studies reported significant negative correlations (Irvine et al. 2016; Parish-Morris et al. 2017), and two have not found a significant association between *UM* production and ASD symptom severity (Gorman et al. 2016; McGregor and Hadden 2018).

Unfilled (or Silent) Pauses

Several studies have also examined the production of unfilled or silent pauses. Both Lake et al. (2011) and Shriberg et al. (2001) reported higher rates of unfilled pauses in ASD. One potential issue with unfilled pauses is the criterion of what counts as an unfilled pause. For example, Lake et al. defined unfilled pauses as 3 sec or longer, whereas Engelhardt et al. (2017) used 250 ms. This may or may not be a problem, as research has only begun to examine the distributions of the length of pauses between ASD and controls to determine whether one has a tendency to produce particularly long or short pauses. In general, the distributions of pauses (in controls) are positively skewed (see Fig. 2 for an example). In monologue production tasks, the group effect (ASD vs. control) on unfilled pauses appears to turn on how groups are matched. Age and gender matched groups tend not to show differences. The Engelhardt et al. study utilized a memorize-and-repeat sentence production paradigm, which relied heavily on memory. Their results showed a

Speaker Versus Listener-Oriented Disfluency,

Fig. 2 Histogram showing an example of the length and frequency of unfilled pauses in typically developing adults



group effect (ASD vs. unmatched controls) over and above variance accounted for by working memory (i.e., working memory ability and ASD status were both significant predictors of unfilled pauses).

Thurber and Tager-Flusberg (1993) also investigated unfilled pauses, but their conclusion was that individuals with ASD expended less effort in constructing narrative discourses (i.e., in a story re-telling task). As a result, they found the opposite pattern from previous studies. That is, ASD participants produced fewer nongrammatical pauses, because their stories were shorter, less complex, and had less lexical diversity, which resulted in between-group differences in cognitive load. In contrast, grammatical pauses (i.e., pauses produced at major syntactic boundaries or points of emphasis in speech) were not significantly different between groups.

Heeman et al. (2010) examined the length of pauses between conversational turns under the hypothesis that individuals with ASD would have less awareness or concern about social cues to minimize silence, or alternatively, whether longer pauses would be due to slower planning and overall slower response times. Thus, they believed that pauses could be due to either speaker-internal factors or poor social-pragmatic communication skills. Their results showed mean pause lengths of 876 ms (control) vs. 1115 ms (ASD). Similar between-group mean differences were observed, specifically following questions. Wiklund and Laakso (2019) also reported longer pauses within a conversational turn. Perhaps more interestingly, Heeman et al. also examined the length of pauses depending on *activity type*. In conversation, the difference between groups following a question was not significant, but in a play activity, there were significant differences and the mean difference between groups was much higher (mean difference of 569 ms). These findings were independent of threshold, as the authors were able to determine the length of silence between all turns in the conversation. However, this study did not control for individual differences measures, and so, to the extent that these data shed light on the speaker- vs listener-oriented issue remains to be determined. That is, studies

examining the length of pauses in dialogue did not consider individual difference variables or ASD symptom severity.

At present, most evidence suggests that unfilled pauses are produced more frequently in individuals with ASD, but at the same time, this effect also seems to be affected by (group) matching variables, which suggests speaker-oriented production issues. Clearly, more work is needed to examine pause frequency differences and length of pause differences based on speaking task. Also, given the findings of Thurber and Tager-Flusberg, more attention concerning the type of pause is warranted.

Repetitions

The first report of abnormal repetitions was provided by Simmons and Baltaxe (1975). They observed high rates of repetitions in four of their seven children with ASD. Other studies have reported similar findings (Lake et al. 2011; Shriberg et al. 2001; Suh et al. 2014), but one study found no significant differences in repetitions (Thurber and Tager-Flusberg 1993). One key issue with repetitions concerns the relationship between repetitions and verbal IQ. Engelhardt and colleagues across a range of (non-ASD) studies have shown that differences in repetitions are highly related to verbal IQ (e.g., Engelhardt et al. 2019; Engelhardt et al. 2013). In an ASD study, when verbal IQ was covaried, the group effect (ASD vs. control) was no longer significant (Engelhardt et al. 2017). This study highlighted a key issue with respect to study protocols and matching of groups on individual difference variables. Verbal IQ is a particularly interesting individual difference measure with respect to ASD because verbal IQ in ASD often shows a large range. That is, a fair number of individuals with ASD have higher verbal IQ than controls, and others, depending on the overall severity of ASD, are generally lower functioning and have lower verbal IQ. At this point, it seems that the tendency to repeat words and phrases is likely due to a specific speaker-internal factor and that if differences in verbal IQ are taken into account, then individuals with ASD do not show higher rates of repetitions than do controls.

It is important to bear in mind, however, that the proportion of variance explained by verbal IQ is typically between 25% and 50%, and so, other (unknown) factors likely contribute to variance in repetitions. At this point, it is also unclear why verbal IQ is related to repetitions. It is also important to note that no study to date has systematically examined repetitions and other language ability measures (e.g., Clinical Evaluation of Language Fundamentals, which assess expressive and receptive language abilities).

Repairs

Repairs are also referred to as revisions and false starts in the literature. With repairs, the speakers stop speaking and then restart with a new word or phrase. As such, these disfluencies reveal instances in which speakers correct, clarify, or elaborate on the content of their output. This type of disfluency presents the most complicated picture in terms of the speaker- vs. listener-oriented debate, because repairs have been shown to be associated with many different individual difference variables. There are also mixed findings in the literature concerning whether individuals with ASD produce more repairs than do controls. However, the majority of studies show that individuals with ASD do produce more repairs (De Marchena and Eigsti 2016; Engelhardt et al. 2017; Shriberg et al. 2001; Sisskin 2006; Suh et al. 2014). The one study which concluded that repairs are a listener-oriented form of disfluency did so simply on the basis that individuals with ASD produced fewer repairs. In a later study, Engelhardt et al. found the opposite, individuals with ASD produced more repairs than controls. Engelhardt et al. reported that the increased tendency to produce repairs was associated with several Autism Quotient subscales (i.e., autism symptom severity). In addition, the group on repair findings held even when covarying individual differences and demographic variables, and so, with the exception of Lake et al., it appears that more repairs are produced by individuals with ASD.

Two additional points are worth mentioning here. The first is the notion of self-corrections. Obviously, certain types of errors in the content

of speech will lead to conversational breakdowns via the risk of mis-communication. Therefore, the only way to “rescue” a listener-oriented conclusion about repairs is to assume that higher rates of repairs are due exclusively (or largely) to situations in which the speaker needs to correct an outright mistake. Similar points about self-corrections were made by both De Marchena and Eigsti (2016) and Wiklund and Laasko (2019). The former found higher rates of repairs in a shared (common ground) condition in controls compared to ASD, suggesting that individuals with ASD did not adjust, correct, or clarify when it was appropriate to do so. Wiklund and Laasko argued that individuals with ASD showed marked deficits in linguistic and morpho-syntactic abilities, but were able to monitor sufficiently well these deficits and to correct in the event of possible misunderstanding for listeners. This again suggests some listener-oriented motivation for the production of repairs, but obviously, more research is needed to definitively understand whether some repairs may have a listener-oriented function.

The second point is that rates of repairs differ based on the speaking task. For example, MacFarlane et al. (2017) found that ADOS dialogue was a much stronger predictor of disfluency rates compared to telling a story from a book, despite not observing differences between ASD and controls. However, MacFarlane et al. included some additional detail about the nature of the repairs. They distinguished whether the repair was a true error (e.g., *turn left...right*), what MacFarlane et al. referred to as a “revision,” or whether it involved a deletion (e.g., *my old dog...my dog.*) or insertion (e.g., *my dog...my old dog.*) of linguistic material. They also classified false starts (e.g., *Dogs...My friend likes dogs.*) as a separate category. This more detailed classification scheme may prove informative in future studies to elucidate the specific form of repairs produced by individuals with ASD, as well as shedding light on the speaker- vs. listener-oriented issue.

In summary, evidence clearly points to individuals with ASD producing more repairs than controls. Also, there is no concrete evidence at this

point to suggest that repairs are a listener-oriented form of disfluency. Instead, the tendency to produce repairs is related to many different types of individual difference variables and also to ASD symptom severity.

Other (Interjections, Prolongations, and Atypical Disfluency)

“Other” forms of disfluency have received much less empirical investigation compared to the types reviewed above in both the clinical and psycholinguistic literatures. This is partially due to the fact that they are produced much less frequently overall and also they do not fit well within the typical classification schemes and as such, are classified as “other.” Obviously, interjections (*I mean, you know*, etc.) could be communicatively beneficial in terms of signaling something important to a listener. No study to date has examined the production of interjections in ASD, but Heeman et al. (2010) did analyze “discourse markers” and “acknowledgments.” Prolongations (e.g., *theeeeeee* vs. *the*) have been noted in the psycholinguistic literature, as a mechanism in which the speaker “buys” themselves more time, presumably due to difficulty with an upcoming element in the speech stream (Arnold et al. 2004). However, again, no study has examined the production of prolongations in ASD.

Atypical disfluencies are quite interesting: They were noted in the original studies in the 1970s but have rarely been classified and/or observed in later studies (cf. Plexico et al. 2010; Scaler Scott et al. 2014). Some common examples are final syllable repetitions (e.g., *speech. . .eech, animal. . .mal*) and stuttering-like disfluency, including part-word perseverations (e.g., *d.d.d.d. d.dog*), single syllable repetitions (e.g., *I.I.I.I*), and dysrhythmic blocks. Readers interested in atypical disfluency and cluttering (i.e., excessively fast speech that overloads the production system) and the incidence rates of these things in ASD should consult Scott (2015), Scaler Scott et al. (2014), and Sisskin (2006).

Summary

This review has focused on how individuals with ASD show differences in disfluency in their

speech outputs compared to controls. Some of these can be clearly linked with speaker-internal factors and some with more listener-oriented functions. It is also important to note that there are several reports that suggest that some types of disfluency are associated with comprehension problems for listeners, in particular, repairs (see Wiklund and Laasko 2019), which suggests that the production of disfluency can be communicatively problematic. Filled pauses provide the clearest example of listener-oriented disfluency, and here, the research shows that individuals with ASD produce fewer *UMs* than do controls. The rate of *UHs* is not affected. Unfilled pauses are produced more frequently in individuals with ASD, and particularly, in unmatched samples. The evidence tends to indicate that unfilled pauses are related to speaker-internal factors and as such, are likely a speaker-oriented form of disfluency. Likewise, repetitions are highly associated with individual differences in verbal IQ, and at this point, not linked to ASD (cf. Suh et al. 2014). Finally, repairs have been linked with executive functions and individual differences in IQ. However, they are also produced more frequently in individuals with ASD. This may be due to more basic language-related impairments. The strength of the relationships between repairs and individual difference variables and ASD symptom severity are much more variable (perhaps due to differences in task demands) as compared to the other types of disfluency.

Future Directions

Below are two “general” future directions (points 1 and 2) which are based on re-occurring themes arising throughout this review. I have also highlighted two “specific” future directions (points 3 and 4) which take the study of disfluency in ASD in novel and exciting new directions.

1. A recurring theme throughout the review was the differences in task demands associated with the speaking situation in which disfluencies were analyzed. For example, does the task involve monologue or dialogue and is the

speech scripted or naturalistic. Many studies have examined disfluencies produced in ADOS assessments, which is an excellent source of data given its widespread use and semi-structured nature. Relatedly, what are the cognitive load differences associated with the speaking task? It is likely that overall rates of disfluency (and type of disfluency produced) will differ depending on the speaking situation and task demands. Further studies are needed to assess disfluency production when speaking with and without eye contact and about emotional topics.

2. With respect to control variables and the matching protocols between groups (ASD vs. control), further research is needed to determine which variables are key so that confounds can be avoided. Past research suggests that demographics (age, gender), individual difference variables (specifically verbal IQ, nonverbal IQ, executive functions), language abilities, and comorbid disorders are all important in some situations. These issues are especially important for speaker-oriented disfluencies. There are also a number of issues that need to be investigated concerning the relationship between verbal IQ and repetitions.
3. A recent study has linked disfluency production to particular genetic signatures in mothers who had a child with a developmental disorder (Klusek et al. 2018). To establish a relationship between an etiologically more basic (causal) factor and disfluent speech is an exciting avenue of research. It remains to future research to determine how these genetic differences are linked to structural-functional brain abnormalities, resulting in a behavioral profile characterized by fluency issues.
4. The time and cost associated with disfluency analysis is immense. It requires hand coding of the data, typically by a research team, in order to gain the requisite inter-rater reliability, especially for more subjective forms of disfluency (e.g., unfilled pauses). If we are to take McGregor and Hadden's suggestion of using disfluency as a clinical-diagnostic marker of ASD seriously, the field needs to simplify, speed, and reduce cost. Therefore, automated

annotation of speech samples is critical. Currently, automated disfluency analyses only detect disfluency generally, not specific categories or subcategories (i.e., filled pauses, repetitions, repairs, etc.) (see Morley et al. 2014).

See Also

- [Pragmatic Language Impairment](#)
- [Pragmatics](#)
- [Reciprocal Communication/Interaction](#)
- [Social Communication](#)
- [Speech/Communication Disabilities](#)
- [Verbal Communication](#)

References and Reading

- Arnold, J. E., Tanenhaus, M. K., Altmann, R. J., & Fagnano, M. (2004). The old and the new: Disfluency and reference resolution. *Psychological Science, 15*, 578–582.
- Baron-Cohen, S., Leslie, A. M., & Firth, U. (1985). Does the autistic child have a “theory of mind”. *Cognition, 21*, 37–46.
- Clark, H. H. (1994). Managing problems in speaking. *Speech Communication, 15*, 243–250.
- Clark, H. H., & Fox Tree, J. E. (2002). Using uh and um in spontaneous speaking. *Cognition, 84*, 73–111.
- De Marchena, A., & Eigsti, I.-M. (2016). The art of common ground: Emergence of a complex pragmatic language skill in adolescents with autism spectrum disorders. *Journal of Child Language, 43*, 43–80.
- Engelhardt, P. E., Nigg, J. T., & Ferreira, F. (2013). Is the fluency of language outputs related to individual differences in intelligence and executive function? *Acta Psychologica, 144*, 424–432.
- Engelhardt, P. E., Alfridijanta, O., McMullon, M. E. G., & Corley, M. (2017). Speaker- vs. listener-oriented disfluency: A re-examination of arguments and assumptions for autism spectrum disorders. *Journal of Autism and Developmental Disorders, 44*, 2885–2898.
- Engelhardt, P. E., McMullon, M. E. G., & Corley, M. (2019). Individual differences in the production of disfluency: A latent variable analysis of memory ability and verbal intelligence. *Quarterly Journal of Experimental Psychology, 72*, 1084–1101.
- Gorman, K., Olson, L., Presmanes Hill, A., Lunsford, R., Heeman, P. A., & van Santen, J. P. H. (2016). *Uh* and *um* in children with autism spectrum disorders or language impairment. *Autism Research, 9*, 854–865.
- Heeman, P. A., Lunsford, R., Selfridge, E., Black, L., & van Santen, J. (2010). Autism and interactional aspects of dialogue. In the proceedings of the 11th annual

- meeting of the special interest group on discourse and dialogue. (pp. 249–252).
- Hill, E. L. (2004). Executive dysfunction in autism. *Trends in Cognitive Sciences*, 8, 26–32.
- Irvine, C. A., Eigsti, I.-M., & Fein, D. A. (2016). *Uh, Um, and autism: Filler disfluencies as pragmatic markers in adolescents with optimal outcomes from autism spectrum disorder. Journal of Autism and Developmental Disorders*, 46, 1061–1070.
- Klusek, et al. (2018). Curvilinear association between language disfluency and FMR1 CGG repeat size across the normal, intermediate, and permutation range. *Frontiers in Genetics*. <https://doi.org/10.3389/fgene.2018.00344>.
- Lake, J. K., Humphreys, K. R., & Cardy, S. (2011). Listener vs. speaker-oriented aspects of speech: Studying the disfluencies of individuals with autism spectrum disorders. *Psychonomic Bulletin and Review*, 18, 135–140.
- Levelt, W. J. M. (1989). *Speaking: From intention to articulation*. Cambridge, MA: MIT.
- MacFarlane, H., Gorman, K., Ingham, R., Presmanes-Hill, A., Papadakis, K., Kiss, G., & van Santen, J. (2017). Quantitative analysis of disfluency in children with autism spectrum disorder or language impairment. *PLoS One*, 12(3), e0173936.
- McGregor, K. K., & Hadden, R. R. (2018). “Um” fillers distinguish children with and without ASD. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-018-3736-1>.
- Morley, E., Hallin, A., Roark, B. (2014). *Challenges in automating maze detection*. In Proceedings of the ACL Workshop on Computational Linguistics and Clinical Psychology.
- Parish-Morris, J., et al. (2017). Linguistic camouflage in girls with autism spectrum disorder. *Molecular Autism*, 8, 48–60.
- Plexico, L. W., Cleary, J. E., McAlpine, A., & Plumb, A. M. (2010). Disfluency characteristics observed in young children with autism Spectrum disorders: A preliminary report. *Perspectives on Fluency and Fluency Disorders*, 20, 42–50.
- Rutter, M. (1970). Autistic children: Infancy to adulthood. *Seminars in Psychiatry*, 2, 435–450.
- Scaler Scott, K., Tetnowski, J. A., Flaitz, J. R., & Yaruss, J. S. (2014). Preliminary study of disfluency in school-aged children with autism. *International Journal of Language and Communication Disorders*, 49, 75–89.
- Scott, K. S. (2015). Dysfluency in autism spectrum disorders. *Procedia – Social and Behavioral Sciences*, 193, 239–245.
- Shriberg, L. D., Paul, R., McSweeney, J. L., Klin, A., Cohen, D. J., & Volkmar, F. R. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning autism and Asperger syndrome. *Journal of Speech, Language, and Hearing Research*, 44, 1097–1115.
- Simmons, J. Q., & Baltaxe, C. (1975). Language patterns of adolescent autistics. *Journal of Autism and Childhood Schizophrenia*, 5, 333–351.
- Sisskin, V. (2006). Speech disfluency in Asperger’s syndrome: Two cases of interest. *Perspectives on Fluency and Fluency Disorders*, 16, 12–14.
- Suh, J., Eigsti, I., Naigles, L., Barton, M., Kelly, E., & Fein, D. (2014). Narrative performance of optimal outcome children and adolescents with a history of an autism spectrum disorder (ASD). *Journal of Autism and Developmental Disorders*, 44, 1681–1694.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders, Vol. 1: Diagnosis, development, neurobiology, and behavior* (3rd ed., pp. 335–364). Hoboken: Wiley.
- Thurber, C., & Tager-Flusberg, H. (1993). Pauses in the narratives produced by autistic, mentally retarded, and normal children as an index of cognitive demand. *Journal of Autism and Developmental Disorders*, 23, 309–322.
- Wiklund, M., & Laasko, M. (2019). Ungrammatical utterances and disfluent speech as causes of comprehension problems in interactions of preadolescents with high functioning autism. *Clinical Linguistics and Phonetics*, 33, 654–676.

Special Education

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Definition

Special education is a broad term that refers to a variety of direct instructional and related services that are available for students ages birth–21 who meet specified criteria for eligibility under the Individuals With Disabilities Education Act (IDEA). These services are designed to give the eligible student a “free and appropriate public education.”

See Also

- [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

- IDEA Partnerships. Retrieved 11 July 2011 from www.fape.org
- Nation Dissemination Center for Children with Disabilities. Retrieved 11 July 2011 from www.nichcy.org
- Turnbull, A., Wehmeyer, M. L., & Turnbull, R. (2009). *Exceptional lives: Special education in today's schools* (pp. 8–21). Upper Saddle River: Prentice-Hall.
- U.S. Department of Education. Retrieved 11 July 2011 from www.ed.gov
- Vaughn, S., Bos, C., & Schumm, J. S. (2010). *Teaching exceptional, diverse, and at-risk students* (pp. 2–7). Upper Saddle River: Prentice-Hall.

Special Education Classroom

- [Self-Contained Classroom](#)

Special Education Needs

- [Special Needs](#)

Special Education Service Utilization in ASD

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Definition

The investigation of what special education services (e.g., speech, occupational, physical therapies; 1:1 aide support; behavior support plans; classroom placements; and academic supports) children with autism spectrum disorder (ASD) currently access.

Historical Background

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by symptoms of social-interaction and social-communication impairments and restricted and repetitive interests and behaviors (American Psychiatric Association [APA] 2013). These prominent features of ASD often limit students' functioning in schools (Wei et al. 2014). In addition to these core diagnostic symptoms, many children with ASD present with problem behavior (e.g., noncompliance, aggression, and disruptive behaviors; Ashburnere et al. 2010), and other associated difficulties. As a result, many children with ASD receive long-term intervention, often in the school setting (Kang-Yi et al. 2016). Generally these services in the schools address symptoms and behavior that interfere with overall learning and functioning in the school setting (Kang-Yi et al. 2016), which may include academic, behavioral, and therapeutic supports (Kang-Yi et al. 2016; McDonald et al. 2019; Wei et al. 2014; White et al. 2007). Given the amount of time spent in the school setting, access to services in schools is critically important for children with ASD (McDonald et al. 2019; Narendorf et al. 2011) and may be the most accessible service option (White et al. 2007). In addition, services in the schools often lead to service provision outside of the school setting (White et al. 2007).

Prevalence estimates of children with ASD have documented an increase of 29% between 2008 and 2010, with the majority of this increase attributed to the growing number of children with ASD without intellectual disability (Center for Disease Control and Prevention [CDC] 2014, 2016). For example, the CDC (2016) documented that in 2012, the number of students identified with ASD with average or above average cognitive ability had risen to 43.9%. Due to the growing number of children with ASD in the schools, special education services are challenged (Yeargin-Allsop et al. 2003).

According to the USDOE (2017) Annual Report, 9.1% of the children ages 6–21 accessing special education services were served under the special education classification of Autism.

However, the number served under the classification of Autism may be misleading and represent an underestimate of the number with a clinical diagnosis of ASD served in special education (CDC 2014; McDonald et al. 2019; Rubenstein et al. 2018). For example, Rubenstein et al. (2018) examined special education trends of 8-year-olds ($n = 6010$) who had been diagnosed with ASD from 2002 to 2010, and discovered that more than 36% of these children did not receive special education eligibility under the classification of Autism during each surveillance year. These findings are a low estimate compared to those reported by the CDC (2014), who found that between 30% and 69% of students diagnosed with ASD received special education services under the classification of Autism. However, Rubenstein et al. (2018) also noted that the number of children accessing special education services via a classification of Autism increased by 3.6 percentage points from 2002 to 2010, with the greatest proportional increase documented amongst Hispanic students diagnosed with ASD.

Given these findings, many students clinically diagnosed with ASD receive special education services under a different educational classification than Autism (CDC 2014; McDonald et al. 2019; Rubenstein et al. 2018; Sansosti 2010; Toomey et al. 2009). These other special education classifications encompass such options as Other Health Impaired (OHI), Speech and Language Impairment (SLI), Emotional Disturbance (ED), Specific Learning Disability (SLD), and Multiply Disabled (MD; CDC 2014; Toomey et al. 2009). McDonald et al. (2019) conducted a survey of special education classifications of children with ASD without ID and found that the majority of children with ASD without ID were classified under Autism (56%), followed by OHI (27%) as the second largest category of classification. This is in contrast to the findings of Toomey et al. (2009), who found that children with ASD without ID were most frequently classified under OHI, while only 17.6% were classified under the Autism category. While the percentage of children classified under Autism for special education services may vary, the distribution of children diagnosed with ASD among variable special

education classifications continues to indicate that the unique features and needs of students should be examined when designing specific academic, social, and behavioral interventions. This is in contrast to school personnel relying on the specific special education category to guide intervention decision-making (Toomey et al. 2009).

Current Knowledge

Given the number of children with ASD accessing special educational services, more information is needed regarding how they are accessing these services. Zuckerman et al. (2017) assessed service use as well as documenting diagnostic age and delay among 722 elementary-school children with ASD, using the CDC's Survey of Pathways to Diagnosis and Services data. Of this sample, 63% of the children with ASD demonstrated a functional delay. Zuckerman et al. (2017) found that children who were diagnosed later (after 4 years of age) were less likely (72%) to access school-based services than peers who had been diagnosed before the age of 4 (90%). Children were diagnosed later were also more likely to take psychotropic medications (Zuckerman et al. 2017). Further, as time-to-diagnosis increased for those children with ASD with functional delays, Zuckerman et al. (2017) found that these children were less likely to receive school-based therapies. Finally, Zuckerman et al. (2017) expressed concern that nearly a quarter of elementary school-aged children with ASD did not receive school-based services.

Of those who have been accessing special education services in the schools, there has been a recent trend of placing students with ASD in inclusive classrooms in the general education setting (Lauderdale-Littin et al. 2013; Martins et al. 2014). For example, the United States Department of Education (USDOE 2012) has reported that between 2004 and 2010, the number of students with ASD attending public schools has doubled. In addition, there was a 244% increase of students with ASD in full-inclusion classrooms from 1992 to 2006 (USDOE 2010). Researchers have found that students with higher cognitive and social

abilities were more apt to be placed in inclusive settings (Lyons et al. 2011; White et al. 2007). Conversely, students with ASD with lower IQs, lower adaptive behavior skills, and higher ASD symptoms were likely to be placed in more restrictive special education settings (Eaves and Ho 1997; White et al. 2007).

Supporting the trend toward inclusion, recent studies have reported a positive impact on the level of social engagement of students with ASD in the inclusion setting (Sansosti and Sansosti 2012). For example, studies examining subgroups of children with ASD without ID have demonstrated that they engage in increased social interaction in the mainstream classroom (Dahle 2003), have larger networks of friends, or are included in peer activities at the same rate as their same-age peers without disabilities (Chamberlain et al. 2007). Others note that an inclusive placement alone may be insufficient to fulfill the social and academic needs of children with ASD (Leyser and Kirk 2004; Sansosti 2010). In addition, special education placement only describes a minute portion of special education service use. Therefore, further examination of services used within the parameters of special education is warranted.

In a study of 101 children with ASD without ID, White et al. (2007) examined relationships between child demographic factors and special education, and found that children with ASD without ID with lower cognitive and language abilities were more likely to receive special education services. In general, service use was highest in the third and fourth grades, and lowest in eighth grade. Further, White et al. (2007) found that the children with ASD's level of social ability was not related to special education service use. This is concerning, given the deleterious effect of children with ASD's social deficits on their performance in the classroom setting (Thomeer et al. 2017), especially with the reported overarching suppression of social ability among children with ASD (White et al. 2007). Finally, the majority of students with ASD in special education received speech therapy, followed by physical and/or occupational therapies (White et al. 2007). Social skills instruction was much less frequently reported for children in the younger

elementary grades, with none of the parents of older children (grades 7 and 8) reporting that their children received this service. Given the potential benefits of social skills instruction in the schools (Thomeer et al. 2017), services that would directly ameliorate social deficits continue to be an area of unmet need (White et al. 2007).

Wei et al. (2014) examined three national datasets of functionally heterogeneous students with ASD for patterns of special education services use. Wei et al. (2014) found that students with ASD and higher levels of ASD symptoms were more likely to receive more special education services, and that service use generally declined with increasing age. In addition, they noted how educational priorities may shift as a student ages, with age moderating how the students experience special education services and supports. For elementary school students, the two most common therapies were speech and language therapy (84.6%), followed by occupational therapy (50.0%). In contrast, a relatively lower percentage of students received a behavior management program (43.8%), service coordination (39.5%), adaptive physical education (39.1%), learning strategies/study skills support (29.5%), or social work services (6.4%). Secondary school children with ASD also followed a similar pattern, with speech services being the most frequently accessed service for children with ASD in special education.

While these service usage results reported by Wei et al. (2014) are consistent with prior research (White et al. 2007), the authors also noted the discrepancy between the widespread social and behavioral impairments experienced by students with ASD and lack of social/behavioral services delivered in schools. Therefore, this represents an area of unmet need in the special education system (Wei et al. 2014; White et al. 2007). In addition, the low percentage of elementary school children with ASD accessing learning supports reported by Wei et al. (2014) further confirmed the assertion that these services are underemphasized in the IEPs developed for students with ASD (White et al. 2007). Finally, Wei et al. (2014) found that 3.4% of elementary/middle school and 6.7% of high school children with ASD did not receive

any of the three services (i.e., speech and language therapy, occupational therapy, behavior management program) thought to directly address ASD symptoms.

McDonald et al. (2019) examined the service use of a sample of 89 children (ages 6–11 years) diagnosed with ASD without ID. The children's IEPs were evaluated for special education classification, type of service received (e.g., speech therapy, occupational therapy, physical therapy, individualized behavior support plan, social skills instruction, counseling, and 1:1 aide), duration of service, and type of placement (i.e., full inclusion, partial inclusion, and self-contained classroom). For the purpose of their study, McDonald et al. (2019) defined full inclusion as the placement for a child who remained in the mainstream, general education setting for the entirety of the school day. Partial inclusion was defined as the placement for children who attended general education for the majority of their school day, but less than 100% of all subjects were taught in the general education setting. Students in this placement typically were removed from the general education setting briefly for more intensive instruction in a specific subject area, such as math and/or reading. Finally, a self-contained classroom was defined as the placement for children who primarily remained in this more restrictive special education classroom (with a smaller teacher-to-student ratio) and only were included into the general education setting for a maximum of two academic classes. McDonald et al. (2019) reported that partial inclusion was the most frequent placement (40%), followed by full inclusion (34%) and self-contained classroom settings (26%). Over half of the participants ($n = 50$, 56%) were classified under Autism, followed by OHI (27%). In addition, the researchers found that children classified under Autism were about twice as likely to receive 1:1 aide services as children with ASD who met other special education classifications. In regards to the whole sample, the most frequently provided services included speech therapy (71%) and occupational therapy (56%) in line with prior research (Wei et al. 2014; White et al. 2007). However, unlike past research, counseling services were the

third reported most frequent service (44%). This is noteworthy, as evidence for the use of cognitive-behavioral strategies with children with ASD without ID is increasing (Ho et al. 2018), although counseling has not been widely examined in the special education service use literature. In contrast with these top three services accessed, social skills instruction and behavior support plans were the least common (17% and 16%, respectively) services rendered.

Although not specifically examining counseling, in a rare study looking at mental health services in the schools, Narendorf et al. (2011) examined a sample of adolescents with ASD ($n = 920$), drawn from the National Longitudinal Transition Study-2 (NLTS2) who qualified for special education services via an educational classification of Autism. Of these youth, 49% of the sample reported using a mental health service in the past, with 47% of these youth accessing mental health services through the special education system in schools. Factors that increased the odds of accessing these services via the school system included being African American, or having a household income below \$50,000 (in comparison to families having a household income greater than \$70,000; Narendorf et al. 2011). Parent involvement in the IEP process, as well as child characteristics of displaying poor social skills, experiencing bullying, or perpetrating bullying were associated with accessing mental health services, but not associated with accessing these services in schools.

Future Directions

Research in the area of special education service use by children with ASD and its relationships with child and placement variables continues to be limited (McDonald et al. 2019; Wei et al. 2014; White et al. 2007). Further research is needed that examines what special education services children with ASD receive, as well as the amounts and types of special education services accessed and their relationship to the children's demographic variables (e.g., age, IQ, level of ASD symptoms,

communication ability, and other adaptive behaviors; Lauderdale-Littin et al. 2013; McDonald et al. 2019; White et al. 2007). Documenting special education service use by children with ASD is important as it may inform the training needed for school personnel who work with children with ASD (McDonald et al. 2019; Wei et al. 2014). In addition, the documentation of special education service use may be a resource used to inform families as to what special education services a child with ASD typically receives (or may need), and what they might be inclined to advocate for (McDonald et al. 2019; Wei et al. 2014). For example, Thomas et al. (2007) found that parents of children with ASD report that not only is speech therapy one of the most widely-used services in special education, but it is considered by many parents as one of the best services (followed by occupational therapy) their child receives in the school. While speech services are often necessary (especially given the documentation of lower communication skills in this population), it is essential to understand the child demographic and behavioral characteristics associated with special education placement and service use decisions (Lauderdale-Littin et al. 2013) and inform families, school personnel, and other stakeholders of these relationships. Given the disparities between level of social/behavior services provided in schools and the social/behavioral needs of children with ASD highlighted by multiple researchers (McDonald et al. 2019; White et al. 2007; Wei et al. 2014), families and school staff of students with ASD should advocate for appropriate social and behavioral interventions in schools. In addition, further examination of how children with ASD are or are not succeeding socially in the various special education placements is warranted (White et al. 2007). This research should also focus its investigation on the potential existence of a social benefit purported to be received from placement in the general education setting (White et al. 2007).

Future research also needs to focus on understanding the barriers to accessing special education services throughout students with ASD's school tenure (Wei et al. 2014). Most importantly,

more examination of the reasons behind the decline of speech and occupational therapies and behavioral services needs to be conducted, as this also indicates that accessing special education services may become more difficult as children with ASD age (Wei et al. 2014). In addition, barriers to minorities accessing special education services needs to be examined, as research has mixed results on the success these subgroups have had in receipt of services, which is typically below their Caucasian peers (Wei et al. 2014), aside from the propensity to access mental health care in the school setting at higher rates (Narendorf et al. 2011). Finally, further examination of the implementation of counseling and other mental health services in the schools should be studied, in order to gain more information as to the implementation of and access to these services across the various age ranges.

See Also

- ▶ [School-Clinic Care Coordination for Youth with ASD](#)
- ▶ [Service Use](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association.
- Ashburnere, J., Ziviani, J., & Rodger, S. (2010). Surviving in the mainstream: Capacity of children with autism spectrum disorders to perform academically and regulate their emotions and behavior at school. *Research in Autism Spectrum Disorders* 4(1), 18–27
- Center for Disease Control and Prevention (CDC). (2014). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 states, United States, 2010. *MMWR*, 63(SS-2), 1–21.
- Center for Disease Control and Prevention (CDC). (2016). Prevalence and characteristics of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 states, United States, 2012. *MMWR*, 65(SS-3), 1–23.
- Chamberlain, B., Kasari, C., & Rotherman-Fuller, E. (2007). Involvement or isolation? The social networks of children with autism in regular classrooms.

- Journal of Autism and Developmental Disorders*, 37, 230–242. <https://doi.org/10.1007/s10803-006-0164-4>.
- Dahle, K. B. (2003). Services to include young children with autism in the general classroom. *Early Childhood Education Journal*, 31, 65–70. <https://doi.org/10.1023/A:1025193020415>.
- Eaves, L. C., & Ho, H. H. (1997). School placement and academic achievement in children with autism spectrum disorder. *Journal of Developmental and Physical Disabilities*, 9, 277–291. <https://doi.org/10.1023/A:1024944226971>.
- Ho, B. P. V., Stephenson, J., & Carter, M. (2018). Cognitive-behavioral approaches for children with autism spectrum disorder: A trend analysis. *Research in Autism Spectrum Disorders*, 45, 27–41. <https://doi.org/10.1016/j.rasd.2017.10.003>.
- Kang-Yi, C. D., Locke, J., Marcus, S. C., Hadley, T. R., & Mandell, D. S. (2016). School-based behavioral health service use and expenditures for children with autism and children with other disorders. *Psychiatric Services*, 67(1), 101–106. <https://doi.org/10.1176/appi.ps.201400505>.
- Lauderdale-Littin, S., Howell, E., & Blacher, J. (2013). Educational placement for children with autism spectrum disorders in public and nonpublic school settings; The impact of social skills and behavior problems. *Education and Training in Autism and Developmental Disabilities*, 48(4), 469–478. Retrieved from: [http://daddceec.org/Portals/0/CEC/Autism_Disabilities/Research/Publications/Education_Training_Development_Disabilities/Full_Journals/ETADD_48\(4\)_469-478.pdf](http://daddceec.org/Portals/0/CEC/Autism_Disabilities/Research/Publications/Education_Training_Development_Disabilities/Full_Journals/ETADD_48(4)_469-478.pdf)
- Leyser, Y., & Kirk, R. (2004). Evaluating inclusion: An examination of parental views and factors influencing their perspectives. *International Journal of Disability, Development, and Education*, 51, 271–285. <https://doi.org/10.1080/1034912042000259233>.
- Lyons, J., Cappadocia, M. C., & Weiss, J. A. (2011). Social characteristics of students with autism spectrum disorders across classroom settings. *Journal on Developmental Disorders*, 17, 77–82. Retrieved from: <http://hdl.handle.net/10315/33012>
- Martins, M., Harris, S. L., & Handelman, J. S. (2014). Support inclusive education. In F. R. Volkmar, S. J. Rogers, R. Paul, & K. A. Pelfrey (Eds.), *Handbook of autism and pervasive developmental disorders. Vol. 2: Assessment, interventions, and policy* (4th ed., pp. 858–870). New York: Wiley.
- McDonald, C. A., Donnelly, J. P., Feldman-Alguire, A. L., Rodgers, J. D., Lopata, C., & Thomeer, M. L. (2019). Special education service use by children with autism spectrum disorder. *Journal of Autism and Developmental Disabilities*, 49, 2437–2446. <https://doi.org/10.1007/s10803-019-03997-z>.
- Narendorf, S. C., Shattuck, P. T., & Sterzing, P. R. (2011). Mental health service use among adolescents with an autism spectrum disorder. *Psychiatric Services*, 62(8), 975–978. <https://doi.org/10.1176/appi.ps.62.8.975>.
- Rubenstein, E., Daniels, J., Schieve, L. A., Christensen, D. L., Van Naarden Braun, K., Rice, C. E., Bakian, A. V., Durkin, M. S., Rosenberg, S. A., Kirby, R. S., & Li-Ching, L. (2018). Trends in special education eligibility among children with autism spectrum disorder, 2002–2010. *Public Health Reports*, 133(1), 85–92. <https://doi.org/10.1177/0033354917739582>.
- Sansosti, J. M., & Sansosti, F. J. (2012). Inclusion for students with high-functioning autism spectrum disorders: Definitions and decision making. *Psychology in the Schools*, 49(10), 917–931. <https://doi.org/10.1002/pits>.
- Sansosti, F. J. (2010). Teaching social skills to children with autism spectrum disorders using tiers of support: A guide for school-based practitioners. *Psychology in the Schools*, 47(3), 257–281. <https://doi.org/10.1002/pits.20469>.
- Thomas, K. C., Morrissey, J. P., & McLaurin, C. (2007). Use of autism-related services by families and children. *Journal of Autism and Developmental Disorders*, 37, 818–829. <https://doi.org/10.1007/s10803-006-0208-9>.
- Thomeer, M. L., McDonald, C. A., Rodgers, J. D., & Lopata, C. (2017). High-functioning autism spectrum disorder: A framework for evidence-based practice. *School Mental Health*. <https://doi.org/10.1007/s12310-017-9236-1>.
- Toomey, J. A., Lopata, C., Volker, M. A., & Thomeer, M. L. (2009). Comprehensive intervention for high-functioning autism spectrum disorders: An in-depth case study. In M. T. Burton (Ed.), *Special education in the 21st century* (pp. 95–118). Hauppauge: Nova Science.
- United States Department of Education. (2010). *32nd Annual report to Congress on the implementation of the individuals with Disabilities Education Act*. Retrieved from <https://www2.ed.gov/about/reports/annual/osep/2010/parts-b-c/32nd-idea-arc.pdf>
- United States Department of Education. (2017). *39th Annual report to Congress on the implementation of the individuals with Disabilities Education Act*. Retrieved from <https://www2.ed.gov/about/reports/annual/osep/2017/parts-b-c/39th-arc-for-idea.pdf>
- United States Department of Education, National Center for Education Statistics. (2012). *Digest of Education Statistics, 2011* (NCES 2012-001). Chapter 2.
- Wei, W., Wagner, M., Christiano, E. R. A., Shattuck, P., & Yu, J. W. (2014). Special education services received by students with autism spectrum disorders from preschool through high school. *The Journal of Special Education*, 48(3), 167–179. <https://doi.org/10.1177/0022466913483576>.
- White, S. W., Scahill, L., Klin, A., Koenig, K., & Volkmar, F. R. (2007). Educational placements and service use patterns of individuals with autism spectrum disorders. *Journal of Autism and Developmental Disabilities*, 37, 1403–1412. <https://doi.org/10.1007/s10803-006-0281-0>.
- Yeargin-Allsop, M., Rice, C., Karapurkar, T., Doernberg, N., Boyle, C., & Murphy, C. (2003). Prevalence of autism in a U.S. metropolitan area. *Journal of the*

American Medical Association, 289, 49–55. <https://doi.org/10.1001/jama.289.1.49>.

Zuckerman, K., Lindly, O. J., & Chavez, A. E. (2017). Timeliness of autism spectrum disorder diagnosis and use of services among U. S. Elementary school-aged children. *Psychiatric Services*, 68(1), 33–40. <https://doi.org/10.1176/appi.ps.201500549>.

Special Needs

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Synonyms

[Accommodations](#); [Modifications](#); [Special education needs](#)

Definition

Special needs is a term often used to describe the needs of individuals with disabilities or who are at risk of developing disabilities and their unique needs as a result of deficits related to a disability or disorder, who require or may require in the future special services or treatment. Needs typically refer to special care, services, or accommodations necessary to support the individual or those supporting the individual with a disability or disorder. Special needs most often refer to intervention, curricula, behavior management, and transition planning; however, individual accommodations and modifications related to deficit of the disability are addressed under this term.

See Also

- [Accommodations](#)
- [Modifications](#)
- [Transition Planning](#)

References and Reading

- Klin, A. (2006). Autism and Asperger's syndrome: An overview. Retrieved 15 Jan 2011 from http://www.scielo.br/scielo.php?script=sci_arttext&pid=S1516-44462006000500002&lng=en&nrm=iso&tlng=en
- National Academy of Sciences and Committee on Educational Interventions for Children with Autism. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Volkmar, F. R., & Wiesner, L. A. (2009). *A practical guide to autism: What every parent, family member, and teacher needs to know*. Hoboken: Wiley.
- Volkmar, F. R., Paul, R., Klin, A., & Cohen, D. (2005). *The handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.

Special Needs Planning (SNP)

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Definition

Special needs planning (SNP) is a process of collecting, coordinating, and consolidating legal, financial, health, and personal wellness information concerning an individual with disabilities to ensure that the individual receives support in the future. SNP activities can help to improve an individual's quality of life and provide resources for goods and services that are not covered under government benefits programs or private health insurance plans. The overarching goal of SNP activities is to create a well-informed plan for an individual's future care and resources.

Principles of Special Needs Planning

SNP activities can assist individuals who have autism spectrum disorder (ASD) in making financial resources available to pay for current and future needs that are not covered by government

Special Needs Planning (SNP), Table 1 Examples of SNP benefits and instruments

Potential benefit	Examples of instruments that provide assistance
Ensuring funds are available to a beneficiary without jeopardizing eligibility for government benefits	Special needs trust (SNT) and Achieving a Better Life Experience account (ABLE or 529A account)
Ensuring assigned individuals available to assist the beneficiary as needed	Decision-making support agreement, guardianship, conservatorship, letter of intent (LOI), Power of Attorney for money and property, and Power of Attorney for healthcare
Ensuring that funds are well managed to meet the beneficiary's needs	Legal or financial agent agreement, decision-making support agreement, guardianship, and conservatorship
Document preferences for future transition-of-care planning to prepare for a time when a beneficiary's current caregivers can no longer provide care	Letter of intent (LOI), Power of Attorney for healthcare, and advance directive

benefits or private health insurance (Marsack-Topolewski and Church 2019; Thomas et al. 2016; Parish et al. 2015; Horlin et al. 2014; and Montes and Halterman 2008; see also Krakovich et al. (2016) and Cidav et al. 2012). The person who receives the benefit of SNP activities is often referred to as the “beneficiary.” SNP has the potential to offer multiple forms of assistance and security to a beneficiary. See Table 1, Examples of SNP benefits and instruments. The individual beneficiary should be involved in the planning process to the greatest extent possible (National Council on Disability 2018). Other individuals involved in the SNP process often include family members, friends, and legal guardians or conservators (Trentacosta et al. 2018).

There are three fundamental phases of SNP activities: (A) Identifying costs for essential needs; (B) Identifying costs for preferred goods and services; and (C) strategizing with guidance from legal and finance professionals. See Table 2, Twelve key steps in the SNP process. The initial steps involve confirming information about the beneficiary. Information collection for the SNP process is often conducted by a close caregiver of the beneficiary. First, the caregivers must document which short and long-term expenses are absolutely essential, and which are merely desirable. The time spent collecting detailed information about the beneficiary's desires and needs will allow legal and financial planners to offer personalized guidance for SNP strategies (Russell and Grant 2010). With clearly organized information about an individual with disabilities' unique

circumstances, professional advisors are better able to provide well-informed SNP guidance.

To gather the necessary information for SNP, individuals with disabilities and/or their caregivers should outline short- and long-term goals for the beneficiary's living, healthcare, wellness, educational, vocational, and emergency needs (Tencza and Forsythe 2019; Lauderdale and Huston 2012). As a next step, the beneficiary or caregiver must create a list of all of the goods and services that are necessary to meet the beneficiary's goals. Each item on the special needs planning list should correlate to an “out of pocket” cost – a good/service that is not covered under the beneficiary's government benefits programs and/or private health insurance. Individuals who are working to collect details to inform a special needs plan must consider that SNP information comes from a variety of sources that include many professional disciplines. One of the preferred means to create a thorough, well-informed summary of the beneficiary's needs is to seek information about the beneficiary's needs from multi-disciplinary groups of experts, particularly those in health and human services fields. See Table 3, Health and human services professionals who may assist during the SNP information collection phase.

Many technical SNP services are performed by an experienced attorney, financial planner, and/or tax advisor (see generally, American Bar Association Diversity and Inclusion Center Commission on Disability Rights Resources 2019). Though many for-profit and non-profit businesses offer

Special Needs Planning (SNP), Table 2 Twelve key steps in the SNP process

A. Identify costs for essential needs not covered by government programs and private health insurance	
Step 1	What are the beneficiary’s short- and long-term goals in the following areas: healthcare, personal wellness, education, and vocation?
Step 2	Which essential goods or services are required for the beneficiary to satisfy the goals identified in Step 1?
Step 3	Which of the goods or services identified in Step 2 are not covered by government benefits programs and/or private health insurance?
Step 4	What are the out-of-pocket costs for goods or services identified in Step 3 (per item/session, monthly, and annually)?
Step 5	Of the goods and services identified in Step 4, which are needed for a limited amount of time and which are permanently needed? Can any of the beneficiary’s healthcare providers provide additional information to help answer this question?
B. Identify costs for preferred goods and services not covered by government programs and private health insurance	
Step 6	Considering the goals identified in Step 1, which goods or services are desirable/preferred for the beneficiary?
Step 7	Which of the goods or services identified in Step 6 are not covered by government benefits programs and/or private health insurance?
Step 8	What is the out-of-pocket cost for goods or services identified in Step 7 (per item/session, monthly, and annually)?
Step 9	Of the goods and services identified in Step 7, which are needed for a limited amount of time and which are permanently needed? Can any of the beneficiary’s healthcare providers provide additional information to help answer this question?
C. Strategize with guidance from legal and finance professionals	
Step 10	Which attorney can best assist in writing and implementing the legal instruments for the special needs plan that is based on the information collected in in Steps 1–9? What is the attorney’s guidance on how to budget for emergency expenses in addition to the goods and services identified in Steps 1–9?
Step 11	Which financial planner can best assist in analyzing wealth management and tax reporting strategies to maximize the funds for the special needs plan?
Step 12	Review the special needs plan annually and update it to reflect any changes in the beneficiary’s needs and circumstances.

generalized SNP forms online, it is a strongly recommended best practice to retain an attorney to write and implement legal instruments that are tailored to the beneficiary’s unique needs. Legal instruments for SNP include, but are not limited to: special needs trusts (SNTs), Achieving a Better Life Experience accounts (ABLE or 529A accounts), Last Will and Testament documents, guardianships, conservatorships, letters of intent (LOIs), Power of Attorney for money and property; Power of Attorney for healthcare; money management services; and supported decision-maker agreements (Andersen and Gary 2018). Because a beneficiary’s preferences, health, and financial circumstances can change over time, it is a recommended best practice for caregivers to review a beneficiary’s special needs plan annually and to work with legal and financial advisors to make updates as needed.

In the United States, the SNP process should not be confused with the term “Medicare Special Needs Plans (“Medicare Advantage”), which are tailored health coverage plans that assist people with specific health conditions in accessing provider specialists, particularly drug formularies, and other unique benefits.

Common Barriers to Special Needs Planning

Over the past several years, multiple qualitative studies have found that a significant proportion of individuals with disabilities and their families are unready or unwilling to make future plans (Walker and Hutchinson 2018). Burke et al. (2018) summarize this concern as follows:

Special Needs Planning (SNP), Table 3 Health and human services professionals who may assist during the SNP information collection phase

Aging studies expert
Art therapist
Music therapist
Nurse practitioner, registered nurse, and/or home health nurse
Occupational therapy (OT) practitioner
Physiatrist
Physical therapy (PT) practitioner
Physician's assistant
Primary physician
Psychologist
Recreational therapist
Registered dietitian
Social worker
Special education expert
Speech pathologist

Special Needs Planning (SNP), Table 4 Commonly cited reasons why individuals do not engage in effective SNP

Lack of available services
Financial challenges
Reluctance of family members
Lack of time
The emotional nature of future planning
Inertia and/or disinterest in SNP issues
Lack of family members to be caregivers
Perceived lack of need due to the existence of current caregivers
Lack of awareness and understanding about SNP processes
Lack of confidence in available housing options
Lack of confidence in available options for retaining SNP professionals
Existence of mutually supportive relationships
Opinion that the beneficiary does not require any goods or services that are not covered by public benefits programs

Although many families want to engage in future planning, families often treat future planning as more aspirational than definitive. . .Parents often do not create actual plans; however, when parents create plans, their wishes for their offspring are more likely to be fulfilled.

Researchers report numerous barriers to future planning using SNP tools (Burke et al. 2018; and Bowey and McGlaughlin 2007). See Table 4, commonly cited reasons why individuals do not engage in effective SNP. Walker and Hutchinson (2019) have identified three overarching themes that arise in SNP scenarios: (1) external barriers to planning involving reservations about available services; (2) internal barriers within family and caregiver groups that prevent planning (e.g., mutual dependency and sense of helplessness); and (3) existence of diverse ideas and misconceptions about planning and ways of managing the future. Though a range of external and internal factors pose as barriers to SNP processes, a growing body of evidences suggests positive outcomes for individual beneficiaries and families once they engage in a future planning process (Brennan et al. 2020; Weiss et al. 2014; Heller and Kramer 2009).

See Also

- Achieving a Better Life Experience Savings Account (ABLE Savings Account, ABLE Account, 529A Savings Plan, and 529A Account)
- Conservatorship (Full Conservatorship and Limited Conservatorship)
- Guardianship
- Letter of Intent
- Power of Attorney (Financial and Property Management)
- Power of Attorney for Healthcare (Durable Healthcare Power of Attorney and Medical Power of Attorney)
- Social Security Amendments of 1965 (or “Medicare Act of 1965” and/or the “Medicaid Act of 1965”)
- Special Needs Trust (SNT)

References and Reading

American Bar Association Diversity and Inclusion Center Commission on Disability Rights Resources. (2019). *State Bars: Disability Entities Directory*. Retrieved from https://www.americanbar.org/groups/diversity/disabilityrights/resources/state_bar_disability_entities/

- Andersen, R., & Gary, S. (2018). *Understanding trusts and estates* (6th ed.). Durham: Academic.
- Bowey, L., & McGlaughlin, A. (2007). Older carers of adults with a learning disability confront the future: Issues and preferences in planning. *British Journal of Social Welfare*, 37(1), 39–54. <https://doi.org/10.1093/bjsw/bcl052>.
- Brennan, D., McCausland, D., O'Donovan, M. A., Eustace-Cook, J., McCallion, P., & McCarron, M. (2020). Approaches to and outcomes of future planning for family carers of adults with an intellectual disability: A systematic review. *Journal of Applied Research in Intellectual Disabilities*. <https://doi.org/10.1111/jar.12742>.
- Burke, M., Arnold, C., & Owen, A. (2018). Identifying the correlates and barriers of future planning among parents of individuals with intellectual and developmental disabilities. *Intellectual and Developmental Disabilities*, 56(2), 90–100. <https://doi.org/10.1352/1934-9556-56.2.90>.
- Cidav, Z., Marcus, S. C., & Mandell, D. S. (2012). Implications of childhood autism for parental employment and earnings. *Pediatrics*, 129(4), 617–623. <https://doi.org/10.1542/peds.2011-2700>.
- Heller, T., & Kramer, J. (2009). Involvement of adult siblings of persons with developmental disabilities in future planning. *Intellectual and Developmental Disabilities*, 47(3), 208–219. <https://doi.org/10.1352/1934-9556-47.3.208>.
- Horlin, C., Falkmer, M., Parsons, R., Albrecht, M. A., & Falkmer, T. (2014). The cost of autism spectrum disorders. *PLoS One*, 9(9), e106552. <https://doi.org/10.1371/journal.pone.0106552>.
- Krakovich, T. M., McGrew, J. H., Yu, Y., & Ruble, L. A. (2016). Stress in parents of children with autism spectrum disorder: An exploration of demands and resources. *Journal of Autism and Developmental Disorders*, 46(6), 2042–2053. <https://doi.org/10.1007/s10803-016-2728-2>.
- Lauderdale, M., & Huston, S. (2012). Financial therapy and planning for families with special needs children. *Journal of Financial Therapy*, 3(1), 62–81. <https://doi.org/10.4148/jft.v3i1.1494>.
- Marsack-Topolewski, C. N., & Church, H. L. (2019). Impact of caregiver burden on quality of life for parents of adult children with autism spectrum disorder. *American Journal on Intellectual and Developmental Disabilities*, 124(2), 145–156. <https://doi.org/10.1352/1944-7558-124.2.145>.
- Montes, G., & Halterman, J. S. (2008). Association of childhood autism spectrum disorders and loss of family income. *Pediatrics*, 121(4), e821–e826. <https://doi.org/10.1542/peds.2007-1594>.
- National Council on Disability. (2018). Beyond guardianship: Toward alternatives that promote greater self-determination. Retrieved from <https://ncd.gov/publications/2018/beyond-guardianship-toward-alternatives>
- Parish, S. L., Thomas, K. C., Williams, C. S., & Crossman, M. K. (2015). Autism and families' financial burden: The association with health insurance coverage. *American Journal on Intellectual and Developmental Disabilities*, 120(2), 166–175. <https://doi.org/10.1352/1944-7558-120.2.166>.
- Russell, L. M., & Grant, A. (2010). *Planning for the future: Give your child with a disability the gift of a safe and happy life* (7th ed.). Palatine, Illinois: Planning For The Future.
- Tencza, M., & Forsythe, L. (2019). Transition-of-care planning: Preparing for the future care of the individual with intellectual and developmental disabilities. *Journal of Intellectual Disabilities*. <https://doi.org/10.1177/1744629519883453>.
- Thomas, K. C., Williams, C. S., deJong, N., & Morrissey, J. P. (2016). Examination of parent insurance ratings, child expenditures, and financial burden among children with autism: A mismatch suggests new hypotheses to test. *Pediatrics*, 137(Supplement 2), S186–S195. <https://doi.org/10.1542/peds.2015-2851Q>.
- Trentacosta, C. J., Irwin, J. L., Crespo, L. M., & Beeghly, M. (2018). Financial hardship and parenting stress in families with young children with autism: Opportunities for preventive intervention. In *Handbook of parent-implemented interventions for very young children with autism* (pp. 79–91). Cham: Springer.
- Walker, R., & Hutchinson, C. (2018). Planning for the future among older parents of adult offspring with intellectual disability living at home and in the community: A systematic review of qualitative studies. *Journal of Intellectual & Developmental Disability*, 43(4), 453–462. <https://doi.org/10.3109/13668250.2017.1310823>.
- Walker, R., & Hutchinson, C. (2019). Care-giving dynamics and futures planning among ageing parents of adult offspring with intellectual disability. *Ageing and Society*, 39(7), 1512–1527. <https://doi.org/10.1017/S0144686X18000144>.
- Weiss, J. A., Wingsiong, A., & Lunsy, Y. (2014). Defining crisis in families of individuals with autism spectrum disorders. *Autism*, 18(8), 985–995. <https://doi.org/10.1177/1362361313508024>.

Special Needs Trust (SNT)

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Definition

A special needs trust (SNT) is a legal instrument established to hold money or property for the benefit of a person with disabilities. The recipient

of SNT assets is commonly referred to as a “beneficiary.” An SNT allows a benefactor, often called a “grantor,” to give money or property to a beneficiary without disrupting the beneficiary’s eligibility for government benefit programs (U.S. Social Security Administration 2020a). Assets that are held in an SNT account for a beneficiary are managed by a trustee. Generally, SNT holdings are not considered as a source of income to a beneficiary. Specific legal requirements for creating, administering, and funding an SNT are set forth in federal statutes, state statutes, and administrative regulations from federal and state agencies (e.g., the Social Security Administration (SSA), Centers for Medicare and Medicaid Services (CMS), and Internal Revenue Service (IRS)).

Principles of Special Needs Trusts

An SNT can be a useful special needs planning tool for individuals with autism spectrum disorder (ASD) and their caregivers to manage financial resources. When properly designed and administered in accordance with legal requirements, SNTs help people with disabilities to receive financial resources for their benefit while also qualifying to receive assistance from government programs like Medicare, Medicaid, and Social Security Incomes (SSI). For this reason, SNTs are sometimes referred to as “supplemental needs trusts.”

Individuals with disabilities cannot receive benefits through Medicaid and other government programs if they own significant financial resources that are not sheltered in an SNT or other special needs planning instrument. SNT legal doctrine considers state and federal government benefits as essential healthcare resources for individuals with disabilities. As a whole, SNT laws safeguard qualified individuals’ access to government benefits so that people can access comprehensive support networks and receive the care that they need. Many people with complex healthcare requirements rely on goods and services from government benefits to complete their activities of daily living (“ADLs” or basic self-care tasks) and instrumental activities of daily

living (“IADLs” or tasks to organize, manage, and interact with one’s environment). An SNT is sometimes employed in instances where an individual has an influx of financial resources and wishes to maintain government benefits (e.g., *Lewis v. Alexander*, 685 F.3d 325, 332 (3d Cir. 2012)). Eligibility for state and federal government program benefits is based on Modified Adjusted Gross Income (MAGI). Assets held in SNTs for a beneficiary do not typically count toward the government’s MAGI calculations (U.S. Centers for Medicare & Medicaid Services 2020a). Before the creation of SNT laws, individuals with disabilities were forced to forego financial gains that placed them above the legal threshold for personal income so that they could retain their access to government benefits.

Expenditures from an SNT are often used to pay for a range of goods and services for the beneficiary that are not covered by public benefits, including certain caregiving and personal wellness costs. Allowances from an SNT can help a beneficiary to pay for short- and long-term costs that enhance his or her quality of life (Jackins et al. 2019). As a general rule in most jurisdictions, SNT funds can only be used for the special and supplemental needs of a person with disabilities, and not their day-to-day living expenses such as food and shelter (e.g., New York Consolidated Laws Estates, Powers and Trusts Law § 7-1.12 (2020)). Because certain types of purchases cannot be made from SNT funds under state and federal laws, a trustee must ensure that all spending from an SNT satisfies all applicable legal requirements.

Individuals who are interested in using an SNT should take care not to confuse an SNT document with a Last Will and Testament, sometimes called a “legal Will” (Andersen and Gary 2018). Both are legal documents that can convey money and property to a loved one; however, they differ in several respects (Wick 2019). In certain cases, if a grantor leaves his or her assets directly to a beneficiary through a Will and without an SNT or other special needs planning tool, the Will’s bequest could render the beneficiary ineligible for government sponsored-benefits. Assets in a Will are governed by probate law processes. All distributions from a Will must be reviewed by a probate

court judge to confirm that the Will is valid and any bequests were not induced by fraud. Because they move through the court system, any asset distribution to a beneficiary in a Will becomes part of public record (Feder and Sitkoff 2016). As a general rule, a properly constructed SNT is not subject to the probate process and will not be deemed as income that could jeopardize the beneficiary’s eligibility for government benefits.

Creation of Special Needs Trusts

Three key parties are required to create an SNT: (1) grantor (the provider of trust assets and often parents, family members, or friends of the trust beneficiary), (2) trustee (the manager of trust assets), and (3) beneficiary (recipient of trust assets) (Table 1). The terms of an SNT are confirmed in a trust document, which constitutes a binding legal contract between a grantor and trustee (Wilcenski and Pleat 2020, April). As a

recommended best practice, an SNT document should always be written by a qualified attorney. Ideally, grantors and beneficiaries (including beneficiary guardians and conservators) should analyze specific legal, investment, and tax strategies in detail with both a tax advisor/financial planner and their special needs planning attorney (Lauderdale and Huston 2012).

Though several non-profit and for-profit organizations offer generalized forms for SNTs online, experts highly recommend obtaining personalized advice from experienced legal and financial professionals who can best tailor the SNT and other special needs planning tools to address particular needs and circumstances. As Hickman-Ladd (2019) explains,

Clients with special needs or a client’s family member with special needs can range from a child to an older adult. The planning will differ from client to client and depend on where each individual with special needs is in his or her life. Therefore, an attorney will consider different planning tools for each client.

Special Needs Trust (SNT), Table 1 Three key parties involved in an SNT

Party name	Description	Overview
Grantor	Provider of trust assets (money or property)	Grantors are often parents, family members, or friends of the trust beneficiary
		A grantor formally appoints a trustee for the SNT in a trust document
		By signing a trust document, grantors specify exactly how they would like the trustee to manage the SNT assets
		A signed trust document also specifies a grantor’s wishes regarding when the SNT assets should be distributed to the beneficiary – an SNT can be designed to take effect during the grantor’s lifetime or upon the grantor’s death
Trustee	Manager of assets	Trustees have a legal duty to manage the SNT for the benefit of the beneficiary
		A trustee must avoid conflicts of interest between his or her personal finances and the beneficiary’s finances
		Because a host of distributions from an SNT are not permitted by state or federal law, trustees must ensure that all SNT expenditures are in accordance with legal requirements
		A trustee can be a specific individual or an organization such as a bank or other financial institution
Beneficiary	Recipient of assets	A beneficiary can receive funds from an SNT without losing his or her eligibility for government-sponsored benefits
		Depending on the wording of the trust document, a beneficiary can receive payments from an SNT immediately after the trust is created or be entitled to funds sometime in the future (e.g., upon the death of the grantor, the beneficiary’s 21st birthday, or other specified date)

Because SNT laws are complex and situation specific, grantors and beneficiaries who engage in “Do-It-Yourself” SNT planning may render themselves liable for tax penalty fees for accidental noncompliance with federal and state requirements. By retaining professional assistance, SNT parties are more likely to avoid legal mistakes and see that their needs are comprehensively addressed. Moreover, professional guidance can help to ensure all short- and long-term goals for the beneficiary’s SNT finances account align with wills, guardianships/conservatorships, and other legal instruments that are specific to the beneficiary’s circumstances.

Types of Special Needs Trusts

There are varying legal requirements for grantors, beneficiaries, and trustees under federal and state laws. Technical legal requirements concerning SNTs include how assets must be executed, funded, managed, allocated after a beneficiary’s

death, and reported to the U.S. Internal Revenue Service (IRS) for tax and income purposes (U.S. Internal Revenue Service 2020). The particular rules that apply in a given situation depend largely on the type of SNT that is created by the grantor and trustee for the beneficiary (Russell and Grant 2010).

In the USA, there are three primary categories of SNT: self-funded trusts, third-party trusts, and pooled trusts (Table 2). Each type of SNT can be designed to hold assets without making the beneficiary ineligible for needs-based public benefits (U.S. Social Security Administration 2020b). SNT terminology sometimes varies among state jurisdictions and individual preferences. The terms “SNT” and “supplemental needs trusts” are often used interchangeably (Wilcenski and Pleat 2016). Nevertheless, individuals who are analyzing SNT issues should be aware that some state laws use the term “SNT” to refer only to self-funded and pooled trusts and define “supplemental needs trust” as solely third-party trusts (Table 3).

Special Needs Trust (SNT), Table 2 Three primary types of SNTs

Special needs trust type	Summary	Occurrences after a beneficiary’s death	Key federal statutes and social security administration regulations
Self-funded trust	SNT funded with the beneficiary’s own assets. Funds may include, but are not limited to: the beneficiary’s income from employment, inheritance, or a personal injury lawsuit settlement. This SNT can only be established for beneficiaries under age of 65	Any remaining assets may be subject to Medicaid Estate Recovery (payment collection by the government)	42 U.S.C. § 1396p(d)(4)(A); POMS SI 01120.200(B)(4)
Third-party trust	SNT established by a third-party and funded entirely with third-party assets. The beneficiary’s own assets cannot be used to fund this trust	Any remaining assets are freely transferable by the grantor (and not subject to Medicaid Estate Recovery)	42 U.S.C. § 1396p(d)(2)(A)(iii)–(iv); POMS SI 01120.200(B)(16)
Pooled trust	SNT created and administered by a nonprofit entity that creates individual trust for the beneficiary to use during his or her lifetime. A pooled trust contains the assets of several different individuals that are maintained in separate accounts for each beneficiary. Account funds are pooled together for investment purposes. This SNT can be established for an individual with disabilities any age	Depending on state law, some or all of the assets remaining in a pooled trust account may revert to the other remaining beneficiaries or be kept by the charity trustee. In cases of first-party pooled trusts, any remaining assets may be subject to Medicaid Estate Recovery. In cases of third-party pooled trusts, any remaining assets are freely transferable by the grantor	42 U.S.C. § 1396p(d)(4)(C); POMS SI 01120.200(B)(9)

Special Needs Trust (SNT), Table 3 Common terminology differences among SNT types

Self-funded trust	Pooled trust	Third-party trust
First-party trust	Pooled income trust	Supplementary trust
OBRA-93 trust, OBRA’ 93 trust, or OBRA trust	OBRA-93 trust, OBRA’ 93 trust or OBRA trust	Pooled supplemental needs trust
d4A trust	d4C trust	Third-party pooled trust
Grantor trust	First-party pooled trust	Self-funded payback pooled trust
Self-settled special needs trusts	Third-party pooled trust	
Self-funded payback trust	Pooled supplemental needs trust	
Medicaid payback trust	Pooled Medicaid payback trust	

As its name suggests, a self-funded SNT (also known as a “first-party SNT,” “OBRA-93 trust,” or “d4A trust”) is financed with a beneficiary’s own assets (42 U.S.C. § 1396p(d)(4)(A) (2020)). Funds for a self-funded SNT may include, but are not limited to, funds from an inheritance, retirement plan, divorce settlement, life insurance policy, or personal injury lawsuit settlement. Under certain conditions, any assets that remain in a self-funded SNT after a Medicaid enrollee has passed away may be used to reimburse the government for Medicaid benefits provided to the beneficiary during his or her lifetime. This process is often called “Medicaid Estate Recovery” (U.S. Centers for Medicare and Medicaid Services 2020b). Under federal law, states may not recover from the estate of a deceased Medicaid enrollee who is survived by a spouse, child under age 21, or a child of any age who is blind or has a serious disability (42 U.S. Code § 1396p (2020)).

In cases of pooled SNTs, a non-profit agency serves as the trustee and maintains a separate account for multiple individual beneficiaries (42 U.S.C. § 1396p(d)(4)(C) (2020)). This type of trust maintains one large account and individual beneficiaries have sub-trust accounts. With each individual trust account in one group, a single trustee or group of trustees can streamline investment and management tasks for the collective SNT pool. Weikel (2018) discusses the efficiencies of pooled trusts stating,

The benefit of a pooled trust is that the sub-trust accounts are pooled together for investment and management purposes, making the cost of

administration low (depending upon the choice of non-profit trustee). Furthermore, pooled trusts comprise professional trustees armed with more sophisticated knowledge of the complexities of SNT administration than the average family member of a disabled beneficiary serving as trustee.

Depending on which state law applies in a given situation, some or all of the assets remaining in the individual’s trust may revert to the other remaining beneficiaries or be kept by the charity trustee after one pooled trust beneficiary dies.

Third-party SNTs require a third-party donor to contribute the assets for the benefit of someone else (i.e., not for themselves) (42 U.S.C. § 1396p (d)(2)(A)(iii)–(iv) (2020)). Typically, if a balance remains in this trust upon the beneficiary’s death, the remaining assets are freely transferable by the grantor and not subject to estate recovery. Third-party SNTs can be included in the grantor’s Will. If an SNT is created through a Will, the trust will not activate and allow the beneficiary to access trust funds until after the grantor’s death. In cases where multiple donors wish to fund an SNT, it is preferable to create an SNT that is independent of a Will.

Key Considerations for Grantors When Creating a Special Needs Trust

During the process of creating a trust, grantors must consider seven primary issues: (1) Which assets (cash, stocks, bonds, real estate, and other property of value) will I put into the trust?;

(2) Who will receive the assets as the trust’s beneficiary?; (3) What is the name of the individual or organization that will manage the assets as the trustee? (Wilcenski and Pleat 2019, March); (4) What are the stipulations for this trust – i.e., should the trust funds be available immediately for the beneficiary or become eligible for withdrawal at a later date? (e.g., Sullivan 2010); (5) In the event I can no longer oversee the trustee’s actions because of a serious disability or injury, should my grantor responsibilities for this SNT refer to my named Power of Attorney?; (6) Can I structure this SNT to coordinate with the beneficiary’s Achieving a Better Life Experience (“ABLE” or “529A”) account?; and (7) Which special needs planning attorney and financial professional can best assist me in creating and implementing this SNT? (Table 4).

In many cases, an SNT is most advantageous for a beneficiary when combined with other special needs planning tools as part of a comprehensive long- and short-term budget plan (Rephan and Groshek 2016) (Table 5). For example, if an SNT beneficiary has an ABLE account, Hershey and Kelly (2019) recommend coordinating the use of relatively lower amounts in an ABLE account in tandem with greater savings in a third-party SNT. In this scenario, the ABLE account should be spent down entirely before the beneficiary’s death to avoid a loss of funds in the Medicaid Estate Recovery process. Funds remaining in the SNT can be freely transferred to another person or charity organization after the beneficiary’s passing (Hershey et al. 2015). All in all, a special needs planning attorney can help to ensure that the terms of a trust document are written correctly to meet the grantor’s goals.

Special Needs Trust (SNT), Table 4 Seven key considerations for grantors when creating an SNT

Issue	Key questions to consider
Type of trust	Which SNT type will best assist the beneficiary? What are the potential challenges and benefits associated with this type?
Content of the trust	Which assets will I put into the trust (e.g., cash, stocks, bonds, real estate, and other property of value)?
Trust funds recipient (beneficiary)	Who will receive the assets as the trust’s beneficiary? How will the SNT assets assist the beneficiary? How can I structure this SNT to best satisfy the beneficiary’s short- and long-term financial needs?
Trust funds manager (trustee)	What is the name of the individual or organization that will manage the assets as the trustee? Can the trustee objectively administer the trust in the best interest of the beneficiary and adhere to the terms of the trust contract?
Contract terms within the trust document (your ideas should be written by a special needs planning attorney using precise legal terms in the trust document)	What are the stipulations for this trust? Which type of SNT should I create? Should the trust funds be available immediately for the beneficiary or become eligible for withdrawal at a later date? How often should trust payments to the beneficiary occur – i.e., monthly installments, annual deposits to the beneficiary’s checking, or Achieving a Better Life Experience (“ABLE” or “529A”) account?
Future plans for the trust with grantor’s Power of Attorney	In the event I can no longer oversee the trustee’s actions because of a serious disability or injury, should my grantor responsibilities for this SNT refer to my named Power of Attorney? Is my Power of Attorney document up to date?
Hiring a special needs planning attorney and financial professional	Which special needs planning attorney and financial professional can best assist me in creating and implementing this SNT?

Special Needs Trust (SNT), Table 5 Comparing Achieving a Better Life Experience (ABLE) accounts and SNTs

Key considerations	Self-funded trust	Third-party trust	Pooled trust	ABLE account
What does the setup process include?	Attorney writes the trust document	Attorney writes the trust document	Grantor typically uses the non-profit organization's trust format, however, the language should also be reviewed by a special needs planning attorney	Each state offers online enrollment for beneficiaries (or their legal guardians) to create an account. State ABLE account programs encourage users to set up their own accounts, but also warn of the need for legal advice from an attorney and financial planner/tax advisor.
Is an attorney often used for the setup?	Yes, to ensure all trust terms are properly written and the grantor's wishes are satisfied	Yes, to ensure all trust terms are properly written and the grantor's wishes are satisfied	Yes, to ensure all trust terms are properly written and the grantor's wishes are satisfied	Though state ABLE account programs encourage do-it-yourself setup, personalized guidance from an attorney and financial planner/tax advisor is strongly recommended
If there is a balance remaining after beneficiary's death, are the assets subject to government payback (Medicaid estate recovery)?	Yes	No	Yes or no, depending on the structure of the trust and state law	Yes
Yearly fees	Yes, typically trustee and/or investment advisor fees	Yes, typically trustee and/or investment advisor fees	Yes, typically trustee and/or investment advisor fees	Yes, account maintenance fees (depending on the beneficiary's investment selections and the state ABLE program's administrative rules)
Yearly contribution limits	No	No	No	Yes, subject to the federal gift tax limit
Maximum contribution limits	No	No	No	Yes (the total amount varies between state ABLE program jurisdictions)

Legislative History of Special Needs Trusts

In 1993, the Omnibus Budget Reconciliation Act (OBRA) created many of the foundations

of current SNT law (Pub. L. No. 103–66 1993). OBRA confirmed that people with a disability to have a right to use an SNT to supplement their government benefits without jeopardizing their eligibility for means-tested

government benefits. Under the original wording of the OBRA law, a self-funded SNT could only be established by the SNT beneficiary's parent, grandparent, or guardian, or by a court.

In 2016, the 21st Century Cures Act amended federal law to expand regulations for creating self-funded SNTs (Pub. Law 114–255 2016). Under this law, individuals with a disability who have legal capacity can establish and make deposits into their own SNT savings accounts (excluding third-party SNTs). A beneficiary's legal capacity to create an SNT must be distinguished from his or her mental ability (Dudley and Edmonds 2018). Legal capacity embodies three pillars of individual legal rights: to have standing to bring and defend lawsuits; to enter into/execute legally binding contracts; and to appreciate the basic challenges and risks involved with an SNT (e.g., legal compliance and income tax reporting requirements). A legal milestone for disability rights, the Cures Act empowers individuals with disabilities who wish to save their own money using an SNT.

See Also

- [Achieving a Better Life Experience Savings Account \(ABLE Savings Account, ABLE Account, 529A Savings Plan, and 529A Account\)](#)
 - [Conservatorship \(Full Conservatorship and Limited Conservatorship\)](#)
 - [Guardianship](#)
 - [Power of Attorney \(Financial and Property Management\)](#)
 - [Power of Attorney for Healthcare \(Durable Healthcare Power of Attorney and Medical Power of Attorney\)](#)
 - [Social Security Amendments of 1965 \(or “Medicare Act of 1965” and/or the “Medicaid Act of 1965”\)](#)
 - [Special Needs Planning \(SNP\)](#)
- ## References and Reading
- 21st Century Cures Act. Pub. Law 114–255, 130 Stat. 1033. (2016). Codified at 42 U.S.C. § 201 et seq. Retrieved from <https://www.govinfo.gov/content/pkg/PLAW-114publ255/pdf/PLAW-114publ255.pdf>
- 42 U.S. Code § 1396p. (2020). Liens, adjustments and recoveries, and transfers of assets: Treatment of trust amounts. Retrieved from <https://www.govinfo.gov/content/pkg/USCODE-2010-title42/html/USCODE-2010-title42-chap7-subchapXIX-sec1396p.htm>
- Andersen, R., & Gary, S. (2018). *Understanding trusts and estates* (6th ed.). Durham: Carolina Academic Press.
- Dudley, K., & Edmonds, N. (2018). Psychology's role in guardianship and conservatorship evaluations. In S. S. Bush & A. L. Heck (Eds.), *Forensic geropsychology: Practice essentials* (pp. 257–279). Washington, DC: American Psychological Association. <https://doi.org/10.1037/0000082-013>.
- Feder, D. J., & Sitkoff, R. H. (2016). Revocable trusts and incapacity planning: More than just a will substitute. *Elder Law Journal*, 24, 1–48. http://www.law.harvard.edu/programs/olin_center/papers/pdf/Sitkoff_857.pdf
- Hershey, L., & Kelly, A. (2019). Using Roth conversions of legacy retirement plans to fund special needs planning. *Journal of Financial Service Professionals*, 73(2), 80–88. Retrieved from https://www.emich.edu/cob/documents/2019_hershey_kelly.pdf
- Hershey, L., Abbey, B., Stanaland, T. B., & Owens, T. L. (2015). When to stretch and when not to stretch an inherited IRA: The special case of the special needs trust. *Journal of Financial Service Professionals*, 69(2), 59–66. Retrieved from https://www.emich.edu/cob/documents/2015_when_to_stretch_and_when_not_to_stretch_an_inherited_ira.pdf
- Hickman-Ladd, C. (2019). Special needs planning: Some things to consider. *American Bar Association Voice of Experience*. Retrieved from https://www.americanbar.org/groups/senior_lawyers/publications/voice_of_experience/2019/october-2019/some-things-to-consider-in-special-needs-trust/
- Jackins, B., Blank, R. S., & Shulman, K. W. (2019). *Managing a special needs trust: A guide for trustees*. Exeter: disAbilities Books Press, LLC.
- Lauderdale, M., & Huston, S. (2012). Financial therapy and planning for families with special needs children. *Journal of Financial Therapy*, 3(1), 62–81. <https://doi.org/10.4148/jft.v3i1.1494>.
- Lewis v. Alexander, 685 F.3d 325, 332. (3d Cir. 2012). Retrieved from <https://caselaw.findlaw.com/us-3rd-circuit/1604090.html>
- New York Consolidated Laws Estates, Powers and Trusts Law § 7-1.12. (2020). Supplemental needs trusts established for persons with severe and chronic or persistent disabilities. Retrieved from <https://www.nysenate.gov/legislation/laws/EPT/7-1.12>

- Omnibus Budget Reconciliation Act of 1993 (OBRA), Pub. L. No. 103–66, 107 Stat. 312–685 Stat. 1025. (1993). Codified at 42 U.S.C. § 1396p. Retrieved from <https://www.govinfo.gov/content/pkg/BILLS-103hr2264enr/pdf/BILLS-103hr2264enr.pdf>
- Rephan, D. A., & Groshek, J. (2016). ABLE act accounts: Achieving a better life experience for individuals with disabilities with tax-preferred savings (and the old reliable special and supplemental needs trust). *Mitchell Hamline Law Review*, 42, 963. Retrieved from <https://open.mitchellhamline.edu/cgi/viewcontent.cgi?article=1034&context=mhlr>
- Russell, L. M., & Grant, A. (2010). *Planning for the future: Give your child with a disability the gift of a safe and happy life* (7th ed.). Palatine: Planning For The Future, Inc.
- Sullivan, P. (2010). Wealth matters: Exploring trusts for the disabled. *The New York Times*. Retrieved from <https://www.nytimes.com/2010/11/06/your-money/06wealth.html>
- U.S. Centers for Medicare & Medicaid Services. (2020a). How to count income & household members: What to include as income. Retrieved from <https://www.healthcare.gov/income-and-household-information/income/#magi>
- U.S. Centers for Medicare & Medicaid Services. (2020b). Estate recovery. <https://www.medicaid.gov/medicaid/eligibility/estate-recovery/index.html>
- U.S. Internal Revenue Service. (2020). U.S. Income Tax Return for Estates and Trusts: Instructions for form 1041 and schedules A, B, G, J, and K-1. Retrieved from <https://www.irs.gov/instructions/i1041#idm140366338350960>
- U.S. Social Security Administration. (2020a). Spotlight on trusts: 2020 Edition. Retrieved at <https://www.ssa.gov/ssi/spotlights/spot-trusts.htm>
- U.S. Social Security Administration. (2020b). SI 01120.203: Exceptions to counting trusts established on or after January 1, 2000. Program Operations Manual System (POMS). Retrieved from <https://secure.ssa.gov/poms.nsf/lnx/0501120203>
- Weikel, E. (2018). Disabling the funds of the disabled: How the sole benefit rule frustrates the administration and purpose of supplemental needs trusts. *Quinnipiac Probate Law Journal*, 32, 60.
- Wilcenski, E., & Pleat, T. (2016). Supplemental needs trusts: The basics. *New York State Bar Association Elder Law Section Program*. Retrieved from <https://nysba.org/NYSBA/Meetings%20Department/Section%20Meetings/Trusts%20and%20Estates/TRUSFA16%20Materials/Combined%20Self%20Settled%20Materials.pdf>
- Wilcenski, E., & Pleat, T. (2019, March). Administration of special needs trusts: Development of an improved approach (Part II). *New York State Bar Association Journal*, 91(2), 12–20. Retrieved from <https://nysba.org/app/uploads/2020/04/March-Journal-2019-WEB.pdf>
- Wilcenski, E., & Pleat, T. (2020, April). Administration of special needs trusts: Development of an improved approach (Part III). *New York State Bar Association Journal*, 92(3), 40–44. Retrieved from https://nysba.org/app/uploads/2020/04/NYSBA-April-2020-Journal_FinalWEBLinks.pdf
- Wick, D. W. (2019). The Expensive, Protective Estate Planning Strategy: A New Paradigm for Estate Planning Design and Administration. *Elder LJ*, 27, 115. Retrieved from: <https://theelderlawjournal.com/wp-content/uploads/2019/05/Wick.pdf>

Special Olympics

Timothy Shriver

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Major Areas or Mission Statement

With sports at the core, Special Olympics stands as a leader in advancing rights, opportunities, and social change for people with intellectual disabilities around the world. In 2011, Special Olympics served nearly four million athletes worldwide. While the majority of the athletes (64.8%) are school-aged (8–21 years old), 33.4% are adults (22+), and 1.8% are young children (2–7), with more males (62%) than females (38%) participating. Special Olympics has been growing rapidly; since 2000, the Special Olympics athlete count has grown from just under one million to the current total of just under million.

Most staff in Special Olympics programs globally are volunteers with a wide variety of expertise, including credentialed sports coaches, health professionals, program administrators, and researchers. In 2011, more than 306,000 coaches supported Special Olympics athletes. In addition, 546,000 partners, or participants without disabilities, are now engaged in Unified Sports, a Special Olympics initiative that includes individuals with and without intellectual disabilities together. Nearly 29,000 of our athletes serve in leadership

positions offered through another initiative, the Athlete Leadership Programs (ALPs).

Because of its inclusive nature, Special Olympics does not collect disability-specific information on its athletes, and, as such, there is no nationwide data on the number of participants in Special Olympics who have autism spectrum disorder (ASD). However, studies suggest that between 6% (Weiss et al. 2003) and 19% (Glidden et al. 2011) of Special Olympics athletes have ASD.

Mission Statement

"The mission of Special Olympics is to provide year-round sports training and athletic competition in a variety of Olympic-type sports for children and adults with intellectual disabilities, giving them continuing opportunities to develop physical fitness, demonstrate courage, experience joy and participate in a sharing of gifts, skills and friendship with their families, other Special Olympics athletes and the community."

Landmark Contributions

Brief History

In 1962, Eunice Kennedy Shriver started a summer day camp for children and adults with intellectual disabilities in her backyard in Maryland. She hoped to not only provide this underserved population with access to recreational opportunities but also to explore their capabilities in a variety of sports and physical activities. Just 6 years later, this small summer camp grew into the First International Special Olympics Summer Games, held at Soldier Field in Chicago, Illinois, USA. Over 1,000 individuals with intellectual disabilities came from 26 U.S. states and Canada to compete in track and field and swimming.

As the movement grew, so too did its national and international recognition as a serious sports movement. In 1971, the U.S. Olympic Committee gave Special Olympics official approval to use the name "Olympics" in the United States, making Special Olympics one of only two organizations authorized to do so. Fifteen years later, the United Nations proclaimed the International Year of

Special Olympics (1986) under the banner "Special Olympics – Uniting the World," and just 2 years later, the International Olympic Committee (IOC) officially endorsed and recognized Special Olympics (1988). The backyard camp had grown into an internationally recognized organization.

The subsequent decades were dedicated to expanding existing Special Olympics activities, as well as implementing new initiatives, such as Unified Sports, which creates an inclusive sports experience for participants with and without disabilities; Healthy Athletes, which provides free health screenings for athletes; and the Global Messenger Program, which offers public speaking and presentation skills training to athletes who are interested in representing the movement internationally. The organization also expanded to include cutting-edge research and evaluation, including the "Multinational Study of Attitudes toward Individuals with Intellectual Disabilities" (Siperstein et al. 2003), the most comprehensive study ever conducted on the subject. When Special Olympics celebrated its 40th anniversary in 2008, it had grown to serve three million athletes in 173 countries around the world. Today, the organization stands as an international leader in research and advocacy for persons with intellectual disabilities. In fact, in 2010, the first Special Olympics Global Congress was held in Marrakech, Morocco, bringing together hundreds of movement leaders from countries around the world to chart the next 5 years of work.

Special Olympics is currently led by:

- Timothy P. Shriver, Ph.D., Chairman and Chief Executive Officer
- J. Brady Lum, President and Chief Operating Officer

Major contributions:

1. Special Olympics has shown that people with disabilities can participate in and benefit from sports and recreation. The movement has demonstrated that the same motivation, desire, and ability to play and compete are present in people, irrespective of the presence of a disability.

2. Special Olympics has documented the developmental significance of sports and recreation across the lifespan for people with disabilities, from motor skill development in young children ages 2–7 to community engagement for adults.
 3. Special Olympics has led a movement of community inclusion by engaging millions of volunteers in meaningful experiences with people with intellectual disabilities, while also creating inclusive sports activities.
 4. Special Olympics has acted as a public education and attitude change vehicle, presenting people with intellectual and developmental disabilities in valued and respected social and community roles all around the world.
 5. Special Olympics has globalized the notion of sports and recreation for people with ID, particularly in developing countries.
 6. Special Olympics has demonstrated its ability to change attitudes – challenging and eliminating the stigma associated with intellectual or developmental disabilities – and instead highlighting the value of and skills possessed by people with ID, inspiring people to become advocates for inclusive education, employment, health care, and public policy.
 7. Special Olympics has pushed the boundaries of scholarly inquiry into the methods and models of personal and social change for people with intellectual disabilities.
 8. Special Olympics has launched a worldwide movement to combat health disparities and discrimination in health-care systems and the largest public health system has established Healthy Athletes, for people with ID in the world.
- Unified Sports: Unified Sports brings together equal numbers of Special Olympics athletes (with intellectual disabilities) and unified partners (peers without disabilities) on sports teams for organized, structured training and competition against other unified teams.
 - Healthy Athletes: Healthy Athletes offers free health screenings and health information to athletes and families at local, regional, and world games.
 - Young Athletes: Young Athletes is a curriculum designed to promote motor and social development for children ages 2½ to 7 through motor play activities and games.
 - Motor Activity Training Program: The Motor Activity Training Program is designed to provide individualized training programs for athletes with severe or profound intellectual disability who are unable to participate in official Special Olympics sports competitions because of their skill and/or functional capabilities.
 - Get Into It: The Get Into It educational resources explore the subjects of inclusion, acceptance, and social justice through the lens of core curriculum requirements and a service-learning framework.
 - Project UNIFY: Project UNIFY is a school-based initiative that uses sports and education programs to activate young people to develop school communities where all youth are agents of change – fostering respect, dignity, and advocacy for people with intellectual disabilities.
 - Athlete Leadership Programs (ALPs): The Athlete Leadership Program allows athletes to explore opportunities for greater participation in Special Olympics beyond sports training and competition as coaches, officials, team captains, spokespeople, and Board and committee members.
 - Research and Policy: Special Olympics conducts cutting-edge research and evaluation to better understand the many challenges faced by people with intellectual disabilities and the impact of Special Olympics on their lives. This research is also a driving force for realizing improved policies, laws, and rights for

Major Activities

Overview of Special Olympics programs:

- Sports Training and Competition: Special Olympics offers a variety of sports training and competitions for people with intellectual disabilities. In 2011, over 53,000 competitions were organized around the world, resulting in an average of 146 competitions every day.

people with intellectual disabilities around the world.

- **Special Olympics Family Support Network:** Family members of a person with intellectual disability often feel confused and alone. Special Olympics Family Support Network provides them acceptance, resources, hope, and a chance to become advocates – making them a valued voice in our movement.
- **Special Olympics Camp Shriver:** A camp experience for people with intellectual disabilities to learn new sports skills, participate in individual and team sports, and build friendships. The camp creates an atmosphere of understanding, learning, and sharing so that new friendships between athletes and participants without intellectual disabilities (partners) are created and continue to thrive once camp is over.

The Value of Special Olympics

There is growing empirical evidence demonstrating the value of Special Olympics for its participants, both with and without disabilities. Studies have shown that Special Olympics athletes gain not only improved sports skills but also improved physical fitness (Balic et al. 2000), increased self-esteem (Castagno 2001), increased self-perceptions of physical competence and social acceptance (Weiss et al. 2003), and improved social competence (Dykens and Cohen 1996). Indeed, athletes and families typically choose to participate in Special Olympics not only because of its sports opportunities but also because it is fun and offers prospects for social interaction (Harada and Siperstein 2008; Harada et al. 2011). There is also evidence that participation in Special Olympics decreases problem behaviors (Rosegard et al. 2001) and increases adaptive behaviors (Favazza and Siperstein *in press*; Favazza et al. *under review*). Further, typically developing individuals who participate in Unified Sports alongside teammates with intellectual disabilities have also demonstrated higher self-esteem, as well as greater willingness to interact with and more positive perceptions of individuals with disabilities (Castagno 2001). In addition, parents of Special

Olympics athletes frequently find that participation in Special Olympics raises their expectations of their son or daughter's abilities (Glidden et al. 2011; Kersh and Siperstein *under review*).

Although the benefits of participation in sports and recreation – and particularly in Special Olympics – have been well established for individuals with intellectual disabilities, only recently has the field begun to recognize the potential impact of sports and recreation on the social and motor development of individuals with autism spectrum disorder (ASD). Children with ASD frequently demonstrate motor impairments (Getchell and Lynch 2007), may be less physically active than their typically developing counterparts (Pan 2008), and are less involved in social and recreational activities with peers than those without disabilities (Solish et al. 2010). However, in their recent review of research on exercise and persons with ASD, Lang et al. (2010) found that persons with ASD participating in regular exercise experienced reductions in stereotypic, aggressive, and off-task behaviors. In addition, studies have suggested that participation in physical activity and recreational programs may have positive social benefits for individuals with autism, such as improved social behavior (Pan 2010) and increased social interaction (Garcia-Villamizar and Dattilo 2010), in addition to personal benefits, such as decreased stress and improved quality of life (Garcia-Villamizar & Dattilo). Of course, given the variability in specific deficits associated with ASD, some individuals may struggle with certain elements of team sports, including verbal and nonverbal communication with peers, loud noises, or personal contact (Crollick et al. 2006). Therefore, to maximize the benefits of sports and recreations, some adaptations – such as visual supports to present clear and predictable expectations (Fittipaldi-Wert and Mowling 2009), smooth transitions between activities (Crollick et al. 2006), and reinforcement of appropriate social and physical behaviors (Reid et al. 2003) – are often necessary.

The inclusive nature of Special Olympics requires that athletes not be singled out on the basis of their specific disabilities, and as a result,

Special Olympics does not have distribution numbers by specific intellectual/developmental disability subcategories. However, there is burgeoning preliminary evidence that individuals with ASD have participated in and enjoyed Unified Sports, inclusive summer camps (Special Olympics Camp Shriver), and traditional Special Olympics competitions. Additionally, families have spoken of a number of benefits of Special Olympics for their child with ASD, particularly in improved social skills and increased opportunities for social interaction (Kersh and Siperstein [under review](#)). Moving forward, Special Olympics will focus on how to best adapt its array of activities in order to accommodate its growing number of participants with ASD.

References and Reading

- Balic, M., Mateos, E., Blasco, C., & Fernhall, B. (2000). Physical fitness levels of physically active and sedentary adults with Down syndrome. *Adapted Physical Activity Quarterly*, 17(3), 310–321.
- Castagno, K. S. (2001). Special Olympics unified sports: Changes in male athletes during a basketball season. *Adapted Physical Activity Quarterly*, 18, 193–206.
- Crollick, J., Mancil, G., & Stopka, C. (2006). Physical activity for children with autism spectrum disorder. *Teaching Elementary Physical Education*, 17(2), 30–34.
- Dykens, E. M., & Cohen, D. J. (1996). Effects of special Olympics international on social competence in persons with mental retardation. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35(2), 223–229.
- Favazza, P. C. & Siperstein, G. N. (in press). Young athletes: A Special Olympics motor skill development program. *Journal for National Association of State Boards of Education*.
- Favazza, P. C., Siperstein, G. N., Zeisel, S., Odom, S. L., & Moskowitz, A. L. (under review). Young Athletes motor curriculum: Impact on motor development. *Adaptive Physical Activity Quarterly*.
- Fittipaldi-Wert, J., & Mowling, C. (2009). Using visual supports for students with autism in physical education. *JOPERD: The Journal of Physical Education, Recreation & Dance*, 80(2), 39–43.
- Frey, G. (2007). Physical activity and youth with developmental disabilities. In *Handbook of developmental disabilities* (pp. 349–365). New York: Guilford Press.
- Frey, G. C., Stanish, H. I., & Temple, V. A. (2008). Physical activity of youth with intellectual disability: Review and research agenda. *Adapted Physical Activity Quarterly*, 25(2), 95–117.
- Garcia-Villamizar, D., & Dattilo, J. (2010). Effects of a leisure programme on quality of life and stress of individuals with ASD. *Journal of Intellectual Disability Research*, 54(7), 611–619.
- Getchell, N., & Lynch, A. (2007). Motor competencies in children with Autism spectrum disorders. *Journal of Sport & Exercise Psychology*, 29, S6–S7.
- Glidden, L. M., Bamberger, K. T., Draheim, A. R., & Kersh, J. (2011). Parent and athlete perceptions of Special Olympics participation: Utility and danger of proxy responding. *Intellectual and Developmental Disabilities*, 49(1), 37–45.
- Harada, C. M., & Siperstein, G. N. (2008). The sport experience of athletes with intellectual disabilities: A national survey of Special Olympics athletes and their families. *Adapted Physical Activity Quarterly*, 26, 1–20.
- Harada, C. M., Siperstein, G. N., Parker, R., & Lenox, D. (2011). Promoting social inclusion for people with intellectual disabilities through sport: Special Olympics International, global sport initiatives, and strategies. In *Disability in the global sport arena: A sporting chance*. Routledge/Taylor & Francis.
- Kersh, J., & Siperstein, G. N. (under review). Mutual benefits of Special Olympics participation for individuals with intellectual and developmental disabilities and their parents. *Adaptive Physical Activity Quarterly*.
- Lang, R., Koegel, L., Ashbaugh, K., Regehr, A., Ence, W., & Smith, W. (2010). Physical exercise and individuals with autism spectrum disorders: A systematic review. *Research in Autism Spectrum Disorders*, 4(4), 565–576.
- McHale, J. P., Vinden, P. G., Bush, L., Richer, D., Shaw, D., & Smith, B. (2005). Patterns of personal and social adjustment among sport-involved and noninvolved urban middle-school children. *Sociology of Sport Journal*, 22(2), 119–136.
- Pan, C. (2008). Objectively measured physical activity between children with autism spectrum disorders and children without disabilities during inclusive recess settings in Taiwan. *Journal of Autism & Developmental Disorders*, 38(7), 1292–1301. <https://doi.org/10.1007/s10803-007-0518-6>.
- Pan, C. (2010). Effects of water exercise swimming program on aquatic skills and social behaviors in children with autism spectrum disorders. *Autism: The International Journal of Research and Practice*, 14(1), 9–28.
- Reid, G., O'Connor, J., & Lloyd, M. (2003). The autism spectrum disorders physical activity instruction: Part III. *Palaestra*, 19(2), 20–26, 47–48.
- Rosegard, E., Pegg, S., & Compton, D. M. (2001). Effect of Unified Sport on maladaptive behaviors among Special Olympics athletes. *World Leisure Journal*, 43(2), 39–48.
- Siperstein, G. N., Norins, J., Corbin, S., & Shriver, T. (2003). *Multinational study of attitudes toward*

individuals with intellectual disabilities: General findings and call to action. Washington, DC: Special Olympics and University of Massachusetts-Boston.

Solish, A., Perry, A., & Minnes, P. (2010). Participation of children with and without disabilities in social, recreational and leisure activities. *Journal of Applied Research in Intellectual Disabilities*, 23(3), 226–236. <https://doi.org/10.1111/j.1468-3148.2009.00525.x>.

Weiss, J., Diamond, T., Demark, J., & Lovald, B. (2003). Involvement in special Olympics and its relations to self-concept and actual competency in participants with developmental disabilities. *Research in Developmental Disabilities*, 24(4), 281–305.

Specific Language Impairment

- ▶ Childhood Aphasia
- ▶ Expressive Dysphasia
- ▶ Expressive Language Disorder

Specific Learning Disability

- ▶ Developmental Disabilities
- ▶ Learning Disability

Specific Learning Disorder

- ▶ Learning Disability

Specific Reading Disability

- ▶ Dyslexia

Spectrum/Continuum of Autism

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Definition

Autism is one of a spectrum of disorders that includes Asperger syndrome, pervasive developmental disorder-not otherwise specified (PDD-NOS), childhood disintegrative disorder (CDD), and Rett's disorder. These disorders are currently classified under the umbrella term of "pervasive developmental disorder (PDD)" in both the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association [APA] 1994) and the International Classification of Diseases (ICD-10; World Health Organization [WHO] 1992), but the term "autism spectrum disorder (ASD)" (also sometimes called autism spectrum condition (ASC)) is now more commonly used. Given the current state of knowledge, ASD appears to be a more useful description than PDD. It identifies autism as the reference point and suggests that at least some of the other disorders classified as PDD (i.e., atypical autism, PDD-NOS, Asperger syndrome) are differentiated from autism primarily by the age of onset, severity of symptoms, and presence of language delay and/or intellectual disability, whereas they are differentiated from non-spectrum disorders on the basis of several qualitative (and presumably biological) variables. Although CDD and Rett's disorder are also currently included in the PDD/ASD category, both of these disorders are exceedingly rare and differ significantly from other ASDs in the pattern of onset. In addition, given that there is now an identified genetic cause for the majority of cases of Rett's syndrome (Amir et al. 1999), it is unlikely that this disorder will be

included in the ASD category in DSM-V. Thus, the following discussion focuses on autism, PDD-NOS, and Asperger syndrome, referred to collectively as ASD.

Historical Background

Individuals with ASD present with diverse sets of symptoms and behaviors that vary as a function of many different factors, including age, language, IQ, and severity of social/communication symptoms and restricted and repetitive behaviors. The concept of a spectrum is therefore useful because it suggests that two individuals “on the spectrum” may look very different from one another even though they are classified under the same diagnostic umbrella. A child who avoids eye contact, flaps his hands, says only one or two words, and shows little interest in social interactions might share a diagnosis of ASD with a child who uses complex sentences and is socially motivated under certain circumstances but lacks the social awareness or skills to know how to behave in social situations or maintain successful peer relationships. Aided in part by advances in standardized diagnostic instruments and by opportunities to study larger and more diverse samples of individuals with ASD, there is increasing awareness that the spectrum of autism encompasses a wide range of difficulties and strengths and that no two individuals with ASD can be expected to look exactly the same.

Within the broader spectrum of autism, current diagnostic classification systems provide guidelines for assigning specific diagnoses (i.e., for determining whether a diagnosis of autism or Asperger syndrome or PDD-NOS is most appropriate). Unfortunately, these diagnoses tend to be highly unreliable across different sites and clinicians (Mahoney et al. 1998; Lord et al. 2012). Particularly with respect to Asperger syndrome, diagnostic conceptualizations vary widely within the field, making it difficult for professionals to agree on or be consistent in their use of terminology (Ozonoff et al. 2000). For example, whereas

some research groups adhere to the strict DSM-IV definition when diagnosing Asperger syndrome, which requires that autism be ruled out first, other groups diagnose Asperger syndrome even if the criteria for autism is also met (Szatmari et al. 2003). Similarly, although PDD-NOS is technically intended as a category to describe atypical autism (ICD-10) or “not quite autism,” it often ends up meaning “mild autism” or is used to classify young children to whom professionals do not yet feel comfortable giving an autism diagnosis (Walker et al. 2004). Disagreement about diagnostic criteria for differentiating within ASDs may help explain why, unlike earlier comparative studies, more recent investigations have not found clear differences between individuals identified as “high functioning autism (HFA)” and those diagnosed with Asperger syndrome. In one study, Bennett et al. (2008) found that adolescent outcomes in autism symptoms and adaptive behavior were better predicted by structural language impairment (defined by the presence of deficits in grammar or syntax) in childhood than by a clinical diagnosis of HFA vs. Asperger syndrome. Repetitive behaviors have also not been found to differ significantly between these diagnostic groups (Cuccaro et al. 2007).

Current Knowledge

From a clinical standpoint, the specific ASD diagnosis a child receives is arguably much less important than the fact that he/she falls somewhere on the autism spectrum. However, inconsistent use of labels often leaves parents confused about what to make of their child’s diagnosis. For example, Asperger syndrome has come to be portrayed in the media as a diagnosis characterized by special skills, high intelligence, and “quirky” personality traits, whereas autism is more commonly described as being associated with low IQ, limited language abilities, aloofness, and a poor prognosis. For parents of a child with ASD, their conceptualization of the disorder, including decisions about what interventions or

resources to seek out and expectations about the future, is likely to differ depending on whether the child receives a diagnosis of autism or another spectrum diagnosis that is perceived as being less severe. Moreover, if a child is initially given a diagnosis of PDD-NOS by one professional but then given a diagnosis of autism by another professional, the parent may erroneously assume that something about the child changed when in fact each professional simply had his/her own idiosyncratic way of deciding on one diagnosis over the other.

Disagreement about how diagnoses are assigned within the spectrum also has a number of important implications for ASD research. For example, if all “mild” cases of autism are referred to as Asperger syndrome or PDD-NOS, then a diagnosis of autism will be more likely to be related to intellectual disability. If Asperger syndrome is defined as any case of mild autism without intellectual disability, samples of children with PDD-NOS will be lower in IQ than if Asperger syndrome has a narrower definition (Klin et al. 2005).

From a neurobiological research perspective, clinical diagnostic designations are most useful if they are associated with different etiologies. But accumulating evidence suggests that these disorders are indeed on the same continuum and are therefore unlikely to be useful as a primary variable by which to stratify samples for etiological investigations. Analyses of datasets that include children with autism, PDD-NOS, and Asperger syndrome, as well as typically developing children and those with non-spectrum disorders, indicate that symptoms in the areas of communication, reciprocal social interaction, and restricted and repetitive behaviors constitute an “autism factor” that differentiates children with ASD *as a group* from children not on the spectrum (Constantino et al. 2004). Therefore, although the severity of symptoms in these domains differs among individual children with ASD, particular impairments in communication and reciprocal social interaction and the presence of restricted and repetitive behaviors set apart

individuals across the whole autism spectrum from those with other disorders (see also Lord and Bishop 2010).

Evidence that diagnostic distinctions within the spectrum may simply represent different degrees of the same disorder has also come from longitudinal follow-ups of children with ASD. Whereas movement “off” the spectrum is rare, it is not uncommon for children to change diagnoses within the spectrum depending on their symptom presentation at different points in development. A sizable proportion of children who initially receive a diagnosis of PDD-NOS in early preschool may go on to be diagnosed with autism in school age when their symptoms become more apparent and/or when the clinician becomes more certain about the diagnosis. Alternatively, some children diagnosed with autism may later receive a diagnosis of PDD-NOS when their symptoms improve (Lord et al. 2006; Stone et al. 1999). Such findings about changes in diagnostic status would be surprising if autism and PDD-NOS were indeed qualitatively distinct, but the concept of a spectrum allows for the idea that a child may move within the spectrum depending on their particular symptom profile while still continuing to meet the criteria for an ASD at each time point.

There is no question that the behavioral phenotypes of individuals with ASD are tremendously heterogeneous in general, or that an individual with a specific ASD designation of Asperger syndrome, for example, may look quite different than someone with a diagnosis of autism. However, research continues to indicate that whereas professionals (and diagnostic instruments) are reliable in indicating whether a child is somewhere on the spectrum or not on the spectrum, there are substantial inconsistencies in how specific ASD diagnoses are assigned by different practitioners (Lord et al. 2012). Therefore, it is likely that all specific ASD designations will be collapsed into a single ASD diagnosis in the forthcoming fifth revision of the DSM (see www.dsm5.org).

This proposed revision for DSM-V has caused significant controversy. Many professionals,

parents, and individuals with ASD feel that it is important to retain specific designations within the spectrum. Despite the issue of generally low levels of inter-rater reliability, it is argued that in some cases, diagnostic distinctions of autism vs. Asperger syndrome vs. PDD-NOS are useful or necessary in helping professionals communicate about different sorts of children or adults with ASD, or in helping families find resources that are appropriate. For example, parenting handbooks or social groups might be designed for individuals with “high functioning autism or Asperger syndrome” because the activities or suggestions are only relevant for those with fluent language. Similarly, a support group for parents of children with autism might be specifically intended to cater to families with children whose ASD symptoms are quite severe. Furthermore, some adults with ASD are more comfortable self-identifying as having Asperger syndrome (as opposed to autism or ASD) because they feel that this diagnosis more accurately reflects their profile of strengths and difficulties than the more classic definition of autism. Nevertheless, as they are currently defined, specific diagnoses within the spectrum have proven ineffective in reliably describing different groups of individuals with ASD.

On the other hand, the variability in language, cognitive functioning, and ASD symptoms and behaviors that exists among those diagnosed with ASD begs for *some* way of differentiating individuals with such discrepant behavioral profiles. Many authors have written about the need for measures of ASD severity. This information is necessary to facilitate communication about, and access to, appropriate services, as well as to measure the effectiveness of interventions and assess long-term outcomes. But the concept of ASD severity is still not well defined. Attempts have been made, through measures like the Social Responsiveness Scale (SRS; Constantino 2002), to develop more continuous measures of autism symptoms that can be used to approximate severity, but more research is needed to determine the extent to which scores on these

measures truly offer information about severity above and beyond what is provided by age and/or IQ. Often when people talk about a child at the “high end of the spectrum,” they are referring to the child’s high IQ or well-developed language abilities. Thus, putting the issues of IQ and language aside, it is not entirely clear what “severe” vs. “average” vs. “mild” ASD would be expected to look like. The ADOS calibrated severity score (Gotham et al. 2009) is a new metric that converts ADOS raw scores into standard scores based on comparisons of other children within the same age range and with similar language abilities. More research is needed to determine the validity of this score as a measure of ASD severity.

Future Directions

To begin to deal with the issue of severity in DSM-V, the proposed revisions stipulate that a diagnosis of ASD will be described in terms of severity in two domains: social-communication symptoms and fixated interests/repetitive behaviors. Any associations with other known genetic or medical conditions, language disorders, intellectual disability, or psychiatric disorders will also be specified (see www.dsm5.org). It will be important to evaluate whether this dimensional approach to “within-ASD” designations results in enhanced inter-rater reliability among professionals compared to the current categorical approach. In addition, it will be important to examine the utility of the new severity dimensions for both clinical (e.g., using different severity designations to track children into various services) and research (e.g., as predictors of outcome to stratify genetics samples) purposes. Undertaking other measure development efforts in the area of ASD severity may also be helpful in providing a means of operationalizing various anchor points along the spectrum (e.g., mild vs. severe ASD) or along multiple spectra (i.e., defined by more narrow behavioral dimensions) relevant to a diagnosis of ASD.

See Also

- [Asperger Syndrome](#)
- [DSM-IV](#)

References and Reading

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: APA Press.
- Amir, R. E., Ignatia, B., Van den Veyver, I. B., Wan, M., Tran, C. Q., Francke, U., et al. (1999). Rett syndrome is caused by mutations in X-linked MECP2 encoding methyl-CpG-binding protein 2. *Nature Genetics*, 23, 185–188.
- Bennett, T., Szatmari, P., Bryson, S., Volden, J., Zwaigenbaum, L., Vaccarella, L., et al. (2008). Differentiating autism and Asperger syndrome on the basis of language delay or impairment. *Journal of Autism and Developmental Disorders*, 38, 616–625.
- Constantino, J. N. (2002). *The social responsiveness scale*. Los Angeles: Western Psychological Services.
- Constantino, J. N., Gruber, C., Davis, S., Hayes, S., Passanante, N., & Przybeck, T. (2004). The factor structure of autistic traits. *Journal of Child Psychology and Psychiatry*, 45, 719–726.
- Cuccaro, M., Nations, L., Brinkley, J., Abramson, R., Wright, H., Hall, A., et al. (2007). A comparison of repetitive behaviors in Asperger's disorder and high functioning autism. *Child Psychiatry and Human Development*, 37, 347–360.
- Gotham, K., Pickles, A., & Lord, C. (2009). Standardizing ADOS scores for a measure of severity in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39, 693–705.
- Klin, A., Pauls, R., Schultz, R., & Volkmar, F. (2005). Three diagnostic approaches to Asperger syndrome: Implications for research. *Journal of Autism and Developmental Disorders*, 35, 221–234.
- Lord, C., & Bishop, S. L. (2010). Autism spectrum disorders: Diagnosis, prevalence, and services for children and families. *Society for Research in Child Development Social Policy Report*, 24, 2.
- Lord, C., Risi, S., DiLavore, P., Shulman, C., Thurm, A., & Pickles, A. (2006). Autism from 2 to 9 years of age. *Archives of General Psychiatry*, 63, 694–701.
- Lord, C., Petkova, E., Hus, V., Gan, W., Lu, F., Martin, D. M., et al. (2012). A multi-site study of the clinical diagnosis of different autism spectrum disorders. *Archives of General Psychiatry*, 69(3), 306–313.
- Mahoney, W. J., Szatmari, P., MacLean, J. E., Bryson, S., Bartolucci, G., Walter, S., et al. (1998). Reliability and accuracy of differentiating pervasive developmental disorder subtypes. *Journal of the American Academy of Child and Adolescent Psychiatry*, 37, 278–285.
- Ozonoff, S., South, M., & Miller, J. N. (2000). DSM-IV-defined Asperger syndrome: Cognitive, behavioral, and early history differentiation from high-functioning autism. *Autism*, 4(1), 29–46.
- Stone, W. L., Lee, E. B., Ashford, L., Brissie, J., Hepburn, S., Coonrad, E., et al. (1999). Can autism be diagnosed accurately in children under 3 years? *Journal of Child Psychology and Psychiatry*, 40, 219–226.
- Szatmari, P., Bryson, S. E., Boyle, M. H., Streiner, D. L., & Duku, E. (2003). Predictors of outcome among high functioning children with autism and Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 44, 520–528.
- Walker, D., Thompson, A., Zwaigenbaum, L., Goldberg, J., Bryson, S., Mahoney, W., et al. (2004). Specifying PDD-NOS: A comparison of PDD-NOS, Asperger syndrome, and autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43, 172–180.
- World Health Organization. (1992). *International Classification of Diseases (ICD-10)* (10th ed.). Geneva: Author.

Speech

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Synonyms

[Talking](#); [Verbalization](#)

Definition

Speech is the physical production of language. Speech is a complex act that requires the coordination of multiple systems including respiration, voicing, articulation, and prosody. Speech begins with respiration. Respiration provides the air supply necessary to create speech sounds, also known as phonemes. As the air moves through the vocal tract into the larynx, the vocal folds are vibrated to produce voiced sounds (e.g., /g/) or remain still to produce voiceless sounds (e.g., /h/). Once the air moves out of the larynx, it continues through the vocal tract and into the mouth where the air is

shaped into sounds by the articulators (e.g., lips, teeth, and tongue). Accurate speech sound production requires appropriate timing, direction, force, speed, and placement of the articulators (American Speech-Language-Hearing Association n.d.; Freed 2000; Zemlin 1998). Producing speech enables individuals to communicate (e.g., expressing wants and needs, asking for information or help).

See Also

- Expressive Language
- Phonemes
- Phonetics
- Phonology
- Prosody

References and Reading

- Freed, D. (2000). *Motor speech disorders: Diagnosis and treatment*. San Diego: Singular.
- What is Language? What is speech? (n.d.). In American-Speech-Language-Hearing-Association typical speech and language development. Retrieved 18 Jan 2011, from http://www.asha.org/public/speech/development/language_speech.htm
- Zemlin, W. R. (1998). *Speech and hearing science: Anatomy and physiology* (4th ed.). Boston: Allyn and Bacon.

Speech and Language Services

- Speech-Language Intervention

Speech Clinician (Not Preferred Title)

- Speech-Language Pathologist (SLP)

Speech Delay

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Definition

Speech delay is the diagnosis given to children who exhibit normal language development, but their speech production skills fall below those expected based on their chronological age and level of cognitive or intellectual functioning (Vinson 2007). Speech-language pathologists use standardized language tests to diagnose speech delay. While the etiology underlying the delay may be unknown, speech delay has been associated with a variety of developmental disorders including autism spectrum disorders. Predictors of long-term speech delay in late-talking toddlers at 30–35 months include limited phonetic inventory, simple syllable structures, more sound errors, greater inconsistency in substitution errors, atypical errors, and slow rate of resolution (Williams and Elbert 2003). Speech therapy may be provided to children with a diagnosis of speech delay with the goal of helping them attain communication skills that are commensurate with their chronological/developmental age (Roth and Worthington 2010).

See Also

- Articulation Disorders
- Speech Impairments
- Speech/Communication Disabilities

References and Reading

- Late Blooming or Language Problem. (n.d.). In American-Speech-Language-Hearing-Association typical speech and language development. Retrieved 3 Feb 2011 from <http://www.asha.org/public/speech/disorders/LateBlooming.htm>

- Roth, F. P., & Worthington, C. K. (2010). *Treatment resource manual for speech-language pathology* (4th ed.). Independence: Cengage Learning.
- Vinson, B. (2007). *Language disorders across the lifespan* (2nd ed.). Clifton Park: Thomson Delmar Learning.
- Williams, A. L., & Elbert, M. (2003). A prospective longitudinal study of phonological development in Late Talkers. *Language, Speech, and Hearing Services in Schools*, 34, 138–153.

Speech Disorder

- ▶ [Articulation Disorders](#)
- ▶ [Speech Impairments](#)

Speech Impairment

- ▶ [Paraphasia](#)

Speech Impairment/Disorder

- ▶ [Speech/Communication Disabilities](#)

Speech Impairments

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Synonyms

[Speech disorder](#)

Definition

Speech impairment is the diagnosis given to an individual when one or more of the components of speech (e.g., articulation, voice, fluency) are

impaired. Speech-language pathologists use standardized tests to diagnose speech impairments. The different categories of speech impairments reflect the underlying etiology of the disorder and/or the impaired components. Childhood speech impairments include apraxia, dysarthria, speech sound disorders, stuttering, and voice. Adult speech impairments include apraxia, dysarthria, stuttering, and voice. An individual's speech impairment may emerge over the course of development, be acquired due to a brain injury, or be associated with a disorder such as cerebral palsy (American-Speech-Language-Hearing-Association n.d.). Speech-language pathologists work with individuals with speech impairments to either help them attain communication skills that are commensurate with their chronological/developmental age or premorbid status or to help them attain functional communication skills that enable them to operate in the daily environment without handicap (Roth and Worthington 2010).

See Also

- ▶ [Apraxia](#)
- ▶ [Dysarthria](#)
- ▶ [Speech](#)

References and Reading

- Adult Speech and Language. (n.d.). In American-Speech-Language-Hearing-Association adult speech and language. Retrieved 25 Jan 2011 from <http://www.asha.org/public/speech/disorders/AdultSandL.htm>
- Child Speech and Language. (n.d.). In American-Speech-Language-Hearing-Association child speech and language. Retrieved 25 Jan 2011 from <http://www.asha.org/public/speech/disorders/ChildSandL.htm>
- Roth, F. P., & Worthington, C. K. (2010). *Treatment resource manual for speech-language pathology* (4th ed.). Independence: Cengage Learning.
- What is Language? What is speech?. (n.d.). In American-Speech-Language-Hearing-Association typical speech and language development. Retrieved 25 Jan 2011 from http://www.asha.org/public/speech/development/language_speech.htm

Speech Morphology

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Synonyms

[Affixes](#); [Bound morphemes](#); [Grammatical morphemes](#)

Definition

Speech morphology deals with the organization of morphemes, or the smallest units of meaning, in spoken language. Morpheme arrangement is governed by morphological rules and, in spoken language, by morphophonological rules. In speech, these rules dictate when pronunciation changes result from the combination of morphemes (e.g., *divine* + *ity* to form *divinity*).

Morphemes are categorized according to structure and function. Free morphemes can stand alone (e.g., *cat*), while bound morphemes must be attached to a free morpheme (e.g., plural *-s*). Bound morphemes can be classified as derivational or inflectional. Derivational morphemes change the class of a word (e.g., the verb *work* is changed to a noun by adding the morpheme *-er*, as in *work* → *worker*), while inflectional morphemes are associated with grammatical changes (e.g., plural, possessive, verb tense). While some individuals with autism spectrum disorders may exhibit normal morphological aspects of speech and language, others may exhibit deficits in grammatical morphology similar to those observed in children with specific language impairment (SLI). There is evidence to indicate that children with SLI have particular difficulty with finite verb morphology.

See Also

- ▶ [Language](#)
- ▶ [Speech](#)

References and Reading

- Gleason, J. B., & Ratner, N. B. (2009). *The development of language* (7th ed.). Boston: Pearson Education.
- Nicolosi, L., Harryman, E., & Kresheck, J. (1996). *Terminology of communication disorders: Speech-language-hearing* (4th ed.). Baltimore: Williams and Wilkins.
- Owens, R. E. (2010). *Language disorders: A functional approach to assessment and intervention* (5th ed.). Boston: Allyn & Bacon.

Speech Pathologist (Not Preferred Title)

- ▶ [Speech-Language Pathologist \(SLP\)](#)

Speech Sound

- ▶ [Phonemes](#)

Speech Sound Disorder

- ▶ [Articulation Disorders](#)

Speech Sound Production

- ▶ [Articulation](#)

Speech Teacher (Not Preferred Title)

- ▶ [Speech-Language Pathologist \(SLP\)](#)

Speech Therapist (Not Preferred Title)

► Speech-Language Pathologist (SLP)

Speech Therapy

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Definition

Speech therapy is a treatment aimed at modifying, minimizing, or resolving a communication disorder (Tomblin 2002).

Historical Background

The first speech defects and treatments were reported in the third and fourth century BC (Klingebeil 1939). The roots of modern day logopedics, known as speech therapy in the United States, began on the European continent (Rieber and Froeschels 1966). The first center that took a special interest in speech functioning and rehabilitation was established in 1895 and located in Vienna. The work at this center was carried out by physicians who went on to form the profession of phoniatics (the medical, research, and treatment of structures involved in speech production). In 1921, a phoniatriest and educationalist came together to initiate a training program for teachers that focused on teaching speech, and by 1928, 39 schools in Vienna were offering remedial speech courses. In the 1900s, Americans began to travel to Germany and other European countries to study speech. As the number of speech practitioners in Europe grew, a decision was made to host an international

congress in 1924, which focused on various aspects of communicative disorders. The International Association of Logopedics and Phoniatics was founded during this congress (DeMontFort 1990).

During this same time, graduate programs were beginning to develop in the United States. One of the first graduate programs was established in 1914 at the University of Wisconsin by Smiley Blanton. In the United States, the first practitioners of speech pathology were professionals and educators who took an interest in helping individuals with speech problems. These practitioners, referred to as “speech correctionists,” were made up of physicians, scholars, and public school administrators all of whom fell under the National Association of Teachers of Speech. In 1925, Carl Seashore, a professor of psychology at the University of Iowa, initiated a meeting aimed at forming an academy for speech correction that was independent of the National Association of Teachers of Speech. The outcome of this meeting was the formation of the American Academy of Speech Correction, which would eventually go on to become the American Speech-Language-Hearing Association (Duchan 2002; van Riper 1981). The number of members of the American Speech-Language-Hearing Association has grown from 25 members in 1925 to 175,673 members in 2016 (American Speech-Language-Hearing Association 2017). Although the roots of modern speech therapy began in Europe and the United States, today, clinicians provide speech therapy to individuals with communicative disorders throughout the world. However, there are significant differences in the length and content of the training programs. Many countries with limited resources do not have training programs even though these countries have citizens with hearing, speech, and language problems. Thus, a primary goal of the International Association of Logopedics and Phoniatics is to establish an international set of essential principles and common ground for those who serve individuals with communication disorders throughout the world (Butler 1990).

Rationale or Underlying Theory

Communication is an essential social tool. Communication is considered disordered when an individual is no longer able to accomplish necessary social functions or the manner in which an individual communicates is viewed negatively (Tomblin 2002). The overall goal of speech therapy is to modify, minimize, or resolve an individual's communication disorder and subsequently improve his or her quality of life. To accomplish this, the speech-language pathologist must identify factors causing and/or exacerbating the problem and then provide appropriate treatment. Treatment may be aimed at targeting areas of weakness, providing support or alternative methods of communication, and/or addressing behaviors and environments that affect communication (Tomblin 2002).

Goals and Objectives

The objective of speech therapy varies depending on the individual. Goals most often fall into one of the two categories: (1) the attainment of communication skills that are commensurate with the individual's chronological/developmental age or premorbid status or (2) the attainment of functional communication skills that enable the individual to function in his or her daily environment (Roth and Worthington 2001).

Treatment Participants

Individuals with a diagnosed communicative disorder are eligible for speech therapy. Communicative disorders do not include speech and language differences that arise from dialect usage or nonnative language (Roth and Worthington 2001).

Treatment Procedures

The treatment of a communicative disorder begins with an assessment of the individual's

communication behaviors. Treatment involves the following steps: (1) determination of treatment goal(s), (2) measurement of the behavior prior to the start of intervention for the purpose of establishing a pretreatment baseline, (3) implementation of intervention, and (4) generalization or carryover of the newly mastered communicative behavior from the clinical setting to the natural environment (Hedge 1988; Roth and Worthington 2001).

Treatment goals may be determined using either a normative or client-specific strategy. The normative strategy for treatment goal selection is based upon typical communication development. Therapy targets are selected and taught in the same order that they emerge developmentally. Normative goal selection is most often used with children who are receiving therapy to address articulation and/or language impairments. Client-specific goal selection involves selection of therapy targets based on an individual's specific needs rather than developmental norms. This includes "consideration of (1) the frequency with which a specific communicative behavior occurs in a client's daily activities, (2) the relative importance of a specific communicative behavior to the client, regardless of how often it occurs, (3) the client's potential for mastery of a given communication skills" (Roth and Worthington 2001, p. 7).

There are often a variety of treatment methods available to target the selected treatment goal. Speech-language pathologists use evidence-based practice, which is the integration of clinical expertise with the best available evidence from systematic research, to determine what intervention method and/or therapy techniques to use (Dollaghan 2007; Reilly et al. 2004).

A baseline measurement of the behavior that will be targeted during speech therapy should be taken prior to the start of intervention. Baseline measures provide a picture of the individual's communicative behaviors prior to treatment and a base against which the effects of various treatments may be measured. More specifically, baseline measurements enable a clinician to document whether or not the targeted behavior has improved. Without pretreatment baseline

measures, clinicians are unable to document whether an intervention was or was not effective (Hedge 1988).

Finally, clinicians must determine whether or not the targeted behavior has been mastered. Mastery of a behavior includes generalization of a targeted behavior to outside of the clinical setting. To facilitate generalization, a variety of stimuli should be used when teaching the targeted behavior, and the audience with whom the therapy targets are practiced should be varied (Roth and Worthington 2001).

Efficacy Information

The National Outcomes Measurement System (NOMS), organized by the American Speech-Language-Hearing Association, is a national database comprised of outcome data from speech-language pathologists (SLPs) and audiologists working with adults and children in school and health-care settings. The results from this database indicate that speech therapy is effective at modifying, minimizing, and/or resolving communication disorders. In the area of adult treatment, intervention is considered effective if there is measurable change from admission to discharge. Measurable changes for patients were found in the areas of memory, swallowing, motor speech, language comprehension, and expression (American Speech-Language-Hearing Association *n.d.-b*). In the area of child treatment, children must make demonstrable progress in targeted areas. Results from prekindergarten data found that more than half of the children made demonstrable progress following SLP intervention in the areas of articulation/intelligibility, cognitive orientation, pragmatics, spoken language comprehension, spoken language production, and swallowing. This included children with lower functional communication and/or swallowing abilities. Thus, the data suggests that speech therapy is effective, as demonstrated by measuring change, in treating both adult and child communicative disorders (American Speech-Language-Hearing Association *n.d.-a*; American Speech-Language-Hearing Association *n.d.-b*; Mullen and Schooling 2010).

Outcome Measurement

To determine outcome measurements, clinicians should compare pretreatment baseline measures to posttreatment measures and/or compare scores from admission and discharge. The differences in pretreatment and posttreatment measures may be used to determine the amount of change that has occurred as a result of treatment (Hedge 1988). The National Outcomes Measurement System, organized by the American Speech-Language-Hearing Association, uses functional communication measures to describe the change in an individual's functional communication and/or swallowing ability over time. These measures are based on disorder-specific scales (Mullen and Schooling 2010).

Qualifications of Treatment Providers

Qualifications needed to provide speech therapy services in the United States include Certification of Clinical Competence (CCC). This certificate, granted by the American Speech-Language-Hearing Association, requires a master's degree from an accredited graduate program, a passing score on the national Praxis exam (a certification examination written and administered by the Educational Testing Service), and completion of a 9-month clinical fellowship under the supervision of a certified speech-language pathologist. Most states license speech-language pathologists. State licensing requirements are most often comparable, although they may not be identical, to the qualifications for the Certificate of Clinical Competence. Speech-language pathologists licensed in other states must apply for licensure in the state in which they are practicing (American Speech-Language-Hearing Association *n.d.-c*).

References and Reading

- American Speech-Language-Hearing Association. (2017). ASHA summary membership and affiliation counts, year-end 2016. Available from www.asha.org.
- American Speech-Language-Hearing Association. (n.d.-a). Pre-Kindergarten NOMS 2009: National report. Retrieved 12 Mar 2011, from http://www.asha.org/members/research/noms/noms_data.htm.

- American Speech-Language-Hearing Association. (n.d.-b). SLP treatment outcomes: Fact sheet. Retrieved 28 Feb 2011, from http://www.asha.org/members/research/noms/noms_data.htm.
- American Speech-Language-Hearing Association. (n.d.-c) Qualifications needed to provide SLP services in health care. Retrieved 28 Feb 2011, from http://www.asha.org/careers/recruitment/healthcare/recruit_appendix.htm.
- Butler, K. G. (1990). IALP today. *American Speech and Hearing Association*, 32, 34.
- DeMontFort, M. (1990). A brief history of IALP: International association of logopedics and phoniatrics. *American Speech and Hearing Association*, 32, 33–35.
- Dollaghan, C. A. (2007). *The handbook for evidence-based practice in communication disorders*. Baltimore: Paul H. Brooks Publishing.
- Duchan, J. F. (2002). What do you know about your profession's history? The ASHA leader online. Retrieved 28 Feb 2011, from <http://www.asha.org/Publications/leader/2002/021224/021224a.htm>.
- Hedge, M. N. (1988). *Treatment procedures in communicative disorders* (3rd ed.). Austin: Pro.Ed.
- Klingebeil, G. M. (1939). The historical background of the modern speech clinic. *The Journal of Speech and Hearing Disorders*, 4, 115–132.
- Mullen, R., & Schooling, T. (2010). The national outcomes measurement systems for pediatric speech-language pathology. *Language, Speech, and Hearing Services in Schools*, 41, 44–60.
- Reilly, S., Douglas, J., & Oates, J. (Eds.). (2004). *Evidence based practice in speech pathology*. London: Whurr.
- Rieber, R. W., & Froeschels, F. (1966). A historical review of the European literature in speech pathology. In R. W. Rieber & R. S. Brubaker (Eds.), *Speech Pathology: An international study of the science* (pp. 5–23). Philadelphia: J.B. Lippincott.
- Roth, F. P., & Worthington, C. K. (2001). *Treatment resource manual for speech language pathology* (2nd ed.). Albany, NY: Singular.
- Roth, F. P., & Worthington, C. K. (2010). *Treatment resource manual for speech-language pathology* (4th ed.). San Diego: Singular Thomson Learning.
- Tomblin, J. B. (2002). Perspectives on diagnosis. In J. B. Tomblin, H. L. Morris, & R. Spriestersbach (Eds.), *Diagnosis in speech-language pathology* (pp. 1–28). San Diego: Singular Thomson Learning.
- van Riper, C. (1981). An early history of ASHA. *ASHA Magazine*, 23, 855–858.

Speech/Communication Disabilities

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Synonyms

Communication disorder; Language impairment/disorder; Speech impairment/disorder

Short Description or Definition

Speech/communication impairments are among the core features of autism spectrum disorders (ASD). Although enormous variability in the development of speech and language is observed in individuals with ASD, even those who have high cognitive and language abilities exhibit some type of communication disability. Deficits can occur at the nonverbal level (e.g., gestures, facial expressions, eye gaze), paralinguistic level (e.g., prosody, intonation), and linguistic level (e.g., language, speech). Communication deficits in the social use of speech and language are particularly salient. Research suggests that a subset of children with ASD also have grammatical deficits similar to children with specific language impairment. In addition, speech sound disorders are evident in a subset of children with ASD. Approximately 20–40% of individuals with autism never develop spoken language, and about 20% exhibit a loss of language skills as toddlers, also known as language regression.

Aspects of communication that are relatively preserved in individuals with ASD include segmental phonology (i.e., the system of speech sounds), syntax, and morphology (i.e., the form or structure of language). Areas of relative difficulty include nonsegmental speech production (e.g., prosody, intonation, stress patterns, vocal quality), semantics (i.e., meaning), and pragmatics (i.e., social use of language).

Speech Therapy (Not Preferred)

► Speech-Language Intervention

Categorization

The *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5; American Psychiatric Association 2013) includes four categories of communication disorders: language disorder, speech sound disorder, childhood-onset fluency disorder, and social (pragmatic) communication disorder. Speech/communication disabilities can be associated with developmental disabilities and syndromes, such as autism spectrum disorders.

Epidemiology

Speech/communication disabilities are core features of ASD. According to Fombonne's (2009) review, the prevalence for all autism spectrum disorders (previously referred to as pervasive developmental disorders) is estimated at 60–70 per 10,000.

Natural History, Prognostic Factors, Outcomes

Communication delays and deficits are often the first indicators of ASD. For example, children later diagnosed with ASD show a lack of interest in faces, voices, and social interactions as infants. Later in development, children with ASD may fail to understand or produce communicative gestures (e.g., pointing) and may be delayed in babbling, vocalizing, and speech development. Lack of communication skills is one of the most significant stress factors for families of children with ASD.

The speech/communicative outcomes of individuals with ASD are extremely variable. About 20% of children with ASD exhibit a regression in speech and language skills during their second year of life. About 40–50% never develops functional speech except for perhaps a few single words (although this percentage may be decreasing due to the positive effects of early identification and intervention). Little is known about the communication skills of nonverbal individuals with ASD because research has focused on verbal individuals.

Those individuals with ASD who do develop speech are often significantly delayed and do not speak until well into their preschool years. High-functioning individuals with ASD may have IQs in the normal range and appear precocious in their vocabulary size and ability to speak in detail on certain topics. Despite these strengths, this population continues to struggle with the comprehension and production of social and non-literal communication.

Several characteristics have been linked to better communicative outcomes. For example, children with ASD who produce and imitate words, exhibit pretend play with objects, communicate with gestures, and show evidence of joint attention have greater rates of vocabulary growth and overall language skills. Negative prognostic indicators include a regression in language skills after a period of development and severe receptive language impairment. Children with lower IQs also tend to have poorer communicative functioning. Even for individuals with high cognitive and language abilities who attend college and are able to live independently, ASD is a lifelong disability that affects social communication and functioning.

Clinical Expression and Pathophysiology

Early Communication Development

Communication is a broad term that encompasses numerous modes of sending and receiving messages, including gestures, body language, facial expressions, language, and speech. Typically developing children exhibit social communicative behaviors beginning in infancy. For example, they turn toward human voices, are fascinated by faces, smile to hold an adult's attention, vocalize, and demonstrate joint attention (e.g., paying attention to the same object as another person). In the first year of life, typically developing infants express a range of communicative intents, such as requesting, greeting, and protesting. For individuals with ASD, communication deficits are apparent almost from birth. One of the first signs of ASD is a lack of responsiveness to social interactions. For example, infants later diagnosed with ASD were described by their

parents as uninterested in human voices or faces. They rarely smiled at others, vocalized in a communicative manner, or engaged in social games such as “peekaboo.” In addition, 1-year-olds who were later diagnosed with ASD exhibited a lack of joint attentional behavior and paid less attention to people in their environment. The communicative intents of children with ASD are primarily to regulate their environment (e.g., getting others to do things for them) rather than for social purposes. They may exhibit unusual gestures, such as pulling an adult’s hand toward a desired object (rather than pointing at the object or verbally requesting it). Other symptoms of communicative deficits include delayed development of pointing gestures, a lack of typical imitation skills, and deficits in pretend and imaginative play. In the second year of life, the receptive language abilities of children with ASD are depressed relative to their expressive abilities. The gap tends to narrow over time, with receptive and expressive language abilities becoming more similar by ages 3–4. Parents of children with ASD report becoming seriously concerned about their children’s development during the toddler years, particularly due to their children’s receptive and expressive language delays. Children with ASD are generally late to begin talking and develop speech and language at a slower rate than typical children. When they do speak, children with ASD are less spontaneous in their communication, and verbal expression is sparse. They also exhibit deficits in social communication, including following politeness rules, turn taking, and engaging in conversation.

Later Language, Speech, and Communicative Development

Language consists of three primary domains: form, content, and use. *Form* involves the structure of language (i.e., phonology, syntax, morphology), *content* involves the meaning of language (i.e., semantics, vocabulary), and *use* refers to the social use of language (e.g., pragmatics). Effective communication involves the interaction of these three domains. For example, language produced by typical individuals is most often syntactically correct, meaningful, and socially

appropriate. In addition, effective communicators can decode other’s communication in terms of its form, content, and use. In individuals with ASD, the domains of language can be dissociated, negatively impacting communication. Specially, the form of language may be preserved (e.g., syntax, morphology, phonology), but the meaning, and especially social use of language, is impaired. More details on the language, speech, and communication characteristics of individuals are included below.

Language Form (Phonology, Morphology, Syntax)

The language form of individuals with ASD who communicate verbally is relatively unimpaired compared to the language domains of content and use. In general, development of language form in children with ASD follows the same sequence as for typically developing children. Syntactic errors that are observed seem to be related to semantic or pragmatic difficulties. In general, the development of language form is commensurate with nonverbal mental age, although a more restricted range of syntactic constructions may be produced. A subgroup of children with ASD may exhibit grammatical deficits similar to children with specific language impairment. This subgroup of children is likely to omit certain grammatical morphemes such as articles, auxiliary and copula verbs, and verb tense markers (e.g., past tense, third person singular).

Language Content (Vocabulary, Semantics)

Language content involves the rules for relating words to meaning. Individuals with ASD exhibit both strengths and impairments in this language domain. For example, research suggests that overall vocabulary knowledge may be a relative strength in individuals with autism spectrum disorders (ASD). In addition, children with ASD use semantic groupings (i.e., word relationships) in typical ways for categorizing and retrieving words. Despite these strengths, notable deficits and unusual characteristics are apparent. For example, the acquisition of words that refer to mental state or emotional concepts may be especially impaired. Individuals with ASD may

have difficulty generalizing the meanings of words to new contexts (e.g., understanding that the word “dog” can refer to many animals, not just the family pet). In addition, they often have difficulty comprehending vocabulary in nonliteral language (e.g., slang, figures of speech, proverbs, metaphors).

Individuals with ASD may produce speech that appears to be irrelevant to the current communicative context. These utterances are referred to as idiosyncratic or metaphorical language. For example, Kanner (1946) described a child with ASD who would yell, “Don’t throw the dog off the balcony!” whenever he was about to throw something. His parents reported that several years earlier, they had been staying in a hotel with a balcony and warned the child not to throw his stuffed toy dog over the railing. Kanner emphasized that this type of utterance was not irrelevant or meaningless. Rather, individuals with ASD attach unique meanings to these “figures of speech” based on specific past experiences.

Language Use (Pragmatics, Social Use of Language)

The social use of language is particularly impaired in individuals with ASD, including for individuals with IQs in the normal range. Deficits in social communication are evident almost from birth, as children with ASD are less responsive to voices and faces. Social skills are learned and applied in transactional contexts, that is, during interactions between communication partners. The deficits in social communication in individuals with ASD result in less social experience, contributing to impaired development and learning. For children who develop verbal language, the speech acts they do exhibit are primarily for regulating their environment and the behaviors of others, rather than for social purposes. Later in development, engaging in conversation appears to be especially difficult. Deficits in eye gaze, intonation, topic maintenance, understanding the communicative intent of others, and providing the appropriate amount of information are apparent. In general, individuals with ASD exhibit impairments participating in communicative interactions

that involve joint reference or shared topics and perspectives.

Speech

Among individuals with ASD who speak, articulation skills are a relative strength and are generally commensurate with mental age, although there may be a higher incidence of residual speech distortion errors on sounds such as /r/, /l/, and /s/ in adults. Despite strengths in speech sound development, paralinguistic aspects of speech (e.g., intonation, prosody) and vocal quality may be atypical, causing communication difficulties. For example, prosody and intonation may be monotonous, inappropriate, or overly dramatic. In addition to expressive deficits, individuals with ASD may have difficulty interpreting the prosody and intonation of others leading to misinterpretation of sarcasm, etc.

Echolalia

Echolalia is the repetition, with similar intonation, of words or phrases spoken by another person. Echolalia can be immediate (e.g., a child repeats, “Are you hungry?” when an adult asks him that question) or delayed (e.g., a child says, “You look sleepy,” to indicate that it is bedtime). Echolalia was once considered to be aberrant, undesirable behavior. Now it is recognized as serving various communicative functions. Echolalia is more common in children with less language ability and tends to decline as language develops. Other populations of children, including typical children, also produce echolalic utterances, although not to the extent observed in children with ASD.

Pronoun Reversal

It has frequently been noted that children with ASD appear to reverse the pronouns in their utterances. For example, they may say, “Pick you up!” instead of, “Pick me up!” These errors are thought to be a result of echolalia.

Evaluation and Differential Diagnosis

As stated earlier, impairments in communication, particularly for social purposes, are core features

of ASD. Therefore, all individuals with ASD will have communication needs, although they will vary depending on the age, level of functioning, and individual characteristics of each person. For very young children, assessment will include evaluation of preverbal communication abilities, including gestures, eye gaze, joint attention, vocalizations, responsiveness to communication, and communicative functions. Observations during play or activities of daily living are important for gaining information about children's communicative behavior and their caregivers' interactional style. The use of checklists while observing the child in naturalistic interactions is common and may be the only feasible method of assessment for many children with ASD who have difficulty participating in structured test formats (see Paul and Norbury 2012).

A variety of parent report/interview assessment tools are available, including the *Receptive-Expressive Emergent Language Test, Third Edition* (REEL-3; Bzoch et al. 2003) and the *Vineland Adaptive Behavior Scales, Third Edition* (Vineland-3; Sparrow et al. 2016). Other assessment tools include direct observation of the child and may include more structured interactions or elicitation of particular behaviors. Examples include the *Communication and Symbolic Behavior and Play Scales-DP* (CSBS; Wetherby and Prizant 2003), the *Peabody Picture Vocabulary Test, Fourth Edition* (PPVT-5; Dunn 2019), and the *Preschool Language Scale, Fifth Edition* (PLS-5; Zimmerman et al. 2011).

In verbal children, language samples may be collected for assessing the expression and use of language in naturalistic settings. Specific areas to assess include responsiveness to speech, mean length of utterance (a measure of syntactic development), word use, echolalia, pronoun use, and pragmatics. Pragmatic analysis may include the range of communicative functions (e.g., directing others, reasoning, empathizing), discourse management, register variation (e.g., politeness), presupposition (e.g., providing enough background information), and manner of communication.

For individuals with ASD who have higher cognitive and language abilities, and who can

participate in formal testing situations, a variety of standardized language assessments are available (see Paul and Norbury 2012). Assessments that focus on pragmatic language or verbal reasoning may be the most useful in identifying core deficits. It is important to supplement standardized testing with informal observations within a variety of naturalistic contexts in order to capture deficits in social communication.

For individuals with ASD who are nonverbal, it is important to assess the various ways that they do communicate. In addition, their ability to use augmentative and alternative communication (AAC) may also be evaluated.

Speech can be assessed through procedures common to the evaluation of any client with a suspected speech sound disorder, depending on the ability of the individual with ASD to participate in a standardized assessment. Common tests include the *Arizona Articulation Proficiency Scale, Fourth Edition* (Arizona-4; Fudala and Stegall 2017) and the *Goldman-Fristoe Test of Articulation 3* (GFTA-3; Goldman and Fristoe 2015). Also, speech can be evaluated through more naturalistic sampling procedures (e.g., recording an inventory of a client's phonetic repertoire during spontaneous speech).

Treatment

For individuals with ASD, research has demonstrated that a range of approaches are effective for promoting communication abilities (cf. ASHA 2017). Common approaches range from naturalistic (e.g., Floor Time, Greenspan et al. 1998) to highly structured, behavioral interventions (e.g., Lovaas et al. 1989). The incorporation of peers as models and/or trainers is also common. According to the National Research Council (2001), educational interventions for individuals with ASD should begin as early as possible, programming should be intensive with repeated and planned teaching opportunities, teacher-student ratios should be low, mechanisms for ongoing assessment and program evaluation should be in place, and family involvement and training is important.

In addition, intervention for individuals with ASD should include spontaneous and functional communication, social skills, play skills, peer interactions, generalization of skills to natural contexts, mechanisms for addressing challenging behaviors, and promoting functional academic skills when appropriate.

For young children in the beginning stages of language development, the goals of intervention include rewarding efforts for communication and speech, expanding vocabulary and communicative functions, encouraging multiword utterances, expanding sentence types, developing emergent literacy, teaching functional use of imitation, and capitalizing on memorized forms. Various evidence-based approaches to facilitating language may be implemented, including prelinguistic milieu teaching, focused stimulation, and conversational recasting. In addition, approaches that focus on parent training, such as *It Takes Two to Talk, More Than Words or Talkability* (developed by The Hanen Centre), have been shown to be effective.

For nonverbal individuals with ASD, the use of augmentative and alternative communication (AAC) strategies is common for facilitating communication. AAC approaches include sign language, Picture Exchange Communication System (PECS), communication boards, and voice output devices. For individuals who are literate, written language can be an effective means of communication and can be used for compensatory purposes (e.g., using a script or communication checklist for functioning within various situations). For example, Social Stories (Gray 1993) employ a story format using visual materials to improve an individual with ASD's social understanding of various situations. Technology (e.g., texting, social networking) may assist individuals with ASD in social communication without the stress of face-to-face interactions. Comprehension monitoring strategies for older individuals with ASD with higher cognitive and language abilities (e.g., checklists) may also be effective.

In general, intervention for individuals with ASD needs to address core deficits in communication and social skills.

See Also

- [Alternative Communication](#)
- [Communication Interventions](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- American Speech-Language-Hearing Association. (2017). *Autism*. Available from www.asha.org/Practice-Portal/Clinical-Topics/Autism/. Accessed 26 June 2017.
- Bzoch, K., League, R., & Brown, V. (2003). *Receptive-expressive emergent language test* (3rd ed.). Circle Pines: American Guidance Service.
- Dunn, D. M. (2019). *Peabody picture vocabulary test* (5th ed.). Bloomington, MN: NCS Pearson.
- Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. *Pediatric Research*, 65, 591–598.
- Fudala, J. B., & Stegall, S. (2017). *Arizona articulation proficiency scale-fourth revision*. Los Angeles: Western Psychological Services.
- Goldman, R., & Fristoe, M. (2015). *Goldman-Fristoe test of articulation 3*. Bloomington: Pearson.
- Gray, C. (1993). *The social story book*. Arlington: Future Horizons.
- Greenspan, S. I., Wieder, S., & Simons, R. (1998). *The child with special needs: Encouraging intellectual and emotional growth*. Reading: Addison-Wesley.
- Kanner, L. (1946). Irrelevant and metaphorical language in early infantile autism. *American Journal of Psychiatry*, 103, 242–246.
- Lovaas, I., Calouri, K., & Jada, J. (1989). The nature of behavioral treatment and research with young autistic persons. In C. Gillberg (Ed.), *Diagnosis and treatment of autism* (pp. 285–305). New York: Plenum Press.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press, Committee on Educational Interventions for Children with Autism, Division of Behavioral and Social Sciences and Education.
- Paul, R., & Norbury, C. (2012). *Language disorders from infancy through adolescence: Listening, speaking, reading, writing, and communicating* (4th ed.). St. Louis: Elsevier.
- Sparrow, S., Cicchetti, D., & Saulnier, C. (2016). *Vineland adaptive behavior scales* (3rd ed.). Minneapolis: Pearson Assessment.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, 3rd ed., pp. 335–364). Hoboken: Wiley.
- The Hanen Centre. <http://www.hanen.org/>. Accessed 15 Aug 2019.

- Wetherby, A., & Prizant, B. (2003). *Communication and symbolic behavior scales developmental profile*. Baltimore: Paul H. Brookes.
- Zimmerman, I. L., Steiner, V. G., & Pond, R. E. (2011). *Preschool language scales* (5th ed.). Bloomington: Pearson.

language therapy may be conducted individually, in small groups, in a classroom, or in the community.

The following speech, language, and swallowing disorders are described, followed by a description of the treatment focus.

Speech-for-the-Self

- [Private Speech](#)

Speech-Language Clinician (Not Preferred Title)

- [Speech-Language Pathologist \(SLP\)](#)

Speech-Language Intervention

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Synonyms

[Communication services](#); [Speech and language services](#); [Speech therapy \(not preferred\)](#); [Speech-language pathology practice](#); [Speech-language pathology services](#)

Definition

The overall objective of speech-language therapy or speech-language pathology services is to optimize individuals' ability to communicate and swallow, thereby improving quality of life. Speech-language pathologists are the professionals who provide speech, language, and swallowing evaluations and treatment. Speech-

Speech Disorders

- Speech sound production – how we say sounds and put sounds together in words
- Fluency (stuttering) – how well speech flows
- Voice – how the voice sounds
- Speech-language therapy – focuses on improving articulation or pronunciation of sounds, improving fluency in increasingly longer utterances, and helping people who sound hoarse, lose their voice easily, talk too loudly or too softly, or talk through their nose

Language Disorders

- Spoken language – how well we understand what we hear and how well we use words to tell others what we are thinking. Loss of language following a stroke or brain injury is known as *aphasia*.
- Written language – how well we understand what we read, how well we read, and how well we write to communicate.
- Speech-language therapy – helps individuals who have problems understanding, speaking, reading, writing, and spelling.

Social Communication or Pragmatic Language Disorders

Social communication, or *pragmatics*, refers to the way in which individuals use language in context. This is one of the defining features of autism. It includes following rules for conversation, such as taking turns, staying on topic, describing an event in sequence, and talking differently depending on the listener and the setting.

Speech-language therapy focuses on helping people adapt their language use depending on the

listener and the setting, learn the rules of conversation and storytelling, and use nonverbal language correctly (e.g., using gestures, knowing how close to stand when someone is talking).

Cognitive-Communication Disorders

People may have problems with memory, attention, problem solving, organization, and other thinking skills that interfere with effective communication.

Speech-language therapy helps individuals with organizing thoughts, recalling information, paying attention, planning, and solving problems.

Severe Communication Disorders

Speech-language therapy is used to assist people with severe communication disorders by providing direct service, by collaborating with teachers and families, and by evaluating, selecting, and developing augmentative and alternative communication (AAC) systems. Such systems may include low-tech communication boards with words or pictures, sign language, and speech-generating devices that produce a synthesized voice through use of a computer.

Feeding and Swallowing Disorders (Dysphagia)

People may have problems sucking, chewing, and swallowing food and liquid; this is known as *dysphagia*. Intervention helps to prevent poor nutrition, weight loss, and other health problems in individuals with dysphagia.

See Also

- [Communication Services](#)
- [Speech and Language Services](#)
- [Speech Therapy](#)
- [Speech-Language Pathologist \(SLP\)](#)
- [Speech-Language Pathology Practice](#)

References and Reading

American Speech-Language-Hearing Association (ASHA). www.asha.org

Speech-Language Pathologist (SLP)

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Synonyms

SLP (acronym for speech-language pathologist); Speech clinician (not preferred title); Speech pathologist (not preferred title); Speech teacher (not preferred title); Speech therapist (not preferred title); Speech-language clinician (not preferred title); Speech-language therapist (not preferred title)

Definition

The American Speech-Language-Hearing Association (ASHA) is the national professional, scientific, and credentialing association for 211,000 members and affiliates who are audiologists; speech-language pathologists; speech, language, and hearing scientists; and audiology and speech-language pathology support personnel. According to ASHA, *speech-language pathologists* are the professionals responsible for the diagnosis, prognosis, prescription, and treatment of speech, language (spoken and written), cognitive communication, social communication, and swallowing disorders. Working within the full range of human communication and its disorders, speech-language pathologists evaluate and treat individuals of all ages who have difficulty speaking, understanding, reading, writing, or swallowing. Individuals with autism typically present with communication disorders, including

delayed speech and language as well as social communication disorders. Speech-language pathologists are often the first professionals to identify that a child has autism. Speech-language pathologists screen, assess, diagnose, and provide intervention services for children and adults with autism. Speech-language pathologists may work independently or as members of a team. In addition, speech-language pathologists may:

- Train future professionals at colleges and universities
- Manage agencies, clinics, or private practices
- Engage in research to enhance knowledge about human communication processes
- Develop new methods and equipment to evaluate problems
- Establish more effective treatments
- Investigate behavioral patterns

Speech-language pathologists, as defined by ASHA, hold the ASHA Certificate of Clinical Competence in Speech-Language Pathology (CCC-SLP). Minimal criteria to become a speech-language pathologist include:

- Master's, doctoral, or other recognized post-baccalaureate degree.
- All graduate coursework and graduate clinical experience required in speech-language pathology must have been initiated and completed in a program accredited by the Council on Academic Accreditation in Audiology and Speech-Language Pathology (CAA) or in a program with CAA candidacy status.
- Successful passing of a national examination in speech-language pathology.
- Completion of a clinical fellowship after completion of academic course work and clinical practicum – the fellowship consists of at least 36 weeks and 1,260 hours, which can be completed full-time or part-time.

Demonstration of continued professional development is mandated for maintenance of the CCC-SLP. Where applicable, speech-language pathologists hold other required credentials (e.g., state licensure, teaching certification).

See Also

- [Speech Clinician \(Not Preferred Title\)](#)
- [Speech Pathologist \(Not Preferred Title\)](#)
- [Speech Therapist \(Not Preferred Title\)](#)
- [Speech-Language Clinician \(Not Preferred Title\)](#)
- [Speech-Language Therapist \(Not Preferred Title\)](#)

References and Reading

American Speech-Language-Hearing Association (ASHA). www.asha.org

Speech-Language Pathology Practice

- [Speech-Language Intervention](#)

Speech-Language Pathology Services

- [Speech-Language Intervention](#)

Speech-Language Therapist (Not Preferred Title)

- [Speech-Language Pathologist \(SLP\)](#)

Sphingolipidoses

- [Gangliosidoses](#)

Spinal Curvature

- [Scoliosis](#)

Spinal Fluid

- ▶ Cerebrospinal Fluid

SPM

- ▶ Sensory Processing Measure
- ▶ Sensory Processing Measure: Preschool (SPM-P)

SPM-P

- ▶ Sensory Processing Measure
- ▶ Sensory Processing Measure: Preschool (SPM-P)

Spoken Language

- ▶ Expressive Language
- ▶ Verbal Communication

Spoken Language Comprehension

- ▶ Listening Comprehension

Sports

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Definition

Sports are physical activities that usually require exertion, effort, and skill. They can be

individual or team oriented and require athletic ability. Many sports are competitive in nature where one individual or team attempts to beat another in a game or in the achievement of a goal. Sports can be played both indoors and outdoors.

Historical Background

The inclusion of individuals with autism spectrum disorders in the USA in sports can be traced directly to the creation of the Special Olympics. Eunice Kennedy Shriver was the founder of the Special Olympics which started as a summer day camp program for children with intellectual disabilities in her own backyard in 1962. In 1968, the first international Special Olympics were held at Soldier Field in Chicago, IL, with athletes from 26 states and Canada competing in track and field and swimming events. Over the past 40 years, the organization has grown to a global movement. It now includes both summer and winter Olympic Games and involves over 3 million athletes in 180 countries (Special Olympics 2011).

A second origin of the inclusion of individuals with autism in sports can be traced back to educational law. In 1975, the Education for All Handicapped Children Act, more commonly referred as P.L. 94-142, made education a right for all children with disabilities. Children with disabilities could no longer be denied an education. It was incumbent upon the local education agency or school district to provide a free and appropriate public education (FAPE) to all children with disabilities. Along with the legal concept of FAPE was the concept of mainstreaming. It was not sufficient for school districts to simply educate students with disabilities separately from nondisabled students. School districts were required to “mainstream” students for an optimal time period that was individualized to the particular student. Physical education classes along with art, lunch, recess, and some academic subjects were written into Individualized Education Plans (IEPs) of students as areas in the curriculum where students with disabilities were mainstreamed or educated side by side with their nondisabled peers.

This landmark legislation also included the concept of ancillary services, i.e., services provided to the student that were not directly educational in nature but helped to support the student in reaching his or her educational goals as a part of his or her IEP. Physical therapy, occupational therapy, and adaptive physical education were some of those ancillary services that helped individuals with autism participate in sports not only in the public school systems but eventually in the greater community as well. P.L. 94-142 was subsequently reauthorized as the Individuals with Disabilities Act (IDEA) and reaffirmed the importance of educating students with disabilities side by side with their nondisabled peers, and the concept of mainstreaming evolved into the concept of inclusion. Physical education classes were one of the primary parts of the curriculum where inclusion was written into a student's IEP. Under IDEA, physical therapy and adaptive physical education were solidified as ancillary services to support students with autism in reaching their IEP goal and helped pave the way to including individuals with autism in sports.

A tertiary influence on the inclusion of individuals with autism in sports needs to be credited to individuals with physical disabilities. In fact, the Paralympics movement predates Special Olympics and the passage of legislation in the USA promoting access for people with disabilities. "In 1948, Sir Ludwig Guttmann organized a sports competition involving World War II veterans with a spinal cord injury in Stoke Mandeville, England. Four years later, competitors from the Netherlands joined the games and an international movement was born. Olympic style games for athletes with a disability were organized for the first time in Rome in 1960, now called Paralympics. In Toronto in 1976, other disability groups were added and the idea of merging together different disability groups for international sport competitions was born. In the same year, the first Paralympic Winter Games took place in Sweden" (International Paralympic Committee 2011).

Disabled veterans and other groups of individuals with physical disabilities fought for access to services and the basic right to not be denied

physical access. These groups fought for the passage of the Vocational Rehabilitation Act of 1975 including Section 504 which forbade public entities which received federal funding from denying the benefits of the services of the institution solely based upon a person's disability. In 1990, the act was reauthorized and renamed as the Americans with Disabilities Act (ADA). These movements contributed to the rise of adapted physical activities where the sports or the equipment were modified so that individuals with physical disabilities could participate in sports. Modifying the social rules of some team sports for individuals on the autism spectrum, therefore, was made easier by these previous modifications. The importance of these movements changing the culture from exclusion to inclusion of people with different abilities cannot be underestimated when considering the inclusion of people with autism in sports.

Current Knowledge

It is imperative that individuals with ASDs develop lifelong habits that incorporate fitness into their daily routine. Sports can be a part of that fitness regime. The USA is faced with an obesity epidemic with over two-thirds of Americans being labeled as overweight or obese (Center for Disease Control [CDC] 2010). Sedentary lifestyles and diet are major contributors to obesity. The sequelae of obesity are serious including heart disease, diabetes, and certain forms of cancer (CDC 2010; Rundle et al. 2010). Additionally, obese individuals suffer a lowered quality of life and are actively discriminated against (CDC 2010). For children, being overweight puts them at greater risk for being the victim of bullying and increased risk of suicidality (CDC 2011a). Individuals who are obese also suffer from a shortened lifespan. The average American will live 77.9 years (CDC 2011b). Those who are labeled obese are expected to decrease not only their own life expectancy but that of the US population as a whole (Steward et al. 2009). However, a positive relationship exists between exercise and longevity. Individuals who exercise regularly have reduced mortality from all causes (Blair

and Brodney 1999; Paffenbarger et al. 1986). For individuals on the autism spectrum, regular moderate to vigorous exercise is associated with increased attention span, a decrease in stereotypies (Elliott et al. 1994), and a decrease in the use of psychotropic medications. Participation in sports is one way for individuals with ASDs to exercise regularly.

Individuals on the autism spectrum are at increased risk for developing obesity. One national health study of children found that children with an ASD were 42% more likely to be labeled obese than children without an ASD (Curtin et al. 2010). In fact, obesity is a well-recognized issue among people with developmental disabilities. A number of factors contribute to individuals with ASDs being more vulnerable to obesity. First, many individuals on the spectrum take medications to treat comorbid psychiatric and behavioral issues, which have either increased appetite or weight gain as a side effect. Second, some individuals on the spectrum have very restrictive self-imposed diets that are high in fat and have little nutritional value. In fact, anecdotal stories of individuals with ASDs eating only “white foods” such as breads and plain pasta suggest that they have a diet replete with carbohydrates that are associated with weight gain (VanBergeijk 2009). Third, some people with ASDs have both coordination problems and problems with proprioception (Weimer et al. 2001) or the ability to know where one’s own body is in space. Many sports require good eye-hand coordination, the ability to engage in multichanneling (Lawson 2001), a good sense of where one’s body is in space, and the ability to track a high-speed object and then perform another task in response (e.g., following a baseball with one’s eye after it leaves the pitcher’s hand and then responding by swinging a baseball bat to hit the approaching ball). The motor coordination nature of many sports conflicts directly with the individual on the spectrum’s disability, thereby discouraging them from participating. Fourth, the social nature of many team sports also discourages participation. Team sports require team members to use both verbal and nonverbal communication where the intent is not always explicit. For example, a

person with an ASD would have to be able to discern the intent of a team member in order to catch a behind-the-back pass or an “alley-oop” in a game of basketball. The person would also have to be flexible and not be too literal in their interpretation of rules of a game in order to be successful and would have to understand concepts like “stealing a base” as being a part of the game and not view it as cheating. The social stressors of team sports may discourage adults with ASDs from engaging in sports and benefiting from exercise. Again, a lack of regular exercise is associated with obesity, diabetes, orthopedic problems, loss of bone density, and heart disease (Pangrazi et al. 2007).

Individual sports may be the best possible solution to the dilemma of the need to exercise through sports. Persons on the autism spectrum could even participate in individual events as a part of a team (e.g., running track and cross country, long jump, or swimming). It would be important to avoid more complex social interactions during these events such as relay races where the stress of attempting to figure out when one is supposed to start one’s leg of a relay can be highly contextualized and requires the reading nonverbal communication.

Swimming has a plethora of benefits as a sport. It is a sport that has low impact upon the joints, and the probability of injury is minimal. Consequently, it is a sport that one can participate in throughout the lifespan. However, for individuals on the autism spectrum, there are additional benefits. For children on the autism spectrum, the number one cause of deaths is accidents. The most frequent accident is drowning followed closely by automobile accidents (Gillberg et al. 2010; Shavelle et al. 2001). By engaging in swimming lessons, individuals on the autism spectrum not only engage in exercise, but they also will learn important survival skills including “drown proofing.” The American Red Cross and YMCAs offer swimming lessons including those adapted for individuals on the spectrum.

As noted previously, many individuals on the spectrum have issues with coordination. Bilateral coordination is particularly poor often with individuals having extreme difficulty doing motions

or actions across their bodies. They frequently have a history of not crawling as infants. Crawling is a complex motor action that involves using alternative halves of the body simultaneously. As infants, this promotes bilateral integration of the brain. In swimming, the crawl (freestyle) or backstroke is one of the few motions that older individuals on the spectrum use alternating halves of the body. While swimming these strokes, the right hand works simultaneously with the left leg, and then the left hand conducts a stroke in coordination with the right leg. This may promote bilateral integration of the brain and improve coordination. The cardiovascular benefits of swimming are well documented.

Walking is an activity that is available to most individuals on the autism spectrum. It requires very little in terms of equipment or training. A sedentary person walks between 1,000 and 3,000 steps a day (www.walking.com). The CDC recommends walking 10,000 steps a day or roughly a little less than 5 miles a day (Besser and Dannenberg 2005). Walking 10,000 steps a day is associated with better cardiac health, reduced stress, better moods, reduced risk of diabetes, lower blood pressure, and improvements in sleep quality (VanBergeijk 2009).

Before starting a pedometer program with individuals on the autism spectrum, an assessment of their skills should be conducted. This includes their knowledge of and ability to implement pedestrian and personal safety. Not only should crossing the street safely be taught, assessed, and reviewed but also the importance of walking with a buddy and not using portable MP3 players set so loudly that they cannot hear oncoming traffic and other dangers. Part of the discussion should also include wearing bright reflective clothing and not wearing hoods that might block peripheral vision. Special care and instruction should be taken around daylight saving time. In the weeks following the change to daylight saving time in the fall, dusk is the most dangerous time for pedestrians. More pedestrians are struck and killed by automobiles at this time than any other time of the year.

When selecting a pedometer, simplicity is the best guide. The more features a pedometer has, the higher the cost will be. More importantly, the

more buttons a pedometer has, the more likely an individual on the spectrum will accidentally reset the device causing possible frustration which can lead to him or her giving up the exercise activity. A simple pedometer with a single reset button and a protective cover is the best option. The device should also have a clip to attach the device to one's belt or waistband and an alligator clip with a tether to avoid losing the device. Some newer-generation MP3 players have pedometers built in as an additional feature to the primary function as a music player.

The pedometers can also be used in combination with persons on the autism spectrum's interest in video games and other pieces of exercise equipment. Wii™ games and Wii Fit™ as well as Dance, Dance, Revolution™ can be used in combination with the pedometers to make exercise fun and engaging. Pedometers can also be used in conjunction with treadmills. They are less effective with elliptical machines and exercise bikes because of the nature of the motion and the manner in which pedometer record movement.

Setting up an incentive and monitoring program will be critical to helping instituting a pedometer program and incorporating walking into a healthy lifestyle. Using behavior modification, individuals should be rewarded for wearing their pedometers and for reaching distance milestones. Graphing the accomplishments and publicly displaying their achievements can instill a healthy sense of competition among participants. Translating the number of steps into mileage and reaching certain well-known geographic landmarks can spur the participants on. Frequent award ceremonies or check-ins are imperative in getting the individuals to incorporate the new behavior into their daily behavior. Incentives should be of interest to the participants, and they should be involved in their selection and the setting of the goals to earn the incentive. If possible, the incentives should also be reinforcing of the activity (e.g., "Million Step Club Member" t-shirts or baseball hats). Challenges and competitions can be issued between subgroups or teams or participants and staff members.

Hiking and backpacking are a natural outgrowth of walking as a form of exercise. Walking

in nature can be a great stress reliever among the peace and quiet of the woods (Rudy 2009) and is considered a perfect sport for children on the autism spectrum (Marinac 2010). The planning of a hike should have a goal of the hiker being able to survive 72 h in the woods in the event he or she becomes lost. Seventy-two hours are the average length of time lost hikers are found by search and rescue teams. Three-quarters of lost hikers requiring search and rescue were simply conducting day hikes with no intention of spending an overnight in the wilderness. Having plenty of water as well as means of extracting and purifying water from natural sources is an essential part of prehike planning. Trail maps, a compass, fire making, and signaling gear are also essential. Orienteering skills should be taught to the individual on the autism spectrum in order for them to be able to navigate and feel a sense of control when in the woods. Hiking can be done as an individual activity, or there are many hiking and conservation groups an individual with an ASD can join where organized trips are planned for the group. If an individual on the spectrum enjoys the outdoors, then kayaking, canoeing, and camping are other recreational activities worth investigating.

Traditional gym memberships are also an option for individuals on the autism spectrum who are interested in maintaining their physical fitness. Many offer running tracks, swimming pools, weight rooms, and organized fitness classes such as yoga, spinning, and cardio classes. For persons with ASDs, it will be important to review social etiquette rules in a gym such as comportment in the locker room, appropriate attire for the various areas of the workout facility, the importance of wiping down the equipment after use, turn taking with the exercise equipment, etc. For those individuals with ASDs who find routine important, gym facilities are excellent environments where they can establish and maintain an exercise routine. Newer-generation gym equipment even combines the use of electronics and computers. Not only can the user watch TV, listen to music, and monitor their speed and heart rates, but they can also play video games as a part of their exercise routine. Some brands of exercise

bikes (both upright and recumbent models) have a video screen where they chase a yellow jersey rider and experience riding in a peloton on a course that the rider selects. Depending on the type of course the rider selects, he or she might be rewarded by seeing a reclusive Yeti or Bigfoot character as a part of the game. With a wireless feature, riders can compete live with other exercise enthusiasts via the World Wide Web (International Fitness Holdings 2011).

If balance and coordination are not issues, then bicycle riding on the street or on a mountain bike trail is another sport that individuals on the autism spectrum could engage in. However, the person's ability to judge traffic and danger should be assessed prior to beginning this activity. Bicycle riding can not only be a great fitness activity but can also alleviate the person's need to rely upon others for transportation. Again, as long as balance is not an issue, then skateboarding or surfing may be considered as a sport for the individual or a form of exercise. With these two activities, protective gear and strong swimming ability are critical.

Martial arts have also been recommended as sports that are excellent for children on the autism spectrum. Among the reported benefits are discipline, increased concentration and memory, better balance and coordination, physical fitness, and increased self-esteem. The repeated practicing of the forms provides the individual with an ASD predictability. The added benefit of martial arts training is that the individual learns self-defense skills. Children with ASDs are often the victims of bullying and as adults are the victims of hate crimes (Sherry 2010).

Horseback riding is a sport that has been proposed not only as a physical fitness activity but as a therapeutic intervention as well. The purported benefits of horseback riding are improved circulation, muscle control, and coordination (Baker 2008). It is also an activity where the participants form bonds with the horses and see the direct benefits of their attempts to communicate with their mounts. A distinction is made between hippotherapy and equine-assisted activities or therapeutic riding. The main differences are who leads the activity and the cost. Hippotherapy is

conducted by a licensed physical or occupational therapist. Consequently, it is considerably more expensive than therapeutic riding which is led by riding instructors. However, some medical insurance may cover hippotherapy (Baker 2008) (see “North American Riding for the Handicapped Association” for a listing of accredited facilities and certified instructors).

The concept of adaptive physical education is one that is derived from the field of special education. Adaptive physical education can be written into a child’s Individualized Education Plan (IEP) under the Individuals with Disabilities Education Act (IDEA). Adaptive physical education is usually taught by someone trained in physical therapy and is used for students who have difficulties with gross motor coordination problems and other types of disabilities who would have difficulty participating in standard physical education classes. Although many individuals on the spectrum have coordination issues, persons with ASDs may benefit from the social skills training aspects of APE. In these classes, the students can be explicitly taught the social rules of team sports and the methods of verbal and nonverbal communication inherent in many games.

A slightly different concept is adaptive physical activity. “Adapted physical activity (APA) is a professional branch of kinesiology/physical education/sport & human movement sciences, which is directed toward persons who require adaptation for participation in the context of physical activity” (International Federation of Adaptive Physical Activity [IFAPA] 2011). Rather than focus upon the therapeutic aspects of working with a population, this approach to sports focuses upon access and adaptation. “In contrast to physical therapies, APA is dedicated to the concepts of empowerment and ecological validity. This means that physical activity of participants is self-driven and targeted towards mastery and excellence” (IFAPA 2011). Although originally this approach evolved from the area of physical disabilities, it is a highly individualized approach that not only involves designing and modifying equipment to maximize participation in a sport but also includes “task criteria (e.g., modifying

skill quality criteria or using a different skill), instructions (e.g., using personal supports, peer tutors, non-verbal instructions, motivational strategies), physical and social environments (e.g., increasing or decreasing court dimensions; segregated vs. inclusive; type of training climate: mastery oriented, collaborative or competitive social environment; degree of peer and parental support), & rules (e.g., double bounce rule in wheelchair tennis)” (IFAPA 2011). The philosophy of this approach is best summarized from its web site: “adapted physical activity is about individualizing instruction, matching personal strengths and interests with appropriate activities and adapting environments to promote full participation in physical activity, regardless of the population being engaged” (IFAPA 2011).

For individuals on the autism spectrum who are truly interested in team sports, the task is to find adaptive physical activities in order to participate in sports as a physical fitness activity. Adaptive leagues are being organized in team sports such as baseball, soccer, bowling, and basketball. A keyword search using “adaptive sports leagues” in an Internet browser can help identify leagues in a local area. If no adaptive leagues are available in the area, then speaking to the organizers and coaches of the local leagues may be a strategy to determine if they have a supportive attitude and philosophy of sportsmanship to include individuals on the autism spectrum.

Adaptive physical activities have been organized for team sports but for high adventure sports as well. This can include skiing, snowboarding, and even scuba diving. With skiing and snowboarding, the individual should be assessed for balance issues, and adaptations should be made accordingly. Prior to scuba diving, participants must be assessed for contraindications to scuba diving which include claustrophobia, asthma, seizure disorder, heart conditions, and potential ear problems. Scuba diving as a sport has, inherent to it, many of the same benefits as swimming but also has additional benefits. Diving teaches participants navigation skills, nonverbal communication, strong reliance on others through the buddy system, emergency management, concentration, and attention to

detail (e.g., time, depth, and air consumption). Some individuals have keen interests in marine biology, and dive trips can be organized around fish and coral identification. Others have interests in history and naval architecture. Nondive time can be spent researching shipwreck sites to explore at a later date on a scuba trip. Many individuals on the autism spectrum are fascinated with computers. The computer can be used not only to research shipwreck sites but other dive sites as well. Dive computers are wrist or console-mounted devices that help a diver keep track of depth, time, and the buildup of nitrogen in the diver's body. Finally, adaptive scuba diving can be the basis for adventure travel for individuals on the autism spectrum (Strayne 2011).

Future Directions

What sport an individual selects to maintain his or her physical fitness is immaterial. What is important is that individuals on the autism spectrum need to participate in 20–30 min of physical activity daily (just like neurotypical individuals) in order to maintain a healthy lifestyle. The risk of obesity and its negative sequelae are higher for individuals on the autism spectrum. Therefore, it is imperative that sports and other forms of exercise become a part of their daily routine. Future research should focus upon what services providers can do to help individuals on the autism spectrum develop a routine where they are engaged in physical activity including sports 20–30 min a day. From an advocacy perspective, the inclusion of individuals on the autism spectrum in sports is an ongoing effort.

See Also

- ▶ [Free Appropriate Public Education](#)
- ▶ [Inclusion](#)
- ▶ [Individuals with Disabilities Education Act \(IDEA\)](#)
- ▶ [Special Olympics](#)
- ▶ [Travel Training](#)
- ▶ [Vocational Rehabilitation Act of 1973](#)

References and Reading

- Appalachian Mountain Club (AMC). <http://www.outdoors.org/recreation/hiking/index.cfm>.
- Baker, J. (2008). Therapeutic horseback riding as a treatment for autism. Retrieved 3 June 2011, from <http://www.regardinghorses.com/2008/03/11/therapeutic-horseback-riding-as-autism-treatment/>.
- Besser, L. M., & Dannenberg, A. L. (2005). Walking to public transit: Steps to help meet physical fitness recommendations. *American Journal of Preventive Medicine*, 29(4), 273–280. Retrieved 8 June 2011, from http://www.cdc.gov/healthypaces/publications/besser_dannenberg.pdf.
- Blair, S. N., & Brodney, S. (1999). Effects of physical inactivity and obesity on morbidity and mortality: Current evidence and research issues. *Medicine and Science in Sports and Exercise*, 31(Suppl 11), S646–S662. Retrieved 6 June 2011, from <http://www.ncbi.nlm.nih.gov/pubmed?term=Blair%20%26%20Brodney%2C%201999>.
- Center for Disease Control, CDC. (2010). Vital signs: State specific obesity prevalence rates among adults -United States, 2009. Retrieved 6 June 2011, from <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm59e0803a1.htm>.
- Center for Disease Control, CDC. (2011a). Bullying among middle and high school students—Massachusetts, 2009. *Morbidity and Mortality Weekly*, 60(15), 465–471. Retrieved 6 June 2011, from http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6015a1.htm?s_cid=mm6015a1_w.
- Center for Disease Control, CDC. (2011b). *Fast facts: Average life expectancy*. Retrieved 6 June 2011, from <http://www.cdc.gov/nchs/fastats/lifexp.htm>.
- Curtin, C., Anderson, S. E., Must, A., & Bandini, L. (2010). The prevalence of obesity in children with autism: A secondary data analysis using nationally representative data from the National Survey of Children's health. *BMC Pediatrics*, 23(10), 11. Retrieved 7 June 2011, from <http://www.ncbi.nlm.nih.gov/pubmed/20178579>.
- Elliott, R. O., Dobbin, A. R., Rose, G. D., & Soper, H. V. (1994). Vigorous, aerobic exercise versus general motor training activities: Effects on maladaptive and stereotypic behaviors of adults with both autism and mental retardation. *Journal of Autism and Developmental Disorders*, 24(5), 565–576. Retrieved 7 June 2011, from <http://www.ncbi.nlm.nih.gov/pubmed/7814306>.
- Gillberg, C., Billstedt, E., Sundh, V., & Gillberg, I. C. (2010). Mortality in autism: A prospective longitudinal community-based study. *Journal of Autism and Developmental Disorders*, 40, 352–357.
- International Federation of Adaptive Physical Activity, IFAPA. (2011). *APA definition and philosophy*. Retrieved 3 June 2011, from <http://www.ifapa.biz/?q=node/7>.
- International Fitness Holdings. (2011). *Expresso Live! Advanced interactive experience for riders*. Retrieved

- 4 June 2011, from http://www.expresso.com/products_services/expresso_live.html.
- International Paralympic Committee. (2011). *Paralympics*. Retrieved 5 June 2011, from http://www.paralympic.org/Paralympic_Games/.
- Lawson, W. (2001). *Understanding and working with the spectrum of autism: An insider's view*. Philadelphia/London: Jessica Kingsley Publishers.
- Marinac, M. K. (2010). *Hiking with autism*. Retrieved 2 June 2011, from http://www.spectrumpublications.com/index.php/myspectrum/hiking_with_autism/.
- North American Riding For the Handicapped Association. <http://www.ncpad.org/organizations/index.php?id=591&state=Colorado&city=Denver>.
- Outdoor Explorations. <http://www.outdoorexplorations.org>, sponsors a few family hikes and backpacking training programs for disabled individuals ages 8 and older, with younger kids eligible for family camp programs.
- Paffenbarger, R. S., Hyde, R. T., Wing, A. L., & Hsieh, C. C. (1986). Physical activity, all-cause mortality, and longevity of college alumni. *New England Journal of Medicine*, 314(10), 605–613. Retrieved 6 June 2011, from PubMed <http://www.ncbi.nlm.nih.gov/pubmed/3945246?dopt=Abstract>.
- Pangrazi, R. P., Beighle, A., & Sidman, C. L. (2007). *Pedometer power: Using pedometers in school and community* (2nd ed.). Champagne: Human Kinetics.
- Rudy, L. J. (2009). *Best sports for kids with autism*. Retrieved 2 June 2011, from <http://autism.about.com/od/childrenandautism/p/sportsideas.htm>.
- Rundle, A. (2005). Molecular epidemiology of physical activity and cancer. *Cancer Epidemiology, Biomarkers & Prevention*, 14(1), 227–236.
- Rundle, A., Richie, J., Steindorf, K., Peluso, M., Overad, K., Raaschou-Nielsen, O., et al. (2010). Physical activity and lung cancer among non-smokers: A pilot molecular epidemiological study within EPIC. *Biomarkers*, 15(1), 20–30. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/20050820>.
- Shavelle, R. M., Strauss, D. J., & Pickett, J. (2001). Causes of death in autism. *Journal of Autism and Developmental Disorders*, 31(6), 569–576.
- Sherry, M. D. (2010). *Disability hate crimes: Does anyone really hate disabled people*. London: Ashgate Publishing.
- Special Olympics. (2011). The history of the Special Olympics. Retrieved 4 June 2011, from <http://www.specialolympics.org/history.aspx>.
- Steward, S. T., Culter, D. M., & Rosen, A. B. (2009). Forecasting the effects of obesity and smoking on U.S. life expectancy. *New England Journal of Medicine*, 361(23), 2252–2260. Retrieved 6 June 2011, from PubMed: <http://www.ncbi.nlm.nih.gov/pubmed/19955525>.
- Strayne, J. (2011). Diveheart training in Chico, CA. Retrieved 4 June 2011, from http://diveheart.org/index.php?option=com_content%26view=article%26id=329:diveheart-training-in-chico-ca%26catid=28:diveheart-events%26Itemid=44.
- VanBergeijk, E. O. (2009). Incorporating exercise in a healthy lifestyle for adults on the autism spectrum. *Autism Spectrum News*, 2(1), 30, 52.
- Weimer, A. K., Schatz, A. M., Lincoln, A., Ballantyne, A. O., & Trauner, D. A. (2001). “Motor” impairment in Asperger syndrome: Evidence for a deficit in proprioception. *Developmental and Behavioral Pediatrics*, 22(2), 92–101.

SRS

► Social Responsiveness Scale

SSIS SEL

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Synonyms

Social skills; social emotional skills; social-emotional learning skills

Description

The *Social Skills Improvement System Social-Emotional Learning Edition* (SSIS SEL Edition; Gresham and Elliott 2017) Rating Forms assist professionals in screening and classifying students aged 3–18 years suspected of having significant social emotional learning (SEL) skill deficits. The SSIS Classwide Intervention Program (CIP) is a manualized and fully digitized online treatment programs for improving 23 social emotional skills identified by low frequency ratings on items on one or more Rating Forms. The SSIS uses a multirater (parents, teachers, and students with at least third-grade reading ability) approach that provides a comprehensive

examination of five SEL competency areas (self-awareness, self-management, social awareness, relationship skills, and responsible decision making) and an overall academic competence scale. The instrument is available online or in paper version and yields norm-referenced scores based on a national sample of children representative of the 2006 US Census.

Given that autism is characterized by impaired social interactions, problems with verbal and nonverbal communication, and repetitive/stereotyped behaviors, the SSIS Rating Forms and related CIP intervention manual offer professionals a comprehensive assessment of key SEL skills and an aligned intervention program to specifically address skills identified as relative weaknesses. The SSIS SEL Edition, unlikely the original SSIS Rating Scales (Gresham and Elliott 2008), does not have a separate set of Problem Behavior scales. Thus, autism spectrum disorder (ASD) behaviors are not directly measured on the SEL Edition Rating Forms, but many SEL behaviors commonly targeted for intervention with children with ASD are assessed and aligned with CIP skill units.

Historical Background

The SSiS Rating Scale originally was published in 1990 as the *Social Skills Rating System* (SSRS; Gresham and Elliott 1990). The SSRS was reportedly used by many professionals as part of their autism screening efforts; however, the SSRS was not designed with autism or autism spectrum disorders in mind. When the revision of the SSiS was being conceptualized, the authors consult with educators serving autistic children and the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., 1994) of the American Psychiatric Association and the National Research Council's report (2001) on *Educating Children with Autism* to ensure they were sensitive to critical identifying behaviors and conditions.

The Collaborative for Academic, Social, and Emotional Learning (CASEL 2013) SEL Framework that features five competency domains

(listed above) inspired the new *SSIS SEL Edition Rating Forms* (SSIS SEL RF) and the *SSIS SEL Edition Screening and Progress Monitoring Scales* (Elliott and Gresham 2017a, b), both published in 2017.

Psychometric Data

The SSIS SEL-RF is comprised of the same 46 social behaviors as the original SSIS Rating Scales, along with five internalizing behaviors (reverse scored). These 51 items, based on a confirmatory factor analysis, represent the five SEL competency domains theorized in the CASEL SEL competency framework. The SSIS SEL Edition Manual provides extensive validity evidence based on test content, internal structure, intercorrelations among scales and subscales, item-total correlations, and relations with other variables (Gresham and Elliott 2017).

Given the restructuring of the SSIS SEL-RF items/scales as compared to the SSIS-RS, modifications of specific subscales prevent direct comparisons; however, total scale internal consistency estimates across forms were comparable between the SSIS SEL-RF and SSIS-RS. Specifically, subscale coefficient alphas on the SSIS SEL-RF ranged from 0.69 (Self-Awareness, Spanish Parent Version) to 0.97 (several subscales across forms), while the lowest alpha for a Composite scale was 0.94. In general, the Self-Awareness subscale yielded slightly lower coefficient alphas than the Self-control subscale, its corresponding subscale on the SSIS-RS. These results are especially apparent on the Parent English and Spanish forms. The internal consistency coefficients for the Responsible Decision Making scale largely equal or exceed those of its related SSIS-RS subscales of Assertion and Responsibility.

A comparison of *teacher ratings* between the SSIS SEL-RF and SSIS-RS forms revealed high levels of agreement across composite and subscale scores ($p < 0.05$). The SSIS SEL-RF Composite ($r = 0.98$) and Core Skills ($r = 0.92$) scales strongly correlated with the SSIS-RS Social Skills scale and demonstrated moderate to high

correlations with the corresponding subscales. Furthermore, significant negative relationships were revealed between teacher ratings on the SSIS SEL Composite and Core Skills scores with the SSIS-RS Problem Behaviors scale and subscale scores, providing evidence for divergent validity of the negative relationship between social emotional skills and problem behaviors.

Similar to the comparisons of teacher ratings, *parent ratings* on the SEL composite scale, Core Skill scale, and related subscales yielded high correlations across the SSIS-RS Social Skills scale and subscales ($p < 0.05$). Concerning divergent validity, all SSIS SEL-RF parent ratings negatively correlated with the SSIS-RS Problem Behavior composite scale and subscales, providing further evidence for differences between positive social and emotional skills as measured by the SSIS SEL and problem behaviors.

Correlations between student ratings on the SSIS SEL-RF and SSIS-RS also revealed strong positive relationships between all composite scales and subscales ($p < 0.05$). These results provide support for the convergent validity between the two forms. Furthermore, ratings between the SSIS SEL-RF skills and the SSIS-RS Problem Behavior composite scale and subscales demonstrated strong negative relationships, presenting additional evidence for divergent validity. An exception to these results was the Internalizing subscale on the SSIS-RS, which did not produce any correlations higher than 0.18 with the SSIS SEL-RF scales. Additionally, the relationship between Social Awareness and Hyperactivity/Impulsivity ratings was also weak ($r = 0.16$).

Correlations between the SSIS-RS and SSIS SEL Edition with the *Behavioral Assessment System for Children*, Second Edition (BASC-2; Reynolds and Kamphaus 2004) are moderate to high, depending on the scales and subscales. Correlations between the SSIS-RF total social skills scores and the socialization scores of the *Vineland Adaptive Behavior Scales*, Second Edition (Vineland II; Sparrow et al. 2005) are also moderate to high for the teacher and parent forms, respectively.

Using a sample of 50 students with an autism spectrum disorder as diagnosed by the DSM-IV-TR, a known group comparison analysis with a nonclinical sample was conducted. On the Teacher and Parent forms of the SSIS, all the mean score differences were statistically significant. The SEL scale means at least 1.5 standard deviations lower than the nonclinical reference sample of students. These results are consistent with expectations that individuals with autism spectrum disorder have major deficiencies in social emotional skills. Thus, lending substantial support for the validity of the scores of this subscale as a measure of behaviors typically exhibited by individuals diagnosed with autism.

Clinical Uses

The SSIS Rating Forms, comprised of the Teacher, Parent, and Student rating forms, are intended to be used as comprehensive measures of children's social emotional skills. In particular, these rating scales are designed to identify social emotional skill acquisition and performance deficits in areas of self-awareness, self-management, social awareness, relationship skills, and responsible decision making. By using the nationally representative norming sample for comparison, it is possible to get a clear comparison between a student suspected of an autism spectrum disorder to a same age and sex comparison group of non-clinical students. The assessment results can be used to identify specific skill deficits that link directly to SSIS CIP intervention skill units, where SEL skills are directly taught via a manualized treatment supported with rich online resources designed to engage students in the process of skill development and generalization.

References and Reading

- Elliott, S. N., & Gresham, F. M. (2017a). *SSIS SEL Classwide intervention program*. Bloomington, MN: Pearson Assessments.

- Elliott, S. N., & Gresham, F. M. (2017b). *SSIS SEL edition screening and progress monitoring scales*. Bloomington, MN: Pearson Assessments.
- Collaborative for Academic, Social, and Emotional Learning. (2012). *2013 CASEL guide: Effective social and emotional learning programs—Preschool and elementary school edition*. Chicago, IL: Author.
- Gresham, F. M., & Elliott, S. N. (1990). *Social skills rating system*.
- Gresham, F. M., & Elliott, S. N. (2008). *Social skills improvement system rating scales manual*. Bloomington, MN: NCS Pearson Inc.
- Gresham, F. M., & Elliott, S. N. (2017). *SSIS SEL edition rating forms manual*. Bloomington, MN: Pearson Assessments.
- Reynolds, C. R., & Kamphaus, R. W. (2004). *Behavior assessment system for children manual* (2nd edn). Bloomington, MN: NCS Pearson Inc.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales: Parent/caregiver rating form* (2nd ed.). Bloomington, MN: NCS Pearson Inc.

SSP

- ▶ [Short Sensory Profile in Autism](#)

SSP-2

- ▶ [Short Sensory Profile in Autism](#)

SSQ-R

- ▶ [Sensory Sensitivity Questionnaire: Revised](#)

Staff Training and Development

- ▶ [Feedback on Provider Work Performance](#)

Stalking

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Definition

Stalking, also referred to in the extant literature as obsessional following, obsessional harassment, and obsessive relational intrusion (Meloy 2001; Rosenfeld and Harmon 2002; Spitzberg and Cupach 2007), consists of a continuum of behaviors involving harassment and threats over time that serve no purpose other than to cause fear in the victim and distress over their safety (Sheridan and Davies 2004).

Historical Background

The Stalking Risk Profile (MacKenzie et al. 2012) categorizes typologies of stalking representing various motivations and precipitating factors.

Rejected stalking results from the dissolution of a close relationship such as former intimate partners. Often the rejected stalker is trying to repair the relationship and with it their sense of self-worth but when this proves fruitless, revenge may be the focus of their behavior.

Resentful stalking occurs when the stalker perceives they have been disrespected, humiliated, or have received unfair treatment. The victims may or may not know the perpetrator but are perceived to have committed the slight or injustice. The genesis of this type of stalking is revenge which can be maintained by the power they feel through demonstrations of the victims' fear. When this type of stalking is directed to an organization, the stalker may feel like they are standing up for themselves against the oppressors.

Predatory stalking typically involves male perpetrators and female victims unknown to the perpetrator but with whom he has developed a

sexual interest. The behaviors can range from voyeurism resulting in sexual gratification to using the stalking as a means of amassing information with the ultimate goal of sexual assault.

Intimacy-seeking stalking results from social isolation and a dearth of emotionally/physically intimate relationships. Intimacy seekers often target a stranger, celebrity, or casual acquaintance and develop delusional beliefs that they are having a relationship with their victim.

Similar to the intimacy seeker, **the incompetent suitor** operates from a position of loneliness though their motivations may be primarily focused on establishing sexual intimacy. The duration of their stalking is typically less than other typologies and is characterized by a mindblindness to the anguish of their victims. This lack of perspective taking is often associated with perpetrators with autism spectrum disorder (ASD).

For the purpose of this chapter, we will focus on **the incompetent suitor** typology and attempt to explain how the characteristics of ASD, in particular social challenges, perspective taking, circumscribed interests, and a strong need for routines and rituals, may be implicated in the commission of a stalking offense.

Current Knowledge

Risk Factors

Desire Versus Skillset to Initiate and Maintain Intimate Relationships

Contrary to popular belief, many people with ASD desire intimate relationships. However, in assessments of their knowledge of sexuality, they score below their typical peers in all areas except body part identification and knowledge of menstruation (McCabe and Cummins 1996). They often have a paucity of vocabulary to describe relationships or identify what makes someone an appropriate target for a romantic relationship. Moreover, for a group of people who have often been the targets of bullying and harassment (Little 2002), small gestures of kindness may be interpreted as romantic overtures. When

asked what makes someone your girlfriend, a man with ASD serving a lengthy prison sentence for stalking reported “You just know.” When plumbed for further information he reported, “She was nice to me.” This juxtaposition of the desire for intimacy with the challenges in understanding what constitutes an intimate relationship may lead to trouble and even criminal charges.

Victim as Circumscribed Interest

Evidence would suggest that the majority of individuals with ASD have circumscribed interests, manifested as part of their experience of their ASD (Klin et al. 2007), and that these circumscribed interests occupy their attention longer and will prevent exploration of other pursuits (Sasson et al. 2008). It has been found that circumscribed interests may become more intense for those with ASD (Anthony et al. 2013) or involve greater emotional attraction (Sasson et al. 2012) than interests do for typically developing (TD) individuals.

As individuals with ASD approach adolescence, they manifest desire to have normal sexual and romantic relations (Dewinter et al. 2015; Stokes and Kaur 2005), and yet, while normal sexual experiences can occur (Dewinter et al. 2015; Ginevra et al. 2015), compared to TD individuals, those with ASD more frequently fail to obtain lasting relationships (Dewinter et al. 2017; Stokes et al. 2007). Moreover, as a group these individuals have reduced sexual knowledge (Ginerva et al. 2016; Stokes and Kaur 2005), which has been associated with sexual offending among a minority of this group (Baarsma et al. 2016).

Few studies have examined offending behaviors, let alone stalking, among those with ASD. One, Stokes et al. (2007), examined 25 individuals with ASD found that this group reported a willingness to pursue strangers, ex partners (where they had these), and celebrities considerably more so than TD individuals and that they would pursue the victim longer if the rejection of this was made explicit. Indeed, individuals with ASD are noted for perseveration in the face of error (cf. Landry and Al-Taie 2016). This is akin to persisting and trying again using the same failed

strategy as was first used seeking the possibility that with practice it may work. Broadbent and Stokes (2013) explored whether the unwanted pursuit might be perversely encouraged by active rejection. They undertook a modified Wisconsin Card Sort Task with 50 individuals with high functioning ASD and 50 TD individuals, where in one condition negative reinforcement was removed. It was found that negative reinforcement trapped individuals with ASD, so that instead of modifying their strategies, those with ASD persisted longer in the face of negative reinforcement yet performed significantly better when offered only positive reinforcement for correct choices. These results suggest the victim may become a circumscribed interest for those with an ASD. Fortunately, recently published results suggest that individuals with ASD can learn strategies to reduce their inappropriate behavior and focus upon a person (Dekker et al. 2015).

Stalking as the Routine/Ritual

People with ASD often have a strong need for routines and rituals (Bishop et al. 2013). When a routine or ritual is disrupted, they often have a need to restart the ritual from the beginning. The daily calls, obsessional following, gift giving, showing up at the victim's place of employment, and other behaviors that constitute obsessional harassment may have become part of the person's routine. This completist mentality, "I have to start from the beginning until I get it right," may lead to criminal stalking, probation or parole violations relative to restraining orders, or even instances of recidivism.

Difficulties with Perspective Taking

Ironically, stalking is largely predicated on the perception of the victim (Ngo and Paternoster 2016) and, however, may be perpetrated due to lack of perspective taking on the part of the person with ASD. From the perspective of the victim, they may think they are simply being kind to a person who appears to have few friends while the person with ASD may assume that this is a romantic gesture. Faced with increasingly uncomfortable attention, the victim may begin to ignore the person, thinking they will leave them alone. This

can be interpreted by the person with ASD as a need to work harder to "win the person back." The victim may then believe that if they speak to the person one more time, and explain themselves, they will be left alone. Unwittingly, they have engaged in intermittent reinforcement of the problem behavior (stalking), which builds the most durable behaviors (Skinner 1954; Broadbent and Stokes 2013). The person with ASD is now faced with a lack of access to their romantic target so they may begin to seek out other places to see the person. This escalation could take the form of driving down the person's street, walking past their home, and showing up at their place of work. Both parties may arrive at the same emotional state when the police are called – terrified and wondering why this is happening to them.

Time Spent Online

People with ASD spend an inordinate amount of time online relative to the time they spend pursuing other leisure activities (Mazurek and Wenstrup 2013). Cyberstalking in the form of obsessional following and harassment may result from the person's proclivity toward online activities and the ease with which they can access personal information about their intended target. Moreover, cyberstalking may be easier than off-line stalking as it can be done from a home location and does involve the degree or social interaction, transportation, or community access skills required for off-line stalking.

Media Influences

The media's influence on romantic knowledge is important. Many individuals consume a considerable amount of media (Nielsen 2014), and it has been found that individuals with ASD consume more (Mazurek and Wenstrup 2013) and are less discerning of this than are TD individuals (Kuo et al. 2015). Those with ASD have been found to be 2.3 times more likely to learn their romantic knowledge from the media than were TD individuals (Stokes et al. 2007). If this source of learning was the entirety of this issue, it would be simple to address. However, part of the trouble is that individuals with ASD tend to take things at face value, misinterpreting the kindness of others as

indicating romantic interest (Attwood 2007). Viewers without autism likely recognize the hyperbole in the popular media when it comes to the glorification of stalking characterized by romantic tenacity that results in obtaining the affections of the object of their desire (Spitzberg and Cadiz 2002). Media images, taken at face value, may become a script, albeit inappropriate, that the person with ASD utilizes in their romantic pursuits believing that this behavior will result in garnering the attention of their intimate target. It is considered likely that impaired theory of mind function may contribute to this phenomenon. It is unsurprising that this misinterpretation will lead to rejection, and while the majority of individuals with ASD do not involve themselves in vengeful violence against others for this rejection, violent vengeful sexual fantasies have been reported in a small number of individuals with ASD (Palermo and Bogaerts 2015). More generally, those with ASD may utilize strategies to initiate romantic contact that would be unusual in their intensity compared to that undertaken by TD individuals. These may include touching, pursuing, and the making of threats (Stokes et al. 2007). The consequence is that the target person may reasonably seek to inform their pursuer that they are not interested, and this then leads the individuals with ASD to become trapped into trying harder to improve their technique in the forlorn hope that their target will recognize the suitability of them as a partner (Broadbent and Stokes 2013).

Future Directions

Individuals with ASD need greater and fuller understanding of how to interact in the complex world of human relationships. It would be ideal to suggest that the TD world could adjust itself to better accommodate those with ASD. However, this is unlikely. Interpreting others' intentions in romantic relationships is fraught with error among TD individuals (de Quadros-Wander and Stokes 2007); it is unlikely that the TD population will modify their evolutionarily derived behaviors to accommodate those with ASD. Therefore, changes in understanding are required among

those with ASD. To this end Dekker et al. (2015) have evolved a promising program of cognitive interventions that appears to assist those with ASD to adjust their thinking and behavior. Additionally, to date, the extent to which romantic rejection is associated with well-being failure in those with ASD has yet to be elucidated, as has their responses to this. Unfortunately, individuals with ASD typically do not receive this type of education until after they have engaged in a behavior that has reached the level of criminality (Griffiths et al. 2002). They may not be aware of the illegality of their behavior nor are they aware of the consequences. Proactive intimacy education should be specifically focused on increasing their understanding of relationship vocabulary and identification of appropriate targets for romantic pursuits, with a focus on developing appropriate replacement behaviors when rejection does occur (Gerhardt 2009). Targeted skill development can provide individuals with ASD with the necessary social tools to pursue the intimate relationships they so often desire.

See Also

- ▶ Sexuality and Problem Behaviors
- ▶ Violence and ASD

References and Reading

- Anthony, L. G., Kenworthy, L., Yerys, B. E., Jankowski, K. F., James, J. D., Harms, M. B., Martin, A., & Wallace, G. L. (2013). Interests in high-functioning autism are more intense, interfering, and idiosyncratic than those in neurotypical development. *Development and Psychopathology*, 25(3), 643–652. doi:10.1017/S0954579413000072.
- Attwood, T. (2007). *The complete guide to Asperger's syndrome*. London: Jessica Kingsley Publishers.
- Baarsma, M. E., Boonmann, C., 't Hart-Kerkhoffs, L. A., de Graaf, H., Doreleijers, T. A. H., Vermeiren, R. R. J. M., & Jansen, L. M. C. (2016). Sexuality and autistic-like symptoms in juvenile sex offenders: A follow-up after 8 years. *Journal of Autism and Developmental Disorders*, 46(8), 2679–2691. doi:10.1007/s10803-016-2805-6.
- Bishop, S. L., Hus, V., Duncan, A., Huerta, M., Gotham, K., Pickles, A., ..., Lord, C. (2013). Subcategories of

- restricted and repetitive behaviors in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(6), 1287–1297.
- Broadbent, J., & Stokes, M. (2013). Removal of negative feedback enhances WCST performance for individuals with Asperger's Syndrome. *Research in Autism Spectrum Disorders*, 7(6), 785–792.
- de Quadros-Wander, S., & Stokes, M. (2007). The effect of mood on opposite-sex judgments of males' commitment and females' sexual intent. *Evolutionary Psychology*, 5(3), 453–475.
- Dekker, L. P., van der Vegt, E. J., Visser, K., Tick, N., Boudestein, F., Verhulst, F. C., Maras, A., & Greaves-Lord, K. (2015). Improving psychosexual knowledge in adolescents with autism spectrum disorder: Pilot of the tackling teenage training program. *Journal of Autism and Developmental Disorders*, 45(6), 1532–1540. doi:10.1007/s10803-014-2301-9.
- Dewinter, J., Vermeiren, R., Vanwesenbeeck, I., Lobbestael, J., & Van Nieuwenhuizen, C. (2015). Sexuality in adolescent boys with autism spectrum disorder: Self-reported behaviours and attitudes. *Journal of Autism and Developmental Disorders*, 45(3), 731–741. doi:10.1007/s10803-014-2226-3.
- Dewinter, J., Van Parys, H., Vermeiren, R., & van Nieuwenhuizen, C. (2017). Adolescent boys with an autism spectrum disorder and their experience of sexuality: An interpretative phenomenological analysis. *Autism*, 21(1), 75–82.
- Ginevra, M. C., Nota, L., & Stokes, M. A. (2015). The differential effects of Autism and Down's syndrome upon sexual behavior. *Autism Research*, 9(1), 131–140. doi:10.1002/aur.1504.
- Klin, A., Danovitch, J. H., Merz, A. B., & Volkmar, F. R. (2007). Circumscribed interests in higher functioning individuals with autism spectrum disorders. *Research and Practice for Persons with Severe Disabilities*, 32(2), 89–100.
- Kuo, M. H., Magill-Evans, J., & Zwaigenbaum, L. (2015). Parental mediation of television viewing and video-gaming of adolescents with autism spectrum disorder and their siblings. *Autism*, 19(6), 724–735. doi:10.1177/1362361314552199.
- Landry, O., & Al-Taie, S. (2016). A Meta-analysis of the Wisconsin Card Sort Task in Autism. *Journal of Autism and Developmental Disorders*, 46(4), 1220–1235. doi:10.1007/s10803-015-2659-3.
- Little, L. (2002). Middle-class mothers' perceptions of peer and sibling victimization among children with Asperger's syndrome and nonverbal learning disorders. *Issues in Comprehensive Pediatric Nursing*, 25(1), 43–57.
- MacKenzie, R. D., McEway, T. E., Pathe, M. T., James, D. V., Ogloff, J. R. P., & Mullen, P. E. (2012). *Stalking risk profile: Guidelines for the assessment and management of stalkers*. Cambridge, MA: Meloy. <https://www.stalkingriskprofile.com/stalking-risk-profile>
- Mazurek, M. O., & Wenstrup, C. (2013). Television, video game and social media use among children with ASD and typically developing siblings. *Journal of Autism and Developmental Disorders*, 43(6), 1258–1271. doi:10.1007/s10803-012-1659-9.
- McCabe, M. P., & Cummins, R. A. (1996). The sexual knowledge, experience, feelings and needs of people with mild intellectual disability. *Education and Training in Mental Retardation and Developmental Disabilities*, 31, 13–21.
- Meloy, J. R. (2001). *The psychology of stalking: Clinical and forensic perspectives*. Academic Press. Cambridge, MA
- Ngo, F. T., & Paternoster, R. (2016). Toward an understanding of the emotional and behavioral reactions to stalking: A partial test of general strain theory. *Crime and Delinquency*, 62(6), 703–727.
- Nielsen Co. (2014). Shifts in viewing. The Cross-Platform Report Q2 2014.
- Palermo, M. T., & Bogaerts, S. (2015). Violent fantasies in young men with autism spectrum disorders: Dangerous or miserable misfits? Duty to protect whom? *International Journal of Offender Therapy and Comparative Criminology*, 1–16. doi:10.1177/0306624X15612719. Published online on 28 Oct 2015.
- Post, M., Haymes, L., Storey, K., Loughrey, T., & Campbell, C. (2012). Understanding stalking behaviors by individuals with autism spectrum disorders and recommended prevention strategies for school settings. *Journal of Autism and Developmental Disabilities*, 49(1), 1–9.
- Post, M., Storey, K., Haymes, L., Campbell, C., & Loughrey, T. (2014). Stalking behaviors by individuals with autism spectrum disorders in employment settings: Understanding stalking behavior and developing appropriate supports. *Education and Training in Autism and Developmental Disabilities*, 49(1), 102–110.
- Rosenfeld, B., & Harmon, R. (2002). Factors associated with violence in stalking and obsessional harassment cases. *Criminal Justice and Behavior*, 29(6), 671–691.
- Sasson, N. J., Turner-Brown, L. M., Holtzclaw, T. N., Lam, K. S. L., & Bodfish, J. W. (2008). Children with autism demonstrate circumscribed attention during passive viewing of complex social and nonsocial picture arrays. *Autism Research*, 1(1), 31–42. doi:10.1002/aur.4.
- Sasson, N. J., Dichter, G. S., & Bodfish, J. W. (2012). Affective responses by adults with autism are reduced to social images but elevated to images related to circumscribed interests. *PloS One*, 7(8), e42457. doi:10.1371/journal.pone.0042457.
- Sheridan, L., & Davies, G. (2004). Stalking. In J. R. Adler (Ed.), *Forensic psychology: Concepts, debates and practice* (pp. 197–216). Cullompton: Willan.
- Skinner, B. F. (1954). *The science of learning and the art of teaching* (pp. 99–113). Cambridge, MA.
- Spitzberg, B. H., & Cadiz, M. (2002). The media construction of stalking stereotypes. *Journal of Criminal Justice and Popular Culture*, 9(3), 128–149.
- Spitzberg, B. H., & Cupach, W. R. (2007). The state of the art of stalking: Taking stock of the emerging literature. *Aggression and Violent Behaviour*, 12, 64–86.

- Stalking Resource Center. (2015). *Stalking information*. National Center for Victims of Crime. Retrieved from <http://www.victimsofcrime.org/our-programs/stalking-resource-center/stalking-information/the-use-of-technology-to-stalk#use>
- Stokes, M., & Kaur, A. (2005). High functioning autism and sexuality: A parental perspective. *Autism*, 9(3), 263–287.
- Stokes, M., Newton, N., & Kaur, A. (2007). Stalking, and social and romantic functioning among of adolescents and adults with autistic spectrum disorder. *Journal of Autism and Developmental Disorders*, 37(10), 1969–1986.
- WHOA. (2014). *Online harassment/cyberstalking statistics*. Retrieved from <http://www.haltabuse.org/resources/stats/index.shtml>

Stammering

- [Fluency and Fluency Disorders](#)

Standard

- [Criterion](#)

Standard Deviation (SD)

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Definition

The standard deviation is a measurement of the amount of variance a data set has from its mean. The larger the standard deviation the greater the variance; the smaller the standard deviation the less variance exists. The standard deviation is represented by the Greek letter sigma σ .

The standard deviation is calculated by finding the square root of the variance or, in other words, finding the square root of the average of the squared deviations of each number from the mean of all the numbers.

See Also

- [Standard Error of the Mean](#)

References and Reading

- Norman, G., & Streiner, D. (2008). *Describing the data with numbers, measures of central tendency and dispersion* (Biostatistics the bare essentials) (Vol. III, pp. 19–29). Shelton: People's Medical Publishing House.
- Rosner, B. (2010). *Descriptive statistics* (Fundamentals of biostatistics) (Vol. VII, pp. 5–31). Boston: Brooks/Cole.
- Weisstein, E., (2011). Standard Deviation. *MathWorld – A Wolfram Web Resource*. Retrieved May 16, 2011, from <http://mathworld.wolfram.com/StandardDeviation.html>

Standard Error of Measurement

- [Measurement Error](#)

Standard Error of the Mean

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Definition

Standard Error of the Mean refers to the error associated with estimating a mean or average score of a population, or a sample (N), where N refers to the Number of subjects.

It is defined mathematically as the standard deviation (S.D.) divided by the square root of N . The logic behind this simple formula is evidenced by the quick realization that the larger N becomes, the smaller the absolute error in estimating the true or population mean.

This same logic is used in polling undertaken in political races. When pollsters claim that the margin of error is, say, plus or minus three points and one candidate shows a lead of only 2% points, they say further that such a result must be interpreted with caution because it is “within the margin of error.” The margin of error is, in turn, based upon the size of the standard error of the mean, such that the larger the standard error, the less accurate the polling results, and vice versa.

Let us now take an example of the usefulness of knowing the standard error of the mean in understanding a hypothetical level of clinician agreement in diagnosing a given child as one of the following: non-autistic; on the autism spectrum, but not meeting full criteria for autism; or meeting current DSM criteria for autism. Suppose that using the Weighted Kappa statistic (Cohen 1968), with the corrected standard error due to Cicchetti and Fleiss (1977) and Fleiss et al. (1969), we discover the following: The overall Percentage of Observed (PO) agreement for our two well-trained clinicians is 94%. Suppose further that the Percentage of Expected (PE) agreement turns out to be 60%. Weighted Kappa is defined as the difference between observed and expected agreement (PO – PE) divided by the maximum difference from expected agreement that is possible (100 – PE).

This translates into : Weighted Kappa–

$$\begin{aligned} &= (\text{PO} - \text{PE}) / (100 - \text{PE}) \\ &= (94 - 60) / (100 - 60) \\ &= .85 \end{aligned}$$

The level of statistical significance of a given Weighted Kappa value is determined by dividing the size of Weighted Kappa by its Standard error of the mean (S.E.). This produces a *z* score such that a value of 1.96 or higher is statistically significant at the .05 level of probability. Suppose our S.E. based upon 250 cases is .425. This produces a *z* of 2.00, which is indeed significant at the .05 level.

But how do we interpret the Weighted Kappa value of .85, for level of clinical or practical significance?

By the criteria of Cicchetti (1994), Cicchetti and Sparrow (1981), and Fleiss et al. (2003), the Weighted Kappa value can be considered Excellent, and by the earlier criteria of Landis and Koch (1977), a Weighted Kappa value of .85 can be considered to be almost perfect.

References and Reading

- Cicchetti, D. V. (1994). Guidelines, criteria, and rules of thumb for evaluating normed and standardized instruments in psychology. *Psychological Assessment*, 6, 284–290.
- Cicchetti, D. V., & Fleiss, J. L. (1977). Comparison of the null distributions of weighted kappa and the C ordinal statistic. *Applied Psychological Measurement*, 1, 195–201.
- Cicchetti, D. V., & Sparrow, S. S. (1981). Developing criteria for establishing inter-rater reliability of specific items: Applications to assessment of adaptive behavior. *American Journal of Mental Deficiency*, 86, 127–137.
- Cohen, J. (1968). Weighted kappa: Nominal scale agreement with provision for scaled disagreement or partial credit. *Psychological Bulletin*, 70, 213–220.
- Fleiss, J. L., Cohen, J., & Everitt, B. S. (1969). Large sample standard errors of kappa and weighted kappa. *Psychological Bulletin*, 72, 323–327.
- Fleiss, J. L., Levin, B., & Paik, M. C. (2003). *Statistical methods for rates and proportions* (3rd ed.). New York: Wiley.
- Landis, J. R., & Koch, G. G. (1977). The measurement of agreement for categorical data. *Biometrics*, 33, 159–174.

Standard Normal Distribution

► Normal Curve

Standard Score

► Z Scores

Standard Scores (Z and T Scores)

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Definition

Standard Scores are raw scores that, for ease of interpretation, are converted to a common scale of measurement, or z distribution, with a mean or average value of 0 and a standard deviation of 1. When sample sizes, or *N*s, are small, say less than 200, standard scores are interpreted as t scores. The simple formula for calculating a z score is: $Z = (X - \bar{X}) / S.D.$ in which: *X* = the aforementioned raw score $\bar{X}$ = the converted average or mean score of the population under investigation *S.D.* = the standard deviation of that population When the sample size is large, a z value of + or -1.96 so-called standard deviation units is statistically significant at the conventional 5% level of statistical significance. Thus, if the difference in average adaptive behavior scores of a large group of Asperger individuals and a large group of high functioning autistic persons resulted in a z value of 2.06, we would conclude, correctly, that the high functioning group had significantly higher overall adaptive behavior scores. However, if we now inspected the average scores of the two groups and discovered that they were standard scores of 104 and 100, respectively, we would likely conclude that although the difference was statistically significant, it was of limited clinical or practical significance. One should note that in the interpretation of the meaning of IQ score levels, scores are not based upon a mean or average of 0 and a standard deviation of 1; but rather -again for ease of interpretation- upon a mean of 100 (average IQ) and a standard deviation of 15. This is true for most, but not all major

IQ tests. As an application, suppose we find in another large-sample study that Asperger individuals have an average IQ of 125 as compared to the average IQ of 100 in a group of non-Asperger general population individuals; and that this was statistically significant, say, at the 1% level. The difference in the size of the two mean IQ levels would be seen to be of clinical or practical significance, as well. It has been noted that a sample size of 200 is considered large; in fact, this is often used by biostatisticians to define a large *N*. This is no doubt related to the simple fact that as *N* gets larger, the t distribution values closer and closer approximate the z distribution values. For example, at an *N* of 120, a t value of 1.98 would be required for statistical significance at the 5% level. Thus a sample size or *N* of 200 virtually guarantees the required z level of 1.96.

See Also

- ▶ [T Scores](#)
- ▶ [Z Scores](#)

Standardization

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Introduction and Definition

Standardization is the process by which a test is administered in a standard or uniform manner from one examinee to another, as well as from one testing milieu to another. This means the test uses a consistent set of rules or instructions to obtain performance scores that should be conceptually more reliable and valid (accurate) than

would be the case if the test were administered in what might be referred to as changing or variable test administration formats – ones that could vary from one test administrator to another, and from one respondent, client, or examinee to another.

Standardization Sample

It is safe to say that the most useful standardization sample would be a large and representative one in the country or other geographic area in which the process takes place.

Standard Scores

Standard scores have exactly the same format, meaning, and interpretation as *z* scores. These scores are based upon a mean or average score of 0 and a standard deviation of 1. A score of 0 would indicate the mean or average score; while one of +1.96 would indicate a score that would be expected to be obtained by only 5% of respondents.

The same logic is used with standard test scores. Most standardized tests such as the Vineland Adaptive Behavior Scales and most cognitive or IQ tests are based upon an average or mean score of 100, with a standard deviation of 15. By the same reasoning as was done for the aforementioned *z* score, this would mean that only 5% of those taking either one of these two tests would be expected to score as high as 115 (again, one standard deviation above the mean or average score of 100).

Interpreting the Meaning of Standard Scores, with Reference to Vineland II Adaptive Behavior Scores

The possible range of Vineland Composite or Domain scores is between more than five standard deviations *below* the mean (or a standard score of

20) to four standard scores *above* the mean (or a score of 160) (Sparrow et al. 2005).

Classifying Levels of Adaptive Behavior on the Basis of a Knowledge of Standard Scores

As noted in Sparrow et al. (2005, p. 65), Vineland Adaptive Levels, like scores on most IQ tests, can be meaningfully classified on the basis of the size of the associated standard score. This can be illustrated, as follows:

Adaptive level	Standard Score Range
High	130 and above
Moderately high	115–129
Adequate	86–114
Moderately low	71–85
Low	70 and below

How Standardized Test Scores Can Be Used to Understand Autistic Behavior

It is expected that a typical pattern of functioning for a person with Asperger Syndrome would be a relatively high IQ score, with a correspondingly relatively low Vineland Socialization standard score. Thus, it would not be unusual for such an individual to have an IQ score of, say, 125 in the moderately high range; but a socialization standard score of 80, or in the moderately low range. In developing an educational plan, emphasis would be mainly on developing and applying techniques to improve interpersonal skills, since cognitive skills are at a very adequate level of functioning.

References and Reading

Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland–II: Vineland adaptive behavior scale, survey forms manual: A revision of the Vineland social maturity scale by Edgar A. Doll* (2nd ed.). Circle Pines: American Guidance Service (AGS).

Standardized Behavior Checklists

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Synonyms

[Rating scale](#); [Report form](#)

Definition

A list of behaviors used to characterize an individual. Checklists can be self-rated, but in the ASD field they are most often completed by informants who know the person well, such as parents, teachers, workshop supervisors, or support staff. Both the items and the response categories are *standardized*, meaning that all users respond to the same questions on the same scale. Often this response scale will be *Likert*-style, which means that responses are ordered along some dimension (e.g., *strongly agree*, *agree*, *neutral*, *disagree*, and *strongly disagree*). Responses are then scored in a standard manner, meaning that it is possible to compare the results across individuals or groups of individuals. Scores may be compared to reference samples, called *normative data*, to determine whether the individual's behavior is similar to his or her peers ("peers" may be defined by any number of characteristics, but usually refers to chronological age and, sometimes, sex). A high-quality standardized checklist will be empirically derived, using standard scale development techniques (e.g., *factor analysis*, *item response theory*), and will have well-established psychometric properties (e.g., *acceptable*, *reliability*, *validity*).

Standardized behavior checklists are used for individuals with autism, both in clinical and

research contexts. Checklist scores are not sufficient for a formal diagnosis of any disorder but may be used by the clinician to inform the diagnostic process. For example, a child's scores on the *Child Behavior Checklist*, the most commonly used standardized behavior checklist in child psychiatry, may be used by the clinician to screen for the presence of externalizing or internalizing disorders. Because checklists are usually faster to administer and less expensive than interview- or observation-based assessments, they are used frequently as outcomes in research. For example, scores on the *Aberrant Behavior Checklist* are often used to evaluate the effectiveness of medication intervention on problem behaviors in children with autism.

See Also

- ▶ [Aberrant Behavior Checklist](#)
- ▶ [Child Behavior Checklist in Autism](#)
- ▶ [Factor Analysis](#)
- ▶ [Validity](#)

References and Reading

- Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, 101(2), 213.
- Clark, L., & Watson, D. (1995). Constructing validity: Basic issues in objective scale development. *Psychological Assessment*, 3, 309–319.
- Downing, S., & Haladyna, T. (Eds.). (2006). *Handbook of test development*. Mahwah: Lawrence Erlbaum Associates.

Standardized Scores

- ▶ [T Scores](#)

Standardized Tests

- ▶ [Language Tests](#)

Standing Balance Assessment

► [Posturography](#)

Stanford-Binet

► [Stanford-Binet Intelligence Scales and Revised Versions](#)

Stanford-Binet Intelligence Scales

► [Stanford-Binet Intelligence Scales and Revised Versions](#)

Stanford-Binet Intelligence Scales and Revised Versions

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Synonyms

[Early Stanford-Binet, Fifth Edition \(Early SB5\);](#)
[SB5;](#) [Stanford-Binet;](#) [Stanford-Binet Intelligence](#)
[Scales;](#) [Stanford-Binet Intelligence Test](#)

Description

The Stanford-Binet Intelligence Scales – Fifth Edition (SB5) – is a widely used individually administered measure of intelligence and cognitive abilities for children and adults ages 2 to 85+. Average testing time is 45–75 min to complete the full-scale IQ battery. An Abbreviated Battery IQ

is available which takes 15–20 min to complete but provides a more restricted assessment of cognitive abilities. The nonverbal IQ domain which can be administered to individuals with limited language abilities, and the verbal IQ domain, which can be administered to individuals with motor and visual impairments, take approximately 30 min each to administer.

The SB5 provides a comprehensive assessment of five factors of cognitive ability: fluid reasoning (ability to solve nonverbal and verbal problems using reasoning skills), knowledge (fund of general information), quantitative processing (ability to work with numbers and solve numerical problems), visual-spatial processing (ability to see patterns, relationships, and spatial orientations), and working memory (ability to store, sort, and transform information in short-term memory), which provide a profile of differential abilities. The SB5 also contains two domain composites: nonverbal IQ (NVIQ) and verbal IQ (VIQ). The five factors are crossed with the two domains resulting in ten subtests. The five factor indexes or the two domains can combine to form the full-scale IQ (FSIQ).

The SB5 has two subtests known as “the routing subtests” which are administered at the beginning of the test to help determine an initial estimate of the examinee’s abilities and to identify an appropriate developmental starting point for the remaining subtests. The nonverbal fluid reasoning subtest (object series and matrices) provides an overview of an examinee’s nonverbal ability, while the verbal knowledge subtest (vocabulary) is an indicator of an examinee’s verbal ability. Excluding “the routing subtests,” items for all subtests are grouped into “testlets” which are arranged by five levels of difficulty for the verbal domain and six levels of difficulty for the nonverbal domain. Many of the SB5 subtests contain more than one item due to the wide range of ages and abilities that each subtest covers. During the administration of the SB5, individuals are administered a series of testlets at each level of functional ability. According to the author, the assessment of several domains in each functional level provides a broader assessment and a larger variety of tasks to maintain the examinee’s interest. Some subtests are

also comprised of different types of “activities” which do not specifically conform to testlets. Activities may extend across several testlets with different levels of difficulty. The SB5 is organized into three item books, one for the routing subtests, one for the verbal subtests, and one for the nonverbal subtests.

The SB5 generates a standard score ($M = 100$) with a standard deviation of 15, which is different than previous versions which had a standard deviation of 16. Subtest scores have a mean of 10 and a standard deviation of 3. The full-scale IQ of the SB5 ranges from 40 to 160.

Historical Background

The SB5 is the newest version of this well-established intelligence assessment that has a long-standing and rich history. The Stanford-Binet is a descendant of the first intelligence test, the 1905 Binet scale. In 1916, Lewis Terman completed an American version of the Binet-Simon Intelligence Scale (SB1). The test was then revised in 1937 (SB2) and renormed in 1960 (Stanford-Binet Intelligence Scale, Form L-M). It was revised again in 1986, resulting in the Stanford-Binet Intelligence Scale – Fourth Edition (SB-IV). The earliest version of the test used item groupings to assess functional ability, which were arranged by levels based on order of difficulty. The 1986 version, SB-IV, changed to a subtest-based point system based on items of increasing difficulty, much like other intelligences tests such as the Wechsler scales. The SB-IV also addressed theories of g measuring crystallized and fluid abilities. The following is a summary of the new features of the fifth edition (Roid 2003b; Strauss et al. 2006):

(a) Expansion of the test to allow the assessment of very low and very high levels of cognitive ability (i.e., extensive high-end items for measuring the highest level of gifted performance and improved low-end items for better measurement of young children, low functioning older children, or adults with mental retardation)

- (b) Restoration of the original toys and manipulatives for assessing preschoolers that had been removed from previous versions
- (c) Increased clinical utility
- (d) Updated materials
- (e) Increased nonverbal items to assist with assessment of examinees with limited English, deafness, or communication disorders
- (f) Increased range of domains measured by the test
- (g) Enhanced memory tasks to provide a comprehensive assessment for adults and the elderly
- (h) Ability to compare verbal and nonverbal performance to assist with evaluating learning disabilities

As a result of these goals and changes, several new subtests were added, but many of the classic subtests were maintained. While the SB-IV measured four factors (verbal, quantitative, abstract/visual reasoning, and short-term memory), the SB5 measures five factors (fluid reasoning, knowledge, quantitative reasoning, visual-spatial processing, and working memory). These factors are based on Cattell-Horn-Carroll theories of intelligence which posits that there are ten broad areas of cognitive ability with narrower abilities underlying these broad factors. Factors that are not measured by the SB5 are auditory processing, long-term retrieval, and processing speed. The SB5 maintained the “routing” subtest technique which was developed in the 1986 version and continues to present items according to function levels. For the SB5, both the routing subtests and functional levels were redesigned using current psychometric methods including item response theory.

Psychometric Data

Normative data for the SB5 were gathered from 4800 individuals between the ages of 2 and 85+ and was closely matched to the 2000 US Census based on age, gender, race/ethnicity, geographic region, and socioeconomic level. The SB5 was also administered to 1365 individuals with mental health and clinical diagnoses (e.g., ADHD,

autism, developmental disability, motor impairment, mental retardation, speech/language impairment, deafness/hard of hearing, and emotional disturbance).

Internal reliability is strong for the full-scale IQ at all ages ($r = .97-.98$). Reliabilities for the nonverbal IQ and verbal IQ are also very high (mean $r = .95$ and $.96$). The Abbreviated Battery IQ is lower but still good (mean $r = .91$, range $.85-.96$). Average reliabilities for the five factor index scores are high ($r = .90-.92$). The average internal consistencies are also respectable ($r = .84$ to $.89$). Overall, test-retest reliability is also good. (Four groups were retested after a test interval of 5–8 days; see Technical Manual.) Full-scale IQ and verbal IQ stability was high for all ages ($r = .93-.95$). Nonverbal IQ was slightly lower ($r = .89-.93$). Abbreviated Battery IQ was also lower but still acceptable ($r = .84-.87$). Subtest and factor index test-retest reliability was generally good ($>.80$); however, the Working Memory Index only obtained adequate reliability in adults. Practice effects were also evaluated and considered to be minimal after a 5–8-day interval. The full-scale IQ increased two to four points, the verbal IQ increased two to three points, and the nonverbal IQ increased two to five points.

Content validity for the SB5 is based on 7 years of development which included literature review, expert advice, user surveys, factor analyses of previous versions, pilot studies, tryout edition, and item response theory modeling. Fairness of the SB5 was evaluated along gender, ethnic, racial, cultural, linguistic, exceptional group status, and religious group status. Fairness evaluation techniques included logical analyses, “offensiveness review” by expert bias reviewers from each of the groups, and various empirical techniques such as conventional item analysis, construct-related studies, and studies of fairness prediction. Based on these evaluations, the SB5 is considered a fair test across groups. Concurrent and criterion validity data were obtained using the following tests: Stanford-Binet Intelligence Scale, Fourth Edition; Stanford-Binet Form L-M; Woodcock-Johnson III; Universal Nonverbal

Intelligence Test; WAIS-III; WIAT-II; WISC-III; and WPPSI-R.

Clinical Uses

The SB5 has a wide variety of uses but is generally considered to be a measure of intelligence and verbal and nonverbal cognitive abilities. SB5 scales are used in the diagnosis of conditions such as intellectual disability (all ages), learning disabilities (all ages), and developmental cognitive delays in young children. It can also be helpful in the assessment of students in programs for the intellectually gifted. Supplemental assessments are usually administered along with the SB5 to assist with assessment and diagnosis. The fifth edition of the SB is designed to provide information for interventions including individual family plans (IEPs) for young children, support from school-to-work transition planning, adolescent and adult career change, employee selection and classification, and adult workers compensation evaluations. The SB5 may also be useful in forensic contexts, as well as part of research assessment batteries investigating abilities and aptitudes. The SB5 does not measure processing speed, an important factor in the assessment of neuropsychological conditions, and therefore, the SB5 may need to be supplemented by other neuropsychological tests to assist with differential diagnoses. Clinical data on patients with traumatic brain injury and Alzheimer’s is also lacking to date. The SB5 is typically administered by psychologists or trained psychometrists with supervision by a psychologist in clinical, counseling, school, or research settings. As with many psychological assessments, the overall usefulness of the SB5 is strengthened when combined with other appropriate assessments to give a well-rounded picture of the examinee.

See Also

- [Cognitive Skills](#)
- [Differential Ability Scales \(DAS and DAS-II\)](#)

- Educational Testing
- Mullen Scales of Early Learning
- Psychological Assessment
- Wechsler Preschool and Primary Scale of Intelligence
- Woodcock-Johnson Cognitive and Achievement Batteries

References and Reading

- Coolican, J., Bryson, S. E., & Zwaigenbaum, L. (2008). Brief report: Data on the Stanford-Binet intelligence scales in (5th ed.) children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38, 190–197.
- Evans, J. J., Floyd, R. G., McGrew, K. S., & Leforgee, M. H. (2001). The relations between measures of Cattell-Horn-Carroll (CHC) cognitive abilities and reading achievement during childhood and adolescence. *School Psychology Review*, 31(2), 246–262.
- Flanagan, D. P. (2000). Wechsler-based CHC cross-battery assessment and reading achievement: Strengthening the validity of interpretations drawn from Wechsler test scores. *School Psychology Quarterly*, 15(3), 295–329.
- Roid, G. H. (2003a). *Stanford-Binet intelligence scales* (5th ed.). Itasca: Riverside.
- Roid, G. H. (2003b). *Stanford-Binet intelligence scales, fifth edition, examiner's manual*. Itasca: Riverside.
- Roid, G. H. (2003c). *Stanford-Binet intelligence scales, fifth edition, technical manual*. Itasca: Riverside.
- Roid, G. H. (2003d). *Stanford-Binet intelligence scales, fifth edition, interpretive manual*. Itasca: Riverside.
- Sattler, J. M. (2008). *Assessment of children: Cognitive foundations* (5th ed.). San Diego: Jerome M. Sattler.
- Strauss, E., Sherman, E. M. S., & Otfried, S. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). New York: Oxford University Press.
- Thorndike, R. L., Hagen, E. P., & Sattler, J. M. (1986). *Stanford-Binet intelligence scale* (4th ed.). Itasca: Riverside.

Stanford-Binet Intelligence Test

- Stanford-Binet Intelligence Scales and Revised Versions

STAR Program

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Definition

The STAR (Strategies for Teaching based on Autism Research) Autism Program uses applied behavior analysis methods to teach skills in the areas of expressive and receptive language, spontaneous language production, functional routines, academic skills, play, and social interaction to children with ASD. It combines the highly structured teaching approach of discrete trial training with a more naturalistic-based approach of pivotal response teaching. Its curriculum was designed to be implemented by a variety of professionals such as special educators and speech-language pathologists, as well as by paraprofessional support staff. The STAR program has three different curriculum modules chosen based on the child's current level of functioning. However, limited empirical evidence is available to support the efficacy of this program.

Historical Background

The STAR program is based on the work of Drs. Joel Arick and David Krug, who initially developed a program combining several behavioral strategies for teaching children with autism including discrete trial training, augmentative communication systems, and the teaching of independence skills. This program was utilized in local school systems. Subsequently, they published a book entitled, *Autistic and Severely Handicapped in the Classroom: Assessment, Behavior Management and Communication Training*, describing the curriculum (Krug et al. 1981). In the late 1990s, Drs. Arick and Krug began a collaboration with

Dr. Ruth Falco and Lauren Loos. This collaboration functioned to help develop a program driven by empirically based teaching methods.

Rationale or Underlying Theory

The purpose of this program is to use research-supported strategies to teach children with autism basic skills including language, pre-academic, social skills, and functional routines. There is a highly structured discrete trial component to teach skills initially, and then a less structured, naturalistic approach is used in an attempt to generalize those skills.

Goals and Objectives

This manualized program includes three levels, each with learning goals in the areas of language, social development, academic skills, and functional routines. Each child begins the program at the level that is congruent with their current developmental skill set.

Treatment Procedures

Skills are initially taught in a discrete trial format in a 1:1 instructional setting and data are collected. Once the skill is mastered, the skill is then taught in a more naturalistic context using a pivotal response teaching method. The second edition of this manualized program is now available and includes multimedia materials.

Efficacy Information

Limited empirical evidence is available to support the efficacy of this program. While there is evidence that supports the techniques used in this treatment approach, the program as a whole has limited data to support its efficacy.

Outcome Measurement

Arick et al. (2004) developed the Functional Assessment and Curriculum for Teaching Everyday Routines as a measure of a student's ability to participate in classroom activities.

Qualifications of Treatment Providers

Unlike many treatment programs for children on the autism spectrum that require implementation by a Board Certified Behavior Analyst, this program is designed to be implemented by a variety of individuals including teachers, support staff, and caregivers.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Pivotal Response Training](#)

References and Reading

- Arick, J., Young, H., Falco, F., Loos, H., Krug, D., Gense, M., et al. (2003). Designing an outcome study to monitor the progress of students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 18, 75–87.
- Arick, J., Nave, G., & Hoffman, T. (2004). *Functional assessment and curriculum for teaching everyday routines*. Portland: STAR Support Center. Retrieved 4 Nov 2010 from <http://www.starautismprogram.com/>
- Arick, J., Loos, L., Falco, R., & Krug, D. (2010). *STAR autism program: A research-based ABA curriculum*. Retrieved 4 Nov 2010 from <http://www.starautismprogram.com/>
- Bacon, E. C., Dufek, S., Schreibman, L., Stahmer, A. C., Pierce, K., & Courchesne, E. (2014). Measuring outcome in an early intervention program for toddlers with autism spectrum disorder: Use of a curriculum-based assessment. *Autism Research and Treatment*, 2014, 1–9.
- Krug, D. (1981). *Autistic & severely handicapped in the classroom: Assessment, behavior management, and communication training*. Portland: ASIEP Education Company.

Startle Reflex

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Synonyms

[Startle response](#)

Definition

A species-specific automatic response to the sudden onset of an acoustic, visual, or tactile stimulus. This response, or reflex, is viewed as a defensive response that varies systematically with the individual's emotional state. The startle response is characterized by a fast series of muscle contractions around the head, neck, and shoulders; the sudden closure of the eyelid (i.e., eyeblink) is the first, fastest, and most reliable component of this reflexive response. The startle reflex is mediated in part by the amygdala. Strength and latency of startle reflex, or eyeblink, have been used to investigate amygdala function in various psychiatric populations with putative amygdala dysfunction (e.g., autism, anxiety).

See Also

- ▶ [Eyeblink Reflexes](#)
- ▶ [Moro Reflex](#)

References and Reading

- Bernier, R., Dawson, G., Panagiotides, H., & Webb, S. (2005). Individuals with autism spectrum disorder show normal responses to a fear potential startle paradigm. *Journal of Autism and Developmental Disorders*, 35, 575–583.
- Davis, M. (2006). Neural systems involved in fear and anxiety measured with fear-potentiated startle. *The American Psychologist*, 61, 741–756.

LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience*, 23, 155–184.

Wilbarger, J. L., McIntosh, D. N., & Winkielman, P. (2009). Startle modulation in autism: Positive affective stimuli enhance startle response. *Neuropsychologia*, 47, 1323–1331.

Startle Response

- ▶ [Eyeblink Reflexes](#)
- ▶ [Startle Reflex](#)

STAT

- ▶ [Screening Tool for Autism in Two-Year-Olds \(STAT\)](#)

State Educational Agency

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Definition

The state education agency (SEA) refers to the state board of education or other agency or officer with the primary responsibilities for oversight and supervision of public elementary and secondary schools. In cases where there is no state board or agency with the authority to conduct supervision over local education agencies (LEAs), the SEA defaults to the officer or agency designated by the governor or in state law.

See Also

- ▶ [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Reading

<http://idea.ed.gov/explore/view/p/%2Croot%2Cdynamic%2CTopicalArea%2C1%2C>

U.S. Department of Education, Office of Special Education Programs 10.04.06. (2006). Building the Legacy: IDEA 2004.

State of Alertness

► Optimal Arousal

Statewide Service Programs

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Definition

Definition: Integrated programs that are explicitly structured to provide, or to directly facilitate the provision of, services to a population of persons with ASD across an entire state. Statewide programs that provide services directly to clients with ASD using a relatively uniform standard of practice are very rare, and so for purposes of comparison we also describe below examples of programs that are formally mandated, designed, and/or contracted to provide *training* to service providers in a specific approach related to ASD treatment and/or education across an entire state (e.g., statewide networks for training in positive behavior supports).

For purposes of this definition, we would only consider programs formally mandated, designed, and/or contracted to provide services across a state to be truly statewide. Private, regional centers may offer excellent services across a region but, absent a formal mandate, we cannot expect them to ensure reasonable access to these services to all residents of a state.

We have also only considered programs established in the United States. Whereas many developed nations have a regional layer of service organization (e.g., départements in France, provinces in Canada, etc.), descriptions of variations from country to country in the organization of this layer are beyond the scope of this entry. Nonetheless, the range of opportunities and challenges offered by statewide service programs in the United States is likely to be comparable to those apparent elsewhere (Doehring and Becker-Cottrill [in preparation](#)).

Historical Background

As part of the broader national trend in the United States between 1890 and 1930, many states developed specialized centers for specific populations that (it was believed) could not be easily accommodated in society: for example, state psychiatric hospitals, state schools for the deaf and for the blind, etc. The development of these centers coincided with the increasingly prominent role of the state in health and education, and was spurred by advances in treatment. Some of these centers grew to be very large, serving thousands of patients who often resided there for much of their lives. With the rise of the disabilities rights movement, the passage of Public Law 94–142, and the move toward deinstitutionalization beginning in the mid-1970s, many states moved away from a reliance on large, stand-alone centers to the development of multisite, community-based programs.

Throughout much of this period, autism remained too rare a disorder to merit its own specialized statewide centers, and we believe that this had a significant impact on the quality of services available. Moreover, because the needs of persons with autism span education and psychiatry, programs often developed in separate service systems, and persons with autism were served in other programs that often failed to recognize the full spectrum of their needs. For example, some adults with autism were served in statewide centers designed to serve adults with schizophrenia, and these programs did not always recognize and address the fundamental deficits in

social and other skills of adults with ASD. Other adults with autism were served in statewide centers designed to serve adults with intellectual disabilities and other psychiatric or conduct disorders, and these programs did not always recognize and address areas of special interest or relatively intact ability. Asperger syndrome was not yet recognized, and the persons affected often received little or no services.

Despite radical changes in the design and delivery of services at the local and the statewide level in the last two decades, the vestiges of this traditional split between educational, health, and psychiatric/behavioral health service systems are still evident in the structure and funding at the statewide level. While we have witnessed an explosion in the range of services designed more specifically for persons with ASD, these are rarely organized via a unitary, statewide structure specific to ASD. They are more often organized via loose partnerships between universities and other specialized centers, or via programs designed to meet the needs of a broader population (see below). These partnerships and programs tend to address a narrow band of the spectrum of needs, and rarely integrate services and supports across traditional service system boundaries.

Rationale or Underlying Theory

With some exceptions (described below), statewide service programs tend to be distinguished less by a specific theoretical orientation than by a structure that ensures reasonable access to services to all eligible clients across the state, or training to the providers who support them. If state agencies seek to influence local service delivery, it is usually through the training provided, or via the broad parameters embodied in licensing and certification requirements.

We describe below various approaches to statewide service delivery. While our description is by no means exhaustive, we believe that these examples characterize the range of approaches used. And while some of the specific programs cited are among the most prominent, no doubt, there

are other examples of innovative and effective programs that we have not described here.

The first, and perhaps best known, statewide service program is the TEACCH (Treatment and Education of Autistic and related Communication-handicapped CHildren) program based at the University of North Carolina (UNC) (Mesibov et al. 2005). While readers seeking more information about the specific theoretical approach are referred to the entry regarding TEACCH, some characteristics regarding their model of service delivery can be highlighted here. First, TEACCH is one of the first attempts to develop and strategically disseminate a specific educational model for persons with ASD across a state using a coordinated program of training. Every year, TEACCH faculty offer comprehensive training that follows a structured curriculum, at multiple sites and including summer sessions, to increase accessibility to educators. Second, TEACCH is also one of the first attempts to take full advantage of the state university infrastructure to increase accessibility to ASD-specific training and services: In addition to its headquarters at UNC-Chapel Hill, TEACCH sites can be found on other campuses of UNC. TEACCH remains one of the few university-based consortia to have offered specific services (e.g., consultation and assessment) in addition to training, and to provide training that spans traditional public school and adult service systems.

Since the establishment of TEACCH, a number of other university-based programs have sought to provide training or services on a statewide basis. The West Virginia Autism Training Center (WV-ATC) at Marshall University was established in 1983 by the state legislature, and has since undertaken a number of statewide programs through a combination of state and federal grants. For example, the Family Focus Positive Behavior Support program was established initially in 1996 through a grant from the Centers for Disease control, and has since expanded through support from the state legislature. The project provides education specialists to facilitate collaboration between family and school and other team members in the development and implementation of behavior support plans across

the state of West Virginia (Becker-Cottrill et al. 2003). The Indiana Autism Resource Center (IRCA), established in 1985 at Indiana University Bloomington, serves as a hub for a wide variety of training events and information about ASD. Both WV-ATC and IRCA also serve statewide needs' assessment functions as designated by their legislatures: WV-ATC established the first state autism registry, and IRCA has conducted triennial needs' assessment to inform state planning. A wider variety of university-based centers providing training across a region have been established or have increased their emphasis on ASD within the past 10 years, many of which are part of the network of University Centers of Excellence on Developmental Disabilities (see www.aucd.org).

There are other examples of statewide service and training programs that are not university based, but that grow out of agencies for public education or welfare. The Delaware Autism Program (DAP) is a consortium of public school programs that together serve the majority of students with ASD in the state (please see the entry for DAP for more information). The Commonwealth of Pennsylvania has implemented a variety of ASD-specific training programs that are primarily organized and/or funded more directly by state agencies. For example, the Pennsylvania Training and Technical Assistance Network (PaTTAN), within the department of Education, has created a network of model classrooms using the verbal behavior and the competent learner models. PaTTAN also helps to convene the National Autism Conference, which includes specific tracks for family members and professionals. The Pennsylvania Bureau of Autism Services was established within the Department of Public Welfare in 2003 to fund and coordinate a wide variety of statewide training and service programs. Many of these specifically complement education-based services by focusing on adults with ASD, or community-based early identification and behavioral support. In general, the most common statewide training programs impacting ASD would be those addressing the implementation of positive behavior supports; though not specific to ASD, these have great potential because challenging behaviors are perhaps the most likely reason for persons with ASD to be excluded from

public schools and other community-based settings.

More recently, a number of federal grant-funded programs have sought to disseminate training in a coordinated way at a statewide level. The National Professional Development Center for Autism (NPDC), funded by the Office of Special Education Programs, U.S. Department of Education, currently involves the collaboration of the University of North Carolina at Chapel Hill (Frank Porter Graham Child Development Institute), the University of Wisconsin (Waisman Center), and the University of California at Davis (M.I.N.D. Institute). The NPDC supports the development of ASD programs within the public schools by helping states to create statewide teams. These teams create model classrooms and develop training programs within their state. The Act Early Campaign orchestrated by the Centers for Disease Control (Daniel et al. 2009) has sought to create cross-agency statewide teams to identify opportunities to improve early detection and intervention in ASD.

Goals and Objectives

In addition to the goals and objectives set by individual clients and their caregivers, the goals most distinct to statewide service programs are those intended to make designated services directly accessible to the full range of eligible clients. In some cases (e.g., early education and intervention services), specific regulations mandate the rapidity with which such services are provided. Consistent with research documenting disparities in early identification and various aspects of intervention (Liptak et al. 2008), we would expect that some statewide service systems may face significant challenges when trying to ensure equal access for individuals and families from racial and ethnic minorities, at or near poverty level, and from rural regions.

Treatment Participants

Eligibility requirements vary from program to program, and usually reflect the general criteria

for accessing the service (e.g., only individuals between 3 and 21 years of age are eligible for services provided or funded via public schools). Because publicly funded statewide service programs are otherwise often designed, if not explicitly mandated, to be freely accessible, eligibility criteria are kept to a minimum. Some of the training programs (e.g., PBS) are intended to support a broad range of children and adolescents.

Treatment Procedures

Recommended treatment procedures reflect the specific focus of the training or service program.

Efficacy Information

While there may be data regarding the efficacy of specific treatment procedures employed or recommended by statewide service programs, there are at present no data that clearly demonstrate the specific efficacy of statewide service programs in providing or facilitating the use of these treatment procedures across a sample of clients that is representative of the population of the state.

Outcome Measurement

While outcome measures for specific treatment procedures employed or recommended by statewide service programs may be described elsewhere, there are at present no models for defining the acceptable outcomes for statewide service programs per se. Statewide service programs seeking to evaluate outcomes related to access could estimate the proportion of eligible individuals who receive services, or who experience significant barriers in accessing services.

Qualifications of Treatment Providers

ASD-specific qualifications for service providers are rarely defined for a specific statewide service or training program. Regulations established by

the state for specific categories of providers (e.g., special education teacher certification, psychologist licensure, etc.) usually represent minimal criteria, and rarely reference ASD-specific competencies. Exceptions include ASD teacher certification requirements or endorsement opportunities implemented in several states with statewide service programs (e.g., teachers in the Delaware Autism Program must complete a graduate-level teaching certificate in ASD to continue teaching students with ASD). The increasing recognition of behavior analysts by state licensure boards, the growing consensus that Board Certification is a minimal entry criteria, and the central role played by behavior analysts in treatment, all suggest that ASD-specific competencies are likely to be first established by states for behavior analysts.

See Also

- ▶ [Board Certified Associate Behavior Analyst](#)
- ▶ [Delaware Autism Program](#)
- ▶ [Positive Behavioral Support](#)
- ▶ [Regional Centers](#)

References and Reading

- Becker-Cottrill, B., McFarland, J., & Anderson, V. (2003). A model of positive behavioral support for individuals with autism and their families: The family focus process. *Focus on Autism and Other Developmental Disabilities*, 18(2).
- Daniel, K. L., Prue, C., Taylor, M. K., Thomas, J., & Scales, M. (2009). Learn the signs. Act early: A campaign to help every child reach his or her full potential. *Public Health*, 123(Suppl 1), e11–e16. e-Supplement: Social Marketing.
- Doehring, P., & Becker-Cottrill, B. (in preparation). *Facing autism nationally: Federal, state, and regional approaches to training, services, research, and policy for persons with an autism spectrum disorder*. Baltimore: Brookes.
- Liptak, G. S., Benzoni, L. B., Mruzek, D. W., Nolan, K. W., Thingvoll, M. A., Wade, C. M., et al. (2008). Disparities in diagnosis and access to health services for children with autism: Data from the National Survey of Children's Health. *Journal of Developmental & Behavioral Pediatrics*, 29(3), 152–160.
- Mesibov, G. B., Shea, V., & Schopler, E. (2005). *The TEACCH approach to autism spectrum disorders*. New York: Springer Science + Business Media.

Statistical Approaches to Subtyping

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Definition

Subtyping or *clustering* is a type of data analysis that seeks to assign elements of a set into groups, or clusters, that are similar (in some sense) to each other, but different from elements in other clusters. It is distinct from *classification*, in which one or more group labels already exist and the algorithm attempts to explain these labels using observed variables; instead, clustering procedures attempt to create such labels from the observed variables directly. For this reason, classification methods are sometimes called “supervised” methods because there are group labels available to “supervise” the partitioning of the data, while clustering methods are termed “unsupervised” because no such labels exist.

Historical Background

As long as there has been data, there has been a desire to find subtypes within data. The historical roots of this emphasis can be seen in taxonomy, where the interest in clustering species into different phylogenetic categories extends back to ancient Greece. However, the statistical approach to subtyping is a relatively recent phenomenon, made possible primarily by the modern computer, which made numerical analysis of large datasets possible. Since then, there has been increasing interest in searching for and understanding subtypes in a wide variety of scientific disciplines, including psychology, where the subtyping of individuals based on their measurements on psychological markers and the categorizing of different types of psychiatric disorders have been frequent topics of research.

Current Knowledge

Three of the most popular and useful approaches to clustering are *k*-means, mixture modeling, and hierarchical clustering. They are all readily available in common statistical software packages such as SPSS, SAS, and R.

K-Means

K-means is a popular clustering algorithm that assigns observations to clusters based on how close they are to cluster centers. The algorithm takes the data in matrix form, with the n observations as rows and the p variables/measurements as columns, and computes distances from each observation to each of k cluster centers using Euclidean distance, the classic distance formula taught in school. *K*-means then assigns each observation to its closest center, recalculates the k cluster means (hence the name), and repeats this back-and-forth process until the cluster assignments do not change anymore and the algorithm has “converged.” An example of a set of clusters obtained via *k*-means with two variables, 100 data points, and $k = 3$ clusters is shown in Fig. 1.

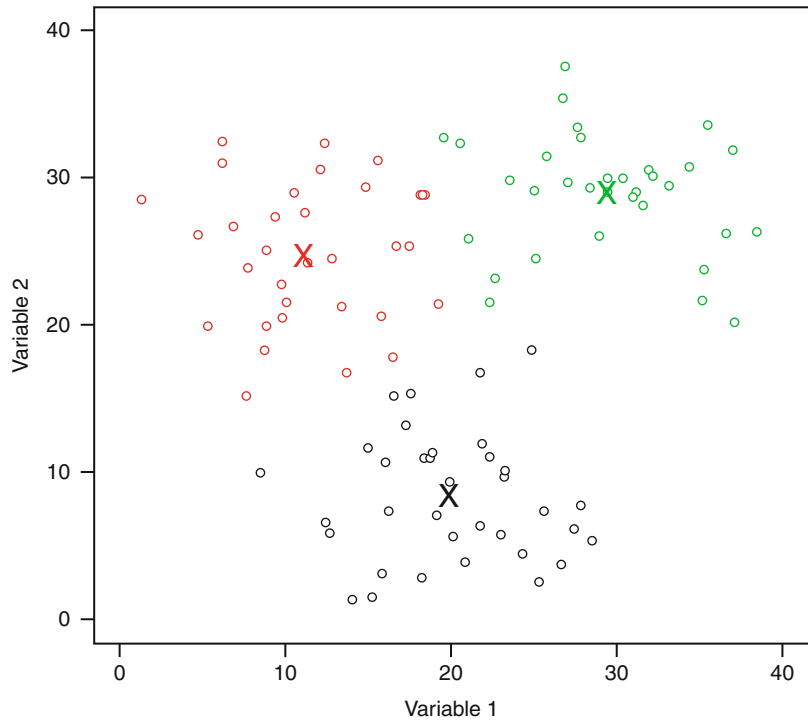
The *k*-means algorithm is computationally very fast, and the clusters it provides tend to be roughly comparable in size and shape. It requires specification of the number of clusters (see section “[Determining the Number of Clusters](#),” below), and making the wrong choice can yield poor clustering results. Also, because it uses Euclidean distance, it works best when all measurements are on the same scale, or else the variable with the largest range will dominate the distances between points; the data can always be scaled to adjust for this, however. Another limitation is that *k*-means is not designed to handle categorical measurements (because the distance between categorical labels, e.g., “Yes,” “No,” and “Maybe,” is undefined).

Mixture Modeling

A broad set of clustering methods are collectively known as *mixture models*. In these methods, a number of “latent” or unknown subgroups are assumed, and a probability distribution for each subgroup is specified. For example, the

Statistical Approaches to Subtyping,

Fig. 1 *K*-means results using $k = 3$ on a sample two-dimensional dataset with 100 observations, with the clusters differentiated by color. The X's indicate the centers of each cluster



observations from each cluster may come from a multivariate Normal distribution, with an unknown mean and standard deviation. Then, for each observation, the model can calculate the probabilities of belonging to each cluster: 90% for Cluster 1, 5% for Cluster 2, etc. The final cluster assignment for each observation is chosen as the cluster with the largest probability.

Mixture models have a lot of advantages. First, unlike in *k*-means where clusters tend to be compact and circular in shape, the latent clusters found in a mixture model can take any arbitrary shape, which provides a lot of freedom for the researcher. Second, the cluster assignments in *k*-means and other methods are absolute – an observation either belongs to a given cluster or it does not, and each observation can only belong to one cluster at a time. In mixture modeling, on the other hand, there is a measure of uncertainty in the cluster assignments, so if an observation is on the border between two cluster regions, it can have a 50–50 chance of belonging to either one. This can be very helpful in understanding how well the cluster assignments describe the data, and in identifying outliers that do not fit easily into any of the clusters.

Like other clustering algorithms, mixture models require you to specify the number of clusters, k . In addition, they also require that the distributions of each cluster be specified as well, which can yield a poor fit to the data if they are chosen incorrectly. While this can make the use of mixture models somewhat more complicated, the added complexity also allows clustering of more interesting and complex types of data, such as longitudinal data. By placing certain assumptions on the correlations between variables, mixture modeling can find clusters in sets of curves; popular variants of mixture modeling that do this are latent cluster analysis and latent trajectory analysis, and they have extensive applications to the longitudinal analysis of autism spectrum disorders.

Hierarchical Clustering

Methods like *k*-means build their clusters by calculating the distance of each cluster to some cluster center. The cluster centers are not observations themselves, they are just arbitrary points. Alternatively, one could build clusters by grouping together observations that are close to each

other, and far apart from the others. This is the strategy employed by hierarchical clustering.

The hierarchical clustering algorithm takes the set of all pairwise distances – distances from every observation to every other observation – and merges observations together into sets if they are very close to each other. The two closest observations get merged first, followed by the next two, and so on. The algorithm also needs one other piece of information: instructions on how to define the distance from a set to other observations (or other sets), given the pairwise distances. This piece is called the *linkage criterion*, and there are many to choose from. Using *single linkage*, the distance from an observation to a set is the minimum distance to any of the members of the set – two sets are close if they have a “single link” making them close together. *Complete linkage* instead uses the maximum distance to any member of the set, and two sets will be close under this criterion only if every pair of observations is close to each other. *Average linkage* attempts a compromise between single and complete linkage, and averages the minimum and the maximum distances. Ward’s method takes a different approach, and combines the observations/sets together that give the smallest increase in variability by merging, so that each new cluster has the smallest possible variance.

Hierarchical clustering continues combining sets until all observations have been merged into a single, all-encompassing cluster. The end result is a *cluster tree* or *cluster dendrogram* made up of nested sets of clusters, where a pair of clusters is merged to give the cluster solution with one fewer label. As such, it provides the cluster solution for all values of k simultaneously, although it is still up to the researcher to determine which value of k to choose. Examples of cluster trees using four different linkage criteria are shown in Fig. 2.

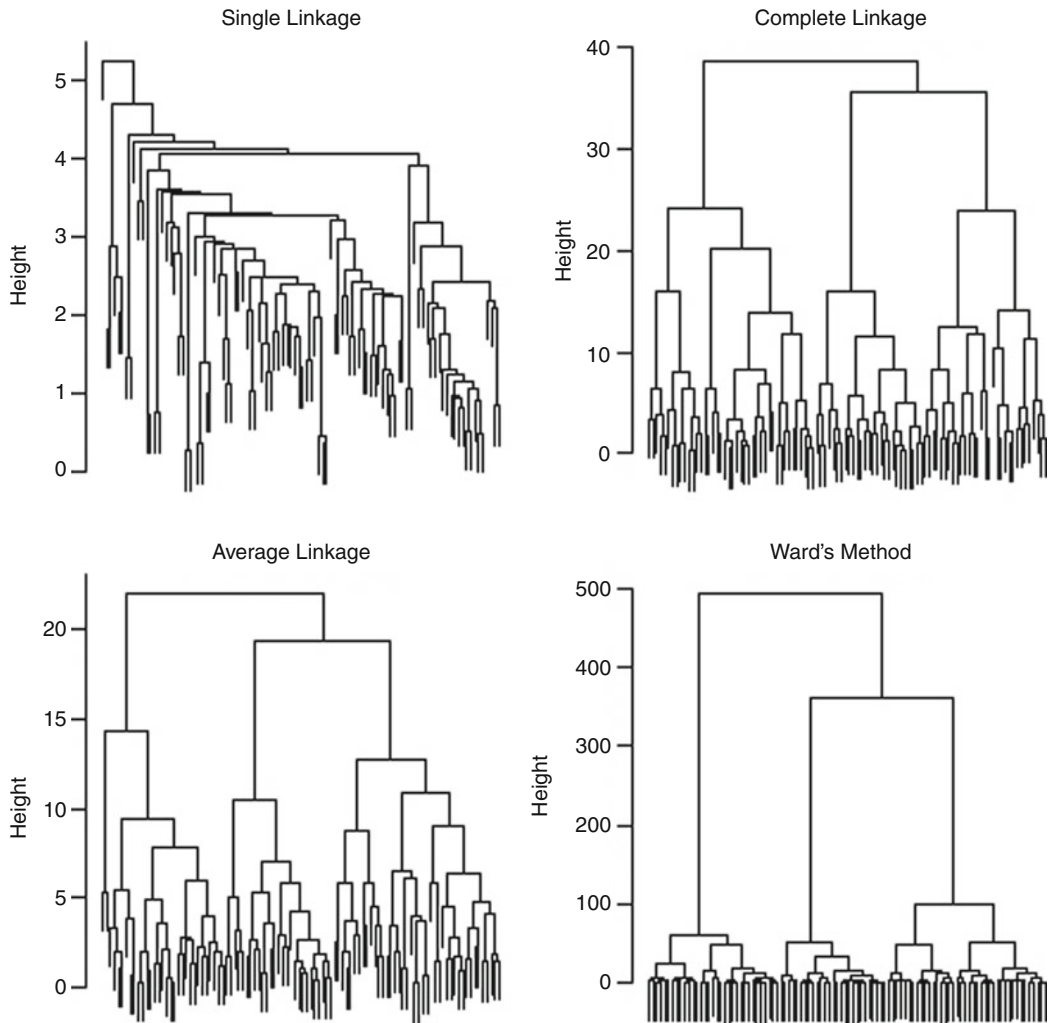
Determining the Number of Clusters

Determining the “correct” number of clusters is one of the most difficult aspects of clustering. Because there is no known “true” grouping in the data (if there were, clustering analysis would not be necessary), there is no way to know how correct any clustering result actually is, and

instead alternative means of assessing the validity of a clustering result must be used. Such methods can be internal, by looking at the statistical differences between clusters on the same variables used to make the clusters, or external, by relying on other variables not included in the clustering analysis to validate the clusters.

Choosing the number of subgroups using internal aspects of the cluster solution is commonly done by means of a *scree plot*. A scree plot graphs the number of clusters, k , on the x-axis, and a measure of variability or dispersion of the data on the y-axis. Typically the y-axis is the *sum of squares*, which is an unscaled version of the sample variance. An example of a scree plot is given in Fig. 3. When $k = 1$, the sum of squares gives the default amount of variability in the data; for larger values of k , the sum of squares will be smaller because the clusters explain some of this variability. The scree plot shows how much explanatory power (measured by a drop in variability) is gained by each additional cluster, with big drops in variability for the first few clusters, but much smaller drops as more and more clusters are added because much of the variability is already captured by earlier clusters. A scree plot can suggest the best choice of k if it displays a “kink” or “elbow,” where the marginal benefit of adding more clusters is relatively small and a more parsimonious clustering is preferable.

External information can also be very useful in choosing the preferred number of clusters. A set of clusters can be validated though hypothesis testing, e.g., comparing differences in means between clusters using Student’s t -test (for two clusters) or an analysis of variance (for three or more clusters). Statistically significant differences between some or all of the clusters on variables not used in the clustering algorithm can indicate meaningful differences between the subgroups. If some clusters do not exhibit statistically significant differences from each other, then they can perhaps be merged together. Care should be taken not to read too much into differences among the variables used to create the clusters, however, because the clustering procedure is designed to maximize differences on these variables, so they cannot serve as outside sources of validation.



Statistical Approaches to Subtyping, Fig. 2 The results of hierarchical clustering using four different linkage criteria on the same dataset used in Fig. 1. Notice that single linkage tends to produce many tiny clusters, while average linkage and Ward's method favor larger groups.

While the overall tree structures are markedly different, on this particular dataset complete linkage, average linkage, and Ward's method give nearly the same subgroupings if the tree is cut into three clusters. This is because the data strongly displays a three-cluster structure, as seen in Fig. 1

Ultimately, the determination of the number of clusters is an art, not a science, and the strategies described here should be treated as rules of thumb. While scree plots and hypothesis tests can give some insight into the right number of clusters, equally important are the principles of parsimony and interpretability. If two clusters appear different on a few variables but not others and are small in size, then they can (and perhaps should) be merged to give fewer subgroups that are easier to understand. Choosing between five and six

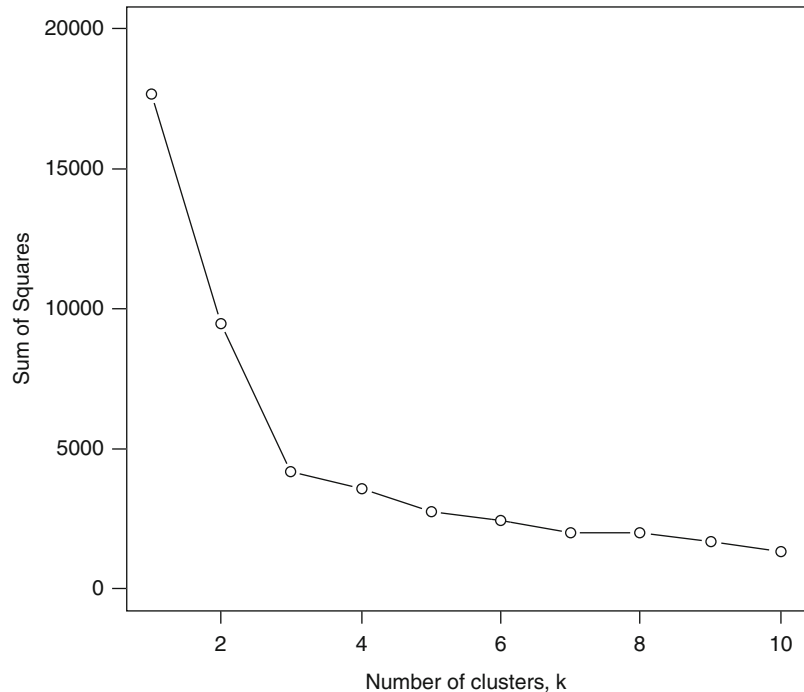
subgroups, for instance, is far less important than understanding what these subgroups represent to the researcher and to his or her scientific field.

Future Directions

The subtyping methods described here work well for datasets in which the number of variables, p , is relatively small compared to the number of observations, n . When the number of variables is much

Statistical Approaches to Subtyping, Fig. 3

A scree plot showing the decreasing sum of squares with increasing k , using k -means on the same data as in Fig. 1. Notice the “kink” at $k = 3$, suggesting three subgroups in the data



larger than the number of observations, the data is said to be *high-dimensional*, and these methods may become computationally slow, or fail to give satisfactory results at all. Such datasets are increasingly more common in genetics and eye-tracking studies, so statistical research is currently ongoing to develop subtyping methods that work well in the high-dimensional setting. A few of these newer methods are subspace clustering, projected clustering, and correlation clustering.

See Also

- [Asperger Syndrome](#)
- [Autism](#)
- [Broader Autism Phenotype](#)
- [Pervasive Developmental Disorder Not Otherwise Specified](#)

References and Reading

Dempster, A. P., Laird, N. M., & Rubin, D. B. (1977). Maximum likelihood from incomplete data via the EM algorithm. *Journal of the Royal Statistical Society, Series B*, 39, 1–31.

- Everitt, B. S., & Hand, D. J. (1981). *Finite mixture distributions*. London/New York: Chapman and Hall.
- Hartigan, J. A. (1975). *Clustering algorithms*. New York/London/Sydney/Toronto: Wiley.
- Hartigan, J. A. (1985). Statistical theory in clustering. *Journal of Classification*, 2, 63–76.
- Hastie, T., Tibshirani, R., & Friedman, J. (2009). *The elements of statistical learning: Data mining, inference, and prediction* (2nd ed.). New York: Springer.
- Jones, B. L., Nagin, D., & Roeder, K. (2001). A SAS procedure based on mixture models for estimating developmental trajectories. *Sociological Methods & Research*, 29, 374–393.
- Kriegel, H.-P., Kröger, P., & Zimek, A. (2009). Clustering high-dimensional data: A survey on subspace clustering, pattern-based clustering, and correlation clustering. *ACM Transactions on Knowledge Discovery from Data*, 3(1), 1–58. New York: ACM.
- MacQueen, J. B. (1967). Some methods for classification and analysis of multivariate observations. In *Proceedings of 5th Berkeley Symposium on Mathematical Statistics and Probability*, Vol. 1. University of California Press, pp. 281–297.
- McLachlan, G. J., & Basford, K. E. (1988). *Mixture models: Inference and applications to clustering*. New York: Marcel Dekker.
- Nagin, D. S., & Tremblay, R. E. (2001). Analyzing developmental trajectories of distinct but related behaviors: A group-based method. *Psychological Methods*, 6(1), 18–34.
- Press, W. H., Teukolsky, S. A., Vetterling, W. T., & Flannery, B. P. (2007). Hierarchical clustering by

- phylogenetic trees. In *Numerical recipes: The art of scientific computing* (3rd ed., pp. 868–863). New York: Cambridge University Press.
- Székely, G. J., & Rizzo, M. L. (2005). Hierarchical clustering via joint between-within distances: Extending Ward's minimum variance method. *Journal of Classification*, 22, 151–183.
- Ward, J. H. (1963). Hierarchical grouping to optimize an objective function. *Journal of the American Statistical Association*, 58(301), 236–244.

Statistical Significance

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Definition

Statistical vs. Clinical Significance: Statistical significance means simply that a result in a given comparison occurs beyond what one would expect by chance alone. Here, and by accepted convention, a comparative result is declared statistically significant if it occurs at or beyond the 5% level. By simple subtraction, this means that there is a 95% possibility that the result did not occur by chance.

The caveat here, and one that many journal editors and even some unenlightened biostatisticians fail to grasp, is that given a large enough number of cases or N, a given comparative result will inevitably occur *beyond chance*, at the 5% level of statistical significance.

In order to guard against this so-called big N phenomenon, enlightened biostatisticians have devised guidelines for defining a result as having reached a level of clinical, as well as statistical significance.

A concrete example, albeit an apocryphal one, can be derived easily from the field of autism spectrum research.

Suppose an inexperienced clinician on a scale of 0–100% agrees with the diagnosis of childhood autism, in 500 cases at 10%. With such a large N of cases, the result turns out to be statistically

significant with a chance probability at the scientifically acceptable level of 5%.

Well, any self-respecting autism expert would tell you that 10% chance-corrected agreement, from a clinical perspective, is poor or trivial.

Clinical significance to the rescue comes in the form of a set of clinical criteria developed by Cicchetti and Sparrow (1981) by which:

- Agreement Clinical
- Level of significance:
 - <40%, Poor
 - 40–59%, Fair
 - 60–74%, Good
 - 75–100%, Excellent

See Also

- [Clinical Significance](#)

References and Reading

- Cicchetti, D. V., & Sparrow, S. S. (1981). Developing criteria for establishing interrater reliability of specific items: Applications to assessment of adaptive behavior. *American Journal of Mental Deficiency*, 86, 127–137.

Stay-Put Requirement

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Synonyms

- [Placement-pending requirement](#)

Definition

In General

“Stay-put requirement” refers to a provision in the Individuals with Disabilities Education Act

(IDEA) (20 U.S.C. § 1415(j) (2011)) that gives a child the right to “stay put” in his or her current educational placement pending resolution of a dispute between the child’s parents and the school district. Under the IDEA, school districts must provide special education and related services to “children with disabilities,” which specifically includes children with autism. When parents and the school district cannot agree on the special education and related services provided to a child, parents and the school district have the right to file a “due process” complaint requesting a hearing before an impartial hearing officer. Once a due process complaint is filed, the child must “stay put” unless the parents and school district agree otherwise (or unless the school district places the child in an “interim alternative educational placement”). For example, if a school district notifies the parents of a child with autism that their child is no longer eligible for special education and related services, the parents have the right to file a due process complaint. Under the IDEA’s stay-put requirement, the child will continue to receive special education and related services pending resolution of the dispute. Similarly, if a school district notifies the parents of a child with autism that the district wants to remove the child from a private special education placement and place the child back in the public schools, and if the parents disagree, the parents have the right to file a due process complaint and invoke “stay put” pending resolution of the dispute.

In the Disciplinary Context

The stay-put requirement has special importance in the disciplinary context. If a child with a disability violates a code of student conduct, school districts are free to suspend the child for up to 10 consecutive days. The rules governing discipline of children with disabilities for more than 10 consecutive days are complex. Generally speaking, if a school district determines that the child’s misconduct was not a “manifestation” of his or her disability, the district may expel a child with a disability or suspend the child for more than 10 consecutive days so long as it provides educational services in an alternative setting. (If the misconduct involves

weapons, drugs, or serious bodily injury, the school district may suspend the child for up to 45 days even if his or her behavior was a manifestation of his or her disability.) Importantly, in the disciplinary context, the filing of a due process complaint does not require that the child “stay put” in his or her “predisciplinary” placement. Rather, the child will remain in his or her alternative educational placement pending resolution of the dispute.

See Also

- Due Process
- Individuals with Disabilities Education Act (IDEA)
- Special Education

References and Reading

- Individual with Disabilities *Education Act (IDEA) regulations*: 34 C.F.R. § 300, et seq. (2011). Retrieved March 14, 2011, from http://ecfr.gpoaccess.gov/cgi/t/text/textidx?c=ecfr&tpl=/ecfrbrowse/Title34/34cfr300_main_02.tpl
- Individuals with Disabilities Education Act (IDEA) statute*: 20 U.S.C. § 1400, et seq. (2011). Retrieved March 14, 2011, from http://www.law.cornell.edu/uscode/html/uscode20/uscode20_00001415%2D%2D%2D%2D0000.html
- Jones, N. L., & Apling, R. N. (2004). Individuals with Disabilities Education Act: Statutory provisions and selected issues. In I. O. Javier (Ed.), *Individuals with Disabilities Education Act (IDEA): Background and issues* (pp. 43–62). Hauppauge: Nova Scotia.
- Latham, P. S., Latham, P. H., & Mandlawitz, M. R. (2008). *Special education law*. Boston: Pearson Education.
- Mooney, T. B. (2008). *A practical guide to Connecticut school law* (6th ed.). Wethersfield: Connecticut Association of Boards of Education.
- Russo, C. J., Osborne, A. G., Jr., Massucci, J. D., & Cattaro, G. M. (2009). *The law of special education and non-public schools: Major challenges in meeting the needs of youth with disabilities*. Lanham: Rowman & Littlefield Education.
- Siegel, L. M. (2007). *The complete IEP guide: How to advocate for your special ed child* (5th ed.). Berkeley: Nolo.
- U.S. Supreme Court. (1988). *Honig v. Doe, et al.* Retrieved March 14, 2011, from <http://www.wrightslaw.com/law/caselaw/ussupct.honig.doe.htm>

- Wright, P., & Wright, P. (2006). *From emotions to advocacy: The special education survival guide* (2nd ed.). Hartfield: Harbor House Law Press.
- Wright, P. W. D., & Wright, P. D. (2009). *Special education law* (2nd ed.). Hartfield: Harbor House Law Press.

their interactions with the environment. Such advances in educational technology offer the opportunities for learners with Autism Spectrum Disorders (ASD) to explore and learn STEM ideas through nontraditional modes of interaction.

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STEM Education and Autism Spectrum Disorder

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Definition

Our fundamental definition of how the fields of educational research and Autism at large interact is through the development of multimodal interactive learning environments as teaching and learning tools to improve the mathematical knowledge and problem-solving skills for learners with Autism Spectrum Disorders (ASD). Such interventions establish a potential importance for their use in special needs education in general. In particular, STEM learning environments offer natural multimodal interactions to enhance the accessibility to complex mathematical and scientific ideas through technological affordances. Multimodal interfaces exist as combined input/output methods or as an alternative input, such as speech inputs (e.g., voice recognition), bodily motion, and touch (e.g., gesture-based interactions). The latter has evolved in recent years with the development of multitouch technologies (e.g., tablet PCs, Interactive Whiteboards, iPads). Combined input/output devices include haptic devices, which integrate visual modes with force feedback loops, offering the user the ability to feel objects or the results of

Historical Background

Learners with Autism Spectrum Disorders represent one of the underrepresented STEM talent pools in the United States (Wei et al. 2012, 2013), since they have an innate attraction to STEM fields (Baron-Cohen et al. 2007; Baron-Cohen 2009) and they are more likely than the general population and other disability groups to choose a STEM major (Wei et al. 2012). Despite their potential to succeed in STEM fields, interest and ability alone may not be sufficient enough to enable a learner with Autism Spectrum Disorders to pursue a STEM major in college (Wei et al. 2015). The data from the National Longitudinal Transition Study-2 (a nationally representative sample of students eligible for special education with an autism spectrum disorder) show that 34.1% of learners with ASD declared a major in STEM field in postsecondary education compared to 22.80% of students in the general population (Chen and Weko 2009). The issue with learners with ASD, however, is postsecondary enrollment, where the rate of learners with ASD is the third lowest of all the disability categories: 30% versus 66% of general population (Wei et al. 2012). The main barriers that learners with ASD face in their learning are related to their challenges in executive functioning and social communication: social interaction, flexible thinking, perspective taking, awareness of and adherence to social rules, language flexibility and discourse skills, motivation, and engagement (Hendricks and Wehman 2009). Research has indicated that learners with ASD use ineffective strategies for problem-solving because they may often focus on irrelevant information, and may not consider outcomes prior to the use of a strategy (Bauminger 2007; Solomon et al. 2004). The problem-solving skills that were studied are not mathematical, but rather related to daily routines, and there is a lack

of research on how students with high-functioning autism can demonstrate executive functions in mathematical problem-solving.

In a review of the literature of Computer-Assisted Instruction (where a computer is used to learn the material and for assessment) between the years of 1997 and 2008, Pennington (2010) inferred that Computer-Assisted Instruction may have promise as an effective intervention for ASD learners, as most of the studies showed effectiveness for teaching academic literacy skills. In 2011, Wainer and Ingersoll concluded that the use of innovative computer technology is a promising strategy for delivering direct intervention to children and adults with ASD. What is missing, according to the authors, is demonstrating the efficacy, effectiveness, and social validity of such interventions across diverse samples of individuals with ASD.

Current Knowledge

The use of computerized environments permits the development of skills in a predictable and controlled environment, while allowing students to work at their own pace and ability level (Golan and Baron-Cohen 2006). These features may be particularly beneficial for learners with Autism Spectrum Disorders given that they often experience discomfort in unpredictable social environments (Charlop-Christy et al. 2000). Learners with ASD typically have strong visual processing skills and a predisposition towards electronic media. Accordingly, interventions using computer technology would be particularly appropriate and motivating (Shane and Albert 2008). The use of interesting visual and auditory stimuli to engage users may be beneficial for learners with ASD given their relative strength in visual processing (Rayner et al. 2009). More importantly, learners with ASD have an innate tendency to gravitate toward STEM fields (Baron-Cohen 2009; Baron-Cohen et al. 2007). They have relatively greater aptitude to analyze or construct rule-based systems to explain the world around them, than to social and emotional reactions to other people's thoughts and feelings (Baron-Cohen 2009).

Systemizing often requires the thinking or skills needed to analyze and construct systems, which also are necessary to perform successfully in many STEM-related fields (Baron-Cohen et al. 2007).

In Mathematics Education research, the use of dynamic interactive representationally rich environments has dominated the design and curriculum space for 25 years with basic and applied research in studies focused on learning, teaching effectiveness, motivation, and collaborative learning (Hegedus and Roschelle 2013). Science Education has utilized similar design spaces but have primarily focused on Logo, and virtual and augmented reality environments focused on real-world issues including climate change and urban planning to name a few. As we build out into a space of learning for all, we notice a lack of attention (or extension) to learners with exceptionalities or specifically, learners with ASD. Multimodal interaction has evolved in various research areas and applications including computer vision/visualization, psychology, and artificial intelligence with increasing use in education particularly in early learning and developmental psychology. Jaimes and Sebe (2007) offer a survey of many of these disciplines including face recognition, facial expression analysis, vocal emotion, gesture recognition, human motion analysis, speech recognition, and eye tracking. They outline how a multimodal interaction can simply be an environment that responds to inputs in more than one modality or communication channel (e.g., speech, gesture, writing) through perceptual, attentive, or enactive interfaces. Dautenhahn (2003) has developed multimodal interactive learning environments as teaching and learning tools for the rehabilitation of children with autism, which establishes a potential importance for their use in special needs education in general.

Future Directions

It is our current goal to understand the interaction of learners with Autism Spectrum Disorders utilizing the current affordances of educational

technology, and to understand their interactivity in a multimodal environment. Learners with ASD have innate attraction to STEM fields and engage well with technology, but are underrepresented in STEM fields and as previous research has shown, inflexibility in thinking hinders their performance. Future work needs to focus on designing and implementing learning environments that focus on improving the executive functions and mathematical problem-solving skill development for learners with ASD. These environments will serve as accommodations for the needs of learners with Autism Spectrum Disorders to learn high-quality mathematics, rather than simplifying the content. Multimodal technologies provide an effective medium through which customizable supports may be provided to learners with ASD that have a variety of needs.

The fields of STEM (particularly Mathematics Education) research needs to fully understand the democratizing effect of examining each and every learner within its design paradigm. Much of the design, efficacy, and large-scale implementation studies through randomized controlled trials over the past two decades can be tested with learners with ASD. More specifically though, we need to focus on what kinds of experimental and methodological supports are necessary to conduct such work. Are traditional research methods necessary and sufficient for such work particularly when we are examining communication skills? Traditional educational research methods focused on discourse and think-aloud protocols, or semi-structured interviewing protocols, might well not be sufficient. Do we focus on less qualitative and more behavioral techniques? Ultimately, our responses are subject to what is needed in schools today: to shift the mindsets of teachers and support staff but ultimately to carefully examine, unpack, and rebuild the art of teaching and learning for each and every learner.

References and Reading

- Baron-Cohen, S. (2009). Autism: The empathizing-systemizing (E-S) theory. *Annals of the New York Academy of Science*, 1156, 68–80.
- Baron-Cohen, S., Wheelwright, S., Burtenshaw, A., & Hobson, E. (2007). Mathematical talent is linked to autism. *Human Nature*, 18(2), 125–131.
- Bauminger, N. (2007). Group social-multimodal intervention for HFASD. *Journal of Autism and Developmental Disorders*, 37(8), 1605–1615.
- Charlop-Christy, M. H., Le, L., & Freeman, K. A. (2000). A comparison of video modeling with in vivo modeling for teaching children with autism. *Journal of Autism and Developmental Disorders*, 30, 537–552.
- Chen, X., & Weko, T. (2009). Students who study science, technology, engineering, and mathematics (STEM) in postsecondary education. US. Department of Education, NCES 2009-161.
- Dautenhahn, K. (2003). Roles and functions of robots in human society: Implications from research in autism therapy. *Robotica*, 21, 443–452.
- Golan, O., & Baron-Cohen, S. (2006). Systemizing empathy: Teaching adults with Asperger syndrome or high-functioning autism to recognize complex emotions using interactive multimedia. *Development and Psychopathology*, 18, 591–617. <https://doi.org/10.1017/S0954579406060305>.
- Hegedus, S., & Roschelle, J. (Eds.). (2013). *Democratizing access to important mathematics through dynamic representations: Contributions and visions from the SimCalc Research Program*. Berlin: Springer.
- Hendricks, D. R., & Wehman, P. (2009). Transition from school to adulthood for youth with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 24(2), 77–88.
- Jaimes, A., & Sebe, N. (2007). Multimodal human computer interaction: A survey. *Computer Vision and Image Understanding*, 108(1–2), 116–134.
- Pennington, R. (2010). Computer-assisted instruction for teaching academic skills to students with autism Spectrum disorders: A review of literature. *Focus on Autism and other Developmental disabilities*, 25, 239. Sage.
- Rayner, C., Denholm, C., & Sigafos, J. (2009). Video-based intervention for individuals with autism: Key questions that remain unanswered. *Research in Autism Spectrum Disorders*, 3(2), 291–303.
- Shane, H., & Albert, P. (2008). Electronic screen media for persons with autism spectrum disorder: Results of a survey. *Journal of Autism and Developmental Disorders*, 38, 1499–1508.
- Solomon, M., Goodlin-Jones, B., & Anders, T. (2004). A social adjustment enhancement intervention for high functioning autism, Asperger's syndrome, and pervasive developmental disorder NOS. *Journal of Autism and Developmental Disorders*, 34(6), 649–668.
- Wainer, A., & Ingersoll, B. (2011). The use of innovative computer technology for teaching social communication to individuals with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 5, 96–107.
- Wei, X., Yu, J., Shattuck, P., McCracken, M., & Blackorby, J. (2012). Science, technology, engineering, and mathematics (STEM) participation among college students with an autism Spectrum disorder. *Journal*

- of *Autism and Developmental Disorders.*, 43, 1539. <https://doi.org/10.1007/s10803-012-1700-z>.
- Wei, X., Christiano, E., Yu, J., Blackorby, J., Shattuck, J., & Newman, L. (2013). Postsecondary pathways and persistence for STEM versus non-STEM majors: Among college students with an autism Spectrum disorder. *Journal of Autism and Developmental Disorders.*, 44, 1159. <https://doi.org/10.1007/s10803-013-1978-5>.
- Wei, X., Yu, J., Shattuck, P., & Blackorby, J. (2015). High school math and science preparation and Post-Secondary STEM preparation for students with an autism Spectrum disorder. *Focus on Developmental Disabilities.* Sage. pp., 32, 1–10. <https://doi.org/10.1177/1088357615588489>.

other, non-ASD, disabilities (Roux et al. 2013). Generally, quality of life is also relatively poor (van Heijst and Geurts 2015) and social inclusion is limited (Howlin et al. 2013).

The Stepped Transition in Education Program for Students with ASD (STEPS) is an individualized therapeutic program to support transition into postsecondary education for adolescents and young adults with ASD. STEPS was designed to be developmentally appropriate, such that students facing high school graduation receive support related to preparing for the transition (Step 1), while those in college receive support related to social inclusion and academic success in post-secondary education (Step 2).

Stepped Transition in Education Program for Students with ASD (STEPS)

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Definition

Students with ASD have unique transition needs that go largely unmet. The transition from adolescence to adulthood (spanning approximately age 18–25) represents a distinct developmental period termed “emerging adulthood” (Arnett 2010). It is during this developmental period that people with ASD who do not have co-occurring intellectual disability appear to be most vulnerable, as they often face loss of services and supports to help them achieve important developmental tasks such as beginning higher education and establishing close and intimate relationships (Taylor and Seltzer 2011). In fact, many students with ASD report feeling ill-prepared for success in the postsecondary domain (Cai and Richdale 2016). Most adults with ASD are employed neither consistently nor gainfully (Engström et al. 2003) and, when employed, they tend to be paid less than young adults with

Historical Background

With heightened awareness of ASD, earlier diagnosis, development of more effective interventions, and greater recognition of ASD among people who do not have co-occurring intellectual disability, the population of cognitively able adolescents and young adults with ASD interested in higher education and career attainment has been steadily rising (White et al. 2011). For a host of reasons, however, young people with ASD often do not enroll in college or, once matriculated, exit prior to graduation. Low levels of educational attainment are associated with later disadvantage in the work place. Although academic supports are often in place to support college students with learning disabilities (Cai and Richdale 2016), support personnel in the university setting often are unprepared to support the host of nonacademic needs with which some students with ASD present (VanBergeijk et al. 2008).

Research has shown that difficulties with self-regulation, social isolation, and task management are among the primary challenges of college students with ASD, and these difficulties may contribute to unsuccessful outcomes (e.g., White et al. 2016a). Additionally, there is evidence that the needs of college students with ASD are fairly unique and distinct from those seen among college students with disabilities such as attention-deficit/hyperactivity disorder (Elias and White

2017). Intervention and support to address these difficulties may contribute to greater odds of success in postsecondary education for students with ASD.

Rationale or Underlying Theory

STEPS is based on cognitive-behavioral theory of change and, as such, the content addresses the student's thoughts, feelings, and behaviors in the here and now. Content of each session is fairly modular and skill-focused. Between-session assignments are used to practice skills and reinforce learning, and every session follows a goal-based agenda.

Mechanistically, STEPS targets two related processes – self-regulation and self-determination. Change in these mechanisms is theoretically believed to mediate observed increases in readiness for transition to college (Step 1) and improvement in college adjustment (Step 2). Self-regulation is a multifaceted construct involving monitoring and modulation of behavior, emotion, and thought (Baumeister et al. 2007). Self-determination includes the ability to identify, set, and then achieve goals.

Among students with disabilities, self-determination has been found to predict adult outcomes (e.g., Chambers et al. 2007). Skills related to advocating for one's needs and appropriate disclosure of the ASD diagnosis are addressed in relation to self-determination. Students in STEPS learn strategies to improve self-regulation as well; cognitive flexibility, stress management approaches, and problem-solving are covered. Self-regulation is related to the difficulties commonly seen in young people with ASD in managing intense emotion (e.g., Mazefsky et al. 2013).

In light of research suggesting limited generalizability of observed treatment effects in ASD samples, and the benefit of parental support to intervention success, STEPS utilizes the input and support of stakeholders in the student's life (e.g., parents) as well as frequent in situ training. In Step 1, students participate in a college immersion experience, and in Step 2, students participate in several social outings with their therapist,

during which they practice skills (e.g., stress management, social initiation). Additionally, there is online content covering the primary STEPS modules, made available to the students, their parents, and identified school personnel, so that skills can be reinforced by other people in the student's life.

Goals and Objectives

STEPS has two primary goals: to promote successful transition into postsecondary education and to improve the college experience. As such, there are two levels within STEPS – Step 1 is for secondary school students planning for graduation and college matriculation and Step 2 is for students enrolled in community college or 4-year university.

Treatment Participants

Step 1 was developed for students with a diagnosis of ASD who are either in secondary school (high school) or who have graduated but are not yet enrolled in college. Students must be 16 years old (so that they are likely in their last 2 years of high school) and have no intellectual disability, given that the goal of the program is to help prepare the student for higher education as well as the verbal demands of the program. The student need not know their postgraduation plans, but STEPS is most appropriate for students who plan to attend either community college or university after high school. Step 2 is for young people with ASD who are college-enrolled. The program is appropriate for students regardless of type of college (e.g., community college, university) or field of training (academic major).

Treatment Procedures

Step 1 is comprised of six counseling sessions, plus a day-long college immersion experience, held over a course of approximately 12–14 weeks. Each student has an identified “school personnel” (oftentimes a teacher who knows the student well)

who participates in the program as well. This identified school personnel receives in-person training, which takes place over the course of two school in-services, during which research-supported strategies for supporting students with ASD during transition from secondary school are presented and discussed. These in-services provide opportunities for role-play, skills practice (among the school personnel), and sharing of ideas among the group of school personnel as well.

Step 2 is comprised of 12–13 individual sessions (student and therapist). These sessions, along with the community-based outings, are held over a 12–16-week period. Each student in Step 2 receives 4–6 community-based outings, the goal of which is to practice new skills related to self-determination and self-regulation in social settings and increase the student's social involvement. As such, outings might involve activities such as joining a new social club on campus or applying a part-time job.

Although students, parents, and school personnel (once identified) are invited to use the STEPS online content, the parents and school personnel are much more actively involved in the program in Step 1 than in Step 2. In Step 2, the student acts more autonomously, and often the parents are not living nearby.

STEPS is individualized to the needs of the particular student. As such, the timing of the sessions, as well as the content and context of the social outings in Step 2, and the goals of the immersion experience of Step 1, are designed to address the needs of the student. Within this flexibility, there are core content sessions for each Step.

Efficacy Information

There is no efficacy data on STEPS at the time of this publication, although a randomized controlled trial (RCT) of the intervention is nearing completion, with a sample of 50 adolescents and adults (aged 16–25) who have ASD. In the preliminary study on feasibility, college students with ASD reported considerable satisfaction with the intervention and there were no drops from intervention (White et al. 2016b). Based on incomplete

data from the current RCT, students and parents have reported that the program is quite helpful (mean = 4.38 and 4.39, respectively, for students and parents, on a 1- to 5-point Likert scale).

Outcome Measurement

STEPS was developed to improve postsecondary outcomes for students with ASD. As such, for Step 1, the primary outcome variable is readiness for college transition, measured with a scale developed for this project that assesses preparedness for transition across three domains: cognitive (expectations, awareness of needs), emotional (fears related to transition), and behavioral (skills needed for postsecondary success). For Step 2, the primary outcome variable is college adjustment, measured with the Student Adaptation to College Questionnaire (SACQ; Baker and Siryk 1999).

In addition, secondary outcomes related to emotion regulation, quality of life, and adaptive skills are assessed throughout the intervention and at 3-month follow-up after STEPS completion. Data are collected from student participants as well as parents and school personnel.

Qualifications of Treatment Providers

Therapists for STEPS should have prior experience working with adolescents and young adults with ASD. In the current RCT, supervision is provided by a licensed clinical psychologist (PhD level) and therapists have been doctoral students in clinical psychology and education.

See Also

- [Adulthood, Transition to](#)
- [College Transitional Programs](#)
- [Emotional Regulation](#)

References and Reading

- Arnett, J. J. (2010). Oh, grow up!: Generational grumbling and the new life stage of emerging adulthood.

- Perspectives on Psychological Science*, 5(1), 89–92. <https://doi.org/10.1177/1745691609357016>.
- Baker, R. W., & Stryk, B. (1999). *Student adaptation to college questionnaire*. Los Angeles: Western Psychological Services.
- Baumeister, R. F., Schmeichel, B. J., & Vohs, K. D. (2007). Self-regulation and the executive function: The self as controlling agent. In A. W. Kruglanski & E. T. Higgins (Eds.), *Social psychology: Handbook of basic principles* (2nd ed., pp. 516–539). New York: Guilford Press.
- Cai, R. Y., & Richdale, A. L. (2016). Educational experiences and needs of higher education students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(1), 31–41. <https://doi.org/10.1007/s10803-015-2535-1>.
- Chambers, C. R., Wehmeyer, M. L., Saito, Y., Lida, K. M., Lee, Y., & Singh, V. (2007). Self-determination: What do we know? Where do we go? *Exceptionality*, 15(1), 3–15. <https://doi.org/10.1080/09362830709336922>.
- Elias, E., & White, S. W. (2017). Autism goes to college: Understanding the needs of a student population on the rise. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-017-3075-7>. Advance online publication.
- Engström, I., Ekström, L., & Emilsson, B. (2003). Psychosocial functioning in a group of Swedish adults with Asperger syndrome or high-functioning autism. *Autism*, 7(1), 99–110.
- van Heijst, B. F. C., & Geurts, H. M. (2015). Quality of life in autism across the lifespan: A meta-analysis. *Autism*, 19(2), 158–167. <https://doi.org/10.1177/1362361313517053>.
- Howlin, P., Moss, P., Savage, S., & Rutter, M. (2013). Social outcomes in mid- to later adulthood among individuals diagnosed with autism and average nonverbal IQ as children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(6), 572–581. <https://doi.org/10.1016/j.jaac.2013.02.017>.
- Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(7), 679–688. <https://doi.org/10.1016/j.jaac.2013.05.006>.
- Roux, A. M., Shattuck, P. T., Cooper, B., Anderson, K. A., Wagner, M., & Narendorf, S. C. (2013). Postsecondary employment experiences among young adults with an autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(9), 931–939. <https://doi.org/10.1016/j.jaac.2013.05.019>.
- Taylor, J. L., & Seltzer, M. M. (2011). Employment and post-secondary educational activities for young adults with autism spectrum disorders during the transition to adulthood. *Journal of Autism and Developmental Disorders*, 41(5), 566–574. <https://doi.org/10.1007/s10803-010-1070-3>.
- VanBergeijk, E., Klin, A., & Volkmar, F. (2008). Supporting more able students on the autism spectrum: College and beyond. *Journal of Autism and Developmental Disorders*, 38(7), 1359–1370. <https://doi.org/10.1007/s10803-007-0524-8>.
- White, S. W., Ollendick, T. H., & Bray, B. C. (2011). College students on the autism spectrum: Prevalence and associated problems. *Autism: The International Journal of Research and Practice*, 15(6), 683–701. <https://doi.org/10.1177/1362361310393363>.
- White, S. W., Elias, R. E., Salinas, C. E., Capriola, N., Conner, C. M., Asselin, S. B., Miyazaki, Y., Mazefsky, C. A., Howlin, P., & Getzel, E. E. (2016a). Students with autism spectrum disorder in college: Results from a preliminary mixed methods needs analysis. *Research in Developmental Disabilities*, 56, 29–40. <https://doi.org/10.1016/j.ridd.2016.05.010>.
- White, S. W., Richey, J. A., Gracanin, D., Coffman, M., Elias, R., LaConte, S., & Ollendick, T. H. (2016b). Psychosocial and computer-assisted intervention for college students with autism spectrum disorder. *Education and Training in Autism and Developmental Disabilities*, 51(3), 307–317.

Steps

► Objective

Stereotyped and Restrictive Behaviors and Sensory Interests (SRS)

► DiBAS-R Validation

Stereotyped Movement Disorder

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Synonyms

Repetitive behavior; Self-injurious behavior; Self-stimulatory behavior; Stereotypy

Short Description or Definition

Stereotyped movement disorder (SMD) refers to motor behaviors characterized by repetition of the

same movements that are seemingly driven and which have no obvious purpose or function (APA 2000, DSM-IV TR). Prior to 1994, SMD was referred to by the American Psychiatric Association as stereotypy/habit disorder. Examples of SMD-related behaviors include hand flapping, body rocking, head rolling, twirling or spinning objects, as well as behaviors that can be self-injurious (e.g., self-hitting, head banging, self-biting). To meet diagnostic criteria for SMD, the behavior needs to interfere with normal activities or result in injury requiring medical treatment and last longer than 4 weeks. Additional criteria require that if mental retardation (currently intellectual and developmental disability) is present, the behavior must be of sufficient severity to become a focus of treatment. In addition, to warrant the diagnosis of SMD, the behavior is not better accounted for by a compulsion (obsessive-compulsive disorder), tic (tic disorder), or stereotypy that is part of a pervasive developmental disorder (PDD) or is not hair pulling (trichotillomania). Finally, the behavior should not be due to the effects of a substance (e.g., a psychostimulant like amphetamine or cocaine) or general medical condition.

In the proposed DSM-V, the relevant work group is recommending that SMD be classified as a neurodevelopmental disorder or an anxiety and obsessive-compulsive spectrum disorder. In addition, in the case of self-injurious responding, the behavior will likely no longer require medical treatment to meet diagnostic criteria. In addition, specification of 4 weeks or longer duration and the requirement that the stereotyped behavior be the focus of treatment will likely not be required for the diagnosis to be made. Instead, the behavior will need to result in clinically significant distress or impairment in important (e.g., social, occupational) areas of functioning (Stein et al. 2010). As in DSM-IV, the stereotypy cannot be consequent to the use of a particular drug or substance or restricted to the symptoms of another disorder (e.g., autism spectrum disorder or ASD, trichotillomania).

The hierarchical exclusion criteria operative in DSM-IV precludes the diagnosis of SMD if stereotypy is part of a pervasive developmental

disorder. With regard to ASD, however, SMD maps on to what has been characterized as the “lower order” or repetitive sensory-motor behavior factor of restricted, repetitive behavior (e.g., Szatmari et al. 2006). Repetitive sensory-motor behaviors are more characteristic of younger or lower functioning individuals with ASD.

Categorization

The SMD diagnosis is most frequently associated with intellectual and developmental disability (IDD), with the occurrence of stereotyped and self-injurious behavior being inversely correlated with degree of intellectual disability (Bodfish and Lewis 2002). Despite this, there has been little or no attempt to ascertain SMD in this population based on the diagnostic criteria reviewed in the previous section, particularly the criterion specifying that stereotyped behavior be the focus of treatment. In individuals with intellectual disability, SMD involving SIB is frequently accompanied by forms of self-restraint (e.g., binding hand or arms in clothing) designed to avoid self-injury, although self-restraint is only temporarily effective (Powell et al. 1996). The available evidence suggests little difference in form or pattern between repetitive motor behaviors in ASD and IDD, although the frequency appears to be higher in the ASD group (Bodfish et al. 2000).

There is growing evidence of the applicability of the SMD diagnosis in non-developmentally delayed individuals (Singer 2009). These stereotypes have been referred to as “primary” stereotypes or “physiological” stereotypes to distinguish them from those associated with a specific clinical disorder. It should be noted, however, that SMD appears to be comorbid with hyperactive behavior or attention-deficit problems as well as anxiety or affective problems. Although there has been little systematic attempt to differentiate stereotyped motor behavior in non-developmentally disabled individuals versus those with autism and related neurodevelopmental disorders, there seems to be considerable overlap in topographies or forms.

Stereotyped motor behavior has also been documented as a common consequence of psychosocial deprivation such as being reared in institutional environments (Bos et al. 2010), severe sensory deficits such as blindness, traumatic brain injury, and dementia, particularly the frontotemporal variety. In addition, stereotyped motor behaviors are commonly observed among typically developing young children. These developmentally appropriate behaviors include thumb-sucking, body rocking, hand flapping, and even transient head banging. These behaviors are self-limited and rarely result in tissue damage. The factors contributing to the progression of normative stereotyped behavior in infants and toddlers to persistent, developmentally inappropriate stereotyped motor behavior are unknown. The frequent expression of topographically similar forms of stereotyped behaviors in typically developing children presents an important challenge to identifying potential SMD in non-developmentally delayed children.

Epidemiology

Prevalence estimates for individuals who meet DSM diagnostic criteria for SMD are not available. There have been attempts, however, to estimate the rates of stereotypy and SIB in samples of individuals with IDD and in non-IDD individuals with “primary” or “physiological” stereotypies. In the former case, stereotypies have been observed in up to 80% of individuals with severe or profound intellectual disability residing in a state residential facility (Bodfish et al. 2000) whereas much lower rates (10–20%) have been observed in individuals who live in the community and have milder intellectual impairment. Self-injury is less common, being observed in 20–25% of individuals who are institutionalized and have severe or profound mental retardation (Bodfish et al.) whereas overall SIB rates vary from 4% to 10% (Arron et al. 2011). Interestingly, SIB rates ranging from 45% to 93% have been reported for individuals with specific genetic syndromes including fragile X, Angelman, cri du chat,

Cornelia de Lange, Prader-Willi, Lowe, and Smith-Magenis (Arron et al.).

The prevalence of complex motor stereotypies in a non-developmentally disabled population has not been established. In a retrospective study of referrals to pediatric neurology-movement disorders clinic, 100 children were identified with persistent “primary” or “physiological” stereotypies. Of these children, 62% were boys, age of onset was 24 months or younger in 80% of cases, and about half displayed comorbid conditions (ADHD, tics, OCD). Castellanos, Ritchie, Marsh, & Rapoport (1996) identified 12 non-IDD adults who met DSM-IV criteria for SMD. Body rocking or thumb-sucking was present in 8 of these 12, and a lifetime history of an affective or anxiety disorder was found for 11 of 12 SMD individuals. A family history of stereotypies, anxiety or mood disorders, tics, and ADHD has also been documented in non-IDD individuals exhibiting complex motor stereotypies (Harris et al. 2008).

Natural History, Prognostic Factors, and Outcomes

Little is known about the developmental timing of the transition from normative to pathological stereotyped behavior. In addition, little is known about environmental, behavioral, or genetic factors that influence or predict the developmental progression of stereotyped behaviors. It does appear, however, that when stereotypies persist beyond what is expected in typical development, they are usually chronic, although not worsening over time. In individuals with ASD, there is some evidence that repetitive sensory-motor behaviors decrease across development (Esbensen et al. 2009; Richler et al. 2010).

Unlike tics or OCD, stereotypies do not appear to have a natural history of waxing and waning. Stereotypies can be interrupted, however, and may vary in frequency depending on the emotional state of the individual. The factor most predictive of a diagnosis of SMD is IDD, with severity of SMD varying with intellectual impairment. Boys are more likely than girls to develop

the disorder with other predictive factors being early psychosocial deprivation, profound sensory impairment, a family history of stereotypies, tics, compulsions, or ADHD, and a comorbid disorder that includes tics, OCD, or ADHD. There is no evidence for birth history being a predictive factor.

Clinical Expression and Pathophysiology

Repetition of invariant sequences of behavior that appear to serve no obvious purpose has long been considered an important dimension of psychopathology. Abnormal repetitive behavior is part of the clinical presentation of a variety of disorders including ASD, OCD, Tourette syndrome, IDD, frontotemporal dementia, schizophrenia, Parkinson's disease, and substance abuse. Such behaviors have been labeled as stereotypies, compulsions, rituals, obsessions, punding, complex tics, self-stimulatory behavior, habits, and perseveration. SMD is more circumscribed, however, and refers to discrete motor responses such as body rocking, head rolling, finger flicking, hand flapping, and repeating the same words or phrases. In some cases, the stereotypy will involve repetitive object manipulation such as spinning or twirling a toy or household object. Other topographies are associated with self-inflicted tissue damage including head banging, biting, head or face slapping, and eye poking. In individuals with IDD, stereotypies can occupy a great deal of the individual's time and interfere with ongoing habilitative efforts and activities. SIB can be quite severe with potential loss of digits or lips from self-biting (e.g., Lesch-Nyhan syndrome) to delivery of self-inflicted blows to the head that have the force of a boxer's jab (Newell et al. 2002).

Beyond its general association with IDD, specific forms of SMD have been associated with specific genetic syndromes: skin picking in Prader-Willi syndrome; self-biting of the lips and fingers in Lesch-Nyhan syndrome; midline hand claspings in Rett syndrome; and hand flapping in fragile X syndrome to name but some (Lewis and Kim 2009). Understanding the pathophysiology

of these genetic syndromes will help inform the neural bases of SMD.

There is only one postmortem study that has linked SMD with a specific neuropathological finding (Lloyd et al. 1981). In this report of three Lesch-Nyhan cases, severe SIB was associated with markedly reduced striatal dopamine concentrations. Later, positron-emission tomography (PET) studies confirmed the loss of dopamine innervation to striatum in Lesch-Nyhan patients (Ernst et al. 1996; Wong et al. 1996). A number of clinical and animal model studies have provided additional support for the importance of alterations in basal ganglia dopamine function in stereotypy and SIB (Turner and Lewis 2002). Neuroimaging findings in ASD have linked repetitive behavior to altered caudate volume although this association involved "higher order" behaviors reflecting resistance to change or insistence on sameness not sensory-motor repetitive behaviors. Kates, Lanham, and Singer (2005), however, compared boys with stereotypies but no other known neurological or neurodevelopmental disorder with matched controls. Decreases in frontal white matter were observed in the stereotypy group even after controlling for total white matter. Caudate volumes did not differ between groups, however, after taking total brain volume into account.

Much of what we know about the pathophysiology of stereotyped motor behaviors comes from studies using various animal models (Lewis et al. 2007). These models can be categorized as induction of stereotyped behavior by (1) targeted CNS insults, (2) pharmacological agents, and (3) environmental restriction or deprivation. These models generally support a preeminent role for alterations (dopaminergic and non-dopaminergic) in cortical-basal ganglia circuitry in the expression of stereotyped behavior. This circuitry involves projections from select areas of cortex to striatum and then on to other basal ganglia (globus pallidus, substantia nigra), then to thalamus and finally back to cortex. At least three parallel but interacting loops have been identified that make up this cortical-basal ganglia circuitry: the motor, limbic, and associative loops. Of these, the motor loop appears to be the best candidate for

mediation of SMD. This loop involves both a direct and indirect pathway from striatum back to cortex. Dysregulation of cortico-striato-thalamocortical circuitry associated with motor disorders is thought to be due to an imbalance between the direct and indirect pathways comprising this circuit. Recent evidence from an animal model suggests that decreased activity of the indirect pathway is associated with higher levels of stereotyped behavior (Lewis and Kim 2009). A review of relevant animal models suggest that stereotyped and self-injurious behavior can result from a variety of perturbations (e.g., drugs, lesions, gene deletions) to this cortical-basal ganglia circuit.

Evaluation and Differential Diagnosis

As SMD should not better be accounted for by a compulsion, tic, or PDD-related stereotypy, careful assessment of the individual will be required to assess the presence of these other disorders. In addition, given the high prevalence of SMD in developmentally disabled individuals, careful assessment of IDD is also required. The diagnosis of SMD may be particularly challenging with regard to differentiation from complex motor tics. Stereotypies, however, typically have an earlier age of onset than tics; involve hands and arms versus face, head, and shoulders; and tend to be longer in duration and often rhythmic in expression. Careful attention should also be paid to assessment of conditions reported to be comorbid with stereotyped motor behavior (e.g., tics, ADHD, OCD). It will also be critical to differentiate developmentally appropriate and developmentally inappropriate stereotyped movements.

Although there is no “gold standard” diagnostic assessment tool for SMD, there are specific instruments designed to assess stereotyped and other repetitive behaviors. One of the more useful is the Repetitive Behavior Scale-Revised (RBS-R; Bodfish et al. 2000) which includes stereotypy and SIB subscales. The RBS-R is an informant-based rating scale with good psychometric properties that provides for both an endorsement of specific forms of stereotyped and self-injurious

behavior as well as ratings of intensity or severity of each item. Other options for assessment include the Childhood Routines Inventory (CRI; Evans et al. 1997), which includes items that load on a “just right” factor and a “repetitive activities” factor, and the Repetitive Behavior Questionnaire (RBQ; Arron et al. 2011), which has been used to examine stereotypy and self-injury in a number of genetic syndromes. Observational measures have also been used to provide more detailed assessments of frequency and duration.

Treatment

Both behavioral and pharmacological interventions have been used to treat stereotyped and self-injurious behavior. With regard to psychotropic medication, the two drug classes most commonly employed include atypical antipsychotics (e.g., risperidone) and selective serotonin reuptake inhibitors (SSRIs; e.g., fluoxetine). A recent multisite study of the SSRI citalopram provided no evidence for its efficacy in treating repetitive behavior in autism (King et al. 2009). There is limited evidence for the utility of atypical antipsychotics in treating stereotyped and self-injurious behavior. Risperidone, the most widely studied drug in this class, appears to have some efficacy for SMD but may be better suited to the treatment of irritability and aggression. In addition, risperidone carries the risk of adverse effects including weight gain and metabolic syndrome. Aripiprazole appears to be useful in the treatment of stereotypies, but significant adverse effects have also been reported. Finally, the opiate antagonist naltrexone has been used in the treatment of self-injury, but reports are mixed as to its efficacy.

Behavioral treatments for SMD are generally based on operant learning principles and administered by behavior analysts (Rapp and Vollmer 2005). These interventions often follow completion of a functional analysis of the stereotyped or self-injurious behavior which is designed to identify existing socially mediated consequences that maintain the behavior. An example of such an assessment might show that whereas stereotypy is not affected by socially mediated

consequences, the self-injury of a particular individual results in either attention or escape from demands and is thereby reinforcing. Alternative appropriate behaviors designed to result in attention or escape can then be shaped and reinforced. This strategy is referred to as differential reinforcement of other (or alternative or incompatible) behaviors (DRO/DRA/DRI). This treatment can also be used in conjunction with response interruption. Another intervention strategy is functional communication training (FCT) which is designed to replace stereotyped or self-injurious behaviors by providing the individual with more appropriate and effective ways to act on the environment and achieve the individual's desired ends. This strategy is particularly useful with nonverbal individuals. Finally, environmental enrichment is used as a way to provide alternative response or behaviors for the individual that can be reinforced.

See Also

- Citalopram
- Habit Reversal
- Repetitive Behavior
- Risperidone
- Sameness, Insistence on
- Self-injurious Behavior
- Stereotypic Behavior

References and Reading

- APA. (2000). *Diagnostic and Statistical Manual of Mental Disorders, Text Revision (DSM-IV-TR)*. Washington, DC: American Psychiatric Publishing.
- Arron, K., Oliver, C., Moss, J., Berg, K., & Burbidge, C. (2011). The prevalence and phenomenology of self-injurious and aggressive behaviour in genetic syndromes. *Journal of Intellectual Disability Research: JIDR*, 55(2), 109–120.
- Bodfish, J. W., & Lewis, M. H. (2002). Self-injury and co-morbid behaviors in developmental, neurological, psychiatric, and genetic disorders. In S. Schroeder, M.-L. Oster-Granite, & T. Thompson (Eds.), *Self-injurious behavior: Gene-brain-behavior relationships* (pp. 23–40). Washington, DC: APA Books.
- Bodfish, J. W., Symons, F. J., Parker, D. E., & Lewis, M. H. (2000). Varieties of repetitive behavior in autism: Comparisons to mental retardation. *Journal of Autism and Developmental Disorders*, 30, 237–243.
- Bos, K. J., Zeanah, C. H., Jr., Smyke, A. T., Fox, N. A., & Nelson, C. A., 3rd. (2010). Stereotypies in children with a history of early institutional care. *Archives of Pediatrics & Adolescent Medicine*, 164(5), 406–411.
- Castellanos, F. X., Ritchie, G. F., Marsh, W. L., & Rapoport, J. L. (1996). DSM-IV stereotypic movement disorder: persistence of stereotypies of infancy in intellectually normal adolescents and adults. *The Journal of Clinical Psychiatry*, 57(3), 116–122.
- Ernst, M., Zametkin, A. J., Matochik, J. A., Pascualvaca, D., Jons, P. H., et al. (1996). Presynaptic dopaminergic deficits in Lesch-Nyhan disease. *The New England Journal of Medicine*, 334(24), 1568–1572.
- Esbensen, A. J., Seltzer, M. M., Lam, K. S., & Bodfish, J. W. (2009). Age-related differences in restricted repetitive behaviors in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(1), 57–66.
- Evans, D. W., Leckman, J. F., Carter, A., Reznick, J. S., Henshaw, D., et al. (1997). Ritual, habit, and perfectionism: the prevalence and development of compulsive-like behavior in normal young children. *Child Development*, 68(1), 58–68.
- Harris, K. M., Mahone, E. M., & Singer, H. S. (2008). Nonautistic motor stereotypies: clinical features and longitudinal follow-up. *Pediatric Neurology*, 38(4), 267–272.
- Kates, W. R., Lanham, D. C., & Singer, H. S. (2005). Frontal white matter reductions in healthy males with complex stereotypies. *Pediatric Neurology*, 32(2), 109–112.
- King, B. H., Hollander, E., Sikich, L., McCracken, J. T., Scahill, L., et al. (2009). Lack of efficacy of citalopram in children with autism spectrum disorders and high levels of repetitive behavior: citalopram ineffective in children with autism. *Archives of General Psychiatry*, 66(6), 583–590.
- Lewis, M., & Kim, S.-J. (2009). The pathophysiology of restricted repetitive behavior. *Journal of Neurodevelopmental Disorders*, 1, 114–132.
- Lewis, M. H., Tanimura, Y., Lee, L. W., & Bodfish, J. W. (2007). Animal models of restricted repetitive behavior in autism. *Behavioural Brain Research*, 176(1), 66–74.
- Lloyd, K. G., Hornykiewicz, O., Davidson, L., Shannak, K., Farley, I., et al. (1981). Biochemical evidence of dysfunction of brain neurotransmitters in the Lesch-Nyhan syndrome. *The New England Journal of Medicine*, 305(19), 1106–1111.
- Newell, K. M., Challis, J. H., Boros, R. L., & Bodfish, J. W. (2002). Further evidence on the dynamics of self-injurious behaviors: impact forces and limb motions. *American Journal on Mental Retardation*, 107(1), 60–68.
- Powell, S. B., Bodfish, J. W., Parker, D. E., Crawford, T., & Lewis, M. H. (1996). Self-restraint and self-injury: Occurrence, association with compulsions, and motivational significance. *American Journal on Mental Retardation*, 101, 41–48.

- Rapp, J. T., & Vollmer, T. R. (2005). Stereotypy I: a review of behavioral assessment and treatment. *Research in Developmental Disabilities*, 26(6), 527–547.
- Richler, J., Huerta, M., Bishop, S. L., & Lord, C. (2010). Developmental trajectories of restricted and repetitive behaviors and interests in children with autism spectrum disorders. *Development and Psychopathology*, 22(1), 55–69.
- Singer, H. S. (2009). Motor stereotypies. *Seminars in Pediatric Neurology*, 16(2), 77–81.
- Stein, D. J., Grant, J. E., Franklin, M. E., Keuthen, N., Lochner, C., et al. (2010). Trichotillomania (hair pulling disorder), skin picking disorder, and stereotypic movement disorder: toward DSM-V. *Depression and Anxiety*, 27(6), 611–626.
- Szatmari, P., Georgiades, S., Bryson, S., Zwaigenbaum, L., Roberts, W., et al. (2006). Investigating the structure of the restricted, repetitive behaviours and interests domain of autism. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 47(6), 582–590.
- Turner, C. A., & Lewis, M. H. (2002). Dopaminergic mechanisms of self-injurious behavior and related disorders. In S. Schroeder, M.-L. Oster-Granite, & T. Thompson (Eds.), *Self injurious behavior: Gene-brain-behavior relationships* (pp. 165–180). Washington, DC: APA Books.
- Wong, D. F., Harris, J. C., Naidu, S., Yokoi, F., Marengo, S., et al. (1996). Dopamine transporters are markedly reduced in Lesch-Nyhan disease in vivo. *Proceedings of the National Academy of Sciences of the United States of America*, 93(11), 5539–5543.

Stereotypic Behavior

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Synonyms

Repetitive behavior; Repetitive movements; Self-injurious behavior; Self-stimulatory behavior; Stereotypic movement disorders; Stereotypies

Definition

Stereotypies

Etymology: Gk, *stereos* + *typos* mark

Stereotypies are repetitive, persistent, non-goal, and apparently purposeless motor actions and speech patterns which are carried out in a rhythmic and uniform way that serves no obvious adaptive functioning and are not explained by other movement disorders or paroxysmal event.

It is necessary to distinguish from stereotypic behavior that is a behavior that looks unusual, strange or inappropriate, a voluntary act that occurs repeatedly in the same way and it is characteristic for that person but does not cause physical damage, therefore includes not only stereotypies but other repetitive behavior or sensory action leading to overestimated frequency compared with stereotypies.

In DSM-5, stereotypic movement disorder (SMD) is included in the neurodevelopmental section, with onset in the early developmental period, as a variety of repetitive and uncontrolled movements for a period of no less than 4 weeks that interferes with activities and may result in self-injury. DSM-5 substituted no functional by purposeless motor actions, added criteria of severity, and include specifiers if associated to self-injury, genetic, medical, and another neurodevelopmental disorders or environmental factors. The ICD-11 definition is very similar to DSM5, but SMD is distinguished from body-focused repetitive behavior disorders, such as trichotillomania and skin-picking disorders, not preceded by cognitive phenomena included under the umbrella of obsessive and related disorders.

Stereotypies usually begin in the first years of life, most of the cases before 2 years of age, the movements are paroxysmal, last for seconds to minutes, appear multiple times a day, and have a fixed pattern. The movements usually abruptly cease when the person is distracted, though they may immediately return. They are not present during the sleep, and phonation may occur during the performance. Stereotypies are usually maintained by the reinforcement of the behavior itself, described as calming and comfortable, and people affected usually does not try to stop them. Stereotypies are not associated to premonitory urge, and they are usually precipitated by overwhelming environment, sensory overload, uncontrollable emotions, thoughts, or intense imagery. They are

different from mannerisms which are unusual repeated performances of goal-directed motor actions or maintenance of an unusual modification of adaptive postures. They are also different from tics that tend to be rapid, random, and associated to premonitory urges or desires to reduce an inner tension.

Primary motor stereotypies (leg shaking, thumb sucking, nail biting, grinding) are common in typically developing children (up to 70%), whereas complex motor stereotypies (hand flapping, waving, finger wiggling, mouth opening, orofacial movements, head nodding) are much less frequent (3–4%). Primary complex motor stereotypies occur also in children without underlying disorders and usually persist along their life being the head nodding the more likely to be resolved in adult life. In typical developing children, stereotypies decrease from 2 years old, and concern arises when persist; they are very intense and frequent, appear with an atypical presentation, or interfere with functioning.

Secondary stereotypies occur associated with other disorders such as developmental disabilities, sensory deprivation or deficits (70–100% of blind people), schizophrenia, autistic spectrum disorders (ASD), genetic disorders such as Rett syndrome, Lesch Nyhan, Cornelia de Lange, Angelman syndrome, Fragile X, Williams syndrome, and Smith-Magenis syndrome, neurological disorders (tumors, Parkinson's disease, dementia, infections, autoimmune disorders, etc.), and drugs, namely, neuroleptics, amphetamines, or cocaine.

Approximately half of the people with autism present stereotypies which are more frequent at younger age, lower intelligence, and with greater symptom severity. Gender is not associated with increased prevalence. In most of cases, triggering factors are found, namely, anxiety, anger, noise, interest in objects, and visual imagery. Sometimes pleasure and anxiety occur during the stereotypies. In children with autism, stereotypies may interfere in the acquisition of new skills, functional performance, and social interaction and may be associated with stigmatization. Although stereotypies are very frequent in people with autism, they are very little understood with little knowledge of pathophysiology and treatment.

It is essential to distinguish stereotypies from other repetitive behaviors and also from tics or seizures in order to reach a better understanding of the phenomena. Although the definition of stereotypies includes to be no functional and purposeless, adolescents and adults with autism argue that stereotypies are a useful coping mechanism that helps them to regulate themselves when experiencing intense emotions, thoughts, and imagery.

Stereotypies are related to dysfunction of prefrontal corticobasal ganglia or cortico-striatal-thalamo-cortical pathways, associated with hyperstimulation of dopaminergic systems. Low level of GABA in anterior cingulate cortex and striatum and in neuroimaging studies volumetric reductions in frontal white matter have also been found.

With respect to treatment, behavior-modifying techniques, habit reversal, and differential reinforcement of other behaviors are the most frequently used treatments although pharmacological treatments are also used without a firm evidence of effectiveness. There is some evidence of parent intervention behavior therapy, with stronger effect in younger children.

See Also

- Repetitive Behavior
- Stereotyped Movement Disorder

References and Reading

- Carcani-Rathwell, I., Rabe-Hasketh, S., & Santosh, P. J. (2006). Repetitive and stereotyped behaviours in pervasive developmental disorders. *Journal of Child Psychology and Psychiatry*, 47, 573–581.
- Kapp, S. K., Steward, R., Crane, L., Elliott, D., Elphick, C., Pellicano, E., & Russell, G. (2019). People should be allowed to do what they like: Autistic adults' views and experiences of stimming. *Autism*, 23(7), 1782–1792.
- Katherine, M. (2018). Stereotypic movement disorders. *Seminars in Pediatric Neurology*, 25, 19–24.
- Melo, C., Ruano, L., Jorge, J., Pinto Ribeiro, T., Oliveira, G., Azevedo, L., & Temudo, T. (2019). Prevalence and determinants of motor stereotypies in autism spectrum disorder: A systematic review and meta-analysis. *Autism*, 25, 1–22.

- Péter, Z., Oliphant, M. E., & Fernandez, T. V. (2017). Motor stereotypies: A pathophysiological review. *Frontiers in Neuroscience, 11*, 171.
- Scahill, L., et al. (2012). Effects of risperidone and parent training on adaptive functioning in children with pervasive developmental disorders and serious behavioral problems. *Journal of the American Academy of Child & Adolescent Psychiatry, 51*(2), 136–146.

Stereotypic Movement Disorders

- [Stereotypic Behavior](#)

Stereotypies

- [Stereotypic Behavior](#)

Stereotypy

- [Stereotyped Movement Disorder](#)

Steroids

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Definition

Steroids are naturally occurring hormones in the body that carry out many important biological functions. Medications that have been developed for steroids are primarily used as anti-inflammatory

medications or hormone replacement therapy. Early case studies suggested that steroid medications could be helpful in children with autism or related developmental disabilities (Lerman et al. 1991; Stefanos et al. 1995). In addition, a case study in a child with concurrent autism and a rare autoimmune disorder condition reported remission of the developmental delays following treatment with prednisone. However, it is generally agreed that steroids are unlikely to be useful in children or adults with autism who do not have other medical conditions that require their use. Steroid medications are well known for adverse effects that are behavioral in psychiatric nature. For example, steroids can cause increased anxiety, depressed mood, or mania in some patients. This may be important in children or adults treated with steroids for medical reasons who suddenly develop psychiatric symptoms.

References and Reading

- Lerman, P., Lerman-Sagie, T., & Kivity, S. (1991). Effect of early corticosteroid therapy for Landau-Kleffner syndrome. *Developmental Medicine and Child Neurology, 33*(3), 257–260.
- Shenoy, S., Arnold, S., & Chatila, T. (2000). Response to steroid therapy in autism secondary to autoimmune lymphoproliferative syndrome. *Journal of Pediatrics, 136*(5), 682–687.
- Stefanos, G. A., Grover, W., & Geller, E. (1995). Case study: corticosteroid treatment of language regression in developmental disorder [see comments]. *Journal of the American Academy of Child and Adolescent Psychiatry, 34*(8), 1107–1111.

Stigmatization

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Synonyms

[Social stigma](#)

Definition

Stigmatization is the social disgrace or disapproval of a personal trait that goes against the norm. The label of “autism” is most stigmatizing when it is misunderstood or used to isolate individuals. For example, individuals with autism have been negatively characterized as being mentally retarded, not intelligent, and socially awkward. Parents of children with autism were incorrectly characterized as having poor parenting skills or not loving their child. These characterizations have led to social isolation and rejection. Fortunately, the stigma or disapproval for autism is decreasing as people learn more about the disorder.

See Also

► [Refrigerator Mother](#)

References and Reading

- Janzen, J. E. (2003). *Understanding the nature of autism: A guide to the autism spectrum disorders* (2nd ed.). San Antonio: PsychCorp.
- Wetherby, A. M., & Prizant, B. M. (2000). *Autism spectrum disorders: A transactional developmental perspective*. Baltimore: Brookes.

Stimulant Medications

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Synonyms

[Psychostimulants](#)

Definition

Stimulants, also called psychostimulants, are medications that enhance dopamine and, to a lesser extent, norepinephrine in the brain. The two most common classes of stimulants are methylphenidate and amphetamines.

Amphetamine: Amphetamines are stimulant medications that enhance the release and block the reuptake of dopamine in the brain. Taken at large doses, this mechanism of action can produce euphoria and increased energy. Because of these effects, amphetamines are subject to abuse. In doses used to treat attention deficit/hyperactivity disorder, however, these stimulant effects are not usually present. The enhanced release of dopamine is presumed to be the source of improved attention and decreased activity.

Amphetamines are associated with many adverse effects that are relevant to children and adults with autism. For example, they can cause insomnia, decreased appetite, increased stereotypic behavior, and irritability. To date, the amphetamines have only been evaluated in small studies in children with autism with equivocal results. It appears that the potency of the amphetamine compounds makes it difficult to find a dose that is helpful, but not associated with dose-limiting adverse effects.

Methylphenidate: Methylphenidate is also a stimulant medication. Although methylphenidate also enhances release and blocks the reuptake of dopamine, it is less potent than the amphetamines. There are many preparations with methylphenidate including immediate-release compound in which the tablet is rapidly absorbed and the duration of action is approximately 4 h. When using the immediate-release compounds, it is likely that the dose will have to be repeated twice or even three times per day. There are also long-acting formulations marketed under several different trade names. These long-acting formulations do not have to be repeated throughout the day. However, various products may only come in particular strengths. Therefore, becoming familiar with the different brands of medications is important for using these long-acting formulations of methylphenidate in the clinic. Methylphenidate also comes in a transdermal skin patch. The skin

patch can be worn for 8–9 h per day and replaced with a new patch each day. The skin patch comes in several strengths and may require some trial and error to achieve comparable dosing with oral forms of methylphenidate. One drawback of the current skin patch preparation is the relatively high cost.

The immediate-release formulation of methylphenidate was studied in a large-scale, multisite trial in children with autism spectrum disorders (Research Units on Pediatric Psychopharmacology 2005). The study included three doses of methylphenidate (low, medium, and high) and placebo in a crossover trial (each subject received the study treatments in random order under double-blind conditions). All active doses of methylphenidate were superior to placebo in this short-term study. In contrast to the benefits observed in typically developing children with attention deficit/hyperactivity disorder, however, the beneficial effects were modest in children with autism spectrum disorders who were hyperactive, impulsive, and distractible. Furthermore, the study showed that methylphenidate doses that are well tolerated in typically developing children with attention deficit/hyperactivity disorder were less well tolerated in children with autism spectrum disorders. Intolerable adverse effects included irritability, insomnia, loss of appetite, and increased stereotypic behavior. These results suggest that when treating children with autism spectrum disorders accompanied by hyperactivity and impulsiveness, the low-to-medium dose range is likely to produce modest benefit. However, attempts to achieve a greater benefit by pushing the dose upward are likely to encounter dose-limiting adverse effects.

See Also

► [Methylphenidate](#)

References and Reading

Research Units on Pediatric Psychopharmacology (RUPP) Autism Network. (2005). Randomized, controlled,

crossover trial of methylphenidate in pervasive developmental disorder. *Archives of General Psychiatry*, 62, 1266–1274.

Ristow, A., Westphal, A., & Scahill, L. (2011). Treating hyperactivity in children with pervasive developmental disorders. In E. Hollander, A. Kolevzon, & J. T. Coyle (Eds.), *Textbook of autism spectrum disorders* (pp. 479–492). Arlington: American Psychiatric Publishing.

Scahill, L., & Boorin, S. G. (2011). Psychopharmacology in children with PDD: Review of current evidence. In B. Reichow, P. Doehring, D. V. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-based practices* (pp. 231–243). New York: Springer.

Stimulus

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Synonyms

[Sensory stimuli](#)

Definition

A stimulus is defined as an environmental event that affects an individual's behavior through their sense modes, such as vision, hearing, smell, taste, touch, kinesthesia, and balance. Additionally, events such as dreams, mental images, and hallucinations are also considered stimuli and may affect behavior; however, these do not activate a specific sense mode. This makes their study and detection more difficult. Stimuli are dynamic and often studied in terms of stimulus changes in the environment, such as a light turning on or off or a sudden loud noise. Stimuli can also be the effects of internal bodily events such as the onset of a toothache, stomach pangs, and dizziness.

Stimuli are important components of a variety of autism treatments. They can be used to provide the context for behavior to be expressed or

withheld, delivered as prompts during learning trials, and – when overselected (as defined below) – can even interfere with learning. In this regard, these stimuli are called antecedents because they happen before the behavior of interest.

A phenomenon termed “stimulus overselectivity” (Lovaas et al. 1971) is often discussed in relation to individuals with a diagnosis of autism. Research has shown that when presented with a complex stimulus, individuals with autism may attend only to certain components of the stimulus (which may be irrelevant to the appropriate response). An implication of stimulus overselectivity is that many individuals with autism have difficulty in learning situations, requiring them to attend to more than one component of a stimulus (i.e., an instruction to put on a blue shirt).

In addition to its role before responding occurs, a stimulus can also influence behavior by following it. When this happens, the stimulus is called a consequence and can serve as a reinforcer or punisher. The use of certain stimuli both before and after the desired responding has been central to applied behavior analytic interventions aimed at enhancing learning, decreasing behavior problems, and improving quality of life for people with autism.

See Also

- [Consequence-Based Interventions](#)
- [Punishment](#)
- [Reinforcer](#)

References and Reading

- Burke, J., & Cerniglia, L. (1990). Stimulus complexity and autistic children's responsivity: Assessing and training a pivotal behavior. *Journal of Autism and Developmental Disorders*, 20, 233–253. <https://doi.org/10.1007/BF02284721>.
- Leader, G., Loughnane, A., McMoreland, C., & Reed, P. (2009). The effect of stimulus salience on over-selectivity. *Journal of Autism and Developmental Disorders*, 39, 330–338. <https://doi.org/10.1007/s10803-008-0626-y>.

- Lovaas, O. I., Schreibman, L., Koegel, R., & Rehm, R. (1971). Selective responding by autistic children to multiple sensory input. *Journal of Abnormal Psychology*, 77, 211–222.
- Michael, J. (2004). *Concepts and principles of behavior analysis* (Rev. ed.). Kalamazoo: Society for the Advancement of Behavior Analysis.
- Schreibman, L., & Lovaas, O. I. (1973). Overselective response to social stimuli by autistic children. *Journal of Abnormal Child Psychology*, 1, 152–168.

Stimulus Control

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Synonyms

[Discrimination](#); [Discrimination training](#); [Discriminative stimulus](#)

Definition

Stimulus control refers to the emission or suppression of a behavior contingent upon the presence or absence of a stimulus. Therefore, behavior is said to be under stimulus control when that behavior occurs more often in the presence of certain antecedent stimuli and does not occur in the absence of the same antecedent stimuli. When a behavior is under stimulus control, the frequency in which the behavior is emitted may be altered. Other measurable dimensions of behavior may also be affected in the presence of the stimulus such as latency to responding, the duration of the response emission, or the intensity in which the behavior is emitted. When a new skill is being taught, prompting hierarchies may be employed to evoke responding in the presence of the correct stimulus and not in the absence of that stimulus. A transfer of stimulus control occurs when those prompts are faded over time, and the behavior is emitted in the presence of the stimulus alone, without supportive prompts in place, and when the behavior does not

occur in the absence of that stimulus. For example, a person is taught to pick up the phone and say “Hello” when the phone rings, and they are taught to suppress the behavior of picking up the phone and saying “Hello” in the absence of the phone ringing.

See Also

- [Chaining](#)
- [Shaping of Behavior](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis (2nd edition)*. Upper Saddle River, NJ: Pearson.
- Miltenberger, R. G. (2011). *Behavior modification: Principles and procedures (5th edition)*. Belmont, CA: Cengage Learning.
- Sidman, M. (2008). Reflections on stimulus control. *The Behavior Analyst, 31*(2), 127–135.

Stimulus Fading

- [Fading](#)

Stimulus Overselectivity

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Definition

Stimulus overselectivity is an attentional abnormality common to individuals with autism spectrum disorder characterized by hyper-attentiveness to selected stimuli in the environment and lack of attention to other relevant stimuli.

Various behavioral and cognitive theories offer explanations for this particular phenomenon.

A behavioral perspective put forth by Lovaas, Schreibman, Koegel, and Rehm (1971), explains stimulus overselectivity as a narrowing of attention to a particular characteristic of a single stimulus or to distinct stimuli in a complex environment. Other authors have related this concept to the term “tunnel vision” (Rincover and Ducharme 1987). The sensory overload hypothesis, another possible explanation for overselectivity, posits that children with autism become overloaded with sensory input and therefore can only attend to a limited amount of sensory information at a given time. Alternatively, cognitive theories suggest that this phenomenon is the result of a generalized attentional bias in visual perception (such as a more detail-oriented style of information processing) in which they are unable to extract context-dependent relevant features from the stimuli or the environment (Hermelin 1976). Treisman (1969) suggested that such a phenomenon might occur if a child failed to switch attention between multiple cues. Stimulus overselectivity is not unique to autism spectrum disorders and has been observed in individuals with intellectual disability as well (Dickson et al. 2006; Wilhelm and Lovaas 1976).

In intervention settings, stimulus overselectivity can impact how the child learns new skills. For example, children with autism have been observed to fail to generalize newly learned skills across novel settings and individuals outside of the original treatment context (Rincover and Koegel 1975). Additionally, if this strategy of processing and responding is not addressed early in treatment, progress in the areas of social behavior and language acquisition can be limited (Rosenblatt et al. 1995). For this reason, some interventions have identified responding to multiple cues as a pivotal area that, if targeted, can have widespread effects on many areas of functioning (Koegel et al. 1999).

References and Reading

- Dickson, C. A., Wang, S. S., Lombard, K. M., & Dube, W. V. (2006). Overselective stimulus control in residential school students with intellectual disabilities.

- Research in Developmental Disabilities*, 27(6), 618–631. <https://doi.org/10.1016/j.ridd.2005.07.004>.
- Hermelin, B. (1976). Coding the sense modalities. In L. Wing (Ed.), *Early childhood autism: Clinical educational and social aspects* (2nd ed., pp. 135–168). Oxford: Pergamon Press.
- Koegel, L. K., Koegel, R. L., Harrower, J. K., & Carter, C. M. (1999). Pivotal response intervention I: Overview of approach. *The Journal of the Association for Persons With Severe Handicaps*, 24(3), 174–185.
- Lovaas, O. I., Schreibman, L., Koegel, R., & Rehm, R. (1971). Selective responding by autistic children to multiple sensory input. *Journal of Abnormal Psychology*, 77(3), 211–222. <https://doi.org/10.1037/h0031015>.
- Rincover, A., & Ducharme, J. M. (1987). Variables influencing stimulus overselectivity and “tunnel vision” in developmentally delayed children. *American Journal of Mental Deficiency*, 91(4), 422–430.
- Rincover, A., & Koegel, R. L. (1975). Setting generality and stimulus control in autistic children. *Journal of Applied Behavior Analysis*, 8(3), 235–246. <https://doi.org/10.1901/jaba.1975.8-235>.
- Rosenblatt, J., Bloom, P., & Koegel, R. L. (1995). Over-selective responding: Description, implications, and intervention. In R. L. Koegel & L. K. Koegel (Eds.), *Teaching children with autism: Strategies for initiating positive interactions and improving learning opportunities* (pp. 33–42). Baltimore: Paul H. Brookes.
- Treisman, A. (1969). Strategies and models of selective attention. *Psychological Review*, 76, 282–299.
- Wilhelm, H., & Lovaas, O. I. (1976). Stimulus over-selectivity: A common feature in autism and mental retardation. *American Journal of Mental Deficiency*, 81(1), 26–31.

Stimulus Preference Assessment

- [Preference and Reinforcer Assessment](#)

Story Grammar Analysis

- [Story Structure Decision Tree](#)

Story Grammar Assessment

- [Story Structure Decision Tree](#)

Story Structure Decision Tree

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Synonyms

[Narrative analysis](#); [Narrative assessment](#); [Story grammar analysis](#); [Story grammar assessment](#)

Definition

In typical development, narrative skills progress from simple descriptive sequences to more complex and elaborated episodic structures. A story structure decision tree is a graphic tool for guiding the narrative analysis of children’s stories. The decision tree is a flow chart containing a series of yes or no questions. Each “yes” answer moves the user to the next question/level, while a “no” response prompts the user to exit the flow chart whereby the child’s level of story grammar development is indicated. For an example, see Westby’s (2005) Story Grammar Decision Tree based on Stein and Glenn’s (1979) classic description of story grammar.

See Also

- [Narrative Assessment](#)

References and Reading

- Hughes, D., McGillivray, L., & Schmidek, M. (1997). *Guide to narrative language: Procedures for assessment*. Eau Claire: Thinking Publications.
- Paul, R. (2007). *Language disorders from infancy through adolescence: Assessment and intervention*. St. Louis: Mosby Elsevier.
- Stein, N., & Glenn, C. (1979). An analysis of story comprehension in elementary school children. In R. Freedle (Ed.), *New directions in discourse processing* (Vol. 2, pp. 53–120). Norwood: Ablex.

- Wallach, G. P. (2008). *Language intervention for school-age students: Setting goals for academic success*. St. Louis: Mosby Elsevier.
- Westby, C. (2005). Assessing and remediating text comprehension problems. In H. Catts & A. Kamhi (Eds.), *Language and reading disabilities* (2nd ed., pp. 157–232). Boston: Allyn & Bacon.

Strabismus

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Synonyms

[Cross eye](#)

Definition

Strabismus refers to misalignment of the eyes typically due to a lack of coordination between the extraocular muscles, which prevents bringing the eye gaze of each eye to the same point in space, thus preventing binocular vision. This will most notably affect stereopsis or depth perception and can have serious consequences with 30–50% of children with strabismus developing amblyopia or vision loss. Strabismus is one of the most common eye problems in children and affects about 4% of children less than 6 years of age. Strabismus can occur in one or both eyes and in any direction. For example, in esotropia, there is inward deviation of the nonfixing eye, and in exotropia, the nonfixing eye outwardly deviates. Strabismus is also classified based on time of onset, such as infantile, acquired, or secondary to another pathological process, such as cataract. Strabismus must be treated early in order to decrease the risk of amblyopia, maintain binocular vision, and minimize the psychosocial effects of looking different than one's peers. Treatment depends on the etiology and may involve a combination of eyeglasses, vision therapy, and

surgery, depending on the underlying reason for the misalignment.

Stranger Anxiety

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Definition

Stranger anxiety, which typically involves an overt display of mild to moderate emotional distress, is identified by observing how infants respond to the approach of an unfamiliar adult. Demonstration of signs of anxiety, or wariness, in response to an approaching stranger is a significant, universally observed, adaptive response that occurs in the course of typical child development. Although there is significant individual variation in the onset and severity of stranger anxiety, it is rare to observe such wariness during the first 6 months of life. However, by 8 months, stranger anxiety is commonly observed, and the phenomenon peaks around 12 months. Some factors that may influence stranger anxiety are temperament and attachment. Fussy, insecurely attached infants are more likely to respond negatively to the approach of a stranger, as compared to easy going, securely attached infants. Additionally, it is important to note that stranger anxiety is also influenced by a range of contextual factors, including whether or not the caregiver is present, the physical characteristics of the approaching stranger, the rapidity with which the stranger approaches, the physical positioning of the child during the approach, and the familiarity of the setting. Although individual differences impact the presentation of stranger anxiety, research suggests that children with autism do indeed display signs of stranger anxiety over the course of their development.

See Also

- [Anxiety](#)
- [Attachment](#)
- [Separation Anxiety Disorder](#)

References and Reading

- Dissanayake, C., & Crossley, S. A. (1996). Proximity and sociable behaviours in autism: Evidence for attachment. *Journal of Child Psychology and Psychiatry*, 37, 149–156.
- Greenberg, D. J., Hillman, D., & Grice, D. (1973). Infant and stranger variables related to stranger anxiety in the first year of life. *Developmental Psychology*, 9, 207–212.
- Sigman, M., & Ungerer, J. A. (1984). Attachment behaviors in autistic children. *Journal of Autism and Developmental Disorders*, 14, 231–244.
- Sroufe, L. A. (1977). Wariness of strangers and the study of infant development. *Child Development*, 48, 731–746.
- Tennes, K. H., & Lampl, E. E. (1964). Stranger and separation anxiety in infancy. *Journal of Nervous and Mental Disease*, 139, 247–254.

Straterra (TM)

- [Atomoxetine](#)

Strengthening

- [Reinforcement](#)

Stress Disorder

- [Posttraumatic Stress Disorder](#)

Stress Reactivity

- [Physiological Measures of Parental Stress](#)

Striatum

- [Basal Ganglia](#)
- [Caudate Nucleus](#)

Strong Narrative Assessment Procedure (SNAP), Second Edition

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Synonyms

[SNAP](#)

Description

The Strong Narrative Assessment Procedure (SNAP; Strong 1998) is an individually administered criterion-based method to measure various skills related to narrative discourse. Children from kindergarten to 8th grade are the appropriate candidates to be administered the SNAP. The SNAP assessment is divided into multiple steps; children recall relevant details of stories, summarize the stories, and answer questions that assess comprehension of the stories following specific instructions. Flexibility in transitioning between verbal and written language is required by students who are administered the SNAP. The ultimate goal of this assessment is to provide an approach to treatment that will support children when engaging in positive social interactions.

Although the targeted age groups for the SNAP procedures are children aged 6–13 years, comparison data are available only for children aged 7–10 years. SNAP is administered using tape recordings as well as corresponding wordless picture books: *Frog, Where are You?* (Mayer 1969), *Frog Goes to Dinner* (Mayer 1974), *A Boy, a Dog*

and a Frog (Mayer 1967), and *One Frog Too Many* (Mayer and Mayer 1975). The book *Frog Goes to Dinner* (Mayer 1974) is administered as a sample test that does not impact the final narrative score. The instructor then administers one of the remaining stories to elicit a sample narrative. The child is asked to listen to the audiotaped narrative while simultaneously looking at the accompanying wordless picture book. The manual suggests that the instructor should create a receptive environment for taking the SNAP by asking the child to listen to the stories through headphones or by the instructor leaving the room to allow the child to focus on the story (Rathvon 2007). After this step, the child is asked to retell the story and answer the 10 questions probing comprehension of the story (five factual and five inferential questions). When retelling the stories, the child is not permitted to use the picture books for assistance. Both the narrative and the responses of the child to the comprehension questions are recorded and transcribed.

Once the child's oral responses are transcribed into written form, the narrative sampling procedure is implemented by the instructor to identify "C-units" (Ward 2007). C-units consist of a main clause, modifiers, and any subordinating clauses. For example, a complete sentence such as "The boy was riding his bike on the street" is a C-unit. Narrative length is determined by counting the total number of words in a C-unit. Each C-unit is used to evaluate cohesion, syntax, grammar, and fluency in literacy (Ward 2007). Fluency is assessed by the total numbers of pauses per C-unit and determines whether self-corrections or abandoned utterances interfere with the child's retelling of the stories. Cohesion refers to linguistic components (e.g., grammar or vocabulary) used by a child to make connections among the sentences in the stories. For instance, conjunctions (e.g., and, but, or) are cohesive elements which connect and create fluency between a series of phrases and simple sentences. The SNAP manual provides examples of cohesive elements which can be administered. Lastly, syntax, i.e., the rules for constructing sentences, is assessed by counting the total number of clauses used in the stories (e.g., subordinate, adverbial, or nominal).

After these linguistic elements are analyzed, the story grammar components of a child's narrative stories are evaluated. Story grammar components are the specific elements that are universal to a narrative. These include information about setting, initiating events, internal responses, attempts to solve problems, and consequences of the stories. The initiating events refer to problems in the stories. For example, "the boy ran over a nail with his bike and got a flat tire" is an initiating event. The initiating events prompt internal responses from the character(s) in the stories. The internal response to the initiating event will be "the boy was very angry that his tire was flat." The initiating event induces attempts/plans to solve the problem. "The boy tried to fill the hole with a piece of gum" is a possible attempt to solve the problem caused by the initiating event. This plan/attempt may not work, leading to a second attempt at fixing the problem. Each narrative is also expected to include the consequence of the attempts. In this story, "the boy fixed the bike so that he was able to ride home" is the consequence of the attempts.

The instructor scores the child's responses to the story comprehension questions. These responses are determined to be either accurate or inaccurate based on the instructor's perspective. The SNAP manual provides instructions to convert a child's raw scores to frequency and percentage of narrative elements. Average scores for children ages 7–10 years are provided as normative data for comparison (Rathvon 2007). A score that falls more than one standard deviation below the mean is considered "inadequate." Means and standard deviations are provided in the manual to interpret the comprehension responses for children who are only 7 years old. Guidelines for scoring and interpreting the comprehension questions included the information for children who are not just 7 years old. However, empirical evidence is not provided to support children beyond 7 years of age (Ward 2007).

Historical Background

Carol J. Strong developed SNAP with the purpose of assessing the narrative skills of children with

language impairments (LI). These children normally demonstrate shorter narratives, more grammatical errors, fewer cohesive devices, and fewer story grammar elements compared to neurotypical children (Swanson et al. 2005; Ukrainetz and Gillam 2009). Although SNAP was originally designed for testing children with LI, it also is utilized to test children with Autism Spectrum Disorder (ASD) who perform poorly in narrative discourse. Research shows that children with ASD produce shorter narrative discourse with fewer connections between related events compared to neurotypical children (Tager-Flusberg 1995). Moreover, oral statements from children with ASD are less complex and syntactically correct (Capps et al. 2000).

In 1988, SNAP was field tested for the first time using samples of neurotypical children ($N = 39$) and children with LI ($N = 39$) ages 8–10 years. The collected narrative samples were analyzed to establish the stability of story length and cohesive adequacy over time (Strong 1989; Strong and Shaver 1991). Strong (1998) received funding from the ASHA foundation and Utah State University to reanalyze the previous samples for fluency, syntax, and story grammar. In 1998, additional data were collected from children aged 7 years ($N = 26$). These data were published in the SNAP manual for comparison purposes.

Psychometric Data

The SNAP test was first implemented in 1988. It collected and analyzed 312 narrative samples obtained from 39 neurotypical children and 39 children with LI, all between 8 and 10 years of age. Another sample included 13 typically developing children and 13 children with LI, all 7 years old. As the sample sizes are not deemed statistically adequate, the data should be evaluated with a caution. Moreover, the participants in the study were limited by geographical homogeneity as all were from the State of Utah.

Measurements of interrater reliability were obtained for both sample groups regarding agreement on transcription of the 312 narrative samples in the 1988 study. There was complete agreement

(i.e., 100% accuracy) over 64% of the total transcript and no more than a three-word disagreement over the remaining 36% of the transcripts (Strong 1998). There also was 99% agreement in Interrater reliability for segmentation of a selected number of transcripts (Strong 1998). For the sample year 1998, interrater reliability of transcription, segmentation, and coding was greater than 90% for all measurements (Strong 1998). In an independent investigation, John et al. (2003) reported interrater reliability for segmentation and coding of transcripts as 81% and 87%. No evidence of test-retest reliability is provided.

There is some evidence supporting the validity of SNAP. Construct validity was administered by comparing mean scores of the groups of neurotypical and language-impaired sample students. In the 1988 sample, only 62% of scores were significantly different between groups, and in the 1998 sample, only 54% of scores were statistically significant (Strong 1998). These results show that SNAP reliably differentiated between neurotypical children and language-impaired children. When children with ASD ($N = 17$) were compared to typically developing children, the groups did not differ on narrative length, semantic and syntactic measures, cohesion, clause development, story grammar, or number of complete episodes (Young et al. 2005). However, the groups did differ in their ability to answer the inferential comprehension questions. Criterion-related validity was not investigated.

By comparing test scores over time, the SNAP manual indicates that the four stimulus stories are equivalent in length, syntactic measures, complexity, cohesive density, and story grammar complexity (Strong 1998). John et al. (2003) found that participants ($N = 61$) showed highest accuracy in retelling the story *A Boy, a Dog and a Frog* (Mayer 1967). Moreover, when the scores of the other two stimulus stories were compared, the comparison of the scores were inferential (the book *Frog, Where are You?* (Mayer 1969) was not included in this analysis due to the sample story). John et al. (2003) suggested that clinicians may want to use *A Boy, a Dog and a Frog* (Mayer 1967) as the practice narrative and use the other three stories for test-retest purposes.

Clinical Uses

The ability to engage in narrative discourse is important for development. Competent use of narrative is critical both for assessing and engaging in social communication. Personal narrative is used on a daily basis to share details of events and other information with others. Narrative use is a pragmatic skill reflecting how an individual uses language in a social context. A speaker, when telling a story, should be able to recognize a listener's knowledge level and comprehension ability. Moreover, he/she should notice nonverbal cues indicating whether the listener appropriately understands the conversation, is bored, or is confused by the communication. Therefore, successful storytelling requires understanding of the perspective of the listener as well as understanding of other social communication skills of the listener.

As previously discussed, children with ASD demonstrate deficits in narrative discourse compared to same age neurotypical peers, especially regarding understanding another's perspective. Theory of Mind (ToM) is the theoretical basis for many interventions designed to improve the ability to understand another's perspective (for a review, see Baron-Cohen et al. 1993). Theory of Mind techniques help children with ASD to acknowledge that people feel and think differently from one another. Children with ASD have difficulty interpreting other people's facial expressions from various social contexts and situations and, as a result, have significant deficits in social interactions. For example, they often lack empathy towards others. These deficits are related to irregular activity within the amygdala and ventromedial prefrontal cortex (vmPFC) of the brain. In addition, these deficit factors are related to inadequacy in narrative discourse. For instance, there is a positive relationship between children's performance on a standard measure of ToM and their frequency of recognizing a character's thoughts and emotions from the narrative stories (Capps et al. 2000; Tager-Flusberg and Sullivan 1995). Children with ASD have poor scores in ToM and a relative inability to interpret the character's emotional details from the stories. Moreover,

children with ASD have lower linguistic skills which contribute to poor performance in storytelling. The poor quality of their storytelling is reflected mainly in their use of simple structure with limited cohesive devices and other literate language features that control to the quality of the story (Strong 1998).

Narratives are integral aspects of social communication and with a fundamental role in developing meaningful relationships with others. Thus, the assessment of narrative competency is important for children with ASD. SNAP was structured to identify areas of concern in narrative discourse in order to guide intervention goals and monitor progress over time. Understanding narrative language is related to an empirical behavioral foundation in children with ASD. Although the SNAP is one method to assess narrative language use in children with ASD, the sample sizes in SNAP studies are too small to provide accurate measures of reliability and validity. The SNAP might be appropriate to use in conjunction with a norm-referenced measure, such as the *Test of Narrative Language* (TNL; Gilliam and Pearson 2004).

See Also

- ▶ [Clinical Assessment](#)
- ▶ [Communication Assessment](#)
- ▶ [Criterion-Referenced Testing](#)
- ▶ [Literacy](#)
- ▶ [Narrative Assessment](#)
- ▶ [Pragmatic Communication](#)
- ▶ [Pragmatic Language Impairment](#)
- ▶ [Pragmatic Language Skills Inventory](#)
- ▶ [Pragmatics](#)
- ▶ [Social Communication](#)
- ▶ [Theory of Mind](#)

References and Reading

- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. J. (Eds.). (1993). *Understanding other minds: perspectives from autism*. Oxford, UK: Oxford University Press.
- Capps, L., Losh, M., & Thurber, C. (2000). "The frog ate the bug and made his mouth sad": Narrative

- competence in children with autism. *Journal of Abnormal Child Psychology*, 28(2), 193–204.
- Diehl, J. J., Bennetto, L., & Young, E. C. (2006). Story recall and narrative coherence of high-functioning children with autism spectrum disorders. *Journal of Abnormal Child Psychology*, 34(1), 87–102.
- Gilliam, R. B., & Pearson, N. A. (2004). *Test of narrative language*. Austin: PRO-ED.
- John, S. F., Lui, M., & Tannock, R. (2003). Children's story retelling and comprehension using a new narrative resource. *Canadian Journal of School Psychology*, 18, 91–113.
- Klapwijk, E. T., Aghajani, M., Colins, O. F., Marijnissen, G. M., Popma, A., van Lang, N. D., & Vermeiren, R. R. (2016). Different brain responses during empathy in autism spectrum disorders versus conduct disorder and callous-unemotional traits. *Journal of Child Psychology and Psychiatry*, 57(6), 737–747. <https://doi.org/10.1111/jcpp.12498>.
- Mayer, M. (1967). *A boy a dog and a frog*. New York: Dial Books for Young Readers.
- Mayer, M. (1969). *Frog, where are you?* New York: Dial Books for Young Readers.
- Mayer, M. (1974). *Frog goes to dinner*. New York: Dial Books for Young Readers.
- Mayer, M., & Mayer, M. (1975). *One frog too many*. New York: Dial Books for Young Readers.
- Rathvon, N. (2007). Review of the strong narrative assessment procedure. In B. S. Plake, J. C. Impara, & R. A. Spies (Eds.), *The seventeenth mental measurements yearbook*. Lincoln: Buros Institute of Mental Measurements. Retrieved on 1 Oct 2010 from Mental Measurements Yearbook database.
- Rosenblau, G., Kliemann, D., Lemme, B., Walter, H., Heekeren, H. R., & Dziobek, I. (2016). The role of the amygdala in naturalistic mentalising in typical development and in autism spectrum disorder. *The British Journal of Psychiatry*, 208(6), 556–564. <https://doi.org/10.1192/bjp.bp.114.159269>.
- Strong, C. J. (1989). *Stability of oral cohesion skills of language-impaired and normally developing school-aged children*. Unpublished doctoral dissertation, Utah State University, Logan.
- Strong, C. J. (1998). *The strong narrative assessment procedure*. Eau Claire: Thinking Publications.
- Strong, C. J., & Shaver, J. P. (1991). Stability of cohesion in the spoken narratives of language-impaired and normally developing school-aged children. *Journal of Speech and Hearing Research*, 34(1), 95–111.
- Swanson, L. A., Fey, M. E., & Hood, L. S. (2005). Use of narrative-based language intervention with children who have specific language impairment. *American Journal of Speech-Language Pathology*, 14(2), 131–143.
- Tager-Flusberg, H. (1995). "Once upon a rabbit": Stories narrated by autistic children. *British Journal of Developmental Psychology*, 13, 45–59.
- Tager-Flusberg, H., & Sullivan, K. (1995). Attributing mental states to story characters: A comparison on narratives produced by autistic and mentally retarded individuals. *Applied Psycholinguistics*, 16, 241–256.
- Ukrainetz, T. A., & Gillam, R. B. (2009). The expressive elaboration of imaginative narratives by children with specific language impairment. *Journal of Speech, Language, and Hearing Research*, 52(4), 883–898.
- Ward, A. (2007). Review of the strong narrative assessment procedure. In B. S. Plake, J. C. Impara, & R. A. Spies (Eds.), *The seventeenth mental measurements yearbook*. Lincoln: Buros Institute of Mental Measurements. Retrieved on 1 Oct 2010 from Mental Measurements Yearbook database.
- Young, E. C., Diehl, J. J., Morris, D., Hyman, S. L., & Bennetto, L. (2005). The use of two language tests to identify pragmatic language problems in children with autism spectrum disorders. *Language, Speech, and Hearing Services in Schools*, 36, 62–72.

Structural Equation Mixture Modeling

► Latent Variable Modeling

Structural Equation Modeling

► Latent Variable Modeling

Structured Behavioral Interventions

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Definition

Structured behavioral intervention is an umbrella term for interventions in which the therapist or teacher organizes the learning environment, selects the activity, and directs the individual with autism. Structured behavioral interventions are often implemented in a one-to-one setting

between a trained therapist and child. The therapist provides a clear cue or antecedent to initiate the desired behavior from the individual and uses positive reinforcement to strengthen the occurrence of the behavior following the antecedent. Often structured behavioral interventions are based on a breakdown of tasks into their component steps with a set plan for linking the steps together as the child masters each step and is gradually moving to more complex tasks. Structured behavioral interventions are often described as “didactic” and are contrasted with interventions in which the therapist follows the child’s lead and aims to expand on activities that the child initiates. Many school- and home-based programs are considered to be structured behavioral interventions, among which approaches based on applied behavioral analysis (ABA) are the most common.

Historical Background

Structured behavioral interventions began to be used in the education of children with autism spectrum disorders in studies on ABA conducted in the early 1960s. These initial studies incorporated experimenter-led tasks showing that children with autism could learn new skills if they received systematic instruction. In the mid-1960s, investigators demonstrated that similar procedures could be implemented in clinical settings and developed an instructional format now called discrete-trial training (DTT). DTT consists of small units of instruction implemented by a teacher in a one-to-one setting with a child. These units of instruction are composed of a cue, prompt, response, consequences, and intertrial interval. For example, in a DTT session aiming to enhance imitative behavior, the instructor would give a verbal cue “Do this” and visual cue (taps head), perhaps with a prompt such as lifting the child’s arm up to elicit the behavior and reinforce the child for displaying the desired behavior.

Other examples of structured behavioral interventions include (1) task analysis and chaining in which complex skills are broken down into their component steps to be taught individually and

then strung together and (2) script training, which focuses on using words, pictures, videos, or live models to show a sequence of appropriate social and verbal responses. Structured behavioral interventions have been implemented in individual and group formats in a range of settings, such as home-based intervention programs, specialized schools, and integrated classrooms.

Rationale or Underlying Theory

The use of structured behavioral interventions is based on the view that individuals with autism require didactic, systematic instruction because they are unable to learn in the same ways young typically developing children learn (e.g., playing creatively and interacting with others) and often have little interest in doing so because they are unresponsive to social reinforcement (e.g., verbal praise from parents). Structured behavioral interventions involve carefully prompting and reinforcing successes and providing instruction at a rapid pace in order to provide many learning opportunities and teach a wide range of skills efficiently.

Goals and Objectives

Structured behavioral interventions aim to place an individual with autism in an environment that optimizes the rate of learning and promotes the individual’s experiences of success. This is done by breaking complex tasks into component steps that can each be taught and built upon (e.g., identifying the steps involved in a task such as toothbrushing and teaching each task separately).

Treatment Participants

People with autism of any age are candidates for a structured behavioral intervention as long as a motivating positive reinforcer can be identified. Research suggests that the younger a participant starts in the intervention, the more significant are his or her improvements.

Treatment Procedures

A number of interventions are considered to be structured behavioral interventions, so specific treatment procedures vary by intervention. As a therapist- or teacher-led intervention, procedures are usually implemented in a one-to-one setting. Generally, each behavioral instruction is cued by a specific antecedent and followed by a consequence designed to either reinforce a correct response or extinguish an incorrect response.

Efficacy Information

Numerous single-case studies indicate that structured teaching approaches such as DTT, task analysis/chaining, and script training can be effective. Early intensive behavioral intervention (EIBI) programs that emphasize such methods (such as the Ivar Lovaas's UCLA You Autism Project) have also reported high success rates such as significant increases in IQ test scores, Vineland adaptive behavior scale scores, and integrated classroom placements.

Outcome Measurement

In single-case studies of structured behavioral interventions, outcome is usually measured by repeated, direct observations of changes in the rate of a specific target behavior, such as an increase in the display of a language skill or a reduction in an aberrant behavior. In EIBI studies, outcome measures usually include standardized tests of intelligence and adaptive behavior.

Qualifications of Treatment Providers

Structured behavioral interventions may be implemented by a variety of treatment providers, such as paraprofessionals, educators, and residential care providers. The interventions are often delivered under the supervision of a behavioral analyst.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Chaining](#)
- [Early Intensive Behavioral Intervention \(EIBI\)](#)

References and Reading

- Brown, F., & Bambara, L. M. (1999). Special series on interventions for young children with autism. *The Journal of the Association for Persons with Severe Handicaps*, 24(3), 131–241.
- Committee on Educational Interventions for Children with Autism National Research Council Staff. (2001). *Educating children with autism*. Washington, DC: National Academic Press.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- Lovaas, O. I. (2003). *Teaching individuals with developmental delays: Basic intervention techniques*. Austin: Pro-Ed.
- Lovaas, O. I., & Smith, T. (1989). A comprehensive behavioral theory of autistic children: Paradigm for research and treatment. *Journal of Behavior Therapy and Experimental Psychiatry*, 20(1), 17–29.
- Maurice, C., Green, G., & Luce, S. C. (Eds.). (1996). *Behavioral intervention for young children with autism: A manual for parents and professionals*. Austin: Pro-Ed.
- McClannahan, L. E. (2005). *Teaching conversation to children with autism: Scripts and script fading*. Bethesda: Woodbine House.
- McEachin, J. J., Smith, T., & Lovaas, O. I. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. *American Journal on Mental Retardation*, 4, 359–372.
- Rogers, S. J. (1998). Empirically supported comprehensive treatments for young children with autism. *Journal of Clinical Child Psychology*, 27, 168–179.
- Sallows, G. O., & Groupner, T. D. (2005). Intensive behavioral treatment for children with autism: Four-year outcome and predictors. *American Journal on Mental Retardation*, 110(6), 417–438.
- Schreibman, L. (2000). Intensive behavioral/psychoeducational treatments for autism: Research needs and future directions. *Journal of Autism and Developmental Disorders*, 30(5), 373–378.
- Smith, T. (2001). Discrete trial training in the treatment of children with autism. *Focus on Autism and Other Developmental Disabilities*, 16(2), 86–92.
- Smith, T., McAdam, D., & Napolitano, D. (2007). Autism and applied behavioral analysis. In P. Sturmey & A. Fitzner (Eds.), *Autism spectrum disorders: Applied behavioral analysis evidence, and practice* (pp. 187–234). Austin: Pro-Ed.

Structured Classrooms

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Synonyms

Visually organized learning environments

Definition

Learning environments are organized so that the curriculum (activities, schedule, physical environment) is clearly visually communicated to both the students and educational personnel (Iovannone et al. 2003). Structured environments have been demonstrated to be an essential part of programming for students with autism spectrum disorders (ASD) (Bodfish 2004; Iovannone et al. 2003; Mesibov and Shea 2010). Structured classrooms for students with ASD utilize the principles of Structured Teaching (an evidence-based approach devised by the TEACCH [Treatment and Education of Autistic and related Communication-handicapped CHildren] Program in North Carolina; Mesibov and Shea 2010).

Key features of structured classrooms are (Ball n.d.; Mesibov and Shea 2010; Mesibov et al. 2004):

- Physical structure – using furniture to demonstrate expectations and reduce distractions.
- Visual schedules – using objects, pictures, or the written word to show the student the sequence of events.
- Visually structured individual tasks that incorporate object, picture, and/or written instructions.
- Organizing a sequence of individual tasks using visual work/activity systems – using objects, pictures, letters, numbers, or the written word; tasks are organized to show the student what they have to do, how many tasks they need to do, how they are progressing,

when they will be finished, and what they are going to do next, for example, lining up the tasks on the students' left and having them move them to their right when completed.

- Using students' relative strengths to support weaker skills. For example, requiring a student to select a picture of the weather (utilizing strong visual skills) during a verbal discussion about the weather (thus supporting relatively weak verbal communication skills).
- Using students' special interests to increase engagement in learning. For example, teaching counting by having a student interested in trains count the number of train cars.
- Strategies to increase students' meaningful, spontaneous communication. For example, teaching a nonverbal student to hand the instructor a cup to request a drink.

Within these features of a structured classroom, the key to student success is that each is individualized according to the strengths and weaknesses of each student (Mesibov et al. 2004).

See Also

- Classroom Structure
- Culture and Autism
- Educational Interventions
- Pictorial Cues/Visual Supports (CR)
- Structured Teaching
- Visual Schedule
- Visual Supports

References and Reading

- Ball, J. (n.d.). Structured classrooms, virtual speaker by talk autism [video]. Retrieved January 25, 2011, from <http://www.talkautism.com/Components/Video/Video.aspx?v=59>
- Bodfish, J. W. (2004). Treating the core features of autism: Are we there yet? *Mental Retardation and Developmental Disabilities Research Reviews*, 10, 318–326.
- Iovannone, R., Dunlap, G., Huber, H., & Kincaid, D. (2003). Effective educational practices for students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 18, 150–165.

- Mesibov, G. B., & Shea, V. (2010). The TEACCH program in the era of evidence-based practice. *Journal of Autism and Developmental Disorders*, 40, 570–579.
- Mesibov, G., Shea, V., & Schopler, E. (2004). *The TEACCH approach to Autism Spectrum Disorders*. New York: Springer.

Structured Descriptive Assessment

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Synonyms

[SDA](#)

Definition

A structured descriptive assessment (SDA) uses principles of applied behavior analysis to describe the causes and consequences of a given behavior. A SDA typically occurs in the child's natural environment (e.g., the child's classroom) with no manipulation of environmental variables. The examiner records the antecedent of the child's behavior, target behavior, and consequence. For example, if the child might be engaging in self-harm, the examiner will record the antecedent of this behavior (e.g., the teacher asking a difficult question to the child), the child's behavior (e.g., biting of his hand), and the consequence (e.g., the child is sent out of the classroom with his aide). This type of assessment is often used to determine the cause of maladaptive behavior. It differs from functional behavior analysis as the examiner does not control for environmental variables.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Functional Behavior Assessment](#)

References and Reading

- Anderson, C., & Long, E. (2002). Use of a structured descriptive assessment methodology to identify variables affecting problem behavior. *Journal of Applied Behavioral Analysis*, 35, 137–154.
- English, C., & Anderson, C. (2006). Evaluation of the treatment utility of the analog functional analysis and the structured descriptive assessment. *Journal of Positive Behavior Interventions*, 8, 212–230.
- Slooman, K. N. (2010). Research trends in descriptive analysis. *The Behavior Analyst Today*, 11(1), 20–35.

Structured Environments

- [Structured Teaching](#)

Structured Teaching

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Synonyms

[Organized work systems](#); [Structured environments](#)

Definition

Structured teaching (often associated with the Treatment and Education of Autistic and Related Communication-Handicapped Children [TEACCH] method) refers to strategies that promote systematic and predictable instruction that supports individuals with autism spectrum disorders to better understand their environment and live in it more independently. Structured teaching provides an environment that is organized with clear concrete visual information that defines the environment. Structure is provided by the adults, the physical organization of the environment, materials, and routine. It is important to

conceptualize that the structured teaching is not faded or removed over time if adjusted to meet the changing needs of the individual with autism spectrum disorder.

The components of structured teaching relate directly to the characteristics of autism spectrum disorder. Many individuals with autism spectrum disorder are visual learners, and structured teaching relies heavily on visual supports. Effective processing of information is often a challenge for individuals with autism spectrum disorders, and structured teaching highlights routines, beginnings, and endings. Attention and sensory deficits impact how individuals with autism spectrum disorders learn, and structured teaching ensures that the environment is modified to reduce distractions and excessive stimulation. Structure teaching benefits individuals with autism spectrum disorder by reducing anxiety through predictability, it increases learning through rule-based strategies, and it promotes independence by encouraging reliance on the system rather than people.

Structured teaching is an effective lifelong tool that capitalizes on organizing the individual with autism spectrum disorder's world, through organizational and temporal structure.

See Also

- [Task Analysis](#)
- [Visual Supports](#)

References and Reading

- Hume, K., & Carnahan, C. (2008). *Steps for implementation: Structured work systems and activity organization*. Chapel Hill: The National Professional Development Center on Autism Spectrum Disorders, Frank Porter Graham Child Development Institute, The University of North Carolina.
- Panerai, S., Ferante, L., Caputo, V., & Impellizzeri, C. (1998). Use of structured teaching for treatment of children with autism and severe and profound mental retardation. *Education and Training in Mental Retardation and Developmental Disabilities*, 33, 367–374.

Student Characteristics and Teachers' Decisions to Use 1:1 Instruction

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Definition

Evidence-based treatments for children with autism spectrum disorder (ASD) include behavioral interventions that teach communication, cognitive, social, and adaptive skills. One-to-one instruction is a critical component of evidence-based instruction for students with ASD, but is not used as often as recommended. Two recent studies suggest that student characteristics may affect teachers' decisions to select types of one-to-one treatment (e.g., pivotal response training [PRT] vs. discrete trial training [DTT]) and implement them in the classroom. In the first, teachers reported choosing DTT for students who had more significant cognitive delays, who struggled with compliance, and who did not imitate or attend in less structured settings (Stahmer et al. 2005). In the second, children's higher sensory symptoms, lower social approach, lower verbal skills, and higher self-regulation difficulties were found to be associated with more frequent 1:1 instruction (Nuske et al. 2019).

Historical Background

There has been a steady rise in the number of children receiving ASD intervention services through schools (U.S. Department of Education

2017), but the type and intensity of treatment vary greatly (White et al. 2007). There is little research on factors that predict the type and intensity of school-based treatment children with ASD receive. Such factors may include different school resources, such as staffing, or teacher characteristics, including teacher preferences, training, pedagogy, and skill (Stahmer et al. 2005). Some studies have found associations between these characteristics and use of behavioral interventions for children with ASD (Hogan et al. 2015), but others have found no association (Segall and Campbell 2012; Morrier et al. 2011; Mandell et al. 2013; Stahmer et al. 2015).

Variability in treatment delivery also may be driven by child characteristics (Stahmer et al. 2005), such as their language level, severity of ASD symptoms, or regulatory difficulties, all of which may consciously or unconsciously drive teachers' decisions about who is best suited for one-to-one (1:1) instruction or a particular type of treatment. The authors have found that child characteristics account for about half of the variance in the frequency with which children received 1:1 intervention in school (Lushin et al., under review), but that study did not investigate whether specific child characteristics are associated with teachers' decisions to provide 1:1 intervention.

Related research has examined whether child characteristics predict outcomes as a result of different types of treatment. Two evidence-based treatment approaches for children with ASD include DTT (Lovaas 2003; Lovaas and Buch 1997) and PRT (Schreibman et al. 2015). Both DTT and PRT are based on principles of applied behavior analysis (ABA; e.g., operant conditioning, systematic reinforcement; Ringdahl et al. 2009; Smith and Iadarola 2015) and are administered by an adult working 1:1 with a child. A key difference between these two approaches is that DTT is adult-directed and incorporates external reinforcement (e.g., token reward for play initiation), while PRT is child-directed and incorporates naturalistic reinforcement (e.g., smiles/engagement for play initiation; Koegel et al. 1999a, b). Both DTT (Anderson et al. 1987; Lovaas 1987; McEachin et al. 1993) and PRT (Koegel et al. 1999b; Nefdt et al. 2010) are effective practices

for children with ASD. Research has indicated that DTT is most effective when implemented in combination with naturalistic interventions to facilitate generalization of skill development across settings and support the development of spontaneous behaviors, such as play initiation (Duffy and Healy 2011; National Research Council 2001; Schreibman et al. 2015; Smith 2001).

Children who made the greatest gains through DTT demonstrated greater social engagement (social approach, joint attention, and imitation), were younger, and had a higher IQ (Smith et al. 2015). Children who made the greatest gains through PRT were younger (Baker-Ericzén et al. 2007), had higher social approach, and had higher expressive language ability (not overall IQ). They also had higher positive affect and appropriate toy contact and lower stereotyped/repetitive vocalizations (Fossum et al. 2018). Research by Schreibman and colleagues suggests that the profile of children who made greater gains with PRT was not the same as those who responded most to DTT (Schreibman et al. 2009; Sherer and Schreibman 2005), indicating utility in identifying unique responder profiles for different treatments. Among children with ASD who received a comprehensive program that incorporates DTT and PRT, fewer self-regulation difficulties over the course of the academic year were associated with higher cognitive skill gains (Nuske et al. 2017). This body of work has therefore identified some overlapping and some unique child characteristics of higher responders to DTT and PRT.

Such research could be used to inform a personalized medicine approach (i.e., individual tailoring of treatment) for children with ASD, by selecting interventions for a child based on their characteristics or behavioral profile (Masi et al. 2017; Ousley and Cermak 2014). Personalized medicine approaches have been recently applied to clinical psychology, so to increase the efficacy of behavioral and cognitive treatments (Drake et al. 2009; Fisher 2015; Jeon and Fava 2015; Kasari et al. 2014; Lei et al. 2012; Ng and Weisz 2016). Individualizing treatments may result in better outcomes. These approaches bring new methodological considerations and challenges

and necessitate research designs that allow for treatment individualization via systematized trial and error, for example, in the case of adaptive, sequential, and crossover designs (for review, Lillie et al. 2011). Importantly, individualization may be most effective when completed a priori to starting treatment. This approach entails selecting treatments based on children's behavioral profile *before* treatment or programming begins, so to potentially avoid spending time using treatments or components of treatments that are not suited to a particular child.

Teachers may be using this approach already, either from reading of the research or from their own experience, when deciding which intervention to use with which children. Indeed, teachers note that individualizing instruction based on a child's strengths and weaknesses is critically important (Stahmer et al. 2005).

Current Knowledge

Teachers report choosing DTT for students who have more significant cognitive delays, who struggle with compliance, and who do not imitate or attend in less structured settings (Stahmer et al. 2005). This study established that teachers do factor in students' characteristics when deciding for whom to implement 1:1 instruction. However, due to the qualitative design of the study, the study was unable to empirically test whether frequency of treatment delivery and teacher selection of treatment approach relate to their students' characteristics. The second study by Nuske et al. (2019) explored this question by quantitatively examining the associations between child characteristics and teacher selection and frequency of use of PRT and/or DTT with children with ASD in their classrooms. Based on the study by Stahmer et al. (2005), researchers hypothesized that teachers would be more likely to use DTT with children who had more severe ASD symptoms, lower cognitive skills, and higher regulatory difficulties. Based on prior research regarding PRT outcomes (Schreibman et al. 2009; Sherer and Schreibman 2005), researchers hypothesized that children with higher social and semantic

pragmatic skills and higher verbal ability would be more likely to receive PRT. Researchers also explored associations with sex and race and described the variability in use of DTT and PRT across children and classrooms.

Results from Nuske et al. (2019) showed that teachers reportedly provided more 1:1 instruction in general to children who have more sensory and self-regulation difficulties, lower verbal skills, and less social approach. Findings suggest that teachers give more treatment to children with more obvious impairments. This is encouraging as it suggests that teachers are tapping into some key observable difficulties of particular children and reportedly allocating 1:1 instruction resources from their classrooms accordingly (Linstead et al. 2017). Previous research has shown that children with more significant impairments make the fewest gains in any treatment (Fossum et al. 2018; Smith et al. 2015; Thurm et al. 2007). Therefore, teachers with more severely impaired students face significant challenges in fostering positive outcomes for their students. The results show that teachers are attempting to address these students' needs by providing them with more individualized intervention.

In the context of limited classroom resources (e.g., time, space, availability of teacher aids), teachers are using 1:1 interventions to treat children who demonstrate the most obvious need. While promising in one sense, it also means that other children may be inadvertently neglected, though they may still benefit from 1:1 treatment. All children in autism support classrooms have an educational classification of ASD, meaning that their school teams have identified significant support needs consistent with ASD. However, those who are easier to manage or who have, relatively speaking, more skills are not receiving potentially beneficial treatment as other children with ASD, though they may have significant developmental delays relative to their typically developing peers. Given that for many children the bulk of their services are received through school, this may mean that many children with ASD are not receiving the 1:1 instruction that they need.

In Nuske et al. (2019), age was not associated with implementation of 1:1 instruction. These

findings suggest that teachers are not considering students' age when implementing either PRT or DTT. In the context of findings of higher positive outcomes in PRT or DTT for younger children with ASD (Baker-Ericzén et al. 2007; Smith et al. 2015), this suggests again that teachers are making decisions preferentially based on perceived students' support needs.

Findings from Nuske et al. (2019) also suggested that teachers did not differentially use DTT or PRT based on child characteristics, despite the theoretical notion that different profiles of children may be better suited to one over the other (Vivanti 2017). Evidence suggests that matching children with ASD to certain treatment styles based on their pretreatment behavioral profile may support positive outcomes (Schreibman et al. 2009; Sherer and Schreibman 2005). However, researchers found the same student characteristics predicted teachers' use of DTT and PRT. Given that the ASD field, as well as the mental health field in general, is moving toward a personalized medicine approach to treatment (i.e., to individualize treatments decision based on individual behavioral profiles; Drake et al. 2009; Jeon and Fava 2015; Ng and Weisz 2016; Stahmer et al. 2011), these results provide important insights into the current state of affairs regarding teachers' decisions about their students' educational programming. The results show that teachers are not reportedly using one treatment type more frequently over the other with children with different characteristics. The research on pretreatment behavioral profiles and optimal treatment fit is promising, but has only recently begun; therefore, further work is needed before teacher consultation models around child-treatment fit can be developed.

Future Directions

First, given that both of the studies on the association between the characteristics of children with ASD and teacher implementation of evidence-based practice rely on teacher's own report of 1:1 instruction use, these data are subject to the social desirability bias, in that teachers may have

reported higher frequencies than were actually implemented. However, this may not differentially apply to children of certain characteristics. Nevertheless, observation methods would have strengthened the measurement of intervention implementation and is recommended for future research.

Second, in Nuske et al. (2019) while ASD symptoms were measured via teacher report, cognitive and language abilities and self-regulation difficulties were measured by study staff. Therefore the latter may not have necessarily mapped onto teacher's own perspectives of children's cognitive and language abilities and self-regulation difficulties and decisions regarding the frequency with which teachers should use 1:1 across different children in their classroom. Notably, however, lower verbal skills and higher self-regulation difficulties were found to be associated with more frequent 1:1 instruction in Nuske et al. (2019). Still, future research should incorporate teachers' measures of cognitive and language abilities and self-regulation difficulties to further investigate this issue.

Third, the extent to which teachers' decisions to use treatments for more severely impacted children were well-informed or considerate of individual student needs was not specifically examined in this study. Future work could identify factors such as the extent to which cognitive biases, intentions, and adherence to coaches' recommendations affect teachers' use of 1:1 intervention.

Finally, since research in this area has just begun, it is not yet known how these findings apply outside of school or community early intervention provider settings. Future research should involve providers across a range of treatment settings, including community behavioral health settings or in private practice.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Classroom Management](#)
- [Classroom Structure](#)
- [Education](#)

- **Elementary and Secondary Education Act (No Child Left Behind)**
- **Free Appropriate Public Education**
- **Pivotal Response Training**
- **Treatment Priorities**

References and Reading

- Anderson, S. R., Avery, D. L., DiPietro, E. K., Edwards, G. L., & Christian, W. P. (1987). Intensive home-based early intervention with Autistic children. *Education and Treatment of Children*, 10(4), 352–366.
- Baker-Ericzen, M. J., Stahmer, A. C., & Burns, A. (2007). Child demographics associated with outcomes in a community-based pivotal response training program. *Journal of Positive Behavior Interventions*, 9(1), 52–60.
- Drake, R. E., Cimpian, D., & Torrey, W. C. (2009). Shared decision making in mental health: Prospects for personalized medicine. *Dialogues in Clinical Neuroscience*, 11(4), 455–463.
- Duffy, C., & Healy, O. (2011). Spontaneous communication in autism spectrum disorder: A review of topographies and interventions. *Research in Autism Spectrum Disorders*, 5(3), 977–983.
- Fisher, A. J. (2015). Toward a dynamic model of psychological assessment: Implications for personalized care. *Journal of Consulting and Clinical Psychology*, 83(4), 825.
- Fossum, K.-L., Williams, L., Garon, N., Bryson, S. E., & Smith, I. M. (2018). Pivotal response treatment for preschoolers with autism spectrum disorder: Defining a predictor profile. *Autism Research*, 11(1), 153–165. <https://doi.org/10.1002/aur.1859>.
- Hogan, A., Knez, N., & Kahng, S. (2015). Evaluating the use of behavioral skills training to improve school staffs' implementation of behavior intervention plans. *Journal of Behavioral Education*, 24(2), 242–254. <https://doi.org/10.1007/s10864-014-9213-9>.
- Jeon, H. J., & Fava, M. (2015). Chapter eight – Novel study designs for clinical trials in mood disorders. In M. Tohen, C. L. Bowden, A. A. Nierenberg, & J. R. Geddes (Eds.), *Clinical trial design challenges in mood disorders* (pp. 87–104). Amsterdam: Elsevier Academic. <https://doi.org/10.1016/B978-0-12-405170-6.00008-7>.
- Kasari, C., Kaiser, A., Goods, K., Nietfeld, J., Mathy, P., Landa, R., . . . Almirall, D. (2014). Communication interventions for minimally verbal children with Autism: Sequential multiple assignment randomized trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 53(6), 635–646. <https://doi.org/10.1016/j.jaac.2014.01.019>
- Koegel, L. K., Koegel, R. L., Harrower, J. K., & Carter, C. M. (1999a). Pivotal response intervention I: Overview of approach. *Journal of the Association for Persons with Severe Handicaps*, 24(3), 174–185. <https://doi.org/10.2511/rpsd.24.3.174>.
- Koegel, L. K., Koegel, R. L., Shoshan, Y., & McNERNEY, E. (1999b). Pivotal response intervention II: Preliminary long-term outcome data. *Journal of the Association for Persons with Severe Handicaps*, 24(3), 186–198.
- Lei, H., Nahum-Shani, I., Lynch, K., Oslin, D., & Murphy, S. A. (2012). A “SMART” design for building individualized treatment sequences. *Annual Review of Clinical Psychology*, 8(1), 21–48. <https://doi.org/10.1146/annurev-clinpsy-032511-143152>.
- Lillie, E. O., Patay, B., Diamant, J., Issell, B., Topol, E. J., & Schork, N. J. (2011). The n-of-1 clinical trial: The ultimate strategy for individualizing medicine? *Personalized Medicine*, 8(2), 161–173. <https://doi.org/10.2217/pme.11.7>.
- Linstead, E., Dixon, D. R., French, R., Granpeesheh, D., Adams, H., German, R., . . . Kornack, J. (2017). Intensity and learning outcomes in the treatment of children with autism spectrum disorder. *Behavior Modification*, 41(2), 229–252.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55(1), 3–9.
- Lovaas, O. I. (2003). *Teaching individuals with developmental delays: Basic intervention techniques*. Austin: PRO-ED.
- Lovaas, O. I., & Buch, G. (1997). Intensive behavioral intervention with young children with autism. In *Prevention and treatment of severe behavior problems: Models and methods in developmental disabilities*. Pacific Grove: Wadsworth Publishing.
- Mandell, D. S., Stahmer, A. C., Shin, S., Xie, M., Reisinger, E., & Marcus, S. C. (2013). The role of treatment fidelity on outcomes during a randomized field trial of an Autism intervention. *Autism: The International Journal of Research and Practice*, 17(3), 281–295.
- Masi, A., DeMayo, M. M., Glozier, N., & Guastella, A. J. (2017). An overview of Autism spectrum disorder, heterogeneity and treatment options. *Neuroscience Bulletin*, 33(2), 183–193. <https://doi.org/10.1007/s12264-017-0100-y>.
- McEachin, J. J., Smith, T., & Lovaas, O. I. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. *American Journal of Mental Retardation: AJMR*, 97(4), 359–372; discussion 373–391.
- Morrier, M. J., Hess, K. L., & Heflin, L. J. (2011). Teacher training for implementation of teaching strategies for students with Autism spectrum disorders. *Teacher Education and Special Education*, 34(2), 119–132. <https://doi.org/10.1177/0888406410376660>.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press. <https://doi.org/10.17226/10017>.
- Nefdt, N., Koegel, R., Singer, G., & Gerber, M. (2010). The use of a self-directed learning program to provide

- introductory training in pivotal response treatment to parents of children with autism. *Journal of Positive Behavior Interventions*, 12(1), 23–32.
- Ng, M. Y., & Weisz, J. R. (2016). Annual research review: Building a science of personalized intervention for youth mental health. *Journal of Child Psychology and Psychiatry*, 57(3), 216–236. <https://doi.org/10.1111/jcpp.12470>.
- Nuske, H. J., Kane, C., Rump, K., Pellecchia, M., Maddox, B., Reisinger Blanch, E., & Mandell, D. (2017). The impact of self-regulation skills on academic outcomes in minimally-verbal school-age children with autism. *International meeting for autism research*.
- Nuske, H. J., Pellecchia, M., Lushin, V., Rump, K., Seidman, M., Ouellette, R. R., . . . Mandell, D. S. (2019). Do student characteristics affect teachers' decisions to use 1:1 instruction? *Journal of Autism and Developmental Disorders*, 49(7), 2864–2872.
- Ousley, O., & Cermak, T. (2014). Autism spectrum disorder: Defining dimensions and subgroups. *Current Developmental Disorders Reports*, 1(1), 20–28. <https://doi.org/10.1007/s40474-013-0003-1>.
- Ringdahl, J. E., Kopelman, T., & Falcomata, T. S. (2009). *Applied behavior analysis and its application to autism and autism related disorders*. New York: Springer. https://doi.org/10.1007/978-1-4419-0088-3_2.
- Schreibman, L., Stahmer, A. C., Barlett, V. C., & Dufek, S. (2009). Brief report: Toward refinement of a predictive behavioral profile for treatment outcome in children with Autism. *Research in Autism Spectrum Disorders*, 3(1), 163–172. <https://doi.org/10.1016/j.rasd.2008.04.008>.
- Schreibman, L., Dawson, G., Stahmer, A. C., Landa, R., Rogers, S. J., McGee, G. G., . . . others. (2015). Naturalistic developmental behavioral interventions: Empirically validated treatments for autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45, 2411–2428.
- Segall, M. J., & Campbell, J. M. (2012). Factors relating to education professionals' classroom practices for the inclusion of students with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6(3), 1156–1167. <https://doi.org/10.1016/j.rasd.2012.02.007>.
- Sherer, M. R., & Schreibman, L. (2005). Individual behavioral profiles and predictors of treatment effectiveness for children with autism. *Journal of Consulting and Clinical Psychology*, 73(3), 525–538. <https://doi.org/10.1037/0022-006X.73.3.525>.
- Smith, T. (2001). Discrete trial training in the treatment of Autism. *Focus on Autism and Other Developmental Disabilities*, 16(2), 86–92. <https://doi.org/10.1177/108835760101600204>.
- Smith, T., & Iadarola, S. (2015). Evidence base update for Autism spectrum disorder. *Journal of Clinical Child and Adolescent Psychology: The Official Journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53*, 44(6), 897–922. <https://doi.org/10.1080/15374416.2015.1077448>.
- Smith, T., Klorman, R., & Mruzek, D. W. (2015). Predicting outcome of community-based early intensive behavioral intervention for children with Autism. *Journal of Abnormal Child Psychology*, 43(7), 1271–1282. <https://doi.org/10.1007/s10802-015-0002-2>.
- Stahmer, A. C., Collings, N. M., & Palinkas, L. A. (2005). Early intervention practices for children with Autism: Descriptions from community providers. *Focus on Autism and Other Developmental Disabilities*, 20(2), 66–79.
- Stahmer, A. C., Schreibman, L., & Cunningham, A. B. (2011). Toward a technology of treatment individualization for young children with autism spectrum disorders. *Brain Research*, 1380, 229–239. <https://doi.org/10.1016/j.brainres.2010.09.043>.
- Stahmer, A. C., Reed, S., Lee, E., Reisinger, E. M., Connell, J. E., & Mandell, D. S. (2015). Training teachers to use evidence-based practices for Autism: Examining procedural implementation fidelity. *Psychology in the Schools*, 52(2), 181–195. <https://doi.org/10.1002/pits.21815>.
- Thurm, A., Lord, C., Lee, L.-C., & Newschaffer, C. (2007). Predictors of language acquisition in preschool children with Autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(9), 1721–1734. <https://doi.org/10.1007/s10803-006-0300-1>.
- U.S. Department of Education. (2017). *39th Annual report to congress on the implementation of the individuals with Disabilities Education Act, 2017*.
- Vivanti, G. (2017). Individualizing and combining treatments in autism spectrum disorder: Four elements for a theory-driven research agenda. *Current Directions in Psychological Science*, 26(2), 114–119.
- White, S. W., Scahill, L., Klin, A., Koenig, K., & Volkmar, F. R. (2007). Educational placements and service use patterns of individuals with Autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(8), 1403–1412. <https://doi.org/10.1007/s10803-006-0281-0>.

Studies to Advance Autism Research and Treatment

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Definition

A federally funded network of studies was mandated by the Children's Health Act (2000) that

was to include at least five centers of excellence in autism research. In response to this mandate, five NIH institutes (NIMH, NICHD, NINDS, NIDCD, and NIEHS) came together to develop a network program. This program of work was concerned with aspects of diagnosis, early detection, prevention, and treatment as well as the etiology of autism. This program was the successor to the original Collaborative Program of Excellence in Autism which had been funded by NICHD and NIDCD.

Following a competitive process, eight centers around the country were funded using the mechanism. These included:

- Boston University (Helen Tager-Flusberg, Ph.D., principal investigator)
- Kennedy Krieger Institute (Rebecca Landa, Ph.D., principal investigator)
- Mt. Sinai Medical School (Eric Hollander, M.D., principal investigator)
- University of California, Los Angeles (Marian Sigman, Ph.D., principal investigator)
- University of North Carolina at Chapel Hill (Joe Piven, M.D., principal investigator)
- University of Rochester (Patricia Rodier, Ph.D., principal investigator)
- University of Washington (Geri Dawson, Ph.D., principal investigator)
- Yale University (Fred Volkmar, M.D., principal investigator)

In addition to studies conducted at each site, the network also worked on a series of collaborative projects.

Klin, A., Saulnier, C. A., Sparrow, S. S., Cicchetti, D. V., Volkmar, F. R., & Lord, C. (2007). Social and communication abilities and disabilities in higher functioning individuals with autism spectrum disorders: The Vineland and the ADOS. *Journal of Autism and Developmental Disorders*, 37(4), 748–759.

Landa, R., & Garrett-Mayer, E. (2006). Development in infants with autism spectrum disorders: A prospective study. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 47(6), 629–638.

Losh, M., & Piven, J. (2007). Social-cognition and the broad autism phenotype: Identifying genetically meaningful phenotypes. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 48(1), 105–112.

Moy, S. S., Nadler, J. J., Young, N. B., Perez, A., Paige Holloway, L., Barbaro, R. P., et al. (2007). Mouse behavioral tasks relevant to autism: Phenotypes of 10 inbred strains. *Behavioural Brain Research*, 176(1), 4–20.

Schultz, R. T. (2005). Developmental deficits in social perception in autism: The role of the amygdala and fusiform face area. *International Journal of Developmental Neuroscience*, 23(2-3), 125–141.

Sigman, M., Spence, S. J., & Wang, A. T. (2006). Autism from developmental and neuropsychological perspectives. *Annual Review of Clinical Psychology*, 2, 327–355.

Tager-Flusberg, H. (2004). Strategies for conducting research on language in autism. *Journal of Autism and Developmental Disorders*, 34(1), 75–80.

Study Design Impact on Prevalence

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References and Reading

- Arndt, T. L., Stodgell, C. J., & Rodier, P. M. (2005). The teratology of autism. *International Journal of Developmental Neuroscience*, 23(2-3), 189–199.
- Dawson, M., Soulieres, I., Gernsbacher, M. A., & Motttron, L. (2007). The level and nature of autistic intelligence. *Psychological Science*, 18(8), 657–662.
- Hollander, E., Bartz, J., Chaplin, W., Phillips, A., Sumner, J., Soorya, L., et al. (2007). Oxytocin increases retention of social cognition in autism. *Biological Psychiatry*, 61(4), 498–503.

Definition

Prevalence of autism spectrum disorders (ASDs) consists in the number of people identified with an ASD in a defined population divided by the total number of people in the population at a given time. Prevalence is usually expressed as x people per 1,000 with an ASD in the area studied. Determining the prevalence of the ASDs depends on the identification of people with an ASD in a defined population. Prevalence studies must first

define the “case definition” by describing who will be counted as having an ASD and by what standards. Some prevalence studies have sought to identify specific subtypes such as autistic disorder or Asperger’s disorder, while others have focused on the whole spectrum. The way ASDs are defined and the methods used to identify the conditions can have an impact on prevalence estimates. One major difference among prevalence studies is whether the focus is on counting individuals who have already been diagnosed or classified as having an ASD or on identifying individuals who have the diagnostic profile of certain ASDs who may or may not have already been diagnosed. There are several methods used to identify people with an ASD in the population. These include counting the number of people already identified with an ASD either through service systems or registries or by conducting surveys asking if a person has an ASD diagnosis. Other methods include trying to not only count people already diagnosed with an ASD but also identify people who meet the diagnostic criteria but have not yet been diagnosed with an ASD. These efforts attempt to screen records or people in the population to find already-known and currently unknown cases of ASDs. All prevalence studies require that you know the base population of people from whom you are trying to identify the individuals with an ASD.

The number of people receiving services for identified ASDs has increased substantially since the early 1990s. This has been documented in the US Department of Education counts of children identified with autism for special education purposes and among statewide service systems for people with developmental disabilities (Centers for Disease Control and Prevention [CDC] 2009, 2012; Newschaffer et al. 2005). However, the number of individuals receiving services for ASDs is likely to be an underestimate of all people with ASDs in the population. In fact, methods that rely on service systems, registries, and surveys of people already diagnosed under-ascertain people without a diagnosis. Epidemiologic studies which systematically screen the population to also identify individuals who meet the case definition but who were not previously classified as having an

ASD generally result in higher and more complete prevalence estimates. Screening can focus on groups at risk who have been identified for some type of developmental concern in order to maximize resources and minimize false positives. A multisite study using this method found that 21% of the children identified with ASDs did not have a previous classification documented in their records (CDC 2012). Screening can try to identify ASDs among the general population. These studies generally report the highest prevalence estimates of ASDs (Baron-Cohen et al. 2009; Kim et al. 2011), but also must address challenges of low response rates and the possibility of over identification of people who have autism-like traits who may not have the constellation of functional impairments necessary to identify autism as a developmental disability. Overall, studies using a variety of methods have recently estimated about 1% of children with ASDs; however, estimates vary based on the methods used. The fact that there have been changes in the conceptualization of autism coupled with gains in community awareness over the past two decades poses challenges in knowing exactly how many people have really had autism or a related condition throughout history. However, it is clear that more people are identified with an ASD today than ever before, resulting in major needs for supports and services across the life span.

See Also

- Incidence
- Prevalence

References and Reading

- Baron-Cohen, S., Scott, F. J., Allison, C., Williams, J., Bolton, P., Matthews, F. E., et al. (2009). Prevalence of autism spectrum conditions: UK school-based population study. *The British Journal of Psychiatry*, 194, 500–509.
- California Department of Developmental Services. (2007). *Changes in the CA caseload: An update June 1987 through June 2007*. Sacramento: California Department of Developmental Services. Accessed December

- 7, 2009, from http://www.dds.ca.gov/Autism/docs/AutismReport_2007.pdf
- Centers for Disease Control and Prevention. (2009). Prevalence of autism spectrum disorders – autism and developmental disabilities monitoring network, United States, 2006. *Morbidity and Mortality Weekly Report Surveillance Summaries*, 58, 1–20.
- Centers for Disease Control and Prevention. (2012). Prevalence of autism spectrum disorders – autism and developmental disabilities monitoring network, United States, 14 sites, 2008. *Morbidity and Mortality Weekly Report Surveillance Summaries*, 61, 1–19.
- Kim, Y. S., Leventhal, B. L., Koh, Y. J., Fombonne, E., Laska, E., Lim, E. C., et al. (2011). Prevalence of autism spectrum disorders in a total population sample. *American Journal of Psychiatry*, 168, 904–912.
- Newschaffer, C., Falb, M., & Gurney, J. (2005). National autism prevalence trends from United States special education data. *Pediatrics*, 115, 277–282.
- Rice, C. E. (2011). The changing prevalence of the autism spectrum disorders. *American Family Physician*, 83(5), 515–520.

Stuttering

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Synonyms

Disfluency

Definition

Stuttering is a speech disorder in which the flow of speech is disrupted by involuntary repetitions (e.g., a-a-a-apple) and prolongations of sounds (e.g., Ssssit down), syllables (e.g., pat-pat-patriotic), words, or phrases; involuntary fillers (e.g., “um,” “like”); or silent pauses in which the speaker is unable to produce sounds. Stuttering can be variable in certain situations depending on the anxiety level connected with that activity. Although the precise etiology is not known, both genetics and neurophysiology are thought to contribute. Speech therapy is the primary treatment. It

generally aims to increase fluency or modify the stuttering behaviors.

Stuttering typically begins during childhood – approximately 2.5% of children under the age of 5 stutter. It can last throughout the lifetime, although approximately 75% of individuals who begin stuttering during the preschool years recover by their early teens. The overall prevalence of stuttering is approximately 1% of the population. If severe and persistent, it can significantly impact academic, social, and vocational outcomes. Differential diagnosis is required to distinguish between word retrieval and language formulation difficulties which can also cause disfluent speech.

See Also

- Fluency and Fluency Disorders
- Speech Therapy

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- American Speech-Language Hearing Association. (2017, March 29). *Stuttering*. Retrieved 17 Mar 2017 from <http://www.asha.org/public/speech/disorders/stuttering.htm>

Subacute Necrotizing Encephalomyelopathy

- Leigh Disease

Subacute Necrotizing Encephalopathy

- Leigh Disease

Sub-cortical Ganglia

► Basal Ganglia

Sub-Saharan Africa and Autism

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Historical Background

Sub-Saharan Africa is the sum of 46 countries located fully or partially south of the Sahara Desert. As a region, Sub-Saharan Africa (SSA) is as heterogeneous as its vast geographical scale would suggest: within it are more than 24 million km², more than a billion people (World Bank 2015), and more than 2000 languages (Epstein and Kole 1998). Because of this intraregion diversity, Pan-African organizations such as the African Union often divide SSA into several local subregions. Nevertheless, SSA is seen as a distinct geographical and political region by many global organizations, including the World Bank and the United Nations (UN) (United Nations 2014; World Bank 2014). Most of the 46 countries in SSA are low income – 41% of all people in SSA live on less than 1.25 USD per day (United Nations 2015). Although almost 90% of the world's children and adolescents live in low- and middle-income countries (LMICs), only about 10% of randomized controlled mental health trials for this population are conducted there (Kieling et al. 2011). Clinical interventions, service developments, research, and policy work for children and adolescents in SSA have focused almost exclusively on communicable diseases such as malaria, tuberculosis (TB), and human immunodeficiency virus (HIV), and on reducing infant mortality (United Nations 2015); there has been comparatively little focus on noncommunicable

diseases and neurodevelopmental disorders such as autism spectrum disorder (ASD) (Franz et al. 2017). Over the past few decades, successful decreases in mortality among children under 5 years of age in SSA (Garenne and Gakusi 2006; Mathers and Loncar 2006; Murray et al. 2007; Rajatnaram et al. 2010; United Nations 2015) have been followed by international calls to increase focus on the needs of children with developmental delays or disabilities, including ASD (Engle et al. 2007; Scherzer et al. 2012).

Autism research and resources within SSA are disproportionately located in South Africa and Nigeria (Abubakar et al. 2016b; Ruparelia et al. 2016), two of the most populous nations in the region and two of the richest by GDP per capita (World Bank 2013). A recent review found that more than half of high-quality peer-reviewed research on autism in SSA was conducted in South Africa (51%) and another 23% in Nigeria (Abubakar et al. 2016b). South Africa also differs from much of the rest of SSA in important ways; for example, it is the only nation in the region with less than 40% of its population under 15 years of age and one of only two regional nations with a fertility rate below the world average (Central Intelligence Agency 2016). For more information about autism research, treatment, and resources in South Africa, refer to its national entry referenced in the “See Also” section of this entry. Research from South Africa will not be discussed in this entry to maintain a complementary focus on the rest of SSA.

The history of autism in Sub-Saharan Africa is a relatively short one. Early thought hypothesized that autism was a culturally bound disorder and essentially nonexistent in SSA. It was not until 1978 that a psychiatrist, Victor Lotter, brought international attention to autism in the region, having identified children with autism in Ghana, Kenya, Nigeria, South Africa, Zambia, and Zimbabwe (Lotter 1978). Thirty of the children screened by Lotter had “autistic-like” behaviors, and nine met DSM criteria for the disorder. Lotter observed that the African diagnosed group was broadly comparable to a group of children diagnosed with autism in Britain in terms of the group's breadth of verbal and intellectual abilities, sex

ratio, breadth of socioeconomic status, and occurrence of epilepsy (Lotter 1978). Lotter also observed that repetitive movements and object-centered ritualistic behaviors were uncommon in the African sample, though contradictory findings have since been presented by several studies (Bakare and Ikegwuonu 2008; Bello-Mojeeed et al. 2011; Dhadphale et al. 1982; Holford 1994; Izuwah et al. 2016; Lagunju et al. 2014; Springer et al. 2013; Szabo and Aber 1992; Wessels and Pompe van Meerdervoort 1979). International dialogue continued regarding the “open question” of the universality of ASD (Sanua 1984), but reviews of case reports around the world have since concluded that ASD has substantial worldwide prevalence (Fombonne 2003). Increasing interest in ASD in Africa is currently demonstrated in, among other indicators, the growing number of regional scientific studies on the topic (Ametepee and Chitiyo 2009; Bakare and Munir 2011a). A recent review concluded that 83% of peer-reviewed research on autism in SSA (including South Africa) had been conducted after 2006, indicating a dramatically increased interest over the previous decade (Abubakar et al. 2016b).

The global prevalence of ASD is now estimated at 1–2% (CDC 2014; Elsabbagh et al. 2012; Kim et al. 2011). Around 50% of the African population is under 18 years of age, and given trends in birth rates around the world, the United Nations predicts that 40% of the world’s children will live in Africa by 2050 (UNICEF 2014). Although no population-based studies of ASD have yet been performed in SSA, due in part to barriers to accessing “gold standard” diagnostic tools in research, it has been suggested that there is little reason to believe regional rates of ASD will be lower than elsewhere (Abubakar et al. 2016b; Durkin et al. 2015; Franz et al. 2017). It has also been suggested that rates of ASD may be higher in SSA, following a pattern of greater reported prevalence of intellectual disabilities in South Africa and LMICs compared to in high-income countries (Adnams 2010; de Vries et al. 2013; World Health Organization 2011). The need for a global perspective on ASDs was publicly affirmed by the World Health Organization (WHO) in 2008 when its General Assembly

adopted a resolution designating the second day of April each year as “World Autism Awareness Day.” In 2013, the WHO Executive Board adopted a resolution entitled “Comprehensive and coordinated efforts for the management of autism spectrum disorders (ASDs)” (World Health Organization 2014). The resolution was supported by more than 60 member countries and calls upon Member States – including 40 countries in SSA – to address the needs of people with ASD worldwide (World Health Organization 2013).

Legal Issues: Mandates for Service

There are currently no legal mandates for ASD-specific services in any country in SSA. Political will to enact such legislation is limited by the lack of empirical data regarding the prevalence of autism in countries in SSA (Abubakar et al. 2016b; Ametepee and Chitiyo 2009; Bakare and Munir 2011a; Franz et al. 2017). Forty countries in SSA are members of the WHO and, as such, adopted the 2013 resolution on autism spectrum disorders (World Health Organization 2013), which urges member states to expand capacity for services, develop and implement policies, promote research and awareness, and focus on community-based treatment. The resolution focuses on an implementation period from 2013 to 2020. Yet while the WHO has firmly acknowledged ASD as a public health concern (World Health Organization 2013, 2014), no countries in SSA currently have effective policies or “good practice” guidelines in place for assessment, treatment, education, and adult support of persons with ASD.

Overview of Current Treatments and Centers

There are very few specialist resources in SSA for individuals with ASD and related neurodevelopmental disorders (de Vries 2016). In 2015, there were only 50 specialist child and adolescents psychiatrists in the entire continent and a much

smaller number of developmental pediatricians (de Vries et al. 2013). Additionally, it has been noted that published cases in Africa tend to be of children from higher socioeconomic backgrounds (Ametepee and Chitiyo 2009). The most likely explanation for this is that wealthier individuals live in closer proximity to medical centers in major cities and are therefore more likely to be able to access services. This observation that socioeconomic status is a correlate for access to services is analogous to similar observations in the USA (Durkin et al. 2010). Nonverbal rates for children with autism in SSA have been reported as high as 71%, compared to only 25% in the USA (Bakare and Munir 2011b), and an autism diagnosis in SSA often co-occurs with epilepsy or intellectual disability. This suggests that reported cases are skewed toward the more severe and recognizable cases with comorbidities, indicating a severely underestimated prevalence of more moderate cases (Bakare and Munir 2011b).

Even when a diagnosis is given, it is difficult to find appropriate services. There are far too few mental healthcare facilities, personnel trained to work with autistic children, and specialist educational and behavioral interventions compared to the estimated prevalence of autism in SSA (Bakare and Munir 2011b). Many Africans have spiritual beliefs about psychiatric disorders, evident in the perceived etiology of autism among both caregivers and the general public. In a survey of Nigerian pediatric and psychiatric nurses, 40% cited preternatural causes of autism such as ancestral spirits or actions of the devil (Bakare et al. 2008). Supernatural explanations of autism can influence treatment-seeking behaviors of caregivers by encouraging use of spiritualists and traditional healers before other professionals.

Treatment in SSA is limited by a lack of validated measures for diagnosing and treating ASD in an African context. Additionally, very little systematic data about ASD in SSA is available, due in part to the absence of centralized bodies tasked with monitoring available services. Designing treatments in this context thus presents a methodologically difficult task. Recent reviews of published literature have emphasized the need to invest more in identifying potential screening

and diagnostic tools for the SSA context, as access to such tools can contribute to early identification of children with ASD (Abubakar et al. 2016b; Ametepee and Chitiyo 2009; Bakare and Munir 2011a; Franz et al. 2017). Preliminary studies have indicated some tools with moderate validity. These include a study in Uganda that adapted and extended the Ten-Question Questionnaire (TQQ) into a 23-item questionnaire, modestly successful in identifying subgroups of children at high risk of being diagnosed with ASD (Kakooza-Mwesige et al. 2014), as well as a study in Tanzania that evaluated a culturally adapted Childhood Autism Rating Scale as a structured tool to diagnose ASD locally – after ensuring that the play interactions, materials used, and social routines used to probe the child's behavior were familiar to the child – concluding that the tool had excellent discriminative validity and acceptable levels of sensitivity and specificity (Harrison et al. 2014). Because of the remarkable cultural and linguistic diversity within SSA (Epstein and Kole 1998), tools that are validated in one region must be systematically validated in each new region. The few intervention studies that exist have limited sample sizes, are largely cross-sectional, and lack evaluation of long-term impact (Abubakar et al. 2016b). Altogether, the precedence of interventions for children with ASD in SSA is not yet adequate to plan large-scale effective intervention strategies in SSA.

As there is no legal mandate for support of families with ASD, the bulk of support and services for families of children with ASD are provided by nongovernmental organizations (NGOs); existing NGOs also raise awareness about autism in Africa (Newton and Chugani 2013). These organizations are a significant part of the ASD treatment landscape in SSA. It should also be noted that the majority of African countries, including South Africa, have few specialist educational facilities for school-aged children with ASD (de Vries 2016). Moreover, in most parts of SSA, there appear to be no residential treatment facilities specifically for adults with autism, either with or without intellectual disability. For a list of some of the organizations engaged in providing resources and services and raising

awareness as of publication, refer to the “[Resources and Readings](#)” section appended to the entry.

Overview of Research Directions

Research output from SSA regarding autism is scarce. This contributes to the broader “10–90% divide” in research: while almost 90% of the world’s children and adolescents live in low- and middle-income countries (LMICs), only about 10% of randomized controlled mental health trials for this population are conducted there (Kieling et al. 2011). Global reviews of peer-reviewed publications on autism have come to the same conclusion. One found only 120 data-driven publications on autism from SSA, even before screening them for methodological rigor, compared to 511,560 publications from North America and 57,577 from Europe (Franz et al. 2017). More than half of published autism research in SSA has been carried out in South Africa alone, and of the research conducted outside South Africa, nearly half of that is from Nigeria (Abubakar et al. 2016b). Research centers in SSA specializing in autism are scarce. For specific research center information sorted by country, refer to the “[Resources and Readings](#)” section appended to the entry.

This lack of literature from SSA may be due to several factors. Firstly, there is a lack of regional expertise in ASD. In 2013, there were only 50 specialist child and adolescent psychiatrists across the entire continent of Africa and a much smaller number of developmental pediatricians (de Vries et al. 2013). Secondly, there is not sufficient investment in autism research to enable widespread methodological rigor (Ruparel et al. 2016). This includes a lack of investment in producing standardized tools that have been validated in the African context, a significant barrier to data-driven research toward screening and diagnosis of autism (Abubakar et al. 2016b). For more information on the preliminary validation of tools for diagnosis and screening, refer to the “[Overview of Current Treatments and Centers](#)” section of the entry. Broader involvement from

multidisciplinary clinical and academic colleagues across all academic medical centers, particularly in the development of validated tools for screening and diagnosis, remains an important need.

Of the limited research in SSA, most focuses on the psychosocial aspects of assisting someone with ASD. This focus on health workers and caregivers for children already diagnosed with ASD may be due to the difficulty of additional diagnosis and the minimal resources available for such a task (Abubakar et al. 2016b; Franz et al. 2017). Additionally, this focus on the psychosocial aspects of ASD may reflect an increasing appreciation for the significant burden on quality of life in African communities due to ASD and a growing interest in managing and averting this burden (Abubakar et al. 2016a). These psychosocial studies largely focus on three themes: (a) awareness levels among various subpopulations, (b) quality of services provided to children with ASD and challenges for caregivers, and (c) sociocultural aspects regarding ASD, including cultural explanatory models for the etiology of ASD (Abubakar et al. 2016b). Most of these studies were carried out in Nigeria by Bakare and colleagues (Abubakar et al. 2016b; Bakare et al. 2008, 2009a, b, c, 2011, 2015). A study in Tanzania developed an intervention to raise awareness of ASD and help caregivers learn some basic behavioral intervention strategies, including teaching of basic social skills (e.g., eye contact and imitating) and self-help skills (e.g., feeding) (Harrison et al. 2014, 2016). This intervention was found to be feasible to implement, and almost all participating caregivers found it to be useful. Findings regarding cultural and social policies are described in greater detail in the “[Cultural Factors and Controversies](#)” section of the entry.

A few studies have attempted to estimate the burden of ASD in SSA. Prevalence of ASD in this region, as in most low- and middle-income countries (LMICs), is still unclear (Bakare and Munir 2011a; Newton and Chugani 2013; World Health Organization 2013). A recent review found only a single population-level study aimed at documenting the prevalence of ASD in a country in SSA (Kakooza-Mwesige et al. 2014) and not a

single case-control study aimed at examining a comprehensive set of potential risk factors (Abubakar et al. 2016a). Most of these studies have used convenience sampling, sourced largely from hospitals and specialist units for children with special needs, which skews the prevalence and comorbidities of ASD in the sample population. For example, a study of 2320 patients at a pediatric neurological clinic in Nigeria diagnosed 54 patients with ASD, at an estimated prevalence of 2.3%, and found neurological comorbidities in 75.5% of those diagnosed (Lagunju et al. 2014). A single community-based study in Uganda surveyed 1169 children aged 2–9 in the Kampala District – half-urban, half-rural – wherein eight children had a positive diagnosis of ASD, resulting in an unadjusted prevalence for ASD of 6.8/1000, which the authors suggested was likely underrepresentative (Kakooza-Mwesige et al. 2014). All studies included in recent reviews have found a higher prevalence among boys compared to girls, in line with sex ratios in other global regions (Abubakar et al. 2016b; Ametepee and Chitiyo 2009; Bakare and Munir 2011a; Franz et al. 2017).

Overview of Training

Autism awareness, even among health professionals, is low in most parts of SSA. While a significant proportion of studies of ASD in SSA have investigated awareness of ASD among various subpopulations, most of these were carried out in a single country, Nigeria. An important step in gauging knowledge of ASD among health workers has been the development of the Knowledge about Child Autism among Health Workers (KCAHW) questionnaire, a validated and standardized tool for such evaluations in the Nigerian context. Bakare and colleagues developed this questionnaire and evaluated its psychometric properties using a sample of psychiatric nurses; they found that the measure had excellent internal consistency and adequate test-retest reliability (Bakare et al. 2008). Using this questionnaire to evaluate a sample of 134 health workers in Nigeria, Bakare and colleagues noted that a significant

percentage still held negative or false beliefs about the etiology, treatability, and preventability of ASD (Bakare et al. 2009a). In a related study, the same sample of health workers exhibited their most salient knowledge gaps in symptoms of obsessive behavior and impairments in social interaction (Bakare et al. 2009b). While general health workers exhibited poor knowledge of ASD, Bakare and colleagues also found that knowledge of ASD was quite good among final-year medical students, though important knowledge gaps were still noted in this population (Bakare et al. 2015). Eseigbe and collaborators (2015) used the KCAHW questionnaire to evaluate knowledge of ASD among a sample of 167 Nigerian medical doctors. They observed that pediatricians and psychiatrists had the best relative knowledge of ASD, that the most significant knowledge gap was regarding the onset of ASD and its comorbidities, and that the least significant gap was regarding communication impairments (Eseigbe et al. 2015). Igwe and collaborators (2011) found that even among Nigerian pediatric and psychiatric nurses, there was a significant deficit in ASD knowledge, particularly regarding the initial onset of ASD (Igwe et al. 2011).

Documented interventions, in peer-reviewed literature, intended to increase awareness of ASD are rare. In Tanzania, one such study reported a successful intervention intended to improve caregiver skills, focusing on basic behavioral strategies such as teaching basic social skills (e.g., making eye contact and imitating) and self-help skills (e.g., feeding) (Harrison et al. 2014, 2016). Education of healthcare workers is crucial for early diagnosis of ASD, so that early intervention programs can be delivered when they are most effective. For specific organizational resources, refer to the “[Resources and Readings](#)” section appended to the entry.

Cultural Factors and Controversies

There is a significant lack of awareness regarding ASD, particularly in rural areas where specialist care is effectively nonexistent (Ruparel et al.

2016). This lack of awareness, in combination with a lack of evidence-based treatments, has contributed to misconceptions about the nature of the disorder and the proper means for diagnosing and managing it. In a survey of Nigerian pediatric and psychiatric nurses, 40% cited preternatural causes of autism such as ancestral spirits or actions of the devil (Bakare et al. 2008). Yet while supernatural powers are a common attributed cause of autism, they are by no means the only common attribution. Attributed etiologies in SSA populations regarding mental health and developmental disorders, including autism, often span a wide range of beliefs, even within an individual. In a survey of caregivers in Ethiopia, explanations for the condition of a child with ASD included a mix of biomedical explanations (e.g., head injuries, given by 30.4% of participants), birth complications (25.5%), and supernatural explanations (e.g., spirit possession (40.2%) and sinful acts (27.5%)) (Tilahun et al. 2016). Gona et al. (2015) conducted in-depth interviews and focus groups with parents and professionals in Kenya – spanning communities in urban and rural settings with high and low literacy levels, Christian and Muslim faiths, and a range of educational levels. In line with the aforementioned survey of caregivers in Ethiopia by Tilahun et al. (2016), all participants in the Kenyan study by Gona et al. (2015) described preternatural causes (evil spirits, witchcraft, and curses) as well as medical causes (infections, drugs, birth complications, and genetics) as contributors to the etiology of ASD (Gona et al. 2015). All participants also indicated use of a range of treatment options, regardless of socioeconomic status or cultural background, that included visits to hospitals for “modern” healthcare treatments as well as consultations with traditional and spiritual healers (Gona et al. 2015). While these studies should not be hastily generalized to the rest of SSA, current literature would suggest that pluralistic perceived etiologies and preferred treatments of ASD in SSA cross socioeconomic, cultural, and religious divides. Accordingly, researchers strongly suggest that healthcare professionals should take care to explore the cultural beliefs of African families and account for the pluralism contained within,

thereby ensuring sensitivity to cultural nuances in beliefs regarding a child’s condition (Gona et al. 2015; Tilahun et al. 2016).

The challenges facing caregivers of children with ASD and caregivers’ common coping strategies are also broad and pluralistic. Common challenges include stigma, lack of appropriate health services, financial burdens, and heavy practical and emotional burdens (Gona et al. 2016). Coping strategies among a sample of Kenyan parents of children with ASD included problem-focused aspects (e.g., diet management and respite care) as well as emotional-focused aspects (e.g., belief in supernatural powers, prayers, and spiritual healing) (Gona et al. 2016). Among caregivers in Ethiopia, coping mechanisms for dealing with stigma of ASD were most commonly support from friends (reported by 76.5% of participants) and prayer (reported by 57.8% of participants) (Tilahun et al. 2016).

Access to specialist education facilities for school-aged children with ASD is rare (de Vries 2016). A survey of caregivers in Ethiopia reported that educational provision was their single greatest unmet need (reported by 74.5% of participants), even greater than treatment by a health professional (reported by 47.1% of participants). A study in Zimbabwe examined common barriers to including children with ASD in mainstream classrooms; these barriers included social rejection, communication impairments, and behavioral challenges of children with ASD (Majoko 2016). Collaborating with stakeholders to promulgate training for regular teachers and to enhance social support services may be effective strategies for encouraging inclusion of children with ASD in mainstream educational settings; these strategies may be more feasible than providing widespread specialist educational facilities (Majoko 2016).

Documented interventions to improve caregiver skills are rare in peer-reviewed literature (Abubakar et al. 2016b; Ametepee and Chitiyo 2009; Bakare and Munir 2011a; Franz et al. 2017). One such intervention was successfully implemented in Tanzania with a focus on improving basic behavioral strategies for caregivers such as teaching basic social skills (e.g., making eye contact and imitating) and self-help skills (e.g., feeding) (Harrison et al.

2014, 2016). In designing an intervention, cultural validation is essential to achieving the desired impact, as local cultural behaviors may substantially impact the manifestation and visibility of ASD. For example, a study in South Africa noted that direct eye contact with elders is discouraged and potentially seen as disrespectful among the general population; in this context, relying on avoidance of eye contact as an indicator for ASD would be less effective (Grinker et al. 2012). Education of the public is crucial for early diagnosis of ASD and minimizing stigma. NGOs are making significant contributions to autism awareness in SSA – refer to the “[Resources and Readings](#)” section appended to the entry for a partial list of specific organizations that are contributing to minimizing the impact of stigma and maximizing the availability of resources. Much of the existing research literature focuses on the psychosocial aspects of ASD for the child and for their families. This may be a favorable indicator of a greater appreciation for the significant burden on quality of life due to ASD in African communities as well as a growing interest in developing best practices for managing and averting this burden (Abubakar et al. 2016b).

See Also

- [Ethiopia and Autism](#)
- [South Africa and Autism](#)

Resources and Readings

1. Botswana
 - a. Camphill Community Rankoromane Trust
Camphill Rankoromane School
P.O. Box 2224, Gaborone
Phone: +267 32303.
Email: camphill.friends@iname.com
Website: <http://camphillbotswana.drupalgardens.com/>
 - b. Motse Wa Badiri Camphill
P.O. Box 142
Otse Botswana
Phone: +267 33727.
Fax: +267 33727.
- Email: mwb@info.bw
Website: www.info.bw/~mwb/progdis.html
- c. Dinaletsana Developmental Centre
Isang Close, Plot 449
Gaborone
Phone: +267 3105105
Fax: +267 3105101
Email: dinaletsana@gmail.com
Website: <http://www.facebook.com/groups/dinaletsana.botswana>
2. Cameroon
 - a. FADINE (Foundation for Africans with Disabilities and Neglect)
Website: <http://www.autismaroundtheglobe.org/countries/Cameroon.asp>
3. Cote D'Ivoire
 - a. Autism Community in Africa-Cote d'Ivoire
Website: <http://autismcommunityofafrica.org>
Phone: +1 443-718-1824
 - b. Autism Community of Africa
Website: http://www.autismaroundtheglobe.org/countries/Ivory_Coast.asp
4. Ethiopia
 - a. Joy-Center for Children with Autism and Related Developmental Disorders
Phone: +251 11 320 6159/60, +251 911 21053.
Email: Joy4autism@yahoo.com
 - b. Nehemiah Autism Center
P.O. Box 6294.
Addis Ababa
Phone: +251 930012652
Email: nehemiah.center@yahoo.com
Website: <http://www.neh-autism.org>
5. Ghana
 - a. Autism Awareness Care and Training (AACT)
P.O. Box OS-3043
Osu, Accra
Phone: +233 21 22472.
Fax: +233 0 21 762 619
Email: aact@ghana.com
Website: www.aact.org.gh
 - b. Haven International Center for Special Education

- Phone: 011 233 242649617
 Email: info@haven-international.org
 Website: <http://mygoalautism.org/haven-project/>
- c. HopeSetters Autism Center
 Golden Age Youth Training Center,
 Community 7
 Tema, Accra
 Phone: +233-24-217-2405/+233-20-984-5445
 Email: info@hopesettters.org
 Website: www.hopesettters.org
- d. MultiKids Academy
 Number M3, (Near America House),
 Ajringano Road
 New Otinshie, East Legon, Greater Accra
 Phone: +233 (0) 202966871
 Email: info@multikidsggh.com
- e. New Horizon Special School
 P.O. Box 0108.
 Osu, Accra
 Phone: +233 (0) 21 77287.
 Fax: +233 (0) 21 77287.
 Email: nhorizongh@gmail.com
 Website: www.newhorizon-school-gh.com
- f. Reyo-Paddock School
 Near American House
 East Legon Area, Accra
 Phone: 233 205 387 561 (Ghana)
 44 7988 526 754/44 7886 388 589
 Email: hanotoo@hotmail.co.uk
- g. Woodfield Manor
 Adenta Municipality
 Phone: 030 393 083.
 Email: woodfieldmanor@aol.co.uk
 Website: <http://www.creatingnewbeginnings.co.uk/woodfield-manor-school-gh>
6. Kenya
 a. Autism Center of Kenya
 Phone: 0734575504 0704308368
 Email: info@autismcenterkenya.or.ke
 Website: <http://www.autismcenterkenya.or.ke/>
 b. Autism Foundation of Kenya and Autism Resource Centre
 P.O. Box 288-00502 Karen Nairobi
 Phone: +254 20 88265.
 Fax: +254 20 88378.
- c. Autism Society of Kenya
 P.O. 1762-00200 Suraj Plaza Ngara
 Basement 1 Limuru Rd., Nairobi
 Phone: 254 0 20 3742582/3742603
 Email: kenyaaautism@yahoo.com
 Website: <http://www.autismkenya.org/programs.html>
- d. National Autistic Center – Kenya NAC-K
 Acorn Special Tutorials 417 Gitanga
 PO Box 21436 00505, Nairobi
 Phone: 254 020 3864436/7 0733 785
 Email: 234acorn@clubinternetk.com
- e. Special Education Professionals
 c/o Gertrude's Children's Hospital
 P.O. Box 42325 Nairobi – 0010.
 Phone: 254 02 3763474 ext. 322
 Email: SEP_professionals@yahoo.com
 Website: www.sepkenya.com
7. Liberia
 a. Liberia Renaissance Foundation
<http://www.autismaroundtheglobe.org/countries/liberia.asp>
8. Malawi
 a. Hope for Autism Foundation in Malawi
<http://www.autismaroundtheglobe.org/countries/Malawi.asp>
9. Namibia
 a. Autism Namibia
 P.O. Box 5043, Windhoek
 Phone: 264 61 224 562
 Fax: 264 61 228 255
 Email: petaut@africaonline.com.na
 Website: www.autism-namibia.org
10. Niger
 a. Association Espoir Pour L'Autisme Au Niger
 BP 11509 Niamey
11. Nigeria
 a. Autism Associates
 1 Bode Thomas St. off Bode Thomas Rd. Onipan, Lagos
 Phone: 234 1 8506353; 234 702 8193870; 234 803 9112839; 234 1 8506354
 Email: info@autismassociatesnigeria.org/autismassociates@yahoo.co.uk

- Website: www.autismassociatesnigeria.org
- b. Blazing Heart Autism Center
16A Circular Road, Elekahia Housing Estate, Port Harcourt
Phone: 08029998884
Fax: 08038852183
Email: info@blazingheartfoundation.org
Website: www.blazingheartautism.org
 - c. Centre for Autism and Developmental Disabilities (CADD Nigeria)
1 Chika Omo St. Off Nnebisi Rd. Asaba, Delta State
Email: caddng@yahoo.com
 - d. National Society for Autism, Nigeria (NASAN)
27 Libreville St. Off Aminu Kano Crescent Wuse II, FCT Abuja
Email: info@societyforautismnigeria.org
Website: www.societyforautismnigeria.org
 - e. Nigerian Autistic Society
P.O. Box 7173 Wuse, Abuja
Phone: 234 0 9 523 667.
 - f. NWATU Autism Foundation
No. 4, Idowu Rufai St. Ago Palace Way-Okota -Lagos P.M.B 039 Festac Town-Lagos
Phone: 00234 1 8992331/00234 8058458995
Email: autism4nigeria@yahoo.co.uk
 - g. OLG Health Foundation and Autism Centre
8 Faith Avenue Woji, Port Harcourt Rivers State
Phone: 234 8030564409
Email: chidi_izuwah@yahoo.co.uk
 - h. The Start Right Centre
Surulere, Lagos
Website: www.autism-smile.co.uk/nigeria/
 - i. The Zamarr Institute
27 Libreville St. Wuse II Abuja
Phone: 2.3480586498e+012
Email: info@thezamarrinstitute.org
Website: www.thezamarrinstitute.org
12. Tanzania
 - a. National Association for Care of Autistics (NACA)
Depr. of Psychiatry Muhimbili Medical Centre
P.O. Box 65293, Dar es Salaam
Phone: 255 51 4320.
Email: bmutag@udsm.ac.tz
Website: <http://www.autismaroundtheglobe.org/countries/tanzania.asp>
 13. Senegal
 - a. Ministry of Health
Building Administratif Avenue Leopold Sedar Senghor, Dakar
Phone: 221 823 10 8.
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 - b. Centre Aminata Mbaye
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Phone/Fax: (00221) 33 867 22 6.
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M. Claude Sarr, Directeur
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- ## References and Reading
- Abubakar, A., Ssewanyana, D., de Vries, P. J., & Newton, C. R. (2016a). Autism spectrum disorders in sub-Saharan Africa. *Lancet Psychiatry*, 3(9), 800–802. [https://doi.org/10.1016/s2215-0366\(16\)30138-9](https://doi.org/10.1016/s2215-0366(16)30138-9).
- Abubakar, A., Ssewanyana, D., & Newton, C. R. (2016b). A systematic review of research on autism spectrum disorders in sub-Saharan Africa. *Behavioural Neurology*, 2016, 3501910. <https://doi.org/10.1155/2016/3501910>.
- Adnams, C. M. (2010). Perspectives of intellectual disability in South Africa: Epidemiology, policy, services for children and adults. *Current Opinion in Psychiatry*, 23, 439–440.
- Ametepee, L. K., & Chitiyo, M. (2009). What we know about autism in Africa: A brief research synthesis. *Journal of the International Association of Special Education*, 10(1), 11–13.
- Bakare, M. O., & Ikegwuonu, N. N. (2008). Childhood autism in a 13 year old boy with oculocutaneous albinism: A case report. *Journal of Medical Case Reports*, 2, 56. <https://doi.org/10.1186/1752-1947-2-56>.
- Bakare, M. O., & Munir, K. M. (2011a). Autism spectrum disorders (ASD) in Africa: A perspective. *African Journal of Psychiatry*, 14(3), 208–210. <https://doi.org/10.4314/ajpsy.v14i3.3>.
- Bakare, M. O., & Munir, K. M. (2011b). Excess of non-verbal cases of autism spectrum disorders presenting to orthodox clinical practice in Africa – A trend possibly resulting from late diagnosis and intervention. *South African Journal of Psychiatry*, 17(4), 118–120.
- Bakare, M. O., Ebigo, P. O., Agomoh, A. O., & Menkiti, N. C. (2008). Knowledge about childhood autism among health workers (KCAHW) questionnaire: Description, reliability and internal consistency. *Clinical Practice and Epidemiology in Mental Health*, 4, 17. <https://doi.org/10.1186/1745-0179-4-17>.
- Bakare, M. O., Agomoh, A. O., Ebigo, P. O., Eaton, J., Okonkwo, K. O., Onwukwe, J. U., & Onyeama, G. M. (2009a). Etiological explanation, treatability and preventability of childhood autism: A survey of Nigerian healthcare workers' opinion. *Annals of General Psychiatry*, 8, 6. <https://doi.org/10.1186/1744-859x-8-6>.
- Bakare, M. O., Ebigo, P. O., Agomoh, A. O., Eaton, J., Onyeama, G. M., Okonkwo, K. O., et al. (2009b). Knowledge about childhood autism and opinion among healthcare workers on availability of facilities and law caring for the needs and rights of children with childhood autism and other developmental disorders in Nigeria. *BMC Pediatrics*, 9, 12. <https://doi.org/10.1186/1471-2431-9-12>.
- Bakare, M. O., Ubochi, V. N., Okoroikpa, I. N., Aguocha, C. M., & Ebigo, P. O. (2009c). Agreement between clinicians' and care givers' assessment of intelligence in Nigerian children with intellectual disability: 'Ratio IQ' as a viable option in the absence of standardized 'deviance IQ' tests in sub-Saharan Africa. *Behavioral and Brain Functions*, 5, 39. <https://doi.org/10.1186/1744-9081-5-39>.
- Bakare, M. O., Igwe, M. N., Odinka, P. C., & Iteke, O. (2011). Neuropsychiatric diagnosis and psychotropic medication prescription patterns in a mental hospital-based child and adolescent psychiatric service in Nigeria. *Journal of Health Care for the Poor and Underserved*, 22(3), 751–755. <https://doi.org/10.1353/hpu.2011.0078>.
- Bakare, M. O., Tunde-Ayinmode, M. F., Adewuya, A. O., Bello-Mojeed, M. A., Sale, S., James, B. O., et al. (2015). Recognition of autism spectrum disorder (ASD) symptoms and knowledge about some other aspects of ASD among final year medical students in Nigeria, sub-Saharan Africa. *BMC Research Notes*, 8, 454. <https://doi.org/10.1186/s13104-015-1433-0>.
- Bello-Mojeed, M. A., Ogun, O. C., Omigbodun, O., Adewuya, A. O., & Ladapo, H. T. O. (2011). Late identification of autistic disorder in Nigeria: An illustration with 2 case reports. *Nigerian Journal of Psychiatry*, 9(2), 31–35.
- Epstein, E. L., & Kole, R. (Eds.). (1998). *The Language of African Literature*. Trenton, NJ: Africa World Press.
- CDC. (2014). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 Sites, United States, 2010. http://www.cdc.gov/mmwr/preview/mmwrhtml/ss6302a1.htm?s_cid=ss6302a1_w. Retrieved 24 Nov 2015.

- Central Intelligence Agency. (2016). The World Factbook. <https://www.cia.gov/library/publications/the-world-factbook/>
- de Vries, P. J. (2016). Thinking globally to meet local needs: Autism spectrum disorders in Africa and other low-resource environments. *Current Opinion in Neurology*, 29(2), 130–136. <https://doi.org/10.1097/wco.0000000000000297>.
- de Vries, P. J., Venter, A., & Jacklin, L. (2013). Behavioural phenotypes and neurodevelopmental disorders in Africa. *Journal of Intellectual Disability Research*, 57, 793–795.
- Dhadphale, M., Lukwago, M. G., & Gajjar, M. (1982). Infantile autism in Kenya. *Indian Journal of Pediatrics*, 49(396), 145–148.
- Durkin, M. S., Maenner, M. J., Meaney, F. J., Levy, S. E., DiGuiseppi, C., Nicholas, J. S., et al. (2010). Socioeconomic inequality in the prevalence of autism spectrum disorder: Evidence from a U.S. cross-sectional study. *PLoS One*, 5(7), e11551. <https://doi.org/10.1371/journal.pone.0011551>.
- Durkin, M. S., Elsabbagh, M., Barbaro, J., Gladstone, M., Happe, F., Hoekstra, R. A., et al. (2015). Autism screening and diagnosis in low resource settings: Challenges and opportunities to enhance research and services worldwide. *Autism Research*, 8(5), 473–476. <https://doi.org/10.1002/aur.1575>.
- Elsabbagh, M., Divan, G., Koh, Y. J., Kim, Y. S., Kauchali, S., Marcin, C., et al. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research*, 5(3), 160–179. <https://doi.org/10.1002/aur.239>.
- Engle, P. L., Black, M. M., Behrman, J. R., Cabral de Mello, M., Gertler, P. J., Kapiriri, L., et al. (2007). Strategies to avoid the loss of developmental potential in more than 200 million children in the developing world. *Lancet*, 369, 229–242.
- Eseigbe, E. E., Nuhu, F. T., Sheikh, T. L., Eseigbe, P., Sanni, K. A., & Olisah, V. O. (2015). Knowledge of childhood autism and challenges of management among medical doctors in Kaduna State, Northwest Nigeria. *Autism Research and Treatment*, 2015, 892301. <https://doi.org/10.1155/2015/892301>.
- Fombonne, E. (2003). Epidemiological surveys of autism and other pervasive developmental disorders: An update. *Journal of Autism and Developmental Disorders*, 33(4), 365–382.
- Franz, L., Chambers, N., von Isonburg, M., & de Vries, P. J. (2017). Autism spectrum disorder in sub-Saharan Africa: A comprehensive scoping review. *Autism Research*. <https://doi.org/10.1002/aur.1766>.
- Garenne, M., & Gakusi, E. (2006). Health transitions in sub-Saharan Africa: Overview of mortality trends in children under 5 years old (1950–2000). *Bulletin of the World Health Organization*, 84, 470–478.
- Gona, J. K., Newton, C. R., Rimba, K., Mapenzi, R., Kihara, M., Van de Vijver, F. J., & Abubakar, A. (2015). Parents' and professionals' perceptions on causes and treatment options for autism spectrum disorders (ASD) in a multicultural context on the Kenyan coast. *PLoS One*, 10(8), e0132729. <https://doi.org/10.1371/journal.pone.0132729>.
- Gona, J. K., Newton, C. R., Rimba, K. K., Mapenzi, R., Kihara, M., Vijver, F. V., & Abubakar, A. (2016). Challenges and coping strategies of parents of children with autism on the Kenyan coast. *Rural and Remote Health*, 16(2), 3517.
- Grinker, R. R., Chambers, N., Njongwe, N., Lagman, A. E., Guthrie, W., Stronach, S., et al. (2012). "Communities" in community engagement: Lessons learned from autism research in South Korea and South Africa. *Autism Research*, 5(3), 201–210. <https://doi.org/10.1002/aur.1229>.
- Harrison, A. J., Zimak, E. H., Sheinkopf, S. J., Manji, K. P., & Morrow, E. M. (2014). Observation-centered approach to ASD assessment in Tanzania. *Intellectual and Developmental Disabilities*, 52(5), 330–347. <https://doi.org/10.1352/1934-9556-52.5.330>.
- Harrison, A. J., Long, K. A., Manji, K. P., & Blane, K. K. (2016). Development of a brief intervention to improve knowledge of autism and behavioral strategies among parents in Tanzania. *Intellectual and Developmental Disabilities*, 54(3), 187–201. <https://doi.org/10.1352/1934-9556-54.3.187>.
- Holford, L. (1994). Asperger's syndrome: A classification struggle. *Southern African Journal of Child & Adolescent Psychiatry*, 6, 47–56.
- Igwe, M. N., Ahanotu, A. C., Bakare, M. O., Achor, J. U., & Igwe, C. (2011). Assessment of knowledge about childhood autism among paediatric and psychiatric nurses in Ebonyi state, Nigeria. *Child and Adolescent Psychiatry and Mental Health*, 5(1), 1. <https://doi.org/10.1186/1753-2000-5-1>.
- Izuwah, D. N., Okoh, B. A., & Alikor, E. A. (2016). Clinical pattern of autism in Nigeria. *Autism Research*, 9(3), 376–381. <https://doi.org/10.1002/aur.1531>.
- Kakooza-Mwesige, A., Ssebyala, K., Karamagi, C., Kiguli, S., Smith, K., Anderson, M. C., et al. (2014). Adaptation of the "ten questions" to screen for autism and other neurodevelopmental disorders in Uganda. *Autism*, 18(4), 447–457. <https://doi.org/10.1177/1362361313475848>.
- Kieling, C., Baker-Henningham, H., Belfer, M., Conti, G., Ertem, I., Omigbodun, O., et al. (2011). Child and adolescent mental health worldwide: Evidence for action. *Lancet*, 378(9801), 1515–1525. [https://doi.org/10.1016/S0140-6736\(11\)60827-1](https://doi.org/10.1016/S0140-6736(11)60827-1).
- Kim, Y. S., Levanthal, B. L., Koh, Y. J., Fombonne, E., Laska, E., Lim, E. C., et al. (2011). Prevalence of autism spectrum disorders in a total population sample. *American Journal of Psychiatry*, 168, 904–912.
- Lagunju, I. A., Bella-Awusah, T. T., & Omigbodun, O. O. (2014). Autistic disorder in Nigeria: Profile and challenges to management. *Epilepsy & Behavior*, 39, 126–129. <https://doi.org/10.1016/j.yebeh.2014.08.020>.
- Lotter, V. (1978). Childhood autism in Africa. *Journal of Child Psychology and Psychiatry*, 19(3), 231–244.
- Majoko, T. (2016). Inclusion of children with autism spectrum disorders: Listening and hearing to voices from the grassroots. *Journal of Autism and Developmental Disorders*, 46(4), 1429–1440. <https://doi.org/10.1007/s10803-015-2685-1>.

- Mathers, C. D., & Loncar, D. (2006). Projections of global mortality and burden of disease from 2002 to 2030. *PLoS Medicine*, 3, e442.
- Murray, C. J., Laakso, T., Shibuya, K., Hill, K., & Lopez, A. D. (2007). Can we achieve Millennium Development Goal 4? New analysis of country trends and forecasts of under-5 mortality to 2015. *Lancet Infectious Diseases*, 370, 1040–1054.
- Newton, C. R., & Chugani, D. C. (2013). The continuing role of ICNA in Africa: How to tackle autism? *Developmental Medicine and Child Neurology*, 55(6), 488–489. <https://doi.org/10.1111/dmcn.12150>.
- Rajatnaram, J. K., Marcus, J. R., Flaxman, A. D., Wang, H., LevinRector, A., Dwyer, L., et al. (2010). Neonatal, postneonatal, childhood, and under-5 mortality for 187 countries, 1970–2010: A systematic analysis of progress towards Millennium Development Goal 4. *Lancet Infectious Diseases*, 375, 1988–2008.
- Ruparelia, K., Abubakar, A., Badoe, E., Bakare, M., Visser, K., Chugani, D. C., et al. (2016). Autism spectrum disorders in Africa: Current challenges in identification, assessment, and treatment: A report on the International Child Neurology Association Meeting on ASD in Africa, Ghana, April 3–5, 2014. *Journal of Child Neurology*, 31(8), 1018–1026. <https://doi.org/10.1177/0883073816635748>.
- Sanua, V. D. (1984). Is infantile autism a universal phenomenon? An open question. *International Journal of Social Psychiatry*, 30(3), 163–177. <https://doi.org/10.1177/002076408403000301>.
- Scherzer, A. L., Chagan, M., Kauchali, S., & Susser, E. (2012). Global perspective on early diagnosis and intervention for children with developmental delays and disabilities. *Developmental Medicine and Child Neurology*, 54, 1079–1084.
- Springer, P. E., van Toorn, R., Laughton, B., & Kidd, M. (2013). Characteristics of children with pervasive developmental disorders attending a developmental clinic in the Western Cape Province, South Africa. *Southern African Journal of Child Health*, 7, 95–99.
- Szabo, C. P., & Aber, D. (1992). Asperger's syndrome: A valid DSM IV diagnostic entity? A case report. *Southern African Journal of Child & Adolescent Psychiatry*, 4, 3–7.
- Tilahun, D., Hanlon, C., Fekadu, A., Tekola, B., Baheretibeb, Y., & Hoekstra, R. A. (2016). Stigma, explanatory models and unmet needs of caregivers of children with developmental disorders in a low-income African country: A cross-sectional facility-based survey. *BMC Health Services Research*, 16, 152. <https://doi.org/10.1186/s12913-016-1383-9>.
- UNICEF. (2014). Generation 2030 Africa. http://www.unicef.org/publications/files/Generation_2030_Africa.pdf. Retrieved 5 Sept 2016
- United Nations. (2014). World and regional groupings Millennium Development Indicators. <http://mdgs.un.org/unsd/mdg/Host.aspx?Content5Data/RegionalGroupings>
- United Nations. (2015). The Millennium Development Goals report 2015. [http://www.un.org/millenniumgoals/2015_MDG_Report/pdf/MDG%202015%20rev%20\(July%202015\).pdf](http://www.un.org/millenniumgoals/2015_MDG_Report/pdf/MDG%202015%20rev%20(July%202015).pdf)
- Wessels, W. H., & Pompe van Meerdervoort, M. (1979). Monozygotic twins with early infantile autism. A case report. *South African Medical Journal*, 55(23), 955–957.
- World Bank. (2013). *Africa development indicators 2012/13*. Washington, DC: World Bank.
- World Bank. (2014). Regions. <http://www.worldbank.org/en/about/annual-report/regions>
- World Bank. (2015). Population, total. Data. Retrieved 1 May 2017
- World Health Organization. (2011). World report on disability 2011. Retrieved from Geneva: www.who.int/disabilities/world_report/2011/en/
- World Health Organization. (2013). Meeting report: Autism spectrum disorders and other developmental disorders: From raising awareness to building capacity. apps.who.int/iris/bitstream/10665/103312/1/9789241506618_eng.pdf.
- World Health Organization. (2014). Autism. Sixty-seventh World Health Assembly, Agenda item 13.4. WHA67.8. May 2014. apps.who.int/gb/ebwha/pdf_files/WHA67/A67_R8-en.pdf.

Substance Use Problems/Disorders in Individuals with ASD

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Introduction

Autism spectrum disorder (ASD) is a heterogeneous condition defined by social deficits, restrictive interests, and repetitive behaviors. Individuals with ASD demonstrate highly variable levels of

intellectual abilities, verbal skills, and adaptive functioning (American Psychiatric Association 2013). Psychiatric and medical comorbidities are frequently co-occurring in ASD, including anxiety, depression, attention deficit hyperactivity disorder (ADHD), obsessive compulsive disorder (OCD), seizure disorder, sleep disorder, and a broad range of various somatic disorders (Bauman 2010; Joshi et al. 2010; Mattila et al. 2010; Muskens et al. 2017; Simonoff et al. 2008). However, little is known about the rates of substance use problems or substance use disorder (SUD) in this group. A recent literature review found that clinical assessment of substance use among individuals with ASD is infrequent, and the overall scientific evidence and understanding of potential relationships between ASD and SUD is very limited (Palmquist et al. 2014).

Current Knowledge

Increasing numbers of individuals with ASD demonstrate high functioning and independent living status (Howlin 2005; Howlin et al. 2004). Additionally, ASD individuals are frequently prescribed medications that may have addictive properties or have misuse or abuse potential (Siegel and Beaulieu 2011; Williamson and Martin 2010). Consequently, the true scope of substance use problems among individuals with ASD can potentially be larger than suggested by researches conducted to date. While an overall prevalence of substance use disorders in individuals with neurodevelopmental disorders is not known, a recent systematic review by Arnevik and Helverschou (2016) reported a range of SUD prevalence rates between 0.7% and 36% among individuals with ASD. The largest study included in the review based on data from a national register in Denmark (Abdallah et al. 2011) included 414 individuals with ASD and reported that only three (0.7%) individuals in the cohort were diagnosed with alcohol related disorders (ARD). In contrast, one of the smallest sample studies included in the review by Mandell and colleagues reported a prevalence of 36% in a sample of 14 psychiatrically hospitalized

individuals with ASD. Most published studies report a lower prevalence of SUD in individuals with ASD than in individuals without ASD. However, small sample sizes and the pronounced selection biases in many of the published reports undermine generalizability or reliability of the reported findings (Arnevik and Helverschou 2016).

A small body of research explored the causes and consequences for substance use among individuals with ASD. It has been reported that while the use of substances can be perceived as helpful by ASD individuals by offering a temporary relieve of anxiety, mood, or other adverse symptoms, it often leads to overall long-term negative outcomes. For example, using substances to overcome social difficulties could have somewhat positive short-term effects, while having a long-term adverse effect on cognitive functioning (Kronenberg et al. 2014).

To summarize, little is known about substance use among individuals with ASD, the rate of co-occurrence is uncertain, and there is a significant lack of studies that address and evaluate interventions for individuals with ASD and substance use disorder. ASD is a relatively rare condition affecting approximately 1.7% of the general population (Baio et al. 2018). Consequently, the overall low ASD prevalence rate poses a significant challenge for research studies aimed to evaluate the rates of co-occurrence of ASD and SUD.

Future Directions

Further epidemiological research is needed to obtain better estimates of prevalence rate of co-occurring ASD and SUD and to assess potential implications for clinical care. As highlighted in the systematic review by Arnevik and Helverschou (2016), research should also focus on SUD treatment for individuals with ASD which is likely to require comprehensive approaches and might be more challenging than treatment for individuals with SUD alone. Research aimed to uncover specific treatment, social support, and other adjunctive services

needs of ASD individuals with SUD is also urgently needed. Furthermore, better understanding of potential causes for substance use or developing a SUD in individuals with ASD to be able to investigate why the rate of SUD is lower in ASD compared to other psychiatric disorders.

References and Reading

- Abdallah, M. W., Greaves-Lord, K., Grove, J., Nørgaard-Pedersen, B., Hougaard, D. M., & Mortensen, E. L. (2011). Psychiatric comorbidities in autism spectrum disorders: findings from a Danish Historic Birth Cohort. *European child & adolescent psychiatry*, 20 (11-12), 599–601.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Publishing.
- Arnevik, E. A., & Helverschou, S. B. (2016). Autism spectrum disorder and co-occurring substance use disorder – A systematic review. *Substance Abuse: Research and Treatment*, 10, SART.S39921. <https://doi.org/10.4137/SART.S39921>.
- Baio, J., Wiggins, L., Christensen, D. L., Maenner, M. J., Daniels, J., Warren, Z., et al. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR Surveillance Summaries*, 67(6), 1.
- Bauman, M. L. (2010). Medical comorbidities in autism: Challenges to diagnosis and treatment. *Neurotherapeutics*, 7(3), 320–327. <https://doi.org/10.1016/j.nurt.2010.06.001>.
- Howlin, P. (2005). Outcomes in autism spectrum disorders. In *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 201–220). John Wiley & Sons Inc, 1997 New York.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45(2), 212–229.
- Joshi, G., Petty, C., Wozniak, J., Henin, A., Fried, R., Galdo, M., et al. (2010). The heavy burden of psychiatric comorbidity in youth with autism spectrum disorders: A large comparative study of a psychiatrically referred population. *Journal of Autism and Developmental Disorders*, 40(11), 1361–1370.
- Kronenberg, L. M., Slager-Visscher, K., Goossens, P. J., van den Brink, W., & van Achtenberg, T. (2014). Everyday life consequences of substance use in adult patients with a substance use disorder (SUD) and co-occurring attention deficit/hyperactivity disorder (ADHD) or autism spectrum disorder (ASD): A patient's perspective. *BMC Psychiatry*, 14(1), 194. <https://doi.org/10.1186/s12888-014-0264-1>.
- Mattila, M.-L., Hurtig, T., Haapsamo, H., Jussila, K., Kuusikko-Gauffin, S., Kielinen, M., et al. (2010). Comorbid psychiatric disorders associated with Asperger syndrome/high-functioning autism: A community-and clinic-based study. *Journal of Autism and Developmental Disorders*, 40(9), 1080–1093.
- Muskens, J. B., Velders, F. P., & Staal, W. G. (2017). Medical comorbidities in children and adolescents with autism spectrum disorders and attention deficit hyperactivity disorders: A systematic review. *European Child & Adolescent Psychiatry*, 26(9), 1093–1103. <https://doi.org/10.1007/s00787-017-1020-0>.
- Palmquist, E., Claeson, A.-S., Neely, G., Stenberg, B., & Nordin, S. (2014). Overlap in prevalence between various types of environmental intolerance. *International Journal of Hygiene and Environmental Health*, 217(4-5), 427–434. <https://doi.org/10.1016/j.ijheh.2013.08.005>.
- Siegel, M., & Beaulieu, A. A. (2011). Psychotropic medications in children with autism spectrum disorders: A systematic review and synthesis for evidence-based practice. *Journal of Autism and Developmental Disorders*, 42(8), 1592–1605. <https://doi.org/10.1007/s10803-011-1399-2>.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(8), 921–929. <https://doi.org/10.1097/CHI.0b013e318179964f>.
- Williamson, E. D., & Martin, A. (2010). Psychotropic medications in autism: Practical considerations for parents. *Journal of Autism and Developmental Disorders*, 42(6), 1249–1255. <https://doi.org/10.1007/s10803-010-1144-2>.

Substantia Grisea

► Gray Matter

Substantia Nigra

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Definition

The substantia nigra consists of large dopamine-producing nerve cells in the midbrain, adjacent to the striatum. The structure is part of the basal

ganglia and consists of two subdivisions, the pars compacta and pars reticulata. This region has functional implications in reward, addiction, and movement, as damage to this area is associated with disorders such as Parkinson's disease.

References and Reading

Lewis, M. H., Tanimura, Y., Lee, L. W., & Bodfish, J. W. (2007). Animal models of restricted repetitive behavior in autism. *Behavioural Brain Research*, 176, 66–74.

Substantial Gainful Activity

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Synonyms

Substantial gainful employment

Definition

Substantial gainful activity is a term used to describe a level of work activity and earnings. It is used by the social security administration to determine if a person is eligible for social security benefits. It refers to any work that is generally performed for pay or profit or intended for profit (even if there is none). It applies to full- or part-time work. It can also apply to self-employment.

Substantial gainful activity is not about whether you can perform your job. It is not about whether you can or cannot find work. It is about whether you can work at any employment activity that is generally available. It includes salaried positions, attending school, or a volunteer activity even if you receive no salary. An activity qualifies as substantial gainful activity based on the amount of income that is or could be earned. You are not eligible for disability benefits if you can perform any work that someone could have been paid for.

The Social Security Administration defines a dollar amount yearly that a person can earn and still maintain their eligibility for SSI or social security benefits. Those who earn (or could earn) over this minimum dollar amount are considered to be engaging in substantial gainful activity and, therefore, ineligible for Social Security benefits.

See Also

► [Employment](#)

References and Reading

Federal Government. (2010). *Social security handbook 2010: Overview of social security programs*. Lanham: Bernam Press.

Morton, D. M. D., III. (2010). *Nolo's guide to social security disability: Getting and keeping your benefits* (5th ed.). Berkeley: NOLO Press.

Social security disability help. <http://www.socialsecurity-disability.org>

Social Security On-line. www.socialsecurity.gov

The Red Book. <http://www.ssa.gov/redbook/eng/introduction.htm>

Substantial Gainful Employment

► [Substantial Gainful Activity](#)

Subtest Scatter

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Definition

Subtest scatter refers to a pattern or profile across subtests in which subtest scores on a standardized test vary a great deal. Most standardized subtest

scores have a mean, or average, score of 10. Children will often score within three points above or below the mean of 10 (i.e., scores between 7 and 13) (or above and below their own mean score across subtests, which may be higher or lower than 10). If significant scatter exists, meaning the difference between the highest and lowest subtest scores is greater than six points, this suggests that the test taker has areas of strength and weakness that need to be further explored. When interpreting standardized test results of an individual with an autism spectrum disorder, it is important for educators, the individual, and/or parents to understand the nature of the weak areas or areas of relative weakness, what skills need to be learned to strengthen those areas (e.g., utilizing strengths to develop skills in area of weakness), and what specific accommodations should be implemented to facilitate the remediation of the weak areas. Understanding areas of strength or relative strength can often inform remediation of weaknesses.

Historical Background

Researchers have been analyzing IQ subtests for more than 70 years (Zachary 1990). Early researchers hypothesized that subtest scatter would predict academic potential. Additionally, uneven subtest scores were assumed to be indicative of psychopathology. Specifically, psychologists speculated that subtest scatter of an individual's scaled scores across subtests on an intelligence battery might be a sign of neurological dysfunction (Drebing et al. 1994), learning disability (McLean et al. 1990), or emotional disability (Drummond 2000). While some studies found subtest scatter to be associated with clinical groups, differences tended to disappear when adequate comparison samples were used (Zimmerman and Woo-Sam 1985). Furthermore, subtest scatter was also found to be unrelated to academic achievement (Hale and Saxe 1983; Kline et al. 1992).

A source of data that has greatly contributed to the understanding of IQ subtest analysis is the Dunedin Multidisciplinary Health and

Development Study (DMHDS; Silva 1990). The DMHDS included a sample of more than 1000 New Zealand children assessed with a battery of psychological, sociological, and medical measures every 2 years from birth to adulthood. Its representative sample, comprehensive assessment battery, and longitudinal design make DMHDS IQ test results unique in the professional literature.

Using data from the DMHDS, Moffitt and Silva (1987) reported on the clinical significance and stability of Wechsler Intelligence Scale for Children-Revised (WISC-R; Wechsler 1974) VIQ and PIQ scatter. They concluded that perinatal, neurological, and health problems did not cause extreme VIQ-PIQ discrepancies and found that neither behavior problems nor motor problems were significantly related to VIQ-PIQ scores. Furthermore, VIQ-PIQ score discrepancies were unreliable across time. That is, the majority of children with extreme VIQ-PIQ score discrepancies did not maintain such a large difference when tested with the WISC-R 2 years later. Thus, they concluded that VIQ-PIQ scatter may not be stable and does not adequately predict psychological diagnoses. The combined results from 94 studies, including 9372 total participants, also demonstrated that subtest scatter and scatter between VIQ and PIQ failed to uniquely distinguish children with learning disabilities (Kavale and Forness 1984). For example, the average VIQ-PIQ difference for children with learning disabilities was only 3.5 points – a difference found in 79% of the normative sample.

Even though subtest scatter was not found to indicate psychopathology or predict academic achievement, psychologists continued to examine individual subtest patterns. Specifically, examining an individual's scaled subtest scores began to be used to identify specific cognitive strengths and weaknesses (Zeidner 2001). Following this logic, a high degree of subtest variability or specific patterns of subtest scores were presumed to substantially invalidate global intelligence indices (Groth-Marnat 1997) so that subtests, rather than IQ composites, became the focus of interpretation. Psychologists believed that such a multidimensional view of intelligence would provide greater insight into the nature of human ability

than summary intellectual indices (Zimmerman and Woo-Sam 1985), particularly when trying to describe an individual's pattern of cognitive performance. Based on these principles, intricate subtest profile interpretation systems have achieved wide popularity in psychological training and practice (Aiken 1996; Groth-Marnat 1997; Kaufman 1994; Sattler 2001). For example, approximately 74% of school psychology training programs place moderate to great emphasis on the use of subtest scores in their individual cognitive assessment courses, and almost all use texts that advocate subtest analysis (Alfonso et al. 2000). As would be expected from such training, school psychologists frequently analyze cognitive subtest profiles in their practice (Pfeiffer et al. 2000). Among their sample of clinicians, for example, Pfeiffer et al. found that almost 70% reported factor scores/profile analysis to be a useful feature of intelligence tests and 29% reported that they derived specific value from individual subtests. Matarazzo and Prifitera (1989) noted that while some supportive WAIS-R subtest scatter research had been published, lack of cross-validation and replication deems interpretation of subtest scatter useful for creating hypothesis about an individual, but the results are not conclusive in and of themselves.

Current Knowledge

Subtest scatter has continued to receive clinical attention despite previous literature reviews demonstrating that it was ineffective for distinguishing clinical from typically functioning groups. Recent research results are remarkably consistent with past reviews, in that results indicate that subtest scatter is an invalid diagnostic indicator and incapable of reliably predicting academic achievement. Several studies are particularly important because they demonstrated no relationship between subtest scatter and academic achievement in large, nationally representative samples of students without disabilities (Kahana et al. 2002; McDermott and Glutting 1997; McGrew and Knopik 1996; Watkins and Glutting 2000; Youngstrom et al. 1999). Based on their review

of IQ subtest scatter research, Kline et al. (1996) suggested that psychologists have pursued scatter analysis with little success and that it is time to move on; this suggestion was further supported by McGrew and Knopik (1996). Given the body of research to date, it seems as though there is no scientific support for the use of subtest scatter to inform diagnosis or prediction.

Future Directions

Although there is general agreement that diagnoses based on IQ subtest scatter should be avoided, the use of subtest profiles and subtest scatter for hypothesis generation is frequently recommended (Sattler 2001; Kaufman and Lichtenberger 1998, 2000; Kamphaus 2001; Groth-Marnat 1997). Specifically, the examiner must first generate hypotheses about an individual's assets and deficits. Next, the examiner must confirm or deny these hypotheses by exploring multiple sources of evidence. Finally, well-validated hypotheses must then be translated into meaningful, practical recommendations, concerning interventions, instructional strategies, and remediation activities (Groth-Marnat 1997).

Treatment recommendations resulting from the hypotheses generated from interpretation of subtest profiles and subtest scatter should follow three basic guidelines. First, subtest profiles must be strongly associated with performance in such socially important endeavors as academic achievement and psychosocial behavior. If subtest profiles are not substantially related to important social criteria, then hypotheses generated from subtest variation may not be useful. Second, hypotheses based on subtest scatter or subtest profiles should be consistently confirmed by multiple pieces of other information, including pursuing additional testing to corroborate observed areas of weakness. Finally, interventions that result from hypotheses based on subtest performance must exhibit treatment validity; that is, they must selectively assist in the improvement of cognitive, achievement, or behavioral weaknesses.

Subtest interpretation systems provide hundreds of hypotheses to consider when IQ subtest

patterns are obtained (Kaufman 1994; Sattler 2001). For example, some comparisons that have been highlighted include the following: low performance on the Picture Arrangement (PA) and Comprehension (CM) subtests suggests poor social adjustment, as seen in autism spectrum disorders; equal VIQ and PIQ scores may indicate an absence of emotional distress; a large difference in performance between digits forward and digits backward indicates anxiety; and low scores on Digit Span (DS), Arithmetic (AR), and Coding (CD) subtests identify anxiety, attentional deficits, or both (Banas 1993; Drummond 2000; Groth-Marnat 1997; Kellerman and Burry 1997).

The relationship between social adjustment and performance on PA and CM subtests has received considerable attention. Lipsitz et al. (1993) administered Wechsler scales and two measures of social adjustment to groups of high-risk and typical comparison children. PA scores showed no relation with either measure of social adjustment. Further, Campbell and McCord (1996) found that PA scores were not significantly better than FSIQ scores in predicting participants' ability to interpret the nonverbal behavior of others. These negative results for the PA subtest have been replicated with different samples, criterion tests, and IQ tests (Beebe et al. 2000; Campbell and McCord 1999). Consequently, making an inference regarding social adjustment/judgment based on PA scores is contraindicated by research.

See Also

► WPPSI-III

References and Reading

- Aiken, L. R. (1996). *Assessment of intellectual functioning* (2nd ed.). New York: Plenum.
- Alfonso, V. C., Oakland, T. D., LaRocca, R., & Spanakos, A. (2000). The course on individual cognitive assessment. *School Psychology Review*, 29, 52–64.
- Banas, N. (1993). *WISC-III prescriptions: How to work creatively with individual learning styles*. Novato: Academic Therapy.
- Beebe, D. W., Pfiffner, L. J., & McBurnett, K. (2000). Evaluation of the validity of the Wechsler Intelligence Scale for Children-Third Edition comprehension and picture arrangement subtests as measures of social intelligence. *Psychological Assessment*, 12, 97–101.
- Campbell, J. M., & McCord, D. M. (1996). The WAIS-R comprehension and picture arrangement subtests as measures of social intelligence: Testing traditional interpretations. *Journal of Psychoeducational Assessment*, 14, 240–249.
- Campbell, J. M., & McCord, D. M. (1999). Measuring social competence with the Wechsler picture arrangement and comprehension subtests. *Assessment*, 6, 215–223.
- Drebing, C., Satz, P., Van Gorp, W., Chervinsky, A., & Uchiyama, C. (1994). WAIS-R intersubtest scatter in patients with dementia of Alzheimer's type. *Journal of Clinical Psychology*, 50, 753–758.
- Drummond, R. J. (2000). *Appraisal procedures for counselors and helping professionals* (4th ed.). Upper Saddle River: Prentice-Hall.
- Groth-Marnat, G. (1997). *Handbook of psychological assessment* (3rd ed.). New York: Wiley.
- Hale, R. L., & Saxe, J. E. (1983). Profile analysis of the Wechsler Intelligence Scale for Children-Revised. *Journal of Psychoeducational Assessment*, 1, 155–162.
- Kahana, S. Y., Youngstrom, E. A., & Glutting, J. J. (2002). Factor and subtest discrepancies on the differential abilities scale: Examining prevalence and validity in predicting academic achievement. *Assessment*, 9, 82–93.
- Kamphaus, R. W. (2001). *Clinical assessment of child and adolescent intelligence* (2nd ed.). Boston: Allyn & Bacon.
- Kaufman, A. S. (1994). *Intelligent testing with the WISC-III*. New York: Wiley.
- Kaufman, A. S., & Lichtenberger, E. O. (1998). Intellectual assessment. In A. S. Bellack & M. Hersen (Eds.), *Comprehensive clinical psychology: Assessment* (Vol. 4, pp. 187–238). New York: Elsevier.
- Kaufman, A. S., & Lichtenberger, E. O. (2000). *Essentials of WISC-III and WPPSI-R assessment*. New York: Wiley.
- Kavale, K. A., & Forness, S. R. (1984). A meta-analysis of the validity of Wechsler scale profiles and recategorizations: Patterns or parodies? *Learning Disabilities Quarterly*, 7, 136–156.
- Kellerman, H., & Burry, A. (1997). *Handbook of psychodiagnostic testing: Analysis of personality in the psychological report* (3rd ed.). Boston: Allyn & Bacon.
- Kline, R. B., Snyder, J., Guilmette, S., & Castellanos, M. (1992). Relative usefulness of elevation, variability, and shape information from WISC-R, K-ABC, and Fourth Edition Stanford-Binet profiles in predicting achievement. *Psychological Assessment*, 4, 426–432.
- Kline, R. B., Snyder, J., & Castellanos, M. (1996). Lessons from the Kaufman Assessment Battery for Children (K-ABC): Towards a new cognitive assessment model. *Psychological Assessment*, 8, 7–17.

- Lipsitz, J. D., Dworkin, R. H., & Erlenmeyer-Kimling, L. (1993). Wechsler comprehension and picture arrangement subtests and social adjustment. *Psychological Assessment*, 5, 430–437.
- Matarazzo, J. D., & Prifitera, A. (1989). Subtest scatter and premorbid intelligence: Lessons from the WAIS-R standardization sample. *Psychological Assessment*, 1, 186–191.
- McDermott, P. A., & Glutting, J. J. (1997). Informing stylistic learning behavior, disposition, and achievement through ability subtests—Or, more illusions of meaning? *School Psychology Review*, 26, 163–175.
- McGrew, K. S., & Knopik, S. N. (1996). The relationship between intra-cognitive scatter on the Woodcock-Johnson Psycho-Educational Battery-Revised and school achievement. *Journal of School Psychology*, 34, 351–364.
- McLean, J. E., Reynolds, C. R., & Kaufman, A. S. (1990). WAIS-R subtest scatter using the profile variability index. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, 2, 289–292.
- Moffitt, T. E., & Silva, P. A. (1987). WISC-R verbal and performance IQ discrepancy in an unselected cohort: Clinical significance and longitudinal stability. *Journal of Consulting and Clinical Psychology*, 55, 768–774.
- Pfeiffer, S. I., Reddy, L. A., Kletzel, J. E., Schmelzer, E. R., & Boyer, L. M. (2000). The practitioner's view of IQ testing and profile analysis. *School Psychology Quarterly*, 15, 376–385.
- Sattler, J. M. (2001). *Assessment of children: Cognitive applications* (4th ed.). San Diego: Author.
- Silva, P. A. (1990). The Dunedin multidisciplinary health and development study: A 15-year longitudinal study. *Pediatric and Perinatal Epidemiology*, 4, 96–127.
- Watkins, M. W., & Glutting, J. J. (2000). Incremental validity of WISC-III profile elevation, scatter, and shape information for predicting reading and math achievement. *Psychological Assessment*, 12, 402–408.
- Wechsler, D. (1974). *Wechsler intelligence scale for children-revised*. New York: Psychological Corporation.
- Youngstrom, E. A., Kogos, J. L., & Glutting, J. J. (1999). Incremental efficacy of differential ability scales factor scores in predicting individual achievement criteria. *School Psychology Quarterly*, 14, 26–39.
- Zachary, R. A. (1990). Wechsler's intelligence scales: Theoretical and practical considerations. *Journal of Psychoeducational Assessment*, 8, 276–289.
- Zeidner, M. (2001). Invited foreword and introduction. In J. J. W. Andrews, D. H. Saklofske, & H. L. Janzen (Eds.), *Handbook of psychoeducational assessment: Ability, achievement, and behavior in children*. New York: Academic.
- Zimmerman, I. L., & Woo-Sam, J. M. (1985). Clinical applications. In B. B. Wolman (Ed.), *Handbook of intelligence: Theories, measurements, and applications* (pp. 873–898). New York: Wiley.

Subtyping Autism

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Definition

Subtypes of autism can be defined as groups of persons with autism spectrum disorders (ASD) who are posited to be more similar to each other than to the larger group of individuals who fall under the broad diagnostic category of ASD. Identification of subtypes may facilitate efforts to understand etiology of ASD as well as differential response to treatment. These subtypes are most often defined by a particular behavior or set of behaviors, such as cognitive or language abilities. However, they may also be defined by other characteristics, such as patterns of development or physical features.

Historical Background

Autism spectrum disorders (ASDs) are characterized by impairments in communication and reciprocal social interaction and the presence of restricted and repetitive behaviors and interests. Although ASDs are defined based on this triad of impairments, the presentation of these symptoms varies widely from one individual to another. Behaviors are further impacted by variability in other factors, such as age, level of language, and cognitive ability. This heterogeneity makes it difficult for researchers seeking to elucidate causes or risk factors for ASD or develop and test the efficacy of interventions. Common sense would suggest that it is unlikely that the same etiological

factors would explain two vastly different phenotypes (e.g., a nonverbal child with profound intellectual disability and a verbally fluent child with a superior IQ). Similarly, it would not be expected that those two individuals would respond equally to the same type of intervention. However, at this point, there is relatively little data to answer these questions.

In an attempt to reduce the variability observed in ASDs, researchers have sought to identify groups of individuals within the autism spectrum with the hope that these “subtypes” may represent a more homogeneous group that has a greater likelihood of being etiologically distinct (Newschaffer et al. 2002) or responsive to a certain type of intervention (see Schreibman and Ingersoll 2011). A summary of some subtypes is provided below; however, it is important to recognize that subtypes in ASD are still being actively researched and definitions are continually being modified to reflect the most recent findings. The question still remains as to whether these subtypes will lead to increased understanding of the etiology or prognosis for some individuals with ASD and whether these will ever have clinical utility, allowing clinicians to better explain the causes of a child’s difficulties or choose the most appropriate treatment for that child.

Categorical Diagnoses

The earliest descriptions of autism described by Kanner in 1944 noted the variability in symptom manifestation and severity. Yet, it was not until 1980, with the American Psychiatric Association’s publication of DSM-III, that such variation was noted in the diagnostic criteria with the category of “Atypical Pervasive Developmental Disorder” included to capture children who did not meet full criteria for infantile autism. This category was renamed “Pervasive Developmental Disorder-not otherwise specified” in DSM-III-R (1987). In addition to Autistic Disorder and PDD-NOS, other categories of ASD (i.e., Pervasive Developmental Disorders) were added in DSM-IV, including Asperger’s disorder, Child Disintegrative Disorder, and Rett’s disorder.

These diagnostic categories were among the early attempts to capture the behavioral

heterogeneity observed in ASD by distinguishing subtypes of persons showing different kinds and levels of symptoms and/or patterns of onset. However, over time, it has been increasingly recognized that, even within these diagnostic subtypes, there remains a great deal of variability in symptom presentation and course. Furthermore, although ASDs are reliably distinguished from typical development and other non-ASD developmental disabilities, differentiations within the autism spectrum (i.e., Autistic Disorder, Asperger’s disorder, and PDD-NOS) have been generally inconsistent (Lord et al. 2012). Efforts to demonstrate measureable differences between groups have not been particularly successful. For example, studies comparing the cognitive profiles of individuals with Asperger’s to those deemed as having “high-functioning autism” (i.e., a diagnosis of Autistic Disorder and IQ in the average to above average range) have provided mixed results. In general, these studies have not supported the categorical distinction made between these two groups (see Joseph 2011).

Given that these diagnostic categories have not yielded meaningful subgroups, the Neurodevelopmental Disorders Work Group has proposed that DSM5 includes a single diagnostic category of ASD with clinical specifiers (e.g., to indicate severity and verbal ability) and associated features (e.g., known genetic etiologies, such as fragile X, intellectual disability, etc.). Previously differentiated categories of Autistic Disorder, Asperger’s, and PDD-NOS will no longer be used in clinical practice.

Cognitive Subtypes

Cognitive abilities observed in ASD range from severely intellectually disabled to performance in the superior range on tests of intellectual functioning. In contrast to early studies suggesting that 75% of children with ASD have intellectual disability (ID), recent estimates are closer to 25–50% (Charman et al. 2011; Joseph 2011). In an early study comparing children with ASD and ID to those who did not have cognitive impairments (i.e., IQs <70 and >70), Bartak and Rutter (1976) reported group differences in ASD-related symptoms as well as educational and employment

outcomes. These authors hypothesized that the origin of autism may differ for children with and without intellectual disability and highlighted the importance of considering intellectual functioning in studies assessing etiological factors in autism. Although studies have found differences in genetic transmission of ASD for individuals with IQs over 70 (see Joseph 2011), results must be interpreted cautiously, as is possible that the identified loci are more generally related to IQ than ASD (Liu et al. 2008). It is also important to note that, while some studies have reported familial clustering of IQ in twin and sibling studies, conclusions are often based upon moderately sized correlations with wide confidence intervals (e.g., Szatmari et al. 2008), and, in many multiplex families, there are vast discrepancies in children's cognitive abilities (see LeCouteur et al. 1996).

Aside from general level of functioning, investigators have also questioned whether particular cognitive profiles, such as a large discrepancy between verbal and performance IQ, might reflect etiological differences in ASD (Joseph 2011). Early studies emphasized strengths in nonverbal abilities compared to language skills in children with ASD (Lockyer and Rutter 1970). Since then, researchers have sought to understand how the early development and severity of symptoms of children with this profile may differ from children with ASD who do not demonstrate this discrepancy. There is some evidence that discrepant cognitive profiles may be associated with enlarged head circumference and increased gray matter volume (see Joseph 2011). While there is no doubt that consideration of large differences in IQ is important to long-term outcomes (e.g., comparing individuals with and without ID; Howlin et al. 2004), the existence of categorical cognitive subtypes is not yet clear.

Language Subtypes

A substantial body of research has focused on whether age of language acquisition should be used to differentiate between Autistic Disorder and Asperger's and whether children with or without a language delay may represent etiologically different subtypes. These subtypes have generated a great deal of interest, given that increased

associations with various chromosomal abnormalities have been found in quantitative trait loci (QTL) analyses for age of language acquisition and linkage analyses using samples stratified by whether or not the proband had delayed language (see Cantor 2011; Lamb 2011 for reviews). However, not all studies using this method have yielded positive results. These inconsistencies could be related to differences across samples. Group comparisons of children with and without language delays show differences in age, cognitive level, and overall severity of symptoms, suggesting that if samples differ on these characteristics, associations with biological mechanisms may not be replicated across studies (Hus et al. 2007). There is also evidence that error in retrospective recall of language milestones, upon which most studies rely, could result in children being inaccurately grouped as language delayed, particularly for children with cognitive delay (Hus et al. 2011). Bennett et al. (2008) have suggested that current language level may be a more meaningful marker than history. While these considerations may help to explain inconsistencies across studies, they also suggest that age of language acquisition could be an index for general level of functioning, rather than a more specific subtype. While children with cognitive impairment may be a group of interest in and of themselves, highlighting associations between biological mechanisms and language subtypes can be misleading if these subtypes actually indicate more global impairments.

A range of current language abilities, apart from different histories, are observed in conjunction with ASD. Tager-Flusberg et al. (2011) briefly highlight three language subtypes within ASD – (1) individuals who are verbally fluent and do not have difficulties with structural aspects of language (e.g., vocabulary, syntax, phonology); (2) individuals who acquire varying degrees of functional language, though acquisition may be delayed, development may be slowed, and they may have ongoing difficulties in different areas of language; and (3) individuals who remain nonverbal (i.e., do not develop the ability to speak).

Many researchers have been particularly interested in the second group, i.e., those who acquire

functional speech but exhibit difficulties that overlap with those associated with specific language impairment (SLI). Although definitions of SLI generally require that language difficulties are independent of ASD, some researchers have questioned whether this criterion is appropriate or if some children should be considered as having a comorbid diagnosis of ASD and SLI (see Tomblin et al. 2011 for review). Similar patterns of neuroanatomical asymmetry have been reported in children with SLI and children with ASD and language impairments. Moreover, genetic variation within CNTNAP2 has been associated with both ASD and SLI; most interestingly, the same genetic polymorphism on this gene was related to parent-reported age of language acquisition in children with ASD and phonological short-term memory in children with SLI (see Tomblin et al. 2011).

In spite of evidence that failure to develop functional speech by age 5 is associated with poorer outcomes for children with ASD (e.g., Venter et al. 1992), much less research has focused on understanding nonverbal children. There is some evidence that children who have not developed language by age 5 are more impaired on early measures of social, communication, and imitation skills (Thurm et al. 2006). However, there has not yet been enough research on nonverbal individuals to know whether they may represent a useful unitary subtype. Several studies have shown a high concordance rate for verbal/nonverbal status in sibling and twin pairs (e.g., Spiker et al. 2002), though results have not been consistent. However, little is known about the biological mechanisms underlying the failure to acquire spoken language (Tager-Flusberg et al. 2011).

Repetitive Behaviors Subtypes

The domain of restricted, repetitive behaviors (RRBs) encompasses a variety of behaviors, ranging from repetitive sensorimotor behaviors (RSM), such as hand and finger mannerisms, to insistence on sameness (IS, e.g., difficulties with changes in routine). Factor analyses of the Autism Diagnostic Interview-Revised support a division of these subtypes of RRBs (Rutter et al. 2003).

While RSM behaviors are frequently associated with lower cognitive and adaptive functioning, IS has been shown to be relatively independent of other phenotypic features (see Richler et al. 2010 for review). Differences in correlates and predictors of change, as well as differences in patterns of change for each subtype of RRB, provide further evidence for distinct profiles of repetitive behaviors in persons with ASD. Several studies have demonstrated genetic associations with IS, further supporting its utility as a distinct subtype; recent evidence for a relationship between distinct genetic regions and RSMA has also been reported (see Coon et al. 2010).

Patterns of Onset

There has been a great deal of interest in patterns of onset of ASD, particularly in children who exhibit a loss of language and/or social skills in the first few years of life. Pickles et al. (2009) provide evidence that regression is highly specific to ASD and suggest that the phenomenon underlying loss may be related to brain development in a subtype of autism. In a sample of multiplex families in which both affected family members had a history of regression, Molloy et al. (2005) found evidence of linkage on 7q and 21q, including regions containing genes expressed in fetal brain. However, several studies have reported that, by later childhood, children with a history of regression do not differ from children without regression on a variety of outcome measures, including intellectual functioning, symptom severity, adaptive behaviors, seizures, and gastrointestinal difficulties. As with language milestones, many studies have relied on retrospective parent report of regression, which may not capture the more subtle losses observed in prospective studies (Ozonoff et al. 2011).

Physical Phenotypes

In addition to subtypes defined by specific patterns of behavior, some researchers have hypothesized that physical phenotypic features may reflect abnormal processes during embryogenesis or related to the genes involved in early development of the brain stem and face (Miles and Hillman 2000; Rodier et al. 1996). Although

dysmorphic features are neither sensitive nor specific to ASD, they may provide insight into the etiological process contributing to risk and/or development of ASD, as well as the prenatal period in which exposure to a teratogen occurred (Dufour-Rainfray et al. 2011).

Miles et al. (2005) have hypothesized that children with congenital anomalies (i.e., significant dysmorphology or microcephaly) may represent an etiologically distinct subtype which they refer to as “complex autism.” In contrast, children with no evidence of dysmorphology define “essential autism.” Compared to children with essential autism, those with complex autism are more cognitively impaired and more likely to have seizures and abnormal brain structures. Children with essential autism were noted to have a higher likelihood of being male and a family history of autism as well as a higher sibling recurrence risk (taking into account specific known genetic disorders). Miles and colleagues suggest that removing children with complex autism (which include children with specific identifiable syndromes, such as Fragile X Syndrome) from linkage and sib-pair analyses will provide a more homogeneous subgroup.

Others have focused on macrocephaly and accelerated trajectories of brain growth as a putative subtype. Pierce and Eyler (2011) posit three types of brain growth in ASD: (1) children who meet criteria for macrocephaly and have persistently enlarged brains; (2) those whose brains demonstrate large changes in size relative to head circumference at birth; and (3) those who do not demonstrate dramatic changes in brain size. However, neither the mechanisms contributing to enlarged brains and/or accelerated patterns of growth nor the relationship between these subtypes and functional outcomes in ASD is well understood (see Miles 2011; Pierce and Eyler 2011). Associations with genes implicated in other disorders (e.g., PTEN) have been demonstrated in children with autism, suggesting that studying the overlap between autism and other genetic disorders associated with macrocephaly may further the understanding of this subtype (Miles 2011). Further studies of the developing brain at the cellular and molecular level will be

important to gain insight into gene expression and mechanisms contributing to accelerated patterns of brain growth (Pierce and Eyler 2011).

In addition to physical phenotypic features, a large body of research has investigated neurochemical subtypes in ASD. One neurotransmitter that has received a significant amount of interest is serotonin (5-HT). Since early studies demonstrated that children with ASD had higher levels of whole blood 5-HT, numerous studies have sought to understand the role that 5-HT may play in ASD (see Posey et al. 2011). Serotonin challenge studies have demonstrated differences in both neuroendocrine function and behavioral symptoms (e.g., increased repetitive and self-injurious behaviors) in some participants with autism compared to controls. In contrast, studies examining the effects of selective serotonin reuptake inhibitors (SSRIs) on reducing RRBs have had mixed results (see King et al. 2009). With respect to underlying mechanisms, genes associated with whole blood 5-HT levels have been posited to play a role in autism susceptibility. Numerous studies have also indicated associations between the 5-HT transporter gene (SLC6A4) and autism, though the region implicated has varied across studies. Results suggest a complex relationship between this gene and autism; it is possible that associations with the short or long allele may represent two differing subtypes (which may explain the variability in effectiveness of SSRIs). Nonetheless, associations between SLC6A4 variants and more severe impairments in nonverbal communication and repetitive behaviors, as well as increases in cortical gray matter, suggest that this gene may increase susceptibility to autism in certain groups of children (see Posey et al. 2011; Veenstra-VanderWeele and Anderson 2011).

Sex

Many researchers have hypothesized reasons for the discrepancy in prevalence of ASD in males versus females, suggesting that affected males and females may demonstrate different cognitive or behavioral phenotypes. Early studies comparing sexes reported that females with ASD were more impaired than males in cognitive functioning (see

Hartley and Sikora 2009 for review). Recent research has begun to suggest that sex differences in cognitive ability may be present in families with one child with ASD and one or more unaffected siblings (i.e., simplex samples), but not multiplex samples (i.e., families with more than one child with ASD; Banach et al. 2008; Spiker et al. 2002).

Studies examining sex differences in ASD-related impairments have provided mixed results. While some studies have found no differences between males and females, others have suggested that, after controlling for cognitive functioning, females may demonstrate milder ASD-related impairments, particularly in terms of RRBs and imitation and play skills (see Hartley and Sikora 2009). Interestingly, behaviors comprising IS have been shown to be relatively independent of sex (Hus et al. 2007; Richler et al. 2010). Retrospective parent report of behaviors during the preschool years suggests that males may have more impaired social and communication skills than females, whereas parental reports of their child's current functioning at older ages indicated that females had more social and communication difficulties than males; other studies reported no differences (see Hartley and Sikora 2009 for review). Although findings with respect to RRBs are somewhat more consistent, it is important to note that these findings have been limited to a few studies of repetitive behaviors in toddlers or preschool- to school-age children. Thus, sex-RRB associations may be somewhat different in adolescents and adults. In contrast, studies examining social and communication impairments tended to have a somewhat wider age range, which may explain the variability in results. Moreover, measures used to diagnose ASD have not been specifically validated in female samples and may not fully capture the behavioral variability demonstrated by females with ASD (Huerta et al. 2011).

There is some evidence for sex-based etiological differences, though results have also not always been consistent. Genetic studies have indicated specific linkage associations with male-only families and others with families including females (e.g., Lamb et al. 2005; Stone et al.

2004). Furthermore, proportion of individuals carrying de novo CNVs is lower (2:1 male to female) than the overall ratio of affected males to females in the population (Xu et al. 2008).

Responders to Treatment

Paralleling the behavioral heterogeneity observed in ASD, there is a great deal of variability in response to treatment. Some researchers have begun to try to understand what factors may influence a child's responsiveness to a given treatment. In young children with autism, baseline child characteristics such as IQ, language level, interest in objects/activities, and initiation of joint attention have predicted response to different treatments. Importantly, certain interventions have been differentially predicted by these characteristics. For example, children who initiated joint attention prior to treatment made more gains in response to responsive education and prelinguistic milieu teaching (REPM), a naturalistic behavioral language intervention; in contrast, children with more limited joint attention skills responded better to the picture exchange communication system (PECS), a structured augmentative communication intervention (see Schreibman and Ingersoll 2011 for review).

Although subgroups of treatment "responders" (i.e., those who have made the most gains in response to receiving an intervention) have been identified based upon behavioral characteristics, because these characteristics are often defined relative to the "nonresponders," it is difficult to know how to define these subtypes outside of the context of the intervention study. That is, if we wanted to decide whether to refer a child to REPM or PECS, at what point do we decide that his or her level of joint attention skills is sufficient to suggest response to REPM or impaired enough to recommend that PECS may be more appropriate? While clearly there is no sufficient data to inform such clinical decisions just yet, studies that did not find expected moderating effects of child characteristics (e.g., joint attention) speculate that the absence of effects may be due to the behavior not being sampled in a sufficient number of contexts (Carter et al. 2011).

Current Knowledge

The goal of subtyping in autism – reducing phenotypic heterogeneity to produce groups more likely to share a common etiology or to predict response to treatment – seems logical. However, this approach has not always yielded the anticipated results, particularly with respect to identifying behaviors that reflect distinct etiologies. While some studies using subtypes to stratify samples have shown increased associations in genetic studies and identified QTLs related to particular subtypes, results have varied. Sometimes associations have not been significant, whereas in other studies, areas implicated have differed. On the other hand, defining subtypes of “responders” to different treatments has yielded promising results in the sense that some behaviors seem to differentially predict responses to certain treatments. However, thus far, only a few studies have characterized “responders” to treatment; many of these subtypes have yet to be replicated, and more research is needed to determine how “responders” to a given treatment respond to similar types of interventions (e.g., naturalistic interventions delivered by a therapist vs. a parent).

Small sample sizes present a significant challenge in the interpretation of discrepant results across studies (Charman et al. 2011). As noted above, finding subtypes that are independent of other characteristics, such as age and IQ, has also been challenging. An additional concern is whether the instruments used to define subtypes result in accurate classification.

Future Directions

Autism research has rapidly expanded over the last several decades, and the development and refinement of diagnostic measures now widely used across studies will facilitate our investigations of subtypes in ASD. Efforts should be made to establish subtypes using multiple measures (i.e., direct observation and parent report), with a focus on current information or corroborating information from retrospective reports with prior documentation whenever possible. The formation

of multisite collaborations and national databases, such as the Simons Foundation Autism Research Initiative (SFARI) and National Database for Autism Research (NDAR), will further promote these types of studies by providing larger samples and increased statistical power. However, we must be cognizant of factors that may influence interpretation of our results from these studies, such as ascertainment biases and site-specific effects. Thus, it will also be important for investigators conducting population-based studies to compile their samples to allow for sufficiently sized samples in which to test subtypes. Collaboration across longitudinal studies is also needed to allow examination of subtypes across several points in time as well as to take a developmental approach to identify subtypes based upon changes in behavior over time (Richler et al. 2010).

With advances in technology providing more sophisticated and sensitive measures of biological mechanisms, our understanding of how brain systems relate to behavior in both clinical and nonclinical samples has increased exponentially in the last decade, thereby enhancing our ability to investigate how behavioral subtypes in ASD may relate to neurobiological mechanisms (Charman et al. 2011). It is important that we use this knowledge to begin to draw and test hypotheses as to how some of the noted genetic variations may influence the behavioral phenotypes with which they are associated. Although some studies have begun to investigate how genes implicated in autism may affect neural mechanisms, there is still a great deal of interest in connecting behavior with particularly genotypes, which overlooks the complexity of the relationship between genes and behavior. It will be important for future investigations of subtypes to examine how gene expression influences brain structure and function to produce behavior in particular groups of individuals as well as how interactions between genes and between genes and the environment influence the brain (Abrahams and Geschwind 2008).

As behavioral subtypes are defined, it is important to remember that not all subtypes will be strongly associated with specific genotypes (Meyer-Lindenberg 2010). Thus, it is also

important that future efforts be made to investigate the clinical utility of a given subtype (e.g., whether certain behavioral subtypes are differentially responsive to particular types of treatment). To facilitate the identification of subtypes of “responders” to treatment, it may be necessary for intervention researchers to establish an agreed upon set of measures that will be considered as potential moderators to treatment. Similar measures across studies will facilitate the establishment of clear-cut parameters to define subtypes of treatment “responders.” Clearly defined subtypes will be particularly important in translating these results to clinical practice, as references to “more impaired skills” relative to the “nonresponders” are not useful in the context of deciding whether a young client is likely to benefit from a given intervention. Furthermore, requiring all studies to collect similar information will allow for comparison of how a subtype of “responders” to one treatment may benefit from other interventions. This would allow for the treatments to which the child is least likely to respond to be “ruled out” while still providing options that can then be considered with respect to other factors influencing response to treatment (e.g., parent and family characteristics, cultural factors, etc.; see Schreibman and Ingersoll 2011). It is also important to highlight that, in addition to defining subtypes of “responders,” significant efforts must also be dedicated to understanding “nonresponders” and to ensure that alternative treatments are identified for these groups. Future studies should also examine treatment response in older children and may consider examining subtypes of children who benefit from group interventions, such as social skills groups.

The search for subtypes to decrease heterogeneity in ASD may lead to a better understanding of the pathophysiology contributing to this complex disorder and enhance our ability to recommend the most appropriate treatments for certain groups of children. While much research has been done in this area, few subtypes have been definitively linked to biological mechanisms and there is not yet sufficient data to clearly define parameters of child characteristics predicting response to treatment. Research in the next decade will be

crucial to determining whether and how subtypes can be used in clinical practice.

See Also

- Endophenotypes
- Factors Affecting Outcomes

References and Reading

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, 9(5), 341–355. <https://doi.org/10.1038/nrg2346>.
- Banach, R., Thompson, A., Szatmari, P., Goldberg, J., Tuff, L., Zwaigenbaum, L., et al. (2008). Brief report: Relationship between non-verbal IQ and gender in autism. *Journal of Autism and Developmental Disorders*, 39(1), 188–193. <https://doi.org/10.1007/s10803-008-0612-4>.
- Bartak, L., & Rutter, M. (1976). Differences between mentally retarded and normally intelligent autistic children. *Journal of Autism and Childhood Schizophrenia*, 6(2), 109–120. <https://doi.org/10.1007/BF01538054>.
- Bennett, T., Szatmari, P., Bryson, S., Volden, J., Zwaigenbaum, L., Vaccarella, L., et al. (2008). Differentiating autism and Asperger syndrome on the basis of language delay or impairment. *Journal of Autism and Developmental Disorders*, 38(4), 616–625. <https://doi.org/10.1007/s10803-007-0428-7>.
- Cantor, R. M. (2011). Autism endophenotypes and quantitative trait loci. In D. Amaral & G. Dawson (Eds.), *Autism spectrum disorders* (1st ed., p. 1456). New York: Oxford University Press.
- Carter, A. S., Messinger, D. S., Stone, W. L., Celimli, S., Nahmias, A. S., & Yoder, P. (2011). A randomized controlled trial of Hanen’s “More Than Words” in toddlers with early autism symptoms. *Journal of Child Psychology and Psychiatry*, 52(7), 741–752. <https://doi.org/10.1111/j.1469-7610.2011.02395.x>.
- Charman, T., Jones, C., Pickles, A., Simonoff, E., Baird, G., & Happé, F. (2011a). Defining the cognitive phenotype of autism. *Brain Research*, 1380, 10–21. <https://doi.org/10.1016/j.brainres.2010.10.075>.
- Charman, T., Pickles, A., Simonoff, E., Chandler, S., Loucas, T., & Baird, G. (2011b). IQ in children with autism spectrum disorders: Data from the Special Needs and Autism Project (SNAP). *Psychological Medicine*, 41(3), 619–627. <https://doi.org/10.1017/S0033291710000991>.
- Coon, H., Villalobos, M. E., Robison, R. J., Camp, N. J., Cannon, D. S., Allen-Brady, K., et al. (2010). Genome-wide linkage using the Social Responsiveness Scale in Utah autism pedigrees. *Molecular Autism*, 1(1), 8. <https://doi.org/10.1186/2040-2392-1-8>.

- Dufour-Rainfray, D., Vourc'h, P., Tourlet, S., Guilloteau, D., Chalon, S., & Andres, C. R. (2011). Fetal exposure to teratogens: Evidence of genes involved in autism. *Neuroscience and Biobehavioral Reviews*, 35(5), 1254–1265. <https://doi.org/10.1016/j.neubiorev.2010.12.013>.
- Hartley, S. L., & Sikora, D. M. (2009). Sex differences in autism spectrum disorder: An examination of developmental functioning, autistic symptoms, and coexisting behavior problems in toddlers. *Journal of Autism and Developmental Disorders*, 39(12), 1715–1722. <https://doi.org/10.1007/s10803-009-0810-8>.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45(2), 212–229. <https://doi.org/10.1111/j.1469-7610.2004.00215.x>.
- Huerta, M., Bishop, S. L., Gotham, K., Hus, V., Lund, S., Buja, A., et al. (2011, May). *Females with autism spectrum disorder: An analysis of the Simons Simplex Collection*. Presented at the International Meeting for Autism Research, San Diego.
- Hus, V., Pickles, A., Cook, E. H., Jr., Risi, S., & Lord, C. (2007). Using the Autism Diagnostic Interview – Revised to increase phenotypic homogeneity in genetic studies of autism. *Biological Psychiatry*, 61(4), 438–448. <https://doi.org/10.1016/j.biopsych.2006.08.044>.
- Hus, V., Taylor, A., & Lord, C. (2011). Telescoping of caregiver report on the Autism Diagnostic Interview – Revised. *Journal of Child Psychology and Psychiatry*, 52(7), 753–760. <https://doi.org/10.1111/j.1469-7610.2011.02398.x>.
- Joseph, R. (2011). The significance of IQ and differential cognitive abilities for understanding ASD. In D. A. Fein (Ed.), *The neuropsychology of autism* (pp. 281–294). New York: Oxford University Press.
- King, B. H., Hollander, E., Sikich, L., McCracken, J. T., Scahill, L., Bregman, J. D., et al. (2009). Lack of efficacy of citalopram in children with autism spectrum disorders and high levels of repetitive behavior: Citalopram ineffective in children with autism. *Archives of General Psychiatry*, 66(6), 583–590. <https://doi.org/10.1001/archgenpsychiatry.2009.30>.
- Lamb, J. A. (2011). Whole genome linkage and association analyses. In D. Amaral & G. Dawson (Eds.), *Autism spectrum disorders* (1st ed., p. 1456). New York: Oxford University Press.
- Lamb, J. A., Barnby, G., Bonora, E., Sykes, N., Bacchelli, E., Blasi, F., et al. (2005). Analysis of IMGSAC autism susceptibility loci: Evidence for sex limited and parent of origin specific effects. *Journal of Medical Genetics*, 42(2), 132–137. <https://doi.org/10.1136/jmg.2004.025668>.
- LeCouteur, A., Bailey, A., Goode, S., Pickles, A., Robertson, S., Gottesman, I., et al. (1996). A broader phenotype of autism: The clinical spectrum in twins. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 37(7), 785–801.
- Liu, X.-Q., Paterson, A. D., Szatmari, P., & The Autism Genome Project Consortium. (2008). Genome-wide linkage analyses of quantitative and categorical autism subphenotypes. *Biological Psychiatry*, 64(7), 561–570. <https://doi.org/10.1016/j.biopsych.2008.05.023>.
- Lockyer, L., & Rutter, M. (1970). A five- to fifteen-year follow-up study of infantile psychosis: IV. Patterns of cognitive ability. *The British Journal of Social and Clinical Psychology*, 9(2), 152–163.
- Lord, C., Petkova, E., Hus, V., Gan, W., Lu, F., Martin, D. M., Ousley, O., et al. (2012). A multisite study of the clinical diagnosis of different autism spectrum disorders. *Archives of General Psychiatry*, 69(3), 306–313. <https://doi.org/10.1001/archgenpsychiatry.2011.148>.
- Meyer-Lindenberg, A. (2010). Intermediate or brainless phenotypes for psychiatric research? *Psychological Medicine*, 40(7), 1057–1062. <https://doi.org/10.1017/S0033291709991929>.
- Miles, J. H. (2011). Autism subgroups from a medical genetics perspective. In D. Amaral & G. Dawson (Eds.), *Autism spectrum disorders*. New York: Oxford University Press.
- Miles, J. H., & Hillman, R. E. (2000). Value of a clinical morphology examination in autism. *American Journal of Medical Genetics*, 91(4), 245–253. [https://doi.org/10.1002/\(SICI\)1096-8628\(20000410\)91:4<245::AID-AJMG1>3.0.CO;2-2](https://doi.org/10.1002/(SICI)1096-8628(20000410)91:4<245::AID-AJMG1>3.0.CO;2-2).
- Miles, J. H., Takahashi, T. N., Bagby, S., Sahota, P. K., Vaslow, D. F., Wang, C. H., et al. (2005). Essential versus complex autism: Definition of fundamental prognostic subtypes. *American Journal of Medical Genetics. Part A*, 135A(2), 171–180. <https://doi.org/10.1002/ajmg.a.30590>.
- Molloy, C. A., Keddache, M., & Martin, L. J. (2005). Evidence for linkage on 21q and 7q in a subset of autism characterized by developmental regression. *Molecular Psychiatry*, 10(8), 741–746. <https://doi.org/10.1038/sj.mp.4001691>.
- Newschaffer, C. J., Fallin, D., & Lee, N. (2002). Heritable and nonheritable risk factors for autism spectrum disorders. *Epidemiologic Reviews*, 24(2), 137–153. <https://doi.org/10.1093/epirev/mxf010>.
- Ozonoff, S., Heung, K., & Thompson, M. (2011). Regression and other patterns of onset. In D. Amaral & G. Dawson (Eds.), *Autism spectrum disorders* (1st ed., p. 1456). New York: Oxford University Press.
- Pickles, A., Simonoff, E., Conti-Ramsden, G., Falcato, M., Simkin, Z., Charman, T., et al. (2009). Loss of language in early development of autism and specific language impairment. *Journal of Child Psychology and Psychiatry*, 50(7), 843–852. <https://doi.org/10.1111/j.1469-7610.2008.02032.x>.
- Pierce, K., & Eyler, L. T. (2011). Structural and functional brain development in ASD: The impact of early brain overgrowth and considerations for treatment. In D. A. Fein (Ed.), *The neuropsychology of autism*. New York: Oxford University Press.
- Posey, D. J., Lodin, Z., Erickson, C. A., & Stigler, K. A. (2011). The neurochemistry of ASD. In D. A. Fein (Ed.), *The neuropsychology of autism*. New York: Oxford University Press.

- Richler, J., Huerta, M., Bishop, S. L., & Lord, C. (2010). Developmental trajectories of restricted and repetitive behaviors and interests in children with autism spectrum disorders. *Development and Psychopathology*, 22(1), 55–69. <https://doi.org/10.1017/S0954579409990265>.
- Rodier, P. M., Ingram, J. L., Tisdale, B., Nelson, S., & Romano, J. (1996). Embryological origin for autism: Developmental anomalies of the cranial nerve motor nuclei. *The Journal of Comparative Neurology*, 370(2), 247–261. [https://doi.org/10.1002/\(SICI\)1096-9861\(19960624\)370:2<247::AID-CNE8>3.0.CO;2-2](https://doi.org/10.1002/(SICI)1096-9861(19960624)370:2<247::AID-CNE8>3.0.CO;2-2).
- Rutter, M., Le Couteur, A., & Lord, C. (2003). *Autism diagnostic interview-revised*. Los Angeles: Western Psychological Services.
- Schreibman, L., & Ingersoll, B. (2011). Naturalistic approaches to early behavioral intervention. In D. Amaral & G. Dawson (Eds.), *Autism spectrum disorders* (1st ed., p. 1456). New York: Oxford University Press.
- Spiker, D., Lotspeich, L. J., Dimiceli, S., Myers, R. M., & Risch, N. (2002). Behavioral phenotypic variation in autism multiplex families: Evidence for a continuous severity gradient. *American Journal of Medical Genetics*, 114(2), 129–136. <https://doi.org/10.1002/ajmg.10188>.
- Stone, J. L., Merriman, B., Cantor, R. M., Yonan, A. L., Gilliam, T. C., Geschwind, D. H., et al. (2004). Evidence for sex-specific risk alleles in autism spectrum disorder. *American Journal of Human Genetics*, 75(6), 1117–1123. <https://doi.org/10.1086/426034>.
- Szatmari, P., Mérette, C., Emond, C., Zwaigenbaum, L., Jones, M. B., Maziade, M., et al. (2008). Decomposing the autism phenotype into familial dimensions. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*, 147B(1), 3–9. <https://doi.org/10.1002/ajmg.b.30561>.
- Tager-Flusberg, H., Edelson, L., & Luyster, R. (2011). Language and communication in autism spectrum disorders. In D. Amaral & G. Dawson (Eds.), *Autism spectrum disorders* (1st ed., p. 1456). New York: Oxford University Press.
- Thurm, A., Lord, C., Lee, L., & Newschaffer, C. J. (2006). Predictors of language acquisition in preschool children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(9), 1721–1734. <https://doi.org/10.1007/s10803-006-0300-1>.
- Tomblin, B. J., McGregor, K., & Bean, A. (2011). Specific language impairment. In D. Amaral & G. Dawson (Eds.), *Autism spectrum disorders* (1st ed., p. 1456). New York: Oxford University Press.
- Veenstra-VanderWeele, J., & Anderson, G. M. (2011). The serotonin system in autism. In E. Hollander, A. Kolevzon, & J. T. Coyle (Eds.), *Textbook of autism spectrum disorders*. Arlington: American Psychiatric Publishing.
- Venter, A., Lord, C., & Schopler, E. (1992). A follow-up study of high-functioning autistic children. *Journal of Child Psychology and Psychiatry*, 33(3), 489–597. <https://doi.org/10.1111/j.1469-7610.1992.tb00887.x>.
- Xu, B., Roos, J. L., Levy, S., van Rensburg, E. J., Gogos, J. A., & Karayiorgou, M. (2008). Strong association of de novo copy number mutations with sporadic schizophrenia. *Nature Genetics*, 40(7), 880–885. <https://doi.org/10.1038/ng.162>.

Success Factors Enabling Employment for Adults on the Autism Spectrum

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Definition

Factors important for improving the outcomes of individuals on the autism spectrum engaging in competitive or open employment.

Historical Background

Individuals on the autism spectrum bring unique strengths and abilities to the workplace and often perform well in job tasks that require a high degree of accuracy in visual perception, systematic information processing, and precise technical abilities (Dreaver et al. 2019). While the employment support needs of this group are diverse, given the right supports and capitalizing on their strengths, individuals on the autism spectrum can be successfully employed in a variety of competitive work environments (Hendricks 2010). Work is a source of economic independence with many

benefits beyond financial gain, including positive effects on physical and mental health, a sense of accomplishment, opportunities for socialization and community engagement, and facilitating a contribution to society, with less reliance on government funding (Scott et al. 2017). However, for many individuals on the autism spectrum, the process of finding and securing a job is a challenging process, often resulting in unemployment. Unemployment rates for individuals on the autism spectrum are higher than that of individuals with other disabilities, and the general population, with these poor employment outcomes observed internationally (Black et al. 2019). Even when individuals on the autism spectrum are employed, it is often in positions well below their education and skill level, working for fewer hours and earning less wages per week compared to those with other disabilities (Shattuck et al. 2012).

Unemployment and underemployment may be attributed to the core characteristics experienced by those on the autism spectrum, including difficulties in social reciprocity and communication, and restricted or repetitive interests or behaviors (Hendricks 2010). While the individual difficulties experienced by people on the autism spectrum in obtaining a job may vary, many find it challenging to promote themselves in a traditional interview, flexibly adjust to new work environments and routines, plan and manage a multi-task workload, respond to unexpected changes in the workplace, recognize expected social norms and meet the communication demands of the workplace (Hendricks 2010; Richards 2012; Shattuck et al. 2012). Workplace settings can be complex, generally relying on self-management skills to navigate and cope with workplace demands. These environments, coupled with a pervasive lack of awareness and understanding in the workplace, and inadequate accommodations underpin the need for individualized vocational support for people on the autism spectrum (Richards 2012).

The high rates of unemployment among individuals on the autism spectrum suggest that factors beyond core characteristics are influencing their employability. It is likely that a variety of environmental factors are barriers to work

participation, including a lack of specific vocational support services, traditional job advertising and interviewing processes, and limited workplace accommodations (Scott et al. 2017). While vocational support services aim to improve employment outcomes by assisting individuals on the autism spectrum with recruitment, the interview process, workplace accommodations, and ongoing support, they remain less than optimal (Nicholas et al. 2014). Vocational support services often overlook social support needs, on-the-job training required by employees on the autism spectrum, and the level of education, support, and training required by employers and coworkers to create an inclusive workplace (Dreaver et al. 2019). These services also have a tendency to treat the needs of individuals on the autism spectrum homogeneously, failing to consider the unique and variable profiles of autism (Richards 2012). Another barrier to employment is employers' attitudes toward hiring and supporting individuals with disabilities (Hernandez and McDonald 2010). Employer attitudes in the workplace are well-recognized as influencing their hiring decisions (Unger 2002). Positive employer attitudes are associated with larger organizations, previous experience working with individuals with disabilities and the perceived benefits of retaining a qualified, reliable employee with low rates of absenteeism (Unger 2002). In contrast, negative employer attitudes are influenced by a lack of knowledge regarding disability, misperceptions relating to the unknown additional costs associated with supervision, training, and workplace accommodations and concerns over lost productivity (Hernandez and McDonald 2010). A recent cost-benefit study addressed employers' misperceptions and demonstrated that the cost of employing an individual on the autism spectrum is similar to that of neurotypical employees, and is not associated with any additional costs over and above that associated with a new employee (Scott et al. 2017). In fact, supporting the employment of individuals on the autism spectrum significantly decreases their lifetime reliance on government funding and reduces the costs associated with loss of productivity (Jacob et al. 2015).

For individuals on the autism spectrum, the desire to work is no different than those of the general working population. However, the potential for individuals on the autism spectrum to engage and contribute to the workplace remains untapped, highlighting the need for greater opportunities to engage in meaningful and competitive employment (Dreaver et al. 2019; Hendricks 2010). Employers have the potential capacity to create such opportunities and remove barriers to work participation by implementing workplace modifications, leading workplace culture, diversifying the workplace, and implementing organizational policies and practices (Erickson et al. 2014). Given the heterogeneity in the support needs of individuals on the autism spectrum in finding and maintaining employment, and the complex dynamics of the workplace environment, the factors required to enable employment success are likely diverse in nature. In order to understand these factors, it is essential to consider employment success as a combination of personal and environmental factors working together, rather than independently, to create the best fit in the workplace.

Current Knowledge

Current evidence regarding employment interventions for adults on the autism spectrum support their effectiveness in improving vocational skills, executive functioning in relation to job performance, and employment status outcomes (Hedley et al. 2016; Scott et al. 2018b). Despite the majority of these interventions having the collective purpose of improving employment outcomes, they are primarily impairment-focused, targeting specific characteristics associated with autism, such as social-communication difficulties and behaviors in the workplace, with little consideration of environmental influences (Scott et al. 2018b). There is an increasing amount of evidence suggesting that rather than only focusing on the individual and their intrinsic challenges to finding and securing employment, addressing changes in the environment may be an important enabler for employment success of individuals on

the autism spectrum (Hedley et al. 2016; Nicholas et al. 2018; Scott et al. 2018b). Therefore, it is critical to consider factors that enable employment success from both a personal and environmental perspective.

Personal Factors

Job Match

Both individuals on the autism spectrum and employers have highlighted job matching as underpinning success in finding and maintaining employment (Dreaver et al. 2019). Job matching is described as the process of establishing a “good match” between the individual on the autism spectrum, in terms of the skills, abilities, strengths and interests, and the job. General characteristics of a “good” job match include the following: (1) recognition of, and opportunity to, build on the skills and special interests of the individual; (2) predictable and structured work tasks; (3) adequate time for learning new tasks; (4) flexible work schedules; and (5) opportunities for growth (Dreaver et al. 2019; Müller et al. 2003). It is important to consider job matching from a holistic perspective, i.e., considering the individual’s skills, abilities, and interests and how these can be harnessed in the workplace, as well as the challenges of traits associated with autism. Vocational support services have a responsibility to ensure that individuals on the autism spectrum are matched to a job based on their skills and abilities required for the job and worksite. They also need to ensure that these individuals are “job-ready” through developing the necessary interpersonal skills and behaviors to meet performance and workplace expectations (Dreaver et al. 2019; Müller et al. 2003). Using a strengths-based approach is a useful way of “profiling an individual’s barriers and facilitators in acquiring a job and mitigating their weaknesses by promoting and supporting their strengths” (Scott et al. 2018b, p. 894). Recent literature is advocating for a strengths-based approach, whereby special interests of individuals on the autism spectrum are utilized in promoting workforce integration (Goldfarb et al. 2019).

Environmental Factors

Employers and Coworkers

Employers are a key environmental factor in the employment process, with their attitudes, management practices, and policies critical in facilitating work participation of individuals on the autism spectrum (Erickson et al. 2014; Unger 2002). However, employers remain a largely untapped resource in the work environment given their potential role in hiring decisions, creating employment opportunities, and providing effective supports to individuals on the autism spectrum (Hagner and Cooney 2005). Given the diverse and individualized workplace supports required by individuals on the autism spectrum, employers need to be well equipped to provide adequate support that encourages successful work participation. If the necessary support is not provided, there is a risk that employers and coworkers will rely on their preconceived ideas, attitudes and stigma toward disability in the workplace to guide their decision-making (Dreaver et al. 2019; Unger 2002). An understanding of autism by employers and coworkers has been identified as a key factor in developing positive workplace relationships (Dreaver et al. 2019). Increased knowledge of autism through autism awareness training and education, as well as open-mindedness to diversifying the workforce, has been associated with reduced workplace conflicts and misunderstandings. As a result, employees on the autism spectrum have experienced an increase in confidence in their workplace relationships, resulting in feeling “accepted” and included (Dreaver et al. 2019; Hagner and Cooney 2005; Müller et al. 2003). Inclusive and supportive workplaces are considered significant factors in determining job satisfaction and retention. Therefore, employers and coworkers who are knowledgeable about autism, supportive and flexible in their practices, and who foster positive relationships are influential in their contribution to the employment success (Dreaver et al. 2019; Hendricks 2010; Nicholas et al. 2018).

Workplace Accommodations

Workplace accommodations are a critical component of employment success. Many workplace challenges encountered by employees on the autism spectrum can be mitigated by implementing a variety of workplace accommodations during both pre-employment and once employed (Black et al. 2019; Khalifa et al. 2019). To successfully navigate these challenges, effective management practices are required, whereby employers identify problems that arise in the workplace promptly, determine the most effective strategies or accommodations required, recognize when and/or whether to implement them, and supervise the outcomes accordingly (Hagner and Cooney 2005). Often employers are quick to identify problems, such as challenging behaviors or difficulties following instructions, yet lack the expertise and knowledge in identifying what strategies to implement to assist their employees on the autism spectrum. In order for workplace accommodations to be effective, employers require support and training from autism-specific or general disability vocational service providers to upskill them in delivering individualized supports (Dreaver et al. 2019; Müller et al. 2003). An increase in employer knowledge and understanding of autism increases their capacity to identify and implement appropriate strategies (Dreaver et al. 2019; Scott et al. 2018a). This is important as workplace accommodations for employees on the autism spectrum may include (1) *social support strategies* (e.g., interpreting instructions, understanding job task expectations, teamwork); (2) *adaptive strategies* (e.g., establishing routine and structure in work tasks, alternative job interviews); (3) *cognitive strategies* (e.g., problem-solving strategies, stress management); (4) *physical strategies* (e.g., accommodations to sensory demands of the environment such as noise and light); and (5) *technology-based strategies* (e.g., use of technology-based tools or devices to improve workplace performance by prompting, directing, and managing tasks) (Dreaver et al. 2019; Khalifa et al. 2019; Müller et al. 2003).

Vocational Support Services

Workplace accommodations are also addressed through vocational support services and

employment programs. Vocational support services address the unique challenges confronted by individuals on the autism spectrum in employment, while advocating for their strengths and abilities in the best-suited job and providing ongoing support to both the employee and the employer in the process (Müller et al. 2003). These support services are accessed by individuals on the autism spectrum throughout the employment process and play a pivotal role in securing and retaining a job (Nicholas et al. 2014). Individuals on the autism spectrum who receive job readiness training, job search assistance, job placement services, and ongoing on-the-job support through vocational support services are significantly more likely to achieve and sustain competitive employment, with increased working hours and wages (Hedley et al. 2016; Hendricks 2010; Nicholas et al. 2018). In addition, employment programs, such as the TEACCH program, Project SEARCH, and the National Autistic Society Prospects all focus on improving employment outcomes either through the use of job coaches, internships, or supported employment services (Hedley et al. 2016). Overall, vocational support services and employment programs are integral in enabling employment success and are often considered as a “bridge” between individuals on the autism spectrum and the employer.

Family

The family system, particularly parents or caregivers of individuals on the autism spectrum, plays an influential role in employment outcomes (Nicholas et al. 2018). Many parents are strong advocates for their adult children and often engage in the employment process, beginning in high school with their child’s transition plan into adult life. Parents are key enablers in achieving vocational success, with many assisting their adult children in searching for job opportunities, approaching local community businesses for potential jobs, creating a job in a family business, and navigating services (Nicholas et al. 2018). This prolonged support and advocacy facilitates achieving employment outcomes for individuals on the autism spectrum.

Policy, Procedures, and Practices

Policies and practices related to the employment of people with disabilities are designed to remove barriers and facilitate work participation. Policies and practices that focus on recruitment and hiring, disability-related awareness training for staff, accessible accommodations, and retention have been shown to reduce barriers in the workplace for employees with disabilities (Erickson et al. 2014). While employment policies and practices vary globally, those, which are characterized by increased government support for vocational support services and employment programs, with strong employer obligations, universal coverage, and comprehensive and accessible incentives, have higher employment rates of people with disabilities (Black et al. 2019). Although policies and practices consider employment outcomes of individuals on the autism spectrum from a broader perspective, they have the power to facilitate work participation at an individual, organizational, and societal level (Erickson et al. 2014).

Future Directions

While it is increasingly clear that environmental supports and modifications, job-matching, and harnessing strengths of individuals on the autism spectrum are essential to improving employment prospects, future efforts must be made to support organizations and employers in this task.

A paradigm shift away from focusing on individual deficits and instead using a biopsychosocial approach, such as the International Classification of Functioning, Disability and Health (ICF) that considers the environment alongside an individual’s strengths and difficulties profile, is needed to support the employment of individuals on the autism spectrum (Black et al. 2019). While there is increasing recognition that addressing the environment is important for success, there are limited interventions that directly target this important factor (Scott et al. 2018b). The Integrated Employment Success Tool™ (IEST) is one evidence-based intervention that targets the environment. The IEST™ seeks to provide employers across a variety of workplaces with the tools necessary to support employees on the

autism spectrum (Scott et al. 2018a). Future research should continue to develop and evaluate interventions that target the workplace environment.

Ensuring a match between the individual and the work task is an important factor for success. This may be a daunting task for employers, who may have difficulty understanding the unique strengths and difficulties profile of each of the employees on the autism spectrum. Tools and supports which assist employers to create an environment and job tasks which harnesses individual unique strengths are needed to facilitate a job-match. It must, however, be noted that within current labor markets, ensuring a match between a job and an individual's interests is not always possible for all employees at all times. Goldfarb et al. (2019) suggest that Social Determination Theory may play a critical role in addressing this potential misalignment between the job and an individual's interests, proposing that extrinsic motivators (such as salary), and ensuring that individuals have the opportunity for choice or autonomy, positive social relatedness, and feelings of competence may contribute to work motivation. An integrative approach, combining a strength-based approach that strives for a person-job match, along with Social Determination Theory, may work to promote long-term motivation and employment success. Future research should seek to understand how employers can not only harness an individual's strengths but also how employers and workplaces can ensure that the basic human needs of autonomy, social relatedness, and competence can be met in the workplace (Goldfarb et al. 2019).

Providing early work experience, for example, in high school, may be an important first step to employment for individuals on the autism spectrum. Work experience can provide youth on the autism spectrum with insights into their own unique strengths and difficulties profile in the workplace, enabling them to explore their interests, develop skills, and gain important insights into the workplace (Lee et al. 2019). Opportunities for work experience should be provided to individuals on the autism spectrum, with a focus on how early work experience may be integrated into schools.

See Also

- [Competitive Employment](#)
- [Employment in Adult Life](#)
- [Work Placement for Individuals with Autism Spectrum Disorder](#)

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References and Reading

- Black, M., Mahdi, S., Milbourn, B., Thompson, C., D'Angelo, A., Ström, E., . . . Bölte, S. (2019). Perspectives of key stakeholders on employment of autistic adults across the United States, Australia, and Sweden. *Autism Research*. <https://doi.org/10.1002/aur.2167>.
- Dreaver, J., Thompson, C., Girdler, S., Adolfsson, M., Black, M. H., & Falkmer, M. (2019). Success factors enabling employment for adults on the autism spectrum from employers' perspective. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03923-3>.
- Erickson, W. A., von Schrader, S., Bruyère, S. M., & VanLooy, S. A. (2014). The employment environment: Employer perspectives, policies, and practices regarding the employment of persons with disabilities. *Rehabilitation Counseling Bulletin*, 57(4), 195–208. <https://doi.org/10.1177/0034355213509841>.
- Goldfarb, Y., Gal, E., & Golan, O. (2019). A conflict of interests: A motivational perspective on special interests and employment success of adults with ASD. *Journal of Autism and Developmental Disorders*, 49(9), 3915–3923. <https://doi.org/10.1007/s10803-019-04098-7>.
- Hagner, D., & Cooney, B. F. (2005). 'I do that for everybody': Supervising employees with autism. *Focus on Autism and Other Developmental Disabilities*, 20(2), 91–97. <https://doi.org/10.1177/10883576050200020501>.
- Hedley, D., Uljarevic, M., Cameron, L., Halder, S., Richdale, A., & Dissanayake, C. (2016). Employment programmes and interventions targeting adults with autism spectrum disorder: A systematic review. *Autism*, 21, 929–941.
- Hendricks, D. (2010). Employment and adults with autism spectrum disorders: Challenges and strategies for success. *Journal of Vocational Rehabilitation*, 32(2), 125–134.
- Hernandez, B., & McDonald, K. (2010). Exploring the costs and benefits of workers with disabilities. *Journal of Rehabilitation*, 76(3), 15–23.
- Jacob, A., Scott, M., Falkmer, M., & Falkmer, T. (2015). The costs and benefits of employing an adult with

autism spectrum disorder: A systematic review. *PLoS One*, 10(10), e0139896. <https://doi.org/10.1371/journal.pone.0139896>.

Khalifa, G., Sharif, Z., Sultan, M., & Di Rezze, B. (2019). Workplace accommodations for adults with autism spectrum disorder: A scoping review. *Disability and Rehabilitation*, 1–16. <https://doi.org/10.1080/09638288.2018.1527952>.

Lee, E. A. L., Black, M. H., Tan, T., Falkmer, T., & Girdler, S. (2019). “I’m destined to ace this”: Work experience placement during high school for Individuals with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49(8), 3089–3101. <https://doi.org/10.1007/s10803-019-04024-x>.

Müller, E., Schuler, A., Burton, B. A., & Yates, G. B. (2003). Meeting the vocational support needs of individuals with Asperger syndrome and other autism spectrum disabilities. *Journal of Vocational Rehabilitation*, 18, 163–175.

Nicholas, D. B., Attridge, M., Zwaigenbaum, L., & Clarke, M. (2014). Vocational support approaches in autism spectrum disorder: A synthesis review of the literature. *Autism*. <https://doi.org/10.1177/1362361313516548>.

Nicholas, D. B., Mitchell, W., Dudley, C., Clarke, M., & Zulla, R. (2018). An ecosystem approach to employment and autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(1), 264–275. <https://doi.org/10.1007/s10803-017-3351-6>.

Richards, J. (2012). Examining the exclusion of employees with Asperger syndrome from the workplace. *Personnel Review*, 41(5), 630–646. <https://doi.org/10.1108/00483481211249148>.

Scott, M., Jacob, A., Hendrie, D., Parsons, R., Girdler, S., Falkmer, T., & Falkmer, M. (2017). Employers’ perception of the costs and the benefits of hiring individuals with autism spectrum disorder in open employment in Australia. *PLoS One*, 12(5), 1–16. <https://doi.org/10.1371/journal.pone.0177607>.

Scott, M., Falkmer, M., Falkmer, T., & Girdler, S. (2018a). Evaluating the effectiveness of an autism-specific workplace tool for employers: A randomised controlled trial. *Journal of Autism and Developmental Disorders*, 48(10), 3377–3392. <https://doi.org/10.1007/s10803-018-3611-0>.

Scott, M., Milbourn, B., Falkmer, M., Black, M., Bölte, S., Halladay, A., . . . Girdler, S. (2018b). Factors impacting employment for people with autism spectrum disorder: A scoping review. *Autism*. <https://doi.org/10.1177/1362361318787789>.

Shattuck, P. T., Narendorf, S. C., Cooper, B., Sterzing, P. R., Wagner, M., & Taylor, J. L. (2012). Post-secondary education and employment among youth with an autism spectrum disorder. *Pediatrics*, 129(6), 1042–1049.

Unger, D. D. (2002). Employers’ attitudes toward persons with disabilities in the workforce: Myths or realities? *Focus on Autism and Other Developmental Disabilities*, 17(1), 2–10. <https://doi.org/10.1177/108835760201700101>.

Successful School Transition

► [Barriers to and Facilitators of Successful Early School Transitions for Children with Autism Spectrum Disorders](#)

Successive Integration

► [Sequential Processing](#)

Successive Processing

► [Sequential Processing](#)

Suicidality in Children and Adolescents with Autism

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Definition

Suicide may be defined as death caused by a self-inflicted injury with the intent to kill oneself. The umbrella term, suicide, encompasses a spectrum of classifications varying in degree of intensity from suicidal ideation (thoughts of wanting to end one’s life) to suicide attempts (completed or aborted efforts).

Historical Background

Research in autism spectrum disorders (ASD) is a nascent field compared to other psychiatric disorders. With recent recognition of more cognitive

able forms of ASD (e.g., Asperger's Disorder in the *Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition [DSM-IV]*), there has been greater focus on comorbidities, including mood disorders and suicide. In this regard, suicide is a minimally studied and poorly understood phenomenon in ASD.

Current Knowledge

In the United States, suicide is the 2nd leading cause of death for children and adolescents between the ages of 10 and 17 (Centers for Disease Control 2014). Specific demographic variables are considered risk factors for suicide. In nonclinical populations, older age (within the period of adolescence) and male sex increase the risk of suicide five-fold (Centers for Disease Control 2015). In addition to age and gender, being victimized or socially isolated by peers and having co-morbid psychiatric disorders, such as depression and anxiety, are risk factors for suicidal ideation and suicidal behavior in typical children and adolescents.

Suicide and Autism. A limited number of studies have investigated suicidal ideation and suicidal behavior in youths with ASD. These studies have yielded varying rates of suicidality. In a Japanese sample of 94 adolescents hospitalized for a suicide attempt, 12 (13%) met the diagnostic criteria for ASD and of those 12, five (42%) had previous histories of suicide attempts. Six of the twelve adolescents (50%) had a history of psychiatric difficulties (Mikami et al. 2009). Similarly, in a small sample of 10 adolescents, 50% endorsed clinically significant levels of suicidal ideation (Shtayermman 2008). Other studies fail to corroborate such a high rate of suicidality in ASD; for example, 20 of 102 (20%) youths diagnosed with ASD and co-morbid anxiety endorsed suicidal thoughts and behavior on a self-report measure, and 11 of the 102 (11%) displayed having suicidal thoughts (Storch et al. 2013). The prevalence of suicidal thoughts in children and adolescents with ASD has also been explored through parent report. In comparison to 0.5% of mothers of typically developing controls ($N=186$), between 13% and 14% of mothers of children with autism

($N=791$) rated their child as having suicidal ideation or attempts as being “sometimes to very often a problem” (Mayes et al. 2013). Given the limited and inconsistent research on suicide in ASD, this represents an important area for further investigation.

Risk Factors

Demographic Variables

Age, gender, race, and socioeconomic status are demonstrated risk factors of suicidal ideation or attempts in children with autism (Mayes et al. 2013). More specifically, youths with ASD were found to be at a higher risk for suicidal ideation or suicidal behavior if they were found to be: 10 years of age or older, of the male sex, of the African American or Hispanic race, and/or of a lower socioeconomic status. Children with ASD over the age of 10 endorsed having suicidal ideation or attempts three times more than children younger than 10 years of age. The frequency of suicidal ideation or attempts was twice as high in males with ASD than in females with ASD (Mayes et al. 2013).

Symptom Severity

Studies have yielded conflicting results about the relationship between the severity of autism symptoms and frequency of suicidal ideation or attempts. Children diagnosed with Autistic Disorder were more likely to have suicidal thoughts or behaviors than those diagnosed with Asperger Disorder (Storch et al. 2013). Additionally, strong negative correlations have been found between level of suicidal ideation and severity of autism symptoms (Shtayermman 2008). This relationship between symptom severity and suicidal ideation is not evident in all samples. Mayes and colleagues (2013) found no relationship between frequency of suicidal ideation or attempts and autism severity.

Comorbidities

Suicidal ideation and suicide attempts are associated with multiple psychiatric illnesses in both typical development and ASD including: depression, anxiety, bi-polar, and posttraumatic stress disorder (PTSD). Depression and anxiety are the

leading co-morbid diagnoses in adolescents with ASD (Storch et al. 2013). Mayes et al. (2013) examined rates of depression in a sample of 350 children and adolescents with ASD (ages 6–16); they found that 54% of youths with higher functioning autism and 42% of youths with lower functioning autism were reported (by their parent) to exhibit depressed mood. Additionally, depression, impulsivity, and behavioral difficulties were significant predictors of suicidal ideation or attempts in children with ASD (Mayes et al. 2013). Similarly, in a sample of 23 children and 14 adolescents with Asperger's Disorder, 35 children and adolescents had at least one additional psychiatric diagnosis (Mukaddes and Fateh 2010). The most common psychiatric disorders for this group were anxiety disorders (54%), disruptive behavior disorders (48%), and mood disorders (37%). Furthermore, 42% of the adolescents were found to have suicidal behaviors; these individuals were diagnosed with both a mood and an anxiety disorder (Mukaddes and Fateh 2010). Other research has found similar rates of suicidal thoughts and behavior in youths with ASD and anxiety (11%) as compared to neurotypical youths without anxiety (14%), suggesting that suicidality is present to a similar degree (Storch et al. 2013; Wunderlich et al. 2001). It has also been found that having a comorbid diagnosis of PTSD is associated with increased risk of suicide in youths with ASD (Storch et al. 2013).

Bullying

Bullying has been defined by researchers as the repeated exposure to aggressive acts, directly or indirectly, over time that are intended to harm the individual physically, emotionally, or socially (Sofronoff et al. 2011; Blake et al. 2012). The act of bullying can come in many forms including: name calling, spreading rumors, purposeful exclusion from events or activities, physical aggression, and targeted cyber-attacks. A review of research investigating suicide and bullying among youths in the general population revealed that victims of bullying exhibited higher levels of suicidal ideation and were more likely to attempt suicide (Klomek et al. 2010; Geoffroy et al. 2016). Furthermore, being bullied at school was found

to be a significant risk factor for suicidal thoughts and behaviors, independent of other established risk factors such as depression, socioeconomic status, and biological sex (Kaminski and Fang 2009).

It has been shown that children and adolescents with ASD are at increased risk of peer victimization, including social isolation and bullying (Locke et al. 2010; Cappadocia et al. 2012). Repeated victimization was found to be significantly higher for both elementary and middle school students with ASD (Blake et al. 2012). Moreover, research has indicated that the rate at which youths with ASD are victimized varies and this variation may be influenced by the perspective of the informant and their ability to report accurately. Bullying has been reported at higher rates by both by children with ASD and their mothers when compared to neurotypical youths and their mothers (Zeedyk et al. 2014). Furthermore, youth's self-report of the emotional impact of bullying was significantly higher for the ASD group compared to neurotypical youths (Zeedyk et al. 2014). Additionally, in a general education setting, teachers reported higher rates of bullying of children with ASD compared to peer and self-reports (Van Roekel et al. 2010).

There are a number of factors that may contribute to the increased rate of bullying experienced by students with ASD. This increased risk may be due to the child's limited ability to understand the social world as well as having other psychiatric disorders present. Children with ASD who have experienced more victimization were more likely to be rated by their parents have having other psychiatric difficulties such as higher levels of anxiety than those children who experienced no peer victimization (Cappadocia et al. 2012). It will be critical for future research to determine whether the increased risk of bullying in ASD is associated with an increased risk for the development of suicidal thoughts and behaviors.

Assessment

There is not currently a specific assessment tool to measure specific suicide risk in individuals with ASD, and there are numerous challenges to

assessing risk in this population. First, youths with ASD commonly have difficulties with expressive language, and this is often particularly pronounced for social-emotional matters, complicating assessment of emotional distress (Losh and Capps 2006). Paired with reduced insight into one's own emotions, these factors decrease the effectiveness of standard risk assessment methods in this population.

Prevention

It is recommended that parents and professionals working with children and adolescents on the autism spectrum take thoughts or mentions of suicide very seriously. Any comments made regarding thoughts of wanting to hurt or kills oneself should be considered significant and promptly brought to the attention of a qualified mental health professional or emergency medical center. Aware of predisposing risk factors, such as a family history of suicide attempts, can support early recognition of suicidal ideation prior to the emergence of suicidal behaviors.

In summation, though suicidality in autism is of great relevance to public health, existing research is scant. Particular areas requiring further investigation include assessment of suicide risk in ASD and strategies for psychoeducation for children and adolescents with ASD.

Future Directions

The aforementioned work highlights multiple topics that must be investigated in the future studies. It will be important to understand how children and adolescents with ASD think about and understand the concept of suicide. This will inform more accurate assessment of risk. Additional exploration of risk factors (e.g., social isolation, comorbid psychiatric disorders), protective factors, and the interplay between these factors is needed to shed light on high suicide prevalence rates for children and adolescents with ASD compared to the general population.

See Also

- Anxiety
- Bullying

References and Reading

- Blake, J. J., Lund, E. M., Zhou, Q., Kwok, O. M., & Benz, M. R. (2012). National prevalence rates of bully victimization among students with disabilities in the United States. *School Psychology Quarterly*, 27(4), 210.
- Cappadocia, M. C., Weiss, J. A., & Pepler, D. (2012). Bullying experiences among children and youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(2), 266–277.
- Centers for Disease Control and Prevention. (2014). Ten leading causes of death by age group, United States – 2014. Retrieved from https://www.cdc.gov/injury/images/lc-charts/leading_causes_of_death_age_group_2014_1050w760h.gif
- Centers for Disease Control and Prevention. (2015). Suicide: At a glance. Retrieved from <https://www.cdc.gov/violenceprevention/pdf/suicide-datasheet-a.pdf>
- Geoffroy, M.-C., Boivin, M., Arseneault, L., Turecki, G., Vitaro, F., Brendgen, M., et al. (2016). Associations between peer victimization and suicidal ideation and suicide attempt during adolescence: Results from a prospective population-based birth cohort. *Journal of the American Academy of Child and Adolescent Psychiatry*, 55(2), 99–105.
- Kaminski, J. W., & Fang, X. (2009). Victimization by peers and adolescent suicide in three US samples. *The Journal of Pediatrics*, 155(5), 683–688.
- Klomek, A. B., Sourander, A., & Gould, M. (2010). The association of suicide and bullying in childhood to young adulthood: A review of cross-sectional and longitudinal research findings. *Canadian Journal of Psychiatry*, 55(5), 282–288.
- Locke, J., Ishjima, E. H., Kasari, C., & London, N. (2010). Loneliness, friendship quality, and the social networks of adolescents with high-functioning autism in an inclusive school setting. *Journal of Research in Special Education Needs*, 10(2), 74–81.
- Losh, M., & Capps, L. (2006). Understanding of emotional experience in autism: Insights from the personal accounts high-functioning children with autism. *Developmental Psychology*, 42(5), 809.
- Mayes, S. D., Gorman, A. A., Hillwig-Garcia, J., & Syed, E. (2013). Suicide ideation and attempts in children with autism. *Research in Autism Spectrum Disorders*, 7(1), 109–119.
- Mikami, K., Inomata, S., Hayakawa, N., Ohnishi, Y., Enseki, Y., Ohya, A., et al. (2009). Frequency and clinical features of pervasive developmental disorder in adolescent suicide attempts. *General Hospital Psychiatry*, 31, 163–166.

- Mukaddes, N. M., & Fateh, R. (2010). High rates of psychiatric co-morbidity in individuals with Asperger's disorder. *The World Journal of Biological Psychiatry*, 11(2-2), 486–492.
- Shtayemman, O. (2008). Suicidal ideation and comorbid disorders in adolescents and young adults diagnosed with Asperger's syndrome: A population at risk. *Journal of Human Behavior in the Social Environment*, 18(3), 301–332.
- Sofronoff, K., Dark, E., & Stone, V. (2011). Social vulnerability and bullying in children with Asperger syndrome. *Autism*. <https://doi.org/10.1177/1362361310365070>.
- Storch, E. A., Sulkowski, M. L., Nadeau, J., Lewin, A. B., Arnold, E. B., Mutch, P. J., . . . Murphy, T. K. (2013). The phenomenology and clinical correlates of suicidal thoughts and behaviors in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(10). <https://doi.org/10.1007/s10803-10013-11795-x>.
- Van Roekel, E., Scholte, R. H. J., & Didden, R. (2010). Bullying among adolescents with autism spectrum disorders: Prevalence and perception. *Journal of Autism and Developmental Disorders*, 40(1), 63–73.
- Wunderlich, U., Bronisch, T., Wittchen, H. U., & Carter, R. (2001). Gender differences in adolescents and young adults with suicidal behaviour. *Acta Psychiatrica Scandinavica*, 104(5), 332–339.
- Zeedyk, S. M., Rodriguez, G., Tipton, L. A., Baker, B. L., & Blacher, J. (2014). Bullying of youth with autism spectrum disorder, intellectual disability, or typical development: Victim and parent perspectives. *Research in Autism Spectrum Disorders*, 8(9), 1173–1183.

Suicide Rates in Adults with Autism

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Definition

Suicide can be defined as the deliberate taking of one's life. A leading cause of death worldwide, it is the most extreme form of a range of behaviors which include suicidal thoughts and attempts. There is no single cause of suicide. Risk factors include presence of mental illness particularly depression, family history of suicide, substance abuse, chronic medical illness, and exposure to

stressful life events. While suicidal attempts occur more commonly in females, completed suicide is more common in males.

Autism and Suicidal Behavior

Prevalence

Suicidal behavior is being recognized as an emerging problem in persons with autism spectrum disorders (ASD), particularly in those with a normal IQ (high-functioning autism, HFA). Although it occurs in both children and adults with ASD, relatively little research has been done on the clinical correlates and mechanisms underlying suicidal behavior in this population. Most of the research on this topic has been done in clinical samples. Prevalence rates based on epidemiological studies of suicidal behavior in individuals with ASD are not known.

Completed Suicide

While suicidal behavior has been described in several recent studies, reports of *completed suicide* in persons with ASD are uncommon. Most of these have involved persons with HFA or those with mild to moderate degree of intellectual disability (ID) with relatively good verbal skills and have been reported in the newspapers. For example, Nikki Bacharach, the 40-year-old daughter of the composer Burt Bacharach, committed suicide by suffocating herself using a plastic bag and helium. She reportedly had a diagnosis of Asperger syndrome (AS) with depression (2013). In a recent Italian study of 26 adults with ASD, two (7.7%) committed suicide; one had PDDNOS, schizophrenia, and alcohol abuse by jumping from a bridge and the other with AS and schizophrenia, by dismemberment (see Raja et al. 2011). In the USA, a recent problem has been that of mass school shootings, characterized by indiscriminate violence against others, followed by suicide of the perpetrator. At least two of the individuals involved in such incidents were known to have been diagnosed with ASD during their lifetime (Farrell 2016; Ghaziuddin 2013).

Indirect evidence of completed suicide in this population also comes from mortality studies of individuals with autism. For example, Mouridsen and colleagues (2008) examined the causes of death in a clinical sample of patients with ASD, aged 2–17 years, in Denmark. They found that the mortality rate was increased in persons with autism compared to that of the rest of the population. Among the 26 deaths recorded, 5 patients died from unnatural causes, 3 in accidents (suffocation and drowning), and 2 by suicide. Both patients who committed suicide were male of average intelligence. One, with a diagnosis of atypical autism, took an overdose of medications, while the other, with a diagnosis of Asperger syndrome, jumped to his death (Mouridsen et al. 2008). Mortality studies of autism are remarkable in suggesting not only higher rates compared to that of the general population but also pointing out the high occurrence of accidents, such as drowning and suffocation (Shavelle et al. 2001). It is possible that suicidal behavior could have contributed to some of these accidents.

Suicidal Ideation and Attempts in Adults

In her paper on Asperger syndrome, Lorna Wing described two patients who had made a suicidal attempt and another who had made suicidal statements (Wing 1981). Subsequently, Wolff and McGuire (1995) reported that out of 49 subjects with “schizoid personality disorder,” many of whom were probably on the autistic spectrum, 10 out of 17 women and 17 out of 32 men had attempted suicide, a rate higher than that in the general population (Wolff and McGuire 1995). Kato and colleagues examined the characteristics of adults with ASD presenting to the emergency room with suicidal behavior (Kato et al. 2013). ASD patients were compared with non-ASD patients on psychiatric diagnoses, frequency of suicidal attempts, and clinical features. Out of the 587 subjects who attempted suicide, 43 (7.3%) had ASD. In a survey of parents of adult children with ASD carried out by the UK National Autism Society (NAS 2001), about a third of parents reported that their son or daughter had experienced mental health problems, out of which about half suffered from depression and 8% from suicidal behavior including attempted

suicide (Barnard et al. 2001). In a recent study, Cassidy et al. (2014) explored suicidal behavior in adults with Asperger syndrome. Two-thirds of the sample reported that they had thought about committing suicide at some point, and 35% had made specific plans to kill themselves.

Suicidal Ideation and Attempts in Children and Adolescents

In a study of 537 subjects with high-functioning autism (HFA, defined as IQ 80) and 254 subjects with low-functioning autism (LFA, IQ <80), 1–16 years of age, subjects’ mothers were asked about suicidal behavior in their offspring. The 13.8% of children with autism rated suicide ideation or attempts as a problem, compared with 0.5% for typical children and 42.9% for depressed children ($\chi^2 = 55.6, p < 0.0001$). There were no differences between high-functioning and low-functioning autistic patients (Mayes et al. 2013). In a retrospective chart review of 233 patients admitted to a specialized service for children with developmental disabilities and comorbid psychiatric disorders, Hardan and Sahl found that 47 (20%) had a past or present history of suicidal ideation or attempt. Three subjects had autism and four had PDDNOS. Suicidal behavior was more common in patients with depression, oppositional defiant disorder (ODD), and post-traumatic stress disorder (PTSD) than in those with autism and profound mental retardation. The authors speculated that autism and/or profound intellectual disability acted as protective factors against suicide (Hardan and Sahl 1999). Based on a chart review, Mikami et al. (2009) reported that 12 (12.8%) out of a consecutive series of 94 adolescent patients (aged under 20 years) admitted to an inpatient unit with suicidal behavior were diagnosed with having ASD. Six cases had Asperger syndrome and six had PDDNOS. Life events, such as bullying, were common. Interestingly, depression was underrepresented in the ASD group compared to the non-ASD group (Mikami et al. 2009).

Presentation

The presentation of suicidal behavior in persons with ASD depends on the level of intelligence and verbal skills. Persons with HFA can better

verbalize their feelings and admit to suicidal thoughts and behavior (Ghaziuddin et al. 2002). A small number of adolescents with ASD, often with average level of intelligence, may present with superficial self-cutting and make repeated statements to die but show no clear suicidal intent on examination. These should be differentiated from low-functioning individuals with ASD, often with the IQ in the severe to profoundly disabled range, who indulge in repetitive self-injurious behaviors such as head banging, self-biting, and other aggressive behaviors. In the latter group, the intent to commit suicide is not expressed. The assessment of suicidal behavior in autistic persons with very low IQ is complicated. Because of communication problems, these patients cannot verbalize their feelings and may, therefore, act out in other ways, such as aggression toward self or others. When present, a history of recent change in behavior with frequent crying, regression of skills, loss of interest in pleasurable activities, and change of sleep or appetite may help in the evaluation. A family history of major mental illness and of completed suicide can further support a diagnosis of suicidal behavior. In general, however, the more intellectually disabled a person is, the more difficult it is to screen for suicidal behavior.

Causes and Risk Factors

As mentioned above, the most common cause of suicidal behavior in the general population is psychiatric illness, particularly depression. The same applies to persons with ASD. Autistic persons develop all types of psychiatric disorders across their life span, especially after puberty. Depression and other mood disorders are particularly common (Ghaziuddin et al. 2002). Both high- and low-functioning individuals with ASD may develop comorbid depression, although the signs and symptoms are relatively easy to elicit in those who are higher functioning, that is, have relatively normal intelligence and verbal skills. In addition to depression, suicidal behavior can also result from bipolar disorder which typically emerges in late adolescence and early adulthood. Suicidal behavior and completed suicide in persons with ASD have also been reported in

association with psychotic disorders, including psychotic depression with hallucinations and delusions (see Raja et al. 2011). In addition, suicidal behavior can also occur in autistic individuals with anxiety disorders. Storch et al. (2013) described suicidal behavior in 102 children and adolescents (7–16 years) with HFA and comorbid anxiety disorder. Eleven percent displayed suicidal thoughts and behaviors. An association was found between depression and a history of trauma. Other risk factors for suicidal behavior in persons with ASD include social isolation, bullying, and impulsivity. A family history of completed suicide is a known risk factor in persons committing suicide. Furthermore, there is some suggestion for shared predisposition for suicide and autism spectrum disorders (Chojnicka et al. 2013).

Management

Crisis Intervention/Inpatient Admission

The first step is to ensure the safety of the suicidal person. All suicidal threats and gestures in persons with ASD should be taken seriously. A systematic assessment should be done of the level of functioning of the individual, of the family and social circumstances, and of any precipitating or risk factors. A detailed psychiatric assessment should be performed focusing on mood symptoms. A history of previous attempts should be elicited. Depending on their level of functioning, adolescents and adults with ASD can express their suicidal thoughts and plans, if questioned in a tactful manner. Clinicians working with lower-functioning individuals with ASD should be trained to screen for suicidal behavior not only by taking a detailed history from the caregivers about changes in the mood and behavior but also by looking for any indirect evidence of suicidal intent in play and interests. If the patient is actively suicidal or psychotic, and if the family is unable to guarantee his safety, a brief admission to a specialized unit should be considered to assess the safety of the patient; clarify the reasons for the suicidal behavior; screen for psychiatric disorders; and give relief and support to the family.

Outpatient Treatment

The goal of outpatient treatment is to help the patient remain safe and gradually return to normality. By using appropriate interventions, improvement made in the hospital should be sustained. Common methods of treatment include medications and individual and family therapy. Parents should be educated to be alert to any clues of self-harm. Finally, appropriate changes should be made in the environment. For example, if the suicidal act occurs due to bullying at school, efforts should be made to involve the school authorities in the treatment program to provide support and protection to the affected person. Parents and caregivers experience severe stress when they realize, often for the first time, that their child not only has a chronic disability (autism) but also a psychiatric disorder. They may themselves need psychological support which is critical to improving the long-term outcome of the affected person.

Prevention

All suicide attempts should be taken seriously. Professional help should be sought and steps taken to minimize the risk of suicide. Caregivers should be trained to look out for clues of suicidal behavior. Those patients who have a history of suicidal behavior and those with a family history of completed or attempted suicide should be carefully observed. When care is provided at home, all necessary changes should be made to ensure safety. These include locking away guns and other weapons in the house, limiting access to pills, placing locks on the doors and windows, etc. If safety cannot be guaranteed at home or if outpatient treatment is not practical, admission to a psychiatric unit should be considered.

Conclusion

Suicidal behavior is probably underreported in persons with ASD. Reasons include a low level of suspicion on the part of clinicians and the difficulty of assessing suicidal behavior in persons

with severe deficits of communication and intelligence. Most of the research has focused on clinical samples of relatively modest size, usually of higher-functioning individuals, with limited details about their psychiatric comorbidity. It remains to be determined if the core autistic deficits raise the risk of suicide in persons with ASD or if this applies only to a subgroup of individuals within the spectrum. The relationship between the level of IQ and suicidality also needs to be clarified as also the role of family-genetic factors and life events. Despite these limitations, the findings emphasize the need for screening for suicidal behavior in all persons with autism and for undertaking systematic studies to facilitate its early detection and treatment.

References and Reading

- Barnard, J., Harvey, V., Potter, D., & Prior, A. (2001). *Ignored or ineligible? The reality for adults with autism spectrum disorders*. London: NAS.
- Cassidy, S., Bradley, P., Robinson, J., Allison, C., McHugh, M., & Baron-Cohen, S. (2014). Suicidal ideation and suicide plans or attempts in adults with Asperger's syndrome attending a specialist diagnostic clinic: A clinical cohort study. *The Lancet Psychiatry*, 1(2), 142–147.
- Chojnicka, I., Gajos, K., Strawa, K., Broda, G., Fudalej, S., Fudalej, M., Stawinski, P., Pawlak, A., Krajewski, P., Wojnar, M., et al. (2013). Possible association between suicide committed under influence of ethanol and a variant in the AUTS2 gene. *PLoS One*. <https://doi.org/10.1371/journal.pone.0057199>.
- Farrell, K. (2016). Killing the killer: Rampage and gun rights as a syndrome. In *Interdisciplinary handbook of trauma and culture* (pp. 353–363). Cham: Springer.
- Ghaziuddin, M., Ghaziuddin, N., & Greden, J. (2002). Depression in persons with autism: Implications for research and clinical care. *Journal of autism and developmental disorders*, 32(4), 299–306.
- Ghaziuddin, M. (2013). Violent behavior in autism spectrum disorder: Is it a fact, or fiction? *Current Psychiatry*, 12(10), 23–32.
- Hardan, A., & Sahl, R. (1999). Suicidal behavior in children and adolescents with developmental disorders. *Research in Developmental Disabilities*, 20, 287–296.
- Howlin, P. (2000). Outcome in adult life for more able people with autism or Asperger syndrome. *Autism*, 4(1), 63–83.
- Kato, K., Mikami, K., Akama, F., Yamada, K., Maehara, M., Kimoto, K., ..., Ichimura, A., Matsumoto, H. (2013). Clinical features of suicide attempts in adults with autism spectrum disorders. *General Hospital Psychiatry*, 35(1), 50–53.

- Mayes, S. D., Gorman, A. A., Hillwig-Garcia, J., & Syed, E. (2013). Suicide ideation and attempts in children with autism. *Research in Autism Spectrum Disorders*, 7(1), 109–119.
- Mikami, K., Inomata, S., Hayakawa, N., Ohnishi, Y., Enseki, Y., Ohya, A., Haruki, Y., Kishi, Y., Shinohara, Y., Ichimura, A., & Matsumoto, H. (2009). Frequency and clinical features of pervasive developmental disorder in adolescent suicide attempts. *General Hospital Psychiatry*, 31(2), 163–166.
- Mouridsen, S. E., Brønnum-Hansen, H., Rich, B., & Isager, T. (2008). Mortality and causes of death in autism spectrum disorders: An update. *Autism*, 12, 403.
- NAS (2001). <http://www.autism.org.uk/working-with/health/mental-health-and-asperger-syndrome.aspx>.
- Raja, J., Azzoni, A., & Frustaci, A. (2011). Autism spectrum disorders and suicidality. *Clinical Practice and Epidemiology in Mental Health*, 7, 97–105.
- Shavelle, R. M., Strauss, D. J., & Pickett, J. (2001). Causes of death in autism. *Journal of Autism and Developmental Disorders*, 31(6), 569–576.
- Storch, E. A., Sulkowski, M. L., Nadeau, J., Lewin, A. B., Arnold, E. B., Mutch, P. J., ..., Murphy, T. K. (2013). The phenomenology and clinical correlates of suicidal thoughts and behaviors in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(10), 2450–2459.
- The USA Today. Burt Bacharach opens up about daughter's suicide. Published 22 May 2013. Accessed 21 Jan 2017.
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11, 115–129.
- Wolff, S., & McGuire, R. J. (1995). Schizoid personality in girls: A follow-up study. What are the links with Asperger's syndrome? *Journal of Child Psychology and Psychiatry*, 36, 793–818.

Sulfatide Lipidosis

- [Metachromatic Leukodystrophy](#)

Sulfatidosis

- [Metachromatic Leukodystrophy](#)

Superior Temporal Sulcus

- [Superior Temporal Sulcus Region](#)

Superior Temporal Sulcus Region

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Synonyms

[Superior temporal sulcus](#)

Definition

The superior temporal sulcus is the sulcus separating the superior temporal gyrus from the middle temporal gyrus in the temporal lobe of the brain. Single-cell recordings in monkeys, and neurophysiological and neuroimaging studies in humans, reveal that cerebral cortex in and near the superior temporal sulcus region plays an important role in social perception. As defined by Allison et al. (2000), social perception “refers to initial stages in the processing of information that culminates in the accurate analysis of the dispositions and intentions of other individuals.” In man and monkey, the superior temporal sulcus regions respond vigorously to biological motion (the motion characteristic of living, animate beings) including movements of the eyes, mouth, hands, arms, and whole body.

See Also

- [Biological Motion](#)

References and Reading

- Allison, T., Puce, A., & McCarthy, G. (2000). Social perception from visual cues: Role of the STS region. *Trends in Cognitive Sciences*, 4(7), 267–278.

Support Trust

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Definition

A support trust is a type of discretionary trust used for estate planning whereby the creator of the trust (the *settlor*) transfers the assets to another (the *trustee*), who then has a duty to hold and manage the assets for the benefit of a third party (the *beneficiary*). If the trust is established by a living settlor, rather than through a will, the trust acts as a will substitute and the transferred estate avoids probate and some estate taxes.

A pure discretionary trust empowers the trustee with absolute discretion to authorize any distributions the trustee considers advisable. The trustee may decide the “time, purpose and amount of all distributions” to one or more beneficiaries (POMS SI §01120.200.B.10). Because the trustee can only be compelled to distribute money from the trust under very restricted circumstances, the assets are protected against invasion by the beneficiary, or the beneficiary’s general creditors.

In contrast, a support trust grants limited discretion upon the trustee allowing distributions solely for the “comfortable support, education, health and maintenance of the beneficiary” (Leslie and Sterk 2006). “Food, shelter, education, and medical care” are eligible categories, but the trustee may still have the discretion to determine the timing and amount of assets to assign to each category (Begley and Canellos 2010).

A common type of support trust provides care for a surviving spouse in “the manner and style to which they are accustomed” (In re Estate of Brown 1987, p. 754). In other instances, a support trust can be drafted to meet the special needs of a minor child, or a mentally incompetent individual.

Special needs trusts are established to provide supplemental funds for a beneficiary who may also be eligible to receive public benefits including SSI or other government benefits. Trust fund

distributions must only supplement public funding; the beneficiary must have no control over the assets, or ability to compel distributions. Administration of such trusts can be complex, requiring specialized knowledge of the entitlement programs available over the course of the incompetent beneficiary’s lifetime.

A support trust may be an effective estate planning tool, depending on the intent of the settlor, the objectives of the trust, and the specific needs of the beneficiary. However, the trust must be carefully drafted to consider potential conflicts between the availability of trust assets to potential creditors, as well as eligibility for public assistance programs. Additionally, problems may arise such as a trustee’s breach of duty to manage the trust in “good faith,” or “in accordance with its terms and purposes,” or “in the interests of the beneficiary” (Uniform Trust Code § 801 et. seq.). This may be demonstrated when the trustee abuses the assigned discretionary power, and the beneficiary is not competent to address the breach. Therefore, one must carefully evaluate the goals of the settlor, the competence and trustworthiness of the trustee, and the long-term needs of the beneficiary in order to determine which type of discretionary trust is the best estate planning option.

See Also

► [Discretionary Trust](#)

References and Reading

Sources

- Andersen, R. (2003). *Understanding trusts and estates*. Newark: Matthew Bender & Company.
- Averill, L. (2005). *Wills, trusts, and future interests*. St. Paul: Thomson/West.
- Begley, T., & Canellos, A. (2010). *Special needs trusts handbook (supplement)*. New York: Aspen.
- In re Estate of Brown, 528 A.2d 752, 754–755 (Vt. 1987).
- Leslie, M., & Sterk, S. (2006). *Trusts and estates*. New York: Foundation Press.
- Programs Operations Manual System: Trusts §SI 01120.200(B)(13). Retrieved March 8, 2011., from <https://secure.ssa.gov/apps10/poms.nsf/lnx/0501120200>

Restatement of Trusts (2nd) §154
 Social Security Act (SSA), §42 U.S.C. §1396p
 Uniform Trust Code §801 (2005) *et. seq.*

Supported Employment

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Definition

The term supported employment has been traced back to the early 1980s when advocates and service providers for individuals with intellectual disabilities became increasingly aware that students were not making the transition from school to the workforce. The official government definition appeared in the mid-1980s and provided the parameters of the concept that still exists today.

Supported employment means:

1. Competitive employment in an integrated setting with ongoing support services for individuals with the most severe disabilities:
 - (a) For whom competitive employment has not traditionally occurred or for whom competitive employment has been interrupted or intermittent as a result of a severe disability
 - (b) Who, because of the nature and severity of their disabilities, need intensive supported employment services from the designated state unit and extended services after transition in order to perform this work
2. Transitional employment for individuals with the most severe disabilities due to mental illness

Some of the major terms in the definition such as competitive employment and ongoing services will be examined more closely in the following

section when supported employment is described as a state's vocational rehabilitation service option known as supported employment services.

A number of models emerged over the years. The one promoted here is the individualized approach where a person with a significant disability receives one-to-one assistance with gaining or maintaining employment in his or her community, a real job for real pay, by using an array of supports provided and/or facilitated by a vocational rehabilitation professional known as a job coach or employment specialist. It has been described as a strategy for changing the mismatch between employment expectations for people with severe disabilities and the limited options in their communities (i.e., workshops or unemployment, etc.).

Historical Background

For many years, individuals with significant disabilities were served within a continuum model of vocational rehabilitation which included day centers, sheltered workshops, and transitional programs. The premise behind this approach was to teach individuals with disabilities the skills needed to become "ready to work." However, movement out of these segregated settings into employment in the community rarely took place, and eventually, the system came under criticism (Wehman 2006; Whitehead 1979). At this time, the belief that all individuals including those with significant disabilities should have the opportunity to achieve as much independence as possible including working in their communities was also beginning to flourish. In addition, a number of researchers were demonstrating that people with moderate to severe intellectual disabilities could learn complex real work tasks and furthermore that these tasks could and should be taught on the job (Bellamy et al. 1979; Gold 1972a, b, 1978; Rusch and Scutz 1979; Wehman 1981; Wehman et al. 1979), instead of in segregated settings. The approach used applied behavior analysis and systematic instruction (Test and Wood 1997). This requires breaking tasks down into stimulus response chains and using

prompting hierarchies and reinforcement to teach them (Bellamy et al. 1979).

At last, no longer were individuals with significant disabilities required to learn prerequisite skills in segregated settings prior to going to work. Instead of training an individual with a disability outside of a workplace to get ready to work (i.e., work readiness) and then locating employment, now research backed the effectiveness of a model that emphasized the effectiveness of obtaining employment first and then training the newly hired employee the skills needed on the job. In the years that followed, a number of projects were funded that demonstrated the effectiveness of training individuals with significant disabilities on the job. The results of these efforts revealed promising practices for a supported employment approach to assist individuals with developmental disabilities with employment (Vogelsberg 1990).

Again, up until this time, most individuals with significant disabilities had often been denied the option to work in their communities. Because of discrimination and oppression and unfounded beliefs regarding personal skills, capacities, and capabilities, individuals with disabilities had often been relegated to segregated environment like day centers and sheltered workshops.

Passage of legislation, such as the Developmental Disabilities Assistance and Bill of Rights Act of 1984 and Title VI, Part C of the Rehabilitation Act Amendments of 1986, in combination with systems change grants funded through Title III of the Rehabilitation Act, provided the basis for the initiation of a series of federal- and state-funded demonstration projects designed to provide opportunities and supports for individuals with severe or significant disabilities to work alongside other citizens in their communities.

Current Knowledge

Through the years, the supported employment approach has been used to assist individuals with developmental disabilities with gaining and maintaining “real work for real pay” (Wehman 2012). Although initially conceived as an

employment support service for individuals with intellectual disabilities, today supported employment has also been instrumental in assisting individuals with mental illness (Bond et al. 2001; Drake et al. 1999), traumatic brain injury (Wehman et al. 1993), autism (Howlin et al. 2005; Wehman et al. 2009), cerebral palsy, physical disabilities (Inge et al. 1998), and other disabilities (Wehman and Revell 1996).

States offer this service under the State Supported Employment Services Program which is under the Authority: 29 U.S.C. 795j–q, unless otherwise noted. What follows are the specifics about the program and the definitions from the Source: 59 FR 8331, Feb. 18, 1993, unless otherwise noted.

Under the State Supported Employment Services Program, the secretary provides grants to assist states in developing and implementing collaborative programs with appropriate entities to provide programs of supported employment services for individuals with the most severe disabilities who require supported employment services to enter or retain competitive employment (Authority: 29 U.S.C. 795j).

A state may provide services under this program to any individual if:

- (a) The individual has been determined eligible for vocational rehabilitation services in accordance with the criteria in section 102(a) (1) of the Act
- (b) The individual has been determined to be an individual with the most severe disabilities
- (c) Supported employment has been identified as the appropriate rehabilitation objective for the individual on the basis of a comprehensive assessment of rehabilitation needs, including an evaluation of rehabilitation, career, and job needs (Authority: 29 U.S.C. 795 m)

Under this program, the following activities are authorized:

- (a) Any particularized assessment that is needed to supplement the comprehensive assessment of rehabilitation needs done under 34 CFR part 361 and that is provided subsequent to

the development of the individualized written rehabilitation program. The supplementary assessment may be provided in circumstances such as the following:

1. A reassessment of the suitability of the placement is warranted.
 2. There is a change in the individual's medical condition.
- (b) Development of and placement in jobs for individuals with the most severe disabilities.
- (c) Provision of supported employment services that are needed to support individuals with the most severe disabilities in employment, such as:
1. Intensive on-the-job skills training and other training provided by skilled job trainers, coworkers, and other qualified individuals, and other services specified in Section 103(a) of the Act in order to achieve and maintain job stability
 2. Follow-up services, including regular contact with employers, trainees with the most severe disabilities, parents, guardians or other representatives of trainees, and other suitable professional and informed advisors in order to reinforce and stabilize the job placement
 3. Discrete postemployment services following transition that are unavailable from an extended services provider and that are necessary to maintain the job placement, such as job station redesign, repair and maintenance of assistive technology, and replacement of prosthetic and orthotic devices (Authority: 29 U.S.C. 795l)

The following regulations apply to the State Supported Employment Services Program:

- (a) The Education Department General Administrative Regulations (EDGAR) as follows:
1. 34 CFR part 76 (State-Administered Programs)
 2. 34 CFR part 77 (Definitions that Apply to Department Regulations)
 3. 34 CFR part 79 (Intergovernmental Review of Department of Education Programs and Activities)

4. 34 CFR part 80 (Uniform Administrative Requirements for Grants and Cooperative Agreements to State and Local Governments)
 5. 34 CFR part 81 (General Education Provisions Act-Enforcement)
 6. 34 CFR part 82 (New Restrictions on Lobbying)
 7. 34 CFR part 85 (Governmentwide Debarment and Suspension (Nonprocurement) and Governmentwide Requirements for Drug-Free Workplace (Grants))
 8. 34 CFR part 86 (Drug-Free Schools and Campuses)
- (b) The regulations in this part 363
- (c) The following regulations in 34 CFR part 361 (The State Vocational Rehabilitation Services Program): §§361.31; 361.32; 361.33; 361.34; 361.35; 361.39; 361.40; 361.41; 361.42; 361.47(a); 361.48; and 361.49

Note: Many of the regulatory provisions cross-referenced in §363.5(c) are affected by statutory changes made by the Rehabilitation Act Amendments of 1992. If these provisions conflict with statutory language, they are superseded by the statutory language. Program regulations for part 361 are being amended to implement statutory changes. When final regulations for part 361 are published, these cross-references will be corrected, if necessary

(Authority: 29 U.S.C. 795j and 711(c)):

- (a) Definitions in 34 CFR part 361. The following terms used in this part are defined in 34 CFR 369.4(b):
- Act
 - Designated state unit
 - Individual with disabilities
 - Individual with severe disabilities
 - State plan
- (b) Definitions in EDGAR. The following terms used in this part are defined in 34 CFR 77.1:
- Fiscal year
 - Nonprofit
 - Private secretary
 - State

(c) *Other definitions.* The following definitions also apply to this part:

1. *Supported employment* means:

(i) Competitive employment in an integrated setting with ongoing support services for individuals with the most severe disabilities:

(A) For whom competitive employment has not traditionally occurred or for whom competitive employment has been interrupted or intermittent as a result of a severe disability

(B) Who, because of the nature and severity of their disabilities, need intensive supported employment services from the designated State unit and extended services after transition in order to perform this work

(ii) Transitional employment for individuals with the most severe disabilities due to mental illness

2. As used in the definition of “supported employment”:

(i) *Competitive employment* means work:

(A) In the competitive labor market that is performed on a full-time or part-time basis in an integrated setting

(B) For which an individual is compensated at or above the minimum wage, but not less than the customary or usual wage paid by the employer for the same or similar work performed by individuals who are not disabled

(ii) *Integrated setting* means a setting typically found in the community in which an individual with the most severe disabilities interacts with non-disabled individuals, other than non-disabled individuals who are providing services to that individual, to the same extent that nondisabled individuals in comparable positions interact with other persons.

(iii) *Supported employment services* means ongoing support services provided by the designated state unit with funds under this part:

(A) For a period not to exceed 18 months, unless under special circumstances a longer period to achieve job stabilization has been jointly agreed to by the individual and the rehabilitation counselor and established in the individualized written rehabilitation program, before an individual with the most severe disabilities makes the transition to extended services

(B) As discrete postemployment services following transition in accordance with §363.4(c) (3)

(iv) *Extended services* means ongoing support services and other appropriate services provided by a state agency, a private nonprofit organization, employer, or any other appropriate resource, from funds other than funds received under this part, part 381, part 376, or part 380, after an individual with the most severe disabilities has made the transition from state vocational rehabilitation agency support.

(v) *Transitional employment* means a series of temporary job placements in competitive work in an integrated work setting with ongoing support services for individuals with the most severe disabilities due to mental illness. In transitional employment, the provision of ongoing support services must include continuing sequential job placements until job permanency is achieved.

(d) *Ongoing support services* means services that are:

(i) Needed to support and maintain an individual with the most severe disabilities in supported employment

- (ii) Based on a determination by the designated state unit of the individual's needs as specified in an individualized written rehabilitation program
- (iii) Furnished by the designated state unit from the time of job placement until transition to extended services, except as provided in §363.4(c) (3) and, following transition, by one or more extended services providers throughout the individual's term of employment in a particular job placement or multiple placements if those placements are being provided under a program of transitional employment. Ongoing support services must include, at a minimum, twice-monthly monitoring at the work site of each individual in supported employment to assess employment stability, unless under special circumstances; especially at the request of the individual, the individualized written rehabilitation program provides for off-site monitoring and, based upon that assessment, the coordination or provision of specific services at or away from the work site that are needed to maintain employment stability. If off-site monitoring is determined to be appropriate, it must, at a minimum, consist of two meetings with the individual and one contact with the employer each month. Ongoing support services consist of:
 - (A) Any particularized assessment needed to supplement the comprehensive assessment of rehabilitation needs
 - (B) The provision of skilled job trainers who accompany the individual for intensive job skill training at the work site
 - (C) Job development and placement
 - (D) Social skills training
 - (E) Regular observation or supervision of the individual
 - (F) Follow-up services such as regular contact with the employers, the

individuals, the parents, family members, guardians, advocates or authorized representatives of the individuals, and other suitable professional and informed advisors, in order to reinforce and stabilize the job placement

- (G) Facilitation of natural supports at the work site
- (H) Any other service identified in the scope of rehabilitation services described in 34 CFR part 361
- (I) Any service similar to the foregoing services

(Authority: 29 U.S.C. 706(18), 711(c), and 795j)

[59 FR 8331, Feb. 18, 1993, as amended at 62 FR 6363, Feb. 11, 1997]

Future Directions

Today, with the right type, level, and intensity of support, more and more individuals with intellectual disabilities are living and working in their communities (Wehman 2006; Wehman et al. 2003). Since its inception, supported employment has assisted individuals with significant disabilities with achieving positive employment outcomes (Wehman et al. 2006; Wehman et al. 1998). However, challenges remain for those who want to work. These individuals lack access to the support needed to gain and maintain work in their communities due to the slow conversion of day programs, slow expansion of program capacity among day programs that now offer integrated options (i.e., supported employment), no or limited individualized supported employment service options in such settings in favor of group models, and a lack of consumer choice and self-determination to help ensure meaningful employment outcomes. In addition, greater attention needs to be paid to employers. It is necessary to further investigate their perceptions, as well as examine more ways to motivate them to hire a person with a significant disability, such as customizing a job.

See Also

- [Employment](#)
- [Entrepreneurial Model](#)
- [School to Work Transition Process](#)
- [Sheltered Workshops](#)
- [Transition Planning](#)

References and Reading

- Bellamy, G. T., Horner, R. H., & Inman, D. P. (1979). *Vocational rehabilitation of severely retarded adults: A direct service technology*. Baltimore: University Park Press.
- Drake, R. E., Becker, D. R., Clark, R. E., & Mueser, K. T. (1999). Research on the individual placement and support model of supported employment. *Psychiatric Quarterly*, 70(4), 289–301.
- Gold, M. (1972a). Stimulus factors in skill training of the retarded on a complex assembly task: Acquisition, transfer and retention. *American Journal of Mental Deficiency*, 76, 517–526.
- Gold, M. (1972b). Factors affecting production by the mentally retarded: Base rates. *Mental Retardation*, 11, 9–11.
- Gold, M. (1978). *Try another Way: Training manual*. Austin: Marc Gold & Associates.
- Howlin, P., Alcock, J., & Burkin, C. (2005). An 8 year follow-up of a specialist supported employment service for high-ability adults with autism or Asperger syndrome. *Autism*, 9(5), 533–549.
- Inge, K., & Moon, S. (2011). Preparing students with low incidence disabilities to work in the community. In J. M. Kauffman & D. P. Hallahan (Eds.), *Handbook of special education*. London: Routledge.
- Inge, K. J., Wehman, P., Strobel, W., Powell, D., & Todd, J. (1998). Supported employment and assistive technology for persons with spinal cord injury: Three illustrations of successful work supports. *Journal of Vocational Rehabilitation*, 10, 141–152.
- Kregel, J., Wehman, P., & Banks, P. D. (1989). The effects of consumer characteristics and type of employment model on individual outcomes in supported employment. *Journal of Applied Behavior Analysis*, 22, 407–415.
- Rusch, F. R., & Scutz, R. P. (1979). Non-sheltered employment of the mentally retarded adults: Research to reality? *Journal on Contemporary Business*, 8(4), 85–89.
- Test, D. W., & Wood, W. M. (1997). Rocket science 101: What supported employment specialists need to know about systematic instruction. *Journal of Vocational Rehabilitation*, 9, 109–120.
- Vogelsberg, R. T. (1986). Competitive employment in Vermont. In F. R. Rusch (Ed.), *Competitive employment: Issues and strategies* (pp. 35–50). Baltimore: Paul H. Brookes.
- Vogelsberg, R. T. (1990). Supported employment in Pennsylvania. In F. R. Rusch (Ed.), *Supported employment: Models, methods and issues* (pp. 45–64). Sycamore: Sycamore.
- Wehman, P. (1981). *Competitive employment: New horizons for severely disabled individuals*. Baltimore: Paul H. Brookes.
- Wehman, P. (2006). *Life beyond the classroom: Transition strategies for young people with disabilities*. Baltimore: Paul H. Brookes.
- Wehman, P. (2012). *Life beyond the classroom: Transition strategies for young people with disabilities*. Baltimore: Paul H. Brookes Publishing Co.
- Wehman, P., & Kregel, J. (1985). A supported work approach to competitive employment for individuals with moderate and severe handicaps. *The Journal of the Association for Persons with Severe Handicaps*, 10, 3–11.
- Wehman, P., & Revell, W. G. (1996). Supported employment from 1986 to 1993: A national program that works. *Focus on Autism and Other Developmental Disabilities*, 11(4), 235.
- Wehman, P., Hill, J. W., & Keohler, F. (1979). Placement of developmentally disabled individuals into competitive employment: Three case studies. *Education and Training of the Mentally Retarded*, 14, 269–276.
- Wehman, P., Kregel, J., Sherron, P., Nguyen, S., Kreutzer, J., Fry, R., et al. (1993). Critical factors associated with the successful supported employment placement of patients with severe traumatic brain injury. *Brain Injury*, 7(1), 31–44.
- Wehman, P., Revell, G., & Kregel, J. (1998). Supported employment: A decade of rapid growth and impact. *American Rehabilitation*, 24(1), 31–43.
- Wehman, P., Revell, G., & Brooke, V. (2003). Competitive employment: Has it become the first choice yet? *Journal of Disability Policy Studies*, 14(3), 163–173.
- Wehman, P., Smith, M., & Schall, C. (2009a). *Transition from school to adulthood for youth and young adults with autism: Growing Up in the real world*. Baltimore: Paul H. Brookes.
- Wehman, P., Smith, M., & Schall, C. (2009b). *Transition from school to adulthood for youth and young adults with autism: Growing Up in the real world*. Baltimore: Paul H. Brookes.
- Whitehead, C. W. (1979). Sheltered workshops in the decade ahead: Work, wages or welfare. In G. T. Bellamy, G. O'Conner, & O. C. Karan (Eds.), *Vocational rehabilitation of severely handicapped persons*.

Supported Employment Group

- [Mobile Work Crew Model](#)

Supported Work

- [Entrepreneurial Model](#)

Supporting

- [Reinforcement](#)

Sustained Treatment Benefits

- [Maintenance of Treatment Effects](#)

Sweden and Autism

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Historical Background

Knowledge about autism in Sweden was very limited until the mid-late 1970s. The predominant view, based on no evidence, was that autism was caused by poor parenting. This view has been difficult to root out and has led to enormous suffering and unwarranted interventions well into the twenty-first century.

Anna-Lisa Annell, professor of child and adolescent psychiatry in Uppsala, had shown interest already in the 1950s in what was at the time referred to as childhood psychosis and its possible links with brain damage, as documented in her thesis on pertussis, possible anoxia attacks, and behavior problems in children.

In the 1960s child psychiatrist Karl Grunewald, later in life head of the Swedish National Board of Health and Welfare, started a campaign/lobbying with a view to improving the situation for young

and older people with intellectual disability. For a long time he was the driving force in the process of shutting down the big institutions for the “mentally retarded” (of whom many had undiagnosed autism). In 1968, the Swedish parliament passed the first law specifically stating the need for support and schooling for individuals with intellectual disability. This law (Omsorgslagen) was updated in 1986, and autism (“childhood psychosis”) was mentioned for the first time in Swedish legislation as a diagnosis that would *translate* support and schooling for affected individuals. This was followed by the 1993 Act concerning Support and Services for Persons with Certain Functional Impairments (LSS) in which *ten different rights* (including the right to a personal assistant) were specified and guaranteed for individuals with intellectual disability, autism and autistic-like conditions, traumatic brain injury with cognitive impairment, and other individuals with major physical or mental impairments in need of major support measures (Table 1). The aims and overall objectives of this development were to guarantee that people with severe disabilities would have as high a quality of life as possible, and that services and support due to lifelong disabilities would be free of charge and not cause extra costs to the individual. The Swedish Education Act updated in 2010 also specifies the right for all individuals – including those with disabilities – to get the best possible educational support so that they might achieve their full potential.

In parallel with Grunewald, Bengt Hagberg, Olle Hansson, and Karl-Gustaf Stukat, professors of Pediatrics and of Special Education in Gothenburg started a large population study of so-called Minimal Brain Dysfunction (MBD). The senior author of this chapter was one of the members of the clinical research team. He was also in charge of the medical services to a group home for children with “psychosis,” and later for all medical services to people with intellectual disability in and around Gothenburg. In planning the MBD study, he had read up about autism and childhood schizophrenia. He read Lorna Wing’s book *Autistic Children* from 1964 and also Hans Asperger’s 1944 paper on autistic psychopathy (later referred to as Asperger syndrome). The concepts of autism

(Mildred Creak’s “version,” Lorna Wing’s triad of social impairments and autistic psychopathy) were used in the MBD study and laid the groundwork for the Gothenburg autism studies, with the first publication on “Maternal age in infantile autism” appearing in 1980 and on “comorbidity” in 1983. From then on, the Gothenburg autism team led the development of research (and also to an extent clinical services) into autism and related disorders in Sweden. In the early 1980s, the only other center in which research and services for autism were developing was in Umeå, where professor Michael Bohman and his team did groundbreaking work around “childhood psychosis.” Psychologist Lena Andersson was one of the first to recognize the high rate of autism in immigrants. She collaborated with the Gothenburg autism group in several different projects (including the Nordic Twin Study of Autism and The Early Diagnosis “Rebecka” Project in the 1980s). For many years she was the secretary of the Autism Society and a key person (together with the chairmen/women of the society) in the spreading of autism knowledge across the country.

The National Autism Society – variously labeled Association for Psychotic Children, Association for Autism and since 2010 Autism and Asperger Syndrome Association – since its inception in 1973 (first chair Gunilla Dahlin, mother of a girl who later was diagnosed with Rett syndrome), has played an increasingly important role in assuring the development of services for individuals with autism in Sweden. In the early 1980s the association had 1000 members, many of whom were professionals working together with a few “powerhouse” parents of affected children. Twenty-five years later there are now 16,000 members, and the association is a strong “agency” with considerable clout vis-a-vis legislators, politicians, and administrators. The second author of this chapter was the chairman for 16 years (up until 2015), during which many of the very positive changes as regards assessment routines and interventions occurred. The society has been instrumental in delivering education (including e-learning) for parents, affected individuals, and professionals.

Legal Issues, Mandates for Service

The LSS-act from 1994 (see above) which guarantees a large number of different rights for people with a diagnosis of autism and autistic-like conditions (see Table 1) has gradually come to be interpreted much more narrowly than was intended by the legislators so that even with an unequivocal diagnosis of autism, individuals affected (or their parents) can no longer be sure that they will get what they need. It is assumed that one of the reasons for the more restrictive interpretation of the “circle of people” that is considered to have lawful right to the services detailed in the LSS-act is the enormous increase in the number of autism diagnoses made in some parts of Sweden (notably Stockholm) over the past 15 years. There is almost definitely “overdiagnosis” of autism in some areas of the country (Arvidsson et al. 2017). When the act was passed, it was assumed that the rate of autism was under half a percent. In Stockholm in 2011 almost 2.5% of 13–17-year-olds had been registered with a diagnosis of autism (Idring et al. 2015). Fewer and fewer symptoms seem to be needed for a clinical diagnosis (Arvidsson et al. 2017). This situation has led to skepticism on the part of those who assign/agree to services under the LSS-act, who argue that mild cases should not be able to avail themselves of equally comprehensive services (including personal assistants) as should those with very severe problems. At the

Sweden and Autism, Table 1 Act concerning Support and Services to Persons with Certain Functional Impairments. The ten rights

1	Advice and other personal support
2	Personal assistance
3	Companion service
4	Contact person
5	Relief service
6	Short-term stays away from home
7	Short-term care for school children over 12
8	Living in family homes or housing with special services for children and young people
9	Housing with special services for adults or other specially adapted housing for adults
10	Daily activities

same time, even though, by and large, awareness about autism has increased greatly over the past two decades, in-depth knowledge, particularly in schools and social services, has remained scarce.

Overview of Current Treatments and Centers

There are now relatively well-functioning child and adolescent autism assessment and diagnostic teams in many Swedish counties (although certainly not in all), usually within child and adolescent mental health services. In many ways, this has contributed to the development of better services in terms of educational and behavioral interventions, particularly for young children with autism. However, it has also, in some places, led to an overfocus on autism as the only problem in children with the disorder. As has been demonstrated by the Gothenburg research group since the beginning of the 1980s, clinically relevant autism usually does not come “alone.” Instead, comorbidities (e.g., intellectual disability, language disorder, motor coordination problems, ADHD, tics, hearing and visual deficits, epilepsy, etc.) are the rule. These associated problems are often highly treatable, but in recent years, the diagnosis of autism has overshadowed them to the extent that they are not always properly diagnosed or treated. In other places, autism-specific knowledge has been watered down to the extent that there is little or no understanding of the special needs for individuals with a diagnosis of autism. By and large though, the situation for young children with autism in Sweden is much better now than 40 years ago, even though the separation of assessment and intervention services remains a problem with child psychiatry usually providing the assessment and habilitation services (without being involved in assessment) the intervention. Teenagers and adults with autism are often misunderstood and misdiagnosed, and “autism-specific” competence in schools and social services is rare. In adult psychiatry there is growing interest in and awareness of autism, but, again, in-depth knowledge is lacking in many places, leading to missed diagnoses, and,

sometimes, unwarranted treatments (such as with atypical neuroleptics for what is considered “psychosis” or “personality disorder” rather than social communication deficits and behavioral peculiarities that are typical of autism spectrum disorders). In Sweden there has, rightly, been a lot of emphasis on early diagnosis and early intervention in autism. The need for very long-term follow-up (indeed sometimes life-long) has often been ignored, and a focus on transition and adulthood is much talked about but not “breaking through.” Unfortunately, services for adults with autism and related disorders are still nowhere near as well developed as are those for younger people.

Overview of Research Directions

The Gothenburg group has been leading the international field in epidemiological autism research for many years. Together with Lorna Wing, who gradually became a close collaborator, the group was first to draw attention to the much higher than previously reported rate of autism, claiming already in the 1980s that the “true” prevalence of the disorder was around 1% of the general population. The group has since shown that the rate of the autism phenotype in the population appears to be very stable over the years (and remaining around 1%), but that the clinical diagnosis of autism is made more and more often, sometimes in cases with neurodevelopmental problems that do not meet the diagnostic criteria for autism. The Swedish epidemiologically based studies of autism (and Asperger syndrome) are unique in an international perspective. Very little in-depth study of epidemiological cohorts is published by other groups.

Autism research in Sweden is holistic and ranges from groundbreaking genetic research (in collaboration with a French group led by professor Thomas Bourgeron), epigenetics (including pioneering work on vitamin D deficiency in autism in Somali immigrants and in pregnant mothers of children who later get a diagnosis of autism), clinical presentation (which has led to the concept of Autism Plus, i.e., that clinically impairing autism is usually associated with

significant comorbidity), early symptoms (including work that suggests that motor-perceptual problems may be the first to signal autism, and there might be a slightly different phenotype in girls), longitudinal population-based studies from childhood into late middle age of low- and high-functioning individuals, twin studies, various intervention RCTs, and to neurochemical, neurophysiological, imaging, and neuropsychological studies. The ultimate goal is to get at “the bigger picture in autism” at the same time as understanding the molecular basis and brain functioning.

Social Policy and Training

Many advances have been achieved in the past 40 years. On-going training of providers (doctors, psychologists, nurses, teachers, and others) remains a top priority for the autism center in Gothenburg and in the past several years in other places in Sweden, including Stockholm. Efforts have been made for the last 30 years, particularly by the National Autism Association, to develop and implement curricula to enhance effective teaching methods and alternative communication in schools, classrooms, and everyday-life situations, often inspired by the TEACCH “philosophy.” The Autism Society has also been promoting the “low-arousal” approach advocated by Bo Hejlskov Elvén and Ross Greene. In the past 15 years, ABA-approaches and early intensive programs (along the lines of Denver Early Start program) have come to the forefront and been strongly advocated by a small group of influential specialists. However, recent Swedish research has not supported the superiority of intensive versus nonintensive interventions in naturalistic settings, and there is now, perhaps, a return to a more eclectic and individually designed approach addressing all the specific needs of each child treatments and other interventions, including education and social support, in each child.

Establishing the evidence base of interventions should be a top priority in the next several years. Also, a stronger focus needs to be directed at adults and old people with autism, who still do not get the attention that they are in need of in our country.

References and Reading

- Arvidsson, O., Lichtenstein, P., Gillberg, C., Lundström S (2017) *Fewer symptoms required for clinical diagnosis of autism: A population based study*. Submitted
- Ildring, S., Lundberg, M., Sturm, H., Dalman, C., Gumpert, C., Rai, D., Lee, B. K., & Magnusson, C. (2015). Changes in prevalence of autism spectrum disorders in 2001–2011: Findings from the Stockholm youth cohort. *J Autism Dev Disord*, 45(6), 1766–1773.
- The Swedish Act concerning Support and Service for Persons with Certain Functional Impairments (LSS) 1993:387

Symbol

► [Icon](#)

Symbol Use

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Definition

The term “symbol use” is often used to describe the deficits in communication that characterize individuals with autism spectrum disorders (ASD; Marans et al. 2005). The capacity for symbol use refers to an individual’s ability to use one thing to represent another. Symbolic behavior is evident in verbal as well as nonverbal communication through the use of

words as labels, and the use of gestures. An additional domain of symbol use relevant to ASD is that of symbolic play, in which non-present elements are represented by objects, gestures, or language during the play (Rogers et al. 2005).

A deficit in symbol use is a core social communication deficit that distinguishes young children with ASD (Wetherby and Woods 2008). At a prelinguistic level, this is seen in a deficit of symbolic behavior. This includes gestures, pretend or imaginative play, and the ability to imitate others (Paul and Sutherland 2005). In higher-functioning individuals, the deficit in the capacity for symbol use is seen in difficulties with using language in a flexible manner, such as responding to language that includes words with multiple meanings, non-literal language, and irony (Marans et al. 2005). An additional area of social communication that might be affected includes symbolic conventions, such as rules of politeness.

See Also

- Symbolic Behavior
- Symbolic Play

References and Reading

- Marans, W. D., Rubin, E., & Laurent, A. (2005). Addressing social communication skills in individuals with high-functioning autism and Asperger syndrome: Critical priorities in educational programming. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.
- Paul, R., & Sutherland, D. (2005). Enhancing early language in children with autism spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.
- Rogers, S. J., Cook, I., & Meryl, A. (2005). Imitation and play in autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed.). Hoboken: Wiley.
- Wetherby, A., & Woods, J. (2008). Developmental approaches to treatment. In K. Chawarska, A. Klin, & F. R. Volkmar (Eds.), *Autism spectrum disorders in infants and toddlers*. New York: The Guilford Press.

Symbolic Behavior

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Definition

Symbolic behavior is an encoding process in which items or actions are used to represent something different (Huttenlocher and Higgins 1978). Engaging in symbolic behavior elicits a network of experiences that is a result of the history of actions that are associated with that particular symbol (Volkmar et al. 2005). The development of a symbol is related to adaptation, in which somatosensory experiences and perceptually guided actions are integrated, and turns the symbol into a substitute for the action (Volkmar et al. 2005). Imitation is also believed to be a prerequisite for the development of symbolic behavior (Volkmar et al.). Language is the most important type of symbolic behavior; it is also evident in storytelling, play and games, and gestures (Greenberg 1971; Jones 1996).

Symbolic behavior functions to create and maintain a common meaning that is shared socially. It validates norms for behaviors and allows individuals to engage in behaviors that connect them together (Harris and Nelson 2007; Jones 1996). Symbolic behavior can also create and maintain social interactions. The use of symbols is reliant upon the expected response from others (Volkmar et al. 2005). It is the expectation of shared meaning that both initiates and perpetuates an interaction when one seeks to engage with others and interpret the symbol to represent the same meaning.

Early symbolic behavior can be seen in pretend play and imitation, where an object or person is

used to represent another (Volkmar et al. 2005). It is concurrent with the development of language, and “marks the existence of a vivid and active internal world for the young child” (Berg and Sternberg 1985, p. 68). Symbolic play usually develops around 2–3 years, although the use of symbolic gestures as a function of communication has been observed in children as young as 11 months of age (Acredolo and Goodwyn 1988). A child’s current level of symbolic behavior can be determined by examining the highest level of play that is spontaneously exhibited by the child (Paul 2007), and is closely related to the child’s understanding of a symbolic system, such as language (Christie 1991).

Historical Background

Children with ASD have difficulties demonstrating symbolic behavior (Baron-Cohen 1987; Charman and Baron-Cohen 1997; Libby et al. 1998; Stahmer 1995; Wing et al. 1977). The diagnostic criteria for autism include a deficit in symbolic play, particularly in pretend and imitative play, observed before the age of 3 years (American Psychiatric Association [APA] 2000). Given the importance of symbolic behavior in the development of linguistic, social, and cognitive skills, several studies have been conducted to both examine the symbolic play skills of children with ASD and develop interventions to improve those skills.

In early studies of symbolic play among children with ASD (Hammes and Langdell 1981; Wing et al. 1977), they were found, overall, to have a lack of spontaneous symbolic play. Those children with ASD who did exhibit symbolic play also had more repetitive and stereotyped play (Wing et al.). Although children with ASD were able to imitate video-modeled play acts similarly to children with intellectual disability, they had significantly fewer spontaneous symbolic acts (Hammes and Langdell 1981). A common theme in the symbolic play literature appears to be that scaffolding (prompting) improves the ability of children with ASD to engage in symbolic play; however, this type of play is still lacking in their

spontaneous play (Blanc et al. 2000, 2005; Lewis and Boucher 1988; Sigman and Ungerer 1984; Volkmar et al. 2005).

There are several areas of symbolic play which can be affected for children with ASD. In general, children with ASD do not spontaneously exhibit symbolic play as frequently as other children (Blanc et al. 2005; Hammes and Langdell 1981; Wing et al. 1977). There is also a lower complexity and variety in the type of symbolic sequences used by children with ASD (Blanc et al. 2005; Wing et al. 1977). It has been hypothesized that these deficits may be a result of dysregulation (Blanc et al. 2005), difficulty with meta-representation (Baron-Cohen 1987; Volkmar et al. 2005), or a lack of generativity of ideas (Lewis and Boucher 1988). Limited symbolic play abilities may also be a result of a lack of social opportunities or more general symbolic thought and language difficulties (Volkmar et al. 2005).

Current Knowledge

Children with ASD have been observed to demonstrate less symbolic behavior compared to their typical peers or peers with other disabilities (Baron-Cohen 1987; Charman and Baron-Cohen 1997; Libby et al. 1998; Stahmer 1995; Volkmar et al. 2005; Wing et al. 1977). Research suggests that children with ASD use less complex nonverbal behavior to communicate, and they demonstrate a disordered pattern that suggests the misuse and misunderstanding of nonverbal communication (APA 2000). Children with ASD are able to imitate symbolic behavior but seem to have more difficulty producing their own symbolic actions (Blanc et al. 2005; Hammes and Langdell 1981; Wing et al. 1977). Additionally, deficits in functional and pretend play have been observed, even after they have been modeled by others (Chawarska et al. 2008).

Children with ASD also demonstrate a qualitatively different symbolic play than typically developing children or those with other disabilities, with more object substitutions, fewer attributions of false properties, and no references to

absent objects (Blanc et al. 2000, 2005; Lewis and Boucher 1988; Sigman and Ungerer 1984; Volkmar et al. 2005). Significant differences in the types of symbolic behaviors used to communicate (i.e., gestures, eye gaze, requesting, and commenting) have also been observed (Stone et al. 1997). It has also been found that children with ASD use unconventional gestures and do not use gestures spontaneously (Chawarska et al. 2008). Overall, children with ASD seem to communicate for different reasons, using different types of symbolic and nonverbal behaviors to communicate, and these differences can be detected as early as 2–3 years of age (Stone et al. 1997).

Children with ASD who are able to demonstrate symbolic play behaviors have significantly higher verbal and nonverbal IQs compared to those who do not (Baron-Cohen 1987). However, even when children with ASD are able to demonstrate symbolic play skills before starting school, they still show poorer play abilities relative to typical peers (McDonough et al. 1997). Various studies have suggested that symbolic play improves after modeling and that children with ASD are able to engage in symbolic play when the symbol is suggested by another person (Blanc et al. 2000, 2005; Lewis and Boucher 1988; Sigman and Ungerer 1984; Volkmar et al. 2005). However, during spontaneous play conditions, children with ASD demonstrate fewer different play behaviors, as well as less functional and sensorimotor play (Volkmar et al.).

Early studies suggested that a relationship existed between language development and symbolic play skills in children with autism (Sigman and Ungerer 1984); however, more recent studies have reported the absence of a relationship between functional and symbolic play and verbal language (Blanc et al. 2005; Charman and Baron-Cohen 1997; Volkmar et al. 2005). Linguistic abilities can remain intact, while symbolic behaviors continue to be a challenge for children with ASD (Blanc et al. 2005). There may be distinct communication difficulties associated with autism (e.g., pragmatic joint attention or unusual lexical patterns) that specifically underlie problems with symbolic behavior (Miller 2007).

Symbolic behavior is commonly measured using the Communication and Symbolic Behavior Scales (CSBS), developed by Wetherby and Prizant (1998). The CSBS consists of a caregiver questionnaire and direct assessment and examines the communicative, social, and symbolic functioning of children up to 6 years old with communication skills between 6 and 24 months old. The CSBS is used to identify at-risk children, guide interventions, and establish profiles that can be monitored over time (Volkmar and Marans 1999).

In addition, the CSBS Developmental Profile (CSBS-DP) is another standardized tool developed by Wetherby and Prizant (Wetherby et al. 2002) to evaluate the communication and symbolic behavior of children from 6 to 24 months old. Based on the CSBS, the CSBS-DP consists of a behavior sample, which is a face-to-face evaluation of a child's interaction with a parent and clinician. Three categories of skills are assessed: social, speech, and symbolic scales. The CSBS-DP has been found to be valuable for predicting later language based on the child's social-communication skills, and positive predictive values for communication delays above 70% for children of ages 9–24 months (Barton et al. 2010; Wetherby 2006). A copy of the caregiver questionnaire used for the CSBS-DP can be found online: <http://www.brookespublishing.com/tools/csbsdp/caregiverquestionnaire.pdf>.

Another method used to evaluate symbolic play in nonverbal children is the Carpenter's Play Scale (Carpenter 1987), which infers a child's symbolic play during four 8-min play scenes with a parent. Using appropriate prompt materials (e.g., tea cups), the actions of the nonverbal child are interpreted to evaluate the presence of symbolic play with those materials (e.g., pretending to drink from the cup). Parents are asked to follow the child's lead throughout the sessions. Similarly, McCune (1995) developed a hierarchical scale on which to rate spontaneous symbolic play behaviors during interactive sessions with a familiar adult using standardized items (e.g., bed and covers, toy food, cars). Both methods use interactional observations to determine the presence of symbolic play behaviors, and have relatively strong validity and reliability.

Another assessment of play skills in children with autism has been adapted from those used with typically developing children. The Structured Play Assessment (SPA; Ungerer and Sigman 1981) is a developmental play assessment that uses several standardized toy sets that are placed in front of the child sequentially to examine how the child plays with the toys. Children are presented with each toy set for approximately 3–4 min, for a total of a 15–20-min videotaped observation. Several features of play are coded, such as the number of different play acts and the diversity of play, which can ultimately provide a developmental profile of the child's play activity level (Kasari et al. 2006).

Future Directions

Given the difficulties of children with ASD to master symbolic play skills, it seems critical to continue focusing both assessment and intervention studies in this area. It remains unclear exactly why children with ASD struggle with symbolic behavior skills, although several hypotheses have been proposed. While some studies suggest that children with ASD lack the ability to manipulate symbols purposefully and meaningfully (Hammes and Langdell 1981), other studies propose dysregulation as a more fundamental challenge underlying symbolic play skills (Blanc et al. 2005). Especially given the mixed nature of the literature on the relationship between communication and language with symbolic play skills, additional studies are needed to better evaluate this area. More information is clearly needed to better identify the multiple underlying issues relevant to symbolic behavior that are specifically affecting children with ASD.

In addition, to identify the underlying causes for difficulties in symbolic play, there are several associated areas that relate to symbolic behavior, which remain to be explored by the current literature. For example, the relationship between symbolic behavior and social interest, social orientation, imitation, developmental skills, etc., among children with ASD has not been fully examined (Volkmar et al. 2005). Both child-

specific (e.g., neuropsychological) and environment-specific (e.g., exposure to symbolic play practice) areas should be explored (Volkmar et al.).

Better identification of symbolic behavior difficulties can also aid in the early diagnosis of children with ASD. Given that difficulties in this area are a core feature of ASD (APA 2000), there is good reason to use this skill as an early marker of the disorder (Blanc et al. 2005). There is also a great need for early intervention strategies to address this core deficit. Recent studies targeting symbolic play among children with ASD have been successful in improving this skill (Kasari et al. 2006; Stahmer 1995; Thorp et al. 1995); however, more studies are needed to replicate and expand these findings. Naturalistic behavioral procedures, such as Pivotal Response Training, appear to be promising approaches to teaching symbolic play skills to children with ASD (Kasari et al. 2006; Stahmer 1995; Thorp et al. 1995). Given that there are now models of interventions to follow in teaching symbolic play skills, those methods should be examined across populations of children with ASD to identify for whom the methods work best.

See Also

- Pretend Play
- Symbolic Thought

References and Reading

- Acredolo, L., & Goodwyn, S. (1988). Symbolic gesturing in normal infants. *Child Development*, 59, 450–466.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author. Text rev.
- Baron-Cohen, S. (1987). Autism and symbolic play. *British Journal of Developmental Psychology*, 5, 139–148.
- Barton, M., Carr, K., Herlihy, L., Knoch, K., & Fein, D. (2010). The identification of autism spectrum disorders in early childhood: A case report. In J. E. Morgan, I. S. Baron, & J. H. Ricker (Eds.), *Casebook of clinical neuropsychology* (pp. 28–37). New York: Oxford University Press.
- Berg, C. A., & Sternberg, R. J. (1985). Metaphoric competence in cognitive and language development. In

- H. W. Reeses (Ed.), *Advances in child development and behavior* (Vol. 19, pp. 49–82). Orlando: Academic Press.
- Blanc, R., Tourrette, C., Deletang, N., Roux, S., Barthelemy, C., & Adrien, J. L. (2000). Regulation of symbolic activity and development of communication in children with autistic disorder. *European Review of Applied Psychology*, 50, 369–381.
- Blanc, R., Adrien, J.-L., Roux, S., & Barthelemy, C. (2005). Dysregulation of pretend play and communication development in children with autism. *Autism*, 9(3), 229–245.
- Carpenter, R. (1987). Play scale. In L. Olswang, C. Stoel-Gammon, T. Coggins, & R. Carpenter (Eds.), *Assessing prelinguistic and early linguistic behaviors in developmentally young children* (pp. 44–77). Seattle: University of Washington Press.
- Charman, T., & Baron-Cohen, S. (1997). Brief report: Prompted pretend play in autism. *Journal of Autism and Developmental Disorders*, 27(3), 325–332.
- Chawarska, K., Klin, A., & Volkmar, F. R. (Eds.). (2008). *Autism spectrum disorders in infants and toddlers: Diagnosis, assessment, and treatment*. New York: Guilford Press.
- Christie, J. F. (Ed.). (1991). *Play and early literacy development*. Albany: State University of New York Press.
- Greenberg, J. H. (1971). *Language, culture, and communication*. Stanford: Stanford University Press.
- Hammes, J. G. W., & Langdell, T. (1981). Precursors of symbol formation and childhood autism. *Journal of Autism and Developmental Disorders*, 11, 331–346.
- Harris, T. E., & Nelson, M. D. (2007). Symbolic behavior. In *Applied organizational communication: Theory and practice in a global environment* (3rd ed., pp. 221–252). New York: Taylor and Francis.
- Huttenlocher, J., & Higgins, E. T. (1978). Issues in the study of symbolic development. In W. A. Collins (Ed.), *Minnesota symposia on child psychology* (Vol. 11, pp. 98–138). Hillsdale: Erlbaum.
- Jones, M. O. (1996). *Studying organizational symbolism: What, how, why?* Thousand Oaks: Sage.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in young children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry*, 47(6), 611–620.
- Lewis, V., & Boucher, J. (1988). Spontaneous, instructed and elicited play in relatively able autistic children. *British Journal of Developmental Psychology*, 6, 325–339.
- Libby, S., Powell, S., Messer, D., & Jordan, R. (1998). Spontaneous play in children with autism: A reappraisal. *Journal of Autism and Developmental Disorders*, 28(6), 487–497.
- McCune, L. (1995). A normative study of representational play at the transition to language. *Developmental Psychology*, 31(2), 206.
- McDonough, L., Stahmer, A., Schreibman, L., & Thompson, S. (1997). Deficits, delays, and distractions: An evaluation of symbolic play and memory in children with autism. *Development and Psychopathology*, 9, 17–41.
- Miller, D. C. (2007). The school neuropsychological model applied to a common developmental disorder: Autism. In *Essentials of school neuropsychological assessment* (pp. 383–393). Hoboken: Wiley.
- Paul, R. (2007). Assessment and intervention for developing language. In *Language disorders from infancy through adolescence: Assessment and intervention*. St. Louis: Elsevier.
- Sigman, M., & Ungerer, J. A. (1984). Cognitive and language skills in autistic, mentally retarded, and normal children. *Developmental Psychology*, 20, 293–302.
- Stahmer, A. C. (1995). Teaching symbolic play skills to children with autism: Pivotal response training. *Journal of Autism and Developmental Disorders*, 25(2), 123–141.
- Stone, W. L., Ousley, O. Y., Yoder, P. J., Hogan, K. L., & Hepburn, S. L. (1997). Nonverbal communication in two- and three-year-old children with autism. *Journal of Autism and Developmental Disorders*, 27, 677–696.
- Thorp, D. M., Stahmer, A. C., & Schreibman, L. (1995). Effects of sociodramatic play training on children with autism. *Journal of Autism and Developmental Disorders*, 25(3), 265–282.
- Ungerer, J. A., & Sigman, M. (1981). Symbolic play and language comprehension in autistic children. *Journal of the American Academy of Child Psychiatry*, 20(2), 318–337.
- Volkmar, F. R., & Marans, W. D. (1999). Measures for assessing pervasive developmental and communication disorders. In D. Shaffer, C. P. Lucas, & J. E. Richters (Eds.), *Diagnostic assessment in child and adolescent psychopathology* (pp. 167–206). New York: Guilford Press.
- Volkmar, F. R., Paul, R., Klin, A., & Cohen, D. (Eds.). (2005). *Handbook of autism and pervasive developmental disorders: Assessment, interventions, and policy* (3rd ed.). Hoboken: Wiley.
- Wetherby, A. M. (2006). Understanding and measuring social communication in children with autism spectrum disorders. In T. Charman & W. Stone (Eds.), *Social and communication development in autism spectrum disorders: Early identification, diagnosis, and intervention* (pp. 3–34). New York: Guilford Press.
- Wetherby, A. M., & Prizant, B. (1998). Communicative, social/affective, and symbolic profiles of young children with autism and pervasive developmental disorders. *American Journal of Speech-Language Pathology*, 7, 79–91.
- Wetherby, A. M., Allen, L., Cleary, J., Kublin, K., & Goldstein, H. (2002). Validity and reliability of the communication and symbolic behavior scales developmental profile with very young children. *Journal of Speech, Language, and Hearing Research*, 45(6), 1202–1218.
- Wing, L., Gould, J., Yeates, S., & Brierley, L. (1977). Symbolic play in severely mentally retarded and autistic children. *Journal of Child Psychology and Psychiatry*, 18, 167–178.

Symbolic Play

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Definition

Play emerges in a specific developmental sequence (Belsky and Most 1981). First, children handle toys in manipulative ways by touching, mouthing, and smelling. Functional play or appropriate play with objects develops at approximately 14 months of age in typically developing children. At about 24 months of age, symbolic play emerges. Symbolic play is behavior that is simulative or nonliteral (Fein 1981) and involves acting as if something is the case when in reality it is not (Leslie 1987). Definitions of symbolic play used in the majority of research with children with autism typically involve the following three symbolic forms of pretense (Leslie 1987): object substitution, in which one object is used to represent another (e.g., using a hairbrush as a telephone); attribution of absent/false properties (e.g., pretending that a stove top is hot when it is not); and imaginary objects present (e.g., pouring tea from an imaginary tea pot). A number of terms have been used interchangeably with symbolic play, including “pretend, fantasy, imagination, and nonrepresentational play” (Jarrold et al. 1993). These terms, however, do not have consistent definitions and may include actions with objects, such as pretending to feed one’s self or a doll with a spoon, that do not show true evidence of symbol use since the child may actually perceive the toys as smaller versions of real objects (Baron-Cohen 1987).

Historical Background

Children with autism exhibit significant deficits in the development of symbolic play (Jarrold et al. 1993). Indeed, a “lack of varied, spontaneous, make-believe play” is a diagnostic criterion for

autism in the DSM-IV (American Psychiatric Association 2000). The view that children with autism have impaired symbolic play was first noted by Kanner in 1943 and has subsequently been supported by a number of studies that have compared the ability of children with autism to engage in symbolic play with other groups of children (e.g., typically developing, language delayed, intellectually disabled), matched in a variety of ways (e.g., chronological age, mental age, language age). Symbolic play has been traditionally measured by observing the child interact with a standard set of toys and recording the number of play actions produced of various types (e.g., functional, symbolic). A commonly used play measure is the Lowe and Costello Symbolic Play Test (Lowe and Costello 1976). Children are presented with four sets of toys, and their spontaneous engagement with the toy is observed. If the child does not spontaneously engage with the toy set, then they are provided with a prompt such as “what can you do with these” (Stanley and Konstantareas 2007). The child’s score from this test is used to determine their play age.

These studies have typically demonstrated that children with autism are significantly less likely to spontaneously produce symbolic play acts than children without autism functioning at a similar developmental level (see Jarrold et al. 1993 for review). The ability to engage in symbolic play appears to be closely related to both nonverbal and verbal skills. For example, in Baron-Cohen’s (1987) study, children with autism who did engage in symbolic play had significantly higher nonverbal mental ages than those who did not. This finding supports a relationship between symbolic play and cognitive development. Further, Stanley and Konstantareas (2007) found that children with autism who exhibited greater cognitive impairment showed lower symbolic play skills, and higher expressive language abilities were associated with better symbolic play skills. However, symbolic play was not significantly related to social development.

Interestingly, although deficits in the spontaneous use of symbolic play have been consistently observed across studies, there is evidence that children with autism can produce symbolic play

acts in a structured, elicited setting. For example, Charman and Baron-Cohen (1993) found that children with autism were able to produce a similar number of object substitutions in response to a prompt as children with mental retardation matched on verbal mental age. However, Sigman and Ungerer (1984) found that although children with autism were able to produce some symbolic acts in response to a prompt, they still produced fewer acts than the control group. In general, studies have found that deficits in the spontaneous production of symbolic acts during play are more pronounced than the ability to produce symbolic acts in a structured situation; however, deficits in both of these abilities appear to be present, particularly in younger children with autism.

A number of theoretical accounts of autism have attempted to explain the deficit in symbolic play (see Jarrold et al. 1993 for review). The *metarepresentational* or *theory of mind* hypothesis holds that children with autism have a specific deficit (Leslie 1987) or delay (Baron-Cohen 1989) in the ability to form metarepresentations or symbolic second-order representations. It is this underlying cognitive deficit that produces the observed lack of symbolic play in children with autism. Several researchers have suggested that an underlying social deficit, either in the ability to affectively connect with others (Hobson 1990) or in intersubjectivity (Rogers and Pennington 1991), disrupts early emotion sharing and attunement between the infant and the parent and leads to later emerging deficits in symbolic play via its impact on the child's ability to recognize others as having differing views of the same situation. The *executive functioning* or *generativity* hypothesis (Harris 1993; Jarrold et al. 1996) suggests that executive functioning problems interfere with the child's ability to generate internal representations or schemas in the absence of prompts or cues. Thus, their spontaneous play remains highly repetitive and stereotyped. Although symbolic play deficits are one of the defining features of the disorder, there is evidence that children with autism exhibit deficits in functional play as well (Jarrold et al. 1993), which has been used to support the view that symbolic play deficits may

be the result of a more general deficit in executive functioning.

Current Knowledge

A number of more recent studies on the symbolic play skills in children with autism have clarified a number of questions regarding the scope and underlying causes of symbolic play deficits. One question is whether symbolic play deficits are due to difficulty in understanding the underlying symbolic representation (competence) or whether they are due to difficulty in generating play ideas (performance). Research with young (Rutherford et al. 2007) as well as older (Bigham 2010) children with autism has suggested that children with autism have difficulty both with the spontaneous use as well as the prompted use of symbolic actions, suggesting a difficulty with competence rather than performance. However, there is also evidence that children with autism's spontaneous symbolic play is more impaired than their prompted symbolic play, suggesting that the ability to generate play ideas may also be impaired.

Another issue in the field is whether play deficits in autism are specific to symbolic actions or are more general, involving functional actions as well. To date, the field has been mixed. Several studies, particularly those involving young children with autism, have suggested that play deficits extend to functional actions as well (Rutherford and Rogers 2003), whereas other studies have found deficits only on symbolic play tasks (Baron-Cohen 1987; Libby et al. 1997). One recent longitudinal study found that children with autism exhibited impairments in both functional and symbolic play when compared to children with typical development or developmental delay in the early preschool period. However, 1 year later, deficits in functional play were no longer present; yet symbolic play skills remained impaired (Rutherford et al. 2007). Thus, there appears to be an improvement in functional play with time, but not in symbolic play.

Several recent studies have attempted to directly test theories proposed to explain symbolic play deficits in children with autism. In one study,

longitudinal predictors of symbolic play were examined in preschoolers with autism, developmental delay, and typical development. Predictors included a spatial reversal task (a task thought to tap executive functions in preschoolers), joint attention (thought to be a precursor to meta-representation or theory of mind), and imitation. Joint attention skills were predictive of gains in symbolic play over time, whereas spatial reversal and imitation performance were not. In a second study with older children with autism, intellectual disability, and typical development, performance on a false belief task (an index of theory of mind ability) was concurrently associated with understanding of symbolic play acts, but not response inhibition (an aspect of executive function). Thus, current work is more supportive of a meta-representation explanation for symbolic play deficits.

Due to the prevalence and persistence of symbolic play deficits in children with autism, a number of intervention approaches have been used to teach symbolic play skills to children with autism (see Lang et al. 2009 for review). For example, Kasari, Freeman and Paparella (2006) used a randomized controlled trial to evaluate a symbolic play intervention that used structured and naturalistic teaching strategies in young children with autism. Children were randomly assigned to receive symbolic play training, joint attention training, or a control group. After 6 weeks, children in the symbolic play training group showed a significantly greater number of different, novel, child-initiated symbolic play acts and higher play level than children in the other two groups. Finally, the researchers found the children were able to generalize their skills from the individualized treatment with a therapist to playing with their caregiver (Kasari et al. 2006). In a follow-up study (Kasari et al. 2008), children in the symbolic play training group (and joint attention training group) made greater gains in language on standardized measures than the children in the control group 1 year later, suggesting that improving symbolic play skills can lead to improvements in language skills. Interestingly, although symbolic play and expressive language ability appear to be closely related in children with

autism (Stanley and Konstantareas 2007), training in language skills did not produce changes in use of symbolic play acts (Stahmer 1995), indicating that symbolic play skills may need to be directly targeted.

Future Directions

The vast majority of research on symbolic play in children with autism has examined these skills at a single time point. Future research is needed that can examine changes in symbolic play over time. In particular, it is important to examine both predictors of gains in symbolic play skills as well as outcomes of gains in symbolic play. Further research that can examine effective methods for improving symbolic play skills in children with autism is needed.

See Also

- ▶ [Executive Function \(EF\)](#)
- ▶ [Play](#)
- ▶ [Play Intervention](#)
- ▶ [Pretend Play](#)
- ▶ [Symbolic Behavior](#)
- ▶ [Theory of Mind](#)

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author. Text revision.
- Baron-Cohen, S. (1987). Autism and symbolic play. *British Journal of Developmental Psychology*, 5, 139–148.
- Baron-Cohen, S. (1989). The autistic child's theory of mind: A case of specific developmental delay. *Journal of Child Psychology and Psychiatry*, 30, 285–297.
- Belsky, J., & Most, R. K. (1981). From exploration to play: A cross-sectional study of infant free play behavior. *Developmental Psychology*, 17, 630–639.
- Bigham, S. (2010). Impaired competence for pretense in children with autism: Exploring potential cognitive predictors. *Journal of Autism and Developmental Disorders*, 40, 30–38.
- Charman, T., & Baron-Cohen, S. (1993). Brief report: Prompted pretend play in autism. *Journal of Autism and Developmental Disorders*, 27, 325–332.

- Fein, G. G. (1981). Pretend play in childhood: An integrative review. *Child Development*, 52, 1095–1118.
- Harris, P. L. (1993). Pretending and planning. In S. Baron-Cohen, H. Tager-Flusberg, & D. Cohen (Eds.), *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.
- Hobson, R. P. (1990). On acquiring knowledge about people and the capacity to pretend: Response to Leslie (1987). *Psychological Review*, 97, 114–121.
- Jarrold, C., Boucher, J., & Smith, P. (1993). Symbolic play in autism: A review. *Journal of Autism and Developmental Disorders*, 23, 281–307.
- Jarrold, C., Boucher, J., & Smith, P. (1996). Generativity deficits in pretend play in autism. *British Journal of Developmental Psychology*, 14, 275–300.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in young children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry*, 47, 611–620.
- Kasari, C., Paparella, T., Freeman, S., & Jahromi, L. B. (2008). Language outcome in autism: Randomized comparison of joint attention and play interventions. *Journal of Consulting and Clinical Psychology*, 76, 125–137.
- Lang, R., O'Reilly, M., Rispoli, M., Shogren, K., Machalicek, W., Sigafoos, J., et al. (2009). Review of interventions to increase functional and symbolic play in children with Autism. *Education and Training in Developmental Disabilities*, 44(4), 481–492.
- Leslie, A. M. (1987). Pretence and representation: The origins of 'theory of mind'. *Psychological Review*, 94, 412–426.
- Libby, S., Powell, S., Messer, D., & Jordan, R. (1997). Imitation of pretend play acts by children with autism and Down syndrome. *Journal of Autism and Developmental Disorders*, 27, 365–383.
- Libby, S., Powell, S., Messer, D., & Jordan, R. (1998). Spontaneous play in children with autism: A reappraisal. *Journal of Autism and Developmental Disorders*, 28, 487–497.
- Lowe, M., & Costello, A. J. (1976). *Manual for the symbolic play test*. Windsor: National Foundation for Educational Research.
- Rogers, S., & Pennington, B. (1991). A theoretical approach to the deficits in infantile autism. *Development and Psychopathology*, 3, 137–162.
- Rutherford, M. D., & Rogers, S. J. (2003). The cognitive underpinnings of pretend play in autism. *Journal of Autism and Developmental Disorders*, 33, 289–302.
- Rutherford, M. D., Young, G. S., Hepburn, S., & Rogers, S. J. (2007). A longitudinal study of pretend play in autism. *Journal of Autism and Developmental Disorders*, 37, 1024–1039.
- Sigman, M., & Ungerer, J. A. (1984). Cognitive and language skills in autistic, mentally retarded, and normal children. *Developmental Psychology*, 20, 293–302.
- Stahmer, A. C. (1995). Teaching symbolic play skills to children with autism using pivotal response training. *Journal of Autism and Developmental Disorders*, 25, 123–141.
- Stanley, G. C., & Konstantareas, M. M. (2007). Symbolic play in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37, 1215–1223.

Symbolic Thought

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Synonyms

[Intuitive thought](#)

Definition

Symbolic thought refers to the use of symbols (e.g., words and images) and mental representations of objects or events to represent the world (Hockenbury and Hockenbury 2002; Rathus 2007). Symbolic thought is one of the major developmental outcomes of language acquisition, and the ability to understand and use symbols allows children to engage in intellectual and social activities that are unique to humans (Carlson and Zelazo 2009; Karpov 2005).

There are two constructs of symbolic thought: mediation and intentionality. In mediation, a symbol is substituted for a real stimulus, and behavior can be controlled by the symbol rather than the stimulus. Intentionality refers to the awareness of the relationship between the symbol and the stimulus (Karpov 2005).

Symbolic thought gradually develops throughout infancy and early childhood and is evident in a child's use of imagination while playing (Carlson and Zelazo 2009; Hockenbury and Hockenbury 2002). The capacity for symbolic thought allows a child to begin abstract thinking and act

independently of what he or she sees and to begin abstract thinking (Karpov 2005). Its development is related to the development of self-reflection and social interaction (Carlson and Zelazo 2009). It is a precursor to sociodramatic play, such as role-playing (Karpov 2005). Children with an immature understanding of symbols may not understand the relationship between symbols and the objects they represent (Hockenbury and Hockenbury 2002).

See Also

- [Piagetian Stages](#)
- [Symbolic Behavior](#)
- [Symbolic Play](#)

References and Reading

- Carlson, S. M., & Zelazo, P. D. (2009). Symbolic thought. In J. B. Benson & M. M. Haith (Eds.), *Language, memory, and cognition in infancy and early childhood* (pp. 485–494). San Diego: Academic.
- Hockenbury, D. H., & Hockenbury, S. E. (2002). Lifespan development. In *Psychology* (3rd ed., pp. 367–408). New York: Worth Publishers.
- Karpov, Y. V. (2005). *The neo-Vygotskian approach to child development*. New York: Cambridge University Press.
- Rathus, S. A. (2007). Early childhood: Cognitive development. In *Childhood and adolescence: Voyages in development* (pp. 294–328). Belmont: Thomson Wadsworth.

synapses, chemical and electrical, differentiated by the method by which these action potentials travel between neurons. In chemical synapses, neurotransmitters are required to pass an action potential across the synaptic cleft, the space between the axon terminal and the dendrite. In electrical synapses, two neurons are physically connected via gap junctions allowing for action potential transmission.

See Also

- [Dendrite](#)
- [Neurotransmitter](#)

References and Reading

- Garber, K. (2007). Autism's cause may reside in abnormalities at the synapse. *Science*, 317, 190–191.

Synaptic Proteins

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Structure

The synapse is a highly specialized junction between two neurons, consisting of a presynaptic cell, a postsynaptic cell, and a space between the membranes of the two cells. There are two kinds of synapses, chemical (neurotransmitter-driven) and electrical, both occurring between neurons; to date, the synaptic proteins implicated in autism are involved in chemical synapses. In a chemical synapse, the presynaptic neuron is responsible for creating chemical neurotransmitters in small membrane-sealed droplets called vesicles, storing these in the presynaptic axon terminal (the part of the presynaptic neuron abutting the synaptic cleft) and releasing them into the synaptic cleft to bind to receptors on the membrane of the postsynaptic

Synapses

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Definition

A synapse is the area forming the junction between two neurons where an action potential passes from the axon terminal of one neuron to the dendrite of another. There are two types of

density, the specialized portion of the postsynaptic neuron on the synaptic cleft directly opposite the presynaptic axon terminal (Dubin 2002; Peça et al. 2011; Purves et al. 2001; Sherwood 2010). Voltage-modulated calcium ion channels on the presynaptic terminal allow an influx of calcium ions which prompts the stored vesicles to fuse with the cell membrane and release their neurotransmitter contents into the cleft (Lodish et al. 2000; Purves et al. 2001). Synapses can be excitatory or inhibitory, depending on whether they involve the release of an excitatory neurotransmitter or an inhibitory neurotransmitter. Binding of excitatory neurotransmitters to their specific receptors on the postsynaptic density causes ion channels linked to the receptors to open, allowing an influx of sodium ions to enter the cell, depolarizing it and increasing the likelihood that it will fire a signal, or action potential, across its synapse to the next neuron in the pathway, and so on. The binding of inhibitory neurotransmitters to their specific receptors on the postsynaptic density, conversely, results in the channels opening to allow influx of chloride ions or efflux of potassium ions, resulting in increasing polarization of the cell and reducing the probability that it will fire an action potential and transmit a signal to the following neuron (Alberts et al. 2002; Lodish et al. 2000; Sherwood 2010). Synaptic proteins play roles in all of this, from synaptic formation and development to synapse maintenance and signal transmission, and are responsible for the structure of both the presynaptic terminal and the postsynaptic density, as well as structural connections spanning the synaptic cleft.

Function

Synapses are essential to neural function and thus all brain activity. By relaying signals from one neuron to another and modulating the strength of the relayed signal (keeping it the same, attenuating it, or amplifying it), they allow for highly complex signals and “messages” between neurons and over long distances (neurologically speaking, i.e., along connections millions of cells long).

In a structure as specialized and involved as the synapse, understandably, synaptic proteins will vary widely in form and function. Some of the proteins are involved in large-scale structural alignment of the synapse, connecting with each other across the cleft anchoring the synapse together, as well as relaying the received signal on the postsynaptic side; these include CASK, neuexins, ERC, Piccolo, Bassoon, and catenins (presynaptic) (Waites and Garner 2011; Ziv and Garner 2004), and various neurotransmitter receptors, neuroligins, PSD95, SAP97, SAPAP, and SHANK genes (postsynaptic). Some, such as cadherins, which interact with each other across the synaptic cleft, and catenins, which interact with the cadherins inside the cell, occur on both sides of the synapse (Peça et al. 2011; Waites and Garner 2011; Ziv and Garner 2004). Others are involved in vesicle trafficking and secretion; these include syntaxin, Snap25, Rim, Rab3a, Munc13, and Munc18 (Ziv and Garner 2004).

Pathophysiology

Given the vital role synapses play in neural function, it is easy to understand how synaptic dysfunction could result in neuropsychiatric conditions. In recent years, a growing number of synaptic proteins have been implicated in the pathogenesis of autism. Nearly all of those that have been associated with autism have been so in the context of rare, likely highly deleterious mutations that would severely disrupt the function of the protein in question. When mutations in synaptic proteins are reported in association with autism, typical implicated pathologies are problems in neuronal migration and connection, as well as abnormal dendritic spines (the protruding structure of which the postsynaptic density is the end) (Waites and Garner 2011). The interacting neuexins (presynaptic) and neuroligins (postsynaptic) neuexin-1 (NRXN1) and neuroligin 4 X-linked (NGLN4) have been implicated several times and have the strongest evidence for involvement in ASD. Mutations in neuroligin 3 have also been identified in patients with ASD, and a mouse model of one mutation

identified in an ASD family demonstrated changes in synaptic function. Downstream of neuroligins in the postsynaptic density are the SHANK genes, binding indirectly to the neuroligins (Südhof 2008). Mutations/deletions in SHANK3, as part of 22q13.3 deletion syndrome, make it one of the most convincing and replicable autism genes (see ► [“SHANK 3”](#) entry). More recently, SHANK2 has also emerged as an autism candidate (Berkel et al. 2010, 2012, reviewed in Penzes et al. 2011). Cadherins have also been recently implicated in autism, including through the finding of a significantly associated SNP mapping between the genes encoding CDH9 and CDH10 (Wang et al. 2009) in the first large-scale genome wide association study in ASD. As noted, this finding has yet to be independently replicated. These types of proteins interact with each other across the synaptic cleft and are the cytoplasmic binding partners of the catenins (Waites and Garner 2011; Ziv and Garner 2004).

The notion that ASD is, at least in part, a reflection of synaptic pathology is supported by the increased rates of autism spectrum disorders in fragile X syndrome and in tuberous sclerosis. The genes that cause these syndromes, namely, FMRP (fragile X mental retardation protein), TSC1 (tuberous sclerosis complex 1), and TSC 2 (tuberous sclerosis complex 2), are known to play important roles in synaptic function.

Further research into these synaptic proteins will result in a greater understanding of their precise roles in synaptic function, and thus clarify how they are involved in autism. This will in turn hopefully allow for better diagnostic methods and treatment options, informed by the presence or absence of causative mutations in specific synaptic genes.

See Also

- [Course of Social Avoidance in Fragile X Syndrome, The](#)
- [Neuroligins](#)
- [NLGN3](#)
- [NRXN1](#)
- [SHANK 3](#)
- [Tuberous Sclerosis Complex](#)

References and Reading

- Alberts, B., Johnson, A., Lewis, J., Raff, M., Roberts, K., & Walter, P. (2002). *Molecular biology of the cell* (4th ed.). New York: Garland Science.
- Berkel, S., Marshall, C. R., Weiss, B., Howe, J., Roeth, R., & Moog, U. (2010). Mutations in the SHANK2 synaptic scaffolding gene in autism spectrum disorder and mental retardation. *Nature Genetics*, 42(6), 489–491.
- Berkel, S., Tang, W., Trevio, M., Vogt, M., Obenhaus, H. A., & Gass, P. (2012). Inherited and de novo SHANK2 variants associated with autism spectrum disorder impair neuronal morphogenesis and physiology. *Human Molecular Genetics*, 21(2), 344–357.
- Dubin, M. W. (2002). *How the brain works*. Malden: Blackwell Science.
- Lodish, H., Berk, A., Zipursky, S. L., Matsudaira, P., Baltimore, D., & Darnell, J. (2000). *Molecular cell biology* (4th ed.). New York: W. H. Freeman.
- Peça, J., Ting, J., & Feng, G. (2011). Snapshot: Autism and the synapse. *Cell*, 147(3), 706, 706.e1–706, 706.e1.
- Penzes, P., Cahill, M. E., Jones, K. A., VanLeeuwen, J. E., & Woolfrey, K. M. (2011). Dendritic spine pathology in neuropsychiatric disorders. *Nature Neuroscience*, 14(3), 285–293.
- Purves, D., Augustine, G. J., Fitzpatrick, D., Katz, L. C., LaMantia, A.-S., & McNamara, J. O. (Eds.). (2001). *Neuroscience* (2nd ed.). Sunderland: Sinauer Associates.
- Sherwood, L. (2010). *Human physiology: From cells to systems* (7th ed.). Belmont: Brooks/Cole.
- Südhof, T. C. (2008). Neuroligins and neuroligins link synaptic function to cognitive disease. *Nature*, 455(7215), 903–911.
- Waites, C. L., & Garner, C. (2011). Presynaptic function in health and disease. *Trends in Neurosciences*, 34(6), 326–337.
- Wang, K., Zhang, H., Ma, D., Bucan, M., Glessner, J. T., & Abrahams, B. S. (2009). Common genetic variants on 5p14.1 associate with autism spectrum disorders. *Nature*, 459(7246), 528–533.
- Ziv, N. E., & Garner, C. (2004). Cellular and molecular mechanisms of presynaptic assembly. *Nature Reviews Neuroscience*, 5(5), 385–399.

Synaptic Pruning

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Definition

Synaptic pruning is the process by which synaptic connections that are weak, usually as a result of

being underused, are eliminated. This process of eliminating weak connections allows the brain to process more frequently encountered and relevant information with greater efficiency. It is part of the developmental process and results in changes in cortical thickness over time.

See Also

- [Synapses](#)
- [Synaptogenesis](#)

References and Reading

Kolb, B., & Whishaw, I. Q. (2009). *An introduction to brain and behavior* (3rd ed.). New York: Worth.

Synaptogenesis

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Definition

Synaptogenesis is the formation of synapses between neurons. This process begins in gestation and continues throughout life. The greatest amount of synapse formation occurs early in life, during what is referred to as a “critical period” in development, during which sensitivity to developmental influences is heightened. Synaptogenesis in early development is coupled with synaptic pruning, the deletion of excess synapses.

See Also

- [Synaptic Pruning](#)

References and Reading

Zhiling, Y., Fujita, E., Tanabe, Y., Yamagata, T., Momoi, T., & Momoi, M. Y. (2008). Mutations in the gene encoding CADM1 are associated with autism spectrum disorder. *Biochemical and Biophysical Research Communications*, 377, 926–929.

Syndrome of Deficits in Attention, Motor Control, and Perception (DAMP)

- [Attention Deficit/Hyperactivity Disorder](#)

Syntax

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Synonyms

[Grammar](#); [Sentence structure](#)

Definition

Syntax is the set of rules and principles that govern the organization of words into sentences in any individual language. In typical development, adult-like syntax is acquired by age 5. In ASD, it has long been thought that syntax is relatively spared and usually on par with cognitive development. However, recent research suggests that a subgroup of speakers with ASD have syntactic deficits above and beyond their cognitive limitations that resemble the deficits seen in specific language disorders.

See Also

- [Expressive Language](#)

References and Reading

- Boucher, J. (2003). Language development in autism. *International Journal of Pediatric Otorhinolaryngology*, 67, 159–163.
- Fletcher, P. (2009). Syntax in child language disorders. In R. Schwartz (Ed.), *Handbook of child language disorders* (pp. 388–405). New York: Psychology Press.
- Stephanie, D., & Julie, F. (2015). Exploring links between language and cognition in autism spectrum disorders: Complement sentences, false belief, and executive functioning. *Journal of Communication Disorders*, 54, 15–31.
- Tager-Flusberg, H., & Joseph, R. M. (2003). Identifying neurocognitive phenotypes in autism. *Philosophical Transactions of the Royal Society of London, Series B*, 358, 303–314.

Systemizing

► Systems Intervention

Systemizing Quotient (SQ-7)

► Self-Report Autism Scales for Adults

Systems Intervention

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Synonyms

Empathizing-systemizing theory; Organizational prevention; Systemizing

Definition

Systems are a set of interacting or interrelated entities forming an integrated whole or larger,

more complex formation. Interventions are acts of intervening or coming between two or more things or acts that occur between two or more points in time. When combined, the terms systems and intervention describe actions that either purposefully or incidentally interfere with a larger organizational configuration. Systems intervention can occur at macro and micro levels. Systems approaches at the larger, macro level are organizational arrangements that enhance and diminish treatment effectiveness. Volkmar et al. (2005) describe systems as being a part of the environment and are found within six classifications, each with its own set of rules and regularities: technical, natural, abstract, social, motoric, and organizational. The interactivity of these six components, each working separately within its own unique parameters and then as a collective whole, coordinate to form a system. In developing systems intervention, consideration for the larger system and each interlocking component with surrounding parameters needs to be analyzed and evaluated for causal roles and relationships.

At the micro level, the concept of systems intervention applies to specific treatments, interventions, or evidence-based practices aimed at intervening by aligning with particular approaches and methodologies that focus on a systematic delivery of intervention following a standardized protocol. For example, the Picture Exchange Communication System (PECS) is a training protocol that is based on applied behavior analysis (ABA) techniques, whereby functional communication is systematically taught using prompting and reinforcement strategies that lead to independent communication. PECS also teaches discrimination of symbols and then sequencing to create simple sentences. In the most advanced phases, individuals are taught to comment and answer direct questions.

Further, with respect to systems and organizations represented within the individual with ASD, it is hypothesized that input-output systems are affected by the disability and the systemizing of information may be compromised given deficit areas involving communication, behavior, and social reciprocity.

See Also

- [ABA](#)
- [Empathizing-Systemizing Theory](#)
- [Organizational Prevention](#)
- [Social](#)
- [Systemizing](#)

References and Reading

- Charlop-Christy, M. H., & Jones, C. (2006). The picture exchange communication system: A nonverbal communication program for children with autism spectrum disorders. In R. J. McCauley & M. E. Fey (Eds.), *Treatment of language disorders in children* (pp. 105–122). Baltimore: Paul H. Brookes Publishing.
- Charlop-Christy, M. H., Carpenter, M., Le, L., LeBlanc, L. A., & Kellet, K. (2002). Using the picture exchange communication system (PECS) with children with autism: Assessment of PECS acquisition, speech, social-communicative behavior, and problem behavior. *Journal of Applied Behavior Analysis*, 35, 213–231. <https://doi.org/10.1901/jaba.2002.35-213>.
- Flay, B. R., Biglan, A., Boruch, R. F., Castro, F. G., Gottfredson, D., Kellam, S., et al. (2004). Standards of evidence: Criteria for efficacy, effectiveness and dissemination. Retrieved 10 Sept 2008 from <http://www.preventionresearch.org/Standards of Evidencebook.pdf>
- Ganz, J. B., & Simpson, R. L. (2004). Effects on communicative requesting and speech development of the picture exchange communication system in children with characteristics of autism. *Journal of Autism and Developmental Disorders*, 34, 395–409. <https://doi.org/10.1023/B:JADD.0000037416.59095.d7>.
- Green, V. A., Pituch, K. A., Itchon, J., Choi, A., O'Reilly, M., & Sigafos, J. (2006). Internet survey of treatments used by parents of children with autism. *Research in Developmental Disabilities*, 27, 70–84. <https://doi.org/10.1016/j.ridd.2004.12.002>.
- Preston, D., & Carter, M. (2009). A review of the efficacy of the picture exchange communication system intervention. *Journal of Autism Developmental Disorders*, 39, 1471–1486.
- Volkmar, F., Paul, R., Klin, A., & Cohen, D. (2005). *Handbook of autism and pervasive developmental disorders* (3rd ed.). New York: Wiley.

T

T Scores

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Synonyms

[Standardized scores](#)

Definition

T scores (or T-scores) are an example of standardized scores, where the mean is equal to 50 and the standard deviation is equal to 10. They are a linear transformation of Z-scores, which have mean 0 and standard deviation 1; a T score can be obtained from a Z-score by the formula $T = 50 + 10Z$. T scores are convenient because scores below 0 and above 100 are virtually impossible; in fact, 99.7% of the time, a T score will lie between 20 and 80, because these limits are 3 standard deviations below and above the mean, respectively. The Mullen Scales of Early Learning is one example of an assessment that reports T scores.

See Also

- [Mullen Scales of Early Learning](#)
- [Standard Scores \(Z and T Scores\)](#)
- [Standardization](#)

References and Reading

- Clark-Carter, D. (2005). T scores. In B. Everitt & D. Howell (Eds.), *Encyclopedia of statistics in behavioral science*. Chichester: Wiley. Retrieved from <http://onlinelibrary.wiley.com/doi/10.1002/0470013192.bsa690/full>
- Mullen, E. M. (1995). *Mullen scales of early learning: AGS edition*. Circle Pines: American Guidance Service.
- Tamhane, A. C., & Dunlop, D. D. (2000). *Statistics and data analysis: From elementary to intermediate* (p. 117). Upper Saddle River: Prentice-Hall.

TABS

- [Temperament and Atypical Behavior Scale Screener \(TABS Screener\)](#)

TABS Screener

- [Temperament and Atypical Behavior Scale Screener \(TABS Screener\)](#)

TACL-4

- [Test for Auditory Comprehension of Language – Fourth Edition](#)
-

Tact

- [Label](#)
-

Tact(s)

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Synonyms

[Label](#); [Referent](#)

Definition

A tact is a verbal action evoked by something in the environment and reinforced by an unspecified consequence (Skinner 1957). For example, if a child who sees a picture of a dog says “Doggy!” and the child’s parent then smiles and replies, “Wow! That *is* a dog,” the child’s statement (i.e., “Doggy!”) would be considered a tact. This term was introduced by B. F. Skinner and refers to a specific type of verbal act within an operant conditioning framework designed to analyze the functions of verbal behavior.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Label](#)
- [Operant Conditioning](#)
- [Skinnerian Categories of Verbal Behavior](#)
- [Verbal Behavior Interventions](#)

References and Reading

Skinner, B. F. (1957). *Verbal behavior*. New York: Appleton-Century-Crofts.

Tactile

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Synonyms

[Touch](#)

Definition

Tactile or touch is a sensory experience that is first detected by receptors in the skin. These receptors then transmit the information into the spinal cord to be further processed. Tactile information is diverse and carried through different pathways in the spinal cord to provide the brain with specificity about the encounter of the skin with the environmental object that produced the touch. For example, tactile information may be used in a discriminatory manner to sort sizes of coins without the use of vision, or the tactile sensation may be protective in nature when an unexpected touch is encountered. The discriminative tactile sensation can be refined with training, as is seen when teaching a person to interpret Braille.

References and Reading

- Gardner, E. P., & Kandel, E. R. (2000). Touch. In E. R. Kandel, J. H. Schwartz, & T. M. Jessell (Eds.), *Principles of neural science* (pp. 451–471). New York: McGraw-Hill.
- Gertz, S. D. (2007). *Liebman’s neuroanatomy made easy and understandable* (7th ed.). Austin: Pro-ED.

Tactile Defensiveness

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Synonyms

[Tactile sensitivity](#); [Touch sensitivity](#)

Definition

Individuals with autism spectrum disorders with tactile hypersensitivity have been commonly reported in the literature (Baranek et al. 1997). Individuals with tactile defensiveness may show reluctance to manipulate various textures and materials based on their texture or may exhibit an exaggerated or persistent response to unexpected touch occurrences and may even avoid situations in which these materials are presented or settings in which unexpected touch may occur. Additionally, these individuals may exhibit hypervigilance to the potential threat of unexpected touch occurrences. Individuals with tactile defensiveness may also restrict their food repertoire based on texture of the food itself. Participation in such activities of daily living as hair cutting, nail cutting, and toothbrushing may also be impacted by tactile defensiveness. Baranek et al. (1997) explored the relationship between tactile hypersensitivity and stereotypes and found that children with ASD and tactile sensitivity were more likely to exhibit visual stereotypes, behavioral rigidities, and repetitive vocalizations.

See Also

- ▶ [Sensory Integration and Praxis Tests \(SIPT\)](#)
- ▶ [Sensory Processing](#)
- ▶ [Touch Sensitivity](#)

References and Reading

- Ayres, A. (1972). *Sensory integration and learning disabilities*. Los Angeles: Western Psychological Services.
- Ayres, A. (1973). *Sensory integration and learning disorders*. Los Angeles: Western Psychological Services.
- Ayres, A. (1979). *Sensory integration and the child*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (1985). *Developmental Dyspraxia and adult onset apraxia*. Torrance: Sensory Integration International.
- Baranek, G. T., Foster, L. G., & Berkson, G. (1997). Tactile defensiveness and stereotypic behaviors. *American Journal of Occupational Therapy*, 51, 91–95.
- Parham, L. D., & Mailloux, Z. (2001). Sensory integration. In J. Case-Smith (Ed.), *Occupational therapy for children* (4th ed.). St. Louis: Mosby.
- Tomchek, S. D. (2010). Sensory processing in individuals with an autism spectrum disorder. In H. M. Kuhaneck & R. Watling (Eds.), *Autism: A comprehensive occupational therapy approach* (pp. 135–161). Bethesda: AOTA Press.

Tactile Defensiveness and Discrimination Test-Revised

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Synonyms

[TDDT-R](#)

Description

This tool is not currently available commercially. According to the published literature, the Tactile Defensiveness and Discrimination Test-Revised (TDDT-R) is comprised of multiple subtests with two of these measuring responses to light touch stimuli. These subtests involve applying light touch to various points on the

examinee's skin via a finger puppet/cotton swab or small stickers. The examinee is observed for behavioral reactions such as withdrawal from the stimulus, scratching or rubbing the skin, or negative facial grimaces. Intensity of responses is coded on a four-point scale with higher scores indicating more symptoms. Test items are administered with vision allowed for training purposes then with vision occluded for measurement of response. Other subtests measure orienting/habituation, tactile discrimination, and sensory seeking behaviors during play-based tasks (e.g., finding hidden objects and exploration of textures such as sand, lotion, etc.).

Scores can be calculated for accuracy of discrimination and for level of tactile defensiveness, under internally and externally controlled conditions. The manual is available from the author; no norms are currently published.

Historical Background

The Tactile Defensiveness and Discrimination Test-Revised (TDDT-R; Baranek 2010; Baranek and Berkson 1994) is a 15-min behavioral observation assessment of tactile processing (hyperresponsiveness and tactile discrimination) for children with autism and other developmental disabilities ages 2 through 14 years. This performance-based measure involves presentation of play-based tactile tasks and observing and coding responses.

Psychometric Data

This tool has been validated in several studies with children with autism, fragile X syndrome, and other developmental disabilities ages 2 through 14 years (Baranek and Berkson 1994; Baranek et al. 2002; Boyd et al. 2010; Watson et al. 2011). Inter-rater reliability has been documented as >90%.

Clinical Uses

This tool is not currently commercially available, so it is unlikely that it will be encountered in clinical practice. However, it continues to be used in research and may be encountered in the literature.

References and Reading

- Baranek, G. T. (2010). *Tactile defensiveness and discrimination test – revised (TDDT-R)*. Unpublished manuscript.
- Baranek, G. T., & Berkson, G. (1994). Tactile defensiveness in children with developmental disabilities: Responsiveness and habituation. *Journal of Autism and Developmental Disorders*, 24, 457–471.
- Baranek, G. T., Chin, Y. H., Hess, L. M. G., Yankee, J. G., Hatton, D. D., & Hooper, S. R. (2002). Sensory processing correlates of occupational performance in children with fragile X syndrome: Preliminary findings. *American Journal of Occupational Therapy*, 56, 538–546.
- Boyd, B. A., Baranek, G. T., Sideris, J., Poe, M., Watson, L. R., Patten, E., et al. (2010). Sensory features and repetitive behaviors in children with autism and developmental delays. *Autism Research*, 3, 1–10.
- Creedon, M. P., & Baranek, G. T. (1988, July). Touch another way: Recognizing and managing tactile defensiveness at home and in the classroom [Summary]. *Proceedings of the Annual Conference of the Autism Society of America* (pp. 49–54). As cited in Baranek & Berkson 1994.
- Watson, L. R., Patten, E., Baranek, G. T., Poe, M., Boyd, B. A., Freuler, A., et al. (2011). Differential associations between sensory response patterns and language, social, and communication measures in children with autism or other developmental disabilities. *Journal of Speech, Language, and Hearing Research*, 54(6), 1562–1576.

Tactile Sensitivity

- [Tactile Defensiveness](#)
- [Touch Sensitivity](#)

Tactile Temporal Resolution

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Definition

Our awareness of surrounding environments is perceived, keeping temporal continuity, which is important for constructing a robust representation of the environment. Temporal limitations to sensory information processing inhibit extraordinary inflow of sensory inputs. For example, in tactile temporal order judgment, in which sensory inputs are successively provided, temporal resolution (as a discriminative limitation) is approximately 50 to 100 (Ide et al. 2019; Takahashi et al. 2013; Yamamoto and Kitazawa 2001). This suggests that we roughly summarize sensory inputs that are delivered within a certain temporal range, as the information of a single unit because of temporal resolution and cognitive processing capacity limitations.

Buhusi and Meck (2005) presented a neural timing process framework which was further sub-divided into three different stages. The millisecond timing stage represents the shortest time scale for neural processing, at ≤ 1 s, and served speech and motor control functions. The stages in interval timing and circadian timing were longer at over ≥ 1 s and ≥ 1 h, respectively, and active

during decision-making, conscious time estimation, and the sleep-wake cycle.

Numerous reports have suggested the presence of enhanced perceptual functions in individuals with autism spectrum disorder (ASD). These enhancements may manifest as elevated detection and discrimination performance (Dakin and Frith 2005). Such studies mostly concentrated on spatial sensory processing, for example, detecting and searching for target stimuli amidst distractive stimuli (Bertone et al. 2005). Fewer works have examined perceptual ability in the temporal domain. Herein we overview studies which examined temporal processing in the brain in experimental psychological and neuroscientific review fields. We then introduce our ongoing studies which are examining the perceptual/cognitive characteristics of internal timing in individuals with ASD and the contribution of extraordinary temporal resolution to symptomatic sensory hyperreactivity.

Historical Background

Although temporal processing has garnered little attention in the autism literature, there is reason to believe that some individuals with ASD exhibit outstanding timing abilities in their daily lives. Colman et al. (1976) found that children with ASD manifested frequent repetitive behaviors and decreased attention to their jobs when they were in the presence of fluorescent lighting that flickered at 60 times per second, rather than incandescent lighting. Their findings suggested that some individuals with ASD possessed an extremely high temporal resolution (i.e., over 60 Hz of visual temporal resolution), sufficient to perceive flickering. This frequency of light flickering would be challenging for neurotypical individuals to identify. Another example is that individuals with ASD frequently complain of tactile hyperreactivity or perceived discomfort in response to skin contact with certain fabric

textures or clothing tags (Dunn 2001). Scratching the skin on a finely textured surface produces high-frequency vibrotactile information. Consequently, complaints of uncomfortable sensations in response to clothing scratches originate from the high temporal resolution of these tactile sensory inputs.

Perceptual time duration is another dimension which reflects temporal limitations for sensory stimuli processing. Honma et al. (2019) studied the accuracy with which individuals with ASD could reproduce various temporal durations. Participants were instructed to wait for an arbitrary time duration (10 or 20 s) and respond by tapping a tablet when they felt the instructed duration had elapsed from stimulus onset. Their results showed less accuracy of reproduction in individuals with ASD than neurotypical controls in their responses to shorter (10 s) duration conditions. Considering that higher individual deviations of reproduction performance in individuals with ASD were demonstrated in the auditory modality (Martin et al. 2010), they might have difficulty precisely identifying the temporal duration of sensory stimuli on an interval timing scale (Buhusi and Meck 2005).

The neural bases of stimulus temporal processing have been broadly surveyed in terms of stimulus duration or estimation of elapsed time from stimulus onset. The basal ganglia and supplementary motor area (SMA) are likely involved in modality-independent temporal processing of sensory inputs. Based on our neuroanatomical knowledge of information processing through the thalamo-cortico-striatal loop, the basal ganglia are suspected to play an important role in timing processes (Buhusi and Meck 2005). This circuit includes the prefrontal cortex and the posterior parietal cortex. Activation of these areas during interval timing tasks has been confirmed by prior investigations. The neural circuit between the cortices and the basal ganglia might form an internal clock for temporal processing (striatal beat frequency model).

Animal pharmacological studies have demonstrated that administration of a dopaminergic agonist (e.g., methamphetamine) into the striatum reduces the estimated time. Conversely,

administration of an antagonist (e.g., haloperidol) prolonged the time. Taken together, these findings suggest that the dopaminergic system can modulate the speed of “internal clock” (Meck 1996). A recent mouse study revealed that activation of dopaminergic neurons in the substantia nigra pars compacta (SNc), a part of basal ganglia, induced a longer estimation of stimulus duration. In contrast, inhibition leads to a shorter estimation of stimulus duration (Soares et al. 2016). Dopaminergic signaling from the ventral tegmental area (VTA) to the frontal cortices may also modulate time perception (Kim and Narayanan 2018). While it is still unclear that somatosensory temporal information could also be processed in the overlapped circuit, neurotransmission might be critical for precise duration or time estimation. Abnormal functioning of the prefrontal cortex and basal ganglia has been frequently reported in individuals with ASD and may lead to aberrant stimulus temporal processing (Allman and Meck 2012).

Current Knowledge

Many studies that used psychophysical techniques examined temporal resolution by evaluating perceptual/cognitive limitations to temporal discrimination of a sensory stimulus when another stimulus is delivered within a short temporal interval. During the simultaneous judgment (SJ) task, participants answer whether or not double stimuli, successively delivered with different short temporal time intervals, are perceived as synchronous or asynchronous inputs. During the temporal order judgment (TOJ) task, respondents judge the order of stimuli delivered during the SJ task. Tactile temporal resolution of normal participants was reported to be about 30–60 ms in response to SJ task of vibrotactile stimuli (Hirsh and Sherrick 1961; Miyazaki et al. 2016) and 60–80 ms in response to the TOJ task (Ide et al. 2019; Takahashi et al. 2013; Yamamoto and Kitazawa 2001). These data imply that different neural circuitry stages might link different tasks since few differences in tactile temporal resolution were reported relative to task features

(i.e., SJ or TOJ). A study that used the SJ task reported that visual temporal resolution was higher in individuals with ASD compared with neurotypical individuals (Falter et al. 2011). That is, individuals with ASD could finely discriminate double visual stimuli presented with short temporal lags. Another TOJ study showed that individuals with ASD manifested lower temporal resolution of vibrotactile stimuli than neurotypical participants; however, individuals with ASD exhibited higher temporal resolution when the stimuli were synchronously delivered with conditioning background stimuli (Tommerdahl et al. 2008).

One study demonstrated the neural correlates of neurotypical individuals during tactile SJ and TOJ using functional magnetic resonance imaging (MRI) (Miyazaki et al. 2016). The SMA and the basal ganglia showed heightened BOLD signals (as a proxy of neural response) during each task in comparison to the resting state. During between-task comparisons, greater activations of the bilateral insula cortices were shown during SJ rather than during TOJ tasks. Conversely, the bilateral dorsal premotor, the left ventral premotor cortices, the superior/inferior parietal lobule, and the thalamus exhibited greater activation during the TOJ task than during the SJ task. These brain regions may also be involved as TOJ neural circuits and during lateralization to the left hemisphere (Takahashi et al. 2013). Moreover, an essential role of neural activation in the parietal-occipital sulcus involving precuneus for proper task performance of TOJ has been shown (Takahashi and Kitazawa 2017).

Neural activation of the basal ganglia-cortices network may be important for tactile temporal resolution tasks as demonstrated in rodents (Kim and Narayanan 2018; Soares et al. 2016). Dopamine projections from the VTA to the cortex may modulate the frequency of cortical oscillations (Meck et al. 2013). Thus, the frequency of cortical oscillation depends on the dynamics of synaptic neurotransmitter and may in turn predict temporal performance. Assuming that a phasic neural oscillation reflects the perceptual processing cycle, stimulus temporal resolution is

likely to be modulated by the perceptual cycle. According to this Temporal Framing Theory (see a review in Baumgarten et al. 2015), if two stimuli were presented during one cycle of a neural oscillation, the stimuli would more likely be perceived as a single stimulus. On the other hand, if the stimuli were presented across two cycles, they would more likely be perceived as two separate stimulations. In line with this theory, faster parieto-occipital alpha-band oscillation predicted more accurate discrimination performance of two successive flashes (Samaha and Postle 2015).

Similarly, the phase of an alpha- or low beta- (8–20 Hz) oscillation in the primary somatosensory cortex predicted discrimination accuracy of electrical pulse stimulations (Baumgarten et al. 2015). A study of tactile TOJ using magnetoencephalography (MEG) demonstrated that the phase of alpha-band oscillation is crucial for proper order judgment (Takahashi and Kitazawa 2017). One recent study which examined the effect of transcranial alternating current stimulation (tACS) for tactile TOJ reported that a 10 Hz electrical stimulation of the bilateral posterior parietal cortices improved temporal resolution (Otsuru et al. 2019).

In line with these reported neural substrates of temporal resolution, Ide et al. (2019) demonstrated that diversity of tactile temporal resolution in individuals with ASD was associated with sensory hyperresponsivity, a diagnostic characteristic of ASD. In that study, participants performed a tactile TOJ task using vibrotactile stimuli. While the tactile temporal resolution of individuals with ASD did not differ from that of neurotypical controls, there was a significant positive correlation between temporal resolution and sensory hyperresponsivity severity assessed using the *Adolescent/Adult Sensory Profile* (AASP) (Brown et al. 2001). Individuals with ASD with higher temporal resolution during the tactile TOJ task tended to report more frequent pathognomonic experiences of sensory hyperresponsivity during their daily lives. In our unpublished study, we analyzed the neural correlates of an individual with ASD who showed extremely heightened tactile temporal resolution

and severe sensory hyperresponsivity. The patient's tactile temporal resolution was almost ten times greater than the controls'. As demonstrated by fMRI during a tactile TOJ task, the patient showed greater BOLD responses in the left superior frontal gyrus, the ventral premotor cortex, and the superior temporal gyrus than the control participants. Consistent with previous TOJ literature, greater activations were found in left hemisphere regions (Miyazaki et al. 2016; Takahashi et al. 2013). A group analysis that pooled all the participants showed that the percentage of correct responses positively correlated left superior frontal gyrus activation and negatively correlated with activation in the right inferior frontal gyrus. We speculated that increased and decreased neural activities in different parts of the frontal area regulated sensory input temporal resolution.

Future Directions

In addition to some studies that addressed characteristics of temporal processing in individuals with ASD, our research suggested individual differences in processing. We also found that excessively high temporal resolution engages diagnostic aspects of sensory hyperresponsivity. The findings may seem to counter previous reports that demonstrated lower accuracy of time reproduction of sensory stimuli, as discussed above (Honma et al. 2019; Martin et al. 2010). For the three stages of timing processes proposed by Buhusi and Meck (2005), the processing of ≤ 1 ms was assumed to be separate from processing of ≥ 1 ms. Studies that reported high temporal resolution in individuals with ASD, including our study, examined ≤ 1 ms millisecond timing processes using psychophysical tasks, such as SJ and TOJ. Such tasks reflect low-level sensory processing (Falter et al. 2011; Ide et al. 2019; Tommerdahl et al. 2008). In contrast, the studies that reported lower performance used second timing processes that were ≥ 1 ms (i.e., interval timing and circadian timing) during reproduction and discrimination tasks (Honma et al. 2019; Martin et al. 2010). These differences

of processing stage in terms of time perception might produce the apparently contradictory temporal resolution findings in individuals with ASD.

Alteration of gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter, is likely the underlying basis of the considerably high temporal resolution of sensory inputs in individuals with ASD and may result in sensory hyperresponsivity (Cellot and Cherubini 2014). Reduced concentration of GABA or density of GABAergic receptors has been observed in broad brain areas of individuals with ASD. These findings suggest an imbalance between neural circuitry excitation and inhibition (E/I) (Cellot and Cherubini 2014). Development of proton magnetic resonance spectroscopy (^1H -MRS) has allowed researchers to perform in vivo measurement of brain metabolites. One study found that neurotypical individuals with higher frequency discrimination performance for vibrotactile stimuli showed lower GABA concentrations in the sensorimotor cortex (Puts et al. 2011). Future studies should examine GABA concentrations in the brains of individuals with ASD and determine if these concentrations are associated with temporal resolution task performance, which relates with sensory hyperresponsivity.

As mentioned above, individuals with ASD occasionally complain about the flickering of fluorescent lights and tickling sensations caused by touching of certain textures to their skin (Colman et al. 1976; Dunn 2001). These atypical perceptual/cognitive experiences may be related to the temporal resolution of sensory inputs. Individuals with ASD frequently report that their sensory hypersensitivity is affected by other psychological conditions, including high stress, anxiety, and so on. Thus, relationships with autonomic nervous system functions are suggested. Since researchers' interests is centered on the contributions of those psychological states to sensory hypersensitivity, revealing the effects of such states on psychophysiological task performance, including temporal resolution, should assist us in developing more effective clinical interventions.

References and Reading

- Allman, M. J., & Meck, W. H. (2012). Pathophysiological distortions in time perception and timed performance. *Brain*, 135(3), 656–677. <https://doi.org/10.1093/brain/awr210>.
- Baumgarten, T. J., Schnitzler, A., & Lange, J. (2015). Beta oscillations define discrete perceptual cycles in the somatosensory domain. *Proceedings of the National Academy of Sciences*, 112(39), 12187–12192. <https://doi.org/10.1073/pnas.1501438112>.
- Bertone, A., Mottron, L., Jelenic, P., & Faubert. (2005). Enhanced and diminished visuo-spatial information processing in autism depends on stimulus complexity. *Brain*, 128, 2430–2441. <https://doi.org/10.1093/brain/awh561>.
- Brown, C., Tollefson, N., Dunn, W., Cromwell, R., & Filion, D. (2001). The adult sensory profile: Measuring patterns of sensory processing. *American Journal of Occupational Therapy*, 55, 75–82.
- Buhusi, C. V., & Meck, W. H. (2005). What makes us tick? Functional and neural mechanisms of interval timing. *Nature Reviews Neuroscience*, 6(10), 755–765. <https://doi.org/10.1038/nrn1764>.
- Cellot, G., & Cherubini, E. (2014). GABAergic signaling as therapeutic target for autism spectrum disorders. *Frontiers in Pediatrics*, 2, 70. <https://doi.org/10.3389/fped.2014.00070>.
- Colman, R. S., Frankel, F., Ritvo, E., & Freeman, B. J. (1976). The effects of fluorescent and incandescent illumination upon repetitive behaviors in autistic children. *Journal of Autism and Childhood Schizophrenia*, 6(2), 157–162.
- Dakin, S., & Frith, U. (2005). Vagaries of visual perception in autism. *Neuron*, 48, 497–507. <https://doi.org/10.1016/j.neuron.2005.10.018>.
- Dunn, W. (2001). The sensations of everyday life: Empirical, theoretical, and pragmatic considerations. *American Journal of Occupational Therapy*, 55(6), 608–620.
- Falter, M. C., Elliot, A. M., & Bailey, J. A. (2011). Enhanced visual temporal resolution in autism spectrum disorders. *PLoS One*, 7(3), e32774. <https://doi.org/10.1371/journal.pone.0032774>.
- Hirsh, I. J., & Sherrick, C. E., Jr. (1961). Perceived order in different sensory modality. *Journal of Experimental Psychology*, 62(5), 423–432. <https://doi.org/10.1037/h0045283>.
- Honma, M., Itoi, C., Midorikawa, A., Terao, Y., Masaoka, Y., Kuroda, T., . . . Kato, N. (2019). Contraction of distance and duration production in autism spectrum disorder. *Scientific Reports*, 9(1), 8806.
- Ide, M., Yaguchi, A., Sano, M., Fukatsu, R., & Wada, M. (2019). Higher tactile temporal resolution as a basis of hypersensitivity in individuals with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49(1), 44–53. <https://doi.org/10.1007/s10803-018-3677-8>.
- Kim, Y. C., & Narayanan, N. S. (2018). Prefrontal D1 dopamine-receptor neurons and delta resonance in interval timing. *Cerebral Cortex*, 29(5), 2051–2060. <https://doi.org/10.1093/cercor/bhy083>.
- Martin, J. S., Poirier, M., & Bowler, D. M. (2010). Brief report: Impaired temporal reproduction performance in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 40(5), 640–646. <https://doi.org/10.1007/s10803-009-0904-3>.
- Meck, W. H. (1996). Neuropharmacology of timing and time perception. *Brain Research. Cognitive Brain Research*, 3(3–4), 227–242. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/8806025>.
- Meck, W. H., Church, R. M., & Matell, M. S. (2013). Hippocampus, time, and memory – A retrospective analysis. *Behavioral Neuroscience*, 127(5), 642–654. <https://doi.org/10.1037/a0034201>.
- Miyazaki, M., Kadota, H., Matsuzaki, K. S., Takeuchi, S., Sekiguchi, H., Aoyama, T., & Kochiyama, T. (2016). Dissociating the neural correlates of tactile temporal order and simultaneity judgements. *Scientific Reports*, 6(1), 23323. <https://doi.org/10.1038/srep23323>.
- Otsuru, N., Kamijo, K., Otsuki, T., Kojima, S., Miyaguchi, S., Saito, K., . . . Onishi, H. (2019). 10 Hz transcranial alternating current stimulation over posterior parietal cortex facilitates tactile temporal order judgment. *Behavioural Brain Research*, 368, 111899. <https://doi.org/10.1016/j.bbr.2019.111899>.
- Puts, N. A. J., Edden, R. A. E., Evans, C. J., McGlone, F., & McGonigle, D. J. (2011). Regionally specific human GABA concentration correlates with tactile discrimination thresholds. *Journal of Neuroscience*, 31(46), 16556–16560. <https://doi.org/10.1523/JNEUROSCI.4489-11.2011>.
- Samaha, J., & Postle, B. R. (2015). The speed of alpha-band oscillations predicts the temporal resolution of visual perception. *Current Biology*, 25(22), 2985–2990. <https://doi.org/10.1016/j.cub.2015.10.007>.
- Soares, S., Atallah, B. V., & Paton, J. J. (2016). Midbrain dopamine neurons control judgment of time. *Science*, 354(6317), 1273–1277. <https://doi.org/10.1126/science.125234>.
- Takahashi, T., & Kitazawa, S. (2017). Modulation of illusory reversal in tactile temporal order by the phase of posterior α rhythm. *The Journal of Neuroscience*, 37(21), 5298–5308. <https://doi.org/10.1523/JNEUROSCI.2899-15.2017>.
- Takahashi, T., Kansaku, K., Wada, M., Shibuya, S., & Kitazawa, S. (2013). Neural correlates of tactile temporal-order judgment in humans: An fMRI study. *Cerebral Cortex*, 23(8), 1952–1964. <https://doi.org/10.1093/cercor/bhs179>.
- Tommerdahl, M., Tannan, V., Holden, K. J., & Baranek, T. G. (2008). Absence of stimulus-driven synchronization effects on sensory perception in autism: Evidence for local underconnectivity? *Behavioral and Brain Functions*, 4, 19. <https://doi.org/10.1186/1744-9081-4-19>.
- Yamamoto, S., & Kitazawa, S. (2001). Reversal of subjective temporal order due to arm crossing. *Nature Neuroscience*, 4(7), 759.

Tailoring Job Skills

- [Job Carving](#)

Takao Syndrome

- [CATCH 22 \(Chromosome 22q11 Deletion Syndrome\)](#)

Talking

- [Speech](#)

Tangentially

- [Flight of Ideas](#)

Tantrum

- [Temper Tantrum](#)

Tardive Dyskinesia

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Synonyms

[Antipsychotic-induced dyskinesia](#); [Chronic dyskinesia](#); [Irreversible dyskinesia](#); [Late-onset dyskinesia](#); [Neuroleptic-induced dyskinesia](#)

Definition

Tardive dyskinesia (TD) is a chronic extrapyramidal adverse effect, consisting of abnormal,

involuntary movements of the tongue, lips, face, trunk, and/or extremities. TD can either occur spontaneously or as an adverse effect during antidopaminergic therapy, particularly long-term antipsychotic treatment, but also after exposure to antidopaminergic medications inhibiting gastric motility (e.g., metoclopramide). The annualized TD risk in adults is approximately 5% with first-generation antipsychotics and $\leq 1\%$ with second-generation antipsychotics (Correll et al. 2004; Correll and Schenk 2008). In children and adolescents, mostly treated with low-dose risperidone, the annualized TD risk was 0.4% (Correll and Kane 2007). The exact pathophysiology of TD is still unknown. Increased TD risk is associated with older age, female sex, White ethnicity, history of acute extrapyramidal side effects, higher daily and cumulative antidopaminergic medication dose, and with first-generation versus second-generation antipsychotics (Margolese et al. 2005a). Patients with central nervous damage and those with preexisting motor abnormalities, such as are not uncommon in patients with autism spectrum disorders, may also be at greater risk for TD (Campbell et al. 1997; Connor et al. 2001; Masi et al. 2003). Measurement of TD severity and determination of TD caseness can be achieved with the Abnormal Involuntary Movement Scale (AIMS) (Guy 1976) or the dyskinesia subscale of the Extrapyramidal Side Effect Scale (ESRS) (Chouinard et al. 1980). The classic definition of TD according to the Schooler-Kane criteria consists of either a rating of mild (Schooler and Kane 1982) in at least two body regions or a rating of at least moderate (Chouinard et al. 1980) in at least one body area. In youth and adults, antidopaminergic treatment of at least 3 months is currently required to diagnose treatment emergent TD, while in the elderly the minimum treatment duration was set as 1 month. However, there is no known biological minimum or required duration of antidopaminergic exposure after which TD can emerge. Nevertheless, TD needs to be differentiated from withdrawal dyskinesia that follows during or closely after antipsychotic discontinuation (or switch), and that can last for several months. In youth, TD may be more reversible than in adults and, especially,

the elderly when discontinuing the offending dopamine blocking medication (Correll and Kane 2007). Except for discontinuing all antipsychotic agents or a switch from an antipsychotic to clozapine, no other treatment strategy has been found to be consistently efficacious for the treatment of TD (Margolese et al. 2005b). Sometimes, antipsychotic dose increase can mask abnormal involuntary movements.

See Also

- ▶ Antipsychotics: Drugs
- ▶ Clozapine
- ▶ Neuroleptics

References and Reading

- Campbell, M., Armenteros, J. L., Malone, R. P., Adams, P. B., Eisenberg, Z. W., & Overall, J. E. (1997). Neuroleptic-related dyskinesias in autistic children: A prospective, longitudinal study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 36(6), 835–843.
- Chouinard, G., Ross-Chouinard, A., Annabel, L., & Jones, B. D. (1980). The extrapyramidal symptom rating scale. *Canadian Journal of Neurological Sciences*, 7, 233.
- Connor, D. F., Fletcher, K. E., & Wood, J. S. (2001). Neuroleptic-related dyskinesias in children and adolescents. *Journal of Clinical Psychiatry*, 62(12), 967–974.
- Correll, C. U., & Kane, J. M. (2007). One-year tardive dyskinesia rates in children and adolescents treated with second-generation antipsychotics: A systematic review. *Journal of Child and Adolescent Psychopharmacology*, 17(5), 647–655.
- Correll, C. U., & Schenk, E. M. (2008). Tardive dyskinesia and new antipsychotics. *Current Opinion in Psychiatry*, 51, 151–156.
- Correll, C. U., Leucht, S., & Kane, J. M. (2004). Lower risk for tardive dyskinesia associated with second-generation antipsychotics: A systematic review of one-year studies. *Current Opinion in Psychiatry*, 16(3), 414–425.
- Guy, W. (1976). *ECDEU assessment manual for psychopharmacology* (Publication ABM 76-338, pp. 534–537). Washington, DC: US Department of Health, Education, and Welfare.
- Margolese, H. C., Chouinard, G., Kolivakis, T. T., Beaulclair, L., & Miller, R. (2005a). Tardive dyskinesia in the era of typical and atypical antipsychotics. Part 1: Pathophysiology and mechanisms of induction. *Canadian Journal of Psychiatry*, 50(9), 541–547.
- Margolese, H. C., Chouinard, G., Kolivakis, T. T., Beaulclair, L., Miller, R., & Annable, L. (2005b). Tardive dyskinesia in the era of typical and atypical antipsychotics. Part 2: Incidence and management strategies in patients with schizophrenia. *Canadian Journal of Psychiatry*, 50(11), 703–714.
- Masi, G., Cosenza, A., Mucci, M., & Brovedani, P. (2003). A 3-year naturalistic study of 53 preschool children with pervasive developmental disorders treated with risperidone. *Journal of Clinical Psychiatry*, 64(9), 1039–1047.
- Schooler, N. R., & Kane, J. M. (1982). Research diagnoses for tardive dyskinesia. *Archives of General Psychiatry*, 39, 486–487.

Target Behavior

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Synonyms

Challenging behavior; Skill; Unit of response

Definition

Target behavior is the behavior identified to be changed, the prescribed behavior. This behavior can be defined either by function or by topography. A functionally defined target behavior identifies a response by its effect on the person or the environment. For example, pica is defined by placing inedible items in the mouth and consuming them. In contrast, topographically based definitions identify a response by its form. Function-based definitions are preferable and should be used wherever possible because they lead to more accurate and reliable measurement systems and because they capture the response class more accurately.

References and Reading

- Martin, G., & Pear, J. (2003). *Behavior modification: What it is and how to do it* (7th ed.). Upper Saddle River: Prentice Hall.

Targeting IEP Social Goals in Summer Camps

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Definitions

After it has been determined that a child qualifies for special education services, an Individualized Educational Program (IEP) is developed by the school with input from parents. The IEP is a written legal document that includes goals, the method for obtaining these goals, and a measurement plan. The term “social” is individually defined depending on the particular IEP goal of the child. For example, for some children appropriately taking turns during a simple activity is a social goal. For others, engaging in appropriate reciprocal verbal conversation is a social goal. Inclusive settings are community environments that have a natural proportion of children with autism spectrum disorder (ASD) or other disability and typically developing peers. In these settings, peers can be used as appropriate role models as well as providing support, participation, and assistance during goal implementation. Paraprofessionals are one-on-one assistants that generally do not hold an advanced higher education degree and are hired to assist and monitor students in the implementation of IEP goals. Paraprofessionals are supervised by individuals with advanced degrees, such as teachers in the school setting. Peer mediation refers to strategies in which peers

are recruited to assist with the implementation of target goals. Priming is a procedure wherein children are exposed to a concept or activity before it is presented in real time. The use of priming has resulted in improved learning and lower levels of disruptive behavior in academic and social settings. Pivotal Response Treatment (PRT) is an intervention package consisting of specific motivational components (e.g., choice, natural rewards, task variation, and rewarding attempts) that have been shown to improve responsiveness, learning, and decrease challenging behaviors when incorporated into the intervention. Ritualistic and repetitive behaviors (RRBs), sometimes referred to in the literature and in clinical practice as stereotypic behaviors or self-stimulatory behaviors, are idiosyncratic repetitive actions that may interfere with learning and socialization. Self-management is a procedure wherein children are taught to discriminate, evaluate, and then monitor their own behaviors. Self-management can be programmed to occur in natural environments, thereby reducing the need for constant vigilance by an adult or treatment provider.

Historical Background

By definition, all children with ASD have difficulty with social communication skills (American Psychiatric Association 2013), and thus, developing interventions for improving this area has been a major goal of treatment programs. Historically, children with ASD were excluded from the educational system because of the lack of effective interventions to improve social communication and other areas (cf. Koegel and Koegel 2019). However, in the 1960s, interventions documenting that these children could learn began emerging in the scientific literature.

During the mid-1970s, laws were passed requiring children with ASD to be included in school settings. Over time, it became evident that interventions for developing social behaviors would be of major importance, and the large number of typically developing peers might make the school setting an ideal place for social interactions. Although social goals were considered to be a

critical area, interventions remained a difficult and relatively undeveloped area in individualized educational programs (IEPs) for children with ASD. Impairments in social communication can lead to academic underachievement, a decrease or complete lack of employment later in life, and fewer opportunities to participate in leisure activities (Holwerda et al. 2012), among other challenges. Thus, the importance of targeting social goals is critical. Despite the fact that many children with ASD report a desire to have friendships (Bauminger and Kasari 2000), they often spend unstructured play periods alone and/or engaging in RRBs rather than socializing in school and other settings where peers are available. Another challenge is that while explicit individualized teaching of social skills is critical for these children (White et al. 2007), often the least trained individuals (e.g., paraprofessionals) are the only staff available to deliver social interventions during unstructured periods. In addition to the need for social intervention during the school year, a related concern expressed by parents and teachers is continuity of treatment and the lack of school services during summer months (Brookman et al. 2003). While many schools offer extended school year programs, these often are short term and do not include typically developing peers, that may be helpful for assisting with the social intervention and who serve as important role models.

Underlying Theory, Goals, and Objectives

The underlying goals and objectives of this research are based on the theory that ASD is largely a performance or motivational deficit rather than an ability deficit (Koegel and Egel 1979; Koegel and Koegel 2019). The theory suggests that children with ASD may acquire socialization skills such as asking a question or initiating to peers; however, they may not perform these skills due to a severe lack of motivation. We have discussed this as the child exhibiting learned helplessness (Koegel and Egel 1979; Koegel and Koegel 2019). Based on a theory of learned helplessness, we hypothesized that the children might actually be capable of performing certain

behaviors, but just do not perform them due to a severe motivational deficit and related difficulty in associating reinforcers with their behavior. We also hypothesized that if an empirically based intervention could be developed to motivate children with ASD to engage in social interactions, there would be rapid and widespread improvement in their behaviors rather than needing to teach the children to exhibit the behaviors one at a time over a long time period. Further, implementing such an intervention using empirically validated motivational procedures should improve socialization with widespread generalization (Koegel and Koegel 2019). Of specific relevance to this chapter is the hypothesis that if such motivational interventions could be implemented so as to target IEP goals throughout the day in summer camp, achievement of the IEP goals would be rapidly acquired, putting the children ahead of schedule. Finally, it has been hypothesized that as a result of massed practice in a setting such as a summer camp where interventions with motivational components could be implemented across settings and peers, generalization and maintenance of social skills would occur.

Treatment Participants

Individualized treatment packages for socialization, described herein, have been empirically shown to be effective with a wide range of children diagnosed with ASD (Koegel et al. 2019). The interventions have been individualized and therefore can be used with a wide age range of children enrolled in summer camps, including elementary school aged children through adolescence. Further, since target goals are individualized, the procedures can be used with a variety of ability levels ranging from minimally verbal children, whose goals may be focused on areas such as turn taking, to highly verbal children with ASD whose goals focus on verbal social communication. While the research on targeting IEP goals in summer camps has focused on children diagnosed with ASD who demonstrate social communication difficulties with peers, we speculate that the

procedures would also be effective with any child having difficulties socializing with peers.

Treatment Procedures

Interventions for difficulties in social communication frequently have been based upon a model of learned helplessness, where children are taught to attempt to make communicative responses, and then reinforced for making those attempts, even if the initial attempts are not entirely accurate. Reinforcing the children's attempts to socially communicate, rather than insisting on their performance of completely correct communicative responses, has resulted in the elimination of learned helplessness and in rapid and large increases in the children's social communication (cf. Koegel and Koegel 2012, 2019). While there is not one standardized model of teaching social skills, it is common for professionals to use motivational procedures and antecedent interventions. Social interventions can be adult-directed (e.g., "show me how you share the candy with your friends"), peer-directed (e.g., using a peer to prompt for turn taking), or child-directed (e.g., setting up an activity around the child's interests to capture motivation for initiations to peers).

Mechanism of Intervention. Related to the mechanism of intervention, by targeting goals in an inclusive camp setting, the children with ASD have extensive opportunities to be exposed to typically developing peer models who can also serve as intervention mediators. It is also noteworthy that paraprofessionals spend a large amount of their time in such inclusive settings and have the potential to be major treatment delivery agents if they were properly trained. Therefore, an additional goal of this research has been to assess whether paraprofessionals can effectively implement the interventions.

In the Koegel et al. (2019) study, participants attended a summer camp for 2 weeks. Intervention consisted of PRT and Positive Behavioral Intervention and Support (PBIS) strategies, which were individualized for each child and their specific social goal. The strategies were designed to eliminate learned helplessness and increase motivation

for social communication. The procedures included reinforcing attempts, using natural reinforcers, following the children's lead in order to identify motivational stimuli, priming, peer-mediated intervention, and self-management. Many of the activities organized by the camp (e.g., ropes, archery, and gymnastics) were new to the participants. Therefore, priming (Koegel and Koegel 2006; Koegel et al. 2003) was a method used to expose the children with ASD to activities before they encountered the activity with their peers in the summer camp. Specifically, the participants were provided with instructions and prompts to follow the rules of the activities (e.g., prompting the child to stand in line with friends and wait for their turn) to anticipate what was expected (e.g., when it's your turn you will get a safety harness and then you can climb up as high as you can).

In order to further promote participation in camp activities, typically developing peers selected based on their requests to assist the child with ASD were recruited to engage with the child with ASD. Paraprofessionals prompted the peers to initiate, take turns, participate in social conversations, and engage in social interactions with the participants based on each child's individualized goals. For example, when engaging in turn-taking, all of the campers were prompted to wait for their turns and to assist the child with ASD if necessary. Self-management, wherein the participants were taught to discriminate and record their occurrences or absence of targeted social behaviors (Koegel and Koegel 2006) was used during everyday activities with peers to increase targets such as social conversation.

We also anticipated that teaching the IEP social goals in an inclusive setting would be highly effective. Inclusive settings have been shown to be a beneficial setting to target social skills for individuals with ASD because willing peers can easily be recruited to model appropriate behavior and assist with intervention (Koegel and Koegel 2019; Rogers 2000; Watkins et al. 2014). Furthermore, peer-mediated interventions targeting social skills are more likely to generalize and maintain overtime when implemented in inclusive settings (Chamberlain et al. 2006; Watkins et al. 2014).

Efficacy Information

There has been a large amount of empirical research in the general area of social communication for children with ASD demonstrating gains if social areas are targeted (Koegel and Koegel 2019). Previous research on PBIS has consistently demonstrated that it is effective in reducing challenging behaviors for children with ASD (Carr et al. 1999; Koegel et al. 1996; Neitzel 2010; Turnbull et al. 2002). Further, PBIS and PRT strategies have been effectively implemented in summer camp settings by trained and supervised paraprofessionals to increase social skills and to reduce disruptive behaviors (Brookman et al. 2003). However, the feasibility of providing continuity with school interventions, by targeting IEP social goals for children with ASD during the summer has not been empirically addressed until recently (Koegel et al. 2019). In this current research on PRT, Koegel et al. (2019) collected data during behavioral observations for successive 10-min intervals during baseline, intervention, and follow-up. Each child showed gains and met their year-long IEP goals during the two-week intensive summer camp, only after the paraprofessionals providing the PRT motivational interventions reached fidelity of implementation. Specifically, whereas all children showed low levels to no social engagement during baseline probes, results demonstrated that following a 2-week summer camp program, with consistent targeted intervention using PRT, all participants made social improvements, reaching their year-long IEP goals within the 2 weeks, and that maintained at follow-up in natural nontreatment environments.

Outcome Measurement

There are various tools used to measure socialization outcomes. The most common method is collecting behavioral observation data on specific behaviors such as rate of initiations, percentage of engagement, level of enthusiasm, or frequency of turn taking. In the empirical research cited above, each participant received intervention during the

summer camp that was chosen from the child's IEP. Target outcomes included social engagement, appropriate eye contact during conversation, turn taking, and providing appropriate details when retelling a recent experience. Behavioral observations during 10-min representative probes were coded for each of the target behaviors. For example, within the probes, engagement was scored as appropriate if the participant engaged in the activity with peers and followed the rules of the game during successive 30s intervals. At the end of each session, the total number of intervals scored as appropriate was divided by the total number of appropriate and inappropriate intervals and multiplied by 100 to yield a percentage of appropriate engagement per session. By collecting frequent data on observable and measurable social behaviors in natural settings with peers, progress made on socialization goals can be evaluated.

Qualifications of Treatment Providers

Professionals who develop and supervise social skills interventions should have a background in the basic principles of applied behavior analysis, functions of behavior, and PBIS, as well as certification for meeting fidelity of implementation in PRT. The providers can include a variety of different people who are certified as proficient in PRT, including special education teachers, psychologists, BCBA providers, speech and language pathologists, and paraprofessionals. In the Koegel et al. (2019) study, one-on-one paraprofessionals supported the children with ASD throughout their camp day. The paraprofessionals were undergraduate students majoring in psychology who had taken an introductory undergraduate course in ASD. Graduate students with at least 4 years of experience working with children with PRT supervised the undergraduate students through in-vivo feedback for about 1 h per day. The graduate students were supervised by university faculty. While most adults, including parents with a relevant background in PRT, and students can deliver social skills interventions, it is important that a highly experienced professional is available to supervise, monitor progress, and provide feedback.

Summary

The results of the above empirical research suggest that inclusive summer camps may be ideal settings for targeting IEP socialization goals, even for just a few weeks, for children with ASD. One-on-one paraprofessionals can be taught to implement goals with fidelity of implementation; however, some background (i.e., coursework), on-site supervision, and ongoing training may be helpful. Overall, the research is optimistic that the important area of socialization may be improved during a short term and intensive intervention implemented by supervised paraprofessionals in a community summer camp setting with typical typically developing peers.

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Publishing.
- Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development*, 71, 447–456.
- Brookman, L., Boettcher, M., Klein, E., Openden, D., Koegel, R. L., & Koegel, L. K. (2003). Facilitating social interactions in a community summer camp setting for children with autism. *Journal of Positive Behavior Interventions*, 5, 249–252.
- Carr, E. G., Levin, L., McConachie, G., Carlson, J. I., Kemp, D. C., Smith, C. E., & McLaughlin, D. M. (1999). Comprehensive multisituational intervention for problem behavior in the community: Long-term maintenance and social validation. *Journal of Positive Behavior Interventions*, 1, 5–25.
- Carr, E. G., Dunlap, G., Horner, R. H., Koegel, R. L., Turnbull, A. P., Sailor, W., . . . Fox, L. (2002). Positive behavior support: Evolution of an applied science. *Journal of Positive Behavior Interventions*, 4, 4–16.
- Chamberlain, B., Kasari, C., & Rotheram-Fuller, E. (2006). Involvement or isolation? The social networks of children with autism in regular classrooms. *Journal of Autism and Developmental Disorders*, 37, 230–242.
- Holwerda, A., Van der Klink, J. J., Groothoff, J. W., & Brouwer, S. (2012). Predictors for work participation in individuals with an autism spectrum disorder: A systematic review. *Journal of Occupational Rehabilitation*, 22, 333–352.
- Koegel, R. L., & Egel, A. L. (1979). Motivating autistic children. *Journal of Abnormal Psychology*, 88, 418.
- Koegel, R. L., & Koegel, L. K. (2006). *Pivotal response treatments for autism: Communication, social & academic development*. Baltimore: Paul H. Brookes Publishing.
- Koegel, R. L., & Koegel, L. K. (2012). *PRT pocket guide*. Baltimore: Paul H. Brookes Publishing Company.
- Koegel, R. L., & Koegel, L. K. (2019). *Pivotal response treatments for autism spectrum disorders*. Baltimore: Paul H. Brookes Publishing.
- Koegel, L. K., Dunlap, G., & Koegel, R. L. (1996). *Positive behavioral support: Including people with difficult behavior in the community*. Baltimore: Paul H. Brookes Publishing.
- Koegel, L. K., Koegel, R. L., Frea, W., & Green-Hopkins, I. (2003). Priming as a method of coordinating educational services for students with autism. *Language, Speech, and Hearing Services in Schools*, 34, 228–235.
- Koegel, L. K., Glugatch, L. B., Koegel, R. L., & Castellon, F. A. (2019). Targeting IEP social goals for children with autism in an inclusive summer camp. *Journal of Autism and Developmental Disorders*, 49, 1–11. <https://doi.org/10.1007/s10803-019-03992-4>.
- Neitzel, J. (2010). Positive behavior supports for children and youth with autism spectrum disorders. *Preventing School Failure: Alternative Education for Children and Youth*, 54, 247–255.
- Rogers, S. J. (2000). Interventions that facilitate socialization in children with autism. *Journal of Autism and Developmental Disorders*, 30, 399–409.
- Turnbull, A., Edmonson, H., Griggs, P., Wickham, D., Sailor, W., Freeman, R., . . . Riffel, L. (2002). A blueprint for schoolwide positive behavior support: Implementation of three components. *Exceptional Children*, 68, 377–402.
- Watkins, L., O'Reilly, M., Kuhn, M., Gevarter, C., Lancioni, G. E., Sigafoos, J., & Lang, R. (2014). A review of peer-mediated social interaction interventions for students with autism in inclusive settings. *Journal of Autism Developmental Disorders*, 45(1070), 1083. <https://doi.org/10.1007/s10803-014-2264>.
- White, S. W., Keonig, K., & Seahill, L. (2007). Social skills development in children with autism spectrum disorders: A review of the intervention research. *Journal of Autism and Developmental Disorders*, 37, 1858–1868.

Task Analysis

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A task analysis is used to break complex tasks into a sequence of smaller steps or actions. For some

individuals on the autism spectrum, even simple tasks can present complex challenges. Having an understanding of all the steps involved in a particular task can assist in identifying any steps that may need extra instruction and will help teach the task in a logical progression. A task analysis is developed using one of the three methods. First, competent individuals who have demonstrated expertise can be observed and steps documented. A second method is to consult experts in performing the required task. And finally, those who are teaching the skill can perform the task themselves and document steps.

As task analyses are developed, it is important to remember the skill level of the person, the age, communication and processing abilities, and prior experiences in performing the task. For those on the autism spectrum, also remember their tendency toward literal interpretation of language. For example, students who have been told to put the peanut butter on the bread when making a peanut butter and jelly sandwich have literally just placed the entire jar on the bread. Below are two examples of task analyses.

Putting a Coat On

1. Pick up the coat by the collar (the inside of the coat should be facing you)
2. Place your right arm in the right sleeve hole
3. Move your arm through until you can see your hand at the other end
4. Now reach behind with your left hand
5. Locate the left sleeve hole
6. Put your arm in the left sleeve hole
7. Move your arm through until you see your hand at the other end
8. Adjust the coat so it is ready to zip

Washing Hands

1. Turn on right faucet
2. Turn on left faucet
3. Place hands under water
4. Dispense soap
5. Rub palms to count of 5
6. Rub back of left hand to count of 5
7. Rub back of right hand to count of 5
8. Turn off water
9. Take paper towel

10. Dry hands to count of 5
11. Throw paper towel away

Skills taught using a task analysis (TA) include daily living skills, such as brushing teeth, bathing, making a meal, and performing a variety of household chores. Task analysis is also useful in desensitization programs such as tolerating haircuts, having teeth cleaned, and tolerating buzzers or loud environments.

Again, the number of steps involved and the wording used will differ depending on the individual. Determining the steps to a TA as well as the starting point for an individual often requires collecting baseline data, and/or examining the individual's ability to complete any or all of the required steps. Once implementation begins, the TA may need to be revised to address any additional needs. It is important that all steps are operationally defined.

Once a task analysis is developed, chaining procedures are used to teach the task. Forward chaining involves teaching the sequence beginning with the first step. Typically, the learner does not move onto the second step until the first step is mastered. In backward chaining, the sequence is taught beginning with the last step. And again, the previous step is not taught until the final step is learned. One final strategy is total task teaching. Using this strategy, the entire skill is taught and support is provided or accommodations made for steps that are problematic. Each of these strategies has benefits. In forward chaining, the individual learns the logical sequence of a task from beginning to end. In backward chaining, the individual immediately understands the benefit of performing the task. In total task training, the individual is able to learn the entire routine without interruptions. In addition, they are able to independently complete any steps that have been previously mastered.

Regardless of the strategy chosen, data has to be collected to document successful completion of the entire routine and progress on individual steps. How an individual progresses through the steps of the task analysis and what strategies are used have to be determined via data collection.

References and Reading

Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson Merrill Prentice Hall.

TDDT-R

► [Tactile Defensiveness and Discrimination Test-Revised](#)

TEACCH Transition Assessment Profile – Computer Version

► [TEACCH Transition Assessment Profile \(TTAP\)](#)

TEACCH Transition Assessment Profile (TTAP)

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Abbreviations

CRS	Cumulative Record of Skills
CSAW	Community Site Assessment Worksheet
DAC	Daily Accomplishment Chart
IDEA	Individuals with Disabilities Education Act of 1997
IEP	Individualized Education Program

Synonyms

[TEACCH transition assessment profile – computer version](#); [Treatment and Education of Autistic and Communication-related handicapped CHildren](#); [TTAP](#); [TTAP-CV](#)

Definition

Treatment and Education of Autistic and Communication-related handicapped CHildren is an evidence-based service, training, and research program for individuals of all ages and skill levels with autism spectrum disorders. Established in the early 1970s by Eric Schopler and colleagues, the TEACCH program has worked with thousands of individuals with autism spectrum disorders and their families.

Description

The TEACCH Transition Assessment Profile (TTAP) by Gary Mesibov, John B. Thomas, S. Michael Chapman, and Eric Schopler (ProEd, Inc. 2007) is a revision of the *Adolescent and Adult Psychoeducational Profile (AAPEP)*. This test was developed for adolescents and adults with autism spectrum disorders, particularly those with transitional needs. The *TTAP* is structured to satisfy those provisions in the Individuals with Disabilities Education Act (IDEA) of 1997 that require adolescents to be evaluated and provided with a transition plan by age 14. In 2011, the *TTAP-CV: TEACCH Transition Assessment Profile, Second Edition – Computer Version* was published as a companion volume to the TTAP. This electronic version supports the development and documentation of goals for transition planning and career/job training with a detailed system of references and tools. These tools provide an opportunity for continuity in ongoing assessment throughout the adolescent and adult work history of an individual with ASD.

Educators, parents, counselors, and care providers can use this tool to assist individuals with autism spectrum disorders to prepare for independence in adult life (i.e., personal development, recreational living, adult integration into employment and residential arrangements, etc.). The *TTAP* will also help providers identify the individual’s principle transition goals, strengths, and weaknesses. Second, a “Cumulative Record of Skills” (CRS), along with several data collection forms, provides an efficient method of ongoing assessment in community-based instruction. The

TTAP can be used to facilitate educational and transitional planning. Emphasis is on evaluating multiple functional skill areas, including both concrete vocational skills and a variety of social, leisure, and conceptual skills that are crucial to effective vocational and residential training. The assessment instrument emphasizes:

1. Assessment for transition
2. Focus on the six major functional areas
3. Assessment and performance in different environmental contexts
4. A scoring system that assists goal development
5. Information on environmental accommodation
6. Identification of preferences for individuals with limited communication skills

Career planning is a lifelong process that requires repeated, ongoing assessment to adjust preferences and skills and to prioritize needs. The TEACCH Transition Assessment Profile creates a system that supports continuity of programming across the lifespan in defining and programming for positive adult outcomes.

The entire system of the TTAP allows users to record and update learner progress toward obtaining skills related to post-school outcomes.

The TTAP provides a way for users to generate reports about individual learners and to further identify necessary transition skills. The system lends itself to periodic review (e.g., the Individualized Education Program process).

Historical Background

In 1988, Gary Mesibov, Eric Schopler, Bruce Schaeffer, and Rhoda Landrus created *The Adolescent and Adult Psychoeducational Profile (AAPEP)*. This instrument was designed as a highly structured, skills-based instrument to evaluate current and potential skills in areas most important for successful independent functioning in the home and community. The instrument was intended to assist in diagnostic assessment as well as in defining objectives for instruction that would support independent community functioning.

As a result of nearly 20 years of supported employment services and adult residential

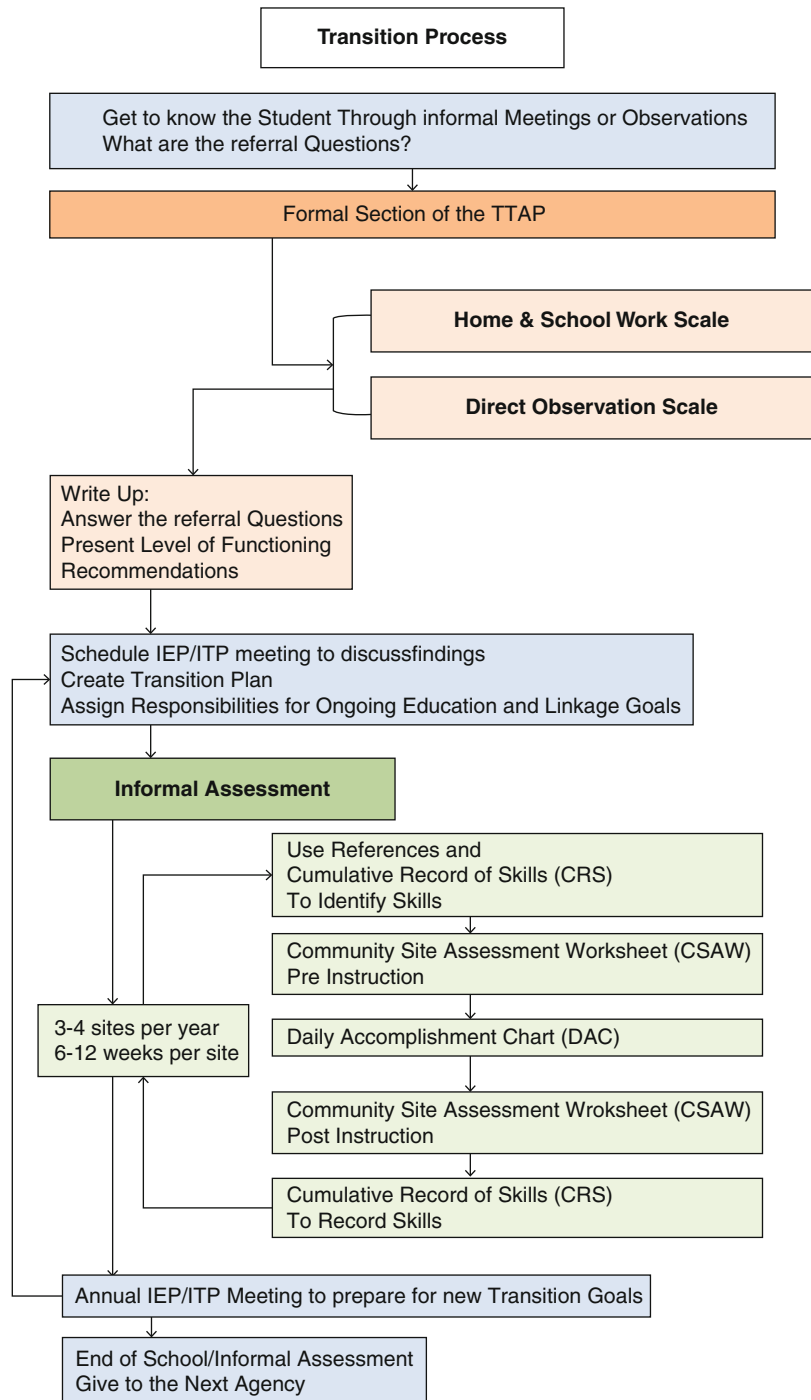
services provided by Division TEACCH, the instrument was redesigned to address the specific needs of individuals with autism spectrum disorders in transition services in public schools and in adult services, both residential and vocational. The skills-based instrument, the AAPEP, was redesigned to address a broader range of cognitive abilities. The “formal assessment” of the AAPEP was redesigned as an “initial assessment of transition needs,” as required by IDEA, the Individuals with Disabilities Education Act. Moreover, a significant expansion of the ongoing assessment process that is required by IDEA was added to assure that both school-based and adult service organizations had a means of documenting their programming and instruction.

The two instruments of the TTAP represent a means of direct experiential evaluation in adolescent and adult services. Inventory assessments, a primary form of adolescent and adult vocational assessments, are frequently ineffective in reliably measuring interests and employment potential for individuals with autism spectrum disorders.

Clinical Uses

The TEACCH Transition Assessment Profile provides both an initial assessment of transition needs and an ongoing documentation system that supports job development, job training, and supported employment service for individuals with autism spectrum disorders. The initial assessment of transition needs fulfills the requirement for such an assessment by age 16 for individuals requiring transition planning within the Individualized Education Program (IEP) process. It identifies potential educational objectives that can be reviewed by the IEP team. The ongoing assessment supports systematic instruction of specific objectives related to positive adult outcomes over the secondary school career of individuals with autism spectrum disorders. Moreover, the documentation system is easily transferred to adult service providers so that continuity of service provision can be addressed efficiently (Fig. 1).

**TEACCH Transition
Assessment Profile
(TTAP), Fig. 1** Transition
process



See Also

- [Adulthood, Transition to](#)
- [Employment](#)
- [Employment in Adult Life](#)
- [Feedback on Provider Work Performance](#)
- [Independent Living](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [School to Work Transition Process](#)
- [Transitional Living](#)

References and Reading

- Clark, G. M. (1998). *Assessment for transition planning*. Austin: PRO-ED.
- Clark, G. M., & Patton, J. R. (2005). *Transition planning inventory (updated version)*. Austin: PRO-ED.
- Cronin, M. E., & Patton, J. R. (1993). *Life skills instruction for all students with special needs*. Austin: PRO-ED.
- Individuals with Disabilities Education Act of 2004, H.R. 1350, 108th Cong.
- Mesibov, G. B., Schopler, E., Schaffer, B., & Landrus, R. (1998). *Individualized assessment and treatment for autistic and developmentally disabled children (Adolescent and adult psychoeducational profile) (Vol. 4)*. Austin: PRO-ED.
- Michaels, C. A. (1998). *Transition to employment*. Austin: PRO-ED.
- Patton, J. R., & Dunn, C. (1998). *Transition from school to young adulthood: Basic concepts and recommended practices*. Austin: PRO-ED.
- Wehman, P. (1998). *Developing transition plans*. Austin: PRO-ED.

overseen by professional consultants and should not be used without the guidance of a behavioral consultant or a speech-language pathologist.

Historical Background

The activities in *Teach Me Language* were reportedly developed over a number of years, following the authors' work on language difficulties for children with pervasive developmental disorders. No other historical information is available.

Rationale or Underlying Theory

Freeman and Dake (1997) suggest that there are no books that give hands-on, explicit instructions for working on the language needs specific to children with autism, Asperger's syndrome, and other related pervasive developmental disorders (PDDs). This was the impetus for their creation of a language manual that would provide exercises and drills to address those language weaknesses typically seen in children with PDDs. The manual capitalizes on the visual strengths reported for children with autism or autistic-like disorders as much of the material within the book uses visual scripts, word or phrase prompts, etc. (Freeman and Dake 1997).

Goals and Objectives

Teach Me Language focuses on six specific goals with chapters describing activities to address each of these goals. They are as follows:

- Improve social language: activities are designed to facilitate the child's ability to speak about a single topic as well as conversing with a peer.
- Increase comprehension and production of general information: activities are designed to support understanding of factual knowledge, an ability to compare and contrast concepts, defining vocabulary, and using true/false concepts based on general information the child learns.
- Improve use of grammar and syntax: activities are designed to facilitate understanding and use of the structure of language to create or assemble original sentences.

Teach Me Language

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Definition

Teach Me Language (Freeman and Dake 1997) is a manualized approach to treatment designed to offer providers specific activities to support language understanding and use for children with autism spectrum disorders (ASDs). It is intended to be used as part of a therapy program already in progress that is

- Increase functional knowledge and written expression: activities are designed to improve recall and communication around personal, daily events.
- Increase language-based academic concepts: activities are designed to support comprehension of academic language, address math word problems, develop dictionary skills, and increase vocabulary related to money, time, calendar concepts, and numbers.

Treatment Participants

Teach Me Language was designed for the child who has already learned basic skills in lower-level language. Children who benefit most are visual learners, and those who can participate in table activities and are relatively compliant, and able to communicate in some way (Freeman and Dake 1997). Freeman and Dake (1997) suggest that the manual should be used as a supplemental support for in-place intervention. Children who are most likely to be responsive to the *Teach Me Language* activities would have a basic receptive and/or expressive vocabulary, i.e., knowing a few animals, basic shapes, and basic colors. They should also be familiar with the numbers 1–10 and recognize the alphabet.

Treatment Procedures

A written schedule is used during treatment to outline language therapy. The authors suggest that there is no developmental sequence to the activities provided; rather, the book is organized topically and should work in the context of the child's strengths and weaknesses. Each drill is introduced, and the child is prompted to give the correct answer. Prompts are faded over time so that the child completes most activities with few to no visual and/or verbal prompts (Freeman and Dake 1997).

Efficacy Information

The exercises in the manual were designed to be effective if the child understands that the therapist

is in charge and sets the agenda. *Teach Me Language* is designed to be used as part of an in-place therapy program. There is no specific research available addressing its efficacy although the principles guiding the activities are founded in behavioral research.

Outcome Measurement

Freeman and Dake (1997) suggest that once the child understands the concept emphasized during each exercise, the difficulty level of the exercise can be adjusted. If the child is struggling with a task after much repetition, then an easier drill is introduced. Children should demonstrate a level of mastery for each task as a sign of improvement.

Qualifications of Treatment Providers

The manual was designed to be used by parents, speech-language pathologists, and/or behavioral interventionists as part of an in progress treatment program where language difficulties are being addressed (Freeman and Dake 1997). The authors structured the book so that parents with minimal experiences could implement the language activities and drills without specialized training (Freeman and Dake 1997). However, the authors reinforce that although parents with minimal experiences could use the manual, they should do so only under the guidance of a behavioral consultant or a speech-language pathologist.

See Also

► [Language Interventions](#)

References and Reading

Freeman, S., & Dake, L. (1997). *Teach me language: A manual for children with autism, Asperger's syndrome and related developmental disorders*. Langley: SKF Books.

Teacher's Aide

► Para-educator

Teaching Abduction-Prevention Skills to Children with Autism

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Definition

Abduction is defined as “any unlawful movement or detention of a child, whether this is attempted or completed” (Collie and Greene 2016; Finkelhor et al. 2002). Children may be abducted by family members or strangers. Although most of the child abductions are committed by family members (Holcombe et al. 1995; Kurt and Kutlu 2019), child abduction by strangers can lead more serious results (e.g., sexual abuse, disappearance, death) in terms of child safety (Miltenberger 2008). A stranger is defined as “a person the victim has had no apparent previous contact or interaction with” (Collie and Greene 2016; Finkelhor et al. 2002).

Mostly child abductions are carried out by persuading the child or verbally enticing the child away from safety, without using physical force (Kurt and Kutlu 2019; Poche et al. 1981). Therefore, it is critical for children to learn how to protect themselves from abduction in order to prevent child abduction cases (Godish et al. 2017). The literature mentions several types of lures used by strangers to abduct children. Lure types are classified under four categories, including simple, authority, incentive (Poche et al. 1981), and assistance (Holcombe et al. 1995; Johnson et al. 2005). The simple lure involves

the request of anyone planning abduction from the child to come with him/her without any incentive or particular reason (e.g., “Will you come with me?”). The authority lure involves convincing the child using someone (e.g., parents, teacher) who has the authority over the child to come with him/her (e.g., “Your mom is looking for you, you must come with me now!”). The incentive lure involves giving something (e.g., surprise, gift, ice cream, candy) to the child if he/she accepts to come with the person planning the abduction (e.g., “I will give you candy if you come with me”). The assistance lure involves asking for the child’s help about something (e.g., “I lost my bunny, can you help me find it?”).

The abduction-prevention skill is defined as “learning what to do when a stranger tries to lure the child to go with him/her” (Tarbox et al. 2014). Acquisition of three behaviors by the child is aimed in teaching of abduction-prevention skills. These behaviors involve (a) clearly rejecting the lures of the stranger (e.g., reacting as “no or no way!”), (b) moving away from the stranger, and (c) telling/reporting about the situation to a family member or a trusted adult (Miltenberger 2008; Poche et al. 1981). Especially, “moving away from the stranger and reporting to a trusted adult” are the most critical components of these three target behaviors (Johnson et al. 2005). The child can avoid the dangerous situation to a great extent by moving away from the person planning to abduct him/her. The child reporting the abduction to a trusted adult can serve to bring the stranger who is planning to abduct him/her or has a tendency to do such an act under control (Miltenberger 2008).

Historical Background

Historically, specialists in the field of education stated that abduction-prevention skills are among the most critical skills due to their vital importance. The main purpose of the studies on abduction-prevention for nearly 50 years has been to protect children with or without developmental disability from physical, sexual, or emotional abuse.

The preliminary experimental research on teaching abduction-prevention skills was carried out during the 1980s. In the published articles, the skills of sexual abuse-prevention and abduction-prevention were focused on simultaneously (Miltenberger and Thiesse-Duffy 1988; Poche et al. 1981, 1988). The first experimental research focusing on protecting children from sexual abuse and abduction was published by Poche et al. in 1981 under the title of “Teaching Self-Protection to Young Children” in the *Journal of Applied Behavior Analysis*. In this study, a multicomponent training package based on a behavioral approach was used to teach normally developing children abduction-prevention skills. The training package included modeling, behavior rehearsal, and social reinforcement. It was observed that participants acquired avoiding strangers and generalized this behavior. However, maintenance data were collected from two of three participants, and only one participant exhibited the target behavior the criterion level in maintenance. The training package used in this research and similar training packages using more than one application together have been called as Behavioral Skills Training (BST) in later years and today (Miltenberger 2008). The specific interventions included in BST vary slightly, but generally role-play, rehearsal, and corrective feedback are involved in BST to teach abduction-prevention skills. In another study conducted in 1988, Poche et al. compared video modeling, video modeling with behavior rehearsal, a standard safety program, and no training in teaching children abduction-prevention skills. The video modeling with behavior rehearsal intervention in this research involved the components of video modeling, role-play, corrective feedback, and praise. In this research, although the best results were found in the video model plus rehearsal condition, some children in each condition accepted the proposal of the person planning the abduction. Therefore, it can be stated that none of the interventions were effective for all of the children. One of the remarkable aspects of this research was that the appearance of strangers in the video footage was presented to resemble those of adults who had previously been

convicted of child abduction and sexual abuse. In both studies, it was stated that children were vulnerable to lures of strangers and the need for educational programs was emphasized (Poche et al. 1981, 1988).

Studies by Poche et al. have been the basis for many other studies on the teaching of sexual abuse and abduction-prevention since the late 1980s (Bergstrom et al. 2014; Collins et al. 1992a; Gast et al. 1993; Gunby et al. 2010; Gunby and Rapp 2014; Haseltine and Miltenberger 1990; Holcombe et al. 1995; Miltenberger and Thiesse-Duffy 1988; Watson et al. 1992). Thus, these studies became a model for the teaching of abduction-prevention skills to individuals with normal development as well as individuals with developmental disabilities. In the 1980s and 1990s, BST was used in almost all of the studies on teaching abduction-prevention skills to individuals with normal development or developmental disabilities (Gast et al. 1993; Haseltine and Miltenberger 1990; Poche et al. 1981).

The first experimental studies on the teaching of abduction-prevention skills to persons with developmental disabilities were published in the 1990s (Collins et al. 1992a; Gast et al. 1993; Haseltine and Miltenberger 1990; Watson et al. 1992). To summarize the findings of these studies, it can be stated that BST was generally effective in teaching abduction-prevention skills to individuals with developmental disabilities, but in some studies, this finding could not be repeated consistently for all participants, and the participants of some studies had difficulty in maintaining their abduction-prevention skills and generalizing them to social environments (Collins et al. 1992a; Gast et al. 1993; Haseltine and Miltenberger 1990). In the 1990s, descriptive studies were conducted in addition to experimental research on teaching abduction-prevention skills to individuals with developmental disabilities. Descriptive studies showed that teachers and parents expressed that the skill of “resisting the lures of strangers” was a critical safety skill (Collins et al. 1991, 1992b). In 2000s, it was stated that learning abduction-prevention skills is vital for all children, but not enough attention was given to developing safety

skills training programs despite child abduction statistics (Johnson et al. 2005).

The first study aimed at teaching abduction-prevention skills for participants with autism was published in 2010 (Gunby et al. 2010). In this study and most of other experimental studies (Akmanoglu and Tekin-Iftar 2011; Bergstrom et al. 2014; Gunby and Rapp 2014; Gunby et al. 2010; Ledbetter-Cho et al. 2016), BST was used. To summarize the findings of these studies published since 2010, it can be stated that BST was effective in teaching abduction-prevention skills to children with ASD and that the participants of these surveys generally maintained target behaviors after the intervention ended and were able to generalize to conditions other than teaching conditions. In addition, there is no significant difference in the way BST is applied in the teaching of abduction-prevention skills to individuals with developmental disabilities, whether ASD or not, and normally developing individuals. BSTs, which consist of multi-component teaching packages used for teaching abduction-prevention skills to individuals with ASD, generally included applications such as instructions, modeling, rehearsal, and feedback (Bergstrom et al. 2014; Gunby et al. 2010; Ledbetter-Cho et al. 2016). However, in two studies published to date, video modeling (Godish et al. 2017) and social stories (Kurt and Kutlu 2019) were used instead of a multi-component teaching package. The findings of these two studies are similar to those of studies investigating the effectiveness of BST in teaching abduction-prevention skills to children with ASD. In other words, according to the findings of these two studies, video model teaching and social stories were also effective in teaching abduction-prevention skills to children with ASD.

As a result, the common element of published research on teaching abduction-prevention skill has been to teach children to recognize the lures of people who want to abduct them, to reject their offers, to move away from their environment, and to inform a trusted adult to control the situation. Teaching practices based on applied behavior analysis were mostly used in teaching these skills.

Current Knowledge

Millions of children disappear every year in the world ("International Centre for Missing and Exploited Children" 2019). According to a report published by the US National Crime Information Center (NCIC) in 2018, there were 424,066 entries for missing children in the United States. About 1% of these individuals had physical or intellectual disabilities. According to the same report, 4925 of these missing children may have been abducted ("Federal Bureau of Investigation" 2019).

The inability of children to protect themselves from abductors may facilitate their abduction. Unfortunately, children with disabilities are at a greater risk of being abducted compared with normally developing children (Godish et al. 2017). The difficulties or delays in communication skills and inabilities in attention, motor control, and cognitive skills that are experienced by children especially with ASD might increase the risk of getting harmed by their environment (Wiseman et al. 2017). Another risk factor is that almost 50% of children with ASD tend to elope from the safe area (Anderson et al. 2012). This situation increases the risk of harm and abduction of children with ASD. Moreover, children with ASD may not be able to distinguish strangers from acquaintances or may be prone to persuasion by strangers due to their inadequacy in social skills (Gunby et al. 2010). Recent studies have shown that children with ASD are more likely to believe and be abducted by lures offered by strangers (Akmanoglu and Tekin-Iftar 2011; Bergstrom et al. 2014; Godish et al. 2017; Gunby and Rapp 2014; Gunby et al. 2010; Kurt and Kutlu 2019; Ledbetter-Cho et al. 2016).

In the literature, the most commonly used method for teaching abduction-prevention skills to children with ASD is training packages, often called BST, where multiple applications are used together. Such a training package can be implemented as in the following example: Firstly, verbal instructions are given (briefly discuss/review the lures and safety response with participant); in other words, the practitioner

informs the participant about the behaviors to be exhibited (e.g., “If a stranger tries to get you to go with him/her, say ‘no,’ move away, and then tell your mom”). Secondly, the practitioner models the correct behavior (e.g., “Practitioner says “No” to abductor, moves away from him/her, and reports the situation to a trusted adult”). Thirdly, role-playing is carried out. In role-playing, the practitioner pretends to be an abductor with the child (“Let’s pretend we are at the supermarket and I’m a stranger”) (Bergstrom et al. 2014). Finally, praise (e.g., thank you for telling me) or corrective feedback is used depending on the child’s performance. Corrective feedback is used when the child does not exhibit the correct behavior at the criterion level (e.g., “You need to walk away from her”). BST training packages might also involve systematic clue and fading processes such as graduated guidance (Akmanoglu and Tekin-Iftar 2011) and constant time delay (Bergstrom et al. 2014). Another application that has been used so far in teaching abduction-prevention skills to children with ASD is video modeling. Video modeling was used alone in one study (Godish et al. 2017) and in combination with other practices within BST in some studies (Gunby et al. 2010; Gunby and Rapp 2014; Ledbetter-Cho et al. 2016) to teach abduction-prevention skills to children with ASD. In the studies using video modeling, video images of abduction-prevention skills exhibited by peer models were shown to children with ASD, and then the participant was given the opportunity to exhibit the same behavior. In a recent study on teaching abduction-prevention skills to children with ASD, social stories were also used (Kurt and Kutlu 2019). In this study, the lure types mentioned in the literature and the expected behaviors of the child with ASD were expressed in short sentences in the story, and these sentences were supported by photographs with peer models (Kurt and Kutlu 2019). An example sentence of a social story about target behavior can be written as follows: “When a person I don’t know wants me to go with him/her by saying that he/she will give me candy, I should go away by saying “no” to him and then tell my mom about it.”

Teaching Abduction-Prevention Skills to Children with Autism, Table 1 An example of scoring charts for teaching abduction prevention skills

Behavior	Score
Agreed to leave with the abductor	0
Did not agree to leave but failed to say “no”	1
Said “no” but did not leave or report the incident	2
Said “no” and left the area but did not report the incident	3
Said “no,” left the area, and immediately reported the incident	4

In published studies, scoring charts were generally used to evaluate the performance of children with ASD on abduction-prevention skills (Johnson et al. 2005). An example of such a chart is presented in Table 1 (Gunby and Rapp 2014).

The following recommendations may be taken into account in the organization of settings and planning of the teaching process for teaching abduction-prevention skills to children with ASD: Firstly, the types of lures (assistance, simple, incentive, authority) mentioned in the literature should be taken into consideration when evaluating the performance of children with ASD on abduction-prevention skills. Children with ASD may be overly sensitive to certain objects, foods, or activities (e.g., going to pizza, playing the piano). Various data collection methods (e.g., family interviews, observations) can identify objects, foods, or activities that children with ASD may be susceptible to. In this way, the performance of children with ASD in terms of incentive lure type can be determined better. Secondly, children with ASD may have difficulty in generalizing their newly learned skills to different settings and people (Dufour and Lanovaz 2017). Therefore, the implementation of efforts on teaching abduction-prevention skills to children with ASD in social settings may facilitate the generalization of these skills. Finally, children with ASD can often perceive visual stimuli more easily than auditory and social stimuli (Nikopoulos and Keenan 2006). Thus, visual aids can be utilized in the teaching programs related to abduction-prevention skills.

Future Directions

Despite its vitality, a limited number of studies have been conducted on teaching abduction-prevention skills to children with ASD. Therefore, the search for the development of more effective and efficient practices for teaching these vital skills to children with ASD should be continued. Future studies might aim at determining and using more efficient practices that can result in better learning. Better learning means that one particular teaching practice results in faster acquisition, higher generalization, and more comprehensive learning than the other (Tekin-Iftar and Kircaali-Iftar 2013; Wolery et al. 1991). In further research, studies can investigate which of the applications used in teaching abduction-prevention skills to individuals with ASD can be more effective and efficient.

Almost all of the published studies used BST, which was created by combining multiple applications, and children with ASD often learned socially valid and important target skills in these studies. Only two studies used social stories and video modeling instead of BST consisting of multicomponent training packages (Godish et al. 2017; Kurt and Kutlu 2019). However, the findings of these studies should be supported by other studies in order to generalize them. Although BST applications, which consist of multicomponent training packages, generally seem to be effective, studies comparing various teaching practices with each other can be planned in order to determine the teaching practices that can be used more effectively and efficiently in teaching abduction-prevention skills to children with ASD. Faster, less time, fewer teaching sessions, less error response rates, and less practitioner preparation have been used as productivity parameters in a large number of researches published so far (Tekin-Iftar and Kircaali-Iftar 2013; Wolery et al. 1991). Therefore, these efficiency parameters can also be used to determine which teaching practices are more efficient in teaching abduction-prevention skills. Moreover, although it has been determined by various studies to be effective, in some studies the BST packages required the use of intrusive adult prompts and more training time

and a higher level of expertise for practitioners (Mancil et al. 2009). Further studies may aim to determine the applications that can be used easily and effectively by nonspecialists (e.g., siblings, parents, caregiver) who are less intrusive to children with ASD.

To prevent the abduction of children with ASD by family members may be planned in future studies. In addition to strangers and family members, children can be abducted by people they know less (Finkelhor et al. 2002). Examples of slight acquaintances include the neighbors that the child never talked to before, house cleaner, a person that the child interacted online for less than a month, or a friend of a friend who once babysat for the victim. Therefore, it is important to conduct studies to protect children with ASD from strangers and family members as well as lures of slight acquaintances.

In studies focused on teaching abduction-prevention skills, children with ASD were taught to protect themselves from strangers who do not use physical force. However, children with ASD can also be abducted by force. Forced abduction can be carried out by strangers, family members, or slight acquaintances. New digital technology tools can be used to prevent situations where children with ASD can be abducted by force. For example, devices with GPS (Global Positioning System) (e.g., tablets, smartphones, smart watches) can show where children are when they are lost or kidnapped. In further research, the use of such technological tools to prevent the abduction of children with ASD can be studied. For example, children with ASD may be instructed to secretly activate the GPS use of smart watches during abduction. Another technology used in the teaching of children with ASD is virtual reality. Virtual reality can reduce the difficulties involved in providing the necessary environments and the sufficient number of people to play the role of strangers, when teaching abduction-prevention skills especially in social settings. Therefore, in further research, the use of virtual reality in teaching abduction-prevention skills can be studied. Moreover, the performance of children with ASD in real environments can be compared to that of virtual reality environments.

In the studies focusing on the teaching of abduction-prevention skills, practitioners have been professionals, and no study has been found in which parents or other family members participate in the teaching process. Participation of family members in the teaching process contributes to the maintenance and generalization of children's acquired skills at home and thus improves the quality of the process. Previous studies show that parents are able to teach many skills to their children, including self-help skills, communication skills, imitation skills, social skills, and joint attention skills (Besler and Kurt 2016). Therefore, family education studies aiming to teach abduction-prevention skills to children with ASD can be carried out, and studies on the effectiveness of teaching offered by family members can be performed.

In addition to children with ASD, parents may be affected by abduction and teaching sessions planned to teach abduction-prevention skills. In particular, children may feel more scared, cautious, or upset, as they are exposed to abduction trials in the experimental process of research (Godish et al. 2017). Therefore, it may be important to collect social validity data from both children with ASD and family members and to take into consideration the findings obtained from these studies in order to evaluate such effects (Callahan et al. 2017).

See Also

- [Safety](#)
- [Stranger Anxiety](#)

References and Reading

- Akmanoglu, N., & Tekin-Iftar, E. (2011). Teaching children with autism how to respond to the lures of strangers. *Autism, 15*, 205–222.
- Anderson, C., Law, J. K., Daniels, A., Rice, C., Mandell, D. S., Hagopian, L., & Law, P. A. (2012). Occurrence and family impact of elopement in children with autism spectrum disorders. *Pediatrics, 130*, 870–877.
- Bergstrom, R., Najdowski, A. J., & Tarbox, J. (2014). A systematic replication of teaching children with autism to respond appropriately to lures from stranger. *Journal of Applied Behavior Analysis, 47*, 1–5.
- Besler, F., & Kurt, O. (2016). Effectiveness of video modeling provided by mothers in teaching play skills to children with autism. *Educational Sciences: Theory & Practice, 16*, 209–230.
- Callahan, K., Hughes, H. L., Mehta, S., Toussaint, K. A., Nichols, S. M., Ma, P. S., Kutlu, M., & Wang, H. (2017). Social validity of evidence-based practices and emerging interventions in autism. *Focus on Autism and Other Developmental Disabilities, 32*, 188–197.
- Collie, C. J. R., & Greene, K. S. (2016). The effectiveness of victim resistance strategies against stranger child abduction: An analysis of attempted and completed cases. *Journal of Investigative Psychology and Offender Profiling, 13*, 277–295.
- Collins, B. C., Wolery, M., & Gast, D. L. (1991). A survey of safety concerns for students with special needs. *Education and Training in Mental Retardation, 26*, 305–318.
- Collins, B. C., Schuster, J. W., & Nelson, C. M. (1992a). Teaching a generalized response to the lures of strangers to adult with severe handicaps. *Exceptionality, 3*, 67–80.
- Collins, B. C., Wolery, M., & Gast, D. L. (1992b). A national survey of safety concerns for students with special needs. *Journal of Developmental and Physical Disabilities, 4*, 263–276.
- Dufour, M., & Lanovaz, M. J. (2017). Comparing two methods to promote generalization of receptive identification in children with autism spectrum disorders. *Developmental Neurorehabilitation, 20*, 463–474.
- Federal Bureau of Investigation. (2019). Retrieved August 19, 2019 from <https://www.fbi.gov/file-repository/2017-ncic-missing-person-and-unidentified-person-statistics.pdf/view>
- Finkelhor, D., Hammer, H., & Sedlak, A. J. (2002). Nonfamily abducted children: National estimates and characteristics. NISMART: *National incidence studies on missing, abducted, runaway, and throwaway children*. Washington, DC: U.S. Department of Justice.
- Gast, D. L., Collins, B. C., Wolery, M., & Jones, R. (1993). Teaching preschool children with disabilities to respond to the lures of strangers. *Exceptional Children, 59*, 301–311.
- Godish, D., Miltenberger, R., & Sanchez, S. (2017). Evaluation of video modeling for teaching abduction prevention skills to children with autism spectrum disorder. *Advances in Neurodevelopmental Disorders, 1*, 168–175.
- Gunby, K. V., & Rapp, J. T. (2014). The use of behavioral skills training and in situ feedback to protect children with autism from abduction lures. *Journal of Applied Behavior Analysis, 47*, 856–860.
- Gunby, K. V., Carr, J. E., & Leblanc, L. A. (2010). Teaching abduction-prevention skills to children with autism. *Journal of Applied Behavior Analysis, 43*, 107–112.

- Haseltine, B., & Miltenberger, R. G. (1990). Teaching self-protection skills to persons with mental retardation. *American Journal of Mental Retardation*, 95, 188–197.
- Holcombe, A., Wolery, M., & Katzenmeyer, J. (1995). Teaching preschoolers to avoid abduction by strangers: Evaluation of maintenance strategies. *Journal of Child and Family Studies*, 2, 177–191.
- International Centre for Missing and Exploited Children. (2019). Retrieved August 10, 2019 from <http://www.icmec.org>
- Johnson, B. M., Miltenberger, R. G., Egemo-Helm, K., Jostad, C. M., Flessner, C., & Gatheridge, B. (2005). Evaluation of behaviour skills training for teaching abduction-prevention skills to young children. *Journal of Applied Behavior Analysis*, 38, 67–78.
- Kurt, O., & Kutlu, M. (2019). Effectiveness of social stories in teaching abduction-prevention skills to children with autism. *Journal of Autism and Developmental Disorders*, 49, 3807–3818.
- Ledbetter-Cho, K., Lang, R., Davenport, K., Moore, M., Lee, A., O'Reilly, M., Watkins, L., & Falcomata, T. (2016). Behavioral skills training to improve the abduction-prevention skills of children with autism. *Behavior Analysis in Practice*, 9, 266–270.
- Mancil, G. R., Haydon, T., & Whitby, P. (2009). Differentiated effects of paper and computer-assisted social stories on inappropriate behavior in children with autism. *Focus on Autism and Other Developmental Disabilities*, 24, 205–215.
- Miltenberger, R. G. (2008). Teaching safety skills to children: Prevention of firearm injury as an exemplar of best practice in assessment, training, and generalization of safety skills. *Behavior Analysis in Practice*, 1, 30–36.
- Miltenberger, R., & Thiesse-Duffy, E. (1988). Evaluation of home-based programs for teaching personal safety skills to children. *Journal of Applied Behavior Analysis*, 21, 81–87.
- Nikopoulos, C., & Keenan, M. (2006). *Video modeling and behaviour analysis*. London: Jessica Kingsley Publishers.
- Poche, C., Brouwer, R., & Swearingen, M. (1981). Teaching self-protection to young children. *Journal of Applied Behavior Analysis*, 2, 169–176.
- Poche, C., Yoder, P., & Miltenberger, R. (1988). Teaching self-protection to children using television techniques. *Journal of Applied Behavior Analysis*, 21, 253–261.
- Tarbox, J., Persicke, A., & Bergstrom, R. (2014). Adaptive. In D. Granpeesheh, J. Tarbox, A. C. Najdowski, & J. Kornack (Eds.), *Evidence-based treatment for children with autism the card model* (pp. 241–260). Waltham: Academic.
- Tekin-Iftar, E., & Kircaali-Iftar, G. (2013). *Ozel egitimde yanlisiz ogretim yontemleri [Errorless teaching procedures in special education]* (2nd ed.). Ankara: Vize Yayincilik.
- Watson, M., Bain, A., & Houghton, S. (1992). A preliminary study in teaching self-protective skills to children with moderate and severe mental retardation. *The Journal of Special Education*, 26, 181–194.
- Wiseman, K. V., McArde, L. E., Bottini, S. B., & Gillis, J. M. (2017). A meta-analysis of safety skill interventions for children, adolescents, and young adults with autism spectrum disorder. *Review Journal of Autism and Developmental Disorders*, 4, 39–49.
- Wolery, M., Doyle, P., Ault, M., Gast, D., Meyer, S., & Stinson, D. (1991). Effects of presenting incidental information in consequent events on future learning. *Journal of Behavioral Education*, 1, 79–104.

Teaching Assistant

► Para-educator

Teaching Interaction Procedure

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Definition

The teaching interaction procedure (TIP) is an applied behavior analysis (ABA)-oriented, systematic instructional procedure of the modeling, practice, and feedback sort. While it, at its core, does contain the basic elements (instruction, opportunity to perform the behavior, and feedback) of discrete trial teaching, the TIP contains additional components, is more complex, and is delivered in a more natural conversational and interactive manner. It has been utilized for nearly four decades with individuals with autism spectrum disorder (ASD) but has been empirically validated only in the last decade. It can be utilized to teach a variety of skills but is especially suited for instruction in the social area.

Historical Background

The TIP was developed in the early 1970s as part of the teaching family model for court-adjudicated

youth (Phillips 1968). It was based on the instructional style used by Lonnie and Elaine Phillips, who served as the original teaching parents in the first community-based ABA-oriented treatment program for delinquent youth, achievement place. Subsequent research showed it to be an effective technique for addressing the skill deficits of that population (e.g., Minkin et al. 1976).

In the late 1970s, it was brought by the author to the UCLA Young Autism Project and has been utilized in home, clinic, school, community, residential, and vocational settings to teach a variety of skills to children, adolescents, and adults on the autism spectrum in the years since.

Rationale or Underlying Theory

Years of field testing as well as a growing body of empirical support have shown that the TIP can be an effective means for developing competencies, in persons with ASD. The nature and components of the procedure allow for instruction in skills from the simple to the complex and sophisticated. Further, as it constitutes a naturalistic and dignifying teaching technique, it has been found to promote independent skill utilization, generalization, and consumer satisfaction.

Goals and Objectives

Often used in conjunction with motivational arrangements (e.g., token economies), the TIP is designed to help address skill deficits (e.g., social, play/leisure, communication, vocational, self-help) in the repertoires of individuals on the autism spectrum. It can also be effectively used to teach replacement and alternate skills to the behavioral excesses (e.g., noncompliance, poor frustration tolerance, negative attention seeking) common to ASD. The ultimate goal of the TIP is to produce meaningful skills, utilized independently in everyday, naturally occurring situations.

Treatment Participants

Because of the interactive and conversationally oriented nature of the TIP, it is typically used with those at the upper end of the autism spectrum. At least moderate communicative and social interaction skills are necessary for adequate participation in the TIP. Further, cognitive capacity of participants allowing for the understanding of such concepts as cause and effect and the relationship between role play practice and the ultimate utilization of skills are typically helpful. The procedure has often been used with young children but, because of its dignifying nature, is particularly fitting for adolescents and adults.

Treatment Procedures

The multistep TIP consists of the following components.

Label and Identification

The first step of the TIP is the labeling and identification of the target behavior or skill. Identification and labeling of a skill involve noting what is being taught and giving it a name. The purpose of this step is to make sure that the learner clearly understands what skill is being taught. Further, having a clear name for the skill can help when priming is used and makes for clear feedback. Several things characterize a good label. The label should be clear. It should capture the skill being taught and do so in as few words as possible. Also, it should consist of language that fits the age level and peer culture of the learner. When identifying the behavior, it is good practice to define what the behavior is and what it is not. Further, such identification should clearly indicate where and when the skill is relevant or an issue. Identification can relate the skill to the student's past history and personal experience. Past or present occurrences or issues in the child's life that point to the area of need can be included (e.g., "Remember when your classmate offered to give you help? Well, we want to work on what to do when that happens.").

Rationale

The second, and a unique component of the TIP, is the provision of a meaningful rationale. A rationale is not just a reason to engage in the behavior but rather naturally occurring, fundamental, and germane to the learner's purpose for engaging in the behavior. ABA often discusses the natural consequences of a behavior, such as when it is recommended that artificial reinforcers (e.g., tokens) be faded to natural, maintaining consequences. These natural consequences should form the substance of rationales. A good rationale includes naturalistic and realistic outcomes and meets the learner's personal needs and desires. This can be the first step in connecting the learner to the natural consequences that will eventually maintain the behavior. This connection will happen once the learner is good enough at the skill being taught to actually achieve those outcomes.

With rationales, learners are more likely to accept instruction. Further, they begin to recognize that they have influence over the outcomes they experience. Good rationales help the learner anticipate outcomes and develop a sense of cause and effect. With time and some teaching, some learners even begin to develop meaningful rationales themselves.

Description/Demonstration

The third component of the TIP is the description and demonstration of the skill being taught. Typically, given the complexity of skills often targeted, only part of a skill is taught in a single TIP session. It is very common that adjustments have to be made in the size or complexity of the segment, once the TIP begins.

The teacher should describe the segment being taught, including all necessary elements. This information might include what is being said and how it is being said – that is, facial expressions, voice tone, gaze, and body language. It may include overt actions, supportive elements (e.g., use of stress management tools such as taking deep breaths), and internal, cognitive pieces (e.g., self-talk or self-instructions). Descriptions should be detailed and

clear. They should also contain understandable, student-friendly language.

In the demonstration portion, the teacher models for the learner all of the components contained within the description. Modeling provides an exact demonstration of the behavior, or component, to be learned. Demonstrations are usually in vivo (happening live in front of the learner) but can be written, pictorial, or consist of video modeling.

Practice

The next step of the TIP is the practice or role play portion. It is critical to learning to make sure that the student has sufficient opportunity to practice the skill and that practice continues until the skill is mastered. What is practiced follows the description/demonstration. That is, attempts should be made to have the learner fully practice what has been modeled.

The student role plays the skill (or, more likely, the portion of the skill) being taught, with the teacher as partner, in a simulation. The teacher should design this step of the TIP in a way that will increase the chance that the student's practice will be successful. Therefore, the practice is most often initially removed from natural circumstances (hence it is role play) and setup in a way that will minimize the obstacles to successful performance. The teacher has control over the form and extent of the practice through setup and planning and by how he or she plays the other party in the role play. In this way, statements, styles, complexity, challenges, emotional content, and other factors can be adjusted either up or down. In time, effort is directed at working the fully learned skill into application in the natural environment.

Feedback

Without feedback, indicating what was correctly performed and what was less than adequate, practice may not result in progress. Without feedback, practice can be an exercise in repetition of mistakes and can result in frustration.

Feedback can be given throughout the TIP. Such feedback often focuses on the student's behavior and quality of their responses during the TIP. For example, this can include his or her cooperation, effort, calmness, participation, processing, persistence, patience, and attention.

The feedback step of the TIP, however, follows the practice. In it, the teacher provides specific feedback on the student's performance, both positive and corrective. The key is specificity: information on how the learner performed all (or as many as possible) of the components that were described and demonstrated should be provided. Positives should be sincere, accurate, and specific. Corrective feedback should be unambiguous, yet supportive and constructive.

Feedback may cycle back to the description/demonstration portion of the TIP as indicated. Opportunities for additional practice (with additional feedback) often follow. There may even be a return to the rationale, with reasons given for the correct performance and for the inclusion of the particular element being retaught. Typically, this would then be followed by additional modeling and practice as indicated.

Optional External Consequence

Often the feedback segment of the TIP is accompanied or followed by the giving of external reinforcement. Such reinforcement may take the form of tokens; direct reinforcement with items, activities, or opportunities; use of a contingency contract; or provision of other artificial motivators. It could involve reinforcement of correct performance of the skill being taught and/or appropriate learning behaviors as noted above.

The provision of external consequences may serve important motivational functions. While praise and positive feedback may have some value for the student with ASD, they may not be sufficient to motivate participation, performance, and skill acquisition. Without sufficient motivation, learning and application of new skills may not occur. Conversely, without skill instruction, all of the motivation in the world is not likely to result in changed performance. Systematic

instruction and consequences are two mutually facilitative and essential components of any ABA teaching procedure. Using external, meaningful consequences can help ensure that the student is motivated to learn and to eventually engage in the targeted skill in naturally occurring situations.

Given their potential benefits, external consequences can be a customary part of the TIP. The application of this component, like all good ABA, depends on the student, what has unfolded during the teaching, and flexible decision-making on the part of the teacher. In time, the goal is to fade the external consequences. This would occur when natural positive consequences (those outcomes introduced when rationales were provided) begin to control and support the utilization of the new skill.

A more extensive description of the TIP is provided in Taubman et al. (2011) and in Cihon et al. (2017).

Efficacy Information

Leaf et al. (2009) provided the first empirical evaluation of the TIP with individuals diagnosed with ASD. That study demonstrated that the TIP was effective for teaching all of its young participants targeted social skills; however, no generalization measures were collected, and maintenance measures were variable. In a follow-up study, Leaf et al. (2010) demonstrated the effectiveness of the TIP in teaching social skills within a group instructional format for individuals, ages ranging from 4 years old to 6 years old, diagnosed with ASD. Leaf et al. (2012) and Kassardjian et al. (2014) in studies comparing the relative effectiveness of the TIP to Social Stories™ both showed the TIP to be more effective and more facilitative of generalization when used to teach individuals with ASD social skills. Kassardjian et al. (2013) showed the successful generalization of social skills taught using the TIP to the natural environment with four individuals with ASD, ages ranging 4–13 years old. Ferguson et al. (2013) effectively used the TIP with video games to teach six children diagnosed with ASD, ages

7–11 years old, team sportsmanship skills and demonstrated generalization of skills taught. Peters et al. (2016) extended Leaf et al. (2010) by demonstrating the effectiveness of the TIP implemented in a group instructional format to improve social skills for four individuals, ages 8–10 years old, diagnosed with ASD. They also showed the maintenance over time of acquired skills and the social validity of the TIP. Ng et al. utilized a modified version of the TIP to effectively teach social skills to four children diagnosed with ASD and an intellectual disability. Finally, Green et al. (2019) illustrated the effective use of the TIP to train staff in the implementation of the TIP with children with ASD.

Outcome Measurement

Results of the use of the TIP can best be assessed through the use of observational measures. For such measurement, brief but clear definitions of appropriate or successful performance (again including all aspects) of the elements, segments, or components of the targeted skill should be created (often referred to as a task analysis). Measures typically involve the recording of the number of elements or segments of the targeted skill successfully learned or performed. A percentage reflecting the number of elements correctly performed divided by the total number of segments is then reported. While such measurement can certainly occur during contrived or training arrangements, it is suggested that ultimately, non-intrusive observation of independent skill performance should occur in natural everyday social situations. Only then can meaningful, true mastery, and actual, independent skill utilization be determined.

Qualifications of Treatment Providers

Those using the TIP should be well trained in the procedure. The training should include didactic instruction on the components of the TIP and their purposes as well as experiential training on its implementation. Training should not only

include application of the steps of the TIP but the manner in which they are delivered. Given the nature of the TIP and the capacity of those typically receiving the procedure, stylistic elements, flexibility in delivery, and concurrent rapport building are particularly important for instructional success. Accompanying training on the TIP with supportive background information on ABA and its basic principles and concepts can provide context and also be helpful.

See Also

► [Progressive Applied Behavior Analysis](#)

References and Reading

- Cihon, J. H., Weinkauff, S. M., & Taubman, M. (2017). Using the teaching interaction procedure to teach social skills for individuals diagnosed with autism spectrum disorder. In J. B. Leaf (Ed.), *Handbook of social skills and autism Spectrum disorder : Assessment, curricula, and intervention* (pp. 313–323). AG Switzerland: Springer International Publishing.
- Ferguson, B. R., Gillis, J. M., & Sevlever, M. (2013). A brief group intervention using video games to teach sportsmanship skills to children with autism spectrum disorders. *Child & Family Behavior Therapy*, 35(4), 293–306.
- Green, D. R., Ferguson, J. L., Cihon, J. H., Torres, N., Leaf, R., McEachin, J., et al. (2019). The teaching interaction procedure as a staff training tool. *Behavior Analysis in Practice*. <https://doi.org/10.1007/s40617-019-00357-2>.
- Kassardjian, A., Rudrud, E., Taubman, M., Leaf, J. B., Edwards, A., Schulze, K., et al. (2013). Utilizing teaching interactions to facilitate social skills in the natural environment. *Education and Training in Autism and Developmental Disabilities*, 48(2), 245–257.
- Kassardjian, A., Leaf, J. B., Ravid, D., Leaf, J. A., Alcalay, A., Dale, S., et al. (2014). Comparing the teaching interaction procedure to social stories: A replication study. *Journal of Autism and Developmental Disorders*, 44(9), 2329–2340.
- Leaf, J. B., Taubman, M., Bloomfield, S., Palos-Rafuse, L., Leaf, R., McEachin, J., & Oppenheim, M. L. (2009). Increasing social skills and pro-social behavior for three children diagnosed with autism through the use of a teaching package. *Research in Autism Spectrum Disorders*, 3(1), 275–289.
- Leaf, J. B., Dotson, W. H., Oppenheim, M. L., Sheldon, J. B., & Sherman, J. A. (2010). The effectiveness of a group teaching interaction procedure for teaching social skills to young children with a pervasive

- developmental disorder. *Research in Autism Spectrum Disorders*, 4(2), 186–198.
- Leaf, J. B., Oppenheim-Leaf, M. L., Call, N. A., Sheldon, J. B., Sherman, J. A., Taubman, M., et al. (2012). Comparing the teaching interaction procedure to social stories for people with autism. *Journal of Applied Behavior Analysis*, 45(2), 281–298.
- Minkin, N., Braukmann, C. J., Minkin, B. L., Timbers, G. D., Timbers, B. J., Fixsen, D. L., et al. (1976). The social validation and training of conversational skills. *Journal of Applied Behavior Analysis*, 9(2), 127–139.
- Ng, A. H. S., Schulze, K., Rudrud, E., & Leaf, J. B. (2016). Using the teaching interaction procedure to teach social skills to children with autism and intellectual disability. *American Journal on Intellectual and Developmental Disabilities*, 121(6), 501–519.
- Peters, B., Tullis, C. A., & Gallagher, P. A. (2016). Effects of a group teaching interaction procedure on the social skills of students with autism spectrum disorders. *Education and Training in Autism and Developmental Disabilities*, 51(4), 421–433.
- Phillips, E. L. (1968). Achievement place: Token reinforcement procedures in a home-style rehabilitation setting for “pre-delinquent” boys. *Journal of Applied Behavior Analysis*, 1(3), 213–223.
- Taubman, M. T., Leaf, R. B., & McEachin, J. (2011). *Crafting connections: Contemporary applied behavior analysis for enriching the social lives of persons with autism spectrum disorder*. New York: DRL Books.

skills considers the developmental and cognitive level of the child, and instruction can begin with very young children.

Historical Background

Childhood sexual abuse is a serious issue in the United States (USA). The latest figures estimate that approximately 57,964 children were confirmed victims of sexual abuse last year in the USA (US Department of Health and Human Services 2019). According to results from three administrations of the National Survey of Children’s Exposure to Violence, Finkelhor et al. (2014) reported an estimated prevalence of 27% for females and 5% for males, or approximately 1 in 4 girls and 1 in 20 boys. The results of CSA on youth are far reaching and can cause a multiplicity of emotional and behavioral problems into adulthood (Pérez-Fuentes et al. 2013). While all children are at risk of victimization, there are some characteristics that make certain children more vulnerable to sexual assault. One of those characteristics is the disability that children may experience, such as autism spectrum disorder (ASD). Certain social-emotional and communication challenges, when present in children with ASD, may be interpreted by sexual offenders as vulnerabilities that they can exploit (Edelson 2010). Research has confirmed that children with disabilities are 2–3 times more likely to be sexually abused (including raped) than children without disabilities (Brown-Lavoie et al. 2014; Sullivan and Knutson 2000). Mandell et al. (2005) found that 16.6% of caregivers reported their child with ASD had been sexually abused. Brown-Lavoie et al. (2014) reported that 78% of their respondents with ASD reported at least one instance of sexual victimization, compared to 47% of a comparison group. There appears to be certain characteristics that children with ASD possess that may make them more vulnerable to abuse including lack of communication, limited understanding of personal boundaries, and difficulty interpreting others’ intentions. Muccigrosso (1991) discusses other attributes of children with disabilities that may put them

Teaching Personal Safety

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Definition

Teaching personal safety skills involves educating children about their private body parts, boundaries with others, personal privacy, and ability to say “no” to touches they do not want or are inappropriate. Conceptualized as part of childhood sexual abuse (CSA) prevention, educating youth about personal safety typically consists of teaching children knowledge and skills related to recognizing, responding to, and reporting potentially abusive situations (three R’s of prevention). Teaching personal safety

at increased risk including lack of knowledge of abuse, socialized to be over compliant, limited or no assertiveness/refusal skills and a potentially restricted range of socialization experiences. The later may lead them to accept professed affection/attention from people who may exploit them sexually.

In response to the growing body of knowledge regarding the scope and consequences of CSA, prevention programs were developed in the late 1970s and widely disseminated in the early to mid-1980s (Wurtele and Kenny 2010). The primary focus of CSA prevention efforts has been to educate children about how to protect themselves through group-based instruction on personal safety. These efforts have traditionally taken place in educational settings, as schools' primary function is to inform and educate (Wurtele and Kenny 2010). These school-based programs typically teach children safety rules, body ownership, private parts of the body, distinguishing types of touches and secrets, who to tell, and where to go for help. A systematic review by Walsh et al. (2015) found that these programs assisted children in learning behaviors that would help them avoid sexual abuse encounters, and that participants remembered what they learned up to 6 months after the program. In addition, participation did not increase fear or anxiety among participants, and those who participated were more likely to tell an adult about abuse compared to those who did not participate in a program. While these programs have existed for some time, there has been little focus on development of programs for children with disabilities, including those with ASD.

Current Knowledge

Prevention programs have historically not made accommodations or addressed children with disabilities, and limited research exists on CSA prevention programs with individuals with developmental disabilities (Lee and Tang 1998; Lumley et al. 1998). There have been a few studies, mostly conducted in the 1990s, using personal safety skills programs with children

with disabilities, but not specifically ASD (see Kim 2010), and only a small number of programs have received empirical support. There are even fewer programs that specifically target the parents of youth with ASD to assist with prevention education and little empirical evidence exists for these programs also. Given the lack of research with children with ASD and personal safety, a few personal safety programs for individuals with intellectual or other disabilities will be examined for potential generalization. It should be noted that in a review of programs that teach abuse protection skills to individuals with disabilities, only one utilized children while all other programs focused on adults (see Doughty and Kane 2010).

There are a few documented cases of teaching body safety skills to youth with disabilities and they have primarily utilized behavioral approaches. Lee and Tang (1998) used Behavioral Skills Training (BST; Wurtele 1986) with Chinese adolescents with mild mental deficits. BST has been empirically supported and is recognized by the California Evidence-Based Clearing House Practices as having promising research evidence as a primary prevention program for abuse. BST includes components such as instructions, modeling, rehearsal, praise, social reinforcement, shaping, and corrective feedback. The teens participated in two 45-min sessions where they practiced discriminating between appropriate and inappropriate touch requests and were taught four self-protection skills: verbal response (e.g., say "No!" in a loud voice), motoric responses (e.g., try to get away or remove yourself from the situation), tell some trusted persons (e.g., parents, teachers) about the incident, and report the person and situation concerning the sexual advance (Lee and Tang 1998). The teens evidenced increases in recognition of appropriate touch and did not over generalize appropriate touches to necessary medical or hygiene touches. They also demonstrated better understanding of sexual abuse issues (e.g., being boss of one's own body, knowing that adults' inappropriate touching of a child is never the fault of a child, touching an adult's private parts is wrong, touching own private parts is acceptable) and differentiating between

inappropriate and appropriate touch situations. Compared to a control group, they were also significantly more likely to verbally refuse inappropriate sexual advances, remove themselves from the abusive situations, tell a resource person about the inappropriate situations and relay what had happened to the resource person. Since some gains were not maintained at 2 months, the authors recommend booster sessions with an emphasis on the nonverbal refusal skill of physically removing oneself from the abusive situations. BST was also implemented with a young boy with ASD, and he made gains in knowledge of personal safety including appropriate touches (Kenny et al. 2013). His caretakers participated in a simultaneous program aimed at helping them create a safe environment for him and review the prevention concepts at home.

Moving beyond youth, Miltenberger et al. (1999) provided ten behavioral skills training sessions (1 h a week) followed by in situ training to five adult females with mild or moderate intellectual disabilities. Training involved presentation of information about sexual behavior and sexual abuse, learning to discriminate sexual abuse from harmless situations, instructions in the use of the sexual abuse prevention skills in response to a sexual solicitation from a staff person, rehearsal of the skills in role plays of a sexual solicitation, praise for correct performance and corrective feedback as needed, and the use of multiple exemplars of sexual solicitations in the role play. The sexual abuse prevention skills taught were not complying with the request, saying “no” (or its equivalent), leaving the area, and reporting the incident to an authority figure. Behavioral skills training resulted in abuse prevention skill acquisition and in situ training produced generalized responding during naturalistic assessments (Miltenberger et al. 1999).

Using a more cognitive, decision-making approach, Khemka and Hickson (2000) used video vignettes with adults with intellectual disabilities that showed potentially abusive situations (e.g., sexual, psychological, and physical). The vignettes were short video clips (<1 min) and included a visual scene overlaid with a verbal narrative. The scenes were problematic real-life

social interpersonal situations that required decision-making by the individual. All participants scored high in their ability to successfully recognize and define a situation involving abuse as a problem situation. However, rather than act independently, the individuals often wanted to seek help from an authority figure. While these are still prevention efforts, they are not as immediate as taking action themselves. These authors recommend that the focus of abuse prevention training should be on fostering independent, generalizable, self-regulatory skills, such as decision-making, assertiveness, and self-empowerment.

Teaching methods need to be adapted to the level of functioning of the target individuals. The concurrent deficits in communication, social skills, and behaviors – which accompany ASD – will have to be considered when creating personal safety training programs. Several materials and curricula exist that may be helpful in training but have not been empirically evaluated. Susan Ludwig’s book, *Sexuality: A Curriculum for Individuals Who Have Difficulty with Traditional Learning Methods* (Ludwig 1993), uses exercises based on drawings of the four “Feelings Faces” to express “happy, sad, mad and scared.” These faces can be used to communicate feelings about various touches (appropriate and not appropriate). O’Day (1983) created a sexual abuse prevention curriculum for use with students with a variety of physical and developmental disabilities. The curriculum includes a list of teaching guidelines, lesson plans (instructions, exercises, handouts, teacher guides), and notes for how to adapt the curriculum for various student impairments. Lessons cover personal safety and myths/facts about sexual assault. Plummer’s (1997) curriculum, *Preventing Sexual Abuse*, provides tips for working with youth with developmental disabilities. She suggests keeping concepts simple and staying within the limits of abilities and attention spans of the youth. Plummer emphasizes repetition to reinforce the learning, as well as utilizing movement, for example, with role plays or cooperative games. Techniques that will be particularly useful with children with developmental disabilities, including ASD, are instruction that is active and participatory, not lecture. Role play and

dramatization are excellent techniques for educating and assessing learning. Reviews have consistently concluded that programs that incorporate modelling (i.e., demonstrating the skill to be learned) and rehearsal (e.g., role plays) are more effective than programs that primarily rely on individual study or passive exposure (Topping and Barron 2009; Walsh et al. 2015; Wurtele 2009; Wurtele and Kenny 2010; Zwi et al. 2007). Given that most parents reported that their children with ASD were usually or sometimes able to follow rules and one- to three-step directions, personal safety training could incorporate simple rules about behavior and touching (Kenny et al. under review).

The program must be simple, use plain language, involve repetition, and be implemented over a lengthy period of time to be as effective as possible. Other strategies include focusing on the concrete, not the abstract and using role plays to build skills through practice. Plummer (1997) recommends that if the prevention training is conducted in a group setting, keep the groups small, with a preferred one-to-four staff to student ratio. Finally, she states parental and teacher involvement is crucial for additional support and reinforcement of material outside of the sessions. Since repetition and reinforcement are necessary for learning, it is imperative that parents, care takers, and educational staff reinforce appropriate behaviors and review concepts.

In addition to using certain techniques in teaching personal safety skills, improving communication skills for those with disabilities is also an important component (Burke et al. 1998). Burke et al. (1998) recommend teaching the names of private parts to youth so that disclosure of abuse is possible. This is consistent with researchers in the field of CSA prevention (Wurtele and Kenny 2011). Often youth are not taught the correct names for basic anatomy and use terms that are not universally understood. Burke et al. (1998) also recommend teaching youth to trust their feelings by validating, rather than dismissing or minimizing, them. Other components of personal safety include helping the individual to identify enjoyable touches and to recognize what is appropriate touch from various individuals (caregivers,

health professionals, etc.) in different circumstances. An understanding of maintaining personal boundaries and for respecting the boundaries of others is another critical component of prevention planning. Children with ASD should be taught a safety plan (e.g., say “NO” loudly, leave and tell someone) when their boundaries are violated.

Personal safety can be viewed as a beginning step in sexuality education. Parents of children with ASD need guidance on sexuality topics, including sexual abuse prevention, and professional assistance in having these discussions. Professionals involved in the lives of children with ASD need to be open to dialogue about these issues and should likely initiate these discussions with parents. As Breuner et al. (2016) state, developmentally appropriate, evidence-based sexuality education over time provided by pediatricians, educators, other professionals, and parents is necessary to assist youth in making informed, positive, and safe choices about healthy relationships, responsible sexual activity, and their reproductive health. Parental concerns should be addressed as they arise with a child’s changing developmental stages. While parents are often reluctant to have such discussions with their children, they are the best educators for their child and can use the opportunity to infuse their values related to sexuality (e.g., premarital sex, abortion, and contraception).

Sexuality education discussions are imperative in personal safety training, as research has shown that lower levels of sexual knowledge are associated with likelihood of being sexually assaulted (Brown-Lavoie et al. 2014). Ballan (2001) further found that all parents were worried about their children being sexually abused and had talked to them about sexual abuse prevention. Some parents of children with ASD fear that discussing sexual-related topics with their children will increase their sexual interest or behaviors (Brown-Lavoie et al. 2014; Konstantareas and Lunskey 1997), although this seems unsubstantiated by research.

Parents of youth with ASD share many of the same concerns as their counterparts with typically developing children, but research has shown that

they have some additional concerns about sexuality education. In the Ballan (2012) study, parents of children with ASD verbalized a strong desire to communicate with their children about sexuality, but felt they were not adequately prepared to do so. In focus groups with parents of children with ASD, Nichols and Blakeley-Smith (2009) found that they felt unclear about what healthy sexuality could look like for their children. Parents also shared concerns that their children struggle with understanding privacy, boundaries, personal space, and not being able to read social cues. All parents worried that their children could be taken advantage of and possibly raped or sexually assaulted. At the same time, they were not sure how much information to give their child, how long to wait until discussing particular topics, and what strategies to use for teaching. The majority of parents in the Ballan (2001) study stated that they would be comfortable communicating about sexuality with their children with ASD, but they questioned their children's ability to comprehend the information as a barrier to discussions.

There are few prevention or sexuality education programs directed specifically at parents of children with ASD. Nichols and Blakeley-Smith (2009) created a group for parents (8 weeks for 1-h per week) to help them discuss certain sexuality topics with their children with ASD and to increase their comfort and competence for these discussions. Parents reported satisfaction with learning, sharing with other parents, and gaining resources for talking with their children. Corona et al. (2016) provided a sexuality education program for teens with ASD and their parents. The program consisted of six 2-h sessions over a 3-month period. Topics included introducing sexuality, puberty, the human body and maturation, masturbation, privacy, friendship development, interpersonal relationships, dating, sexual activity, personal safety, legal issues, and electronic communication. The program relied on techniques shown to promote learning in individuals with ASD including direct, clear, and specific descriptions of the session's topic. When possible, verbal conversation and prompts were accompanied by visual representations of the

subject matter. Participants were consistently and frequently rewarded for their willingness to participate and their compliance with group rules. When necessary, an assistant used verbal and visual reminders of appropriate behavior (Corona et al. 2016). Parents were in simultaneous sessions, which covered the same topics but were also provided ways to support their child. Parents reported having discussed significantly more sexuality topics with their adolescents following the program and decreased concerns about some of the topics. Meister et al. (1994) conducted a six-session parent group aimed at increasing parents' competence in handling issues of sexuality with their adolescent children with ASD. The program included discussion of sexual development, masturbation, preventing sexual abuse, and talking to one's children about sexuality. Group participants completed measures of parent stress and tracked progress on goals they had set for themselves but results of these assessments were not reported.

In sum, programs that utilize behavioral skills training and incorporate teaching techniques known to be effective with children with ASD appear to be the most promising. Educating parents about CSA and the potential risks to their children, as well as ways they can help to ensure a safe environment is an important first step in prevention. It is imperative that parents, caretakers, and schools join in prevention efforts so that youth with ASD receive repeated instruction over time. Booster sessions for youth can help ensure skills are maintained over time. Developing programs that incorporate exceptional education methods while considering potential cognitive and emotional deficits of youth needs to be achieved. Adapting programs that have been used with adults with disabilities may be appropriate, but must consider the developmental level of the child.

Future Directions

While some work has been done in the area of teaching personal safety for children with ASD,

prevention specialists need to develop empirically supported programs that target this group of vulnerable youth. Teaching personal safety to youth with ASD should be a comprehensive approach that begins at home with parents as teachers, continues formally in school, and includes repeated exposure to safety concepts. In addition to targeted programming, Muccigrosso (1991) recommends parents of children with disabilities begin early in their child's life to teach them independence skills, decision-making, and assertive skills – all of which are general protective factors against sexual abuse and can be honed in later prevention programs.

Best practices for personal safety programs suggest that enlisting parents as their children's teacher is the most effective way to teach such skills. This allows for repeated exposure in a natural environment. Additionally, training for parents is critical to increase their awareness of the problem of sexual abuse in youth. Providing opportunities for parents to role-play discussions with their children will be an important component of programming. For programming to be truly effective, it will need to be more than a onetime event. Successful programs should include school-based curriculum and parental involvement; education to ensure that concepts are reinforced in the natural environment (Muccigrosso 1991). It is recommended that frequent booster training in CSA education and body safety training be provided for individuals with ASD, so as to demonstrate enhanced knowledge and skill development, and ensure a sufficient level of maintenance and generalization (Kenny et al. 2013).

Based on their prevention work with mentally deficient Chinese adolescents, Lee and Tang (1998) make a number of recommendations for future programming. These include expanding the complexity of inappropriate request recognition to include more ambiguous circumstances, as well as an emphasis on a behavioral approach that embodies modeling, behavioral rehearsal, and reinforcement. More specifically, vignettes for behavioral rehearsal could cover settings and

people familiar to the child including teachers, aides, and exceptional education staff. Lee and Tang (1998) also recommend inclusion of general sexuality education for children with ASD.

Sexuality education experts will need to brainstorm strategies to overcome challenges and obstacles in teaching children with ASD. As many experts have stated, sexual education is essential for reducing the risk of sexual abuse among children with disabilities. Teaching children about sexuality in a healthy, appropriate manner is necessary or they may learn about it through abusive, exploitive relationships. Sexuality educators will need to join forces with exceptional education specialists to create curriculum for children with ASD, as well as their families, which considers the range of cognitive and verbal limitations these youth may possess. Unique instructional strategies will need to be developed to ensure appropriate instruction that leads to learning for these populations. Sullivan and Caterino (2008) recommend that educators should be prepared to address sexuality with children with ASD in schools through interventions informed by individuals' levels of cognitive functioning, social skills, and awareness.

There is a need to systematically evaluate existing programs and determine their effectiveness. Empirical evaluation of these programs is imperative to ensure children with ASD are receiving the best, most efficacious programming. Guaranteeing that programs meet certain standards and are accessible to school districts or parents is critical. While a number of programs exist, they may not meet rigorous evaluation standards. Children with ASD are vulnerable to abuse and teaching personal safety should be an educational priority to ensure they live a life free from abuse.

See Also

- [Child Abuse in Autism](#)
- [Peer Victimization](#)
- [Safety](#)

- ▶ Sex Education
- ▶ Sexuality in Autism
- ▶ Social Behaviors and Social Impairment
- ▶ Social Stories
- ▶ Teaching Abduction-Prevention Skills to Children with Autism

References and Reading

- Ballan, M. (2001). Parents as sexuality educators for their children with developmental disabilities. *Sexuality Information and Education Council of the United States Report*, 29, 14–19.
- Ballan, M. S. (2012). Parental perspectives of communication about sexuality in families of children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(5), 676–684.
- Breuner, C. C., Mattson, G., American Academy of Pediatrics Committee on Adolescence, & American Academy of Pediatrics Committee on Psychosocial Aspects of Child and Family Health. (2016). Sexuality education for children and adolescents. *Pediatrics*, 138(2), e1–e11.
- Briggs, F., & Hawkins, R. M. F. (1994). Follow-up data on the effectiveness of New Zealand's national school-based child protection program. *Child Abuse & Neglect*, 18, 635–643.
- Brown-Lavoie, S. M., Viecili, M. A., & Weiss, J. A. (2014). Sexual knowledge and victimization in adults with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(9), 2185–2196.
- Burke, L., Bedard, C., & Ludwig, S. (1998). Dealing with sexual abuse of adults with a developmental disability who also have impaired communication: Supportive procedures for detection, disclosure and follow-up. *The Canadian Journal of Human Sexuality*, 7, 79–91.
- Corona, L., Fox, S., Christodulu, K., & Worlock, J. (2016). Providing education on sexuality and relationships to adolescents with Autism Spectrum Disorder and their parents. *Sexuality & Disability*, 34(2), 199–214.
- Dekker, L. P., van der Vegt, E. J. M., van der Ende, J., Tick, N., Louwerse, A., Maras, A., & Verhulst, F. C. (2017). Psychosexual functioning of cognitively-able adolescents with Autism Spectrum Disorder compared to typically developing peers: The development and testing of the teen transition inventory- a self- and parent report questionnaire on psychosexual functioning. *Journal of Autism and Developmental Disorders*, 47(6), 1716–1738.
- Doughty, A., & Kane, L. (2010). Teaching abuse-protection skills to people with intellectual, disabilities: A review of the literature. *Research in Developmental Disabilities*, 31(2), 331–337.
- Edelson, M. (2010). Sexual abuse of children with autism: Factors that increase risk and interfere with recognition of abuse. *Disabilities Study Quarterly*, 30(1).
- Finkelhor, D., Shattuck, A., Turner, H. A., & Hamby, S. L. (2014). The lifetime prevalence of child sexual abuse and sexual assault assessed in late adolescence. *Journal of Adolescent Health*, 55(3), 329–333.
- Kenny, M. C., Bennett, K., Dougery, J., & Steele, F. (2013). Teaching general safety and body safety training skills to a Latino preschool male with Autism. *Journal of Child and Family Studies*, 22(8), 1092–1102.
- Kenny, M., Crocco, C., & Long, H. (under review). Parents' plans to communicate about sexuality and child sexual abuse with their children with Autism Spectrum Disorder. *Journal of Autism and Developmental Disabilities*.
- Khemka, I., & Hickson, L. (2000). Decision-making by adults with mental retardation in simulated situations of abuse. *American Journal on Mental Retardation*, 38, 15–26.
- Kim, Y. (2010). Personal safety programs for children with intellectual disabilities. *Education and Training in Autism and Developmental Disabilities*, 45(2), 312–319.
- Koller, R. (2000). Sexuality and adolescents with autism. *Sexuality and Disability*, 18(2), 125–135.
- Konstantareas, M. M., & Lunsy, Y. J. (1997). Sociosexual knowledge, experience, attitudes, and interests of individuals with autistic disorder and developmental delay. *Journal of Autism and Developmental Disorders*, 27(4), 397–413.
- Lee, Y. K., & Tang, C. S. (1998). Evaluation of a sexual abuse prevention program for female Chinese adolescents with mild mental retardation. *American Journal on Mental Retardation*, 103(2), 105–116.
- Ludwig, S. E. (1993). *Sexuality a curriculum for individuals who have difficulty with traditional learning methods*. York: The Regional Municipality of York Public Health.
- Lumley, V. A., Miltenberger, R. G., Long, E. S., Rapp, J. T., & Roberts, J. A. (1998). Evaluation of a sexual abuse prevention program for adults with mental retardation. *Journal of Applied Behavior Analysis*, 31, 91–101.
- Mandell, D. S., Walrath, C. M., Manteuffel, B., Sgro, G., & Pinto-Martin, J. A. (2005). The prevalence and correlates of abuse among children with autism served in comprehensive community-based mental health settings. *Child Abuse & Neglect*, 29(12), 1359–1372.
- Meister, C., Norlock, D., Honeyman, S., & Pierce, K. (1994). Sexuality and autism: A parenting skills enhancement group. *Canadian Journal of Human Sexuality*, 3(3), 283–289.
- Miltenberger, R. G., Roberts, J. A., Ellingson, S., Galensky, T., Rapp, J. T., Long, E. S., et al. (1999). Training and generalization of sexual abuse prevention skills for women with mental retardation. *Journal of Applied Behavior Analysis*, 32, 385–388.

- Muccigrosso, L. (1991). Sexual abuse prevention strategies and programs for persons with developmental disabilities. *Sexuality and Disability*, 9, 261–271.
- Nichols, S., & Blakeley-Smith, A. (2009). “I’m not sure we’re ready for this...”: Working with families toward facilitating healthy sexuality for individuals with autism spectrum disorders. *Social Work in Mental Health*, 8(1), 72–91.
- O’Day, B. (1983). *Preventing sexual abuse of persons with disabilities: A curriculum for hearing impaired, physically disabled, blind and mentally retarded students*. Santa Cruz: ETR Associates.
- Pérez-Fuentes, G., Olsson, M., Villegas, L., Morcillo, C., Wang, S., & Blanco, C. (2013). Prevalence and correlates of child sexual abuse: A national study. *Comprehensive Psychiatry*, 54(1), 16–27.
- Plummer, C. (1997). *Preventing sexual abuse* (2nd ed.). Holmes Beach: Learning Publications.
- Sullivan, A., & Caterino, L. (2008). Addressing the sexuality and sex education of individuals with Autism Spectrum Disorders. *Education and Treatment of Children*, 31(3), 381.
- Sullivan, P. M., & Knutson, J. F. (2000). Maltreatment and disabilities: A population-based epidemiological study. *Child Abuse & Neglect*, 24(10), 1257–1273.
- Topping, K. J., & Barron, I. G. (2009). School-based child sexual abuse prevention programs: A review of effectiveness. *Review of Educational Research*, 79(1), 431–463.
- U.S. Department of Health & Human Services, Administration for Children and Families, Children’s Bureau. (2019). *Child maltreatment 2017*. Available from: <https://www.acf.hhs.gov/sites/default/files/cb/cm2017.pdf>
- Walsh, K., Zwi, K., Woolfenden, S., & Shlonsky, A. (2015). School-based education programmes for the prevention of child sexual abuse. *Cochrane Database of Systematic Reviews*, 16(4), Art. No.: CD004380. <https://doi.org/10.1002/14651858.CD004380.pub3>.
- Wurtele, S. K. (1986). *Body safety training*. Author: Colorado Springs.
- Wurtele, S. K. (2009). Preventing sexual abuse of children in the 21st century: Preparing for challenges and opportunities. *Journal of Child Sexual Abuse*, 18, 1–18.
- Wurtele, S. K., & Kenny, M. C. (2010). Primary prevention of child sexual abuse: Child- and parent-focused approaches. In K. L. Kaufman (Ed.), *The prevention of sexual violence: A practitioner’s sourcebook* (pp. 107–119). Holyoke: Neari Press.
- Wurtele, S., & Kenny, M. (2011). Normative sexuality development in childhood: Implications for developmental guidance and prevention of childhood sexual abuse. *Counseling and Human Development*, 43(9), 1–24.
- Zwi, K., Woolfenden, S., Wheeler, D., O’Brien, T., Tait, P., & Williams, K. (2007). School-based education programs for the prevention of child sexual abuse. *Campbell Systematic Reviews*, 5. <http://campbellcollaboration.org/lib/project/28/>

Team Approach

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Definition

Team approach is a model involving a team of professionals with complementary backgrounds and skills working together toward common goals.

This approach is increasingly advocated by scholars and policy makers as a means of assuring quality of outcomes and quality of work environment.

Comprehensive intervention models for individuals with autism spectrum disorder (ASD) require the expertise of professionals from multiple disciplines (e.g., physicians, psychologists, speech and language therapists, occupational therapists, special educators). International guidelines (Department of Children and Families 2003; Committee on Educational Interventions for Children with Autism 2001; Roberts and Prior 2006) highlight the importance of employing a collaborative approach of teamwork in providing effective intervention for ASD. Family involvement is a key component of a collaborative intervention approach, especially for successful infant-toddler interventions.

Three collaborative models exist:

- *Multidisciplinary team*. Professionals work in parallel. Individual therapists perform their assessments, goal planning, and treatment according to discipline-specific tasks and hierarchical lines of authority. There is little overlap between the team members.
- *Interdisciplinary team*. Communication and interdependence between professionals is encouraged, and team members contribute their own professional expertise to establish goals and deliver therapy; however, each

member delivers discipline-specific therapy (e.g., the speech and language therapist in the team is responsible for the language component of the intervention).

- *Transdisciplinary team.* Professionals collaborate to produce a single integrated intervention plan. Team members have or develop skills across a range of different disciplines, as they share and integrate their expertise to formulate and implement mutually agreed goals. A high degree of communication and cooperation among the team members occurs within this model. Each member delivers different aspects of the treatment that might or might not relate to their background (e.g., the speech and language therapist in the team might implement fine motor, cognitive, and language intervention).

Research suggests that the most appropriate team approach to address the needs of individuals with ASD is the transdisciplinary approach (Klin et al. 2005; Roberts and Prior 2005). This approach might also serve the purpose of fostering individual team members' capacity to work in a cohesive and cooperative way, with positive effects on work satisfaction and motivation (Vivanti et al. 2017). Moreover, families feel that compared to other models, a transdisciplinary team is more powerful, less intrusive, and more successful in promoting active participation in the treatment programs (Bell et al. 2009).

Further research in this area is necessary, in order to (1) understand the impact of different teamwork approaches across different settings (e.g., childcare, school, clinical setting); (2) address the challenges involved in coordinating team members, ensuring ongoing communication, organizing training, and providing unitary guidelines; (3) address the issue of establishing and maintaining a single clear line of communication with the family.

See Also

- [Advocacy](#)
- [Classroom Structure](#)
- [Clinical Assessment](#)

- [Ecological Inventory](#)
- [Ecological Validity](#)
- [Family Therapy](#)
- [Functional Ecological Approach](#)
- [Interdisciplinary Team](#)

References and Reading

- Bell, A., Corfield, M., Davies, J., & Richardson, N. (2009). Collaborative transdisciplinary intervention in early years – Putting theory into practice. *Child: Care, Health and Development*, 36(1), 142–148.
- Committee on Educational Interventions for Children with Autism. (2001). *Educating children with autism*. Washington: National Research Council.
- Department of Children, Schools and Families. (2003). *Every child matters*. London: Department of Children, School and Families.
- Klin, A., Saulnier, C. D., Tsatsanis, K. D., & Volkmar, F. R. (2005). Clinical evaluation in autism spectrum disorders: Psychological assessment within a transdisciplinary framework. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed.). New York: Wiley.
- Roberts, J. M. A., & Prior, M. (2006). *A review of the research to identify the most effective models of practice in early intervention of children with autism spectrum disorders*. Adelaide: Australian Government Department of Health and Ageing.
- Vivanti, G., Capes, K., Duncan, E., Dawson, G., & Rogers, S. J. (2017). Setting up the G-ESDM Team and Learning Environment. In *Implementing the group-based early start Denver model for preschoolers with autism* (pp. 43–57). Cham: Springer.

Teglutik

- [Riluzole](#)

TELD

- [Test of Early Language Development \(TELD\)](#)

TELD-2

- [Test of Early Language Development \(TELD\)](#)

TELD-3

► Test of Early Language Development (TELD)

Telegraphic Speech

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Synonyms

Early multiword utterances; Three-word phrases and sentences

Definition

Telegraphic speech is a concise message characterized by the use of three-word short phrases or sentences made up of main content words such as nouns and verbs and void of function words and grammatical morphemes such as articles (e.g., *the*, *a*), auxiliaries or modals (e.g., *is*, *are*, *can*), prepositions (e.g., *in*, *on*), and tense morphemes (e.g., *-ing*, *-ed*, *-s*). The omission of certain words and grammatical morphemes resembles what is typically seen in a brief telegram, hence the term *telegraphic speech*. Telegraphic speech is seen developmentally when a child moves beyond the two-word, relational stage of language development and begins to express longer, three-word sentences using a finite set of grammatical categories, such as nouns, verbs, and adjectives.

Information regarding language developmental milestones (including the use of telegraphic speech) of children with autism is somewhat lacking. Autism is often diagnosed when a child is 3–4 years old following a history of language delay or regression, and the developmental language milestones of children with autism are most often determined by a retrospective parent report of the presence or absence of language behaviors during infancy, toddler, and preschool years.

There are few longitudinal studies of language development in verbal children with autism, although there is some evidence that, when verbal, development of sentence structure is similar to that of typically developing children.

See Also

- Grammar
- Language Acquisition
- Language Disorder
- Phrase Length
- Syntax

References and Reading

- Brown, R. (1973). *A first language: The early stages*. Cambridge, MA: Harvard University Press.
- Demuth, K. (1994). On the underspecification of functional categories in early grammars. In B. Lust, M. Suner, & J. Whitman (Eds.), *Syntactic theory and first language acquisition: Cross-linguistic perspectives* (pp. 119–1234). Hillsdale: Erlbaum.
- Hoff, E. (2001). The development of syntax and morphology: Learning the structure of language. In E. Hoff (Ed.), *Language development* (2nd ed., pp. 201–257). Stamford: Thomson Learning.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 335–364). New York: Wiley.

Telehealth: An Effective Model for Providing Behavior Analytic Interventions to Individuals with ASD

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Definition

Telehealth involves the use of technology for the delivery of health-related services. It encompasses

the use of computers, mobile phones, and other digital devices for the communication between an expert and a service-recipient. Technological strategies involve the use of computer-based video conferencing, online teaching platforms, live coaching, and related content (Bearss et al. 2017).

Historical Background

Interventions based on applied behavior analysis (ABA) are considered evidence-based practice for individuals with autism spectrum disorder (ASD) and their families (Reichow et al. 2018). Recent studies have proven that access to ABA-based interventions early enough during child development can lead to optimal outcomes (Orinstein et al. 2014).

Albeit this need for highly qualified professionals who can deliver effective interventions to individuals with ASD, the availability of coaching for their caregivers and university-based education in behavior analysis for early-career professionals is scarce. This has resulted in an insufficient number of professionals being credentialed as Board Certified Behavior Analysts (BCBA[®]) and a consequent gap in service provision for individuals with ASD. With autism prevalence now standing at 1 in 59 newborns in the USA (Centers for Disease Control and Prevention 2018) and a total of 70,000 individuals living with autism in the UK (National Autistic Society 2019), the need for BCBAs to reach under-served populations is more pressing than ever before. To illustrate the magnitude of this lack of quality provision with exact numbers, only 376 BCBAs are found in the UK (BACB 2019), translated to a ratio of 1 BCBA per 186 individuals with ASD. With this panorama, it is not surprising that high burnout rates have been reported among early-career BCBAs (Dounavi et al. 2019) with obvious negative consequences of such phenomenon to the quality of services offered and the well-being of individuals with ASD and their caregivers (Weiss et al. 2014). The effects of such shortage are felt strongly in rural areas, where lack of service availability has led to reduced parental satisfaction (Murphy and Ruble 2012).

By exploring new delivery models, researchers can investigate how best to disseminate good training practices and support as many individuals as possible. One model which has the potential to increase the reach of behavior analysis is telehealth, a method already showing promise in studies assessing its effectiveness for the provision of medical treatment for numerous conditions, including diabetes, heart disease, and depression (Department of Health 2011).

Current Knowledge

To date there have been 28 published studies which have solely utilized a telehealth platform to provide behavior analytic services to individuals with ASD (Ferguson et al. 2018). These have demonstrated favorable outcomes and shown the flexibility and efficiency of telehealth across participants, settings, and interventions.

Telehealth can adopt a two-pronged approach to training, adapting proven face-to-face techniques onto the telehealth model. The first approach includes an initial training phase that utilizes technology to provide trainees with the theoretical knowledge on a chosen topic. This usually incorporates established training strategies, such as modeling, role-play, and feedback. Preliminary training has been recognized as a key component in achieving procedural fidelity in behavior analytic interventions (Symes et al. 2006), and telehealth may be the ideal platform for delivering this training in a convenient and cost-effective manner, with a greater level of flexibility than traditional models.

The second approach employed is live coaching. Coaching can be defined as the provision of individualized training and feedback on the implementation of the intervention of choice; it can be conducted via telephone, email, or video-conferencing. Feedback can be provided live or retrospectively via recorded footage. Again, known face-to-face training strategies can be successfully incorporated into a telehealth coaching session including modeling of appropriate behavior, self-monitoring, error correction, behavior skills training, and behavior-specific feedback.

Training packages have most commonly incorporated a combination of initial online training and coaching techniques in a “hybrid” approach (e.g., Simacek et al. 2017).

The technology used in telehealth-based training can be easy to obtain. Video-conferencing software used to conduct didactic training and video coaching is often free to access and readily available (e.g., Skype, Viber, Google Hangouts, or iChat). Hardware such as personal computers, web cameras, or tablets are often already found in the intervention settings. When devices or software are purchased, these are in-expensive and readily available. An important consideration when adopting a telehealth-based approach to training is to ensure compliance with data protection regulations, which vary dependent on region and should be used to guide the selection of appropriate tools (see the Healthcare Insurance Portability and Accountability Act, HIPAA, established in 1996 in the USA or the General Data Protection Regulation, GDPR, enforced in Europe in 2018). Compliance with these regulations is an ethical and legal necessity, and professionals considering incorporating a telehealth component should work to ensure they use technology compliant with the regulations of their region in terms of the security, privacy, and management of data (Hall and McGraw 2014).

The telehealth model has the ability to successfully train a variety of participants employed across different sectors, including schools, hospitals, and family homes (e.g., Ingersoll et al. 2016). The potential of telehealth to bring together multidisciplinary teams mirrors the reality of on-site ASD services where interdisciplinary teams work together to plan provision (Department of Education Department of Health 2015; Dillenburger et al. 2014). In fact, telehealth can be a valuable tool in introducing expertise that would otherwise be unavailable. With that being said, the use of telehealth to train participants other than parents is somewhat limited and further research in this area is warranted.

Providing parents with training to carry out interventions has been researched more extensively and results have indicated this can be done to a high degree of fidelity, translating

parent-mediated interventions as evidence-based practice (EBP) (Wong et al. 2014). Parent training has been highlighted as an essential component of a successful ASD intervention (National Research Council 2001). Using telehealth for parent training has the additional bonus of training being conducted in ecologically relevant settings, i.e., both the parents and the child are being taught skills directly in the environment where they will be most beneficial if used. Additionally, given young age at the start of intervention is a crucial predictor of success (Perry et al. 2011), telehealth is an excellent manner of bringing science in family homes before children have even attended their first educational setting. In fact, existing research on the use of telehealth has primarily been conducted with participants under 6 years old confirming positive outcomes (Ferguson et al. 2018).

A telehealth training model has been used effectively across several ABA-based strategies, displaying the flexibility of the approach. The most common use of the platform has been to determine the function of behavior deemed challenging by utilizing functional behavior assessments (FBA) to determine why the behavior may be occurring or what the individual is communicating when they exhibit this behavior. Once a function has been identified, telehealth can be used to train parents or professionals strategies to teach an appropriate replacement behavior which will allow the individual to access the same outcome in a socially acceptable manner, a process called functional communication training (FCT) (e.g., Simacek et al. 2017). For the most part, functional behavior assessment and functional communication training conducted through telehealth have been successful resulting in the reduction of the behavior considered challenging. Useful analysis of telehealth interventions which were not successful at decreasing the targeted behavior have indicated that failures occurred due to low procedural fidelity displayed by trainees or due to an evolution in the behavior function (Schieltz et al. 2018).

The effectiveness of telehealth has also been demonstrated with naturalistic behavioral interventions and strategies, including Naturalistic

Developmental Behavioral Interventions (NDBI) and incidental teaching (e.g., Neely et al. 2016). These interventions focus on child-directed activities using the participant's motivation in the natural environment to promote and create learning opportunities. They aim to increase communication, imitation, and play skills and enhance the interactions between adult and child. Findings indicate positive outcomes in terms of upskilling parents and professionals. Trainees have been taught ways to play and interact with their child and have demonstrated an ability to use and create teachable moments to a high level of fidelity. The outcomes for the children have been more mixed, with some children making great gains and others less significant. However, this variability again could be dependent on the ability to train to high fidelity (Barkaia et al. 2017).

In order for telehealth to be deemed an EBP, enough high-quality empirical evidence needs to be accumulated (Reichow et al. 2008). When considering existing evidence for the use of telehealth, desired quality indicators are not met. However, as studies primarily score low due to omissions in reporting as opposed to severe methodological flaws, it would be nonsensical to dismiss past research and not embrace the potential of a telehealth as a promising model for the delivery of established behavior interventions (see Ferguson et al. 2018 for a detailed review).

Future Directions

The use of telehealth as a model for the delivery of behavioral interventions and training is promising, although more quality research needs to be undertaken. The existing body of research indicates that this model can successfully be used to train interventionists; however, there needs to be more research to ensure that existing data cover the breadth of behavior analytic strategies and result in best child outcomes. For example, there is scarce evidence on the effectiveness of Discrete Trial Teaching when interventions or parents are trained through telehealth or on the use of telehealth to promote the acquisition of functional living skills.

Greater awareness and adherence to research quality measures will ensure evidence is robust enough to provide an overall designation of telehealth as an EBP. In addition, follow-up data are missing from the majority of published studies; therefore, a more longitudinal perspective is required in order to increase our confidence in claiming that telehealth-based training can be used to create long-term, meaningful changes in behavior.

As previously described, existing research has primarily focused on FBA and associated FCT or naturalistic interventions. Training in other behavior analytic strategies has not been widely researched; in particular, there is a dearth of evidence focusing on using telehealth to support comprehensive ABA provision, where a number of programs are introduced simultaneously to teach skills across developmental domains in an individualized "packaged" intervention. Comprehensive packages such as "Early Intensive Behavioral Interventions" (EIBI) have a strong evidence base (Reichow 2012) and are often the most common form of behavior analytic provision. Although FBA, FCT, and naturalistic interventions can constitute effective components of EIBI, future research focusing on the combination of strategies would test the potential of telehealth to facilitate intensive training and supervision. Despite individual differences in intervention outcome success, varied results have often been dependent on procedural fidelity. Research has aimed to identify factors which may predict success (Ingersoll et al. 2016), but further research is needed in this area.

Similar limitations are also apparent when examining the age range of participants in prior research, with the majority of past studies focusing on children under the age of six. ABA-based interventions can have great success with both a teenage and adult age cohort (Santiago et al. 2016) and future research focusing on telehealth applications with older students would increase the reach of this model.

In sum, the use of a telehealth for the provision of training and ongoing support to parents or professionals has shown to be both feasible and effective. Those wishing to utilize telehealth training

in their practice or conduct research on its effectiveness should do so with recognition to its limitations but more importantly by making improvements in research rigor that will increase our confidence that telehealth is an EBP. Overall, telehealth should be seen as a promising development in the evolution of ABA provision that is in line with wider changes in social interactions in the twenty-first century.

References and Reading

- BACB. (2019). Retrieved from <https://www.bacb.com/find-a-certificant/>
- Barkaia, A., Stokes, T. F., & Mikiashvili, T. (2017). Inter-continental telehealth coaching of therapists to improve verbalizations by children with autism. *Journal of Applied Behavior Analysis*, 50(3), 582–589. <https://doi.org/10.1002/jaba.391>
- Bearss, K., Burrell, T. L., Challa, S. A., Postorino, V., Gillespie, S. E., Crooks, C., & Seahill, L. (2017). Feasibility of parent training via telehealth for children with autism spectrum disorder and disruptive behavior: A demonstration pilot. *Journal of Autism and Developmental Disorders*, 48(4), 1–11. <https://doi.org/10.1007/s10803-017-3363-2>
- Centers for Disease Control and Prevention (2018). *Autism Spectrum Disorder (ASD): Data & Statistics on Autism Spectrum Disorders*. Last accessed on 14/03/2020 at <https://www.cdc.gov/ncbddd/autism/data.html>
- Department of Education Department of Health. (2015). *Special educational needs and disability code of practice: 0 to 25 years*. Statutory guidance for organisations which work with and support children and young people who have special educational needs or disabilities.
- Department of Health. (2011). *Whole system demonstrator program*.
- Dillenburger, K., Röttgers, H.-R., Dounavi, K., Sparkman, C., Keenan, M., Thyer, B., & Nikopoulos, C. (2014). Multidisciplinary teamwork in autism: Can one size fit all? *The Australian Educational and Developmental Psychologist*, 31(2), 97–112. <https://doi.org/10.1017/edp.2014.13>
- Dounavi, K., Fennell, B., & Early, E. (2019). Supervision for certification in the field of applied behaviour analysis: Characteristics and relationship with job satisfaction, burnout, work demands, and support. *International Journal of Environmental Research and Public Health*, 16(12). <https://doi.org/10.3390/ijerph16122098>
- Ferguson, J., Craig, E. A., & Dounavi, K. (2018). Telehealth as a model for providing behaviour analytic interventions to individuals with autism spectrum disorder: A systematic review. *Journal of Autism and Developmental Disorders*. Springer US. <https://doi.org/10.1007/s10803-018-3724-5>
- Hall, B. J. L., & McGraw, D. (2014). For telehealth to succeed, privacy and security risks must be identified and addressed. *Health Affairs*, 33, 216–221. <https://doi.org/10.1377/hlthaff.2013.0997>
- Ingersoll, B., Wainer, A. L., Berger, N. I., Pickard, K. E., & Bonter, N. (2016). Comparison of a self-directed and therapist-assisted telehealth parent-mediated intervention for children with ASD: A pilot RCT. *Journal of Autism and Developmental Disorders*, 46(7), 2275–2284. <https://doi.org/10.1007/s10803-016-2755-z>
- Murphy, M. A., & Ruble, L. A. (2012). A comparative study of rurality and urbanicity on access to and satisfaction with services for children with autism spectrum disorders. *Rural Special Education Quarterly*, 31(3), 3–11.
- National Autistic Society. (2019). *Autism facts and history*. Retrieved from <https://www.autism.org.uk/about/what-is/myths-facts-stats.aspx>
- National Research Council. (2001). *Educating children with autism*. Washington, DC: The National Academies Press.
- Neely, L., Rispoli, M., Gerow, S., & Hong, E. R. (2016). Preparing interventionists via telepractice in incidental teaching for children with autism. *Journal of Behavioral Education*, 25(4), 393–416. <https://doi.org/10.1007/s10864-016-9250-7>
- Orinstein, A. J., Helt, M., Troyb, E., Tyson, K. E., Barton, M. L., Eigsti, I. M., . . . Fein, D. A. (2014). Intervention for optimal outcome in children and adolescents with a history of Autism. *Journal of Developmental and Behavioral Pediatrics*, 35(4), 247–256. <https://doi.org/10.1097/DBP.0000000000000037>
- Perry, A., Cummings, A., Dunn, J., Freeman, N. L., Hughes, S., Managhan, T., . . . Williams, J. (2011). Research in autism spectrum disorders predictors of outcome for children receiving intensive behavioral intervention in a large, community-based program. *Research in Autism Spectrum Disorders*, 5, 592–603. <https://doi.org/10.1016/j.rasd.2010.07.003>
- Plantiveau, C., Dounavi, K., & Virués-Ortega, J. (2018). High levels of burnout among early-career board-certified behavior analysts with low collegial support in the work environment. *European Journal of Behavior Analysis*, 19(2), 195–207. <https://doi.org/10.1080/15021149.2018.1438339>
- Reichow, B. (2012). Overview of meta-analyses on early intensive behavioral intervention for young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(4), 512–520. <https://doi.org/10.1007/s10803-011-1218-9>
- Reichow, B., Volkmar, F. R., & Cicchetti, D. V. (2008). Development of the evaluative method for evaluating and determining evidence-based practices in autism. *Journal of Autism and Developmental Disorders*, 38(7), 1311–1319. <https://doi.org/10.1007/s10803-007-0517-7>
- Reichow, B., Hume, K., Barton, E. E., & Boyd, B. A. (2018). Early intensive behavioral intervention (EIBI) for young children with autism spectrum disorders (ASD). *Cochrane Database of Systematic Reviews*,

- 2018(5). <https://doi.org/10.1002/14651858.CD009260.pub3>.
- Santiago, J. L., Hanley, G. P., Moore, K., & Jin, C. S. (2016). The generality of interview-informed functional analyses: Systematic replications in school and home. *Journal of Autism and Developmental Disorders*, 46(3), 797–811. <https://doi.org/10.1007/s10803-015-2617-0>.
- Schieltz, K. M., Romani, P. W., Wacker, D. P., Suess, A. N., Huang, P., Berg, W. K., . . . Kopelman, T. G. (2018). Single-case analysis to determine reasons for failure of behavioral treatment via telehealth. *Remedial and Special Education*, 39(2), 95–105. <https://doi.org/10.1177/0741932517743791>
- Simacek, J., Dimian, A. F., & McComas, J. J. (2017). Communication intervention for young children with severe neurodevelopmental disabilities via telehealth. *Journal of Autism and Developmental Disorders*, 47(3), 744–767. <https://doi.org/10.1007/s10803-016-3006-z>.
- Symes, M. D., Remington, B., Brown, T., & Hastings, R. P. (2006). Early intensive behavioral intervention for children with autism: Therapists' perspectives on achieving procedural fidelity. *Research in Developmental Disabilities*, 27, 30–42. <https://doi.org/10.1016/j.ridd.2004.07.007>.
- Weiss, J. A., Wingsiong, A., & Lunskey, Y. (2014). Defining crisis in families of individuals with autism spectrum disorders. *Autism*, 18(8), 985–995. <https://doi.org/10.1177/1362361313508024>.
- Wong, C., Odom, S. L., Hume, K., Cox, A. W., Fettig, A., Kucharczyk, S., . . . Schultz, T. R. (2014). *Evidence-based practices for children, youth, and young adults with autism spectrum disorder*. Retrieved from <https://autismpdc.fpg.unc.edu/sites/autismpdc.fpg.unc.edu/files/2014-EBP-Report.pdf>

Temper Tantrum

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Synonyms

Tantrum

Definition

The term “temper tantrum” or “tantrum” refers to a set of disruptive, undesirable behaviors and

emotional distress in response to unmet wants or needs. Typical behaviors that often comprise a tantrum episode include: crying, screaming, yelling, falling to the floor, hitting, kicking, throwing objects, destroying property, and/or self-injury. Temper tantrums are natural during early childhood development (Kaneshiro 2007). While tantrum behaviors usually decrease in frequency, duration, and intensity for most children, chronic, prolonged, and severe tantrum behaviors can be a serious problem with many children in the Autism Spectrum (Schreibman 2005). These problem behaviors contribute greatly to the challenges and stress faced by parents and schools and pose a significant barrier to effective social and educational development (National Research Council 2001).

Tantrums are often related to deficits in communication and serve as an alternative form of request. Functional behavior assessments are used to determine what causes and maintains a problem behavior like temper tantrum. Common causes or functions of tantrum behavior include gaining the attention of another person, gaining access to preferred items or activities, and avoiding non-preferred activities or settings. For individuals with autism, tantrums may be triggered by idiosyncratic and seemingly benign events such as changes in routine, sensory aversions, or violations of ritual or compulsion (Schreibman 2005). The likelihood of a tantrum may be increased by other factors such as lack of sleep, illness, hunger, or other physiological factors. Once the causes of tantrum have been identified, decreasing tantrum behaviors often involves teaching appropriate communication strategies to request (i.e., functional communication training), strategies to prevent the likelihood of tantrum (i.e., antecedent-based interventions), self-regulation training (i.e., self-management interventions), and extinction procedures. Psychopharmacological treatments have also been shown effective in reducing tantrums and associated problem behaviors in children with autism and related disorders (Portes et al. 2003).

See Also

- [Extinction Procedures](#)
- [Functional Behavior Assessment](#)
- [Functional Communication Training](#)
- [Self-Management Interventions](#)

References and Reading

- Durand, M. (1990). *Severe behavior problems: A functional communication training approach*. New York: The Guilford Press.
- Kaneshiro, N. (2007). *Temper tantrums*. Retrieved 24 Apr 2011, from U.S. National Library of Medicine, National Institutes of Health site. <http://www.nlm.nih.gov/medlineplus/ency/article/001922.htm>
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Portes, V., Hagerman, R., & Hendren, R. (2003). Pharmacotherapy. In S. Ozonoff, S. J. Rogers, & R. L. Hendren (Eds.), *Autism spectrum disorders: A research review for practitioners* (pp. 55–85). Washington, DC: American Psychiatric.
- Schreibman, L. (2005). *The science and fiction of autism*. Cambridge, MA: Harvard University Press.

behavioral characteristics (Henderson and Wachs 2007). Differences exist in the latency, intensity, and threshold of children's reactions to their environments as well as in the ability to maintain or regain a state of equilibrium using self-regulatory processes, such as effortful control (Rothbart and Derryberry 1981). As a group, children with autism are characterized by lower levels of extraversion and positive affect, higher levels of negative affect, and more difficulty regulating attention and emotion based on both parent- (De Pauw et al. 2011; Konstantareas and Stewart 2006) and self-reported assessments (Schwartz et al. 2009). The infant siblings of children with autism (ASD-Sibs) display a distinct profile of temperament compared to the infant siblings of typically developing children that is characterized by lower levels of positive affect, higher levels of negative affect, and difficulty controlling attention and behavior. ASD-Sibs who go on to receive a diagnosis themselves are further characterized by low levels of behavioral approach, or sensitivity to social rewards (Garon et al. 2009). These findings demonstrate both concurrent and predictive associations between temperament and ASD.

Importantly, there is just as much heterogeneity in mean levels and profiles of temperament among children with autism as there is for typically developing children. Several studies suggest that temperament may be an important predictor of individual differences in symptom presentation, adaptive functioning, and comorbid psychopathology. For example, among children with ASD, those with more symptoms are rated by parents as being lower in effortful control (Konstantareas and Stewart 2006), lower in surgency, and higher in negative affect (De Pauw et al. 2011). Similarly, in a sample of higher functioning children and adolescents with autism (HFA) and a typically developing comparison sample, Schwartz et al. (2009) found that children with HFA were rated by their parents as having more internalizing problems. However, over and above this effect of diagnostic group, lower self-reported surgency was associated with higher parent-reported internalizing problems for both TD and HFA participants. Similarly, after

Temperament

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Synonyms

[Personality](#)

Definition

Temperament describes early appearing, biologically based, and relatively stable individual differences in physiological, emotional, and

controlling for the effects of diagnostic group, higher levels of self-reported effortful control were associated with fewer externalizing problems for both TD and HFA participants. Together, these findings suggest that temperament may provide important additional information related to symptom presentation and comorbid behavioral and emotional problems in children with autism. In addition, given comparable patterns of associations with important adjustment outcomes for children with ASD and typically developing children, the mechanisms linking temperament to adjustment are comparable regardless of diagnostic status (De Pauw et al. 2011; Schwartz et al. 2009).

Given the inherent methodological challenges of relying exclusively on parent- and/or self-reported measures of temperament (i.e., reporter biases), it is suggested that multimethod assessment approaches be employed that incorporate multiple informants' perspectives and include direct observational assessments of temperament. A better understanding of individual differences in temperament is important for identifying early risk factors and better tailoring prevention and early intervention efforts to fit the individual needs of children with autism.

See Also

- [Executive Function \(EF\)](#)
- [Motivation](#)
- [Self-regulation](#)

References and Reading

- De Pauw, S. S. W., Mervielde, I., Van Leeuwen, K. G., & De Clercq, B. J. (2011). How temperament and personality contribute to the maladjustment of children with Autism. *Journal of Autism and Developmental Disorders*, 41, 196–212.
- Garon, N., Bryson, S., Zwaigenbaum, L., Smith, I., Brian, J., Roberts, W., et al. (2009). Temperament and its relation to Autistic symptoms in a high-risk infant sib cohort. *Journal of Abnormal Child Psychology*, 37, 59–78.
- Henderson, H. A., & Wachs, T. D. (2007). Temperament theory and the study of cognition-emotion interactions across development. *Developmental Review*, 27, 396–427.
- Konstantareas, M. M., & Stewart, K. (2006). Affect regulation and temperament in children with Autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 36, 143–154.
- Rothbart, M. K., & Derryberry, D. (1981). Development of individual differences in temperament. In M. E. Lamb & A. Brown (Eds.), *Advances in developmental psychology* (Vol. 1, pp. 37–86). Hillsdale: Erlbaum.
- Schwartz, C. B., Henderson, H. A., Inge, A. P., Zahka, N. E., Coman, D. C., Kojkowski, N. M., et al. (2009). Temperament as a predictor of symptomatology and adaptive functioning in adolescents with high-functioning Autism. *Journal of Autism and Developmental Disorders*, 39, 842–855.

Temperament and Atypical Behavior Scale Screener (TABS Screener)

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Synonyms

[TABS](#); [TABS screener](#)

Description

The Temperament and Atypical Behavior Scale (TABS) is a norm-referenced assessment of developmentally dysfunctional behavior or behavior problems for infants and children aged 11–71 months (Neisworth et al. 1999). The purpose of the TABS is to identify children with atypical development or those at risk for atypical development in order to allow for early intervention planning. The TABS contains two components: the TABS Screener and the TABS Assessment Tool. The TABS Screener is a brief 15-item questionnaire that can be completed in approximately 5 min and is used to determine if a child requires a more thorough assessment of developmental aspects related to temperament and self-regulation. The TABS Assessment Tool

contains 55 questions about the child's behavior that can be completed in approximately 15 min. Both the Screener and Assessment Tool should be completed by a parent or professional who is familiar with the child's daily behavior (Neisworth et al. 1999). A "yes" on this measure indicates that the behavior is a current or recent problem, and a "no" indicates that the behavior is not a problem or does not apply to the child. Some of the questions require a subjective judgment of frequency, duration, or intensity of behaviors.

The questions from the TABS Assessment Tool load onto four factors: detached, hypersensitive/active, underreactive, and dysregulated. Behaviors consistent with the detached factor include such things as being withdrawn, unengaged, or disconnected from everyday life that involves other individuals. These behaviors may often be seen in individuals with autism spectrum disorder. The second factor, hypersensitive/active, reflects overreactive, impulsive, and highly active behaviors. Often these behaviors are associated with attention-deficit/hyperactivity disorder (ADHD). The third factor, underreactive, refers to behavior that is often unresponsive and requires intense stimuli to elicit a response from the child. The fourth factor, dysregulated, contains behaviors that indicate the child has trouble controlling behavior that has a neurological basis, and may have trouble sleeping and hypersensitivity to contact. Overall, the four factors are used to determine if a child's temperament and self-regulation are atypical (Neisworth et al. 1999, p. 3).

Historical Background

The TABS Assessment Tool was based on more than 10 years of research by its authors on the temperament and self-regulatory styles associated with developmental delay. The authors of this measure aimed to overcome many of the conceptual and methodological limitations of applying commonly used adult measures of behavior problems and psychopathology to infants and young children. The goal of this measure is to detect early behavior problems and to help practitioners

to devise an intervention plan for young children. The scale is based on research in the areas of typical self-regulation development, characteristics of atypical self-regulatory development, and specific self-regulatory problems associated with diagnosed neurodevelopmental/neurobehavioral disorders (Neisworth et al. 1999, p. 7). The TABS authors define temperament as a biologically based behavioral style that influences dimensions of behavior and self-regulation. More specifically, self-regulation is the ability to control and monitor behaviors according to social demands. The measure identifies unusual temperamental and self-regulatory behaviors, or indicators, that have been previously associated with neurodevelopmental disabilities, such as autism spectrum disorder, with items that focus on neurobehavioral markers, or early atypical behavioral patterns thought to have a neurological basis (Neisworth et al.).

Psychometric Data

The scores derived from TABS reflect overall self-regulatory problems, with higher raw scores indicating more atypical self-regulation. Calculating the Temperament and Regulatory Index (TRI) from the TABS Assessment Tool allows for interpretation of raw scores. The authors suggest that using TABS normative cutoffs, a TRI score of 5 indicates at-risk status and a TRI of 8 or higher is indicative of severe behavioral dysfunction.

The TABS normative sample consisted of 621 children (11–71 months of age), 52% of whom were 2 years old or younger. Approximately 60% of the sample's gender was not identified. The scores of children with disabilities in this sample were compared to the scores of children without disabilities; children with disabilities consistently scored higher, indicating poorer performance (Neisworth et al. 1999).

The internal consistency of the TABS Assessment Tool was determined using a split-half reliability procedure, which divided each subtest in half and then correlated the halves of each subtest. The reliability coefficients for children not at risk, for children with disabilities, and for the pooled

sample were .88, .95, and .95, respectively (Neisworth et al. 1999). The authors do not recommend using TABS subtest profiles for diagnostic purposes, as some of the subtests have insufficient reliability for this purpose. Internal consistency of the TABS Screener was determined using coefficient alpha, yielding an alpha of .83 (Neisworth et al. 1999).

With respect to the validity of the TABS Assessment Tool, the authors report that the measure shows a factor structure consistent with theoretical models proposed by Zero To Three: National Center for Infants, Toddler, and Families in 1994. With respect to accuracy of the TABS Screener, about 72% of the children who are screened as at risk or as having a disability will have been screened accurately (Neisworth et al. 1999).

Clinical Uses

The scores derived from TABS may allow clinicians to identify those children who may have a need for early intervention, to assess children's behaviors before the start of intervention, and to help therapists monitor and evaluate progress over the course of intervention. The scores derived from TABS can be used by state agencies as a standardized criteria necessary for families to meet eligibility for government-funded intervention programs or wraparound support (i.e., services that lend support to the family and the child) (Neisworth et al. 1999).

See Also

- [Emotion Regulation](#)
- [Temperament](#)

References and Reading

Neisworth, J. T., Bagnato, S. J., Salvia, J., & Hunt, F. M. (1999). *TABS manual for the temperament and atypical behavior scale: Early childhood indicators of developmental dysfunctions*. Baltimore: Paul H. Brookes.

Temporal Cortex

- [Temporal Lobes](#)

Temporal Lobes

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Synonyms

[Temporal cortex](#)

Definition

The temporal lobes are bilateral portions of the cerebral cortex. They are demarcated at the top by the Sylvian fissure and extend to the ventral (lower) most surface on the brain. The temporal lobes are involved in a variety of key cognitive functions. For instance, they are home to auditory cortex (responsible for the perception and processing of sound). Moreover, portions of the temporal lobes are responsible for high-level semantic processing in language, recognition of letters and words, and processing faces and other complex objects. The medial temporal lobes, especially the hippocampus, also play a key role in the formation and retrieval of long-term memories. Wernicke's area, which spans the region between temporal and parietal lobes, plays a key role (in tandem with Broca's area, which is in the frontal lobe) in language.

See Also

- [Cerebral Cortex](#)
- [Inferior Temporal Area](#)
- [Superior Temporal Sulcus Region](#)

References and Reading

- Hazlett, H. C., Poe, M. D., Gerig, G., Styner, M., Chappell, C., Smith, R. G., et al. (2011). Early brain overgrowth in autism associated with an increase in cortical surface area before age 2 years. *Archives of General Psychiatry*, 68(5), 467–476.
- Zilbovicius, M., Boddaert, N., Belin, P., Poline, J. B., Remy, P., Mangin, J. F., et al. (2000). Temporal lobe dysfunction in childhood autism: A PET study. *The American Journal of Psychiatry*, 157(12), 1988–1993.

Tenex

- [Guanfacine](#)

Teratology

- [Toxicology](#)

Test Age

- [Age Equivalents](#)

Test Bias

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Definition

Test bias refers to the systematic under- or over-prediction of a particular trait or status based upon one's group membership. Groups may include individuals of different races, ethnicities, genders, and socioeconomic statuses. A test may be considered biased because it was not developed using a standardization sample that is representative of the populations on which it is used. According to Sattler and Hoge (2006),

procedures for evaluating assessment instruments for test bias include: (1) examining content for evidence of cultural bias, (2) examining construct validity to determine whether the test measures the same constructs or behavioral dimensions for different groups, and (3) examining criterion-related validity, for example, by determining whether a measure significantly predicted success in treatment for one group but not another. Ortiz and Dynda (2005) argue that, when considering the cultural fairness of a test, test bias is a limited construct as it conflates race and ethnicity in the construction of norm groups and evaluation of a test's psychometric properties. They suggest that when considering tests used with individuals from culturally and linguistically diverse groups, the validity of the test is dependent upon the extent to which evaluation methods and procedures used were appropriate and successful in accounting for the known influence of culture and language on test performance during the evaluation.

See Also

- [Culture and Autism](#)
- [Standardization](#)
- [Validity](#)

References and Reading

- American Psychological Association. (1990). *Guidelines for providers of psychological services to ethnic, linguistic, and culturally diverse populations*. Washington, DC: American Psychological Association.
- American Psychological Association. (2003). Guidelines on multicultural education, training, research, practice, and organizational change for psychologists. *American Psychologist*, 58, 377–402.
- Ortiz, S. O., & Dynda, A. M. (2005). Use of intelligence tests with culturally and linguistically diverse populations. In D. P. Flanagan & P. L. Harrison (Eds.), *Contemporary intellectual assessment: Theories, tests, and issues* (2nd ed., pp. 545–556). New York: Guilford Press.
- Sattler, J. M., & Hoge, R. D. (2006). *Assessment of children: Behavioral, social, and clinical foundations* (5th ed.). San Diego: Jerome M. Sattler.

Test for Auditory Comprehension of Language – Fourth Edition

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Synonyms

TACL-4

Description

The TACL-4 measures language in the areas of syntax and semantics for children ages 3–12 (three sets of stimuli for ages 3–5, 6–10, and 11–12). The test aims to establish what areas of language comprehension are challenging for children (Brown 1986). Three subtests make up the TACL and include test vocabulary, grammatical morphemes, and elaborated phrases and sentences. The length of administrations is between 15 and 20 min. The TACL-4 includes basals and ceilings to reduce frustration in testing and to reduce the time needed to administer the test. This test is appropriate for use by speech-language pathologists, educational diagnosticians, and professionals in related fields, requiring some formal training in assessment.

As a receptive assessment, the test uses visual stimuli presented in a standing flip book. Selection of answers from three picture choices is performed by pointing with no verbal expressive language required. The pictures are realistic, hand-drawn, color pictures of items, avoiding biases (such as race, sex, age, socioeconomic) where possible. The arrangement of correct images is distributed equally in left, middle, and right positions. Instructions for use of the test are clear and easy to administer. Administration uses the same phrase “Show me” throughout the test. Scoring uses 1 for correct and 0 for incorrect (or no response). Correct answers are indicated on the score sheet as A, B, or C for position on page.

Scoring for the TACL-4 uses age equivalents, percentile ranks, scaled scores with a mean of 10 and standard deviation of 3, and a receptive language index (composite score) with a mean of 100 and standard deviation of 15. These measures can support qualification for language services for children who fall below the norms. The TACL-4 is co-normed with the Test of Expressive Language, both of which are designed to be used with children of all abilities (TEXL; Carrow-Woolfolk and Allen 2014). Both assessments are intended to produce cross-battery measures of linguistic systems, characteristics, and general oral language (Carrow-Woolfolk 2014). Comprehensive Scoring Supplement is available to allow comparison of scores and/or combining of scores to produce a more comprehensive measure of a child’s language ability.

Historical Background

The original version of the TACL came about in the early 1960s by Elizabeth Woolfolk (Carrow-Woolfolk 2014). Woolfolk’s inspiration came from an interaction with a 3-year-old boy, who was unable to comprehend or respond to spoken language, maintain eye contact, and struggled with anger outbursts. Woolfolk studied the word categories and patterns they followed. The creation of the TACL was an attempt to identify issues in using these categories and patterns and a means to promote earlier intervention for future clients in similar situations. The initial focus was to evaluate the morphological, syntactical, and lexical components of language because this combination is missing from other receptive language measures (Blood and Greenberg 1978). Background research for the original TACL was based on 40 children, and the results were used to assist with item revision, presentation, order, etc. Once a model assessment was complete, it was used on 159 students (ages 2;10-7;9), controlled for similar IQ, no hearing or speech issues, and monolingual. Over the span of the next decade, the second and third versions of the TACL were released, but it wasn’t until 2013 that the TACL-4 was released. This update was

important because the data collected was from a more diverse demographic. Additionally, the TACL-4 age range is expanded to include 10;0–12;11 and requires less time to administer, and the floor and ceiling effects are greatly reduced in comparison to previous versions.

Psychometric Data

Standardization of the TACL-4 was determined by a sample size of 1,142 children with ages ranging from 3 years, 0 month to 12 years, and 11 months. The normative sample's characteristics were nationally representative for school-age population based on the characteristic's comparable percentages of those in the *Statistical Abstracts of the United States 2013*. The pictures chosen for the test have been designed to be as unbiased as possible, with an equal representation of all demographics throughout the assessment.

Reliability looked at the assessments subtests and the assessment as a whole. Reliability coefficients for content sampling and inter-scorer differences were all above 0.95. Time sampling's highest coefficient was 0.85 for receptive language index and lowest coefficient was 0.71 for grammatical morphemes. These scores indicate high reliability in the TACL-4.

Assessment of criterion-predictive validity was determined by comparing the TACL-4 with three similar assessments: Clinical Evaluation of Language Fundamentals-Fourth Edition (CELF-4; Semel et al. 2003), Clinical Evaluation of Language Fundamentals Preschool-Second Edition (CELF Preschool-2; Wiig et al. 2004), Oral and Written Language Scales-Second Edition (OWLS-II; Carrow-Woolfolk 2011), Diagnostic Achievement Battery-Fourth Edition (DAB-4; Newcomer, 2014), and Test of Expressive Language (TEXL; Carrow-Woolfolk and Allen 2014). The receptive, expressive and general language of the 3 assessments were compared to the TACL-4 by computing each assessment's language measures, means, standard deviation, and diagnostic accuracy. The correlation between all tests and measures of each test was at a magnitude between large and very large, meaning the

coefficients fell in a range between 0.70 and 1.00, with receptive language being the strongest of measures and supporting the TACL-4 as accurately testing areas of oral language. Additionally, TACL-4 standard scores will be similar to the scores from assessments of oral language based on means and standard deviation effect sizes which were small to trivial for receptive language tests and trivial to moderate for expressive and general language. For diagnostic accuracy, the specificity index results for each assessment reveal that the TACL-4 has high accuracy when predicting similar scores of receptive language assessments than assessments that looked at expressive and general language. The high accuracy gives strong validity in the TACL-4 in predicting children who do and do not have a language impairment.

Construct identification validity was measured based on various components. Relationship to age was strongly correlated, obtaining a magnitude of very large correlation with coefficients ranging from 0.73 to 0.76 for each subtest. Relationship to academic achievement was assessed comparing six achievement tests that assessed different literacy skills. The correlation coefficients compared to the various assessments ranged from 0.5 to 0.76, showing that the assessments all tested related, but different abilities indicating a relationship between the TACL-4 and achievement. The relationship to intelligence was revealed through comparison to various intelligence tests. The scores between the Abbreviated Battery Review of the Stanford-Binet Intelligence Scales-Fifth Edition (SB5 ABIQ; Roid 2003) and the Brief Intellectual Ability section from the WJ III assessments and the TACL-4 revealed coefficient scores ranging from 0.58 to 0.82, indicating high support for construct identification validity for each subtest. Subtest interrelationship coefficients for each subtest ranged from 0.42 to 0.49, indicating that the subtests all assess individual aspects of Auditory Comprehension. The validity coefficients indicate that the TACL-4 is a valid assessment for oral language comprehension. A review search of the TACL-4 did not reveal any recent articles since the TACL-4 revision.

Clinical Uses

The TACL-4 tests a child's receptive language in the domains of spoken vocabulary, grammar, and syntax. It is well-suited for use with English-speaking children in schools in the United States. Past research has also concluded that the use of the TACL is successful in measuring the receptive language age of children with intellectual disabilities (Beisler and Tsai 1987). There are also significant associations between a child's language comprehension and executive functioning skills (Lyberg-Åhlander et al. 2015). The TACL-4 would be best suited for a child who achieves low scores in the reception section of a broader assessment. The clinician can then use the scores of the TACL-4 to further refine understanding of areas of challenge and develop a specific intervention for the child. This tool is beneficial to assess receptive language for a child who is nonverbal due to a physical disability.

See Also

- Auditory Discrimination
- Auditory Processing
- Central Auditory Processing Disorder

References and Reading

- Beisler, J. M., & Tsai, L. Y. (1987). Comparisons between autistic and nonautistic children on the test for auditory comprehension of language. *Journal of Autism and Developmental Disorders*, 17(1), 95–102. <https://doi.org/10.1007/bf01487262>.
- Blood, G. W., & Greenberg, B. R. (1978). Normative data for the revised score sheet of the test for auditory comprehension of language. *Language, Speech, and Hearing Services in Schools*, 9(4), 210–212. <https://doi.org/10.1044/0161-1461.0904.210>.
- Brown, J. R. (1986). An evaluation of the test for auditory comprehension of language. *Ear and Hearing*, 7(4), 255–256. <https://doi.org/10.1097/00003446-19860800-00006>.
- Carrow-Woolfolk, E. (2011). *OWLS-II: Oral and Written Language Scales*. Western Psychological Services.
- Carrow-Woolfolk, E., & Allen, E. (2014). *Test of expressive language*. Austin: PRO-ED, Inc.
- Lyberg-Åhlander, V., Haake, M., & Brännström, J. (2015). Does the speakers voice quality influence children's performance on a language comprehension test? *International Journal of Speech-Language Pathology*, 17(1), 63–73. <https://doi.org/10.3109/17549507.2014.898098>.
- Roid, G. H. (2003). *Stanford Binet intelligence scales* (5th ed.). Itasca, IL: Riverside Publishing.
- Semel, E. M., Wiig, E. H., & Secord, W. A. (2003). *Clinical Evaluation of Language Fundamentals* (4th ed.). San Antonio, TX: The Psychological Corporation.
- Wiig, E. H., Secord, W., & Semel, E. M. (2004). *CELF preschool 2: clinical evaluation of language fundamentals preschool*. Pearson/PsychCorp.

Test of Early Language Development (TELD)

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Synonyms

TELD; TELD-2; TELD-3

Description

The Test of Early Language-3rd edition (TELD-3) is a test developed for the assessment of the comprehension and production of English spoken language (focusing on syntax and semantics) in children aged 2;0 to 7;11 years, toddlers to second grade students. Not to be confused with the Test of Language Development (TOLD), now in its 4th edition, for the assessment of oral language in 4;0 to 17;11 year-olds (in primary and intermediate versions). Newcomer and Hammill. Test of Language Development, Fourth Edition (TOLD-4), Western Psychological Association.

Test format and scoring (as reported in Backlund, Morreale, and Suen 2001):

The TELD-3 is to be administered individually. It contains two forms, (A and B). Each form has two subtests, one for Expressive Language

and one for Receptive Language. The Receptive Language subtest contains 37 items; Form A has 24 semantic items and 13 syntax items, while Form B has 25 semantic items and 12 syntax items. The Expressive Language subtest contains 39 items; Form A has 22 semantic items and 17 syntax items, while form B has 24 semantic items and 15 syntax items.

The test materials are easy to use and well prepared. For each item, the child is given a verbal direction and then shown a stimulus object or a picture. Then the child is invited to respond to prompts for each item. This response is scored by the examiner.

The 3rd edition yields an Overall Spoken Language score as well as separate Receptive and Expressive Language subscores. Standard scores (mean of 100 and standard deviation of 15), percentiles, and age equivalents are provided for subtests and for a composite score.

Normative information is reported with respect to geographical residence, gender, race, ethnicity, education, and socioeconomic status, and is stratified by age. Color pictures and manipulatives are included in the test kit. Testing time is reported to take 15–45 min.

Applications in Research on Autism Spectrum Disorders

Although the TELD appears often in the typical language development, stuttering and specific language impairment literatures, as of 2011 it has yet to be applied widely in Autism Spectrum Disorder (ASD) research. Potential uses are for characterizing profile of functioning, matching between comparison groups in experimental studies, and identifying language impairment.

Waterhouse et al. (1996) employed the first edition of the TELD to characterize language level in part of their study sample of 194 preschool children suspected of ASD. Their study compared four systems for the diagnosis of autism (DSM-III, DSM-III-R, DSM-IV, and ICD-IO) and sought to quantitatively identify subgroups of children with pervasive developmental disorder. Their results identified lower- and higher-functioning subgroups with considerable overlap,

suggesting a continuum of pervasive developmental disorders.

Koegel, Carter, and Koegel (2003) used the first edition of the TELD for initial characterization of two children with ASD in a multiple-baseline intervention study focused on increasing the use of grammatical morphemes.

Herbert and colleagues (2003, 2004) have conducted a series of neuroimaging studies examining increased cerebral white matter volume in children with Developmental Language Disorder and High Functioning ASD, relative to typically developing children. All participants had Performance IQs greater than 80. Characterization of language in potential participants with non-ASD Developmental Language Disorder was done using the TELD (first edition) as well as mean length of utterance. Inclusion in the Developmental Language Disorder group required scores that fell one standard deviation or lower below the mean on the TELD. Similar language characterization was unfortunately not conducted for the ASD group. Herbert et al. highlight a similar distribution of white matter enlargement in both clinical groups, and raise the possibility that they fall along the same spectrum. However, this comparison needs to be examined in a sample where the language ability of all participants is characterized, to understand the relationship between anatomical changes and behavior. Overlap and differences in white matter volume profiles between the two clinical groups warrant further study.

Applications in Research on Theory of Mind and Language Development

In contrast, the TELD has been used extensively with typically developing children, especially in research focusing on the relationship between language and theory of mind abilities. This practice likely has its roots in Astington and Jenkins (1999) seminal article on the developmental relationship between language and theory of mind, where they used the TELD to assess language skills three times over a 7-month period in 3-year-olds. They report that earlier language abilities predicted later theory-of-mind performance (controlling for earlier theory of mind), but not the

reverse. Subsequently, the same measure was used in number of other studies on related topics (Cassidy et al. 2005; Cheung et al. 2004; Farrar et al. 2009; Ng et al. 2010).

Historical Background

The Test of Early Language Development was developed around two of three central features of language identified by Bloom and Lahey (1978): content (knowledge of objects, relationships among objects and events - semantics and reference) and form (phonology, syntax, morphology). Language use, the third feature, was not incorporated in the assessment. It was designed to be a quick and easy-to-administer screener of spoken language for young children.

The first edition of the TELD was published in 1986 for use with children aged 3;0 to 7;11. The standardization sample included 1,184 “typical” students who are comparable, where possible, to a US national sample with respect to demographic characteristics.

The second edition, TELD-2, was published in 1991. It provided one overall spoken language score with no subscales. Sorensen and Bradley-Johnson (1993) noted a lack of test-retest reliability by age group as a concern for this edition. The standardization sample included 1,329 students approximating the 1990 US census with respect to demographic characteristics; 150 children were included per age group.

The third edition (TELD-3) was published in 1999. It is designed to assess English spoken language skills at early ages (from 2;0 to 7;11). The TELD-3 has two subtests, Receptive Language and Expressive Language, and yields an overall Spoken Language score. This edition addressed previous criticisms by division of items into Receptive Language and Expressive Language scales, development of standard scores, more effective coverage for children at the upper and lower age ranges of ability, new studies supporting reliability and validity, increased normative data, more visually appealing, color pictures, more accurate wording, and

clearer instructions for test administration (Backlund et al. 2001). This edition includes all necessary manipulatives in the test kit, unlike previous versions.

The TELD-3:S is a Spanish translation and adaptation of the TELD:3 that was published in 2007 to assess early Spanish spoken language in the same age range (from 2;0 to 7;11), (Ramos and Ramos 2007).

Psychometric Data

The TELD-3 has high reliability and excellent validity.

Demographics: The TELD-3 was standardized on 2,217 children representing 35 US states. The normative population was representative of the US population as reported in the 1997 *Statistical Abstract of the United States* (U.S. Bureau of the Census 1997). The test website reports that a wide range of both mainstream and minority populations, including gender, racial, ethnic, linguistic, and disability categories, was included in the normative sample.

Limiting bias: The TELD-3 was examined to ensure that little or no bias relative to gender, disability, racial, socioeconomic, or ethnic groups existed. Reliability and validity information is provided for different mainstream and minority subgroups.

Reliability: Adequate test reliability (coefficient alpha, test/retest, immediate test/retest with equivalent forms, and interscorer) was established.

Validity: Content-description validity was established through careful selection of items, controlled vocabulary, construct review by a panel of language experts, conventional item analysis, differential item functioning analysis, and form equivalence.

Criterion-prediction validity was established by correlating TELD-3 standard scores with a variety of widely recognized measures of language ability (i.e., CELF Preschool, EOWPVT, PLS-3, PPVT-Revised, ROWPVT, and TOLDP:3).

Construct-identification validity was established by studying (a) the relationship of

the TELD-3 standardized scores with age, IQ, and academic achievement and (b) the ability of the TELD-3's standard scores to differentiate groups with known language problems from those without such problems.

Clinical Uses

The TELD-3 is used for the spoken language (syntax and semantics) assessment of children aged 2;0 to 7;11 years, toddlers to Grade 2. The authors identify five purposes of the test: to identify candidates for early intervention, to identify strengths and weaknesses of individuals, to document progress in an intervention program, to serve as a research tool in language development, and to accompany other assessment techniques (Hresko et al. 1999).

Though clinical usage as described above does appear to be a potential use of this test, Backlund et al. (2001) caution that the authors did not report any evidence of these applications, such as positive predictive power indices or over- or underreferral indices, to support its use for identification. There is also no evidence in the manual such as treatment validity to support its use to identify strengths and weaknesses or to document progress in intervention. A literature review in June 2011 did not identify any published studies employing the TELD in ASD assessment or as an outcome measure in intervention research.

The TELD-3 does include two different forms (A and B) of items, making it useful for longitudinal and intervention studies where retesting over short periods of time on the same items is often an issue. Its effectiveness in such applications will need to be determined by future research.

See Also

- [Expressive Language](#)
- [Language Tests](#)
- [Preschool Language Scale – 5](#)
- [Receptive Language](#)

References and Reading

- Astington, J. W., & Jenkins, J. M. (1999). A longitudinal study of the relation between language and theory-of-mind development. *Developmental Psychology*, 35, 1311–1320.
- Backlund, P., Morreale, S., & Suen, H. (2001). Review of the test of early language development-3rd edition. In B. S. Plake & J. C. Impara (Eds.), *The fourteenth mental measurements yearbook*. Lincoln: Buros Institute of Mental Measurements.
- Bartlett, A., Slade, D., & Bellerose, P. C. (1987). Test review: The test of early language development (TELD). *The Reading Teacher*, 40, 546–548.
- Bloom, L., & Lahey, M. (1978). *Language development and language disorders*. New York: Wiley.
- Cassidy, K. W., Fineberg, D. S., Brown, K., & Perkins, A. (2005). Theory of mind may be contagious, but you don't catch it from your twin. *Child Development*, 76, 97–106.
- Cheung, H., Hsuan-Chih, C., Creed, N., Ng, L., Wang, S. P., & Mo, L. (2004). Relative roles of general and complementation language in theory-of-mind development: Evidence from Cantonese and English. *Child Development*, 75, 1155–1170.
- Dale, P. S., & Henderson, V. L. (1987). An evaluation of the test of early language development as a measure of receptive and expressive language. *Language, Speech, and Hearing Services in Schools*, 18, 179–187.
- Farrar, M. J., Johnson, B., Tompkins, V., Easters, M., Zilisi-Medus, A., & Benigno, J. P. (2009). Language and theory of mind in preschool children with specific language impairment. *Journal of Communication Disorders*, 42, 428–441.
- Herbert, M. R., Ziegler, D. A., Makris, N., Bakardjiev, A., Hodgson, J., Adrien, K. T., et al. (2003). Larger brain and white matter volumes in children with developmental language disorder. *Developmental Science*, 6, F11–F22.
- Herbert, M. R., Ziegler, D. A., Makris, N., Filipek, P. A., Kemper, T. L., Normandin, J. J., et al. (2004). Localization of white matter volume increase in autism and developmental language disorder. *Annals of Neurology*, 55(4), 530–540.
- Hresko, W. P., Reid, D. K., & Hamill, D. D. (1981). *Test of early language development*. Austin: Pro-Ed.
- Hresko, W. P., Reid, D. K., & Hamill, D. D. (1991). *Test of early language development* (2nd ed.). Austin: Pro-Ed.
- Hresko, W. P., Reid, D. K., & Hamill, D. D. (1999). *Test of early language development* (3rd ed.). San Antonio: Pearson Assessment.
- Koegel, L. K., Carter, C. M., & Koegel, R. L. (2003). Teaching children with autism self-initiations as a pivotal response. *Topics in Language Disorders*, 23(2), 134–145.
- Ng, L., Cheung, H., & Xiao, W. (2010). False belief, complementation language, and contextual bias in preschoolers. *International Journal of Behavioral Development*, 34(2), 168–179.

- Paul, R. (2005). Assessing communication in autism spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Assessment, interventions, and policy) (Vol. 2, 3rd ed., pp. 799–816). Hoboken: Wiley.
- Ramos, M., & Ramos, J. (2007). *Test of early language development-3rd edition, Spanish*. Austin: Pro-Ed. <http://www.proedinc.com/customer/productView.aspx?ID=4008>
- Sorensen, R. J., & Bradley-Johnson, S. (1993). Book review: Test of early language development (2nd ed). *Journal of Psychoeducational Assessment*, 11(4), 370–374.
- U.S. Bureau of the Census. (1997). Statistical abstract of the United States: (116th edition) Washington, DC, 1996. <http://www.census.gov/compendia/statab/>
- Waterhouse, L., Morris, R., Allen, D., Dunn, M., Fein, D., Feinstein, C., et al. (1996). Diagnosis and classification in autism. *Journal of Autism and Developmental Disorders*, 26(1), 59–86.

Test of Early Language Skill

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Synonyms

[ELS](#); [ELS checklist](#)

Description

The *Early Language Skills Checklist* (ELS Checklist) is an observation-based assessment for use by preschool teachers and childcare workers for assessing language abilities in children between the ages of 3 and 5. The ELS Checklist consists of 59 items falling under four broad categories: Attention Control and Listening Skills, Receptive Language, Expressive Language, and Use of Language. The authors caution that the ELS Checklist is intended for assessing children whose first language is English and should not be used for making clinical decisions about children who speak English as a second language. The ELS Checklist uses both assessment tasks (i.e., elicitation procedures) and

naturalistic observations that are carried out over the span of 2–3 days within different communicative contexts. Total administration time is approximately 30 min. Items are scored as “yes” or “no” based on whether the child is able to do the task. If a child can perform the most developmentally advanced item within a set, the easier items do not need to be administered. In some cases, skills need to be observed on at least three occasions to ensure mastery before a score of “yes” is given.

Historical Background

The ELS Checklist was developed in Great Britain and designed for monitoring children’s progress in language development according to *Code of Practice on the Identification and Assessment of Special Educational Needs in England and Wales* (1996), and also in line with similar requirements put forth by Scotland and Northern Ireland. Only one edition of the ELS Checklist has been published to date, and it is currently out of print.

Psychometric Data

No psychometric data are provided.

Clinical Uses

The ELS Checklist provides information about general developmental expectations for each item. Teachers can use these guidelines to help identify children who may have significant language delays. Strengths and weaknesses in children’s language development can be identified by comparing results across the four categories assessed. In addition, performance can be compared across settings and communication partners (e.g., group vs. one-on-one interactions). Teachers can record children’s results on the Early Language Skills Profile across multiple administrations to track growth (or lack thereof).

The manual provides guidelines for setting goals and intervention ideas for promoting language development.

Although the ELS Checklist is described as an easy-to-use “observation-based” assessment for use by “nursery or reception staff,” many of the procedures include elicitation methods (e.g., asking the child to identify rhymes or body parts) or carefully engineered small group interactions (e.g., observing the child with two to three peers who are “neither too outspoken nor withdrawn,” while engaged in a specific type of activity). In addition, examiners are required to elicit language samples which are subsequently analyzed for verb tense use and sentence structure. The user manual recommends videotaping the language samples for later scoring. It seems that for most early childhood teachers operating within a typical classroom setting these procedures would not be feasible (e.g., engineering interactions, videotaping language samples). In addition, a high level of expertise in language assessment is needed to elicit the desired behaviors and perform the analyses (e.g., collect a high-quality language sample and identify verb tense use).

The ELS checklist requires that children between the ages of 3 and 5 be able to follow directions and, in many cases, provide verbal responses. Therefore, it may not be appropriate for children with ASD in this age range who are nonverbal and/or exhibit significant developmental delays. In addition, the ELS Checklist is currently out of print. Suitable alternatives for the assessment of language may include:

Vineland Adaptive Behavior Scales, 2nd Edition
Communication and Symbolic Behavior Scales
Preschool Language Scale, 5th Edition
Test of Early Language Development, 3rd Edition

See Also

- ▶ [Communication and Symbolic Behavior Scale](#)
- ▶ [Communication Assessment](#)
- ▶ [Test of Early Language Development \(TELD\)](#)
- ▶ [Vineland Adaptive Behavior Scales \(VABS\)](#)

References and Reading

- Boyle, J., & McLellan, E. (1998). *Early language skills checklist: Observation-based assessment for early education*. London: Hodder & Stoughton.
- Wright, S. H. (1999). Book review: Early language skills checklist: User's handbook. *Child Language Teaching and Therapy*, 15(3), 285.

Test of Early Mathematics Ability-3

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Description

The *Test of Early Mathematics Ability, Third Edition* (TEMA-3, Ginsburg and Baroody 2003), is a standardized, individually administered norm-referenced test of mathematics performance, with norms for children who are 3–8 years old. The test takes about 40 min to administer and is intended to tap both informal and formal mathematics. Informal mathematics involves skills and knowledge typically acquired outside of school, such as counting skills and understanding of relative magnitude (e.g., 7 is more than 3). Formal mathematics involves skills and knowledge that children usually learn in school, such as base ten concepts (e.g., identifying ones, tens, and hundreds in a written numeral) and procedures for adding or subtracting two-digit numbers with regrouping (e.g., calculating the correct answer for $23 + 49$ or $60 - 27$). It should be noted, however, that the test yields only a comprehensive (total) score, not individual subtest scores for different areas of math.

The TEMA-3 provides age equivalents, grade equivalents, percentile ranks, and standard scores that have a mean of 100 and a standard deviation of 15. For interpreting children's performance, standard scores or percentile ranks should be used. There are two forms of the test, A and B, with procedures to equate scores on the two forms

so that they can be used interchangeably (e.g., to pretest and posttest before and after intervention). The third edition of the test comes with a book of assessment probes and instructional activities. Educators can use the assessment probes to further pinpoint individual children's math weaknesses and the instructional activities to improve children's skills in specific math areas.

Psychometric Data

According to information from the test publisher, the standardization sample for the *TEMA-3* comprised 1,219 children with characteristics (breakdown of socioeconomic status, race/ethnicity, etc.) roughly comparable to those in the 2001 U.S. Census. Internal test reliabilities for various age groups are in the .90s, which are excellent; some alternate-form and test-retest reliabilities are a bit lower but still all in the .80s or .90s. The validity of the test as a measure of children's mathematical performance has been established experimentally (Brassard and Boehm 2007; Dumont and Willis 2007). The third edition of the test also includes numerous bias studies demonstrating that the test is not biased in terms of gender or ethnicity.

Clinical Uses

The *TEMA-3* is easy to administer and useful for identifying children ages 3–8 years old who are either mathematically advanced or at risk in mathematics. For this age range, the test could also be helpful as part of a broader comprehensive evaluation for special education, especially for children suspected of disabilities involving mathematics or, conversely, those believed to be mathematically gifted. In addition, the *TEMA-3* can be employed as an indicator of children's overall progress in a math intervention or special education program. Because of the lack of individual subtest scores, the test is somewhat less useful as a detailed math diagnostic measure or indicator of children's progress in specific math skills. However, consideration of specific test items and use

of the assessment probes can provide additional information beyond the overall *TEMA-3* score.

See Also

- [Age Equivalents](#)
- [Educational Testing](#)
- [Test Bias](#)
- [Validity](#)

References and Reading

- Brassard, M., & Boehm, A. (2007). *Preschool assessment: Principles and practices*. New York: Guilford.
- Dumont, R., & Willis, O. (2007). Test of early mathematics ability, third edition (test review). In C. R. Reynolds & E. Fletcher-Janzen (Eds.), *Encyclopedia of special education* (Vol. 3, pp. 1992–1993). Hoboken: Wiley.
- Ginsburg, H., & Baroody, A. (2003). *Test of early mathematics ability* (3rd ed.). Austin: Pro-Ed.

Test of Narrative Language

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Synonyms

[TNL 2](#)

Description

The Test of Narrative Language – Second Edition (TNL-2) is designed to assess the ability of an individual in understanding and relating stories from and to communication partners. Linking to the collective understanding of the development of narrative language and correlation to success in other aspects of communication, such as literacy, the TNL-2 supports the examiner in assessing

comprehension and expression of three types of narratives: scripts, personal narratives, and fictional narratives. The TNL-2 is normed for individuals age 4 years 0 months through 15 years 11 months and includes visual stimuli for six tasks which include a comprehension and production task for each of the three types of narratives. Additionally, with the exception of the script narrative, different topics are presented for the types of narratives. Comprehension questions for the script narrative include five literal and nine inferential questions related specifically to the problem outlined. The personal narrative questions include six literal and six inferential questions regarding the characters, events, and consequences from the story. Last, the fictional narrative questions include four literal and eight inferential regarding characters, events, and consequences, while also focusing on responding to why and how questions, thus requiring perspective and non-literal interpretation. Production requires the individual to verbally express narratives that include key thematic elements, linguistic devices of temporal and causal conjunctions, grammatical accuracy, and character dialogue. Slight variation between the narrative type is the direct retell of the script narrative, creating a story related to the presented pictures that may be familiar, and creating a fictional story that intentionally includes a topic and/or character to ensure lack of familiarity.

The TNL-2 tasks have no basal, ceiling, or time limits. There is indication of an average time of administration falling between 15 and 30 min. Additionally, the authors suggest encouraging examinees to not spend unnecessary time over specific comprehension questions and to expect that most individuals will be capable of planning the oral narrative within 1 min. Digital recording is recommended to assist with scoring.

Scoring of each task for both comprehension and production results in total raw scores which are translated into age equivalent, percentile rank, and scaled scores for a total comprehension and production performance rating. These are then combined to establish a composite performance referred to as the Narrative Language Ability Index, resulting in percentile rank and standard

score. Both the scaled scores for the comprehension and production subtest performance and standard score for the composite are translated into descriptive terms aligning to the following rubric: very poor, poor, below average, average, above average, superior, and very superior. Standard error of measurement are provided to assist with interpretation of the scores. Three areas of observation are recorded to assist in analyzing possible variables that could impact outcomes, including speech production, fluency, and voice. The authors provide additional information regarding potential floor and ceiling effects, indicating no ceiling effects and possibly mild floor effects at the 4-year-old level; thus scores at the lower range for this age group should be interpreted with caution. Lastly, the authors provide information and guidance regarding interpretation of the results and consideration of cultural influences.

Historical Background

Since the 1980s and 1990s, and perhaps earlier, the link between narrative language and cognition, socialization and academic achievement has been expressed in the literature (Mandler 1984; Merritt and Liles 1989; van Dijk 1981; Applebee 1978). Of note is the critical link narratives provide for children between the contextualized language of conversation and the decontextualized language of the classroom, possibly allowing for greater success in an academic setting (Paul et al. 2018). Individuals with language disorders may present with challenges in their use of narration, thus be disadvantaged in achieving success both socially and academically. This is particularly noted in individuals with autism (ASD). Individuals with ASD may evidence microstructure yet lack the macrostructure required to convey a clear and coherent narrative. For example, they may include irrelevant information (Norbury and Bishop 2003).

The challenge presented to the authors was identifying the best approach to assess narrative language that encompasses critical aspects of narration at both the micro- and macro-level while providing a standardized method of comparison to

same age peers. Most standardized instruments assess isolated aspects of language needed for narration and not narration as an outcome. Although there exists agreed upon characteristics of narrative language that include structure, modality, type of narrative, and the development of narrative in children, a measure that was standardized was still needed. Therefore, the authors developed the original TNL to meet this need. The first iteration of the TNL was first published in 2004 and designed to reduce the need of transcribing children's stories. The TNL provided explicit prompts that could ease this process of transcription while providing a standard approach to assessing narration. Based on feedback and current research, the authors began a process of revision in 2012 (Gillam and Pearson 2017). Improvements include new standardization data, larger normative sample, pictures to support all stories, pictures added color and are of higher interest for children, increased number of comprehension questions, scoring for production (oral narratives) is consistent across all, increased data regarding test validity, and diagnostic accuracy.

Psychometric Data

Standardization was established on a normative sample of 1310 children age 4 years 0 months to 15 years 11 months and representative of the United States per comparison of percentages with the ProQuest Statistical Abstract of the United States 2015 and the Digest of Education Statistics 2014 (as cited by Gillam and Pearson 2017). Stratification of selected demographic categories at each age interval further demonstrates normative representation (Gillam and Pearson 2017). Recruited examiners met specific indicated criteria, represented each of four US demographic regions, and were trained in the administration of the assessment.

Overall reliability for the TNL-2 exceeds the desirable level of a coefficient of above 0.80. The authors provide results to three types of correlation coefficients to express test reliability. A Cronbach's (1951) coefficient alpha revealed 0.81 for comprehension subtest, 0.87 for production subtest, and 0.90 for composite of Narrative

Language Ability. Collectively these reveal a strong level of reliability for the overall assessment of narrative language across examinees. Test-retest reliability revealed a coefficient of 0.93 for the composite of Narrative Language Ability and 0.85 and 0.82 for the Comprehension and Production subtests, respectively. Lastly, interscorer reliability resulted in a coefficient of 0.99 for composites, Comprehension and Production.

Regarding test validity, the authors provide extensive measures to assess and meet expectations of test validity. First they provide detailed explanation and rationale to meet content-description validity, revealing the literature regarding narrative type and both the comprehension and production option. Additionally, they included various quantitative measures that link to content-description validity. For example, a quantitative item discrimination correlation revealed satisfactory to larger than satisfactory correlation between items and overall test score. The authors provide a criterion-prediction validity analysis comparing the TNL-2 subtest and composite raw scores with The Systematic Analysis of Language Transcripts software (SALT; Miller et al. 2011). SALT is used to calculate word-level analysis, sentence-level analysis, and story analysis of a narrative. Recordings of 142 of the children samples were selected for this transcription and language analysis using their Alien's story. Correlations between SALT scores and the TNL-2 ranged from moderate on the comprehension subtests to large on both production and composite scores, revealing scores on the TNL-2 as related to the types of language measures commonly applied to narrative language samples. Additional information regarding performance by differing diagnostic groups reveals the capability of the TNL-2 to correlate to those groups known to have compromised language as a result of their diagnosis.

Clinical Uses

The outcomes from the TNL-2 can provide initial understanding of the individual's language skills

underlying their narrative comprehension and production. The outcomes can be used to indicate general estimate of language ability and be a guide for further assessment and evaluation. The assessment may be beneficial to assessing growth overtime as well. Use of the assessment to select long-term goals is recommended and not recommended for short-term objectives. It may be possible for the examiner to conduct an error analysis of the individuals produced narratives to identify macrostructure and microstructure strengths and challenges to inform short-term objectives.

See Also

- ▶ [Narrative Assessment](#)
- ▶ [Narrative Language in ASD](#)
- ▶ [Strong Narrative Assessment Procedure \(SNAP\), Second Edition](#)

References and Reading

- Applebee, A. (1978). *The child's concept of story*. Chicago: University of Chicago Press.
- Cronbach, L. J. (1951). Coefficient alpha and the internal structure of tests. *Psychometrika*, 16, 297–334.
- Gillam, R. B., & Pearson, N. A. (2017). *Test of narrative language* (2nd ed.). Austin: Pro-ed.
- Mandler, J. M. (1984). *Stories, scripts, and scenes: Aspects of schema theory*. New York: Psychology Press.
- Merritt, D. D., & Liles, B. Z. (1989). Narrative analysis: Clinical applications of story generation and story retelling. *Journal of Speech and Hearing Disorders*, 54, 438–447.
- Miller, J. F., Andriacchi, K., & Nockerts, A. (2011). *Assessing language production using SALT software: A clinician's guide to language sample analysis*. Middleton: SALT Software.
- Norbury, C. F., & Bishop, D. V. M. (2003). Narrative skills of children with communication impairments. *International Journal of Language and Communication Disorders*, 38(3), 287–313.
- Paul, R., Norbury, C. F., & Gosse, C. (2018). *Language disorders from infancy through adolescence* (5th ed.). St. Louis: Elsevier.
- van Dijk, T. A. (1981). Episodes as units of discourse analysis. In D. Tannen (Ed.), *Analyzing discourse: Text and task*. Washington, DC: Georgetown University Press.

Test of Nonverbal Intelligence (TONI-4)

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Synonyms

TONI

Description

The Test of Nonverbal Intelligence, Fourth Edition (TONI-4), is an individually administered cognitive assessment instrument constructed in 2010 to assess general intellectual functioning. The test may serve to support an understanding of intellectual impairment, treatment therapy, or any special service needs. This tool is able to eliminate complex language and motor requirements that typically influence the results of more traditional measures of intellectual ability. This test is not designed for those who may be visually impaired. This assessment tool, published by Pro-Ed in Austin, Texas, can be administered individually in 15–20 min to children aged 6:0 and adults aged 89:11. Results exhibit skill areas measuring intelligence, aptitude, abstract reasoning, and problem solving. The publishers offer this “language-free” test as an alternative assessment measure for those who may be challenged with language, hearing, and/or motor abilities. This may also help to serve populations of individuals who may be unfamiliar with American culture. Directions may be delivered in English, Spanish, French, German, Chinese, Vietnamese, Korean, and Tagalog. In the complete test kit, there is a manual, picture book, critical reviews, and research findings (1982–2009), Form A and Form B record forms.

The first 19 questions are designed for ages 6–9 years, and the remaining questions are

designed for ages 10 and older. Each correct response is scored as 1 point and incorrect responses are scored with a 0. Each item contains one or more of the eight salient characteristics:

1. Shape
2. Position
3. Direction
4. Rotation
5. Contiguity
6. Shading
7. Size
8. Movement

A basal is achieved when the highest level at which an individual scores five consecutive correct responses is recorded. A ceiling is achieved when the third error out of five consecutive responses is recorded. Both TONI-4 forms can be used for research purposes and for periodic reevaluation. Scores are available as index scores where the mean is 100 and the standard deviation is 15. Percentiles, age equivalents, and descriptive terms (IQ greater than 130 = very superior, 121–130 = superior, 111–120 = above average, 90–110 = average, 80–89 = below average, 79 and below = poor) are presented. The manual provides a corresponding descriptive rating with each index score interval.

The oral instructions are simply stated. Individuals being administered the TONI-4 may respond with gestures including pointing, nodding, and blinking, thereby making this test available to both language-challenged and motorically challenged individuals. The 60 TONI-4 test items are arranged, as with other cognitive assessment tools, in an easy-to-difficult order. The authors have chosen items intended not to be influenced or challenged by educational, cultural, or experiential backgrounds. This assessment measure then can provide cognitive information for a diverse population of both children and adults for whom language and/or motor challenges may exist. The TONI-4 can be accessible for a wide range of varying needs and allow examiners and test takers to achieve results in a relatively short period of time.

Historical Background

The TONI-4 exhibits some decrease in cultural language factors which often influence verbally based tests. This 2010 updated current version of the TONI-3 has newly added several major non-English languages for non-English examinees who do not require nonverbal instructions. This is an important change allowing this cognitive measure to be administered with verbal instructions to a variety of diverse and/or multicultural individuals. These expanded languages could offer support for this tool to be a potentially welcome addition in a highly diverse school district.

In this most recent version of the Test of Nonverbal Intelligence (TONI-4), there is new normative data which has been collected and stratified. There is also the addition of six items from the TONI-3 to increase the level of challenge of the assessment measure. These items were added to address the previous concern which found that the test was not well suited for those who may be found to be in the gifted and talented population. The new items are stated to have improved the floor of both forms. Items have been reordered to balance the forms in the TONI-4.

The TONI-4 Kit includes:

Examiner's manual

Picture book

Critical reviews and research findings
(1982–2009)

50 Form A answer booklets and record forms

50 Form B answer booklets and record forms

Sturdy storage box

Some potential weaknesses of the TONI-4 emerge when considering that this assessment tool is designed for those who may be culturally or linguistically challenged. These notations include a small (23%) normative sample of individuals who were administered the test using the nonverbal instructions. Again, given that this tool is designed for those who may not be fluent with language, it is important to consider that the information gathered from the sample size is largely based on responses delivered through a verbal mode. Seventy-seven percent of participants in

the sample size utilized the verbal format. Further, none of the sample participants were given the non-English verbal formats of the test within the sampled population. Perhaps additional numbers within a sample size for the future, both within the population of nonverbal and within the population on non-English speakers, may be considered given that these persons are those for whom the test is especially designed to assess accurately. However, it is critical to note that stratification for the TONI-4 illustrates percentages which are consistent with the representative population of the United States.

Psychometric Data

Test Construction

The TONI-4 is an individually administered cognitive assessment tool which examines problem solving skills in a range of persons from 6 years 0 months through 89 years, 11 months. Each item contains features: shape, position, direction, rotation, shading, size, and movement. The more differences, the more complicated types and the greater number of rules the item has. Each form, A and B, has 60 items by adding the beginning and the end of the test to reduce the floor and ceiling effects, and the two were equally allocated in item similarity and difficulty by sorting all the items in difficulty.

Clinical Uses

The TONI-4 may serve as a strong and viable alternative as an individually administered cognitive measure for those individuals who may be challenged by language, hearing, motor, or American culture. Where instruments such as a WISC-IV, WAIS-IV, SB5, or WJ Cognitive Assessment do not suit, the TONI-4 may allow a closer look at individual abilities, especially problem solving and abstract reasoning, and support any diagnosis regarding intellectual functioning. The TONI-4 can be a strong alternative to other more verbally laden tools of individual assessment. It could also be utilized to support a special services need. To

utilize and interpret the TONI-4, test administrators need formal training and a fundamental understanding of mental ability testing and measurement.

Test Construction

The TONI-4 is an individually administered measure used to assess problem solving without a requirement of the testee of a language or motor skill. Each item contains features of shape, position, direction, rotation, shading, size, and movement. The greater the difficulty, the more complicated types and the greater the number of rules each item contains. Examiners can use this tool to help shape hypotheses for learning and planning that do not bias children or adults with language or culturally laden items. Indeed, the directions may be administered in a variety of languages.

There are two test forms: form A and form B. Each contains 60 items. Adding items at the beginning and at the end of the two forms was done to reduce the floor and the ceiling. Effects of the two were equally allocated in item similarity and difficulty by sorting all the items by difficulty. If three errors occur within five items, the last incorrect item is the ceiling. Conversely, with five correct responses, the last item is the floor item. If a test-taker cannot understand the expectation after two times of a training item, the testing should be ceased. Otherwise, an examiner would discontinue testing once a ceiling has been reached.

Item Analysis

A point-biserial correlation between each item and the total scores resulted in enough magnitude for positive content validity. Item difficulty averaged about 50% with items ranging between 15% and 85% which is an acceptable degree for item discrimination. The distribution was evenly spread.

Item Response Theory

A logistic regression was used on all test items. Three dichotomous groups were compared by effect sizes (e.g., male vs. female, African American vs. non-African American, and Hispanic

vs. non-Hispanic). The results show the items are unlikely to be biased. Items that did show moderate (34–70%) or large bias (greater than 70%) were deleted and replaced with other nonbiased items.

Standardization Sample

The robust standardization sample included 2,272 participants from 31 of the United States. Those states were further broken down and examined by regions within the United States including: South, West, Northeast, and Midwest. All assessments were performed by experienced trained specialists. The verbal instructions were delivered in English, and this form was used for 77% of the normative sample. The remaining used the nonverbal instructions provided. The sample was proportionally stratified relative to the United States population at the time the test was developed. Age, race, gender, location, parental education, and socioeconomic status were all considered, each of which was hypostasized to potentially influence cognitive ability.

Reliability and Validity

Test-retest ability showed ($N = 63$) consistency for both forms. This was true for immediate and delayed administrations. Interrater reliability showed strength among experts who administered the tool. These findings may be interesting for those who may wish to use this tool to update their understanding of an individual who is language challenged and where planning for needs may be better understood.

Strengths

The first strength of the TONI-4 is that it can be administered with clear and easy-to-understand instructions in 15–20 min to a diverse array of children and adults aged 6–89 years. That a cognitive assessment instrument can be administered in a short period of time to such a wide range of individuals is an asset by itself. Beyond this, however, it is possible to provide oral directions in a variety of languages serving the linguistically diverse national population as well as nonverbal directions to those who may benefit to grasp without a language requirement. Test-taking responses

do not have motor or language-based requirements, making this again accessible to a wide range of individuals with varying needs.

The manual for the TONI-4 provides instructions that are both verbal and nonverbal. There is evidence of a decrease in the cultural factors. There is new normative data that has been collected and stratified and six items were added to increase the measure's level of challenge. This is in response to earlier critique that the tool did not assess for those qualified as gifted and talented. These items improved the floor of both forms. Items were also reordered to balance the forms. In short, this tool may reach a population subset of individuals that have been a challenge to assess with accurate well-normed tools previously.

Weaknesses

The normative sample was tested with the English version. 23% of the sample was administered the test using the non-verbal instructions. Therefore, some of the stratification subgroups may be perceived to be small (e.g., exceptionality subgroup 13% and adults with disability 7%); however, it is noted that this was representative of the United States population at the time the norming was completed. Still, with smaller groups, there may be some question about the reliability and validity. Additional numbers may be useful for the future given that the test authors are offering this tool to support certain populations.

Summary

The TONI-4 is considered to be user friendly. It can be a time-efficient cognitive measure that can be individually administered to a multicultural and physically diverse population of children and adults aged 6–89 years. This cognitive measure does not require language or advanced motor skill of the participant. Visual skills are required. The TONI-4 could be seen as a potential tool for schools to utilize who have diverse student populations. Those taking the test have the option of responding with simple gestures such as pointing, nodding, or blinking which opens up opportunities to use this tool with diverse

populations of learners. Professionals, then, can consider this assessment as part of a battery to examine general intellectual functioning, diagnose intellectual impairments, and verify referrals for special services and for research purposes.

See Also

- Behavior
- Education
- Occupational Therapy (OT)
- Special Needs

References and Reading

- Brown, L., Shechenou, R. J., & Johnsen, S. K. (2010). *Test of nonverbal intelligence-4 (TONI-4)*. Test review. Austin: PRO-ED.
- Ritter, N., Kilinc, E., Navruz, B., & Bae, Y. (2011). Test review: L. Brown, R. J. Sherbenou, & S. K. Johnsen Test of Nonverbal Intelligence-4 (TONI-4). Austin, TX: PRO-ED. *Journal of Psychoeducational Assessment*, 29, 484–488. <https://doi.org/10.1177/0734282911400400>.

Test of Playfulness (ToP)

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Synonyms

ToP

Description

The Test of Playfulness (ToP) is an assessment designed for measuring the play of individuals between the ages of 6 months and 18 years (Bundy 2010). It is composed of 29 items that are scored following an observation of the individual's free play in both indoor and outdoor

settings. It has been recommended that the ToP is most reliable when completed after play sessions that are 15 minutes in length. The ToP is completed by a trained observer. Items are scored on a 4-point Likert scale with respect to three dimensions: Extent (0 = rarely or never, 3 = almost always), Intensity (0 = not, 3 = highly), and Skillfulness (0 = unskilled, 3 = highly skilled).

Scoring the ToP utilizes a test-specific Keyform, which depicts the relative difficulty of each item plotted against the means and standard deviations for the items. Recording the item scores on this form produces a total score ranging from −7 to 7.

Historical Background

The initial version of the ToP was a 60-item assessment completed by a rater who scored from videotaped segments of free play. It has undergone three revisions, leading to the current version (Version 4). The first and most significant revision took place in 1993. Items were eliminated from the previous version. Those that were eliminated were items in which the statistical analyses indicated did not contribute toward the construct of playfulness or were difficult to observe reliably. New items were also added in order to increase the ability of the measure to detect subtle changes as a result of intervention. In addition, the scoring procedure was revised to include the Intensity and Skillfulness scoring scales. The second revision took place in 1997, which consisted of subtle revisions to the item set, with a subsequent minor revision clarifying scoring criteria. The most recent revision to ToP was completed in 2000. This revision came about in response to the growing number of examiners who were using the ToP to assess children with autism spectrum disorders (ASD). The goal of this revision was to make the ToP more sensitive to changes in playfulness as a result of intervention, as well as to add a greater number of easier items to adequately capture the play skills of children at lower levels of functioning. Seven new items were added to address this goal.

Psychometric Data

Throughout the development of the ToP, data have been collected from nearly 2000 children. Rasch analysis was used to validate the measure. This type of analysis measures the extent to which the difficulty of items corresponds to the ability of the individual. In other words, this analysis indicates whether more capable individuals (in this case, more playful) have higher scores on the ToP. Data from 96% of the items, 93% of the participants, and 95% of the raters have met this assumption, thus indicating that the ToP items reflect a unidimensional construct of playfulness.

The test-retest reliability of the ToP has been examined in one published study (O'Brien and Shirley 2001). Overall, it appears that the stability of the test scores is high. However, some variability in scores is expected as a function of the environment in which the play observations are conducted. In situations with several potential play activities or partners, the reliability of the test scores have been found to be lower (Skard and Bundy 2008). Researchers have suggested that if a child is to be evaluated on the ToP twice (e.g., to assess change in playfulness following an intervention), the child should be alone or with a playmate in both assessments in order to increase consistency between the play settings.

Clinical Uses

The ToP was developed as a tool to be used by occupational therapists as a formal assessment of the playfulness of their clients. The authors of the ToP indicate that it should be used by clinicians who desire to assess and promote play and playfulness in their young clients. Although it was originally intended to be used by occupational therapists, members of other professions are encouraged to use the ToP in assessments that would benefit from an evaluation of playfulness. As play skills are often impaired in children with ASD, the ToP may be a useful addition to the assessment of this population (Baranek et al. 2005).

See Also

- [Play](#)
- [Play Intervention](#)

References and Reading

- Baranek, G. T., Parham, L. D., & Bodfish, J. W. (2005). Sensory and motor features in autism: Assessment and intervention. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 831–861). Hoboken: Wiley.
- Bundy, A. (2010). *Test of playfulness manual*. Boulder: Colorado State University.
- O'Brien, J. C., & Shirley, R. J. (2001). Does playfulness change over time? A preliminary look using the test of playfulness. *Occupational Journal of Research*, 21, 132–139.
- Skard, G., & Bundy, A. C. (2008). Test of playfulness. In L. D. Parham & L. S. Fazio (Eds.), *Play in occupational therapy for children* (2nd ed., pp. 71–93). St. Louis: Mosby Elsevier.

Test of Pragmatic Language-2 (TOPL-2)

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Synonyms

[TOPL-2](#)

Description

The *Test of Pragmatic Language, Second Edition* (TOPL-2; Phelps-Terasaki and Phelps-Gunn 2007), is a standardized, norm-referenced test designed to effectively assess pragmatic language ability in students and provide in-depth, comprehensive analysis of social communication in context. Its principal uses are to identify individuals with social language deficits, determine individual strengths and weaknesses, develop a remedial

program, assess metapragmatics, and provide a tool for continued research in pragmatic language skills.

In the pragmatic language model of the TOPL-2, social communication is represented as an interconnected function of a student's ability to appropriately analyze, respond, and integrate situational, discourse, and semantic contexts of language (Phelps-Terasaki and Phelps-Gunn 2007). The TOPL-2 uses narrative and story contexts that reflect the dynamics of daily social interaction typically experienced by school-age children. Verbal and picture prompts are used to create a story or problem situation, and the student is asked to generate responses that make sense, fit the situation and context, and are likely to be successful, which is to say that they "work" effectively.

The TOPL-2 is developed for use with individuals between the ages 6 and 18 years. It can be individually administered in 45–60 min. The TOPL-2's 43 items measure various aspects of pragmatics (i.e., physical context, audience, topic, purpose, visual-gestural cues, and abstractions). Nineteen of the 43 items contain a pragmatic evaluation (PE) subcomponent, which measures the student's ability to monitor and appraise their own social language usage. The test's 43 items combine to produce a standard score, called the Pragmatic Language Usage Index. Administration of the PE items is optional but gives the examiner further information which may be used in individual educational plans and remedial plans.

Historical Background

The *Test of Pragmatic Language* (TOPL) was published in 1997 in response to the need for a standardized assessment instrument to measure the use of language in a social context. The TOPL-2, published in 2007, made improvements and refinements of the test based on new research in social language, new ideas for more in-depth layers of evaluation, and user feedback on the first edition of the test.

The TOPL-2 model is based on Norris and Hoffman's (1993) model of pragmatic language

known as the Situational-Discourse-Semantic (S-D-S) model. The S-D-S model serves as a framework for conceptualizing language in school-aged children. Language functioning in the S-D-S model falls under three contexts: situational context, discourse context, and semantic context.

The TOPL-2 authors adapted Norris and Hoffman's model and further developed subcomponents for each S-D-S context. The situational context represents the physical environment and the audience to which the conversation is directed. The discourse context concerns the function and effectiveness of language and is represented by the topic and purpose subcomponents of the TOPL-2 model. Semantic context refers to the aspects of language that are used to convey ideas or concepts. Semantic concepts in the TOPL-2 model have three subcomponents: visual-gestural cues, abstract language, and pragmatic evaluation.

Psychometric Data

The TOPL-2 was normed on a representative sample of 1136 students reflective of US school-age children. Data were matched to US Census reports on race, gender, rural or urban residence, ethnicity, family income, parent education, and disability. Raw scores on the TOPL-2 can be converted to standard scores (mean of 100 and a SD of 15), percentile ranks, and age equivalents.

The reliability of the TOPL-2 was estimated using Cronbach's coefficient alpha (content sampling), test-retest (time sampling), and correlation between scorers (scorer differences). The average coefficient alpha for the Pragmatic Language Usage Index at 13 age intervals yields an average reliability coefficient of .91. The TOPL-2 test-retest coefficients exceed .90, showing good time stability. TOPL-2's interscorer reliability was .98.

The TOPL-2's validity was estimated using content-description, criterion-related, and constructed-identification methods. Items of the TOPL-2 were written to reflect Norris and Hoffman's (1993) S-D-S model and rated by 12 experts regarding the relevance of each item to subcomponents of the TOPL-2. Item analysis

studies indicated a median item difficulty of approximately .50 and median item discrimination coefficients which exceed .35 at every age. Items on the TOPL-2 were tested for bias using logistic regression procedures. Only one item indicated bias toward gender or ethnic subgroups.

Criterion-prediction studies compared examinee performance on the TOPL-2 with performance on the *Comprehensive Assessment of Spoken Language* (CASL; Carrow-Woolfolk 1999), *Pragmatic Language Skills Inventory* (PLSI; Gilliam and Miller 2005), and *Test of Adolescent and Adult Language, Fourth Edition* (TOAL-4; Hammill et al. 2007). The relationship between the TOPL-2 and criterion tests ranged from .80 to .97.

Other validity studies described the TOPL-2's relationship to age and IQ. The performance of various subgroups was analyzed as well. The correlation between chronological age and performance on TOPL-2 was .64. The performance of selected subgroups of gender, ethnic background, and exceptionality subgroups was within expectations. The correlation between the TOPL-2 index and the *Wechsler Intelligence Scale for Children, Third Edition* (WISC-III; Wechsler 1991) was .52.

Clinical Uses

TOPL was originally designed for use by speech-language pathologists. However, with the ever-increasing emphasis on social skills and conflict resolution in students, TOPL-2 test provides essential information for all team members: school psychologists, counselors, clinical psychologists, and special education teachers and specialists. These varied team members can use the TOPL-2 as part of a full individual evaluation and program planning.

The TOPL-2 can be used for a variety of purposes such as to identify students who have difficulty using social language, to determine a student's progress in pragmatic language development, to measure social language deficits in certain populations or to explore the relationship between social language ability and emotional

behavioral problems and academic performance, and to aid in evaluating specialized interventions.

References and Reading

- Brinton, B., & Fujiki, M. (1999). Social interaction behaviors of children with specific language impairment. *Topics in Language Disorders, 19*, 49–69.
- Brinton, B., Fujiki, M., & McKee, L. (1998). The negotiation skills of children with specific language impairment. *Journal of Speech, Language, and Hearing Research, 41*, 927–940.
- Carrow-Woolfolk, E. (1999). *Comprehensive assessment of spoken language*. Circle Pines: American Guidance Service.
- Gilliam, J., & Miller, L. (2005). *Pragmatic language skills inventory*. Austin: PRO ED.
- Hammill, D. D., Brown, V. L., Larsen, S. C., & Wiederholt, J. L. (2007). *Test of adolescent and adult language* (4th ed.). Austin: PRO ED.
- Nippold, M. (1994). Persuasive talk in social contexts: Development, assessment, and intervention. *Topics in Language Disorders, 14*, 1–12.
- Nippold, M. (2000). Language development during the adolescent years: Aspects of pragmatics, syntax, and semantics. *Topics in Language Disorders, 20*, 15–28.
- Norris, J., & Hoffman, P. (1993). *Whole language intervention for school-age children*. San Diego: Singular.
- Ochoa, S. H. (1995). Review of the test of pragmatic language. In J. C. Conoley & J. Impara (Eds.), *The twelfth mental measurements yearbook* (pp. 1059–1060). Lincoln: The University of Nebraska Press.
- Phelps-Terasaki, D., & Phelps-Gunn, T. (1992). *Test of pragmatic language*. Austin: PRO-ED.
- Phelps-Terasaki, D., & Phelps-Gunn, T. (2007). *Test of pragmatic language development* (2nd ed.). Austin: PRO-ED.
- Wechsler, D. (1991). *Wechsler intelligence scale for children* (3rd ed.). San Antonio: Psychological Corp.

Test of Sensory Functioning in Infants

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Synonyms

TSFI

Description

This assessment tool offers an objective method to examine the sensory functioning of infants aged 4–18 months. In an effort to provide an objective method to determine whether, and to what extent, an infant has sensory processing deficits, this tool provides a score for each of the 24 items. These test item scores are then calculated to establish an overall score of sensory processing and reactivity (total test score), as well as a score within each of the following sensory subdomains of sensory functioning:

1. Reactivity to tactile deep pressure
2. Visual tactile integration
3. Adaptive motor function
4. Ocular motor control
5. Reactivity to vestibular stimulation

The above subdomains were identified for inclusion in this tool for specific applicability to infant behavior. For example, measuring how an infant responds when touched in sensitive areas (i.e., soles of feet, stomach, or mouth) is important as tolerance to touch experiences is critical for mother-infant interaction. Additionally, a child who does not tolerate touch (reactivity to tactile deep pressure) or movement experiences (reactivity to vestibular stimulation) “may avoid tactile contact, being held and moved in space, and may avert his or her gaze to avoid face-to-face interaction” (DeGangi and Greenspan 1989, p. 4). Observing how a child visually locates and tolerates the touch of various objects is important as these are some of the first steps to object exploration and then creating plans of action on how to manipulate an object.

It is suggested that a practitioner (i.e., pediatrician, psychologist, infant educator, occupational therapist, physical therapist), with a baseline understanding of sensory processing, allows 2 h to learn the items prior to implementation. The assessment manual is easy to follow, and the specific instructions for item implementation are outlined with pictures to assist. A score of 0, 1, 2, or 3 is received depending on the child’s response to each item. The higher the

score indicates a more integrated, organized, or normal response. Lower scores qualify other responses as no response, partial response, disorganized response, hypo-reactive response, or adverse or mildly defensive reaction. The score tallies in each subdomain then result in a “normal,” “at risk,” or “deficient” score profile. It should be noted that at the time of development, 1989, practitioners only had their clinical judgment to determine if sensory issues were present in an infant. Thus, this criterion-referenced assessment tool was a marked improvement for the time and thus assisted in clinical practice as well as research.

Historical Background

Well-known and respected early childhood specialists, Georgia DeGangi, Ph.D., OTR (occupational therapist who now practices in clinical psychology) and Stanley Greenspan, MD (who later developed the Floortime Approach™), developed this objective tool to observe and measure the sensory processing abilities of infants. At the time, the only formalized measurements of children of this age were to measure motor and neurological functions but not the possible underlying sensory dysfunction which resulted in these identified motor or perceptual difficulties. The thought, based on sensory integration theory (Ayres 1964, 1972, 1979), was that intervention directed at potential underlying sensory processing difficulties would be most effective in addressing any resulting motor or perceptual deficits. A fuller description of Ayres’ theory of sensory integration can be found in the links below titled ► [“Ayres, A. Jean”](#), ► [“Sensory Processing”](#), and ► [“Sensory Integration \(SI\) Therapy”](#).

Psychometric Data

The test manual specifically outlines each step of the sampling and statistical procedures. However, it should be noted that there were some sampling difficulties specifically associated

with distinguishing typical irregularities (i.e., colic which might be noted up until 6 months of age) from clinical issues measured by the assessment.

Table 1 outlines the components of the assessment process including the psychometric procedures associated with the development of this criterion-referenced assessment tool. In summary, the total test score can be used reliably and validly for screening decisions, and the subtests can be used reliably and validly under certain age or diagnostic conditions based on the following information:

- Content validity: The test has a moderate to high degree of content validity.
- Construct validity:
 - Due to false error rates, the test should not be used on infants with delays or regulatory disorder until the infant reaches 10 months of age (DeGangi and Greenspan 1989, p. 29).
 - The subdomains of behavior investigated by the TSFI were distinctly different from other scales and assessments available at the time of development.
- Interobserver reliability: There were high levels of consistency in the identification of the classification designated for each item.
- Test-retest reliability:
 - Stability was good for visual tactile integration, ocular motor control, and the total test score.
 - Stability was fair for reactivity to tactile deep pressure.
 - Stability for reactivity to vestibular stimulation and adaptive motor functions were not strong enough to base decisions on, and a retest of these items should occur to substantiate initial findings of normalcy or dysfunction.

Clinical Uses

Any assessment tool should be used in combination with other tools in order to gain the most

Test of Sensory Functioning in Infants, Table 1 TSFI assessment review

Test name: Test of Sensory Functions in Infants (TSFI)
Author(s): Georgia A. DeGangi, PhD, OTR and Stanley I. Greenspan, MD
Publisher: Western Psychological Services, 625 Alaska Ave, Torrance, CA 90503; 800-648-8857
Technical information: 2 h of practice before administering
Age range(s): 4–18 months
Assessment type: Criterion referenced
Reliability: 1. <i>Interobserver reliability</i> : Obtained through 2 observers (PT & OT) for the same set of responses; 41 infants participated within 7–12-month age ranges. Intraclass correlations for the subtests and total test were high with coefficients ranging from .88 to .99 2. <i>Decision-consistency reliability</i> : The p_o index of decision consistency was used to determine “the proportion of children classified as normal and delayed on repeated testings” indicating a degree of confidence for the decision (i.e., the stability of the decisions). A sample of 26 “normal” infants was tested twice using a 1–5-day interval of retests. The age range consisted of 4–18 months consisting of 10 boys and 16 girls. Utilizing five observers, observer errors of measurement were affected because the same observer was not always able to conduct the retest on the same infant. The p_o index displayed that the five subdomains and total test ranged from 81% to 96%, indicating high levels of overall classification consistency 3. <i>Test-retest reliability</i> : The Pearson product-moment was used to calculate the stability of individual scores between the test and retest (five observers). In addition, some evidence indicates that some sensory integrative functions may be unstable (Ayres 1980), and this may affect the stability of coefficients Results: Good: Visual tactile integration, ocular motor, and total test scores Fair: Reactivity to tactile deep pressure Poor: Reactivity to vestibular stimulation and adaptive motor functions Twenty of the 26 infants were between the ages of 4 and 6 months old, thus leading to more variability in responses. This concludes that more extensive research should take place
Validity: 1. <i>Content validity</i> : A two-stage judgmental review occurred to determine test validity. Congruence and representativeness were rated by eight judges. They rated the subtests of the assessment with regard to their ability to overall measure the infant population’s sensory functioning. Congruence was rated either as poor, moderate, or high for each item <i>Item-behavior congruence</i> : High ratings (75–85%) for item-behavior congruence for all items. Two of the eight judges rated moderate for the items of the vestibular

(continued)

Test of Sensory Functioning in Infants, Table 1
(continued)

stimulation and reactivity to deep pressure. One judge gave a low rating for the adaptive motor function test and the visual tactile integration test

Representativeness: High ratings (87%) for all subtests except for a rating of 75% which was used for the reactivity to vestibular stimulation. The concept of “stranger anxiety” was discussed as a possible influence on the vestibular score. Several items (several domains) were ultimately discarded as not sufficiently discriminating or differentiating the sample

2. *Construct validity:* Construct validity evidence was found within the item, subtest, and test and was based on criterion-group performance comparisons. The validity was found by computing a discrimination index displaying the difference between group item performance and the mean score for regulatory, normal, and delayed disorder groups. Subtest intercorrelations were computed to display evidence that each subtest measures a different subdomain of behavior and, thus, are worthy of administration

Item validity: Out of the 44 test items, 20 were taken out after item analysis since they did not discriminate between the groups of delayed and normal or regulatory disorder infants, or were not sensitive to the normal infants’ developmental status

Decision validity: The focus of these cut-off decision procedures was to minimize the false-normal error rate (thought to be the more serious type of error in the diagnostic or screening process). Results were as follows:

False-normal error rate across age ranges:

Reactivity to tactile deep pressure and reactivity to vestibular stimulation subtests: between 0% and 59%

Adaptive motor functions, visual tactile integration, and ocular motor control: between 29% and 100%

False-normal error rate for 4–6-month-old age range: 75% for the total test with adaptive motor functions and visual tactile integration subtests least effective as these functions are just beginning to emerge

False-normal error rate for 7–18-month-old age range: between 14% and 45% for the total test

False-delayed error rate was minimized by setting the cut-off scores: ranged from 1% to 29% for the various subtests and age ranges, and from 7% to 19% for the total test

Test structure: Low coefficients between subtests indicate that this subtest is measuring a distinctly different function. Results, as follows, furnish evidence of the benefit of administering all five subtests:

Very low coefficients were found for the correlations of reactivity to vestibular stimulation and other subtests

Very low coefficients were found for the correlations of reactivity to tactile deep pressure and the other subtests

High correlations of .40–.47 were found between the adaptive motor functions, visual tactile integration, and ocular motor control subtests. This is not all that surprising as each of these measures a motor response or a motor output to a sensory response

(continued)

Test of Sensory Functioning in Infants, Table 1
(continued)

All coefficients were relatively low, ranging from .02 to .47, indicating that each subtest is measuring distinctly different sensory functions from the others

Obtaining information: 24 items should be administered individually, and in one setting, items should be administered exactly as described in the manual

Family’s role in the assessment process: The caregiver must be present during administration to hold the child and/or assist if needed

Time to administer: 20 min

Time to score: 5 min

Materials included in the test kit? X yes no

Describe materials: hand puppets, tennis ball, yarn, paper, furry mitt, etc.

Testing procedures

Is the tool easy to learn and administer? X yes no

How much training or practice is required? 2 h

Who can administer the test? Designed for pediatricians, psychologists, infant educators, occupational therapists, and physical therapists. A background in sensory processing is recommended

Is the manual easy to follow/understand? X yes no

Are the forms easy to follow/understand? X yes no

Domains: Reactivity to tactile deep pressure, adaptive motor functions, visual tactile integration, ocular motor control, and reactivity to vestibular stimulation

Outcome: Each subdomain/subtest will identify or determine an adverse, mildly defensive, or integrative response or reaction. It can also be used in conjunction with other developmental tests to provide an overall indicator of the child’s developmental functioning

comprehensive picture of a child’s functioning. The TSFI was intended to provide information related to the five subdomains noted above as these categories of sensory functioning were thought to be the most impacting on the development of efficient sensory integration in infants. The intent was to administer this assessment to “infants with regulatory disorder, developmental delay, and those at risk for later learning and sensory processing disorders (e.g., high-risk premature infants)” (DeGangi and Greenspan 1989, p. 2).

This tool continues to be utilized today in clinical practice as it is a very structured and organized method to investigate the sensory

processing abilities in this age group. Other standardized tools attempting to reliably measure sensory processing in this age group include the *Sensory Profile – Infant/Toddler* (Dunn 2002), *Sensory Processing Assessment* (Baraneck 2010), and *Sensory Processing Measure – Infant/Toddler* which is currently in development by the authors of the *Sensory Processing Measure* (Parham et al. 2007) and *Sensory Processing Measure - Preschool* (Kuhaneck et al. 2010).

See Also

- Ayres, A. Jean
- DeGangi-Berk Test of Sensory Integration
- Evaluation of Sensory Processing
- Occupational Therapy (OT)
- Sensory Diet
- Sensory Integration (SI) Therapy
- Sensory Integration and Praxis Test
- Sensory Processing
- Sensory Processing Assessment
- Sensory Processing Measure

References and Reading

- Ayres, A. J. (1964). Tactile functions: Their relation to hyperactive and perceptual motor behavior. *American Journal of Occupational Therapy*, 18(1), 6–11.
- Ayres, A. J. (1972). *Sensory integration and learning disorders*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (1979). *Sensory integration and the child*. Los Angeles: Western Psychological Services.
- Baraneck, G. (2010). Sensory processing assessment for young children (SPA)©1999: Version 2.2: Research Version (3/15/2010).
- DeGangi, G. A., & Greenspan, S. I. (1989). *Test of sensory functions in infants (TSFI) manual*. Los Angeles: Western Psychological Services.
- Dunn, W. (2002). *The infant toddler sensory profile*. San Antonio: Psychological Corporation.
- Kuhaneck, M. H., Ecker, C. E., Parham, L. D., Henry, D. A., & Glennon, T. J. (2010). *Sensory processing measure-preschool (SPM-P): Manual*. Los Angeles: Western Psychological Services.
- Parham, L. D., Ecker, C., Miller-Kuhaneck, H., Henry, D. A., & Glennon, T. J. (2007). *Sensory processing measure (SPM): Manual*. Los Angeles: Western Psychological Services.

Test of Written Language

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Synonyms

Test of Written Language, Fourth Edition (TOWL-4)

Description

The Test of Written Language, Fourth Edition (TOWL-4; Hammill and Larsen 2009), is designed to be a comprehensive assessment of the writing abilities of students between the ages of 9 and 17 years and 11 months. The testing materials include a student response booklet (Forms A and B), a record/story scoring form and a supplemental practice scoring sheet, illustrations to elicit writing samples, and an examiner's manual.

The TOWL-4 is comprised of seven subtests intended to sample a wide range of writing competencies. Five subtests (Vocabulary, Spelling, Punctuation, Logical Sentences, and Sentence Combining) use a contrived format intended to assess isolated units of written language (e.g., use of punctuation, spelling from dictation). An additional two subtests (Contextual Conventions, Story Composition) follow a spontaneous format designed to assess a students' functional writing ability. "Such assessment focuses on evaluating the quality of actual passages composed by students. To write effectively in everyday life or school situations, students must be proficient in using all of the writing components" (Hammill and Larsen 2009, p. 4).

The test begins with the collection of the spontaneous writing sample. The student is shown a sample illustration (one that shows preparations for a birthday party where chaos has erupted). The administrator reads an "example of a good story" to accompany the picture and explains what

makes this a good story (e.g., it has a clear beginning, middle, and end; people in the story have names and emotions). The student is asked to write a story about another picture and to “try to make your story as interesting as you can.” Students are given 20 min (5 to plan and 15 to write) to finish the story.

The test continues with the administration of contrived contexts. Examples of each task are given to ensure that the student understands what is being asked. No time limits are imposed for these tasks but students are encouraged to progress rapidly and not spend too much time on any particular item. Items in each subtest increase in difficulty. Testing always begins with the first item and progresses until the ceiling (three incorrect responses in a row) is reached.

The first subtest, Vocabulary, includes 33 items. For each item, a written word is presented (it is never spoken), and the student is asked to incorporate the word into a sentence without changing it in any way (e.g., “ran” can be incorporated into “I ran to the store”). Subtests 2 and 3 (Spelling and Punctuation, respectively) are administered using 26 sentence dictation items (e.g., “You can go with me” or “Because of the confusion, she sought legal help”). Subtest 4, Logical Sentences, uses 22 sentences that contain some form of flawed logic. The child is asked to correct each sentence (e.g., “The dog moored loudly” should be changed to “The dog barked loudly”) by marking those changes on the form (e.g., crossing out, writing in). Subtest 5, Sentence Combining, includes 23 items. Each item consists of 2–4 sentences that can be combined into a single sentence (e.g., “Tom is big” and “Tom is a man” can be combined to form “Tom is a big man”). For each of the subtests comprising the contrived portion of the TOWL-4, correct answers are provided in the examiner’s manual. Well-reasoned guidance is also provided to help administrators make decisions for sentences that are difficult to score.

Two additional subtests are intended to evaluate the quality of the student’s spontaneous language sample gathered at the beginning of the test. Subtest 6, Contextual Conventions, contains 21 items which rate a number of contextual

dimensions. Each item is accompanied by either a nominal or ordinal scale. For example, the first item (“Sentences begin with a capital letter”) uses an ordinal 0 (three mistakes or more), 1 (one to two mistakes), or 2 (no mistakes). By contrast, item 6 (“Uses a question mark”) employs a nominal 0 (no) or 1 (yes) system. Subtest 7, Story Composition, is similar in design, but it contains 11 items intended to tap aspects of story composition (e.g., sequence, plot). These examples are ones in which there is a rather clear criterion to justify a rating. This is not true, however, for all items in the spontaneous format. For example, item 6 of the Story Composition subscale requires a rating for “Story Action or Energy Level (Pace).” Here, the administrator is asked to make a judgment of either 0 (plodding, stumbling, none), 1 (interesting, sustained), or 2 (exciting, compelling, exceptional). Unlike the scoring of the subscales for the contrived formats, little guidance is offered, and to the degree that these ratings are subjective, errors will be introduced during scoring. A summary of the subscales comprising the TOWL-4 is presented in the Table 1.

Scores can be generated for each subtest as well as composite scores for the contrived and spontaneous tasks, and an overall writing score. Scores are available in the form of raw scores, percentile ranks, grade and age equivalents, and scaled scores which are particularly useful for making comparisons to the normative sample.

Historical Background

The original TOWL (Hammill and Larsen 1978) was developed to respond to the need for a comprehensive test of written language. At that time, only the Picture Story Language Test (Myklebust 1965) was available and concern was growing over the outdated, unrepresentative normative data and the validity of the tool more generally (Hammill and Larsen 2009). Since its original publication, developers of the TOWL recognized the need to assess written language using both contrived and spontaneous formats. Contrived formats are desirable because they are more traditional and, thus, more familiar to test

Test of Written Language, Table 1 Description of subtests on the Test of Written Language

<i>Contrived formats</i>	Description
Subtest 1: vocabulary	The student is asked to incorporate a single written word into a written sentence (33 items)
Subtest 2: spelling	The student writes from dictation, and spelling is inspected for correctness (26 items)
Subtest 3: punctuation	The student writes from dictation, and punctuation is inspected for correctness (26 items)
Subtest 4: logical sentences	The student is asked to identify and correct logical errors within sentences (22 items)
Subtest 5: sentence combining	The student is asked to combine relatively short and simple sentences into a single, more complex, sentence (23 items)
<i>Spontaneous formats</i>	
Subtest 6: contextual conventions	The examiner rates the quality of the student's spontaneous narrative with regard to several writing conventions (e.g., correct capitalization) (21 items)
Subtest 7: story composition	The examiner rates the quality of the student's spontaneous narrative with regard to several dimensions of story composition (e.g., sequence) (11 items)

administrators. They are also well suited for isolating and assessing discrete units of written discourse (e.g., spelling, punctuation). On the other hand, spontaneous formats (i.e., written story elicitation in response to a visual stimulus) were also seen as essential to content validity. In analysis of the spontaneous format, the story can be scrutinized for the quality of its composition, creativity, and intellectual merits (Hammill and Larsen) which is not possible using the contrived format. This basic distinction between contrived and spontaneous formats for analyzing written language underlies the design of the original TOWL (Hammill and Larsen 1978) and all of its revisions (i.e., Hammill and Larsen 1988, 1996, 2009). What has changed with each revision is the number and content of the subscales that make up the contrived and spontaneous formats and the specific administration and scoring procedures that accompany them.

Although the original TOWL (Hammill and Larsen 1978) was generally described by reviewers as an ambitious and commendable undertaking and one that represented a real contribution, criticism was reported. Major questions surrounded the characteristics of the normative sample and whether the test met industry standards with regard to reliability and validity (Hammill and Larsen 2009). The TOWL-2 was undertaken as the first major revision of the test, and “reviews for the TOWL-2 were even more encouraging that those for the TOWL” (Hammill and Larsen 2009, p. viii). Nonetheless, some questions about the description of the normative sample persisted. New concerns were also raised about the time length required to administer the TOWL-2 and subjectivity when scoring the spontaneous subtests (Davis 1990; Jacobson 1991). As Hammill and Larsen (2009) attest, “in our efforts to improve the statistical and theoretical properties of the test, we inadvertently made it cumbersome to give and tedious to score” (p. ix). The next major revision, the TOWL-3 (Hammill and Larsen 1996) was developed to overcome these difficulties and strengthen reliability and validity. Reviewers of the TOWL-3 were generally supportive of the psychometric properties of the test and agreed that the TOWL-3 was useful as a research and clinical tool. On the other hand, questions were raised about the presence of ceiling and floor effects (e.g., Bucy and Swerdlik 1998) which seriously limited the usefulness of the test when given to older or younger children. Moreover, concerns about the subjectivity of scoring (particularly with regard to the spontaneous format) remained. Thus, “although the TOWL-3 substantially improved over previous versions, a degree of subjectivity remains in the scoring procedures” with many of the subscales requiring judgments that are “inherently subjective and likely to provide a significant source of error” (Hansen 1998, p. 1071).

In the examiner's manual of the TOWL-4, Hammill and Larsen (2009) describe how the current version seeks to improve upon the test in several ways. Improvements include the use of normative data representative of US census statistics, the availability of both grade- and age-based

norms, and more conclusive evidence as to the measure's reliability and validity. Unfortunately, the problem of ceiling and floor effects continues for individual subscales but the authors note that neither of these occurs for the composite scores (Hammill and Larsen 2009). The psychometric properties of the TOWL-4 are briefly described below.

Psychometric Data

The TOWL-4 was normed on a sample of 2,205 individuals (ages 9–17 years) living in the United States. Demographic data were evaluated with regard to geographic area, gender, ethnicity, income, parental education, and exceptionality status, including individuals with autism spectrum disorder (ASD; 1% of sample). Normative data for all categories are comparable to recent US census statistics.

Several types of reliability of the TOWL-4 have been documented. Internal consistency, or the degree to which items on a test measure the same content domain, indicates a moderate to high degree of consistency for subscale and composite scores. An evaluation of alternate-form reliability was needed to establish the equivalency of the A and B test forms. Good reliability was achieved. The test-retest reliability over a 2-week interval is also acceptable indicating good temporal stability. The TOWL-4 should also have adequate interscorer reliability to demonstrate that scores are stable when different individuals administer the test. This is particularly important given previous concerns about the subjectivity and difficulty in scoring the test, especially the Contextual Conventions and Story Composition subtests of the spontaneous formats. Although the lowest estimates occurred with the Story Composition subtest, acceptable interscorer reliability was found between two trained raters who were familiar with TOWL-4 scoring procedures.

The validity of the TOWL-4 has also been assessed. First, the TOWL-4 appears to have adequate content validity, which is the degree to which the test covers the intended content domain. Indeed, the content of the TOWL-4 was developed

in line with the writing development literature, and the developers are convincing in their justification of each of the seven subscales included in the TOWL-4. To further support content validity, item analysis was conducted. Only items with conservative discriminating power and difficulty levels between 15% and 85% were included. Convergent validity, based on the ability of TOWL-4 scores to correlate with scores on a variety of performance measures, is moderate to very large. For example, subscale and composite TOWL-4 scores are significantly correlated with other literacy (e.g., the Test of Reading Comprehension – Fourth Edition; Brown et al. 2009) and intelligence (e.g., the Wechsler Intelligence Scale for Children – Fourth Edition; Wechsler 2003) measures. The authors of the TOWL-4 have demonstrated criterion-related validity by obtaining the expected correlations between TOWL-4 scores, age, and grade level.

In summary, the TOWL-4 is highly regarded as a comprehensive assessment of written language. Of course, users should be aware of the features that complicate administration and interpretation of the measure. Chief among these are the presence of ceiling and floor effects for many of the individual subscales, which makes their use for both older and younger children problematic. In addition, administrators require a degree of practice and familiarity with the measure before it can be administered and scored efficiently and reliably.

Clinical Uses

The TOWL-4 was designed to serve as a comprehensive assessment of written language. More specifically, its purposes are to identify students who may need special help with writing, to document students' writing strengths and weaknesses, to guide education planning, and to monitor the effectiveness of programs designed to support writing skills. Despite inclusion of a small number of individuals with ASD in the normative sample, the TOWL-4 was not developed for use with this population. It is intended for students with developing writing skills who may be struggling to achieve literacy milestones typical of their age- or grade-matched peers.

Of course, this could apply to special populations including those with ASD, especially those with higher cognitive and language skills for whom writing development has been identified as a goal in the student's education planning. Thus, the TOWL-4 may have some utility for professionals working with students with ASD in an educational setting and could be administered as part of a larger battery of tests designed to inform education programming across verbal and non-verbal means.

Clearly, each clinical application raises questions to consider when determining the appropriateness of the TOWL-4 and interpretation of scores for special populations. One advantage of the TOWL-4 is that it includes alternate forms that may be particularly useful in evaluating the effects of education programming. However, one serious concern involves the presence of floor and ceiling effects that are known to affect TOWL-4 subscale scores. This makes the literal interpretation of all derived scores perilous for older and younger individuals (with and without ASD). When ceiling or floor effects occur, professionals are advised to consider only the composite and overall writing scores. Of course, derived scores need not be obtained at all. Although the inspection of raw scores does not allow for comparison to the normative group (which is not equivalent to a population of students with ASD), informal inspection of the patterns of performance across subtests to identify relative strength and weakness areas in the context of dynamic assessment seems more justified.

See Also

- [Academic Skills](#)
- [Achievement Testing](#)
- [Agraphia](#)
- [Asperger Syndrome](#)
- [Ceiling Effect](#)
- [Dysgraphia](#)
- [Functional Assessment](#)
- [High-Functioning Autism \(HFA\)](#)
- [Literacy](#)
- [Wechsler Preschool and Primary Scale of Intelligence](#)

References and Reading

- Brown, V., Wiederholt, J. L., & Hammill, D. D. (2009). *Test of reading comprehension* (4th ed.). Austin: Pro-Ed.
- Bucy, J., & Swerdlik, M. (1998). Review of the test of written language – fourth edition (TOWL-4). In J. C. Impara & B. S. Plake (Eds.), *The thirteenth mental measurements yearbook* (pp. 1072–1074). Lincoln: The Buros Institute of Mental Measurements.
- Davis, J. M. (1990). Test of written language-2. *Journal of Psychoeducational Assessment*, 8, 185–189.
- Hammill, D. D., & Larsen, S. C. (1978). *Test of written language*. Austin: Pro-Ed.
- Hammill, D. D., & Larsen, S. C. (1988). *Test of written language* (2nd ed.). Austin: Pro-Ed.
- Hammill, D. D., & Larsen, S. C. (1996). *Test of written language* (3rd ed.). Austin: Pro-Ed.
- Hammill, D. D., & Larsen, S. C. (2009). *Test of written language* (4th ed.). Austin: Pro-Ed.
- Hansen, J. B. (1998). Review of the test of written language – fourth edition (TOWL-4). In J. C. Impara & B. S. Plake (Eds.), *The thirteenth mental measurements yearbook* (pp. 1070–1072). Lincoln: The Buros Institute of Mental Measurements.
- Jacobson, J. E. (1991). Test of written language-2 (TOWL-2). *Journal of Reading*, 34, 663–665.
- Myklebust, H. L. (1965). *Picture story language test*. New York: Grune & Stratton.
- Wechsler, D. (2003). *The wechsler intelligence scale for children* (4th ed.). San Antonio: Psychological Corporation.

Test of Written Language, Fourth Edition (TOWL-4)

- [Test of Written Language](#)

Thalamus

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Definition

The thalamus is a brain structure located just above the midbrain near the center. It is a paired structure

(one in each hemisphere of the brain) shaped like small elongated spheres. Composed of many subregions each with unique functions and connections in the brain, the thalamus serves many different roles. One primary role is as a relay station between sensory input and the brain's primary sensory processing regions. All sensory input except olfaction stops first at the thalamus before reaching the neocortex. One major theory for thalamic function describes the thalamus as a sensory gating structure, in which sensory information is processed and filtered, perhaps for relevance, before proceeding to the brain (McCormick and Bal 1994). In addition to relaying sensory information to the cortex, the thalamus also receives top-down input from frontal cortex, which then proceeds to vast brain areas, including sensory association regions. The thalamus also plays an important role in regulating motor behavior. Finally, the thalamus is involved in regulating arousal and wakefulness through connections with the cortex, which may be involved in consciousness. Lesions to the thalamus can cause patients to enter into comatose states, as well as affecting motor, sensory, attention, and higher cognitive functions.

References and Reading

McCormick, D. A., & Bal, T. (1994). Sensory gating mechanisms of the thalamus. *Current Opinion in Neurobiology*, 4(4), 550–556.

Thalidomide

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Synonyms

[N-Pthaloylglutamide](#)

Indications

Thalidomide is a potent teratogen.

Because of observed potential to increase tumor necrosis factor alpha production in vivo and interleukin-2 and interferon gamma in vivo, thalidomide has limited utility in the dermatologic manifestations of HIV, Behcet's disease, and graft-versus-host disease and is now being investigated for use in myelodysplastic disorders.

Mechanisms of Action

The teratogenic effect may be related to vascular disruption. As noted, it impacts immune function including increasing production of tumor necrosis factor alpha in vitro and interleukin-2 and interferon gamma in vivo.

Specific Compounds and Properties

The chemical name is *N*-pthaloylglutamide.

Clinical Use (Including Side Effects)

Thalidomide was initially developed in 1954 and licensed in 40 countries around the world for treatment of anxiety, insomnia, gastritis, and vomiting. It was marketed as safe for use in pregnancy. It was never approved by the Food and Drug Administration in the USA. By the early 1960s, increasing numbers of children were identified in Europe who were born with limb deformities. This, and eye movement abnormalities, was associated with thalidomide use by the mother between days 20 and 36 of pregnancy. Many years later, ophthalmologists Drs. Judy Miller and Kristen Stromland noticed that there was an increased rate of autism among individuals exposed to thalidomide in utero who were participating in their studies related to eye movement. This observation led to the association of autism with an environmental teratogen. Dr. Patricia Rodier worked out that this was likely to be due to impact on early development of the brain stem and resulted in her development of an animal model for early brain injury based on

valproic acid exposure since thalidomide has different toxicities in some animals.

The reasons why thalidomide is important in the study of autism include:

1. The impact on regulation and testing requirements for new medications brought to market regarding teratogenic potential
2. The association of autism with a known environmental toxin in a subgroup of affected individuals
3. The subsequent inquiry that led from early brain injury to investigation of early developmental genes as etiologic for autism

See Also

- ▶ [Rubella](#)
- ▶ [Toxicology](#)

References and Reading

- Kontogiannis, V., & Powell, R. J. (2000). Use of thalidomide in dermatologic indications. *BioDrugs*, 13(4), 255–265.
- Miller, M. T., & STromland, K. K. (2011). What can be learned from the thalidomide experience: An ophthalmologic perspective. *Current Opinion in Ophthalmology*, 22(5), 356–364.
- Rodier, P. M. (2004). Environmental causes of central nervous system maldevelopment. *Pediatrics*, 113, 1076–1083.
- Millrine, D., & Kishimoto, T. (2017). A brighter side to thalidomide: Its potential use in immunological disorders. *Trends in Molecular Medicine*, 23(4), 348–361. <https://doi.org/10.1016/j.molmed2017.02.006.Epub>. PMID 28285807.

Theoretical Models and Autism

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Definition

In this entry, the authors want to introduce you to some theoretical models of autism based on the

three main models of disability – the medical model, the social model, and the bio/psycho/social model – which we will review briefly first. But there are two terminology matters to consider before we begin. We explain why we use autism-first language in this entry. And we explain some terms introduced by Ian Hacking and Carol Thomas, an understanding of which is crucial to our discussion of the theoretical models.

We use autism-first language throughout this entry. That is because this form of language is generally preferred to person-first language by autistic adults. Although Dunn and Andrews argue that both forms should be used “to address the concerns of disability groups while promoting human dignity and maintaining scientific and professional rigor” (2015, p. 255), we are unaware of any evidence that respecting the views of the majority of autistic adults in this regard impacts adversely on scientific and professional rigor.

We also need to reflect on some terms introduced by Ian Hacking (1999) and Carol Thomas (1999, 2004) which form part of the framework of models. The terms we refer to are “indifferent kind” and “interactive kind” (Hacking) and “impairment effects” (Thomas). We also need to consider the essential differences between the main models of disability, i.e., the medical, social, and bio/psycho/social models. Those who regard autism as cognitive, perceptual, and sensory difference object to the term “impairment.” They would presumably also object to the term “impairment effects.” We use this term in our framework of autism models for the same reason Thomas introduced it, to distinguish between the inherent effects of an impairment and the additional, avoidable, societal effects. Those who agree with us that autism involves a set of *differences* (we do not subscribe to the view that all autistic individuals are inherently *impaired*) could substitute “difference effects” or “inherent disadvantages” for “impairment effects.”

In *The Social Construction of What?* Hacking (1999) discusses the increasingly widespread use of the concept of social construction and of things being socially constructed using examples such as authorship, the child viewer of television, danger, emotions, gender, and illness. He argues that in many cases it is only superficially correct to

refer to something as being socially constructed since every concept people invent is socially constructed in the trivial sense that all human concepts are the result of social interaction. The two terms he has introduced are designed to highlight the distinction between the kind of things that can interact with the people who are of that kind and those things that do not interact in that way; the former falling into the category of “interactive kind,” and the latter in the category of “indifferent kind.” Examples should make the difference clear. Hacking uses the concept of the “woman refugee” to explicate the idea of an interactive kind; the important point here being that when a woman knows that she is classified as a “woman refugee” she may, unknowingly or not, begin to act *as* a woman refugee, that is, behaving in ways she would not if unaware of her classification. In the same way, an autistic person may start analyzing their behavior after receiving a diagnosis of autism, explaining past behavior from the perspective of autism, and perhaps being less likely to mimic nonautistic behavior (especially in a setting where autism is accepted as natural human difference). Thus, a diagnosis of autism may interact with the individual diagnosed to change behavior and is, therefore, an interactive kind. The opposite of an interactive kind is an indifferent kind where no interaction effect between the concept and the thing itself is possible. For instance, to use a couple of Hacking’s examples, neither quarks nor microbes, even though the latter interacts with human beings, are aware of what they do and so are said to be indifferent to their classification as quarks and microbes (we think that “unaware” would have been a better choice of term as “indifferent” implies the holding of a view by a thinking being). Likewise, a cognitive difference in autism is indifferent to, or unaware of, its status as a cognitive difference prior to the autism being identified and understanding of the difference having developed.

Much has been written about disability models. We only have space to draw attention to the fundamental difference between the medical model of disability and the social model of disability and position the bio/psycho/social model. While the medical model places the “fault” for being

disabled fairly and squarely with the individual, under the social model disability is seen as the result of attitudes and barriers imposed by society over and above an individual’s impairments. Unlike the other two models, the bio/psycho/social model acknowledges biological, psychological, and social disabling/disadvantaging effects.

Realizing that not all disabling effects are the fault of society, Thomas introduced the term “impairment effects” to enable a distinction to be drawn between the inherently disabling effects of a perceived impairment and the effects of the attitudinal and physical barriers placed in the way of individuals with impairments by society. In the context of autism one might want to say, for example, that the difficulties arising from sensory sensitivities may not always be something that society can do anything to ameliorate. But failure to appreciate the strengths in autism, and enable an individual to make the most of those strengths, is a barrier that society should work to remove.

Historical Background

We now introduce the medical, social, and bio/psycho/social models of disability before considering autism from the perspectives of these three models, comparing the perspectives, and drawing some conclusions.

Medical Model of Disability

The historical background here is that autism, inevitably because it relates to a series of medical diagnoses beginning with “infantile autism” in the ninth revision of the World Health Organization’s International Classification of Diseases (ICD-9) published in 1979, and a year later in the third edition of the American Psychiatric Association’s Diagnostic and Statistical Manual (DSM-III), has been seen through the lens of the medical model of disability (also known as the individual model). The medical model of disability has been described by a Cerebral Palsy group as:

a model by which disability is the result of a physical condition, is intrinsic to the individual (it is part of that individuals own body), may reduce the individuals quality of life, and causes clear

disadvantages to the individual. By this model, a compassionate or just society should invest resources to attempt to cure disabilities medically or to improve functioning

(<http://www.livingwithcerebralspalsy.com/social-disability.php>)

The main criticism of the medical model of disability is that it equates disability solely with impairment, ignoring the involvement of social, cultural, and environmental factors in the construction of disability. The medical model has been said to attach blame to the individual for their disability and in that respect it is, at least, consistent with what Reindal refers to as the “many historical examples of individualising social problems: explaining poverty and unemployment as a result of idleness and character weakness; crime as a result of ‘moral insanity’” (Reindal 2008, p. 141). In this context, with disability being seen as the fault of the disabled person, society has little or no responsibility for looking after them, let alone for implementing change in order to reduce the impacts of societal barriers (because there is no recognition of the barrier concept). Over time, societal attitudes began to change and disability was increasingly seen as a personal tragedy for the “unfortunate individual with impairments.” As Reindal writes, “This approach . . . promoted attitudes of paternalism and mechanisms of dependency . . . within society” (Reindal 2008, p. 141) which led to a more caring or charitable attitude towards disabled people, but still with no acknowledgment that society was itself the primary cause of disability.

Social Model of Disability

A great deal remains to be done to ensure that people with disabilities are enabled to take their rightful places as full-fledged citizens within society, there is still much lip service paid to antidiscrimination, and the practice of “exclusion through nominal inclusion” (Thomas 2006, p. 177) is, perhaps, more prevalent than substantive inclusion. However, the advent of the social model of disability in the mid-1970s has had a profound impact on disability advocacy and

outcomes since its development through the work of the Union of the Physically Impaired Against Segregation (UPIAS), including the disabled scholar and activist Michael Oliver. The Cerebral Palsy group referred to in relation to the medical model of disability describe the social model of disability as follows:

the ‘social model’ of disability, makes a clear distinction between the impairment itself (such as a medical condition that makes a person unable to walk) and the disabling effects of society in relation to that impairment. In simple terms, it is not the inability to walk that prevents a person entering a building unaided but the existence of stairs that are inaccessible to a wheelchair-user. In other words, ‘disability’ is socially constructed.

(<http://www.livingwithcerebralspalsy.com/social-disability.php>)

While it is probably true to state that there is more rhetoric from organizations claiming to apply the principles of the social model of disability than actual application of those principles, even critics of the social model must accept that its development and increasing level of adoption has led to major benefits for disabled people. With eminent justification, this model is often referred to as the “big idea” of the disability movement.

Bio/Psycho/Social Model of Disability

The International Classification of Functioning Disability and Health (ICF) is the framework developed by the World Health Organization (WHO) for measuring health and disability at both individual and population levels. It was officially endorsed by all 191 WHO Member States at the 54th World Health Assembly held on 22 May 2001. The ICF is a classification of health and health-related domains based on bodily, individual, societal, and environmental perspectives through placing individuals on lists of bodily functions, activity limitations, and environmental factors. The ICF is said to complement the ICD diagnostic manual, referred to earlier, which comprises a classification of diagnoses based on etiology. The WHO has written that:

The ICF puts the notions of ‘health’ and ‘disability’ in a new light. It acknowledges that every human being can experience a decrement in health and

thereby experience some degree of disability. Disability is not something that only happens to a minority of humanity. The ICF thus ‘mainstreams’ the experience of disability and recognises it as a universal human experience. By shifting the focus from cause to impact it places all health conditions on an equal footing allowing them to be compared using a common metric – the ruler of health and disability. Furthermore ICF takes into account the social aspects of disability and does not see disability only as a ‘medical’ or ‘biological’ dysfunction.

https://ec.europa.eu/eip/ageing/standards/healthcare/e-health/icf-browser_en

The WHO regard the ICF as following what they call a biopsychosocial model of disability that “synthesizes what is true in the medical and social models, without making the mistake each makes in reducing the whole, complex notion of disability to one of its aspects” (WHO, p. 9). Reindal writes that the understanding of disability within the ICF is about restricted activities and an inability to do things that others can do, that it “is a weaker framework for theorising about empowerment and autonomy for disabled people, as goals for participation rest on norms related to normality” (Reindal 2008, p. 138) and, perhaps most important of all given the importance of oppression to the founders of the social model, the issue of oppression is lost in Reindal’s view.

Current Knowledge: Theoretical Models of Autism and Autism Theory

We aim to explain the five elements of a proposed framework of models of autism (see Table 1), which could also be described as a framework of perspectives on autism from which autism theory – which we discuss in a separate entry – can be viewed. These perspectives include one in which autism is regarded as nothing more than a label applied by classificatory systems to something that is a reified category with no existence other than as a conglomeration of unconnected behaviors (i.e., it is entirely socially constructed). The other four perspectives regard autism as something “real” in that it has a biological/neurological cause, at least in part, as well as socially constructed aspects. The opposite of the “denial”

Theoretical Models and Autism, Table 1 A framework of perspectives on autism (autism models)

Category	Kind	Cause
1. Autism as nothing more than a label – social model		
Diagnosis (label)	Interactive kind	Socially constructed
2. Autism as cognitive/perceptual/sensory difference – social model		
Differences	Indifferent kind	Bio/psycho causation
Societal effects pre-diagnosis	Indifferent kind	Socially constructed
Societal effects post-diagnosis	Interactive kind	Socially constructed
Diagnosis ^a (label)	Interactive kind	Socially constructed
3. Autism as disability in a lay sense – social model		
Impairments	Indifferent kind	Bio/psycho causation
Impairment effects	Indifferent kind	Bio/psycho causation
Disability (societal effects) prediagnosis	Indifferent kind	Socially constructed
Disability (societal effects) postdiagnosis	Interactive kind	Socially constructed
Diagnosis (label)	Interactive kind	Socially constructed
4. Autism as disability in a lay sense – bio/psycho/social model		
Impairments	Indifferent kind	Bio/psycho causation
Impairment effects	Indifferent kind	Bio/psycho causation
Societal effects pre-diagnosis	Indifferent kind	Socially constructed
Societal effects post-diagnosis	Interactive kind	Socially constructed
Diagnosis (label)	Interactive kind	Socially constructed
5. Autism as disability in a lay sense – medical model		
Impairments	Indifferent kind	Bio/psycho causation
Disability	Indifferent kind	Bio/psycho causation
Societal effects pre-diagnosis ^b	Indifferent kind	Socially constructed
Societal effects post-diagnosis ^b	Interactive kind	Socially constructed
Diagnosis (label)	Interactive kind	Socially constructed

^aThose who see autism as a set of differences do not consider that it should be included in diagnostic manuals
^bSeemingly unacknowledged societal effects

of those who consider autism to be no more than a label is the medical model perspective whereby autism is generally considered to be entirely biological/neurological in nature. However, in our view, even a medical model perspective on autism should incorporate socially constructed elements as we hope to make clear. We now consider each of the five sections of our framework of perspectives on autism.

We understand why Runswick-Cole (2016, p. 27, our italics) writes that it would be far better to have a support service for people currently labeled with autism “that labels people, *but only with their names*.” She stresses the importance of practitioners asking questions about a person rather than a disorder and offering to help them achieve their aspirations “rather than intervening to ‘correct’ perceived symptoms and deficits.” We agree with Runswick-Cole that it is essential to consider an individual’s unique needs, especially as the effects of autism vary so greatly. It is also unarguable that the label of autism is generally needed to enable access to support where it is available. However, although some scholars may consider dispensing with this label to be a valid longer-term objective, we disagree, as the label has helped many autistic individuals to achieve a better understanding of themselves. There are even, as Goodley (2016, p. 147) points out, those who “have suggested that autism is nothing more than a myth perpetuated by the biopolitics of psychiatry and psychology.” This is not the place to mount a challenge to those who believe autism is *only* a label. However, holding this view is to be utterly dismissive of leading clinical expertise beginning with Asperger and Kanner, the experiences of those for whom becoming aware that they are autistic has been transformational, and the experiences of parents of autistic children.

We now move on to consider the other elements of the framework: autism as cognitive/perceptual/sensory difference, and as disability, from the perspectives of the three main disability models: the medical model, social model, and bio/psycho/social model. Members of the general public would usually regard autism as a disability in the sense of being something inherent in the individual that has an adverse effect on their

ability to function in society to the fullest extent (though it should be noted that such judgements are made from a predominant neurotype (nonautistic) perspective and, therefore, may not be valid for all autistic individuals). This lay understanding of disability contrasts with the social model perspective on disability as attitudinal and physical barriers socially constructed by society in addition to impairments/differences. Whether understood as impairments or differences, matters caused biologically/neurologically are indifferent kinds according to Hacking’s definition because there is no interaction effect between them and the individual autistic person; the impairments/differences are simply what they are. We think that the societal effects are also indifferent kinds prior to receipt of a diagnosis – because at this stage they cannot interact with the individual and change behavior – but may transform into interactive kinds after diagnosis when the diagnosed individual becomes aware of them and reflects on their effects on him or her. Even in the case of the model regarding autism as nothing more than a label, we are in no doubt that a diagnosis is an interactive kind because it can interact with the individual and thus carries the potential to change behavior.

We argue that autism involves impairments/differences, impairment effects as understood by Thomas, and societal effects. The social difficulties and sensory sensitivities in autism may have disabling effects to which society adds a layer of attitudinal and physical barriers that further disable. Although the medical model regards autism as a set of impairments that themselves disable an individual, it seems to us that those who adopt a medical model perspective should acknowledge that societal effects do exist; those who consider that impairments result in disability should accept that impairments do not cause disabling attitudinal and physical barriers. Ignoring the label-only model, there is little difference between the models. In each case, there are impairments/differences (which may disadvantage), impairment effects, an overlay of disabling societal effects, and the potential for behavior change following receipt of a diagnosis.

When seemingly unacknowledged societal effects are included in a medical model

perspective, the result is not unlike a bio/psycho/social model. If a social model perspective does not ignore impairments/differences, and the impairment effects Thomas wrote of, it is also virtually indistinguishable from a bio/psycho/social model. We do not consider that rejection of a medical model perspective on autism of necessity requires autism to be viewed as a social construct in a nontrivial sense as Shyman (2015, p. 73) appears to believe having written that “the medical model of viewing autism is overtly rejected in favor of viewing it as a social construct.” [In a trivial sense, autism and every other diagnosis, together with the diagnostic manuals themselves, and the psychiatry and psychology that led to the development of these manuals, are socially constructed.] We argue that it is the idea of autism *as a disorder* that is socially constructed in a nontrivial sense and that medical diagnosis would not be as necessary as it is currently if society accommodated the differences in autism, while stressing the importance of identifying individuals who are autistic. Formal *identification* (diagnosis) of autism is essential to: (1) help autistic individuals to better understand themselves; (2) assist those who live and work with autistic people to support them; and (3) enable the authorities to implement ad hoc environmental adaptations for those who need them. To be clear, we are saying no more than that it is wrong to medicalize a set of cognitive, perceptual, and sensory differences. We do not deny the effects autism can have on the lives of individuals and their families/carers. Neither do we challenge the role of clinicians in relation to autism while it remains necessary for individuals to be diagnosed as autistic to secure protection under disability discrimination legislation (such as the UK’s Equality Act, 2010) and access to whatever local authority support is available.

Future Directions: For Autism Research

Irrespective of an individual’s perspective on autism, especially given our view that there is less difference between the three main models of disability than there may appear to be, many

members of a sizeable community of autistic people need support. This section is a combination of a reflection on the Pellicano et al. paper (2014) alongside a summary of our rationale for certain types of research being more advantageous than others because of our belief that research in autism should be focused primarily on supporting individuals with cognitive, perceptual, and sensory differences to achieve the fullest life possible in every respect (education, relationships, work, etc.). Pellicano and colleagues identified the following six types of funded autism research over a 3-year period specific to the UK (along with the percentage of funding allocated to each type of research over those 3 years):

1. Biology, brain, and cognition – 56%
2. Treatment and interventions – 18%
3. Causes – 15%
4. Diagnosis, symptoms, and behaviors – 5%
5. Services – 5%
6. Societal issues – 1%

The reaction from the autism community was almost entirely negative, with autistic adults suggesting that research follows a nonautistic agenda, and parents reflecting that research fails to accurately reflect the reality of the lived experience of autistic individuals. It is not possible to simply identify what is “good” research and what is “bad” research in terms of type, but it is worth identifying questions that might enable researchers to ascertain the purpose of their research and what potential impact it might have. This, in turn, might enable funders to understand the efficacy and purpose of research aims. We suggest the following questions provide a structure within which research can be framed:

1. Does the research engage directly with the autism community?
2. Does the research engage with autistic individuals as “subjects” or as co-researchers?
3. What potential impact might the research have on autistic individuals?
4. What impact might the research have on those associated with autism (e.g., parents, carers, professionals)?

5. Is the main purpose of the research to directly or indirectly influence quality of life for the autistic population?
6. Does the research intend to establish new knowledge that can influence practice that will have a positive influence within the autism community?
7. How might the research enable practitioners to develop better practice?
8. How involved are autistic people in the aims of the research and the project design?
9. Does the research fulfil or acknowledge any criteria identified by the autism community as needing investigation?

Clearly this list of questions is not exhaustive, but it does provide a structure within which the purpose of research and its potential links to practice can be explored. Chown et al. (2017) have developed a more detailed framework of criteria for what they refer to as “inclusive” autism research, i.e., research which is both emancipatory and participatory. Neither we nor they dismiss research that may not influence the day-to-day lived experience of autistic individuals entirely; however, it is acknowledged that the current funding of autism research does not reflect the views of the autism community and we suggest that this needs addressing as a matter of urgency.

See Also

► [Autism Theory](#)

References and Reading

- Chown, N., Robinson, J., Beardon, L., Downing, J., Hughes, L., Leatherland, J., Fox, K., Hickman, L., & MacGregor, D. (2017). Improving research about us, with us: A draft framework for inclusive autism research. *Disability and Society*, 32(5), 1–15.
- Dunn, D. S., & Andrews, E. E. (2015). Person-first and identity-first language: Developing psychologists’ cultural competence using disability language. *American Psychologist*, 70(3), 255–264.
- Goodley, D. (2016). Autism and the human. In K. Runswick-Cole, R. Mallett, & S. Timimi (Eds.), *Re-thinking autism: Diagnosis, identity and equality*. London: Jessica Kingsley Publishers.
- Hacking, I. (1999). *The social construction of what?* Harvard: Harvard University Press.
- Pellicano, E., Dinsmore, A., & Charman, T. (2014). What should autism research focus upon? Community views and priorities from the United Kingdom. *Autism*, 18(7), 756–770.
- Reindal, S. M. (2008). A social relational model of disability: A theoretical framework for special needs education? *European Journal of Special Needs Education*, 23(2), 135–146.
- Runswick-Cole, K. (2016). Understanding this thing called autism. In K. Runswick-Cole, R. Mallett, & S. Timimi (Eds.), *Re-thinking autism: Diagnosis, identity and equality*. London: Jessica Kingsley Publishers.
- Shyman, E. (2015). *Besieged by behavior analysis for autism spectrum disorder: A treatise for comprehensive educational approaches*. Lanham: Lexington Books.
- Thomas, C. (1999). *Female forms: Experiencing and understanding disability*. New York: McGraw-Hill Education.
- Thomas, C. (2004). How is disability understood? An examination of sociological approaches. *Disability & Society*, 19(6), 569–583.
- Thomas, C. (2006). Disability and gender: Reflections on theory and research. *Scandinavian Journal of Disability Research*, 8(2–3), 177–185.

Theories of Language Development

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Definition

Language acquisition presents children with a tremendous challenge: how to determine the mappings between meanings in their world, and the complex acoustic signal of speech. Children must segment the running stream of sounds that they hear into words and phrases, and determine the regularities, or systematicities, of the language. Furthermore, they accomplish this task in the absence of direct instruction and even (for example, in the case of Quiche Mayan) while hearing little language directed at themselves (Pye 1986). As such, the acquisition of human language has been described as the single most impressive

intellectual accomplishment of individual humans (Pinker 1984).

There are five systematic features of language that a learner must master in order to understand and produce a spoken or signed language. Note that while this review focuses on spoken languages, there is such significant overlap with signed languages; see the ► [“American Sign Language \(ASL\)”](#) entry for more information. *First*, they must learn the phonology – that is, the inventory of sounds that make up the language. Sounds are considered to be distinct phonemes when they are “minimally contrastive,” such that two words which differ only by that sound, are recognized as distinct words (“*pin*” vs. “*pen*” is a minimal pair in English, whereas “*pint*” vs. “*pint^h*” with a final “*t*” accompanied by a puff of air is noncontrastive). Children learning English must master an inventory of 12 distinct vowels and 24 consonants (depending on their dialect); classical Arabic, 6 vowels and 30 consonants; and Swedish, 17 vowels and 18 consonants. The phonology of a language corresponds to a degree to its written alphabet, but is not identical (and indeed, in English, the alphabet/phonology correspondence is particularly weak, making the acquisition of reading in English a particularly lengthy and error-prone process).

Second, the learner must learn an inventory of words; that is, she must determine what concepts are encoded as words, and map those concepts onto specific sequences of sounds. The acquisition of a language’s vocabulary, or *lexicon*, is a challenging proposition; some languages convey concepts that, in other languages, are unspecified (for example, the concepts *melancholy* and *gloomy* are conveyed by a single term in many other languages). Typically developing children produce first words by around 10–11 months. These early words generally consist of names for specific individuals (*mama*, *Sarah*), objects (*dog*, *banana*), or substances (*juice*); other early emerging word types include action verbs (*give*), adjectives (*pretty*), and some social expressions (*hi*, *no*). Abstract terms, such as mental state verbs (*think*, *remember*), and those expressing nonpresent objects or events, are produced later. Children bring a set of useful constraints or biases to this learning problem;

for example, they are guided by an assumption of “mutual exclusivity,” which suggests that novel concepts should be expressed using novel words (Markman 1990). Research indicates that children with autism spectrum disorder share at least some of these important biases (e.g., mutual exclusivity; de Marchena et al. 2011), but not all (e.g., the shape bias; Tek et al. 2008).

Third, the learner must determine how words are meaningfully combined in the language, a system of combinatorial rules known as syntax (also called grammar). In English, a descriptive noun phrase might consist of a determiner (e.g., *the*), an adjective (*fuzzy*), and a noun (*rabbit*); sentences in English are structured as subject-verb-object. During the second and third years of life, children’s utterances increase in length and complexity, and increasingly incorporate function words (*if*, *the*, *over*); they also incorporate advanced syntactic devices such as relative clauses (*the cookie that I ate yesterday*), yes/no questions (*can I have that cookie?*), and if/then statements (*if I eat my dinner, can I have a cookie?*).

Fourth, children must learn how to mark tense, gender, number, aspect, register (formality or politeness), and so on, via either phrasal combinations or via morphological markers. “Morphemes” are the smallest meaningful units of language; morphological development refers to the development and understanding of these units and how such units are combined into words. For example, *junper*, *jumped*, *jumps*, and *jumpy*, all use the morpheme “*jump*” in combination with morphemes *-er*, *-ed*, *-s*, and *-y*. In many languages (typically those where word order is free to vary), syntactic information is conveyed primarily through morphological markers; thus, many theories of language structure collapse morphology and syntax into a single process (morphosyntax).

Finally, in addition to phonological, lexical, morphological, and syntactic development, children must learn to use language as a social tool. Pragmatic language refers to a speaker’s ability to select of words that are appropriate to the social context (“register,” i.e., speaking more formally to a professor than to a peer, avoiding taboo words in many situations); negotiating when it is one’s turn in the conversation; the choice of referential expressions

(*a* vs. *the*); use of indirect speech (*it's very cold in here*), sarcasm (*nice shirt, Mom*), or metaphor (*you had that one hidden up your sleeve*); repair of miscommunications; the use of prosody (the melody or rhythm of the voice); the use of perspective in language (*he thought that she was going home*); and nonverbal linguistic functions (e.g., eye contact, body language, and gestures). Discourse is a closely related concept, which refers to the production of longer connected streams of speech, as in the telling of a story or joke. Narrative ability is important for communication as well as for the structuring of one's own thoughts.

Historical Background

According to Herodotus (Fromkin et al. 1974), an Egyptian pharaoh, Psammetichus the First, conducted one of the first known experiments on language acquisition. Hoping to uncover the “original” language, he caused two newborn babies to be left with an isolated shepherd, who was ordered not to speak with them. The shepherd monitored their speech sounds; as the first word uttered sounded like “bread” spoken in Phrygian, Psammetichus concluded that Phrygian was the original or primary language of the world. Later work has also attempted to build on the experiences of feral or wild-reared children in order to explore the process of language acquisition in the absence of or given inadequate language input (Candland 1993); one challenge is to disentangle language input from atypical social experiences.

Another classic formulation described in theories of language acquisition is a problem posed by the philosopher Quine, commonly known as the “gavagai” thought experiment (Quine 1960). Quine asks us to imagine standing in an alien world, next to a native from that world; when a rabbit runs past, the native points and says, “gavagai!” While *gavagai* might refer to the concept *rabbit*, there is a nearly infinite number of alternative interpretations, such as *brown*, *furry*, *four-legged*, *speedy*, *hopping*, *delicious*, *watch out!* or, even, *an assembly of rabbit parts*. How can a learner determine what semantic concepts

are conveyed, when they happen to hear an isolated word spoken?

Faced with the challenges of learning what sounds should cohere to form words, the challenge of determining word meanings, and the challenge of learning how words should be further combined, one might assume that an important component of language acquisition is the instruction of adults. Indeed, a typical educated parent in the USA may act according to the assumption that she is teaching her infant to speak. Somewhat surprisingly, however, children receive very little explicit instruction in their language; what is more, they appear to receive little negative (error-correcting) feedback about their own speech. That is, when children produce a speech error, it is rarely explicitly corrected. Furthermore, children are famously resistant to such corrections. McNeill reports a now-famous anecdote about this resistance to error correction (McNeill 1970, p. 106): Child: *Nobody don't like me*. Mother: *No, say “nobody likes me.”* Child: *Nobody don't like me* (eight repetitions). Mother: *Listen carefully: say, “nobody likes me.”* Child: *Oh! Nobody don't likes me*.

Current Knowledge

As reviewed above, the challenge of learning a language is a formidable one. Given the difficulties, it is remarkable how uniform the process of acquisition is. It follows a similar developmental progression across children, despite quite striking differences in the structure of the language being learned, culture, individual differences in intelligence or sociability, parent factors, and so on. That said, however, not all children develop functional language skills; in addition to the clinical implications of this fact, the study of delays and deficits in acquisition can help to elucidate the nature of the language acquisition process, by throwing into sharper relief the developmental course of language acquisition (Cicchetti and Rogosch 1996; Curtiss et al. 1992).

One of the primary theories of language acquisition is associated with “nativist” approaches to human development. The enormous complexity

of language, and the relatively limited direct “teaching” of language (along with the uniformity of acquisition of language milestones), has led scholars in the nativist or innate school to propose that language development proceeds according to the growth or unfolding of an innate language faculty, just as physical development proceeds in the absence of specific instruction or special input (e.g., Chomsky 1986; Fodor 1983). Chomsky formulated one of the most influential theories of language that applies to the nativist approach, the principles and parameters framework (and its subsequent iterations and revisions, Government and Binding, and the Minimalist approach). In this model, humans have innately specified knowledge about language: a set of principles shared by all human languages (e.g., that all grammatical sentences must have a (possibly covert) subject) and a set of parameters that specify how a given language instantiates specific syntactic qualities (e.g., that the subject should be overtly expressed, or not); parameters are essentially binary decision processes. Under this account, children require little language input; they only require enough input to direct them in how to set those particular parameters.

The “constructionist” perspective provides an alternative perspective (see Elman et al. 1996); in this account, general and nonlinguistic learning capacities may account for much or all of language knowledge. Although support for related theoretical positions dwindled after Chomsky’s (1959) influential and overwhelming critique of behaviorism, more recent research suggests an important role for domain-general learning capacities such as tracking the statistics of words or sounds in language input (Saffran et al. 1996). The history of research in language development has been marked by disagreement between the nativist and constructionist positions; currently, many researchers in the field accept some middle ground between the strict nativist and the purely empiricist approaches, suggesting that children may draw on some innately specified biases as well as on domain-general learning processes.

There are several important models that fall in the middle ground of, or otherwise negotiate between, the nativist and empiricist perspectives.

One usage-based account, the “verb island” hypothesis (Tomasello 2000), for example, suggests that children acquire syntactic knowledge gradually, item by item, and that much of their early knowledge consists of memorized two- or three-word structures (with relatively little transfer of morphological structure across verbs). Experiments presenting children with novel (nonsense) verbs indicate a sharp change between ages 3 and 4. Specifically, children aged 3–4 are able to extend novel verbs to abstract syntactic categories; for example, they are able to use a verb heard only in a passive sentence frame and utilize it in an active sentence frame. However, children aged 2–3 years have difficulty extending verbs between transitive and intransitive frames.

Connectionist approaches to acquisition have also provided a set of tools for addressing the trade-off between the apparent poverty of syntactic input to the learner (i.e., the “learnability” problem) and the role of innate structures and processes in development; thus, while often described as simply a version of empiricism, connectionism and empiricism are not necessarily identical (Plunkett 1997). “Connectionism” refers to computational models (either theoretical or actually implemented using computer learning algorithms) that take input (typically, somewhat abstracted in nature from actual language input) and feeds that input forward (e.g., through a single layer of units or nodes) to a set of output units. For example, a network model of past tense formation in English might receive as input an abstracted phonological representation of the verb stem (*jump*, *swim*); activation (i.e., which units are ready to “fire” at a given time) passes from input units to a connected set of output units; those units receiving the strongest input activations are most likely to be active in the output (e.g., the corresponding past tense form: *jumped*, *swam*). The output activations are compared to the target activation pattern (e.g., the correct past tense form), and any discrepancies form an error signal which influences the network connections. Connectionist models have helped demonstrate “proof of principle” that a single learning system is capable of generating such apparently distinct outputs as the regular past tense form (*-ed*) along

with the irregular forms. Connectionist models have also helped to generate and test models of concept formation and vocabulary acquisition (see Plunkett 1997). Work of the past decade has examined children's ability to make use of distributional aspects of language input (e.g., Saffran et al. 2007) and demonstrated both the availability of potentially informative information in the statistics of language input, as well as learners' ability to access that information. Few computational models of ASD have been formulated (though see, e.g., McClelland 2000), though this approach may increasingly offer researchers ways to develop and test hypotheses about human acquisition.

Among the many methodological innovations in child language research, several bear particular mention: the language database approach. Research in this field has often made use of "diary studies" in which investigators track the spontaneous utterances produced by an individual child over time. This is a valuable resource for that individual, but it becomes a highly useful resource if it is available to others. The CHILDES website project has assembled written transcripts of children's speech from 34 languages, several clinical populations, and children in bilingual acquisition contexts. This publicly available, searchable, and downloadable database has been used as a resource in more than 3,000 publications from its inception through 2008. It can be accessed at <http://childes.psy.cmu.edu/>. MacWhinney, who is the principal investigator on the CHILDES database, spearheaded the development of a language transcription system (CLAN), also available on that website.

Another recent innovation in child language research is the availability of the LENA (Language ENvironment Analysis) system. A small recorder, generally worn as a backpack, can record a child's voice as well as other voices and sounds in the child's environment, across varied settings (home, playground, preschool, therapy sessions, etc.). After recording, software algorithms are able to automatically tag each speech sound according to its sound source (adult, child, male, female, etc.), and to label them as speech or nonspeech (Oller et al. 2010). While the LENA system does not lend itself to fine-grained analyses of specific narrow aspects of acquisition, it permits researchers

to collect large corpora from large groups of participants (e.g., Warlaumont et al. 2014).

Future Directions

Researchers in the field of language acquisition have often turned to ASD as a sort of "natural laboratory" in which to test theories of acquisition because studies of this disorder offer the possibility of examining meaningful differences in ability across a wide range of language, social, and cognitive domains. At the same time, research in this area must grapple with the subtleties in performance and ability that are part and parcel of a developmental disorder; development is rarely as neatly "packaged" as we might hope. For example, in typical development, comprehension seems to outpace production in early language acquisition; thus, children who do not produce function words (e.g., determiners) show effects in comprehension and memory when those elements are left out, suggesting some sensitivity and awareness of these elements. Children with ASD appear to provide a distinct picture, with some data suggesting that their production may outpace their comprehension. Research in the field must reconcile this somewhat bewildering inconsistency with a finding in typical development that is, as yet, unexplained. Open questions remain about acquisition in autism remain to be addressed, using cross-linguistic studies (e.g., Brynskov et al. 2017), eyetracking (e.g., Schuh et al. 2016), and psychophysiological tools such as EEG and MRI, among others.

See Also

- ▶ [American Sign Language \(ASL\)](#)
- ▶ [Grammar](#)
- ▶ [Language](#)
- ▶ [Language Acquisition](#)
- ▶ [Mean Length of Utterance \(MLU\)](#)
- ▶ [Phonology](#)
- ▶ [Pragmatics](#)
- ▶ [Prosody](#)
- ▶ [Syntax](#)

References and Reading

- Brynskov, C., Eigsti, I. M., Jørgensen, M., Lemcke, S., Bohn, O.-S., & Krøjgaard, P. (2017). Syntax and morphology in Danish-speaking children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(2), 373–383. <https://doi.org/10.1007/s10803-016-2962-7>.
- Candland, D. K. (1993). *Feral children and clever animals: Reflections on human nature*. New York: Oxford University Press.
- Chomsky, N. (1959). Review of skinner's 'verbal behavior'. *Language*, 35, 26–58.
- Chomsky, N. (1986). *Knowledge of language: Its nature, origin, and use*. New York: Praeger.
- Cicchetti, D., & Rogosch, F. (1996). Developmental pathways: Diversity in process and outcome. *Development and Psychopathology*, 8(4), 597–896.
- Curtiss, S., Katz, W., & Tallal, P. (1992). Delay versus deviance in the language acquisition of language-impaired children. *Journal of Speech and Hearing Research*, 35(2), 373–383.
- de Marchena, A., Eigsti, I. M., Worek, A., Ono, K. E., & Snedeker, J. (2011). Mutual exclusivity in autism spectrum disorders: Testing the pragmatic hypothesis. *Cognition*, 119, 96–113. <https://doi.org/10.1016/j.cognition.2010.12.011>.
- Elman, J. L., Bates, E. A., Johnson, M. H., Karmiloff-Smith, A., Parisi, D., & Plunkett, K. (1996). *Rethinking innateness: A connectionist perspective on development*. Cambridge, MA: MIT Press.
- Fodor, J. A. (1983). *The modularity of mind*. Cambridge, MA: MIT Press.
- Fromkin, V., Krashen, S., Curtiss, S., Rigler, D., & Rigler, M. (1974). The development of language in Genie: A case of language acquisition beyond the "critical period". *Brain and Language*, 1(1), 81–107.
- Markman, E. M. (1990). Constraints children place on word meanings. *Cognitive Science*, 14, 57–77.
- McClelland, J. L. (2000). The basis of hyperspecificity in autism: A preliminary suggestion based on properties of neural nets. *Journal of Autism and Developmental Disorders*, 30(5), 497–502.
- McNeill, D. (1970). *The acquisition of language: The study of developmental psycholinguistics*. New York: Harper & Row.
- Oller, D. K., Niyogi, P., Gray, S., Richards, J. A., Gilkerson, J., Xu, D., et al. (2010). Automated vocal analysis of naturalistic recordings from children with autism, language delay, and typical development. *Proceedings of the National Academy of Sciences*, 107(30), 13354–13359. <https://doi.org/10.1073/pnas.1003882107>.
- Pinker, S. (1984). *Language learnability and language development*. Cambridge: Harvard University Press.
- Plunkett, K. (1997). Theories of early language acquisition. *Trends Cognition Sciences*, 1(4), 146–153. [https://doi.org/10.1016/S1364-6613\(1997\)01039-01035](https://doi.org/10.1016/S1364-6613(1997)01039-01035).
- Pye, C. (1986). Quiche Mayan speech to children. *Journal of Child Language*, 13, 85–100.
- Quine, W. V. (1960). *Word and object*. Cambridge, MA: MIT Press.
- Saffran, J. R., Aslin, R. N., & Newport, E. L. (1996). Statistical learning by 8-month-old infants. *Science*, 274, 1926–1928.
- Saffran, J. R., Pollak, S. D., Seibel, R. L., & Shkolnik, A. (2007). Dog is a dog is a dog: Infant rule learning is not specific to language. *Cognition*, 105(3), 669–680. Epub 2006 Dec 2026.
- Schuh, J. M., Eigsti, I. M., & Mirman, D. (2016). Referential communication in autism spectrum disorder: The roles of working memory and theory of mind. *Autism Research*. <https://doi.org/10.1002/aur.1632>.
- Tek, S., Jaffery, G., Fein, D., & Naigles, L. R. (2008). Do children with autism spectrum disorders show a shape bias in word learning? *Autism Research*, 1(4), 208–222. <https://doi.org/10.1002/aur.38>.
- Tomasello, M. (2000). The item-based nature of children's early syntactic development. *Trends Cognition Sciences*, 4(4), 156–163.
- Warlaumont, A. S., Richards, J. A., Gilkerson, J., & Oller, D. K. (2014). A social feedback loop for speech development and its reduction in autism. *Psychological Science*, 25(7), 1314–1324. <https://doi.org/10.1177/0956797614531023>.

Theory of Mind

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Definition

Theory of mind (ToM) refers to the capacity to attribute mental states to others and oneself in order to explain and predict behavior. ToM is an evolved psychological ability, most highly developed in humans, specialized in the rapid attribution of beliefs, intentions, desires, or knowledge to others and oneself and in the spontaneous understanding that others have mental states that may differ from one's own.

The attribution of mental states to others pervades human cognition. When watching others, we automatically interpret their current behavior in the light of the intentions they might have. If we

see a man running as a bus passes by, for example, we will be inclined to explain his behavior by the fact that he *intends* to catch the bus, and this will lead us to predict that he will hop on the bus if he does manage to get to the bus stop in time. Attributing mental states and predicting behavior on that basis can thus be seen as our most natural way to grasp the social world.

Historical Background

Historically, the false belief task was considered to be the best test of ToM abilities: passing the test meant that one was able to represent others' mental states and failing the test was taken as evidence that one was impaired representing others' mental states. In false belief tasks, the participant is typically put in a position where she needs to take someone's belief into account so as to provide the appropriate answer to a question. Crucially, the belief she needs to take into account differs from her own (hence the term "false" belief). Standard false belief tasks have led to an industry of studies among which the unexpected location task is most common. In the "Sally-Anne" paradigm (the initial version of the unexpected location task), the location of an object is changed in the absence of a character who then has to look for the object. Children are told a story involving two dolls, Sally and Anne, playing with a marble. Sally puts the marble away in a basket and leaves the room. In Sally's absence, Anne takes the marble out and plays with it. Once she has finished playing, she puts the marble away in a box. Sally returns and the child is asked where Sally will look for the marble. The child passes the task if she answers that Sally will look where she first put the marble; the child fails the task if she answers that Sally will look in the box (where the marble really is) (for descriptions of other similar tasks, see Baron-Cohen 1995).

Using these types of tasks, a substantive body of research led to the following threefold conclusion: (1) ToM is specifically human and does not appear to be part of other animals' psychological makeup (including nonhuman primates),

(2) typically developing children master ToM types of reasoning from around the age of four and rarely succeed in false belief tasks before that age (for a recent review see, Wellman et al. 2001), and (3) individuals on the autism spectrum face important difficulties with false belief tasks, which can be taken as evidence of a specific ToM impairment in the condition (Baron-Cohen 1995). Over the past decade, however, each of these conclusions has been challenged and current knowledge about ToM in typical and atypical development has been considerably reviewed as a result.

Current Knowledge

A considerable amount of recent research has aimed to elucidate the phylogenetic and ontogenetic roots of ToM and findings emanating from these fields of research are especially relevant to better understand the consequences of ToM dysfunctions in autism spectrum disorders (ASDs).

Philogeny. For a long time, ToM was construed as a uniquely human feature, and even our nearest relatives were thought to be unable to understand the psychological states of others. It was agreed that they could understand others' behaviors, but they were thought to do so using a behaviorist rather than a mentalistic stance. This view, however, was recently challenged by a number of findings suggesting that apes are not fully blind to others' mental states. In experimental settings, there is now robust evidence that chimpanzees are able to grasp actions underlying goals and intentions and that they understand that others perceive the world in a way that may differ from their own.

Evidence that chimpanzees understand the actions of others not just in terms of behaviors but also in terms of goals and intentions include the fact that they react differently when facing an actor who is unwilling versus unable to give them food, that they distinguish accidental and intentional behaviors, and that they are able to imitate the intended goal of an action even when the demonstrator failed to achieve that goal (for a review of 10 relevant studies, see Call and

Tomasello 2008). Evidence that chimpanzees are sensitive to others' perceptions include chimps' ability to follow the gaze of others (and the associated expectation that something relevant should be found at the target location), their understanding that gaze is stopped by opaque occluders, their use of gestures restricted to contexts when they know that others see and attend to what they are doing, and their use of all that knowledge for food competition purposes (i.e., pick the food that others are not looking at or cannot see because of an occluder) (for a review of 16 relevant studies, see Call & Tomasello).

Whether these skills are indicative of true ToM is still hotly disputed. In particular, evidence that chimpanzees are able to understand *beliefs* is still lacking. To date, every attempt at demonstrating that chimpanzees can succeed in understanding false beliefs, considered to be the only acid test of ToM, has failed.

To conclude, though evidence for fully human-like ToM is lacking, recent research now unambiguously indicates that chimpanzees do much more than simply reading overt behavior: they understand goals, intentions, perceptions, and knowledge of others and use these mentalistic mechanisms to make sense of and predict other's behaviors.

Ontogeny. Much research has investigated the early precursors to the development of a fully functional ToM, such as gaze following, perspective taking, or goal attribution, which all develop within the first 1 or 2 years of life. Infants start following the gaze of others as early as 3–6 months and use pointing gestures by around 1 year. Infants also demonstrate an early ability to take the perspective of others and make inferences regarding what others can see and cannot see. For instance, they know that one cannot see a target with closed eyes or under a blindfold. Infants as young as 14 months also take into account various kinds of obstacles in determining what others can see. In particular, they understand that an opaque screen blocks people's vision or that an adult looking behind a barrier can see things that they cannot see (Tomasello 2008).

Infants are also able to attribute goals to agents from about 7 to 12 months of age. For instance, in a well-known series of habituation studies, Csibra, Gergely, and colleagues demonstrated how infants interpret an agent's behavior in the light of the rational goal she might be pursuing (many relevant papers are available on Gergely Csibra's website). In the classic study, infants are habituated to a large dot jumping over an obstacle and approaching a small dot. When the obstacle is removed, infants are surprised if the large dot continues to jump while approaching the small dot (despite the fact that the path of movement is identical to the one they had been habituated to). However, if the large dot stops jumping and goes straight to the small dot, infants are not surprised (despite the fact that the path of movement differs from the one the infant has been habituated to). These results are evidence in favor of the idea that infants are able to attribute goals to moving agents. These behaviors (e.g., gaze and point following, perspective taking, goal attribution, etc.) all appear within the first 2 years of life and constitute early manifestations of our innate ability to understand mental states.

As far as the understanding of beliefs is concerned, the consensual view was that it emerged later on in development, at around the age of 4. Over the past few years, however, new paradigms have demonstrated that reasoning about beliefs can appear much earlier in development than previously taught. For instance, infants as young as 15 months old or even 13 months old are surprised when the actor's behavior does not match her true or false belief regarding the situation. In one experiment, for example, the infant sees an agent take a toy out of one of two boxes. The toy is then moved from one box to the other, either in the presence of the agent (true belief condition) or in her absence (false belief condition). The infant then sees the agent reach for the toy in one of the two boxes. Infants' looking times are used to measure their degree of surprise. Results indicate that infants look significantly longer when the agent's behavior is incongruent rather than congruent with her false belief (i.e., when she reaches for the box containing the toy) (Baillargeon et al. 2010).

Evidence in favor of early ToM skills is also compatible with a wealth of research in developmental pragmatics indicating that preverbal infants are able to engage in inferential communication that requires taking the audience's perspective. For instance, much research demonstrates that young children's pointing behaviors are best understood by positing that they are in some sense trying to influence the audience's mental states. Infants as young as 12 months spontaneously point to help an adult by providing her with needed or desirable information (Tomasello 2008). Conversely, infants are able to interpret adults' points and gaze direction as cues to their communicative intentions. In particular, infants use these behaviors in word-learning situations as crucial cues to the speaker's referential intent (Bloom 2000). Moreover, recent experiments demonstrate that 2–4-year-olds do not blindly follow gaze (or points), but consider the linguistic and pragmatic context when learning a new word. More strikingly still, recent research demonstrates that manipulating whether or not a communicator has a false belief leads toddlers to different interpretations of the same communicative act, thereby demonstrating early mental state attribution in pragmatic contexts (Baillargeon et al. 2010).

Taken together, these results indicate that infants with very limited verbal abilities understand goals, intentions, and even beliefs. This suggests that ToM is an essential part of our psychological makeup and confirms that there is a strong innate component to ToM.

ToM Dysfunction in Autism Spectrum Disorders. Among the various cognitive accounts of autism, the hypothesis of a primary deficit in ToM has had a considerable influence in both psychology research and intervention strategies. In this framework, an impaired ability to understand other people's minds is at the very heart of the autistic symptomatology: by preventing access to the full range of mental states and efficient mind reading, a deficit in ToM would lead to abnormalities in social development, in communication development, in empathy, and in imitation, all of which require taking other people's perspective.

The ToM deficit hypothesis of autism was put forward after Baron-Cohen, Leslie, and Frith demonstrated that children with autism had massive problems dealing with the Sally-Anne task (Baron-Cohen et al. 1985). In this seminal study, a large majority of typically developing children and of children with moderate learning difficulties passed the test while most children with autism did not. Numerous studies later replicated those findings, bringing further support to the idea that the social and communicative difficulties found in ASDs are rooted in a primary ToM deficit (Baron-Cohen 1995, 2000).

However, it must be noted that there is a subset of children with autism who do pass the Sally-Anne test, which could jeopardize the assumption that the ToM deficit is universal. This criticism led to more research using more sophisticated ToM paradigms, such as second-order false belief tasks. In these tasks, the participant has to represent what someone thinks of what another person thinks. For instance, in the ice cream van task, two characters (John and Mary) are independently informed about the unexpected change of location of an ice cream van. John thus knows that the van has moved, and so does Mary; however, both wrongly *believe* that the other doesn't *know* about this unexpected change in location. Children's understanding of this second-order belief structure is tested by asking them "Where does John think Mary will go for the ice cream?"

Using this paradigm with children with autism, Baron-Cohen (1989) found that 90% of TD children and 60% of children with Down syndrome passed the test. In sharp contrast, none of the children with autism succeeded. This led to the conclusion that although some individuals demonstrate that they can use first-order ToM, they remain unable to handle a two-order false belief task, thereby indicating that they do not have a fully representational ToM. But these results were later criticized because a significant proportion of young adults with a high-functioning ASD appear to succeed even in second-order ToM tests.

Criticisms to these original findings led to the development of two – mutually compatible – hypotheses. The first one is that individuals with

autism who do pass first- and second-order tests come to do so with a significant delay. This fits well with results found in Happé's (1995) meta-analysis of 13 false belief studies showing that the minimum verbal mental age (VMA) at which participants pass false belief tasks is 3.62 years in TD children and 5.5 years in children with ASD. Under the assumption that there is a critical period for the development of numerous cognitive skills, this delay could account for persisting deficits in the communicative and social realms.

The second hypothesis is that surface-level performance is to be distinguished from actual competence. It is indeed possible that the individuals with an ASD who pass ToM tests use strategies that greatly differ from ordinary ToM mechanisms. One should thus refrain from assuming that intact performance reflects intact competence. This is in line with the fact that more advanced tests of ToM reveal impaired performance even in individuals on the highest end of the spectrum. In the Strange Stories test (Happé 1994), for instance, participants are asked to justify why a character might have chosen to say what he says in a complex mentalistic situation. For example, a prisoner gets captured by enemy troops and upon being asked where the rest of his camp is hidden, the prisoner decides to reveal the exact location in the hope that the enemy will believe that he's lying and therefore send troops to the opposite location. Understanding this use of "double bluff" is a complex mind reading achievement that turns out to be especially challenging for individuals with autism, including for those individuals who do pass second-order theory of mind tests. Similarly, Baron-Cohen and colleagues found that adults with a high-functioning ASD who were able to pass first- and second-order standard ToM tests were nonetheless impaired in a subtle form of tests, assessing the ability to "read the mind in the eyes." In this test, participants look at pictures of the eye region displaying specific emotions and select the appropriate emotional adjective best describing the person's mental state (e.g., despondent, relieved, excited, shy). The fact that adults on the highest end of the autism spectrum are less proficient than control populations

in these kinds of tests suggests that difficulties dealing with psychological states are long lasting and can persist in even milder forms of the condition.

Future Directions

Standard false belief tasks and more subtle tests of mind reading have lent support to the idea that ToM deficits are widespread in ASDs. Whether this impairment is primary or simply consecutive to more basic deficits, however, is still debated. In particular, some researchers have argued that ToM deficits may be the consequence of a more primary deficit in social orienting or social attention. More specifically, this alternative hypothesis posits that autism is characterized by a primary disturbance in the motivational and executive processes that prioritize orienting to social stimuli. In this framework, decreased expertise in social cognition and ToM would be the result of reduced time spent attending to the social world (see e.g., Dawson et al. 2002; Schultz 2005). In one case then, impaired ToM is the result of impaired social attention. In the other, impaired ToM is the primary deficit behind decreased social attention. Telling these two hypotheses apart is the matter of further empirical investigation, perhaps through comparison of different clinical groups or longitudinal studies of individual differences within the autism spectrum.

See Also

- [Attributions \(First Order/Second Order\)](#)
- [Presupposition](#)
- [Social Cognitive Interventions](#)

References and Reading

- Baillargeon, R., Scott, R., & He, Z. (2010). False-belief understanding in infants. *Trends in Cognitive Sciences*, 14(3), 110–118.
- Baron-Cohen, S. (1989). The autistic child's theory of mind—a case of specific developmental delay. *Journal*

of Child Psychology and Psychiatry, and Allied Disciplines, 30(2), 285–297.

- Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory of mind*. Cambridge: MIT Press.
- Baron-Cohen, S. (2000). Theory of mind and autism: A 15-year review. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds.), *Understanding other minds: Perspectives from developmental cognitive neuroscience* (pp. 3–21). Oxford: Oxford University Press.
- Baron-Cohen, S., Leslie, A., & Frith, U. (1985). Does the autistic child have a “theory of mind”. *Cognition*, 21(1), 13–125.
- Bloom, P. (2000). *How children learn the meanings of words*. Cambridge: The MIT Press.
- Call, J., & Tomasello, M. (2008). Does the chimpanzee have a theory of mind? 30 years later. *Trends in Cognitive Sciences*, 12(5), 187–192.
- Dawson, G., Munson, J., Estes, A., Osterling, J., McPartland, J., Toth, K., et al. (2002). Neurocognitive function and joint attention ability in young children with autism spectrum disorder versus developmental delay. *Child Development*, 73(2), 345–358.
- Happé, F. (1994). An advanced test of theory of mind: Understanding of story characters’ thoughts and feelings by able autistic, mentally handicapped, and normal children and adults. *Journal of Autism and Developmental Disorders*, 24(2), 129–154.
- Happé, F. (1995). The role of age and verbal ability in the theory of mind task performance of subjects with autism. *Child Development*, 66(3), 843–855.
- Schultz, R. (2005). Developmental deficits in social perception in autism: The role of the amygdala and fusiform face area. *International Journal of Developmental Neuroscience*, 23(2–3), 125–141.
- Tomasello, M. (2008). *Origins of human communication*. Cambridge, MA: MIT Press.
- Wellman, H. M., Cross, D., & Watson, J. (2001). Meta-analysis of theory-of-mind development: The truth about false belief. *Child Development*, 72(3), 655–684.

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Thimerosal

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Definition

In 1999, the Public Health Service and the American Academy of Pediatrics issued a letter to pharmaceutical companies to remove thimerosal, an ethylmercury-containing preservative, from vaccines as quickly as possible. During the previous decade, as more vaccines were added to the infant vaccination schedule, some of which contained thimerosal, infants were exposed to greater quantities of ethylmercury. By 1999, it was possible for infants to receive as much as 187.5 ug of ethylmercury in the first 6 months of life. Regulators were worried that this quantity of mercury might cause subtle signs of mercury toxicity. Because no studies existed to refute this concern, public health officials cited the precautionary principle and urged removal.

The precipitous and frightening manner in which thimerosal was removed from vaccines in the late 1990s gave birth to several parent advocacy groups, specifically, *Generation Rescue*, *Moms Against Mercury*, and *Safe Minds*. All of these groups believed that mercury had been removed from vaccines because the public health community believed not that it *might* cause harm, but rather that it *had* caused harm. These groups were particularly concerned that thimerosal in vaccines had caused autism.

Although parents’ concern that mercury in vaccines could cause autism was understandable – especially given the manner in which it was removed – several toxicological and biological facts should have been reassuring. First, signs and symptoms of mercury poisoning are clearly distinct from those of autism. Second, ethylmercury is excreted from the body far more quickly than environmental mercury

(methylmercury). Third, the quantities of mercury contained in breast milk and infant formula likely to be ingested in the first 6 months of life were greater than the quantity of mercury contained in vaccines.

Epidemiological studies determining whether thimerosal in vaccines caused autism took advantage of several natural experiments. Western European nations had removed thimerosal from all vaccines by 1991. Also, some Canadian provinces used thimerosal-containing vaccines, whereas others used vaccines containing lesser quantities of thimerosal or no thimerosal. Investigators working in Europe, Canada, and the United States showed that children who received thimerosal-containing vaccines were not more likely to develop autism spectrum disorder or even subtle signs of mercury poisoning than children who had received thimerosal-free vaccines or vaccines containing lesser quantities of thimerosal. Scientists and public health officials are now comfortable that existing studies have refuted the concern that thimerosal in vaccines caused autism.

References and Reading

- Andrews, N., Miller, E., Grant, A., Stowe, J., Osborne, V., Taylor, B., Fombonne, E., Zakarian, R., Bennett, A., Meng, L., McLean-Heyward, D., Madsen, K., Lauritsen, M., Pedersen, C., Thorsen, P., Plesner, A. M., Andersen, P. H., et al. (2004). Thimerosal exposure in infants and developmental disorders: A retrospective cohort study in the United Kingdom does not support a causal association. *Pediatrics*, 114, 584–591.
- Fombonne, E., Zakarian, R., Bennett, A., et al. (2006). Pervasive developmental disorders in Montreal, Quebec, Canada: Prevalence and links with immunization. *Pediatrics*, 118, 139–150.
- Heron, J., & Golding, J. (2004). Thimerosal exposure in infants and developmental disorders: A prospective cohort study in the United Kingdom does not support a causal association. *Pediatrics*, 114, 577–583.
- Hviid, A., Stellfeld, M., Wohlfahrt, J., & Melbye, M. (2003). Association between thimerosal-containing vaccine and autism. *Journal of the American Medical Association*, 290, 1763–1766.
- Institute of Medicine. (2001). *Immunization safety review: Thimerosal-containing vaccines and neurodevelopmental disorders*. Washington, DC: National Academies Press.
- Institute of Medicine. (2004). *Immunization safety review: Vaccines and autism*. Washington, DC: National Academies Press.
- Madsen, K., Lauritsen, M., Pedersen, C., et al. (2003). Thimerosal and the occurrence of autism: Negative ecological evidence from Danish population-based data. *Pediatrics*, 112, 604–606.
- Nelson, K., & Bauman, M. (2003). Thimerosal and autism? *Pediatrics*, 111, 674–679.
- Offit, P. (2007). Thimerosal and vaccines—a cautionary tale. *The New England Journal of Medicine*, 357, 1278–1279.
- Pichichero, M., Gentile, A., Giglio, N., Umide, V., Clarkson, T., Cernichiar, E., et al. (2008). Mercury levels in newborns and infants after receipt of thimerosal-containing vaccines. *Pediatrics*, 121, e208–e214.
- Schechter, R., & Grether, J. (2008). Continuing increases in autism reported to California’s development services system. *Archives of General Psychiatry*, 65, 19–24.
- Stehr-Green, P., Tull, P., Stellfeld, M., Mortenson, P. B., Simpson, D., Thompson, W., Price, C., Goodson, B., Shay, D. K., Benson, P., Hinrichsen, V. L., et al. (2005). Autism and thimerosal-containing vaccines: Lack of consistent evidence for an association. *American Journal of Preventive Medicine*, 25, 101–106.
- Thompson, W., Price, C., Goodson, B., et al. (2007). Early thimerosal exposure and neuropsychological outcomes at 7 to 10 years. *The New England Journal of Medicine*, 357, 1281–1292.

Thioridazine

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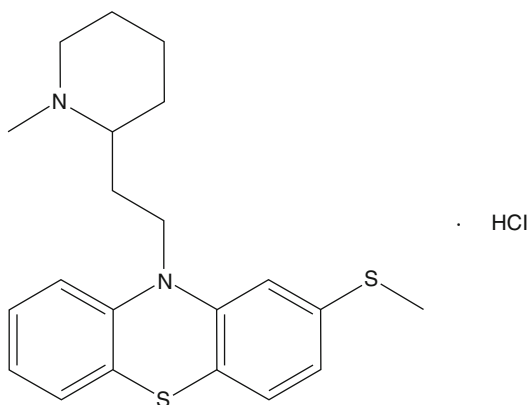
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Synonyms

Mellaril; Mellaril-S

Definition



Thioridazine is a prescription drug in the group of piperidine phenothiazines in the family of first-generation antipsychotics which has the chemical formula $C_{21}H_{26}N_2S_2 \cdot HCl$. This drug was initially FDA-approved for medical use in the year 1962. This compound has relatively low potency compared to the other first-generation antipsychotics, and its mechanism of action is thought to involve anticholinergic binding. This drug is FDA-approved for the treatment of schizophrenia and can be used to treat aggression and positive symptoms. Observed side effects include drowsiness/sedation, orthostatic hypotension, tachycardia, electrocardiogram (ECG) abnormalities, cardiac arrhythmias, anticholinergic effects, sexual dysfunction, galactorrhea, weight gain, photosensitivity, rashes, and pigmentary retinopathy. Thioridazine has significant potential adverse drug-drug interactions when coadministered with fluvoxamine, fluoxetine, paroxetine, sertraline, pindolol, propranolol, cisapride, fluoroquinolone antibiotics, many other antipsychotics, and tricyclic antidepressants among other drugs.

See Also

► [Antipsychotics: Drugs](#)

References and Reading

- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001). *Principles and practice of psychopharmacotherapy* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
- Stahl, S. M. (2000). Antipsychotic agents. In *Essential psychopharmacology: Neuroscientific basis and clinical applications* (pp. 401–458). Cambridge, MA: Cambridge University Press.
- Thioridazine. (n.d.). Retrieved from the ChemSpider Wiki: <http://www.chemspider.com/Chemical-Structure.5253.html>.
- U.S. Food and Drug Administration. (2011). *Drugs@FDA*. Retrieved from: <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm>.
- Wilkaitis, J., Mulvihill, T., & Nasrallah, H. A. (2006). Classic antipsychotic medications. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 211–228). Washington, DC: American Psychiatric Publishing.

Thiothixene

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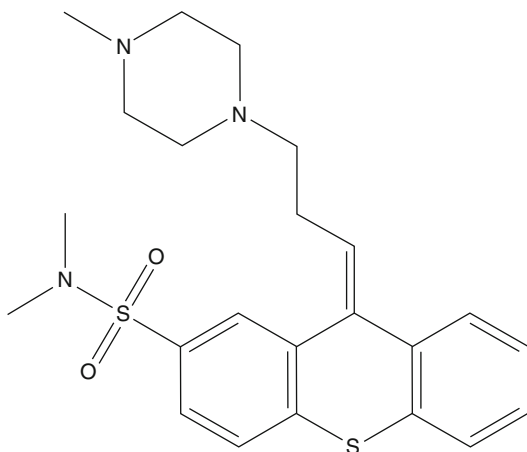
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Synonyms

cis-N,N-dimethyl-9-[3-(4-methyl-1-piperazinyl)-propylidene] thioxanthene-2-sulfonamide; Navane; Thiothixene hydrochloride; Thiothixene hydrochloride intensol

Definition



Thiothixene is a prescription drug in the group of thioxanthenes in the family of first-generation antipsychotics which has the chemical formula $C_{23}H_{29}N_3O_2S_2$. This drug was initially FDA-approved for medical use in the year 1967. This drug is FDA-approved for the treatment of schizophrenia and can be used to treat borderline personality disorder and schizotypal personality disorder. Observed side effects include drowsiness/sedation, insomnia/agitation, Parkinsonism, akathisia, and weight gain.

See Also

► [Antipsychotics: Drugs](#)

References and Reading

- Janicak, P. G., Davis, J. M., Preskorn, S. H., & Ayd, F. J. (2001). *Principles and practice of psychopharmacotherapy* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
- Stahl, S. M. (2000). Antipsychotic agents. In *Essential psychopharmacology: Neuroscientific basis and clinical applications* (pp. 401–458). Cambridge, MA: Cambridge University Press.
- U.S. Food and Drug Administration. (2011). *Drugs@FDA*. Retrieved from: <http://www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm>.

Wagner, K. D. (2006). Treatment of childhood and adolescent disorders. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 575–645). Washington, DC: American Psychiatric Publishing.

Wilkaitis, J., Mulvihill, T., & Nasrallah, H. A. (2006). Classic antipsychotic medications. In A. F. Schatzberg & C. B. Nemeroff (Eds.), *Essentials of clinical psychopharmacology* (2nd ed., pp. 211–228). Washington, DC: American Psychiatric Publishing.

Thiothixene

► [Navane](#)

Thiothixene Hydrochloride

► [Thiothixene](#)

Thiothixene Hydrochloride Intensol

► [Thiothixene](#)

Third Edition

► [Wechsler Preschool and Primary Scale of Intelligence](#)

Thorazine

► [Chlorpromazine](#)

Threat Detection

► [Reduced Prioritization of Facial Threat in ASD](#)

Threat Perception

- [Reduced Prioritization of Facial Threat in ASD](#)

Threat Superiority

- [Reduced Prioritization of Facial Threat in ASD](#)

Three-Word Phrases and Sentences

- [Telegraphic Speech](#)

Thyrotropin, Thyroid-Stimulating Hormone (TSH)

- [Maternal Hypothyroidism and Autism](#)

Thyroxine (T)

- [Maternal Hypothyroidism and Autism](#)

Tics

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Definition

Tics are the defining feature of Tourette syndrome. Tics are typically rapid, jerky movements

that may also involve vocalizations. Common tics include eye blinking, facial movements, head jerking, shoulder jerking, and abdominal tensing. Common vocal tics include throat clearing, grunting, coughing, hooting, and making various animal noises. Although tics are the defining feature of Tourette syndrome, tics may also occur in other conditions. Tic-like behaviors do occur in children with autism spectrum disorders.

See Also

- [Tourette Syndrome](#)

References and Reading

- Canitano, R., & Vivanti, G. (2007). Tics and Tourette syndrome in autism spectrum disorders. *Autism, 11*(1), 19–28.
- Larson, T., Anckarsater, H., Gillberg, C., Stahlberg, O., Carlstrom, E., Kadesjo, B., et al. (2010). The autism-tics, AD/HD and other comorbidities inventory (A-TAC): Further validation of a telephone interview for epidemiological research [Comparative Study Research Support, Non-U.S. Gov't]. *BMC Psychiatry, 10*, 1.
- Realmuto, G. M., & Main, B. (1982). Coincidence of Tourette's disorder and infantile autism. *Journal of Autism and Developmental Disorders, 12*(4), 367–372.
- Scahill, L., King, R. A., Lombroso, P., Sukhodolsky, D. G., & Leckman, J. F. (2011). Assessment and treatment of Tourette syndrome and other tic disorders. In A. Martin, L. Scahill, & C. Kratochvil (Eds.), *Pediatric psychopharmacology: Principles and practice* (pp. 516–530). New York: Oxford University Press.

TIME

- [Toddler Infant Motor Evaluation](#)

Time Experience in Autism Spectrum Disorder

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Abbreviations

ASD Autism spectrum disorder

Definition

The term *time experience* has been used in philosophy and descriptive psychopathology to delineate both the explicit and implicit inner experience of time. Explicit time experience (or “macro layer”) entails those aspects of time which we are consciously aware of – or may become aware of – such as the different dimensions of time encompassing past, present, and future or feelings such as boredom and time pressure (Vogel et al. 2018a). Implicit time experience (or “micro layer”) describes the unconscious prerequisites for perception, thinking, and behavior, usually with a focus on the continuity and future directionality of our subjective experience. Importantly, time experience as the experience of time passing by only marginally relates to our time perception (e.g., the ability to explicitly judge time intervals according to the clock or to generate time intervals via button press etc.) (Wearden 2015; Vogel et al. 2018a). With respect to psychopathology in general, time experience provides a holistic understanding of an individual syndrome in terms of a person’s inner experience (Stanghellini 2009, 2010). It has been speculated that the time experience of individuals with ASD differs from those without ASD (Zukauskas et al.

2009; Hohwy et al. 2016; Vogel et al. 2018a, 2019a). The respective proposals state that the processes underlying implicit time experience take more time in individuals with ASD, due to a more detailed perception of their environment. This might affect the intrapersonal coordination of information processing and might cause inter-subjective asynchronies potentially impeding both perceptual processing and complex behavior (Bloch et al. 2019). Persons with ASD may consequently experience continuous interruptions of their time caused by their environment. As a result, rituals and structured repetitive behavior are used to increase predictability, decrease future anxiety, and avoid interruption.

Historical Background

Research on time experience has a long-standing tradition in philosophy and psychiatry. Its roots go back to the philosophical discipline of phenomenology (Kupke 2009). Among the most prominent theories are the works of Edmund Husserl, Henri Bergson, and Martin Heidegger (Bergson 2001; Heidegger 2008; Husserl 2012). Their ideas were adopted by psychiatrists including Erwin Straus, Ludwig Binswanger, Eugene Minkowski, and Viktor Emil von Gebsattel. This early interdisciplinary approach has since inspired many other psychiatrists, psychologists, and philosophers alike. The subsequent research has largely focused on the phenomenological description of mental disorders, such as major depression (von Gebsattel 1928, 1954; Straus 1928; Lewis 1932; Binswanger 1960; Minkowski 1933; Fuchs 2001, 2013; Kupke 2005; Wyllie 2005; Ratcliffe 2012; Stanghellini et al. 2017; Vogel et al. 2018b), mania (Minkowski 1923; Binswanger 1960; Moskalewicz and Schwartz 2018; Martin et al. 2019), and schizophrenia (Bouman and Grünbaum 1929; Lewis 1932; Minkowski 1933; Vogeley and Kupke 2006; Fuchs 2007a; Kupke 2009; Stanghellini et al. 2015; Moskalewicz 2018; Vogel et al. 2019b). Some studies were

also dedicated to obsessive-compulsive disorder (von Gebsattel 1928, 1954), addictive behavior (von Gebsattel 1954; Moskalewicz 2016b), eating disorders, or personality disorders (Fuchs 2007b; Stanghellini and Mancini 2019). So far only very few studies have focused primarily on ASD (Zukauskas et al. 2009; Hohwy et al. 2016; Vogel et al. 2019b). However, an extensive and increasing body of research on time perception (e.g., the ability to explicitly judge time intervals according to the clock or to generate time intervals via button press etc.) has yielded interesting results for ASD and in turn inspired research on time experience (for review see Allman and Meck 2011; Falter and Noreika 2014; Allman and Falter 2015; Casassus et al. 2019).

Current Knowledge

“Lived Time”

The concept underlying time experience as a whole is commonly referred to as “lived time” (Straus 1928; von Gebsattel 1928; Fuchs 2001, 2013; Broome 2005; Kupke 2005; Wyllie 2005; Gallagher 2012; Moskalewicz 2016a; Stanghellini et al. 2015). In simple terms, it supposes that human consciousness is not only dependent on time as a prerequisite to our basic existence but that it is the direction of time which provides goals and meaning: actions were prepared in the immediate or far away past and are being performed in the present in order to influence the near to distant future (Vogel et al. 2018a).

Time experience may be differentiated into an explicit, controlled, and conscious aspect and an implicit, automatic, and unconscious aspect. Importantly, this division is conceptual; it is much harder to draw clear and distinctive lines between the two concepts in the empirical domain (Vogel et al. 2018a). However, from a scholarly perspective, the division into such an implicit and an explicit time experience enables an orderly scientific approach to the otherwise elusive research topic of human time.

Time is not static, but dynamic. According to the theory of lived time, the essential constituent of its dynamicity is the directionality toward the

future. This dynamic character has consequences for the experience of the present that may vary in its temporal extension depending on its relationship to the future (as determined by the past). For example, depending on a current situation and its demands, the “present” could refer to an hour, a particular day, or a life phase – or even all three. We live our time from the past in the present into the future (Kupke 2009; Vogel et al. 2018a). In emphasis of its future directionality, lived time has also been called the “becoming” (“Werden”) (von Gebsattel 1954; Straus 1947; Minkowski 1933; Fuchs 2013; Vogel et al. 2018a, b) or “striving” (“Streben”) (Minkowski 1923, p. 220; Minkowski 1933).

Explicit Time Experience

Explicit time experience (or “macro layer of time”) (for terminology see Fuchs 2005; Vogel et al. 2018a) entails the three dimensions of the structure of time: past, present, and future. Along these dimensions, we construct our individual autobiographical narratives and identities (Carr 1986; Stanghellini and Mancini 2017, p. 56 ff). Humans seemingly effortlessly locate themselves, their respective present, and their memories of the past on a virtual time line. Also, they devise plans and prospects for the future on that time line. For each individual these biographic events are inter-related and thereby provide each other with meaning and significance (Stanghellini 2017; Stanghellini and Mancini 2017, p. 57 ff). As long as the execution of the present aligns with our ideas about our past and future, we normally do not pay attention to time: explicit time blends with implicit time (Fuchs 2005; Vogel et al. 2018a). However, if our present activity in some way does not fit our narrative or is out of tune with our plans, we increasingly become aware of time (Vogel et al. 2018a).

The dynamic character can be illustrated by extreme cases of time pressure and boredom. Under time pressure, the available time is experienced as not sufficient to perform a particular action to reach a particular goal. As a consequence, the present appears to be passing too quickly to attain the said goal. If, however, we are bored, the present does not seem sufficiently

(full)filled. It may seem meaningless or empty in light of our plans and wishes. In terms of lived time, this can be understood as an inability to pursue a preferred activity or goal, and it explains why someone bored would prefer a different (more satisfying) activity (Fuchs 2005; Elpidorou 2018). This tension between present and future directionality causes time to be experienced as passing more slowly. Contrarily to both time pressure and boredom, we experience the so-called flow state when being completely immersed in lived time. During flow, external requirements and own abilities are optimally balanced (Csikszentmihályi 1990).

The explicit experience of time has been suggested as altered in a variety of psychiatric conditions (Straus 1928; von Gebattel 1928; Lewis 1932; Fuchs 2001, 2013; Broome 2005; Kupke 2005; Wyllie 2005; Gallagher 2012; Moskalewicz 2016a; Stanghellini et al. 2015; Vogel et al. 2018a). For example, its disturbances are a frequently reported symptom of depressive disorders. It has repeatedly been proposed that in major depression the future directionality of time experience loses extension, and the personal future appears closed off. Depressed patients lose their inherent ability to influence the future in the present. The present is rendered meaningless, the past unchangeably shameful, and the passage of time turns into a dragging and repetitious continuance of suffering. The emerging syndrome of disturbed time experience in the case of depression has been referred to as “blocked future” (von Gebattel 1928; Straus 1928; Fuchs 2001; Wyllie 2005; Stanghellini et al. 2017), “disturbance of (vital) becoming” (Straus 1928; von Gebattel 1954; Minkowski 1933; Fuchs 2013), “disturbance of protention” (Binswanger 1960), or “disorder/disturbance of conation” (Stanghellini et al. 2017) (for review see Vogel et al. 2018a, b).

With regard to time experience, ASD stands out among psychiatric diagnoses as the conceptualization of ASD in the International Classification of Diseases (ICD) (World Health Organization. 1992), and the Diagnostic and Statistical Manual of Mental Disorders (DSM) (American Psychiatric Association 2013)

contains a distinctly temporal symptom: “restricted, repetitive patterns of behavior, interests, or activities” (American Psychiatric Association 2013, p. 50). Temporal structure, repetitive routine, difficulty or inability to spontaneity, and insistence on sameness have recently been described in terms of time experience as part of an overarching syndrome. In a qualitative study consulting adults with high-functioning ASD about inner time experience, Vogel et al. (2019a) were able to describe symptom formation in terms of time experience. Building on earlier qualitative research (Zukauskas et al. 2009; Vogel et al. 2018a), the results indicate that routine was described as bringing about a pleasant fading out of the felt passage of time as an uninhibited immersion into lived time. Non-surprisingly, disruptions of routine were experienced as unpleasant. Due to a felt unpredictability, individuals with ASD reported a particular fear and anxiety concerning the very close to near future. This fear seemed to inspire excessive planning and structuring of the future through further routine. As long as the plan succeeded, time experience followed the same rules in persons with and without ASD.

However, plans can be neither flawless nor rigidly adhered to in everyday life without a certain ability to take into account unpredictability and surprise. Hence, the pleasant continuation of routine is usually interrupted at some point – especially when it comes to particularly unpredictable and complex situations such as social interactions. These interruptions are an external cause of a fragmentation of time experience. This discontinuation of the time line relates to the experience of past and future events that are seemingly factual and isolated from the present (Zukauskas et al. 2009; Vogel et al. 2019a). Prospectively overcoming and retrospectively making sense of these separate temporal entities seems to necessitate a considerable degree of effort. Hence it has been argued that the degree of cognitive functioning and overall resources attributable to planning and integrating of temporal structure determines symptom severity and potentially also depressive comorbidity in ASD (Boucher 2001; Fein et al. 2013).

Implicit Time Experience

Implicit time experience (or “micro layer of time”) (for terminology see Fuchs 2005; Vogel et al. 2018a) refers to the unconscious basis of experience. Its most important foundations are continuity and the future directionality of this subpersonal, unconscious basis of consciousness/experience (Dainton 2006; Fuchs 2013; Wiese 2017). It is this fundamental continuity and directionality that makes possible the similar principles in explicit time experience (Vogel et al. 2018a). The same principles have recently found their way into neuroscientific concepts as the putative basic requirements of consciousness in the brain (Hohwy et al. 2016; Wiese 2017; Vogel et al. 2018a). A variety of psychological and neurophysiological processes have been proposed as underlying the supposed alterations in time perception and experience in ASD (e.g., genetic variances (Wimpory et al. 2002; Nicholas, B. et al. 2007) or hormonal dysregulation (Tordjman et al. 2015)). Concerning the theory of interrupted time experience, examining its underlying implicit mechanisms requires the examination of the unconscious processes constituting the flow or stream of consciousness (Vogel et al. 2018a). As a starting point of inquiry, it has recently been suggested that predictive processing or Bayesian perceptual inference constitutes the passage of time (Hohwy et al. 2016; also see Vogel et al. 2018a). The major advantage of this line of reasoning is that it elegantly combines both phenomenological ideas grounded largely in the Husserlian analysis of time consciousness and trending neuroscientific theories (Hohwy et al. 2016; Wiese 2017).

Predictive processing in the brain assumes that the brain calculates its output (i.e., perceptions and experience) through the combination of preconceived top-down inferences and bottom-up perceptual information (e.g., Friston et al. 2014). Put simply, at any point in time, the brain creates a hypothesis of what its next perceptual input will most likely be. This prediction is based on the previous input, i.e., earlier experience and directly preceding percepts. This hypothesis is called a prior. The prior is compared to the actual perception. As long as

perceptual input and prior match, the prior remains active and the output remains unchanged. If incoming information and prior do not match, a prediction error is calculated. In case of a prediction error, the neural system passes the error along a hierarchical organization of alternative potential priors in order to explain the error (i.e., unexpected or unusual percept). This hierarchy ranges from lower priors reflecting simple, specific assumptions on the causes and contents of perception to higher priors reflecting general beliefs. The more “unexpected” a new perceptual input, the higher the resulting prediction error will be, and the further it potentially will have to travel up the hierarchy until it has been explained away and resolved. The result from this calculation is called the posterior. The active prior is subsequently replaced by the posterior which thereby becomes the next percept’s better fitting prior. The transition from prior to new prior has been postulated to produce the experience of temporal flow (Hohwy et al. 2016).

Theorists on Bayesian processing in the brain have proposed so-called hypo-priors for ASD (Pellicano and Burr 2012; Hohwy et al. 2016), i.e., priors which prioritize more perceptual input and hence provide more perceptual detail. A brain with higher-detailed priors would need more time to process and extract relevant information, comparatively slowing down processing speed and the internal flow of information (Hohwy et al. 2016). If the resultant speed of prior changes drops below the speed of changes in the external world, the external changes might appear as happening too fast (Gepner and Féron 2009; Hohwy et al. 2016). In other words, due to a more detailed perception, individuals with ASD may not be able to process information fast enough to keep up with their surroundings. The need to process new, fast incoming and especially unexpected, spontaneous information effectively interrupts the currently ongoing processing of information. Although the prior updating process does not directly reflect experienced speed of time, the divergence of internal and external temporal flow may become aware in changes in the explicit velocity of time (Fuchs 2005; Vogel et al. 2018a).

Interrupted Time Experience

Complementarily to these qualitative descriptions and hypotheses, observations from experimental psychology can also be read as indications of an interrupted time experience in ASD. Wimpory et al. (2002) proposed a “Social Timing Hypothesis of Autism.” According to the hypothesis, persons with ASD cannot sufficiently synchronize with others on a behavioral level, due to suspected anomalies in clock genes (Nicholas et al. 2007). Boucher et al. (2007) studied children with ASD and found impairment in diachronic thinking. Diachronic thought (Montangero 1992) entails the ability to imagine past or future stages of a present situation, object permanence, and the ability to infer a process from successive events and vice versa. In other words, children with ASD may have a differing concept of temporal continuity or a delayed development thereof. Concerning the passage of time and events, an environment changing too fast due to aberrant connectivity and neuronal synchronization has been discussed as constituting ASD (Gepner and Féron 2009). Atypicalities in intrapersonal synchrony have been speculated to cause interpersonal asynchronies in ASD (Bloch et al. 2019). Although it still remains unclear, whether these and other variations in ASD are due to distinct impairments in time perception, they have been suggested to produce difficulties in interpersonal and communicative coordination (for review see Allman and Meck 2011; Falter and Noreika 2014; Allman and Falter 2015; Casassus et al. 2019). In accordance with interrupted time experience, stereotyped behavior may serve as a compensatory mechanism for these deficits (Allman et al. 2011).

The integrated analysis of time and temporality in ASD understands patients’ symptoms and individual syndromes as a meaningful whole (Stanghellini 2009, 2010) which emerges from a distinct experience of time. Repeat interruptions by an environment that is too fast or too unpredictable cause subjective difficulties depending on the availability of cognitive resources. Due to their innate complexity and relatively high spontaneity, we may assume that communicative situations are especially difficult.

Symptoms such as repetitions, routine, insistence on sameness, and intensified focus (American Psychiatric Association 2013, p.50) can be interpreted as compensatory mechanisms to reestablish or guarantee undisturbed time experience.

Future Directions

Interrupted time experience in ASD remains a hypothesis primarily derived from qualitative exploration. As with other findings on time and ASD, the reliability and specificity of the theory remain uncertain (see Falter and Noreika 2014 for discussion; and Casassus et al. 2019 for review). However, it holds tremendous potential for further investigation of time experience in ASD. In its explorative and observational character and the need to contextualize and understand the inner lived experience of persons with ASD, interrupted time experience can become informative for research on the putative underpinnings of human perception and experience in ASD, and beyond.

A potential neural target for research on time experience both generally and in ASD is the so-called default mode of the brain. The default mode (or resting state) is the brain’s activity measurable while the brain is not engaged in any specific task (Raichle et al. 2001; Northoff 2016a). Changes in the temporospatial activity of the default mode have recently been put forward as being related to psychopathological phenomena of time experience (Northoff 2016a, b; Northoff and Stanghellini 2016). Should the concept prove true, alterations in the brain’s resting state as repeatedly described in ASD (e.g., Weng et al. 2010; Anderson et al. 2011; Joshi et al. 2017) could be used to explain altered time experience.

Future research should further attempt an operationalization of interrupted time experience as a descriptive syndrome. Further conceptualization and the inclusion into a standardized diagnostic protocol allows for testing of the experiential hypothesis. If such a descriptive approach succeeds in clinical practice, it would inform and specify diagnostic procedure, foster our

Time Experience in Autism Spectrum Disorder, Table 1 Descriptive view of the syndrome of interrupted time experience in ASD. (Amended acc. to Vogel et al. 2019a)

Experience	Definition	Suggestions for related questions
The faded out passage of time	The passage of time is hardly or vaguely felt. A bad sense of time. Fading is usually amplified by structured and routine activity	“Do you have a good or a bad sense of time?”; “How do you keep track of time?”; “What makes you forget about time?”
The present as confined activity	The present consists of one distinct activity, usually deemed important, necessary, or meaningful. Routine is experienced as beneficial. Interruptions are experienced as distressing	“What does ‘the present’ mean to you?”; “How does routine/repetition influence your experience of the present?”; “Is the present a point in time, or is it extended?”
The past as isolated memories	The past is described as a repetition of facts. Although meaningful and influential, the past is not necessarily part of the lived present	“What do your past experiences (<i>or a specific memory</i>) mean to you?”; “Do your past experiences influence your decisions? If yes, how?”
The planned future	The future is experienced as uncertain and frightening. The future is rigorously planned to avoid unexpected interruption	“What does the future mean to you?”; “Are you afraid of the future/the unexpected?”; “Do you distinguish between a near and a far-away future?” “Do you feel that it is possible to plan for the future?”; “Do you spend much time/effort on planning? Describe to me, how you would plan your day?”; “Why do you make plans?”

understanding of persons with ASD, and help identify, therapeutically employ, and improve strengths and resources.

Vogel et al. (2019a) have provided a table (Table 1) describing a potential syndromic description of interrupted time experience. We have amended the table with explorative questions designed to approach, identify, and potentially understand the time experience of a patient with suspected ASD in a diagnostic interview. Of course these questions are preliminary, are difficult to understand, have never been used systematically, and will need further study before being able to be more widely used in clinical practice. Additionally quantifying psychometric scales need to be developed both to facilitate standardized clinical application and a broader use in scientific research.

References and Reading

Allman, M., & Falter, C. (2015). Abnormal timing and time perception in autism spectrum disorder? A review of the evidence. In A. Vatakis & M. Allman (Eds.), *Time distortions in mind – Temporal processing in clinical populations* (pp. 37–56). Leiden: Brill Academic Publishers.

Allman, M. J., & Meck, W. H. (2011). Pathophysiological distortions in time perception and timed performance. *Brain*, 135(3), 656–677.

Allman, M. J., DeLeon, I. G., & Wearden, J. H. (2011). Psychophysical assessment of timing in individuals with autism. *American Journal on Intellectual and Developmental Disabilities*, 116(2), 165–178.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Publishing.

Anderson, J. S., Nielsen, J. A., Froehlich, A. L., Dubray, M. B., Druzgal, T. J., Cariello, A. N., et al. (2011). Functional connectivity magnetic resonance imaging classification of autism. *Brain*, 134, 3739–3751.

Bergson, H. (2001). *Time and free will: An essay on the immediate data of consciousness*. Mineola, New York: Dover Publications.

Binswanger, L. (1960). *Melancholie und Manie*. Pfullingen: Neske.

Bloch, C., Falter-Wagner, C., Georgescu, A. L., & Vogeley, K. (2019). INTRApersonal synchrony as constituent of INTERpersonal synchrony and its relevance for autism spectrum disorder. *Frontiers in Robotics and AI*, 6, 73.

Boucher, J. (2001). Time-parsing and autism. In C. Hoerl & T. McCormack (Eds.), *Time and memory: Issues in philosophy and psychology* (pp. 111–135). Oxford: Oxford University Press.

Boucher, J., Pons, F., Lind, S., & Williams, D. (2007). Temporal cognition in children with autistic spectrum disorders: Tests of diachronic thinking. *Journal of Autism and Developmental Disorders*, 37(8), 1413–1429.

- Bouman, L., & Grünbaum, A. A. (1929). Eine Störung der Chronognose und ihre Bedeutung im betreffenden Symptombild. *Monatsschrift für Psychiatrie und Neurologie*, 73(1), 1–39.
- Broome, M. R. (2005). Suffering and eternal recurrence of the same: The neuroscience, psychopathology, and philosophy of time. *Philosophy, Psychiatry, and Psychology*, 12(3), 187–194.
- Carr, D. (1986). *Time, narrative, and history*. Bloomington: University of Indiana Press.
- Casassus, M., Poliakoff, E., Gowen, E., Poole, D., & Jones, L. A. (2019). Time perception and autistic spectrum condition: A systematic review. *Autism Research*, 12(10), 1440–1462.
- Csikszentmihályi, M. (1990). *Flow: The Psychology of Optimal Experience*. New York: Harper & Row.
- Dainton, B. (2006). *Stream of consciousness: Unity and continuity in conscious experience*. London, UK: Taylor & Francis.
- Elpidorou, A. (2018). The bored mind is a guiding mind: Toward a regulatory theory of boredom. *Phenomenology and the Cognitive Sciences*, 17(3), 455–484.
- Falter, C. M., & Noreika, V. (2014). Time processing in developmental disorders: A comparative view. In *Subjective time: The philosophy, psychology, and neuroscience of temporality*. Cambridge: MIT Press.
- Fein, D., Barton, M., Eigsti, I. M., Kelley, E., Naigles, L., Schultz, R. T., et al. (2013). Optimal outcome in individuals with a history of autism. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 54(2), 195–205.
- Friston, K. J., Stephan, K. E., Montague, R., & Dolan, R. J. (2014). Computational psychiatry: The brain as a phantastic organ. *The Lancet Psychiatry*, 1(2), 148–158.
- Fuchs, T. (2001). Melancholia as a desynchronization: Towards a psychopathology of interpersonal time. *Psychopathology*, 34(4), 179–186.
- Fuchs, T. (2005). Implicit and explicit temporality. *Philosophy, Psychiatry, and Psychology*, 12(3), 195–198.
- Fuchs, T. (2007a). The temporal structure of intentionality and its disturbance in schizophrenia. *Psychopathology*, 40(4), 229–235.
- Fuchs, T. (2007b). Fragmented selves: Temporality and identity in borderline personality disorder. *Psychopathology*, 40(6), 379–387.
- Fuchs, T. (2013). Temporality and psychopathology. *Phenomenology and the Cognitive Sciences*, 12(1), 75–104.
- Gallagher, S. (2012). Time, emotion, and depression. *Emotion Review*, 4(2), 127–132.
- Gepner, B., & Féron, F. (2009). Autism: A world changing too fast for a mis-wired brain? *Neuroscience and Biobehavioral Reviews*, 33(8), 1227–1242.
- Heidegger, M. (2008). *Being and Time*. New York: HarperPerennial.
- Hohwy, J., Paton, B., & Palmer, C. (2016). Distrusting the present. *Phenomenology and the Cognitive Sciences*, 15(3), 315–335.
- Husserl, E. (2012). *On the phenomenology of the Consciousness of internal Time (1893–1917)* (Vol. 4). Springer Science & Business Media.
- Joshi, G., Arnold Anteraper, S., Patil, K., Semwal, M., Goldin, R., Furtak, S., et al. (2017). Integration and segregation of default mode network resting-state functional connectivity in transition-age males with high-functioning autism spectrum disorder: A proof of concept study. *Brain Connectivity*, 7(9), brain.2016.0483.
- Kupke, C. (2005). Lived time and to live time: A critical comment on a paper by Martin Wyllie. *Philosophy, Psychiatry, and Psychology*, 12(3), 199–203.
- Kupke, C. (2009). *Der Begriff Zeit in der Psychopathologie*. Berlin: Parodos Verlag.
- Lewis, A. (1932). The experience of time in mental disorder. *Proceedings of the Royal Society of Medicine*, 25(5), 611–620.
- Martin, W., Gergel, T., & Owen, G. S. (2019). Manic temporality. *Philosophical Psychology*, 32(1), 72–97.
- Minkowski, E. (1923). Bleulers Schizoidie und Syntonie und das Zeiterlebnis. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, 82(1), 212–230.
- Minkowski, E. (1933). *Le temps vécu: Etudes phénoménologiques et psychopathologique*. Paris: Delachaux Collection de l'Evolution Psychiatrique.
- Montangero, J. (1992). The development of a diachronic perspective in children. In *Time, action and cognition* (pp. 55–65). Dordrecht: Springer.
- Moskalewicz, M. (2016a). Disturbed temporalities. Insights from phenomenological psychiatry. *Time & Society*, 25(2), 234–252.
- Moskalewicz, M. (2016b). Lived time disturbances of drug addiction therapy newcomers. A qualitative, field phenomenology case study at Monar-Markot Center in Poland. *International Journal of Mental Health and Addiction*, 14(6), 1023–1038.
- Moskalewicz, M. (2018). Toward a unified view of time: Erwin W. Straus' phenomenological psychopathology of temporal experience. *Phenomenology and the Cognitive Sciences*, 17(1), 65–80.
- Moskalewicz, M., & Schwartz, M. A. (2018). Temporal experience in mania. *Phenomenology and the Cognitive Sciences*, 1–14.
- Nicholas, B., Rudrasingham, V., Nash, S., Kirov, G., Owen, M. J., & Wimpory, D. C. (2007). Association of Per1 and Npas2 with autistic disorder: Support for the clock genes/social timing hypothesis. *Molecular Psychiatry*, 12(6), 581.
- Northoff, G. (2016a). How do resting state changes in depression translate into psychopathological symptoms? From “spatiotemporal correspondence” to “spatiotemporal psychopathology”. *Current Opinion in Psychiatry*, 29(1), 18–24.

- Northoff, G. (2016b). Spatiotemporal psychopathology I: No rest for the brain's resting state activity in depression? Spatiotemporal psychopathology of depressive symptoms. *Journal of Affective Disorders*, 190, 854–866.
- Northoff, G., & Stanghellini, G. (2016). How to link brain and experience? Spatiotemporal psychopathology of the lived body. *Frontiers in Human Neuroscience*, 10, 76.
- Pellicano, E., & Burr, D. (2012). When the world becomes 'too real': A Bayesian explanation of autistic perception. *Trends in Cognitive Sciences*, 16(10), 504–510.
- Raichle, M. E., MacLeod, A. M., Snyder, A. Z., Powers, W. J., Gusnard, D. A., & Shulman, G. L. (2001). A default mode of brain function. *Proceedings of the National Academy of Sciences*, 98(2), 676–682.
- Ratcliffe, M. (2012). Varieties of temporal experience in depression. *The Journal of Medicine and Philosophy*, 37(2), 114–138.
- Stanghellini, G. (2009). The meanings of psychopathology. *Current Opinion in Psychiatry*, 22(6), 559–564.
- Stanghellini, G. (2010). A hermeneutic framework for psychopathology. *Psychopathology*, 43(5), 319–326.
- Stanghellini, G. (2017). *Lost in dialogue*. Oxford: Oxford University Press.
- Stanghellini, G., & Mancini, M. (2017). *The therapeutic interview. Emotions, values, and the life-world*. Cambridge: Cambridge University Press.
- Stanghellini, G., & Mancini, M. (2019). Abnormal time experiences in persons with feeding and eating disorder: A naturalistic explorative study. *Phenomenology and the Cognitive Sciences*, 1–15.
- Stanghellini, G., Ballerini, M., Presenza, S., Mancini, M., Raballo, A., Blasi, S., & Cutting, J. (2015). Psychopathology of lived time: Abnormal time experience in persons with schizophrenia. *Schizophrenia Bulletin*, 42(1), 45–55.
- Stanghellini, G., Ballerini, M., Presenza, S., Mancini, M., Northoff, G., & Cutting, J. (2017). Abnormal time experiences in major depression: An empirical qualitative study. *Psychopathology*, 50(2), 125–140.
- Straus, E. W. (1928). Das Zeiterlebnis in der endogenen Depression und in der psychopathischen Verstimmung. *Monatsschrift für Psychiatrie und Neurologie*, 68, 640–656.
- Straus, E. W. (1947). Disorders of personal time in depressive states. *Southern Medical Journal*, 40(3), 254–259.
- Tordjman, S., Davlantis, K. S., Georgieff, N., Geoffray, M. M., Speranza, M., Anderson, G. M., et al. (2015). Autism as a disorder of biological and behavioral rhythms: Toward new therapeutic perspectives. *Frontiers in Pediatrics*, 3(1).
- Vogel, D. H. V., Falter-Wagner, C. M., Schoofs, T., Krämer, K., Kupke, C., & Vogeley, K. (2018a). Flow and structure of time experience—concept, empirical validation and implications for psychopathology. *Phenomenology and the Cognitive Sciences*, 1–24.
- Vogel, D. H. V., Krämer, K., Schoofs, T., Kupke, C., & Vogeley, K. (2018b). Disturbed experience of time in depression – Evidence from content analysis. *Frontiers in Human Neuroscience*, 12, 66.
- Vogel, D. H. V., Beeker, T., Haidt, T., Kupke, C., Heinze, M., & Vogeley, K. (2019a). Disturbed time experience during and after psychosis. *Schizophrenia Research: Cognition*, 17, 100136.
- Vogel, D., Falter-Wagner, C. M., Schoofs, T., Krämer, K., Kupke, C., & Vogeley, K. (2019b). Interrupted time experience in autism spectrum disorder: Empirical evidence from content analysis. *Journal of Autism and Developmental Disorders*, 49(1), 22–33.
- Vogeley, K., & Kupke, C. (2006). Disturbances of time consciousness from a phenomenological and a neuroscientific perspective. *Schizophrenia Bulletin*, 33(1), 157–165.
- von Gebsattel, V. E. F. (1928). Zeitbezogenes Zwangsdenken in der Melancholie. *Nervenarzt*, 1, 275–287.
- von Gebsattel, V. E. F. (1954). Störungen des Werdens und des Zeiterlebens psychiatrischer Erkrankungen. In *Prolegomena einer Medizinischen Anthropologie*. Berlin/Göttingen/Heidelberg: Springer.
- Wearden, J. H. (2015). Passage of time judgements. *Consciousness and Cognition*, 38(2014), 165–171.
- Weng, S. J., Wiggins, J. L., Peltier, S. J., Carrasco, M., Risi, S., Lord, C., & Monk, C. S. (2010). Alterations of resting state functional connectivity in the default network in adolescents with autism spectrum disorders. *Brain Research*, 1313, 202–214.
- Wiese, W. (2017). Predictive processing and the phenomenology of time consciousness. In T. Metzinger & W. Wiese (Eds.), *Philosophy and predictive processing*. Frankfurt am Main: MIND Group.
- Wimpory, D., Nicholas, B., & Nash, S. (2002). Social timing, clock genes and autism: A new hypothesis. *Journal of Intellectual Disability Research*, 46(4), 352–358.
- World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organization.
- Wyllie, M. (2005). Lived time and psychopathology. *Philosophy, Psychiatry, and Psychology*, 12(3), 173–185.
- Zukauskas, P. R., Siltan, N., & Assumpção, F. B., Jr. (2009). Temporality and Asperger's syndrome. *Journal of Phenomenological Psychology*, 40(1), 85–106.

Time Interval

► [Latency, Second Edition](#)

Time Management

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Definition

Time management is the process through which a person plans for, initiates, and completes one or more activities consistent with a predetermined sequence and/or schedule. Time management is important for many everyday activities, such as getting ready for school or work, completing responsibilities on time, arranging priorities, etc. For individuals with autism spectrum disorders (ASDs), the time management process can be very challenging to complete independently. Some individuals with ASDs may need explicit teaching with initial or ongoing support to manage their time effectively.

To help address time management issues, researchers and practitioners have used a variety of supports. Visual supports may be used to help arrange several activities into a logical order. Once arranged, visual supports then show a particular sequence to be followed (e.g., activity schedules; McClannahan and Krantz 2010). While visual support can be used to help a person with an ASD complete a routine without assistance from another person, this does not mean he or she is independently completing the entire time management process.

Learning to manage one's time requires consideration of a wide variety of variables, such as relevant activities, time frames, and resources. Interventions designed to improve time management may need to address each of these areas if the person is to learn to manage their time independently. For individuals who have not yet mastered time management, visual supports, such as activity schedules, may be particularly effective in increasing independent completion of even

complex routines with little or no intervention from care givers during the activities.

References and Reading

McClannahan, L. E., & Krantz, P. J. (2010). *Activity schedules for children with autism: Teaching independent behavior* (2nd ed.). Bethesda: Woodbine House.

Time Sampling Procedures

► Discontinuous Data Collection

Time Trends in Diagnosis

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Definition

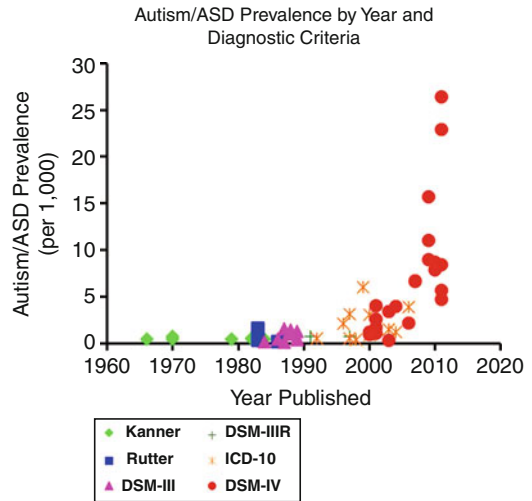
Autism is one of several pervasive developmental disorders (PDDs) characterized by atypical development in socialization, communication, and behavior. Symptoms of autism and related conditions are typically present before age 3 years and often are accompanied by unusual patterns in cognitive functioning, learning, attention, and sensory processing. In the past decade, the conditions of autistic disorder, Asperger's disorder, and pervasive developmental disorder – not otherwise specified – have been referred to as the “autism spectrum disorders” (ASDs). ASDs are defined by a pattern of development which emerges in the first few years of life, and there is currently no biologic test to confirm the diagnosis. Therefore, these developmental disabilities are typically diagnosed by professionals who have an advanced degree and training in the evaluation of developmental status, such as physicians and psychologists. Determining that a child's

developmental profile is consistent with one of the ASDs requires consideration of the way the child communicates, interacts, behaves, learns, and plays and is guided by diagnostic criteria. Since autism was first described about 70 years ago, these criteria have changed.

Historical Background

Autism was first described in 1943 by psychiatrist Leo Kanner working at Johns Hopkins University (Kanner 1943). Dr. Kanner described 11 children referred to him because of unusual development. He noted common patterns among the children's behavior including "extreme autism, obsessiveness, stereotypy, and echolalia." He also noted similarities to children with schizophrenia but offered that autism was unique in the "extreme aloneness" and lack of social contact with "intelligent relation to objects" from very early in life. Also in the 1940s, Dr. Hans Asperger of Austria was also describing a group of children he saw clinically who had significant social impairments but more typical language content than the children described by Kanner. They were not aware of each other's work, but both used the term "autism" to describe a unique group of children. Although there are reports of socially isolated individuals with unusual behaviors throughout history, these efforts were the first to describe the developmental pattern as a specific psychiatric condition (Rutter and Schopler 1987).

Kanner's description (1943; Eisenberg and Kanner 1956) of autism provided the dominant guidance for describing the features of autism until the 1970s. Although Kanner noted that autism had a unique profile different from schizophrenia, autism was classified as a form of early-onset schizophrenia (Creak et al. 1964) in the American Psychiatric Association's Diagnostic and Statistical Manual (DSM) (American Psychiatric Association [APA] 1952, 1968) (see Fig. 1 summarizing autism diagnostic criteria). The DSM is the standard for clinical diagnosis of psychiatric disorders which impacts the way in which professionals identify and label these



Time Trends in Diagnosis, Fig. 1 Summary of Autism/ASD prevalence by year and diagnostic criteria

conditions. The DSM is periodically revised. During the 1970s, there was a shift to distinguish autism from childhood schizophrenia with the recognition that the latter usually involves a period of typical functioning with the later emergence of unusual thoughts and behaviors and a loss of contact with reality. Criteria for autism by Rutter (1978) and Ritvo and Freeman (1978) emphasized the early onset of autism in the first three years of life and the differences and overlap between autism and mental retardation. Around the same time, Lorna Wing translated Asperger's earlier descriptions of more verbal children with autism into English. She also described social, communication, and behavioral features as the core of autism but noted that there is a continuum of social functioning indicated by three groups: "aloof," "passive," and "active but odd" (Wing and Gould 1979).

The *DSM-III* (APA 1980) distinguished autism as one of the "pervasive developmental disorders" and severed the connection to schizophrenia. Autism was now classified as one of several related developmental disorders where the typical social and language skills and behaviors do not occur as expected from very early in life. The next edition, *DSM III-R* (APA 1987), revised the specific criteria which make up the social,

communication, and behavioral symptoms of autism and the lowest threshold diagnosis of PDD-NOS. More significant changes were made with the DSM-IV (APA 1994) and the subsequent text revision (APA 2000) which mirrored the broadening of conditions included as a PDD for the International Classification of Diseases, tenth edition (ICD-10) (WHO 1992). Starting in the early 1990s, Asperger's disorder was included for the first time as a PDD. Since the publication of the *ICD-10* and *DSM IV* and *IVTR* criteria for the PDDs in the early 1990s, a great deal has changed in the identification and conceptualization of autism and related disorders. As noted earlier, three of the conditions are now referred to as the ASDs to reflect the "spectrum" of deficits and skills across multiple domains which share core social, communication, and behavioral features present during the first 3 years of life. However, within this spectrum, individual features and functioning across domains may vary from mildly to severely affected. In addition, there is a recognition that ASDs can co-occur across the whole range of intellectual functioning and with other developmental and medical conditions.

Current Knowledge

In the past 20 years, there have been increases in the numbers of people identified with an ASD compared to the past. This has sparked debate about how much of the increases in the prevalence of ASDs are explained either by changes in identification patterns and, therefore, an artifact of changing diagnostic criteria and awareness or by a true increase in symptoms among children born in more recent times. One issue to examine is whether the changing diagnostic criteria for the ASDs have influenced estimates of the prevalence of ASDs over time. The first epidemiologic studies of autism prevalence in the population were done by Lotter in the UK (1966, 1967) and were based on Kanner's criteria. Since that time, many studies based on each of the major diagnostic criteria have estimated the prevalence of ASDs (see graph). From the 1940s until the

1980s, autism primarily referred to more severely affected individuals with autistic disorder and was thought to be rare, affecting approximately one in every 2,000 (0.05%) children (Fombonne 2009; Rutter 2005). Several studies using *ICD-10* and *DSM-IV* have been done in industrialized countries identifying not only autism but the wider spectrum with surprising consistency indicating a best estimate of combined ASD prevalence at 6 or 7 of every 1,000 (0.6–0.7%) children with ASDs (CDC 2007; Fombonne 2009). These estimates are more than 10 times higher than estimates using earlier criteria. However, some of the most recent population-based studies have documented even higher ASD prevalence estimates of >1% (Baird et al. 2006; Baron-Cohen et al. 2009; CDC 2009; Honda et al. 2005; Kadesjo et al. 1999; Kogan et al. 2009) and even as high as 2.6% (Kim et al. 2011; Roelfsema et al. 2012) among children in areas of Asia, Europe, and North America. Can the difference of 1 in 2,000 children to about 1 in 100 be accounted for by the shift from seeing autism as a severe, rare disorder to the broader spectrum? One effort to retrospectively apply more modern criteria to an older study estimated a prevalence of about 4 per 1,000 children, indicating that older studies underestimated prevalence as they considered a more strict interpretation of autism (Heussler et al. 2001). Although earlier studies likely underestimated ASD prevalence by today's standards, there are many other factors to take into account when sorting out time trends in ASD identification. For example, the age of onset required for diagnosis has changed over time and can influence ASD prevalence estimates. In addition, there are multiple identification and risk factors which could also influence the identification and/or development of an ASD.

Few studies have tracked the same population over time to evaluate ASD prevalence changes. The Autism and Developmental Disabilities Monitoring (ADDM) Network [established by the Centers for Disease Control and Prevention (CDC) in 2007 to characterize and track the prevalence of ASDs among children in multiple areas of the United States] reported an overall 57%

increase in identified ASD prevalence among 8-year-old children over a 4-year period from 2002 to 2006 (Centers for Disease Control and Prevention [CDC] 2009). On average, ASD prevalence increased across all sex, racial/ethnic, and cognitive functioning subgroups. Although some of the changes in ASD prevalence could be attributed to improved identification in the areas studied, particularly of some subgroups such as Hispanic children and children without intellectual disabilities, these patterns were not consistent among sites and cannot completely explain a greater than 50% prevalence increase in such a short time period. These findings, in addition to trends across multiple single studies, raise questions about what other identification and potential risk factors in addition to changes in diagnostic criteria are influencing increasing prevalence estimates of ASDs. At this point, it is clear that shifts in the diagnostic criteria for ASDs have had an impact on the identified prevalence, but no single factor explains the changes identified in ASD prevalence over time, and much needs to be done to understand the relative contribution of the multiple factors involved.

Kanner Criteria (1943)

- Inability to relate to people; language does not convey meaning; food refusal; fear of noises; limited but good relations with objects; present from the beginning of life

Eisenberg and Kanner (1956)

- Extreme self-isolation (lack of affective contact) and extreme repetitive and ritualistic behavior (perseveration of sameness) DSM-I (1952) and DSM-II (1968)
- Part of schizophrenia, childhood type manifested by autistic, atypical, and withdrawn behavior; unevenness, immaturity, or inadequacy of development

Creak et al. (1964)

- Schizophrenic syndrome in childhood including gross and sustained impairment of

emotional relationships with people, lack of self-awareness, preoccupation with objects, resistance to change, abnormal perceptual and sensory issues, excessive anxiety, delayed language, unusual activity levels, unusual intellectual skills

Rutter Criteria (1978)

- Emphasized social and language development delayed and unusual for the child's intellectual ability; insistence on sameness, abnormal pre-occupations, or resistance to change; onset before 30 months

DSM-III (1980)

- Differentiated autism from schizophrenia (not a psychiatric disorder, but developmental)
- Concept of "PDD" introduced: infantile autism; childhood-onset PDD; atypical PDD
- Autism: Pervasive lack of responsiveness to other people (autism); gross deficits in language development or peculiar speech patterns; bizarre responses to the environment; onset before 30 months
- Childhood-onset PDD: gross and sustained impairment in social relationships with at least three of the following: excessive anxiety, inappropriate affect, resistance to change, movement oddities, abnormalities of speech, sensory issues, self-injury; onset between 30 months and 12 years

DSM-III-R (1987)

- Concept of PDD continued: autism and PDD-NOS
- *Autism: has eight criteria indicating qualitative impairment in reciprocal social interaction, in verbal and nonverbal communication and in imaginative activity, and in markedly restricted repertoire of activities and interests; onset during infancy or early childhood*
- PDD-NOS: qualitative impairment in the development of reciprocal social interaction and of verbal and nonverbal communication skills; may have a markedly restricted repertoire of activities and interests

DSM-IV (1994) and DSM-IV TR (2000)

- Similar to ICD-10 (1992)
- Expanded PDD concept: autistic disorder; Asperger syndrome; Rett syndrome; CDD; PDD-NOS
- Autism: has six criteria indicating qualitative impairment in social interaction and communication, and restricted, repetitive, and stereotyped patterns of behavior; onset prior to 36 months
- PDD-NOS: Severe and pervasive impairment in the development of reciprocal social interaction or verbal and nonverbal communication skills, or when stereotyped behavior, interests, and activities are present; includes “atypical autism” presentations that do not meet the criteria for autistic disorder because of late age at onset, atypical symptomatology, or sub-threshold symptomatology, or all of these

DSM 5 (estimated 2013)

- Proposes to collapse PDD subtypes into a single category of “autism spectrum disorders”
- Proposed ASD: persistent deficits in social communication and social interaction across contexts not accounted for by general developmental delays; restricted, repetitive patterns of behavior, interests, or activities; present in early childhood; symptoms limit and impair everyday functioning
- <http://www.dsm5.org>

Future Directions

Despite the fact that ASD prevalence estimates based on the DSM-IVTR criteria continue to increase, there will be another revision of the diagnostic criteria for autism and related conditions with the upcoming publication of the fifth edition of the DSM (<http://www.dsm5.org>) anticipated for 2013. The current proposed criteria collapse several of the PDD subtypes into one category of autism spectrum disorders. As it stands now, there is greater focus on distinguishing core social communication and interaction and repetitive behaviors, interests, and activity symptoms of ASDs in the context of language level, cognitive functioning,

and other indicators of functioning. In the clinical and research fields, there is a move away from the categorical identification of autism as being present or not present with the recognition that some of the core domains associated with ASDs are distributed in a more continuous way in the population (Constantino 2011). There is also increasing interest improving the utility of diagnoses to inform everyday supports and services for people with ASDs. This includes incorporating information on developmental changes and better understanding impact of ASD characteristics on daily functioning (Happé 2011; Lord 2011). The significance of the upcoming revised diagnostic criteria for the ASDs remains to be seen, but impact on identified prevalence of ASDs is highly likely (Mattila et al. 2011).

See Also

- ▶ [Alternative Diagnostic Concepts](#)
- ▶ [Diagnosis and Classification](#)
- ▶ [DSM-III](#)
- ▶ [DSM-III-R](#)
- ▶ [DSM-IV](#)
- ▶ [Epidemiology](#)
- ▶ [Incidence](#)
- ▶ [Prevalence](#)
- ▶ [Study Design Impact on Prevalence](#)

References and Reading

- American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders (Rev. I-IV)*. Washington, DC: Author.
- American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders (Rev. I-IV)*. Washington, DC: Author.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders (Rev. I-IV)*. Washington, DC: Author.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders (Rev. I-IV)*. Washington, DC: Author.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (Rev. I-IV)*. Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (Rev. I-IV)*. Washington, DC: Author.

- Asperger, H. (1944). Die "autistischen Psychopathen" im Kindesalter. *Archiv für psychiatrie und Nervenkrankheiten*, 117, 76–136. (Reprinted in *Autism and Asperger syndrome*, by Frith, U. (Ed.). (1991). Cambridge, UK: Cambridge University Press).
- Baird, G., Simonoff, E., Pickles, A., Chandler, S., Loucas, T., Meldrum, D., et al. (2006). Prevalence of disorders of the autism spectrum in a population cohort of children in South Thames: The special needs and autism project (SNAP). *Lancet*, 368, 210–215.
- Baron-Cohen, S., Scott, F. J., Allison, C., Williams, J., Bolton, P., Matthews, F. E., et al. (2009). Prevalence of autism spectrum conditions: UK school-based population study. *The British Journal of Psychiatry*, 194, 500–509.
- Centers for Disease Control and Prevention (CDC). (2007). Prevalence of autism spectrum disorders—autism and developmental disabilities monitoring network, six sites, United States, 2000. *Morbidity and Mortality Weekly Report Surveillance Summaries*, 56 (SS-1), 1–11.
- Centers for Disease Control and Prevention (CDC). (2009). Prevalence of autism spectrum disorders – Autism and developmental disabilities monitoring network, United States, 2006. *Morbidity and Mortality Weekly Report Surveillance Summaries*, 58(SS-10), 1–20.
- Constantino, J. N. (2011). The quantitative nature of autistic social impairment. *Pediatric Research*, 69(5 Pt 2), 55R–62R.
- Creak, M., Cameron, K., Cowie, V., Ini, S., Mac Keith, R., Mitchell, G., et al. (1964). Schizophrenic syndrome in childhood: Further progress report of a working party. *Developmental Medicine and Child Neurology*, 6, 530–535.
- Eisenberg, L., & Kanner, L. (1956). Childhood schizophrenia symposium, 1955. 6. Early infantile autism, 1943–55. *American Journal of Orthopsychiatry*, 26, 556–566.
- Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. *Pediatric Research*, 65, 591–598.
- Grinker, R. R. (2007). *Unstrange minds*. New York: Basic Books. <http://www.unstrange.com>
- Happé, F. (2011). Criteria, categories, and continua: Autism and related disorders in DSM-5. *Journal of the American Academy of Child and Adolescent Psychiatry*, 50(6), 540–542.
- Heussler, H., Polnay, L., Marder, E., Standen, P., Chin, L. U., & Butler, N. (2001). Prevalence of autism in early 1970s may have been underestimated. *BMJ*, 323(7313), 633.
- Honda, H., Shimizu, Y., Imai, M., & Nitto, Y. (2005). Cumulative incidence of childhood autism: A total population study of better accuracy and precision. *Developmental Medicine & Child Neurology*, 47, 10–18.
- Kadesjo, B., Gillberg, C., & Hagberg, B. (1999). Brief report: Autism and Asperger Syndrome in seven-year-old children: A total population study. *Journal of Autism and Developmental Disorders*, 29, 327–331.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kanner, L. (1956). *The autistic child in adolescence*. Paper presented at the American Academy Psychiatric Association. Atlantic City.
- Kim, Y. S., Leventhal, B. L., Koh, Y. J., Fombonne, E., Laska, E., Lim, E. C., et al. (2011). Prevalence of autism spectrum disorders in a total population sample. *American Journal of Psychiatry*, 168, 904–912.
- Kogan, M. D., Blumberg, S. J., Schieve, L. A., Boyle, C. A., Perrin, J. M., Ghandour, R. M., et al. (2009). The prevalence of parent-reported diagnosis of autism spectrum disorder among children in the United States, 2007. *Pediatrics*. [Epub]. Accessed December 7, 2009. <http://pediatrics.aappublications.org/cgi/reprint/124/5/1395>
- Lord, C. (2011). Epidemiology: How common is autism? *Nature*, 474(7350), 166–168.
- Lotter, V. (1966). Epidemiology of autistic conditions in young children: I. Prevalence. *Social Psychiatry*, 1, 124–137.
- Lotter, V. (1967). Epidemiology of autistic conditions in young children: II. Some characteristics of the parents and children. *Social Psychiatry*, 1, 163–173.
- Mattila, M. L., Kielinen, M., Linna, S. L., Jussila, K., Ebeling, H., Bloigu, R., et al. (2011). Autism spectrum disorders according to DSM-IV-TR and comparison with DSM-5 draft criteria: An epidemiological study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 50(6), 583–592.
- Rice, C. (2007). *Prevalence of autism spectrum disorders – Autism and developmental disabilities monitoring network*. Atlanta: Centers for Disease Control.
- Ritvo, E. R., & Freeman, B. J. (1978). Current research on the syndrome of autism: Introduction. The National Society for Autistic Children's definition of the syndrome of autism. *Journal of the American Academy of Child Psychiatry*, 17(4), 565–575.
- Roelfsema MT, Hoekstra RA, Allison C, Wheelwright S, Brayne C, Matthews FE, Baron-Cohen S. (2012). Are autism spectrum conditions more prevalent in an information-technology region? *The Journal of Autism and Developmental Disorders*, 42(5), 734–739. <http://www.ncbi.nlm.nih.gov/pubmed/21681590>
- Rutter, M. (1978). Diagnosis and definition. In M. Rutter & E. Schopler (Eds.), *Autism. A reappraisal of concepts and treatment*. New York: Plenum Press.
- Rutter, M. (2005). Incidence of autism spectrum disorders: Changes over time and their meaning. *Acta Paediatrica*, 94, 2–15.
- Rutter, M., & Schopler, E. (1987). Autism and pervasive developmental disorders: Concepts and diagnostic issues. *The Journal of Autism and Developmental Disorders*, 17(2), 159–186. <http://www.ncbi.nlm.nih.gov/pubmed/3610994>
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11, 115–129.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children:

Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9(1), 11–29.

World Health Organization. (1992). *ICD-10 Classifications of mental and behavioral disorder: Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organization.

Time-Out

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Synonyms

[Time-out from positive reinforcement](#)

Definition

Time-out is a procedure that involves removing an individual from positive reinforcement. It is typically employed as a punishment technique. Often, it involves removing an individual from a particular setting in which they displayed inappropriate behaviors and placing the individual in a non-reinforcing setting, such as a chair in the corner, their bed/crib, and outside the classroom. Time-out also may include removal of items or materials that are reinforcing. The term “time-out” was first coined by Arthur Staats in the late 1950s as an effective procedure he used with his typically developing children to decrease their disruptive behaviors, such as crying (Staats 1971). Montrose

Wolf and others (e.g., Allen et al. 1964) used the procedure more systematically as a behavior management technique in which individuals are removed from positive reinforcement. While this technique is generally effective with typically developing children, it may serve as a (negative) reinforcer for children with autism, who may find some of the demands of social interaction aversive and time alone rewarding (Solnick et al. 1977). Specifically, social isolation may not be aversive to a child with autism; thus, time-out may be rewarding to the child. Time-out is often used in place of physical punishment but is most effective when used with other procedures, such as extinction, teaching appropriate replacement behaviors, and differential reinforcement of appropriate behaviors.

See Also

- [Negative Reinforcement](#)
- [Punishment](#)
- [Type II Punishment, Second Edition](#)

References and Reading

- Allen, K. E., Hart, B., Buell, J. S., Harris, F. R., & Wolf, M. (1964). Effects of social reinforcement on isolate behavior of a nursery school child. *Child Development*, 35(2), 511–518.
- Risley, T. R. (1968). The effects and side effects of punishing the autistic behaviors of a deviant child. *Journal of Applied Behavior Analysis*, 1(1), 21–34.
- Solnick, J. V., Rincover, A., & Peterson, C. R. (1977). Some determinants of the reinforcing and punishing effects of timeout. *Journal of Applied Behavior Analysis*, 10(3), 415–424.
- Staats, A. W. (1971). *Child learning, intelligence, and personality: Principles of a behavioral interaction approach*. New York: Harper & Row.

Time-Out from Positive Reinforcement

- [Time-Out](#)

Timothy Syndrome

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Synonyms

Heart-hand syndrome; Timothy Syndrome – 1 (TS1); Timothy Syndrome – 2 (TS2)

Short Description or Definition

Timothy syndrome (TiS) is a rare multisystem developmental disorder caused by a single de novo mutation in the $Ca_v1.2$ L-type calcium channel gene *CACNA1C*. TiS is characterized by physical malformations, cardiac defects, and autism. Individuals with TiS exhibit dysmorphic facial features such as rounded faces, flat nasal bridges, baldness, and abnormal dental anatomy. Patients also possess webbing of toes and fingers (syndactyly). Cardiac deficits of TiS include a severe prolongation of the QT interval (electrocardiographic indication of prolonged heartbeat), significant arrhythmia, and congenital heart disease. Developmental delays associated with TiS include language, motor, and cognitive impairments. Language is affected in the domains of articulation, reception, and expression. Cognitive features include impairment in communication, socialization, daily living skills, as well as intellectual disability. Individuals with TiS meet the criteria for autism spectrum disorder.

Categorization

Phenotypic concurrence of cardiac arrhythmia, long QT, and syndactyly (webbing of fingers and toes) are indicative of TiS (Marks et al. 1995);

DNA sequence analysis is used to confirm TiS (Frohler et al. 2014). Depending on the point mutation in either the mutually exclusive exon 8 or exon 8A, TiS diagnoses are categorized into two subtypes: TS1 (G406R in exon 8A) and TS2 (G406R or G402S in exon 8). The exon 8A-containing variant of $Ca_v1.2$ mRNA is primarily expressed during development and at lower levels (~20%) than the dominant exon 8 (Panagiotakos et al. 2019). A glycine to arginine substitution at residue 406 (G406R) in the alternative splicing variant of exon 8A of the *CACNA1C* gene encoding $Ca_v1.2$ calcium channel confirms typical Timothy Syndrome variant 1 (TS1) (Splawski et al. 2004). The atypical Timothy Syndrome variant 2 (TS2), a more severe form of TiS, results from the G406R mutation occurring in the more highly and persistently expressed exon 8 (Panagiotakos et al. 2019). Patients with atypical TiS do not present with syndactyly (Splawski et al. 2005), a particular phenotypic distinction from TS1. Additionally, TS2 can arise from two mutations of $Ca_v1.2$, in exon 8: G406R and G402S. The G406R mutation in atypical TS2 leads to many of the same cardiac and neurological phenotypes as classic TS1, including diagnosable ASD-associated behaviors. Patients with atypical TS2 from a G402S mutation also possess significant cardiac symptoms, including QT interval elongation as well as some neurological symptoms. Though TS2 patients with the G402S mutation possess degrees of intellectual disability and some developmental delay (Diep and Seaver 2015; Hiippala et al. 2015; Splawski et al. 2005), these patients do not present with cognitive deficits associated specifically with Autism Spectrum Disorder (ASD), an important distinction from the G406R variant of TS2 (Hiippala et al. 2015; Splawski et al. 2005). There have also been singular cases of patients presenting with TiS who exhibited mutations in *CACNA1C* gene but not within exon 8/8A (Boczek et al. 2015; Landstrom et al. 2016).

Epidemiology

TiS results from de novo mutations and is extremely rare (Splawski et al. 2004). Inheritance patterns of TiS are sporadic; parents of affected

children typically do not have TiS (Marks et al. 1995; Splawski et al. 2004). However, genetic mosaicism, in which a two or more cell populations in an individual express different genotypes, can lead to inheritance of the mutation in offspring (Gao et al. 2013; Splawski et al. 2004, 2005).

Natural History, Prognostic Factors, and Outcomes

Timothy Syndrome was initially identified in the early 1990s as a novel cardiac arrhythmia that presented with webbing of fingers and toes (syndactyly) and described as a unique form of heart-hand syndrome (HHS). TiS was first identified in Germany by Dr. Reichenbach who recorded a significantly elongated QT-interval and syndactyly in a new born. Long QT-interval and synostoses of the carpus in the father suggested a significant genetic component to this syndrome (Reichenbach et al. 1992). Shortly after, Dr. Mark Keating reported several case studies of infants possessing the same phenotypic association of cardiac arrhythmia and syndactyly. The three children were hypothesized to have the same novel form of long QT-syndrome and were at high risk of sudden death. The patients also possessed other congenital cardiac malformations, but only long QT-interval and syndactyly were consistent across individuals. Dr. Keating and colleagues hypothesized a genetic mechanism for this disease (Marks et al. 1995). The syndrome was named Timothy Syndrome after Katherine Timothy, a nurse at the University of Utah who originally identified this phenotype across different infants. As medical treatment of TiS improved and extended the lives of patients, it became clear that TiS was a major multisystem disorder affecting the heart, skin, eyes, teeth, immune system, and brain (Splawski et al. 2004).

Cardiac abnormalities in TiS are severe and are associated with sudden death (Marks et al. 1995). All children with TiS possess elongated QT intervals and arrhythmias, with 70% of patients experiencing a life-threatening arrhythmic episode. Arrhythmia in TiS can occur as ventricular tachycardia or bradycardia from atrioventricular block (AV block). TiS mutations also result in

hypertrophic cardiomyopathy and ventricular systolic dysfunction (Lo et al. 2005). Patients with TiS also possess congenital heart disease including patent ductus arteriosus (60%), patent foramen ovale (30%), ventricular septal defects (20%), tetralogy of Fallot (6%), and cardiomegaly (35%). Because of the severe cardiac arrhythmia in TiS, many children with TiS die early in infancy and childhood. The average age of death in children with TiS is 2.5–3 years (Splawski et al. 2004).

Children with TiS experience a number of medical difficulties in addition to heart abnormalities. Skin and facial abnormalities are the early phenotypic features of TiS. All children with TiS have syndactyly of the fingers and occasionally toes, as well as baldness at birth. Many children with TiS will have other facial and skin features. Identifiable facial dysmorphism includes rounded faces, flat nasal bridges, thin upper lips, and receding upper jaws. Dentition development of children with TiS is abnormal, characterized by small teeth with soft, poorly formed enamel (Papineau and Wilson 2014; Splawski et al. 2004). Other facial difficulties in TiS patients include nearsightedness (25%) and sinusitis (30%). Children with TiS are likely to present with pulmonary complications such as pneumonia, bronchitis, and pulmonary hypertension, as well as gastrointestinal difficulties. Episodic hypocalcemia and hypoglycemia are present in about 33% of patients. Surviving children exhibit significant development delays in language and motor skills, along with various general cognitive impairments. Nearly 60–80% of surviving patients with the G406R TiS mutation meet the criteria for autism spectrum disorder while patients with the G402S mutation exhibit intellectual disability and developmental delay without autism (see section “[Evaluation and Differential Diagnosis](#)”). Seizures are experienced by 21% of patients with TiS.

Clinical Expression and Pathophysiology of Timothy Syndrome

Intellectual disability occurs in about 25% of individuals with TiS, though a number of other

cognitive impairments are also present. All adaptive functioning is impaired in TiS. Communication is significantly affected and language delay manifests early in development. Many children with TiS do not babble during infancy. Other language skills that are affected in TiS include articulation, reception, and expression. Other areas of adaptive functioning such as socialization, personal care, and daily living skills are impaired. The association between TiS and ASD is very high. Between 60% and 80% of patients with either TS1 or TS2 G406R mutations that survive the significant cardiac defects are diagnosed with ASD (Splawski et al. 2004). TiS is a syndromic autism spectrum disorder, an ASD with known genetic etiology that occurs in conjunction other specific phenotypes (Sztainberg and Zoghbi 2016).

TiS has attracted research interest because it arises from a known single amino acid mutation in a prominent signaling protein ($\text{Ca}_v1.2$) that generates ASD with high likelihood. Consistent with other lines of evidence for an important role of calcium channels in ASD (Lu et al. 2012), TiS provides a unique opportunity for pinpointing and dissecting mechanisms potentially involved in ASD. Building on the known genetics associated with TiS, investigations of altered neurological mechanisms have yielded valuable knowledge regarding activity-dependent neuronal signaling. Studies of TiS-associated mutations in animal models and induced pluripotent stem cells (iPSCs) have revealed important details about altered pathophysiological mechanisms underlying the disease (Bader et al. 2011; Bett et al. 2012; Krey et al. 2013). Both the G406R and G402S mutations result in excess calcium influx via the $\text{Ca}_v1.2$ channel upon neuronal depolarization (Splawski et al. 2005). This excess calcium influx is the result of altered channel inactivation (Barrett and Tsien 2008). Both mutations results in alterations in voltage-dependent activation. The G406R mutation exhibits a potentiating leftward 10 mV shift in the voltage-dependent activation of $\text{Ca}_v1.2$ (Splawski et al. 2005) while the G402S mutation exhibits a rightward shift. Additionally, the G406R mutation has be shown to exhibit

abnormal voltage-dependent conformational signaling (Li et al. 2016). Neurons expressing the G406R TiS mutation show excessive activation of activity-dependent genes specifically after increases in neuronal activity (Li et al. 2016). Both the G402S and G406R mutation confers defects in calcium-dependent inactivation and contributes to arrhythmogenesis (Dick et al. 2016). Furthermore, developmental studies have identified excessive activity-dependent retraction of dendrites (Krey et al. 2013) both of which are not associated with the increased calcium influx of $\text{Ca}_v1.2$ with a TiS mutation (Krey et al. 2013; Li et al. 2016), as well as aberrant migration and neurite formation during development in TiS animal models (Kamijo et al. 2018) and cultured human brain organoids (Birey et al. 2017). Genetic investigations in both human and animal models of TiS have revealed effects of the TS1 mutation on the alternative mRNA splicing of exon 8 and exon 8A (Panagiotakos et al. 2019). In addition to alterations in the electrophysiological functions of $\text{Ca}_v1.2$, the TS1 mutation confers altered exon 8A expression that results in continuous post-developmental expression of the pathological channel (Panagiotakos et al. 2019). Glial development and function are also affected by the TiS mutation. Myelinating oligodendrocytes possess increased calcium influx, mature at faster rates, and exhibit increased myelination during postnatal development. Animal models of TiS display readily discernable ASD-associated behaviors such as reduced socialization and increases in repetitive and perseverative behavior. The findings from these behavioral and mechanistic studies have important implications for broader questions about the underlying pathophysiological mechanisms in ASD (Bader et al. 2011; Mullins et al. 2016; Sztainberg and Zoghbi 2016).

Evaluation and Differential Diagnosis

Though TiS is characterized by many neurological and developmental features, it is typically suggested by the presence of cardiac QT

elongation accompanied by syndactyly and other facial deformities (Marks et al. 1995). Diagnosis can be suspect in a child before birth if fetal heart rates are found to be bradycardic. After birth, children with TiS can develop sudden hypoxia and present with cyanosis, a bluish discoloration of the skin. Subsequent cardiac evaluation of infants with TiS reveals 2:1 AV block as well as prolonged QT interval. However, there are forms of TiS where patients do not exhibit syndactyly. In order to properly diagnose TiS, single-strand conformational polymorphism sequence analysis or DNA sequencing of the CACNA1C gene is utilized (Splawski et al. 2004). It is important to identify TiS-associated mutations in the various exons of CACNA1C, as many of them are exclusively alternatively spliced (Tang et al. 2011). These mutations are gain-of-function mutations in the $\text{Ca}_v1.2$ L-type calcium channel. They are both genetically and phenotypically distinct from other heart-hand syndromes (HHS), such as Holt-Oram Syndrome (HOS). While patients with HOS generally present with congenital heart defects and upper-limb anomalies, they do not exhibit the QT interval elongation or cognitive impairments seen in TiS (Basson et al. 1994).

Treatment

TiS is a complex disorder that significantly affects multiple systems of the body. Its diverse phenotypic expression in various domains presents difficulties in targeting intervention and treatments for the many cardiac, neurological, immunologic, and other symptoms. Primary attention of treatment and intervention is placed on mitigating severity of life-threatening cardiac arrhythmias and associated heart defects. Despite the syndrome's rare nature, information regarding medical background and intervention is readily available from National Organization for Rare Disorders (NORD) and the Sudden Arrhythmia Death Syndromes Foundation (SADS).

The leading cause of death in patients with TiS is ventricular tachycardia and fibrillation. Because of its rarity, no significant data is

available regarding the effectiveness of numerous cardiac treatments. Beta-blockers have been suggested as a standard treatment for children with TiS. Whether beta-blockers are effective forms of treatment remains in question. Additional novel cardiac treatments such as sodium channel blockers have been proposed, with case studies describing their effectiveness (Gao et al. 2013). However, broad and long-term efficacy of these treatments remain in question. As the TiS-associated mutation occurs in L-type calcium channel, treatment with L-type calcium channel blockers have also been suggested. Studies in animal models have shown potential but have not been proven to be effective in humans with TiS. Other cardiac-specific interventions include the use of pacemakers and defibrillators concurrent with drug treatments for patients with TiS. However, immune systems of children with TiS are significantly affected. Consequently, any intervention utilizing internally placed devices poses a risk for infection. Additionally, anesthesia is known to trigger arrhythmias in TiS. Thus, any surgical intervention required to repair congenital cardiac defects will require extreme care by treating physicians and caretakers.

While the average age of death in TiS is 2.5–3 yrs., children have been known to survive into their teenage years. Neuropsychiatric evaluations of individuals with TiS reveals significant speech and developmental delays, intellectual disability, and autism spectrum disorder. Delays in development can be alleviated through a broad and integrated therapies such as social integration, speech, and physical therapies. Early intervention is key to show marked benefit in treating the numerous neuropsychiatric and cognitive symptoms of children with TiS.

See Also

- [Animal Models](#)
- [Genetic Disorders](#)
- [Plasticity, Neural](#)
- [Single-Nucleotide Polymorphism](#)
- [Synaptic Proteins](#)

References and Reading

- Bader, P. L., Faizi, M., Kim, L. H., Owen, S. F., Tadross, M. R., Alfa, R. W., Bett, G. C. L., Tsien, R. W., Rasmusson, R. L., & Shamloo, M. (2011). Mouse model of Timothy syndrome recapitulates triad of autistic traits. *Proceedings of the National Academy of Sciences of the United States of America*, 108, 15432–15437.
- Barrett, C. F., & Tsien, R. W. (2008). The Timothy syndrome mutation differentially affects voltage- and calcium-dependent inactivation of CaV1.2 L-type calcium channels. *Proceedings of the National Academy of Sciences of the United States of America*, 105, 2157–2162.
- Basson, C. T., Cowley, G. S., Solomon, S. D., Weissman, B., Poznanski, A. K., Traill, T. A., Seidman, J. G., & Seidman, C. E. (1994). The clinical and genetic spectrum of the Holt-Oram syndrome (heart-hand syndrome). *The New England Journal of Medicine*, 330, 885–891.
- Bett, G. C., Lis, A., Wersinger, S. R., Baizer, J. S., Duffey, M. E., & Rasmusson, R. L. (2012). A mouse model of timothy syndrome: A complex autistic disorder resulting from a point mutation in Cav1.2. *North American Journal of Medicine & Science (Boston)*, 5, 135–140.
- Birey, F., Andersen, J., Makinson, C. D., Islam, S., Wei, W., Huber, N., Fan, H. C., Metzler, K. R. C., Panagiotakos, G., Thom, N., et al. (2017). Assembly of functionally integrated human forebrain spheroids. *Nature*, 545, 54–59.
- Boczek, N. J., Miller, E. M., Ye, D., Nesterenko, V. V., Tester, D. J., Antzelevitch, C., Ciosek, R. J., Ackerman, M. J., & Ware, S. M. (2015). Novel Timothy syndrome mutation leading to increase in CACNA1C window current. *Heart Rhythm*, 12, 211–219.
- Dick, I. E., Joshi-Mukherjee, R., Yang, W., & Yue, D. T. (2016). Arrhythmogenesis in Timothy Syndrome is associated with defects in Ca(2+)-dependent inactivation. *Nature Communications*, 7, 10370.
- Diep, V., & Seaver, L. H. (2015). Long QT syndrome with craniofacial, digital, and neurologic features: Is it useful to distinguish between Timothy syndrome types 1 and 2? *American Journal of Medical Genetics Part A*, 167A, 2780–2785.
- Frohler, S., Kieslich, M., Langnick, C., Feldkamp, M., Opgen-Rhein, B., Berger, F., Will, J. C., & Chen, W. (2014). Exome sequencing helped the fine diagnosis of two siblings afflicted with atypical Timothy syndrome (TS2). *BMC Medical Genetics*, 15, 48.
- Gao, Y., Xue, X., Hu, D., Liu, W., Yuan, Y., Sun, H., Li, L., Timothy, K. W., Zhang, L., Li, C., et al. (2013). Inhibition of late sodium current by mexiletine: A novel pharmacotherapeutic approach in timothy syndrome. *Circulation. Arrhythmia and Electrophysiology*, 6, 614–622.
- Hiippala, A., Tallila, J., Myllykangas, S., Koskenvuo, J. W., & Alastalo, T. P. (2015). Expanding the phenotype of Timothy syndrome type 2: An adolescent with ventricular fibrillation but normal development. *American Journal of Medical Genetics Part A*, 167A, 629–634.
- Kamijo, S., Ishii, Y., Horigane, S. I., Suzuki, K., Ohkura, M., Nakai, J., Fujii, H., Takemoto-Kimura, S., & Bito, H. (2018). A critical neurodevelopmental role for L-type voltage-gated calcium channels in neurite extension and radial migration. *The Journal of Neuroscience*, 38, 5551–5566.
- Krey, J., Pasca, S., Shecheglovitov, A., Yazawa, M., Schwemberger, R., Rasmusson, R., & Dolmetsch, R. (2013). Timothy syndrome is associated with activity-dependent dendritic retraction in rodent and human neurons. *Nature Neuroscience*, 16, 201–212.
- Landstrom, A. P., Boczek, N. J., Ye, D., Miyake, C. Y., De la Uz, C. M., Allen, H. D., Ackerman, M. J., & Kim, J. J. (2016). Novel long QT syndrome-associated missense mutation, L762F, in CACNA1C-encoded L-type calcium channel imparts a slower inactivation tau and increased sustained and window current. *International Journal of Cardiology*, 220, 290–298.
- Li, B., Tadross, M. R., & Tsien, R. W. (2016). Sequential ionic and conformational signaling by calcium channels drives neuronal gene expression. *Science*, 351, 863–867.
- Lo, A. N. S. M., Wilde, A. A., van Erven, L., & Blom, N. A. (2005). Syndactyly and long QT syndrome (CaV1.2 missense mutation G406R) is associated with hypertrophic cardiomyopathy. *Heart Rhythm*, 2, 1365–1368.
- Lu, A. T., Dai, X., Martinez-Agosto, J. A., & Cantor, R. M. (2012). Support for calcium channel gene defects in autism spectrum disorders. *Molecular Autism*, 3, 18.
- Marks, M. L., Whisler, S. L., Clericuzio, C., & Keating, M. (1995). A new form of long QT syndrome associated with syndactyly. *Journal of the American College of Cardiology*, 25, 59–64.
- Mullins, C., Fishell, G., & Tsien, R. W. (2016). Unifying views of autism spectrum disorders: A consideration of autoregulatory feedback loops. *Neuron*, 89, 1131–1156.
- Panagiotakos, G., Haveles, C., Arjun, A., Petrova, R., Rana, A., Portmann, T., Pasca, S. P., Palmer, T. D., & Dolmetsch, R. E. (2019). Aberrant calcium channel splicing drives defects in cortical differentiation in Timothy Syndrome. *eLife*, 8, e51037.
- Papineau, S. D., & Wilson, S. (2014). Dentition abnormalities in a Timothy syndrome patient with a novel genetic mutation: A case report. *Pediatric Dentistry*, 36, 245–249.
- Reichenbach, H., Meister, E. M., & Theile, H. (1992). The heart-hand syndrome. A new variant of disorders of heart conduction and syndactyly including osseous changes in hands and feet. *Kinderärztliche Praxis*, 60, 54–56.

- Splawski, I., Timothy, K. W., Sharpe, L. M., Decher, N., Kumar, P., Bloise, R., Napolitano, C., Schwartz, P. J., Joseph, R. M., Condouris, K., et al. (2004). Ca(V)1.2 calcium channel dysfunction causes a multisystem disorder including arrhythmia and autism. *Cell*, 119, 19–31.
- Splawski, I., Timothy, K. W., Decher, N., Kumar, P., Sachse, F. B., Beggs, A. H., Sanguinetti, M. C., & Keating, M. T. (2005). Severe arrhythmia disorder caused by cardiac L-type calcium channel mutations. *Proceedings of the National Academy of Sciences of the United States of America*, 102, 8089–8096. discussion 8086–8088.
- Sztainberg, Y., & Zoghbi, H. Y. (2016). Lessons learned from studying syndromic autism spectrum disorders. *Nature Neuroscience*, 19, 1408–1417.
- Tang, Z. Z., Sharma, S., Zheng, S., Chawla, G., Nikolic, J., & Black, D. L. (2011). Regulation of the mutually exclusive exons 8a and 8 in the CaV1.2 calcium channel transcript by polypyrimidine tract-binding protein. *The Journal of Biological Chemistry*, 286, 10007–10016.

Timothy Syndrome – 1 (TS1)

► [Timothy Syndrome](#)

Timothy Syndrome – 2 (TS2)

► [Timothy Syndrome](#)

Title IX

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Definition

Title IX is a federal civil rights law that prohibits any college or university that receives federal funding from discriminating against an individual upon the basis of their sex. This includes female, male, and

gender-nonconforming individuals. Recently it has been used to address sexual discrimination in a variety of forms including sexual harassment that involves both sexual violence and other forms of harassment not involving violence. Sexual violence includes but is not limited to sexual assault, rape, sexual battery, and sexual coercion. The goal of this legislation is to prevent these forms of violence from seriously limiting or even denying a student’s ability to participate or benefit from the school’s educational programs and activities. The law covers behaviors that occur both on and off campus.

According to the US Department of Education, the Office of Civil Rights (OCR) is responsible for the enforcement of Title IX as well as many other laws. The scope of Title IX according to OCR is the following:

Title IX applies to institutions that receive federal financial assistance from ED, including state and local educational agencies. These agencies include approximately 16,500 local school districts, 7,000 postsecondary institutions, as well as charter schools, for-profit schools, libraries, and museums. Also included are vocational rehabilitation agencies and education agencies of 50 states, the District of Columbia, and territories and possessions of the United States.

Educational programs and activities that receive ED funds must operate in a nondiscriminatory manner. Some key issue areas in which recipients have Title IX obligations are: recruitment, admissions, and counseling; financial assistance; athletics; sex-based harassment; treatment of pregnant and parenting students; discipline; single-sex education; and employment. Also, a recipient may not retaliate against any person for opposing an unlawful educational practice or policy, or made charges, testified or participated in any complaint action under Title IX. For a recipient to retaliate in any way is considered a violation of Title IX. The ED Title IX regulations (volume 34, Code of Federal Regulations, Part 106) provide additional information about the forms of discrimination prohibited by Title IX. (OCR [2015](#))

Historical Background

On June 23, 1972, President Richard Nixon signed Title IX of the Educational Amendments into law (U.S. Department of Justice [2015](#)). Initially Title IX was used in the 1970s to leverage equal opportunity for female athletes in university

sports programs (NCAA 2017). It was also used to protect pregnant students from being denied educational opportunities in both secondary and postsecondary institutions. This act amended the Higher Education Act of 1965, the Vocational Education Act of 1963, the General Education Provisions Act, and the Elementary and Secondary Education Act of 1965. The nickname of the Act is the Educational Amendments Act of 1972, rather than the longer title of all of the various acts it amended.

Current Knowledge

Recent media attention has highlighted the problem of sexual assault on college campuses. According to the National Sexual Violence Resource Center (2016), 1 in 5 women and 1 in 71 men will be raped in their lifetimes. College-aged students in general are at a much higher risk of sexual assault than other facets of the population. In fact, 11.2% of all students experience rape or sexual assault through physical force, violence, or incapacitation (among all graduate and undergraduate students) (RAINN 2016). According to RAINN, among undergraduate students, 23.1% of females and 5.4% of males experience rape or sexual assault through physical force, violence, or incapacitation (2016). A survey from the Association of American Universities (2015) confirmed the data with a finding that 23% of female college students experienced unwanted sexual contact. Well over 1/5 of transgender, gender-queer, and gender-nonconforming individuals have been sexually assaulted (RAINN 2016) which was also found to be the case in the AAU survey. The last finding is particularly important given the fact that recent research in the area of autism has found a higher incidence of gender nonconformity among ASD students. In a study conducted in the Netherlands, de Vries and her team (2010) found an incidence of 7.8% of the children, and adolescents in their sample had gender identity disorder. This prevalence is ten times greater than incidence that is found in the non-ASD population. Strang et al. (2014) found that participants with ASD in their study were 7.59

times more likely to express gender variance. There are approximately a ½ dozen studies that link autism and the expression of gender variance.

Clearly, students with ASDs, who have difficulty reading social situations and have difficulty self-advocating, are at higher risk of sexual assault than their nondisabled peers. They may be pressured into sexual behaviors or into drinking alcohol or recreational drug use. An individual cannot give consent to sexual activity if he or she is incapacitated due to drugs or alcohol. “A majority of all the victims of any nonconsensual sexual contact reported using alcohol or drugs prior to the incident: ranging from 61.7 percent to 93.8 percent,” in the 127 universities surveyed (Fisher et al. 2016, p. 39). “Victims of any nonconsensual sexual contact perceived that the offender was under the influence of alcohol or drugs prior to the incident (ranging from 66.7% to 83.8% across types of nonconsensual sexual contact)” (Fisher et al. 2016, p. 21).

Future Directions

Students on the autism spectrum need to understand that they must give affirmative consent for each and every sexual activity they are to engage in. This means they must agree to the sexual contact voluntarily and that the agreement is mutual. The agreement can be verbal or nonverbal. The nonverbal agreement is where students on the autism spectrum have the most difficulty. They may misunderstand or misread nonverbal cues. The consent should be given in a manner that both parties can understand. Consent must be given for each act and upon each occasion. What was okay between two parties on 1 day may not be okay upon another day. What can be even more confusing is that consent can be withdrawn at any time. Consequently, the sexual activity must stop immediately. The consent also must be given without any force, coercion, or intimidation. Silence is not consent. A lack of resistance, likewise, is not consent either. Future research should be aimed at how best to teach students on the spectrum of these complex social concepts.

In an effort to fit in, some students on the autism spectrum may be duped into saying or doing something to a fellow student that could be construed as sexual harassment. Nondisabled peers may goad a student on the spectrum into saying something about another student's anatomy, making a sexually suggestive comment or joke, or even kissing or groping another student. Students on the autism spectrum should read the university's student code of conduct in reference to the definitions of sexual harassment.

Most often male students on the spectrum may develop a romantic interest in a female student and not know how to express the interest in an appropriate manner. Here, the student on the autism spectrum should again consult the student code of conduct. Many times, these students are in violation of the student code of conduct in relation to the rules against stalking. These students find themselves facing disciplinary proceedings and not understanding why this is happening. High school transition programs should include social skills training in the areas of assertiveness, sexual harassment, and appropriate platonic and romantic relationships.

If you are a student on the autism spectrum or if you care for an individual on the spectrum and that person is a victim of sex-based discrimination, know that there are professionals at colleges and universities nationwide that are dedicated to helping students with this difficult situation. Look for your college's Title IX coordinator's name and contact information. He or she has a number of resources at her or his disposal to help you including counseling and health referrals, as well as support in filing reports and helping to insure your safety from further harm. It is imperative that a student receive help and support from family, friends, and health-care providers during this most difficult period. Future research needs to be aimed at preventing sexual assault among the ASD population attending college.

If you are a student on the autism spectrum who is accused of violating your college's code of conduct, know that the Title IX coordinator's job is to help support you through the process as well. He or she will inform you as to what exactly is going to happen, i.e., what the process will be,

what your rights are, what prohibitions you might face during the process, and finally what are the possible outcomes of the process. Tell the Title IX person the exact nature of your disability and what reasonable accommodations you might need, including designating a support person or advisor to you – who can help guide you through the process. Find out whether the college follows an investigator model or a judiciary or hearing model. An investigatory model may be easier for students on the spectrum to negotiate because it involves less people than a hearing panel model. Read your college's Title IX web page as well as the student code of conduct. Finally, future research should investigate how best to support victims of sexual assault who are on the autism spectrum, as well as individuals on the autism spectrum who are accused of stalking, sexual harassment, and sexual assault. A focus of this research should be what reasonable accommodations under the Americans with Disabilities Act would best help students with ASDs in these situations.

See Also

► [Americans with Disabilities Act](#)

References and Reading

- Association of American Universities. (2015). *AAU climate survey on sexual assault and sexual misconduct*. <http://www.aau.edu/Climate-Survey.aspx?id=16525>.
- de Vries, A. L. C., Noens, I. L. J., Cohen-Kettenis, P. T., van Berckelaer-Onnes, I. A., & Doreleijers, T. A. (2010). Autism spectrum disorders in gender dysphoric children and adolescents. *Journal of Autism and Developmental Disorders*, 40(8), 930–936. <https://doi.org/10.1007/s10803-010-0935-9>.
- Fisher, B. S., Peterson, S., Cantor, D., Townsend, R., & Sun, H. (2016). *Characteristics of nonconsensual sexual contact incidents: Penetration and sexual touching by force or while incapacitated*. Westat, Rockville, MD.
- National Sexual Violence Resource Center. (2016). Statistics about sexual violence. Retrieved from http://www.nsvrc.org/sites/default/files/publications_nsvrc_factsheet_media-packet_statistics-about-sexual-violence_0.pdf.
- National College Athletics Association (NCAA). (2017). Title IX Frequently Asked Questions | NCAA.org –

- The official site of Retrieved from www.ncaa.org/about/resources/inclusion/title-ix-frequently-asked-questions. 3 Sept 2017.
- RAINN. (2016). Campus sexual violence statistics. Retrieved from <https://www.rainn.org/statistics/campus-sexual-violence>.
- Strang, J. F., Kenworthy, L., Dominska, A., Sokoloff, J., Kenealy, L. E., Berl, M., Walsh, K., Menvielle, E., Slesaransky-Poe, G., Kim, K. E., Luong-Tran, C., Meagher, H., & Wallace, G. L. (2014). Increased gender variance in autism spectrum disorders and attention deficit hyperactivity disorder. *Archives of Sexual Behavior*, 43, 1525–1533. <https://doi.org/10.1007/s10508-0140285-3>.
- U.S. Department of Education. (2015). *Title IX and sex discrimination*. Retrieved from https://www2.ed.gov/about/offices/list/ocr/docs/tix_dis.html. 3 Sept 2017.
- U.S. Department of Justice. (2015). Overview of Title IX of the Education Amendments of 1972, 20 U.S.C. Retrieved from <https://www.justice.gov/ct/overview-title-ix-education-amendments-1972-20-usc-1681-et-seq>. 3 Sept 2017.

the 0–3 age group. Assessments for infants often include tests of neonatal reflexes and developmental motor milestones expected in the first year of life. Motor assessment for toddlers usually includes emerging use of writing and eating utensils, object manipulation, and play skills. This entry focuses specifically on the T.I.M.E. standardized assessment.

This test was designed to provide a comprehensive assessment of motor abilities, in conjunction with occupational and functional performance in children 4 months to 3½ years of age. It takes 10–20 min to administer to younger children and 20–40 min to administer to older children. The five primary subtests of the T.I.M.E. are mobility, stability, motor organization, social/emotional abilities, and functional performance.

TNL 2

► Test of Narrative Language

Toddler Infant Motor Evaluation

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Synonyms

TIME

Description

Toddler infant motor evaluation can be thought of as a generic term for assessment of various aspects of motor development and skills in babies and young children, although there is also a specific assessment known as the Toddler and Infant Motor Evaluation (T.I.M.E.). There are many developmental tests that include motor scales for

Historical Background

The T.I.M.E. was developed by Lucy Jane Miller, Ph.D., OTR, and Gale Roid, Ph.D., in 1994. It was designed to be a comprehensive assessment of motor development and quality of motor functions.

Psychometric Data

Standardization

Test items for the T.I.M.E. were developed following item generation and testing with a variety of ages and groups. The final test is standardized on a sample of 875 children (731 typically developing and the remainder children with motor delays or concerns). The normative sample is stratified based on the 1990 US census. The sample was divided into 10 age groups (0–12 months divided in 3-month intervals, 13–24 months in 4-month intervals, and 25–48 months in 6-month intervals).

Reliability

Internal consistency ranges from .72 to .97.

Test-retest – in a sample of 33 children (9% with motor delays) with a retest of 1–3 weeks, the test-retest correlation coefficient ranged from .965 to .998.

Interrater reliability – in a sample of 31 children tested by two independent examiners, the correlation coefficient ranged from .897 to .996.

Validity

Content validity was initially developed through expert analysis. Construct validity was established through age trends, factor analysis, and studies of dimensionality. Discriminant validity and classification accuracy were determined through a series of analyses, which demonstrated false positives between 1.1% and 11.8%, false negatives between 1.0% and 3.2%, specificity between 85.9% and 98.6%, and sensitivity between 80.6% and 97.2%.

Clinical Uses

The T.I.M.E. is conceptualized as a diagnostic assessment designed for use by a licensed or highly trained physical or occupational therapist, adaptive physical education teacher, or others with specialized training in the motor domain.

References and Reading

- Mahoney, G., Robinson, C., & Perales, F. (2004). Early motor intervention: The need for a new treatment paradigm. *Infants and Young Children*, 17(4), 291–300.
- Miller, L. J., & Roid, G. H. (1993). Sequence comparison methodology for the analysis of movement patterns in infants and toddlers with and without motor delays. *American Journal of Occupational Therapy*, 47(4), 339–347.
- Miller, L. J., & Roid, G. H. (1994). *The T.I.M.E.: Toddler and infant motor evaluation*. San Antonio: The Psychological Corporation.

Definition

Humans exhibit plantigrade walking: They walk on the full soles (bottoms) of their feet. Toe walking involves walking on the balls (distal metatarsals/metatarsal heads) of the feet with the heel (calcaneus) raised up off the ground. Typically developing children will intermittently walk on their toes for a short while after first beginning ambulation. When the equinus gait is noticeably present after 3 months of walking, it is referred to as persistent toe walking. In the past, this was considered a sign of possible spastic cerebral palsy, but many children who toe walk do not exhibit any other signs of neuromotor disorder (hypertonicity, hyperreflexia, and upgoing Babinski reflexes). Orthopedics sometimes attributed this gait to a shortened tendo Achilles (short heel cord) even in the absence of a shortened ligament. Children with a neuromotor problem such as spastic diplegia will toe walk with their knees flexed, while children with idiopathic toe walking will toe walk with their knees extended. Since toe walking may be inhibited by certain types of shoes, its presence should be assessed with shoes off. Persistent toe walking has been noted to be more common in children across the entire spectrum of neurodevelopmental disorders apart from any motor diagnosis, and to be even more common in children with their language skills more severely impaired than the other developmental streams. Among children with language disorders, children with autism exhibit the highest persistence of toe walking, with close to 50% of 5-year-old children with autism continuing to exhibit at least an intermittent equinus gait (Accardo and Whitman 1989; Accardo et al. 1992; Shulman et al. 1997). The persistence of toe walking in children with autism may contribute to secondary motor deformity by producing a genuine shortening of the tendo Achilles. Treatments for this secondary deformity can include stretching exercises, casting or braces, Botox injections, and, in rare cases, heel cord lengthening surgery. Toe walking seems to be more correlated with the language component of autism, since it is not appreciably increased in children with Asperger Syndrome where language is less affected (Barrow et al. 2011). The etiology behind the

Toe Walking

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Synonyms

Digitigrade gait; Equinus gait; Idiopathic toe walking; Persistent toe walking

association between toe walking and autism/language disorders remains unclear. It may be related to a sensory disorder or to the persistence of a remnant of the infantile tonic labyrinthine reflex.

See Also

► [Language Disorder](#)

References and Reading

- Accardo, P. J., & Whitman, B. Y. (1989). Toe walking: A marker for language disorders in the developmentally disabled. *Clinical Pediatrics*, 28, 347–350.
- Accardo, P. J., Morrow, J., Heaney, M. S., Whitman, B. Y., & Tomazic, T. (1992). Toe walking and language development. *Clinical Pediatrics*, 31, 158–160.
- Barrow, W., Jaworski, M., & Accardo, P. J. (2011). Persistent toe walking in autism. *Journal of Child Neurology*, 26, 619–621.
- Shulman, L. H., Sala, D. A., Chu, M. L. Y., McCaul, P. R., & Sandler, B. J. (1997). Developmental implications of idiopathic toe walking. *Journal of Pediatrics*, 130, 541–546.

Tofranil

► [Imipramine](#)

Toilet Training

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Definition

The process of achieving continence by applying a systematic approach to teach another to gain control over urine and bowel movements. Successful toilet training is a combination of attaining continence while also completing the chain of behaviors associated with toileting including traveling to the

bathroom, undressing, voiding into the toilet, redressing, washing hands, and leaving the bathroom. Teaching continence in toilet training is often achieved using one or more of the following teaching components and strategies: scheduled sitting, urine alarms, reinforcement for in-toilet voids, hydration, prompting and prompt fading, over-correction, and environmental restitution.

Historical Background

The systematic teaching of toilet training was born in the 1960s paralleling the rise of behaviorism. Toilet training is one of the cardinal skills that lent itself to behavioral research in that it was relatively easy to study in terms of both target behavior descriptions and clear, definable outcomes. That is, voids were distinctly defined and mutually agreeable with little room for error in definition or observation. Similar to most of the behavioral research coming out of this era, the early work on toileting was conducted largely with institutionalized persons globally developmentally delayed or disabled, both children and adults, acting as samples of convenience. Early work focused on systematic presentation of prompts to void and positive reinforcement for accurate voiding behavior. The beginning body of toilet training research was furthered by Drs. Nathaniel Azrin and Richard Foxx who are credited with developing the rapid toilet training (RTT) method, the most cited and comprehensive toilet training protocol, and widely adapted for everyone from typically developing children to profound developmental disabilities such as autism to subspecialty, rare diagnoses such as Angelman syndrome. The novelty and appeal of the RTT approach was two-fold in that it provided a comprehensive approach targeting all toileting behaviors at once (i.e., bladder and bowel continence, as well as associated self-help skills of undressing and redressing) and achieved results in an extraordinary short amount of time (median of 4 days to train to independent toileting behavior). The original RTT method included hydrating the individuals for increased opportunity to void, using graduated guidance with prompt fading as

individuals were more successful with the set elimination schedule, contingency management with positive reinforcement for dry pants checked systematically and in-toilet voids and punishment consisting of overcorrection and environmental restitution for accidents and out-of-toilet voids. Despite the success of RTT, the protocol is often disaggregated and components used individually as training protocols in and of themselves. This reduction of protocol is largely due to shifts in treatment procedures and setting locations. Most notably, the use of punishment in primary treatment protocols has fallen by the wayside in support of more popular positive behavior support interventions and institutions have been replaced by home and school treatment settings, extending privacy rights with reduced apparatus needs.

Current Knowledge

The current state of the science for toilet training research supports the use of the Foxx and Azrin protocol, as well as its empirically supported derivatives. The RTT method has been largely adapted and widely accepted for its use in positive reinforcement protocols and as it applies to individuals with autism. Empirical evidence has repeatedly shown that an intensive toilet training method with positive reinforcement for target behaviors and void-specific contingency management is effective and, in turn, effective with children with autism. Most toileting studies published within the past 20 years focus largely on RTT-derived protocols and particularly in the area of positive reinforcement based training (see Kroeger and Sorensen-Burnworth 2009 for review; Kroeger and Sorensen-Burnworth 2010). Other areas of foci include stimulus controls studies and behavior-specific problem solving (e.g., Hagopian et al. 1993; Luiselli 1996). That is, the research has moved to cases where the established protocols have failed and revised the protocol to problem solve specifically at the target behavior. For example, if the individual only voids when in water, the intervention begins with first gaining control over the behavior in water. Non-academic professionals and paraprofessionals

also successfully implement the translational model of toilet training with a focus on the positive reinforcement contingent upon scheduled sittings. Often when the research becomes practice, residual issues are noted to occur including additional bowel movement training, communication to use the restroom, deficits in redressing, and non-self-initiated voiding.

Future Directions

Future research endeavors include expansion of trainer characteristics, as well as problem-solving residual issues. A number of toileting protocols exist within the literature, many of them successful and capable of generalization to a wide variety of individuals within the autism and developmental disability population. While most of the programs are modified versions of the toileting protocol presented by Foxx and Azrin, studies focus on abbreviating the protocols while also reducing steps, components, and professional training involvement. That is, the trend is to make less complicated the toileting protocols previously originated. Future areas of toileting literature should focus on the following: (1) collateral behaviors pivotal to successful toileting training, including communication, self-initiation, and bowel movement training; (2) exploration of age and functioning limits (i.e., how young to train and how cognitively disabled is trainable); and (3) review of necessary prerequisite skills suggested present before initiating toilet training. Such investigations will serve to continue the body of literature surrounding toilet training, while at the same time providing improved quality of life for individuals with autism spectrum disorders and intellectual disability.

See Also

- [Adaptive Behavior](#)
- [Constipation](#)
- [Functional Assessment and Curriculum for Teaching Everyday Routines](#)
- [Functional Life Skills](#)

- ▶ Prompt Hierarchy
- ▶ Prompting
- ▶ Visual Supports

References and Reading

- Ando, H. (1977). Training autistic children to urinate in the toilet through operant conditioning. *Journal of Autism and Childhood Schizophrenia*, 7, 151–163.
- Averink, M., Melein, L., & Duker, P. C. (2005). Establishing diurnal bladder control with the response restriction method: Extended study on its effectiveness. *Research in Developmental Disabilities*, 26, 143–151.
- Azrin, N. H., & Foxx, R. M. (1971). A rapid method of toilet training the institutionalized retarded. *Journal of Applied Behavior Analysis*, 4, 89–99.
- Azrin, N. H., Bugle, C., & O'Brien, F. (1971). Behavioral engineering: Two apparatuses for toilet training retarded children. *Journal of Applied Behavior Analysis*, 4, 249–252.
- Cicero, F. R., & Pfadt, A. (2002). Investigation of a reinforcement-based toilet training procedure for children with autism. *Research in Developmental Disabilities*, 23, 319–331.
- Dalrymple, N. J., & Ruble, L. A. (1992). Toilet training and behaviors of people with autism: Parent views. *Journal of Autism and Developmental Disorders*, 22, 265–275.
- Duker, P. C., Averink, M., & Melein, L. (2001). Response restriction as a method to establish diurnal bladder control. *American Journal on Mental Retardation*, 106, 209–215.
- Foxx, R. M., & Azrin, N. H. (1973). *Toilet training persons with developmental disabilities: A rapid program for day and nighttime independent toileting*. Harrisburg: Help Services Press.
- Hagopian, L. P., Fisher, W., Piazza, C. C., & Wierzbicki, J. J. (1993). A water-prompting procedure for the treatment of urinary incontinence. *Journal of Applied Behavior Analysis*, 26, 473–474.
- Hyams, G., McCoull, K., Smith, P. S., & Tyrer, S. P. (1992). Behavioural continence training in mental handicap: A 10-year follow-up study. *Journal of Intellectual Disability Research*, 36, 551–558.
- Kroeger, K. A., & Sorensen-Burnworth, R. (2009). Toilet training individuals with autism and other developmental disabilities: A critical review. *Research in Autism Spectrum Disorder*. <https://doi.org/10.1016/j.rasd.2009.01.005>.
- Kroeger, K. A., & Sorensen-Burnworth, R. (2010). A parent training model for toilet training children with autism. *Journal of Intellectual Disability Research*. <https://doi.org/10.1111/j.1365-2788.2010.01286.x>.
- Leaf, R., & McEachin, J. (1999). *A work in progress: Behavior management strategies and a curriculum for intensive behavioral treatment of autism*. New York: Autism Partnership.
- LeBlanc, L. A., Carr, J. E., Crossett, S. E., Bennett, C. M., & Detweiler, D. D. (2005). Intensive outpatient behavioral treatment of primary urinary incontinence of children with autism. *Focus on Autism and Other Developmental Disabilities*, 20, 98–105.
- Lott, J. D., & Kroeger, K. A. (2004). Self-help skills in persons with mental retardation. In J. L. Matson, R. B. Laud, & M. L. Matson (Eds.), *Behavior modification for persons with developmental disabilities: Treatment and supports* (Vol. II). New York: National Association for the Dually Diagnosed.
- Luiselli, J. K. (1996). A case study evaluation of a transfer-of-stimulus control toilet training procedure for a child with pervasive developmental disorder. *Focus on Autism and Other Developmental Disabilities*, 11, 158–162.

Token Economy

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Definition

The token economy is a highly individualized, reinforcement-based, behavior change system, derived from the principles of operant conditioning that can be used with individuals or groups. The specific components of a token economy include (a) the identification of measurable and observable target behaviors (e.g., verbal utterances, academic readiness behaviors) by intervention agents (e.g., teachers, parents), (b) the administering of tokens (e.g., points, check marks, real or imitation money), which function as generalized conditioned reinforcers, by the intervention agent to the participant, and (c) the exchange of tokens by the participant for a variety of backup reinforcers (e.g., line leader, computer time, snacks). Generally speaking, participants earn tokens contingent on the occurrence of target behaviors; subsequently participants exchange the tokens for a variety of backup reinforcers (Cooper et al. 2007). Likely the most widely known and

used token in the world is money. Money is not initially reinforcing but acquires the properties of a reinforcer after multiple pairings with another reinforcer; it is a conditioned reinforcer that can be exchanged for a wide variety of backup reinforcers (e.g., food, shelter, clothing, entertainment, transportation).

Historical Background

The origins of the token economy system can be traced back to Wolfe (1936) and Cowles (1937) during which time investigators provided tokens to chimpanzees when they engaged in correct responses, and later trained the chimpanzees to exchange the tokens for grapes. However, few token economy systems were utilized with humans prior to the 1960s. Pioneering research conducted by Ayllon and Azrin (1965, 1968) with adult psychiatric inpatients, as well as Staats and Wolf with children (Staats et al. 1962, 1964), resulted in the peak of token economy research with humans in the 1970s and 1980s.

The all-encompassing literature surrounding the token economy is beyond the scope of this entry (for a review of the literature see Kazdin 1982; Kazdin and Bootzin 1972; Matson and Boisjoli 2009; O'Leary and Drabman 1971); however, the contributions of the system are noteworthy and a brief mention of various previous research conducted on token economies including those outside of the autism literature demonstrate the robust nature of the system. The token economy has been shown to increase a variety of appropriate behaviors and decrease a plethora of inappropriate behaviors, in a wide array of settings, with diverse populations, such as spontaneous appropriate sentences in children with autism (Hung 1978), safety performance in open-pit mining (Fox et al. 1987), letter recognition and labeling with preschool children (Kincaid and Weisberg 1978), dietary compliance for children on hemodialysis (Magrab and Papadopoulou 1977), personal hygiene, personal management, ward work, and social skills with chronic psychiatric inpatients (Nelson and Cone 1979), school

achievement of children with Down's syndrome (Dalton et al. 1973), academic readiness behaviors of children with developmental disabilities (Playnick et al. 2010), academic performance of children with autism (Charlop-Christy and Haymes 1998), encouraging car pool formation on a university campus (Jacobs et al. 1982), cleaning and hygiene behaviors of maximum security correctional institution inmates (Milan and McKee 1976), and conversation skills for adolescents with autism (McGee et al. 1984).

The above paragraph is an indicator of the depth and breadth of token economy research and by the early 1970s there were sufficient studies conducted on the efficacy of token economies (although, not all specific to autism) (e.g., Birnbrauer et al. 1965; Broden et al. 1970; Bushell et al. 1968; Girardeau and Spradlin 1964; Hewett et al. 1969; Hunt et al. 1968; Hunt and Zimmerman 1969; Meichenbaum et al. 1968; O'Leary et al. 1969; Phillips 1968; Schaefer and Martin 1966; Tyler and Brown 1968) to warrant a comprehensive literature review. O'Leary and Drabman (1971) conducted a literature review of token economy systems in the classroom. The authors reviewed the literature, identified variables that could potentially influence token economy effectiveness, and provided an in-depth discussion regarding the concern that token economies did not necessarily result in generalization across situations or maintain upon withdrawal of the program. Additionally, Kazdin and Bootzin (1972) published an evaluative review of the token economy. This review of the token economy differed from that of O'Leary and Drabman in that it was not limited to the use of token economies in the classroom and it provided a discussion on the methodology of token economy investigations. However, there was one salient similarity between the reviews, and that was the lengthy discussion regarding stimulus and response generalization limitations and suggested procedures to increase generalization in future token economy investigations.

Early studies conducted with individuals with autism did not address generalization; however, they did demonstrate the efficacy of token

economy systems with individuals with autism on both basic and complex behaviors. Ferster and DeMyer (1961, 1962) taught individuals with autism a basic response (key pressing) through the use of tokens (i.e., coins), which could be inserted into a vending machine to obtain a variety of reinforcers (e.g., food). Metz (1965) extended the research by increasing the complexity of the target behavior. Metz reinforced nonverbal imitative responses of children with autism, with tokens, which could be exchanged for food. However, subsequent research may have been influenced by the suggestions provided by the O'Leary and Drabman (1971) and Kazdin and Bootzin (1972) reviews, as many studies specific to individuals with autism did attempt to assess the generalization and maintenance of behaviors learned during token economy interventions during the late 1970s and 1980s. Hung (1977) investigated the effect of token value manipulations on spontaneous question-asking and generalization. Results indicated that the occurrence of generalized spontaneous question-asking was minimal until token values were manipulated and increased. McGee et al. (1984) taught individuals with autism to make positive and negative assertions in the context of two game situations to increase conversational skills during leisure time with the use of a token economy system. Generalization from trained to spontaneous assertions did occur within response classes. A particularly impressive demonstration of generalization occurred in the investigation by Krantz, Zalsenski, Hall, Fenske, and McClannahan (1981) in which a token economy was used to teach individuals with autism complex expressive speech (i.e., the use of nouns, verbs, shapes/size, and color adjectives). Results indicated all children generalized their skills to a new teacher, a new classroom, and a new set of stimulus materials. Handen, Apolito, and Seltzer (1984) illustrated the assessment of maintenance. The authors used a token economy to decrease the rate of repetitive speech in an adolescent with autism and found that low rates of repetitive speech maintained at both a 9- and 14-month follow-up.

Kazdin (1982) published a follow-up paper to the Kazdin and Bootzin (1972) review that

discussed the significant issues related to effective token economy implementation such as staff training, client resistance, response maintenance, integrity of treatment, administrative and organizational issues, and dissemination of the token economy. However, the overall lack of current literature specific to the implementation of token economies with individuals with autism prohibits an analysis of research related to the issues raised by Kazdin nearly 30 years ago.

Current Knowledge

As previously stated, minimal research on the token economy related to individuals with autism is currently being conducted. In fact, Matson and Boisjoli (2009) conducted a review of the literature surrounding the token economy for children with intellectual disabilities and autism and found only seven investigations specifically related to individuals with autism (Handen et al. 1984; Hung 1977; Kahng et al. 2003; McDonald and Hemmes 2003; Odom et al. 1985; Steeves et al. 1970; Tarbox et al. 2006).

A majority of the studies conducted during this time frame fall into one of two categories. One, researchers were investigating the effects of the token economy as the independent variable on various dependent variables. For example, McDonald and Hemmes (2003) investigated the use of a token economy and prompts to increase social initiations by an adolescent with autism and reciprocity effects. Social initiations by the participant were higher during the token economy training conditions. Additionally, spontaneous staff initiations toward the youth increased during treatment when the frequency of the youth's initiations increased. Kahng et al. (2003) increased food acceptance in a preschool-aged child who had speech delay and possible pervasive developmental disorder by using escape from a meal and a token economy. The child could earn tokens by accepting a bite of food and she could exchange the tokens for meal termination. Results indicated that this condition (as compared to differential positive reinforcement of alternative behavior and differential positive reinforcement of

alternative behavior plus physical guidance) was effective at increasing the number of bites accepted and decreasing the instances of food refusal. Finally, Tarbox et al. (2006) used a token economy to increase the attending behavior of a young child with autism. Attending behaviors increased; however, the behaviors did not maintain during conditions in which the tokens were not exchanged for backup reinforcers and delay to exchange of the backup reinforcers occurred.

Two, investigators were interested in comparing various treatment approaches and the token economy is one component within the training conditions, that is not specifically being manipulated or assessed. For example, Gena, Couloura, and Kymissis (2005) conducted a comparison of video modeling and in vivo modeling to teach preschoolers affective behavior. However, both conditions included the use of verbal praise and a token economy. The authors reported that both treatments (in vivo and video modeling) successfully increased affective responding by all participants and generalization occurred across responses to untrained scenarios, the child's mother, new therapists, and time. Additionally, Hilton and Seal (2007) conducted a comparative analysis of applied behavior analysis (ABA) and Developmental, Individual Difference, Relationship-Based model (DIR) in twin brothers with autism. The ABA program, among other things, consisted of a token economy. The authors reported variable improvement in both the ABA and DIR programs.

However, there is a third category that includes research that has begun to investigate some of the salient issues raised by Kazdin's (1982) review. First, Kazdin iterated the importance of staff training and the relationship to procedural integrity within the constraints of existing settings, stating, "too little attention has been accorded the manner in which the program is monitored and implemented" (p. 437). Plavnick et al. (2010) assessed the effects of self-monitoring on the procedural integrity of token economy implementation by staff in a special education classroom. Additionally, the academic readiness behaviors (i.e., appropriate sitting and vocalizing) of students with autism and Williams syndrome were

assessed during conditions in which staff were (1) trained on token economy implementation, (2) independently implemented the token economy, and (3) self-monitored their implementation of the token economy. Results suggested that procedural integrity was high during the self-monitoring condition for the staff and the percentage of intervals in which students engaged in academic readiness behaviors increased during conditions of high procedural integrity. Second, Kazdin discussed the notion of client resistance to the token economy and subsequently suggested the creative selection of reinforcers and opportunities for clients to have input into the system. Charlop-Christy and Haymes (1998) investigated an important component of the token economy that involved creative tokens and participant input. The authors investigated the effectiveness of typical tokens (stars) and objects of obsession as tokens ("micromachine cards or trucks, the letter 'A,' names of characters from videos, and plastic beads") on task performance for children with autism. The participants performed with greater accuracy during the obsessions as tokens conditions, and although the authors provided many considerations for such results, it began to address the leading issues that Kazdin outlined many decades prior.

Based on the review of the literature above, it may be apparent that token economy systems vary greatly in terms of simplicity and complexity. The design and implementation of a token economy system can be cumbersome and requires meticulous planning and execution; however, if done properly, the system can be highly effective. Although future research is needed to address all the facets of token economy systems in relation to individuals with autism; the likelihood of implementing a successful token economy can be increased by utilizing the basic steps below:

1. Intervention agent identification.
 - (a) Determine who will administer tokens. Intervention agents may include one person (e.g., school professional, parent) or everyone employed in a school building (e.g., general educators, special educators,

- paraprofessional, bus drivers, administrators, cafeteria workers).
- (b) Administer detailed training to all individuals who have regular contact with the participant(s). Include procedures that specify the participants, target behaviors, tokens, rules regarding the distribution and exchange of tokens, identification of and availability of backup reinforcers, protocol for not meeting criteria, policies regarding unused tokens, plans for generalization, maintenance, and withdrawal of the token economy, and so forth.
 - (c) Monitor intervention agents.
 - (d) Evaluate and respond to the needs of the intervention agents.
2. Participant identification.
 - (a) Determine who will participate in the token economy. Participants may include, for example, just one individual, groups within a classroom, or all students within a school district.
 - (b) Determine criteria for the inclusion, exclusion, and removal of participants in the token economy.
 3. Target behavior identification. Myles, Moran, Ormsbee, & Downing (1992) provide the following guidelines regarding target behavior identification.
 - (a) Select observable and measureable target behaviors. These may be the same behaviors for all participants or different behaviors for each participant.
 - (b) Determine the criteria for each target behavior. For example, if "on-task" is the target behavior, how many seconds, minutes, or instances of on-task behavior need to occur before a token will be distributed.
 - (c) Require first a few, easy behaviors that gradually increase as the individual becomes successful.
 - (d) Ensure that the participant has prerequisite skills necessary to perform the target behavior.
 4. Token identification.
 - (a) Select tokens that are safe (cannot be swallowed or cause injury), unique (so that participants cannot buy, access, accumulate tokens independent of the occurrence of the target behavior), durable, readily available, and inexpensive (Cooper et al. 2007). The research is inconclusive regarding the comparative efficacy of neutral or highly desirable tokens (see Charlop-Christy and Haymes 1998 for one such investigation).
 - (b) Note that praise should always be paired with token distribution to increase the likelihood that praise will eventually function as a conditioned or generalized conditioned reinforcer.
 5. Backup reinforcer identification.
 - (a) Reinforcers should be nonintrusive and occur naturally in the environment (e.g., music, books, teacher helper) and should never prohibit the individual from accessing basic needs (e.g., food, warmth, hygiene products).
 - (b) If the above reinforcers prove to be ineffective, reinforcers that are not naturally occurring (e.g., candy, pop, video-game time) may be considered.
 - (c) Determine the price of the backup reinforcers. For example, highly preferred items may cost more than low preference items or items may be auctioned. Additionally, the cost of backup reinforcers may increase or the value of tokens may decrease as the participant becomes more proficient in the target behavior (Cooper et al. 2007).
 6. Cooper et al. (2007) suggest field-testing the system prior to implementation. Data from the field-test can be used to make adjustments to the system prior to full-scale implementation.
 7. Finally, participant training. Training procedures will vary based on the functioning level of the participant. For higher functioning participants, an explanation may suffice. However, lower functioning participants may require explicit training, models, physical and verbal prompts (Cooper et al. 1987). Snell (1983) offers a helpful step-by-step training protocol for training lower functioning individuals to use a token economy.

An optional element that can be used in conjunction with the reinforcement-based token economy is response cost. Response cost is a form of punishment by removal, in which reinforcement is removed based on the occurrence of inappropriate behavior and results in the decreased likelihood of such future behavior. For example, an individual with autism may earn tokens for the use of spontaneous communicative acts (e.g., unprompted exchange of picture symbols, unprompted vocal verbal mands, unprompted gestures) and lose points (i.e., response cost) for disruptive behaviors (e.g., humming, tongue clicking, aggression). Therefore, in a 30-min period, the individual may earn 10 tokens for spontaneous communicative acts, and lose 2 tokens for instances of aggression, in which case the individual would have 8 tokens available for exchange of backup reinforcers. There are many serious considerations that should be contemplated before deciding upon the use of any punishment-based system, and response cost is not an exception. For more information regarding these considerations, please see Cooper et al. (1987, 2007).

Future Directions

The future direction of token economy research specific to individuals with autism is largely unknown. Historically, there was a flurry of research during the 1970s and 1980s, but has nearly ceased in recent decades. There are still many unanswered questions regarding token economy systems and individuals with autism. For example, it would be important to, (a) consider the feasibility of training intervention agents such as parents, teachers, caregivers, speech and language pathologists, or individuals other than researchers, (b) assess the effects of procedural integrity on participant skill acquisition, (c) identify the ideal schedules of reinforcement to maintain performance upon the removal of token economies, (d) study the effects of participant input on reinforcer and contingency selection, (e) address all aspects of stimulus and response generalization, (f) study efficacy of token economies in relation to participant demographics, such as age, cognitive

level, and specific diagnosis on the spectrum, (g) compare individual versus group token economies, and (h) compare the token economy to other treatment options in terms of time, cost, resources, feasibility, utility, social validity, and efficacy. However, ultimately the future direction of autism research as it relates to the token economy will be determined by the research questions that investigators deem most crucial, applicable, and feasible at that point in time. The possibilities are endless and the future is quite exciting.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavior Analysis](#)
- [Behavior Modification](#)
- [Early Intensive Behavioral Intervention \(EIBI\)](#)
- [Positive Reinforcement](#)
- [Reinforcer](#)
- [Response Cost](#)
- [Social Skill Interventions](#)

References and Reading

- Ayllon, T., & Azrin, N. H. (1965). The measurement and reinforcement of behavior of psychotics. *Journal of Experimental Analysis of Behavior*, 8, 356–383.
- Ayllon, T., & Azrin, N. H. (1968). *The token economy*. New York: Appleton.
- Birnbrauer, J. S., Wolf, M. M., Kidder, J. D., & Tague, C. E. (1965). Classroom behavior of retarded pupils with token reinforcement. *Journal of Experimental Child Psychology*, 2, 219–235.
- Brodin, M., Hall, R. V., Dunlap, A., & Clark, R. (1970). Effects of teacher attention and a token reinforcement system in a junior high school special education class. *Exceptional Children*, 36, 241–349.
- Bushell, D., Wrobel, P., & Michaelis, M. (1968). Applying “group” contingencies to the classroom study behavior of preschool children. *Journal of Applied Behavior Analysis*, 1, 55–62.
- Charlop-Christy, M. H., & Haymes, L. K. (1998). Using objects of obsession as token reinforcers for children with autism. *Journal of Autism and Developmental Disorders*, 28(3), 189–198.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (1987). *Applied behavior analysis*. Upper Saddle River: Prentice Hall.
- Cooper, J. O., Herron, T. E., & Heward, W. L. (2007). *Applied behavior analysis*. Upper Saddle River: Prentice Hall.

- Cowles, J. T. (1937). Food-tokens as incentives for learning by chimpanzees. *Comparative Psychology Monograph*, 14 (5, Serial No. 71).
- Dalton, A. J., Rubino, C. A., & Hislop, M. W. (1973). Some effects of token rewards on school achievement of children with Down's syndrome. *Journal of Applied Behavior Analysis*, 6, 251–259.
- Ferster, C. B., & DeMyer, M. K. (1961). The development of performances in autistic children in an automatically controlled environment. *Journal of Chronic Diseases*, 13, 312–345.
- Ferster, C. B., & DeMyer, M. K. (1962). A method for the experimental analysis of the behavior of autistic children. *The American Journal of Orthopsychiatry*, 1, 87–110.
- Fox, D. K., Hopkins, B. L., & Anger, W. K. (1987). The long-term effects of a token economy on safety performance in open-pit mining. *Journal of Applied Behavior Analysis*, 20, 215–224.
- Gena, A., Couloura, S., & Kymissis, E. (2005). Modifying the affective behavior of preschoolers with autism using in-vivo or video modeling and reinforcement contingencies. *Journal of Autism and Developmental Disorders*, 35, 545–556.
- Girardeau, F. L., & Spradlin, J. E. (1964). Token rewards in a cottage program. *Mental Retardation*, 2, 345–351.
- Handen, B. L., Apolito, P. M., & Seltzer, G. B. (1984). Use of differential reinforcement of low rates of behavior to decrease repetitive speech in an autistic adolescent. *Journal of Behavior Therapy and Experimental Psychiatry*, 15, 359–364.
- Hewett, F. M., Taylor, F. D., & Artuso, A. A. (1969). The Santa Monica project: Evaluation of an engineered classroom design with emotionally disturbed children. *Exceptional Children*, 35, 523–529.
- Hilton, J. C., & Seal, B. C. (2007). Brief report: Comparative ABA and DIR trails in twin brothers with autism. *Journal of Autism and Developmental Disorders*, 37, 1197–1201.
- Hung, D. W. (1977). Generalization of "curiosity" questioning behavior in autistic children. *Journal of Behavior Therapy and Experimental Psychiatry*, 8, 237–245.
- Hung, D. W. (1978). Using self-stimulation as reinforcement for autistic children. *Journal of Autism and Childhood Schizophrenia*, 8, 355–365.
- Hunt, J. G., & Zimmerman, J. (1969). Stimulating productivity in a simulated sheltered workshop setting. *American Journal of Mental Deficiency*, 74, 43–49.
- Hunt, J. G., Fitzhugh, L. C., & Fitzhugh, K. B. (1968). Teaching "exit-ward" patients appropriate personal appearance by using reinforcement techniques. *American Journal of Mental Deficiency*, 73, 41–45.
- Jacobs, H. E., Fairbanks, D., Poche, C. E., & Bailey, J. S. (1982). Multiple incentives in encouraging car pool formation on a university campus. *Journal of Applied Behavior Analysis*, 15, 141–149.
- Kahng, S. W., Boscoe, J. H., & Byrne, S. (2003). The use of an escape contingency and a token economy to increase food acceptance. *Journal of Applied Behavior Analysis*, 36, 349–353.
- Kazdin, A. E. (1982). The token economy: A decade later. *Journal of Applied Behavior Analysis*, 15, 431–445.
- Kazdin, A. E., & Bootzin, R. R. (1972). The token economy: An evaluative review. *Journal of Applied Behavior Analysis*, 5, 343–372.
- Kincaid, M. S., & Weisberg, P. (1978). Alphabet letters as tokens: Training preschool children in letter recognition and labeling during a token exchange period. *Journal of Applied Behavior Analysis*, 11, 199.
- Krantz, P. J., Zalsenski, S., Hall, L. J., Fenske, E. C., & McClannahan, L. E. (1981). Teaching complex language to autistic children. *Analysis and Intervention in Developmental Disabilities*, 1, 259–297.
- Magrab, P. R., & Papadopoulou, Z. L. (1977). The effect of a token economy on dietary compliance for children on hemodialysis. *Journal of Applied Behavior Analysis*, 10, 573–578.
- Matson, J. L., & Boisjoli, J. A. (2009). The token economy for children with intellectual disability and/or autism: A review. *Research in Developmental Disabilities*, 30, 240–248.
- McDonald, M. E., & Hemmes, N. S. (2003). Increases in social imitation toward an adolescent with autism: Reciprocity effects. *Research in Developmental Disabilities*, 24, 453–465.
- McGee, G. G., Krantz, P. J., & McClannahan, L. E. (1984). Conversational skills for autistic adolescents: Teaching assertiveness in naturalistic game settings. *Journal of Autism and Developmental Disorders*, 14, 319–330.
- Meichenbaum, D. H., Bowers, K., & Ross, R. R. (1968). Modification of classroom behavior of institutionalized female adolescent offenders. *Behaviour Research and Therapy*, 6, 343–353.
- Metz, J. R. (1965). Conditioning generalized imitation in autistic children. *Journal of Experimental Child Psychology*, 2, 389–399.
- Milan, M. A., & McKee, J. M. (1976). The cellblock token economy: Token reinforcement procedures in a maximum security correctional institution for adult male felons. *Journal of Applied Behavior Analysis*, 9, 253–275.
- Myles, B. S., Moran, M. R., Ormsbee, C. K., & Downing, J. A. (1992). Guidelines for establishing and maintaining token economies. *Intervention in School and Clinic*, 27, 164–169.
- Nelson, G. L., & Cone, J. D. (1979). Multiple-baseline analysis of a token economy for psychiatric inpatients. *Journal of Applied Behavior Analysis*, 12, 255–271.
- O'Leary, K. D., & Drabman, R. (1971). Token reinforcement programs in the classroom: A review. *Psychological Bulletin*, 75, 379–398.
- O'Leary, K. D., Becker, W. C., Evans, M. B., & Saudargas, R. A. (1969). A token reinforcement program in a public school: A replication and systematic analysis. *Journal of Applied Behavior Analysis*, 2, 3–13.
- Odom, S. L., Hoyson, M., Jamieson, B., & Strain, P. S. (1985). Increasing handicapped preschooler's peer social interactions: Cross-setting and component analysis. *Journal of Applied Behavior Analysis*, 18, 3–16.
- Phillips, E. L. (1968). Achievement place: Token reinforcement procedures in a home-style rehabilitation

- setting for “pre-delinquent” boys. *Journal of Applied Behavior Analysis*, 1, 213–224.
- Plavnick, J. B., Ferreri, S. F., & Maupin, A. N. (2010). The effects of self-monitoring on the procedural integrity of a behavioral intervention for young children with developmental disabilities. *Journal of Applied Behavior Analysis*, 43, 315–320.
- Schaefer, H. H., & Martin, P. L. (1966). Behavioral therapy for “apathy” of hospitalized schizophrenics. *Psychological Report*, 19, 1147–1158.
- Snell, M. E. (1983). Implementing and monitoring the IEP: Intervention strategies. In M. E. Snell (Ed.), *Systematic instruction of the moderately and severely handicapped* (2nd ed., pp. 113–145). Columbus: Charles E. Merrill.
- Staats, A. W., Staats, C. K., Schutz, R. E., & Wolf, M. M. (1962). The conditioning of textual responses using “extrinsic” reinforcers. *Journal of Experimental Analysis of Behavior*, 4, 33–40.
- Staats, A., Finley, J., Minke, K. A., Wolf, M., & Brooks, C. (1964). A reinforcer system and experimental procedures for laboratory study of reading acquisition. *Child Development*, 35, 209–231.
- Steeves, J. M., Martin, G. L., & Pear, J. J. (1970). Self-imposed time-out by autistic children during an operant training program. *Behavior Therapy*, 1, 371–381.
- Tarbox, R. S., Ghezzi, P. M., & Wilson, G. (2006). The effects of a token reinforcement on attending in a young child with autism. *Behavioral Interventions*, 21, 155–164.
- Tyler, V. O., & Brown, G. D. (1968). Token reinforcement of academic performance with institutionalized delinquent boys. *Journal of Educational Psychology*, 59, 164–168.
- Wolfe, J. B. (1936). Effectiveness of token rewards for chimpanzees. *Comparative Psychology Monograph*, 11, (5, Series No. 60).

Token Test for Children – Second Edition (TTFC-2)

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Synonyms

TTFC-2

Description

The *Token Test for Children, Second Edition* (TTFC-2) is a quick 10- to 15-min screening

measure intended for identifying receptive language difficulties in children ages 3–12 years and 11 months. The overall score reflects the child’s ability to follow multiple-step oral directions, understand spoken language components, and recall short-term auditory memory. Syntactic structures, knowledge of prepositional and relational concepts, and semantic development are among the linguistic components assessed. The TTFC-2 manual notes that the test is an effective addition to comprehensive language assessment as a means of gathering targeted information of receptive language skills. Results from the TTFC-2 provide specified information that allows clinicians to glean the relative impact of receptive language difficulties and “subtle language deficits” on functional language and communication outcomes (Harris et al. 1983). Harris et al.’s (1983) exploration reveals that there is a parallel relationship between dichotic listening tasks and TTFC results, wherein results on the TTFC aligned with higher deficits in dichotic listening. Thus, the TTFC provides an accurate indicator of receptive language abilities that can be used when investigating relationships among receptive language abilities and other auditory language tasks. In this way, the TTFC-2 is a useful measure that provides critical information about receptive language deficits as a variable in overall language outcomes.

The TTFC-2 manual highlights that some training is necessary in order to ensure that internal consistency measures are accurate. Thus, the TTFC-2 may be administered by speech-language pathologists, school psychologists, special education teachers, education specialists, and those with training in standardized test administration (DiSimoni et al. 2007). Administration protocol begins with three unscored practice items before the evaluation. The test consists of 46 verbally transmitted commands that are divided into 4 parts according to level of complexity. The clinician is required to administer all 46 test items in *successive* order given the exception of children who cannot successfully complete the practice items.

Following the test administrator’s command, the test-taker is required to manipulate the

provided tokens under the constraints of the command. Commands are varied in length and complexity and utilize the variety of token colors and shapes to add complexity to each portion of the test. The materials required for administration include the TTFC-2 manual, 20 token manipulatives that differ in size (small and large), shape (circle and square) and color (green, blue, red, yellow, and white), and the provided test sheets (Alkhamra and Al-Jazi, 2016). The data recorded on the test sheets provides a measurement of test-taker success as a function of their ability or inability to successfully complete the verbal commands. Thus, score interpretation of the TTFC-2 identifies children that are significantly below their peers in receptive language abilities.

Five distinct types of informative scores, namely, standard score, percentile rank, standard error of measurement (SEM), descriptive rating, and listening age, are obtained from the recorded raw score. The standard score, age equivalents, and percentile ranks are converted from the raw score, and the SEM is a measure of age and coefficient alpha. As a final measure, six descriptive ratings ranging from very poor to very superior are obtained from the percentile ranks and standard scores (DiSimoni et al. 2007).

The scores on the TTFC-2 are a measure of language comprehension through the receptive mode. All scoring for the TTFC-2 is scored as correct (1) or incorrect (0). To receive a correct score, the task must be performed with 100% accuracy; no partial credit is received. Distractibility, impulsiveness, as well as a severe physical disability should be considered as possible confounding factors that may account for depressed scores (DiSimoni et al. 2007). Thus, lower scores on the TTFC-2 resulting from the inability to successfully carry out the commands may indicate the presence of a developmental disorder or extreme impairment of visual processing ability.

Historical Background

The original *Token Test*, designed by DeRenzi and Vignolo (1962), sought to measure receptive

language comprehension in adults with aphasia by way of following directions applied to token manipulatives. Several adaptations were made following the earliest rendition, all of which measured the patient's ability to manipulate the tokens upon five sets of oral commands that systematically increased in both length and complexity.

The emergence of research by DiSimoni in 1978 questioned the *Token Test's* efficacy when evaluating children for several reasons, including lack of age norms, socioeconomic group comparisons, and lack of clarity in data collection. This research prompted DiSimoni's development of the *Token Test for Children* (TTFC) in 1978, which integrated the variety of command sequences and use of tokens present in the original test with standardized administration protocol and increased sample size to forge its viability for children aged 3 years to 12 years and 6 months.

Following the development of the TTFC, later research suggested that despite improvements in standardization and clarity, deficits in the psychometric stability of the test remained present. In 2007, DiSimoni, Ehrler, and McGhee developed the second edition, the TTFC-2, with detail to improving validity, reliability, and normative data to be critically representative of the demographics of the current US population. The TTFC-2 aims to improve shortcomings by providing targeted performance data based on a variety of ethnic, gender, disability, and race discrepancies.

Psychometric Data

Standardization data represents the total population by matching sample criterion to the overall geographic region, gender, race, and exceptionality data noted in the US Census data report in 2004–2005. The standardization population consisted of 1310 children from 22 states and 136 geographically diverse communities. Various exceptionalities were presented in the inclusion of children with specific language impairment (SLI). The normative sample mirrors the current population, where 7% of the normative sample were children with SLI and 7.42% of the total

population is constituted by those with SLI (DiSimoni et al. 2007).

The representation of the sample spans across three targeted subgroups. These subgroups consist of children who were classified as representative of the mainstream, minority, or exceptional population. The mainstream subgroup includes male and female Caucasians while the minority subgroup includes male and female African Americans, Hispanic Americans, Asian Americans, and American Indians. Finally, the exceptionality subgroup includes those children who are gifted and talented as well as individuals with autism, attention-deficit/hyperactivity disorders, articulation disorders, intellectual disabilities, learning disabilities, or SLI (DiSimoni et al. 2007).

The test reliability, validity, and score comparison results indicate consistencies and demonstrate the efficacy of the battery. Reliability is substantially positive with coefficients that range from an average of 0.96 for test-retest reliability to 0.99 for interscorer reliability. Validity coefficients further evidence consistencies through construct-identification and criterion-prediction validity that both indicated validity as a measure of five testable questions and three independent studies. Evidence of validity is further propagated by conventional and differential item analysis techniques that revealed accuracy in capturing the variance of receptive language skills as well as a lack of bias in the language of the command stimuli. As a final measure of validity, the scores and measurements from the TTFC-2 were directly compared with the Test of Early Language Development (TELD-3), the Receptive One-Word Picture Vocabulary Test (ROWPVT-2000), and the Expressive One-Word Picture Vocabulary Test (EOWPVT-2000) to reveal significant correlations with coefficients at the 0.001 level. Thus, the reliability and validity measures support the efficacy and psychometric stability of the TTFC-2 (DiSimoni et al. 2007).

Clinical Uses

The literature reveals that the TTFC-2 is not only a useful tool for children who speak Standard

American English, but also holds clinical utility when serving several diverse cultural-linguistic communities. Research by Alkhamra and Al-Jazi (2016) unveil the Arabic Token Test for Children (A-TTFC) as a valid, reliable, and appropriate measure of receptive language in the assessment of children who are native speakers of Arabic. The A-TTFC was developed as a translation of the TTFC, and as a translated test, the “content of the test and the construct measures most likely do not change. . .the test score is therefore less threatened” (Alkhamra and Al-Jazi 2016, p. 184). The study investigates this probability by systematically evaluating the performance of 397 Jordanian children on the A-TTFC. Prior to their evaluation, each child was tested to ensure their ability to hear the linguistic commands via administration of the Ling Six Sound Test. Data collection to determine the efficacy in standardization of the A-TTFC was performed through factor analysis, bilingual validation, and age difference analysis in order to “support the accuracy of the translation process [as well as] the validity of the A-TTFC” (Alkhamra and Al-Jazi 2016, p. 189). Thus, the A-TTFC provides a formal tool for receptive language assessment in native Arabic-speaking children that maintains the same clinical strengths and utility as the TTFC-2 for native English speakers.

In addition to the A-TTFC, research by Gallardo et al. (2011) uncovers many available versions of the TTFC that have been developed in Dutch, German, Khmer, Kannada, Polish, Portuguese, and several Spanish-speaking populations. Yet, the authors posit that many of these versions may not be viable for use in practice, as there is a lack of standardization and psychometric evidence that is necessary for efficacy (Gallardo et al. 2011). Thus, Gallardo et al.’s study sought to explore these measures of efficacy in the Revised Token Test (RTT) in 250 Spanish-speaking children aged 4–12 years old. Analyses of vocabulary knowledge and intelligence were recorded as a means of obtaining language performance overall before administering the tests. The RTT results were then correlated with the results on the prerequisite testing in order to determine criterion validity. Additional measures of factorial analysis and analysis of variance were carried out to arrive at a statistical

outcome. Results from this study indicate that the RTT is a reliable and valuable tool for the assessment of Spanish-speaking children due to its psychometric sensitivity. While the authors outlined these positive outcomes, they caution clinicians that further research into the normative data and increased sample size is warranted prior to use of the RTT with Spanish-speaking children (Gallardo et al. 2011).

See Also

- [Receptive Language](#)
- [Receptive Language Disorders](#)
- [Receptive-Expressive Emergent Language Test \(REEL-3\)](#)

References and Reading

- Alkhamra, R., & Al-Jazi, A. (2016). Validity and reliability of the Arabic token test for children. *International Journal of Language & Communication Disorders*, 51(2), 183–191.
- Brownell, R. (2000a). *Receptive one-word picture vocabulary Test-2000 edition*. Novato: Academic Therapy Publications.
- Brownell, R. (2000b). *Expressive one-word picture vocabulary test – 2000 edition*. Novato: Academic Therapy Publications.
- DeRenzi, E., & Vignolo, L. (1962). *Token test*: A sensitive test to detect receptive disturbances in aphasics. *Brain*, 85, 665–678.
- DiSimoni, F. (1978). *Token test for children*. Austin: PRO-ED.
- DiSimoni, F., McGhee, R., & Ehrler, D. (2007). *The token test for children* (2nd ed.). Austin: PRO-ED.
- Gallardo, G., Guàrdia, J., Villaseñor, T., & McNeil, M. (2011). Psychometric data for the revised token test in normally developing Mexican children ages 4–12 years. *Archives of Clinical Neuropsychology*, 26(3), 225–234.
- Harris, V. L., Keith, R. W., & Novak, K. K. (1983). Relationship between two dichotic listening tests and the token test for children. *Ear and Hearing*, 4(6), 278–282.
- Hresko, W. P., Reid, D. K., & Hammill, D. D. (1999). *Test of early language development-third edition*. Austin: PRO-ED.

Tolerance for Delay

- [Wait Training](#)

Tone of Voice

- [Intonation](#)

TONI

- [Test of Nonverbal Intelligence \(TONI-4\)](#)

Tonic-Clonic Seizure

- [Grand Mal Seizure](#)

ToP

- [Test of Playfulness \(ToP\)](#)

Topamax (Topiramate)

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Synonyms

[Topiramate](#)

Definition

Topamax is an anticonvulsant. The drug works by inhibiting glutamate activity (major excitatory neurotransmitter), attenuating activity of Na⁺ channels and high-voltage-activated Ca²⁺ channels (preventing depolarization, i.e., excitation),

and further potentiating gamma-aminobutyric acid (GABA) receptor-mediated inhibitory cell signaling. While there is some evidence of its effectiveness in ASD, further validation is required. Topamax's mechanism of action is in concordance with theories of increased excitation in ASD patients. Side effects may include weight loss, somnolence, and anorexia.

See Also

► [Anticonvulsants](#)

References and Reading

- Canitano, R. (2005). Clinical experience with topiramate to counteract neuroleptic induced weight gain in 10 individuals with autistic spectrum disorders. *Brain and Development*, 27(3), 228–232.
- Hardan, A. Y., Jou, R. J., & Handen, B. L. (2004). A retrospective assessment of topiramate in children and adolescents with pervasive developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 14(3), 426–432.
- Janowsky, D. S., Kraus, J. E., Barnhill, J., Elamir, B., & Davis, J. M. (2003). Effects of topiramate on aggressive, self-injurious, and disruptive/destructive behaviors in the intellectually disabled: An open-label retrospective study. *Journal of Clinical Psychopharmacology*, 23(5), 500–504.
- Mazzone, L., & Ruta, L. (2006). Topiramate in children with autistic spectrum disorders. *Brain and Development*, 28(10), 668.
- Rezaei, V., Mohammadi, M. R., Ghanizadeh, A., Sahraian, A., Tabrizi, M., Rezazadeh, S. A., & Akhondzadeh, S. (2010). Double-blind, placebo-controlled trial of risperidone plus topiramate in children with autistic disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 34(7), 1269–1272.

Topic Management

► [Discourse Management](#)

Topiramate

► [Topamax \(Topiramate\)](#)

TOPL-2

► [Test of Pragmatic Language-2 \(TOPL-2\)](#)

Total Communication (TC) Approach

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Definition

Total communication (TC) is an educational philosophy which was introduced to the field of deaf education in the 1960s (Schow and Nerbonne 2007). TC utilizes all modalities of communication (spoken, signed, and written) as well as lipreading, and gestures in the education of deaf children. Simultaneous communication (SimCom) is typically used in education settings that boast a TC approach (Power et al. 2008). Users of SimCom speak and sign simultaneously. Because American Sign Language (ASL) uses a different grammar and syntax from English, out of necessity the sign system that is used during SimCom is some form of manually coded English or a form of pidgin sign language. Because other forms of communication are used with children with autism, the total communication approach includes not only speech, signing, gestures, and written language, but also picture communication and voice output communication devices.

T

Historical Background

TC has been used in some form since the beginning of deaf education in the sixteenth century (Evans 1982). It was not until the mid-1800s that an oral-only approach began to take hold of the field of deaf education. In 1880, deaf educators from Europe and the United States met at the International Congress at Milan to discuss the

then-current debate over which educational philosophy (oral-only or a sign and spoken combined method) should be used. The results of the Congress at Milan led to a predominately oral-only approach in European countries and in the United States. The purely oral-only approach dominated the field of deaf education until the 1960s when David Denton at the Maryland School for the Deaf developed the idea for the TC approach (Lowenbraun et al. 1980).

In the 1960s, many researchers and educators of the deaf began to question the oral-only methods of teaching due to the alarmingly high numbers of illiterate or barely literate deaf students (Boatner 1965; Schein and Bushnag 1962; Trybus and Kachmer 1977). Also available at the time was research that focused on the comparisons of deaf children of hearing parents and deaf children of deaf parents. It was found that deaf children of deaf parents outperformed deaf children of hearing parents in measures of academic achievement and literacy skills (Quigley 1969; Stevenson 1964). These differences were attributed to the use of American Sign Language among the deaf families.

Both then and now, the total communication approach is not implemented homogenously. Stuckless (1976) was the first to point out that total communication “means different things to different people.” The inclusion of finger spelling to a largely oral-only approach is enough to label some programs TC. On the opposite end of the spectrum, programs that are based primarily on American Sign Language as the language of instruction and include written English label the educational philosophy TC. The following definition of TC was provided in 1976, and it remains the same today. TC is a “philosophy incorporating the appropriate aural, manual, and oral modes of communication in order to ensure effective communication with and among hearing impaired persons” (Garretson 1976, p. 300).

Although not labeled “total communication,” the approach was first used with children with autism in the 1970s. Carr et al. (1978) were among the first to use sign language with children with ASDs. The use of signs for nonverbal children with ASDs is still employed widely today. In

addition to the use of signs, picture symbols, picture communication boards, and low-tech and high-tech augmentative and alternative communication (AAC) devices are used with children with ASDs, and the approach is also termed “total communication” because any form or modality of communication is applied.

Rationale or Underlying Theory

The rationale for the TC approach has its beginnings in deaf education. In the 1960s, the attention turned to manual approaches of communication and education due to research that showed a more favorable outcome in literacy skills for children who were either born to deaf parents or who were educated through sign language (Hester 1963; Meadow 1968; Quigley 1969). This finding has been echoed in the literature more recently (Hermans et al. 2008; Strong and Prinz 2000). The basic findings correlate signing ability and reading ability such that those with better signing skills are better readers. It makes sense that those children who are better signers would be in an environment with much sign exposure such as in a household with deaf parents who use ASL.

Related to this is a proposal first described by Cummins in 1979 called the linguistic interdependence theory. The theory was originally meant to suggest an adjustment to bilingual education in a way that would incorporate the basic tenets of the model. Cummins (1979) asserts that the competence that can be achieved in a second language (L2) is dependent on and restricted by the competence individuals have in their first language (L1). Additionally, children must reach a threshold competency level in their L1 before learning an L2 becomes too cognitively taxing. Although the linguistic interdependence theory was not meant to be applied to deaf children, many see a parallel between the obstacles deaf children face when learning written English and the obstacles a second-language learner faces. Indeed, if a child learns ASL, which is a separate language from English, learning written and spoken English is an issue of a bilingual learner.

Regarding individuals with ASDs, the TC approach has been applied due to research that began in the 1970s related to the speech outcomes of children with autism who were taught manual signs. More recently, related to the idea of multiple intelligences (Gardner 1993) is the notion that, much like the normally developing population, individuals with ASDs are a heterogeneous group with different strengths and learning styles. Specifically, it has been found that some individuals with ASDs have differences in the way they process information (Harris et al. 1990; Siegel et al. 1996). Hermelin and O’Conner (1970) were the first to state that some individuals with ASDs process visuospatial information better than auditory information. Additionally, it has been found that the memory of individuals with ASDs is superior for visual information compared to auditory information (Prior and Chen 1976). Finally, research on the attention of individuals with ASDs supports the notion that they have difficulty selecting the most salient features of a stimulus (Frith and Baron-Cohen 1987).

The use of other forms of augmentative communication for individuals with ASDs seems logical based on the strengths that they possess. The use of sign language relieves the burden of having to process verbal information and builds on the strength of processing visuospatial information. The memory difficulties of individuals with ASDs can be alleviated by using picture communication symbols which do not require them to rely on their own memories to create a message. Individuals with ASDs can attend to the symbols for as long as necessary to comprehend the information, alleviating the difficulties related to attention needed for verbal information transfer. Sign language is seen as a viable option for nonverbal individuals with ASDs due to the ease with which the therapist can physically manipulate the hands for manual articulation compared to manipulating the mouth during speech production.

Goals and Objectives

It is estimated that 30–50% of individuals with autism spectrum disorders will not acquire

functional speech (National Research Council 2001; Luyster et al. 2005); the goal of the total communication approach is functional communication. Although verbal speech may not be possible for all individuals with autism, communication through any means is the ultimate goal.

Treatment Participants

Any individual who is unable to communicate to the level expected for their age is a candidate for augmentative and alternative communication of any form (Beukelman and Mirenda 2005). Therefore, because communication impairments are a hallmark of ASDs (Mirenda 2009), many individuals with ASDs are candidates for a total communication approach.

Treatment Procedures

Because the total communication approach is an eclectic one, there is no one set of procedures to be applied. Typically, whether referring to the use of manual signs or picture communication, the common practice is to begin training a small number of lexical items for requesting highly motivating objects (Beukelman and Mirenda 2005). Once the use of symbolic communication is established, more lexical items are introduced and are used for other communicative acts such as commenting and questioning.

Beukelman and Mirenda (2005) also support the use of natural consequences when applying AAC solutions to beginning communicators. For example, when using picture symbols to request an item, the language facilitator should provide the item that is requested even if the facilitator knows the request was a mistake or knows that the AAC user does not want the item. Giving the non-preferred item, and allowing for the natural consequence, teaches the AAC user that they must pay attention to their own communicative interactions and that they must weight their options carefully.

Efficacy Information

Efficacy research in the field of AAC is a relatively new addition to the literature. Bedrosian (1999) states that much early research in the field, as it should have been, was devoted to descriptive studies relating to describing the communication of AAC users. Since that publication, many more research studies have been conducted that are devoted to the efficacy of AAC for specific populations. Autism is one of those populations that have been widely studied. Overwhelmingly, the use of AAC has resulted in increased language skills in individuals with ASDs over treatment approaches that focus on speech alone. For most individuals with ASDs, accessing their relative strength in the visual domain has resulted in faster and more complex language growth in both signing and speaking. The use of manual signing in combination with speech training has been shown to increase language skill. The use of non-electronic-aided systems such as picture use has also been shown to increase functional communication, and a wide range of individuals with ASDs have been able to make use of this type of communication. High-tech AAC use has been shown to increase language abilities and speech output in individuals with autism as well. See Goldstein (2002) and Mirenda (2002) for reviews.

A meta-analysis of available research related to AAC use was conducted by Millar et al. (2006). Although the meta-analysis was not focused only on individuals with autism, the major finding was that use of AAC does “not have a negative impact on speech production” (p. 257), and in fact, speech production increased in individuals ages 2–60 years as a result of AAC interventions and across a range of different AAC interventions (aided and unaided).

Outcome Measurement

Because the goal of AAC use is functional communication, the outcome measurement should be the same. Functional communication will be defined differently based on the

cognitive skills of the individual and the type of AAC system that is in place.

Qualifications of Treatment Providers

AAC interventions are most typically introduced by a speech-language pathologist. Unfortunately, many speech-language pathologists do not report having adequate training or education in the field of AAC (King 1998; Marvin et al. 2003; Simpson et al. 1999), and a survey of education programs for speech-language pathologists has uncovered a need for better education in this area (Ratcliff et al. 2008). Although this is the case, speech-language pathologists are the best equipped of all professionals who work with individuals with autism to provide intervention that includes AAC. A listing of speech-language pathologists who are certified by the American Speech Language Hearing Association can be found on their website. A few short questions posed to the speech-language pathologist can reveal whether they are comfortable with the area of AAC.

See Also

- ▶ [Alternative Communication](#)
- ▶ [American Sign Language \(ASL\)](#)
- ▶ [Assistive Devices](#)
- ▶ [Communication Board](#)
- ▶ [Low-Technology Device](#)
- ▶ [Manual Sign](#)
- ▶ [Pictorial Cues/Visual Supports \(CR\)](#)
- ▶ [Sign Language](#)
- ▶ [Voice Output Communication Aids](#)

References and Reading

- Bedrosian, J. L. (1999). AAC efficacy research: Challenges for the new century. *Augmentative and Alternative Communication*, 15, 2–3.
- Beukelman, D. R., & Mirenda, P. (2005). *Augmentative and alternative communication: Supporting children and adults with complex communication needs*. Baltimore: Brooks Publishing.
- Boatner, E. B. (1965). *The need of a realistic approach to the education of the deaf*. Paper presented to the joint

- coll. vention of the California Association of Parents of Deaf and Hard of Hearing Children, California Association of Teachers of the Deaf and Hard of Hearing, and California Association of the Deaf.
- Carr, E. G., Binkoff, J. A., Kologinsky, E., & Eddy, M. (1978). Acquisition of sign language by autistic children I: Expressive labeling. *Journal of Applied Behavior Analysis*, 11, 489–501.
- Cummins, J. (1979). Linguistic interdependence and the educational development of bilingual children. *Review of Educational Research*, 49, 222–251.
- Evans, L. (1982). *Total communication. Structure and strategy*. Washington, DC: Gallaudet University Press.
- Frith, U., & Baron-Cohen, S. (1987). Perception in autistic children. In D. J. Cohen & A. M. Donnellan (Eds.), *Handbook of autism and pervasive developmental disorders*. New York: Wiley.
- Gardner, H. (1993). *Multiple intelligences: The theory in practice*. New York: Basic Books.
- Garretson, M. (1976). Total communication. *The Volta Review*, 78, 88–95.
- Goldstein, H. (2002). Communication intervention for children with autism: A review of treatment efficacy. *Journal of Autism and Developmental Disorders*, 32, 373–396.
- Harris, S., Handleman, J., & Burton, J. (1990). The Stanford Binet profiles of young children with autism. *Special Services in the School*, 6, 135–143.
- Hermans, D., Knoors, H., Orel, E., & Verhoeven, L. (2008). The relationship between the reading and signing skills of deaf children in bilingual education programs. *Journal of Deaf Studies and Deaf Education*, 13, 518–530.
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. London: Pergamon.
- Hester, M. S. (1963). Manual communication. In P. V. Doctor (Ed.), *Proceedings of interviews of the Council on Education of the Deaf and of the 41st meeting of the convention of American Institute of the Deaf*. Washington, DC: Gallaudet College.
- King, J. (1998). Preliminary survey of speech-language pathologists providing AAC services in health care settings in Nebraska. *Augmentative and Alternative Communication*, 14, 222–227.
- Lowenbraun, S., Appelman, K., & Callahan, J. (1980). *Teaching the hearing impaired through total communication*. Columbus: Charles E. Merrill.
- Luyster, R., Richler, J., Risi, S., Hsu, W., Dawson, G., Bernier, R., et al. (2005). Early regression in social communication in autism spectrum disorders: A CPEA study. *Developmental Neuropsychology*, 27, 311–336.
- Marvin, L. A., Montano, J. J., Fusco, L. M., & Gould, E. P. (2003). Speech-language pathologists' perception of their training and experience in using Alternative and Augmentative Communication. *Contemporary Issues in Communication Sciences and Disorders*, 30, 76–83.
- Meadow, K. P. (1968). Early manual communication in relation to deaf child's intelligence, social and communicative functioning. *American Annals of the Deaf*, 113, 21–41.
- Millar, D. C., Light, J. C., & Schlosser, R. W. (2006). The impact of augmentative and alternative communication intervention on the speech production of individuals with developmental disabilities: A research review. *Journal of Speech, Language, and Hearing Research*, 49, 248–264.
- Mirenda, P. (2002). Toward functional augmentative and alternative communication for students with autism: Manual signs, graphic symbols, and voice output communication aids. *Language, Speech, and Hearing Services in Schools*, 34, 203–216.
- Mirenda, P. (2009). Introduction to AAC for individuals with autism spectrum disorders. In P. Mirenda & T. Iacono (Eds.), *Autism spectrum disorders and AAC*. Baltimore: Paul H. Brookes.
- National Research Council. (2001). *Educating children with autism*. Committee on Educational Interventions for Children with Autism, Division of Behavioral and Social Sciences and Education. Washington, DC: National Academies Press.
- Power, D., Hyde, M., & Leigh, G. (2008). Learning English from signed English: An impossible task? *American Annals of the Deaf*, 153, 37–47.
- Prior, M., & Chen, C. (1976). Short-term and serial memory in autistic, retarded, and normal children. *Journal of Autism and Childhood Schizophrenia*, 6, 121–131.
- Quigley, S. P. (1969). The deaf and the hard of hearing. *Review of Educational Research*, 39, 103–123.
- Ratcliff, A., Koul, R., & Lloyd, L. L. (2008). Preparation in augmentative and alternative communication: An update for speech-Language Pathology training. *American Journal of Speech-Language Pathology*, 17, 48–59.
- Schein, J. D., & Bushnag, S. (1962). Higher education for the deaf in the United States: A retrospective investigation. *American Annals of the Deaf*, 107, 416–420.
- Schow, R. L., & Nerbonne, M. A. (2007). *Introduction to audiologic rehabilitation* (5th ed.). Boston: Pearson.
- Siegel, J., Minshew, N., & Goldstein, G. (1996). Wechsler IQ profiles in diagnosis of high-functioning autism. *Journal of Autism and Developmental Disorders*, 26, 389–406.
- Simpson, K., Beukelman, D., & Bird, A. (1999). Survey of school speech and language service provision to students with severe communication impairments in Nebraska. *Augmentative and Alternative Communication*, 14, 212–221.
- Stevenson, E. (1964). A study of the educational achievement of deaf children of deaf parents. *California News*, 80, 143.
- Strong, M., & Prinz, P. (2000). Is American Sign Language skill related to English literacy? In C. Chamberlain, J. P. Morford, & R. I. Mayberry (Eds.), *Language acquisition by eye* (pp. 131–141). Mahwah: Earlbaum.
- Stuckless, E. R. (1976). Manual and graphic communication. *Volta Review*, 78, 96–101.

Trybus, R., & Kachmer, M. (1977). School achievement scores of hearing-impaired children: National data on achievement status and growth patterns. *American Annals of the Deaf*, 122, 62–69.

Touch

► Tactile

Touch Pressure

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Definition

Touch pressure is a specific form of touch sensation, recording the amount of push on the receptor with the ultimate goal to avoid skin damage. The sensation of touch pressure is detected by skin receptors designed to provide graded information to the brain through spinal cord pathways. This touch pressure can be noticed, but if not perceived to be a threat to the integrity of the skin, then no action is taken. Occasionally, the term touch pressure is used to refer to the use of therapeutic touch in the form of massage or touch therapy to calm children who are diagnosed with autism.

References and Reading

- Field, T., Lasko, D., Mundy, P., Henteleff, T., Kabat, S., Talpins, S., et al. (1997). Brief report: Autistic children's attentiveness and responsivity improve after touch therapy. *Journal of Autism and Developmental Disorders*, 27, 333–338.
- Gardner, E. P., & Kandel, E. R. (2000). Touch. In E. R. Kandel, J. H. Schwartz, & T. M. Jessell (Eds.), *Principles of neural science* (pp. 451–471). New York: McGraw-Hill.
- Gertz, S. D. (2007). *Liebman's neuroanatomy made easy and understandable* (7th ed.). Austin: Pro-ED.

Touch Processing and Social Behavior in ASD

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Definition

Deficits in sensory functioning were recently recognized under the repetitive behavior criteria for the diagnosis of autism spectrum disorder (ASD) (APA 2013). It is estimated that atypical responses to sensory stimuli are present in 90% of individuals diagnosed with ASD (Tavassoli et al. 2014; Tomchek and Dunn 2007), with the sense of touch described by parents as one of the most impaired (Hilton et al. 2010; Lane et al. 2010).

Touch is a sensory modality used in the context of affiliative behaviors and social interaction, and some have proposed that it precedes more complex forms of social-emotional skill development, such as language or emotional expressiveness (Hertenstein et al. 2006). Touch is important to human social bonding and affect communication because it is a primary channel of communication in the first year of life (Gallace and Spence 2010; Morrison et al. 2010). ASD is a disorder characterized by a lack of social-emotional reciprocity, and a relationship between the tactile domain and other core symptoms of the disorder, namely, social problems, has been suggested. Specifically, it is hypothesized that aberrant touch processing is a potential precursor to these deficits.

Abnormal touch processing in ASD is often characterized by abnormal responses to textures, preoccupations with sensory features of objects, or problems habituating to prior sensory experiences (Hilton et al. 2010; Lane et al. 2010). Other

reports include defensive reactions and lower pleasantness ratings to tactile stimuli (Tomchek and Dunn 2007). Abnormal responses to touch in ASD seem to be present, prodromally, in the first year of life (Kadlaskar et al. 2019). Specifically, infants who are later diagnosed with ASD fail to shift their attention in response to caregiver touch, and even when they do attend to their mother's touch, they seem to orient away from it. In addition, infants who attend less to caregiver's touch at 12 months old present more ASD symptomatology as measured by the autistic diagnostic observation tool (ADOS) at age 3 (Kadlaskar et al. 2019). There seems to be evidence of tactile disfunction early in ASD, but the abnormal responses are heterogeneous and may have different etiologies.

Missing tactile stimuli in the environment (sensory hyporesponsiveness) has been found previously in young children with ASD (Foss-Feig et al. 2012). However, there are other mechanisms by which sensory impairments may relate to symptoms of ASD. For example, the degree to which a child seeks (sensory seeking) or is bothered by tactile stimuli (sensory hyperresponsiveness) can also result in failing to attend to appropriate stimuli in the environment and consequently result in missed learning opportunities (Baranek et al. 2007).

Atypical behavioral responses to touch may directly impact the way children and adolescents interact with others, both contemporaneously, and over time. It is hypothesized that children engaging in sensory seeking behaviors are too distracted with enhancing their sensory experiences and therefore miss social cues in their environment (Foss-Feig et al. 2012). Others suggest that hyperresponsiveness/tactile defensiveness is the pattern that mostly affects the ability to socialize in ASD (Miguel et al. 2017) and is in fact one of the patterns of behavior that best differentiates ASD from typically developing children. However, hyporesponsiveness to touch seems to be the pattern that gathers greater consensus among researchers on the cascading effects on development over time (Foss-Feig et al. 2012; Miguel et al. 2017). Children who miss tactile experiences due to hyporesponsiveness will have fewer opportunities to engage meaningfully and learn from their physical environment and social experiences.

The relationship between tactile and social processing in ASD may occur at multiple levels, and it is not yet well understood where in the biological hierarchy (e.g., sensory receptors, perception, attention, cognition) there is a breakdown that results in ASD behavioral characteristics (phenotype). Some studies point to differences in how tactile stimuli are processed at a peripheral level, namely, touch detection and stimuli discrimination, but findings are inconclusive (Riquelme et al. 2016). Others suggest differences in neurotransmitters – specifically GABAergic function (the major inhibitory neurotransmitter in the central nervous system), crucial for balance between excitatory and inhibitory tactile input at a cortical level (Tavassoli et al. 2016). A specific class of tactile mechanoreceptors, C-tactile (CT) neurons, involved in encoding the valence/pleasantness of a stimulus (Loken et al. 2009), have been hypothesized to underlie the atypical responses in ASD. By encoding the valence of a stimulus, these fibers are intrinsically related to social cognition and our ability to bond with others. Children with ASD present more aversive reactions to touch delivered to CT-innervated body regions, such as the face and arm, compared to non-CT-innervated regions, such as the palm of the hand (Cascio et al. 2016). Moreover, at a brain level, children with ASD show diminished responses in social-affective brain networks compared to typically developing children following CT-targeted touch stimulation (Kaiser et al. 2016).

Touch processing seems to be altered in ASD; however, how heterogeneous behavioral responses to touch are related to social-communication problems in ASD is under deliberation. Atypical touch processing, whether characterized as hyporesponsiveness, sensory seeking, or hyperresponsiveness, can result in reduced exposure to important stimuli causing an altered trajectory of the developing social brain in infancy. Altered tactile processing emerges before many of the social and communicative impairments associated with ASD and has strong theoretical grounding as a developmental precursor to social-communicative behavior. By understanding the behaviors and mechanisms that underlie atypical touch processing, we can design and improve diagnostic and treatment tools for these individuals.

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References and Reading

- APA. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- Baranek, G. T., Boyd, B. A., Poe, M. D., David, F. J., & Watson, L. R. (2007). Hyperresponsive sensory patterns in young children with autism, developmental delay, and typical development. *American Journal of Mental Retardation*, 112(4), 233–245. [https://doi.org/10.1352/0895-8017\(2007\)112\[233:HSPIYC\]2.0.CO;2](https://doi.org/10.1352/0895-8017(2007)112[233:HSPIYC]2.0.CO;2).
- Cascio, C. J., Gu, C., Schauder, K. B., Key, A. P., & Yoder, P. (2015). Somatosensory event-related potentials and association with tactile behavioral responsiveness patterns in children with ASD. *Brain Topography*, 28(6), 895–903. <https://doi.org/10.1007/s10548-015-0439-1>.
- Cascio, C. J., Lorenzi, J., & Baranek, G. T. (2016). Self-reported pleasantness ratings and examiner-coded defensiveness in response to touch in children with ASD: Effects of stimulus material and bodily location. *Journal of Autism and Developmental Disorders*, 46(5), 1528–1537. <https://doi.org/10.1007/s10803-013-1961-1>.
- Foss-Feig, J. H., Heacock, J. L., & Cascio, C. J. (2012). Tactile responsiveness patterns and their association with Core features in autism Spectrum disorders. *Research in Autism Spectrum Disorder*, 6(1), 337–344. <https://doi.org/10.1016/j.rasd.2011.06.007>.
- Gallace, A., & Spence, C. (2010). The science of interpersonal touch: An overview. *Neuroscience and Biobehavioral Reviews*, 34(2), 246–259. <https://doi.org/10.1016/j.neubiorev.2008.10.004>.
- Hertenstein, M. J., Verkamp, J. M., Kerestes, A. M., & Holmes, R. M. (2006). The communicative functions of touch in humans, nonhuman primates, and rats: A review and synthesis of the empirical research. *Genetic, Social, and General Psychology Monographs*, 132(1), 5–94. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/17345871>.
- Hilton, C. L., Harper, J. D., Kueker, R. H., Lang, A. R., Abbacchi, A. M., Todorov, A., & LaVesser, P. D. (2010). Sensory responsiveness as a predictor of social severity in children with high functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(8), 937–945. <https://doi.org/10.1007/s10803-010-0944-8>.
- Kadlaskar, G., Seidl, A., Tager-Flusberg, H., Nelson, C. A., & Keehn, B. (2019). Atypical response to caregiver touch in infants at high risk for autism Spectrum disorder. *Journal of Autism and Developmental Disorders*, 49(7), 2946–2955. <https://doi.org/10.1007/s10803-019-04021-0>.
- Kaiser, M. D., Yang, D. Y., Voos, A. C., Bennett, R. H., Gordon, I., Pretzsch, C., et al. (2016). Brain mechanisms for processing affective (and nonaffective) touch are atypical in autism. *Cerebral Cortex*, 26(6), 2705–2714. <https://doi.org/10.1093/cercor/bhv125>.
- Lane, A. E., Young, R. L., Baker, A. E., & Angley, M. T. (2010). Sensory processing subtypes in autism: Association with adaptive behavior. *Journal of Autism and Developmental Disorders*, 40(1), 112–122. <https://doi.org/10.1007/s10803-009-0840-2>.
- Loken, L. S., Wessberg, J., Morrison, I., McGlone, F., & Olausson, H. (2009). Coding of pleasant touch by unmyelinated afferents in humans. *Nature Neuroscience*, 12(5), 547–548. <https://doi.org/10.1038/nn.2312>.
- Miguel, H. O., Sampaio, A., Martinez-Regueiro, R., Gomez-Guerrero, L., Lopez-Doriga, C. G., Gomez, S., et al. (2017). Touch processing and social behavior in ASD. *Journal of Autism and Developmental Disorders*, 47(8), 2425–2433. <https://doi.org/10.1007/s10803-017-3163-8>.
- Morrison, I., Loken, L. S., & Olausson, H. (2010). The skin as a social organ. *Experimental Brain Research*, 204(3), 305–314. <https://doi.org/10.1007/s00221-009-2007-y>.
- Riquelme, I., Hatem, S. M., & Montoya, P. (2016). Abnormal pressure pain, touch sensitivity, proprioception, and manual dexterity in children with autism Spectrum disorders. *Neural Plasticity*, 2016, 1723401. <https://doi.org/10.1155/2016/1723401>.
- Tavassoli, T., Miller, L. J., Schoen, S. A., Nielsen, D. M., & Baron-Cohen, S. (2014). Sensory over-responsivity in adults with autism spectrum conditions. *Autism*, 18(4), 428–432. <https://doi.org/10.1177/1362361313477246>.
- Tavassoli, T., Bellesheim, K., Tommerdahl, M., Holden, J. M., Kolevzon, A., & Buxbaum, J. D. (2016). Altered tactile processing in children with autism spectrum disorder. *Autism Research*, 9(6), 616–620. <https://doi.org/10.1002/aur.1563>.
- Tomchek, S. D., & Dunn, W. (2007). Sensory processing in children with and without autism: A comparative study using the short sensory profile. *The American Journal of Occupational Therapy*, 61(2), 190–200. <https://doi.org/10.5014/ajot.61.2.190>.

Touch Sensitivity

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Synonyms

Sensory integration and praxis tests (SIPT);
Sensory processing; Tactile sensitivity

Definition

Touch sensitivity, or sensitivity to tactile stimuli, has been noted in children with autism spectrum disorders (Baranek et al. 1997). Children with tactile hypersensitivity may show a reluctance to manipulate various toys, materials, or foods based on their texture or may exhibit exaggerated or persistent responses to unexpected touch occurrences and may even avoid situations in which these events or unusual textures may occur. Some children may be hypervigilant to the potential threat of unexpected touch which may ultimately compromise successful participation in a designated activity. Clothing selection, play materials, and food choices may also be restricted if they are perceived to be unpleasant to the individual. Some individuals may exhibit hyposensitivity to touch or an underreaction to touch occurrences. These individuals may display a more passive response to tactile stimuli and may not notice when clothing is twisted on their body or food is left on their face. Tool usage may also be affected as the individual may not have adequate feedback in the hand for effective manipulation.

See Also

► [Tactile Defensiveness](#)

References and Reading

- Ayres, A. (1972). *Sensory integration and learning disabilities*. Los Angeles: Western Psychological Services.
- Ayres, A. (1973). *Sensory integration and learning disorders*. Los Angeles: Western Psychological Services.
- Ayres, A. (1979). *Sensory integration and the child*. Los Angeles: Western Psychological Services.
- Ayres, A. J. (1985). *Developmental Dyspraxia and adult onset apraxia*. Torrance: Sensory Integration International.
- Baranek, G. T., Foster, L. G., & Berkson, G. (1997). Tactile defensiveness and stereotypic behaviors. *American Journal of Occupational Therapy*, 51, 91–95.
- Parham, L. D., & Mailloux, Z. (2001). Sensory integration. In J. Case-Smith (Ed.), *Occupational therapy for children* (4th ed.). St. Louis: Mosby.

Tomchek, S. D. (2010). Sensory processing in individuals with an autism spectrum disorder. In H. M. Kuhaneck & R. Watling (Eds.), *Autism: A comprehensive occupational therapy approach* (pp. 135–161). Bethesda: AOTA Press.

Tourette Disorder

► [Tourette Syndrome](#)

Tourette Syndrome

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Synonyms

[Gilles de la Tourette syndrome](#); [Tourette disorder](#); [Tourette's syndrome](#)

Short Description or Definition

Tourette syndrome is a childhood-onset neuropsychiatric disorder characterized by multiple motor and vocal tics that occur for at least a year in duration.

Categorization

Tic Disorder

Epidemiology

Transient tics, which have a duration of less than 6 months, are common in childhood. Estimates suggest that 4–24% of school-aged children experience tics (Khalifa and von Knorring 2003; Scahill et al. 2005). Roughly one-quarter of children experience chronic motor or vocal tics. Chronic tics have a duration of at least 1 year. Once thought to be much rarer, the current

lifetime prevalence estimates for Tourette syndrome ranges from 0.1% to 1%. Tic disorders are much more common in children with pervasive developmental disorders and in special education populations than would be expected by chance (Canitano and Vivanti 2007; Kurlan et al. 2001). Studies have estimated that the occurrence of Tourette syndrome may be greater than 10% among autistic children (Canitano and Vivanti 2007).

Prevalence of tic disorders peak during the late first decade and early second decades of life due to the clinical course of the disorder and are roughly one-third as prevalent in adulthood (Bloch and Leckman 2009; Bloch et al. 2006; Leckman et al. 1998). There is a male predominance of TS with boys about twice as likely to be affected by tic disorders as girls (Scahill et al. 2005).

Natural History, Prognostic Factors, and Outcomes

The onset of TS is usually characterized by the appearance of simple, transient motor tics that affect the face (typically eye blinking) around the age of 5–7 (Leckman et al. 1998). Over time, these simple motor tics generally progress in a rostrocaudal direction affecting other areas of the face, followed by the head, neck, arms, and, lastly and less frequently, the lower extremities. With time, vocal tics often appear, tics become increasingly complex, and premonitory urges appear. Premonitory urges are feelings of tightness, tension, or itching that are accompanied by a mounting sense of discomfort or anxiety that can be relieved only by the performance of a tic (Leckman et al. 1993). With increasing awareness of premonitory urges, TS patients begin to exhibit a variable degree of voluntary control over tic performance. However, this voluntary control should be likened to that governing control of eye blinking. Eye blinking and tics can both be inhibited voluntarily, but only for a limited period of time and only with mounting discomfort. Thus, some adult TS patients are able to demonstrate nearly complete control over when expression of their tics will occur. However, when complete or

near complete control of tics is present, resistance to the mounting tension of premonitory urges can produce mental and physical exhaustion even more impairing and distracting than the tics themselves.

The severity of tics in TS waxes and wanes throughout the course of the disorder (Peterson and Leckman 1998). Tics are highly variable from minute to minute, hour to hour, day to day, week to week, and month to month. Tic episodes occur in bouts, which in turn also tend to cluster. Tic symptoms, however, can be exacerbated by stress, fatigue, extremes of temperature, and external stimuli (i.e., in echolalia tics). Intentional movements attenuate tic occurrence over the affected area, and intense involvement and concentration in activities tends to dissipate tic symptoms.

TS symptoms generally peak in severity between the ages of ten and twelve. Tic severity typically begins to decrease with the onset of adolescence (Bloch et al. 2006; Leckman et al. 1998). Reduction in tic severity generally ends by the early twenties. Although a small minority of TS patients does experience catastrophic outcomes in adulthood, on the whole, individuals rarely experience either a sustained worsening or improvement of their symptoms after their mid-20s. One-half to two-thirds of individuals with TS experience a marked reduction of symptoms by their late teens and early twenties, with one-third to one-half of these patients becoming virtually asymptomatic in adulthood.

Evaluation and Differential Diagnosis

A diagnosis of Tourette syndrome or chronic tic disorders is made through clinical history and observation. A childhood onset, the waxing-and-waning severity, and changing nature of tic symptoms are critical in establishing the diagnosis. In older children, the presence of premonitory urges and the exacerbation of tics in response to stress and fatigue are also helpful clues in establishing the diagnosis. In children, tics often need to be distinguished from stereotypies of stereotyped movement disorders or pervasive developmental disorders. Stereotypies typically have an earlier

age of onset than tics, are bilateral rather than unilateral, and have a soothing quality. Table 1 contrasts the attributes of tics and stereotypies. Complex tics often also need to be distinguished from compulsions of obsessive-compulsive disorder. Making this particularly difficult is the high comorbidity between the two conditions. Compulsions are usually performed in response to an obsession and preceded by anxiety, worry, or concern, whereas tics are generally performed in response to a physical sensation or premonitory urges. Compulsions typically are more elaborate than tics and are more likely to resemble “normal” behavior. Often, both a diagnosis of OCD and a tic disorder are warranted in the same individual.

The tics, which are the most prominent feature of TS, are often neither the first nor the most impairing psychological disturbance TS patients endure. Thus, a thorough evaluation for common comorbid conditions is also critical (Scahill et al. 2006). In screening for comorbidities in TS, ADHD, OCD, disruptive behaviors, and pervasive developmental disorders are of principle importance. Children with TS have higher rates of OCD, ADHD, PDD, and disinhibited speech and behavior compared to the general population. In the natural course of comorbid psychiatric illness in TS, ADHD symptoms, when they occur typically, precede the onset of tic symptoms by a couple of years, whereas OC symptoms typically present around the age of 12–13 after tics have reached their peak severity (Bloch et al. 2006). Approximately half of children with Tourette syndrome experience comorbid ADHD and an even

greater proportion of children with tic disorders reaching clinical attention. Roughly one-third to one-half of Tourette syndrome patients will experience clinically significant OCD symptoms during the course of their lifetime.

Educating the patient, his family members, teachers, and peers is among the most important interventions available to the clinician. It should be undertaken for nearly all patients with tic disorders. Family psychoeducation should focus on:

1. The tics are not voluntary or meant to be intentionally provocative. They typically occur in “bouts” when tics will appear in rapid succession followed by a tic-free interval.
2. Premonitory urges – the physical sensations like an itch or before a sneeze that many patients experience prior to performing tics. TS is a sensorimotor disorder. There is also a momentary sense of relief that follows the completion of the tic.
3. Suppressibility of tics – many patients are able to suppress their tics over the short term, but often this is at the expense of increasing discomfort and distraction due to the premonitory urges. So it is not uncommon for a child to have relatively minor tics at school and then return home to let loose a bout of tics.
4. The natural waxing-and-waning course of tic symptoms – that there will be increases and decreases in tic severity over time. Due to the natural ebb and flow of symptoms in the illness, any interventions started during an

Tourette Syndrome, Table 1 Comparison between tics and stereotypies

	Tics	Stereotypies
Typical age of onset	4–8 Years	2–3 Years
Course of symptoms	Waxing and waning	Constant
Timing of movements	Brief and sudden	Continuous, rhythmic, and prolonged
Typical movements	Eye blinking, facial grimace, throat clearing	Arm flapping, rocking
Characteristics of movements	Typically unilateral	Often bilateral
Exacerbated by. . .	Stress, fatigue	Excitement
Premonitory urges	Present	Absent
Suppressibility	Often for short periods of time	Rare
Comorbid conditions	OCD, ADHD	PDD, autism spectrum disorders
Treatment	Neuroleptic, $\alpha 2$ agonists	No response to medication

exacerbation of tic severity may appear successful due to a natural decline in symptom severity.

5. The natural history of tic disorders – the illness tends not to be relentlessly progressive and symptoms usually improve by adulthood. This information often contradicts the impressions gained from the available lay literature on TS that typically focuses on the most extreme cases – which do typically occur in adulthood.
6. Exacerbating and alleviating factors – tics tend to be exacerbated by fatigue, sleeplessness, stress, excitement, and possibly changes in temperature and streptococcal infections. Tics often tend to be alleviated when a child is deeply engaged in a motor activity such as sports, playing musical instruments, and dancing. The exact exacerbating and alleviating factors are highly individualized to the patient. Healthy habits such as good sleep hygiene and regular exercise can only improve tic symptoms.
7. Education on comorbid ADHD, OCD, learning disabilities, and disruptive or disinhibited behaviors when present. Improving these disorders often leads to an improvement in the tics.
8. Provide resources to help educate parents and teachers about tic disorders. These are available at the Tourette Syndrome Association website: <http://www.tsa-usa.org> and <http://www.tourettesyndrome.net>.

Treatment

Given the waxing and waning course of tics and the decline in tic severity that usually occurs in adolescence, any intervention performed in response to symptom exacerbation will usually be followed by clinical improvement. The goal of treatment should be minimizing the level of social and educational impairment caused by tics rather than completely eliminating tics, which will be unsuccessful in the majority of cases. Many cases of TS can be successfully managed without medication. When coexisting conditions such as

ADHD, OCD, depression, or ODD are present, it is usually better to treat these “comorbid” conditions first, as successful treatment of these disorders often will diminish tic severity (Scahill et al. 2006).

Pharmacologic Interventions

Antipsychotic medications, dopamine D₂ receptor antagonists, are the most effective tic-suppressing medications (Scahill et al. 2006; Singer 2010). Among the typical antipsychotics, haloperidol and pimozide have the most studied and have the most convincing data demonstrating efficacy. Although antipsychotics have the greatest efficacy in treating tics, they are generally not recommended as first-line treatment because of poor tolerability. Common potential side effects include tardive dyskinesia, acute dystonic reactions, sedation, depression, school and social phobias, and/or weight gain. In many instances by starting at low doses and adjusting the dosage upward slowly, clinicians can avoid these side effects. The goal should be to use as little of these medications as possible to render the tics “tolerable.” Efforts to stop the tics completely often risk overmedication and increased side effects.

Due to the extrapyramidal side effects associated with typical antipsychotics, atypical antipsychotics, such as risperidone, olanzapine, and ziprasidone, are now the most widely used medications to treat tic symptoms. These agents have potent 5-HT₂ blocking effects as well as more modest blocking effects on dopamine D₂. There are currently double-blind clinical trials that have supported the efficacy of risperidone, olanzapine, and ziprasidone (Singer 2010). Atypical antipsychotics, especially olanzapine and risperidone, are associated with significant weight gain and sedation and increased risk of diabetes. Ziprasidone use can be associated with QT prolongation in children; so serial monitoring with electrocardiograms may be necessary. Due to the significant metabolic side effects associated with atypical antipsychotics, even though they are more effective at treating tics than other classes of medications, they are not recommended as a first-line intervention.

Clonidine and guanfacine are potent α_2 receptor agonists that are thought to reduce central noradrenergic activity. Although less effective in relieving tics compared to the antipsychotic medication, α_2 receptor agonists have the advantage of being better tolerated and also improving comorbid ADHD symptoms in patients with tics (Scahill et al. 2006; Singer 2010). The principal side effect associated with clonidine use is sedation which occurs in 10–20% of subjects and which usually abates with continued use. Other side effects include dry mouth, transient hypotension, and rare episodes of worsening behavior. Clonidine should be tapered and not withdrawn abruptly, to reduce the likelihood of symptom or blood pressure rebound. Guanfacine is generally preferred to clonidine because it is less sedating and not associated with rebound hypertension following withdrawal.

When comorbid ADHD is the most impairing illness in a patient presenting with comorbid tics, the stimulants methylphenidate and dextroamphetamine derivatives are still first-line agents for the medical management of ADHD. Although clinical trials of meta-analysis of psychostimulant use in children with ADHD and comorbid tics suggest that these medications do not worsen tic severity, there still exists an FDA warning against the use of psychostimulants in children with tic disorders or a family history of TS (Bloch et al. 2009). This FDA warning was exclusively based on data from clinical case reports and case series that reported worsening of tics when children were exposed to psychostimulants. For this reason, non-stimulant treatments are often used in the treatment of children with ADHD and comorbid tics. α_2 receptor agonists have the advantage of demonstrated efficacy for both ADHD and tic symptoms. Furthermore, the combination of clonidine and methylphenidate has been demonstrated to be more effective in treating ADHD symptoms in children with comorbid tics than either medication alone. Atomoxetine has also been demonstrated to be effective in the treatment of ADHD symptoms in children with comorbid tics and may also be modestly helpful in the treatment of tics.

Behavioral

Behavioral therapy has shown significant promise in the treatment of tic disorders. Behavioral therapy has been demonstrated to be significantly more effective than supportive therapy in reducing tic severity in children with TS in a large, randomized, multicenter trial using blinded raters (Piacentini et al. 2010). Behavioral therapy has also been shown to significantly reduce tic symptoms in adults with TS when compared to supportive therapy in randomized and unblinded trials (Deckersbach et al. 2005; Wilhelm et al. 2003). The measured effects of behavioral therapy in reducing tic severity have been similar to the most effective pharmacological agents used to treat tics.

Behavioral therapy for tics consists of two main components: (1) awareness training and (2) competing response practice (Woods 2001). Awareness training consists of 4 components designed to increase an individual's awareness of his own tics. These components include the following: (1) response description, in which the patient learns how to describe tic movements and reenacts them into a mirror; (2) response detection, in which the therapist aids the patient in tic detection by pointing out each tic immediately after it occurs in the session; (3) early warning procedure, in which an individual learns how to identify the earliest signs of tic occurrence; and (4) situational awareness training, in which an analysis is conducted to identify the high-risk situations where tics are most likely to occur. Competing response practice involves teaching individuals to produce an incompatible physical response (i.e., isometric contraction of tic-opposing muscles) contingent upon the urge to perform a tic.

Although behavioral therapy has significant evidence of efficacy and does not have the side effect burden of the medications used to treat tics, dissemination remains a significant barrier to the widespread implementation of this treatment for tics. Furthermore, the efficacy of behavioral therapy has not been demonstrated in special populations of children with tic disorders such as those with comorbid ADHD, pervasive developmental disorders, or intellectual disability.

See Also

- ▶ [Attention Deficit/Hyperactivity Disorder](#)
- ▶ [Obsessive-Compulsive Disorder \(OCD\)](#)

References and Reading

- Bloch, M. H., & Leckman, J. F. (2009). Clinical course of Tourette syndrome. *Journal of Psychosomatic Research*, 67(6), 497–501.
- Bloch, M. H., Peterson, B. S., Scahill, L., Otko, J., Katsoyich, L., Zhang, H., et al. (2006). Adulthood outcome of tic and obsessive-compulsive symptom severity in children with Tourette syndrome. *Archives of Pediatrics & Adolescent Medicine*, 160(1), 65–69.
- Bloch, M. H., Panza, K. E., Landeros-Weisenberger, A., & Leckman, J. F. (2009). Meta-analysis: Treatment of attention-deficit/hyperactivity disorder in children with comorbid tic disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(9), 884–893.
- Canitano, R., & Vivanti, G. (2007). Tics and Tourette syndrome in autism spectrum disorders. *Autism*, 11(1), 19–28.
- Deckersbach, T., Rauch, S., Buhlmann, U., & Wilhelm, S. (2005). Habit reversal versus supportive psychotherapy in Tourette's disorder: A randomized controlled trial and predictors of treatment response. *Behaviour Research and Therapy*, 44(8), 1079–1090.
- Khalifa, N., & von Knorring, A. L. (2003). Prevalence of tic disorders and Tourette syndrome in a Swedish school population. *Developmental Medicine and Child Neurology*, 45(5), 315–319.
- Kurlan, R., McDermott, M. P., Deeley, C., Como, P. G., Brower, C., Eapen, S., et al. (2001). Prevalence of tics in schoolchildren and association with placement in special education. *Neurology*, 57(8), 1383–1388.
- Leckman, J. F., Walker, D. E., & Cohen, D. J. (1993). Premonitory urges in Tourette's syndrome. *The American Journal of Psychiatry*, 150(1), 98–102.
- Leckman, J. F., Zhang, H., Vitale, A., Lahnin, F., Lynch, K., Bondi, C., et al. (1998). Course of tic severity in Tourette syndrome: The first two decades. *Pediatrics*, 102(1 Pt 1), 14–19.
- Peterson, B. S., & Leckman, J. F. (1998). The temporal dynamics of tics in Gilles de la Tourette syndrome. *Biological Psychiatry*, 44(12), 1337–1348.
- Piacentini, J., Woods, D. W., Scahill, L., Wilhelm, S., Peterson, A. L., Chang, S., et al. (2010). Behavior therapy for children with Tourette disorder: A randomized controlled trial. *Journal of the American Medical Association*, 303(19), 1929–1937.
- Scahill, L., Sukhodolsky, D. G., Williams, S. K., & Leckman, J. F. (2005). Public health significance of tic disorders in children and adolescents. *Advances in Neurology*, 96, 240–248.
- Scahill, L., Erenberg, G., Berlin, C. M., Jr., Budman, C., Coffey, B. J., Jankovic, J., et al. (2006). Contemporary assessment and pharmacotherapy of Tourette syndrome. *NeuroRx*, 3(2), 192–206.
- Singer, H. S. (2010). Treatment of tics and Tourette syndrome. *Current Treatment Options in Neurology*, 12(6), 539–561.
- Wilhelm, S., Deckersbach, T., Coffey, B. J., Bohné, A., Peterson, A. L., & Baer, L. (2003). Habit reversal versus supportive psychotherapy for Tourette's disorder: A randomized controlled trial. *The American Journal of Psychiatry*, 160(6), 1175–1177.
- Woods, D. W. (2001). Habit reversal treatment manual for tic disorders. In D. W. Woods & R. M. Miltenberger (Eds.), *Tic disorders, Trichotillomania, and other repetitive behavior disorders: Behavioral approaches to analysis and treatment* (pp. 97–132). Boston: Kluwer Academic Publishers.

Tourette's Syndrome

- ▶ [Tourette Syndrome](#)

Toxicology

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Synonyms

Teratology

Definition

Toxicology is the branch of science that studies the adverse effects of chemicals and environmental exposures on living organisms. It includes aspects of biology, chemistry, and medicine in the scientific approaches used. It studies the symptoms, mechanisms, measurement, and treatment of poisonous substances that people and other living things are exposed to.

The science of toxicology identifies chemical compounds and environmental events that

negatively impact health. It was initially considered as the study of poisons and has been expanded with the technological impact on the environment. The study of dose and effect is central to toxicology. Many compounds, such as lead, are toxic or harmful above certain levels of exposure. The risk for injury increases with the level of exposure. Toxicity refers to the biologic effect of a substrate or compound. The Society of Toxicology defines toxicology as “the study of the adverse effects of chemical, physical, or biologic agents on living organisms and the ecosystem, including the prevention and amelioration of such adverse effects.” Toxicologists are important to the study of the potential role of exposures to environmental agents such as air pollution and maternal use of medication such as selective serotonin reuptake inhibitors during brain development.

The scientists who study toxicology approach question regarding the safety of compounds and events living things are exposed to from the fields of biology, chemistry, medicine, and environmental science, among others. Neurotoxicologists study the effects of chemicals and environmental events on the brain and brain development.

See Also

- ▶ [Metallothionein](#)
- ▶ [Thalidomide](#)

References and Reading

- Halladay, A. K., Amaral, D., Ascher, M., Bolvar, V. J., Bowman, A., DiCicco, B. E., et al. (2009). Animal models of autism spectrum disorder: Information for neurotoxicologists. *Neurotoxicology*, 30(5), 811–821.
- Kaplan, Y. C., Keskin-Arslan, E., Acar, S., & Sozmen, K. (2017). Maternal SSRI discontinuation, use, psychiatric disorder and the risk of autism in children: a meta-analysis of cohort studies. *British Journal of Clinical Pharmacology*, 83(12), 2798. PMID:28734011.
- Lam, J., Sutton, P., Kalkbrenner, A., Windham, G., Halladay, A., Koustas, E., Lawler, C., Davidson, L., Daniels, N., Newschaffer, C., & Woodruff, T. (2016). A systematic review and meta-analysis of multiple airborne pollutants and autism Spectrum disorder. *PLoS One*, 11(9), e0161851. <https://doi.org/10.1371/journal.pone.0161851>. PMID:27653281.
- Society for Toxicology. www.toxicology.org. Accessed 24 Sept 2017.
- Winneke, G. (2011). Developmental aspects of environmental neurotoxicology: Lessons from lead and polychlorinated biphenyls. *Journal of the Neurological Sciences*, 308(1–2), 9–15.

Train-and-Hope Strategy

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Definition

Intervention goals for individuals with autism spectrum disorders (ASD) often include increasing the use of one or more target skills within the treatment sessions. Moreover, comprehensive interventions include strategies to support the generalization of the new target skills. Generalization is the ability to use newly acquired skills with other materials, people (e.g., family members, teachers, peers), and settings (e.g., home, work, school, recess), that differ from the treatment sessions. The phrase “*train and hope*” was identified in Stokes and Baer’s (1977) classic review of treatment generalization strategies. “*Train and hope*” refers to teaching individuals a desired skill within a treatment session and hoping that the individual will generalize the use of that skill without implementation of a predetermined plan or strategy to facilitate generalization. Instead of merely “*hoping*” for generalization, Stokes and Baer (1977) recommended implementation of explicit and systematic strategies to promote generalization. These strategies include using sequential modification, training sufficient exemplars, programming common stimuli, using natural maintaining contingencies, training loosely, using indiscriminable contingencies, mediating generalization by teaching individuals to monitor their own performance, and training to generalize. Please see generalization and maintenance for an explanation of these generalization strategies.

See Also

► Generalization and Maintenance

References and Reading

- Kashinath, S., Woods, J., & Goldstein, H. (2006). Enhancing generalized teaching strategy use in daily routines by parents of children with autism. *Journal of Speech, Language & Hearing Research*, 49(3), 466–485. [https://doi.org/10.1044/1092-4388\(2006/036](https://doi.org/10.1044/1092-4388(2006/036)
- Stokes, T., & Baer, D. (1977). An implicit technology of generalization. *Journal of Applied Behavior Analysis*, 10, 349–367.
- Strain, P., Schwartz, I., & Bovey, E. (2007). Social skills intervention for young children with autism: Programmatic research findings and implementation issues. In W. H. Brown, S. L. Odom, & S. R. McConnell (Eds.), *Social competence in young children* (pp. 253–272). Baltimore: Paul H. Brooks Publishing.
- Whalen, C. (Ed.). (2009). *Real life, real progress for children with autism spectrum disorders: Strategies for successful generalization in natural environments*. Baltimore: Paul H. Brooks Publishing.

Trainer Implementation

► Treatment Fidelity

Training Parents of Youth with Autism to Advocate for Adult Disability Services

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Definition

Parents of individuals with autism spectrum disorder (ASD) often advocate throughout their lifespans on behalf of their offspring. An advocate is “someone who acts on someone’s behalf or for

their best interest” (Alper et al. 1995). With respect to individuals with ASD, parent advocacy can take on many forms ranging from advocating to access and obtain individual services to advocating for systemic changes to a service delivery system. With respect to adult services for individuals with ASD, parents’ knowledge about the adult service delivery system, feelings of empowerment, and skills to identify, access, and receive needed services can result in effective parent advocacy.

Historical Background

Upon aging out of special education services, individuals with ASD shift from an entitlement driven system (i.e., special education services) to an eligibility driven system (i.e., adult services). Although adult services exist, after exiting special education, many adults with ASD lack needed services (Shattuck et al. 2011). Without services, individuals with developmental disabilities, including ASD, may make little progress (Taylor and Hodapp 2012). Indeed, youth with ASD may go for years without needed services and rely on family to provide support.

Barriers to Receiving Adult Services for Youth with ASD

Although there are many barriers to accessing adult services, for brevity, only three barriers will be listed here. First, the adult service delivery system is fragmented. Comprised of a variety of agencies, services, and funding sources, the adult service delivery system stretches across multiple domains including but not limited to employment, health, leisure and recreation, housing or community living, financial assistance, and education. Different policies dictate regulations for certain adult services. Health services, for example, are (in part) outlined by the Patient Protection and Affordable Care Act while community living and housing services are (in part) regulated by Section 8 of the Housing Act of 1937. Further, in addition to having a variety of policies relate to different services, some policies overlap. For example, agencies that receive any federal

funding must comply with Section 504 of the American Rehabilitation Act, which provides accommodations and services to individuals with disabilities, including ASD. Such agencies could include post-secondary education institutions, places of employment, and leisure and recreational facilities as long as they receive some federal funding. Due to the fragmented nature of the service delivery system including the different policies and regulations, parents struggle to access and obtain services for their offspring with ASD.

Second, in addition to being fragmented, adult services are also complicated because, in some instances, certain policies and services impact one's eligibility and reception of other services. For example, in a given state, the Division of Vocational Rehabilitative Services generally helps individuals with disabilities, including ASD, find, secure, and maintain employment. Unfortunately, many states have waiting lists for Vocational Rehabilitative Services. Often, in these waiting lists, individuals with disabilities are prioritized by their level of needs. In some states, the level of need is, in part, determined by whether the individual is receiving supplemental security income (SSI). If the individual is receiving SSI, then the individual has a higher priority on the waiting list. Parents would have to learn how to navigate the SSI regulations to access Vocational Rehabilitative services. Thus, the onus is often put on parents to understand the rules of different service delivery systems as well as how the systems relate to one another.

Third and finally, adult services are difficult to navigate and receive because they are often eligibility driven. Parents of youth with ASD may have grown accustomed to their offspring being entitled to receive special education services, which are federally required regardless of the cost to the school. Adult services, however, are eligibility driven, meaning youth with ASD must qualify to receive services.

For example, youth with ASD may be eligible to receive a Home and Community Based Services (HCBS) Medicaid Waiver – this waiver reimburses states for providing supports including employment, transportation, and therapies. One issue with the HCBS Medicaid Waiver is that

43 out of 50 states have waiting lists for this waiver (Research and Training Center on Community Living 2013). Depending on the state, it could take years before a youth with ASD is eligible to receive a Medicaid Waiver. Further, eligibility for an HCBS waiver varies by state. In some states, an individual must have an intellectual disability to be eligible for the waiver, whereas other states require individuals to have a developmental disability (i.e., eligibility is not limited to an intellectual disability) to be eligible for the waiver. Thus, depending on the state, individuals with ASD may not even be eligible for the waiver. This issue of varying eligibility can lead to inter- and intrastate variation. Specifically, individuals with ASD may be “locked in” to residing in states where they are eligible for Medicaid waiver services feeling they are unable to move to a state wherein they would not be eligible.

Need for Parent Advocacy

Given that the onus is on parents to identify, access, and receive adult services for their offspring with ASD, parents need advocacy skills. When parents are knowledgeable and advocate for their children, parents report that transition planning and the subsequent shift to adult services is more seamless (Timmons et al. 2004). Yet, many parents of individuals with developmental disabilities, including ASD, report struggling to advocate and to understand the adult service delivery system (Hetherington et al. 2010). As such, there is a strong need for parent advocacy training interventions about adult services. Although the research about parent advocacy is scant, the limited extant research indicates that parent advocacy interventions lead to increases in knowledge and perceived advocacy skills to navigate service delivery systems (Burke et al. 2016).

Current Knowledge

Because the systems of services and supports for individuals with ASD who are moving from high school to adult life are very complex, it is important to understand what choices are available and

how enrollment in some services may affect access to other services. To help in the decision-making process for parents of young adults with ASD in Tennessee, a comprehensive 12-week parent advocacy curriculum, entitled the Volunteer Advocacy Program – Transition (VAP-T), is used to train parents. Funding for this work was received from the National Institute of Mental Health. The training provides information on various services and post-secondary, advocacy tips for parents of young adults with ASD, as well as a Letter of Intent form.

The Letter of Intent is not a legal document; it is a record of information about the person with a disability. This includes family information and a record of the other topics found in the VAP-T curriculum. Included in this document would be: the services and programs that have been applied for in the past, the outcome of those applications, and the programs and services that are currently in place. The information found in a Letter of Intent is important for anyone supporting a family member throughout his or her life-course. The document helps families plan and assess life-goals from year to year. Parents in the VAP-T are encouraged to complete a couple of pages of the Letter of Intent each week that coordinates with the topic covered. Notably, the Letter of Intent should be updated as changes occur and, otherwise, at least annually.

The 12 sessions in the VAP-T curriculum are 2.5 h each in consecutive weeks and are held in the evening to accommodate parents who often have active careers in this stage of family life. Because the sessions are offered during the dinner hour, a light meal or snack is provided. Each week's agenda includes PowerPoint presentations by subject matter experts, activities for participants, time for discussion among participants across three sites, and resources specific to the topic covered and the physical location of participants. Participants and facilitators in all locations received a notebook containing all materials, activity sheets, and resources.

The group facilitator and subject matter experts are present with a group at the host site. In collaboration with distance sites, the training is provided using video conferencing, so that two

other groups of parents can join remotely. Additionally, if participants cannot attend at the host or remote sites then, on occasion, they can join from a home computer. All sessions are video recorded for later viewing if a participant misses a session. Support to the remote sites includes a meeting before the start of the training to go over the curriculum, group facilitation techniques, and technology.

The 12-week curriculum includes the following topics:

1. Introduction to the Project – Syllabus, Speaker Information, Acronyms Guide, Letter of Intent
2. Person-Centered Thinking and Planning
3. Secondary Education
4. Supplemental Security Income (SSI)
5. Social Security Disability Income (SSDI) & Family Support Program
6. Employment 1 – Types, Training, Ticket to Work
7. Employment 2 – Vocational Rehab and the Workforce Innovation and Opportunity Act (WIOA)
8. Post-Secondary Education
9. Employment and Community First Program (i.e., a Medicaid Waiver Program)
10. Conservatorship & Special Needs Trusts, Achieving a Better Life Experience Act of 2014 (ABLE)
11. TennCare (Medicaid) and Medicare
12. Housing and Non-Adversarial Advocacy

During the development of the VAP-T curriculum, an advisory board consisting of parents, self-advocates, and community partners provided input and feedback on the syllabus and course content. The initial curriculum was provided to a pilot group of parents of adults with ASD at the host location only. Pilot group participants were individuals invited by members of the team and colleagues. Based on feedback of the pilot group, the curriculum was shortened, and then delivered it to a newly recruited group of participants that included the host and distance sites.

It is necessary to update the curriculum each time it is delivered, to reflect all policy changes,

eligibility changes, new programs, etc. It is also necessary to plan months in advance with speakers to fit into busy schedules and to identify new subject matter experts as people change positions or as new topics are added for new programs in the state.

Goals of the curriculum are that parents gain knowledge about services as well as gain greater confidence as advocates for their sons and daughters. Feedback from evaluations confirmed that those goals were met, even with parents who are highly engaged in the disability community and who were knowledgeable on various parts of the curriculum before participating in the training (Taylor et al. 2017). Additionally, families valued the opportunity to have discussions with representatives of local disability organizations and programs and to have their contact information. And finally, participants found value in getting to know other parents who are in various places along their journeys with their sons and daughters, learning from one another, and forming new relationships that could last beyond the group. This is an especially valuable outcome, since parents often feel isolated after their children leave the school system that had, for 12 plus years, put them in naturally occurring contact with other parents.

One of the biggest challenges is making this curriculum accessible to larger numbers of parents of transition-age sons and daughters with ASD. For example, a 2.5 h weekly commitment to attend a training session is not possible for some people. Also, some parents may not live close enough to join the in-person training or have broadband internet access to the training. In addition, the curriculum as written is specific to Tennessee. Some programs and services such as eligibility for SSI are the same in all states. However, some programs are specific to a given state such as how Medicaid dollars are allocated in programs that support people in the community, high school transition programs, and local post-secondary college programs, as well as others. These challenges make it difficult to determine how to scale-up the project to reach other families of individuals with ASD.

Future Directions

There are several next steps. One next step is to develop a curriculum that can be adopted nationally, state-by-state, in a format that provides flexibility in the way information is delivered. In this new curriculum, there will be video pre-recorded sessions that contain basic information about systems and services for each transition topic. This general information on each topic in the curriculum will include program and service descriptions and the information that is applicable in any state but would not contain state specific information. Parents will be able to view these recorded sessions on one topic each week. They can be viewed on-line, or the recordings can be shown to a group, in-person. An alternative format would be to provide a DVD of the presentations to individuals. All participants will receive course materials by email or in print.

At individual sites, the facilitators will identify local and state-wide subject matter experts to supplement the basic information. The experts may be from disability organizations and agencies; they would present on programs in their specific state. This type of presentation would follow the viewing of the basic information on the weekly topic. Facilitators will have the flexibility to offer the supplemental material in a variety of ways: to groups that are physically present; to groups who join remotely; or to individual participants that join from remote locations in an on-line classroom style. Each of these delivery methods provides the supportive benefit of the group experience.

In addition to offering access to the instructional materials in a variety of formats, future iterations of the curriculum should be adapted to be culturally responsive and available to diverse families. Although parent advocacy training interventions are needed for all families, these interventions may be especially needed for culturally diverse families as (compared to White families) they receive significantly less services (Magaña et al. 2013).

Collaboration with multicultural community programs and feedback from families of diverse backgrounds will be important in the development and delivery of culturally responsive training material.

Finally, a future step is to gather further data to show the value of providing this type of training to parents of youth with ASD. Having an evidence-based intervention like the VAP-T increases the likelihood that disability organizations and other partnering community organizations would be motivated to make this training available to parents. To increase capacity of parent advocates, a Train-The-Trainer (TTT) curriculum could be developed and technical assistance could be offered to support community investment in this type of project for parents of youth with ASD.

See Also

- [Asperger Syndrome Training & Employment Partnership \(ASTEP\)](#)
- [College Transitional Programs](#)
- [Estate Planning](#)
- [Higher Education Opportunity Act of 2008 \(HEOA\)](#)
- [Interventions to Support Transition to Adulthood for Individuals with Autism Spectrum Disorder](#)
- [US Social Policy and Autism Spectrum Disorders](#)

References and Reading

- Alper, S., Schloss, P. J., & Schloss, C. N. (1995). Families of children with disabilities in elementary and middle school: Advocacy model and strategies. *Exceptional Children, 62*, 261–270.
- Burke, M. M., Goldman, S. E., Hart, M., & Hodapp, R. M. (2016). Evaluating the efficacy of a special education advocacy training program. *Journal of Policy and Practice in Intellectual Disabilities, 13*, 269–276.
- Hetherington, S. A., Durant-Jones, L., Johnson, K., Nolan, K., Smith, E., Taylor-Brown, S., & Tuttle, J. (2010). The lived experiences of adolescents with disabilities and their parents in transition planning. *Focus on Autism and Other Developmental Disabilities, 25*, 163–172.
- Magaña, S., Lopez, K., Aguinaga, A., & Morton, H. (2013). Access to diagnosis and treatment services among Latino children with autism spectrum disorders. *Intellectual and Developmental Disabilities, 51*, 141–153.
- Research and Training Center on Community Living. (2013). Residential services for persons with intellectual or developmental disabilities: Status and trends through 2011. Available at <https://rtc3.umn.edu/risp/docs/risp2011.pdf>
- Shattuck, P. T., Wagner, M., Narendorf, S., Sterzing, P., & Hensley, M. (2011). Post-high school service use among young adults with an autism spectrum disorder. *Archives of Pediatrics and Adolescent Medicine, 165*, 141–146.
- Taylor, J. L., & Hodapp, R. M. (2012). Doing nothing: Adults with disabilities with no daily activities and their siblings. *American Journal on Intellectual and Developmental Disabilities, 117*, 67–79.
- Taylor, J. L., Hodapp, R. M., Burke, M. M., Rabideau, C., & Waitz-Kudla, S. N. (2017). Training parents of youth with autism spectrum disorders to advocate for adult disability services: Results from a pilot randomized controlled trial. *Journal of Autism and Developmental Disorders, 47*, 846–857.
- Timmons, J. C., Butterworth, J., Whitney-Thomas, J., Allen, D., & McIntyre, J. P. (2004). Managing service delivery systems and the role of parents during their children's transitions. *Journal of Rehabilitation, 70*, 19–26.

Training the General Case

- [General Case Programming \(GCP\)](#)

Transactional Support (SCERTS) Model

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Definition

Social Communication Emotional Regulation Transactional Support (SCERTS) is an innovative educational model developed by Prizant et al. (1993). It is a lifespan model that is designed to help a child become a competent and confident social communicator, while reducing/preventing problematic behaviors that interfere with learning and the development of relationships. This comprehensive intervention approach is dedicated to facilitating the socioemotional and communication abilities of children with ASD in the context

of the home and school (Prizant et al. 2003; Prizant et al. 2004).

Historical Background

The SCERTS model was developed over two decades of clinical and empirical work and is consistent with recommended evidence-based practice (Prizant et al. 2003). The social-pragmatic framework of the model has been a focus of the researchers for several years. The model was developed in response to feedback/encouragement from clinicians and researchers in the field of ASD and also parents of children with ASD who were seeking an alternative route to the traditional education-based approaches. The model reflects an understanding of communication in ASD (both conventional and unconventional) that includes behavioral intentions and communicative functioning. The model is built on previous research supporting the relationship between social-emotional development, communication, and emotional regulation (Prizant 1999; Prizant et al. 1990; Prizant and Meyer 1993; Prizant and Wetherby 1990a, b).

Rationale or Underlying Theory

The SCERTS model emphasizes helping persons with autism to achieve “authentic progress,” which is defined as the ability to learn and spontaneously apply functional and relevant skills in a variety of settings and with a variety of partners. *Social communication* is referred to as the development of spontaneous and functional conversation and the building of secure and trusting relationships with adults. *Emotional regulation* is defined as the ability to maintain a well-regulated emotional state to cope with daily stressors. *Transactional support* is defined as the implementation of supports to respond to a child’s needs and interests. In addition, it helps partners modify and adapt the environment and enhances learning through the use of visual schedules and picture communication (Prizant et al. 2006a). The SCERTS model is a multidimensional approach

that allows professionals to continue using other therapy approaches (i.e., ABA) so long as they reflect one of the main components of SCERTS (social communication, emotional regulation, and transactional relationships). The SCERTS model also takes into consideration that most learning during childhood takes place in the context of daily social activities. With other models, there is often a gap in the support provided to those who serve as supports in the child’s life, such as para-professionals and parents. The SCERTS model takes into account this gap in the form of *transactional supports*, as the model considers the progress made by the child’s partners and how they are able to offer transactional support (i.e., learning supports) across contexts.

Goals and Objectives

The overarching goal of the SCERTS model is to provide children with the support to successfully participate in developmentally appropriate activities with peers, family members, and adult partners in a variety of settings (Prizant et al. 2003, 2004).

There are different goals for each of the three components of the SCERTS model. A sample of these goals is outlined below. Prizant et al. (2003) propounded several goals for each area that can be seen in full in their publication.

Research over the past two decades in the area of ASD has shown two major areas of challenge in terms of *social communication* that are addressed by the SCERTS model: the capacity for joint attention and the capacity for symbol use (Prizant et al. 2003). The capacity for joint attention is seen in a child’s ability to consider the attentional focus of another and to draw another’s attention to events/objects of mutual interest. This ability is considered to be the foundation for the development of language, social relationships, and social-conversational skills (Prizant et al.). The SCERTS model has three levels of goals for joint attention: the prelinguistic level, the emerging language level, and the advanced language level. At the prelinguistic level, goals include establishing shared affect, anticipatory behaviors (i.e., social referencing), and early intentional

behaviors, such as coordinating gestures with a physical gaze. Other goals at this level include increasing frequency/rate of communication bids, expanding the range of communicative functions, developing strategies for communication breakdowns, and developing the ability to communicate intent across persons, environments, and activities (Prizant et al.). The emerging language-level goals include expanding communicative intent, the ability to coordinate attention/affect through gaze, and increasing reciprocity. The advanced language-level joint attention goals include increasing the ability to communicate about past and future events, developing the ability to modify topic selections, and increasing the ability to interpret and use language flexibly (Prizant et al.).

The capacity for symbol use in children with ASD is typically impaired. It is seen through a limited ability to use conventional hand gestures (waving bye-bye, pointing), the use of unconventional vocal development and verbal behavior, and limitations in symbolic play and functional object use (Prizant et al. 2003). Goals at the prelinguistic level for symbol use include establishing consistent means for expressing intent, replacing unacceptable communicative means with acceptable ones, developing the use of more formal augmentative/alternative systems to communicate intentions, and developing the use of multiple gestural and vocal communication means (Prizant et al.). Goals at the emerging language level for symbol use include acquiring core vocabulary to serve a range of communicative functions, expanding vocabulary for semantic relations, expanding representational play themes, and expanding the ability to combine words to show semantic relationships. Goals at the advanced language level include achieving higher level linguistic forms that have differences in meaning (pronouns, tense markers, etc.), increasing the ability to use and interpret nonverbal behavior, and acquiring tools to use language as a tool for emotional regulation (Prizant et al.).

The SCERTS model targets *emotional regulation* by developing goals that are targeted toward the development of *self-regulatory* and *mutual-regulatory* abilities. There are two developmental levels of goals for self-regulatory and mutual-regulatory abilities: *prelinguistic/sensory-motor*

goals and *cognitive-linguistic goals*. Goals at the prelinguistic/sensory-motor level for self-regulation include increasing the child's ability to acquire and use sensory-motor strategies as supports and expanding the use of sensory-motor strategies to support transitions within daily routines. Goals at the cognitive-linguistic level for self-regulation include increasing the child's ability to initiate and utilize cognitive-linguistic strategies to support transitions through daily routines and expanding the ability to use cognitive-linguistic strategies to support attention throughout the day (Prizant et al. 2003). Goals at the *prelinguistic/sensory-motor level for mutual regulation* include increasing the ability to maintain engagement and attention to activities through responding to signs of dysfunction, increasing the ability to use socially acceptable gestures for social control functions, and developing strategies through nonspeech transactional supports to assist with the expression of arousal and emotional states (Prizant et al.). Goals at the cognitive-linguistic level for mutual regulation include increasing acquisitions of vocabulary that permits assistance requests and organizing supports when there is dysregulation, increasing the ability to use specific vocabulary for emotional/arousal state, and increasing the ability to identify and express emotional/arousal state and regulating strategies both with and without the use of a visual schedule (Prizant et al.).

The SCERTS model targets *transactional support* by making goals for *interpersonal support*, *educational and learning support*, and *family support*. Interpersonal support goals support the interactions between the target child and communication partners. Goals in this area include looking at the interaction styles of communication partners that support or serve as barriers to successful interactions, creating efforts across communication partners to use communication styles that support successful interactions, and designing and implementing peer-play interactions that support good language, social, and play models (Prizant et al. 2003). Goals for *educational and learning supports* include designing and implementing organizational and visual supports and designing learning/living environments that support emotional regulation and social communication. *Family support* includes providing

families with educational support to understand the nature of their child's disability and ways to support their child's development and providing emotional support in one-on-one and group settings (Prizant et al.).

Treatment Participants

Children with autism (up to 10 years old), in their preschool/elementary years, are the most appropriate participants. Although the intervention is targeted toward this population, it is not exclusive in nature. SCERTS can also benefit children with significant challenges in social communication and emotional regulation who need transactional support throughout the day. Children with developmental disabilities, communication disorders, and sensory-processing disorders who exhibit social communication and emotional regulation challenges could also benefit from the SCERTS model. Further, the model can be applied to verbal and nonverbal children (Prizant et al. 2003).

Treatment Procedures

The SCERTS model was designed to help professionals assess and establish goals based upon performance in the areas of social communication, emotional regulation, and transactional supports (see description under [Goals and Objectives](#)). The SCERTS Assessment Process (SAP) is considered to be an ongoing assessment process. It is designed to establish a profile of the child's strengths and needs, determine goals that are meaningful and motivational based upon their profile and functional skills, select appropriate teaching strategies and contexts for learning, determine the necessary level of transactional supports (whether it be learning support, support to families, or support among professionals), and monitor progress over time (Prizant et al. 2003).

Efficacy Information

Landsman (2007) did a review of Prizant et al. (2006) SCERTS manual. This review examined

volumes one and two. Volume one describes the SCERTS Assessment Process (SAP), and volume two explains the intervention component. Landsman (2007) states that one of the best features of the SCERTS manual is that it is practice and research driven, as opposed to other interventions that are purely research driven. There is very little research solely dedicated to the efficacy of SCERTS in practice, although the principles guiding the approach are grounded in research on what is known about social communication and emotional regulation.

Outcome Measurement

The SCERTS model uses an evolving assessment process. After the initial assessment of a child and goals are set, the child's progress in the areas of social communication and emotional regulation is monitored through daily logs, weekly logs, and also put into updated quarterlies. This ongoing monitoring of progress allows the team to assess if a child is making progress and if changes need to be made in transactional support. The data-collection procedure is not like other methodologies, as the SCERTS model data gathering involves not "one-on-one" response tracking forms but instead requires a consensus among team members about the child's performance and achievements with natural partners and across natural contexts. Core questions about social communication, emotional regulation, transactional support, and the relationship among the components of the SCERTS model are continually addressed during the process. The SCERTS model is "curriculum-based" or criterion-referenced. There is no normative data to compare a child's results; rather, a child's progress is tracked over time and compared to previously demonstrated skills or abilities.

Qualifications of Treatment Providers

The SCERTS model is most beneficial when it is initiated by an interdisciplinary team, as family members and professionals can contribute input that best addresses a child's needs. Providers

might include speech-language pathologists, general educators, special educators, and other related service providers (e.g., occupational therapist, nurse, social worker, psychologist, physical therapist).

See Also

- Emotional Regulation
- Social Communication

References and Reading

- Bristol, M. N., & Schopler, E. (1984). A developmental perspective on stress and coping in families of autistic children. In J. Blacher (Ed.), *Severely handicapped young children and their families: Research and review* (pp. 91–141). New York: Academic.
- Landsman, M. (2007). *Next to the heart: Where autism should be. A review of the SCERTS model*. PsycCRITIQUES. http://www.barryprizant.com/files/scertsmodel/the_scerts_model_review_from_american_psychological_ass.pdf
- National Research Council. (2001). *Educating children with autism. Committee on educational interventions for children with autism: Division of behavioral and social sciences and education*. Washington, DC: National Academy Press.
- Prelock, P. (2006). *Autism spectrum disorders: Issues in assessment and intervention*. Austin: Pro-Ed.
- Prizant, B. M. (1999). Early intervention: Young children with communication and emotional/behavioral disorders. In D. Rogers-Adkinson & P. Griffith (Eds.), *Communication and psychiatric disorders in children*. San Diego: Singular.
- Prizant, B. M., & Meyer, E. C. (1993). Socioemotional aspects of communication disorders in young children and their families. *American Journal of Speech-Language Pathology*, 2, 56–71.
- Prizant, B., & Wetherby, A. (1990a). Toward an integrated view of early language and communication development and socioemotional development. *Topics in Language Disorders*, 10, 1–16.
- Prizant, B. M., & Wetherby, A. M. (1990b). Incorporating a socioemotional perspective in early communication assessment. *Zero to Three*, 11, 1–12.
- Prizant, B. M., Audet, L., Burke, G., Hummel, L., Maher, S., & Theodore, G. (1990). Communication disorders and emotional/behavioral disorders in children. *Journal of Speech and Hearing Disorders*, 55, 179–192.
- Prizant, B. M., Wetherby, A. M., Rubin, E., & Laurent, A. C. (2003). The SCERTS model: A transactional, family-centered approach to enhancing communication and socio-emotional abilities of children with autism spectrum disorders. *Infants and Young Children*, 16(4), 296–316.
- Prizant, B. M., Wetherby, A. M., Rubin, E., Laurent, A. C., & Rydell, P. (2004). *The SCERTS model: Enhancing communication and socioemotional abilities of children with autism spectrum disorders*. Port Chester: National Professional Resources.
- Prizant, B. M., Wetherby, A. M., Rubin, E., Laurent, A. C., & Rydell, P. J. (2006a). *The SCERTS Model: A comprehensive educational approach for children with autism spectrum disorders: Volume I assessment*. Baltimore: Paul H. Brookes.
- Prizant, B. M., Wetherby, A. M., Rubin, E., Laurent, A. C., & Rydell, P. J. (2006b). *The SCERTS Model: A comprehensive educational approach for children with autism spectrum disorders: Volume II program planning & intervention*. Baltimore: Paul H. Brookes.
- Wetherby, A. M., & Prizant, B. M. (1993). *Communication and symbolic behavior scales*. Chicago: Riverside.
- Wetherby, A., & Prizant, B. (2002). *Communication and symbolic behavior scales developmental profile* (1st normed ed.). Baltimore: Brookes.

Transactional Supports

- Interpersonal Supports

Transdisciplinary Assessment and Intervention

- Role Release

Transdisciplinary Team

- Interdisciplinary Team

Transfer of Function

- Role Release

Transformative Autism Studies (Woods et al. 2018)

► Critical Autism Studies

Transition Cue

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Definition

A transition cue can be any prompt given to an individual to represent or notify of an upcoming change from one activity to the next. Transition cues can be visual, verbal, auditory, or tangible and aim to pair concrete stimuli with a more abstract occasion. Using these cues helps to create a clear concrete representation of the transition as well as an established routine for recognizing and making the transition. Some examples of transition cues are ringing a bell or using a timer, singing a specific song, clapping hands or hand signals, picture cues, object cues, or any combination of these.

References and Reading

- Cooper, J., Heron, T., & Heward, W. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson Education.
- Dettmer, S., Simpson, R., Myles, B., & Ganz, J. (2000). The use of visual supports to facilitate transitions of students with autism. *Focus on Autism and Other Developmental Disabilities*, 15, 163–169.
<http://www.iidc.indiana.edu/?pageId=399>
- Hume, K. (2008). Transition time: Helping individuals on the autism spectrum move successfully from one activity to another. *The Reporter*, 13(2), 6–10.
- Thiemann, K. S., & Goldstein, H. (2001). Social stories, written text cues, and video feedback: Effects on social communication of children with autism. *Journal of Applied Behavior Analysis*, 34, 425–446.

Transition Planning

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Definition

A legally required process for planning a student's transition services. During transition planning, the IEP team must discuss and decide whether the student needs transition services and activities (e.g., instruction, related services, community experiences) to prepare for the different domains of adulthood (postsecondary education, vocational education, employment, adult services, independent living, etc.) and if so set goals, develop strategies, and outline tasks and schedules to accomplish the goals in the student's individualized education program (IEP) or in some States on a document sometimes referred to as the Transition IEP or plan.

The focus within IDEA on school to adult life transition is an important step in improving adult outcomes for students with disabilities. As stated in IDEA §300.43, (a) *Transition services* means a coordinated set of activities for a child with a disability that-

1. Is designed to be within a result-oriented process, that is focused on improving the academic and functional achievement of the child with a disability to facilitate the child's movement from school to post-school activities, including postsecondary education, vocational education, integrated employment (including supported employment), continuing and adult education, adult services, independent living, or community participation;
2. Is based on the individual child's needs, taking into account the child's strengths, preferences, and interests; and includes-

- (i) Instruction;
- (ii) Related services;
- (iii) Community experiences;
- (iv) The development of employment and other post-school adult living objectives; and
- (v) If appropriate, acquisition of daily living skills and provision of a functional vocational evaluation.

(b) Transition services for children with disabilities may be special education, if provided as specially designed instruction, or a related service, if required to assist a child with a disability to benefit from special education.

From §300.320(b):

(b) *Transition services.* Beginning not later than the first IEP to be in effect when the child turns 16, or younger if determined appropriate by the IEP Team, and updated annually, thereafter, the IEP must include-

1. Appropriate measurable postsecondary goals based upon age appropriate transition assessments related to training, education, employment, and, where appropriate, independent living skills; and
2. The transition services (including courses of study) needed to assist the child in reaching those goals.

Historical Background

The transition from school to adulthood is critical in the lives of all students. For a student with autism spectrum disorder (ASD), transition of any kind can be challenging and the transition from school to postsecondary education or employment.

The curriculum for any student should be based on desired long-term outcomes. However, unlike their nondisabled peers, students with disabilities often seemed unprepared to achieve success as adults, despite the fact that they received special education. As a result, the Office of Special Education and Rehabilitation Services established transition as a priority and funding priority in the early 1980s.

Over the next 10 years time, Madeline Will proposed Bridges from School to Work as a conceptual model for transition and required transition planning. In addition, Halpern developed a conceptual model of transition. By the end of the decade, OSERS funded a Transition Study from 1987 to 1993 that included over 8,000 youth ages 13–21. In 1990, IDEA is passed and included provisions for transition services, and a systems change in transition priority was established in 1991. In 1992, the Rehabilitation Act was amended to include the same definition of transition as IDEA. In 1994, The School-to-Work Opportunities Act was passed that required the inclusion of all students. In 1996, the National Transition Alliance was funded to provide technical assistance to states to improve transition of outcomes of youth with disabilities. However, many states were too slow in implementing transition services requirements and failed to meet minimal levels of compliance (Johnson et al. 2002).

As mentioned earlier, transition services for students with disabilities were addressed for the first time in the 1990 amendment to the IDEA. Subsequent amendments to the IDEA have continued to strengthen and promote the vision for youth with disabilities to transition from education to employment and community living.

The IDEA 2004 reauthorization included requirements to develop and obtain measurable transition goals that articulate the intended post-graduation achievements of the student. This requirement went into effect July 1, 2005.

Transition planning has expanded since the 1980s from the narrow focus on a student's post-school environment which often focused only on employment to include curriculum and instruction to teach self-determination and work skills, and transition assessment systems and processes (Cobb and Alwell 2009). This evolution reflects emphases on the whole person and quality of life today.

Current Knowledge

Transition planning for a student with ASD should begin early in their academic career to

ensure appropriate planning of assessment, coursework, opportunities, and diploma status. For some students, this may be needed when they are young. The law, IDEA 2004, states transition planning is mandatory at age 16. Notably, by this time, coursework and diploma status has often been determined, thus limiting post-secondary opportunities for some students.

In order to systematically address transition services, the following three steps may prove useful: assessment to inform transition planning, development of goals and objectives, documentation and implementation.

During the assessment phase, assembly of key team members is significant to the success during the subsequent phases. Parents, teachers, and other support providers will work together to collect the necessary background documentation on the student. Assessments can be a variety of measures including formal assessments such as published assessments or checklists; informal assessments such as interviews, person-centered planning tools, or review of records; and clinical observations. Combined, these tools can be completed on an ongoing basis and offer a comprehensive picture of the student. These assessments are then used to inform transition planning.

Sitlington and Clark (2007) identified eight areas that should be considered when assessing the academic and functional performance of students:

1. Interests
2. Preferences
3. Cognitive development and academic achievement performance
4. Adaptive behavior
5. Interpersonal relationship skills
6. Emotional development and mental health
7. Employability and community skills
8. Community participation

While the components of the assessment vary based on the student's age and areas of individual need, a comprehensive assessment should include career awareness assessments, vocational evaluation and assessments, assessments of independent living and community skills, and assessment of self-determination.

Patton and Dunn (1997) indicated that there are two types of goals that can result from transition planning: instructional and linkages to community services and agencies. Cronin et al. (2008) note that both academic and functional skills need to be addressed.

Future Directions

With thoughtful planning and appropriate accommodations, the transition process for a student with autism can be clearer, which means less confusing and infinitely more successful. This is accomplished by following best practices and as needed using creative strategies to specifically address the behavioral and educational needs of a student with autism spectrum disorder (ASD). Autism presents as a compilation of characteristics that consist of sensory abnormalities and impairments in communication and social development. The severity of these characteristics falls along a continuum that generally determines the level of functioning of the student. The characteristics evident in ASD manifest in many ways including challenging behavior, difficulties in communication, and processing of information.

Challenging behaviors, particularly, can make transitions even more difficult to navigate successfully. Although students with ASD share many common characteristics, all students have unique strengths and needs that must be evaluated to inform transition planning.

Although self-determination training has been part of transition planning since IDEA 1999, today it is receiving much more attention such as writing self-advocacy goals in to the IEP related to transition. However, many special educators are not prepared to teach the skills of self-determination and do not know how to infuse them into the curriculum Wehmeyer et al. (2000).

Interagency and interdisciplinary planning is essential. However, in practice, professionals often overlook involving various adult agencies in transition planning (Patton 2004). And the requirement by law for vocational rehabilitation to be involved may not be adhered to by some. Family participation is also lacking. Transition planning is

not effective without family involvement. Furthermore, they are not able to benefit from it.

Sitlington (2008) states research is needed to examine and document the effectiveness of major approaches to instructing adolescents with disabilities. A review of transition literature by Test et al. (2009) reports no or limited studies addressing these issues. Furthermore, even in the realm of best practices, these are rarely implemented. Future research should provide empirical evidence of best practices. Sitlington (2008) goes on to also state that research should consider variables related to all aspects of adult living in follow-along studies and relate individual's education and transition planning experiences to follow-along findings.

See Also

- [Employment in Adult Life](#)
- [Employment Specialist](#)
- [Supported Employment](#)

References and Reading

- Cobb, R. B., & Alwell, M. (2009). Transition planning/coordination interventions for youth with disabilities: A systematic review. *Career Development for Exceptional Individuals*, 32, 70–81.
- Cronin, M. E., Lemoine, M. F., & Wheeler, S. C. (2008). Developing life skills. In G. Blalock, J. R. Patton, P. Kohler, & D. Bassett (Eds.), *Transition and students with learning disabilities: Facilitating movement from school to adult life* (2nd ed., pp. 203–224). Austin: Hammill Institute on Disabilities.
- Individuals with Disabilities Education Improvement Act (IDEIA). (2004). Public Law no. 108-446, 118 Stat. 2647 [Amending 20 USC 1400 et seq.].
- Johnson, D. R., Stodden, R. A., & Emanuel, E. J. (2002). Curricular challenges facing secondary education and transition services. *Exceptional Children*, 68, 519–531.
- Patton, J. R. (2004). Transition issues: Processes, practices and perspectives. In A. M. Sorrells, H. J. Reithe, & P. T. Sindelair (Eds.), *Critical issues in special education: Access, diversity and accountability* (pp. 108–124). Boston: Pearson.
- Patton, J. R., & Dunn, C. (1997). *Dream. Summary of performance for graduating seniors and exiting aging out students. Transition planning process*. Austin: ProEd.
- Patton, J. R., & Dunn, C. (1998). *Transition from school to young adulthood: Basic concepts and recommended practices*. Austin: Pro-ED.
- Sitlington, P. L. (2008). Transition to life in the community. In G. Blalock, J. R. Patton, P. Kohler, & D. Bassett (Eds.), *Transition and students with learning disabilities: Facilitating movement from school to adult life* (2nd ed.). Austin: Hammill Institute on Disabilities, 307.
- Sitlington, P. L., & Clark, G. M. (2006). *Transition education and services for adolescents with disabilities* (4th ed.). Boston: Allyn and Bacon.
- Sitlington, P. L., & Clark, G. M. (2007). The transition assessment process and IDEIA 2004. *Assessment for Effective Intervention*, 32(3), 133–142.
- Test, D. W., Fowler, C. H., Richter, S. M., White, J., Mazzotti, V., Walker, A. R., & Korterling, L. (2009). Evidenced based practices in secondary transition. *Career Development for Exceptional Individuals*, 32, 115–128.
- Wehman, P. (2012). *Life beyond the classroom*. Baltimore: Paul H Brookes.
- Wehman, P., Datlow Smith, M., & Schall, C. (2009). *Autism and the transition to adulthood success beyond the classroom*. Baltimore: Paul H Brookes.
- Wehmeyer, M. L., Palmer, S. B., Agran, M., Mithaug, D. E., & Martin, J. E. (2000). Promoting causal agency: The self determined learning model of instruction. *Exceptional Children*, 66, 439–453.
- Will, M. (1984). *OSERS programming adults with disabilities: Bridges from school to working life*. Washington, DC: Office of Special Education and Rehabilitative Services.

Transition Support Practices

- [Barriers to and Facilitators of Successful Early School Transitions for Children with Autism Spectrum Disorders](#)

Transitional Living

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Definition

Transitional living is a type of living arrangement in which an individual is learning a new way of life in preparation for going out on their own. This living

model allows an individual to be taught how to gradually ease into the process of conducting their life in a different, more productive way.

Many older teenagers and young adults with autism spectrum disorders, specifically high-functioning autism or Asperger's disorder, struggle with the social competencies and adaptive skills that are necessary to live independently, work successfully, and enjoy their community. Transitional living provides these individuals with an environment that focuses on teaching and supporting these critical skills, with the opportunity to use and practice these skills across multiple settings and with diverse populations. Fading of supports needs to be gradual, with opportunities for feedback.

It is also important that, once the individual with autism spectrum disorder has transitioned into an independent living arrangement, monitoring and feedback continue in order to promote success and address issues that may compromise that success.

See Also

- [Benhaven Residential Services](#)
- [Independent Living](#)
- [Transition Planning](#)

References and Reading

- Nerney, T., & Shumway, D. (1996). *Beyond managed care: Self-determination for people with disabilities* (1st ed.). Durham: University of New Hampshire Press.
- Schwartz, A. E., McRoy, R. G., & Downs, C. A. (2004). Adolescent mothers in a transitional living facility; an exploratory study of support networks and attachment patterns. *Journal of Adolescent Research, 19*(1), 85–112.
- Wehman, P., Smith, M. D., & Schall, C. (2009). *Autism and the transition to adulthood success beyond the classroom*. Baltimore: Paul H. Brookes.

Trauma

- [Injuries in Children with ASD](#)

Travel Training

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Definition

Travel training is a systematic approach to teaching individuals on the autism spectrum to use mass transit. According to Groce (1996a), "Travel training is short term, comprehensive, intensive instruction designed to teach students with disabilities how to travel safely and independently on public transportation. The goal of travel training is to train students to travel independently to a regularly visited destination and back" (p. 2). Without the ability to travel independently, an individual with autism is isolated and must rely upon others to transport them to work, social activities, and other activities of daily living (e.g., grocery shopping).

Historical Background

The history of travel training is scant at best. With the passage of the Individuals with Disabilities Education Act (IDEA) in the 1990s, educators acknowledged the need to train individuals with disabilities regarding how to access and use public transportation to increase their independence and employability. Publications were primarily in the form of newsletters and were written by special educators in large urban settings that had well-established public mass transit systems. Individuals with autism were not specifically studied nor were they specifically commented upon regarding a need for specialized instruction. Individuals on the spectrum were simply subsumed under the general category of individuals with developmental disabilities.

Current Knowledge

Driving an automobile is a highly complex skill that requires the ability to read or predict the intentions of other drivers. It also requires drivers to make instantaneous decisions when other vehicles drive in a manner that breaks the rules of the road or drive in an unpredictable manner. Individuals with autism have been described as having a lack of theory of mind (ToM) or the ability to predict the actions or intentions of others. Consequently, many individuals, even those that are higher functioning on the autism spectrum, will not learn how to drive. This severely limits a person socially and economically. According to the National Center on Workforce and Disability, the lack of reliable transportation is one of the most significant barriers to employment to individuals with a variety of disabilities (NCWD 2011).

Therefore, travel training skills are essential in maximizing the employability and independence of individuals on the autism spectrum.

Travel training must be broken down into subsets of skills that should be taught and learned in a developmentally sequential and appropriate manner. Three meta-skills that need to be assessed and mastered prior to independent travel are “(1) Awareness of personal space – meaning a clear understanding of where their personal space ends and others begins. . . ; (2) An awareness of their environment. . . ; and (3) The ability to recognize and respond to danger” (Groce 1996b, p. 10). Before travel training can begin in earnest, a child on the autism spectrum should be taught basic pedestrian skills including walking in a crosswalk, looking both ways when crossing the street, using pedestrian crossings with “Walk” and “Don’t Walk” signs and symbols, etc. In vivo practice crossing the streets is essential, and observations of pedestrian safety skills should be conducted. Some individuals on the autism spectrum because of their literalness or concreteness have been observed to wait for an extraordinary length of time for a crosswalk light to change because they did not realize that they had to push a button to activate the crosswalk light. Others have frozen in a panic because the light

changed from “Walk” to “Don’t Walk” while they were in the middle of the street. Although pedestrians legally have the right of way, they are still at risk of being struck by a motor vehicle even in a crosswalk. In one New York City study, 40% of pedestrians were struck by a car while walking in a crosswalk and accounted for 52% of all traffic fatalities (Solomonow and Gastel 2010). Individuals on the autism need to learn to anticipate the possibility of a car running a red light and must continuously scan the environment for danger. Confusion concerning what to do when a traffic light malfunctions also needs to be dealt with in training as well as developing the individual’s problem-solving skills. Reading basic traffic signs should be incorporated in pedestrian skills training.

For a child on the autism spectrum, part of the difficulty will lie in the area of sensory integration issues. Public transportation is often loud, crowded, and noisy. Smells of bus fumes and odors on the subway platforms can overwhelm even the most hardened traveler. Parents can help a child on the autism spectrum by previewing the sights, sounds, and smells for their child prior to traveling on the subway, train, or bus. Children that are hypersensitive to sound may use a MP3 player or ear plugs as a means of coping with the high-pitched sounds of brakes or pneumatic lowering devices found on city busses. A child who is hypersensitive to smell can use a handkerchief perhaps coated with a soothing scent to cope with the smells of a subway platform. With young children on the autism spectrum, the initial goals of traveling on mass transit are simply to habituate them to the sensory experiences associated with public transportation.

By incorporating the use of mass transit in the family’s daily life, the child with autism will have an easier time transitioning from traveling with his or her family to traveling independently. Parents should use public transportation in planning fun outings for the family to the beach, a museum, a baseball game, or some activity that is of particular interest to the child on the spectrum. Pairing the use of public transportation with fun and interesting activities for the child will help increase the probability of success. Early

trips with the young child should emphasize safety and problem-solving skills (e.g., not standing too close to the edge of the platform or the curb; procedures to enact should the child become separated from his parents while on a subway train versus a bus; and how to identify appropriate people to ask for help) (VanBergeijk 2009a). A natural show-and-tell approach by parents with their children can be used on daily outings on mass transit. Parents can name parts of the bus or train such as the stop request button or cord, exit, etc. and show the child familiar landmarks along frequently traveled routes especially those right before a destination stop. These natural teachable moments should include locating route maps and neighborhood maps. Learning to read these maps and general navigation skills are critical subskills to independent travel.

As the child ages, the trips should shift from parent-led to child-led outings. In this approach, the parent becomes the pupil, and the child shows the parent where to safely stand, find maps, locate schedules, and review procedures of what to do if separated. During this phase of parent-led travel training, pretravel planning can be introduced. Here, the parent can show the child how to locate information using the Internet regarding types of transportation to take, routes to follow, transfers involved, schedules, and varying fares. Not only should the child learn how to locate information reading the local transit system or regional rail travel, but they should also learn to use commercially available GPS/mapping sites or route-optimizing web sites. This will increase the child's sense of mastery and decrease their anxiety regarding travel.

Some higher functioning individuals on the autism spectrum such as those with Asperger syndrome will use their profound rote memorization skills to memorize subway maps and schedules. This can give parents a false sense of security that their child "knows" how to travel independently. The child will also believe he or she knows everything there is to know about traveling independently. What should be practiced and even staged are changes in service or "missed" trains and busses. These unexpected service interruptions or changes in the routine of schedule

can lead to meltdowns or panic. Practicing problem-solving skills in these situations is critical in training individuals on the autism spectrum to travel independently. The use of cell phones with preprogrammed telephone numbers should be practiced. During a meltdown or panic attack, the individual may not remember even frequently dialed telephone numbers. Well-labeled speed dial numbers will help alleviate some of the panic associated with a sudden and unexpected change in schedule or service.

At what age a child should travel or begin to learn to travel independently depends upon the developmental and cognitive level of the child. The child who appears to have mastered the aforementioned skills may be ready to try short tests. Since travel training is a complex skill, parents will need to use successive approximations to shape this behavior. As the child reaches junior high or high school age and if they are cognitively and developmentally able, the child can travel alone one stop along a well-traveled and known subway or bus route. Older siblings and other relatives can help in the process by waiting ahead of time at the destination while the parent(s) put the student on the subway or bus. The number of stops an older child travels alone can be increased along the well-traveled route as his or her confidence increases until the child has increased the number of stops traveled without parents to a desired destination. One suggested final destination to practice traveling to is the child's school. The child on the autism spectrum should be explicitly taught to travel with at least one other person or travel with groups of people. Enlisting willing classmates to travel with the child on the spectrum will help increase safety and normalize the experience of traveling to school without one's parent. Once the older child on the autism spectrum has mastered the familiar trip to school, other frequently traveled routes should be also traveled alone in successively longer intervals between stops. Finally, practicing transfers between busses and subways is an even more complex skill that should be attempted after mastering single modes of transportation.

Training using fixed route transit as described above or using paratransit systems offered

by schools or offices of developmental disabilities or vocational rehabilitative services should be considered when parents are not successful training their children to travel independently. The formal travel training can be incorporated into a student's individualized education plan as a part of a formal transition plan beginning at age 14 under the Individuals with Disabilities Education Act. Postgraduation or after the age of 21 years old, formal travel training will be the responsibility of either the state's vocational rehabilitative services office or state office of developmental disabilities.

Voorhees (1996) described a seven-phase process that formal travel training programs should employ when working with individuals with a variety of cognitive disabilities. These phases include the following:

- Phase 1: instruction in detail of specific route-related skills such as pedestrian skills, paying fares, interacting with community workers and strangers, and appropriate behaviors in public.
- Phase 2: observation and verification by a travel trainer of the skills learned in phase 1.
- Phase 3: emergency procedures training (e.g., missing a bus or train, losing a fare card, getting on the wrong bus or train, etc.).
- Phase 4: assessment of a student's skills in dealing with strangers.
- Phase 5: involves the student being observed from a distance by a travel trainer with the student's full knowledge of the travel trainer's presence.
- Phase 6: the student is observed covertly and with no knowledge of the observation and assessment of his or her skills.
- Phase 7: follow-up covert observations on a periodic basis.

A formalized program of this nature can be implemented by the school district, a state office of vocational rehabilitative services, or a social service agency subcontracted by the state to provide those services. Aspects of formal travel training programs may also be a part of the curriculum at college-based comprehensive transition programs.

Learning travel training skills are essential for individuals on the autism spectrum in order

for them to fully integrate into adult life and independent living. This is self-evident to many people. However, traveling for pleasure may not appear as an obvious need. Traveling for pleasure both domestically and overseas helps to increase the quality of life for individuals with ASDs. It provides them with opportunities to learn new skills, try new foods, make friendships, and accumulate fond memories which can be the basis for conversations with new people they meet.

Traveling for pleasure for individuals on the spectrum often involves family vacations. As the child grows older, he or she can join summer travel programs for individuals with ASDs or other types of disabilities. Many of these summer travel programs are affiliated with summer camps that serve a wide variety of campers with disabilities. Often, these are returning campers who are older and are looking for new experiences. Other travel programs that are for pleasure are offered by postsecondary independent living programs or comprehensive transition programs.

The traveling for pleasure programs can build upon travel training programs that aid individuals in traveling to and from home and work. Some programs simply offer guided tours with no specific curriculum. Others offer a more formalized curriculum which could begin months before the actual trip overseas.

The curriculum can include the following aspects: (1) how to apply for a passport and/or a visa; (2) how to use public transportation to reach the airport; (3) the purchasing of tickets, tours, and accommodations; (4) packing suitcases with appropriate clothing for a trip; (5) what to expect when going through airport security; (6) how to answer airport security questions; (7) words or phrases not to use in an airport or aboard an airplane; (8) forbidden items not to pack in a suitcase or bring aboard an airplane; (9) monetary exchanges; (10) personal safety when traveling abroad; (11) completing customs declaration forms; (12) the use of international calling cards; (13) dealing with jet lag; (14) local natural and historic sites of interest; (15) cultural do's and don'ts; and (16) cuisine of the destination country. As a way to both quell anxieties and stoke positive anticipation of an upcoming trip, some international travel training programs host an event

prior to the trip in order to introduce traveling companions to each other, show a slide show of points of interest in the destination country to group members, and serve a dinner featuring foods from the country where the group will be traveling. Many individuals on the spectrum have limited palates or food choices and will be anxious about what they will eat or drink. By hosting a dinner with the foods they may encounter on the trip, the program helps reduce the individual's anticipatory anxiety over food and helps them identify foods they may like to eat on the trip.

When choosing an international travel training trip for an individual on the spectrum, it is important to assess how the program will provide participants with structure, predictability, and safety. The ratio of chaperones to participants will depend upon the level of functioning of the participants. The lower the level of functioning, the lower the ratio of chaperones to participants should be. While traveling, the group should be further subdivided into units where a chaperone is solely responsible for the whereabouts of a smaller number of individuals. Throughout the day, the group leader should ask chaperones to conduct a head count. The group leader should structure the day so that each day is similar (e.g., wake up calls and breakfast set at the same time, early morning preview meeting of the day's activities, early evening debriefing meeting with a preview of the following day's activities, escorting the participants to pay phones to call home at the same time and providing assistance with calling cards, etc.). The group leader will have to review emergency procedures with the group such as what to do if lost and hand each participant a business card with the name of the hotel that the group is staying at and insure the participant places the card in his or her wallet that he or she carries at all times. Periodically, the group leader will also have to remind participants about taking care of their daily hygiene, dress codes for different venues, and taking prescribed medications. With a change in time zones, participants can become particularly confused about when to take their medications. Staff must help them transition to the new time zone and new schedule of taking medications. When engaged in adventure

activities (e.g., zip lining, snorkeling, outrigger canoeing, etc.), participants should be given an option that does not involve a perceived risk. Some chaperones should offer an alternative activity that they supervise while the other group members engage in the adventure activity.

Finding a travel program for individuals with any kind of disability can be a challenge. Airports are beginning to offer programs to families with children on the spectrum to help them cope with the stressors of travel (Strunsky 2011; WALB News 2011). Some boarding schools and comprehensive transition programs offer this type of programming as an extracurricular activity (VanBergeijk 2009a). The National Clearinghouse on Disability and Exchange provides information regarding foreign student exchange (Mobility International USA 2011). Using the Internet search engines with keyword searches such as "special needs camps and travel" or some other similar phrasing will yield a list of camps and travel programs which cater to individuals with disabilities.

Teaching an individual travel training skills is essential to his or her independence. It increases the person's freedom and overall quality of life. Mastery of travel training skills also contributes to an individual's increased self-esteem. This is a complex skill that can be taught systematically throughout a person's development. Refresher sessions may be necessary as changes occur in public transportation.

Future Directions

There is very little research in the field of travel training. The majority of recently published studies focus upon individuals with physical disabilities with a particular emphasis upon individuals with visual impairments. Currently, there are no published research studies that are specific to individuals on the autism spectrum. In the future, research studies should focus upon this population and conduct efficacy-based research comparing parent-led training versus curricula developed by social service and employment agencies and by school districts. Distinctions should be made in the research between fixed route travel skills using

mass transit or paratransit services, flexible route travel training skills, and travel for pleasure skills.

Travel training for employment and independent living skills should be incorporated in a student's individualized education plan (IEP) under the Individuals with Disabilities Education Act (IDEA) or as a part of an individualized plan for employment (IPE). The travel training can be added to the IEP as a part of the transition plan when the student reaches age 14. Pedestrian and other safety skills can be added to the IEP earlier in the process. The trend in the future will be that travel training will be more often formally incorporated into IEPs and IPEs as parents become aware of this possibility.

See Also

- [Individualized Plan for Employment \(IPE\)](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Transition Planning](#)

References and Reading

- Groce, M. M. (1996a). An introduction to travel training. *Transition summary: Travel training for youth with disabilities* (pp. 2–5). National Information Center Children and Youth with Disabilities (NICHY), Newark, NJ.
- Groce, M. M. (1996b). A model of a travel training program: The New York City Board of Education Travel Training Program. *Transition summary: Travel training for youth with disabilities* (pp. 10–13). June 9. National Information Center Children and Youth with Disabilities (NICHY), Newark, NJ.
- Mobility International USA. (2011). Autism spectrum/Asperger and international exchange.
- National Center on Workforce and Disability. (2011). Transportation for people with disabilities. Retrieved 11 May 2011, from http://www.onestops.info/article.php?article_id=124.
- National Clearing House on Disability and Exchange Mobility International USA. (n.d.). Autism spectrum/Asperger and international exchange. Retrieved 11 May 2011, from <http://www.miusa.org/ncde/tipsheets/autismtips/?searchterm=autism>.
- Solberg, M. M., Fickas, S., Lemoncello, R., & Hung, P. F. (2009). Validation of the action communities transportation model for individuals with cognitive impairments. *Disabilities and Rehabilitation*, 31(11), 887–897.
- Solomonow, S., & Gastel, S. (2010). NYC DOT, NYPD announce new initiatives to improve safety for pedestrians, motorists and cyclists. New York City Department of Transportation press release. Retrieved 11 May 2011, from http://www.nyc.gov/html/dot/html/pr2010/pr10_053.shtml.
- Strunsky, S. (2011). Newark airport program helps autistic children cope with air travel. *The Star Ledger*. Retrieved 11 May 2011, from http://www.nj.com/news/index.ssf/2011/02/nj_autistic_children_brave_con.html.
- VanBergeijk, E. O. (2009a). Travel advice for higher functioning individuals on the autism spectrum. *Exceptional Parent*, 39(12), 44–45.
- VanBergeijk, E. O. (2009b). Travel training for higher functioning individuals on the autism spectrum. *Autism Spectrum News*, 1(3), 32–40.
- Voorhees, P. (1996). Travel training for persons with cognitive or physical disabilities: An overview. *Transition summary: Travel training for youth with disabilities* (pp. 7–9). June 9. National Information Center Children and Youth with Disabilities (NICHY), Newark, NJ.
- WALB News. (2011). *Program eases travel stress for families with autistic children*. The Autism News. Retrieved 11 May 2011, from <http://www.theautismnews.com/2011/01/31/program-eases-travel-stress-for-families-with-autistic-children/>.

Traveling Teacher

- [Itinerant Teacher](#)

Trazodone

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Synonyms

[Desyrel](#)

Definition

Trazodone is an older antidepressant medication that has a mixed mechanism of action affecting multiple neurotransmitter systems. It is often used in adults as an adjunctive treatment for depression, particularly to aid with sleep. It has not been evaluated in children or adults with autism. The use of trazodone is avoided in males due to multiple reports of priapism. Priapism is an acute problem characterized by prolonged and painful erection.

See Also

► [Depressive Disorders](#)

References and Reading

- Gedye, A. (1991). Trazodone reduced aggressive and self-injurious movements in a mentally handicapped male patient with autism [letter]. *Journal of Clinical Psychopharmacology*, 11(4), 275–276.
- Kem, D. L., Posey, D. J., & McDougle, C. J. (2002). Priapism associated with trazodone in an adolescent with autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(7), 758.
- Martin, A. M., Scahill, L., & Kratochvil, C. J. *Pediatric psychopharmacology* (pp. 263–274). New York: Oxford University Press.

Treatment Adherence

► [Treatment Fidelity](#)

Treatment and Education of Autistic and Communication-Related Handicapped Children

► [TEACCH Transition Assessment Profile \(TTAP\)](#)

Treatment and Education of Autistic and Related Communication-Handicapped Children

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Definition

TEACCH was originally an acronym for *Treatment and Education of Autistic and related Communication-handicapped CHildren* (although TEACCH now serves individuals of all ages). TEACCH is based at the University of North Carolina at Chapel Hill and is a nationally and internationally known model of conceptualizing autism spectrum disorders (ASD) and delivering services and supports (Mesibov et al. 2005).

Historical Background

TEACCH began at the University of North Carolina at Chapel Hill in 1972 as a continuation and expansion of a federally funded grant to the late Eric Schopler, Ph.D. for clinical work with children with autism and their families. When the federal grant ended, Dr. Schopler and the families successfully lobbied the North Carolina General Assembly to continue funding the program. TEACCH now includes regional centers across North Carolina that provide diagnostic evaluations, parent coaching, social and support groups, and other clinical services. TEACCH also operates model early intervention and supported employment programs and a residential/vocational program (the Carolina Living and Learning Center) and provides individual consultations and professional training workshops. The directorship of TEACCH passed from Dr. Schopler to Gary Mesibov, Ph.D. in 1993 and then to Laura Klinger, Ph.D. in 2011.

Rationale or Underlying Theory

TEACCH developed the concept of the “Culture of Autism” to convey the idea that like cultures, autism spectrum disorders yield characteristic patterns of thinking, communicating, and behaving. This concept is broader than, although compatible with, standard diagnostic criteria. TEACCH proposes that teachers, therapists, and parents should function as “cross-cultural interpreters” who translate the expectations of the neurotypical world into forms that people with ASD understand. Therefore, understanding the Culture of Autism is a prerequisite for developing the most effective interventions.

Features of the Culture of Autism include (1) scattered cognitive skills, with relative strengths in visual understanding and relative weakness in auditory processing; (2) tendency to focus on details rather than on the underlying, integrated meaning of the details (i.e., limited central coherence; Frith 2003); (3) distractibility or other atypical patterns of focusing and disengaging attention; (4) difficulties with generating organizational strategies; (5) limited ability to understand the perspectives and experiences of other people; (6) difficulties with generalizing from one situation to another; (7) difficulties with time concepts and sequencing in time; (8) atypically strong interests and impulses; and (9) sensory and perceptual differences. Many individuals with ASD also have excessive levels of anxiety. Further, all have some form of communication disorder, which varies across individuals, ranging from no speech and extremely limited receptive language to excessive, awkward speech and occasional receptive difficulty with nonliteral communication such as “body language,” verbal sarcasm, and idioms.

Goals and Objectives

The TEACCH approach involves strategies both for teaching new skills and for designing environmental supports. One long-term goal of the TEACCH approach is developing skills for daily living so that individuals function with the highest

possible degree of independence given their neuropsychological impairments. Other goals involve meeting fundamental human needs of engagement in productive, personally meaningful activities and feelings of dignity and security.

Treatment Participants

The TEACCH approach is appropriate for individuals with ASD at all ages and functioning levels. It can be used to help individuals with ASD learn new skills or participate appropriately in any setting, including, but not limited to:

- Schools and child care centers (from preschool to graduate school, from special education settings to typical classes)
- Home (e.g., learning to participate in chores, mealtimes, leisure time, homework)
- Community activities and errands (e.g., standing in a checkout line, sitting through a religious service, participating in grocery shopping, getting a haircut)
- Medical, dental, and therapy appointments (e.g., remaining cooperative through the steps involved in examination or treatment, following the sequence of activities in speech/language or occupational therapy sessions)
- Work sites (e.g., competitive employment, supported employment, volunteer work with a job coach, sheltered employment settings)

Treatment Procedures

The overall TEACCH approach is called “Structured TEACCHing.” The fundamental principles of Structured TEACCHing are:

- Individualize interventions based on the person’s specific pattern of difficulty, strengths, and special interests.
- Use visual or written supports to supplement auditory language-based information.
- Make the sequence of events predictable and understandable using visual means, such as a schedule of upcoming activities (depending on

the individual's skills, this may take the form of objects, picture, or written words).

- Organize the physical environment and visually highlight important features of both the surroundings (such as where to sit, stand, walk, drive, etc.) and tasks (such as where to focus attention or put materials).
- Work specifically on flexibility and generalization by changing the sequence of activities and introducing new elements (e.g., location, materials, staff or peers, etc.).
- Stimulate and support meaningful, self-initiated communication (in contrast to prompted speech or rote memory).
- Collaborate with families.

TEACCH Structured TEACCHing suggests that for teaching new skills or in situations of behavioral difficulties, visual answers should be provided for the following questions:

- Where am I supposed to be?
- What am I supposed to do?
- How much will I do? How long will I do this?
- How can I see that I am making progress, and how can I see that I have finished?
- What will I do next?

Techniques for answering these questions vary widely based on the individual's age and skill level and the setting. For young and developmentally delayed learners, typical techniques for answering "where" include defining learning spaces through furniture placement and blocking out sources of distraction. For older and developmentally advanced individuals, it is often useful to provide visual guidance (or options) about where to sit, where to put possessions, and how to get from place to place in the school or work setting.

Techniques for showing "what" generally involve clear visual directions (often accompanied with brief verbal directions) about the steps in the task and the sequence of tasks to be completed. For example, a developmentally young learner might have a system consisting of different shapes on his worktable. Each shape would correspond to a shape on a box containing a task. The person would know *what* task to do by taking a

shape from the table and matching it to the box with the same shape. The person would know *how much* work there was by the number of shapes on the worktable. For example, if there were three shapes, that would mean there were three tasks to complete during the work session. *Progress* would be understood by seeing the shapes on the table decrease and seeing the boxes moving into the finished area as tasks were completed. The person would know that the session was *finished* when all of the shapes had been removed from the worktable. A more developmentally advanced person might have a written system with a list of tasks labeled in words (such as "science, PE, social studies" or "staple, fold, insert into envelope, sort by zip code"). The individual would know *what* to do based on the written words corresponding to the labels on each of the books, folders, or other containers in the work area. *How much* work would be seen by the number of items written on the list or by a written indication of the time the activity would end. The person would see that *progress* was being made as each item on the list was crossed off. When all the words were crossed off, that would be the sign that the work was *finished*.

Techniques for showing "how much" or "how long" include (1) having materials organized into containers that become emptier as the task moves toward completion, (2) having a list of activities that are checked off or removed as they are completed, and (3) using a visual timer that provides information about the passage of time and arrival of the end of the activity. Consistent with these techniques, "finished" is shown by empty containers of materials, a completed checklist, or a visual and/or auditory cue from a timer or clock. When activities are completed, there is typically a mechanism for guiding the individual to engage in a leisure time activity or to look back to the schedule to find the next activity.

Efficacy Information

A current trend in psychology and related disciplines suggests that randomized controlled trials (RCTs) are the best way to demonstrate efficacy.

(In an RCT, a single variable is manipulated, while all other factors are effectively neutralized through random assignment of subjects to treatment or control groups.) However, the RCT methodology is not universally accepted as the best approach for assessing comprehensive ASD treatment programs (Mesibov and Shea 2011; Reichow et al. 2011), and there are currently no published RCTs of TEACCH versus other methodologies, although one quasi-experimental study is in progress (Odom 2007). Several recent studies using other research methodologies have provided evidence of the efficacy of Structured TEACCHing techniques (Mesibov and Shea 2010). Also, the National Standards Report (National Autism Center 2009) identified Structured TEACCHing by name as an “emerging treatment” and called several components of Structured TEACCHing “established treatments” – specifically Antecedent Packages and Schedules. Similarly, the National Professional Development Center on Autism Spectrum Disorders (<http://autismpdc.fpg.unc.edu/content/briefs>) lists as “Evidence-Based Practices for Children and Youth with ASD” the following Structured TEACCHing practices: Antecedent-Based Interventions, Structured Work Systems, and Visual Supports, in addition to widely used practices also used at TEACCH such as prompting, reinforcement, task analysis, functional communication training, social narratives, and parent-implemented training.

Outcome Measurement

Adult outcome research in the field of ASD has included various measures, including standardized test scores (cognitive, language, academic, adaptive, etc.); attainment of an academic high school diploma or college degree; living arrangements; employment status, wages, and benefits; number and quality of social interactions; and individual ratings of life satisfaction (Howlin 2005; Shea and Mesibov 2005). However, long-term outcome research is fairly limited, and there are no peer-reviewed studies comparing the outcome in adulthood of individuals treated in childhood with different treatment approaches.

Qualifications of Treatment Providers

TEACCH center directors have historically been doctoral-level clinical psychologists. The staffs at the centers come from a variety of backgrounds including education, psychology, rehabilitation counseling, social work, and being the parent of a child with ASD. There are no specific licensure requirements.

See Also

- Childhood Autism Rating Scale
- Mesibov, Gary
- Schopler, Eric
- Visual Supports
- Visual Thinking

References and Reading

- Bolick, T. (2001). *Asperger syndrome and adolescence: Helping preteens and teens get ready for the real world*. Gloucester: Fair Winds.
- Bolick, T. (2004). *Asperger syndrome and young children: Building skills for the real world*. Gloucester: Fair Winds.
- Eckenrode, L., Fennell, P., & Hearsey, K. (2003). *Tasks galore*. Raleigh: Tasks Galore. Retrieved from <http://www.tasksgalore.com>.
- Faherty, C. (2000). *What does it mean to me? A workbook explaining self-awareness and life lessons to the child or youth with high functioning autism or Asperger's*. Arlington: Future Horizons.
- Frith, U. (2003). *Autism: Explaining the enigma* (2nd ed.). Oxford: Blackwell.
- Hodgdon, L. A. (1995). *Visual strategies for improving communication: Practical supports for school and home*. Troy: QuirkRoberts.
- Hodgdon, L. A. (1999). *Solving behavior problems in autism: Improving communication with visual strategies*. Troy: QuirkRoberts.
- Howlin, P. (2005). Outcomes in autism spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Diagnosis, development, neurobiology, and behavior, 3rd ed., Vol. 1, pp. 201–220). Hoboken: Wiley.
- Janzen, J. (2003). *Understanding the nature of autism: A guide to the autism spectrum disorders* (2nd ed.). San Antonio: Therapy Skill Builders.
- McAfee, J. (2001). *Navigating the social world: A curriculum for individuals with Asperger's syndrome, high functioning autism and related disorders*. Arlington: Future Horizons.

- Mesibov, G. B., & Shea, V. (2011). Evidence-based practices and autism. *Autism: The International Journal of Research and Practice*, 15(1), 114–133. <https://doi.org/10.1177/1362361309348070>.
- Mesibov, G. B., Shea, V., & Schopler, E. (2005). *The TEACCH approach to autism spectrum disorders*. New York: Springer.
- National Autism Center. (2009). *National standards report*. Randolph: Author.
- Odom, S. L. (2007). *Comparison of two comprehensive treatment models for preschool-aged children with autism spectrum disorders and their families* (Institute of Education Sciences no. R324B07219). [Abstract]. Retrieved from <http://ies.ed.gov/ncser/projects/grant.asp?ProgID=42&grantid=556&NameID=77>
- Reichow, B., Doehring, P., Cicchetti, D. V., & Volkmar, F. R. (2011). *Evidence-based practices and treatments for children with autism*. New York: Springer.
- Shea, V., & Mesibov, G. B. (2005). Adolescents and adults with autism. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Diagnosis, development, neurobiology, and behavior, 3rd ed., Vol. 1, pp. 288–311). Hoboken: Wiley.

Treatment Fidelity

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Synonyms

Accuracy of treatment implementation; Fidelity of implementation; Intervention integrity; Procedural fidelity; Trainer implementation; Treatment adherence; Treatment integrity

Definition

The degree to which a treatment condition is implemented as intended. It refers to whether the treatment condition manipulated the independent variable as planned and is an important methodological component in empirical interventions. A high level of treatment fidelity requires adequate staff training, adherence to intervention procedures, and ongoing support and monitoring of

implementation. Treatment manuals are typically provided to allow for written descriptions of the intervention procedures. Treatment fidelity data and reliability should be provided within scientific reports of a treatment evaluation. It is critical that treatments are routinely assessed for treatment fidelity to ensure that the process is standardized and consistent. Furthermore, the level of treatment fidelity is a factor in treatment replication and generalization across settings.

See Also

► [Procedural Fidelity](#)

References and Reading

- Detrich, R. (1999). Increasing treatment fidelity by matching interventions to contextual variables within the educational setting. *School Psychology Review*, 28(4), 608–620.
- Gearing, R. E., El-Bassel, N., Ghesquiere, A., Baldwin, S., Gillies, J., & Ngeow, E. (2011). Major ingredients of fidelity: A review and scientific guide to improving quality of intervention research implementation. *Clinical Psychology Review*, 31, 79–88.
- McHugh, R. K., Murray, H., & Barlow, D. (2009). Balancing fidelity and adaptation in the dissemination of empirically-supported treatments: The promise of transdiagnostic interventions. *Behavior Research and Therapy*, 47, 946–953.
- Monchor, F., & Prinz, R. (1991). Treatment fidelity in outcome studies. *Clinical Psychology Review*, 11, 247–266.

Treatment for Higher-Order Restricted, Repetitive Behaviors

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Synonyms

[Behavioral flexibility](#); [Flexibility](#)

Definition

Treatments for higher-order restricted, repetitive behaviors (H-RRBs) aim to improve the skills of people with ASD to appropriately vary their behaviors and interests to situational demands. Specifically, to adaptively vary from following preferred idiosyncratic rituals or routines, shift one's activities and topics of conversations from restricted interests, reasonably adjust to changes in routines and schedules, and vary from engaging in repetitive play actions. These treatments focus on addressing impairments in behavioral flexibility observed in many people with ASD. Interventions targeting H-RRBs are aimed to promote skills in flexibly adjusting to situational demands while teaching skills, either directly or indirectly, to appropriately regulate emotions and behaviors to improve the overall adaptability of people with ASD.

Higher-order restricted, repetitive behaviors are considered to be distinct from sensory motor repetitive behaviors given that these sets of behaviors and interests seem to be cognitively mediated rather than driven by sensory and repetitive motor movement needs. In fact, there is evidence indicating that H-RRBs are not as heavily influenced by developmental and language factors as sensory and repetitive motor behaviors are, indicating that H-RRBs may be unique characteristics of ASD, rather than a function of developmental delay.

The rationale for an intervention to target this subcomponent of restricted repetitive behaviors (RRBs) is that impairments in behavior flexibility can contribute to increased functional impairment for people with ASD and their families. Challenges with adaptively and flexibly adjusting from preferred interests and behaviors are associated with emotion dysregulation and behavior problems that can contribute to functional impairment across many areas of life. In children, impairments stemming for H-RRBs have been associated with interfering with social, communication, and adaptive functioning. Difficulty varying from preferred interests and behaviors and appropriately adjusting to situational demands

have been linked with negatively affecting peer and family interactions, interfering with the completion of daily activities in the home, and limiting the overall learning opportunities of people with ASD (e.g., early academic learning).

Developmentally, insistence on sameness behaviors (e.g., need to follow idiosyncratic changes, difficulty with changes) seems to be a common aspect of childhood that begins to emerge and peak in the preschool years and then becomes less prominent as children exit their elementary-school age years. However, in ASD, the degree of emotional reactivity and behavior challenges associated with varying from these interests and behaviors is more frequent and severe and can persist longer across the life span in comparison to typical development. In addition, although these behaviors and interests can be a source of positive feelings and comfort for all people, particularly those with ASD, the emotional and behavioral challenges that arise for people with ASD when they are faced with situations that differ from their H-RRBs can be very limiting and cause a greater degree of distress than that of typically developing people.

Interventions that specifically target behavior flexibility are emerging either as the singular focus or as part of a broader intervention package that spends a portion of the treatment targeting H-RRBs. The interventions commonly include behavioral intervention strategies based on the principles of applied behavior analysis (ABA), such as teaching replacement behaviors, shaping, and differential reinforcement. Some of these interventions are also including additional treatment strategies such as cognitive reappraisal and emotional regulation skills that aim to improve behavior flexibility. These more recent aspects include skill building in the development of adaptive thoughts, problem-solving strategies, and coping strategies. Intervention modalities include group intervention, individual child treatment, and parent-mediated intervention.

As of now additional research on further understanding H-RRBs and the development of effective and empirically supported interventions

targeting this symptom domain of ASD continue to be a growing need. Historically, the focus of the field has been on the social-communication challenges inherent in ASD and developing treatments to target improvement in this area of functioning. Therefore, interventions targeting H-RRBs have been slower to develop, but has been gaining more momentum as the understanding of the impact and nature of H-RRBs has been evolving.

See Also

- [Behaviorism](#)
- [Cognitive Flexibility](#)
- [Functional Behavior-Based Cognitive-Behavioral Therapy for Obsessive-Compulsive Behavior in Children with ASD](#)
- [Operant Conditioning](#)
- [Restricted Interest](#)
- [Shaping of Behavior](#)

References and Reading

- Bishop, S. L., Hus, V., Duncan, A., Huerta, M., Gotham, K., Pickles, A., Kreiger, A., Buja, A., Lund, S., & Lord, C. (2013). Subcategories of restricted and repetitive behaviors in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(6), 1287–1297. <https://doi.org/10.1007/s10803-012-1671-0>. PubMed PMID: 23065116; PubMed Central PMCID: PMC3579001.
- Boyd, B. A., Woodard, C. R., & Bodfish, J. W. (2011). Feasibility of exposure response prevention to treat repetitive behaviors of children with autism and intellectual disabilities: A brief report. *Autism*, 17, 196–204.
- Evans, D. W., Uljarević, M., Lusk, L. G., Loth, E., & Frazier, T. (2017). Development of two dimensional measures of restricted and repetitive behavior in parents and children. *Journal of the American Academy of Child Adolescent Psychiatry*, 56(1), 51–58. <https://doi.org/10.1016/j.jaac.2016.10.014>. Epub 2016 Nov 3. PubMed PMID: 27993229.
- Harrop, C. (2015). Evidence-based, parent-mediated interventions for young children with autism spectrum disorder: The case of restricted and repetitive behaviors. *Autism*, 19(6), 662–672. <https://doi.org/10.1177/1362361314545685>.
- Kenworthy, L., Anthony, L. G., Naiman, D. Q., Cannon, L., Wills, M. C., Luong-Tran, C., Werner, M. A., Alexander, K. C., Strang, J., Bal, E., Sokoloff, J. L., & Wallace, G. L. (2014). Randomized controlled effectiveness trial of executive function intervention for children on the autism spectrum. *Journal of Child Psychology and Psychiatry*, 55(4), 374–383. <https://doi.org/10.1111/jcpp.12161>. Epub 2013 Nov 21. PubMed PMID: 24256459; PubMed Central PMCID: PMC4532389.
- Klin, A., Danovitch, J. H., Merz, A. B., & Volkmar, F. (2007). Circumscribed interests in higher functioning individuals with autism spectrum disorders: An exploratory study. *Research and Practice for Persons with Severe Disabilities*, 32(2), 89–100.
- Lin, C. E., & Koegel, R. L. (2018). Treatment for higher-order restricted repetitive behaviors (H-RRB) in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 48(11), 3831–3845.
- Richler, J., Bishop, S. L., Kleinke, J. R., & Lord, C. (2007). Restricted and repetitive behaviors in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(1), 73–85.
- Troyb, E., Knoch, K., Herlihy, L., Stevens, M. C., Chen, C., Barton, M., et al. (2016). Restricted and repetitive behaviors as predictors of outcome in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 46(4), 1282–1296. <https://doi.org/10.1007/s10803-015-2668-2>.
- Ventola, P. E., Yang, D., Abdullahi, S. M., Paisley, C. A., Braconnier, M. L., & Sukhodolsky, D. G. (2016). Brief report: Reduced restricted and repetitive behaviors after pivotal response treatment. *Journal of Autism and Developmental Disorders*, 46(8), 2813–2820. <https://doi.org/10.1007/s10803-016-2813-6>.

Treatment Gain Retention

- [Maintenance of Treatment Effects](#)

Treatment Integrity

- [Treatment Fidelity](#)

Treatment Preference Assessment

- [Treatment Preference Evaluation](#)

Treatment Preference Evaluation

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Synonyms

Treatment preference assessment

Definition

Treatment preference evaluation is a procedure used to examine the preference between two or more treatment alternatives for individuals with autism and other developmental disabilities. Results of the treatment preference evaluation can help professionals establish treatment acceptability and social validity (Kodak et al. 2016; Yuan et al. 2019). During the evaluation, the individuals are first guided to choose and experience each treatment to ensure that they have been exposed to the options before they make their selection (Hanley et al. 1997). Then, they are asked to choose their preferred treatment followed by the selected treatment session. During the evaluations, the more preferred treatment will have more selections. Previous studies have used this evaluation procedure to reveal preferences of children with autism for various procedures such as response redirection and blocking (Giles et al. 2012) as well as different error-correction arrangements (Kodak et al. 2016; Yuan et al. 2019). Visual stimuli (e.g., colored paper) representing different treatment options were used in these studies for children to make the selections. Presenting treatment alternatives in the visual forms may be particularly useful for those who have limited verbal communication skills so that they can express preferences by

selecting the associated visual stimuli (Yuan et al. 2019).

Even though alternative assessments of treatment acceptability using rating scales and questionnaires can also be completed by stakeholders such as parents and practitioners and may provide valuable input, preferences and values of the stakeholders may not always align with those of the individuals who directly receive the treatment (Hanley et al. 2005). Indeed, existing literature reports that individuals with autism and developmental disabilities sometimes prefer the treatment alternatives that may not be obvious to others (e.g., Hanley et al. 2005; Yuan et al. 2019). Additionally, individual preference may also have an impact on treatment efficacy (Kodak et al. 2016; Yuan et al. 2019). For example, a preferred treatment could lead to a higher level of compliance and engagement. Coupled with effective treatment, the high level of engagement might result in an even greater likelihood of success. By contrast, a treatment that is not preferred can produce noncompliance and problem behavior (Yuan et al. 2019). Thus, practitioners should include opportunities for individuals with autism to show their treatment preferences during the treatment-selection process, especially when the treatment alternatives are equally viable and may produce similar effects.

See Also

► Preference and Reinforcer Assessment

References and Reading

- Giles, A. F., St. Peter, C. C., Pence, S. T., & Gibson, A. B. (2012). Preference for blocking or response redirection during stereotypy treatment. *Research in Developmental Disabilities, 33*(6), 1691–1700. <https://doi.org/10.1016/j.ridd.2012.05.008>.
- Hanley, G. P., Piazza, C. C., Fisher, W. W., Contrucci, S. A., & Maglieri, K. A. (1997). Evaluation of client preference for function-based treatment packages. *Journal of Applied Behavior Analysis, 30*(3), 459–473. <https://doi.org/10.1901/jaba.1997.30-459>.
- Hanley, G. P., Piazza, C. C., Fisher, W. W., & Maglieri, K. A. (2005). On the effectiveness of and preference for punishment and extinction components of function-based interventions. *Journal of Applied Behavior Analysis, 38*(1), 51–65. <https://doi.org/10.1901/jaba.2005.6-04>.

- Kodak, T., Campbell, V., Bergmann, S., LeBlanc, B., Kurtz-Nelson, E., Cariveau, T., et al. (2016). Examination of efficacious, efficient, and socially valid error-correction procedures to teach sight words and prepositions to children with autism spectrum disorder. *Journal of Applied Behavior Analysis*, 49(3), 532–547. <https://doi.org/10.1002/jaba.310>.
- Yuan, C., Hua, Y., & Zhu, J. (2019). The role of reinforcement in multiple response repetition error correction and treatment preference of Chinese children with autism. *Journal of Autism and Developmental Disorders*, 49(9), 3704–3715. <https://doi.org/10.1007/s10803-019-04086-x>.

Treatment Priorities

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Definition

Treatment priorities must be individualized and consider the unique needs of the individual with autism as well as the needs of the relevant stakeholders in the individual's natural environments. Particularly important in developing treatment priorities is social validation or the concept that the most important and beneficial behaviors to teach should result in a valuable and meaningful difference in the individual's life. It is recommended that the individual with autism's own personal goals and expectations are addressed and incorporated accordingly into the treatment plan. The treatment priorities of the parents or caretakers for the individual with autism are also an important aspect of intervention, and their concerns should be taken into account. In order to address this issue, it is important that the individuals themselves, their parents, caretakers, and other members of the individual's support group actively partake in contributing to the treatment priorities. Research suggests that

parents and caretakers rank skills such as making friends, pedestrian safety skills, and appropriate interaction skills as a part of their top ten priorities in the treatment outcome of the individual with autism. Thus, treatment priorities relating to safety, social skills, communication, and academics, among others, should be addressed.

All treatment priority goals should include quantifiable variables so that response to intervention and the intervention itself can be empirically and objectively evaluated. The success of treatment should not be contingent merely on the reduction of negative behavior but also on the increase of and improvements in positive behaviors.

The acceptability and ease of intervention implementation for the individual with autism must also be considered so that treatment is introduced as smoothly and efficiently as possible. Intervention procedures should always be executed in a way such that the individual with autism is treated with respect and dignity. Moreover, interventions should reflect an individualized and age-appropriate treatment plan in order to facilitate optimal social integration with peer groups. In addition, the implementation of treatment should be achieved in a naturalistic and comfortable way so that intervention can be executed consistently and frequently throughout the waking hours of the individual with autism's day for faster gains and greater generalization and maintenance.

Also important when considering treatment priorities are ecocultural variables. This necessitates that treatment goals are developed within an individual's natural settings and with consideration of cultural and psychological diversity. Another important consideration in developing treatment priorities is the least restrictive environment. That is, if a behavior is likely to result in the placement of an individual in a more restrictive educational or community setting, the modification of such behavior would be a treatment priority. Individuals with autism should have the opportunity to engage in and have access to the same basic privileges as his or her respective peers, and the overarching goal of treatment is to achieve this level of equality.

References and Reading

- Berry, J. W. (2001). Contextual studies of cognitive adaptation. In J. M. Collis & S. Messick (Eds.), *Intelligence and personality: Bridging the gap in theory and measurement* (pp. 319–333). Mahwah: Lawrence Erlbaum Associates.
- Crockett, J. B. (1999). *The least restrictive environment: Its origins and interpretations in special education*. Mahwah: Lawrence Erlbaum Associates.
- Kazdin, A. E. (1977). Assessing the clinical or applied importance of behavior change through social validation. *Behavior Modification, 1*, 427–452.
- Koegel, L. K., Dunlap, G., & Koegel, R. L. (1996). *Positive behavioral support: Including people with difficult behavior in the community*. Baltimore: Paul H. Brookes.
- Koegel, L., Matos-Freeden, R., Lang, R., & Koegel, R. (2011). Interventions for children with Autism spectrum disorders in inclusive school settings. *Cognitive and Behavioral Practice, 18*(3), 421–588.
- Lord, C. (1988). Enhancing communication in adolescents with Autism. *Topics in Language Disorders, 9*, 72–81.
- Pituch, K. A., Green, V. A., Didden, R., Lang, R. B., O'Reilly, M. F., Lancioni, G. E., et al. (2011). Parent reported treatment priorities for children with Autism spectrum disorders. *Research in Autism Spectrum Disorders, 5*, 135–143.

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Short Biography

Dr. Darold Treffert is the world's leading authority on the autistic savant syndrome – a term he coined. He has been studying savant syndrome since he met his first savant in 1962, when he developed a children's unit at a hospital in Wisconsin, USA. He is a clinical professor in the Department of Psychiatry at the University of Wisconsin and on the staff of the Behavioral Health Department of St. Agnes Hospital in Fond du Lac, Wisconsin. He maintains a very active web site on savant syndrome at www.savantsyndrome.com through the Wisconsin Medical Society. He is also the author of *Extraordinary People: Understanding Savant Syndrome* (1989) – the first comprehensive book on the condition – and *Islands of Genius: The Bountiful Mind of the Autistic, Acquired and Sudden Savant* (2010). He

was a consultant on the movie, *Rain Man*, in which Dustin Hoffman won an Oscar for his portrayal of an autistic savant.

Dr. Treffert's work has appeared in numerous publications, including *Time*, *People*, *Newsweek*, *USA Today* and *Scientific American*, and he has participated in many documentaries on savant syndrome and autistic disorder. He also has over 50 articles in professional journals. He has been listed in *The Best Doctors in America*, by peer selection, beginning in 1979.

References and Reading

- Treffert, D. A. (1988). The idiot savant: A review of the syndrome. *American Journal of Psychiatry, 145*(5), 563–572.
- Treffert, D. (1989). *Extraordinary people*. New York: Bantam.
- Treffert, D. (2010). *Islands of genius: The bountiful mind of the autistic, acquired and sudden savant*. London: Jessica Kingsley.
- Treffert, D. A., & Wallace, G. L. (2002). Islands of genius. Artistic brilliance and a dazzling memory can sometimes accompany autism and other developmental disorders. *Scientific American, 286*(6), 76–85.

Trexan

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Synonyms

[Naltrexone](#)

Definition

Trexan or naltrexone is an opiate antagonist used as an adjunct in the treatment of opioid dependence. It became of interest in the 1970s and

1980s as a potential treatment for autism based on the potential role of the endogenous opioid system in autism. There have been several small studies using naltrexone in children with autism. These studies indicate that the medication is generally well tolerated but did not rarely shown to be beneficial for any outcome measured. Outcomes that have been of the interest in these studies include an improved communication, decreased self-injurious behavior, and decreased hyperactivity. The adverse effects of naltrexone include loss of energy, drowsiness, and dizziness.

References and Reading

Ristow, A., Westphal, A., & Scahill, L. (2011). Treating hyperactivity in children with pervasive developmental disorders. In E. Hollander, A. Kolevzon, & J. T. Coyle (Eds.), *Textbook of autism spectrum disorders* (pp. 479–492). Arlington: American Psychiatric Publishing.

TRIAD Social Skills Assessment

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Synonyms

Vanderbilt Treatment and Research Institute for Autism Spectrum Disorders (TRIAD) Social Skills Assessment (TSSA)

Description

The social challenges that children with autism experience are pervasive and lifelong. With direct intervention, children with autism can acquire the tools necessary for interacting with others. Many researchers have documented the beneficial effects of teaching social skills to children with autism.

Children with autism experience specific social challenges that are different from children with other developmental disabilities, such as understanding emotions and how to recognize and communicate feelings, knowing how to start and maintain interactions appropriately, and understanding other people's perspectives. Because of their unique challenges, many of the tools available for assessing social skills do not address the level of detail needed for generating program plans for children with autism. In order to teach social skills, we need to determine what each child's individual needs are in order to monitor the effectiveness of teaching plans.

The TSSA is designed for children ages 6–12 years who have basic reading skills at the first grade level. It is criterion-based and assesses knowledge and skills in three areas: (a) cognitive, (b) behavioral, and (c) affective. The *cognitive* areas assess the child's ability to understand other people's perspectives. The *behavioral* aspects determine the child's ability to initiate and maintain interactions and respond appropriately to other people. The *affective* components evaluate the child's abilities to understand basic and complex emotions.

Four sources of information are incorporated into the assessment: (1) parent report, (2) teacher report, (3) observation, and (4) direct child interaction. The parent information provides descriptions of the child's social skills in home and community settings. The teacher information provides descriptions of social skills in the school setting. Parents and teachers also both rate interfering behaviors exhibited by the child. Finally, the direct child assessment provides additional information of the child's skills as observed during interaction with a clinician.

The information from the various sources should be interpreted in terms of the child's

strengths and challenges. The TSSA provides a sample report that can be used to identify teaching goals for school, home, and clinical settings. The TSSA can be downloaded from the following web page: <http://uwreadilab.com/tools-materials/>.

Historical Background

The Vanderbilt Treatment and Research Institute for Autism Spectrum Disorders (TRIAD) was established within the Department of Pediatrics at Vanderbilt University Medical Center in 1998 by Dr. Wendy Stone. Dr. Stone recognized a need in the community for outreach and intervention services for children with autism. In 2005, TRIAD became a partner with the Vanderbilt Kennedy Center for Research on Human Development in order to continue to develop larger and more integrated autism research, training, and clinical programs.

The TSSA was developed originally by TRIAD autism specialists to address the need for a relatively brief, easy-to-administer tool for evaluating the complex social profiles of children with autism spectrum disorders, identifying strengths and challenges in the social domain, and providing recommendations for intervention planning through individualized goals and specific strategies. Used in-house for many years, the TSSA recently has been updated for use in a variety of settings, including the school and community.

Psychometric Data

There are currently no research studies that provide normative or quantitative information about a child's individual performance on the parent and teacher questionnaires or specific testing items. However, very important and helpful information can be gathered from each section of this assessment that will guide recommendations for treatment interventions.

Clinical Uses

The TSSA does not require specific training or a degree. However, the examiner should have

knowledge about the characteristics of autism as well as communication and social skill development. The intent is that this instrument be administered by special educators, speech pathologists, licensed therapists (e.g., occupational therapist, physical therapist), or school psychologists.

The TSSA is an integrated assessment that provides valuable information about a child's social and behavioral strengths and concerns. The information obtained from each section of the TSSA is meant to be a subjective and qualitative guide about the child's functioning in multiple contexts, including home, school, and the direct assessment setting.

The assessment report should include the following sections: background and referral information, a description of the assessment instrument and the child's participation and effort, interaction style, and interfering behaviors, as well as summaries of parent and teacher reports and direct child interaction, and recommendations for goals and interventions. A guideline for the interpretation and content of each section is specified in the manual.

See Also

- [Social Behaviors and Social Impairment](#)
- [Social Interventions](#)
- [Social Responsiveness Scale](#)
- [Social Skill Interventions](#)

References and Reading

- Barnhill, G. P., Cook, K. T., Tebbenkamp, K., & Myles, B. S. (2002). The effectiveness of social skills intervention targeting nonverbal communication for adolescents with Asperger syndrome and related pervasive developmental delays. *Focus on Autism and Other Developmental Disabilities*, 17(2), 112–118.
- Bary, T. D., Klinger, L. G., Lee, J. M., Palardy, N., Gilmore, T., & Bodin, S. D. (2003). Examining the effectiveness of an outpatient clinic-based social skills group for high-functioning children with autism. *Journal of Autism and Developmental Disorders*, 33(6), 685–701.
- Bauminger, N. (2007). Brief report: Individual social-multiple modal intervention for HFASD. *Journal of Autism and Developmental Disorders*, 37(8), 1593–1604.
- Crager, D. E., & Horvath, L. S. (2003). The application of social skills training in the treatment of a child with Asperger's disorder. *Clinical Care Studies*, 2(1), 34–49.

- Hwang, B., & Hughes, C. (2000). The effects of social interactive training on early social communicative skills of children with autism. *Journal of Autism and Developmental Disorders*, 30(4), 331–343.
- Kroeger, K. A., Schultz, J. R., & Newsom, C. (2007). A comparison of two group-delivered social skills programs for young children with autism. *Journal of Autism and Developmental Disorders*, 37(5), 808–817.
- Lopata, C., Thomeer, M. L., Volker, M. A., & Nida, R. E. (2006). Effectiveness of a cognitive-behavioral treatment on the social behaviors of children with Asperger disorder. *Focus on Autism and Other Developmental Disabilities*, 21(4), 237–244.
- Murray, D. S., Ruble, L. A., Willis, H., & Molloy, C. A. (2009). Parent and teacher report of social skills in children with autism spectrum disorders. *Language, Speech, and Hearing Services in Schools*, 40, 109–115.
- Ruble, L. A., Willis, H., & Crabtree, V. (2008). Social skills group therapy for autism spectrum disorders. *Journal of Clinical Case Studies*, 7, 287–300.
- Tse, J., Strulovitch, J., Tagalakis, V., Meng, L., & Fombonne, E. (2007). Social skills training for adolescents with Asperger syndrome and high-functioning autism. *Journal of Autism and Developmental Disorders*, 37(10), 1960–1968.

Trial

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Definition

A (discrete) trial represents an isolated opportunity for an organism to make a single operant response in the presence of a discriminative stimulus. Each discrete trial must contain three components: an antecedent discriminative stimulus, a response, and a consequence. Successive trials are separated by intertrial intervals during which no discriminative stimuli are presented and operant responses are either precluded or are not reinforced.

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Skinner, B. F. (1953). *Science and human behavior*. New York: Macmillan.

Triaminic Thin Strips® Children's Cough and Runny Nose [OTC]

► [Diphenhydramine](#)

Triazolam and Autism

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Synonyms

[Halcion](#)

Definition

Sedation of intellectually disabled patients, such as autism spectrum disorders (ASD), is often applied before invasive or lengthy surgical procedures (Lim et al. 2018). It is estimated that 30% of patients with intellectual disabilities are uncooperative before receiving general anesthesia (Lim et al. 2018). In order to avoid hard restraints, three general sedatives, midazolam, ketamine, and dexmedetomidine, are utilized to calm ASD patients before receiving general anesthesia (Lim et al. 2018). Recently, physicians began administering triazolam after government restrictions on midazolam use in certain countries. To determine whether triazolam was equally efficacious as midazolam, a study consisting of 97 patients with intellectual disabilities were divided into two groups receiving either midazolam or triazolam. Analysis of cooperation after drug administration showed anesthesia induction without physical restraint was possible in 56.0% of patients in the midazolam group and in 46.8% of patients in the triazolam group ($P = 0.312$) (Lim et al. 2018). Therefore, triazolam is an equivalent alternative for sedating uncooperative patients before receiving general anesthesia. However, sedatives pose

an increased risk of respiratory depression, hypoxia, or pulmonary aspiration (Lim et al. 2018). It is encouraged for clinicians to pursue alternative means for calming ASD patients using family or other close caregivers throughout the pre- and postoperative stages of surgical intervention rather than sedation.

See Also

- Midazolam
- Zyprexa

References and Reading

Lim, S. W., So, E., Yun, H. J., Karm, M.-H., Chang, J., Lee, H., . . . Seo, K.-S. (2018). Analysis of the effect of oral midazolam and triazolam premedication before general anesthesia in patients with disabilities with difficulty in cooperation. *Journal of Dental Anesthesia and Pain Medicine*, 18(4), 245. <https://doi.org/10.1724/jdapm.2018.18.4.245>.

Trichotillomania

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Synonyms

Chronic hairpulling

Short Description or Definition

Trichotillomania is characterized by recurrent hairpulling that causes noticeable hair loss and significant distress or impairment. A diagnosis of trichotillomania according to DSM-IV also requires that an individual sometimes experience urges prior to pulling and a sense of pleasure, gratification, or relief after pulling. Individuals who do not report urges prior to and a sense of relief after pulling are generally given a diagnosis

of chronic hairpulling. Inability to report sensory phenomenon is common and developmentally appropriate in younger children; thus, there is a strong probability that these diagnostic criteria will be removed from future diagnostic criteria.

Categorization

Impulse Control Disorder

Epidemiology

Trichotillomania has an estimated lifetime prevalence of roughly 0.6%. However, the estimated lifetime prevalence increases to roughly 3% when preceding urges and pleasure/relief afterward are removed from the diagnostic criteria. The prevalence of trichotillomania in patients with pervasive developmental disorders is consistent with these estimates.

Trichotillomania patients have a high rate of comorbid illnesses, with approximately 82% of adults presenting for treatment experiencing at least one other axis I psychiatric disorder (Woods et al. 2006a). Common comorbidities include depression, anxiety disorders, obsessive-compulsive disorder, substance use disorders, posttraumatic stress disorder, and other body-focused impulse control disorders. Trichotillomania in adults has a strong female predominance and a chronic, waxing and waning course.

Natural History, Prognostic Factors, and Outcomes

Trichotillomania has an average age of onset around 13 years. Longitudinal studies of adults with trichotillomania have demonstrated little improvement in symptoms over time (Keuthen et al. 2001). Longitudinal studies have not been conducted in order to establish the long-term prognosis of children with trichotillomania, although it is clear from adult samples that a majority of trichotillomania cases arise in childhood. Hairpulling in young children (less than

5 years of age) is considered a behavior consistent with developmentally appropriate environmental exploration and is usually self-limited. It is currently unclear how the discovery and implementation of therapies with some evidence of short-term efficacy for treating trichotillomania will affect the long-term prognosis of the illness.

Evaluation and Differential Diagnosis

A proper assessment in a patient with trichotillomania involves getting a detailed history of their hairpulling. When discussing hairpulling behaviors, it is important to discuss (1) antecedent cognitions, behaviors, and feelings prior to pulling, (2) the settings in which pulling occurs, (3) body locations from which pulling occurs, and (4) postpulling behavior. Trichotillomania patients commonly experience emotions such as boredom, tension, and anxiety prior to hairpulling episodes and/or a physical urge to pull. Commonly experienced cognitions prior to hairpulling include beliefs about the inappropriate appearance of certain hairs (gray, coarse, etc.), that hairlines or lengths of hair need to be symmetrical, or that she is unattractive or unlovable because of her appearance. Common postpulling behaviors involve biting, rubbing, or eating the hair. Also, discarding of the hairs in fairly stereotyped ways is the norm. In patients who ingest their own hair, trichobezoars – conglomerations of hair and food that form in the gastrointestinal tract – are of particular concern as they can lead to weight loss, iron deficiency anemia, malabsorption, and even gastrointestinal tract obstructions. Frequently, younger children and individuals with developmental disabilities will have difficulty reporting these feelings. In these cases, having a parent journal the child's hairpulling behaviors can be effective.

Trichotillomania patients also usually have specific places where they pull, i.e., the bedroom or the bathroom. The most common sites of hairpulling are the scalp (73%), eyebrows (56%), eyelashes (53%), pubic region (46%), and legs (15%) (Woods et al. 2006a). Adults with trichotillomania tend to pull from more sites than

children. A physical examination of areas of hairpulling can uncover areas of irritation, follicle damage, and atypical regrowth of hair that are common in patients with trichotillomania. Rating scales such as the Massachusetts General Hospital Hairpulling Scale, a patient self-report form, and the National Institute of Health Trichotillomania Severity Scale, a clinician-rated form, are useful in measuring the severity of trichotillomania symptoms and tracking changes in symptom severity over time.

Treatment

Nonpharmacological behavioral treatments should be regarded as the treatment of choice for both children and adults with trichotillomania. Habit reversal therapy (HRT) is a behavioral therapy designed for the treatment of trichotillomania and tics. HRT is a manualized, behavioral technique that is administered over a period of 2–3 months with a maintenance period for relapse prevention. HRT involves several different components – self-monitoring, awareness training, stimulus control, and competing response training. The self-monitoring component of HRT requires patients to keep records of their hairpulling. Awareness training works to increase patient awareness of hairpulling behaviors and of high-risk situations that increase the risk of hairpulling. Stimulus control employs interventions designed to decrease the opportunities to pull and to intervene or prevent pulling behaviors. Competing response training involves teaching a patient to engage in a physically incompatible behavior with pulling for a set period of time when they feel the urge to pull. In HRT, patients are only permitted to pull after they have engaged in the competing response behaviors.

In three randomized, parallel-group studies, HRT demonstrated superior efficacy compared to wait-list or placebo controls (Ninan et al. 2000; van Minnen et al. 2003; Woods et al. 2006b). HRT also demonstrated superiority to pharmacotherapy with fluoxetine and clomipramine in two of these randomized trials (Ninan et al. 2000; van Minnen et al. 2003).

A meta-analysis demonstrated that in randomized, controlled trials of trichotillomania, HRT has demonstrated superior effect sizes compared to pharmacological agents for trichotillomania (Bloch et al. 2007). Further trials are needed to demonstrate that HRT will maintain efficacy when compared to control conditions that account for the nonspecific aspects of therapy (i.e., supportive psychotherapy and psychoeducation).

The main problem with the use of HRT to treat trichotillomania is the lack of availability of clinicians experienced in this behavioral technique. It is unknown the degree to which HRT efficacy is diminished when it is practiced by clinicians less experienced in HRT and trichotillomania. An online website, stoppingpulling.com, provides many of the common HRT techniques in a self-help format for motivated patients who do not have access to an experienced therapist. Furthermore, many patients with trichotillomania do not experience remission of symptoms with behavioral therapy and thus other treatments are needed. Conducting HRT in patients with developmental or intellectual disability is challenging and remains understudied.

Selective serotonin reuptake inhibitors (SSRI) are the most commonly utilized intervention to treat trichotillomania (Woods et al. 2006a). Initial open-label trials showed improvement over time in trichotillomania patients taking SSRI. However, in four randomized, blinded, placebo-controlled trials, SSRIs have failed to show benefit compared to placebo (Christenson et al. 1991; Dougherty et al. 2006; Streichenwein and Thornby 1995; van Minnen et al. 2003). Meta-analysis which combined the results of the four previously conducted trials similarly failed to show any evidence of an improvement compared to placebo treatment (Bloch et al. 2007). Although there is substantial evidence that pharmacological treatment with SSRI is no more effective than placebo in the treatment of primary hairpulling in trichotillomania patients, these medications still may be quite effective in treating comorbid illness in these patients. Depression, anxiety disorders, and posttraumatic stress disorder are all common comorbidities in trichotillomania patients and have substantial evidence

demonstrating improvements with SSRI pharmacotherapy. When SSRI pharmacotherapy is initiated in a trichotillomania patient, the goal of therapy should be to specifically target comorbid illness that is impairing to the patient, as SSRI pharmacotherapy for primary trichotillomania has little evidence of efficacy.

Clomipramine is a tricyclic antidepressant (TCA) that has been extensively studied in the treatment of trichotillomania. Initial results from a 10-week, randomized, double-blind, crossover study of 13 women which compared clomipramine, a serotonergically selective TCA, to desipramine, a nonselective TCA, demonstrated substantial improvement in trichotillomania patients treated with clomipramine (Swedo et al. 1989). However, most of the patients in this trial experience a relapse in their symptoms after longer-term treatment with clomipramine. Another small, 9-week, randomized, placebo-controlled parallel group study showed some increased improvement of trichotillomania symptoms compared to placebo but not to the level of statistical significance (Ninan et al. 2000). Clomipramine was very poorly tolerated in this study, with 40% of subjects dropping out early due to side effects. Common side effects associated with clomipramine include weight gain, anticholinergic symptoms, and sedation. Meta-analysis of randomized trials with clomipramine suggests modest short-term benefits when compared to control conditions. However, evidence suggests that benefits from clomipramine may be short-lived. Randomized trials have suggested that both SSRIs and clomipramine are inferior to HRT.

A double-blind placebo-controlled trial in 50 adults with trichotillomania demonstrated the efficacy of N-acetylcysteine (NAC), a glutamate-modulating agent with antioxidant properties. Fifty-six percent of the subjects given NAC demonstrated significant improvement at 12 weeks compared with only 16% in the placebo group (Grant et al. 2009). N-acetylcysteine showed statistically significant improvement compared with placebo by 6 weeks of treatment. There are no published reports of the use of NAC to treat childhood TTM. However, NAC has been used safely with minimal side effects in the

treatment of acetaminophen overdose in children. N-acetylcysteine doses used to treat acetaminophen overdose are orders of magnitude higher than the 2,400 mg/day suggested for TTM. Possible side effects of NAC include nausea, flatulence, and asthma exacerbation. NAC is available as an over-the-counter supplement for a cost of \$5–15 a month.

A randomized, double-blind, placebo-controlled trial of 25 adults with trichotillomania has also demonstrated the efficacy of olanzapine. Eighty-five percent of subjects receiving olanzapine reported significant improvement compared to 17% in the placebo group after 12 weeks of treatment (Van Ameringen et al. 2006). Olanzapine also demonstrated efficacy on primary measures of trichotillomania in the trial. Because of the significant adverse effects associated with olanzapine such as weight gain, sedation, and metabolic syndromes, even if effective, olanzapine is unlikely to be a first-line treatment for trichotillomania. Case series and case reports have suggested the efficacy of other antipsychotics; however, none have been studied in randomized controlled trials. Most other antipsychotic agents also share a similar side effect profile to olanzapine.

Case report level data suggests the possible efficacy of other pharmacological agents such as riluzole, topiramate, and naltrexone. Skepticism is warranted when viewing the likely efficacy of any treatment of trichotillomania studied in an unblinded, uncontrolled fashion. Many patients with trichotillomania improve over the short term regardless of treatment after presenting for initial treatment. Greater familiarity and psychoeducation about the disorder, meeting other individuals with the disorder, supportive clinicians, and engaging in any active intervention against the disorder are all powerful forces toward patient improvement. These aspects of treatment along with the waxing and waning course of trichotillomania make it difficult to assess efficacy in uncontrolled trials.

Support groups can also be very helpful for treatment for trichotillomania. The Trichotillomania Learning Center based in Santa Cruz, California, can be quite helpful in providing treatment

referrals and support to individuals experiencing trichotillomania. Often times, hearing other individuals' stories, coping mechanisms, and strategies for trichotillomania can be quite helpful in living with and overcoming this illness. The Trichotillomania Learning Center can be contacted through their website at www.trich.org.

References and Reading

- Bloch, M. H., Landeros-Weisenberger, A., Dombrowski, P., Kelmendi, B., Wegner, R., Nudel, J., et al. (2007). Systematic review: Pharmacological and behavioral treatment for trichotillomania. *Biological Psychiatry*, 62(8), 839–846.
- Christenson, G. A., Mackenzie, T. B., Mitchell, J. E., & Callies, A. L. (1991). A placebo-controlled, double-blind crossover study of fluoxetine in trichotillomania. *American Journal of Psychiatry*, 148(11), 1566–1571.
- Dougherty, D. D., Loh, R., Jenike, M. A., & Keuthen, N. J. (2006). Single modality versus dual modality treatment for trichotillomania: Sertraline, behavioral therapy, or both? *Journal of Clinical Psychiatry*, 67(7), 1086–1092.
- Grant, J. E., Odlaug, B. L., & Kim, S. W. (2009). N-acetylcysteine, a glutamate modulator, in the treatment of trichotillomania: A double-blind, placebo-controlled study. *Archives of General Psychiatry*, 66(7), 756–763.
- Keuthen, N. J., Fraim, C., Deckersbach, T., Dougherty, D. D., Baer, L., & Jenike, M. A. (2001). Longitudinal follow-up of naturalistic treatment outcome in patients with trichotillomania. *Journal of Clinical Psychiatry*, 62(2), 101–107.
- Ninan, P. T., Rothbaum, B. O., Marsteller, F. A., Knight, B. T., & Eccard, M. B. (2000). A placebo-controlled trial of cognitive-behavioral therapy and clomipramine in trichotillomania. *Journal of Clinical Psychiatry*, 61(1), 47–50.
- Streichenwein, S. M., & Thornby, J. I. (1995). A long-term, double-blind, placebo-controlled crossover trial of the efficacy of fluoxetine for trichotillomania. *American Journal of Psychiatry*, 152(8), 1192–1196.
- Swedo, S. E., Leonard, H. L., Rapoport, J. L., Lenane, M. C., Goldberger, E. L., & Cheslow, D. L. (1989). A double-blind comparison of clomipramine and desipramine in the treatment of trichotillomania (hair pulling). *New England Journal of Medicine*, 321(8), 497–501.
- Van Ameringen, M. A., Mancini, C. L., Collins, S., Oakman, J. M., & Farvolden, P. (2006). A 12-week, double-blind trial of olanzapine and placebo in the treatment of trichotillomania. Paper presented at the American Psychiatric Association 159th annual meeting, Toronto.
- van Minnen, A., Hoogduin, K. A., Keijsers, G. P., Hellenbrand, I., & Hendriks, G. J. (2003). Treatment of trichotillomania with behavioral therapy or

fluoxetine: A randomized, waiting-list controlled study. *Archives of General Psychiatry*, 60(5), 517–522.

Woods, D. W., Flessner, C. A., Franklin, M. E., Keuthen, N. J., Goodwin, R. D., Stein, D. J., et al. (2006a). The Trichotillomania Impact Project (TIP): Exploring phenomenology, functional impairment, and treatment utilization. *Journal of Clinical Psychiatry*, 67(12), 1877–1888.

Woods, D. W., Wetterneck, C. T., & Flessner, C. A. (2006b). A controlled evaluation of acceptance and commitment therapy plus habit reversal for trichotillomania. *Behaviour Research and Therapy*, 44, 639–656.

Tricyclic Antidepressants (TCAs)

► Antidepressants

Triennial Evaluation and Triennial Review

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Synonyms

Reevaluation

Definition

A review of the progress of a student receiving special education services must be conducted at least every 3 years, or more frequently if requested, by the school's Individual Education Planning (IEP) team in order to determine whether the student continues to be eligible for special education services. If, at that time, the IEP team determines that additional information is needed to make this determination, a triennial evaluation will be conducted. The triennial evaluation typically consists of the use of a combination of formal and informal assessment data by a number of professionals within the school

system. Written parental consent must be provided before the evaluations can be completed. For people with Autism Spectrum Disorders (ASD), a comprehensive assessment typically evaluates functioning in a number of areas including cognition, communication, academic achievement, and social-emotional and behavioral development. As part of the triennial review process, assessments are completed in specific areas of concerns. This may include psychological, educational, speech and language, occupational therapy, or physical therapy evaluations by school professionals.

See Also

- Individual Education Plan
- Individuals with Disabilities Education Act (IDEA)
- Special Education

References and Reading

- Goldstein, S., Naglieri, J. A., & Ozonoff, S. (2009). *Assessment of autism spectrum disorders*. New York: The Guilford Press.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: The National Academy of Sciences.
- United States Department of Education, Office of Special Education Programs. (2004). Changes in initial evaluation and reevaluation. Retrieved from <http://idea.ed.gov/>.

Trifluoperazine

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Synonyms

Stelazine™

Definition

One of the older (first-generation) neuroleptics of the phenothiazine group. This agent has been used most widely in the treatment of schizophrenia and less frequently for other conditions, e.g., Alzheimer's. This agent was occasionally used in the past for treatment of agitation/stereotyped movements in autism. With other agents in this class, it impacts various systems as with other agents in this group; side effects can include movement problems (including tardive dyskinesia). It may also lower the seizure threshold (a particular issue for individuals with autism and seizures). Its primary neurochemical action to be blockade of dopamine D₁ and D₂ receptors in the brain. In autism, the use of this, and similar first-generation neuroleptics, has largely been replaced by the newer "second-generation" (atypical) neuroleptics, e.g., risperidone.

See Also

- [Dopamine](#)
- [Risperidone](#)

References and Reading

- Joshi, G., Petty, C., Wozniak, J., Henin, A., Fried, R., Galdo, M., et al. (2010). The heavy burden of psychiatric comorbidity in youth with autism spectrum disorders: A large comparative study of a psychiatrically referred population. *Journal of Autism & Developmental Disorders*, 40(11), 1361–1370.
- McCracken, J. T., McGough, J., Shah, B., Cronin, P., Hong, D., Aman, M. G., et al. (2002). Risperidone in children with autism and serious behavioral problems. *New England Journal of Medicine*, 347(5), 314–321.
- Scahill, L., & Martin, A. (2005). Psychopharmacology. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 2, pp. 1102–1122). Hoboken: Wiley.
- Volkmar, F., & Wiesner, L. (2009). *A practical guide to autism*. Hoboken: Wiley.

Trilafon

- [Perphenazine](#)

Trisomy 21

- [Down Syndrome](#)

True Negative Rate

- [Sensitivity and Specificity](#)

True Positive Rate

- [Sensitivity and Specificity](#)

Trust

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Definition

In General

A trust is a financial arrangement whereby one person, the trustee, holds property (usually in the form of money) for the benefit of another person, the beneficiary. The person who establishes the trust and whose property is held in the trust is called the settlor. The trustee has legal title to the trust property (also known as the "corpus" of the trust) which allows him to invest the money as a prudent investor would. Over the years, specialized trusts emerged to fit the unique needs of individuals and they currently number in the hundreds. Trusts are particularly useful for ensuring responsible inheritance management, avoiding certain estate and gift taxes and preventing the need for a guardian in the case of incapacity.

To illustrate the basic formulation and function of a trust, let us assume that A and B are the parents of a child, C, diagnosed with an ASD. In order to assure that C is financially supported, either during their lifetime or after their death,

A and B set up a trust through trust company T.C., Inc. wherein an employee of T.C., Inc. will be charged with dealing with the day-to-day management of the trust. A and B then transfer \$1 million into the trust and the trustee; T.O. is bound by the trust document to manage and distribute the money for the sole benefit of C, the beneficiary of the trust.

Important Considerations and Financial Planning

Because a trust is a legal agreement that represents an understanding of a financial arrangement, it is usually (and, in some jurisdictions, required to be) in written form. Thus, the settlor may customize the trust to meet his or her individual needs. Using our example from earlier, A and B can enter a provision into the trust agreement that requires the trustee to distribute money only to pay for the expenses of C that are a direct result of their ASD treatment.

Trusts are an ideal mechanism for protecting assets. First, trust agreements can be structured (most commonly through the inclusion of a so-called spendthrift clause) to create discretionary trusts that shelter the trust assets from creditors. In a discretionary trust, the settlor can postpone and delegate to the trustee the decision of to whom to make distributions, in what amounts, and, more importantly, when. Say, for example, that C incurs a large amount of debt and C's creditors wish to go after the money in the trust that A and B set up for C. A spendthrift clause prohibits the creditor from satisfying the debt from the trust assets because the trustee has the sole authority to distribute the assets.

See Also

- [Beneficiary](#)
- [Discretionary Trust](#)
- [Support Trust](#)
- [Trustee](#)

References and Reading

Beyer, G. W. (2007). *Wills, trusts, and estates: Examples and explanations* (4th ed.). New York: Aspen.

Dukeminier, J., Sitkoff, R., & Lindgren, J. (2009). *Wills, trusts, and estates* (8th ed.). New York: Aspen.

Garner, B. A. (2009). Black's law dictionary. In B. A. Garner (Ed.), *Black's law dictionary* (9th ed., pp. 1647–1648). Dallas: West Publishing.

Larson, A. (n.d.). *The living trust*. Retrieved March 10, 2011, from ExpertLaw.Com. http://www.expertlaw.com/library/estate_planning/trusts.html

Trustee

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Definition

A trustee is a person or entity who holds the assets of a trust for the benefit of a beneficiary. A person who becomes a trustee is granted legal title of a trust and is responsible for managing and controlling all trust assets for the benefit of the beneficiary.

Serving the Role of Trustee

In order to become a trustee, a person must be competent and of legal age. Anyone with the ability to own and transfer property can serve the role of trustee. Once named a trustee, a person must be identified as such by the trust document and the trustee must formally accept the role. Depending on the state, a trustee may be required to take a sworn oath of office and/or post a bond in order to accept the role.

Trustee Duties and Obligations

A trustee's key responsibility involves controlling and protecting the trust. In administering the trust, the trustee must exercise the highest degree of care and he/she has a duty of loyalty to the trust and its beneficiary. The trustee must act in good faith and avoid any conflicts of interest. The trustee must follow any instructions provided by the person (s) who established the trust. The trustee must also exercise great care in financial transactions involving the trust. All transactions must be accurately recorded by the trustee and accounting must be provided to the beneficiary.

Multiple Trustees

There can be more than one person acting as trustee of a trust. If there is more than one trustee, all designated trustees must make joint decisions regarding the trust. Some states require all trustees to be in agreement before any decision related to the trust is made. Other states require only a majority of the trustees to agree on a decision.

Compensation of Trustees

A trust may or may not explicitly state whether a trustee is entitled to compensation. In cases where the trust is silent on the issue of compensation, many states have laws which set forth that a trustee is entitled to some compensation.

See Also

- [Beneficiary](#)
- [Discretionary Trust](#)
- [Support Trust](#)
- [Trust](#)

References and Reading

- Dukeminier, J., Sitkoff, R. H., & Lindgren, J. (2009). *Wills, trusts, and estates* (8th ed.). New York: Aspen.
- Garner, B. A. (2009). *Black's law dictionary* (9th ed.). St. Paul: West Publishing.
- Jackins, B. D., Blank, R. S., Shulman, K. W., & Onello, H. H. (2010). *Managing a special needs trust: A guide for trustees*. Brookline: Disabilities Books.

Tryptophan

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Synonyms

[L-Tryptophan](#)

Definition

One of the amino acids (essential building blocks of proteins). Tryptophan is found in many dietary products and is a dietary precursor of a number of compounds including serotonin, melatonin, niacin, and others. Tryptophan is available in health food stores as a dietary supplement and has been advocated as an aid for sleep, although research data are mixed on this point. Its main relevance to psychiatry in general (and to autism in particular) has to do with its relationship to serotonin (both in autism and in depression).

See Also

- [Neurochemistry](#)
- [Serotonin](#)

References and Reading

- Anderson, G. M., & Hoshino, Y. (2005). Neurochemical studies of autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 453–472). Hoboken: Wiley.

TS Bourneville's Disease

- [Tuberous Sclerosis Complex](#)

TSC Tuberous Sclerosis

- [Tuberous Sclerosis Complex](#)

TSFI

- [Test of Sensory Functioning in Infants](#)

TSI

- ▶ [DeGangi-Berk Test of Sensory Integration](#)

TTAP

- ▶ [TEACCH Transition Assessment Profile \(TTAP\)](#)

TTAP-CV

- ▶ [TEACCH Transition Assessment Profile \(TTAP\)](#)

TTFC-2

- ▶ [Token Test for Children – Second Edition \(TTFC-2\)](#)

Tuberous Sclerosis Alliance

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Major Areas or Mission Statement

In 1974, four mothers of children diagnosed with Tuberous Sclerosis Complex (TSC) formed the Tuberous Sclerosis Alliance (TS Alliance) to provide fellowship, generate awareness, pursue knowledge, and provide hope to those who shared the common bond of TSC. Today, the TS Alliance has grown to almost 1,000 active members with

over 10,000 on its mailing list. It is the only national voluntary health organization dedicated to finding a cure for TSC while improving the lives of those affected through the following activities:

- The stimulation and sponsorship of research into the diagnosis, cause, management, and cure of TSC, a genetic disorder that can affect multiple organ systems
- The development of programs providing individuals with TSC and their families access to support services, resource information, and health care professionals engaged in the diagnosis, treatment, and management of TSC
- The development and implementation of public and professional programs designed to heighten awareness of TSC, thereby prompting early, accurate diagnosis and effective treatment

Landmark Contributions

In the quest to find a cure for TSC, the TS Alliance has contributed more than \$15 million to fund significant scientific breakthroughs. TS Alliance-sponsored research helped to:

- Identify two genes that cause TSC
1993: TSC2 gene on chromosome 16 is identified by a European consortium.
1997: TSC1 gene on chromosome 9 is identified by an International Consortium.
- Develop a genetic test
2002: Dr. Vicky Whittemore at TS Alliance facilitated the transfer of clinical genetic testing from two academic centers (Dr. Katherine Sims at Massachusetts General Hospital and Dr. Hope Northrup at Baylor University in Houston) to Athena Diagnostics, a fee-for-service commercial laboratory. Dr. Northrup helped Athena develop the molecular diagnostic tests for TSC1 and TSC2.
- Initiate the first clinical trial with sirolimus (Rapamune[®]), an FDA-approved drug that

showed promise of meaningful treatment. The trial was expanded into a Phase II, multicenter study co-funded by the National Cancer Institute at the National Institutes of Health. A Phase II Multi-Center Study of Rapamycin for Treating Kidney Angiomyolipomas in TSC or LAM (*Lymphangioliomyomatosis*).

Major Activities

TS Alliance Grants Program: Active since 1984, the TS Alliance has raised funds and administered a grants program to support basic, translational, and clinical research. The TS Alliance encourages the researchers to share information and resources, thus expediting research results and their translation into improved clinical care of individuals with TSC. Grants include postdoctoral fellowship awards as well as principal investigator support for scientists studying TSC and autism. For more information about the TS Alliance Grants Program go to <http://www.tsalliance.org/pages.aspx?content=24>.

In 2006 launched the TSC Natural History Database, a web-based, password-protected central research repository of information about individuals with TSC that includes demographics, genotypes, conditions affecting various organs in the human body, diagnostic and follow-up tests, and treatments. This project is intended to gather information over a participant's lifetime to ultimately improve the care of those affected by TSC, by:

1. Identifying cohorts of adults and children with TSC for future clinical studies and/or clinical trials for treatment of epilepsy, autism, tumor growth, and other manifestations of TSC
2. Improving the understanding of the progression of TSC over an individual's life span
3. Serving as a resource that will hasten the discovery of new treatments and therapies for individuals with TSC and for other causes of epilepsy, autism, cancer, and heart and lung disease

In 2010 launched the TSC Drug Screening Program to support pre-clinical development and testing of new therapies related to neurological and neurocognitive symptoms associated with TSC. TS Alliance is funding research for the steps between compound identification and testing for efficacy in human clinical trials in order to accelerate the development of a potential therapeutic agent for individuals with neurocognitive symptoms associated with TSC.

See Also

► [Tuberous Sclerosis Complex](#)

References and Reading

- Ann, H. (2011). *Guidelines for the assessment of cognitive and behavioral issues in TSC*. Tuberous Sclerosis Association. Retrieved March 2011, from <http://www.tsalliance.org/pages.aspx?content=589>
- Dabora, S. L., Jozwiak, S., Franz, D. N., Roberts, P. S., Nieto, A., Chung, J., et al. (2001). Mutational analysis in a cohort of 224 tuberous sclerosis patients indicates increased severity of TSC2, compared with TSC1, disease in multiple organs. *American Journal of Human Genetics*, 68, 64–80.
- Ehninger, D., Sano, Y., de Vries, P. J., Dies, K., Franz, D., Geschwind, D. H., et al. (2010). Gestational immune activation and Tsc2 haploinsufficiency cooperate to disrupt fetal survival and may perturb social behavior in adult mice. *Molecular Psychiatry*. <https://doi.org/10.1038/mp.2010.115>.
- European Chromosome 16 Tuberous Sclerosis Consortium. (1993). Identification and characterization of the tuberous sclerosis gene on chromosome 16. *Cell*, 31, 1305–1315.
- Kwiatkowski, D. J., Whittemore, V. H., & Thiele, E. A. (2010). *Tuberous sclerosis complex: Genes, clinical features, and therapeutics*. Weinheim: Wiley-Blackwell.
- Multimedia resource from the Herscot Center for TSC at Massachusetts General Hospital. (2006). http://www2.massgeneral.org/livingwithtsc/affects/mental_health.htm
- Van Slegtenhorst, M., de Hoogt, R., Hermans, C., Nellist, M., Janssen, B., Verhoef, S., et al. (1997). Identification of the tuberous sclerosis gene TSC1 on chromosome 9q34. *Science*, 277, 805–808.

Tuberous Sclerosis Complex

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Synonyms

TS Bourneville's disease; TSC tuberous sclerosis

Short Description or Definition

Tuberous Sclerosis Complex (TSC) is a rare neurogenetic disorder characterized by abnormalities in multiple systems including brain, skin, eyes, heart, lungs, and kidneys (Crino et al. 2006). It is one of the single gene disorders that has been most closely associated with autism spectrum disorders (ASD) and many researchers believe that it can be a good model for understanding the pathophysiology of ASD (Tsai and Sahin 2011).

TSC is caused by mutations in one of two genes (*TSC1* or *TSC2*) which result in the development of benign tumors (hamartomas) in many organs. These gene mutations can either be inherited in an autosomal dominant fashion or occur de novo. They are known to disrupt a key signaling pathway involved in cell size regulation and therefore allow for abnormal cell growth in a number of different systems. Most relevant to ASD are lesions in the brain. The most common lesions are cortical tubers, nests of giant cells, which can cause seizures and are posited to be associated with the cognitive, learning, and behavioral problems (including ASD) that are commonly seen in this disorder. Subependymal giant cell astrocytomas (SEGAs) are so-called benign tumors in the brain that can lead to significant morbidity. Other organ system involvement includes a variety of lesions on the skin as well as fingernails and teeth along with hamartomatous growths in the retina, heart, kidneys, and lungs. Clinical expression and outcome is highly

variable and largely dependent on the specific manifestations of the disorder in a given patient.

Categorization

TSC is categorized as a rare neurogenetic disorder because it is known to be caused by specific gene mutations. It is also considered a neurocutaneous disorder because individuals with TSC have findings both on the skin and in the brain.

Diagnosis is made according to the presence of clinical features (major and minor) (Roach et al. 1998) and can be confirmed by gene mutation analysis.

Major features include findings in the following systems:

- Brain (cortical tuber, subependymal nodule, subependymal giant cell astrocytoma)
- Skin or nails (facial angiofibromas, three or more hypopigmented macules or ash leaf spots, presence of a shagreen patch or a connective tissue nevus, nontraumatic ungula or periungual fibroma)
- Eye (Multiple retinal nodular hamartomas)
- Heart (Cardiac rhabdomyoma)
- Lungs (Lymphangioleiomyomatosis/LAM)
- Kidneys (Renal angiomyolipoma)

Minor features include findings in multiple systems: pits in dental enamel, hamartomatous rectal polyps, bone cysts, cerebral white-matter "migration tracts," gingival fibromas, nonrenal hamartoma, retinal achromic patch, "confetti" skin lesions, multiple renal cysts.

Epidemiology

TSC is considered a rare disorder with prevalence estimated at 1 in 6000. There are up to 40,000 individuals with TSC in the US and one to two million worldwide. There are no known risk factors according to gender, ethnic, or racial groups (NINDS fact sheet).

ASD occurs in up to 40% of individuals with TSC. However, TSC is a relatively rare cause of

ASD with most studies reporting only 1–3% of large autism samples having TSC. Despite much research, the exact factors associated with increased risk of developing ASD in individuals with TSC are still not known.

Natural History, Prognostic Factors, Outcomes

Some cases of TSC are diagnosed very early in life (Datta et al. 2008). The presence of a cardiac rhabdomyoma may even be picked up on fetal ultrasound allowing for diagnosis in the newborn period. Early onset seizures called infantile spasms (IS) are often associated with TSC and careful physical examination and brain imaging can lead to a diagnosis in the first year of life. However, in milder cases, the disorder is often not diagnosed until the skin manifestations arise in later childhood and/or behavioral and learning deficits become more obvious.

This potential for early diagnosis may be especially important for the development of ASD. There are currently intensive research efforts attempting to discover the specific risk factors for ASD and other neurobehavioral outcomes with the idea that we might be able to develop preventative strategies.

TSC is a lifelong disorder and clinical manifestations can change over time. It is crucial that patients have long-term follow-up with providers familiar with the myriad of symptoms associated with the disorder.

Prognosis is extremely variable and dependent on the manifestation of the disorder in the individual patient. Even within the same family (with the same genetic mutation), there are widely variable presentations. For instance, some individuals with TSC only have isolated skin lesions. Alternatively, in other patients, severe renal disease can result in kidney failure, while others may be affected with severe epilepsy, intellectual disability and autism.

Why some individuals with TSC develop ASD is still unknown, but various researchers have suggested that risk factors include worsened seizures, intellectual disability, more tuber burden

(either numbers or volume), or even specific location of tubers in the temporal lobe and/or cerebellum. And more recent data suggests that the neurocognitive phenotype in TSC may be related to subtle abnormalities in the white matter. These findings are unrelated to tuber burden and more suggestive of the idea of aberrant connectivity in the brain, a leading theory for the pathogenesis of ASD (Tsai and Sahin 2011).

Clinical Expression and Pathophysiology

As mentioned, the clinical expression of this disorder is extremely heterogeneous and depends on the burden of lesions in different organ systems (Tuberous Sclerosis Alliance website).

CNS symptoms include seizures often manifesting early in life as infantile spasms. Early and aggressive management of this epilepsy syndrome is important because uncontrolled spasms have been associated with poor neurodevelopmental outcomes. Later seizures can manifest as complex partial epilepsy of differing severity. Some individuals suffer from Lennox–Gastaut Syndrome, a severe epileptic condition associated with multiple seizure types, intellectual disability, and a characteristic EEG pattern of slow spike and wave SEGAs tend to occur during childhood (peak incidence ~9 years of age) and are often associated with signs of increased intracranial pressure (headaches, vomiting). The neurobehavioral expression of TSC ranges from mild learning difficulties or ADHD to severe intellectual disability and autism.

Typical skin findings including hypopigmented macules, facial angiofibromas, and periungual or subungual fibromas are not usually associated with significant morbidity. Whereas nail findings are rare to see in children, facial angiofibromas are not usually present at birth but can appear in early childhood.

Most cardiac rhabdomyomas are completely asymptomatic. However, large tumors in infants can be the cause of congestive heart failure and arrhythmias. These tend to regress in size over time and should be followed regularly with cardiac echo and EKG until they have disappeared or

stabilized in size. As mentioned, with continued improvements in fetal ultrasound, these tumors are now being diagnosed prenatally which allows for much earlier diagnosis of TSC.

The two most common kidney lesions are benign renal angiomyolipomas and cysts, both of which can be asymptomatic or cause destruction of kidney tissue over time. The major morbidity is secondary to bleeding. Other lesions such as malignant angiomyolipoma, oncocytomas, and renal cell carcinoma are rare but can occur. Renal imaging at routine intervals is essential.

Lung involvement most commonly involves lymphangioleiomyomatosis (LAM) which occurs exclusively in women and usually appears after age 20. Significant pathology is associated with breathing difficulties including shortness of breath or coughing or even spontaneous pneumothorax (collapsed lung).

Eye involvement involves hamartomas which appear as flat opaque lesions or as multinodular “mulberry” lesions in the retina. These lesions are usually asymptomatic but serious vision problems from bleeding or retinal detachment have been reported.

Because of intense research efforts, much is now known about the pathophysiology of this disorder. The genes responsible for the disorder (*TSC1* and *TSC2*) code for the proteins hamartin and tuberlin. These two proteins together are involved in regulating the mTOR complex which is part of a key signaling pathway controlling protein synthesis and cell size. Mutations in one or the other gene with subsequent reduction of protein will result in overactivity of the mTOR complex and uncontrolled protein synthesis and cell growth (Tsai and Sahin 2011).

Evaluation and Differential Diagnosis

Evaluation involves a thorough physical examination looking for skin, nail, and tooth findings. An ultraviolet light (called a Wood’s lamp) is often needed to identify the hypopigmented macules, especially in individuals with light skin. Imaging of the brain with MRI is necessary to identify the cortical tubers as well as the presence

of subependymal nodules or SEGA. Because of the frequent comorbid neurobehavioral problems, it has been recommended that all children with TSC undergo thorough behavioral and cognitive assessments (de Vries et al. 2005).

For non-CNS disease, the evaluation involves a number of different specialists. Ophthalmological exam can reveal the retinal changes. Renal ultrasound is recommended at time of diagnosis and at regular follow-up intervals to identify and follow angiomyolipomas. Similarly, echocardiogram and EKG are essential to identify the cardiac rhabdomyomas and to screen for any cardiac arrhythmias. Recent research has shown that many women with TSC have minor, usually asymptomatic lung involvement which can be identified through chest CT. It is currently recommended that women be screened initially in adolescence or at the time of diagnosis for older women.

Differential diagnosis really depends on the signs and symptoms exhibited by the individual. Unfortunately, most manifestations of TSC are not specific when they occur in isolation and thus it is the constellation of symptoms that will steer the clinician toward this diagnosis. Neurobehavioral problems (ASD, intellectual disability, and learning deficits) can have a number of different etiologies, so practitioners need to be aware of the possibility of TSC and look for other signs. Likewise, infantile spasms can be associated with other neurogenetic conditions, structural brain lesions such as focal cortical dysplasias, metabolic disorders, and can even be idiopathic (West Syndrome). However, when seen in a baby with hypopigmented macules and/or tubers on MRI the diagnosis is evident. There are a few signs that are more pathognomonic of the disorder such as the cardiac rhabdomyomas and facial angiofibromas. But the eye, renal, and lung involvement can be sporadic or associated with other diseases.

Treatment

The majority of treatments are aimed at specific symptoms associated with the disorder, but as we

understand more about the pathophysiology of TSC, there is intensifying interest in discovering treatments that specifically target the molecular pathways underlying the condition.

Currently CNS manifestations are treated with a wide variety of approaches (Tuberous Sclerosis Alliance website). Seizures are typically treated with anticonvulsant medications. Infantile spasms associated with TSC are particularly sensitive to vigabatrin. A number of other anticonvulsant medications are used for seizures in children and adults. Vagal nerve stimulation, special diets, and even focal resection epilepsy surgery have also been shown to be effective. The neurobehavioral manifestations including autism are treated with typical behavioral interventions. It is important to note that there are no current data suggesting that these treatments are any more or less effective for the ASD, intellectual, and learning deficits in patient with TSC than in other patients.

Facial angiofibromas can be treated with laser therapy. Renal angiomyolipomas can be surgically resected or treated with embolization procedures that are tissue sparing. Heart lesions and eye lesions usually do not need treatment.

The most exciting prospect in the investigation of new treatment opportunities involves the study of specific molecular compounds designed to impact the signaling pathway known to underlie this disorder. Exciting animal model work has shown that rapamycin (an mTOR inhibitor) can actually prevent the development of epilepsy in mice (Meikle et al. 2007; Zeng et al. 2008). Similarly the neurobehavioral or learning deficits have been reversed in another mouse model (Ehninger et al. 2008). Everolimus has recently received FDA approval for treatment of the SEGAs that are not amenable to surgical resection as well as angiomyolipomas. There are currently clinical trials examining the neurobehavioral effects of this class of medication in humans.

See Also

- Autism
- Epilepsy

References and Reading

- Crino, P. B., Nathanson, K. L., et al. (2006). The tuberous sclerosis complex. *The New England Journal of Medicine*, 355(13), 1345–1356.
- Datta, A. N., Hahn, C. D., et al. (2008). Clinical presentation and diagnosis of tuberous sclerosis complex in infancy. *Journal of Child Neurology*, 23(3), 268–273.
- de Vries, P., Humphrey, A., McCartney, D., Prather, P., Bolton, P., Hunt, A., et al. (2005). Consensus clinical guidelines for the assessment of cognitive and behavioural problems in Tuberous Sclerosis. *European Child & Adolescent Psychiatry*, 14(4), 183–190.
- Ehninger, D., Han, S., et al. (2008). Reversal of learning deficits in a Tsc2+/- mouse model of tuberous sclerosis. *Nature Medicine*, 14(8), 843–848.
- Meikle, L., Talos, D. M., et al. (2007). A mouse model of tuberous sclerosis: Neuronal loss of TSC1 causes dysplastic and ectopic neurons, reduced myelination, seizure activity, and limited survival. *Journal of Neuroscience*, 27(21), 5546–5558.
- NINDS fact sheet. See http://www.ninds.nih.gov/disorders/tuberous_sclerosis/detail_tuberous_sclerosis.htm
- Roach, E. S., Gomez, M. R., et al. (1998). Tuberous sclerosis complex consensus conference: Revised clinical diagnostic criteria. *Journal of Child Neurology*, 13(12), 624–628.
- Tsai, P., & Sahin, M. (2011). Mechanisms of neurocognitive dysfunction and therapeutic considerations in tuberous sclerosis complex. *Current Opinion in Neurology*, 24(2), 106–113.
- Tuberous Sclerosis Alliance website. <http://www.tsalliance.org/>
- Zeng, L. H., Xu, L., et al. (2008). Rapamycin prevents epilepsy in a mouse model of tuberous sclerosis complex. *Annals of Neurology*, 63(4), 444–453.

Turkey and Autism

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Historical Background

In Turkey, the earlier work with autism has been limited to academic centers in three major cities of Istanbul, Ankara, and Izmir, where the major medical schools were. The first medical center

dedicated to autism was established in 1989 at the Ankara University also where the first department of child psychiatry had been founded by Dr Mualla Ozturk in 1973. Drs Atalay Yörükoğlu in Ankara and Rıdvan Cebiroğlu in Istanbul were also among the academic leaders who clinically utilized the concept of autism as described by Leo Kanner. The DSM criteria after the publication of the DSM III in 1980 were quickly accepted in Turkey and became part of the standard evaluations.

Turkey currently hosts more than 30 university departments of child psychiatry and two state training hospitals with Child Psychiatry Departments that provide diagnosis and some of the treatment services for autism.

The common misperception of the association between autism and cold, distant, over-intellectual mothering with high socioeconomic status was prevalent until the late 1990s, despite the medical model in the academic centers. However, in parallel to the proliferation of the national and worldwide research into the etiology and pathogenesis, autism is now almost unanimously considered as a neurobiologically based disorder of social emotional development evident in social, behavioral, and communication domains by the professionals from diverse backgrounds (from special education to genetics to child psychiatry to psychology). Currently, studies conducted with families and teachers of children with autism show that autism remains to be perceived as an “abnormality” and to be stigmatized.

In Turkey, legislative efforts to meet the educational needs of individuals with disabilities date back to 1952 (Birkan 2007). Until 1995, children with autism received education no different than designed for those with intellectual disabilities. In 1999, following the Ministry of Education’s project for “Autistic Children’s Education (OCEP)” and its implementation in 2000 for children between the ages of 3 and 15, local counseling centers and educational units for children with autism were established (http://mevzuat.meb.gov.tr/html/2567_0.html, 2004). The aim was defined as to set the standards of service to individuals with autism, in accordance with the 19 items of the Charter of Rights for Persons

with Autism presented at the 4th Autism-Europe Congress, Den Haag, May 10, 1992 (<http://www.autismeurope.org/files/files/ae-eypd-edu-final-2-eng.pdf>).

Since the beginning of 1990s, numerous foundations and societies have been formed related to autism, especially with the aims of organizing and providing special educational services that were needed. These include but are not limited to Ilgi, IZEV, TODEV, OYAD, ODER, and TOHU-M. The Autism Platform that brings together these entities has worked in the lobbying, advocacy, and public communication efforts in order to raise awareness and support infrastructural work (Darica et al. 1992).

Most of these foundations and societies have focused their work on providing counseling, guidance, referral, support, special education, education about autism, and related services to families with autism.

The shortage of adequately trained professionals to provide the special education services is a main challenge in meeting the children’s special educational needs. Currently, there are more than thirty universities with departments of Special Education, although, a small number of them have a focus on autism.

Legal Issues, Mandates for Service

The new legislative studies on disabilities began in the second half of 1990s, after a long interval since the initial legislative efforts in 1950s. In 1997, an official position in the government was declared for the administration of the services for the disabled. In 2005, the first law, briefly referred to as “The Law of the Disabled” (Law Nr: 5378), was passed (Birkan 2007; Özgökçeler and Alper 2010). According to this law, individuals with disabilities have access not only to the services to meet their constitutional and international rights but also access to certain services and accommodations commensurate with their disabilities. According to this law, the state is mandated to provide reimbursement to the family of the child with autism for child care as well as support for the child’s special educational needs. The state is also obliged to

create job opportunities for all individuals with disabilities. Early diagnosis and prevention services were also mandated by this law. Although recent major amendments were made in 2012, the law continues to lack regulations regarding vocational training opportunities and employment opportunities both in the private sector as well as the public sector (<http://www.mevzuat.gov.tr/MevzuatMetin/1.5.5378.pdf>).

Overview of Current Treatments and Centers

After the first center for the education of children with autism (OCEM) was established in 1999, there has been a rapid increase in the number of OCEMs throughout the country. An OCEM can either be independent or within the body of a mainstream public school as a special class. Depending on their developmental level and social adjustment, children with autism can also be inclusion students within mainstream classrooms (Aksüt et al. 2000).

Children with autism are also entitled to receive special education and rehabilitation services provided or reimbursed by the state, although the number of hours designated (8 h per month, 2013) for such services is far from sufficient (Yıldırım 2007). The type of the services offered to each child is determined by the RAM, described in an Individualized Education Plan (BEP) (Alpaytaç 2009).

There are currently 49 OCEMs in 28 cities in Turkey. As of 2012, the number of children receiving educational services at these centers was 2,066. As of 2012, in grades 1 through 8, the number of children with autism who are inclusion students is 1,537, and the number of children who are enrolled in a “special class” is 2,022. The proportion of children with autism is quite small if we consider that at 1,605 “educational centers” across the country, at primary education level, 241,746 children with a variety of intellectual disabilities are served (Aksüt 2001).

Unfortunately, special educational services are limited beyond the 8th grade for children with autism.

The number of Vocational Training Centers for Autistic Children was 14 in 2012.

Besides these government-supported services, the families of children with autism and the foundations and societies working with autism assume an active role in introducing and educating the parents on different modes of education and treatment services and disseminating the knowledge to the professionals and the families (such as the web portal by TOHUM foundation prepared based on PCDI method at the address <http://www.tohumotizmportali.org/>).

Overview of Research Directions

The epidemiological facts of autism in Turkey are roughly estimated by the estimates of the WHO, that is, one in 250. An epidemiological research for identifying the prevalence of autism in Turkey is urgently warranted for better planning of the services and allocation of the resources.

Based on the 1/250 ratio, we can assume that there should be 271,000 individuals with autism in Turkey, 81,000 of whom between the ages of 0 and 14. Considering that autism does not only affect the individual but also their families, their doctors and caregivers, as well as their educators, it would be a close approximation to assume that autism is a part of 1,626,000 individuals’ lives; 567,000 in the 0–14 age group.

There are a number of publications from the ongoing research in pharmacology, genetics, and neuroimaging of autism in Turkey. Many studies on the phenomenology and clinical observations of autism have also been published. However, there is a continued need for epidemiological studies for an accurate estimate of the prevalence of autism in Turkey. As is the case for the rest of the world, early identification and intervention studies remain as major needs.

Social Policy and Training

A collaborative research by the State Planning Organization (DPT) and Turkish Scientific Research Council (TUBITAK) in 2002 revealed

that individuals with disabilities are facing great social disadvantages and experiencing the practical, everyday aspects of this social inequality.

Awareness studies and campaigns have been instrumental in making autism a part of the daily language as well as in increasing the parental sensitivity to social emotional development.

A recent event during the writing of this item was a general meeting organized by the Ministry of Family and Social Policies in April 2, 2013. In this meeting, the ministry, alongside the foundations and organizations working in the field of autism, generated an action plan which included points regarding increasing the involvement of individuals with autism in the general social environments, increasing education services and opportunities beyond K-8, increasing media coverage on autism to raise awareness, and increasing early identification and intervention resources.

See Also

► [Culture and Autism](#)

References and Reading

- Aksüt, M. (2001). "Yeni Bin Yılın Yeni Eğitim Merkezleri (OÇEM'ler) ve Otistik Bireylerin Eğitimi AKÜ Sosyal Bilimler Dergisi Cilt III. Sayı 2, Sayfa 56–71 Aralık 2001 Afyon.
- Aksüt, M. et al. (2000). Otistik Çocuklar Eğitim Programı, MEB. Ankara.
- Alpaytaç, S. (2009). Otizm Üzerine Türkiye'den Bir Örnek Vaka İncelemesi.
- Birkan, B. (2007). "Dünden Bugüne Otizm" Özürlüler 2007 Kongre Bildirileri Kitabı. http://www.ozurlulerkongresi.org/2013/assets/Uploads/gecmis-kongreler/2007_kongre-bildiri_kitabi.pdf
- Darıca, N., Abidoğlu, Ü., & Gümüşçü Ş. (1992). Otizm ve Otistik Çocuklar. Özgür Yayınları.
- http://mevzuat.meb.gov.tr/html/2567_0.html
- <http://www.autismeurope.org/files/files/ae-cypd-edu-final-2-eng.pdf>
- <http://www.mevzuat.gov.tr/MevzuatMetin/1.5.5378.pdf>
- Milli Eğitim Bakanlığı Otistik Çocuklar Eğitim Merkezleri Yönergesi. Tebliğler Dergisi: ARALIK 2004/2567.
- Özgökçeler, S., & Alper, Y. (2010). Özürlüler Kanunu'nun Sosyal Model Açısından Değerlendirilmesi. *İşletme ve Ekonomi Araştırmaları Dergisi, C 1*(2010):1.
- Yıldırım, T. (2007). Özürlü Hakları Kapsamında İdarenin Yükümlülükleri. Özürlüler 2007 Kongre Bildirileri Kitabı.

Turner Syndrome

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Synonyms

[Ullrich-Turner syndrome](#)

Definition

A genetic condition in girls in which only one of two X chromosomes is present. In this condition, the individual is phenotypically female and is at risk for a range of difficulties. It is named after Henry Turner who described cases in the 1930s. Various forms of Turner's syndrome have been identified. These can include a complete or a partial absence of one X chromosome. In some cases, only some cells are missing the chromosome (mosaic Turner's syndrome). Diagnosis is sometimes made during the pregnancy (e.g., by amniocentesis or sometimes by ultrasound). A karyotype analysis confirms the diagnosis. Various forms of Turner's syndrome have been identified. These can include a complete or a partial absence of the X chromosome.

Physical abnormalities include low-set ears, webbed neck, short stature, and other features. Medical problems include amenorrhea and sterility and heart as well as visual and hearing problems. A substantial minority of cases exhibit cardiac and cardiovascular abnormalities including coarctation of the aorta and a defective aortic valve. Hormonal factors can inhibit skeletal growth. It is common for the fourth finger/toe to be short. Osteoporosis is frequent due to lack of estrogen and can lead to scoliosis. Abnormalities in other areas include problems with the kidneys and thyroid (typically taking the form of hypothyroidism), and there is an increased risk for diabetes. Those with the condition are typically

infertile, but with modern technology, they may be able to have a child following IVF.

Although sometimes associated with intellectual deficiency, Turner's syndrome is more frequently associated with a range of cognitive problems often consistent with the profile of non-verbal learning disability. These problems can be associated with other difficulties, including in adaptive skills and motor areas. Math skills are frequently impacted. There have been some reports of Turner's syndrome co-occurring with autism.

Individuals with the condition can lead active and productive lives. Medical and learning difficulties can present obstacles and should be a focus of intervention.

See Also

- [Nonverbal Learning Disabilities \(NLD\)](#)

References and Reading

- Donnelly, S. L., Wolpert, C. M., Menold, M. M., Bass, M. P., Gilbert, J. R., Cuccaro, M. L., Delong, G. R., & Pericak-Vance, M. A. (2000). Female with autistic disorder and monosomy X (Turner syndrome): Parent-of-origin effect of the X chromosome. *American Journal of Medical Genetics*, 96(3), 312–316.
- Ross, J., Zinn, A., & McCauley, E. (2000). Neurodevelopmental and psychosocial aspects of Turner syndrome. *Mental Retardation & Developmental Disabilities Research Reviews*, 6(2), 135–141.
- Rourke, B. (Ed.). (1994). *Syndrome of nonverbal learning disabilities: Manifestations in neurological disease disorder and dysfunction*. New York: Guilford Press.
- Skuse, D. H., James, R. S., Bishop, D. V., Coppin, B., Dalton, P., Aamodt-Leeper, G., et al. (1997). Evidence from Turner's syndrome of an imprinted X-linked locus affecting cognitive function. *Nature*, 387(6634), 705–708.
- Skuse, D. H., Mandy, W. P. L., & Scourfield, J. (2005). Measuring autistic traits: Heritability, reliability and validity of the social and communication disorders checklist. *British Journal of Psychiatry*, 187, 568–572.
- Yorifuji, T., Muroi, J., Kawai, M., Uematsu, A., Sasaki, H., Momoi, T., et al. (1998). Uniparental and functional X disomy in Turner syndrome patients with unexplained mental retardation and X derived marker chromosomes. *Journal of Medical Genetics*, 35(7), 539–544.

Turn-Taking

- [Discourse Management](#)

Tutor

- [Para-educator](#)

Twilite® [OTC]

- [Diphenhydramine](#)

Twin Studies in Autism

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Definition

Before researchers undertake studies aimed at identifying the genetic factors that contribute to a disorder like autism, whose specific causes are largely unknown, it is useful to attempt to determine the balance between genetic and environmental contributors. The reason is that if it were determined that a disorder were caused mainly by environmental factors, then the yield from genetic research might be quite low. Twin studies are one means of estimating the relative contributions of genes and environment to a phenotype.

The study of the rates of disease in twins can help elucidate the extent to which environmental versus genetic factors contribute to a particular disease or syndrome. When twins share the same condition, they are said to be “concordant” for the illness. When they do not share the disorder, they are said to be “discordant” for the disease. By comparing how frequently monozygotic twins are concordant for a disease (the “monozygotic

concordance rate”) with how frequently dizygotic twins are concordant for the same disease (the “dizygotic concordance rate”), one can estimate the degree to which genetic versus environmental factors contribute to disease.

If monozygotic twins are highly concordant for a disease and dizygotic twins significantly less concordant for a disease, this suggests that genetics plays a larger role than environmental factors in causing the illness. The rationale for this is that for each pair of twins, regardless of whether they are monozygotic or dizygotic, environmental factors are held essentially constant – the twin pairs result from the same pregnancy, are most often raised in the same household, and so are thought to be exposed to largely the same environmental factors. Thus, the main variable that could alter the concordance rate is the genetic composition of each twin pair. Since monozygotic twins share identical genetic material and dizygotic twins do not, a high concordance rate for monozygotic twins and a relatively lower one for dizygotic twins suggests each pair of monozygotic twins shares some identical genetic abnormalities that are contributing to their shared disease, while each pair of dizygotic twins, who do not by definition share identical genetic material, do not share identical genetic abnormalities and thus, do not share the same disease. If on the other hand, monozygotic twins have a rate of concordance for a disease that is similar to the rate for dizygotic twins, then environmental factors are likely to be contributing more than genetic factors to the disorder.

Twin studies in autism have shown that monozygotic twins are concordant for autism significantly more frequently than dizygotic twins. One of the most important initial studies conducted by Bailey et al. in England, found that 60% of monozygotic twins studied were concordant for autism compared to none of the dizygotic twins. When the definition of illness was broadened to include Autism Spectrum Disorders (ASDs) and not strictly autism, the concordance rate for monozygotic twins was greater than 90% and the rate for dizygotic twins was only 10%, giving a heritability estimate of approximately 90%. As discussed above, the high monozygotic concordance and the relatively low dizygotic concordance indicates that genetic factors contribute significantly to ASDs.

These findings have generally been corroborated by more recent studies, such as those conducted by Rosenberg et al. and Taniai et al. In these more recent studies, the dizygotic concordance was higher than those reported by Bailey, suggesting that environmental factors play a more significant role than initially calculated. Similarly, one of the most recent twin studies conducted by Hallmayer et al. reported a monozygotic concordance rate of 77%, and a dizygotic concordance rate of 31%. This most recent report provided the lowest estimate to date of the contribution of genes to ASD (~45%). Not surprisingly, the study quickly generated both proponents and detractors. However, when viewed in the broad context, this study, as have all twin studies of ASD to date, supported considerable genetic as well as environmental contributors to ASD.

See Also

- [Genetics](#)
- [Monozygotic \(MZ\) Twins](#)

References and Reading

- Bailey, A., Le Couteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda, E., et al. (1995). Autism as a strongly genetic disorder: Evidence from a British twin study. *Psychological Medicine*, 25(1), 63.
- Fraga, M. F., Ballestar, E., Paz, M. F., Ropero, S., Setien, F., & Ballestar, M. L. (2005). Epigenetic differences arise during the lifetime of monozygotic twins. *Proceedings of the National Academy of Sciences of the United States of America*, 102(30), 10604.
- Hallmayer, J., Cleveland, S., Torres, A., Phillips, J., Cohen, B., Torigoe, T., et al. (2011). Genetic heritability and shared environmental factors among twin pairs with autism. *Archives of General Psychiatry*, 68(11), 1095–1102.
- Haque, F. N., Gottesman, I. I., & Wong, A. H. (2009). Not really identical: Epigenetic differences in monozygotic twins and implications for twin studies in psychiatry. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics*, 151C(2), 136–141.
- Machin, G. A. (1996). Some causes of genotypic and phenotypic discordance in monozygotic twin pairs. *American Journal of Medical Genetics*, 61(3), 216–228.
- Reeve, E. C. R., & Black, I. (2001). *Encyclopedia of genetics*. London: Routledge.
- Ronald, A., & Hoekstra, R. A. (2011). Autism spectrum disorders and autistic traits: A decade of new twin

- studies. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 156(3), 255–274.
- Rosenberg, R. E., Law, J. K., Yenokyan, G., McGready, J., Kaufmann, W. E., & Law, P. A. (2009). Characteristics and concordance of autism spectrum disorders among 277 twin pairs. *Archives of Pediatrics and Adolescent Medicine*, 163(10), 907.
- Strachan, T., & Read, A. P. (2004). *Human molecular genetics*. New York: Garland Press.
- Taniai, H., Nishiyama, T., Miyachi, T., Imaeda, M., & Sumi, S. (2008). Genetic influences on the broad spectrum of autism: Study of proband-ascertained twins. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 147B(6), 844–849.

Type I Error

- [False Positive](#)

Type II Punishment

- [Omission Training](#)

Type II Punishment, Second Edition

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Synonyms

[Negative punishment](#); [Response cost](#); [Time-out](#)

Definition

Type 2 punishment refers to the removal of reinforcing or preferred stimulus following a behavior. This consequence for a behavior results in a decrease in the future frequency of the

behavior. Across the continuum of least restrictive procedures, Type 2 punishment is considered less aversive than Type 1, or positive punishment procedures. Type 2 punishment, like any punishment procedure, should be employed only after reinforcement-based procedures have failed to be effective. Common types of Type 2 punishment include time-out and response cost procedures.

See Also

- [Behavior Plan](#)
- [Challenging Behavior](#)
- [Punishment](#)

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson.
- Donaldson, J. M., & Vollmer, T. R. (2011). An evaluation and comparison of time-out procedures with and without release contingencies. *Journal of Applied Behavior Analysis*, 44(4), 693–705.
- Laprime, A. P., & Dietrich, G. (2013). An evaluation of a treatment package consisting of discrimination training and differential reinforcement with response cost and a social story on vocal stereotypy for a preschooler with autism in a preschool classroom. *Education and Treatment of Children*, 37(3), 407–430.

Typical

- [Neurotypical](#)

Typical Length

- [Mean Length of Utterance \(MLU\)](#)

Typically Developing

- [Neurotypical](#)

U

U.S. Family and Medical Leave Act

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Definition

Family and Medical Leave Act.

The Family and Medical Leave Act (FMLA) of 1993 is a Federal law which requires employers to provide unpaid leave for employees who have a serious medical condition requiring time off their job or time off to care for a family member. The law was signed into law by President Clinton and came into effect on August 5, 1993.

The Act provides for 12 weeks of unpaid, job-protected leave within a 12-month period for various reasons, including caring for a new child, caring for a seriously ill family member, recovering from the employee's own serious illness, caring for an injured family member who has been in military service, and addressing exigencies which stem from the active deployment of a family member.

The National Defense Authorization Act for Fiscal Year 2010 amends military family leave entitlements under the Family and Medical Leave Act (FMLA) to expand coverage for military caregiver leave. Additionally, the Airline Flight Crew Technical Corrections Act, Public Law 111-119, also modified eligibility requirements for flight crew members.

References and Reading

- Gerstel, N., & McGonagle, K. (1999). Job leaves and the limits of the Family and Medical Leave Act: The effects of gender, race, and family. *Work and Occupations*, 26(4), 510–534.
- Ruhm, C. J. (1997). Policy watch: The Family and Medical Leave Act. *The Journal of Economic Perspectives*, 11(3), 175–186.
- United States Department of Labor. (2011, 1 April). Family and Medical Leave Act. Retrieved 22 March 2011 from <http://www.dol.gov/whd/fmla/>
- Waldfogel, J. (2001). Family and medical leave: Evidence from the 2000 surveys. *Monthly Labor Review*, 124(9), 17–23.
- Young, A. K. (1998). Assessing the Family and Medical Leave Act in terms of gender equality, work/family balance, and the needs of children. *Michigan Journal of Gender and Law*, 5(1), 114–155.

UCLA Young Autism Project

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Definition

The UCLA Young Autism Project (YAP) is an applied behavior analytic intervention model for preschool children diagnosed with autism spectrum disorders. Based on the work of O. Ivar Lovaas, the treatment consists of 40 h per week of intensive one-to-one therapy over a period of 2–3 years, usually beginning when children are under the age of 3½ years.

Historical Background

The UCLA Young Autism Project has its origins in research conducted in the 1960s on the use of operant conditioning in the treatment of children with autism (DeMye et al. 1981). These early studies were quite successful in demonstrating immediate gains in adaptive skills such as language and reductions in problem behavior such as aggression and self-injury, but long-term outcomes were poor, with most children reverting to pretreatment levels of functioning.

The UCLA YAP was initiated by Lovaas in 1970 in an effort to improve outcomes. It differed from previous ABA programs in its focus on young children, delivery of treatment in the home with parental participation, use of a comprehensive curriculum, and recruitment of undergraduates who worked in teams to provide 40 h per week of instruction. In 1987, Lovaas summarized the initial results of this project in a landmark study published in the *Journal of Consulting and Clinical Psychology*, which was later supplemented by a long-term follow-up study of

these same children by McEachin, Smith, and Lovaas (1993). The compelling results reported by Lovaas and colleagues were unprecedented in the early intervention literature and prompted both excitement and heated controversy within the field.

In order to validate his findings, Lovaas recognized the need for replication of the results across multiple studies. Replication projects of the UCLA YAP were instituted at other sites beginning in the 1990s, including Smith, Groen and Wynn (2000) study and the National Institute of Mental Health Multisite Young Autism Project (Cohen et al. 2006; Sallows and Graupner 2005), among other independent replications. Although these studies did not produce outcomes of the magnitude originally reported by Lovaas (1987), they did confirm his primary finding that the UCLA model of early intensive behavioral intervention (EIBI) can be effective in accelerating the development of some children with autism (Smith et al. 2008).

The YAP closed when Lovaas retired from UCLA in 2004, but the intervention model continues to be implemented through the Lovaas Institute for Early Intervention and replication sites.

Rationale or Underlying Theory

According to Smith and Lovaas (1998), the Young Autism Project was designed to address the large discrepancy in learning styles between children on the spectrum and their typically developing peers. While most children learn by exploring and interacting with their environments in novel and creative ways, the idiosyncratic behaviors and restricted interests of children with autism generally prevent them from engaging in this style of learning. Lovaas theorized that a highly intensive intervention was needed to account for these lost learning opportunities and would be most effective if implemented early in the child's life across school, home, and community settings (Smith et al. 2008). The intervention is also designed to maximize success and minimize failures for the child in order to reduce the frustration

and ensuing avoidance behaviors that often accompany typical teaching situations. Ultimately, the YAP aims to construct a functional educational environment that meets the unique needs of children with autism (Smith and Lovaas 1998).

Goals and Objectives

The ultimate goal of the YAP is to maximize behavioral treatment gains for children with autism during most of their waking hours and across all significant environments (Lovaas 1987). By creating educational environments that cater to the specific needs of children on the autism spectrum, the YAP aims to match the learning opportunities available to typically developing children and improve global functioning (Smith and Lovaas 1998).

Treatment Participants

Ideal treatment participants meet criteria for autism as defined in the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association [APA] 1994) and generally begin treatment before the age of 42 months. According to some reports, best outcomes are generally attained by participants who are able to acquire speech early in treatment, while those who have difficulty with this skill tend to demonstrate poorer outcomes.

Treatment Procedures

The treatment, as originally prescribed, calls for an average of 40 h per week of intensive one-to-one behavior therapy over a period of 2–3 years and is conducted in the subject's home, school, and community by a team of therapists and the subject's parents. The treatment is based in reinforcement (operant) theory and relies on shaping behaviors through reinforcement of successive approximations, the use of prompting and fading procedures, and the reduction or reshaping of

aggressive behaviors into more socially acceptable, functional forms of behavior (Smith and Lovaas 1998). Discrimination learning is used to help the child generalize learned skills across other environments.

In the first year of treatment, the primary instructional method is discrete trial teaching, focusing on self-help skills, elementary receptive language skills, simple verbal and nonverbal imitation, and establishing the beginnings of appropriate toy play. Once these skills have been mastered, the second year of treatment is geared toward teaching expressive language skills and interactive play with peers, as well as preparing subjects to enter preschool. Subjects are first taught how to engage in standard preschool activities in the home and are gradually introduced into the school setting over time. Once subjects master these milestones in the second year, the third year of treatment emphasizes general school-based tasks, such as basic reading and writing skills, interactive play with peers, and observational learning (or learning by watching others learn) (Smith and Lovaas 1998). The goal of this gradual introduction to the classroom environment is to ease the transition into kindergarten and ultimately ensure the child's success in a general educational setting.

Efficacy Information

The original UCLA study examined three separate groups: the experimental group and Control Groups 1 and 2. Control Group 1 received 10 h of behavior therapy per week, while those in Control Group 2 received special education services in local public schools without input from or contact with the UCLA project. At follow-up, subjects in the experimental group demonstrated significantly higher educational placements and IQ scores than subjects in either control group. Of the 19 experimental group subjects, 47% ($n = 9$) were eventually determined to be "normally functioning," compared to 2% ($n = 1$) of the control group subjects (Lovaas 1987). A further follow-up was conducted at age 12, and it was found that subjects in the experimental group maintained

these gains and also scored higher than control group subjects on measures of personality and adaptive behavior (McEachin et al. 1993).

Opportunities for replication have been somewhat restricted due to the intensity and duration of the intervention. Of those replication studies that have been completed, all have reported significant gains for children in the experimental condition, although these results have not been of the same magnitude as those of the original study (Howlin et al. 2009). In addition to the feasibility of replication, some aspects of the methodology used by Lovaas have sparked controversy among critics who question the validity of his results (Gresham and MacMillan 1998; Howlin et al. 2009). Many of the replication studies have attempted to address these methodological weaknesses. Despite these issues and the need for further replication, early intensive behavioral interventions based on the UCLA model are widely regarded as highly effective for the treatment of some young children autism (Howlin et al. 2009; Reichow and Wolery 2009).

Outcome Measurement

In the original study (1987), Lovaas incorporated two primary outcome measurements: the subject's posttreatment IQ score and first-grade educational placement, between the ages of 6 and 7 years. The choice of IQ measure was determined according to the subject's developmental level and most frequently included the Wechsler Intelligence Scale for Children-Revised, the Stanford-Binet Intelligence Scale, and the Peabody Picture Vocabulary Test. Information about first-grade placement was assessed by parent report and validated by the educational institution.

Qualifications of Treatment Providers

The clinicians administering the behavioral interventions in the original study (Lovaas 1987) were undergraduate students with superior performance in a course on behavioral treatment, as well as senior staff members with 2 years or

more of experience with the YAP (Smith and Lovaas 1998). All clinicians were trained and supervised by doctoral-level psychologists with many years of experience in treating children with autism.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Early Intensive Behavioral Intervention \(EIBI\)](#)
- [Lovaas Approach](#)
- [Lovaas, O. Ivar](#)

References and Reading

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Cohen, H., Amerine-Dickens, M., & Smith, T. (2006). Early intensive behavioral treatment: Replication of the UCLA model in a community setting. *Journal of Developmental and Behavioral Pediatrics*, 27(2), 145–155.
- DeMyer, M. K., Hingtgen, J. N., & Jackson, R. K. (1981). Infantile autism reviewed: A decade of research. *Schizophrenia Bulletin*, 7, 388–451.
- Gresham, F. M., & MacMillan, D. L. (1998). Early intervention project: Can its claims be substantiated and its effects replicated? *Journal of Autism and Developmental Disorders*, 28, 5–13.
- Gresham, F. M., Beebe-Frankenberger, M. E., & MacMillan, D. L. (1999). A selective review of treatments for children with autism: Description and methodological considerations. *School Psychology Review*, 28(4), 559–575.
- Howlin, P., Magiati, I., & Charman, T. (2009). Systematic review of early intensive behavioral interventions for children with autism. *American Journal on Intellectual and Developmental Disabilities*, 114(1), 23–41.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55(1), 3–9.
- Lovaas, O. I. (1993). The development of a treatment-research project for developmentally disabled and autistic children. *Journal of Applied Behavior Analysis*, 26(4), 617–630.
- McEachin, J. J., Smith, T., & Lovaas, O. I. (1993). Long-term outcome for children with autism who received early intensive behavioural treatment. *American Journal of Mental Retardation*, 97(4), 359–372.
- Reichow, B., & Wolery, M. (2009). Comprehensive synthesis of early intervention behavioral interventions for

young children with autism based on the UCLA Young Autism Project model. *Journal of Autism and Developmental Disorders*, 39, 23–41.

- Sallows, G., & Graupner, T. D. (2005). Intensive behavioral treatment for children with autism: Four-year outcome and predictors. *American Journal of Mental Retardation*, 110(6), 417–438.
- Smith, T., & Lovaas, O. I. (1998). Intensive and early behavioral intervention with autism: The UCLA Young Autism Project. *Infants & Young Children*, 10(3), 67–78.
- Smith, R., Groen, A. D., & Wynn, J. W. (2000). Randomized trial of intensive early intervention for children with pervasive developmental disorder. *American Journal on Mental Retardation*, 105, 269–285.
- Smith, T., Mruzek, D. W., & Peyton, R. (2008). A study in perseverance: The emergence of early intensive behavioral intervention. In E. Cipani (Ed.), *Triumphs in early autism treatment*. New York: Springer.

Ullrich-Turner Syndrome

► Turner Syndrome

Uncertainty Processing in Autism

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Definition

A summary of theory and research on the relevance of uncertainty processing to core symptoms of autism spectrum disorder.

Historical Background

Autism spectrum disorder (ASD) is often associated with a difficulty in processing and a lack of preference for uncertainty in the environment. Since the first description of ASD by Leo Kanner, ASD has been associated with a preference for sameness and predictability. Kanner described this cluster of symptoms as “an anxiously obsessive desire for the maintenance of sameness” and explained that “changes of routine, of furniture arrangement, of a pattern, of the order in which every day acts are carried out, can drive him [a child with ASD] to despair” (Kanner 1944, pp. 215). Other early conceptualizations have suggested that individuals with ASD tend to have extremely negative reactions to unexpected events (Baron-Cohen 1989; Dawson and Lewy 1989; Hutt et al. 1964).

Since the first inclusion of autism in the *Diagnostic and Statistical Manual of Mental Disorders* (a classification system for all psychiatric disorders), the diagnostic criteria for autism have always included atypical responses to uncertainty as a core symptom. The *Diagnostic and Statistical Manual of Mental Disorders*, third Edition (DSM-III), the first version to include autism, describes “Bizarre responses to various aspects of the environment, e.g., resistance to change” as a symptom of infantile autism (APA 1980). The Revised DSM-III (DSM-III-R) includes the following symptoms of autistic disorder: “Marked distress over changes in trivial aspects of environment (for example, when a vase is moved from usual position)” and “Unreasonable insistence on following routines in precise detail (for example, insisting that exactly the same route always be followed when shopping)” (APA 1987). The DSM-IV describes “Apparently inflexible adherence to specific, nonfunctional routines or rituals” as a possible symptom of autistic disorder (APA 1994). Most recently, the DSM-5 includes the following as a symptom of ASD: “inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behavior (e.g., extreme distress at small changes, difficulties with transitions, rigid thinking patterns, greeting rituals, need to take same route or eat the same food every day)” (APA 2013).

Current Knowledge

Relations Between Uncertainty Processing and Autism Symptoms and Features

Impairments in processing uncertainty, and impairments in the related construct of prediction, are increasingly being recognized as important features of ASD that are linked to core symptoms and co-occurring conditions associated with ASD. Previous research indicates that children with ASD prefer to engage in predictable or repetitive tasks (Boucher 1977) and demonstrate increased attention to and engagement with objects presented underpredictable versus unpredictable conditions (Ferrara and Hill 1980). Children with ASD also show an exaggerated response to changes in their environment (Kootz et al. 1982) and reduced exploration of novel or unfamiliar environments (Pierce and Courchesne 2001). Individuals with ASD also report that changes in their schedules or transitions are one of the primary sources of stress in their lives (Gillott and Standen 2007).

The two defining features of ASD, impaired processing of social and communicative information and restricted repetitive behaviors (APA 2013), both have relevance to uncertainty processing. First, a preference for sameness and aversion toward uncertainty would undoubtedly impact social functioning in individuals with ASD given the inherent unpredictable nature of social stimuli in the naturalistic environment. The complexity and dynamicity of human interactions make predicting social behavior extremely difficult, even for the most socially skilled individuals. Yet, for individuals with ASD, fundamental deficits in perspective-taking and social attention likely make social stimuli even more unpredictable. Thus, a preference for predictability in ASD, along with pervasive social deficits, may ultimately reduce motivation to attend to and interact with social stimuli in ASD.

In contrast to this pattern of reduced social motivation, individuals with ASD often have intense, specialized, and restricted interests referred to as *circumscribed interests* (APA 2013). These interests are more intense and less flexible than interests in typically developing (TD) children and frequently

interfere with the development of social relationships and participation in functional activities (Klin et al. 2007; Turner-Brown et al. 2011). Interestingly, the most common circumscribed interests in ASD are predictable objects, events, or functions (e.g., machines, mechanical systems, vehicles, computers) (Baron-Cohen and Wheelwright 1999; Turner-Brown et al. 2011). Likewise, for decades it has also been recognized that some individuals with ASD have enhanced skills in certain domains. These so-called islets of abilities (Shah and Frith 1983) typically are in topics related to mathematics, calendar calculations, and visual search tasks (Pring 2005). Typically, what these domains have in common is that they are rule-based and predictable, thereby minimizing ambiguity and uncertainty. More recent evidence also indicates that an intolerance of uncertainty is associated with sensory sensitivity (Neil et al. 2016) and repetitive behaviors in ASD (Vasa et al. 2018).

Therefore, both areas of preserved abilities and impairments in ASD share an affinity for certainty and an aversion of uncertainty. For this reason, understanding uncertainty processing in ASD is likely of prime importance to gain insight into cognitive systems with etiologic relevance to the disorder.

More broadly, two recent theoretical models have suggested that a core deficit in ASD may be related to how individuals with ASD process uncertain events and develop predictions based on these events. The first model, proposed by Sinha et al. (2014), suggests that individuals with ASD have an impaired ability to understand predictive relationships in the environment. According to this model, an inability to effectively develop and test predictive hypotheses results in individuals with ASD living in a constantly unpredictable and uncertain environment. Insistence on sameness and avoidance of social interaction thus minimizes the impact of this deficit. A second theoretical model, developed by Van de Cruys et al. (2014), builds upon the first model by suggesting that impaired predictive coding in ASD is the result of an inability to learn from relevant predictive cues in a dynamic and uncertain environment. According to this model, individuals with ASD are overly attuned to uncertainty in the environment as they

expect every situation to meet very precise predictions based on past events. Thus, they place too much weight on irrelevant violations of their expectations and predictive coding becomes shaped by environmental noise or random variability rather than relevant cues. The authors suggest that this effect is particularly robust in complex and dynamic situations such as social interactions. Accordingly, individuals with ASD may prefer to engage in repetitive and stereotyped behaviors with known contingencies, while avoiding the relative uncertainty of social interactions.

Intolerance of Uncertainty and Anxiety in ASD

Processing uncertainty in the environment is a critical component of learning, and the drive to resolve uncertainty has been proposed to be a principal motivating force for nearly all human behavior (Kagan 1972). The preference for predictability in ASD bears notable resemblance to a construct that has been primarily studied in the context of anxiety disorders referred to as “intolerance of uncertainty” (Rodgers et al. 2012). Individuals with intolerance of uncertainty find uncertain situations stressful and distressing, tend to interpret ambiguous information as threatening, and find it difficult to function in the face of uncertainty (Buhr and Dugas 2009). An intolerance of uncertainty is a characteristic of a range of anxiety disorders, including generalized anxiety disorder (Ladouceur et al. 2000b), obsessive-compulsive disorder (Tolin et al. 2003), and social anxiety disorder. Although the causal relationship between intolerance of uncertainty and anxiety warrants further research, it is clear that intolerance of uncertainty serves as a broad vulnerability factor for anxiety disorders (Carleton 2012). In addition, intolerance of uncertainty is a common intervention target in anxiety disorders, and treatment-related reductions in the intolerance of uncertainty are associated with reduced anxiety in individuals with anxiety disorders (Wilkinson et al. 2011).

As many as 50–75% of individuals with ASD meet criteria for an anxiety disorder (Mosner et al. 2019), and individuals with ASD often meet criteria for multiple anxiety disorders (Simonoff et al. 2008). A growing body of research is focused on understanding intolerance of uncertainty in

autism and has consistently found elevated intolerance of uncertainty in children and adults with autism (Rodgers et al. 2017). There is evidence for strong positive correlations between anxiety and IU in ASD (Boulter et al. 2013; Chamberlain et al. 2013; Wigham et al. 2014) and evidence that intolerance of uncertainty mediates the relationship between anxiety and ASD, suggesting that intolerance of uncertainty may play a causal role in the development of anxiety in ASD (Boulter et al. 2013). However, it remains unclear the extent to which the aversion to uncertainty in ASD is characterized by beliefs that uncertainty necessarily results in negative outcomes as is the case in anxiety disorders (Koerner and Dugas 2006, 2008). Kerns et al. (2014) suggested that distress associated with uncertainty may be an important factor in the development of anxiety in ASD, highlighting that intolerance of uncertainty is an important mechanism contributing to the emergence of anxiety in ASD and an appropriate target for ASD intervention. Boulter et al. (2014) found that intolerance of uncertainty and anxiety were correlated in ASD and that intolerance of uncertainty mediates the ASD-anxiety association (in other words, intolerance of uncertainty may be a causal factor of anxiety in ASD). According to this model, insistence on sameness in ASD may serve as a maladaptive coping strategy aimed at reducing uncertainty and thus anxiety. Additionally, in a group of children with ASD receiving treatment for anxiety, high levels of pretreatment intolerance of uncertainty predicted poorer treatment response (Keefer et al. 2017). Further, in a study designed to validate a new self- and parent-report measure of anxiety in children with ASD using factor analysis, Rodgers et al. (2016) identified four anxiety factors in ASD, one of which was an uncertainty intolerance subscale.

The “insistence on sameness” symptom observed in most individuals with ASD shares many qualities with intolerance of uncertainty. This symptom domain includes repetitive thoughts and actions, behavioral rigidity, a reliance on routines, resistance to change, and obsessive adherence to rituals. Ritualistic behaviors and insistence on sameness in ASD may reflect behaviors that function to minimize uncertainty in the

environment. Additionally, insistence on sameness behaviors in ASD may reflect, at least in part, processes to reduce anxiety in the face of intolerance of uncertainty. Anxiety disorders are characterized by active avoidance of high uncertainty situations to avoid accompanying negative emotions. Outside of the context of ASD, anxiety is modulated by predictability even if the uncertain outcomes do not have aversive consequence (Grupe and Nitschke 2013). Additionally, anxiety is a cause of ritualistic and repetitive behaviors, including benign leg-swinging in typically developing children (Soussignan and Koch 1985) and self-injurious stereotypies in animal model studies (Fox 1986). Across species, there is compelling evidence that ritualistic and repetitive behaviors that manifest under conditions of uncertainty or unpredictability are anxiolytic, serving to elicit calm under conditions of stress (Eilam et al. 2011). In this manner, insistence on sameness and associated ritualistic behaviors in the context of uncertainty may be adaptive responses to anxiety that is prompted by intolerance of uncertainty.

Indeed, anxiety symptoms and disorders are highly prevalent in ASD and anxiety is a common treatment target for individuals with ASD. Further, there appears to be a clear linkage between symptoms related to intolerance of uncertainty and anxiety in ASD. For example, in children with ASD with clinically significant anxiety, Rodgers et al. (2012) found a correlation between anxiety symptoms and insistence on sameness behaviors, but not other ASD symptoms. Similarly, Abramson et al. (2005) reported that children with increased insistence on sameness behaviors were more likely to have parents with anxiety symptoms.

Emotional Regulation in ASD

Although the core features of ASD are in the domains of social communication and restricted and repetitive behaviors, ASD is characterized by broad impairments in affective expression and regulation, including tantrums, aggression, self-injury, anxiety, and irritability (Lecavalier 2006). Emotion regulation is a critical adaptive response that appears to be impaired in ASD, and these

impairments may contribute to the increased rates of internalizing disorders in ASD given that such disorders are themselves characterized by impaired emotion regulation (White et al. 2009). Moreover, emotion regulation impairments in ASD are associated with the overall severity of all core ASD symptoms (Samson et al. 2014) and compromise the quality and quantity of early social interactions (White et al. 2014). Children with ASD frequently display decreased positive and increased negative emotional responses and poor recognition of emotions (Mazefsky et al. 2013). As already discussed, intolerance of uncertainty is a hallmark of anxiety disorders and is an associated feature of a number of ASD characteristics, including insistence on sameness and rigid adherence to routines. Additionally, given that, under conditions of uncertainty, ritualistic and repetitive behaviors may help to reduce anxiety, it may be the case that impaired emotion regulation may similarly be a risk factor for intolerance of uncertainty given impairments with emotion regulation that characterize ASD. In other words, impaired emotion regulation, combined with intolerance of uncertainty, may together potentiate anxiety symptoms that are so commonly observed in ASD. Indeed, there is some emerging evidence that intolerance of uncertainty is associated with emotional dysregulation in ASD, even when controlling for symptoms of anxiety (Vasa et al. 2018).

Treatment Models for Uncertainty Processing

Outside of the context of ASD, most theories of psychosocial interventions for intolerance of uncertainty rely on cognitive strategies, including changing negative assumptions about uncertainty and seeking additional information. Based on a model in which uncertainty is a core deficit in anxiety disorders, Ladouceur and colleagues (2000a) designed a cognitive behavioral treatment for intolerance of uncertainty that addresses the four main components of intolerance of uncertainty. This treatment involved explaining the concept of intolerance of uncertainty; how to recognize, accept, and tolerate uncertainty in adaptive ways; the distinction between real and

imagined negative outcomes; reevaluation of potentially positive beliefs about uncertainty; problem-solving training; and finally, exposure. A randomized controlled trial of adults with anxiety disorders found improvement in intolerance of uncertainty, anxiety symptoms, worry, and depressive symptoms that were maintained at 6- and 12-month posttreatment assessments, with 77% of the sample no longer meeting diagnostic criteria for anxiety disorders at both assessments (Ladouceur et al. 2000a). This treatment model suggests that intolerance of uncertainty and uncertainty processing are amenable to psychosocial intervention.

Although treatment methods that use cognitive strategies to cope with the intolerance of uncertainty in ASD are beginning to be evaluated, it is largely still unknown to what extent these approaches would be beneficial in the ASD population, and which patient characteristics might predict better treatment response. In general, individuals with ASD who have more skilled communication abilities are good candidates for psychosocial interventions (Wyman and Claro 2019), and future clinical trials should evaluate the efficacy of psychosocial interventions in targeting intolerance of uncertainty in ASD. In an initial study in this area, Keefer et al. (2017) examined whether intolerance of uncertainty affected outcomes following cognitive behavioral therapy for anxiety in children with ASD. They found that higher levels of pretreatment intolerance of uncertainty predicted higher anxiety before and after treatment, suggesting that targeting the intolerance of uncertainty may improve outcomes for therapy in youth with ASD. Finally, Rogers and colleagues (2018, 2017) recently developed “Coping with Uncertainty in Everyday Situations for Autism (CUES-A©),” a manualized treatment that targets intolerance of uncertainty in children with ASD. This intervention trains parents to help their children to manage intolerance of uncertainty. A recent initial validation study suggested that the intervention is feasible, acceptable, and valued by families. These results indicate that larger trials are warranted to evaluate the true effectiveness of this intervention.

Behavioral Measures of Uncertainty Processing

Behavioral differences have also been found in terms of responding to uncertainty. Using a task referred to as the “beads task,” which has been well-validated for measuring individual differences in responses to uncertainty (Broome et al. 2007; Ladouceur et al. 1997), adolescents with ASD tend to seek more information in an uncertain context before making a decision relative to controls (Brosnan et al. 2014). However, it may also be important to note that another study involving an adult sample found that the ASD group chose fewer beads (i.e., sought less information) than the control group (Jänsch and Hare 2014), suggesting that adults with ASD may use a “jumping to conclusions” approach (i.e., choosing only one or two beads before making a decision) as a coping strategy for avoiding the experience of uncertainty.

Neurobiological Measures of Uncertainty Processing

There is also emerging evidence from neurobiological studies for atypical uncertainty processing in ASD. For instance, an event-related potential referred to as the “mismatch negativity (MMN)” indexes differences in the processing of unexpected events. Evidence for the MMN response in ASD is somewhat mixed. In individuals with ASD, the MMN response has been found to be reduced (Seri et al. 1999), increased (Ferri et al. 2003; Gomot et al. 2002), and, in some cases, statistically equivalent (Čeponienė et al. 2003; Kemner et al. 1995). In addition, studies of the P3a (an event-related potential response related to novelty processing) has consistently found attenuated P3a responses in children with ASD (Čeponienė et al. 2003; Courchesne et al. 1985, 1994; Dawson et al. 1988; Kemner et al. 1995; Lincoln et al. 1993; Oades et al. 1988), indicating that children with ASD may be impaired in processing unexpected change.

Using a passive functional MRI task examining the processing of unexpected stimuli within a series of predictable stimuli, Gomot et al. (2006) found that children with ASD showed relatively

reduced activation of the left anterior cingulate during the processing of unexpected stimuli. When this study was repeated as an active choice task, Gomot et al. (2008) found that children with ASD showed relatively increased activity in the right superior/middle and inferior frontal gyrus, right precentral gyrus, right postcentral gyrus, left inferior parietal lobule, and left middle frontal gyrus, as well as attenuated activation in the right caudate. Children with ASD were also faster at detecting the unexpected stimulus. When viewed in the light of the findings of Gomot et al. (2006), these results may suggest that children with ASD are able to process uncertain stimuli more effectively when their attention is directed toward it. However, it is important to note that Gomot et al. (2006) did not collect behavioral data and further research in this area is clearly needed in order to support these findings and rule out possible alternate explanations. In particular, attentional or multisensory processing deficits in ASD may explain this pattern of results. In addition, because response times were faster in the ASD group than the control group in this study, the hemodynamic response (as measured by fMRI) may have simply occurred earlier in the ASD group than the control group.

Future Directions

Despite evidence for the high prevalence of intolerance of uncertainty and insistence on sameness in ASD (Lam and Aman 2007; Richler et al. 2007) and evidence that sameness and ritualistic behaviors in ASD significantly contribute to the stress of caregivers and adults with ASD (Boulter et al. 2014; Gabriels et al. 2005; Gillott and Standen 2007), the study of uncertainty processing in ASD is clearly an emerging field. Although intolerance of uncertainty has been studied extensively in individuals with anxiety disorders, the recognition of its relevance to core and associated ASD symptoms is a relatively new topic of inquiry. Although emerging data suggests that intolerance of uncertainty is related to insistence on sameness symptoms, anxiety, and emotion regulation impairments in ASD, research is needed to evaluate the

extent to which the intolerance of uncertainty is associated with other ASD features. For example, intolerance of uncertainty in ASD has been found to be related to sensory sensitivities (Neil et al. 2016), and, together with anxiety, intolerance of uncertainty has been shown to mediate relations between sensory sensitivities and repetitive behaviors in children with ASD (Wigham et al. 2015). Additionally, Vasa et al. (2018) found that the severity of intolerance of uncertainty was related to the magnitude of social communication deficits, repetitive behaviors, and emotion dysregulation in children with ASD *while controlling for anxiety*. This important finding highlights the far-reaching impact of intolerance of uncertainty as it is related to core ASD symptoms. However, the extent to which interventions that target intolerance of uncertainty in ASD also impact the core symptoms of ASD remains to be evaluated.

A better understanding of the role of uncertainty processing in ASD may have important implications for identifying the active ingredients in effective behavioral interventions for ASD. Many empirically validated interventions for ASD are designed to increase predictability and minimize uncertainty, such as applied behavior analysis (ABA). The study of uncertainty processing in ASD may also inform the development of early intervention programs. If the uncertainty of social stimuli decreases their motivational salience, infants and toddlers at risk for ASD may choose to avoid or ignore social stimuli more and more over the course of development, ultimately resulting in greater social and communicative impairments later in life. Consequently, early intervention programs that increase the predictability and consistency of social stimuli may help to prevent children from following this aberrant developmental trajectory. Interventions for ASD may also incorporate a component that directly addresses the elevated levels of intolerance of uncertainty in this population, such as the intervention designed by Rodgers et al. (2017).

Given the transdiagnostic nature of uncertainty processing, it is likely that the lessons learned about cognitive mechanisms of intolerance of uncertainty in other psychiatric disorders, particularly anxiety disorders, may guide research into

intolerance of uncertainty and its treatment in ASD. The evidence highlighting the success of psychosocial treatments in targeting intolerance of uncertainty in anxiety disorders (Ladouceur et al. 2000a) combined with the high rates of anxiety disorders in ASD (Leyfer et al. 2006), suggest that lessons learned from studying and treating intolerance of uncertainty in anxiety disorder may be a promising starting point to more fully investigate intolerance of uncertainty and ultimately develop treatments for intolerance of uncertainty in ASD.

References and Reading

- Abramson, R. K., Ravan, S. A., Wright, H. H., Wieduwilt, K., Wolpert, C. M., Donnelly, S. A., et al. (2005). The relationship between restrictive and repetitive behaviors in individuals with autism and obsessive compulsive symptoms in parents. *Child Psychiatry and Human Development*, 36(2), 155–165. <https://doi.org/10.1007/s10578-005-2973-7>.
- APA. (1980). *Diagnostic and statistical manual of mental disorders*. 3rd ed. Washington, D.C.: American Psychiatric Association.
- APA. (1987). *Diagnostic and statistical manual of mental disorders*. 3rd ed., revised. Washington, D.C.: American Psychiatric Association.
- APA. (1994). *Diagnostic and statistical manual of mental disorders: DSM-IV* (4th ed.). Washington, DC: American Psychiatric Association.
- APA. (2013). *Diagnostic and statistical manual of mental disorders: DSM-V* (5th ed.). Washington, DC: American Psychiatric Association.
- Baron-Cohen, S. (1989). Do autistic children have obsessions and compulsions? *British Journal of Clinical Psychology*, 28(3), 193–200.
- Baron-Cohen, S., & Wheelwright, S. (1999). ‘Obsessions’ in children with autism or Asperger syndrome. Content analysis in terms of core domains of cognition. *The British Journal of Psychiatry*, 175(5), 484–490.
- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a “theory of mind”? *Cognition*, 21(1), 37–46.
- Boucher, J. (1977). Alternation and sequencing behaviour, and response to novelty in autistic children. *Journal of Child Psychology and Psychiatry*, 18(1), 67–72.
- Boulter, C., Freeston, M., South, M., & Rodgers, J. (2013). Intolerance of Uncertainty as a Framework for Understanding Anxiety in Children and Adolescents with Autism Spectrum Disorders. *Journal of Autism and Developmental Disorders*, 1–12.
- Boulter, C., Freeston, M., South, M., & Rodgers, J. (2014). Intolerance of uncertainty as a framework for understanding anxiety in children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 44(6), 1391–1402. <https://doi.org/10.1007/s10803-013-2001-x>.
- Broome, M. R., Johns, L. C., Valli, I., Woolley, J. B., Tabraham, P., Brett, C., et al. (2007). Delusion formation and reasoning biases in those at clinical high risk for psychosis. *The British Journal of Psychiatry Supplement*, 51, s38–s42. <https://doi.org/10.1192/bjp.191.51.s38>.
- Brosnan, M., Chapman, E., & Ashwin, C. (2014). Adolescents with autism spectrum disorder show a circumspect reasoning bias rather than ‘jumping-to-conclusions’. *Journal of Autism and Developmental Disorders*, 44(3), 513–520. <https://doi.org/10.1007/s10803-013-1897-5>.
- Buhr, K., & Dugas, M. J. (2009). The role of fear of anxiety and intolerance of uncertainty in worry: An experimental manipulation. *Behaviour Research and Therapy*, 47(3), 215–223. <https://doi.org/10.1016/j.brat.2008.12.004>.
- Carleton, R. N. (2012). The intolerance of uncertainty construct in the context of anxiety disorders: Theoretical and practical perspectives. *Expert Review of Neurotherapeutics*, 12(8), 937–947. <https://doi.org/10.1586/ern.12.82>.
- Čeponienė, R., Lepistö, T., Shestakova, A., Vanhala, R., Alku, P., Nääätänen, R., & Yaguchi, K. (2003). Speech-sound-selective auditory impairment in children with autism: They can perceive but do not attend. *Proceedings of the National Academy of Sciences of the United States of America*, 100(9), 5567.
- Chamberlain, P. D., Rodgers, J., Crowley, M. J., White, S. E., Freeston, M. H., & South, M. (2013). A potentiated startle study of uncertainty and contextual anxiety in adolescents diagnosed with autism spectrum disorder. *Molecular Autism*, 4(1), 1–11.
- Courchesne, E., Lincoln, A. J., Kilman, B. A., & Galambos, R. (1985). Event-related brain potential correlates of the processing of novel visual and auditory information in autism. *Journal of Autism and Developmental Disorders*, 15(1), 55–76.
- Courchesne, E., Townsend, J., Akshoomoff, N. A., Saitoh, O., Yeung-Courchesne, R., Lincoln, A. J., et al. (1994). Impairment in shifting attention in autistic and cerebellar patients. *Behavioral Neuroscience*, 108(5), 848.
- Dawson, G., & Lewy, A. (1989). Arousal, attention, and the socioemotional impairments of individuals with autism. In G. Dawson (Ed.), *Autism: Nature, diagnosis, and treatment* (pp. 49–74). New York: Guilford Press.
- Dawson, G., Finley, C., Phillips, S., Galpert, L., & Lewy, A. (1988). Reduced P3 amplitude of the event-related brain potential: Its relationship to language ability in autism. *Journal of Autism and Developmental Disorders*, 18(4), 493–504.
- Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., & Liaw, J. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology*, 40(2), 271–282.
- Eilam, D., Izhar, R., & Mort, J. (2011). Threat detection: Behavioral practices in animals and humans.

- Neuroscience and Biobehavioral Reviews*, 35(4), 999–1006. <https://doi.org/10.1016/j.neubiorev.2010.08.002>.
- Ferrara, C., & Hill, S. D. (1980). The responsiveness of autistic children to the predictability of social and non-social toys. *Journal of Autism and Developmental Disorders*, 10(1), 51–57.
- Ferri, R., Elia, M., Agarwal, N., Lanuzza, B., Musumeci, S. A., & Pennisi, G. (2003). The mismatch negativity and the P3a components of the auditory event-related potentials in autistic low-functioning subjects. *Clinical Neurophysiology*, 114(9), 1671–1680.
- Fox, M. W. (1986). *Laboratory animal husbandry: Ethology, welfare, and experimental variables*. Albany: State University of New York Press.
- Gabriels, R. L., Cuccaro, M. L., Hill, D. E., Ivers, B. J., & Goldson, E. (2005). Repetitive behaviors in autism: Relationships with associated clinical features. *Research in Developmental Disabilities*, 26(2), 169–181.
- Gillott, A., & Standen, P. J. (2007). Levels of anxiety and sources of stress in adults with autism. *Journal of Intellectual Disabilities*, 11(4), 359–370. <https://doi.org/10.1177/1744629507083585>.
- Gomot, M., Giard, M. H., Adrien, J. L., Barthelemy, C., & Bruneau, N. (2002). Hypersensitivity to acoustic change in children with autism: Electrophysiological evidence of left frontal cortex dysfunctioning. *Psychophysiology*, 39(5), 577–584.
- Gomot, M., Bernard, F. A., Davis, M. H., Belmonte, M. K., Ashwin, C., Bullmore, E. T., & Baron-Cohen, S. (2006). Change detection in children with autism: An auditory event-related fMRI study. *NeuroImage*, 29(2), 475–484.
- Gomot, M., Belmonte, M. K., Bullmore, E. T., Bernard, F. A., & Baron-Cohen, S. (2008). Brain hyper-reactivity to auditory novel targets in children with high-functioning autism. *Brain*, 131(9), 2479–2488.
- Grupe, D. W., & Nitschke, J. B. (2013). Uncertainty and anticipation in anxiety: An integrated neurobiological and psychological perspective. *Nature Reviews. Neuroscience*, 14(7), 488–501. <https://doi.org/10.1038/nrn3524>.
- Hutt, C., Hutt, S. J., Lee, D., & Ounsted, C. (1964). Arousal and childhood autism. *Nature*, 204, 908–909.
- Jänsch, C., & Hare, D. J. (2014). An investigation of the “jumping to conclusions” data-gathering bias and paranoid thoughts in Asperger syndrome. *Journal of Autism and Developmental Disorders*, 44(1), 111–119.
- Kagan, J. (1972). Motives and development. *Journal of Personality and Social Psychology*, 22(1), 51.
- Kanner, L. (1944). Early infantile autism. *The Journal of Pediatrics*, 25(3), 211–217.
- Keefer, A., Kreiser, N. L., Singh, V., Blakeley-Smith, A., Duncan, A., Johnson, C., et al. (2017). Intolerance of uncertainty predicts anxiety outcomes following CBT in youth with ASD. *Journal of Autism and Developmental Disorders*, 47(12), 3949–3958. <https://doi.org/10.1007/s10803-016-2852-z>.
- Kemner, C., Verbaten, M. N., Cuperus, J. M., Camfferman, G., & van Engeland, H. (1995). Auditory event-related brain potentials in autistic children and three different control groups. *Biological Psychiatry*, 38(3), 150–165.
- Kerns, C. M., Kendall, P. C., Berry, L., Souders, M. C., Franklin, M. E., Schultz, R. T., et al. (2014). Traditional and atypical presentations of anxiety in youth with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(11), 2851–2861. <https://doi.org/10.1007/s10803-014-2141-7>.
- Koerner, N., & Dugas, M. J. (2006). A cognitive model of generalized anxiety disorder: The role of intolerance of uncertainty. In *Worry and its psychological disorders* (pp. 201–216). Chichester: Wiley Publishing.
- Koerner, N., & Dugas, M. J. (2008). An investigation of appraisals in individuals vulnerable to excessive worry: The role of intolerance of uncertainty. *Cognitive Therapy and Research*, 32(5), 619–638.
- Kootz, J. P., Marinelli, B., & Cohen, D. J. (1982). Modulation of response to environmental stimulation in autistic children. *Journal of Autism and Developmental Disorders*, 12(2), 185–193.
- Ladouceur, R., Talbot, F., & Dugas, M. J. (1997). Behavioral expressions of intolerance of uncertainty in worry. Experimental findings. *Behavior Modification*, 21(3), 355–371. <https://doi.org/10.1177/01454455970213006>.
- Ladouceur, R., Dugas, M. J., Freeston, M. H., Léger, E., Gagnon, F., & Thibodeau, N. (2000a). Efficacy of a cognitive-behavioral treatment for generalized anxiety disorder: Evaluation in a controlled clinical trial. *Journal of Consulting and Clinical Psychology*, 68(6), 957.
- Ladouceur, R., Gosselin, P., & Dugas, M. J. (2000b). Experimental manipulation of intolerance of uncertainty: A study of a theoretical model of worry. *Behaviour Research and Therapy*, 38(9), 933–941.
- Lam, K. S. L., & Aman, M. G. (2007). The repetitive behavior scale-revised: Independent validation in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(5), 855–866.
- Lecavalier, L. (2006). Behavioral and emotional problems in young people with pervasive developmental disorders: Relative prevalence, effects of subject characteristics, and empirical classification. *Journal of Autism and Developmental Disorders*, 36(8), 1101–1114. <https://doi.org/10.1007/s10803-006-0147-5>.
- Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., et al. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, 36(7), 849–861. <https://doi.org/10.1007/s10803-006-0123-0>.
- Lincoln, A. J., Courchesne, E., Harms, L., & Allen, M. (1993). Contextual probability evaluation in autistic, receptive developmental language disorder, and control children: Event-related brain potential evidence. *Journal of Autism and Developmental Disorders*, 23(1), 37–58.
- Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The

- role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(7), 679–688. <https://doi.org/10.1016/j.jaac.2013.05.006>.
- Mosner, M. G., Kinard, J. L., Shah, J. S., McWeeny, S., Greene, R. K., Mazefsky, C. A., & Dichter, G. S. (2019). Rates of co-occurring psychiatric disorders in autism spectrum disorder using the mini international neuropsychiatric interview. *Journal of Autism and Developmental Disorders*, 49, 3819–3832.
- Neil, L., Olsson, N. C., & Pellicano, E. (2016). The relationship between intolerance of uncertainty, sensory sensitivities, and anxiety in autistic and typically developing children. *Journal of Autism and Developmental Disorders*, 46(6), 1962–1973. <https://doi.org/10.1007/s10803-016-2721-9>.
- Oades, R. D., Walker, M. K., Geffen, L. B., & Stern, L. M. (1988). Event-related potentials in autistic and healthy children on an auditory choice reaction time task. *International Journal of Psychophysiology*, 6(1), 25–37.
- Pierce, K., & Courchesne, E. (2001). Evidence for a cerebellar role in reduced exploration and stereotyped behavior in autism. *Biological Psychiatry*, 49(8), 655–664.
- Pring, L. (2005). Savant talent. *Developmental Medicine and Child Neurology*, 47(7), 500–503.
- Richler, J., Bishop, S. L., Kleinke, J. R., & Lord, C. (2007). Restricted and repetitive behaviors in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(1), 73–85.
- Rodgers, J., Glod, M., Connolly, B., & McConachie, H. (2012). The relationship between anxiety and repetitive behaviours in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 42(11), 2404–9.
- Rodgers, J., Wigham, S., McConachie, H., Freeston, M., Honey, E., & Parr, J. R. (2016). Development of the anxiety scale for children with autism spectrum disorder (ASC-ASD). *Autism Research*, 9(11), 1205–1215. <https://doi.org/10.1002/aur.1603>.
- Rodgers, J., Hodgson, A., Shields, K., Wright, C., Honey, E., & Freeston, M. (2017). Towards a treatment for intolerance of uncertainty in young people with autism spectrum disorder: Development of the coping with uncertainty in everyday situations (CUES(c)) Programme. *Journal of Autism and Developmental Disorders*, 47(12), 3959–3966. <https://doi.org/10.1007/s10803-016-2924-0>.
- Rodgers, J., Herrema, R., Honey, E., & Freeston, M. (2018). Towards a treatment for intolerance of uncertainty for autistic adults: A single case experimental design study. *Journal of Autism and Developmental Disorders*, 48(8), 2832–2845. <https://doi.org/10.1007/s10803-018-3550-9>.
- Samson, A. C., Phillips, J. M., Parker, K. J., Shah, S., Gross, J. J., & Hardan, A. Y. (2014). Emotion dysregulation and the core features of autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 44(7), 1766–1772. <https://doi.org/10.1007/s10803-013-2022-5>.
- Seri, S., Cerquiglini, A., Pisani, F., & Curatolo, P. (1999). Autism in tuberous sclerosis: Evoked potential evidence for a deficit in auditory sensory processing. *Clinical Neurophysiology*, 110(10), 1825–1830.
- Shah, A., & Frith, U. (1983). An islet of ability in autistic children: A research note. *Journal of Child Psychology and Psychiatry*, 24(4), 613–620.
- Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(8), 921–929. <https://doi.org/10.1097/CHI.0b013e318179964f>.
- Sinha, P., Kjelgaard, M. M., Gandhi, T. K., Tsourides, K., Cardinaux, A. L., Pantazis, D., et al. (2014). Autism as a disorder of prediction. *Proceedings of the National Academy of Sciences*, 111(42), 15220–15225.
- Soussignan, R., & Koch, P. (1985). Rhythmical stereotypies (leg-swinging) associated with reductions in heart-rate in normal school children. *Biological Psychology*, 21(3), 161–167.
- Tolin, D. F., Abramowitz, J. S., Brigidi, B. D., & Foa, E. B. (2003). Intolerance of uncertainty in obsessive-compulsive disorder. *Journal of Anxiety Disorders*, 17(2), 233–242.
- Turner-Brown, L. M., Lam, K. S., Holtzclaw, T. N., Dichter, G. S., & Bodfish, J. W. (2011). Phenomenology and measurement of circumscribed interests in autism spectrum disorders. *Autism*, 15(4), 437–456. <https://doi.org/10.1177/1362361310386507>.
- Van de Cruys, S., Evers, K., Van der Hallen, R., & Van Eylen, L. (2014). Precise minds in uncertain worlds: Predictive coding in autism. *Psychological Review*, 121(4), 649–675.
- Vasa, R. A., Kreiser, N. L., Keefer, A., Singh, V., & Mostofsky, S. H. (2018). Relationships between autism spectrum disorder and intolerance of uncertainty. *Autism Research*, 11(4), 636–644. <https://doi.org/10.1002/aur.1916>.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29(3), 216–229. <https://doi.org/10.1016/j.cpr.2009.01.003>.
- White, S. W., Mazefsky, C. A., Dichter, G. S., Chiu, P. H., Richey, J. A., & Ollendick, T. H. (2014). Social-cognitive, physiological, and neural mechanisms underlying emotion regulation impairments: Understanding anxiety in autism spectrum disorder. *International Journal of Developmental Neuroscience*, 39, 22–36. <https://doi.org/10.1016/j.jdevneu.2014.05.012>.
- Wigham, S., Rodgers, J., South, M., McConachie, H., & Freeston, M. (2014). The interplay between sensory processing abnormalities, intolerance of uncertainty, anxiety and restricted and repetitive behaviours in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 1–10.
- Wigham, S., Rodgers, J., South, M., McConachie, H., & Freeston, M. (2015). The interplay between sensory

processing abnormalities, intolerance of uncertainty, anxiety and restricted and repetitive behaviours in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(4), 943–952. <https://doi.org/10.1007/s10803-014-2248-x>.

Wilkinson, A., Freeston, M., & Meares, K. (2011). *CBT for worry and generalised anxiety disorder*. Los Angeles: Sage.

Wyman, J., & Claro, A. (2019). The UCLA PEERS school-based program: Treatment outcomes for improving social functioning in adolescents and young adults with autism Spectrum disorder and those with cognitive deficits. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03943-z>.

Under-arousal

► Hypo-arousal

Understanding Parent's Intervention Choices for Their Child with ASD

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Definition

Understanding the decisions made by parents when choosing interventions for their child with autism spectrum disorder.

Historical Background

Autism spectrum disorder (ASD) predisposes an individual with a number of core symptoms and comorbid problem behaviors that are well documented but as yet adequately explained. While modern medicine exists to protect, promote, or restore health, the complexities inherent in a neurodevelopmental disorder such as ASD create scientific challenges when seeking to elucidate its biological underpinnings. This lack of

understanding, in turn, leads to clinical challenges in assessment, diagnosis, treatment, and prevention. Currently, there is no universally accepted theory of ASD causation, nor is there a cure. However, the absence of an etiological base has not prevented the establishment of numerous ASD-related interventions, which in number now run into the hundreds. In the medical sense, an intervention exists to either eliminate or reduce a set of target symptoms. Since its establishment as a diagnostic category, formalized in the *Diagnostic and Statistical Manual of Mental Disorders* (Version 3; 1980) by the American Psychiatric Association, ASD interventions have continued being symptomatic and not curative. Of these interventions, many have been shown to be ineffective or are argued to be unethical, unsafe, or else have yet to be evaluated despite their widespread use. As such, there exists no universal standard of care across health professionals, and consequently in many parts of the world, it is the parents who have historically shouldered the responsibility of choosing their child's ASD-related interventions. Though the choices available to parents have increased over the last 50 or so years, the diverse symptomology of their child's disorder means no "one-stop shop" for treatment exists, which further complicates the decisions parents have to make while under pressure to "get it right."

Only 60 years ago the cause of ASD was largely thought to arise from children being raised in emotionally static environments by "refrigerator mothers" whose lack of affection and closeness to their child interfered with attachment and personality development. Consequently, intervention choice was limited to psychoanalytical approaches specifically designed to undo the development damage wrought by parents. In the later part of 1960s and 1970s, the refrigerator mother hypothesis was debunked and put to rest, only to be replaced by another hypothesis that was later to be discredited, the autism/vaccination hypothesis. Not only would it take decades to finally discredit explanations based on the effects of vaccines and other environmental pollutants (e.g., lead), but the biological interventions they gave rise to, such as chelation therapy and secretin supplementation, served only to install parents with false hope and deliver little in

the way of efficacy. By the 1980s, an increasing awareness of the genetic contribution to ASD emerged, though the provision of such knowledge was not immediately translated into new approaches to intervention. In 1987, what has become known as the Lovaas method, or more latterly intensive applied behavioral analysis (iABA) therapy, was described. Though multiple and diverse behavioral approaches to the treatment of ASD had been reported previously, for example, electric shock therapy in the 1950s, the Lovaas approach emphasized early intervention typified by high-intensity treatment regimens delivered one-on-one in clinical settings and then continued by parents at home. While fringe biomedical interventions such as electroconvulsive therapy and medications exist in contemporary clinical practice, the behavioral techniques evolving from the Lovaas method have been the most scrutinized and arguably the most supported by scientific data.

In contemporary times the increasing awareness of ASD has unleashed a multitude of interventions claiming, with or without evidence, to mitigate symptoms or even offering a total cure. Broadly, interventions are classed as biologically based, targeting a specific physiological process that has been found, or hypothesized, to be dysfunctional, or psychologically based, in which the goal is to increase or decrease target behaviors through established conditioning techniques. In clinical practice the most common biological treatments are prescription medicines, where different families of drugs generally focus on a specific symptom or comorbid condition, as opposed to targeting ASD in the broader sense. In the USA, for example, the prescription of antidepressant, anxiolytic, antipsychotic, seizure-control, mood stabilizer, and stimulant medication occurs alongside medication targeting gastrointestinal- or sleep-related difficulties (Goin-Kochel et al. 2009). Beyond the clinic, parents may administer complementary and alternative medicines (CAMs) to their children or subject their children to dietary regimens such as wheat-free and/or dairy-free diets or high doses of vitamins. Of the psychological interventions, speech language therapy, occupational therapy, iABA therapy, and orthodox behavioral therapies are the most

commonly funded and therefore most engaged internationally. Of note, while many of the psychological interventions differ substantially in their target symptoms and behaviors, they still apply the treatment modality characteristics of behavioral therapy such as operant, classical, and vicarious conditioning.

Though some advocate strongly for a parental voice when choosing interventions for their child, the time-critical nature of ASD-related interventions requires parents to make important decisions at a time when they may be in shock and highly emotionally charged following the delivery of their child's ASD diagnosis. While parents have a duty of care to their child, pressure to immediately begin interventions can be overwhelming, and parents may experience choice overload in the face of the sheer number of available interventions and conflicting information about their efficacy. In the face of inadequate information, and beset by negative emotions and feelings of worry for their child's future, parents will often subscribe to any intervention on offer while adopting a wait-and-see approach. Of concern, rather than choosing multiple interventions as a strategy to treat a complex disorder, the concurrent use of evidence-based and non-evidence-based interventions is more likely a representation of the sheer desperation and frustration felt by parents. Accordingly, research suggests that at any one time, parents are trying up to seven types of intervention with their child, which in turn may be placing immense strain on parents and the greater family unit. That some of these interventions are ineffective and financially punitive demonstrates that parents are not getting the support they need when making decisions about their child's interventions. To that end, in order to better support parents in their intervention decisions, it is important to catalogue the reasons why parents engage or avoid specific interventions.

Current Knowledge

Inspection of the research to date suggests that parental decisions regarding their child's ASD-related interventions can be grossly classified into

three outcomes. First, and for whatever reason, parents are in opposition to the intervention, and therefore the decision is made for their child not to engage it. Second, the parent wants their child to participate in the intervention and has the psychological and physical resources to ensure that their child receives that treatment. Third, there is a strong desire on the part of the parent for their child to receive a particular intervention, though for whatever reason there are barriers preventing the child's engagement. For the first and second outcomes, it is of interest to understand what is driving the parent's decision, especially in relation to child characteristics or external influences. For the second and third outcomes, the interest lies in those facilitating or impeding factors, both internal and external to the parents, that determine whether their child receives a specific intervention or not. In relation to the second outcome, it is also of interest to know why parents, having started their child in a particular intervention, make the decision to continue or discontinue it.

When a parent that has the means and circumstances to enrol their child into an intervention and expresses opposition to do so, then their decision is typically motivated by ideology, skepticism, concerns for child safety or acceptance, and, relatedly, professionalism or personability of intervention providers. In terms of ideology, cultural, family, and religious factors may influence how parents attribute causality to their child's condition and consequently their choice of intervention. Interventions that do not conform to a family's belief or culture may be sternly opposed, and data from the USA suggests that one in five Americans are exposing their children to faith-based therapies (Miller et al. 2012). The notion that parental skepticism may be driving the nonengagement of some interventions indicates that some parents may in fact be referencing scientific evidence and not conforming to the "initiate-wait-then-evaluate" pattern floated in the literature. Remarkably, however, skepticism may not be related to whether an intervention has received backing from the scientific community. In an Australasian study (Shepherd et al. 2018), it was reported that iABA therapy, which is considered the gold standard intervention to which others should be

benchmarked, attracted equivalent numbers of skeptical parents as dietary interventions which are arguably a polar opposite in terms of established efficacy. Further, proven effectiveness of an intervention may not carry much weighting if parents prioritize symptoms that are not targeted by that intervention and are perceived as less urgent, and the individual needs of the child commonly determine if an intervention is rejected. In terms of safety and intervention side effects, it appears that parents have reluctance to engage with biological treatments when their children are younger, where medicine is tried as a last resort. Generally, however, parents find it difficult to judge the safety of interventions, and whether an intervention is evidence-based or not may hold little sway over how parents perceive risk. Finally, parents may hear word of mouth that a particular service provider has been disrespectful to parents or children, leading them to outrightly reject the idea of their children being treated by that provider and thus taint that intervention modality.

Signing up children for ASD-related interventions typically brings substantial time and financial pressures to parents, and yet the evidence shows that children are on average exposed to 3–4 interventions at any one time. It is therefore of interest to understand the reasons why parents take on this "tyranny of interventions" and why they choose the interventions they do. In the ASD literature, it has been argued that the major influence of parental decision-making is endorsement from others, including medical professionals and therapists, friends and relatives, other parents of a child with ASD, and even celebrity promotion of "fad" interventions. Overall, it appears that parents place greater weighting on advice dispensed from professionals (e.g., medical doctors, therapists, and teachers) than from nonprofessionals. However, this may be a double-edged sword as when seeking advice from professionals, parents may be vulnerable to professional recommendations based on "professional opinion" rather than guided by evidence-based practice. This is of concern as it is often those professionals involved at the time of diagnosis that have a major influence on a child's intervention trajectory. Rightly or wrongly, however, parents generally view

medical professionals as trusted authority figures, and as such professionals need to base their recommendations on empirically supported interventions and not on personal beliefs. A further influence, though one has yet to be studied in sufficient detail, is the influence of the Internet and social media upon parental intervention decisions. Though findings to date have been mixed, the fact that parents rely heavily upon social media as a source of social support would suggest that other parents with ASD children maybe exerting a greater dominance than hitherto detected in past studies (Shepherd et al., 2020). An issue arises here in that taking advice from another parent with an ASD child may not lead to the best intervention outcome given the heterogeneity of symptom profiles across the autistic spectrum.

For many parents, the load of managing an ASD child, a family, and work life impedes the ability to access or interpret the body of scientific literature dedicated to intervention efficacy. Studies generally show that when selecting interventions for their children, parents are not especially motivated by whether the intervention is backed up by empirical evidence or not, as can be seen with the uptake rates of unvalidated dietary interventions. Instead, parents may be guided by “gut feelings” about services and the prioritizing of staff knowledge, experience, and skills over scientific evidence. This can be compared to what is known as the “dodo effect” in psychological practice, whereby the rapport (or “therapeutic alliance”) between a client and their therapist is argued to better predict the treatment outcome than the type of therapy itself. Intuitively, however, it is understandable why parents may be drawn to intervention services with staff possessing strong interpersonal skills and clinical experience, and because many services begin with an initial assessment of the child, parents are provided with an opportunity to form opinions of the staff and decide whether or not to engage with this intervention provider. What is clear, however, is that emotional factors weigh heavily on the decisions that parents make, and the general consensus is that parents are more swayed by glitzy advertisements freighted anecdotal reports and

“word of mouth” than scientific evidence. What is more, this shunning of the scientific evidence is not dependent upon the level of education, where even those with university-level qualifications may be vulnerable to “gut feelings” or parental intuitions that are driven by fear of missing out on the “next big thing” in ASD intervention or by worry of regression in their child’s progress to date. Consequently, parents may not disclose the use of interventions that lack empirical support to their doctors and therapists, suggesting that on some level they have a knowledge that their decisions may be cavalier or even dangerous to their child but at the same time not wanting to be criticized or pressured to disengage.

Fortunately, across the limited number of studies investigating parental choices of their child’s ASD-related interventions, it is clear that most decisions are child-centric and that the individual needs of the child are the overriding factor. Specifically, symptom type and symptom severity appear to carry the greatest weight when parents are mulling their child’s interventions; for example, speech language therapy would be selected to target communication difficulties. These findings indicate that either parents are considering the functional abilities of their child and prioritizing accordingly or else the services on offer are doing an effective job of matching interventions to typical ASD symptoms and severity thereof. However, not only does parental rating of their child’s ASD symptom severity guide intervention choice, but proportionality also determines the number of interventions engaged. In the USA, differences in parental intervention choice have been shown to vary as a function of child diagnosis, both for the types and numbers of interventions chosen. Pertinently, and with reference to DSM-3 diagnostic criteria, those with autism or PDD-NOS (pervasive developmental disorder-not otherwise specified) typically engage in more interventions than those with Asperger’s syndrome, and they are generally more psychological in nature. Beyond the core symptoms of ASD, parents also have to grapple with comorbid problem behaviors and deficits in everyday skills that impede child function. In terms of the latter, parents may be highly motivated to engage interventions that

address deficits in areas such as road sense, friendship, joint attention, and safety with strangers. Thus for parents, the presence of comorbidities and the lack of age-appropriate skills serve to further complicate intervention decisions.

Few studies exist that probe the reasons why parents choose to terminate interventions once they are in progress, though it has been suggested that, beyond the expected discontinuation that occurs when therapy goals are realized, such terminations are commonplace. On occasion discontinuation occurs when the child reaches an age, whereby that intervention is no longer funded, even though parents are satisfied with the outcomes of the intervention itself. Related cost is a common reason given by parents to terminate an intervention, and in countries where iABA therapy is not funded, parents on average incomes or less would struggle to meet payments. Research has shown that if parents begin to doubt an intervention's impact, they will likely cease engagement and potentially move to an untried intervention. This is especially evident as the child moves towards adolescence, where there is a noted shift from psychological to biological interventions. As the child gets older, the use of prescribed medicines increases, possibly driven by the emergence of problem behaviors during puberty that are otherwise not being treated by psychological interventions. That being the case, the discontinuation rate of medications is also reported to be high, possibly due to drugs that from the outset were designed to target other conditions and were either not benefitting the child or were causing unpleasant side effects. Predictably, parents will abandon interventions if they perceive their child to be in pain or discomfort. Similarly, dietary interventions are commonly ended by parents due to a perceived lack of progress, constituting wasted time and financial resources that would have been better spent on validated interventions. However, even though parents may be dissatisfied with an intervention, they may still persevere if it provides them with respite.

Related to those parents who are able to engage their child in a specific ASD intervention are those parents who desire to engage but encounter barriers. On this point it is important to acknowledge that the nonengagement of an intervention does not

always signal parental opposition *per se*. Firstly, some interventions are simply not available to be selected by parents due to factors such as geographical location. Where an intervention is not offered in the immediate locality of the family, the only choices are to miss out or uproot the family and move to a place where the intervention is offered. For other parents, the intervention may be within their commuting range, but they are just unaware of the service's existence. In this scenario, it may be that practitioners are failing to fully inform parents about the broad spectrum of available interventions in their area. It has been suggested that in clinical practice, therapists are reluctant to inform parents about evidence-based interventions out of concern that the parents cannot afford it. Indeed, for some parents it may be that the intervention is accessible, but they do not have the financial resources for their child to engage it. Typically, expense is a primary reason why a parent might want to engage their child in a particular intervention but does not, and consequently government-funded interventions are much more likely to be chosen than those interventions that are self-funded. There is no evidence that parents who do not oppose a specific intervention and can afford to enrol their child still refuse to engage on the basis of cost. Instead, there is evidence that parents borrow or obtain funding by other means to get their child the support they need, including bank loans and, in the case of stem cell implants, "crowdfunding" over the internet.

The inability to effectively manage time given the demands of caring for a child with ASD might also be expected to make parents avoid particular interventions, but there is currently mixed evidence as to whether time constraints are a barrier to their child engaging an intervention, nor are time-related barriers commonly cited as a reason to discontinue an already-engaged intervention. On the face of it, this comes as a surprise, given the extensive parental involvement demanded by many of the mainstream psychological interventions, and as such may reflect parent's commitment to their child. Indeed, one study discusses the importance of intervention "fit" in relation to families' schedule and routines (Goin-Kochel et al. 2007) and the pressure placed on parents to oversee home-based

interventions. Despite the daunting task of collaborating in their child's interventions, most parents seem to "just get on with it" and engage in interventions irrespective of the resulting stress or the strength of their support networks to sustain them. Finally, child characteristics such as age and extreme problem behaviors may also prevent parents from choosing some types of interventions. For psychological interventions age is an important consideration as the child needs to be mature enough for a structured approach, while for biological approaches the use of elimination diets seems more popular in early childhood, perhaps as parents have greater control of their child's diet. Further, it appears that practitioners are less likely to advocate medication until children enter adolescence.

Future Directions

While cases of ASD are increasing globally as an expected consequence of population growth, there is convincing evidence suggesting that the prevalence rates of ASD are also increasing. Consequently, the economic burden of ASD is likely to increase, and not only does this increase demand for effective interventions, but it also entails parents to exhibit prudence when strategizing their child's intervention trajectories as demand and cost of interventions increase. In the defense of parents and their choices, the first step towards intervention optimization lies in the development and evaluation of a broader range of ASD-related interventions, and it would be unfair at the current time to judge parents on their choices when the selection of empirically assessed interventions is so thin. The next step entails formal support sources to be equipped with the knowledge to guide parents in their intervention decisions. Currently it is not clear whether parents are prioritizing other factors (e.g., "word of mouth") over research evidence because they are unaware of the importance or existence of research or if they are dismissing its importance in favor of other factors, including emotion-based factors such as "gut feelings." Though reluctant to engage in interventions that may be ineffective,

expensive, time-consuming, or even harmful to their child, parents may find themselves being unduly influenced by therapists or other parents with ASD children or else relying on gut feelings when they don't have the time or know-how to access research evidence. Future researchers may investigate whether the provision of information about evidence-based practice would influence the weighting that parents place on research evidence in their decision-making. On this matter, when considering external influences, more investigation needs to occur around the weighting that parents place on the informational sources themselves, that is, is the medium the message?

Research shows that when parents and practitioners collaborate in decision-making processes, the parents feel more invested and included in their child's health care (Herbert 2014). However, to facilitate joint decision-making processes, professionals need to be mindful that what they believe works best for the child may not be what works best for the parents or the greater family. With this in mind, and acknowledging that parents are the ultimate authorities of their child's treatment, professionals need to formulate treatment paths that optimize intervention outcomes while also being compatible with the larger family system. To this end it might be useful to produce a set of clinical protocols that can be adopted universally and continuously improved and adapted over time. However, challenges in producing a formalized set of practice guidelines exist in as much as substantial variations exist internationally and even within nations. Consequently, what will be effective in one locality may not be in another.

A further issue involves considering legislative frameworks and whether or not intervention choices should in fact be sitting entirely with the parent. Pertinently, the freedom of choice bestowed upon parents may create cases where children receive no intervention at all, which can arguably be considered the equivalent of withholding treatment. Ethically, the withholding of treatment is justifiable only when to do so is in the best interests of the child, and the "best interests" argument often underlies legal criteria which focus on the child's future quality of life, the effectiveness of treatments, and the broader costs versus benefits of

treatments. In the ASD context, it is not uncommon for parents to experience denial immediately following the diagnosis of their child or the emergence of their child's core symptoms. As such, they may refuse to enrol their children in interventions during time-critical windows of development. In such cases should medical professionals be seeking compulsory treatment orders for the child? Further, by not engaging their child in interventions, might parents be committing the equivalent of medical malpractice, and does this non-engagement constitute a form of negligence to which they should be held accountable? Alternatively, what if a parent insists on unproven interventions that are harming their child? Either way, the amount of responsibility put upon parents is one that evidently requires further discussion at the legal, medical, and societal levels.

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.
- Bowker, A., D'Angelo, N. M., Hicks, R., & Wells, K. (2011). Interventions for autism: Parental choices and perceptions of change. *Journal of Autism and Developmental Disorders*, 41, 1373–1382.
- Carlson, S., Carter, M., & Stephenson, J. (2013). A review of declared factors identified by parents of children with autism spectrum disorders (ASD) in making intervention decisions. *Research in Autism Spectrum Disorders*, 7, 369–381.
- Carlson, S., Carter, M., & Stephenson, J. (2015). Decision-making regarding early intervention by parents of children with autism spectrum disorder. *Journal of Developmental and Physical Disabilities*, 27, 285–305.
- Goin-Kochel, R. P., Myers, B. J., & Mackintosh, V. H. (2007). Parental reports on the use of treatments and therapies for children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 1, 195–209.
- Goin-Kochel, R. P., Mackintosh, V. H., & Myers, B. J. (2009). Parental reports on the efficacy of interventions and therapies for their children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 3, 528–537.
- Hebert, E. B. (2014). Factors affecting parental decision-making regarding interventions for their child with autism. *Focus on Autism and Other Developmental Disabilities*, 29(2), 111–124.
- Matson, J. L., & Williams, L. W. (2015). The curious selection process of treatments for autism spectrum disorders. *Research in Autism Spectrum Disorders*, 9, 21–25.
- Miller, V. A., Schreck, K. A., Mulick, J. A., & Butter, E. (2012). Factors related to parents' choices of treatments for their children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6, 87–95.
- Shepherd, D., Csako, R., Landon, J., Goedeke, S., & Ty, K. (2018). Documenting and understanding parent's intervention choices for their child with ASD. *Journal of Autism and Developmental Disorders*, 48(4), 988–1001.
- Shepherd, D., Goedeke, S., Landon, J., & Mead, J. (2020). The types and functions of social supports used by parents caring for a child with Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders*, <https://doi.org/10.1007/s10803-019-04359-5>. [Epub ahead of print].

Understanding Social Communication Differences in ASD and First-Degree Relatives

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Definition

Social communication deficits are a defining feature of autism spectrum disorder (ASD). Evidence suggests that first-degree relatives of individuals with ASD also demonstrate differences in social communication that mirror those observed in ASD in quality but are subclinical in expression and not associated with impairment. Differences in attention and social processing may underlie the social communication impairments in ASD and more subtle differences observed in relatives. Understanding the roots of social communication phenotypes among clinically unaffected first-degree relatives of individuals with ASD may be a particularly powerful way to understand the

cognitive, neurobiological, and genetic contributors to core symptoms observed in ASD. This entry focuses on allocation of visual attention as a possible contributing mechanism to social communication profiles in individuals with ASD and their parents.

Historical Background

When first formally describing autism in 1943, child psychiatrist Leo Kanner observed that the parents of individuals with autism spectrum disorder (ASD) often exhibited subtle personality traits, such as social reticence, that mirrored in quality certain features of autism. Unfortunately, at that time this observation was misinterpreted as evidence that a detached parenting style was a causal factor in autism. It would be several decades before the genetic heritability of ASD was firmly established by the landmark twin study of Folstein and Rutter (1977), who also demonstrated among co-twins who did not have ASD features that resembled those observed in ASD in quality but that were not as severe in expression. Substantial evidence now exists documenting the strong genetic basis of ASD, and a number of high confidence risk loci have been identified (Chaste et al. 2017; Tick et al. 2016). In spite of strong genetic effects, however, the causes of ASD are heterogeneous, with most cases due to polygenic genetic effects, and less than 1% explained by any specific molecular genetic variant. Studying subclinical traits observed in family members of ASD is one means of reducing etiologic heterogeneity, by identifying heritable traits that can be used to stratify families into more homogenous subgroups. Further, the study of such traits in relatives may also help inform etiologic mechanisms of ASD by helping to decompose the complex ASD phenotype into more fundamental component features with clearer ties to underlying biology and in the absence of clinical comorbidities commonly observed in ASD (Losh et al. 2008; Lowe et al. 2015).

The constellation of subclinical traits observed in relatives has been referred to as the Broad

Autism Phenotype, or BAP. Such traits appear to be more strongly expressed among parents from multiplex families than simplex families, suggesting that they are sensitive indices of genetic liability to ASD (Bernier et al. 2012; Losh et al. 2008; Virkud et al. 2009). Given the ubiquity of social communication deficits in ASD, an ongoing area of research has been to characterize social communication features of the BAP, as well as potential cognitive underpinnings of social communication differences, in individuals with ASD and first-degree relatives. This entry focuses specifically on this line of research and in particular an investigation into concurrent narration and visual attention in individuals with ASD and parents.

Current Knowledge

Social Communication in Autism Spectrum Disorder (ASD)

Social communication differences are universally observed in ASD. Difficulties include difficulties with reciprocity (i.e., building on a conversational partner's statement), overly formal language, off topic remarks, introduction of topics without providing adequate background information, and difficulties with suprasegmental features of speech such as reduced variation of intonation (de Villiers et al. 2007; Diehl and Paul 2012, 2013; Diehl et al. 2009; Lam and Yeung 2012; Klusek et al. 2014). Prior work suggests that individuals with ASD also demonstrate difficulties with narration (i.e., storytelling), with particular difficulty imbuing narratives with a psychological perspective to explain protagonists' actions and motivations (Colle et al. 2008; Lee et al. 2017, 2019; Losh and Capps 2003; Losh and Gordon 2014; Loveland et al. 1990; Loveland and Tunali 1993; Tager-Flusberg and Sullivan 1995). Further, individuals with ASD appear to exhibit the greatest difficulty in communicative contexts that are less-structured, emotionally evocative, and open-ended, with relative strengths in highly structured contexts such as wordless picture books (Losh and Capps 2003; Lee et al. 2019).

Social Communication in First-Degree Relatives of Individuals with ASD

Relatives of individuals with ASD may also demonstrate social-communicative differences. Prior work has demonstrated differences in conversational contexts in parents with ASD that are more subtly expressed but qualitatively similar to those observed in ASD. Differences observed include overly lengthy conversational turns, topic preoccupation, tangential responses, and greater rates of total pragmatic (i.e., social) language violations in semi-naturalistic conversations (Di Michele et al. 2007; Hurley et al. 2007; Landa et al. 1992; Losh et al. 2008, 2012; Ruser et al. 2007). Differences in narrative skill have also been observed in parents (Landa et al. 1991). Landa and colleagues presented parents of individuals with ASD and adults without children with ASD open-ended story prompts. They found that a subgroup of parents of individuals with ASD produced narratives lower in complexity and coherence than those of controls (Landa et al. 1991). This finding is consistent with a number of studies showing that differences are evident only in a subgroup who demonstrate the personality and social-communicative features of the BAP. For instance, parents with the BAP have been shown to exhibit subtle differences in social cognition, including differences in reading emotional states from faces, biological motion, and complex emotional scenes (Adolphs et al. 2008; Losh and Piven 2007; Losh et al. 2009). BAP-specific differences have also been observed in studies of language processing in ASD and first-degree relatives, where parents with the BAP showed reduced language automaticity and differences in eye-voice coordination (a measure of efficient language processing) during rapid automatized naming tasks (Losh et al. 2010; Nayar et al. 2018). Together, these findings suggest that specific language and social phenotypes could reflect genetic liability to ASD and be useful in identifying subgroups of families who may be more similar in underlying etiologic factors.

Origins of Social Communication Differences in ASD and First-Degree Relatives

A critical next step in understanding the social communication differences in ASD and first-

degree relatives is to examine the mechanistic origins of such differences. Evidence suggests that impairments in social cognition are strongly related to the social communication difficulties in ASD, where problems reading emotions and psychological states can seriously limit conversation and narrative skills (e.g., Capps et al. 1998; Losh and Capps 2003; Ochs and Capps 2001; Tager-Flusberg and Sullivan 1995). Differences among first-degree relatives have also been observed in neuropsychological domains that are conceptually related to, and have been hypothesized to underlie, social communication difficulties in ASD. For example, as noted previously, parents of individuals with ASD may also show subtle differences in social cognition, such as reduced ability to identify emotions from eyes or interpret biological motion (Baron-Cohen and Hammer 1997; Losh et al. 2009; Losh and Piven 2007) and also exhibit differences in brain activation during such tasks (Baron-Cohen et al. 2006). Studies have also shown that differences in social cognition are correlated with social communication skills in parents of individuals with ASD (Losh and Piven 2007; Losh et al. 2009), suggesting that differences in social cognition may represent a genetically meaningful feature contributing to the core symptomatology of ASD.

Analysis of eye gaze may reveal mechanistic factors underlying social cognitive and subsequent social communication differences in ASD and among first-degree relatives. A substantial body of research suggests differences in allocation of visual attention in ASD. For instance, individuals with ASD demonstrate difficulty following the gaze of individuals in joint attention paradigms (Leekam et al. 2000) and show reduced fixations to social stimuli, particularly for scenes with multiple individuals (Klin et al. 2002; Rice et al. 2012; Sasson et al. 2008; see Chita-Tegmark 2016, for review), and reduced fixation to the eye region of faces and atypical visual scanning of faces (e.g., Corden et al. 2008, Dalton et al. 2005, Hernandez et al. 2009, Pelphrey et al. 2002, and Rutherford and Towns 2009), particularly when conversing about emotional topics (Hutchins and Brien 2016). Such attentional differences might seriously undercut social-communicative competence, making it difficult,

for instance, to read and track an interlocutor's interests and involvement in unfolding exchange, or focus on topics that are socially and psychologically salient to communication partners. Attentional differences during dynamic social scenes have indeed been found to predict greater language and social-communicative impairment in individuals with ASD (Jones et al. 2008; Klin et al. 2002; Righi et al. 2018; Speer et al. 2007). First-degree relatives of individuals with ASD may also show differences in visual attention, such as reduced attention to socially relevant aspects of videos (Groen et al. 2012), and differences in approaches to processing facial expression in those parents who show features of the BAP (Adolphs et al. 2008). These parallel differences suggest that visual attention may be an important mechanism contributing to the social-communicative phenotype in ASD.

In an attempt to further characterize social-communicative phenotypes and potential contributions of visual attention differences in individuals with ASD and first-degree relatives, Lee et al. (2019) examined narrative quality and patterns of visual attention during narration across two different contexts in individuals with ASD and their parents. The first was a more structured task during which individuals were asked to narrate a wordless picture book one page at a time. In this way, visual attention was captured simultaneously with narration. During the second task, both groups were presented with emotionally evocative, ambiguous images from the Thematic Apperception Test for 8 seconds and then asked to generate a narrative. This methodology allowed for comparison of narrative skills in individuals with ASD and first-degree relatives in the same social-communicative contexts, to further explore the role of context in observed differences and to explore how differences in visual attention might relate to differences in social communication.

Results indicated that individuals with ASD and parents of individuals with ASD demonstrated parallel patterns of performance, in that both groups showed differences in narrative quality from controls during the more open-ended task only. Further, subtle differences in visual attention were observed in both individuals with ASD and parents, with more pronounced differences

observed for those parents with the BAP. Findings indicated context-specific patterns of visual attention differences that were not always in line with those observed in prior work (although no prior studies have examined visual attention during narration). For example, both individuals with ASD and parents with the BAP allocated a greater proportion of visual attention to elements of the setting of a visually complex scene with many background details. However, individuals with ASD and parents with the BAP attended more to faces when telling stories about scenes where facial expressions figured prominently and the emotional content was more ambiguous. It may be the case that because groups were explicitly directed to narrate (including a direct prompt to say what characters were thinking and feeling), increased attention to social stimuli in these ambiguous contexts was needed to interpret characters' emotional states.

Interestingly, relationships between patterns of visual attention and narrative quality were not detected for areas in which individuals with ASD and parents of individuals with ASD differed from controls. For example, in response to the image in which individuals with ASD fixated more intensively on the setting than controls, it was an increased attention to characters' bodies that was related to poorer quality narratives. In other words, results suggested the possibility that individuals with ASD and first-degree relatives not only differ in allocation of visual attention but may also differ in how they make use of visual information to inform the content and quality of their narratives.

Although more work is needed to clarify the direct relationships between visual attention and social communication, findings from this study support earlier work showing that including relatives in studies investigating mechanisms underlying core features of ASD (such as social-communication differences), particularly as revealed through eye tracking, may be a fruitful avenue to identify neurocognitive contributors to social communication in ASD that may also index genetic liability. Findings documenting differences in visual attention also relate to prior findings in first-degree relatives suggesting more fundamental differences in the efficiency of eye

movements that map more directly to specific brain structures, such as the cerebellum. For example, Mosconi et al. (2010) found that first-degree relatives of individuals with ASD demonstrated ocular-motor differences such as minor inaccuracies in direction of eye movement and subtly reduced inhibition of eye movements. Differences in eye-voice coordination have also been observed in ASD and among parents in nonsocial, rapid automatized naming tasks requiring fluid scanning and naming of colors, objects, letters, and numbers (Nayar et al. 2018). Of note, similar differences in eye-voice coordination have been noted in carriers of the *FMR1* premutation (Nayar et al. 2019). Full mutations of the *FMR1* gene cause fragile X syndrome, the most common inherited cause of intellectual disability and also the monogenic disorder most frequently associated with ASD (e.g., Zafeiriou et al. 2013). Findings showing overlap between carriers of the *FMR1* premutation, who do not present with confounding intellectual disabilities associated with FXS, and parents of individuals with ASD, suggest a possible link between a gene known to confer risk to ASD and ocular-motor differences observed in ASD and in relatives. Together, such findings highlight how eye tracking may be applied to identify mechanistic differences, which may map more closely to underlying genetic and neurobiological variation, and potentially lead to downstream differences in the efficiency of allocation of visual attention in social settings and subsequent highly complex social-communicative phenotypes. Overall, identifying overlapping phenotypic patterns in ASD and among parents, integrating assessments of both social communication and allocation of visual attention, may clarify phenotypes that could be used in future studies of key features indexing genetic liability and the basis of the complex social-communicative impairments observed in ASD.

Future Directions

Future research should continue to employ a range of assessment methodologies to characterize social communication in ASD and incorporate

assessments in parents and other biological relatives to help clarify the complex ways in which genetic liability to ASD might express in clinically unaffected family members. Given work suggesting the impact of context on differences observed in both individuals with ASD and first-degree relatives, with greater differences observed in less-structured contexts, the development of more naturalistic assessments will be particularly important in elucidating features of social communication shared across groups. Further, incorporation of multiple methodologies, with additional research on concurrent eye tracking with language production, may shed light on neurocognitive underpinnings of these differences. It will also be important to compare first-degree relatives of individuals with ASD to first-degree relatives of other genetically based disorders impacting language (e.g., specific language impairment; Ruser et al. 2007), where social communication differences have also been observed. Such comparisons may clarify the specificity of findings to ASD as opposed to language-related disorders more generally.

Taken together, characterizing social communication in relationship to eye movement in individuals with ASD and first-degree relatives can help to guide investigations of gene-brain-behavior relationships in ASD. A robust body of work suggests that subtle differences in social communication occur at a greater rate in first-degree relatives of individuals with ASD (and particularly subgroups demonstrating broader features of the BAP) that mirror deficits observed in ASD and that these groups also show parallel differences in underlying neurocognitive factors proposed to contribute to social-communicative differences in ASD. Future research should continue to employ multiple methodologies, such as assessment of visual attention and other proposed neurocognitive contributors to social communication, to characterize this key phenotype in first-degree relatives.

See Also

- [Broader Autism Phenotype](#)
- [Endophenotypes](#)

- Narrative Analysis
- Pragmatic Language
- Social Communication

References and Reading

- Adolphs, R., Spezio, M. L., Parlier, M., & Piven, J. (2008). Distinct face-processing strategies in parents of autistic children. *Current Biology*, 18(14), 1090–1093. <https://doi.org/10.1016/j.cub.2008.06.073>.
- Baron-Cohen, S., & Hammer, J. (1997). Parents of children with Asperger syndrome: What is the cognitive phenotype? *Journal of Cognitive Neuroscience*, 9, 548–554.
- Baron-Cohen, S., Ring, H., Chitnis, X., Wheelwright, S., Gregory, L., Williams, S., Brammer, M., & Bullmore, E. (2006). fMRI of parents of children with Asperger syndrome: A pilot study. *Brain and Cognition*, 61(1), 122–130.
- Bernier, R., Gerdtts, J., Munson, J., Dawson, G., & Estes, A. (2012). Evidence for broader autism phenotype characteristics in parents from multiple-incidence autism families. *Autism Research*, 5(1), 13–20. <https://doi.org/10.1002/aur.226>.
- Bolton, P., Macdonald, H., Pickles, A., Rios, P., Goode, S., Crowson, M., et al. (1994). A case-control family history study of autism. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 35(5), 877–900.
- Capps, L., Kehres, J., & Sigman, M. (1998). Conversational abilities among children with autism and children with developmental delays. *Autism*, 2, 325–344.
- Chaste, P., Roeder, K., & Devlin, B. (2017). The yin and yang of autism genetics: How rare de novo and common variations affect liability. *Annual Review of Genomics and Human Genetics*, 18, 167–187. <https://doi.org/10.1146/annurev-genom-083115-022647>.
- Chita-Tegmark, M. (2016). Social attention in ASD: A review and meta-analysis of eye-tracking studies. *Research in Developmental Disabilities*, 48, 79–93. <https://doi.org/10.1016/j.ridd.2015.10.011>.
- Colle, L., Baron-Cohen, S., Wheelwright, S., & van der Lely, H. K. (2008). Narrative discourse in adults with high functioning autism or Asperger syndrome. *Journal of Autism and Developmental Disorders*, 38, 28–40. <https://doi.org/10.1007/s10803-007-0357-5>.
- Corden, B., Chilvers, R., & Skuse, D. (2008). Avoidance of emotionally arousing stimuli predicts social-perceptual impairment in Asperger's syndrome. *Neuropsychologia*, 46, 137–147. <https://doi.org/10.1016/j.neuropsychologia.2007.08.005>.
- Dalton, K. M., Nacewicz, B. M., Johnstone, T., Schaefer, H. S., Gernsbacher, M. A., Goldsmith, H. H., et al. (2005). Gaze fixation and the neural circuitry of face processing in autism. *Nature Neuroscience*, 8, 519–526. <https://doi.org/10.1038/nn1421>.
- de Villiers, J., Fine, J., Ginsberg, G., Vaccarella, L., & Szatmari, P. (2007). Brief report: A scale for rating conversational impairment in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 37(7), 1375–1380. <https://doi.org/10.1007/s10803-006-0264-1>.
- Di Michele, V., Mazza, M., Cerbo, R., Roncone, R., & Casacchia, M. (2007). Deficits in pragmatic conversation as manifestation of genetic liability in autism. *Clinical Neuropsychiatry: Journal of Treatment Evaluation*, 4(4), 144–151.
- Diehl, J. J., & Paul, R. (2012). Acoustic differences in the imitation of prosodic patterns in children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 6(1), 123–134. <https://doi.org/10.1016/j.rasd.2011.03.012>.
- Diehl, J. J., & Paul, R. (2013). Acoustic and perceptual measurements of prosody production on the profiling elements of prosodic systems in children by children with autism spectrum disorders. *Applied Psycholinguistics*, 34(1), 135–161. <https://doi.org/10.1017/S0142716411000646>.
- Diehl, J. J., Watson, D., Bennetto, L., McDonough, J., & Gunlogson, C. (2009). An acoustic analysis of prosody in high-functioning autism. *Applied Psycholinguistics*, 30(3), 385–404. <https://doi.org/10.1017/S0142716409090201>.
- Folstein, S. E., & Rutter, M. (1977). Infantile autism: A genetic study of 21 twin pairs. *Journal of Child Psychology and Psychiatry*, 18, 297–321.
- Groen, W. B., Rommelse, N., de Wit, T., Zwiers, M. P., van Meerendonck, D., van der Gaag, R. J., et al. (2012). Visual scanning in very young children with autism and their unaffected parents. *Autism Research and Treatment*, 2012, 748467. <https://doi.org/10.1155/2012/748467>.
- Hernandez, N., Metzger, A., Magné, R., Bonnet-Brilhaut, F., Roux, S., Barthelemy, C., et al. (2009). Exploration of core features of a human face by healthy and autistic adults analyzed by visual scanning. *Neuropsychologia*, 47, 1004–1012. <https://doi.org/10.1016/j.neuropsychologia.2008.10.023>.
- Hurley, R. S. E., Losh, M., Parlier, M., Reznick, J. S., & Piven, J. (2007). The broad autism phenotype questionnaire. *Journal of Autism and Developmental Disorders*, 37, 1679–1690.
- Hutchins, T. L., & Brien, A. (2016). Conversational topic moderates social attention in autism spectrum disorder: Talking about emotions is like driving in a snowstorm. *Research in Autism Spectrum Disorders*, 26, 99. <https://doi.org/10.1016/j.rasd.2016.03.006>.
- Jones, W., Carr, K., & Klin, A. (2008). Absence of preferential looking to the eyes of approaching adults predicts level of social disability in 2-year-old toddlers with autism spectrum disorder. *Archives of General Psychiatry*, 65(8), 946–954. <https://doi.org/10.1001/archpsyc.65.8.946>.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809–816.
- Klusek, J., Losh, M., & Martin, G. E. (2014). A comparison of pragmatic language in boys with autism and fragile X syndrome. *Journal of Speech*,

- Language, and Hearing Research*, 57(5), 1692–1707. <https://doi.org/10.1111/jcpp.12267>.
- Lam, Y. G., & Yeung, S. S. S. (2012). Towards a convergent account of pragmatic language deficits in children with high-functioning autism: Depicting the phenotype using the Pragmatic Rating Scale. *Research in Autism Spectrum Disorders*, 6(2), 792–797. <https://doi.org/10.1016/j.rasd.2011.08.004>.
- Landa, R., Folstein, S. E., & Isaacs, C. (1991). Spontaneous narrative-discourse performance of parents of autistic individuals. *Journal of Speech and Hearing Research*, 34(6), 1339–1345.
- Landa, R., Piven, J., Wzorek, M. M., Gayle, J. O., Chase, G. A., & Folstein, S. E. (1992). Social language use in parents of autistic individuals. *Psychological Medicine*, 22(1), 245–254.
- Lee, M., Martin, G. E., Hogan, A., Hano, D., Gordon, P. C., & Losh, M. (2017). What's the story? A computational analysis of narrative competence in autism. *Autism*. <https://doi.org/10.1177/1362361316677957>.
- Lee, M., Nayar, K., Maltman, N., Hamburger, D., Martin, G. E., Gordon, P. C., & Losh, M. (2019). Understanding social communication differences in autism spectrum disorder and first-degree relatives: A study of looking and speaking. *Journal of Autism and Developmental Disorders*. epub ahead of print. <https://doi.org/10.1007/s10803-019-03969-3>.
- Leekam, S. R., Lopez, B., & Moore, C. (2000). Attention and joint attention in preschool children with autism. *Developmental Psychology*, 36(2), 261–273. <https://doi.org/10.1037/0012-1649.36.2.261>.
- Losh, M., & Capps, L. (2003). Narrative ability in high-functioning children with autism or Asperger's syndrome. *Journal of Autism and Developmental Disorders*, 33(3), 239–251.
- Losh, M., & Gordon, P. C. (2014). Quantifying narrative ability in autism spectrum disorder: A computational linguistic analysis of narrative coherence. *Journal of Autism and Developmental Disorders*, 44(12), 3016–3025. <https://doi.org/10.1007/s10803-014-2158-y>.
- Losh, M., & Piven, J. (2007). Social-cognition and the broad autism phenotype: Identifying genetically meaningful phenotypes. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 48(1). <https://doi.org/10.1111/j.1469-7610.2006.01594.x>.
- Losh, M., Childress, D., Lam, K., & Piven, J. (2008). Defining key features of the broad autism phenotype: A comparison across parents of multiple- and single-incidence autism families. *American Journal of Medical Genetics. Part B: Neuropsychiatric Genetics*, 147B(4). <https://doi.org/10.1002/ajmg.b.30612>.
- Losh, M., Adolphs, R., Poe, M. D., Couture, S. M., Penn, D. L., Baranek, G. T., & Piven, J. (2009). Neuropsychological profile of autism and the broad autism phenotype. *Archives of General Psychiatry*, 66(5), 518–526.
- Losh, M., Klusek, J., Martin, G. E., Sideris, J., Parlier, M., & Piven, J. (2012). Defining genetically meaningful language and personality traits in relatives of individuals with fragile X syndrome and relatives of individuals with autism. *American Journal of Medical Genetics. Part B: Neuropsychiatric Genetics*, 159B(6), 660–668. <https://doi.org/10.1002/ajmg.b.32070>.
- Loveland, K. A., & Tunali, B. (1993). Narrative language in autism and the theory of mind hypothesis: A wider perspective. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds.), *Understanding other minds: Perspectives from autism*. Oxford, UK: Oxford University Press.
- Loveland, K. A., McEvoy, R. E., & Tunali, B. (1990). Narrative story telling in autism and Down's syndrome. *British Journal of Developmental Psychology*, 8, 9–23.
- Lowe, J. K., Werling, D. M., Constantino, J. N., Cantor, R. M., & Geschwind, D. H. (2015). Social responsiveness, an autism endophenotype: Genomewide significant linkage to two regions on chromosome 8. *American Journal of Psychiatry*, 172, 266–275. <https://doi.org/10.1176/appi.ajp.2014.14050576>.
- Mosconi, M. W., Kay, M., D'Cruz, A., et al. (2010). Neurobehavioral abnormalities in first-degree relatives of individuals with autism. *Archives of General Psychiatry*, 67(8), 830–840. <https://doi.org/10.1001/archgenpsychiatry.2010.87>.
- Nayar, K., Gordon, P. C., Martin, G. E., Hogan, A. L., La Valle, C., McKinney, W., Lee, M., Norton, E. S., & Losh, M. (2018). Links between looking and speaking in autism and first-degree relatives: Insights into the expression of genetic liability to autism. *Molecular Autism*, 9(1), 51.
- Nayar, K., McKinney, W., Hogan, A. L., Martin, G. E., La Valle, C., Sharp, K., Berry-Kravis, E., Norton, E. S., Gordon, P. C., & Losh, M. (2019). Language processing skills linked to FMR1 variation: A study of gaze-language coordination during rapid automatized naming among women with the FMR1 premutation. *PloS one*, 14(7).
- Ochs, E., & Capps, L. (2001). *Living narrative: Creating lives in everyday storytelling*. Cambridge, MA: Harvard University Press.
- Pelphrey, K. A., Sasson, N. J., Reznick, J. S., Paul, G., Goldman, B. D., & Piven, J. (2002). Visual scanning of faces in autism. *Journal of Autism and Developmental Disorders*, 32, 249–261. <https://doi.org/10.1023/A:1016374617369>.
- Piven, J., Wzorek, M., Landa, R., Lainhart, J., Bolton, P., Chase, G. A., et al. (1994). Personality characteristics of the parents of autistic individuals. *Psychological Medicine*, 24(3), 783–795.
- Piven, J., Palmer, P., Landa, R., Santangelo, S., Jacobi, D., & Childress, D. (1997). Personality and language characteristics in parents from multiple-incidence autism families. *American Journal of Medical Genetics. Part B: Neuropsychiatric Genetics*, 74(4), 398–411.
- Rice, K., Moriuchi, J. M., Jones, W., & Klin, A. (2012). Parsing heterogeneity in autism spectrum disorders: Visual scanning of dynamic social scenes in school-aged children. *Journal of the American Academy of*

- Child and Adolescent Psychiatry*, 51(3), 238–248. <https://doi.org/10.1016/j.jaac.2011.12.017>.
- Righi, G., Tenenbaum, E. J., McCormick, C., Blossom, M., Amso, D., & Sheinkopf, S. J. (2018). Sensitivity to audio-visual synchrony and its relation to language abilities in children with and without ASD. *Autism Research*. <https://doi.org/10.1002/aur.1918>.
- Ruser, T. F., Arin, D., Dowd, M., Putnam, S., Winklosky, B., Rosen-Sheidley, B., et al. (2007). Communicative competence in parents of children with autism and parents of children with specific language impairment. *Journal of Autism and Developmental Disorders*, 37(7). <https://doi.org/10.1007/s10803-006-0274-z>.
- Rutherford, M. D., & Towns, A. M. (2009). Scan path differences and similarities during emotion perception in those with and without autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38, 1371–1381. <https://doi.org/10.1007/s10803-007-0525-7>.
- Sasson, N. J., Turner-Brown, L. M., Holtzclaw, T. N., Lam, K. S. L., & Bodfish, J. W. (2008). Children with autism demonstrate circumscribed attention during passive viewing of complex social and nonsocial picture arrays. *Autism Research*, 1(1), 31–42. <https://doi.org/10.1002/aur.4>.
- Speer, L. L., Cook, A. E., McMahon, W. M., & Clark, E. (2007). Face processing in children with autism: Effects of stimulus contents and type. *Autism*, 11(3), 265–277. <https://doi.org/10.1177/1362361307076925>.
- Spezio, M. L., Adolphs, R., Hurley, R. S., & Piven, J. (2007). Abnormal use of facial information in high-functioning autism. *Journal of Autism and Developmental Disorders*, 37(5), 929–939. <https://doi.org/10.1007/s10803-006-0232-9>.
- Tager-Flusberg, H., & Sullivan, K. (1995). Attributing mental states to story characters: A comparison of narratives produced by autistic and mentally retarded individuals. *Applied PsychoLinguistics*, 16(3), 241–256.
- Tick, B., Bolton, P., Happ, F., Rutter, M., & Rijdsdijk, F. (2016). Heritability of autism spectrum disorders: A meta-analysis of twin studies. *Journal of Child Psychology and Psychiatry*, 57, 585–595.
- Virkud, Y. V., Todd, R. D., Abbacchi, A. M., Zhang, Y., & Constantino, J. N. (2009). Familial aggregation of quantitative autistic traits in multiplex versus simplex autism. *American Journal of Medical Genetics. Part B: Neuropsychiatric Genetics*, 150B(3), 328–334. <https://doi.org/10.1002/ajmg.b.30810>.
- Zafeiriou, D. I., Ververi, A., Dafoulis, V., Kalyva, E., & Vargiami, E. (2013). Autism spectrum disorders: The quest for genetic syndromes. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 162B(4), 327–366.

Uniform Transfers to Minors

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Definition

The Uniform Transfers to Minors Act, the successor to the Uniform Gifts to Minors Act, is a uniform law that has been adopted in 48 of 50 states, with two states enacting substantially similar statutes. The Uniform Transfers to Minors Act allows for parents and other donors to transfer property or assets to minors. Because a minor does not have the legal capacity to manage property, the property can be transferred to a trust for the benefit of a minor. This accomplishes the donor's goal of having the property benefit the minor, but this setup still has oversight by a trustee who does have the legal capacity to manage the property and use it for the benefit of the minor. When the minor reaches age 21, the property in the trust is distributed to the minor.

One benefit of this kind of a transfer of property is that the donor, usually a parent or grandparent, can appoint the custodian of the property. The custodian is the person appointed to hold the property for the benefit of the minor. To create a custodianship, the donor can simply fill out a form available at a bank or other financial institution. By doing this, the donor can ensure that he or she is deciding who is keeping the property for the benefit of the minor. Under Section 14(a) of the Uniform Transfers to Minors Act, a custodian can use the property for the benefit of the minor without a court order when the custodian deems it advisable. Under Section 12 of the Uniform Transfers to Minors Act, the custodian can invest and manage the property as long as they are doing so in accordance with the “prudent person” standard.

The other main benefit to using a trust under the Uniform Transfers to Minors Act is the significant tax benefit. If property is given to a minor in this form, the gift of property will qualify as a present interest and therefore be eligible for the federal annual gift tax exclusion. In 2010, the

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annual exclusion allowed for a person to give away \$13,000 to another person without paying any gift tax as long as it was a gift of a present interest. According to the Internal Revenue Service (“IRS”), a present interest is the “unrestricted right to the immediate use, possession, or enjoyment of property or the income from such property” (Treas. Reg. § 25.2503(b)). Under the Internal Revenue Code, for a gift to be a gift of a present interest to a minor and eligible for the annual gift tax exclusion, it must meet three requirements that can be written into the terms of a trust under the Uniform Transfers to Minors Act. These provisions allow property to be used for the minor before age 6. If the property is not used before age 21, the statute mandates that it be distributed to the minor at age 21. If the minor dies before reaching age 21, the property passes through the minor’s estate or as appointed by the minor. Finally, the statute prohibits any substantial restriction on the trustee’s discretion to use the income from the trust (I.R.C. § 2503(c) and Treas. Reg. § 25.2503-4(b)).

The Uniform Transfers to Minors Acts enables a family to make gifts to a minor in a structured and cost-effective manner. Not only will the family be able to ensure that the minor will be taken supported, but they will also be able to take advantage of significant tax benefits by using this structure.

References and Reading

- Beyer, G. W. (2007). *Wills, trusts, and estates*. New York: Aspen.
- Dukeminier, J., Johanson, S. M., Lindgren, J., & Sitkoff, R. H. (2005). *Wills, trusts, and estates*. New York: Aspen.
- I.R.C. § 2503(b) (2012).
- I.R.C. § 2503(c) (2012).
- Treas. Reg. § 25.2503-3(b) (2012).
- Treas. Reg. § 25.2503-4(b) (1985).
- Uniform Transfers to Minors Act § 12 (2012).
- Uniform Transfers to Minors Act § 14(a) (2012).

Unipolar Depression

- [Mood Disorders](#)

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- [Diphenhydramine](#)

Unisom[®] SleepMelts[™] [OTC]

- [Diphenhydramine](#)

Unit of Response

- [Target Behavior](#)

United Kingdom (UK)

- [Autism Family Experience Questionnaire \(AFEQ\)](#)

Upright/Inverted Figures

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Synonyms

[Facial inversion effect](#); [Inversion effect](#)

Definition

The use of upright/inverted figures is a research paradigm in which an image typically expected to be in one orientation (i.e., the “upright” orientation) is shown upside down (i.e., the “inverted” orientation) in order to disrupt typical visual expectations. The discrepancy between performance on a marker task, such as recognition (e.g., “who is shown in this picture?”), in upright

and inverted conditions is known as the inversion effect. Since the low-level perceptual differences between an upright and corresponding inverted image are minimal (e.g., both would be equally bright, contain the exact same contrast profiles, the same colors, etc.), the inversion effect is assumed to be associated with removing the upright image from its original context, that is, shattering configural processing or holistic orientation (Rakover and Cahlon 2001). One class of commonly used upright/inverted figures is human faces. When faces are shown upright, they are easily recognized and classified. However, when faces are shown upside down, identification becomes more labored and processing more difficult. This phenomenon is the face inversion effect, and often seen in older typically developing children and adults (Gauthier et al. 2010). Children and adults with autism have been shown to exhibit the face inversion effect to a lesser degree than controls, and the results of many studies have suggested that individuals with autism process faces in a featural or piecemeal manner (i.e., focused on specific individual features) rather than in the configural or holistic manner employed by typical peers (Bowler 2007). Faces are not the only class of stimuli that exhibit atypical inversion effects in individuals with autism. For example, the preference for stimuli such as point-light displays of people walking shown side by side, one upright, and one inverted, is heavily skewed toward upright walking in typically developing individuals but not in individuals with autism (Kaiser and Shiffrar 2009).

See Also

- Enhanced Perceptual Functioning
- Face Perception
- Face Recognition
- Weak Central Coherence

References and Reading

Bowler, D. M. (2007). *Autism spectrum disorders: Psychological theory and research*. Chichester: John Wiley and Sons.

Gauthier, I., Tarr, M. J., & Bub, D. (2010). *Perceptual expertise: Bridging brain and behavior*. Oxford: Oxford University Press.

Kaiser, M. D., & Shiffrar, M. (2009). The visual perception of motion by observers with autism spectrum disorders: A review and synthesis. *Psychonomic Bulletin & Review*, 16(5), 761–777. <https://doi.org/10.3758/PBR.16.5.761>.

Rakover, S. S., & Cahlon, B. (2001). *Face recognition: Cognitive and computational processes*. Amsterdam: John Benjamins.

Uruguay and Autism

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Background and History

The diagnostic concept of autism was introduced in Uruguay by Professor Luis E. Prego Silva in 1951, after returning from an internship as a child and adolescent psychiatrist at the Harriet Lane Clinic of the John Hopkins Medical School in Baltimore, Maryland. At that time, Professor Prego created a multidisciplinary team aiming at conducting research and treatment for children with autism, in the Department of Child Psychiatry of the Pedro Visca Hospital (Main Pediatric hospital of the time) (Cherro and Trenchi 1997).

However, the influence of psychoanalytic thought in the River Plate area determined an important delay in the expansion of the understanding of autism as a neurodevelopmental disorder. While treatment modalities advanced in other regions of the world, in Uruguay, they were heavily influenced by the psychoanalytic theory. During many decades, intervention was

centered in unstructured programs based on psychoanalytic psychotherapy.

During the 1990s, with the stimulus of Professor Miguel Cherro Aguerre, the incorporation of DSM III-R, and then, DSM IV to the educational programs of Child and Adolescent Psychiatrists first, and then, to other pediatric specialties, allowed the progressive unification of diagnostic criteria. It was at that time that the first parental advocacy group began. The publication of the book *Autism, Revising Concepts* by Professor Prego Silva (1999) gave place to the integration of different theoretical approaches and therapeutic practices.

In 1987 the first public educational center for autism and infantile psychosis was created. This institution was meant to provide education to those children with severe autism. During a decade, students of Child and Adolescent Psychiatry carried out compulsory internships in the above-mentioned center, where they participated in a multidisciplinary team. Regretfully, there was no further advance and no other public centers were created.

In 2003, a meeting that highlighted the difficulties and needs from the diagnostic, therapeutic, preventive, and juridical stand-point was carried out, with the support of the PAHO, the National Minor Institute and an international table for Childhood with special needs (MINCAD 2003). In the national psychiatry field, consensus was reached to incorporate DSM IV and CIE 10 and abandon the use of other nosographies (Garrido and Viola 2006).

In the year 2005, responding to the increase in the need of improving early detection, diagnosis, and treatment of children with Autism Spectrum Disorders (ASD), a specialized outpatient clinic for diagnosis and treatment of children and adolescents with ASD was created at the Centro Hospitalario Pereira Rossell (main public pediatric hospital). As part of the National Childhood Health Program developed by the Ministry of Health, early signs for ASD and other developmental disorders were incorporated to the child's health record (tool of universal and compulsory use within Uruguay) (Programa Nacional de Salud de la Niñez-MSP 2007). Afterwards, in the framework of the Integrated National Health

System (SNIS, from its initials in Spanish), systematic developmental surveillance was established, in the first level of attention and a National Guideline for developmental surveillance for children aged 0–5 was elaborated (Programa Nacional de Salud de la Niñez-MSP 2010). These instruments, together with a greater awareness about Autism in society as a whole, contributed to earlier diagnosis.

Parent advocacy groups grew and expanded, with the creation of some treatment centers in the capital city (i.e., Montevideo) as well as in some of the provinces. In the year 2010, the Uruguayan Autism Federation (FAU, from its initials in Spanish) was created. The Federation, which gathers most of parental associations of persons with ASD, obtained participation in state organisms which implement and control social policies that address disability (National disability commission, group for the educational improvement of persons with autism from the National Administration of Public Education, etc.).

Legal Issues, Mandates for Service

In 1989, there was an unsuccessful attempt to enact the protection of people with disabilities (Poder Legislativo ROU 1989). It was only many years later, in 2010, that a law concerning rights of persons with disabilities was enacted: Law 18.651 for the Integral Protection of persons with Disabilities (Poder Legislativo ROU 2010). The aforementioned law considered the ratification of Uruguay of the Convention on the rights of the Child (UN 1989) and the Convention on the Rights of Persons with Disabilities (UN 2006).

A direct benefit that resulted from Law 18.651 is that many children, adolescents, and adults diagnosed with ASD with severe disability were contemplated within the Personal Assistants Program, in which personal assistants are financed by the Uruguayan state. Articles 35° to 37° of this law established the state's responsibility in the implementation of strategies to support and contribute to early detection of the disability, in providing timely attention, and the mandatory declaration of persons with disabling conditions, regardless of their age. The greatest contribution

has been the Protocol for Educational Inclusion of Persons with Disabilities in educational centers (Decreto N° 72/017), since in Uruguay there was a considerable delay concerning guarantees for inclusive education for children and adolescents with ASD and other disabilities.

Yet, apart from this legislation for the protection of rights of people with disabilities in general, there is still no other legal recognition of Autism in Uruguay. In 2014, as an initiative of parent advocacy groups and some legislators, a bill to create an Integral System of Prevention and Treatment for ASD was presented to the Public Health and Social Assistance Commission. This bill has not yet been considered in Parliament.

Overview of Current Treatments and Centers

Since 2007, the Health System in Uruguay is organized as an integrated system (SNIS) that joins public and private health providers through a National Health fund (Poder Legislativo ROU 2007). The SNIS widened general health coverage of the population, particularly for children and adolescents. Every person with ASD has access to a pediatrician, and to specialists in pediatric neurology and child and adolescent psychiatry. However, the access to treatment and rehabilitation continues to be considerably uneven and partial.

According to an extensive survey for parents of persons with ASD, conducted in six Latin American countries in 2016 (Garrido and García 2017), in Uruguay 64% of patients had received language therapy at some point and 73% psychomotor treatment. These treatments are covered by the state for a maximum of 3 years. Despite strong international evidence, other treatments such as behavioral therapy, cognitive therapy, and occupational therapy are not financed by the Health System.

In Uruguay there is an important network of multidisciplinary centers, which are distributed across the country. They all provide attention from professionals of different areas (language skills, psychomotor skills, psychology, and pedagogy). There are centers which provide comprehensive specific attention for patients with ASD,

organized and managed by parental associations, with partial support from the state and others run by groups of independent professionals. These centers are still scarce and access to them is not universal nor equitable.

Regarding the educational system, there are two public schools specialized in ASD, one in the capital city and another in the province (Salto City), with a very limited capacity. There is a greater number of schools (82) specialized in intellectual disability and distributed throughout the country. Support teachers that come from special schools and cooperate with the inclusion of children with special educational needs are available. However, the number of support teachers remains limited, and there is a considerable gap between the need and offer of this service, added to a heterogeneous distribution throughout the country.

One third of children and adolescents with ASD attend special education centers for intellectual disability, and only 26% of those who attend regular schools receive some kind of additional support in the classroom, according to a survey conducted with caretakers in 2016 (Garrido and García 2017).

Despite of the fact that Uruguay has a greater number of qualified human resources in different disciplines than other countries in the region, it is still essential to continue to go forward in specific qualification and improve accessibility to more intensive evidence-based treatments.

Overview of Research Directions

Research in the autism field is scarce in Uruguay. This is partially due to the obstacles in obtaining financial support and to the fact that clinicians distribute their time between assistance to patients, teaching, and research. Research projects, centered in the University of the Republic (Udelar, for its acronym in Spanish) and in one private university (Uruguayan Catholic University), are in some cases financially supported by the National Agency for Investigation and Innovation (ANII, for its initials in Spanish).

There are no epidemiological studies for ASD in Uruguay. The first estimation of prevalence is

0.9% for Uruguayan children under the age of 4, and was drawn from the data of the First National Survey of Nutrition Child Development and Health (ENDIS) (Cabella and Vigoritto 2016). This survey included the administration of the Child Behavior Checklist (CBCL 1 ½–5 for parents) to determine emotional and behavioral problems in preschoolers. It relied on the use of the withdrawal scale and pervasive developmental disorders scale of the instrument. Specificity and sensibility of both scales had been established previously for Uruguayan preschoolers (Garrido 2015).

The Latin American Autism Network (REAL from Spanish initials) has been created recently. REAL gathers various countries in the region and has enabled cooperative work. In 2016, six of its members (Argentina, Brazil, Chile, Uruguay, Dominican Republic, and Venezuela), with the support of Autism Speaks, carried out a family survey, about the needs, impact, and stigma perceived by caretakers of persons with ASD (Rattazzi 2016). Regarding Uruguay, information concerning diagnosis, treatments, schooling, impact in families, quality of life, and participation in parental groups was gathered from 382 families.

A new collaborative ongoing project, in the framework of REAL, is the adaptation and validation of the Parents Skills Training Program (PST), developed by the WHO and Autism Speaks. This will allow a greater accessibility to early intervention for children with ASD in many Latin American countries.

In the specialized outpatient clinic for ASD at Pereira Rossell Hospital, clinical research about conditions associated with ASD were carried out. These include Sleep Characteristics in Preschoolers with ASD (Tailanián 2015), Early Language and Social Skills Regression (Amigo 2014), and ADHD (Garrido and Ceretta 2013).

Overview of Training

Since 2003, a module concerning ASD has been included in the child psychiatry course for pediatric residents. The latter is compulsory and is

centered in early detection. The implementation of the surveillance guide for children aged 0–5 was accompanied by training on normal development and frequent developmental disorders, for pediatricians and family doctors throughout the country. ASD was specially considered.

In 2013, the Uruguayan Association of Child and Adolescent Psychiatrists carried out an ADOS workshop, where 30 child and adolescent psychiatrists (one third of all child and adolescent psychiatrists in Uruguay) were trained in the administration of the instrument as a complement for clinical diagnosis.

Clinical psychologists, speech language therapists, psychomotor skills therapists, occupational therapists, and educators receive training in different intervention models, but the public offer of specific training is still very limited.

In the framework of an agreement of the Faculty of Medicine (UDELAR), and the University of New México, USA, the project for Extension for Community Healthcare Outcomes (ECHO) has been implemented in Uruguay, under the coordination of Prof. Henry Cohen. The aim of project ECHO is to democratize knowledge and improve the attention to complex and prevalent health problems. Since 2015, the Department of Child and Adolescent Psychiatry of the Faculty of Medicine (Udelar) carries on teleclinic ECHO-ASD with the objective of training first level health professionals in the country from different health providers, in early detection and comprehensive treatment for patients with ASD (Santoro et al. 2015).

The University, through the specialized ASD outpatient clinic of the Pediatric Hospital, has cooperated with the educational sector training resources from early childhood, primary and secondary education by carrying out conferences, workshops, and courses nation-wide.

Social Policy and Current Controversies

In the last years, awareness in the general population, regarding ASD has increased. Among other causes, this is due to the growth of the parental movement and the constitution of the Uruguayan

Autism Federation. As noted earlier, the FAU has obtained participation within programs and organisms that define social policies related to disability. It is important to highlight the existing cooperation between parental associations and many professionals. In this cooperative framework, a variety of initiatives regarding ASD have been carried out, such as support groups, some of the above mentioned research projects, and attention centers. Yet, the access to diagnosis, medical attention, and specialized treatment is not universal nor equitable. It is important to consider that there is still an average gap of 34 months between first parental concerns and diagnosis (Montiel Nava 2017).

Social security in Uruguay contemplates a pension for persons with severe disabilities, the provision of special assistance (personal assistants), in the framework of the National Care System, to those highly dependent individuals, as well as the access to certain treatment modalities.

As mentioned previously, Uruguay has an important network of multidisciplinary centers that provide attention in different areas. Rehabilitation treatments, in the above mentioned centers, are financed by the state, through social security, and given to children with specific developmental disorders and other emotional or behavioral problems. However, even though interdisciplinary treatment is provided to children and adolescents, comprehensive specific treatment for ASD is not fully financed by the state. This kind of treatment is mainly financed by the families of children with ASD. The incorporation of evidence-based treatments to the SNIS is still required. Certain treatments that show little or no evidence but are still used by families continues to be controversial.

Social security In Uruguay also contemplates that persons with severe disabilities receive a pension from the state.

In spite of the earlier noted difficulties and challenges regarding ASD in our country, Uruguay is undergoing a favorable period, and it is in the path of improving early detection, treatment strategies, and social inclusion for persons with ASD. Greater awareness about ASD in the Uruguayan society is notorious, as well as a rising

interest of professionals to become more knowledgeable in the subject.

See Also

- AGESIC – Desarrollando el Uruguay Digital: Carné Nacional de Salud del Niño y de la Niña; <https://www.agesic.gub.uy/innovaportal/v/3079/1/agesic/carne-de-salud-del-nino-y-de-la-nina-electronico.html>
- Convención sobre los Derechos de las Personas con Discapacidad; <http://www.un.org/spanish/disabilities/default.asp?id=497>
- Convención sobre los Derechos del Niño; https://www.unicef.org/uruguay/spanish/CDN_20_boceto_final.pdf
- DSM 5; <https://www.psychiatry.org/psychiatrists/practice/dsm>
- Ministerio de Salud Pública Uruguay – Guía Nacional de vigilancia del Desarrollo; http://www.msp.gub.uy/sites/default/files/archivos_adjuntos/Guia%20vigilancia%20desarrollo%20del%20nino_1.pdf
- Unidad Especializada en Trastornos del Espectro Autista – Hospital Pereira Rossell – Facultad de Medicina UR; <http://www.unidadteauruguay.org/>

References and Reading

- Amigo, C. (2014). *Regresion temprana del lenguaje y habilidades sociales en niños con diagnóstico de trastorno del espectro autista*. Montevideo: UDELAR.
- Cabella, W., & Vigoritto, A. (2016). *Salud, Nutrición y Desarrollo en la Primera Infancia en Uruguay*. Montevideo: Instituto Nacional de Estadística.
- Cherro, M., & Trenchi, N. (1997). International perspectives. Central America and south America. In *Handbook of autism* (2nd ed., pp. 982–988). Hoboken: Wiley.
- Garrido, G. (2015). Jornadas Regionales ASEBA en Montevideo. Presentado en Jornadas Regionales ASEBA en Montevideo. Montevideo.
- Garrido, G., & Ceretta, L. (2013). Trastorno por Déficit Atencional e Hiperactividad en niños preescolares con TEA. Presentado en XIX Congreso Latinoamericano de FLAPIA – Federación Latinoamericana de Psiquiatría de la Infancia, Adolescencia, Familia y Profesiones Afines. Colonia.

- Garrido, G., & García, R. (2017). *Profile of health and educational services received by people with ASD and satisfaction of families in Latin America*. San Francisco: Poster presentado en IMFAR 2017.
- Garrido, G., & Viola, L. (2006). Criterios actuales para la clasificación de los trastornos profundos del desarrollo. *Revista de Psiquiatría de Uruguay*, 70(2), 140–150.
- MINCAD. (2003). Dificultades y necesidades del punto de vista diagnóstico, terapéutico, preventivo y jurídico en el caso de los trastornos del espectro autista infantil. OPS.
- Montiel Nava, C. (2017). *Diferencias entre la fecha de primeras preocupaciones de los padres y la fecha de diagnóstico en TEA*. San Francisco: Presentado en IMFAR.
- Poder Legislativo ROU. (2010). Ley 18.651 Protección Integral de las Personas con Discapacidad.
- Prego Silva, L. E. (1999). *Autismos: Revisando conceptos*. Montevideo: Trilce.
- Programa Nacional de Salud de la Niñez. MSP. (2007). Carné de salud de la niña.
- Programa Nacional de Salud de la Niñez. MSP. (2010). Guía para la Vigilancia del Desarrollo del Niño y de la Niña Menores de 5 años.
- Rattazzi, A. (2016). Implementation of the Latin American autism Spectrum network Cargiver needs survey. Presentado en IMFAR 2016. Baltimore.
- Santoro, A., Garrido, G., Kanopa, V., & Giacheto, G. (2015). Proyecto ECHO: Una estrategia para mejorar la detección y atención de las personas con Autismo. *Opción Médica*, 53, 6–12.
- Tailanián, N. (2015). *Características del sueño en preescolares con Trastorno del Espectro Autista*. Montevideo: UDELAR.

US Social Policy and Autism Spectrum Disorders

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Introduction

Any formal decision made by a publicly mandated organization which is intended to directly or indirectly improve practices addressing the welfare of a population of people with an autism spectrum

disorder or ASD and their legal guardians in the United States.

Inclusion/Exclusion Criteria

Though many individuals and organizations may act in ways to improve welfare of people with ASD, this definition of social policy focuses on a subset of actions and excludes others.

- *Formal decisions* shift the emphasis to practices specified in some written form, as opposed to unwritten, informal, hypothesized practices (e.g., historical trends, institutional culture, and so on). Examples of such formal decisions include laws (and their associated regulations), professional practice standards, and formal position statements as long as these directly address practice.
- The focus on *practices addressing a population* excludes decisions made on a case-by-case basis, and emphasizes practices intended to address a specific population defined a priori, perhaps including a reference to other characteristics (age, presence of intellectual disability, and so on).
- Emphasizing practices affecting *people with ASD* exclude those originally developed to benefit people with related disabilities (such as intellectual disabilities), even those practices that people with ASD might benefit from them.
- Decisions that *indirectly improve practices addressing the welfare* of people with ASD allow the discussion of policy to be extended beyond services and supports provided directly to people with ASD, to actions that may benefit those professionals providing the services, such as training and applied research (see below).
- Decisions made *by an organization* exclude the actions taken by individual professionals in the care of their patients, students, or clients.
- Decisions made by a *publicly mandated organization* focus on practices intended to be universally accessible and exclude policies set by nongovernmental agencies unless the agency has a formal or de facto mandate to establish policy governing a broad population of people

with ASD and the groups of professionals who work with them (for example, the American Academy of Pediatrics).

- Including *legal guardians* acknowledges the primary role played by parents and caregivers, and their own need for support.

A more detailed discussion of social policy pertaining to ASD is available elsewhere (Doehring 2013)

Historical Background

Throughout history, the welfare of people with ASD has been affected by a wide range of social policies as defined above. Though the focus here is on those policies specifically designed for the population of people with ASD, a number of policies for related populations enacted in the 1960s and 1970s in the United States are worthy of note.

Early Legislation

The Rehabilitation Act (1973) was the first act to address the notion of equal access of people with disabilities. The Developmental Disabilities Assistance and Bill of Rights Act (or DD) introduced the term “developmental disability” to describe a range of significant lifelong disabilities with an early onset that included autism and focused on broad systems change, professional development, and legal protections. The passage of the Education for All Handicapped Children Act in 1975, renamed the Individuals with Disabilities Education Act in 1990, was another watershed in the development of school-based services and supports for people with disabilities, including those with ASD. In the field of mental health, the trend towards deinstitutionalization forced the development of new community-based systems of supports for those persons whose psychiatric conditions or developmental disabilities appeared to prohibit living at home as children or living independently as adults. These policies and trends, together with others, were part of a growing awareness and acceptance of disabilities beginning in the 1960s which led

directly to an increased commitment to develop community-based supports. To this day, these policies and trends continue to impact services for a broader population that includes people with ASD.

The Role of Advocates

Most changes in social policy regarding ASD over the past 35 years can be traced to the work of advocacy organizations. These organizations initially sought to raise awareness and, over time, urge the development of specific programs of services, research, and training. The establishment of Autism Society of America in 1965 gave advocates a voice at the state and national level through a network of local chapters and fostered collaboration between caregivers and professionals in formulating policy recommendations based on the presumption that autism is a lifelong developmental disability. Other groups such as Cure Autism Now and National Alliance for Autism Research (later merged into Autism Speaks) sought to define policies and set priorities for autism research often based the characterization of autism as a disease that can be cured or ameliorated. The advocacy organizations initially sought to raise awareness of autism by identifying it as a distinct disability meriting specialized educational and mental health services. State advocacy groups led policy initiatives to create a first wave of autism-specific programs in states such as Delaware, West Virginia, and Indiana in the 1970s and 1980s. These programs provided a range of services at the state level, such as disseminating information, providing training, developing and delivering services, and conducting research (Doehring 2013).

The Role of Research

The contribution of ASD research in the evolution of social policy is more complex (Doehring 2014). Within the broader community, the growth of social policies specifically designed for people with ASD has generally paralleled our understanding of the disability and the refinements in the definition of autism in the DSM. Within the scientific community, awareness grew with research specifically targeting autism and with

the rise of autism-specific periodicals beginning with the establishment of the *Journal of Autism and Childhood Schizophrenia* in 1971. Despite continued advances in the scientific understanding of ASD throughout the 1970s, 1980s, and 1990s, the adoption of new categories and labels did not result in immediate or universal changes in associated social policy. For example, autism emerged as a distinct disability in 1980 in the DSM-III following advances in research, but new state and federal regulation defining autism for the purpose of providing educational services emerged slowly and inconsistently and in many cases still fails to capture all of the defining features of autism. A broad range of studies contributed to the decision to include Asperger Syndrome or AS in the DSM-IV in 1994, but few public policies specifically and explicitly include AS. Part of the lag can probably be attributed to the relative lack of research directly relevant to social policy and inconsistent collaboration between researchers, service providers, policymakers, and advocates (Doehring 2013; Doehring 2014).

In other cases, advocates were able to draw on research findings to press their case for changes in social policy. This is illustrated by a second wave of initiatives intended to increase services for people with ASD. For example, Lovaas' (1987) claims – that almost one-half of preschool children who received early and intensive behavioral intervention or EIBI “recovered” and were not significantly different from typical kindergarteners – were repeatedly cited in costly lawsuits brought by individual parents to education agencies. These lawsuits effectively changed policy in pockets across the country, leading to the creation of autism-specific programs that sometimes provided more intensive services similar to those described by Lovaas. These changes almost always occurred only, however, at the local level – for example, in specific school districts or in county early intervention programs. As a result, services to young children with autism varied widely from one region or town to another, with services less likely to change in areas where advocates lacked organization and resources to lobby for change. Nonetheless, these successes set the

stage for more ambitious reforms led by parent advocates described below.

Research on the prevalence of ASD begun at around the same time also contributed indirectly to the development of related social policy. The first series of research sites in what would become the Autism and Developmental Disorders Monitoring (ADDM) Network was initially funded in 2000 in response to preliminary reports suggesting dramatically increased rates of autism in Brick Township, New Jersey (Peacock et al. 2013). Initial findings suggested markedly higher rates of ASD than previously thought and led some advocates to attribute apparent increases in prevalence to the increased use of the mercury-based preservative Thimerisol in childhood vaccines. This led to a change in national policy, phasing Thimerisol out of almost all vaccines, even though the hypothesized links between vaccine policies and rates of autism were disproven. Continued reports of increased prevalence fuelled public speculation about an “autism epidemic” and subsequently drove many policy initiatives at the local, state, and national level to increase services, training, and research.

Current Knowledge

There are too many potentially pertinent social policies to describe here, and no systematic survey of ASD policies enacted at the local, state, and national level has been conducted (see Doehring 2013 and 2014 for discussion of related issues) in the United States, let alone in other countries. This entry therefore seeks to provide a summary of the more significant social policies undertaken at the state and national level in the last 15 years in the United States, focusing within each area (services, training, research, advocacy on comprehensive reform) on local and regional policies only when state and national policies are absent. Lists of state and national policies can be found on the websites of advocacy organizations such as Autism Society of America (www.autism-society.org) and Autism Speaks (www.autismspeaks.org) and other networks such as the Association of University Centers on Disabilities (www.aucd.org). The National

Conference of State Legislators also maintains a searchable database of all state laws addressing autism (<http://www.ncsl.org/research/health/autism-policy-issues-overview.aspx>).

ASD Information and Awareness

The social policies most commonly adopted are those designed to increase awareness of and disseminate information about ASD and to coordinate efforts to establish other policies. That such policies are most common should not be surprising; these are also the core strategies adopted by most advocacy groups and are critical to building consensus towards more substantial policy. For example, many states have initiated some form of policy intended to gather information about ASD, though such policies can vary in important ways: the initiative can be time-limited (e.g., an ASD task force) or more permanent (a standing committee focused on ASD or a subcommittee of a governor's council on disabilities); they may address ASD more broadly or focus on a specific issue (e.g., ASD screening and diagnosis); they may be limited to the actions of a single government agency or seek to build interdisciplinary or cross-agency collaboration; and the policy may seek or require the involvement of advocates or advocacy groups. The products of such policy can also vary widely: from broad position statements to a published report with specific practice recommendations and from forming an advisory committee to identifying a person within a government agency to be responsible for coordinating information and advocacy to creating a new government agency with specific responsibilities to oversee services, training, research, and/or policy. Many of these steps are evident in the evolution of ASD policy in Pennsylvania: a cross-agency task force that included many advocates generated a report with very specific policy recommendations and that resulted in the creation of a new government bureau with specific responsibilities for overseeing services, training, research, and policy development within in a state department (Wexler et al. 2013).

Other efforts have been undertaken at the federal level. Many federal departments have convened workgroups or committees to gather

information and develop policy. For example, the Interagency Autism Coordinating Committee within the US Department of Health and Human Services (iacc.hhs.gov), first authorized under the Children's Health Act of 2000 and then reauthorized and broadened under the Combating Autism Act of 2006, produces detailed reports intended to inform and coordinate federal agency policies. The Learn the Signs Act Early (LTSAE) initiative undertaken by the Centers for Disease Control (<http://www.cdc.gov/ncbddd/actearly/index.html>) is perhaps the most coordinated federal effort to date. Focusing on early identification of ASD and related disabilities, LTSAE has led to the development of detailed publications for professionals and the general public, and facilitated the development of state-level teams to disseminate training and information, and to engage the full range of stakeholders in the creation of state plans (Peacock et al. 2013). In many cases, such federal efforts solicit the involvement of non-governmental organizations through grants and contracts: for example, the state summits convened through LTSAE were coordinated through a contract by the Centers for Disease Control and Prevention with the Association of University Centers on Disabilities, while the National Autism Resource and Information Center was created through a grant to The Arc of the United States (www.autismnow.org).

ASD Services

With the exception of initiatives related to health insurance (see below), the majority of social policies intended to directly increase services have been undertaken not at the state or national level, but at the local level (Doehring 2013). These policies include decisions made by schools and early intervention programs regarding the amount of services provided to children with ASD, the emphasis on particular programs and approaches such as applied behavior analysis, and the creation of ASD-specific positions or intervention programs. Such policies are adopted haphazardly across the United States, sometimes through the efforts of advocates, and other times through the decision of an individual school district or county leader. Given the costs associated with increased

and improved services, and the seminal role of parent advocates, it is not surprising that such policy changes are more likely to have been enacted in more affluent suburban regions. While a number of state-level initiatives targeting training to, consultation with, and oversight of service providers have been undertaken (see below) few state-level programs directly increase services, except perhaps for comprehensive centers established in West Virginia (Becker-Cottrill 2013) and Delaware (Doeiring and Winterling 2013), and other programs focussed on specific statewide needs (Carbone et al. 2013).

Current social policies with the greatest likelihood of directly increasing the amount and availability of services are those related to insurance reform, although the impact is likely to vary significantly from state to state. With the support of organizations such as Autism Speaks, advocacy groups have successfully lobbied for the inclusion of mandated benefits related to the provision of applied behavior analysis in insurance regulations, beginning with Indiana in 2001 (at the time of writing, 34 states had enacted such reforms). While the exact legislation varies from state to state, it can include provisions for more than \$30 000 of direct services and supports in the home and community annually, but is often restricted to a subset of health insurance plans, may exclude adults, does not address education-based services, depends on the availability of qualified providers, and may require that parents navigate often-confusing insurance procedures. Advocates lobbied for inclusion of similar benefits in the Affordable Care Act (ACA), passed in 2010 to extend health care benefits to all Americans. In addition to extending services important to people with ASD and traditionally included in health care (prescription drug coverage and so on), ACA includes ASD screening at 18 and 24 months as well as “habilitative” services and “behavioral health care” as required benefits in any insurance sold within the ACA “marketplace.” At the time of writing, the impact of these changes depends on state-by-state interpretation of key statutes of the law and its associated regulations.

ASD Training

In contrast to ASD services, a number of policies have been enacted at the state and national level to address training. The National Professional Development Center on Autism Spectrum Disorders was a consortium of university-based centers, funded by the Office of Special Education Programs (OSEP) of the US Department of Education, to build training capacity state by state within the public school system (Cox et al. 2013). A number of states have undertaken training and consultation initiatives targeting educators (Stickle and Hoffmeier 2013; Pratt 2013; Wexler et al. 2013). The network of programs in Leadership Education in Neurodevelopmental and related Disorders (LEND) added autism-specific training to its curriculum targeting a broad range of graduate-level trainees in health and education. At the time of writing, the network included 43 LEND programs across 37 states and was supported through grants provided through the Maternal Child Health Bureau.

Other policy initiatives seek to establish general standards for training and certification. For example, a set of national standards for teachers of children with autism was developed by the Network of Autism Training and Technical Assistance Programs (an affiliate of Autism Society of America) in 2009, and subsequently endorsed by National Council for Accreditation of Teacher Education (NCATE) and the Council for Exceptional Children. Many states have adopted Autism Teacher endorsements that formally recognize a postgraduate program of specialized training, while states such as Delaware require that all public school teachers of children with autism complete a postgraduate certificate from an accredited program. Many states have also moved to formally recognize Board Certified Behavior Analysts (BCBAs) by adopting the licensure requirement of the Behavior Analysis Certification Board (BACB). Autism Insurance regulations adopted in some states have stipulated that only behavior analysis services supervised by a BCBA are eligible for reimbursement, even though training in autism is not required for BCBAs.

ASD Practice Standards

Other social policies with important implications for ASD training and services center on formal endorsement of written standards for specific assessment and intervention practices, enacted by professional and governmental entities. National organizations of physicians and their subspecialties have endorsed a variety of policy or consensus statements regarding specific practices, including the identification and management of autism (Johnson et al. 2007; Myers et al. 2007), the identification and management of gastrointestinal disorders in people with ASD (Buie et al. 2010), and recommending against the use of auditory integration training and facilitated communication (American Academy of Pediatrics 1998)

Other federally funded initiatives have sought to establish standards of practice, although the relevance to social policy cannot be clearly established because the path to implementation is often unclear. For example, the National Research Council reviewed the available research and developed a broad set of recommendations regarding the education of children with ASD (Committee on Educational Interventions for Children with Autism 2001). The aforementioned National Professional Development Center on Autism Spectrum Disorders (NPDC) conducted systematic reviews of the outcome research to establish a list of evidence based practices (EBP) (Cox et al. 2013) had constituted the backbone of the NPDC's program of training. While the work of both groups has been widely cited, these reviewers know of no public entities responsible for delivering services that made a formal commitment to require that their services and training rely on these recommendations.

ASD Research

A wide range of national research initiatives related to ASD have been undertaken, although many – if not the vast majority – of these did not contribute directly and immediately to improved practices, and so their relevance to social policy is unclear. For example, different federally funded autism programs – the Collaborative Programs for

Excellence in Autism initiated in 1997, and the Studies to Advance Autism Research and Treatment initiated in 2000 – were merged and expanded in the Autism Centers of Excellence Programs through the Combating Autism Act. The vast majority of projects have focused, however, on the causes, characteristics, and developmental trajectory of ASD, as opposed to validating and promoting the widespread adoption of effective treatments. The Autism Treatment Networks originally established by Autism Speaks, and then repackaged as the Autism Intervention Research Network on Physical Health (AIR-P) with support from the Health Resources Services Agency in 2007, has sought to develop recommendations and toolkits for practitioners. The AIR-P may represent the public program with the greatest potential to improve community-based practice, but the impact has ultimately been limited by the lack of any formal endorsement of these recommendations by a public or professional entity providing services. The reports of the ADDM network, that continue to refine estimates of prevalence, also continue to have perhaps the greatest impact of policy.

Individual states have also established other research programs intended to gather data needed to inform public policy. For example, a number of states have passed legislation to establish autism registries to, among other goals, identify the number of people in the state with ASD and who require services. It is unclear how such registries have influenced policy decisions, especially given the availability of more rigorous assessments of prevalence available through the ADDM network. Pennsylvania's efforts represent perhaps the most coordinated effort to conduct applied research to inform state policy. A census of all Pennsylvanians receiving health or education services under an ASD label revealed significant county-to-county variability in rates of actual identification or administrative prevalence, as opposed to prevalence projections derived from CDC estimates (Lawer and Mandell 2009). These discrepancies, together with a needs assessment that generated a series of reports about access to and satisfaction with services, now guide state

initiatives to develop and improve services (Wexler et al. 2013).

Comprehensive Reform

The majority of policies are enacted in relative isolation from one another, addressing a specific element (e.g., services or training or research or further policy development) perhaps within a specific domain (health or education or community services). Occasionally, policies are enacted that intended to foment more comprehensive reforms. For example, the Combating Autism Act (CAA) signed into law in 2006 (PL 109-416) and authorizing almost \$1 billion in additional spending on autism represented the largest and most comprehensive legislation focused on autism to date. CAA expanded a wide range of programs through a variety of federal agencies: research and coordination at the National Institutes of Health (NIH), ASD awareness and surveillance activities at the Centers for Disease Control and Prevention (CDC), and training of health professionals within the Health Resources Services Administration to screen, diagnose (or rule out), and provide ASD interventions. CAA also promoted the development of programs to inform the public about ASD (including the National Autism Resource and Information Center), and redefined and enhanced the mandate of the Interagency Autism Coordinating Committee.

Future Directions

Social policy with respect to ASD is constantly evolving, with increased understanding of the needs of the population, changes in government leaders and structures, and shifting societal values.

DSM-V

While the DSM-V allows a DSM-IV diagnosis of ASD to be carried forward, it is unclear whether all individuals previously eligible for a diagnosis of Asperger syndrome or PDD-NOS under the DSM-IV would merit a DSM-V diagnosis of ASD. This may require changes to social policies to accommodate the needs of individuals rendered

ineligible for specialized services and supports under this revised nomenclature.

Improving and Increasing Services at the Local Level

Despite decades of research and advocacy, huge gaps persist between social policy at the national level and day-to-day practice at the local level. National programs like LEND, Learn the Signs Act Early, and the National Professional Development Center demonstrate innovations in raising awareness and training professionals, but the amount, accessibility, and quality of services still vary tremendously from one community to another, with traditionally underserved populations being especially vulnerable. Though some advocates and policymakers have sought to prioritize more comprehensive reforms in services, there is currently little chance for new federal programs addressing the services and supports needs of those on the spectrum, given the current national focus on reducing the deficit and the size of the federal government. Regardless, any substantial changes to services at the local level would have to be carefully coordinated with changes at the state and national level in training and policy, and guided by research focused on implementation. (Doehring 2013)

The Identity of the ASD Community

It also remains unclear whether the autism community can or should speak with a single voice. Perhaps some philosophical differences are inevitable given the breadth of the autism spectrum; some persons with ASD require intensive, life-long services and supports associated with their developmental disabilities; and others who are able to live independently seek recognition and respect for the ways they are different. These differences often translate into competing priorities, with some seeking more research on prevention or intensive treatment and others focused more on civil rights and services and supports for adults. Despite efforts to differentiate ASD from other disabilities, individuals with ASD and intellectual disability or ID may in fact have more in common with those whose ID uncomplicated by ASD, than those with ASD but not ID (Doehring 2013). This may indicate potential

benefits from an alliance with the community of those with developmental disabilities. Interest and attention has also shifted over the past 5–10 years to encompass the needs and services required for adults with ASD, as the children identified in the “epidemic” beginning in the 1990s grow into adults who can no longer benefit from educational entitlements.

See Also

- [American Academy of Pediatrics](#)
- [Autism Society](#)
- [Autism Speaks](#)
- [Delaware Autism Program](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [National Research Council](#)
- [Studies to Advance Autism Research and Treatment](#)

References and Reading

- American Academy of Pediatrics. (1998). Auditory integration training and facilitated communication for autism. *Pediatrics*, 102, 431–433.
- Becker-Cottrill, B. (2013). West Virginia Autism Training Center: Statewide programs for behavior support, paraprofessional training, college students with Asperger syndrome, and the autism spectrum disorders registry. In P. Doehring (Ed.), *Autism services across America: A roadmap for improving state and national education, research, and training programs*. Baltimore: Paul H. Brookes.
- Buie, T., Campbell, D. B., Fuchs, G. J., III, Furuta, G. T., Levy, J., Vandewater, J., et al. (2010). Evaluation, diagnosis, and treatment of gastrointestinal disorders in individuals with ASDs: A consensus report. *Pediatrics*, 125(Suppl 1), S1–18.
- Carbone, P. S., Winter, S., Randall, H., & Holt, J. (2013). Utah: Promoting early detection and community-based support through medical homes and satellite clinics. In P. Doehring (Ed.), *Autism services across America: Road maps for improving state and national education, research, and training programs*. Baltimore: Paul H. Brookes.
- Committee on Educational Interventions for Children with Autism, (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Cox, A., Brock, M., Odom, S., Rogers, S., Sullivan, L., Tuchman-Ginsberg, E., et al. (2013). National Professional Development Center on ASD: An emerging national educational strategy. In P. Doehring (Ed.), *Autism services across America: Road maps for improving state and national education, research, and training programs*. Baltimore: Paul H. Brookes.
- Doehring, P. (2013). *Autism services across America: Road maps for improving state and national education, research, and training programs*. Baltimore: Paul H. Brookes.
- Doehring, P. (2014). Translating research into effective social policy. In F. Volkmar, R. Paul, S. Rogers, & K. Pelphrey (Eds.), *Handbook of Autism and Pervasive Developmental Disorders: volume 2, assessment, interventions, and policy* (4th ed.). Hoboken: Wiley.
- Doehring, P., & Winterling, V. (2013). Delaware Autism Program: Statewide educational services in the public schools. In P. Doehring (Ed.), *Autism services across America: Road maps for improving state and national education, research, and training programs*. Baltimore: Paul H. Brookes.
- Johnson, C. P., Myers, S. M., & The Council on Children with Disabilities. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120, 1183–1215.
- Lawer, L., & Mandell, D. S. (2009). *Pennsylvania autism census project: Final report*. Harrisburg: Bureau of Autism Services, Pennsylvania Department of Public Welfare.
- Lovaas, I. O. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting & Clinical Psychology*, 55, 3–9.
- Myers, S. M., Johnson, C. P., & The Council on Children With Disabilities. (2007). Management of children with autism spectrum disorders. *Pediatrics*, 120, 1162–1182.
- Peacock, G., Lin, S. C., Bogin, J., Rhodes, C., & Wolf, R. (2013). Learn the Signs. Act early: The public health approach to the early identification of children at risk for autism spectrum disorders and other developmental disabilities. In P. Doehring (Ed.), *Autism services across America: Road maps for improving state and national education, research, and training programs*. Baltimore: Paul H. Brookes.
- Pratt, C. (2013). Indiana Resource Center for Autism: Promoting increased capacity statewide through training, information, and policy. In P. Doehring (Ed.), *Autism services across America: Road maps for improving state and national education, research, and training programs*. Baltimore: Paul H. Brookes.
- Stickle, L., & Hoffmeier, S. (2013). The Kansas Instructional Support Network: Developing collaborative teams for support of students with complex needs and for ASD identification. In P. Doehring (Ed.), *Autism services across America: Road maps for improving state and national education, research, and training programs*. Baltimore: Paul H. Brookes.
- Wexler, E., Scutta, C., Miklos, M., Wall-Cote, N., & Doehring, P. (2013). The Pennsylvania Bureau of Autism Services and the Department of Education: Improving educational and community services across the commonwealth. In P. Doehring (Ed.), *Autism services across America: Road maps for improving state and national education, research, and training programs*. Baltimore: Paul H. Brookes.

USA and Autism

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Historical Background

There is a long and distinguished (and complex) history of work on research, clinical care, and training on autism and related disorders in the USA. Of course, the starting point for this discussion is the land breaking description, in 1943, of autism by Leo Kanner. His remarkably enduring, phenomenologically based description of autism highlighted key vulnerabilities in the area of social interaction and unusual environmental responsiveness and became the conceptual basis for all subsequent definitions. At the same time, it also serves to mislead research in some ways, e.g., the use of the term autism suggested a possible conduit with schizophrenia, and his observation of high levels of parental educational attainment also suggested, to some, a possible contribution of experience to psychopathology. During the 1950s in particular, the emphasis on “blaming” parents (particularly mothers) took a decidedly unfortunate turn, and only in the 1960s and particularly in the 1970s did scientific opinion begin to shift as the strong brain and genetic bases of autism began to be appreciated. The early work of the psychologist and parent, Bernard Rimland, was key in this effort (Rimland 1964) as was the work in the 1970s suggesting the validity of autism (Kolvin 1971) and its strong genetic and neurological basis (Folstein and Rutter 1977; Ornitz and Ritvo 1968) and the importance of structured treatment programs (Bartak and Rutter 1973). The pioneering work on the neurochemistry of autism (specifically on serotonin) by Daniel X. Freedman and colleagues was also important (Hanley et al. 1977) as was the early work on pharmacological treatment (Campbell et al. 1971). Early confusion about the

distinctiveness of autism complicated much of the early work, but a growing appreciation of its importance and validity as a condition stimulated a marked increase in research further facilitated by the official recognition of infantile autism in 1980 in the third edition of the Diagnostic and Statistical Manual (DSM-III, APA 1980).

Several other events important in the history of autism program occurred in the 1960s with the organization of the National Autism Society in the USA (parallel to the organization of a corresponding institution in the UK). This group, founded in 1965, began to take a more active role in advocacy and support for both clinical working and training as well as research (NSAC 1978). Reorganized as the Autism Society of America, this group was involved in establishing legal mandates for educational services and for legislation at the state and national levels. In addition to the ASA, a number of other parent-founded foundations and advocacy groups have become active including the National Alliance for Autism Research (NAAR), Cure Autism Now (CAN), Autism Speaks, and Autism Science Foundation. Particularly during the 1990s, a critical body of work began to be available, and federal initiatives to support research began to increase. Notable programs included the Centers of Excellence for Programs in Autism (CPEA) led through the National Institute of Child Health and Human Development by then Director Dwayne Alexander and program officer Marie Bristol-Power were succeeded by support from multiple NIH institutes. These included the Studies to Advance Autism Research and Treatment (STAART) and Autism Center of Excellence Programs. The impact of these initiatives both on research and well-trained investigators has been enormous.

The recognition of autism as a brain-based disorder with a need for structured treatment programs inspired a large body of work on behavioral and special educational interventions (Ferster 1972; Lovaas 1987). Unfortunately, schools often did not wish to provide services or felt unable to do so, and, as a result, parents were frequently involved in developing their own school programs, many of which continue to exist today – sometimes with changes in mission

and orientation over the years. This situation began to dramatically change with the advent of Public Law 94-142 and, along with other advances, has been associated with a significant change in long-term outcome (Howlin 2013).

Many of the earliest programs developed for autism have evolved over the years – often moving from center-based to more home- and community-based programs (see National Research Council 2001). The advent of programs combining behavioral and developmental approaches to treatment is particularly exciting as is the development of regional and statewide programs like Division TEACCH in North Carolina under the leadership of Eric Schopler and Gary Mesibov.

Another important historical development includes founding of the first journal devoted to autism in 1971 – the *Journal of Autism and Developmental Disorders*. Originally called the *Journal of Autism and Childhood Schizophrenia*, Leo Kanner served as the first editor. Over the years, Kanner was succeeded by Eric Schopler and Michael Rutter, then by Gary Mesibov, and most recently by Fred Volkmar. A number of other journals with varying degrees of focus on autism have now appeared, and the advent of an increasing body of peer-reviewed research has undoubtedly contributed to the significant increase in research in the field. A final important area to note has been the development of models of training for individuals working as teachers and professionals in the field. This work has aimed to increase the quality and quantity of teachers, professionals, and others knowledgeable about autism and its treatment (Volkmar et al. 1988).

Legal Issues, Mandates for Service

As noted, the passage, in 1975, of Public Law 94-142, now codified as IDEA (Individuals with Disabilities Education Act), was a watershed event in helping establish eligibility of children for school-based services. The intention of this act was to ensure that states and school districts ensure access to a free appropriate public education (FAPE) to children with disabilities of various

types, all children with disabilities (Mandlawitz 2005). Both follow-up legislation and various state and, particularly federal, judicial decisions have clarified important aspects of this mandate. It is important to note that this law differs in important respects from another statute, the Americans with Disabilities Act of 1990, a law concerned with protecting individuals with discrimination based on a disability. While this act has some relevance to children, it becomes particularly important as students transition from high school to vocational and college programs where access to service is not an automatic right (although reasonable accommodation for a disability is) (Wolff et al. 2009).

Overview of Current Treatments and Centers

The range and diversity of treatment programs and centers has increased dramatically in the past decade. As noted above, a range of programs have been developed, and a number now have substantial empirical research support with many now regarded as evidence based (Reichow et al. 2011). Over the past decade, there has been a growing awareness of the importance of peer exposure and mainstreaming in the acquisition of social skills (Tobin et al. 2012). At the same time, there has been recognition that, in some cases, more intensive behavioral and educational supports must be provided – sometimes in non-mainstream settings. Preparation of teachers and support personnel in schools has also been recognized as increasingly important (Wolery et al. 1993). As noted above, a range of models of intervention are now available and include both strongly behaviorally based programs, those that have a more developmental orientation, others that might best be described as eclectic, and, finally, some that combine aspects of multiple approaches. As with other treatments in psychology and medicine, initial results of intervention programs sometimes are the most positive and are tempered by subsequent experience as programs move from university-based clinical settings to more “real world” applications.

A recurrent tension in treatment research has been the numerous interventions that technically fall in the no established category – these include complementary and alternative treatments (Volkmar and Wiesner 2009). In some areas of intervention research, there has not been a strong tradition of carefully controlled research so information is limited (but sometimes growing). In other areas, research is minimal but treatments are promising, and in yet other instances, treatments are said to be incapable of empirical proof – this latter category is of greatest concern particularly when a child is removed from treatments with some research bare and/or enrolled in programs that entail some actual risk of direct harm to the child.

Overview of Research Directions

Most significant research programs within the country function within the context of college/university settings with a strong commitment to research and training. While there are some important obstacles to research (particularly treatment research), there has been a major expansion over the past decade with hundreds of peer-reviewed scientific papers appearing each year. Advances have been made in multiple areas including brain development and understanding of the social brain in autism (Insel and Fernald 2004; Dawson et al. 2005) of basic genetic mechanisms (State and Levitt 2011) and potential brain effects/correlates of treatment (Voos et al. 2012). Research on potential pharmacological interventions has also significantly advanced and, recently, potential interventions that might impact social disability. The multisite study of risperidone, now approved as an effective intervention for agitation and irritability in autism, is an excellent example of such work (McCracken et al. 2002). At the same time, of course, sometimes initially promising results are not replicated in subsequent studies. The dearth of intervention research, particularly focused on adults, remains a major area of concern. The support of research by both the government and by a range of foundations (including Autism Speaks, Autism Science

Foundation, and Simons Foundation) remains critically important.

Social Policy and Training

Many advances in the on-going training of providers (teachers, health-care professionals, and others) remain a top national priority. As noted above, efforts have been made to develop and implement curricula to enhance effective teaching methods in schools and classrooms (Harris et al. 2005). New approaches to training are now underway and include Internet-based services, publicly available materials, and privately produced materials. Establishing the evidence base of proposed treatments remains a priority as does the critical need for work on older individuals with autism.

See Also

- ▶ [Autism Science Foundation](#)
- ▶ [Autism Society](#)
- ▶ [Autism Speaks](#)
- ▶ [DSM-III](#)
- ▶ [ICD 10 Research Diagnostic Guidelines](#)
- ▶ [Kanner, Leo](#)

References and Reading

- American Psychiatric Association. (1980). *Diagnostic and statistical manual*. Washington, DC: APA Press.
- Bartak, L., & Rutter, M. (1973). Special educational treatment of autistic children: A comparative study. 1. Design of study and characteristics of units. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, 14(3), 161–179.
- Campbell, M., Fish, B., Shapiro, T., & Floyd, A., Jr. (1971). Imipramine in preschool autistic and schizophrenic children. *Journal of Autism & Childhood Schizophrenia*, 1(3), 267–282.
- Dawson, G., Webb, S. J., Wijsman, E., Schellenberg, G., Estes, A., Munson, J., & Faja, S. (2005). Neurocognitive and electrophysiological evidence of altered face processing in parents of children with autism: Implications for a model of abnormal development of social brain circuitry in autism. *Development & Psychopathology*, 17(3), 679–697.

- Ferster, C. B. (1972). Clinical reinforcement. *Seminars in Psychiatry*, 4(2), 101–111.
- Folstein, S., & Rutter, M. (1977). Genetic influences and infantile autism. *Nature*, 265(5596), 726–728.
- Hanley, H. G., Stahl, S. M., & Freedman, D. X. (1977). Hyperserotonemia and amine metabolites in autistic and retarded children. *Archives of General Psychiatry*, 34(5), 521–531.
- Howlin, P. (2013). Outcomes in adults with autism spectrum disorders. In F. Volkmar, S. Rogers, R. Paul, & K. Pelphrey (Eds.), *Handbook of autism* (4th ed.). Hoboken: Wiley.
- Insel, T. R., & Fernald, R. D. (2004). How the brain processes social information: Searching for the social brain. *Annual Review of Neuroscience*, 27, 697–722, Annual Reviews, US.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kolvin, I. (1971). Studies in the childhood psychoses. I. Diagnostic criteria and classification. *British Journal of Psychiatry*, 118(545), 381–384.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting & Clinical Psychology*, 55(1), 3–9.
- Mandlawitz, M. R. (2005). Educating children with autism: Current legal issues. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 1161–1173). Hoboken: Wiley.
- McCracken, J. T., McGough, J., Shah, B., Cronin, P., Hong, D., Aman, M. G., et al. (2002). Risperidone in children with autism and serious behavioral problems. *New England Journal of Medicine*, 347(5), 314–321.
- National Research Council. (2001). *Educating young children with autism*. Washington, DC: National Academy Press.
- NSAC. (1978). National society for autistic children definition of the syndrome of autism. *Journal of Autism & Childhood Schizophrenia*, 8(2), 162–169.
- Ornitz, E. M., & Ritvo, E. R. (1968). Perceptual inconsistency in early infantile autism. The syndrome of early infant autism and its variants including certain cases of childhood schizophrenia. *Archives of General Psychiatry*, 18(1), 76–98.
- Reichow, B., Doehring, P., Cicchetti, D. V., & Volkmar, F. R. (2011). Evidence-based practices and treatments for children with autism. In *Evidence-based practices and treatments for children with autism*. New York: Springer Science/Business Media; US: xvi, 408.
- Rimland, B. (1964). *Infantile autism: The syndrome and its implications for a neural theory of behavior*. New York: Appleton.
- Tobin, H., Staunton, S., Mandy, W., Skuse, D., Hellriegel, J., Baykaner, O., Anderson, S., & Murin, M. (2012). A qualitative examination of parental experiences of the transition to mainstream secondary school for children with an autism spectrum disorder. *Educational and Child Psychology*, 29(1), 75–85.
- Volkmar, F., & Wiesner, L. (2009). *A practical guide to autism*. Hoboken: Wiley.
- Volkmar, F. R., Moss, N. E., & Lettich, A. (1988). Undergraduate education about autism. *Journal of Autism & Developmental Disorders*, 18(4), 695–696.
- Wolery, M., Brookfield, J., Huffman, K., Schroeder, C., et al. (1993). Preparation in preschool mainstreaming as reported by general early education faculty. *Journal of Early Intervention*, 17(3), 298–308.
- Wolff, L. E., Brown, J. T., & Bork, G. R. (2009). *Students with Asperger syndrome: A guide for college personnel*. Shawnee Mission: APC Publishing.

Use of Language

► Pragmatics

Use of Language in Context

► Pragmatics

Using Shaping to Increase Foods Consumed by Children with Autism

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Definition

Shaping is an instructional approach that involves increasing the probability of a response through the gradual transformation of a current level of responding due to the successive approximation of a targeted class of behaviors. Shaping procedures involve the consideration of the current relevant repertoire, terminal goals, systematic approaches to the terminal goal, and maintenance (Goldiamond 1974). In the treatment of food

selectivity, shaping can be used to increase the variety or amount of food consumed. This is accomplished by providing reinforcement for the target level of food acceptance determined by considering both the present level of food acceptance and the terminal goal for acceptance and providing reinforcement contingent upon the display of the target behavior. The level of food acceptance is increased over time until the child meets the terminal goal.

Historical Background

Historically, shaping has been demonstrated to be effective both across and within response topographies. For example, Isaacs et al. (1960) shaped the eye movements of an individual diagnosed with schizophrenia to lip movements, speech sounds, and ultimately recognizable words. Wolf et al. (1964) reinforced successive approximations (i.e., touching eye glasses, picking up eye glasses, putting glasses up to face) to the end goal of wearing eye glasses in a preschooler who was in danger of losing his sight if he did not wear his glasses. Rea and Williams (2002) used shaping to increase the duration of an individual's breath holding prior to measuring exhaled carbon monoxide levels during smoking cessation treatment.

Current Knowledge

Interventions used to increase the acceptance of new foods with children with autism commonly include one or more of the following components based on learning theory: escape extinction, differential reinforcement of alternative behavior (DRA), noncontingent reinforcement, and some form of systematic exposure or antecedent intervention (Chawner et al. 2019; Silbaugh et al. 2016). Multicomponent treatment packages that include escape extinction with DRA and demand fading demonstrate effectiveness in treating food selectivity across multiple settings and with parents or therapists as implementers (Russo et al. 2019; Najdowski et al. 2010; Najdowski et al. 2003; Silbaugh et al. 2016). For example, Russo

et al. (2019) increased the variety of foods consumed across all food groups for two adolescent males with autism by providing antecedent choice for both the preferred and nonpreferred target food, implementing escape extinction of inappropriate mealtime behavior and systematically increasing the number of bites and variety of food presented in order to meet criterion for reinforcement. However, given that DRA is indicated as a preferred treatment by caregivers to treat food selectivity compared to escape extinction (Vazquez et al. 2019), it is not surprising that systematic exposure techniques such as demand fading and shaping with DRA are evaluated more often than escape extinction to increase the variety of foods consumed in children with ASD (Chawner et al. 2019). Effective shaping interventions that do not include escape extinction generally include a multistep hierarchy of graduated exposure that the child moves up as he or she meets criteria and moves down as a result of consecutive errors or engaging in refusal behaviors. Response requirements in hierarchical exposure vary from beginning with tolerating the food in the same room (Tanner and Andreone 2015) to touching the target food with the hands or lips (Hodges et al. 2017; Penrod et al. 2012) and ultimately conclude with swallowing or consuming the food. Tanner and Andreone (2015) evaluated a 12-step hierarchy with a child with food selectivity and ASD. The hierarchy gradually increased the demands over a span of 9 months from tolerating target foods in the same room to consuming the target foods and resulted in acceptance of 50 previously refused foods.

Luiselli et al. (2005) increased the amount of milk a young girl with autism drank by gradually increasing the amount she was required to drink and providing reinforcement contingent upon consumption of the liquid. Koegal et al. (2012) increased food variety in three children with autism who had selective diets by using a systematic hierarchical sequence (i.e., seven-level hierarchy) and provided individualized reinforcement upon the child's mastery of required steps. This resulted in successful expansion of food repertoire among all three participants. Hodges et al. (2017) used differential reinforcement and shaping to

increase the variety of foods accepted by children with autism who demonstrated significant feeding inflexibility. They were introduced to four new food items via hierarchical exposure, which involved systematically increasing the desired response with the food item. Following the intervention, all participants accepted the targeted foods.

Future Directions

More research is needed to identify interventions with long-term therapeutic effects, particularly for increasing variety of foods accepted (Bandini et al. 2017; Chawner et al. 2019; Koegal et al. 2012). Long-term follow-up measurements of observed treatment outcomes may help better identify treatments best suited for long-term generalized therapeutic effects. Furthermore, some individuals may continue to exhibit feeding challenges throughout the lifespan. Indeed, in one longitudinal study of children with ASD, while food refusal improved over time, there were no increases in the number of foods accepted 7 years after the initial measurement point (Bandini et al. 2017). However, in a recent systematic review, only half of the included articles ($n = 36$) included follow-up measures (Chawner et al. 2019). Without replicated demonstrations of long-term results, confidence in interventions assumed to be efficacious is weakened (Chawner et al. 2019).

There is also a need for greater evaluation of the social validity of commonly used feeding intervention components. In particular, caregiver ratings of acceptability of interventions used are necessary, because caregivers are the most common natural implementers of mealtime intervention strategies outside of treatment sessions (Vazquez et al. 2019). It is also possible that more intrusive interventions may be viewed as more acceptable if maladaptive behaviors are more severe (Vazquez et al. 2019).

Finally, because of the prevalence of multi-component interventions, future researchers may evaluate individual strategies that are effective through component analyses (Chawner et al. 2019; Najdowski et al. 2010). Component

analyses may help in identifying mechanisms of behavior change, thus streamlining the iterative intervention process (Najdowski et al. 2010). Alternatively, component analyses that may also illuminate multicomponent interventions are necessary because of the highly complex nature of feeding behaviors (Chawner et al. 2019).

References and Reading

- Bandini, L. G., Curtin, C., Phillips, S., Anderson, S. E., Maslin, M., & Must, A. (2017). Changes in food selectivity in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 47(2), 439–446.
- Chawner, L. R., Blundell-Birtill, P., & Hetherington, M. M. (2019). Interventions for increasing acceptance of new foods among children and adults with developmental disorders: A systematic review. *Journal of Autism and Developmental Disorders*, 49, 3504–3525.
- Goldiamond, I. (1974). Toward a constructional approach to social problems: Ethical and constitutional issues raised by applied behavior analysis. *Behaviorism*, 2(1), 1–84.
- Hodges, A., Davis, T., Crandall, M., Phipps, L., & Weston, R. (2017). Using shaping to increase foods consumed by children with autism. *Journal of Autism and Developmental Disorders*, 47, 2471–2479.
- Isaacs, W., Thomas, J., & Goldiamond, I. (1960). Application of operant conditioning to reinstate verbal behavior in psychotics. *Journal of Speech and Hearing Disorders*, 25, 8–12.
- Koegal, R., Bharoocha, A., Ribnick, C., Ribnick, R., Bucio, M., Fredeen, R., & Koegal, L. (2012). Using individualized reinforcers and hierarchical exposure to increase food flexibility in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42, 1574–1581.
- Luiselli, J. K., Ricciardi, J. N., & Gilligan, K. (2005). Liquid fading to establish milk consumption by a child with autism. *Behavioral Interventions*, 20, 155–162.
- Najdowski, A. C., Wallace, M. D., Reagon, K., Penrod, B., Higbee, T. S., & Tarbox, J. (2010). Utilizing a home-based parent training approach in the treatment of food selectivity. *Behavioral Interventions*, 25, 89–107.
- Najdowski, A. D., Wallace, M. D., Doney, J., & Ghezzi, P. M. (2003). Parental assessment and treatment of food selectivity in natural settings. *Journal of Applied Behavior Analysis*, 36, 383–386.
- Penrod, B., Gardella, L., & Fernand, J. (2012). An evaluation of a progressive high-probability instructional sequence combined with low-probability demand fading in the treatment of food selectivity. *Journal of Applied Behavior Analysis*, 45, 527–537.
- Rea, J., & Williams, D. (2002). Shaping exhale durations for breath CO detection for med with mild mental

- retardation. *Journal of Applied Behavior Analysis*, 35, 415–418.
- Russo, S. R., Croner, J., Smith, S., Chirinos, M., & Weiss, M. J. (2019). A further refinement of procedures addressing food selectivity. *Behavioral Interventions*, 34(4), 1–9.
- Silbaugh, B., Penrod, B., Whelan, C., Hernandez, D., Wingate, H., Falcomata, T., & Lang, R. (2016). A systematic synthesis of behavioral interventions for food selectivity of children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 3, 345–357.
- Tanner, A., & Andreone, B. E. (2015). Using graduated exposure and differential reinforcement to increase food repertoire in a child with autism. *Behavior Analysis in Practice*, 8(2), 223–240.
- Vazquez, M., Fryling, M., & Hernandez, A. (2019). Assessment of parental acceptability and preference for behavioral interventions for feeding problems. *Behavior Modification*, 43, 273–287.
- Wolf, M. M., Risley, T. R., & Mees, H. L. (1964). Application of operant conditioning procedures to improve behaviour problems of an autistic child. *Behavior Research and Therapy*, 1, 305–312.

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2007-2015
Emeritus Professor of Cognitive Development,
Institute of Cognitive Neuroscience, Univer-
sity College London, 2006-present.

Major Honors and Awards

- Prix Jean Nicod, jointly with Chris Frith, 2014
William James Fellow Award, Association for
Psychological Science, 2013
Honorary Fellow British Science Association,
2013
Honorary Dame Commander of the Order of the
British Empire., 2012
Honorary Degree Cambridge University, 2012
Foreign Associate of the National Academy of
Sciences, Section 52 - Psychology and Cogni-
tive Sciences, 2012
Centre for Cognitive Science University of Turin:
Mind and Brain Prize 2010
Experimental Psychology Society 38th Sir
Frederic Bartlett Lecture 6th January 2010
European Latsis Prize, jointly with Chris Frith,
2009
British Psychological Society, Research Board
Life Time Achievement Award, 2009
Elected Member of the German Academy of Sci-
ences Leopoldina, 2008
Honorary Fellow Newnham College Cambridge,
2008
UKRC Women of Outstanding Achievement in
SET, 2008
Foreign Member of the Royal Society of Arts and
Sciences in Göteborg, 2008
Lifetime Achievement Award, International
Society for Autism Research (INSAR), 2007
Samuel T. Orton Award International Dyslexia
Association, 2007
Elected Honorary Fellow of the British Psycho-
logical Society, 2006
President of the Experimental Psychology
Society, 2006-08

Fellow of the Royal Society, 2005-07-09
 Burghölzli Award, University of Zuerich,
 Department of Psychiatry
 Robert Sommer Award, University of Giessen,
 Medical School
 Laurea Honoris Causa, Palermo University, 2004
 Jean-Louis Signoret Prize of the Ipsen Founda-
 tion, 2003
 Fellow of the British Academy, 2001
 Fellow of the Academy of Medical Sciences,
 2001
 Honorary Doctorate, University of Göteborg,
 Sweden, 1998
 Member of the Academia Europaea, 1992
 The President's Award of the British Psychologi-
 cal Society for distinguished contributions to
 psychological knowledge, 1990

Landmark Clinical, Scientific, and Professional Contributions

Having joined her PhD supervisors Neil O'Connor and Beate Hermelin in pioneering the cognitive experimental study of autism, Uta Frith's first landmark contribution was the influential "Theory of Mind" deficit account, developed and tested with fellow CDU scientist Alan Leslie and their then-PhD student, Simon Baron-Cohen. Their empirical paper showing that most children with autism failed a simple false belief test (Baron-Cohen et al. 1985) has since been cited more than 2,500 times (ISI WoS) and launched decades of research into "mentalizing." A decade later, Uta and Chris Frith were the first to use functional neuroimaging to investigate the brain basis of typical and atypical mentalizing, work for which they later received the prestigious Jean Nicod Prize.

Frith's scholarly yet accessible 1989 book, *Autism: Explaining the Enigma*, explained mentalizing deficits in autism to a wider audience and also introduced the concept of weak "central coherence", reduced global and increased local processing, revealed in superior performance on, e.g., Block design, and embedded figures tests, as shown in Frith's studies with her PhD student Amita Shah. Frith can be credited with being the

first scientist to suggest that more can be learnt about autism by studying assets than by studying deficits. In 2010 she edited a book *Autism and Talent* with past PhD student and long-time collaborator, Francesca Happé.

Frith has been a major advocate for cognitive theories of autism (and dyslexia), and her work on Causal Modelling, with John Morton, explained the importance of distinguishing levels of explanation: biological, cognitive (which includes affective), and behavioral – with environmental interplay possible at each level.

Her other notable contributions to autism science include pioneering work on education and the brain (on which she published a book, *The Learning Brain*, with Sarah-Jayne Blakemore in 2005), and autism in history (including her book on the historical case of Hugh Blair, with Rab Houston in 2000). Frith also provided the first English translation of Hans Asperger's 1944 paper, in her influential 1991 edited book, *Asperger Syndrome*

Uta Frith is also well known as a supporter of women in science, having mentored and championed many female scientists, and founded the group "Science and Shopping." She is often in the media and has presented several science programs including the BBC's Horizon.

Short Biography

Born Uta Aurnhammer in 1941 in Rockenhausen, Uta Frith grew up in Kaiserlautern, Germany. It was not until 1964 that she came to England, to the Institute of Psychiatry (IoP) in London, where she met her future husband and longtime collaborator, Chris Frith, and trained as a clinical psychologist. Originally planning a PhD on OCD, she became fascinated by autism and was delighted to find the authors of a key experimental paper she was reading were on the IoP faculty. Neil O'Connor and Beate Hermelin saw her ability at once and took her on first as a PhD student and then, even before her thesis was submitted, as a research scientist in the newly established MRC Developmental Psychology Unit. Uta remained an MRC staff scientist for more than 35 years,

helping to choose the other faculty at the subsequent MRC Cognitive Development Unit. It was at the CDU that the “Theory of Mind” deficit account of autism was conceived and tested. At the same time, Uta was at the forefront of cognitive studies of dyslexia – and she has maintained her fascination with both types of neurodiversity throughout her career.

When the CDU closed in 1998, Uta became Deputy Director of the new Institute of Cognitive Neuroscience at UCL – which neighbors the Wellcome Trust Functional Imaging Laboratory in Queen’s Square, where with Chris Frith and other colleagues she conducted the first functional neuroimaging studies of Theory of Mind. She remains an emeritus professor at UCL and is currently working on book projects with Chris, including a graphic novel about social cognition. Uta is active in public understanding of science through her media work and is a champion for gender equality in science; she is currently chair of the Royal Society’s Diversity Committee.

Apart from a vast array of landmark papers, and six books, Uta is notable for her legacy in terms of nurturing the next generations of scientists: her wide and long-standing collaborations, her generous support for students and junior colleagues, and the large and growing group of researchers who are proud to count themselves as the academic children or grandchildren of Uta Frith.

See Also

- [Beate Hermelin](#)
- [Theory of Mind](#)
- [Weak Central Coherence](#)

References and Reading

- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a “theory of mind”? *Cognition*, 21, 37–46.
- Blakemore, S. J., & Frith, U. (2005). *The learning brain: Lessons for education*. Oxford: Blackwell publishing.
- Fletcher, P. C., Happe, F., Frith, U., Baker, S. C., Dolan, R. J., Frackowiak, R. S., & Frith, C. D. (1995). Other minds in the brain: a functional imaging

- study of “theory of mind” in story comprehension. *Cognition*, 57(2), 109–128.
- Frith, U. (1989). *Autism: Explaining the enigma* (Vol. 1989). Oxford: Blackwell Scientific Publications.
- Frith, U. (Ed.). (1991). *Autism and Asperger syndrome*. Cambridge: Cambridge University Press.
- Frith, U. (2008). *Autism: A very short introduction* (Vol. 195). Oxford: Oxford University Press.
- Frith, C. D., & Frith, U. (2012). Mechanisms of social cognition. *Annual Review of Psychology*, 63, 287–313.
- Frith, U. E., & Hill, E. E. (2003). *Autism: Mind and brain*. Oxford: Oxford University Press.
- Happé, F., & Frith, U. (2006). The weak coherence account: detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36(1), 5–25.
- Happé, F., & Frith, U. (2010). Autism and talent (Vol. 364, No. 1522). Oxford: Oxford University Press.
- Happe, F., Ehlers, S., Fletcher, P., Frith, U., Johansson, M., Gillberg, C., et al. (1996). ‘Theory of mind’ in the brain. Evidence from a PET scan study of Asperger syndrome. *Neuroreport*, 8(1), 197–201.
- Houston, R., & Frith, U. (2000). Autism in history. The case of Hugh Blair of Borgue. Oxford: Blackwell.
- Senju, A., Southgate, V., White, S., & Frith, U. (2009). Mindblind eyes: An absence of spontaneous theory of mind in Asperger syndrome. *Science*, 325(5942), 883–885.
- Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry*, 34(8), 1351–1364.

Utilitarian Attention

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Definition

The term “utilitarian attention” is intended to describe a style displayed by persons with autism spectrum disorders (ASD) across various components of attentional processing. The term utilitarian, based on the root word utility, implies a functional significance as would be the case when attention occurs in ways and is guided by considerations that lead to its most efficient use.

The implementation of the utilitarianism may be manifested in different ways such as the unique processing of stimuli and information that imparts meaning or guidance in a specific context or for a specific task, and by behavior that is driven by data or task requirements rather than general heuristics or information in the environment that may be of increased interest or relevance to others but incidental to the task at hand.

This notion of utilitarian attention represents an extension of earlier empirically based strength-oriented conceptualizations in the areas of visual search (O’Riordan et al. 2001), perception (O’Riordan and Plaisted 2001; Plaisted et al. 1998; Russo et al. 2012), and the iterations of the enhanced perceptual functioning model (Mottron and Burack 2001; Mottron et al. 2006), all of which stand in contrast to the more common emphasis on deficits, including in the area of attention. Thus, the essential premise of the notion of utilitarian attention is that attention is not inherently deficient among persons with ASD and may even be more efficient and effective in certain cases. Utilitarian attention is also consistent with the contemporary notion that the identification of alternative styles, biases, and ways of being is essential to understanding attentional functioning among both persons with ASD (Burack et al. 2016) and the more general population (Burack et al. 2017; Ristic and Enns 2015). In this context, the emphasis on utilitarian attention is not simply about assessing whether attentional abilities are better or worse than that which is evident among comparison groups of appropriately matched persons from the neurotypical population, but rather is meant to highlight styles, biases, and ways of attending that are unique to persons with ASD and the ways that they affect the efficiency of attention. This framework is consistent with the recently posed question about the attentional processing of persons with ASD, “How I attend - not how well do I attend” (Burack et al. 2016).

Historical Background

The term “utilitarian attention” was initially used by Brodeur et al. (2018) in the title and the interpretation of the findings of a study of visual

attention in which the participants (children) with ASD seemed to benefit from the presence of nontarget stimuli in the visual field that appeared to impede the performance of a comparison group of mental age matched typically developing participants (children). It was intended to be understood within the context of the key components of both classic and contemporary conceptualizations of attention.

The concept of attention is a longstanding and relatively intensively studied construct. Its formal scientific conceptualization is usually dated back to William James’ iconic and oft-cited observation that “Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. Focalization, concentration, of consciousness are of its essence” (James 1890, pp. 403–404). Ironically, James’ apparent attempt to highlight the simplicity and obviousness of the concept of attention also serves to underscore that attention is far from a monolithic construct as it involves a multiplicity of components that are complexly intertwined with each other and in relation to the individual and the world around them.

Current Knowledge

As the study of attention, and its development through the life span, has grown in the century since James’ seminal observation, several foundational issues have emerged as central to any relevant framework. One, attention is not anything tangible, but rather is a construct based on observed behavior that has been forwarded to facilitate the understanding of the complex, multimodal, and ever-changing world in which we live and of the abundant dynamic information, stimuli, and events within. Two, attention is an umbrella term used for many different, albeit conceptually related, functions or components that are delineated and categorized differently across the many different conceptualizations or frameworks of attention. Three, unique developmental trajectories can be identified in relation to the demands, efficiency, and effectiveness for each of these attention components. Four, the real-

world demands on attention processes can change in meaningful ways in relation to the individual's age, environment, and transactions with the environment. Five, attentional processes have been identified within and across each of the modalities and these processes can operate in concert with and/or in competition with each other. Six, attention is captured by and directed to events, stimuli, and pieces of information that move in space and change in time. Seven, the context of the attention task influences its difficulty as other concurrent cognitive functions may either enhance or detract from performance.

In contradistinction to James' point of "Everyone knows what attention is," these points are intended to highlight the increasing levels of nuance and complexity in providing some general understanding of attention and the difficulty in articulating any single characteristic of attention that can be applied universally across the conceptualization of attention and its components for all stimuli or information, across different settings, at different developmental stages. This level of deconstruction of the notion of attention is inevitably magnified in considering its manifestation among persons with ASD for whom any type of functioning might vary considerably in relation to the vast differences within the population with regard to developmental level of functioning, level of symptomatology, and etiology. Thus, the claim for utilitarian attention is not meant as necessarily universal with regard to all components of attention nor for all persons with ASD, but rather as insight into unique, and often unexpected, findings of strengths or patterns of performance and as a framework for interpreting the biases and styles of processing displayed on certain tasks.

Although the notion of utilitarian attention among persons with ASD is recent and has not yet been tested directly, its different manifestations appear to be supported by findings from a range of studies on various components of attending. The Brodeur et al. (2018) study was designed to assess the visual filtering component of attention, which is typically depicted as the process of focusing on some stimulus or event while ignoring, or filtering, other potentially distracting non-target information in the environment. The

experimental paradigm was a flanker task, commonly used in studies of filtering, in which participants are required to respond to the identity of a target stimulus that is presented between flanker stimuli which, depending on their physical attributes and location in relation to the target stimulus, appear to serve as distractors that can impede performance as indicated by reaction times (RTs) and/or error rates. In their task that was administered to a group of children with ASD and a group of typically developing (TD) children matched at a mental age (MA) level of approximately 7.4 years, Brodeur et al. instituted both spatial and temporal manipulations. They presented the target stimulus in the middle of a computer screen and flanked it with nontarget stimuli (i.e., flankers) that were presented 180° to the right or the left of the target at three different spatial distances (2.32, 6.92, and 13.78 visual degrees) and, in the most novel aspect of the paradigm, with four conditions of temporal delays (0, 150, 300, and 450 ms) in the onset between the presentation of the target and the flankers. With regard to the spatial effect, the TD children showed the expected distracting effects as their RTs were slower in the conditions in which the flankers were at closer proximity to the target, but apparently unaffected in the condition in which the distractors were considerably more distant from the target. With regard to the temporal effect, the performance of these participants appeared to be unaffected by the presence or lengths of the temporal delays between the onset of the target and flankers. In comparison, the participants with ASD displayed a quite different pattern of performance. They showed no spatial effect in relation to distance with similar RTs for the three conditions of flanker distances, but displayed effects of the temporal relationship with faster RTs in the conditions in which the distractors appeared either simultaneously with the target or very briefly after it (150 ms as opposed to 300 or 450 ms).

Based on these findings, Brodeur et al. concluded that the children with ASD were able to utilize the flankers in the simultaneous and 150 ms conditions in such a way as to create a window of attention that facilitated their focus on the target, thereby speeding up their RTs. Thus, rather than

being distracted by the flanker stimuli, as appeared to be the case with the TD participants, the participants with ASD appeared to adaptively utilize the spatial location of the flankers as cues to help focus attention on the target stimulus, regardless of the distance of the flankers from the target when the target and flanker stimuli were closely linked temporally. This apparently unique pattern, in which the participants with ASD were able to benefit from the flanker stimuli, was interpreted by Brodeur et al. as indicative of utilitarian attention.

Brodeur et al.'s case for utilitarian attention based on the ability to use the flankers as cues is consistent with earlier findings by Burack (1994) on another filtering task that also involved a flanker paradigm. In that study, Burack (1994) found that only a group of low functioning persons with autism, as compared to two groups of mental age (MA)-matched individuals with intellectual disability and a group of MA-matched typically developing persons, showed enhanced RTs in identifying a target stimulus that appeared in the middle of the screen in conditions with no distractors when a visual window was superimposed around the middle of a screen. Other evidence from the study indicated that the persons with ASD were generally less efficient than the other groups in modulating, or adjusting, the attentional lens to focus on the target as their focus of attention was distributed more broadly and extended out farther from the target. Thus, as the presence of a window was only associated with improvements in their performance, the persons with ASD seemed to have imposed a utilitarian approach to attending by using the window as an external facilitator to effectively narrow the lens, or width of spatial attention, a task which otherwise seemed to be difficult for them. Within this context, the findings from the Brodeur et al. study appear to provide further evidence of the facilitative effects of a spatial framing cue even when comprised of discrete stimuli, such as the flankers, rather than a complete framing of the target. This all suggests that one aspect of the utilitarianism in attention might involve some way of cognitively interpreting, processing, or manipulating nontarget stimuli in the environment

such that they uniquely serve as beneficial cues to persons with ASD, although they may be deleterious to the performance of other persons.

Iarocci et al. (2006) suggested a second type of utilitarian attention when they concluded that the children with high functioning ASD showed a more "data -driven" approach as compared to both verbal and nonverbal MA-matched groups of typically developing children in their two-experiment study of visual search for global and local stimuli. In the first experiment, the children with ASD were as able as the TD participants to access targets at either level of visual structure on a visual search task in which the effects of structural bias were manipulated to favor access to either the local or global targets. However, group differences were noted in the second experiment in which a structural global bias was pitted against an implicit task bias that favored the local level, with the probabilities that target would appear at the local level varying from unlikely (30%) to likely (70%) within the blocks of trials. The children with ASD displayed similar levels of sensitivity to the biasing manipulations for both the global and local targets, while also showing the smallest task independent bias in favor of global targets. Iarocci et al. argued that the sensitivity to bias shown by children in this experiment cannot be interpreted as a response bias, as the biasing manipulation only affected the likelihood that the target stimulus would appear at either global or local level, and did not affect the likelihood of the actual response. Rather, Iarocci et al. concluded that the sensitivity to bias shown by the children with autism indicated a unique enhanced ability to overcome, or ignore, task independent biases to either the local or the global level of processing that typically interfere with the performance of other persons.

In another study in which the findings that can be interpreted as reflecting "data-driven" behavior, Ristic et al. (2005) challenged the prevailing notion that persons with ASD were unable, or at least severely impaired in the ability, to follow eye gaze. In doing so, they contrasted the notion of the "social power" of eye gaze in the everyday functioning of typically developing (TD) persons that leads to the inherent following of eye movement

in all faces, even clearly unrealistic faces such as schematic ones, with a pattern of responding among persons with ASD both that indicated an ability to follow and make meaning of eye gaze and that is inherently more efficient, or cost effective, as it is only used when the data from the environment suggests its utility.

Ristic et al. (2005) presented high functioning adolescents and young adults with ASD and a group of chronological age- and IQ-matched typically developing participants with a centrally presented schematic face whose pupils could move from side to side to cue the location of a target presented to its right or left. This comparison involved two conditions – a nonpredictive condition in which the cue only predicted the location of the target on 50% of trials and a predictive gaze condition in which the cue direction predicted the location of the target 80% of the time. As expected both from previous evidence indicating the salience of eyes in the general population and from the narrative of eye gaze following deficits among persons with ASD, the TD participants, but not those with ASD, displayed faster RTs for congruent cue-target trials than for incongruent cue-target trials in the nonpredictive condition. However, any support for a deficit was dispelled in the predictive condition on which the participants with ASD, like the TD participants, displayed faster RTs for congruent cue-target trials than for incongruent cue-target trials in the predictive condition, suggesting that both groups used the cue in an advantageous way to guide their anticipation of where the target would appear. These findings indicate an essential difference in the utility of attention between persons with ASD and their neurotypical peers, whose attending appeared to be governed by a heuristic that prioritizes social information. Whereas eye gaze automatically captured the attention of the TD participants regardless of whether it provided any information about the subsequent location of a target, the participants with ASD appeared to display a more utilitarian approach to attending by only following eye gaze in situations in which it was useful to successful task completion.

In a similar study in which eye-tracking technology was used to monitor attention to eyes and faces in both an object search and gaze-reading task, Del Bianco et al. (2018) found that proportional looking time to faces was task-dependent among a group of adults with ASD but not for a comparison group of TD adults. Although the groups displayed similar proportions of first fixations to the faces, only the participants with ASD looked at faces more when it was most beneficial to do so; in the gaze-reading task. Del Bianco et al. speculated that the performance of the persons with ASD might reflect less efficient attending, although the increased task-dependent behavior certainly suggests a utilitarian approach to attending, especially in the context of the focus on eyes and faces which is so often cited as a source of deficit among persons with ASD.

Joosten et al. (2016) similarly examined gaze and visual search strategies in children with ASD and neurotypical children using a video of a magic trick as the to-be-attended event. Eye movements were recorded toward both the gaze of the magician and other objects in the video during the initial viewing of the trick, and again after the participants were informed that magicians use gaze-aversion tactics to draw the audience's attention away from specific areas to ensure the success of the trick. Consistent with the argument that the attention of persons with ASD is less governed by general heuristics (e.g., social information prioritization), the a priori attention of the neurotypical children was characterized by a bias to attend to gaze, whereas the children with ASD showed no such search pattern as they showed little preference for eyes or gaze-cued stimuli. However, after learning about the magician's gaze aversion tactic, both groups adjusted their attentional style to look less at eyes and gaze-cued objects. In addition to highlighting that persons with ASD, like TD persons, can use instructions to adopt an optimal attention strategy to increase the likelihood of successful task performance, these findings are further support for the notion that the attention of persons with ASD is less influenced than that of TD persons by social stimuli that have been shown to impede task performance in some circumstances.

Future Directions

As is the case with any construct, identifying the source of utilitarian attention, which is found among at least some persons with ASD on at least some types of tasks, is a conceptual exercise that can involve many possibilities. The enhanced perceptual functioning model (Mottron and Burack 2001; Mottron et al. 2006), based on extensive evidence of superior visual and auditory sensory capacity among persons with ASD, provides one foundation for understanding why the attentional processing of persons with ASD might be particularly utilitarian. As perceptual processes are thought to temporally precede those of attention and other aspects of cognition, faster or better perception might pave the way for more economical or efficient “downstream” processing, including that of attention. Alternatively, Pellicano and Burr (2012) offer a Bayesian account in which perception is based on the interplay between basic sensory capacities and prior probabilities, based on prior knowledge about the world. Among persons with ASD, perceptual input may be interpreted more accurately due to attenuated Bayesian priors, referred to as “hypopriors,” that lead to perception that is less biased by accrued experiences with the perceptual environment. Such a model is consistent with utilitarian attention in that perception, presumably related to attention components, is governed less by heuristics and more by data-driven or current, task specific, utilitarian information.

Commensurate with contemporary perspectives in which the emphasis is on individual styles, experiences, motivation, background knowledge, needs, and interests that affect attention with specific information in particular contexts (Burack et al. 2016; Krauzlis et al. 2014; Ristic and Enns 2015), the hypothesized link between engineering and ASD and the related notion that persons with ASD show advanced levels of folk physics (Baron-Cohen et al. 1997; Wheelwright and Baron-Cohen 1998) suggest that persons with ASD might be uniquely able to benefit from the abilities both to construct facilitative visual cues and efficiently utilize task-relevant data in the

environment while ignoring other information or events that distract others.

A first step in future scholarship would be to revisit and reassess key findings from the literature on attention among persons with ASD, especially cases of particular strengths, in order to identify other examples of attending that can be interpreted as utilitarian. These instances should be examined within the contexts of the various theoretical perspectives that might explain utilitarian attention. The information from this conceptual exercise will inform the quest to delineate the environmental settings, attentional tasks, and individual characteristics that are most likely to elicit utilitarian attention. With this type of information, researchers and practitioners will be able to facilitate the development of educational and vocational opportunities for persons with ASD that allow them to benefit from their particularly unique ways of attending to and processing information in the world around them.

See Also

- [Enhanced Perceptual Functioning](#)
- [Gaze](#)
- [Global Versus Local Processing](#)
- [Perception](#)
- [Sensory Overresponsivity as a Predictor of Amplitude Discrimination Performance in Youth with ASD](#)

References and Reading

- Baron-Cohen, S., Wheelwright, S., Stott, C., Bolton, P., & Goodyer, I. (1997). Is there a link between engineering and autism. *Autism, 1*, 101–109.
- Brodeur, D. A., Stewart, J., Dawkins, T., & Burack, J. A. (2018). Utilitarian attention by children with autism spectrum disorder on a filtering task. *Journal of Autism and Developmental Disorders, 48*, 1419–1427. <https://doi.org/10.1007/s10803-018-3619-5>.
- Burack, J. A. (1994). Selective attention deficits in persons with autism: Preliminary evidence for an inefficient attentional lens. *Journal of Abnormal Psychology, 103*, 535–543.
- Burack, J. A., Russo, N., Kovshoff, H., Fernandes, T. P., Ringo, J., Landry, O., & Iarocci, G. (2016). How I attend – Not how well do I attend: Rethinking

- developmental frameworks of attention and cognition in autism spectrum disorder and typical development. *Journal of Cognition and Development*, 17, 553–567. <https://doi.org/10.1080/15248372.2016.1197226>.
- Burack, J. A., Jefferies, L. N., Ringo, J., & Landry, O. (2017). Attention. In B. Hopkins, E. Geangu, & S. Linkenauer (Eds.), *The Cambridge encyclopedia of child development*. New York: Cambridge University Press.
- Del Bianco, T., Mazzoni, N., Bentenuto, A., & Venuti, P. (2018). An investigation of attention to faces and eyes: Looking time is task-dependent in autism spectrum disorder. *Frontiers in Psychology*, 9. <https://doi.org/10.3389/fpsyg.2018.02629/full>.
- Iarocci, G., Burack, J. A., Shore, D. I., Mottron, L., & Enns, J. T. (2006). Local-global visual processing in high-functioning children with autism: Structural vs. implicit task biases. *Journal of Autism and Developmental Disorders*, 36, 117–129. <https://doi.org/10.1007/s10803-005-0045-2>.
- James, W. (1890). *The principles of psychology*. New York: H. Holt.
- Joosten, A., Girdler, S., Albrecht, M. A., Horlin, C., Falkmer, M., Leung, D., Ordqvist, A., Fleischer, H., & Falkmer, T. (2016). Gaze and visual search strategies of children with Asperger syndrome/high functioning autism viewing a magic trick. *Developmental Neurorehabilitation*, 19, 95–102. <https://doi.org/10.3109/17518423.2014.913081>.
- Krauzlis, R. J., Bollimunta, A., Arcizet, F., & Wang, L. (2014). Attention as an effect not a cause. *Trends in Cognitive Sciences*, 18, 457–464.
- Mottron, L., & Burack, J. A. (2001). Enhanced perceptual functioning in the development of autism. In J. A. Burack, T. Charman, N. Yirmiya, & P. R. Zelazo (Eds.), *The development of autism: Perspectives from theory and research* (pp. 131–148). Mahwah: Lawrence Erlbaum Associates.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36, 27–43. <https://doi.org/10.1007/s10803-005-0040-7>.
- O’Riordan, M. A., Plaisted, K. C., Driver, J., & Baron-Cohen, S. (2001). Superior visual search in autism. *Journal of Experimental Psychology: Human Perception and Performance*, 27, 719–730. <https://doi.org/10.1037/0096-1523.27.3.719>.
- O’Riordan, M., & Plaisted, K. (2001). Enhanced discrimination in autism. *The Quarterly Journal of Experimental Psychology: Section A*, 54(4), 961–979.
- Pellicano, E., & Burr, D. (2012). When the world becomes “too real”: A Bayesian explanation of autistic perception. *Trends in Cognitive Sciences*, 16, 504–510. <https://doi.org/10.1016/j.tics.2012.08.009>.
- Plaisted, K., O’Riordan, M., & Baron-Cohen, S. (1998). Enhanced discrimination of novel, highly similar stimuli by adults with autism during a perceptual learning task. *Journal of Child Psychology and Psychiatry*, 39(5), 765–775.
- Ristic, J., & Enns, J. T. (2015). The changing face of attentional development. *Current Directions in Psychological Science*, 24(1), 24–31.
- Ristic, J., Mottron, L., Kelland Friesen, C., Iarocci, G., Burack, J. A., & Kingstone, A. (2005). Eyes are special but not for everyone: The case of autism. *Cognitive Brain Research*, 24, 715–718. <https://doi.org/10.1016/j.cogbrainres.2005.02.007>.
- Russo, N., Mottron, L., Burack, J. A., & Jemel, B. (2012). Parameters of semantic multisensory integration depend on timing and modality order among people on the autism spectrum: Evidence from event-related potentials. *Neuropsychologia*, 50, 2131–2141.
- Wheelwright, S., & Baron-Cohen, S. (1998). The link between autism and skills such as engineering, maths, physics and computing: A reply to Jarrold and Routh. *Autism*, 2, 281–2289.

Utility of Repeated Autism Screening at 18 and 24 Months

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Definition

Importance of repeated screening for autism spectrum disorder (ASD), following a negative (i.e., no risk) screening, in order to maximize identification of toddlers with ASD and other neurodevelopmental disorders.

Historical Background

Early diagnosis of ASD, followed by targeted intensive intervention services, improves children’s outcomes (Anderson et al. 2014; MacDonald et al. 2014; Rogers and Vismara 2008). Despite the fact that ASD symptoms typically emerge within the first 1–2 years of life and caregivers often report concerns early in their children’s development (Crais et al. 2014; Hazlett et al. 2017; Maestro et al. 2005; Ozonoff et al. 2010; Zwaigenbaum

et al. 2005), a large proportion of children are not diagnosed until they are 3 or 4 years old (Centers for Disease Control and Prevention [CDC], 2018). Routine screening at well-child care visits reduces children's age at diagnosis and mitigates discrepancies in age at diagnosis for children of various racial and socioeconomic status backgrounds (Herlihy et al. 2014). Accordingly, the American Academy of Pediatrics (AAP) recommends screening all children for ASD at 18 and 24 months (Duby et al. 2006; Johnson and Myers 2007). Despite the recommendation to screen at both time points, and the clear benefits of screening, many pediatricians do not practice repeated screening. Indeed, barriers including a lack of time and limited staff to implement screening procedures prohibit screening in many pediatrician practices and are likely to make repeated screening particularly challenging (Arunyanart et al. 2012; Zuckerman et al. 2013). As well, caregivers may have reservations about repeating screening after their children recently screened negative. As data increasingly supports the ability to diagnose ASD earlier (Pierce et al. 2019), as well as the importance of early detection and intervention, it is important to identify the optimal screening time points that enable detection of the largest number of children who would benefit from intervention.

Current Knowledge

To our knowledge, only one manuscript has evaluated the utility of the AAP's recommendation to screen for ASD at 18 and 24 months (Dai et al. 2019). However, researchers have compared the benefits of 18- and 24-month screening as well as the positive predictive value (PPV) of screening at each age.

Symptoms of ASD typically emerge by 18 months, and a diagnosis at that age is considered stable (Ozonoff et al. 2015). In light of literature suggesting the importance of starting intervention as early as possible, during a time of brain plasticity, the AAP encourages routine screening for ASD when children are 18 months of age (Duby et al. 2006; Johnson and Myers 2007). The Modified Checklist for Autism in

Toddlers, Revised, with Follow-Up (M-CHAT-R/F) is the most widely used screener for ASD, has been translated to over 40 languages, and is recommended by the AAP. It is validated for use in children as young as 16 months (Robins et al. 2014).

Still, approximately 20–40% of children with ASD appear to either develop typically or exhibit very mild symptoms until their second year of life, but then experience either a regression of skills or a later emergence of ASD symptoms (Barton et al. 2012; Gupta et al. 2007). In light of this developmental trajectory, these children would not be detected by 18-month screening. Therefore, the AAP encourages repeated screening at 24 months, in order to increase the likelihood of identifying children who were missed by an earlier screening. Indeed, the M-CHAT-R/F PPV is stronger at 24 months relative to 18 months, particularly for children who are drawn from a general population (i.e., low-risk) sample (Pandey et al. 2008; Sturmer et al. 2017).

Caregivers and providers may be reluctant to re-screen children who previously screened negative. However, recent research suggests that 24-month screening is essential to identify children who were missed by an earlier screening. Specifically, a study of 7781 participants who screened negative on the M-CHAT(–R)/F at 18 months, and were rescreened at 24 months, found that 0.4% ($n = 32$) screened positive on the M-CHAT(–R)/F at 24 months (Dai et al. 2019). Twenty of those “at-risk” children attended a diagnostic evaluation, and, of those, 80% ($n = 16$) received a developmental diagnosis (50% ASD ($n = 10$), 30% ($n = 6$) other developmental disorder) warranting intervention. Children who received an ASD diagnosis based on their positive 24-month screening, after having a negative 18-month screening, did not differ on demographics (gender, race, ethnicity, maternal education, or annual income) nor overall developmental functioning, from children who received an ASD diagnosis based on a positive 18-month screening (Dai et al. 2019).

Although only 0.4% of children screened positive at 24 months after screening negative at 18 months, the majority of children who attended

an evaluation as a result of their repeated screening met criteria for a developmental diagnosis. The opportunity to identify additional children who will benefit from early intervention services, in our view, outweighs the risks and possible inconveniences of executing this repeated ASD screening procedure. Thus, Dai et al.'s (2019) results support the AAP's recommendation to screen for ASD at 18 and 24 months in order to facilitate early referral to intervention services while increasing the likelihood of identifying children who were missed by an earlier screener.

Future Directions

The growing emphasis on early diagnosis and intervention underscores the need to determine the optimal ages for ASD screening. Given that barriers are preventing some practices from following AAP recommendations of repeated screening, it is critical for research on the utility of repeated screening to inform clinical practice. Since only one study has evaluated the utility of repeated screening at 18 and 24 months, additional research is needed to replicate results of Dai et al. (2019) with a larger sample of children who screen negative at 18 months and then are diagnosed with ASD following a positive 24-month screening.

Additionally, the AAP's recommendation for screening at 18 and 24 months was made before researchers demonstrated the ability to reliably diagnose ASD within the first year of life. Accordingly, additional research is warranted in order to determine whether 18 and 24 months are the best ages for screening, or if repeated screening at other ages enables detection of a greater number of children at risk.

Finally, the advance of technology enables the M-CHAT-R/F to be administered online. This saves time for pediatricians and may reduce the burden of repeated screening. However, future research is necessary to compare the sensitivity and specificity of the M-CHAT-R/F when it is administered electronically, compared to paper screening followed by a phone interview.

Although there are barriers to routine screening, its utility in helping to detect children with

ASD, reduce the age of diagnosis, and decrease racial disparities in age at diagnosis makes it a critical component of well-child care visits and developmental care. Accordingly, pediatricians are encouraged to continue to screen for ASD during well-child care visits, even if children previously screened negative. Additionally, it is important to continue to encourage caregivers to complete ASD screeners and to follow through on recommendations for evaluations, when warranted.

See Also

- Early Diagnosis
- Screening Measures

References and Reading

- Anderson, D. K., Liang, J. W., & Lord, C. (2014). Predicting young adult outcome among more and less cognitively able individuals with autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 55(5), 485–494.
- Arunyanart, W., Fenick, A., Ukritchon, S., Imjaijit, W., Northrup, V., & Weitzman, C. (2012). Developmental and autism screening: A survey across six states. *Infants & Young Children*, 25(3), 175–187.
- Barton, M. L., Dumont-Mathieu, T., & Fein, D. (2012). Screening young children for autism spectrum disorders in primary practice. *Journal of Autism and Developmental Disorders*, 42, 1165–1174.
- Centers for Disease Control and Prevention. (2018). Prevalence of autism spectrum disorder among children aged 8 years – Autism and developmental disabilities monitoring network, 11 sites, United States, 2014. 27th Apr, Morbidity and Mortality Weekly Report Surveillance Summaries.
- Crais, E. R., McComish, C. S., Humphreys, B. P., Watson, L. R., Baranek, G. T., Reznick, J. S., et al. (2014). Pediatric healthcare professionals' views on autism spectrum disorder screening at 12–18 months. *Journal of Autism and Developmental Disorders*, 44, 2311–2328.
- Dai, Y. G., Miller, L. E., Ramsey, R. K., Robins, D. L., Fein, D. A., & Dumont-Mathieu, T. (2019). Incremental utility of 24-month autism spectrum disorder screening after negative 18-month screening. *Journal of Autism and Developmental Disorders*. Advance online publication. <https://doi.org/10.1007/s10803-019-03959-5>.
- Duby, J. C., Lipkin, P. H., Macias, M. M., Wegner, L. M., Duncan, P., Hagan, J. F., et al. (2006). Identifying infants and young children with developmental disorders in the medical home: An algorithm for

- developmental surveillance and screening. *Pediatrics*, 118(1), 405–420.
- Gupta, V. B., Hyman, S. L., Johnson, C. P., Bryant, J., Byers, B., Kallen, R., et al. (2007). Identifying children with autism early? *Pediatrics*, 119, 152–153.
- Hazlett, H. C., Gu, H., Munsell, B. C., Kim, S. H., & Styner, M. (2017). Wolff, J. J. . . . the IBIS Network. *Early brain development in infants at high risk for autism spectrum disorder*. *Nature*, 542, 348–351.
- Herlihy, L. E., Brooks, B., Dumont-Mathieu, T., Barton, M. L., Fein, D., Chen, C. M., & Robins, D. L. (2014). Standardized screening facilitates timely diagnosis of autism spectrum disorders in a diverse sample of low-risk toddlers. *Journal of developmental and behavioral pediatrics: JDBP*, 35(2), 85.
- Johnson, C. P., & Myers, S. M. (2007). American academy of pediatrics, council on children with disabilities. Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, 120(5), 1183–1215.
- MacDonald, R., Parry-Cruwys, D., Dupere, S., & Ahearn, W. (2014). Assessing progress and outcome of early intensive behavioral intervention for toddlers with autism. *Research in Developmental Disabilities*, 35(12), 3632–3644.
- Maestro, S., Muratori, F., Cesari, A., Cavallaro, M. C., Paziante, A., Pecini, C., et al. (2005). Course of autism signs in the first year of life. *Psychopathology*, 38, 26–31.
- Ozonoff, S., Iosif, A.-M., Baguio, F., Cook, I. C., Hill, M. M., Hutman, T., et al. (2010). A prospective study of the emergence of early behavioral signs of autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(3), 256–266.
- Ozonoff, S., Young, G. S., Landa, R. J., Brian, J., Bryson, S., Charman, T., Chawarska, K., Macari, S. L., Messinger, D., Stone, W. L., & Zwaigenbaum, L. (2015). Diagnostic stability in young children at risk for autism spectrum disorder: a baby siblings research consortium study. *Journal of Child Psychology and Psychiatry*, 56(9), 988–998.
- Pandey, J., Verbalis, A., Robins, D. L., Boorstein, H., Klin, A., Babitz, T., et al. (2008). Screening for autism in older and younger toddlers with the Modified Checklist for Autism in Toddlers. *Autism*, 12(5), 513–535.
- Pierce, K., Gazestani, V. H., Bacon, E., Barnes, C. C., Cha, D., Nalabolu, S., et al. (2019). Evaluation of the diagnostic stability of the early autism spectrum disorder phenotype in the general population starting at 12 months. *JAMA Pediatrics*. Advance online publication. <https://doi.org/10.1001/jamapediatrics.2019.0624>.
- Robins, D. L., Casagrande, K., Barton, M., Chen, C.-M. A., Dumont-Mathieu, T., & Fein, D. (2014). Validation of the Modified Checklist for Autism in Toddlers, Revised with Follow-up (M-CHAT-R/F). *Pediatrics*, 133, 37–45.
- Rogers, S. J., & Vismara, L. A. (2008). Evidence-based comprehensive treatments for early autism. *Journal of Clinical Child & Adolescent Psychology*, 37(1), 8–38.
- Sturner, R., Howard, B., Bergmann, P., Stewart, L., & Afarian, T. E. (2017). Comparison of autism screening in younger and older toddlers. *Journal of autism and developmental disorders*, 47(10), 3180–3188.
- Zuckerman, K. E., Mattox, K., Donelan, K., Batbayar, O., Baghaee, A., & Bethell, C. (2013). Pediatrician identification of Latino children at risk for autism spectrum disorder. *Pediatrics*, 132(3), 445–453.
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, 23, 143–152.

Uzgiris-Hunt Ordinal Scales of Infant Development

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Description

The Uzgiris-Hunt Ordinal Scales of Psychological Development (U-H Scales; Uzgiris and Hunt 1975) are a set of six scales of cognitive development designed for infants from 1 to 24 months of age. Scales were inspired by the work of Piaget (see entry ► “[Piagetian Stages](#)”) and thus are grounded in the theory that development is an “epigenetic process of evolving new, more complex, hierarchical levels of organization in intellect and motivation” (Uzgiris and Hunt, p. 47). The Scales include: Scale I: The Development of Visual Pursuit and the Permanence of Objects, Scale II: The Development of Means for Obtaining Desired Environmental Events, Scale IIIa: The Development of Vocal Imitation, Scale IIIb: The Development of Gestural Imitation, Scale IV: The Development of Operational Causality, Scale V: The Construction of Object Relations in Space, and Scale VI: The Development of Schemes for Relating to Objects.

Historical Background

Deriving from Piaget’s theory of cognitive development in the first 2 years of life (see entry ► “[Sensorimotor Development](#)”), the Uzgiris-Hunt Ordinal Scales of Psychological

Development (Uzgiris and Hunt 1975; hereafter referred to as U-H Scales) were developed as an alternative to contemporaneous infant developmental tests such as the Bayley or the Gesell. Uzgiris and Hunt argued that those tests, simply downward extensions of intelligence tests devised for older children and adults, reflected particular assumptions regarding development of intelligence in infancy. These assumptions included the following: that development in infancy reflects a unitary process ("general intelligence"; Spearman 1904); that individual differences in intelligence are synonymous with differential rates of achievement in relation to chronological age, in other words, that developmental quotient (DQ) is the precursor to IQ and continuous with it; and that developmental skills evolve incrementally, without regard to relationships among achievements or to the possibility of a hierarchical progression (Uzgiris and Hunt 1975). (Although the notion of age-referenced norms was anathema to the authors, because of wider interest, ages at which most infants in the original sample passed individual items are listed in the manual. Indeed, the size of the original sample would preclude the use of the suggested ages as true age norms.)

To challenge each of these assumptions, the authors developed the U-H Scales. First, based on Piagetian concepts, they designed six separate scales that were thought to represent "relatively independent branches" (Uzgiris and Hunt 1975, p. 15) of ability. This was a novel idea in infant assessment at that time; for example, the Bayley Scales had only a few years earlier been divided into a Mental Scale and a Motor Scale. Second, achievements in each domain were proposed to follow a consistent order, yet without being linked to a particular chronological age. From emerging research on the effects of deprivation (e.g., orphanage settings; Provence and Lipton 1962) as well as enrichment (e.g., nursery schools; Skeels et al. 1938) on development, Uzgiris and Hunt noted the critical role of the environment and thus the reduced importance of chronological age in intellectual ability. Thus, the authors intended to dissociate the measurement of intelligence from age and thus from an *a priori* rate of development. Taken to support this view was the finding that

infant DQs were not predictive of IQs in adolescence and adulthood (see Hunt 1961) despite having high split-half reliability as well as high test-retest reliability. Finally, the scales were constructed such that each item built directly on the accomplishments of the previous items. And once again, the Scales were rooted firmly in Piagetian concepts, the authors being strongly influenced by his previous scientific endeavors as a biologist: "We have also presumed that this hierarchical organization of abilities and motives consists of coordinations and differentiations among the several sensorimotor organizations already present at birth, and that progress toward the symbolic representations and regulations comprising competence and intelligence undergoes an epigenetic development analogous to the embryonic development of organ structures from the single cell constituting the fertilized ovum" (Uzgiris and Hunt 1975, p. 15). This notion of the coordination of progressively more complex systems into a hierarchical organization is illustrated by the following description of how basic actions develop into goal-directed activity: "At the simplest level, visual following over wide arcs depends upon a coordination of eye movements with head movements. At a somewhat more complex level comes the sensorimotor coordination of Piaget's second sensorimotor stage. Here, arm movements and grasping become coordinated with looking in the visually-directed reaching...and looking becomes coordinated with listening as things heard become something to look at in the behavior of auditory localization. Here, also, vocalization becomes coordinated with listening as the infant begins to recognize repeatedly encountered sounds and to manifest pseudo-imitation of familiar vocal patterns. During the third and fourth stages, according to Piaget's observations, these first habitual coordinations become coordinated at a new hierarchical level in various means-ends relationships" (Uzgiris and Hunt 1975, p. 32).

In addition to being able to measure development of infants along these six dimensions (rates of which were anticipated to be relatively independent) and compare them to each other, the authors expected that other researchers would

conduct the necessary studies to provide age norms. A further goal was to develop a tool that would facilitate research on the influence of various kinds of circumstances (i.e., child-rearing situations) on development of the various skills contained in the Scales. Finally, the authors foresaw utility in the U-H Scales to measure rates of change as well as guide efforts of intervention programs for young children.

Development of the Instrument

Piaget's manuscripts with their rich and detailed descriptions of his own infants' behavior offered a wealth of examples upon which an assessment tool could be based, the authors recognized. Their goal was to draw from these many examples and arrange them in order, both items that required eliciting *situations* as well as those that were simply observations of spontaneous behavior given a set of toys. From the volumes *The Origins of Intelligence in Children* (Piaget 1936) and *The Construction of Reality in the Child* (Piaget 1937), the authors selected a number of eliciting situations that could be reproduced in a lab setting within a brief period of time. In addition, they compiled *critical actions* associated with each eliciting situation, actions which were proposed to mean that an infant had attained a specific milestone of functioning in each domain of development. Thus began a phase of exploratory testing, inter-rater reliability, and revision that included samples of infants from 1 to 24 months of age. The first sample included 42 infants, the second included 23, and the final phase of study tested 84 infants. The final revision of the assessment was comprised of 73 eliciting situations across six scales: Scale I: The Development of Visual Pursuit and the Permanence of Objects; Scale II: The Development of Means for Obtaining Desired Environmental Events; Scale IIIa: The Development of Vocal Imitation; Scale IIIb: The Development of Gestural Imitation; Scale IV: The Development of Operational Causality; Scale V: The Construction of Object Relations in Space; and Scale VI: The Development of Schemes for Relating to Objects. Notably, the scales were constructed such that quality of performance could be documented, rather than using

the "pass/fail" criterion typical of many assessment measures. Instead, several alternative responses are listed in addition to the *critical actions* (responses that qualified as passing a given level of functioning) so that various types of "failures" could be recorded.

Scale I: The Development of Visual Pursuit and the Permanence of Objects

The items in this series focus on the development of the concept of objects, or, as Piaget referred to it, the construction of objects. At the beginning is the basic scheme of looking. The first step beyond basic looking is the visual pursuit of objects, which gradually progresses to permit following objects through larger and larger arcs until pursuit is completed for an arc of 180°. Later achievements include finding partially and fully hidden objects (object permanence) and then visible and invisible displacements of objects.

Scale II: The Development of Means for Obtaining Desired Environmental Events

This scale involves the actions infants engage in either to cause events or to obtain desired objects. Items range from visually directed grasping to pulling a string to obtain an object to using a stick to obtain a toy out of reach. "Increasing differentiation of actions-as-means from actions-as-ends, increasing determination of means by the envisioned end leading to subordination of means to ends, and increasing anticipation regarding the appropriateness of particular means seem to characterize progress along this sequence" (Uzgiris and Hunt 1975, p. 109).

Scale IIIa: The Development of Vocal Imitation

The beginning of the vocal imitation sequence involves cooing and recognition of the infant's own sounds and progresses to vocalizing in response to another's sounds and finally repeating new words.

Scale IIIb: The Development of Gestural Imitation
Items on this scale concern responses to the gestures of others, and the scale begins with a gestural response to a familiar gesture. Later items include imitation with objects, imitation of visible

gestures, and, finally, imitation of invisible gestures (not visible to the child, e.g., tapping one's own head or clucking one's tongue).

Scale IV: The Development of Operational Causality

The beginnings of comprehension of causality involve repeating actions that result in an interesting spectacle. As this ability develops, the infant will hand a spinning toy back to the examiner after a demonstration, then attempt to activate it himself/herself, and finally will attempt to activate novel objects not yet demonstrated.

Scale V: The Construction of Object Relations in Space

This series focuses on development of implicit understanding of and construction of object relations in space. Early items include accurately localizing the source of sounds, reconstructing the trajectory of rapidly falling objects, recognizing rotation of a three-dimensional object in space, and building a tower of blocks.

Scale VI: The Development of Schemes for Relating to Objects

The steps in this sequence basically describe the early development of play with toys and objects prior to representational (pretend) play. Beginning with simple mouthing and visual inspection and ending with naming objects, several steps in between include actions observed in the first year of life: simple manipulation such as hitting, shaking, dropping, and throwing, followed by so-called *socially instigated* behaviors such as showing and functional play ("indicating appreciation of their usual function"; Uzgiris and Hunt 1987, p. 124).

Clinical Uses

In a follow-up volume, Uzgiris and Hunt (1987) listed 137 research papers that had employed the U-H Scales to date, either in part or in its entirety. Several chapters in the volume were devoted to major areas of inquiry using the Scales, including measurement of progress in early intervention

studies (see Seibert 1987), and developmental assessment of children with mental retardation (Curcio and Houlihan 1987; Kahn 1987), Down Syndrome (Cicchetti and Mans-Wagener 1987), and motor impairments (Robinson and Rosenberg 1987).

As had been done previously in the context of mental retardation/intellectual disability by a host of researchers, measurement of sensorimotor development in autism was first undertaken in order to assess intellectual functioning in severely affected nonverbal children (Curcio 1978). The U-H Scales had recently been published, and was the instrument of choice for many researchers studying cognitive development in children with autism. The resulting studies reported no deficits in children with autism in most areas of sensorimotor development compared to mental-age-matched peers, with the notable exception of gestural imitation. Furthermore, a relative strength in object permanence skills was often observed in the children with autism (Dawson and McKissick 1984; Morgan et al. 1989; Sigman and Ungerer 1981, 1984; Wetherby and Gaines 1982). See Dunst (1980) for an attempt to provide more traditional norms for these scales.

See Also

- [Object Permanence](#)
- [Piagetian Stages](#)
- [Sensorimotor Development](#)

References and Reading

- Cicchetti, D., & Mans-Wagener, L. (1987). Sequences, stages, and structures in the organization of cognitive development in infants with Down syndrome. In I. C. Uzgiris & J. M. Hunt (Eds.), *Infant performance and experience: New findings with the ordinal scales* (pp. 281–310). Urbana: University of Illinois Press.
- Curcio, F. (1978). Sensorimotor functioning and communication in mute autistic children. *Journal of Autism and Childhood Schizophrenia*, 8(3), 281–292.
- Curcio, F., & Houlihan, J. (1987). Varieties of organization between domains of sensorimotor intelligence in normal and atypical populations. In I. C. Uzgiris & J. M.

- Hunt (Eds.), *Infant performance and experience: New findings with the ordinal scales* (pp. 98–130). Urbana: University of Illinois Press.
- Dawson, G., & McKissick, F. C. (1984). Self-recognition in autistic children. *Journal of Autism and Developmental Disorders*, 14(4), 383–394.
- Dunst, C. J. (1980). *A clinical and educational manual for use with the Uzgiris and Hunt scales of infant psychological development*. Austin: Pro-Ed.
- Hunt, J. M. (1961). *Intelligence and experience*. New York: Ronald Press.
- Kahn, J. V. (1987). Uses of the scales with mentally retarded populations. In I. C. Uzgiris & J. M. Hunt (Eds.), *Infant Performance and Experience: New Findings with the Ordinal Scales*. Urbana/Chicago: University of Illinois Press.
- Morgan, S. B., Cutrer, P. S., Coplin, J. W., & Rodrigue, J. R. (1989). Do autistic children differ from retarded and normal children in Piagetian sensorimotor functioning? *Journal of Child Psychology and Psychiatry*, 30(6), 857–864.
- Piaget, J. (1936). *The origins of intelligence in children*. New York: International Universities Press.
- Piaget, J. (1937). *The construction of reality in the child*. New York: Basic Books.
- Piaget, J. (1952). *The origins of intelligence in children*. New York: International Universities Press.
- Piaget, J. (1954). *The construction of reality in the child*. New York: Basic Books.
- Provence, S., & Lipton, R. C. (1962). *Infants in institutions*. New York: International University Press.
- Robinson, C., & Rosenberg, S. (1987). A strategy for assessing infants with motor impairments. In I. C. Uzgiris & J. M. Hunt (Eds.), *Infant performance and experience: New findings with the ordinal scales* (pp. 311–339). Urbana: University of Illinois Press.
- Seibert, J. (1987). Uses of the scales in early intervention. In I. C. Uzgiris & J. M. Hunt (Eds.), *Infant performance and experience: New findings with the ordinal scales* (pp. 340–370). Urbana: University of Illinois Press.
- Sigman, M., & Ungerer, J. (1981). Sensorimotor skills and language comprehension in autistic children. *Journal of Abnormal Child Psychology*, 9(2), 149–165.
- Sigman, M., & Ungerer, J. (1984). Cognitive and language skills in autistic, mentally retarded, and normal children. *Developmental Psychology*, 20(2), 293–302.
- Skeels, H. M., Updegraff, R., Wellman, L., & Williams, H. M. (1938). A study of environmental stimulation: An orphanage preschool project. *University of Iowa Studies of Child Welfare*, 15, 4.
- Spearman, C. (1904). “General intelligence” objectively determined and measured. *American Journal of Psychology*, 15, 201–293.
- Uzgiris, I. C., & Hunt, J. M. (1975). *Assessment in infancy: Ordinal scales of psychological development*. Urbana: University of Illinois Press.
- Uzgiris, I. C., & Hunt, J. M. (1987). *Infant performance and experience: New findings with the ordinal scales*. Urbana: University of Illinois Press.
- Wetherby, A. M., & Gaines, B. (1982). Cognition and language development in Autism. *The Journal of Speech and Hearing Disorders*, 47(1), 63–70.

V1

► Visual Cortex

VABS-II: Vineland Adaptive Behavior Scales-II

► PEAK Relational Training System

Vaccinations and Autism

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Definition

In 1998, Andrew Wakefield and co-workers published an article in *The Lancet* claiming that the combination measles-mumps-rubella (MMR) vaccine caused autism (see ► [Measles-Mumps-Rubella \(MMR\) Vaccination](#)). One year later, following a pointed directive by the United States Public Health Service and the American Academy of Pediatrics (AAP) to remove thimerosal (an ethylmercury-containing preservative) from vaccines, parents became concerned that mercury

caused autism. During the next ten years, epidemiological studies showed that both of these hypotheses were incorrect.

A few years later, the hypothesis again shifted – this time to the more diffuse and more difficult to test notion that too many vaccines given too early caused autism. While it is unethical to prospectively test children who either have or have not been vaccinated to determine differences in neurodevelopmental outcomes, one study has addressed this question retrospectively.

Michael Smith and Charles Woods took advantage of a study performed and published by William Thompson and co-workers at the Centers for Disease Control and Prevention (CDC). Thompson evaluated more than 1,000 children: first, by carefully examining the medical records of every child to determine how much mercury the child had received either prenatally in RhoGam or postnatally in vaccines and second, by prospectively administering more than 40 neurodevelopmental and psychological tests to determine if the neurological outcome could be correlated with the quantity of mercury to which the child had been exposed. Thompson found no relationship between mercury exposure and autistic spectrum disorder.

Smith and Woods mined data from the Thompson study to answer a different question. They noted that some parents had immunized their children according to the CDC/AAP schedule while others had chosen to delay, withhold, separate, or space out vaccines. Separating these children into

two groups, they found no relationship between the timing of vaccination and neurological outcome.

It is likely that the hypotheses concerning the relationship between vaccination and autism spectrum disorder will continue to shift to include adjuvants, manufacturing residuals, inactivating agents, or growth factors. All hypotheses are testable but are likely to be fruitless endeavors.

References and Reading

- Smith, M., & Woods, C. (2010). On-time vaccine receipt in the first year does not adversely affect neuropsychological outcomes. *Pediatrics*, 125, 1–8.
- Thompson, W., Price, C., Goodson, B., Shay, D. K., Benson, P., Hinrichsen, V. L., et al. (2007). Early thimerosal exposure and neuropsychological outcomes at 7 to 10 years. *The New England Journal of Medicine*, 357, 1281–1292.

Validity

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Definition

The term “validity” refers to the extent to which a concept or measurement accurately corresponds to the real world.

In psychiatry and psychology, validity is often discussed in terms of its application to psychometrics, or the study of psychological measurement. This application of the term is known as test validity, which refers to the degree to which a test measures what it claims to measure (Anastasi and Urbina 1997). Test validity is important because it determines the inferences that can be made based on the test results.

In evaluating the validity of a test, several lines of evidence can be considered to support its validity. In particular, three principal types of validity must be considered: content validity, criterion

validity, and construct validity (Lord and Corsello 2005; Sattler 2008). Content validity refers to the degree that the items on a test accurately represent the domain that the test is aiming to measure. Building content validity into a test involves careful selection of items to include, with particular focus on the thoroughness of the item sample. Criterion validity refers to the relationship between the test score and a criterion variable, such as other measures or outcomes, which has already been determined as valid. Oftentimes, a diagnosis is used as the criterion of reference, in which cases this analysis is referred to as establishing diagnostic validity. Finally, construct validity refers to the extent to which a test accurately measures a psychological construct or trait. It is evaluated by examining the correlations between the test and different measures of the same construct, as well as correlations between the test of interests and those designed to measure different constructs. Positive correlations between measures of the same domain and the absence of correlations between measures of different domains provide evidence of construct validity.

An additional use of the term in psychiatry concerns assessing the validity of diagnostic categories, or the extent to which the definition of the diagnosis accurately describes its presentation. An influential paper, published in 1970, listed five criteria for establishing the validity of psychiatric diagnoses (Robins and Guze 1970). First, the disorder must have a clear clinical description, including symptomology, demographic characteristics, and etiology. Second, laboratory tests must be conducted to further inform the clinical description. Third, the disorder of interest must be differentiated from related disorders. The fourth criterion involves conducting follow-up studies to establish the characteristic course of the disorder and provide evidence of diagnostic stability. Finally, family studies should be conducted to determine the extent to which the disorder is heritable.

See Also

► [Psychological Assessment](#)

References and Reading

- Anastasi, A., & Urbina, S. (1997). *Psychological testing* (7th ed.). Upper Saddle River: Prentice Hall.
- Lord, C., & Corsello, C. (2005). Diagnostic instruments in autistic spectrum disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 730–771). Hoboken: Wiley.
- Robins, E., & Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry*, 126, 983–987.
- Sattler, J. M. (2008). *Assessment of children: Cognitive foundations* (5th ed.). La Mesa: Jerome M. Sattler, Publisher, Inc..

Valium

- [Diazepam](#)

Valproate

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Synonyms

[Divalproex sodium](#) (Depakote, Depakote ER/CP); [Valproate sodium](#) (Depacon); [Valproic acid](#) (Depakene and Stavzor)

Definition

Valproate is a broad spectrum anticonvulsant used to treat seizures, manic episodes, and migraines. The exact mechanism of action underlining each of the drug's clinical effects remains unclear. However, a wide range of actions have been reported to contribute to the drug's diverse effects, such as: increasing the levels of gamma-aminobutyric acid (GABA) in the brain, inhibiting voltage-gated sodium channels, reducing levels of excitatory amino acid (such as beta-hydrobutyric

acid), inhibiting calcium channels, modulating dopaminergic and serotonergic transmission, and inhibiting histone deacetylase.

Common side effects reported include tremors, confusion, fatigue, dizziness, nausea, weight gain, abdominal cramps, and abnormal liver functioning. Other potential side effects include liver damage (i.e., hepatotoxicity), metabolic and endocrine adverse effects, and teratogenicity. Prenatal exposure to valproate is associated with delayed cognitive development and congenital malformations. Additionally, prenatal use of valproate significantly increases the risk for autism spectrum disorder. Studies have shown that individuals exposed to valproate during prenatal development may be up to three times more likely to develop autism, when compared to individuals who were not exposed to valproate.

See Also

- [Epilepsy](#)
- [Seizure](#)

References and Reading

- Christensen, J., Grønberg, T. K., Sørensen, M. J., Schendel, D., Parner, E. T., Pedersen, L. H., & Vestergaard, M. (2013). Prenatal valproate exposure and risk of autism spectrum disorders and childhood autism. *JAMA*, 309, 1696–1703.
- Tomson, T., Battino, D., & Perucca, E. (2016). Valproic acid after five decades of use in epilepsy: Time to reconsider the indications of a time-honoured drug. *The Lancet Neurology*, 15, 210–218.

Valproate Sodium (Depacon)

- [Valproate](#)

Valproic Acid

- [Depakene](#)

Valproic Acid (Depakene and Stavzor)

- ▶ [Valproate](#)

Vanderbilt Treatment and Research Institute for Autism Spectrum Disorders (TRIAD) Social Skills Assessment (TSSA)

- ▶ [TRIAD Social Skills Assessment](#)

Vanspar

- ▶ [Buspirone](#)

Variable Expressivity of Genes

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Definition

It describes the phenomenon of differing clinical features or phenotype among individuals carrying the same gene allele or genotype. Expressivity differs from *penetrance*, which describes the probability that a genetic variation (or allele) will yield the phenotype at all. Variable expression of a phenotype may be influenced by other genetic variations, epigenetic factors, or environment. This term is often used in conjunction with explanations of variable severity of autism-related traits within families. Certain copy number variants (CNVs) associated with autism display variable expressivity, showing association with other neurodevelopmental disorders, including schizophrenia, epilepsy, and intellectual disability.

See Also

- ▶ [Epigenetic Mechanisms](#)
- ▶ [Pleiotropy](#)

References and Reading

- McCarthy, S., Makarov, V., Kirov, G., Addington, A., McClellan, J., Yoon, S., et al. (2009). Microduplications of 16p11.2 are associated with schizophrenia. *Nature Genetics*, 41(11), 1223–1227.
- Sanders, S. J., Ercan-Sencicek, A. G., Hus, V., Luo, R., Murtha, M. T., Moreno-De-Luca, D., et al. (2011). Multiple recurrent de novo copy number variations (CNVs), including duplications of the 7q11.23 Williams-Buren syndrome region, are strongly associated with autism. *Neuron*, 70(5), 863–885.
- Weiss, L., Shen, Y., Korn, J., Arking, D., Miller, D., Fossdal, R., et al. (2008). Association between microdeletion and microduplication at 16p11.2 and autism. *The New England Journal of Medicine*, 358(7), 667–675.

VB-MAPP

- ▶ [Verbal Behavior Milestones Assessment and Placement Program \(VB-MAPP\)](#)

VB-MAPP: Verbal Behavior Milestones and Placement Program

- ▶ [PEAK Relational Training System](#)

VCFS

- ▶ [Developmental, Psychiatric, and Genetic Profiles of the 22q11.2 Deletion Syndrome](#)

Velocardiofacial Syndrome

- ▶ [CATCH 22 \(Chromosome 22q11 Deletion Syndrome\)](#)

Venlafaxine

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Synonyms

Brand names: [Effexor](#) (immediate release); [Effexor XR](#) (extended release)

Indications

According to the official prescribing information, venlafaxine is FDA-approved to treat major depressive disorder (MDD), generalized anxiety disorder (GAD), social anxiety disorder, and panic disorder with or without agoraphobia in adults (Wyeth Pharmaceuticals 2009). It is not approved for children or adolescents under the age of 18 years.

Venlafaxine is not approved for the treatment of autism spectrum disorders (ASDs). ASDs include the DSM-IV-TR diagnoses of autistic disorder, Asperger's disorder, and pervasive developmental disorder, not otherwise specified (PDD-NOS). The decision to use venlafaxine in the treatment of established clinical indications that may co-occur with ASDs, as mentioned above, or for commonly observed symptoms of ASDs, such as hyperactivity, inattention, irritability, aggression, repetitive behaviors, and social impairment, can be made on an individual basis by the treating practitioner. This will be discussed in more detail in the "[Clinical Uses](#)" section.

Mechanisms of Action

Mechanism of Action According to Stahl's *Essential Psychopharmacology* (2008)

Venlafaxine is a serotonin norepinephrine reuptake inhibitor (SNRI), resulting in reuptake blockade of both serotonin and norepinephrine at the level of neuronal synapses and dendrites. This differs from selective serotonin reuptake inhibitors (SSRIs), which block only serotonin reuptake. Usually, the neurotransmitters serotonin and norepinephrine are released from the presynaptic axon terminal to travel across the synaptic cleft and attach to the postsynaptic axon terminal. Any excess neurotransmitter remaining in the cleft is picked up by their respective reuptake transporters in the presynaptic axon to be inactivated for later use or degraded. Serotonin has long been one of the neurotransmitters implicated in the pathophysiology of depression and anxiety disorders. It is hypothesized that a depletion of serotonin available to act on the postsynaptic axon results in symptoms such as depression, nervousness, guilt, loneliness, apathy, and avolition. Antidepressants with serotonin reuptake inhibition, like SSRIs and SNRIs, block serotonin from reuptake at the presynaptic reuptake transporter. This provides excess serotonin to attach to postsynaptic receptors and exerts the intended antidepressant and anxiolytic effects. Additionally, norepinephrine has been found to regulate serotonin release and reuptake. When norepinephrine floods the synaptic cleft, it is taken up by presynaptic alpha-2 receptors, which then decreases the amount of serotonin released at the axon. Norepinephrine can also be taken up by alpha-2 receptors on neuronal dendrites, which sends a signal to the neuron to decrease the amount of serotonin released at the axon terminal. Conversely, norepinephrine that is taken up by dendritic alpha-1 receptors sends a signal to the axon to accelerate serotonin release. This interplay is further complicated by specific serotonin receptors regulating the release of norepinephrine. Furthermore, dopamine is a third neurotransmitter whose regulation is influenced by serotonin and norepinephrine and which likely plays a role in the pathophysiology of depression and anxiety

disorders. When serotonin and norepinephrine reuptake transporters are blocked via SNRIs, dopamine is increased in the prefrontal cortex. This can lead some patients to feel more energized and less fatigued, as well as demonstrate improvements in concentration and attention. The global consequence is that multiple neurotransmitters regulate multiple brain pathways via positive and negative feedback loops. SNRIs capitalize on these complex relationships and are targeted to treat symptoms experienced in mood and anxiety disorders.

Symptoms of depression include depressed mood, apathy, sleep disturbance (typically sleeplessness), both mental and physical fatigue, slowed executive function, psychomotor retardation or agitation, changes in appetite (usually decreased), suicidal thinking, and feelings of guilt or worthlessness. The pathophysiology of these symptoms is thought to result from complicated interactions between serotonin, norepinephrine, and dopamine in various areas of the brain. Depressed mood is thought to stem from hypoactivity of all three neurotransmitters in the amygdala and ventromedial prefrontal cortex. Apathy is hypothesized to be the result of norepinephrine and dopamine dysfunction in the prefrontal cortex, hypothalamus, and nucleus accumbens. Sleep disturbance is linked to all three aforementioned neurotransmitters in the prefrontal cortex, hypothalamus, and basal forebrain. Mental fatigue is thought to be intimately linked to hypoactivity of norepinephrine on noradrenergic receptors in the prefrontal cortex and physical fatigue by hypoactivity of noradrenergic receptors in the descending spinal cord. Dopamine hypoactivity in the prefrontal cortex also contributes to mental fatigue, but dopamine dysfunction is linked to physical fatigue in the striatum, nucleus accumbens, and hypothalamus. Norepinephrine and dopamine dysfunction in the dorsolateral prefrontal cortex is implicated in slowed executive functioning. Psychomotor agitation or retardation is linked to hypoactivity of all three neurotransmitters in the prefrontal cortex, serotonin and dopamine in the striatum and nucleus accumbens, and serotonin and norepinephrine in the cerebellum. Serotonin

hypoactivity in the hypothalamus is implicated in decreased appetite commonly experienced in depression, whereas serotonin hypoactivity in both the ventromedial prefrontal and orbital frontal cortices can result in suicidal ideation. Feelings of guilt or worthlessness are often attributed to serotonin hypoactivity in the ventromedial prefrontal cortex and amygdala.

A number of medications that effectively treat depression also effectively treat anxiety disorders due to an overlap in symptomatology. Anxiety disorders include GAD, obsessive-compulsive disorder (OCD), panic disorder, social anxiety disorder, and posttraumatic stress disorder (PTSD), with core symptoms of all five being anxiety, fear, and worry. Anxiety and fear are due to dysfunction in the amygdala, in part, which synthesizes input from multiple brain areas to determine the need for a fear response. This area is regulated by a number of neurotransmitters, including serotonin and norepinephrine, so these are subsequently implicated in the pathophysiology of anxiety and fear. Both SSRIs and SNRIs increase the amount of serotonin available to the postsynaptic neuron by blocking the serotonin reuptake transporter in the presynaptic neuron and are effective at reducing anxiety and fear in all five anxiety disorders. However, not all SSRIs or SNRIs are approved for the treatment of all five anxiety disorders; prescribers should rely on data demonstrating their efficacy when choosing a treatment. Interestingly, serotonin reuptake transporters have been found to differ genetically from person to person, with certain types leading to differences in how fearful stimuli are processed. This may determine a person's genetic predisposition to anxiety or fear, providing additional information to the prescriber when choosing a treatment. The symptom of worry, on the other hand, is thought to be due to overactivation of a cortico-striatal-thalamic-cortical feedback loop. Like the pathophysiology of anxiety, multiple neurotransmitters are also believed to regulate this loop, including serotonin and norepinephrine. Therefore, certain SSRIs and SNRIs, which manage these neurotransmitters, are often effective at managing worry and obsessional thinking associated with OCD.

Venlafaxine's inhibition of serotonin and norepinephrine reuptake begins immediately, but its antidepressant and anxiolytic effects are often not experienced for up to 6–8 weeks. This is thought to be due to gradual changes on serotonin receptor sensitivity. The belief is that decreased amounts of serotonin hypothesized to occur in depression and anxiety disorders causes the postsynaptic axon to upregulate the number of postsynaptic axonal receptors expressed. In other words, a decreased amount of serotonin in the synaptic cleft results in an increased amount of receptors at the ready. When venlafaxine enters the picture, the level of serotonin in the cleft rises. Postsynaptic receptors sense the serotonin increase and send messages to the axon nucleus about the changes in the synaptic cleft. The nucleus begins downregulating the amount of serotonin receptors ready to receive serotonin. These changes are gradual and are believed to take 6–8 weeks before fully completed, reflecting the amount of time before many patients begin feeling relief from their symptoms.

Venlafaxine's serotonin and norepinephrine reuptake is dose dependent, with the most potent serotonin inhibition occurring at low doses and moderate norepinephrine inhibition occurring at higher doses (Wyeth Pharmaceuticals 2009). In a dosing range of 75–225 mg/day, many patients experience mostly serotonergic blockade effects whereas others experience dual serotonin and norepinephrine blockade effects (Stahl 2009). In a dosing range between 225 and 375 mg/day, most patients experience dual serotonin and norepinephrine blockade effects. Venlafaxine is a substrate for the CYP450 2D6 enzyme. It is metabolized to the major active metabolite O-desmethylvenlafaxine, also known as desvenlafaxine in the liver (Wyeth Pharmaceuticals 2009). O-Desmethylvenlafaxine has higher norepinephrine reuptake inhibition compared to venlafaxine, so administration of the drug results in variable levels of venlafaxine and O-desmethylvenlafaxine in the blood, which can lead to variable norepinephrine inhibition. Given this variability, caution must be taken to appropriately titrate venlafaxine, especially if the patient is taking other medications metabolized by

the CYP450 2D6 enzyme. The advent of desvenlafaxine for the treatment of depression and anxiety disorders, also known by its brand name Pristiq, allows metabolism by CYP450 2D6 to be bypassed.

According to the prescribing information, a single oral dose of venlafaxine had 92% absorption with an absolute bioavailability of 45% (Wyeth Pharmaceuticals 2009). Venlafaxine has been marketed in two forms, Effexor immediate release and Effexor XR (extended release). Effexor XR has a slower rate of absorption but has comparable absorption to the immediate release version, offering the benefit of single daily dosing. Subsequently, Effexor XR is the common form prescribed. Absorption is neither affected by food nor time of day of administration. Given that venlafaxine is metabolized in the liver and excreted by the kidney, dosing adjustments should be made in patients with liver and renal impairment, although there is room for adjustment given patient variability in liver metabolism and renal clearance. In liver impairment, the dose should be decreased by as much as 50% or more. In renal impairment, the dose should be decreased from 25% to 50%, whereas in patients receiving hemodialysis, the dose should be decreased as much as 50%. Venlafaxine has a dose-dependent effect on blood pressure, causing sustained hypertension at 300 mg/day or above, and should therefore be used with caution in patients with hypertension or cardiac conditions (Stahl 2009). Dosing adjustments are not required based on gender or age in adults (Wyeth Pharmaceuticals 2009). Steady-state plasma levels of venlafaxine are achieved no less than 4 days after administration or a dose adjustment.

Specific Compounds and Properties

(R/S)-1-[2-(dimethylamino)-1-(4-methoxyphenyl)ethyl] cyclohexanol hydrochloride or (±)-1-[α-[(dimethylamino)methyl]-p-methoxybenzyl] cyclohexanol hydrochloride.

Empirical formula: $C_{17}H_{27}NO_2 \cdot HCl$.

Molecular weight: 313.87.

Clinical Use (Including Side Effects)

FDA-Approved Clinical Uses

According to the official prescribing information, venlafaxine is FDA-approved to treat major depressive disorder (MDD), generalized anxiety disorder (GAD), social anxiety disorder, and panic disorder with or without agoraphobia in adults only (Wyeth Pharmaceuticals 2009). There are currently no FDA-approved uses in children or adolescents. For the treatment of MDD and GAD in adults, Effexor XR is typically started at 75 mg once daily, although some patients may require a starting dose of 37.5 mg/day for 4–7 days before increasing to 75 mg/day. For patients with moderate depression who do not demonstrate improvement in depressive symptoms, Effexor XR can be increased by 75 mg at least every 4 days to a maximum dose of 225 mg/day. In one study examining patients with severe depression, the maximum mean daily dose to achieve symptom control was 350 mg/day, but data exploring the use of Effexor XR above 225 mg/day is limited. According to Stahl, “non-responders at lower doses should try higher doses to be assured the benefits of dual SNRI action,” and up to 600 mg/day has been given (Stahl 2009). Again, there is a possibility for sustained hypertension in doses above 300 mg/day, so prescribers should proceed with caution when considering doses above the recommended dose.

For the treatment of panic disorder in adults, Effexor XR should be started at 37.5 mg/day for 7 days, followed by 75 mg/day for 1 week, and then weekly dosing increases of 75 mg/day to a maximum of 225 mg/day as needed. For the treatment of social anxiety disorder, the daily dose recommendation is 75 mg/day. Switching between Effexor immediate release to Effexor XR can be done with equivalent dosing, although some dosing adjustments may be required based on the clinical response. Effexor immediate release is given in divided daily doses, usually twice or three times daily. It is not clear how long patients should be treated with venlafaxine for maintenance of the aforementioned disorders, so the need for this medication and dosing requirements should be occasionally reassessed. When

discontinuing Effexor XR, the dose should be decreased by 75 mg/day each week, although some patients may require longer taper periods, as long as many months.

According to Stahl, the goal of treatment is complete remission of symptoms and prevention of future relapses, so venlafaxine is taken both throughout and in between clinical relapses (Stahl 2009). Although venlafaxine can eliminate symptoms while being taken, it does not cure depression or anxiety disorders and symptoms can reoccur after the medicine has been stopped. After the first episode of depression in the treatment of adults with MDD, venlafaxine should be taken for 1 year following symptom relief. After the second or any subsequent episodes of depression, treatment with venlafaxine may be indefinite to avoid relapse of symptoms. When treating GAD with venlafaxine, remission for symptoms may be delayed as long as 6 months, requiring adjunctive, short-term treatment of symptom control (Stahl 2008). Once GAD symptoms are controlled, treatment with venlafaxine may be indefinite (Stahl 2009).

Clinical Uses of Venlafaxine in Autism Spectrum Disorders (ASDs)

Researchers have sought various pharmacological treatments to effectively manage symptoms associated with autistic disorder, Asperger’s disorder, and PDD-NOS. Core symptoms include interfering repetitive behaviors, social impairment, and communication impairment, with associated symptoms generally including hyperactivity, inattention, irritability, and aggression. There are currently no randomized, placebo-controlled research studies exploring the use of venlafaxine in treating ASDs, so data supporting its utility in this area is sparse. However, there is one open retrospective clinical report (Hollander et al. 2000) that examined 10 children, adolescents, and young adults (nine males and one female, aged 3–21 years) with the aforementioned ASDs who took venlafaxine for control of core symptoms of ASDs as well as for attention-deficit/hyperactivity disorder (ADHD) symptoms. Using SNRIs for the treatment of symptoms associated with ASDs is based on previous

observations that norepinephrine reuptake inhibitors had been useful in controlling hyperactivity and that some serotonin reuptake inhibitors, like clomipramine, fluoxetine, fluvoxamine, and sertraline, may have some benefit on other symptoms (Cook Jr. et al. 1992; Gordon et al. 1993; McDougale et al. 1996; Steingard et al. 1997). Also, venlafaxine was shown to demonstrate utility in controlling symptoms of ADHD in adults (Findling et al. 1996), which has since been observed in a pediatric population (Findling et al. 2007). The Hollander et al. report used low doses of venlafaxine between 6.25 and 50 mg/day. Six of the ten subjects were considered responders, rated as being “much improved” or “very much improved” on the CGI-I scale. In those patients, all three core dimensions of autism were improved, including repetitive behavior, social relatedness, and communication. Most specifically, the responders demonstrated decreased obsessional thinking, repetitive behaviors, and abnormal vocalizations, as well as improved eye contact, socialization, contextual language use, and complexity of play. Five of the six responders also had improvements in ADHD symptoms. The researchers observed behavioral activation in some patients and warned that this may stem from serotonin reuptake inhibition as evidenced by prior reports (Sanchez et al. 1996). They were reassured that the tendency for venlafaxine to induce clinically significant, dose-dependent hypertension (usually above 300 mg/day) would not affect their subjects as the maximum dose used was 50 mg/day (Thase 1998). This study is limited by the small sample size, the younger ages of the participants, two of the subjects’ concomitant use of other medication, and the retrospective design. Therefore, prescribers should be cautious in generalizing these results to the larger population of patients with ASDs. There have been isolated case reports suggesting the utility of using venlafaxine in the treatment of self-injurious behavior and for ADHD symptoms in patients with ASDs (Carminati et al. 2006), as well as one case report that suggested a concern for increased agitation in a patient with autism and developmental disabilities taking venlafaxine (Marshall et al. 2003). However, there are

currently no randomized, placebo-controlled studies investigating the efficacy of venlafaxine in treating ASDs, so the results of these studies should be regarded with caution before generalizing their utility in treating the greater population of children and adults with ASDs.

Side Effects

Venlafaxine’s side effect profile is a consequence of both serotonin and norepinephrine reuptake blockade on receptors throughout the body (Stahl 2008). Motor activation or tremor can result from acute stimulation by norepinephrine of beta-1 and/or 2 adrenergic receptors in the cerebellum or peripheral sympathetic nervous system. Stimulated cardiac beta-1 receptors can cause changes in heart rate, and changes in blood pressure may result from stimulation of noradrenergic receptors in the brain stem cardiovascular center and descending spinal cord. According to the prescribing information, treatment with Effexor XR above 300 mg/day was associated with sustained hypertension defined as treatment-emergent supine diastolic blood pressure (SDBP) ≥ 90 mmHg and ≥ 10 mmHg above baseline for three consecutive on-therapy visits, although an insufficient number of patients received Effexor XR above 300 mg/day to effectively evaluate this response (Wyeth Pharmaceuticals 2009). Agitation may be experienced from the stimulation of noradrenergic receptors in the amygdala or limbic cortex (Stahl 2008). According to Stahl, there may be “pseudo-anticholinergic” side effects from a reduction of parasympathetic tone, resulting from stimulation of noradrenergic receptors in the sympathetic nervous system (Stahl 2008). In other medications, direct blockade of muscarinic receptors results in classic anticholinergic side effects that include dry mouth, urinary retention, and constipation. SNRIs have no effect on muscarinic receptors, but they do increase sympathetic tone via stimulation of noradrenergic receptors, creating a similar albeit milder effect.

On the other hand, stimulation of serotonin receptor subtypes (5-HT_{2A}, 5-HT_{2C}, 5-HT₃, and 5-HT₄) likely causes other notable side effects. A small amount of increased serotonin shortly

after initiating therapy is often enough to mediate some side effects, even if the clinical benefit is not yet apparent to the patient. Patients who are treated for MDD are at increased risk for experiencing suicidal thinking and behavior. Antidepressants as a class have been shown to increase the risk of suicidal thinking and behavior in children, adolescents, and young adults (ages 18–24) with MDD and other psychiatric disorders (Wyeth Pharmaceuticals 2009). Such risk should be carefully considered when prescribing venlafaxine in the pediatric and young adult population, although venlafaxine is not currently approved for use in populations younger than 18 years. According to Stahl (2009), gastrointestinal side effects can include decreased appetite, nausea, diarrhea, or decreased appetite. Sexual dysfunction can include abnormal ejaculation, abnormal orgasm, or impotence. Central nervous system side effects include rare seizures, headache, nervousness, insomnia, or sedation. Endocrine side effects include syndrome of inappropriate antidiuretic hormone (SIADH) and hyponatremia. Patients with undiagnosed bipolar disorder may be vulnerable to the activating properties of venlafaxine, resulting in hypomania or mania, although this is rare. There has been some reported weight gain in patients taking Effexor, but this is not an expected side effect.

See Also

- Antidepressant Medications
- Anxiolytic Drugs
- Anxiolytics
- Depressive Disorder
- Generalized Anxiety Disorder
- Norepinephrine
- Serotonin
- Serotonin Reuptake Inhibitors (SRIs)

References and Reading

Carminati, G. G., Deriaz, N., et al. (2006). Low-dose venlafaxine in three adolescents and young adults

- with autistic disorder improves self-injurious behavior and attention deficit/hyperactivity disorders (ADHD)-like symptoms. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 30(2), 312–315.
- Cook, E. H., Jr., Rowlett, R., et al. (1992). Fluoxetine treatment of children and adults with autistic disorder and mental retardation. *Journal of the American Academy of Child and Adolescent Psychiatry*, 31(4), 739–745.
- Findling, R. L., Schwartz, M. A., et al. (1996). Venlafaxine in adults with attention-deficit/hyperactivity disorder: An open clinical trial. *The Journal of Clinical Psychiatry*, 57(5), 184–189.
- Findling, R. L., Greenhill, L. L., et al. (2007). Venlafaxine in the treatment of children and adolescents with attention-deficit/hyperactivity disorder. *Journal of Child and Adolescent Psychopharmacology*, 17(4), 433–445.
- Gordon, C. T., State, R. C., et al. (1993). A double-blind comparison of clomipramine, desipramine, and placebo in the treatment of autistic disorder. *Archives of General Psychiatry*, 50(6), 441–447.
- Hollander, E., Kaplan, A., et al. (2000). Venlafaxine in children, adolescents, and young adults with autism spectrum disorders: An open retrospective clinical report. *Journal of Child Neurology*, 15(2), 132–135.
- Marshall, B. L., Napolitano, D. A., et al. (2003). Venlafaxine and increased aggression in a female with autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 42(4), 383–384.
- McDougle, C. J., Naylor, S. T., et al. (1996). A double-blind, placebo-controlled study of fluvoxamine in adults with autistic disorder. *Archives of General Psychiatry*, 53(11), 1001–1008.
- Sanchez, L. E., Campbell, M., et al. (1996). A pilot study of clomipramine in young autistic children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35(4), 537–544.
- Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (Vol. 741, 3rd ed., pp. 474–488). New York: Cambridge University Press.
- Stahl, S. M. (2009). *Stahl's essential psychopharmacology: The prescriber's guide* (3rd ed., pp. 579–584). New York: Cambridge University Press.
- Steingard, R. J., Zimnitsky, B., et al. (1997). Sertraline treatment of transition-associated anxiety and agitation in children with autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, 7(1), 9–15.
- Thase, M. E. (1998). Effects of venlafaxine on blood pressure: A meta-analysis of original data from 3744 depressed patients. *The Journal of Clinical Psychiatry*, 59(10), 502–508.
- Wyeth Pharmaceuticals prescribing information. (2009). Retrieved from <http://labeling.pfizer.com/showlabeling.aspx?id=100>.

Ventricles

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Definition

A ventricle is a chamber or space in the brain which contains cerebrospinal fluid. There are four ventricles in the brain, all of which are contiguous with the central canal of the spinal cord. The ventricles of the brain are the fourth ventricle, the third ventricle, and two lateral ventricles.

See Also

► [Cerebrospinal Fluid](#)

References and Reading

Brambilla, P., Hardan, A., di Nemi, S. U., Perez, J., Soares, J. C., & Barale, F. (2003). Brain anatomy and development in autism: Review of structural MRI studies. *Brain Research Bulletin*, 61, 557–569.

Ventriculography

► [Pneumoencephalography](#)

Verbal Apraxia

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Synonyms

[Apraxia of speech \(AOS\)](#); [Articulatory apraxia \(or dyspraxia\)](#); [Childhood apraxia of speech](#)

[\(CAS\)](#); [Developmental apraxia of speech \(DAS\)](#); [Developmental dyspraxia](#); [Developmental verbal apraxia \(or dyspraxia\)](#); [Dyspraxia](#); [Verbal dyspraxia](#)

Short Description or Definition

Verbal Apraxia (VA) is a speech disorder, or category of speech disorders, in which an individual has difficulty consistently forming and executing motor plans necessary for the articulation and ordering of sounds in words. This difficulty is not due to weakness, slowness, or incoordination of muscles of the mouth and throat, and is frequently attributed to impaired translation of linguistic plans into motor speech patterns. While VA has recently become a topic of interest to providers and families of individuals with communication difficulties, including those with Autism Spectrum Disorders, it has been described and investigated by neurologists, psychologists, and speech pathologists for much longer.

In 1865, Broca first described a similar articulatory language deficit, which he termed aphemia, although this was largely subsumed into the more common term aphasia until the concept of apraxia of speech began to develop in the 1950s and 1960s. The development of VA as a well-defined, discrete entity has been slow, largely because of difficulty establishing whether, indeed, it does resemble other established apraxias (e.g., motor apraxias) wherein automatic function of discrete muscle movements are intact. The problem of assessing discrete elements of speech production outside of speech has dogged efforts to arrive at a standard definition that does not overlap excessively with other disorders.

The concept of a developmental VA was initially proposed by Morley, Court, and Miller in 1954 as a constellation of symptoms similar to those defined in VA in adults that could be used to describe a group of children with speech deficits despite objectively normal development of orofacial and lingual anatomy. The developmental or childhood form of VA becomes manifest during acquisition of language skills, particularly during attempts at complex articulations.

Providers, researchers, and families are interested in determining whether individuals with ASD might have higher rates of DVA, and whether DVA might lead to or exacerbate problems with language development in this group. However, recent evidence suggests that in spite of difficulty with other apraxias, individuals with autism do not show a particularly high rate of DVA, and in fact show speech errors that are easily differentiated from errors shown in children with DVA.

Categorization

Verbal apraxia is variably considered as a discrete entity or disease, a syndrome or symptom cluster, or a category of disorders with more specific subtypes.

It can be classified broadly as developmental, meaning it presents during initial language acquisition, or acquired, meaning it results from later life injury. Within this developmental category, further subdivisions are made on etiological grounds, separating deficits due to known neurological disease from those due to the complex neurobehavioral presentations of genetic or metabolic syndromes, and also from deficits without a known cause, which are referred to as idiopathic.

Just as there is no formal, widely agreed upon definition, there is considerable variety in the terminology and in the classification schemes, and use of particular terms may indicate a provider or researcher's perspective on this controversial diagnosis. For example, the use of "verbal" may indicate emphasis on linguistic deficits over gross or fine motor problems. Likewise, the prefix "dys" often indicates the assumption that the disorder is present from birth, while the prefix "a" often indicates suspicion that the disorder was acquired after birth.

Epidemiology

Incidence of VA is difficult to calculate precisely given the disagreement on its definition and the variety of diagnostic criteria employed. In its

acquired form it is reported in as many as two thirds of patients with aphasia, but rarely reported in isolation. Estimates on the prevalence of the developmental form based on rates of diagnosis in various pediatric clinics indicate population rates between 0.125% and 1.3%. Developmental verbal apraxia (DVA) is more common in children with relatives having communication disorders or learning disabilities. It is diagnosed more frequently in boys, although this could reflect an ascertainment bias if boys are more likely to be referred for speech and language evaluation. Individuals with VA may also have additional apraxia diagnoses, but it is unclear whether these diagnoses are more common in individuals with VA than in individuals without a VA diagnosis.

Natural History, Prognostic Factors, Outcomes

Natural History

Acquired VA most often develops as a consequence of stroke, although it may also be caused by tumors or traumatic brain injuries. It is also described in neurodegenerative diseases, particularly frontotemporal dementias and diseases involving the basal ganglia. It generally occurs with other language deficits (aphasia) or speech deficits (dysarthria).

Children with DVA often present as quiet infants, with parents reporting decreased vocal sounds (e.g., cooing, babbling) compared to unaffected children. It has also been suggested that children with difficulty nursing as infants or "messy eaters" later in development may be exhibiting early signs of VA. Authors who include such children in descriptions of developmental VA report observing a variety of feeding concerns, including frequent choking on food or drink, drooling, or strong food preferences related to oral-motor difficulties. These early symptoms raise the question of whether these children have isolated VA or a concurrent orofacial sensorimotor apraxia.

As they develop, children with DVA tend to acquire and use words and phrases at older ages than their peers. They may develop complex

gestures and non-speech sounds (e.g., grunting) in order to communicate needs with caregivers. Notably, despite their difficulties with productive language, these children often have relatively preserved comprehension of the others’ speech.

Prognostic Factors

Recovery in acquired VA depends on the nature of the causative lesion; fixed insults such as stroke or trauma produce a maximal deficit initially followed by a variable degree of recovery, while expanding lesions such as tumor or degenerative processes often cause progressively worsening symptoms. In addition, prognosis is informed by the age at which the individual acquired the underlying lesion as well as the severity and size of the lesion. Prognostic factors for DVA are less clear, and it is often seen as a disorder that is resistant to treatment. (This may be due to its occasional use as a catch-all diagnosis for those failing to benefit from intervention). However, when progress is seen, it is best accounted for by initial severity of the problem and type and length of services received.

Outcomes

Clinical expression of VA can fluctuate over time, ranging from mild, indicating trouble with only occasional sounds, to severe, in which an individual may not be able to communicate effectively with speech. In acquired apraxia, although

recovery may plateau after 1 year, some improvement may be observed up to 10 years out of an initial insult. While some individuals with DVA may show great improvement with intensive treatment, most individuals will continue to experience symptoms of the disorder throughout the lifetime, although there are scattered, rare reports of complete resolution.

Clinical Expression and Pathophysiology

Verbal apraxia is perhaps more easily recognized than defined, with a characteristic “effortful articulatory groping to produce speech sounds.” Even so, there is great disagreement as to what symptoms characterize DVA, even among practitioners from the same field. The most commonly cited symptoms include difficulty putting together the sounds to form words. In the acquired VA literature, most authors point to a core set of deficits, including highly inconsistent and unpredictable errors (as opposed to the predictable errors of dysarthria due to weakness or spasticity), with primarily phonetic, but also prosodic and non-phonetic features. Many of these same deficits are noted in DVA (Table 1). In acquired VA, reading, writing, and receptive language deficits should be absent, although this distinction may be less clear in individuals with DVA who have yet to acquire written language. Some authors note oral-

Verbal Apraxia, Table 1 Features of verbal apraxia

General symptom	Specific symptom	Example
Difficulty forming sounds	Groping	Unnecessary movements of the lips, tongue, or jaw prior to speaking
	Difficulty with “voiced” consonants	Saying “cap” instead of “cab,” “pat” instead of “pad”
	Leaving out sounds	“Unbelievable” becomes “unbeevable”
	Adding sounds	“Eraser” becomes “eraseler”
	Reversing sounds	Saying “miskate” instead of “mistake”
Difficulty increases with complexity	Difficulty repeating a word	Repeat “buttercup” yields “buttercup, puddercut, buttergup”
	Disappearance of words	Loss of a previously mastered word
	Different day-to-day performance	Severity varies day to day
Odd speech characteristics	Incorrect use of prosody	Using an “angry voice” when feeling happy
	Nasal tone	Child sounds as if they are “talking through the nose”

lingual apraxia (difficulty with complex oral or tongue non-speech movements with intact component movements) should be absent, and still others expand the definition to include those with oral sensory deficits.

Localization of verbal apraxia is difficult, with most authors suggesting problems in the dominant frontal lobe, specifically the inferior frontal gyrus (i.e., Broca's area), which is commonly implicated in the eponymous aphasia. Other regions that have been investigated include the insular cortex, ill-defined subcortical regions, and the parietal lobe. Lesions in these regions are implicated in non-verbal apraxias, and are thought to interrupt connections between motor centers and storage of learned motor engrams. The fundamental problem in VA, as envisioned by Darley and others, is a disruption of the "motor speech programmer," wherein received abstract linguistic plans cannot be translated into the specific sequence of motor movements required for speech. There is no evidence that children with DVA show specific brain lesions or differences in brain structure. However, the concept of a disconnection between linguistic processing and motor planning has been proposed, and multiple specific neuro-linguistic processes implicated, including oral-motor programming, pre-articulatory sequencing, auditory processing, perceptual abilities and their link to memory, the process of selecting and retrieving required sounds, and many others. There is some evidence that DVA is heritable. However, it is difficult to study the genetics of the disorder given disagreement regarding diagnostic criteria.

Clinical expression of DVA in children with ASDs can be difficult to disentangle from general language delay. In addition, children with ASDs, like children with DVA, frequently show difficulty with prosody production. However, speech errors in DVA are marked by their inconsistency (e.g., making different errors from day to day), while they may be more persistent in the speech of an individual with an ASD.

Evaluation and Differential Diagnosis

Evaluation for apraxia of speech is most often undertaken by speech pathologists, who utilize a

variety of skills, checklists, and testing instruments to form their conclusions. As there is no single diagnostic test, repeated visits over time are used to determine if the diagnosis is a good fit. One test used to diagnose DVA is the Motor Speech Evaluation (MSE), which includes assessment of vowel prolongation, sequential and alternating diadochokinesis (e.g., speeded repetition of phonemes), as well as repetition of words of increasing syllable length (e.g., *jab jabber, jabberin*), repetition of sentences, and reading aloud.

Other tools are often used to rule out differential possibilities like aphasia or dysarthria, including the Frenchay test for dysarthria and the Boston Diagnostic Aphasia Exam. In addition, the clinician must exclude developmental delay of speech, in which speech development is slow but otherwise typical. Other common comorbidities include poor vocabulary, incorrect grammar, difficulty in clearly organizing spoken information, problems with reading, writing, spelling, or math, coordination or motor skill problems, and chewing and swallowing difficulties. In individuals where concerns primarily relate to written speech, dyslexia may better account for symptoms.

In addition to the above assessments, many clinicians rely on checklists from seminal papers in the field, and when diagnosing acquired VA, speech pathologists appear to have good intra- and interrater reliability. Most studies suggest that this is not the case for DVA. In a study of 75 Speech Language Pathologists asked to name the most essential symptoms for diagnosis of DVA, 50 different symptoms were named, and none were listed by more than half of all respondents. Despite this variability, a diagnosis of DVA can be generally given if a child's symptoms fit some or all of the descriptive criteria in Table 1, and if the difficulties do not result from paralysis or weakness of the muscles involved in speech.

While children with ASDs and children with DVA both show speech deficits, differential diagnosis can be made based on symptoms specific to each disorder. Child with ASDs will also show marked difficulty or awkwardness during social interactions along with odd or stereotyped behaviors or interests, while these symptoms are not

noted in children with DVA. Children with DVA can be distinguished generally by the presence of inconsistent errors, intact receptive language skills, and typical nonverbal social interactions.

Treatment

There are currently no pharmacological or medical treatments for Developmental or Acquired Verbal Apraxia. Speech therapists instead utilize a variety of neuropsychological and rehabilitative approaches. No one approach has been shown to be most effective, but improvement in symptoms usually requires intensive 1:1 therapy. The most common approaches used to treat both types of VA are focused on linguistic goals, motor-programming goals, or a combination thereof. Therapists focused on linguistics will help the child learn sounds and rules about sounds and sound sequences. This is more commonly utilized with preschoolers and very young elementary-aged children. Other providers focus on motor-programming skills. These involve teaching the child to make sounds and sequences of sounds through repetition. Many therapists use both of methods in treatment. Additional methods include using specific sensory and gestural cuing to facilitate fluent speech. For children with a more severe form of DVA, a therapist might recommend the use of additional communication methods, including sign language, picture or communication notebooks, or an electronic communication device. Finally, the above treatments may be paired with physical therapy, especially for children who have nonverbal motoric deficits. For children and adults with other communication disorders, including ASDs, these goals can be incorporated into speech-language therapy along with goals specific to the comorbid disorder.

See Also

- Aphasia
- Apraxia
- Dysarthria
- Dyslexia
- Dyspraxia

- Phonological Deficits
- Phonological Disorders
- Prosody
- Speech Delay
- Speech Impairments
- Speech Therapy
- Speech-Language Pathologist (SLP)

References and Reading

- American Speech-Language-Hearing Association. (2007). *Childhood apraxia of speech* (Position statement). Retrieved from www.asha.org/policy
- Darley, F. L. (1969). *Aphasia: Input and output disturbances in speech and language processing*. Paper presented at the American Speech and Hearing Association.
- Duffy, J. R. (2006). Apraxia of speech in degenerative neurologic disease. *Aphasiology*, 20, 511–527.
- Ferry, P. C., Hall, S. M., & Hicks, J. L. (1975). Dilapidated speech- developmental verbal dyspraxia. *Developmental Medicine and Child Neurology*, 17, 749–756.
- Forrest, K. (2003). Diagnostic criteria of developmental apraxia of speech used by clinical speech-language pathologists. *American Journal of Speech-Language Pathology*, 12, 376–380.
- Hall, P. K. (2000). A letter to the parent(s) of a child with developmental apraxia of speech, parts I–IV. *Language, Speech, and Hearing Services in Schools*, 31, 169–181.
- Haynes, S. (1985). Developmental apraxia of speech: Symptoms and treatment. In D. F. Johns (Ed.), *Clinical management of neurogenic communication disorders* (2nd ed., pp. 259–266). Boston: Little, Brown and Company.
- McNett, K. T., & Armbruster, A. P. (2008). Acquired disorders of swallowing, cognition, speech and language in the older adult. In J. I. Sirven & B. L. Malamut (Eds.), *Clinical neurology in the older adult* (pp. 127–137). Philadelphia: Wolters Kluwer.
- Morley, M., Court, D., & Miller, H. (1954). Developmental dysarthria. *British Medical Journal*, 1, 463–467.
- Mumby, K., Bowen, A., & Hesketh, A. (2007). Apraxia of speech: How reliable are speech and language therapists' diagnoses? *Clinical Rehabilitation*, 21, 760–767.
- Ogar, J., Willock, S., Baldo, J., Wilkins, D., Ludy, C., & Dronkers, N. (2006). Clinical and anatomical correlates of apraxia of speech. *Brain and Language*, 97, 343–350.
- Shriberg, L. D., Aram, D. M., & Kwiatkowski, J. (1997a). Developmental apraxia of speech. 1. Descriptive and theoretical perspectives. *Journal of Speech Language and Hearing Research*, 40, 273–285.
- Shriberg, L. D., Aram, D. M., & Kwiatkowski, J. (1997b). Developmental apraxia of speech. 2. Toward a diagnostic marker. *Journal of Speech Language and Hearing Research*, 40, 286–312.

- Shriberg, L. D., Aram, D. M., & Kwiatkowski, J. (1997c). Developmental apraxia of speech. 3. A subtype marked by inappropriate stress. *Journal of Speech Language and Hearing Research*, 40, 313–337.
- Shriberg, L. D., Paul, R., Black, L. M., & van Santen, J. P. (2011). The hypothesis of apraxia of speech in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41(14), 405–426.
- Stackhouse, J. (1992). Developmental verbal dyspraxia: (1) A review and critique. *European Journal of Disorders of Communication*, 27, 19–34.
- Stackhouse, J., & Snowling, M. (1992). Developmental verbal dyspraxia: (2) A developmental perspective on 2 case-studies. *European Journal of Disorders of Communication*, 27, 35–54.
- Wertz, R. T. (1985). Neuropathologies of speech and language: An introduction to patient management. In D. F. Johns (Ed.), *Clinical management of neurogenic communication disorders* (2nd ed., pp. 1–96). Boston: Little, Brown and Company.
- Wertz, R. T., Rosenbek, J., & Deal, J. (1970). *A review of 228 cases of apraxia of speech: Classification, etiology, and localization*. Paper presented at the American Speech and Hearing Association Convention.
- Yoss, K. A., & Darley, F. L. (1974). Developmental apraxia of speech in children with defective articulation. *Journal of Speech and Hearing Research*, 17, 399–416.

well. Verbal Auditory Agnosia is a language disorder due to brain dysfunction, similar to aphasia.

Categorization

Individuals with Verbal Auditory Agnosia are unable to recognize that acoustic streams of complex sounds (containing periodic energy) spoken by humans (i.e., the fundamental frequency of the vocal cord vibration) can convey meaning, despite normal auditory sensitivity. As shown in Table 1, they can detect the presence of sound and detect changes in sound whether the changes are verbal or nonverbal. The difficulty occurs at the next level of processing, where individuals are unable to recognize speech as linguistic information, yet they can recognize and name nonverbal sounds (e.g., a dog barking). The next level of auditory processing, comprehension, is also affected for linguistic information such that they have difficulty answering simple questions. However, individuals with Verbal Auditory Agnosia could appropriately associate a siren sound with a plausible scenario in which it might occur, outside of a jail.

Verbal Auditory Agnosia

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Synonyms

[Auditory processing disorder](#); [Auditory verbal agnosia](#); [Cortical deafness](#); [Wernicke's aphasia](#)

Short Description or Definition

Verbal Auditory Agnosia is the inability to recognize linguistic stimuli presented verbally, despite normal hearing and intelligence. In most cases, it is acquired via brain injury, but can be congenital as

Epidemiology

Verbal Auditory Agnosia is considered rare and therefore there are no reports on the prevalence or incidence of this condition. The International Statistical Classification of Diseases and Related Health Problems (ICD-9) only includes a reference to “agnosia” under the code 784.69 “Other,” which also includes acalculia, agraphia, and apraxia. The 784.69 is a subcategory under 784.6 “Other Symbolic Dysfunction.” In the new classification available in 2013 (ICD-10), there will be a code for agnosia, R481, but it will not be specific to verbal auditory agnosia. Of the limited reports in the literature which refer to a complex of symptoms which may include auditory agnosia, only five authors provide information regarding the demographics of individual cases who specifically present Verbal Auditory Agnosia. Of these, there were reports of three

Verbal Auditory Agnosia, Table 1 Hierarchy of auditory processing

	Hierarchy of auditory processing			
	Human sounds		Non-human sounds	
	Periodic	Aperiodic	Periodic	Aperiodic
	"Linguistic"	Non-linguistic	Music	<i>Environ. sounds</i>
<i>Awareness:</i>	Respond to sound by raising hand when sound is heard			
Sample stimulus	"book"	<Cough>	♪ Happy birthday ♪	<i>Siren</i>
Sample response	Raise hand in response to word	Raise hand in response to cough sound	Raise hand in response to music	Raise hand in response to siren sound
VAA Ability?	Yes	Yes	Yes	Yes
<i>Discrimination:</i>	Detect a change in sound by pointing to the one that is different			
Sample stimulus	"book-shoe-book"	<Cough-laugh-cough>	♪HB♪ ♪RRR♪ ♪HB♪	<i>Siren – dog bark -siren</i>
Sample response	Point to #2	Point to #2	Point to #2	Point to #2
VAA ability	Yes	Yes	Yes	Yes
<i>Identification:</i>	Associate sound with a label by pointing to a corresponding picture/object			
Sample stimulus	"book"	<Coughing>	♪ Happy Birthday ♪	<i>Siren</i>
Sample response	Point to picture of book	Point to picture of someone with hand over his mouth	Point to picture of a birthday cake	Point to picture of a police car
VAA ability	No	Yes	Yes	Yes
<i>Comprehension:</i>	Associate sound with past experiences by selecting a related but not actual picture			
Sample stimulus	"Which one do we read?"	<Coughing>	♪ Happy birthday ♪	<i>Siren</i>
Sample response	Point to picture of book	Point to picture of a bottle of medicine	Point to a picture of people at party	Point to a picture of a jail
VAA ability	No	Yes	Yes	Yes

Note: VAA Verbal Auditory Agnosia, Environ Environmental; ♪HB♪ ♪RRR♪ ♪HB♪ Happy Birthday, Row Row Row your Boat, Happy Birthday

females, ages 7, 19, and 40 years, and two males, ages 7 and 51 years. The onset varied and was suggestive of being an acquired condition such as stroke (two cases) or secondary to a congenital condition such as a syndrome or developmental disorder (three cases).

Natural History, Prognostic Factors, Outcomes

The earliest report of someone with Verbal Auditory Agnosia was in 1930. It is considered a rare condition that someone with normal hearing would have such difficulty with recognition of words, yet be capable of recognizing environmental sounds. The prognosis is generally better than that for someone with Wernicke's aphasia, which

also affects the temporal lobe, because individuals with Verbal Auditory Agnosia still have relatively good spoken language skills. Furthermore, the receptive difficulty is limited to the auditory domain so that other domains such as vision, which may be stronger, may be used to facilitate communication and learning through written language. Depending upon the age of onset if caused by a cerebral insult, it can impact the degree to which the production of speech is affected. For example, if the Verbal Auditory Agnosia occurs during the early years of acquiring speech, the acoustic patterns or templates, upon which the individual's own speech is compared, may not develop. Hence, the auditory/motor coordination for speech production is impaired. In contrast, if the Verbal Auditory Agnosia onset occurs after the development of fully intelligible speech, the

general prognosis remains high because more compensatory strategies may be developed based on the strength of the production skills.

Clinical Expression and Pathophysiology

While the cause of Verbal Auditory Agnosia is unknown, research shows that those with the condition have sustained damage to the left temporal lobe. This region of the brain contains Wernicke's area, where speech is processed and meaning is derived from the spoken word. Receptive language deficits can almost always be linked to an infarct or abnormality in Wernicke's area.

Individuals with Verbal Auditory Agnosia are unable to recognize that acoustic streams of complex sounds containing periodic energy, (i.e., the fundamental frequency of the vocal cord vibration) can convey meaning despite normal auditory sensitivity. This disorder may be associated with Landau-Kleffner syndrome, in which an individual also suffers from convulsions and seizures. These individuals may gradually lose their ability to speak along with their impaired ability to discriminate speech sounds spoken by others.

Evaluation and Differential Diagnosis

An appropriate evaluation for Verbal Auditory Agnosia includes four steps: (a) evaluate sound awareness (b) evaluate sound discrimination (c) evaluate auditory identification/recognition (d) evaluate auditory comprehension.

The first step in the evaluation process is to determine that hearing sensitivity is within normal limits. If a standard audiometric evaluation cannot be completed, electrophysiological techniques, such as measurement of electrical responses to sound at the brainstem, Auditory Brainstem Response (ABR), should be used. Hearing sensitivity tests are aimed at evaluation at the simplest level of processing, awareness, and are known as speech awareness or detection thresholds. A response may range from eye widening in an infant to raising one's hand to indicate detection. Although auditory discrimination, the next level

of auditory processing, can be evaluated in many ways, there are no tests of discrimination routinely used in the audiological battery. The evaluation of discrimination, or one's ability to detect a change in the acoustic stimulus, is accomplished in young infants by using a dishabituation response recorded as a change in sucking or heart rate. In toddlers, the dishabituation response may be a head turn. When paired with appropriate visual training, children's speech discrimination may be tested with the oddity paradigm as young as 4 years of age. In the oddity paradigm, three stimuli are presented, two of which are acoustically identical. The child's task is to identify which of the three stimuli was different. By age 5 or 6, a same/different paradigm is often used to evaluate discrimination. For adults, discrimination of speech features has most often been measured using an ABX procedure in which the listener determines if the first (A) or second (B) stimulus is the same as the last one (X). In all of these procedures, the patient is required to respond only when a change has been perceived in some acoustic dimension of the stimulus, such as the intensity, frequency, or timing. Therefore, when assessing auditory discrimination, the patient is merely comparing stimuli. One commercially available tests of discrimination include the Goldman-Fristoe-Woodcock Test of Auditory Discrimination (1970), which has norms for ages 3:8 to 70+ years.

The next level, auditory identification/recognition, may be evaluated by asking the listener to choose an object, point to a picture, or produce the stimulus in written or verbal form. Individuals must recognize the sequence of sounds and then associate it with some previous experience. Recognition tasks differ from the next level of processing in that no semantic processing is required. That is, the patient may be able to imitate the word "book" but not be able to define it or use it in a sentence. Typical audiological speech assessments at the recognition level of processing include the speech recognition threshold and the word/sentence recognition score. It is important to note that audiologists typically perform recognition tests as a function of intensity level. A recognition test is usually administered at the threshold level and at

one or more supra-threshold levels. It is becoming increasingly common to also include another recognition test at a supra-threshold level in the presence of background noise.

Auditory comprehension, the final level of auditory processing, is not routinely assessed during the clinical audiological battery. Following simple commands or answering “wh-” questions at a fixed intensity level in a sound booth removes the possibility of errors resulting from distractions or intensity variations with live-voice presentation.

In addition to the audiological battery, age-appropriate language tests may be used. The individual with VAA will have difficulty with any task that requires identification and/or comprehension of verbal stimuli. There is currently no one test that will allow assessment of identification of nonspeech sounds in the environment such as horns, animals, or music in contrast to the identification and comprehension of words and/or phrases. The ability to identify nonspeech sounds combined with an inability to identify words/phrases are the classic symptoms of one with Verbal Auditory Agnosia.

Treatment

There has been limited research on the treatment methods for Verbal Auditory Agnosia because there have been so few cases reported. Most of the literature has been focused on the differential diagnosis of these individuals using techniques such as cortical event-related potentials, auditory brainstem responses, magnetic resonance imaging, SPECT imaging, and cranial computed tomography. Treatment methods vary for Verbal Auditory Agnosia based on individual severity. Strategies used to treat auditory processing disorder (APD) might also be effective because they are designed to strengthen processing via the auditory channel. Individuals with this diagnosis present with a delay in cortical components, suggesting they need more time to process speech. Therapy strategies include:

- Giving frequent breaks in listening time
- Limiting background noises in the environment

- Providing more pauses in speech stimuli to accommodate slower processing speeds
- Computer-based auditory training exercises
- Assistive listening devices such as FM systems

There are no standard psychopharmacological treatments for VAA; however, there has been one report of using diazepam with one individual who presented symptoms of VAA (Nagafuchi et al. 1993). The 7-year-old participant with VAA was injected with 10 mg of diazepam, which was followed by a daily dose of 2 mg for 2 years. The individual with VAA returned to within normal limits on auditory discrimination tasks involving monosyllabic words. This study has not been replicated, so its effect on all individuals with VAA is unknown.

Another approach is to capitalize on one's strengths in other areas of functioning to increase verbal processing. For example, if an individual with VAA is a strong reader, communication strategies that involve the printed words would be effective. A progressive approach of pairing the auditory label with the printed word to convey meaning may be tried to bridge associations between the linguistic concepts that may ultimately transfer to the auditory domain. Computerized auditory training programs also may be used to increase independent efforts to improve auditory processing.

See Also

- Aphasia
- Auditory Processing Disorder
- Auditory Verbal Agnosia
- Cortical Deafness
- Wernicke's Aphasia

References and Reading

- American Speech-Language-Hearing Association. (2005). *(Central) Auditory processing disorders* (Technical report). Retrieved from www.asha.org/policy
- Carmona, C., Casado, I., Fernández-Rojas, J., Garín, J., & Rayo, J. I. (1995). Verbal auditory agnosia: SPECT

- study of the brain. *Revista de Neurologia*, 23(123), 1047–1050.
- Cooper, J., & Ferry, P. (1978). Acquired auditory verbal agnosia and seizures in childhood. *The Journal of Speech and Hearing Disorders*, 43(2), 176–184.
- Duran, M. H. C., Guimarães, C. A., Medeiros, L. L., & Guerreiro, M. M. (2009). Landau-Kleffner syndrome: Long-term follow-up. *Brain & Development*, 31(1), 58–63. <https://doi.org/10.1016/j.braindev.2008.09.007>.
- Goldman, R., Fristoe, M., & Woodcock, R. (1974). *G-F-W diagnostic auditory discrimination test*. Circle Pines: American Guidance Service.
- Goldstein, M. N. (1974). Auditory agnosia for speech (“Pure word deafness”): A historical review with current implications. *Brain and Language*, 1, 195–204.
- Guerreiro, M. M., Camargo, E. E., Kato, M., Menezes Netto, J. R., Silva, E. A., Scotoni, A. E., et al. (1996). Brain single photon emission computed tomography imaging in Landau-Kleffner syndrome. *Epilepsia*, 37(1), 60–67.
- Kaga, M., Kon, K., Uno, A., Horiguchi, T., Yoneyama, H., & Inagaki, M. (2002). Auditory perception in auditory neuropathy: Clinical similarity with auditory verbal agnosia. *Brain & Development*, 24(3), 197–202.
- Klein, S. K., Kurtzberg, D., Brattson, A., Kreuzer, J. A., Stapells, D. R., Dunn, M. A., et al. (1995). Electrophysiologic manifestations of impaired temporal lobe auditory processing in verbal auditory agnosia. *Brain and Language*, 51(3), 383–405. <https://doi.org/10.1006/brln.1995.1067>.
- Klein, S., Tuchman, R., & Rapin, I. (2000). The influence of premorbid language skills and behavior on language recovery in children with verbal auditory agnosia. *Journal of Child Neurology*, 15(1), 36–43.
- Nagafuchi, M., Iinuma, K., Yamamoto, K., & Kitahara, T. (1993). Diazepam therapy of verbal auditory agnosia. *Brain and Language*, 45, 180–188.
- Papathanasiou, I., Macfarlane, S., & Heron, C. (1998). A case of verbal auditory agnosia: Missing the word ... missing the sound. ... *International Journal of Language & Communication Disorders/Royal College of Speech & Language Therapists*, 33, 214–217.
- Shoumaker, R., Ajax, E., & Schenkenberg, T. (1977). Pure word deafness (Auditory verbal agnosia). *Diseases of the Nervous System*, 38(4), 293–299.
- Thibodeau, L. (2006). Computer based auditory training for children with auditory processing disorders. In G. Chermak & F. Musiek (Eds.), *Handbook of (central) auditory processing disorder: Vol. 2: Comprehensive Intervention*. San Diego: Plural.
- Wang, E., Peach, R. K., Xu, Y., Schneek, M., & Manry, C. (2000). Perception of dynamic acoustic patterns by an individual with unilateral verbal auditory agnosia. *Brain and Language*, 73(3), 442–455. <https://doi.org/10.1006/brln.2000.2319>.
- Zhu, R., Lv, Z., Shan, C., Xu, M., & Luo, B. (2010). Pure word deafness associated with extrapontine myelinolysis. *Journal of Zhejiang University Science B*, 11(11), 842–847.

Verbal Behavior

► Skinnerian Categories of Verbal Behavior

Verbal Behavior Assessment

► Verbal Behavior Milestones Assessment and Placement Program (VB-MAPP)

Verbal Behavior Interventions

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Definition

Verbal Behavior, Verbal Behavior intervention, or Applied Verbal Behavior (AVB) is an application of Applied Behavior Analysis (ABA) therapy that incorporates B. F. Skinner’s 1957 functional analysis of the acquisition of language in individuals. Skinner’s book only describes verbal behavior and does not specifically describe how to apply verbal behavior in an intervention setting. The primary focus in Verbal Behavior intervention is to identify deficits in an individual’s functional language and skills to determine how to best capture a child’s motivation. This motivation helps to pair the value of a word and the word itself; this is done through behavior training. Verbal Behavior intervention used with individuals diagnosed with Autism Spectrum Disorders (ASD) often works on mastering four distinct components of Skinner’s verbal behavior (i.e., mands, tacts, echoics, and intraverbals). Additional verbal intervention can be completed on receptive skills (in the areas of

function, feature, classification), textual, written (dictation), and imitation.

Historical Background

In 1957, Skinner wrote *Verbal Behavior*. In his book, Skinner argued that communication is a form of behavior that follows the same principles as other forms of behavior. Furthermore, Skinner stated that verbal behavior is subject to the same controlling variables as all other voluntary behavior. However, verbal behavior is indirect and mediated by the behavior of other people and nonverbal behavior is direct and mediated by the environment. Because of this specific distinction, Skinner believed that verbal behavior required a separate functional analysis.

Since the introduction of Skinner's theory of verbal behavior in Applied Behavior Analysis (ABA) therapy with individuals with ASD, the incorporation of this form of intervention has grown in its popularity as successful behavioral programs for individuals with ASD possess verbal behavior targets (i.e., communication) as well as social behavior targets, motor targets, and self-help targets. Skinner's original theory has not changed, but the implementation of Verbal Behavior intervention continues to progress as more is learned about how children, both with and without ASD, develop language.

Rationale or Underlying Theory

According to Skinner, verbal behavior can be grouped into four distinct units or operants as he identified them. The four operants are: (1) mands, (2) tacts, (3) echoics, and (4) intraverbals. Each operant serves a different and specific function in the acquisition of verbal behavior and have been incorporated into Verbal Behavior intervention for individuals with an ASD diagnosis.

Mands are verbal requests for something that an individual desires or wants. It is believed that if individuals with an ASD diagnosis do not learn mands, they are not able to control their environment and social settings. It is theorized that

learning mands makes the acquisition of echoics and tacts easier for the individual. An example of a mand is when a child is learning to say the word "cookie" for requesting a cookie. The word itself is reinforced when the child is given the cookie after successfully saying the word "cookie" and, according to Skinner's verbal behavior theory, the child will use the word "cookie" again when the same context is presented to the child.

Tact is the ability to label an object. Tacts are elicited by the use of a specific object. For example, if a child sees a picture of a dog and says the word "dog," the child is labeling the item (i.e., the picture) with a specific word (i.e., the word "dog"). A pure tact is when labeling occurs naturally whereas an impure tact is when labeling occurs through manipulation.

Echoic is the ability to imitate natural sounds. Echoic builds on the principle that most people learn by imitation. Reinforcement of echoic responses will increase the likelihood of future echoic responses to occur in an individual without prompting. In Verbal Behavior intervention, echoics include vocal imitation as well as sign language. It is believed that if a child is able to echo the word, the child will be motivated to use the same word again in the future in order to obtain the same object.

Intraverbal is a prerequisite for more advanced verbal skills like conversation or social language. It is responding to another individual's verbal response and can be motivated by social reinforcement. Intraverbals can be simple conversation, categorizations, or verbal responses to another person's verbal behavior; they are an essential goal of Verbal Behavior intervention. Intraverbal responses to the language of another person are usually in response to "Wh-" question (i.e., "Who?" "What?" "Where?" "When?" "Why?"). An example of an intraverbal is if a parent asks a child, "Where do you go swimming?" and the child responds, "Pool." An example of an intraverbal fill-in is if a parent asks a child, "You go swimming in the . . ." and the child completes the sentence "Pool." Intraverbals allow individuals to discuss stimuli that are not present and without the presence of a visual cue prompt.

Verbal Behavior intervention teaches mands, tacts, echoics, and intraverbals in such a manner that there is very specific and systematic reinforcement for each specific verbal response. In Skinner's functional analysis of language, he proposed that language is a behavior that is caused by environmental variables such as reinforcement, motivation, extinction, and punishment. When intervention systematically manipulates these variables, therapists are able to increase motivation for a client to engage in a targeted verbal response (vocal or nonvocal).

Goals and Objectives

The primary goal of Verbal Behavior intervention is to promote language development in any individual with language deficits. Verbal Behavior intervention aims to capture the motivation of an individual to pair the value and function of a word and the word itself. The understanding, acquisition, and production of verbal behavior from the targeted individual means vocal behavior, but also includes sign language and the use of augmented speaking devices. Sign language and augmented speaking devices are extremely helpful while teaching mands, tacts, and even intraverbals. Through a behavioral therapist, the individual learns the relationship between language use and requests to obtain desired items or activities.

Some examples of objectives from a Verbal Behavior intervention session are to increase the frequency of independent requests for toileting (i.e., mand for "toilet") and to increase the frequency of independent requests for a break during a session, help during a session, or stop the session. These mands are important for communication because they provide immediate access to toileting or to a break. Additionally, these mands are positive replacement behaviors because the use of them will produce immediate access to toileting when needed or a break from a non-preferred activity. Another example of an individual's Verbal Behavior intervention session would be the use of mands for information. Once the mands for that individual are successful

for basic needs and in high establishing operations/motivating operations (or EO/MO) situations, the therapist may then work to create situations where requests for information help the individual gain access to more motivating items. For example, if a child wants to color but needs crayons, the therapist will teach the mand for crayons or the location of crayons when crayons are not present.

Treatment Participants

Verbal Behavior intervention is used with children who have deficits in language and may be applied to individuals with or without ASD. Although it may be effective in teaching some communication skills to children with ASD, additional research is still needed to determine if the procedures in Verbal Behavior interventions are effective for teaching complex, flexible, and generalized verbal skills for individuals with ASD.

Mands need to be taught first for children diagnosed with ASD. Individuals are then taught tacts and the different function between a mand and a tact. That is, individuals must understand the difference between requesting desired objects (mand) and labeling objects (tact). After these concepts are learned, individuals with ASD are then taught intraverbals. This is perhaps the most important verbal behavior component of the intervention as individuals learn to answer various "Wh-" questions. Echoics are also an important part of the intervention for individuals diagnosed with ASD. Echoic learning in the natural environment is critical to the generalization of learned skills and imitation and implementation of social language in the natural environment.

All individuals diagnosed with ASD with a language deficit can benefit from Verbal Behavior intervention. Verbal Behavior intervention provides continual reinforcement and opportunities to continue increasing the fluency of mands, tacts, echoics, and intraverbals. Additionally, the skills that are being taught in Verbal Behavior intervention are key components of social and communication behavior.

Treatment Procedures

The structure of Verbal Behavior intervention is based on the format of ABA therapy, with a therapist working directly with a child in a structured setting with predetermined individualized goals. Verbal Behavior intervention is designed to motivate a child to learn language by finding a pairing between a word and the value of the word. Verbal Behavior intervention also emphasizes the function of words and language instead of the form. In Verbal Behavior intervention, more importance is placed on mands than tacts stemming from the belief that the use of language is more important than just the knowledge of language.

The format of a discrete trial is in the order of stimulus-response-consequence. Sessions contain a combination of verbal operants with the opportunity of the mand. Verbal request for a desired item or activity is the targeted response. Mands are first taught to the individual. The intervention should be structured in a formal training session and in the child's natural environment so that verbal and social interactions may be generalized and practiced with peers, parents, and others outside of the formal therapy environment. The therapist should make the intervention as fun as possible for the child. This is done by using frequent mand trials, varying the pace of the sessions, the tone of voice of the therapist, using errorless learning procedures instead of using the word "no," and minimizing the use of punishments. Verbal Behavior intervention generally involves 30 or more h per week of scheduled therapy. Families are highly encouraged to use the skills learned in the structured sessions in their daily lives.

Verbal Behavior intervention uses reinforcers at the beginning of therapy with an individual, and eventually fades them out of the therapy to a more natural reinforcement schedule. However, fading is not done with mands as the reinforcer for the targeted behavior is not praise but the requested item. Reinforcers are functionally related to the type of response the child is producing. Reinforcers, such as food and toys, are then faded to natural reinforcers that maintain the specific type of response elicited from the child.

Efficacy Information

To date, research studies on the efficacy of the use of Verbal Behavior intervention to increase vocalizations in nonverbal children are inconclusive. Inconclusive empirically supported research findings on the efficacy of Verbal Behavior intervention stem from several issues. First, it is challenging for a research study to appropriately separate the exact component of Verbal Behavior intervention that contributes to the verbal gains seen in the participants (Bondy et al. 2010). Second, a majority of the research looking at the potential benefits of Verbal Behavior intervention in individuals, with and without language deficits, focus on interventions targeting a single operant (Carr and Firth 2005), thus making findings not applicable to Verbal Behavior intervention as a whole. The lack of outcome studies, especially in the ASD literature, continues to contribute to the lack of evidence-based support for Verbal Behavior intervention and is primarily due to the complexities of measuring and replicating intervention studies. However, research findings do exist on the independent benefits of mands, tacts, and intraverbals (Sautter and LeBlanc 2006). Each operant has been proven to provide verbal gains in individuals with language deficits, both with and without ASD.

Further research is needed to determine whether the approaches used in Verbal Behavior intervention are in fact unique to ABA therapy or if they are simply another way to label tools and skills already used in conventional ABA therapy. Additional research is also needed to test the overall effectiveness of the use of curricula based on Skinner's analysis of verbal behavior as well as outcome studies with larger sample sizes.

Outcome Measurement

The Verbal Behavior Milestones Assessment and Placement Program (VB-MAPP) is a criterion-referenced assessment tool, curriculum guide, and skills tracking system that is designed for children with ASD and other individuals who

demonstrate language deficits. The VB-MAPP is comprised of five components that collectively provide: (a) a baseline level of performance (the VB-MAPP Milestones Assessment), (b) a direction for intervention (the VB-MAPP Barrier Assessment), (c) a system for tracking skills acquisition (the VB-MAPP Transition Assessment), (d) a framework for curriculum development (the VB-MAPP Task Analysis and Skills Tracking), and (e) a guide for placement recommendations (the VB-MAPP Placement and IEP Goals).

Qualifications of Treatment Providers

Verbal Behavior intervention is conducted by Board Certified Behavioral Analysts (BCBA), psychologists, speech therapists, special education teachers, and other professionals who have been trained in ABA therapy and BCBA certified. Professionals who are not BCBA certified, such as Board Certified Assistant Behavioral Analysts (BCaBA), may conduct Verbal Behavior intervention under the direct supervision of a certified BCBA.

To apply for BCBA certification, an individual must have at least a Masters level degree in a human service field, such as Behavior Analysis, psychology, or special education. Individuals must also have completed coursework in behavior analysis that cover ethical considerations, behavioral assessment, experimental evaluation of interventions, measuring and interpreting behavioral data, and behavioral change procedures. An individual must also have obtained supervised training from a certified BCBA. An application needs to be completed and approved. The individual must also take and pass a standardized examination. An individual may also obtain BCBA certification if he/she has at least 1 year faculty appointment at a university or college where he/she must have (1) taught a class on the principles of behavior, single-subject research methods, applications of basic principles of behavior in applied settings, and ethical issues and (2) conducted and published research on behavior analysis. An individual may also apply for BCBA certification if he/she has a doctoral degree for at

least 10 years in behavior analysis, psychology, education, or another related field approved by the Behavioral Analyst Certification Board (BACB).

To obtain BCBA certification, an individual must then pass a standardized exam. The BCBA exam consists of 150 multiple-choice questions and the BCaBA exam consists of 132 multiple-choice questions. Both exams cover 10 content areas: (1) ethical considerations; (2) definition and characteristics; (3) principles, processes, and concepts; (4) behavioral assessment; (5) experimental evaluation of interventions; (6) measurement of behavior; (7) displaying and interpreting behavioral data; (8) selecting intervention outcomes and strategies; (9) behavior change procedures; and (10) systems support. Examiners are given 4 h to complete the exam. The exams are administered on computers and are offered three times a year in 1-month testing windows.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Behavior Analysis](#)
- [Behavior Analyst Certification Board](#)
- [Behaviorism](#)
- [Mands](#)
- [Skinnerian Categories of Verbal Behavior](#)

References and Reading

- Barbera, M. L., & Rasmussen, T. (2007). *The verbal behavior approach: How to teach children with autism and related disorders*. London: Jessica Kingsley.
- Bondy, A., Esch, B. E., Esch, J., & Sundberg, J. (2010). Questions on verbal behavior and its application to individuals with autism: An interview with the experts. *Behavior Analyst Today*, 11, 186–205.
- Carr, J. E., & Firth, A. M. (2005). The verbal behavior approach to early and intensive behavioral intervention for autism: A call for additional empirical support. *Journal of Early and Intensive Behavioral Intervention*, 2, 18–27.
- Drash, P. W., High, R. L., & Tudor, R. M. (1999). Using mand training to establish an echoic repertoire in young children with autism. *The Analysis of Verbal Behavior*, 16, 29–44.
- Fisher, W. W., Piazza, C. C., & Roane, H. S. (2011). *Handbook of applied behavior analysis*. New York: Guilford Press.

- Kates-McElrath, K., & Axelrod, S. (2006). Behavioral intervention for autism: A distinction between two behavior analytic approaches. *The Behavior Analyst Today*, 7, 242–252.
- Koegel, L. K. (2000). Interventions to facilitate communication in autism. *Journal of Autism and Developmental Disorders*, 30, 383–391.
- Koegel, L. K., Koegel, R. L., Green-Hopkins, I., & Carter Barnest, C. (2010). Brief report: Question-asking and collateral language acquisition in children with autism. *Journal of Autism and Developmental Disorders*, 40, 509–515.
- Koegel, R. L., Shirotova, L., & Koegel, L. K. (2009). Brief report: Using individualized orienting cues to facilitate first-word acquisition in non-responders with autism. *Journal of Autism and Developmental Disorders*, 39, 1587–1592.
- Lord, C. (2000). Communication: Achievements and future directions for intervention research in communication and autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 30, 393–398.
- Lovaas, O. I. (1977). *The autistic child: Language development through behavior modification*. New York: Irvington Publishers.
- Lovaas, O. I., Koegel, R., Simmons, J. Q., & Stevens, J. (1973). Some generalization and follow-up measures on autistic children in behavior therapy. *Journal of Applied Behavior Analysis*, 6, 131–165.
- Miguel, C. F., Petursdottir, A. I., & Carr, J. E. (2005). The effects of multiple-tact and receptive-discrimination timing on the acquisition of intraverbal behavior. *The Analysis of Verbal Behavior*, 21, 27–41.
- Partington, J. W., & Sundberg, M. L. (1998). *The assessment of basic language and learning skills (the ABLLS)*. Pleasant Hill: Behavior Analysts.
- Sautter, R. A., & LeBlanc, L. A. (2006). Empirical applications of Skinner's analysis of verbal behavior with humans. *The Analysis of Verbal Behavior*, 22, 35–48.
- Sigafoos, J., Roberts, D., Kerr, M., Couzens, D., & Kerr, M. (1994). Opportunities for communication in classrooms serving children with developmental disabilities. *Journal of Autism and Developmental Disorders*, 24, 259–279.
- Skinner, B. F. (1957). *Verbal behavior*. Acton: Copley.
- Sundberg, M. L. (1998). Realizing the potential of Skinner's analysis of verbal behavior. *The Analysis of Verbal Behavior*, 15, 143–147.
- Sundberg, M. L., & Michael, J. L. (2001). The benefits of Skinner's analysis of verbal behavior for children with autism. *Behavior Modification*, 25, 698–724.
- Sundberg, M. L., & Partington, J. W. (1998). *Teaching language to children with autism or other developmental disabilities*. Pleasant Hill: Behavior Analysts.
- Yamamoto, J. (1994). Functional analysis of verbal behavior in handicapped children. In S. C. Hayes, L. J. Hayes, M. Masaya, & K. Ono (Eds.), *Behavior analysis of language and cognition* (pp. 107–122). Reno: Context Press.

Verbal Behavior Milestones Assessment and Placement Program (VB-MAPP)

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Synonyms

Criterion-referenced assessment; VB-MAPP; Verbal behavior assessment

Description

The Verbal Behavior Milestones Assessment and Placement Program (VB-MAPP), developed by Mark L. Sundberg, Ph.D., BCBA-D, is a criterion-referenced assessment that provides individualized information regarding a learner's current level of responding across a variety of verbal and learning behavior skills that typically emerge by age 48 months. Within the VB-MAPP is the Early Echoic Skills Assessment (EESA) subtest, developed by Barbara E. Esch, Ph.D., BCBA-D, CCC-SLP, which is used in combination with the VB-MAPP. The information gained through the completion of the VB-MAPP may be used to drive academic and behavioral programming including the development of goals and objectives (Sundberg 2014).

The *VB-MAPP Protocol*. The *VB-MAPP Protocol* is the testing booklet used for administration of the assessment. One protocol booklet should be used per learner, and each protocol booklet may be used for up to four administrations of the VB-MAPP. The *VB-MAPP Protocol* contains the VB-MAPP Milestones Assessment, Early Echoic Skills Assessment (ESSA), VB-MAPP Barriers Assessment, VB-MAPP Transition Assessment, and a Skills Tracking Chart for the VB-MAPP Task Analysis and Supporting Skills section (Sundberg 2008).

Milestones Assessment. The Milestones Assessment determines the baseline levels for

170 skills across 16 developmental domains: mand; tact; listener behavior; visual perceptual/match-to-sample (VP/MTS); play; social; imitation; echoic skills; vocal behavior; listener responding by feature, function, and class (LRFFC); intraverbals; group; linguistics; reading; writing; and math (Sundberg 2008). The domains are grouped into three levels based upon typical skills observed for a learner within the specified developmental age range (Level 1: 0–18 months, Level 2: 18–30 months, and Level 3: 30–48 months). The age ranges are available to easily group the skills into developmental ranges and should not be used to provide a developmental age-equivalency score. The Milestones Assessment involves either direct testing, observation, either testing or observation, or timed observation of the assessed skill. The assessment method for each individual skill is stated on the scoring page next to each individual skill, and a comments/notes section is provided below the skill domain area. Each individual skill receives a score of 0 (did not demonstrate skill), 1/2 (partially demonstrated skill), or 1 (demonstrated skill). The score is placed in the grid to the left of the skill description labelled 1st, 2nd, 3rd, and 4th, which represents the current testing administration number. The sum of all five skills per domain is transferred to the corresponding testing administration grid located above the skill domain area. Within the Milestones Assessment is the EESA subtest. The EESA assesses as learner's echoic skills (i.e., vocal imitation) across five separate groups including simple and reduplicated syllables, two-syllable combinations, three-syllable combinations, prosody within spoken phrases (e.g., emphasize syllables), and prosody across other contexts (e.g., pitch, volume, and duration; Sundberg 2008).

Barriers Assessment. The Barriers Assessment determines the learner's baseline levels across 24 areas that commonly contribute to decreased language development and reduced learning rates (Sundberg 2008). Examples of common barriers include prompt dependency, failure to generalize, reinforcement dependency, and obsessive-compulsive behavior. Each barrier is scored on a 0–4 Likert scale ranging from 0 to 4; a low score

indicates that the assessed barrier does not impact the learner, and a high score indicates a severe barrier for the learner (Sundberg 2008).

Transition Assessment. The Transition Assessment determines the effectiveness of the learners current programming and whether the learner has the prerequisite skills needed to learn in a more naturalistic arrangement (Sundberg 2014). The Transition Assessment provides information that may lead to a better understanding of the appropriate learning format and educational setting for the learner. Similar to the Barriers Assessment, each skill has an associated grid to document the score. Scores in the Transition Assessment range from 1 to 5; a low score indicates a more intensive and individualized instructional setting which may be more appropriate for the learner, and a high score indicates that the learner may learn most effectively in a more naturalistic educational environment. The transition skills are grouped into three larger categories: VB-MAPP Scores and Academic Independence, Learning Patterns, and Self-Help, Spontaneity, and Self-Direction. Examples of assessed skills include *works independently on academic tasks*, *retention of new skills*, and *adaptability to change* (Sundberg 2008).

Task Analysis and Supporting Skills Tracking Guide. The Task Analysis and Supporting Skills Tracking Guide component included with the *VB-MAPP Protocol* includes identified prerequisite and supporting skills to encourage further development for milestone skills across the Milestones Assessment (Sundberg 2014). Each milestone skill is listed with suggested prerequisite and supporting skills. Supporting skills are indicated with an asterisk, whereas prerequisite skills do not have a symbol. An example of a prerequisite skill is that the learner should “put 2 similar items together 2 times” prior to demonstrating the milestone skill of “matches any 10 identical items” (Sundberg 2008, pp. 41). An example of a supporting skill is that the learner “completes a 3-piece inset puzzle without physical prompts” (Sundberg 2008, pp. 41). When a prerequisite or supporting skill meets mastery, the testing administrator should indicate that the objective is met by placing a mark in the corresponding box. Items

that indicate a prerequisite or supporting skill met mastery should be transferred to the Skills Tracking Chart which provides the assessor with a visual reference of the learner's progress (Sundberg 2008).

The VB-MAPP Guide. The *VB-MAPP Guide* is the user manual for the VB-MAPP assessment. It provides background information on the overall assessment; a brief introduction to B. F. Skinner's *Verbal Behavior* (1957); assessment tool administration guidelines; scoring instructions for the Milestones, Barriers, and Transition Assessments; and a guide for program development including suggested goals and objectives for Individualized Educational Plans (Sundberg 2014). An introductory review of verbal behavior is provided in the *VB-MAPP Guide* to orient the administrator to the necessary concepts of verbal behavior that are required to complete the assessment. The administration guidelines describe general strategies to increase the effectiveness of the assessment process. The scoring instructions provide additional information regarding the skill being assessed such as appropriate sample materials, an example of how to present the skill, and specific criteria for scoring. Suggested learning objectives are separated into levels that correspond to the levels on the Milestones Assessment (Sundberg 2014).

Historical Background

The VB-MAPP is based on Skinner's *Verbal Behavior* (1957). Skinner used the term *verbal behavior* intentionally to differentiate his theory from more traditional theories of speech and language development. He wrote that the term *speech* was too limiting in that it excluded other types of communication such as sign language, exchanges via pictures, and writing. He also proposed that the word *language* was too broad as it encompassed entire languages (e.g., English). Traditional theories of language are broken primarily into three categories: receptive language, expressive language, and pragmatic language. However, Skinner identified several specific components of verbal behavior, referred to as verbal operants. Skinner categorized verbal behavior

into two main categories – the behavior of the speaker and that of the listener (1957). The VB-MAPP assesses speaker behavior across several verbal operants including mands, tacts, intraverbals, echoics, motor imitation as related to sign language, textual, copying a text, and transcription. The VB-MAPP also assesses for listener skills such as following instructions (Sundberg 2008).

Psychometric Data

The VB-MAPP is a criterion-referenced assessment (Sundberg 2014). Criterion-referenced assessments are designed to provide information regarding a learner's performance on specific testing items over the course of time in order to identify progress (Powers et al. 2014). Although the VB-MAPP provides age ranges for each milestone skill level, the assessment is not designed to provide a standardized score, and therefore a limitation of this assessment tool includes the inability to compare the learner's scores to their same-aged peer group.

The Milestones Assessment. The Milestones Assessment Scoring Form is a color-coded visual grid that represents each of the three skill levels. Each level contains columns with a maximum score of 5 for each of the relevant developmental domains assessed in that level. Each individual milestone score is transferred to the Milestones Assessment Scoring Form and represented on the visual grid as either a blank (0), partially filled (1/2), or fully filled (1) scoring box. The total sum of each skill domain area across all three levels is then combined to determine the total assessment score which has a maximum score of 170. Progress is demonstrated and tracked on the Scoring Form for up to four testing administrations.

The EESA. The EESA is initially scored separate from the Milestones Assessment; however, the EESA score is then incorporated into the Milestones Assessment Scoring. There are 100 possible points on the EESA. Points are assigned based on the production of correct sounds. Each item may be scored as (1) point for all correct sounds; (1/2) point for recognizable

approximations with the correct vowel sounds, addition of syllables, and/or addition of consonant sounds; or (0) points for no response, unrecognizable approximations, omitted syllables, and/or incorrect vowel usage (Sundberg 2014).

The Barriers Assessment. The scores from the VB-MAPP Barriers Assessment should be transferred to the corresponding Scoring Form. The Barriers Assessment includes a Likert scale (0–4) in which assessor scores the child on 24 potential barriers to learning. A score of 0 on the Likert scale indicates the barrier is not a concern, while a score of 4 indicates the barrier is of significant concern. A scores of 2, 3, or 4 warrant additional intervention to prevent the barrier from becoming worse (Sundberg 2014). There is a possible total of 96 points on the VB-MAPP Barriers Assessment. Similar to the Milestones Assessment, each area has an individual grid to write the score for the current testing administration. Individual scores are transferred to the Scoring Form which is comprised of 24 individual smaller grids, separated by each barrier area, and can document scoring information for four testing administrations (Sundberg 2008).

The Transition Assessment. The scores from the VB-MAPP Transition Assessment should be represented on the Transition Assessment Scoring Form. The Transition Assessment provides a Likert scale (1–5) with scores ranging between 4 and 5 indicating that the child may benefit from opportunities in a mainstream learning environment with a low student to teacher ratio; however, it is recommended to use behavioral technology in coordination with more typically teaching methods. Scores within the 0–2 range indicate that the child should primarily receive an individualized program with one-to-one direct instruction, use of behavioral technology, and sufficient oversight by qualified professionals (Sundberg 2014). Individual scores are transferred to the Scoring Form which is comprised of 18 individual smaller grids, separated by each barrier area, and can document scoring information for four testing administrations (Sundberg 2008).

Clinical Use

The VB-MAPP assessment is designed to identify skill deficits for learners with speech and language delays that impact their learning, regardless of age and/or diagnosis. Although a comprehensive knowledge of applied behavior analysis (ABA) and B.F. Skinner's *Verbal Behavior* (1957) are not required, it is important that the administrator understands verbal behavior at a basic level. Additionally, assessors should be able to identify any unintentional influences on responding evoked by inappropriate prompting procedures. Information obtained through the assessment will be beneficial for parents and educators in determining the learner's skill acquisition needs and barriers to learning, which guides the learner's educational team with planning for the individualized educational needs of the child.

See Also

- [Applied Behavior Analysis \(ABA\)](#)
- [Intraverbals](#)
- [Mands](#)
- [Reinforcement](#)
- [Skinner's Verbal Behavior](#)
- [Tact](#)
- [Verbal Behavior](#)

References and Reading

- Powers, M. D., Palmieri, M. J., Egan, S. M., Rohrer, J. L., Nulty, E. C., & Forte, S. (2014). Behavioral assessment of individuals with autism: Current practice and future directions. In F. R. Volkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. II, 4th ed., pp. 695–736). Hoboken: Wiley.
- Skinner, B. F. (1957). *Verbal behavior*. Ann Arbor: Copley Custom Textbooks, XanEdu Publishing.
- Sundberg, M. L. (2008). *The verbal behavior milestones assessment and placement program: The VB-MAPP protocol*. Concord: AVB Press.
- Sundberg, M. L. (2014). *The verbal behavior milestones assessment and placement program: The VB-MAPP guide* (2nd ed.). Concord: AVB Press.

Verbal Communication

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Synonyms

Fluent speech; Functional speech; Spoken language

Definition

Verbal communication refers to the production of spoken language to send an intentional message to a listener. Verbal and nonverbal communication abilities are considered to represent a core deficit in the diagnosis of autism. Indeed, the presence of fluent spoken language (in the form of regular and nonimitative use of multiword utterances) during the preschool years is a robust predictor of positive long-term outcomes for children with autism. In the research literature, the acquisition of fluent spoken language is sometimes referred to as functional speech. The domain of verbal communication can be divided into several component areas: semantics (vocabulary), syntax (grammar), and pragmatics (the social uses of language). Often, pragmatics is the area of spoken language that is most challenging for individuals with autism.

See Also

- Expressive Language
- Speech

References and Reading

Anderson, D. K., Lord, C., Risi, S., DiLavore, P. S., Shulman, C., Thurm, A., et al. (2007). Patterns of growth in verbal abilities among children with autism spectrum disorder. *Journal of Consulting and Clinical Psychology*, 75, 594–604.

Eigsti, I., de Marchena, A. B., Schuh, J. M., & Kelley, E. (2011). Language acquisition in autism spectrum disorders: A developmental review. *Research in Autism Spectrum Disorders*, 5, 681–691.

Howlin, P. (2007). The outcome in adult life for people with ASD. In F. R. Volkmar & F. R. Volkmar (Eds.), *Autism and pervasive developmental disorders* (2nd ed., pp. 269–306). New York: Cambridge University Press.

Stone, W. L., & Yoder, P. J. (2001). Predicting spoken language level in children with autism spectrum disorders. *Autism*, 5, 341–361.

Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, R. Paul, A. Klin, D. Cohen, F. R. Volkmar, R. Paul, et al. (Eds.), *Handbook of autism and pervasive developmental disorders, Vol. 1: Diagnosis, development, neurobiology, and behavior* (3rd ed., pp. 335–364). Hoboken: Wiley.

Tager-Flusberg, H., Rogers, S., Cooper, J., Landa, R., Lord, C., Paul, R., et al. (2009). Defining spoken language benchmarks and selecting measures of expressive language development for young children with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, 52, 643–652.

Verbal Comprehension

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Synonyms

Language comprehension; Language understanding; Receptive language

Definition

Verbal comprehension is the ability to understand spoken language. Early in typical development, a relative advantage of verbal comprehension over spoken language ability is observed when language abilities are measured using both parent report and direct assessment measures. An atypical profile of language comprehension and production has been reported for young children with autism by several groups of researchers. Two

atypical profiles have been described. In one profile, the relative advantage of receptive over expressive language is decreased (i.e., the receptive-expressive gap is smaller) for children with a diagnosis of autism, but not PDD. In the other profile, young children with autism actually demonstrate lower age-equivalent scores for language comprehension than they do for production. Many common assessment instruments include subtests designed to measure verbal comprehension of language, including the Vineland Adaptive Behavior Scales, the infant subscale of the MacArthur-Bates Communicative Development Inventory, the Preschool Language Scales, and the Mullen Scales of Early Learning. Other authors report that, in slightly older children with autism, verbal comprehension no longer lags behind expressive language in that standard scores for language comprehension are found to be commensurate with scores for spoken language. It has been suggested that the comprehension of language in a conversational context may provide challenges for individuals with autism that are not present within a standardized testing context. For example, the need to respond to non-verbal cues that support semantic meaning may interfere with the verbal comprehension ability of individuals with autism.

References and Reading

- Charman, T., Baron-Cohen, S., Swettenham, J., Baird, G., Drew, A., & Cox, A. (2003). Predicting language outcome in infants with autism and pervasive developmental disorder. *International Journal of Language & Communication Disorders*, 38, 265–285.
- Hudry, K., Leadbitter, K., Temple, K., Slonims, V., McConachie, H., Aldred, C., et al. (2010). Preschoolers with autism show greater impairment in receptive compared with expressive language abilities. *International Journal of Language & Communication Disorders*, 45, 681–690.
- Kjelgaard, M. M., & Tager-Flusberg, H. (2001). An investigation of language impairment in autism: Implications for genetic subgroups. *Language & Cognitive Processes*, 16, 287–308.
- Luyster, R. J., Kadlec, M., Carter, A., & Tager-Flusberg, H. (2008). Language assessment and development in toddlers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38, 1426–1438.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. R. Volkmar, R. Paul, A. Klin, D. Cohen, F. R. Volkmar, R. Paul, et al. (Eds.), *Handbook of autism and pervasive developmental disorders (Diagnosis, development, neurobiology, and behavior)* (Vol. 1, 3rd ed., pp. 335–364). Hoboken: Wiley.
- Weismer, S. E., Lord, C., & Esler, A. (2010). Early language patterns of toddlers on the autism spectrum compared with toddlers with developmental delay. *Journal of Autism and Developmental Disorder*, 40, 1259–1273.

Verbal Comprehension Index

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Definition

The Verbal Comprehension Index (VCI) is one of the indices representing the major components of intelligence in two of the Wechsler Intelligence Scales: the Wechsler Adult Intelligence Scale (WAIS) and the Wechsler Intelligence Scale for Children (WISC). It is thought to provide a relatively pure measure of verbal abilities, free of the influences of auditory attention and concentration. In the fourth edition of the WAIS (WAIS-IV), the VCI is comprised of three main subscales, with an additional supplemental scale: Similarities, which measures abstract verbal reasoning; Vocabulary, in which words must be defined singly, without context; Information, which tests general knowledge of history, art, culture, and politics; and Comprehension (supplemental), which measures the ability to understand abstract or idiomatic expressions. In the fourth edition of the WISC (WISC-IV), the VCI consists of the Vocabulary, Similarity, and Comprehension subtests, with Information and Word Reasoning, a task which provides clues leading to a specific word, as supplemental scales.

Research has consistently shown that children and adults diagnosed with Autistic Disorder generally obtain scores consistently lower on the VCI

than they do on nonverbal indices of the WAIS. In contrast, individuals with Asperger's disorder tend to earn their two highest subtest scores in Information and Vocabulary, reflecting the VCI as a relative strength.

See Also

► [Processing Speed Index](#)

References and Reading

- Bowler, D. (2007). *Autism spectrum disorders: Psychological theory and research*. Hoboken: Wiley.
- Flanagan, D. P., & Kaufman, A. S. (2009). *Essentials of WISC-IV assessment* (2nd ed.). Hoboken: Wiley.
- Lichtenberger, E. O., & Kaufman, A. S. (2009). *Essentials of WAIS-IV assessment*. Hoboken: Wiley.

Verbal Dyspraxia

► [Verbal Apraxia](#)

Verbal Fluency

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Synonyms

[Category fluency](#); [Controlled oral word association](#); [FAS-test](#); [Letter fluency](#); [Phonemic fluency](#); [Semantic fluency](#); [Word fluency](#)

Definition

Verbal fluency refers to the ability to spontaneously generate as many words as possible within a given time period, according to a set rule. There

are two types of verbal fluency that are commonly tested: phonemic fluency and semantic fluency. During a phonemic fluency task, a person is asked to generate as many words as possible beginning with a specific letter of the alphabet. During a semantic fluency task, the person is asked to generate as many words as possible from a specific semantic category (e.g., animals), irrespective of which letter of the alphabet the words begin with. Typically, there is a 60 s time limit to complete individual verbal fluency trials, and performance is the sum of correct words generated within the allotted time period. A number of different errors can be calculated, including repetition and intrusion errors. Two underlying abilities govern verbal fluency performance: clustering and switching. Clustering is the tendency to produce a set of words within a particular subcategory (e.g., farm animals on a semantic fluency task requiring animal words). Switching refers to the ability to shift from one subcategory to the next in order to maximize performance (e.g., shifting from farm animals to pets, once generation of farm animals slows). Performance on verbal fluency tasks is associated with frontal and temporal lobe functioning. Verbal fluency is impaired in individuals with high-functioning autism when their performance is compared to people without ASD. Conversely, individuals with Asperger syndrome appear to have intact verbal fluency abilities.

See Also

► [Executive Function \(EF\)](#)

References and Reading

- Geurts, H. M., Verté, S., Oosterlaan, J., Roeyers, H., & Sergeant, J. A. (2004). How specific are executive functioning deficits in attention deficit hyperactivity disorder and autism? *Journal of Child Psychology and Psychiatry*, 45, 836–854.
- Lezak, M. D. (1995). *Neuropsychological assessment*. Oxford: Oxford University Press.
- Spek, A., Schatorjé, T., Scholte, E., & van Berckelaer-Onnes, I. (2009). Verbal fluency in adults with high

- functioning autism or Asperger's syndrome. *Neuropsychologia*, 47, 652–656.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). New York: Oxford University Press.
- Troyer, A. K., Moscovitch, M., & Winocur, G. (1997). Clustering and switching as two components of verbal fluency: Evidence from younger and older healthy adults. *Neuropsychology*, 11, 138–146.
- Turner, M. A. (1999). Generating novel ideas: Fluency performance in high-functioning and learning disabled individuals with autism. *Journal of Child Psychology and Psychiatry*, 40, 189–201.

Verbal Intelligence

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Definition

Verbal intelligence refers to specific human language-based skills which are considered to reflect latent general abilities. Despite historical disagreement about the precise place and fundamental nature of verbal intelligence, widespread agreement about its importance is evident in its omnipresence across all major hierarchical models of human intelligence. A person's verbal intelligence is assessed through performance on one or more specific tests involving receptive and/or expressive spoken language. While these tests assess a limited range of specific verbal abilities, they are also intended to estimate, or to contribute to an estimation of, a person's general intelligence. Verbal intelligence tests contrast with performance or nonverbal intelligence tests, which may in fact require verbal skills (e.g., the comprehension of spoken instructions) but primarily are considered measures of other abilities, such as visuospatial perception or processing speed. Scores on verbal and performance intelligence tests can be combined to generate full-scale IQ scores.

The use of vocabulary tests in assessing verbal intelligence is nearly universal. In these tests, individuals are asked to demonstrate their understanding of single words, for example, by providing a spoken definition, by indicating one of several presented pictures in response to a spoken word, or by naming a single picture. Other kinds of verbal intelligence tests, often administered in addition to vocabulary tests, require specific skills in a variety of areas. Examples include identifying associations between words (verbal abstract reasoning), providing factual responses to general knowledge questions, and demonstrating comprehension of social conventions and practical "common sense."

As is evident in autism research, verbal intelligence scores can be derived in many different ways and from many different tests. A single test of vocabulary, such as the Peabody Picture Vocabulary Test (PPVT) or its variants, may be used. Alternatively, intelligence test batteries combine several verbal subtests to produce a verbal index score or Verbal IQ (VIQ), as in Wechsler Scales and the Stanford-Binet. Some intelligence test batteries also provide abbreviated versions in which verbal intelligence scores are derived from a vocabulary subtest either alone or combined with another verbal subtest. It is important to note that tests which assess verbal intelligence are culture specific and evolve over time with respect to their specific content. Thus, whether a person has or has not been educated within a specific culture affects their measured verbal intelligence. What kinds of tests are or are not included when measuring verbal intelligence may also be subject to change. For example, Wechsler scales subtests once included as measures of VIQ were later indexed as measures of working memory (e.g., tests of arithmetic ability and digit recall).

Verbal intelligence scores can be reported as IQs or other standard scores, as percentiles, age equivalents, or raw scores. Developmental tests designed for infants and preschool children (e.g., Mullen or Bayley scales) as well as tests of adaptive behavior (e.g., Vineland) are commonly used to assess autistic individuals and include language-related domain scores. However, these reflect developmental or adaptive levels rather

than verbal intelligence. Further, the content of some language-related items in developmental and adaptive tests may be similar to diagnostic signs of autism and therefore overlap with items in autism diagnostic instruments.

Historical Background

Unusual patterns of verbal abilities are prominent throughout the first formal descriptions of autistic individuals. None of the 11 children described by Leo Kanner responded typically when spoken to; a majority presented as though deaf. The eight children who developed speech (with or without delays) and the three classified as mute did not, in Kanner's view, fundamentally differ in their failure to engage in appropriate verbal communication. The speaking children displayed immediate and delayed echolalia, reversed pronouns, and a lack of spontaneous or typically meaningful sentences. Words were used in idiosyncratic or rigid and literal ways. Strong rote memory was evident, as were verbal strengths in naming objects, using plurals and tenses, and reciting information within formats or categories (e.g., poems, psalms, the alphabet, lists of animals, and presidents). As Kanner (p. 243) observed, "long and unusual words were learned and retained with remarkable facility." In later papers, he acknowledged that seemingly irrelevant or nonsense utterances in fact conveyed meaning via metaphors and other atypical but decipherable verbal associations. While autistic children showed marked resistance to teaching, regardless they learned – displaying improvements over time including spontaneous nonechoed sentences with correct pronouns. Even "mute" autistics sometimes spoke in full sentences in emergencies (Kanner 1973).

The four autistic children described by Hans Asperger had verbal abilities at least as idiosyncratic as those encountered by Kanner. They spoke like adults, responded unpredictably or inappropriately – if at all – when spoken to, and were nearly impossible to teach. Both Kanner and Asperger relied far more on description than

test scores in assessing verbal abilities; both doubted that autistic children's intelligence could be fairly estimated via conventional testing.

In 1945, Scheerer et al. reported an extended case study of "L," an autistic savant (calendar calculation, music) who excelled in uninstructed memory of word, letter, and number sequences; and of names, places, and events associated with dates (e.g., birthdays). Several attempts to measure L's intelligence were made over time with varying degrees of success. As a child L – when testable – achieved normal range IQs but as an adolescent his Stanford-Binet IQ was 50. Verbal subtest performance ranged from strengths in immediate memory (of digits and sentences) to limited or idiosyncratic performance in tests of analogies, comprehension, reasoning, and vocabulary. For example, he defined "gown" as "something I have on" (p. 9). Aspects of information he processed well were deemed outlandish and irrelevant, while his unusual verbal abilities suggested that "rote memory without comprehension of social contents and symbols may operate astoundingly well" (p. 14) to the point that impairments are concealed.

Anticipating important directions in later research, Scheerer et al. also questioned Kanner's view of autism as primarily affective. Instead they proposed that L's strengths and deficits were underpinned by a fundamental inability to understand or form concepts, symbols, and abstractions. Similar views of autism flourished in the 1960s and 1970s, as summarized in Wing and Ricks' 1975 overview of language and communication in autism. Intelligence measures, including verbal tests, were important in the emerging characterization of autism as a cognitive phenotype. Hermelin and O'Connor (1970) reported a series of seminal experiments in which autistic and non-autistic children were matched according to intelligence test scores. In a core group of autistic children whose mean age was 10 years, performance scores (Merrill-Palmer) were higher than verbal scores (PPVT). However, two children had verbal age equivalents above 10 years, well exceeding their performance scores. Hermelin and O'Connor found that autistic children organize and recall verbal information atypically, with

diminished dependence on meaning, categories, and context.

Within the same time period, Rutter and colleagues published a series of papers reporting a longitudinal study of 63 individuals originally identified as autistic or “psychotic” in the 1950s. In adolescence (mean age 15 years), Wechsler scores ranged from untestable to above average. Performance (PIQ), VIQ, and full-scale means for testable individuals were all in the 70s – and in turn higher than receptive vocabulary (PPVT) and adaptive ability (Vineland) scores. More autistics had higher PIQ than VIQ compared to the reverse pattern, with a Block Design peak and a wide scatter of subtest scores (Lockyer and Rutter 1970). The highest verbal subtest score was Digit Span, the lowest Comprehension. Rutter (1966) noted that on verbal tasks, many autistics were untestable but a minority in contrast achieved their best scores, including three individuals where this unexpected pattern was “very marked.” Yet these three, one of whom excelled in math and music, still had low Comprehension scores and a history of “very backward speech development” (p. 73). Across the full autistic sample, 31 spoke by age 5 and 7 developed speech afterward, as late as age 11; one-third received no education whatsoever, while a majority received 2 years or less. Individual VIQ scores changed (both increases and decreases) on average 17 points from IQ approximations recorded at preschool age, but the group mean did not change.

To investigate the extent and specificity of verbal impairments in the autistic cognitive phenotype, Bartak et al. (1975) characterized and tested small groups of autistic and nonautistic language disabled (“dysphasic”) boys. All the children had PIQ scores of at least 70 and were matched on this measure. The autistic boys (mean age 7 years) were distinct from their controls in achieving mean Wechsler PIQ (94–97) which dramatically exceeded their Wechsler VIQ (67, when testable), PPVT (52), and Vineland (70) scores. Their distinctively scattered Wechsler subtest scores included a relative Block Design peak and a greater range within the verbal subtests, which included an extremely low Comprehension score. Interpreting this and his earlier

follow-up study, Rutter proposed a primary role for a severe distinctive verbal impairment in autism, with scattered PIQ subtest scores (e.g., high Block Design, low Coding) speculated to result from test differences in verbal loading.

A follow-up study of the boys in Bartak et al. (1975) into young adulthood found the autistics were distinctive in their trajectory of verbal abilities (Mawhood et al. 2000). Over time, the autistics’, but not the nonautistics’, Wechsler VIQ increased significantly – a group average of 16 points, with individual increases up to 50 points. The number of autistics who could be tested on all Wechsler subtests doubled (from 9 to 18, out of 19), while the PIQ-VIQ discrepancy was no longer significant. No subtest scores were reported. On PPVT, the autistic group also did significantly better over time, largely due to losses in the controls. While mean differences between the two groups had diminished, the autistic adults displayed much higher variation in scores on the verbal tests (Wechsler VIQ, PPVT).

Starting in the 1980s, studies using false belief and other social-specific tasks proliferated, raising questions about appropriate matching strategies and instruments, and whether inconsistent results could be attributed to inadequate accounting for group differences in verbal intelligence (Bowler 2007, for an overview). Meanwhile, studies including autistic Wechsler subtest and PIQ-VIQ profiles slowly accumulated, with 16 collected in Siegel et al. (1996), who also reported a new study. Overall, the PIQ-VIQ discrepancy pattern was inconsistent across studies. This is unsurprising given that the studies spanned 31 years, included very small samples across different age ranges, had different inclusion criteria, used different test versions, and encompassed different diagnostic definitions and practices. Nevertheless, Block Design robustly stood out as the highest Performance subtest score (all studies) and Comprehension was similarly robust as lowest Verbal score (all but one study).

The 1980s also marked the gradual introduction of Asperger syndrome as a separate autistic spectrum diagnosis, a change formalized in the 1994 DSM-IV, followed shortly by Klin et al.

(1995) report of no VIQ-PIQ discrepancy in autism, but a large Wechsler VIQ over PIQ discrepancy in Asperger syndrome. Ehlers et al. (1997) reported a similar result, with an Asperger subtest profile dominated by Verbal peaks (Vocabulary, Similarities, Information) and low scores in the Performance subtests Coding and Object Assembly.

While the views of autism touched upon above have fluctuated in their prominence or have been modified over time, the historical importance of verbal intelligence in autism research continues to the present. Thus verbal intelligence tests have been and continue to be used in cognitive profiles of autism: to assess different specific verbal abilities and to contrast verbal intelligence with other abilities. Verbal intelligence tests continue to be used as matching variables, as measures of changes in ability over time, in attempts to form or confirm autism subgroups, in case studies of individuals with exceptional or savant abilities, and as a way to differentiate autistic individuals from both the typical population and other atypical populations. Descriptions of aspects of autistic verbal abilities (e.g., lack of overt response to speech sounds; idiosyncrasies of speech such as pronoun reversal) that date back to Kanner's seminal observations remain important within diagnostic instruments and in characterizing developmental trajectories in autism.

Current Knowledge

Empirical Findings from Verbal Intelligence Tests

The diagnosis of autism is consistent with all possible levels of measured verbal intelligence. In other words, verbal intelligence in autism, as assessed via different standard measures, spans the full range from a VIQ of 150 or more to an assigned score of "untestable." High variance with respect to specific verbal abilities is durably characteristic of autism within and across individuals, encompassing multiple dimensions, and including sometimes dramatic incongruities between high and low abilities. Addressing such high variance by creating autism or autistic

spectrum subgroups is frequently invoked as a high priority, but as yet, little consensus as to valid subgroups has emerged. Widespread disagreement over how and whether to distinguish Asperger syndrome from autism has led to a welter of inconsistent findings regarding cognitive profiles, including patterns of verbal intelligence scores, across the autistic spectrum. DSM-IV autism subgroups, including Asperger syndrome, are now lumped together as a single autistic spectrum diagnosis in the DSM-5, where "with or without language impairment" is one of several specifiers but a history of speech delay is not considered.

However, regardless of diagnostic confusion and numerous other confounds (see "[Historical Background](#)" above), some findings persist, including autistics' difficulty with the Wechsler verbal subtest Comprehension. In a population-based study of school-aged children, Charman et al. (2011) found Comprehension scores significantly below Wechsler subtest means across various subgroups as well as in the full group of autistic children who were testable. No other finding involving Wechsler subtests achieved such consistency in the broadly defined autistic spectrum sample. Another persistent finding is adaptive ability scores which are lower than measured verbal intelligence, a discrepancy that can be extreme in autistic individuals with average or high VIQ scores. For example, in a clinic-based sample of autistic spectrum children and adolescents whose VIQ was at least 70, mean VIQ was 107 (range 70–150) in contrast with mean Vineland composite scores of 55 (range 25–79; Klin et al. 2007).

Large individual variations in trajectories of specific verbal abilities over time have also been reported as characteristic of autism. In a follow-up to mean age 29, Howlin et al. (2004, p. 219) reported "considerable movement in verbal IQ levels over time" in some individuals, with a small group-level improvement. Of the 68 autistics tested, 14 fell into the normal range of VIQ in childhood, while 32 scored at this level as adults. Thirteen of 31 individuals who as children had verbal intelligence scores of less than 30, or could not be tested at all, scored

significantly higher as adults – nine of them in the normal range. Neither verbal intelligence measured in childhood nor development of speech by 5 years of age – a milestone popularly claimed to be crucial – was a good predictor of a range of adult outcomes. A later follow-up of the same sample to mean age 44 found high variance in VIQ-PIQ discrepancies (from VIQ 50 points lower than PIQ, to 38 points higher) and a “small but steady increase” in VIQ across timepoints in testable individuals (Howlin et al. 2014).

In the same direction, Anderson et al. (2007) investigated verbal ability trajectories within childhood, starting from an original diagnosis of autism, PDD-NOS, or other developmental disability at age 2, to outcomes at a mean age of 9 years. Children were assessed with a mix of developmental and IQ tests. The main finding was higher variance in verbal ability outcomes in the autistic spectrum children, particularly those whose specific diagnosis was autism. In these children, “improvement can range from minimal to dramatic” (p. 594) with many trajectories collected at either extreme. While group mean verbal abilities were lower for autistic than nonautistic children, the exceptionally wide range of autistic outcomes was such that a greater number of autistic children achieved average level or better verbal intelligence scores at age 9.

Case studies of autistic individuals further add to the picture of remarkable variance in verbal intelligence and apparent incongruities across numerous dimensions. Autistics who have little in the way of functional speech and are therefore classified as “nonverbal” may also have high measured verbal intelligence. For example, Bonneh et al. (2008) report a Wechsler VIQ of 128 while Gernsbacher (2004) reports a PPVT standard score of 160, in case studies of minimally speaking autistic children tested respectively at 12 and 6 years of age. Conversely, a nonspeaking autistic adult is reported unscorable on PPVT, suggesting verbal intelligence so low it cannot be measured. However, he also has outstanding performance (estimated IQ of 140) on Raven’s Matrices – a general intelligence test which does not include verbal material yet for nonautistics requires verbal

abilities. The same autistic adult demonstrates outstanding calculation skills whose acquisition, even at a much more basic level, would depend on verbal abilities in nonautistics (Anderson et al. 1999). An incongruity between very low or untestable VIQ and normal-range or high Raven scores has also been displayed by some autistic children (Dawson et al. 2007; Courchesne et al. 2015). The important question of how to fairly assess verbal intelligence in minimally speaking autistics, who in addition may have difficulty pointing, has belatedly appeared in the literature (Kasari et al. 2013), but studies are as yet sparse and preliminary.

Underlying Abilities and Theoretical Accounts

With respect to early development, both echolalia and hyperlexia are well established as characteristic of autism and both indicate that autistics acquire verbal abilities in an atypical way. Autistics may enter into language by first learning its complex structure rather than by first learning its meaning, as is the typical pattern. For example, evidence suggests an early atypical developmental pattern of discrepantly high expressive versus receptive verbal abilities, at least in what has been considered the specific diagnosis of autism (e.g., Hudry et al. 2010). However, findings may be in part dependent on adaptive and parent-report measures, and again are within a context of high individual variance across all verbal measures.

Beyond early development, where autistics’ verbal abilities may be difficult to assess, numerous findings have confirmed that autistics of all ages perceive, organize, and recall verbal information in atypical ways. Autistics are characterized by strong phonological processing of language and by enhanced perception of speech. In neither case is there a consistently straightforward or predictable trade-off with other aspects of verbal information processing. Exceptional absolute pitch for speech, for instance, has been found in an autistic individual with a characteristic delay in speech development – but the person went on to become fluent in numerous languages (Heaton et al. 2008).

In addition, although autistics may organize and recall verbal information without using typical categories, they are also capable of forming categories when instructed to. The typicality, or not, of category formation displayed by autistics thus has depended on specific task instructions (Bowler 2007). Similarly, autistics' use of context when processing verbal information atypically encompasses both local (e.g., immediately proximate) and global (e.g., sentence-level) contexts. This does not imply an inability to use wider contexts but does demonstrate that more immediate contextual possibilities are not discarded via typically mandatory top-down processes (Henderson et al. 2011). In turn, this is consistent with the idiosyncratic use of language in autism, including unusual vocabulary and word associations. Further in this direction, autistics show a consistent pattern of difficulty with episodic but not semantic verbal memory, and also difficulty with organizing verbal information into expected, typical narratives (e.g., Diehl et al. 2006). Autistics would therefore be expected to perform poorly on tests which require verbal information to be organized into conventional or "common sense" type narratives, as in the Wechsler Comprehension subtest.

Overall, existing findings suggest that aspects of verbal information which automatically take precedence in nonautistic information processing hierarchies do not play the same role in autistics (Mottron et al. 2006). For autistic individuals, depending on circumstances and task demands, this may result in both advantages and disadvantages, which in turn are evident in uneven development and verbal intelligence profiles within and across individuals.

Future Directions

- Individuals diagnosed with neurodevelopmental genetic syndromes (e.g., fragile X, Williams syndrome) may also meet autism criteria on various instruments. Whether these individuals are in fact autistic is an increasingly important research question which cognitive

profiles, including verbal intelligence measures, may help address.

- The challenge of fairly testing verbal intelligence in all autistics, including those with atypical or minimal speech, was apparent in the first published descriptions of autism. Despite progress in technology – touch screens, smart phones, tablets, applications – this area remains neglected.
- New technologies should also be explored as opportunities for autistics to develop and display verbal abilities. For example, given the opportunity, autistics may demonstrate their ability to learn and adapt technologies for communication in both expected and unexpected ways.
- There has been little interest in the atypical kinds, quantities, and arrangements of information – including verbal information – from which autistics learn well. Availability (or not) of verbal information remains overlooked as a plausible factor in the high variance of verbal abilities in autism.
- Autistics with excellent adult outcomes, but atypical development and patterns of specific verbal abilities, have appeared in the literature throughout autism research history. Yet these individuals, and the patterns of verbal ability associated with successful autistic outcomes, remain understudied.
- The relationship between speech delay and later verbal intelligence, including in adulthood, remains understudied. Whether longer or shorter or no speech delay, or for that matter precocious speech, consistently leads to better or worse verbal (and other) autistic outcomes is an important but neglected research question.
- High variance in verbal intelligence is widely regarded as an obstacle to autism research which ideally should be eliminated through subgrouping. However, attempts to compose consensual autism subgroups have not been successful. One future direction is to consider high variance in verbal outcomes not as an impediment to research but as informative about the nature of autism and the possibilities of autistic individuals.

See Also

- [Nonverbal Intelligence](#)
- [Savant Skills \(in Autism\)](#)
- [Vocabulary](#)

References and Reading

- Anderson, M., O'Connor, N., & Hermelin, B. (1999). A specific calculating ability. *Intelligence*, 26, 383–403.
- Anderson, D. K., Lord, C., Risi, S., DiLavore, P. S., Shulman, C., Thurm, A., Welch, K., & Pickles, A. (2007). Patterns of growth in verbal abilities among children with autism spectrum disorder. *Journal of Consulting and Clinical Psychology*, 75, 594–604.
- Asperger, H. (1944/1991). Autistic psychopathology in childhood (Frith, U., Trans.). In: U. Frith, (Ed.), *Autism and asperger syndrome* (pp. 37–92). Cambridge, UK: Cambridge University Press.
- Bartak, L., Rutter, M., & Cox, A. (1975). A comparative study of infantile autism and specific developmental language disorder, I. the children. *The British Journal of Psychiatry*, 126, 127–145.
- Bonneh, Y. S., Belmonte, M. K., Pei, F., Iversen, P. E., Kenet, T., Akshoomoff, N., Adini, Y., Simon, H. J., Moore, C. I., Houde, J. F., & Merzenich, M. M. (2008). Cross-modal extinction in a boy with severely autistic behaviour and high verbal intelligence. *Cognitive Neuropsychology*, 25, 635–652.
- Bowler, D. (2007). *Autism spectrum disorders: Psychological theory and research*. Hove: Wiley.
- Charman, T., Pickles, A., Simonoff, E., Chandler, S., Loucas, T., & Baird, G. (2011). IQ in children with autism spectrum disorders: Data from the special needs and autism project (SNAP). *Psychological Medicine*, 41, 619–627.
- Courchesne, V., Meilleur, A. A. S., Poulin-Lord, M. P., Dawson, M., & Soulières, I. (2015). Autistic children at risk of being underestimated: school-based pilot study of a strength-informed assessment. *Molecular autism*, 6(1), 12.
- Dawson, M., Soulières, I., Gernsbacher, M. A., & Mottron, L. (2007). The level and nature of autistic intelligence. *Psychological Science*, 18, 657–662.
- Diehl, J. J., Bennetto, L., & Young, E. C. (2006). Story recall and narrative coherence of high-functioning children with autism spectrum disorders. *Journal of Abnormal Child Psychology*, 34, 87–102.
- Ehlers, S., Nyden, S., Gillberg, C., Sandberg, A. D., Dahlgren, S. O., Hjelmquist, E., & Oden, A. (1997). Asperger syndrome, autism and attention disorders: A comparative study of the cognitive profiles of 120 children. *Journal of Child Psychology and Psychiatry*, 38, 207–217.
- Gernsbacher, M. A. (2004). Language without speech: A case study. *Journal of Developmental and Learning Disorders*, 8, 123–138.
- Heaton, P., Davis, R. E., & Happé, F. G. (2008). Research note: Exceptional absolute pitch perception for spoken words in an able adult with autism. *Neuropsychologia*, 46, 2095–2098.
- Henderson, L. M., Clarke, P. J., & Snowling, M. J. (2011). Accessing and selecting word meaning in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 52(9), 964–973.
- Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children*. Oxford: Pergamon Press.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45, 212–229.
- Howlin, P., Savage, S., Moss, P., Tempier, A., & Rutter, M. (2014). Cognitive and language skills in adults with autism: A 40-year follow-up. *Journal of Child Psychology and Psychiatry*, 55(1), 49–58.
- Hudry, K., Leadbitter, K., Temple, K., Slonims, V., McConachie, H., Aldred, C., Howlin, P., Charman, T., & PACT Consortium. (2010). Preschoolers with autism show greater impairment in receptive compared with expressive language abilities. *International Journal of Language & Communication Disorders*, 45, 681–690.
- Kanner, L. (1973). *Childhood psychosis: Initial studies and new insights*. New York: Winston/Wiley.
- Kasari, C., Brady, N., Lord, C., & Tager-Flusberg, H. (2013). Assessing the minimally verbal school-aged child with autism spectrum disorder. *Autism Research*, 6(6), 479–493.
- Klin, A., Volkmar, F. R., Sparrow, S. S., Cicchetti, D. V., & Rourke, B. P. (1995). Validity and neuropsychological characterization of Asperger syndrome: Convergence with nonverbal learning disabilities syndrome. *Journal of Child Psychology and Psychiatry*, 36, 1127–1140.
- Klin, A., Saulnier, C. A., Sparrow, S. S., Cicchetti, D. V., Volkmar, F. R., & Lord, C. (2007). Social and communication abilities and disabilities in higher functioning individuals with autism spectrum disorders: The Vineland and the ADOS. *Journal of Autism and Developmental Disorders*, 37, 748–759.
- Lockyer, L., & Rutter, M. (1970). A five to fifteen year follow-up study of infantile psychosis, IV. Patterns of cognitive ability. *The British Journal of Social and Clinical Psychology*, 9, 152–163.
- Mawhood, L., Howlin, P., & Rutter, M. (2000). Autism and developmental receptive language disorder – A comparative follow-up in early adult life. I: Cognitive and language outcomes. *Journal of Child Psychology and Psychiatry*, 41, 547–559.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. A. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36, 27–43.
- Ricks, D. M., & Wing, L. (1976). Language, communication, and the use of symbols in normal and autistic children. *Journal of Autism and Childhood Schizophrenia*, 5, 191–221.

- Rutter, M. (1966). Behavioural and cognitive characteristics of a series of psychotic children. In L. Wing (Ed.), *Early childhood autism: Clinical, educational and social aspects* (pp. 51–81). Oxford: Pergamon Press.
- Scheerer, M., Rothmann, E., & Goldstein, K. (1945). A case of “idiot-savant”: An experimental study of personality organization. *Psychological Monographs*, 58, 1–63.
- Siegel, D. J., Minshew, N. J., & Goldstein, G. (1996). Wechsler IQ profiles in diagnosis of high-functioning autism. *Journal of Autism and Developmental Disorders*, 26, 389–306.

with pragmatic language, and poor construction of a narrative.

See Also

- [Auditory Verbal Learning](#)
- [Pragmatics](#)
- [Semantic Memory](#)
- [Weak Central Coherence](#)

Verbal Semantic Coherence

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Synonyms

[Weak central coherence](#)

Definition

Verbal semantic coherence refers to the ability to use the broader context of a story or sentence, and the semantic relationships between words to aid understanding and interpretation of spoken and written language. The concept of verbal semantic coherence stems from Weak Central Coherence Theory, which posits that individuals with autism spectrum disorder (ASD) have a cognitive style that involves detail-focused processing, rather than global or gist processing of information. In other words, people with ASD are more likely to see the individual trees rather than the forest. This detail-focused cognitive style has been observed while individuals with ASD engage in verbal tasks. Examples of impaired verbal semantic coherence in autism include difficulties using sentence context to interpret the meaning of homographs and heteronyms (words with one spelling, but two meanings and two pronunciations), poor recall of semantically related words, difficulties

References and Reading

- Diehl, J. J., Bennetto, L., & Young, E. C. (2006). Story recall and narrative coherence of high-functioning children with autism spectrum disorders. *Journal of Abnormal Child Psychology*, 34, 87–102.
- Frith, U., & Snowling, M. (1983). Reading for meaning and reading for sound in autistic and dyslexic children. *British Journal of Developmental Psychology*, 1, 329–342.
- Happé, F. (1999). Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Science*, 3, 216–222.
- Happé, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36, 5–25.
- Hermelin, B., & O'Connor, N. (1967). Remembering of words by psychotic and subnormal children. *British Journal of Psychology*, 58, 213–218.
- López, B., & Leekam, S. R. (2003). Do children with autism fail to process information in context? *Journal of Child Psychology and Psychiatry*, 44, 285–300.

Verbalization

- [Speech](#)

Versed

- [Midazolam](#)

Very Early-Onset Schizophrenia

- [Childhood Schizophrenia](#)

Vestibular Function in Children with Autism

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Synonyms

[Equilibrium](#); [Sense of balance](#)

Definition

Vestibular function refers to the ability to maintain balance and postural control, a stabilized vision during head movements, and spatial orientation. The vestibular system is a complex sensorimotor system which comprises the peripheral vestibular apparatus or labyrinth, the postural muscles, visual system, brainstem, cerebellum, and the cortex. The peripheral portion of the vestibular system is located in the inner ear and consists of three semi-circular canals and two otolith organs (i.e., saccule and utricle) providing complementary information about rotational and translational head movements relative to gravity. It gives together with centrally integrated proprioception and visual stimuli an internal representation of the environment and movements through it. In addition, a stabilized vision during head movements and postural control are reflexively maintained by the vestibulo-ocular, vestibulospinal, and vestibulo-collic reflexes. In case of a dysfunction in this complex system, postural imbalance and dizziness are often the most reported complaints in adults. In contrast, these symptoms are frequently underestimated in children, since children are often not able to communicate their complaints properly and a vestibular impairment often has a different clinical course compared to that in adults. Nevertheless, when a vestibular dysfunction occurs at birth or in early stages of life, an impact on motor, cognitive,

psychosocial, and educational performances may be expected.

Similar to children with a severe vestibular dysfunction, children with autism often present with vestibular-related complaints such as avoidance behavior, gross and fine motor impairment, static and dynamic postural instability, delayed motor milestones, and/or poor motor coordination. Additionally, compared to typically developing children, research indicated that postural performances in children with autism were found to be the most aberrant in conditions where they could rely on their vestibular input only, which is also suggestive for a vestibular deficit. Therefore, it has been suggested that a (peripheral or central) vestibular dysfunction may contribute to the phenotype or behavioral features in a subset of children with autism spectrum disorder, which has been supported by studies using vestibular function testing in this population. Moreover, some children with autism are known to have a preoccupation with spinning objects or repetitive movements, such as spinning or shaking their head or have difficulties in integrating vestibular input. However, due to heterogeneous findings in studies mainly focusing on the function of the horizontal semicircular canals only (only one fifth of the peripheral vestibular system), a more extensive and objective elaboration on whether an accompanying vestibular dysfunction is present in this population is highly recommended.

See Also

- [Sensory Impairment in Autism](#)
- [Vestibular System in Autism](#)

References and Reading

- Carson, T. B., Wilkes, B. J., Patel, K., Pineda, J. L., Ko, J. H., Newell, K. M., et al. (2017). Vestibulo-ocular reflex function in children with high-functioning autism spectrum disorders. *Autism Research*, 10(2), 251–266. <https://doi.org/10.1002/aur.1642>.

- Furman, J. M., Osorio, M. J., & Minshew, N. J. (2015). Visual and vestibular induced eye movements in verbal children and adults with autism. *Autism Research*, 8(6), 658–667. <https://doi.org/10.1002/aur.1481>.
- Goldberg, M. C., Landa, R., Lasker, A., Cooper, L., & Zee, D. S. (2000). Evidence of normal cerebellar control of the vestibulo-ocular reflex (VOR) in children with high-functioning autism. *Journal of Autism and Developmental Disorders*, 30(6), 519–524.
- Ornitz, E. M., Atwell, C. W., Kaplan, A. R., & Westlake, J. R. (1985). Brain-stem dysfunction in autism. Results of vestibular stimulation. *Archives of General Psychiatry*, 42(10), 1018–1025.
- Van Hecke, R., Danneels, M., Dhooge, I., Van Waelvelde, H., Wiersema, J. R., Deconinck, F. J. A., & Maes, L. (2019). Vestibular function in children with neurodevelopmental disorders: A systematic review. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-019-04059-0>.

Vestibular System in Autism

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Definition

Vestibular System

A system in the body that is responsible for maintaining balance, posture, and the body's orientation in space. This system also regulates locomotion and other movements and keeps objects in visual focus as the body moves.

The vestibular system is comprised of the vestibular apparatus itself, the vestibulocochlear nerve, and those parts of the brain that interpret and respond to information derived from these structures.

Historical Background

Abnormal modulation of sensory input (over and under responding) has been a pathognomonic feature of autistic disorder (AD) since it was first described by Kanner (1968). Of particular note is the fact that response levels may not remain

static, but can alternate over time in the same individual. This characteristic, called “Perceptual Inconstancy” (Ornitz and Ritvo 1968) underlies the diagnostic criteria for AD listed in the DSM-IV-TR (American Psychiatric Association 2000) as, “stereotyped and repetitive motor mannerisms” (e.g., hand or finger flapping or twisting, or complex whole-body movements). The proposed DSM V (American Psychiatric Association 2010) lists these symptoms as, “Hyper- or hypo-reactivity to sensory input or unusual interest in sensory aspects of environment; (such as apparent indifference to pain/heat/cold, adverse response to specific sounds or textures, excessive smelling or touching of objects, fascination with lights or spinning objects).”

Abnormal modulation affects all sensory systems (Ritvo 2006; Leekam et al. 2007; Schoen et al. 2009, DSM-IV-TR and proposed DSM 5, Reynolds and Lane 2008; Minshew and Hobson 2008), for example, many AD infants and children are initially referred for evaluation of possible deafness. These children may not respond at times to speech or loud noises, and at other times cover their ears and cry in response to soft sounds. Alternating sensitivity to temperature is shown in AD children who demand wool sweaters and heavy coats in the summer, but will only wear light skimpy clothes in the winter. Response to pain varies from ultra sensitive to nonexistent, even with major injuries. Visual detailed scrutiny may alternate with bumping into things as if they were not seen. Hypersensitivity to the texture of food is responsible for the often-reported behavior of spitting out lumpy items, a problem many parents solve intuitively by using a blender. And, lastly, many AD children become preoccupied with obtaining certain specific odors, while at other times will ignore noxious fumes.

Current Knowledge

Symptoms Due to Abnormal Modulation of Vestibular Input

Under responsiveness to vestibular input leads to several types of repetitive behaviors. For example, many children and adults with AD spend hours

each day on swings, jumping on trampolines, and twirling themselves around. They do this without becoming dizzy or nauseous, and report that it is “soothing” and “comforting” (Ritvo 2006).

Staring at rotating objects such as phonograph records and fans, head shaking, body rocking, and finger flapping in front of the eyes are other common ways AD individuals induce nystagmus and vestibular input.

Over responsiveness to vestibular input leads to the opposite kind of behaviors. Maintaining rigid postures, placing themselves in unusual prone or upside down positions, refusing to get out of bed, and hiding in dark places are behaviors that AD individuals use to diminish vestibular input and maintain equilibrium.

Symptoms due to both over and under modulation of vestibular input, like those in the other sensory systems, tend to decrease in frequency and duration with age, although, in some, they remain lifelong (Ritvo 2006).

Neurophysiologic Studies

Several neurophysiologic studies begun in the 1970s attempted to identify objective measures of abnormal vestibular processing. One pilot study identified a subset of AD children who showed decreased post rotator nystagmus (PRN) in the light, when visual fixation is possible, but not in the dark (Ritvo et al. 1969). However, replication studies failed to identify specific PRN differences between large groups of AD and age and sex matched non-AD children that could be of diagnostic significance (Ornitz 1970). A review of the recent literature failed to find subsequent studies that identified specific pathology in the vestibular system (peripheral or central) pathognomonic of autism, but concluded that more research in this area is warranted by previous findings (Rogers and Ozonoff 2005; Reynolds and Lane 2008).

Therapeutic Implications of Abnormal Vestibular Modulation

Based on the clinical observations of Jane Ayers in the 1970s (Ayers 1974), occupational therapists and sensory integration therapists developed interventions aimed at assessing and improving symptoms of abnormal vestibular modulation.

These interventions have gained worldwide application based on positive clinical experience, but to date no objective studies of their efficacy have been published (Kinnealey et al. 1995; Watling et al. 2001; Tomchek and Dunn 2007).

References and Reading

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders*. (4th ed. text revision). Washington, DC.
- American Psychiatric Association. *DSM- 5*. (2010). <http://www.dsm5.org>
- Ayres, A. J. (1974). *The development of sensory integrative theory and practice: A collection of the works of A Jean Ayres*. Dubuque: Kendall/Hunt Publishing.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Kanner, L. (1968). Autistic disturbances of affective contact. *Acta Paedopsychiatrica*, 35(4), 100–136.
- Kinnealey, M., Oliver, B., & Wilbarger, P. (1995). A phenomenological study of sensory defensiveness in adults. *American Journal of Occupational Therapy*, 49, 444–451.
- Leekam, S. R., Libby, S. J., Wing, L., & Gould, J. (2007). Describing the sensory abnormalities of children and adults with autism. *Journal of Autism and Developmental Disorders*, 37, 894–910.
- Minshew, N. J., & Hobson, J. A. (2008). Sensory sensitivities and performance on sensory perceptual tasks in high-functioning individuals with autism. *Journal of Autism and Developmental Disorders*, 38, 1485–1498.
- Ornitz, E. M. (1970). Vestibular dysfunction in schizophrenia and childhood autism. *Comprehensive Psychiatry*, 11, 159–173.
- Ornitz, E. M., & Ritvo, E. R. (1968). Perceptual inconstancy in early infantile autism. *Archives of General Psychiatry*, 18, 76–98.
- Reynolds, S., & Lane, S. J. (2008). Diagnostic validity of sensory over-responsivity: A review of the literature and case reports. *Journal of Autism and Developmental Disorders*, 38, 516–529.
- Ritvo, E. R. (2006). *Understanding the nature of autism and Asperger disorder*. London: Jessica Kingsley Publishers.
- Ritvo, E. R., Ornitz, E. M., Eviatar, A., Markham, C. H., Brown, M., & Mason, A. (1969). Decreased post-rotatory Nystagmus in early infantile autism. *Neurology*, 19, 653–658.
- Rogers, S. J., & Ozonoff, S. (2005). Annotation: what do we know about sensory dysfunction in autism? A critical review of the empirical evidence. *Journal of Child Psychology and Psychiatry*, 46, 1255–1268.
- Schoen, S. A., Miller, L. J., Brett-Green, B. A., & Nielsen, D. M. (2009). Physiological and behavioral differences in sensory processing: A comparison of children with

- autism spectrum disorder and sensory modulation disorder. *Frontiers in Integrative Neuroscience*, 3, 29.
- Tomchek, S. D., & Dunn, W. (2007). Sensory processing in children with and without autism: A comparative study using the short sensory profile. *American Journal of Occupational Therapy*, 61, 190–200.
- Watling, R. L., Deitz, J., & White, O. (2001). Comparison of sensory profile scores of young children with and without autism spectrum disorders. *American Journal of Occupational Therapy*, 55, 416–423.

Victimization

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Definition

There is no consensus on what victimization is or what it should cover. The dictionary definition includes victimization of crime, of unjust punishment and of fraud. Victimization is often classified by the source of the offense or the nature of the attack. Two forms of victimization receive disproportionate amount of attention by the research community, child abuse and peer victimization or bullying, while other forms of victimization are not systematically studied. This entry deals with those unstudied sources of victimization following the dictionary definition of crime, unjust punishment, and fraud.

Historical Background

Although undoubtedly victimization of persons with ASD is as old as the disease itself, there has been and continues to be an almost complete paucity of research on the topic.

Current Knowledge

Much of what is known about criminal victimization is not specific to persons with ASD. People with disabilities are two to three times more likely

to be victims of violent crime (rape/sexual assault, robbery, simple, and aggravated assault). In addition, people with ASD are also at higher risk of financial exploitation (Volkmar et al. 2005). About 15% of crime victims with disabilities report that they suspect they were targeted because of their disability (Harrell and Rand 2010). Because of their inability to perceive or understand social clues, people with ASD have been called the “perfect victims” (Debbaudt 2002).

Victimization by direct caregivers (e.g., parents) is often treated as a form of child abuse, although such victimization may occur beyond the age of 18. Peer victimization has been studied repeatedly mostly among children with ASD in the school context (see “► Bullying”). Little is known about peer victimization among adults with ASD.

Victimization by educators and professional caregivers was recently documented in a report from the US Government Accountability Office on the use of seclusions and restraints that resulted in deaths or abuse (United States Government Accountability Office 2009). Given that there is no systematic research on victimization by school personnel, the prevalence of this form of victimization is unknown.

Additionally, persons with ASD can be victimized by law enforcement. When the police comes into contact with persons with ASD spectrum disorders because they are witnesses, perpetrators, or victims of crime, untrained officers can easily mistake ASD symptomatology for suspicious behavior. ASD symptomatology (e.g., absence of eye contact, repeating what an officer says, not understanding body language, not following commands, confessing to crimes they did not commit, persistent interest on aspects of a situation others believe irrelevant) can be easily confused with indications of guilt by officers unfamiliar with ASD during informal questioning, arrest, and formal interrogation. Sometimes such mistakes may lead to criminal convictions of innocent persons with ASD disorders; other times the information provided by a person with ASD is thought to be unreliable (Debbaudt 2002). Consequently, the Federal Bureau of Investigation and a number of ASD advocacy organizations (in particular the Autism

Society) are working with police and other first respondents to educate them about ASD (Debbaudt and Rothman 2001).

As is the case for many other people with incurable diseases, many persons with ASD and their families become victims of those who market untested remedies and effective treatments or cures for ASD. Distinguishing between legitimate and responsible complementary and alternative medicine (CAM) practice and quackery can be very difficult, particularly for desperate parents of children with ASD at a time when the use of CAM is increasing among the general population. Quackery is characterized by exaggerated claims about the likely benefits of the product or service for the typical patient, as well as its higher likelihood to result in financial, emotional or physical harm, including unwarranted delays in seeking appropriate diagnosis and treatment.

In the United States, both the Federal Drug Administration and the Federal Trade Commission have responsibility for consumer protection, yet both agencies lack adequate resources to police the many attempts to promote services and therapies of questionable value to people with ASD. In rare cases, the FDA or the FTC has sent warnings to marketers of unapproved therapies or products (e.g., unapproved chelation products (Federal Drug Administration 2010)). Yet, many CAM approaches are outside of the jurisdiction of the FDA and current regulatory practice in the United States requires the government to prove evidence of harm. Additionally, many of these approaches are not subject to adverse event reporting of any kind. Thus, it is unsurprising that many cases of misrepresentation of alleged benefits are not investigated or prosecuted, as they often go unreported by the victims. As in other forms of victimization, there are no scientific studies on the prevalence and nature of financial, emotional, or physical harms that occur due to quackery, nor any studies to determine if people with ASD or their close relatives can distinguish between legitimate CAM and quackery. There are however many published accounts showing that quackery and charlatanism related to ASD therapies and

“cures” is widespread (Fitzpatrick 2008; Offit 2008; Schreibman 2007).

Finally, persons of ASD are also victims of fraud when researchers commit scientific misconduct on ASD-related studies. The most famous cause was the Andrew Wakefield’s 1998 study published in *The Lancet* expressing concerns that the MMR vaccination was linked to ASD. The dissemination of the claim through the media resulted in a loss of confidence in the MMR vaccination program in the United States and United Kingdom. For many families with children with ASD, the study also became a justification to seek treatment and cures outside of the medical establishment, thus delaying appropriate and timely treatment and exposing them to an increased risk of financial, emotional, and physical harm. In addition, many families decided not to vaccinate children who did not have ASD for fear of contracting ASD. In 2004, many of the Dr. Wakefield’s coauthors retracted the problematic interpretation (Murch et al. 2004). The UK’s General Medical Counsel erased Dr. Wakefield’s name from the medical register in 2010 and the original paper was subsequently retracted from *the Lancet* (Greenhalgh 2010).

Future Directions

There is no question that persons with ASD are at higher risk of various forms of victimization, yet scientific studies have not been systematically conducted to determine the prevalence of the various types of victimizations for persons with ASD and their families. With the exception of bullying and child abuse, there are no systematic efforts to determine the prevalence, causes, and remedies of the various forms of victimization presented in this entry. People with ASD will continue to be at higher risk of these various forms of victimization until studies elucidate their plight.

See Also

► [Bullying](#)

References and Reading

- Debbaudt, D. (2002). *ASD, advocates and law enforcement professionals: Recognizing and reducing risk situations for people with ASD spectrum disorders*. Philadelphia: Jessica Kingsley.
- Debbaudt, D., & Rothman, D. (2001). Contact with Individuals with ASD. *FBI Law Enforcement Bulletin*, 70(4), 20–24.
- Federal Drug Administration. (2010). FDA issues warnings to marketers of unapproved “chelation” products 2011, from <http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm229320.htm>
- Fitzpatrick, M. (2008). *Defeating ASD: A damaging delusion*. London: Routledge.
- Greenhalgh, T. (2010). Observations on GMC Wakefield verdict: Why did it take the Lancet so long? *British Medical Journal*, 340, c664.
- Harrell, E., & Rand, M. R. (2010). Crime against people with disabilities, 2008. *Bureau of Justice Statistics Statistical Tables*, NCJ(231328).
- Murch, S. H., Anthony, A., Casson, D. H., Malik, M., Berelowitz, M., Dhillon, A. P., et al. (2004). Retraction of an interpretation. *The Lancet*, 363(9411), 750.
- Offit, P. A. (2008). *ASD's false prophets: Bad science, risky medicine and the search for a cure*. New York: Columbia University Press.
- Schreibman, L. (2007). *The science and fiction of ASD*. Cambridge, MA: Harvard University Press.
- United States Government Accountability Office. (2009). *Seclusions and restraints: Selected cases of death and abuse at public and private schools and treatment centers*. Washington, DC: Testimony before the committee on Education and Labor, House of Representatives.
- Volkmar, F. R., Paul, R., Klin, A., & Cohen, D. (2005). *Handbook of ASD and pervasive developmental disorders* (Vol. 1). New York: Wiley.

and a toy truck with which to play, but then pushes the toys away without making a choice. Simply replaying the video would be feedback and probably ineffective, so a replay would be used as an opportunity to teach an adaptive response. Thus the feedback itself might not be educational or therapeutic, but may serve to present “teaching moments,” which may be effective if developmentally appropriate.

Video feedforward entails the portrayal of an event as a future success, beyond current capability, achieved through filming and editing techniques. That is, a person will see a video of themselves succeeding in something where they normally fail. For example, the child above would be also be offered, say, a truck, filmed from a different angle, maybe even prompted to say “thank you.” The separate shots are then edited together to show the child adaptively choosing and receiving a toy, in this case, a truck. So the “teaching moment” is shown on video and the feedback errors are not used.

Thus feedback is effective when it allows feedforward to take place, either through additional training or therapy or through appropriately recorded and edited video. With individuals for whom learning comes easily, just seeing and hearing the video may be enough to create spontaneous feedforward and future adaptive responses to the event.

Video Feedback and Feedforward

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Definition

Video feedback entails the replay of a recorded event with the intent that participants will learn improved behavior for that situation. For example, a child may be offered a choice of a toy bear

Historical Background

The first use of the term “feedback” was in the 1920s, in reference to that dreadful oscillation in sound systems turned in on themselves. Then feedback progressed through physics and biology, later psychology and education, to reference information about the impact on the environment of systems and organisms, and then the potential influence of that information in a “feedback loop.” The Concise Oxford Dictionary (2006, p. 521) defines feedback in psychology and biology as the “modification or control of a process or system by its results or effects.” A common biological example is the blink reflex, by which the eyelid moistens a drying eyeball. Examples in psychology are seldom so

clear cut. Feedback is routine in sports, where a video may be shown with analysis of what worked and more emphasis on what did not work. Any feedback may be disputed or have a negative emotional impact. To be used constructively, is it still feedback? Or does it become “feedforward?”

Feedforward is even more recent terminology. Although the terminology was not used, the concept had its most famous exploration in the 1940s by British air force scientists, led by Norbet Wiener. London was being attacked by German aircraft who tried to make their approach unpredictable with sudden changes of speed and direction (see Galison 1994). Wiener developed systems to predict the unpredictable so that a missile could be launched to intercept the plane on its future path. It was not until the 1970s that cybernetics embraced feedforward (e.g., Lee 1980) and psychology flirted with it (Björkman 1972). In cognitive behavioral psychology, feedforward is recognized as a personal image of future successful behavior, not previously achieved (Dowrick 1991, 2012).

Current Knowledge

Video feedback. While there are many studies using different forms of video with individuals with autism, there are not many using video feedback. For example, a review in 2005 (Ayres and Langone 2005) of 15 studies for social and daily living skills in children with autism, all used variations of video modelling, not feedback. There were peer and self-models, adults, multimedia, and point-of-view modeling. All were successful, although it should be noted that most included other strategies such as discussions and prompts. None used video feedback. More recently, a study by Robinson (2011) found video feedback effective in interventions with the communication skills of four young boys with autism – when the video was used with paraprofessionals delivering the skills training – not used with the children.

Lofthouse et al. (2012) list 19 “complementary and alternative treatments for autism,” from melatonin and B12 to acupuncture and massage. There is no mention of “video feedback,” either because it is “mainstream” rather than

“alternative” or because there are no reports of it. The review notes the “most empirical support” for (mainstream) applied behavior analysis. However, that requires “30–40 h of treatment per week for several years,” thus the impetus for alternative treatments.

There are studies using oral or social feedback but with little promise (unless one considers reinforcement “feedback”). For example, Bedford et al. (2013) found a failure to learn from social feedback in children with autism who had been held back in their vocabulary development around age 2 years. However, they did not mention the use of video or other possibilities.

Reviews of video feedback with general clinical populations do not help with autism. For example, there are numerous reviews of Video Interaction Guidance (e.g., Kennedy et al. 2011) around issues of interest in autism – for example, family studies including child clinical participants. But neither the data nor the references seem to mention “autism.” The authors do provide overall evidence that video feedback can be used effectively to improve parenting skills and parent–child interactions. It seems likely that video feedback, when used constructively for parenting, classroom management, etc., can benefit children and adults with autism. But there is little evidence that video feedback can benefit individuals with autism directly for skills or behavior development, unless it provides feedforward.

Video feedforward. Previous studies have demonstrated the effectiveness of video feedforward to improve various psychological conditions, for example, reading skills (Dowrick et al. 2006; Robson et al. 2015), sport performance (Ste-Marie et al. 2011), communication (Smith et al. 2014), and presentation skills (Murphy and Barry 2016). These are self-modeling interventions that acknowledge the procedure as “feedforward.” Most studies have focused on primary school-age children, often children with special needs, although some have included adults (see Dowrick 2012) and those with elite skills, such as gymnastics (Dowrick 1989) and swimming (Clark and Ste-Marie 2007). In a thesis study of four children aged from 9 to 17 years, Holly Smith produced remarkable results using video feedforward. The youth were all on the autistic spectrum, some severe, with serious phobias

of dogs. At first, two were treated with peer models, two with VSM: the first showed no effects; the second, moderate effects. Then all videos were redone to feature feedforward – with immediate and dramatic results. Phobic reactions disappeared, replaced by enjoyment, in some cases patting the test dogs and one request to own a similar dog (Smith 2018).

In all conditions, the *speed* of behavior change is most notable. In particular, the children with autism in the Smith et al. study, mentioned above, transformed their ability to use picture communication in a matter of days or short weeks. Such communication had previously proved so challenging, they had not gained skills in years of instruction.

Dowrick et al. (2006) implemented video feedforward on children with special needs who have difficulties in reading. Robson et al. (2015) support Dowrick et al.'s (2006) findings by showing the benefit of video feedforward on reading fluency. The participants were 11 children who watched the edited video of themselves reading fluently. The findings showed that feedforward helped them to read more fluently, as measured by accuracy, comprehension, and speed.

Ste-Marie and colleagues (2011) expanded the use of video feedforward to help children improving their performances in sport, including trampoline skills. Smith et al. (2014) used video feedforward to enhance the picture-based communication skills of two children with autism and one adult with Down syndrome in their social environments. Furthermore, Murphy and Barry (2016) showed video feedforward contributions in improving presentation skills of university students. Those findings indicate that video feedforward has been used in various settings for target behaviors which show promise for further research and applications to expand the use in other psychological aspects and settings.

Future Directions

Recognition of feedforward will improve as more self-modeling studies acknowledge the “feedforward”/“positive self-review” distinction. Clearly, interventions with feedforward show

remarkably superior speed and efficacy of behavior change. The literature will be notably advanced by the proper recognition of feedforward as distinct from the carefree or careless reference to feedback as a catch-all category for all video or other recorded replay.

References and Reading

- Ayres, K. M., & Langone, J. (2005). Intervention and instruction with video for students with autism: A review of the literature. *Education and Training in Developmental Disabilities, 40*, 183–196.
- Bedford, R., Gliga, T., Frame, K., Hudry, K., Chandler, S., Johnson, M. H., & Charman, T. (2013). Failure to learn from feedback underlies word learning difficulties in toddlers at risk for autism. *Journal of Child Language, 40*, 29–46.
- Björkman, M. (1972). Feedforward and feedback as determiners of knowledge and policy: Notes on a neglected issue. *Scandinavian Journal of Psychology, 13*, 152–158.
- Clark, S. E., & Ste-Marie, D. M. (2007). The impact of self-as-a-model interventions on children's self-regulation of learning and swimming performance. *Journal of Sports Sciences, 25*, 577–586.
- Dowrick, P. W. (1989). Videotraining strategies for beginners, champions, and injured athletes. In A. A. Turner (Ed.), *Proceedings of American College of Sports Medicine, Alaska Regional Conference 1987* (pp. 1–9). Anchorage: University of Alaska.
- Dowrick, P. W. (1991). Feedforward and self modeling. In P. W. Dowrick (Ed.), *Practical guide to using video in the behavioral sciences*. New York: Wiley.
- Dowrick, P. W. (2012). Self model theory: Learning from the future. *WIREs Cognitive Science, 3*, 215–230. <https://doi.org/10.1002/wcs.1156>.
- Dowrick, P. W., Kim-Rupnow, W., & Power, T. J. (2006). Video feedforward for reading. *The Journal of Special Education, 39*(4), 194–207. <https://doi.org/10.1177/00224669060390040101>.
- Galisson, P. (1994). The ontology of the enemy: Norbert Wiener and the cybernetic vision. *Critical Inquiry, 21*, 228–266.
- Kennedy, H., Landor, M., & Todd, L. (2011). *Video interaction guidance: A relationship-based intervention to promote attunement, empathy and wellbeing*. London: Jessica Kingsley.
- Lee, W. A. (1980). Anticipatory control of postural and task muscles during rapid arm flexion. *Journal of Motor Behavior, 12*, 185–196.
- Lofthouse, N., Hendren, R., Hurt, E., Arnold, L. E., & Butter, E. (2012). A review of complementary and alternative treatments for autism spectrum disorders. *Autism Research and Treatment, 2012*, 870391. <https://doi.org/10.1155/2012/870391>.
- Murphy, K., & Barry, S. (2016). Feed-forward: Students gaining more from assessment via deeper engagement

- in video-recorded presentations. *Assessment & Evaluation in Higher Education*, 41, 213–227. <https://doi.org/10.1080/02602938.2014.99620>.
- Robinson, S. E. (2011). Teaching paraprofessionals of students with autism to implement pivotal response treatment in inclusive school settings using a brief video feedback training package. *Focus on Autism and Other Developmental Disabilities*, 26, 105–118.
- Robson, C., Blampied, N., & Walker, L. (2015). Effects of feedforward video self-modelling on reading fluency and comprehension. *Behaviour Change*, 32(1), 46–58. <https://doi.org/10.1017/bec.2014.29>.
- Smith, H. (2018). *The use of video modelling to reduce fear and teach appropriate response and safe behaviours in children with autism spectrum disorder with a severe fear of dogs*. Unpublished master's thesis, University of Canterbury, Christchurch.
- Smith, J., Hand, L., & Dowrick, P. W. (2014). Video feedforward for rapid learning of a picture-based communication system. *Journal of Autism and Developmental Disorders*, 44(4), 926–936. <https://doi.org/10.1007/s10803-013-1946-0>.
- Ste-Marie, D. M., Vertes, K., Rymal, A. M., & Martini, R. (2011). Feedforward self-modeling enhances skill acquisition in children learning trampoline skills. *Frontiers in Psychology*, 2(2), 155. <https://doi.org/10.3389/fpsyg.2011.00155>.

Video Games and Violence

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Definition

Adolescents with autism spectrum disorder (ASD) spend a considerable amount of their leisure time playing video games to the exclusion of other pursuits. Often the content of these games is violent. It is unclear what impact the consumption of violent video games (VVG) has on the behavior of adolescents with ASD or whether this results in increased risk for contact with the juvenile justice system (JJS).

Historical Background

Following the mass shooting of children and school personnel in Newtown, Connecticut,

there is an emerging body of research specifically focused on video game use in children and adolescents with ASD. The shooter, Adam Lanza, was a young man with ASD who was known to play violent video games for several hours a day, including a first-person-shooter game entitled *School Shooter* (Sedensky 2013). The object of *School Shooter* (Checkerboard Studios 2011) is to murder as many schoolchildren and staff members as possible, using weapons similar to those utilized in the Columbine massacre. After completing the shootings, the gamer is prompted to commit suicide before being apprehended by law enforcement. Adam Lanza walked into Sandy Hook Elementary School, shot and killed 20 schoolchildren and six staff members, and then committed suicide prior to being apprehended by police. Norwegian mass shooter Anders Breivik received a diagnosis of ASD after murdering 77 people. Breivik wrote in his 1500 page manifesto that he utilized the video game *Call of Duty: Modern Warfare 2* and *World of Warcraft* as training videos for the attack. He photographed himself dressed in garb emulating characters from the games including a military uniform, a hazardous material suit, and a special operations soldier. At his trial, he made a statement related to how history would view him which was very similar in nature the script from the game *Call of Duty*. He reported playing both games for up to 15 h a day in preparation for the massacre (Breivik 2009). This raises the question, are violent video games serving as video model for mass shootings perpetrated by people with ASD?

Adolescents with autism spectrum disorders (ASD) spend approximately 4.4 h per day watching television and playing video games. This amount is greater than the time spent pursuing any other leisure activities (Mazurek and Wenstrup 2013). Parents reported considerable difficulty related to the behaviors displayed by their children with ASD when efforts were made to monitor and/or restrict access to media and video games. Longitudinal research by Willoughby et al. (2012) on the effects of VVG consumption on behavior in neurotypical adolescents suggests that continual violent video game play predicts the slope of aggression at a level reaching statistical significance. Moreover, sustained playing of

violent video games reliably predicted higher levels of aggression over time. Their findings suggest that continual nonviolent video (NVG) game play does not significantly predict scores of aggression.

There is limited research that focuses specifically on adolescents with ASD and the relationship between VVG and aberrant behavior. What is unknown is what effect the amount of VVG consumption has on measures of maladaptive behavior in adolescents with ASD and contact with the JJS. Maladaptive behaviors are defined as behaviors that impede participation in activities of daily living and may include property destruction, non-compliance, withdrawal, self-injury, and aggression (Shattuck et al. 2007).

Current Knowledge

Vulnerability to the Impact of Violent Video Games

Specific cognitive, social, and sensory functions are involved in the interaction with video game play (Durkin 2010). Each of these areas is impacted when a person has a diagnosis of ASD (DSM-5, APA 2013). Markey and Markey (2010) examined the personality traits that may make an individual more vulnerable to the effects of violent video games (VVGs). These researchers posited that preexisting dispositions increase the susceptibility of certain individuals to the impact of VVGs. Using the five-factor model of personality traits (McCrae and Costa 1999), they developed a spherical model to determine how the confluence of specific traits may render a person most vulnerable to the deleterious effects of VVGs. The model suggested that three orthogonal traits were moderating variables of VVGs. These traits included high neuroticism, low levels of agreeableness, and low conscientiousness. It is possible that the corollary to this model in the population of people with ASD is ToM deficits, belief persistence, difficulties with syllogistic reasoning, and difficulties with emotional regulation. As a result, they may be particularly susceptible to allegory or images of violent hyperbole, and it is possible that they may take the visual information presented in a VVG at face value and emulate

it. Comorbid psychiatric disorders could potentially serve as a moderating variable exacerbating the person's susceptibility to VVGs.

Violent Video Games and Aberrant Behavior

Anderson et al. (2004) completed a meta-analysis on the robust body of literature that demonstrates a relationship between consumption of virtual violence and actual aggression. The mechanism that accounts for this relationship is the subject of intense interest. A possible explanation for the relationship between VVG and increases in measures of aggression is that virtual violence is abstract and therefore pleasurable, allowing for a level of moral disengagement with violent actions. Many gamers describe a lack of remorse over engaging in virtual violence and derive great pleasure from playing VVGs (Ladas 2002).

As computer graphics become more sophisticated, the characters, both protagonists and antagonists, become more identifiable as social entities. These enhanced graphics allow for a level of immersion that has been linked to heightened enjoyment in game play (Skalski et al. 2006). Some VVGs come with a "blood on" feature, which allows the gamer to control whether they see the results of their carnage. Research has demonstrated that the "blood on" condition results in higher scores on measures of aggression than the "blood off" condition (Farrar et al. 2006). Hartmann and Vorderer (2010) have hypothesized that enjoyment of virtual violence may be contingent upon maximizing rewarding aspects of the game (feelings of power and/or success) while minimizing negative costs (guilt). This cost-benefit equation is likely impacted by both the amount of play and the role violence itself plays within the game. High-use gamers may become inured to images of violence and the resultant suffering displayed within the game (Carnagey et al. 2007). Gamers who are able to justify their behaviors because the antagonist is engaging in morally reprehensible acts may feel absolved of the guilt and distress associated with acts of virtual violence (Opatow 1990).

Another possible antecedent to this increase in aggressive behaviors may be the denial of humanness to the victim. Greitemeyer and McLatchie (2011) examined the role ascribing

or denying humanness to the victim in video games played in measures of violence and aggression. Visual hyperbole is often used to portray the antagonists in VVGs. These larger-than-life characters are imbued with nonhuman characteristics which may lead to the gamer engaging in cognitive distortions, allowing themselves to justify the intensity of their game-playing violence to vanquish the enemy (Smith et al. 2003). Greitemeyer and McLatchie (2011) hypothesized that this dehumanization and justification of violence could transfer from the screen to real life through the type of moral disengagement that assuages guilt and provides a pathway for aggression toward victims (Bandura et al. 1996).

These concepts of desensitization, feelings of power, and denying humanness to victims may have particular relevance to gamers with ASD. Many adolescents with ASD have been the victims of bullying and marginalization (Little 2002). They spend the majority of their leisure time playing video games that may provide their sole source of feelings of empowerment. In the game *One Life* (Chalk 2015), an FPS game, players only have one life and losing it means they are locked out of the game forever. This adds an element of reality to game play heretofore unseen. Players also have the ability to extend mercy or humiliate their victims prior to vanquishing them. The game boasts that no other game will provide players with the feelings of power and control over others. Players are rewarded for ignoring the cries of pain, rage, and appeals for mercy from their victims. In an ultimate act of degradation, players can urinate on their victims. From a behavior analytic perspective, the immediate and continuous schedules of reinforcement for aggression followed by intermittent schedules of reinforcement as the gamer advances through levels have the potential to build extremely durable aggressive mind-sets for the gamer with ASD.

People with ASD often have a concrete and literal sense of right and wrong and as such may find it easier to morally disengage than the neurotypical gamer. The ability to morally disengage requires the gamer to deny humanness to the victim, an act that is facilitated by viewing the

victim's acts as repugnant, thus justifying the vanquishing of the victim. This abates feelings of guilt and distress associated with virtual violence (Hartmann and Vorderer 2010).

Greitemeyer and McLatchie (2011) completed a series of two experiments designed to determine if type of video game (violent, neutral, pro-social) impacted measures of aggressive behavior. Playing VVG increased dehumanization toward others and was linked to increased measures of aggressive behavior. Specifically, the researchers found that players of VVG ascribed fewer human-centric emotions to others, reducing the opposing gamers, in a sense, to the level of animals, machines, or objects. This concept has interesting implications for gamers with ASD, many of whom experience difficulty in facial emotional recognition as the result of the social difficulties inherent in their diagnosis (O'Connor et al. 2005).

The role of VVG as a factor in conferring increased risk of aberrant behavior in adolescents with ASD is unclear. The answer to this question may serve to explain the distress and problem behavior exhibited by people with ASD when efforts are made to place parameters on their gaming (Mazurek and Wenstrup 2013). Engelhardt et al. (in press) recently completed a study in which adults with ASD as well as adults who were neurotypical were randomized to different video game playing conditions: VVG versus NVG. Measures of aggressive thought accessibility, aggressive affect, and aggressive behavior were taken following the consumption of VVGs. Compared with their neurotypical peers, adults with ASD were not differentially impacted by viewing VVG (Engelhardt et al. in press). These findings may have limited applicability to the current study as the adults were exposed to only three brief video game sessions (15, 10, and 10 min) for a total of 35 min of game play, and this figure is considerably less than the average 2.4 h of daily game play reported by parents of teenagers with ASD (Mazurek and Wenstrup 2013). Therefore, there may be something of a dose-response relationship in which long-term consumption of VVGs has a different effect on gamers with ASD than acute, short-term consumption.

A study by Krcmar and Lachlan (2009) examined the association between length of VVG play and aggression and found a positive relationship between habitual game play and physical aggression. A study which examined the impact of VVG on measures of aggression found that there were significant increases from pre-exposure baselines after participants played first-person-shooter games (Barlett et al. 2007). Thus, long-term exposure to violent images may lead to an increase in aggression as the result of regularly primed aggressive mind-sets.

A pilot study examined the relationship between amount of VVG consumption, scores on the subscales of the Aberrant Behavior Checklist (ABC), and contact with the JJS in a group of adolescents with ASD and comorbid psychiatric conditions who were placed in a Neuropsychiatric Special Care Unit (Sperry 2016). There was a considerable range in the amount of weekly game play (3–28 h per week) with an average of 8.5 h. The majority of these games (67%) contained violent content. There was no statistically significant relationship between amount of VVG and contact with the JJS which may be a function of the NSC serving as a diversionary, alternative placement to the JJS. Parents setting limits on the video gaming were consistently listed as precipitating factors in the outbursts that led to placement in the NSC. Adolescents who played the most violent games the most frequently had the highest scores on the ABC subscales of hyperactivity, irritability, lethargy/social withdrawal, and inappropriate speech.

Internet Addiction

There is a growing body of literature on the Internet gaming disorder (IGD), which has been identified in the emerging measures and models section of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)* as a disorder that merits additional clinical research prior to being included in the main text (American Psychiatric Association 2013). People with IGD experience clinically significant levels of distress when limits are put on their gaming, but they also experience this distress when playing compulsively, which

compromises other activities of daily life including work and school.

In one of the largest studies to date ($N = 7069$), Grüsser et al. (2006) found that nearly 12% of study participants met diagnostic criteria for addiction (including craving, feelings of relief when playing, and increasing game play) relative to their gaming behaviors. Based on their responses to an online questionnaire, participants fell into two groups: pathological gamers who played an average of 4.70 h per day and non-pathological gamers who played an average of 2.49 h per day. There were statistically significant between-group differences, with the pathological gamers reporting greater anticipated relief of withdrawal when playing. They also demonstrated statistically significant higher levels of craving than non-pathological gamers. Addiction may be the result of the neurological response to the positive reinforcement inherent in the games and the success the individual experiences while playing. During their time away from gaming, they experience feelings associated with drug withdrawal.

Circumscribed interests may be part of the constellation of characteristics that can render people with ASD more vulnerable to the impact of VVG. Klin et al. (2007) first posited the idea that video game addiction may be an example of a circumscribed interest. In a study by Romano et al. (2014), the relationship between scores on the autism spectrum quotient questionnaire (AQ; Baron-Cohen et al. 2001), scores on the Spielberger trait anxiety inventory (STAI-T; Spielberger 1983), scores on the Beck depression index (BDI; Beck et al. 1961), and scores on the Internet addiction test (IAT; Young 1998) were examined. The IAT measures the extent to which activities of daily living are compromised by time spent on the Internet. Statistically, significant relationships were found between scores on the AQ, STAI-T, and degree of Internet addiction. People with higher AQ scores, meaning they had more autistic traits, tended to score high on the IAT with anxiety serving as a moderating variable. Interestingly, participants with high AQ scores and low STAI-T scores reported the highest levels of Internet addiction. As the IAT does not specifically

measure how time is spent on the Internet, it is difficult to determine if the participants with high AQ scores and high anxiety were pursuing special interests, engaging in social media, or playing VVGs. In other words, some of the aforementioned activities may have a soothing effect while others may serve to escalate anxiety.

Mazurek et al. (2012) completed a study based on data from the National Longitudinal Transition Study-2 (NLTS-2). The vast majority of adolescents with ASD (64.2%) spent the bulk of their free time consuming television and video games. When video games and television viewing were separated, 41.4% spent the greater part of their free time consuming video games. When compared to adolescents with other disabilities, teens with ASD spent more time playing video games, and these differences reached the level of statistical significance. The amount of consumption created considerable interference with their activities of daily living and functioning. Only 28% of neurotypical teens qualify as high consumers of video games compared to 60.3% of adolescents with ASD. The greatest predictors of high use for teens with ASD included a higher IQ and access to technology in their homes.

Game Genres

In 2014, half of the ten top-selling video games were classified as VVG (Kain 2015). These include first-person-shooter games (FPSG) and role-playing games (RPG). First-person-shooter games (FPSG) allow gamers to immerse themselves into a three-dimensional world with the console screen serving as their means of experiencing the action from the point of view of the shooter (Schneider 2004). Contrary to popular belief, FPSGs are not always played in isolation. A study by Jansz and Tanis (2007) found that the majority of gamers who played FPSGs (80%) joined a clan, which allowed them to collaborate and compete with gamers at similar skill levels. This suggests there is a social motive to the play of neurotypical gamers, though it is unclear if people with ASD play FPSG in the same way or for the same purposes. Barlett et al. (2007) found significant increases in levels of aggression in participants after playing

FPSGs for a mere 15 min. They propose that priming for aggression occurs when a person is exposed to VVG which triggers aggressive thoughts and ultimately aggressive actions. This high level of primed aggression is exacerbated by the amount of blood and gore that accompany an aggressive action within the game.

Role-playing games (RPGs) differ from FPSGs in that they allow the person with ASD to immerse themselves in the game by taking on an avatar within a fantasy world, setting out on quests, and exploring against the backdrop of a story line or dialogue. Gamers who engage with RPGs tend to play longer (23–25 h a week versus 16 h per week for FPS gamers) (Ng and Wiemer-Hastings 2005; Jansz and Tanis 2007) and develop substantial attachments to their character (Lewis et al. 2008). These character attachments are formed as the gamer's actions shape the development of the story line, and the gamer becomes more immersed and integrated with his/her avatar. In their study on character attachment, Lewis et al. (2008) found that gamers with stronger character attachments were motivated by fantasy seeking, social interaction, and diversion. This raises a question regarding the degree to which a gamer with ASD would form attachments with a character in an RPG (Kowert and Oldmeadow 2014). Does a competent avatar allow a person with ASD to feel more competent and less socially anxious? Does the avatar become the surrogate friend in the absence of real-world friendships, thus limiting real-world social experiences and exacerbating social impairments?

Mazurek and Engelhardt (2013) examined the relationship between video game play and scores on measures of problem behavior in a sample of 169 boys with a diagnosis of ASD. Video games were identified as fitting into the category of RPG or FPSG. In this study, the amount of video game consumption did not demonstrate an association with maladaptive behaviors. However, there was a statistically significant positive relationship between type of game (RPGs) and scores on measures of oppositional behavior. There was also a positive relationship, though not reaching the level of statistical significance, between FPSGs and oppositional

behaviors. Additionally, there was a statistically significant positive relationship between FPSGs and addictive gaming behaviors.

Chung et al. (2015) examined the effects of active video game play (AVG) on the social behaviors of children with ASD. In this single-subject design study, children with ASD played with their siblings with measures taken on joint positive affect, reciprocal conversation, and aggression. Primary analysis suggested that AVGs have an inconsistent effect on the socialization of children with ASD and their siblings. Of particular interest, however, was a finding in the supplementary analysis of the augmented reality (AR) condition. The AR was originally conceived to provide a bridge between sedentary and active game play and to introduce the more cooperative style of play with the sibling. Rather than using an avatar, the AR condition used a silhouette or photo-realistic image of the gamer. The AR condition was statistically significantly more preferred by the majority of participants with ASD, and they reported a more positive experience with the AR condition.

Social learning theory (Bandura and Walters 1977; Bandura 1997) has demonstrated the impact of modeling on children's acquisition of social, emotional, behavioral, and functional skills. According to Bandura's social learning theory, children pay particular attention to models who share their salient demographic characteristics and models perceived to be competent. These models can continue to influence the child beyond the context of the original observation and do not require reinforcement for behavioral modeling to occur. Social learning theory may explain why the AR conditions in the Chung et al. (2015) study were preferable to the children with ASD. It may also begin to explain the underlying mechanism for VVGs as potential video models of aberrant behavior in adolescents with ASD.

Violent Video Games as Video Models

Ferguson and Kilburn (2010) have raised the issue of selection to explain the relationship between VVG play and aggression. It is possible that people who are naturally more aggressive play more violent video games. Przybylski et al. (2009)

found that while the level of violence in a video game did not enhance enjoyment, those with higher scores on measures of trait aggression strongly preferred VVGs over NVG, and this was strongly predictive of their future choices of VVGs. Alternately, violent video games may result in increases in aggressive behavior. There is ample research that supports the finding that observation of violence in the home, community, and school is detrimental to children and increases their risk of engaging in violent behavior (Cummings et al. 2010; Henrich and Shahar 2013). Bushman and Huesmann (2013) support the position that there is an interaction between aggression-prone personalities and social cognitive learning, including observational learning. Randomized, controlled studies have demonstrated that decreasing access to media violence and teaching children rules against mimicking violence they see have both shown a decrease in aggression (Moller et al. 2012).

Video modeling is an evidence-based strategy utilized to teach pro-social behaviors to people with ASD that capitalizes on the strong visual learning skills of this population. Point-of-view modeling enables the person with ASD to see what the behavior looks like from the standpoint of the person engaging in the target behavior. These pro-social behaviors can include ending a conversation appropriately, asking for help, or entering into an ongoing conversation (Bellini and Akullian 2007). Video modeling allows for the repeated and consistent use of models that do not vary from time to time as they would likely do with a live model and has been successful in teaching and maintaining imitation skills in children with ASD (Cardon and Wilcox 2011). Video modeling is an effective tool for teaching pro-social behaviors, though it is not clear to what extent VVGs are impacting aberrant behavior by providing antisocial models for adolescents with ASD. Strong visual skills, circumscribed interests, difficulties in syllogistic reasoning, deficits in ToM, and problematic/addictive gaming behaviors may serve as a confluence of vulnerabilities rendering the adolescent with ASD vulnerable to emulating the behaviors viewed during hours of VVG playing.

Future Directions

Future research should consider the factors that may be contributing to mass shootings perpetrated by young men with ASD and comorbid psychiatric disorders who spend significant amounts of time playing VVGs. Does their propensity toward visual learning make them more vulnerable to violent images? Are deficits in perspective taking that make it difficult for them to consider how their behavior impacts others, being exacerbated by the dehumanization of victims within video games? Is this violent modeling generalizing to real-life behaviors? Are some people with ASD more impacted by violent images than others and if so, what variables moderate that impact? Are the feelings of empowerment provided by video games serving to galvanize aberrant behavior?

It is incumbent upon the ASD community, including people with ASD, advocates, families, and professionals, to demonstrate an understanding of the impact of violence consumption on people with ASD through the development of reasonable game play guidelines. Components of VVGs that may make them appealing to the player (pace, level of competitiveness, graphics, frequency of rewards) could be identified, and alternative games could be substituted that would still allow the person with ASD access to an equivalent gaming experience without subjecting them to graphic violence. This may serve as a mechanism for families to provide acceptable gaming alternatives thus avoiding the resultant problem behaviors that come with placing restrictions on their video game play.

While these questions are being investigated, we must consider the amount and type of violence adolescents with ASD are routinely consuming and work toward developing nonviolent alternative interests both within and outside of the digital world.

See Also

- ▶ [Aberrant Behavior Checklist](#)
- ▶ [Internet Safety](#)
- ▶ [Video Modeling/Video Self-Modeling](#)
- ▶ [Violence and ASD](#)

References and Reading

- Adachi, P. J., & Willoughby, T. (2011). The effect of violent video games on aggression: Is it more than just violence? *Aggression and Violent Behavior, 16*, 55–62.
- Aman, M. G., Singh, N. N., Stewart, A. W., & Field, C. J. (1985). The aberrant behavior checklist: A behavior rating scale for the assessment of treatment effects. *American Journal of Mental Deficiency, 89*, 485–491.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). <http://dx.doi.org/10.1176/appi.books.9780890425596.910646>
- Anderson, C. A., & Bushman, B. J. (2001). Effects of violent video games on aggressive behavior, aggressive cognition, aggressive affect, physiological arousal, and prosocial behavior: A meta-analytic review of the scientific literature. *Psychological Science, 12*(5), 353–359.
- Anderson, C. A., Carnagey, N. L., Flanagan, M., Benjamin, A. J., Eubanks, J., & Valentine, J. C. (2004). Violent video games: Specific effects of violent content on aggressive thoughts and behavior. *Advances in Experimental Social Psychology, 36*, 200–251.
- Bandura, A. (1997). Editorial. *American Journal of Health Promotion, 12*(1), 8–10.
- Bandura, A., & Walters, R. H. (1977). *Social learning theory*. General Learning Press, NY.
- Bandura, A., Barbaranelli, C., Caprara, G. V., & Pastorelli, C. (1996). Mechanisms of moral disengagement in the exercise of moral agency. *Journal of Personality and Social Psychology, 71*(2), 364.
- Barlett, C. P., Harris, R. J., & Baldassaro, R. (2007). Longer you play, the more hostile you feel: Examination of first person shooter video games and aggression during video game play. *Aggressive Behavior, 33*(6), 486–497.
- Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory of mind*. Cambridge, MA: The MIT Press.
- Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from Asperger syndrome/high-functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders, 31*(1), 5–17.
- Barry-Walsh, J. B., & Mullen, P. E. (2004). Forensic aspects of Asperger's Syndrome. *Journal of Forensic Psychiatry and Psychology, 15*, 96–107.
- Beck, A. T., Ward, C., & Mendelson, M. (1961). Beck depression inventory (BDI). *Archives of General Psychiatry, 4*(6), 561–571.
- Bellini, S., & Akullian, J. (2007). A meta-analysis of video modeling and video self-modeling interventions for children and adolescents with autism spectrum disorders. *Exceptional Children, 73*(3), 264–287.
- Benedetti, W. (2007, April 20). Were video games to blame for the massacre? Pundits rushed to judge industry, gamers in the wake of the shooting. *MSNBC*. Retrieved from <http://www.nbcnews.com/id/18220228/ns/>

- technology_and_science-games/t/were-video-games-bla-me-massacre/#.VmcX32fru70
- Breivik, A. B. (2009). 2083: A European Declaration of Independence. Unpublished manifesto. Retrieved from https://fas.org/programs/tap/_docs/2083_A_European_Declaration_of_Independence.pdf
- Bushman, B. J., & Huesmann, L. R. (2013). Twenty-five years of research on violence in digital games and aggression revisited: A reply to Elson and Ferguson (2013). *European Psychologist*. Advance online publication. doi:10.1027/1016-9040/a000164.
- Cardon, T. A., & Wilcox, M. J. (2011). Promoting imitation in young children with autism: A comparison of reciprocal imitation training and video modeling. *Journal of Autism and Developmental Disorders*, 41(5), 654–666.
- Camagey, N. L., Anderson, C. A., & Bushman, B. J. (2007). The effect of video game violence on physiological desensitization to real-life violence. *Journal of Experimental Social Psychology*, 43(3), 489–496.
- Chalk, A. Review of the video game *One Life*, produced by One Life, 2015 (10.15.15). Retrieved from <http://www.pcgamer.com/one-life-is-a-multiplayer-fps-that-locks-you-out-forever-when-you-die/>
- Checkerboard Studios. (2011). *School Shooter North American Tour*: Half-Life 2. Checkerboard Studios. Lulzy Games. Information retrieved from https://en.wikipedia.org/wiki/School_Shooter:_North_American_Tour_2012
- Chung, P. J., Vanderbilt, D. L., & Soares, N. S. (2015). Social behaviors and active videogame play in children with autism spectrum disorder. *Games for Health Journal*, 4, 225–234.
- Cummings, E. M., Merrilees, C. E., Schermerhorn, A. C., Goecke-Morey, M. C., Shirlow, P., & Cairns, E. (2010). Testing a social ecological model for relations between political violence and child adjustment in Northern Ireland. *Development and Psychopathology*, 22, 405–418.
- Durkin, K. (2010). Videogames and young people with developmental disorders. *Review of General Psychology*, 14(2), 122–140.
- Engelhardt, C. R., Mazurek, M. O., Hilgard, J., Rouders, J. N., & Bartholow, B. D. (in press). Effects of violent video game exposure on aggressive behavior, aggressive thought accessibility, and aggressive affect among adults with and without autism spectrum disorder. Retrieved from http://pcl.missouri.edu/sites/default/files/Engelhardt.etal_PsySci.2015.pdf
- Farrar, K. M., Krcmar, M., & Nowak, K. L. (2006). Contextual features of violent video games, mental models, and aggression. *Journal of Communication*, 56(2), 387–405.
- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, 39, 175–191.
- Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods*, 41, 1149–1160.
- Ferguson, C. J., & Kilburn, J. (2010). Much ado about nothing: The misestimation and over interpretation of violent video game effects in Eastern and Western Nations: Comment on Anderson et al. (2010). *Psychological Bulletin*, 136, 174–178. doi:10.1037/a0018566.
- Field, A. (2013). *Discovering statistics using IBM SPSS statistics*. Los Angeles: Sage.
- Gabriels, R. L., Agnew, J. A., Beresford, C., Morrow, M. A., Mesibov, G., & Wamboldt, M. (2012). Improving psychiatric hospital care for pediatric patients with autism spectrum disorders and intellectual disabilities. *Autism Research and Treatment*, 2012, 1–7.
- Greitemeyer, T., & McLatchie, N. (2011). Denying humanness to others a newly discovered mechanism by which violent video games increase aggressive behavior. *Psychological Science*, 22, 659.
- Grüsser, S. M., Thalemann, R., & Griffiths, M. D. (2006). Excessive computer game playing: Evidence for addiction and aggression? *Cyberpsychology & Behavior*, 10(2), 290–292.
- Harris, P. A., Taylor, R., Thielke, R., Payne, J., Gonzalez, N., & Conde, J. G. (2009). Research electronic data capture (REDCap) – A metadata-driven methodology and workflow process for providing translational research informatics support. *Journal of Biomedical Informatics*, 42(2), 377–381.
- Hartmann, T., & Vorderer, P. (2010). It's okay to shoot a character: Moral disengagement in violent video games. *Journal of Communication*, 60(1), 94–119.
- Health Insurance Portability and Accountability Act. Pub. L. No. 104–191, § 264, 110 Stat. 1936.
- Hellings, J. A., Nickel, E. J., Weckbaugh, M., McCarter, K., Mosier, M., & Schroeder, S. R. (2005). The overt aggression scale for rating aggression in outpatient youth with autistic disorder: Preliminary findings. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 17(1), 29–35.
- Henrich, C. C., & Shahar, G. (2013). Effects of exposure to rocket attacks on adolescent distress and violence: A 4-year longitudinal study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(6), 619–627.
- Hillbrand, M., & Sondik, T. (2012). Commentary: Treatment and violence risk mitigation in high-functioning autism spectrum individuals. *Journal of the American Academy of Psychiatry and the Law Online*, 40(2), 191–192.
- Jansz, J., & Tanis, M. (2007). Appeal of playing online first person shooter games. *Cyberpsychology & Behavior*, 10(1), 133–136.
- Kain, E. (2015). The top ten best-selling video games of 2014. *Forbes Magazine*. <http://www.forbes.com/sites/#/sites/erikkain/2015/01/19/the-top-ten-best-selling-video-games-of-2014/>
- Klin, A., Danovitch, J. H., Merz, A. B., & Volkmar, F. R. (2007). Circumscribed interests in higher functioning individuals with autism spectrum disorders: An exploratory study. *Research and Practice for Persons with Severe Disabilities*, 32(2), 89–100.
- Kowert, R., & Oldmeadow, J. A. (2014). Playing for social comfort: Online video game play as a social

- accommodator for the insecurely attached. *Computers in Human Behavior*, 53, 556.
- Krcmar, M., & Lachlan, K. (2009). Aggressive outcomes and videogame play: The role of length of play and the mechanisms at work. *Media Psychology*, 12(3), 249–267.
- Ladas, M. (2002). *Brutal players? Uses and effects of violent video games*. Frankfurt, M: Peter Lang.
- Laurent, A. C., & Rubin, E. (2004). Challenges in emotional regulation in Asperger syndrome and high-functioning autism. *Topics in Language Disorders*, 24(4), 286–297.
- Leatherdale, S. T., & Wong, S. (2009). Association between sedentary behavior, physical activity, and obesity: Inactivity among active kids. *Preventing Chronic Disease*, 6(1), A26.
- Lerner, M. D., Haque, O. S., Northrup, E. C., Lawer, L., & Bursztajn, H. J. (2012). Emerging perspectives on adolescents and young adults with high-functioning autism spectrum disorders, violence, and criminal law. *Journal of the American Academy of Psychiatry and the Law Online*, 40(2), 177–190.
- Lewis, M. L., Weber, R., & Bowman, N. D. (2008). “They may be pixels, but they’re MY pixels:” Developing a metric of character attachment in role-playing video games. *Cyberpsychology & Behavior*, 11(4), 515–518.
- Linkous, D., & Carter, K. F. (2009). Responding to the shootings at Virginia Tech: Planning and preparedness. *Journal of Emergency Nursing*, 35, 321–325.
- Little, L. (2002). Middle-class mother’s perceptions of peer and sibling victimization among children with Asperger’s syndrome and nonverbal learning disorders. *Issues in Comprehensive Pediatric Nursing*, 25, 43–57. doi:10.1080/014608602753504847.
- Mandell, D. S. (2008). Psychiatric hospitalization among children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(6), 1059–1065.
- Markey, P. M., & Markey, C. N. (2010). Vulnerability to violent video games: A review and integration of personality research. *Review of General Psychology*, 14(2), 82.
- Matthews, B., & Ross, L. (2010). Research methods: A practical guide for the social sciences. London: Pearson.
- Mazurek, M. O., & Engelhardt, C. R. (2013). Video game use in boys with autism spectrum disorder, ADHD, or typical development. *Pediatrics*, 132(2), 260–266.
- Mazurek, M. O., & Wenstrup, C. (2013). Television, video game and social media use among children with ASD and typically developing siblings. *Journal of Autism and Developmental Disorders*, 43(6), 1258–1271.
- Mazurek, M. O., Shattuck, P. T., Wagner, M., & Cooper, B. P. (2012). Prevalence and correlates of screen-based media use among youths with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42, 1757–1767.
- McCrae, R. R., & Costa Jr., P. T. (1999). A five-factor theory of personality. *Handbook of Personality: Theory and Research*, 2, 139–153.
- Moller, I., Krahe, B., Busching, R., & Krause, C. (2012). Efficacy of an intervention to reduce the use of media violence and aggression: An experimental evaluation with adolescents in Germany. *Journal of Youth and Adolescence*, 41, 105–120.
- Morsanyi, K., & Handley, S. J. (2012). Reasoning on the basis of fantasy content: Two studies with high-functioning autistic adolescents. *Journal of Autism and Developmental Disorders*, 42(11), 2297–2311.
- National Disability Rights Network. (2007). *Youth with disabilities in the juvenile justice system*. Retrieved from http://www.ndrn.org/images/Documents/Issues/Juvenile_Justice/NDRN_JDAI_handout_prevalence_92607.pdf
- Ng, B. D., & Wiemer-Hastings, P. (2005). Addiction to the internet and online gaming. *Cyberpsychology & Behavior*, 8(2), 110–113.
- O’Connor, K., Hamm, J. P., & Kirk, I. J. (2005). The neurophysiological correlates of face processing in adults and children with Asperger’s syndrome. *Brain and Cognition*, 59(1), 82–95.
- Opotow, S. (1990). Moral exclusion and injustice: An introduction. *Journal of Social Issues*, 46(1), 1–20.
- Przybylski, A. K., Ryan, R. M., & Rigby, C. S. (2009). The motivating role of violence in video games. *Personality and Social Psychology Bulletin*, 35(2), 243–259.
- Puth, M. T., Neuhauser, M., & Ruxton, G. D. (2014). Effective use of Pearson’s product-moment correlation coefficient. *Animal Behaviour*, 93, 183–189.
- Romano, M., Truzoli, R., Osborne, L. A., & Reed, P. (2014). The relationship between autism quotient, anxiety, and internet addiction. *Research in Autism Spectrum Disorders*, 8(11), 1521–1526.
- Samaniego, F., Nordhaus, W., DaVanzo, J., Bradburn, N., Altonji, J., & Ver Ploeg, M. (Eds.). (2000). *Time-use measurement and research: Report of a workshop*. Washington, DC: National Academies Press.
- Schneider, E. F. (2004). Death with a story. *Human Communication Research*, 30(3), 361–375.
- School Shooter North American Tour 2012. (2011). Half-Life 2. *Checkerboard Studios. Lulzy Games*. Information retrieved from https://en.wikipedia.org/wiki/School_Shooter:_North_American_Tour_2012
- Sedensky, S. J. (2013). *Report of the state’s attorney for the judicial district of Danbury on the shootings at Sandy Hook Elementary School and 36 Yogananda Street, Newtown, Connecticut on December 14, 2012*. Danbury: Office of the Attorney General.
- Shapiro, E. (2015, October 17). Oregon college shooting survivor describes gunman as acting ‘Like he was playing a video game’. *ABC News*. Retrieved from <http://abcnews.go.com/US/oregon-college-shooting-survivor-describes-gunman-acting-playing/story?id=34542192>
- Shattuck, P. T., Mailick-Seltzer, M., Greenberg, J. S., Orsmond, G. I., Bolt, D., Kring, S., Lounds, J., & Lord, C. (2007). Change in autism symptoms and maladaptive behaviors in adolescents and adults with autism spectrum disorder. *Journal of Autism and Developmental Disabilities*, 37, 1735–1747.
- Skalski, P., Lange, R., & Tamborini, R. (2006). Mapping the way to fun: The effect of video game interfaces on

- presence and enjoyment. In *Proceedings of the ninth annual international workshop on presence*, Conference Proceedings. University of Minnesota (pp. 63–64).
- Smith, S. L., Lachlan, K., & Tamborini, R. (2003). Popular video games: Quantifying the presentation of violence and its context. *Journal of Broadcasting & Electronic Media*, 47(1), 58–76.
- Sperry, L. (2016). *Analysis of violent video game consumption, scores on the aberrant behavior checklist and contact with the juvenile justice system: Retrospective chart review of neuropsychiatric special care populations*, Unpublished Dissertation.
- Spielberger, C. D. (1983). *State-trait anxiety inventory STAI (form Y)*. Palo Alto: Consulting Psychologists Press.
- United States Census Bureau. (2014). State and county quick facts. Retrieved from <http://quickfacts.census.gov/qfd/states/08000.html>
- Werling, D. M., & Geschwind, D. H. (2013). Sex differences in autism spectrum disorders. *Current Opinion in Neurology*, 26(2), 146.
- Willoughby, T., Adachi, P. J., & Good, M. (2012). A longitudinal study of the association between violent video game play and aggression among adolescents. *Developmental Psychology*, 48, 1044–1057.
- Young, K. (1998). *Caught in the net*. New York: Wiley.
- Yudofsky, S. C., Silver, J. M., Jackson, W., Endicott, J., & Williams, D. (1986). The Overt Aggression Scale for the objective rating of verbal and physical aggression. *The American Journal of Psychiatry*, 143, 35.

Video Games, Use of

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Definition

Video games are electronic media with three key attributes: they display content visually, they are interactive, and they are typically used for entertainment or have entertainment-like qualities. Video games encompass a wide array of formats, applications, and designs, ranging from simple hand-held gaming devices depicting black and white rasterized characters, to fully immersive virtual reality simulators.

In general, video games are separate from electronic screen media, because electronic screen

media do not necessarily require an interactive component. Video games are separate from computer-assisted instruction and computer-based intervention, as video games are typically associated with entertainment, and computer-assisted instruction and computer-based intervention involve a therapeutic or didactic element or intention. Similarly, a distinction can be drawn between software, in general, and video games (which can be a specific type of software), as the goal in most software is typically to facilitate an external goal in the most efficient way possible (e.g., summing numbers, looking up information on the internet, or reading an Encyclopedia of Autism entry); the goal for video games typically lies within the game itself or is defined by the game (e.g., to “win” the game, to solve a puzzle, or to “beat” another player).

It is important to note that the line between video games and other forms of interactive electronic media is somewhat indistinct. It could be argued that some computer-based interventions are, in fact, video games, since they rely on games (in particular, other video games) as a basis for design (e.g., the Memory game, played with emotional faces, for individuals with autism). Nevertheless, the term “video game” is sometimes avoided by researchers who wish to highlight the advantages and potential of this media format while avoiding the perceived negative connotations associated with, for example, video game violence or obsessive video game play that serves no positive remedial goal.

Historical Background

History of Video Games

One of the first reports of video games was filed in a patent, “Cathode-ray tube amusement device,” in 1947 by Goldsmith and Ray (1948; Mitra 2010). This patent described a game system in which a cathode-ray tube was used as the face of the console, displaying a single moving point, representing a missile fired toward drawn figures overlaid upon the tube. Over the next two decades, other video games slowly emerged, driven by advancements in computer technologies and increasing sophistication with computer

programming. Notable games during this time included “OXO,” a tic-tac-toe program written as part of a University of Cambridge Ph.D. dissertation, by Alexander Douglas in 1952; “Tennis for Two,” a tennis game played on an oscilloscope screen at Brookhaven Nation Labs, by William Higinbotham in 1958; and “Spacewar!,” a two-player vector-based space combat game designed to showcase the Digital Equipment Corporation’s recently developed computer, the PDP-1, by MIT students Steve Russell and others in 1962 (Burnham 2001; Donovan 2010).

Through the 1960s, video games were primarily viewed as sophisticated but impractical novelties, due to their size and expense. However, as the cost and size of electronics and microprocessors began to rapidly decrease, video games became more viable as a form of entertainment. In the 1970s, beginning with the successes of coin-operated arcade systems such as Pong, video games came into vogue and quickly became significant segments of the entertainment market (Donovan 2010). The late 1970s saw commercial successes by companies such as Atari, and by the early 1980s video games had moved into the home in the form of consoles and personal computers, ushering in the modern era where video games have never diminished from public consciousness (Donovan 2010; Kent 2001).

Video games have continued to innovate and redefine themselves (Donovan 2010). In the early 2000s, internet games became popular, allowing players to participate in games with tens or thousands of other players at the same time. Simultaneously, mobile devices like cell phones began to allow gaming on devices that were cheap and portable and therefore ubiquitous. Today, this interconnectivity and ubiquity of access define some of the most modern aspects, and modern challenges, of video game technology.

Types of Video Games

The taxonomy of video games is a complex subject (e.g., see Apperley 2006; Wolf 2002). However, one useful classification scheme involves the discussion of video game “genres.” A review by

Yuan et al. (2010) identifies the following (non-exhaustive and non-mutually exclusive) categories of games: (1) first-person shooter, (2) strategy games, (3) sports games, (4) role-playing games, (5) puzzle games, (6) racing games, (7) dance/rhythm games, and (8) adventure games.

Another distinction in video games is between “serious video games” versus “non-serious video games.” Serious video games represent games intended to fulfill a role other than (or in addition to) pure entertainment, with the typical assumption that they will foster some specific skill acquisition or broader learning (Rego et al. 2010; Susi et al. 2007). Others, however, argue that such a distinction is artificial, and that even games not originally intended to be “serious” can be used for serious intentions, such as education (Breuer and Bente 2010; Squire 2003).

The Demographics of Video Games and Video Game Players

Today, video games collectively represent a more than \$67-billion-a-year industry globally, putting the industry on equal footing with the music and film industries (Gartner 2011). In the USA, video game platforms are popular and widespread. Consumer spending for the video game industry in the USA in 2010 was \$25 billion, with 72% of households playing (Entertainment Software Association 2011).

More males than females play video games, but recent estimates by the Entertainment Software Association (2011) suggest that a sizeable 42% of game players are women and that females aged 18 and older are a group of game players three times larger than males 17 years of age and younger. Despite this, it is well known that women, along with minorities, are underrepresented as characters in video games (Terlecki et al. 2010; Williams et al. 2009). Furthermore, research has found that the representation of women in video games is often sexually stereotyped, potentially contributing to feelings of inadequacy and the internalization of stereotypes, especially by young females (Dietz 1998; Dill and Thill 2007).

Positive and Negative Effects of Video Games

Video games can have both positive and negative qualities (Kirriemuir and McFarlane 2004; Lee and Peng 2006; Mitchell and Savill-Smith 2004) and, as with other forms of media, video games have had their share of controversy. One of the first video games to generate bad publicity for the fledgling industry was the video game *Death Race* (Exidy 1976), a game where players controlled a car and received points for driving over people (Donovan 2010). This began a long disagreement over the role of violence in video games in potentially encouraging or exacerbating aggressive or violent tendencies, a disagreement that continues to this day (Mitchell and Savill-Smith 2004). While some meta-analytical research suggests that violent video game play is causally linked to increases in real-world aggression and decreases in prosocial behavior (Anderson and Bushman 2001; Craig 2004), other researchers challenge these assertions (Ferguson 2007, 2010). Some argue that violent video games might be a cathartic experience for players, allowing players to release pent-up aggression, anxieties, and energy, though evidence to this effect is scant (Lee and Peng 2006).

Similarly, the widespread popularity of video games and internet video games, in particular, has resulted in increasing focus on the phenomenon of video game addiction (Young 1998). A recent review of internet gaming addiction (Kuss and Griffiths 2011) suggests that individuals with video game addictions share similarities with individuals with other forms of addiction in terms of neural activation patterns (e.g., see Ko et al. 2009), atypical personality traits (e.g., bipolar disorder and depression; see Shapira et al. 2000; Young and Rogers 1998), and poor maintenance of real-world social relationships (Allison 2006). Yet, Kuss and Griffiths (2011) point out that gaming addiction exists on a spectrum of severity, where most video game players do not exhibit concerning levels of video game play.

The flip side to video game addiction is that the games are designed, of course, to be as fun as possible. While addictions that interfere with daily living are certainly an unfortunate consequence, they can be seen as the natural extension

of the continuum of the qualities that make certain games popular, namely, their addictive nature. What makes one game more playable and replayable than another has been a constant question for video game designers (Salen and Zimmerman 2004). One useful perspective involves the concept of “flow” (Csikszentmihalyi 1991). Flow is a state in which a person operates at his or her peak ability, with focused motivation and full involvement dedicated to overcoming or completing a challenging task. The ability to evoke flow in its users has been described as an attribute of highly successful video games (Chen 2007), with play that walks a fine line between anxiety and boredom (Cowley et al. 2008). These flow states have been described as extremely meaningful, enjoyable, and illuminating experiences; the ability of video games to instill a flow state into players could, from this point of view, be described as a positive experience. For example, Bowman (1982; as discussed in Squire 2003) analyzed the Pac-Man video game in the context of flow, and identified qualities that promoted flow states during its gameplay: “skills and challenges are progressively balanced, goals are clear, feedback is immediate and unambiguous, and relevant stimuli can be differentiated from irrelevant stimuli.” Bowman suggested that educators should aim for these qualities to design learning environments that are engaging.

Yet, detractors of video games would be quick to point out that video game play involves consumption rather than creation, resulting in no tangible benefits to either the player or the world at large. Such arguments parallel historical arguments against other forms of media, such as television viewing, and there is a real concern that, to some extent, excessive video game play may be displacing other activities such as schoolwork. These concerns are supported by some studies, especially for violent games played by excessive video game players (Gentile and Gentile 2007; Harris and Williams 1985), but seem to be a lesser concern for the majority of video game players, who do not play excessively (Christopher 2011; Gentile and Stone 2005). For example, a study by Durkin and Barber (2002), which looked at 1,304 16-year-old high school students, found that video

game play was associated with positive attributes (e.g., positive mental health and friendship networks) rather than negative ones (poor school performance). A similar concern has been raised regarding the possibility that video game play may be displacing sports and other physical recreation (Kirriemuir and McFarlane 2004; Vandewater et al. 2004). On the other hand, the evolution of video game technologies has seen the advent of games that can place physical demands on players, for example, by requiring players to swing a paddle or to dance. Studies of these physical video games suggest that they can generate the same level of physical activity as light jogging or walking (Maddison et al. 2007).

It can be argued that video games are in and of themselves neither good nor bad, but rather it is in their use, abuse, and interpretation in which such distinctions are made. Video games, from one point of view, are just a particular form of game; games in turn, are a specific form of play. Developmental psychologists have long argued that play is important for children (e.g., Bruner et al. 1976; Piaget 1962; Vygotsky 1978), and modern views place games and play as developmentally critical to cognitive development, abstract thinking, self-identity, and social and cultural learning (Bornstein and O'Reilly 1993; Johnson et al. 2005; Pellegrini and Smith 1998). Similarly, the role of games in fostering or facilitating learning has been examined (Boocock and Schild 1968; DeVries and Edwards 1973; James 1966; though see Inbar and Stoll 1970). However, it is clear that the intentions of the designers of games are paramount. For example, educational video games are one set of games that seek to achieve positive goals for their users. Yet even within this domain, grounding the design of video games from perspectives of development and learning theory may be necessary in order to help designers achieve the most positive impact (Verenikina et al. 2003).

History of Educational Video Games and Edutainment

Though computers had been used for educational purposes before (see Suppes 1966), one of the earliest widely used educational video games, *Oregon Trail*, was created by Don Rawitsch, Bill Heinemann, and Paul Dillenberger in 1971 as a

demonstration for a history class that Rawitsch was teaching (Lussenhop 2011). This video game was an all-text adventure presented as an exercise in resource management (Perron and Wolf 2008). Later, while working for Minnesota Education Computing Consortium (Haugo 1973), Rawitsch expanded upon the educational aspects of the game, creating an early precursor of modern day video game “edutainment.”

The term “edutainment,” a combination of “education” and “entertainment,” represents programs that perform educational functions while providing the entertainment value of regular games (Okan 2003; Resnick 2004). Many of the most popular educational video games that followed *Oregon Trail*, such as *Reader Rabbit* (The Learning Company 1984) and *Math Blaster* (Eckert and Davidson 1987), were edutainment titles, structured in order to make drill-and-practice exercises more palatable to young children. Yet, this strategy has left some video game theorists and computer education experts wanting. For example, Mitchel Resnick criticizes modern edutainment software companies as being unnecessarily rigid in their views regarding learning and education, preferring instead a playful approach toward learning that focuses on intrinsic rather than extrinsic rewards (Resnick 2004). Similarly, Simon Egenfeldt-Nielsen noted that edutainment computer games are easily identified by reward structures that are completely disconnected from the educational aspects of the game (Egenfeldt-Nielsen 2007) and was likewise critical of their superficial perspectives on learning.

Superficial or not, educational video games gained great popularity in the 1980s, in part due to the increasing availability of computers in the classroom (Becker 1991) and in part due to the attractive notion of combining the “tedium of education” with the fun of games. Most studies examining whether video games could indeed foster learning showed positive results (for reviews, see Egenfeldt-Nielsen 2006; Kirriemuir and McFarlane 2004; Mitchell and Savill-Smith 2004; Squire 2003). For example, Din and Calao (2001) showed that children playing educational video games designed for kindergarten students improved their ability to spell and decode words when compared to control children; similarly,

Thomas et al. (1997) showed that an adventure video game designed to teach safe-sex skills to high-risk adolescents was effective in improving knowledge as well as perceptions of self-efficacy. Critics of the educational video game literature, however, argue that proof of learning is only the first bar to be hurdled in proving video games a useful tool in the classroom experience (Egenfeldt-Nielsen 2006, 2007), and that a more appropriate comparison is to show that video games provide additional gains in comparison to more standard forms of teaching.

Like video games in general, educational video games continue to evolve. Egenfeldt-Nielsen (2007) describes a model where three generations of video games in education correspond to the broader evolution of psychological and educational perspectives and beliefs. Egenfeldt-Nielsen notes that the first generation can be defined by a behaviorist viewpoint that sees drill-and-practice in the context of extrinsic reward (i.e., most edutainment titles) as a sufficient route by which to inculcate learning. The second generation is defined by a cognitive approach, a view that sees intrinsic motivation and internal mental states as central to learning (e.g., games which encourage a discovery process such as *Super Tangrams*; see Sedighian and Klawe 1996). Between the second and third generations, there is a constructionist perspective, which is similar to the cognitive approach, but emphasizes the role of tangible objects and experience, as exemplified by “micro-world” simulation video games, such as *SimCity* (Games and Squire 2011). The third generation, according to Egenfeldt-Nielsen, is the sociocultural approach, one which sees educational video games themselves as only one piece of the educational puzzle, with a broader context that includes teachers, other students, and the transformation of knowledge through exploration, examination, play, and discourse.

Current Knowledge

Recreational Use of Video Games in Individuals with ASD

Video games have many qualities that make them attractive to some individuals with ASD. An early

assessment of computers in use with autism listed three reasons why computers are attractive to children with autism: computers are nonsocial, they are consistent and predictable, and the child can control and determine the pace of an experience (Swettenham 1996). Other aspects of video interfaces that appeal to individuals with ASD differ from the attractions for typically developed individuals. For example, Mineo et al. (2009) studied the use of multiple forms of electronic-screen media among students with ASD and discussed the attraction of electronic-screen media for individuals with ASD in the context of qualities, such as the de-emphasis on language skills, the constrained viewing area requiring limited attention, its visual modality, and the ability to repeat experiences over and over. Also, Durkin (2010) has noted that the sensory experience of children with autism may be different than typically developing individuals (see also Simmons et al. 2009), and that they appear to have superior performance on some visual tasks but not others.

The absorbing, repetitive, and rewarding nature of computers and computer games has the potential to harm or help individuals with ASD. In the home, nontherapeutic games might encourage social disengagement by displacing social interaction, as is seen in excessive video game players who are otherwise typically developing. Anecdotal reports on ASD video game use and social disengagement are not uncommon, and have been supported by survey data. A study in Mazurek et al. 2011 by Mazurek, Shattuck, Wagner, and Cooper found that 41% of youths with ASD spend most of their free time playing video games, a higher percentage than for other developmentally disabled youths, and higher in comparison to the 18% of youths in the general population who are considered to be high users of video games. Compared to adolescents with typical development, adolescents with Asperger syndrome are less likely to use cell phones for social communication and more likely to use them for non-communicative activities, like games (Durkin et al. 2010). Interestingly, a survey of 23 youths with Asperger syndrome found that several of them listed video games as an apparent special interest. But interviews revealed that the youths often used video games and other more socially

acceptable interests to mask separate special interests that were perceived to be less socially acceptable (Winter-Messiers 2007). These individuals often used video games as a social bridge to help them interact with typical individuals.

Therapeutic Use of Video Games in Individuals with ASD

Computer games have shown the potential to cause brain function changes in typically developed individuals. In particular, action video games have been shown to induce a broad class of changes such as contrast sensitivity, spatial resolution, visual attention, and visuomotor coordination (Spence and Feng 2010). However, others have pointed out methodological shortcomings in studies showing benefits of action video games (Boot et al. 2011). Computers have also been used to target brain training in subgroups of typical individuals. In infants, computers have been used to train visual attention (Wass et al. 2011). In the elderly, cognitive training has been used to attempt to maintain or improve cognitive abilities (Schmiedek et al. 2010), although another study showed a failure of such training to generalize to closely related untrained tasks (Owen et al. 2010).

Because of the increased attraction for computers, video games, and other electronic screen media shown by many individuals with ASD, some educators have suggested that video games (educational or otherwise) might be an effective tool in the therapy and education of individuals with ASD (see ► [“Computer-Based Intervention Assistive Technology”](#) specifically, but also ► [“Video Modeling/Video Self-Modeling”](#) and ► [“Video Instruction”](#)). Whereas computer-assisted instruction (CAI) has been used to understand and treat deficits associated with autism since the late 1960s and early 1970s (Colby 1968, 1973), the use of a video game approach (Chaffin et al. 1982) for therapeutic goals has been a relatively recent research focus. The distinction between computer-assisted instruction (CAI) and computer games is not easy to make. A natural approach is to assert that games involve “play,” and instruction does not. However, CAI often crosses the line into video games. For example,

the study of TeachTown, a CAI program, used animated games as rewards for correct responses (Whalen et al. 2010). Its reinforcers were designed by professional video game designers (Whalen et al. 2006), and the program has been described as a suite of “game-like tests” (Wilkinson et al. 2008).

Video games and CAI have been used in several studies to teach foundational social skills to teach children with ASD. The typical goals of such training are to teach kids to look at faces (Trepagnier et al. 2006), to identify emotions or faces (Beaumont and Sofronoff 2008; Tanaka et al. 2010), to express emotions (Cockburn et al. 2008), and to play collaboratively with other children (Battocchi et al. 2010; Gal et al. 2009). Though skill and knowledge acquisition are the main use of video games for individuals with ASD, games can also be used as rewards in conventional therapy, and as adjuncts to conventional therapy.

One class of computer games involve extending non-computer games into the computer realm. Gal et al. (2009) and Battocchi et al. (2010) are examples of computerized interventions that use tablet input to build on the ideas of collaborative play therapy, where two children must collaborate to accomplish goals on the tablet device. One potential advantage of such computerized therapies is that the computer can easily be tailored to special interests of individuals with ASD to increase interest and engagement. Such tailoring has been used for interventions using non-computer-based games (Baker 2000). Morris et al. (2010) describe successful use of such tailoring for a computer-based exposure therapy, in which an individual with ASD was at first uninterested in the computer therapy, but became deeply engaged by a specifically tailored version of the software that used images of his special interest: newborn babies. They created software that quickly produced background-free images from the internet that were tailored to any specific special interest. They then used this software to customize computer games to the specific special interests of nine individuals with an ASD diagnosis and showed that seven of them preferred to play the customized versions.

Several groups have used video games or video game concepts in larger studies of individuals with ASD (Hopkins et al. 2011; Tanaka et al. 2010; Whalen et al. 2010). Hopkins et al. (2011) developed a computer-based social skills training program called *FaceSay* and employed it in a 6-week randomized control study of 49 high-functioning and low-functioning children with ASD. The results of this study showed that *FaceSay* was useful in teaching emotion recognition skills to low-functioning children with ASD and improving their social interaction. High-functioning children in the study gained in these skills as well as in face recognition ability. Tanaka and others (2010), used a suite of video game-like programs designed to teach face processing skills to children with ASD (the *Let's Face It!* battery), and showed that after just 20 h of intervention using the programs, children in a treatment group ($N = 47$) showed improvements in critical face processing skills compared to controls ($N = 37$). Whalen et al. (2010) used a combined CAI/behavioral intervention approach called *Teach Town: Basics* in a 3-month intervention study using 40 min of activity per school day for 22 children with ASD, comparing to a control group that received regular school instruction ($N = 25$). Whalen found that the intervention group receiving *Teach Town: Basics* showed gains in receptive language and expressive vocabulary compared to the control group. These three studies provide evidence that video games may be an effective tool in providing services to individuals with ASD.

Concerns, Limitations, and Recommendations

There is good evidence that video games may provide a teaching format that helps individuals with ASD learn. However, as highlighted by Egenfeldt-Nielsen (2007), to show that learning is taking place through video games is only the first step in proving the efficacy of video game formats as a therapeutic aid; the careful choice of control conditions (e.g., comparably intense and well-designed non-video-game therapies) will be critical in identifying the advantages and disadvantages of video games in individuals with ASD.

In particular, it is important to note that research shows video game use, and more concerningly, excessive video game use, may be more prevalent in individuals with ASD than in individuals with other developmental issues. For very attractive and fun video games, there is a concern that over time, despite initial intentions, their use by individuals with ASD may become repetitive and more purely stimulatory, ceasing to provide social, communicative, and other educational gains. Furthermore, there is the possibility that skills mastered through CAI and educational video games may not translate or generalize to real-world situations. Though the promise of this technology is great, the dangers are also real, and care must be taken to monitor the utility and pitfalls of this technology. For this reason, it is recommended that video games used for treatment purposes in individuals with autism be part of a combined intervention, under the supervision of experienced clinicians.

Future Directions

Technology is advancing rapidly, and with these advances, the promise of video games and automatic instruction for individuals with ASD seems to grow larger daily. Today, tablet devices, like the iPad, use finger touch as the basis of a look-and-touch interface that is intuitive for young children and accessible to people with developmental disorders (Henderson and Yeow 2012). Console controllers like the Wii and the Kinect use input based on sensing body motion; the use of such body motion in games is highly intuitive and immersive and may provide a physical aspect to video games, with potential benefits to skill generalization. Simultaneously, internet accessibility is becoming ubiquitous, increasing the possibility that individuals with ASD may be able to develop social networks and foster social development through nontraditional means.

From the research point of view, future directions include more large and well-controlled studies of the efficacy of video games, including studies of generalizability of skill acquisition; more concerted efforts to understand the nature

of what makes video games appealing to certain individuals with ASD, and how to manage the potential for abuse; and a continuing focus on the educational role that video games may play for individuals with ASD, with particular attention toward how video games should be tailored to the characteristics of an individual on the spectrum.

See Also

- Computer-Based Intervention Assistive Technology
- Video Instruction
- Video Modeling/Video Self-modeling

References and Reading

- Allison, S. E. (2006). The development of the self in the era of the internet and role-playing fantasy games. *The American Journal of Psychiatry*, 163(3), 381–385. <https://doi.org/10.1176/appi.ajp.163.3.381>.
- Anderson, C. A., & Bushman, B. J. (2001). Effects of violent video games on aggressive behavior, aggressive cognition, aggressive affect, physiological arousal, and prosocial behavior: A meta-analytic review of the scientific literature. *Psychological Science*, 12(5), 353–359. Retrieved February 10, 2012.
- Apperley, T. H. (2006). Genre and game studies: Toward a critical approach to video game genres. *Simulation & Gaming*, 37(1), 6–23. <https://doi.org/10.1177/1046878105282278>.
- Baker, M. J. (2000). Incorporating the thematic ritualistic behaviors of children with autism into games. *Journal of Positive Behavior Interventions*, 2(2), 66–84. <https://doi.org/10.1177/109830070000200201>.
- Battocchi, A., Ben-Sasson, A., Esposito, G., Gal, E., Pianesi, F., Tomasini, D., et al. (2010). Collaborative puzzle game: A tabletop interface for fostering collaborative skills in children with autism spectrum disorders. *Journal of Assistive Technologies*, 4(1), 4–13. <https://doi.org/10.5042/jat.2010.0040>.
- Beaumont, R., & Sofronoff, K. (2008). A multi-component social skills intervention for children with Asperger syndrome: The Junior detective training program. *Journal of Child Psychology and Psychiatry*, 49(7), 743–753. <https://doi.org/10.1111/j.1469-7610.2008.01920.x>.
- Becker, H. J. (1991). How computers are used in United States schools: Basic data from the 1989 IEA computers in education survey. *Journal of Educational Computing Research*, 7(4), 385–406.
- Boocock, S. S., & Schild, E. O. (1968). *Simulation games in learning*. Beverly Hills: Sage.
- Boot, W. R., Blakely, D. P., & Simons, D. J. (2011). Do action video games improve perception and cognition? *Frontiers in Psychology*, 2. <https://doi.org/10.3389/fpsyg.2011.00226>.
- Bornstein, M. H., & O'Reilly, A. W. (1993). *The role of play in the development of thought*. San Francisco: Jossey-Bass.
- Bowman, R. F. (1982). A Pac-Man theory of motivation. Tactical implications for classroom instruction. *Educational Technology*, 22(9), 14–17.
- Breuer, J. S., & Bente, G. (2010). Why so serious? On the relation of serious games and learning. *Eludamos. Journal for Computer Game Culture*, 4(1), 7–24.
- Bruner, J. S., Jolly, A., & Sylva, K. (1976). *Its role in development and evolution*. New York: Penguin Books.
- Burnham, V. (2001). *Supercade: A visual history of the videogame age 1971–1984*. Cambridge, MA: MIT Press.
- Chaffin, J. D., Maxwell, B., & Thompson, B. (1982). ARC-ED curriculum: The application of video game formats to educational software. *Exceptional Children*, 49(2), 173–178.
- Chen, J. (2007). Flow in games (and everything else). *Communications of the ACM*, 50(4), 31–34. <https://doi.org/10.1145/1232743.1232769>.
- Christopher, J. F. (2011). The influence of television and video game use on attention and school problems: A multivariate analysis with other risk factors controlled. *Journal of Psychiatric Research*, 5(6), 808–813. <https://doi.org/10.1016/j.jpsychires.2010.11.010>.
- Cockburn, J., Bartlett, M., Tanaka, J., Movellan, J., Pierce, M., & Schultz, R. (2008). *SmileMaze: A tutoring system in real-time facial expression perception and production in children with autism spectrum disorder*. International Conference on Automatic Face and Gesture Recognition, Workshop on Facial and Bodily expression for Control and Adaptation of Games.
- Colby, K. M. (1968). Computer-aided language development in nonspeaking children. *Archives of General Psychiatry*, 19(6), 641–651. <https://doi.org/10.1001/archpsyc.1968.01740120001001>.
- Colby, K. M. (1973). The rationale for computer-based treatment of language difficulties in nonspeaking autistic children. *Journal of Autism and Childhood Schizophrenia*, 3(3), 254–260. <https://doi.org/10.1007/BF01538283>.
- Cowley, B., Charles, D., Black, M., & Hickey, R. (2008). Toward an understanding of flow in video games. *Computers in Entertainment*, 6(2), 20:1–20:27. <https://doi.org/10.1145/1371216.1371223>.
- Craig, A. A. (2004). An update on the effects of playing violent video games. *Journal of Adolescence*, 27(1), 113–122. <https://doi.org/10.1016/j.adolescence.2003.10.009>.
- Csikszentmihalyi, M. (1991). *Flow: The psychology of optimal experience*. New York: Harper Perennial.
- DeVries, D. L., & Edwards, K. J. (1973). Learning games and student teams: Their effects on classroom process. *American Educational Research Journal*, 10(4), 307.

- Dietz, T. L. (1998). An examination of violence and gender role portrayals in video games: Implications for gender socialization and aggressive behavior. *Sex Roles*, 38(5), 425–442.
- Dill, K. E., & Thill, K. P. (2007). Video game characters and the socialization of gender roles: Young people's perceptions mirror sexist media depictions. *Sex Roles*, 57(11), 851–864.
- Din, F. S., & Calao, J. (2001). The effects of playing educational video games on kindergarten achievement. *Child Study Journal*, 31(2), 95.
- Donovan, T. (2010). *Replay: The history of video games*. East Sussex: Yellow Ant.
- Durkin, K. (2010). Videogames and young people with developmental disorders. *Review of General Psychology*, 14(2), 122–140. <https://doi.org/10.1037/a0019438>.
- Durkin, K., & Barber, B. (2002). Not so doomed: Computer game play and positive adolescent development. *Journal of Applied Developmental Psychology*, 23(4), 373–392.
- Durkin, K., Whitehouse, A., Jaquet, E., Ziatas, K., & Walker, A. J. (2010). Cell phone use by adolescents with Asperger syndrome. *Research in Autism Spectrum Disorders*, 4(2), 314–318. <https://doi.org/10.1016/j.rasd.2009.09.017>.
- Eckert, R., & Davidson, J. (1987). *Math blaster plus (computer software)*. Torrance: Davidson & Associates.
- Egenfeldt-Nielsen, S. (2006). Overview of research on the educational use of video games. *Digital kompetanse*, 1(3), 184–213.
- Egenfeldt-Nielsen, S. (2007). Third generation educational use of computer games. *Journal of Educational Multimedia and Hypermedia*, 16(3), 263.
- Entertainment Software Association. (2011). Essential facts about the computer and video game industry: 2011 sales, demographic, and usage data. Retrieved February 6, 2012, from <http://www.theesa.com/facts/gameplayer.asp>
- Exidy. (1976). *Death Race [Arcade Game]*.
- Ferguson, C. J. (2007). The good, the bad and the ugly: A meta-analytic review of positive and negative effects of violent video games. *Psychiatric Quarterly*, 78(4), 309–316. <https://doi.org/10.1007/s1126-007-9056-9>.
- Ferguson, C. J. (2010). Blazing angels or resident evil? Can violent video games be a force for good? *Review of General Psychology*, 14(2), 68.
- Gal, E., Bauminger, N., Goren-Bar, D., Pianesi, F., Stock, O., Zancanaro, M., et al. (2009). Enhancing social communication of children with high-functioning autism through a co-located interface. *AI & Society*, 24(1), 75–84. <https://doi.org/10.1007/s00146-009-0199-0>.
- Games, A., & Squire, K. D. (2011). Searching for the fun in learning: A historical perspective on the evolution of educational video games. In S. Tobias & J. D. Fletcher (Eds.), *Computer games and instruction* (pp. 17–46). US: Information Age Publishing.
- Gartner, (2011, July 5). Gartner says spending on gaming to exceed \$74 billion in 2011. *Gartner Newsroom*. Retrieved February 6, 2012, from <http://www.gartner.com/it/page.jsp?id=1737414>
- Gentile, D. A., & Gentile, J. R. (2007). Violent video games as exemplary teachers: A conceptual analysis. *Journal of Youth and Adolescence*, 37(2), 127–141. <https://doi.org/10.1007/s10964-007-9206-2>.
- Gentile, D. A., & Stone, W. (2005). Violent video game effects on children and adolescents. A review of the literature. *Minerva Pediatrica*, 57(6), 337.
- Goldsmith, T. T., Jr., & Ray, M. E. (1948). Cathode-ray tube amusement device. *US Patent*, 2(455), 992.
- Harris, M. B., & Williams, R. (1985). Video games and school performance. *Education*.
- Haugo, J. E. (1973, April). Minnesota Educational Computing Consortium. Retrieved February 9, 2012, from <http://www.eric.ed.gov/ERICWebPortal/contentdelivery/servlet/ERICServlet?accno=ED087434>
- Henderson, S., & Yeow, J. (2012). iPad in education: A case study of iPad adoption and use in a primary school. *Hawaii International Conference on System Sciences* (Vol. 0, pp. 78–87). Los Alamitos: IEEE Computer Society. <http://doi.ieeecomputersociety.org/10.1109/HICSS.2012.390>
- Hopkins, I. M., Gower, M. W., Perez, T. A., Smith, D. S., Amthor, F. R., Casey Wimsatt, F., et al. (2011). Avatar assistant: Improving social skills in students with an ASD through a computer-based intervention. *Journal of Autism and Developmental Disorders*, 41(11), 1543–1555. <https://doi.org/10.1007/s10803-011-1179-z>.
- Inbar, M., & Stoll, C. S. (1970). Games and learning. *Interchange*, 1(2), 53–61.
- James, H. H. (1966). An exploratory study of active games in learning of number concepts by first grade boys and girls. *Perceptual and Motor Skills*, 23(2), 341–342.
- Johnson, J. E., Christie, J. F., & Wardle, F. (2005). *Play, development, and early education*. Boston: Allyn & Bacon.
- Kent, S. L. (2001). *The ultimate history of video games: From Pong to Pokemon – The story behind the craze that touched our lives and changed the world* (1st ed.). New York: Three Rivers Press.
- Kirriemuir, J., & McFarlane, A. (2004). *Literature review in games and learning: A report for NESTA Futurelab*. NESTA Futurelab series report 8, 5.
- Ko, C. H., Liu, G. C., Hsiao, S., Yen, J. Y., Yang, M. J., Lin, W. C., et al. (2009). Brain activities associated with gaming urge of online gaming addiction. *Journal of Psychiatric Research*, 43(7), 739–747.
- Kuss, D. J., & Griffiths, M. D. (2011). Internet gaming addiction: A systematic review of empirical research. *International Journal of Mental Health and Addiction*. <https://doi.org/10.1007/s11469-011-9318-5>.
- Lee, K. M., & Peng, W. (2006). What do we know about social and psychological effects of computer games? A comprehensive review of the current literature. In *Playing video games: Motives, responses, and consequences* (pp. 327–345). Mahwah: Lawrence Erlbaum Associates.

- Lussenhop, J. (2011, January 19). *Minneapolis Oregon trail: How three Minnesotans forged its path – City pages*. Retrieved February 9, 2012, from <http://www.citypages.com/content/printVersion/1740595/>
- Maddison, R., Mhurchu, C. N., Jull, A., Jiang, Y., Prapavessis, H., & Rodgers, A. (2007). Energy expended playing video console games: An opportunity to increase children's physical activity? *Pediatric Exercise Science*, 19(3), 334.
- Mazurek, M. O., Shattuck, P. T., Wagner, M., & Cooper, B. P. (2011). Prevalence and correlates of screen-based media use among youths with autism spectrum disorders. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-011-1413-8>.
- Mineo, B. A., Ziegler, W., Gill, S., & Salkin, D. (2009). Engagement with electronic screen media among students with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 39(1), 172–187.
- Mitchell, A., & Savill-Smith, C. (2004). *The use of computer and video games for learning: A review of the literature*.
- Mitra, A. (2010). *Digital games: Computers at play*. New York: Infobase Publishing.
- Morris, R. R., Kirschbaum, C. R., & Picard, R. W. (2010). Broadening accessibility through special interests: a new approach for software customization. *Proceedings of the 12th International ACM SIGACCESS Conference on Computers and Accessibility*, ASSETS'10 (pp. 171–178). New York: ACM. <https://doi.org/10.1145/1878803.1878834>.
- Okan, Z. (2003). Edutainment: Is learning at risk? *British Journal of Educational Technology*, 34(3), 255–264. <https://doi.org/10.1111/1467-8535.00325>.
- Owen, A. M., Hampshire, A., Grah, J. A., Stenton, R., Dajani, S., Burns, A. S., et al. (2010). Putting brain training to the test. *Nature*, 465(7299), 775–778. <https://doi.org/10.1038/nature09042>.
- Pellegrini, A. D., & Smith, P. K. (1998). The development of play during childhood: Forms and possible functions. *Child and Adolescent Mental Health*, 3(2), 51–57.
- Perron, B., & Wolf, M. J. P. (2008). *Video game theory reader two*. Taylor & Francis.
- Piaget, J. (1962). *Play, dreams and imitation* (Vol. 24). New York: Norton.
- Rego, P., Moreira, P. M., & Reis, L. P. (2010). Serious games for rehabilitation: A survey and a classification towards a taxonomy. *2010 5th Iberian Conference on Information Systems and Technologies (CISTI)* (pp. 1–6). Presented at the 2010 5th Iberian Conference on Information Systems and Technologies (CISTI), IEEE.
- Resnick, M. (2004). Edutainment? No thanks. I prefer playful learning. *Associazione Civita Report on Edutainment*, 14.
- Salen, K., & Zimmerman, E. (2004). *Rules of play. Game design fundamentals*. Cambridge: The MIT Press.
- Schmiedek, F., Lövdén, M., & Lindenberger, U. (2010). Hundred days of cognitive training enhance broad cognitive abilities in adulthood: Findings from the COGITO study. *Frontiers in Aging Neuroscience*, 2. <https://doi.org/10.3389/fnagi.2010.00027>.
- Sedighian, K., & Klawe, M. (1996). Super Tangrams: a child-centered approach to designing a computer supported mathematics learning environment. *Proceedings of the 1996 International Conference on Learning Sciences*, ICLS'96 (pp. 490–495). International Society of the Learning Sciences. Retrieved February 10, 2012, from <http://dl.acm.org/citation.cfm?id=1161135.1161208>
- Shapira, N. A., Goldsmith, T. D., Keck, P. E., Jr., Khosla, U. M., & McElroy, S. L. (2000). Psychiatric features of individuals with problematic internet use. *Journal of Affective Disorders*, 57(1–3), 267–272. [https://doi.org/10.1016/S0165-0327\(99\)00107-X](https://doi.org/10.1016/S0165-0327(99)00107-X).
- Simmons, D. R., Robertson, A. E., McKay, L. S., Toal, E., McAleer, P., & Pollick, F. E. (2009). Vision in autism spectrum disorders. *Vision Research*, 49(22), 2705–2739. <https://doi.org/10.1016/j.visres.2009.08.005>.
- Spence, I., & Feng, J. (2010). Video games and spatial cognition. *Review of General Psychology*, 14(2), 92–104. <https://doi.org/10.1037/a0019491>.
- Squire, K. (2003). Video games in education. *International Journal of Intelligent Simulations and Gaming*, 2, 49–62.
- Suppes, P. (1966). *The uses of computers in education*. San Francisco: Freeman Press.
- Susi, T., Johannesson, M., & Backlund, P. (2007). Serious games-An overview. Skövde: University of Skövde (Technical Report HS-IKI-TR-07-001).
- Swettenham, J. (1996). Can children with autism be taught to understand false belief using computers? *Journal of Child Psychology and Psychiatry*, 37(2), 157–165. <https://doi.org/10.1111/j.1469-7610.1996.tb01387.x>.
- Tanaka, J. W., Wolf, J. M., Klaiman, C., Koenig, K., Cockburn, J., Herlihy, L., et al. (2010). Using computerized games to teach face recognition skills to children with autism spectrum disorder: The let's face it! program. *Journal of Child Psychology and Psychiatry*, 51(8), 944–952. <https://doi.org/10.1111/j.1469-7610.2010.02258.x>.
- Terlecki, M., Brown, J., Harner-Steci, L., Irvin-Hannum, J., Marchetto-Ryan, N., Ruhl, L., et al. (2010). Sex differences and similarities in video game experience, preferences, and self-efficacy: Implications for the gaming industry. *Current Psychology*, 30(1), 22–33. <https://doi.org/10.1007/s12144-010-9095-5>.
- The Learning Company. (1984). *Reader rabbit*.
- Thomas, R., Cahill, J., & Santilli, L. (1997). Using an interactive computer game to increase skill and self-efficacy regarding safer sex negotiation: field test results. *Health Education & Behavior*, 24(1), 71–86. <https://doi.org/10.1177/109019819702400108>.
- Trepagnier, C. Y., Sebrechts, M. M., Finkelmeyer, A., Stewart, W., Woodford, J., & Coleman, M. (2006). Simulating social interaction to address deficits of autistic spectrum disorder in children. *Cyberpsychology & Behavior*, 9(2), 213–217. <https://doi.org/10.1089/cpb.2006.9.213>.

- Vandewater, E. A., Shim, M., & Caplovitz, A. G. (2004). Linking obesity and activity level with children's television and video game use. *Journal of Adolescence*, 27(1), 71–85.
- Verenikina, I., Harris, P., & Lysaght, P. (2003). Child's play: Computer games, theories of play and children's development. ACM International Conference Proceeding Series; Vol. 98: *Proceedings of the International Federation for Information Processing Working Group*. Presented at the International Federation for Information Processing Working Group 3.5 Open Conference, Vol. 3. Sydney: Association for Computing Machinery, Inc, New York, pp. 99–106.
- Vygotsky, L. (1978). The role of play in development. *Mind in society the development of higher psychological processes* (M. Cole, Trans.). Cambridge, MA: Harvard University Press, pp. 92–104.
- Wass, S., Porayska-Pomsta, K., & Johnson, M. H. (2011). Training attentional control in infancy. *Current Biology*, 21(18), 1543–1547. <https://doi.org/10.1016/j.cub.2011.08.004>.
- Whalen, C., Liden, L., Ingersoll, B., Dallaire, E., & Liden, S. (2006). Behavioral improvements associated with computer-assisted instruction for children with developmental disabilities. *The Journal of Speech and Language Pathology-Applied Behavior Analysis*, 1(1), 11–26.
- Whalen, C., Moss, D., Ilan, A. B., Vaupel, M., Fielding, P., Macdonald, K., et al. (2010). Efficacy of teachtown: Basics computer-assisted intervention for the intensive comprehensive autism program in Los Angeles unified school district. *Autism*, 14(3), 179–197. <https://doi.org/10.1177/1362361310363282>.
- Wilkinson, N., Ang, R. P., & Goh, D. H. (2008). Online video game therapy for mental health concerns: A review. *The International Journal of Social Psychiatry*, 54(4), 370–382. <https://doi.org/10.1177/0020764008091659>.
- Williams, D., Martins, N., Consalvo, M., & Ivory, J. D. (2009). The virtual census: Representations of gender, race and age in video games. *New Media & Society*, 11(5), 815–834. <https://doi.org/10.1177/1461444809105354>.
- Winter-Messiers, M. A. (2007). From tarantulas to toilet brushes. *Remedial and Special Education*, 28(3), 140.
- Wolf, M. J. (2002). Genre and the video game. *The medium of the video game*, pp. 113–134.
- Young, K. S. (1998). Internet addiction: The emergence of a new clinical disorder. *Cyberpsychology & Behavior*, 1(3), 237–244. <https://doi.org/10.1089/cpb.1998.1.237>.
- Young, K. S., & Rogers, R. C. (1998). The relationship between depression and Internet addiction. *Cyberpsychology & Behavior*, 1(1), 25–28.
- Yuan, B., Folmer, E., & Harris, F. C. (2010). Game accessibility: A survey. *Universal Access in the Information Society*, 10(1), 81–100. <https://doi.org/10.1007/s10209-010-0189-5>.

Video Instruction

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Definition

Video instruction is a teaching technique where learners perform specific target skills (e.g., social, communication, academic, daily chores, vocational, recreational) in natural activity contexts immediately after watching a customized video in which a model performs the expected target behaviors. The video model could be same-age peers, siblings, adults, or the learners themselves. Video instruction comprises several techniques including video modeling, video self-modeling, point-of-view video modeling, and video prompting.

Video modeling is described as a process where (1) a video clip is created that shows a model performing the target skill in a specific activity context in its entirety; (2) a learner is asked to watch the video clip prior to entering the same activity context; (3) an instructor provides prompts and reinforcers to the learner for attending to relevant stimuli; and (4) the learner imitates the observed behavior when provided with the opportunity to perform the same skill.

Video self-modeling is where learners themselves serve as a model by performing the desired target behavior. In this process (1) *only* the exemplary target behavior of the learner is recorded and displayed on the video minus instructor prompts and reinforcers; (2) the learner observes his/her own adaptive behavior in the video; (3) the learner is provided with opportunities to imitate his/her own target behavior in the activity context after watching the video; and (4) after repeated practice, the learner performs the desired target behavior.

Watching oneself performing the desired target behavior increases the learner's self-perception of competence (Mason et al. 2016).

Point-of-view video modeling has been defined as the process of videotaping elements of the physical environment or activity context from the *visual perspective or vantage point* of the learner who needs to acquire and/or master the desired target responses (Hine and Wolery 2006; Mason et al. 2013; Spencer et al. 2015). In this process:

1. The videographer keeps the camcorder at the eye level of the learner while sequentially recording the hands of a model completing the steps of an activity. If the video clip is to teach a transition routine, then only the physical settings are recorded.
2. The learner reviews the video clip and observes exactly how to navigate the task or routine from start to finish.
3. The learner performs the behaviors observed in the video clip.

Repeated review of such video clips is more likely to promote visual comprehension, increase familiarity with materials and settings, and provide a "picture" of the completed process, ostensibly reducing task anxiety and inappropriate behavior.

Whereas video modeling demonstrates an entire set of target behaviors in a sequential manner, **video prompting** breaks down a chain of tasks into individual clips, making it easier for learners to perform each step accurately before advancing to the next step in the activity chain (Sigafoos et al. 2007; Wu et al. 2016).

During **video feedback**, the target student and the instructor view the video clip recorded during a natural activity context. Based on the observed behaviors of the student, the instructor provides specific performance feedback including reinforcers for accurate responding and corrective feedback for incorrect responding. It is believed that the specific focus on the target behaviors increases the probability of correct responding in the future (Thiemann and Goldstein 2001).

Historical Background

The concept of modeling for observational learning dates as far back as 48 years to the social learning theory exemplified by Albert Bandura (Bandura 1969, 1976). This theory emphasized the importance of observing and modeling behaviors, attitudes, and emotional reactions of others. This theory explained human behavior in terms of continuous reciprocal interaction between cognitive, behavioral, and environmental influences (Bandura 1969, 1976). Bandura's early work focused primarily on neurotypical individuals and those with psychological disorders utilizing the behavior modification framework.

Inspired by Bandura's work on observational learning, Creer and Miklich (1970), as cited in Dowrick (1999), asked a young boy who had asthma and behavior problems to role-play adaptive behavior. Authors reported that the role-play per se did not make a difference but watching himself on the video improved the boy's behavior. Creer and Miklich then coined the phrase "self-modeling" to describe this process (Dowrick 1999). During the 1970s, also inspired by observational learning, Hosford and his colleagues had started to use "self-as-a-model" in behavioral counseling sessions with adults on the assumption that a similarity between the model and observer contributed to positive effects (as cited in Dowrick 1999). Based on these reports and his own work on self-efficacy, Bandura advocated for the use of film as a teaching tool in 1976 to enable children to perform desired behaviors after watching themselves acting appropriately.

Video instruction has evolved since the 1970s as an intervention strategy of choice. Its effectiveness has been documented in the literature with regard to training with a variety of populations (e.g., children and adults with and without a disability) on a variety of skills including athletics, academic tasks, social interaction, and functional life skills.

Previous and current research reviews have indicated the effectiveness of video instruction for learners with an autism spectrum disorder on a variety of target skills including social-communication (Green et al. 2017; Lee et al.

2017; Macpherson et al. 2015; Shukla-Mehta et al. 2010), daily living (Mechling et al. 2015), academic (Yakubova et al. 2016), vocational (Seaman and Cannella-Malone 2016), and recreational skills (Sherrow et al. 2016).

While several previous studies reported the use of video modeling as the only intervention component (MacDonald et al. 2005; Nikopoulos and Keenan 2007), recent research and reviews have focused on evaluating the parameters of video instruction packages through comparison studies (Bennett et al. 2017; Mason et al. 2016).

Rationale or Underlying Theory

Autism spectrum disorder is classified as a neurodevelopmental disorder that significantly affects social interaction, verbal and nonverbal communication, interests, activities, and behavior across the lifespan. Awareness and acknowledgment of these skill deficits reiterates the need for using evidence-based practices for learners with ASD across the lifespan. Recent research reviews have presented several compelling reasons for continuing the use of video instruction for learners with ASD to increase a variety of skills (Bennett et al. 2017; Mason et al. 2016; Shukla-Mehta et al. 2010).

Positive outcomes have been documented for learners with ASD when video instruction compared to other methods was used to teach social interaction (Malmberg et al. 2015; McDowell et al. 2015) and gaming skills (Spriggs et al. 2016). With repeated practice and feedback, such skills are more likely to generalize to natural environments (Jones et al. 2014; Smith et al. 2016). Additionally, because video clips can be easily accessed, opportunities to repeatedly watch video clips may result in mastery of skills in a relatively shorter period of time.

Video instruction utilizes systematic instruction; thus, learners are more likely to discriminate between accurate and inaccurate responses. Also, receiving visual and verbal feedback on one's own behavior is often more effective than receiving skills instruction alone. Performance feedback provides concrete information to learners with

ASD and promotes comprehension regarding the accuracy of their response. Video instruction is more likely to deliver focused and systematic visual and verbal feedback when compared to live instruction (Spriggs et al. 2016).

The fact that video clips can be accessed across multiple platforms of mobile devices makes this teaching method not only effective and efficient (Bennett et al. 2017; Mason et al. 2016) but also age and culture appropriate (Odom et al. 2015; Rajendran 2013). Additionally, it facilitates the ultimate goal of independent functioning in adolescents and adults (Ayres et al. 2013; Seaman and Cannella-Malone 2016).

Goals and Objectives

The purpose of video instruction is to allow learners to observe the appropriate actions for performing the target behavior immediately prior to performing the response or skills in the natural environment, increasing the probability of successful performance. Specifically, video instruction is designed to increase a variety of target skills including social-communication, daily living, academic, vocational, and recreational skills. The additional purpose of video instruction is to decrease the problem behavior of individuals (Green et al. 2017).

Treatment Participants

Video instruction has been found to be effective across all age groups of neurotypical and neurodiverse individuals including those with ASD, ADHD, emotional and behavioral disorder (EBD), developmental disability, and language and communication disorder; however, a vast majority of the studies have involved learners with ASD. Learners are more likely to benefit from video instruction if they have skills in (1) attending to relevant stimuli (e.g., cues and prompts in the video); (2) imitating physical and social behavior of the model; (3) visual processing and comprehension of the video content; (4) matching-to-sample and spatial ability to

generalize the target behaviors in real activity settings after having observed the video; and (5) attending to a video clip of at least 1 min in length.

Treatment Procedures

The key procedures of implementing video instruction include both preparing the learning environment and delivering the instructional procedures. Based on previous and current reviews of research on video instruction, the use of systematic instructional procedures based on the principles of behavior is very effective for skills instruction for learners with ASD (Bennett et al. 2017; Mason et al. 2016; Odom et al. 2015; Shukla-Mehta et al. 2010). The step-by-step procedures are as follows:

1. Identify or pinpoint the target responses that need to be increased or decreased and operationally define or describe these behaviors. Develop a unit of measurement to measure the behavior.
2. Identify the setting or activity context in which the target behavior needs to be changed.
3. Identify the model (e.g., adult, peer, sibling, or learner himself/herself) who will display the appropriate behavior and participate in the video production. In existing research, the type of model (i.e., peer, self, or adult) did not appear to affect learning of the target responses in any significant manner.
4. Develop a script or list of actions that the model and other participants (e.g., teacher, peer, or parent) will use for the video demonstration. Role-play or practice the “scene” that needs to be recorded whether it is transition from one environment to another or a complex social interaction sequence. If the model is the learner with ASD, then it may be important to keep a recording device available at all times to capture the target behavior when it is displayed.
5. Plan for the type of video instruction method to use (i.e., video modeling, video self-modeling, point-of-view modeling, etc.) in order to adjust the distance and area of focus for creating the video clip.
6. Prepare the instructional context where the video clip needs to be recorded prior to creating the instructional video. For discrete behaviors of short durations, a 3–5-minute video clip is generally considered to be adequate. Plan on when, where, and how frequently should the learner watch the video. For children who were reinforced by watching videos, repeated viewing (3–4 times) has been known to be effective.
7. Let the learner watch the video clip immediately prior to the natural activity context in which the target skill needs to be displayed. During the viewing, the teacher might use prompts to direct the learner’s attention to the relevant environmental cues and to the model’s appropriate behavior. If the concept is complex (e.g., perspective taking), the teacher may pause the video to ask questions to ensure the learner’s comprehension. The correct response may be reinforced, whereas an incorrect response may lead to reviewing the video clip until a correct response is emitted. If video feedback is an integral component of video instruction, then the instructor and learner can watch the recorded performance to provide constructive feedback.
8. Provide the learner an opportunity to display the observed behavior in the natural activity context. Accurate display of the target behavior may result in natural reinforcers (e.g., a compliment or smile from a peer, task completion).
9. Teach the learner to use the technological device when using video self-modeling or video prompting for self-instruction purposes.
10. Whether video instruction occurs in the context of research or clinical activity, it is recommended that instructors take baseline, intervention, maintenance, and generalization data to demonstrate the effectiveness of this teaching method.

Efficacy Information

Based on recent research reviews exclusively on video instruction for individuals with ASD, sufficient evidence exists to support it as an evidence-based practice for teaching a variety of skills (Bennett et al. 2017; Mason et al. 2016; Shukla-Mehta et al. 2010). While research reviews have also been conducted by Odom et al. (2015) and Seaman and Cannella-Malone (2016), they involve the use of various types of technological interventions for adolescents and adults including but not exclusively video modeling.

Bennett et al. (2017) reviewed 24 studies involving a total of 89 participants (3–22 years) who were diagnosed with ASD and intellectual disability (ID) or other comorbid diagnoses. Their primary purpose was to compare different intervention components to determine which ones were the most effective in producing desired outcomes (e.g., type of model; narration versus no narration; screen sizes; video perspective; custom versus commercial videos; simultaneous VM versus video priming or delayed VM; combined comparisons; and miscellaneous studies where a comparison was between two or more teaching techniques). Results showed no differential outcomes across any of the components that largely favored one over the others. The only exception was where function-based vs. nonfunctional videos were more effective for participants. Because this review included a wide variety of target skills, the outcomes do not associate specific components with specific target skills.

In a meta-analysis of the effectiveness of video self-modeling (VSM), Mason et al. (2016) reviewed 14 studies that met the quality standards established by What Works Clearinghouse (Kratochwill et al. 2010). The review included 50 participants ranging from pre-kindergarten to post-secondary settings across a variety of disability categories including ASD (62%), ID (14%), EBD (18%), and learning disability (LD; 8%). Overall results on the effectiveness of VSM (used alone or with other components) indicated a moderate magnitude of change as a function of intervention. Thus, authors conducted additional analyses to identify potential moderators (p. 632).

In general, VSM was found to be more effective for learners with ASD and EBD, specifically involving positive self-review as opposed to editing video to only include target behavior. Additionally, VSM alone was more effective as opposed to combining it with other intervention components to both change specific target behaviors.

The review of research by Shukla-Mehta et al. (2010) included 40 studies with a total of 126 participants (2.5 to 20 years) where video instruction was used to increase social-communication skills of learners with ASD. Results showed that 23 of 30 (77%) VM studies reported clear acquisition of target responses for all participants with or without other instructional components. Of the seven studies that utilized VSM, only three reported effectiveness in teaching social and communication responses. Similarly, only three studies have utilized point-of-view modeling and all reported to improve target behaviors. Since then many studies have utilized various types of video instruction methods for teaching a variety of target skills. In a study that compared VSM with and without peer training, Bellini et al. (2016) showed that VSM alone increased unprompted social engagement for all three participants. The addition of peer training did not lead to changes in social engagement when compared to the VSM-only phase.

Overall, while various methods of video instruction have been documented to be effective, future research needs to focus on isolating specific VM methods by disability category and level of functioning while controlling for methodological rigor.

Outcome Measurement

The outcomes of video instruction have been measured mostly based on directly observable discrete target responses of participants. Additionally, outcomes were measured in terms of skill acquisition, maintenance, or generalization in the natural activity contexts. In addition to objective measures for assessing behavior change, outcomes assessment can also include social

validation. Social validity is central to the belief that video instruction will be used more frequently in applied settings by practitioners and parents if it is shown to be successful in increasing social and functional skills of learners with ASD.

Qualifications of Treatment Providers

Interventionists or treatment providers who are well versed in the use of systematic instruction procedures including modeling, prompting, corrective feedback, and contingent reinforcement are more likely to be successful in implementing video instruction with individuals with ASD. However, recent research (Allen et al. 2015) showed that parents can effectively use video instruction to increase functional life skills of an adolescent with ASD and ID.

See Also

- ▶ Video Feedback and Feedforward
- ▶ Video Modeling/Video Self-Modeling
- ▶ Video Prompting

References and Reading

- Allen, K. D., Vatland, C., Bowen, S. L., & Burke, R. V. (2015). An evaluation of parent-produced video self-modeling to improve independence in an adolescent with intellectual developmental disorder and an autism spectrum disorder: A controlled case study. *Behavior Modification, 39*(4), 542–556. <https://doi.org/10.1177/0145445515583247>.
- Ayres, K. M., Mechling, L., & Sansosti, F. J. (2013). The use of mobile technologies to assist with life skills/independence of students with moderate/severe intellectual disability and/or autism spectrum disorders: Considerations for the future of school psychology. *Psychology in the Schools, 50*(3), 259–271. <https://doi.org/10.1002/pits.21673>.
- Bandura, A. (1969). *Principles of behavior modification*. New York: Holt, Reinhart, & Winston.
- Bandura, A. (1976). Effecting change through participant modeling. In J. D. Krumboltz & C. E. Thorensen (Eds.), *Counseling methods*. New York: Holt, Rinehart and Winston.
- Bellini, S., Gardner, L., Hudock, R., & Kashima-Ellingson, Y. (2016). The use of video self-modeling and peer training to increase social engagement in preschool children on the autism spectrum. *School Psychology Forum, 10*(2), 207–219.
- Bennett, K. D., Aljehany, M. S., & Altaf, E. M. (2017). Systematic review of video-based instruction component and parametric analyses. *Journal of Special Education Technology, 32*(2), 80–90. <https://doi.org/10.1177/0162643417690255>.
- Dowrick, P. (1999). A review of self-modeling and related interventions. *Applied and Preventive Psychology, 8*, 23–39.
- Green, V. A., Prior, T., Smart, E., Boelema, T., Drysdale, H., Harcourt, S., et al. (2017). The use of individualized video modeling to enhance positive peer interactions in three preschool children. *Education & Treatment of Children, 40*(3), 353–378.
- Hine, J. F., & Wolery, M. (2006). Using point-of-view video modeling to teach play to preschoolers with autism. *Topics in Early Childhood Special Education, 26*, 83–93.
- Jones, J., Lerman, D. C., & Lechago, S. (2014). Assessing stimulus control and promoting generalization via video modeling when teaching social responses to children with autism. *Journal of Applied Behavior Analysis, 47*, 37–50.
- Kratochwill, T. R., Hitchcock, J., Homer, R. H., Levin, J. R., Odom, S. L., Rindskopf, D. M. & Shadish, W. R. (2010). Single-case designs technical documentation. Retrieved from What Works Clearinghouse website: http://ies.ed.gov/ncee/wwc/pdf/wwc_scd.pdf
- Lee, S. Y., Lo, Y., & Lo, Y. (2017). Teaching functional play skills to a young child with autism spectrum disorder through video self-modeling. *Journal of Autism and Developmental Disorders, 47*(8), 2295–2306. <https://doi.org/10.1007/s10803-017-3147-8>.
- MacDonald, R., Clark, M., Garrigan, E., & Vangala, M. (2005). Using video modeling to teach pretend play to children with autism. *Behavioral Interventions, 20*, 225–238.
- Macpherson, K., Charlop, M. H., & Miltenberger, C. A. (2015). Using portable video modeling technology to increase the compliment behaviors of children with autism during athletic group play. *Journal of Autism and Developmental Disorders, 45*, 3836–3845. <https://doi.org/10.1007/s10803-014-2072-3>.
- Malmberg, D. B., Charlop, M. H., & Gershfeld, S. J. (2015). A two experiment treatment comparison study: Teaching social skills to children with autism spectrum disorder. *Journal of Developmental and Physical Disabilities, 27*(3), 375–392. <https://doi.org/10.1007/s10882-015-9420-x>.
- Mason, R. A., Davis, H. S., Boles, M. B., & Goodwyn, F. (2013). Efficacy of point-of-view video modeling: A meta-analysis. *Remedial and Special Education, 34*(6), 333–345. <https://doi.org/10.1177/0741932513486298>.
- Mason, R. A., Davis, H. S., Ayres, K. M., Davis, J. L., & Mason, B. A. (2016). Video self-modeling for individuals with disabilities: A best-evidence, single case meta-analysis. *Journal of Developmental and Physical Disabilities, 28*(4), 623–642. <https://doi.org/10.1007/s10882-016-9484-2>.

- McDowell, L. S., Gutierrez, A., & Bennett, K. D. (2015). Analysis of live modeling plus prompting and video modeling for teaching imitation to children with autism. *Behavioral Interventions*, 30(4), 333–351.
- Mechling, L., Bryant, K., Spencer, G., & Ayres, K. (2015). Comparison of methods for demonstrating passage of time when using computer-based video prompting. *Education and Training in Autism and Developmental Disabilities*, 50(1), 56–70.
- Nikopoulos, C. K., & Keenan, M. (2007). Using video modeling to teach complex social sequences to children with autism. *Journal of Autism Developmental Disorders*, 37, 678–693.
- Odom, S. L., Thompson, J. L., Hedges, S., Boyd, B. A., Dykstra, J. R., Duda, M. A., et al. (2015). Technology-aided interventions and instruction for adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 45(12), 3805–3819. <https://doi.org/10.1007/s10803-014-2320-6>.
- Rajendran, G. (2013). Virtual environments and autism: A developmental psychopathological approach. *Journal of Computer Assisted Learning*, 29(4), 334–347. <https://doi.org/10.1111/jcal.12006>.
- Seaman, R. L., & Cannella-Malone, H. I. (2016). Vocational skills interventions for adults with autism spectrum disorder: A review of the literature. *Journal of Developmental and Physical Disabilities*, 28(3), 479–494. <https://doi.org/10.1007/s10882-016-9479-z>.
- Sherrow, L. A., Spriggs, A. D., & Knight, V. F. (2016). Using video models to teach students with disabilities to play the wii. *Focus on Autism and Other Developmental Disabilities*, 31(4), 312–320. <https://doi.org/10.1177/1088357615583469>.
- Shukla-Mehta, S., Miller, T., & Callahan, K. J. (2010). Evaluating the effectiveness of video instruction on social and communication skills training for children with autism spectrum disorders: A review of the literature. *Focus on Autism and Other Developmental Disabilities*, 25(1), 23–36. <https://doi.org/10.1177/1088357609352901>.
- Sigafoos, J., O'Reilly, M., Cannella, H., Edrisinha, C., de la Cruz, B., Upadhyaya, M., et al. (2007). Evaluation of a video prompting and fading procedure for teaching dishwashing skills to adults with developmental disabilities. *Journal of Behavioral Education*, 16, 93–109.
- Smith, K. A., Ayres, K. A., Alexander, J., Ledford, J. R., Shepley, C., & Shepley, S. B. (2016). Initiation and generalization of self-instructional skills in adolescents with autism and intellectual disability. *Journal of Autism and Developmental Disorders*, 46(4), 1196–1209. <https://doi.org/10.1007/s10803-015-2654-8>.
- Spencer, G. P., Mechling, L. C., & Ivey, A. N. (2015). Comparison of three video perspectives when using video prompting by students with moderate intellectual disability. *Education and Training in Autism and Developmental Disabilities*, 50(3), 330.
- Spriggs, A. D., Gast, D. L., & Knight, V. F. (2016). Video modeling and observational learning to teach gaming access to students with ASD. *Journal of Autism and Developmental Disorders*, 46(9), 2845–2858. <https://doi.org/10.1007/s10803-016-2824-3>.
- Thiemann, L. S., & Goldstein, H. (2001). Social stories, written text cues, and video feedback: Effects on social communication of children with autism. *Journal of Applied Behavior Analysis*, 34, 425–446.
- Wu, P., Cannella-Malone, H. I., Wheaton, J. E., & Tullis, C. A. (2016). Using video prompting with different fading procedures to teach daily living skills: A preliminary examination. *Focus on Autism and Other Developmental Disabilities*, 31(2), 129–139. <https://doi.org/10.1177/1088357614533594>.
- Yakubova, G., Hughes, E. M., & Shinaberry, M. (2016). Learning with technology: Video modeling with concrete–representational–abstract sequencing for students with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 46(7), 2349–2362. <https://doi.org/10.1007/s10803-016-2768-7>.

Video Modeling/Video Self-Modeling

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Definition

Video modeling is the use of video to instruct an individual on desired skills or behaviors by viewing someone demonstrating those skills or behaviors. The video model can take several forms including watching an adult, peer, oneself (also known as video self-modeling), or an animation. This instructional approach is designed to teach new or improve existing skills or behaviors. Video modeling is also used to replace or eliminate challenging or nonproductive behaviors. All behavior viewed on a video model is positive modeling of what should be done versus what should not be done. Inappropriate behaviors or observed errors are removed from the videos. Video modeling helps focus an individual's attention on the most relevant behaviors in the video so that with practice and rehearsal the individual learns and demonstrates the targeted behavior modeled (Prelock 2006; Prelock et al. 2011). Video modeling helps an individual translate and generalize what they learned in a video-modeling session to skill

acquisition in many aspects of daily life (Charlop-Christy et al. 2000; Shipley-Benamou et al. 2002).

Historical Background

Bandura's (1969) early work on social learning theory and the power of modeling to support learning as well as Dowrick & Dove, (1980) research on video modeling, including video self-modeling, provides the underlying historical framework for using video models and video self-models to increase positive behaviors. In fact, in the early 1970s, video self-modeling was used as an intervention strategy to support the learning and behavior of individuals with developmental disabilities. With the ongoing work of Marjorie Charlop-Christy, Scott Bellini, and Tom Buggey, interest in video modeling and video self-modeling as viable intervention strategies for children with ASD has grown. Positive outcomes have been reported for increasing play behavior, greetings, answering and asking questions, conversation language, and perspective taking and self-help skills, to name a few. With rapid expansion of and increased access to video-editing software, the use of video modeling and video self-modeling is likely to see a dramatic increase.

Rationale or Underlying Theory

Video modeling is grounded in social learning theory (Bandura 1969) and the influence of models on observational learning. Bandura (1969, 1977) describes two underlying theoretical considerations that support the use of video modeling and, in particular, video self-modeling. He found that the most effective models were those who shared attributes (e.g., gender, age, race, ability – those slightly more advanced than the observer) with the observer. He also described self-efficacy or how confident one feels about carrying out a task as critical to learning success. Video self-modeling is built on both of these theoretical foundations in that individuals observe themselves carrying out tasks with success.

Since it is often difficult for individuals with autism to focus their attention, the use of video that highlights relevant stimuli facilitates an individual's attention on what is most important for learning to occur (Prelock 2006). Video is also less socially threatening, does not require immediate responses, allows for repeated viewings, and focuses on the visual strengths frequently reported for children with ASD (Grandin 2006). Further, video self-modeling might affect memory. Kehle et al. (2002) propose that children viewing themselves performing positive behaviors might supplant their memories of inappropriate behaviors. Additional research is needed to more carefully examine each of these perspectives as an explanation of why video modeling and video self-modeling appear to lead to behavior change.

Goals and Objectives

Several goals have been addressed using video modeling. Much of the research has focused on facilitating social skills including emotion recognition (Golan and Baron-Cohen 2006); time engaged during play (Bellini et al. 2007); conversational speech, spontaneous greetings, labeling emotions, and perspective taking (Charlop and Milstein 1989; Charlop-Christy et al. 2000; Charlop-Christy and Daneshvar 2003; LeBlanc et al. 2003); and other social language skills (Buggey 2005; Maione and Mirenda 2006; Scattone 2008). Some recent efforts have investigated the use of video modeling to support academic tasks (Delano 2007). Communication goals that might be a focus for video modeling or video self-modeling include naming, responding to questions, asking questions, participating in reciprocal conversation, and improving prosody and intonation (Prelock 2006).

Treatment Participants

Video modeling appears to be an appropriate intervention across a range of developmental levels and ages. For children with ASD, the reported research focuses on children 3–15 years

of age, although some researchers describe using video modeling with preschool children through older adults. Video modeling has been applied to children across the autism spectrum with varying cognitive and linguistic abilities (Buggey 2005, 2012; Delano 2007; Sansosti and Powell-Smith 2008; Scattone 2008). Some evidence suggests that individuals with both severe autism and cognitive impairment may not benefit from video modeling, yet several researchers have reported positive outcomes for children with moderate to severe autism (Bellini et al. 2007; Buggey et al. 1999; LeBlanc et al. 2003).

Treatment Procedures

There are several steps to consider in the implementation of video modeling. First, a decision needs to be made about the behavior to be targeted. This should be a team decision, and the targeted behavior should be operationally defined so that it can be observed, measured, and is specific to the individual with ASD (Charlop-Christy 2004; Prelock et al. 2011). A task analysis is completed next so that the individual steps for the video model can be itemized. This should be guided by observations of individuals who are typically developing, and input should be gathered from those who know the individual best (e.g., parents, teachers). It is important to incorporate a motivating theme in what is being modeled based on the particular interest of the child or individual with ASD.

Actors for video modeling can be familiar or unfamiliar adults or peers who are able to follow a script that has been outlined by the team. The actors for video self-modeling would include the individual with ASD performing a task that is slightly beyond his/her current observed ability. Notably, there are two types of video self-modeling, *feedforward* and *positive self-review*. In feedforward, an individual is shown a video of himself/herself performing a new, developmentally appropriate behavior that the team hopes will facilitate forward movement in performing that behavior. Positive self-review includes the individual with autism performing a task he/she

already knows how to do, so performance success can be reinforced, maintained, and generalized.

When preparing the video, actors should speak slowly and clearly, and target behaviors should be clearly observable and exaggerated when appropriate. The actors' facial expression should be shown. There should be minimal distracters in the video so the individual can focus on the most relevant cues. When viewing the video, attention can be prompted by pointing and saying "watch" or "look."

Following the video viewing, it is important to debrief with the individual, reviewing what was seen and heard, identifying the targeted language, noting the prosody and emotional expression of the models, and talking about variations in the scenarios (Charlop and Milstein 1989; Prelock et al. 2011). To support the child's generalized use of the targeted behavior being modeled, all communication and interaction attempts should be encouraged and reinforced. The target behavior should be demonstrated about 75–80% of the time before determining acquisition, and at least two observations of the video should occur before learning acquisition is assessed (Charlop-Christy 2004; Prelock et al. 2011).

Efficacy Information

Dowrick and colleagues first described the effectiveness of video modeling when they used this intervention to facilitate skill development in children with a variety of disabilities (Dowrick and Dove 1980; Dowrick and Hood 1981; Dowrick and Raeburn 1995). Charlop-Christy and her colleagues then applied video modeling to children with ASD (Charlop and Milstein 1989; Charlop-Christy and Daneshvar 2003; Charlop-Christy et al. 2000; LeBlanc et al. 2003). Over the last 25 years, research has investigated a variety of outcomes in the areas of communication, social, and academic skills following implementation of video modeling for children with ASD.

Spontaneous requesting (Wert and Neisworth 2003), recognizing emotions in speech and facial expressions (Corbett 2003), compliment giving, initiations and responses (Apple et al. 2005), and

language production (Buggey 2005; Charlop-Christy et al. 2000), including responding to questions and making verbal requests (Buggey et al. 1999), asking questions (Charlop and Milstein 1989), and increasing conversational speech (Charlop and Milstein 1989; Charlop-Christy et al. 2000; Nikopoulos and Keenan 2003, 2004, 2007; Ogletree and Fischer 1995; Sherer et al. 2001), have all been reported as yielding positive communication outcomes following video modeling.

A variety of social skills have also been facilitated through video modeling, including play behaviors such as reciprocal play (Nikopoulos and Keenan 2003; Sancho et al. 2010), motor and verbal sequences (D'Ateno et al. 2003), independent play (Charlop-Christy et al. 2000), play-related comments (Taylor et al. 1999), and socio-dramatic play (Dauphin et al. 2004; Nikopoulos and Keenan 2003) and more social engagement behaviors like complying, greeting and sharing (Simpson et al. 2004), spontaneous greeting (Charlop-Christy et al. 2000), and social initiations (Buggey 2005, 2013; Nikopoulos and Keenan 2004). Various aspect of theory of mind have been taught using video modeling including first-order false-belief tasks (Charlop-Christy and Daneshvar 2003) and other perspective-taking activities such as recognition of facial expressions, gestures, and intonation in the context of play (LeBlanc et al. 2003). In fact, Gena and colleagues (2005) found video modeling to be as effective as in vivo modeling in increasing affective responding (i.e., sympathy, appreciation, and disapproval) for preschoolers in the context of play with peers. Video-based group instruction was successful in increasing social initiations such as joining an activity and social awareness such as asking about another person's interest and offering assistance (Plavnick et al. 2013).

Academic classroom skills have also been supported through video modeling. Adolescents with ASD learned to solve math problems (Burton et al. 2013) after watching their successful performance. Preschoolers with ASD successfully increased their social communication in the classroom using either in vivo or video modeling (Wilson 2013).

When comparing the effects of live modeling to video modeling on increasing spontaneous greetings, conversational speech, play, comprehension following story reading, emotion recognition, and self-help skills, results favored video modeling which led to faster skill acquisition and better generalization and was more cost effective (Charlop-Christy et al. 2000). In addition, Sherer et al. (2001) found no differences between children watching themselves and children watching other children or adults when examining children's ability to respond to and ask questions.

Video modeling appears to be an evidence-based intervention that supports generalized learning and skill maintenance (Prelock et al. 2011). A review of the literature suggests video modeling is a promising intervention to support social communication and functional skills (Ayres and Langone 2005; Bellini and Akullian 2007). In fact, the National Standards Project (National Autism Center 2009, 2015) describes modeling, including video modeling, as an established intervention that increases communication, cognitive, social, play, and personal responsibility skills and decreases problem behaviors for children ages 3–18 on the autism spectrum. Additional research is needed to examine the impact of video modeling on children with more limited verbal and cognitive abilities.

Outcome Measurement

There are no specific tools or instruments suggested for measuring treatment outcomes, although most clinical researchers using this method suggest that a functional behavior assessment should occur prior to implementation to determine the targeted behaviors (Sturmey 2003), data collection should follow a single-subject design approach (Kazdin 1982), and curriculum-based measurements should be considered in assessing effectiveness across contexts (Salvia and Hughes 1990). Following these recommendations, baseline rates of behaviors should be documented and then tracked through intervention and maintenance. Graphic representations can also be made to display visual evidence of

change. Charting progress on targeted behaviors is also possible through curriculum-based measurements if team members are examining change in the context of the classroom as compared to the performance expected of other students in the classroom. Parents can have a critical role in the measurement of outcomes, particularly if the desired behaviors are primarily observed in the home, although documenting generalization of behaviors across home and school settings is ideal.

Qualifications of Treatment Providers

There is no specific training required to implement video modeling other than being familiar with the equipment and software used to create and edit the video models or video self-models. Models require the basic skills to act out a pre-determined script that is targeting the desired behavior. Parents, teachers, speech-language pathologists, and other related service providers may be required to take on primary responsibility for planning and editing the videos or particular aspects of the video depending on the targeted behaviors and the requirements for language, engagement, physical positioning, behavior, etc.

See Also

- [Modeling](#)
- [Self-Recognition](#)
- [Social Skill Interventions](#)
- [Video Instruction](#)

References and Reading

- Apple, A. L., Billingsley, F., Schwartz, I. S., & Carr, E. G. (2005). Effects of video modeling alone and with self-management on compliment-giving behaviors of children with high-functioning ASD. *Journal of Positive Behavior Interventions*, 7(1), 33–46.
- Ayres, K. M., & Langone, J. (2005). Intervention and instruction with video for students with autism: A review of the literature. *Education and Training in Developmental Disabilities*, 40(2), 183–196.
- Bandura, A. (1969). *Principles of behavior modification*. New York: Holt, Rinehart & Winston.
- Bandura, A. (1977). *Self-efficacy: The exercise of control*. New York: Freeman.
- Bellini, S., & Akullian, J. (2007). A meta-analysis of video modeling and video self-modeling interventions for children and adolescents with autism spectrum disorders. *Exceptional Children*, 73, 261–284.
- Bellini, S., Akullian, J., & Hopf, A. (2007). Increasing social engagement in young children with autism spectrum disorders using video self-modeling. *School Psychology Review*, 36, 80–90.
- Buggey, T. (2005). Applications of video self-modeling with children with autism in a small private school. *Focus on Autism and Other Developmental Disabilities*, 20, 180–204.
- Buggey, T. (2012). Video modeling applications for persons with autism. In P. A. Prelock & R. J. McCauley (Eds.), *Treatment of autism spectrum disorders: Evidence-based intervention strategies for communication and social interaction* (pp. 345–369). Baltimore: Paul H. Brookes.
- Buggey, T. (2013). The use of self-modeling to promote social interactions among young children. *Focus on Autism & Other Developmental Disabilities*, 28(4), 202–211.
- Buggey, T., Toombs, K., Gardner, P., & Cervetti, M. (1999). Self-modeling as a technique to train response behaviors in children with autism. *Journal of Positive Behavior Intervention*, 1, 205–214.
- Burton, C. E., Anderson, D. H., Prater, M. A., & Dyches, T. T. (2013). Video self-modeling on an iPad to teach functional math skills to adolescents with autism and intellectual disability. *Focus on Autism & Other Developmental Disabilities*, 28(2), 67–77.
- Charlop, M. H., & Milstein, J. P. (1989). Teaching autistic children conversational speech using video modeling. *Journal of Applied Behavior Analysis*, 22(3), 275–285.
- Charlop-Christy, M. H. (2004). Using video modeling to teach perspective taking to children with autism. Presentation at the Annual Vermont Summer Autism Institute, Burlington.
- Charlop-Christy, M., & Daneshvar, S. (2003). Using video modeling to teach perspective taking to children with autism. *Journal of Positive Behavior Interventions*, 36(2), 12–21.
- Charlop-Christy, M., Le, L., & Freeman, K. (2000). A comparison of video modeling with in vivo modeling for teaching children with autism. *Journal of Autism and Developmental Disorders*, 30, 537–552.
- Corbett, B. A. (2003). Video modeling: A window into the world of autism. *The Behavior Analyst Today*, 4, 367–375.
- D'Ateno, P., Mangiapanello, K., & Taylor, B. A. (2003). Using video modeling to teach complex play sequences to a preschooler with autism. *Journal of Positive Behavior Interventions*, 5(1), 5–11.
- Dauphin, M., Kinney, E. M., & Stromer, R. (2004). Using video-enhanced activity schedules and matrix training

- to teach sociodramatic play to a child with autism. *Journal of Positive Behavior Interventions*, 6, 238–250.
- Delano, M. E. (2007). Improving written language performance of adolescents with Asperger syndrome. *Journal of Applied Behavior Analysis*, 40(2), 342–351.
- Dowrick, P. W., & Dove, C. (1980). The use of self-modeling to improve the swimming performance of spina bifida children. *Journal of Applied Behavior Analysis*, 13, 51–56.
- Dowrick, P. W., & Hood, M. (1981). Comparison of self-modeling and small cash incentives in a sheltered workshop. *Journal of Applied Psychology*, 66, 394–397.
- Dowrick, P. W., & Raeburn, J. M. (1995). Self-modeling: Rapid skill training for children with physical disabilities. *Journal of Developmental and Physical Disabilities*, 7, 25–37.
- Gena, A., Couloura, S., & Kymissis, E. (2005). Modifying the affective behavior of preschoolers with autism using in-vivo or video modeling and reinforcement contingencies. *Journal of Autism and Developmental Disorders*, 35 (5), 545–556.
- Golan, O., & Baron-Cohen, S. (2006). Systemizing empathy: Teaching adults with Asperger syndrome or high-functioning autism to recognize complex emotions using interactive multimedia. *Development and Psychopathology*, 18(2), 591–617.
- Grandin, T. (2006). *Thinking in pictures: And other reports from my life with autism*. New York: Vintage Books.
- Kazdin, A. E. (1982). *Single-case research designs: Methods for clinical and applied settings*. New York: Oxford University Press.
- Kehle, T. J., Bray, M. A., Margiano, S. G., Theodore, L. A., & Zhou, Z. (2002). Self-modeling as an effective intervention for students with serious emotional disturbance: Are we modifying children's memories? *Psychology in the Schools*, 39(2), 203–207.
- LeBlanc, L. A., Coates, A. M., Daneshvar, S., Charlop-Christy, M. H., Morris, C., & Lancaster, B. M. (2003). Using video modeling and reinforcement to teach perspective-taking skills to children with autism. *Journal of Applied Behavior Analysis*, 36, 253–257.
- Maione, L., & Mirenda, P. (2006). Effects of video modeling and video feedback on peer-directed social language skills of a child with autism. *Journal of Positive Behavior Interventions*, 8(2), 106–118.
- National Autism Center. (2009). *National Standards Project, Phase 1: Addressing the need for evidence-based practice guidelines for ASD*. www.nationalautismcenter.org.
- National Autism Center (2015). *National Standards Project, Phase 2: Addressing the need for evidence-based practice guidelines for ASD*. www.nationalautismcenter.org.
- Nikopoulos, C. K., & Keenan, M. (2003). Promoting social initiation in children with autism. *Behavioral Interventions*, 18(2), 87–108.
- Nikopoulos, C. K., & Keenan, M. (2004). Effects of video modeling on training and generalisation of social initiation and reciprocal play by children with autism. *European Journal of Behaviour Analysis*, 5(1), 1–13.
- Nikopoulos, C. K., & Keenan, M. (2007). Using video modeling to teach complex social sequences to children with autism. *Journal of Autism and Developmental Disorders*, 37(4), 678–693.
- Ogletree, B. T., & Fischer, M. A. (1995). An innovative language treatment for a child with high functioning autism. *Focus on Autistic Behavior*, 10, 1–10.
- Plavnick, J. B., Sam, A. M., Hume, K., & Odom, S. L. (2013). Effects of video-based group instruction for adolescents with autism spectrum disorders. *Exceptional Children*, 80(1), 67–83.
- Prelock, P. A. (2006). *Communication assessment and intervention in Autism Spectrum Disorders*. Austin: Pro-Ed.
- Prelock, P. A., Paul, R., & Allen, E. (2011). Evidence-based treatments in communication for children with autism spectrum disorders. In B. Reichow, P. Doehring, D. V. Cicchetti, & F. R. Volkmar (Eds.), *Evidence-base treatments for children with autism* (pp. 93–169). New York: Springer.
- Salvia, J., & Hughes, C. (1990). *Curriculum-based assessment: Testing what is taught*. New York: Macmillan.
- Sancho, K., Sidener, T. M., Reeve, S. A., & Sidener, D. W. (2010). Two variations of video modeling interventions for teaching play skills to children with autism. *Education & Treatment of Children*, 33(3), 421–442.
- Sansosti, F. J., & Powell-Smith, K. A. (2008). Using computer-presented social stories and video models to increase the social communication skills of children with high-functioning autism spectrum disorders. *Journal of Positive Behavior Interventions*, 10(3), 162–178.
- Scattone, D. (2008). Enhancing the conversation skills of a boy with Asperger's disorder through social stories and video modeling. *Journal of Autism and Developmental Disorders*, 38(2), 395–400.
- Sherer, M., Pierce, K. L., Paredes, S., Kisacky, K. L., Ingersoll, B., & Schreibman, L. (2001). Enhancing conversation skills in children with autism via video technology: Which is better, "self" or "other" as a model? *Behavior Modification*, 25(1), 140–158.
- Shipley-Benamou, R., Lutzker, J. R., & Taubman, M. (2002). Teaching daily living skills to children with autism through instructional video modeling. *Journal of Positive Behavior Interventions*, 4(3), 166–177.
- Simpson, A., Langone, J., & Ayres, K. M. (2004). Embedded video and computer based instruction to improve social skills for students with autism. *Education & Training in Developmental Disabilities*, 39(3), 240–252.
- Sturmey, P. (2003). Video technology and persons with autism and other developmental disabilities: An emerging technology for PBS. *Journal of Positive Behavior Interventions*, 5, 3–4.
- Taylor, B. A., Levin, L., & Jasper, S. (1999). Increasing play-related statements in children with autism toward their siblings: Effects of video modeling. *Journal of*

- Developmental and Physical Disabilities*, 11(3), 253–264.
- Wert, B. Y., & Neisworth, J. T. (2003). Effects of video self-modeling on spontaneous requesting in children with autism. *Journal of Positive Behavior Interventions*, 5, 300–305.
- Wilson, K. P. (2013). Teaching social-communication skills to preschoolers with autism: Efficacy of video versus in vivo modeling in the classroom. *Journal of Autism and Developmental Disorders*, 43, 1819–1831.

Video Prompting

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Definition

Video prompting (VP) is one of the several video-based instruction strategies, and it involves a student observing an actor performing individual task steps on a video before imitating those task steps. The process is comprised of the following sequence: The student observes a single step, and then the student performs that step. The student repeats this process until all task steps are completed. Additional prompts and performance feedback are required for some individuals, and these should be faded as soon as possible to promote the independent demonstration of the skills or the independent use of the VP system.

Historical Background

Video-based instruction (VBI) is based, in part, on Albert Bandura's Social Learning Theory (Bellini and Akullian 2007). In that seminal work, Bandura (1977) posited that in addition to learning behaviors through direct experience, humans could also acquire behaviors by observing others emit behavior. Bandura reasoned that the ability to imitate others makes for efficient learning that

results in the large behavior repertoires that humans develop. Others have supported the idea that imitation is a critical feature of learning that is essential for developing a variety of important skills (Cooper et al. 2020). VBI is also based on other behavioral procedures such as task analysis, behavior chaining, and performance feedback (Banda et al. 2011).

VBI has been implemented for decades with individuals with developmental disabilities (DD), including people who experience autism spectrum disorder (ASD; Banda et al. 2011; Bellini and Akullian 2007). VBI incorporates two main strategies that include video modeling (VM) and VP; there are additional variations based on these overall VBI techniques. The main difference between VM and VP is related to the video presentation. When using VM, an interventionist plays the video of an actor performing an entire task before instructing the student to imitate what they observed on the recording. When using VP, the interventionist plays a video of one step of a task being completed by an actor prior to instructing the student to model what they viewed, and this continues until all steps have been presented using the video recordings, as well as all steps having been completed or attempted by the student (Aljehany and Bennett 2019).

VP is a VBI strategy that is used with behavior chains (i.e., behaviors comprised of multiple steps, such as making a sandwich). Earlier research suggested that people performed better when completing a behavior chain while viewing smaller portions of video compared to viewing longer video sections (Margolius & Sheffield, as cited in LeGrice 1989). LeGrice (1989), applying the aforementioned research finding, was among the first researchers to explore the effects of VP on the skill acquisition of chained behaviors among individuals with DD. In that study, LeGrice (1989) successfully used VP as part of a time delay strategy to teach individuals with intellectual disability (ID) to use a video recorder and a personal computer. Participants were given an opportunity to perform the target behaviors. If the participants did not respond, or if they responded in error, a video prompt was delivered up to three times. The researcher showed the participants a video clip of

an actor completing the corresponding task step and then asked the participants to imitate what they observed. In this sense, VP was used as a consequence strategy.

Soon after the LeGrice (1989) study, Tiong et al. (1992) examined the effects of VP when teaching individuals with ID fire safety skills (e.g., exiting a building). Similar to the LeGrice (1989) study, Tiong et al. (1992) used VP as part of a time delay strategy whereby participants were given an opportunity to respond. However, contingent upon no response or an error, the researchers showed participants a video prompt of the correct behavior, and this video was shown up to three times. Similar to the previously discussed study, VP was used as a consequence strategy. Results indicated that participants acquired the correct responses, there was evidence of generalization, and the responses maintained following the removal of the intervention.

In a later study, Norman et al. (2001) investigated VM combined with VP to teach children with DD three self-help skills including cleaning sunglasses, putting on a watch, and using a zipper to close a jacket. Norman et al. (2001) started sessions by showing a complete video model of the entire task. This video model was followed by a zero-second time delay whereby the researchers then showed participants a video prompt of the first task step to be completed. The time delay for showing participants the video prompt eventually increased to 5 sec. Results indicated that VM combined with VP was successful in teaching the students the targeted skills. In addition to these positive results, there is an important distinction between the study by Norman et al. and those by LeGrice (1989) and Tiong et al. (1992): Norman and colleagues used the VM and the VP sequence, at least initially, as an antecedent strategy rather than a consequence strategy. Although VP eventually became a consequence strategy in the Norman et al. study, this variation is notable in that the researchers implemented VP as a system to match errorless learning parameters.

Graves et al. (2005) also used VM combined with VP to teach three secondary students with ID to complete cooking tasks. Similar to Norman et al. (2001), Graves et al. (2005) initially used

VM and VP as antecedent strategies to reduce the likelihood of student errors while learning the targeted tasks. VP eventually became a consequence strategy as the study progressed, and the results indicated that the participants acquired the cooking skills with anecdotal reports of generalization and maintenance.

An additional, albeit subtle, variation of this research line was conducted by Sigafoos et al. (2005). In this study, the researchers examined VP (without the use of VM) on the acquisition and maintenance of adults with DD making a snack. Intervention sessions consisted of researchers playing a video prompt before requesting participants to complete a given step. Sigafoos et al. continued to use VP as an antecedent intervention throughout the study, and this represents an approach to VP that potentially reduces errors committed by participants. Results suggested that two of the three participants acquired and maintained the skill; however, VP was not helpful for the remaining participant.

In a follow-up study, Sigafoos et al. (2007) examined the effects of VP as an antecedent strategy to improve a daily living skill (DLS) – in this case, dishwashing – among adults with DD. The VP procedure was implemented identically to the Sigafoos et al. (2005) study. Results suggested that participants acquired the skills; however, unlike previous research, they did not maintain the skill when VP was removed. Attempts to fade VP by progressively combining steps until the procedure resembled VM were successful with two of the three participants. The remaining participant's responding was variable.

The results related to maintenance led to Sigafoos et al. (2007) questioning whether VP might lead to a level of prompt dependency for some participants. That is, some participants would continue to require video prompts to complete tasks. For some researchers, practitioners, caregivers, and students, this dependency might be of concern since the use of devices to prompt behavior might not be suitable in all settings (Sigafoos et al. 2007).

The studies described represent a sampling of the development of the VP strategy; the presentation of these studies does not imply a specific

timeline nor does it represent an exhaustive sample of the work during the timeframe of the reviewed studies. The critical point is that the tactic emerged as a means of helping individuals learn tasks comprised of multiple steps given that early research demonstrated that shorter video clips, focused on fewer behaviors, were effective when teaching people skills (LeGrice 1989). The application of VP with individuals with DD proved successful, and researchers applied variations (e.g., as an antecedent strategy and as a consequence strategy) with success. The development of VP continued during the ensuing years, and there is ample research available today to suggest that the practice is evidence-based under certain circumstances.

Current Knowledge

Over the last several decades, researchers have explored the effects of VP on the acquisition, maintenance, and generalization of skills among individuals with DD, including those people experiencing ASD with or without ID. Many of these studies have been organized into systematic reviews of the literature and meta-analyses, and our understanding of the effects of VP has improved as a result of this work.

The effectiveness of VP on the acquisition of chained behaviors has been well-documented. One of the first research teams to synthesize the research on VP was conducted by Banda et al. (2011). That research demonstrated that VP was effective for teaching a variety of chained behaviors to individuals with ASD with or without ID. In their review of the literature, Gardner and Wolfe (2013) reported that VP was effective when teaching chained behaviors to individuals with ASD. A subsequent review by Domire and Wolfe (2014) reported similar findings. In a recent meta-analysis, Aljehany and Bennett (2019) reported that VP was an effective instructional strategy for teaching DLS; their results showed the effect size of VP was in the high-moderate range. Among these review studies, the chained behaviors frequently explored were DLSs (e.g., cooking, setting tables, laundry skills, tying shoes,

and washing dishes), community skills (e.g., using debit cards), vocational skills (e.g., cleaning, making photocopies), and safety skills (e.g., exiting a building).

Maintenance and generalization of skills were noted among some of the studies in the aforementioned systematic reviews. Indeed, Banda et al. (2011) and Domire and Wolf (2014) reported that some researchers demonstrated positive results related to maintenance and generalization of skills. However, issues with maintenance were also noted in some studies. Overall, these features of learning – that is, maintenance and generalization – seem to be understudied, and additional work is needed.

In addition to the overall effects of VP, researchers have explored parameters of VP, including the perspective of the videos, the actors serving as models in the videos, differing screen sizes, and the use of other procedures as part of a VP intervention package, to name a few (Bennett et al. 2017). The perspective from which the video is recorded, and subsequently played for those receiving VP, can be from (a) a first-person, or point-of-view, perspective; (b) a third-person, or spectator, perspective; or (c) a combination of both of these. A first-person perspective shows learners a video of an actor performing a task; however, only the actor's arms and hands are in view. A third-person perspective shows students a video of the actor completing tasks from a distance such that most or all of the actor's body is in view while completing the task. Researchers have used all three perspectives across VBI studies with success. When it comes to VP, in particular, researchers have reported that the strategy has been effective using either perspective, as well as the combined perspective (Domire and Wolfe 2014). Moreover, Bennett et al. (2017) reported that there were no differences in two comparison studies whereby researchers examined differences related to the video perspectives. Notwithstanding these findings, a first-person perspective has the advantage of reducing additional stimuli that might be less relevant to task completion, and this feature could benefit individuals with ASD who are known to have issues with over-selectivity and attending to tasks (Gardner and Wolfe 2013).

Another parameter of VBI that received attention in the literature is related to the actors performing tasks in the videos. Bandura (1977) suggested that imitation of others' behaviors could be affected by the degree to which the actor, or model, is similar to the individual learning skills. In the overall VBI literature, there are numerous examples of various models used as actors in the videos, including self as the model, children as the models, and adolescent and adults as the models. In the Bennett et al. (2017) review on parameters of VBI, comparison of models was examined by researchers with inconclusive results. In some cases, self as the model (when compared to peer or adult models) was more effective for a limited number of participants. In other instances, self as the model, adult as the model, and peer as the model were equally effective (Bennett et al. 2017). In demonstration studies where the model type was not compared, there have been successful reports of VM and VP with children modeling adult actors, adolescents modeling adult actors, and adults modeling adult actors (Gardner and Wolfe 2013). Thus, specific guidance is difficult to offer at the present moment, and additional research seems to be warranted when examining the effects of model type when implementing VM or VP (Bennett et al. 2017).

In recent years, and as a result of improvements in mobile technology, researchers have investigated the effects of different screen sizes when using VBI. Among the advances in technology are the screen sizes of video playback devices. Devices used in past VBI research included televisions, desktop computer monitors, laptop computers, tablet computers, and smartphones, to name a few. To date, there are inconclusive results related to this parameter of VBI (Bennett et al. 2017). Some researchers found little differences in student performance when comparing large to small screen sizes using VM (Miltenberger and Charlop 2015), while other researchers found larger screen sizes to be more effective for participants when using VM (Mechling and Ayres 2012). Recently, Bennett et al. (2016) found that results between two different screen sizes were negligible for two participants, with the remaining

participant performing better with a larger screen size when using VP. Considering the corpus of work examining screen sizes with VBI, Bennett et al. (2016) posited that differences in student performance could be unique to the characteristics of the students or the tasks they are learning to complete.

Other intervention components have been included with VP (Aljehany and Bennett 2019; Banda et al. 2011). Some of these additional strategies include the use of voice-over instructions that accompany each video step, the use of response prompts and fading systems (e.g., least-to-most prompting, graduated guidance), and error correction (Aljehany and Bennett 2019). Voice-over instruction provides students with an auditory cue as part of the video, and the inclusion of this extra cue can help some individuals perform the demonstrated behavior (Bennett et al. 2017). However, as pointed out by Bennett et al. (2017), a few individuals seemed to perform better without the use of voice-over narration. Thus, caregivers and practitioners should record the video with narration and then test if the narration is useful to the student. Adjusting this parameter, if initially recorded, is as simple as lowering the volume (Bennett et al. 2017).

In addition to voice-over narration, researchers have examined the effects of additional response prompting and fading systems to help learners follow video prompts. In a recent meta-analysis of VP, Aljehany and Bennett (2019) reported that the use of response prompting and fading systems, as well as error correction, was needed for some participants with ASD and ID. If these extra strategies are needed, it is critical that implementers fade these extra prompts so that the videos evoke student behavior absent of additional assistance by others. Ideally, the students learn to model the video demonstrations on their own so that they can prompt their behavior, especially as they approach new tasks (Smith et al. 2015).

The promise of any VBI system, including VP, is that individuals with ASD and similar developmental disabilities learn a system that can prompt their behaviors independent of caregivers or professionals. The improvements in mobile technology (e.g., tablets, smartphones, and smartwatches)

offer the potential for greater independence as students learn to become VBI users (Smith et al. 2016). To date, however, there have been a limited number of studies whereby researchers examined the effects of VBI as a self-instruction system. In the existing research on self-instruction, implementers completed some tasks for the students while setting up the self-instruction sessions, and this limits the results of the extant data (Smith et al. 2015, 2016). Smith et al. (2016) completed one of the few studies whereby researchers demonstrated that individuals with ASD and ID could learn to self-instruct using VM on a smartphone. The results of this study are promising, but additional work is needed in this area.

Future Directions

Although the use of VP has been well-studied over recent decades, there are still areas that need to be addressed. First, many studies on the use of VP have focused on teaching DLSs. Additional work is needed to learn if VP is useful in developing other skills among individuals with ASD, including academic skills and social skills. Second, Aljehany and Bennett (2019) reported that VP was most frequently studied with adolescent and adult participants. There were too few studies and participants where researchers examined VP with elementary-aged students. Moreover, there were even fewer studies and participants that included preschool-aged children. Given these findings, additional research is needed to inform caregivers and practitioners the extent to which VP is effective with younger populations. Third, as illustrated by Banda et al. (2011), Domire and Wolfe (2014), and Smith et al. (2016), additional studies are needed to assess the generalization potential of VBI strategies, including VP. This issue is more pronounced considering that VBI strategies, paired with mobile technology, have the potential to serve as a self-instruction methodology or a self-remediation technique (Smith et al. 2016). Fourth, additional work is needed to explore methods to fade VP so that individuals perform tasks in complete independence. As Sigafos et al. (2007) argued, there could be instances in

which it is not feasible or appropriate for individuals with disabilities to use VP. For instance, some tasks, or task steps, require careful attention (e.g., construction), and the use of mobile technology as a self-instruction or self-prompting device could compromise the safety of the person performing the task or others nearby. Finally, as technology continues to advance, becomes more portable, and begins to simulate reality (e.g., portable devices used in virtual reality simulations), the integration of VP tactics with this technology becomes possible. However, research into how such technology affects individuals with ASD, the practicality of using such technology in home and community settings, and the feasibility of integrating the instructional strategies with advance technology is needed (Pham et al. 2019).

Although additional work is required to refine some of the parameters surrounding the use of VP, as well as research on integrating VP with more advanced technological developments, the practice is well-suited for helping people with ASD. Years of research have demonstrated that the strategy is useful for developing skills among this population of learners. Caregivers and teachers should consider using this strategy when circumstances permit.

See Also

► Video Modeling/Video Self-Modeling

References and Reading

- Aljehany, M. S., & Bennett, K. D. (2019). Meta-analysis of video prompting to teach daily living skills to individuals with autism spectrum disorder. *Journal of Special Education Technology*, 34(1), 17–26. <https://doi.org/10.1177/0162643418780495>.
- Banda, D. R., Dogoe, M. S., & Matuszny, R. M. (2011). Review of video prompting studies with persons with developmental disabilities. *Education and Training in Autism and Developmental Disabilities*, 46, 514–527.
- Bandura, A. (1977). *Social learning theory*. Englewood Cliffs: Prentice-Hall.
- Bellini, S., & Akullian, J. (2007). A meta-analysis of video modeling and video self-modeling interventions for children and adolescents with autism spectrum

- disorders. *Exceptional Children*, 73, 264–287. <https://doi.org/10.1177/001440290707300301>.
- Bennett, K. D., Aljheny, M., & Altaf, E. (2017). Systematic review of video-based instruction component and parametric analyses. *Journal of Special Education Technology*, 32, 80–90. <https://doi.org/10.1177/0162643417690255>.
- Bennett, K. D., Gutierrez, A., & Loughrey, T. (2016). Comparison of screen sizes when using video prompting to teach adolescents with autism. *Education and Training in Autism and Developmental Disabilities*, 51, 379–390.
- Cooper, J. O., Heron, T. E., & Heward, W. L. (2020). *Applied behavior analysis* (3rd ed.). Hoboken: Pearson Educational.
- Domire, S. C., & Wolfe, P. (2014). Effects of video prompting techniques on teaching daily living skills to children with autism spectrum disorders: A review. *Research and Practice for Persons with Severe Disabilities*, 39, 211–226. <https://doi.org/10.1177/1540796914555578>.
- Gardner, S., & Wolfe, P. (2013). Use of video modeling and video prompting interventions for teaching daily living skills to individuals with autism spectrum disorders: A review. *Research and Practice for Persons with Severe Disabilities*, 38, 73–87. <https://doi.org/10.2511/027494813807714555>.
- Graves, T. B., Collins, B. C., Schuster, J. W., & Kleinert, H. (2005). Using video prompting to teach cooking skills to secondary students with moderate disabilities. *Education and Training in Developmental Disabilities*, 40, 34–46.
- LeGrice, B. (1989). Video prompting and increasing assistance: A comparison of two prompting methods (unpublished master's thesis). University of Canterbury.
- Mechling, L. C., & Ayres, K. M. (2012). A comparative study: Completion of fine motor office related tasks by high school students with autism using video models on large and small screen sizes. *Journal of Autism and Developmental Disorders*, 42, 2364–2373. <https://doi.org/10.1007/s10803-012-1484-1>.
- Miltenberger, C. A., & Charlop, M. H. (2015). The comparative effectiveness of portable video modeling vs. traditional video modeling interventions with children with autism spectrum disorders. *Journal of Developmental and Physical Disabilities*, 27, 341–358. <https://doi.org/10.1007/s10882-014-9416-y>.
- Norman, J. M., Collins, B. C., & Schuster, J. W. (2001). Using an instructional package including video technology to teach self-help skills to elementary students with mental disabilities. *Journal of Special Education Technology*, 16(3), 5–18.
- Pham, A. V., Bennett, K. D., & Zetina, H. (2019). Technology-aided interventions for individuals with autism: Implications for policy and practice. *Policy Insights from the Behavioral and Brain Sciences*, 6(2), 202–209. <https://doi.org/10.1177/2372732219857750>.
- Sigafoos, J., O'Reilly, M., Cannella, H., Edrisinha, C., de la Cruz, B., Upadhyaya, M., et al. (2007). Evaluation of a video prompting and fading procedure for teaching dish washing skills to adults with developmental disabilities. *Journal of Behavioral Education*, 16, 93–109. <https://doi.org/10.1007/S10864-006-9004-z>.
- Sigafoos, J., O'Reilly, M., Cannella, H., Upadhyaya, M., Edrisinha, C., Lancioni, G. E., et al. (2005). Computer-presented video prompting for teaching microwave oven use to three adults with developmental disabilities. *Journal of Behavioral Education*, 14, 189–201. <https://doi.org/10.1007/s10864-005-6297-2>.
- Smith, K. A., Ayres, K. A., Alexander, J., Ledford, J. R., Shepley, C., & Shepley, S. B. (2016). Initiation and generalization of self-instructional skills in adolescents with autism and intellectual disability. *Journal of Autism and Developmental Disorders*, 46, 1196–1209. <https://doi.org/10.1007/s10803-015-2654-8>.
- Smith, K. A., Shepley, S. B., Alexander, J. L., & Ayres, K. M. (2015). The independent use of self-instructions for the acquisition of untrained multi-step tasks for individuals with an intellectual disability: A review of the literature. *Research in Developmental Disabilities*, 40, 19–30. <https://doi.org/10.1016/j.ridd.2015.01.010>.
- Tiong, S., Blampied, N. M., & LeGrice, B. (1992). Training community-living, intellectually handicapped people in fire safety using video prompting. *Behaviour Change*, 9(2), 65–72. <https://doi.org/10.1017/S0813483900006379>.

Video Tape Analysis Studies

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Definition

This entry on videotape analysis studies considers how researchers use video to study autism, especially in terms of diagnosis, skill assessment, behavioral expression, and lived experience. The studies primarily represent research from the United States.

Historical Background

Live observation of human behaviors and interactions is a basic investigative tool used in the humanities and social sciences, and video recording is now used as an addition to or substitute for this research activity. Ethnographic and

ethnomethodological theories and methods are often manifested in video footage that is spontaneous, with wide-angle shots of social activities occurring in their typical settings. It can be analyzed in terms of conversational and discursive features, meaning-making actions and processes, social activities and routines, and speech acts and events, for example, which are often represented in the form of transcripts. Psychological theories and methods are often manifested in video footage that is considered semi-structured or structured, where spontaneous behaviors are captured and/or elicited in prearranged scenarios. Depending on the behavior in question, close or wide framing of shots occurs. It can be analyzed using discrete behavioral coding, or translating an observation of gaze, facial expression, gesture, or word production, for example, into a number, where any type of outward behavior can be defined and measured in an effort to predict and/or understand its relationship to another behavior. Erickson (1986) argues that ethnographic/ethnomethodological approaches are “interpretive” or focused on how meaning is made in human life and psychological approaches are “positivistic” or focused on cause-and-effect relationships between human behaviors. The former, he says, primarily intends to “particularize” human experience or locate it within a historical and social time and place. The latter, he continues, primarily intends to “generalize” human experience or identify patterns of behavior that may hold true despite environmental influences. Depending on researchers’ training and the phenomenon they want to investigate, they may thus approach the collection and analysis of video recording differently. However, given the permanent documentation of human behavior that video provides, a combination of such approaches to recordings may also be applied.

Current Knowledge

Video to Diagnose (Retrospective and Prospective)

One of the most representative types of autism video analysis studies is that of the retrospective use of home videos for the purpose of

investigating the emergence of autism in infants. Faced with questions about the possible early expression of behavioral differences and how, in particular, that might correspond to early diagnosis and intervention, researchers often turn to families for help. The most commonly cited study, by Osterling and Dawson (1994), compared the home videos of 11 children with autism and 11 neurotypical children as they celebrated their first birthdays. Though not the first researchers to use home videos (see the same article for a discussion of the earliest work by Massie in the late 1970s), their decision to focus on such an event allowed them to match children from the two groups in terms of activity, age, and setting, which strengthened their research design in comparison to previous studies. As a result, their findings still receive significant attention. In particular, the absence of social interaction action behaviors, especially orienting to names and others, pointing, and showing objects, predicted a later autism diagnosis. Adding to the scope of previous studies, Baranek (1999) also used home videos to measure sensory-motor functioning in addition to social functioning in 32 infants who were divided into groups with neurotypical development or a later diagnosis of autism or developmental delay. Focusing on scenarios of social play, Baranek determined that between 74% and 85% of the time-specific variables, including mouthing and playing with objects and bodily posturing, significantly predicted which developmental group the infants would later fit into. This reflected similar findings from earlier video analysis by Losche (1990). Baranek thus recommended using sensory-motor functioning measurements as supplements to screening tools for autism. Similarly, Colgan, Lanter, McComish, Watson, Crais, & Baranek (2006) found a correlation between less variety of gestures in infancy and later autism diagnosis. These retrospective studies used behavioral coding to measure and analyze video data. For example, Adrien, Lenoir, Martineau, Perrot, Haneury, Larmande, & Sauvage (1993) used a standardized assessment to code behavioral functioning on a 0–4 scale. Meanwhile, other studies developed original coding procedures based on behavioral categories

identified in previous studies. Whether building off of existing measures or meeting the needs of a particular study, retrospective coding of video produced evidence that early behavioral differences could be identified in a way that might predict a later diagnosis of autism. It also provided the foundation for researchers to begin prospective video studies and to test the validity of these findings.

Unlike the retrospective studies, prospective studies often use video collected from structured assessments and/or as a supplement to other measures (these assessments and measures will be discussed in more detail in the next section). Driven by evidence that autism is usually genetically based, such studies largely examine the behavior of infant siblings of older children who have been diagnosed with autism. In recent work, Ozonoff, Iosif, Baguio, Cook, Hill, Hutman, Rogers, Rozga, Sangha, Sigman, Steinfeld, & Young (2010) coded the frequency of social behaviors and engagement from video of standardized visual subtest for 25 infant siblings who were later diagnosed with autism and 25 gender-matched neurotypical children at 6, 12, 18, 24, and 36 months. After 12 months of age, they found significantly less sharing of gaze, smiles, and vocalizations with examiners in the autism group. Using this same sample of children, Rozga, Hutman, Young, Rogers, Ozonoff, Dapretto, & Sigman (2011) coded nonverbal communication and social interaction abilities of neurotypical infants and infant siblings who either went on to receive a later diagnosis of autism or not. In a lab setting, they videotaped semi-structured play between caregivers and children and a social communication assessment. This permitted coding of gaze, joint attention, smiles, and vocalizations at 6, 12, 24, and 36 months for most of their 167 subjects. Like Ozonoff et al. 2010 they found that autism symptoms were not identifiable before 12 months. In line with these findings, Hutman, Rozga, DeLaurentis, Barnwell, Sugar, & Sigman (2010) found a comparatively lower reaction to distress behaviors between the ages of 12 and 36 months for infant siblings later diagnosed with autism. Furthermore, Wetherby, Woods, Allen, Cleary, Dickinson, & Lord (2004)

also used video from a communication and symbolic skills assessment for children between the ages of 18 and 21 months and found that at least nine behaviors indicated autism versus other atypical and typical development. This included difficulties with coordinating and orienting gaze, responding to one's name, repetitive movements of the body or with objects, and unusual prosody. In sum, prospective videotape studies further specify the findings of retrospective home videos and lend more support to the validity of early behavioral differences being predictive of a later autism diagnosis.

Video to Assess and Treat

As indicated in the previous section, semi-structured and structured assessments that are administered in a lab setting are often used in combination with video as well as other measures in large-scale, multifaceted, and sometimes multi-site studies. In order to facilitate the exchange of information, researchers often use the same or similar protocol and video coding procedures. Some of the most common, replicated videotape studies for autism have measured emotional expression, engagement states, joint attention and other nonverbal communication, functional and symbolic play levels, verbal abilities, and coordinated or synchronized interactional behaviors.

Many different semi-structured and structured assessments are used to measure a variety of domains of functioning in children with autism. The behaviors, when coded across time, can also be used to evaluate the efficacy of a treatment approach. For example, an early intervention targeting joint attention may measure the child's joint attention at pre- and post-intervention to evaluate program efficacy. A few of the most noteworthy assessments include the Early Social Communication Scale (ESCS; Mundy et al. 1986), the Structured Play Assessment (Ungerer and Sigman 1981), maternal synchrony (Siller and Sigman 2008), and joint engagement coding (Adamson et al. 2009). The ESCS is a semi-structured assessment that provides social communication probes to children with autism and is later coded for initiations and responses to joint

attention. The Structured Play Assessment is also a semi-structured assessment that provides children the opportunity to display functional and symbolic play acts; frequency and type of play acts are coded from the videotape. Both the maternal synchrony code and joint engagement coding systems are coded from a brief videotaped mother-child interaction where mothers and children are instructed to “play as they normally would” with their child. The maternal synchrony code focuses on several dimensions of the interaction including maternal indicating behaviors, maternal verbal behaviors, and the child’s toy-directed attention. The joint engagement coding system is a state coding system that compiles the duration of time that the child’s attention is directed to another person, objects, or shifting attention between the two. These measures have been applied to the study of children with autism both using cross-sectional and longitudinal designs (Siller and Sigman 2008), as well as used as measures of treatment outcome in early intervention research (Kasari et al. 2006, 2008, 2010).

Researchers also integrate video-based analysis to compare and potentially improve autism intervention and treatment. For example, Rogers, Hayden, Hepburn, Charlifue-Smith, Hall, & Hayes (2006) determined that Denver Model and PROMPT therapies produced similar language outcomes. Additionally, Kasari et al. (2010) demonstrated the positive impact of parent-mediated intervention on toddlers’ communication skills. While video is also used in various ways to treat autism-related issues (i.e., reflecting on videos of oneself to learn adaptive social skills), those are beyond the scope of this entry.

Video to Contextualize, Describe (Naturalistic: Generalizing, Particularizing)

Naturalistic videos of spontaneous behaviors in real-life social settings provide a way for researchers to explain their findings in relation to the varied expression of lived experience. When collected by researchers who apply ethnographic or ethnomethodological approaches, videos function as a form of documentation of everyday life. They can be transcribed in terms of nonverbal and

verbal features and closely analyzed in terms of the resources that individuals use to make sense of a given social moment. Ochs, Kremer-Sadlik, Sirota, and Solomon (2004), for example, demonstrate that video analysis conducted within an overall ethnographic study can be used to further investigate how specific children with autism cope with aspects of social functioning that are culturally and historically situated. They apply conversation and discourse analysis, provide detailed descriptions of social organization, and identify how the features of conversational sequences, specific situations, and implied cultural norms can either provide support or limitations for individuals with autism. Similarly, Ochs, Solomon, and Sterponi (2005) apply the same style of video microanalysis to a cross-cultural comparison of autism therapy. They find that treatment outcomes can be affected by body positioning, speech tempo, voice pitch, and other features of communication that are often culturally specific and subconsciously enacted. In the same analytic tradition, Kremer-Sadlik (2004) demonstrates in part how caregivers use question and answer sequences and reformulations of speech to enhance the participation of their children with autism in family activities. Similarly, Maynard (2005) analyzes the administration of a standardized assessment of commonsense reasoning. He demonstrates how an apparently straightforward question that is presented by examiners to children with autism is actually underpinned by different types of practical logic that impact children’s responses and the meaning of the exam’s results. As previously mentioned, such studies represent an effort to “particularize” (Erickson 1986) the experience of autism, and they typically focus on very few cases or individuals at a time.

Naturalistic video can also be used to “generalize” (Erickson 1986), though sometimes the social situations may be somewhat structured. In such cases, spontaneous interactions may be transcribed as in the above studies, and then verbal features may be quantified and statistically analyzed. Also, the behaviors of larger groups of individuals or “samples” may be coded frame-by-frame, in specific categories or intervals, or

for specific durations or frequencies and then statistically analyzed. For example, Tager-Flusberg, Calkins, Nolin, Baumberger, Anderson, & Chadwickdias (1990) compared the language acquisition of six children with autism and six with Down syndrome by analyzing 100 samples of their spontaneous speech while playing with their mothers at home over a span of 14 months. In part, they measured lengths of utterances, word order, and word usage and found that the children in the study followed the same developmental trajectory for language as neurotypical children. Similarly, Capps, Losh, and Thurber (2000) compared the language of 13 children with autism, 13 with Down syndrome, and 13 neurotypically developing while telling stories from a book and participating in a semi-structured conversation with an intervention. They applied a preexisting transcription system to analyze speech structures and an original coding system to analyze evaluative practices in storytelling. After a statistical analysis, they determined that the discreet components did not significantly differ between the groups, but how children with autism linked them did. Similarly, from transcripts, Losh and Capps (2006) coded emotional experiences recounted by neurotypical children and those with autism during the administration of a conversation-based emotion elicitation procedure. Using a discourse analysis perspective in combination with a 0–3 scale of situational appropriateness, they identified statistical differences in aspects of how the two groups interpreted and represented their emotional experiences. Swensen, Kelley, Fein, and Naigles (2007) also used video documentation to investigate the relationship between gaze and word-order comprehension in very young children with autism and neurotypical development. Such studies seek to find general patterns of behavior within more natural settings in order to produce findings that may have universal implications.

Future Directions

As technological advances make video equipment and related analytic software more accessible,

affordable, and efficient, videotape studies present promising future directions. In addition to the refinement of measures based on naturalistic, semi-structured, and structured video for purposes of diagnosis, assessment, description, intervention, and treatment of autism, such studies support creative, holistic, and representative approaches. For example, bridging a divide between qualitative and quantitative research paradigms, video facilitates mixed-method designs. Most often, this means that video is being used to capture human interaction in natural environments, which is then coded and/or transcribed. While the reality is that such uses of video are not usually truly combining methodologies but rather standing them side by side, they are nevertheless a reflection of researchers attempting to apply and connect their work to real-world settings. This can be seen in studies that videotape spontaneous interactions in clinics, homes, and schools, for example, and result in generalizable findings that may be used to improve educational and intervention services (Grey et al. 2007; Kasari et al. 2006; Tardif et al. 1995). This is also seen in studies that use videos to document and detail specific behaviors in specific contexts, as with the Thunberg, Ahlsen, and Sandberg (2007) description of four children's use of speech-generating devices in their homes and with the Stribling, Rae, and Dickerson (2007) description of a girl's use of different forms of repetition in her classroom. Video creates additional opportunities as well. In terms of investigating issues for which clinical and standardized assessments may be problematic (i.e., assessing intelligence in nonverbal individuals with autism or relating daily functioning capacities with cognitive test scores), ecological validation, or the comparison of assessment rationale/scores with that of everyday behaviors, is made possible. This can provide clinicians with more information about how standardized measures might relate to daily life. Seal and Bonvillian (1997), for instance, correlated standardized measures of apraxia to the videotaped sign language production of 14 children and adolescents with autism in classroom settings and determined a relation between movement ability and vocabulary size. Though their study was not designed for

purposes of ecological validation, it provides an example of potential applications. Such studies may help to modify or supplement standardized assessments, if needed. Finally, not only researchers can collect video. The involvement of individuals with autism, caregivers, teachers, and others in research may be facilitated by the exchange of personal videos, making collaborative efforts more common.

See Also

- ▶ Video Games, Use of
- ▶ Video Instruction

References and Reading

- Adamson, L. B., Bakeman, R., Deckner, D. F., & Ronski, M. (2009). Joint engagement and the emergence of language in children with autism and Down syndrome. *Journal of Autism and Developmental Disorders*, 39(1), 84–96.
- Adrien, J. L., Lenoir, P., Martineau, J., Perrot, A., Haneury, L., Larmande, C., & Sauvage, D. (1993). Blind ratings of early symptoms of autism based upon family home movies. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32(3), 617–626.
- Baranek, G. T. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders*, 29(3), 213–224.
- Capps, L., Losh, M., & Thurber, C. (2000). “The frog ate the bug and made his mouth sad”: Narrative competence in children with autism. *Journal of Abnormal Child Psychology*, 28(2), 193–204.
- Colgan, S. E., Lanter, E., McComish, C., Watson, L. R., Crais, E. R., & Baranek, G. T. (2006). Analysis of social interaction gestures in infants with autism. *Child Neuropsychology*, 12(4–5), 307–319.
- Erickson, F. (1986). Qualitative methods in research on teaching. In M. Wittrock (Ed.), *Handbook of research on teaching* (3rd ed.). New York: Macmillan.
- Grey, I. M., Bruton, C., Honan, R., McGuinness, R., & Daly, M. (2007). Co-operative learning for children with an Autistic Spectrum Disorder (ASD) in mainstream and special class settings: An exploratory study. *Educational Psychology in Practice*, 23(4), 317–327.
- Gulsrud, A. C., Jahromi, L. B., & Kasari, C. (2010). The co-regulation of emotions between mothers and their children with autism. *Journal of Autism and Developmental Disorders*, 40(2), 227–237.
- Hutman, T., Rozga, A., DeLaurentis, A. D., Barnwell, J. M., Sugar, C. A., & Sigman, M. (2010). Response to distress in infants at risk for autism: A prospective longitudinal study. *Journal of Child Psychology and Psychiatry*, 51(9), 1010–1020.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in young children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry*, 47(6), 611–620.
- Kasari, C., Paparella, T., Freeman, S., & Jahromi, L. B. (2008). Language outcome in autism: Randomized comparison of joint attention and play interventions. *Journal of Consulting and Clinical Psychology*, 76(1), 125–137.
- Kasari, C., Gulsrud, A. C., Wong, C., Kwon, S., & Locke, J. (2010). Randomized controlled caregiver mediated joint engagement intervention for toddlers with autism. *Journal of Autism and Developmental Disorders*, 40(9), 1045–1056.
- Kremer-Sadlik, T. (2004). How children with autism and Asperger syndrome respond to questions: A ‘naturalistic’ theory of mind task. *Discourse Studies*, 6(2), 185–206.
- Losche, G. (1990). Sensorimotor and action development in autistic children from infancy to early childhood. *Journal of Child Psychology and Psychiatry*, 31, 749–761.
- Losh, M., & Capps, L. (2006). Understanding of emotional experience in autism: Insights from the personal accounts of high-functioning children with autism. *Developmental Psychology*, 42(5), 809–818.
- Luyster, R. J., Kadlec, M. B., Carter, A., & Tager-Flusberg, H. (2008). Language assessment and development in toddlers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(8), 1426–1438.
- Maynard, D. W. (2005). Social actions, gestalt coherence, and designations of disability: Lessons from and about autism. *Social Problems*, 52(4), 499–524.
- Mundy, P., Sigman, M., Ungerer, J., & Sherman, T. (1986). Defining the social deficits of autism: The contribution of nonverbal-communication measures. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 27(5), 657–669.
- Ochs, E., Kremer-Sadlik, T., Sirota, K. G., & Solomon, O. (2004). Autism and the social world: An anthropological perspective. *Discourse Studies*, 6(2), 147–183.
- Ochs, E., Solomon, O., & Sterponi, L. (2005). Limitations and transformations of habitus in child-directed communication. *Discourse Studies*, 7(4–5), 547–583.
- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of 1st birthday videotapes. *Journal of Autism and Developmental Disorders*, 24(3), 247–257.
- Ozonoff, S., Iosif, A. M., Baguio, F., Cook, I. C., Hill, M. M., Hutman, T., et al. (2010). A prospective study of the emergence of early behavioral signs of autism.

Journal of the American Academy of Child and Adolescent Psychiatry, 49(3), 256–266.

- Rogers, S. J., Hayden, D., Hepburn, S., Charlifue-Smith, R., Hall, T., & Hayes, A. (2006). Teaching young nonverbal children with autism useful speech: A pilot study of the Denver model and PROMPT interventions. *Journal of Autism and Developmental Disorders*, 36(8), 1007–1024.
- Rozga, A., Hutman, T., Young, G. S., Rogers, S. J., Ozonoff, S., Dapretto, M., & Sigman, M. (2011). Behavioral profiles of affected and unaffected siblings of children with autism: Contribution of measures of mother-infant interaction and nonverbal communication. *Journal of Autism and Developmental Disorders*, 41, 287–301.
- Seal, B. C., & Bonvillian, J. D. (1997). Sign language and motor functioning in students with autistic disorder. *Journal of Autism and Developmental Disorders*, 27(4), 437–466.
- Siller, M., & Sigman, M. (2002). The behaviors of parents of children with autism predict the subsequent development of their children's communication. *Journal of Autism and Developmental Disorders*, 32(2), 77–89.
- Siller, M., & Sigman, M. (2008). Modeling longitudinal change in the language abilities of children with autism: Parent behaviors and child characteristics as predictors of change. *Developmental Psychology*, 44(6), 1691–1704.
- Stribling, P., Rae, J., & Dickerson, P. (2007). Two forms of spoken repetition in a girl with autism. *International Journal of Language & Communication Disorders*, 42(4), 427–444.
- Swensen, L. D., Kelley, E., Fein, D., & Naigles, L. R. (2007). Processes of language acquisition in children with autism: Evidence from preferential looking. *Child Development*, 78(2), 542–557.
- Tager-Flusberg, H., Calkins, S., Nolin, T., Baumberger, T., Anderson, M., & Chadwick, A. (1990). A longitudinal study of language acquisition in autistic and Down syndrome children. *Journal of Autism and Developmental Disorders*, 20(1), 1–21.
- Tardif, C., Plumet, M. H., Beaudichon, J., Waller, D., Bouvard, M., & Leboyer, M. (1995). Micro-analysis of social interactions between autistic children and normal adults in semi-structured play situations. *International Journal of Behavioral Development*, 18(4), 727–747.
- Thunberg, G., Ahlsen, E., & Sandberg, A. D. (2007). Children with autistic spectrum disorders and speech-generating devices: Communication in different activities at home. *Clinical Linguistics & Phonetics*, 21(6), 457–479.
- Ungerer, J., & Sigman, M. (1981). Symbolic play and language comprehension in autistic children. *American Academy of Child Psychiatry*, 20, 318–337.
- Wetherby, A. M., Woods, J., Allen, L., Cleary, J., Dickinson, H., & Lord, C. (2004). Early indicators of autism spectrum disorders in the second year of life. *Journal of Autism and Developmental Disorders*, 34(5), 473–493.

Vineland Adaptive Behavior Scales (VABS)

► [Autism Family Experience Questionnaire \(AFEQ\)](#)

► [Autism Family Experience Questionnaire \(AFEQ\), The](#)

Vineland III

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Synonyms

[Vineland-3](#)

Description

The Vineland Adaptive Behavior Scales, Third Edition (Vineland-3; 2016) is the third revision of the first standardized measure of adaptive behavior, the Vineland Social Maturity Scale (VSMS) (Doll 1935). Adaptive behavior can be defined as a collection of conceptual, social, and practical skills that are necessary to live independently and function appropriately in one's daily environments. The Vineland Adaptive Behavior Scales (Vineland ABS), the first revision of the VSMS, was published in 1984 (survey and expanded forms) and 1985 (classroom edition). The second revision, the Vineland II, was published in 2005 (survey and parent forms).

The Teacher Rating Form of the Vineland-II was published in 2006, and the Expanded Interview Form was published in 2008. Together, the Vineland ABS, Vineland II, and Vineland-3 are the most widely used measures of adaptive behavior.

The Vineland-3 assesses three core adaptive behavior domains: communication, including receptive, expressive, and written communication; daily living skills, or the ability to take care of one's daily needs, which include personal (e.g., dressing and hygiene), domestic (e.g., household chores), and community (e.g., managing money) activities; and socialization, or the ability to form developmentally appropriate interpersonal relationships, engage in developmentally appropriate play and leisure activities, and demonstrate developmentally appropriate coping skills. The gross and fine motor skills of children ages 9 and younger can be assessed within the motor skills domain. However, to be consistent with the Diagnostic and Statistical Manual, Fifth Edition (DSM-5) criteria for intellectual disability, which excludes motor impairments, the motor skills domain of the Vineland-3 is now optional. In addition, the optional maladaptive behavior domain, which measures maladaptive behaviors that may interfere with an individual's adaptive functioning, is now available for the Interview, Parent/Caregiver, and Teacher Forms. The maladaptive behavior domain includes optional maladaptive internalizing (e.g., symptoms of anxiety and depression), externalizing (e.g., symptoms of hyperactivity and impulsivity), and critical items that measure more severe maladaptive behaviors but are not scored as a subscale or composite (e.g., suicidality, fire setting).

Like the Vineland II, the Vineland-3 has three comprehensive administration forms: the Interview Form (formerly Survey Interview and Expanded Interview Forms combined), the Parent/Caregiver Form (formerly Parent/Caregiver Rating Form), and the Teacher Form (formerly Teacher Rating Form). The Vineland-3 also includes a brief domain-level administration version of each of the three comprehensive administration forms. The brief Domain-Level Forms consist of fewer items than the comprehensive forms and are generally used to determine diagnostic eligibility for

intellectual disability. The six forms are available for administration via paper and pencil or online using Pearson's Q-global™ platform, an online-testing platform that provides automated scoring and reporting. The Interview and Parent/Caregiver Forms can be administered for individuals across the lifespan (from birth through age 90 years). The Teacher Form can be administered to children ages 3 to 21 years. The brief Domain-Level Forms can be administered to ages 3 to 21 years.

The Vineland-3 yields an overall adaptive behavior composite (ABC) standard score (mean of 100 and a standard deviation of 15) as well as standard scores for each of the adaptive behavior domains, percentile ranks, adaptive levels, and stanines. The communication, daily living skills, socialization, and motor skills domains are comprised of age-based subdomains. In the comprehensive versions of the Vineland-3, *v*-scaled scores (mean of 15 and an *SD* of 3) and age equivalents are also derived for the subdomains. The Domain-Level Forms yield an ABC and domain standard scores.

The Vineland ABS is the most commonly used measure of adaptive behavior among individuals with autism spectrum disorder (ASD). Adaptive behavior is an important determinant of prognosis in ASD. Given the hallmark features of ASD, it is not surprising that individuals with ASD exhibit deficits in their adaptive behavior. Researchers have attempted to uncover a typical profile of adaptive behavior among individuals with ASD. However, the pattern of adaptive behavior observed among individuals with ASD differ based on several important factors, including participant's cognitive abilities and age as well as the research methodology (e.g., use of standard scores or age-equivalent scores). Generally, individuals with ASD who have average or above average cognitive abilities demonstrate a profile of adaptive behavior that is characterized by relatively strong daily living skills, significant deficits in socialization skills, and moderate deficits in communication (Kanne et al. 2011). While this profile of adaptive behavior is often referred to as the "typical" autism profile of adaptive behavior, other profiles of adaptive functioning have been found among individuals with ASD. For example,

the Vineland-II profiles of toddlers with below average cognitive functioning are characterized by higher socialization skills followed by lower communication and daily living skills (Ray-Subramanian et al. 2011). To date, no studies investigating the adaptive behavior of individuals with ASD using the Vineland-3 have been published. Of notable importance is the significant gap between cognition and adaptive functioning in individuals with ASD without cognitive impairment that appears to be greater in older vs. younger individuals (e.g., Klin et al. 2007; Kanne et al. 2011). Yet, this gap does not appear to exist in individuals with ASD who have cognitive impairment. In fact, recent studies actually show adaptive skills in these individuals to fall above cognitive level (e.g., Perry et al. 2009; Kanne et al. 2011).

Historical Background

The Vineland-3 measures the construct of adaptive behavior. Adaptive behavior is an age-based construct that is rooted in the definition of intellectual disability (formerly mental retardation). The Vineland ABS, (Sparrow et al. 1984a, b, 1985) represented a significant revision of the VSMS (Doll 1935). The Vineland II (Sparrow et al. 2005, 2006, 2008) and Vineland-3 are revisions of the Vineland ABS. The revision from the Vineland-II to the Vineland-3 included updated norms and major content updates (e.g., modified items and new items). These content updates were made to improve the cultural sensitivity of items and to reflect cultural changes and new scientific knowledge of developmental disabilities since the publication of the Vineland-II. Additionally, the items on the Parent/Caregiver Form were reworded to reflect a fifth-grade reading level. For this reason, the Interview and Parent/Caregiver Forms were normed separately, unlike the Vineland-II where they were normed together given the identical wording of items.

Unlike previous versions of the Vineland, the Vineland-3 can be administered online using Q-global™, which provides computerized scoring and reporting for both online and paper-and-

pencil administrations of the Vineland. In the online administration of the Vineland-3, the program calculates basals and ceilings, and suggests additional probes and interview questions to improve the ease of administration. Score reporting includes item-level comparisons that are provided to monitor progress and to make interrater comparisons. Furthermore, intervention planning recommendations are provided at the item-level.

Psychometric Data

The Vineland-3 was standardized on 2560 individuals from birth to 90 years of age. The standardization sample was stratified according to the 2014 US census on sex, race/ethnicity, individuals' or parents' education level, and geographic region. Additionally, data were collected on seven clinical samples that align with the Individuals with Disabilities Education Act (IDEA) disability categories. The seven clinical samples include autism, developmental delay, emotional disturbance, intellectual disability, specific learning disability, speech or language impairment, and all other IDEA disability categories. Research suggests that individuals in these clinical samples often demonstrate distinct patterns of adaptive behavior on the ABS that may discriminate between groups.

The Vineland-3 has strong psychometric properties. Interrater and inter-interviewer reliability coefficients ranged from 0.70 to 0.81 for the Comprehensive Interview Form and 0.69 to 0.84 for the Domain-Level Interview Form. Internal Consistency reliability ranged from 0.90 to 0.98 for the Comprehensive Interview Form and from 0.86 to 0.97 for the Domain-Level Form. For individuals ages 13 and older, test-retest reliabilities for the Comprehensive Interview Form ranged from 0.81 to 0.92 and from 0.73 to 0.88 for the Domain-Level Form. These represent good to excellent levels of reliability by the criteria of Cicchetti and Sparrow (1981); whereby: <0.40 = Poor; 0.40–0.59 = Fair; 0.60–0.74 = Good; and ≥ 0.75 = Excellent levels of chance-corrected interrater agreement; and for internal consistency,

whereby <70 = Poor; 70–79 = Fair; 80–89 = Good; and 90 and above = Excellent (Cicchetti 1994).

The validity of the Vineland-3 has been examined based on the content, structure, and relationships with other measures of adaptive behavior, including the Vineland-II and the Adaptive Behavior Assessment System, Third Edition (ABAS-3), as well as measures of developmental functioning (e.g., the Bayley Scales of Infant Development, Third Edition).

Clinical Uses

The Vineland scales are most frequently used to evaluate whether an individual meets diagnostic criteria for intellectual disability, as an individual with intellectual disability must have significant deficits in both their cognitive and adaptive functioning. Recently, the Vineland scales have been used to aid in the diagnoses of many disorders beyond intellectual disability, including ASD. Because the Vineland-3 measures adaptive behavior from birth onward, it is an ideal tool for developmental evaluations. In addition to assisting with diagnostic classifications, the Vineland-3 can be useful in program planning and evaluation. Although all forms of the Vineland-3 can be used in program planning, the comprehensive versions of the Vineland-3 cover a much broader range of adaptive behaviors that can be identified and targeted for intervention than the Domain-Level versions. There is also a new Intervention Guidance that examiners can use to determine specific adaptive delays to work on in intervention programming. Research suggests that interventions that aim to improve specific adaptive behaviors in high-functioning individuals with ASD may lead to better outcomes than those that target autistic symptomatology (e.g., Farley et al. 2009). One of the goals of most interventions is to improve how well an individual functions in everyday life, therefore, the Vineland-3 is an excellent progress monitoring tool as it is sensitive to small changes in adaptive functioning and can be used more frequently than other assessment tools (e.g., cognitive measures). The

Vineland scales are commonly used measures of adaptive functioning in research studies. In fact, the Vineland scales are the most commonly used adaptive behavior measure in the extant ASD literature. The Vineland-3 is anticipated to be as commonly used in future research as its predecessors. It should also be noted that the Vineland Adaptive Behavior Scales have been designated by the National Institutes of Health's National Database for Autism Research as the adaptive behavior instrument of choice for multisite studies of Autism.

See Also

► [Vineland Adaptive Behavior Scales \(VABS\)](#)

References and Reading

- Cicchetti, D. V. (1994). Guidelines, criteria, and rules of thumb for evaluating normed and standardized instruments in psychology. *Psychological Assessment*, 6, 284–290.
- Cicchetti, D. V., & Sparrow, S. S. (1981). Developing criteria for establishing interrater reliability of specific items: Applications to assessment of adaptive behavior. *American Journal of Mental Deficiency*, 86, 127–137.
- Doll, E. A. (1935). A genetic scale of social maturity. *The American Journal of Orthopsychiatry*, 5, 180–188.
- Farley, M. A., McMahon, W. M., Fombonne, E., Jenson, W. R., Miller, J., Gardner, M., Block, H., Pingree, C. B., Ritvo, E. R., Ritvo, R. A., & Coon, H. (2009). Twenty-year outcome for individuals with autism and average or near-average cognitive abilities. *Autism Research*, 2(2), 109–118.
- Kanne, S. M., Gerber, A. J., Quirnbach, L. M., Sparrow, S. S., Cicchetti, D. V., & Saulnier, C. A. (2011). The role of adaptive behavior in autism spectrum disorders: Implications for functional outcome. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-010-1126-4>.
- Klin, A., Saulnier, C. A., Sparrow, S. S., Cicchetti, D. V., Volkmar, F. R., & Lord, C. (2007). Social and communication abilities and disabilities in higher functioning individuals with autism spectrum disorders: The Vineland and the ADOS. *Journal of Autism and Developmental Disorders*, 37, 748–759.
- Perry, A., Flanagan, H. E., Dunn Geier, J., & Freeman, N. L. (2009). Brief report: The Vineland adaptive behavior scales in young children with ASD at different cognitive levels. *Journal of Autism and Developmental Disorders*, 39(7), 1066–1078.

- Ray-Subramanian, C. E., Huai, N., & Weismer, S. (2011). Brief report: Adaptive behavior and cognitive skills for toddlers on the autism spectrum. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-010-1083-y>.
- Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1984a). *The Vineland adaptive behavior scales: Interview edition, survey form*. Circle Pines: American Guidance Service.
- Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1984b). *The Vineland adaptive behavior scales: Interview edition, expanded form*. Circle Pines: American Guidance Service.
- Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1985). *The Vineland adaptive behavior scales: Classroom edition*. Circle Pines: American Guidance Service.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales: Second edition (Vineland II), survey form/parent/caregiver rating form*. Bloomington: Pearson Assessments.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2006). *Vineland adaptive behavior scales: Second edition (Vineland II), teacher report form*. Bloomington: Pearson Assessments.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2008). *Vineland adaptive behavior scales: Second edition (Vineland II), expanded interview form*. Bloomington: Pearson Assessments.
- Sparrow, S. S., Cicchetti, D. V., & Saulnier, C. A. (2016). *Vineland adaptive behavior scales: Third edition (Vineland-3)*. Bloomington: NCS Pearson.

Vineland-3

► Vineland III

Violence and ASD

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Definition

Violence is defined as the purposeful and malicious injury of another person (Blackburn 1993).

From a behavior analytic perspective, it can present in multiple expressions, be preceded by various antecedents, and be maintained by numerous consequences. Instrumental violence refers to acts that are goal oriented, measured, and dispassionate (Berkowitz 1993). Expressive violence is characterized by impulsivity and mercurial emotions and may be in response to an intolerable level of physical or emotional dysregulation (McGuire 2008).

Historical Background

Violence is not a core feature of autism spectrum disorder (ASD) and is not our intent to vilify people with ASD. Indeed, people with disabilities are three times more likely to be victims of nonfatal crimes than people without disabilities. A reported 94% of people with Asperger's syndrome (AS) have been the victims of bullying (Little 2002). According to a report from the US Department of Justice (Harrell 2015), people with disabilities were the victims of 1.3 million violent crimes in 2012. This included the crimes of assault (simple and aggravated), rape, sexual assault, and robbery. With that context in mind, there are times when people with ASD do come in contact with the criminal justice system. When people on the autism spectrum do commit violence, this may be due to a multitude of factors related to their ASD including lack of access to circumscribed interest, violation of a routine, response to stress, sensory response, a circumscribed interest in violence, communication challenges, and difficulties reading social situations. Substance abuse and comorbid psychopathology confer significant increased risk of violent offending (Langstrom et al. 2009).

What remains unclear is the prevalence of people with ASD who commit violent crimes relative to their prevalence in general society. What factors place a person with ASD at risk to engage in a violent act and are these the same risk factors that confer increased risk of violence in the neurotypical population?

Prevalence in the Criminal Justice System

People with ASD are not inherently violent, though they may be overrepresented within the criminal justice system relative to their proportion in general society. The difficulty with making this assertion confidently lies, naturally, in a current lack of solid data. There are, however, a number of important aspects of sampling and psychological assessment – specifically, its deficiencies with regard to ASD – that suggest the overrepresentation of people with ASD within the criminal justice system is possible and perhaps likely.

A 2014 study demonstrated the difficulty of finding reliable samples of adult offenders with ASD (Allely et al. 2014). Here the authors examined a large group of serial and mass murderers for evidence of psychosocial stressors such as abuse or neglect, head injury, and ASD. While the authors made an admirable attempt to source their work with databases, books, and peer-reviewed articles, the resulting ASD condition remained overly broad, with the categories “definite,” “highly probable,” and “possible” based, out of necessity, on some amount of conjecture from source data.

The age of a person with ASD presents another sampling issue. As assessment and diagnosis of ASD has become much more widespread, public perception is that the prevalence of ASD in the general population is increasing. This is likely not the case, as wider availability of qualified diagnostic professionals, broader diagnostic criteria, and better record keeping all contribute to a rise in both prevalence and incidence estimates (Elsabbagh et al. 2012). A recent epidemiological study in the UK found that indeed, the prevalence of ASD appears to be consistent across age groups (Brugha et al. 2011). When we consider this in light of the fact that youth with ASD are, at least in some jurisdictions, less likely to be prosecuted due to their diagnosis (Cheely et al. 2011), we must also consider the possibility that older adults, who are less likely to have been diagnosed, are also unlikely to have had ASD presented as a mitigating circumstance in prosecution. The prevalence of ASD among those within the criminal justice system may be higher than the general population due to lack of diagnosis because of

age, and the lack of diagnosis itself presents a problem for estimation sampling.

Tools for the assessment of violence risk are an important part of the clinician’s toolkit. They have seen a number of refinements over the last five decades, with statistical prediction of risk in the second generation of assessments, the inclusion of dynamic risk factors (e.g., substance abuse, interpersonal conflict) in the third generation, and integration with interventions and assessment of rehabilitation progress in the fourth generation (Campbell et al. 2009). While Campbell et al. and others have demonstrated the utility of dynamic factors in assessment of neurotypical populations (Schwalbe 2007; Walters 2006), it should be noted that exactly how dynamic factors affect the likelihood of violence in persons with ASD is not well understood. The result is that a clinician who wishes to assess the risk of violence in a person with ASD either realizes he lacks a complete set of tools for doing so or uses an assessment that does not have demonstrated validity with ASD populations.

Clinical judgment can be utilized to assess risk of violence, and this was in fact the basis for first-generation assessments. But it is prone to error and bias (Grove et al. 2000). Compounding this is a possible pattern: People with ASD may be more likely to self-report violent behavior than other types of law-breaking behavior (Woodbury-Smith et al. 2006), resulting in a higher judgment-based violence risk assessment.

Risk Factors

In criminology, risk predictor variables are dynamic factors that include the consideration of “risk of harm” and “risk level.” Risk of harm reflects the extent and nature of the behavior that may occur. Risk level is a measure of the likelihood of the behavior occurring (McSherry 2004). Kawakami et al. (2012) found that for people with ASD, risk of engaging in criminal behavior was significantly correlated with a history of abuse and neglect as well as a late diagnosis and resultant delayed onset of services.

In a UK study of 16 adults with a clinical diagnosis of Asperger’s who had been in contact with the criminal justice system due to offending

behaviors, Allen et al. (2008) identified behavioral difficulties as well as precipitating and predisposing factors related to the commission of criminal acts. Utilizing a project-created questionnaire administered to service providers involved with the participants, the researchers identified the following extant behavioral difficulties: verbal aggression (88%), physical aggression (75%), property destruction (69%), displaying sexually inappropriate behavior (38%), substance abuse (38%), and overactivity (38%). The service providers also identified the following underlying factors which they believed predisposed the people with ASD to commit crimes: lack of concern for others (94%), social naivety (88%), lack of awareness of outcome (82%), impulsivity (63%), misinterpretation of rules (63%), and obsessional interests (44%). Factors that occasioned offending behaviors included social rejection (69%), bullying (50%), sexual rejection (50%), family conflict (50%), decline in mental health (31%), changes of residence (25%), change in care provision/support (19%), and grief (13%). The cumulative effect of stress was identified most often as the immediate trigger for an offending behavior.

Current Knowledge

Instrumental Violence

Instrumental violence involves acts that are likely to be perpetrated in an emotionally detached way and represent a means to an end behavior (Harkins et al. 2012). The following features of ASD may be implicated in the commission of acts of instrumental violence.

Violation of a Routine/Lack of Access to Circumscribed Interest

Ehlers and Gillberg (1993) noted that people on the autism spectrum often enforced rigid routines not only on themselves but on others as well. In a study of patients on the autism spectrum in a forensic psychiatric setting, Hare et al. (1999) found that 84% had nonfunctional routines which they adhered to in an inflexible manner.

These routines included holding sticks in each hand, listening to the same music over and over, touching things, and flushing toilets. The study also examined differences in circumscribed interests and routines between patients with and without ASD. Statistically significant between groups differences were found ($X^2 = 115.40$ df 2, $p < .0001$). When these routines are violated, a person on the spectrum may engage in by-all-means-necessary behavior to maintain that routine, resulting in harm to themselves or others (Hare et al. 1999).

An all-consuming preoccupation with circumscribed interests and the denial of access to those interests may also serve as a reliable trigger for violence (Barry-Walsh and Mullen 2004). Haskins and Silva (2006) posit that circumscribed interests combined with deficits in theory of mind (ToM) and weak central coherence may result in the development of intense and fixated interests uninhibited by future consequences and social norms that result in criminal behavior. Moreover, differences in perspective taking and executive functioning (Allen et al. 2008) may place them at risk for not considering the impact of their behavior on others or the legal consequences of their actions.

Circumscribed Interest in Violence

Hare et al. (1999) found that 25% of the forensic patients with ASD in their study had circumscribed interests in violence, including Nazism. In some of the cases, the violent interests were connected to the crime committed.

On December 14, 2012, Adam Lanza, a young man on the autism spectrum and comorbid mental health issues, shot and killed his mother with a .22 caliber rifle. He then drove to Sandy Hook Elementary School. He was heavily armed and shot through the school's security doors. He shot and killed the school principal and psychologist. He entered two first grade classrooms and shot and killed a total of 20 children and four adults with a Bushmaster rifle. Adam Lanza died of a self-inflicted gunshot wound to the head (Sedensky 2013).

Lanza's circumscribed interests included mass killings in general and the Columbine school

massacre in particular. He was trained in the use of high-powered weapons and had easy access to guns and ammunition. Lanza had a history of being bullied and difficulty at school. He had poor relationships with his family and an absence of friendships. Mental health professionals who had provided services for Lanza reported that there was nothing in his behavior that could have foreseen his actions, but a search of his home revealed that Lanza had an intense preoccupation with mass shootings. Lanza's circumscribed interest in violence appears to have started as early as elementary school, when in fifth grade he wrote a story entitled "Big Book of Granny." In this story, Granny hides a gun in her cane and shoots people, including children. Among the evidence found in his home was an Excel spreadsheet of mass murders dating back to 1891 that described mass murders and the number of casualties. A second spreadsheet was found that read like a task analysis of how to commit a mass murder (Sedensky 2013).

Expressive Violence

Expressive violence describes acts that are impulsive and reactive and may result from unendurable levels of sensory and physiological arousal that can exacerbate communication challenges. The following features of ASD may be implicated in the commission of acts of expressive violence.

Sensory Response

The DSM 5 (APA 2013) includes hyper- or hypo-reactivity to sensory input as part of the diagnostic criteria for ASD. This includes tactile defensiveness, atypical reactions, and at times extreme distress in response to sensory input. Sensory impairments combined with co-occurring intellectual disabilities and psychiatric disorders render a person more susceptible to developing problem behavior (Carvill 2001). There have been cases in which sensory processing challenges have been implicated in the commission of violent acts by people with ASD. Mawson et al. (1985) describe a man with ASD who engaged in an act of aggression toward a barking dog and its owner as the result

of his sensitivity to high-pitched sounds. Schwartz-Watts (2005) describes a man with ASD who shot and killed an 8-year-old after the boy ran over his foot with a bicycle. The courts ruled that tactile defensiveness precipitated the violent behavior. These cases demonstrate that untenable sensory experiences may lead to a response of such significant magnitude that it results in a criminal act.

Communication Challenges

Impairments in receptive communication can make it difficult for the person with ASD to understand what is being asked of them, while deficits in expressive communication can make it difficult to get their wants and needs met. Difficulties in the use and understanding of nonverbal behaviors may set the stage for misunderstandings with others and have the potential to negatively impact the ability to accurately read social situations. Combined, these communication challenges may lead to confusion, stress, and irritation which can be expressed as problem behavior (Dominick et al. 2007). Stress has a deleterious effect on the ability of a person with ASD to comprehend and utilize their language which can often result in aggressive acts as an alternative form of communication (Baron 2006).

Difficulties Reading Social Situations

In the general population, the social features that place people at risk for a propensity toward violent behavior later in life include poor social skills, poorly developed social networks, and defiance of authority (Farrington and Loeber 2000). A core feature of ASD is a qualitative impairment in social interactions and delays in the development of social reasoning. People with ASD experience challenges establishing and maintaining friendships, have difficulty interpreting social cues, and often engage in improper social behaviors (Siponmaa et al. 2001). While it is more likely that people with ASD will be duped into victimization as the result of their social naiveté, there are times when their misunderstanding of social rules and misreading social cues may lead them to commit an act of violence (Murrie et al. 2002). Barry-Walsh

and Mullen (2004) describe the case of a man who attempted to choke a young woman after she rebuffed his sexual overtures.

It is essential to reiterate that violence is not a core feature of ASD. The core features of ASD including social, communication, sensory, and behavioral challenges may render the person more vulnerable to environmental triggers, which could result in acts of violence.

Future Directions

It is clear that the features of ASD can lead people with the disorder to commit acts of violence that are qualitatively different from those committed by people without ASD in terms of the triggers and topography. Much more study of both populations within the criminal justice system is necessary to accurately determine proportional rates of incarceration for people with ASD and to work toward just mitigation in sentencing and prevention of recidivism. Future research should focus on the expression of behavioral challenges and associated features endemic to autism which might indicate future acts of violence. This could serve as a mechanism to develop appropriate interventions with a greater likelihood of success in reducing recidivism, as they consider the underlying mechanisms specific to ASD that resulted in violent behavior.

Additional research is also needed to understand the similarities and differences that exist in risk estimation between the population of offenders with ASD and comorbid psychiatric conditions and offenders who represent the neurotypical population. Current risk assessments do not consider factors unique to ASD, and data from this research would ultimately inform (perhaps transform) risk assessment procedures and guide more accurate understanding and communication of these risks.

See Also

- ▶ [Diminished Responsibility](#)
- ▶ [Video Games and Violence](#)

References and Readings

- Allely, C. S., Minnis, H., Thompson, L., Wilson, P., & Gillberg, C. (2014). Neurodevelopmental and psychosocial risk factors in serial killer and mass murderers. *Aggression and Violent Behavior, 19*, 288–301.
- Allen, D., Evans, C., Hider, A., Hawkins, S., Peckett, H., & Morgan, H. (2008). Offending behavior in adults with Asperger's Syndrome. *Journal of Autism and Developmental Disorders, 38*, 748–758.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- Baron, M. G. (2006). *Stress and coping in autism*. New York: Oxford University Press.
- Barry-Walsh, J., & Mullen, P. (2004). Forensic aspects of Asperger's Syndrome. *The Journal of Forensic Psychiatry and Psychology, 15*(1), 96–107.
- Berkowitz, L. (1993). *Aggression: Its causes, consequences, and control*. New York: McGraw-Hill Book Company.
- Blackburn, R. (1993). *The psychology of criminal conduct: Theory, research and practice*. London: John Wiley & Sons, Ltd.
- Bjorkly, S. (2009). Risk and dynamics of violence in Asperger's syndrome: A systematic review of the literature. *Aggression and Violent Behavior, 14*(5), 306–312.
- Brugha, T. S., McManus, S., Bankart, J., Scott, F., Purdon, S., Smith, J., Bebbington, P., Jenkins, R., & Meltzer, H. (2011). Epidemiology of autism spectrum disorders in adults in the community in England. *Archives of General Psychiatry, 68*(5), 459–466.
- Campbell, M. A., French, S., & Gendreau, P. (2009). The prediction of violence in adult offenders: A meta-analytic comparison of instruments and methods of assessment. *Criminal Justice and Behavior, 36*(6), 567–590.
- Carvill, S. (2001). Sensory impairments, intellectual disability and psychiatry. *Journal of Intellectual Disability Research, 45*(6), 467–483.
- Cheely, C. A., Carpenter, L. A., Letourneau, E. J., Nicholas, J. S., Charles, J., & King, L. B. (2011). The prevalence of youth with autism spectrum disorders in the criminal justice system. *Journal of Autism and Developmental Disorders, 42*, 1856–1862.
- Dixon, L., & Bowen, E. (2012). Intimate partner violence and stalking. In G. Davies & A. Beech (Eds.), *Forensic psychology: Crime, justice, law, interventions* (pp. 189–208). West Sussex: John Wiley & Sons, Ltd..
- Dominick, K. C., Davis, N. O., Lainhart, J., Tager-Flusberg, H., & Folstein, S. (2007). Atypical behaviors in children with autism and children with a history of language impairment. *Research in Developmental Disabilities, 28*(2), 145–162.
- Ehlers, S., & Gillberg, C. (1993). The epidemiology of Asperger syndrome: A total population study. *Journal of Child Psychology & Psychiatry, 34*, 1327–1350.

- Elsabbagh, M., Divan, G., Koh, Y., Kim, Y., Kauchali, S., Marcin, C., Montiel-Nava, C., Patel, V., Paula, C. S., Wang, C., Yasamy, M. T., & Fombonne, E. (2012). Global prevalence of autism and other pervasive developmental disorders. *International Society for Autism Research*, 5(3), 160–179.
- Farrington, D. P., & Loeber, R. (2000). Epidemiology of juvenile violence. *Juvenile Violence*, 9, 733–748.
- Grove, W. M., Zald, D. H., Lebow, B. S., Snitz, B. E., & Nelson, C. (2000). Clinical versus mechanical prediction: A meta-analysis. *Psychological Assessment*, 12, 19–30.
- Hare, D. J., Gould, J., Mills, R., & Wing, L. (1999). *A preliminary study of individuals with autistic spectrum disorders in three special hospitals in England*. London: National Autistic Society.
- Harkins, L., Ware, J., & Mann, R. (2012). Interventions with dangerous offenders. In G. Davies & A. Beech (Eds.), *Forensic psychology: Crime, justice, law, interventions* (pp. 349–368). London: John Wiley and Sons, Ltd.
- Harrell, E. (2015). Crime against persons with disabilities, 2009–2013–statistical tables.
- Haskins, B., & Silva, J. (2006). Asperger's disorder and criminal behavior: forensic psychiatric considerations. *Journal of the American Academy of Psychiatry and the Law Online*, 34(3), 374–384.
- Kawakami, C., Ohnishi, M., Sugiyama, T., Someki, F., Nakamura, K., & Tsujii, M. (2012). The risk factors for criminal behaviour in high-functioning autism spectrum disorders (HFASDs): a comparison of childhood adversities between individuals with HFASDs who exhibit criminal behaviour and those with HFASD and no criminal histories. *Research in Autism Spectrum Disorders*, 6, 949–957.
- Langstrom, N., Grann, M., Ruchkin, V., Sjostedt, G., & Fazel, S. (2009). Factors for violent offending in autism spectrum disorder: A national study of hospitalized individuals. *Journal of Interpersonal Violence*, 24, 1358–1370.
- Lerner, M. D., Haque, O. S., Northrup, E. C., Lawer, L., & Bursztajn, H. J. (2012). Emerging perspectives on adolescents and young adults with high-functioning autism spectrum disorders, violence, and criminal law. *Journal of the American Academy of Psychiatry and the Law Online*, 40(2), 177–190.
- Little, L. (2002). Middle-class mothers' perceptions of peer and sibling victimization among children with Asperger's syndrome and nonverbal learning disorders. *Issues in Comprehensive Pediatric Nursing*, 25(1), 43–57.
- Mawson, D., Grounds, A., & Tantam, D. (1985). Violence and Asperger's syndrome. *British Journal of Psychiatry*, 147, 566–569.
- McGuire, J. (2008). A review of effective interventions for reducing aggression and violence. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 363(1503), 2577–2597.
- McSherry, B. (2004). Risk assessment by mental health professionals and the prevention of future violent behaviour. *Trends and Issues in Crime and Criminal Justice*, 281.
- Murrie, D., Warren, J., Kristiansson, M., & Dietz, P. E. (2002). Asperger's syndrome in forensic settings. *International Journal of Forensic Mental Health*, 1, 59–70.
- Schwalbe, C. S. (2007). Risk assessment for juvenile justice: A meta-analysis. *Law and Human Behavior*, 31, 449–462.
- Schwartz-Watts, D. (2005). Asperger's disorder and murder. *Journal of the American Academy of Psychiatry and the Law*, 33(3), 390–393.
- Siponmaa, L., Kristiansson, M., Jonson, C., Nyden, A., & Gillberg, C. (2001). Juvenile and young mentally disordered offenders: The role of child neuropsychiatric disorders. *Journal of the American Academy of Psychiatry and the Law*, 29, 420–426.
- Sedensky, S. J. (2013). *Report of the state's attorney for the judicial district of Danbury on the shootings at Sandy Hook Elementary School and 36 Yogananda Street, Newtown, Connecticut on December 14, 2012*. Danbury Connecticut: Office of the Attorney General.
- Tantam, D. (1988). Lifelong eccentricity and social isolation: 1. Psychiatric, social and forensic aspects. *British Journal of Psychiatry*, 153, 777–782.
- Walters, G. D. (2006). Risk-appraisal versus self-report in the prediction of criminal justice outcomes. *Criminal Justice and Behavior*, 33, 179–304.
- Woodbury-Smith, M. R., Clare, I. C. H., Holland, A. J., & Kearns, A. (2006). High functioning autistic spectrum disorders, offending and other law-breaking: Findings from a community sample. *Journal of Forensic Psychiatry & Psychology*, 17(1), 108–120.

Violent/Criminal Behavior in Autism

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Definition

Criminal behavior refers to any acts or omissions that are deemed unlawful, i.e., are contrary to the laws set out in statute by a legislative authority. Such behavior results in a victim, either through (1) direct injury or harm, or (2) damage or loss to their property.

Historical Background

Hans Asperger himself was the first to identify the risk of problematic behaviors when he described four children with core deficits in social interaction, communication, and inflexible patterns of behavior (Asperger 1944, trans. Frith 1991). Among them, three had been excluded from school as a result of aggressive behavior toward their peers. Asperger generalizes these behaviors as representing “autistic acts of malice,” which “appear to be calculated” (Asperger 1944, p. 77) and he adds that “with uncanny certainty, the children manage to do whatever is the most unpleasant or hurtful in a particular situation.” (Asperger 1944, p. 77) However, he does indicate that “because of their poorly developed emotionality they cannot sense how much they hurt others” (Asperger 1944, p. 77), an allusion to both their impairments of empathy and mentalizing (also referred to as “theory of mind”).

Current Knowledge

Unlawful behavior among individuals with autism spectrum disorders is an uncommon occurrence. Indeed, people with ASDs are probably more likely to be victims rather than perpetrators. However, a small number of people with ASDs will, unfortunately, find themselves in contact with the criminal justice system as defendants in connection with alleged criminal behavior, and it is therefore important for clinicians to understand patterns of such behaviors and the relevant etiological factors, as this will guide management and preventative strategies. Moreover, an awareness of the characteristics of ASDs among criminal justice system workers is vital in ensuring that the vulnerabilities of this group are not overlooked and that their rights as vulnerable witnesses or defendants are respected. In this entry, the literature concerning unlawful behavior among people with ASDs is discussed, and the clinical implications of this are expounded. In order to provide context, a discussion of criminological theory is first provided.

Much of the literature is concerned with individuals diagnosed with Asperger syndrome, although, based on current nosology, there is no reason to believe that the behaviors described are specific to Asperger’s vis-à-vis other higher functioning autism spectrum disorders. In contrast, it seems reasonable to believe that among those with ASDs and associated moderate to severe global intellectual disability (mental retardation in the DSM-IV), the risk of unlawful behavior is probably very small.

Overview of Theories of Criminal Behavior: Research with a criminological agenda has identified a number of factors that are associated with an increased vulnerability to offending, including both individual characteristics and factors in the environment. For example, individuals convicted for violent offenses have been shown to score relatively highly on extraversion and neuroticism, dimensions of personality first described by Eysenck. Moreover, two currently recognized personality disorders, antisocial personality disorder (ASPD) and psychopathy, both defined according to the presence of antisocial behaviors, are overrepresented in the criminal justice system and incarcerated populations (Fazel and Danesh 2002). Similarly, individuals with certain mental disorders also appear to have a raised vulnerability (Hodgins 2002). In particular, there is a modest but significant risk of criminal behavior by people with schizophrenia and major affective disorders. Several factors have been identified for both groups as mediating this risk, including associated antisocial behavior since childhood and drug and alcohol abuse. Moreover, among those with schizophrenia, there is some evidence that particular characteristics of the psychosis (such as, e.g., the presence of command hallucinations) may increase vulnerability to criminal behavior (Hodgins 2002).

Longitudinal studies have attempted to integrate environmental and individual factors that operate in a probabilistic way (Farrington 2002) and have identified factors related to family (such as large family size, poor housing, poor or inconsistent parenting, and a parent with a history of convictions), peer group (having friends who are delinquent), and the individual (low IQ and poor

school achievement, truancy, aggressive behavior, and hyperactivity-impulsivity-inattention) as accounting for significant variance in the risk of subsequent offending behavior.

Unfortunately, there are no studies as robust as these that specifically examine factors associated with criminal behavior among those with ASDs. However, there is no reason to believe that the factors identified by Farrington and colleagues will be any less important than in the general population. What is of particular interest, however, is whether factors related to the core autism phenotype increase the propensity to criminal behavior.

ASDs and Criminal Behavior: The Literature: A number of case studies and maximum security psychiatric hospital studies (“special hospitals” in the UK) have raised the awareness that some people with higher functioning ASDs come into contact with the criminal justice system as a result of alleged offending (summarized in Dein and Woodbury-Smith 2010). This has led to (1) speculation concerning whether there is an association between ASDs and offending, an issue that has remained unresolved in the absence of a prevalence study, and (2) an attempt to understand some of the factors associated with offending by the men and women described, an issue that has been beyond the scope of the methodology used.

Wing, in her seminal paper of 1981, “Asperger Syndrome: A Clinical Account,” described a small number of individuals who had a propensity for violent behavior (Wing 1981). One child, for example, with a special interest in chemistry, injured a colleague during the course of a “scientific” experiment. Wing also commented more specifically on how a lack of social understanding could lead to contact with the criminal justice system: “he has no idea of how to indicate his interest and attract a partner in a socially acceptable fashion. If he has a strong sex drive, he may approach or touch or kiss a stranger, or someone much older or younger than himself and as a consequence find himself in trouble with the police” (Wing 1981, p. 116).

Tantam (1988) also studied adults with a lifelong history of social isolation and eccentricity (Tantam 1988), many of whom also met the

diagnostic criteria for AS. Among the 60 cases, forensic information was available on 54 of whom 24 (44%) were reported to have engaged in unlawful behaviors but only 12 (22%) of whom were charged. Fourteen of the 54 had committed offenses punishable by imprisonment, including criminal damage (4), assault (3), arson (3), indecent exposure (3), and attempted rape (1). Clearly, without a comparison group, it is not possible to say whether the prevalence of offending in this group was high. It is also unclear which of those exhibiting problematic behavior met the diagnostic criteria for AS.

In a similar manner, Wolff described a group of children who were socially isolated from their peers (Wolff 2000). Crucially, hers was a longitudinal study, allowing outcomes into adulthood to be examined. In considering the schizoid boys, there was no evidence of any raised prevalence of delinquency, measured by self-report methodology, compared to a control group. However, the self-report results of the schizoid girls indicated that they had rates of delinquency similar to the boys and higher than the control girls. Wolff also approached the Scottish Criminal Records Office to undertake a search of official convictions for the 109 schizoid boys and 32 schizoid girls and the matched controls. Among the population control group, 22% of men and 5% of women had convictions, whereas in contrast 32% of the schizoid men and 34.5% of the schizoid women had convictions. When compared with the control sample, recruited from a psychiatric clinic, the rates for schizoid men were comparable (34.5% of control men with a conviction), whereas the rates for schizoid women were still significantly higher (15.5% of control women with a conviction). Of note is that there was no difference in the nature of offenses between the schizoid and control groups, and none had committed a particularly violent offense. However, 3 years after this survey was undertaken, one of the schizoid men had entered the home of a housewife by posing as a priest, attacked her with a poker, and sexually assaulted her (Wolff 2000).

The numerous case studies published since have described a number of different unlawful behaviors, and it is fair to say that no pattern

concerning type of offense has emerged. While a few have described more serious offenses, such as murder, this is the exception rather than the rule. What does seem true, as discussed subsequently, is that most transgressions occur as a result of social naïveté, a rigid interpretation of rules or interpersonal misunderstanding along with the desire for relationships with others. As a result, people with AS may be arrested following altercations with others, while offenses may take the form of harassment or more seriously sexual assault.

Importantly, being case studies, these publications say nothing about the prevalence of criminal behavior, which remains unknown. While two special hospital studies in the UK have described an overrepresentation of ASDs in such facilities, community studies have indicated that the prevalence is probably no higher than in the general population (Woodbury-Smith et al. 2006). However, what is most interesting regarding these case studies is the insight they provide as to the possible etiological factors that led to the problematic behaviors in each case, as is described in the next section.

The Factors Associated with Offending Among Individuals with ASDs: People with ASDs are generally law-abiding citizens. Indeed, the rigidity of thought that is characteristic of the disorder may result in a very strict adherence to rules. For example, a person with an ASD may be horrified by transgressions as minor as traveling marginally over the speed limit. Strikingly, however, even this intense adherence to rules may result in inadvertent contact with the criminal justice system. By way of a trivial example, imagine the person with AS who confronts someone for talking in the library. He may either risk being assaulted himself, or if his annoyance escalates, he may push the person involved, thereby exposing himself to the risk of charges of assault. Similar infringements have been described in the literature.

One commonly described mediator of problematic behaviors is the pursuit of a circumscribed interest. Such interests form a core part of the phenotype, particularly among those who are higher functioning, and may take different

forms. Often, they involve the acquisition of knowledge, but other forms include collecting and the pursuit of an activity such as a sport or hobby. Regarding the latter, it is their intensity that distinguishes them from the pursuit of such activities among their typically developed peers. In other situations, some people with AS may become particularly interested in certain figures, either from history or present times.

In the research literature, a number of case studies have been described in which people with ASDs have apparently engaged in unlawful behaviors during the pursuit of a circumscribed interest (discussed in Woodbury-Smith et al. 2010). These include theft and burglary in the context of a circumscribed interest, as well as assault. Woodbury-Smith et al. (2010) systematically studied a sample of 21 intellectually able offenders with ASDs, among whom a putative relationship could be established between their index offense and circumscribed interest in six. In addition, when the interests of this group were compared with 23 matched adults with ASDs with no history of offending, interests of a violent nature were overrepresented in the former group.

While offending in the context of a circumscribed interest has formed the focus of several case studies, in day-to-day terms, most transgressions are likely to occur in the setting of social misunderstandings. People with ASDs may be blunt, are likely to speak their mind, and may come across as rude. This may result in offense being taken by another person, and an altercation may follow. In this current discussion, this is particularly pertinent when someone with an ASD is in court as a witness or accused and may come across as arrogant as a result of their communicative bluntness. More generally speaking, people with ASDs may fail to appreciate situations from another's point of view or may be unable to read others' nonverbal cues.

Unlawful transgressions as a result of the social impairment are, perhaps, most likely to occur among isolated young adults who wish for relationships with others but do not necessarily know how to go about achieving such a relationship. They may, therefore, make clumsy or

inappropriate approaches. Moreover, this may be coupled with an inability to read the fearful and/or angry nonverbal cues of (disinterested) others. Under normal situations, these cues would act in an inhibitory manner, but without such mechanisms, the person with AS may continue their attempts at engagement. There is little doubt that in certain situations, this can lead to more serious transgressions. This mechanism may go some way to explain the cases of harassment and/or stalking described in the literature by people with ASDs. Similarly, some cases of inappropriate sexual behavior, including indecent exposure and sexual assault, may be extreme cases of the same mechanism.

There is certainly some evidence to suggest that social transgressions may be associated with an inability to read nonverbal cues and, in particular, the identification of expressions of fear in others (Woodbury-Smith et al. 2005). Interestingly, Woodbury-Smith et al. (2005) also found that the offenders with ASD were significantly better than their non-offending ASD counterparts in theory of mind. This suggests that the relationship between ASDs and unlawful behavior is more complicated, particularly as such neuropsychological profile has also been found among those labeled as psychopaths. It is possible that a small group of people with ASDs may be predisposed to criminality by way of a “double hit,” whereby an additional disorder exists side by side with the ASD as a result of the individual being exposed to both sets of risk factors (Rogers et al. 2006). As a model, this requires further explanation, but it does offer insight that could guide management.

These same mechanisms may also explain some of the cases of aggression that have been described in the literature, including those described by Asperger himself. Asperger believed that such behaviors were indicative of “autistic acts of malice,” characterized by “calculated behavior” (Asperger 1944, op. cit., p. 77) which results in behavior that is the most “unpleasant or hurtful in a particular situation” (Asperger 1944, p. 77). However, an important distinction needs to be made between acts that are purposeful, and

carried out with the intent of upsetting or hurting another person, and acts that are carried out with little understanding of the impact they may have on the other person. Although Asperger used the word “malice,” suggesting a purposeful attempt to hurt emotionally, he is quick to point out that the person with the syndrome he describes “cannot sense how much they hurt others” (Asperger 1944, p. 77). The implication, therefore, is that aggression and other transgressive and unlawful behaviors resulting in a victim may occur without a full appreciation of the distress provoked.

Asperger Syndrome and Criminal Responsibility: A person is deemed not criminally responsible if he lacks the capacity to appreciate the nature and quality of their action or know that their act was wrong. The interpretation of this piece of statute has been the focus of debate and disagreement, particularly with relation to the word “wrong.” In narrow terms, “wrong” may be construed as “breaking the law.” However, in broad terms, it includes knowing that something is both “morally” and “legally” wrong and, in this sense, requires the person to have a knowledge of the law of their jurisdiction but also to have a moral code by which certain (amoral) behaviors are inhibited by them (i.e., having the ability at a cognitive level to empathize with others). In this broader sense, therefore, it might be argued that certain people with ASDs lack the capacity for criminal responsibility. There is no clear guidance on this matter, however (see Fine and Kennett 2004 for a discussion of mental responsibility in connection with psychopathy).

Management Implications: Recognizing that a small number of people with ASDs will come into contact with the criminal justice system, it is important for the key people in the legal arena to be aware of the diagnosis, because of the implications it may have for fitness to plead and stand trial, and criminal responsibility (Dein and Woodbury-Smith 2010). Moreover, people with ASDs may be vulnerable (e.g., suggestible) during the police interview, and their behavior in the courtroom may bias a jury if they do not know the person has the diagnosis. The rehabilitation needs of this group also needs consideration. In the

absence of clear, evidence-based guidelines, assessment and management demand pragmatism (Clare and Woodbury-Smith 2009). However, certain therapeutic practices used for children with ASDs, such as social skills training, may be usefully translated into the adult population to teach a wider set of social rules. Moreover, techniques such as cognitive-behavioral strategies, role playing, and computer-based emotional simulation may go some way to teach “moral capacity,” although the effectiveness of these measures for this purpose has not been tested. Similarly, strategies to reduce problematic circumscribed interests, such as CBT, are not well founded in evidence, although in practical terms, reduction of boredom and the introduction of structure and positive life experiences are likely to help.

Future Directions

In summary, there is still much unknown about the true extent of unlawful behavior among people with ASDs as well as relevant risk and protective factors. Nonetheless, what does seem reasonable to conclude is that a small yet significant number of people with ASDs, particularly those who are functioning within (or just below) the normal range of intelligence, may come into contact with the criminal justice system as a result of alleged unlawful behavior. These infringements are on the whole relatively minor. Their importance lies in the fact that in many cases, a relationship between the behavior and the core clinical and neuropsychological phenotype of the disorder can be hypothesized. Much research is still required, but in the meantime, awareness of the diagnosis among the mental health, police, and legal professions is vital to ensure that people with ASDs are given the best opportunities of rehabilitation, and their vulnerabilities are adequately recognized.

See Also

- [Asperger Syndrome](#)
- [Diminished Responsibility](#)

References and Reading

- Asperger, H. (1944). Die “autistischen Psychopathen” im Kindersalter. *Archiv für psychiatrie und Nervenkrankheiten*, 117, 76–136. In U. Frith (Ed. & Trans.), *Autism and Asperger's Syndrome*. Cambridge: Cambridge University Press. (1991).
- Clare, I. C. H., & Woodbury-Smith, M. (2009). Autism spectrum conditions. In S. Young, M. Kopelman, & G. Gudjonsson (Eds.), *Forensic neuropsychology in practice* (pp. 109–134). Oxford: Oxford University Press.
- Dein, K., & Woodbury-Smith, M. (2010). Asperger syndrome and criminal behaviour. *Advances in Psychiatric Treatment*, 16, 37–43.
- Farrington, D. P. (2002). Key results from the first forty years of the Cambridge study in delinquent development. In T. A. Krohn (Ed.), *Taking stock of delinquency: An overview of findings from contemporary longitudinal studies* (pp. 137–183). New York: Kluwer Academic/Plenum Publishers.
- Fazel, S., & Danesh, J. (2002). Serious mental disorder in 23 000 prisoners: A systematic review of 62 surveys. *Lancet*, 359, 545–550.
- Fine, C., & Kennett, J. (2004). Mental impairment, moral understanding and criminal responsibility: Psychopathy and the purposes of punishment. *International Journal of Law & Psychiatry*, 27, 425–443.
- Hodgins, S. (2002). Research priorities in forensic mental health. *International Journal of Forensic Mental Health*, 1(1), 7–23.
- Rogers, J. S. C., Viding, E., Blair, R. J. R., Frith, U., & Happe, F. (2006). Autism spectrum disorder and psychopathy: Shared cognitive underpinnings or double hit? *Psychological Medicine*, 36, 1789–1798.
- Tantam, D. (1988). Lifelong eccentricity and social isolation. I. Psychiatric, social, and forensic aspects. *British Journal of Psychiatry*, 153, 777–782.
- Wing, L. (1981). Asperger syndrome: A clinical account. *Psychological Medicine*, 11, 115–130.
- Wolff, S. (2000). Schizoid personality in childhood and Asperger syndrome. In A. Klin & F. R. Volkmar (Eds.), *Asperger syndrome* (pp. 278–305). New York: The Guilford Press.
- Woodbury-Smith, M., Clare, I. C. H., Holland, A., Kearns, A., Staufenberg, E., & Watson, P. (2005). A case-control study of offenders with high functioning autistic spectrum disorders. *Journal of Forensic Psychiatry and Psychology*, 16(4), 747–763.
- Woodbury-Smith, M., Clare, I. C. H., Holland, A., & Kearns, A. (2006). High functioning autistic spectrum disorders, offending and other law-breaking: Findings from a community sample. *Journal of Forensic Psychiatry and Psychology*, 17(1), 108–120.
- Woodbury-Smith, M., Clare, I. C. H., Holland, A., Kearns, A., Staufenberg, E., & Watson, P. (2010). Circumscribed interests among offenders with autistic spectrum disorders: A case-control study. *Journal of Forensic Psychiatry and Psychology*, 21(3), 366–377.

Visiting Teacher

► [Itinerant Teacher](#)

Visual Attention to Eyes

► [Mutual Gaze](#)

Visual Cortex

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Synonyms

[Primary visual cortex](#); [V1](#)

Definition

The visual cortex consists of those parts of the brain associated with processing the visual sensory input. Anatomically, these regions correspond most closely to the occipital lobe. Functionally, the visual cortex consists of multiple retinotopically organized maps, i.e., spatially organized by the way that visual input is received on the retina.

See Also

- [Inferior Temporal Area](#)
- [Occipital Lobe](#)
- [Primary Sensory Areas](#)

References and Reading

Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (2000). *Principles of neural science* (4th ed., p. 324). New York: McGraw-Hill.

Visual Evoked Potential (VEP)

► [Evoked Potentials](#)

Visual Narrative Comprehension

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Synonyms

[Comics](#); [Narrative comprehension](#); [Narrative understanding](#); [Visual narrative sequences](#)

Definition

Visual narrative comprehension refers to the access of semantic information in a story presented as pictures, such as a comic or graphic novel. Like language, sequential image comprehension is characterized by two processing streams. First, readers must integrate semantic information across images and update a mental model of the unfolding scene or story (Magliano and Zacks 2011; Zwaan and Radvansky 1998). Second, a “narrative grammar” organizes and constrains this semantic information with categorical roles embedded in hierarchic constituent structures, similar to the syntactic structure of sentences (Cohn 2013; Cohn et al. 2012). According to the theory of visual narrative grammar (Cohn 2013, 2019), these categorical roles include Establishers (panels that set up an interaction or event), Initials (panels which initiate the event in the narrative arc), Peaks (panels which depict the height or climax of the narrative arc), and Releases (panels which resolve the event or interaction). While these categories are similar to traditional notions of narrative (e.g., set up, rising action, climax, resolution), such roles are not

descriptors of meaning itself but are determined through interactions between the bottom-up semantic content in images and the top-down sequential context (Cohn 2013, 2014). That is, the same panel can serve as a peak or an initial depending on its placement in the visual narrative sequence and its role in the narrative. Just as meaningful information in a sentence can be independent of its syntax, meaningful information in a story is independent from the narrative structure ordering that meaning (e.g., Brewer 1985; Cohn 2013). Thus, this narrative grammar is separate from semantics, just as syntactic structure is separate from – yet interfaced with – meaning in sentences (Cohn et al. 2012).

Visual narrative comprehension is commonly assessed behaviorally by asking participants to arrange randomly presented pictures in a temporal order to construct a coherent story. The produced story is scored as either “correct,” if the pictures are ordered in the way expected by the test designers, or “incorrect,” if the pictures are placed in a different order. Such tasks are included in many intelligence tests such as the Wechsler intelligence scales, including the Wechsler Intelligence Scale for Children (WISC; Wechsler 2014) and the Wechsler Adult Intelligence Scale (WAIS; Wechsler 2008), which both include a picture arrangement subsection.

Comprehension can also be assessed using neuroimaging techniques such as electroencephalography (EEG), which measures the electrical activity of the brain via electrodes placed on the scalp. Brain responses that are time-locked to the onset of a specific stimulus are known as event-related potentials (ERPs) and reflect various stages of cognitive processing of that stimulus. The *N400* component of the ERP, a negative-going deflection peaking approximately 400 milliseconds (ms) after stimulus presentation, has been established as an index of semantic processing (Kutas and Federmeier 2011). In typically developing individuals, *N400* amplitude is reduced for semantically congruent stimuli (e.g., contextually congruent words or comic panels at the end of a narrative) relative to semantically incongruent stimuli (e.g., contextually incongruent words or

comic panels; Kutas and Federmeier 2011; Kutas and Hillyard 1980; Lau et al. 2008). The amplitude difference between congruent and incongruent conditions is often referred to as the *N400* effect and is taken to reflect semantic understanding, with smaller *N400* effects representative of poorer comprehension.

While it is well-documented that individuals with ASD show impairments in understanding linguistic narratives (Braeutigam et al. 2008; Dunn and Bates 2005; Dunn et al. 1999; Jolliffe and Baron-Cohen 2000; Kaland et al. 2007; Mason et al. 2008; McCleery et al. 2010; Nuske and Bavin 2011; Pijnacker et al. 2010; Strandburg et al. 1993), fewer studies have investigated comprehension of visual narratives. Those that have suggest that narrative comprehension impairments persist even for nonlinguistic modalities. For instance, children with ASD showed impairments in picture arrangement for stories requiring ToM (Baron-Cohen et al. 1986). Children with ASD have also shown deficits compared to TD children in inferring missing panels from visual event sequences (Davis et al. 2007) and predicting the final image of event sequences (Zalla et al. 2010). Electrophysiological studies have also documented impairments in visual narrative comprehension in individuals with ASD, with this population showing smaller *N400* effects compared to typically developing individuals for incongruent panels at the end of a visual narrative sequence (Coderre et al. 2018).

It is commonly assumed in the autism field that including visual supports when reading will alleviate processing difficulties, as they play to the “visual strengths” of these individuals (Coderre 2019). However, the emerging evidence documenting impairments in visual narrative comprehension in individuals with ASD may point to domain-general impairments in narrative comprehension that persist even for visual modalities. Visual narrative comprehension relies on many of the same domain-general cognitive skills as linguistic narrative comprehension, such as executive control, working memory, theory of mind, and inference generation (Bower and Morrow 1990; Cohn 2014; Cohn and Kutas

2015; Cohn and Wittenberg 2016; Gernsbacher 1997; Linderholm et al. 2004; Magliano et al. 2016; McCloud 1993). Because visual and linguistic narrative comprehension draw on similar cognitive skills, impairments in these underlying skills may translate into impaired comprehension for both visual and linguistic modalities. Further work is needed to fully understand the factors that play into narrative comprehension impairments in individuals with ASD.

See Also

- Academic Skills
- Literacy
- Narrative Assessment
- Narrative Language in ASD

References and Reading

- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1986). Mechanical, behavioural and intentional understanding of picture stories in autistic children. *British Journal of Developmental Psychology*, 4(2), 113–125.
- Bower, G. H., & Morrow, D. G. (1990). Mental models in narrative comprehension. *Science*, 247(4938), 44–48.
- Braeutigam, S., Swithenby, S. J., & Bailey, A. J. (2008). Contextual integration the unusual way: A magnetoencephalographic study of responses to semantic violation in individuals with autism spectrum disorders. *European Journal of Neuroscience*, 27, 1026–1036.
- Brewer, W. F. (1985). The story schema: Universal and culture-specific properties. In D. R. Olson, N. Torrance, & A. Hildyard (Eds.), *Literacy language and learning* (pp. 167–194). Cambridge: Cambridge University Press.
- Coderre, E. L. (2019). Dismantling the “visual ease assumption”: A review of visual narrative processing in clinical populations. *Topics in Cognitive Science*. <https://doi.org/10.1111/tops.12446>
- Coderre, E. L., Cohn, N., Slipper, S. K., Chemenok, M., Ledoux, K., & Gordon, B. (2018). Visual and linguistic narrative comprehension in autism spectrum disorders: Neural evidence for modality-independent impairments. *Brain and Language*, 186, 44–59.
- Cohn, N. (2013). Visual narrative structure. *Cognitive Science*, 37(3), 413–452.
- Cohn, N. (2014). You’re a good structure, Charlie Brown: The distribution of narrative categories in comic strips. *Cognitive Science*, 38(7), 1317–1359.
- Cohn, N. (2019). Your brain on comics: A cognitive model of visual narrative comprehension. *Topics in Cognitive Science*, 1–35. <https://doi.org/10.1111/tops.12421>
- Cohn, N., & Kutas, M. (2015). Getting a cue before getting a clue: Event-related potentials to inference in visual narrative comprehension. *Neuropsychologia*, 77, 267–278.
- Cohn, N., & Wittenberg, E. (2016). Action starring narratives and events: Structure and inference in visual narrative comprehension. *Journal of Cognitive Psychology*, 27(7), 812–828.
- Cohn, N., Paczynski, M., Jackendoff, R., Holcomb, P. J., & Kuperberg, G. R. (2012). (Pea)nuts and bolts of visual narrative: Structure and meaning in sequential image comprehension. *Cognitive Psychology*, 65, 1–38.
- Davis, M., Dautenhahn, K., Nehaniv, C. L., & Powell, S. D. (2007). The narrative construction of our (social) world: Steps towards an interactive learning environment for children with autism. *Universal Access in the Information Society*, 6(2), 145–157.
- Dunn, M. A., & Bates, J. C. (2005). Developmental change in neutral processing of words by children with autism. *Journal of Autism and Developmental Disorders*, 35(3), 361–376.
- Dunn, M. A., Gaughan, H., Jr., Kreuzer, J., & Kurtzberg, D. (1999). Electrophysiologic correlates of semantic classification in autistic and normal children. *Developmental Neuropsychology*, 16(1), 79–99.
- Gernsbacher, M. A. (1997). Two decades of structure building. *Discourse Processes*, 23(3), 265–304.
- Jolliffe, T., & Baron-Cohen, S. (2000). Linguistic processing in high-functioning adults with autism or Asperger’s syndrome. Is global coherence impaired? *Psychological Medicine*, 30, 1169–1187.
- Kaland, N., Smith, L., & Mortensen, E. L. (2007). Response times of children and adolescents with Asperger syndrome on an “advanced” test of theory of mind. *Journal of Autism and Developmental Disorders*, 37(2), 197–209.
- Kutas, M., & Federmeier, K. D. (2011). Thirty years and counting: Finding meaning in the N400 component of the event-related brain potential (ERP). *Annual Review of Psychology*, 62, 621–647.
- Kutas, M., & Hillyard, S. (1980). Reading senseless sentences: Brain potentials reflect semantic incongruity. *Science*, 207(4427), 203–205.
- Lau, E. F., Phillips, C., & Poeppel, D. (2008). A cortical network for semantics: (De)constructing the N400. *Nature Reviews Neuroscience*, 9(12), 920–933.
- Linderholm, T., Gernsbacher, M. A., van den Broek, P., Neninde, L., Robertson, R. R. W., & Sundermier, B. (2004). Suppression of story character goals during reading. *Discourse Processes*, 37(1), 67–78.
- Magliano, J. P., & Zacks, J. M. (2011). The impact of continuity editing in narrative film on event segmentation. *Cognitive Science*, 35(8), 1489–1517.
- Magliano, J. P., Larson, A. M., Higgs, K., & Loschky, L. C. (2016). The relative roles of visuospatial and linguistic

working memory systems in generating inferences during visual narrative comprehension. *Memory & Cognition*, 44, 207–219.

- Mason, R., Williams, D., Kana, R., Minshew, N. J., & Just, M. A. (2008). Theory of mind disruption and recruitment of the right hemisphere during narrative comprehension in autism. *Neuropsychologia*, 46(1), 269–280.
- McCleery, J. P., Ceponiene, R., Burner, K. M., Townsend, J., Kinnear, M., & Schreibman, L. (2010). Neural correlates of verbal and nonverbal semantic integration in children with autism spectrum disorders. *Journal of Child Psychology and Psychiatry*, 51(3), 277–286.
- McCloud, S. (1993). *Understanding comics: The invisible art*. New York: Harper Collins.
- Nuske, H. J., & Bavin, E. L. (2011). Narrative comprehension in 4-7-year-old children with autism: Testing the Weak Central Coherence account. *International Journal of Language & Communication Disorders*, 46(1), 108–119.
- Pijnacker, J., Geurts, B., van Lambalgen, M., Buitelaar, J., & Hagoort, P. (2010). Exceptions and anomalies: An ERP study on context sensitivity in autism. *Neuropsychologia*, 48(10), 2940–2951.
- Strandburg, R. J., Marsh, J. T., Brown, W. S., Asarnow, R. F., Guthrie, D., & Higa, J. (1993). Event-related potentials in high-functioning adult autistics: Linguistic and nonlinguistic visual information processing tasks. *Neuropsychologia*, 31(5), 413–434.
- Wechsler, D. (2008). *Wechsler Adult Intelligence Scale – Fourth edition (WAIS-IV)*. London, UK: Pearson Education.
- Wechsler, D. (2014). *Wechsler Intelligence Scale for Children-fifth edition (WISC-V): Administration and scoring manual*. London, UK: Pearson Education.
- Zalla, T., Labryère, N., Clément, A., & Georgieff, N. (2010). Predicting ensuing actions in children and adolescents with autism spectrum disorders. *Experimental Brain Research*, 201, 809–819.
- Zwaan, R., & Radvansky, G. (1998). Situation models in language comprehension and memory. *Psychological Bulletin*, 123(2), 162–185.

Visual Narrative Sequences

► Visual Narrative Comprehension

Visual Orienting to Eyes

► Mutual Gaze

Visual Processing

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Definition

Visual processing in autism: the lows and highs

Historical Background

Atypical behavioral performance, at multiple levels of visual processing, has been observed in individuals with autism, and unsurprisingly, changes to the neural mechanisms subserving these visual behaviors have often been documented, as well. Perhaps, most intriguing is the finding that, while some visual behaviors appear to be compromised in individuals with autism, relative to typical individuals, others appear to be superior relative to their typical counterparts, and *enhanced perceptual functioning* (Motttron et al. 2006) is increasingly considered part of the autism phenotype. Here, an overview of visual processing in autism from lower through mid- to higher levels of processing is presented. Recent findings concerning the psychological and neural mechanisms mediating these atypicalities as well as some ongoing controversies and debates are summarized; for example, the review considers whether atypicalities in earlier levels potentially have a snowball or cascaded effect such that the observed differences in the computation and representation of visual information in higher-order levels in the autism population might arise from these lower-level perturbations, and there is a call for additional investigations to address the outstanding issues.

It is important to note that alterations in visual processing in autism are many and varied, and reviewing all of these is well beyond the scope of this summary. The focus, then, is mostly restricted to examining the functions of the

cortical ventral visual pathway (although dorsal cortical function will be briefly considered) in individuals with autism. Thus, there is no discussion of optometric functions in autism although there is a high prevalence of strabismus, refractive error, altered ocular alignment, and difficulty with voluntary pursuit eye movements, among other visual symptoms; for a recent short review, see Simmons et al. (2009). It is also the case that there are cognitive or higher-order visual functions that are adversely affected in autism, including, among others, atypical saccadic behavior, alterations in attentional function, unusual socially directed pointing, difficulties with interpretation of gestures, unusual eye contact, difficulty with the interpretation of facial expressions, imitation, and difficulty following the gaze of others and engaging joint attention. This review does not elaborate on these higher-order behaviors in this section although some of these symptoms are critical to the phenotype of autism and are included in the Autism Diagnostic Observation Schedule (ADOS) (Lord et al. 2000).

Current Knowledge

Lower-Level Vision: Striate and Extrastriate Cortex

Many studies of lower-level vision in autism have concluded that elementary visuo-perceptual skills are, at least, preserved or possibly even enhanced in individuals with autism (Ashwin et al. 2009; Mottron et al. 2006, 2009; Soulières et al. 2009), relative to typical controls. Recent studies have demonstrated sensory hypersensitivity with better detection thresholds in vision in individuals with autism, compared with their typical counterparts (Baron-Cohen et al. 2009), and have documented excellent attention to detail in these same individuals (for more on this topic, also see later sections), although the latter may be a direct product of the sensory hypersensitivity per se (Joseph et al. 2009; Mottron and Burack 2001). Of interest, a recent report by Ashwin et al. (2009) described significantly better visual acuity (20:7) in adults with autism compared with control subjects (20:13), an acuity so superior that it lies in

the range reported for birds of prey (although note that this may not hold generically; Keita et al. 2010). However, previous studies with children with ASDs found unexceptional acuity and, indeed, reported that many of the children actually exhibited inferior acuity (Emigh and Procyk 1992). Further investigation into the acuity characteristics of individuals with autism is clearly necessary. See also (Dakin and Frith 2005; Simmons et al. 2009) for further discussion of early sensory processing skills in autism. Support for the reported enhancement of early perception in autism is also gleaned from experiments which show that whereas typical controls' peripheral target perception is adversely affected by the presence of flankers (stimuli in the spatial surround), individuals with autism may have enhanced focus and, thus, be relatively immune to this crowding effect (Baldassi et al. 2009). Finally, neuroimaging studies of adults with autism suggest that the retinotopic organization in early visual cortex may be comparable to that of typical adults, with typical topographic representation of foveal and peripheral fields in early visual cortex (Hadjikhani et al. 2004a).

One fairly consistent finding of atypical lower-level visual processing in autism concerns the extraction or representation of fundamental aspects of incoming percepts. For example, atypicalities in spatial frequency processing in autism have been documented in several recent studies. One such study compared early visual evoked potentials (VEPs) elicited to sine-wave gratings of different spatial frequencies (Jemel et al. 2010) in autism and control participants. Whereas VEP contrast responses to low- and high-spatial-frequency gratings did not differ between the groups, in the autism group, early VEPs to mid-spatial-frequency gratings were similar to those obtained in response to high-spatial-frequency gratings. These data indicate an alteration in the functional segregation or selectivity of early visual channels, especially those responsible for processing mid- and high-frequency spatial scales in autism. Alterations in VEPs elicited by Gabor patches of varying spatial frequency in individuals with autism have also been documented by Milne et al. (2009). They found that the onset of

even a simple visual stimulus perturbs the activity of a widespread network including sensory, parietal, and limbic areas and that the observed reduction in spectral power across multiple regions may reflect a reduction in the synchrony and size of the neural networks recruited during perceptual processing.

This reported alteration in neural integration may also account for the relative difficulty these individuals experience in identifying complex texture-defined gratings (Bertone et al. 2003, 2005). Whereas these individuals are more accurate at detecting the orientation of first-order gratings (simple, luminance-defined) than controls, they are less accurate at identifying the second-order gratings. Bertone et al. (2005) hypothesize that because simple visual stimuli can be processed in a single brain area (typically V1), perception of first-order gratings may be unaffected. Complex stimuli, on the other hand, are processed in multiple brain areas, and the reduced efficiency of neurointegrative mechanisms in autism (see below for further discussion of the topic of connectivity) and perhaps weak central coherence (Shah and Frith 1993) may explain the reduced ability to perceive more complex stimuli.

Consistent with this hypothesis, VEPs recorded while autism participants observed orientation-based texture and contour stimuli forming coherent global patterns revealed the absence of certain harmonics, characteristic of global form processing, and even when harmonics could be elicited in the autism group, the amount of organization required to do so was higher than in the nonautism control group (Pei et al. 2009; Vandenbroucke et al. 2008). Together, these findings indicate that early aspects of high spatial frequency and form extraction are atypical in adults with autism, especially as stimuli become more complex and multiple brain regions are required.

As alluded to above, these findings of atypical lower-level visual processing can be accommodated by a view that argues that vision in autism is mediated by a system with increased local connectivity and a dysfunction of long-range intracortical connections, which are necessary for integrative higher-order visual processes.

Specifically, increased local connectivity, particularly in posterior, sensory parts of the cerebral cortex, may account for the unusual sensory “magnification” strengths and enhanced perception of local features as well as the increased focus (and immunity to distracters) observed in some cases of autism (Belmonte et al. 2004). At the same time, there is reduced neurointegration and so perception of stimuli that are complex or necessarily engage a range of cortical regions is adversely affected.

The altered local lateral connectivity in autism might originate from an imbalance in excitatory/inhibitory neural signaling, resulting in changes regarding elementary feature extraction. Predictably, then, as outlined by accounts of enhanced perceptual functioning, there is increased physiological engagement of occipital cortex and occipitoparietal areas typically involved in primary perceptual functions, coupled with reduced frontal activity (Mottron et al. 2006; Samson et al. 2011) that may explain the autistic perceptual endophenotype (Mottron et al. 2009, 2006).

Mid-level Vision: Ventral Occipitotemporal Cortex

Perceptual Grouping

In addition to the atypicalities observed in early parts of the visual system, there are also atypicalities noted in processes requiring perceptual organization. For example, individuals with autism are able to identify fine stimulus features in spatial tasks like block design (Jolliffe and Baron-Cohen 1997; Shah and Frith 1983) and are notoriously faster and more accurate at finding embedded information, such as in the Embedded Figures Task (Happé and Frith 2006). This profile is also consistent with the notion that brain activity in participants with autism during such tasks is higher bilaterally in occipital cortex and in the cerebellum, as well as in the left posterior parietal cortex, compared to typical controls, but it is lower in frontal cortex (Lee et al. 2007; Manjaly et al. 2007; Ring et al. 1999). Interestingly, even when visual behavioral performance is equated across the control and autism group in an embedded figures-type task, the autism individuals

continue to show heightened activation in the posterior parietal cortex and occipital areas and less activation in the left dorsolateral prefrontal and inferior parietal areas. Furthermore, there is decreased functional connectivity between higher-order working memory/executive areas and visuospatial regions in the autism participants (Damarla et al. 2010; Schipul et al. 2011), reflecting the difficulty with long-range connectivity and neural integration discussed above (for a similar recent result, see Liu et al. 2011).

Several studies have reported superior abilities to detect local targets in visual search tasks in autism (Joseph et al. 2009; Plaisted et al. 1999), which is associated with higher occipital cortex activity (Keehn et al. 2008). Individuals with autism are also better able to ignore the influence of increasing numbers of distracters during visual search (O’Riordan and Plaisted 2001), and they outperform controls in change blindness tasks, detecting more errors/changes in visual scenes (Smith and Milne 2009; see also discussion of crowding phenomenon above). These superior visual search skills may come at a cost, however. Individuals with autism are apparently limited in their ability to derive organized wholes from perceptual parts, which is critical for many aspects of higher-order visual processing such as object recognition. This integration difficulty has been linked to limited use of gestalt grouping heuristics (Brosnan et al. 2004; Farran and Brosnan 2011), the failure to process interelement relationships (Behrmann et al. 2006; for similar results in children and adolescents with autism, see Scherf et al. 2008), and/or the failure to consider the entire visual context (Happé and Frith 2006; Liu et al. 2011). Yet others have suggested that the problem lies in limited abilities to integrate local elements specifically into a coherent global shape (Plaisted et al. 1999; Rinehart et al. 2000; Wang et al. 2007). Note, however, that there are some studies that report typical processing of both the global and local information in hierarchical visual stimuli (Iarocci et al. 2006; Mottron et al. 2003; Ozonoff et al. 1994; Plaisted et al. 2006). Similarly, a recent study reports increased sensitivity to mirror symmetry in autism, again indicative of parallel access to both local and global

information and the possibility that individuals with autism may extract from noisy environments genuine regularities, which elude nonautistic observers (Perreault et al. 2011). Thus, whether there is undue reliance on local elements and/or difficulty in achieving a global percept (or possibly neither) in autism continues to be a source of much debate in the field. Whether these different profiles are indicative of heterogeneity in the population per se or whether the different results emerge from the adoption of different paradigms and analyses remains to be determined.

Motion Perception

As with perceptual grouping, detecting coherent motion involves the integration of multiple local elements, and there is also significant controversy about whether observers with ASD differ from typical observers in their visual analyses of movement. Some initial studies suggested that observers with autism experience marked deficits in their visual sensitivity to coherent motion in random dot displays but not to point-light displays of human motion (Milne et al. 2002; Spencer et al. 2000). More recent evidence suggests exactly the opposite: that observers with autism do not differ from typical observers in their visual sensitivity to coherent motion in random dot displays but do differ from typical observers in their visual sensitivity to human motion (for review of this literature, see Kaiser and Shiffrar 2009). For example, one recent study that directly compared human motion perception versus object motion (tractor motion) revealed that whereas typical observers are more sensitive to the former than the latter, observers with autism showed equivalent levels of sensitivity to both (Kaiser et al. 2010). The explanation offered for this latter result is that the visual systems of individuals with autism may not be tuned for the detection of socially relevant information such as the presence of another person. An explanation offered for some of these findings is that autism observers are likely able to detect coherent movement (Atkinson 2009; Blake et al. 2003) but that, during development, the posterior superior temporal sulcus might not be increasingly tuned to differentiate human motion from object motion in autism (Pelphrey and Carter 2008).

In sum, the studies examining mid-level vision in autism usually reveal atypicalities in perceptual grouping and in motion perception, although the particular profile of the findings is not perfectly consistent across every study. Taken together, however, the weight of the evidence favors atypicalities in mid-level vision in autism, and these atypicalities appear to hold across children, adolescents, and adults with autism. It has been suggested that, from a developmental perspective, the atypical emergence of these perceptual organizational processes may contribute directly to disruptions in the processing of visually presented objects, which may, in turn, fundamentally influence the development of major aspects of the social and emotional deficits characteristic of autism (for recent discussion of social deficit and complexity of this deficit; see New et al. 2010). Additionally, the reduction of visual sensitivity to human movements could compromise important social behaviors including, for example, gesture comprehension, and in this way, foundational visual processes could adversely impact not only higher-order visual processes but also could have direct impact on social behaviors, too.

Higher-Level Vision: Beyond the Calcarine Sulcus

Just as atypicalities in early sensory vision in autism impact mid-level grouping and integration abilities, so too do atypicalities in mid-level vision have direct consequences for higher-level visual skills. Moreover, this cascade may disproportionately affect some types of stimuli, such as faces, in which the input similarity is especially high and the featural differences themselves do not suffice to support differentiation of individual exemplars (Boucher and Lewis 1992; Davies et al. 1994; Hobson et al. 1988; Joseph and Tanaka 2003; Klin et al. 2002; Lahaie et al. 2006). Given that all faces have two eyes, a nose, and a mouth and that these all share the same first-order configuration (eyes above nose above mouth), it has been argued that faces, to a greater degree than any other object type, rely on the ability to derive a second-order or holistic representation, the very ability that seems unduly taxed in autism.

To evaluate the relation between the local visual processing bias and impaired face and object processing skills in autism, Behrmann et al. (2006) tested adults with autism, whom we already knew evinced a local bias across two very different perceptual organization tasks, in several same/different judgments tasks with faces, common and novel objects. Not only were the adults with autism slower than typical adults when making the same/different similarity judgments, but more relevant for the current discussion, they were disproportionately slower in their similarity judgments as the stimuli became increasingly similar. These results reveal the disproportionate difficulty in the more demanding tasks that require perceptual binding of the elemental features, and it is in these cases that featural distinctions do not suffice, and integration of the features into higher-order configurations become essential. Additionally, performance in these more taxing face and novel object discrimination tasks was significantly correlated with the local bias on the global/local task. Although correlation is not causation, the relationship between performance in face and object processing and the local bias appears clear in the autistic adult individuals and is highly suggestive of an association between them.

The atypicality in face processing has also been associated, in many studies, with altered neural activation in higher-order regions of visual cortex, most notably the fusiform gyrus. Many studies have found reduced BOLD activation in this region for faces compared with other visual classes (for a few examples, see Corbett et al. 2009; Critchley et al. 2000; Dalton et al. 2005; Hubl et al. 2003; Pierce et al. 2001; Schultz et al. 2000; Wang et al. 2004) although a few studies have failed to replicate this finding (Hadjikhani et al. 2004a, b; Pierce et al. 2004). The analysis of the cortical substrate of face perception has focused on the preeminent FFA but also on a more widely distributed network of brain areas involved in face processing, and, at least in one study, hypoactivation has been identified more broadly in regions including the right amygdala, inferior frontal cortex, superior temporal sulcus,

and face-related somatosensory and premotor cortex (Hadjikhani et al. 2007).

In another study using naturalistic, real-time movies of unfamiliar faces, buildings, and navigation through open fields, the cortical activation profile from the typical group largely replicated the standard findings with extensive activation for faces and objects and activation (albeit less extensive) for building/scene-related displays (Humphreys et al. 2008). In contrast, there was a clear reduction in face-related activity throughout the ventral visual pathway in the autism group. Interestingly, and consistent with those studies showing enhancement in object-related activation (Pierce et al. 2004), there was more extensive object-related activity in lateral occipital cortex for the autism than comparison group bilaterally.

Adolescents with autism, under the same experimental conditions, failed to show consistent face-selective activation in classical face regions, revealing that the alteration in functional topography of face-related cortex in autism is present early on. Additionally, for those few adolescents with autism who did exhibit face-selective activation, face-selective cortex was located in traditionally object-related regions, supporting the hypothesis that perceptual processing of faces in autism may be more akin to the perceptual processing of common objects in typically developing individuals. Such alterations could result from direct pathology to regions within the face-processing network, like the fusiform gyrus (Hadjikhani et al. 2007), and/or to the structural and functional connections between such regions (Thomas et al. 2011). Also, alterations in the visual experiences that individuals with autism have with faces as a result of social aversion and/or excessive focus on features may configure these regions in the face-processing network atypically (Grelotti et al. 2005; Hadjikhani et al. 2004b).

Taken together, there is substantial behavioral and neuroimaging evidence to suggest that individuals with autism exhibit heightened sensitivity to elemental features in visual objects, which may interfere with their ability to perceptual

group these elements to perceive the holistic gestalt of the image. This profile of atypical visual processing may be especially detrimental to face processing, which relies heavily on holistic processing or relational representation of elemental features. In contrast, it may boost processing of common objects, which can be easily discriminated based on differences in elemental features.

Finally, a recent meta-analysis based on 26 published functional magnetic resonance imaging (fMRI) studies of face, object, and word visual processing in adults with autism has indicated that individuals with autism exhibit generally stronger engagement of visual processing regions, including striate and extrastriate regions, and parietal regions, including the precuneus and intraparietal sulcus, than controls (Samson et al. 2011). More controversially, this review states that these results do not support the claim that there is *hypoactivation* in face-selective regions in autism although, consistent with previous studies (e.g., Scherf et al. 2010), they do suggest that the locus of fusiform face-selective activation may be atypical in autism and located in regions that are object-selective in controls. Suffice it to say, much remains to be done to characterize the cortical profile in individuals with autism engaged in high-level visual processing tasks, and the correspondence between the brain activity and behavior remains to be clarified, too.

Future Directions

Conclusions

The unusual and varied visual processing skills in autism reviewed here span early to late stages of the visual processing stream. Disparities between individuals with autism and typical individuals are observed in tasks engaging lower-level visual features, and atypicalities are observed under conditions engaging mid- and higher-level visual stages, too. This widespread set of altered visual behaviors and their neural correlate is not easily accounted for by views that argue for a primary social deficit in autism (Scherf et al. 2008), and

thus, there is increasing recognition of the rather pervasive perceptual deficit, too. For example, Batty et al. (2011) showed that, when verbal age was matched across a group of children with autism and typical controls, their ERP profiles, recorded during an implicit emotional recognition task, differed only in the amplitude of the early P1 component. This led the authors to suggest that the emotional and facial processing difficulties in autism could start from atypicalities in visual perceptual processes involving rapid feedback to primary visual areas and have subsequent impact on holistic processing. Whether the social and perceptual deficits are independent or interdependent remains to be determined. While some have suggested that the atypical perceptual processing in children with autism is a secondary effect of profound social-communicative deficits that alter the nature of the child's early environmental inputs and disturb the normal course of brain growth and organization, the perceptual difficulties, themselves, may contribute to the alterations in social behavior.

As abundantly evident, many key issues remain to be addressed including broad questions such as what neurobiological events trigger the unusual psychological and neural processes that underlie the altered perceptual profile in autism. More specific questions such as the integrity of particular brain areas, such as the fusiform face area in autism, also remain to be determined. Characterizing the atypical visual behaviors in fine-grained detail and their underlying neural correlates is critical, too, and although individuals with autism might evince some enhanced visual skills, these are not necessarily advantageous, and the highs and lows of their visual experience remain to be fully characterized. Finally, understanding the impact of these atypical skills on daily living and the potential for intervention remain at the top of the list of issues to be addressed.

See Also

► Functional MRI

References and Reading

- Ashwin, E., Ashwin, C., Rhydderch, D., Howells, J., & Baron-Cohen, S. (2009). Eagle-eyed visual acuity: An experimental investigation of enhanced perception in autism. *Biological Psychiatry*, 65(1), 17–21.
- Atkinson, A. P. (2009). Impaired recognition of emotions from body movements is associated with elevated motion coherence thresholds in autism spectrum disorders. *Neuropsychologia*, 47(13), 3023–3029.
- Baldassi, S., Pei, F., Megna, N., Recupero, G., Viespoli, M., Igliozi, R., et al. (2009). Search superiority in autism within, but not outside the crowding regime. *Vision Research*, 49(16), 2151–2156.
- Baron-Cohen, S., Ashwin, E., Ashwin, C., Tavassoli, T., & Chakrabarti, B. (2009). Talent in autism: Hyper-systemizing, hyper-attention to detail and sensory hypersensitivity. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 364(1522), 1377–1383.
- Batty, M., Meaux, E., Wittemeyer, K., Roge, B., & Taylor, M. J. (2011). Early processing of emotional faces in children with autism: An event-related potential study. *Journal of Experimental Child Psychology*, 109(4), 430–444.
- Behrmann, M., Avidan, G., Leonard, G. L., Kimchi, R., Luna, B., Humphreys, K., et al. (2006). Configural processing in autism and its relationship to face processing. *Neuropsychologia*, 44(1), 110–129.
- Belmonte, M. K., Allen, G., Beckel-Mitchener, A., Boulanger, L. M., Carper, R. A., & Webb, S. J. (2004). Autism and abnormal development of brain connectivity. *Journal of Neuroscience*, 24(42), 9228–9231.
- Bertone, A., Mottron, L., Jelenic, P., & Faubert, J. (2003). Motion perception in autism: A “complex” issue. *Journal of Cognitive Neuroscience*, 15(2), 218–225.
- Bertone, A., Mottron, L., Jelenic, P., & Faubert, J. (2005). Enhanced and diminished visuo-spatial information processing in autism depends on stimulus complexity. *Brain*, 128, 2430–2441.
- Bird, G., Catmur, C., Silani, G., Frith, C., & Frith, U. (2006). Attention does not modulate neural responses to social stimuli in autism spectrum disorders. *Neuroimage*, 31(4), 1614–1624.
- Blake, R., Turner, L. M., Smoski, M. J., Pozdol, S. L., & Stone, W. L. (2003). Visual recognition of biological motion is impaired in children with autism. *Psychological Science*, 14(2), 150–157.
- Boucher, J., & Lewis, V. (1992). Unfamiliar face recognition in relatively able autistic children. *Journal of Child Psychology and Psychiatry*, 33(5), 843–859.
- Brosnan, M. J., Scott, F. J., Fox, S., & Pye, J. (2004). Gestalt processing in autism: Failure to process perceptual relationships and the implications for contextual understanding. *Journal of Child Psychology and Psychiatry*, 45(3), 459–469.
- Corbett, B. A., Carmean, V., Ravizza, S., Wendelken, C., Henry, M. L., Carter, C., et al. (2009). A functional and structural study of emotion and face processing in

- children with autism. *Psychiatry Research*, 173(3), 196–205.
- Critchley, H. D., Daly, E. M., Bullmore, E. T., Williams, S. C. R., Amelsvoort, T. V., Robertson, D. M., et al. (2000). The functional neuroanatomy of social behaviour: Changes in cerebral blood flow when people with autistic disorder process facial expressions. *Brain*, 123, 2203–2212.
- Dakin, S., & Frith, U. (2005). Vagaries of visual perception in autism. *Neuron*, 48(3), 497–507.
- Dalton, K. M., Nacewicz, B. M., Johnstone, T., Schaefer, H. S., Gernsbacher, M. A., Goldsmith, H. H., et al. (2005). Gaze fixation and the neural circuitry of face processing in autism. *Nature Neuroscience*, 8(4), 519–526.
- Damarla, S. R., Keller, T. A., Kana, R. K., Cherkassky, V. L., Williams, D. L., Minshew, N. J., et al. (2010). Cortical underconnectivity coupled with preserved visuospatial cognition in autism: Evidence from an fMRI study of an embedded figures task. *Autism Research*, 3(5), 273–279. <https://doi.org/10.1002/aur.153>.
- Davies, S., Bishop, D., Manstead, A. S. R., & Tatum, D. (1994). Face perception in children with autism and Asperger's syndrome. *Journal of Child Psychology and Psychiatry*, 35(6), 1033–1057.
- Emigh, S. J., & Procyk, C. M. (1992). Assessment of visual function in autistic children. *Optometry and Vision Science*, 69(6), 433–439.
- Farran, E. K., & Brosnan, M. J. (2011). Perceptual grouping abilities in individuals with autism spectrum disorder; exploring patterns of ability in relation to grouping type and levels of development. *Autism Research*, 4, 1–10.
- Grelotti, D. J., Klin, A. J., Gauthier, I., Skudlarski, P., Cohen, D. J., Gore, J. C., et al. (2005). fMRI activation of the fusiform gyrus and amygdala to cartoon characters but not to faces in a boy with autism. *Neuropsychologia*, 43(3), 373–385.
- Hadjikhani, N., Chabris, C. F., Joseph, R. M., Clark, J., McGrath, L., Aharon, I., et al. (2004a). Early visual cortex organization in autism: An fMRI study. *Neuroreport*, 15(2), 267–270.
- Hadjikhani, N., Joseph, R. M., Snyder, J., Chabris, C. F., Clark, J., Steele, S., et al. (2004b). Activation of the fusiform gyrus when individuals with autism spectrum disorder view faces. *Neuroimage*, 22(3), 1141–1150.
- Hadjikhani, N., Joseph, R. M., Snyder, J., & Tager-Flusberg, H. (2006). Abnormal activation of the social brain during face perception in autism. *Human Brain Mapping*, 28, 441–449.
- Hadjikhani, N., Joseph, R. M., Snyder, J., & Tager-Flusberg, H. (2007). Abnormal activation of the social brain during face perception in autism. *Human Brain Mapping*, 28(5), 441–449.
- Happé, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36(1), 5–25.
- Hobson, R. P., Ouston, J., & Lee, A. (1988). What's in a face? The case of autism. *British Journal of Psychology*, 79(Pt 4), 441–453.
- Hubl, D., Bolte, S., Feineis-Matthews, S., Lanfermann, H., Federspiel, A., Strik, W., et al. (2003). Functional imbalance of visual pathways indicates alternative face processing strategies in autism. *Neurology*, 61(9), 1232–1237.
- Humphreys, K., Hasson, U., Avidan, G., Minshew, N., & Behrmann, M. (2008). Cortical patterns of category-selective activation for faces, places and objects in adults with autism. *Autism Research*, 1(1), 52–63.
- Iarocci, G., Burack, J. A., Shore, D. I., Mottron, L., & Enns, J. T. (2006). Global-Local visual processing in high functioning children with autism: Structural vs. implicit task biases. *Journal of Autism and Developmental Disorders*, 36, 1–13.
- Jemel, B., Mimeault, D., Saint-Amour, D., Hosen, A., & Mottron, L. (2010). VEP contrast sensitivity responses reveal reduced functional segregation of mid and high filters of visual channels in autism. *Journal of Vision*, 10(6), 13.
- Jolliffe, T., & Baron-Cohen, S. (1997). Are people with autism and Asperger syndrome faster than normal on the embedded figures test? *Journal of Child Psychology and Psychiatry*, 38(5), 527–534.
- Joseph, R. M., & Tanaka, J. (2003). Holistic and part-based face recognition in children with autism. *Journal of Child Psychology and Psychiatry*, 44(4), 529–542.
- Joseph, R. M., Keehn, B., Connolly, C., Wolfe, J. M., & Horowitz, T. S. (2009). Why is visual search superior in autism spectrum disorders? *Developmental Science*, 12(6), 1083–1096.
- Kaiser, M. D., & Shiffrar, M. (2009). The visual perception of motion by observers with autism spectrum disorders: A review and synthesis. *Psychonomic Bulletin and Review*, 16(5), 761–777.
- Kaiser, M. D., Delmolino, L., Tanaka, J. W., & Shiffrar, M. (2010). Comparison of visual sensitivity to human and object motion in autism spectrum disorder. *Autism Research*, 3, 1–5.
- Keehn, B., Brenner, L., Palmer, E., Lincoln, A. J., & Muller, R. A. (2008). Functional brain organization for visual search in ASD. *Journal of International Neuropsychological Society*, 14(6), 990–1003.
- Keita, L., Mottron, L., & Bertone, A. (2010). Far visual acuity is unremarkable in autism: Do we need to focus on crowding? *Autism Research*, 3(6), 333–341. <https://doi.org/10.1002/aur.164>.
- Kimchi, R. (1998). Uniform connectedness and grouping in the perceptual organization of hierarchical patterns. *Journal of Experimental Psychology: Human Perception and Performance*, 24(2), 1105–1118.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of

- naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809–816.
- Lahaie, A., Mottron, L., Arguin, M., Berthiaume, C., Jemel, B., & Saumier, D. (2006). Face perception in high-functioning autistic adults: Evidence for superior processing of face parts, not for a configural face-processing deficit. *Neuropsychology*, 20(1), 30–41.
- Lee, P. S., Foss-Feig, J., Henderson, J. G., Kenworthy, L. E., Gilotty, L., Gaillard, W. D., et al. (2007). Atypical neural substrates of embedded figures task performance in children with autism spectrum disorder. *Neuroimage*, 38(1), 184–193.
- Liu, Y., Cherkassky, V. L., Minshew, N. J., & Just, M. A. (2011). Autonomy of lower-level perception from global processing in autism: Evidence from brain activation and functional connectivity. *Neuropsychologia*, 49(7), 2105–2111.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Leventhal, B. L., DiLavore, P. C., et al. (2000). The autism diagnostic observation schedule – generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders*, 24, 659–685.
- Manjaly, Z. M., Bruning, N., Neufang, S., Stephan, K. E., Brieber, S., Marshall, J. C., et al. (2007). Neurophysiological correlates of relatively enhanced local visual search in autistic adolescents. *Neuroimage*, 35(1), 283–291.
- Milne, E., Swettenham, J., Hansen, P., Campbell, R., Jeffries, H., & Plaisted, K. (2002). High motion coherence thresholds in children with autism. *Journal of Child Psychology and Psychiatry*, 43(2), 255–263.
- Milne, E., Scope, A., Pascalis, O., Buckley, D., & Makeig, S. (2009). Independent component analysis reveals atypical electroencephalographic activity during visual perception in individuals with autism. *Biological Psychiatry*, 65(1), 22–30.
- Mottron, L., & Burack, J. A. (2001). *Enhanced perceptual functioning in the development of autism*. Mahwah: Erlbaum.
- Mottron, L., Burack, J. A., Iarocci, G., Belleville, S., & Enns, J. T. (2003). Locally oriented perception with intact global processing among adolescents with high-functioning autism: Evidence from multiple paradigms. *Journal of Child Psychology and Psychiatry*, 44(6), 904–913.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36(1), 27–43.
- Mottron, L., Dawson, M., & Soulières, I. (2009). Enhanced perception in savant syndrome: Patterns, structure and creativity. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 364(1522), 1385–1391.
- Navon, D. (1977). Forest before trees: The precedence of global features in visual perception. *Cognitive Psychology*, 9, 353–383.
- New, J. J., Schultz, R. T., Wolf, J., Niehaus, J. L., Klin, A., German, T. C., et al. (2010). The scope of social attention deficits in autism: Prioritized orienting to people and animals in static natural scenes. *Neuropsychologia*, 48(1), 51–59.
- O’Riordan, M., & Plaisted, K. C. (2001). Enhanced discrimination in autism. *Quarterly Journal of Experimental Psychology*, 54A(4), 961–979.
- Ogai, M., Matsumoto, H., Suzuki, K., Ozawa, F., Fukuda, R., Uchiyama, I., et al. (2003). fMRI study of recognition of facial expressions in high-functioning autistic patients. *Neuroreport*, 14(4), 559–563.
- Ozonoff, S., Strayer, D. L., McMahon, W. M., & Filloux, F. (1994). Executive function abilities in autism and Tourette syndrome: An information processing approach. *Journal of Child Psychology and Psychiatry*, 35(6), 1015–1032.
- Pei, F., Baldassi, S., Procida, G., Igliozi, R., Tancredi, R., Muratori, F., et al. (2009). Neural correlates of texture and contour integration in children with autism spectrum disorders. *Vision Research*, 49, 2140–2150.
- Pelphrey, K. A., & Carter, E. J. (2008). Brain mechanisms for social perception: Lessons from autism and typical development. *Annals of the New York Academy of Sciences*, 1145, 283–299.
- Perreault, A., Gurnsey, R., Dawson, M., Mottron, L., & Bertone, A. (2011). Increased sensitivity to mirror symmetry in autism. *PLoS One*, 6(4), e19519.
- Pierce, K., Muller, R. A., Ambrose, J., Allen, G., & Courchesne, E. (2001). Face processing occurs outside the fusiform “face area” in autism: Evidence from functional MRI. *Brain*, 124(Pt 10), 2059–2073.
- Pierce, K., Haist, F., Sedaghat, F., & Courchesne, E. (2004). The brain response to personally familiar faces in autism: Findings of fusiform activity and beyond. *Brain*, 127(Pt 12), 2703–2716.
- Plaisted, K., Swettenham, J., & Rees, L. (1999). Children with autism show local precedence in a divided attention task and global precedence in a selective attention task. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 40(5), 733–742.
- Plaisted, K., Dobler, V., Bell, S., & Davis, G. (2006). The microgenesis of global perception in autism. *Journal of Autism and Developmental Disorders*, 36(1), 107–116.
- Rinehart, N., Bradshaw, J., Moss, S., Brereton, A., & Tonge, B. (2000). Atypical interference of local detail on global processing in high-functioning autism and Asperger’s disorders. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 41(6), 769–788.
- Ring, H. A., Baron-Cohen, S., Wheelwright, S., Williams, S. C. R., Brammer, M., Andrew, C., et al. (1999). Cerebral correlates of preserved cognitive skills in autism. *Brain*, 122, 1305–1315.
- Samson, F., Mottron, L., Soulières, I., & Zeffiro, T. A. (2011). Enhanced visual functioning in autism: An ALE meta-analysis. *Human Brain Mapping*. <https://doi.org/10.1002/hbm.21307>.
- Scherf, K. S., Luna, B., Kimchi, R., Minshew, N., & Behrmann, M. (2008). Missing the big picture:

- Impaired development of global shape processing in autism. *Autism Research*, 1(2), 114–129.
- Scherf, K. S., Luna, B., Minshew, N., & Behrmann, M. (2010). Location, location, location: Alterations in the functional topography of face- but not object- or place-related cortex in adolescents with autism. *Frontiers in Human Neuroscience*, 4, 26.
- Schipul, S. E., Keller, T. A., & Just, M. A. (2011). Inter-regional brain communication and its disturbance in autism. *Frontiers in Systems Neuroscience*, 5, 10. <https://doi.org/10.3389/fnsys.2011.00010>.
- Schultz, R. T., Gauthier, I., Klin, A., Fulbright, R. K., Anderson, A. W., Volkmar, F., et al. (2000). Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and Asperger syndrome. *Archives of General Psychiatry*, 57, 331–340.
- Shah, A., & Frith, U. (1983). An islet of ability in autism: A research note. *Journal of Child Psychology and Psychiatry*, 24, 613–620.
- Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry*, 34(8), 1351–1364.
- Simmons, D. R., Robertson, A. E., McKay, L. S., Toal, E., McAleer, P., & Pollick, F. E. (2009). Vision in autism spectrum disorders. *Vision Research*, 49(22), 2705–2739.
- Smith, H., & Milne, E. (2009). Reduced change blindness suggests enhanced attention to detail in individuals with autism. *Journal of Child Psychology and Psychiatry*, 50(3), 300–306.
- Soulieres, I., Dawson, M., Samson, F., Barbeau, E. B., Sahyoun, C. P., Strangman, G. E., et al. (2009). Enhanced visual processing contributes to matrix reasoning in autism. *Human Brain Mapping*, 30(12), 4082–4107.
- Spencer, J., O'Brien, J., Riggs, K., Braddick, O., Atkinson, J., & Wattam-Bell, J. (2000). Motion processing in autism: Evidence for a dorsal stream deficiency. *Neuroreport*, 11(12), 2765–2767.
- Thomas, C., Humphreys, K., Jung, K. J., Minshew, N., & Behrmann, M. (2011). The anatomy of the callosal and visual-association pathways in high-functioning autism: A DTI tractography study. *Cortex; a journal devoted to the study of the nervous system and behavior*, 47(7), 863–873.
- Vandenbroucke, M. W., Scholte, H. S., Engeland, H. V., Lamme, V. A., & Kemner, C. (2008). A neural substrate for atypical low-level visual processing in autism spectrum disorder. *Brain*, 131(4), 1013–1024.
- Wang, A. T., Dapretto, M., Hariri, A. R., Sigman, M., & Bookheimer, S. Y. (2004). Neural correlates of facial affect processing in children and adolescents with autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43(4), 481–490.
- Wang, L., Mottron, L., Peng, D., Berthiaume, C., & Dawson, M. (2007). Local bias and local-to-global interference without global deficit: A robust finding in autism under various conditions of attention, exposure time and visual angle. *Cognitive Neuropsychology*, 24(5), 550–574.

Visual Responses to Implicit Emotional Faces

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Synonyms

Incidental emotional processing

Definition

Individuals with ASD present with difficulties in understanding and maintaining social interactions, and impaired emotional face processing is a crucial feature of ASD that might be responsible for impairments in the social cognition domain. As acknowledged by a large literature, children and adults with ASD have difficulties in interpreting and categorizing emotional faces, which might be related to avoidance of the eye region of the face. For example, those with ASD

might avoid direct gaze from others and thus not respond in an adapted manner to the social content of others' facial expressions (e.g., not returning a smile). Nevertheless, considerable heterogeneity characterizes both behavioural and neuroimaging studies (Jemel et al. 2006), showing that different results can be found depending on the participants' demographics (e.g., age, severity of autism, language, and/or intellectual level) or on experimental design choices.

To investigate emotional face processing in experimental settings, individuals are often asked to recognize emotions explicitly by detecting or categorizing specific expressions, thus orienting the subject's attention toward the emotional content (top-down process). In everyday life, however, the emotional content of faces is often implicitly processed outside of conscious awareness, allowing one to both understand and communicate intentions with others rapidly in an automatic way (bottom-up process). To study implicit emotional face processing, instructions are usually given to the participants to recognize a face feature other than the emotion itself (e.g., gender) or to recognize another object or stimulus feature while the facial emotions are irrelevant. This allows the researcher to record behavioural responses to the task, therefore confirming task compliance, and at the same time investigate at the brain level implicit or incidental emotional face processing.

Studies have shown that while explicit emotional processing might be intact in some individuals with ASD, atypicalities can be found during implicit tasks (see Kana et al. 2016). It has been suggested that explicit instructions could help people with ASD process the social world more efficiently, while the automaticity of implicit emotion processing would not allow the compensatory mechanisms and may contribute to difficulties observed in ASD (Kana et al. 2016). Although individuals with ASD might explicitly recognize emotional facial expressions as well as typical peers, some studies have shown atypical brain activity, suggesting that individuals with ASD recruit different brain regions to recognize emotions accurately (Wong et al. 2008).

Together with difficulties in the communication and social domains, atypical sensory processing, including hypo- and hyper-responsiveness, is now considered an important diagnostic feature of ASD. In the visual modality, this presents via clinical signs such as light-gazing, shaking fingers in front of the eyes, pressing on the eyes, or lateral glances but also via an increasing literature showing atypical patterns of brain responses to visual stimuli (see Simmons et al. 2009). Given both the sensory and social difficulties in autism, it is crucial to consider faces, not only as important social stimuli but also as visual stimuli.

For instance, to better understand emotional face atypicalities, it is essential to study brain responses from the earliest visual stages, including those preceding face-sensitive responses. To this end, several neuroimaging techniques can be used, such as MEG/EEG or fMRI, to characterize the temporal and/or spatial nature of the brain activity and highlight possible group differences. The N170, a component recorded via the EEG over the temporal regions and reflecting the neural processing of face stimuli, is often abnormal in individuals with ASD. Importantly, the P100 component preceding the N170 and recorded over the occipital region has also been found atypical in children and adults with ASD, suggesting that the earlier stages of visual processing are also affected with face stimuli. In a similar vein, in addition to atypical activation of regions implicated in the processing of faces (e.g., fusiform gyri), abnormal patterns of activation in the occipital cortex have been reported by source analyses (MEG/EEG) (Kovarski et al. 2019; Wong et al. 2008) and fMRI studies.

Taken together, these investigations highlight that brain processing differences exist from the first stages of visual processing, occurring early in time and likely affecting successively higher-level processing. Thus, early visual disturbances appear to be affected in individuals with ASD and can have detrimental consequences on the processing of emotional faces. As the nature of the task is known to modulate brain activity,

using implicit emotional tasks allows the identification of these visual disturbances independently of the frontoparietal projections to the visual cortex, which are modulated by explicit attention to emotions (Vuilleumier and Driver 2007).

See Also

- [Face Processing in Autism: Active Avoidance of the Eyes Versus Passive Indifference](#)
- [Perception](#)
- [Sensory Impairment in Autism](#)
- [Social Behaviors and Social Impairment](#)

References and Reading

- Jemel, B., Mottron, L., & Dawson, M. (2006). Impaired face processing in autism: Fact or artifact? *Journal of Autism and Developmental Disorders*, 36(1), 91–106. <https://doi.org/10.1007/s10803-005-0050-5>.
- Kana, R. K., Patriquin, M. A., Black, B. S., Channell, M. M., & Wicker, B. (2016). Altered medial frontal and superior temporal response to implicit processing of emotions in autism. *Autism Research*, 9(1), 55–66. <https://doi.org/10.1002/aur.1496>.
- Kovarski, K., Mennella, R., Wong, S. M., Dunkley, B. T., Taylor, M. J., & Batty, M. (2019). Enhanced early visual responses during implicit emotional faces processing in Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders*, 49(3), 871–886. <https://doi.org/10.1007/s10803-018-3787-3>.
- Simmons, D. R., Robertson, A. E., McKay, L. S., Toal, E., McAleer, P., & Pollick, F. E. (2009). Vision in autism spectrum disorders. *Vision Research*, 49(22), 2705–2739. <https://doi.org/10.1016/j.visres.2009.08.005>.
- Vuilleumier, P., & Driver, J. (2007). Modulation of visual processing by attention and emotion: Windows on causal interactions between human brain regions. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 362(1481), 837–855. <https://doi.org/10.1098/rstb.2007.2092>.
- Wong, T. K., Fung, P. C., Chua, S. E., & McAlonan, G. M. (2008). Abnormal spatiotemporal processing of emotional facial expressions in childhood autism: Dipole source analysis of event-related potentials. *The European Journal of Neuroscience*, 28(2), 407–416. <https://doi.org/10.1111/j.1460-9568.2008.06328.x>.

Visual Scanning

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Definition

Visual scanning refers to the ocular strategies employed for exploring various classes of visual stimuli including faces, objects, and scenery. Scanning patterns reflect the viewer's strategy for acquiring visual information. Therefore, visual scanning precedes visual information processing. Ocular strategies, movements and fixations measured with eye-tracking technology, are the focus of this section.

Historical Background

The suggestion that people with autism view the world differently than neurotypical individuals is not new. An early study of facial recognition indicated that children with autism were better able to judge facial identity than controls using only the eye or mouth region rather than images of the whole face. Younger children showed particularly heavy reliance on the mouth region (Langdell 1978). The observation that children with ASD use different information than their typically developing peers suggests the likelihood that they also explore faces differently. An investigation of looking targets during a solitary play task indicated that toddlers with ASD looked less to people and more to objects, demonstrated fewer shifts of attention toward people and more shifts of attention toward objects than typically developing toddlers (Swettenham et al. 1998). Similarly, retrospective videotape studies of infants later diagnosed with autism indicate that these infants look less to other people's faces

than typically developing infants (Osterling and Dawson 1994). Cumulatively, these studies suggest that children with ASD scan faces and social scenes differently than controls. The literature reviewed below focuses on recent research that used eye-tracking technology to characterize differences in visual scanning patterns between people with and without autism spectrum disorders.

Current Knowledge

Many studies have reported that children and adults with ASD scan faces, objects, and scenery differently than controls, but the evidence is far from consistent. Some studies find no evidence of atypical visual scanning (e.g., Rutherford and Towns 2008; Van der Geest et al. 2002a). Other studies report atypical visual scan patterns only in certain conditions (e.g., Speer et al. 2007) or in certain age-groups (e.g., Nakano et al. 2010).

Visual Scanning of Faces: Fixation Patterns

Most studies that report atypical scanning patterns in response to faces base this claim on participants' gaze fixation patterns during eye-tracking measures in which images of faces are presented on a video monitor. Individuals with ASD fixate longer (Anderson et al. 2006; Chawarska and Shic 2009; Pelphrey et al. 2002; Trepagnier et al. 2002) and more frequently (Pelphrey et al. 2002) on the periphery of faces and less on internal facial features compared to controls. When participants do look at the internal features of the face, individuals with autism look longer to the mouth (Jones et al. 2008; Klin and Jones 2008; Klin et al. 2002; Neumann et al. 2006) and less to eyes relative to controls (Boraston et al. 2008; Corden et al. 2008; Dalton et al. 2005; Hernandez et al. 2009; Jones et al. 2008; Klin et al. 2002; Klin and Jones 2008; Pelphrey et al. 2002; Speer et al. 2007; Sterling et al. 2008). The observation that adults with ASD look less than controls to the eyes and mouth of still images (Sterling et al. 2008) is compatible with preference for peripheral rather than core features of the face (Anderson et al. 2006; Chawarska and Shic 2009; Pelphrey et al. 2002).

One study found evidence of age-related differences in face-scanning behaviors: children with ASD (age 3–9) looked less to the mouth than controls, but the pattern was reversed in adults (Nakano et al. 2010). Variations of these results have also been reported. For instance, 3- to 6-year-old children with ASD looked longer at the mouth than at the eyes compared to typically developing controls. However, the scanning patterns demonstrated by the ASD group did not differ from that of a younger group of neurotypical controls, 1–3 years of age (von Hofsten et al. 2009). Although 6-month-old siblings of children with autism looked more to the mouth than the eyes (Merin et al. 2007), a follow-up on the same children found no link between eye-mouth preferences at 6 months and autism symptoms or diagnostic classification at 24 months (Young et al. 2009). A study that reported equal fixation time between groups also reported fewer fixations on the eye region by children with ASD than controls (van der Geest et al. 2002b). A study involving adolescents with ASD reported atypical scanning of faces, but differences were in the opposite direction of many of the studies cited above. In response to grayscale images of faces, adolescents with and without ASD fixated longer on the upper region of the face than the lower region, but the ASD group fixated even longer on the upper region than typically developing controls (McPartland et al. 2010).

Multidimensional scaling has been employed to analyze participants' gaze patterns with respect to timing and spatial coordinates simultaneously (Nakano et al. 2010). This analytic method revealed highly consistent scanning patterns among typically developing children and adults and highly variable results among children and adults with ASD. Scan patterns of ASD participants differed considerably from each other, and the ASD group as a whole differed significantly from controls.

Attention Shifting

Eye movements and gaze shifts are measures of interest in studies about face scanning. Two studies report less shifting of visual attention by

individuals with ASD compared to controls during the presentation of dynamic social stimuli (Nakano et al. 2010; von Hofsten et al. 2009). One study investigated visual scanning of dynamic faces using video stimuli in which models were speaking. When the auditory modality was combined with the visual modality, children and adults with ASD shifted their attention away from a speaking model earlier than controls, who continued to watch the model's face until she stopped speaking (Nakano et al. 2010). These results are consistent with the observation that toddlers with ASD diverted their attention away from faces and toward objects more rapidly than controls (Chawarska et al. 2010).

Stimulus Effects

In general, the studies or conditions within studies that did not find evidence of atypical visual scanning of faces by individuals with ASD presented still images of faces (Rutherford and Towns 2008; Speer et al. 2007; Van der Geest et al. 2002b). However, numerous studies that employed still images have reported atypical visual scanning of faces in participants with ASD (Anderson et al. 2006; Boraston et al. 2008; Chawarska and Shic 2009; Corden et al. 2008; Dalton et al. 2005; Falck-Ytter et al. 2010; Hernandez et al. 2009; Neumann et al. 2006; Pelphrey et al. 2002; Sterling et al. 2008). Differences in face-scanning patterns were observed more consistently in studies that presented participants with dynamic social stimuli (Jones et al. 2008; Klin et al. 2002; Klin and Jones 2008; Nakano et al. 2010; Speer et al. 2007; von Hofsten et al. 2009). This pattern of results suggests that visual stimuli that are most similar to real-life visual experience are most effective at capturing atypical scan patterns in people with autism (Klin et al. 2003). Other characteristics of visual stimuli that may contribute to the magnitude of group differences include the color and size of the stimuli and duration of presentation (McPartland et al. 2010). Grayscale and static images may elicit similar visual scanning patterns in typical and ASD participants (Speer et al. 2007).

One study sought to address the effects of stimulus type and presented images of people in

isolation and in social contexts, in both still and dynamic formats (Speer et al. 2007). All images also contained nonsocial scenic elements. The ASD group (males, 9–18 years old) looked less at the eyes and more at the bodies of people presented dynamically in groups, but no group differences were observed in the other three conditions (Speer et al. 2007). Another factor that has been associated with differences in result patterns is the duration of stimulus exposure (McPartland et al. 2010). Studies that presented still images for longer durations did not report less looking to eyes by participants with ASD (Van der Geest et al. 2002b (10 s), McPartland et al. 2010 (8 s)), whereas atypical patterns in participants with ASD were reported in studies that presented images more briefly (e.g., Pelphrey et al. 2002 (2 s)).

Age Effects

The studies and portions of studies in which atypical visual scanning behaviors were not observed involved adolescents (Speer et al. 2007; Van der Geest et al. 2002b) and adults (Rutherford and Towns 2008). One study that compared visual scanning of faces between age-groups reported atypical scan patterns in both children and adults, but the differences between ASD and neurotypical participants changed across development: relative to controls, children with ASD fixated less on the mouth, whereas adults with ASD fixated more on the mouth (Nakano et al. 2010). The concordance of results between the child cohort evaluated by Nakano and the results reported for the same developmental period by von Hofsten et al. (2009) suggests that preferential looking to the mouth may be adaptive early in development. Consistent with this claim, a positive correlation was observed in siblings of children with autism in duration of fixation on the mouth at 6 months of age and language skills at 24 months (Young et al. 2009).

Visual Scanning of Scenes That Contain Social Stimuli

A relatively small body of literature has described the visual scan patterns of individuals with ASD in response to complex scenes that include social

stimuli as well as objects and/or scenery. These studies complement what is known about processing of faces presented in isolation by exploring participants' differential preferences among classes of visual stimuli. The stimuli presented in these studies aim to portray situations that individuals may encounter in their everyday lives (Speer et al. 2007). Relative to controls, adolescents with autism look less to the eyes, more to the body, and more to objects (Klin et al. 2002). This indicates that people with autism do not spontaneously access the same information as controls. Comparable results indicate that adolescents with autism fixate less on faces than on bodies and scenic background (Riby and Hancock 2009). A study that presented human figures among multiple objects did not report differences in looking patterns between children with autism and neurotypical children (Van der Geest et al. 2002a). The Klin study employed dynamic live-action video footage, and the van der Geest study employed static cartoonlike images. The Riby study sequentially presented live-action and cartoon footage in a dynamic format. These distinctions suggest the possibility that, while people with ASD do not differ in looking patterns in all contexts, differences in scanning patterns are more likely to be evident in the context that most closely resembles everyday experience. This interpretation is supported by a fourth study (Nakano et al. 2010) that explored looking patterns in response to live-action video footage of a young girl, who is initially silent, but then announces her name. At the same time that she speaks, a caption appears at the bottom of the screen, providing her name and age in written form. The authors report that children (age 3–9) and adults with ASD looked longer at the text than child and adult controls, respectively (Nakano et al. 2010). The looking patterns of children with ASD were surprising given that most could not read letters. Although text has visual properties that distinguish it from objects and scenery, the finding suggests that children and adults with ASD demonstrate less preferential attention to social stimuli than neurotypical controls. Attenuated preference for social stimuli has also been reported in a study of toddlers with ASD (mean

age = 32 months; Chawarska et al. 2010). Collectively, this pattern of results suggests the possibility that selective attention to nonsocial information during everyday encounters interferes with the achievement of expertise in processing informative social phenomena.

Visual Scanning of Nonsocial Stimuli

Two recent studies have assessed scan patterns of children (Anderson et al. 2006) and adolescents (McPartland et al. 2010) with ASD relative to neurotypical controls in response to static nonsocial visual stimuli. The nonsocial stimuli employed in the Anderson study include animal faces, toys, and landscapes. In addition to evaluating participants' scanning behaviors in response to human faces, the McPartland study presented adolescents with monkey faces, two-dimensional geometric patterns, and three-dimensional curvilinear objects. Reported visual scan patterns differ between the two studies and the associated age-groups. Adolescents with ASD looked to upper portion of geometric shapes and curvilinear objects longer than typically developing controls (McPartland et al. 2010). Among children 1–6 years of age, the only category of stimuli that captured different looking patterns between groups was landscapes (Anderson et al. 2006). In response to still images of landscapes (clouds, rocks, grass, and water), children with ASD demonstrated shorter fixations and less total fixation time than typically developing children and children with cognitive delays. The authors suggest that shorter looking at landscapes may reflect more rapid processing of this class of visual information by children with ASD. This interpretation is consistent with the finding that children with ASD perform better than typically developing children on object-recognition tasks (e.g., Trepagnier et al. 2002). The small sample observed in the Anderson study argues for the need to replicate these findings.

Correlates of Atypical Scanning Patterns

Several of the studies described above have reported associations between visual scanning patterns and other areas of functioning associated with autism. If atypical patterns of visual scanning

are associated with severity of social impairment, this would suggest that a common mechanism underlies strategies for acquiring social information and functioning in the social world. Alternatively, if individuals with autism consistently acquire different information than neurotypical individuals, this may constrain social learning opportunities and alter the development of the social brain. Strong relationships between scanning patterns and autistic symptomatology would suggest that atypical visual scanning is a core feature of autism. Alternatively, scan patterns may be disrupted in a subgroup of people with autism. In support of this latter possibility, a study of 5-year-olds reported that the preference for looking at the mouth relative to the eyes was present in a subset of individuals with autism whose social impairments were more severe than their communication impairments (Falck-Ytter et al. 2010). Other studies have articulated results that apply to the full sample of participants with ASD, less looking to the eyes corresponded with other impairments in people with ASD across developmental stages. These include greater social impairment in 2-year-olds (Jones et al. 2008), low levels of social responsiveness in adolescents (Speer et al. 2007), and higher levels of self-reported social anxiety in adults (Trepagnier et al. 2002). More looking to the mouth at 6 months appeared to be adaptive to the development of communication skills in 24-month-olds (Young et al. 2009), but more looking to the mouth corresponds with worse social outcomes in participants in the wide span from 2 years (Falck-Ytter et al. 2010) to 18 years of age (Speer et al. 2007).

A different pattern of associations was reported when social stimuli were presented in scenes that also contained nonsocial stimuli. In that context, adolescents' longer fixations on the mouth were associated with *greater* social adaptation and *lower* autistic social impairment. In the same study, fixation time on objects was associated with lower social adaptation and greater autistic social impairment (Klin et al. 2002). Differences in patterns of association between scanning behaviors and social functioning confirm that the importance of looking targets varies in relation to

the context. Future research should explore looking patterns to faces in isolation as well as looking patterns to scenes that include faces in order to clarify the implications of preferential attention to one type of stimuli or the other.

Among 4-year-olds with ASD ($n = 9$), decreased looking time to landscapes corresponded with lower levels of restricted and repetitive behaviors (Anderson et al. 2006).

Summary

Support for the claim that visual scanning of social stimuli distinguishes people with ASD from neurotypical controls is plentiful, but not entirely consistent. Some studies find no differences between groups, and others find inconsistent patterns of distinction. Fewer efforts have been made to explore patterns of visual scanning in response to nonsocial stimuli, but there is some evidence of atypical scanning patterns in response to images of objects, patterns, and scenery. The inconsistent findings among studies in this domain of research may be attributable to format of visual stimuli, duration of stimulus presentation, and the age or developmental level of research participants.

Future Directions

Studies of visual scanning behavior show promise for characterizing atypical information-gathering processes in individuals with autism. The summary of findings reported above suggests that additional research is needed to clarify effects of age, stimulus format, and research design with respect to the behaviors of interest. These factors can be evaluated in isolation such that various formats of stimulus can be compared within person as well as between groups. Longitudinal research is needed to determine whether visual scanning patterns change across time. Comparison of stimulus formats across time would also help to clarify relationships with other developmental measures such as communication, social skills, and restricted and repetitive behaviors.

Future research should take steps to standardize means of measuring visual scanning behavior between research sites given the possibility that results differ systematically among eye-tracking devices. The precision needed to characterize and compare scanning behaviors argues for the continued fine-tuning and evaluation of instrumentation used for conducting this kind of research.

See Also

- [Face Perception](#)
- [Face Recognition](#)
- [Perception](#)
- [Perceptual Development](#)
- [Visual Processing](#)

References and Reading

- Anderson, C. J., Colombo, J., & Jill Shaddy, D. (2006). Visual scanning and pupillary responses in young children with autism spectrum disorder. *Journal of Clinical and Experimental Neuropsychology*, 28(7), 1238–1256.
- Boraston, Z. L., Corden, B., Miles, L. K., Skuse, D. H., & Blakemore, S. J. (2008). Brief report: Perception of genuine and posed smiles by individuals with autism. *Journal of Autism and Developmental Disorders*, 38(3), 574–580.
- Chawarska, K., & Shic, F. (2009). Looking but not seeing: Atypical visual scanning and recognition of faces in 2 and 4-year-old children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 39(12), 1663–1672.
- Chawarska, K., Volkmar, F., & Klin, A. (2010). Limited attentional bias for faces in toddlers with autism spectrum disorders. *Archives of General Psychiatry*, 67(2), 178–185.
- Corden, B., Chilvers, R., & Skuse, D. (2008). Avoidance of emotionally arousing stimuli predicts social-perceptual impairment in Asperger's syndrome. *Neuropsychologia*, 46(1), 137–147.
- Dalton, K. M., Nacewicz, B. M., Johnstone, T., Schaefer, H. S., Gernsbacher, M. A., Goldsmith, H. H., et al. (2005). Gaze fixation and the neural circuitry of face processing in autism. *Nature Neuroscience*, 8(4), 519–526.
- Falck-Ytter, T., Fernell, E., Gillberg, C., & Von Hofsten, C. (2010). Face scanning distinguishes social from communication impairments in autism. *Developmental Science*, 13(6), 864–875.
- Hernandez, N., Metzger, A., Magné, R., Bonnet-Brilhault, F., Roux, S., Barthelemy, C., et al. (2009). Exploration of core features of a human face by healthy and autistic adults analyzed by visual scanning. *Neuropsychologia*, 47(4), 1004–1012.
- Jones, W., Carr, K., & Klin, A. (2008). Absence of preferential looking to the eyes of approaching adults predicts level of social disability in 2-year-old toddlers with autism spectrum disorder. *Archives of General Psychiatry*, 65(8), 946–954.
- Klin, A., & Jones, W. (2008). Altered face scanning and impaired recognition of biological motion in a 15-month-old infant with autism. *Developmental Science*, 11(1), 40–46.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, 59(9), 809–816.
- Klin, A., Jones, W., Schultz, R., & Volkmar, F. (2003). The enactive mind, or from actions to cognition: Lessons from autism. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 358(1430), 345.
- Langdell, T. (1978). Recognition of faces: An approach to the study of autism. *Journal of Child Psychology and Psychiatry*, 19(3), 255–268.
- McPartland, J. C., Webb, S. J., Keehn, B., & Dawson, G. (2010). Patterns of visual attention to faces and objects in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 41(2), 148–157.
- Merin, N., Young, G. S., Ozonoff, S., & Rogers, S. J. (2007). Visual fixation patterns during reciprocal social interaction distinguish a subgroup of 6-month-old infants at-risk for autism from comparison infants. *Journal of Autism and Developmental Disorders*, 37(1), 108–121.
- Nakano, T., Tanaka, K., Endo, Y., Yamane, Y., Yamamoto, T., Nakano, Y., et al. (2010). Atypical gaze patterns in children and adults with autism spectrum disorders dissociated from developmental changes in gaze behaviour. *Proceedings of the Royal Society B: Biological Sciences*, 277(1696), 2935.
- Neumann, D., Spezio, M. L., Piven, J., & Adolphs, R. (2006). Looking you in the mouth: Abnormal gaze in autism resulting from impaired top-down modulation of visual attention. *Social Cognitive and Affective Neuroscience*, 1(3), 194.
- Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders*, 24(3), 247–257.
- Pelphrey, K. A., Sasson, N. J., Reznick, J. S., Paul, G., Goldman, B. D., & Piven, J. (2002). Visual scanning of faces in autism. *Journal of Autism and Developmental Disorders*, 32(4), 249–261.
- Riby, D., & Hancock, P. J. B. (2009). Looking at movies and cartoons: Eye-Tracking evidence from Williams syndrome and autism. *Journal of Intellectual Disability Research*, 53(2), 169–181.
- Rutherford, M. D., & Towns, A. M. (2008). Scan path differences and similarities during emotion perception

- in those with and without autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(7), 1371–1381.
- Speer, L. L., Cook, A. E., McMahon, W. M., & Clark, E. (2007). Face processing in children with autism. *Autism*, 11(3), 265.
- Spezio, M. L., Adolphs, R., Hurley, R. S. E., & Piven, J. (2007). Analysis of face gaze in autism using. *Neuropsychologia*, 45(1), 144–151.
- Sterling, L., Dawson, G., Webb, S., Murias, M., Munson, J., Panagiotides, H., et al. (2008). The role of face familiarity in eye tracking of faces by individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(9), 1666–1675.
- Swettenham, J., Baron-Cohen, S., Charman, T., Cox, A., Baird, G., Drew, A., et al. (1998). The frequency and distribution of spontaneous attention shifts between social and nonsocial stimuli in autistic, typically developing, and nonautistic developmentally delayed infants. *Journal of Child Psychology and Psychiatry*, 39(5), 747–753.
- Trepagnier, C., Sebrechts, M. M., & Peterson, R. (2002). Atypical face gaze in autism. *Cyberpsychology & Behavior*, 5(3), 213–217.
- Van der Geest, J. N., Kemner, C., Camfferman, G., Verbaten, M. N., & Van Engeland, H. (2002a). Looking at images with human figures: Comparison between autistic and normal children. *Journal of Autism and Developmental Disorders*, 32(2), 69–75.
- Van der Geest, J. N., Kemner, C., Verbaten, M. N., & Van Engeland, H. (2002b). Gaze behavior of children with pervasive developmental disorder toward human faces: A fixation time study. *Journal of Child Psychology and Psychiatry*, 43(5), 669–678.
- von Hofsten, C., Uhlig, H., Adell, M., & Kochukhova, O. (2009). How children with autism look at events. *Research in Autism Spectrum Disorders*, 3(2), 556–569.
- Young, G. S., Merin, N., Rogers, S. J., & Ozonoff, S. (2009). Gaze behavior and affect at 6 months: Predicting clinical outcomes and language development in typically developing infants and infants at risk for autism. *Developmental Science*, 12(5), 798–814.

Visual Schedule

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Synonyms

[Picture schedule](#)

Definition

A visual schedule is a type of visual support used to help children anticipate transitions and upcoming activities (Vaughn et al. 2005) and to understand the expected agenda for a given period (e.g., daily schedule, class period schedule). Visual schedules typically consist of pictures, or a combination of pictures and words, that represent the expected activities in the order in which they are to be completed. Visual schedules can be created for an individual child or for an entire classroom.

See Also

- [Pictorial Cues/Visual Supports \(CR\)](#)
- [Visual Supports](#)

References and Reading

- Vaughn, B., Fox, L., & Lentini, R. (2005). *Creating teaching tools for young children with challenging behavior*. Tampa: University of South Florida.

Visual Schedules

- [Daily Routines](#)

Visual Supports

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Definition

Visual supports can be a type of augmentative communication. Visual supports are graphic cues used to increase an individual's communication skills and understanding of expectations by providing graphic models, prompts, or reminders of

how to participate successfully in interactions and routines. Visual supports can be used to make choices or provide expectations for behavior. Examples of visual supports include picture schedules, video modeling, and Social Stories™.

Historical Background

Some individuals with autism are able to take advantage of visual input more effectively than verbal information. This may be interpreted as individuals with autism having better visual skills than auditory skills. Visual supports may be effective because they are more permanent while verbal information is more transient. The concrete nature of visual supports (e.g., a visual symbol has a specific, unchanging referent) may be the reason that these systems are effective for individuals with autism.

Visual support systems can be used to improve the functioning of individuals with autism. Visual supports allow the individual to organize, understand, and remember information. While using visual supports, individuals are able to participate more readily and effectively in communicative and social interactions. Until the individual has learned the expected behavior, the visual support offers a permanent reference. There is evidence that communication and social interaction skills increase while challenging behaviors decrease as the individual begins to understand the expectations and is provided with a means to communicate appropriately.

Current Knowledge

Visual supports can be a type of augmentative communication. These supports draw upon an individual's strength in understanding visual information since the supports are permanent and easy to understand. Individuals with autism may need the extra details and information that visual supports provide before he/she can respond appropriately.

The types of visual supports and symbols used will be determined by the purpose of the system as well as the individual's needs and abilities.

A symbol, or icon, is a visual representation of a word or concept. These symbols may represent real objects or ideas. When choosing symbols, the symbols should be meaningful to the system user. Examples of symbols include real objects, photographs of objects, simple drawings, printed words, and printed words paired with photographs or drawings. Symbols are more likely to be effective if they are not distracting and irrelevant information is eliminated. After the symbols are chosen, the system user needs to be taught what the symbols represent and how the symbols work as part of the visual support system. In addition, visual supports should be easy for the individual to use and manipulate, sturdy, and flexible (i.e., symbols can change as the situation or individual changes). Supports should be inexpensive and quick to produce as an individual may use numerous systems throughout the day.

There are many examples of visual supports including picture schedules, video modeling, and Social Stories. Picture schedules, photographic activity schedules, and calendar systems visually represent a sequence of events and provide information on when and where something will take place. In addition, the schedule provides information on what will happen next allowing an individual to anticipate future transitions. These systems allow for self-management and prepare the individual for future events. The calendar may include transition cues (i.e., an object or symbol card) which allow the individual to move independently from one activity to another and decrease their reliance on an adult to verbally prompt them. Activity schedules or mini-calendars can be used to show the sequence of steps of an individual activity. Individuals with autism may be better able to complete a routine or activity when the steps are presented in a meaningful way with a clear start and finish. Picture schedules or calendar systems have been used to increase task completion and engagement.

Some visual supports can be used to teach behavioral expectations. Video modeling provides an individual with autism a model of an appropriate behavior via videotape. For example, if the target behavior is social initiations, the person in the videotape would demonstrate an appropriate example of social initiation. Conversely,

video self-modeling provides a model of the actual individual engaging in an appropriate behavior. In this case, the individual with autism would watch a videotape of himself/herself engaging in an appropriate example of social initiation. Video modeling and video self-modeling have been used with individuals with autism to increase conversational skills, initiations, and play behaviors as well as decrease challenging behaviors.

Social Stories are short stories written to describe a situation or behavior that may be problematic for the individual such as initiating and responding during a conversation, responding appropriately, and being flexible to changes to a routine. These stories are usually written in the first person and may or may not contain pictures. The story text describes the situation or behavior in clear terms and includes information on who is involved, what is happening, and where it is happening. The story may include information on social cues and how the individual with autism can respond as well as how other people may feel or react. These stories can be used in a variety of settings (e.g., home, school, day care, community outings, etc.) and may be effective for children with autism, Asperger's syndrome, language impairment, and other developmental disabilities. Social Stories may be effective because they specifically address a deficit in individuals with autism. Social Stories have been used to decrease challenging behaviors (e.g., shouting, tantrums) and increase socially appropriate behaviors (e.g., following directions, maintaining eye contact).

Other examples of visual supports include checklists, visual cues, pictorial prompts, and activity choice boards. These supports may be used to increase task completion, engagement, and choice-making abilities.

Caregivers, educators, and therapists will need to consider many factors to determine which visual support system is most appropriate for an individual. The cost of the system and time to produce the system are two factors to consider. For example, video self-modeling may be a very effective strategy but may be costly for some individuals and take too much time to produce and edit. On the other hand, simple line drawings are inexpensive and quick to produce but may not

meet the needs of some individuals. The physical size of the system also should be taken into consideration. Large calendar systems may be effective for some individuals, but often will need to be shrunk to make them more portable. The smaller system, however, should contain the same information and level of detail as the larger system initially, though it may be possible to fade out details to a more normalized reminder system. Some systems may be more effective for certain behaviors. For example, when engaging in a sequential routine, a dynamic system may be most appropriate as pictures can be added to and removed from the visual support. When an individual completes a step in the routine, he/she removes the associated picture for that step and moves to the next step.

Positive changes in behavior have been documented when using visual support systems where written directions or expectations are supported by pictures or symbols. The pictures or symbols give the individual a static example that he/she can refer to until the expected behavior is learned.

Future Directions

Future research should focus on systems and symbols that produce generalized effects. To improve generalization, multiple exemplars of a symbol within a system may be necessary. During video modeling, multiple examples of social initiation may produce stronger effects than a single example. Multiple examples or symbols to represent the same concept within a Social Story may help individuals generalize their behaviors. For instance, if the story is being used to teach verbal responses, it may be useful to provide multiple examples of verbal responses within the story text. During system development, how the target behaviors may generalize across settings, activities, and people should be considered. A visual support may be very effective at home with a parent but not effective at school with a teacher. Additional training or adaptations to increase generalization should be part of the planning process.

Future research also should focus on maintaining improved behavior outcomes over

time, with and without a system in place. Immediate behavior improvements may be caused by the novelty of a new visual support. Behavior change maintained over time would indicate that the change was a result of the visual support and not simply a novelty effect. Maintaining behavior change after the support has been removed also should be planned. Are individuals able to maintain behavior change over time when using the system? Is the individual able to function as well without the system? If not, are smaller, less intrusive systems able to produce similar results? Adaptations to increase independence should be part of the visual support planning process, especially for older individuals.

See Also

- [Icon](#)
- [Iconic Systems](#)
- [Picture Exchange Communication System](#)
- [Social Stories](#)
- [Video Instruction](#)
- [Video Modeling/Video Self-modeling](#)
- [Visual Schedule](#)

References and Reading

- American Speech-Language-Hearing Association. (2006). *Guidelines for speech-language pathologists in diagnosis, assessment, and treatment of autism spectrum disorders across the life span* (Guidelines). American Speech-Language-Hearing Association. Retrieved 28 Mar 2011 from www.asha.org/policy.
- Bellini, S., & Akullian, J. (2007). A meta-analysis of video modeling and video self-modeling interventions for children and adolescents with autism spectrum disorders. *Exceptional Children*, 73(3), 264–287.
- Beukelman, D. R., & Mirenda, P. (1992). *Augmentative and alternative communication: Management of severe communication disorders in children and adults*. Baltimore: Paul H. Brookes.
- Buggey, T., Hoomes, G., Sherberger, M. E., & Williams, S. (2011). Facilitating social initiations of preschoolers with autism spectrum disorders using video self-modeling. *Focus on Autism and Other Developmental Disabilities*, 26(1), 25–36.
- Dettmer, S., Simpson, R. L., Myles, B. S., & Ganz, J. B. (2000). The use of visual supports to facilitate transitions of students with autism. *Focus on Autism and Other Developmental Disabilities*, 15, 163–169. <https://doi.org/10.1177/108835760001500307>.
- Hodgdon, L. (2000). *Visual supports for improving communication*. Troy: QuirkRoberts.
- Iovannone, R., Dunlap, G., Huber, H., & Kincaid, D. (2003). Effective educational practices for students with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, 18, 150–165. <https://doi.org/10.1177/10883576030180030301>.
- Janzen, J. E. (2003). *Understanding the nature of autism: A guide to the autism spectrum disorders* (2nd ed.). San Antonio: PsychCorp.
- Johnston, S., Nelson, C., Evans, J., & Palazolo, K. (2003). The use of visual supports in teaching young children with autism spectrum disorder to initiate interactions. *Augmentative and Alternative Communication*, 19, 86–103.
- Mirenda, P., & Erickson, K. (2000). Augmentative communication and literacy. In A. Wetherby & B. Prizant (Eds.), *Autism spectrum disorders: A transaction development perspective* (pp. 333–367). Baltimore: Paul H. Brookes.
- Odom, S. L., Brown, W. H., Frey, T., Karasu, N., Smith-Canter, L. L., & Strain, P. S. (2003). Evidence-based practices for young children with autism: Contributions for single-subject design research. *Focus on Autism and Other Developmental Disabilities*, 18, 166–175. <https://doi.org/10.1177/10883576030180030401>.
- Quill, K. A. (1995). *Teaching children with autism: Strategies to enhance communication and socialization*. New York: Delmar.
- Quill, K. A. (1997). Instructional considerations for young children with autism: The rationale for visually cued instruction. *Focus on Autism and Other Developmental Disabilities*, 27(6), 697–714.
- Test, D. W., Richter, S., Knight, V., & Spooner, F. (2011). A comprehensive review and meta-analysis of the social stories literature. *Focus on Autism and Other Developmental Disabilities*, 26(1), 49–62.
- Thiemann, K. S., & Goldstein, H. (2001). Social stories, written text cues, and video feedback: Effects on social communication of children with autism. *Journal of Applied Behavior Analysis*, 34, 425–446.

Visual Thinking

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Synonyms

[Picture thinking](#)

Definition

In her 1995 book, *Thinking in pictures and other reports from my life with autism*, Temple Grandin describes visual thinking as the ability to think in pictures. Grandin describes how she automatically converts written and spoken words into pictures. She is able to view these visualizations as a running videotape in her mind.

References and Reading

Grandin, T. (1995). *Thinking in pictures and other reports from my life with autism*. New York: Vintage.

Visual/Somatosensory Cognitive Potentials

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Definition

Cognitive potentials are electrophysiological responses to internal or external events that are derived from recordings of brain activity made with an electroencephalogram, or EEG. To record EEG, sensors that detect electrical activity are placed on the participant's scalp and secured with a net, cap, or glue. With millisecond-level resolution, these sensors record the electrical activity that naturally propagates to the scalp from thousands of synchronously firing cortical pyramidal neurons. Cognitive potentials and other measures derived from EEG recordings can thus provide important information about the neurophysiological mechanisms underlying behavioral symptoms of developmental disabilities like autism.

Cognitive potentials are derived by time-locked averaging of segments of EEG recorded during a series of similar events (such as pressing

a button or viewing a particular type of stimulus). This averaging technique enables brain activity related to the event to be at least partially dissociated from other processes because randomly distributed "background" brain activity is minimized, while activity that occurs with a reliable temporal relation to the stimulus is retained. The resulting "event-related potential" (ERP) waveform has a characteristic pattern of peaks and troughs that have been termed "components," and the amplitude and latency of particular components are typically sensitive to particular types of event.

Components are typically labeled by polarity (P for a positive-going deflection, N for a negative-going deflection) and absolute or relative timing (e.g., the P300 is a positive-going deflection that peaks approximately 300 ms after an event occurred or the P3 is the third positive-going deflection after an event occurred). Component characteristics like latency and amplitude can be measured to examine the speed and efficiency of particular cognitive processes, although it is also recognized that visible components likely represent the summation on the scalp of several different underlying sources of neural activity. Early components that are related to sensory processing are commonly termed "evoked potentials" and typically occur within 300 ms of an event; later components occurring between 300 and 2,000 ms of the event are often referred to as "cognitive potentials" and are the focus of this section. Cognitive potentials likely reflect neural activity associated with "higher-level" cognitive processes like memory, attention, error detection, inhibition, and semantic processing and can thus provide insight into the neural correlates of cognitive functions that appear atypical in autism.

Historical Background

The use of EEG to measure brain activity in humans dates back to Hans Berger's recordings of the brain activity of his young son in the 1920s, although Richard Caton had earlier recorded responses to sensory stimuli in animals in

nineteenth-century Liverpool (for a history of the early use of EEG, see Brazier 1961). The event-related potential (ERP) technique was developed in the late 1940s and at that time involved the photographic superposition of several time-locked EEG traces. Although later analog-averaging techniques increased efficiency, it was the introduction of digital signal processing methods that made ERP feasible for larger research studies. In the twentieth century, the information technology revolution transformed the recording, acquisition, and analysis of EEG and enabled the introduction of high-density electrode arrays, faster sampling rates, and more sophisticated analytic techniques.

The potential of EEG for revealing information about the nature of neural impairments in autism was recognized from the early development of the techniques, with the first published reports of event-related potentials in autism emerging in the 1960s. Most early studies focused on the P300, a clearly visible positive-going deflection that is elicited by a wide range of events and is modulated by factors such as attention, probability, novelty, and task relevance. With the development of increasingly powerful acquisition and analysis systems, work expanded to examine other components over multiple electrodes. Although the majority of EEG studies have been conducted with older and higher-functioning participants, the advent of high-impedance EEG recording arrays that afford more rapid application and do not require scalp abrasion has facilitated more recent work with individuals with autism who are younger and have fewer adaptive skills. Such developments in EEG technology have led to an expanding body of knowledge about the neural correlates of a range of cognitive abilities in autism, described in the next sections.

Current Knowledge

Face Recognition

Many individuals with autism report difficulties with recognizing familiar faces, and interpreting the signals provided by familiar faces is central to successful social interaction. Understanding the

source and development of difficulties with face processing facilitates the design of effective interventions and may also provide deeper insight into the process of symptom development in autism. Recent models of the development of face processing emphasize the interaction between “expected” experience with faces in the child’s environment and congenital commitment of neural regions with the computational and anatomical qualities optimal for processing complex stimuli like faces. Are face recognition difficulties in autism related to congenital dysfunction in these neural regions, or could they be related to disruptions to the specialization process caused by a lifetime of reduced self-directed experience with faces? Testing the early development of face-processing skills in children with autism is central to testing such proposals.

In a series of studies, Dawson, Webb, Jones, and colleagues have used event-related potentials to examine the development of neural responses to familiar and unfamiliar faces in individuals with autism from toddlerhood to adulthood. Event-related potentials can provide particularly valuable developmental information because the same methodology and paradigms can be used across a wide age range. In this set of studies, participants watched repeated pictures of an unfamiliar face and a highly familiar face (a parent or loved one) while EEG was recorded with a high-density sensor array. Key cognitive potentials examined in the younger populations included the Nc, a negative-going deflection observed over frontocentral regions and associated with attention; in adulthood, analyses focused on the posterotemporal N250 and N400, negative-going deflections associated with identity processing.

In an initial study, Dawson et al. (2002) found that typically developing 3- to 4-year-old children showed a significantly more negative Nc response to an unfamiliar face than a familiar face, indicating that they recognized the familiar face. In contrast, in 3–4-year-old children with autism, ERP responses to familiar and unfamiliar faces did not significantly differ over any examined component. Critically, responses to familiar and unfamiliar objects in the group with autism were relatively typical, suggesting that disruptions to

familiarity processing may be face-specific. Since the familiar face was a picture of the child's mother, could this mean that children with autism did not recognize a picture of their primary caregiver? Possibly, but subsequent studies support an alternate explanation. In typical development, Nc responses to faces change over early childhood such that initially larger (more negative) responses to a highly familiar face during infancy become larger responses to the face of a stranger by age 3–4 years, with a transitional period around the end of the second year of life during which responses to highly familiar and unfamiliar faces are not significantly different. Possibly, the 3–4-year-old children with autism tested by Dawson et al. (2002) were showing the pattern associated with younger typically developing children: the transitional stage between larger neural responses to a familiar versus unfamiliar stimulus. If so, this might suggest that children with autism may have a developmental delay in the effect of facial familiarity on the neural correlates of attention.

To test this possibility, Webb et al. (2011) examined event-related responses to familiar and unfamiliar faces in 18–30-month-old toddlers with autism and two groups of typically developing toddlers. The first group of typically developing toddlers were chronologically age-matched to the toddlers with autism and as expected did not show differential responses to the familiar and unfamiliar faces. The second group of typically developing toddlers was aged between 12 and 18 months and had socialization skills that matched the group of older toddlers with autism. Replicating previous work, this group of toddlers showed greater Nc responses to the familiar than the unfamiliar face. Critically, we predicted that the 18- to 30-month-old toddlers with autism would show Nc responses to the familiar and unfamiliar face that matched those of the 12- to 18-month-old typically developing toddlers. This prediction was confirmed: 18- to 30-month-old toddlers with autism showed significantly greater Nc responses to the highly familiar face than the unfamiliar face. Further, stronger socialization skills as measured by parent report were correlated with a more mature pattern of neural response to familiarity in both groups. These

findings confirm that toddlers with autism can recognize their parent from a static picture, but suggest that the modulation of attention to faces by familiarity was delayed in line with the development of socialization skills in this group. This is consistent with the possibility that developmental delays in face recognition in children with autism may in part be related to children's reduced self-directed social experiences with faces in their natural environment.

Consistent with the possibility that disruptions in the early neural correlates of familiar-face recognition are development delays rather than absolute impairments, Webb et al. (2010) found that N250 and N400 responses to highly familiar and unfamiliar faces did not significantly differ between a group of adults with high-functioning autism and a group of age- and IQ-matched neurotypical controls. Although it remains possible that low-functioning adults with autism would show continued impairment, these results suggest that early neural correlates of face familiarity processing have become relatively neurotypical for at least some adults with autism. Interestingly, this group of adults did show behavioral impairments in a face recognition battery that required them to identify a set of previously presented faces from related distracters. Investigating the neural correlates of encoding and recognition of initially unfamiliar faces may reveal impairments that are not apparent when examining responses to a highly familiar face with affective significance. In addition, deficits in “executive” processes like selective attention, decision-making, resistance to interference, or forming face-context associations may also make a stronger contribution to behavioral or “real-life” difficulties with face recognition in adulthood than deficits in the systems involved in generating the earliest signals that a face is familiar. Addressing these questions may provide important insight into the deficits that underlie difficulties with face recognition in individuals with autism and may suggest fruitful avenues for intervention.

Emotion Processing

Expressing and interpreting emotions can be challenging for many individuals with autism, but the

neurophysiological disruptions that underlie this difficulty remain unclear. Recording EEG responses to pictures of faces depicting different emotional expressions provides one way to examine the integrity of very early facial emotion-processing systems in autism. In an initial study, Dawson and colleagues studied neural responses to neutral and fearful faces in 3–4-year-old children with autism or typical development (Dawson et al. 2004). While neural responses to fearful and neutral faces differed within 300 ms of stimulus presentation for typically developing children, the group of children with autism showed no significant differences in neural response to the two types of stimulus over any examined component. Interestingly, faster responses to emotional faces at the posterior N300 were correlated with increased social attention in children with autism, suggesting that more efficient processing of facial emotions was related to stronger social skills in this group. Consistent with developmental delay in this area, cognitive potential responses to emotional expressions in older children with autism appear more neurotypical, although the neural sources underlying scalp-recorded activity may be weaker and/or slower than those observed in typically developing children (Wong et al. 2008). Possibly, individuals with autism “catch up” with their neurotypical peers in simple emotion-processing tasks, although impairments may remain in more complex aspects of emotion processing (e.g., integration with gaze, parallel processing with the semantic content of an interaction, or rapid transitions between different emotions). Examining the longitudinal developmental trajectory of neural responses to emotional expressions in children with autism would provide insight into this possibility.

Attention

Although not part of the diagnostic triad, difficulties with attention are common in children on the spectrum and often present significant challenges to caregivers and therapists. One event-related component that is clearly modulated by attention is P300, a well-characterized component that likely reflects neural generators in the temporal,

parietal, and frontal areas. P300 is often examined during “oddball” tasks, in which frequent, infrequent, and “novel” (or distinctive) stimuli are presented in visual, auditory, or somatosensory modalities. Typically, P300 responses are larger to infrequent and novel stimuli because attention is engaged by their unusual or unpredicted nature. Recent work suggests P300 comprises two separable components: the early frontocentral P3a, peaking between 250 and 280 ms after stimulus presentation and related to the engagement of attention and novelty processing, and the later parietal P3b, peaking around 300 ms and more sensitive to stimulus probability. Examining P3a and P3b can thus provide insight into the neurophysiological and cognitive mechanisms that underlie problems with attention in autism. Further, since atypicalities in attention are some of the more commonly medicated associated symptoms of autism, examining responses in P300 paradigms could identify potentially sensitive indices of treatment response.

In one of the earliest studies to find atypical P300 responses to visual stimuli in autism, Ciesielski, Courchesne and Elmasian (1990) tested 14 high-functioning adults with autism and 14 age-matched controls. Participants saw a series of green or red flashes, mixed in with a series of 1- or 2-kHz sounds. Each block contained frequent and rare flashes and sounds, and participants were asked to press a button in response to either the rare sounds (the focused auditory attention condition) or the rare flashes (the focused visual attention condition). Results showed that the group with autism showed significantly diminished P3 responses to the targets in both auditory and visual modalities, and this was also observed in a subgroup of adults with autism whose behavioral performance matched that of the neurotypical control group. Since diminished P300 responses are thought to reflect reduced attention, these results may suggest that the capacity for rapid goal-directed modulation of attention may be atypical in autism. However, there must be significant compensatory mechanisms that enable successful task performance despite atypical early neural responses to the stimuli. Identifying these compensatory mechanisms and their

developmental trajectories may be important in identifying effective intervention strategies for attention problems in autism.

Notably, not all studies have observed atypical visual P300 responses in autism, and when disruptions are observed, they are generally milder than for other sensory modalities. For example, Courchesne, Lincoln, Kilman and Galambos (1985) conducted a visual and auditory target detection task with high-functioning adults and adolescents with autism and chronological age-matched controls. Atypicalities in the P300 response to targets were more clearly apparent for auditory than visual stimuli. In a further example, Kemner, Verbaten, Cuperus, Cafferman and Engeland (1994) used oddball tasks with abstract figures and electromagnetic pulses with age-matched groups of 7- to 13-year-old children with autism, typical development, ADHD, or dyslexia. The group with autism showed *larger* P300 responses to novel visual stimuli, but *diminished* somatosensory P300 responses relative to neurotypical controls (though not relative to the other control groups). Taken together, findings suggest that attention may operate with differential efficiency across sensory modalities in autism. However, it is also possible that variability in adaptive or cognitive skill between participant groups is a critical factor. For example, Salmond, Vargha-Khardem, Gadian, de Haan and Baldewag (2007) studied auditory P300 responses in an oddball task with low- and high-functioning 8- to 20-year-old individuals with autism. While the low-functioning group showed a slower P3a and a diminished P3b response to targets, the high-functioning group did not differ from controls in the latency or amplitude of any component. Further, in a group of 5- to 11-year-old children with autism, Gomot et al. (2010) found that an atypically large P3a to deviant sounds was related to decreased tolerance of change, as reported by trained nurses who cared for the children in a hospital psychiatric day-care unit. These studies provide supporting evidence that attention is related to symptom variability in autism and suggest that interventions targeting domains of attention may have broad effects for individuals with autism.

In addition to measuring the ability to “tune in” to relevant stimuli, P300 can be used to examine the ability to “tune out” stimuli that are not relevant to an ongoing task. In paradigms where participants are asked to respond to targets and ignore nontargets, P300 responses to nontargets are typically diminished. However, Sokhadze et al. (2009) found that relative to neurotypical controls, P3b responses to nontargets were larger and responses to targets were smaller in a group of adolescents and adults with autism. This “flatter” response profile suggests that the group with autism were less able to focus their attention on a selected stimulus type. The authors suggest that early inefficient perceptual filtering may lead to a greater need to actively inhibit nontarget stimuli at a later processing stage. Interestingly, in this study, P300 was examined as part of a suite of outcome measures for testing the efficacy of low-frequency repetitive transcranial magnetic stimulation (rTMS) of dorsolateral prefrontal cortex for symptoms of autism. In individuals with autism who received rTMS, the amplitude of P3a response to nontargets significantly decreased between pre- and post-TMS assessment points, while no changes were observed in the wait-list control group. The authors suggest that TMS treatment may have improved selective attention by strengthening the inhibitory surround of minicolumns in dorsolateral prefrontal cortex. This study illustrates the possible benefits of including event-related potential measures in treatment research, both because of their potential sensitivity to subtle treatment effects and because they may provide greater insight into neurophysiological mechanisms underlying treatment response.

P300 responses can also be used to study spatial attention shifting in autism. Many individuals with autism have difficulty shifting their attention between one object and another or shifting gaze between a person and the object to which they are referring. To understand more about attention shifting in autism, Townsend et al. (2001) examined P300 in nine high-functioning adults with autism and chronological age-matched controls. Adults were presented with an array of five squares and instructed to look at a fixation cross that remained in the center of the screen. On each

trial, one of the squares would change color to indicate the upcoming location of the target stimulus on that trial. Participants were asked to respond to target stimuli, but not to respond to stimuli that appeared in noncued locations. Because participants had to pay attention to the cued box while looking at the central stimulus, this is a test of covert attention shifting. EEG results showed that the frontal P3a response to peripheral stimuli was delayed or absent in individuals with autism, and behavioral results also showed lower detection accuracy for peripheral versus central stimuli in the autism group only. Since P3a is typically associated with attention engagement, the atypical P3a response may indicate that the covert spatial orienting system operates less efficiently in autism. Further, a range of evidence suggests that the integrity of the cerebellum can modulate the cortical activity that contributes to the frontal P3a. Townsend and colleagues thus suggest that impairments in attention shifting in individuals with autism may be related to the influence of cerebellar pathology on frontal and parietal spatial systems. In addition to providing important insight into attention impairments in autism, this study illustrates how cognitive potentials allow researchers to begin to investigate the neuropathological and neurophysiological correlates of behavioral challenges in autism.

Goal-Directed Learning

Individuals with autism often have difficulty with cognitive skills required for flexible, goal-directed behavior like inhibition, planning, and regulatory control. These “executive functions” also appear to be related to variability in adaptive functioning and sociocommunicative development in individuals with autism (e.g., Munson et al. 2008) and are thus important potential predictors of developmental outcome. One executive skill critical to goal-directed learning is the ability to modify behavior based on internally or externally generated error signals, and cognitive potentials can provide insight into the integrity of those signals in autism. For example, making an error is associated with the frontal error-related negativity (ERN), which occurs within 500 ms of the

participant’s response and is thought to reflect internal error monitoring. Examining the integrity of ERN thus provides insight into the integrity of internally generated error signals in autism.

In an initial study, Henderson et al. (2006) found that high-functioning 11-year-old children with autism showed a larger ERN than IQ-matched controls or lower-functioning children with autism. Since a larger ERN would be typically associated with more effective error monitoring, these findings might suggest that children with autism require particularly strong executive functioning skills to develop language. However, several subsequent studies have obtained different results. For example, Vlamings, Jonkman, Hoeksma, van Engeland and Kemner (2008) found that 10-year-old children with autism had a smaller ERN than typically developing controls and that this was not associated with IQ. Source analysis suggested that the reduced ERN reflected diminished brain activity in the anterior cingulate, suggesting that disruptions in error processing may be related to pathology in this brain region. Similarly, South, Larson, Krauskopf and Clawson (2010) also observed a reduced ERN in a group of 8- to 18-year-olds with autism; correlations were observed with parent reports of social behaviors, suggesting relations between error processing and social symptoms in autism. Tying together these findings, Santesso et al. (2010) found a reduced ERN in high-functioning adults with autism, linked this to reduced activity in the anterior cingulate, and observed relations between increased anterior cingulate activity and better social skills. Taken together, the weight of evidence suggests that individuals with autism may have diminished activity in the anterior cingulate gyrus and a corresponding difficulty with response monitoring and error awareness that may be related to their degree of social impairment.

If individuals with autism have difficulty generating internal error signals, might external feedback be particularly important to learning? The presentation of external feedback is associated with a negative-going deflection called feedback-related negativity (FRN) that is typically more negative to losses or unexpected

outcomes than gains or expected outcomes. A recent study of children and adolescents with autism observed relatively neurotypical FRNs to concrete rewards (Larson et al. 2010), despite the diminished ERN that was observed in a substantially overlapping group of children (reported in South et al. 2010). Larson et al. (2010) suggest that the response to internally generated error signals may be more impaired than response to concrete, direct feedback in autism. Although more work is required to consolidate these findings, if children with autism are more able to benefit from external feedback than internally generated error signals, there may be important implications for the design of intervention programs.

Movement-Related Potentials

The presence of stereotyped and repetitive motor mannerisms is currently one of the diagnostic criteria for autism. Although there are many hypotheses as to the sources of such behaviors, diminished volitional control of movement could contribute to restricted movement patterns. The movement-related potential (MRP) or Bereitschaftspotential is a negative-going deflection observed when movements are internally generated. The MRP typically peaks when the movement is made and is maximal over central electrodes. Studying the MRP may provide insight into volitional control of movement in autism.

Two related studies have examined the MRP in groups of adults with autism or neurotypical development. Rinehart et al. (2006) used a paradigm in which participants had to press illuminated buttons until the light turned off, such that the duration of each button press was full cued. In this task, the group of adults with high-functioning autism showed a slower MRP offset than the other groups, suggesting that activity associated with movement preparation took longer to dissipate after the movement was generated. In a follow-up study, Enticott, Bradshaw, Iansek, Tonge and Rinehart (2009) modified the paradigm to decrease the provision of external cues for movement generation; participants were asked to press a button at set intervals, with no

external cues to which button to press or when. In this version of the task, the MRP peaked significantly earlier in adults with high-functioning autism than those with Asperger's syndrome and neurotypical adults, again consistent with disruptions in movement preparation. Difficulties with movement preparation may contribute to difficulty with sport or other activities that require fine-grained movement control and could possibly contribute to decreased movement generativity in autism. Studying relations between the MRP and restrictive and repetitive behaviors would be an important next step in this area.

Future Directions

Developments in technology continually afford new research questions in the field of cognitive potentials. The advent of high-density electrode arrays and more powerful source analysis techniques will enable researchers to ask more detailed questions about the neural sources underlying scalp-recorded potentials. However, it is important that the field strives to be representative of the full autism spectrum. The development of low-profile rapid application electrode arrays facilitates the use of EEG with infants, young children, and individuals with autism with more limited adaptive skills. Nonetheless, appropriate use of EEG with these populations requires extensive practice, experience, and training, and developing and disseminating gold-standard procedures for collecting and analyzing data from these relatively understudied groups is an important future goal.

Examining the relation between cognitive potentials and genetic variation is also an important goal for the field. Understanding how particular neurophysiological or cognitive processes relate to genetic risk markers in children with autism or their family members will help us evaluate whether cognitive potentials could provide valuable endophenotypes of autism. There is promising evidence that family members of individuals with autism may show disrupted event-related responses to social stimuli that resemble

those observed in some individuals with autism (Dawson et al. 2005), and examining these responses in relation to particular genetic markers is an important next step.

A further important direction is the integration of cognitive potentials into treatment studies. Many treatment studies currently use global indicators that are not specific to the neural systems that are the hypothesized target of the drug. Furthermore, many more global outcome measures may not be sensitive to subtle changes that occur soon after the drug regime begins and, if reliant on parent report, may suffer from inflated placebo effects. Measuring cognitive potentials may provide a more sensitive, specific, and objective complementary means of assessing response to treatment. Treatment-related ERP changes have been observed with pharmaceutical compounds (Verbaten et al. 1996) and transcranial magnetic stimulation (Sokhadze et al. 2009), indicating that this is a promising approach. One current limitation to the use of EEG in treatment studies is the lack of a standardized “battery” of tasks designed to load on particular cognitive systems; developing such a battery would be an important step forward.

Finally, understanding the developmental emergence of symptoms of autism is a critical step toward identifying critical periods and potential targets for intervention. EEG is a technique that can be used across the lifespan and is therefore ideally suited to longitudinal or cross-sectional investigations of neurophysiological development in autism. However, there are currently very few studies of under-fives with autism. The current wave of infant sibling studies will provide more information about very early development in children at risk for developing autism, and several labs are examining event-related potentials in this population. These studies will provide valuable information about the early emergence of neurophysiological patterns observed in older children and adults and enable questions about their developmental origins. Understanding the neurodevelopmental trajectories that result in an autism diagnosis is critical to developing effective systems for early detection and early intervention, key steps toward

improving the lives of individuals with autism and their families.

See Also

- Attention
- Electroencephalography
- Error-Related Negativity
- Event-Related Potential
- Evoked Potentials
- Executive Function (EF)
- Face Recognition
- Neurophysiology
- Visual Evoked Potential (VEP)

References and Reading

- Brazier, M. A. B. (1961). *A history of the electrical activity of the brain; The first half-century*. New York: Macmillan.
- Ciesielski, K. T., Courchesne, E., & Elmasian, R. (1990). Effects of focused selective attention tasks on event-related potentials in autistic and normal individuals. *Electroencephalography and Clinical Neurophysiology*, 75, 207–220.
- Courchesne, E., Lincoln, A. J., Kilman, B. A., & Galambos, R. (1985). Event-related brain potential correlates of the processing of novel visual and auditory information in autism. *Journal of Autism and Developmental Disorders*, 15, 55–76.
- Dawson, G., Carver, L., Meltzoff, A. N., Panagiotides, H., McPartland, J., & Webb, S. J. (2002). Neural correlates of face and object recognition in young children with autism spectrum disorder, developmental delay, and typical development. *Child Development*, 73, 700–717.
- Dawson, G., Webb, S., Carver, L., Panagiotides, H., & McPartland, J. (2004). Young children with autism show atypical brain responses to fearful versus neutral facial expressions of emotion. *Developmental Science*, 7, 340–349.
- Dawson, G., Webb, S. J., Wijsman, E., Schellenberg, G., Estes, A., Munson, J., et al. (2005). Neurocognitive and electrophysiological evidence of altered face processing in parents of children with autism: Implications for a model of abnormal development of social brain circuitry in autism. *Development and Psychopathology*, 17, 679–697.
- Enticott, P. G., Bradshaw, J. L., Iansek, R., Tonge, B. J., & Rinehard, N. J. (2009). Electrophysiological signs of supplementary-motor-area deficits in high-functioning autism but not Asperger syndrome: An examination of internally cued movement-related potentials. *Developmental Medicine and Child Neurology*, 51, 787–791.

- Gomot, M., Blanc, R., Clery, H., Sylvie, R., Barthelemy, C., & Bruneau, N. (2010). Candidate electrophysiological endophenotypes of hyper-reactivity to change in autism. *Journal of Autism and Developmental Disorders*. <https://doi.org/10.1007/s10803-010-1091-y>.
- Henderson, H., Schwartz, C., Mundy, P., Burnette, C., Sutton, S., Zahka, N., et al. (2006). Response monitoring, the error-related negativity, and differences in social behavior in autism. *Brain and Cognition*, 61, 96–109.
- Kemner, C., Verbaten, M. N., Cuperus, J. M., Cafferman, G., & Engeland, V. (1994). Visual and somatosensory event-related brain potentials in autistic children and three different control groups. *Electroencephalography and Clinical Neurophysiology*, 92, 225–237.
- Larson, M. J., South, M., Krauskopf, E., Clawson, A., & Crowley, M. J. (2010). Feedback and reward processing in high-functioning autism. *Psychiatry Research*. <https://doi.org/10.1016/j.psychres.2010.11.006>.
- Munson, J., Faja, S., Meltzoff, A., Abbott, R., & Dawson, G. (2008). Neurocognitive predictors of social and communicative developmental trajectories in pre-schoolers with autism spectrum disorders. *Journal of the International Neuropsychological Society*, 14, 956–966.
- Rinehart, N. J., Tonge, B. J., Bradshaw, J. L., Iansek, R., Enticott, P. G., & Johnson, K. A. (2006). Movement-related potentials in high-functioning autism and Asperger disorder. *Developmental Medicine and Child Neurology*, 48, 272–277.
- Salmond, C. H., Vargha-Khadem, F., Gadian, D. G., de Haan, M., & Baldewag, T. (2007). Heterogeneity in the patterns of neural abnormality in autistic spectrum disorders: Evidence from ERP and MRI. *Cortex*, 43, 686–699.
- Santesso, D. L., Drmic, I. E., Jetha, M. K., Bryson, S. E., Goldberg, J. O., & Hall, G. B. (2010). An event-related source localization study of response monitoring and social impairments in autism spectrum disorder. *Psychophysiology*, 48, 241–251.
- Sokhadze, E. M., El-Baz, A., Baruth, J., Mathai, G., Sears, L., & Casanova, M. F. (2009). Effects of low frequency repetitive transcranial magnetic stimulation (rTMS) on gamma frequency oscillations and event-related potentials during processing of illusory figures in autism. *Journal of Autism and Developmental Disorders*, 39, 619–634.
- South, M., Larson, M. J., Krauskopf, E., & Clawson, A. (2010). Error processing in high-functioning autism spectrum disorders. *Biological Psychology*, 85, 242–251.
- Townsend, J., Westerfield, M., Leaver, E., Makeig, S., Jung, T.-P., & Pierce, K. (2001). Event-related brain response abnormalities in autism: Evidence for impaired cerebello-frontal spatial attention networks. *Cognitive Brain Research*, 11, 127–145.
- Verbaten, M. N., Kemner, C., Buitelaar, J. K., van Ree, J. M., van Beijsterveld, C. E. M., & van Engeland, H. (1996). Effects of ORG-2766 on brain event-related potentials of autistic children. *Psychiatry Research*, 63, 33–45.
- Vlamings, P. H. J. M., Jonkman, L. M., Hoeksma, M. R., van Engeland, H., & Kemner, C. (2008). Reduced error monitoring in children with autism spectrum disorder: An ERP study. *European Journal of Neuroscience*, 28, 399–406.
- Webb, S. J., Jones, E. J. H., Merkle, K., Oskin, N., Murias, M., & Greenson, J. (2010). Response to familiar faces, newly familiar faces, and novel faces as assessed by ERPs is intact in adults with autism. *International Journal of Psychophysiology*, 77, 106–117.
- Webb, S. J., Jones, E. J. H., Merkle, K., Venema, K., Greenson, J., & Murias, M. (2011). Developmental change in the ERP responses to familiar faces in toddlers with autism spectrum disorders versus typical development. *Child Development*, 82(6), 1868–1886.
- Wong, T. K., Fung, P. C., Chua, S. E., & McAlonan, G. M. (2008). Abnormal spatiotemporal processing of emotional facial expressions in childhood autism: Dipole source analysis of event-related potential. *European Journal of Neuroscience*, 28, 407–416.

Visually Organized Learning Environments

► Structured Classrooms

Visual-Motor Ability

► Visual-Motor Function

Visual-Motor Function

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Synonyms

Visual-motor ability; Visual-motor integration;
Visual-motor skills; Visuomotor ability;
Visuomotor function; Visuomotor skills

Definition

Visual-motor function is the integration between visual perception and motor skills. More specifically, visual-motor function is the ability to draw or copy forms or to perform constructive tasks integrating both visual perception and motor skills. Visual-motor function involves the ability to coordinate vision with the movements of the body. Due to the body, head, eyes, and limbs being constantly in motion, every time an individual wants to carry out an action, calculations and decisions about orientation, motion, and location need to be made. The parietal cortex is the part of the brain that is responsible for processing and integrating somatosensory, visual, and auditory information and plays an important role in producing planned movements. The cerebellum, brainstem, and frontal lobe are also involved in visual-motor abilities. Visual-motor function deficits can lead to learning disabilities, neuropsychological deficits, and behavioral problems. For example, deficits in visual-motor function may lead to problems with fine motor tasks that rely heavily on visual feedback such as writing, drawing, painting, building structures with blocks, stringing beads, repairing things, and playing games. Visual agnosia and apraxia are other disorders associated with visual-motor function deficits. The presence of visual, sensory, or motor deficits is not specific to but is very common in autism. The Bender Visual-Motor Gestalt Test, Second Edition (BG-II), Beery-Buktenica Developmental Test of Visual-Motor Integration (Beery VMI), and the Wide Range Assessment of Visual-Motor Abilities (WRAVMA) are common psychological assessments that assess visual-motor function.

See Also

- ▶ [Apraxia](#)
- ▶ [Beery-Buktenica Developmental Test of Visual-Motor Integration](#)
- ▶ [Peabody Developmental Motor Scales \(PDMS\)](#)
- ▶ [Sensory Impairment in Autism](#)
- ▶ [Wide-Range Assessment of Visual-Motor Abilities](#)

References and Reading

- Dawson, G. (1996). Neuropsychology of autism: A report on the state of the science. *Journal of Autism and Developmental Disorders*, 26, 179–184.
- Dawson, G., & Watling, R. (2000). Interventions to facilitate auditory, visual, and motor integration in autism: A review of the evidence. *Journal of Autism and Developmental Disorders*, 30, 415–421.
- Kolb, B., & Whishaw, I. Q. (2009). The parietal lobes. In B. Kolb & I. Q. Whishaw (Eds.), *Fundamentals of human neuropsychology* (6th ed., pp. 376–401). New York: Worth.
- Lopata, C., Hamm, E. M., Volker, M. A., & Sowinski, J. E. (2007). Motor and visuomotor skills of children with Asperger's disorder: Preliminary findings. *Perceptual and Motor Skills*, 104, 1183–1192.
- Volker, M. A., Lopata, C., Vujnovic, R. K., Smerbeck, A. M., Toomey, J. A., Rodgers, J. D., et al. (2010). Comparison of the Bender Gestalt-II and VMI-V in samples of typical children and children with high-functioning autism spectrum disorders. *Journal of Psychoeducational Assessment*, 29, 187–200.

Visual-Motor Integration

- ▶ [Visual-Motor Function](#)
 - ▶ [Visual-Motor Integration, Developmental \(VMI\) Test](#)
-

Visual-Motor Integration, Developmental (VMI) Test

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Abbreviations

AS	Asperger's syndrome
ASD	Autism spectrum disorder
Beery	Beery-Buktenica Developmental
VMI	Test of Visual-Motor Integration, sixth edition

BG-II	Bender Visual-Motor Gestalt Test second edition
DTVP-2	Developmental Test of Visual Perception – second edition
FRTVMI	Full Range Test of Visual Motor integration
HFASD	High-functioning autism spectrum disorders
MCT	Beery VMI Motor Coordination Test (MCT)
PDD	Pervasive developmental disorders
RtI	Response to intervention
VPT	Beery VMI Visual Perception Test
WISC-R	Revised Wechsler Intelligence Scale for Children

Synonyms

Beery VMI; Beery VMI motor coordination test; Beery VMI visual perception test; Beery-Buktenica developmental test of visual-motor integration; MCT; Visual-motor integration; VMI; VPT; Wide-range assessment of visual-motor abilities; WRAYMA

Description

The *Beery-Buktenica Developmental Test of Visual-Motor Integration* (Beery VMI) is a visual-motor screening tool used to identify children, adolescents, adults, and seniors, aged 2–100 years, who are experiencing difficulty coordinating visual perception and motor (finger and hand) movements (Beery and Beery 2010). The Beery VMI consists of 30 geometric designs of increasing complexity that participants are asked to copy. The specific Beery VMI items are listed in Table 1. It can be administered to individuals or groups and takes about 10 min to complete. The 30-item Full Form can be used with all age groups, 2–100 years, while the 21-item Short Form can be used with children ages 2 through 7 years.

Two additional standardized tests, the *Beery VMI Visual Perception Test* (VPT) and the *Beery VMI Motor Coordination Test* (MCT) are

Visual-Motor Integration, Developmental (VMI) Test, Table 1 Item descriptions for the *Developmental Test of Visual-Motor Integration Sixth Edition*^a

Item number	Item description	Age norm (years: months)
1 ^b	Imitated mark or scribble	1:1
2 ^b	Spontaneous scribble	1:4
3 ^b	Contained scribble	1:9
4	Vertical line (imitated copy of design)	2:0
5	Horizontal line (imitated copy of design)	2:6
6	Circle (imitated copy of design)	2:9
7	Vertical line (copy design that is already present)	2:10
8	Horizontal line (copy design that is already present)	3:0
9	Circle (copy design that is already present)	3:0
10	Vertical-horizontal cross	4:1
11	Right oblique line	4:4
12	Square	4:6
13	Left oblique line	4:7
14	Oblique cross	4:11
15	Triangle	5:3
16	Open square and circle	5:6
17	Three-line cross	5:9
18	Directional arrows	6:5
19	Two-dimensional rings	6:9
20	Six-circle triangle	7:5
21	Circle and tilted square	7:11
22	Vertical diamond	8:1
23	Tilted triangles	8:11
24	Eight-dot circle	9:6
25	Wertheimer's hexagons	10:11
26	Horizontal diamond	11:2
27	Three-dimensional rings	12:6
28	Necker cube	12:8
29	Tapered box	13:2
30	Three-dimensional star	13:8

^aBeery and Beery (2010)

^bBeery VMI items 1, 2, and 3 do not need to be administered if a child is able to copy one or more of items 4, 5, or 6. The Beery VMI Short Form consists of items 1–21 and is designed for use with children aged 2–7 years. The Beery VMI Full Form contains all 30 items and can be used with participants aged 2–100 years

also available for child and adult participants to complete, and this facilitates the comparison of an individual's separate visual and motor performance skills. The VPT requires examinees to identify a target design among choices, and the MCT requires examinees to trace geometric shapes with a dashed outline using a pencil without an eraser. Each supplemental test takes about 5 min to complete. "The two tests use the same stimulus forms as the Beery VMI, unlike other visual-motor test batteries that mistakenly compare less related stimuli and tasks" (Beery and Beery 2010, p. 2). Although administration of the VPT and MCT are not necessary, it is suggested that if they are used, the Beery VMI should be administered first, followed by the VPT and then the MCT.

The Beery VMI is used by psychologists, educators, and allied health professionals to assess children and adults with presenting known or suspected developmental, behavioral, learning, neurological, or psychological difficulties including individuals with autism spectrum disorder (ASD) (Fulkerson and Freeman 1980; Lopata et al. 2007; Novales 2006; Volker et al. 2010). Individuals administering the test must have Examiner B qualifications, which includes a graduate degree in a related field (such as psychology, occupational therapy, education, speech therapy, physiotherapy, or health sciences) or equivalent training to complete the assessment. The Beery VMI is a paper and pencil test that must be hand-scored by the examiner.

The scoring sheet is included as part of the Beery VMI test booklet. Copied shapes and designs receive either a score of "1" for being correct or "0" (no-score) for items not copied correctly (Beery and Beery 2010). Specific guidelines regarding scoring are provided in the manual including pictorial examples as well as specific written scoring criteria for each geometric form. Accurate scoring requires the use of a protractor to make judgments about accuracy of angles, etc. The administration of the Beery VMI, VPT, and MCT items and scoring is stopped after three consecutive forms have received a "no-score." The raw score consists of the total number of correct forms until the ceiling score is reached

(e.g., three consecutive wrong items). The raw score is converted to the standard score using the conversion tables provided and then the standard score is used to calculate the percentile rank for the child's specific age range. Tables for each of these scores are provided in the test manual. The test manual also contains tables to convert raw scores into age equivalents.

Four supplemental materials are available in addition to the Beery VMI that can be used to enhance the teaching of visual-motor integration skills to children include the following:

- The *Beery VMI Stepping Stones Parent Checklist* that includes about 200 of the "Stepping Stones" milestones that a parent can observe and note on a check list (Beery and Beery 2010).
- The *Beery VMI Developmental Teaching Activities* that contains about 250 activities geared to assist children from birth to age 6 develop skills to promote skill development (Beery and Beery 2010).
- The *Beery VMI My Book of Shapes* that provides 100 activities that may help with the learning of letter and numeral shapes (Beery and Beery 2010).
- The *Beery VMI My Book of Letters and Numbers* that extends upon the *My Book of Shapes* activities and provides 100 additional exercises designed to provide more practice with numerals and upper- and lower-case letters (Beery and Beery 2010).

Historical Background

The Beery VMI, consisting of 24 items, was first published by Beery in 1967 and has retained its original forms, characteristics, and strengths. Geometric forms, rather than letters or numbers, were selected as the basis for the test's items, to minimize the impact of culture and/or education. "The Beery VMI was standardized six times between 1964 and 2010 with a total of more than 13,000 children. It was also nationally standardized in 2006 with 1,021 adults" (Beery and Beery 2010, p. 1). In the 1997 version, two supplemental

standardized tests – the *Beery VMI Visual Perception Test* (VPT) and *Beery VMI Motor Coordination Test* (MCT) – were added which allowed users to statistically compare an individual’s visual and motor contributions to visual-motor integration. The Beery VMI was revised in 2004 consisting of 30 geometric forms and included an extended age range for its norms down to 2 years of age (Beery and Berry 2004). As well, the authors identified 600 “Stepping Stones” or milestones that may serve as precursors for visual-motor integration for children from birth through to age 6. The Stepping Stones have been listed in both the Parent Checklist and in the Beery VMI test manual as a reference guide. Developmental Teaching Activities based on the Stepping Stones were also developed for this edition. During the 2006 revision of the Beery VMI, adult norms were added to include participants aged 19–99 years, 11 months. “The Beery VMI and its supplemental *Visual Perception* and *Motor Coordination* tests were renormed in late 2009 and 2010 on children obtained from the four major census regions in the United States” (Beery and Beery 2010, p. 100).

Psychometric Data

A wealth of psychometric data has been reported for the Beery VMI during its six revisions. The information reported here focuses on the most recent reliability and validity data reported in the sixth edition of the Beery VMI manual published in 2010. Types of reliability data often reported includes internal consistency, test-retest/time sampling reliability, inter-rater/inter-scorer reliability, and intra-rater reliability. For participants aged 2–17, the Cronbach alpha coefficients for the Beery VMI ranged from 0.79 to 0.89 with a mean of 0.82, for the VPT ranged from 0.74 to 0.87 with a mean of 0.81, and for the MCT ranged from 0.71 to 0.89 with a mean of 0.82. For the adult group aged 19–100 years, the Cronbach alpha coefficients for the Beery VMI ranged from 0.85 to 0.94 with a mean of 0.89, for the VPT ranged from 0.83 to 0.93 with a mean of 0.88, and for the MCT ranged from 0.84 to 0.89 with a mean of 0.86 (Beery and Beery 2010). The

Standard Errors of Measurement (SEMs) for the Beery VMI, VPT, and MCT designed for children ages 2–17 were 5.5, 6, and 6, respectively (Beery and Beery 2010). The SEMs for the Beery VMI, VPT, and MCT for adults ages 18–100 years were 5, 5, and 6, respectively (Beery and Beery 2010).

Related to test-retest reliability, the Beery VMI sixth edition, VPT, and MCT were administered to 142 children between the ages of 5 and 12 years presenting with a range of abilities. “The time between initial administration and the retest averaged 14 days. The overall test-retest coefficients were .88 for the Beery VMI, .84 for Visual Perception, and .85 for Motor Coordination” (Beery and Beery 2010, p. 107). In the Beery VMI manual, 1-week test-retest reliability coefficients for a sample of 20 adult participants aged 60–69 years were reported as 0.88, 0.86, and 0.84 for the Beery VMI, VPT, and MCT, respectively (Beery and Beery 2010).

For inter-rater reliability, the Beery VMI Manual reported the results of two individuals who independently scored 100 Beery VMI, VPT, and MCT forms randomly selected from the sample of the children’s norming group. “The resulting interscorer reliabilities were .93 for the Beery VMI, .98 for Visual Perception, and .94 for Motor Coordination” (Beery and Beery 2010, p. 108). Similarly, based on a randomly selected sample of 25 adults from the norming sample, inter-rater reliability coefficients of 0.94, 0.97, and 0.92 were obtained from the Beery VMI, VPT, and MCTs, respectively (Beery and Beery 2010). No information on intra-rater reliability was reported in the Beery VMI manual for child or adult versions of the Beery VMI, VPT, and MCT.

Types of validity often reported for tests include content validity, criterion-related reliability, and construct validity. Two subtypes of criterion-related reliability frequently included are concurrent validity and predictive validity while subtypes of construct validity often reported include factor analysis validity, discriminant validity, convergent validity, divergent validity, and rating scale validity. Content validity was established for the Beery VMI, VPT, and MCT through an extensive literature review and based on clinical experience of the author. Concurrent

validity evidence was established by correlating the Beery VMI with the *Copying* subtest of the *Developmental Test of Visual Perception – 2nd edition* (DTVP-2) and the *Drawing* subtest of the *Wide Range Assessment of Visual-Motor Abilities* (WRAVMA). The correlation between the Beery VMI and the DTVP-2 Copying subtest was 0.75 and 0.52 with the WRAVMA *Drawing* subtest. A correlation of 0.62 was obtained between the VPT and the DTVP-2 *Position-in-Space* subtest while a correlation of 0.65 was obtained between the MCT and the DTVP-2 *Eye-Coordination* subtest (Beery and Beery 2010). “These results generally support the validity of the Beery VMI and its supplemental tests, although correlations are only moderately high between the Beery VMI and the newer, less well-developed geometric form-copying tests [the DVTP-2 and the WRAMA]” (Beery and Beery 2010, p. 111).

Predictive validity evidence of the Beery VMI has also been reported with visual-motor integration (VMI) skills being found to be effective predictors of reading difficulties, letter identification, reading readiness, school-related problems, school achievement, and school grade failures and retentions in children (Goldstein and Britt 1994; Pianta and McCoy 1997). “Generally, researchers have found the Beery VMI to be a valuable predictor, particularly when used in combination with other measures” (Beery and Beery 2010, p. 121).

Extensive construct validity information is reported in the Beery VMI manual in the form of seven hypotheses. Firstly, the skills measured by the Beery VMI, VPT, and MCT are related to chronological age. The Pearson correlation coefficients between the Beery VMI, VPT, and MCT and the ages of the children’s total norming sample were 0.89, 0.85, and 0.84, respectively. Secondly, it was hypothesized that the abilities measured by the Beery VMI, VPT, and MCT would at least be moderately correlated with one another. The correlation between the Beery VMI and VPT was 0.36, between the Beery VMI and the MCT was 0.30, and between the VPT and the MCT was 0.46 (Beery and Beery 2010). Thirdly, the authors provided evidence that the VPT and

MCT each measure a part, but not the entirety, of what the Beery VMI measures. In other words, the “Beery VMI whole is greater than the sum of its parts [the VPT and MCT]” (Beery and Beery 2010, p. 115).

Fourthly, it was proposed that the Beery VMI, VPT, and MCT would be related to the nonverbal aspects of intelligence. The manual reports correlations between the *Revised Wechsler Intelligence Scale for Children* (WISC-R) and the Beery VMI, VPT, and MCT for a sample of 17 children between the ages of 6 and 12 years. The correlations between the WISC-R Verbal IQ and the Beery VMI, VPT, and MCT were 0.48, 0.43, and 0.41; between the WISC-R Performance IQ and the Beery tests were 0.66, 0.58, and 0.55; and between the WISC-R Full IQ and the Beery tests were 0.62, 0.54, and 0.51, respectively (Beery and Beery 2010). The fifth proposition was that the Beery VMI, VPT, and MCT would be related to academic achievement. In the manual correlations between the *Comprehensive Test of Basic Skills* and the three Beery tests were reported as 0.63, 0.29, and 0.40 (Beery and Beery 2010). The sixth hypothesis stated that the Beery VMI, VPT, and MCT measure similar traits and are effective in measuring persons. Using the Rasch Measurement Model, evidence of high item and person separation indices were reported thus confirming this hypothesis.

The seventh hypothesis proposed that the Beery VMI, VPT, and MCT would be sensitive to certain disabling conditions with groups of participants with known diagnoses would obtain lower test scores. A multitude of studies have been published documenting that the Beery tests are able to differentiate groups with a variety of clinical conditions including learning disabilities, dyspraxia, dysgraphia, mental retardation, visual impairment, ASD, spina bifida, fetal alcohol syndrome, leukemia, Tourette syndrome, cerebrovascular accident, traumatic brain injury, Parkinson’s disease, amyotrophic lateral sclerosis, multiple sclerosis, dementia, and Guillain-Barre Syndrome to name a few.

The Beery VMI has five main weaknesses in relation to its psychometric properties. Firstly, no intra-rater reliability data is reported in the test

manual. Secondly, with the extension of the Beery VMI to include 2-year olds as well as adults aged 19–100 years, it is not clear whether the current version adequately measures VMI in these two participant age groups since much of the reliability and validity data reported in the test manual are based on studies that used earlier editions of the Beery VMI (Graham 2007). Thirdly, limited construct validity information about the VPT and the MCT are reported in the test manual. Fourthly, the Beery VMI manual cites a number of studies as evidence of its predictive validity; however, few of them are less than 5-years old. The rest of the cited studies are quite dated being 10–25+ years old (McKnight and Chandler 2007). Finally, the Beery VMI manual does not report any validity information related to the consequences of its use, either positive or negative (American Educational Research Association, American Psychological Association and National Council on Measurement in Education 1999).

In a recent study, Lim et al. (2015) examined the cross-cultural validity of the Beery VMI by establishing whether there was a difference in children's visual-motor integration performance between Singaporean and American preschoolers as well as if there were any differences in the visual-integration skills of three ethnic groups residing in Singapore: Chinese, Malay, and Indian preschoolers. This would provide insights about the use and applicability of the Beery VMI in cross-cultural contexts like Singapore. The Beery VMI 5th edition was completed by 385 preschoolers (mean age = 63.3 months) from randomly selected schools in Singapore. The Beery VMI scores of the Singaporean preschoolers were compared with the standardization norms from the United States. The results indicated that there were statistically significant differences between Singaporean and American preschoolers, Chinese and Malay preschoolers from Singapore, and Chinese and Indians preschoolers in Singapore. Lim et al. (2015) concluded that preschool-age children "from different cultural and ethnic groups had different VMI performance. Certain cultural beliefs and practices may affect VMI performance" (p. 213).

Brown et al. (2011) investigated the convergent validity between the Beery VMI and the Full Range Test of Visual Motor Integration (FRTVMI) amongst three groups of participants: 73 children aged 5–10 years (mean age 7.5 years, SD = 2.20), 19 adolescents aged 11–17 years (13.1 years, SD = 2.16), and 61 adults (mean age 31.82 years, SD = 11.20). The Beery VMI and FRTVMI scores for the three participant age groups were all significantly correlated. "Overall, the DTVM and the FRTVMI exhibited large levels of convergent validity with each other, indicating that the two tests appear to measure similar visual-motor integration constructs" (Brown et al. 2011, p. 295).

Using the Rasch model, Brown et al. (2009a, b) inspected the construct validity of the Beery VMI 5th edition. The study involved 400 typically developing children from Victoria, Australia, aged 5–12 years of age. The findings indicated that none of the Beery VMI items "exhibited RMM misfit due to goodness-of-fit mean square (MnSq) infit statistics and standardised z (ZStd) scores being outside the specified acceptable range" (Brown et al. 2009, p. 393). However, Beery VMI item nine demonstrated differential item functioning based on gender. Several of the Beery VMI items were found to have similar levels of difficulty. For example, VMI items 26, 27, and 29; items 18, 22, and 24; and items 4, 5, and 11, were found to have the same level of challenge and this may indicate that some of the items may be redundant. Similarly, the Beery VMI scale item logit measure order did not correspond to that presented in the Beery VMI manual. "Theoretically, the VMI items are developmentally ordered; however, this ordering was not mirrored by the item logit difficulty scores obtained" (Brown et al. 2009, p. 393). In a second study involving the same group of participants, Brown et al. (2009) explored the factor structure of the Beery VMI. The factor analysis results indicated that the Beery VMI generated six viable factors thus demonstrating multidimensionality rather than one overall visual-motor integration factor, in other words, unidimensionality.

Wuang and Su (2009) investigated the measurement properties of the Beery VMI using

Rasch analysis. A sample of 454 children with an intellectual disability aged 4–12 years of age completed the Beery VMI. The following features were examined: unidimensionality, item fit to the model, differential item functioning, item targeting, and discriminative validity. Wang and Su (2009) generated a nine-item version of the Beery VMI that was considered valid and reliable for use with children presenting with an intellectual disability instead of the current 30 item version. However, changes to the Beery VMI items can only be made by the test's authors and the copyright holders.

One of the primary strengths of the Beery VMI is its long history of psychometric validation and reliability studies starting in 1967. It is also easy to use, provides detailed scoring guidelines, and has been standardized on a large sample group. It is widely used internationally and can be administered, scored, and interpreted by a number of health and education professionals. This adds to the versatility and applicability of the Beery VMI as a clinical assessment and research measure. However, one limitation noted by McCrimmon et al. (2012) about the most recent edition of the Beery VMI relates to its “lack of current validity and reliability research. Although the newest edition is based on a strong foundation, much of the psychometric data reported in the manual pertain to previous versions” (p. 592). Therefore it would appear that more current psychometric studies need to be completed. It is recommended that the Beery VMI would be a useful, informative, and appropriate assessment to use with children and adolescents presenting with ASD.

Clinical Uses

“The Beery VMI is designed to assess the extent to which individuals can integrate their visual and motor abilities (eye-hand coordination)” (Beery and Beery 2010, p. 1). The Beery VMI, VPT, and MCT can be used for a variety of clinical purposes in a variety of settings with a variety of client groups. As well, the Beery tests can be used on their own or as part of a larger battery of tests. It is often used by psychologists, occupational

therapists, educators, neurologists, pediatricians, speech/language pathologists, physiotherapists, optometrists, rehabilitation medicine physicians, low-vision specialists, and others to assess children and adults. The Beery VMI, VPT, and MCT can be used in acute care hospitals, rehabilitation centers, geriatric care centers, community health centers, private practice settings, early intervention centers, and primary and secondary school settings. The Beery tests have also been widely used to assess the VMI skills of variety of diagnostic groups including those with ASD (see section above). The Beery VMI provides standard scores for children aged 2-years old, which is rare among psychological assessments. In addition, the Beery VMI is designed to be sensitive to developmental change making it useful in testing the effectiveness of interventions and within a Response to Intervention (RtI) framework.

The Beery VMI is often included as part of a comprehensive neuropsychological test battery or as part of a psychoeducational assessment battery completed by psychologists. Educators, psychologists, and occupational therapists often use the Beery tests when evaluating or screening preschool-age and school-age children presenting with known or suspected developmental delays or learning disabilities. For example, the Beery VMI is frequently utilized in early intervention settings by therapists or by kindergarten teachers to screen children's VMI skills (Dankert et al. 2003). Occupational therapists will often use the Beery VMI, VPT, and MCT to assess children referred to them presenting with sensory-motor problem or printing/writing difficulties (Case-Smith 2002). Optometrists frequently use the Beery tests as part of an initial assessment that includes perceptual and VMI skills (Sorter and Kulp 2003a). Psychologists and occupational therapists might use the Beery tests in rehabilitation settings to assess the VMI abilities of adults who have sustained a traumatic brain injury or older adults who have had a stroke (Hall et al. 1996). “Through early identification, it is hoped that further difficulties can be prevented or remediated by appropriate educational, medical, or other interventions” (Beery and Beery 2010, p. 10).

The clinical purposes the Beery VMI, VPT, and MCT could be used for are establishing the baseline of a client's VMI skills (often done in the context of an initial assessment), assisting with the formulation of a client's intervention goals, monitoring a client's progress after receiving intervention/remedial services, or providing evidence to justify the need for a client to receive continued services (Sorter and Kulp 2003b). The Beery tests can also be used for research purposes such as evaluating the effectiveness of educational, psychological, therapeutic, or medical services provided to clients (Dawson and Watling 2000). For example, Pfeiffer et al. (2015) scrutinized the use of Beery VMI as a potential outcome measure for hand writing interventions with first and second grade primary school students. The study involved 207 students divided into two groups. The first group completed a formal handwriting program while the control group received standard programing. The two groups completed the Beery VMI pre- and post-completion of the handwriting and standard instruction programs. When the pre- and post-completion Beery VMI scores were compared, no significant difference was detected. Pfeiffer et al. (2015) suggests that the Beery VMI may not be the most appropriate outcome measure to uncover clinically significant or skill-related changes in handwriting remediation interventions involving school-age children.

Individuals diagnosed with ASD, Asperger's Syndrome (AS), and other related pervasive developmental disorders (PDD) often present with VMI problems (Baranek et al. 2005; Dowd et al. 2012; Duffield et al. 2013; Fournier et al. 2010; Fuentes et al. 2010; MacDonald et al. 2014; Mayes and Calhoun 2003a; McPhillips et al. 2014; Novales 2006; Volker et al. 2010). "ASD evaluations need to include assessments of visual-motor control" (Beery and Beery 2010, p. 119). Numerous studies have indicated that the VMI skills of individuals with ASD and AS are often below average. For example, Volker et al. (2010) assessed the VMI skills of 60 children with high-functioning autism spectrum disorders (HFASDs) and 46 typically developing children using the *Bender Visual-Motor Gestalt Test Second Edition*

(BG-II) and Beery VMI. After statistically controlling for IQ, the HFASD group scored significantly lower than the typically developing group on the BG-II and Beery VMI with greater *motor* involvement. In another recent study, Green et al. (2016) examined the visual-motor integration skills of individuals who presented ASD. An all-male sample consisting of two contrasting groups were recruited into the study: 56 participants with ASD (ages 3 to 23) and 36 typically developing participants. Both groups completed the Beery VMI. The findings indicated that the two groups were significantly different in relation to their visual-motor integration skills as measured by the Beery VMI.

McDonald (2013) compared the visual-motor skills of children presenting with HFASDs. Ninety children with HFASD and 52 typically developing children completed the BG-II using the KOPPITZ-2 scoring system and the Beery VMI. In addition, a subsample of 33 matched pairs was analyzed to examine potential visual-motor differences between groups, while controlling for age, gender, ethnicity, parent education, and verbal ability. McDonald (2013) reported that the children presenting with HFASD had significantly lower scores on the BG-II and the Beery VMI, both samples scored significantly lower on the MCT than on the VPT, and a moderate level correlation was obtained between the BG-II and the Beery VMI composite score. These results are similar to those reported by Volker et al. (2010) and Schlooz and Hulstijn (2012).

In a study by Kimberly (2014), 45 participants between the ages of 8 and 14 years of age completed the forty-five participants completed the BG-II, Beery VMI 5th edition, *NEPSY Second Edition, Test of Visual Perceptual Skills-3, Navon Task, Kaufman Test of Educational Achievement, Second Edition, Kaufman Brief Intelligence Test, Second Edition, Behavior Assessment Scale for Children, Second Edition, and Autism Spectrum Screening Questionnaire*. The sample was composed of 22 males with autism spectrum disorder and 23 were typically developing. Kimberly (2014) found that there were no significant differences between the two groups on the BG-II, Beery VMI 5th edition, *Test of Visual Perceptual Skills-*

3, and *Navon Task*. However, students diagnosed with ASD have poorer performance on the *NEPSY Second Edition*. Regression analyses for visual-motor integration indicated the best predictors for the Beery VMI 5th edition was the *Test of Visual Perceptual Skills-3*. It also determined that the Beery VMI 5th edition was predictive all areas related to academic achievement (Kimberly 2014).

Lopata et al. (2007) assessed the gross motor, fine motor, and visuomotor skills of 17 boys, 6–13 years old, with AS. Statistically significant deficits were found for the sample's scores on three types of motor skills. Using the WRAVMA, Novales (2006) assessed the VMI skills of 63 children and adolescents with AS ranging in age from 8 to 18 years (with a mean age of 11 years, 2 months) and found that “individuals with AS performed significantly more poorly on most measures of visual-motor ability when compared to neurotypically developing peers” (p. 2260). In a similar study, Mayes and Calhoun (2003a, b) assessed 63 children with autism ranging in age from 6 to 15 years using a number of IQ tests and found that the children had significantly low scores VMI skills. Therefore, the Beery VMI, VPT, and MCT can play an important role in the assessment of and intervention planning for individuals presenting with ASD, AS, and PDD. In summary, “the primary purpose of the Beery VMI is to help identify, through early screening, significant difficulties that some children [and adults] have integrating, or coordinating, their visual-perceptual and motor (finger and hand movements) abilities” (Beery and Beery 2010, p. 10). The Beery VMI is recommended as an appropriate assessment for use with children, adolescents, and adults presenting with ASD.

See Also

- ▶ Bender Visual-Motor Gestalt Test II
- ▶ Bruininks-Oseretsky Test of Motor Proficiency
- ▶ Fine Motor Development
- ▶ Occupational Therapy (OT)
- ▶ Peabody Developmental Motor Scales (PDMS)
- ▶ Perception
- ▶ Sensorimotor Development

- ▶ Standardized Tests
- ▶ Visual-Motor Function
- ▶ VMI

References and Reading

- American Educational Research Association, American Psychological Association, & National Council on Measurement in Education. (1999). *Standards for educational and psychological testing*. Washington, DC: American Psychological Association.
- Baranek, G. T., Parham, L. D., & Bodfish, J. W. (2005). Sensory and motor features in autism: Assessment and intervention. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Assessment, interventions and policy, Vol. 2, pp. 831–857). Hoboken: Wiley.
- Beery, K. E., & Beery, N. A. (2010). *The Beery-Buktenica Developmental Test of Visual-Motor Integration (Beery VMI) with Supplemental Developmental Tests of Visual Perception and Motor Coordination and stepping stones age norms: Administration, scoring and teaching manual*. Minneapolis: NCS Pearson.
- Beery, K. E., & Berry, N. A. (2004). *The Beery-Buktenica Developmental Test of Visual-Motor Integration (Beery VMI) with Supplemental Developmental Tests of Visual Perception and Motor Coordination: Administration, scoring and teaching manual* (5th ed.). Minneapolis: NCS Pearson.
- Brown, T., Unsworth, C., & Lyons, C. (2009a). An evaluation of the construct validity of the Developmental Test of Visual-Motor Integration using the Rasch Measurement Model. *Australian Occupational Therapy Journal*, 56(6), 393–402.
- Brown, T., Unsworth, C., & Lyons, C. (2009b). Factor structure of four visual – motor instruments commonly used to evaluate school-age children. *American Journal of Occupational Therapy*, 63(6), 710–723.
- Brown, T., Chinner, A., & Stagnitti, K. (2011). Convergent validity of two visual motor integration tests. *British Journal of Occupational Therapy*, 74(6), 295–303.
- Case-Smith, J. (2002). Effectiveness of school-based occupational therapy intervention on handwriting. *American Journal of Occupational Therapy*, 56(1), 17–25.
- Dankert, H. L., Davies, P. L., & Gavin, W. J. (2003). Occupational therapy effects on visual-motor skills in preschool children. *American Journal of Occupational Therapy*, 57(5), 542–549.
- Dawson, G., & Watling, R. (2000). Interventions to facilitate auditory, visual, and motor integration in autism: A review of the evidence. *Journal of Autism and Developmental Disorders*, 30, 415–421.
- Dowd, A. M., McGinley, J. L., Taffe, J. R., & Rinehart, N. J. (2012). Do planning and visual integration difficulties underpin motor dysfunction in autism? A kinematic study of young children with autism.

- Journal of Autism and Developmental Disorders*, 42(8), 1539–1548.
- Duffield, T. C., Trontel, H. G., Bigler, E. D., Froehlich, A., Prigge, M. B., Travers, B., & Lainhart, J. (2013). Neuropsychological investigation of motor impairments in autism. *Journal of Clinical and Experimental Neuropsychology*, 35(8), 867–881.
- Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism spectrum disorders: A synthesis and meta-analysis. *Journal of Autism and Developmental Disorders*, 40(10), 1227–1240.
- Fuentes, C. T., Mostofsky, S. H., & Bastian, A. J. (2010). Perceptual reasoning predicts handwriting impairments in adolescents with autism. *Neurology*, 75(20), 1825–1829.
- Fulkerson, S. C., & Freeman, W. M. (1980). Perceptual-motor deficiency in autistic children. *Perceptual and Motor Skills*, 50(1), 331–336.
- Goldstein, D. J., & Britt, T. W., Jr. (1994). Visual-motor coordination and intelligence as predictors of reading, mathematics, and written language ability. *Perceptual and Motor Skills*, 78, 819–823.
- Graham, R. (2007). Review of the Beery-Buktenica Developmental Test of Visual-Motor Integration. In K. F. Geisinger, R. A. Spies, J. F. Carlson, & B. S. Plake (Eds.), *The seventeenth mental measurements yearbook* (5th ed.). Lincoln: Buros Institute of Mental Measurements.
- Green, R. R., Bigler, E. D., Froehlich, A., Prigge, M. D., Travers, B. G., Cariello, A. N., & ... Lainhart, J. E. (2016). Beery VMI performance in autism spectrum disorder. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 22(7), 795–817.
- Hall, S., Pinkerston, S. L., Szalda-Petree, A. C., & Coronis, A. R. (1996). The performance of healthy older adults on the Continuous Visual Memory Test and the Visual Motor Integration Test. *Journal of Clinical Psychology*, 52(4), 449–454.
- Kimberly, O. (2014). Visual, motor, and visual-motor integration difficulties in students with autism spectrum disorders. *Dissertation Abstracts International Section A: Humanities and Social Sciences*, 75(1-A(E)).
- Lim, C. Y., Tan, P. C., Koh, C., Koh, E., Guo, H., Yusoff, N. D., & ... Tan, T. (2015). Beery-Buktenica Developmental Test of Visual-Motor Integration (Beery-VMI): lessons from exploration of cultural variations in visual-motor integration performance of pre-schoolers. *Child: Care, Health and Development*, 41(2), 213–221.
- Lopata, C., Hamm, E. M., Volker, M. A., Sowinski, J. E., & Thomeer, M. L. (2007). Motor and visuomotor skills of children with Asperger's disorder: Preliminary findings. *Perceptual and Motor Skills*, 104(3), 1183–1192.
- MacDonald, M., Lord, C., & Ulrich, D. A. (2014). Motor skills and calibrated autism severity in young children with autism spectrum disorder. *Adapted Physical Activity Quarterly*, 31(2), 95–105.
- Mayes, S. D., & Calhoun, S. L. (2003a). Ability profiles in children with autism: Influence of age and IQ. *Autism*, 7, 65–80.
- Mayes, S. D., & Calhoun, S. L. (2003b). Analysis of WISC-III, Stanford-Binet:IV, and academic achievement test scores in children with autism. *Journal of Autism and Developmental Disorders*, 33(3), 329–341.
- McCrimmon, A. W., Altomare, A. A., Matchullis, R. L., & Jitlina, K. (2012). Test review of the Beery Developmental Test of Visual-Motor Integration (6th ed.). *Journal of Psychoeducational Assessment*, 30(6), 588–592.
- McDonald, C. A. (2013). Visual-motor performance of children with high-functioning autism spectrum disorders: A comparison of the Beery VMI-VI and the KOPPITZ-2 developmental scoring system for the Bender Visual Motor Gestalt TEST-II. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 73(11-B(E)).
- McKnight, T., & Chandler, T. (2007). Review of the Beery-Buktenica Developmental Test of Visual-Motor Integration. In K. F. Geisinger, R. A. Spies, J. F. Carlson, & B. S. Plake (Eds.), *The seventeenth mental measurements yearbook* (5th ed.). Lincoln: Buros Institute of Mental Measurements.
- McPhillips, M., Finlay, J., Bejerot, S., & Hanley, M. (2014). Motor deficits in children with autism spectrum disorder: A cross-syndrome study. *Autism Research*, 7(6), 664–676.
- Novales, B. D. (2006). Visual-motor abilities in individuals with Asperger syndrome. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 67(4-B), 2260.
- Pfeiffer, B., Moskowitz, B., Paoletti, A., Brusilovskiy, E., Zylstra, S. E., & Murray, T. (2015). Developmental Test of Visual-Motor Integration (VMI): An effective outcome measure for handwriting interventions for kindergarten, first-grade, and second-grade students? *American Journal of Occupational Therapy*, 69(4), 1–7.
- Pianta, R. C., & McCoy, S. J. (1997). The first day of school: The predictive validity of early school screening. *Journal of Applied Developmental Psychology*, 8(1), 1–22.
- Sorter, J. M., & Kulp, M. T. (2003a). Clinical value of the Beery Visual-Motor Integration Supplemental Tests of Visual Perception and Motor Coordination. *Optometry and Vision Science*, 80(4), 312–315.
- Sorter, J. M., & Kulp, M. T. (2003b). Are the results of the Beery-Buktenica Developmental Test of Visual-Motor Integration and its subtests related to achievement test scores? *Optometry and Vision Science*, 80(11), 758–763.
- Volker, M. A., Lopata, C., Vujnovic, R. K., Smerbeck, A. M., Toomey, J. A., Rodgers, J. D., et al. (2010). Comparison of the Bender Gestalt-II and VMI-V in samples of typical children and children with high-functioning autism spectrum disorders. *Journal of Psychoeducational Assessment*, 28(3), 187–200.
- Wuang, Y., & Su, C. (2009). Rasch analysis of the Developmental Test of Visual-Motor Integration in children with intellectual disabilities. *Research in Developmental Disabilities*, 30(5), 1044–1053.

Visual-Motor Skills

► Visual-Motor Function

Visual-Spatial Ability

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Definition

Visual-spatial ability is a broad term that emphasizes processes such as image generation, storage, retrieval, and transformation and includes a collection of skills in the areas of spatial relations, visualization, visual memory, closure speed, and spatial scanning (Mather and Wendling 2005). Because visual-spatial ability is a multi-dimensional construct, it is possible for an individual to have a relative strength in one area of visual-spatial ability (e.g., visual memory of objects) and a relative weakness in another (e.g., spatial relations; Mather & Wendling).

The visual environment can be organized into a meaningful whole through visual perception (Grieve 2000). Visual perception of an object requires the integration of several features, such as color, depth, separating shapes and objects from their background, and form constancy. Visual object recognition involves integrating visual perception with previous knowledge of known objects. Spatial ability, on the other hand, involves the integration of visual scanning of space, mental representation of the relative position of one's body parts in space, ability to perform the necessary movements for a task, and understanding of the topographical environment (Grieve 2000).

Visual-spatial ability is an area typically measured as part of neuropsychological examinations and may include the assessment of perceptual skills, constructional skills, and spatial awareness (Sattler and Hoge 2006). The

Perceptual Reasoning Index on the Wechsler Intelligence Scale for Children, Fourth Edition, which is comprised of the Block Design, Picture Concepts, Matrix Reasoning, and Picture Completion subtests, offers a broad measure of graphomotor, spatial, and visual scanning abilities (Sattler & Hoge). An example of a test that measures visual-motor perception is the Bender-Gestalt II. This test includes 16 stimulus cards that each contains a line drawing. Test takers are asked to copy each drawing on a blank piece of paper. A 5-point rating scale is used to score each item, and age-based standard scores are available for the measure (Sattler & Hoge). Another example is the Beery-Buktenica Developmental Test of Visual-Motor Integration, Fifth Edition (Beery VMI-Fifth Edition). This assessment includes 30 items that incorporate spontaneous drawing and copying designs from the stimulus book. Each item is scored as either a 0 or 1, and age-based standard scores can be calculated (Sattler & Hoge).

Historical Background

Components of visual-spatial ability, such as object perception and measuring basic properties of vision, have been researched by psychologists since the nineteenth century (Goldstein 2005). As behaviorism and its emphasis on observable behavior, rather than mental processes, became more prevalent in the first half of the twentieth century, less attention was paid to studying cognition. However, the field of psychology returned to an interest in mental processes later in the twentieth century (Goldstein 2005). Modern technology, such as brain imaging techniques, has allowed researchers to directly measure physiological activity related to cognition, including visual-spatial processes.

Current Knowledge

Neuroimaging research has found some overlap in brain activity during processes that involve visual

perception and visual imagery (Sternberg 2006). This research provides support for the functional-equivalence hypothesis, which states that, even though visual perception and visual imagery are not identical, they are functionally equivalent (Sternberg 2006).

Research findings have also indicated that visual and spatial imagery may be mentally represented differently (Sternberg 2006). Visual imagery is the use of images that represent characteristics such as shape and color, whereas spatial imagery represents characteristics such as depth, distance, and orientation.

Researchers have also examined how visual-spatial skills develop across the lifespan. Basic spatial understanding, including skills such as spatial visualization, begins to develop in children at a young age (Sternberg 2006). Spatial visualization refers to the capacity to orient oneself in one's surroundings and to mentally manipulate images of objects (Sternberg 2006). From childhood through young adulthood, the speed of mental rotation increases. Object familiarity has also been shown to increase mental rotation speed. Middle-aged adults generally have slower mental rotation speeds than young adults but have similar response times for image scanning tasks (Sternberg 2006).

Studies have not generally found a strong relationship between visual-spatial ability and academic performance, although some studies have found evidence of an association between visual-spatial ability and math performance (Mather and Wendling 2005). Performance on visual-spatial tasks has been shown to be influenced by speed, attention, motivation, working memory, and visual-motor coordination (Mather & Wendling). Research has found that visual sensory impairment can affect the perception and storage of visual cues, which contribute to visual-spatial ability (Baum and Katz 2010).

Future Directions

Current research on cognition, which includes visual-spatial ability, has emphasized an interdisciplinary approach to cognitive neuroscience

and has utilized brain imaging techniques to examine relationships between particular brain structures and specific cognitive processes (Goldstein 2005; Sternberg 2006). It is likely that these trends will continue and further contribute to researchers' understanding of visual-spatial ability.

See Also

- [Visual Processing](#)
- [Visual Scanning](#)

References and Reading

- Baum, C. M., & Katz, N. (2010). Occupational therapy approach to assessing the relationship between cognition and function. In T. D. Marcotte & I. Grant (Eds.), *Neuropsychology of everyday functioning*. New York: Guilford Press.
- Goldstein, E. B. (2005). *Cognitive psychology: Connecting mind, research, and everyday experience*. Belmont: Thomson Wadsworth.
- Grieve, J. (2000). *Neuropsychology for occupational therapists: Assessment of perception and cognition* (2nd ed.). Oxford: Blackwell Science.
- Mather, N., & Wendling, B. J. (2005). Linking cognitive assessment results to academic interventions for students with learning disabilities. In D. P. Flanagan & P. L. Harrison (Eds.), *Contemporary intellectual assessment: Theories, tests, and issues* (2nd ed., pp. 269–294). New York: Guilford Press.
- Sattler, J. M., & Hoge, R. D. (2006). *Assessment of children: Behavioral, social, and clinical foundations* (5th ed.). San Diego: Jerome M. Sattler.
- Sternberg, R. J. (2006). *Cognitive psychology* (4th ed.). Belmont: Thomson Wadsworth.

Visuomotor Ability

- [Visual-Motor Function](#)

Visuomotor Function

- [Visual-Motor Function](#)

Visuomotor Skills

- [Visual-Motor Function](#)

Vitamin B8

- [Inositol: Definition](#)

Vivactil

- [Protriptyline](#)

VMI

- [Beery-Buktenica Developmental Test of Visual-Motor Integration](#)
- [Visual-Motor Integration, Developmental \(VMI\) Test](#)

Vocable

- [Protowords](#)

Vocabulary

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Synonyms

[Lexicon](#); [Messages](#); [Words](#)

Definition

Vocabulary is a set of words that a person knows. Typically, vocabulary is described as receptive and expressive. Receptive vocabulary refers to the words that are understood by a person, typically assessed by having the person point to a picture that is named. Expressive vocabulary refers to the words that a person can produce and is typically assessed by showing the person a picture and having them name the word depicted. Individuals with autism may use speech, sign, typing, or pointing to picture symbols to demonstrate their expressive vocabulary skills. People with autism may have vocabulary skills that are within the normal range of functioning or may have an impairment in one or both types of vocabulary.

See Also

- [Expressive Language](#)
- [Peabody Picture Vocabulary Test](#)
- [Receptive Vocabulary](#)
- [Word Use](#)

References and Reading

Kjelgaard, M. M., & Tager-Flusberg, H. (2001). An investigation of language impairment in autism: Implications for genetic subgroups. *Language and Cognitive Processes*, 16(2/3), 287–308.

Vocalization

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Synonyms

[Babbling](#); [Cooing](#); [Prelinguistic sounds](#)

Definition

Vocalizations are sounds produced by children before they learn to talk. For children with ASD, vocalizations may continue to be used throughout the life span, particularly if spoken language does not develop. Vocalizations can be divided into two categories: speech-like and nonspeech. Speech-like vocalizations include consonant and vowel sounds (e.g., baba, daba) and are often referred to as babbling or cooing. Nonspeech vocalizations are natural, vegetative sounds that do not resemble speech, such as crying, laughing, burping, as well as uncommon sound productions such as high-pitched squeals and low-pitched growls. A higher frequency and longer persistence of atypical vocalizations has been found to be present in children with ASD and in infants at high risk for the syndrome, before spoken language emerges.

See Also

- ▶ [Babbling](#)
- ▶ [Prelinguistic Sounds](#)

References and Reading

- Patten, E., Belardi, K., Baranek, G. T., Watson, L. R., Labban, J. D., & Oller, D. K. (2014). Vocal patterns in infants with autism spectrum disorder: Canonical babbling status and vocalization frequency. *Journal of Autism and Developmental Disorders*, 44(10), 2413–2428.
- Paul, R., Fuerst, Y., Ramsay, G., Chawarska, K., & Klin, A. (2011). Out of the mouths of babes: Vocal production in infant siblings of children with ASD. *Journal of Child Psychology and Psychiatry*, 52, 588–598.
- Schoen, E., Paul, R., & Chawarska, K. (2011). Phonology and vocal behavior in toddlers with autism spectrum disorders. *Autism Research*, 4(3), 177–188.

Vocational Counseling

- ▶ [Department of Vocational Rehabilitation](#)

Vocational Counselor

- ▶ [Vocational Evaluator](#)

Vocational Evaluator

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Synonyms

[Job counselor](#); [Vocational counselor](#)

Definition

A vocational evaluator is a professional whose goal is to help people match a career path to their interests, abilities, knowledge, personality, and skills. The evaluator's focus is to help integrate their clients into the workplace. The evaluator minimally holds a bachelor's degree with vocational evaluation specialization. Evaluators typically work in settings such as hospitals, schools, public agencies, and independent living centers and are sometimes in private practice. A certified vocational evaluator (CVE) has received national certification by the Commission on Certification of Work Adjustment and Vocational Evaluation Specialists (CCWAVES).

When evaluating clients on the autistic spectrum, the vocational evaluator will use a variety of instruments. These will provide information about the client's medical, psychological, social, vocational, educational, economical, and cultural history and abilities. The evaluator may assess such areas as being able to understand socially appropriate behaviors, the quality of communication skills, functional academics, and problem-solving skills. The evaluator will also look at the client's work-related behaviors, such as the ability follow directions and to complete tasks successfully, maintain an appropriate rate of work, follow rules, stay on task, and complete multistep

assignments. The outcome of the evaluation is a comprehensive report containing specific recommendations. This report can be used to guide the client and/or counselor in developing appropriate client employment goals.

See Also

- [Employment Specialist](#)

References and Reading

Commission on Certification of Work Adjustment and Vocational Evaluation Specialists (CCWAVES), Rolling Meadows.
VEWAA, National Rehabilitation Association, Reston.
www.VEWAA.org.

Vocational Rehabilitation Act of 1973

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Definition

The Vocational Rehabilitation Act of 1973 Title V was put in place to correct the problem of discrimination against people with disabilities in the United States. Affirmative action programs were established in Title V, Sections 501, 502, 503, and 504. Individuals who qualify as having a disability have experienced discrimination both because of negative attitudes in regard to their ability to be an effective employee as well as the physical barriers at work facilities. The Title V of the Vocational Rehabilitation Act requires private employers with federal contracts over \$2,500 to take affirmative action to hire individuals with a mental or physical disability. While this means that employers must make reasonable accommodations for disabled employees, it does not mean they must hire unqualified individuals. There are

additional sections of the Act that provide vocational counseling, training assistance, and job placement for individuals with severe disabilities.

In the context of the Vocational Rehabilitation Act, the term “disabled individual” means “any person who (1) has a physical or mental impairment which substantially limits one or more of such person’s major life activities, (2) has a record of such impairment, or (3) is regarded as having such an impairment.” This definition is closely related to the definition provided by the Americans with Disabilities Act.

The Vocational Rehabilitation Act does not require employers to hire or retain a disabled person if the individual has a contagious disease that poses a direct threat to the health and safety of others, and the individual cannot be accommodated. Also, employment is not required if the disability prevents the individual from being able to perform a required part of the job or if the individual would be considered unqualified for the job regardless of their disease or disability.

See Also

- [Americans with Disabilities Act](#)
- [Department of Vocational Rehabilitation](#)
- [Vocational Evaluator](#)
- [Vocational Training](#)

References and Reading

Zirkel, P. A. (2003). Conducting legally defensible Section 504/ADA eligibility determinations. *West’s Education Law Reporter*, 176, 1–11.
Zirkel, P. A. (2004). *Section 504, the ADA, and the schools*. Horsham: LRP Publications.
Zirkel, P. A. (2009). A step-by-step process for Section 504/ADA eligibility determinations. *West’s Education Law Reporter*, 239(2), 333–343.

Vocational Rehabilitation Specialist

- [Employment Specialist](#)

Vocational Trainer

► [Job Coach](#)

Vocational Training

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Definition

Vocational training is a form of education that is practically oriented. Individuals who are enrolled in vocational training are learning skills specific to a particular occupation or job. These occupations tend to be manual jobs by nature and are in the mechanical arts or some other form of industry, agriculture, or trade. The education is less academically oriented with an emphasis upon practical skills and apprenticeship work. The students learn the trade by doing supervised work. Extensive practical experience is given in lieu of learning theory, technical knowledge, or following a liberal arts education.

Historical Background

The history of vocational training is as long as humans have been involved in organized activity for survival and commerce. The training of individuals was conducted by the family, tribe, or later village members. Often, young children of impoverished families were put in the care of skilled craftsman or better-educated individuals as a way of the child to learn vocational skills as an apprentice or indentured servant. This insured both the child's and the family's survival.

During the later part of the medieval and into the Renaissance periods, Western Europe would see the rise of the university which led to a new

educational and economical structure and system. The impact of the rise of the university would be upon the class system. No longer would only the nobility be able to read and write, but a new class of citizens would have this skill set. A university education led to new jobs in the economic structure. A university education began to be a viable path to economic survival for an individual. Apprenticeships were now not the only way to learn vocational skills and gain employment.

The industrial revolution also contributed to the change in the way individuals learned job skills. The economic structure shifted from cottage-based industries where family members taught children how to work and create products to be sold outside of the home and to mass-produced products manufactured in factories to be sold to the public on a large scale. The creation of the assembly line method meant that individuals went from being skilled craftsmen responsible for the creation of a product from start to finish to workers who specialized in the completion of one aspect of the overall product. This was often repetitive and dangerous work. The industrial revolution also shifted where the population lived. Prior to the industrial revolution, the greatest proportion of the population lived in agrarian centers or sparsely populated villages surrounded by farms. With the advent of the factory, the population shifted to large urban centers living near the factories where they were employed. "Improvements in transportation and communication - roads, canals, and railroads - brought communities closer together, stimulating the exchange of goods and services as well as the growth of cities" (Barlow 1976, p. 31).

There was no coordinated effort to begin vocational training in the United States or abroad. An educational system was in its nascent stage in the United States. Each state was responsible for the creation of an educational system since there were no constitutional provisions for a national educational system. Horace Mann, Secretary of the Massachusetts State Board of Education (1837–1849), would be credited with the revolutionary concept of free, public, universal education. The concept of the common school was beginning to take root in the country. The

common school was what we would refer to as primary education in today's terms. Secondary education was a radical idea, and the notion of public tax dollars being spent to fund schools was highly controversial. High schools, which would become the home base for vocational training, were rare. According to Barlow (1976), there were only 60 high schools in the entire country by 1850. The Manual Labor Movement, the Hampton Institute, the Morrill Act, and the Kalamazoo Case all converged during this time period to influence the formation of vocational education.

The Manual Labor Movement was led by farmers, mechanics, and artists. In England, in the early nineteenth century, the Mechanics' Institute was formed. It attempted to "regain the educational values lost with the coming of the factory system" (Barlow 1976, p. 32). During the 1820s, the Franklin Institute, the Maryland Institute for the Promotion of Mechanic Arts, and the Ohio Institute of Cincinnati were established in the cities of Philadelphia, Baltimore, and Cincinnati, respectively. These institutes all had reading rooms, libraries, public lectures, cabinets of models and apparatuses, and day and night schools, among their offerings. Educational reformers such as Emma Willard, Catherine Beecher, Mary Lyon, and Ellen Richards began to question the prevailing notion that the woman's place was in the home. Lyon established Mount Holyoke Female Seminary in 1837. Women began to attend postsecondary training, and Elmira College granted the first college degrees to women in 1855 (Barlow 1976). The home economics movement was born during this time period which was a precursor to independent living skills training for individuals on the autism spectrum.

The Hampton Institute is the acknowledged beginning of the Trade School Movement. After the Civil War, a great debate raged in the country regarding whether freed Negroes should have a classical education or an industrial education. As early as 1853, Frederick Douglass advocated for industrial schools for Negroes, while Harriet Beecher Stowe provided financial backing for either an industrial school or a classical education

for freed Negroes. General Samuel Chapman Armstrong commanded a regiment of Negro soldiers during the war and worked as the superintendent of education under the Freedmen's Bureau. He organized the Hampton Institute for the training of Negroes in skilled manual labor. Armstrong viewed a skilled Negro labor force as essential to the reconstruction of the South post-Civil War, and he felt that a liberal education was essential to improving the Negroes social status (Barlow 1976). The Hampton Institute offered vocational training in trades, many of which are still offered in vocational training centers to this day (e.g., plumbing, carpentry, mechanics, upholstery).

The passage of the Morrill Act in 1862 made it possible for states to establish universities. The idea was the brain child of Jonathon Baldwin Turner who believed that society was composed of two classes: a professional and an industrial class. His dream was to establish an industrial university in each state. To fund his dream, he proposed that federal aid be generated through the sale of public lands. Farmers in his home state of Illinois enthusiastically supported the idea. "The act of donating public lands to establish colleges for the benefit of agriculture and the mechanical arts called for land grants to states based on their representation in Congress—30,000 acres for each senator and representative" (Barlow 1976, p. 38). President Lincoln signed the bill into law on July 2, 1862.

The Kalamazoo case represented a major turning point in education and the establishment of vocational training in high schools. The majority of the educational reform thrust was focused upon primary school or the common school as it was called. Students were not expected to receive a public education beyond the 8th grade. In 1874, the Michigan Supreme Court heard the case where citizens from Kalamazoo brought forth a suit against the school districting disputing the right to collect taxes to support local public high schools. The court ruled against the plaintiffs finding an inconsistency between the provision of primary schools and state universities and forcing parents to find private education to prepare students for higher studies. This provided the right

to equal access to higher education for all. It established the right of school districts to levy taxes to support high schools. However, it would not be until well into the twentieth century that vocational training would become a part of the high school curriculum (Barlow 1976).

In the United States after the American Civil War, Abner Flexner began a crusade to reform the medical training of doctors and later higher education as a whole. Doctors were primarily trained through apprenticeships with no standards for their training and no standards for the people who trained them. Virtually anyone could call themselves a doctor, and any institution could call themselves a medical school (Starr 1982). Flexner led the reform of not only medical schools but university education in general. This led to the closing of over half of the medical schools across the country. It also led to the questioning of many groups as to whether or not they were indeed a profession and what a profession was in general. Did they as a group have a unique skill set, an ethical code, and a transmittable knowledge base? Having a transmittable knowledge base distinguished a profession from a trade. It also meant that professionals went onto higher education through the nation's system of colleges and universities, whereas skilled craftsman learned their craft through vocational or trade schools.

Rationale or Underlying Theory

The rationale for training individuals with ASDs specifically to work is that many have a desire to work and be productive members of society. Otherwise, society must support these individuals who have an average life expectancy that is well over 70 years of life. Although no specific epidemiological data are available on the unemployment rates of people with ASDs, anecdotal evidence suggests their unemployment rate is around 90% (Gerhardt 2011, personal communication). The costs of not providing vocational training are staggering from a fiscal and emotional perspective. Tax credits are available to employers who train and hire individuals with disabilities.

Goals and Objectives

The goals of vocational training for individuals with ASDs are to match the interests, aptitudes, and job availability with the individual in order for the person to work in a setting with the highest level of independence and community integration possible. This means the vocational goals must be individualized. In addition to specific job skills, the objectives should include the adoption on prevocational skills that will guarantee employment success. These objectives should include demonstrating appropriate dress for employment; good hygiene; the ability to cook and feed one's self before, during, and after working; and the ability to transport one's self to and from work.

Treatment Participants

Treatment participants are those individuals on the autism spectrum who have a desire to learn how to work.

Treatment Procedures

Vocational training is not a treatment *per se*. Rather, it is an educational approach that focuses upon teaching skills that are directly related to a particular occupation. The training is practical in nature and focuses upon how to complete a task or job. This differs from professional training where the education is less directly related to tasks performed by a specific occupation. Professional training often teaches theory, ethics, and how to think and problem solve. These skills are applied generally to a wide variety of work-related situations. Vocational training, by contrast, teaches how to complete specific tasks.

Prior to engaging in vocational training of an individual with autism, one must complete a thorough evaluation of the person's strengths, areas of difficulty, interests, and aptitudes. For younger individuals, the evaluations can be conducted through the local school district. IQ tests and assessments of reading, writing, and math ability are conducted on a triennial basis as

a part of the special education system and are part of writing an Individualized Education Plan (IEP) under the Individuals with Disabilities Education Act (IDEA). Under IDEA, students beginning at age 14 should have a transition plan written describing the goals for the student and how the student will reach those goals. The goals in the transition plan typically are focused upon vocational training or postsecondary education. The evaluators will need to conduct vocational assessments and examine things like fine motor skills, especially if the individual is interested in an occupation that involves a great deal of eye-hand coordination. An occupational therapist who is a part of the school-based support team can conduct such an assessment. Part of the occupational therapist's evaluation may include an assessment of assistive technology. Functional assessments for assistive technology are not covered by IDEA. These assessments are a part of Assistive Technology Act of 2004, P.L. 108-364; 29 U.S.C 3001 et seq.

Older individuals with autism can receive vocational assessments through state offices of vocational and rehabilitative services. The state office of vocational rehabilitative services or office of persons with developmental disabilities may subcontract these services to private not for profit social service agencies. The exact names of these offices vary by state. Consequently, keyword searches for these offices require the use of a variety of synonyms. These state services will not only evaluate the individual through the use of paper and pencil or computerized aptitude tests and interest inventories, but they will conduct observations of the individuals in work settings to evaluate the goodness of fit between the individual and the job setting. Working with the individual employees of the office of vocational and rehabilitative services will develop an Individualized Plan for Employment (IPE) also known as an Individualized Employment Plan (IEP) which is distinct and separate from an Individualized Educational Plan (IEP) under IDEA. The Individualized Plan for Employment (IPE) is a document that outlines the plan for the individual seeking employment through a series of behaviorally specific goals with fixed deadlines

and outcomes. It will specify the type of training he or she will receive and who will provide the services and where those services will take place. This IPE can help the person obtain assistive technology in order to attend a community college. The IPE can also prescribe other types of supports that the person will need in order to be successfully employed. The other types of support can be learning to drive a car or travel training on mass transit, so the individual can get to work or to attend resume writing workshops or job interview clinics.

After the individual has been evaluated and assessed and his or interests and aptitudes have been determined, then an assessment of the current and future job markets should be conducted to insure the long-term employment of the individual with autism. One excellent source of information is the US Department of Labor, Bureau of Labor Statistics. This department publishes a number of documents which are free to the public. One document in particular, the *Occupational Outlook Handbook* (OOH), is available on a yearly basis. This report informs readers the type of training and education needed by occupational title, the expected earnings of individuals in a vocation, the expected job prospects, a description of what workers do in an occupation, and the working conditions someone faces with a particular job title.

Once an individual with autism has had their aptitudes, abilities, and vocational interests assessed and those have matched with the employment outlook for a particular vocation, then the individual must decide where to obtain the vocational training. There are three general places to obtain training for future employment: traditional vocational training programs, supported academic programs, and comprehensive transition and postsecondary programs (CTPs). Traditional vocational training programs may begin in high school. Some are integrated into the standard high school curriculum. Others are structured as alternative high schools. A third permutation of the traditional vocational training center is a county or municipal training center that draws from a larger catchment area than the local high school. This third permutation may include

students who are receiving postsecondary training, and these training programs often work with state offices of vocational and rehabilitative services. A final option for traditional vocational training is a private vocational training or “tech” school. The final option is not one that an individual on the autism spectrum should have to pursue. Private vocational training centers charge a tuition, and their students should be eligible for federal student aid. However, students on the autism spectrum should be entitled to vocational training through state offices of vocational rehabilitative services or state offices of persons with developmental disabilities. They do not need to pay tuition for vocational training.

Most people are familiar with the variety of occupations available to students who attend vocational training. The most common occupations that readily come to mind are automotive repair, plumbing, carpentry, electrical repair, cosmetology, culinary arts, clerical skills, and retail. However, there are a number of other occupations that do not readily come to mind. Some vocational centers offer veterinary assistant, small animal care, physical therapy and occupational therapy assistant, digital photography, and personal trainer certificate programs. Many individuals on the autism spectrum who are higher functioning have an acute interest in computers. However, many do not see the utility in obtaining a liberal arts degree in computer science, computer engineering, or computer graphics. They refuse to take the English or humanities courses that are required to fulfill the degree program because the course is not directly related to working with computers. Many vocational training centers will offer certification courses in computer programming, networking, and/or repair. Others will offer certification courses in specific software applications. Data entry for individuals on the autism spectrum, who are interested in this occupation, is often a good choice for those individuals who prefer predictability and routine. Some businesses have found that individuals on the autism spectrum who are in data entry fields have higher accuracy rates than their neurotypical peers (Bennett 2009). Consulting the *Occupational Outlook Handbook* for fields that have the most

projected openings will help maximize the chances of the person becoming employed.

The second place or type of program where higher functioning individuals on the autism spectrum can receive vocational training is through supported academic programs at colleges and universities. The training one receives at college or university is less directly related to a specific vocation. The notion is that the liberal arts education is meant to teach an individual how to think and problem solve. It is up to the individual to apply what he or she has learned to the work environment. Experiential learning through service learning programs or internships is the most direct form of vocational training at colleges or universities. Supported academic programs at colleges and universities are charged with insuring that the institution is in compliance with the Americans with Disabilities Act. This often occurs through the college’s Disabled Student Services (DSS) office (although some colleges are beginning to offer programs specifically designed to support higher functioning individuals on the autism spectrum). This office is charged with insuring that the individual with a disability is not denied access to or the benefits of the university solely on the basis of his or her disability. The student must be “otherwise qualified” to attend the university which means they must have the intellectual ability to meet the academic demands of a degree program. The DSS office will assist the student with a disability by either altering the environment (e.g., providing notetakers or allowing testing in alternative venues) or by modifying the behaviors or the skills of the student (e.g., teaching organizational skills or providing stress management workshops). The range of remedial and supportive services is wide due to how the organization interprets ADA. The DSS offices are generally very good at providing reasonable accommodations that involve academic interventions. Colleges are less adept at providing accommodations in the social or sensory integration realms.

Many higher functioning individuals on the autism spectrum are quite comfortable with the structure of the academic demands of college

courses. Individuals on the autism spectrum have difficulty, not with the academic demands but rather with the independent living, executive functioning, and social skills demands of living in a university environment (VanBergeijk et al. 2008). In order for universities to be successful places for vocational training for individuals with ASDs, they must provide the structure and scaffolding necessary to address these deficits. Otherwise, students with ASDs will not be able to access the benefits of the university degree because of the nature of their disability. Colleges must target students with ASDs to also participate in resume writing, interviewing, and job search workshops in order for these students to be successfully employed. The Health Center at George Washington University and Thinkcollege.net are two sources that are helpful in identifying supported academic programs for students with a variety of disabilities. The third place students with autism can receive vocational training is a Comprehensive Transition and Postsecondary program (CTP). CTPs combine aspects of traditional vocational training programs and supported academic programs. This hybrid model focuses upon vocational, independent living, social, and academic skills training. These programs are more individually tailored and do not necessarily result in a college degree. Their goals are to either transition the individual into the world of work and independent living or transition the individual with an intellectual disability into a degree-bearing program full time. A CTP can last longer than a traditional 4-year degree program. Most CTPs have four key components: (1) supported employment and work skills training, (2) adult life skills training (e.g., financial management, grocery shopping, laundry, and home maintenance), (3) social and personal relationships counseling and training, and (4) encouraged social involvement in the community. The best practices of CTPs are (a) using group modality, (b) involving the family, (c) developing community partnerships, (d) taking a long-term approach by following up with graduates, and (e) ongoing evaluation. Involvement of the family distinguishes this model from supported academic programs and traditional vocational training

programs where the expectation is that the family will generally not be involved in the process.

There are two subtypes of Comprehensive Transition and Postsecondary Programs (CTPs), and the distinction is important. The first subtype is a college-affiliated CTP. Generally, college-affiliated CTPs are either not for profit or for profit private social service agencies that are based out of an apartment complex or group housing situation. They are not formally a part of a college or university. However, they may have a memorandum of understanding with a local community college which outlines the relationship between the agency, the college, and the students with a variety of disabilities, not just autism spectrum disorders. College-affiliated CTPs are ideal for individuals on the autism spectrum who have difficulty generalizing independent living skills across environments. Individuals are taught independent living skills in their own apartments. The second type of CTP is the college-based CTP. College-based CTPs are an official program or department of the college or university and draw upon its infrastructure. The distinction between the two subtypes is important in terms of Federal Financial Aid. Students with intellectual disabilities who are enrolled in a college-based CTP that is recognized by the US Department of Education are eligible to complete the Free Application for Federal Student Aid (FAFSA). By completing the FAFSA, students with intellectual disabilities including autism may receive Pell Grants, Federal Supplemental Education Opportunity Grants (FSEOG), and student work study monies. Students with intellectual disabilities who are enrolled in college-affiliated CTPs are *not* eligible for federal financial aid.

Efficacy Information

Vocational training in a postsecondary environment is critical to employment for people with any type of disability and especially for those on the autism spectrum. Recall the unemployment rate for individuals with ASDs is around 90%. Obtaining any type of postsecondary training

significantly increases the probability that the person with disabilities will be employed. “In fact, employment rates for people with disabilities demonstrate a stronger positive correlation between level of education and the employment rate than is seen in statistical trends for the general population. . . for individuals with disabilities a university education is highly correlated with vocational options and financial success” (Wehman 2001, pp. 248–249).

Outcome Measurement

The ultimate outcome for individuals on the autism spectrum is that they are employed in a competitive work environment with no supports and they are able to fully support themselves. However, there are many other outcomes between this outcome and unemployment and being fully dependent upon others for survival. If competitive employment is the apex of an employment pyramid, then the next level of independence is customized employment. According to Gerhardt (2009), customized employment is a “. . . highly specialized derivative of supported employment, supported employment tends to match individuals with previously existing jobs in the community. Customized employment. . . works to create highly individualized, yet economically viable, jobs through active employer negotiation. . .” (p. 20). Supported individualized placements are a third possible employment outcome where individuals with ASDs are placed in an existing job and are supported by a job coach who provides on the job training. Slowly, the supports of the job coach are withdrawn, while the individual develops natural supports in the environment. A variant of this model is the enclave or cluster model according to Gerhardt (2009). In this model, a job coach works with a small group of people with disabilities at a community location. A permutation of the enclave model is the work crew approach, where mobile work crews provide contracted services across a geographic area (e.g., cleaning services or lawn care). Entrepreneurial models involve 1–2 individuals with ASDs who develop a business based upon their own interests.

An example of a successful entrepreneurial model started by individuals with ASDs is an auto detailing business. The last two employment outcomes for people with ASDs provide the least integration. Day habilitation programs focus upon “pre-employment” skills and activities of daily living to individuals living in congregate care. There is little risk of being fired at this type of employment facility because of behavioral issues. “The least integrated work setting for individuals with ASDs is the sheltered workshop where the focus is solely upon providing enclave like employment experience for adults with developmental disabilities” (Gerhardt 2009, p. 23).

Qualifications of Treatment Providers

Traditional vocational training centers are often operated by local or county school districts and receive their auspice from either the state office of education or a state office of vocational and rehabilitative services. Supported academic programs and college-based CTPs operate under the auspices of a college or university. The college or university reports to the state office of education and also the Middle States Commission on Higher Education. Colleges must routinely go through a reaccreditation process through the Middle States Commission on Higher Education. Before applying to a college and enrolling in a supported academic program or college-based CTP, the consumer must insure the college is accredited. Otherwise, the degree or certificate granted may be worthless. College-based CTPs also are under the auspices of the US Department of Education if their students receive federal financial aid. College-affiliated CTPs are not necessarily regulated and do not report to a specific government agency or college administrative office. They are often private businesses.

In terms of the qualifications of instructional personnel at vocational training center, instructors should have the highest levels of certification possible in their trade. Support personnel in disabled student services offices will have a variety of related bachelor’s degrees, and some will have master’s degrees in fields such as higher

education administration, counseling degrees, and human resources. Because comprehensive transition and postsecondary programs provide a wider range of support services, the range of professional qualifications of the staff will be greater. The qualifications at a minimum should be a bachelor's degree in a helping or education profession. Many will have master's degrees in social work, education, special education, assistive technology, occupational therapy, nursing, etc. Key administrative personnel should have doctorate degrees in education, social work, vocational and rehabilitative sciences, and occupational therapy.

See Also

- [Apprenticeships](#)
- [Augmentative and Assistive Technology](#)
- [Individualized Plan for Employment \(IPE\)](#)
- [Individuals with Disabilities Education Act \(IDEA\)](#)
- [Vocational Rehabilitation Act of 1973](#)

References and Reading

- Assistive Technology Act of 2004, P.L. 108-364; 29 U.S.C 3001 et seq. Retrieved July 24 2011, from <http://www2.ed.gov/programs/atsg/legislation.html>.
- Baker, J. (2005). *Preparing for life: The complete guide for transitioning to adulthood for those with autism and Asperger's syndrome*. Arlington: Future Horizons.
- Barlow, M. (1976). 200 years of vocational education: 1776–1976; independent action, 1826–1876. *American Vocational Journal*, 51(5), 31–40.
- Bennett, D. (2009). Thorkil Sonne: Recruit autistics. *Wired Magazine* 17(10); September 21, 2009. Retrieved 5 Oct 2009, from: http://www.wired.com/techbiz/people/magazine/17-10/ff_smartlist_sonne.
- Edmonds, G., & Beardon, L. (Eds.). (2008). *Asperger syndrome and employment: Adults speak out about Asperger syndrome*. London: Jessica Kingsley.
- Freedman, S. (2010). *Developing college skills in students with autism and Asperger's syndrome*. London: Jessica Kingsley.
- Gerhardt, P. (2009). *The current state of affairs for adults with autism* (draft). Advancing Futures for Adults with Autism: Think Tank. January 21, 2009. New York.
- Gerhardt, P. (2011). Personal Communication.
- Grandin, T., & Duffy, K. (2008). *Developing talents: Careers for individuals with Asperger syndrome and high functioning autism*. Overland Park: Autism Asperger.
- Kochhar-Bryant, C., Bassett, D. S., & Webb, K. W. (2009). *Transition to post-secondary education for students with disabilities*. Thousand Oaks: Corwin.
- McDonnell, J., & Hardman, M. L. (2010). *Successful transition programs: Pathways for students with intellectual and developmental disabilities*. Thousand Oaks: Sage.
- Starr, P. (1982). *The social transformation of American medicine*. New York: Basic Books.
- U.S. Department of Labor, Bureau of Labor Statistics. (2011). *Occupation outlook handbook 2011*. Retrieved 24 July 2011, from <http://www.bls.gov/oco/>.
- VanBergeijk, E. O., Klin, A., & Volkmar, F. (2008). Supporting more able students on the autism spectrum: College and beyond. *Journal of Autism and Developmental Disorders*, 38, 1359–1370.
- Wehman, P. (2001). *Life beyond the classroom: Transition strategies for young people with disabilities* (3rd ed.). Baltimore: Paul H. Brookes.

Voice Output Communication Aids

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Definition

Voice output communication aids (VOCAs), also called speech-generating devices (SGDs), are high-tech, augmentative, and alternative communication (AAC) devices that produce speech for an individual who has limited or no means to communicate orally. These AAC systems produce a synthesized or digitized voice for the user. A digitized voice is a live voice that has been recorded and played back. Synthesized voices (also known as text-to-speech engines) are those that are created by programs called speech synthesizers. Different algorithms are used to create synthesized speech. Some programs use a database of recorded speech and then divide the sample into individual phonemes or diphones (two phoneme combinations) which are then combined to form spoken words. Alternately, whole words can be stored and reproduced. The benefits of a digitized live voice are the naturalness and intelligibility of the voice output. The drawback is the time and effort required to record a voice to

capture every message an AAC user would like to speak. Synthesized speech is judged in terms of naturalness and intelligibility. In the past, synthesized voices sounded very robotic, but the field has made great strides in creating synthesized voices that sound more natural.

VOCAs come in many different shapes and sizes and with a variety of features. The BIGmack (distributed by AbleNet) is a single message switch which produces a digitized voice. When the user presses the switch, the device plays a prerecorded message such as, “I’m hungry.” The BIGmack is an example of a simple VOCA for an individual who either does not need or cannot use complex communication. The Maestro (produced by DynaVox) is an example of a VOCA on the other end of the complexity continuum. The Maestro is a dynamic device in which the user can combine symbols to form long messages. Messages can also be stored for more efficient access in the future.

VOCAs are used with a variety of individuals with speech and language impairments. Some populations include individuals with autism, Down’s syndrome, spinal cord injury, traumatic brain injury, aphasia, dysarthria, amyotrophic lateral sclerosis (ALS), cerebral palsy, and apraxia of speech.

Historical Background

Early AAC strategies in the 1970s for individuals with autism spectrum disorders (ASDs) focused heavily on the use of sign language. The use of visual symbols with individuals with ASDs began in the 1980s. The use of VOCAs is a relatively recent development in the field of AAC mainly due to the wider availability of the technology.

For more information on the history of the field of AAC, see the entries on Alternative Communication, Pictorial Cues, and Total Communication Approach.

Rationale or Underlying Theory

The rationale for using VOCAs is to provide an individual with a voice. Mirenda (2001) described

some additional advantages for the use of VOCAs. These include the ideas that “they have the potential to be easily integrated into everyday environments with unfamiliar people” (p. 146) and their use can “facilitate natural interpersonal interactions and socialization by virtue of the speech output they provide” (p. 147).

The rationale for using augmentative and alternative communication can be found in the term itself. AAC is first augmentative. The purpose for this type of intervention is to augment or supplement the speech an individual naturally possess. For some individuals, however, this intervention is an alternative form of communication. These individuals have no means of verbal speech and so need to implement an alternative form. AAC is the means by which these individuals communicate.

Goals and Objectives

The goal for the implementation of VOCAs as with other forms of augmentative and alternative communication (AAC) is functional communication. However, communication with AAC is excruciatingly slow as it has been estimated that an average AAC user communicates at approximately 15 words per minute (Foulds 1987) compared to 150–250 words per minute for speakers (Goldman-Eisler 1986). Therefore, rate of communication should not be expected to occur as fast as spoken communication for individuals who use VOCAs.

Treatment Participants

Any individual who has impaired communication is a candidate for AAC in general. Therefore, because communication impairments are a hallmark of autism spectrum disorders (ASDs) (Mirenda 2009), most individuals with ASDs are candidates for a total communication approach. Specifically for VOCAs, many individuals with more severe impairments do not use the more complex or powerful devices (Rispoli et al. 2010). Developmentally, the strategy for many clinicians has been to introduce picture symbols first and then transition the child to a VOCA when

they are “ready.” Cress (2010) suggests that it is “never too early to introduce VOCAs.” She states that a child can use AAC to affect the world and that simple VOCAs can be programmed as cause/effect toys.

Treatment Procedures

According to a review by Rispoli et al. (2010), the two main methods of introducing VOCAs are discrete trial training (DTT) and milieu teaching. When requesting was the communication goal, Rispoli et al. (2010) found that DTT was the most common method of instruction, while milieu teaching was common when the goal was social interactions and conversational skills.

Discrete trial training is a procedure used in Applied Behavior Analysis (ABA) approaches. During DTT, the professional and the child work for short periods of time in a structured setting. Five steps make up each trial:

1. Cue – The professional provides the child with a stimulus.
2. Prompt – The professional provides assistance to the child. Hand-over-hand assistance is provided during the early stages of learning the behavior and is slowly faded.
3. Response – The child provides a response to the cue.
4. Consequence – The professional provides praise or a reward for a correct response or indicates to the child if an incorrect response was given.
5. Intertrial interval – A 1–5-s interval is given before the next cue.

Milieu teaching techniques are used during the child’s everyday activities and are often considered errorless learning. For example, a child who uses a VOCA may express a desire to join a game other children are playing. The professional may help the child choose the correct message on their device such as, “Can I play too?”

See the encyclopedia entries in this volume for more information on these treatment approaches.

Efficacy Information

Van der Meer and Rispoli (2010) conducted a review of intervention studies which used VOCAs with children with autism spectrum disorders (ASDs). The studies reviewed were conducted between 1998 and 2009. Most of the studies (83%) employed a single-subject research design. There were no instances of a large group design using randomized control trials. Requesting was the most frequently targeted communication skill. The majority of the studies (78%) had positive outcomes for the targeted communication skill and were also classified as demonstrating conclusive evidence. The authors cautioned that although encouraging, not all studies reviewed demonstrated conclusive evidence and so the results should be interpreted carefully. Generalization or some form of follow-up was only included in a small number of studies demonstrating a need for further research in this area. Overall, VOCAs were shown to be a “potentially effective option for teaching communication skills to children with ASD” (p. 304).

Outcome Measurement

Because the goal of any AAC use is functional communication, the outcome measurement should be the same. Functional communication of course will be defined differently based on the cognitive skills of the individual and the type of AAC system that is in place.

Qualifications of Treatment Providers

AAC interventions are most typically introduced by a speech-language pathologist. Unfortunately, many speech-language pathologists do not report having adequate training or education in the field of AAC (King 1998; Marvin et al. 2003; Simpson et al. 1999), and a survey of education programs for speech-language pathologists has uncovered a need for better education in this area (Ratcliff et al. 2008). Although this is the case, speech-language pathologists are the best equipped of all

professionals who work with individuals with autism to provide intervention that includes AAC. A listing of speech-language pathologists who are certified by the American Speech-Language Hearing Association can be found on their website. A few short questions posed to the speech-language pathologist can reveal whether they are comfortable with the area of AAC.

See Also

- [Alternative Communication](#)
- [Assistive Devices](#)
- [Communication Board](#)
- [Low-Technology Device](#)
- [Pictorial Cues/Visual Supports \(CR\)](#)
- [Total Communication \(TC\) Approach](#)

References and Reading

- Cress, C. J. (2010, April). *Strategies for incorporating AAC into basic communication interactions for children and adults*. Presentation at the annual meeting of the California Speech-Language-Hearing Association.
- Foulds, R. (1987). Guest editorial. *Augmentative and Alternative Communication*, 3, 169.
- Goldman-Eisler, F. (1986). *Cycle linguistics: Experiments in spontaneous speech*. New York: Academic Press.
- King, J. (1998). Preliminary survey of speech-language pathologists providing AAC services in health care settings in Nebraska. *Augmentative and Alternative Communication*, 14, 222–227.
- Marvin, L. A., Montano, J. J., Fusco, L. M., & Gould, E. P. (2003). Speech-language pathologists' perception of their training and experience in using Alternative and Augmentative Communication. *Contemporary Issues in Communication Sciences and Disorders*, 30, 76–83.
- Mirenda, P. (2001). Autism, augmentative communication, and assistive technology: What do we really know? *Focus on Autism and other Developmental Disabilities*, 16, 141–151.
- Mirenda, P. (2009). Introduction to AAC for individuals with autism spectrum disorders. In P. Mirenda & T. Iacono (Eds.), *Autism spectrum disorders and AAC*. Baltimore: Paul H. Brookes Publishing.
- Ratcliff, A., Koul, R., & Lloyd, L. L. (2008). Preparation in augmentative and alternative communication: An update for speech-language pathology training. *American Journal of Speech-Language Pathology*, 17, 48–59.
- Rispoli, M. J., Franco, J. H., van der Meer, L., Lang, R., & Camargo, S. (2010). The use of speech generating devices in communication interventions for individuals with developmental disabilities: A review of the literature. *Developmental Neurorehabilitation*, 13, 276–293.
- Simpson, K., Beukelman, D., & Bird, A. (1999). Survey of school speech and language service provision to students with severe communication impairments in Nebraska. *Augmentative and Alternative Communication*, 14, 212–221.
- Van der Meer, L. A., & Rispoli, M. (2010). Communication interventions involving speech-generating devices for children with autism: A review of the literature. *Developmental Neurorehabilitation*, 13, 294–306.

VPT

- [Visual-Motor Integration, Developmental \(VMI\) Test](#)

Wait Training

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Synonyms

[Tolerance for delay](#)

Definition

Wait training is synonymous with teaching “tolerance for delay.” For example, some people with ASD may become restless and disruptive when they are told to “wait” after making a request. Or, they may have difficulty waiting for a future activity to occur. It is first necessary to identify the situations and conditions that will be the focus of wait training. Next, a defined wait period is selected, usually a short duration a person can comfortably tolerate and then increased gradually over time. In illustration, a child or adult may be unable to wait before eating at a favorite restaurant. Wait training in this setting would begin by informing the person that she/he will have to “wait to be called” to the table. Training can be supplemented with visual cues such as a small card showing a red circle (“wait”) on one side and a green circle (“go”) on the other side. When displaying the red side of the card, a parent or care

provider would praise the person for “waiting” or possibly allow her/him access to preferred objects such as a music source (iPod) or book throughout the wait period. When it is time to be seated in the restaurant, the person would be shown the green side of the card, praised again, and allowed to go to the table. This final step of wait training, gaining access to the highly preferred activity, is intended to reinforce the preceding sequence of behaviors, namely, entering the restaurant, sitting quietly, and remaining occupied until the meal begins. With routine training, visual cues and social reinforcement eventually can be withdrawn so that an initial “wait” instruction is sufficient. Of note, training someone to wait in one setting or under certain conditions may not extend to other settings and conditions unless similar training is implemented there.

References and Reading

- Cooper, J. O., Heron, T. E., & Heward, W. L. (2007). *Applied behavior analysis* (2nd ed.). Upper Saddle River: Pearson-Merrill Prentice Hall.
- Durand, V. M. (2002). *Severe behavior problems: A functional communication training approach*. New York: Guilford Press.
- Reichle, J., Johnson, L., Monn, E., & Harris, M. (2010). Task engagement and escape maintained challenging behavior: Differential effects of general and explicit cues when implementing a signaled delay in the delivery of reinforcement. *Journal of Autism and Developmental Disorders*, 40, 709–720.

Warwick-Edinburgh Mental Wellbeing Scale (WEMWBS)

- [Autism Family Experience Questionnaire \(AFEQ\)](#)
- [Autism Family Experience Questionnaire \(AFEQ\), The](#)

WCST

- [Wisconsin Card Sorting Test \(WCST\)](#)

Weak Central Coherence

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Definition

The term “central coherence” refers to the “neurotypical” (NT, i.e., non-autistic) tendency to pull information together and process information in context, looking for the “big picture” and drawing out meaning, often at the expense of details. By contrast, “weak central coherence” refers to the tendency in ASD to attend to and remember details rather than global form or meaning.

Historical Background

The term “central coherence” was coined by Uta Frith in her influential 1989 book “Autism: Explaining the Enigma.” This drive for coherence, in Frith’s words, “pulls together large amounts of information” like the tributaries of a river, and “without this type of high-level cohesion, pieces of information would just remain pieces, be they small pieces or large pieces”

(p. 97). She hypothesized that the drive for coherence was weaker in people with ASD and suggested that this would make them *better* than neurotypicals (NTs) at some tasks. Together with Amita Shah, she demonstrated that people with ASD were better than IQ-matched comparison groups on the Block Design and Embedded Figures Tests, demonstrating facility in seeing the parts without being distracted by the whole picture.

The concept of weak coherence is reflected in Kanner’s first reports of autism as involving an “*inability to experience wholes without full attention to the constituent parts. . . A situation, a performance, a sentence is not regarded as complete if it is not made up of exactly the same elements that were present at the time the child was first confronted with it. If the slightest ingredient is altered or removed, the total situation is no longer the same and therefore is not accepted as such*” (Kanner 1943, p. 246). Kanner’s interest in global–local processing in ASD, and its relationship with insistence on sameness, is reflected in the title of his 1951 paper, “The conception of wholes and parts in early infantile autism.”

Current Knowledge

The notion that people with ASD have a more detail-focused processing style than NTs has become popular not only with researchers but also with teachers, parents, and those with ASD. A recent poster by the National Autistic Society in the UK showed a close-up of an eye, with the words, “*When a person with autism walks into a room, the first thing they see is: A pillow with a coffee stain shaped like Africa, a train ticket sticking out of a magazine, 25 floorboards, a remote control, a paperclip on the mantelpiece, a marble under the chair, a crack in the ceiling, 12 grapes in a bowl, a piece of gum, a book of stamps. . . so it’s not surprising they ignore you completely.*” The idea of weak coherence fits with descriptions by individuals with ASD of their own experience: for example, Gunilla Gerland (1997) writes, “*Every little bit of fact seemed to land in its own compartment in my head and refused to be linked*

with any other.” The emphasis on understanding strengths, as well as weaknesses, in ASD has been particularly welcomed and has led to “weak” coherence often being referred to instead as “detail-focused” processing.

Studies to date suggest that weak coherence is a cognitive *style*, not deficit, and may be best conceptualized as an information-processing bias towards local or featural information (Booth and Happé 2010). This bias can be overcome, just as NTs can overcome their preference for meaning to memorize “meaningless” information such as telephone numbers or bank codes. So, for example, people with ASC *can* read text for meaning, but unless asked to do so explicitly, their default approach may be to read a story as if it were just a list of words (e.g. not using context to disambiguate homographs; Happé 1997). A natural eye for detail appears to play a part in the unusually high rate of savant skills in ASC (Happé and Vital 2009), facilitating, for example, absolute pitch as a basis for further musical skills. Even the child with ASC who is terribly distressed by a familiar ornament being moved a fraction of an inch is demonstrating a special skill and attention to detail that few NTs possess.

Whether the good eye and memory for detail in ASD comes at the expense of processing the meaning and bigger picture is a topic of much debate. Several alternative accounts of superior local processing in ASC (including Baron-Cohen’s “hyper-systemizing” and Mottron’s “enhanced perceptual functioning” theories) propose that global processing is intact in ASD (Mottron et al. 2006). Recently, Happé and Booth (2008) have suggested that weak coherence may be the result of two somewhat independent aspects, superior featural processing and poor global processing, with different subgroups within ASD showing only the former, only the latter, or both, of these.

Studies to date suggest that detail-focused processing bias is not secondary to so-called executive dysfunction: impulsivity and poor planning in ADHD, for example, do not result in the characteristic assets or deficits associated with weak coherence in ASD (Booth et al. 2003). The relationship between weak coherence and “Theory of

Mind” deficits is less clear, although a degree of independence seems likely as, for example, some fathers of children with ASD show excellent eye for detail without accompanying social difficulties (Happé et al. 2001). Weak coherence may also be relevant to other special groups; for example, recent work suggests superior eye for detail in some women with eating disorders (Lopez et al. 2008).

To date, the brain basis of weak coherence is unclear, although the theory chimes well with suggestions of reduced functional connectivity, stronger local than long-range neural connections, and reduced top-down modulation (e.g., Belmonte et al. 2004). The working hypothesis is that weak coherence is among the features of the “broader autism phenotype,” inherited in many cases of ASC. Importantly, ongoing work suggests an overlap between genetic influences on ASC-like eye for detail and genetic influences on talent in maths, music, art, or memory (Happé and Vital 2009).

Future Directions

At least three areas are likely to be important in future research on weak central coherence. First, functional brain imaging studies are needed to better understand the neural basis of this cognitive style, in ASD and in the general population. Second, research on the link between detail-focused style and talent might inform educational approaches to encourage talent development in individuals with ASD and those without. Finally, an important aim for future research is to develop educational interventions based on the weak coherence hypothesis, helping children with ASD learn to see the bigger picture where needed while also capitalizing on their natural strength in noticing and remembering details.

See Also

- [Global Versus Local Processing](#)
- [Interests, Circumscribed/All Absorbing](#)
- [Verbal Semantic Coherence](#)

References and Reading

- Belmonte, M. K., Allen, G., Beckel-Mitchener, A., Boulanger, L. M., Carper, R. A., & Webb, S. J. (2004). Autism and abnormal development of brain connectivity. *The Journal of Neuroscience*, 24, 9228–9231.
- Booth, R. D. L., & Happé, F. (2010). “Hunting with a knife and... fork”: Examining central coherence in autism, attention deficit/hyperactivity disorder, and typical development with a linguistic task. *Journal of Experimental Child Psychology*, 107, 377–393.
- Booth, R., Charlton, R., Hughes, C., & Happé, F. (2003). Disentangling weak coherence and executive dysfunction: Planning drawing in Autism and ADHD. *Philosophical Transactions of the Royal Society (Special Issue: 'Autism: Mind and Brain')*, 358(1430), 387–392.
- Frith, U. (1989). *Autism: Explaining the enigma*. Oxford: Blackwell.
- Gerland, G. (1997). *A real person: Life on the outside* (English trans: Tate, J.). London: Souvenir Press.
- Happé, F. G. E. (1997). Central coherence and theory of mind in autism: Reading homographs in context. *British Journal of Developmental Psychology*, 15, 1–12.
- Happé, F. G. E., & Booth, R. D. L. (2008). The power of the positive: Revisiting weak coherence in autism spectrum disorders. *The Quarterly Journal of Experimental Psychology*, 61, 50–63.
- Happé, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 36, 5–25.
- Happé, F., & Frith, U. (Eds.). (2010). *Autism and talent*. Oxford: Oxford University Press.
- Happé, F., & Vital, P. (2009). What aspects of autism predispose to talent? *Philosophical Transactions of the Royal Society-Series B*, 364, 1369–1375.
- Happé, F., Briskman, J., & Frith, U. (2001). Exploring the cognitive phenotype of autism: Weak “central coherence” in parents and siblings of children with autism I. Experimental tests. *Journal of Child Psychology and Psychiatry*, 42, 299–307.
- Happé, F., Booth, R., Charlton, R., & Hughes, C. (2006). Executive function deficits in Autism Spectrum Disorders and Attention-Deficit/Hyperactivity Disorder: Examining profiles across domains and ages. *Brain and Cognition*, 61, 25–39.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kanner, L. (1951). The conception of wholes and parts in early infantile autism. *American Journal of Psychiatry*, 108, 23–26.
- Lopez, C., Tchanturia, K., Stahl, D., Booth, R., Holliday, J., & Treasure, J. (2008). An examination of the concept of central coherence in women with anorexia nervosa. *International Journal of Eating Disorders*, 41, 143–152.
- Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in

autism: An update and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36, 27–43.

Wechsler Memory Scale (All Versions)

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Description

The Wechsler Memory Scale (WMS) is a neurocognitive assessment tool designed for individuals aged 16–90 years old. Since memory functioning cannot be measured directly, it must be assessed via other cognitive skills, like verbal ability (Holdnack et al. 2011). The WMS measures different facets of human memory, including visual memory, auditory memory, and working memory.

Historical Background

Introduced in 1945, the original WMS has undergone several major revisions, including Russell's WMS-Revised (1975, 1988), the WMS-R (1987), WMS-III (1997), and the WMS-III Abbreviated (2002). Each of these revisions built upon the previous version and reflected evolving theories about memory. Published in 2009, the WMS-IV is the most current version and is designed to complement the Wechsler Adult Intelligence Scale (WAIS-IV); this is very helpful in cases where the goal of testing is to compare an individual's memory functions with his/her other cognitive abilities. The validation sample of the WMS-IV consisted of 100 participants per age band, for a total sample of 1400; the co-validation WAIS-IV/WMS-IV sample consisted of 900 individuals (Holdnack et al. 2011). These samples were stratified nationally by sex, education level, ethnicity, and region. The WMS-IV is innovative in that it

also includes an Older Adult Battery, a modified version of the WMS-IV specifically for examinees between the ages of 65 and 90 years old. The goals put forth by the publisher in designing the WMS-IV include: improving clinical sensitivity, decreasing testing time for older adults, improving forensic utility, reducing confounding factors, eliminating repetition or overlap with the WAIS, improving assessment of working memory, and improving ease of scoring and administration.

Psychometric Data

The WMS-IV consists of seven subtests, four of which are new to this edition and three that are adapted from the WMS-III. The subtests have generally high internal consistencies, ranging from 0.97 to 0.74, and are even higher among some clinical populations, such as individuals with Alzheimer's disease. The subtests are generally presented in the following order:

General Cognitive Screener

The Brief Cognitive Status Exam (BCSE) is a screener for significant cognitive impairment, which if present, could significantly impact test performance. The BCSE contains items that assess orientation to time, incidental recall, mental control, planning/visual perceptual processing, inhibitory control, and verbal productivity. Performance on this subtest is classified as Average, Low, Moderately Low, and Very Low. The BCSE was introduced in the WMS-IV, replacing the former Mental Control, and Information and Orientation subtests. Of the seven subtests of the WMS-IV, this is the only one that is considered to be optional.

Logical Memory

In Logical Memory I, the examinee is presented with information orally, in a story format, and is immediately asked to recall as many specific details about the story as possible. The general WMS-IV contains two stories; the Older Adult Battery contains only one story that is presented twice. In Logical Memory II, the examinee is asked to recall specific details about the

information following a 20–30 min delay, and also to answer some yes or no questions about it. Thus, Part I measures auditory immediate memory whereas Part II measures auditory delayed memory and auditory delayed recognition. Logical Memory was included in previous versions of the WMS and was modified slightly for the WMS-IV. These changes include eliminating a repetition of a story in the general battery, making small factual changes within a story to address clinician feedback, and including a new story in the Older Adult Battery. Logical Memory I has an internal reliability of 0.82 (0.86 for the Older Adult Battery) and Logical Memory II has an internal reliability of 0.85 (0.87 for the Older Adult Battery). The test-retest reliability of Logical Memory I is 0.72 (0.77 for the Older Adult Battery) and of Logical Memory II is 0.67 (0.71 for the Older Adult Battery).

Verbal Paired Associates

In Verbal Paired Associates I, the examinee is presented with a series of word pairs and is immediately asked to recall them, following a prompt including the first word of the pair. In Verbal Paired Associates II, the examinee is asked to recall these word pairs following a 20–30 min delay (again, following a prompt including the first word of the pair) and also to identify which pairs were presented earlier, from a list of words pairs. Part I of Verbal Paired Associates measures immediate learning of verbal associations and Part II measures delayed cued recall for these verbal associations, as well as long-term auditory recall. The California Verbal Learning Test – Second Edition (CVLT-II) is another measure that requires verbal list-learning and measures auditory verbal memory, and it is similar enough to the Verbal Paired Associates subtest that it is identified as an acceptable alternative to the WMS-IV. The CVLT-II can be used as a substitute for Verbal Paired Associates if an examinee has particular difficulty with words pairs that are not semantically related. Verbal Paired Associates was included in previous versions of the WMS and was modified significantly for the WMS-IV. These changes include the addition of easier items to increase the range of scores, and the

addition of Word Recall, an optional free recall condition that requires the examinee to recall, without cues, as many of the original word pairs as possible. Verbal Paired Associates I has an internal reliability of 0.94 (0.93 for the Older Adult Battery) and Verbal Paired Associates II has an internal reliability of 0.85 (0.74 for the Older Adult Battery). The test-retest reliability of Verbal Paired Associates I is 0.76 (also 0.76 for the Older Adult Battery) and of Verbal Paired Associates II is 0.76 (0.77 for the Older Adult Battery).

Designs

In Designs I, the examinee is presented with a series of grids that contain black and white geometric designs for 10 s. Next, the examinee is presented with grids containing new designs, and is asked to identify the locations of the previous designs within them. In Designs II, the examinee is presented with a series of grids and is asked to identify the locations of the original designs following a 20–30 min delay; additionally, the examinee is asked, in a multiple-choice format, to indicate which grids have the correct original designs and locations following a delay. This subtest differentiates one's capacity for visual details and spatial recall and was introduced in the WMS-IV, replacing the former Family Pictures and Faces subtests. The internal reliability of both Designs I and Designs II is 0.85; the test-retest reliability of Designs I is 0.73 of Designs II is 0.72. Designs is excluded from the Older Adult Battery.

Visual Reproduction

In Visual Reproduction I, the examinee is shown a series of five designs for 10 s each. Then, the picture is removed and the examinee is asked immediately to draw it from memory. In Visual Reproduction II, the examinee must draw the same target designs following a 20–30 min delay, without any cues. Also, the examinee must identify the target designs in a multiple-choice format following a delay. An optional condition requires the examinee to copy the original designs while the target design is displayed; this serves as a control for the examinee's visuospatial

skills. Overall, the Visual Reproduction subtest measures short- and long-term visual memory and long-term visual recall. It was included in previous versions of the WMS and was modified significantly for the WMS-IV. These changes include quicker and easier scoring rules for examiners, a return to a 7-item visual discrimination format, and the copy condition, described above. Visual Reproduction I has an internal reliability of 0.93 (also 0.93 for the Older Adult Battery) and Visual Reproduction II has an internal reliability of 0.97 (0.96 for the Older Adult Battery). The test-retest reliability of Visual Reproduction I is 0.62 (0.79 for the Older Adult Battery) and of Visual Reproduction II is 0.59 (0.64 for the Older Adult Battery).

Spatial Addition

In Spatial Addition, the examinee is shown a grid with a pattern of blue and/or red dots, which is immediately followed with a second grid with blue and/or red dots. In these trials, the blue dots are the target and the red dots are the distractor. The examinee then is given another grid, along with white cards and cards with blue dots, and is asked to perform addition, based on rules presented through the previous two grids, to show where the dots should appear on the new grid. This subtest measures spatial working memory and requires the storage and manipulation of visual information, as well as the ability to ignore competing stimuli. Spatial Addition was introduced in the WMS-IV, replacing the former Spatial Span subtest. The internal reliability of Spatial Addition is 0.91; its test-retest reliability is 0.74. Spatial Addition is excluded from the Older Adult Battery.

Symbol Span

In Symbol Span, the examinee is presented with novel symbols in a specific order and is then asked to identify them, in that order, on a page with many different symbols. This subtest measures visual-sequencing working memory, or the capacity to retain a mental image of a symbol and its relative spatial position. Symbol Span was introduced in the WMS-IV as a visual complement to the WAIS-IV subtest Digit Span. The internal

reliability of Symbol Span is 0.88 (0.84 for the Older Adult Battery) and its test-retest reliability is 0.72 (0.69 for the Older Adult Battery).

Older Adult Battery

The Older Adult Battery is a modified version of the WMS-IV that is specifically for examinees between the ages of 65 and 90. It does not contain the Designs and Spatial Addition subtests and requires only half the time as the complete WMS-IV (45 min, as opposed to 75–90 min). Because the Spatial Addition subtest is not administered, a Working Memory Index Score is not calculated for the Older Adult Battery.

Calculation of Index Scores

Subtest raw scores are converted into scaled scores that have a mean of 10 and a standard deviation of 3. Subtests contribute to five indices: Immediate Memory Index (Logical Memory I, Designs I, Visual Reproduction I, and Verbal Paired Associates I), Delayed Memory Index (Logical Memory II, Designs II, Visual Reproduction II, and Verbal Paired Associates II), Visual Working Memory Index (Spatial Addition and Symbol Span), Auditory Memory Index (Logical Memory I and II, and Verbal Paired Associates I and II), and Visual Memory Index (Designs I and II, and Visual Reproduction I and II). Standard scores are derived for each of these five indices, with a mean of 100 and a standard deviation of 15.

Index scores are the basis for interpreting WMS-IV performance as they are more psychometrically sound than individual subtest scores. The internal reliabilities of the indices are very high, ranging from 0.93 (Visual Working Memory Index) to 0.98 (Visual Memory Index) for the 16–90 battery, and from 0.92 (Delayed Memory Index) to 0.97 (Visual Memory Index) for the Older Adult Battery. The test-retest reliability of the indices is good, ranging from 0.79 (Delayed Memory Index) to 0.82 (Visual Working Memory Index) for the 16–90 battery, and from 0.79 (Visual Memory Index) to 0.84 (Immediate Memory Index) for the Older Adult Battery. The Immediate Memory Index indicates one's capacity to encode and retrieve both verbal and visual information very shortly after its presentation; the

Delayed Memory Index indicates one's capacity to perform the same functions but after a longer period of time. The Visual Working Memory Index captures one's ability to work with information that is presented visually, including retaining, organizing, and manipulating it. The Auditory Memory Index indicates one's capacity for encoding and retrieving information that is presented verbally, both immediately and following a delay, and the Visual Memory Index measures the same abilities, except for information that is visual in nature. Index-level contrast scaled scores are calculated to determine whether differences between index scores for a given examinee are statistically significant or within the expected ranges of variation.

Clinical Uses

There are a number of factors that can negatively impact one's performance on the WMS subtests, including disabilities (visual, auditory, physical, speech, and language), lack of familiarity with the testing format, and limited English proficiency. The balance of verbal and nonverbal tests helps to account for these factors. Also, conditions such as Alzheimer's disease, schizophrenia, traumatic brain injury, and intellectual disability have been associated with lower scores on the WMS. There are also factors that can falsely inflate one's performance. Memory and learning measures are subject to significant practice effects, in which one's performance increases with increased exposure to the testing materials. On the WMS-IV, these increases may approach one standard deviation. Therefore, retesting within a short time period (for example, 1–3 months) can lead to invalid results.

As the WMS-IV is a relatively new instrument, little research has documented how individuals with ASD perform on it. Twenty-two individuals with autism were included in the official validation sample of the WMS-IV and preliminary studies suggested that they, along with adults with schizophrenia and traumatic brain injury, performed in the Low Average range on Designs I and II. Additionally, participants with autism

performed significantly worse on the Spatial Addition and Symbol Span subtests than those without autism. It is important to keep in mind that these results are based on a very small sample and that additional research is needed to understand how individuals with ASD may perform on the new WMS-IV subtests.

Previous research compared the performance of individuals with ASD and that of individuals without ASD on the WMS-III. While people with and without ASD performed comparably on memory for words pairs, stories, and a verbal working memory task, individuals with ASD performed worse on immediate and delayed recall of faces and family scenes – which are not included in the WMS-IV – and on a test of spatial working memory (Williams et al. 2005). Other research has examined memory function in adolescents and young adults with ASD with measures other than the WMS. For example, Minshew and Goldstein (2001) found that individuals with ASD performed worse than those without ASD on some tasks comparable to those included in the WMS, including a list-learning task, and an immediate and delayed story recall, and that these results may have been due to weaknesses in higher-level mental organization and a failure to attend to contextual or semantic clues.

See Also

► [Memory](#)

References and Reading

- Delis, D., Kramer, J. H., Kaplan, E., & Ober, B. (2000). *The California Verbal Learning Test* (2nd ed.). San Antonio: The Psychological Corporation.
- Drozdzick, L. W., Holdnack, J. A., & Hilsabeck, R. C. (2011). *Essentials of WMS-IV assessment*. Hoboken: Wiley.
- Groth-Marnat, G. (2009). *Handbook of psychological assessment* (5th ed.). Hoboken: Wiley.
- Holdnack, J. A., & Drozdick, L. W. (2009). *Clinical and psychometric properties of the new WMS-IV*. Retrieved from http://www.pearsonassessments.com/hai/images/products/wms-iv/WMS-IV_Ins_Posters.pdf
- Holdnack, J. A., Zhou, X., Larrabee, G. J., Millis, S. R., & Salthouse, T. A. (2011). Confirmatory factor analysis of the WAIS-IV/WMS-IV. *Assessment*, 18(2), 178–191.
- Minshew, N. J., & Goldstein, G. (2001). The pattern of intact and impaired memory functions in autism. *Journal of Child Psychology and Psychiatry*, 42(8), 1095–1101.
- NCS Pearson Inc. (2009). *Interpretive report of WMS-IV testing*. Retrieved from http://www.pearsonassessments.com/NR/rdonlyres/BBB89AF8-CF8F-4E74-A374-B59471B3223D/0/WMSIV_Writer_Report_21yrMale.pdf
- Pearson Education Inc. (2008). *WMS-IV: Clinical features of the new edition* (PowerPoint slides). Retrieved from http://psychcorp.pearsonassessments.com/NR/rdonlyres/77AB9FD3-5181-4265-9C89-E00CEA5D0561/0/WMSIV_2_06_08.pdf
- Wechsler, D. (1997). *Wechsler memory scale* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2009). *Wechsler memory scale* (4th ed.). San Antonio: Pearson.
- Williams, D. L., Goldstein, G., & Minshew, N. J. (2005). Impaired memory for faces and social scenes in autism: Clinical implications of memory dysfunction. *Archives of Clinical Neuropsychology*, 20(1), 1–15.

Wechsler Preschool and Primary Scale of Intelligence

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Synonyms

[Psychological Assessment](#); [WPPSI-III](#); [WPPSI-III – Wechsler Preschool and Primary Scale of Intelligence, Third Edition](#)

Abbreviations

FSIQ	Full scale IQ
GLC	General language composite
IQ	Intellectual quotient
PIQ	Performance IQ
PSQ	Processing speed quotient
VIQ	Verbal IQ

Description

The Wechsler Preschool and Primary Scale of Intelligence – Third Edition (WPPSI-III) is an individually administered clinical instrument for assessing the intelligence of children aged 2 years 6 months through 7 years 3 months (2:6–7:3). Its age range is divided into two age bands (2:6–3:11 and 4:0–7:3) each with its own battery of subtests. The WPPSI-III provides composite scores that represent intellectual functioning in specified cognitive domains such as Verbal IQ and Performance IQ. The verbal areas are considered auditory/vocal tasks (auditory input and vocal output), and the performance areas are considered visual/vocal (visual input and vocal output) and visual/motor tasks (visual input and motoric output). The WPPSI-III also provides a composite score that represents a child's general intellectual ability (Full Scale IQ). All standard scores have a mean of 100 and a standard deviation of 15. All subtests yield a scaled score with a mean of 10 and a standard deviation of 3. Administration time is between 60 and 75 min in an individual format. Testing materials can be obtained through the publishing company (Pearson) to a qualified examiner. Computerized scoring and interpretation is available.

Structure

There are three types of subtests: core, supplemental, and optional.

Core. Completion of the core subtests yields Full Scale IQ (FSIQ), Verbal IQ (VIQ), and Performance IQ (PIQ). Composite scores can be judged in relation to norms and can guide clinical judgment.

Ages 2:6–3:11

- Receptive Vocabulary
- Information
- Block Design
- Object Assembly

Full Scale IQ = Verbal Domains, Performance Domains

Verbal = Receptive Vocabulary and Information

Performance = Block Design and Object Assembly

Ages 4:0–7:3

- Information
- Vocabulary
- Word Reasoning
- Block Design
- Matrix Reasoning
- Picture Concepts
- Coding

Full Scale IQ = Verbal Domains, Performance Domains, and Coding Subtest

Verbal = Information, Vocabulary, Word Reasoning

Performance = Block Design, Matrix Reasoning, Picture Concepts

Coding Subtest

Supplemental. These subtests can be used to substitute for core subtests and derive additional composite scores. Processing Speed Quotient (PSQ) for ages 4:0–7:3. General Language Composite (GLC) for ages 2:6–3:11.

Ages 2:6–3:11

- Picture Naming

General Language Composite (GLC) = Picture Naming and Receptive Vocabulary (from Core subtests)

Ages 4:0–7:3

- Symbol Search
- Comprehension
- Picture Completion
- Similarities
- Object Assembly

Processing Speed Quotient (PSQ) = Coding (from Core subtests) and Symbol Search

Optional. These subtests are not used to substitute for any core subtest.

Ages 4:0–7:3

- Receptive Vocabulary
- Picture Naming

General Language Composite (GLC) = Receptive Vocabulary and Picture Naming

Some subtests can be core, supplemental, or optional depending on the age of the child (e.g., Receptive Vocabulary subtest is a core verbal subtest for ages 2:6–3:11 but is optional for ages 4:0–7:3).

Supplemental Verbal subtests are Comprehension and Similarities. Either of these subtests may be substituted for one core Verbal subtests. Supplemental Performance Subtests are Picture Completion and Object Assembly. Either of these subtests may be substituted for one core Performance subtest. Symbol Search is a supplemental Processing Speed subtest and can be used to either substitute for the core Processing Speed subtest (Coding) or added to the Coding to then derive the Processing Speed Quotient (PSQ).

Optional subtests may be used to provide measure of expressive and receptive vocabulary ability in older children with language delays but may not be substituted for core Verbal subtests. Further, Receptive Vocabulary and Picture Naming allow the practitioner to calculate the General Language Composite.

Description of Subtests

Information. (2:6–7:3 Core, Verbal, 34 items including 6 picture and 28 verbal). This subtest is designed to assess a child's ability to acquire, retain, and retrieve general factual knowledge. This knowledge is referred to as a general fund of knowledge that includes crystallized intelligence, long-term memory, and the ability to retain and retrieve knowledge from school and the environment. General information is acquired from experience and education, remote verbal memory, understanding, and associative thinking. Other skills that could contribute include auditory perception and comprehension and verbal expressive ability. The picture ($n = 6$) items do not require a verbal response and were designed for the youngest children. They require the child to respond to a question by choosing a picture from a field of four commonplace objects. Verbal items require a brief verbal response from each child.

Vocabulary. (4:0–7:3 Core, Verbal, 25 items including 5 picture 20 verbal). This subtest is designed to assess a child's understanding of spoken words, learning ability, general range of ideas,

verbal information acquired from experience and education, and kind and quality of expressive language. Focus is on child's word knowledge and verbal concept formation that includes a child's fund of knowledge, learning ability, long-term memory, and degree of language development. Other abilities that could contribute include auditory perception and comprehension, verbal conceptualization, abstract thinking, and verbal expression. For the picture items, the child names the pictures that are displayed in a stimulus book (one per page). For the verbal items, the child gives definitions for words that the examiner reads aloud.

Word Reasoning. (4:0–7:3 Core, Verbal, 28 items). This subtest is designed to measure reasoning with verbal material and includes successive clues for the child to identify the underlying concept. The clues are in a series and are increasingly specific. This task assesses verbal comprehension, analogic and general reasoning ability, ability to integrate and synthesize different types of information, verbal abstraction, and domain knowledge, and the ability to generate alternative concepts.

Comprehension. (4:0–7:3 Supplemental, Verbal, 20 items). This subtest measures social judgment and maturity, common sense, verbal reasoning based upon experience, practical intelligence/knowledge of conventional standards of behavior, and conceptualization. This subtest measures the ability to evaluate and utilize past experience, verbal comprehension and expression, and the ability to demonstrate practical information. All items require the child to answer questions based on the understanding of general principals and social situations.

Similarities. (4:0–7:3 Supplemental, Verbal, 24 items). This subtest measures abstract and concrete verbal reasoning, concept formation, logical thought processes, associative thinking, and remote memory. It involves auditory comprehension, memory, the ability to distinguish between nonessential and essential features, and verbal expression. The subtest questions involve sentence completion with a response that reflects a shared characteristic. The child is read an incomplete sentence that contains two concepts sharing a common characteristic.

Receptive Vocabulary. (2:6–3:11 Core, Verbal/4:0–7:3 Optional, Verbal, 38 items). This subtest measures a child's understanding of spoken words, understanding of verbal directives, auditory and visual discrimination, auditory memory and processing, learning ability, general range of ideas, ability to integrate visual perception and auditory input, verbal information acquired from experience and education, and kind and quality of expressive language. Responses may be influenced by phonological memory and working memory. This subtest is relatively unaffected by emotional disturbance but is highly susceptible to cultural background and level of education. It is the best single measure of intelligence in the entire battery. The child looks at a group of four pictures and points to the one the examiner names aloud.

Picture Naming. (2:6–3:11 Supplemental, Verbal/4:0–7:3 Optional Verbal, 30 items). This subtest measures expressive language ability, word retrieval from long-term memory, and association of visual stimuli with language. The child names a picture that is displayed in the stimulus book.

Block Design. (2:6–7:3 Core, Performance, 20 items). This subtest measures the ability to perceive, analyze, synthesize, and reproduce abstract visual stimuli. It involves nonverbal concept formation, visual perception and organization, simultaneous processing, visual-motor coordination, learning, spatial relationships, and the ability to separate figure and ground in visual stimuli as well as the ability to plan and organize. For younger children, skills involve visual observation and matching ability as well as the ability to integrate visual and motor processes. Children are required to recreate a design created in the child's view within a specified time limit. This subtest is divided into two parts (A and B). Part A (items 1–10) involves one-color blocks and rotation is not penalized. Items 1–6 have two trials. Thus, it was designed for the youngest children. Part B (items 11–20) involves viewing and constructing a model or picture in a stimulus book, the use of one- or two-color blocks, and rotations of 30° or more are penalized.

Matrix Reasoning. (4:0–7:3 Core, Performance, 29 items). This highly reliable estimate of general intellectual ability measures fluid

reasoning. Matrix reasoning tasks are relatively culture-fair and language-free requiring no hand manipulation. This reliable measure of visual information processing and abstract reasoning skills utilizes four types of matrices. The first is continuous and discrete pattern completion, classification, analogic reasoning, and serial reasoning. The child looks at an incomplete matrix/grid and selects the missing piece to properly complete the matrix from 4 to 5 response options.

Picture Concepts. (4:0–7:3 Core, Performance, 28 items). This subtest measures fluid reasoning, perceptual organization, and abstract categorical reasoning without a verbal response. Items are developmentally sequenced to reflect increasing demands on abstract reasoning abilities. Easier questions are more concrete (e.g., color, shape, appearance), and the more difficult items require responses based upon more abstract representations (e.g., functions of an object). From each of two or three rows of object, the child selects objects that form a group with a common characteristic based upon an underlying concept.

Picture Completion. (4:0–7:3 Supplemental, Performance, 32 items). This subtest is designed to measure visual alertness to surroundings, remote visual memory, visual perception and organization, concentration, visual attention to detail, and ability to isolate essential from nonessential detail. All items require the child to view a picture and point to or name the important part missing from the picture.

Object Assembly. (2:6–3:11 Core, Performance/4:0–7:3 Supplemental, Performance, 14 items). This subtest measures immediate perception of a total configuration, integration and synthesis of part-whole relationships, nonverbal reasoning, and trial and error learning, spatial ability, visual-motor-spatial coordination, cognitive flexibility, and persistence. The child is presented with a standardized configuration of puzzle pieces and allowed 90 s to fit the pieces together to form a meaningful whole.

Symbol Search. (7:0–7:3 Supplemental, Processing Speed, 120 s). This subtest assesses visual discrimination, short-term visual memory, visual motor coordination, cognitive flexibility, and concentration. It also may involve auditory

comprehension, perceptual organization, and planning and learning ability. The child scans a search group and indicates whether a target symbol appears in the search group by marking the appropriate symbol with a pencil.

Coding. (4:0–7:3 Core, Processing Speed, 120 s). This subtest measures ability to associate meaning with symbols, visual-motor coordination and dexterity (pencil manipulation), flexibility and speed in learning tasks, short-term memory, visual perception, visual scanning ability, cognitive flexibility, attention, and motivation. It also may involve visual and sequential processing. The child copies symbols that are paired with simple geometric shapes. Using a key, the child draws each symbol in its corresponding shape.

Historical Background

In the early twentieth century, prevalent theories of intelligence emphasized a single, underlying construct of intelligence as largely responsible for an individual's performance on all mental tasks. The definition of intelligence, or cognitive ability, evolved as professionals developed ways to measure it. Indeed, experts have agreed that there are overwhelmingly three elements that make up the most important aspects of cognitive ability. They are the ability to deal with abstractions (ideas, symbols, relationships, concepts, and principles), the ability to solve problems (to deal with new situations, not simply to make well-practiced responses to familiar situations), and the ability to learn (the ability to grasp and use abstractions of the kind involving words and other symbols). Psychologists such as Spearman (1904) and Cattell (1941, 1957) introduced the theory that intelligence was comprised of two general factors. The first is fluid intelligence or generalized intelligence which is defined as the ability to perceive relationships independent of previous specific practice or instruction regarding these relationships. It is the ability to manipulate and think abstractly and solve problems. It is considered independent of learning. The second is crystallized intelligence which involves learning from contexts and materials previously learned (e.g.,

reading comprehension, vocabulary exams). This intelligence is based upon facts and rooted in experiences. It grows as people age and accumulate new knowledge and understanding.

Tests to measure intelligence, or cognitive ability, were developed in the latter half of the nineteenth century and reflected the social milieu in which they occurred. The majority of tests were developed for school-aged children, while the assessment of preschool-age children came later and is a relative newcomer in the history of testing.

In 1905, Binet and Simon developed tasks to measure the intelligence of children within the Paris public schools in response to the French government commission aimed at developing methods to identify children who would not benefit from regular education. These tasks were primarily language oriented, emphasizing judgment, memory, comprehension, and reasoning. After two revisions (1908, 1911), the scale extended from age 3 to 13 and included five adult tests. Others revised the assessment again in 1912, 1914, and 1916 and extended the assessment downward to 2 months of age (although these early tests were methodologically lacking) and to the United States (Terman and his colleagues). The primary focus of intelligence testing continued to be aimed at identifying intellectual deficiencies.

Around the time the US entered into World War I, the focus of intelligence testing began to change. There was a need to use an intelligence measure to screen recruits. The Army Alpha was created and included a large verbal component, and thus the Army Beta was subsequently developed to address the recruits with limited literacy skills to provide a nonverbal measure of intelligence.

In 1925, the understanding of “developmental schedules” began to evolve as Gesell identified four areas of development (motor, language, adaptive, and personal social) in 10 age levels (birth, 4, 6, 9, 12, 18, 24, 36, 48, and 60 months). This work helped guide the understanding of early developmental profiles which then were subsequently used in the development of tests for infants and preschoolers. Key infant and

preschool assessments of development and cognition were published in the first half of the twentieth century (Merrill-Palmer Scale of Mental Tests – Stutsman 1931; Minnesota Preschool Scale – Goodenough 1926; the Bayley California First-Year Mental Scale – Bayley 1933; and the Iowa Test for Young Children – Fillmore 1936); however, these assessments focused more on mental and physical growth than on intelligence. Tests for infant preschool *intelligence* were developed in the 1940s (Cattell Infant Intelligence Scale – Cattell 1940; Northwest Infant Intelligence Scale – Gilliland 1949; the Leiter International Performance Scale – Leiter 1948; the Full-Range Picture Vocabulary Test – Ammons and Ammons 1948; and the most widely used Stanford-Binet – 1949). These assessments were primarily designed as psychometric tools.

David Wechsler had strong clinical skills and statistical training with his extensive experience in testing gained initially from the World War I examinations and screenings in which he participated. His developed assessments were first introduced in the mid-1930s and weighted verbal and nonverbal abilities equally which was innovative at that time. As the social culture in the 1950s began to expand to the concept of identifying and diagnosing the nature of learning disabilities in children, the intelligence tests that focused on measuring more discrete aspects of an individual's cognitive functioning became very valuable. Wechsler's goals were to create batteries that yielded dynamic clinical information from his chosen set of tasks.

The original assessment was the Wechsler-Bellevue that was based upon the premise that intelligence is a global entity, characterizing the individual's behavior as a whole, and it is also specific because it is composed of elements or abilities that are distinct from each other. Thus, he developed subtests that highlighted the cognitive aspects of intelligence: abstract reasoning, perceptual organization, verbal comprehension, quantitative reasoning, memory, and processing speed. Wechsler's concept of intelligence was not a two-factor structure of intelligence. Instead, the two areas (verbal and performance) are different measures of intelligence, not measures of

different kinds of intelligence. He concluded that the Verbal and Performance subtests were only one of several ways in which the tests could be grouped.

In the 1960s, the US federal government became involved in education (e.g., Head Start), and this involvement warranted the assessment of preschool children. There was a need for effective program evaluation at the preschool level (both concurrent and predictive). In 1967, the Wechsler Preschool and Primary Scale of Intelligence (WPPSI) was developed to meet that need. It was a downward extension of the Wechsler Intelligence Scale for Children, and it had simpler items and an appropriately aged standardization sample; however, the age range was 4 years to 6 ½ years which did not address the need of early start programs. In 1975, again due to federal involvement in education through Public Law 94-142, the Education for All Handicapped Children Act, there was a need to evaluate and diagnose children's levels of functioning. These laws directly affected the continued development of standardized testing, and the WPPSI was revised in 1989 (WPPSI-R) and expanded the age range to include 3 years through 7 years 3 months as well as the addition of both easier and more difficult items to the original subtests. Age determined starting points were introduced to reduce the overall administration time. In 2002, a third revision (WPPSI-III) was published to first update the theoretical foundations by additional composite scores, enhancing the measure of fluid reasoning and incorporating measures of processing speed. Second, the assessment was revised to increase the developmental appropriateness by dividing the assessment into two age bands: 2 years 6 months to 3 years 11 months and 4 years to 7 years 3 months. Further, the instructions were simplified, the test materials were updated, time constraints and expressive language confounds were reduced, and items/queries/prompts were added as options. Third, the assessment was designed to enhance the clinical utility by increasing the number of special group studies (gifted, developmental delay, mental retardation, etc.), extending the age range, and updating statistical linkage to measures of achievement. Fourth,

psychometric properties were improved by updating the norms, by adding additional evidence of reliability and validity, by extending the floor and ceiling, and by reexamining bias. Finally, user-friendliness was increased by reducing the testing time, reorganizing the manual and the record form, and simplifying administration procedures. The WPPSI-III addresses the developmental period that involves some of the most profound changes in children's cognitive abilities.

Psychometric Data

Development and Standardization Procedures

The WPPSI-III development was a 4-year iterative process where each phase of development leads to further refinements of the scale. The AERA 1999 Standards for Educational and Psychological Testing served as primary resources. Test development occurred in five general stages: conceptual development, pilot, national tryout, standardization, and final assembly and evaluation.

Conceptual Development. An advisory panel with nationally recognized experts in school psychology, clinical psychology, neuropsychology, and cognitive development was assembled to work with the team throughout the process. Early in the development, a group of 106 professionals participated in a focus group to refine goals and assist in the formulation of the initial scales. Further, two telephone surveys were conducted with users of the WPPSI-R and professionals in childhood assessment. Finally, semi-structured surveys of other experts and examiners were conducted at all stages of test development to rate the research versions of the scale on such qualities as developmental appropriateness, user-friendliness, and clinical utility. In all, results were summarized and discussed, and at all stages of development, modifications to the working blueprint and research versions of the scale were integrated.

Pilot Stage. The goal of the pilot stage was to produce a version of the scale for use in the next stage (the national tryout stage). The focus was to

examine any content issues, relevance of items, adequacy of floors and ceilings, the clarity of instruction, identification of responses processes, administration, scoring, and item bias.

National Tryout Stage. The version used included 17 subtests, and data was obtained from a stratified sample of 424 children representatives of key demographic variables in the national population. Stratification occurred along age, sex, race, parent education level, and geographic region. Further, special group data were collected (e.g., groups of participants with mental retardation, intellectual giftedness, and language disorder as well as oversample of African American and Hispanic children) to provide additional understanding of the adequacy of the subtests and the clinical utility of the scale.

Standardization Sample. After collecting data from the national tryout stage, a standardization edition of the WPPSI-II was created. All preceding research questions were reexamined using similar research methods, but at this stage, the focus was on the derivation of norms and the provision of reliability, validity, and clinical utility. The WPPSI-III was standardized on a sample of 1700 children aged 2:6–7:3 who were chosen to match closely with the 2000 US census data on the variables of gender, geographic region (Northeast, South, Midwest, and West), parent education (0–8 years, 9–11 years, 12 years which was high school degree or equivalent, 13–15 years which was some college or associate's degree, and 16 or more), and ethnicity (Whites, African Americans, Hispanics, Asians, and other). The sample was divided into nine age-groups of 200 each (2:6–2:11, 3:0–3:5, 3:6–3:11, 4:0–4:5, 4:6–4:11, 5:0–5:5, 5:6–5:11, 6:0–6:11) and 7:0–7:3 which included 100 participants. The group was evenly split in gender.

Trained recruiters and independent examiners identified the children meeting the specified inclusion criteria for the standardization sample. Both the child and parents were paid an incentive fee for participation. Subjects were excluded if they were tested on any intelligence measure in the previous 6 months, had uncorrected visual and/or hearing loss, were not English speaking, had an upper extremity disability that would affect

motor performance, were admitted to a hospital or psychiatric facility, were taking medications that might depress test performance, or had a previously diagnosed physical condition or illness that might depress test performance.

Final Assembly. The consistency of the item sets, instructions, and stimulus material was evaluated throughout the development process and retained, modified, or deleted. Further, a special study was conducted to examine the possible effects of a new testing order using a within-subjects design. Sixty children aged 4:0–7:3 were administered 14 subtests in the proposed order for the final scale and matched to 60 children from the standardization sample, and no significant differences were found. To establish a quality assurance procedure, qualified examiners were found, a computer program automatically checked the values entered by scorers, and scoring studies were conducted to determine the underlying response process.

Statistical Properties

Reliability. Reliability refers to the accuracy, consistency, and stability of test scores across situations. True scores are hypothetical and defined as what an examinee would score if the test were perfectly reliable. The difference between a true score and an examinee's obtained score is measurement error. A reliable test would thus have very small measurement error as it would be consistent within one administration and on different occasions.

Internal Consistency. The evidence for internal consistency was obtained using the normative sample and the split-half method which was used in the WPPSI-R and divides subtest items with an odd-even split to form two half-tests. The reliability coefficient of the subtest is the correlation between the total scores of the two half-tests corrected by the Spearman-Brown formula for the full subtest. Coding and Symbol Search are timed subtests, and thus test-retest stability coefficients were used as reliability estimates. For the overall standardization sample, the average reliability coefficients range from .84 to .95 which are excellent ($\leq .90$) to good. The average internal consistency coefficients for Verbal IQ = .95,

Performance IQ = .93, Processing Speed Quotient = .89, General Language Composite = .93, and Full Scale IQ = .96.

Test-Retest Stability. The test-retest stability for the WPPSI-III was obtained on a sample of 157 children (13–27 children from each of the nine age-groups). The WPPSI III was administered a second time to establish stability to a group of children at an interval of a mean of 26 days (range 14–50 days). The sample was stratified (39.5% female, 66.2% White, 12.1% African American, 17.2% Hispanic, and 4.5% other). Average test-retest coefficients for the following were: Verbal IQ = .91, Performance IQ = .86, Processing Speed Quotient = .86, General Language Composite = .91, and Full Scale IQ = .92. The largest practice effects (scores increasing from first testing to second testing) across age bands were 5–6 points for Performance IQ and Processing Speech Quotient, while the average for all ages across the other domains were just under three points.

Validity. There are three major types of validity: content (does the assessment adequately sample relevant aspects of the construct being measured), criterion-related (scores are related to specified external criteria), and construct (when the construct measured by the test was actually measured).

The WPPSI-III research team established validity by utilizing evidence from the test content, the response process, the internal structure, and the relationship to other variables. First, using intercorrelation studies, all intersubtest correlations on the WPPSI-III were statistically significant. For both age bands, the presence of moderate to high correlations supports the premise that a general intelligence factor is present. Second, factor analytic studies were used to evaluate the internal construct structure of the WPPSI-III. For both age bands, results were consistent with the predicted factor model proposed. In short, the WPPSI-III is a two-factor test, Verbal and Performance. For ages 4:0–7:3, a third factor includes Processing Speed. When only the core subtests were analyzed, the WPPSI-III subtests each loaded on its predicted factor, with the exception of Picture Concepts at ages 6:0–7:3. It loaded onto

a Verbal subtest rather than on its intended factor (Performance). Finally, the relationship to other measures by locating constructions within a nomological network of known variables was conducted. The WPPSI-III was compared to the WPPSI-R, WISC-III, Bayley Scales of Infant Development-II, and Differential Abilities Scale. The global scales of these instruments correlated strongly with the WPPSI-III (.80–.89). The WPPSI-III Verbal Scale correlated substantially higher with the verbal scales of the WPPSI-R, WISC-III, and DAS than with the nonverbal scales of these instruments. The WPPSI IV and Wechsler Individual Achievement Tests II were both administered to 208 children to examine the relationship between the cognitive measure and academic achievement. The WIAT-II composites were variably correlated with the domain IQ scores of the WPPSI-III ranging from .31 (Processing Speed Index and WIAT-II Reading) to .78 (Full Scale IQ and Total Achievement WIAT-II).

Clinical Uses

Special Subgroups

Because the WPPSI-III is often used as part of a diagnostic assessment, its clinical utility and specificity was examined through a number of special group studies conducted concurrently with the scale's standardization. These samples were not randomly selected; instead, they were from a variety of educational and clinical settings. The sample sizes were small, and only group performance is reported. The following describes the special interest groups and their performance in the WPPSI-III:

- Intellectually gifted – this group obtained significantly higher Verbal, Performance, and Full Scale IQ than children in the normal population.
- Children with mild or moderate mental retardation. For the mild severity subgroup, composite score for the group for Full Scale IQ was 62.1. For the moderate severity subgroup, average Full Scale IQ for the group was 53.1.

- Children with developmental delays. Children in this group performed significantly worse than a matched control group with average Full Scale IQ score at 81.8.
- Children with developmental risk factors. On all domains, IQ ranged from 85.7 to 88.6 with the exception of Processing Speed where the average IQ for the group was 90.1.
- Children with autistic disorder. Consistent with research, the Performance IQ (mean 88.2) was significantly higher than the Verbal IQ (mean 70.6).
- Children with expressive language disorder. Both groups' mean composite score differences were greatest for Verbal IQ (mean 90.6) and Full Scale IQ (mean 90.1).
- Children with mixed receptive-expressive language disorder. Average Verbal IQ was 83.1, Performance IQ was 85.2, Processing Speed IQ was 82.7, Full Scale IQ was 81.9, and General Language Composite was 86.7 representing significantly lower mean scores than a matched control group.
- Children with Limited English Proficiency. No differences were found for Processing Speed and Performance IQ, but Verbal IQ averaged 80.2 and General Language Composite averaged 79.2.
- Children with attention-deficit hyperactivity disorder. In the research and clinical literature, traditional IQ scores have not been found to be useful in discriminating children and adults with ADHD. Children with the disorder typically achieve scores near the normative range. In this subsample, all scores (subtests and domain scores) were between 93.8 (Verbal IQ) and 97.4 (Performance IQ).
- Children with motor impairment. As expected, children with motor impairments scored significantly lower than the normative population on the Performance IQ (average 87.7) and marginally significantly lower on the Processing Speed IQ (average 89.9).

Results from the special group studies indicate that the WPPSI-III is valid and clinically useful (with the exception of the ADHD category, but this is not specific to the WPPSI-III but general to all intelligence tests).

Interpretation

- WPPSI-III scores should never be interpreted in isolation.
- Standard scores enable practitioners to compare scores within the WPPSI-III and between the WPPSI-III and other related measures.
- Age-corrected standard scores also allow for a comparison of children's cognitive functioning across other children of similar age.
- Other information (percentile ranks, descriptive classification, test-age equivalents) can also be used to benefit interpretation.

Profile analyses can be identified from both intraindividual and interindividual perspectives by comparing the child's score patterns across subtests or to the appropriate normative reference group. This can aid in identifying meaningful patterns of strengths and weaknesses.

See Also

- ▶ [Cognitive Skills](#)
- ▶ [Developmental Disabilities](#)
- ▶ [Intelligence Quotient](#)
- ▶ [Nonverbal Intelligence](#)
- ▶ [Psychological Assessment](#)
- ▶ [Verbal Intelligence](#)

References and Reading

- Ammons, R. B., & Ammons, H. S. (1948). *Full range picture vocabulary test*. Missoula: Psychological Tests Specialists.
- Bayley, N. (1933). *California first year mental scale*. Berkeley: University of California Press.
- Bayley, N. (1993). *Bayley scales of infant development* (2nd ed.). San Antonio: The Psychological Corporation.
- Binet, A., & Simon, T. (1905). A new method for the diagnosis of the intellectual level of abnormal persons. *L'Année Psychologique*, 11, 191–244.
- Cattell, P. (1940). *The measurement of intelligence of infants and young children*. Rolling Meadows: Riverside Publishing.
- Cattell, R. B. (1941). Some theoretical issues in adult intelligence testing. *Psychological Bulletin*, 38, 592.
- Cattell, R. B. (1957). *Personality and motivation structure and measurement*. New York: World book.
- Fillmore, E. A. (1936). *Iowa tests for young children* (Vol. 11). Iowa: University of Iowa Studies in Child Welfare.
- Gilliland, A. R. (1949). *Northwestern intelligence tests. Test A for infants 4–12 weeks old*. Boston: Houghton Mifflin.
- Goodenough, F. (1926). *Measures of intelligence by drawings, the Minnesota preschool scale*. New York: Arno Press.
- Leiter, K. (1948). *The Leiter international performance scales*. Los Angeles: Western Psychological Services.
- Lichtenberger, E. O., & Kaufman, A. S. (2002). *Essentials of WPPSI-III assessment*. Hoboken: Wiley.
- Sattler, J., & Dumont, R. (2004). *Assessment of children: WISC-IV and WPPSI-III supplement*. La Mesa: Sattler Publisher.
- Spearman, C. (1904). "General intelligence:" Objectively determined and measured. *The American Journal of Psychology*, 15, 201–293.
- Stutsman, R. (1931). *Merrill Palmer scale of mental tests*. Yonkers-on-Hudson: World Book Company.
- Terman, L. M. (1916). *The measurement of intelligence: An explanation of and a complete guide for the use of the Stanford revision and extension of the Binet-Simon intelligence scale*. Oxford: Houghton Mifflin.
- Wechsler, D. (1939). *Wechsler-Bellevue intelligence scale*. New York: The Psychological Corporation.
- Wechsler, D. (1949). *Wechsler intelligence scale for children*. New York: The Psychological Corporation.
- Wechsler, D. (1967). *Wechsler preschool and primary scales of intelligence*. New York: The Psychological Corporation.
- Wechsler, D. (1989). *Wechsler preschool and primary scales of intelligence-revised*. San Antonio: The Psychological Corporation.
- Wechsler, D. (1991). *Wechsler preschool and primary scales of intelligence* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (1997). *Wechsler adult intelligence scale* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2001). *Wechsler individual achievement test* (2nd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2002). *WPPSI-III technical and interpretive manual*. San Antonio: The Psychological Corporation.
- Woodcock, R. W., McGraw, K. S., & Mather, N. (2001). *Woodcock-Johnson III*. Itasca: Riverside Publishing.

Wechsler Test of Adult Reading

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Description

The *Wechsler Test of Adult Reading* (WTAR; Pearson 2001) was developed for estimating pre-

morbid (pre-injury) intellectual functioning and pre-injury IQ and memory abilities. The WTAR takes less than 10 min to administer and is appropriate for individuals between the ages of 16–89 years of age. It is composed of 50 words that have atypical grapheme to phoneme translations. A grapheme is a letter or set of letters that correspond to a sound, or phoneme, such as the *f* in the word fan, the *ph* in the word phone, or the *gh* in the word tough. The reason for relying on words that have unusual or irregular pronunciations is to test prior word knowledge by minimizing the individual's ability to apply standard pronunciation rules. Thus, the test attempts to rely on the individual's prior, or pre-injury, learning or knowledge of the word list that is presented. The rationale for this approach is that reading recognition is relatively stable, even when there is evidence of cognitive decline due to aging, illness, or brain injury.

The WTAR record form includes the WTAR stimulus words with correct pronunciations and item scoring with an area for recording demographic data, the individual's raw score, standard score, percentile rank, and a space for predicting pre-morbid IQ and memory functioning from the current WTAR performance.

An advantage of the WTAR is that it was co-normed with the United States (US) and United Kingdom (UK) versions of the *Wechsler Adult Intelligence Scale*®-Third Edition (WAIS®-III) and *Wechsler Memory Scale*®-Third Edition (WMS®-III). Estimating pre-morbid or pre-injury level of intellectual functioning may be useful in treatment planning. This co-norming allowed for the development of equations for predicting both the WAIS-III and WMS index scores from the WTAR. A large representative norming sample that was matched to both the US and UK populations was employed to provide demographic data that improves accurate prediction of pre-morbid IQ in neurological cases. There is extensive between group validity for individuals with Alzheimer's disease, Huntington's disease, Parkinson's disease, Korsakoff's syndrome, and Traumatic Brain Injury. However, there is limited information about individuals with autism spectrum disorders.

A key part of the rationale of the WTAR is the assumption of a normal development of reading skills prior to injury or cognitive decline. Individuals with ASD tend to perform well (and consistent with their level of cognitive ability) on mechanical reading and spelling (Minschew et al. 1994). Indeed, decoding or basic word identification, which is the skill that is assessed on the WTAR, is generally an area of strength amid poor reading comprehension (e.g., O'Connor and Hermelin 1994; Patti and Lupinetti 1993). Therefore, the WTAR may overestimate pre-morbid functioning. Just as assessment of single-word comprehension may overestimate general (sentence and paragraph) reading level (Newman et al. 2007), the WTAR may overestimate prior cognitive capacities. Moreover, given that a majority of individuals with ASD are routinely assessed throughout childhood and adolescence to determine their need for academic accommodations, assessment of pre-morbid functioning may be best accomplished through a review or earlier testing reports.

See Also

- [Reading](#)
- [Reading Skills](#)
- [Wechsler Preschool and Primary Scale of Intelligence](#)
- [Wechsler Memory Scale \(All Versions\)](#)

References and Reading

- Minschew, N. J., Goldstein, G., Taylor, H. G., & Siegel, D. J. (1994). Academic achievement in high functioning autistic individuals. *Journal of Clinical and Experimental Neuropsychology*, 16(2), 261–270.
- Newman, T. M., Macomber, D., Naples, A., Babitz, T., Volkmar, F., & Grigorenko, E. L. (2007). Hyperlexia in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(4), 760–774.
- O'Connor, N., & Hermelin, B. (1994). Two autistic savant readers. *Journal of Autism and Developmental Disorders*, 24(4), 501–515.
- Patti, P. J., & Lupinetti, L. (1993). Brief report: Implications of hyperlexia in an autistic savant. *Journal of Autism and Developmental Disorders*, 23(2), 397–405.

Wechsler, D. (1997). *Wechsler memory scale* (3rd ed.). San Antonio: The Psychological Corporation.

Wechsler, D. (2003). *Wechsler intelligence scale for children* (4th ed.). San Antonio: The Psychological Corporation.

Wechsler, David

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David Wechsler (Fig. 1) was born on January 12, 1896, the youngest of seven children, to Moses Wechsler, a merchant, and Leah (Pascal) Wechsler, a shopkeeper, in Lespedi, Romania. The family immigrated to the United States in 1902 when famine and poor economic conditions in Romania led to “worsening the scapegoating of Jews and resulting in severe applications of existing anti-Jewish decrees” (Wasserman 2012, pp. 30–31). He died at the age of 85 in New York City on May 2, 1981.

His experiences just before, during, and after America’s entry into the World War I paved the way for him to become the world’s leading expert in intelligence testing and clinical assessment. In 1917, working with testing pioneer Robert S. Woodworth at Columbia University, Wechsler earned his M.A. in experimental psychopathology



Wechsler, David, Fig. 1 Photo of David Wechsler (*Wechsler* is a trademark, in the United States and/or other countries, of Pearson Education, Inc. or its affiliate (s))

on retention in Korsakoff’s psychosis; that same year, he also worked under E. G. Boring, scoring army intelligence tests, as a civilian volunteer. After induction, while serving in the army’s psychology division in Fort Logan, Texas, Corporal Wechsler administered individual intelligence tests, including the Stanford-Binet, to recruits who could not be validly assessed by the army group tests (e.g., illiterates, suspected malingerers). In 1919, as an army student at the University of London, he worked closely with Karl Pearson (who developed the coefficient of correlation) and Charles Spearman (who promoted the theory of general intelligence or “g”). He then studied at the Sorbonne for 2 years, specializing in experimental and physiological psychology (Edwards 1994). By the time he earned his Ph.D. in 1925, under Woodworth at Columbia (on the measurement of emotional reactions via the galvanic skin response), his meteoric career was already on the rise. As Wasserman (2012) notes, “Columbia was one of the few major universities that provided graduate experimental psychology training with a willingness to address applied problems, termed *experimental abnormal psychology* by Woodworth” (p. 31) (and later called *clinical psychology*).

“In many ways, David Wechsler was an unexpected success – coming to the United States as a child amid a flood of Eastern European immigrants, losing both parents by the age of 10, compiling a relatively ordinary academic record in high school and college (while graduating early),”... “and not having become a naturalized citizen prior to the war. Even so, these risk factors may have been somewhat ameliorated by the following: (1) the guidance of his accomplished older brother, pioneering neurologist Israel S. Wechsler, who became his caretaker and role model; (2) the opportunity to provide military service as an army mental test examiner, which allowed him to thereby quickly learn about assessment and make key professional contacts; and (3) receiving his graduate education and professional psychology training at an opportune time and place in the development of what eventually would become clinical psychology.” (Wasserman 2012, p. 30)

In the mid-1920s, Wechsler was in the first wave of clinical psychologists, a breed of scientist-practitioners that represented a notable departure from the purely academic and experimental tradition of the American Psychological Association (APA). He was among the first to set up a private clinical practice and, in 1932, became chief psychologist at Bellevue Psychiatric Hospital, a post he held until 1967. Concurrently (1933–1967), he was a faculty member at New York University College of Medicine. Wechsler's test development techniques (combining verbal and nonverbal skills to produce a truly global measure of intelligence, a notion first sparked by scoring the group-administered Army Alpha and Army Beta, verbal and nonverbal tests, respectively); (Yoakum and Yerkes 1920) and his clinical approach to the assessment of intelligence are among the greatest innovations in applied psychology during the twentieth century. "Probably the work of no other psychologists, including Freud or Pavlov, has so *directly* impinged upon the lives of so many people" (Matarazzo 1981, p. 1542). French pioneer Alfred Binet developed the first intelligence test in 1905 (the Binet-Simon) and Stanford psychologist Lewis Terman translated and adapted Binet's work within the United States to produce the Stanford-Binet Intelligence Scale in 1916. However, it was American psychologist David Wechsler who dramatically, and permanently, changed the face of intelligence testing when he published the Wechsler-Bellevue (W-B), for ages 7–69 years, in 1939 (known in the literature as the W-B Form I because Form II was published in 1946).

Wechsler's intelligence (or "IQ") tests departed from Binet's and Terman's approach to measuring mental ability. Binet and Terman used mostly verbal tasks to measure intelligence, whereas Wechsler added a performance (nonverbal) scale to a verbal scale, with both contributing equally to a person's full-scale IQ. The Binet-Simon and Stanford-Binet offered a single score, global IQ, plus mental age (MA); the W-B offered verbal, performance, and full-scale IQs, along with a profile of scaled scores on 11 separate subtests (one of which, vocabulary, was an alternate and did not contribute to the IQs). The 1916 Stanford-Binet and its subsequent editions in 1937, 1960, and

1972 (coauthored by Maud Merrill) were routinely administered to children, adolescents, and adults; however, they were never standardized (normed) on adult populations. Consequently, when Wechsler published the W-B, standardized on children and adults (including older adults), he effectively developed the first real test of adult intelligence. And, in 1939, he replaced the ratio IQ that had become a staple in the Binet scales with the "deviation IQ." Ratio IQs were based on an old formula that compared mental age to chronological age and represented a "rubber yardstick" because 1 year's growth is quite different at age 5, 9, or 15; the outmoded formula also was not applicable to adults. By contrast, deviation IQs are standard scores with a mean of 100 and a standard deviation (SD) of 15 for all three IQs at all ages. They are derived from the concepts of the normal curve and standard deviations, which avoid problems associated with ratio IQs. Wasserman (2012) points out that "Wechsler deserves credit for popularizing the deviation IQ, although the Otis Self-Administering Tests and the Otis Group Intelligence Scale had already used similar deviation-based composite scores in the 1920s" (p. 35).

Wechsler's (1939) definition of intelligence has been widely quoted:

Intelligence is the aggregate or global capacity of the individual to act purposefully, to think rationally and to deal effectively with his environment. It is global because it characterizes the individual's behavior as a whole; it is an aggregate because it is composed of elements or abilities which, though not entirely independent, are qualitatively differentiable. By measurement of these abilities, we ultimately evaluate intelligence. But intelligence is not identical with the mere sum of these abilities, however inclusive. (p. 3)

In this definition, he shows the influence of Spearman's *g* on his theory but also makes it clear that there is more to intelligence than *g*. But Wechsler's lasting imprint was as a test developer, not as a theorist. And, ultimately, his greatest contribution may have been his clinical approach to the measurement of intelligence. When the Stanford-Binet reigned supreme prior to the

publication of the W-B, the main approach to test interpretation was *psychometric*, with the focus on the precise IQ, its percentile rank, the band of error surrounding the IQ, and group differences in mean IQ (e.g., urban versus rural children); the main book on Stanford-Binet interpretation was written by McNemar (1942), a statistician. Wechsler changed all that. He believed that intelligence was part of personality and that personality variables affected how a person performed on an IQ test. He encouraged examiners to interpret the IQs and subtest scaled scores within the context of the clinical behaviors observed during the evaluation and the reasons for referral (emotional disturbance, dementia) and to interpret responses to specific items (e.g., verbal answers on social comprehension questions) in terms of their clinical content. Thanks in large part to his experiences as an examiner during wartime, his clinical acumen, and his responsibilities at Bellevue Hospital, which brought him into one-on-one contact with individual patients from diverse backgrounds with an array of diagnoses, Wechsler (in conjunction with innovators such as Rapaport et al. 1945) changed IQ measurement from *psychometric testing* to *clinical assessment*. That distinction still characterizes the training of clinical psychologists and neuropsychologists worldwide.

An immediate benefit of the Wechsler approach to IQ testing was the large number of reliable and valid scores yielded by his tests – three IQs and an array of a dozen or so scaled scores on separate subtests. The full-scale IQ provides a global measure of intelligence (as was provided by the Stanford-Binet). However, the addition of separate verbal and performance IQs allowed examiners to determine whether a person was better able to express his or her intelligence via verbal comprehension and expression or nonverbally via the manipulation of concrete materials. And the subtest scaled scores – standard scores with a mean of 10 and SD of 3 for all subtests at all ages – permitted clinicians to examine a person's profile of strengths and weaknesses on cognitive tasks such as information, similarities, block design, and picture arrangement.

The separate IQs and the profile of scaled scores provided a breakthrough for researchers and clinicians who needed to go beyond a single, global IQ to better understand a person's abilities and disabilities. The global IQ masked areas of strength and weakness. The W-B, and later the 1949 Wechsler Intelligence Scale for Children (WISC) and 1955 Wechsler Adult Intelligence Scale (WAIS), spawned state-of-the-art research with patients diagnosed with neurological impairment in the left versus right hemisphere (Reitan 1955; Meyer and Jones 1957), with children and adolescents referred for reading and learning disabilities (Bannatyne 1971), and with individuals diagnosed with autism (Murata et al. 1974; Wassing 1965). The comparison of verbal IQ with performance IQ (V-P discrepancy) was especially crucial for understanding the strengths and weaknesses of individuals with left-hemisphere damage ($P > V$), right-hemisphere damage ($V > P$), and autism ($P > V$). The separate IQs also contributed mightily to understanding the differential effects of aging on intelligence (Feingold 1950; Jarvik et al. 1962) and to the theory of fluid and crystallized intelligence (Horn and Cattell 1966).

The profile of scaled scores was found to be particularly useful for identifying children with reading disabilities and for understanding the language problems of individuals with autism or other language disorders (e.g., comparing their performance on verbal subtests that required much verbal expression, such as vocabulary, with their performance on tasks like information that required little verbal expression). For reviews of studies on Wechsler profiles for children and adults diagnosed with autism spectrum disorders, including Asperger's disorder, consult Barnhill et al. (2000), Klin et al. (2000), and Lichtenberger and Greenberg (2009).

The Stanford-Binet remained the most popular IQ test into the early 1960s, even after the WISC and WAIS were published. However, as the fields of learning disabilities, neuropsychological assessment, and autism began to grow exponentially during the 1960s and started to dominate clinical practice in schools and clinics, Wechsler's scales surpassed the Stanford-Binet. By the time

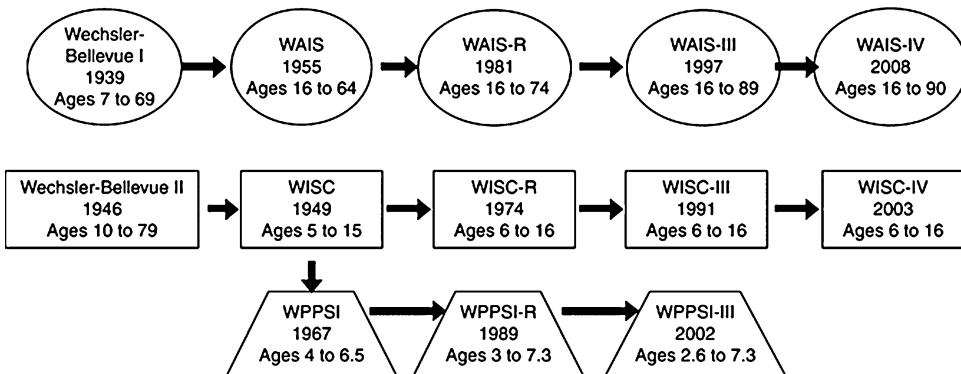
the WISC revision (WISC-R) was published in 1974, Wechsler's scales for children and adults were by far number one in the United States, and his scales have remained number one, worldwide, into the second decade of the twenty-first century. The WISC-IV and WAIS-IV are dominant, and the Wechsler Preschool and Primary Scale of Intelligence, Third Edition (WPPSI-III) is a popular choice for assessing young children. Fig. 2 shows the history of Wechsler's scales, from the 1939 W-B Form I to the 2008 WAIS-IV. Not shown in the figure is Wechsler's widely used *memory* test for adolescents and adults, the Wechsler Memory Scale (WMS), now in its fourth edition (WMS-IV).

"For his master's thesis completed in 1917, Wechsler patched together a clinical memory battery from existing published and unpublished tests [...] . . . a pattern he was later to follow with intelligence tests" (Wasserman 2012, p. 31). But his approach to test construction does not diminish his amazing innovations, including his insistence (in a Binet-dominated world) that nonverbal assessment must accompany verbal assessment to yield a truly global measure of a person's intelligence or his assertions that IQs and scaled-score profiles are only meaningful when interpreted within a rich clinical context, points that he introduced to the world with eloquence in his landmark articles and books (e.g., Wechsler 1928, 1930, 1935, 1939, 1944, 1950, 1958, 1971, 1975). And his genius did not diminish with age. When

I worked with Wechsler on the revision of the WISC in the early 1970s, "he was in his mid 1970s and as active and involved in his tests as ever. He showed me notebooks filled with new items, including comic strips he had cut out from newspapers to adapt for nonverbal test items. With his own tool kit, he had constructed a variety of wooden dolls and formboards, always in search of new ways of measuring mental ability." (Kaufman 2009b, p. 34)

He even analyzed himself: "After completing his fellowship at the Sorbonne, Wechsler traveled through France, Switzerland, and Italy, before reluctantly returning to the United States.... His ambivalence about returning, as disclosed to Edwards (1974), was reflected in his 1922 paper on the psychopathology of indecision" (Wasserman 2012, p. 33).

And his ideas were consistently decades, even generations, ahead of his time. Wechsler (1935) was an early advocate of measuring adaptive behavior skills, urging that daily behaviors, social demands, and functional living skills be considered alongside IQ test results before assigning a mental deficiency diagnosis. As J. D. Wasserman (personal communication, July 27, 2011) pointed out, Wechsler's approach is "entirely congruent with even contemporary standards for diagnosing intellectual disability (Schalock and The AAIDD Ad Hoc Committee on Terminology and Classification 2010)." He also reflected current philosophies by urging caution and



Wechsler, David, Fig. 2 History of Wechsler scales (Note. From *Essentials of WAIS-IV Assessment* (p. 8, "Don't Forget"), by E. O. Lichtenberger and A. S.

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sensitivity about the consequences of applying labels such as mental deficiency and genius based solely on IQ, because “Too much is at stake” (Wechsler 1971, p. 54).

In Wechsler’s lifetime, his scales were organized around the distinction between V and P IQs. Subsequent versions have emphasized four indexes, two of which replaced V IQ and P IQ (verbal comprehension index, or VCI, and perceptual reasoning index, or PRI), and two of which were new: working memory index (WMI) and processing speed index (PSI). L. G. Weiss (personal communication, July 26, 2011) noted that Wechsler’s “early notions on the importance of mental manipulation (Arithmetic/Digit Span) and mental speed (Digit Symbol/Coding) were borne out by modern research and subsequently fleshed out into WMI and PSI, which increased his test’s sensitivity to clinical disorders and extended his influence well into the twenty-first century.”

In a 1976 interview, Wechsler credited Woodworth and E. L. Thorndike as contributing most to his intellectual development, and he also praised Augusta Bronner and William Healy for refining his clinical skills: “they were both wonderful clinicians and they were the first, as I recall, who had discussions of every individual case at which first the social worker would present her history, then the psychiatrist, then the psychologist, and they either praised the individual and so forth on the basis of these conferences” (Hargus and Wasserman 1993).

References and Reading

- Bannatyne, A. (1971). *Language, reading, and learning disabilities*. Springfield: Charles C Thomas.
- Barnhill, G., Hagiwara, T., Myles, B. S., & Simpson, R. L. (2000). Asperger syndrome: A study of 37 children and adolescents. *Focus on Autism and Other Developmental Disabilities*, 15(3), 146–153.
- Edwards, A. E. (Ed.). (1974). *Selected papers of David Wechsler*. New York: Academic Press.
- Edwards, A. J. (1994). Wechsler, David (1896–1981). In R. J. Sternberg (Ed.), *Encyclopedia of intelligence* (Vol. 1, pp. 1134–1136). New York: Macmillan.
- Feingold, A. (1950). *A psychometric study of senescent twins*. Unpublished doctoral dissertation, Columbia University, New York.
- Hargus, M. E., & Wasserman, J. D. (1993). *Inside Wechsler’s library: Insights, incongruities, and the “lost WAIS items.”* Paper presented at the first annual South Padre Island interdisciplinary conference on cognitive assessment of children and youth in school and clinical settings, South Padre Island, TX.
- Horn, J. L., & Cattell, R. B. (1966). Refinement and test of the theory of fluid and crystallized intelligence. *Journal of Educational Psychology*, 57, 253–270.
- Jarvik, L. F., Kallman, F. J., & Falek, A. (1962). Intellectual changes in aged twins. *Journal of Gerontology*, 17, 289–294.
- Kaufman, A. S. (2009a). *IQ testing 101*. New York: Springer.
- Kaufman, A. S. (2009b). Preface—Dr. Wechsler remembered. In E. O. Lichtenberger & A. S. Kaufman (Eds.), *Essentials of WAIS-IV assessment* (pp. xiii–xixx). Hoboken: Wiley.
- Kaufman, A. S. (2010). Foreword. In L. G. Weiss, D. H. Saklofske, D. L. Coalson, & S. E. Raiford (Eds.), *WAIS-IV clinical use and interpretation* (pp. xiii–xxxi). San Diego: Academic Press.
- Klin, A., Sparrow, S. S., Marans, W. D., Carter, A., & Volkmar, F. R. (2000). Assessment issues in children and adolescents with Asperger syndrome. In A. Klin, F. R. Volkmar, & S. S. Sparrow (Eds.), *Asperger syndrome* (pp. 309–339). New York: Guilford Press.
- Lichtenberger, E. O., & Greenberg, D. (2009). Autistic-spectrum disorders. In D. P. Flanagan & A. S. Kaufman (Eds.), *Essentials of WISC-IV assessment* (2nd ed., pp. 242–250). Hoboken: Wiley.
- Matarazzo, J. D. (1981). Obituary: David Wechsler (1896–1981). *American Psychologist*, 36, 1542–1543.
- McNemar, Q. (1942). *The revision of the Stanford-Binet scale*. Boston: Houghton-Mifflin.
- Meyer, V., & Jones, H. G. (1957). Patterns of cognitive test performance as functions of the lateral localization of cerebral abnormalities in the temporal lobe. *Journal of Mental Science*, 103, 758–772.
- Murata, T., Nawa, A., & Okuma, H. (1974). Patterns of intelligence in autistic children: I. Analyses by the WISC. *Kyushu Neuro-Psychiatry*, 20, 206–212.
- Rapaport, D., Gill, M., & Schafer, R. (1945). *Diagnostic psychological testing* (Vol. 1). Chicago, IL: Year Book.
- Reitan, R. M. (1955). Certain differential effects of left and right cerebral lesions in human adults. *Journal of Comparative and Physiological Psychology*, 48, 474–477.
- Schalock, R. L., & The AAIDD Ad Hoc Committee on Terminology and Classification. (2010). *Intellectual disability: Definition, classification, and systems of supports* (11th ed.). Washington, DC: American Association on Intellectual and Developmental Disabilities.
- Terman, L. M. (1916). *The measurement of intelligence*. Boston: Houghton Mifflin.
- Terman, L. M., & Merrill, M. A. (1937). *Measuring intelligence*. Boston: Houghton Mifflin.
- Terman, L. M., & Merrill, M. A. (1960). *Stanford-Binet intelligence scale*. Boston: Houghton Mifflin.
- Terman, L. M., & Merrill, M. A. (1973). *Stanford-Binet intelligence scale: 1972 (norms editions)*. Boston: Houghton Mifflin.

- Wasserman, J. D. (2012). A history of intelligence assessment: The unfinished tapestry. In D. P. Flanagan & P. L. Harrison (Eds.), *Contemporary intellectual assessment: Theories, tests, and issues* (3rd ed., pp. 3–55). New York: Guilford Press.
- Wassing, H. E. (1965). Cognitive functioning in early infantile autism: An examination of four cases by means of the Wechsler intelligence scale for children. *Acta Paedopsychiatrica: International Journal of Child & Adolescent Psychiatry*, 32, 122–135.
- Wechsler, D. (1922). Quelques Remarques sur la Psychopathologie de l'Indecision [Some remarks on the psychopathology of indecision]. *Journal de Psychologie*, 19, 47–54.
- Wechsler, D. (1928). Psychometric tests. In I. S. Wechsler (Ed.), *A textbook of clinical neurology* (pp. 104–116). Philadelphia: W. B. Saunders.
- Wechsler, D. (1930). The range of human capacities. *The Scientific Monthly*, 31, 35–39.
- Wechsler, D. (1935). *The range of human capacities*. Baltimore: Williams & Wilkins.
- Wechsler, D. (1939). *The measurement of adult intelligence*. Baltimore: Williams & Wilkins.
- Wechsler, D. (1944). *The measurement of adult intelligence* (3rd ed.). Baltimore: Williams & Wilkins.
- Wechsler, D. (1945). A standardized memory scale for clinical use. *Journal of Psychology*, 19, 87–95.
- Wechsler, D. (1946). *The Wechsler-Bellevue intelligence scale, form II*. New York: The Psychological Corporation.
- Wechsler, D. (1949). *Manual for the Wechsler intelligence scale for children (WISC)*. New York: The Psychological Corporation.
- Wechsler, D. (1950). Cognitive, conative and non-intellective intelligence. *American Psychologist*, 5, 78–83.
- Wechsler, D. (1955). *Manual for the Wechsler adult intelligence scale (WAIS)*. San Antonio: The Psychological Corporation.
- Wechsler, D. (1958). *Measurement and appraisal of adult intelligence* (4th ed.). Baltimore: Williams & Wilkins.
- Wechsler, D. (1971). Intelligence: Definition, theory, and the I.Q. In R. Cancro (Ed.), *Intelligence: Genetic and environmental influences* (pp. 50–55). New York: Grune and Stratton.
- Wechsler, D. (1974). *Manual for the Wechsler intelligence scale for children-revised (WISC-R)*. San Antonio: The Psychological Corporation.
- Wechsler, D. (1975). Intelligence defined and undefined: A relativistic appraisal. *American Psychologist*, 30, 135–139.
- Wechsler, D. (1981). *Manual for the Wechsler adult intelligence scale-revised (WAIS-R)*. San Antonio: The Psychological Corporation.
- Wechsler, D. (1987). *Manual for the Wechsler memory scale-revised (WMS-R)*. San Antonio: The Psychological Corporation.
- Wechsler, D. (1989). *Manual for the Wechsler preschool and primary scale of intelligence-revised (WPPSI-R)*. San Antonio: The Psychological Corporation.
- Wechsler, D. (1991). *Manual for the Wechsler intelligence scale for children (WISC-III)* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (1997a). *Wechsler memory scale, (WMS-III)* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (1997b). *Wechsler adult intelligence scale (WAIS-III)* (3rd ed.). San Antonio, TX: The Psychological Corporation.
- Wechsler, D. (2002). *Wechsler preschool and primary scale of intelligence (WPPSI-III)* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2003). *Wechsler intelligence scale for children (WISC-IV) administration and scoring manual* (4th ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2008). *Wechsler adult intelligence scale (WAIS-IV)* (4th ed.). San Antonio: Pearson.
- Wechsler, D. (2009). *Wechsler memory scale* (4th ed.). San Antonio: Pearson.
- Yoakum, C. S., & Yerkes, R. M. (1920). *Army mental tests*. New York: Holt.

Wellbutrin

► Bupropion

Wernicke, Karl (1848–1905)

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Name and Degrees

- Carl Wernicke (May 15, 1848–June 15, 1905)
- Doctor of Medicine (Psychiatry)
- University of Breslau

Major Appointments (Institution, Location, Dates)

- Professor of Psychiatry and Nervous Diseases
- University of Halle
- Halle, Germany
- 1904
- Associate Professor of Neurology and Psychiatry
- University of Breslau

- Wroclaw, Poland
- 1885
- Scientist
- Department of Psychiatry and Nervous Diseases
- Berlin Charité Hospital
- Berlin, Germany
- 1878

Landmark Clinical, Scientific, and Professional Contributions

Wernicke is best known for his theory of aphasia, publishing a manuscript entitled “The Aphasia Symptom Complex” in 1874 when he was 26 years old. This publication catapulted Wernicke as the leader in aphasia research. Several years later, Wernicke published a textbook on brain diseases. In this book, he described a condition called “pseudoencephalitic haemorrhagica superior,” later to be known as “Wernicke’s encephalopathy.” In addition to his work on aphasia, Wernicke pioneered a technique for drainage of cerebral spinal fluid in patients with hydrocephalus.

Short Biography

Karl Wernicke was born in Wroclaw, Poland, in 1848. He studied medicine at the University of Breslau. Upon completion of his medical degree, Wernicke completed training in psychiatry. Wernicke worked under and collaborated with a variety of neuroscientists and neuropsychiatrists prior to designing his theory of aphasia. Wernicke discovered that damage to the left posterior, superior temporal gyrus resulted in deficits in language comprehension. This region of the brain is commonly referred to as “Wernicke’s Area,” and the syndrome in which damage to this region of the brain occurs often results in what is now known as “Wernicke’s aphasia.” Following Wernicke’s publication of his aphasia manuscript, he was appointed to the Department of Psychiatry and Nervous Disease of the Berlin Charité Hospital in 1878. During his time at the hospital, there was a significant discord between Wernicke and the administration. This conflict

forced Wernicke into private practice. Despite this conflict, Wernicke remained active in both clinical care and research activities. He continued to publish and pioneer new techniques in the field of neuroscience. In 1885, Wernicke became Professor of Psychiatry at Breslau Psychiatric Hospital at the university where he opened an outpatient clinic for individuals with neurological pathology. Unfortunately, there was tension between Wernicke and the hospital. Wernicke was ultimately forced to leave his position. In 1904, Wernicke was offered the Chair of Psychiatry and Nervous Diseases at University of Halle. Wernicke continued his work in neurological disease at the university until his untimely death due to a bicycle accident in 1905 at the age of 58. Although Wernicke died relatively early, his work continues to have significant influence in the fields of neurology and psychiatry.

See Also

- ▶ [Aphasia](#)
- ▶ [Psychiatrist](#)
- ▶ [Receptive Language](#)

References and Reading

- Lanczik, M., & Keil, G. (1991). Carl Wernicke’s localization theory and its significance for the development of scientific psychiatry. *History of Psychiatry*, 2, 171–180.
- Pillmann, F. (2003). Pioneers in neurology: Carl Wernicke (1848–1905). *Journal of Neurology*, 250, 1390–1391.

Wernicke’s Aphasia

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Synonyms

[Fluent aphasia](#); [Receptive aphasia](#); [Sensory aphasia](#)

Short Description or Definition

Wernicke's aphasia is a condition that is associated with damage to Brodmann area 22 in the left posterior superior temporal gyrus. The disorder is named for Karl Wernicke, the individual who is credited with first recognizing it. Wernicke's aphasia is also known as fluent aphasia or receptive aphasia as deficits tend to be in the content of the spoken message rather than speech output. As such, individuals with Wernicke's aphasia use sentences that are intact in terms of syntax, grammar, rate of speaking, and prosody but demonstrate difficulties with content such as vocabulary. Error patterns include word substitutions, additions, and difficulties with word order. In addition, patients demonstrate extreme difficulty in comprehending both what they themselves are saying and what is being said to them.

Categorization

Wernicke's aphasia is considered a fluent aphasia within larger aphasia classification systems.

Epidemiology

Estimates of the prevalence of Wernicke's aphasia in the larger population are largely unknown, though it has been estimated that 80,000 people develop aphasia in the United States each year.

Clinical Expression and Pathophysiology

Wernicke's aphasia often occurs as the result of damage to left posterior superior temporal gyrus. The condition manifests itself as a fluent aphasia in which an individual demonstrates relatively fluent language output. However, this output is characterized by the use of extra words, "made-up" words, or words that are out of order. In addition, language comprehension skills are largely impaired in individuals with Wernicke's aphasia.

Evaluation and Differential Diagnosis

Please see ► [“Aphasia”](#).

Treatment

Treatment for Wernicke's aphasia is often multifaceted and is typically individualized based on the patient's profile of strengths and needs. Individuals with aphasia often enroll in formal speech-language therapy to address functional communication in a variety of settings in which they are expected to communicate. Therapy goals are focused on maximizing the individual's ability to communicate effectively with peers and family members, given residual strengths. For individuals with Wernicke's aphasia, it is critical to support both comprehension and production; although speech/language output is fluent, patients often do not comprehend what they are saying or what is being said to them.

Although some individuals recover completely, individuals with aphasia often experience lifelong deficits. In these cases, family member and patient support groups are often a critical piece of the therapeutic process as the patient and family learn to manage their new situation.

Please see ► [“Aphasia”](#) for a list of general treatment strategies.

See Also

► [Verbal Auditory Agnosia](#)

References and Reading

- American Speech-Language-Hearing Association, ASHA. (2008). Incidence and prevalence of speech, voice, and language disorders in adults in the United States. Retrieved 1 May 2011 from www.asha.org/research/reports/speech_voice_language.htm
- Barresi, B., Goodglass, H., & Kaplan, E. (2001). *The assessment of aphasia and related disorders*. Hagerstown: Lippincott Williams & Wilkins.
- Chapey, R. (2008). *Language intervention strategies in aphasia and related neurogenic communication*

- disorders*. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins.
- Goodglass, H., Kaplan, E., & Barresi, B. (2000). *Boston diagnostic aphasia examination, third edition (BDAE-3)*. Austin: Pro-Ed.
- Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *The Boston naming test*. Philadelphia: Lea and Febiger.
- Kent, R. D. (1994). *Reference manual for communicative sciences and disorders: Speech and language*. Austin: Pro-Ed.
- Kertesz, A. (2006). *Western aphasia battery- revised (WAB-R)*. Austin: Pro-Ed.
- Lapointe, L. L. (2004). *Aphasia and related neurogenic language disorders*. New York: Thieme Medical.
- National Institute on Deafness and Other Communication Disorders. Aphasia. Retrieved 1 May 2011 from <http://www.nidcd.nih.gov/health/voice/aphasia.htm>

West Syndrome

- [Infantile Spasms/West Syndrome](#)

Whole-Genome Sequencing

- [Next-Generation Sequencing](#)

Wide Range Assessment of Memory and Learning (WRAML)

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Synonyms

[WRAML-2](#); [WRAML2](#)

Description

The WRAML2 (Sheslow and Adams 2003) is a clinical assessment instrument designed to evaluate memory and learning in children and adults (ages 5–90 years). The test is administered individually

with the examiner presenting items and recording responses. Three index scores are derived from six core subtests assessing verbal memory, visual memory, and attention-concentration. In addition, a general memory standard score can be calculated. Supplementary subtests provide the opportunity to assess working memory, recognition memory, and delayed recall. A more brief “memory screening” option is also available.

Content

In all, the WRAML2 is comprised of 6 core subtests and 11 optional subtests, generating 9 possible global index scores. Several subtests from the original WRAML (Sheslow and Adams 1990) are now optional (e.g., Sentence Memory) or limited to a specific age group (e.g., Sound Symbol for ages 5–8 years; Verbal Working Memory and Symbolic Working Memory for individuals 9 years of age and older). New additions include two subtests to assess working memory and four recognition memory tasks. In addition, items have been updated on existing subtests, such as contemporary full-color scenes on the Picture Memory subtest, revised stories in story memory (lengthened to accommodate adults), and more contemporary language on the Sentence Memory subtest. One subtest from the WRAML, Visual Learning, has been eliminated.

The core battery can be individually administered in under an hour, and a memory screening form, composed of four subtests, requires 10–15 min administration time and correlates highly ($r = .91$) with the full test.

The WRAML2 yields standard scores, scaled scores, and percentiles. The test also provides several qualitative analyses scores. Age equivalents are available for the child and preadolescent age groups. Subtest raw scores are converted into scaled scores based on a mean of 10 and standard deviation of 3. Index scores are derived from the sum of the relevant subtest scores and reported with a mean of 100 and standard deviation of 15 for age-based performance comparisons. A profile grid is provided allowing the examiner to plot scores across subtests and indexes for interpretation. A computerized scoring program is also available.

The global domains (underlined) and subtests (*italicized*) of the WRAML2 are described briefly below:

- *Verbal Memory Index*. An index of how well the individual is able to learn and recall both meaningful verbal information and relatively rote verbal information.
- *Story Memory Subtest*. A measure of memory for contextualized or meaningful verbal information. Two short stories are read to the examinee; following each story, the examinee is asked to orally recall as much of the story as he/she can remember.
- *Verbal Learning Subtest*. A measure of how well the individual actively learns, with practice opportunities, and is able to recall meaningful verbal information that is without context. The evaluator reads a list of common, single-syllable words followed by an immediate free recall trial; this list is repeated across four trials.
- *Visual Memory Index*. An index of how well the individual can learn and recall both meaningful (i.e., pictorial) and minimally related, rote (i.e., design) visual information.
- *Design Memory Subtest*. A measure of memory for visual material that is minimally meaningful. Five cards, each with an array of geometric shapes, are exposed, and then after a brief delay, the examinee is asked to draw what is remembered.
- *Picture Memory Subtest*. A measure of nonverbal immediate memory for contextualized information or meaningful information. The examinee is shown a colorful, everyday scene that he/she can scan for 10 s before it is removed. Then a similar alternate scene is immediately presented, and the participant must determine the elements that have been moved, changed, or added.
- *Attention/Concentration Index*. An index of how well the individual can learn and recall relatively nonmeaningful rote, sequential information. The tasks comprising this index require brief attentional demands and/or immediate rote recall abilities.
- *Finger Windows Subtest*. A measure of nonverbal, rote sequential recall; the examinee is asked to repeat gradually more difficult visual sequential patterns demonstrated by the examiner.
- *Number Letter Subtest*. A measure of auditory, rote sequential recall; the examinee is asked to repeat a series of items consisting of progressively longer number and letter sequences that are dictated by the examiner.
- *General Memory Index*. An index of overall memory functioning derived from performance on the six core subtests above.
- *Working Memory Index*. An index of how well the individual can operate on (e.g., add to, reorganize, or manipulate) and retain information that is held in the short-term memory buffer (i.e., in an active, online state).
- *Verbal Working Memory* (for ages 9 years and older). A measure of how well an individual retains information that is manipulated while it is in short-term memory. The examinee listens to a list of nouns that are both animals and nonanimals and, immediately thereafter, is required to recall the words in a specified reorganized order.
- *Symbolic Working Memory* (for ages 9 years and older). A measure of how well a person actively operates on and retains symbolic information (e.g., numbers, letters) prior to recall. Memory and attention are important for this task, and executive skills are also involved in finding an appropriate learning strategy.
- *Verbal Recognition*. An index of how well specific verbal information that was presented previously during this testing session is recognized following a short delay (15–20 min).
- *Story Memory Recognition*. A measure of recognition memory using a multiple-choice format with questions from each previously presented story. A comparison can be made between free recall (in the immediate recall and the delayed recall story memory formats) and recognition.
- *Verbal Learning Recognition*. Provides a measure of recognition recall of verbal information

presented previously as part of the verbal learning subtest.

- *Visual Recognition.* An index of how well the individual can recognize specific nonverbal or visual information that was presented previously in the session (i.e., approximately 15–20 min earlier).
- *Design Memory Recognition.* A measure of recognition recall of visual information that was viewed approximately 20 min earlier. The examinee is asked to look at and distinguish between designs that were and were not presented earlier in the testing session. It involves attention to visual detail and executive skills.
- *Picture Memory Recognition.* A measure of recognition of previously presented meaningful visual information or contextualized visual information, such as that found in pictures, in scenes, or in the environment.
- *General Recognition.* In contrast to memory retrieval as represented in the verbal memory index and the visual memory index scores, the general recognition index provides an estimate of how well the individual can *recognize* specific verbal and visual information that was presented previously (i.e., approximately 15–20 min earlier). This information is relevant to initial encoding strategies.
- *Delay Recall.* No global index score is provided for delayed recall; however, for each individual subtest, retention scores are generated providing a measure of memory decay over time.
- *Story Memory Delay Recall.* A measure of memory for a meaningful verbal narrative over a 10–15-min delayed interval.
- *Verbal Learning Delay Recall.* A measure of memory retrieval after the initial learning of new verbal information (approximately 15 min after initial learning).
- *Sound Symbol Delay Recall* (for ages 8 and younger). A measure of memory retrieval for paired associates (visual-auditory verbal) over a short delay.
- *Additional Subtests*
- *Sentence Memory.* A measure of immediate verbal memory skills similar to those needed

to follow novel verbal directions or to relay a brief phone message accurately. On this subtest, the examinee is required to repeat dictated single sentences of increasing length.

- *Sound Symbol* (for ages 8 and younger). A measure of paired-associate memory and learning. The examiner shows a symbol appearing on a page of the easel booklet and then identifies its sound that the child then repeats. The child is asked to try to recall that same sound when he/she sees the same symbol.

Historical Background

The first edition of the WRAML was published in 1990, filling a need for a comprehensive well-normed battery assessing children's memory functioning. The original WRAML was normed for children ages 5–17 years. The second edition, published in 2003, provides updated and extended norms (ages 5–90 years) as well as new test material. Focus groups were used to obtain first-hand information about improving the test; an item “tryout” was followed by a standardization program. Only one subtest (Visual Learning) was eliminated in the revision.

Psychometric Data

Norms. The standardization edition of the WRAML2 was administered to 1200 individuals across 22 states, representing different geographical regions. A national stratified sampling technique was used, controlling for age, sex, race, region, and education, with efforts to make the normative sample mirror the 2001 US Census. Slight variations in the normative sample from census data were corrected with a statistical weighting procedure.

Reliability. Scores from the WRAML2 are highly reliable. Reliability coefficients for the core battery Verbal Memory Index, Visual Memory Index, and attention/Concentration Index are .92, .89, and .86A, respectively. The alpha reliability for the general index is .93. Internal

consistency for the six core subtests is also shown to be very good, with Cronbach's alpha coefficients ranging from .82 to .96. The range is wider for the optional subtests (.63–.96). Test-retest reliability indicates a substantial learning effect from one test time to another (median administration lag of 49 days) with an overall gain of approximately +1 scaled score on subtests. Therefore, reliability correlations are lower with correlations between .53 and .85 for core subtests and indexes and .47 to .80 for optional subtests. Inter-rater reliability was quite high (.98).

Validity. Internal validity was assessed via examination of item content, subtest intercorrelations, exploratory factor analyses, confirmatory factor analysis, and differential item functioning. Item separation reliabilities, indicating how well the items define the variable being measured, were high (.90–1.00 for all subtests). Intercorrelations of the WRAML2 indexes and subtests were high for the older group (9 years to adult) but more variable in younger children. Results from factor analysis studies supported the internal validity of the WRAML2.

External validity was examined by comparing scores on the WRAML2 with related memory and learning measures, such as the Wechsler Memory Scale-III (WMS-III), Children's Memory Scale (CMS), and the California Verbal Learning Test-II (CVLT-II). Correlations were moderate (e.g., $r = .60$ for the WRAML2 General Memory Index score and the WMS-III General Memory Index score and $r = .49$ for the CMS). Moderate correlations were also found with cognitive measures (i.e., Wechsler Adult Intelligence Scale [WAIS-III] and the Wechsler Intelligence Scale for Children, Third Edition [WISC-II]) and with achievement tests (i.e., Wide Range Achievement Test 3 [WRAT3], and the Woodcock-Johnson, Third Edition [WJ-III] Tests of Achievement).

attentional difficulties, administration of the WRAML2 may help to clarify the contributions of attention, learning, and/or memory problems. This measure may also be used to assess memory impairment following head injury. The manual contains information about small studies involving learning disabled children and adults with traumatic brain injury, Alzheimer's disease, and alcohol abuse, suggesting WRAML2 sensitivity to these influences.

See Also

- [California Verbal Learning Test, Children's Version \(CVLT-C\)](#)
- [Memory](#)
- [Memory Assessment](#)
- [Memory Development](#)
- [Wechsler Preschool and Primary Scale of Intelligence](#)
- [Wechsler Memory Scale \(All Versions\)](#)
- [Woodcock-Johnson Cognitive and Achievement Batteries](#)

References and Reading

- Dunn, T. M., & Haynes, S. D. (2005). Test review of the Wide Range Assessment of Memory and Learning, Second Edition. In B. S. Plake & R. A. Spies (Eds.), *The sixteenth mental measurements yearbook [Electronic version]*. Retrieved from the Buros Institute's Test Reviews Online website: <http://www.unl.edu/buros>
- Sheslow, D., & Adams, W. (1990). *Wide range assessment of memory and learning*. Wilmington: Jastak Associates.
- Sheslow, D., & Adams, W. (2003). *Wide range assessment of memory and learning* (2nd ed.). Lutz: Psychological Assessment Resources.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary*. New York: Oxford University Press.

Clinical Uses

Learning and memory are fundamental to academic success. Therefore, an instrument such as the WRAML2 may be highly useful when evaluating individuals with a learning disability and/or school-related problems. For children with

Wide-Range Assessment of Visual-Motor Abilities

- [Visual-Motor Integration, Developmental \(VMI\) Test](#)

Wilbarger Protocol

- ▶ [Deep Pressure Proprioception Touch Technique](#)
-

Williams-Beuren Region Duplication

- ▶ [7q11.23 Duplications](#)
-

Wing Subgroups Questionnaire (WSQ)

- ▶ [Behavior Development Questionnaire](#)
 - ▶ [Behavioral Development Questionnaire](#)
-

Wing, Lorna

Adam Feinstein
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Landmark Clinical, Scientific, and Professional Contributions

In 1979, Lorna Wing and her Maudsley colleague, Judith Gould, published a landmark study of 173 children in Camberwell, South London, who had autistic features or had an IQ of under 50, or both. As Wing told Adam Feinstein in 2009, there were certainly some children who beautifully fitted Leo Kanner's 1943 criteria for autism, "but there was a huge collection in the middle who could not be put into either category. Very few fitted Asperger's syndrome, because they virtually all had an IQ of under 60 and none were mainstreamed." From their findings, Wing and Gould concluded that there was clearly a broader autism phenotype. This inspired them to introduce the concept of the triad of impairments in autism: deficits in social relations, communication, and imagination, a concept still used by diagnosticians today. Some researchers, like Francesca Happé at London's Institute of Psychiatry, maintain that, while the Wing-Gould version does indeed form

a true unitary triad, the DSM-IV and ID-10 notion of repetitive behavior – rather than imagination – as the third element has meant that the triad becomes "fractionable" (in the sense that the three elements may have separate causes at the genetic, neurological, and cognitive levels).

Short Biography

Dr. Lorna Wing, FRCPsych, born on October 7, 1928, is an English psychiatrist and physician and one of the world's leading autism authorities.

As a result of having an autistic daughter, Suzie, she became involved in researching developmental disorders, particularly autism spectrum disorders. She joined with other parents of autistic children to found the National Autistic Society (NAS) in the United Kingdom in 1962. She currently works part time as a consultant psychiatrist at the NAS Centre for Social and Communication Disorders at Elliot House.

Wing is the author of many books and academic papers, including *Asperger's Syndrome: a Clinical Account*, a 1981 academic paper that popularized the research of Hans Asperger and introduced the term "Asperger's syndrome." Although groundbreaking and influential, Wing herself cautioned in her 1981 paper that "it must be pointed out that the people described by the present author all had problems of adjustment or superimposed psychiatric illnesses severe enough to necessitate referral to a psychiatric clinic ... (and) the series described here is probably biased towards those with more severe handicaps."

References and Reading

- Burgoine, E., & Wing, L. (1983). Identical triplets with Asperger's syndrome. *British Journal of Psychiatry*, 143, 261–265.
- Feinstein, A. (2010). *A history of autism: Conversations with the pioneers*. Oxford, UK: Wiley-Blackwell.
- Wing, L. (1980). Childhood autism and social class: A question of selection? *British Journal of Psychiatry*, 137, 410–417.
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11(1), 115–129. <https://doi.org/10.1017/S0033291700053332>. PMID 7208735. Retrieved 15 Aug 2007.
- Wing, L. (1991). The relationship between Asperger's syndrome and Kanner's autism. In U. Frith (Ed.),

Autism and Asperger syndrome. Cambridge: Cambridge University Press.

- Wing, L. (1992). Manifestations of social problems in high functioning autistic people. In E. Schopler & G. Mesibov (Eds.), *High functioning individuals with autism*. New York: Plenum Press.
- Wing, L., & Attwood, A. (1987). Syndromes of autism and atypical development. In D. Cohen & A. Donnellan (Eds.), *Handbook of autism and pervasive disorders*. New York: Wiley.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11–29.
- Wing, L., & Potter, D. (2002). The epidemiology of autistic spectrum disorders: is the prevalence rising? *Mental Retardation and Developmental Disabilities Research Reviews*, 8(3), 151–161. <https://doi.org/10.1002/mrdd.10029>. PMID 12216059.

Books

- (1964). *Autistic children*.
- (1966). *Physiological measures, sedative drugs and morbid anxiety*, with M.H. Lader.
- (1969). *Children apart: Autistic children and their families*.
- (1969). *Teaching Autistic children: Guidelines for teachers*.
- (1971). *Autistic children: A guide for parents*.
- (1975). *Early childhood autism: Clinical, educational and social aspects* (editor).
- (1975). *What is operant conditioning?*
- (1988). *Aspects of autism: Biological research* (editor).
- (1989). *Hospital closure and the resettlement of residents: Case of Darenth park mental Handicap hospital*.
- (1995). *Autistic spectrum disorders: An aid to diagnosis*.
- (1996). *The autistic spectrum: A guide for parents and professionals*.
- Waites, J., & Swinbourne, H. (2002). *Smiling at shadows: A mother's journey raising an autistic child*.

Winkleman v. Parma City School District (2007 Rights Under IDEA)

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Definition

In General

In this case, the U.S. Supreme Court held that the Individuals with Disabilities Education Act (IDEA) grants parents independent, enforceable

rights. Parents are not limited to bringing claims on behalf of their child or claims based on procedural and reimbursement-related matters, but may sue to enforce their own rights to a “free appropriate public education” (FAPE) for their children.

The Supreme Court based its decision on statutory analysis, legislative intent, and policy considerations (Steiner 2008, p. 1172). IDEA entitles parents to voice their concerns as part of the individualized education program and (IEP) team as well as to submit a complaint concerning the adequacy of the education, the IEP’s construction, or other related matters. “Any party aggrieved,” including a parent, also has the right to bring a civil action based on that complaint. Additionally, IDEA’s express purpose is to protect “the rights of children with disabilities and parents of such children.” The court held that IDEA was intended to confer separate rights to parents of children with disabilities.

Implications for ASD Students

Since parents have their own independent rights under IDEA, a parent of a student with an autism spectrum disorder (added to IDEA’s list of disabilities in 1991), whether or not he or she can afford an attorney, now has the right to enforce his or her rights as a parent to obtain a quality education for their child. This makes IDEA a more substantive and robust reform of special education law (McNeal).

Litigation Strategies

While the decision appears clear-cut, it has led to significant confusion and problems in the lower courts (Zirkel 2009, p. 1). It triggers many larger issues, which are not resolved by the court. For example, there is no clear distinction between the child’s rights under IDEA and the rights of the parents and no sense of what remedy there is for a violation of parental rights. In addition, there is a concern that this decision allows parents to represent their children’s substantive interests pro se which violates common law principles and public policy considerations (Steiner 2008, p. 1181). These considerations seek to ensure that children are represented by those that can best represent them. Still, the decision does clearly allow parents

to lower the potential costs of litigation by suing without representation.

References and Reading

- Individuals with Disabilities Education Act, (IDEA) 20 U.S.C. §§1400, *et seq.* (2006).
- McNeal, L. (2010). Access granted: The Winkelman Case Ushers in a New Era in Parental Advocacy. *Brigham Young University Education and Law Journal*, 10, 129–147.
- Steiner, L. (2008). Playing lawyers: The implications of endowing parents with substantive rights under IDEA in *Winkelman v. Parma City School District*, 127 S. CT. 1994 (2007). *Harvard Journal of Law and Public Policy*, 31, 1169–1181.
- Winkelman v. Parma City School District, 550 U.S. 516 (2007).
- Zirkel, P. A. (2009, October 29). The problematic progeny of *Winkelman v. Parma City School District*. *West's Education Law Reporter*, 284, 1–17.

WISC

► WISC – V

WISC – V

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Synonyms

WISC

Description

The Wechsler Intelligence Scale for Children–Fifth Edition (WISC–V) is a standardized clinical assessment instrument. The clinical instrument assesses general cognitive abilities in children ages 6–16 years 11 months; specifically, it assesses general problem-solving and reasoning skills in verbal, nonverbal, and visual domains, as

well as working memory and processing speed in children and adolescents. This test is individually administered, scored, and interpreted by a trained clinician. Administration typically can be completed within 60–90 min. The WISC–V results in standard scores according to age-referenced norms from a national sample; standard scores can be calculated for specific cognitive indices as well as one or more global indicators of cognitive ability including a Full-Scale Intelligence Quotient (Full-Scale IQ).

The WISC–V framework consists of a Full-Scale Index (or Full-Scale IQ), in addition to primary, ancillary, and complementary indices that are made up of various combinations of primary, secondary, and complementary subtests that are the actual tasks administered by the clinician to the individual. The primary indices that form the core of the cognitive measure are Verbal Comprehension (VCI), Visual Spatial (VSI), Fluid Reasoning (FRI), Working Memory (WMI), and Processing Speed (PSI). These indices are the result of a five-factor model of cognitive abilities (Wechsler 2014). The five primary indices are derived from ten primary subtests: Vocabulary, Similarities, Block Design, Visual Puzzles, Matrix Reasoning, Figure Weights, Digit Span, Picture Span, Coding, and Symbol Search. Two primary subtests make up each index score, with complementary subtests rounding out a fuller measurement of the cognitive abilities of each index area.

The Verbal Comprehension Index (VCI) is comprised of the primary subtests, Similarities and Vocabulary, and the secondary subtests, Information and Comprehension. The VCI subtests assess an individual's ability to access and apply acquired word knowledge. The application of this knowledge involves verbal concept formation, reasoning, and expression. The Visual Spatial Index (VSI), which is comprised of two primary subtests, Block Design and Visual Puzzles, assesses an individual's ability to evaluate visual details and to understand visual spatial relationships to construct geometric designs from a model. The ability to construct designs requires visual spatial reasoning, integration and synthesis of part-whole relationships, attentiveness to visual detail, and visual-motor integration. The Fluid Reasoning Index (FRI) measures a person's

ability to detect the underlying conceptual relationships among visual objects and to use reasoning to identify and apply rules. Identification and application of conceptual relationships require inductive and quantitative reasoning, broad visual intelligence, simultaneous processing, and abstract thinking. The FRI is comprised of two primary subtests, Matrix Reasoning and Figure Weights, and two secondary subtests, Picture Concepts and Arithmetic. The Working Memory Index (WMI) assesses the ability to register, maintain, and manipulate visual information through the Picture Span subtest and auditory information through the Digit Span subtest. Letter–Number sequencing is a secondary subtest within the WMI that further assesses working memory through an auditory information task. The last primary index, Processing Speed, measures an individual's speed and accuracy of visual identification, decision-making, and decision implementation. Performance on the two primary PSI subtests, Coding and Symbol Search, and one secondary subtest, Cancellation, is related to visual scanning, visual discrimination, short-term visual memory, visuomotor coordination, and concentration.

In total, the WISC–V consists of 21 subtests, only ten of which are considered primary subtests and essential to calculating primary index scores. Seven of the ten primary subtests combine to arrive at the Full-Scale IQ (Similarities, Vocabulary, Block Design, Matrix Reasoning, Figure Weights, Digit Span, and Coding). The remaining 11 subtests are considered secondary or complementary. Secondary subtests (Information, Picture Concepts, Letter–Number Sequencing, Cancellation, Comprehension, and Arithmetic) can be used as a substitute for a related primary subtest to calculate the Full-Scale IQ. Additionally, secondary subtests are recommended for administration in order to provide more in-depth information about a child's intellectual functioning and for clinical decision-making, as they make up ancillary index scales. Complementary subtests (Naming Speed Literacy, Naming Speed Quantity, Immediate Symbol Translation, Delayed Symbol Translation, and Recognition Symbol Translation) are not

designed as measures of intelligence but rather to aid in assessment of cognitive processing associated with academic learning. The Complementary subtests comprise the complementary index scales of Naming Speed, Symbol Translation, and Storage and Retrieval.

The WISC–V provides global indicators of cognitive functioning via the Full-Scale IQ, as well as ancillary indices, specifically, the Nonverbal Index, General Ability Index, and Cognitive Proficiency Index. The ancillary indices may be interpreted as a better estimate of a given child's overall cognitive abilities than the Full-Scale IQ based on the individual's performance profile. More specifically, when there is an unusually high amount of variability between subtest scores that comprise the Full-Scale IQ, this Index score is not considered to be a good measure of the child's global intellectual ability, and it is recommended that other global estimates of cognitive functioning be reported. The Nonverbal Index is a measure of general cognitive ability that minimizes expressive language demands. The General Ability Index is an estimate of cognitive ability that reduces emphasis on working memory and processing speed, and it is thought to be a more pure representation of crystallized intelligence, which is heavily based on language and previously learned information. Lastly, the Cognitive Proficiency Index is the summary index made up of the four working memory and processing speed subtests, thus providing a global estimate of a child's cognitive efficiency for manipulating and rapidly processing information.

Historical Background

Intelligence tests were first developed in the early 1900s to more objectively assess and quantify aspects of disability and mental illness, in addition to qualifications or capabilities of an individual. There are various theories and definitions of intelligence and thus various theories on how to measure cognitive functioning (Sattler 2008). David Wechsler (see ► [Wechsler, David](#)), the creator of the original Wechsler scales that have evolved into the current WISC–V, considered intelligence to be

a global factor. He designed his original intelligence scale to consider factors contributing to the effective intelligence of the individual with the overall IQ score representing an index of general mental ability. Thus, from its outset, the Wechsler intelligence scales are rooted in a factor theory of intelligence – espousing to the theory of a general intelligence that is comprised of multidimensional cognitive abilities.

Wechsler developed the Wechsler–Bellevue Intelligence Scale in 1939 for use with adults, and in 1949, the first edition of the Wechsler Intelligence Scale for Children (WISC; Wechsler 1949) was created as a downward extension of the Wechsler–Bellevue scale. The WISC was the first intelligence test created for use in children, specifically children ages 5 to 15 years old (Sattler 2008). The original scales consisted of 12 subtests, many of which remain on the current version in an updated form. The subtests were organized into Verbal and Performance scales resulting in a Verbal IQ (VIQ), Performance IQ (PIQ), and Full-Scale IQ. In 1974, the first revision was produced, the Wechsler Intelligence Scale for Children–Revised (WISC–R; Wechsler 1974). The revision changed the targeted age range for the instrument to 6- to 16-year-old children. After Wechsler’s death in 1982, Psych Corp produced subsequent editions of the WISC and continues to list David Wechsler as the author through the current version. The third edition (WISC–III; Wechsler 1991) was the first revision to introduce significant content and structure changes to the measure. A new subtest for processing speed was developed for the WISC–III, and the measure was organized into more specific indices of cognitive functioning beyond the overarching composites of VIQ, PIQ, and FSIQ: Verbal Comprehension (VCI), Perceptual Organization (POI), Freedom from Distractibility (FDI), and Processing Speed (PSI). The next revision, WISC–IV, was developed in 2003 (Wechsler 2003). The development of the WISC–IV again ushered in major changes to subtest content and structure including updated item content and scoring procedures. The original VIQ, PIQ, and FSIQ were dropped and formally replaced with the specific indices developed on the WISC–III that were revised to reflect the

cognitive abilities assessed by the WISC: the Verbal Comprehension Index (VCI); the Perceptual Reasoning Index (PRI), formally named Perceptual Organization; the Working Memory Index (WMI), formerly named the Freedom from Distractibility; and the Processing Speed Index (PSI).

The WISC–V is the most recent revision of the WISC. One of the major changes in the new edition is the update to the theoretical foundation of cognitive functioning and intellectual assessment through the use of a five-factor model of test structure. The transition from a four- to five-factor model was born from factor analytic research of the WISC–IV and the Wechsler Adult Intelligence Scale–Fourth Edition (WAIS–IV; Wechsler 2008) (Benson et al. 2010; Keith et al. 2006; Weiss et al. 2013a, b), as well confirmatory factor analysis studies in the development of the WISC–V content (Wechsler 2014). The former Perceptual Reasoning Index was replaced by two indices, the Fluid Reasoning Index (FRI) and the Visual Spatial Index (VSI), to more accurately parse out cognitive abilities assessed within the WISC–IV PRI. Thirteen subtests were retained from the WISC–IV (Block Design, Similarities, Matrix Reasoning, Digit Span, Coding, Vocabulary, Symbol Search, Information, Picture Concepts, Letter–Number Sequencing, Cancellation, Comprehension, and Arithmetic). Eight new subtests were added (Figure Weights, Visual Puzzles, Picture Span, Naming Speed Literacy, Naming Speed Quantity, Immediate Symbol Translation, Delayed Symbol Translation, and Recognition Symbol Translation) to expand the cognitive abilities assessed by the measure and round out existing indices, while two subtests (Word Reasoning and Picture Completion) were dropped due to redundancy and limited utility. Thus the WISC–V consists of 21 subtests compared to the 12 subtests of the original WISC (Wechsler 1949) and 15 subtests on its immediate predecessor, the WISC–IV (Wechsler 2003). Furthermore, additional Ancillary and Complementary Indices were introduced to provide clinicians the opportunity to more robustly assess various cognitive abilities with the use of a single intelligence test. The new edition also provides updated normative data for scoring, reduced language in instructions,

changes in subtest scoring procedures, additional process subscores for subtests, and changes to score terminology and qualitative descriptors. There is also the ability to conduct an ability–achievement discrepancy analysis for various standardized achievement tests. Finally, the WISC–V introduces the capability to administer the measure via an iPad.

Psychometric Data

The WISC–V is a norm-referenced test that has been standardized on a clearly defined group and scaled so that each score reflects a rank within the normative group. Therefore, children are evaluated on how their performance compares with other children in their age group from a representative sample of children. After each subtest is scored, raw points are summed and converted to scaled scores according to normative data. An IQ is then computed by comparing the sum of scaled scores with the scores earned by a representative sample of peers in the same age group. Performance on the WISC–V indices, including Full-Scale IQ, is measured by a Standard Score. Standard Scores on the WISC–V have a range of 40–160, a mean of 100, and a standard deviation of 15. The WISC–V provides qualitative descriptors for specific ranges of Standard Scores: Extremely High (score 130 and above), Very High (120–129), High Average (110–119), Average (90–109), Low Average (80–89), Very Low (70–79), and Extremely Low (69 and below).

The WISC–V provides updated normative data collected from a representative sample of 2200 children stratified on the demographic variables of age, sex, race/ethnicity, parent education level, and geographic region from the years 2013 to 2014. The normative sample included typically developing children and children from the following identified special groups: intellectually gifted, intellectual disability–mild/moderate severity, borderline intellectual functioning, specific learning disorder – reading/reading and written expression/mathematics, attention deficit/hyperactivity disorder, disruptive behavior, traumatic brain injury, English language learners, and autism spectrum disorder with/without language impairment. Performance from the standardization

sample was used to inform starting points, discontinuity rules, and base rates for process scores, and the resulting raw scores were used to establish age equivalents and the normative standard scores for subtest and index scores for the measure.

The reliability and validity of the WISC–V has been broadly studied and is generally accepted as good. Rigorous reliability and validity analysis was conducted during test development of the WISC–V. WISC–V subtests demonstrate consistent or improved reliability from WISC–IV reliability findings (Wechsler 2014). Reliability of index scores is comparable or equal to corresponding indices on the WISC–IV. Importantly, the test’s reliability remains strong for special populations. Test–retest reliability for subtests is deemed adequately stable, while the primary index scores, with the exception of the Fluid Reasoning Index, demonstrate good to excellent stability (Wechsler 2014). WISC–V Full-Scale IQ scores also demonstrate excellent stability across two testing points. The WISC–V boasts very high interrater reliability, particularly for subtest scoring that requires greater examiner judgment (e.g., Similarities, Vocabulary, Information, and Comprehension) (Wechsler 2014).

The WISC scales have a long history of validity research with each revision, and the WISC–V builds on the strong foundation of content and construct validity for measuring intelligence (Flanagan and Harrison 2012; Flanagan and Kaufman 2009; Sattler 2008). The new five-factor structure of the WISC–V was based on current developments in intelligence theory and assessment, as well as confirmatory factor analytic research that further validated the five-factor structure of the WISC–V (Benson et al. 2010; Keith et al. 2006; Weiss et al. 2013a, b). WISC–V IQ scores were shown to correlate well with other established measures of intelligence, as well as having expected associations with other measures of a child’s functioning, such as academic achievement, adaptive skills, and behavior (Wechsler, 2014). The WISC–V reliability and validity extends to children with ASD with and without language impairment. Importantly, although the WISC–V as an instrument is deemed a valid tool for measuring an individual’s cognitive ability, the examiner administering the test to a given child

must assess whether the scores on that particular test are valid for that child based on the child's presentation, environmental, and testing factors.

Clinical Uses

The purpose of assessment includes screening, diagnosis, problem-solving, counseling and rehabilitation, and progress evaluation (see Psychological Assessment) (Sattler 2008). The WISC–V is used to assess and quantify a child's cognitive functioning in both global terms (Full-Scale IQ) and more specific cognitive abilities thought to comprise overall intelligence (i.e., Verbal Comprehension, Fluid Reasoning, Visual Spatial, Working Memory, and Processing Speed). Results from the WISC–V allow a clinician to describe a child's present cognitive functioning in reference to his or her peer group and to identify the nature of specific cognitive deficits and strengths to inform diagnostic conceptualization, and intervention planning, and progress.

In terms of diagnostic utility, the WISC–V is often used to establish a diagnosis of intellectual disability based on Full-Scale IQ score or another global index of cognitive functioning; the WISC–V is used in conjunction with assessment of an individual's adaptive behavior using a separate instrument to establish the presence and severity of an intellectual disability. The WISC–V also holds specific clinical utility for individuals with ASD. Psychological assessment that includes cognitive assessment is deemed the clinical standard of practice in the assessment of ASD as outlined by the American Academy of Child and Adolescent Psychiatry (AACAP) (Volkmar et al. 2014). Cognitive testing in ASD assessment is indicated to contextualize a child's social-communication difficulties and behavioral functioning relative to their overall development, as well as to inform treatment planning (Volkmar et al. 2014). Furthermore, in the *Diagnostic and Statistical Manual of Mental Disorders*, 5th Edition (DSM-5; APA 2013), diagnostic criteria and characterization for ASD include specifiers to describe a child's presentation, including level of intellectual functioning or impairment. Therefore, inclusion of a standardized intelligence measure, such as the WISC–V, is

essential to arrive at a comprehensive and complete diagnosis of ASD that includes use of specifiers and level of severity rating within the DSM-5 framework. Moving into treatment planning and service allocation, the WISC–V, or another standardized measure of intelligence, is often required as part of the documentation process to confirm and characterize a child's disability, including ASD. An assessment that includes cognitive testing is often required in order for a child and family to access related government-funded services at the national and state level. The Wechsler scales are widely accepted by government agencies as an established intelligence instrument for both children and adults.

It is important that clinical use of the WISC–V with children with ASD be undertaken by trained professionals who are knowledgeable about test content, competent in standardized administration and scoring procedures, and clinically sensitive to interpret the resulting scores with understanding of the strengths and weaknesses of the WISC–V in assessing this clinical subgroup. To enhance the clinical utility of the WISC–V, targeted analysis of test performance was conducted on children with ASD with and without language impairment (Wechsler 2014). Performance results for both special subgroups of children with ASD replicated cognitive data from previous iterations of the WISC, indicating the WISC–V continues to be sensitive to identifying and quantifying cognitive strengths and weaknesses of children with ASD. A common cognitive profile for children with ASD with language impairment is lower performance than typically developing peers across all primary indices of the WISC–V, with relatively weaker verbal task performance and relatively higher visual spatial task performance within their cognitive profiles. It is thought that children with ASD with language impairment perform lower on a unitary measure of intelligence (i.e., Full-Scale IQ) than matched typically developing children due to the greater global cognitive deficits associated with this ASD presentation (Klinger et al. 2012; Wechsler 2003, 2012, 2014). Children with ASD without language impairment tend to have WISC–V performance profiles that are relatively consistent with their matched typically developing peers, with the exception of a significantly lower score on the

Working Memory Index resulting in a lower Cognitive Proficiency Index score. Additionally, this clinical subgroup demonstrated greater differences at the subtest level compared to matched peers indicating deficits in working memory and processing speed tasks with subtle language weaknesses (Mayes and Calhoun 2008; Wechsler 2014). In general, it is considered sound clinical practice to deem test scores estimates or approximations of the domains assessed and never to focus exclusively on the child's test scores (Sattler 2008). Furthermore, there are various strategies a clinician can use when interpreting scores from the WISC–V, particularly in children with varied cognitive abilities (e.g., profile analysis, significant strengths and weaknesses relative to base rates, use of other global indicators of intelligence) (Sattler 2008). The WISC–V is typically used as one part of a multifaceted developmental or psychological assessment. Assessment conclusions and recommendations should be based on multiple sources of information obtained, and not based solely on the WISC–V.

See Also

- [Psychological Assessment](#)
- [Wechsler, David](#)

References and Reading

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Association.
- Benson, N., Hulac, D. M., & Kranzler, J. H. (2010). Independent examination of the Wechsler Adult Intelligence Scale—Fourth Edition (WAIS–IV): What does the WAIS–IV measure? *Psychological Assessment*, 22(1), 121–130.
- Flanagan, D. P., & Harrison, P. L. (Eds.). (2012). *Contemporary intellectual assessment: Theories, tests, and issues* (3rd ed.). New York: Guilford Press.
- Flanagan, D. P., & Kaufman, A. S. (2009). *Essential of WISC–IV assessment* (2nd ed.). Hoboken: John Wiley & Sons.
- Keith, T. Z., Fine, J. G., Taub, G. E., Reynolds, M. R., & Kranzler, J. H. (2006). Higher order, multisample, confirmatory factor analysis of the Wechsler Intelligence Scale for Children—Fourth Edition: What does it measure? *School Psychology Review*, 35(1), 108–127.
- Klinger, L. G., O'Kelley, S. E., Mussey, H. L., Goldstein, S., & DeVries, M. (2012). Assessment of intellectual functioning in autism spectrum disorder. In D. P. Flanagan & P. L. Harrison (Eds.), *Contemporary intellectual assessment: Theories, tests, and issues* (3rd ed., pp. 670–686). New York: Guilford Press.
- Mayes, S. D., & Calhoun, S. L. (2008). WISC–IV and WIAT–II profiles in children with high functioning autism. *Journal of Autism and Developmental Disorders*, 38, 429–439.
- Sattler, J. M. (2008). *Assessment of children: Cognitive foundations* (5th ed.). La Mesa: Jerome M. Sattler Publisher.
- Volkmar, F., Siegel, M., Woodbury-Smith, M., King, B., McCracken, J., State, M., & AACAP, C. G. I. (2014). Practice parameter for the assessment and treatment of children and adolescents with autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(2), 237–257.
- Wechsler, D. (1949). *Wechsler intelligence scale for children*. San Antonio: The Psychological Corporation.
- Wechsler, D. (1974). *Wechsler intelligence scale for children—revised*. San Antonio: The Psychological Corporation.
- Wechsler, D. (1991). *The Wechsler intelligence scale for children* (3rd ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2003). *The Wechsler intelligence scale for children* (4th ed.). San Antonio: The Psychological Corporation.
- Wechsler, D. (2008). *Wechsler adult intelligence scale* (4th ed.). San Antonio: Pearson.
- Wechsler, D. (2014). *The Wechsler intelligence scale for children* (5th ed.). Bloomington: The Psychological Corporation.
- Weiss, L. G., Keith, T. Z., Zhu, J., & Chen, H. (2013a). WAIS–IV clinical validation of the four- and five-factor interpretive approaches [Special Edition]. *Journal of Psychoeducational Assessment*, 31(2), 94–113.
- Weiss, L. G., Keith, T. Z., Zhu, J., & Chen, H. (2013b). WISC–IV and clinical validation of the four- and five-factor interpretive approaches [Special Edition]. *Journal of Psychoeducational Assessment*, 31(2), 114–131.

Wisconsin Card Sorting Test (WCST)

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Synonyms

WCST

Description

The Wisconsin Card Sorting Test (WCST) is a measure of executive function that is used both clinically and within research. The WCST requires the participant to sort a set of cards according to implicit rules and based on the limited corrective feedback provided by the examiner. The participant's responses can be analyzed to produce separate indices of sources of difficulty on the test. The value of the WCST, therefore, lies in its sensitivity for detecting and characterizing aspects of executive dysfunction.

The WCST consists of two identical sets of 64 response cards that differ on three possible perceptual sorting dimensions: color (red, blue, yellow, or green), form (crosses, circles, triangles, or stars), and number of figures (one, two, three, or four). Four target cards that represent the range of dimensions (one red triangle, two green stars, three yellow crosses, and four blue circles) are placed in a prescribed order in front of the participant and they are handed one set of the response cards. The participant is instructed to match each card in turn with one of the four target cards. The examiner has a preselected sorting dimension (color) that the participant has to deduce using corrective feedback (i.e., whether they are correct or incorrect on a given card sort). At no point is the participant explicitly told how the cards should be sorted. Once the participant has achieved ten consecutive correct matches the sorting dimension is changed by the examiner. The examiner makes no explicit reference to this change; rather, the participant needs to deduce the shift by using the corrective feedback. The test continues with a new sorting dimension being introduced (according to a prescribed order) each time the participant achieves ten consecutive correct matches. The test is completed when six sets of sorts have been successfully achieved or when the two sets of response cards have been used, whichever occurs sooner. During administration, each card sort is marked by the examiner for the dimensions that it matches (i.e., color, form, figure, others). Analysis of the participant's responses enables calculation of a range of scores. These include number of trials administered, total number of correct trials, number of errors (total,

perseverative, and non-perseverative), perseverative responses, conceptual level responses (an index of the participant's insight into the sorting principles), number of categories completed, trials to complete first category (an index of initial conceptualization before a shift in set is required), failure to maintain set (an index of the inability to maintain the correct sorting dimension), and learning to learn (an index of improved efficiency in deducing the correct sorting dimension).

Historical Background

The WCST was originally designed to measure abstract reasoning and cognitive set shifting in healthy adults (Berg 1948), but became popular within the clinical neuropsychological field for detecting frontal lobe dysfunction. Various iterations of the test have been published, some including significant differences in administration or scoring, which has made interpretation across studies difficult. The version of the manual published in 1981 (Heaton 1981) was presented as a definitive standardized version. This manual was revised and expanded in 1993 and is the basis of the description above (Heaton et al. 1993). This full version of the test should take between 20–30 min to administer. More recently, shorter versions of the WCST have been developed (Kongs et al. 2000; Schretlen 2010). Computerized versions, which are both administered and scored online, are also now available (Heaton and Staff 2003a, 2003b).

Psychometric Data

The manual produced by Heaton et al. (1993) includes detailed normative data for 899 individuals aged 6½–89 years. Normative tables provide T scores, standard scores, and percentile scores for percent and total error scores (total, perseverative, and non-perseverative) as well as the percent and total perseverative responses and percent conceptual level responses. Percentile ranges are available for the number of categories completed, trials to complete first category, failure to maintain set,

and learning to learn. Descriptive statistics are also available for adults with a range of cerebral lesions (frontal, frontal plus, diffuse, non-frontal) and for children and adolescents with cerebral lesions, traumatic brain injury, seizure disorder, attention deficit disorder, and reading disorder.

Clinical Uses

The profile of performance across the WCST is used to provide a nuanced measure of executive dysfunction. For example, the data provides information about the ability to learn and retain new information, perseveration, the ability to generate problem-solving strategies, concept formation, and conceptual set shifting. The test is used in a wide range of clinical populations where executive dysfunction might be present, including those with acquired brain injury, neurodegenerative disease, and schizophrenia.

See Also

- [Cognitive Flexibility](#)
- [Executive Function \(EF\)](#)
- [Frontal Lobe Syndrome](#)
- [Perseveration](#)

References and Reading

- Berg, E. A. (1948). A simple objective test for measuring flexibility in thinking. *Journal of General Psychology*, 39, 15–22.
- Heaton, R. K. (1981). *Wisconsin card sorting test manual*. Lutz: Psychological Assessment Resources.
- Heaton, R. K., & Staff, P. A. R. (2003a). *Wisconsin card sorting test: Computer version 4-research edition (WCST: CV4)*. Lutz: Psychological Assessment Resources.
- Heaton, R. K., & Staff, P. A. R. (2003b). *Wisconsin card sorting test-64: Computer version 2-research edition (WCST-64:CV2)*. Lutz: Psychological Assessment Resources.
- Heaton, R. K., Chelune, G. J., Talley, J. L., Kay, G. G., & Curtiss, G. (1993). *Wisconsin card sorting test manual: Revised and expanded*. Lutz: Psychological Assessment Resources.
- Kongs, S. K., Thompson, L. L., Iverson, G. L., & Heaton, R. K. (2000). *Wisconsin card sorting test-64 card version (WCST-64)*. Lutz: Psychological Assessment Resources.
- Schretlen, D. J. (2010). *Modified Wisconsin card sorting test (M-WCST)*. Lutz: Psychological Assessment Resources.

Within-Subject Design

- [Qualitative Versus Quantitative Approaches](#)

WJ-IV

- [Woodcock-Johnson Cognitive and Achievement Batteries](#)

WJ-IV Ach

- [Woodcock-Johnson Cognitive and Achievement Batteries](#)

WJ-IV Cog

- [Woodcock-Johnson Cognitive and Achievement Batteries](#)

WJ-IV OL

- [Woodcock-Johnson Cognitive and Achievement Batteries](#)

Wolfe, Sula

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Name and Degrees

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Major Appointments (Institution, Location, Dates)

Professor of Psychiatry, University of Edinburgh

Major Honors and Awards

- Member Royal College of Psychiatry (1957)
- Fellow Royal College of Psychiatry (1972)
- Honorary Fellow of Psychiatry, University of Edinburgh

Landmark Clinical, Scientific, and Professional Contributions

Dr. Wolff's clinical and scientific work centered on the responses to stress and the development of personality, particularly of children who were socially isolated. Her work on the concept of schizoid disorder was conducted just at the time of the groundswell of interest in Asperger's syndrome and other autism spectrum conditions. Her books *Children Under Stress* (Wolff 1969, 2nd edition 1981) and *Loners: The Life Path of Unusual Children* (Wolff 1995) are particularly notable. Her dedication to longitudinal follow-up of the children she had worked with enriched her research and publications. Her study of the children she originally termed schizoid noted that in

adulthood, many remained social impaired and a minority of cases had difficulties such as schizophrenia and other problems. She was the mentor to many students who became leaders in the field.

Short Biography

Sula Wolff, one of the founders of child psychiatry in Great Britain, was born in Berlin. Along with her family, she immigrated to the United Kingdom and enrolled at Oxford.

She studied medicine at Oxford and then served as a pediatrician before beginning training in psychiatry at the Maudsley Hospital and completed her training in Scotland. She also worked both in South Africa and the United States. She was the first child psychiatrist to work in South Africa. She began her long association with the Royal Hospital for Sick Children in Edinburgh in 1966. From 1947 to 1955 she held a range of appointments at various hospitals.

See Also

- [Asperger's Disorder](#)
- [Schizophrenia](#)

References and Reading

- Chick, J., Waterhouse, L., & Wolff, S. (1979). Psychological construing in schizoid children grown up. *The British Journal of Psychiatry*, 135, 425–430. Royal College of Psychiatrists, UK.
- Wolff, S. (1969). *Children under stress*. London: Penguin.
- Wolff, S. (1989). Schizoid disorders of childhood and adolescence. In C. G. Last & M. Hersen (Eds.), *Handbook of child psychiatric diagnosis* (Wiley series on personality processes, pp. 209–232). Oxford: Wiley.
- Wolff, S. (1991a). Asperger's syndrome [comment]. *Archives of Disease in Childhood*, 66(2), 178–179.
- Wolff, S. (1991b). Childhood autism: Its diagnosis, nature, and treatment. *Archives of Disease in Childhood*, 66(6), 737–741.
- Wolff, S. (1991c). "Schizoid" personality in childhood and adult life. III: The childhood picture. *The British Journal of Psychiatry*, 159, 629–635.

Wolff, S. (1995). *Loners: The life path of unusual children*. London: Routledge.

Wolff, S. (1999). Personality disorders. In S. D. Netherton, D. Holmes, et al. (Eds.), *Child and adolescent psychological disorders: A comprehensive textbook* (Oxford textbooks in clinical psychology, pp. 477–497). London: Oxford University Press.

Wolff, S. (2000). Schizoid personality in childhood and Asperger syndrome. In A. Klin & F. R. Volkmar (Eds.), *Asperger syndrome* (pp. 278–305). New York: The Guilford Press.

Wolff, S. (2004). The history of autism. *European Child & Adolescent Psychiatry*, 13(4), 201–208.

Woodcock Johnson Test of Achievement, Fourth Edition

► [Woodcock-Johnson Cognitive and Achievement Batteries](#)

Woodcock Johnson Tests of Cognitive Abilities, Fourth Edition

► [Woodcock-Johnson Cognitive and Achievement Batteries](#)

Woodcock Johnson, Fourth Edition

► [Woodcock-Johnson Cognitive and Achievement Batteries](#)

Woodcock-Johnson Cognitive and Achievement Batteries

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Synonyms

[WJ-IV Ach](#); [WJ-IV Cog](#); [WJ-IV OL](#); [WJ-IV](#); [Woodcock Johnson Test of Achievement, Fourth](#)

[Edition](#); [Woodcock Johnson Tests of Cognitive Abilities, Fourth Edition](#); [Woodcock Johnson, Fourth Edition](#)

Abbreviations

BIA	Brief intellectual ability
G	General intelligence
Ga	Auditory processing
GAI	General intellectual ability
Gc	Crystallized intelligence
Gf	Fluid intelligence
Glr	Long-term retrieval
Grw	Reading/writing ability
Gs	Processing speed
Gv	Visual-spatial thinking
Gwm	Short-term working memory

Description

The Woodcock Johnson, Fourth Edition (WJ-IV) is a comprehensive set of tests to assess intellectual ability, academic achievement, and oral language ability. The WJ-IV includes three batteries: (1) the WJ-IV Tests of Cognitive Abilities (WJ-IV Cog; Schrank et al. 2014d), which assess overall intellectual ability and specific cognitive abilities; (2) the WJ-IV Tests of Achievement (WJ-IV Ach; Schrank et al. 2014a), which assess academic achievement, and (3) the WJ-IV Tests of Oral Language (WJ-IV OL; Schrank et al. 2014b), which assess oral language. Each battery is composed of a number of subtests. The test items on any given subtest are ordered from easiest to most difficult, and the tests utilize basal and ceiling rules to limit the range of items to those best suited to the examinee’s ability level. All stimulus materials are presented in a stimulus book, with no use of manipulatives. Some subtests involve paper and pencil or listening to an audio recording. A complete description of subtests included in each of the batteries follows.

WJ-IV Cog Battery

The WJ-IV Cog battery is composed of eighteen subtests; ten of these make up the standard battery, while an additional eight make up the extended

battery (McGrew et al. 2014). The standard battery includes the following subtests:

Subtest 1: Oral vocabulary. This task includes two parts: synonyms and antonyms. It provides a measure of lexical knowledge and language development.

Subtest 2: Number series. On this task, the examinee is presented with a series of numbers and is required to determine the numerical pattern in order to identify the missing number. This is a measure of quantitative reasoning.

Subtest 3: Verbal attention. This subtest requires the examinee to listen to a series of animal names and digits and then answer specific questions regarding their sequence. This provides a measure of attentional control and auditory working memory.

Subtest 4: Letter-pattern matching. This is a 3-min task that requires the examinee to locate and circle identical letter patterns as quickly as possible. This subtest becomes increasingly difficult, moving from single- to four-letter patterns. This is a measure of perceptual speed.

Subtest 5: Phonological processing. This subtest consists of three parts: (1) word access, which requires the examinee to say a word that has a specific beginning, middle, or ending sound; (2) word fluency, which requires the examinee to name as many words that he/she can recall within 1 min; and (3) substitution, which requires the examinee to substitute part of a word to create a new word. It measures phonetic coding and the speed of lexical access.

Subtest 6: Story recall. In this subtest, the examinee listens to a story and then must answer questions about the story. This measures listening ability, narrative comprehension, and recall.

Subtest 7: Visualization. This task is composed of two parts: (1) spatial relations, where the examinee must determine which puzzle pieces create a target shape, and (2) block rotation, where the examinee is presented with a series of three-dimensional patterns and must identify which two patterns would match the target pattern when rotated. This provides a measure of visual processing.

Subtest 8: General information. In this subtest, the examinee is asked a set of “where” and “what” factual questions. This task measures acquired verbal knowledge.

Subtest 9: Concept formation. This task requires the examinee to identify the rule that determines why some shapes are inside a box and others are not (i.e., the rule may be that shapes are inside the box if they are both big and yellow). This is a measure of inductive reasoning and cognitive flexibility.

Subtest 10: Numbers reversed. On this task, the examiner recites a series of numbers, and the examinee must repeat them back in reverse order. This is a measure of working memory.

The WJ-IV Cog Extended Battery includes the following additional subtests:

Subtest 11: Number-pattern matching. This speeded task is similar to letter-pattern matching but requires the examinee to locate and circle pairs of identical numbers rather than letters. It increases in difficulty, moving from single- to triple-digit numbers, and has a 3-min time limit. This is another measure of perceptual speed.

Subtest 12: Nonword repetition. On this subtest, the examinee must listen to and repeat a series of nonsense words. It provides a measure of phonological processing, including auditory processing and working memory.

Subtest 13: Visual-auditory learning. On this task, the examinee is required to learn an association between words and pictures and then use this information to decode a string of pictures and identify the corresponding sentence. This task measures associative learning and memory.

Subtest 14: Picture recognition. On this task, the examinee is shown a picture or multiple pictures. The examinee is then asked to identify the picture(s) they just saw from another set of pictures. This task measures visual memory.

Subtest 15: Analysis-synthesis. In this task, the examinee is provided with a set of color “equations” as well as rules about color equivalencies (e.g., “black” is equivalent to “blue and yellow”). The examinee’s task is to determine

which colors are missing from an equation in order to make the two sides of the equation equal to one another. This task provides a measure of sequential reasoning and problem solving.

Subtest 16: Object-number sequencing. On this subtest, the examiner says list words including objects (animals and foods) and numbers. The examinee's task is to say the numbers first, in the order the examiner said them, and then the objects in the order the examiner said them. This task provides another measure of working memory.

Subtest 17: Pair cancellation. On this task, the examinee is asked to scan through a set of pictures on a page to identify a pair of items, which they must then circle. For example, they may be asked to circle any time they see a dog followed by a ball. This task measures attention and concentration.

Subtest 18: Memory for words. On this subtest, the examiner provides a set of words, and the examinee must repeat back the words in the same order in which he or she heard them. This provides a measure of memory span.

The WJ-IV Cog subtests are grouped into seven broad areas of ability (i.e., fluid reasoning, comprehension-knowledge, short-term working memory, cognitive processing speed, auditory processing, long-term retrieval, and visual processing). The fluid reasoning domain consists of two subtests: *number series*, which contributes to the quantitative reasoning subdomain, and *concept formation*. There are also two subtests within the comprehension-knowledge domain: *oral vocabulary*, which contributes to the vocabulary subdomain, and *general information*. In terms of short-term working memory, the two subtests (*verbal attention* and *numbers reversed*) fall within the cognitive efficiency subdomain. The cognitive processing speed domain is composed of two subtests: *letter-pattern matching*, which contributes to the perceptual speed and cognitive efficiency subdomains, and *pair cancellation*. Within the auditory processing domain, there are two subtests: *phonological processing* and *non-word repetition*. The long-term retrieval domain

includes the *story recall* and *visual-auditory learning* subtests. There are also two subtests (*visualization* and *picture recognition*) included in the visual processing domain.

WJ-IV Ach Battery

As is the case with the cognitive battery, the WJ-IV Ach includes both a standard and extended battery. The standard battery includes 11 subtests, while the extended battery includes an additional 9 subtests (McGrew et al. 2014).

The WJ-IV Ach Standard Battery includes the following subtests:

Subtest 1: Letter-word identification. On this subtest, the examinee is asked to name letters and read written words. This task measures reading decoding and sight word recognition.

Subtest 2: Applied problems. This is a paper-and-pencil task in which the examinee must solve mathematical "word problems." This task measures quantitative reasoning, math achievement, and math knowledge.

Subtest 3: Spelling. In this task, the examinee is asked to write dictated letters and words. This provides a measure of spelling ability.

Subtest 4: Passage comprehension. On this subtest, the examinee reads a written passage and must "fill in the blank" by supplying a word that is missing from the sentence or paragraph. This measures reading comprehension and verbal language comprehension.

Subtest 5: Calculation. This subtest is a paper and pencil computation task, in which the examinee must complete mathematical computations ranging from simple addition to calculus. This provides a measure of mathematical achievement.

Subtest 6: Writing samples. On this subtest, the examinee must write sentences based on supplied instructions. This provides a measure of writing ability.

Subtest 7: Word attack. On this subtest, the examinee is asked to read and pronounce nonsense words. This provides a measure of reading decoding and phonetic coding.

Subtest 8: Oral reading. This task requires the examinee to read sentences of increasing

difficulty out loud to the examiner. This is a measure of reading accuracy and prosody.

Subtest 9: Sentence reading fluency. On this task, the examinee must read simple sentences and answer “yes” or “no” to indicate whether the statement is accurate (i.e., to ensure comprehension) within a 3-min time limit. This task provides a measure of reading speed.

Subtest 10: Math facts fluency. On this subtest, the examinee must complete simple paper-and-pencil calculations as quickly as possible within a 3-min time limit. This measures math achievement and numerical facility.

Subtest 11: Sentence writing fluency. In this task, the examinee must write as many sentences (using three provided words) as possible within a 5-min time limit. This provides a measure of writing speed.

The WJ-IV Ach Extended Battery includes the following subtests:

Subtest 12: Reading recall. In this subtest, the examinee reads a story silently and then must recite the story to the examiner. This measures narrative comprehension and recall.

Subtest 13: Number matrices. On this task, the examinee is presented with a number matrix and must solve for the missing number. This provides a measure of quantitative reasoning.

Subtest 14: Editing. On this subtest, the examinee is asked to orally provide a correction for errors in writing. This task measures language development and English usage.

Subtest 15: Word reading fluency. This task requires the examinee to locate and circle words that go together as quickly as possible. This measures word knowledge and semantic fluency.

Subtest 16: Spelling of sounds. On this task, the examinee is asked to write down the spelling of nonsense words heard orally. This provides a measure of spelling ability and phonetic coding and analysis.

Subtest 17: Reading vocabulary. On this task, the examinee is asked to provide synonyms and antonyms. This task measures verbal language comprehension and lexical knowledge.

Subtest 18: Science. This task involves answering factual questions to measure the examinee’s knowledge of the sciences (i.e., anatomy, biology, chemistry, geology, medicine, and physics).

Subtest 19: Social studies. This task involves answering factual questions to measure the examinee’s knowledge of history, economics, geography, government, and psychology.

Subtest 20: Humanities. This task involves answering factual questions to assess the examinee’s knowledge of art, music, and literature.

Just as the WJ-IV Cog subtests are grouped into areas of broad ability, the WJ-IV Ach subtests are grouped into curricular areas (reading, mathematics, written language, and academic knowledge). Within the reading domain are four subdomains: (1) basic reading skills (composed of the *letter-word identification* and *word attack* subtests), (2) reading comprehension (composed of the *passage comprehension* and *reading vocabulary* subtests), (3) reading fluency (composed of the *oral reading* and *sentence reading fluency* subtests), and (4) reading rate (composed of the *sentence reading fluency* and *word reading fluency* subtests). The Mathematics domain contains two subdomains: (1) math calculation skills (composed of the *calculation* subtest and the *math facts fluency* subtest) and (2) math problem solving (composed of the *applied problems* and *number matrices* subtests). The written language domain also includes two subdomains: (1) basic writing skills (composed of the *spelling* and *editing* subtests) and (2) written expression (composed of the *writing samples* and *sentence writing fluency* subtests). The Academic Knowledge domain does not include subdomains and is composed of the *science*, *social studies*, and *humanities* subtests. The remaining subtests are considered supplemental subtests. The WJ-IV Ach also includes additional cross-cluster domains.

WJ-IV OL Battery

The WJ-IV OL battery consists of twelve subtests, including three subtests for English-Spanish

bilingual individuals (McGrew et al. 2014). The battery includes the following subtests:

Subtest 1: Picture vocabulary. On this task, the examinee is asked to name or label a picture (or point to a picture that the examiner named). This subtest measures language development and lexical knowledge.

Subtest 2: Oral comprehension. This task involves listening to passages and identifying the missing words. This task measures listening ability as well as lexical knowledge and language development.

Subtest 3: Segmentation. This task requires the examinee to break words down into smaller parts (i.e., syllables or phonemes). This measures phonetic coding.

Subtest 4: Rapid picture naming. On this task, the examinee is required to name as many items pictured as possible, within a given time limit. This subtest measures naming facility.

Subtest 5: Sentence repetition. This task involves listening to sentences and repeating the words in the correct sequence. This subtest is a measure of listening ability and memory span.

Subtest 6: Understanding directions. In this task, the examinee must point to aspects of a picture in response to oral directions. This provides a measure of language development and listening ability.

Subtest 7: Sound blending. In this task, the examinee is asked to combine orally presented sounds in order to form a whole word. This subtest is a measure of phonetic coding and synthesis.

Subtest 8: Retrieval fluency. On this task, the examinee is given a category and must name as many items within that category as possible within a time limit. This provides a measure of ideational fluency.

Subtest 9: Sound awareness. This task involves rhyming, sound deletion, sound substitution, and sound reversal. This provides a measure of phonetic coding, analysis, and synthesis.

Subtest 10: Vocabulario Sobre Dibujos. This is the Spanish version of *picture vocabulary*.

Subtest 11: Comprensión Oral. This is the Spanish version of *oral comprehension*.

Subtest 12: Comprensión de Indicaciones. This is the Spanish version of *understanding directions*.

The WJ-OL subtests are clustered within four oral language subdomains: (1) oral expression (which consists of the *picture vocabulary* and *sentence repetition* subtests), (2) listening comprehension (which consists of the *oral comprehension* and *listening comprehension* subtests), (3) phonetic coding (which consists of the *segmentation* and *sound blending* subtests), and (4) speed of lexical access (which consists of the *rapid picture naming* and *retrieval fluency* subtests).

Historical Background

The WJ-IV battery was developed based upon contemporary revisions to the Cattell-Horn-Carroll (CHC) theory of cognitive ability (Schneider and McGrew 2012). This theory blends elements of Cattell and Horn's Gf-Gc theory (Cattell 1941; Horn 1965) with Carroll's Three-Stratum theory.

Cattell and Horn's Gf-Gc Theory

Cattell (1941, 1943, 1950) described two broad cognitive abilities that make up general intelligence (G): fluid intelligence (Gf) and crystallized intelligence (Gc). Gf refers to the ability to reason and problem solve, including "drawing inferences, concept formation, classification, generating and testing hypotheses, identifying relations, comprehending implications, problem solving, extrapolating, and transforming information," as well as deductive and inductive reasoning (McGrew 2009). Gc refers to the knowledge that is acquired through acculturation, including knowledge of language, factual information, and cultural concepts. Horn (1965) later expanded the theory to include four additional specific cognitive abilities: Short-term memory (Gsm), long-term retrieval (Glr), processing speed (Gs), and visual-spatial thinking (Gv). Gsm refers to the ability to immediately hold information in one's consciousness, such that the information quickly

decays if one does not transfer this information to other memory systems. Glr refers to the ability to store information in long-term memory, thus making it available for later retrieval. Gs refers to the speed at which an individual is able to perform simple cognitive tasks. Gv refers to the individual's ability to perform visual, perceptual, or spatial cognitive tasks. Horn and Stankov (1982) added the construct of auditory processing (Ga), which refers to the ability to perceive, organize, synthesize, and interpret sound. In 1989, the Woodcock-Johnson Revised battery (WJ-R; Woodcock and Johnson 1989) was published based on the above seven cognitive factors. Following its publication, Horn (1988, 1989) described the additional construct of quantitative ability (Gq), which refers to the individual's store of mathematical or numerical knowledge, acquired through education. In 1998, Woodcock expanded the theory to include the construct of reading/writing ability (Grw), which includes abilities for reading decoding, spelling, reading comprehension, and written narrative construction. Lastly, Schneider and McGrew (2012) modified the existing theoretical constructs to support recent findings within the neuroscience literature. This included replacing Gsm with short-term working memory (Gwm) to account for attentional control and the manipulation of information, expanding the Glr construct to include measures of lexical naming, and integrating sound memory within the operationalization of Ga.

Carroll's Three-Stratum Theory

Carroll (1993) conducted a factor analysis of 461 psychometric data sets, including some data taken from the norming sample of the original *WJ Psycho-Educational Battery*. Based on this analysis, Carroll identified three levels of cognitive ability. Stratum I refers to the narrowest of cognitive abilities, in which Carroll identified 69 different narrow abilities. Stratum II refers to broad categories of cognitive ability, in which Carroll identified fluid intelligence, crystallized intelligence, general memory and learning, broad visual perception, broad auditory perception, broad retrieval ability, broad

cognitive speediness, and processing speed. Finally, Stratum III refers to general intelligence or the broadest factor describing a person's overall cognitive ability.

As McGrew (2009) notes, the commonalities between these domains and those included in Cattell and Horn's theory led theorists to combine them into a single theory of intelligence (CHC Theory). The structure of the WJ-IV assessment battery is based upon this combined theory, in that the subtests (which measure narrow, Stratum I abilities) are grouped into factors (Stratum II), which combine to generate a measure of general intelligence (Stratum III). Stratum II abilities assessed by the battery include comprehension/knowledge (Gc), long-term retrieval (Glr), visual processing (Gv), auditory processing (Ga), fluid reasoning (Gf), processing speed (Gs), short-term working memory (Gsm), reading-writing (Grw), quantitative knowledge (Gq), and domain-specific knowledge. The WJ-IV Cog battery includes two measures of g: general intellectual ability (GIA) or brief intellectual ability (BIA), the latter being based on just three subtests. Likewise, the WJ-IV Ach and OL batteries include an overall achievement and language score.

Psychometric Data

The WJ-IV Cog, Ach, and OL batteries are co-normed, meaning that the three batteries were normed using the same normative sample. The normative sample consists of 7416 subjects, drawn from 47 "geographically diverse" communities in the United States. The norms provide a preschool sample (children ages 2–5 who were not yet enrolled in kindergarten), a kindergarten–grade 12 sample, an undergraduate and graduate student sample, and an adult sample. The norms cover ages 24 months through over 90 years. Sampling controlled for geographical region, community size, sex, country of birth, race, Hispanic status, type of school or university attended, parent education, educational level achieved by adults, occupational status of adults, and type of occupation. Normative sampling was based on the 2010 census projections.

Because the WJ-IV Cog, Ach, and OL batteries are co-normed, the battery also includes discrepancy norms, which allow the examiner to determine the significance of a discrepancy found between an examinee's cognitive, achievement, and language test scores. The battery also provides for intra-cognitive, intra-achievement, and intra-language discrepancy norms, by comparing an individual's performance on one subtest to their average performance across all subtests.

Reliability

The WJ-IV Technical Manual (McGrew et al. 2014) provides extensive reliability data, including measures of standard error (as an indication of the precision of an individual's test scores), internal consistency, alternate forms (reliability ranging from 0.79 to 0.95), test-retest reliability for WJ-IV Cog standard battery (median reliability ranging from 0.86 to 0.93), test-retest reliability for WJ-IV Ach standard battery (median reliability ranging from 0.89 to 0.94), test-retest reliability for WJ-IV OL battery (median reliability ranging from 0.80 to 0.94), and test-retest reliability for speeded tests (median reliability ranging from 0.85 to 0.95).

Validity

Construct validity for the WJ-IV Cog battery was established through confirmatory factor analyses, which fit the WJ-IV subtests to the CHC factor structure. All cognitive subtests loaded on a single factor, suggesting that they were not influenced by variance unrelated to the construct of G. Further, low correlations were found between cognitive battery clusters, suggesting that the clusters measure distinct things. Results of the achievement battery analyses showed less distinction among domains.

To further assess construct validity, 1056 validity study subjects were recruited (in addition to the normative sample) to compare results of the WJ-IV battery to other tests of cognitive ability and achievement. Correlations between the GIA score and overall IQ score from a number of other cognitive tests (i.e., Wechsler Preschool and

Primary Scales of Intelligence, Wechsler Intelligence Scales for Children, Wechsler Adult Intelligence Scale, Differential Ability Scales, Kaufman Assessment Battery for Children, and Stanford Binet) generally fell within the 0.80 range. In terms of the WJ-IV Ach battery, the total achievement score was highly correlated with the total achievement score from the Wechsler Individual Achievement Test (0.90) and the overall composite from the Kaufman Test of Educational Achievement (0.91). The WJ-OL oral language domain is strongly correlated (0.60–0.80 range) with global oral language scores from the Clinical Evaluation of Language Fundamentals, Comprehensive Assessment of Spoken Language, Oral and Written Language Scales, Kaufman Assessment Battery for Children, and Wechsler Individual Achievement Test.

Clinical Uses

The WJ-IV batteries have a number of clinical uses, including identifying cognitive, academic, and language skills for the purpose of educational planning. While the cognitive and oral language batteries are useful in the diagnosis of intellectual disabilities and specific language disorders, all batteries can aid in the diagnosis of learning disorders. Because of the CHC factor structure and inclusion of intraindividual discrepancy norms, the battery is useful in identifying an individual's profile of strengths and weaknesses, which can aid in educational and treatment planning. This is particularly useful for assessing individuals with autism spectrum disorders, who often demonstrate a high degree of variability among specific areas of cognitive ability.

See Also

- [Achievement Testing](#)
- [Cognitive Skills](#)
- [Differential Ability Scales](#)
- [Educational Testing](#)

- [Psychological Assessment](#)
- [Stanford-Binet Intelligence Scales](#)
- [Wechsler Preschool and Primary Scale of Intelligence](#)

References and Reading

- Carroll, J. B. (1993). *Human cognitive abilities: A survey of factor analytic studies*. New York: Cambridge University Press.
- Cattell, R. B. (1941). Some theoretical issues in adult intelligence testing. *Psychological Bulletin*, 38, 592.
- Cattell, R. B. (1943). The measurement of adult intelligence. *Psychological Bulletin*, 40, 153–193.
- Cattell, R. B. (1950). *Personality: A systematic theoretical and factorial study*. New York: McGraw-Hill.
- Horn, J. L. (1965). *Fluid and crystallized intelligence* (Unpublished doctoral dissertation). Urbana-Champaign: University of Illinois.
- Horn, J. L. (1985). Remodeling old models of intelligence. In B. B. Wolman (Ed.), *Handbook of intelligence: Theories, measurements, and applications* (pp. 267–300). New York: John Wiley.
- Horn, J. L. (1988). Thinking about human abilities. In J. R. Nesselrode & R. B. Cattell (Eds.), *Handbook of multivariate psychology* (2nd ed., pp. 645–865). New York: Academic Press.
- Horn, J. L. (1989). Models for intelligence. In R. Linn (Ed.), *Intelligence: Measurement, theory and public policy* (pp. 29–73). Urbana: University of Illinois Press.
- Horn, J. L., & Stankov, L. (1982). Auditory and visual factors of intelligence. *Intelligence*, 6, 165–185.
- McGrew, K. S. (2009). CHC theory and the human cognitive abilities project: Standing on the shoulders of the giants of psychometric intelligence research. *Intelligence*, 37, 1–10.
- McGrew, K. S., LaForte, E. M., & Schrank, F. A. (2014). *Technical manual. Woodcock-Johnson IV*. Rolling Meadows: Riverside.
- Schneider, W. J., & McGrew, K. S. (2012). The Cattell-horn-Carroll model of intelligence. In D. P. Flanagan & P. L. Harrison (Eds.), *Contemporary intellectual assessment: Theories, tests, and, issues* (3rd ed., pp. 99–144). New York: Guilford Press.
- Schrank, F. A., Mather, N., & McGrew, K. S. (2014a). *Woodcock-Johnson IV tests of oral language*. Rolling Meadows: Riverside.
- Schrank, F. A., Mather, N., & McGrew, K. S. (2014b). *Woodcock-Johnson IV tests of achievement*. Rolling Meadows: Riverside.
- Schrank, F. A., McGrew, K. S., & Mather, N. (2014c). *Woodcock-Johnson IV*. Rolling Meadows: Riverside.
- Schrank, F. A., McGrew, K. S., & Mather, N. (2014d). *Woodcock-Johnson IV tests of cognitive abilities*. Rolling Meadows: Riverside.
- Woodcock, R. W. (1998). Extending Gf-Gc theory into practice. In J. J. McArdle & R. W. Woodcock (Eds.), *Human cognitive abilities in theory and practice* (pp. 137–156). Mahwah: Lawrence Erlbaum.
- Woodcock, R. W., & Johnson, M. B. (1989). *Woodcock-Johnson psycho-educational battery - revised*. Itasca: Riverside Publishing.

Word Fluency

- [Verbal Fluency](#)

Word Recognition

- [Decoding Skills](#)

Word Use

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Definition

In language development, word use refers to how a word is mapped to the world. For example, initially a child may produce the word “dog” to refer to all animals with four legs, or perhaps just to refer to the family pet. The mapping between word and referent shifts over time until the term is used to refer to the category conventionally associated with the word in the language community, in this case the category of dogs.

Individuals with Autism Spectrum Disorder (ASD) have been observed to use words in non-conventional ways well beyond the early stages of language development. Different types of unusual

word use have been noted: mapping a word to an aspect of the context present when it was first heard; attributing a personal, idiosyncratic meaning to a word that is conventionally used in a different way (e.g., “furnace” for “stomach”); slight modification of the sound of words so that the meaning remains comprehensible (e.g., “cuts and bluesers” for “cuts and bruises”); and overly precise or pedantic use of words for the situation (e.g., “vignette” for “cartoon”).

Whereas nonconventional word use declines with increasing language ability in typical development and developmental disorders other than ASD, it has been found to increase along with language ability in ASD, being more prominent in highly verbal individuals.

See Also

- [Idiosyncratic Language](#)
- [Pragmatic Communication](#)

References and Reading

- Bowerman, M. (1976). Semantic factors in the acquisition of rules for word use and sentence construction. In D. Morehead & A. Morehead (Eds.), *Directions in normal and deficient child language*. Baltimore: University Park Press.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child*, 2, 217–250.
- Mayes, L., Volkmar, F., Hooks, M., & Cicchetti, D. (1993). Differentiating pervasive developmental disorder not otherwise specified from autism and language disorders. *Journal of Autism and Developmental Disorders*, 23(1), 79–90.
- Tager-Flusberg, H., Paul, R., & Lord, C. (2005). Language and communication in autism. In F. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders*. Hoboken: Wiley.
- Volden, J., & Lord, C. (1991). Neologisms and idiosyncratic language in autistic speakers. *Journal of Autism and Developmental Disorders*, 21, 109–130.

Words

- [Vocabulary](#)

Work Placement for Individuals with Autism Spectrum Disorder

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Definition

Work placements are periods of supervised work that provide a person with the opportunity to gain practical training and experience within an organization. The duration of work placements can vary, ranging from a few days to several months, and can be paid or unpaid. This entry focuses on work placements for high school students with autism spectrum disorder (ASD), presenting current knowledge on how work placements can support their transition into employment.

Historical Background

The transition to adult life for youth with ASD is a phase full of uncertainty, anxiety, and challenges (Schall et al. 2012). Outcomes for adults with ASD are poor and, in many cases, are poorer than those with other disabilities. Participating less in postsecondary education and employment than the general population (Shattuck et al. 2012), youth with ASD are two times less likely to obtain a certificate, diploma, or bachelor's degree or higher than people with other disabilities (Australian Bureau of Statistics 2017). Adults with ASD are employed at a rate 50–75% lower than those without ASD or with other disabilities (International Society for Autism Research 2018).

Employment is an important major life area of adulthood, providing a key mechanism promoting social inclusion, enabling financial independence, and fostering well-being and quality of life (Lee and Carter 2012). Despite individuals with ASD commonly possessing specific skills and abilities beneficial in employment (Jones et al. 2018; Lee et al. 2019) including attention to detail, technical abilities, visual perception, and a preference for repetitive or monotonous tasks (Bölte et al. 2019), the core challenges of ASD (Strickland et al. 2013) and the lack of support after exiting high school for many pose a barrier to finding successful employment (Wehman et al. 2012).

Interventions seeking to strengthen adaptive behaviors while reducing the maladaptive behaviors associated with ASD among high-school-aged students and young adults demonstrate limited effectiveness in improving competitive employment outcomes for youth with ASD (Wehman et al. 2017). Conversely, emerging research suggests that work placement programs during high school may improve competitive employment outcomes for youth with ASD (Wehman et al. 2017). Evidence is emerging that for youth with ASD actually experiencing work, by spending time in real work environments such as in work placements, is likely one of the most promising strategies in reducing the underemployment and unemployment experienced by youth with ASD. Developing manualized and replicable approaches, which build pathways to employment for youth with ASD by mapping strengths and interests to employment opportunities, build skills through work placements, and culminate in community-based competitive employment, is an area of priority for both service providers and autism researchers (Wehman et al. 2017).

Current Knowledge

An emerging body of evidence points toward the importance of preparing youth with ASD for employment. Work experience in high school predicts later employment success (Whittenburg et al. 2018), while those with access to transition

programs and support services are more likely to gain employment (Hendricks 2010). Practical work experience opportunities can help youth with ASD in recognizing their career-related strengths and interests, identifying their goals, improving their employment-related skills, and expanding their knowledge of occupational options (Lee et al. 2019). These experiences support pathway planning for postsecondary education and careers and build work readiness (Lee et al. 2019). A recent Australian-based study exploring work experience in Information and Communication Technology (ICT)-related fields for adolescents with ASD highlighted that even short-term work placements ranging between 5 and 10 days can enable these adolescents to gain practical insight into the real workplaces, explore and extend their interests, increase their confidence, and plan for the future (Lee et al. 2019).

While research is still emerging, there is evidence to suggest that work placements have a long-term and positive impact on employment outcomes (Wehman et al. 2017). Wehman et al. (2017) conducted a randomized control trial evaluation of the “Project SEARCH Plus ASD Supports program” in the United States. Youth with ASD assigned to the Project SEARCH group, or the intervention group, participated in a 9-month internship program during their last year of high school, and in comparison with those receiving usual high school education services (the control group), those in the intervention group demonstrated higher rates of community-based employment and worked more hours per week at graduation and at 3-month and 12-month follow-up. The majority of Project SEARCH participants were still employed 12 months after the intervention, providing high-level evidence that community-based work placement is beneficial in promoting the employment outcomes of youth with ASD (Wehman et al. 2017).

Several important factors contribute to the success of work placement for youth with ASD including job match, job coach support, and environmental factors. Similar to their typically developing peers, young people with ASD are motivated by the nature of the job (Beyer et al.

2016; Scott et al. 2015), with a good job match influencing their work performance in relation to initiative, productivity, and interest in the task (Beyer et al. 2016). Harnessing the strengths and interests of adolescents with ASD in work placements (Lee et al. 2019), by crafting work experience to ensure assigned tasks capture and align with their interests and aspirations and that their needs are met (Wehman et al. 2017), is key in ensuring successful short-term work placements. Profiling the skills of youth with ASD prior to commencing work placement through comprehensive skills assessment enables adolescents and employers to develop placements likely to be the most beneficial in promoting their skill development and motivation (Beyer et al. 2016). Matching work tasks to the skills and abilities of youth with ASD enables them to see their potential and abilities as valuable future employees, strengthening their skills and knowledge in areas of interest, assisting them in planning for future education and career pathways (Lee et al. 2019).

Assistant positions in retail, catering (in the kitchen and in service roles), horticulture, child care, information technology (IT), cleaning, warehousing, mechanics, leisure and sport, and hair and beauty provide many work placement opportunities for youth with ASD (Beyer et al. 2016). Job coaching is an important strategy in promoting success in work placements for youth with ASD, particularly those with developmental or intellectual disability. Job coaches are allocated based on the needs of youth with ASD and take a person-centered approach to mapping an individual's career pathway, preparing them for employment, and supporting them in overcoming barriers. A study in the United Kingdom with youth with ASD aged 15–21 years with severe and complex needs found that a job coach improved their acquisition of work skills, including communicating with others, concentrating on tasks, remembering and following instructions, working without support, undertaking a range of tasks, and taking initiative (Beyer et al. 2016). Collectively, these findings suggest that job coach support combined with work placement experiences may be helpful in assisting youth with ASD in preparing for employment.

In addition to job coaching, behavioral supports are beneficial in supporting youth with ASD who experience difficulty understanding workplace expectations. Positive behavior support programs can promote an understanding of appropriate workplace behaviors, such as maintaining personal space and using appropriate language (Wehman et al. 2012). In the “Project SEARCH Plus ASD Supports program” evaluation conducted in the United States, on-site job coaches and combined with behavioral supports delivered promising results in helping students with ASD to gain and maintain community-based competitive employment (Wehman et al. 2017).

Targeting environmental factors is a key consideration for work placement interventions. Environmental factors extend beyond the physical environment and layout of workspaces to include the commitment, attitudes, and understanding of work colleagues and management and their knowledge of the needs of people with ASD (Lee et al. 2019). The social model of disability suggests that disability can often be minimized or avoided through an accommodating environment (physical, cognitive, or emotional) and the provision of assistive tools (den Houting 2019). Leadership from senior staff and management in promoting an autism-friendly work environment and a balanced understanding of ASD, which includes recognizing the strengths and challenges of autistic individuals, is crucial in enabling successful work placement experiences (Bölte et al. 2019). Preparing workplaces prior to placing youth with ASD, through education aimed at promoting both an understanding of ASD and appropriate communication strategies, can help to ensure positive experiences. Qualitative evaluation of a short-term work placement by Lee et al. (2019) found that providing autism education to supervisors and staff was a key factor in understanding the strengths and challenges of youth with ASD enabling job matching and ultimately underpinned the program.

Other contextual factors influencing the success of transition to employment for people with ASD include the support of family members and personal factors such as independence and social

skills (Beyer et al. 2016). Family members may at times be protective of their young people with ASD, inadvertently limiting their exposure to new experiences (Beyer et al. 2016). Service providers aiming to support youth with ASD and their families through the transition process should be well informed and clear in the messages they convey to families in regard to the available employment options (Beyer et al. 2016). Programs aimed at improving independence and social skills, such as peer mentoring, can work toward minimizing personal barriers to employment (Carter et al. 2012; Olsson et al. 2015).

Work placements for youth with ASD are not possible without the support from businesses and organizations, highlighting the importance of autism service providers partnering with industry. Whittenburg et al. (2018) outlined a four-step guide for transition specialists aiming to identify and engage with local business partners in developing integrated work placements for young people with ASD: (1) identifying potential business partners and targeting businesses based on student strengths, preferences, and skills; (2) engaging in introductory meetings with the goal of clarifying roles and responsibilities of both work experience students and their employers; (3) engaging in productive conversations with employers, learning about their business, and scoping how the partnership could meet specific business needs; and (4) enabling opportunities for youth with ASD and employers to connect through job trials, a tour of the business, or inviting a business representative to school events. Placing youth with ASD with employers and organizations is potentially beneficial to business, given youth with ASD often take new perspectives and have diverse ideas (Lee et al. 2019), can positively impact the attitudes of staff and customers, and reflect positively on the company image (Beyer et al. 2016).

Future Directions

Work placements improve the employment outcomes of youth with ASD, providing opportunities for developing and practicing essential work

and social skills important in employment. Work placements provide the opportunity for employers to observe the skills and abilities and recognize the value of employees with ASD. Work placement opportunities increase the chances of youth with ASD in successfully gaining and maintaining employment after graduation, despite the challenges and impairments associated with a diagnosis of ASD (Schall et al. 2015). However, there is a need for more research aiming to develop work placement frameworks, replicable across industries. Future research should also consider the costs and intensity of intervention required to achieve meaningful employment outcomes (Wehman et al. 2017). Fostering links between service providers and high schools, with the shared goal of bridging the gap between school and employment, has the potential to improve employment outcomes for these young people. While work placements show promise as a means of improving employment outcomes for youth with ASD, the success of such approaches depends on cooperation between education and service providers and industry.

See Also

- [Adulthood, Transition to](#)
- [Employment](#)
- [Employment in Adult Life](#)
- [Success Factors Enabling Employment for Adults on the Autism Spectrum](#)

References and Reading

- Australian Bureau of Statistics. (2017). *Disability, ageing and carers, Australia: Summary of findings, 2015; cat. no. 4430.0*. Canberra: Australian Bureau of Statistics.
- Beyer, S., Meek, A., & Davies, A. (2016). Supported work experience and its impact on young people with intellectual disabilities, their families and employers. *Advances in Mental Health and Intellectual Disabilities*, 10(3), 207–220. <https://doi.org/10.1108/amhid-05-2014-0015>.
- Bölte, S., Mahdi, S., de Vries, P. J., Granlund, M., Robison, J. E., Shulman, C., . . . Selb, M. (2019). The gestalt of functioning in autism spectrum disorder: Results of the international conference to develop

- final consensus international classification of functioning, disability and health core sets. *Autism*, 23(2), 449–467. <https://doi.org/10.1177/1362361318755522>.
- Carter, W. E., Austin, D., & Trainor, A. A. (2012). Predictors of postschool employment outcomes for young adults with severe disabilities. *Journal of Disability Policy Studies*, 23(1), 50–63.
- den Houting, J. (2019). Neurodiversity: An insider's perspective. *Autism*, 23(2), 271–273. <https://doi.org/10.1177/1362361318820762>.
- Hendricks, D. (2010). Employment and adults with autism spectrum disorders: Challenges and strategies for success. *Journal of Vocational Rehabilitation*, 32, 125–134. <https://doi.org/10.3233/JVR-2010-0502>.
- International Society for Autism Research. (2018). *Getting people with autism to work: An international society for autism research policy brief*. Retrieved May 21, 2019, from https://cdn.ymaws.com/www.autism-insar.org/resource/resmgr/files/policybriefs/2018-insar_policy_brief.pdf
- Jones, M., Bölte, S., Falkmer, M., Milbourne, B., Tan, T., Sheehy, L., & Girdler, S. (2018). *A strength-based program for adolescents with autism*. Research report no. 17/18. Bankwest Curtin Economics Centre.
- Lee, G. K., & Carter, E. W. (2012). Preparing transition-age students with high-functioning autism spectrum disorders for meaningful work. *Psychology in the Schools*, 49(10), 988–1000. <https://doi.org/10.1002/pits.21651>.
- Lee, E. A. L., Black, M. H., Tan, T., Falkmer, T., & Girdler, S. (2019). “I’m destined to ace this”: Work experience placement during high school for individuals with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 49(8), 3089–3101. <https://doi.org/10.1007/s10803-019-04024-x>.
- Olsson, N. C., Tammimies, K., & Bölte, S. (2015). Manualized social skills group training for children and adolescents with higher functioning autism spectrum disorder: Protocol of a naturalistic multicenter, randomized controlled trial. *Translational Developmental Psychiatry*, 3(1). <https://doi.org/10.3402/tdp.v3.29825>.
- Schall, C., Wehman, P., & McDonough, J. L. (2012). Transition from school to work for students with autism spectrum disorders: Understanding the process and achieving better outcomes. *Pediatric Clinics of North America*, 59(1), 189–202, xii. <https://doi.org/10.1016/j.pcl.2011.10.009>.
- Schall, C. M., Wehman, P. H., Brooke, V., Graham, C., McDonough, J., Brooke, A., ... Allen, J. (2015). Employment interventions for individuals with ASD: The relative efficacy of supported employment with or without prior project SEARCH training. *Journal of Autism & Developmental Disorders*, 45, 3990–4001. <https://doi.org/10.1007/s10803-015-2426-5>.
- Scott, M., Falkmer, M., Girdler, S., & Falkmer, T. (2015). Viewpoints on factors for successful employment for adults with autism spectrum disorder. *PLoS One*, 10(10), e0139281. <https://doi.org/10.1371/journal.pone.0139281>.
- Shattuck, P. T., Narendorf, S. C., Cooper, B., Sterzing, P. R., Wagner, M., & Taylor, J. L. (2012). Postsecondary education and employment among youth with an autism spectrum disorder. *Pediatrics*, 129(6), 1042–1049. <https://doi.org/10.1542/peds.2011-2864>.
- Strickland, D. C., Coles, C. D., & Southern, L. B. (2013). JobTIPS: A transition to employment program for individuals with autism spectrum disorders. *Journal of Autism & Developmental Disorders*, 43(10), 2472–2483. <https://doi.org/10.1007/s10803-013-1800-4>.
- Wehman, P., Schall, C., McDonough, J., Molinelli, A., Riehle, E., Ham, W., & Thiss, W. R. (2012). Project SEARCH for youth with autism spectrum disorders. *Journal of Positive Behavior Interventions*, 15(3), 144–155. <https://doi.org/10.1177/1098300712459760>.
- Wehman, P., Schall, C. M., McDonough, J., Graham, C., Brooke, V., Riehle, J. E., ... Avellone, L. (2017). Effects of an employer-based intervention on employment outcomes for youth with significant support needs due to autism. *Autism*, 21(3), 276–290. <https://doi.org/10.1177/1362361316635826>.
- Whittenburg, H. N., Sims, K. A., Wehman, P., & Walther-Thomas, C. (2018). Strategies for developing work experiences for youth with intellectual and developmental disabilities. *Career Development and Transition for Exceptional Individuals*, 42, 1–6. <https://doi.org/10.1177/2165143418813900>.

Work Team

► Mobile Work Crew Model

Workforce Innovations Opportunity Act

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Definition

On July 22, 2014, President Barack Obama signed The Workforce Innovation Opportunity Act

(H.R. 803) into law. This reauthorized the Workforce Innovation Act of 1998 for an additional 6 years from 2015–2020. “WIOA opens the door to states’ greater use of sector partnerships and career pathway models and includes higher levels of accountability and outcome data reporting” (National Skills Coalition 2016). This is the most significant piece of legislation for people with disabilities since the passage of the Americans with Disabilities Act of 1990. A central focus of this piece of legislation is upon transition aged youth. It has reallocated 15% of State Offices of Vocational Rehabilitative Services funding to be spent on this age group. The goal of this legislation is to obtain competitive integrated employment for job seekers. State agencies can no longer make direct referrals to sheltered workshops placing individuals with disabilities in sub-minimum wage jobs (34 CFR Parts 361, 363, 397). In the event such a placement is necessary the law requires outside periodic evaluations (Butz 2016). Under WIOA, the performance benchmarks for state offices of Vocational Rehabilitative (VR) Services have also changed. No longer will a consumer’s case be dropped from the VR caseload after the individual has worked 90 days. This practice led to the agencies measuring their success by the number of cases they closed. Now the emphasis is upon longevity in a career and the generation of a living wage. It also calls for better integration of services for consumers through the creation of one-stop centers where all of an individual’s needs related to employment can be addressed. WIOA specifically calls for “. . . transition services for students with disabilities that facilitate the transition from school to post-secondary life, such as achievement of an employment outcome in competitive integrated employment, or pre-employment transition services. . . (H.R. 803-233).”

The Secretary of Education proposed changes to Vocational Rehabilitative Services and State Supported Employment Services program (Supported Employment program) (34 CFR Part 363). According to the proposed changes “Giving workers with disabilities the supports and the opportunity to acquire the skills that they need to pursue in-demand jobs and careers is critical to

growing our economy, ensuring that everyone who works hard is rewarded, and building a strong middle class. To help achieve this priority for individuals with disabilities, the Rehabilitation Act of 1973, as amended by WIOA, seeks to empower individuals with disabilities to maximize employment, economic self-sufficiency, independence, and inclusion and integration into society” (34 CFR Parts 361, 363, and 397. P. 5). WIOA seeks to streamline services and coordinate both services and funding on federal, state, and local levels, making it easier for job seekers with disabilities to connect with employers. The Act also places a heightened emphasis on competitive integrative employment for people with disabilities (34 CFR Parts 361, 363, and 397. P. 8) regardless of the extent of the disability. “The term competitive integrated employment means work that is performed on a full –time or part-time basis (including self-employment)- (A) for which an individual is compensated at a rate that shall not be less than the higher of the rate specified in the Fair Labor Standards Act of 1938 or the rate specified in the applicable state or local minimum wage law. . .” (H.R. 803-209).

Aside from placing an emphasis on streamlining federal, state and local services, and placing individuals with disabilities in competitive employment, a third emphasis is on students with a disability and youth with a disability. The law provides for a continuum of services. There are new requirements to provide pre-employment transition services as well as transition services to students and youth with a disability. It is worth noting that pre-employment transition services can now be funded, such services were excluded in previous versions of the act. Students and youth on the autism spectrum need training in many of the soft skills critical to obtaining and keeping employment (e.g., interview skills, dressing appropriately for the environment, independent living skills, travel training, appropriate peer and supervisor communications, etc.). Training and education for these activities were not previously funded. The changes in the Act are designed to maximize the potential of these individuals to transition to postsecondary education and employment.

WIOA has adopted the definition of an “Individual with a Disability” as identified under section 3 of the Americans with Disabilities Act (42 U.S.C. 12102) (H.R. 803-209). The law also adopted language from the Individuals with Disabilities Education Act (20 U.S.C. 1414(d)(1) (A) (i) (VIII) in describing students with a disability who were eligible for the new emphasis on pre-employment training. Essentially students who were eligible for special education services and received those services qualify for this support. The law broadens the definition to include individuals who qualified as individuals with a disability under Section 504 (H.R. 803-212). The age range with which Vocational Rehabilitative Services can work with youth with disabilities has been extended under WIOA when compared to IDEA. Under WIOA, “The term ‘youth with a disability’ means an individual with a disability who – (i) is not younger than 14 years of age; and (ii) is not older than 24 years of age” (H.R. 803-213).

Institutions of Higher Education (IHE) can, in fact, provide pre-employment and employment transition services to individuals with disabilities under WIOA. The statute specifies performance measures for the state agencies which include measuring the percentage of program participants who obtained a secondary diploma or its equivalent, or enrolled in an education or training program leading to a recognized postsecondary credential. A “recognized postsecondary credential” is defined in WIOA sec.3(52) as “a credential consisting of an industry-recognized certificate or certification, a certificate of completion of an apprenticeship, a license recognized by the State involved or the Federal Government or an associate or baccalaureate degree” (34 Code of Federal Regulations (CFR) Parts 361 and 463. P. 55983). Furthermore, the Act’s definition of one-stop delivery systems and entities that can serve as one-stop partners indicates this reality. “The one-stop delivery system brings together workforce development, educational, and human resource services in a seamless customer-focused service delivery network that enhances access to the programs’ services and improves long-term employment outcomes for individuals receiving

assistance” (34 Code of Federal Regulations (CFR) Parts 361 and 463. P. 56009). Institutions of Higher Education are explicitly cited under the definition of one-stop operators under WIOA: “One-stop operators may be a single entity (public, private, or nonprofit) or consortium of entities. . .the types of entities that may be a one-stop entity include: (1) An institution of higher education. . .” (34 Code of Federal Regulations (CFR) Parts 361 and 463. P. 56014).

The Workforce Innovation Opportunity Act mandates required activities that fall under the provision of pre-employment transition services. These include “(1) job exploration counseling; (2) work based learning experiences, which may include in-school or after school activities, or experience outside the traditional settings (including internships), that is provided in an integrated environment to the maximum extent possible; (3) counseling or opportunities for enrollment in comprehensive transition or post-secondary educational programs at institutions of higher education; (4) workplace readiness training to develop social skills and independent living; and (instruction in self-advocacy, which may include peer mentoring” (H.R. 803-234).

WIOA identifies six primary indicators of performance for adult and dislocated worker programs, AEFLA programs, and the VR programs. These are (1) the percentage of participants who are in unsubsidized employment during the second quarter after exit from the program; (2) the percentage of participants who are in unsubsidized employment during the fourth quarter after exit from the program; (3) median earnings of participants in unsubsidized employment during these two time periods; (4) the percentage of those participants enrolled in education or a training program who attained a recognized postsecondary credential or a secondary school diploma; (5) the percentage of participants who, during the a program year, are in an education or training program that leads to a recognized postsecondary credential or employment who are achieving measurable skill gains; and (6) effectiveness in serving employers (34 Code of Federal Regulations (CFR) Parts 361 and 463. P. 56028).

Historical Background

The most immediate precursors to the Workforce Innovation Opportunity Act were the Job Training and Partnership Act (JTPA) of 1982 and the Workforce Investment Act (WIA) of 1998.

The Workforce Investment Act, and its later reauthorization (and significant expansion) with the Workforce Innovation Opportunities Act, attempts to integrate the intentions of nearly a century of legislation addressing the need for independence and community-based work opportunities for individuals not participating in the workforce for either economic reasons or due to the challenges of a disability. The components of WIOA that are most germane to young adults and adults with a diagnosis of autism represent an integration of three distinct legislative agendas.

Some legislations, such as the Wagner-Peyser Act (1933), the Manpower Development Training Act (1962), the Comprehensive Employment and Training Act (1973), and the Job Training and Partnership Act (1982), focused primarily on assisting individuals not participating in the workforce for economic and educational reasons not necessarily related to any disabilities. All of these acts provide vocational support for individuals with a disability, but it was not the precise focus of any of the acts. The Rehabilitation Act (1973) and the American's with Disabilities Act (1992) more specifically address the provision of supports for individuals with disabilities seeking to find work. The Individuals with Disabilities Education Improvement Act (2004) and certain provisions of the Higher Education Opportunity Act (2008) address the importance of preparing young adults with a disability to transition from an educational setting to independent living and work. The WIOA is the most comprehensive attempt by Congress to combine the initiatives and the agendas of these three distinct strains of legislation in one act.

One of the first pieces of legislation advocated for by President Franklin D. Roosevelt was the Wagner-Peyser Act, which created a national network of public employment services offices that remains in effect to this day. There had been prior, less successful, attempts at creating similar

national networks. "The [national employment network] continues to serve as the foundation for the national One-Stop delivery system established by the workforce Investment Act of 1998" (O'Leary and Eberts 2008, p. 1). A key component of the WIOA reauthorization of the Workforce Investment Act is the One-Stop aspect of accessibility for those seeking assistance.

Current Knowledge

There are few empirically based research studies on the employment of individuals on the autism spectrum. We can only glean information from studies that measure the effectiveness of an intervention or surveys that examine employment with individuals with disabilities in general. Wehman et al. (2013) found that students on the autism spectrum who were involved in a randomized clinical trial of job training had an 87.5% employment rate post-study. This was statistically and clinically significant. The control group, referred to as the business as usual group (i.e., students working with their high schools and state office of Vocational Rehabilitative Services), had only a 6.25% employment rate. Moore and Schelling (2015) did not look specifically at the autism population. However, they did examine the population of students with an intellectual disability which includes some individuals, but not all individuals on the autism spectrum. Their research indicated that 9 out of 10 students with an intellectual disability who graduated from a post-secondary program were employed with 2 years of the study. They compared their data to the findings of the National Longitudinal Transition Study 2 where only about half of the graduates of high school with an intellectual disability had been employed in the last 2 years. Anecdotally, there are some reports of the unemployment rate being as high as 90% in the autism population. This does not include a significant number of higher functioning individuals on the spectrum who are underemployed. Interestingly, Diamant (2015) found that neither IQ nor academic ability were better predictors of employment and independent living for people on the autism spectrum

than their ability to successfully and consistently complete activities of daily living (ADLs).

Future Directions

For those transition aged youth considering attending a college-based transition program paying for a program of this nature can be a major obstacle. Although WIOA will allow state offices of Vocational Rehabilitative (VR) services to pay for these programs, it will only pay for a fraction of the cost. Typically, VR will pay for the tuition for the courses at in-state public university rates. This often will not cover the room and board expenses for the student to reside on campus. Should the student on the autism spectrum decide to go out of state or go to a private university, the cost differential can be substantial. Legislators need to align the Higher Education Opportunity Act of 2008 with future amendments and reauthorizations of WIOA.

A critical component to fostering a student on the autism spectrum's ability to find and maintain employment includes learning independent living skills. Under HEOA colleges were able to create Comprehensive Transition and Post-Secondary (CTP) programs that were approved by the US Department of Education. This allowed students with ID or DD (including those with autism) to enroll in CTPs where there was a curriculum with specialized courses to address the nature of their disabilities (including independent living skills) and to receive a specific advising system. However, at least 51% of their time in the program needs to be with nondisabled peers. Often this is accomplished not only through academic course work, but also in internship placements and on-campus living. By better aligning WIOA with HEOA, students who opt to attend a private or out of state college with a US DOE approved CTP could be eligible for not only monies from WIOA that were disbursed through state offices of VR, but also these students would be eligible for Federal Student Aid under Title IV of HEOA. The grants under Title IV include Pell Grants, Federal Supplemental Education Opportunity Grants (FSEOG), and Student Work Study monies.

These grants do not need to be repaid and would help defray some of the room and board costs not covered by WIOA. Unfortunately, students with autism who are enrolled in a US DOE approved CTP are not eligible for both subsidized and unsubsidized federal student loans under HEOA. By aligning these two pieces of legislation and by lifting the provision against student loan disbursement for students in CTPs, more students on the autism spectrum could attend college-based Comprehensive Transition Programs, practice living independently, and learn independent living skills that would help them keep and maintain employment.

Clearly, future directions will require more research not only into the effects of WIOA on length of employment and wages, but also on the effects of many of its provisions including the provision of pre-employment training, peer mentoring, and one-stop centers for employment services. Research into disability-specific outcomes including autism will be paramount.

See Also

- ▶ [Americans with Disabilities Act](#)
- ▶ [Competitive Employment](#)
- ▶ [Higher Education Opportunity Act of 2008 \(HEOA\)](#)
- ▶ [Intellectual Disability](#)
- ▶ [Section 504 of the Rehabilitation Act of 1973](#)
- ▶ [Vocational Rehabilitation Act of 1973](#)

References and Reading

- 20 Code of Federal Regulations (CFR) Parts, 676, 677, and 678. Department of Labor. Employment and Training Administration. (n.d.).
- 34 Code of Federal Regulations (CFR) Parts 361 and 463. Department of Education. Workforce Innovation and Opportunity, Joint Rule for Unified and Combined State Plans, Performance Accountability, the One-Stop System Joint Provisions; Final Rule. (n.d.).
- 34 CFR Parts 361, 363, and 397. RIN 1820-AB70. [Docket ID ED-2015-OSERS-0001]. State Vocational Rehabilitation Services program; State Supported Employment Services program; Limitations on Use of Subminimum Wage. (n.d.). <https://s3.amazonaws.com/public-inspection.federalregister.gov/2015-05538.pdf>

- Butz, B. (2016). Policy works. Personal Communication 25 Aug 2016. www.disabilitypolicyworks.org
- Diamant, M. (2015). As more with autism reach adulthood clues to success emerge; *Disability Scoop*. Retrieved from <http://www.disabilityscoop.com/2015/5/14/as-autism-adulthood-clues/20299>
- Moore, E. J., & Schelling, A. (2015). Postsecondary inclusion for individuals with intellectual disabilities and its effects on employment. *Journal of Intellectual Disabilities*, 19, 130.
- National Skills Coalition. (2016). *Federal policy. The workforce innovation and opportunity act*. Retrieved from <http://www.nationalskillscoalition.org/federal-policy/workforce-investment-act>. 28 Sept 2016.
- O’Leary, C. J., & Eberts, R. W. (2008). The Wagner-Peyser act and U.S. employment service: Seventy-five years of matching job seekers and employers. Report prepared for Center for Employment Security Education and Research (SESER); National Association of State Workforce Agencies (NASWA). <http://research.upjohn.org/reports/29>
- U.S. Department of Labor. (2016). *The workforce innovation and opportunity act*. Retrieved from <https://www.doleta.gov/wioa/>. 28 Sept 2016.
- Wehman, P. H., et al. (2013). Competitive employment for youth with autism spectrum disorders: Early results from a randomized clinical trial. *Journal of Autism and Developmental Disorders*, 44, 487. <https://doi.org/10.1007/s10803-013-1892>.

Working for Pay

- [Employment](#)

Worry

- [Anxiety](#)

Wounds

- [Injuries in Children with ASD](#)

WPPSI-III

- [Wechsler Preschool and Primary Scale of Intelligence](#)

WPPSI-III – Wechsler Preschool and Primary Scale of Intelligence

- [Wechsler Preschool and Primary Scale of Intelligence](#)

WRAML2

- [Wide Range Assessment of Memory and Learning \(WRAML\)](#)

WRAML-2

- [Wide Range Assessment of Memory and Learning \(WRAML\)](#)

Wraparound Services

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Definition

Wraparound services is a phrase used to describe a comprehensive set of services for an individual with a disability, and/or their family, that addresses all of their educational, vocational, and independent living needs across all parts of their day. Such services may come from a single service provider or from several service providers working in collaboration with one another. Either way, wraparound services usually involves the use of a single service coordinator.

It is reported that the term was first coined by Dr. Lenore B. Behar in an article, “A state model for child mental health services: The North Carolina experience,” published in a 1986 issue of *Children Today*. In that article, she describes

wraparound services as a way to “surround multi-problem youngsters and families with services rather than with institutional walls, and to customize these services to the individual needs and strengths of the child and family” (LenoreBehar.com). The origins of the term specifically relate to services to children and young adults with a mental health diagnosis. Over time, the term has been adopted in various forms by most human service systems serving children and adults, including those systems serving individuals with a developmental disability. A wraparound, or continuum of care, approach to service delivery for individuals with a diagnosis of autism has been identified since the earliest days of the recognition of the syndrome. However, the word “wraparound” or the terms “wraparound process” or “wraparound services” in terms of services to individuals with a developmental disability are usually used as a descriptive term elaborating on a particular therapeutic approach. Examples of these would be applied behavior analysis, person-centered planning, or family-centered care.

Historical Background

The term “wraparound services” has its origins in services to children and youth with a mental health diagnosis in response to a perceived fragmented and disjointed service delivery system. Even in these early uses, the broken service delivery system in question overlapped with the service systems for youth with developmental disabilities as well as the juvenile justice system. Over time, the term has come to be a generic term used in multiple service delivery systems serving youth in need describing a comprehensive set of services for an individual and/or their family that addresses all of their educational, vocational, and independent living needs across all parts of their day.

Origins of the Wraparound Process

In the early 1980s, North Carolina’s Division of Mental Health, Developmental Disabilities, and

Substance Abuse Services responded to a lawsuit, known as the “Willie M.” lawsuit, on behalf of children diagnosed with severe emotional disabilities. The state office used the consent agreement as a means of developing “a comprehensive continuum of community-based care for high-risk and hard-to-manage children and adolescents” (p. 118, Jenson and Fraser 2005). In 1986 Dr. Lenore Behar, then the Chief of Child Mental Health Services for the state of North Carolina, coined the term “wraparound” in an article (Behar 1986) describing the service system for youth with a mental health diagnosis that North Carolina had developed in response to the Willie M. litigation. At that time, she described wraparound services as a way to “surround multi-problem youngsters and families with services rather than with institutional walls, and to customize these services to the individual needs and strengths of the child and family” (LenoreBehar.com/wraparound, n.d.).

Beginning in the early 1970s, the Kaleidoscope program in Chicago, led by Executive Director Karl Dennis, began to develop a community-based, family-centered approach to providing services to children with severe emotional disabilities who had been institutionalized across the country. While the Kaleidoscope model was not initially characterized as wraparound services, it is later identified as being consistent with the philosophies of the wraparound process and one for the earliest examples of the application of wraparound approach. The Kaleidoscope model envisions wraparound process as having five essential features: development of a service plan by an interdisciplinary team, community based, family focused, unconditional, and cost-effective. In the mid-1980s, the Kaleidoscope program provided consultation to officials in the state of Alaska in the development of a new program called the Alaska Youth Initiative. The program, lead by John VanDenBerg, was one of the earliest (along with North Carolina) statewide efforts to use a wraparound service model as a means of returning youth with complex mental health needs who had been placed out of state to community-based settings.

Federally Funded Programs and Wraparound Services

Wraparound services have been a central component of several federal mental health-related initiatives. The National Institute of Mental Health has funded a program called the Child and Adolescent Services System Program (CASSP) since the early 1980s. This program originally funded by Congress in 1984 grew out of a national study that found that two-thirds of all children with severe emotional disturbances were not receiving appropriate services. The CASSP approach, identified as consistent with the wraparound process, advocates for six core principles: child centered, family focused, community based, multisystem, culturally competent, and least restrictive/intrusive.

The Substance Abuse and Mental Health Services Administrations has given grants to communities across the country via its Comprehensive Community Mental Health Services for Children and Families program. The wraparound process has been a main component of a majority of these initiatives.

Early and Periodic Screening, Diagnosis, and Treatment (EPSDT) is a mandated Medicaid service for children and youth. While not specifically designed to support wraparound services, numerous states have used EPSDT as a means of funding community-based wraparound services for children and youth with mental health diagnoses as well as children and youth with developmental disabilities.

Wraparound-Type Services for Individuals with a Developmental Disability

A majority of the service models for individuals with a diagnosis of autism are consistent with the basic philosophies of the wraparound process as delineated by the National Wraparound Initiative (NWI). As delineated in the current knowledge section, NWI has identified ten principles of the wraparound process: family voice and choice, team based, natural supports, collaboration, community based, culturally competent, individualized, strengths based, unconditional, and outcome based. Two particular service models

for individuals with autism consistent with a majority of these principles are person-centered planning and Applied Behavior Analysis.

Person-centered planning is an approach aimed at improving the quality of services for individuals with a developmental disability mostly associated with the work of John O'Brien. In their consideration of the history of person-centered planning, Lyle O'Brien and O'Brien (2000) identify four concurrent initiatives in the early 1980s that they see as the foundations of what ultimately becomes known as person-centered planning. The Canadian National Institute on Mental Retardation was training staff and families in a process of 24-hour planning for individuals with severe disabilities. Beth Mount in Georgia was developing an approach she called Personal Futures Planning. Jack Yates in Massachusetts was developing the Program Design Sessions for individuals moving out of an institutional setting, and Wisconsin's system was identifying the capacities it would need to develop in order to deliver individualized services (p. 3). In turn, those involved in all of these initiatives were influenced by the definition of the principal of normalization as defined by Wolf Wolfensberger and L. Glenn in their book (and related training programs) *Program Analysis of Service Systems* (Pass) (1975).

Current Knowledge

Applied Behavior Analysis

As noted elsewhere in this chapter, the term "wraparound" is used both generically and increasingly in a very purposeful way to refer to a particular means of planning and service delivery ascribing to a specific set of agreed-upon principles. The generic use of the term refers to any instance of a comprehensive set of services that addresses all of a person's educational, vocational, and independent living needs across all parts of their day. The more concrete use of the term being advocated by the National Wraparound Initiative is described below.

The current best practices models of services for individuals with a diagnosis on the autism

spectrum stress team-based comprehensive approaches that span both the whole day and the whole year. There is not a lot of evidence that the majority of the treatment efforts for individuals with a diagnosis of autism are affiliated with the National Wraparound Initiative. However, it is clear that some of the national advisors for the National Wraparound Initiative are involved in services to individuals with a diagnosis of autism and other developmental disabilities. It is also clear that the most dominant approaches to providing services to individuals with a diagnosis with autism, such as applied behavior analysis and person-centered planning are focused on team-based and 24-h inclusive approaches. The main assumptions of the best practice approaches to providing services to individuals with a diagnosis of autism are consistent with a majority of the core principals of the wraparound process as delineated by the National Wraparound Initiative.

Ten Principles of the Wraparound Process as Developed by the National Wraparound Initiative

In 2003, a group of community leaders and researchers who were part of the larger practice community of wraparound services convened a national meeting to consider the necessary and sufficient components of the wraparound process. The meeting, hosted by the Research and Training Center on Family Support and Children's Mental Health, was the beginnings of what became the advisory group to the National Wraparound Initiative (NWI). The National Wraparound Initiative represents a group of "stakeholders-including families, youth, providers, researchers, trainers, administrators and others- [who] came together in a collaborative effort to better specify the wrap-around practice model, compile specific strategies and tools, and disseminate information about how to implement wraparound in a way that can achieve positive outcomes for youth and families" (National Wraparound Initiative [n.d.](#)).

A major initiative of the National Wraparound Initiative has been an online Resource Guide to Wraparound. A key component of this resource guide is a set of ten principles of wraparound developed by consensus by the advisors and

principals of the National Wraparound Initiative. These ten principles are as follow:

1. Family voice and choice: An emphasis on the primary importance of goals and perspective of the individual receiving services and their family and advocates in the development of the wraparound process. This principle stresses the importance of intentional activities to illicit and includes the perspective of the individual receiving services and their family and advocates.
2. Team based: This principal stresses the importance of collaborative effort of family members, professionals, and other stakeholders, committed to the family's well-being over an extended period of time. The choice of team members should be largely driven by the person receiving services and their family and advocates.
3. Natural Supports: To the greatest extent possible, a wraparound plan of service should utilize the natural support systems of family members, friends, neighbors, church, and community members. The plan should also include the regular support structures that exist in the community via school systems, church congregations, community centers, local government, etc.
4. Collaboration: The decision-making process in developing a wraparound plan of service should be based on a consensus approach that includes input from all team members.
5. Community based: Wraparound services should adhere to a principle of provision in the least restrictive setting possible.
6. Culturally competent: Team designation, service planning, and service delivery should demonstrate "respect for the values, preferences, beliefs, culture and identity of the child/youth and family, and their community" (Bruns et al. 2008, p.7).
7. Individualized: Wraparound services need to be uniquely developed for the individual in need and their family. The planning for services should draw upon the best empirical evidence of effective treatment and upon community and professional experience.

However, the services should not be assembled from a static list of available services.

8. Strengths based: A key in the development of a wraparound plan of service is to identify, “build on, and enhance the capabilities, knowledge, skills, and assets of the child and family, their community, and other team members” (p. 8).
9. Unconditional: The origins of the wraparound process grew out of a need to provide quality services to individuals with severe and complex behaviors. It is understood at the outset that this will be a difficult and challenging process. Inherent in the development of a wraparound plan of service is a commitment to see the process through despite setbacks and unanticipated behavior, events, or outcomes. There needs to be an unwavering commitment of the part of the team to continually adapt the plan of service until progress is made and there is consensus that a wraparound process is not longer needed.
10. Outcome based: Wraparound plans of service identify measurable outcomes and indicators of progress and success. The team measures and evaluates these measures on an ongoing basis and modifies plans accordingly (Bruns et al. 2008).

Future Directions

The future direction of wraparound services will depend upon the research community identifying more empirically based interventions specifically designed to help ameliorate the behaviors associated with autism spectrum disorders that interfere with social communication, independent living, vocational skills acquisition, and community integration.

See Also

- Applied Behavior Analysis (ABA)
- Ecological Model of Autism

References and Reading

- Behar, L. B. (1986). A state model for child mental health services: The North Carolina experience. *Children Today*, 15(3), 16–21.
- Bruns, E. J., Walker, J. S., & The National Wraparound Initiative Advisory Group. (2008). Ten principles of the wraparound process. In E. J. Bruns & J. S. Walker (Eds.), *The resource guide to wraparound*. Portland: The National Wraparound Initiative.
- Bruns, E. J., Walker, J. S., Zabel, M., Matarese, M., Estep, K., Harburger, D., Mosby, M., & Pires, S. A. (2010). Intervening in the lives of youth with complex behavioral health challenges and their families: The role of the wraparound process. *American Journal of Community Psychology*, 46, 314–331.
- Dennis, K. W., & Lourie, I. S. (2006). *Everything is normal until proven otherwise: A book about wraparound services*. Washington, DC: Child Welfare League of America.
- Fisher, W. W., Piazza, C. C., & Roane, H. S. (Eds.). (2011). *Handbook of applied behavior analysis*. New York: Guilford Press.
- Jenson, J. M., & Fraser, M. W. (Eds.). (2005). *Social policy for children and families: A risk and resilience perspective*. Thousand Oaks: Sage.
- Lyle O'Brien, C., & O'Brien, J. (2000). The origins of person-centered planning: A community of practice perspective. Retrieved from http://thechp.syr.edu/PCP_History.pdf
- O'Brien, J. (1989). *Improving the quality of services for people with developmental disabilities: It's everybody's business*. Baltimore: Paul Brookes.
- The National Wraparound Initiative. (n.d.). The national wraparound initiative: About NWI. Retrieved 26 June 2012. <http://www.nwi.pdx.edu/index.shtml>
- Walke, J. S., Bruns, E. J., & Penn, M. (2008). Individualized services in systems of care: The wraparound process. In B. A. Stroul & G. M. Blau (Eds.), *The system of care handbook: Transforming mental health services for children, youth, and families*. Baltimore: Paul Brookes.
- Wolfensberger, W. (1972). *The principle of normalization in human services*. Downsview: National Institute on Mental Retardation.
- Wolfensberger, W., & Glenn, L. (1975). *Program analysis of service systems (PASS)*. Downsview: National Institute on Mental Retardation.

WRAVMA

- Visual-Motor Integration, Developmental (VMI) Test

Writing

► Literacy

Writing Disorders

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Short Description or Definition

Writing is considered to have two basic components: the process and product of writing. Writing processes include “planning (prewriting), organizing, drafting, reflecting, revising, and editing. . . as well as forming letters and sequences of letters into words” (American Speech Language Hearing Association [ASHA] 2001), while the products of writing are the final form of the composition produced as either handwritten or word-processed text. Writing involves not only the physical process of forming letters and sequences of letters into words and using appropriate writing mechanics, such as punctuation and capitalization, then, but also consists of the planning, organizing, drafting, reflecting, revising, and editing that leads to the final product (Paul and Norbury 2012; Scott et al. 2009). Children who find writing difficult early in their education often perform poorly on academic measures as they progress through the grades (Nancollis et al. 2005). Children with speech-language disabilities, including those with ASD, are at risk for writing difficulties (Puranik et al. 2008).

Assessment

Writing can be assessed informally, using rubrics to rate the quality of various components of the writing process and product (e.g., Paul and Norbury (2012); Westby and Clauser 2005). In addition, standardized tests are available for the identification of writing disorders. These may be

administered by a speech-language pathologist, learning disability specialist, or special educator. They include, for example, the *Oral and Written Language Scales* (Carrow-Woolfolk 1996), the *Test of Adolescent and Adult Language-4* (Hammill et al. 2007), the *Test of Written Expression* (McGhee et al. 1995), the *Test of Written English* (Anderson and Thompson 1988), the *Test of Written Language-4* (Hammill and Larsen 2009), the *Woodcock Language Proficiency Battery-Revised* (Woodcock 1991), the *Writing Process Test* (Warden and Hutchinson 1992), and the *Written Language Assessment* (Grill and Kerwin 1990).

Categorization

The characteristics of writing disorders, according to Scott et al. (2009), include:

- Poor *planning, monitoring, evaluating*, and *revising* work. Children with writing difficulties have been described as having a “stream of consciousness” writing style (Graham and Harris 1999). Because of this, they demonstrate text-structure problems (with paragraph organization, transitions between paragraphs, and story plots). They often do not recognize and correct errors as they write (Roth 2000) or go back over their writing to correct errors later (Scott 2005).
- Difficulty with *writing mechanics*. Graham and Harris (1999) and MacArthur (2000) observe that children with writing difficulties struggle with spelling, capitalization, punctuation, and handwriting. In addition, they often write sentences that are shorter and simpler than those of their peers. They have fewer clauses per sentence, they overuse *and* to begin sentences, and often omit grammatical word endings, such as *-ed* (to express past tense).
- *Sparse writing*. Children who struggle with writing write less and get less practice writing (Wong 2000). Because of their language deficits, these children often have limited vocabularies and may not know the right word to

express their ideas. They may forget the main point, plan, or the structure of their text because they got stuck on the mechanical process of writing. These findings were confirmed by Scott (2005), who found that problems with thinking through and controlling the steps of writing create difficulty in both excluding unimportant material as well as including appropriate information.

- *Ineffective revision.* Children with writing difficulties tend to view revising as proofreading. Revisions are typically made at the sentence level, without considering changing content (Roth 2000).

Epidemiology

There are no precise estimates of prevalence for writing disorders, although the National Center for Learning Disabilities estimates that 5% of school-aged children have learning disorders; most who have disabilities in the area of reading will have writing disorders, as well.

Natural History, Prognostic Factors, and Outcomes

Natural History: Some children may have difficulty with the physical act of writing, the ability to form letters, as early as the preschool years. This specific deficit in handwriting is often referred to as *dysgraphia*. For most children, writing problems will be identified in 2–4th grade, when school-sponsored writing and spelling are targeted within the curriculum. These difficulties usually accompany reading problems and executive function difficulties and can persist throughout development, into adulthood. However, many adolescents and adults can develop compensatory strategies to that help them cope with their weaknesses and can find careers that allow them to use their strengths for successful adjustment.

Prognostic Factors: Children with reading disorders are at risk for writing disorders, as well.

Outcomes: Although deficits in spelling, handwriting, and composition often persist into

adulthood, many adults find ways to be successful in work and life.

Clinical Expression and Pathophysiology

No pathophysiological signs have been identified that are specific to developmental writing disorders, although adults with acquired dysgraphia are generally found to have lesions in the parieto-occipital region.

Evaluation and Differential Diagnosis

Evaluation and Differential Diagnoses: Writing disorders that are specific to letter formation and handwriting (dysgraphia) need to be distinguished for general motor weakness, spasticity, and incoordination that would affect processes other than writing, such as self-feeding and other hand skills. Since writing disorders almost always occur in conjunction with reading disorders, the two do not require differential diagnosis, but writing ability should always be evaluated in students with reading disorder.

Treatment

Intervention

Several aspects of writing may be addressed in an intervention program, including:

- *Writing mechanics.* Children who have specific difficulties with writing mechanics may demonstrate poor handwriting skills may fatigue or experience hand cramps quickly when writing, and may be “poor spellers.” If this is the case, the use of assistive technology, such as word processing, can be utilized effectively to reduce fine-motor demands of handwriting (MacArthur 2000). In addition, the spell check function in a word processing program can be helpful for a child who experiences difficulty with writing mechanics and spelling (MacArthur 2000). Working with word prediction software in intervention is also an option

for children with problems in writing mechanics. This software predicts a word to type, based on what has been written so far, reducing the stress of both word finding and spelling.

- *Planning and composing.* One effective strategy for improving writing process is *Self-Regulated Strategy Development* (SRSD; Graham et al. 2000). There are six “stages” in the SRSD: (1) develop background knowledge; (2) identify strategy, goals, and significance; (3) modeling of the strategy; (4) memorization of the strategy; (5) collaborative practice; and (6) independent practice. A variety of other strategies are described in Paul and Norbury (2012).
- *Revising.* Children with writing disorders may benefit from the use of assistive technology, such as speech synthesis software that allows the computer to read the composition aloud, while highlighting the words being spoken. The student can then use oral language skills to make revisions. Collaborating with classmates or teachers during revision may also be helpful (MacArthur 2000; Wong 2000). Students can be divided into small groups for peer revisions. Each child is instructed to provide, for example, two positive comments and two suggestions for improvement for their classmates’ compositions. The students might also ask questions about unclear or underdeveloped aspects of compositions.
- *Visual aids and graphic organizers* have also been shown to improve writing. Visual outlines in the form of flow charts and maps (“webs”) can help students organize the writing process (Hyerle 2004). These can be used as a basis for creating a more mature composition.

Additional examples of intervention approaches for writing difficulties can be found in Paul and Norbury (2012).

See Also

- [Dysgraphia](#)
- [Learning Disabilities](#)

References and Reading

- American Speech Language Hearing Association. (2001). Roles and responsibilities of speech-language pathologists with respect to reading and writing in children and adolescents (position statement, executive summary of guidelines, technical report). *ASHA Supplement 21*, (pp. 17–27). Rockville: Author.
- Anderson, V., & Thompson, S. (1988). *Test of written english (TWE)*. Novato: Academic Therapy.
- Apel, K., & Masterson, J. (2001). Theory guided spelling assessment and intervention: A case study. *Language, Speech, and Hearing Services in School*, 32, 182–195.
- Berninger, V. (2000). Development of language by hand and its connections with language by ear, mouth, and eye. *Topics in Language Disorders*, 20, 65–84.
- Carrow-Woolfolk, E. (1996). *Oral and written language scales (OWLS)*. Chicago: Riverside Publishing.
- Graham, S., & Harris, K. (1999). Assessment and intervention in overcoming writing difficulties: An illustration from the self-regulated strategy development model. *Language, Speech, and Hearing Services in School*, 30, 255–264.
- Graham, S., Harris, K., & Troia, G. (2000). Self-regulated strategy development revisited: Teaching writing strategies to struggling writers. *Topics in Language Disorders*, 20, 1–14.
- Grill, I. J., & Kerwin, M. M. (1990). *Written Language Assessment Test*. Novato: Academic Therapy Publications.
- Hammill, D. D., Brown, V. L., Larsen, S. C., & Wiederholt, J. L. (2007). *Test of adolescent and adult language-4*. Austin: Pro-Ed.
- Hammill, D. D., & Larsen, S. C. (2009). *Test of written language-4 (TOWL-4)*. Novato: Academic Therapy Publications.
- Hyerle, D. (Ed.). (2004). *Student successes with thinking maps: School-based research, results, and models for achievement using visual tools*. Thousand Oaks: Corwin Press.
- Lanter, E., & Watson, L. (2008). Promoting literacy in students with ASD: The basics for the SLP. *Language, Speech, and Hearing Services in Schools*, 39, 33–43.
- MacArthur, C. (2000). New tools for writing: Assistive technology for students with writing difficulties. *Topics in Language Disorders*, 20, 85–100.
- McGhee, R., Bryant, B., Larson, S., & Rivera, D. (1995). *Test of written expression models for achievement using visual tools*. Thousand Oaks: Corwin Press.
- Nancollis, A., Lawrie, B., & Dodd, B. (2005). Phonological awareness intervention and the acquisition of literacy skills in children from deprived social backgrounds. *Language, Speech, and Hearing Services in School*, 36, 325–335.
- Paul, R., & Norbury, C. (2012). *Language disorders from infancy through adolescence: Listening, speaking, reading, writing, and communicating* (4th ed.). St. Louis: Elsevier.
- Puranik, C., Lombardino, L., & Altmann, L. (2008). Assessing the microstructure of written language

- using a retelling paradigm. *American Journal of Speech-Language Pathology*, 17, 107–120.
- Roth, F. (2000). Narrative writing: Development and teaching with children with writing difficulties. *Topics in Language Disorders*, 20, 15–28.
- Scott, C. (2005). Learning to write. In H. W. Catts & A. G. Kamhi (Eds.), *Language and reading disabilities* (2nd ed., pp. 233–273). Boston: Pearson.
- Scott, C., Fahey, C., Soukup, K., Walsh, K., Einck, R., Gale, J., et al. (2009, November). *Four letters to parents about children's writing impairments*. Presented at National Convention of the American Speech-Language-Hearing Association, New Orleans.
- Westby, C., & Clauser, P. (2005). The right stuff for writing: Assessing and facilitating written language. In H. Catts & A. Kamhi (Eds.), *Language and reading disabilities* (2nd ed., pp. 274–340). Boston: Allyn & Bacon.
- Warden, M., & Hutchinson, T. (1992). *Writing process test*. Austin: Pro-Ed.
- Wong, B. (2000). Writing strategies instruction for expository essays for adolescents with and without learning disabilities. *Topics in Language Disorders*, 20, 29–40.
- Woodcock, R. W. (1991). *Woodcock language proficiency battery-Revised*. Chicago: Riverside Publishing.

Writing Interventions for Individuals with Autism Spectrum Disorder

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Definition

Writing is a means to communicate and express ideas, thoughts, feelings, and information. Writing interventions refer to effective and evidence-based instructional practices used to support individuals in developing varied elements of writing including but not limited to letter formation, handwriting, length of writing, sentence structure, and quality of writing. Writing interventions often target specific genres of writing including but not limited to descriptive writing, expository writing, narrative writing, and persuasive writing.

Historical Background

The topic of writing interventions and students with autism spectrum disorder (ASD) was not widely considered prior to the twenty-first century. Preceding the enactment of legislation in the 1970s, students with disabilities were denied access to general education. In 1973, Section 504 of the Rehabilitation Act was signed, mandating the inclusion of individuals with disabilities in all programs that receive federal funding, including public schools. In 1975 the Education for All Handicapped Children Act (P.L. 94-142) was signed mandating the rights of students with disabilities to a free and appropriate education in the least restrictive environment. Despite these mandates including students in public education, there was a prevailing assumption that most individuals with ASD would not benefit from instructional time spent on academic skills including writing, and instead the primary focus of education was to address behavioral and life skills.

In 1990, P.L. 94-142 was renamed as the Individuals with Disabilities Education Act (IDEA). IDEA was reauthorized in 1997 with an impactful amendment that required students with disabilities to participate in local and state assessments. IDEA was further reauthorized as the Individuals with Disabilities Education Improvement Act (IDEIA) in 2004, mandating students with disabilities receive academic instruction founded in scientifically based research. These mandates have brought about increased expectations for the academic performance of students with disabilities and the increasing inclusion of students with disabilities in general education classrooms. This includes access to the general education curriculum and participation in high-stakes testing for students with ASD in our public schools.

Students with ASD, however, have been found to struggle with academic performance, including in the domain of written expression, yet skill in writing is paramount to success in local and state assessments. Moreover, developing skill in functional writing as a communication tool is especially important for the subset of individuals with autism spectrum disorder (ASD) with limited

or unreliable speech. Faced with the mandates of IDEIA (2004), many teachers have found themselves unprepared to provide instruction that meets the individualized needs of students with ASD as well as prepares them to meet state standards. This established a need for high-quality writing interventions for students with ASD. Fortunately, there has been increased attention on how to enhance the academic performance of students with ASD in the area of writing.

Current Knowledge

Individuals with ASD often experience reduced success in writing, placing them at a disadvantage in communicating their thoughts, fully participating in school, or gaining employment (Pennington and Delano 2012). Specific writing difficulties include using fewer words and making more spelling errors than their neurotypical peers and having weaker overall structure in their writing (Asaro-Saddler 2014, 2016). In a study of the learning profiles of children with high-functioning autism, Mayes and Calhoun (2008) report that despite strength in visual and verbal reasoning, students with ASD have writing weaknesses, including difficulty with handwriting and with expressing thoughts on paper. Additionally, difficulty with perceptual and visual motor tasks has been found to impact functional handwriting including length, speed, and legibility of writing (Ashburner et al. 2012).

Differences in executive functioning and theory of mind (ToM) have frequently been reported to impact the writing of individuals with ASD. Executive functioning differences may result in difficulty generating, planning, and integrating ideas in writing for individuals with ASD, and as a result instructional practices aimed at supporting the planning, organization, and self-monitoring of writing emerge as essential (Delano 2007). Differences in ToM, the ability to consider the thoughts or feelings of others (Baron-Cohen et al. 1985), have been reported as impacting the ability of individuals with ASD to write persuasively (Asaro-Saddler and Bak 2014) and the quality of both narrative and expository texts

(Brown and Klein 2011). More recently, an emphasis on ToM differences as opposed to deficits has led to consideration of how ToM variations may also emerge as strengths for individuals with autism (see Atherton et al. 2019). For example, strength in imagination and attention to detail may enrich the narrative story writing of individuals with ASD.

Writing Interventions

Numerous interventions have been identified to improve the writing of individuals with ASD. Many of these writing interventions stem from practices identified as evidence-based (EBPs) for instructing students with ASD in a variety of skill areas (see Reichow et al. 2008). Such EBPs include reinforcement, task analysis, prompting, time delay, video modeling, and use of visual supports (National Professional Development Center on ASD 2014). In fact, combining multiple instructional practices to individualize writing intervention for individuals with ASD has emerged as a common practice with interventions frequently combining visual supports, such as a graphic organizer, along with positive reinforcement (Accardo et al. 2019). Reviews of the literature detail writing interventions to support individuals with ASD (see Pennington and Delano 2012; Asaro-Saddler 2014; Accardo et al. 2019). Recommended writing interventions along with examples of implementation follow.

Self-regulated strategy development. Self-regulated strategy development (SRSD) is an instructional package that includes the teaching of academic strategies for writing combined with instruction in self-regulation. Through SRSD, teachers individualize instruction and guide students to use writing strategies while also providing support for student goal setting, self-regulation, and motivation. Instruction includes six stages: (1) developing background information needed for writing; (2) discussing the purpose and benefit of the strategy to be used; (3) modeling of the strategy, often through teacher think-alouds; (4) memorizing the strategy steps, often through the use of a mnemonic (e.g., the mnemonic POW+TREE for the strategy steps: Pick an idea, Organize notes, Write, Topic sentence,

three or more Reasons, Ending, Examine); (5) supporting students in use of the strategy; and (6) independence, evidenced by student generalization of the strategy across a variety of tasks (Santangelo et al. 2008). SRSD is individualized with instruction at each stage continuing until student mastery through a criterion-based approach.

SRSD has emerged as a highly effective writing intervention for individuals with learning disabilities as well as individuals with ASD. In a study of the use of SRSD with three elementary-age students with ASD, students were taught to use the POW+ Tree strategy and the WWW, What-2, and How-2 writing strategy, given a choice of writing tasks, a motivational “rocket” chart, and visual supports. All of the students increased the number of story elements used, the holistic quality, and the number of words when writing stories (Asaro-Saddler 2014). Similarly, in a study of SRSD which included choice of writing tasks, self-regulation charts, cue cards, and a computer graphic organizer program, all of the students aged 18–20 increased the quality of argument-driven writing and writing duration (using the strategy DATE + SCORES) and quality appeared to generalize to prompts in a college writing course (Jackson et al. 2018). In a review of the research literature on the effectiveness of SRSD with individual with ASD, Asaro-Saddler (2016) concluded that most students with ASD participating in SRSD instruction increased both the quality and quantity of their writing. In addition, students increased their planning time and their use of self-regulatory strategies. Since many students with ASD are reported to have difficulty with executive functioning tasks such as planning, organizing, and self-monitoring, the SRSD method may help these students to organize their thoughts and to check the quality of their writing.

Graphic organizers. The use of visual information during instruction, such as through graphic organizers and pictorial representations, allows students to process information faster and easier. Graphic organizers provide a visual representation of information to enhance learning. As a component of the SRSD method, graphic organizers have been shown to be part of an effective

instructional package for enhancing the writing skills of individuals with ASD. For example, Bishop et al. (2015) implemented a graphic organizer package to improve the persuasive writing of three middle school students with ASD. The graphic organizer had spaces for planning – a brainstorming box, three boxes for reasons, a counter-argument box, an introduction box, and a conclusion box. After training with the graphic organizer, all three students improved on all three of the outcome measures – correct writing sequences (defined as “two adjacent writing units that are correct within the context of what is written”), total words written, and a rubric score.

Behavioral Principles

Prompting. Instructional methods derived from applied behavior analysis (ABA) are also recommended to enhance the writing of individuals with ASD (see ► “Progressive Applied Behavior Analysis”), often in combination with other instructional methods. One such method is prompting. For example, response prompting was combined with sentence frames to improve the sentence writing of 7–12-year-old students with moderate intellectual disability and ASD (Pennington et al. 2018). A sentence frame is a scripted portion of a sentence – for example, “I want _____” – that can be completed by using a targeted written response such as “I want the candy.” During instruction, the teacher presented the student with a preferred item and the sentence frame and prompted the student to respond by saying “Write a sentence to tell me what you want.” If the student failed to respond within 10 s, the teacher prompted the student using a predetermined prompt hierarchy. If the student responded correcting the teacher provided either p-raise or a tangible reinforcer. Using these procedures, each of the students produced three types of sentences and maintained their responding following the intervention.

Task Analysis. Similarly, a task analysis approach can be used to enhance the writing of individuals with ASD. For example, Lee et al. (2016) used a task analysis approach to identify the steps needed to teach three skills: identification of key ideas in a paragraph, identifying

supporting details and recording information on a graphic organizer, and composing informational text. Two students with significant intellectual disabilities, one of whom was identified as autistic, were instructed on writing skills using the task analyses as the framework for the instruction. In addition, during instruction teachers used a graphic organizer that consisted of written prompts for each of the three target skills and utilized a least intrusive prompt approach if the student did not respond during the lesson. Following instruction, both of the students made significant improvements on all three skills.

Reinforcement. Reinforcement of behavior is another ABA method that has frequently been used to teach writing skills. In their review of the research on writing interventions for students with ASD, Pennington and Delano (2012) identified 11 studies in which reinforcement was used. A number of different methods were identified, including the delivery of tokens, a computer-delivered auditory fanfare for correct responding, and a reinforcing video clip. Since student preferences can vary widely, it is important to assess individual student response to reinforcers before choosing a method to use to reinforce student responses to writing tasks.

Assistive Technologies

Computer-aided instruction. Assistive technologies, including computer-aided instruction, voice output communication aids (VOCAs), multimedia presentations, and video modeling, have been successfully used to improve the writing of individuals with ASD. Using computer-aided instruction (CAI), Stromer et al. (1996) developed software to teach a student with intellectual disability and ASD to improve spelling. The student was presented with a sample word on the computer screen which, when touched, disappeared and was replaced by a set of letters. The student could then select the correct letters to spell the target word. When the student responded correctly, the computer lit up with a flashing display, and the student received a token that could be exchanged for a preferred item. The student with ASD was able to successfully spell words using this method, and

spelling improvement was found to generalize to other tasks.

Additional studies provide example of how CAI, used in combination with other instructional practices, can be used to teach writing skills to individuals with ASD. Pennington et al. (2014) combined simultaneous prompting, an errorless teaching procedure involving the delivery of a controlling prompt immediately following a discriminative stimulus, with CAI to teach story writing to five students with autism (see Pennington et al. 2010, 2014). Story templates were created using commercially available software, and the participants were prompted to create a story using words and pictures on the story template. In both studies the researchers found that the combination of simultaneous prompting with computer-aided instruction improved the ability of the students to construct stories. Additionally, students showed evidence of generalizing their story construction skills through increased verbal responses.

Voice output communication aids. Assistive technology includes the use of voice output communication aids to support the written expression of individuals with ASD. Two studies by Schlosser and colleagues combined the use of a voice output communication aid (Lightwriter 35) with a copy-cover-compare method to teach spelling to students with ASD (see Schlosser et al. 1998; Schlosser and Blischak 2004). In the first study, a student with ASD significantly increased correct spelling of words and maintained the improvement after the intervention was completed. In the follow-up study in 2004, the investigators used a similar copy-cover-compare method to teach spelling combined with the use of three different forms of feedback – speech, print, and speech + print – with four students with ASD. Results were similar to that found in the earlier study in that all of the participants increased their correct spelling. However, only two of the four students were able to generalize their skills to new words. There was no single type of feedback that was superior, but the results suggest that some individuals with ASD may respond better to print feedback, while others may prefer feedback in the form of speech.

Video modeling. Video modeling has been found to be an evidence-based practice for improving the learning and behavior of individuals with ASD (Wong et al. 2015). When applied to writing instruction, video modeling has been found to enhance essay writing (Delano 2007) and spelling (Kinney et al. 2003; Kagohara et al. 2012). For example, SRSD was delivered via video self-modeling to three young adults with Asperger syndrome (Delano 2007). The young adults participated in making two videos of themselves modeling the use of a strategy – one to increase the number of words written and the other to enhance essay writing. The students watched the video at the beginning of each intervention session. Following the interventions, all of the students increased the number of words used in their essay writing and increased the number of functional elements (e.g., premises, reasons, conclusions, and elaborations) in their writing. In other studies video modeling has been used to improve spelling. Kinney et al. (2003) developed video models that showed one of the researchers writing a word in response to a prompt from an off-camera investigator. After writing the word, the first investigator said the word. These videos were subsequently used by the classroom teacher to teach new words to the student. The researchers reported that the student readily participated in the instructional sessions, learned a large number of new words, and maintained her knowledge of most of the new words over time. Kagohara et al. (2012) sought to increase the use of the spell-check function of two students with ASD through the use of video modeling. Both of the students successfully learned to use the spell-checker and continued to use the method after the video modeling intervention was discontinued.

Future Directions

Studies of writing interventions have clearly established that individuals with ASD can benefit from such instruction. This is true for both those individuals with lower measured cognitive functioning and those with IQ scores in the average to

higher range. A number of different instructional practices have been found to improve specific writing skills although the most effective approaches often use a combination of methods including self-regulation, strategy instruction, graphic organizers, reinforcement, prompting, and technology. Future research should focus on which of these methods is most useful for the improvement of specific skills (e.g., spelling, written expression, planning) and for which subset of individuals with ASD (e.g., those with lower measured cognitive ability or those higher cognitive functioning).

Additionally, the effectiveness of technology to enhance writing skills has only begun to be explored. The proliferation of technology, in particular speech recognition software and text-to-speech software, has yet to be examined as potential methods for enhancing the written language skills of individuals with ASD. When combined with some of the other evidence-based practices for improving writing skills, the impact of both technology and these practices can potentially be multiplied. The future of research and practice in enhancing the written language skills of individuals with ASD is very promising. The research demonstrates that writing skills can be successfully taught and that individuals with ASD can potentially be more successful both in school and in post-secondary education as well as in employment.

See Also

- ▶ [Academic Skills](#)
- ▶ [Computer-Assisted Instruction to Teach Academic Skills](#)
- ▶ [Education](#)
- ▶ [Inclusion](#)
- ▶ [Progressive Applied Behavior Analysis](#)
- ▶ [Video Modeling/Video Self-Modeling](#)

References and Reading

- Accardo, A. L., Finnegan, E. G., Kuder, S. J., & Bomgardner, E. M. (2019). Writing interventions for individuals with autism spectrum disorder: A research synthesis. *Journal of Autism and*

- Developmental Disorders*. <https://doi.org/10.1007/s10803-019-03955-9>.
- Asaro-Saddler, K. (2014). Self-regulated strategy development: Effects on writers with autism spectrum disorders. *Education and Training in Autism and Developmental Disabilities*, 49, 78–91. Retrieved from <http://www.jstor.org/stable/23880656>
- Asaro-Saddler, K. (2016). Writing instruction and self-regulation for students with autism spectrum disorders: A systematic review of the literature. *Topics in Language Disorders*, 36(3), 266–283. <https://doi.org/10.1097/TLD.0000000000000093>.
- Asaro-Saddler, K., & Bak, N. (2014). Persuasive writing and self-regulation training: Pairing writers with autism spectrum disorder. *Journal of Special Education*, 48, 92–105.
- Ashburner, J., Ziviani, J., & Pennington, A. (2012). The introduction of keyboarding to children with autism spectrum disorders with handwriting difficulties: A help or a hindrance? *Australasian Journal of Special Education*, 36, 32–61.
- Atherton, G., Lummis, B., Day, S. X., & Cross, L. (2019). What am I thinking? Perspective-taking from the perspective of adolescents with autism. *Autism*, 23(5), 1186–1200. <https://doi.org/10.1177/1362361318793409>.
- Baron-Cohen, S., Leslie, A., & Frith, U. (1985). Does the autistic child have “theory of mind”? *Cognition*, 21, 31–46. [https://doi.org/10.1016/0010-0277\(85\)90022-8](https://doi.org/10.1016/0010-0277(85)90022-8).
- Bishop, A. E., Sawyer, M., Alber-Morgan, S. R., & Boggs, M. (2015). Effects of a graphic organizer training package on the persuasive writing of middle school students with autism. *Education and Training in Autism and Developmental Disabilities*, 50, 290–302. Stable URL: <http://www.jstor.org/stable/24827511>
- Brown, H. M., & Klein, P. D. (2011). Writing, Asperger syndrome and theory of mind. *Journal of Autism and Developmental Disorders*, 41(11), 1464–1474. <https://doi.org/10.1007/s10803-010-1168-7>.
- Delano, M. E. (2007). Use of strategy instruction to improve the story writing skills of a student with Asperger syndrome. *Focus on Autism and Other Developmental Disabilities*, 22(4), 252. <https://doi.org/10.1177/10883576070220040701>.
- Individuals with Disabilities Education Improvement Act of 2004, 20 U.S.C §1400, Pub. L. No. 108–446.
- Jackson, L., Duffy, M., Brady, M., & McCormick, J. (2018). Effects of learning strategy training on the writing performance of college students with Asperger’s syndrome. *Journal of Autism and Developmental Disorders*, 48(3), 708–721.
- Kagohara, D. M., Sigafos, J., Achmadi, D., O’Reilly, M., & Lancioni, G. (2012). Teaching children with autism spectrum disorders to check the spelling of words. *Research in Autism Spectrum Disorders*, 6, 304–310.
- Kinney, E. M., Vedora, J., & Stromer, R. (2003). Computer-presented video models to teach generative spelling to a child with an autism spectrum disorder. *Journal of Positive Behavior Interventions*, 5, 22–29.
- Lee, A., Browder, D., Hawley, K., Flowers, C., & Wakeman, S. (2016). Teaching writing in response to text to students with developmental disabilities who participate in alternate assessments. *Education and Training in Autism and Developmental Disabilities*, 51(3), 238–251.
- Mayes, S., & Calhoun, S. L. (2008). WISC-IV and WIAT-II profiles in children with high functioning autism. *Journal of Autism and Developmental Disorders*, 38(3), 428–439. <https://doi.org/10.1007/s10803-007-0410-4>.
- National Professional Development Center on Autism Spectrum Disorder. (2014). *Evidence-based practices*. Retrieved 4 Aug 2019, from <https://autismpdc.fpg.unc.edu/national-professional-development-center-autism-spectrum-disorder>
- Pennington, R. C., & Delano, M. E. (2012). Writing instruction for students with autism spectrum disorders: A review of literature. *Focus on Autism and Other Developmental Disabilities*, 27(3), 158–167. <https://doi.org/10.1177/1088357612451318>.
- Pennington, R. C., Ault, M. J., Schuster, J. W., & Sanders, A. (2010). Using simultaneous prompting and computer-assisted instruction to teach story writing to students with autism. *Assistive Technology Outcomes and Benefits*, 7(1), 24–38.
- Pennington, R. C., Collins, B. C., Stenheff, D. M., Turner, K., & Gunselman, K. (2014). Using simultaneous prompting and computer-assisted instruction to teach narrative writing skills to students with autism. *Education and Training in Autism and Developmental Disabilities*, 49, 396–414. Stable URL: <http://www.jstor.org/stable/23881260>
- Pennington, R., Flick, A., & Smith-Wehr, K. (2018). The use of response prompting and frames for teaching sentence writing to students with moderate intellectual disability. *Focus on Autism and Other Developmental Disabilities*, 33(3), 142–149. <https://doi.org/10.1177/1088357616673568>.
- Reichow, B., Volkmar, F. R., & Cicchetti, D. V. (2008). Development of the evaluative method for evaluating and determining evidence-based practices in autism. *Journal of Autism and Developmental Disorders*, 38(7), 1311–1399. <https://doi.org/10.1007/s10803-007-0517-7>.
- Santangelo, T., Harris, K. R., & Graham, S. (2008). Using self-regulated strategy development to support students who have “trubol giting thangs into werds”. *Remedial and Special Education*, 29(2), 78–89.
- Schlosser, R. W., & Blischak, D. M. (2004). Effects of speech and print feedback on spelling by children with autism. *Journal of Speech, Language, and Hearing Research*, 47(4), 848–862. [https://doi.org/10.1044/1092-4388\(2004/063\)](https://doi.org/10.1044/1092-4388(2004/063)).
- Schlosser, R. W., Blischak, D. M., Belfiore, P. J., Bartley, C., & Barnett, N. (1998). Effects of synthetic speech output and orthographic feedback on spelling in a student with autism: A preliminary study. *Journal of Autism and Developmental Disorders*, 28(4), 309–319.
- Stromer, R., Mackay, H. A., Howell, S. R., & McVay, A. A. (1996). Teaching computer-based spelling to

individuals with developmental and hearing disabilities: Transfer of stimulus control to writing tasks. *Journal of Applied Behavior Analysis*, 29, 25–42.

Wong, C., Odom, S., Hume, K., Cox, A., Fettig, A., Kucharczyk, S., Brock, M., Plavnick, J., Fleury, V., & Schultz, T. (2015). Evidence-based practices for children, youth, and young adults with autism spectrum disorder: A comprehensive review. *Journal of Autism and Developmental Disorders*, 45(7), 1951–1966. <https://doi.org/10.1007/s10803-014-2351-z>.

Written Communication

► Written Language

Written Language

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Synonyms

Written communication

Definition

Written language is the written form of communication which includes both reading and writing. Although written language may at first be considered to simply be oral language in its written form, the two are quite different in that oral language rules are innate whereas written language is acquired through explicit education.

Written language, whether reading or writing, requires basic language abilities. These include phonological processing (understanding that words are made of discrete sounds, then associating letters with these sounds, i.e., decoding), vocabulary, and syntax (grammar). Skilled reading and writing further require an awareness of what is being read or written in order to construct meaning. Given characteristic and varying

difficulties in language in individuals with autism spectrum disorders (ASD), the bidirectional relationship between oral and written language poses challenges to written language development.

Further, although reading and writing are based upon the same basic language skills, they are differing forms of written communication with different levels of cognitive demand; these demands present additional challenges to those with ASD learning to read and write. Specifically, writing is more complex than reading because successful written expression requires adequate executive processes (Hooper 2009), that is, processes for planning, organizing, translating (thoughts into words), revising, and editing. Furthermore, writing puts greater demands on internal working memory than does reading does (Berninger and Winn 2006). Additionally, written expression requires handwriting skills, “pen to paper.”

See Also

- Dysgraphia
- Reading
- Writing Disorders

References and Reading

- Berninger, V. W., & Winn, W. D. (2006). Implications of advancements in brain research and technology for writing development, writing instruction, and educational evolution. In C. MacArthur, S. Graham, & J. Fitzgerald (Eds.), *The writing handbook* (pp. 96–114). New York: The Guilford Press.
- Brown, H. M., & Klein, P. D. (2011). Writing, Asperger Syndrome and theory of mind. *Journal of Autism and Developmental Disorders*, 1(1/5), 1–11. <https://doi.org/10.1007/s10803-010-1168-7>.
- Hooper, S. R. (2009). *Biological processes underlying written language acquisition*. *Encyclopedia of Language and Literacy Development* (pp. 1–9). London: Canadian Language and Literacy Research Network. Retrieved July 26, 2011, from <http://www.literacyencyclopedia.ca/pdfs/topic.php?topId=288>.
- Kushki, A., Chau, T., & Anagnostou, E. (2011). Handwriting difficulties in children with autism spectrum disorders: A scoping review. *Journal of Autism and Developmental Disorders*, 1, 2(25), 1–2(25), 11. <https://doi.org/10.1007/s10803-011-1206-0>.

Nation, K., Clarke, P., Wright, B., & Williams, C. (2006). Patterns of reading ability in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 36(7), 911–919. <https://doi.org/10.1007/s10803-006-0130-1>.

Wrong Decision

► False Positive

X

X and Y Chromosomes

► [Sex Chromosomes](#)

Xanax

► [Alprazolam](#)

Xanax (Alprazolam)

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Synonyms

[Alprazolam](#); [Benzodiazepine](#)

Definition

Xanax, the brand name of Alprazolam, is a member of the benzodiazepine (BZD) family, a category of drugs commonly prescribed for conditions such as insomnia, anxiety, agitation, muscle spasticity, and epilepsy. Xanax is classified as a high-potency benzodiazepine and is known to have a short-lasting anxiolytic effect, meaning the drug reduces anxiety and has a half-life of

approximately 6–27 h half life; it is consequently often prescribed for panic disorder and anxiety-related disorders, such as generalized anxiety disorder (GAD) and obsessive compulsive disorder (OCD). To treat anxiety, Xanax is most commonly taken by mouth in 0.25–0.5 mg tablets up to three times per day. For panic disorder, it is recommended the drug be taken at a maximum of 6–10 mg per day.

Concerning the mechanism of action, Xanax binds to GABA-A receptors, ion channels that have an affinity towards chloride. When the compound binds at two transected subunits of the complex, it in turn alters the shape of the complex's chloride channels and allows GABA to bind. GABA, a common inhibitory neurotransmitter, is known to interfere with brain activity and the connectivity of brain structures, therapeutically resulting in sedation and relaxation of the body. Furthermore, when Xanax binds to subunits of the GABA-A receptor complex, specific isoforms of those subunits regulate the effects of sedation, antianxiety, hypnosis, and amnesia.

Because of its fast-acting properties, Xanax can provide more immediate relief for individuals with anxiety, compared the use of SSRIs or buspirone. Yet the administration of Xanax may also result in impairments in executive functioning, affecting the patient's alertness, diligence, and decision-making. Because of the drug's high potency and high lipid solubility, the possibility of memory deficits, such as anterograde amnesia and impairments in delayed recall, are risk factors.

Rebound anxiety is also a possible side effect when withdrawing from Xanax, which results in a worse return of the original symptoms. Rebound anxiety is more often to occur with a benzodiazepine like Xanax, because of its shorter elimination half-life compared to other BZDs, such as diazepam. In addition to rebound, the fast-acting, short-lived properties of Xanax also expose the patient to risks of withdrawal symptoms and dependence.

In terms of Xanax and its relation to autism, anxiety-related disorders are a common comorbidity of autism spectrum disorders (ASD). Multiple classes of drugs, such as antidepressants, stimulants, anticonvulsants, and antipsychotics are used by youth with ASD to treat comorbid or related diagnoses. Compared to the above classes, benzodiazepines appear to be less commonly used within the community. It is important to note that while benzodiazepines are sometimes taken to directly treat a related diagnosis such as anxiety disorder or epilepsy, they are not prescribed to treat the core symptoms of ASD.

Additionally, there is some recent evidence that suggests that the GABA system may be involved with the pathophysiology of ASD. Several mouse models have shown a reduction in GABAergic interneurons, thus maintaining an imbalance of excitation and inhibition in the brain. This imbalance is thought to be associated with the characteristics of the autism phenotype.

See Also

- [Anxiolytic Drugs](#)
- [Lorazepam](#)

References and Reading

- Chouinard, G. (2004). Issues in the clinical use of benzodiazepines: Potency, withdrawal, and rebound. *The Journal of Clinical Psychiatry*, 65, 7–12.
- Griffin, C. E., Kaye, A. M., Bueno, F. R., & Kaye, A. D. (2013). Benzodiazepine pharmacology and central nervous system-mediated effects. *The Ochsner Journal*, 13, 214–223.
- Oswald, D. P., & Sonenklar, N. A. (2007). Medication use among children with autism spectrum disorders. *Journal of Child and Adolescent Psychopharmacology*, 17, 348–355.
- Rudolph, U., & Möhler, H. (2014). GABAA receptor subtypes: Therapeutic potential in down syndrome, affective disorders, schizophrenia, and autism. *Annual Review of Pharmacology and Toxicology*, 54, 483–507.
- Verster, J. C., Volkerts, E. R., & Verbaten, M. N. (2002). Effects of alprazolam on driving ability, memory functioning and psychomotor performance: A randomized, placebo-controlled study. *Neuropsychopharmacology*, 27, 260–269.

Xanax XR

- [Alprazolam](#)

Xeristar

- [Duloxetine: Definition](#)

X-Linked Traits

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Definition

The traits of an organism are its characteristics. These can be normal characteristics such as stature and eye color or abnormal or disease characteristics, such as blindness or the symptoms of autism. Traits are influenced by two main factors – the environment and an organism's genetic material, the biological instructions for the development and functioning of a living organism.

These instructions are contained in a molecule called deoxyribonucleic acid (DNA). The instructions are spelled out in a sequence or code of four chemical units called nucleobases (or bases). This molecule is contained within nearly every cell of an organism, cells being the building blocks of organisms. Certain segments of the DNA

molecule called genes contain the code for creating the components of cells, most importantly, molecules called proteins.

DNA is passed from one generation to the next. In humans, the DNA is not comprised of a single long molecule but rather is divided up into a set of smaller pieces that correspond to structures called chromosomes, which contain both a single long DNA molecule and proteins. By definition, cells of males contain a chromosome X and a chromosome Y while cells of females contain two of chromosome X. The X and Y chromosomes are referred to as the sex chromosomes. Each parent passes one of his or her sex chromosomes to each offspring. By definition, male offspring can inherit their X chromosome only from their mothers (as the father's Y chromosome is necessary for the creation of male offspring), while female offspring inherit one X chromosome from each parent.

Sex chromosomes stand in contrast to the other human chromosomes that are called autosomes. Autosomes are paired in an individual so that each carries two of chromosome 1, for example. One member of the pair is inherited from the individual's father and the other from the mother.

Traits that result from the sequence of DNA on the X chromosome are called X-linked traits. Rare variations in the DNA sequence are sometimes called mutations. When mutations in the genes on the X chromosome lead to disease, the disease itself is referred to as an X-linked disease. Color blindness is an example of such a disease. Fragile X syndrome, an intellectual disability syndrome that is associated with an increased risk for autism, is another example of an X-linked disease.

In autosomes, mutations in a gene on a single chromosome of a pair can lead to a disease or contribute to disease risk. In the most dramatic case in which a single variation or mutation present on only one of the two chromosomes in a pair leads to a phenotype, this is called a dominant mutation. However, in some cases, a mutation in a gene on one chromosome of a pair will not be sufficient to result in disease because a non-mutated copy of the gene that exists on the other chromosome compensates. Only when mutations in the gene occur on both chromosomes in a pair will the disease occur. These are called recessive

mutations. In autosomes, these rules by and large do not vary on the basis of the sex of an individual.

X-linked mutations can be dominant and recessive as well. However, since not all the genes on chromosome X are represented on chromosome Y, the inheritance pattern of X-linked dominant and recessive mutations differs from that of analogous mutations on autosomes.

In males, an X-linked recessive disease mutation will result in disease because there is not an additional nonmutated X chromosome to compensate. In females, however, such a mutation would only result in disease if the other chromosome X also had a disease mutation in the gene in question. If the gene on only one of a female's X chromosomes was mutated, then the non-mutated X chromosome would compensate, and the individual female would not be affected by the disease. Such females would be called "carriers" of the disease since they carry a disease gene but do not manifest the disease.

In X-linked recessive disorders, a common pattern of inheritance within families, therefore, is as follows. The disease is seen to be passed from apparently healthy mothers who are carriers to their sons but not to their daughters.

In X-linked dominant diseases, a mutation on only one copy of chromosome X is sufficient to result in disease. Thus, even females with one mutated X chromosome and one nonmutated one will have the disease.

The inheritance pattern commonly seen in X-linked dominant disorders depends on which parent carries the mutated chromosome X. If of the parents, only the father has the mutated chromosome, he will pass the disease to all of his daughters and none of his sons since he passes only chromosome Y to his sons. If of the parents, only the mother has the mutated chromosome, she will pass the disease in equal proportion to daughters and sons.

It is important to note that this nomenclature evolved in reference to so-called Mendelian inheritance. These are cases in which there is a highly reliable relationship between the presence of a dominant, recessive, or X-linked mutation and the presence of a disorder or disease. Autism is thought, in contrast, to be a complex genetic disorder in which many different variations do not

cause but rather contribute to the risk for the emergence of the phenotype. These variations, while not as predictive as those found in Mendelian disorders, may also show dominant, recessive, or X-linked effects. As noted, there are also examples of genetic syndromes, such as Fragile X, that are associated with an increased rate of ASD. Finally, there have also been rare cases of genes contributing to “nonsyndromic” autism showing Mendelian inheritance, including X-linked inheritance.

See Also

- [DNA](#)
- [Genetics](#)
- [Recessive Genes](#)
- [Variable Expressivity of Genes](#)

References and Readings

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, 9(5), 341.
- Alberts, B., Bray, D., Lewis, L., Raff, M., Roberts, K., & Watson, J. D. (2002). *The cell*. New York: Garland Science.
- Reeve, E. C. R., & Black, I. (2001). *Encyclopedia of genetics*. New York: Routledge.
- Strachan, T., & Read, A. P. (2004). *Human molecular genetics*. New York: Garland Press.
- Watson, J. D., & Crick, F. H. C. (1953). Molecular structure of nucleic acids. *Nature*, 171(4356), 737–738.

X-Ray Computed Tomography

- [Computed Tomography](#)

Yale Global Tic Severity Scale

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Synonyms

YGTSS

Definition

Yale Global Tic Severity Scale: The YGTSS is a commonly used measure to document the severity of motor and phonic tics. It is performed by a clinician interview. The clinician reviews a list of possible tics that may have occurred over the past week, first motor and then vocal tics. Once the tics that have occurred over the past week are established, the interviewer goes on to ask about the frequency of the tics, the intensity or forcefulness of the tics, the complexity of the tics, and the extent to which the tics are directly interfering in the person's daily life. These dimensions are rated for both motor and phonic tics. The combined total is often used as a measure for overall tic

severity. The YGTSS also includes an overall impairment scale rated from 0 to 50. A score of 0 would indicate that the presence of Tourette syndrome has no negative impact on a person's life. By contrast, a score of 50 would indicate marked interference and disability associated with the presence of Tourette syndrome. The overall impairment caused by Tourette syndrome may not be completely driven by the tic severity. Some individuals with mild tics may be deeply troubled and embarrassed by the tics. By contrast, some individuals with marked tics seem to proceed with life without too much difficulty.

See Also

- [Tics](#)
- [Tourette's Syndrome](#)

References and Reading

- Leckman, J. F., Riddle, M. A., Hardin, M. T., Ort, S. I., Swartz, K. L., Stevenson, J., & Cohen, D. J. (1989). The Yale Global Tic Severity Scale: Initial testing of a clinician-rated scale of tic severity. *Journal of the American Academy of Child Psychiatry*, 28(4), 566–573.
- Piacentini, J., Woods, D. W., Scahill, L., Wilhelm, S., Peterson, A., Chang, S., et al. (2010). Behavior therapy for children with Tourette disorder: A randomized controlled trial. *Journal of the American Medical Association*, 303(19), 1929–1937.

Yale In Vivo Pragmatic Protocol

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Synonyms

YiPP

Description

The Yale in vivo Pragmatic Protocol is an assessment tool that measures pragmatic language through a semi-structured conversational task in verbal children aged 9–17. It contains a series of predetermined probes to collect information on a variety of conversational speech acts. Within this 30–40-min conversation, the examiner inserts 23 pragmatic probes within five conversational domains (discourse management, communicative function, conversational repair, presupposition, register variation). If the child produces a pragmatic language behavior in response to the probe, the conversation continues and the next probe is administered. If the child does not respond to the probe, the examiner systematically provides a series of prompts to determine whether or not these prompts are helpful in the production of the pragmatic behavior.

Historical Background

The YiPP was developed by Dr. Rhea Paul and piloted at the Yale Child Study Center.

Psychometric Data

This measure is not standardized but yields qualitative information on pragmatic language skills. Norm-reference to a group of typically developing age-mates is provided.

Clinical Uses

This assessment provides a naturalistic means to assess pragmatic language difficulties commonly seen in children with autism spectrum disorders. Due to the probes used in this assessment, it requires the child to speak in full sentences. Thus, this evaluation is best suited for higher functioning children on the autism spectrum. It yields qualitative information in five domains of pragmatic language. The information derived from this tool can be useful for intervention programming.

See Also

- ▶ [Conversational Manner](#)
- ▶ [Discourse Management](#)
- ▶ [Pragmatic Communication](#)

References and Readings

- Paul, R. (2005). Assessing communication in autism spectrum disorders. In D. Cohen & F. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 799–816). New York: Wiley.
- Schoen, E., & Paul, R. (2009). *Assessing pragmatic language in high-functioning autism*. Poster presented at the symposium on research in child language disorders, Madison.
- Simmons, E. S., Paul, R., & Volkmar, F. (2014). Assessing pragmatic language in autism spectrum disorder: The Yale in vivo pragmatic protocol. *Journal of Speech, Language, and Hearing Research*, 57, 2162–2173.

Yeast Infection

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Definition

Various types of yeast can cause infection in different parts of the body. The most common cause

of yeast infection is *Candida albicans*. It can be oral thrush or with vaginitis. Males can also exhibit genital infection. At times, yeast infection can be systemic and potentially very serious (usually this occurs when the individual has some other condition that compromises the immune system, e.g., AIDS). On the skin surface or oral mucosa, yeast infection causes inflammation. Yeast is diagnosed based on microscopic examination and search for characteristic yeast organisms. A range of antifungal drugs is used to treat yeast infection. These can be topical or systemic.

In addition to the medically accepted yeast infections (noted above), others have proposed that “subclinical” yeast infection can cause a wide range of problems and can be treated by a special diet. The notion behind this diet is that yeast infection, sometimes acquired at the time of vaginal birth, causes autism. The diet consists of having children avoid food that contains yeast or fermented foods, perhaps combined with medications used in treatment of yeast infections. Although dramatic claims have been made, the treatment is unproven. (Smith et al. 2014).

Clearly systematic data on this intervention cannot be regarded as having a solid evidence base. Despite this many parents wish to pursue this (or other complementary/alternative) treatment options. Typically dietary interventions focused on yeast advise parents to avoid foods for the child that contain yeast (baked goods) or which are fermented (soy sauce) or aged (cheeses). Sugar is also to be avoided. Occasionally parents will wish to pursue long-term anti-fungal treatments – again with no clear evidence of yeast infection. As noted above, although dramatic claims have been made for these treatments, substantive scientific data are lacking, and the hypothesis of “yeast overgrowth” in children with autism has no scientific basis. Helping parents make informed treatment decisions is an important aspect of medical care in autism.

References and Reading

- Committee on Children with Disabilities. (2001). American Academy of Pediatrics: Counseling families who choose complementary and alternative medicine for their child with chronic illness or disability. *Pediatrics*, 107(3), 598–601.
- Hyman, S. L., & Levy, S. E. (2005). Introduction: Novel therapies in developmental disabilities, hope, reason, and evidence. *Mental Retardation and Developmental Disabilities Research Reviews*, 11(2), 107–109.
- Jacobson, J. W., Foxx, R. M., & Mulick, J. A. (Eds.). (2005). *Controversial therapies for developmental disabilities: Fad, fashion and science in professional practice*. Mahwah: Lawrence Erlbaum Associates.
- Levy, S. E., Mandell, D. S., Merhar, S., Ittenbach, R. F., & Pinto-Martin, J. A. (2003). Use of complementary and alternative medicine among children recently diagnosed with autistic spectrum disorder. *Journal of Developmental and Behavioral Pediatrics*, 24(6), 418–423.
- Perrin, J. M., Coury, D. L., Hyman, S. L., Cole, L., Reynolds, A. M., & Clemons, T. (2012). Complementary and alternative medicine use in a large pediatric autism sample. *Pediatrics*, 130(Suppl 2), S77–S82.
- Smith, T., Oakes, L., & Selver, K. (2014). Alternative treatments. In F. R. Volkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (4th ed., pp. 1051–1069). Hoboken: Wiley.
- Volkmar, F., & Wiesner, L. (2009). *A practical guide to autism*. Hoboken: Wiley.

Yentreve

- [Duloxetine: Definition](#)

YGTSS

- [Yale Global Tic Severity Scale](#)

YiPP

- [Yale In Vivo Pragmatic Protocol](#)

Z

Z Scores

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Synonyms

[Standard score](#)

Definition

The Z score (or standard score) is a standardized way to convert raw scores into values expressed in terms of standard deviation units. It is calculated by taking a raw score and subtracting the mean score, then dividing the result by the standard deviation. (The mean score and standard deviation used in the calculation are specific to the population or test from which the raw score is obtained).

$$Z = (\text{raw score} - \text{meanscore}) / \text{standarddeviation}$$

A Z score of 1.0 indicates that the raw score was 1 standard deviation above the mean. A Z score of -1.0 indicates that the raw score was 1 standard deviation below the mean.

See Also

► [Standard Deviation \(SD\)](#)

References and Reading

Norman, G., & Streiner, D. (2008). *The normal distribution* (Biostatistics the bare essentials) (pp. 32–33). Shelton: People's Medical Publishing House.

Zactin

► [Prozac™ \(Fluoxetine\)](#)

Zaponex

► [Clozapine](#)

Zebrafish Models

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Definition

In recent years, large-scale human genetics studies have led to considerable advances in our

understanding of the biology of autism spectrum disorders (ASD). In particular, these studies have resulted in the identification of a growing list of ASD risk genes that are beginning to converge on common biological mechanisms (De Rubeis et al. 2014; Iossifov et al. 2014; Sanders et al. 2015). At the same time, scientists now face the challenge of leveraging these genetic findings to elucidate the neural circuit mechanisms underlying ASD and to identify novel pharmacotherapies that selectively target these mechanisms. Here, scientists have utilized model systems to advance from risk gene discovery to the elucidation of basic neurobiological mechanisms. These systems include mouse “knockout” models, in which the function of a particular risk gene is disrupted, as well as human induced pluripotent stem cells (iPSCs), which are generated from the cells of an affected individual carrying a mutation in a specific risk gene. More recently, there is growing interest in the use of zebrafish as a system for the functional analysis of risk genes in ASD. There are several unique features of the zebrafish that make it an optimal system for this purpose. First, zebrafish embryos undergo rapid external development and are optically transparent, allowing for direct visualization of basic processes of vertebrate brain development at early stages. Second, zebrafish have large progenies and their larvae are small and highly tractable in the laboratory. These features facilitate their use in high-throughput pharmacological screens, which are not possible in rodents, given their greater size and complexity. Third, zebrafish provide an in vivo system for investigating the effect of risk gene disruption on the neural circuitry underlying simple, quantifiable behaviors, which is a notable limitation of in vitro methods, including human iPSCs. Finally, recent advances in technologies that allow scientists to target genes of interest in zebrafish, together with the inherent ease of genetic manipulation in this system, have contributed to its growing popularity as a tool for the functional analysis of ASD risk genes. As the list of genes that are strongly associated with ASD continues to grow, zebrafish are likely to emerge as a key player in the identification of convergent

biological pathways involving multiple genes and the discovery of novel pharmacological candidates for further investigation in ASD.

Historical Background

Until recently, the major limitation of the use of zebrafish for the functional analysis of risk genes in ASD and other neurodevelopmental disorders was the lack of efficient methods for selectively disrupting a gene of interest. While mouse “knockouts” were generated by isolating stem cells, such an approach was not feasible in zebrafish due to their rapid embryonic development. Indeed, it was only within the past 10 years that the technology for generating zebrafish lacking the function of a specific risk gene became widely available. For this reason, early studies of zebrafish primarily relied on *forward genetics* approaches (Granato and Nusslein-Volhard 1996), in which scientists search for an unidentified gene that when disrupted leads to an observed structural or behavioral phenotype. In particular, a large-scale screen in the mid-1990s was a tour de force that established zebrafish as a valuable system for studying basic mechanisms in genetics and developmental biology. This series of studies, in which zebrafish carrying randomly induced mutations were assessed for a range of morphological and behavioral phenotypes, led to the discovery of hundreds of genes involved in fundamental processes of vertebrate development, including axon pathfinding and locomotion (Baier et al. 1996; Granato et al. 1996; Karlstrom et al. 1996). However, the ability to target a gene associated with a human disorder in zebrafish remained limited. For example, early *reverse genetics* approaches in zebrafish, such as TILLING (Targeted Induced Local Lesions in Genomes), required screening thousands of zebrafish carrying chemically induced mutations to identify a deleterious mutation in a gene of interest (Moens et al. 2008). Another approach to study gene function in zebrafish that is relatively simple to perform in the laboratory is the use of *morpholinos*, which are sequences of nucleotides

that are introduced into zebrafish embryos and transiently reduce the expression of a gene of interest during early developmental stages. While this “knockdown” approach has contributed insights into the role of genes in basic developmental processes, drawbacks include its potential for inducing off-target effects and non-specific phenotypes (Kok et al. 2015). For this reason, scientists have argued that it is important to examine the developmental effects of gene disruption in zebrafish carrying deleterious, heritable mutations in a gene of interest in conjunction with phenotypes resulting from morpholino-induced gene “knockdown” (Kok et al. 2015).

In the late 2000s, the introduction of targeted nuclease technology transformed reverse genetics studies in zebrafish, enabling researchers to selectively induce heritable mutations in a gene of interest (Doyon et al. 2008; Meng et al. 2008). This technology expanded the range of experimental possibilities in zebrafish, allowing researchers to capitalize fully on the advantages of this system for the functional analysis of genes associated with human disorders, including ASD and other neurodevelopmental disorders. Zinc finger nucleases, which are chimeric fusion proteins designed to bind to a gene of interest and disrupt the gene at the target site, were the first available tools. However, this approach was cost-prohibitive and, in some cases, characterized by low efficiencies. More recently, the advent of *CRISPR (clustered regularly interspaced short palindromic repeats)/Cas9* technology, which hijacks an adaptive immune mechanism used by bacteria for protection against viruses to allow scientists to target genes of interest (Jinek et al. 2012), has revolutionized gene editing in zebrafish as well as other model systems. Compared to earlier gene targeting methods in zebrafish, CRISPR/Cas9 offers superior flexibility and efficiency (Hwang et al. 2013). Given these advantages, along with their reasonable cost, this method has allowed scientists to harness the full potential of zebrafish for the functional analysis of ASD risk genes, which has the potential in the next several years to lead to important insights into the biology of ASD.

Current Knowledge

Despite the obvious evolutionary distance between zebrafish and humans, it is important to observe that there is a reasonable degree of conservation between the two systems at the genetic, molecular, and pharmacological levels. First, zebrafish are vertebrates and share the same major brain subdivisions as humans, including the forebrain, midbrain, hindbrain, and spinal cord (Guo 2009). Neurotransmitter systems and neural cell types are also largely conserved. Second, there is evidence for conservation of pharmacological pathways in zebrafish and other vertebrates, based on similarities in their behavioral responses to psychoactive agents (Guo 2009; Rihel et al. 2010). Third, there is conservation at the genomic level, such that over 80% of genes associated with human disorders have an orthologous gene in zebrafish (McCammon and Sive 2015). Given the advantages of the zebrafish system, as discussed above, zebrafish represent a reasonable “balance” between experimental manipulability on the one hand and conservation on the other (McCammon and Sive 2015). Taken together, the relative conservation of zebrafish at the level of central nervous system structure, genetics, and pharmacological pathways provides support for its use as a translational tool for investigating the function of genes that are strongly associated with ASD in basic neurodevelopmental processes (Ijaz and Hoffman 2016).

At the same time, there are limits to translating findings from zebrafish or any animal system to human disorders. First, there are notable anatomical differences between zebrafish and human brains, including the lack of a neocortex, which is present in mice and clearly limits the translation of findings from zebrafish to humans (Guo 2009; McCammon and Sive 2015). Second, there is less conservation between genes in zebrafish compared to mammalian systems, which may complicate the ability to identify the orthologs of human genes in zebrafish (McCammon and Sive 2015). Third, it is impossible for any animal system to recapitulate the complex range of clinical features in ASD or any neuropsychiatric disorder. For this

reason, it is important to emphasize that the goal of studying zebrafish is to leverage the unique advantages of this relatively simple system to analyze the function of ASD risk genes in basic neurodevelopmental processes.

A number of studies have capitalized on the advantages of zebrafish to investigate the role of ASD risk genes in nervous system development, with many of these studies employing a morpholino-based approach, as described above, to reduce the expression of genes of interest. For example, Kozol et al. (2015) knocked down two ASD-associated genes, *SYNGAP1* (*Synaptic Ras GTPase Activating Protein 1*) and *SHANK3* (*SH3 and Multiple Ankyrin Repeat Domains 3*), in zebrafish, and found that reduced expression was associated with abnormal swimming and escape behaviors, as well as developmental delay and microcephaly. In addition, Blaker-Lee et al. (2012) studied the effect in zebrafish embryos of reducing the expression of 22 genes found in the region of human 16p11.2. Copy number variants in this region are strongly associated with ASD and other neuropsychiatric disorders, including schizophrenia and intellectual disability, yet the contribution of individual genes in the region to ASD is poorly understood. By performing a series of morphological and behavioral assays, the authors found that the reduced expression of many of these genes led to brain and behavioral abnormalities, such as decreased brain ventricle size, abnormal midbrain structure, or alterations in spontaneous movement and touch response (Blaker-Lee et al. 2012). Another zebrafish study found that reduced expression and overexpression of one of the genes in this region led to macrocephaly and microcephaly, respectively, suggesting dose-dependent effects (Golzio et al. 2012). At the same time, it is important to observe that morpholinos are limited by their potential for inducing off-target effects, such that there is growing emphasis on confirming phenotypes resulting from gene knockdown in mutants, where gene function is permanently disrupted (Kok et al. 2015). With the advent of CRISPR/Cas9 technology, this goal is becoming increasingly feasible.

Recent studies have also capitalized on the large progenies of zebrafish and the small size of their embryos and larvae to develop quantitative simple behavioral assays for conducting high-throughput pharmacological screens. For example, Rihel et al. (2010) tracked the locomotor activity of zebrafish larvae automatically on a light-dark cycle in response to hundreds of psychoactive compounds to analyze the effects of these drugs on their rest-wake cycle behaviors. Intriguingly, this study demonstrated that psychoactive compounds could be correctly classified by their mechanism of action based solely on the readout of their behavioral effects in zebrafish larvae. In addition, Kokel et al. (2010) screened thousands of compounds for their effects on the photomotor response, a characteristic motor response exhibited by zebrafish embryos following exposure to a light stimulus, and found that their behavioral responses could be used to correctly categorize novel psychotropic compounds. These studies reveal the potential of large-scale screens of simple behavioral responses in zebrafish to uncover pharmacological mechanisms. In a recent study, Hoffman et al. (2016) utilized this automated behavioral profiling approach to analyze the locomotor activity of zebrafish carrying deleterious mutations in the ASD risk gene, *CNTNAP2* (*Contactin Associated Protein-like 2*). This study found that these zebrafish mutants displayed deficits in inhibitory neurons, had increased sensitivity to drug-induced seizures, and were hyperactive at night. By comparing the abnormal behavioral profile of mutants to the known effects of over 500 compounds in control fish (Rihel et al. 2010), this study found that estrogenic compounds, including the plant-derived estrogen, biochanin A, were able to selectively rescue the abnormal behavioral phenotype in mutants, identifying a novel pharmacological pathway not previously associated with *CNTNAP2* (Hoffman et al. 2016). In another study, Baraban et al. (2013) conducted a large-scale drug screen to identify compounds that suppress spontaneous seizure-like behaviors in zebrafish carrying a loss-of-function mutation in a gene that is closely related to *SCN1A* (*Sodium Voltage-Gated Channel Alpha Subunit 1*), which is associated with a rare

syndrome of intractable epilepsy called Dravet syndrome. Of the 320 compounds tested, the authors identified a single compound, clemizole, which could reverse abnormal seizure-associated behaviors and electrophysiological abnormalities in mutants (Baraban et al. 2013). These studies highlight the potential of the zebrafish system as a *first-pass screening approach* to identify novel pharmacological candidates with relevance to ASD and other neurodevelopmental disorders for further investigation in mammalian systems.

Future Directions

There are a number of emerging technologies that are likely in the next several years to expand the repertoire of questions that scientists can address using the zebrafish system. First, genetically encoded fluorescent calcium indicators are allowing scientists to image the activity of individual cells throughout the brain of a live larval zebrafish at high resolution, which is possible given their transparent heads. These live imaging assays are increasingly being used to study the neural circuitry underlying simple behavioral tasks, such as the optomotor response and prey capture (Filosa et al. 2016; Muto et al. 2013; Portugues et al. 2015). By visualizing brain activity in zebrafish lacking the function of ASD risk genes, scientists can begin to explore how the disruption of these genes leads to abnormalities in specific neural circuits. Second, the advent of CRISPR/Cas9 gene-editing technologies is facilitating the targeting of multiple ASD risk genes in zebrafish, providing scientists with the opportunity to leverage this approach to investigate the effect of multiple ASD risk genes on brain development simultaneously. Third, high-throughput, automated pharmacological screens based on simple larval behaviors, as described above, have the potential to serve as an important first-pass screening tool to uncover neurochemical pathways that are disrupted due to loss of ASD risk genes and novel pharmacological candidates for further evaluation in mammalian systems. Therefore, the zebrafish system holds considerable translational promise, in conjunction with mouse and cell

culture models, to advance our understanding of neurobiological mechanisms in ASD. Taken together, given the unique features of this system, the zebrafish has the potential to emerge as a key contributor to the functional analysis of ASD risk genes and the elucidation of convergent biological and pharmacological mechanisms in ASD.

See Also

- ▶ [Animal Models](#)
- ▶ [Candidate Genes in Autism](#)
- ▶ [Genetics](#)
- ▶ [Neuroscience](#)
- ▶ [Recessive Genes](#)

References and Reading

- Baier, H., Klostermann, S., Trowe, T., Karlstrom, R. O., Nusslein-Volhard, C., & Bonhoeffer, F. (1996). Genetic dissection of the retinotectal projection. *Development*, 123, 415–425.
- Baraban, S. C., Dinday, M. T., & Hortopan, G. A. (2013). Drug screening in Scn1a zebrafish mutant identifies clemizole as a potential Dravet syndrome treatment. *Nature Communications*, 4, 2410.
- Blaker-Lee, A., Gupta, S., McCammon, J. M., De Rienzo, G., & Sive, H. (2012). Zebrafish homologs of genes within 16p11.2, a genomic region associated with brain disorders, are active during brain development, and include two deletion dosage sensor genes. *Disease Models & Mechanisms*, 5, 834–851.
- De Rubeis, S., He, X., Goldberg, A. P., Poultney, C. S., Samocha, K., Cicek, A. E., Kou, Y., Liu, L., Fromer, M., Walker, S., et al. (2014). Synaptic, transcriptional and chromatin genes disrupted in autism. *Nature*, 515, 209–215.
- Doyon, Y., McCammon, J. M., Miller, J. C., Faraji, F., Ngo, C., Katibah, G. E., Amora, R., Hocking, T. D., Zhang, L., Rebar, E. J., et al. (2008). Heritable targeted gene disruption in zebrafish using designed zinc-finger nucleases. *Nature Biotechnology*, 26, 702–708.
- Filosa, A., Barker, A. J., Dal Maschio, M., & Baier, H. (2016). Feeding state modulates behavioral choice and processing of Prey Stimuli in the Zebrafish Tectum. *Neuron*, 90, 596–608.
- Golzio, C., Willer, J., Talkowski, M. E., Oh, E. C., Taniguchi, Y., Jacquemont, S., Reymond, A., Sun, M., Sawa, A., Gusella, J. F., et al. (2012). KCTD13 is a major driver of mirrored neuroanatomical phenotypes of the 16p11.2 copy number variant. *Nature*, 485, 363–367.

- Granato, M., & Nusslein-Volhard, C. (1996). Fishing for genes controlling development. *Current Opinion in Genetics & Development*, 6, 461–468.
- Granato, M., van Eeden, F. J., Schach, U., Trowe, T., Brand, M., Furutani-Seiki, M., Haffter, P., Hammerschmidt, M., Heisenberg, C. P., Jiang, Y. J., et al. (1996). Genes controlling and mediating locomotion behavior of the zebrafish embryo and larva. *Development*, 123, 399–413.
- Guo, S. (2009). Using zebrafish to assess the impact of drugs on neural development and function. *Expert Opinion on Drug Discovery*, 4, 715–726.
- Hoffman, E. J., Turner, K. J., Fernandez, J. M., Cifuentes, D., Ghosh, M., Ijaz, S., Jain, R. A., Kubo, F., Bill, B. R., Baier, H., et al. (2016). Estrogens suppress a behavioral phenotype in Zebrafish mutants of the autism risk gene, CNTNAP2. *Neuron*, 89, 725–733.
- Hwang, W. Y., Fu, Y., Reyon, D., Maeder, M. L., Tsai, S. Q., Sander, J. D., Peterson, R. T., Yeh, J. R., & Joung, J. K. (2013). Efficient genome editing in zebrafish using a CRISPR-Cas system. *Nature Biotechnology*, 31, 227–229.
- Ijaz, S., & Hoffman, E. J. (2016). Zebrafish: A translational model system for studying neuropsychiatric disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 55, 746–748.
- Iossifov, I., O’Roak, B. J., Sanders, S. J., Ronemus, M., Krumm, N., Levy, D., Stessman, H. A., Witherspoon, K. T., Vives, L., Patterson, K. E., et al. (2014). The contribution of de novo coding mutations to autism spectrum disorder. *Nature*, 515, 216–221.
- Jinek, M., Chylinski, K., Fonfara, I., Hauer, M., Doudna, J. A., & Charpentier, E. (2012). A programmable dual-RNA-guided DNA endonuclease in adaptive bacterial immunity. *Science*, 337, 816–821.
- Karlstrom, R. O., Trowe, T., Klostermann, S., Baier, H., Brand, M., Crawford, A. D., Grunewald, B., Haffter, P., Hoffmann, H., Meyer, S. U., et al. (1996). Zebrafish mutations affecting retinotectal axon pathfinding. *Development*, 123, 427–438.
- Kok, F. O., Shin, M., Ni, C. W., Gupta, A., Grosse, A. S., van Impel, A., Kirchmaier, B. C., Peterson-Maduro, J., Kourkoulis, G., Male, I., et al. (2015). Reverse genetic screening reveals poor correlation between Morpholino-induced and mutant phenotypes in zebrafish. *Developmental Cell*, 32, 97–108.
- Kokel, D., Bryan, J., Laggner, C., White, R., Cheung, C. Y., Mateus, R., Healey, D., Kim, S., Werdich, A. A., Haggarty, S. J., et al. (2010). Rapid behavior-based identification of neuroactive small molecules in the zebrafish. *Nature Chemical Biology*, 6, 231–237.
- Kozol, R. A., Cukier, H. N., Zou, B., Mayo, V., De Rubeis, S., Cai, G., Griswold, A. J., Whitehead, P. L., Haines, J. L., Gilbert, J. R., et al. (2015). Two knockdown models of the autism genes SYNGAP1 and SHANK3 in zebrafish produce similar behavioral phenotypes associated with embryonic disruptions of brain morphogenesis. *Human Molecular Genetics*, 24, 4006–4023.
- McCammon, J. M., & Sive, H. (2015). Challenges in understanding psychiatric disorders and developing therapeutics: A role for zebrafish. *Disease Models & Mechanisms*, 8, 647–656.
- Meng, X., Noyes, M. B., Zhu, L. J., Lawson, N. D., & Wolfe, S. A. (2008). Targeted gene inactivation in zebrafish using engineered zinc-finger nucleases. *Nature Biotechnology*, 26, 695–701.
- Moens, C. B., Donn, T. M., Wolf-Saxon, E. R., & Ma, T. P. (2008). Reverse genetics in zebrafish by TILLING. *Briefings in Functional Genomics & Proteomics*, 7, 454–459.
- Muto, A., Ohkura, M., Abe, G., Nakai, J., & Kawakami, K. (2013). Real-time visualization of neuronal activity during perception. *Current Biology: CB*, 23, 307–311.
- Portugues, R., Haesemeyer, M., Blum, M. L., & Engert, F. (2015). Whole-field visual motion drives swimming in larval zebrafish via a stochastic process. *The Journal of Experimental Biology*, 218, 1433–1443.
- Rihel, J., Prober, D. A., Arvanites, A., Lam, K., Zimmerman, S., Jang, S., Haggarty, S. J., Kokel, D., Rubin, L. L., Peterson, R. T., et al. (2010). Zebrafish behavioral profiling links drugs to biological targets and rest/wake regulation. *Science*, 327, 348–351.
- Sanders, S. J., He, X., Willsey, A. J., Ercan-Sencicek, A. G., Samocha, K. E., Cicek, A. E., Murtha, M. T., Bal, V. H., Bishop, S. L., Dong, S., et al. (2015). Insights into autism spectrum disorder genomic architecture and biology from 71 risk loci. *Neuron*, 87, 1215–1233.

Zeldox

► [Geodon \(Ziprasidone\)](#)

Zero Reject

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Definition

The term *zero reject* refers to the requirement that an individual with a disability recognized by the Individuals with Disabilities Education Act (IDEA) cannot be denied access to special education and necessary related services in the United States. This explicitly implies that a local education agency (LEA) must make an affirmative effort to determine eligibility for special education

services, develop a free and appropriate individualized educational plan, implement that plan in the least restrictive environment, and demonstrate that the plan is effectively addressing the educational needs of a student with a disability. The LEA does not have the right to refuse (reject) determination of need, or provision of services, under federal law.

See Also

- [Individuals with Disabilities Education Act \(IDEA\)](#)

References and Readings

- Mandlawitz, M. R. (2005). Educating children with autism: Current legal issues. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 1161–1173). Hoboken: Wiley.
- PL 105-17. (1997). *Individuals with Disabilities Education Act Amendments (IDEA)*

Ziprasidone

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Synonyms

[Geodon](#)

Definition

Ziprasidone is atypical antipsychotic medication that has been on the marketplace since early in the 2000s. It is distinguished from others of the class in that it appears to be less likely to cause weight

gain. There are a few studies that have examined the benefits of ziprasidone in children or adults with autism, so information on dose and effectiveness is incomplete. Although rare, a drawback of ziprasidone is that it has a potential for affecting electrical signals in the heart. Most clinicians do not consider it as a first-line treatment because of this concern.

See Also

- [Antipsychotics: Drugs](#)

References and Reading

- Duggal, H. S. (2007). Ziprasidone for maladaptive behavior and attention-deficit/hyperactivity disorder symptoms in autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, 17(2), 261–263.
- Martin, A., Scahill, L., & Christopher, K. (2010). *Pediatric psychopharmacology: Principles and practice* (2nd ed.). New York: Oxford.
- McDougale, C. J., & Posey, D. J. (2011). Assessment and treatment of autistic and other pervasive developmental disorders. In A. Martin, L. Scahill, & C. Kratochvil (Eds.), *Pediatric psychopharmacology: Principles and practice* (pp. 547–560). New York: Oxford University Press.
- McDougale, C. J., Kem, D. L., & Posey, D. J. (2002). Case series: use of ziprasidone for maladaptive symptoms in youths with autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 41(8), 921–927.

Zoloft

- [Sertraline](#)

Zolpidem

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Synonyms

[Ambien](#); [Gamma-aminobutyric agonist](#)

Definition

Despite its high prevalence, few pharmacological treatments outside antihistamines and melatonin exist for sleep disorders among pediatric populations (Chevreuil et al. 2010). Few clinical trials have examined whether benzodiazepines, such as Zolpidem, may benefit autistic spectrum disorder (ASD) children with sleep disorders (Chevreuil et al. 2010). A recent case report suggests Zolpidem was effective in reducing catatonic behavior in an adolescent with ASD (Zaw and Bates 1997). However, murine models suggest zolpidem exacerbates social and cognitive deficits observed in ASD children (Banerjee et al. 2012; Han et al. 2014). Further clinical trials are required to elucidate the mechanism, therapeutic use, and side effects of Zolpidem among ASD children with sleep disorders.

See Also

- Gabapentin
- Midazolam
- Zyprexa

References and Reading

- Banerjee, A., García-Oscos, F., Roychowdhury, S., Galindo, L. C., Hall, S., Kilgard, M. P., & Atzori, M. (2012). Impairment of cortical GABAergic synaptic transmission in an environmental rat model of autism. *The International Journal of Neuropsychopharmacology*, 16(06), 1309–1318. <https://doi.org/10.1017/s1461145712001216>.
- Chevreuil, C., Polard, E., Gicquel, G., Frémaux, T., & Bentué-Ferrer, D. (2010). Pharmacologic treatment of insomnia in children and adolescent psychiatric patients. *Thérapie*, 65(1), 1–12. <https://doi.org/10.2515/therapie/2010001>.
- Han, S., Tai, C., Jones, C. J., Scheuer, T., & Catterall, W. A. (2014). Enhancement of inhibitory neurotransmission by GABA A receptors having α 2,3 -subunits Ameliorates behavioral deficits in a mouse model of autism. *Neuron*, 81(6), 1282–1289. <https://doi.org/10.1016/j.neuron.2014.01.016>.
- Zaw, Z. F., & Bates, G. D. L. (1997). Replication of zolpidem test for catatonia in an adolescent. *The Lancet*, 349(9069), 1914. [https://doi.org/10.1016/s0140-6736\(05\)63915-3](https://doi.org/10.1016/s0140-6736(05)63915-3).

Zonalon

- Adapin

Zone of Proximal Development (ZPD)

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Definition

A concept developed by the Russian psychologist Lev Vygotsky (1896–1934) to describe the point where a child (or adult) is most able to learn, i.e., just past the point at which a skill/task/activity has been mastered but not so difficult as to be impossible for the child to learn. From the educator's point of view, this suggests giving the child materials/tasks that are carefully selected to enable the child to advance without overwhelming them. Put another way, this notion refers to tasks that the child is capable of learning and performing (often initially with guidance) before these become fully independent activities. High interest in the topic was based on his awareness that for most children certain activities (e.g., language learning) were more effortless than work in other areas (e.g., mathematics). His work also reflected an awareness of distinguishing between development and teaching and has implications for theories like those of Jean Piaget on cognitive development. In Vygotsky's view, a middle ground was needed between self-directed learning and explicit educational instruction. For him a good teacher was one who directed teaching at the child's specific level of ability. His death at a young age meant that he did not fully develop the concept.

The concept of the zone of proximal development prefigures in important ways certain current approaches to treatment and intervention (e.g., in learning difficulties). Other approaches, e.g., Maria Montessori's teaching methods, similarly attempt to match activities with skills that are emerging for the individual child. Other theorists have referred to these concepts in slightly different ways, e.g., J. McV. Hunt (1961) referred to the "problem of the match," that is of being aware of exactly where the child was developmentally and providing a slightly more challenging task. The notion has also been viewed as an important aspect of scaffolding helping the child learn from peers as well as teachers. In this process, the teacher or a knowledgeable peer help a student who is just at his or her zone of proximal development to learning a more challenging task. Clearly an awareness of where the child is actually at is critical in this regard. The concept has been extensively used in educational settings. It has implications not only for teacher-directed learning but the assistance of peers as well.

The social context of children's learning is important given the usual indication of the child to learn by imitation, and this imitation and ability to learn socially provides an important tool for learning. Thus by teaching children at a level slightly above current skill levels, parents and teachers help them expand their abilities. The concept applies not only to more straightforward school-related tasks but other complex activities, e.g., riding a bicycle, learning to drive a car. It is important for parents and teachers to avoid teaching outside the zone of proximal development; conversely, it is also important that parents/teachers are able to sensibly withdraw scaffolding when a child has mastered, or is well on his or her way to mastering, an activity. The use of older peers/mentors can be important in helping the child learning a slightly more challenging task.

For individuals with autism spectrum disorders, an awareness of the child's levels of functioning in specific areas is critical given that some skills (e.g., nonverbal tasks like puzzles) are learned with greater ease than other (e.g.,

language-based) tasks. As with typically developing children, teachers and peers can be helpful in facilitating learning in the child with ASD as tasks are "pitched" superficially at an appropriate level and within the ability of the child with ASD to learn.

See Also

- [Education](#)
- [Social Cognition](#)

References and Reading

- Crain, W. (2010). *Theories of development: Concepts and applications* (6th ed.). Upper Saddle River: Prentice Hall.
- Harwood, R., Miller, S. A., & Vasta, R. (2008). *Child psychology: Development in a changing society*. Hoboken: Wiley.
- Hunt, J. McV. (1961). *Intelligence and experience*. New York: Ronald Press.
- Montessori, M. (1913). *Pedagogical anthropology*. London: William Heinemann.
- Vygotsky, L. S. (1978). *Mind and society: The development of higher psychological processes*. Cambridge, MA: Harvard University Press.

Zyban

- [Bupropion](#)

Zygosity

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Synonyms

- [Allele similarity](#)

Definition

It describes DNA sequence similarity or origin at a particular genetic locus or between twins. In diploid organisms, where one allele is usually inherited from the mother and one from the father, zygosity describes whether these homologous alleles have identical (*homozygous*) or different (*heterozygous*) DNA sequence. A diploid individual may also be missing one (*hemizygous*) or both (*nullizygous*) alleles. Alleles are *autozygous* if they originate from a common ancestor via nonrandom mating or inbreeding; *autozygous* alleles are homozygous. Alternatively, alleles that coincide via random mating are *allozygous* and may be homozygous or heterozygous. Twins are described as *monozygotic* (MZ) when a single egg is fertilized to form one zygote, which later divides into two embryos. *Dizygotic* (DZ) twins occur when two eggs are fertilized independently, yielding two zygotes; on average, they share 50% of their DNA sequence, similar to nontwin siblings. Comparisons of concordance (shared diagnosis) rates between MZ and DZ twin pairs are used to establish the overall heritability of conditions or phenotypes in “twin studies.” Multiple twin studies have been conducted in autism spectrum disorders and demonstrate a significant role for genetic factors in the etiology of these syndromes.

See Also

- [Dizygotic \(DZ\) Twins](#)
- [Monozygotic \(MZ\) Twins](#)

References and Reading

- Hallmayer, J., Cleveland, S., Torres, A., Phillips, J., Cohen, B., Torigoe, T., et al. (2011). Genetic heritability and shared environmental factors among twin pairs with autism. *Archives of General Psychiatry*, 68(11), 1095–1102.
- Lodish, H. F. (2000). *Molecular cell biology* (4th ed.). New York: W.H. Freeman.
- Rosenberg, R. E., Law, J. K., Yenokyan, G., McGready, J., Kaufmann, W. E., & Law, P. A. (2009). Characteristics and concordance of autism spectrum disorders among 277 twin pairs. *Archives of Pediatrics & Adolescent Medicine*, 163(10), 907–914.

Zyprexa

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Synonyms

[Olanzapine](#)

Definition

Despite advancements in cognitive-behavior therapies, no single pharmacological therapy has successfully treated the disruptive-compulsive behaviors of Autism Spectrum Disorders (ASD), which include hand-flapping, tantrums, and self-injurious behaviors (Fido and Al-Saad 2008). Among the pharmacological agents examined to reduce disruptive-compulsive behaviors, atypical antipsychotics, including olanzapine, showed the most promise (Fido and Al-Saad 2008; Malek-Ahmadi and Simonds 1998; Malone et al. 2001). Specifically, ASD children prescribed olanzapine showed significant reductions in irritability, hyperactivity, and lethargy (Fido and Al-Saad 2008; Malek-Ahmadi and Simonds 1998; Malone et al. 2001). Unlike typical antipsychotic medications, atypical antipsychotic medications block postsynaptic dopamine and serotonin receptors and have a lower risk of extrapyramidal symptoms, such as dyskinesias, among children and adolescents (Malone et al. 2001). However, patients prescribed olanzapine experience several side-effects including: hyperlipidemia, hyperprolactinemia, weight gain, and tardive dyskinesia (Fido and Al-Saad 2008). Other case reports have also reported hyponatremia and excessive masturbation from prolonged use of olanzapine among ASD patients (Chiang et al. 2013; Hergüner 2010). In addition, it remains uncertain whether long-term use of olanzapine produces detrimental effect to learning and development of ASD children (Fido and Al-Saad 2008). Further studies of olanzapine are required to

determine its long-term side-effects and potential applications for treating symptoms of ASD.

See Also

- Midazolam
- Neurontin (Gabapentine)

References and Reading

- Chiang, C.-I., Lin, Y.-H., & Hsieh, M. H. (2013). Olanzapine-induced hyponatremia in a patient with autism. *Journal of Child and Adolescent Psychopharmacology*, 23(10), 699–700. <https://doi.org/10.1089/cap.2013.0055>.
- Fido, A., & Al-Saad, S. (2008). Olanzapine in the treatment of behavioral problems associated with autism: An open-label trial in Kuwait. *Medical Principles and Practice*, 17(5), 415–418. <https://doi.org/10.1159/000141508>.
- Hergüner, S. (2010). Excessive masturbation associated with olanzapine in a pediatric case. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 34(7), 1349–1350. <https://doi.org/10.1016/j.pnpbp.2010.06.017>.
- Malek-Ahmadi, P., & Simonds, J. F. (1998). Olanzapine for autistic disorder with hyperactivity. *Journal of the American Academy of Child & Adolescent Psychiatry*, 37(9), 902–903. <https://doi.org/10.1097/00004583-199809000-00006>.
- Malone, R. P., Cater, J., Sheikh, R. M., Choudhury, M. S., & Delaney, M. A. (2001). Olanzapine versus haloperidol in children with autistic disorder: An open pilot study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 40(8), 887–894. <https://doi.org/10.1097/00004583-200108000-00009>.